Sports and Traumatology
Series Editor: Philippe Landreau

Bernard Roger
Ali Guermazi
Abdalla Skaf *Editors*

Muscle Injuries in Sport Athletes

Clinical Essentials and Imaging Findings

Springer

Sports and Traumatology

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As more and more people are getting involved in sports, even the elderly, sports traumatology has become a recognized medical specialty. In sports exercises, every joint and every anatomical region can become the location of a traumatic injury: an acute trauma, a series of repeated microtraumas or even an overuse pathology. Different sports activities may produce different and specific traumas in the same anatomical region.The aim of the book series 'Sports and Traumatology' is to present in each book a description of the state of the art on treating the broad range of lesions and the mechanisms in sports activities that cause them. Sports physicians, surgeons, rehabilitation specialists and physiotherapists will find books that address their daily clinical and therapeutic concerns.

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Clinical Essentials and Imaging Findings

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Foreword

Myology is one of the great frontlines for musculoskeletal medicine across all ages and types of athletes.

The series editor, Dr. Philipp Landreau, an orthopedic surgeon, certainty has served the readership well in selecting an outstanding group of editors for this increasingly important field. The editorial team comprises a powerful triumvirate of leaders in the field, Bernard Roger, Ali Guermazi, and Abdalla Skaf. The editors are known to me not only by reputation and through professional organizations but by personal interactions demonstrating a passion and commitment to excellence for musculoskeletal medicine and education. In addition to the editors, as with many medical texts, one of the pivotal features is the chapter authors. The authors recruited by the editors were carefully selected not only for the knowledge in the domain but for the capability to effectively communicate in written form on the topics.

This well-written text is accompanied by high-quality figures, tables, and images (of course) that complement and augment the descriptions. These contributors really know the material through clinical practice, and this book consolidates the current knowledge of the fields of muscle biology, disease, and therapy that we are all engaged in. This is a significant work as muscle physiology, injuries, and imaging are timely areas of research and new knowledge expansion.

There is a logical organization, and each chapter represents a mini exposition of a particular topic that can stand on its own. As a compendium, the chapters provide a very comprehensive repository of a broad spectrum of sports-related muscle injuries. The topics range from basic to advanced, with equal regard given to common and esoteric conditions, not easily found in one tome. As an author myself, I find this work highly engaging, educational, and easy to digest.

In my estimation as a practitioner with over 20 years' experience, this book is a worthwhile addition to the library of any musculoskeletal medicine provider.

Enjoy and learn!

John A. Carrino (Vice Chairman)

Preface

“Tests conducted this morning on first-team player Lionel Messi have ruled out a hamstring injury in the right leg and he is merely suffering some mild discomfort,” Barcelona FC said in the statement on their website (www.fcbarcelona.es). The test was an MRI!

Indeed muscle injuries are probably the most common injuries in athletes, and probably every athlete will have a muscle injury at least once in their career. What is important then is the extent of the muscle injury, associated lesions, and the time to return to play. Nowadays, imaging is playing an important role in the diagnosis, follow-up, and treatment of muscle injuries, and radiologists and clinicians work in concert to help the injured athlete at every step from diagnosis to preventing recurrence.

The book contains 30 chapters and starts with Part I which focuses on general principles: from the physiology, to general imaging semiology, to treatment. Part II treats nontraumatic muscle injury. Part III is short and contains a chapter about extrinsic muscular injury, while Part IV focuses on intrinsic muscular injury. Part V introduces clinical cases with emphasis on imaging. Finally, Part VI focuses on imaging specifics with a chapter on plasma rich in growth factors for the treatment of muscle injury, a chapter on new advanced imaging techniques, and finally a chapter on body composition measurement using dual-energy x-ray absorptiometry.

We recognize that some of the information in the book may be subjective, but readers can appreciate that the authors of this book are world-recognized specialists. Their years of accumulated experience illuminate their opinions and help to make the book even more valuable to its readers.

We are indebted to all the authors who generously and enthusiastically accepted our invitation to be part of this project and delivered outstanding contributions. We hope that all readers will find this book helpful in daily practice and that the sophisticated information and quality of text and images will be of genuine help to all our colleagues, whether they are dealing with patients playing on an amateur or recreational level or are faced with the specific challenges of evaluating and treating professional athletes with muscle injuries.

We would like to dedicate this book to Professors Gerard Saillant and Karim Khan for their genuine support of our work. We also want to thank our wives Nathalie, Noura, and Caroline and kids Charlène, Romane, Cécilia, Sofia, Dorra, Elias, Valentina, Manel, and Helena for their patience while we were completing this volume.

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Quotes

“Knowledge without action is vanity, and action without knowledge is insanity.”

Abū Hāmid Muhammad ibn Muhammad al-Ghazālī (Algazelus) 1058–1111

“Everyone discusses my art and pretends to understand, as if it were necessary to understand, when it is simply necessary to love.”

Claude Monet 1840–1926

“Success is not final, failure is not fatal. It is the courage to continue that counts.”

Winston Churchill 1874–1965

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Part I
General Principles

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1. Muscle Physiology in Athletes

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Abstract
Skeletal muscle exhibits plasticity in response to physical training. The functional consequences of these adaptations are determined by training volume, intensity and frequency. One could oppose the practice of endurance sports to sports involving strength and speed. Endurance exercise leads to physiological and biochemical adaptations in skeletal muscle which sustain aerobic metabolism capacity such as mitochondrial biogenesis, angiogenesis, and fiber type transformation. Strength training stimulates synthesis of contractile proteins that are responsible for muscle hypertrophy and increased maximal contractile force output. The increase in muscle mass observed in response to strength training is related to hypertrophy of cellular components, with an increase in their number referred to as hyperplasia. These adaptive changes are responsible for the improvement of physical performance. This review focuses on the mechanisms involved in these adaptations. Modifications of muscle typology under the effect of training result from three main factors: nerve stimulation, mechanical stress resulting from the type of physical activity, and the metabolic response to effort. Beside these main factors of muscle adaptation, hormonal response and nutrition can modulate their expression. Recent findings have revealed some of the mechanisms of various signal transduction pathways and gene expression programs in exercise-induced skeletal muscle adaptations. It is now possible to study the effects of various training interventions on a variety of signaling proteins and early-response genes in skeletal muscle. A practical question is whether it is possible to relate muscle structural and

functional capacities to performance. Physiological and possibly pathological structural modifications are appreciated through the various imaging techniques, such as dual energy X-ray absorptiometry (DEXA), radiography and computed tomography, magnetic resonance imaging and ultrasound, all of which have been applied to the study of how changes in muscle mass are effected by training. Measuring cross-sectional surface area by means of ultrasound helps evaluate with precision the increase in segmental cross-sectional surface area. A non-invasive measurement of the effects of training on muscle typology can be realized using magnetic resonance spectrometry, ^{31}P-MRS.

1.1 Introduction

To begin, a brief overview of the normal structural and functional mechanism of skeletal muscle is given.

1.1.1 Skeletal Muscle Function

The skeletal muscle is the only organ that ensures the biomechanical work of locomotion. It is able to transform the biochemical energy contained in energetic substrates into mechanical energy. Its structure and functional capabilities are adapted to the various types of constraints it endures. The level of physical activity, defined in terms of work load and intensity, influence the total muscle mass as well as the muscle's metabolic and contractile properties. According to the type of physical training, muscle adaptations will be focused either on development of muscle power or the capacity to sustain prolonged work. This leads to the distinction between the effects of strength training and the effects of endurance training. Recently, our interest has centered on the effects of workouts that combine strength and endurance. The muscle's ability to adapt to the various demands is referred to as muscle plasticity. To understand the hierarchy of such factors of muscle adaptation to training conditions, the first step is to give a short description of muscle structure and muscle contraction mechanisms. This serves as a basis for explaining the molecular mechanisms involved in muscle plasticity which are beginning to be well determined.

1.1.2 Muscle Fiber Composition

Myofibrils represent the histological entity of the striated muscle. The particular structure of the myofibrils gives the skeletal muscle its "striated" appearance. The proteins that make up such myofibrils are organized as sarcomeres delimited by Z-shaped striae. Sarcomeres are organized in series along the muscle fibers. From the sarcomere's periphery to its center, there is a succession of I bands (clear, called isotopic) and A bands (dark, called anisotropic). I bands consist of fine filaments made up of two spiral chains of globular actin enabling control of the contractile property of

actomyosin filaments by calcium. A bands consist of thick filaments of myosin molecules, the main contractile element of muscles.

1.1.3 Muscle Contraction Mechanism

Muscle contractions result from the transformation of chemical energy into mechanical energy through the slip of contractile protein filaments, actin and myosin. Chemical energy is provided by the hydrolysis of adenosine triphosphate (ATP) under the influence of the ATPasic activity of the myosin head. The hydrolyzing activity of this enzyme regulates the slip speed between the myofilaments. The contractile process mainly depends on the nerve command which regulates motricity at the central level of the organism. This nerve control is the contractility excitation factor which is based on the transmission of information from the muscle periphery to the inner area of the cell by coupling excitation and contraction. This coupling and the contraction-release cycle of the muscle fiber are directly related to an increase in the concentration of intracytoplasmic ionized calcium [32]. The rise in calcium produces an interaction between the actin molecules and the myosin head which conditions the slip of filaments and so ensures the mechanical phenomenon of muscle contraction. Release follows the contraction. After having been brought in contact with the ATPasic site of the myosin molecule head, the ATP is hydrolyzed and the link between actin and myosin is broken. This time corresponds to the re-uptake of calcium by the sarcoplasmic reticulum; these various stages are energy consuming. Power, resistance to fatigue, and contraction speed are dependent on the nature of contractile proteins and the metabolic resources of each muscle fiber. There are several types of muscle fibers that can be classified into different types based on their contractile and metabolic properties (reviewed by Schiaffino and Reggiani [41]). There are two major motor units: slow type motor units (slow type I), characterized by the slowness of their contraction, the low value of their mechanical power, and their resistance to fatigue. By contrast, fast type motor units are characterized by their fast contraction and high power. They are classified as fast-fatigable (fast twitch IIb/IIx) or fast-resistant (fast twitch IIa), according to their resistance to fatigue. IIx fibers are the fastest and have a strictly anaerobic metabolism. IIa fibers have a mixed aerobic and anaerobic metabolism. Contractile properties, notably the contraction speed of the motor unit, are closely dependent on the speed of ATP hydrolysis, which in turn, depends on polymorphism of contractile proteins. This variability is largely determined by the various types of myosin heavy chains. The type of fibers is determined through myosin heavy chains. The fibers listed as IIb in the classification based on ATPase coloring actually contain type IIx myosin heavy chains. In humans, only type IIx fibers are expressed. Each myosin molecule is formed by the association of two polypeptide chains said to be “heavy” that are called myosin heavy chains and four light polypeptide chains that are called myosin light chains. In humans

there are three isoforms of myosin heavy chains–slow type I, fast types IIa and IIx. Slow type I is expressed in slow fibers and the two others in fast fibers. The various isoforms of light chains play a role in the sensitivity of the ATPase site of heavy chains by modulating sensitivity to calcium. Type I myosin heavy chain produces an actomyosin interaction which is slower than rapid type chains. The modifications of myosin isoforms are the most relevant indicators of muscle remodeling in response to training [43].

The metabolic properties depend on enzymatic resources and mitochondrial density. Type I fibers have a strong mitochondrial density and enzymes directing metabolism towards oxidative pathways. They are capable of using carbohydrate or lipid substrates and they are also the seat of the oxidation of certain amino acids during muscular work. Rapid fibers are classified into two sub-sets that differ in terms of their metabolic capabilities. Type IIa fatigue-resistant fibers are able to ensure a substantial oxidative metabolism, while type IIb/IIx fatigable rapid fibers have an essentially anaerobic metabolism.

1.1.4 The Capillary Network

The capillary network of the striated muscle is of paramount importance in as much as it represents the exchange interface between the vascular bed and the muscle fiber. It is the transport and distribution system of oxygen and substrates. The diffusion of capillary oxygen at the center of the fiber is an essential factor for muscle metabolism during the contraction phase and during recovery. It is essential to emphasize that the capillary layout is of considerable importance for the assimilation of substrates and the release of metabolites. The number of capillaries that come in contact with a fiber and thereby contribute to its supply depends on the metabolic property of the latter. Physical training increases capillary density in muscle fibers.

1.2 Effects of Physical Training on Muscle Structures

1.2.1 Effects of Endurance Training

When we attempt to map out the effects of training on muscles, we oppose the practice of endurance sports to sports involving strength and speed. The main adaptation of muscle to endurance sports results from the capacity to maintain a substantial flow of energy for a prolonged time without fatigue. Therefore, the use of ATP should be continually compensated by energy processes that balance energy supply and use. This process requires a sufficient oxygen flow and an availability of energy substrates in the muscle. Three major types of adaptations result from this type of training: an increase in mitochondria, a modification of muscle types, and orientation of the use of energy substrates. To fulfil such conditions, endurance training increases the density of

mitochondria, spurs the activity of enzymes of the Krebs cycle, favors capillary proliferation around muscles, and increases intra-muscular glycogen and triglyceride reserves. The density of mitochondria increases relatively rapidly at the start of training. A 40 % increase in the total volume of mitochondria in the quadriceps of sedentary individuals can be seen after six weeks of endurance training [24]. This adaptation is partly responsible for the increase in maximal oxygen uptake. Increasing the volume of mitochondria per muscle unit allows preferential use of lipids which is facilitated by an increase in intra-muscle lipid content [26]. It has been well established that endurance training causes a transition from glycolytic type fast fibers (type II) to slower oxidative type fibers (type I). The effects of endurance training have been a controversial issue for many years. Early studies on the typical features of subjects according to physical activity showed that among the main locomotor muscles such as the quadriceps, endurance athletes had a predominance of type I fibers that could reach up to 90 % of total fibers whereas sprinters had a predominance of type II fibers. Such differences have been attributed to natural aptitude rather than training. Discussion remains open to what extent such muscle modifications in athletes who succeed in endurance disciplines is the result of training or genes [39]. This issue has been explored in studies of monozygotic and dizygotic twins [11], the results of which indicate that 25–50 % of the variability in muscle typology is hereditary. Among the many genes that have been identified as able to influence global response to training, a peroxisome proliferator activated receptor α (PPARα) can be isolated from the gene that regulates actinic production (ACTN3 gene); both genes are associated with muscle typology [37]. The α-Actinin-3 is a muscle protein that interacts with metabolic and nervous signals of adaptation to training. The *ACTN3* 577XX type polymorphism is more common in endurance athletes [16]. It has now been well established that endurance training on top of genetic factors causes a transformation of muscle fibers towards a predominance of slow fibers and also produces a transition from type IIb glycolytic fast fibers to type IIa oxidative fast fibers [42]. This type of transition is advantageous because it reduces muscular fatigue considerably. Oxidative slow fibers have a section surface weaker than fast fibers; this enables a better diffusion of oxygen towards the mitochondria but also has the disadvantage of reducing force capacity per motor unit.

1.2.2 Effects of Strength Training

Several factors determine strength production by muscles. These are successively the surface of the muscle section, the typology of the muscle fibers, the number of motor units solicited and an anatomic insertion parameter of muscles on the bone segments, known as the pennation angle. The ability of an athlete to develop significant strength mainly depends on structural and neural factors [9]. Structural factors relate to muscle

composition itself whereas neural factors relate to the use of motor units. The increase in muscle mass observed in response to strength training is related to a hypertrophy of cellular components, with an increase in their number referred to as hyperplasia.

1.2.2.1 Hypertrophy

Muscular strength production depends directly on the muscle section [27]. Strength training rapidly produces an increase in the section surface of muscle fibers. Studies indicate an increase of 5–10 % in the lower limbs and 15–30 % on the upper limbs after 10–15 weeks of training [3]. Chronic muscle hypertrophy is an indication of structural modifications in muscles: multiplication and hypertrophy of myofibrils, and an increase in the connective tissue. This hypertrophy phenomenon is very important in athletes who have been undergoing strength training for several years and can be translated by an increase of over 80 % in the cross section surface of muscle fibers compared to the sedentary population. Hypertrophy relates to all types of muscle fibers with predominance of type IIb; the surface increase of IIa fibers is particularly clear in the upper limb muscles of weightlifters [28]. The hypertrophy mechanism results from the stimulation of satellite cells situated at the periphery of each fiber. Satellite cells are myogenic precursor cells situated between the sarcolemma and the basal plate of muscle fibers that induce the proliferation of the number of nuclei. The increase in fiber size results from the addition of the number of nuclei [51]. The same mechanism is involved in muscle repair after an injury [40]. This parallel action supports the theory that hypertrophy is regulated by the production of micro-lesions as a result of training and which, during repair, facilitate muscle hypertrophy [21]. Several studies underscore an increase in the contents of satellite cells in response to strength training [28]. However, this activation of satellite cells might have several physiological explanations. In one view, activation of such cells is an indication of their powers of regeneration in response to small lesions resulting from strength training. Or perhaps such activation is an indication of the incorporation of new nuclei by muscle fibers which could participate in addressing fiber synthesis needs and therefore muscle hypertrophy. Satellite cells multiply (proliferation step) and fuse with each other and with pre-existing muscle fibers (differentiation step) to enable muscle tissue regeneration [15]. The increase in muscle volume also results from the development of muscle envelopes as the connective tissue adapts to the mechanical constraints of training; the increase in the strength of this connective tissue is the consequence of the regeneration of muscle lesions due to physical exercise.

1.2.2.2 Possibility of an Increase in Muscle Cell Hyperplasia

The phenomenon of hypertrophy of muscle fibers is supported by multiplication of the muscle fibers: the hyperplasia phenomenon. The increase in the number of muscle cells

in response to strength training was first demonstrated in animal models. In humans, the exact contribution of hypertrophy and hyperplasia in the increase of muscle volume remains controversial [19]. The mechanism responsible for this phenomenon could be stimulation of myogenesis by growth factors. Muscle hypertrophy and hyperplasia together make up a protective mechanism by which unusually intense contraction stimuli are distributed over a larger muscle mass, therefore offering relative protection against overload, as the load borne by each muscle fiber is reduced.

1.2.2.3 Effect of Strength Training on Muscle Typology

Muscle power is largely dependent on the maximum contractile speed. Apart from muscle mass itself, the muscle's composition of the various types of fibers influences strength. The more important density in myofibrils of type IIA fast fibers and especially type IIX improve upon the speed and strength of muscle contraction. High level weightlifters who have regularly undergone strength training for several years have a higher percentage of type IIA rapid fibers and a lower percentage of type I slow fibers [28]. Change in density also leads to modification of muscular metabolism. Hypertrophy in response to strength training modifies muscular metabolism by reducing oxidative capabilities. These result from the decrease of mitochondria density in muscles, due to an increase in the quantity of myofibrillar proteins. The decrease in mitochondrial density is associated with a decrease in the activity of Krebs cycle oxidative enzymes [12].

1.2.2.4 Role of Nerve Stimulation

The observed rapid increase in strength at the beginning of strength training before the appearance of structural phenomena is attributed to nervous factors of adaptation to this type of training [20]. Such neural factors are highlighted by the increase in EMG activity which indicates an increase in the number of recruited motor units and an improvement of motor unit synchronization [35]. Motor units are recruited according to their size, from smaller to bigger, referred to as Henneman's size principle [22]. Subjects who undergo strength training are therefore able to activate a more important number of motor units within the solicited muscle, thus allowing them to develop greater strength. The structures responsible for this adaptation are situated at the peripheral stage of the neuromuscular junction but also at the cortical level of the motor command [17]. This adaptation occurs on a very short term after only a few weeks of training and is associated with an increase in the stiffness of the muscle-tendon torque, which favors muscle performance [50].

1.2.2.5 The Role of Muscle Architecture

Muscle contraction strength is influenced by a stress-length relation. For each muscle fiber there is an optimal tension which, according to the initial model by Sonnenblick and Skelton [46], enables a greater juncture between actin and myosin filaments. Anatomically, the arrangement of individual fibers affects their traction angle (pennation) as well as the articulation angle which influence muscle length and the capacity to produce strength and contraction speed. The decrease in pennation angle helps obtain an optimum length when relaxed to favor contraction speed. The longer the fibers are, the more they have of sarcomeres in series, which is favorable to speed; sprinters have more lengthening of muscle fibers than long distance runners [2]. Kumagai et al. [33] showed significant differences in the pennation angle between two groups of sprinters: 10 s sprinters have longer fiber bundles than 11 s sprinters, as well as a lower pennation angle and are more effective at producing speed. Moreover, the pennation angle also increases total muscle mass connected to a tendon [29]. This has a genetic aspect but may also be a result of training. Muscle hypertrophy in response to strength training is associated with a decrease in the pennation angle [1].

1.3 Mechanisms of Muscular Plasticity in Response to Training

The modification of muscle type under the effect of training results from three main factors: nerve stimulation; the nature of the mechanical stress from each type of physical activity; and the metabolic response to effort, hormonal response and nutrition which can modulate the expression of these factors [30]. The stem cells situated on the periphery of muscle fibers, the satellite cells, allow muscular plasticity in response to physical training [28]. We shall list the main muscle development factors and their mechanisms.

1.3.1 Neural Factors

The nature of nervous control probably plays the main role; experiments on animal models have well indicated that it is possible to transform the typology of a muscle by varying the nature of nerve stimulation [25]. Nervous control of motor skills acts initially on calcium entry into the muscle cell. Calcium transmembrane entry activates the cyclical release of the intracellular calcium stored in the sarcoplasmic reticulum. Calcium is fixed on binding proteins called calmodulins. The modulation of calcium flow activates phosphatases called calcineurins, which at the end of the chain activate a nuclear transcription factor, the nuclear factor of activated T cells, or NFAT [38]. This cascade of events acts on the metabolic and structural differentiation of muscles. The calcium signal activates the muscle degradation pathways. Degradation is a precondition to the renewal process. There are several degradation pathways that are

dependent on calcium activation or energy status and that may or may not undergo lysosomal activation. Such processes come into play during muscle regeneration after immobilization [48]. The activation of this signaling pathway seems to be primarily responsible for the transition towards oxidative fibers, mitochondrial angiogenesis and biogenesis under the effect of endurance training [54]. An important element in the transmission of neural messages via calcineurin is the role of the myogenic family. Myogenins, four in number (MyoD, MRF4, myogenin, Myf5), are proteins that interact with the genome and regulate the gene expression of muscle proteins. Myogenic regulatory factors are a family of transcription factors known to play an important role in myogenesis or during the initial training of skeletal muscle. Parallel to this function of coordination of the myogenes, myogenic regulatory factors have been invoked to explain the transitions between the various types of fibers in adult muscles. They are particularly involved in the increase of muscle oxidative capabilities in response to training [45]. The action of myogenins is balanced by a powerful factor that limits muscle growth by acting at the level of the expression of muscle genes, namely myostatin. Myostatin belongs to the transforming growth factor family. It permanently inhibits muscle mass development [34].

1.3.2 Mechanical Stress

Mechanical stimuli resulting from physical activity act on the muscle membrane; their translation into intracellular signals is mediated by age, sex, pre-existing muscle architecture and metabolic status; such signals modulate the transcription and translation of contractile and metabolic proteins. Mechanical stress can be passive in response to muscle stretching or active due to the shortening of contraction. Stress is accounted for at the level of the membrane by a system of adhesion molecules, integrins; such proteins act on the transduction factors (mitogen activated protein kinase, MAPk) which stimulate the factors of stimulation of genome expression (cJUN, HSP70). Other muscle tension sensors are situated inside the contractile machine. The effectiveness of this system is highlighted by the fact that simple prolonged tension in a muscle helps maintain a part of muscle mass. From a practical point of view, knowledge of such mechanical factors helps to adapt the physical activity to promote muscle development. To optimize muscle development, activities need to impose a level of mechanical stress which is sufficient to the purpose and acceptable to the subject. This principle finds its application in training or rehabilitation methods that use eccentric muscular work. This work consists of stretching the muscle against its insertion points during a muscle contraction to slow down lengthening. This type of stress is obtained when landing from a downward jump; it is sought in plyometric-type workouts.

1.3.3 Metabolic Factors

The energy flow of muscle contraction leads to a decrease in the intramuscular energy charge. The result is an increase in the AMP/ATP ratio. The activation of an enzyme system AMPkinase is considered a sensor of the cell's energy status. This system acts on muscular protein synthesis. The AMPk pathway stimulates the expression of specific genes of muscle adaptation to increase energy expenditure; these are mainly the genes encoding mitochondrial proteins [6]. The increase in AMPk activity inhibits protein synthesis during the muscle exercise phase by reducing the activity of an intracellular signaling system, the mammalian target of rapamycin, or mTOR [14]. During the recovery phase, activity in this system rebounds. This biphasic action explains the fact that the increase in muscular protein synthesis in response to muscular exercise occurs during the recovery phase and seems to be related to the recharge of energy. Another metabolic factor that acts on muscle development is influenced by the level of muscle oxygenation. It is the hypoxic inductible factor. Magnetic resonance spectroscopy techniques have highlighted a decrease in local oxygen stress at the beginning of muscle contraction. This local hypoxemia is said to stimulate the production of hypoxic inductible factor; this action may favor the biogenesis of mitochondria [44]. Knowledge of the exact role of such metabolic factors shows that the adaptation of muscles to physical training occurs after substantial metabolic stress. These elements explain the fact that short intense intermittent work helps to obtain metabolic adaptation to muscle exercise.

1.3.4 Role of Hormones

Several hormones play a role in muscle development. Schematically, one can distinguish hormonal axes that act mainly on the development of muscle mass and hormones that act on muscle tissue differentiation. The increase in muscle mass results from the coordinated actions of steroid hormones, the somatotrope axis and insulin. Studies that have attempted to describe their respective roles have analyzed the evolution of such hormones in response to different types of training. Steroids effect muscle development at all stages of life; it is very clear during puberty in males. The decrease of all steroids with age in both sexes might also be associated with a decrease in muscle mass and a concomitant increase in body fat. Physical training influences circulating concentrations of gonadal steroids. Such variations are small, however, compared to the pharmacological doses involved in the increase of muscle volume [8]. In contrast, the response of the somatotrope axis is important under the effect of muscle exercise and training [52]. Insulin growth factor IGF-1 is also stimulated during physical exercise and plays a role in the response of muscle anabolism [31]. The role of hormones on the typological determinism of muscle fibers seems mainly assigned to thyroid hormones. Thyroid hormones seem indispensable in inducing an increase in the percentage of fast fibers; their deletion prevents fast contractile protein synthesis.

Thyroid hormones act on the expression of fast contractile proteins by means of the myogenin system [18].

1.3.5 Role of Nutrition

It has been well proven that the muscle growth depends on both protein contribution and total calorie intake [10]. The existence of a threshold below which muscle growth stops and then becomes negative has helped define minimal intakes. A study on nitrogen balance in various types of athletes has helped measure the effects of the type of endurance or strength training on nitrogen balance and body composition [49]. The results of the study show that protein intake adequate to balance nitrogen balance is 1.2 g of proteins/kg/day for muscle building and 1.6 g proteins/kg/day for enduring athletes. The nature of ingested amino acids influences the level of protein synthesis; leucine availability is a major determining factor of muscle protein synthesis under the effect of strength training.

Moreover, physical activity is the source of a substantial increase in energy metabolism that may be responsible for muscle damage by means of free radical production [4]. Under the effect of intense exercise, during which oxygen uptake can be multiplied by a factor of 20 and that of skeletal muscle may increase up to 200 times, skeletal muscle mitochondria become the site for the formation of free radicals derived from oxygen [13]. Furthermore, within active muscles, highly transient ischemia occurs. During exercise or after, ischemic areas are rapidly reinfused thereby contributing to the production of free radicals [53]. Such free radicals are responsible for cell damage and especially cell micro-lesions. The lesions are responsible for an inflammatory syndrome by means of the production of pro-inflammatory cytokines (TNF-α, IL-6, IL-1β). The consequences of such cell damage are assessed via the increasing the plasma concentration of intracellular enzymes (CK, LDH, PK, AST) and pro-inflammatory cytokines (IL-1Ra, IL-6, TNF-α). The susceptibility of an organ to damage induced by free radicals depends on the balance between oxidative stress and antioxidant capacity. Defense systems are made of an endogenous device of enzymatic nature represented among others by superoxide dismutase (SOD) and catalases. In the skeletal muscle, cytosolic SOD activity represents 65–85 % of total antioxidant activity. Dietary intake can play an important role by providing antioxidant vitamins (A, C, and E) and enzyme co-factor trace elements of antioxidant enzymes (zinc, copper, and selenium). Another reactive species, nitrogen monoxide (or NO) is also produced during physical exercise by two major enzyme systems, the NO synthases and the independent pathway of NO synthases. An increase in the production of NO can have, on the one hand, a favorable action on muscle function by increasing local blood flow and, on the other hand, an adverse action because the concomitant production at the same site of NO and superoxide is very harmful by giving birth to the peroxynitrite radical that, in turn, can

break down leading to the formation of free radicals. The intake of nutrients stimulating NO production causes the pro-oxidant balance to tip in in favor of muscle protection; mainly those containing arginine, citrulline and nitrate [7].

1.4 Practical Applications

1.4.1 How to Assess Muscle Mass

How to assess muscle mass is a practical question within the framework of the assessment of muscle capabilities and monitoring of training; it relates to measuring the total muscle mass and segmental muscle mass. A precise answer to this question helps to adapt strength training plans and to verify the effectiveness of the various types of nutritional intakes used to optimize molecular mass. A method is chosen based either on its precision or its ease of implementation. Several methods are possible; the reference method for measuring total muscle mass is hydrostatic weighing, which consists of measuring body density by weighing the body in water. The complexity of this method makes it difficult to apply in practice, which justifies the need for image-based methods. These methods can use ultrasound, dual energy X-ray absorptiometry (DEXA), X-ray scanner and MRI. Adding to these imaging methods are bioelectrical impedance and skin-fold measurement. Nelson et al. [36] have shown that single-slice computed tomography (CT) scanning is the most precise method compared to DEXA or bioelectrical impedance for measuring fine variations of segmental muscle mass at the end of a prolonged strength training period. The major disadvantage of this method is the administration of radiation. More recently, magnetic resonance imaging has proved to be the best method for measuring total and segmental muscle mass [3]. This study which aimed to measure the effects of strength training in a year also highlights the limits of bioelectrical impedance. This technique is highly influenced by the variations of the fluid compartment that can occur irrespective of muscle mass gain. Measuring section surface by means of ultrasound helps evaluate with precision the increase in segmental section surface [5]. This method has the advantage that it does not produce ionizing radiations, it is less costly, and is performed with easy-to-carry material.

1.4.2 How to Assess Muscle Typology

Classification of muscle typology relies on the histomorphometric analysis of muscle sample taken by biopsy. This method can hardly be applied in the follow up of training, which is why non-invasive methods for determining muscle typology are needed. A first approach indirectly assesses the typology of a muscle group by means of muscle performance tests. The most stringent approach links functional results according to the speed obtained on an isokinetic dynamometer; results are adapted to the assessment of the percentage of fast and slow fibers in lower limb muscles such as the quadriceps

[47]. Non-invasive measurement of the effects of training on muscle typology can be realized using magnetic resonance spectrometry, ^{31}P-MRS. An analysis of the ratio [PCr]/[P*i*] [PCr]/[ATP in a 1.5 T magnetic resonance imager is relatively well correlated with the distribution of type II fibers in lower limb muscles [23].

1.5 Conclusion

Knowing the various mechanisms that regulate the plasticity of muscle helps to adapt muscle training to optimize muscle function and performance. Functional responses in terms of strength, speed and resistance to fatigue help evaluate the response to the various types of training; performance tests are adapted to this provision. They can be administered by trainers. Physiological and possibly pathological structural modifications can be appreciated through the various imaging techniques. The choice of the type of imaging will depend on whether the total or segmental muscle mass is being measured, by the availability of material, and by the competence of the operators. The interpretation is necessarily medicalized.

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2. Functional Anatomy of the Muscle

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Abstract
For around 20 years, structural organization of muscle has been correlated with innervation and the functional capacities of muscle, via the concept of neuromuscular compartments. Additionally, it is now accepted that intramuscular fibrous structures and tendons form an integral part of muscle organization. Particular attention should therefore be given to the connective tissue network and myotendinous junctions. In this chapter, we have chosen to present first the macroscopic architecture of muscle and the concept of neuromuscular compartments. In the second part, we consider the major types of muscle organization for both limb muscles and static muscles. The organization of tendons is developed in the third part, based on examples of injuries suffered by athletes, such as the proximal hamstring complex or the femoral portion of the iliopsoas muscle. Finally in the last part, the issue of muscle variations is discussed using examples that illustrate their functional and morphological repercussions.

2.1 Introduction

The functional anatomy of muscle is a very broad topic. For many years, muscles were viewed and studied from a purely descriptive and morphological perspective. Over the last twenty years, their structural organization has been correlated with their functional

abilities and innervation, particularly via the concept of neuromuscular compartments. Additionally, it is now accepted that intramuscular fibrous structures and tendons form an integral part of muscle organization. Particular attention should therefore be given to the connective tissue network and myotendinous junctions.

In this chapter, we have chosen to present first the macroscopic architecture of muscle and the concept of neuromuscular compartments, detailing the example of the triceps surae muscle, an important muscle in athletes. In the second part, we consider the major types of muscle organization for both limb muscles and static muscles, such as periscapular muscles. The organization of tendons is developed in the third part, based on examples of injuries suffered by athletes, such as the proximal hamstring complex or the femoral portion of the iliopsoas muscle. Finally, the issue of muscle variations is discussed using examples that illustrate their functional and morphological repercussions, especially in imaging.

2.2 Muscle Architecture

The architecture of a given muscle is the major determinant of its function. The main components of this architecture are muscle fibers, which are inserted on intramuscular fibrous structures. The latter form a connective tissue network, a “fibrous skeleton” of muscle, including intramuscular aponeuroses which then condense into tendons inserted on the bone. Each muscle is surrounded by a fibrous envelope, of varying density, called the muscle fascia. Muscular force, produced by the contraction of muscle fibers, is transmitted to the connective tissue network that contains myotendinous and myofascial pathways.

Muscle architecture helps define the concept of muscle compartments as separate functional subunits, formed of morphologically and functionally identical muscle fibers. Each compartment is innervated by an individual nerve branch and controlled by a specific spinal neuronal population [12, 46, 54].

To better understand muscle architecture, it is important to detail the organization of muscle compartments. It is also useful to present the musculotendinous organization of the triceps surae muscle, given its specificity in humans and the frequency of its injuries to this muscle in athletes.

2.2.1 Muscle Compartments

The concept of muscle compartments is now widely accepted. Muscle anatomy has been progressively reviewed in light of this concept, enabling a better understanding of the principles of muscle innervation. According to muscular partitioning, a muscle may be differentially activated depending on the required function of the muscle, thus allowing multifunctional muscles to contribute to a variety of movements [47]. In practice, this

elaborate macroscopic organization helps to understand the different types of musculotendinous injuries. It must also be the basis of surgical considerations of muscle transfers and is also useful for the placement of electrodes in electromyographic studies.

The partitioning of the hamstrings has been well studied. Their nerves emanate from the sciatic nerve or tibial nerve. The semitendinosus muscle comprises two distinct partitions that are arranged in a series and are divided by a tendinous inscription. A single muscle nerve innervates each partition. The semimembranosus muscle includes three distinct and superposed compartments. The superior and inferior compartments are innervated by individual nerve branches. The nerve of the superior compartment also innervates the postero-medial head of the adductor magnus muscle. The middle compartment is sometimes innervated by a collateral branch of one of the other two nerves. The long head of the biceps femoris muscle is organized into two compartments innervated by specific nerves [56] (Fig. 2.1).

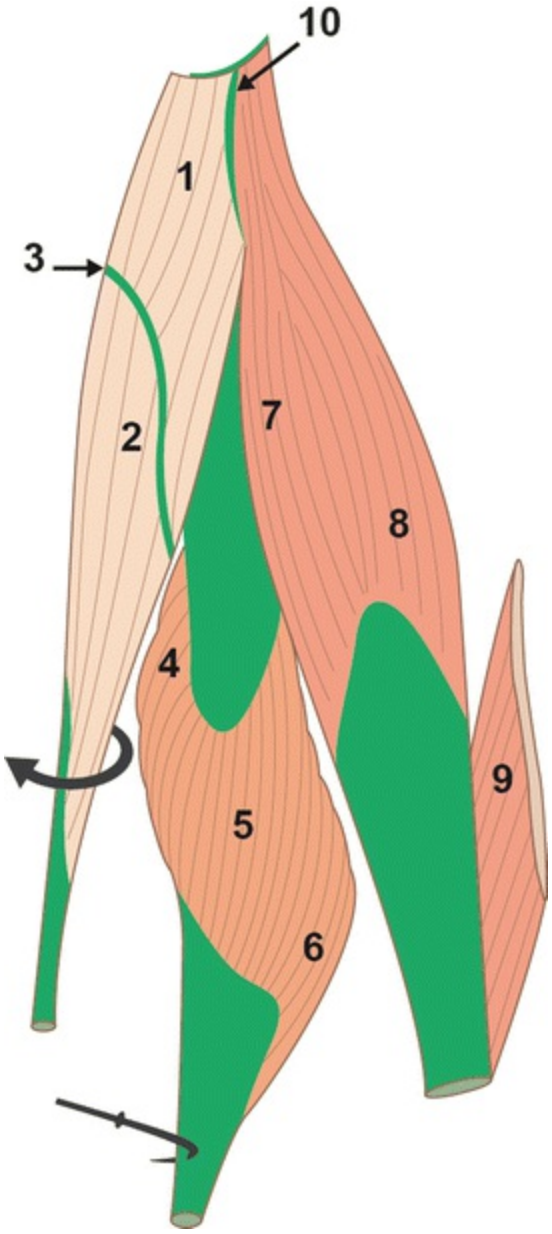

Fig. 2.1 The hamstring muscle complex. (*1*) Superior compartment of semitendinosus – (*2*) Inferior compartment of semitendinosus – (*3*) Tendinous inscription of semitendinosus – (*4*) Superior compartment of semimembranosus – (*5*) Middle compartment of semimembranosus – (*6*) Inferior compartment of semimembranosus – (*7*) Medial compartment of the long head of the biceps femoris muscle – (*8*) Lateral compartment of the long head of the biceps femoris muscle – (*9*) Short head of the biceps femoris muscle – (*10*) Common tendon of the semitendinosus and long head of the biceps femoris muscles

The study of neuromuscular partitioning of the extensor carpi radialis muscles was based on extra- and intramuscular innervations. The extensor carpi radialis longus muscle comprises a superficial compartment and a deep compartment, each innervated by a specific branch of the radial nerve, dividing into the anterior and posterior branches. Partitioning of the extensor carpi radialis brevis muscle is more complex comprising two to four compartments. The increased number of neuromuscular partitions in extensor carpi radialis brevis when compared to extensor carpi radialis

longus could be due to the need for more differential recruitment in this muscle depending on force requirements [37].

The serratus anterior muscle can be separated into three parts based on its origin and insertion. The superior part originates from the first and second ribs, and is inserted onto the superior angle of the scapula. The middle part, originating from the second and third ribs, inserts on the medial border of the scapula and the inferior part originates from the inferior and third ribs and inserts on the inferior angle of the scapula. The long thoracic nerve supplies the three parts. The superior part receives additional innervation by a nerve branch also innervating the levator scapulae muscle. The inferior part is additionally innervated by branches of the intercostal nerves. Understanding the characteristics of the innervation in each part is useful in identifying the cause of dysfunction of the muscle [33].

2.2.2 Architecture of the Triceps Surae Muscle

The triceps surae is a musculotendinous complex involved in balance and walking. It is a heavily used muscle in athletes. The complexity of its architecture is linked to its fundamental role during the various phases of walking and running.

The triceps surae muscle-tendon complex, innervated by the tibial nerve, is classically composed of two gastrocnemius muscle heads and the soleus muscle. Both gastrocnemius heads are in fact the same muscle. The gastrocnemius muscle is composed of two independent heads, the lateral head and medial head (Fig. 2.2).

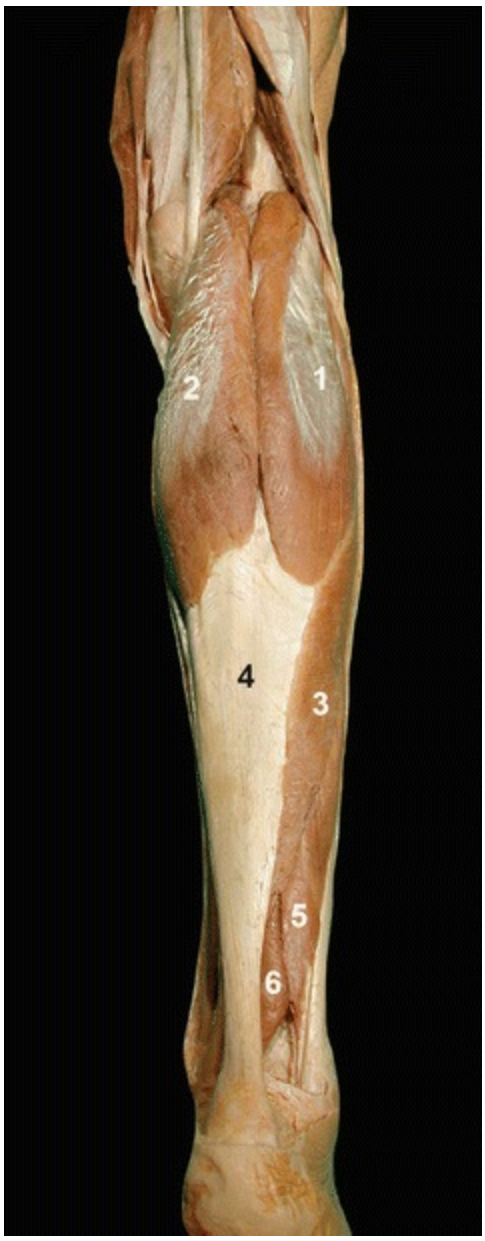

Fig. 2.2 Posterior view of the right leg shows the lateral gastrocnemius muscle (*1*), the medial gastrocnemius muscle (*2*), the soleus muscle (*3*), the calcaneal tendon (*4*), the fibularis brevis muscle (*5*) and the flexor hallucis longus muscle (*6*)

The study of compartments enables a different conceptualization of the architecture of the triceps surae complex. Gastrocnemius is a digastric muscle with two juxtaposed bellies and each head has proximal insertions and a common terminal aponeurosis. The lateral head is divided into three muscle compartments [46]. The medial head is organized in a single compartment [27]. The soleus muscle can be divided into two compartments [13, 25]. The dorsal portion is situated between a dorsal aponeurosis and a ventral aponeurosis, which can be incomplete. Short muscular fibers inserted on these aponeuroses are oriented obliquely from the rear towards the front, from top to bottom, converging towards the center of the muscle. The ventral portion is inserted into the

ventral concavity of the ventral aponeurosis of the dorsal portion. It is a bi-pinnate muscle with an intramuscular septum at its center [35] (Fig. 2.3). The terminal aponeurosis of the gastrocnemius muscle binds to the dorsal aponeurosis of the soleus muscle to which the intramuscular septum is attached. This distal fibrous unit becomes a powerful tendon, the calcaneal tendon, common to the heads of the gastrocnemius and the soleus muscle. The long tendon of the plantaris muscle slides between the gastrocnemius and soleus muscles, attaching on the medial aspect of the calcaneal tendon.

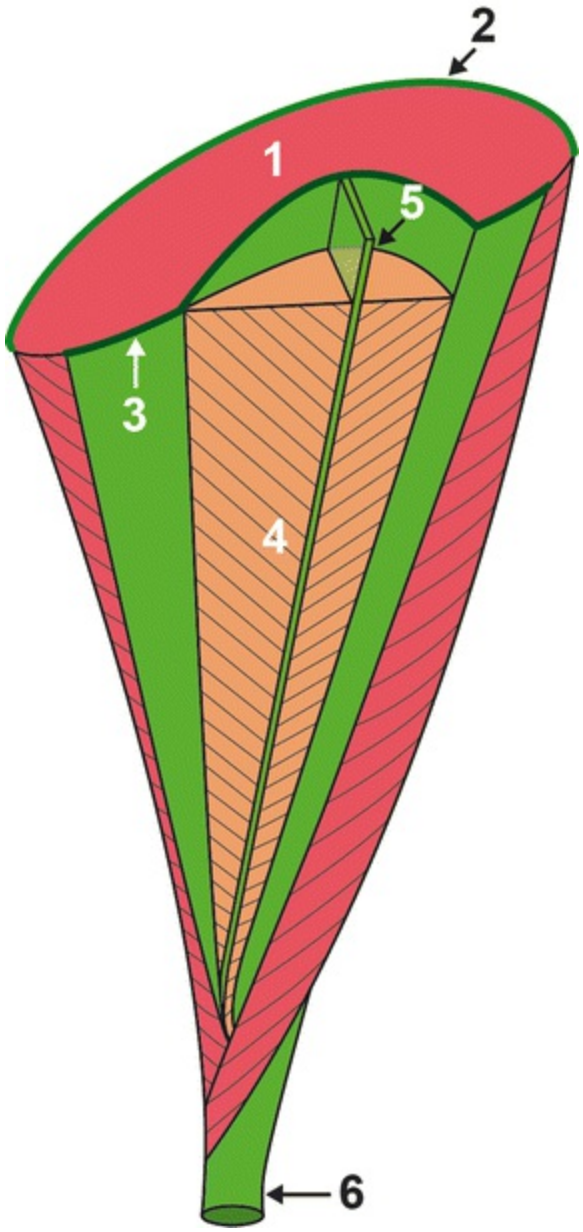

Fig. 2.3 Schematic drawing of the soleus muscle shows the different compartments. (*1*) Dorsal portion of the muscle – (*2*) Dorsal aponeurosis – (*3*) Ventral aponeurosis – (*4*) Ventral portion of the muscle – (*5*) Septum of the ventral portion –(*6*) Calcaneal tendon

Innervation of the lateral head of the gastrocnemius muscle is provided by two or three nerve branches [35, 45, 46]. Innervation of the medial head is extremely variable and systematization is not feasible [55]. The soleus muscle is innervated by two nerve pedicles. The dorsal nerve for the dorsal compartment divides rapidly in two to three branches that provide branches to the medial and lateral parts of the muscle. The ventral nerve divides usually into two branches which in turn provide branches for each half of the bi-pinnate part of the muscle [28, 35].

The architectural complexity of the muscle is demonstrated by its functional complexity but the physiological action of each part remains unclear [28]. However, this organization of muscle, aponeurosis and tendon demonstrates extensive areas of muscle insertions. Each is susceptible to sustaining injury [3].

2.3 Functional Organization of Muscles

The muscles of the human body are functionally organized. Organization differs significantly between the static muscles of the trunk and the dynamic segmental muscles of the limbs.

Limb muscles are organized in compartments which contain muscles that activate or coordinate movement. It is important to consider the functional terminology of the muscles involved in movements such as flexing the forearm.

Some muscles are both static and dynamic, and their organization is more complex. Periscapular muscles are part of this muscle group and are arranged in a particular muscle system presented in this chapter.

Morphological classification of muscles is antiquated. Muscles were often described at a time when functional aspects were poorly taken into account. Morphological classifications are therefore sometimes ill-adapted to biomechanical reality, as is the case of the deltoid and pectoralis major muscles. This example of functional proximity will also be discussed.

2.3.1 Functional Organization of Limb Muscles

Morphologically, the dynamic muscles of the limbs are mainly long. In each limb segment they are organized into muscular compartments. Each muscular compartment is separated from the adjacent compartments by a septum, a fibrous structure, which inserts on the bone and fascia of the circular envelope of the corresponding member segment. This fascia is of varying thickness depending on the muscle power imposed. For example, in the lower limb, these fasciae are thick so as to contain muscle expansion during contraction and maintain maximum power. In pathological conditions, this barrier to muscle expansion becomes a source of increased pressure in the compartment, creating compartment syndrome, occurring frequently in athletes due to the

repeated activity imposed by the sport concerned.

In the same muscle compartment, some muscles only mobilize a single joint, and are called monoarticular muscles. Others, polyarticular muscles, cross several joints, and can be organized in several muscle compartments. Each limb has a primary function, such as flexion-extension of a joint, integrated into a more specific function, such as prehension in the upper limbs or walking and running in the lower limbs.

Furthermore muscles have specific roles in a given movement. The main activated muscle which produces analytical movement is called the agonist muscle. In the same muscular compartment and adjacent compartments, some muscles participate in the same movement without being the main mobiliser. These muscles are called “congener” muscles. To harmonize movement, other muscles or muscle bundles may act in opposition to the agonist and “congener” muscles. These are the antagonist muscles; they control the speed and accuracy of movement, moderating the action of the agonist muscle. Synergist muscle is another type of functional group of muscles. A synergist muscle is involved concomitantly in the considered movement. It may be an agonist or “congener” muscle, which produce and control movement, or antagonists that neutralize or moderate the execution of muscle movement.

To illustrate the functional organization of the segmental muscles, we will consider flexion of the forearm. It involves three bones, the humerus, radius and ulna. Their epiphyses are grouped into a single joint complex, the elbow joint. The three main flexor muscles of the elbow are the biceps brachii, brachialis and brachioradialis. The brachialis muscle, located in the ventral compartment of the arm is strictly mono-articular, crossing only the elbow joint. The biceps brachii muscle, also located in the same compartment, is polyarticular, passes over the proximal joints of the shoulder and the elbow joint complex. The brachioradialis muscle is also polyarticular, passing over the elbow joints and the distal radio-ulnar joint. It belongs to the ventral compartment of the arm and the lateral compartment of the forearm.

The brachialis muscle is the agonist in flexion of the forearm. It is involved only in this movement. The biceps brachii and brachioradialis muscles are “congeners”. They have other functions in addition to that of elbow flexion. Thus, the biceps brachii muscle is involved in the abduction of the arm and supination of the forearm. The brachioradialis is involved in pronation and supination of the forearm. The main antagonist of elbow flexion is the triceps brachii muscle, located in the dorsal compartment of the arm. The radialis extensor carpi muscles are also antagonists, involved in moderating the end of flexion movement. All these muscles are synergist in flexion of the elbow (Fig. 2.4).

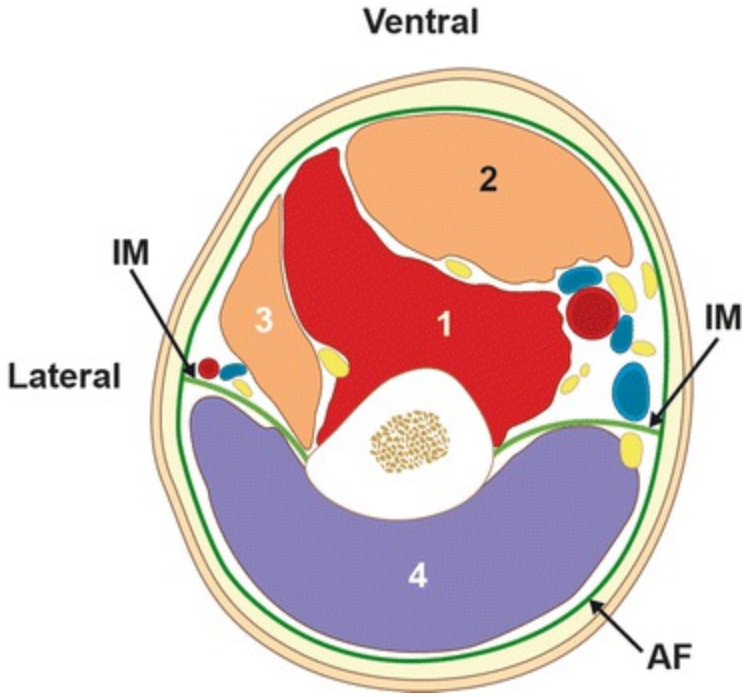

Fig. 2.4 Horizontal section of the arm showing the ventral and dorsal compartments separated by the intermuscular septum (*IM*) and surrounded by the arm fascia (*AF*). Concerning the forearm flexion, the agonist muscle is the brachialis muscle (*1*), the biceps brachialis (*2*) and brachioradialis (*3*) muscles are "congener" muscles and the triceps brachialis (*4*) is an antagonist muscle

2.3.2 The Scapulothoracic Functional Complex

Another type of elaborate muscle organization is that of the periscapular muscular complex. This complex has two functions: stabilizing the scapula and optimizing movements of the upper limb. Functionally, the muscles inserted on the scapula are usually classified into two major groups: the scapulohumeral muscles which help to position the arm in space and the scapulothoracic, or axiothoracic, muscles, which coordinate scapulothoracic movements.

The scapulohumeral muscles include the supraspinatus, infraspinatus, teres major, teres minor, deltoid, biceps brachii, long head of triceps brachii and coracobrachialis muscles. They regulate activities of the glenohumeral articulation and provide power to the humerus.

The scapula is essential in coordinating upper extremity activity. Other than its attachment to the acromioclavicular and sternoclavicular joints, the scapula does not have any other attachments to the thorax. Its stability is provided by the surrounding musculature, the scapulothoracic muscles. They include the trapezius, serratus anterior, rhomboid and levator scapulae muscles. The main convergence of the muscles of this group is the medial or spinal border of the scapula. Indeed, the tendinous attachment of the serratus anterior muscle combines with the ventral tendinous insertions of the levator scapulae and rhomboid muscles, enveloping the entire medial border of the

scapula. Fibers of the latissimus dorsi muscle cover the inferior angle of the scapula during their oblique course. There is no osseous attachment of the latissimus fibers to the inferior angle of the scapula.

The scapulothoracic "articulation" differs from the other joints of the shoulder complex, as there is no articular cartilage, synovium, or capsule but a series of bursal and muscular planes, which allow sliding. The scapulothoracic joint is defined by soft tissue apposition. The subscapularis muscle spreads across the concave ventral scapula, lying over the serratus anterior muscle and the convex thoracic cage.

From a muscular functional perspective, the movements of the scapulothoracic joint are facilitated by three layers of muscles and bursae. The superficial layer includes the trapezius, and latissimus dorsi which is not attached to the inferior angle of the scapula. In some cases a well-defined bursa is present between the superior fibers of the latissimus dorsi and the inferior angle of the scapula. When absent, the space is filled with areolar tissue. The middle layer is formed by the rhomboid and levator scapulae muscles. The scapulotrapezial bursa is always present between the fibers of the middle and inferior trapezius and the superomedial scapula. The deep layer includes the serratus anterior and subscapularis muscles. The scapulothoracic bursa is always present between the thoracic cage and the deep surface of the serratus anterior. A bursa is not always present between the superficial surface of the serratus anterior and the subscapularis [24, 53]. This functional complex enables the scapula to slide on the external surface of the thorax, due to the scapulothoracic articulation, and to perform rotation and translation movements which are not independent of one another.

Instability of this system can result in scapular winging, an abnormal scapulothoracic posture and motion. In high-level athletes, periscapular weakness resulting from overuse may manifest as this type of dysfunction. Loss of motion of the scapula in the different planes can affect a thrower's power and place a strain on the posterior shoulder capsule [14, 29].

2.3.3 The "Deltoid–Pectoralis" Complex

Morphological classification of muscular mass of the human body by the first anatomists was based on purely topographical, even aesthetic, criteria. The potential function of the different muscle bundles has not always been taken into account. This is particularly the case of the clavicular heads of the deltoid and pectoralis major muscles.

The deltoid muscle is a large, thick, triangular muscle that superficially envelops the shoulder joint. The deltoid muscle is divided into three anatomical parts. The anterior deltoid originates from the lateral third of the clavicle. The middle deltoid originates from the acromion and the posterior deltoid originates from the scapular spine. The muscle fibers of the deltoid converge into a V-shaped pattern, inserting on the humeral shaft.

The pectoralis major muscle is a large muscle of the anterior chest wall. The muscle is divided into a clavicular head, inserted on the medial portion of the clavicle, and a sternal head, inserted on the sternum and the adjacent sterno-costal cartilages. From its broad medial insertions, muscular fibers transform into a tendon inserted on the humeral diaphysis.

From a functional point of view, the clavicular head of the pectoralis major muscle and the anterior deltoid are very similar muscles. They act as arm flexors. The anterior deltoid is only slightly involved in abduction, the major function of the deltoid muscle. The clavicular head of the pectoralis major muscle is not really involved in adduction of the upper limb, the main function of the pectoralis major muscle.

These two heads are moreover very similar in structure, which clearly differs from that of the muscles to which they are attached. The deltoid muscle has a complex anatomical structure with intramuscular tendons which should be taken into account when assessing the function of the muscle. The structure of the clavicular portion of the deltoid muscle is however different and more simple than that of other segments of the muscle [26, 41]. Similarly, the intramuscular structure of the pectoralis major muscle is complex. Whereas the sternal head is divided into six or seven segments, the clavicular head is compact and unsegmented [15]. This more simple structure is similar to that of the anterior portion of the deltoid muscle.

Furthermore, the tendons corresponding to these two muscular heads are very close to one another. Concerning the deltoid muscle, the tendons of the three different parts (anterior, middle and posterior) insert individually into the humeral shaft and form three discrete lines [39, 41]. The anterior insertion line corresponding to the clavicular portion of the muscle is well individualized and separated from the lines of the middle and posterior parts that are closer to one another and far from the anterior line. Organization of the tendon of the pectoralis major muscle is also complex. Medially, the tendon consists of two distinct layers, anterior and posterior, that are separated by fat tissue. Laterally, the tendon fuses prior to its insertion at the lateral lip of the intertubercular sulcus of the humerus. The two layers are broad and flat and are continuous inferiorly. The anterior layer courses in an inferolateral direction, whereas the posterior layer courses superolaterally. The anterior layer corresponds to the clavicular head, even if in some cases bundles of the sternal head project onto the anterior layer. At the musculotendinous junction, the clavicular head overlaps all the sternal segments deep to it [15].

There is a marked anatomic proximity between the tendon of the anterior part of the deltoid muscle and that of the clavicular head of the pectoralis major muscle [23]. Proximity and these distal interconnections further enhance the common function of both muscles and focus their muscle power.

This anatomical knowledge of these two muscle bundles is important to consider in clinical practice. It is fundamental for surgical approaches and repair of the

deltopectoral region. This information is essential in rehabilitation of the shoulder, especially in athletes.

2.4 Organization of Tendons

Muscles produce force that is transmitted via connective tissue networks and tendons to the skeletal system in order to propel movements. This sometimes complex intramuscular network organization has been previously discussed (cf. muscular structure).

The tendon is the fibrous structure necessary for the muscle to insert on the bone. The surface of tendinous insertion causes bone relief, defined as ridges, tuberosities or even processes. There are tendons of origin and termination. Tendons can be flat and thin, thus defined as an aponeurosis. Within the same muscle, a tendon can develop gradually into an aponeurosis.

In some cases, muscle fibers are inserted directly onto a bone structure without an interposing tendon. Moreover, some muscular fibers are inserted directly on structures other than bone, such as on a septum or fascia. A fascia is defined as connective tissue sheets enveloping muscles or muscle groups and which do not contain tendon fibers of muscle origin.

The myotendinous junction corresponds to the macroscopic insertion area of muscle fibers on a tendon or aponeurosis. It depends on the configuration and structure of the muscle. For the same muscle, it may be subjected to variations (Fig. 2.5).

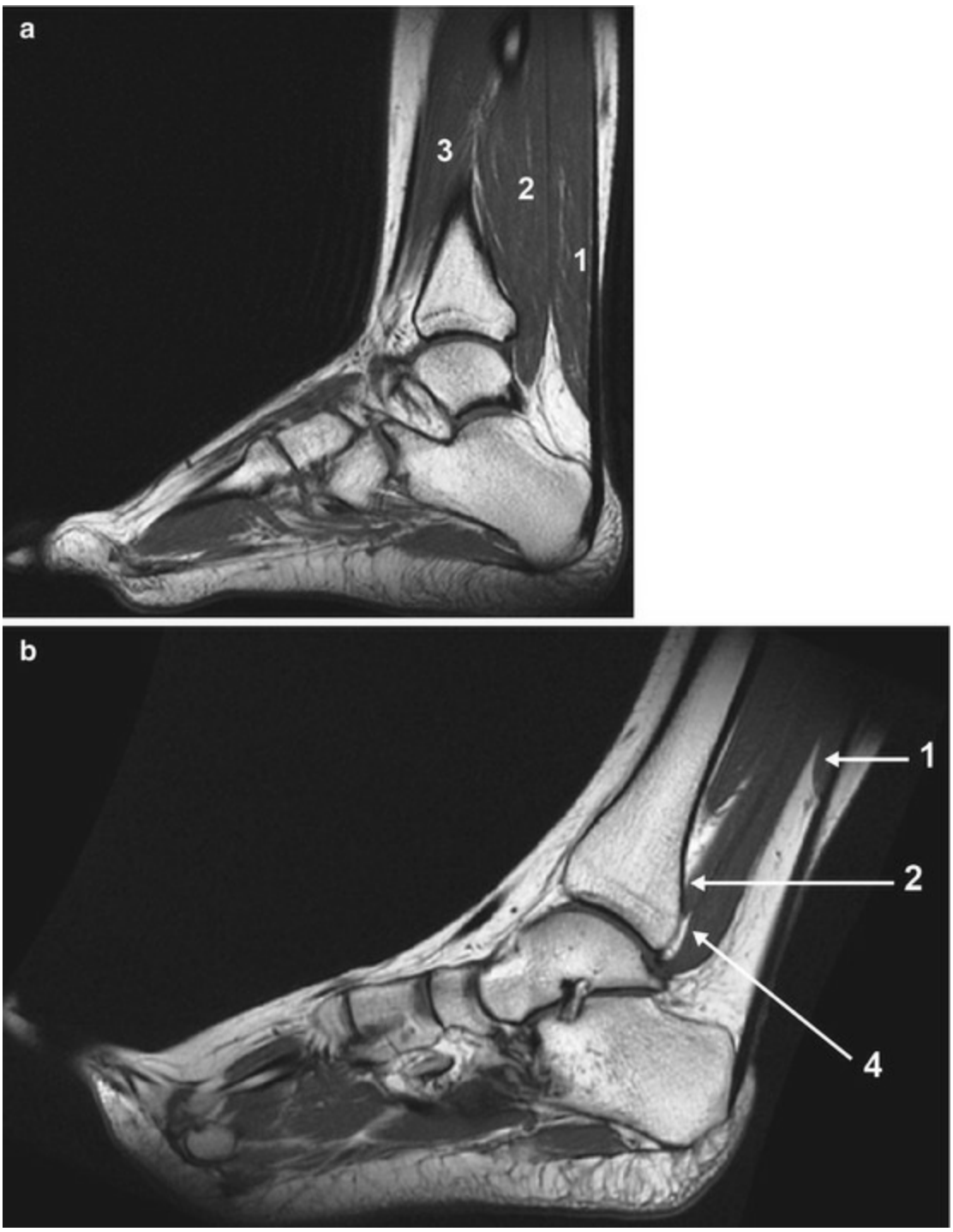

Fig. 2.5 Variations of levels of the soleus myotendinous junction. (**a**) Low myotendinous junction. (**b**) High myotendinous junction. (*1*) Soleus muscle – (*2*) Flexor digitorum longus muscle – (*3*) Extensor digitorum longus muscle – (*4*) Flexor hallucis longus muscle

Tendon structures may sometimes have a complex organization that is important to consider in pathology, especially in athletes. To illustrate these concepts of organization, we will present the anatomy of the proximal hamstring muscle complex and the femoral portion of the iliopsoas muscle.

2.4.1 The Proximal Hamstring Muscle Complex

The hamstring refers to the three muscles located in the posterior compartment of the thigh: semimembranosus, semitendinosus and biceps femoris (long and short heads). These muscles are bi-articulate, extending the hip joint and flexing the knee joint.

The ischial tuberosity is the site of origin of the hamstring muscles, except for the short head of the biceps femoris, which arises from the middle third of the linea aspera and the lateral supracondylar ridge of the femur. The proximal region of the hamstring muscles is characterized by complex architecture with overlapping tendons and inter-relations between muscles. The raphe, a tendinous inscription dividing the semitendinosus muscle into two distinct parts, belongs to this proximal complex. The semitendinosus muscle is considered to be a digastric muscle with opposite bellies due to this raphe.

The areas of origin of the semitendinosus, semimembranosus and long head of the biceps femoris muscles are clearly divided into two parts on the ischial tuberosity. The anteromedial part is occupied by semitendinosus and the long head of the biceps femoris muscle which are highly adjoined. The posteromedial part is occupied by the origin of semimembranosus. These two parts are divided by a vertical ridge delimiting the anteromedial and posterolateral facets. A small portion of semitendinosus is inserted on a small inferior facet of the ischial tuberosity. The adductor magnus muscle has also a tendinous slip originating from the infero- and anteromedial aspect of the ischial tuberosity, but is usually not considered a component of the hamstring muscle complex [4, 52].

The close relationships between the long head of the biceps femoris muscle and the semitendinosus muscle have led to the description of a common head and a common tendon. Biceps femoris and semitendinosus usually separate from their common tendon 9 cm away from the ischial tuberosity. Most of the common head is constituted by semitendinosus. In this common tendon, the long head of the biceps femoris muscle consists of the tendinous part and semitendinosus mainly consists of the muscular part. The tendinous portions of the proximal parts of these muscles can be delimited. The long head of the biceps femoris muscle is composed of a thick and long tendon and semitendinosus contains a thin and short tendon. The tendon of the long head of the biceps femoris muscle is widely connected to the sacrotuberous ligament [4, 43, 52].

The semimembranosus muscle originates just laterally to the common insertion of the semitendinosus and biceps femoris muscles, posterior to the origin of the quadratus femoris muscle. The most proximal part of semimembranosus tendon is joined to the common tendon of semitendinosus and the long head of the biceps femoris and separates around 2.5 cm away from the ischial tuberosity [4, 31, 43].

Knowledge of the anatomy of this tendinous complex is important in understanding pathologies of this region. Hamstring muscle strain is one of the most common injuries

in sports medicine. The long head of the biceps femoris muscle is the most commonly injured muscle of the hamstring. The most vulnerable part of the tendon-muscle-bone unit is the musculotendinous junction. Strain on the musculotendinous junction is usually caused by eccentric load during either acceleration or deceleration. Distinction can be made between two mechanisms of injury: hamstring injuries sustained during high speed running, usually affecting semitendinosus, and hamstring injuries sustained during stretching with a combination of extensive hip flexion and knee extension, usually located in semimembranosus. Most hamstring strains or tears can be treated conservatively, but proximal hamstring avulsions can cause significant disability and may need surgery. Surgical intervention must respect the myotendinous organization of the proximal part of the hamstrings (Fig. 2.1).

2.4.2 The Femoral Portion of the Iliopsoas Muscle

The femoral portion of the iliopsoas muscle is a good example of the organization of a myotendinous complex.

The psoas major muscle is a long fusiform muscle, originating from the transverse processes of the lumbar vertebrae. It courses caudally to lie anterior to the capsula of the hip. The iliacus muscle is a flat triangular muscle that arises from the two thirds of the iliaca fossa and the inner lip of the iliac crest. The fibers converge to insert on the lateral side of the psoas major muscle. In some cases these two muscular fasciae are accompanied by the psoas minor muscle. This inconstant muscle originates on the vertebral bodies of the twelfth thoracic vertebra and the first lumbar vertebra. Its thin muscular body orients to the surface of the major psoas muscle and finishes as a tendon which joins the inguinal ligament.

The femoral portion of the iliopsoas muscle forms a musculotendinous complex with a variable and elaborate organization. This musculotendinous complex usually consists of a main tendon, arising from the psoas muscle, an accessory tendon arising from the iliacus muscle, proper muscular fibers belonging to the iliacus muscle and an iliopectineal bursa [51].

The main tendon originates in the muscular body of the psoas major muscle above the inguinal ligament. Psoas major fibers insert on the anterior surface of the main tendon. Initially in a frontal plane, it exhibits a characteristic rotation which progressively transforms its ventral surface into a medial surface. It spreads out to cover the lesser trochanter and fixes directly onto the lesser trochanter without interposition of a gliding apparatus. The accessory tendon has a more lateral position than the main tendon. The most medial iliac muscular fibers insert on its anterior surface and then fuse with the main tendon. The fusion between both tendons is progressive, leaving a 6–8 cm long crevice on the posterior surface of the tendon formed by the union of the main and accessory tendons. The iliopectineal bursa has 6 cm high and 3 cm wide

cavity. It extends from the iliopectineal eminence to the lower extremity of the femoral head. On the upper part, the bursa is divided into a medial compartment for the main tendon and a lateral compartment for the accessory tendon. Proper muscular fibers of the iliac muscles join this musculotendinous complex. The most lateral fibers, originating particularly from the ventral portion of the iliac crest, terminate with no tendon on the anterior surface of the lesser trochanter and in the infratrochanteric region. The most inferior muscular fibers of the iliac muscle join the main tendon, passing around its ventromedial part.

An ilio-infratrochanteric muscular bundle, sometimes referred to as the iliacus minor muscle, is present in a deeper position under the iliopsoas tendon. Its fibers arise from interspinous incisor and anterior inferior iliac spine above the insertion of the rectus femoris muscle. The bundle runs along the anterolateral edge of the iliacus muscle and inserts without a tendon onto the anterior surface of the lesser trochanter of the femur and on the infratrochanteric area [51].

Description of these tendons is difficult due to terminological confusion. Indeed the terms “iliopsoas tendon” and “psoas tendon” have been used interchangeably in the literature. In some cases, the psoas tendon splits partially into two bundles [11, 49, 51]. A recent anatomic study confirms that two or more distinct tendons are not rare [36].

This musculotendinous complex is visible on ultrasound. The different components may be individualized. Only the most inferior muscular fibers of the iliac muscle, that join the psoas tendon and pass around its ventromedial part, are the most difficult to visualize. This discrepancy may be explained by poor demonstration of the interface between the inferior muscular fibers of the iliac muscle and the psoas muscle [16].

The femoral portion of the iliopsoas muscle is implicated as the cause of internal snapping hip syndrome. Hip snapping is caused by a tendon catching on the hip. It is felt and heard by the patient and in some cases, especially in athletes, can become painful. Hip snapping has special importance in sports that demand repeated elevations of the leg over the horizontal line in abduction. Lateral snapping, the most frequent, corresponds to the sudden slip of the iliotibial band over the greater trochanter. Internal hip snapping, less frequent, is the consequence of the sudden slip of the iliopsoas tendon over the iliopectineal eminence on the anterior edge of the pelvis. The mechanism of the snapping of the iliopsoas tendon is still debated. A complex anatomic relationship exists at the musculotendinous junction between the iliopsoas tendon and the anterior structures of the hip joint. Dynamic ultrasound studies help to understand this mechanism. Snapping may be caused by the iliopsoas tendon suddenly slipping around the iliac muscle, abruptly connecting with the pubic bone and producing an audible snap. The potential role of a bifid tendon of the psoas muscle has also been debated. In this case the bifid tendon heads would slip over one another [11] (Fig. 2.6).

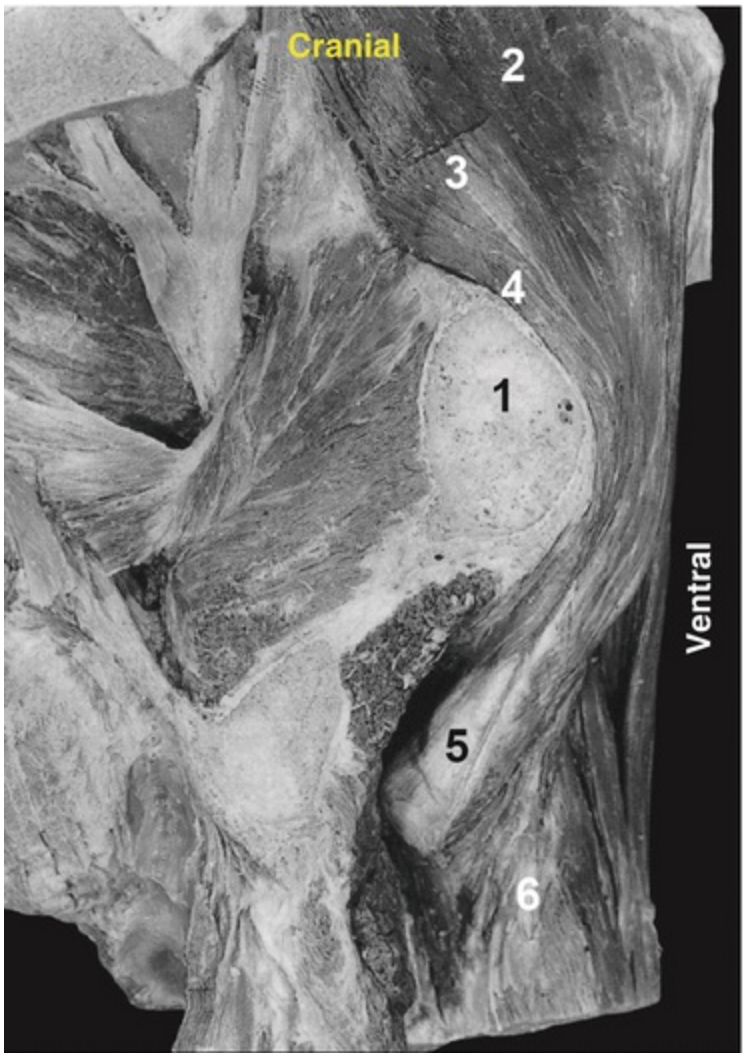

Fig. 2.6 Endopelvic view of an anatomic sagittal cut of the pelvis. The specific course of the femoral portion of the iliopsoas muscle is visible. (*1*) Iliopectineal eminence – (*2*) Iliacus muscle – (*3*) Psoas major muscle – (*4*) Proper muscular fibers of the iliacus muscle – (*5*) Main tendon of the iliopsoas muscle – (*6*) Ilio-infratrochanteric muscular bundle

2.5 Muscular Variations

Variations are numerous in human anatomy and muscles are no exception to this rule. The majority of these variant muscles are found incidentally during imaging explorations or surgery. Nevertheless, knowledge of muscle variations is important because they are potentially symptomatic, particularly in athletes. They may be caused by unusual truncal nerve compression and can also be expressed by palpable peri-articular swelling.

The advent of MRI has led to renewed interest in these variant muscles which also pose problems for radiologists in imaging interpretation or for the surgeon exploring a site of limb nerve compression.

To illustrate these variations, it is important to consider some significant examples

of muscle variations common in athletes. We will discuss the reversed palmaris longus muscle and the supernumerary bundles of the flexor pollicis longus muscle, which can compress the nerve branches of the forearm muscle, and muscle variations in the ankle region, which frequently present diagnosis problems.

2.5.1 Reversed Palmaris Longus Muscle

The palmaris longus muscle is a superficial flexor muscle of the forearm innervated by the median nerve. The standard proximal origin of the palmaris longus muscle is the common flexor tendon at the medial epicondyle of the humerus. This common tendon is shared by the flexor digitorum superficialis, the pronator teres, the flexor carpi radialis and the flexor carpi ulnaris muscles. The palmaris longus muscle lies in the superficial muscular group on the anterior forearm, just under the skin and the fascia of the forearm, between the flexor carpi radialis muscle laterally and the flexor carpi ulnaris muscle medially. Fibers normally extend from the distal tendon to the flexor retinaculum and, when these flatten, are incorporated into the palmar aponeurosis.

It has been long known that the palmaris longus muscle has many variations. These variations, well-described in large anatomic series, include full agenesis, variations in the location of the belly portion, duplication, triplication or abnormal insertions of the distal tendon. The most common is agenesis of the muscle, occurring in about 15 % of cases [38].

Distal muscle abnormalities of this muscle include the reversed palmaris longus muscle, where a muscular belly replaces the distal tendon [32]. The proximal part of the muscle is tendinous (Fig. 2.7). In some very rare cases, the muscle may have proximal and distal bellies, forming a digastric muscle.

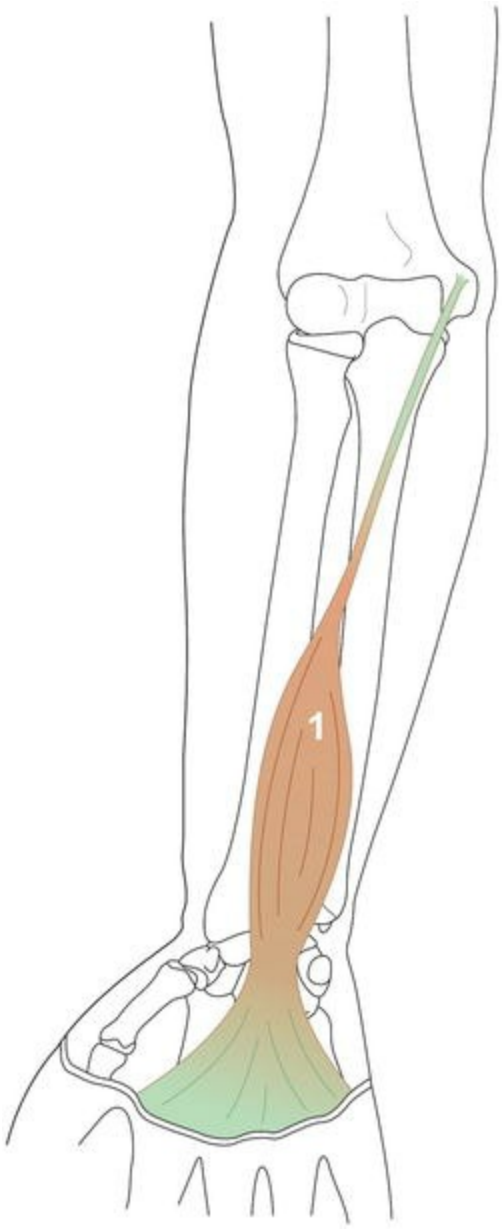

Fig. 2.7 A reversed palmaris longus muscle. A muscular belly (*1*) replaces the distal tendon

Because of the close anatomic relationship to the median nerve in the distal forearm, the reversed palmaris longus muscle can compress the median nerve and cause carpal tunnel-like syndrome symptoms. This variant distal belly increases in volume during exercise and can cause local pressure on the median nerve. This is particularly frequent in athletes using their wrist flexors intensively and repeatedly (mountain biking, racquet sports, etc.). Neurological symptomatology is essentially sensitive and frequently paroxysmal appearing during or after exercise involving the flexor muscles [10].

Clinical examination may help positive diagnosis by showing the existence of swelling in the palmar area of the wrist. Electroneuromyographic examination is often normal because is performed when the median nerve is not compressed. Ultrasound and/or MRI examinations can confirm this variant belly as close to the median nerve

(Fig. 2.8). Surgical excision of this distal muscle belly is curative [17, 30].

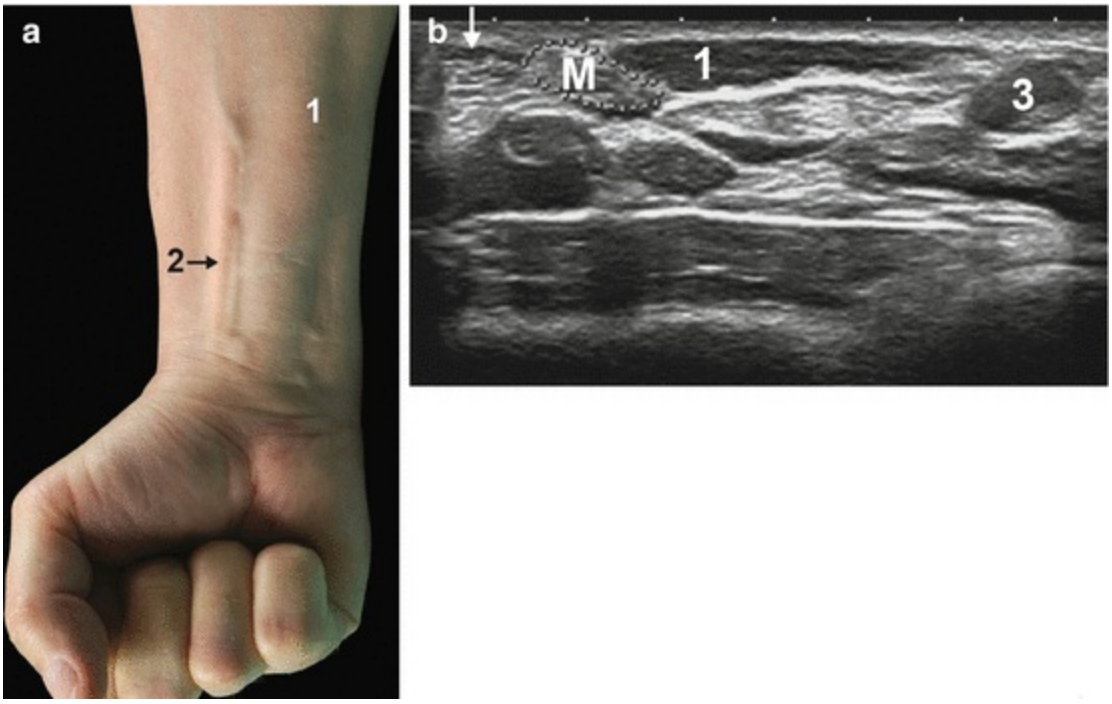

Fig. 2.8. (**a**) Swelling in the palmar area of the wrist corresponding to a reversed palmaris longus muscle (*1*) next to the tendon of the flexor radialis carpi muscle (*2*). (**b**) Transverse ultrasound image of the distal ventral forearm shows a reversed palmaris longus muscle (*1*) close to the median nerve (*M*). The variant belly (*1*) is located between the distal tendon of the flexor radialis carpi muscle (*arrow*) and the radial artery (*3*)

Compression of the ulnar nerve by a reversed palmaris longus muscle is rarer and occurs rather in cases of two or three headed reversed palmaris longus muscle [1].

2.5.2 Aberrant Belly of the Flexor Pollicis Longus Muscle (Gantzer's Muscle)

Two different aberrant bellies in the deep flexor region of the forearm have been described. These variants heads, inserting into either the flexor digitorum superficialis or the flexor pollicis longus, were named the Gantzer's muscle, after Gantzer who is recognized for having described it in 1813.

These two accessory heads can be joined, but the presence of only one accessory head of the deep flexor muscle of the fingers is infrequent and cannot anatomically compress nerve structures. The presence of an additional head of the long flexor muscle of the thumb is however much more common, seen in 46–66 % of cases, and may be involved in the compression of the anterior interosseous nerve. This accessory muscle is often the only one to be referred to as Gantzer's muscle [21, 34, 44].

The flexor pollicis longus muscle inserts on the upper third of the radial diaphysis and the adjacent interosseous membrane. Its forearm tendon begins at the junction of the

middle and proximal third of the forearm, continues to the wrist and ends on the distal phalanx of the thumb. At the forearm, the muscle is located in the same plane as the flexor digitorum profundus muscle. When present, the accessory flexor pollicis longus muscle inserts into the medial epicondyle of the humerus by the common tendon of the flexor muscles on the coronoid process of the ulna and the intermuscular fascia between the superficial and deep flexor muscles of the fingers. It usually ends on the ulnar side of the long flexor muscle of the thumb [21, 34, 44].

The presence of variant bellies of the flexor pollicis longus muscles in the anterior compartment of the forearm may result in painful compartment syndrome due to decreased availability of space. Furthermore, the supernumerary muscle passes fairly consistently between the median and anterior interosseous nerves and may compress the latter [48] (Fig. 2.9). Anterior interosseous nerve compression by Gantzer's muscle has been frequently observed and surgically confirmed [8, 50].

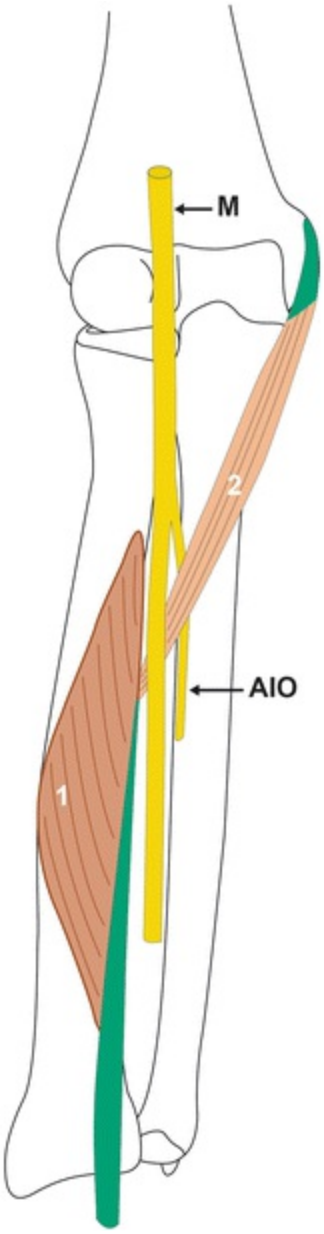

Fig. 2.9 Schematic drawing of a variant flexor pollicis longus muscle. An abnormal muscle (*2*) is inserted on the medial epicondyle and joins the flexor pollicis longus muscle (*1*). The belly passes through the median nerve (*M*) and the anterior interosseous nerve (*AIO*)

2.5.3 Muscular Variations of the Ankle Region

Many muscle variations of the periarticular muscles of the ankle have been described. Some are common, such as the presence of the fibularis tertius muscle, others are rarer, such as the presence of a fibularis quartus muscle, accessory soleus muscle or flexor accessorius digitorum longus. These variations should be considered in the differential diagnosis of chronic ankle pain and sometimes in the diagnosis of tarsal tunnel syndrome, especially in athletes. MRI is the method of choice in identifying these variant muscles.

2.5.3.1 Fibularis Tertius Muscle

The presence of a fibularis tertius muscle is a very common variation. The muscle is present in 75–90 % of cases. It is a relatively small muscle found in the anterior compartment of the leg, also comprising the tibialis anterior, extensor hallucis longus and extensor digitorum longus muscles.

The fibularis tertius muscle can sometimes originate with the extensor digitorum longus muscle. It originates from the anterior surface of the distal shaft of the fibula and interosseous membrane. The tendon of the fibularis tertius muscle passes anterior to the ankle joint under the superior extensor retinaculum extensor. It has a postero-lateral position relative to the tendon of the extensor digitorum longus muscle and inserts at the base of the fourth and fifth metatarsal [22, 40] (Fig. 2.10).

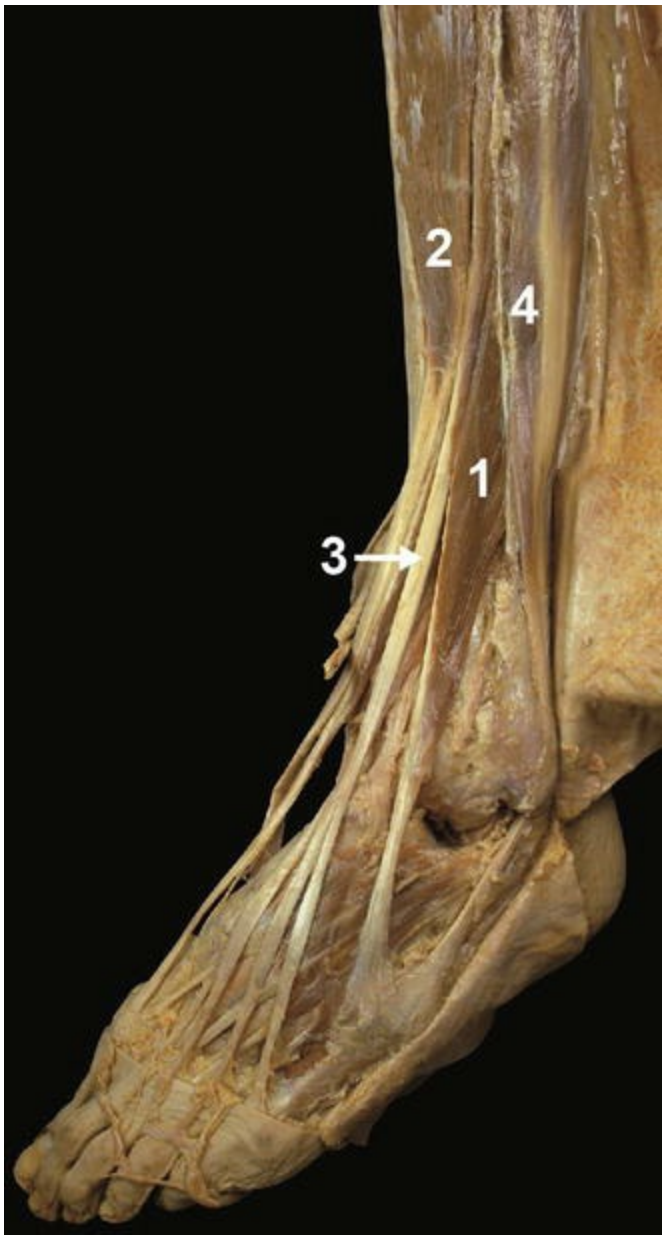

Fig. 2.10 Anatomic dissection of a fibularis tertius muscle (*1*) located between, medially, the tibialis anterior muscle (*2*) and the extensor digitorum longus muscle (*3*) and laterally, the fibularis longus muscle (*4*)

The fibularis tertius muscle is rarely pathogenic, but can sometimes cause snapping and ankle pain [42]. It can also lead to misinterpretation of MRIs of the ankle.

2.5.3.2 Fibularis Quartus Muscle

The fibularis quartus muscle is the only accessory muscle that passes the lateral compartment of the ankle. Its reported incidence varies from 6 to 21.7 %. The fibularis quartus muscle mostly originates from the peroneus brevis muscle, distal third of the fibula or the peroneus longus muscle. The muscle is located posteriorly to the peroneus brevis and longus tendons. Distally, it inserts most commonly at the lateral area of the calcaneus onto the retrotrochlear eminentia, which can be hypertrophic in the presence of this variant muscle. Its distal insertion shows variety including peroneal trochlea of the calcaneus, cuboid bone or fifth metatarsal base. As a result of these insertions, many different muscles that cause ambiguous nomenclature in literature have been described: peroneus calcaneus externus, peroneus medius, etc. All these accessory muscles should be collected under a single title, fibularis quartus muscle, to prevent terminological confusion [2, 5, 19, 57].

The fibularis quartus muscle is supposed to be involved in the lateral stabilization of the foot and can be associated with many pathologies of the lateral ankle compartment. As it will cause stenosis in the lateral compartment, it may cause degeneration or tear in the peroneus brevis tendon. In some cases, the fibularis quartus muscle may present bifurcated insertion around the peroneus brevis [5]. The possibility of a fibularis quartus muscle should be kept in mind in patients with post-traumatic ankle pain, instability or complaints of loss of strength. The fibularis quartus muscle is usually very visible on MRI.

2.5.3.3 Flexor Digitorum Accessorius Longus Muscle

The flexor digitorum accessorius longus muscle is the second most commonly occurring muscle anomaly of the ankle region, and the first in the medial compartment. Its incidence is around 4–12 %. The muscle may arise from any structure in the posterior compartment of the leg, especially on the flexor hallucis longus muscle or the transverse intermuscular septum. In some cases, the level of origin of the flexor digitorum accessorius longus muscle is the tarsal tunnel (Fig. 2.11). Different types of this variant muscle have been described depending on its site of origin and its situation to the posterior tibial neurovascular bundle. The distal insertion site of the muscle is usually the medial border of medial head of quadratus plantae muscle [6, 19].

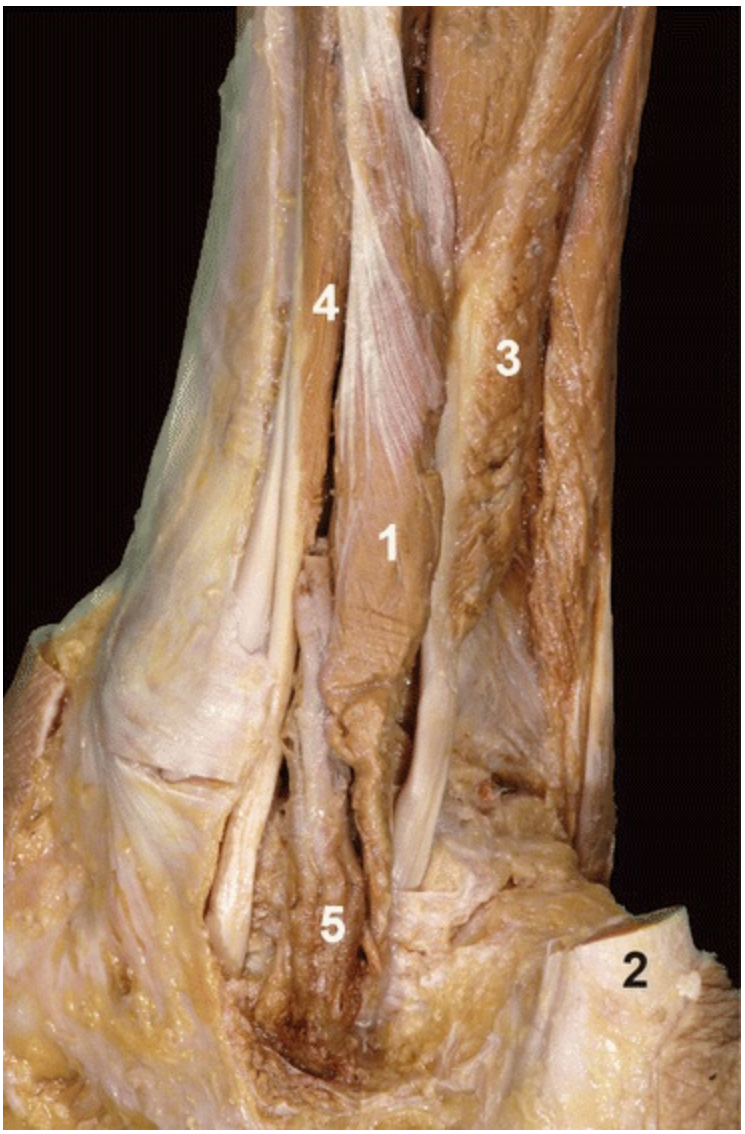

Fig. 2.11 Anatomic dissection of the medial aspect of the ankle shows a flexor digitorum accessorius longus muscle (*1*) close to the flexor hallucis longus muscle (*3*) and the flexor digitorum longus muscle (*4*). The calcaneal tendon has been removed (*2*)

The flexor digitorum accessorius longus muscle is considered as a possible cause of tarsal tunnel syndrome, compression of the tibial nerve within the tarsal tunnel that results in pain and sensory disturbances of the foot. The belly of the variant muscle within the tarsal canal can cause increased compression on the tibial nerve. As a result of repetitive irritation within the tarsal tunnel, the flexor digitorum accessorius longus muscle can also lead to flexor hallucis longus muscle tenosynovitis [6, 19].

2.5.3.4 Accessory Soleus Muscle

The accessory soleus muscle is an anomalous muscle of the medial compartment of the ankle. Its embryological origins may derive from an anomalous splitting of a soleus anlage from the tibial side of a common flexor mass. The prevalence of the accessory

soleus muscle in cadaveric specimens ranges from 0.7 to 5.5 % and is rarely bilateral. The most common form of insertion of the accessory soleus muscle is proximal attachment to the soleus muscle and distal attachment to the calcaneus via a separate tendon medial to the calcaneus tendon. Two other distal types of attachment may be observed: tendinous attachment to the calcaneus tendon and fleshy attachment to the calcaneus [9, 18].

The accessory soleus muscle is frequently symptomatic in athletes because of the increase in muscle mass induced by activity. It may lead to pain and swelling posteromedial to the medial malleolus [7]. This soft tissue prominence may be misdiagnosed as synovioma or lipoma. Differential diagnosis of the presence of an accessory soleus muscle is a low myotendinous junction. Ultrasound is useful in confirming the presence of a soleus accessorius muscle.

2.6 Conclusion

The descriptive and morphological anatomy of muscles is often presented in an instructive and simplified manner. This perspective is insufficient to grasp the complexity of the musculo-aponeurotic and tendinous architecture. Knowledge of the concept of neuromuscular compartments is essential to the understanding of muscular function and pathology. Certain muscular variations induced by this compartmentalization are likely to interfere with the vascular and nervous environment, especially during extreme physical exertion encountered by sportsmen.

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3. Muscular Responses During and Following Acute Physical Activity Under Heat Stress

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Abstract
Acute exercise provides a unique challenge to several regulatory systems. From a circulatory and metabolic perspective, exercise is associated with a rise in systemic blood flow, which allows for the adequate delivery of oxygen to the working musculature, as well as the dissipation of metabolically generated heat to the environment. Prolonged exercise or repeated contractions also lead to the development of fatigue, characterized by a reduction in the ability to generate force or power. This loss of strength has both peripheral and central mechanistic causes, which are influenced by increases in whole-body temperature. Hence, when exercise is performed in hot environmental conditions, the development of hyperthermia contributes to alter skeletal muscle performance. The aim of this chapter is to provide a brief overview of the circulatory and thermoregulatory responses associated with acute dynamic exercise, as well as the influence of heat stress on the development of skeletal muscle fatigue. Skeletal muscle damage will also be addressed to provide a wider perspective on what occurs within active muscles during and following acute exercise.

3.1 Introduction

It is well established that acute exercise provides a unique challenge to several regulatory systems. From a circulatory and metabolic perspective exercise is associated

with a rise in systemic blood flow, which allows for the adequate delivery of oxygen to the working musculature, as well as the dissipation of metabolically generated heat to the environment. Prolonged exercise or repeated contractions also lead to the development of fatigue, characterized by a reduction in the ability to generate force or power [22]. This loss of strength has both peripheral and central mechanistic causes, which are influenced by increases in whole-body temperature. Hence, when exercise is performed in hot environmental conditions, the development of hyperthermia contributes to alter skeletal muscle performance. This chapter aims to provide a brief overview of the circulatory and thermoregulatory responses associated with acute dynamic exercise, as well as the role of heat stress on the development of skeletal muscle fatigue. Skeletal muscle damage will also be addressed to provide a wider perspective on what occurs within active muscles during and following acute exercise.

3.2 Circulatory and Thermoregulatory Responses to Dynamic Exercise

3.2.1 Skeletal Muscle Blood Flow

Skeletal muscle blood flow is a key component to optimizing both brief and prolonged exercise performance. Accordingly, skeletal muscles possess the circulatory capacity to provide adequate blood supply for various exercise tasks. Part of this ability stems from the variety and composition of skeletal muscles fibers. For example, red muscle fibers are rich in myoglobin and oxidative enzymes, which gives them a reddish color. These fibers also contain three times the number of capillaries per fiber as white fibers, which are generally recognized as glycolytic [19].

At rest, skeletal muscle blood flow varies between 750 and 1000 ml.min^{-1}, or 2–4 ml.100 g^{-1} of muscle per minute. This constitutes about 15–20 % of cardiac output and an oxygen uptake of approximately 25 % of the resting metabolic rate [49]. However, oxygen uptake in individual muscles can increase 50-fold when transitioning from rest to maximal exercise. The working musculature can thus receive 80–85 % of maximal cardiac output during whole-body exercise at maximal aerobic capacity (VO_2max). In a 75 kg individual, skeletal muscles constitute about 30 kg (40–45 %) of overall body mass. With a VO_2max of 3500 ml.min^{-1} and a maximal cardiac output of 22,000 ml.min^{-1}, total (i.e. systemic) muscle blood flow in this individual could reach or exceed 18,000 ml.min^{-1} (60 ml.100 g^{-1}.min^{-1}) [49, 50]. However, during intense whole-body exercise such as running, only about 50 % of total muscle mass is strongly engaged. Thus, if 15 kg of muscle mass were uniformly engaged, blood flow to the active musculature would increase to 120 ml.100 g^{-1}.min^{-1}. In an individual with a VO_2max of

6300 ml.min^{-1} and an identical muscle mass, such as an elite endurance athlete, total muscle blood flow would be 33,600 ml.min^{-1} (i.e. approximately 224 ml.100 g^{-1}.min^{-1}). During cycling exercise, which requires less overall muscle mass (10 kg), muscle perfusion could theoretically increase to 336 ml.100 g^{-1}.min^{-1}.

The upper limit of muscle perfusion in well-trained cyclists has been reported to reach upward of 380 ml.100 g^{-1}.min^{-1} during isolated, single leg maximal knee exercise [6]. During such exercise the absolute increase in muscle blood flow can rise from 300 to 10,000 ml.min^{-1} in less than 10 s [52]. This represents a tremendous circulatory capacity and compliance in the muscle vasculature in response to vigorous exercise. It also reinforces the notion that muscle blood flow is intimately linked with the oxygen demand of the exercising muscles [3]. To meet this demand, several circulatory adjustments occur to ensure that perfusion pressure and vascular conductance requirements are met, especially during whole-body exercise. For example, blood pressure is not only maintained but increases in relation to exercise intensity. Compliant vascular regions (i.e. splanchnic and cutaneous) vasoconstrict to allow a redistribution of blood towards the active muscles, which initiate the muscle pump and concomitantly increase the volume of circulating blood (Table 3.1). Overall, muscle blood flow stabilizes within 30–90 s depending on exercise intensity, remaining relatively stable during extended periods of steady-state exercise [53].

Table 3.1 Estimated blood flow of the major vascular beds at rest and during exercise at VO_2max in temperate environmental conditions in a healthy and active human subject

Vascular region	Blood flow (ml.min^{-1})	
	Rest	VO_2max
Brain	750	850
Coronary	250	1000
Kidneys	1200	360
Muscle - Inactive	1000	850
Muscle - Active		21,840
Skin	500	300
Splanchnic	1500	350
Other	300	100
Total	**5500**	**25,000**

Adapted from Wade and Bishop [61] and Rowell [50]

3.2.2 Skeletal Muscle Heat Production

Active muscles are a major site of endogenous heat production and storage. Indeed,

exercise performed at 80–90 % VO_2max could increase core temperature by 1 °C every 5–8 min if heat was not dissipated to the environment. This is due to the low mechanical efficiency of humans, as approximately 70–75 % of the energy derived from metabolism during exercise is released as heat, rather than converted into movement [24]. However, the circulation of blood ensures temperature regulation, which is an essential physiological function that controls the exchange of heat between the different human body segments and the environment.

At the onset of exercise, heat production in active muscles increases sharply and markedly from a resting temperature of about 34 °C. During short intense efforts the production of heat increases until the termination of exercise. For example, during a 3 min maximal effort of the knee extensors, heat production is doubled, with 50 % of the increase occurring in the first minute [26]. In contrast, the rate of rise in muscle temperature is curtailed and production eventually plateaus during steady state dynamic exercise. Nevertheless, the absolute increase in skeletal muscle temperature when transitioning from rest to prolonged intense dynamic exercise can exceed 5 °C [30]. The increased heat production is linked to both anaerobic and aerobic energy pathways. The initial rise in temperature is associated with an anaerobic energy turnover, until a shift to aerobic metabolism occurs (i.e. glycogenolysis). Interestingly, exercise performed under heat stress has been shown to enhance glycogen utilization in the contracting musculature [17, 18]. However, the development of skeletal muscle fatigue during prolonged exercise in the heat appears to be unrelated to substrate availability [16].

Rather, exercise in the heat leads to a significant amount of blood being redirected towards the skin for the purpose of heat dissipation. Although this redistribution does not compromise muscle blood flow during submaximal exercise [35, 54], it does exacerbate the cardiovascular response [51]. As a result, VO_2max is reduced during protracted exercise in the heat, which leads to an increase in relative exercise intensity for any given workload [4, 39, 64]. Ultimately, the increase in intensity results in the early cessation of constant rate exercise, or the reduction of power output/running speed during self-paced efforts. At the level of the muscle, the combination of an accumulation of heat and prolonged contractile activity have been shown to compromise cellular and neuromuscular function, which are likely to impair performance [25, 41, 59].

3.3 Skeletal Muscle Fatigue and Heat Stress

A moderate increase in skeletal muscle temperature enhances brief explosive performance (e.g. sprinting and jumping) by improving metabolic function, nerve conduction and conformational changes associated with muscle contraction [2, 12, 20]. Muscle contractile function is also improved in a heated muscle, as demonstrated by increases in the rates of force development and relaxation (Fig. 3.1). Although these

increases benefit short-term power output and force development, they require faster motor neuron firing rates to maintain the fusion of force during prolonged efforts (e.g. maximal voluntary isometric contractions). Thus, a sustained contraction performed at a given absolute force with a heated muscle will result in a more rapid decline in force, compared with a cooler muscle, as firing rates decrease [12].

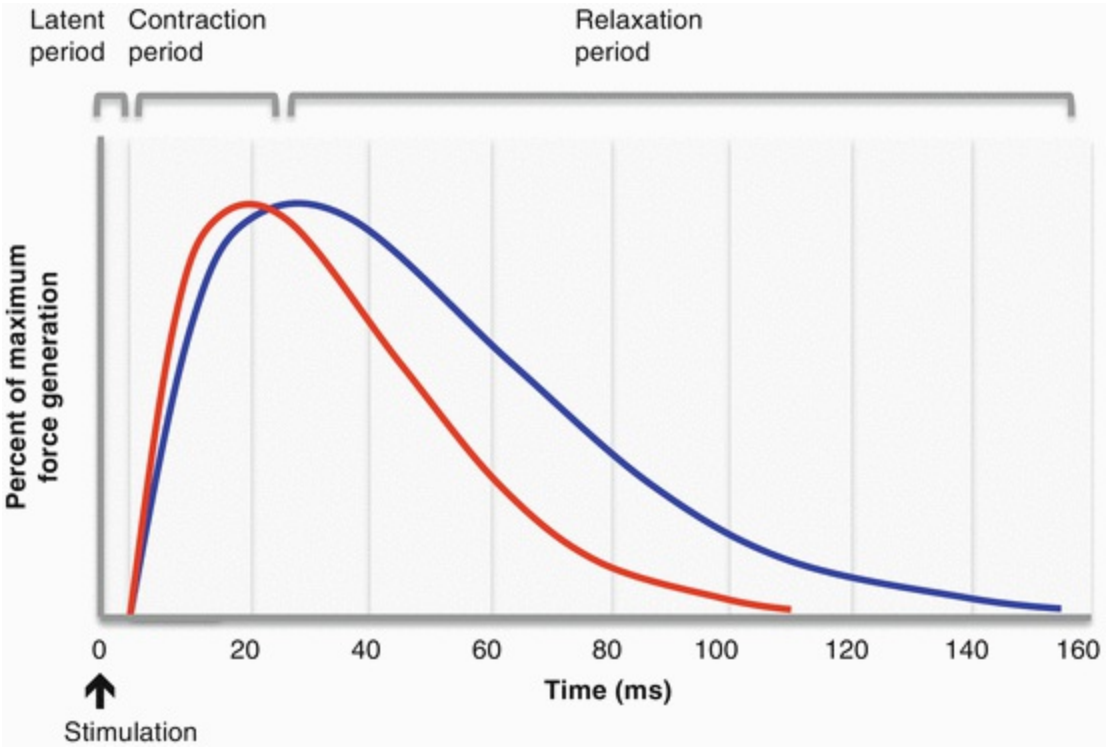

Fig. 3.1 Muscle contractile response to a single brief threshold stimulus (i.e. twitch) in normothermic (*blue*) and hyperthermic (*red*) skeletal muscle. A twitch has three phases: a latent period, a period of contraction, and a period of relaxation. From this response, peak twitch force, time to peak force, half-relaxation time, along with rates of force development and relaxation may be calculated to evaluate the changes in the contractile function induced by exercise or heat stress

Comparative studies of mammalian muscle contractions in vitro and performance in vivo have also indicated that muscle fatigue is temperature-sensitive. It appears that optimal muscle temperature for performance, the temperature at which less fatigue develops, is achieved around 30 °C [48, 58]. However, these results may not relate directly to exercising humans, as resting core and muscle temperature are typically 7 °C and 4 °C higher, respectively. Nevertheless, in humans performing whole-body dynamic exercise there is a dose-response relationship between temperature and performance [47]. This relationship is non-linear and dependent on central (i.e. core) and local (i.e. muscle) temperatures, whereby it is a rise in muscle temperature that improves power output [14] and a rise in core temperature that leads to impairment [47]. For example, mean power output during a repeated sprint protocol on a cycle ergometer was improved in hot ambient conditions with the attainment of a core temperature of ~38.1

°C, versus ~37.7 °C in temperate conditions [23]. Conversely, repeated sprinting ability was reduced when core reached ~39.5 °C in the heat, compared with ~38 °C in cool conditions [10].

Part of the loss in performance associated with the attainment of an elevated core temperature has been attributed to a hyperthermia-induced reduction in central neural drive to exercising muscles (i.e. central fatigue) [32, 37]. Indeed, several studies have shown that passive hyperthermia is associated with a loss of strength, concomitant with reductions in voluntary activation [32, 40, 41, 45, 60]. However, the role of exercise-induced hyperthermia in exacerbating central fatigue remains unclear. It was previously shown that voluntary activation and force production progressively decrease during a sustained (120 s) maximal voluntary isometric contraction following exhaustive cycling in the heat, but remain elevated after exercise in cool conditions [37]. Conversely, more recent studies have reported that voluntary activation and strength losses are equivalent during brief (5 s) and sustained (20 s) contractions conducted after self-paced and incremental exercise in hot and cool conditions [42, 46]. Part of the discrepancy may relate to the length of contraction. After 30 s of maximal isometric effort pain becomes increasingly severe, altering the perception of sensations originating from the active musculature, which leads to uncertainty as to the level of force being exerted [5]. Moreover, a lack of motivation during prolonged contractions might reduce central activation to the appropriate motor neurons, resulting in the loss of force [13].

During exercise in the heat, fatigue is also mediated by adjustments occurring beyond the neuromuscular junction (i.e. peripheral fatigue) [40–42, 45]. These peripheral adjustments contribute to the preferential development of fatigue at low frequencies of stimulation (i.e. low-frequency fatigue) [11]. Consequently, force production in hot fatigued muscles decreases at lower stimulation frequencies, shifting the force-frequency relationship toward higher frequencies (Fig. 3.2) [43]. This phenomenon has been attributed to a failure in excitation–contraction coupling, stemming from a reduction in sarcoplasmic reticulum Ca^{2+} release [11, 27, 63], and/or a conduction block of the action potential [2, 20]. It has also been linked to a reactive oxygen species mediated decrease in myofibrillar Ca^{2+} sensitivity [7]. Interestingly, the influence of the failure in excitation–contraction coupling is negligible at high frequencies of stimulation, as the total number of action potentials increases. As a result, force production in hot fatigued muscles decreases at lower excitation frequencies, but remains elevated or only slightly decreases at higher frequencies.

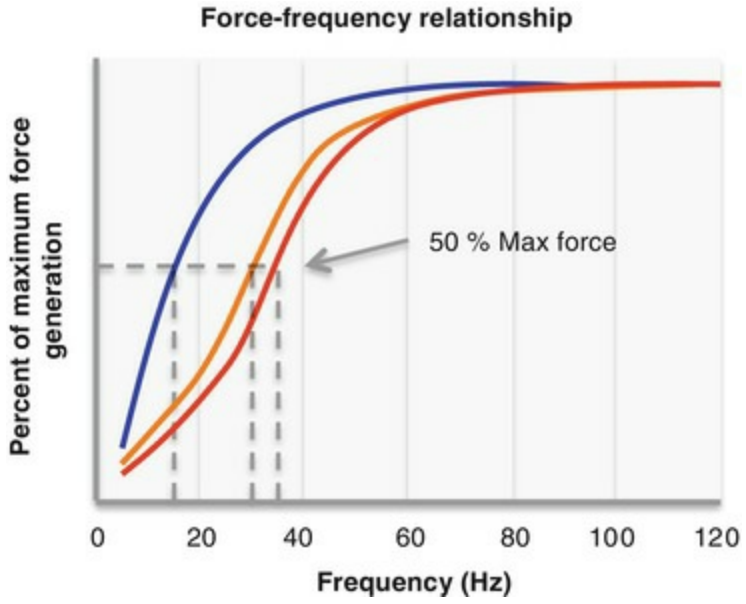

Fig. 3.2 Force production capacity relative to maximum force output in normothermic-unfatigued (*blue*), hyperthermic-unfatigued (*orange*) and hyperthermic-fatigued (*red*) skeletal muscle. A rightward displacement of the force-frequency relationship occurs at lower excitation frequencies due to the development of hyperthermia and low-frequency-fatigue. In hyperthermic-fatigued muscle, a greater excitation frequency is required to attain 50 % of maximal force generation, an effect that diminishes as frequency increases. Adapted from Périard et al. [43]

3.4 Exercise-Induced Muscle Damage

Muscle damage stemming from physical activity occurs following unfamiliar forms of exercise or extended periods of intense exercise. Interestingly, it appears that muscle damage and the recovery of functional performance are not aggravated following strenuous exercise under heat stress, compared with exercise undertaken in temperate conditions [36]. On the other hand, movements that involve eccentric muscle loading or lengthening contractions tend to exacerbate the damage and increase the risk of injury [8, 44]. Fast-twitch muscle fibers are especially susceptible to eccentric damage. This is likely due to structural differences between fiber phenotypes, as well as the greater level of generated tension and lower oxidative capacity of fast-twitch fibers [44]. Characteristically, exercise-induced muscle damage causes an immediate loss of strength, which can take a number of days to recover (Fig. 3.3). Swelling and soreness within the muscle develop thereafter (1 or 2 days), in conjunction with the release of soluble muscle proteins such as creatine kinase. Acute and chronic inflammatory responses are also associated with damaging exercise.

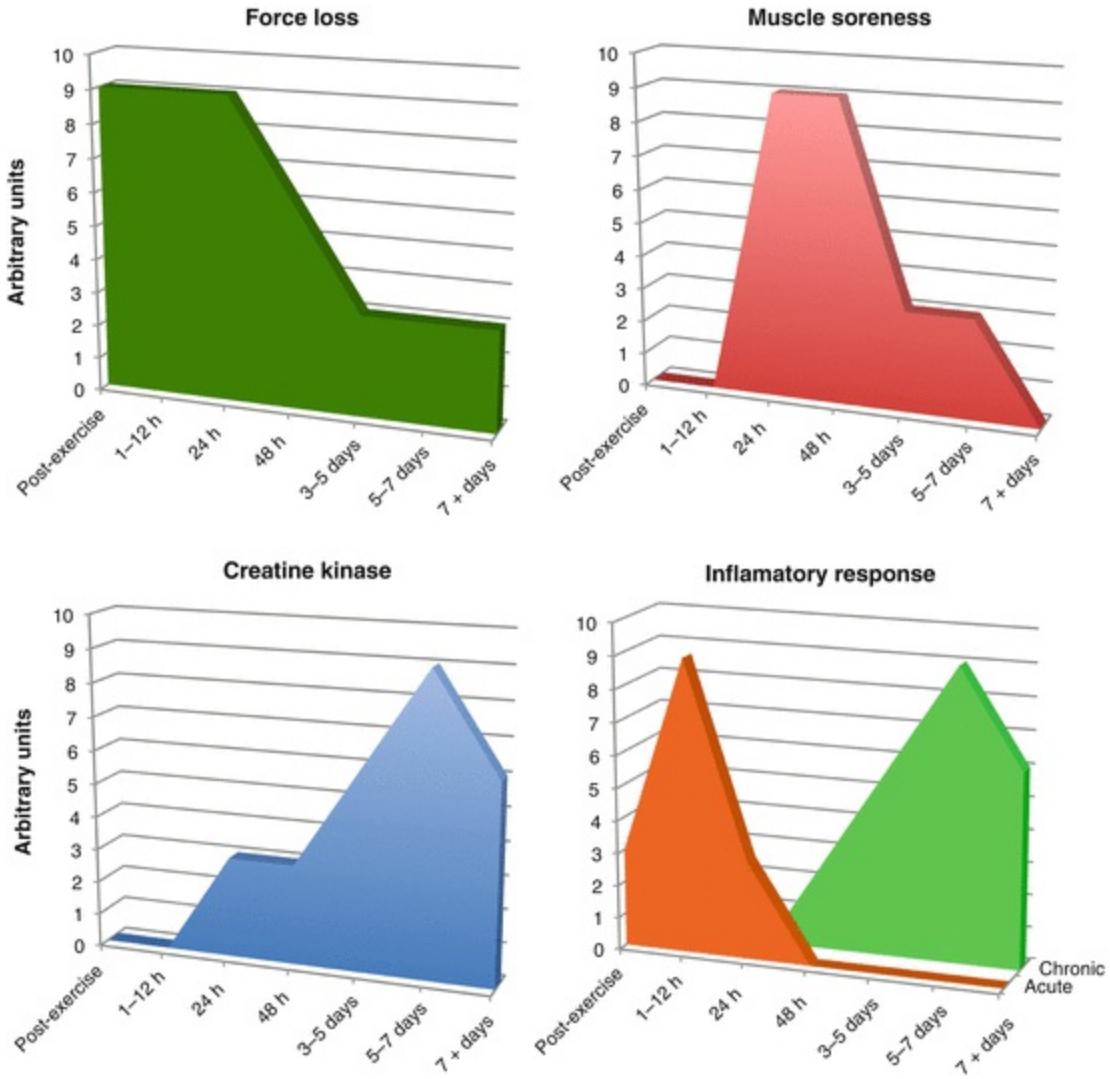

Fig. 3.3 Time course of adjustments in skeletal muscle damage following maximal eccentric exercise. Force loss, muscle soreness, creatine kinase activity, and inflammation (acute and chronic) are plotted on a relative 10-point scale (0 = no increase, 10 = large increase) of arbitrary units (AU) (Adapted from Clarkson and Hubal [8])

The precise mechanism that induces muscle damage is not fully understood. The initial trauma appears to originate from mechanical disruptions to actomyosin bonds within the muscle fibers. It is suggested that stretching the muscle beyond 140 % of its optimal length results in unfamiliar strain to the fibers [15]. This causes a linear deformation to certain sarcomeres within myofibrils and leads to membrane deformation, particularly in T-tubules, as well as disruption to calcium homeostasis. Consequently, damage occurs in response to the tearing of membranes and opening of stretch activated channels [1, 15]. Following the initial trauma, alterations in excitation-contraction coupling occur in conjunction with the inflammatory processes, which are characterized by infiltration of fluid and plasma proteins into the site of trauma [8]. The extent of the muscle damage is dose-dependent, as greater volumes of exercise produce greater trauma. Interestingly, the magnitude of trauma during a subsequent bout of

similar damaging exercise is reduced relative to the first bout [31]. This phenomenon is called the 'repeated bout effect' and can last for several months [56]. It is proposed that this adaptation stems from a strengthening of the connective tissue, a greater efficiency in motor unit recruitment and synchronization, a better distribution of workload amongst fibers, and a greater contribution of synergistic muscles during work.

In humans, the direct assessment of muscle damage is difficult. It requires the analysis of muscle biopsies, or the interpretation of magnetic resonance imaging (MRI). Although biopsies provide information about myofibrillar disturbances and Z-line streaming, the characterization is specific to a certain portion of muscle. As such, biopsies may not represent the state of the entire muscle [8]. Conversely, MRI assesses damage through the edemic response in the whole muscle by using signal intensity changes (i.e. T2 relaxation time). This technique is naturally better tolerated, as it is non-invasive. However, MRI interpretation remains problematic, as it may not be clear what the changes in the images indicate. As such, indirect markers of muscle damage are often used to evaluate the level of trauma associated with damaging exercise. These markers offer strong insight into the level of damage. The three indirect markers most commonly used are subjective soreness, blood proteins, and maximal voluntary force production [62].

3.4.1 Muscle Soreness

Following a bout of damaging exercise, muscle soreness does not appear for several hours, peaking at 24–48 h after exercise. This response is called delayed onset muscle soreness (DOMS). The level of soreness is associated with the level of muscle damage incurred, which is typically characterized by the type of activity performed. For example, non-weight bearing exercise such as cycling, which produces very little muscle damage, will result in minimal soreness. In contrast, maximal repeated eccentric contractions of the knee extensors will produce significant levels of muscle soreness. Part of the soreness is derived from swelling and pressure within the muscle. As fibers become enlarged, intramuscular pressure rises to cause pain [21]. The sensation of soreness also stems from the release of noxious chemicals (e.g. histamines, prostaglandins). These activate type III and IV muscle afferents, which trigger the sensation of pain [38]. However, muscle nociceptors can become sensitized to these noxious chemical mediators. In such circumstances, the mechanical deformation and swelling of the fibers act as the main physical stimulus for pain/soreness [28]. Interestingly, peak levels of soreness occur long before peak swelling.

3.4.2 Creatine Kinase and Myoglobin Activity

Hematologically, several muscle enzymes (e.g. lactate dehydrogenase, aspartate aminotransferase) and proteins (e.g. myoglobin, troponin) can be measured following

exercise to indirectly assess muscle damage. Due to its significant magnitude of increase relative to other proteins, creatine kinase (CK) in the bloodstream has been utilized most often as a blood marker. Depending on the mode and intensity of exercise, CK values can peak between 12–24 h (e.g. downhill running), or 4–6 days (e.g. maximal eccentric contractions) [9, 57]. A particular limitation with using CK however, is the large variation in response among individuals [8]. Notwithstanding, CK activity in the blood is generally associated with the level of damage assessed via MRI. It therefore represents an indirect relative marker of muscle damage. Similarly, when muscle fibers are damaged, myoglobin in the cells is released into the bloodstream (i.e. rhabdomyolysis). The presence of myoglobin in urine and serum is indicative of skeletal muscle trauma, inflammation and ischemia. In extreme circumstances it may indicate a heart attack, or malignant hyperthermia. As such, myoglobinuria represents a useful marker of muscle damage during exercise [36], especially in hot environmental conditions since hyperthermia may aggravate rhabdomyolysis [33].

3.4.3 Muscle Strength

The sustained loss of force production capacity following exercise, especially eccentric exercise, is considered to be one of the most valid and reliable markers of muscle damage [62]. The loss of force associated with intense/maximal eccentric exercise can lead to decrements in the ability to produce force between 50 and 65 % of pre-exercise baseline/control values. Recovery from such damage can take over a week [34, 55]. In contrast, strength losses of 10–30 % have been observed in less severe eccentric protocols causing minimal damage. The time course of recovery following such protocols is reduced to 24 h. During concentric exercise, although the loss of force is similar (10–30 %), recovery is much shorter and occurs within hours following the cessation of exercise [29].

3.5 Conclusion

Skeletal muscles undergo several adjustments when transitioning from rest to exercise. The magnitude of these adjustments relates to the intensity of exercise, the muscle group involved, and the composition of the muscle fibers within that musculature. The initiation of exercise and recruitment of skeletal muscles causes blood flow and heat production to rise locally. As exercise progresses, circulatory adjustments ensure adequate blood is supplied to the working musculature and metabolic heat is dissipated to the environment. Depending on exercise mode, intensity and duration, fatigue develops to various extents. When exercise is performed in hot ambient conditions and whole-body temperature increases, skeletal muscle fatigue develops in response to alterations occurring both centrally and peripherally. These alterations involve a

reduction in central neural drive, as well as disturbances in cellular and neuromuscular function. When exercise is performed with predominantly eccentric movements, skeletal muscle damage is likely to occur. However, the recovery process may be curtailed when damaging bouts of exercise are repeated. Understanding the interaction of these processes, from fatigue to damage, and the role of heat stress on performance, may allow athletes and coaches to optimize training and minimize skeletal muscle injuries.

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4. Epidemiology and Clinical Features of Muscle Injuries

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Abstract
Muscle injuries are extremely frequent in professional and amateur sports. Muscle strains, particularly of the hamstrings, are the most frequent injuries in football (soccer) and Australian football. Muscle injuries can be classified in relation to the time of absence into minor, moderate or SEVERE. One of the main problems in muscle injuries is the recurrence rate and this is especially common in the hamstrings group. The clinical presentation of muscle injuries is highly variable and depends on many factors related the mechanism, intensity and type of trauma. Understanding the injury as well as its cause is crucial for the development of specific treatment and prevention strategies.

4.1 Introduction

Muscle injuries are extremely frequent in professional and amateur sports; their clinical presentation varies widely and a greater knowledge of the epidemiology, clinical

features and classification will aid health workers in choosing the best treatment for each athlete.

4.2 Epidemiology

Muscle strains are the most frequent injuries in football (soccer) and Australian football [1–4], where they constitute approximately a third of all injuries [5–7] and are responsible for more than a quarter of the total absences caused by injuries [5]. Players average 0.6 muscle injuries per season [5] while clubs average five hamstring strains per season [8], with 90 days and 15 matches missed per club per season [8]. This works out to six new hamstring strains per club per season [3, 4]. Quadriceps injuries may cause an even longer absence [5].

In American football the offensive players sustain most of the hamstring injuries (45.1 %) with the wide receivers suffer 12–20 % of the injuries [9, 10]; 81.5 % are noncontact injuries, with those mainly (71 %) occurring during sprinting. In hockey, the offensive players account for the majority of groin injuries [11]. Men have a higher incidence of hamstring injuries [12] and there is a higher rate of injuries during games than during practice [8, 12]. Quadriceps and adductor injuries are more common in the kicking leg [6, 13] and have a higher injury rate in the preseason [6] in football games and are less likely to occur in the first quarter of each half [5]. In American football the majority of muscle injuries occur during the 7-week pre-season [9] but no difference between game periods was found in hockey [11]. Table 4.1 shows the incidence of muscle injuries in different sports.

Table 4.1 Incidence of muscle injuries in several sports

Author/year	n	Sport	Incidence	Location	Time lost (days, average)
Arnason [1]	306	Football	8.4 inj/1000 mh 0.8 inj/1000 th	Hamstrings	
Bradley [14]	36	Football	9 inj/1000 hp	Hamstrings	
Brooks [15]	546	Rugby	5.6 inj/1000 mh 0.27 inj/1000 th	Hamstrings only	14
Carling [16]	46	Football	4.51 inj/1000 mh 0.94 inj/1000 th	Hamstrings	7.5
Ekstrand [5]	2299	Football	8.70 inj/1000 mh 1.37 inj/1000 th	Hamstrings	14.4 ± 18.5
Elliott [9]	–	American football	2.7 inj/1000 AE mh 0.47 inj/1000 AE th	Hamstrings only	13.2
Emery [11]	1292	Hockey	1.33 inj/1000 AE	Groin only	
Engebretsen [17]	508	Football	1.8 inj/1000 mh 0.3 inj/1000 th	Hamstrings only	

Feeley [10]	696	American football	4.07 inj/1000 AE mh 1.79 inj/1000 AE th	Hamstrings only	
Fuller [18][a]	626	Rugby	22.4 inj/1000 mh 1.5 inj/1000 th	Hamstrings	M = 19.5 T = 10.7

Legend: All subjects were male. *Location* most frequent site of muscle injury, *inj* injury/injuries, *mh* match hours played, *th* training hours played, *hp* hours played (match and training), *AE* athlete-exposures (one athlete participating in either one practice or one game). *M* matching injury *T* training injury

[a]Incidence by injuries/1000 player-hours

Of the hamstring complex, the biceps femoris is the most frequently injured [8, 19, 20], followed by the semimembranosus and then the semitendinosus [19] with the musculotendinous junction the most frequent site of injury [20]. There is no consensus on when a hamstring injury is more likely to happen, during the terminal swing phase as a consequence of the eccentric contraction [21] or during the early stance phase due to the high external joint moments [22].

Muscle injuries can be classified in relation to the time of absence in: minor (<7 days), moderate (7–21 or 28 days) and severe (>21 or 28 days) [5, 9, 17]. Minor injuries account for 33–42 % of the total injuries, moderate injuries 40–47 % and severe injuries from 11 to 18 % [5, 9, 17].

One of the main problems in muscle injuries is recurrence which is even more common in the hamstrings muscle group [12, 16]. In football, most commonly midfielders and forwards re-injure the hamstrings and defenders re-injure the quadriceps [16]. Re-injuries cause a longer absence period than first muscle injuries [2, 5] with recurrence rates from 20 to 33 % [4]. Visser et al. [23], in a systematic review, found that the main risk factors for recurrent hamstring injuries are an initial grade 1 hamstring injury, a large volume first injury and ipsilateral ACL reconstruction.

4.3 Clinical Features

The clinical presentation of muscle injuries is highly variable and depends on many factors related to the mechanism, intensity and type of trauma [24, 25]. Understanding of the injury as well as its cause is crucial for the development of specific treatment and prevention strategies. Injuries associated with direct trauma, with muscle contusion or laceration, occur most commonly in the muscle belly with intramuscular hematoma. Injuries associated with indirect trauma are characterized by muscle strain at the myotendinous transition with minimal deformation of the tendon. These injuries most often occur due to the forces generated by vigorous eccentric contraction [26–29].

Muscle injuries affect mainly the lower limbs and specifically superficial and bi-

articulated muscles, such as the hamstrings and rectus femoris. Typically, individuals experience sudden pain after physical exertion associated with variable functional disability. In more severe injuries, there may be a deformity represented by a muscle gap. The presence of a hematoma is variable and has no direct correlation with the severity of the injury. Injuries of extensive muscle groups or in muscles that do not have agonist muscles may cause the muscle to lose strength. Pain improves during the first few weeks, but there is still the possibility of disruptions of the new strands of fibrosis that may be formed between the lesion periphery and the surrounding muscles; this could lead to pain of varying intensity and new hematomas.

In minor injuries, pain is localized and edema may be present, albeit small, as structural damage is minimal. In moderate injuries, symptoms are more severe and a muscle gap may be palpable, but usually not visible; also a hematoma and localized edema are present. In severe injuries, in which there is complete muscle rupture, loss of function and a visible gap, as well as important edema and hemorrhage, increasing the local inflammatory response with augmented redness, pain and tenderness; hematoma size is variable and loss of function can be observed almost immediately.

Considering the wide variety of clinical presentations and the existence of many different classifications, some sort of consensus view seems necessary. A recent consensus statement, published in 2013, divided muscle injuries into two groups: indirect injury and direct trauma [30].

Indirect muscle injuries are also divided into two groups: functional damage and structural damage. Functional lesions include late-onset muscle soreness, muscle fatigue and neuromuscular diseases. Structural lesions are, generally, partial ruptures that can progress to complete ruptures. Originally, strain injuries were classified as structural lesions, but this has changed because a strain refers to a biomechanical phenomenon and not an anatomical feature.

Lesions caused by direct trauma are divided into contusions and lacerations. There is a clear history of an external trauma and the formation of an extra-muscular hematoma is frequent. The use of a single classification system is necessary to assure the exchange of information in clinical research on a regular and reliable basis [30].

4.4 Risk Factors

A recent systematic review and meta-analysis [31] evaluated the risk factors for hamstring muscle injuries in sports and found that older age, increased quadriceps peak torque and past history of hamstring injury were associated with increased risk of hamstring muscle injury. Table 4.2 shows the main risk factors involved in muscle injuries. There are particular intrinsic characteristics of the individuals and their training patterns that are more frequent in those with muscle injuries. Of all the risk factors the ones that have superior clinical evidence are the presence of type II fibers

and a history of previous injuries. Other characteristics of lesser value are gender, age, stretching deficiency and muscle imbalance.

Table 4.2 Risk factors for muscle injuries in sports

Author/year	n	Sport	Risk factor
Arnason [1]	306	Football	HMT: Age, previous inj Groin: previous inj, ↓ ROM hip abd
Bradley [14]	36	Football	Low ROM of knee and hip flexors
Croisier [32]	462	Football	HMT: muscle imbalance
Ekstrand [5]	2299	Football	Calf: age
Emery [11]	1292	Hockey	Groin: previous injury, no training in the off-season
Engebretsen [17]	508	Football	HMT: previous injury
Gabbe [33]	126	Australian Football	HMT: older age (> 23 year), decreased QDs flexibility
Gabbe [34]	222	Australian Football	HMT: older age (> 20 year), previous injury
Hagglund [35]	197	Football	HMT: older age, previous injury Groin: previous injury
Hagglund [6]	1401	Football	HMT, adductors, calf: previous injury Calf: age
Orchard [36]	37	Australian Football	HMT: low HQ ratio, lower HMT strength in the injured thigh
Orchard [13]	1607	Australian Football	HMT, QDs, calf: previous injury HMT and calf: age
Sugiura [37]	30	Track & Field	HMT: HMT eccentric weakness and concentric weakness of hip extensors (<60°/s)
Tyler [38]	47	Ice Hockey	Adductors: adductor strength <80 % of abductor strength
Witvrouw [39]	146	Football	HMT and QD: lower flexibility
Yamamoto [40]	64	Track & Field	HMTs: muscle imbalance (isometric)
Yeung [41]	44	Track & Field	HMTs: decreased HQ ratio at 180°/s

Legend: All subjects were male. *HMT* hamstrings, *inj* injury/injuries, *ROM* range of motion, *abd* abduction, *QD* quadriceps, *HQ* hamstring-to-quadriceps

4.5 Prevention

Several studies have evaluated the impact of stretching and a flexibility training program on the incidence of muscle strains in sports. Some have found no difference [42] while others have found a positive result for injury prevention [43].

The same is true for hamstring strengthening as a prevention program for hamstring

injuries. The most frequently used method for training is the eccentric training program with the Nordic hamstring lowers [42]. Some found that it made no difference [34, 44] while others found a positive effect for injury prevention [42, 43]. Arnason [42] found that an eccentric strengthening program reduced the incidence of hamstring injuries by 65 % but had no effect on injury severity or proportion of re-injuries. Asklin [43] also found that a preseason eccentric strengthening program resulted in a lower occurrence of injuries (20 % vs. 66.7 %). A Cochrane review [45] concluded that there is no evidence, from randomized clinical trials, of the effectiveness of interventions used to prevent hamstring injuries.

4.6 Conclusion

Muscle injuries are very frequent in sports and account for long periods away from physical activities. They affect mainly the lower limbs with the hamstring complex the most frequently injured and also the most prone to re-injuries.

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5. Role of Clinical Evaluation for the Diagnosis of Acute and Chronic Muscle Injuries

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Abstract
Sports-related muscle injuries occur predominantly in the thigh and calf muscles including hip adductors, quadriceps, and hamstring muscles. Rapid and accurate diagnosis is the key to a successful outcome after a muscle injury. Clinically, it is important to differentiate extrinsic lesions from intrinsic lesions as well as to determine their severity. Extrinsic lesions result from a blunt impact – kick, strike –that injures the muscle directly causing a variable degree of damage ranging from simple contusion to laceration of muscle fibers. Intrinsic lesions occur when muscle fibers are stretched more or less violently. The anatomic severity of the injury is not easy to determine but many lesions can be well differentiated with careful physical examination. Until quite recently, clinicians used a wide variety of relatively vague terms such as pulled muscle, strain, overstretching, tear, or rupture to describe these injuries. The current classification attempts to provide a more objective evaluation system (grades 0–4) and offers the advantage of providing a good description of each type of lesion which facilitates the diagnostic procedure. Clinical tests (passive stretching, active contraction against resistance) and palpation are helpful to guide complementary explorations to a specific muscle group or muscle.

5.1 Introduction

Sports-related muscle injuries occur predominantly in the powerful thigh and calf muscles used to jump, run and kick: hip adductors; quadriceps, especially the rectus femoris; hamstring muscles; triceps surae. Rapid and accurate diagnosis is the key to a successful outcome after muscle injury. Any delay increases the risk of serious consequences. Clinically, it is important to differentiate extrinsic lesions from intrinsic lesions and benign lesions that will heal readily from severe lesions that require prolonged treatment. Extrinsic lesions result from a blunt impact – kick, strike –that injures the muscle directly causing a variable degree of damage ranging from simple contusion to laceration of muscle fibers. Intrinsic lesions occur when muscle fibers are stretched more or less violently. The anatomic severity of the injury is not easy to determine but many lesions can be well differentiated with careful physical examination. Until quite recently, clinicians used a wide variety of relatively vague terms such as pulled muscle, strain, overstretching, tear, or rupture to describe these injuries. The current classification attempts to provide a more objective evaluation system by grading the injuries from 0 to 4 [1, 2].

5.2 Acute Muscle Injuries

5.2.1 The Clinical Diagnosis

The clinical diagnosis relies on two fundamental and complementary elements: history taking and physical examination. For most cases, the clinical diagnosis will describe the precise localization of the injury and its potential severity.

5.2.1.1 Clinical History

The information collected by taking the patient's history is essential to understanding the severity of the injury. Data collected will concern the characteristic features of the pain and associated signs as well as the functional impact [3].

- *Pain at the moment of the accident*

Unlike the pain caused by tendon or ligament injury, the pain caused by muscle injury provides information on the severity of the lesion in only a very small number of cases. For most accidents, pain occurs suddenly, described as like being stabbed, kicked or hit hard with a ball. In certain circumstances, essentially after a benign lesion, pain will develop progressively.

- *Associated signs*

A crack or a pop may be audible. Sometimes the patient reports a shock wave

running through the muscle, or describes a tight knot snapping shut.

- *Functional impact*

For the majority of severe muscle injuries, functional impairment is the rule. The clinical presentation varies depending on the muscle and the specific site of the injury. For instance, following a lesion without avulsion affecting the upper part of the rectus femoris, patients readily report nothing more than an uncomfortable feeling. But after a tear in the mid portion of the muscle they may limp or even be unable to stand on the injured side.

The functional impact of a benign lesion is usually mild to moderate but important enough to prevent the patient from continuing the sports activity.

5.2.1.2 Physical Examination

The well-conducted physical examination provides information essential for the diagnosis.

- *Inspection* to search for swelling affecting a more or less extensive area of soft tissue, increased limb volume, local or more distant ecchymosis (Figs. 5.1 and 5.2).

Fig. 5.1 Acute injury to the medial gastrocnemius: increased calf volume and characteristic forefoot gait

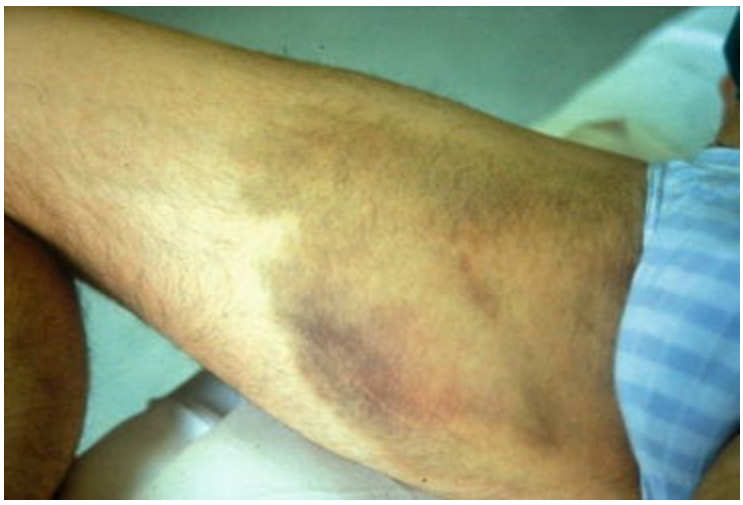

Fig. 5.2 Acute injury of the hip adductors: early development of a large hematoma

- *Palpation* to search for a more or less localized tenseness and/or crepitus (Fig. 5.3). In some cases, exquisite pain is discovered within the contracted mass, in others a depressed area may be palpated in the muscle. In many cases the muscle is abnormally taut.

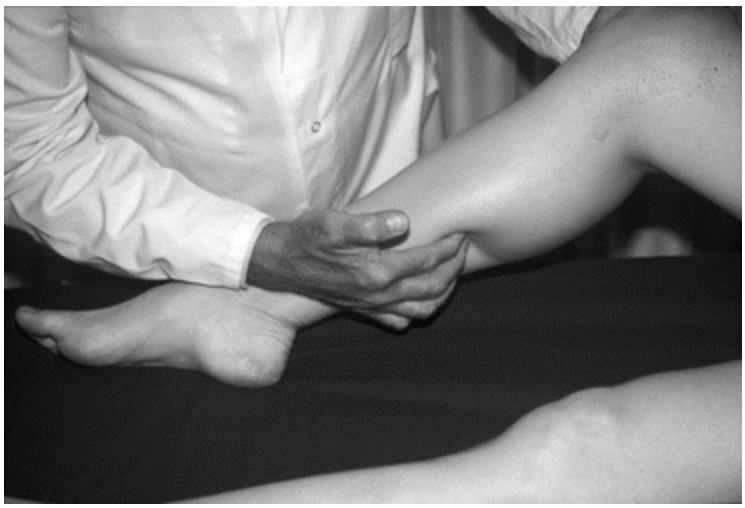

Fig. 5.3 Acute injury of the medial gastrocnemius: palpation disclosed a painful defect at the base of the muscle

- *Passive stretching* is a routine practice: the adductor muscles are studied with the patient in the supine position, the hip in abduction; the hamstring muscles are evaluated by raising the leg in the supine position or by anterior flexion of the trunk in the upright position; the quadriceps is examined by flexing the knee while the patient is in the prone position; the triceps is explored by passive dorsiflexion of the foot with the knee first extended then flexed in order to dissociate the gastrocnemius from the soleus (Fig. 5.4)

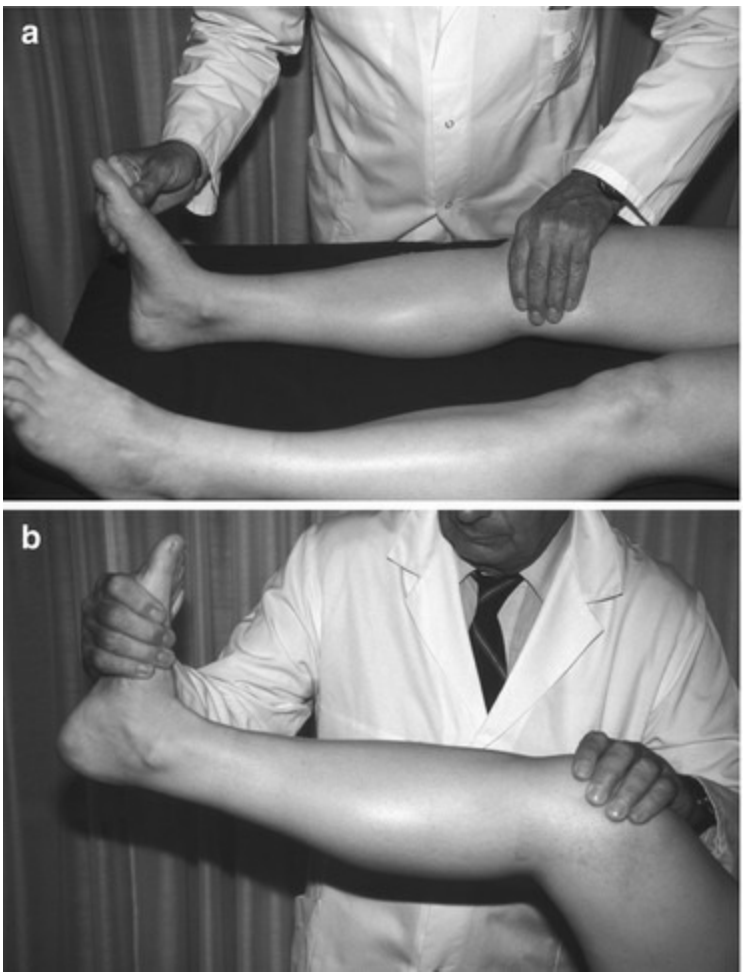

Fig. 5.4 Gastrocnemius injury: passive knee flexion is limited and painful on the extended knee (a) and returns to normal on the flexed knee (b)

.

- *Contraction against resistance* provides information on important elements depending on the result (possible, reduced resistance, impossible); the development, or not, of a depression or on the contrary a globular mass may be difficult to interpret: healthy muscles may be replacing the function of an injured muscle; soft tissue edema can mask a muscle deformity that will only be seen later after the swelling has subsided.

5.2.2 Clinical Presentations [4–6]

5.2.2.1 Muscle Contusion

The clinical presentation is variable, depending upon the severity of the trauma.

- *Benign contusion* The initial traumatic event may have gone unnoticed, the athlete being able to continue sports activities. Painful discomfort may develop later

leading to a retrospective recognition of the symptoms. Similarly, repeated microtrauma can lead to an insidious presentation with late-onset diffuse pain, in an imprecise localization, affecting the entire muscle.

The functional impotency can be marked, maximal before warm-up, rescinding with exercise and reappearing with forced movements or fatigue.

The physical examination reveals a slightly tense muscle compartment with edema and a taut muscle. Passive mobilization of the joints above and below the injury does not trigger pain. Inversely, mobilization against resistance is painful, providing objective pain-equivalent evidence of decreased resistance compared with the healthy side.

- *Severe contusion* A sudden shock (kick, blunt trauma) may produce a severe contusion. The injured muscle is usually contracted when the traumatic event occurs. The characteristic feature is the formation of a hematoma due to rupture of the capillaries or small vessels. Laceration of a certain volume of muscle fibers may be associated. The hematoma may spread over the peripheral aspect of the muscle, form under the aponeurosis, or remain localized within the muscle mass (Fig. 5.5).

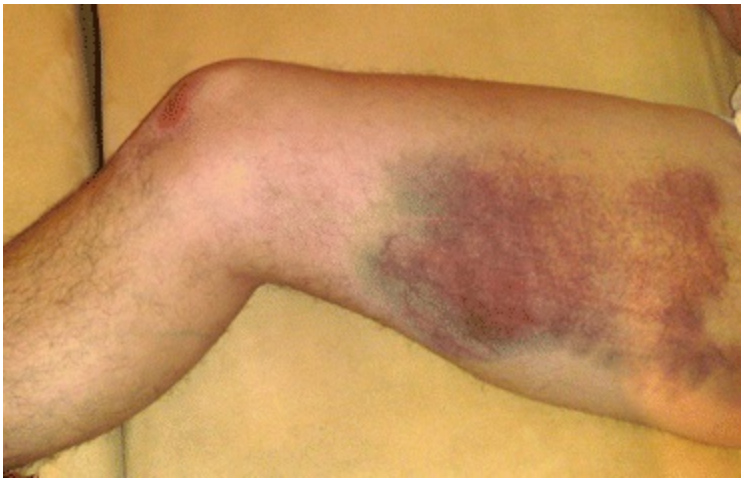

Fig. 5.5 Diffusion of the hematoma 72 h after injury

The patient reports a precise highly painful event followed by immediate and often total functional impotency.

At inspection the muscle is abnormally stiff and increased in volume. Palpation reveals exquisite pain, swelling, and painful stiffness of the injured area. In certain cases, any attempt for active contraction reveals complete impotency.

The clinical course varies with the severity of the injury. For a benign contusion, athletes generally return to sports activities rapidly. If the contusion is severe, recovery depends on the resorption of the hematoma which is generally uneventful once the

hematoma has spread into the subcutaneous tissues or formed a distal ecchymosis (Fig. 5.6).

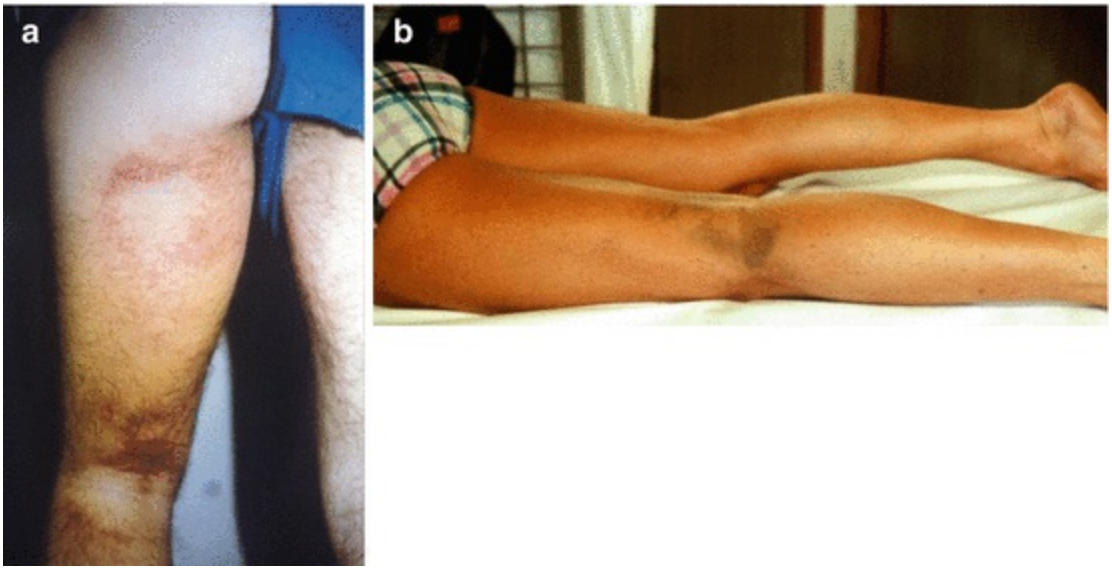

Fig. 5.6 Large ecchymosis on the posterior aspect of the thigh after proximal hamstring injury (**a**). Ecchymosis that developed three days after a pulled hamstrings (**b**)

Occasionally, fibrous scar tissue or an encysted or calcified hematoma may be a source of pain.

5.2.2.2 Overstretching

Theoretically, overstretching corresponds to stretching beyond the physiological limits of the muscle's elastic capacity without causing anatomic damage. In practice, overstretching generally involves some damage to a few muscle fibers that rupture or tear out of the aponeurosis. These tears are much localized and are followed by the formation of a small serohematic cavity.

Pain onset is generally sudden but can be progressive in certain cases. Functional impairment is minimal or absent.

Sports activities can be continued but at the cost of painful discomfort and reduced performance.

The physical examination provides little information. There is no increase in muscle volume and no painful points at palpation, but the muscle is "tender" over the entire length. Contracture is moderate. Passive stretching may reproduce the pain, contrasting with the discomfort triggered by active contraction against resistance.

The clinical course is rapidly favorable with full recovery in a few days.

5.2.2.3 Muscle Tears

A muscle tear corresponds to the rupture of a more or less significant portion of muscle fibers.

Pain onset is sudden and intense. Patients report it was like being stabbed. All activity is stopped immediately.

The physical examination finds a swollen portion of the limb. There may be a subcutaneous ecchymosis that in some cases only develops a few days later. At palpation, a point of pain is found in the zone of contracture. In certain specific situations, particularly in the event of injury to a superficial muscle, a defect can be palpated in the muscle mass. Total impotency is the rule, but if contraction remains possible, the pain is very intense during isometric testing.

5.2.2.4 Muscle Ruptures

Muscle ruptures can be partial or total.

The incident is a sudden event causing intense pain during a muscle movement. There may be a crack or a pop. Functional impotency is total and generally immediate but sometimes delayed.

At the physical examination, there is a major tumefaction of a muscle segment (Fig. 5.7). An ecchymosis develops early and can be voluminous. It appears at the site of the muscle injury. Palpation may find a more or less voluminous hematoma that is sometimes fluctuant. In certain cases, there is a painful defect. Active contraction is rarely possible. When present, and if the swelling is not to important, a contracting tumefaction can be palpated underlying the rupture.

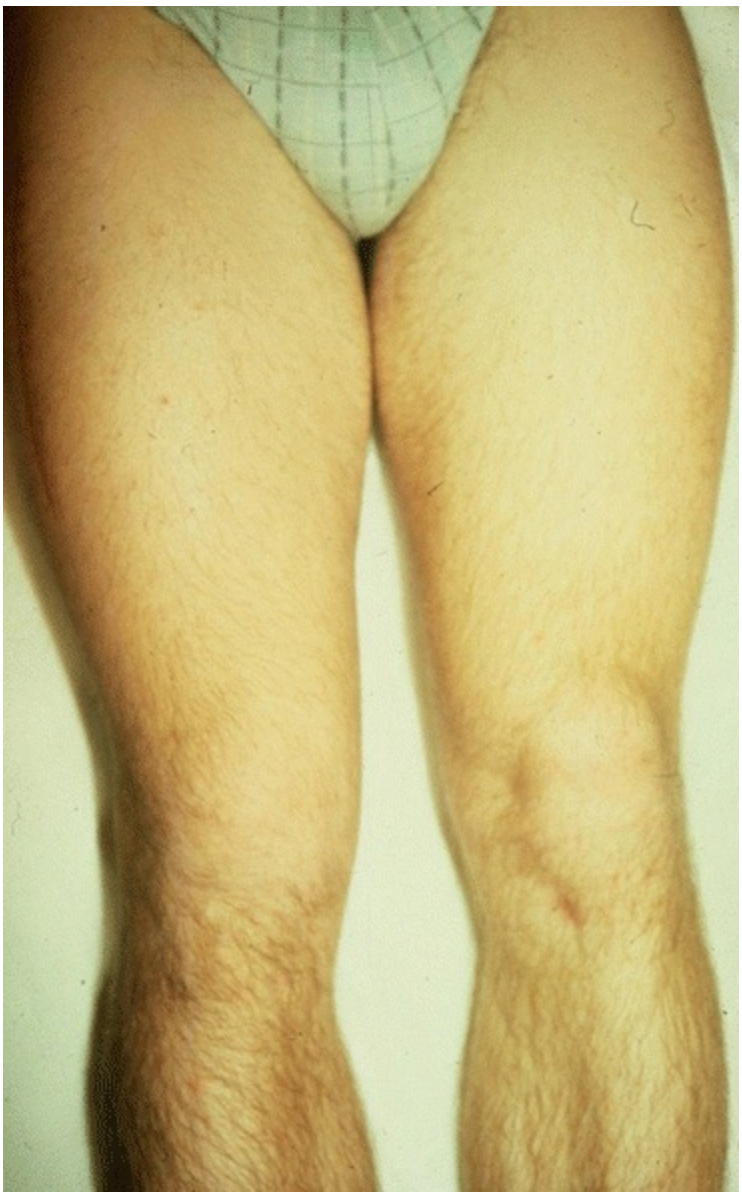

Fig. 5.7 Swollen thigh and right knee after direct impact (knee) on the anterior aspect of the thigh

5.2.3 Current Classification of Muscle Injuries

Four grades are described [2–4].

5.2.3.1 Grade 0

Reversible disorder affecting a small number of muscle fibers.

Symptoms are moderate: pain, muscle contracture and reduced force. Recovery is complete in a few hours.

5.2.3.2 Grade 1

Irreversible disorder affecting a small number muscle fibers and the integrity of the

supportive connective tissues.

The symptoms are similar to those in grade 0 but with more intense pain and contracture. The reduction in force is more marked. Muscle fiber regeneration ensures total recovery in a few days. Patients can return to sports activities when the pain has resolved.

5.2.3.3 Grade 2

Irreversible disorder affecting a small number of muscle fibers and moderate injury of the supportive connective tissues without excessive disorganization. There is no hematoma, even at ultrasound.

The symptoms are intense pain occurring during a sports movement not imposing immediate interruption. The functional impact is variable depending on the localization.

The clinical course is rapidly favorable. Good quality repair can be obtained in 10–15 days but not *ad integrum*. Return to sports is based on the clinical tests: no pain at contraction against resistance and stretching.

5.2.3.4 Grade 3

Injury affecting numerous muscle fibers with marked involvement of the supportive connective tissue that becomes disorganized with formation of a generally localized hematoma.

Symptoms are acute pain during a sports activity imposing immediate interruption. Marked functional impotency.

Recovery is long but of variable duration: 4–6 weeks, up to 12 weeks. Length of recovery time depends on the severity of the anatomic damage: number of muscle fibers injured, volume of the hematoma, status of the aponeurosis. Recovery also depends on the quality of care, from the acute phase to the remodeling phase.

5.2.3.5 Grade 4

Partial or total muscle rupture, with massive injury to the supportive connective tissues and formation of a voluminous and diffuse hematoma (Fig. 5.8).

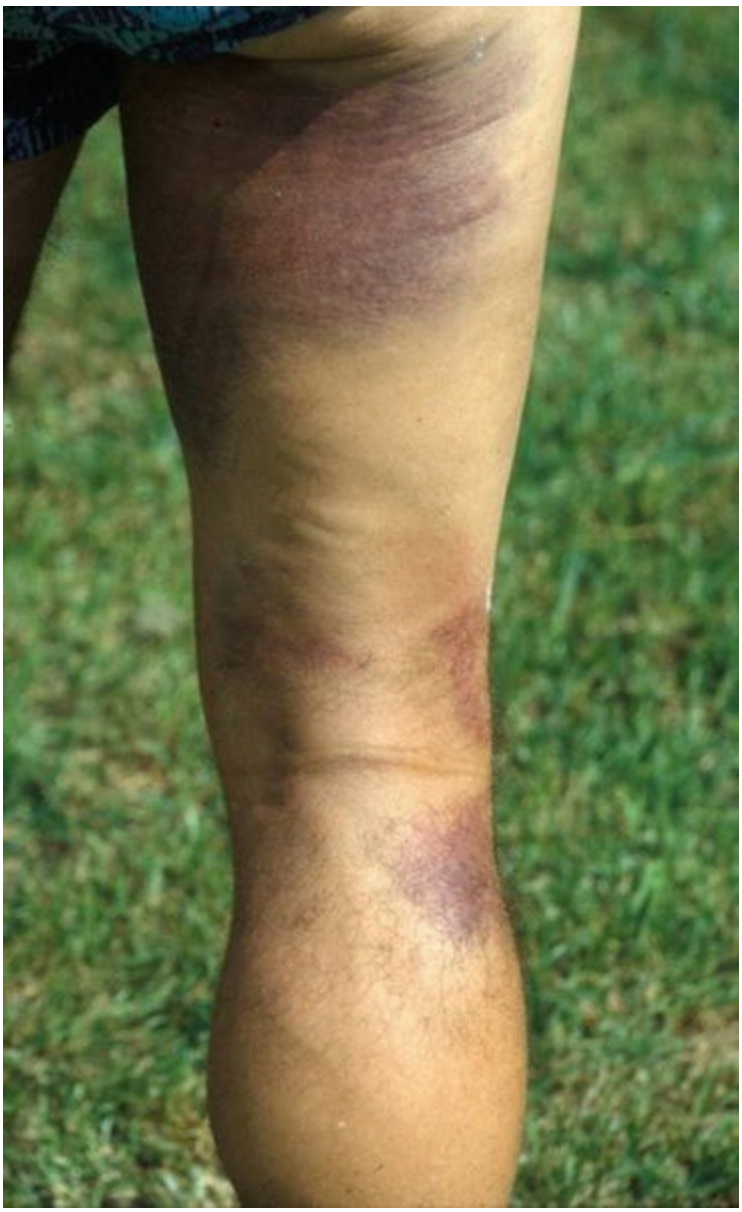

Fig. 5.8 Very voluminous ecchymosis on the thigh and leg after disinsertion from the ischiatic tubercle

The symptoms are violent pain occurring during a sports movement imposing immediate interruption of the activity. Functional impotency is total.

The clinical course is not as poor as could be expected. For total ruptures, the gap between the two muscle stumps is wide so that the fibrous scar tissue is not subjected to traction. Pain subsides. The loss of function is tolerated particularly well if the ruptured muscle is part of a muscle group in which other muscles can compensate for the functional impairment.

In conclusion, it is generally possible to establish a clinical diagnosis of the severity of a recent muscle injury.

The diagnosis must be based on careful history taking to determine the precise circumstances of pain onset and the functional impact of the injury.

The physical examination is conducted according to a rigorous protocol including inspection, palpation, active contraction, passive stretching and contraction against resistance.

In the majority of cases, the physical examination is sufficient to establish the diagnosis of severity. For certain cases, complementary investigations will be needed, particular ultrasound.

5.3 Chronic Muscle Injuries

5.3.1 Introduction

Classifications proposed for trauma-induced chronic muscle injuries are basically anatomopathological classifications, especially since the clinical presentations can be ambiguous [7, 8].

Currently, three major categories of inappropriate healing resulting in functional impairment are described:

- nodular formations (scar tissue granuloma, fibrous nodule, sclerotic formation) result from the proliferation of poorly organized fibrous tissue developed during the healing process after muscle injury due to lacerations, tears, ruptures or avulsions. This nodular tissue does not have the elastic quality of muscles;
- the cysts or pseudocysts develop by progressive encapsulation of residual effusion creating an intramuscular fluid-filled cavity surrounded by a zone of sclerotic tissue;
- intramuscular calcifications may take on many forms, localized strings or very diffuse localizations. Ossification phenomena are also observed, leading to a muscular osteoma.

5.3.2 Scar Tissue Granuloma, Fibrous Nodule, Sclerotic Formation

These three nodular formations result from anarchic proliferation of fibrous tissue disorganized by the muscle injury (laceration, tear, rupture, avulsion).

Reason for Seeking Care

Most patients consult because of chronic pain provoked by repeated exercise or specific movements resulting from muscle contractions that stretch or distract the scar tissue. The pain may also be caused by repeated focal strains.

History

Most patients report a typical history starting after a sudden painful inaugural event, usually an indirect trauma, that led to an immediate interruption of the sports activity. After three weeks or so without sports, the muscle involved is still tender when stretching or contracting. Attempts to return to sports activities are unsuccessful because the patient perceives an analogous traumatic phenomenon, but with less pain for a shorter time. After interrupting the activity for several months, a final attempt is marked by recurrent pain.

Physical Examination

Muscle atrophy may be observed in a limb segment or muscle compartment (Figs. 5.9 and 5.10). Exceptionally, a hard scar formation can be palpated. Muscle contraction against resistance (manual or mechanical loading) and passive stretching reproduce the pain. The limitation of muscle force is difficult to determine because of the pain.

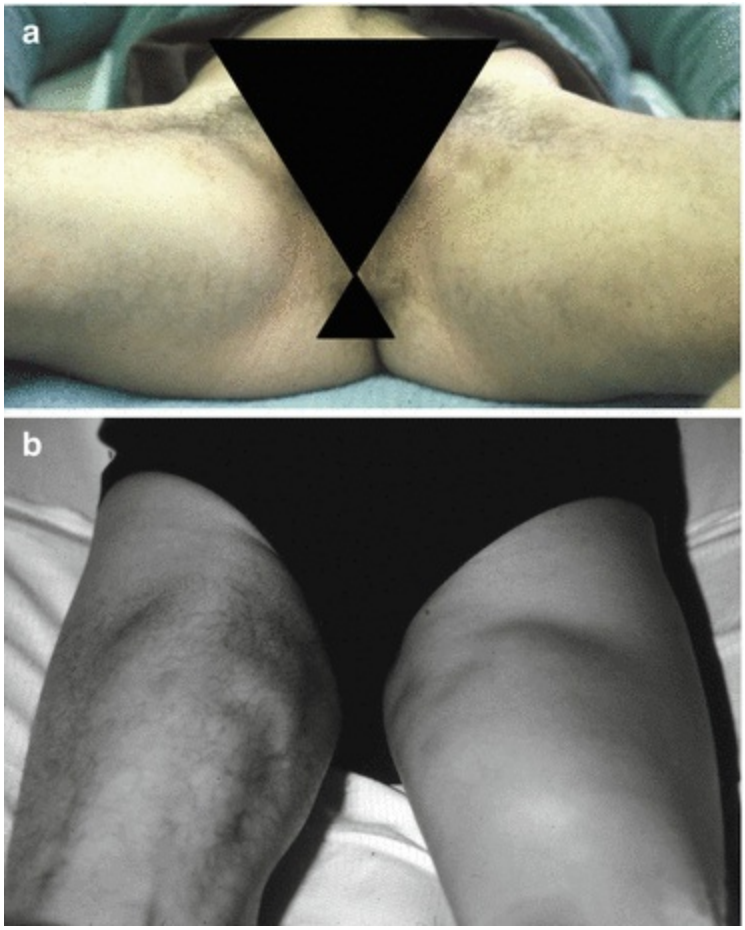

Fig. 5.9 Old injuries of the adductors (**a**) and hamstrings (**b**) revealing complete disinsertion

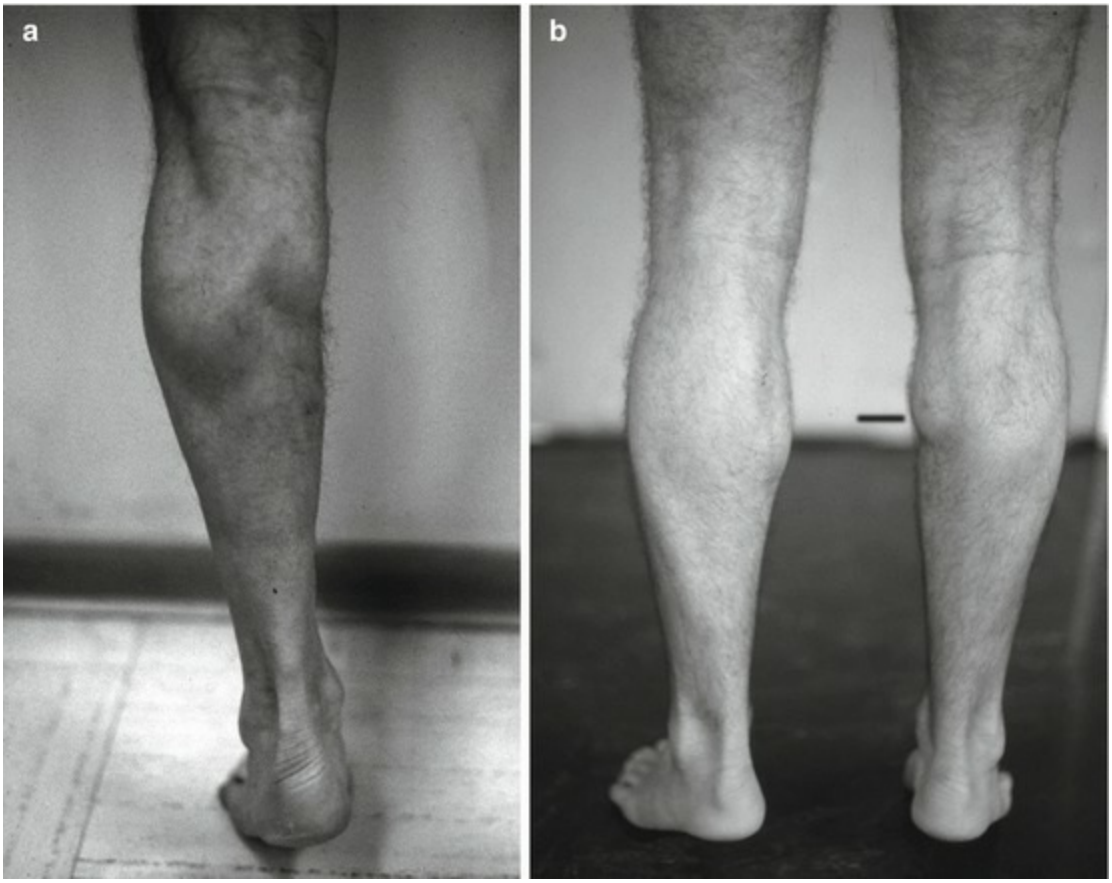

Fig. 5.10 Fibrous degeneration of the medial gastrocnemius (**a**, **b**)

5.3.3 Cystic Lesions

Cysts or pseudo-cysts develop when the initial hematoma or residual effusion is encapsulated by a zone of dense sclerotic tissue. These lesions are generally observed in the rectus femoris, the hamstrings, and the triceps surae after distal disinsertion of the medial gastrocnemius.The patient consults for well-focalized exercise-induced pain reproduced by specific movements. There is a history of a muscle tear.If the localization is sufficiently superficial (rectus femoris, medial gastrocnemius), the cyst can sometimes be palpated as a renitent tumor perceived or not by the patient. Palpation however does not provide any information for deep cysts (hamstrings). At best, certain muscles may appear abnormally tight or painful at passive stretching or contraction against resistance. Such tests are however non-specific.

5.3.4 Muscle Calcifications: Ossification Phenomena

Relatively superficial moniliform calcifications secondary to short longitudinal lacerations must be distinguished from more voluminous calcifications as observed in ossifying myositis or periostic ossifications.

5.3.4.1 Moniliform Calcifications

Functional symptoms are not very specific. The patient describes chronic exercise-induced pain but when questioned fails to recall a specific traumatic event, especially in case of successive micro-lacerations.

After repeated contractions, it is sometimes possible to palpate painful nodular indurations and/or a thickened aponeurotic reaction over a string of calcifications.

5.3.4.2 More Extensive Calcifications

In soccer players, these "ossifications" have been described as osteomas in the rectus femoris, the hip adductors, the sartorius, or the hamstring muscles a few weeks after a direct trauma (contusion) caused by a tackle or a missed kick.

Post-trauma muscle osteoma can take on the form of an ossifying myositis or periostic ossification depending on the intramuscular or peripheral localization of the initial hematoma formed after the muscle injury.

The pathogenic mechanisms remain a matter of debate. Certain authors accept the classical notion of calcification of the hematoma only for periostic ossification. The problem remains as to how an intramuscular fibrous scar tissue becomes calcified.

The patient generally consults for chronic exercise-induced pain that developed after an acute event. A rather hard mass may have formed in the painful area a few weeks after a sharp pain during a missed kick or on the medial aspect of the thigh after a violent spread.

The physical examination reveals signs of muscle suffering at passive stretching and contraction against resistance or a palpable tumor in the muscle concerned [9].

5.4 Conclusion

It is not possible to establish a coherent classification of sports-related chronic post-trauma muscle lesions on the basis of the initial injury, the specific movement incriminated, the muscle involved, or the non-specific clinical presentation. The current classification system based on anatomopathological elements offers the advantage of providing a good description of each type of lesion which facilitates the diagnostic procedure. Thus, patients consult for painful exercise-induced functional discomfort that developed a few weeks after a traumatic event identified by history taking. It is then important to determine the precise mechanism of the event. Clinical tests (passive stretching, active contraction against resistance) and palpation are helpful to guide complementary explorations to a specific muscle group or muscle.

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6. Imaging Semiology: Ultrasound and MRI in the Assessment of Muscle Injury

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Abstract
Competitive athletes are at a high risk for acute and repetitive muscle injury. Although the clinical examination remains the core of any patient evaluation, radiology plays an increasingly important role in the initial assessment and follow-up of muscle injury. Muscle injuries are responsible for a large proportion of time lost to competition. Appropriate management decisions, return to training and competition, and prediction of injury recurrence may all be influenced by appropriate imaging. Magnetic resonance imaging (MRI) has been applied to muscle injuries for more than a decade and has evolved into a valuable tool for the routine evaluation of traumatic muscle injuries in athletes. Ultrasound remains a popular alternative modality for the assessment of acute muscle injury and has some definite advantages over MRI: lower cost and greater availability, short imaging time, no contraindications (e.g. pacemakers), capability for dynamic imaging and comparison with the contralateral side. However, user dependence and lower sensitivity tend to outweigh some of these advantages. This chapter will outline the semiology of muscle injuries, that is describe different types and signs of musculotendinous injuries including musculotendinous strain, muscle contusion and avulsion injury. The focus of this chapter is on clinically applicable techniques of standard MRI and ultrasound.

6.1 Introduction

Football players, dancers, runners and jumpers, and other competitive athletes are at high risk for acute and repetitive muscle injury [1]. Although the clinical examination remains the core of any patient evaluation, radiology plays an increasingly important role in the initial assessment and follow-up [2]. Muscle injuries are responsible for a large proportion of time lost to competition [3], and for all professional athletes rapid return to training and competition is a priority. However, it is also important not to return to competition too soon, when the risk of recurrent injury is high [2]. Management decisions, return to training and competition, and prediction of injury recurrence may all be influenced by appropriate imaging [4–8].

Magnetic resonance imaging (MRI) has been applied to muscle injuries for more than a decade [9–12] and with the advent of high field systems and dedicated imaging protocols, it has evolved into a valuable tool for the routine evaluation of traumatic muscle injuries in athletes [13].

Ultrasound remains a popular alternative modality for the assessment of acute muscle injury and has some definite advantages over MRI, such as lower cost and greater availability, short imaging time, no contraindications (e.g. pacemakers), capability for dynamic imaging and comparison with the contralateral side [14]. However, user dependence and lower sensitivity tend to outweigh some of these advantages [14].

This chapter will outline the semiology of muscle injuries, that is describe different types and signs of musculotendinous injuries including musculotendinous strain, muscle contusion and avulsion injury. Secondly, characteristics of both MRI and ultrasound, including advantages and limitations of each will be discussed. Finally, the natural history of muscle injuries and clinical relevance of imaging will be summarized. The focus of this chapter is on clinically applicable techniques of standard MRI and ultrasound.

6.2 Semiology: Types of Musculotendinous Injuries and Imaging Features

6.2.1 Musculotendinous Strains and Tears

Musculotendinous strains and tears may be caused by a single traumatic event from excessive stretching on musculotendinous fibers (as in high-speed runners) [1], from movements involving excessive range over sequential joints (in dancers) [1], or as a result of eccentric contractions (in football players) [15]. The lesion is commonly located at the musculotendinous junction, usually involving the superficial muscle layers, but the location may vary depending on the mechanism of injury. Clinically,

musculotendinous strains can be classified as grade 1, grade 2, and grade 3 based on absent, mild, or complete loss of muscle function [11, 16]. A recent consensus statement expanded this classification and introduced an additional grade of "neuromuscular muscle disorder", and it differentiates "functional muscle disorders", which are imaging negative or exhibit edema only, from "structural injury" [2]. Validation of this system, which was based entirely on clinical experience, is still lacking but the purpose of any grading system must be to help predict the convalescence period and to design an appropriate rehabilitation program [17]. Muscle strains typically affect the muscles that extend across two joints, have a high proportion of fast-contracting type II fibers and fusiform shape, and undergo eccentric contractions [11, 18]. In the lower extremities, the hamstrings, rectus femoris and gastrocnemius muscles are commonly involved.

In musculotendinous strain without tear (i.e. grade 1 injuries), some fiber disruptions are seen as a result of a stretch injury, but muscle functions are maintained and treatment is conservative. On MRI, interstitial edema and hemorrhage are present at the musculotendinous junction and extend into the adjacent muscle fascicles, producing a feathery appearance (i.e. hyperintensity) on fluid-sensitive sequences [19, 20] (Fig. 6.1). However, up to 45 % of clinically diagnosed grade 1 hamstring injuries may have a normal appearance on MRI according to one study [4]. On ultrasound, the lesion may present as hyperechogenic [21], or hypoechogenic, or may appear normal [21] (Fig. 6.2).

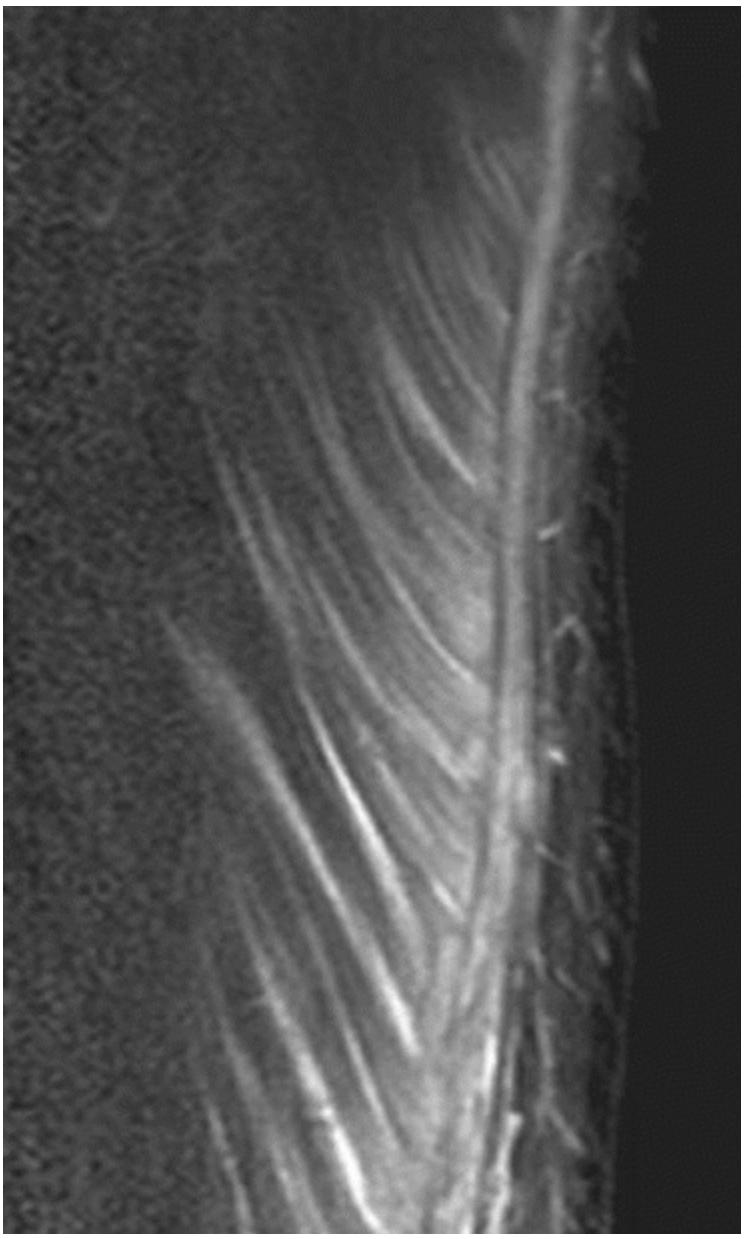

Fig. 6.1 Extensive grade 1 muscle injury on MRI. Coronal T2-weighted fat-suppressed image depicts typical diffuse feathery appearance (of semimembranosus muscle in this example). This effect is due to fluid deposition within the muscle, which leads to a clear definition of the secondary muscle bundles. No circumscribed hematoma is seen in grade 1 lesions

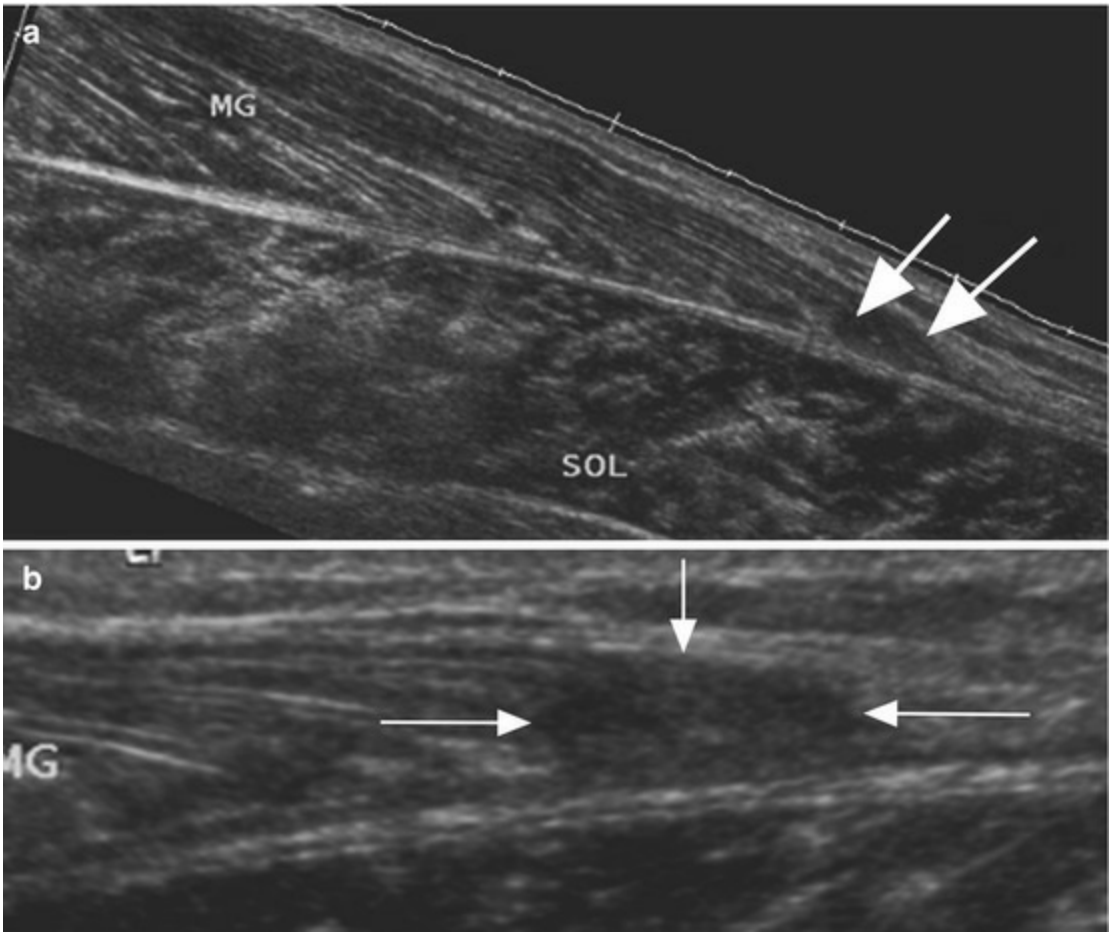

Fig. 6.2 A 27-year-old footballer with a grade 1 injury on ultrasound, presenting with left calf pain. (**a**) On ultrasound examination, a small hypoechogenic area measuring 1 × 0.4 cm was noted (*arrows*), corresponding with a grade 1 strain of the medial head of the gastrocnemius. (**b**) No evidence of hematoma or other abnormality was observed, but only circumscribed, non-fluid-equivalent hypoechogenicity (*arrows*). MRI was not performed

In the presence of partial tears of fibers without retraction (grade 2 injuries), there is a mild loss of muscle function. On MRI, in addition to interstitial edema and hemorrhage, hematoma at the musculotendinous junction and perifascial fluid collection appear as fluid-equivalent hyperintensity on fluid-sensitive sequences. On ultrasound, these pathologic features are depicted as hypoechogenic. Disruption of muscle fibers will be depicted as notable echo inhomogeneity (Fig. 6.3). Treatment of partial tears is also conservative.

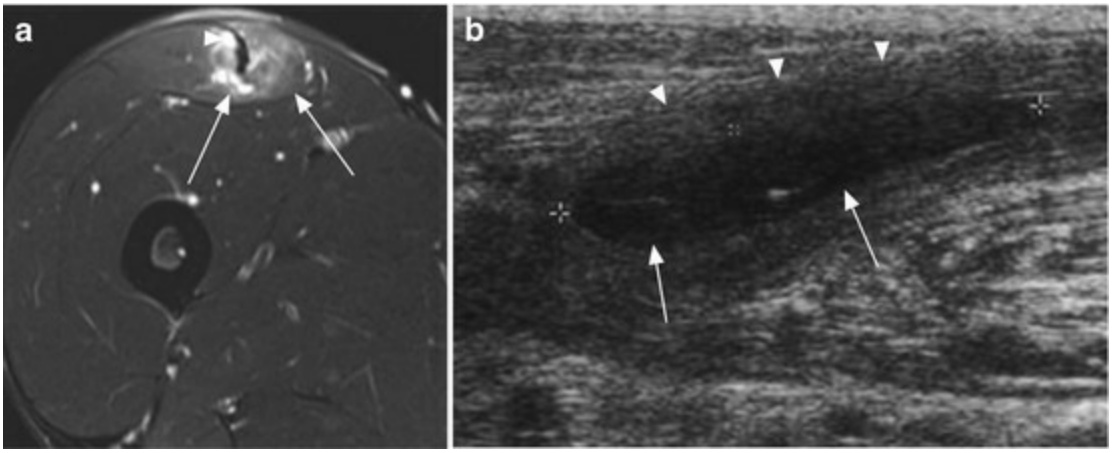

Fig. 6.3 A 26-year-old football player with a Grade 2 muscle injury of the rectus femoris muscle. (**a**) Axial T2-weighted MRI shows diffuse hyperintensity surrounding the central tendon (*arrows*). In addition, there are small patches of fluid-equivalent signal intensity (*arrowhead*) defining this lesion as a grade 2 muscle strain. (**b**) Longitudinal ultrasound image shows liquid-equivalent hematoma (hypoechogenic area depicted by *arrows*). In addition, there are more diffuse edematous changes seen surrounding the hematoma (*arrowheads*)

Complete musculotendinous rupture (grade 3 injury) is commonly accompanied by a hematoma. The diagnosis is usually made on clinical grounds, i.e. complete loss of muscle function, with palpable gap and muscle fiber retraction. Surgical repair is an option, depending on the location of the rupture [22], and both MRI and ultrasound may be useful for preoperative assessment of the extent of retraction [11]. Extensive acute edema and hemorrhage may limit accurate evaluation of the injured muscle. If the tears are left untreated, the ends may become rounded and tether to adjacent muscles or fascia [23].

6.2.2 Muscle Contusion

Muscle contusions result from direct trauma [18]. The injury consists of a well-defined sequence of events involving microscopic rupture and damage to muscle cells, macroscopic defects in muscle bellies, infiltrative bleeding, and inflammation. As a complication, myositis ossificans traumatica may develop [24]. Unlike strains, these traumas usually occur deep in the muscle belly and tend to be less symptomatic than strains. Severity depends on the site of impact, the activation status of the muscles involved, the age of the patient, and the presence of fatigue [25].

On ultrasound, muscle contusion is characterized by discontinuity of normal muscle architecture, with ill-defined hyperechogenicity that may cross fascial boundaries [21]. MRI varies according to severity of injury, but typically there is a feathery appearance of diffuse muscle edema on short tau inversion recovery and fat-suppressed T2-weighted images [9] (Fig. 6.4). Increased muscle girth can be observed but there are no other architectural changes, such as fiber discontinuity or laxity. In case of severe trauma with muscle fiber disruptions, deep intramuscular hematoma is seen [11]. Signal

intensity within the hematoma is influenced by the concentration of protein, methemoglobin, magnetic susceptibility at high field strength, and tissue clearance [26]. Acute hematomas (<48 h) are typically isointense on T1-weighted images, and subacute hematomas (<30 days) appear hyperintense relative to muscle on both T1-weighted and fluid-sensitive sequences secondary to methemoglobin accumulation [19]. As the hematoma evolves, a wide range of MR signal intensity can be seen within the collection, depending on the age of degradation products (Fig. 6.5). Chronic hematoma characteristically shows a hypointense rim on all pulse sequences due to hemosiderin. As blood degradation products are reabsorbed over 6–8 weeks, the size of the hematoma will decrease [27].

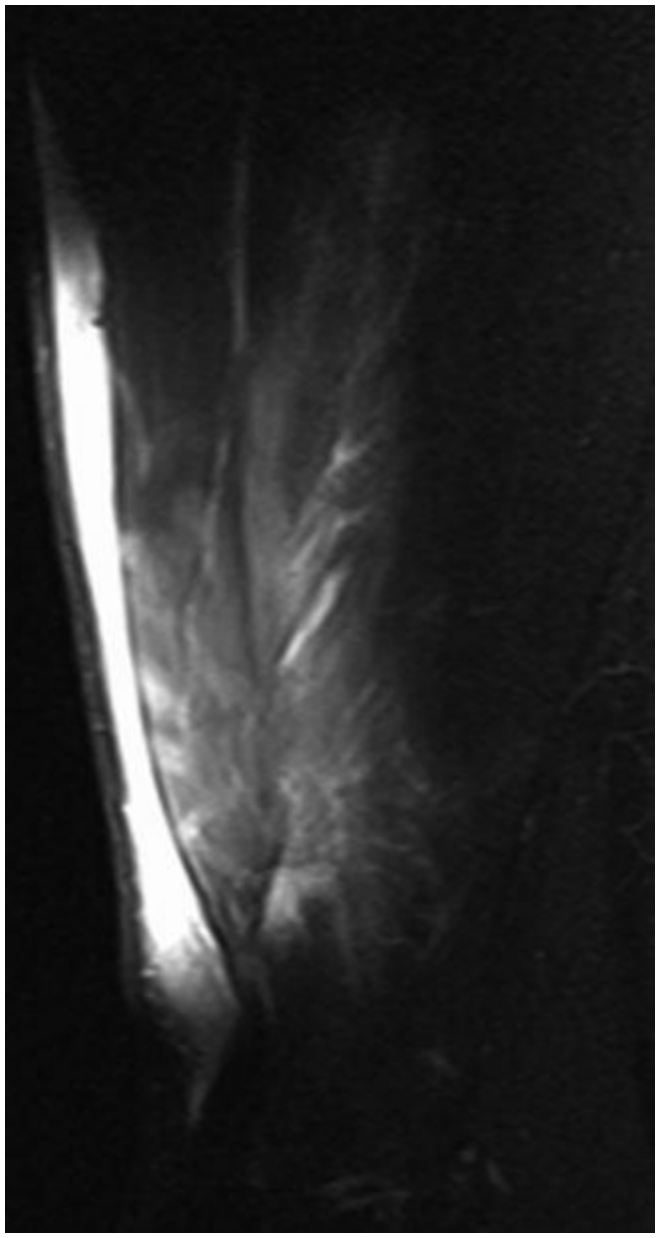

Fig. 6.4 A 22-year-old rugby player presenting with stiffness of the right thigh after receiving a direct blow to the right anterior thigh during a tackle. Sagittal STIR MRI show diffuse intramuscular hyperintensity, consistent with

contusion injury of the rectus femoris, vastus lateralis and vastus intermedius muscles. A large epifascial hematoma is noted superficially. This sagittal image demonstrates the longitudinal extent of the contusion injury and the hematoma

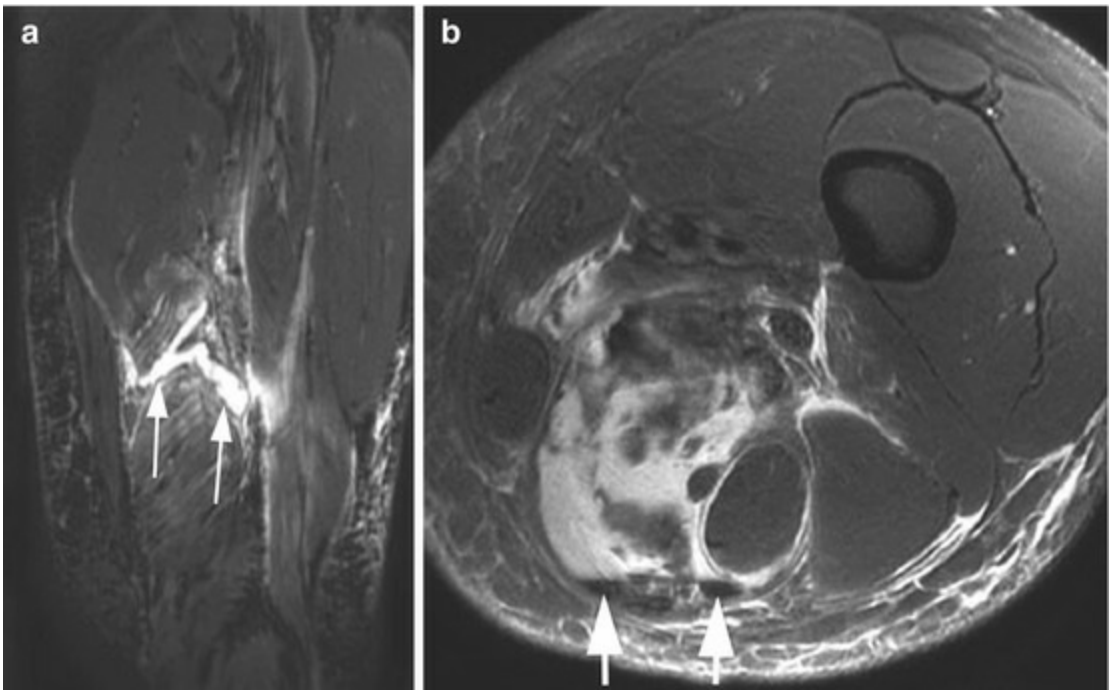

Fig. 6.5 Complete tear of the semimembranosus muscle with minor retraction and hemorrhage. (**a**) Coronal proton density-weighted fat-suppressed MRI shows linear hematoma and complete disruption of the muscle belly (*arrows*). (**b**) Axial proton density-weighted fat-suppressed MRI shows extent of hematoma involving the whole cross-sectional area of the muscle and sedimentation within the hematoma characterized by susceptibility artifacts (*arrowheads*)

6.2.3 Avulsion Injury

Acute avulsion injury results from extreme, unbalanced and often eccentric muscular contractions, and patients with such injuries present with severe pain and loss of function [28]. Adolescents are particularly vulnerable to avulsion injuries because of the inherent weakness of the apophyses. The many apophyses in the pelvis and hip are common sites of avulsion injuries. The single most common site of apophyseal avulsion is at the ischial tuberosity [29]. Cheerleaders, sprinters, gymnasts, track athletes, American football players, and baseball players are commonly affected [29]. Treatment for avulsion injury is generally conservative and the prognosis is good, but non-union may occur.

In acute avulsion injury, periosteal stripping with hematoma at a tendon attachment site can be depicted by MRI. A wavy appearance and retraction of the torn end of the tendon with fragments of bone/cartilage are characteristic. The redundant tendon edge may be lying in a large fluid collection/hematoma (Fig. 6.6). Ultrasound evaluation is useful, but may be difficult due to the presence of a hematoma of mixed echogenicity which has echogenicity similar to the avulsed tendon [21].

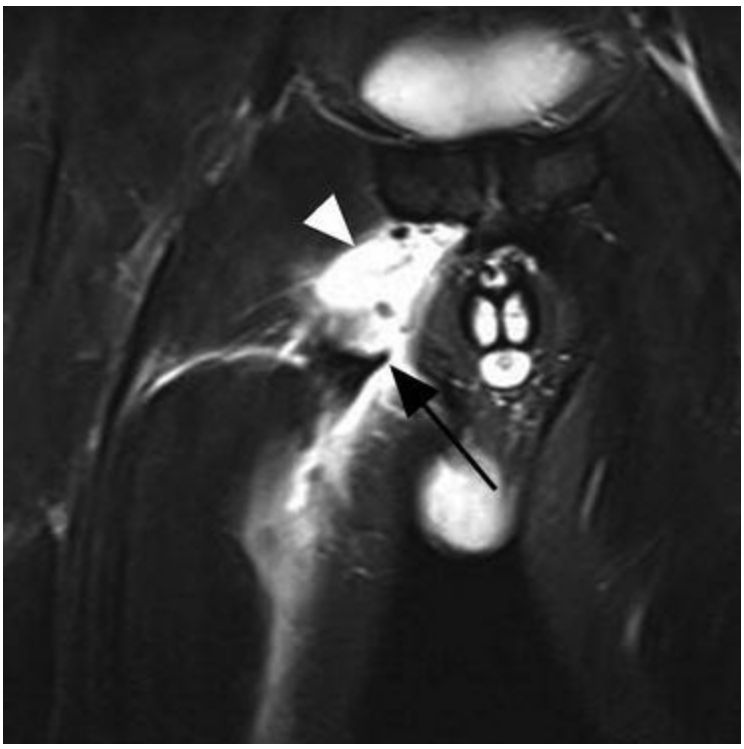

Fig. 6.6 A 21-year-old male javelin thrower presenting with a sudden onset of right-sided groin pain secondary to an avulsion injury. Coronal fat-suppressed T2-weighted MRI demonstrates a wavy appearance and retraction of the torn end of adductor longus tendon (*arrow*) with surrounding fluid-equivalent hyperintensity representing hematoma (*arrowhead*). Small hypointense fragments of avulsed cortical bone from the symphyseal attachment are also noted

6.2.4 Chronic and Repetitive Injuries

Imaging features of chronic musculotendinous injury include muscle or tendon retraction or compensatory hypertrophy, muscle atrophy and formation of scar tissue (fibrosis) [30]. In chronic injuries, T1-weighted images may be normal in low grade injuries, but the fluid-sensitive sequences are helpful for detection of symptomatic old tears which are depicted as abnormally hyperintense [27]. There may be associated surrounding edema and hemorrhage due to re-injury at the site [27]. Scar tissue may be observed as early as 6 weeks after initial injury [14]. On MRI, scar formation appears as hypointense on all pulse sequences and, on ultrasound, areas of scar tissue have irregular morphological features and show heterogeneous echogenicity [31]. It is important to identify the scar tissue because recurrent injuries can occur in close proximity due likely to elasticity differences and altered contractility [27].

6.3 Imaging Modalities

6.3.1 Magnetic Resonance Imaging

MRI is commonly performed to locate the lesion and assess its severity. Under normal

circumstances, images from only the affected area are acquired using a surface coil, but the appropriate coil should be selected to obtain the desired field of view. Imaging of the contralateral side is performed in exceptional cases only (e.g. bilateral injury). Contrast enhancement is rarely needed except to distinguish solid from cystic lesions or to diagnose muscle infarction [10]. To correlate imaging with clinical findings, a skin marker is placed over the area of symptoms (Fig. 6.7). Extent of injuries and associated architectural distortion is assessed using axial, sagittal and coronal images oriented along the long and short axes of the involved musculotendinous unit. The axial plane is useful to assess muscle contours and to delineate the musculotendinous junction and its exact anatomical relation with focal lesions [10], while coronal and sagittal planes are used to assess the longitudinal extent of injury [11].

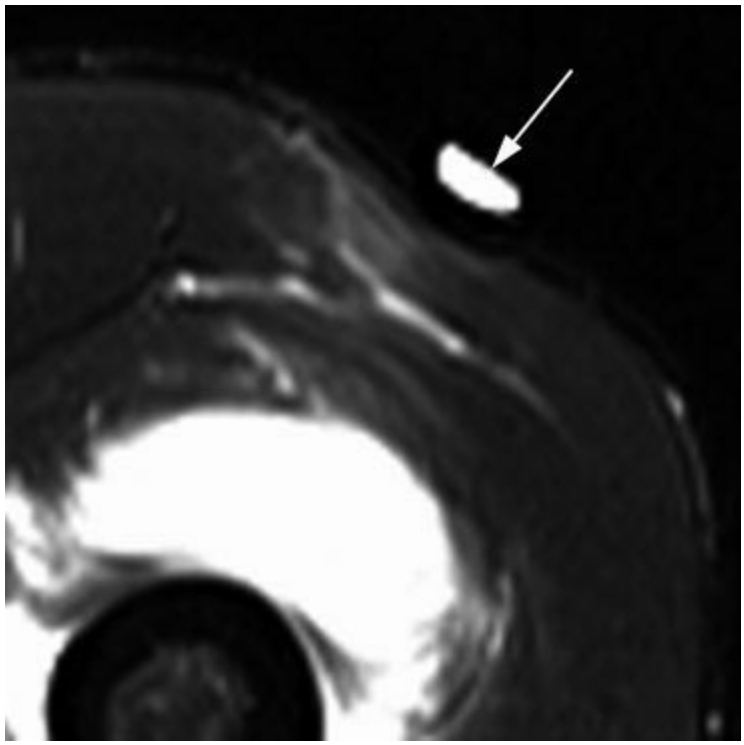

Fig. 6.7 Placing a skin marker (*arrow*) prior to the examination is helpful to define the clinically relevant anatomical region. The marker is commonly a capsule filled with fish oil or vegetable oil. This is especially important in cases of repeated injury in order to differentiate old from incident lesions

Normal skeletal muscles show intermediate to low signal intensity on both T1-weighted (short TR/short TE) and T2-weighted or short tau inversion recovery (STIR) (long TR/long TE) images compared to other soft tissues [19]. Alterations in water content in the affected musculotendinous units are common to all forms of acute traumatic injuries (Figs. 6.2, 6.3, and 6.4) [9–11]. Fluid-sensitive sequences, i.e. fat-suppressed T2-weighted or proton density-weighted turbo spin echo, and STIR sequences are suitable for detecting edematous changes (hyperintensity with a "feathery" appearance) in the musculotendinous unit, and to delineate and locate

intramuscular or perifascial fluid collections or hematomas as hyperintensity [10, 32]. Such sequences can depict abnormal hyperintensities at the site of symptomatic old tears [27]. T1-weighted turbo spin-echo sequences are used to visualize atrophy and fatty infiltration and to differentiate between hemorrhage/hematoma (hyperintense) and edema (hypointense) [11], but they are less sensitive for depiction of soft tissue abnormalities [19]. In chronic muscle injuries, T1-weighted images may not show any signal abnormalities in small tears [27].

6.3.2 Ultrasound

Ultrasound is inexpensive and widely available, and is helpful in the initial assessment of injury in the clinic. Unlike MRI, ultrasound allows a dynamic examination, which aids in clarifying the diagnosis. Power Doppler is useful for identifying hyperemia associated with acute injuries [27]. Hematomas may be drained under ultrasound guidance after liquefaction of the hematoma has occurred. The sensitivity of ultrasound to post-traumatic fluid collections in the acute stage has been shown to be equal to the sensitivity of MRI [31]. However, the sensitivity of ultrasound for detecting ongoing muscle healing during recovery is not as high as the sensitivity of MRI [4]. A study involving Australian football players showed that follow-up MRI 6 weeks after hamstring injury detected persistent abnormalities in 36 % of athletes, whereas the 6-week follow-up ultrasound demonstrated residual abnormalities in only 24 % of patients. It is postulated that the lower sensitivity of ultrasound in prediction of convalescence time is due to underestimation of the degree of injury and to areas of subtle edema that cannot be detected. Overall, the disadvantages of ultrasound seem to outweigh the advantages compared to MRI especially for follow-up imaging [14], because it cannot differentiate with certainty between old and new lesions and it is very difficult to reproduce exactly the same imaging position/plane at baseline and follow-up visits.

6.4 Natural History of Muscle Injuries and Clinical Relevance of Imaging

At follow-up, grade 1 injuries may manifest as a region of hyperechogenicity in up to 50 % of cases on ultrasound [33]. In such cases, normal healing is typically evident as a decrease in size or resolution of the area of hypoechogenicity, together with return of normal muscle architecture and echotexture [32]. More severe injuries may be characterized by the presence of hypoechoic regions indicative of fluid adjacent to muscle fibrils or the epimysium. Resolution or notable reduction in the amount of fluid is expected during the normal healing process. Any hematoma or fluid collection should decrease in size, and macroscopic muscle tears may show echogenicity of the margins

of the tear as healing occurs. As the healing process progresses, small tears may fill with echogenic material, which is presumed to be scar tissue [32]. Scar tissue formation at the site of injury can be seen at 6 weeks [27]. However, ultrasound is less sensitive than MRI to residual muscle injury during follow-up [14].

MRI of healing muscle injuries typically shows gradual resolution of fluid between muscle fascicles and in relation to the epimysium, together with gradual reduction in the extent and intensity of T2 signal within muscle. The degree of resolution of T2 hyperintensity resolution varies depending on the severity of the initial injury, but in many cases MR signal abnormalities have not resolved by 6 weeks despite return to competition, especially when the initial injury was severe. Persistent high T2 signal suggests ongoing healing and resolution of injury in keeping with the fact that the ultrastructural healing process continues for weeks to months, even after the time when athletes usually return to competition [32]. Hypointensity may also be seen during muscle healing, and reflects the formation of scar tissue and/or hemosiderin deposition following hemorrhage. These changes may contribute to the susceptibility artifacts seen on T2-weighted images during follow-up [27]. During the first few weeks of healing, there may be thickening and T2 hyperintensity of the central intramuscular tendon at the site of injury. As maturation of the scar occurs, T2 hypointensity replaces the hyperintensity. In a recent study of athletes with grade 1 and 2 hamstring injuries, follow-up MRI was performed 5–23 months post-injury [30]. Hypointensity representing scar tissue was seen along the musculotendinous junction in 11 of 14 subjects (79 %). Muscle volume reduction following the injury of long head of biceps was observed, but fatty infiltration was infrequent on these follow-up MRI [30] (Fig. 6.8).

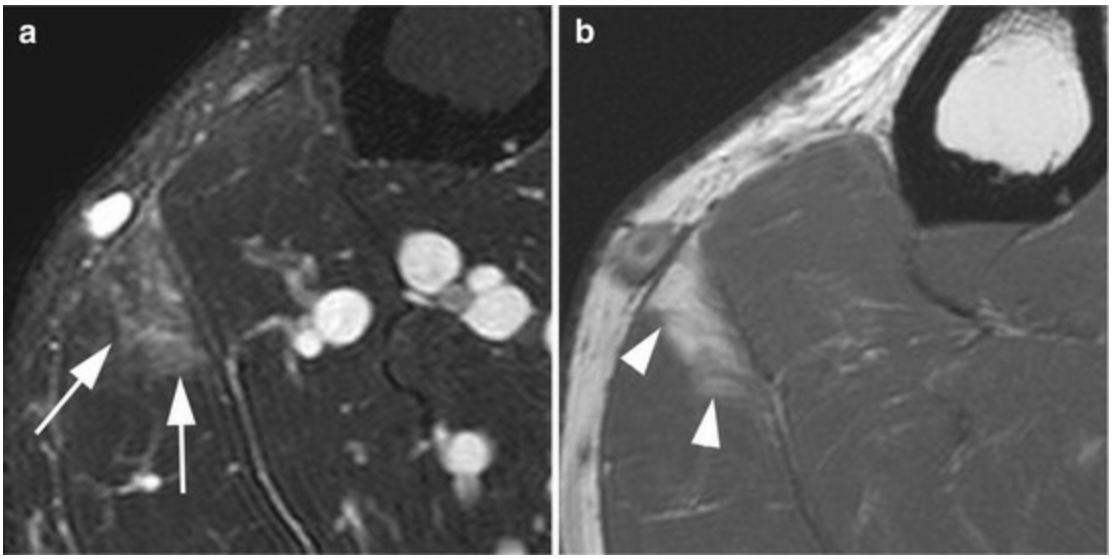

Fig. 6.8 Evolution of muscle injury over time. (**a**) Axial T2-weighted fat-suppressed MRI depicts circumscribed muscle strain of the medial head of the gastrocnemius muscle as a hyperintense edematous area (*arrows*). (**b**) T1-weighted spin-echo MRI acquired 3 years later, shows focal fatty replacement in the previously injured area

(*arrowheads*), a rare finding as most low grade muscle strains will resolve without structural sequelae

There are high demands on sports medicine physicians to quantify the prognosis and to predict when the athlete can return to training and competition. Imaging may assist in the prognostication of the healing process and in predicting the risk of recurrence, but the decision on return to play cannot be dependent on the imaging findings alone and must be balanced against the clinical situation [2, 34]. It has been shown that athletes with a normal MRI in the presence of clinically suspected muscle injury require a shorter convalescence interval (1–2 weeks) [4] and have a lower recurrence rate [32] than those with an abnormal MRI. However, when there is an abnormality on MRI or ultrasound, there is no conclusive evidence that the extent of the abnormality can predict the risk of recurrent injury, whether the images are acquired shortly after the injury or just prior to returning to competition [32].

Imaging parameters used to estimate the extent of muscle injury include the percentage of the cross-sectional area of the affected muscle, the craniocaudal length of the muscle lesion adjacent to the musculotendinous junction, and the approximate volume of muscle injury [32]. These parameters are associated with the duration of absence from competition and thus may guide clinicians in managing muscle injuries [5, 14, 35, 36]. A complete tear of the hamstring, as well as hamstring injuries involving more than 50 % of the muscle cross-sectional area, hemorrhage, fluid collections and distal myotendinous involvement, were all associated with a longer recovery time [35, 36]. A study of sprinters demonstrated that time to return to pre-injury level condition following hamstring injury involving the proximal tendon was significantly longer (approximately 35 weeks) than when the proximal tendon was not involved (less than 15 weeks) [1]. In regard to the quadriceps, sprinters with acute injury involving the central tendon of the rectus femoris were shown to have a mean recovery time of 26.8 days, which was significantly longer than those who sustained injury to the peripheral tendon of the rectus femoris (9.2 days) and vasti muscles (4.4 days). Other studies also showed that the larger the size of the injury on MRI the greater the risk of recurrence [8, 37]. Follow-up imaging may thus provide additional information to support clinical progress through a rehabilitation program [30], although many athletes will return to activity before the MRI findings are resolved [7].

High quality imaging is of great clinical relevance in planning and guiding athlete rehabilitation. It is well established that MRI is the best technique for monitoring the muscle healing process, although this is tempered by the need to let the clinical evaluation guide return-to-play decisions [14]. No official guidelines on the role of MRI or ultrasound in evaluating muscle injuries in high-level athletes have been published. An algorithm that integrates clinical and imaging information into an explicit management plan would be useful and needs to be developed [2, 32].

6.5 Conclusion

MRI seems to be the current imaging method of choice for the initial diagnosis and follow-up of acute musculotendinous injuries, including strain, contusion and avulsion injuries. Ultrasound plays an important role as an adjunct imaging method, but seems less accurate than MRI for assessing the extent of the injury and it cannot differentiate between new and old injuries. It is of great clinical importance to use high quality imaging to plan and guide athlete rehabilitation, although the clinical evaluation itself must guide return-to-play decisions.

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7. Treatment of Muscle Injury

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Abstract
Muscle injuries are one of the most frequent lesions in sports. They can be treated conservatively or operatively. Return to play should be based on the patient's ability to stretch the injured muscle as much as the contralateral healthy muscle, pain-free use of the injured muscle in sports-specific movements (mainly in eccentric exercise), comparable strength between injured and healthy muscles and functional tests. Strategies can be adopted to minimize the recurrence rate and to enhance performance. Novel gene and cell therapies including stem cell therapy are emerging but need more research before they are ready for routine clinical use.

7.1 Introduction

Muscle injuries are one of the most frequent sports lesions [1]. To understand the injury, one must consider the timing of the injury, whether acute or chronic; the type of lesion, whether structural or functional (based on the Munich classification [2]); the grade of the injury in complementary exams, mainly MRI; the specific muscle or muscles

involved; the topography, whether bone avulsion, interstitial or myotendinous junction; and whether it is a first tear, or a recurrence of an old injury [3]. It is critical to provide the best mechanical and biological environment to ensure rapid, complete healing and to prevent a second tear [1, 3].

7.2 Conservative Treatment

The conservative treatment known as PRICE (protection, rest, ice, compression, elevation) is the initial treatment [4]. For two to three days (up to 5 days for more severe injuries), protection should include immobilization or the use of crutches to minimize weight bearing. A brief period of immobilization for the first few days to limit hemorrhage and edema formation can provide time for the new granulation tissue to gain the tensile strength needed to withstand the forces of muscle contraction [3]. Prolonged immobilization is discouraged because of the detrimental long-term effects that could alter the biomechanical properties of the muscle-tendon unit: significant hypotrophy of the healthy muscle fibers, excessive deposition of connective tissue within the muscle tissue, and substantially delayed recovery of the injured skeletal muscle [1, 3, 4].

Ice cold packs can be applied several times a day, not directly on the skin, for approximately 20 min, always considering the adipose thickness around the injury [5]. Compression aims to prevent additional swelling and blood loss: an elastic compression bandage may be helpful [3]. Complications like compartmental syndrome can be avoided with prompt hematoma drainage guided by ultrasound in some specific situations [1, 4].

Nonsteroidal anti-inflammatory agents may also be used for pain relief for the first 2–3 days. The prolonged use of this medication can contribute to an increase in fibrous tissue formation [6]. Muscle relaxants can provide relief of discomfort associated with acute painful muscle injuries in young patients. Among older adults they are associated with sedation and confusion, which may lead to an increased risk of falls and injuries [7]. Caution should be considered before they are prescribed, especially in the elderly.

As the pain and swelling subside, physical therapy will help improve range of motion and strength [1]. This phase should be followed by controlled isometric, isotonic, and isokinetic contractions of the injured muscle group with increasing levels of intensity [3, 4]. Passive painless stretching of the injury is beneficial as it reduces muscle stiffness. Elongation can be performed by gradual stretching, beginning with shifts of 10–15 seconds at a time and then proceeding up to a period of one minute [8].

Ultrasound therapy can contribute to pain relief and assist the initial stage of muscle regeneration [9]. Neuromuscular electrical stimulation can be used to stimulate the muscle group involved and attenuate the hypotrophy related to the injury. The amplitude and frequency of the current should be modulated according to the equipment, type of injury and muscle size. The principle is that random and synchronous recruitment of the

activated motor units, dependent on the position of electrodes from axonal terminals, can evoke an electrical muscle activity [4, 9].

Low-level laser therapy has been used to decrease the inflammatory response, as it enhances the synthesis of collagen and promotes a better environment for muscle repair [10, 11]. Hyperbaric oxygen therapy has been tried as another therapeutic option to improve blood supply to augment the aerobic metabolism and hasten recovery after muscle injury in an animal model [12].

7.3 Operative Treatment

The indications for surgical intervention (Fig. 7.1) after a muscle injury are: bone avulsion or complete tears in the origin of the tendon most commonly involving the biceps femoris, semitendinosus and semimembranosus insertion in the proximal hamstrings with a retraction greater than 2 cm; a large intramuscular hematoma, a complete (grade III) tear of a muscle with few or no agonist muscles, or a partial (grade II) strain if more than half of the muscle belly is torn [13, 14].

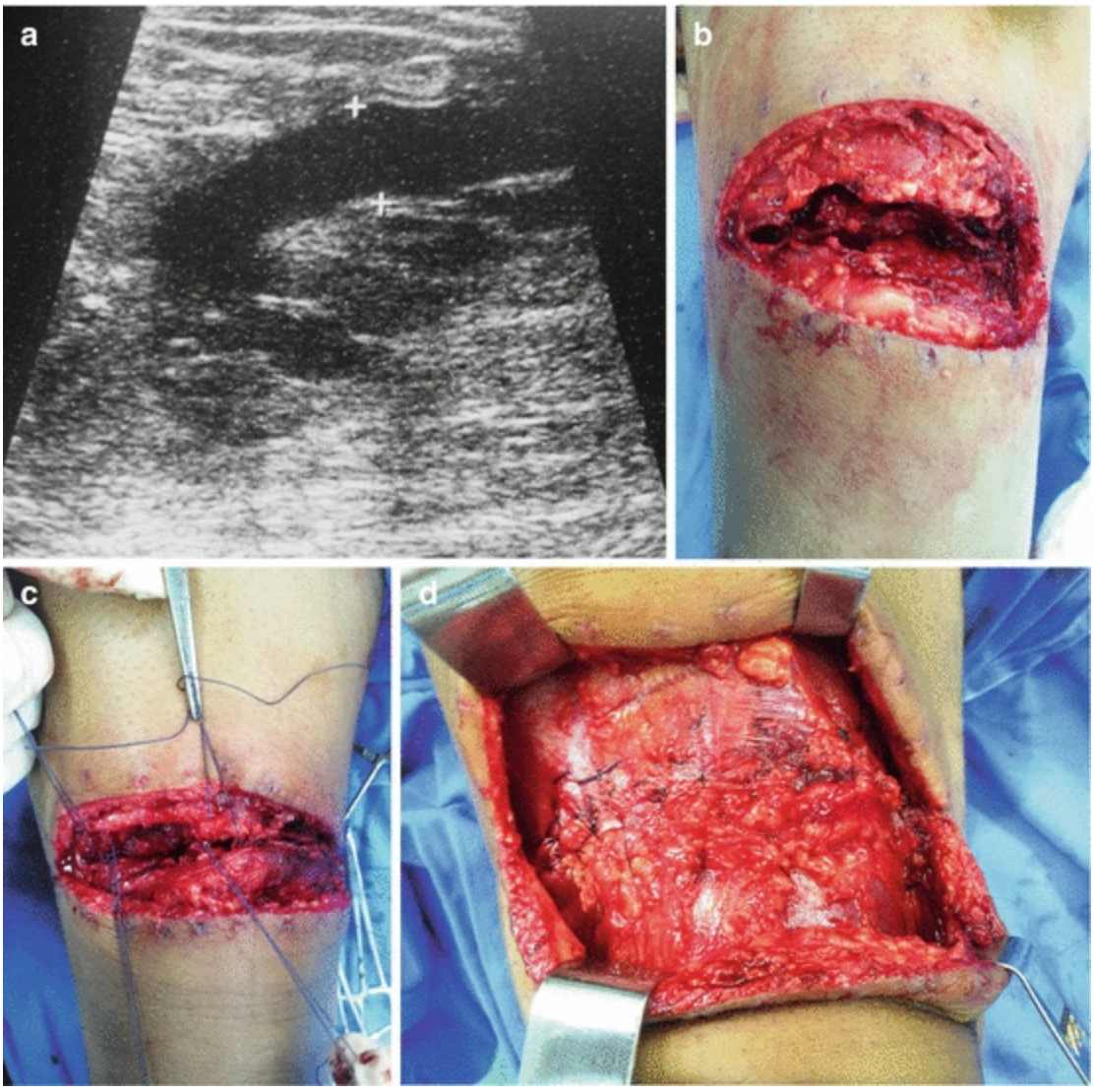

Fig. 7.1 (**a**) Transversal ultrasound image shows a complete rupture of the quadriceps muscle. (**b**) Intraoperative view of the quadriceps lesion. (**c**) The lesion after sutures. (**d**) Final aspect of the suture

Partial tears with an avulsion involving one or two tendons with greater than 2 cm retraction, or a musculotendinous avulsion injury to the direct head of the rectus femoris that is symptomatic for more than three months should be considered for an operative management, depending on the severity of symptoms, or functional limitations in patients unwilling or unable to modify their activity levels [15].

7.4 Return to Play and Prevention

Reconditioning the injured muscle group is mandatory. Return to play should be based on the patient's ability to stretch the injured muscle as much as the contralateral healthy muscle, pain-free use of the injured muscle in sport-specific movements (mainly in eccentric exercise), comparable strength between injured and healthy muscles and

functional tests [16].

There are strategies that can be adopted to minimize the recurrence rate and to enhance performance.

A. An appropriate warm-up is protective because it increases range of motion and reduces stiffness. Warm up should occur before any exercise session or sports activity, including practice. This will help increase speed and endurance [17].

B. Avoid muscle tightness by stretching slowly and gradually, holding each stretch to give the muscle time to respond and lengthen.

C. A regular program of strengthening exercises should be instituted.

D. Recovery of strength and muscular endurance should focus on the transitional period due to inactivity and deconditioning. The relative state of fatigue may be increased making the muscles more susceptible to injury. Training with low levels of resistance and many repetitions should be recommended [4, 18, 19].

7.5 Future Therapies

Novel treatments for muscle healing—such as the use of growth factors and cytokines—are still under investigation in Europe and have not been approved by the U.S. Food and Drug Administration (FDA). Hyperthermia is emerging as an effective treatment for some injuries. Rapid, precise application of heat at the injured site improves and accelerates the healing process. It stimulates muscle precursor cells and protein synthesis.

Gene and cellular therapy appear to be the two main future therapies for muscle injury treatment. Gene therapy is the addition of genes into cells with the aim of restoring function, establishing new cells or even enhancing already existing activities through the expression of introduced genes [20]. Thus, gene therapy can be applied to most diseases, whether genetic or acquired. The therapeutic gene is introduced into the patient in two ways: in vivo or ex vivo. The choice of one type or another depends on a number of factors such as the molecular pathology of the disease, the target cell, the size and type of the therapeutic gene and the duration of the therapy [20]. The system used for transferring therapeutic genes into the target cell is called a vector which can be a viral-based structure or a plasmid [20].

A major obstacle to the application of gene therapy in clinical practice is the construction of suitable vectors to ensure patient safety and a high transfection rate to

allow for a sufficient level of expression of the therapeutic gene for the treatment of the disease. The plasmid vectors are superior to the viral system in terms of biosecurity, but are generally inferior in terms of efficiency of gene transfer [21]. Because there were deaths associated with the use of viral vectors, research has turned to the development of new systems of non-viral gene transfer [21]. Electroporation [22], sonoporation [23] use of lipoplexes [24], polyplexes [25], transfection by hydrodynamics [26] and nucleofection [27] are examples of non-viral gene transfer that are efficient and comparable to the viral systems. As currently available gene transfer systems permit the transfer of most genes with variable sizes, many diseases can be treated by gene therapy [21].

There have been few studies involving gene therapy and muscle injuries. The plasmid is the vector of choice due to ease of manipulation, and gene expression for only a short period of time, lower cost of production on a large scale and safer drug formulation [20]. Many modifications can be easily performed on plasmid vectors to enhance gene expression and ease gene transfer [20].

Most studies use growth factors to enhance angiogenesis and myogenesis. Piccioni et al. [28] have shown that the Shh gene (Sonic Hedgehog) was able to activate the expression of myogenic and angiogenic factors thus increasing the capacity of muscle regeneration after injury. The IGF-1 (insulin-like growth factor) gene has been used to treat muscle injuries. It was effective in regenerating muscle fibers and was able to attract bone marrow stem cells to the injury site and, thus, accelerate tissue repair [29, 30]. Arsic et al. [31] constructed an adenovirus expressing VEGF (vascular endothelial growth factor) and injected it five days after the injury; the treated group showed better and faster muscle regeneration [31]. Vectors and genes that allow faster and improved recovery of injured tissue are the objectives of gene therapy for the treatment of muscle injuries. Generally, the use of a vector expressing a factor is much cheaper than the injection of the protein itself. And the effect lasts longer.

Cell therapy involves the use of cells for treatment. In the case of muscle damage, the cells that have been used more frequently in preclinical trials are mesenchymal stem cells (MSC) and muscle-derived stem cells (MDSC).

MSCs are non-hematopoietic multipotent stem cells that adhere to culture plates [32]. MSCs have the ability to renew and differentiate into multiple lineages of connective tissue, including bone, fat, cartilage, tendon, muscle, and bone marrow stromal cells [32]. These cells were first described by Friedenstein who found that MSCs adhere to culture plates, resemble fibroblasts in vitro, and form colonies [33]. MSCs are present in all adult tissues and in the wall of fetal tissue vessels as part of the pericyte population [34]. In vivo, MSCs are identified by expressing CD146 and CD271, and within the adipose tissue as part as the CD34 positive population [35]. A very rich source of MSC is the adipose tissue and these cells are called ADSCs (adipose-derived stem cells). Peçanha et al. [36] showed that the ADSCs were able to

accelerate muscle recovery in treated animals.

The MDSC are multipotent stem cells that were isolated from mouse skeletal muscle and are obtained through a series of steps of plating on collagen plates [37, 38]. The MDSC have a high expression of Sca-1, very low levels of viementin (a fibroblast marker), low levels of desmin and other markers of muscle differentiation [38]. Ota et al. [39] showed that injection of MDSCs four days after injury increased angiogenesis and reduced scar tissue. This group found high levels of VEGF one week after injection. In addition, MDSCs expressed high levels of antioxidant and GSH (glutathione) and superoxide dismutase, allowing greater survival of these cells after injection [40, 41].

Currently the major concern is to retain these living cells at the injury site so that they can have a prolonged effect; many researchers have turned their attention to this problem. Distefano et al. [42] used electrical stimulus and the cells remained longer at the site of injury. Park et al. [43] used losartan, an antihypertensive, and achieved a greater reduction in fibrosis following injury.

7.6 Conclusion

Treatment of muscle injuries depends on knowledge of the cause and nature of the injury. Treatment can be conservative or surgical. Gene and cell therapies are promising but much more research is need before we can proceed to a clinical protocol that is safe and efficient. The combination of the properties of stem cells with growth factors such as VEGF and IGF may, in the future, provide faster recovery from muscle injuries.

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8. Therapeutic Alternatives: Principles and Results

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Abstract
Muscle injuries are the most common traumas occurring during sports practice. The heterogeneity of the severity of these injuries probably explains the lack of clinical studies on their treatment. Accordingly, treatment principles are derived from empirical tests or from studies with low levels of proof. The RICE protocol (rest, ice, compression and elevation) is universally recognized. The aim is to fight pain and bleeding immediately after injury and during the first 5 days after the initial trauma. Mobilization of the muscle is recommended as soon as possible when pain at rest has disappeared. Muscle strengthening and stretching should be carried out gradually to induce remobilization without re-rupture and to allow return to daily activities. Rehabilitation programs, based on progressive eccentric, agility and trunk stabilization exercises help to organize the muscle scar and to recover full muscle strength and flexibility. Retraining programs can be built around the specific practices of the sport when no pain is present during muscle contraction and stretching. The duration of these phases depends on the type of muscle injury and the individual possibility of scaring. Evaluations using an isokinetic dynamometer or testing on the field can be used to follow and manage treatment before recovery and return to competition. Several therapeutic alternatives are presented to decrease the duration of the muscle treatment but always need to be evaluated in studies with good methodology.

8.1 Introduction

Treatment of musculoskeletal injuries is a real challenge. The complexity of the muscle anatomy and the variety of muscle injuries explain why their treatment is still empirical and relies on individualized strategies. The amount of time out of competition is difficult to predict in spite of the contributions of ultrasound, magnetic resonance imaging and muscle strength evaluation by isokinetic dynamometer [10, 11, 13, 14]. That is why the risk of re-injury is high, a third of all hamstring injuries in soccer players [27]. The presence of a previous injury is the most powerful predictive risk factor of the occurrence of a muscle injury [6]. To better understand the treatment of muscle injuries, we analyse the various principles and therapeutic results in terms of efficiency.

8.2 Method

In the field, the use and efficacy of the various treatments of musculoskeletal injuries is based on common clinical sense, empirical knowledge and the therapist's experience. This approach although interesting is insufficient to allow any scientific generalization. Analysis of the literature, based on a search of MEDLINE from 1966 to 2013, allows a stronger approach but is limited by the lack of double-blind randomized studies. Some useful information can still be gleaned from studies with less than ideal methodologies. All in all, 16 references were found with the best level of proof, although only four were of real significance because of their methodological quality [7, 25, 34, 35].

8.3 Results

8.3.1 The Acute Phase of Muscle Injury

The RICE protocol (rest, ice, compression and elevation) is an empirical, pragmatic approach with an efficacy unanimously accepted in spite of the lack of scientific validation [4]. The optimal duration of ice application is not well known but is generally proposed as 15–20 min every 6 h during the first 3–5 days, according to personalized protocols [5]. The therapeutic purpose is to limit bleeding by vasoconstriction, and to fight swelling and pain by inhibiting the nerve receptors and sensory conduction. However, the cold increases bleeding by changing coagulation and hemostasis while compression would be more effective to reduce the intramuscular blood flow into the injured area [19] (Fig. 8.1). From memory, no study has shown the effect of cold on muscle injuries in humans. It seems nevertheless logical to think that the efficiency of the cold decreases with the depth of the injury [5]. Elevating the injured limb above the level of the heart results in a decrease in hydrostatic pressure

and, subsequently, reduces the accumulation of interstitial fluid. Complete cessation of participation in sport is essential because rest avoids aggravating the injury by reducing the size of the hematoma and, subsequently the size of the connective tissue scar. Using crutches to walk without putting pressure on the injured leg may be necessary, as judged by the level of pain. Such support should be withdrawn as soon as possible, again according to the amount of pain.

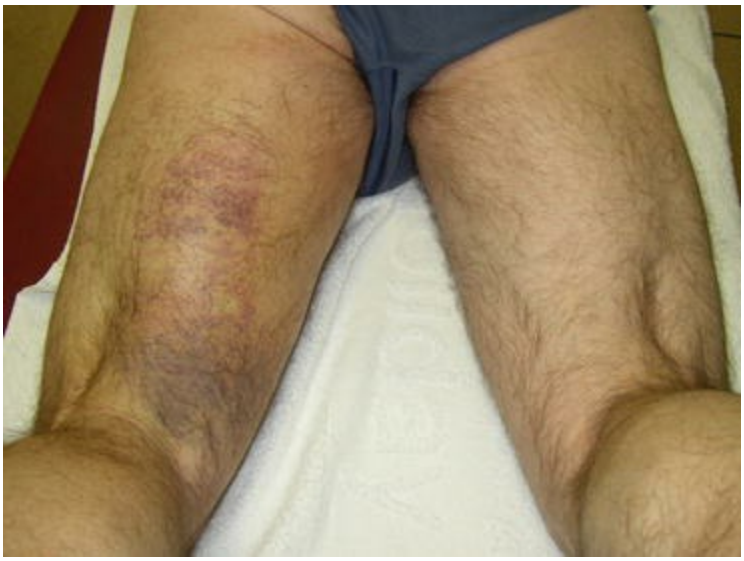

Fig. 8.1 Bleeding after hamstring (biceps femoris) injury

8.3.2 The Rehabilitation Phase

8.3.2.1 Musculoskeletal Exercises

Early active mobilization has been recommended since 1953 according to Woodard, and should start as soon as spontaneous motion is painless [20, 21]. It must be progressive with validation step by step. From a more analytical point of view, eccentric exercises should be started as soon as walking is painless, to allow the muscle to contract as well as to stretch. The myofibers and their conjunctive envelopes should heal according to lines of force connected to these drives. These exercises are usually progressive, carried out according to internal, and then average and external muscle course, that is with a muscle length always higher. It is recommended that these exercises begin slowly, at 10° per second for example, increasing to 30° per second with submaximal muscle recruitment to remain pain free. This type of rehabilitation is more or less easy to perform according to the site of the muscle injury. To control the joint movement, eccentric exercises could be started manually or with an isokinetic dynamometer [17] (Fig. 8.2). However, compensation by the muscle heads is possible in cases of poly-joint muscle injury such as injury to the hamstring. For example, the biceps femoris is easily recruited during knee flexion and can compensate for the

injured internal rotator muscles of the other knee.

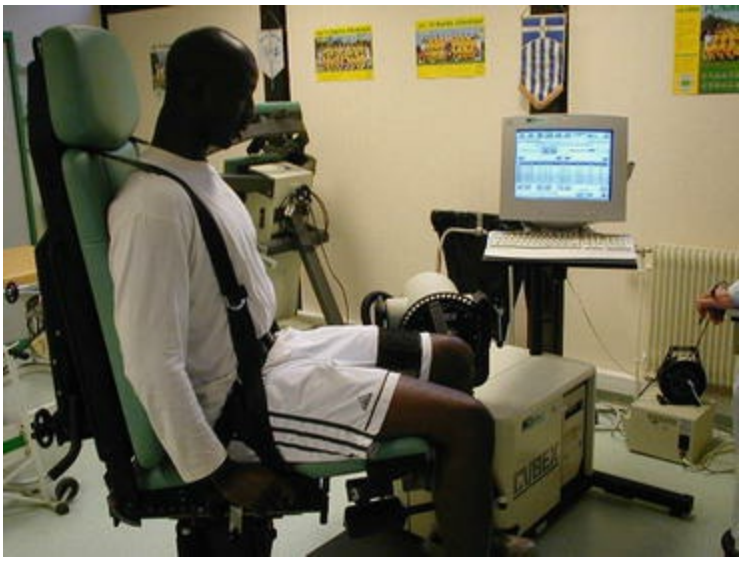

Fig. 8.2 Isokinetic muscle strength assessment

The hamstring muscles have been studied in particular because the muscle is frequently injured. The four solid methodological studies mentioned previously allow some certainty with regard to stretching, trunk stabilization, eccentric exercises associated with running and the lack of efficacy of the sacroiliac manipulations [26].

Stretching four times a day for 30 s is more effective than stretching only once a day. A study of two groups of 40 randomized injured subjects, found that those who stretched four times a day returned to play 1.8 days earlier, on average, than those who stretched once a day [25].

Agility and trunk stabilization using isometric trunk training and one-limb balance exercises allows better results in terms of protection against re-injury and rapid return to sport activity (22 days ± 8 against 37 days ± 25) compared with stretching and strengthening of the injured hamstring muscle [34]. However, that study included only 12 subjects. More recently, this type of exercise was blind compared with eccentric strengthening associated with running in 51 randomized patients. No difference was found between the two types of rehabilitation in terms of return to play which averaged between 25 and 28 days [36]. At return to play, there were still signs of trauma visible on MRI, even though most of the patients were clinically pain-free. Four subjects were reinjured during the 12 month follow-up, with two of them at the very end of their rehabilitation period.

Passive stretching with sacroiliac manipulation is not effective [7]. However, the results makes sense when the two groups, treated and control, are compared before treatment. In fact, a meta-analysis on this subject by Mason et al. summarizes very well the misunderstanding of treatment of muscle injuries [26]. Only some parameters are

studied: range of motion of the joint, muscle strength or re-injury. There is no single definition of time to return to play, despite harmonization plans proposed by various sports federations. Certain interesting parameters have been little studied: pain, muscular atrophy, or patient satisfaction. The number and the duration of treatments remain empirical while the effect of simple rest has never been studied.

8.3.2.2 Physiotherapy and Massages

Therapeutic ultrasound produces vibrations and heat, which aggravate the injury during the acute phase, and thus it is used only at the end of the treatment to decrease pain and tenderness while the collagen tissues heal. The efficacy of treatment has not been validated in humans or animals [32].

Electrical muscle stimulation is the application of an electrical current to the skin to provoke a muscle contraction. The myofibers are recruited, however, contrary to the physiological process. The fibers contract directly and locally under electrodes according to the quantity of current delivered, with no possibility of voluntary control of the contractions. Temporal and spatial recruitment are not respected, and there is some risk of worsening the injury during its acute phase.

Massage is mechanical palpation of the skin to exert varying pressure on the muscle according to the spatial orientation of the muscle. This treatment can aggravate the injury during the acute phase by manipulating the site of pain. Any link with between massage and muscular calcification, however, has not been proved [2].

8.3.3 The Retraining Phase

The objective of this phase is to return the injured subject to the same competitive level while avoiding re-injury (Fig. 8.3). To reach this goal, the concept of analytical symmetry was developed to reduce and eliminate pain [9]. Pain is estimated during maximal muscle stretching, maximal resisted muscle strengthening and during palpation of the injured muscle. No pain and tenderness can be present during daily activities. Bike riding can begin when walking is painless and symmetric. Footing is deemed to be recovered when stairs can be ascended and descended without pain. Increasing the intensity of running depends on the degree of muscle tenderness, as recommended for any muscle injury at the level of the lower limb [16, 35]. These activities maintain cardiovascular fitness.

Fig. 8.3 The retraining phase in the field

From a clinical point of view, muscle symmetry is evaluated on recovery of the range of motion of the joint, and the elasticity and strength of the muscle, measured with an isokinetic dynamometer if possible. To return to competition, the difference in concentric and eccentric strength between the injured and the healthy side must be less than 10 %, except for any expected after-effects (Fig. 8.4). By identifying and calculating the degree of imbalance between agonist and opposing muscles, injuries could be reduced by three fourths [8].

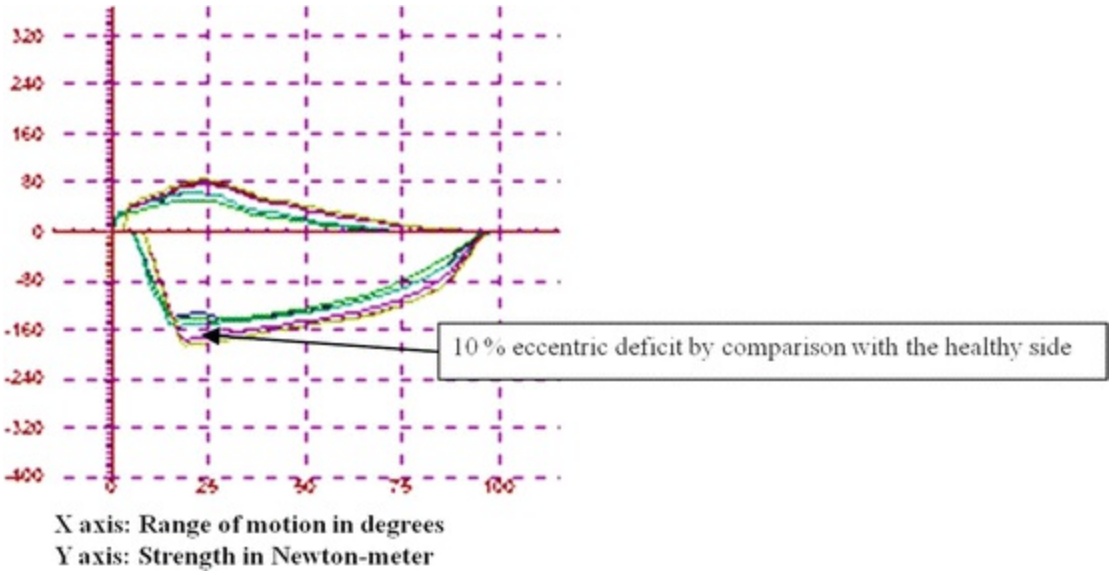

Fig. 8.4 A football player with an eccentric isokinetic strength deficit 2 months after a grade II hamstring injury

From a more global point of view, the return to the playing field also depends on training. A period of retraining is inescapable after several weeks of reduced activity while the injury heals [29, 36]. Merely ceasing all activity, without injury, will cause a 1 % loss of strength per week and the loss of aerobic capacity is 10 % per week until the sedentary level is reached. This sedentary level is often underestimated in high-level athletes who have practiced their sport since adolescence or childhood.

8.3.4 Therapeutic Alternatives and Future Treatments

Several treatments have been tried or are the subjects of trials with the aim of early return to elite competition. Time lost to play is a significant financial cost for professional clubs [12]. In the absence of strong level I studies, these treatments are considered empirical. Time will tell whether the promising results already described will be proved scientifically.

8.3.4.1 Anti-inflammatory

Nonsteroidal anti-inflammatory drugs (NSAIDs) are sometimes proposed for temporary relief during the acute phase to limit the inflammatory cell reaction. No adverse effects on the healing process or on tensile strength have been shown. However, NSAIDs should not be used during the first 3–5 days after injury because they delay the phagocytosis phase of necrosis. On the other hand, the long-term cures showed negative effects on muscle regeneration [28].

Corticoids must not be used because they decrease muscle regeneration and prolong the healing process [2]. Intramuscular corticosteroid injection decreases pain but the association with an anesthetic drug masks any analgesic role they may play. There is no proof that they shorten the duration of healing in the absence of any controlled studies and despite a retrospective study of 431 (American) football players [24].

8.3.4.2 Hyperbaric Oxygen Therapy [3]

This technique was developed from animal experiments with the aim of favoring soft tissue healing by providing additional oxygen. The treatment places the injured subject in a compression chamber with 100 % oxygen 45 min twice a day for 5 days. In humans, this process has been used in several therapeutic indications: ankle and medial collateral knee sprain or prevention of delayed onset muscle soreness after eccentric exercise. At present, the utility of hyperbaric oxygen therapy for treating muscle injuries has not been studied. Nevertheless, this treatment has already been proposed for treating injuries in professional soccer players for example. The limits of this treatment are its cost and limited accessibility.

8.3.4.3 Autologous Serum, Actovegin and Traumeel®

Several animal studies on tissue regeneration have reported interesting results when they injected autologous conditioned serum at the level of the muscle injury. Injured subjects who were treated with autologous serum returned to play 6 days sooner, on average, than those who were treated with Traumeel®: 16 days versus 22 days [37]. Traumeel® was then considered as a homeopathic mixture taken as placebo. Numerous study biases were present. The two groups were of different size, 18 in the group treated by autologous serum and 11 in the Traumeel® group. Muscle injuries were not homogenous with an abdominal muscle injury in the treated group that was cured in 8 days, which significantly improved the results.

A conflict of interest was inherent in the use of Traumeel® as the study was sponsored by the pharmaceutical company that marketed the drug [33]. In some studies, Traumeel® was considered as an NSAID, in terms of reducing symptoms of inflammation and improving mobility with a favorable safety profile; other studies treated it as a placebo [37].

Actovegin is a protein-free derivative of calf's blood. It is used via intra-muscular or -vascular injection to accelerate healing. In recent years, Actovegin was forbidden by the World Anti-Doping Agency because it improves cellular oxygen transport and elite cyclists were using it to enhance their performance. Users were required to wait 8 days before returning to practice and the ban was widely advertised [23].

8.3.4.4 Platelet Rich Plasmas

It is thought that this treatment induces growth factors such as IGF1 and VEGF by injecting the plasma directly into the muscle injury [1]. At least five different manufacturing processes have been described and several injured athletes have already been treated according to open methodological studies [22]. Only two controlled randomized studies exist about muscle injuries but they are in rats and mice [30]. A decrease in the duration of recovery from 21 to 14 days was shown, as determined by measurement of muscle strength and histological analysis of tissue regeneration. Whether these results can be applied to humans has not been confirmed, although several pilot studies are in progress with results not yet published [15]. Numerous questions remain about the manufacture of platelet rich plasma–which platelet concentration, which platelet activation, association with white blood cells or normalization to the pH and so on; and about the therapeutic strategy–Which grade of muscle injury to inject, what is the optimal timing of injections, how many injections, is anesthetic preinjection necessary, what are the adverse effects on the muscle and for the patient and on [30].

8.3.4.5 The Future and the Current Experiments [18]

The American team of Huard et al. published several animal studies with the aim of regenerating muscle by inhibiting muscle fibrosis. It was suggested that inhibiting the myostatine expression or blocking TGF β-1 action by using γ interferon, suramin or still losartan. In spite of good results, the research was not pursued, to our knowledge, because of the potential adverse effects in humans. It was then thought to develop muscle-derived stem cells to produce scar tissue rather than healthy skeletal muscle fibers [18, 31]. These exploratory treatments may enable the use of muscle stem cell-based gene therapy and tissue engineering to improve tissue regeneration.

8.4 Conclusion

In the absence of robust scientific study of the treatment of muscle injuries, it is nonetheless possible to propose a therapeutic regimen. After an injury, the objective is to avoid hematoma and especially to moderate the effects of the injury with the RICE protocol, which is a basic and pragmatic treatment. Nonsteroidal anti-inflammatories, if indicated, could be proposed a few days after the destruction phase of the injury. The alternative therapeutics are to use on second rank by carefully weighing the indications if attempting to decrease the time to return to play without risking re-injury. When spontaneous muscle pain has disappeared, the rehabilitation phase can begin to encourage organized tissue healing. Mobilization of the muscles can be accomplished through walking, climbing stairs and cycle riding which are well supported physical activities for this purpose. Recovery is based on exercise to regain muscle elasticity and strength in infra-painful according to components under maximal eccentric contraction at first. In the absence of pain, after complete recovery of the joint range of motion, the retraining phase can begin to regain muscle capacity in terms of endurance, strength, speed and sport-specific agility. Return to competition can be advised after recovery of muscle symmetry and aerobic activities. The progressive validation of the stages, sprint, jump etc. is best established with tests on the field. The duration of the time away from activity is thus connected to the restoration of muscle function (strength-speed-elasticity), which occurs thanks to sufficient healing of the histological components of contractile structures and muscle soft tissues.

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Part II
Non-traumatic Muscle Injury

Bernard Roger, Ali Guermazi and Abdalla Skaf (eds.), Muscle Injuries in Sport Athletes, Sports and Traumatology,
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9. Non Traumatic Muscular Injury

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Abstract
Nontraumatic muscular injuries are defined by the fact that they occur without a clear direct or indirect traumatic event. Concurrently to the latter and despite including a wide spectrum of diseases, nontraumatic injuries often share the common features of insidious onset and a clinically unclear origin. In this regard imaging may play, despite its varying accuracy, a significant role in confirming the presence of a muscular suffering and/or the presence of an anatomic variant that may become painful in itself or responsible for an impingement with neighboring structures such as other muscles, nerves or vessels.

9.1 Introduction

There is ample epidemiologic evidence that high levels of physical activity are associated with specific overuse syndromes. Exercise can reveal a pre-existing abnormality or induce specific lesions.

Pain is the cardinal symptom. Abnormal fatigue upon exertion, dysesthesia, paresthesia, are less common. All symptoms have a purely mechanical time pattern. The diagnosis requires careful elimination of non-exertion-induced conditions e.g., rheumatic, neurologic, endocrine, vascular and drug-induced disorders [1].

9.1.1 Myopathies Revealed by Exercise

In some cases the presence of myalgia can reveal muscular disease. A rigorous clinical examination is necessary to justify the request for complementary examinations (frequently leading to muscular biopsy) and often necessary to confirm the diagnosis. The major complication, rhabdomyolysis, must be precisely evaluated in regard to aptitude, partial or total inaptitude to sport.

Muscular diseases revealed by physical exercise are most frequently related to metabolic myopathies. The majority of these hereditary diseases are often revealed in young babies, but in some cases are detected only in the young adult and, generally, in respect to sports activities.

It should be simple to eliminate the diagnosis of classical cramp, which most often occurs after prolonged exercise of unusual intensity. This type of cramp is localized, unilateral, and yields in a few minutes after exercise.

Metabolic myopathies, conversely, are generally expressed by localized pains (but very often symmetrical) even diffuse, accompanied or not by an impression of muscular weakness. We observe a gradual decrease of performance, accompanied sometimes by myoglobinuria. It seems likely that exercise may be useful in these pathologies, because it may affect the disease itself [2].

McArdle disease (deficient phosphorylase) and Tarui disease (deficient phosphofructokinase) are revealed by fatigue and cramping during exercise, particularly with intense and short exercises. Performance decreases very gradually. CPK is low or not high, with inconstant episodes of myoglobinuria. In all cases there poor production of lactic acid during exercise tests on a cycle ergometer [3].

Myoadenylate deaminase deficiency is less common [4] but can also be revealed by clinical symptoms of exercise intolerance with elevated serum creatine kinase. (Myoadenylate deaminase, the enzyme in the purine cycle, catalyzes the deamination of adenosine monophosphate into inosine monophosphate [5]) The enzymatic deficit involves a disturbance of the cycle of purins and is expressed by a lesser increase in ammonia. Exercise tests on a cycle ergometer can reveal a dramatic increase of ammonia.

Carnitine palmitoyltransferase II deficiency [6] is expressed by myalgias and muscular weakness after prolonged exercises. It is frequently accompanied by myoglobinuria and elevated levels of CPK. The deficit induces a reduction in the transfer capacities and mitochondrial fatty acid oxidation.

Mitochondrial myopathies can be confirmed by muscle biopsy with enzymology testing and by assessing reaction activity of the respiratory chain complexes, including complex I (NADH ubiquinone oxidoreductase), complex II (succinate-ubiquinone reductase), complex III (ubiquinol-cytochrome c oxidoreductase), complex IV (cytochrome c oxidase), and citrate synthase activity, In these cases exercise tests on a cycle ergometer or treadmill can reveal early production of lactic acid [7].

The use of MRI in the field of metabolic myopathies has been scarcely studied in the

literature. A few reports have emphasized the presence of a progressive muscular fatty replacement without significant muscular atrophy in McArdle disease [8]. This finding often contrasts with the low disability induced by the disease. Concurrently, other studies have demonstrated a significantly lower increase in T2 signal in muscles in patients with metabolic myopathies than in healthy subjects [9]. Despite its reported high sensitivity, this technique is not widely used in clinical practice.

9.1.2 Exercise-Induced Muscle Damage

Exercise-induced muscle damage can be caused by unaccustomed exercise, with muscle soreness, swelling and decreasing force production. This discomfort is exacerbated when exercises include an eccentric component.

Eccentric work is characterized by an elongation of the muscle during simultaneous contraction. It produces more hypertrophy compared to concentric contractions with less energy cost. Investigations have demonstrated myofibrillar disruption in particular within fast-twitch fibers. Discomfort increases within the first hours after exercise, with a peak between 1–3 days and disappears after 5 or 6 days. This phenomenon is referred to as delayed onset muscle soreness (DOMS) [10].

Despite substantial research, the mechanism of damage remains unclear. A number of theories have been proposed: muscle spasm, connective tissue damage, muscle damage, local inflammation. A general consensus is that DOMS cannot been explained by a single theory. We must take into account the high tensile forces produced by eccentric exercise, concomitant damage to the sarcolemma, local inflammation, accumulation of potassium, histamine and kinins, and increased intramuscular pressure which creates a mechanical stimulus for pain receptors [11].

9.2 Clinical Examination

DOMS is classified as a type I muscle strain injury. Tenderness is localized in the distal portion of the muscle (in particular the quadriceps, hamstring and triceps surae).

On clinical examination, palpation of the muscle is painful. Isometric testing and stretching are painful and a strength deficit can be observed. In all cases, sometime in the first 7 days blood samples show a dramatic increase of muscular enzymes such as the CK or LDH (but also myoglobin) and metabolites of conjunctive degradation (in particular hydroxyproline and hydroxylysine).

9.3 Imaging

Short term imaging confirms the presence of edematous changes seen on both MRI and ultrasound (Fig. 9.1).

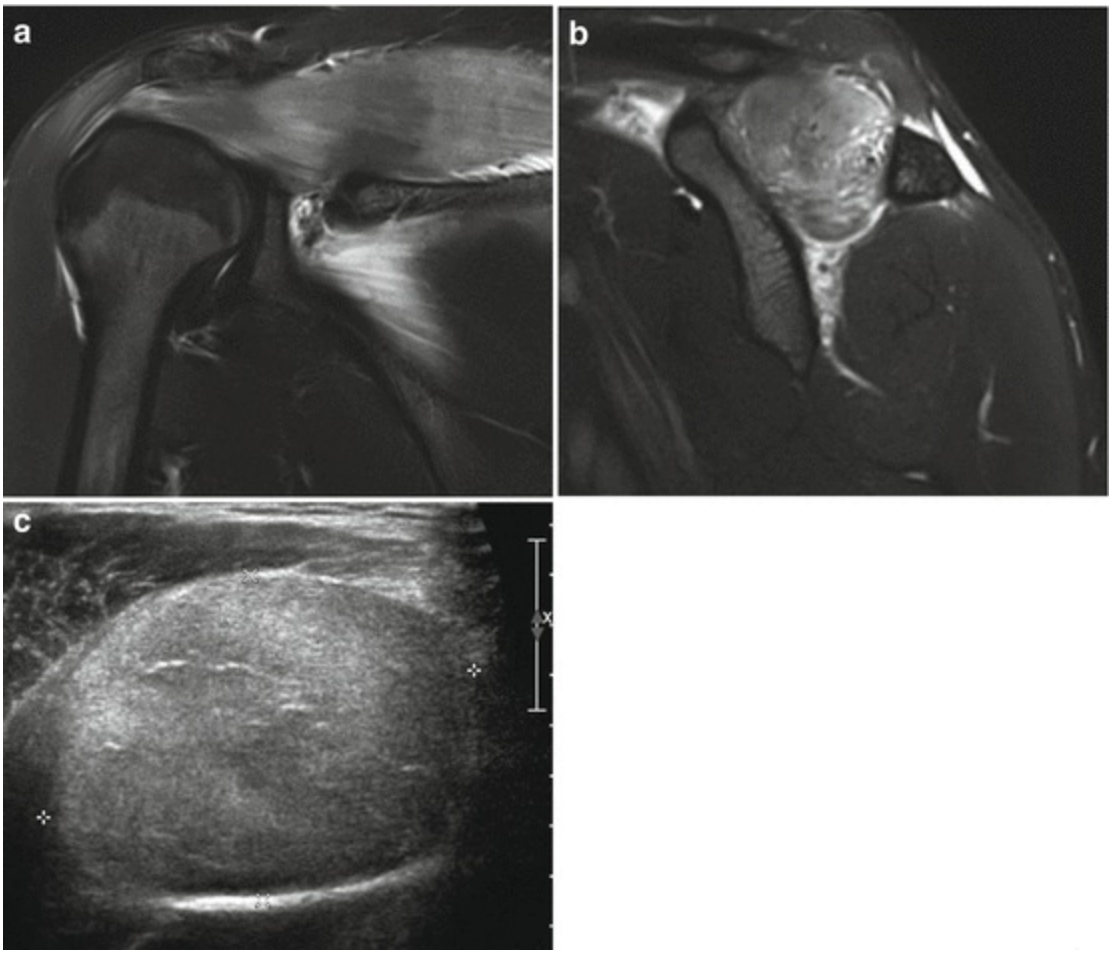

Fig. 9.1 A 20-year-old male patient with overexertion of the upper-arms during exercise in the early afternoon. After a delay of 5 h, the patients reported rapidly progressive pain and disability of the right shoulder. Coronal (**a**) and sagittal (**b**) fat-suppressed T2-weighted MRI performed the following day confirm the presence of edema and swelling of the supraspinatus muscle typical of a DOMS syndrome. Transverse ultrasound image (**c**) confirms both swelling and hyperechoic edema of the muscle

9.4 Treatment and Prevention

At present, various treatments like cryotherapy, stretching, massages, compression, anti-inflammatory drugs, antioxidants, homeopathy, and physiotherapy, have been proposed but with very little evidence for their efficacy [12].

9.4.1 Muscular Lesions

Apart from extrinsic and intrinsic muscular lesions, two types of clinical syndromes are often revealed by physical exertion: chronic compartment syndromes and accessory muscle syndromes.

9.4.1.1 Chronic Compartment Syndromes

Since the first reports in 1975 [13], the number of cases of chronic compartment syndrome due to prolonged exercise – running, hiking, roller skating, motocross, wind surfing [14] – has dramatically increased.

An increase in intratissue pressure within a compartment enclosed in an unyielding fascia defines the condition. Ischemia can be the ultimate result. There is normally an intricate balance between capillary pressure, intratissue pressure, venous pressure, and osmotic protein pressure. Muscular contraction is normally associated with an inflow of water into the muscle. If the compartment is abnormally tight or the contraction excessively intense, the pressure within the muscle rises to abnormally high values during relaxation, causing symptoms. This is probably an oversimplification of the pathophysiology of chronic compartment syndrome. In a muscle biopsy study, Wallensten [15] found that patients had higher percentages of slow fibers with a high oxydative potential, hypotrophy of the same fibers, and decreased production of lactic acid, compared with the controls. However, these abnormalities could be either causes or consequences of the compartment syndrome. The most frequent compartment syndromes concern the lower leg, divided into three muscular compartments, one antero-lateral, one deep posterior, and one superficial posterior [16]. Involvement of the antero-lateral compartment is common in all published studies, whereas involvement of the superficial posterior compartment is rare and consistently accompanied with involvement of the deep posterior compartment. But all compartments can be concerned, in particular the forearm and the foot.

Pain is the main symptom. It is usually confined to the involved compartment and initially occurs at a given level of activity, although the interval to occurrence of pain tends to decrease over time. Fullness of the compartment upon palpation is common. These manifestations resolve completely within an average of ten minutes with rest.

Patients with antero-lateral compartment syndrome sometimes have one or several muscular herniations, transient foot-drop, or sensory loss in the territory of the superficial fibular nerve.

Intramuscular pressures at rest and at cessation of the activity that induces the symptoms should be measured to confirm the diagnosis. The measurement technique was first described by Whitesides [14]. A number of devices for intramuscular pressure measurement are now available, including the Stic Catheter (Stryker Instruments) that provides fast, highly reproducible results. Pressures do not normally exceed 15 mmHg at rest and 30 mmHg (25 mmHg for forearm) after exertion. While patients with chronic compartment syndrome often have normal basal values, their values after exertion are abnormally high and return to normal levels only after several minutes.

Because intramuscular pressure measurement remains an invasive procedure, MRI has been proposed as an alternative for diagnosis. Numerous studies have shown an increase in T1 and T2 relaxation time in muscles involved in compartment syndrome [17–21]. In a study of 13 military recruits, Eskelin has shown an excellent correlation

between the increase of intracompartmental pressures and MR signal intensity on T2-weighted images at rest and after standard dynamic exercise on a treadmill (Fig. 9.2) [17]. This technique requires, however, the use of parameters that are sensitive to muscle water content. As absolute values of signal intensity may be hindered by technical factors such as field inhomogeneity, the author suggests better accuracy from normalized values using ratios of T2 signal intensity compared to subcutaneous fat, tibial bone marrow or superficial posterior compartment muscles. As an example, when tibial bone marrow was used as the normalization tissue, the increase in T2 signal measurement was 24 % ±2 in the control group and 98 % in symptomatic patients with more than 40 mmHg pressure on manometry [17]. Similarly, with the use of fasciotomy as the gold standard, Verleisdonk et al. have shown an increase in the signal intensity ratio between anterior and posterior superficial compartments of 21 % (12.2–32 %) after exercise, with values of 3.9 % and −1.8 % in the control and post fasciotomy groups, respectively [18]. More recently, Litwiller et al. have proposed improving the technique by promoting an in-scanner exercise added to a novel dual birdcage coil. An in-scanner exercise device means the patient can exercise and stress the muscles without getting in and out of the machine [19]. This technique drastically shortens the delay between exercise and acquisition of images, thus limiting the risk of false negatives since signal modifications related to exercise are known to fade in a few minutes. Concurrently, the use of a dedicated dual birdcage allows reduced field inhomogeneity and increases signal to noise ratio. In that study, a threshold of 1.54 for the ratio of relative T2-weighted signal intensity increase compared to baseline offered a sensitivity of 96 %, specificity of 90 % and accuracy of 96 % [19, 20]. Development of such a technique may nevertheless be limited to medical care structures with a high volume of patients with chronic compartment syndromes.

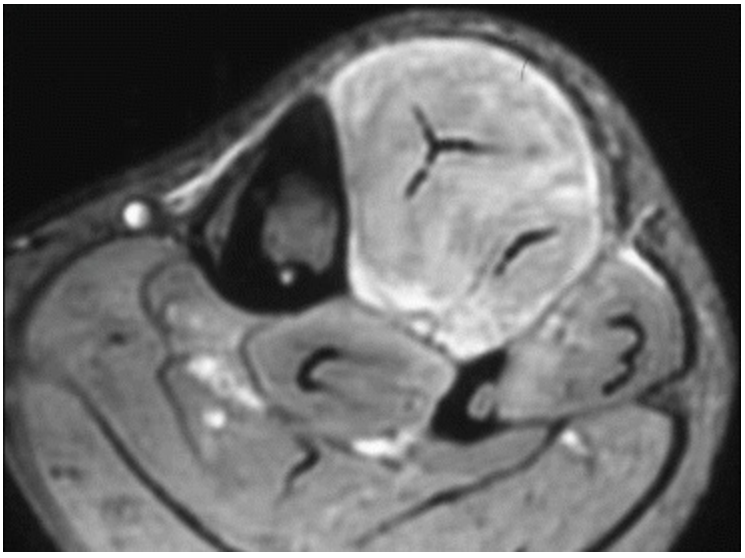

Fig. 9.2 A 32-year-old patient with compartment syndrome of the left leg. Axial fat-suppressed T2-weighted MRI of the left leg after running on a treadmill shows hyperintensity of the muscle in the anterior compartment of the leg

Compartment syndromes in the forearm were studied in a population of motocross racers. That study, using MRI, found the flexor digitorum superficialis and profundus to be predominantly involved [21].

Rare acute exertional compartment syndromes have been reported in the literature [22, 23]. In an emergency, MRI may demonstrate edema in all of the fibers of the involved muscle, thus confirming the diagnosis in the absence of any reported trauma [22]. In a case that required urgent surgery of the adductor muscles, signal intensity was found to be normal four months postoperatively while the normal fiber structure required additional months to return to normal [23].

Surgical fasciotomy is the only way to resolve symptoms and avoid progression towards acute compartment syndrome. Rorabeck [24] has described the surgical procedure for each compartment. Patients should refrain from athletic activities for one month after the procedure. Most can resume activities at the presurgical level at the end of the second postoperative month.

9.4.1.2 Variant Muscles

Variant muscles include accessory muscles, hypertrophy of normal muscles, abnormal trajectory of muscles and abnormal organization in transverse or longitudinal planes of muscles.

An *accessory muscle* is a supernumerary structure that shares some similarities, especially of trajectory, with a normally existing one from which it usually inherits its name. While the frequent presence of accessory muscles has long been reported in anatomic studies [23], exertion-related leg pain caused by these muscles is a recently and infrequently identified condition. Awareness of this syndrome is important to avoid errors in diagnosis and treatment. Most subjects, however, have an accessory muscle, an abnormality whose embryology and phylogeny have been extensively studied by Gordon [25]. Patients are invariably young adults who engage in sports (running, ball games, motor bicycle, wind surfing) several times a week and who experience heaviness or cramping pain in the muscle compartment upon exertion. The pain resolves in few minutes with rest. We have observed this pathology in many cases, for forearm, thigh, leg and foot. Examination after exercise can, in some cases, demonstrate swelling, which sometimes fluctuates upon palpation but hardens during contraction of the muscle. The swelling is present in both legs in more than half the patients. At rest we can observe local hypertrophy, in particular during contraction of the involved muscle.

An accessory soleus is one of the most typical example of accessory muscle in the lower leg. Proximally, the muscle inserts on the soleal line of the tibia and fibula in the lower third of the leg to reach the distal end of the calcaneal tendon or the upper or medial edge of the calcaneal bone [25, 26] (Figs. 9.3 and 9.4). Distal insertion of the muscle can be fully muscular or through a thin tendon [26–28]. Such details can be

accurately depicted with ultrasound or MRI that confirm its typical muscular appearance and explore its relationship to neighboring structures [26]. An accessory soleus usually partly fills up a wide amount of the Kager fat pad [29, 30]. MRI is also especially efficient in differentiating this variant muscle from others that may lie in the posteromedial ankle, namely the flexor digitorum accessorius longus (FDAL) and tibiocalcaneal internus muscles [30]. On axial T1-weighted images, the accessory soleus remains posterior and superficial to the flexor retinaculum and reaches the calcaneus. Conversely, the FDAL is located anteriorly to the retinaculum and posteriorly to the flexor hallucis longus tendon to insert onto the flexor digitorum longus distally (Figs. 9.5 and 9.6) [30]. Due to this position, the FDAL lies in close vicinity with the posterior tibial neurovascular bundle. If PCI and FDAL may be confused because both travel anteriorly to the flexor retinaculum, the latter differentiates itself by the fact it lies laterally to the flexor hallucis longus, travelling with the latter through the calcaneal groove to insert onto a small tubercule beneath the sustentaculum tali, on the medial aspect of the anterior calcaneus [30].

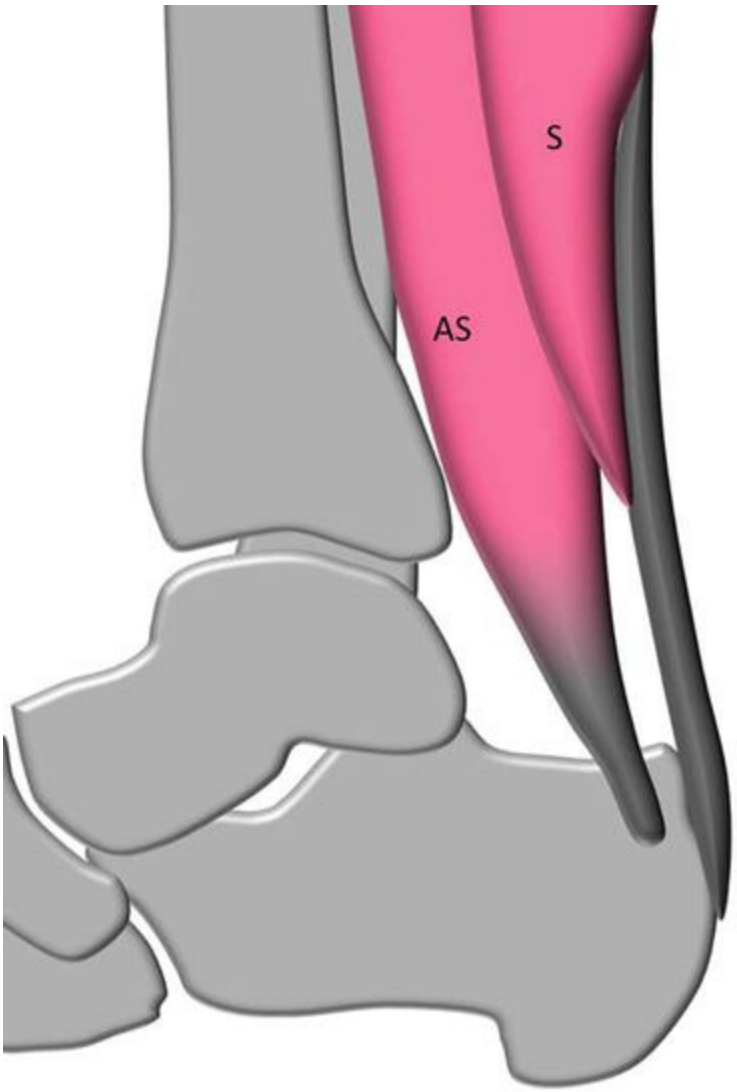

Fig. 9.3 The accessory soleus (*AS*) (medial view). Soleus (*S*)

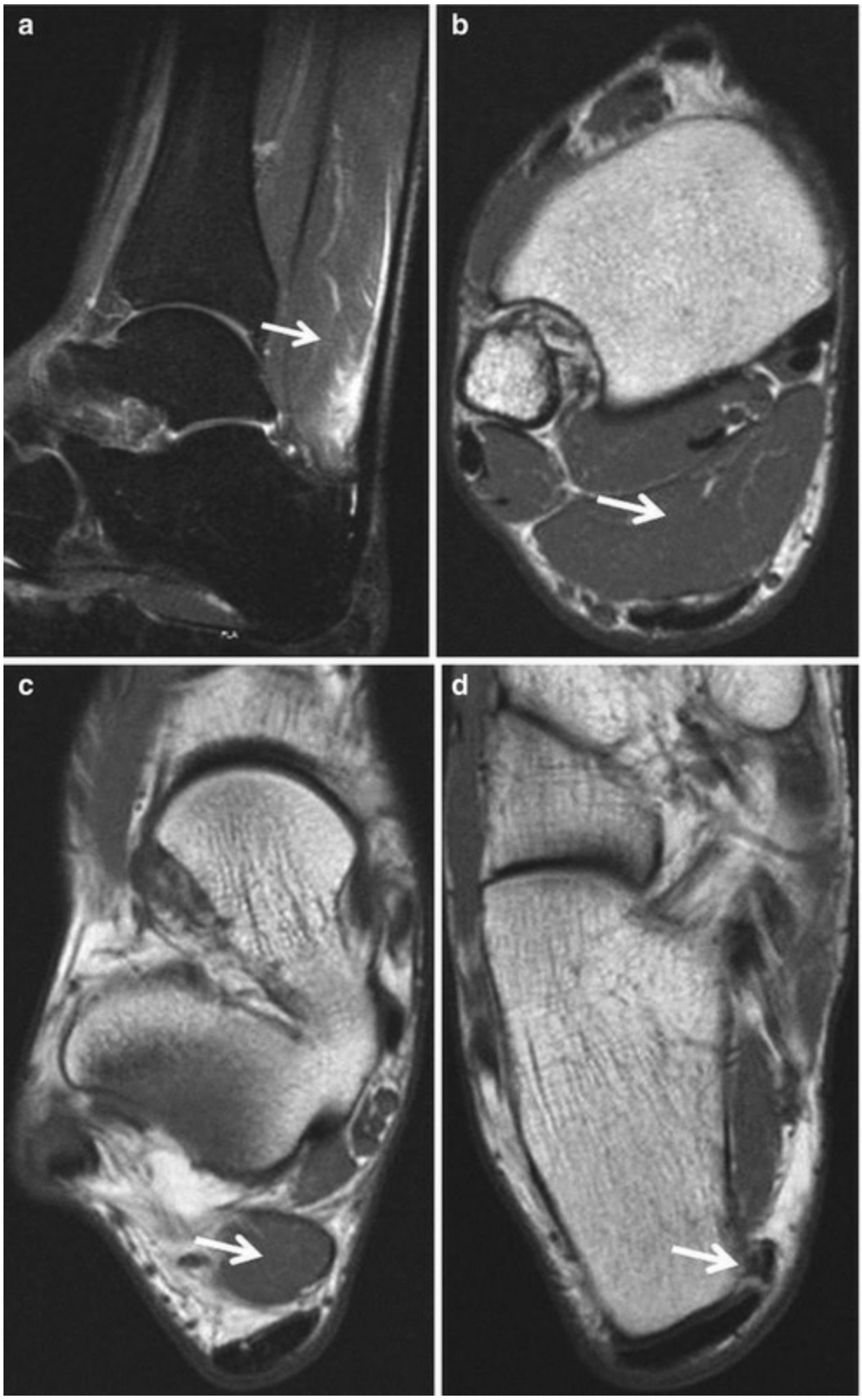

Fig. 9.4 A 25-year-old patient with recurrent pain in the lower leg during athletic activity. Sagittal STIR MRI (**a**) shows accessory soleus with spontaneous hyperintensity due to muscular strain (*arrow*). Axial T1-weighted MRIs (**b**–**d**) show the insertion of the accessory soleus (*arrow*) on the medial aspect of the calcaneus

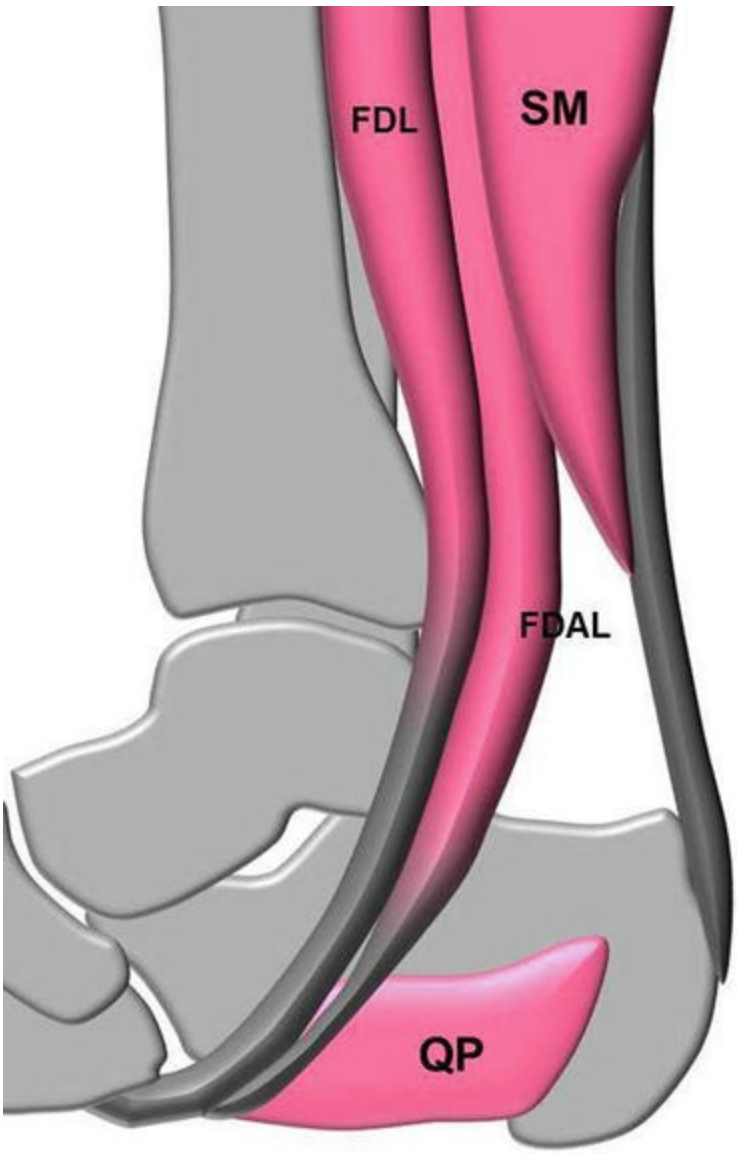

Fig. 9.5 The flexor digitorum accessorius longus (*FDAL*) (medial view). Soleus muscle (*SM*), Flexor digitorum longus (*FDL*) quartus peroneus (*QP*)

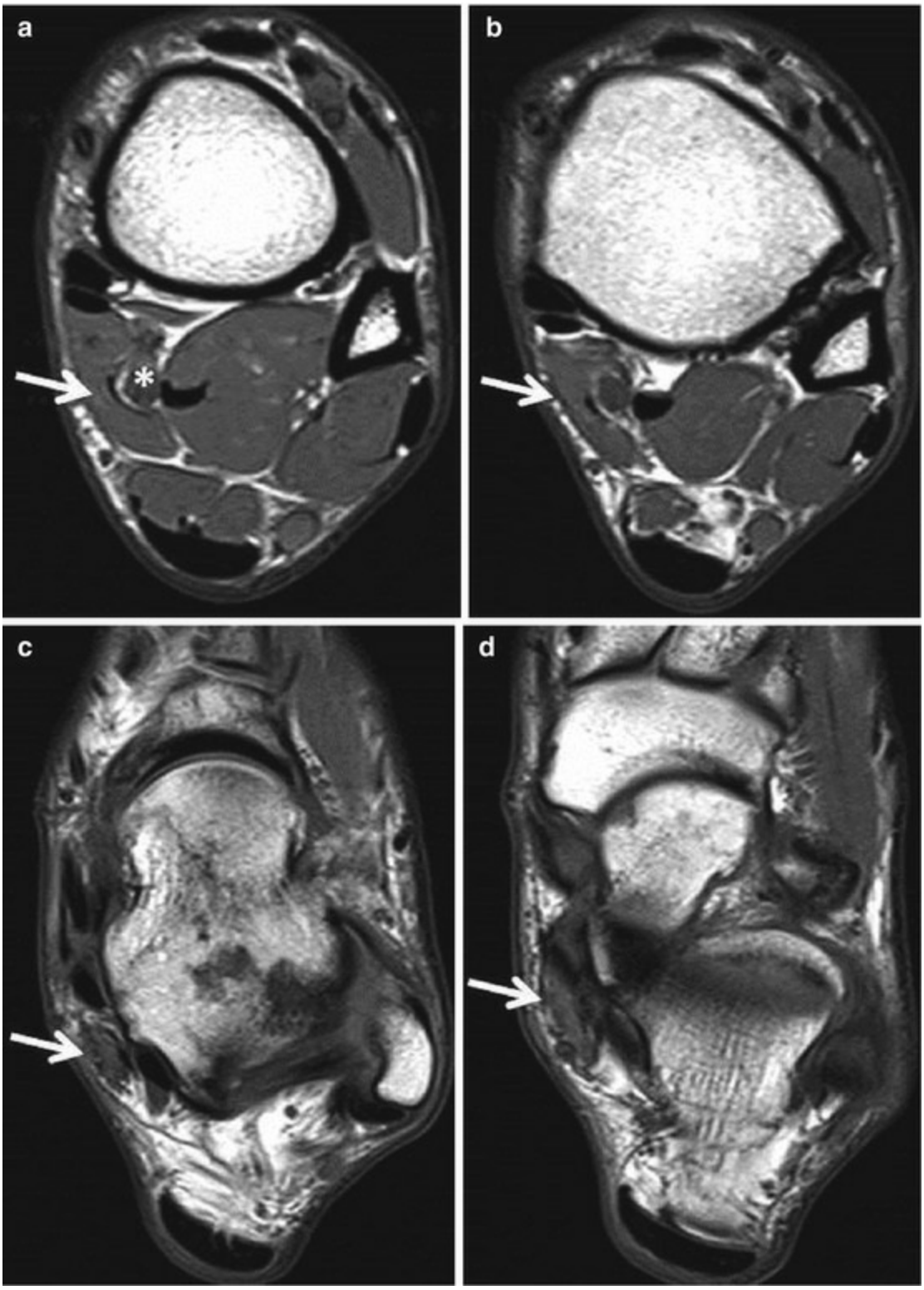

Fig. 9.6 An 18-year-old patient with posterior tibial nerve syndrome. Axial T1-weighted MRIs (**a**–**d**) show the presence of a flexor digitorum accessorius longus (FDAL) muscle (*arrow*), embedding the posterior tibial nerve and reaching the FDL distally. Intraneural cyst of the posterior tibial nerve is visible (*asterisk*)

An accessory soleus may have multiple clinical presentations, including the vast majority of pathophysiologic ways from which a variant muscle can become

symptomatic (Table 9.1). Mass effect related to the muscle may clinically raise the suspicion of a tumoral lump and require exploration with imaging [30]. As mentioned earlier, the muscle may bulge and occupy an anatomical space that is usually free of any mass effect, leading to a compartment-like syndrome. In contrast with transient modifications of signal encountered in compartment syndromes, the fact that modifications of signal on T2-weighted images are sometimes encountered demonstrates the occurrence of muscular strain within the muscle [26]. This may result, in some cases, from the inadaptation of the muscle to mechanical stress. Finally, close proximity of the accessory muscle and neurovascular bundle may lead to tunnel syndrome. In a study of 18 athletes, Kinoshita showed variant muscles to impinge on the posterior tibial nerve in four cases, including three FDAL and one accessory soleus [31]. Two other possible complications of accessory muscles, that to date have not been reported with the accessory soleus, may occur in the ankle in the vicinity of an accessory muscle. Snapping of the peroneus tertius is reported when impinging with the lateral talar dome during flexion-extension of the ankle [32]. Tenosynovitis of a flexor hallucis longus is reported when impinging on an FDAL [33, 34] or peroneocalcaneus internus muscle [34].

Table 9.1 Supernumerary muscles

		Trajectory	Key point to depiction with MRI (on axial view)	Compartment syndrome/muscular strain	Nerve compression	Vasc comp	Tendinopat
Anconeus epitrochlearis muscle	Elbow	From medial epicondyle Inserts onto olecranon	Additional muscular fibers covering the ulnar nerve above the level of the flexor carpi ulnaris (Osborne ligament)		Present Ulnar nerve [46]		
Accessory flexor pollicis longus muscle (Gantzer muscle)	Forearm	From medial epicondyle or coronoid process inserts onto the ulnar aspect of the flexor pollicis longus	Additional muscle in the vicinity of FPL		Present anterior interosseous nerve [47, 48]		
Accessory flexor digitorum	Hand	From humerus, ulna or radius	Presence of muscular		Present median nerve		

superficialis indicis muscle		through the carpal tunnel inserts onto the middle phalanx of the index	fibers or an additional tendon under the retinaculum of the carpal tunnel or the palm of the hand [48, 49]		[38, 50]		
Accessory flexor digiti minimi	Hand	From distal ulna through Guyon canal inserts onto the proximal phalanx of fifth finger or flexor digiti minimi	Presence of muscular fibers or tendon within the Guyon canal [50]		Present ulnar nerve [52]		
Accessory abductor digiti minimi	Hand	From antebrachial fascia and pisiform bone through Guyon canal shares the insertion of the abductor digiti minimi muscle [53]	Presence of a muscular layer on the superficial border of the Guyon canal [50, 51]		Present ulnar nerve [54]		
Palmaris longus	Hand	From the common flexor origin inserts onto the palmar fascia	Additional tendon located medially to the flexor carpi radialis. May be duplicated, digastric, fully muscular or reversed [55]	Present [36]	Present median ulnar nerve [36]		
Extensor digitorum brevis manus muscle	Hand	From dorsal wrist capsule inserts onto the extensor hood of the index or middle finger	Presence of muscular fibers between the second and third rays on the superficial aspect of the dorsum of the hand [55, 56]	Present [57]			
Gastrocnemius	Knee	Refer to	Refer to the	Present		Present	

medius		entrapment of the popliteal artery section	section on entrapment of the popliteal artery			popliteal artery	
Peroneus tertius	Ankle	From lower third of the leg inserts onto the basis of fifth metatarsal bone	Laterally to extensor digitorum longus				
Peroneus quartus	Ankle	From lower one third of the leg to calcaneal or cuboid bones or to peroneus longus tendon inserts onto the lateral calcaneus near the tubercule of the calcaneofibular ligament	Separate structure in close vicinity with peroneus longus and brevis [58, 59]				Present [60] peroneus brevis
Flexor digitorum accessorius longus	Ankle	From posterior compartment bones or muscles travels anteriorly to the flexor retinaculum. Inserts onto the flexor digitorum longus	Additional muscle deep to the flexor retinaculum, posterior to FHL and medial to the neurovascular bundle (in close vicinity) [30, 61]		Present posterior tibial nerve [62, 63]		Present FH [33]
Peroneocalcaneus internus	Ankle	From lower fibula travels anteriorly to the flexor retinaculum inserts on the calcaneal bone below the sustentaculum tali	Additional muscle deep to the flexor retinaculum and posterolateral to FHL, thus separated from the neurovascular bundle by the latter [30, 64]		Present posterior tibial nerve [34]		Present FH [34]
Accessory soleus	Ankle	From the deep	Additional	Present [26]	Present		

		aspect of soleus muscle or fibular bone travels through Kager fat pad, posteriorly to the flexor retinaculum. Inserts on the upper or medial aspect of the calcaneus with tendinous or muscle fibers	muscle superficial to flexor retinaculum, deep to the calcaneal tendon [26, 30]		posterior tibial nerve [31]		

In some cases, exploration of clinical swelling with imaging does not depict a proper accessory muscle but only reports *hypertrophy of an existing muscle*. This is especially frequent in the proximal leg as this situation is also responsible for arterial entrapment in the course of so-called "functional" popliteal artery syndrome.

In the forearm, Ryu has advocated that pain and discomfort related to accessory muscles result from shear phenomena, a condition reported as the "supernumerary muscle belly syndrome" [35]. The author suggests that most accessory muscles are attached to neighboring muscles that have a different excursion during motion. Contraction of the two muscles into two diverging directions may lead to restriction and burning pain that is usually localized on the distal one-third of the forearm. An anomalous palmaris longus muscle attached to a flexor digitorum superficialis or flexor pollicis brevis accounts for the most typical examples. Surgical excision usually offers complete relief of symptoms [35].

Similarly to muscular hypertrophy, *abnormal trajectory of a muscle* is another well-known situation that is especially prevalent in the popliteal fossa where muscular fibers may impinge with the neurovascular bundle. Details on such disorders will be treated elsewhere in this book. At the knee, we have found a tendinous lip of the medial gastrocnemius to impinge with the semi-membranosus (Fig. 9.7).

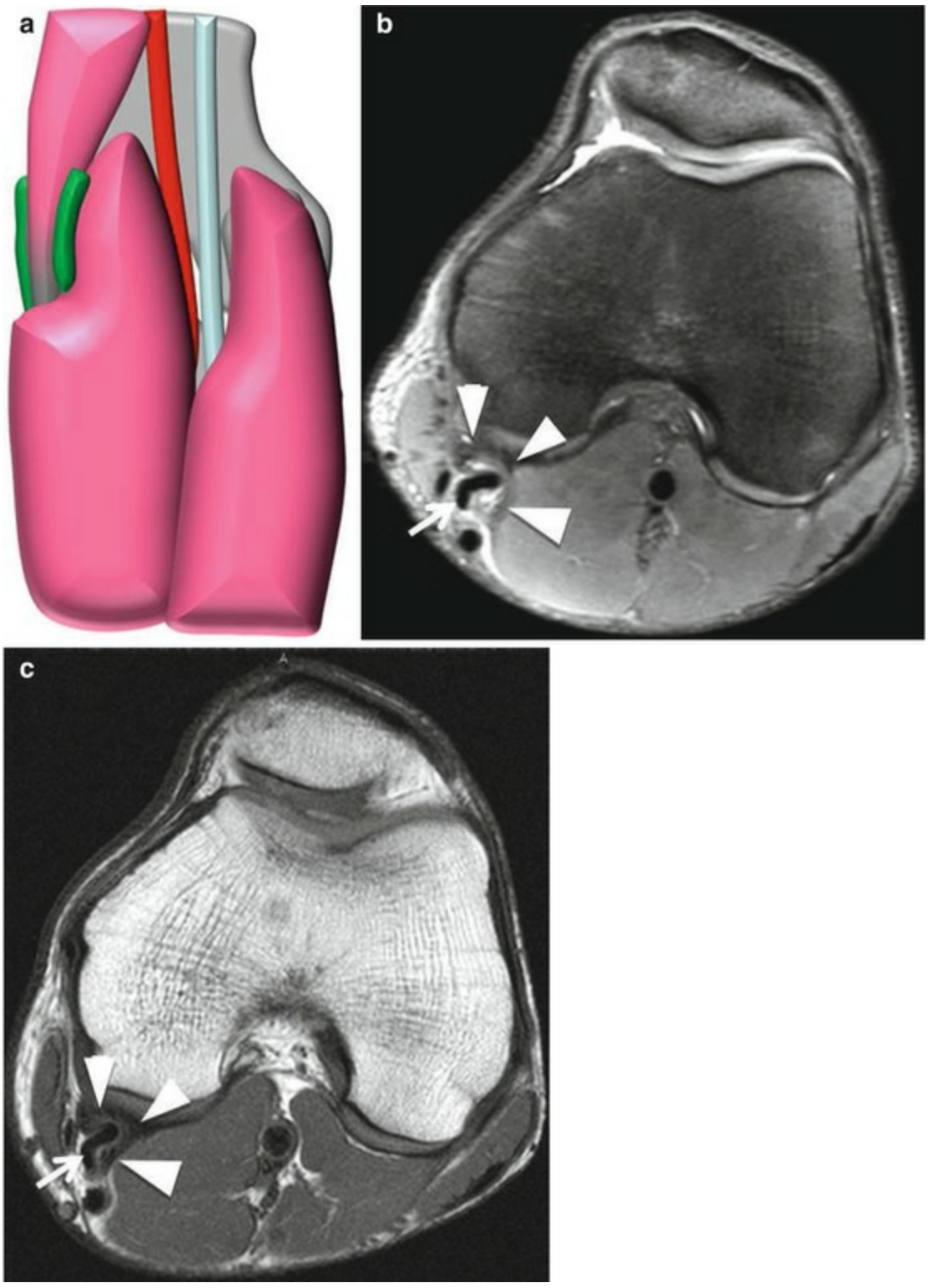

Fig. 9.7 A 32-year-old male patient with postero-medial pain of the knee when running. Posterior view of the right knee (**a**) shows the impingement of the proximal gastrocnemius and distal semi-membranosus tendons. Axial proton density- (**b**) and T1-weighted (**c**) MRI of the right knee demonstrate the proximal fibers of the gastrocnemius muscle (*arrowheads*) embedding themselves into the semi-membranosus tendon (*arrow*). Mild edema surrounding the semi-membranosus tendon is visible. Ultrasound-guided block-test with steroids resolved symptoms for a few weeks, thus confirming the presence of an extra articular impingement

Numerous *abnormalities in longitudinal organization of a muscle* may be encountered. In this regard, the palmaris longus muscle is a good model for all types of such disorders. The muscle may be fully muscular without a tendon, have a low musculotendinous junction, be digastric or even have a reverse organization [36]. Due to the mass effect they provide especially in the distal limb, such modifications of shape are known to favor tunnel syndromes. Carpal tunnel syndromes have been reported when a too distal musculotendinous junction of the superficial flexor indicis muscle or a too proximal musculotendinous junction of the lumbricals are present [37]. Diagnosis with imaging helps in preoperative planning as the presence of a variant muscle leads to open carpal tunnel release instead of an endoscopic procedure [38]. At the ankle, the low lying myotendinous junction of the peroneus brevis has been thought to favor tenosynovitis of the tendon [39]. Another study, however, has shown this finding to be frequent in the general population [40]. Impingement of an accessory muscle with the posterior tibial nerve has been mentioned above [31].

Finally, *abnormal organization of the muscle in the transverse plane* may be encountered rarely and lead to asymmetry of contraction of the muscle. Typically the presence of an ectopic cordlike tendon, converting it from unipennate to bipennate, may lead to asymmetrical contraction of the muscle when compared to the contralateral side. This may suggest the presence of a muscular herniation or a fibrous scar of a previous high grade muscular tear (Fig. 9.8).

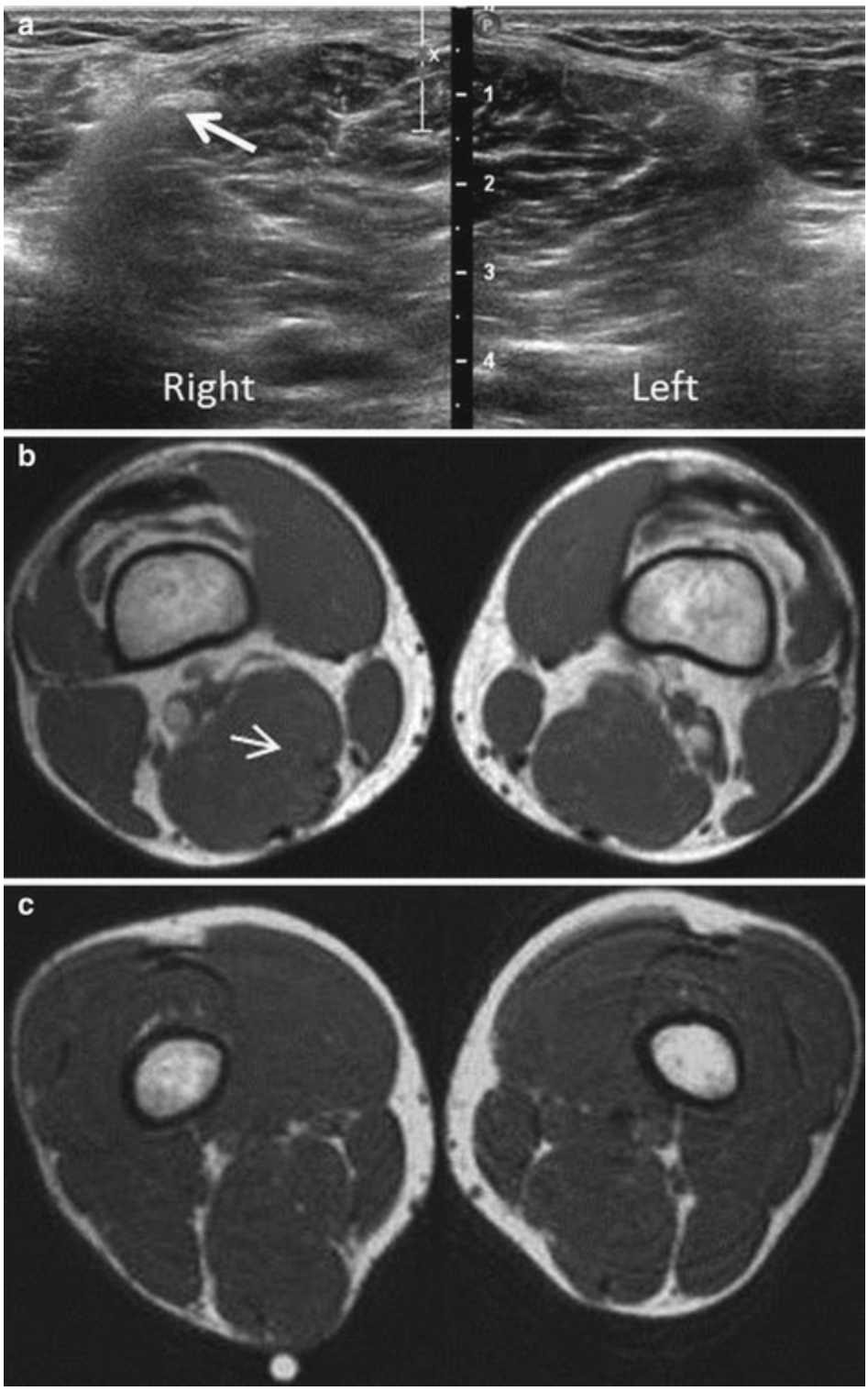

Fig. 9.8 A 16-year-old male patient with a painful posterior lump of the thigh during exercise, thought to be a muscular herniation. Transverse ultrasound image (**a**) of the thigh rules out herniation and shows a bipennate appearance of the muscle around an internal tendon (*arrow*), compared to the contralateral side. Axial T1-weighted MRI (**b**) confirms the findings of the ultrasound. Additional axial T1-weighted MRI (**c**) during contraction of the thigh confirms that congenital bipennate organization of the muscle explains non symmetrical contraction of the muscle in this young adult

The main variant muscles of upper and lower limbs are listed in Tables 9.1 and 9.2. It is interesting to notice that most cases encountered in the literature are accessory muscles while other types with internal abnormalities are less prevalent in the symptomatic population, or simply less reported in the literature.

Table 9.2 Anatomic variations leading to popliteal artery entrapment syndrome

	Aberrant muscular slip	Aberrant trajectory of the artery	Separation between artery and vein	Muscle hypertrophy
1	No	Yes	Yes	No
2	Yes	No	Yes	No
3	Yes	No	Yes	No
4	Yes	No	Yes	No
5	Variable	Variable	No	No
6	No	No	No	Yes

Surgery is the only treatment, in case of major problems. The accessory muscle should be removed if possible. However, incision of the aponeurosis and fascia have been effective, suggesting that the source of the symptoms may be a chronic compartment syndrome. In one case we could demonstrate the relation between these two syndromes, with a patient with unilateral muscle hypertrophy, with pressure at rest of 70 mmHg.

9.4.2 Vascular Compressions

Vascular compression should be routinely considered when patients present with onset of pain at a given level of physical exertion followed by resolution of the pain after a few minutes of rest. This pattern of pain suggests soleus syndrome, popliteal artery entrapment or an adventitial cyst of popliteal artery.

9.4.2.1 Soleus Syndrome

This exceedingly rare condition is due to venous compression with or without arterial compression. Tenderness of the calf upon exertion with edema and cyanosis of the calf and ankle is the characteristic picture. Examination of lateral phlebography views taken during dynamic manoeuvers demonstrates venous compression at the arch of the soleus muscle. Surgery is required to remove the anatomic obstruction.

9.4.2.2 Entrapment of the Popliteal Artery

This syndrome is difficult to diagnose. Both sides are affected occasionally. Most patients are young adults who hike, bicycle or swim on a regular basis. Vascular

claudication may occur in this area which is usually free of atherosclerosis. At the stage of simple compression, intermittent claudication that varies with the type of activity is the most common symptom. While they are always normal at rest in the involved population of young athletes, the posterior tibial and dorsalis pedis pulses are sometimes decreased when the knee is extended and the ankle actively flexed.

Doppler ultrasound should be performed first when popliteal compression is clinically suspected. Ultrasound offers high accuracy and allows reliable selection of patients with compression of the popliteal artery. Absence of abnormality on Doppler ultrasound indicates another diagnosis, such as compartment syndrome. The popliteal artery is explored with the probe in a longitudinal plane. In chronic cases, stenosis of the vessel may be seen spontaneously while color and pulsed Doppler imaging confirm the presence of aliasing artifacts and acceleration of flow. In some instances, subsequent loss of laminar flow may lead to aneurysm with partial or complete thrombosis [41]. Such lesions may explain why some patients, in whom compression has been neglected, may truly develop the disease with an acute thrombosis of the leg.

In athletes or patients with high physical demands, evolution towards thrombosis and functional impairment is usually prevented by systematic surgery. Besides assessing the degree of stenosis of the artery, preoperative planning requires accurate understanding of the cause of the compression. The hamstring tendons, greater adductus and solear arcade are rarely involved, while the proximal end of the medial gastrocnemius is the main cause of arterial compression due to a wide spectrum of variations of both insertion and trajectory [42].

Love and Whelan [43], followed by Rich [44], proposed a classification of six types of muscular patterns that may lead to popliteal artery entrapment syndrome (PAES), depending on the presence of supernumerary muscular bundles and their position relative to the neurovascular bundle (Fig. 9.9).

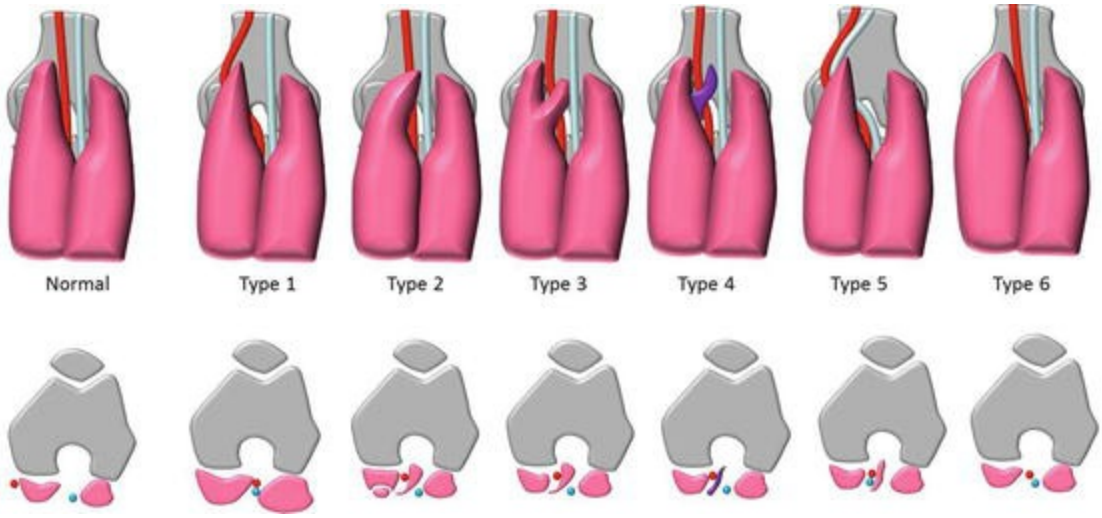

Fig. 9.9 Classification of popliteal artery entrapment syndrome

On both CT and MRI, axial views are especially fitted to assess the organization of the muscles and their relationship to the neighboring vessels [45]. Four main features should be systematically assessed to classify the origin of PAES: aberrant position of the medial gastrocnemian muscle; presence of an additional muscular bundle; separation between artery and vein; size of the medial gastrocnemian muscle (Fig. 9.9; Table 9.2). Concurrently with the axial views, enhancement of the vessels is performed to search for stenosis at rest and during dynamic maneuvers. On both CT and MR angiography, patients are placed supine. The first acquisition is performed in neutral position and after plantar flexion of the ankle, thus allowing assessment of chronic damage (including stenosis, thrombosis or aneurysm) and/or dynamic damages related to muscular compression (Figs. 9.10 and 9.11).

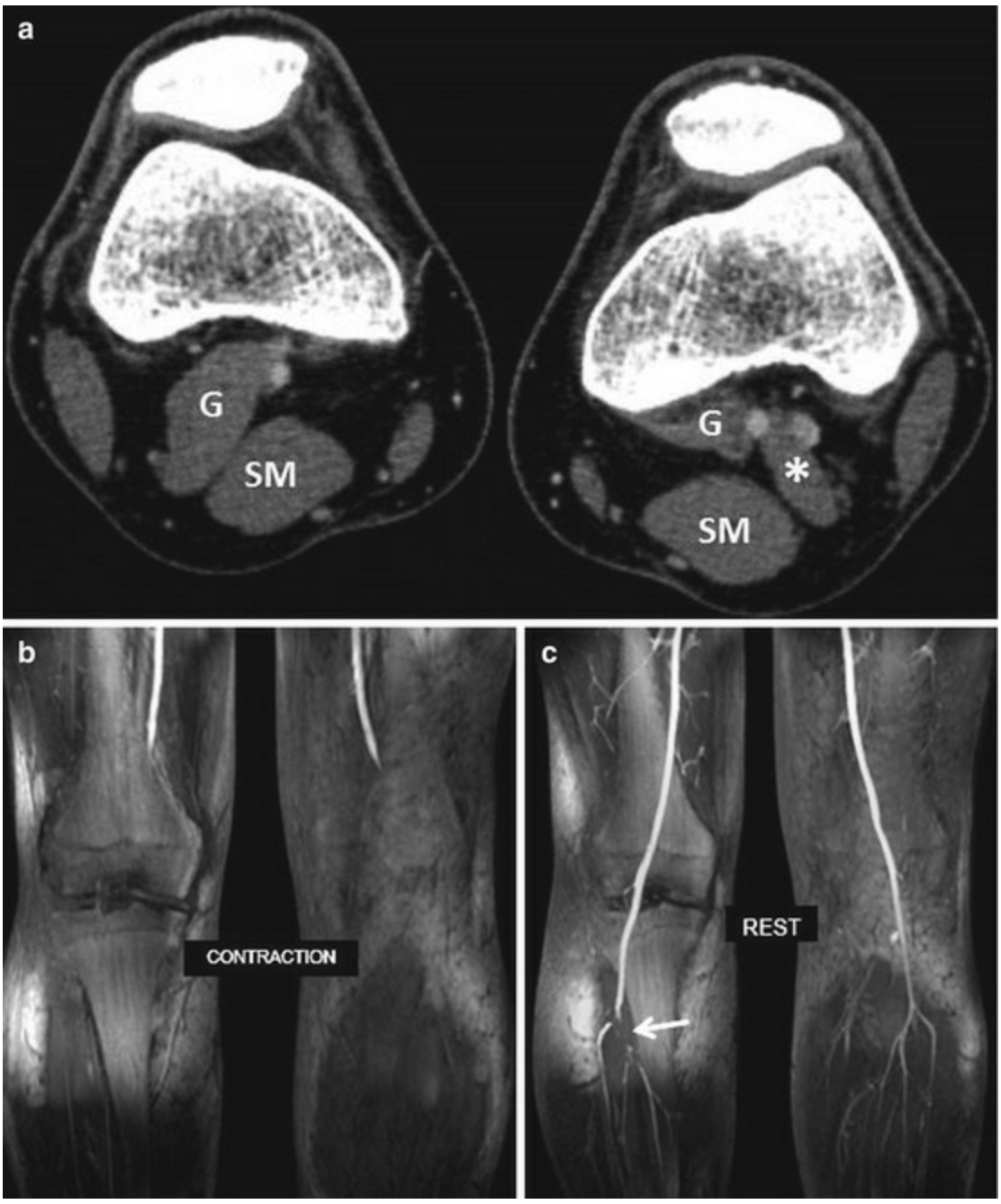

Fig. 9.10 A 29-year-old male patient with a history of subacute ischemia of the right leg and bilateral popliteal artery entrapment syndrome. Axial CT image (**a**) of the knees shows abnormal anatomy of the popliteal fossa with vascular enhancement. On the right side, medial gastrocnemius (*G*) travels laterally to popliteal vessels, resulting in a type 5 PAES. Semi-membranosus (*SM*) is visible superficially. On the left side, an accessory slip (*asterisk*) of the medial gastrocnemius lies between popliteal artery and vein, resulting in a type 3 PAES. Coronal MR angiography reformatted maximum intensity projection images (**b**, **c**) demonstrate complete stenosis of the proximal popliteal artery during contraction (**b**) of the triceps surae. At rest (**c**) residual stenosis of the tibiofibular trunk and proximal anterior tibial arteries is visible (*arrow*), resulting from a past acute thrombosis (Image courtesy of Dr. Antoine Larralde, University Hospital, Rennes, France)

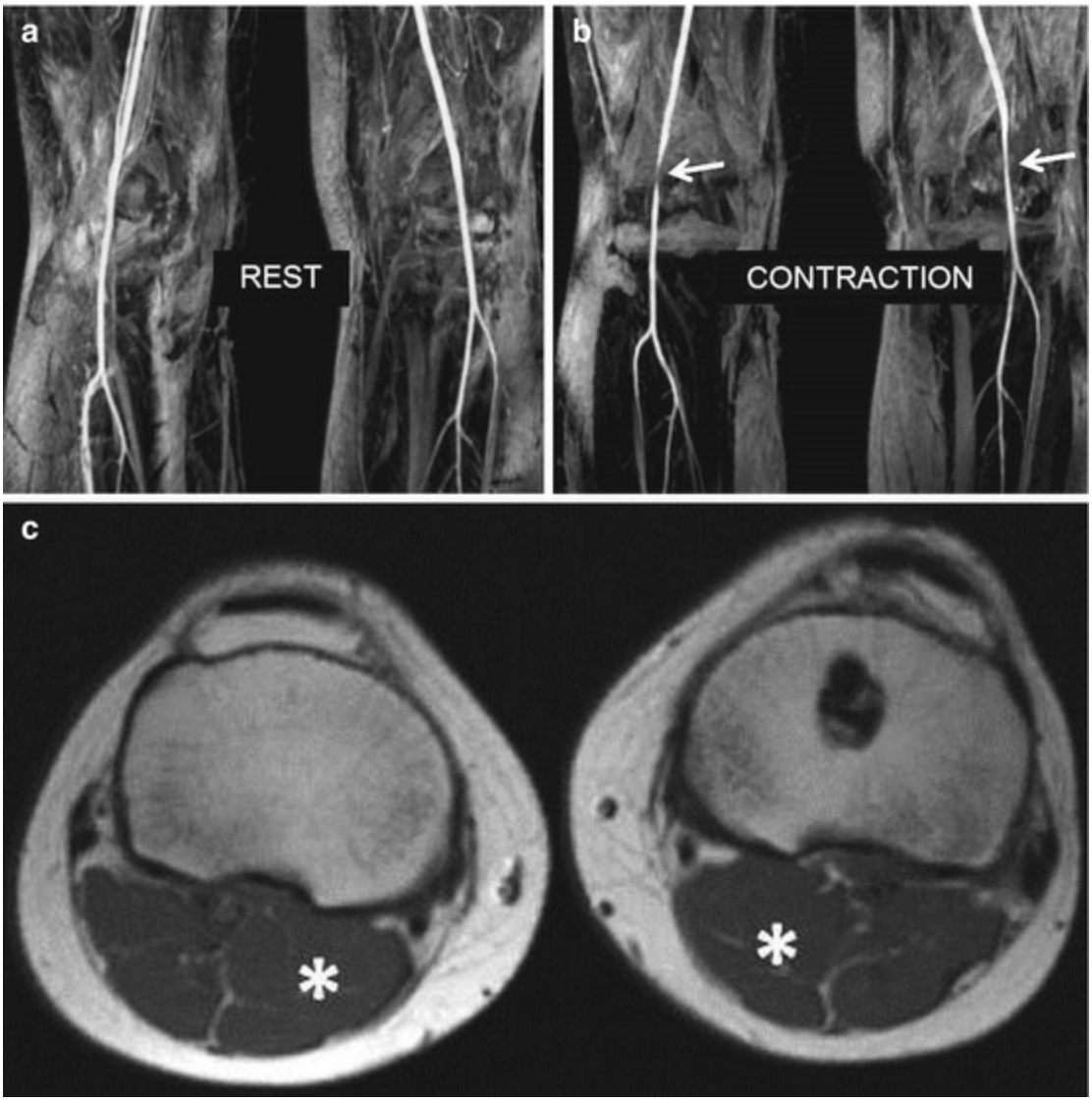

Fig. 9.11 A 23-year-old female patient with a history of chronic leg pain on exertion and functional popliteal entrapment syndrome. Coronal MR angiography reformatted maximum intensity projection (**a**, **b**) demonstrate significant stenosis of the proximal popliteal artery (*arrows*) during contraction of the triceps surae. Axial T1-weighted MRI (**c**) of the popliteal fossa confirms mild hypertrophy of the proximal medial gastrocnemius muscles (*asterisks*)

9.5 Conclusion

Non-traumatic muscular injuries usually share, along with traumatic injuries, the common feature of a progressive and insidious onset. In some instances, imaging may be relevant when it demonstrates the presence of edematous remodeling of muscles, such as in DOMS, or when it emphasizes the presence of an anatomic variant responsible for an impingement with neighboring structures. On the other hand, the limited sensitivity of routine imaging, and especially MRI, should be considered in the diagnosis of other conditions such as metabolic diseases or compartment syndrome.

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10. General Considerations on Muscle Denervation in Sports Activities: Shoulder Entrapment Syndromes and Compressive Neuropathies

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Abstract
It is not uncommon to see nerve entrapment syndromes in athletes. As a consequence of the Wallerian degeneration of a motor nerve, muscle denervation can occur. The denervated muscle fiber will develop a vasogenic edema. These abnormalities are seen electromyographically and with MRI at 48 h. MRI is the key imaging technique for diagnosis of shoulder neuropathy, determining the location of the trapped nerve, and highlighting intrinsic or extrinsic lesions causing compression of the nerve. In this chapter, the basic imaging findings of muscle denervation are reviewed, the value of the imaging techniques is explained and the shoulder neuropathy syndromes associated with denervation are described.

10.1 Introduction

Sports activities are not uncommonly associated with nerve entrapment syndromes. With Wallerian degeneration of the motor nerves, the pathology leads to muscle denervation.

Ultrasound is a very efficient technique for evaluating distal nerves. MRI, however, is the key to making the diagnosis and determining the cause of nerve compression.

In this chapter, the basic imaging findings of muscle denervation are reviewed, the value of the imaging techniques is explained and the shoulder neuropathy syndromes associated with denervation are described.

10.2 General Considerations

10.2.1 Pathophysiology

A peripheral nerve is a cord-like collection of axons (nerve fibers) with a specific concentric organization designed to guide, protect and nourish neuronal fibers within it. The main structure is the axon, which is a long extension of the neuronal cell body, specialized in transmitting nerve impulses. Support cells called Schwann cells constantly sheath it. Both myelinated and unmyelinated peripheral nerve fibers lie in a loose connective tissue called the endoneurium, made of fibroblasts, a collagen matrix and blood capillaries. They are grouped in bundles, each bundle demarcated by a solid concentric cell layer, the perineurium. Several fascicles (from a few units to a hundred) are grouped together to form the actual nerve trunk, which is separated from the surrounding environment by a dense fibrous connective tissue, the epineurium, within which many blood vessels, the vasa nervorum, circulate [1].

After section or crush injury, axons forming the peripheral nerves are able to regenerate. In the first hours after injury, the axon and the Schwann cells forming its myelin sheath begin to degenerate. This process starts immediately distal to the site of injury and follows a proximal to distal path. This phenomenon known as Wallerian degeneration lasts between 1 and 2 weeks, depending on the length of the nerve involved. Macrophages then clean up the cell debris and release growth factors, which stimulate axonal development. Regenerative nerve buds advance distally, at a rate of 1–2 mm per day, guided and stimulated by the remaining endoneural environment. Should the nerve margins be too far apart, the axonal buds will no longer have guidance and may grow anarchically, forming a mass, the amputation or traumatic neuroma. This axonal regrowth sequence only occurs completely and effectively in clear-cut nerve section or focal nerve compression.

Muscle denervation is the consequence of Wallerian degeneration of a motor nerve. Denervated muscle fibers will first develop vasogenic edema. These abnormalities are seen electromyographically and with MRI at 48 h after injury, and a typical histological appearance is seen after 3 weeks. If denervation persists, metabolic changes within the muscle will progress to muscle fiber atrophy and to a fat-content increase. Chronic denervation leads to diffuse fatty infiltration of the affected muscles after several months [2–7].

10.2.2 Nerve Injury Grading System

Traumatic nerve injuries involve a wide spectrum of damage, ranging from simple compression to total destruction with tissue loss. Several nerve damage classifications have been described to reflect the various possible levels of nerve damage. The most widely used is the five stage Sunderland classification (1978) inspired by the work of Seddon (1943) [1, 8–10]:

- Sunderland 1 (neuropraxia according to Seddon): a local conduction block secondary to focal demyelination, therefore involving destruction of the myelin sheath but with no damage to the axon or rupture of the endoneural support tissue. Recovery occurs fully within 12 weeks;
- Sunderland 2 (axonotmesis according to Seddon): this involves a loss of axonal continuity, with complete distal Wallerian degeneration. The supporting connective tissue is preserved and the endoneural tubes guide the proximal to distal axonal regrowth. Complete recovery usually occurs but requires several months, depending on the distance, which has to be re-innervated;
- Sunderland 3: damage locally destroys the axon, the myelin sheath and the endoneural tubes. The perineurium and epineurium remain intact. Regrowth is variable but generally incomplete because of incorrect orientation of the fibers and endoneural scarring with fiber trapping;
- Sunderland 4: only the epineurium remains intact with endoneural scarring and loss of endoneural and perineural continuity. The scarring prevents regrowth of the fibers and results in the formation of a neuroma;
- Sunderland 5 (neurotmesis according to Seddon): the nerve is totally transected, affecting the endoneurium, epineurium and perineurium. As in stage 4, only surgery can offer hope of nerve regrowth.

10.3 Etiologies of Neuropathies in Athletes

In sports activities, muscle denervation is usually associated with nerve entrapment syndromes. Nerves are susceptible to compression, damage, and eventual impairment of their terminal function as they traverse fibromuscular, fibrous, osteofibrous, and bony canals or “tunnels” [11] (Fig. 10.1). These entrapment syndromes will be emphasized in the second part of this chapter dedicated to shoulder neuropathies.

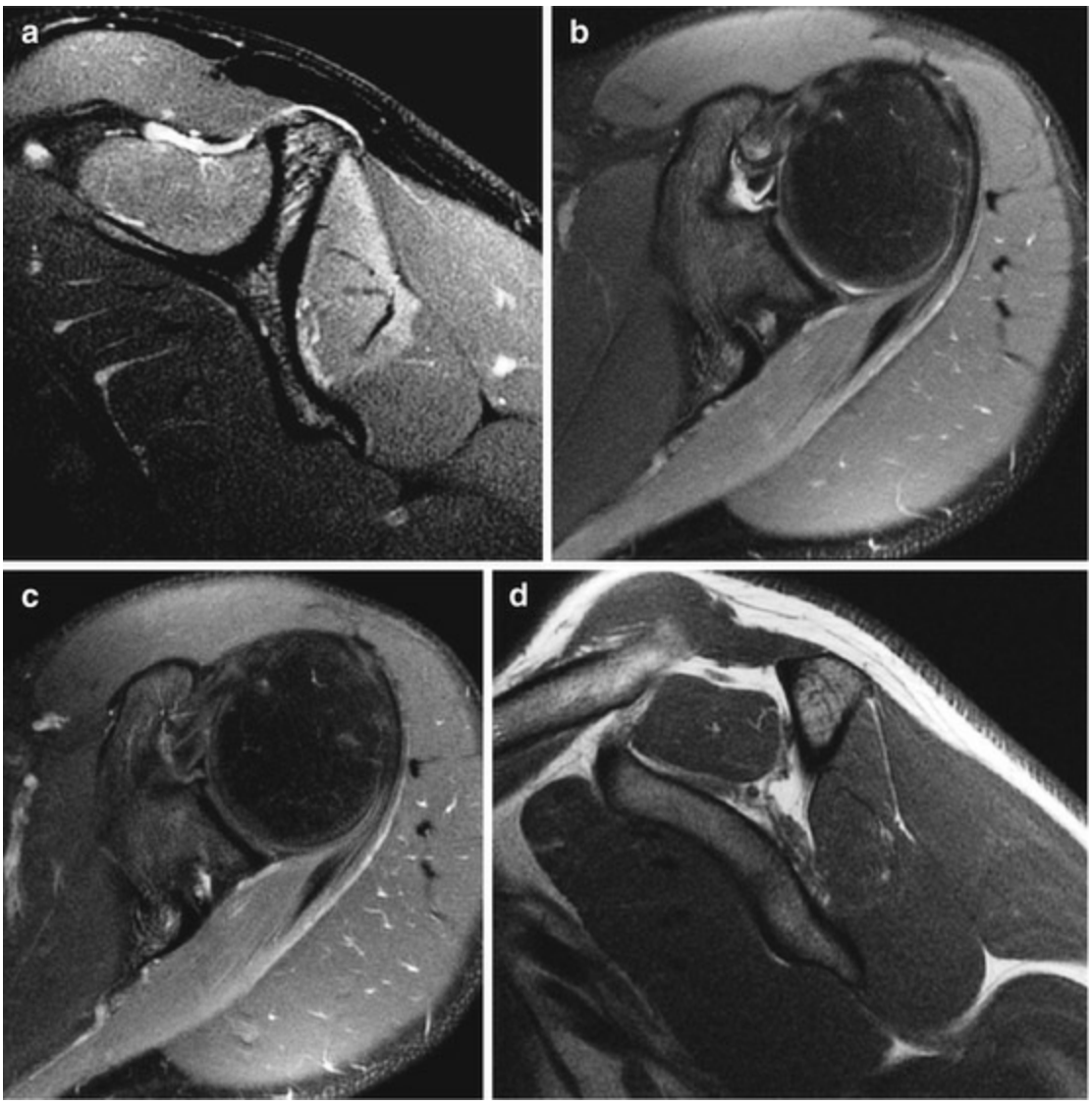


Fig. 10.1 Suprascapular neuropathy at the scapular notch in a 27-year-old patient. Sagittal (**a**) and axial (**b**) fat-suppressed T2-weighted MRI show edema of the supraspinatus and infraspinatus muscles. (**c**) Axial contrast-enhanced fat-suppressed T1-weighted MRI shows a homogeneous enhancement of the infraspinatus muscle. (**d**) Sagittal T1-weighted MRI shows muscle atrophy and signal increase of the supraspinatus and infraspinatus muscles

Peripheral nerve damage may also be due to many other causes. An acute trauma is also a frequent situation in athletes (direct contusion, blunt trauma, acute stretch injury…) (Figs. 10.2 and 10.3). Most of them are associated with complete recovery.

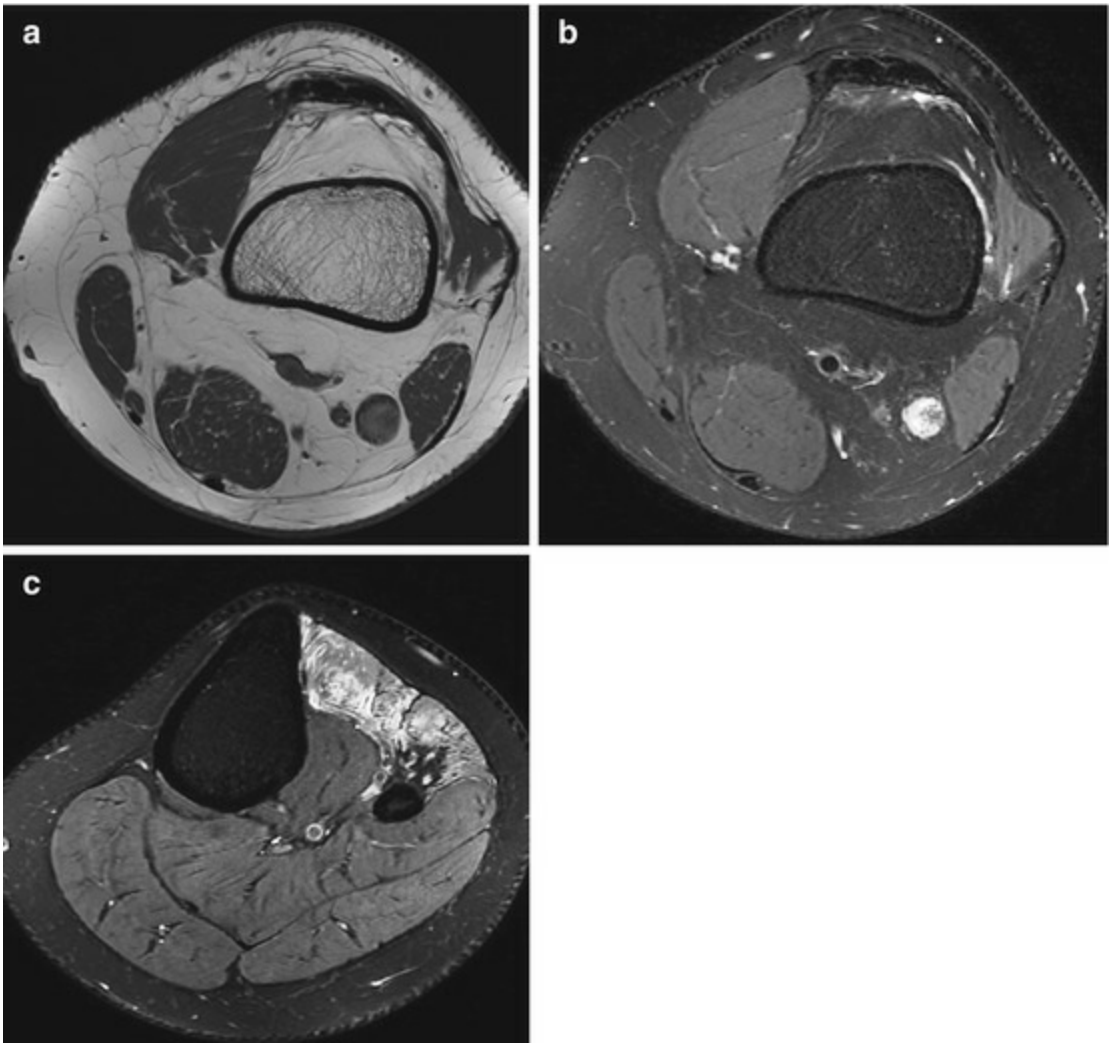


Fig. 10.2 A 26-year-old soccer player with a hematoma of the common peroneal nerve after a knee trauma. Axial (**a**) T1- and (**b**) fat-suppressed T2-weighted MRI show an enlargement of the common peroneal nerve with a loss of the fascicular structure, above the level of fibular head. (**c**) Axial fat-suppressed T2-weighted MRI shows patchy high signal in tibialis anterior, extensor digitorum, extensor hallucis longus muscles

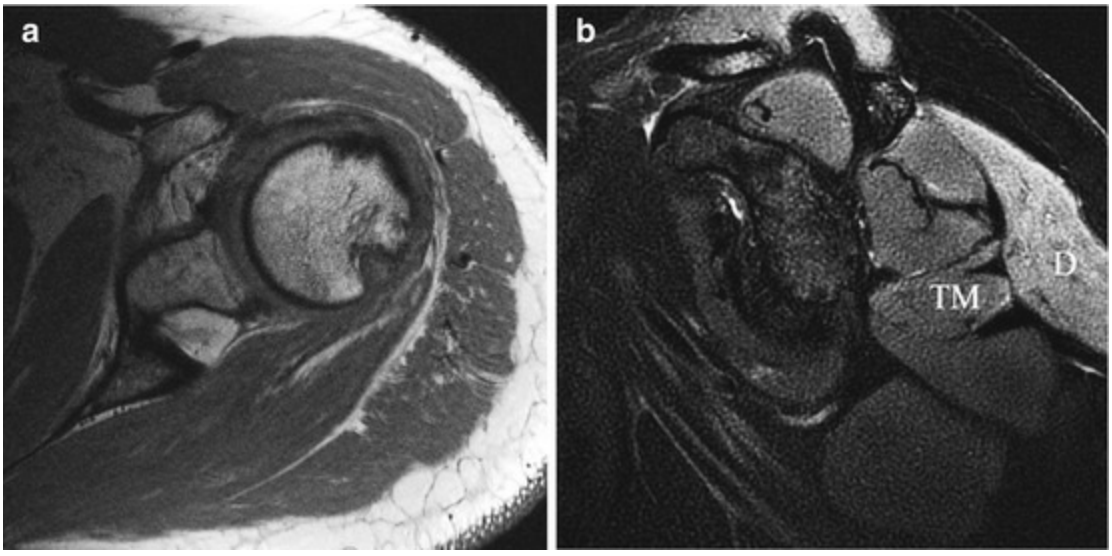
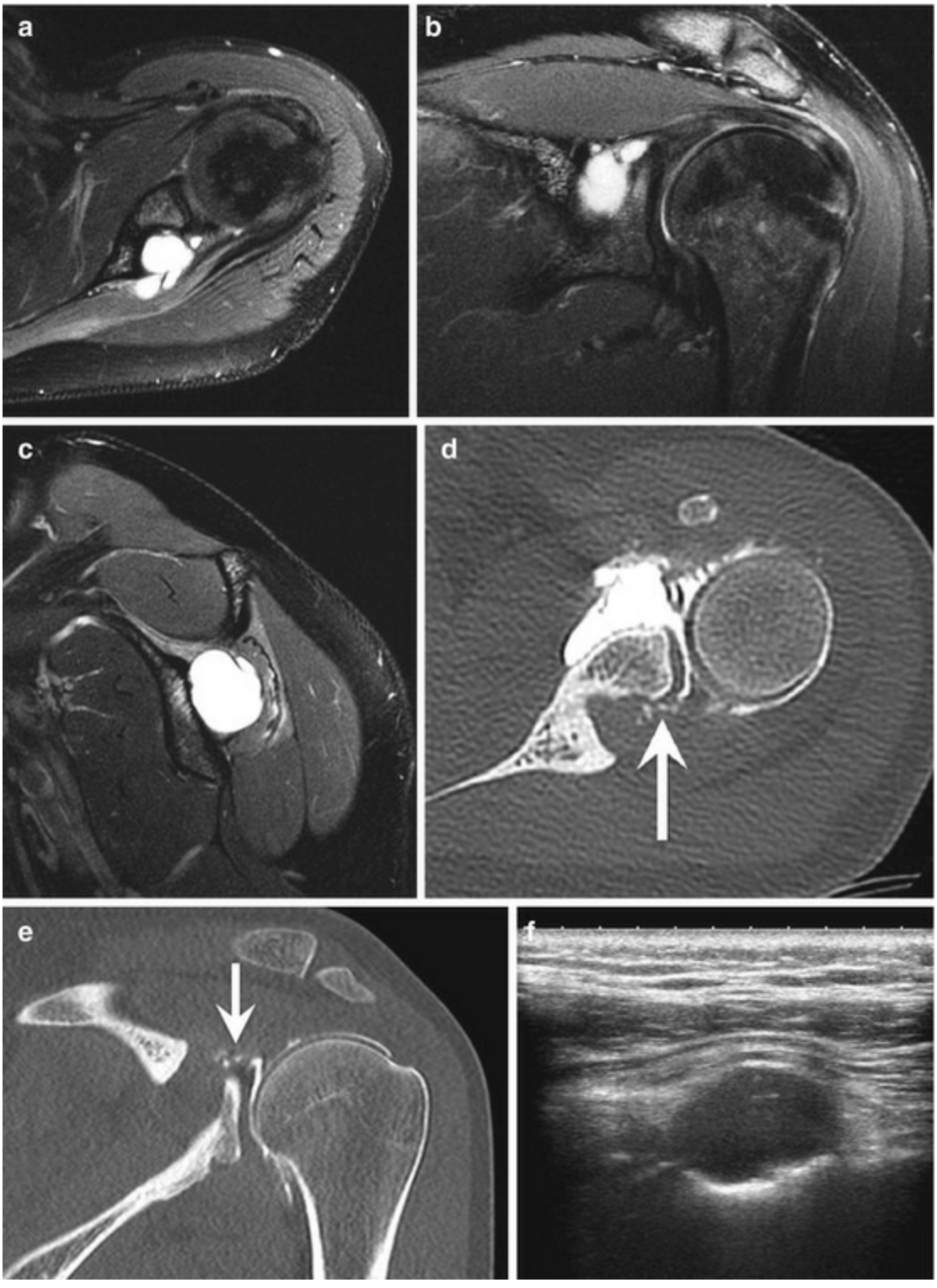


Fig. 10.3 Axillary nerve trauma after an anterior glenohumeral dislocation. (**a**) Axial T1-weighted MRI shows a Hill-Sachs lesion and fatty degeneration of the deltoid muscle. (**b**) Sagittal fat-suppressed T2-weighted MRI shows mild muscle atrophy and a high signal of the teres minor (*TM*) and deltoid (*D*) muscles

The nerves close to joints may be compressed by synovial and ganglion cysts, both of them being connected to a joint (Figs. 10.4 and 10.5). According to Spinner, a connection to an articular branch of the nerve, cyst fluid propagation along the path of least resistance, and the variation of intra cystic fluid pressure explain the pathophysiology and the different aspect of intra-neural ganglion cysts [12]. Finally, as for all patients, neurogenic tumors, soft tissue or bone tumors and any inflammatory tissue compressing the nerves may affect athletes (Figs. 10.6, 10.7, and 10.8).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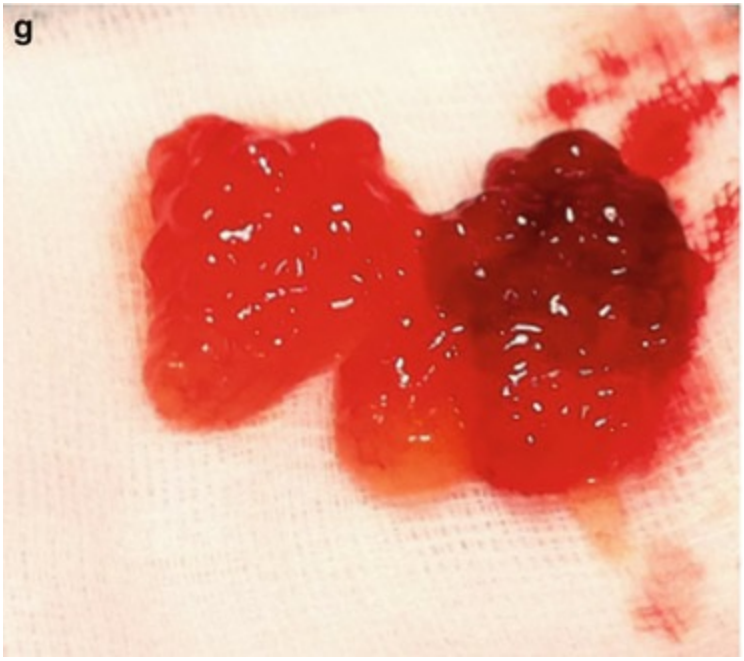


Fig. 10.4 A 28-year-old woman with a spinoglenoidal cyst and a posterosuperior impingement of the shoulder. (**a**) Axial, (**b**) coronal and (**c**) sagittal fat-suppressed T2-weighted MRI show a large cyst of the spinoglenoidal notch responsible for compression of the suprascapular nerve. Note the muscle atrophy and edema of the infraspinatus muscle. MRI also shows some heterogeneity of the posterosuperior labrum with a suspicion of a tear. (**d**) Axial and (**e**) coronal reformatted CT arthrography images clearly demonstrates the posterosuperior labral tear connecting with the cyst (*arrow*). Due to its highly viscous content, the cyst is not completely filled by the contrast medium. (**f**) Short axis ultrasound image shows the cyst. (**g**) Picture shows the gelatinous aspect of the cyst content

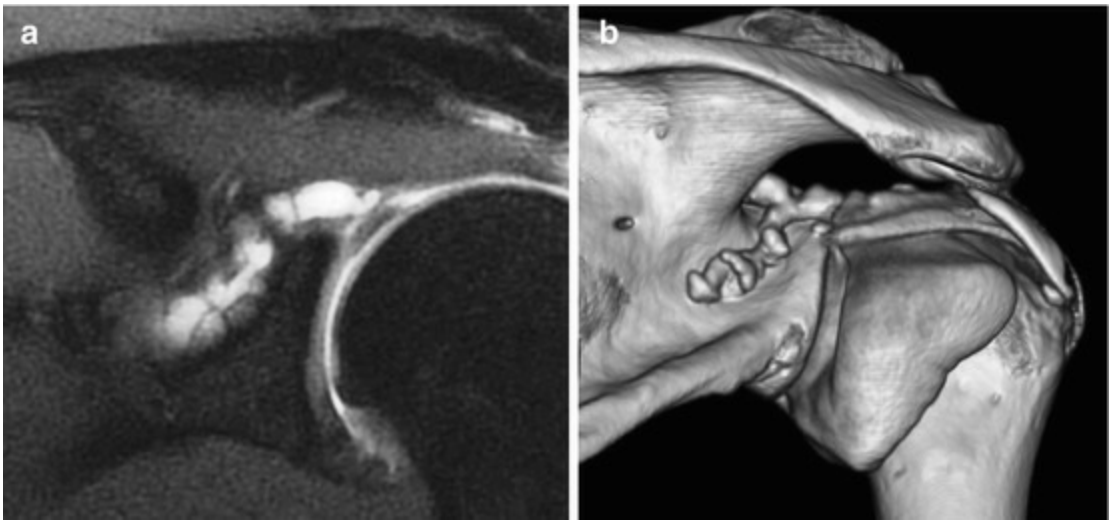


Fig. 10.5 Spinoglenoidal cyst associated with a SLAP lesion. Its long and tortuous path is in agreement with Spinner's theory [12]. (**a**) Coronal fat-suppressed T1-weighted MR arthrography image with fat suppression shows multiseptated associated with the SLAP lesion. (**b**) Volume rendering CT arthrography image confirms the MRI findings. (From Blum et al. [25])

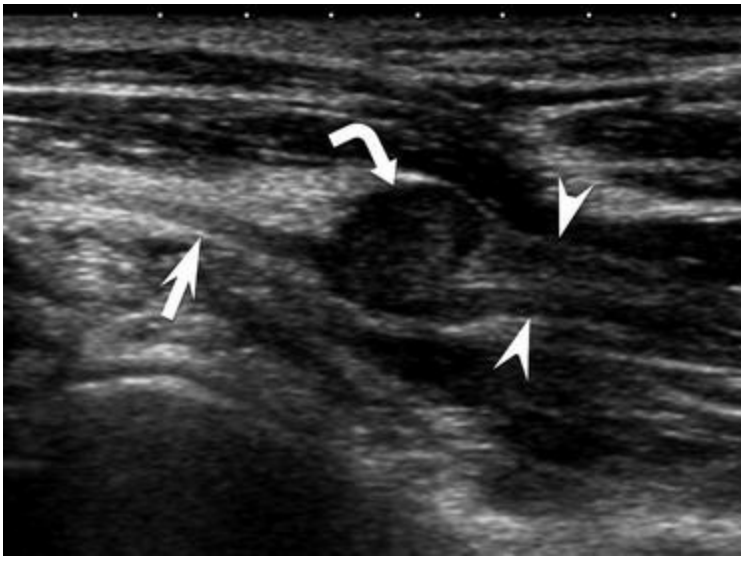


Fig. 10.6 A 34-year-old patient with schwannoma of the radial nerve. Longitudinal ultrasound of the radial nerve shows a schwannoma (*curved arrow*) affecting the radial nerve (*straight arrow*) encompassing its division into superficial and deep branches (*arrowheads*)

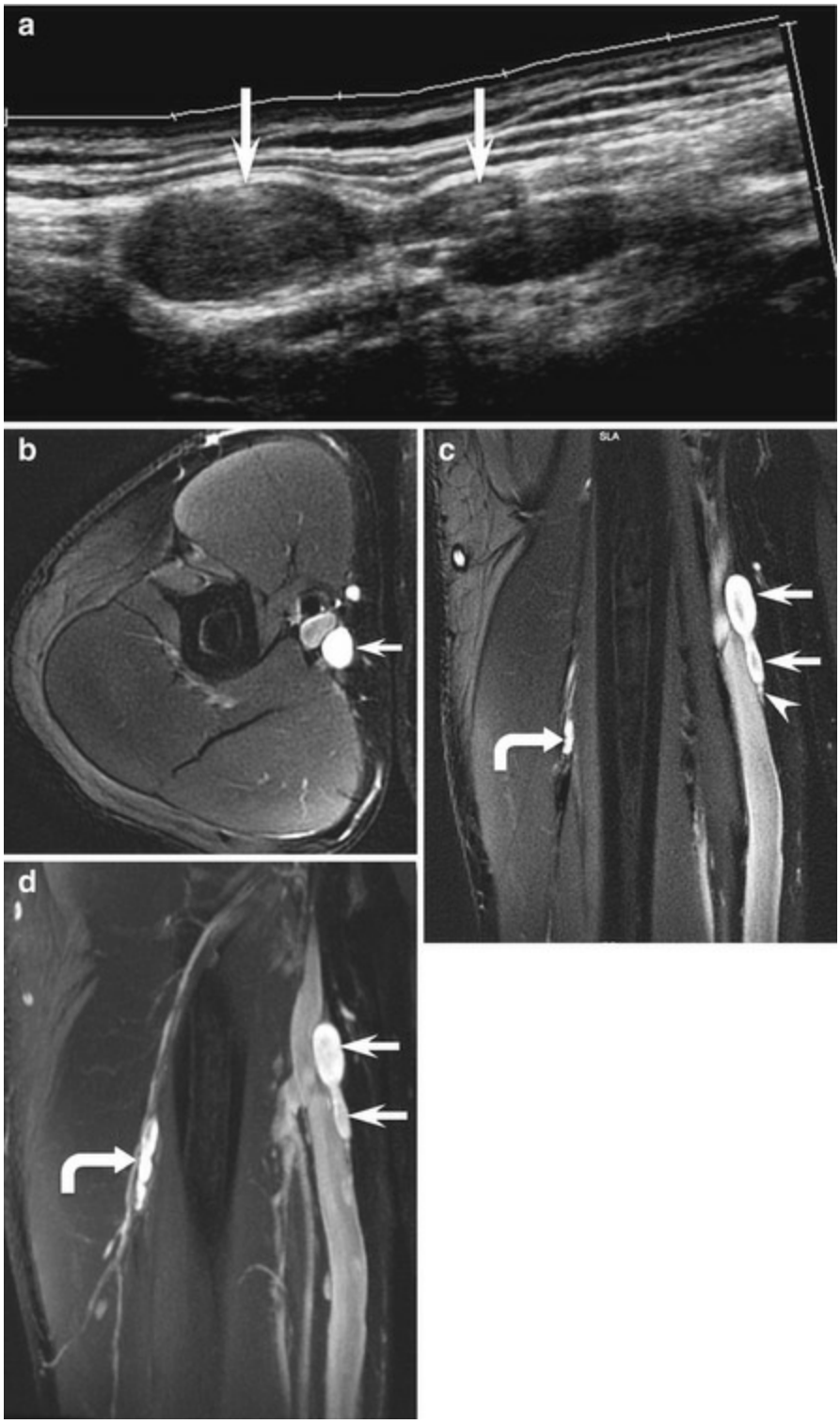
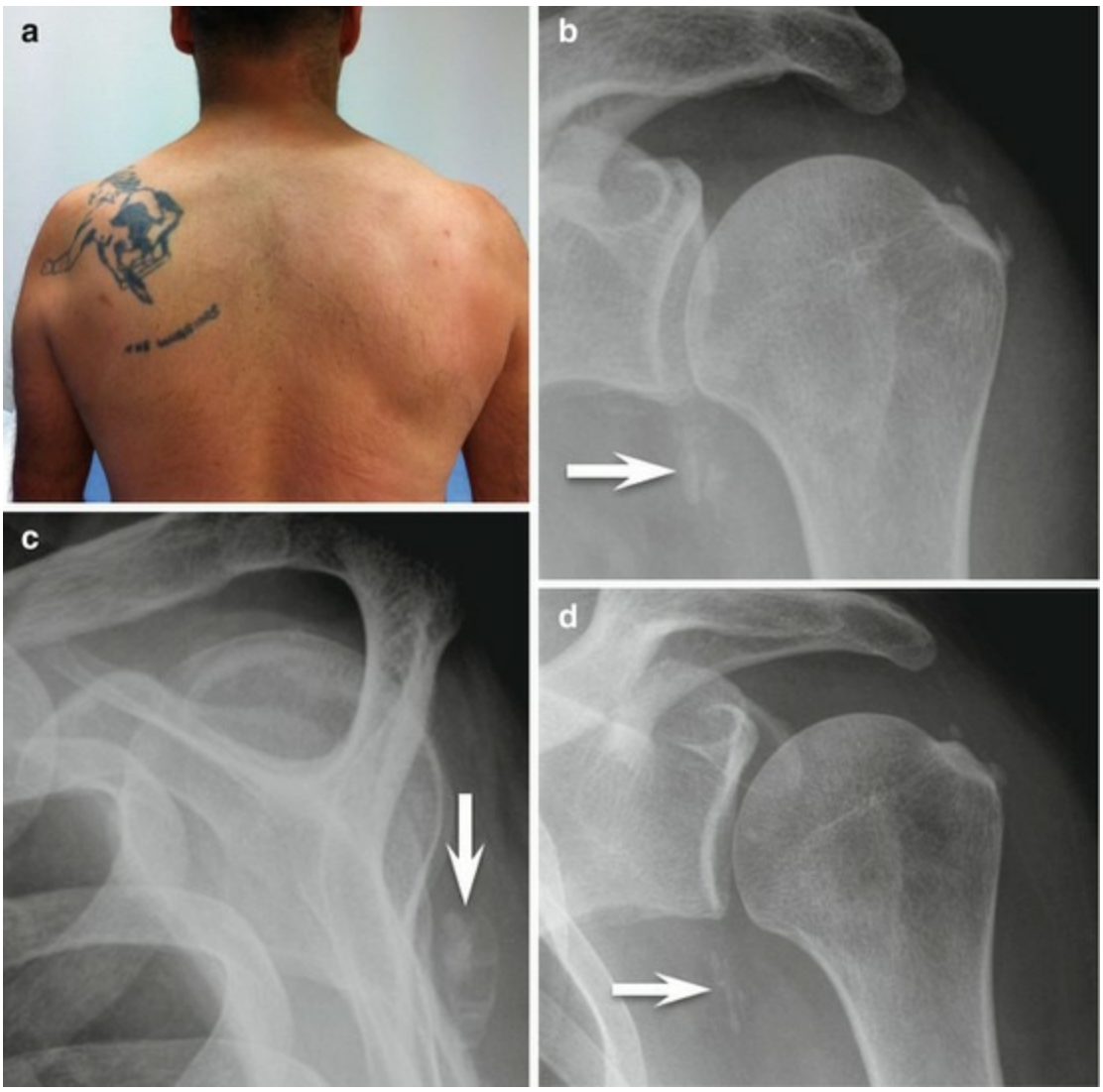


Fig. 10.7 Multiple small neurogenic tumors of the medial antebrachial cutaneous and radial nerves. (**a**) Ultrasound shows two small neurogenic tumors (*arrows*) affecting the medial antebrachial cutaneous nerve. (**b**) Axial, (**c**) coronal and (**d**) coronal multiplanar volume reformation fat-suppressed T2-weighted 3 T MRI show two small nerve tumors (*arrows*) affecting the medial antebrachial cutaneous nerve (*arrowhead*) and tiny neurogenic tumors of the radial nerve (*curved arrow*). Note the vascular inflow signal reduction and the absence of ghost artifacts caused by vascular pulsatility facilitating the depiction of the tumors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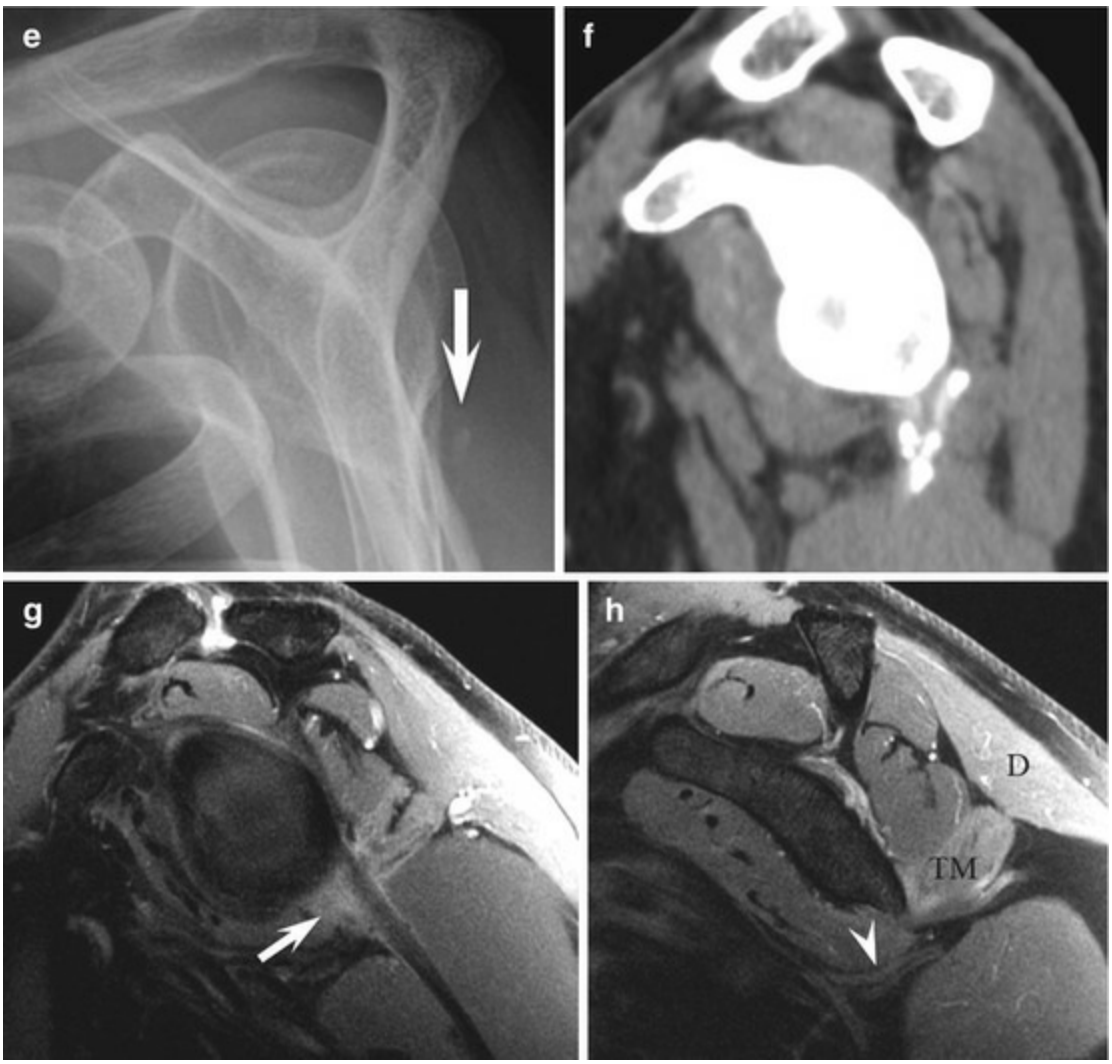

Fig. 10.8 Quadrilateral space syndrome of the left shoulder due to calcific tendonitis of the triceps tendon at the resorptive stage, 2 months after trauma. (**a**) Photograph of the patient showing atrophy of the deltoid and teres minor muscles. Initial (**b**) anteroposterior and (**c**) Y-view radiographs show calcific tendonitis of the supraspinatus tendon and the triceps tendon (*arrow*). Follow-up (**d**) anteroposterior and (**e**) Y-view radiographs 2 months later show a partial resorption of calcium hydroxyapatite deposits of the triceps. (**f**) Sagittal reformatted CT image shows the calcification affecting the triceps tendon. (**g**, **h**) Sagittal fat-suppressed T2-weighted MRI show muscle edema of the teres minor (*TM*) and deltoid (*D*) muscles as well as some inflammatory tissue (*arrow*) affecting the triceps tendon and the upper part of the quadrilateral space. Note the good depiction of the axillary nerve (*arrowhead*)

Iatrogenic nerve injuries in one series accounted for 17.4 % of all traumatic nerve injuries [13]. Nerve injuries can result from direct surgical trauma, mechanical stress on a nerve due to faulty positioning during anesthesia, injection of neurotoxic substances into a nerve, compression by a hematoma secondary to drawing blood or through anticoagulation, tourniquets, dressings, casts or orthotic devices (Fig. 10.9). Sites especially likely to be affected include the carpal tunnel and wrist, as well as the knee and the shoulder [14–16]. Neurologic complications associated with regional anesthesia are uncommon. Although intraneural injection during regional anesthesia has a higher incidence than previously appreciated it is not necessarily associated with

nerve injury [17].

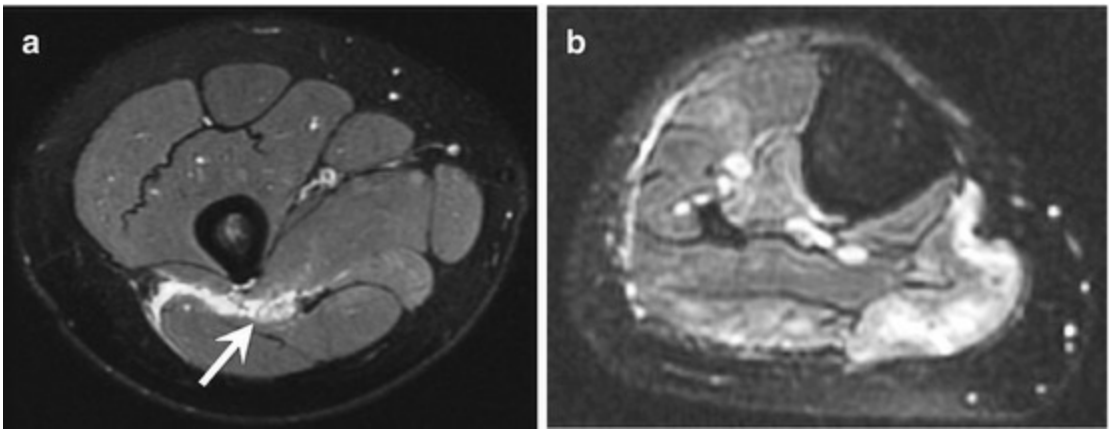

Fig. 10.9 A 26-year-old woman with an iatrogenic sciatic nerve injury due to a nerve block and resultant permanent residual deficits. (**a**) Axial fat-suppressed T2-weighted MRI shows an enlargement of the sciatic nerve (*arrow*) surrounded by soft tissue edema. (**b**) Axial fat-suppressed T2-weighted MRI of the calf shows patchy muscle edema mostly affecting the gastrocnemius muscles

Disabled athletes face many challenges during training and competition [18]. Stump pain is a common problem following limb amputation. The etiology is often multifactorial and the treatment challenging [19, 20]. Nerve section is responsible for scar tissue formation, so-called traumatic neuroma that occurs at the end of an injured nerve, usually 1–12 months after amputation (Fig. 10.10). During amputation, careful management of the peripheral nerves is critical to minimize painful neuroma formation. Most traumatic neuromas are asymptomatic but a painful neuroma makes it virtually impossible to mount a well-fitting prosthesis socket. MRI is the key examination to localize the neuroma, show its relationship with adjacent bone and eventual heterotopic bone formation, make the differential diagnosis with stump bursitis and evaluate the soft-tissue coverage [21–23].

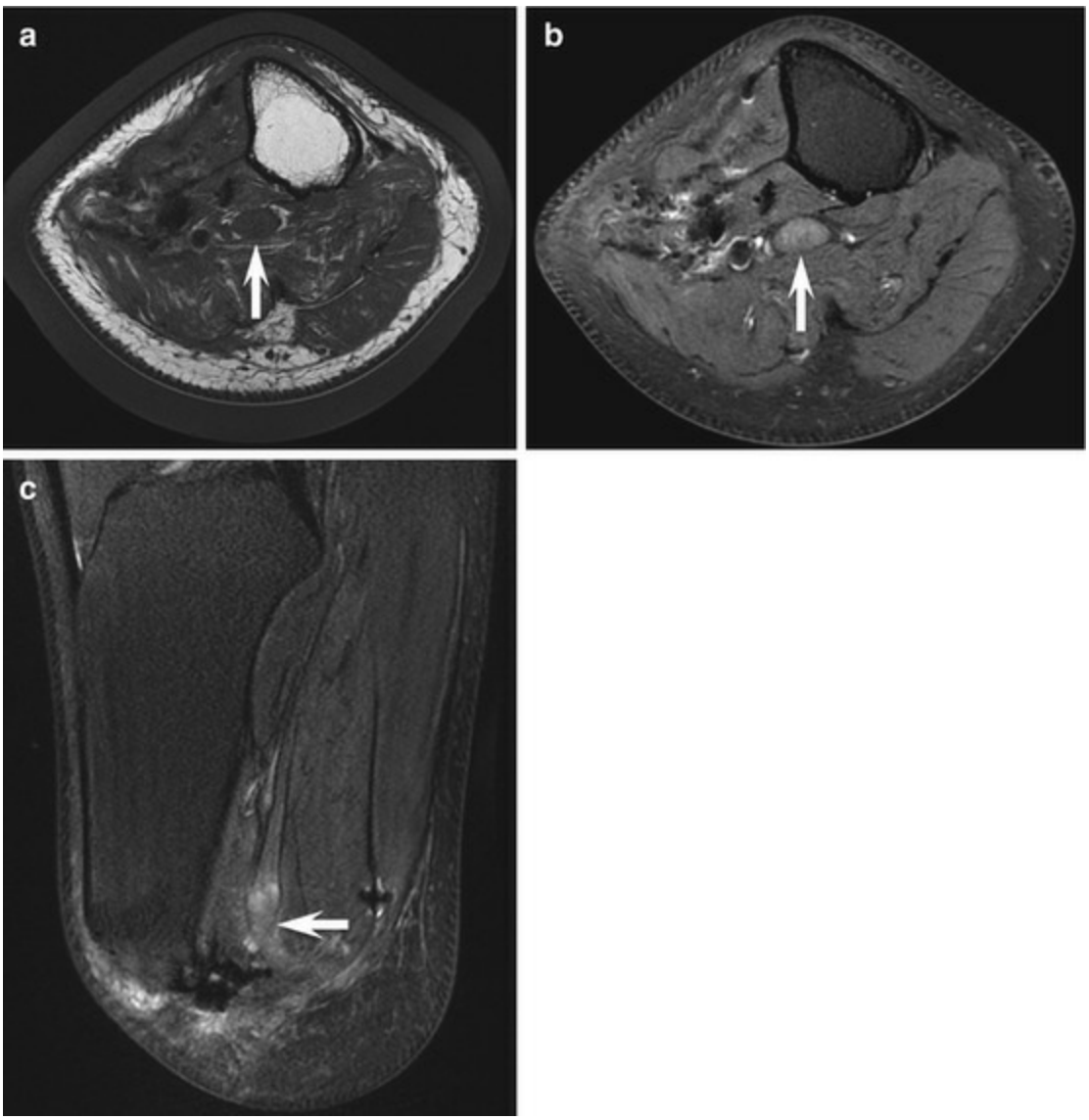

Fig. 10.10 Neuroma of the posterior tibial nerve after leg amputation. Axial (**a**) unenhanced and (**b**) contrast-enhanced fat-suppressed and T1-weighted MRI show the neuroma (*arrow*). (**c**) Sagittal fat-suppressed T2-weighted MRI shows the neuroma covered by muscles. Note also bursitis under the lower extremity of the tibia

Parsonage-Turner syndrome, also called brachial neuritis and idiopathic neuralgic amyotrophy, is the main differential diagnosis of entrapment syndromes as it may simulate suprascapular nerve entrapment, quadrilateral space syndrome or long thoracic nerve neuropathy (Figs. 10.11 and 10.12) [24–26]. This syndrome is characterized by a sudden onset of severe shoulder pain lasting about 2–4 weeks. It affects young men without a history of trauma; pain is followed by paralysis and atrophy of muscles of the scapular girdle and sometimes of more distal arm muscles. The anarchic distribution of muscle denervation depending on which of the brachial plexus nerves are affected is the hallmark of this syndrome. Parsonage-Turner syndrome can affect almost any nerve in the brachial plexus, although damage in the upper and middle trunk distribution with involvement of the long thoracic and/or suprascapular nerve occurs most frequently

(70–97 %) [26–28]. Other nerve trunks can be affected (phrenic nerve, cranial nerves). The presence of sensory signs is common. Bilateral but asymmetrical symptoms occur in 2–34 % of cases [27]. Persistent neuropathic pain may follow the acute setting. The clinical diagnosis is confirmed by EMG or MRI, which plays a major role in the differential diagnosis.

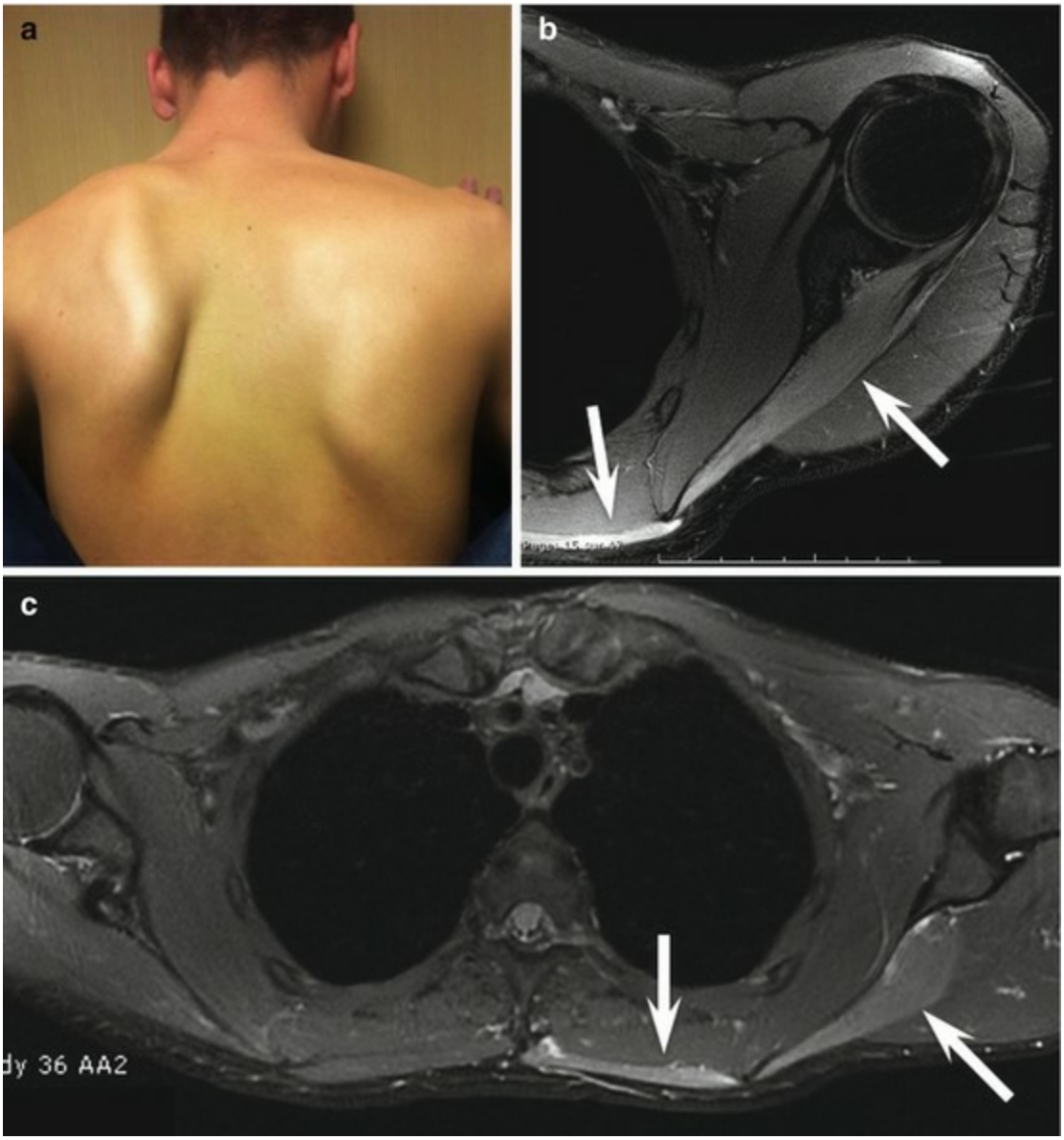

Fig. 10.11 A 22-year-old man with Parsonage-Turner syndrome due to a left suprascapular and accessory nerves palsy. (**a**) Photograph of the patient showing a winging scapula of the left shoulder. (**b**) Axial fat-suppressed T2-weighted MRI shows muscle edema of the infraspinatus and trapezius muscles (*arrows*). (**c**) Axial water-imaging with a three-point Dixon MRI shows the muscle edema of the infraspinatus and trapezius muscles (*arrows*). Note the good signal homogeneity of the image

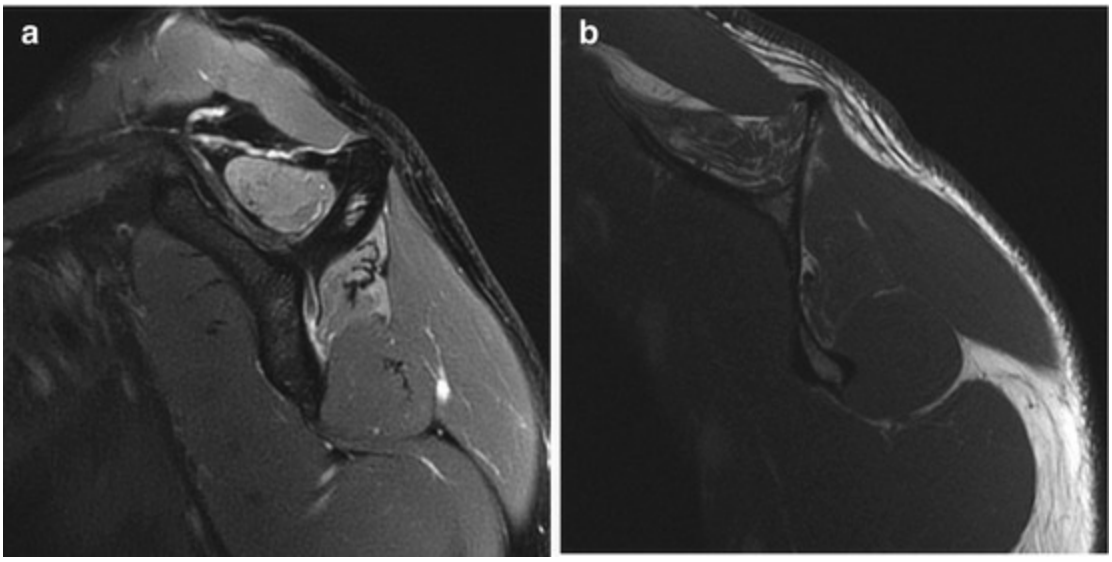

Fig. 10.12 A 43-year-old man with Parsonage-Turner syndrome due to an isolated right suprascapular nerve palsy mimicking a nerve entrapment syndrome. (**a**) Sagittal fat-suppressed T2-weighted MRI shows a muscle edema of the supraspinatus and infraspinatus muscles. (**b**) Sagittal T1-weighted MRI shows atrophy and fatty degeneration of the supraspinatus and infraspinatus muscles. Note the fatty degeneration is more severe for the supraspinatus muscle

The origin of this predominantly multifocal axonal damage to the brachial plexus is still poorly understood. Immune dysfunction remains the most plausible hypothesis in the presence of infectious or vaccine triggers, and in some cases of antimyelin antibodies.

Finally, some degenerative changes of the nerves and some inflammatory neuropathies remain of unknown origin (Figs. 10.13 and 10.14).

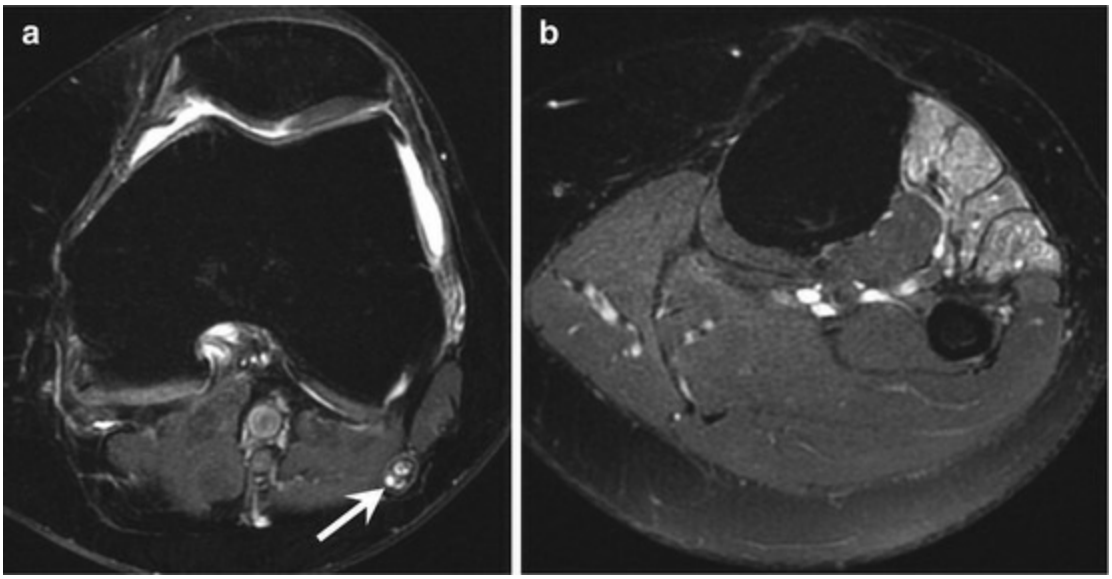

Fig. 10.13 Idiopathic fibrosis and cystic degeneration of the common peroneal nerve. (**a**, **b**) Axial fat-suppressed T2-weighted MRI show an enlargement and a cystic degeneration of the common peroneal nerve (*arrow*) and edema of the tibialis anterior, extensor digitorum, extensor hallucis longus muscles

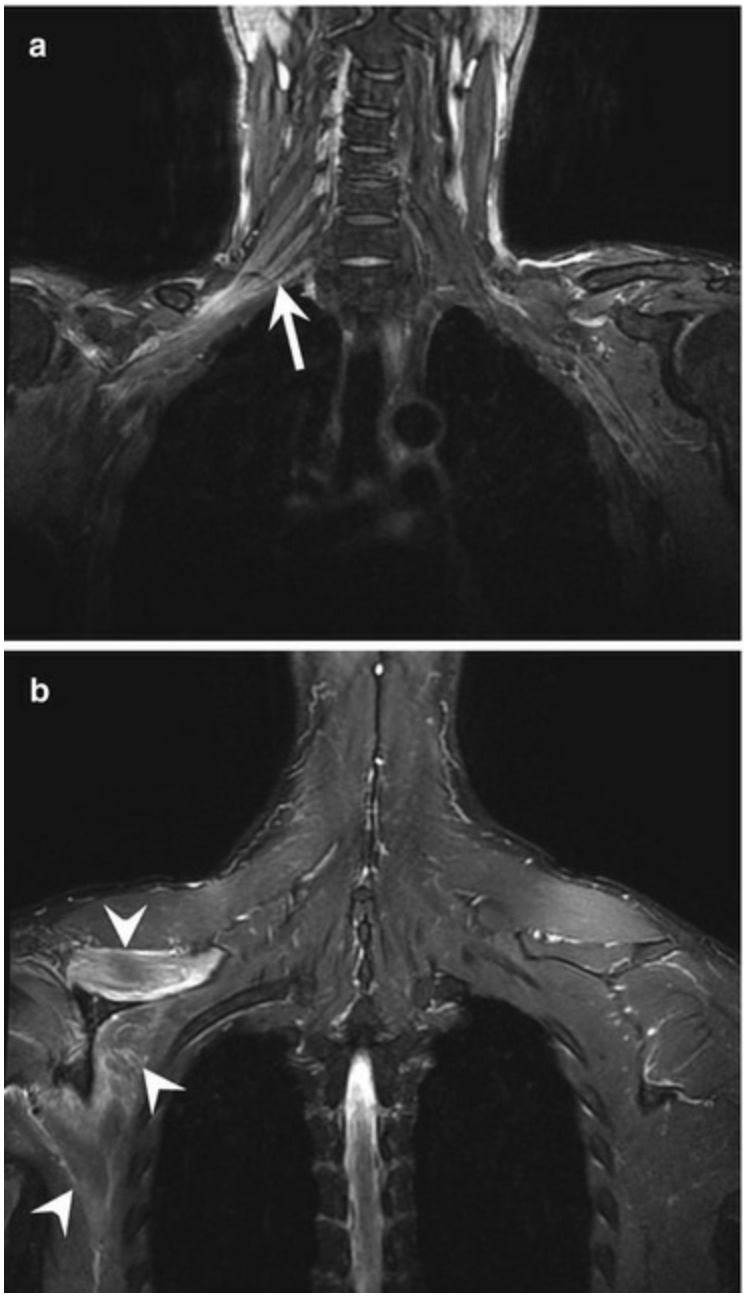

Fig. 10.14 A 37-year-old patient with inflammatory polyneuropathy of unknown origin. (**a**, **b**) Coronal water-imaging with a three-point Dixon sequence MRI show an enlargement of the right cervical roots (*arrow*) and a muscle edema of the muscles of the right scapular girdle (*arrowheads*)

10.4 Imaging Modalities of Neuropathies and Muscle Denervation

High-resolution ultrasound and magnetic resonance imaging (MRI) are the two imaging methods of choice for the study of peripheral nerves. Conventional radiography and CT scans have a more limited input. CT arthrography (with delayed acquisitions) may play a role to detect the joint anomalies associated with a ganglion cyst and show its

connection with the joint.

10.4.1 Ultrasound

High-resolution ultrasound is currently the imaging modality of choice for the examination of peripheral nerves, particularly because of the unrivaled spatial resolution it provides [29–31]. In addition, ultrasound is a noninvasive and low-cost technique and has two major advantages: It allows dynamic imaging (as in the assessment of subluxation of the ulnar nerve in the elbow) and analysis of the entire length of peripheral nerves along their anatomical course. The transducer frequency is chosen based on the size of the nerve and the depth of the anatomical region to be examined.

Using musculoskeletal presets and high frequency linear probes (10–17 MHz), the majority of peripheral nerve trunks, i.e. the median, radial, ulnar, sciatic, common fibular and tibial nerves, can be examined. However, nerves that are more proximal may be more difficult to analyze. The nerve is identified and its perineuronal environment studied on axial sections in which the peripheral nerve appears as an oval structure, consisting of a network of hypoechogenic fascicles separated by hyperechogenic septa. In longitudinal sections, it appears as a tube containing hypoechogenic bands separated by hyperechogenic lines (Fig. 10.15). The characteristic fascicular structure allows peripheral nerves to be distinguished from tendons, which have a fibrillar echo structure.

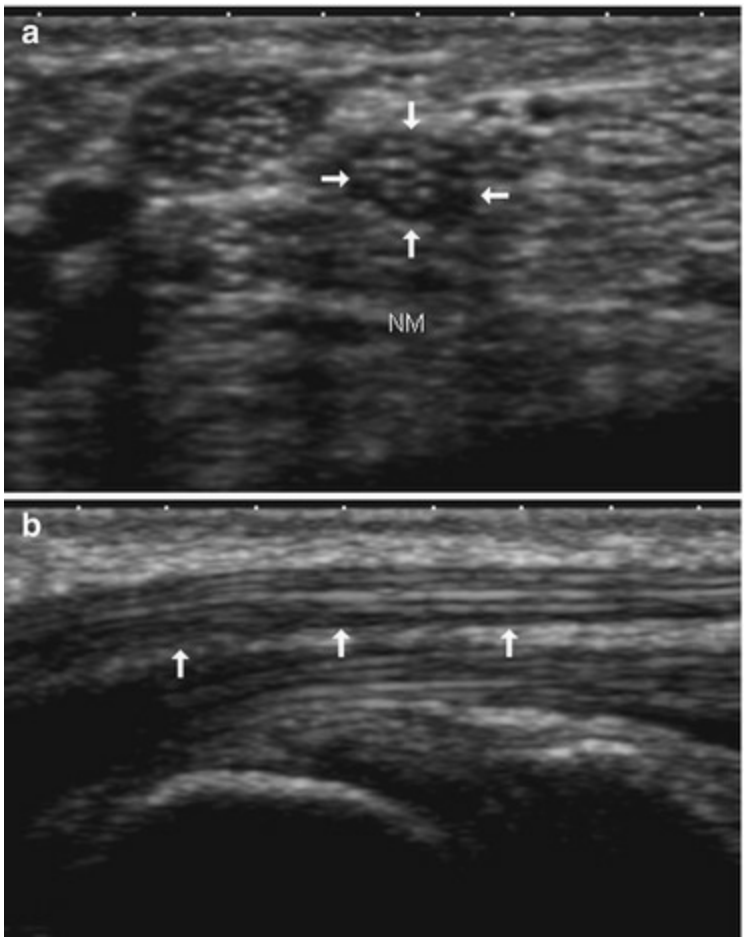

Fig. 10.15 Normal appearance of the median nerve. (**a**) Short axis ultrasound image shows the peripheral median nerve (*arrows*) as an oval structure, consisting of a network of hypoechogenic fascicles separated by hyperechogenic septa. (**b**) Longitudinal ultrasound image shows the median nerve (*arrows*) as a tube containing hypoechogenic bands separated by hyperechogenic lines

Ultrasound plays three roles in the diagnosis of entrapment syndromes [1, 32]:

- to look for nerve morphology abnormalities as a result of compression, which may produce two major signs:
  - segmental changes in diameter, usually focal thinning at the point of compression and downstream enlargement immediately after the compression; an increase in nerve diameter may also be seen proximal to the compressed area.
  - loss of the usual fascicular echo structure, the nerve becoming hypoechogenic and the fascicles are difficult to visualize.
- to identify lesions in the perineuronal environment responsible for compression: soft tissue tumors, musculoskeletal abnormalities such as synovial cysts, tenosynovitis or supernumerary tendons and vascular abnormalities (Figs. 10.4, 10.6, and 10.7).

- to guide cyst aspiration and/or corticosteroid injection.

Ultrasound may also depict the consequences of muscle denervation. On ultrasound, fatty degeneration and muscle atrophy appear as increased echogenicity of the muscle and resultant poor differentiation between the tendon and the muscle. Fatty atrophy also results in decreased muscle bulk. However, these anomalies are not specific and ultrasound is not able to depict muscle edema with any certainty.

Contrast-enhanced ultrasound could play a role in the future as animal studies have shown that it enables quantitative measurement of nerve perfusion and shows a significant signal enhancement of denervated muscle [33].

10.4.2 MRI

MRI is the most efficient technique for diagnosing neuropathies and entrapment syndromes. Improvements over the last couple of decades–high-field units, improved coil design and more robust sequences–have increased image quality with higher spatial resolution, better signal homogeneity and vascular inflow signal reduction, allowing better delineation of the nerves and an enhanced depiction of muscle edema associated with denervation.

10.4.2.1 Technical aspects

T1 sequences allow good identification of the peripheral nerves, which appear on cross-sectional images as numerous small hypointense dots (corresponding to the nerve fascicles) surrounded by high signal-intensity connective tissue (corresponding to the epineurium) that contains a certain amount of fat. With fat-suppressed T2-weighted sequences, peripheral nerves appear isointense to mildly hyperintense compared with normal muscle. Nerve fascicles, which contain endoneural fluid, may have slightly higher signal intensity than the surrounding connective tissue (Fig. 10.16). STIR, periodically rotated overlapping parallel lines with enhanced reconstruction (PROPELLER®, BLADE®) sequence and Dixon three-point sequence are very sensitive to fluid and may be used as an alternative to T2-weighted fast spin-echo sequences. MR neurography based on 3D volume acquisitions may also improve the depiction of nerves anomalies. It provides high quality isotropic images with the possibility of curvilinear multiplanar reconstructions and post-processing such as MIP or image fusion, which are particularly useful in examining complex anatomical structures like the brachial plexus [34–36].

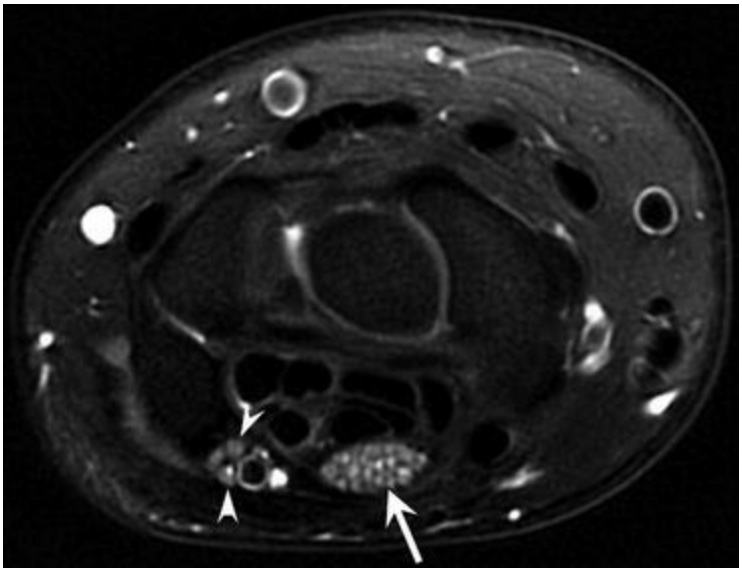

Fig. 10.16 Axial fat-suppressed T2-weighted MRI of the median nerve (*arrow*) and ulnar nerve (*arrowheads*). Peripheral nerves appear isointense to mildly hyperintense as compared with normal muscle. Nerve fascicles, which contain endoneural fluid, may have slightly higher signal intensity than the surrounding connective tissue

It is noteworthy that even with longer echo times, the magic angle phenomenon may increase the signal intensity of the nerves when the nerve fibers are oriented at an angle of about 55° to the constant magnetic induction field B0. However, neuropathic lesions are clearly distinguishable from an artificial increase of intraneural T2 by the magic angle effect [37, 38].

Diffusion tensor imaging, a technique for imaging anisotropy based on the measurements related to the molecular motion of water, provides information on peripheral nerves, which are characterized by an anisotropic arrangement of the nerve fibers. The molecular motion of water preferentially occurs along the axis of the nerve fibers, whereas it is much less in the direction perpendicular to this axis. Tractography (or fiber tracking) allows visualization of 3D fiber tracts via a mathematical representation (Fig. 10.17). Tractography could have clinical applications in entrapment neuropathies such as carpal tunnel syndrome and in peripheral nerves injuries [39–42].

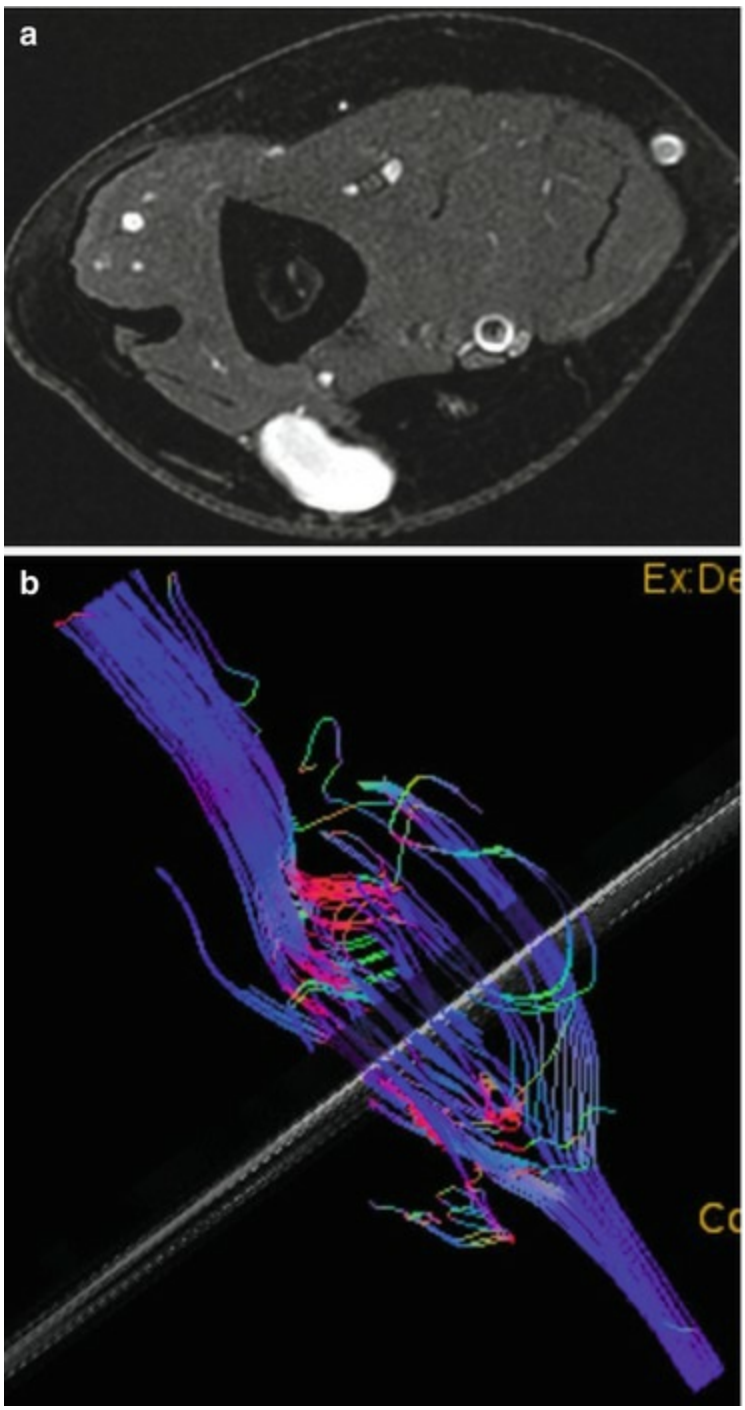

Fig. 10.17 Schwannoma of the ulnar nerve. (**a**) Axial fat-suppressed T2-weighted MRI shows the nerve is enlarged and hyperintense. (**b**) Tractography shows disorganization of the nerve fibers

MRI plays a major role in the diagnosis of entrapment syndromes by identifying nerve anomalies, lesions in the perineural environment and muscle denervation [43].

10.4.2.2 Pathological Features of the Morphology, Signal and Course of the Nerve

– flattening of the nerve, particularly in areas liable to be compressed, is abnormal. This sign is particularly valuable if the flattening is segmental and associated with an increase in diameter of the upstream nerve segment;

– an increase in diameter, particularly a segmental increase preceded and followed by a nerve of normal diameter, is also considered pathological;

– hyperintensity on "neurography" sequences: this is reported to be the result of decrease in axonoplasmic flow due to neuronal degeneration and peri- and endoneural edema;

– loss of the fascicular structure on T2-weighted sequences with fat suppression also indicates disease and is due to the same causes;

– moderate contrast enhancement on T1-weighted sequences after gadolinium injection, reflects a breach of the blood-nerve barrier;

– a deviation or change in direction of a nerve is evidence for compression or a pathological adhesion. It is therefore an excellent criterion to assess entrapment syndromes, particularly for recurrences or failures after surgery [1].

Note that MRI can be useful for the diagnosis of inflammatory polyneuropathies by demonstrating contrast uptake and increased diameter of the nerve roots [44]. MRI is also extremely useful for examining the perineural environment highlighting intrinsic or extrinsic compressive lesions [25, 45].

10.4.2.3 MRI of Muscle denervation

Finally, MRI is the only imaging technique that clearly shows the consequences of muscle denervation. Initially the muscle remains normal in size and morphology, with clear global hyperintensity on T2-weighted sequences. The edema is visible early, experimentally as soon as 48 h, and persists throughout the acute and subacute phase of denervation, usually for less than 10 weeks and very rarely for more than 6 months. The intensity of the T2 signal increase is reported to be proportional to the severity of nerve damage [3, 46, 47].

However, muscle edema may be missing on MRI when inappropriate sequences with insufficient sensitivity to fluid are used, when the image quality is poor or when the denervation is either minor or chronic (Table 10.1; Fig. 10.18).

Table 10.1 Causes of lack of muscle edema on MRI in compressive neuropathy

Compression of sensory branches only
Minor denervation
Chronic denervation
Poor image quality

Inappropriate sequence

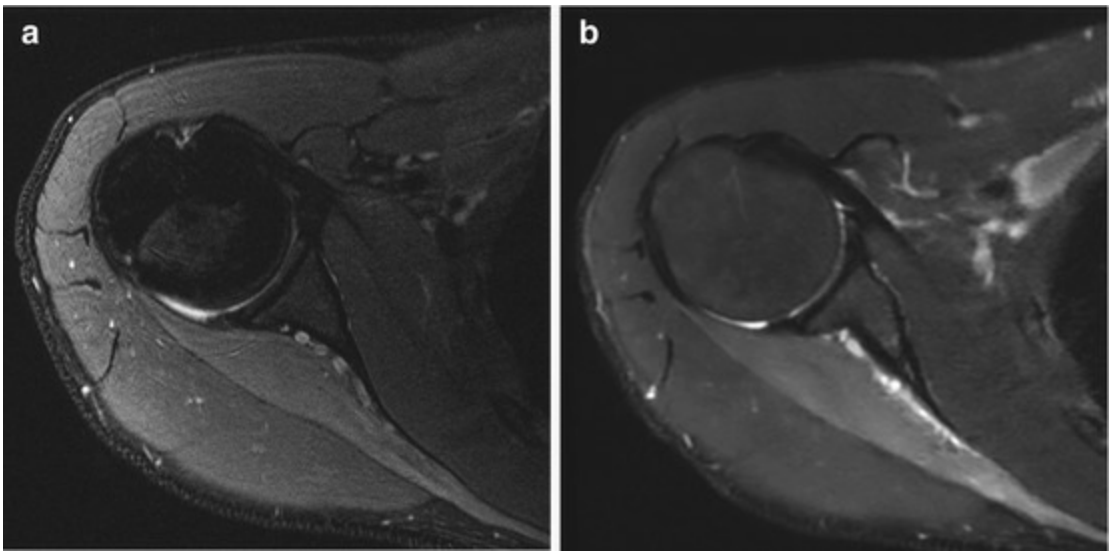

Fig. 10.18 A 29-year-old man with suprascapular nerve entrapment at the suprascapular notch. Axial (**a**) fat-suppressed and (**b**) water-image IDEAL T2-weighted MRI show muscle edema is more visible on image b for different reasons: the signal-to-noise ratio is better and the signal homogeneity is better. However, the spatial resolution is lower on image b

Muscle edema usually affects the entire muscle with the same intensity. In some situations, edema is more prominent around the myotendinous junction. The significance of this pattern is not clear. It may reflect increased vascularity and capillary permeability around the myotendinous junction in the subacute phase. In our experience, this finding is more frequent in Parsonage-Turner syndrome than in entrapment syndromes [25, 48]. Significant muscle enhancement after gadolinium injection is seen in this phase [2].

Secondarily, loss of muscle volume (muscle atrophy) associated with hyperintense T1-signal (fatty degeneration) is seen. This chronic phase of denervation, which occurs in the months following the initial lesion, persists if re-innervation does not occur. Uncommonly, muscle denervation is associated with muscle hypertrophy (Fig. 10.19) [49]. In Parsonage-Turner syndrome, acute and chronic muscle denervation may co-exist.

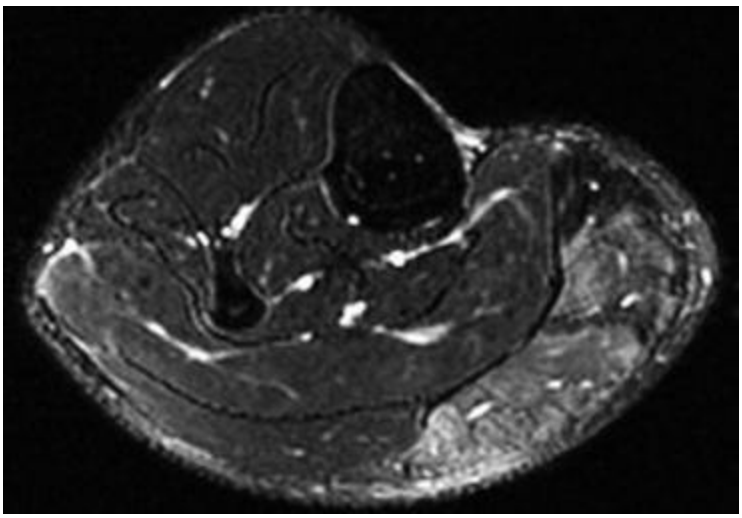

Fig. 10.19 Neurogenic muscle hypertrophy associated with an S1 radiculopathy due to a disk herniation (From Zabel et al. [49]). Axial fat-suppressed T2-weighted MRI of the calf shows enlargement, edema and fatty degeneration of the medial gastrocnemius

Finally, MRI can be used to map out denervated muscles and thus to diagnose and localize entrapment or compressive neuropathies when direct visualization of compressive anatomic structures is not possible with MRI or ultrasound [50–52].

10.5 Entrapment Neuropathies of the Shoulder

Entrapment neuropathies are relatively common, accounting for about 2 % of cases of sport-related shoulder pain. The most frequent one is the suprascapular nerve entrapment syndrome. Quadrilateral space syndrome, long thoracic nerve and accessory nerve neuropathies are less frequent. Parsonage-Turner syndrome is the main differential diagnosis [25, 46, 53, 54].

10.5.1 Suprascapular Neuropathy

Suprascapular neuropathy was first described by Thomas in the French literature in 1936 [55]. Suprascapular neuropathy is one of the most frequent manifestations of injury to the peripheral branches of the brachial plexus in athletes. It is particularly common among volleyball and tennis players, but other sports may also be implicated (notably throwing sports and weightlifting). Trauma is usually related to stretching and/or contusion of the nerve. Depending on the movement the patient made, nerve damage occurs either in the suprascapular notch or, more distally, in the spinoglenoid notch. Compression by a labral or mucoid cyst is also common. The site of compression determines the effect on the muscles [56–61].

10.5.1.1 Anatomy

The suprascapular nerve is a peripheral mixed sensorimotor nerve arising from the superior trunk of the brachial plexus (C5–C6) and in 15–22 % of cases C4. It provides motor innervation of the supraspinatus and infraspinatus muscles, and sensory branches innervate the subacromial bursa, acromioclavicular and glenohumeral joints and sometimes the lateral shoulder. Downstream from its origin in the brachial plexus, the suprascapular nerve crosses the posterior cervical triangle and then the deep face of the trapezius muscle in the direction of the suprascapular notch, which it crosses to enter the supraspinous fossa.

The suprascapular notch varies considerably in size and shape, and Rengachary has classified six types [62–64]. A U-shaped form is most common and least likely to damage the suprascapular nerve. Narrow (9 % of cases) or closed (4 %) notches are theoretically more pathogenic but this has not been proven (Fig. 10.20).

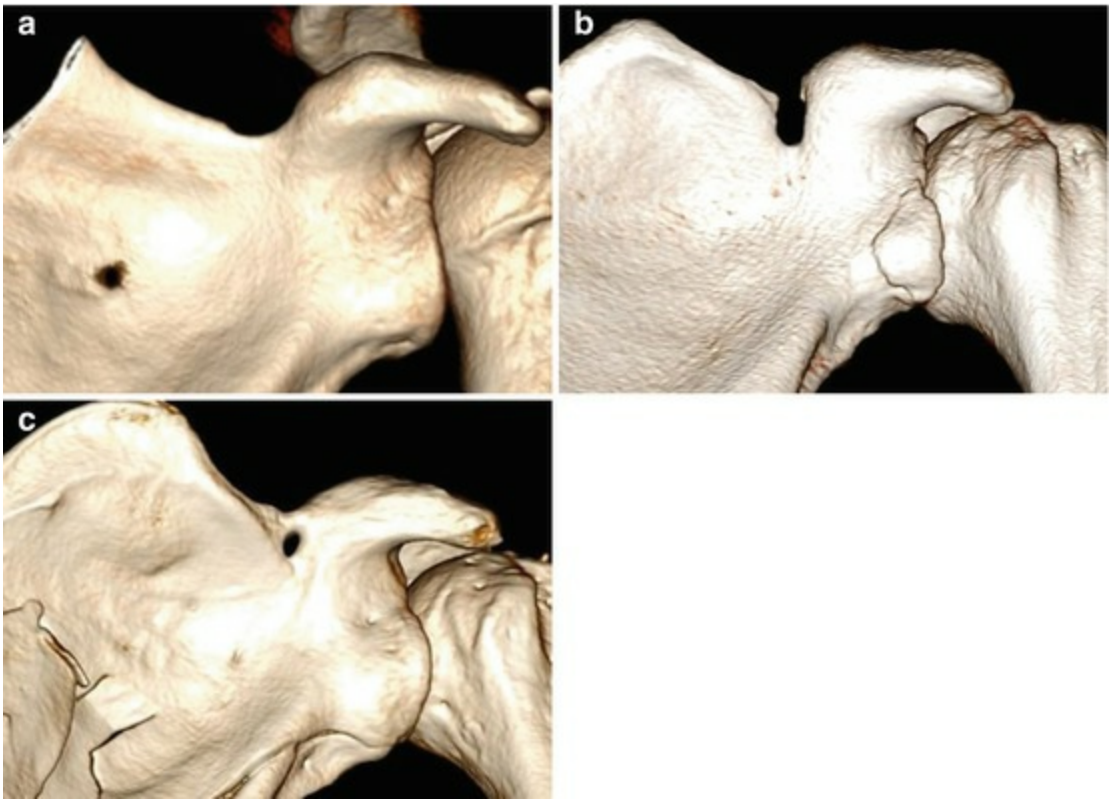

Fig. 10.20 Volume rendering technique show the great morphological diversity of the suprascapular notch in patients without neuropathy. (**a**) flared notch (**b**) indented notch (**c**) closed notch (From Blum et al. [25])

In most cases, one or two branches of the nerve provide motor innervation for the supraspinatus muscle 1 cm after its emergence from the channel. These branches sometimes originate upstream from the suprascapular notch. If so, they pass into the suprascapular notch and accompany the main trunk of the nerve. More rarely, they pass over the transverse scapular ligament. The superior articular sensory branch originates

before the suprascapular notch and crosses it by accompanying the principal trunk. It provides sensory innervation of the coracoacromial and coracoclavicular ligaments, the subacromial bursa and the acromioclavicular joint.

After the suprascapular notch, the main trunk of the suprascapular nerve crosses the supraspinatus fossa under the corresponding muscle and travels downwards and outward in the direction of the spinoglenoid notch located in the lateral edge of the spine of the scapula. This nerve is more or less flat against the bone and passes under the fascia of the supraspinatus. Before reaching the spinoglenoid notch, a sensory nerve branches off to the posterior wall of the glenohumeral joint capsule. The nerve passes this notch and then takes a route between the medial scapula and the deep face of the infraspinatus muscle while splitting into a number of motor branches to the infraspinatus. It is accompanied in that course by suprascapular vessels that also pass into the spinoglenoid notch. Beyond the spinoglenoid notch, the nerve makes an acute medial turn around the base of the scapular spine, traveling along the scapular body and sending two or more branches into the infraspinatus muscle.

Broadly, the nerve takes an erratic course with three fixed points: the cervical origin, the suprascapular notch and the spinoglenoid notch. At the suprascapular notch, it innervates the supraspinatus and infraspinatus muscles. One centimeter later, its main trunk innervates only the infraspinatus muscle.

10.5.1.2 Pathophysiology

The suprascapular nerve can be directly compressed by a labral cyst, a mucoid cyst or a tumor. It can also be injured during trauma (fractured scapula) or, exceptionally, during surgery.

In an athlete, it is usually the result of entrapment at the suprascapular notch or the spinoglenoid notch (Fig. 10.1) [46, 58, 65]. The origin of trauma is multifactorial. Repetitive movement and harsh or extensive movements of the scapula put the nerve under tension and injury occurs about one of the two fixed points. Inflammation and resulting swelling make it even more vulnerable, creating a vicious circle. Anatomical features that predispose to trauma include a narrow suprascapular notch with sharp edges and a thick and rigid transverse scapular ligament. In the spinoglenoid notch, nerve entrapment may be due to increased tension of the spinoglenoid ligament in adduction and internal rotation, which corresponds to the follow-through phase of throwing. Entrapment at the spinoglenoid notch can also cause intimal lesions of the suprascapular artery and be responsible for microemboli in the vasa nervorum of the suprascapular nerve. In most cases, the nerve is injured at the scapular notch. Nerve injury at the spinoglenoid notch has been described principally in volleyball players [58, 66, 67].

After authentic entrapment syndromes, a mucoid cyst is the lesion most frequently

implicated in suprascapular nerve compression. Cysts develop from a labral tear, and a superior cyst is normally associated with a superior labral tear from anterior to posterior (SLAP lesion) or a posterosuperior impingement syndrome. A posterior cyst is related to either occult posterior instability or glenohumeral degeneration with rupture of the posterior labrum (Fig. 10.4). Rarely, cysts grow at the expense of the joint capsule with an intact labrum.

In the vast majority of cases, nerve damage at the scapular notch causes denervation of the supraspinatus and infraspinatus muscles. More distal truncal impairment leads to isolated involvement of the infraspinatus. Compression downstream of the spinoglenoid notch by an extrinsic factor can affect only some of the terminal branches of the nerve and cause a partial denervation of the infraspinatus muscle.

10.5.1.3 Diagnosis

The diagnosis is often delayed as the symptoms are not specific and pain is usually moderate or even absent. Proximal injury is more painful, probably due to involvement of sensory branches. Isolated infraspinatus muscle atrophy or combined supraspinatus and infraspinatus muscle atrophy are the most relevant signs, especially in patients practicing overhead sports activities. C5 or C6 neuropathy and lesions affecting the rotator cuff tendons are important to eliminate.

The diagnosis can be confirmed by EMG but MRI is the key examination to confirm the diagnosis (muscle edema, atrophy, fatty degeneration) and determine the cause and location of compression. An isolated edema of the infraspinatus muscle is related to a spinoglenoidal notch nerve compression whereas a combined edema of the supraspinatus and infraspinatus muscles indicate compression of the nerve at the suprascapular notch. In case of entrapment syndrome, no other lesion is identified. However, suprascapular nerve compression by a synovial or a ganglion cyst is also frequent. Note that not all cysts are responsible for nerve compression and their identification with ultrasound or CT arthrography does not imply suprascapular nerve neuropathy. Finally, MRI usually demonstrates the connection with the labral tears in case of a SLAP lesion, a postero-superior impingement or a posterior glenohumeral instability.

10.5.1.4 Treatment

Treatment is primarily medical. Evidence of a cyst causing compression may prompt percutaneous puncture-infiltration guided by ultrasound or CT but this approach is efficient in only about 50 % of cases. When the content of the cyst is very thick and viscous, its removal may be difficult (and corticosteroids are less effective). Besides, some cysts can recur. In all cases, functional rehabilitation must be undertaken early. Surgical intervention is considered when medical treatment fails (less than 40 % of

cases). Identifying the region of compression is very helpful for planning surgery, for which open and arthroscopic techniques may be considered.

10.5.2 Axillary Neuropathy and the Quadrilateral Space Syndrome

Quadrilateral space syndrome is a rare chronic impingement syndrome described by Cahill in 1980; it affects the axillary nerve plus the posterior circumflex humeral artery in the quadrilateral space compressed by intermuscular fibrous bands [68]. The axillary nerve may also be compressed by a posteroinferior labral cyst [69, 70]. These compressive phenomena should be distinguished from acute trauma to the axillary nerve occurring during acute anterior dislocation or fracture of the humeral head.

10.5.2.1 Anatomy

The axillary nerve is a terminal branch of the posterior cord of the brachial plexus and derives from the ventral rami of C5 and C6. It runs obliquely across the inferolateral border of the subscapularis and passes through the quadrilateral space accompanied by the posterior humeral circumflex artery. The axillary nerve is the most superior structure in the space. In most cases, the axillary nerve divides in the quadrilateral space into anterior (superior), and posterior (inferior) branches [71]. The anterior branch takes a tortuous path around the surgical neck of the humerus and supplies the anterior and middle parts of the deltoid muscle. The posterior branch provides motor innervation to the subscapular muscle, the teres minor muscle, and often the posterior portion of the deltoid, plus sensory innervation of the posterolateral shoulder.

10.5.2.2 Pathophysiology

In the quadrilateral space syndrome, the posterior humeral circumflex artery and the axillary nerve could be compressed by intermuscular fibrous bands. The origin of these bands is unknown but they could be induced by sport-related trauma. The axillary nerve or its branches may also be compressed by a hematoma, a tumor, a posteroinferior labral or paralabral cyst (between 6 and 9 o'clock), a bony callus or hypertrophy of the muscles bounding the quadrilateral space. Other cases of axillary nerve injury involve trauma (humeral neck fracture, acute glenohumeral dislocation) and surgical or arthroscopic intervention.

10.5.2.3 Diagnosis

The quadrilateral space syndrome predominantly affects younger patients and athletes (20–35 years) involved in throwing sports. The presentation is essentially one of

subacute pain and paresthesia of the shoulder. The pain is exacerbated when the arm is held up in abduction and external rotation for one minute. Skin paraesthesia in the sensory distribution of the axillary nerve may also occur. Deltoid atrophy is rarely present. Neurological examination is normal.

EMG is often normal and currently, diagnosis is determined with MRI by the discovery of edema of the teres minor muscle, isolated or associated with deltoid muscle edema. MRI may also demonstrate an inferior labral cyst or a compressive lesion responsible for nerve lesion (Figs. 10.8, 10.21, and 10.22). The origin of isolated atrophy and fatty degeneration of the teres minor (without any muscle edema) remains debatable and anyway is not associated with clinical symptoms.

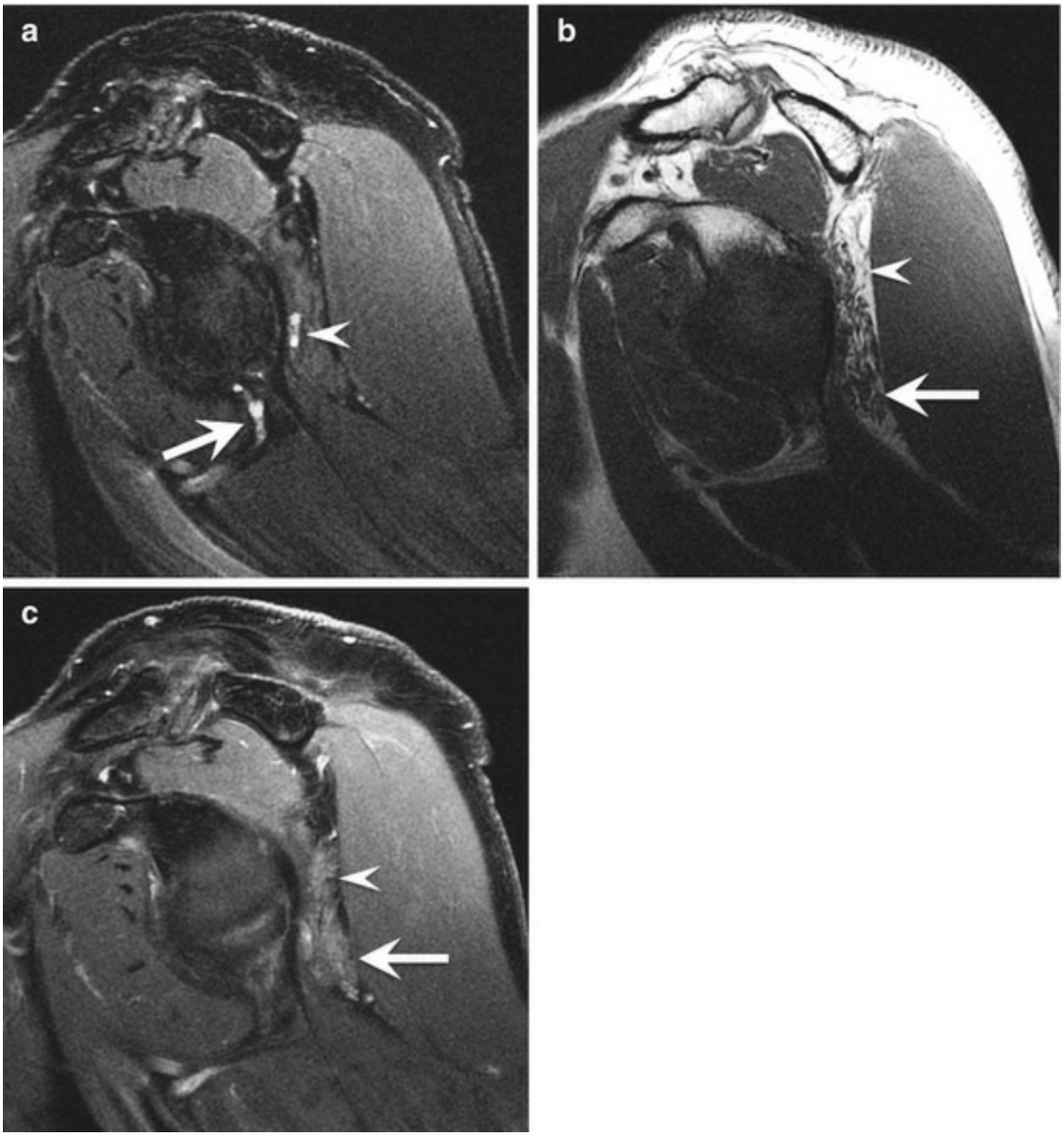

Fig. 10.21 Suprascapular nerve and axillary nerve neuropathies due to labral cysts. (**a**) Sagittal fat-suppressed T2-weighted MRI shows two labral cysts, the lower (*arrow*) extending into the quadrilateral space, the posterior (*arrowhead*) extending medially towards the spinoglenoid notch. (**b**) Sagittal T1-weighted MRI shows infraspinatus

(*arrowhead*) and teres minor (*arrow*) muscles atrophy and fatty degeneration. (**c**) Sagittal fat-suppressed contrast-enhanced T1-weighted MRI shows mild hypervascularization of the infraspinatus (*arrowhead*) and teres minor (*arrow*) muscles

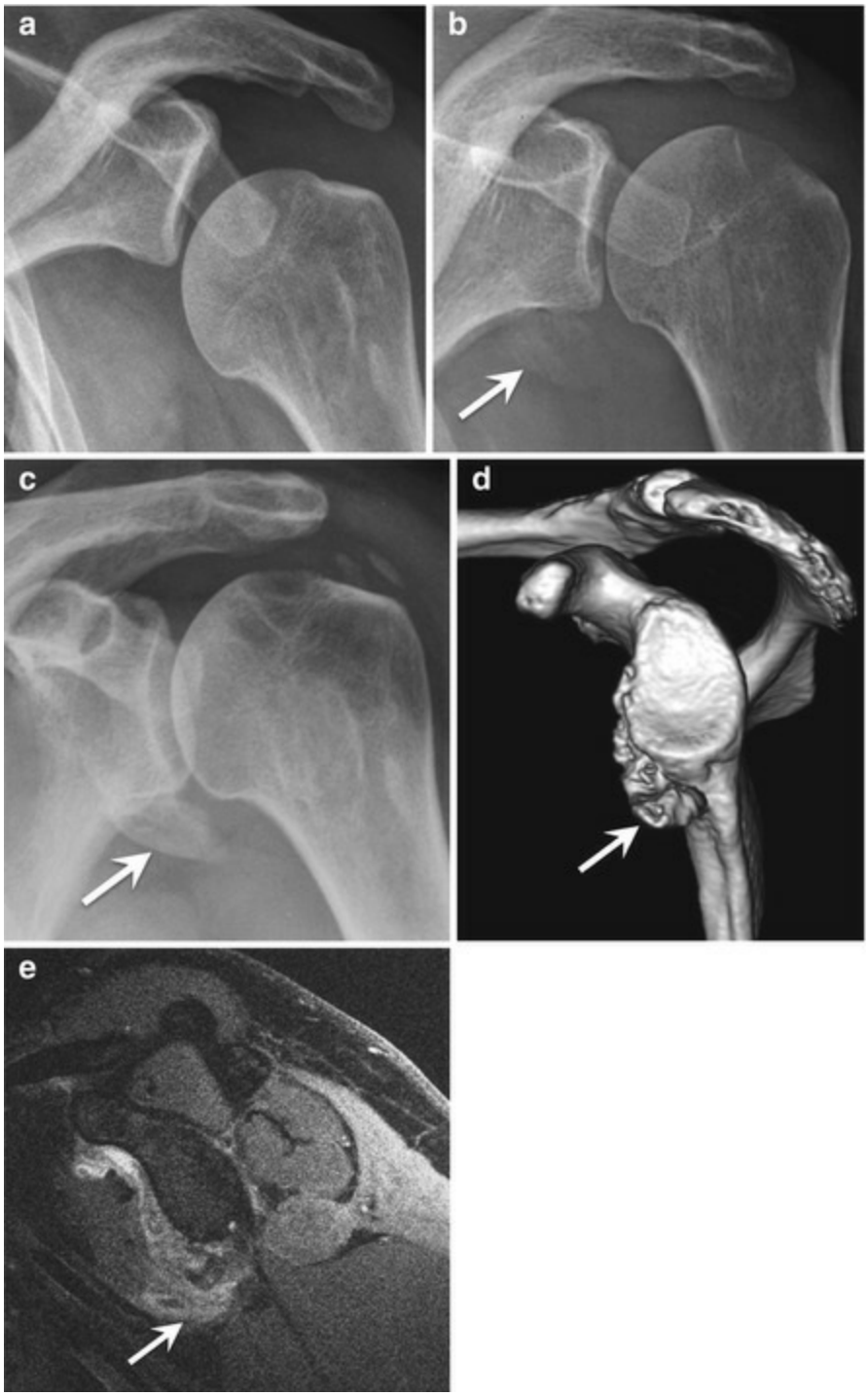

Fig. 10.22 A 46-year-old man after an anterior glenohumeral dislocation and subsequent myositis ossificans of the subscapularis muscle. (**a**) Anteroposterior radiograph after dislocation reduction shows no glenoid fracture. (**b**) Anteroposterior radiograph 1 month after the trauma shows a fuzzy ossification under the scapular neck (*arrow*). (**c**) Anteroposterior radiograph 4 months after the trauma shows ossification at the anteroinferior portion of the scapular neck (*arrow*). (**d**) 3D CT image 4 months after the trauma shows the extent of the ossification under the scapular neck (*arrow*). (**e**) Sagittal fat-suppressed T2-weighted MRI shows the extension of the myositis ossificans in the

quadrilateral space (*arrow*) and some muscle edema of the deltoid and teres minor muscles

10.5.2.4 Treatment

Treatment is essentially medical, primarily NSAIDs and corticosteroid injections, and physiotherapeutic.

10.5.3 Neuropathy of the Long Thoracic Nerve

Damage to the long thoracic nerve paralyzes the anterior serratus muscle, which generates a medial winging of the scapula and a deficit in active forward flexion. It has been described in practitioners of many sports [24, 72–74].

10.5.3.1 Anatomy

The anatomy of the long thoracic nerve (or Charles Bell nerve) is not fully elucidated. It is a pure motor nerve exclusively responsible for innervation of the anterior serratus muscle. It arises from the anterior branches of roots C5, C6, often C7 and less commonly C4. The long thoracic nerve then dives deep to the brachial plexus and the clavicle to pass over the first rib within the axillary sheath. At the posterior angle of the second rib, the nerve is embedded in the fascia of the serratus anterior muscle and descends inferiorly between the middle and posterior axillary lines to innervate the serratus anterior muscle. There are two recognized critical points for the long thoracic nerve: the scalenus medius muscle, and the serratus anterior fascia at the level of the second rib.

10.5.3.2 Pathophysiology

Damage to the long thoracic nerve has been described in many sports including weightlifting, bodybuilding, basketball, squash, tennis, javelin throwing, swimming, and American football. The responsible mechanisms are controversial, but excessive traction seems to be incriminated in most cases. Head rotation and lateral bending of the neck opposite the arm that performs an elevation movement, cause elongation of the nerve between its two fixed points: the middle scalenus and its attachment to the anterior upper serratus muscle.

Long thoracic nerve neuropathy may also be related to prolonged carrying of a heavy load or a direct blow to the shoulder. In addition, it has been described after anesthesia (related to positioning) and after surgery.

10.5.3.3 Diagnosis

Pain and functional impairment are variable, but the deficit in active forward flexion of

the arm past the horizontal is relatively consistent. The diagnosis may be made clinically. Upon physical examination, classical or medial scapular winging is usually evident at rest, with the medial and inferior borders closer to the spine and lifted superiorly when compared to the normal side (Tables 10.2 and 10.3) [24]. This detachment is more pronounced at the beginning of elevation. In milder forms, the most frequent, it appears when the patient pushes against a wall with a "push-up" movement.

Table 10.2 Classification of scapular winging (From Kuhn et al. [73])

Primary scapula winging	
Neurologic origin	Spinal accessory nerve (trapezius palsy) Long thoracic nerve (serratus anterior palsy) Dorsal scapular nerve (rhomboids palsy)
Osseous origin	Osteochondromas Fracture, malunions
Soft-tissue origin	Contractural winging Muscle avulsion or agenesis Scapulothoracic bursitis
Secondary scapula winging	Accompanies glenohumeral disorders and should resolve once that disorder has been addressed
Voluntary scapula winging	

Table 10.3 Neurogenic causes of scapular winging (Adapted from Martin and Fish [24])

	Medial winging	**Lateral winging**	**Lateral winging**
Injured nerve	Long thoracic	Spinal accessory	Dorsal scapular
Muscle palsy	Serratus anterior	Trapezius	Rhomboids
Physical exam	Arm flexion; push-up motion against a wall	Arm abduction; external rotation against resistance	Arm extension from full flexion
Position of the scapula compared to normal	Entire scapula displaced more medial and superior	Superior angle more laterally displaced	Inferior angle more laterally displaced

Diagnosis is clinical. It is confirmed by EMG and possibly by MRI, which shows signs of denervation (edema, atrophy) of the anterior serratus muscle (Fig. 10.23). The serratus anterior muscle is sometimes outside the usual field of exploration of shoulder MRI and standard joint examination may miss the anomalies. A shoulder coil with a large field is sufficient in thin patients. A spine coil and a multi-element body coil give results that are more satisfactory.

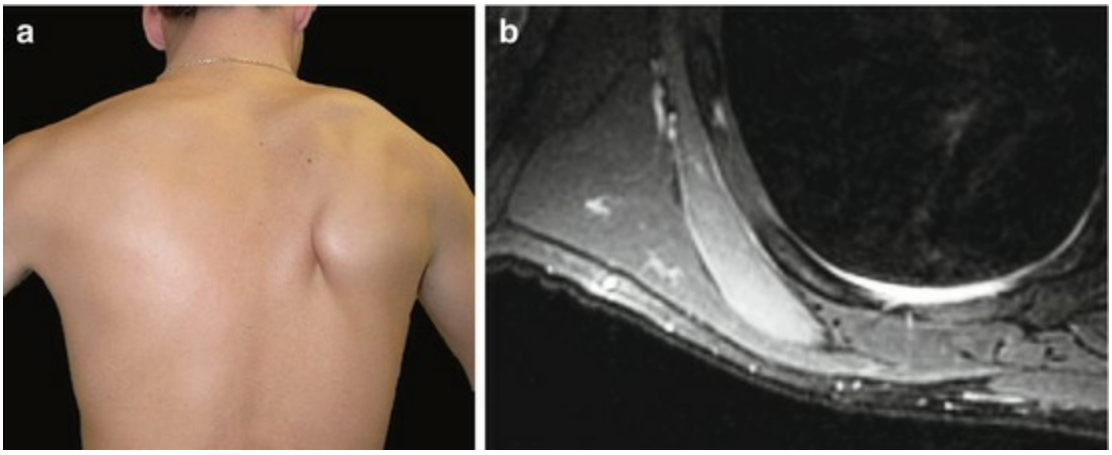

Fig. 10.23 A football (soccer) goalkeeper following a fall on the apex of the shoulder and resultant right long thoracic neuropathy. (**a**) Photograph of the patient shows medial winging of the right scapula. (**b**) Axial fat-suppressed MRI confirms the diagnosis and reveals edema of the right anterior serratus muscle

10.5.3.4 Treatment

As with other neuropathies, treatment is essentially medical. Prognosis is favorable in 80 % of cases, with an average duration of 8 months (range 1–24 months). When medical treatment fails, and in severe forms, surgical stabilization of the scapula on the thorax should be considered.

10.5.4 Accessory Nerve Neuropathy

Neuropathy of the accessory nerve (spinal) is mainly iatrogenic. It may also be due to direct trauma to the posterior cervical region or, uncommonly, sporting activity [72, 75, 76].

10.5.4.1 Anatomy

The accessory nerve provides motor innervation to the trapezius and sternocleidomastoid muscles. Its lateral branch emerges from its posterior border at C3–C4. It then crosses the posterior cervical triangle to reach the trapezius.

10.5.4.2 Pathophysiology

The lateral branch of the accessory nerve is particularly vulnerable in the posterior cervical triangle. A lesion at this level leads to paralysis of the trapezius muscle. By far the most common cause of spinal accessory nerve palsy is iatrogenic damage, primarily sustained during surgery for cervical lymph node biopsy and cervical mass excision. Sports activities are rarely responsible for spinal accessory nerve palsy. The mechanism may involve stretching or compression.

10.5.4.3 Diagnosis

Accessory neuropathy has been described in wrestlers (crossface), weightlifters, and hockey players (direct impact). Complete paralysis of the trapezius profoundly alters shoulder function, but there are many well-tolerated partial forms. Patients present with non-specific shoulder pain and a deficit in arm abduction. Displacement of the scapula is always present, but depends on the extent of muscle denervation. Typically, winging is minimal and is accentuated during arm abduction, with the scapula moving upwards with the superior angle more lateral to the midline than the inferior angle (Tables 10.2 and 10.3) [24].

The EMG is difficult to interpret, especially in the first month. MRI is indicated in difficult cases to confirm the diagnosis. Centered on the trapezius and anterior serratus muscles, it reveals classic signs of denervation affecting the trapezius and allows for elimination of long thoracic neuropathy (Fig. 10.24).

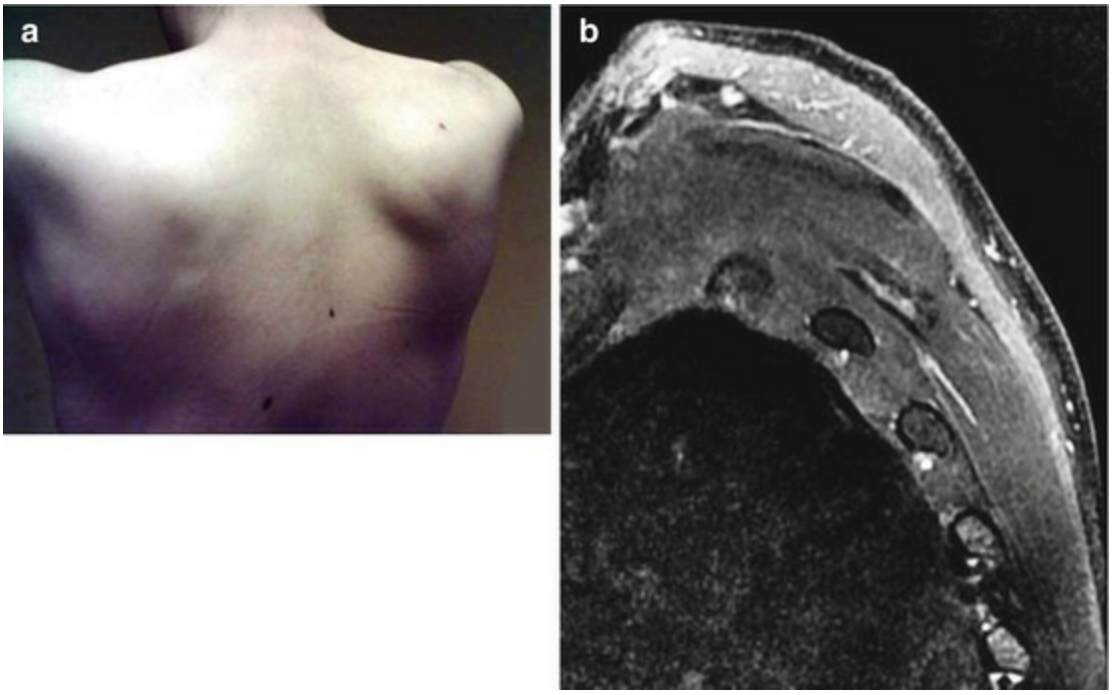

Fig. 10.24 A 43-year-old patient with neuropathy in the left accessory nerve. (**a**) Photograph of the patient shows left scapular winging. (**b**) Strictly oblique sagittal fat-suppressed T2-weighted MRI of the shoulder shows edema of the trapezius

10.5.4.4 Treatment

Treatment is functional. Evolution is often favorable over 6–18 months.

10.5.5 Dorsal Scapular Nerve Neuropathy

Weakness of the greater and lesser rhomboid muscles is a rare source of scapular

winging but these lesions are probably under diagnosed. Most of them are related to an entrapment syndrome, probably in the scalenus medius muscle [24, 73, 77].

10.5.5.1 Anatomy

The dorsal scapular nerve originates from the C5 spinal nerve. It pierces the middle scalenus muscle and travels posteriorly between the posterior scalenus muscle and the serratus posterior superior and levator scapulae muscles. The dorsal scapular nerve innervates the rhomboid major and minor muscles and, occasionally, the levator scapulae.

10.5.5.2 Pathophysiology

Dorsal scapular nerve entrapment may develop through various mechanisms.

In general, dorsal scapular nerve entrapments are attributed to the hypertrophy of the middle scalenus muscle. Trauma of the dorsal scapular nerve could be due to the stretching of scalenus muscles such as whiplash injury. It may be associated to lesions of the suprascapular nerve or of the long thoracic nerve, which has an adjacent course.

10.5.5.3 Diagnosis

Entrapment of the dorsal scapular nerve is easily overlooked as a cause of upper-arm pain. Rhomboid weakness produces a very subtle winging of the scapula, with the scapula laterally translated and the inferior angle rotated laterally (Tables 10.2 and 10.3). The winging is increased with overhead elevation of the humerus. MRI shows the denervation of the rhomboid muscles as long as the acquisition is centered on the right anatomical region. A standard shoulder exploration will fail to show any anomaly (Fig. 10.25).

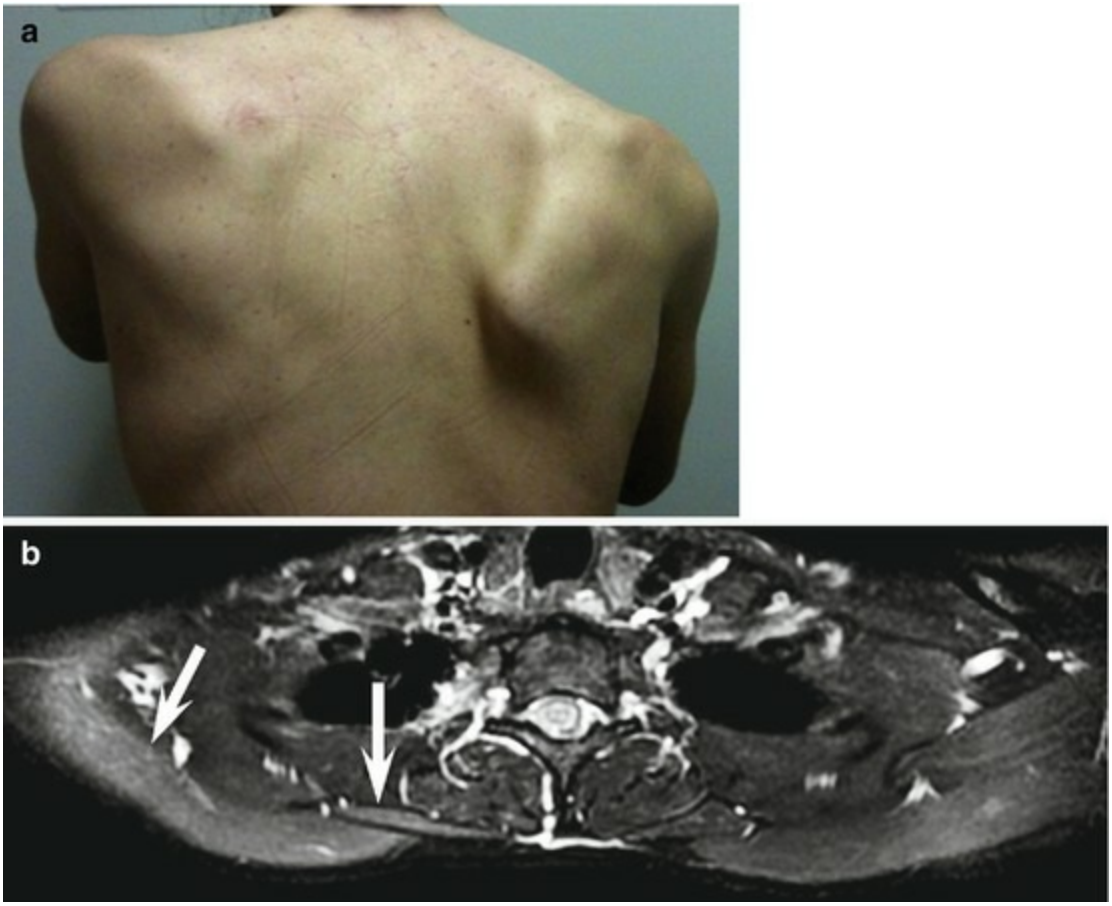

Fig. 10.25 A 19-year-old man with Parsonage-Turner Syndrome. (**a**) Photograph shows right scapular winging. (**b**) Axial water-imaging IDEAL MRI shows edema in the right trapezius and rhomboid muscles (*arrows*)

10.5.5.4 Treatment

Treatment is based on rehabilitation. Some authors suggest a surgical decompression of the dorsal scapular nerve by transecting the scalene anterior and medius muscles.

10.5.6 Proximal Neuropathy of the Musculocutaneous Nerve

Proximal neuropathies affecting only the musculocutaneous nerve are rare [78].

10.5.6.1 Anatomy

The musculocutaneous nerve is a major peripheral nerve of the upper limb. It arises from the lateral (secondary anterolateral trunk) of the brachial plexus. Its origin is C5, C6 and sometimes C4 or C7. In the axilla, it is situated outside the median nerve and the axillary artery. Then it travels downward and outward before entering the coracobrachialis muscle. It runs between the bundles and then passes between the brachialis in the back and the biceps in the front and along the lateral biceps to reach the external bicipital groove. In its course, it branches to the coracobrachialis, biceps and

brachial muscles. Its terminal branches are purely sensory (anterior, posterior and lateral forearm). There are numerous anatomical variations.

10.5.6.2 Pathophysiology

During violent extension of the arm (throwing sports), the musculocutaneous nerve can be stretched between two fixed points: its origin and where it enters the coracobrachialis muscle. In sports such as cycling, rowing, and weightlifting, prolonged isometric contraction of the coracobrachialis muscle may compress the musculocutaneous nerve and/or associated vessels and generate nerve damage.

10.5.6.3 Diagnosis

Pain and functional impairment are negligible. The patient presents with impaired strength of flexion and supination of the forearms, wasting of the muscles of the anterior arm, radial hypoesthesia of the forearm and abolished biceps reflex. The presence of anastomoses of the median/musculocutaneous nerves helps to explain the extension of altered sensitivity in the sensory median nerve territory.

The clinical picture simulates C6 nerve root involvement. Differential diagnosis includes distal bicep rupture. The diagnosis is confirmed by EMG. MRI is of limited value. It may show edema of the coracobrachialis, brachialis and brachoradialis muscles.

10.5.6.4 Treatment

In cases that relate to compression on effort, the course is usually favorable without the need for surgical treatment.

10.6 Conclusion

Muscle denervation is the consequence of the Wallerian degeneration of a motor nerve. The denervated muscle fiber will at first develop a vasogenic edema. These abnormalities are seen on EMG and with MRI at 48 h. MRI is the key examination to reach the positive diagnosis of shoulder neuropathy, determine the location of entrapments, and highlight intrinsic or extrinsic compressive lesions.

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Part III
Extrinsic Muscular Injury

Bernard Roger, Ali Guermazi and Abdalla Skaf (eds.), Muscle Injuries in Sport Athletes, Sports and Traumatology,
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11. Muscle Contusions: Extrinsic Muscle Lesions

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Abstract
Muscle contusions or extrinsic muscle lesions are frequently encountered in sports medicine; indeed they represent the second most common cause of morbidity in athletes. Participation in contact sports is a major risk factor. Because of the high frequency and the severity of muscle contusions, some of the governing bodies in some sports, like American football and ice hockey, have implemented prevention strategies and changed their regulations by authorizing helmets and protective padding. However, there is no validated scientific evidence to prove that these measures provide any meaningful benefit. The site of the muscle contusion depends on the type of sport, with the lower limb being the most commonly cited location in the literature. Despite its high incidence, there is no evidence from studies human on the optimal treatment. In the literature, contusions of the quadriceps muscle are the most well studied. On the other hand, numerous animal studies have shed light on the pathogenesis and the healing process of muscle lesions. The healing process must be clearly understood to provide the most efficient treatment plan. Moreover a thorough clinical examination associated with imaging is required. A detailed grading system of the images helps to develop the optimal treatment strategy.

11.1 Introduction

Muscle contusions or extrinsic muscle lesions are a frequent injury in sports medicine,

as a result of direct trauma or hit. They are the second leading cause of morbidity in athletes [1]. In some muscle groups, like the quadriceps, they are the second most common type of lesion, after intrinsic muscle lesions [1].

Participation in contact sports is a major risk factor. Soccer, probably the most popular game around the world, is a major source of muscle contusions in the lower limb, commonly known as a “charley horse” or “cork thigh”. In epidemiological studies on ice hockey, muscle contusions made up half of the injuries in a season. In rugby, the number of tackles is strongly correlated with an increase of muscular protein in blood, a sign of muscle cell damage [2]. Because of the high incidence and the severity of muscle contusions, some of the governing bodies of sports, like American football and ice hockey, have implemented prevention strategies and changed their regulations by authorizing helmets and protective padding. Meanwhile there is no scientific evidence to show any real benefit from these precautions [4]. Fighting sports (with or without weapons), sports with equipment and outdoor sports also present a risk of extrinsic muscle injuries.

The site of the muscle contusion depends of the sport with the lower limb as the most cited in the literature. For the quadriceps, the vastus lateralis and especially the vastus intermedius are commonly injured. For the triceps suralis, the gastrocnemius lateralis and the soleus are the most injured. At the level of the upper limb, the brachialis and deltoid muscles may be affected [5].

Despite its high incidence, there is no scientific evidence from studies in humans to indicate the optimal treatment. In the literature, contusions of the quadriceps are the most studied and the management is usually the same as at other sites [7, 11]. On the other hand, animal studies shed some light on the pathogenesis and healing of muscle lesions [11].

11.2 Types and Mechanism

Muscle contusions can be divided into two main types: with or without breach of the skin Extrinsic muscle lesions with breach of the skin are less common and follow an impact with contending surface like spikes, studded shoes and ice skates. Depending on the kinetics and the force applied during the impact, the sub-cutaneous tissue, the aponeurosis and then the muscle fibers may be lacerated. Muscle contusion without breach of the skin is usually induced by a direct hit from an opponent (elbow, knee), from sports equipment (hockey stick), a ball thrown at a high velocity [9] or from outdoor hazards.

Animal studies have shown that contusion injuries result from two concomitant debilitating factors: a direct crush on the underlying tissues and an indirect propagation of forces creating a deep compression of tissues on bony structures [6]. Aggravating risk factors have been highlighted; the seriousness of the muscle lesion depends on the

kinetic energy and the surface of the wounding object. The smaller the impact the worse the lesions are [6]. Crisco et al. showed that a contracted muscle is better able to absorb and to disseminate the received energy than a resting or passively stretched muscle [11]. Contraction of a muscle during the impact should be seen as a protective factor despite some common wide-spread beliefs [6, 16]. Finally, muscle fatigue also seems to be a risk factor for severe muscle injuries.

11.3 Pathogenesis

11.3.1 Injury Phase

The impact causes reversible and irreversible damage to the muscle and connective tissues. Due to the sudden increase in the local pressure, the vascular obstruction and the acute temporary ischemia, some tissue damage will be reversible within 24 h [12]. But some definite lesions with local necrosis are seen due to contracture of the myofilaments and tears associated with intercellular edema. Connective tissue like the peripheral aponeurosis or intra-muscular aponeurosis may also be injured. Blood vessels expanding through the connective tissue may be ruptured and hematoma may develop [13]. Muscle hematoma may be either intramuscular or intermuscular [9]. A precise analysis of the connective tissue may be of help in the prognosis.

11.3.2 Healing Phase

Jarvinen et al. [14, 15] and Beiner [1, 9] have shown that an inflammatory reaction starts 12 h after the injury, including invasion of neutrophil polynuclear cells within the intercellular edema. This phase progressively regresses after 4 days. Cleaning the muscle is effective 4–6 days post-injury. Then the proliferation phase starts: Mauro satellite cells allow development and the replication of the myoblasts and myofibers [16]. At the same time, healing of the connective tissue and neo-vascularization progresses. After 3 weeks, functional muscle fibers have replaced the necrotic tissue. The remodeling phase completes the healing process, as muscle fibers and scar tissue adapt to the mechanical forces.

11.4 Muscle Contusion Classification

Different grading systems are available, with some based on clinical examination [7, 14] and others on imaging [10]. Most of them can be used together and describe three stages: minor, moderate and severe. The quadriceps muscle is the most common site of contusions and the most studied; a classification system specific to that site has been developed by Jackson and Feagin [7]. Range of motion deficit in knee flexion and inability to walk 12–24 h after injury are the main criteria as described on Table 11.1.

Table 11.1 Classification of quadriceps muscle contusions

Classification	Active flexion ROM	Walking
Minor	>90°	Normal
Moderate	45–90°	Antalgic
Severe	<45°	Severe limping

11.4.1 Minor Muscle Contusion

The athlete may or may not remember the incident and usually can keep playing. Soreness increases after effort or the following morning. During clinical examination, the articular active range of motion related to the injured muscle is limited from 5 to 20 %. Local palpation is tender and a minimal loss of force is found during contraction testing [9]. Imaging shows a diffuse swelling, but no structural damage or dilacerations of muscle fibers and no hematoma [10].

11.4.2 Moderate Muscle Contusion

The athlete remembers the incident but can continue playing despite increasing stiffness during breaks (half time). During clinical examination, the athlete is limping; the articular active range of motion linked to the injured muscle is limited to 20–50 %. Palpation and resisted contraction are painful and associated with loss of force [9]. Imaging shows damage to the muscular tissue of less than 50 % of the axial surface of the muscle with a non-collected spreading hematoma [10].

11.4.3 Severe Muscle Contusion

The athlete remembers the impact very well and usually had to stop playing. Bleeding progresses quickly and is difficult to control. Clinical examination reveals a deformity of the muscle with an increased volume sometimes associated with delayed ecchymosis. Palpation is very painful. Walking is difficult. The articular active range of motion related to the injured muscle is limited by more than 50 %. Loss of force is important [9]. Imaging shows damage to more than 50 % of the axial surface with a collected hematoma. The hematoma can be either intramuscular or perimuscular if the peripheral aponeurosis is torn [10].

11.5 Radiological Strategies

11.5.1 Diagnostic

Immediately after the injury, X-ray can be used if an associated bony lesion or other injuries are suspected.

Real time ultrasound seems to be the most used due to its accessibility, its cost and the ability to perform a guided puncture of any collected hematoma. The examination should be performed after at least 48 h [10] to avoid underestimating the seriousness of the contusion and to allow blood collection (Figs. 11.1–11.5). Ultrasound allows precise diagnosis of the injured muscle, measurement of the lesion size and a calculation of the axial injured surface. The examination should look carefully at the superficial aponeurosis. In some cases, local hematoma will be limited to the subcutaneous tissue without a muscle tissue lesion; the superficial aponeurosis will then be intact and show a concave shape. With a lesion of the aponeurosis, pain is inversely proportional to the size of the lesion. Small tears are very painful; large lesions allow the hematoma to drain and decrease intramuscular pressure. In the rare case of an isolated lesion, comparison with the non-injured side will show localized thickening inducing vascular and neural pain.

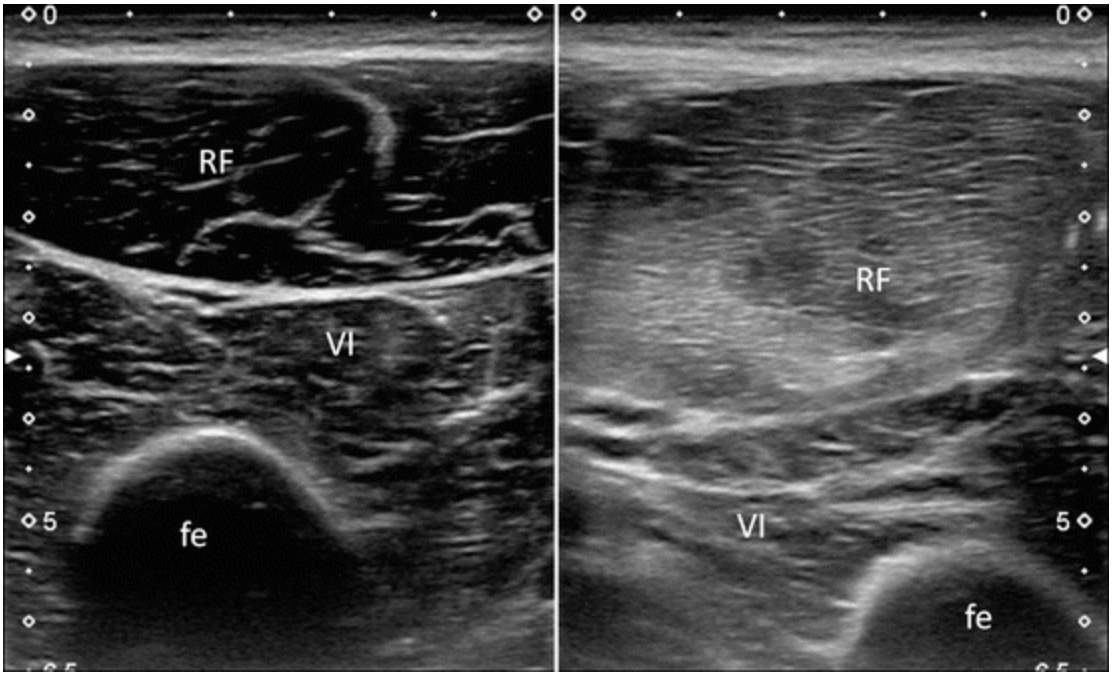

Fig. 11.1 Transverse ultrasound image at the mid third of the anterior thigh shows moderate diffuse hyperechoic signal within the left rectus femoris muscle (>50 % of muscle surface area). The normal right rectus femoris muscle is shown for comparison. *RF* rectus femoris, *VI* vastus intermedius, *fe* femur

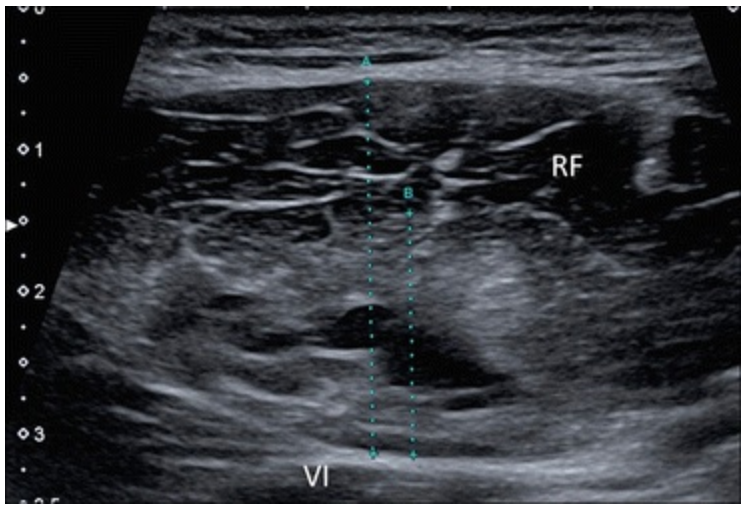

Fig. 11.2 Transverse ultrasound image at the mid third of the anterior thigh shows severe heterogeneous hyperechoic signal with anechoic area signaling a hematoma at the rectus femoris (>50 % of muscle surface area). *RF* rectus femoris, *VI* vastus intermedius

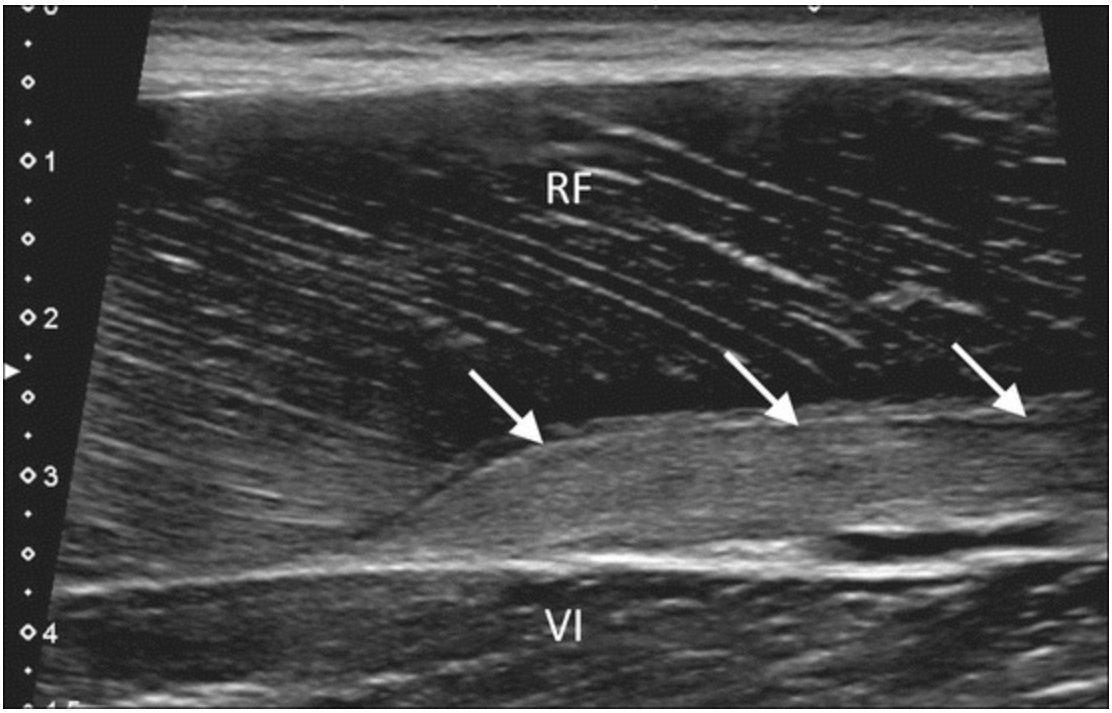

Fig. 11.3 Longitudinal ultrasound image at the mid third of the anterior thigh shows chronic hyperechoic and organized fibrotic tissue (*arrows*) between the rectus femoris and vastus intermedius muscles aponeurosis from chronic hematoma. *RF* rectus femoris, *VI* vastus intermedius

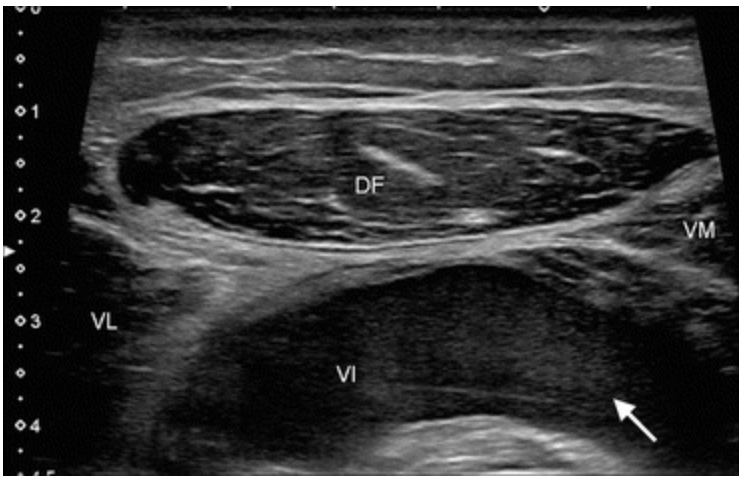

Fig. 11.4 Transverse ultrasound image at the mid third of anterior thigh shows severe anechoic signal within the vastus intermedius due to collected hematoma. *DF* rectus femoris, *VI* vastus intermedius, *VL* vastus lateralis, *VM* vastus medialis, *fe* femur

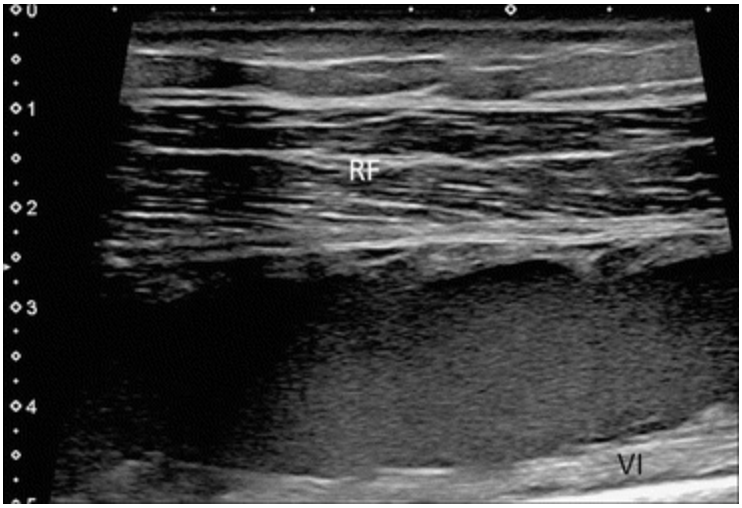

Fig. 11.5 Longitudinal ultrasound image at the mid third of the anterior thigh shows severe anechoic signal within the vastus intermedius due to a collected hematoma. *RF* rectus femoris, *VI* vastus intermedius

If the muscle lesion is deep (e.g. the soleus) or ultrasound is not available, MRI remains the gold standard. With MRI the size of the lesion, local edema, the hematoma and muscle tissue damage can be investigated (Figs. 11.6–11.9).

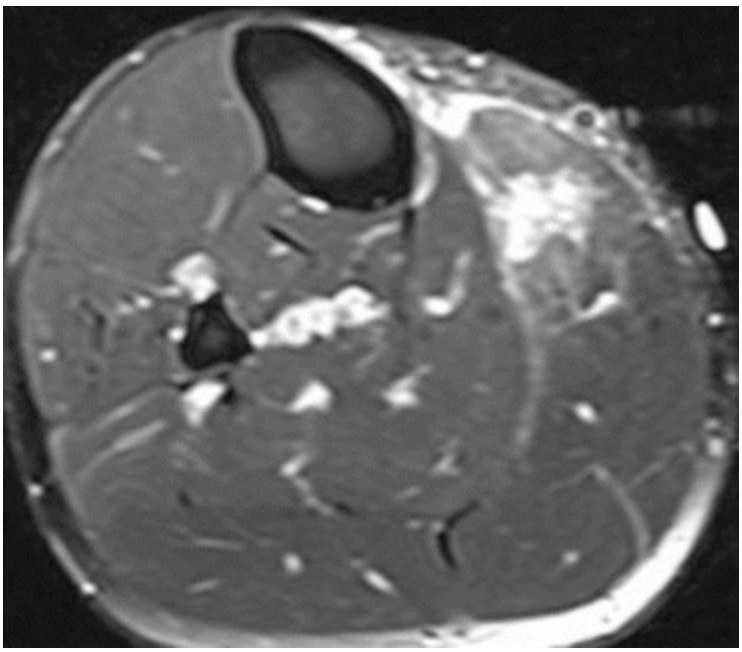

Fig. 11.6 Axial fat-suppressed T2-weighted MRI of right mid-calf shows severe muscle contusion of the gastrocnemius lateralis associated with rupture of the deep and superficial aponeurosis

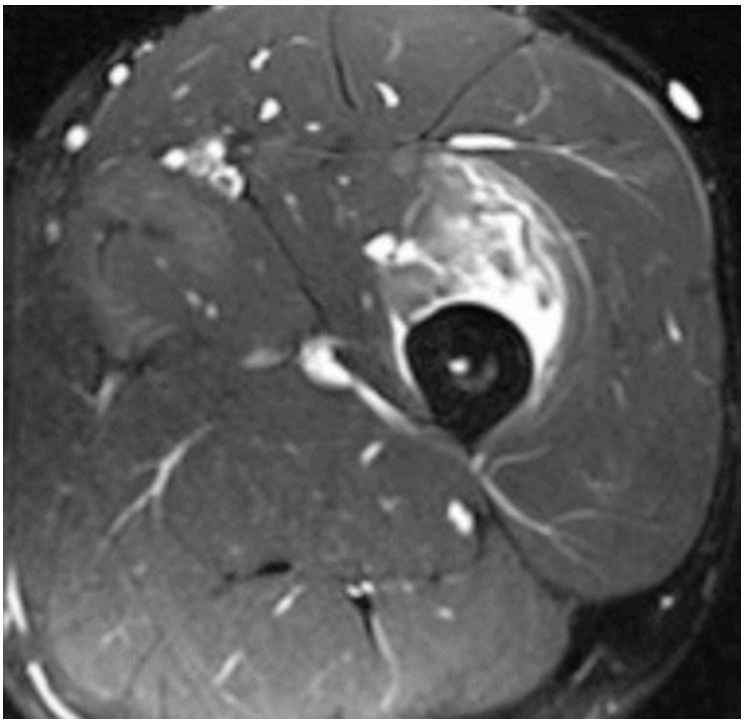

Fig. 11.7 Axial fat-suppressed T2-weighted MRI of right proximal thigh shows severe muscle contusion of the vastus intermedius

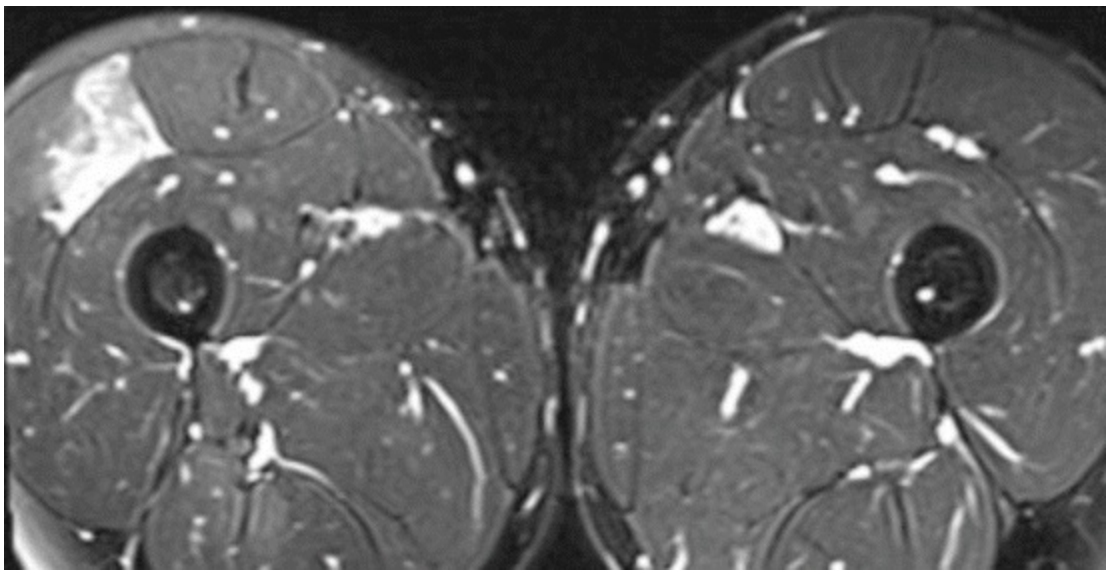

Fig. 11.8 Axial fat-suppressed T2-weighted MRI of right mid-thigh discloses moderate muscle contusion of the vastus lateralis without aponeurosis rupture

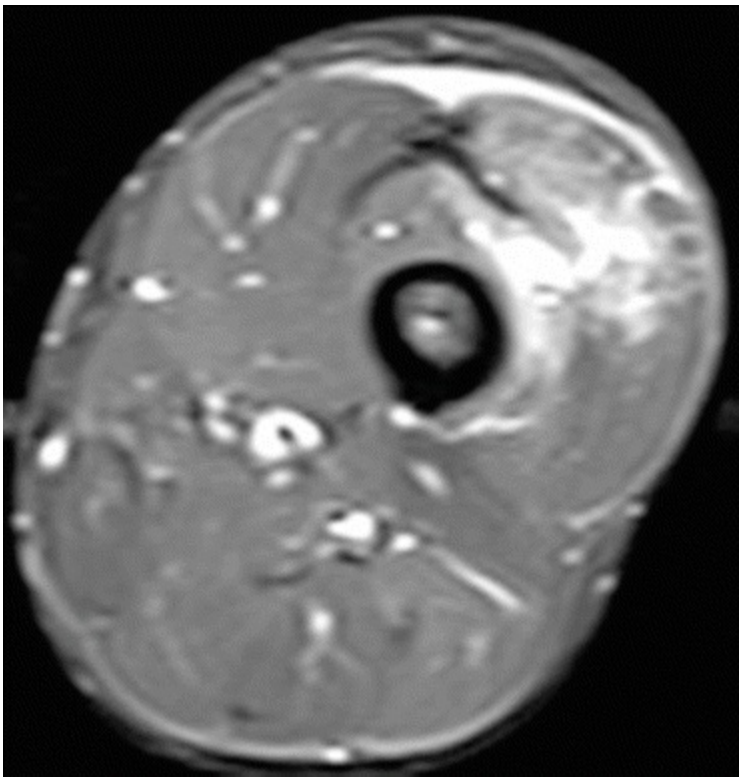

Fig. 11.9 Axial fat-suppressed T2-weighted MRI of left mid-thigh shows combined severe muscle contusion of the vastus lateralis and vastus intermedius

11.5.2 Follow-Up

The healing process can be followed with real time ultrasound. Doppler signal activity, initially localized on the central zone of the lesion, will progressively migrate to the

peripheral zone and slowly disappear.

A few days after hematoma evacuation, an ultrasound examination should be undertaken to rule out any recurrent bleeding.

MRI is the most sensitive examination to follow muscle healing Figure 11.10. Hematoma will decrease after 10 days [

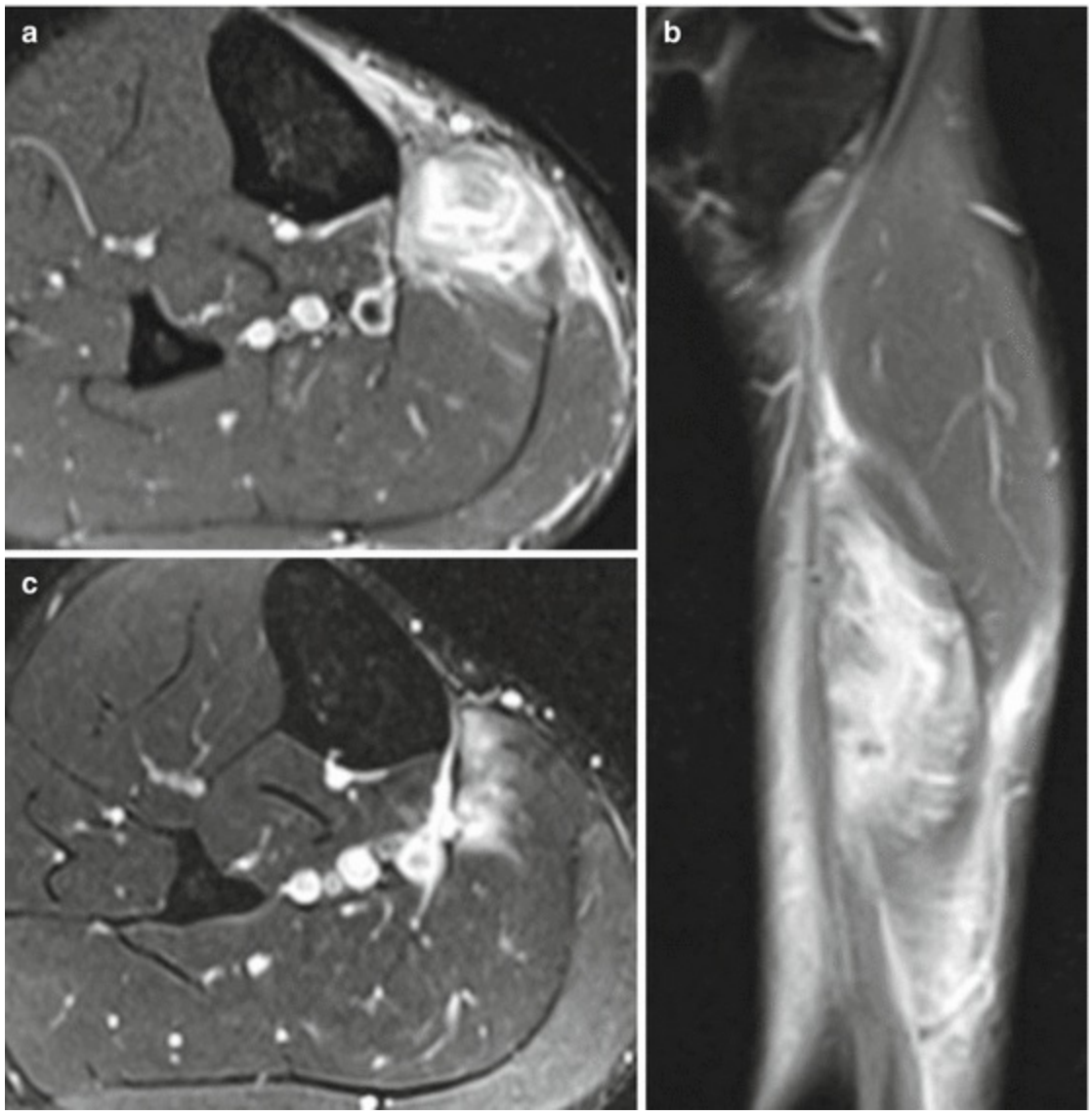

Fig. 11.10 Axial (**a**) and sagittal (**b**) fat-suppressed T2-weighted MRI of the right mid-calf show severe muscle contusion of the soleus associated with rupture of the superficial aponeurosis. Follow-up axial fat-suppressed T2-weighted MRI (**c**) shows partial healing of the muscle lesion with important decrease of the hyperintense lesion

17]. The MRI signal of the hematoma will change with time, as the hemoglobin degrades and scar tissue develops. In the acute phase, the hematoma will appear isointense, surrounded by a hyperintense ring in T1-weighted sequences. T2 sequences show an early hypersignal. In the chronic phase, T2 shows a dark peripheral ring due to the local accumulation of hemosiderin. In T1-weighted sequences, inhomogeneous

hypersignal can be associated with recurrent bleeding [17]. Intramuscular hypersignal in proton density or T2-weighted sequences will persist over time despite a positive clinical and functional evolution [18, 19]. Nonetheless, the use of MRI in deciding when athletes can return to play is still a matter of debate [19].

11.6 Treatment and Management Care

11.6.1 Open Muscle Contusion

Initial management focuses on the penetrating lesion. A precise examination is necessary to extract any foreign body and to evaluate the damaged tissues. After debridement of the necrotic tissue, cutaneous and subcutaneous layers are sutured. Suturing the aponeurosis is still debated due to the risk of compartment syndrome or chronic functional impairment. There is no scientific evidence on the optimal management of such conditions. Once the risk of infection is controlled, management becomes similar to a closed muscle contusion. The status of tetanus immunization should be checked.

11.6.2 Closed Muscle Contusion

11.6.2.1 Acute Phase, the First Forty-Eight Hours

Despite the high incidence of contusion injuries among sports injuries, care is based on experience and consensus. The results of some studies with animal models have been widely applied to humans [14, 15].

In the acute phase, the RICE protocol (rest, ice, compression, and elevation) is common and accepted. This provides general guidelines for the clinician, but the protocol is quite vague, especially on timing and frequency.

Jarvinen showed that rest associated with 24 h of immobilization optimizes healing in animal studies [14]. Some case studies advocate immobilization in a stretched position for a short time [9, 20]. Studies on quadriceps contusions recommend immobilization of the knee at 120°.

In 2004, a literature review on ice and cryotherapy concluded that there was no scientific evidence of therapeutic benefit to muscle lesions. In other types of injuries, ice is beneficial only as a local analgesic [21]. Depending on the depth of the lesion, ice should be applied for 20 min every 4 h during the first 48 h following injury.

Compression and ice have a synergistic effect [21]. Compression alone seems to be less effective on deep muscle groups, especially with strong subjects.

Elevation increases the drainage of the muscle edema and the lymphatic system [9].

Electrotherapy, low frequency pulsed ultrasound and low energy laser are cited in the literature for their role in promoting tissue regeneration and limiting scar tissue. Strong scientific proof to support these types of therapy is, however, still lacking.

NSAIDS and corticosteroids provide an initial analgesic effect but delay the tissue healing process [6, 15]. Their use in such injuries has been stopped.

To summarize, within the 48 first hours following the muscle contusion, moderate to severe lesions will benefit from immobilization in a stretched position for 24 h. Regular icing and compression should be added.

11.6.2.2 After Forty-Eight Hours

Clinical examination and radiological imaging are used to evaluate the gravity of the injury. The clinician can tailor management with progressive mechanical loading of the injured muscle. If needed, the collected intramuscular hematoma can be evacuated with ultrasound guidance. Immediate ice and compression after the procedure may decrease the risk of recurrent bleeding. Clinical and radiological controls can be used to follow healing and detect any local complications.

In the early stage, rehabilitation aims to restore pain free active articular range of motion of the injured limb. The mechanical load is increased progressively using different contraction modalities with open and closed kinetic chain exercises. Finally functional rehabilitation moves to sport-specific exercises. Progress depends on the symptoms. On average, return to play is possible 13 days after a minor lesion, 19 days after a moderate lesion and 21 days after a major muscle contusion [8].

Protective padding in the injured area can decrease the risk of recurrence or complication [9].

11.7 Complications

11.7.1 Compartment Syndrome

This is a rare complication of a muscle contusion. Intra-muscular bleeding associated with an intact aponeurosis increases the pressure within the muscle compartment. Muscle necrosis results from local hypoperfusion and hypoxia. This complication necessitates an emergency surgical aponeurotomy.

11.7.2 Muscle Hernia

Muscle hernia is due to a ruptured superficial aponeurosis. Muscle fibers will incarcerate into the open aponeurosis resulting in a muscle hernia. This can be assessed clinically or with real time ultrasound during dynamic contraction. A small breach induces painful contraction, but a large breach is often better tolerated [10].

11.7.3 Myositis Ossificans Traumatica

Myositis ossificans traumatica is a non-malignant ectopic ossification of the injured

muscle (Figure 11.11). Incidence varies between 9 and 20 % [

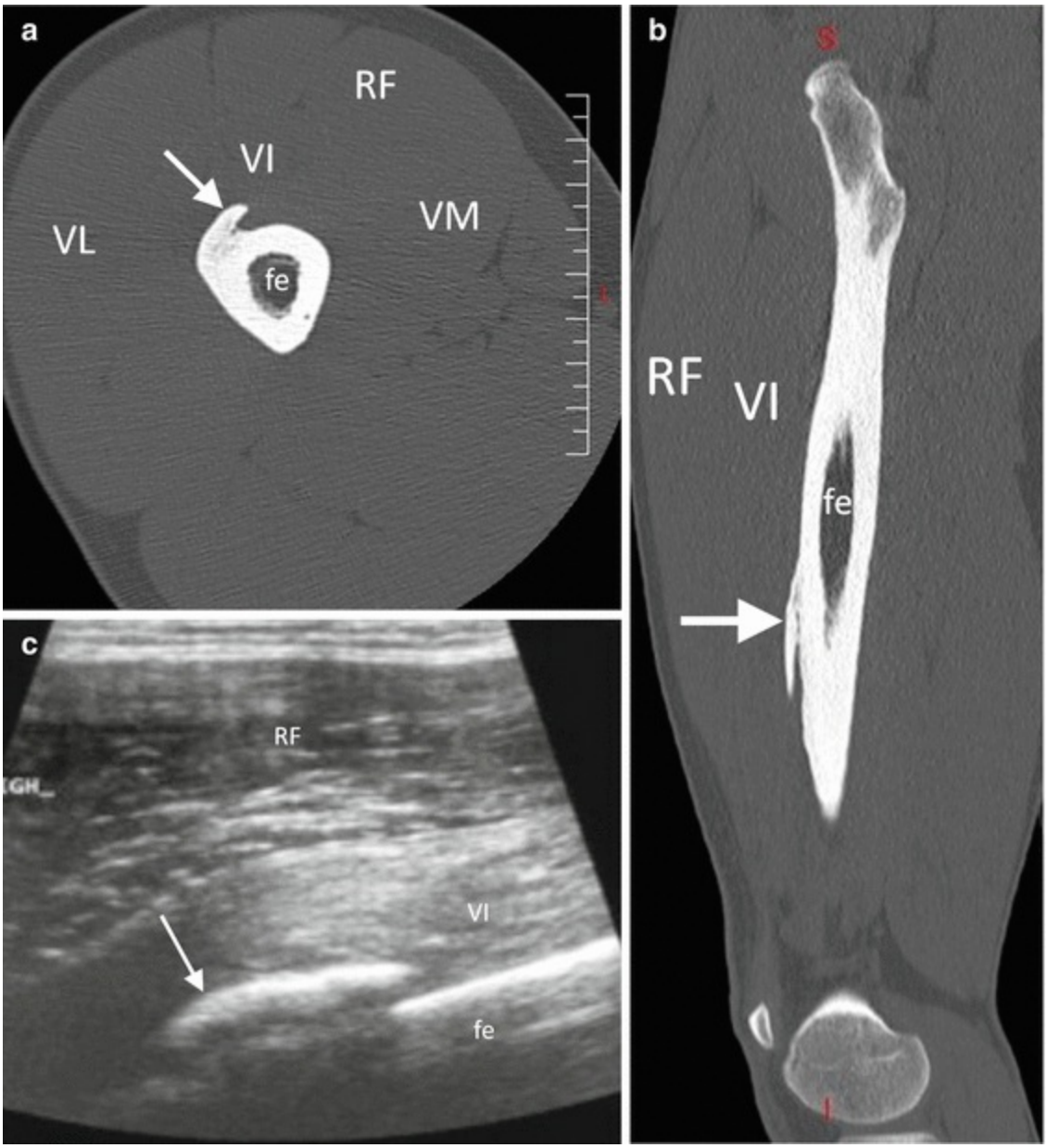

Fig. 11.11 Axial (**a**) and sagittal reformatted (**b**) CT images demonstrate chronic coalescence of the ossification (*arrow*) from the vastus intermedius with femur cortical bone at the mid third of the anterior thigh in keeping with myositis ossificans traumatica. Longitudinal ultrasound image (**c**) at the mid third of the anterior thigh shows hyperechoic linear structure with shadowing effect due to myositis (*arrow*). *RF* rectus femoris, *VI* vastus intermedius, *VL* vastus lateralis, *VM* vastus medialis, *Fe* femur

22]. The severity of the lesion influences its development; minor lesions are associated with up to 9 % of cases of myositis ossificans; moderate and severe lesions in 17–72 % of such cases [22]. Recurrent injuries and young age are two additional risk factors cited in the literature [5, 9, 22]. The quadriceps and brachial muscles are the most often cited locations. The pathogenesis remains debated [22]. Myositis usually develops 2–3

weeks post-trauma. Progressive worsening of the range of motion of the injured limb is associated with a palpable hard mass within the muscle.

11.8 Conclusion

Muscle contusions are among the most common injuries in sports medicine. The healing process of the muscle tissue must be understood to provide the most efficient treatment plan. A thorough clinical examination associated with imaging is required to investigate the condition. Application of a detailed grading system facilitates development of the optimal treatment strategy. Severe muscle contusions have a high rate of complications; in the worst case they can end an athletic career.

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Part IV
Intrinsic Muscular Injury

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12. The Protective Role of Cervical Spinal Muscle Masses in Sports Related Trauma

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Abstract
The cervical spine is an anatomically complex structure whose essential roles are to orient the head and the sensory system and to protect the spinal cord. In developed countries, sports related accidents constitute the fourth most common cause of spinal cord trauma. They are the second most frequent case of spinal cord injuries for those under the age of 30. All sports are potentially affected even though it would seem that highly kinetic physical activities or contact sports would be the most concerned. In this chapter, we will first review the anatomy of the cervical spine and then describe the biomechanical basis of the protective effect of the cervical musculature and the neurophysiological aspects of trauma prevention. We will describe how cervical muscles can be evaluated and close with a brief overview of preventative strategies of the French Rugby Federation will be described.

12.1 Introduction

The cervical spine is an extremely solicited zone in most sports. It also supports the

head which orients the sensory organs (visual, vestibular and auditory). These sensory organs constitute the starting point of information collection about ongoing activity. This anatomical structure also must protect the effector organ of the locomotive apparel: the spinal cord.

In developed countries, sports related accidents constitute the fourth most common cause of spinal cord trauma. However, for those under the age of 30 years, they are the second most frequent cause [1]. Currently the life expectancy of an individual with a medullary injury reaches approximately that of the general population; treatment may add an extremely heavy financial burden to the human and social drama caused by those accidents [2].

All sports can be potentially affected even though it may seem that significant kinetic physical activities or contact sports are the most concerned of all [3–11].

12.2 Anatomical Review

The cervical spine is an anatomically complex structure whose essential roles are to orient the head and the sensory system as well as to protect the spinal cord. This implies a capacity for both rigidity and flexibility given that the structure is extremely mobile with over 6000 daily movements [12].

Experts differentiate on the morphological and functional levels:

- The superior cervical spine consists of two vertebrae: Atlas (C1) and axis (C2) on top of which the skull swivels (C0).
- The lower cervical spine consists of five vertebrae (C3 to C7).

The vertebrae articulate between each other via three structures: the intervertebral disc anteriorly and the large articular processes posteriorly, one to the left and the other to the right. There is an elaborate ligamentory system in place that largely contributes to the stability of this structure.

The neck muscles, 48 in all, are of the skeletal striated type. The vast majority are polyarticular and distributed in symmetrical pairs. On the functional level we can differentiate three muscle groups (Table 12.1; Fig. 12.1):

- The anterior group, with its longus colli and longus capitis.
- The lateral group, with its scalene muscles, levator scapulae muscles and the sternocleidomastoids.
- The posterior group which can be divided into three levels: the superficial (trapezius), the median (splenius capitis and splenius colli), the deep (semispinalis muscles, transversospinalis, and suboccipital muscles for C1C2).

Table 12.1 Neck muscles

Groups	Muscles
Posterior	Trapezius (1), rhomboideus (2), splenius capitis (3), semi spinalis (4), multifidus (5), longissimus cervicis (6), splenius cervicis (8)
Lateral	Ilio costalis (7), levator scapula (9),posterior (10), middle (11) and anterior scalene (15), sternocleidomastoid (16)
Anterior	Longus colli (12 + 13 + 14), infrahyoid muscles: omohyoid (18) et sternohyoid (19), platysma (17)

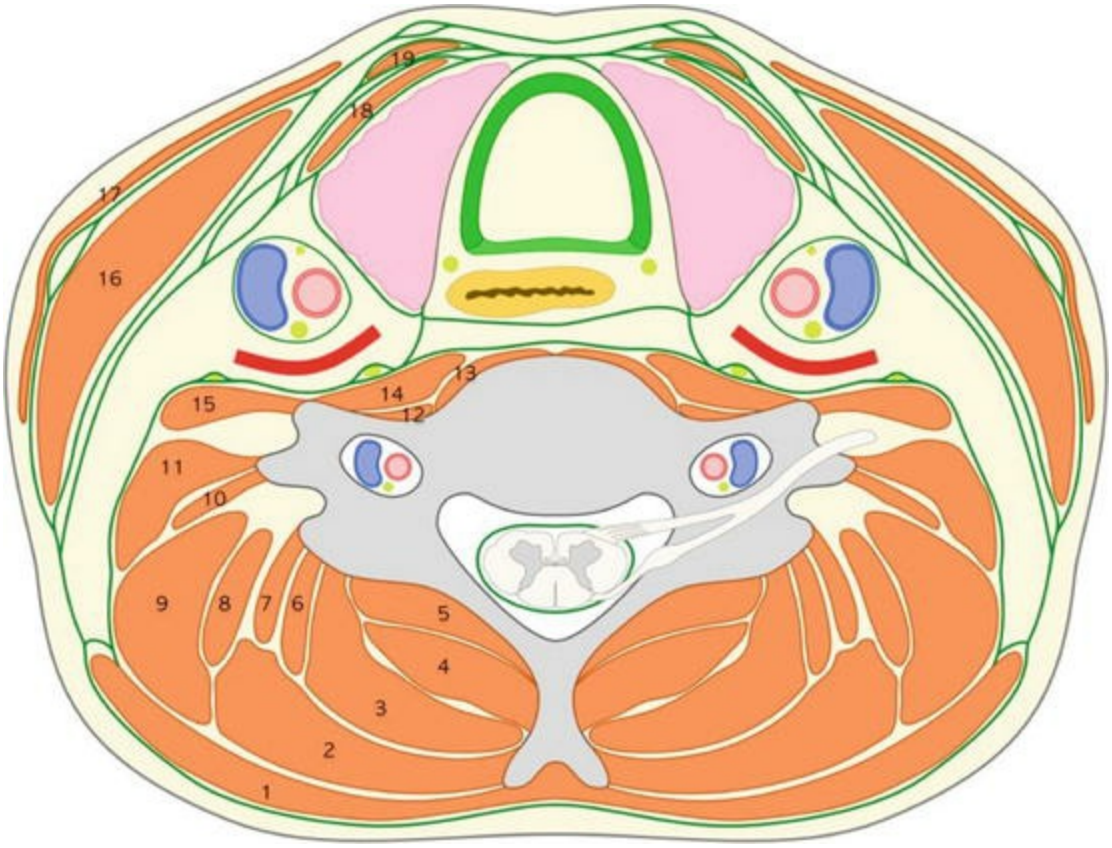

Fig. 12.1 Paraspinal cervical muscles-cross section at C6

They serve two essential roles in the function of the cervical spine:

– *Motion of the head and neck segment* **.** It is hard to precisely define the individual function of each muscle; in addition each differs regarding the head position, the chief muscle in action and the unilateral or bilateral character of muscle activation [13].

– *Stability and rigidity of the neck* **.** In fact, the line of action for most of these muscles is parallel to the spine with a pennation angle ranging between 0 and 30°, but mostly less than 3° for the majority given that there is little specific morphological data about the deep muscles [14–16].

Despite the fact that all muscles contribute to these two functions [17], it seems that the deep muscles are more involved in maintaining postural stability, whereas the

superficial muscles are involved in movement of the cervical spine. This notion conforms with several biomechanical and electrophysiological studies [18]. Finally, Schomacher has recently shown that the recruitment threshold of muscle fibers of these deep muscles varies according to the spinal level, with a lower recruitment threshold for the inferior cervical spine [19].

Volume varies significantly from person to person, and thus there is much variation in the forces generated by muscle masses [20]. As for the skeletal striated muscles, consider the correlation between the cross sectional area of a muscle and its potential generated force, a factor that seems to explain the observed differences between men and women [21–24]. In parallel to the volume variation, muscle insertion variations can be noticed particularly for the deepest muscles of the neck [16].

12.3 Biomechanical Basis of the Cervical Musculature Protective Effect

There are several morphological parameters that influence the stability of a cervical spine under external constraint. Those elements have been cited in the literature review of Stemper in 2011 [25].

– Vertebral body morphology**:** larger vertebrae are associated with a higher rupture threshold.

– Cervical spine curvature: kyphotic spines seem to be at a higher risk of traumatic lesion.

– Articular facet orientation: articular processes of smaller volume and vertical orientation offer less significant stability.

These parameters define *passive stability,* whereas *active stability* is ensured by the paraspinal cervical muscle.

Even with the lack of studies that define paraspinal muscular incompetence as a risk factor of serious accidents, it has been known for several years that this musculature intervenes during impacts to decrease the extent of the motion [26, 27]. This protective mechanism consists of a simultaneous contraction of all the muscles inducing an axial contraction of the whole cervical spine [28, 29].

At the experimental level, some evidence seems to imply that the muscle mass plays a role of “active protection” during a cervical spine trauma. Recently published laboratory measurements suggest that an important muscle force of the cervical spine reflects more efficient active protection in the case of a violent trauma of the head [30]. These results seem like they would be useful for screening of individuals at risk of severe injury but they were not confirmed by a study Mihalik undertook on 37 ice hockey players during one season [31]: Analysis of the acceleration meter in the

players' helmets showed there was no less-violent trauma in subjects with higher muscle force peaks.

The biomechanical role of the paraspinal muscles as a passive protector has been significantly less studied. Nonetheless, its importance seems to be far from negligible and its role increases along with the stretching constraints. The deep muscles are involved first then the superficial larger ones which have more significant leverage [32, 33]. Regarding this subject, analysis by finite element method seems to be promising, allowing simulations of different accidents. The use of a valid experimental model has recently established that the muscular absorption of constraints linked to a traumatic shock were minimal in the case of traumas in hyperextension. This result suggests higher spine vulnerability in this position [33].

Finally, as it ages, the cervical spine becomes rigid and loses its flexibility [34]. Biomechanically, this loss of flexibility exposes the subject to an increased risk of spinal lesions [35, 36]. This will in turn expose athletes at the end of their career to an increased risk of cervical osteoligamentary rupture in the case of an excessively hyperflexed posture. Therefore an athlete at career's end should be extra vigilant about preparing the cervical spine muscles for action.

12.4 Neurophysiological Aspect of Prevention

When exposed to a violent impact, the response-time is one of the critical parameters. It has been studied in particular for whiplash type accidents. EMG of the sternocleidomastoid muscle reveals that the latent time after sudden acceleration is slightly longer for men than women, about 8 ms and becoming even longer with age [29, 37, 38]. Overall, displacement of the cervical spine will occur between 20 and 40 ms after an impact causing displacement of the head [39, 40]. The muscular delay in activation occurs around 120 to 150 ms with the peak of activation ranging between 200 and 250 ms in subjects aged between 20 and 30 exposed to the most violent traumas [41]. During a direct shock impacting the top of the head, which happens with the spear tackle in American football [4, 42], spinal lesions were noticeable within 2 ms [40].

This muscular contraction is divided into two phases: a reflex contraction and a voluntary contraction [29]. Brault also notes the risk of a muscular lesion if the reflex contraction arises after the head has already started a movement. As for trunk muscles, Moorhouse and Granata estimate that the reflex muscular activity is around 40 % responsible for maintaining stability when facing a postural perturbation [43].

These activation delay notions are of extreme importance in some collision sports where spinal cervical trauma is associated with skull trauma that might lead to a concussion [44]. At that moment, the player on the verge of losing consciousness can no longer "defend" himself through muscular recruitment allowing him to rigidify his spine.

As with all striated muscles of the human body, spinal muscle masses are also

subject to fatigue. In fact, maintaining the head in a desired position requires a large recruitment of muscles, particularly if the spine is subject to supramaximal constraints during an intense physical effort. A contraction is called exhausting if it is maintained at a tension level superior to 15 % of the maximal force of a given subject, the endurance being defined by the amount of time during which such a contraction can be maintained [45]. These elements might explain why the majority of accidents occur in the beginning of the season when physical preparation hasn't yet reached its objectives, as suggested by Mall who has analyzed the collected data from the (American) National Football League over a period of 11 years [46], although other authors find that these lesions become more frequent as the season progresses [47]. As for age related effects, studies indicate that the delay in action of the muscular recruitment becomes progressively longer and the muscular healing following intense physical activity becomes longer as well, although this has not been specifically proven in the case of spinal muscles [48].

12.5 Efficacy of Muscular Reinforcement Programs in Sports Related Trauma

Many authors recommend implementation of cervical spine physical preparation programs for high risk activities following a traumatic event [49, 50]. Furthermore, in some disciplines, the condition of the cervical spine constitutes a performance factor that allows the player to get the better of the adversary.

Numerous studies have proved that a reinforcement program is effective for improving the performance of the cervical musculature [51–53]. Various preparation schemes using isometric or static protocols have been described, although neither is clearly superior. Note that even though the benefits of conditioning the cervical musculature are recognized with regards to maximal isometric force, dynamic stability under constraint does not seem to be significantly improved.

12.6 Cervical Muscle Evaluation

After a problem affecting the cervical spine, many authors agree that return to play cannot be expected until the muscular force has returned to normal [49].

On the other hand, paraspinal muscular incompetence would be an interesting risk factor to screen for in a severe accident.

On this basis, it seems useful to evaluate two parameters:

– The maximal isometric muscle force.

– Muscle fatigability.

The maximal muscular force can be measured through isometric or dynamic

methods. Isometric measurements are mostly used. To mention a few:

- Manual muscle testing: In which the evaluator rates the force of some muscle groups from 0 to 5.
- Handheld dynamometry: Evaluation modalities remain the same; however the rating is achieved through a dynamometer.
- Fixed frame dynamometry: The head and trunk are fixed; the measures are taken by a dynamometer.
- Isokinetic dynamometry

Muscular fatigability is defined as the loss of the muscle's capacity to generate a force. This refers us to the definition of endurance as the capacity to maintain repeated muscular efforts or generate a force in a given period. Assessment of these parameters is rarely used in clinical practice. To mention a few:

- Subjective methods: the subject describes the fatigue.
- Clinical tests ("time dependent methods"): evaluation of the maximal duration of maintaining a posture, the maximal number of repetitions, etc. EMG: the appearance of electrical modifications. These methods lack standardization.

So far, there is no normal value for these measures. In fact, it seems that the large variability in evaluation protocols as well as the heterogeneity of the studied population explains to a high extent the difficulty in obtaining reliable and reproducible values [54, 55]. In this context, consider the study by Oliver who has assessed a sample of premier league rugby players in an attempt to define some norms in regard to their positions, keeping in mind that some are more exposed to spine trauma [56]. Unfortunately, these measuring systems and the expertise of a well-trained operator can be hard to obtain. Couvet considered this problem and asked how to determine whether performance on isometric tests could be correlated to simple evaluations. The results, awaiting confirmation on larger series, seem to indicate that simple and inexpensive evaluations could contribute in keeping away, from some high risk positions, those subjects who failed to pass these tests. The French Rugby Federation has since implemented a technical passport partially based on those results [57].

Finally, qualitative analyses of muscular mass seem promising in high level sports fields but they are currently still at their beginning and do not allow definition of precise objectives in terms of physical preparation. The development of dual-energy X-ray absorptiometry (DXA) which is a three component model, allows analysis of bone mineral density in addition to muscle mass and fat mass [58, 59]. Volume analyses are not routinely used, although it seems that single muscle area analysis would help to account for the importance of the whole musculature [60].

12.7 Preventive Strategies of the French Rugby Federation

Rugby is a contact sport and players are exposed to cervical spinal cord accidents [47, 61–64]. Many studies have identified the risks associated with periods of the game and with positions. (Fig. 12.2).

Fig. 12.2 The scrum. Forward is the most exposed position, with an average of 15 scrums per game

For several years now, the French Rugby Federation has implemented a preventive policy aiming to limit the number of severe medullary accidents related to the practice of rugby. We can clearly describe the annual prospective epidemiological data collection of these accidents, rules modifications, as well as screening measures of subjects at risk based on the clinical assessment with cervical spine MRI cuts for professionals and players registered on the sports ministry lists [5, 65].

In France, the ability of a player to move towards a high risk position is evaluated on two levels:

– *On the medical level:* Every player should pass a medical visit at the beginning of the season, and submit a medical certificate of suitability. It is at this point that the physician can decide whether the athletes can participate, based on the clinical assessment. An MRI of the cervical spine should be prescribed in complex cases. The criteria set in 2002 are constantly being updated according as new data becomes available.

The player receives a certificate stating that he is “capable of playing in first line”, and which the referee should verify before each game.

– *On the technical level:* The federation has recently implemented a validation procedure for the ability to play in front positions. It is based on a functional muscular evaluation of the cervical spine and on the technical skills of the player in some game situations. The certificate indicates «first line player» and will be valid for the entire career in sports of the player, except in the case of a prolonged suspension.

This evaluation has two stages.

- The first takes place in the clubs under the supervision of a trained specialist. It consists of biometric and muscular evaluation completed by a number of tests to screen for any cervical paraspinal muscular incompetence. (Fig. 12.3).
- The second takes place in a federal structure under the supervision of a technical counselor of the federation. The main goal is to verify the technical and muscular ability in simulated game situations. (Fig. 12.4).

Fig. 12.3 Trunk stability exercices: with the four limbs (**a**) and with the neck (**b**) (from "Passeport joueur de 1ere ligne" – Photo credits I. Picarel)


a
b


c
d

Fig. 12.4 Scrummaging assessment for front row players: position before the impact (**a**), the clench (**b**), pushing exercice for three players (**c**) and for one player (**d**), pushing exercice under pressure (from "Passeport joueur de 1ere ligne" – Photo credits I. Picarel)

12.8 Conclusion

There are several factors establishing the protective aspect of the cervical spinal musculature in traumatology. The current challenge is to determine whether there are indicators that allow "a priori" screening for muscular incompetence that may predispose the player to injury. Current preventive measures are based on muscular reinforcement, allowing improvement of the isometric maximal force. There are ongoing efforts to define a training regimen that allows acceleration of recruitment delays which in turn would be beneficial for the prevention of injuries.

With age, the sportsman's cervical spine becomes rigid, the action delays of muscular recruitment increase and the healing following an intense physical effort gets longer. Even though it seems that muscular atrophy at the level of the cervical spine starts much later, towards the seventh decade, we can assume that the aging athlete is exposed to a higher risk of injury and will need to compensate by a significant muscular preparation which will grant him active and passive protections when facing high energy traumas. The other important element is training that promotes playing techniques that help avoid high risk situations through the integration of neuromuscular recruitment strategies.

In the end, there will always be some situations where the muscular protection alone

is overwhelmed, either by intensity or by speed of trauma. Such situations inherent to life activities could be avoided through other measures implemented by the sports federations such as rules modifications, educational programs and protective gear etc.

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13. Abdominal Wall Injuries

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Abstract
The rectus abdominis, transversus abdominis and oblique muscles form the abdominal wall. Indirect trauma is the most frequent, often occurring in tennis or soccer. Spontaneous hematoma in the elderly population or endometriosis may also cause disorders of the abdominal wall muscles. Ultrasound is a readily-available tool for diagnosing injuries but its accuracy depends on the operator's experience. MRI is performed to confirm diagnosis or in high-level athletes to show areas of high-signal intensity in case of strain. Motion artifacts can be a problem and care should be taken when positioning the patient and the coils. Appropriate software can overcome some motion artifacts to improve image quality. Preventative treatment is fundamental in pre-season rehabilitation to avoid such injuries. Rest and physiotherapy are the main treatment for strains.

13.1 Introduction

Abdominal wall injuries may involve the rectus abdominis, transverse and oblique muscles. Strains are encountered in athletes but others conditions such as spontaneous hematoma or endometriosis may occur. Clinical symptoms include pain and tenderness but an accurate diagnosis depends on imaging. Ultrasound is usually performed first although it is not as accurate as MRI. In this chapter we describe the anatomic structures

of the abdominal wall and match the role of each imaging technique with the description of the injury patterns.

13.2 Anatomy

13.2.1 Rectus Abdominus

The recti abdomini muscles are paired muscles located on the anterior part of the abdomen, separated at the midline by the linea alba. Each rectus abdominis muscle has a medial head which arises from the anterior surface of the pubic symphysis and a larger lateral one which originates from the upper border of the pubic crest. Those two heads form a muscle belly which is surrounded by a large sheath and insert onto the fifth to seventh costal cartilages.

Each muscle belly is interrupted by three transverse fibrous bands which run deep to the anterior part of rectus sheath. The linea alba is a central fibrous structure resulting from the fusion of the oblique and transversus abdominis muscles which runs vertically from the xiphoid process to the pubic symphysis.

The recti abdomini muscles play an important role in trunk flexion and stability in athletes (Fig. 13.1) [1].

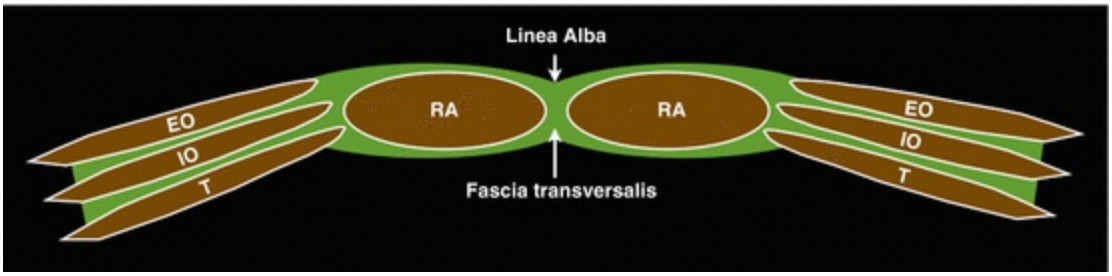

Fig. 13.1 Schematic drawing of the abdominal wall muscles. *RA* rectus abdominis, *EO* external oblique, *IO* internal oblique, *T* transverse muscle

13.2.2 Oblique Muscles and Transversus Abdominis

The external oblique is located on the lateral and anterior parts of the abdomen. It arises from eight fleshy digitations which emerge themselves from the 5th to 12th ribs and that have an oblique inferior and anterior course. The fibers emerging from the lowest ribs have a vertical course and insert on the anterior half of the outer lip of the iliac crest. The fibers from the middle and upper ribs have an oblique inferior and anterior course and become gradually aponeurotic before crossing the linea alba.

The internal oblique muscle is the intermediate layer of the abdomen wall musculature located deep to the external oblique muscle and superficial to the transverse abdominal muscle. It arises from the thoracolumbar fascia, the anterior two-

thirds of the iliac crest and the lateral part of the inguinal ligament. Its fibers run perpendicular to the external oblique muscle, have a superomedial course and end on the inferior edge of the three lowest ribs and on the linea alba.

The oblique muscles are active during rotating and side-bending the trunk and during exhalation as an antagonist of the diaphragm.

The transversus abdominis muscle emerges from the inner lip of the iliac crest, the inguinal ligament, thoracolumbar fascia and the costal cartilages from the 7th to 12th ribs. It has a transverse course and becomes gradually aponeurotic before inserting on the xiphoid process and the linea alba. The lowest fibers have a distinct inferomedial course and join fibers of the internal oblique forming the inguinal aponeurotic falx (or conjoint tendon) which ends at the pubic crest forming the medial part of the posterior wall of the superficial inguinal ring.

Oblique muscles and the transversus abdominis act by compressing the abdominal content and have a limited role in flexion and rotation of the trunk (Fig. 13.2) [2].

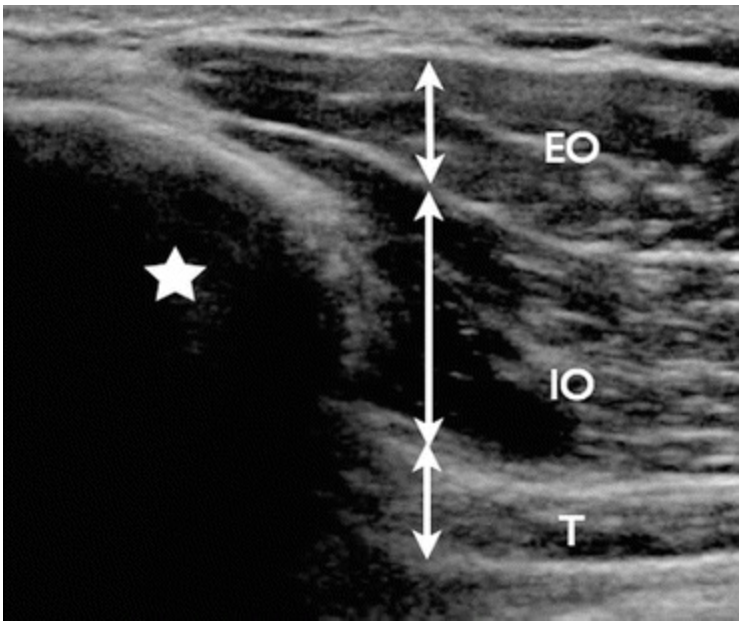

Fig. 13.2 Normal transverse ultrasound image with a 17–5 MHz transducer shows normal abdominal wall anatomy at the level of the iliac crest (*arrow*). *EO* external oblique, *IO* internal oblique, *T* transverse muscle

13.2.3 Normal Variants

Abdominal muscle size is greater in males than in females when assessing muscle thickness with ultrasound at rest and during contraction [3]. Symmetry of the abdominal wall muscles is subject to change according to activity. One study found no clinically discernable differences in the left and right lateral abdominal wall muscles (transversus abdominis, internal and external obliques) in single-sided rowers despite the asymmetric functional demand [4]. Nevertheless, asymmetry of the rectus abdominis is

seen in the non-active population as well as in athletes. Sports like soccer or tennis attenuate the pattern of asymmetry because they induce muscle hypertrophy of the non-dominant side in proximal regions and the dominant side in regions closer to the pubic symphysis. It remains to be determined whether hypertrophy of the rectus abdominis in soccer players may increase the risk of injury [5, 6].

13.3 Clinical Findings

13.3.1 Rectus Abdominis

Rectus abdominis muscle injuries are secondary effects of direct blows to the abdomen or indirect trauma by sudden or repetitive trunk movement, either rotation or flexion/extension. One retrospective study reported a prevalence rate of 29.5 % in the career of a professional tennis player. Injury happens during the cocking phase of the service motion when eccentric overload followed by forced contraction applies on the non-dominant rectus abdominis. Those muscle strains have also been described in many sports such as soccer, handball, baseball and their prevalence seems to increase especially in early season. Usually, the lesion is located in the rectus abdominis on the side of the nondominant arm or foot. Pain is usually peri- or infra-umbilical and is exacerbated by eccentric or concentric contraction. There is usually no ecchymosis because strains are mainly low-grade injuries [7–11].

A spontaneous rectus sheath hematoma can occur in elderly adults without history of trauma and with or without anticoagulation treatment [12]. Care should be taken in case of an abdominal mass with cyclical pain symptoms in middle-age women. Extra pelvic endometriosis which involves the abdominal wall muscles is an uncommon finding but it should be considered in the differential diagnosis of any abdominal swelling with or without a previous history of surgery [13]. A rare case of myositis ossificans involving the rectus abdominis muscle has been described in which the complete lack of history of trauma led to the suspicion of a malignant tumor, which was proved surgically [14].

No specific complications have been described besides those common to muscle strains. A fibrous scar can be responsible for chronic tenderness and pain and is usually treated by conventional physiotherapy and does not require further exploration. In our experience, some fibrous scars are responsible for small nerve entrapment and/or neuroma; they can be treated by surgical excision.

13.3.2 Oblique Muscles and Transversus Abdominis

Side strains are mainly encountered in cricketers, javelin throwers, rowers, and ice hockey players. Patients present with sudden pain and point tenderness in the region of the lower costal margin. It is due to a tear of the internal oblique muscle from its rib or costal cartilage origin. Internal oblique muscle strains may also occur at the proximal

insertion onto the iliac crest resulting from eccentric, unbalanced trunk rotation in tennis players. Such injuries may extend to the external oblique muscle [2, 8, 15]. Isolated external oblique muscle strains have been described in elite ice hockey players and usually cause groin pain.

The main differential diagnosis is rib stress fracture that occur in rowers, swimmers, golfers, and canoeists and which results from the repetitive forces exerted by the external oblique and serratus anterior muscles [16].

13.4 Imaging Findings

13.4.1 Rectus Abdominis

Ultrasound is an easy and commonly available technique that enables contralateral examination and appears to be sensitive to tears when used with a high-resolution transducer. Acute injuries are easier to diagnose when bleeding is present within the fibril disruption and the muscle edema. Hematoma appears anechoic at the acute phase and its echogenicity slightly decreases with healing. In our experience and in one study, MRI does not provide any additional information but the images are easier to understand [1]. Hematoma appears as a well-delineated high signal intensity area on both T1- and T2-weighted images within the muscle. Fluid-fluid levels and a concentric ring sign are commonly observed [17]. Hypertrophy of the rectus musculature on the non-dominant side is a normal finding which should not be taken for pathology. Coronal and sagittal images are the most useful for assessing the degree of fibril disruption and retraction.

Some studies have established that acute injury almost always occurs in the deep epimysium at the infra-umbilical level, approximately 5 cm from the umbilicus. Medial and anterior injuries are extremely rare. Tears may involve the whole thickness of the muscle or they can be partial [2, 9].

At a chronic stage, fibrous scars appear hyperechoic with acoustic shadowing. Dynamic ultrasound allows visualization of the irregular and retracted margins. Calcifications are more uncommon and result from hematoma. On MRI, scars have low signal intensity (Figs. 13.3, 13.4, 13.5, 13.6, and 13.7).

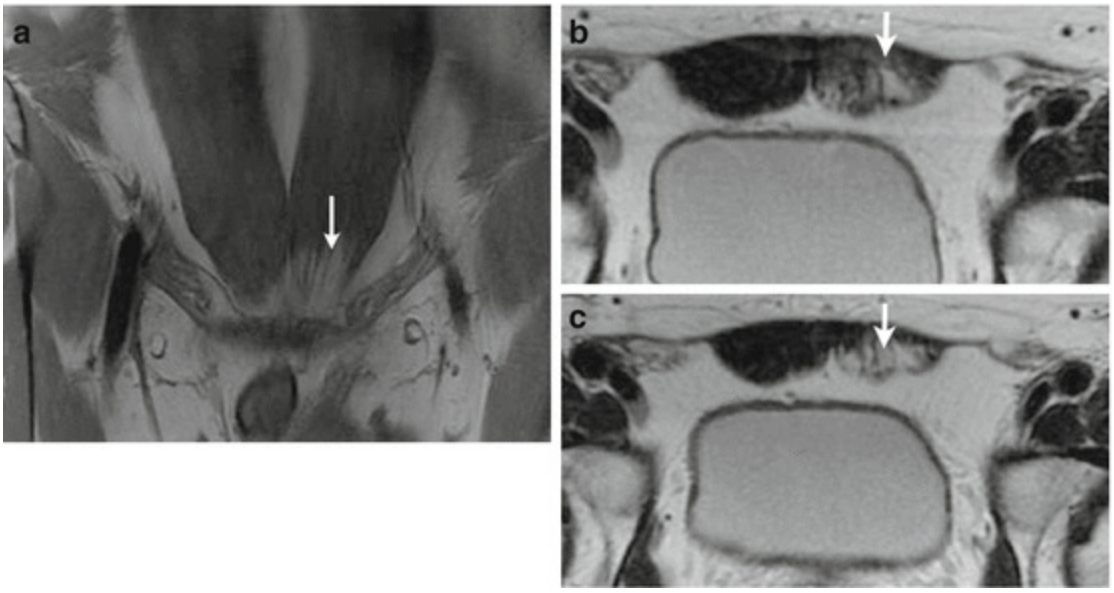

Fig. 13.3 Coronal (**a**) and axial (**b**, **c**) T1-weighted MRI show left rectus abdominis amyotrophy (*arrows*) compared to the opposite one

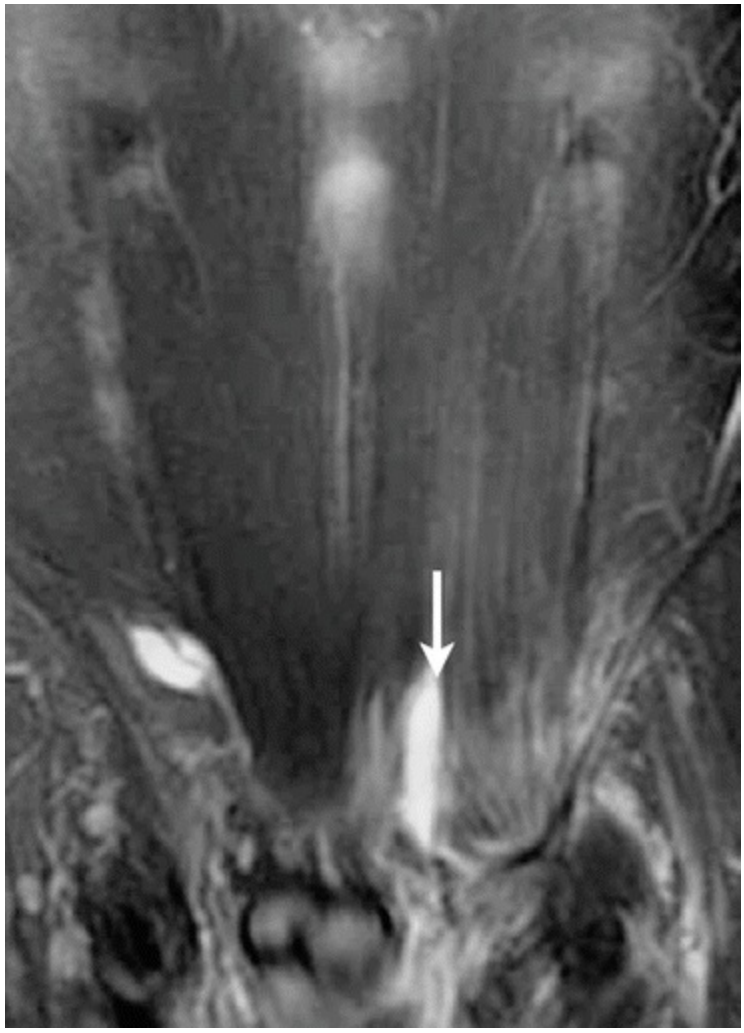

Fig. 13.4 Coronal fat-suppressed T2-weighted MRI shows high signal intensity area due to left rectus abdominis

strain (*arrow*)

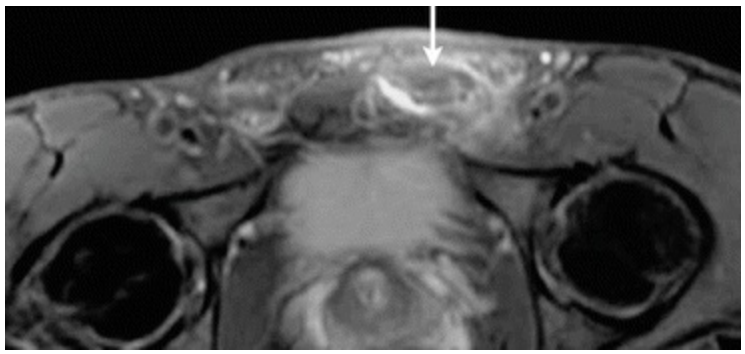

Fig. 13.5 Axial fat-suppressed T2-weighted MRI shows high signal intensity area due to left rectus abdominis strain (*arrow*)

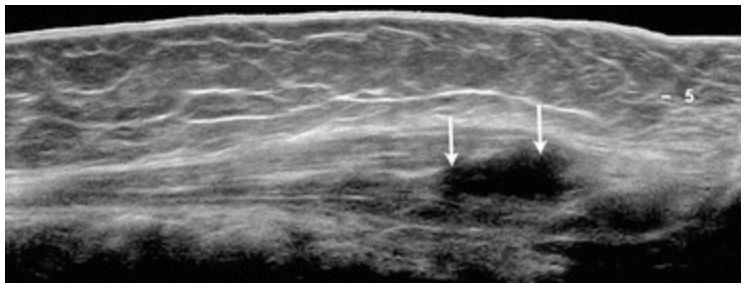

Fig. 13.6 Longitudinal ultrasound image with extended field-of-view shows anechoic hematoma (*arrows*) within rectus abdominis muscle following indirect trauma in a professional tennis player

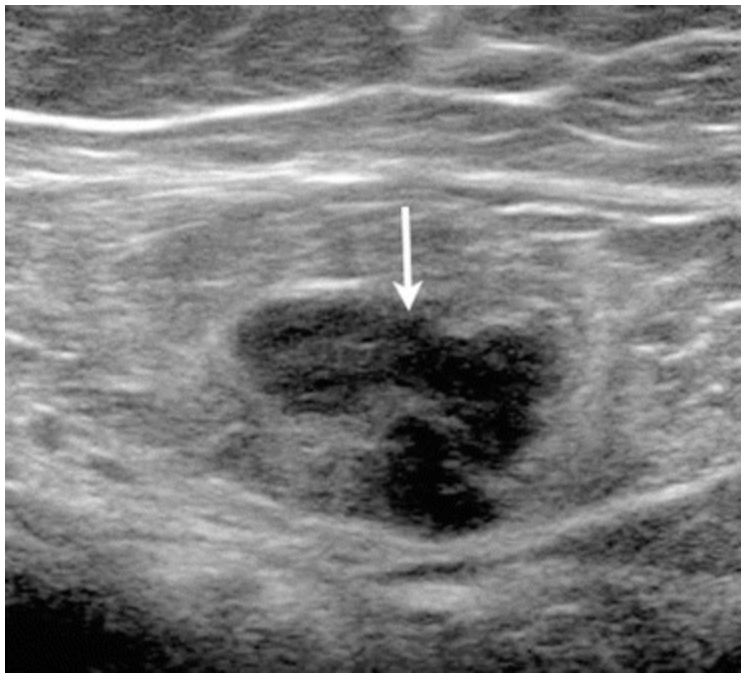

Fig. 13.7 Transverse ultrasound image shows anechoic hematoma (*arrow*) within rectus abdominis muscle following indirect trauma in a professional tennis player

13.4.2 Oblique Muscles and Transversus Abdominis

Side strain injury results from a tear of the internal oblique muscle from the undersurface of one of the lower four ribs or costal cartilages. Muscle edema and hemorrhage are usually observed and bleeding passes through the surrounding fascias. The average length of the tear is ranges from 1 to 3 cm. Stripping of the periosteum from the undersurface of the rib may also be present [15, 16]. Fluid-sensitive sequences in a sagittal oblique plane allow better assessment of the tear. Respiratory and motion artifacts are frequent and decrease image quality. Care should be taken when positioning the patient and surface coils. Software has been developed to reduce such artifacts which are common when assessing oblique muscles at their proximal origin. Ultrasound may show muscular edema with fibrillar disruption and hematoma. Contralateral examination is required to confirm the diagnosis of strain. Color Doppler is useful for the end of the healing process as hyperemia fades [2]. More distal injuries involving the internal oblique muscle close to the iliac crest have been described and have the same aspects (Fig. 13.8).

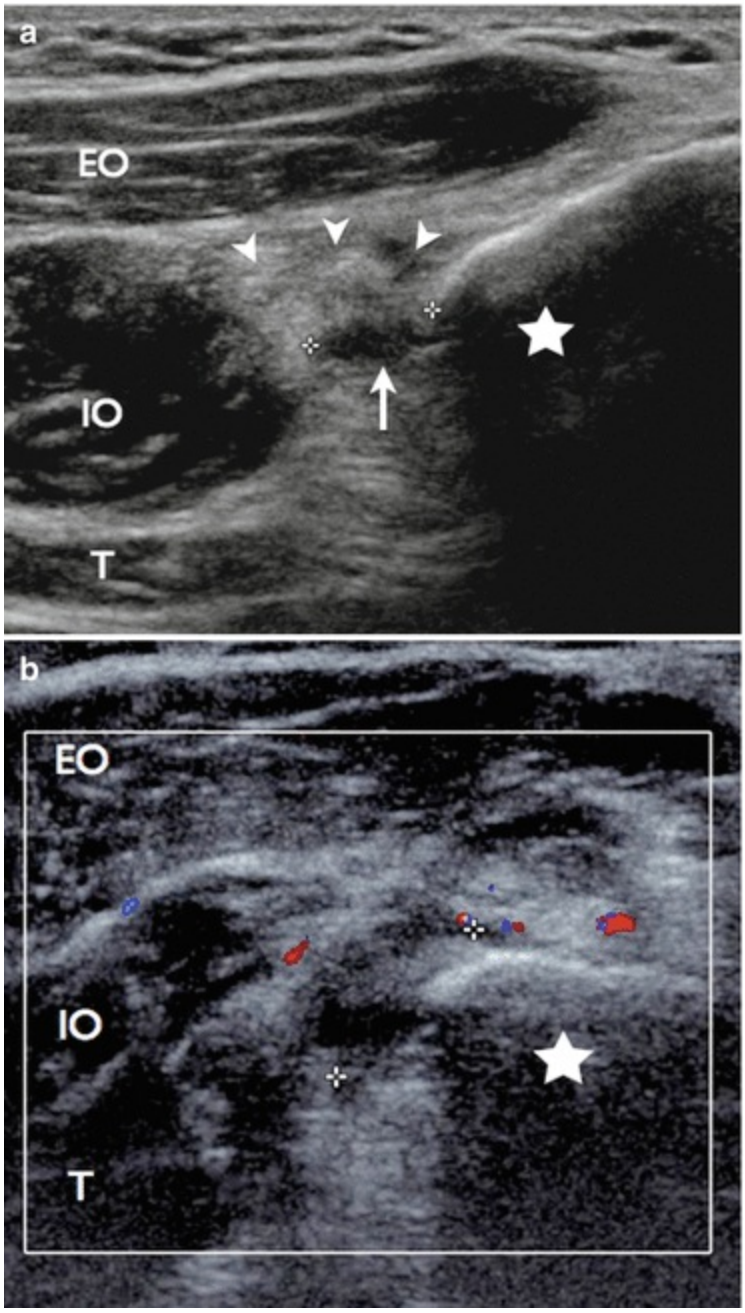

Fig. 13.8 Transverse ultrasound image with a 17–5 MHz transducer at the level of iliac crest. (**a**) Grade 2 strain of the internal oblique muscle appears heterogeneous with hypoechoic bleeding (*arrow*) and surrounding hyperechoic edema (*arrowheads*). (**b**) Color Doppler shows peripheral hyperemia. *EO* external oblique, *IO* internal oblique, *T* transverse muscle

13.5 Treatment

Preventive core strengthening exercise programs and rehabilitation techniques emphasizing eccentrics and plyometric strengthening of the abdominal wall muscles are commonly assumed to prevent such injuries and help reduce recurrences in athletes [10].

At an acute stage, initial treatment consists of rest, girdle compression, cryotherapy, oral non-steroidal anti-inflammatory drugs and physiotherapy. In the first week after onset isometric strengthening is indicated whereas concentric strengthening exercises start at week two, together with light stretching as well as aerobic conditioning. Usual recovery time is from 4 to 10 weeks [8].

Therapeutic injections of steroids and anesthetic under ultrasound guidance have been used to speed recovery and rehabilitation in professional baseball pitchers with acute side strains but are not currently used in our institution [18].

13.6 Conclusion

Although abdominal wall injuries are uncommon, it is necessary to know their distinctive patterns of injuries that may occur in athletes. Side strain injury occurs at the proximal insertion site of the internal oblique whereas rectus abdominis tears may be proximal or distal. MRI of the abdominal wall should be performed with sequences that minimize motion artifacts.

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14. Adductor Muscles Injuries

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Abstract
Adductor injuries are common among athletes. Because of the complex anatomical relationships between different muscle groups at the groin region and possibility of referred pain at this level, accurate clinical diagnosis can be difficult. Imaging plays a central role in diagnosing and directing management of adductor muscles injuries. This chapter offers an overview of the normal anatomy of the adductor and different imaging findings, mainly magnetic resonance imaging, of adductor injuries. Awareness of different types of adductor injuries and understanding their pathophysiology is paramount for musculoskeletal radiologists who must evaluate sports injuries.

14.1 Introduction

Adductor injuries are very common among athletes. Caused by twisting and turning, they are reported to have the highest incidence among all other match injuries in English

rugby players [1, 2]. Incidence of adductor injuries is higher among backs (3.1/1000 player hours) than forwards (2.0/1000 player hours) [1, 2]. High running loads, change of direction and kicking are generally considered the mechanisms for this type of sports injury [3]. There is a much confusion in the terminology of adductor injuries, with various terms used such as athletic pubalgia, groin pain, and osteitis pubis. While pain in the groin region may be secondary to different muscle groups, e.g., adductor and iliopsoas, it may also have a pelvic, genitourinary, gastrointestinal, or even neurologic etiology [4]. In addition, the adductor compartment encompasses a large anatomic area, with only its proximal insertion located at the groin. Injuries to more distal components of the muscle, e.g., myotendinous junction, do not necessarily result in groin pain.

In this chapter we will review the normal anatomy of the adductor muscles with an emphasis on their proximal insertions at the pubic region, describe technical considerations for imaging of these lesions, and explain different patterns of adductor injuries while emphasizing the clinical relevance of different imaging findings based on current knowledge in the field. To avoid confusion, and for didactic purposes, we will separate injuries to the myotendinous junction and muscle (muscle belly) from lesions to the tendon and enthesis at the proximal insertion.

14.2 Anatomy

The adductor muscles include the gracilis, pectineus, adductor longus (AL), brevis and magnus. All five muscles cross the hip joint but only the gracilis reaches beyond the knee. Although they are known collectively as adductors, their function is more complex as they also act from below, playing an important role in balancing the trunk on the lower limbs during walking [5].

14.2.1 Adductor Muscles

14.2.1.1 Adductor Longus

Along with the rectus abdominis (RA), the adductor longus is considered the most robust muscle for maintaining the stability of the anterior pelvis [6]. The AL lies anterior to the adductors magnus and brevis, and posterior to the pectineus. Its triangular belly arises from a narrow tendon and fibrocartilaginous enthesis off the anterior margin of the pubis, below the pubic crest [7]. At this level, both AL and RA attach in continuity via a single common sheath (RA-AL aponeurosis), blending with the underlying fibrocartilaginous disk. Awareness of this intimate anatomical relationship is important, as it is related to combined injuries to AL and RA attachments. At the proximal myotendinous junction, tendon fibers are predominantly anterior while the posterior surface mainly consists of muscle tissue. In addition the proximal tendon is less vascular towards the enthesis, which is important when considering the

pathogenesis and pattern of injury of adductor muscle group [7]. The muscle belly broadens caudally as it inserts on the middle third of the femoral linea aspera. The deep femoral artery supplies most of the muscle belly through a branch referred to as the artery of the adductors. The muscle is innervated by the anterior branch of the obturator nerve (L2, L3, and L4 branches) [6]. The adductor longus acts to adduct the thigh and contributes to hip flexion and medial rotation [8].

14.2.1.2 Adductor Brevis

The adductor brevis is located immediately posterior to the AL and anterior to the adductor magnus. It has a narrow origin from the anterior pubic body and inferior pubic ramus and becomes broader as it travels inferiorly. It attaches on the linea aspera, cranial to the site of insertion of the adductor magnus muscle. Similar to the AL, the adductor brevis has a fibrocartilaginous enthesis and an intramuscular tendon [7]. It is supplied by the artery of the adductors from deep femoral artery, and innervated by obturator nerve (L2 and L3) [6].

14.2.1.3 Adductor Magnus

The adductor magnus is a massive triangular muscle that originates at the lower margin of the inferior pubic ramus, and the ischial ramus and tuberosity. Different orientations of muscle fibers arise from each these sites. The uppermost fibers (from the inferior pubic ramus) are more anterior and more horizontally oriented, and insert on the gluteal tuberosity of the femur, medial to the insertion of the gluteus maximus. This part of the muscle is sometimes called the *adductor minimus* [5, 9]. The ischial ramus fibers travel obliquely and insert via an aponeurosis on the lower femoral linea aspera and medial femoral condylar ridge. The ischial tuberosity fibers taper to a rounded tendon that inserts on the adductor tubercle of the medial femoral condyle. Because the adductor magnus is so large, it is supplied by a number of arteries. The deep femoral artery mainly supplies the anterior part, and the medial circumflex femoral and popliteal arteries supply the posterior part. The muscle is innervated by the obturator nerve and tibial division of the sciatic nerve (L2, L3, and L4) [5].

14.2.1.4 Gracilis

The gracilis is the most superficial of the adductor group, overlying the adductor magnus and brevis. Its fibrocartilaginous enthesis [7] arises from the anterior aspect of the pubic body and the inferior pubic ramus by a thin and flat belly, and narrows inferiorly to form a rounded tendon that crosses the medial femoral condyle. The distal tendon inserts on the upper anteromedial tibia as part of the pes anserinus. Its main blood supply is from the artery of the adductors. It is innervated by the obturator nerve

(L2 and L3). The gracilis flexes the leg and rotates it medially, and may also act as an adductor of the thigh [5, 6].

14.2.1.5 Pectineus

The pectineus is a flat rectangular muscle in the femoral triangle, which overlies the AL. It arises from the pubic tubercle, pecten pubis and a portion of iliopectineal eminence. Distally it inserts on the posterior upper femur between the lesser trochanter and linea aspera. It is mainly supplied by the medial circumflex artery, and innervated by the femoral nerve (L2 and L3 branches), as well as the accessory obturator nerve [6]. The pectineus adducts the thigh and flexes it on the pelvis [5].

14.2.2 Symphyseal Joint and Proximal Insertions of Adductor Muscles

The symphyseal joint is important to consider when discussing injuries to the adductor group, given its intimate relationship with the adductor insertions. The symphyseal joint is a fibrocartilaginous joint with a central disc interposed between two hyaline cartilage-covered joint surfaces. Normally, these joint surfaces are smooth and well delineated, the central disc is contained within the joint capsule and there is often a small physiological fluid-filled space inside the disc, called a primary cleft [10]. At the anteroinferior margins of the symphyseal joint lies the arcuate ligament, which attaches to the symphyseal joint capsule and both pubic tubercles. This robust and clinically relevant structure is adherent to the pubic periosteum and apophyseal cartilage, forming a thick fibrocartilaginous plate, which in turn serves as the superficial attachment site for many core muscles and tendons [11].

The RA muscles are paired anteriorly and blend with the AL tendon origin to form a contiguous fibrous aponeurosis spanning the anterior pubic bones. The left and right RA-AL aponeuroses converge at the midline, where a thinner fibrous region extends anterior to the pubic symphysis forming the midline pubic plate [12]. Other adductor muscles originate from the paired pubic bones near the midline in close proximity to the RA-AL aponeurosis, including the most anterior pectineus, the adductor brevis just posterior to the AL, the more posterolateral adductor magnus, and the posteromedial gracilis. In addition to the complex musculoskeletal anatomy surrounding the pubic symphysis, the superficial inguinal ring is immediately lateral to the RA-AL aponeurosis contributing to the confounding clinical presentation of groin pain in athletes [12].

14.3 Imaging Modalities and Technical Considerations

14.3.1 Radiographs

Antero-posterior radiographs of the weight-bearing pelvis are obtained to assess bone, symphysis pubis, and coxofemoral joint space, and even detect a possible pelvic tilt. Soft tissue calcifications may be seen in the area surrounding the symphysis pubis [13].

14.3.2 Ultrasound

Ultrasound is often considered first line imaging due to its greater availability. The patient is examined with a linear probe in supine position with the thigh abducted at 30 degrees, externally rotated and the knee bent. The frequency of the transducer should be adjusted to the patient anatomy. The operator first identifies clinically the bulk of the AL in the anteromedial aspect of the thigh. It is helpful to identify the central aponeurosis in ultrasound images, and then follow it by moving the transducer proximally until the tendon is visualized. Axial and longitudinal images can be obtained and compared to the other side [13].

14.3.3 MR Examination

A dedicated MRI protocol is the study of choice for a specific subset of young athletic patients with groin pain suggesting an adductor injury. It is generally performed in supine position for patient comfort; the bladder should be emptied just before the MRI acquisition. Either 1.5 T or 3 T systems should provide high quality imaging of the pubic region. Coil selection and positioning are more important than field strength with modern scanners. Three large field-of-view sequences of the pelvis are first obtained using a body coil. A coronal T1-weighted sequence is useful for assessment of marrow abnormalities. A coronal short tau inversion recovery (STIR) is useful for both osseous and soft tissue pathology, as well as detection of abnormal fluid, and allows for homogeneous suppression of fat, despite relatively poor signal-to-noise ratio. An axial large field-of-view fast spin echo T2-weighted fat-suppressed sequence allows adequate contrast and coverage to identify marrow edema, hip flexor injury, and even visceral pelvic lesions [14].

14.4 Adductor Injuries

14.4.1 Adductor Injury of the Muscle and Myotendinous Junction

Strains to the adductor muscles and myotendinous junctions are very common with activity [6, 12] (Figs. 14.1 and 14.2). When isolated, an adductor injury is most commonly centered at the proximal myotendinous junction of the AL, with the adductor magnus and pectineus less frequently injured [12] (Fig. 14.3). Tears limited to the muscle and myotendinous junction seem to have a favorable prognosis, as most patients

usually do well with conservative management [12]. MRI protocol should include the full distal extent of pathology in the thigh [12]. Several classification systems have been published to categorize the severity of muscle injuries. [15–20]. The clinical relevance of these classifications is not proven, and the only consistently reported high level evidence in muscle injuries simply suggests that a "negative" MRI carries a better prognosis than a "positive" MRI [15, 21–23].

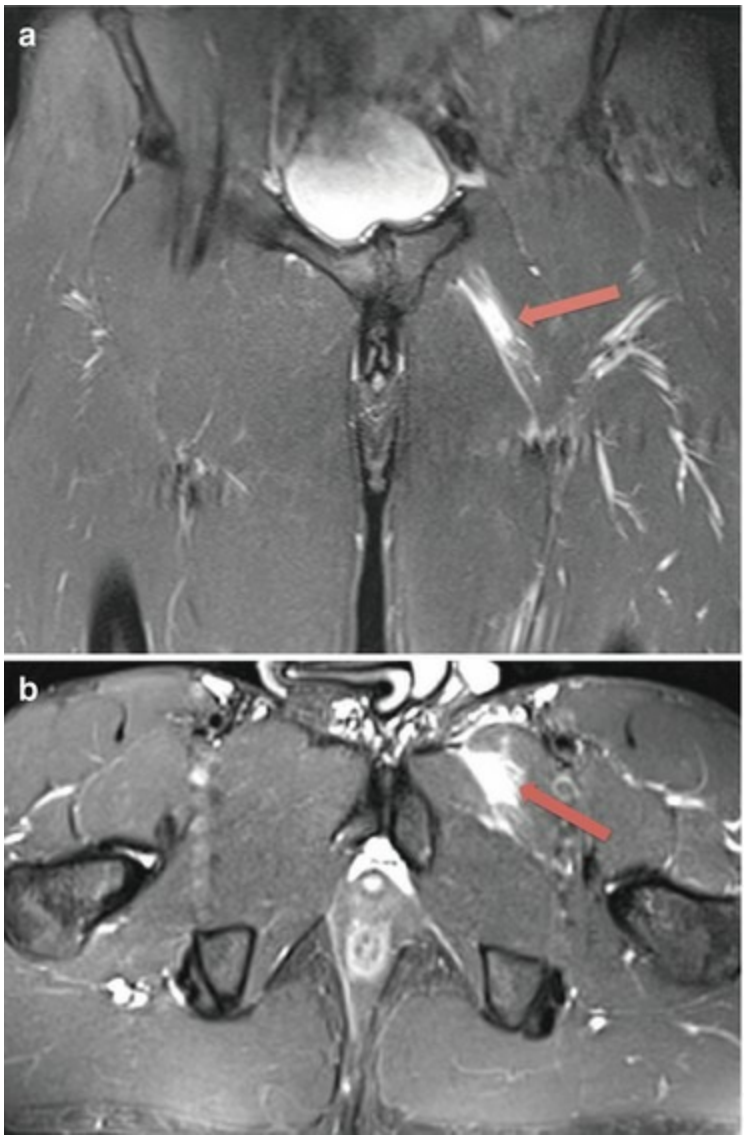

Fig. 14.1 A 28-year-old male athlete with an injury to the myotendinous junction of the left adductor longus (AL). (**a**) Coronal and (**b**) axial short tau inversion recovery MRI show intrasubstance high signal intensity (*arrow*) at the proximal portion of the left AL, distant from its osseous attachment, consistent with an injury to the myotendinous junction

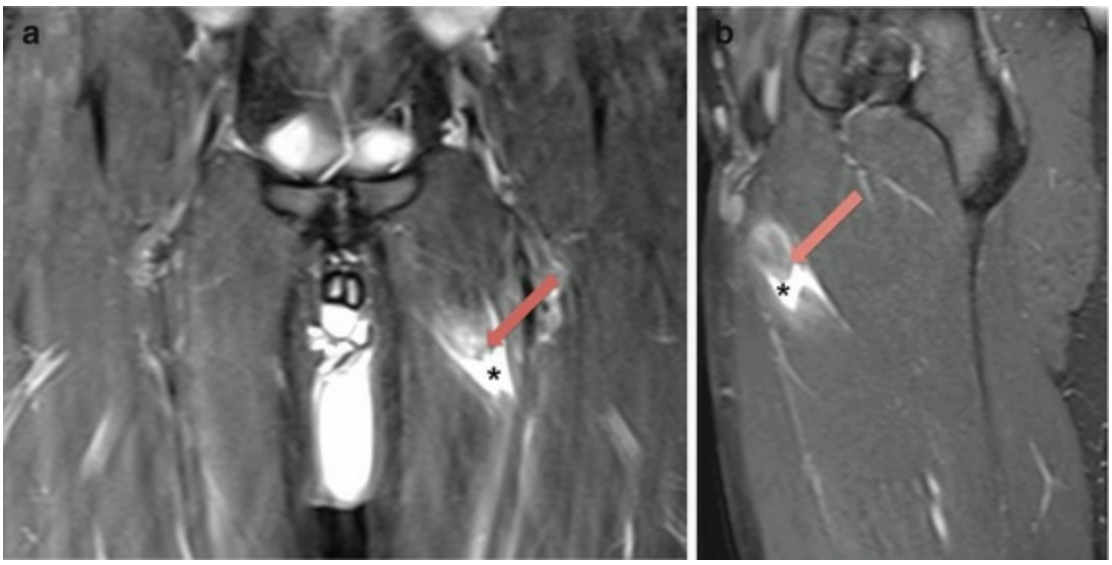

Fig. 14.2 A 20-year-old male athlete with a tear of the right adductor longus at the proximal myotendinous junction. (**a**) Coronal and (**b**) sagittal short tau inversion recovery MRI show a tear at the myotendinous junction with retraction of the proximal fibers (*arrow*). Note the presence of hemorrhage delineating the torn muscle (*asterisk*)

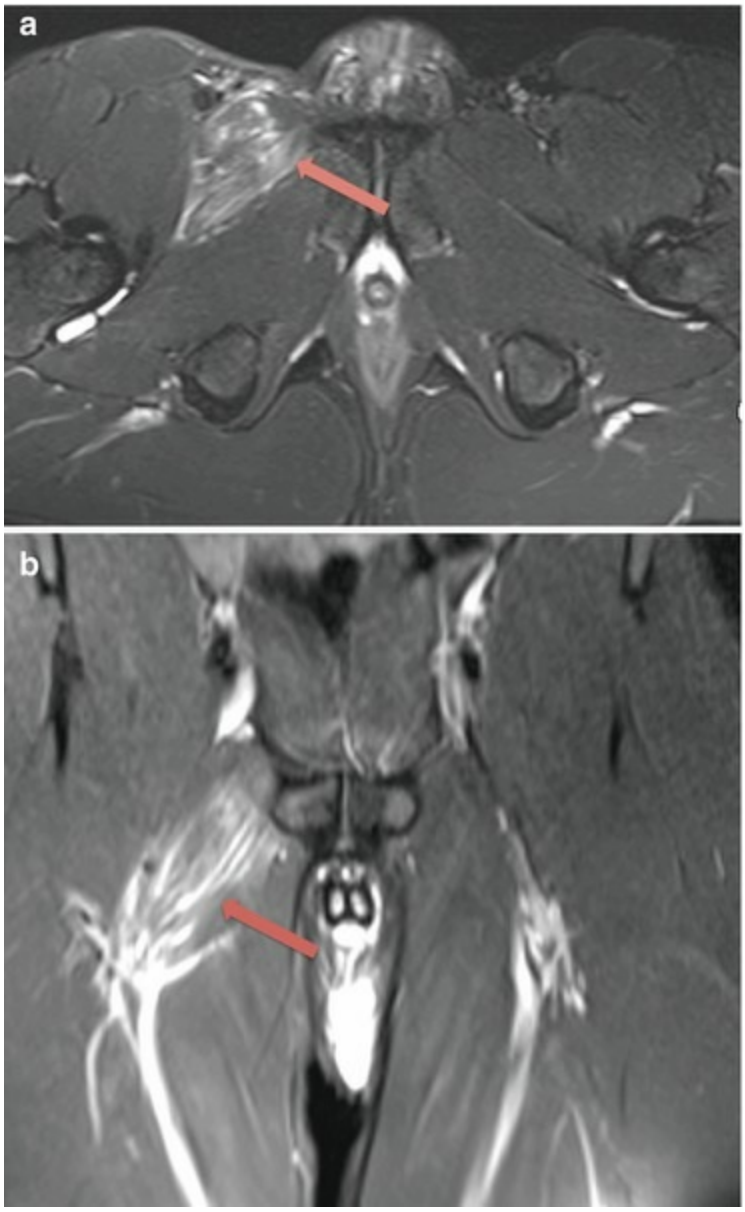

Fig. 14.3 A 24-year-old male athlete with an intrasubstance tear of the right pectineus muscle. (**a**) Axial, and (**b**) coronal short tau inversion recovery MRI show right pectineus muscle is enlarged and displays intra-substance high signal intensity interspersed within muscle fibers (*arrow*), in keeping with muscle strain. The proximal attachment of the right pectineus is intact

MR findings of acute muscle strain include enlargement and increased signal intensity in fluid-sensitive sequences dissecting muscle fibers, resulting in "feathery edema" appearance. Perifascial edema can also be seen. These abnormalities may outline adjacent femoral neurovascular structures, given the proximity of the adductor canal [24].

Associated hemorrhage may appear as fluid-like intensity tracking along fascia and muscle fibers. Hematoma appears as a homogeneous fluid collection in the early phase. Long-standing organized hematomas tend to be more heterogeneous. Chronic injuries may show fibrous scarring, which appears as hypointense fascial thickening in fluid

sensitive images. In ultrasound, there is loss of the normal pennate appearance of the muscle, with areas of altered echogenicity, and loss of perimysial striation adjacent to the myotendinous junction. In complete tears, interruption and retraction of muscle fibers can be seen usually with surrounding fluid collection corresponding to hematoma [25].

Recurrent and chronic adductor muscle strain, extensive intermuscular fluid and intramuscular edema can cause an "exercise-induced compartment syndrome" [12]. If suspected, axial T2-weighted images should be acquired before and after exercise to compare the degree of mass effect [12].

14.4.1.1 Baseball Pitcher-Hockey Goalie Syndrome

Rarely the AL can be herniated through its epimysium, causing a prolonged course of pain. This entity called "baseball pitcher-hockey goalie syndrome" may be seen along with a chronic or repaired injury to the RA-AL aponeurosis [12, 14]. It is thought that an epimysial defect may result from chronic repetitive stress at the sites of relative weakness, such as entry points of neurovascular structures [14, 26]. Although this injury occurs distal to the tendon insertion, patients often report groin pain [6] – likely because of associated RA-AL injury. A detailed medical history (acute onset of pain worse after stretching) and careful physical examination (site of pain distal to the pubic symphysis, over the herniated area) may suggest this diagnosis [6]. MRI findings are not well described although one may see focal edema in the AL muscle belly, with a focal bulge at the site of herniation [27]. "Baseball pitcher-hockey goalie syndrome" is reported to respond poorly to conservative management and treatment of recalcitrant pain requires surgical epimysiotomy and debridement [6].

14.4.2 Adductor Injury of the Tendon and Enthesis

Because of the anatomic complexity of the groin region, and intimate relationships of tendons from different muscle groups at this level (such as the iliacus and adductor), there is much confusion about the terminology used to designate the clinical entity related to adductor tendon and insertion injuries. Terms such as athletic pubalgia, osteitis pubis, and sports hernia have been used [28]. To overcome this confusion, a group of experts in sports medicine met in Doha in 2014 to reach an agreement on the definitions and classifications of different entities that cause groin pain [29]. They concluded by suggesting the term "adductor-related groin pain", defined as tenderness *and* pain on resisted adduction testing at the insertion sites of adductor muscles [29].

Two prospective studies have used clinical examination and imaging for diagnostic confirmation of diagnosis of acute adductor-related groin pain [3, 30]. Only the more recent study by Serner et al. included MRI findings [3]. Among 110 athletes with acute groin injuries, Holmich et al. found a high prevalence of clinically defined adductor

injuries (73 individuals, 66 %) [3]. However, only 54 of the 73 (73 %) were examined with MRI, which showed the AL to be most commonly affected (50 cases, 93 %), followed by the pectineus (10 cases, 19 %), and adductor brevis (9 cases, 17 %). One gracilis and one adductor magnus were affected (2 %) [3]. The same study showed a better correlation between clinical and radiological findings for clinically diagnosed adductor-related groin pain when compared with injuries to the iliopsoas, rectus femoris and sartorius. When multiple adductor muscles are involved, injuries of the AL, adductor brevis and pectineus are often combined [3]. Avulsion and retraction of the AL should prompt assessment of the caudal RA, as these injuries are often seen concurrently [14]. Unfortunately very little is known about diagnostic reliability of cross-sectional imaging for adductor injuries [4].

A recent review of the literature reported four main consistent radiological findings for long-standing symphyseal and adductor-related groin pain across studies: pathology of the adductor muscle insertions, pubic bone marrow edema, the "secondary cleft sign", and degenerative changes of the symphyseal joint [4].

14.4.2.1 Pathology of the Adductor Tendons

On MRI the affected tendons, mostly the AL, may show several findings including edema, contrast enhancement following gadolinium injection [31–33], and partial or total disruption, i.e. avulsion. In this case there is retraction of the distal portion of the tendon with associated fluid signal collection at the site of rupture, related to hematoma (Figs. 14.4, 14.5, and 14.6). Robinson et al. reported that abnormal enhancement at the site of enthesis shows good correlation with the site of pain [32]. In chronic injuries, tendon thickening can be seen [6, 34]. In this case, there is enlargement and hypointense signal of the tendon on fluid-sensitive images with possible focal rounded hypointensity representing hydroxyapatite deposition tendinosis [6, 14].

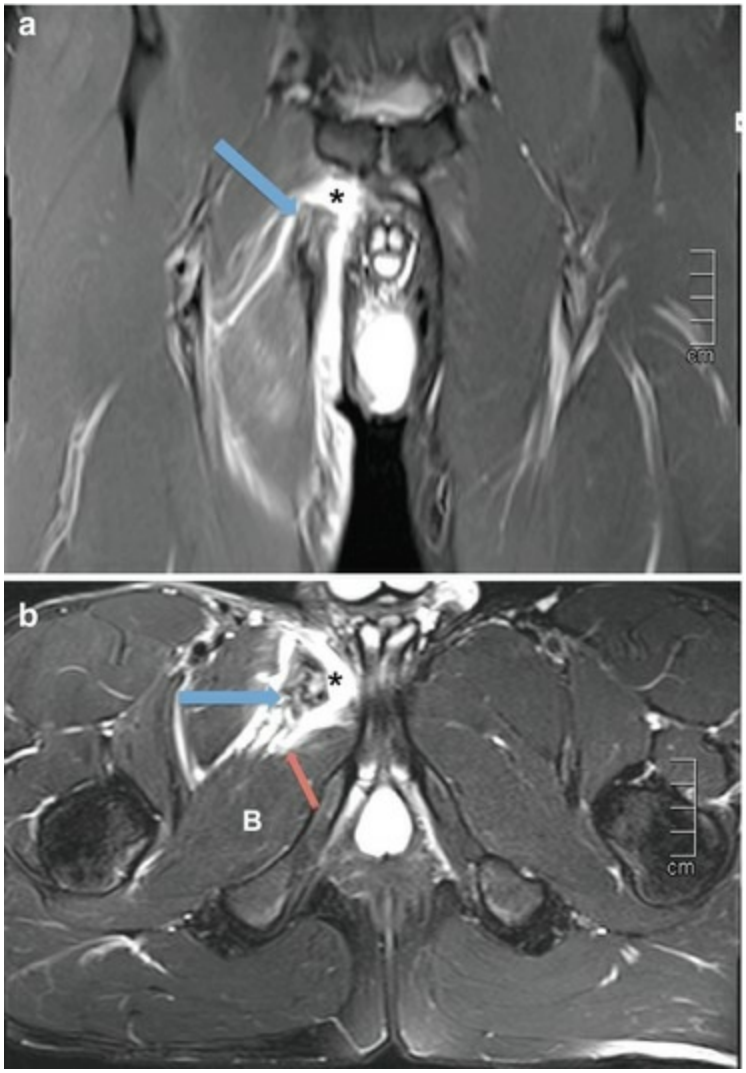

Fig. 14.4 A 21-year-old male athlete with avulsion tears to the right adductor longus (AL) and brevis muscles. (**a**) Coronal and (**b**) axial short tau inversion recovery MRI show complete discontinuity with retraction of the proximal attachment of the right AL (*blue arrow*) surrounded by fluid, consistent with hemorrhage (*asterisk*). In the axial image, the tendon rupture appears to involve the adductor brevis (**b**), as suggested by the irregularity of its anterior surface (*red arrow*)

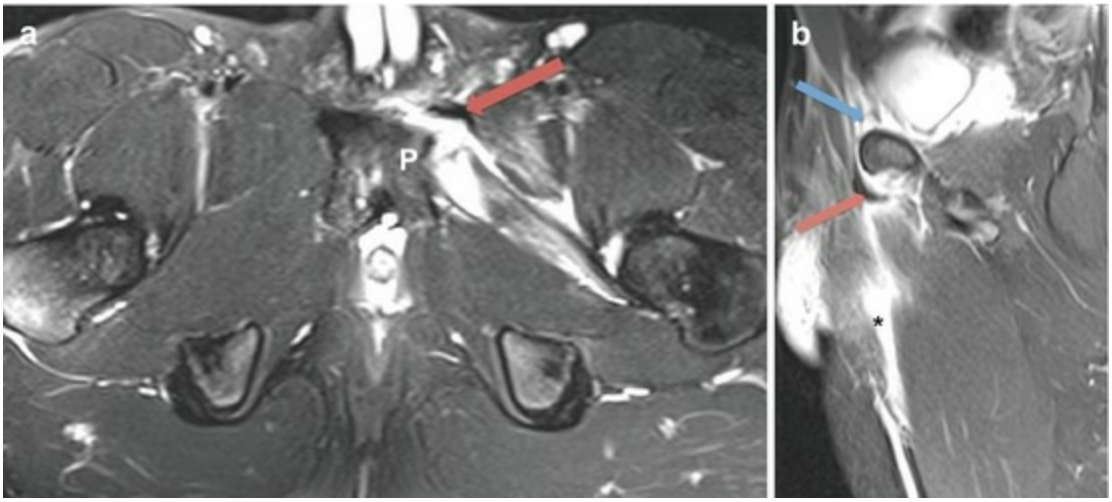

Fig. 14.5 A 19-year-old male athlete with tenoperiosteal detachment of the rectus abdominis-adductor longus (RA-AL) aponeurosis. (**a**) Axial and (**b**) coronal short tau inversion recovery MRI show avulsion of the proximal tendon of the left adductor longus (AL) with tenoperiosteal detachment of the RA-AL aponeurosis (*red arrow*) from the pubic tubercle (*P*). The tear extends to the distal rectus abdominis attachment (*blue arrow*). Note fluid signals tracking along the torn AL muscle (*asterisk*), consistent with hemorrhage

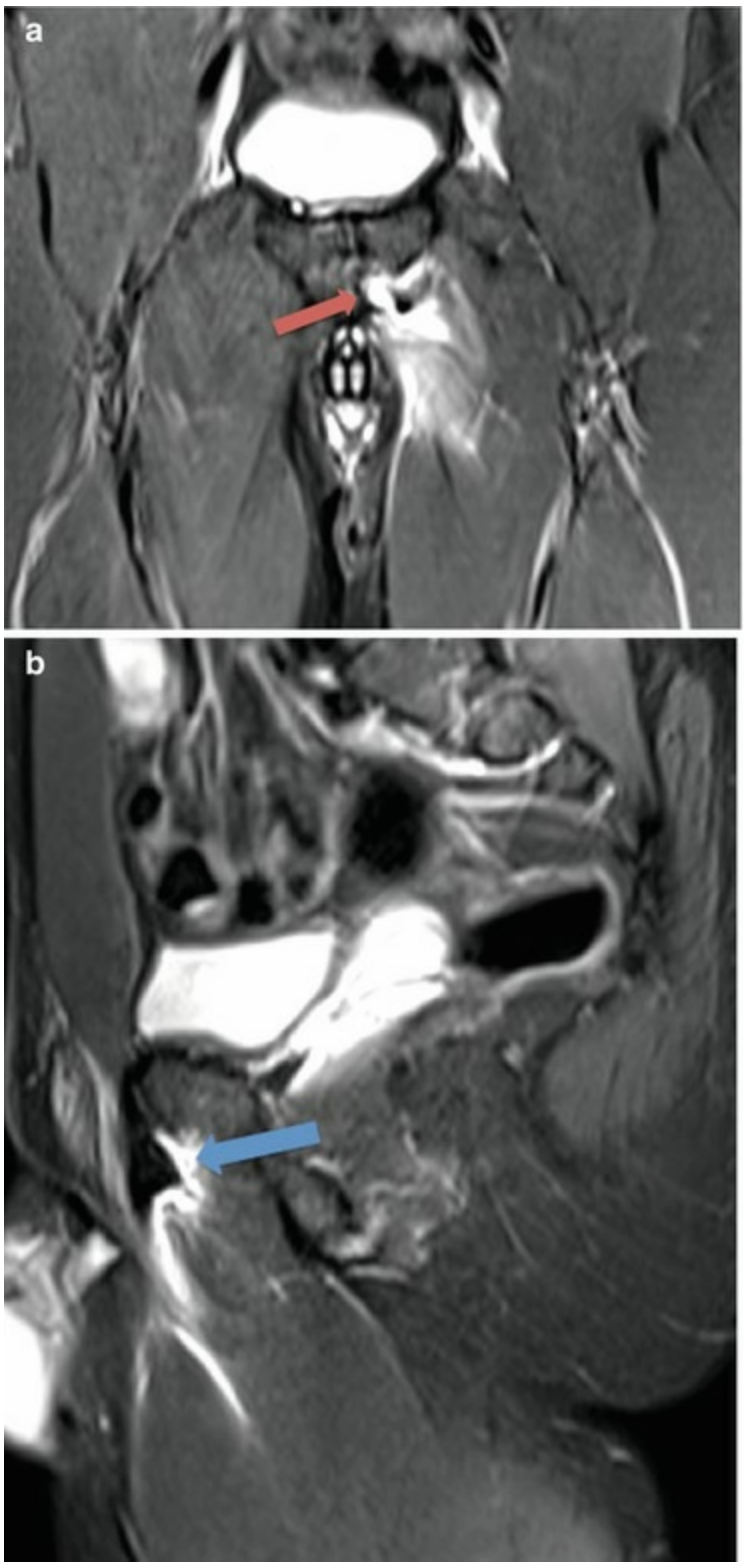

Fig. 14.6 A 31-year-old male athlete with adductor longus (AL) proximal tendon avulsion and detachment of the rectus abdominis-adductor longus (RA-AL) aponeurosis. (**a**) Coronal and (**b**) sagittal short tau inversion recovery MRI show avulsion of the proximal tendon of the left AL with an osseous defect at the left pubic tubercle (*red arrow*). Sagittal image shows tenoperiosteal detachment of RA-AL aponeurosis (*blue arrow*)

On ultrasound, detection of tendon changes is made easier by comparing the contralateral side. Unfortunately changes appreciated by ultrasound in adductor injuries are not always distinguishable from uncomplicated tendinosis or normal variations in athletes. Color Doppler imaging may show hyperemia, but it is not a consistent finding [13].

RA-AL Aponeurosis Injury

Adductor tendon avulsion is most commonly associated with injury to the RA-AL aponeurosis, which in turn can predispose to large multitendon adductor tears. For this reason, careful assessment of any small detachment of the RA-AL aponeurosis is necessary in case of injury to the AL tendon [12]. This occurs lateral to midline, at the attachment site to the pubic tubercle. This tenoperiosteal disruption may be most visible on axial and sagittal fluid-sensitive images acquired approximately 1–2 cm lateral to the pubic symphysis, where it appears as irregular areas of fluid signal intensity undermining the aponeurosis (Figs. 14.5 and 14.6) [6]. Unilateral RA-AL aponeurosis lesions can range in severity from very small lateral edge detachments to severe distractive tears with the RA retracted cephalad and AL retracted distally into the medial thigh. Chronic unilateral RA-AL lesions may manifest on MRI as atrophy of the RA in cross section just above the pubis, or as mild asymmetric bone marrow edema at the pubic tubercle in the smaller field of view sequences. When a tear to the proximal adductor insertion is diagnosed, it is crucial to look for an associated RA injury, as treatment of the adductor injury without addressing the RA can lead to recurrence and protracted pain [12].

Recently, Coker and Zoga described a subset of severe RA-AL aponeurosis tears that combines pectineus and AL avulsion, termed "*combined anterior adductor avulsion lesion*" [12]. Although the MRI appearance is dramatic, the combined avulsed tendon group can be reduced and fixed with relative ease with surgery, and athletes often return to play within 8 weeks [12]. In contrast, organized soft tissue hematoma in the setting of high grade RA-AL aponeurosis lesions can serve as a harbinger of delayed return to activity, and collections can localize superficial to the pubis, insinuate into the inguinal canal, or even extend into the peritoneal cavity. Coronal oblique images are often very useful in localizing fluid collection with regard to the inguinal canal.

Midline Pubic Plate Lesion

In addition to the RA-AL aponeurosis detachment, there is another dominant pattern of injury, which occurs at the midline anterior to the pubic tubercle. This injury pattern is

termed "*midline pubic plate lesion*", and often extends through the lateral edge of RA-AL aponeurosis, unilaterally or bilaterally [12]. Other associated injuries are also often asymmetric, including secondary cleft, bone marrow edema and osseous resorption. This asymmetry correlates with clinical findings with pain usually predominant on the side of greater bone marrow edema or osseous resorption [12].

Incipient Breach

Another MR feature was recently described by Coker and Zoga, the "*incipient breach*", which is thought to occur at the early stage of midline pubic plate lesions. Sagittal fluid-sensitive MRIs reveal a very small detachment of the fibrous plate at midline from the anterior aspect of the symphyseal capsule with disruption of the antero-inferior pubic symphysis. It is hypothesized that the incipient breach may indicate the site of initial injury, which is potentially helpful for repair [12].

14.4.2.2 "Secondary Cleft" Sign

The so-called "secondary cleft" sign is an apparent curvilinear area extending inferiorly from the central symphyseal fibrocartilaginous cleft along the anteroinferior margin of the pubic body. Although its clinical significance is still debatable, some authors have interpreted it as a possible consequence of a micro-tear or traction force at the site of the pubic bone, and thus as another sign of a lesion in the attachment site of AL and gracilis [6, 35]. It was initially described at arthrography of the pubic symphysis, and later reported in unenhanced MRIs obtained with fluid-sensitive pulse sequences, on which it appears as a curvilinear region of high signal intensity adjoining the pubic symphysis (Fig. 14.7) [35–37]. Its prevalence in athletes with symphyseal pain varies between 52 % [38] and 88 % [35] and is reported to correspond to the site of pain in all cases. Interestingly the secondary cleft sign has not been found among asymptomatic individuals whether they be athletes [35, 37] or sedentary non-athletes [38].

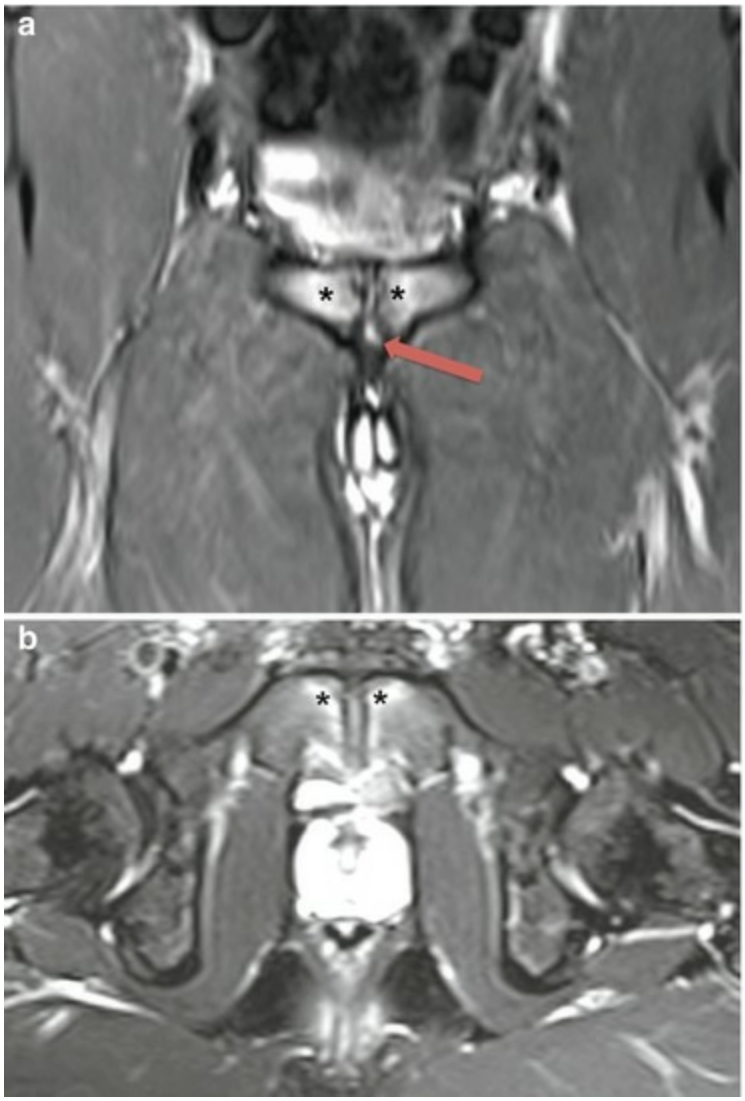

Fig. 14.7 A 25-year-old male athlete with pubic bone marrow edema and "secondary cleft sign", presenting with a 10-weeks history of left groin pain. (**a**) Coronal and (**b**) axial short tau inversion recovery MRI show bilateral anterior pubic bone marrow edema (*asterisks*) and linear hyperintense signal along the anteroinferior margin of the left pubic tubercle (*arrow*), consistent with secondary cleft sign

14.4.2.3 Pubic Bone Marrow Edema

As discussed in the "RA-AL aponeurosis" section, pubic bone marrow edema is suggestive of an aponeurotic lesion. It is typically located at the anterior-inferior aspect of the pubic body, deep to the RA-AL aponeurotic attachment (Fig. 14.7) [32]. In a retrospective study including 52 athletes, anterior pubic edema and enhancement showed reproducible correlation with the site of pain on clinical examination [32]. There is no reliable and reproducible grading scale for pubic bone marrow edema, and thus its assessment remains subjective [4]. Pubic bone marrow edema may be seen in asymptomatic athletes; the reported prevalence varies widely from 0 % [35] to 61–65 % [39, 40]. It is reported, however, to be more prevalent and more severe in

symptomatic athletes [4].

14.4.2.4 Osseous Changes to the Symphyseal Joint

Osseous changes around this joint include erosions and irregularities, subchondral sclerosis and cysts, joint space widening or narrowing, central disc herniation, as well as anterior and posterior osteophytes [6, 41].

A presentation of advanced osseous changes is osteitis pubis, where repetitive injuries result in inflammatory response with osteitis and periostitis. This pattern frequently overlaps with RA and AL tendon dysfunction [42]. In case of acute onset, symptoms and imaging findings can be indistinguishable from septic arthritis and osteomyelitis of the pubic symphysis [6]. However pubic symphyseal infection is uncommon in the athletic population [6]. Radiography shows subchondral sclerosis, symphyseal irregularity, and bone resorption. MRI shows diffuse edema extending from the subchondral plate, often involving both pubic bodies [43].

14.5 Conclusion

Musculoskeletal imagers should be familiar with imaging patterns, including an isolated adductor injury, RA-AL aponeurosis lesion, midline pubic plate lesion, and various musculoskeletal and visceral concomitant pathologies. Understanding the pathophysiology of these injuries and their accompanying MRI findings can play an important role in building an effective treatment strategy.

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15. Iliopsoas Muscles Injuries

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Abstract
The anatomy of the iliopsoas is complex, extending from the lumbar fossa to the upper thigh. Traumatic lesions of this muscle are rare and often misunderstood. Team sports and canoeing are the activities that most commonly produce traumas of the psoas. The lesions are to be found in three highly distinct locations. Lesions of the fleshy body lead to a noisy clinical picture with large hematoma. Injuries to the myotendinous junction are often small in size, resulting in very late diagnosis. Low disinsertions usually occur in older athletes. They are immediately disabling. Ultrasound or MRI scans will confirm the diagnosis at an early stage. Medical and functional treatment is the rule, except when there is a compressive hematoma or displaced avulsion of the lesser trochanter, in which case surgery may be considered.

15.1 Introduction

The iliopsoas as the main hip flexor is solicited in many sporting gestures. Its injuries during sports practice are rarely described and often unknown. Lesions can occur

throughout the path of the long muscle causing very different clinical issues. In most cases, the peculiarity of traumatic lesions of the psoas lies in the difficulty of early diagnosis. This allows simple management. But the complexity of the anatomy of the groin and the large number of possible lesions often lead to an array of chronic lesions with more difficult management. This chapter will take the reader thru the anatomy and the pathophysiology of traumatic lesions of the iliopsoas. The various lesions are listed and the many differential diagnoses as well. Clinical and para-clinical diagnostic processes are detailed as well as the management of acute and chronic injuries.

15.2 Anatomy

The iliopsoas muscle is formed of the psoas and iliacus muscles. It is paired, symmetrical and polyarticular.

15.2.1 The iliacus

The *iliacus* is a thick, wide, fan-shaped muscle that fills a large part of the internal iliac fossa. It is inserted on one side into the internal iliac fossa, except for its anterior inferior part, and the edge of the internal iliac fossa on the other, most notably the inner lip of the iliac crest, the iliac spine and the innominate notch and the base of the sacrum. The muscle fibers of the iliacus then converge towards the distal part of the psoas major and its tendon.

15.2.2 The Psoas Major

The *psoas major* is inserted into the anterior and lateral aspect of the twelfth dorsal vertebra and the five lumbar vertebrae via fibrous arches through which the lumbar vessels and the rami of the sympathetic nervous system travel. It then continues downwards, forwards and outwards. It goes across the deep aspect of the iliac fossa where it joins the iliacus. Next, it changes direction, running obliquely downwards, outwards and rearwards. At this point it forms the myotendinous junction as it goes over the iliopectineal eminence then immediately to the fore of the acetabular capsule and the anterior acetabular labrum [1]. At this level the tendinous and muscular proportions are equivalent over a transversal section of psoas major [1].

15.2.3 The Myotendinous Junction of the Iliopsoas(or Femoral Portion)

The *myotendinous junction of the iliopsoas (or femoral portion)* is histologically different. It is formed very high up with a double tendinous plate which progressively thickens to the rear of the femoral arch and a muscle component. Insertion takes place at

the summit of the lesser trochanter [2]. This myotendinous junction comprises three parts [3]. *The first part* is formed by the main tendon of psoas major which forms gradually from this muscle to constitute the anterior fibers of the tendon at the inguinal ligament. Initially oriented in a strictly frontal plane, this tendon undergoes a characteristic spiral rotation. The ventral fibers become medial and the dorsal fibers lateral. In a more lateral position, *the second part* is formed by the accessory tendon which follows on from the most medial muscle fibers of the iliacus. This tendon then gradually merges with the main tendon over a height of 6–8 cm. *The third part* is formed by the most lateral fibers of the iliacus with a constant pure muscle fascicle, progressing over the iliopectineal eminence below the main tendon and then inserting into the lesser trochanter [3]. When it passes immediately to the fore of the hip, the iliopsoas muscle is separated from it by a synovial bursa which often adjoins the joint cavity, [2, 4] most notably in incidences of symptomatic clicking [5]. This synovial bursa is the largest in the body and can reach a height of 6–7 cm and a width of 3–4 cm [3, 5]. The upper part of the synovial bursa is divided into two compartments, medial for the main tendon and lateral for the accessory tendon [3]. It runs irregularly downwards, but also upwards, coming between the tendinous deep aspect of psoas major and the bony margin, and for this reason is known as the iliopectineal bursa.

15.2.4 Insertion of the Iliopsoas Muscle into the Lesser Trochante

Insertion of the iliopsoas muscle into the lesser trochanter takes place via the main tendon, extending the psoas major, and the accessory tendon which has joined it. These two tendons run directly towards the lesser trochanter where they are inserted without interposition.

The iliacus is inserted directly into the anterior side of the lesser trochanter and beneath it, without forming a tendon (subtrochanteric muscle bundle) [3].

15.3 Physiopathology

The iliopsoas is the main hip flexor, acting on the thigh or the trunk depending on the fixed point. It is also capable of imparting a lateral rotational movement to the hip and contributes to the lateral inclination of the trunk. It also plays a part in the motor pattern of many movements in sports (kicking a ball, leg stance, whitewater canoeing draw stroke…). It plays a fundamental role in the statics of human beings when they are standing and in this respect is significant in lordosis.

Several types of lesions are encountered in sports pathology, differing according to their location and their nature.

15.3.1 Traumatic Lesions of the Iliopso

Traumatic lesions of the iliopsoas are rare and barely described in the literature. Certain lesions of the fleshy body in the retroperitoneal space or the pelvis can cause extensive bleeding and a compressive hematoma (Fig. 15.1).

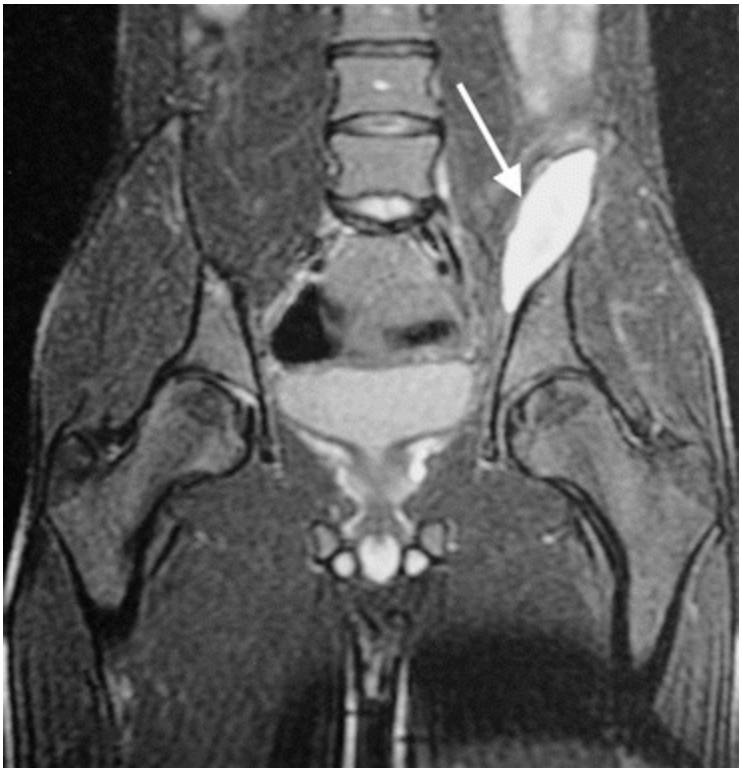

Fig. 15.1 Coronal contrast-enhanced T1-weighted SPIR pelvic MRI shows hypersignal at the myotendinous junction of the left iliopsoas muscle and its iliac insertion with hematoma (*arrow*)

15.3.2 Acute Traumatic Lesions of the Myotendinous Junction

Acute traumatic lesions of the myotendinous junction occur in sports which feature repeated hip flexions, stiffness of the psoas, and episodic or random flexion against resistance. These include a backward kick of the ball by a football player, or the lateral stance of the scrum-half as he removes the ball from a ruck. In canoeing the powerful draw stroke is the most damaging movement. With the paddle behind the canoeist and his thighs held by the boat, the trunk switches violently from extension to flexion. All these movements combine an eccentric contraction of the iliopsoas muscle. It is therefore logical that along this stretched muscle-tendon chain, the vulnerable area is the reflexion of the myotendinous junction on the iliopubic ramus.

15.3.3 Chronic Lesions of the Myotendinous Junction

Chronic lesions of the myotendinous junction follow on from an untreated acute lesion. The debilitating pain of the first few days is often replaced by a remission with persistent discomfort in certain movements. If it does not heal or the healing is not solid enough before resumption of sports activity, recurrence is common with chronic pains similar to those observed far more frequently in the hamstrings, caused by healing of poor functional quality.

15.3.4 Traumatic Low Ruptures of the Tendon

Traumatic low ruptures of the tendon, with or without an avulsion injury of the enthesis on the lesser trochanter [6], are less common. These low ruptures mainly seem to occur in patients aged over 65. Diagnosis is often delayed because the clinical presentation of these ruptures is deceptive [7]. They sometimes appear spontaneously with a myotendinous retraction [8].

Younger patients more often present *enthesitis or apophysis avulsion of the lesser trochanter* [9]. Ossification of the apophysis appears at around 8–9 years of age and fusion occurs at around 16–18 years. The lesion most often comes further to an excessive contraction of the iliopsoas muscle on a hip in abduction and/or in hyperextension.

Conflicts between the deep aspect of the iliopsoas tendon and the hip joint, with *clicking*, have been described by various authors [10]. More recently, thanks to complementary dynamic ultrasound [11], the painful *snapping* of the anterior face of the hip has been connected to instability due to a sudden turn of the psoas tendon by the iliopectineal eminence.

Bursitis of the iliopsoas has also seen developments [4]. It is very often secondary to a pre-existing pathology of the adjoining hip joint (inflammatory or degenerative). Bursopathies of intrinsic origin, not adjoining the joint, are encountered when there is a conflict with the iliopectineal eminence or in the event of activities with overstress.

Certain non-traumatic lesions of the iliopsoas have also been described. Among them we can mention *abscesses* or *hematomas* complicating an anticoagulant treatment, as well as *osteochondrosis* of the lesser trochanter. The sometimes extensive *ossifications* are secondary to hematomas, to neurological deficits or to surgery of the hip joint.

15.4 Epidemiology

Traumatic lesions of the iliopsoas are rare. [6] Prevalence of myotendinous and low tendinous lesions in the general population is around 0.66 %. Etiologies differ according to age, with a predominance of traumatic lesions of the myotendinous junction among people doing sport before the age of 65. In this area, basketball, rugby, football

and canoeing are the sports that must commonly cause trauma to the psoas major. After the age of 65 there is a predominance of complete ruptures of the tendon with disinsertion appearing spontaneously [12], whereas enthesopathy or apophysis avulsion is more common among children and adolescents.

15.5 Clinical Examination

15.5.1 Muscle Lesions Proximal to the Iliopsoas

Traumas to the iliopsoas can affect the proximal part upstream of the myotendinous junction. It is a rare lesion, of the muscle only, in the lumbar fossa or the iliac fossa. Clinicians should be particularly attentive when this trauma occurs in patients with hemophilia, coagulation disorder or those taking an anticoagulant treatment. The heavily vascularized and innervated environment will result in a particular clinical picture. They start suddenly, for example with an uncontrolled slide or blocked hip flexion. The symptomatology is unclear from the outset with deep pains, and lack of movement on palpation. Complications may appear rapidly due to the extensive hematoma which develops in the muscle, with psoitis [1] causing permanent flexion of the hip and cruralgia with superficial loss in sensitivity on the anterior aspect of the thigh and partial motor loss in the quadriceps. If diagnosis and therapy are not performed in time, post-traumatic ossifying myositis may occur [13].

15.5.2 Lesions of the Myotendinous Junction

This is the most common location although it has very rarely been documented in the literature. In a previous work we assessed the sensitivity of clinical signs in a series of 33 cases [14]. The reason for consultation is the appearance of anterior hip pain which is either sudden (45 %) or rapidly progressing (55 %). It occurs in the middle of an activity, particularly when kicking a ball or changing foot position in team sports, or a violent draw stroke in kayaking. Team sports represent 69 % of cases.

Limping and psoitis are initially present in one third of cases and five times out of six the lesion affects the dominant side. In 50 % of cases, these unclear symptoms are replaced after a few days by an insidious picture that systematically evolves to chronic mode if the diagnosis is not done initially. Average treatment time is 5 days when performed early, rising to 3 months on average for injuries treated late.

The examination starts with a search for negative signs. Hip joint mobility is conserved. No clicking is observed. Palpation of the bony structures is painless, particularly on the anterior superior iliac spine (avulsion) and the ischiopubic ramus (stress fracture). The neurovascular examination is normal. There is no hernia.

The pain is in the groin, limiting the rear step. Pain on stretching is only present in 18 % of cases. It should be located with the patient prone, the clinician’s hand blocking

pelvic retroversion, and the patient's hip in slight abduction (Fig. 15.2).

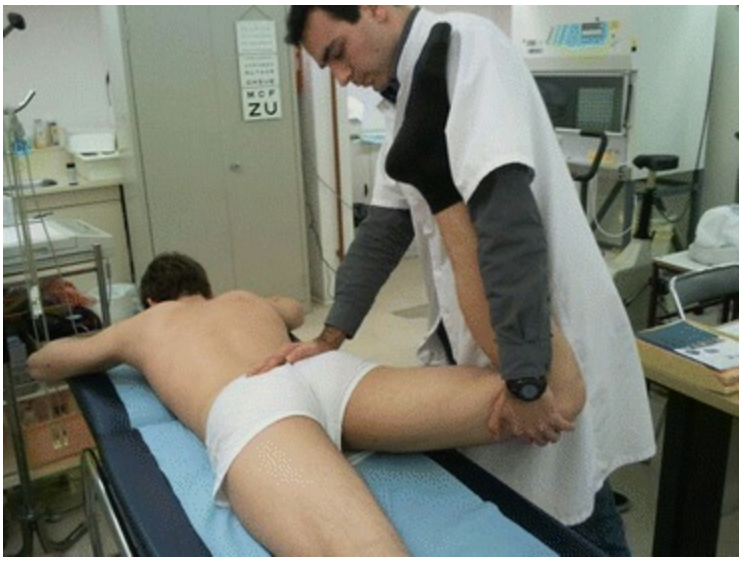

Fig. 15.2 Locating pain by stretching the iliopsoas

In 78 % of cases the pain occurs in contraction against resistance in the supine position, with the knee in extension and the hip in slight abduction (Fig. 15.3). This sign is all the more valuable if there is no pain when the knee is extended against resistance, bringing into play another hip extensor, the anterior femoral muscle.

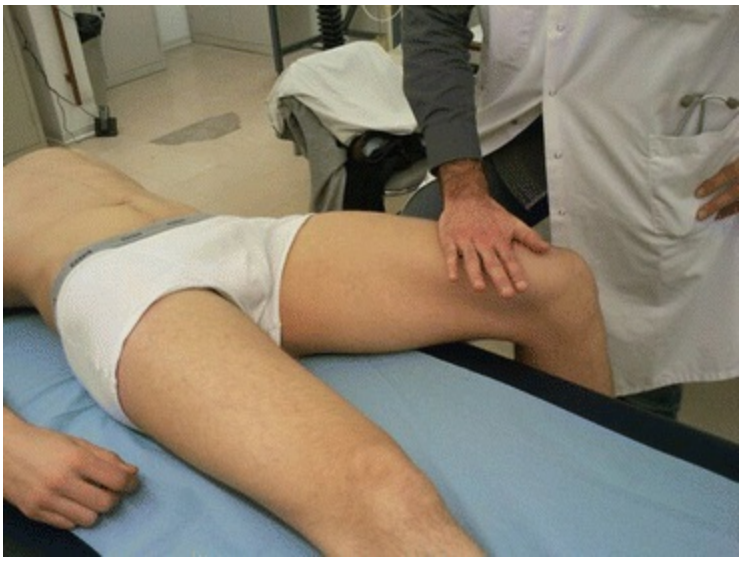

Fig. 15.3 Locating pain by contracting the iliopsoas against resistance

Pain on palpation, which is sharper but far less specific, is present in 89 % of cases. It should be located with the hip semi-flexed, just outside the femoral neurovascular bundle and inside the sartorius muscle.

Similarly, pain should systematically be located by contracting the neighboring

muscles, in particular the external adductor and obturator muscles which may also have an intrinsic lesion. The examination is completed by locating stiffness in the anterior pelvic chains (53 % of cases), a manifestation of athletic pubalgia (16 % of cases) or lumbago (13 % of cases) or other differential diagnoses as shown in Table 15.1 (Sect. 15.7).

Table 15.1 Differential diagnoses

Other lesions of muscles and tendons	Lesions of the sartorius, lesions of the anterior femoral, lesions of the adductors, notably the pectineal
Nearby bone disorders	Stress fracture, avascular necrosis
Hip joint disorders	Early osteoarthritis [8], lesions of the labrum (with or without rupture), anterior conflict, bursitis
Pubalgia- Groin pain	
Osteochondrosis	Lesser trochanter, anterior superior iliac spine
Femoral hernias	
Medical disorders	Hematoma of the psoas (traumatic in hemophiliacs or iatrogenic in VKA overdose), abscess of the psoas, peripheral lymphadenopathies, calcifications
Vascular lesions	Endofibrosis of the external iliac artery
Neurological lesions	Radiculalgia, ductal syndromes affecting the ilio-inguinalis or the femoralis (crural) [9]

15.5.3 Apophysitis and Distal Disinsertion at the Lesser Trochanter

Tendon disinsertion, combined or not with an avulsion fracture of the lesser trochanter, is a rare accident which is most likely to occur to older men. These accidents are sudden, caused when a movement is countered by an opponent or by a sharp, uncontrolled change in foot position. Football (soccer) is the sport that produces it most commonly [15]. Anterior hip pain is immediate and disabling. It leads to retraction. The athlete is incapable of raising the lower limb when lying down. Active hip flexion is impossible, particularly in the sitting position. A full clinical study of the hip is disrupted by the extent of the initial pain and should be resumed after a few days of rest. At this stage the examination may find bruising of the anterior-medial thigh, heading down towards the knee.

Adolescents may present a similar acute picture with apophyseal avulsion or a chronic picture with apophysitis during growth. Boys who do football, athletics and skating seem to be the most affected [16]. Healing occurs by fusion of the secondary ossification centers over 3–4 months with a total break from sport.

15.5.4 “Anterior” Hip Clicking and Snapping

These are lesions that also concern the myotendinous junction region. This symptomatic

clicking corresponds to the sudden movement or even turn [11] of one of the myotendinous parts of the iliopsoas on the iliopectineal eminence when certain movements are made, and is often accompanied by bursitis. These pathologies evoke a conflict which is encouraged by the anatomical context [17]. The patients are young and describe more or less painful snapping during the active switch from flexion to extension of the hip. The symptoms evolve in a fluctuating manner which does not systematically occur at the same time as physical effort. A physical examination should seek to reproduce an audible snapping, with the patient standing during the switch from flexion to extension-abduction-lateral rotation [16]. Passive mobilization of the hip produces no result. Dynamic ultrasound contributes strongly to the diagnosis [11]. Conservative treatment is recommended.

15.6 Further Examinations

15.6.1 Standard X-ray

This examination is usually normal.

In the event of an osseous lesion of the lesser trochanter, an x-ray may show a partial or full avulsion, displaced upwards and inwards to varying degrees. This lesion occurs in adolescents from the age of around 10–12, when secondary ossification centers appear in the pelvis. This avulsion may also be encountered in young adults due to late, variable cementing of this apophysis, sometimes after they are 20 years old [15]. This examination also allows monitoring of evolution, which is usually positive.

In certain patients these x-rays show osseous signs of associated lesions or can guide the physician towards another pathology: stress fracture, pubic symphysis's, anterior hip conflict due to impingement.

15.6.2 CT-Scan

The utility of a CT scan is very limited and its indications are restricted for this pathology.

This examination should mainly be used for a better analysis of the bone and joint morphology at the rear of the iliopsoas and its tendon, and to highlight any osseous origin of a conflict with clicking or snapping [17].

Conflict with: the iliopectineal eminence; the femoral head and the capsule; the crest of the lesser trochanter; exostosis or calcification.

A prior arthrographic study with intra-articular opacification does not appear to provide any further essential information for the diagnosis or the examination.

15.6.3 Ultrasound

This examination makes a significant contribution provided that it is done early, rigorously, comparatively and dynamically. The patient is supine then in lateral position, with a full bladder for a study of the pelvic portion of the muscle. A linear probe is used (10–13 MHz). The pelvic and abdominal portion of the iliopsoas (to the fore of the iliac crest and outside the lumbar rachis) is difficult to analyze due to digestive interpositions. The general echo pattern of the muscle is regular, made up of fine hypoechoic images arranged obliquely to the main axis of the muscle [18, 19].

In their prefemoral trajectory, the myotendinous junction and the distal tendon of the iliopsoas are very hypoechoic. The lesions are highlighted well on comparative horizontal and sagittal planes. There may be a slight peritendinous detachment or a sero-hematic hypoechoic collection located on the deep aspect. The size of the lesion contributes to its classification. When the examination is conducted late, it may show fine calcifications, sources of shadow cones in the same zone.

In the event of distal damage there is a hypoechoic thickening of the tendon with small calcified spicules from the bone surface. These signs of enthesopathy are accompanied by hyper-vascularization clearly evidenced in a Doppler examination.

This examination also serves to locate an iliopsoas bursopathy, the origin of which, tendinous or articular, remains to be clarified. The impact of the collection on the adjacent structures, particularly the tendon of the iliopsoas, is easy to analyze, as is its content which is most often anechoic, or heterogeneous in the event of bleeding.

The examination is complemented by a dynamic study [11] with muscle contraction by elevation of the lower limb, and with rotation in various hip movements. This study highlights a clicking of the tendon of the iliopsoas, most notably in horizontal cross-sections. There is a sharp movement of the tendon when it moves over the iliopectineal eminence (when the hip switches from flexion to active extension) with a dull, sometimes painful snapping. This dynamic study is also useful in assessing the relationships between the tendon and iliopsoas bursitis.

15.6.4 Magnetic Resonance Imaging

This is the benchmark examination because it directly shows the muscles and tendons, allowing the lesions to be broken down precisely: topography of the area in the three planes, type and severity. It offers semiological arguments in tissue characterization thanks to the use of various sequences with T1, T2 and PD weighting (proton density) and the fat signal suppression technique. The T1 sequences give anatomical details while the T2 sequences are more semiological, highlighting pathological zones in hypersignal.

MRI will also detect any underlying bone or joint damage, a lesion of the soft parts around the hip, and fluid collections. This examination is thus immediately useful in the event of any diagnostic doubts or ultrasound/clinical disagreement, as well as for an

exploration of chronic lesions.

In the T1-weighted sequences the normal muscle presents homogenous signal intensity. The inferior part of the muscle has a weaker signal as the tendon fibers gradually replace the muscle fibers, and the tendon presents a hyposignal. The insertion zone is harder to analyze because the iliopsoas tendon and the cortical bone of the lesser trochanter present the same hyposignal. In T2 with fat-signal saturation, the hyposignal from the muscle increases and the anatomical aspect is less easy to analyze (Fig. 15.4). The hyposignal in Tl from stagnant liquids (effusion, edemas, blood…) becomes a hypersignal in T2 [19, 20].

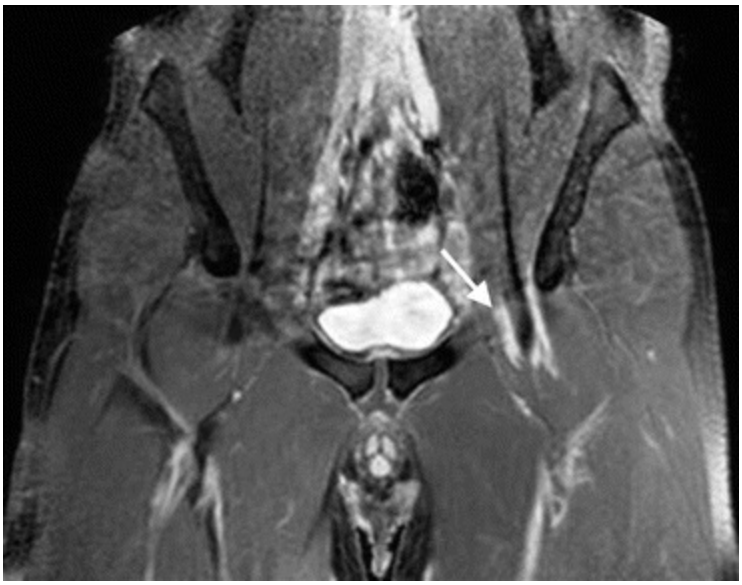

Fig. 15.4 Coronal contrast-enhanced T1-weighted SPIR pelvic MRI shows hypersignal at the myotendinous junction of the left iliopsoas (*arrow*)

If the three planes in T2 with fat-signal saturation are combined to perform a complete examination, the comparative horizontal planes (Fig. 15.5) are indispensable to locate the lesion precisely. At a minimum they are combined with sagittal planes to assess the extent in height. An analysis of the morphological changes provides clarifications regarding the nature and location of the lesion. The hypersignals that are visible in the T2 sequences with fat-signal suppression should be differentiated according to whether they represent an edema, a layer of fluid or a sero-hematic collection (hypersignal from the myotendinous junction, which may be accompanied by an intra-muscular edema and a sub-aponeurotic fluid line, for example). The point of the comparative study is to highlight small lesions (irregular, scalloped appearance of the deep aspect of the myotendinous junction by the bony margin, most notably). Chronic lesions are studied in the T2 sequences with fat-signal suppression, and particularly in the Tl sequences with fat-signal suppression and intravenous injection of gadolinium.

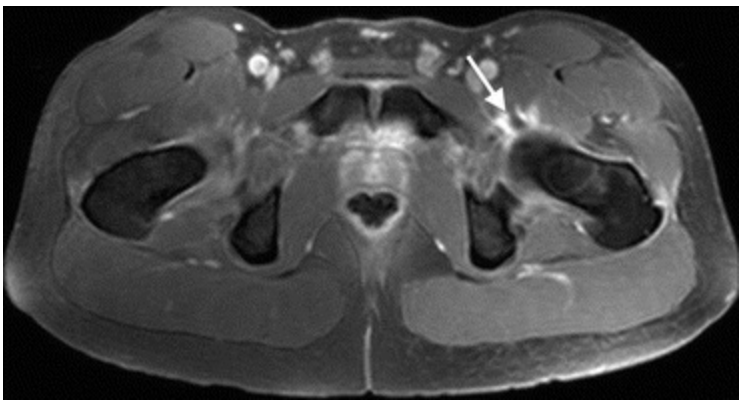

Fig. 15.5 Axial contrast-enhanced T1-weighted SPIR pelvic MRI shows hypersignal at the myotendinous junction of the iliopsoas (*arrow*)

As is often the case with chronic muscle lesions, anomalies are difficult to detect and are sometimes shown only by the presence of a fibrous scar in T1 and T2 hyposignal. There is no hypersignal if there is no associated edematous or inflammatory phenomenon. Its presence confirms the topographic diagnosis and conditions local manipulation.

In certain incidences of clicking to the fore of the hip, an MRI scan may reveal discrete anomalies in the posterior face of the terminal part of the iliopsoas and the tendon. Sagittal views (Fig. 15.6) [21] show a fibrous thickening, small dot-shaped hypersignal ranges with T2 weighting, and a fluid section, also in hypersignal from the bursa [22, 23]. Tomography views can highlight a protrusion of the iliopectineal eminence [17].

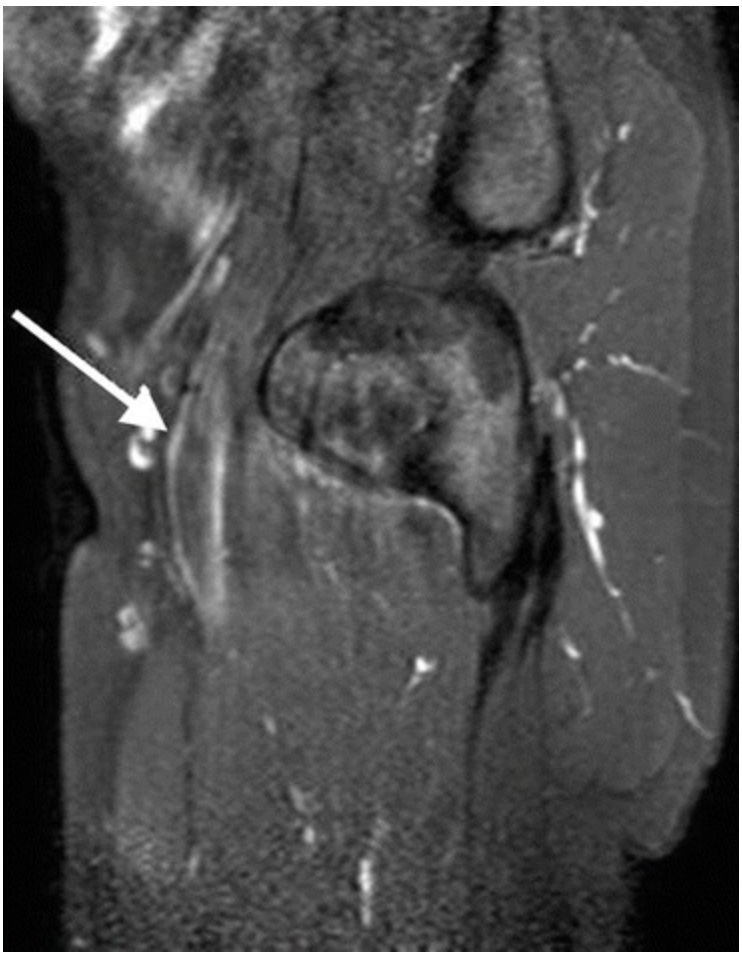

Fig. 15.6 Sagittal contrast-enhanced T1-weighted SPIR pelvic MRI shows anterior hypersignal at the myotendinous junction of the iliopsoas (*arrow*)

15.7 Differential Diagnoses

The differential diagnoses of the iliopsoas lesions and, more broadly, the etiologies of anterior hip pains, are listed in Table 15.1.

15.8 Treatment

If the diagnosis *is performed early*, the lesion is classified after the results of the clinical and sonographic examinations according to the Durey and Rodineau criteria [24].

For *lesions presenting low or medium severity*, the first phase of treatment attempts to limit the extent of the lesion and the hematoma, a source of fibrous scarring and chronic evolution. Selective rest should be imposed, allowing the patient's general physical condition to be maintained and the other weak points to be worked on by strengthening the upper limbs or swimming with floaters. The healing mechanism and

re-education start as soon as the clinical tests are painless. Thereafter, an active strengthening program lasting 4 weeks may be carried out. The first 2 weeks involve concentric contraction work, and the last 2 weeks eccentric (abduction and adduction) and proprioceptive contraction. These exercises stabilize the hip and maintain a good flexor/extensor balance. [25] They should be coupled with stretching of the pelvic chains to maintain the suppleness of the psoas. An analysis of the wounding movement serves as a guide to re-adaptation and is used to organize the patient's return to the sports field and prevent a repeat injury. Healing takes 2–6 weeks.

For high lesions of the iliopsoas, close clinical and paraclinical monitoring is implemented. The occurrence of a *compressive hematoma of the psoas* requires a check on the coagulation function and a surgical opinion.

Low lesions are tendon disinsertions. Orthopedic and functional care is sufficient in most cases for a good result. The extent of retraction may lead to a surgical opinion. *In avulsions of the lesser trochanter*, a significant displacement may require surgery with the fragment screwed in. In other cases, functional treatment after 3 weeks of rest lying down gives good results, even when a detached fragment persists [26]. Full resumption of sport can be discussed from the third month.

When *treatment is late,* the way complications are treated conditions the prognosis. If the lesion of the junction is calcified, an indomethacin treatment is offered by some physicians and, above all, clinical and x-ray monitoring is required. It is usually a fibrous scar which hinders the biomechanics of the myotendinous junction. First-line treatment involves analytical and then global functional re-education, associated with selective rest. Muscle balance should be studied carefully. Some physicians recommend strengthening the hip rotators [23, 27].

Infiltration of the fibrous zone may be necessary when competitive movement is resumed, usually with good results. Bearing in mind the complex anatomical environment of this region, this treatment is only indicated if the other treatments fail, after first confirming the diagnosis via imaging and pinpointing the injection point by means of a sonograph.

Secondary prevention is systematic, aiming to detect movement errors and intrinsic factors. With chronic forms the time required for a return to the same level of physical activity is three to 12 months (5 months on average), emphasizing the problems of late treatment and the importance of early diagnosis.

For certain conflicts between the deep aspect of the iliopsoas and the hip joint, the treatment may be arthroscopic, but this is rarely necessary.

15.9 Conclusion

Traumatic pathologies of the iliopsoas are rare, little known and little documented. Two modes of occurrence are classically described. The lesion may be caused by an

accident, with the lesion in the middle of the muscle body, of the muscle-tendon junction or a low rupture, or by a low-intensity trauma with a small lesion, usually located at the myotendinous junction. These traumatic lesions of the iliopsoas are mainly located at three very different topographic levels. At the top there are lesions of the fleshy body of the muscle and the main complication is the formation of a compressive hematoma. The second level corresponds to the myotendinous junction of the iliopsoas. This histological transition zone is also a region of anatomical transition. As the muscle moves from the pelvis to the thigh it is reflected on a bone depression located between the anterior superior iliac spine and the iliopectineal eminence. Lastly, the third lesional level is located by the lower insertion into the lesser trochanter (tendon, bone and peritendinous space).

Diagnosis should be performed early in order to avoid the all too common path towards chronic symptomatology. It may be complicated by the rarity of these disorders, by the complex anatomical environment and by the falsely benign evolution of the initial picture. The first clinical examination should strive to locate pain on palpation and flexion against resistance of the hip.

Analysis of the imaging results becomes more difficult the later it occurs. Among athletes, in an intrinsic traumatic context this diagnosis should be evoked in case of anterior hip pain. When the standard x-ray result is normal, the diagnosis should be carried out by ultrasound or MRI, clarifying the location and nature of the lesion, its size, and even the extent of retraction in the event of rupture. A large compressive hematoma indicates evacuation-puncture under imaging control.

The athlete's profile analysis and the imaging will determine the choice of treatment. Lastly, stiffness of the anterior pelvic muscle chains appears to be a factor favoring this type of lesion. We recommend preventive work involving active and passive stretching.

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16. Pathology of the Rectus Femoris

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Abstract
The quadriceps is the most commonly injured muscle in sports that require sprinting or kicking, such as soccer. The rectus femoris (RF) is the most commonly injured quadriceps muscle especially in young athletic individuals. Imaging is an important adjunct to clinical examination for management of RF injuries in athletes. The aim of this chapter is to summarize the current state of knowledge of sport related injuries of the RF. After a comprehensive review of anatomy and the mechanisms of injury, we will focus on the imaging findings from proximal attachment injuries of the RF, while emphasizing the most relevant points for making the prognosis.

16.1 Introduction

The quadriceps is the most commonly injured muscle in sports that require sprinting or kicking, such as soccer. Lesions of the quadriceps represent 32 % of muscle strains in a 5-year study of European soccer players [1]. The rectus femoris (RF) is the most commonly injured quadriceps muscle, representing up to 68 % of quadriceps injuries [2, 3] usually in young athletic individuals [4]. Imaging is an important adjunct to clinical examination, and although a number of papers have been dedicated to RF injuries, few of them correlated radiological with clinical results [3, 5]. The aim of this chapter is to summarize the current state of knowledge of sport related injuries of the

RF. After a comprehensive review of anatomy and the mechanisms of injury, we will focus on the imaging findings from RF proximal attachment injuries, while emphasizing the most relevant points for making the prognosis.

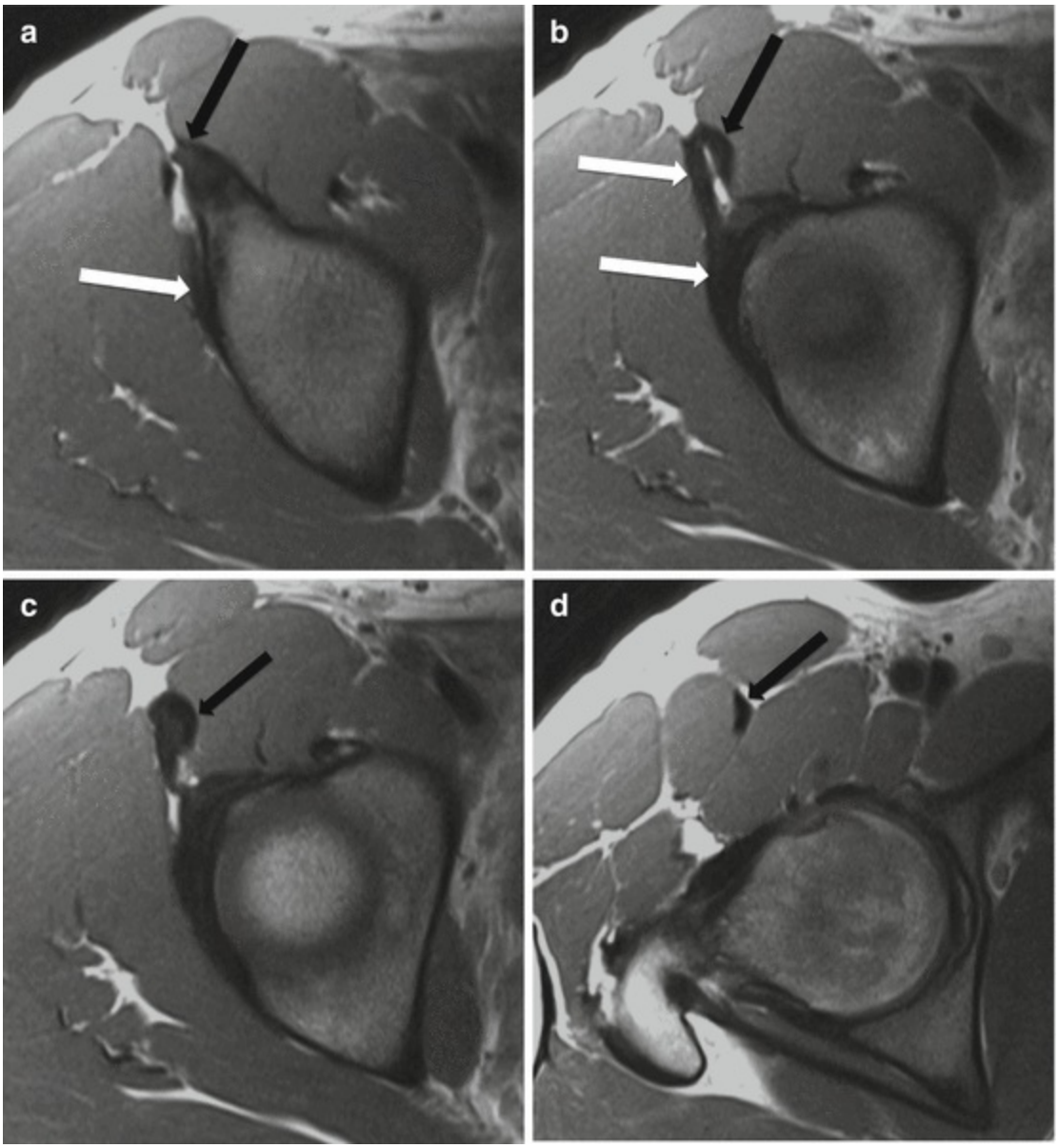
16.2 Anatomy and Normal Radiological Appearance

The RF muscle is a long and fusiform muscle that forms the anterior superficial portion of the quadriceps muscle group. A detailed description of its normal anatomy, especially of the deep tendon, was provided only in 1995 by Hasselman et al. [6] They reported that the direct head arises from the anterior inferior iliac spine (AIIS), while the more posterior and inferior indirect head originates from the superior acetabular ridge and hip capsule. Only a few centimeters below their origin, the two heads form a conjoint tendon. The superficial conjoint tendon, made mostly of fibers of the direct head, then blends with the anterior fascia of the muscle; the posterior conjoint tendon, made mostly of fibers of the indirect head, which is initially rounded, lies in the most medial aspect of the muscle. As it progresses distally the indirect head flattens out laterally, rotates and migrates to the middle of the muscle belly forming a deep myotendinous junction. By its end in the distal third of the muscle, the deep tendon—also referred to as the "deep tendon of the indirect head"—is flat and twisted 90° to a nearly vertical position. In their original description, Hasselman et al. described the presence of the deep tendon—and the muscle fibers attached to it—as a "small muscle within the RF muscle" [6] (Fig. 16.1).

Fig. 16.1 Muscle within muscle appearance of the rectus femoris muscle. The indirect head inserts on the anterior inferior iliac spine (*black arrow*). The muscle fibers arising from the deep tendon of the indirect head (*black arrowheads*) form a small muscle within the rectus femoris. The *grey arrow* points to the direct head

On axial magnetic resonance imaging (MRI) both heads appear as linear low-signal structures arising from their respective bony insertions. The deep tendon transfers from a globular structure at its proximal part to a boomerang-like structure located anteriorly and medially to the muscle fibers. The anterior component of the conjoint tendon (superficial tendon) blends more distally with the anterior fascia of the RF muscle. The more posterior portion of the conjoined tendon (deep tendon) gradually becomes embedded within the muscle belly of the RF muscle, forming a deep tendon with a long intrasubstance muscle tendon junction (Fig. 16.2). On coronal images the two origins of

the RF tendon are also clearly seen. Anteriorly the origin of the direct head from the AIIS is visualized as a relatively thick, somewhat rectangular low-signal structure. Slightly more posteriorly, the indirect head appears—in very close proximity to the joint capsule—as a linear low-signal structure highlighted by fat and thinner than the direct head [7].



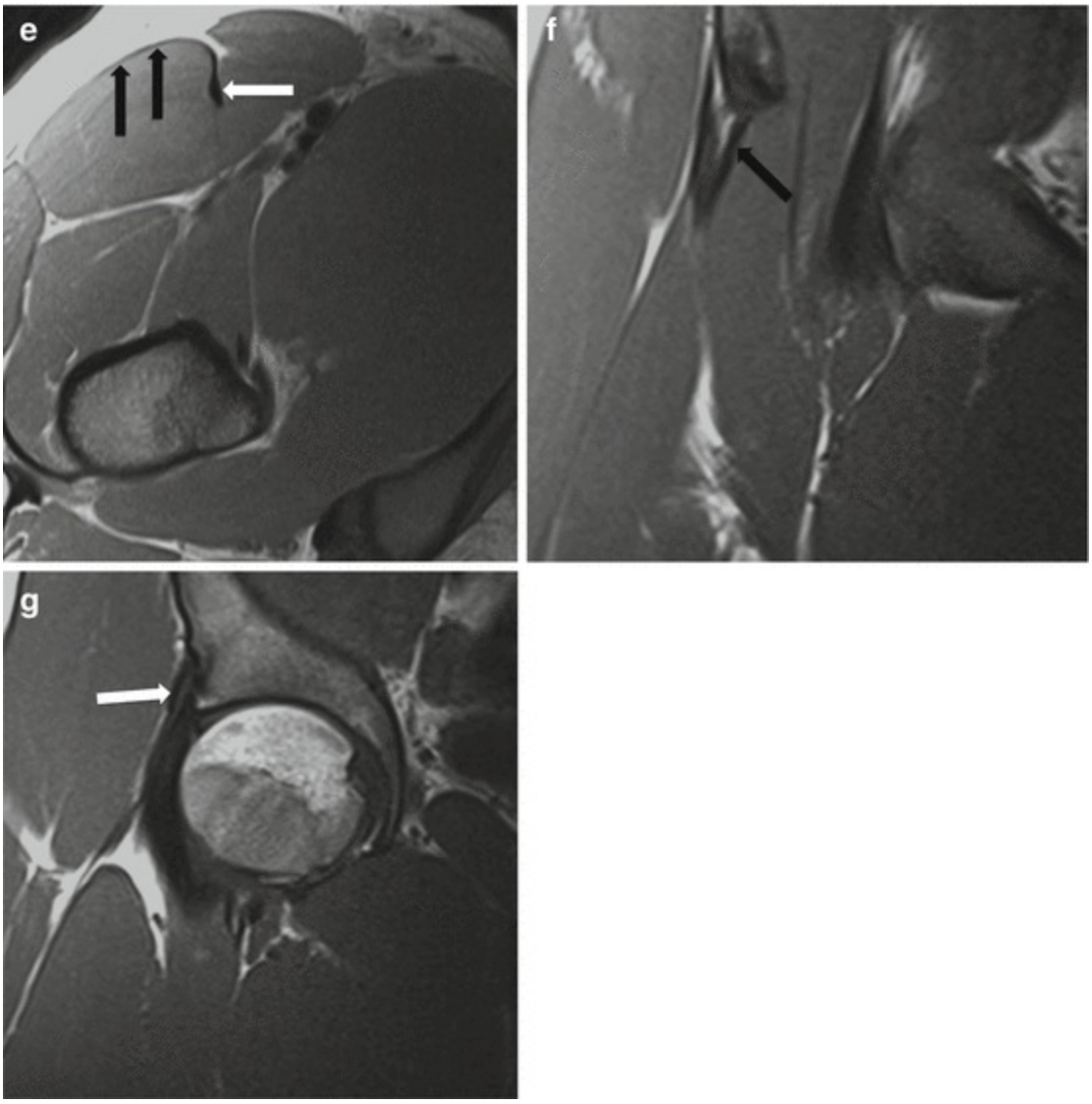


Fig. 16.2 A healthy 15-year-old male athlete with normal MRI anatomy of the proximal right rectus femoris. (**a**–**e**) Axial T1-weighted MRI. (**a**) The direct head inserts at the anterior inferior iliac spine (*black arrow*) while the indirect head is slightly more posterior inserting at the superior ridge at the anterolateral aspect of the acetabulum (*white arrow*). (**b**) The indirect tendon is seen in its full horizontal oblique course (*white arrows*) and is immediately posterior to the direct tendon (**c**) Slightly caudally both tendons merge into the conjoint tendon (*arrow*). (**d**) The rounded tendon progressively flattens as it travels distally, lying in the most medial aspect of the muscle (*arrow*). (**e**) While progressing along the muscle, fibers from the direct tendon form the superficial tendon (*black arrows*) and those continuing the indirect tendon migrate to a sagittal position (*white arrow*). (**f**, **g**) Coronal T1-weighted MRI show the insertion of the direct head off the anterior inferior iliac spine (*black arrow*) and the attachment of the indirect head to the superior acetabular ridge (*white arrow*)

On ultrasound, the insertion of the direct tendon is easily detected in axial scans from the easily-palpated AIIS downward. The indirect head follows an oblique course and is examined with an oblique axial scan at the lateral aspect of the uppermost portion of the thigh. More distal scans reveals the particular internal architecture of the RF with the deep aponeurosis represented by a mildly curved, comma-shaped hyper echoic structure—normally quite distinct from the muscle tissue—extending as far as the lower

third of the muscle. The superficial tendon, however, is visualized as a thickening of the muscle fascia [8].

16.3 Mechanisms of Injury

In general there are three main etiopathogenic mechanisms of muscle injury: (1) direct trauma—resulting in contusion; (2) indirect trauma—resulting in strains; and (3) lacerations. The latter mechanism is uncommon in sport related lesions, and is excluded from this chapter. The quadriceps muscle is subject to both direct and indirect energy trauma. It has, however, been shown that the mechanism of injury usually depends on which muscle within the quadriceps is involved: vastus medialis and lateralis muscles are more often exposed to direct trauma while lesions of the RF usually result from indirect energy trauma. Indirect trauma to the RF causes the muscle to rupture at its weakest point, which is the myotendinous junction in adults (strains) and the apophyseal growth cartilage in children [8, 9].

The RF has all the characteristics of frequently injured muscles. It is biarticular, spanning the hip and knee and thus responsible for decelerating both; it has a high proportion of type II muscle fibers which generate more tension on contraction; and it acts in an eccentric manner [6, 10–12]. The incriminating role of kicking has been well described [13]. In the final portion of the backswing phase, the hip is hyperextended and the knee is flexed, stretching the biarticular RF muscle and putting it in passive insufficiency, its weakest position, especially at the proximal third. At the onset of the forward swing there is massive eccentric muscular recruitment of the quadriceps and iliopsoas muscles. Forceful muscular contraction in a stretched quadriceps muscle can lead to sprain [6, 9, 13]. This is what typically happens when, for example, soccer or rugby players unexpectedly encounter irregular or slippery turf as they are about to kick the ball, and they try to compensate by extending the hip [8, 14]. It also occurs when one loses one's footing during abrupt deceleration, an event that is common in all sports that involve running.

16.4 Risk Factors

The causes of muscle strain injury are multifactorial. Past muscle strain injury is perhaps the most recognized risk factor [15, 16]. Other proposed risk factors include low muscle strength, muscle fatigue, age, lack of warm-up, muscle temperature, and poor flexibility [15, 17]. For quadriceps muscle strains in Australian Rules footballers, Orchard found that both recent (less than 8 weeks) and remote quadriceps strain injury, recent hamstring strain, the dominant kicking leg, short stature, and ground hardness were all associated with increased risk [16]. Orchard described these clinical strains over a 7-year period in the national competition; the injuries were not routinely

assessed by MRI, and therefore we do not know what patterns might have been revealed.

16.5 Imaging Techniques

Cross sectional imaging, MRI and ultrasound are commonly indicated for professional and/or elite amateur athletes when both the athlete and others (coach, trainer, manager) need accurate diagnosis and prognosis.

16.5.1 Conventional Radiographs

Conventional x-rays are helpful in detecting osseous fracture in cases of avulsion fracture. Anteroposterior x-rays of the pelvis can be helpful in this case, as can the oblique alar view. X-rays also show mineralization and ossification in chronic lesions of the RF [8].

16.5.2 Ultrasound

The examination is best done with a multifrequency (5–12 MHz) linear transducer. If there is substantial muscle hypertrophy a 5-MHz transducer is preferable as it offers better visualization of the deep planes. A systematic approach will allow complete exploration of the RF and reveal even small lesions that can be easily missed. Axial scans from the AIIS downward are performed first as they offer a panoramic view. They are followed by oblique coronal views at the proximal insertion. Information obtained during the static examination can be supplemented with a dynamic examination performed during isometric contraction. This approach is sometimes more suitable for detecting small partial tears. When the muscle plane has been fully explored, the distal tendon is scanned. During this phase, the knee is flexed approximately 30° to straighten the tendon and to eliminate anisotropy artifacts which result in hypoechoic areas that can be mistaken for focal tendinopathy or even partial ruptures [8].

16.5.3 MRI

Although each patient is unique, certain generalizations are helpful in designing an appropriate MRI protocol. At an absolute minimum, each examination should include at least two orthogonal planes and two different pulse sequences. In addition to the requisite axial plane the second long-axis plane can be either coronal or sagittal. In addition the examination should include a combination of T1-weighted—for high resolution and “anatomical images”—and fat-suppressed fluid-weighted images to detect pathologic changes [7, 18].

Gradient-echo sequences help in detecting the presence of hemosiderin by

accentuating certain paramagnetic effects. The administration of gadolinium based contrast material is generally not necessary. Occasionally intravenous gadolinium administration can be helpful, particularly when a clinically suspected muscle injury is not visualized on T2-weighted and inversion-recovery fast spin-echo images. Torn muscle fibers may be more conspicuous after gadolinium administration, particularly when there is extensive hemorrhage and edema. Several cases have been reported in which professional athletes had muscle strains that were not diagnosed on T2-weighted and inversion-recovery fast spin-echo images, but were visualized on contrast-enhanced T1-weighted images [18–20].

16.6 Avulsion Fractures at Tendon Insertions

Avulsion fractures can involve any of the three osseous insertions of the RF: the AIIS for the direct head, the superior lateral acetabular ridge for the indirect head, and the patellar sleeve for the distal insertion. Unlike strains, which most commonly involve the indirect head, avulsion fractures usually involve the AIIS, the site of insertion of the direct head [9, 21–23]. This type of fracture is also called “sprinter’s fracture” [24]. In children the presence of growth cartilage at the tendon insertion makes the bone-tendon junction more vulnerable to indirect energy trauma than the myotendinous junction. The patient reports a sudden pain and eventually feeling a break when kicking the ball. Pain is usually worst at the site of fracture, which is frequently at the AIIS, or the lateral upper thigh when the indirect head is involved.

Avulsion fractures are readily detected on conventional radiographs. Anterior-posterior x-rays of the pelvis, as well as the oblique alar view, will clearly reveal the avulsion of the cartilage (Fig. 16.3). The degree of retraction and the size of the bone fragment can be evaluated. X-rays taken in athletes years after avulsion fractures show hypertrophy of the AIIS, which appears as a large ossification that projects into the inferior soft tissue. Ultrasound confirms the diagnosis. The avulsed fragment is seen as a hyperechoic structure of variable size lying at some distance from the AIIS [8]. On MRI avulsion fractures appear as hypointense linear abnormalities on T1-weighted imaging of bone underlying the origin of the RF. The osseous fragment and donor site are also evident, as is any hematoma interposed between the avulsed fragment and adjacent bone. The signal intensity of the hematoma depends on the age of the injury, although in the acute phase the hematoma is likely to show decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted images [9, 25] (Fig. 16.4). Treatment is usually conservative (short period of bed rest followed by progressive weight bearing with crutches) with rapid resolution of pain and return to playing condition in a relatively short period (6 weeks) [26]. Surgery is indicated for long standing symptomatic proximal avulsion after failure of nonoperative treatment [27, 28].

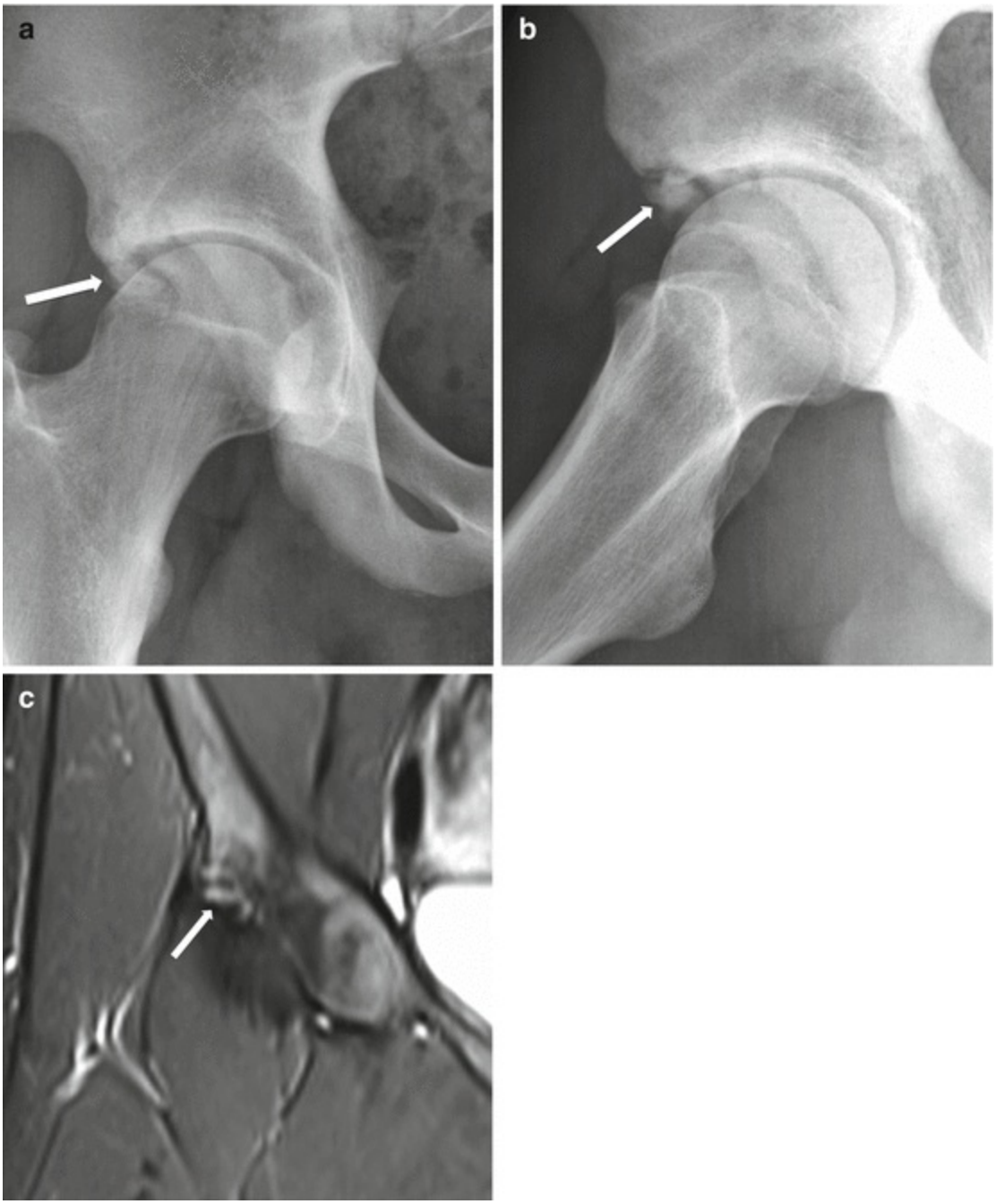


Fig. 16.3 A 16-year-old female athlete with an old fracture avulsion of the right superior acetabular ridge at the insertion of the indirect head of rectus femoris. (**a**) Anteroposterior and (**b**) frog leg lateral x-rays of the right hip show an osseous fragment detached from the superior acetabulum (*arrow*). (**c**) Coronal fat-suppressed T2-weighted MRI displays fracture of the superior acetabulum at the insertion of the indirect head (*arrow*)

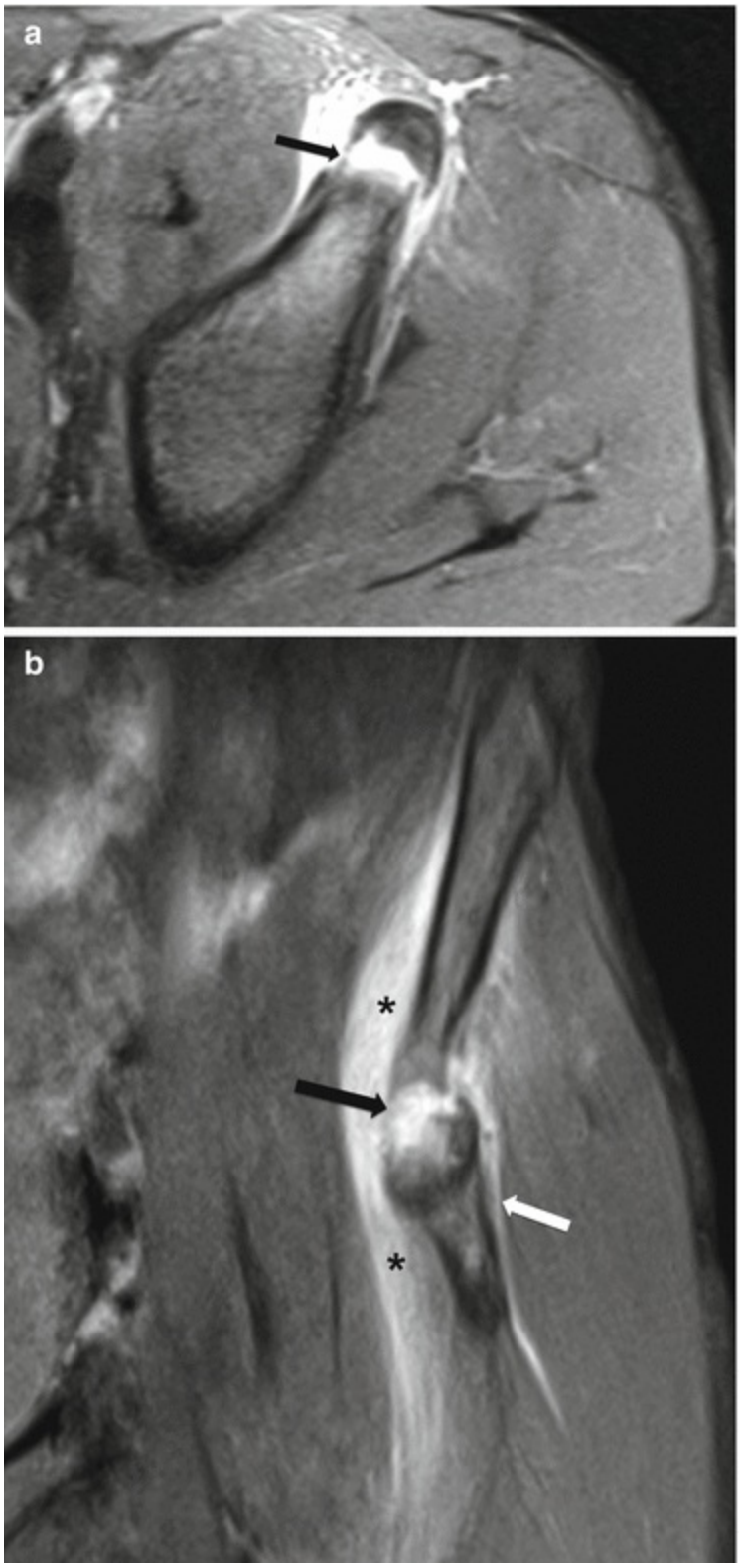


Fig. 16.4 A 14-year-old male athlete with an acute avulsion fracture of the anterior inferior iliac spine. (**a**) Axial and (**b**) coronal fat-suppressed T2-weighted MRI show fracture of the anterior inferior iliac spine (*black arrow*), attachment site of the direct tendon of rectus femoris (*white arrow*). Note the presence of feathery edema of the iliacus muscle (*asterisks*)

16.7 Myotendinous Injuries of the RF

The clinical diagnosis of myotendinous injuries is usually based on a three-point scale: (1) for mild; (2) for partial; and (3) for a complete tear [29]. Mild injuries have no discernible loss of strength or motion restriction. Partial tears demonstrate some loss of strength or motion that is not complete, unlike type 3 injuries [2]. Strain injury is associated with inflammation, edema, and sometimes hemorrhage with proliferation of inflammatory cells and fibroblastic activity in the first 24–48 h [2]. Histological animal models of muscle stretch injury have shown that myotendinous injury results in inflammation, bleeding and muscle fiber necrosis initially. This destructive phase is followed by a concomitant repair and remodeling phase involving recruitment of progenitor cells, scar formation, and remodeling of organized tissue [30].

Clinically, the patient may present with sharp pain associated with movement and impaired mobility. The injured area can be located with precision by the patient and verified by a careful examination showing maximal tenderness over the midline of the thigh [3]. Injuries of the origin of the reflected tendon may mimic hip pain or a lesion of the tensor fascia lata. The patient reports a sensation that something in the hip was displaced during the trauma [8]. When the lesion involves disruption of the distal muscle fibers from the posterior tendon of insertion, however, proximal retraction of the entire muscle belly is observed, resulting in a mass that migrates proximally to the groin with muscle activation. This mass is sometimes mistaken for a soft-tissue neoplasm [6, 25, 31]. Other clinical findings include localized swelling, loss of knee extension, thigh asymmetry and a palpable defect with a retracted mass (in complete ruptures). Prompt diagnosis (within a few days of the muscle trauma) is essential to ensure timely and complete healing and to reduce the likelihood of recurrence. Most important at the time of diagnosis is to differentiate benign injuries from serious injuries that may require protracted rehabilitation. Unfortunately, making the distinction is difficult by clinical examination alone, especially in the first week after injury. Imaging studies however show a significant relationship between initial findings and prognosis [3].

16.8 Imaging Findings

RF injuries are commonly classified on MRI as:

- *Grade 1:* Bright signal on fluid-sensitive sequences representing fluid and hemorrhage around the myotendinous junction extending into the adjacent muscle, creating a feathery appearance.
- *Grade 2:* More severe and may show a thin or irregular appearance of the myotendinous junction itself along with edema and hemorrhage (increased T2 signal intensity) that often tracks along the fascial plane.

- *Grade 3:* Complete disruption and discontinuity of muscle typically at the myotendinous junction with complete replacement of organized collagen by fluid signal on fluid sensitive sequences. There is often an associated wavy tendon morphology and retraction of the muscle. Surrounding edema or hemorrhage is usually extensive.

There are also ultrasound classifications for RF injuries [8, 32]. The following were reported by Petroons et al. and modified by Balius et al. [5, 32]:

- *Grade 1:* Ill-defined hyper or hypoechoic area without objective fibrillar discontinuity or inflammation of the fascia.
- *Grade 2:* Lesions represent a partial fibrillar discontinuity.
- *Grade 3:* Complete discontinuity of the fibrillar structure.

Although these—or similar—classifications are often reported in the radiological literature their relevance and clinical and prognostic value are not always clear. Grade 3 usually portends a longer rehabilitation time [33], and one study showed that ultrasound grade 2 correlated with longer rehabilitation times than grade 1 [5]. To the best of our knowledge there is no report correlating MRI classification of RF injuries with prognosis. Moreover these classifications are usually not validated by reliability and reproducibility studies [7, 9, 18, 34–37], and we suspect that their usefulness is limited.

Ouellette et al. reported that most RF injuries involve the reflected head (94 % of the cases) with the direct head and conjoint tendon less likely to be injured [9]. However Cross et al. noted equal involvement of both reflected and direct head in Australian Rules Football players. Injuries to the origin of the RF appear as increased signal intensity within and surrounding the tendon insertion. Surrounding fluid may be present. Discontinuity of the tendon indicates rupture, which may be partial or complete (Figs. 16.5, 16.6, and 16.7). It is important to note that when the injury does not show any sign of rupture (no fluid signal intensity within the tendon, no discontinuity of fibers) the lesion is sometimes referred to as a strain as opposed to a partial or complete tear. Most RF origin lesions are partial tears [9]. In addition, because the indirect head inserts on to the supra acetabular ridge and joint capsule, MR arthrography can play a role in detecting reflected head origin by detecting leakage of intraarticular contrast material into the periarticular soft tissues. Similarly, associated labral tears are also easily identified by MR arthrography [9]. Chronic lesions show thickening, redundancy and wavy contours of the injured tendon. Mineralization or heterotopic ossification can also be seen. Both are easily detected on x-rays and appear as globular low-signal intensity areas on both T1- and T2-weighted images surrounding the tendon extremity. Injuries to the RF insertions are usually treated non-operatively. Surgery may be indicated in case of heterotopic ossification with limitation of movement [38].

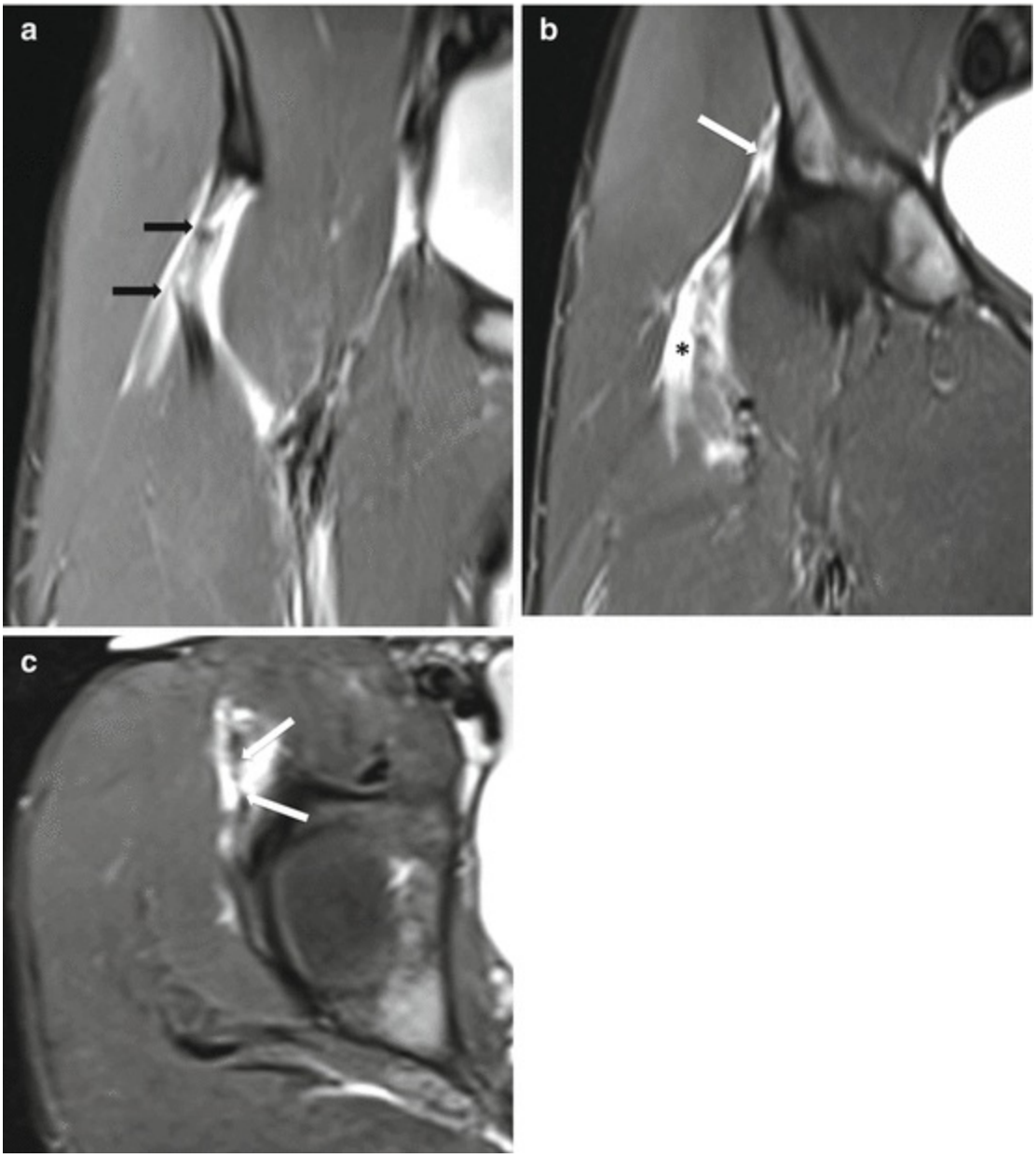


Fig. 16.5 A 19-year-old male athlete with right partial tear of both direct and indirect heads of rectus femoris insertions. (**a**, **b**) Coronal fat-suppressed T2-weighted MRI show partial thickness discontinuity of the direct head close to the anterior inferior iliac spine (*black arrows*) and the indirect head at its attachment to superior acetabular ridge and the hip joint capsule (*white arrow*). Fluid collection surrounding the direct head is noted (*asterisk*). (**c**) Axial fat-suppressed T2-weighted MRI displays partial thickness tear of the indirect head (*arrows*)

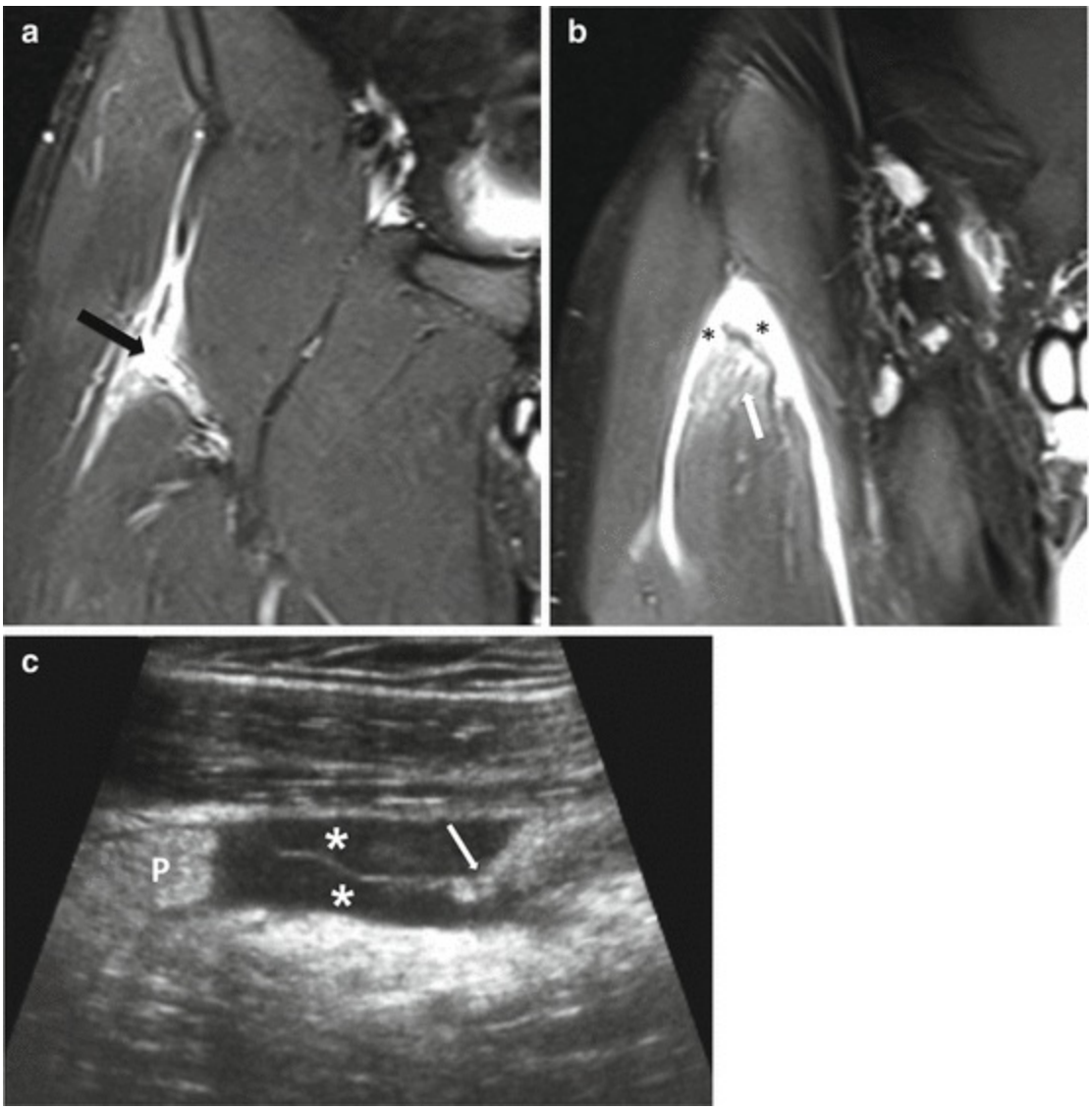


Fig. 16.6 A 31-year-old soccer player with complete rupture of the conjoint tendon of the right rectus femoris. (**a**, **b**) Coronal fat-suppressed T2-weighted MRI. (**a**) Full thickness discontinuity of the conjoint tendon of the rectus femoris (*arrow*). (**b**) Feathery edema within the upper pole of the rectus femoris in keeping with interstitial edema and hemorrhage (*arrow*). Fluid collection surrounding the upper pole of the rectus femoris is noted (*asterisks*). (**c**) Sagittal ultrasound image shows complete tear of the proximal tendon (*P*). The distal stump is pointed with a *white arrow*. The fluid collection appears anechoic (*asterisks*)

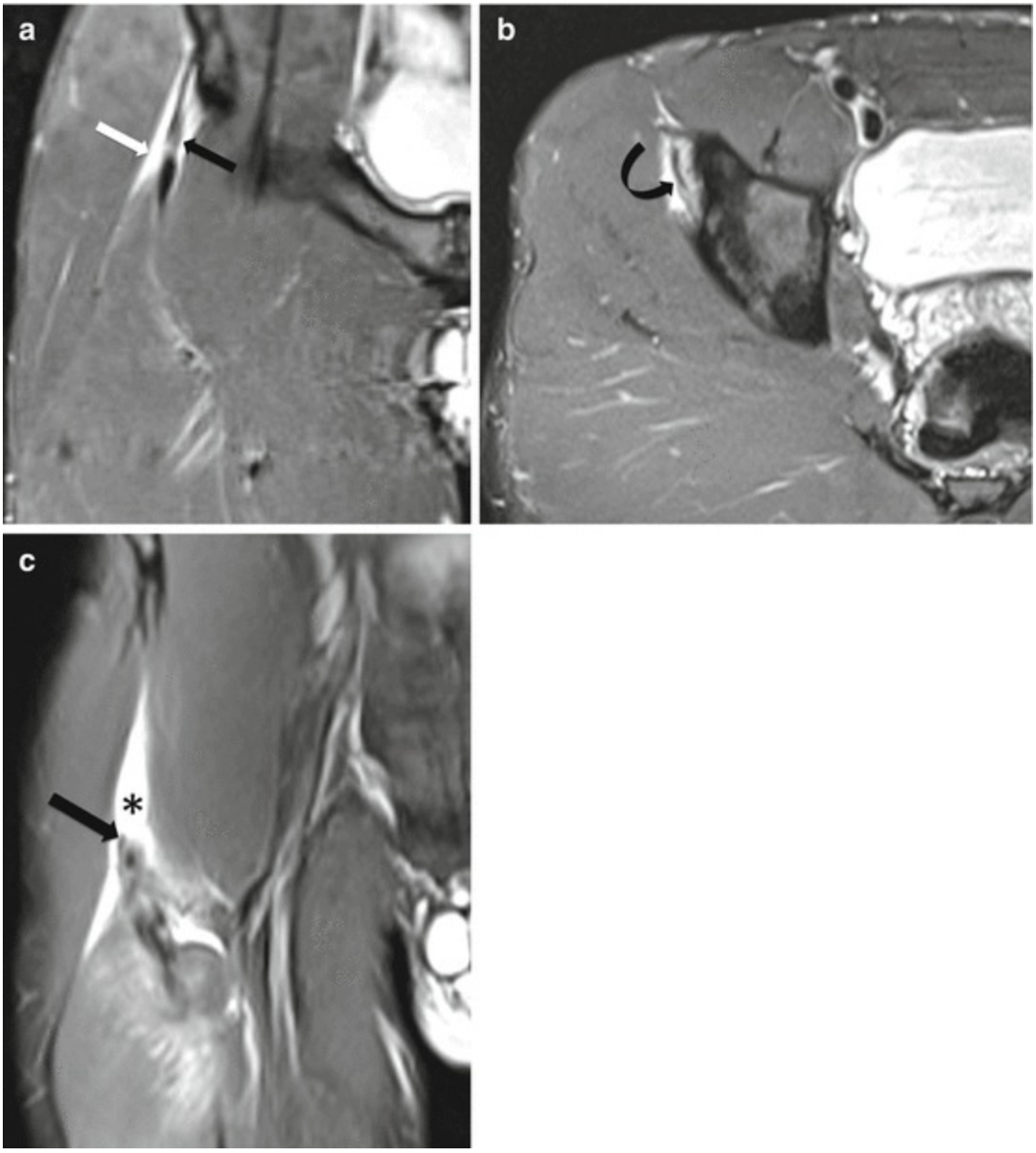


Fig. 16.7 A 19-year-old male athlete presenting with groin pain after forceful soccer ball kicking. (**a**) Coronal and (**b**) axial fat-suppressed T2-weighted MRI show partial tear of both direct (*black straight arrow*) and indirect (*black curved arrow*) heads of rectus femoris. Moderate fluid collection is noted around the proximal tendon insertion indicating an acute lesion (*white straight arrow*). (**c**) Coronal fat-suppressed T2-weighted MRI 11 months later shows complete tear of the proximal attachment of the rectus femoris with a 4 cm retraction of the distal stump (*arrow*). An increase of fluid collection is also noted (*asterisk*)

Injuries to the deep and superficial myotendinous junction most commonly show a pattern referred to as a "bull's eye" consisting of bright signal surrounding the low-signal deep tendon on multiple consecutive axial T1 or fluid-sensitive images (or both) and after administration of intravenous gadolinium (Fig. 16.8) [7]. The presence of fluid and hemorrhage is consistent with an acute lesion (Figs. 16.5, 16.6, and 16.7). Gradient-echo images may demonstrate susceptibility artifacts in the area of hemorrhage. Ultrasound findings can be transposed to MRI findings. The bull's eye aspect consists of

a hypoechoic area encircling the central aponeurosis and surrounded by a hyperechoic circular zone, probably representing edema and hemorrhage (Fig. 16.9). The central aponeurosis can be partially masked by hyperechoic blood which makes it barely detectable [39]. The muscle may also appear swollen, showing a globular appearance with a larger central area of both hypoechoic and hyperechoic zones. Color Doppler sonograms may show peripheral flow signal related to the surrounding hyperemia. More severe cases display complete disruption of the insertion of the muscle fibers in the central aponeurosis. The latter appears then as a linear hyperechoic continuous structure crossing an anechoic area interposed between retracted muscle ends.

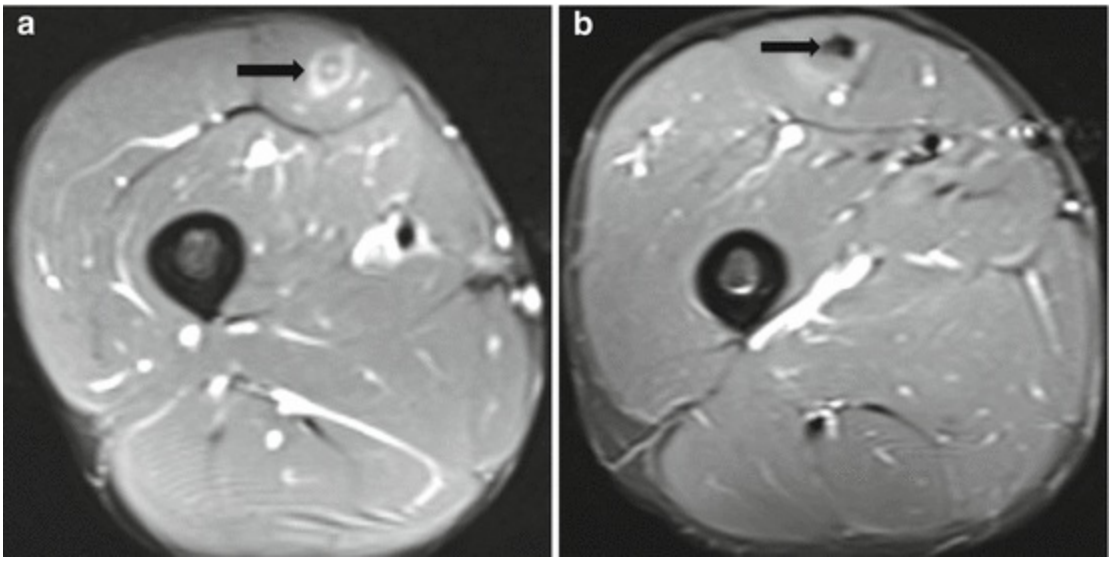


Fig. 16.8 A 22-year-old male athlete with a central injury of the deep myotendinous junction. Axial fat-suppressed T2-weighted MRI (**a**) at initial presentation and (**b**) 12 months later. (**a**) High-signal ring around the deep tendon with bull's eye appearance consistent with central injury (*arrow*). (**b**) Twelve months later, fibrous encasement appears as low-signal thickening of the deep tendon (*arrow*)

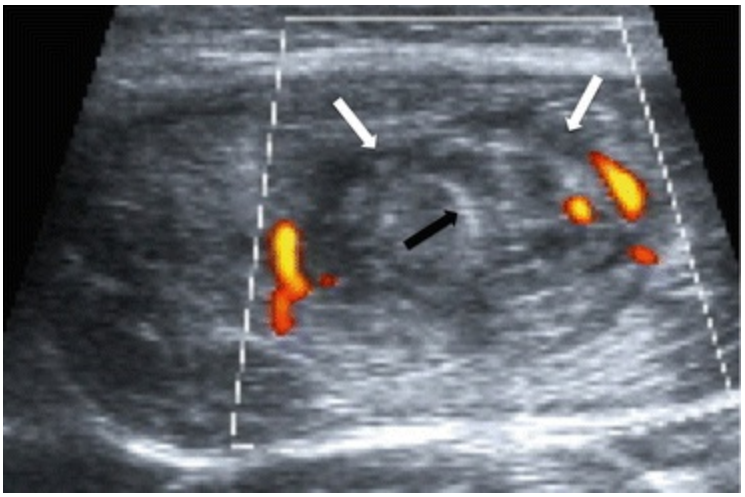


Fig. 16.9 A 20-year-old male athlete with deep myotendinous strain of the rectus femoris. Axial color Doppler image at the mid part of the thigh shows the bull's eye; the intramuscular hematoma (*white arrows*) is centered by a comma-

shaped hyperechoic linear structure, the deep tendon (*black arrow*). The color Doppler shows flow signal related to the surrounding hyperemia

Chronic tears of the deep tendon typically show fibrous encasement of the deep tendon with scar tissue formation that is hypointense on T1- and T2-weighted images (Figs. 16.8, 16.10, 16.11, and 16.12). Fibrous scars can sometimes be mistaken for a soft tissue neoplasm, but the typical cylindrical shape corresponding to the tendon of the indirect head should correct the diagnosis. If the differential diagnosis includes a neoplastic process, such as malignant fibrous histiocytoma, intravenous gadolinium should be administered. Fibrous scars can be detected in ultrasound as a hyperechoic image interposed between muscle fibers and tendon. Chronic lesions can also present a distinct pseudocyst at the site of chronic fibrous encasement appearing as a focal hyperintense area on T2-weighted MRI with well-circumscribed walls (Fig. 16.13). Focal fatty replacement is also found in chronic lesions where it appears as a focal bright area in T1-weighted images adjacent to the tear (Fig. 16.10) [7].

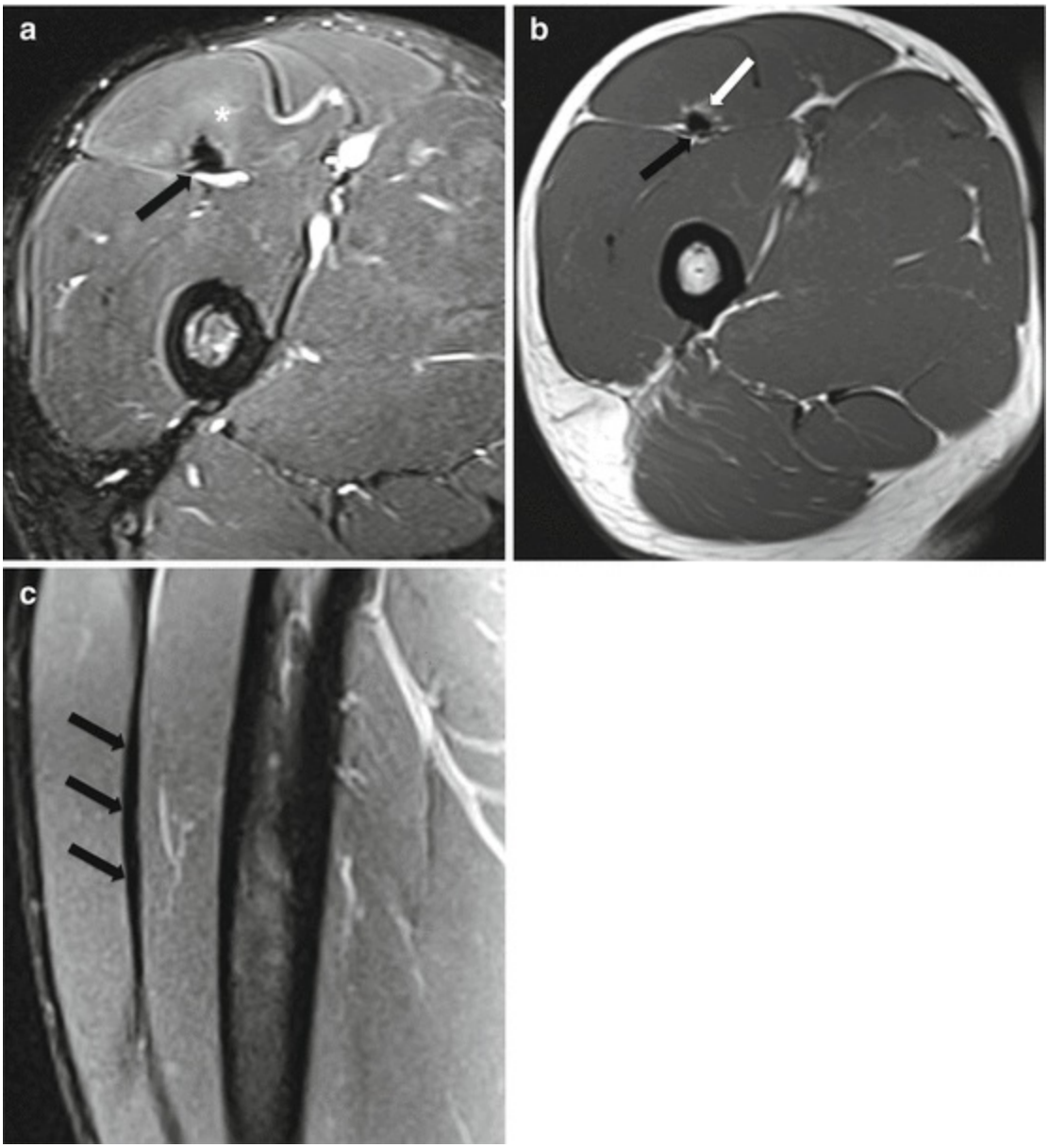


Fig. 16.10 A 35-year-old male athlete with a chronic peripheral myotendinous injury at the mid belly of left rectus femoris. Axial (**a**) fat-suppressed T2- and (**b**) T1-weighted MRI show a low-signal fibrous scar at the point of contact of posterior myotendinous junction (*black arrow*) with slight hyperintensity within the muscle fibers (*asterisk*) and focal fatty infiltration (*white arrow*) in keeping with an old injury. (**c**) Sagittal fat-suppressed T2-weighted MRI show the longitudinal course of the fibrous scar (*arrows*)

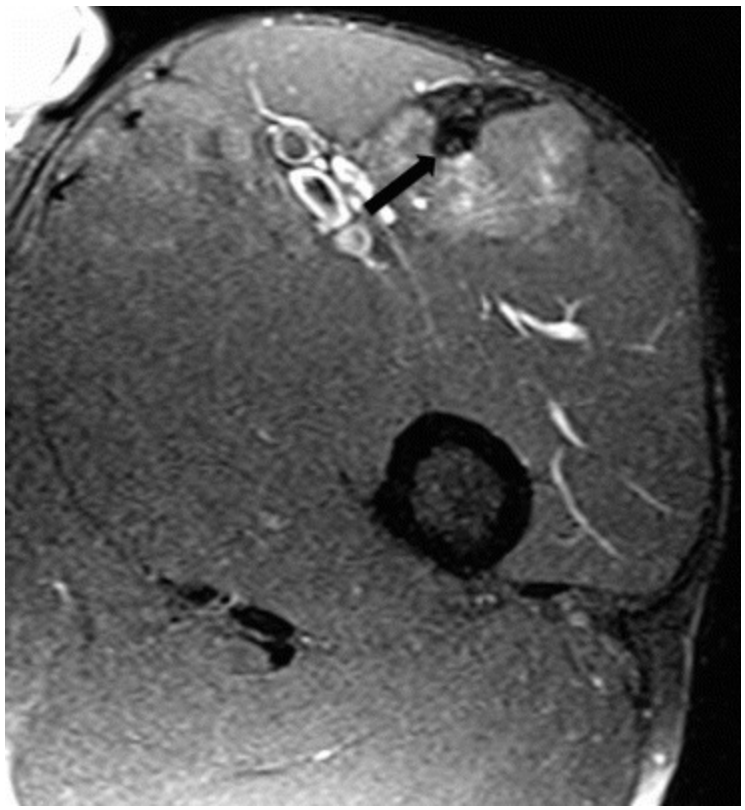


Fig. 16.11 A 32-year-old female athlete with fibrous encasement of the deep rectus femoris tendon three months after injury. Axial fat-suppressed T2-weighted MRI shows thickened and hypointense superficial and deep tendons (*arrow*)

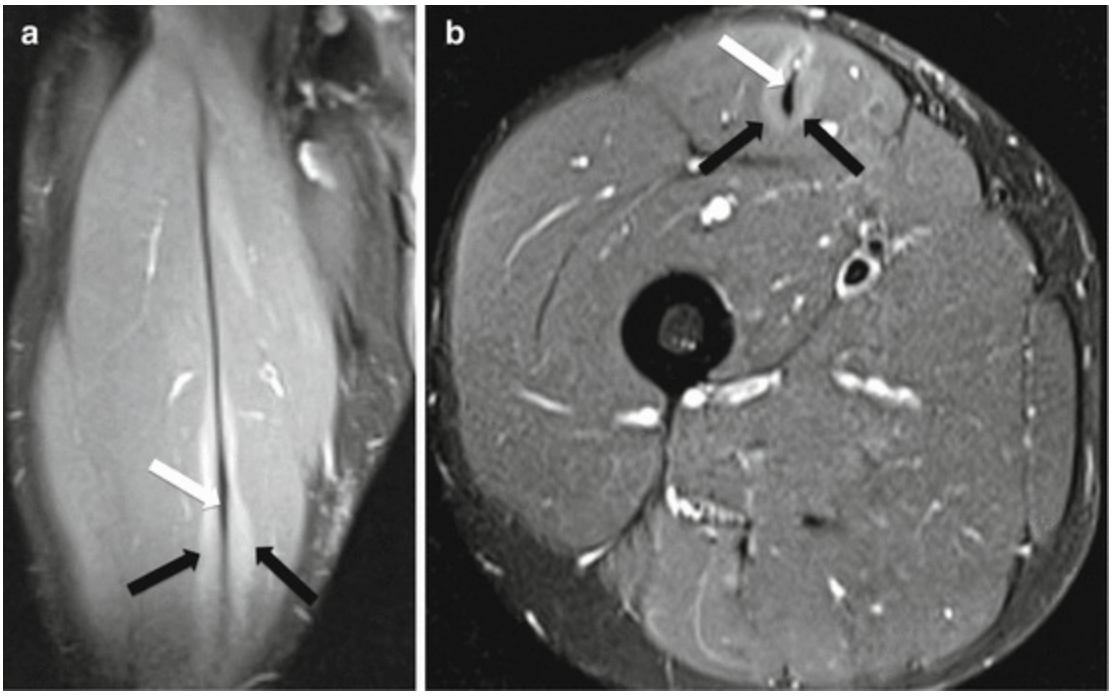


Fig. 16.12 A 23-year-old male athlete with a fibrous scar of the rectus femoris deep tendon. (**a**) Coronal and (**b**) axial fat-suppressed T2-weighted MRI show thickened and hypointense deep tendon (*white arrows*) surrounded by increased signal intensity consistent with edema (*black arrows*)

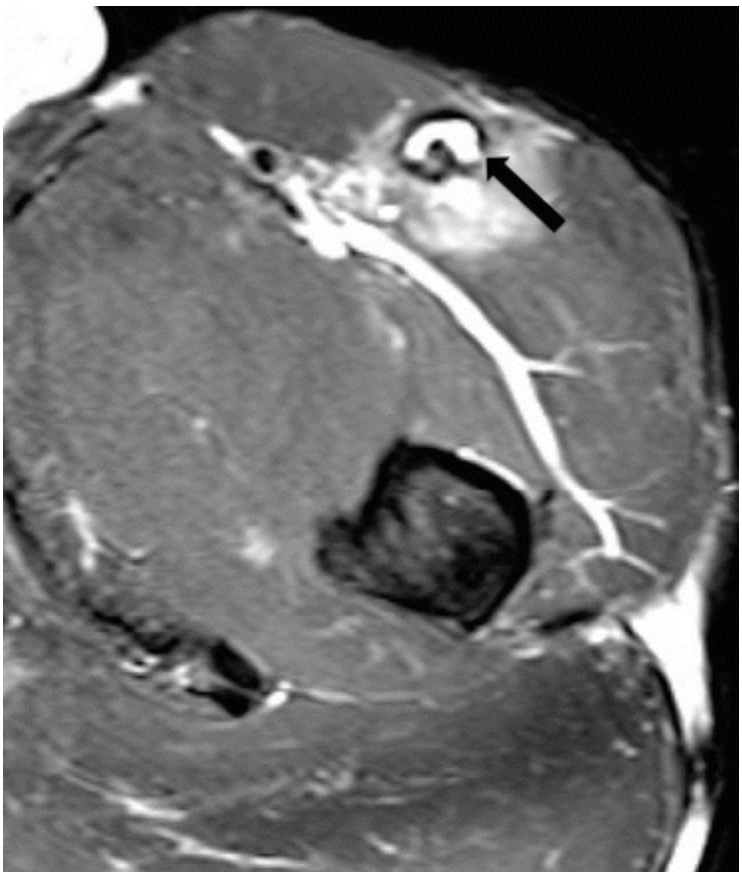


Fig. 16.13 A 32-year-old female athlete with a chronic deep rectus femoris tendon injury. Axial fat-suppressed T2-weighted MRI shows a focal area of bright signal circumscribed by well-defined hypointense walls (*arrow*) in keeping with a pseudo cyst

The role of imaging is not limited only to the initial diagnosis and follow-up of RF injuries. For instance the following radiological findings have been shown to have prognostic value:

- ***Site of injury***: Central tendon involvement (Figs. 16.14 and 16.15) carries a less favorable prognosis than superficial tendon involvement (Fig. 16.16) and is considered the red-flag injury that heralds a protracted rehabilitation [3, 5]. Cross et al. [3] reported an average rehabilitation interval of 27 days for RF central tendon injuries—three times the average interval for direct head strains. However the prognostic value of the longitudinal site of injury (proximal, middle or distal) is less clear with contradictory reports in the literature [3, 5].
- ***Size of injury***: The size of the muscle strain injury assessed by estimating the cross sectional area as a percentage—CSA% as calculated by Walton et al. [40]—and its length are both independently predictive of the rehabilitation time. In case of deep tendon strains, the rehabilitation time of injuries over 13 cm, 32 days, is more than twice that of lesions that are less than 7 cm, which require 14 days [3].

- ***Complete tears***: complete tears also carry the least favorable prognosis but their number is very limited in studies correlating imaging and prognosis of RF injuries.

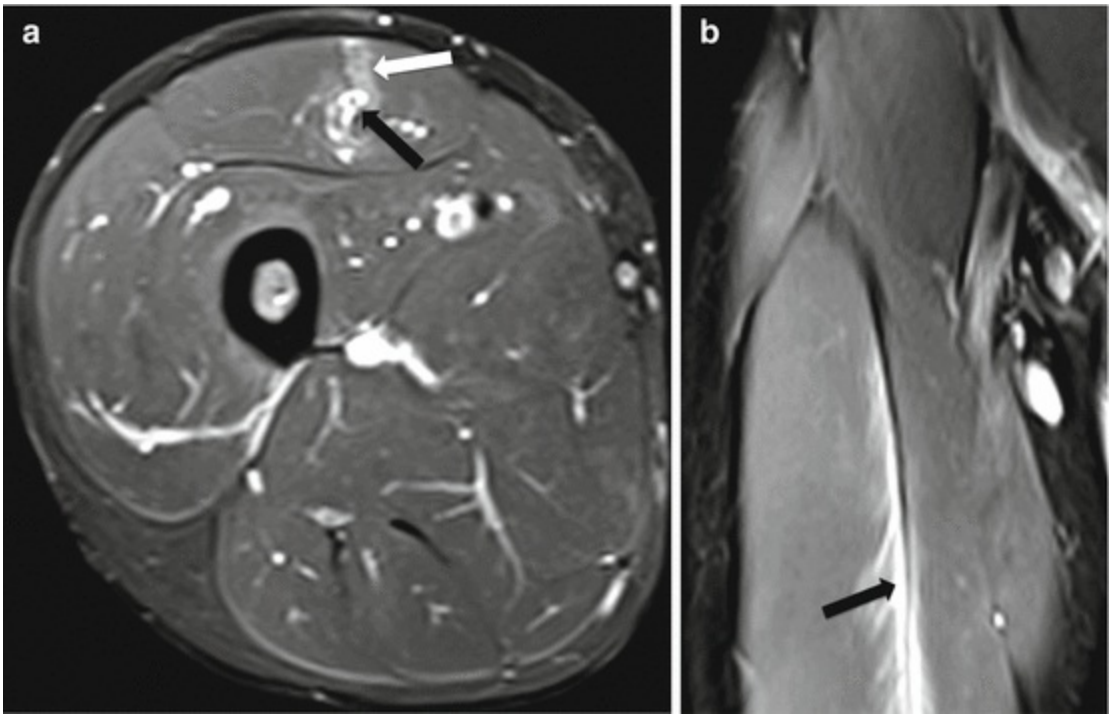


Fig. 16.14 A 23-year-old male athlete with a central myotendinous injury of the deep tendon of the right rectus femoris. (**a**) Axial and (**b**) coronal fat-suppressed T2-weighted MRI show increased signal intensity around the deep tendon consistent with grade I strain (*white arrow*) with mild fluid collection adjacent to the deep tendon (*black arrow*)

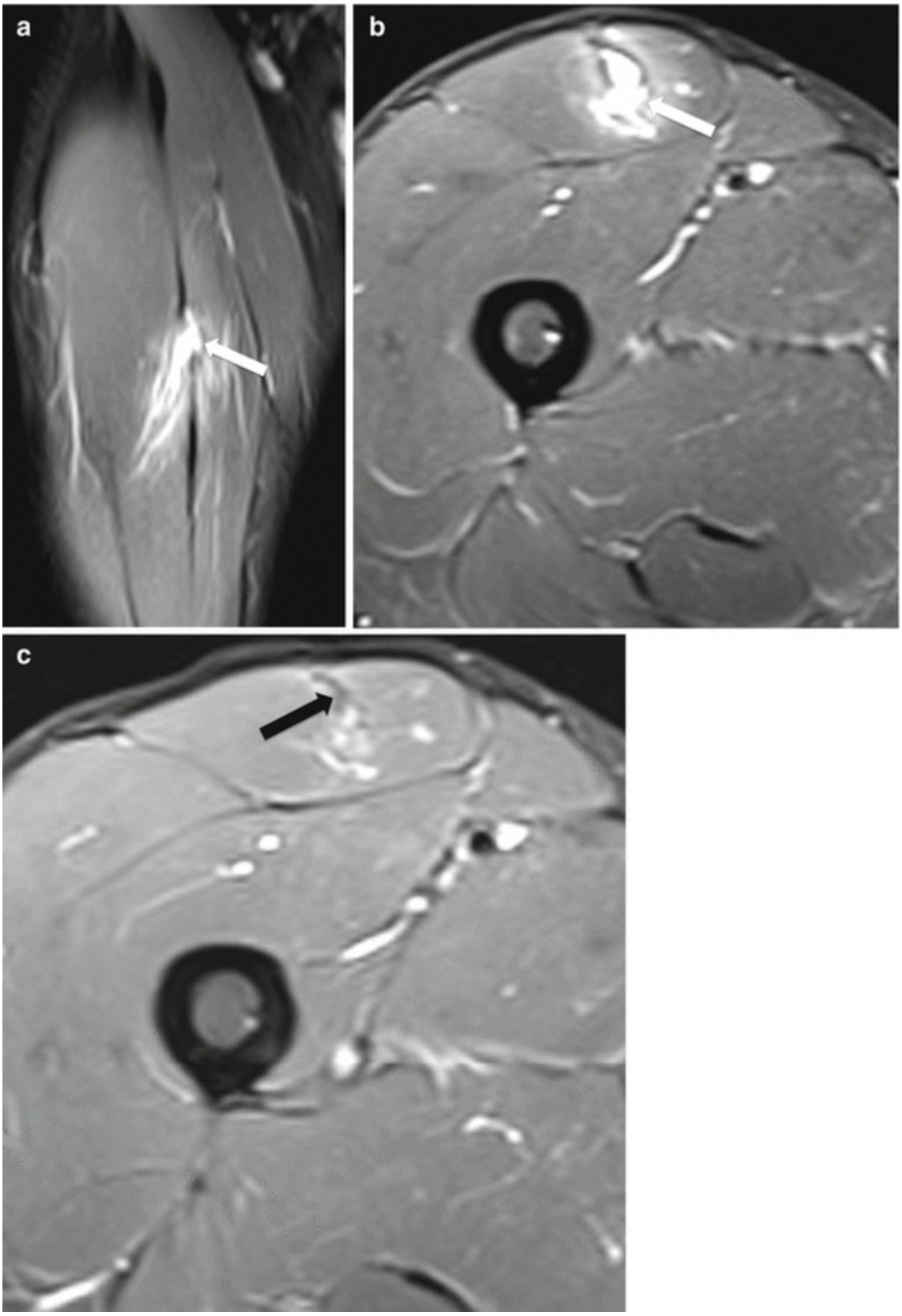


Fig. 16.15 A 25-year-old male athlete with a central myotendinous injury of the right rectus femoris. (**a**) Coronal and (**b**) axial fat-suppressed T2-weighted MRI show a partial tear of the deep myotendinous junction of the rectus femoris associated with fluid collection (*arrow*). (**c**) Axial fat-suppressed T2-weighted MRI performed 10 months later show resolution of the hematoma and low-signal thickening of the deep tendon consistent with a fibrous scar (*arrow*)

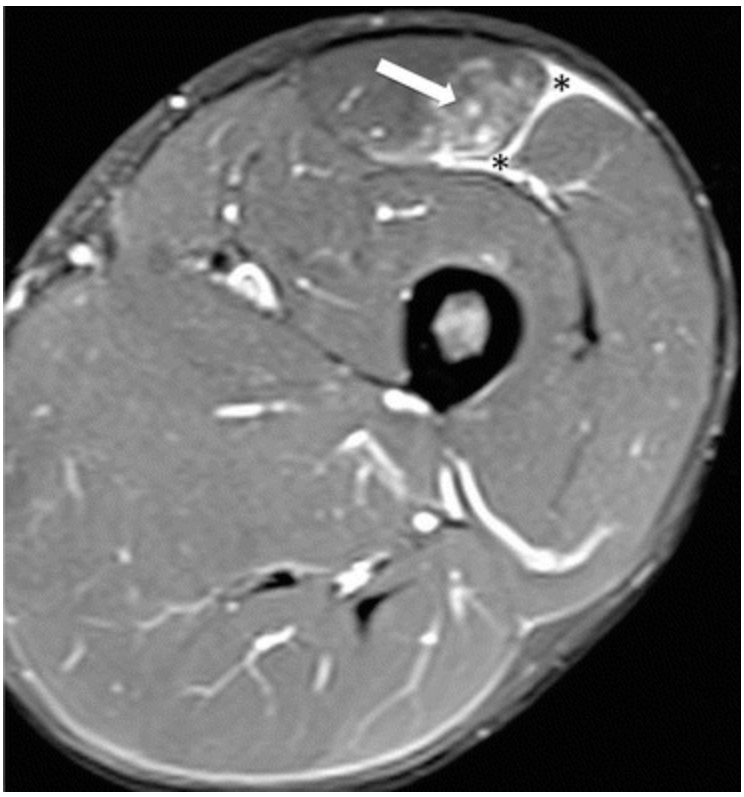


Fig. 16.16 A 35-year old male athlete with a peripheral myotendinous injury of the right rectus femoris in. Axial fat-suppressed T2-weighted MRI shows a peripheral hyperintensity within the mid-belly of the rectus femoris associated (*arrow*) with fluid collection between the rectus femoris, vastus lateralis and vastus intermedius (*asterisks*). These findings are in keeping with grade II peripheral strain

16.9 Treatment

The majority of cases improve with conservative treatment, and surgery is rarely indicated. Compartment syndrome, complete tears, and large hematomas, however, usually require surgery. Conservative treatment consists of the RICE protocol (rest, ice, compression and elevation), physical therapy (stretching and strengthening exercise), non-steroidal anti-inflammatory agents, and gradual return to activity. Nonetheless, studies on nonoperative and operative treatment of RF central aponeurosis strains are lacking. A survey of the (American) National Football League reported on outcomes of nonoperative treatment of RF avulsions from the AIIS, but did not address injuries to the central aponeurosis specifically [41]. For those patients with chronic symptoms from central aponeurosis tears in whom nonoperative treatment failed, Wittstein et al. reported that delayed excision of the torn reflected head can reduce pain, although athletes who do a lot of kicking are still likely to have difficulty competing at high levels despite significant reduction in symptoms postoperatively [42].

16.10 Direct Energy Related Lesions (Contusions) and Delayed Onset Muscle Soreness

Contusions by direct trauma and delayed-onset muscle soreness result in pain at the site of direct trauma that resolves in a few weeks. Both entities can present as increased muscle size with increased signal intensity that is diffuse or geographic with feathery margins that look just like a muscle strain. This type of deceptive similarity is referred to as an "MRI look-alike" and emphasized by Shellock and Fleckenstein [43]. A thorough clinical history should always be sought to differentiate strains from contusion and delayed-onset muscle soreness. A particular complication pertaining to direct trauma is myositis ossificans, which has been reported in up to 9 % of quadriceps contusions. In such cases, pain is often intense and disabling, associated with palpable thickening of the muscle [8, 44]. Five risk factors are recognized as associated with the development of myositis ossificans: knee motion less than 120°, injury occurring during football, previous quadriceps injury, delay in treatment greater than 32 days, and ipsilateral knee effusion [44]. Inappropriate therapy (heat, deep muscle relaxant massages) and excessive premature exertion while the muscle is still painful are also thought to be aggravating factors [8]. Conventional radiographs show heterotopic ossification at the site of impact. Myositis ossificans appears as a focus of low-signal intensity on both T1- and T2-weighted MRI. Myositis ossificans can be treated through arthroscopic excision [8].

16.11 Conclusion

We described an overview of the current state of knowledge of sport related injuries of the RF. MRI seems to be the current imaging modality of choice for diagnosis and follow-up of acute RF injuries, including strain, contusion and avulsion injuries. Ultrasound also plays an important role as an adjunct imaging technique, but it is limited by operator dependency and inability to differentiate between new and old injuries. It is important to use high quality imaging to plan and guide rehabilitation after RF injury, although the decision for timing of return-to-play remains largely dependent on clinical examination findings.

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17. Posterior Compartment of the Thigh Muscles Injuries

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Abstract
Hamstring muscles are the most frequent sites of muscle injury in professional and recreational athletes. As clinical evaluation alone is often insufficient to determine appropriate treatment and prognosis, imaging studies, particularly ultrasound and magnetic resonance imaging, play key roles in the assessment of hamstring lesions. In this chapter, we will describe the anatomy of the hamstring muscle complex and imaging findings of hamstring injuries. A brief overview of treatment options for hamstring injuries will also be given.

17.1 Introduction

The muscles of the posterior compartment of the thigh, also called the hamstring muscle complex (HMC), are the most frequent sites of muscle injury in professional and recreational athletes. Such injuries often lead to physical activity limitations and decreased performance. The incidence of these injuries has increased further with increased physical activity in the general population and high demands on professional

athletes.

As clinical evaluation alone is often insufficient to determine appropriate treatment and prognosis, imaging studies, particularly ultrasound and magnetic resonance imaging (MRI), play key roles in the assessment of HMC lesions. The goals of imaging examination are to confirm the injury; determine its location and extent, with comprehensive assessment of the nature of injury; and guide the choice of treatment (conservative versus surgical) and accompanying approach to muscle recovery. In general, the prognosis of HMC injuries is good, even in cases of avulsion, if they are diagnosed early and treated appropriately.

17.2 Anatomy of the Posterior Muscular Compartment of the Thigh

The posterior muscular compartment of the thigh is composed of three muscles that constitute the HMC: the biceps femoris, semitendinosus, and semimembranosus muscles.

17.2.1 Biceps Femoris Muscle

The biceps femoris is considered a double muscle with long and short heads. The long head arises from the medial facet of the ischial tuberosity via a conjoint tendon shared with the semitendinosus muscle. The short head arises from the lateral lip of the linea aspera, between the adductor magnus and vastus lateralis, the lateral supracondylar line, and intermuscular septum of the femur [1, 2].

The fibers of the long head form a fusiform muscle belly, which passes obliquely and inferolaterally, ending in an aponeurosis that receives the fibers of the short head. This aponeurosis becomes gradually contracted into a tendon, which is inserted into the lateral side of the fibular head, the lateral tibial condyle, and the fascia of the leg [2, 3].

The long and short heads of the biceps femoris muscle are innervated by the tibial portion and peroneal branch of the sciatic nerve, respectively [2, 4, 5]. The muscle's vascular supply is derived from the anastomoses of several arteries: the perforating branches of the profunda femoris artery, the inferior gluteal artery, and the popliteal artery.

Anatomic variation may be present in the form of slips between the hamstring muscles, especially between the short and long heads of the biceps femoris, resulting in variation in the extents of origin and insertion points and reduced flexibility [6]. Occasionally, the short head of the biceps femoris muscle is absent and additional heads arise from the ischial tuberosity, the linea aspera, and the medial supracondylar ridge of the femur [6]. Rarely, the short and long heads of this muscle do not share an insertion point [2, 4].

17.2.2 Semitendinosus Muscle

The semitendinosus is a single muscle, aptly named due to the great length of its distal tendon (about half of its length is tendinous), situated at the posterior and medial aspects of the thigh. It arises from the inferomedial impression of the upper portion of the ischial tuberosity in common with the long head of the biceps femoris muscle.

Distal to the ischial tuberosity, the semitendinosus muscle becomes bulbous and lies posterior to the semimembranosus tendon. More distally, in the middle of the thigh, it forms a long and round tendon. This elongated distal tendon passes along the medial side of the popliteal fossa, then curves around the medial tibial condyle, passes over the medial collateral ligament of the knee, and inserts into the upper part of the medial surface of the proximal tibia [2, 4, 7]. At its insertion, the semitendinous tendon lies behind the tendon of the sartorius and below that of the gracilis. These three tendons form the pes anserinus, so named because it resembles the foot of a goose.

The semitendinosus muscle is supplied by two distinct branches of the tibial nerve, which also innervates the other hamstring muscles. The muscle's vascular supply is derived from the inferior gluteal artery and perforating arteries.

17.2.3 Semimembranosus Muscle

The semimembranosus muscle originates on the superolateral aspect of the ischial tuberosity as a long, flat tendon (or membrane) that runs anteromedial to the other hamstring tendons. The muscle belly begins about halfway down the thigh and has a cordlike appearance and a typical sharp medial border [2, 5]. It has numerous short unipennate and multipennate fibers, maximizing the number of muscle fibrils per unit area.

This muscle has multiple complex distal insertions via five tendinous arms [1, 2, 4]:

- the anterior arm to the medial aspect of the proximal tibia;
- the direct arm to the posteromedial aspect of the tibia;
- the inferior arm to the medial aspect of the tibia, just above the insertion of the tibial collateral ligament;
- the capsular arm, contiguous with the posterior oblique ligament; and
- the oblique popliteal ligament.

The first three arms lie deeper than the tibial collateral ligament, presenting a J-shaped bursa between this ligament and the semimembranosus tendon attachments [8, 9].

The muscle is supplied by a branch arising from the tibial division of the sciatic nerve [2, 3]. The tibial nerve consists of the anterior divisions of ventral nerve roots from L4 through S3. These nerve roots are part of a larger nerve network called the

sacral plexus. The muscle's vascular supply is derived from the profunda femoris and gluteal arteries.

The semimembranosus muscle can be large and occasionally exists as a double muscle, arising mainly from the sacrotuberous ligament. Also, small slips of the semimembranosus tendon insert on the femur, adductor magnus, and posterior horn of the lateral meniscus [2, 8–12]. Conversely, the semimembranosus muscle may be absent [10].

17.3 Biomechanical Features of the Hamstring Muscle Complex and Predisposing Factors for Injury

The HMC is an important hip extensor and knee flexor in the gait cycle. Upon heel strike, it also decelerates the anterior translation of the tibia during knee extension, acting as a dynamic stabilizer together with the static stabilizer, the anterior cruciate ligament. Once foot strike has occurred, the HMC is elongated to optimal length over the hip and knee joints, providing hip extension and, again, knee stabilization [2, 13–15].

Both heads of the biceps femoris muscle perform knee flexion. The long head is a weaker knee flexor when the hip is extended and a weaker hip extender when the knee is flexed. When the knee is semi-flexed, the biceps femoris causes slight lateral rotation of the leg due to its oblique direction [2, 13–15].

The semitendinosus and semimembranosus muscles also contribute to hip extension and knee flexion. These muscles aid in medial rotation of the tibia on the femur when the knee is flexed and medially rotate the femur when the hip is extended [2, 13–15].

Several factors predispose the HMC to injury [2]. The dual innervation of the biceps femoris heads may result in asynchrony in the coordination and/or intensity of the contraction of the two muscle bellies, which may be cause for the high incidence of injuries, especially in the deep portion (myofascial interface) of the long head. The rapid shift in HMC function from the stabilization of knee flexion to hip extension during walking also predisposes these muscles to injury. The biarticular character of the HMC also contributes to the high rate of injury, especially given the disproportionate strength of the antagonist quadriceps muscle group. An imbalance in quadriceps/hamstring contraction exceeding 60 %, or a difference of more than 10 % in HMC contraction between an individual's legs, may be a factor contributing to injury. The prime function of the HMC is eccentric contraction, in which the muscle contracts while being passively lengthened. Injuries occur more frequently during eccentric contraction because contraction stress overlaps extension (stretching). Furthermore, damage is more pronounced in myofibrillar muscles with large proportions of type 2 (fast-twitch) fibers, such as the HMC, which are capable of producing more tension at a greater rate than

other fibers.

17.4 Clinical Findings of Lesions of the Posterior Muscular Compartment of the Thigh

In the clinical evaluation of HMC injuries, the first step is to determine the level of injury. Lesions may be proximally located at the origin of the semimembranosus and conjoint tendons on the ischial tuberosity, with the clinical scenario of pain, difficulty in walking, sitting, and knee flexion, antalgic hip flexion and, rarely, bruises and swelling due to the depth of the lesion.

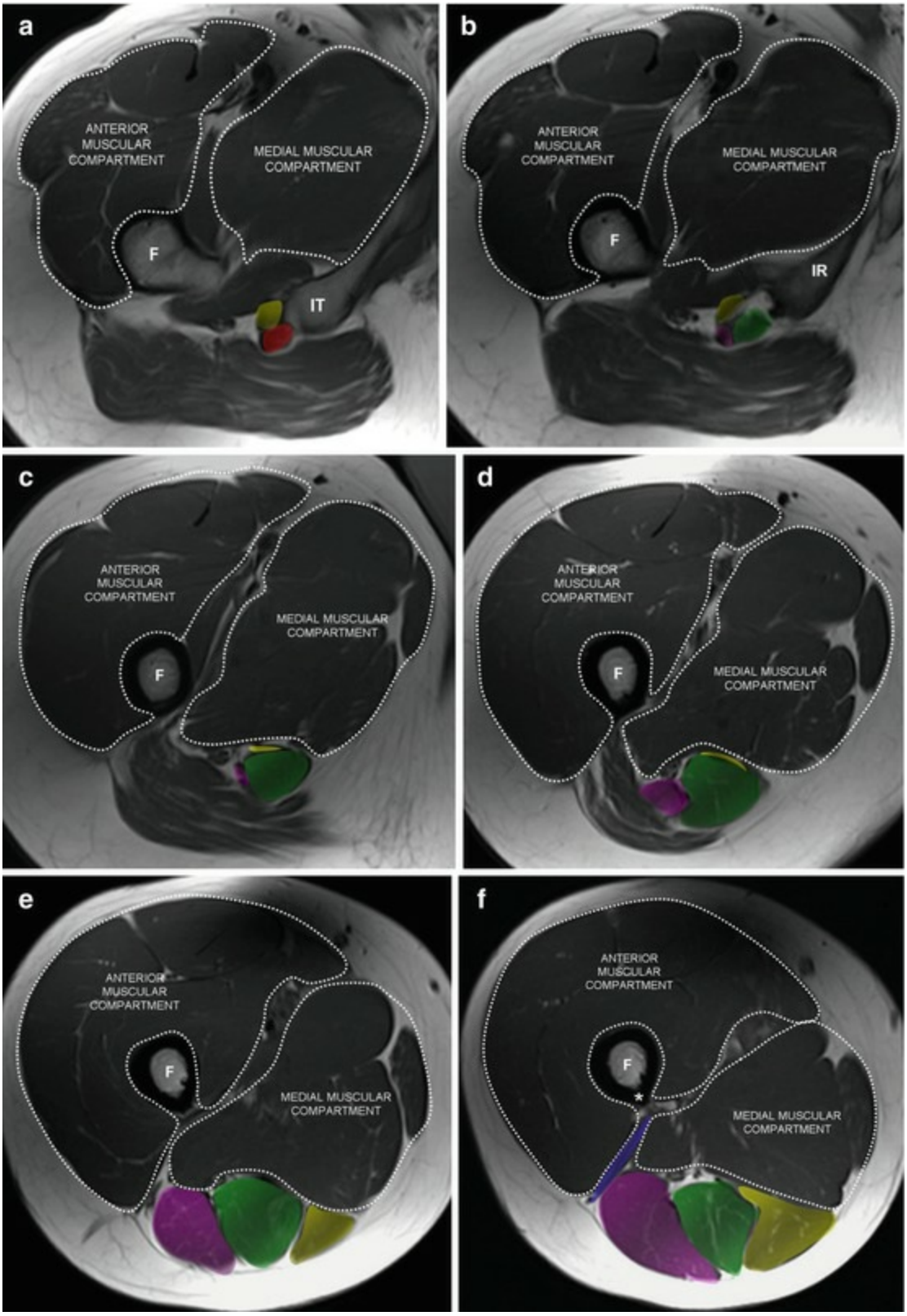
Physical examination of proximal HMC lesions is difficult because of the large muscle mass in this region and the depth of the lesion, but the most notable clinical aspects are pain located inferior to the gluteal fold on palpation and difficulty with knee flexion and posterior thigh elevation. Due to the potential presence of bony avulsions, diagnostic workup with plain radiography, computed tomography, and especially MRI may be necessary. Ultrasound is difficult to perform due to the depth of the structures and overlapping large muscle mass and subcutaneous adipose tissue.

Various degrees of injury to the muscle body may occur, and MRI has enabled accurate lesion staging. The clinical presentation of this injury type is always pain in the posterior thigh, usually after exertion (e.g., sprinting), and may include bruising, difficulty with/antalgic knee flexion, and difficulty with walking that may require the use of crutches. Physical examination is sufficient to achieve an accurate diagnosis in most cases, and the findings are well correlated with the site of injury.

Most distal lesions are related to insertional tendinopathies, notably in the biceps femoris, where avulsion of the fibular dome can be observed. This type of injury is uncommon, but it presents clinically as pain and complete functional disability.

17.5 Normal Imaging Findings

On MRI, two rounded areas of low signal intensity on all sequences in the region of the origin on the ischial tuberosity represent the semimembranosus tendon superolaterally and the conjoint tendon of the biceps femoris and semitendinosus muscles inferomedially. The semitendinosus quickly forms a muscle as it passes into the thigh, posteromedial to the biceps femoris tendon [2, 16–18]. These two muscles can be visualized by MRI until their distal insertions on the lateral side of the fibular head (biceps femoris) and the anteromedial aspect of the proximal tibia (semitendinosus). The complex anatomy of the five tendinous arms of the semimembranosus muscle cannot be distinguished well by MRI (Fig. 17.1) [2, 16–18].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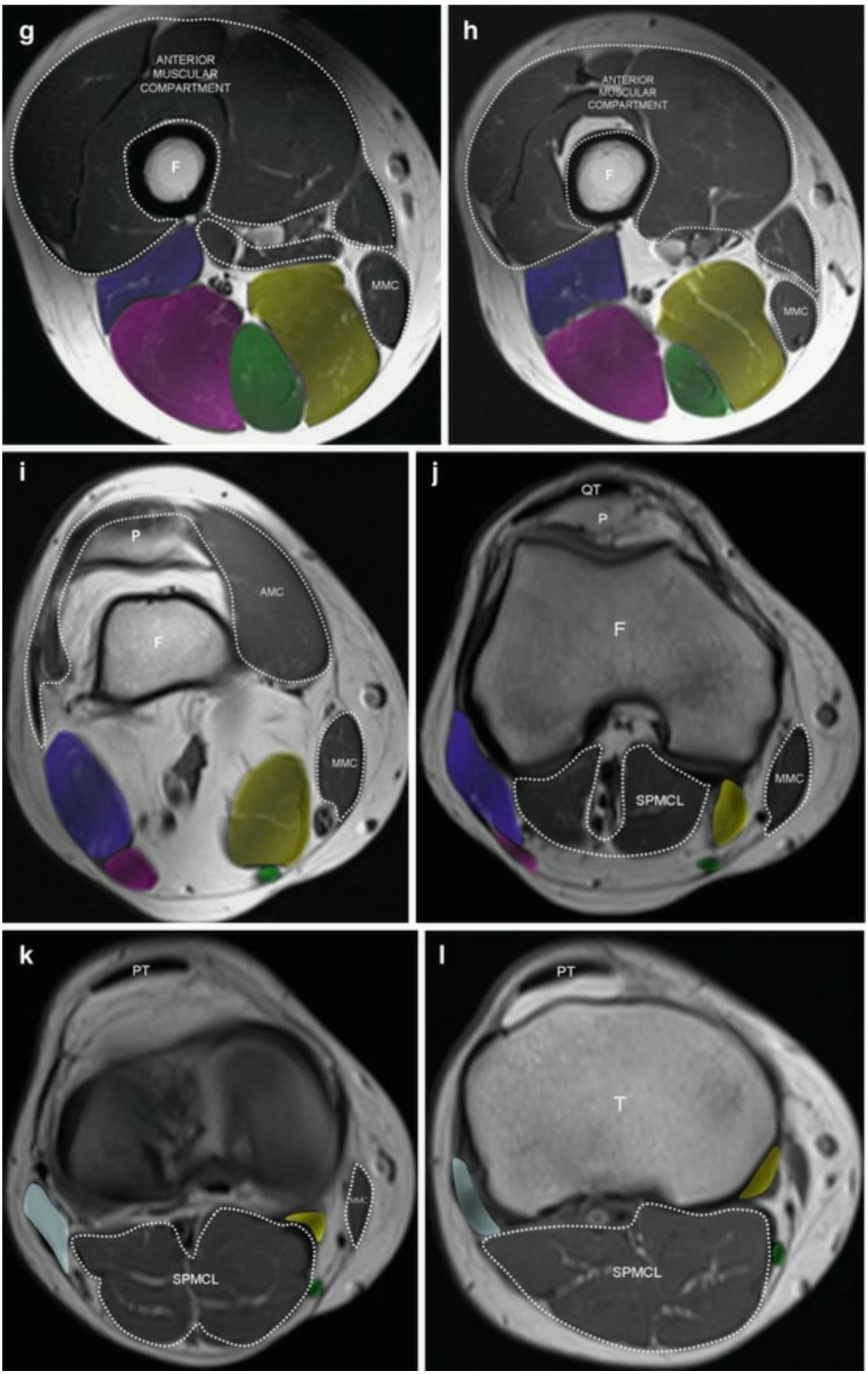
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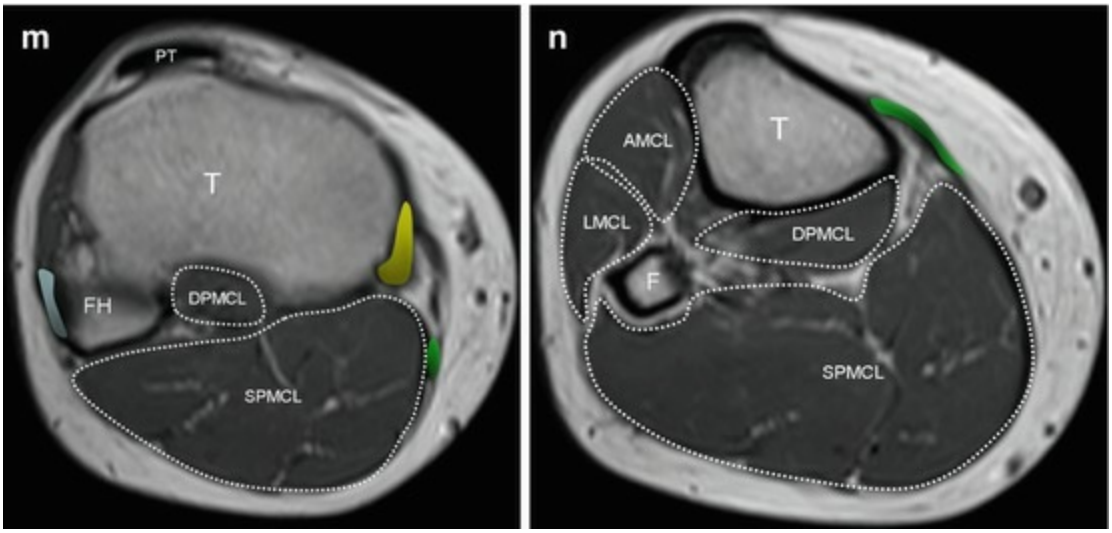


Fig. 17.1 (**a**) Axial T1-weighted MRI shows the long head of the biceps femoris arising together with the semitendinosus tendon (*red*) on the inferomedial aspect of the ischial tuberosity (*IT*). Note also the semimembranosus tendon (*yellow*) on the superolateral aspect of the IT. *F* femur. (**b**) In the plan distal to A, the long head of the biceps femoris (*pink*), semitendinosus (*green*), and semimembranosus (*yellow*) tendons are visible. Note that the semitendinosus muscle becomes bulbous distally in (**c**). *F* femur, *IR* ischial ramus. (**d**) Axial plan of the proximal middle third of the thigh shows the muscle bellies of the long head of the biceps femoris (*pink*) and semitendinosus (*green*). Note the semimembranosus muscle belly (*yellow*) distally in (**e**). *F* femur. (**f**) The three muscle bellies have about the same areas in the middle third of the thigh, and the short head of the biceps femoris tendon (*blue*) arises from the lateral lip of the linea aspera of the femur (*). The belly of this muscle is visible distally in (**g**). Note also the muscle bellies of the long head of the biceps femoris (*pink*), semitendinosus (*green*), and semimembranosus (*yellow*). *F* femur, *MMC* medial muscular compartment. (**h**) Bellies of the short (*blue*) and long (*pink*) heads of the biceps femoris, semitendinosus (*green*), and semimembranosus (*yellow*) muscles in the distal third of the thigh. Note the semitendinosus tendon (*green*) distally in (**i**) and (**j**). *F* femur, *P* patella, *AMC* anterior muscular compartment, *QT* quadriceps tendon, *SPMCL* superficial posterior muscular compartment of the leg. (**k**) Axial plan of the knee shows fused fibers of the long and short heads of the biceps femoris (*light blue*), semitendinosus (*green*), and semimembranosus (*yellow*) tendons. *PT* patellar tendon. (**l**) Axial plan of the knee shows the insertions of the semimembranosus tendon (direct arm; *yellow*) on the medial tibial condyle. The insertions of the biceps femoris (*light blue*) on the lateral side of the fibular head (*FH*) and the anterior arm of the semimembranosus tendon (*yellow*) are visible in (**m**). *T* tibia, *DPMCL* deep posterior muscular compartment of the leg. (**n**) Axial plan of the proximal leg shows the insertions of the semitendinosus tendon (*green*) on the superomedial surface of the tibia (*T*). At its insertion, the semitendinosus tendon lies posterior to the tendon of the sartorius and inferior to that of the gracilis. These three tendons form the pes anserinus. *F* fibula, *AMCL* anterior muscular compartment of the leg, *LMCL* lateral muscular compartment of the leg

On ultrasound, the same two rounded areas at the ischial tuberosity appear as echogenic structures. Distally, the muscle bellies present a fibrillar pattern like that of other skeletal muscles. The distal insertions of the biceps femoris and, especially, the semitendinosus muscles can be identified by ultrasound. This modality is also suitable for evaluation of the pes anserinus tendons. As with MRI, the multiple insertions of the semimembranosus muscle tendons are not well distinguished by ultrasound.

17.6 Acute HMC Lesions

17.6.1 Proximal Lesions

HMC lesions typically occur in the region of the musculotendinous junction (MTJ), a 10–12 cm zone of transition that is the weakest point in the bone-tendon-muscle complex in adults. In children and adolescents, the apophysis is the weakest biomechanical link in the musculotendinous unit (Fig. 17.2) [2]. Injuries to the hamstring origin usually occur after extreme, unbalanced, and often eccentric muscle contraction [19], and the risk of such injury can be increased by any condition that affects muscular function, such as fatigue, poor flexibility, and previous injury [20].

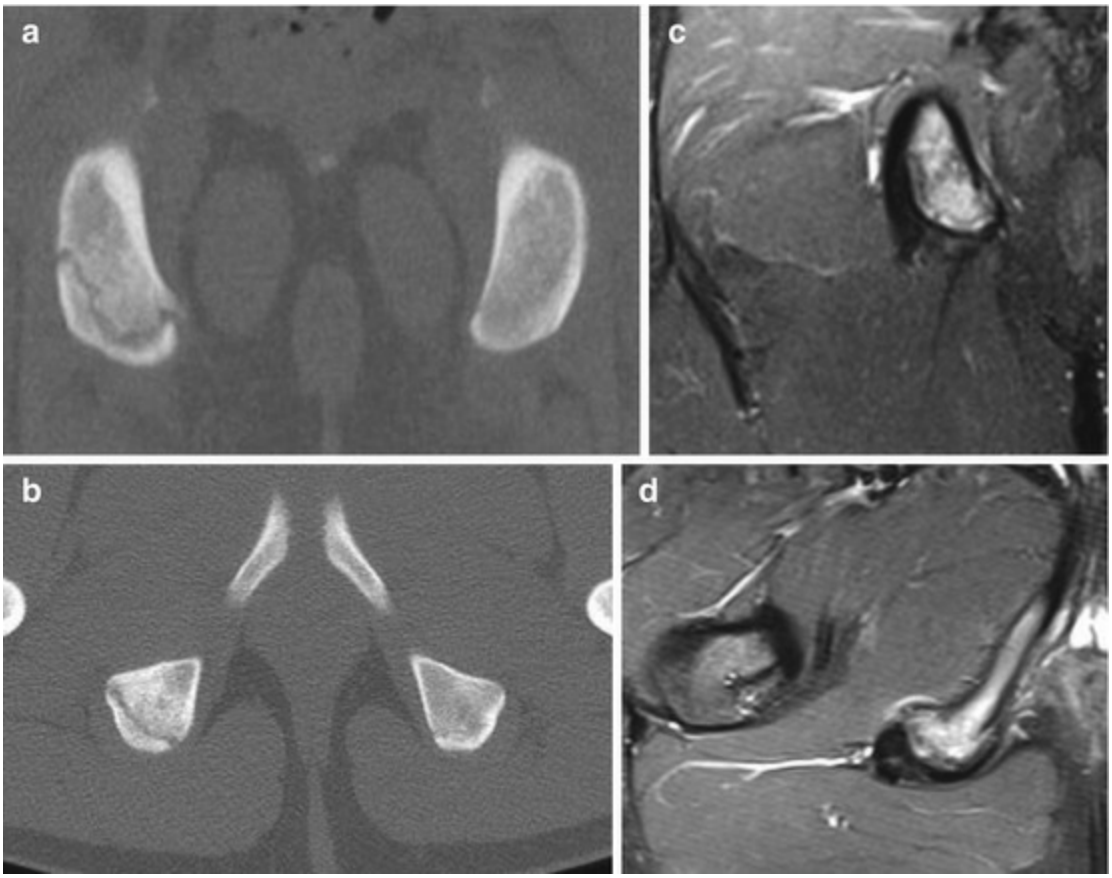


Fig. 17.2 A 17-year-old professional male gymnast with apophsitis. Coronal (**a**) and axial (**b**) CT images show widening and irregularity of the physis. Coronal (**c**) and axial (**d**) fat-suppressed proton density-weighted MRI also show physeal widening associated with bone marrow edema

17.6.1.1 Ischiogluteal Bursitis

The ischiogluteal bursa is a variable anatomical structure located between the gluteus maximus and ischial tuberosity [21]. Ischiogluteal bursitis is an uncommon disorder that can cause referred pain in the posterior thigh and resemble clinical manifestations of

injury to the hamstring muscle.

This lesion was historically encountered in weavers due to irritation of or intermittent pressure on the ischial tuberosity from prolonged sitting, and was thus called "weaver's bottom." Currently, it predominantly affects athletes engaging in sports that cause acute or chronic shearing force on the ischial tuberosity, such as canoeing, horseback riding, and wheelchair racing in paraplegic patients [21, 22].

MRI findings usually include hyperintensity on T2-weighted sequences, with enlarged bursae that may contain blood-fluid levels and septations [22]. Enhancement of the bursal wall, synovial proliferation, and mural nodules can be seen following contrast injection [22, 23].

MRI and advanced imaging techniques also play an important role in differential diagnosis. Intrabursal synovial proliferation can present as a solid mass, making the exclusion of other conditions, such as pigmented villonodular synovitis, synovial sarcoma, synovial hemangioma, and non-calcified synovial chondromatosis, crucial [21, 22].

17.6.1.2 Proximal Hamstring Tendinopathy

High hamstring tendinopathy is a less common overuse injury that manifests clinically as the subacute onset of deep buttock or thigh pain exacerbated by repetitive activity, such as long-distance running, and often aggravated by sitting. MRI findings include increased tendon size, peritendinous edema with a distal feathery pattern, ischial tuberosity edema, and increased internal T1 and T2 signal intensity (Figs. 17.3 and 17.4).

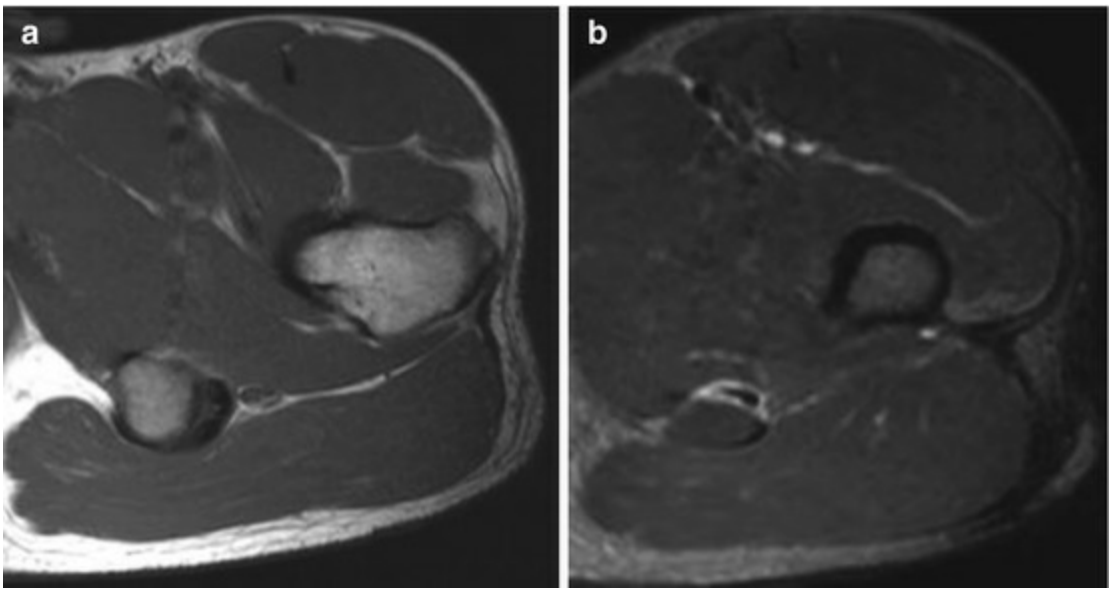


Fig. 17.3 A 21-year-old male soccer player with proximal tendinopathy of the semimembranosus. Axial T1-weighted MRI (**a**) shows subtle areas of increased intratendinous signal intensity and axial STIR image (**b**) shows edema

surrounding the proximal semimembranosus tendon

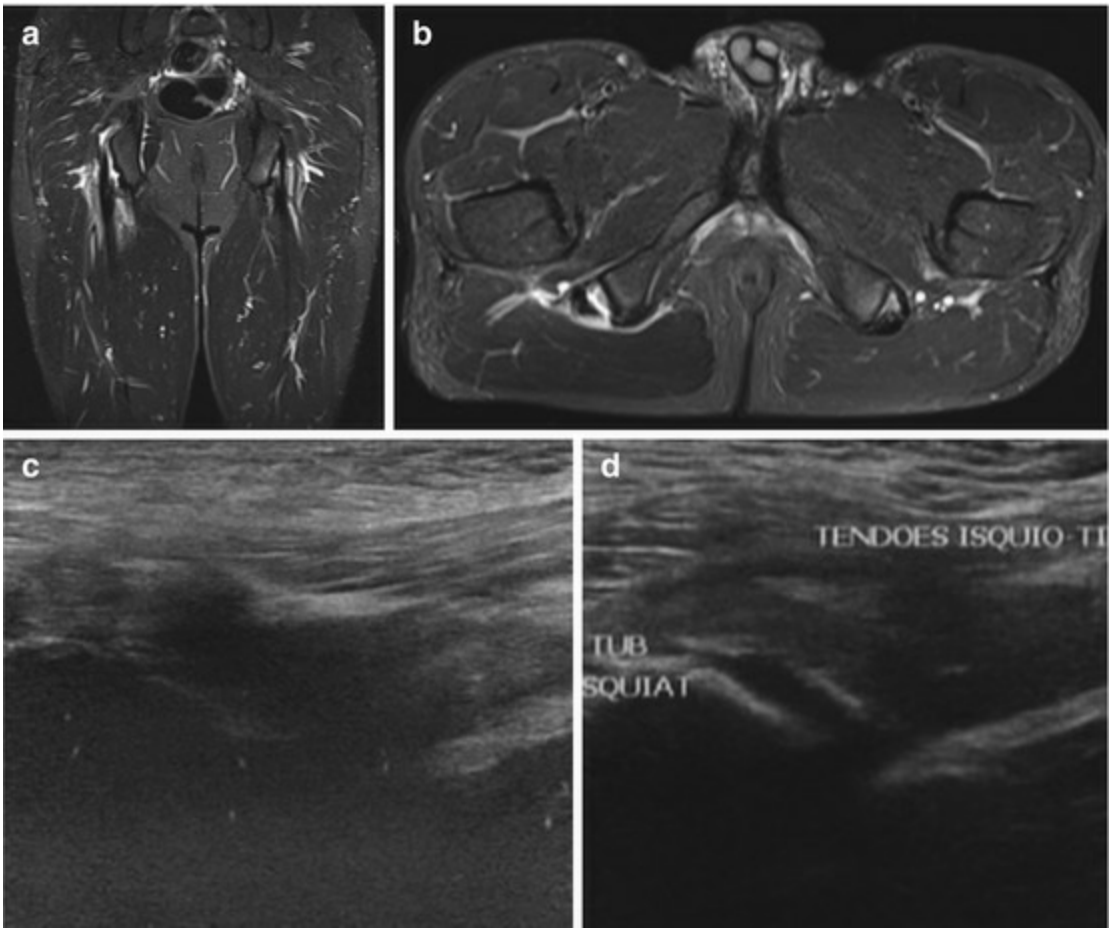


Fig. 17.4 A 33-year-old male professional soccer player. Coronal (**a**) and axial (**b**) STIR MRI show partial rupture of the right hamstring tendons from the ischial tuberosity at their origin. Note also proximal tendinopathy of the left hamstring tendons associated with mild bone marrow edema. (**c**) Posterior longitudinal ultrasound image used to guide the injection of platelet-rich plasma shows retraction of the hamstring muscle insertions. (**d**) Ultrasound examination performed 30 days after platelet-rich plasma injection reveals reduction of the tendinous gap and partial recovery of the fibrillar pattern

17.6.1.3 Avulsion Injuries

Complete avulsion of the HMC from the ischial tuberosity is uncommon in adults. It is often caused by falling or slipping involving forceful forward hip flexion while the knee is extended, resulting in violent overstretching of the hamstring muscles [24]. This kind of injury has been documented predominantly in cross-country and downhill skiers, water skiers, runners, and athletes who commonly perform splits, such as ballet dancers and gymnasts [24–26]. The conjoint tendon is most commonly affected, usually in association with complete or partial tearing of the semimembranosus muscle [7].

The early diagnosis of tendon avulsion is important because acute surgical intervention has a better outcome than do chronic repairs, defined as operative reapproximation of avulsed tendons four weeks after the index trauma [26, 27].

Avulsion injuries at the ischial tuberosity are more common than distal avulsion and usually occur without an osseous fragment in adults [28, 29]. MRI typically demonstrates low signal intensity of the injured and retracted tendon, surrounded by high signal intensity of the proximal muscle belly due to edema or hemorrhage in acute cases (Fig. 17.5) [28]. Ultrasound has limited use in the evaluation of proximal avulsion because of the depth of injury. In young patients, as the apophysis is the weakest link, a displaced osseous fragment can be detected by radiography (Fig. 17.6).

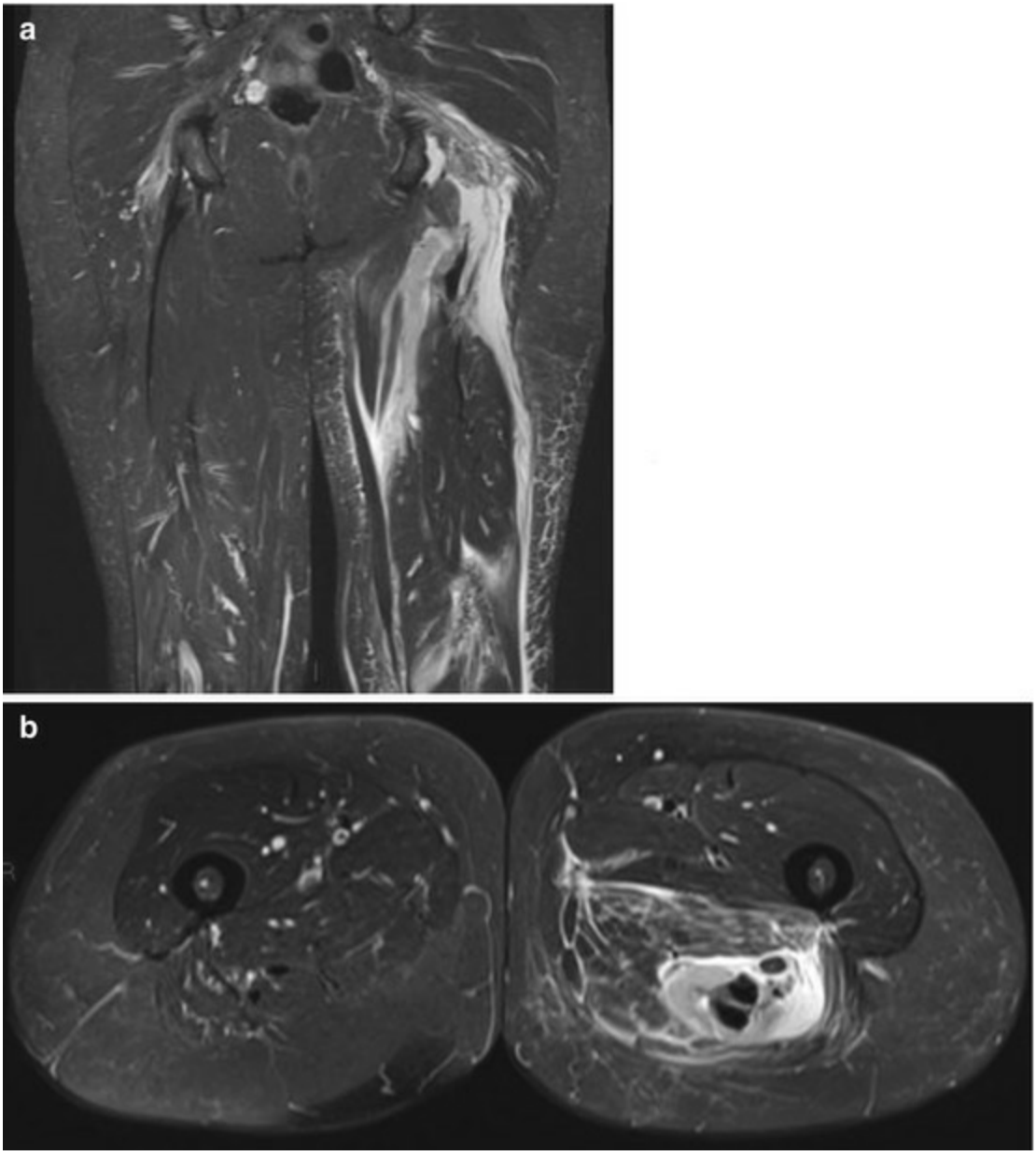
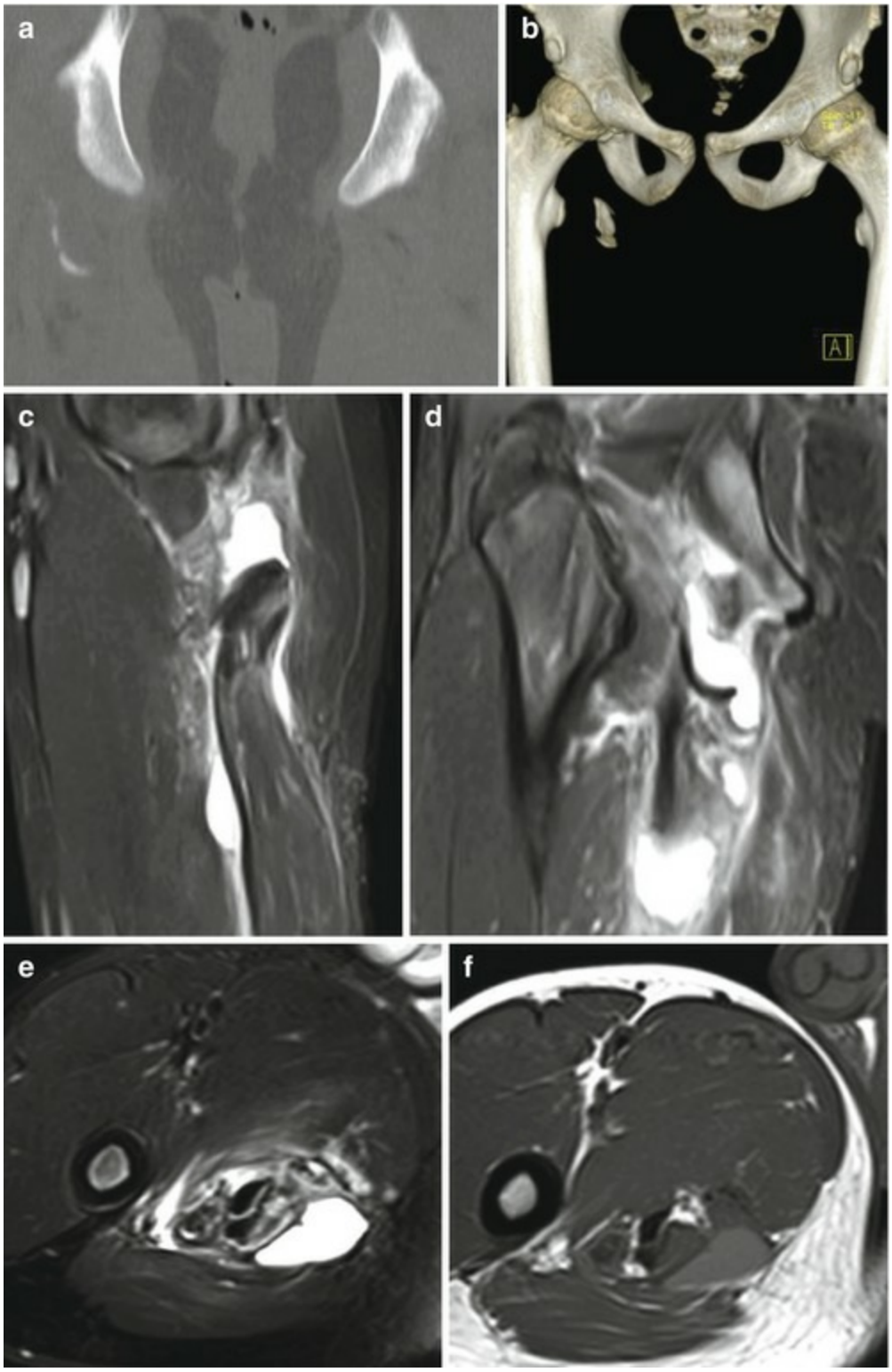


Fig. 17.5 A 66-year-old woman with complete proximal hamstring avulsions after trauma. (**a**) Coronal STIR MRI shows torn and retracted hamstring tendons. Note the presence of a fluid-filled gap at the ischial tuberosity, indicating the avulsion site. (**b**) Axial STIR MRI reveals a large intermuscular hematoma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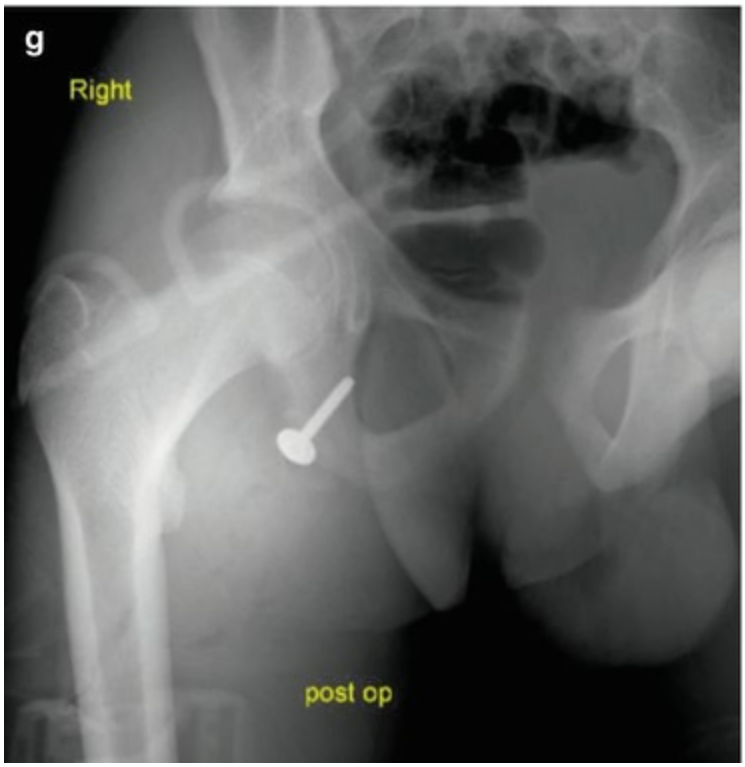


Fig. 17.6 A 13-year-old boy with apophyseal avulsion fracture of the ischial tuberosity. Coronal MRI (**a**) and 3D reformatted CT image (**b**) demonstrate the presence of a displaced fragment of the inferolateral portion of the right ischial tuberosity. Sagittal (**c**) and coronal (**d**) fat-suppressed proton density-weighted MRI show the retracted and torn hamstring tendons. Note also the associated hematoma on axial fat-suppressed proton density (**e**) and T1-weighted (**f**) MRI. (**g**) Anteroposterior radiograph of the right hip taken after surgical treatment reveals internal fixation of the ischial apophysis

Subacute or chronic avulsion may mimic infectious or neoplastic process, and functional MRI may help to exclude conditions such as osteosarcoma or osteochondroma in the differential diagnosis. Muscular atrophy and fatty replacement can also be seen in chronic injuries.

17.6.2 Hamstring Strain

Acute hamstring strains are common in individuals engaging in various sports that involve acceleration, deceleration, jumping, and rapid changes of direction, such as track and field, soccer, and American football [30–32]. Such injuries occur most commonly in the MTJ, where they can be confused with pure intramuscular lesions. They also occur commonly at the myofascial junction, mainly in the long head of the biceps femoris, along the aponeurotic interface with the short head.

Muscles that undergo eccentric contraction, that cross two joints, and that contain high proportions of fast-twitch fibers are at greatest risk of strain. The biceps femoris is by far the most commonly strained muscle, followed by the semimembranosus [33]. Muscle strains are usually caused by overly forceful muscle contraction. A different

type of hamstring strain has been identified in dancers, in which the mechanism involves excessive slow-speed stretching combined with hip flexion and knee extension, as opposed to rapid cutting maneuvers. The semimembranosus muscle is most frequently involved in this type of injury [33, 34].

The types of injury and sports activity influence recovery time; for instance, rehabilitation times are shorter in soccer and American football players than in elite sprinters [30, 32, 33, 35]. This difference is related to the level of exertion; a member of a soccer team playing at 85 % of full capacity will not influence the competitiveness of the team overall, whereas sprinting at only 85 % capacity will make a difference in the final results of competition [33].

Hamstring strain has a high rate of recurrence [36]. Inadequate rehabilitation, early return to sports activity, and factors related to modifications after initial muscle injury, such as weakness, presence of scar tissue, and biomechanical alterations [36, 37], may be responsible for reinjury, which may cause a longer absence from normal activities than the initial injury [30, 38, 39].

17.6.2.1 Grading Muscular Injuries

The degree of strain can be classified into three grades according to the spectrum of injury to facilitate communication among physicians and guide patient management. Alternative classification schemes that consider the site of injury (proximal MTJ, muscle belly, or distal MTJ) and muscular structures involved (intramuscular, myofascial, perifascial, or myotendinous) have been proposed [40].

Grade 1 (Figs. 17.7, 17.8, and 17.9)

This grade describes microscopic injuries to the muscle or tendon, in which 0 % to <5 % of muscle fibers are ruptured. The most common MRI findings are focal or diffuse areas of high signal intensity on fluid-sensitive sequences due to edema and hemorrhage, centered at the main MTJ, surrounding the intramuscular part of the tendon, or in the periphery of the muscle at the myofascial junction [31, 41–43], with intact but potentially distorted myotendinous fibers. The edema track along muscle fascicles may produce a feathery appearance [44]. Associated edema extending into the muscle belly or deep fascia is common and may be extensive, but the injury is classified as grade 1 in the absence of gaps within muscle fibers [45].

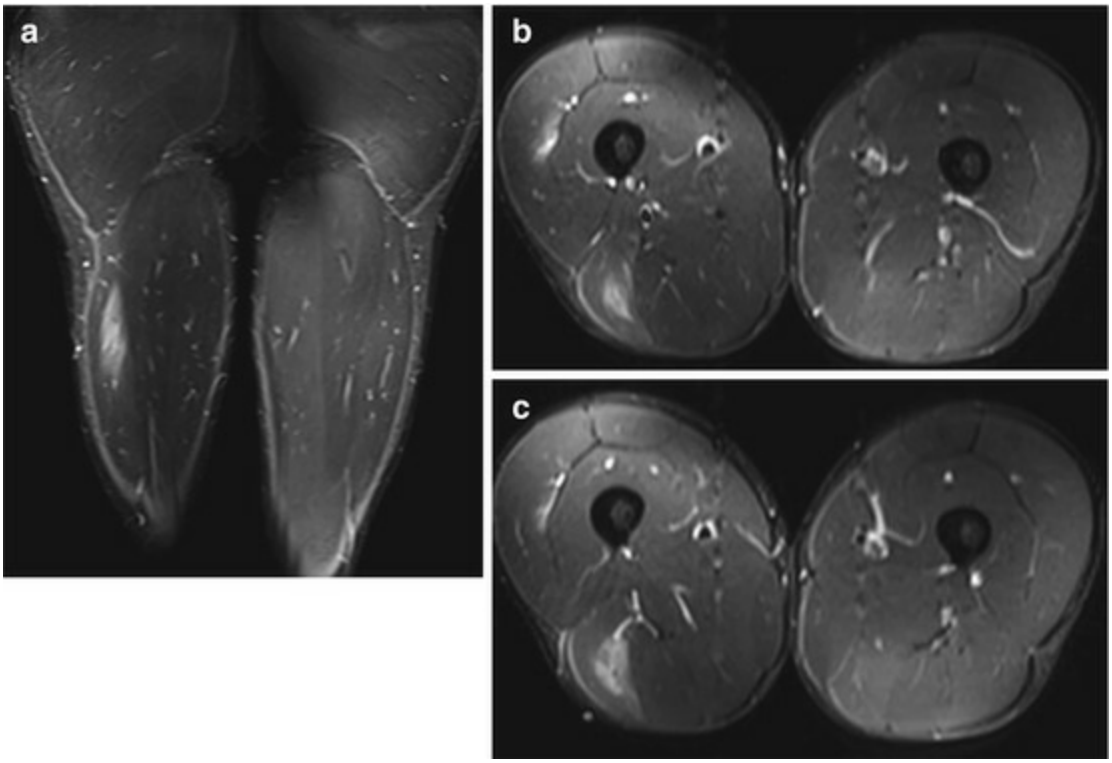


Fig. 17.7 A 27-year-old male professional soccer player with proximal myotendinous junction strain. He presented with pain in the posterior thigh after a match. Coronal (**a**) and axial (**b**, **c**) STIR MRI show feathery intramuscular edema at the proximal myotendinous junction of the biceps femoris, consistent with a grade 1 strain

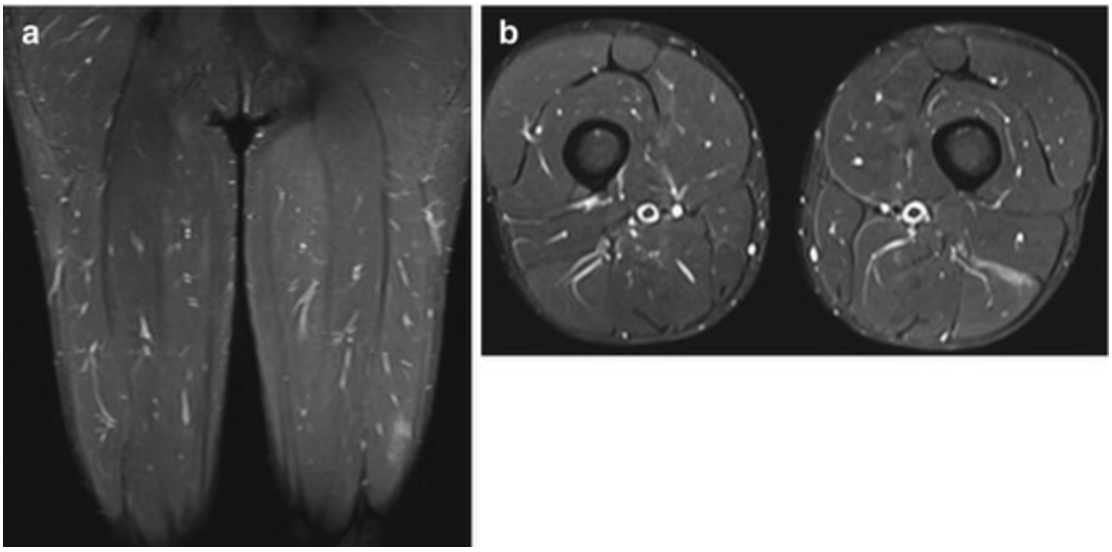


Fig. 17.8 A 27-year-old male professional soccer player with proximal myotendinous junction strain. He presented with pain in the posterior thigh after a match. Coronal (**a**) and axial (**b**) STIR MRI show feathery intramuscular edema at the proximal myotendinous junction of the biceps femoris, consistent with a grade 1 strain

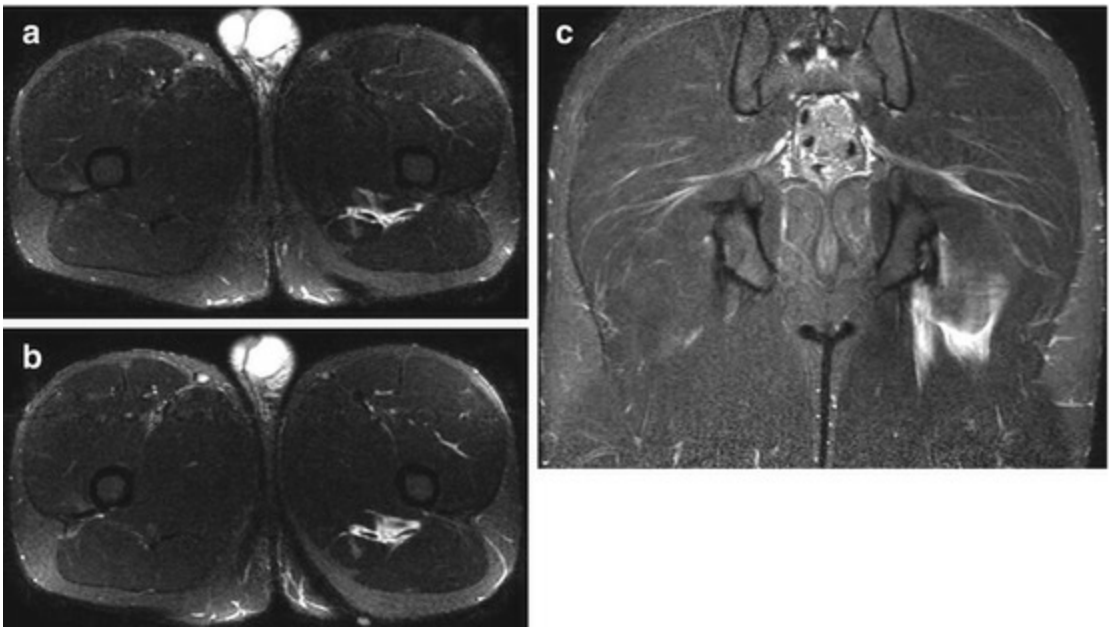


Fig. 17.9 A 21-year-old male professional soccer player presenting with discomfort in the posterior thigh after a training session. Axial (**a**, **b**) and coronal (**c**) STIR MRI show edema in the proximal myotendinous junction of the semitendinosus, with little fluid in the intermuscular space but no muscle fiber disruption, consistent with a grade 1 injury

Grade 2 (Figs. 17.10, 17.11, and 17.12)

A grade 2 designation represents a more serious injury characterized by macroscopic fiber disruption, with some intact fibers near the lesion site. In the acute setting, MRI often shows loss of the normally low-signal intensity intramuscular tendon on T1-weighted images [31]. T1-weighted imaging is less sensitive for depicting muscle injury and is best used to demonstrate areas of hyper- or hypointensity if hemorrhage has occurred, which is age dependent [41, 46]. On T2-weighted sequences, acute grade 2 strains appear hyperintense due to the presence of edema. Considerable retraction of muscle fibers is visible on T1- and T2-weighted sequences [47]. Increased muscle disruption expands the dimensions of abnormality on MRI and ultrasound and increases the probability of visualizing edema and actual fiber disruption [41].

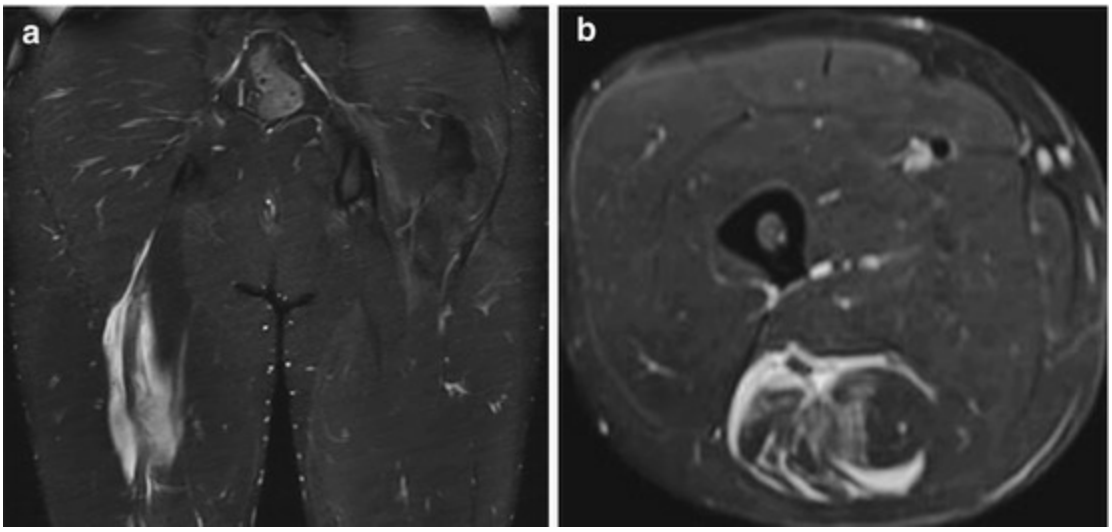
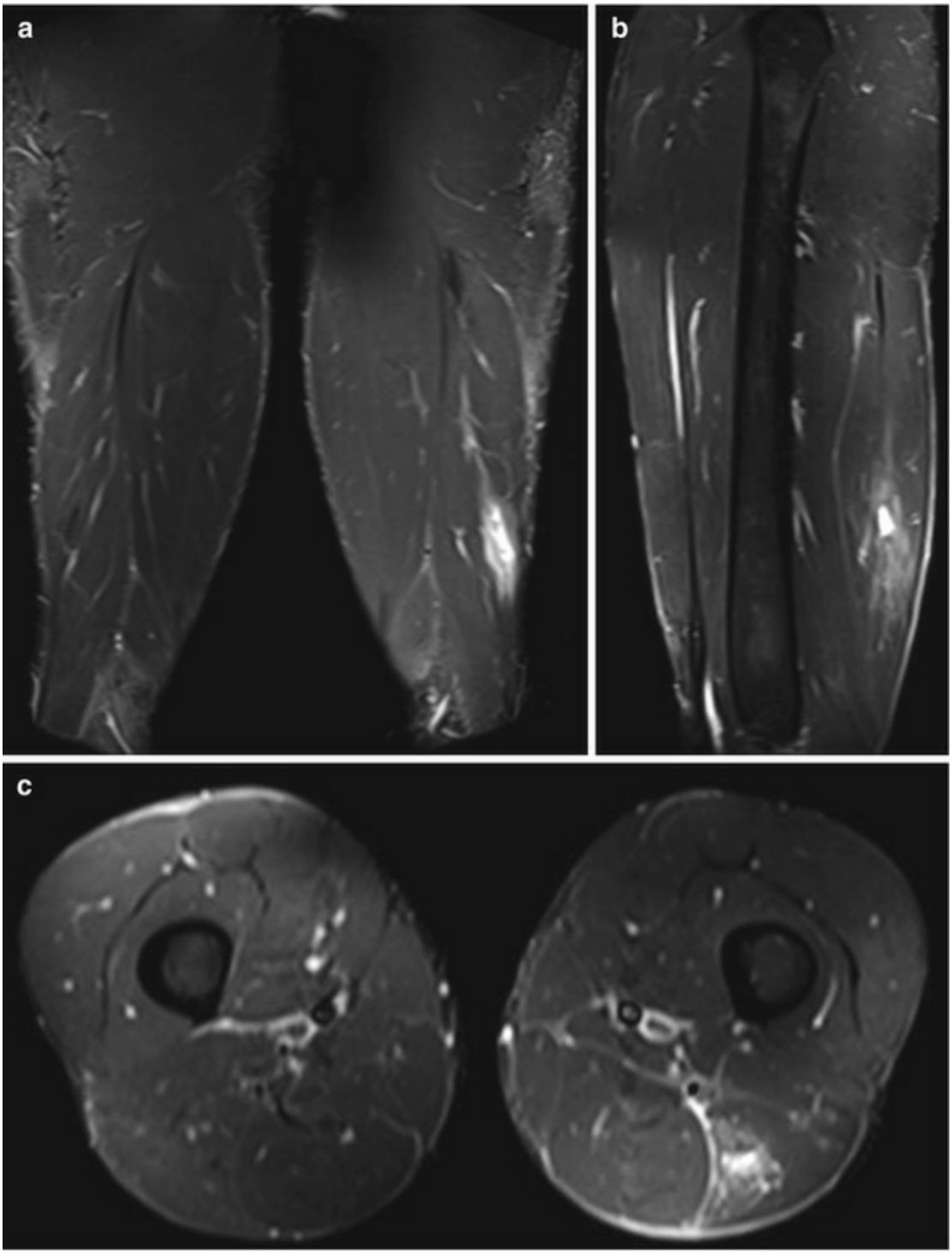


Fig. 17.10 A 35-year-old male professional racecar driver with a grade 2 strain of the biceps femoris. Coronal (**a**) and axial (**b**) STIR MRI show partial rupture of the proximal myotendinous junction of the biceps femoris, with edema adjacent to the conjoint tendon extending into the biceps femoris muscle and a moderate amount of intermuscular fluid. Edema is also present at the proximal myotendinous junction of the semitendinosus, indicating an associated grade 1 strain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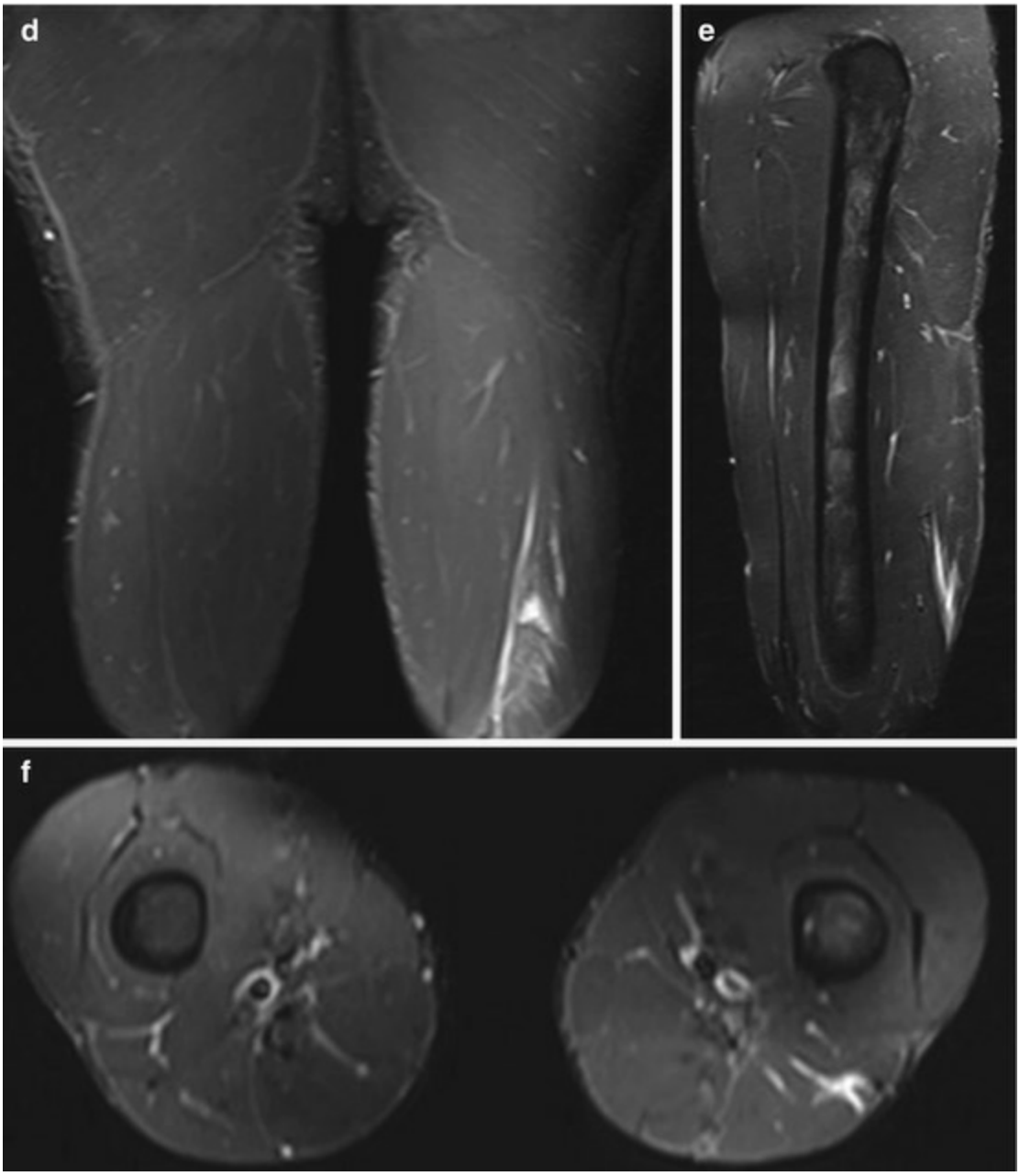


Fig. 17.11 A 33-year-old male professional soccer player with a grade 2 strain of the short head of the biceps femoris. Coronal (**a**), sagittal (**b**), and axial (**c**) STIR MRI show edema and intramuscular hematoma at the short head of the biceps femoris, associated with a small amount of intermuscular fluid. Follow-up STIR MRI (**d**–**f**) obtained in the same planes one month after the initial examination demonstrate the reduction of edema and hematoma

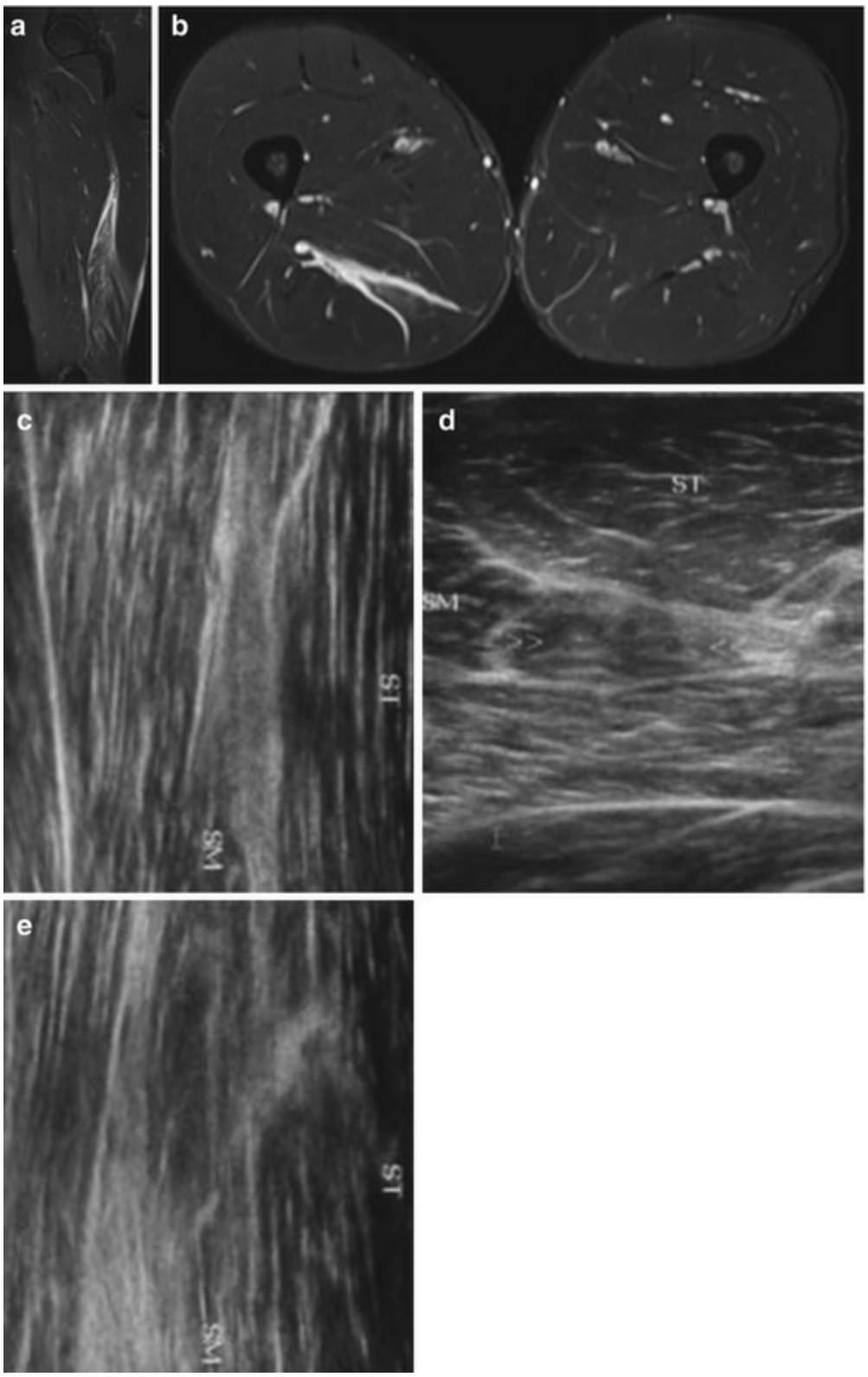


Fig. 17.12 A 34-year-old male professional soccer player with a grade 2 strain of the right semimembranosus muscle. Sagittal (**a**) and axial (**b**) STIR MRI show irregularity of the proximal myotendinous junction of the semimembranosus and intermuscular fluid. Corresponding sagittal (**c**) and axial (**d**) ultrasound images depict a hyperechoic semimembranosus muscle with myofibrillar disruption. Sagittal ultrasound image (**e**) shows the 20-gauge needle in place for ultrasound-guided injection of platelet-rich plasma

As a grade 2 injury partly disrupts the myotendinous fibers, an inter- or intramuscular hematoma may form. Intramuscular hematoma is highly characteristic of grade 2 strains and may form within the space vacated by the lesion. A hematoma focally enlarges the affected compartment and may be found to contain subacute or chronic blood products on T1-weighted images, with echogenicity varying depending on the age and amount of internal liquefaction. Low signal intensity on T1- and T2-weighted sequences due to fibrosis or hemosiderin may be seen in old grade 2 strains.

Grade 3

Grade 3 strains are rare and involve complete disruption of the entire muscular cross section, usually at the MTJ, with or without retraction of the damaged muscle [47]. Such injuries are usually a result of violent contraction against firm resistance. On MRI, complete discontinuity of the muscle, commonly accompanied by muscle fiber laxity, is apparent. The muscle stump appears hyperintense on T2-weighted sequences and the muscle gap is almost always filled by hematoma and effusion. The "bell clapper" sign refers to the retracted muscle fragment floating within a hematoma [47]. MRI is also useful for assessing the extent of tendon retraction for preoperative planning [48]. Grade 3 strains usually require early surgical intervention to prevent permanent retraction and scar formation.

17.6.3 Distal Lesions

Distal hamstring injuries are usually associated with other knee injuries; isolated injury is rare [49]. They usually occur in sprinters or due to hyperextension of the knee [17, 50]. Direct trauma causes muscle contusion or bruising, but it rarely affects the HMC due to its posterior and less exposed location. Indirect trauma causes most sports-related muscle injuries, which manifest as muscle strains or tendon avulsions. A single violent stretch can avulse a tendon from its bone anchor [51], but the majority of stretching injuries are strains caused by eccentric stretching, which occurs when a muscle contracts at the same time that it is being lengthened [52].

Few published reports have focused on distal HMC lesions. The most significant report [50] describes a series of 18 distal hamstring tears in athletes, all of which occurred during sports activity, typically sprinting, with no direct trauma. The biceps femoris was injured in 11 patients, the semimembranosus in five patients, and the semitendinosus was injured in two patients. These findings are consistent with reports showing that the long head of the biceps femoris is the most commonly injured muscle, followed by the semimembranosus and semitendinosus [7, 8, 31, 33, 39].

17.7 Chronic Lesions

17.7.1 Chronic Tendinopathy

The causes and pathophysiology of chronic tendinopathy in humans have not been scientifically proven, but an association has been proposed between the tendon's failed healing response to repetitive stretching and mechanical overload and the development of tendinosis [53].

Koulouris and Connell [7] found imaging features of preexisting hamstring enthesopathy in three of five patients with acute partial tears of the hamstring muscles. Pathological changes in the proximal hamstring tendons may expose the muscles to partial and complete ruptures, at least in some cases. This finding has also been described in other tendon ruptures.

Because of this region's complex anatomy, chronic gluteal and posterior thigh pain often poses a diagnostic challenge. The main conditions that should be considered in the differential diagnosis are piriformis syndrome, stress fracture, apophysitis, avulsion fracture, bursitis, posterior femoral compartment syndrome, partial hamstring tear, lumbar radicular pain, and other causes of sciatic pain, such as soft-tissue tumors [21, 54, 55].

Sciatic nerve irritation is occasionally related to and may aggravate the pain caused by proximal hamstring tendinopathy [56]. The mechanism of such irritation may involve scarring and adhesions around the nerve caused by repetitive stretching and continuous overload of the proximal hamstring tendons. Another proposed mechanism is impingement of the sciatic nerve because of a swollen and thickened tendon insertion on the lateral part of the ischial tuberosity [53]. However, pain originating from the tendon is difficult to differentiate from that originating from nerve involvement.

Distal hamstring tendinopathy can lead to posterolateral and posteromedial knee pain. Causes of pain in this area include meniscal tears, osteoarthritis, tendinopathy or bursitis of the pes anserine, semimembranosus bursitis (Fig. 17.13), popliteal (Baker) cysts, gastrocnemius or popliteus muscle strain, and medial or lateral collateral ligament sprain. Friction and repetitive eccentric tendon loading can lead to degenerative changes in distal hamstring tendons and insertions, and irritation of the bursa.

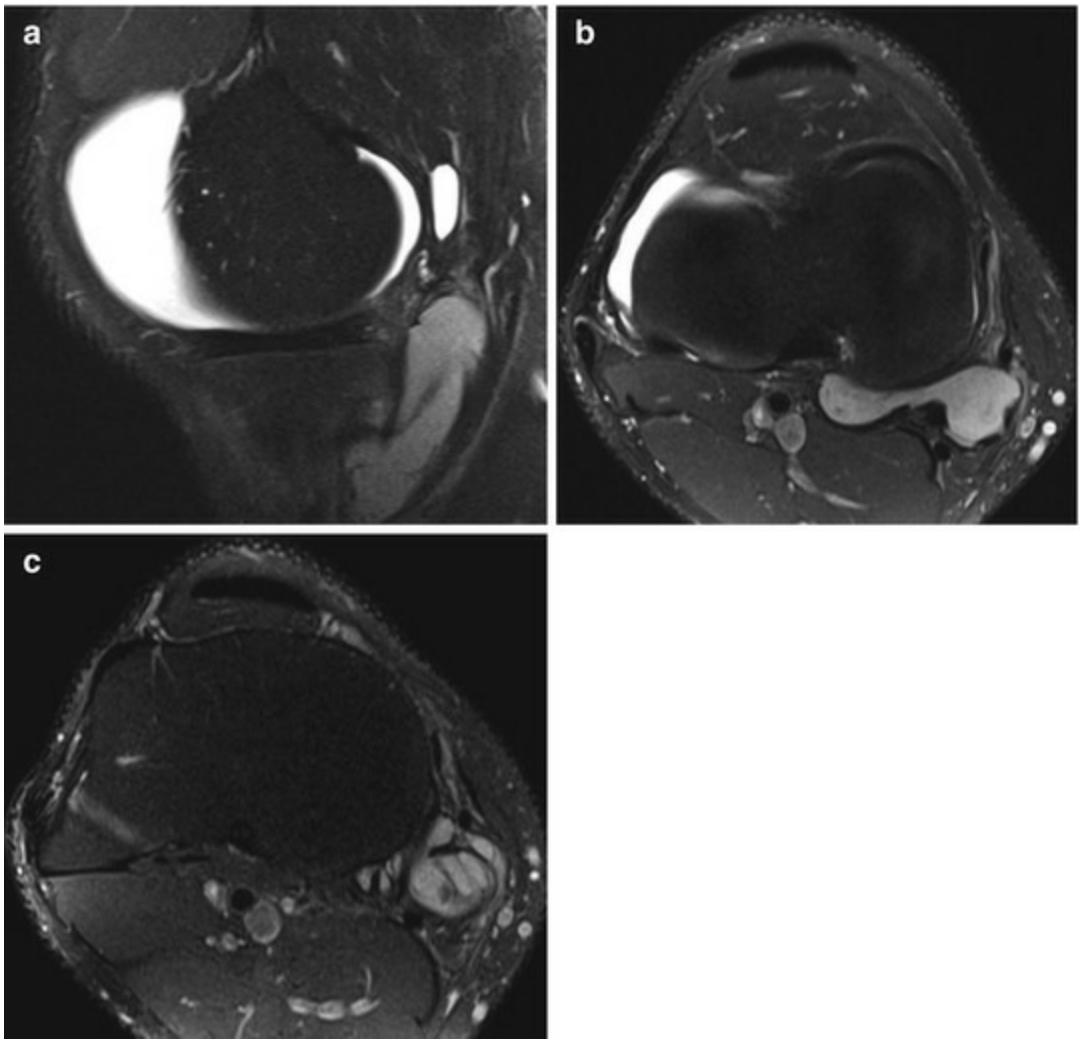


Fig. 17.13 A 23-year-old man with semimembranosus bursitis, presenting with medial knee pain. Sagittal (**a**) and axial (**b**, **c**) fat-suppressed proton density-weighted MR arthrograms show an abnormally enlarged bursa associated with synovial thickening

Two types of patient are predisposed to developing distal hamstring tendinopathy: young athletes due to overuse and, more commonly, middle-aged and elderly patients. In elderly patients with osteoarthritis, tendinosis can develop in the anterior reflected semimembranosus tendon insertion secondary to adjacent osteophytes on the joint line. These patients also frequently suffer from concomitant pes anserine tendonitis [57]. Total knee replacement components can also cause secondary semimembranosus tendinopathy.

MRI is recommended to confirm the diagnosis of hamstring tendinopathy and to help rule out other causes of pain. It provides detailed anatomical information and enables the identification of pathological changes in the tendons. Typical MRI findings of hamstring tendinosis include increased tendon girth and intrasubstance signal heterogeneity [58, 59]. Reactive edema of entheses, such as the ischial tuberosity, and even cortical defects are occasionally noted [53]. Tendon thickening with increased

signal intensity on T1-weighted and proton-density images and without bright signal tear on T2-weighted images is diagnostic of tendinosis.

Occasionally, MRI reveals a markedly thickened and edematous sciatic nerve, which may explain patients' neuropathic symptoms. Surrounding bursal fluid is another possible finding when distal tendinosis is suspected.

17.7.2 Chronic Tears

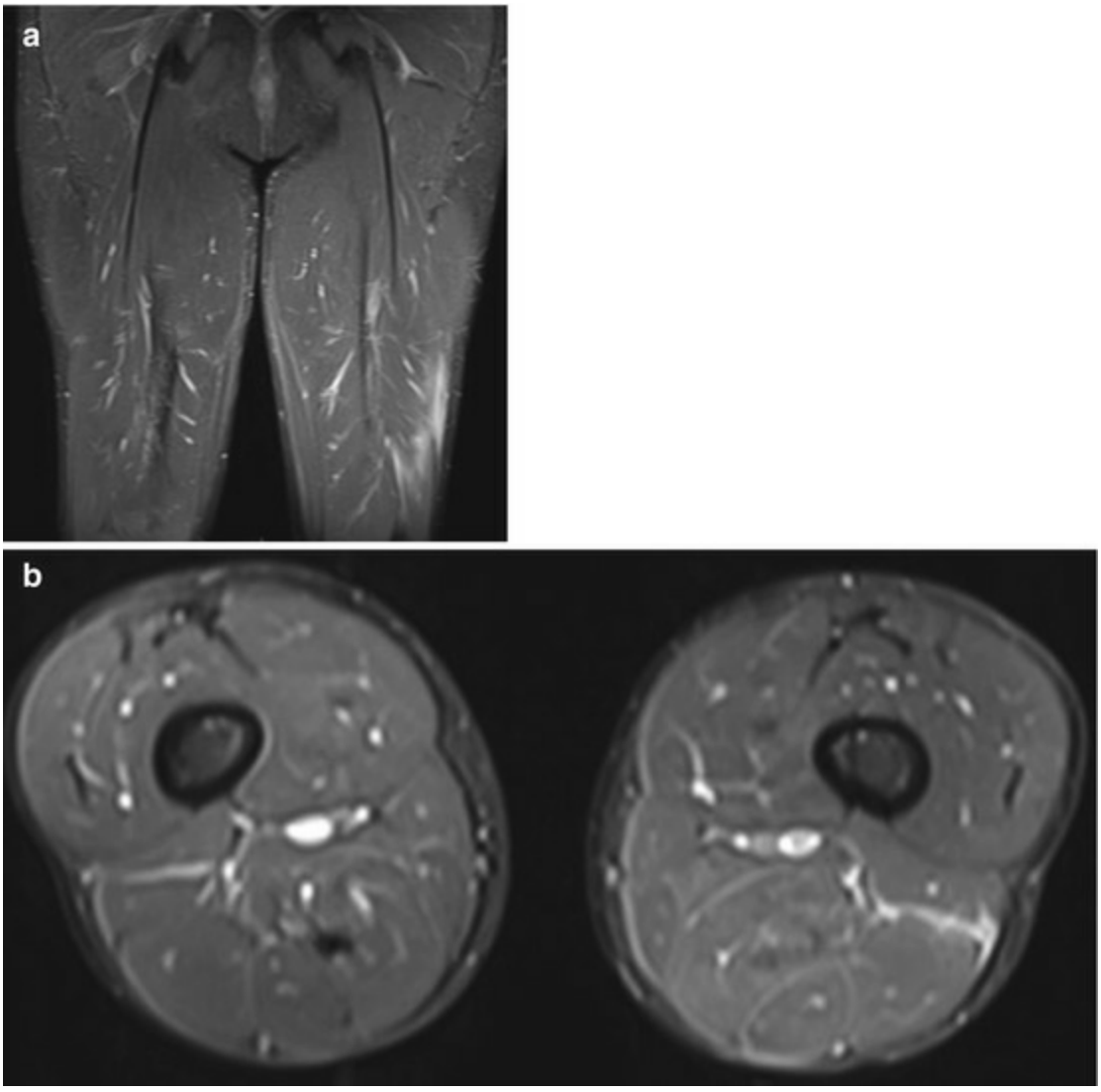
After a disruptive shearing type of injury, the affected area of a skeletal muscle undergoes remodeling. In grade 1 strains, in which the sarcolemma is intact, regeneration occurs due to the proliferation of myocytes [47]. In grade 2 and 3 strains, two healing processes occur: regeneration and the formation of fibrous scar tissue. The frequency of scar tissue formation increases with the extent of the initial tear.

Scar tissue formation and fibrosis commence as early as 7 days after injury, as detected histopathologically [51, 60]. These processes are not usually observed radiologically until the scar tissue has matured and become macroscopically visible [41]. Excessive scarring and adhesion formation can impair muscle function and reduce biomechanical properties [61].

The key objective in the imaging examination of chronic tears is the identification of scar tissue, as strain may recur near regions in which normal muscle contractility and mobility are impaired due to shortening and tethering [60]. The neurovascular bundle should also be assessed routinely because chronic injury may cause tethering of the sciatic nerve [62].

The appearance of chronic injuries is less predictable than those of acute strain and avulsion. The absence of fluid collection at tendon margins, fatty infiltration of the muscle, reduction in hamstring muscle volume, and the presence of fibrosis and/or scar tissue are all suggestive of chronic hamstring injury [7].

Scar tissue has low signal intensity on all MRI pulse sequences; because it forms at the site of injury, it appears as areas of nodularity adjacent to or within the normal low signal of the tendon (Fig. 17.14) [63]. This appearance may be confused with an area of focal, discrete tendon disruption, with associated retraction. The adjacent muscle fibers may appear hyperintense on T2-weighted images due to degeneration, chronic inflammation, and the presence of granulation tissue [47].


a
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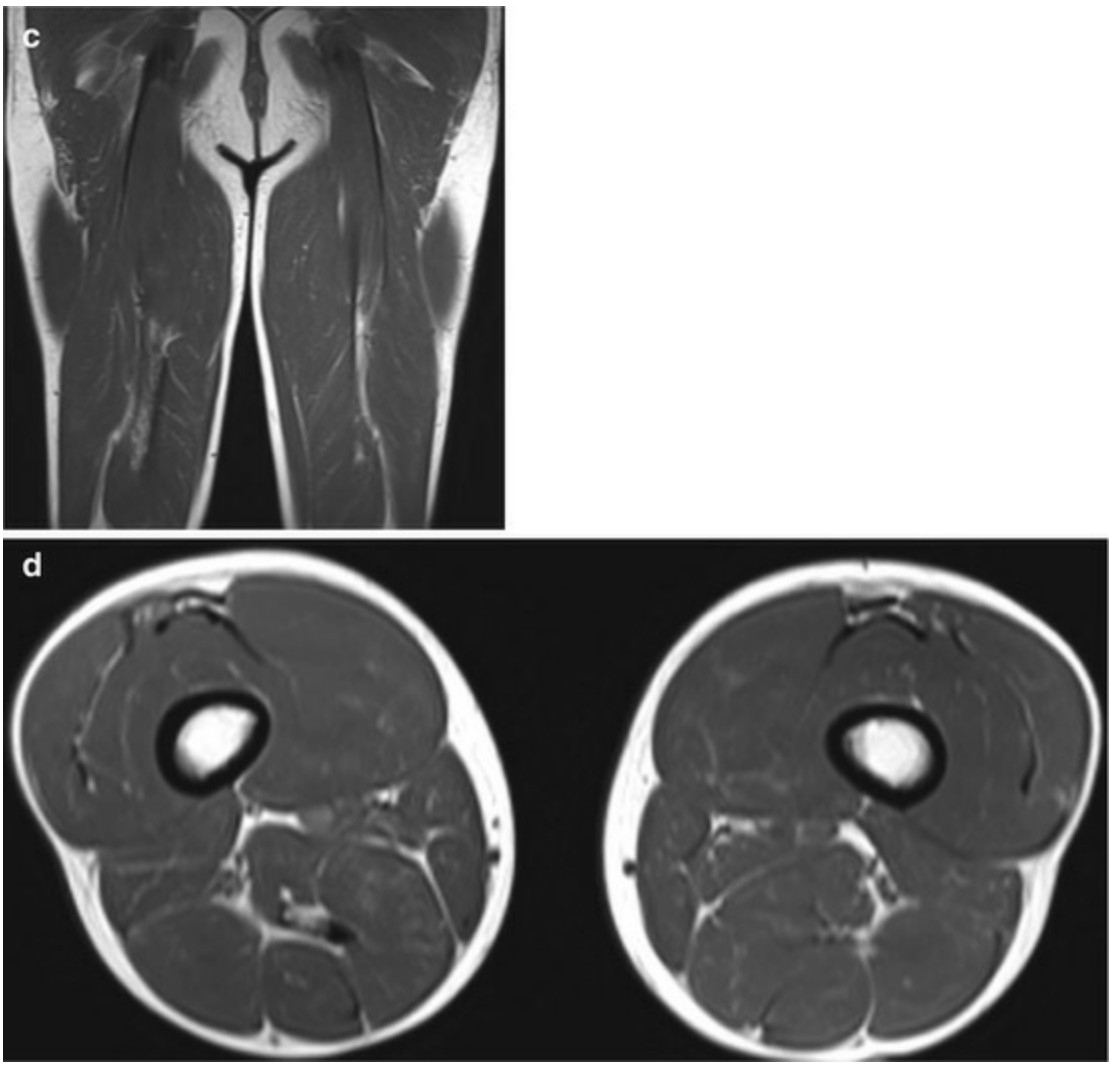

Fig. 17.14 A 30-year-old male professional soccer player with a grade 1 perifascial strain of the left biceps femoris. Feathery interstitial edema is visible within the short head of the biceps femoris on coronal (**a**) and axial (**b**) fat-suppressed proton density-weighted MRI. Note also a longitudinal area of low signal intensity in the right semimembranosus muscle on all pulse sequences, better visualized on coronal (**c**) and axial (**d**) T1-weighted MRI, indicating scar tissue from a previous strain

Scar tissue is usually treated with a conservative stretching program. However, surgical removal is warranted in a certain portion of recalcitrant cases.

17.7.3 Muscle Atrophy

Chronic injuries frequently demonstrate atrophy and fatty replacement of muscles [16, 64]. Muscle atrophy can be extremely rapid, developing 5–10 days after immobilization, and can become irreversible within 4 months [65]. On MRI, it manifests as reduced muscle size with fatty infiltration. Fatty changes appear as diffuse areas of increased signal intensity within the muscle on T1-weighted images (Fig. 17.15) [16, 64]. Comparison with the contralateral extremity is important in evaluating

the relative symmetry of muscle groups [16], particularly in subtle cases.

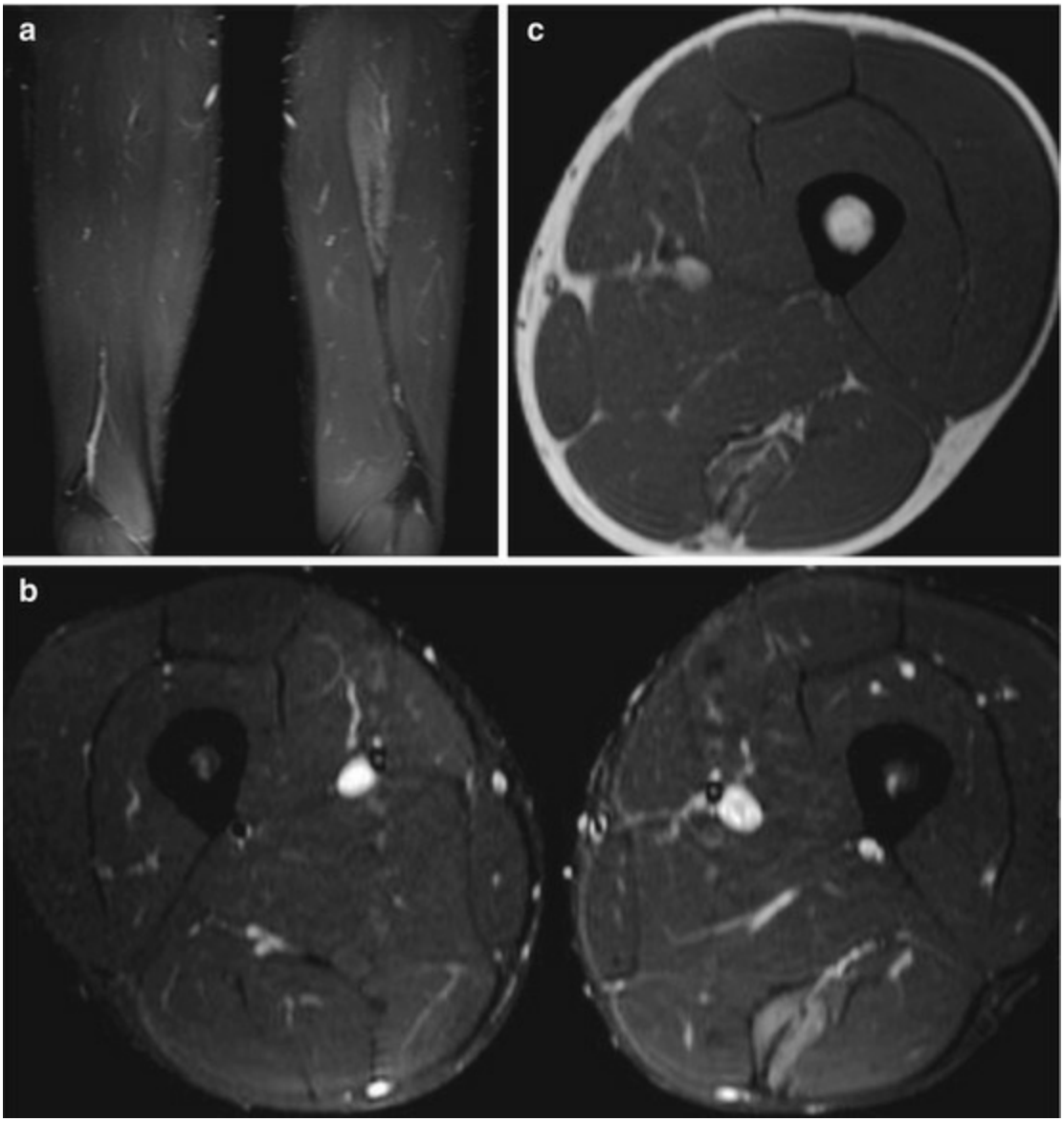

Fig. 17.15 A 19-year-old male professional soccer player with chronic injury of the semitendinosus muscle. Coronal (**a**) and axial (**b**) STIR MRI show hyperintensity and atrophy of the semitendinosus muscle with a longitudinal, hypointense scar. Axial T1-weighted MRI (**c**) reveals fatty infiltration, indicating an old injury

Fatty infiltration occurs in the late stages of many pathological conditions involving skeletal muscles [66]. Muscle atrophy and fatty infiltration can also be caused by idiopathic myopathies, denervated muscles, neurogenic disorder [67, 68], chronic disuse as a late finding after severe muscle injury or chronic tendon tear, and corticosteroid use.

Complete rupture of the MTJ, on the other hand, selectively involves one muscle or conjoined attached muscles and can be diagnosed on the basis of atrophy and fatty changes, as well as the inability to identify the distal musculotendinous attachment, on

MRI. Potential clues to this diagnosis include the identification of tendon interruption and retraction of the muscle belly.

17.8 Treatment

The treatment of proximal lesions is conservative in most cases, consisting of rest, ice, and physical therapy. Complete rehabilitation usually requires about 6–8 weeks. Surgical treatment is indicated in cases of proximal avulsion, with or without an ischial bone fragment. The whole block is set with anchors and sutures or soft-tissue sutures. Modern treatment techniques, such as cell therapy with platelet-rich plasma, are currently available, but remain controversial. However, this procedure has shown promising results in some studies and in our experience, and can be considered an adjuvant treatment of high lesions in the posterior muscles of the thigh (Fig. 17.4).

Conservative treatment of muscle injuries with rest, physical therapy, and cryotherapy is usually sufficient. The adjuvant use of platelet-rich plasma has been reported to reduce recovery time by 30–50 % and decrease recurrence, with better healing quality. The gradual return to normal physical activity requires additional muscle strengthening to regain full competitive capacity. Surgical treatment of such injuries is rarely indicated, but attention must be paid to the potential for hematoma formation, which may require ultrasound-guided percutaneous drainage, and compartment syndrome, which is uncommon in this region.

A distal biceps femoris injury characterized by avulsion involving a fragment of the fibular head requires fixation with screws and, in some cases, surgical exploration of the peroneal nerve branch, which is located anatomically in this region.

In summary, the treatment of HMC injuries is based on rest, physical therapy, and analgesic administration. Functional recovery is achieved with strengthening and stretching exercises in preparation for the resumption of sports performance. Good nutrition and the reestablishment of a balance between muscle groups are also important. Surgical treatment is restricted to specific cases, usually involving bone and tendon avulsions. The isokinetic evaluation of elite athletes allows them to resume competitive activity with confidence because it provides accurate information about the status of the muscle group.

17.9 Conclusion

HMC lesions are among the most frequent injuries in athletes, and their incidence is also increasing in the general population. Clinical evaluation is often inconclusive or insufficient, and imaging studies, especially MRI and ultrasound, are often required to further evaluate such injuries. Imaging evaluation was formerly limited to professional and elite athletes, but with increasing sports activity in the general population,

musculoskeletal radiologists must have knowledge of and familiarity with muscle injuries in general and those of the HMC in particular.

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18. Hip Short External Rotator Muscles Injuries

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Abstract
Traumatic injuries to the short external rotator muscles of the hip are rare. They occur mainly in young athletes and are often difficult to diagnose clinically. Moreover, the mechanisms at their origin are quite nonspecific. MRI, however, is very useful for exploring and assessing these and related injuries. This examination is essential to assessment of the injury because several different muscles of the pelvic girdle may be damaged. Short external rotator injuries are generally specific to their distal tendon. They are essentially partial tears that can extend to the insertion of the fleshy muscle into the bone. The prognosis for this injury is good with rapid pain relief within several days and a return to sports in nearly all cases.

18.1 Introduction

The short external rotator muscles of the hip are a deep muscle group less well known than the gluteal, iliopsoas, abdominal wall, adductor, and hamstring muscles and are rarely mentioned first when considering the cause of pain, regardless of whether or not its origin was traumatic.

Because of numerous adjacent musculotendinous and nerve structures, this muscle group can also be the source of local non-traumatic pain from impingement. This non-traumatic pain mainly involves the piriformis and the quadratus femoris. It is rare and often difficult to analyze in practice, but it is relatively well known to doctors.

Traumatic injuries of the short external rotators of the hip are still rarer, as demonstrated by the lack of literature on this subject. The symptoms of these injuries are nonspecific and thus difficult to analyze.

Beyond the rare cases of direct impact traumas, most of the cases we have encountered occur in young, high-level or elite athletes, who practice their sport regularly. These injuries often occur during periods of intense sports activity or training for a competitive event.

In our experience, a quarter of these injuries are associated with injury to other muscles (e.g., rectus femoris, iliopsoas, great adductor, and hamstrings). Similarly, concomitant damage to several of the short external rotator muscles of the hip is possible but infrequent (approximately one case in 8 in our series).

These muscles are explored mainly by MRI. Standard radiography is used for the immediate post-trauma assessment to rule out bone injury. Ultrasound is generally not especially useful and is very often limited by the patient's body morphotype. Moreover, the deep anatomical location of these muscles makes them difficult to access, for part of the muscle structure is hidden by pieces of bone.

Traumatic injuries to these muscles are seen by morphologic and, especially, signal abnormalities of T2-weighted, fat-suppressed MRI sequences. They show hyperintense edematous areas, sometimes with small fluid collections that may correspond to small hematomas.

The anatomical injuries encountered most often are multiple tears or localized partial tendon avulsion. We have never observed a complete rupture or avulsion.

As a general rule, the course of these injuries is fast and favorable, with functional discomfort that diminishes very quickly, within hours or days. Physical and sports activity resumes within several days of the acute phase and without sequelae. Most of the time, therefore, the prognosis of these injuries is good.

This chapter will review the different short external rotator muscles of the hip and look at examples of traumatic injuries, without considering chronic pain syndromes. We will discuss some particularities of the morphology, context of occurrence, and symptoms of these injuries.

18.2 Short External Rotator Muscles of the Hip: Anatomy

There are six of these short external rotator muscles of the hip, situated deep on the dorsal side of the coxofemoral joint [1, 2].

Their insertions make it possible to identify two subgroups:

- two short external rotator muscles originating within the pelvis:
  - the piriformis
  - and the internal obturator

- four short external rotator muscles originating outside the pelvis:
  - the gemellus muscles
  - the quadratus femoris
  - and the external obturator

All these muscles originate at different points in or around the pelvis, but all attach into the greater trochanter.

18.2.1 The Piriformis

Origin: it starts on the anterior surface of the sacrum (vertebrae S2–S4) from fleshy digitations surrounding the ventral sacral foramina (Fig. 18.1).

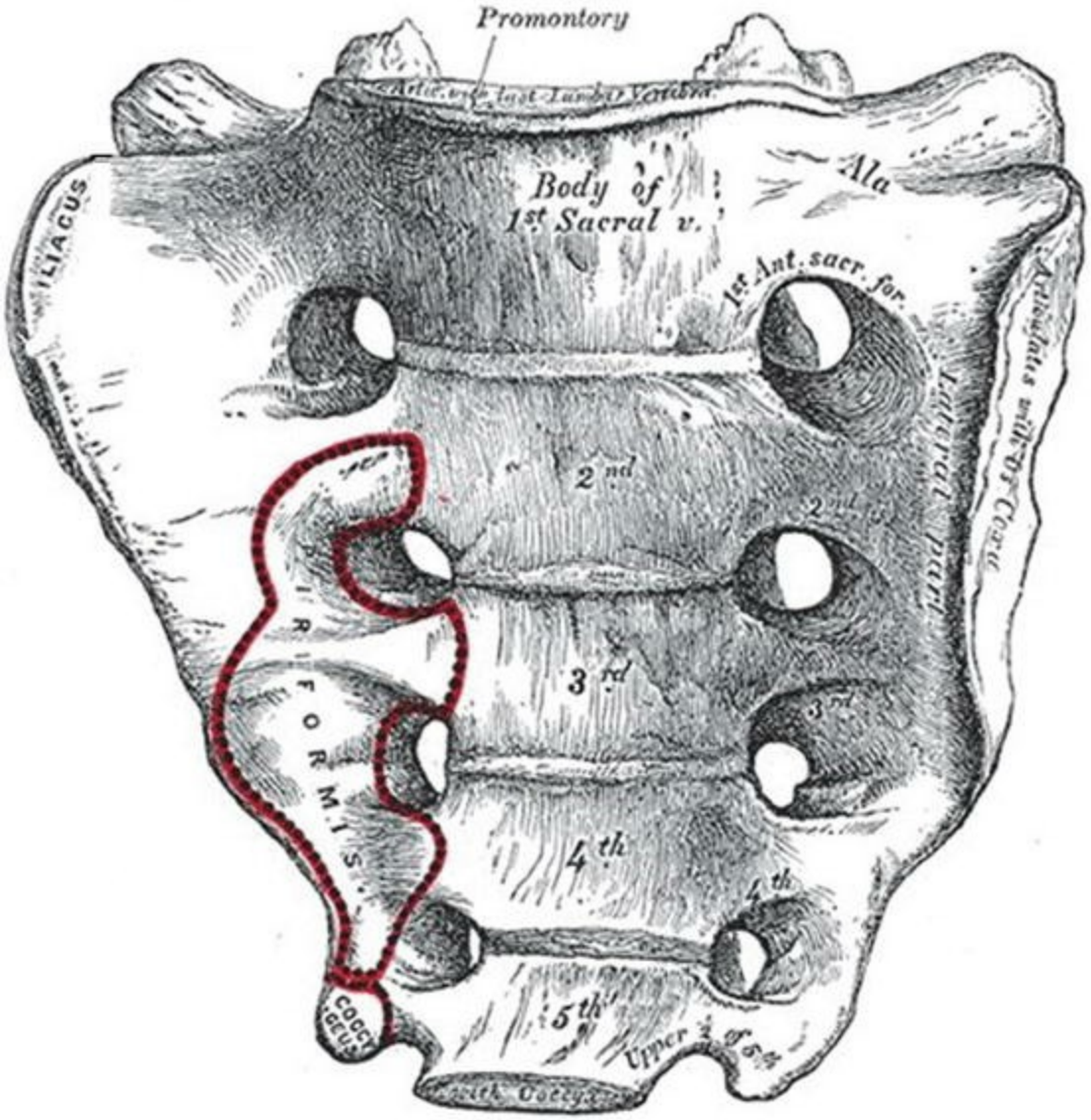

Fig. 18.1 Anterior view of the sacrum with the insertion site of the piriformis muscle

Course: The body of the piriformis is triangular with a medially oriented base. It is disposed transversally, pressed against the ventral surface of the sacrum (Fig. 18.2 and 18.3). At its origin, its fibers are in contact with the roots of the sacral plexus. It leaves the true pelvis through the greater sciatic foramen, thus forming the suprapiriform foramen and the infrapiriform foramen (through which the sciatic nerve usually passes). It then travels transversally and dorsally to the hip joint and inserts into the superior medial surface of the greater trochanter (Fig. 18.4).

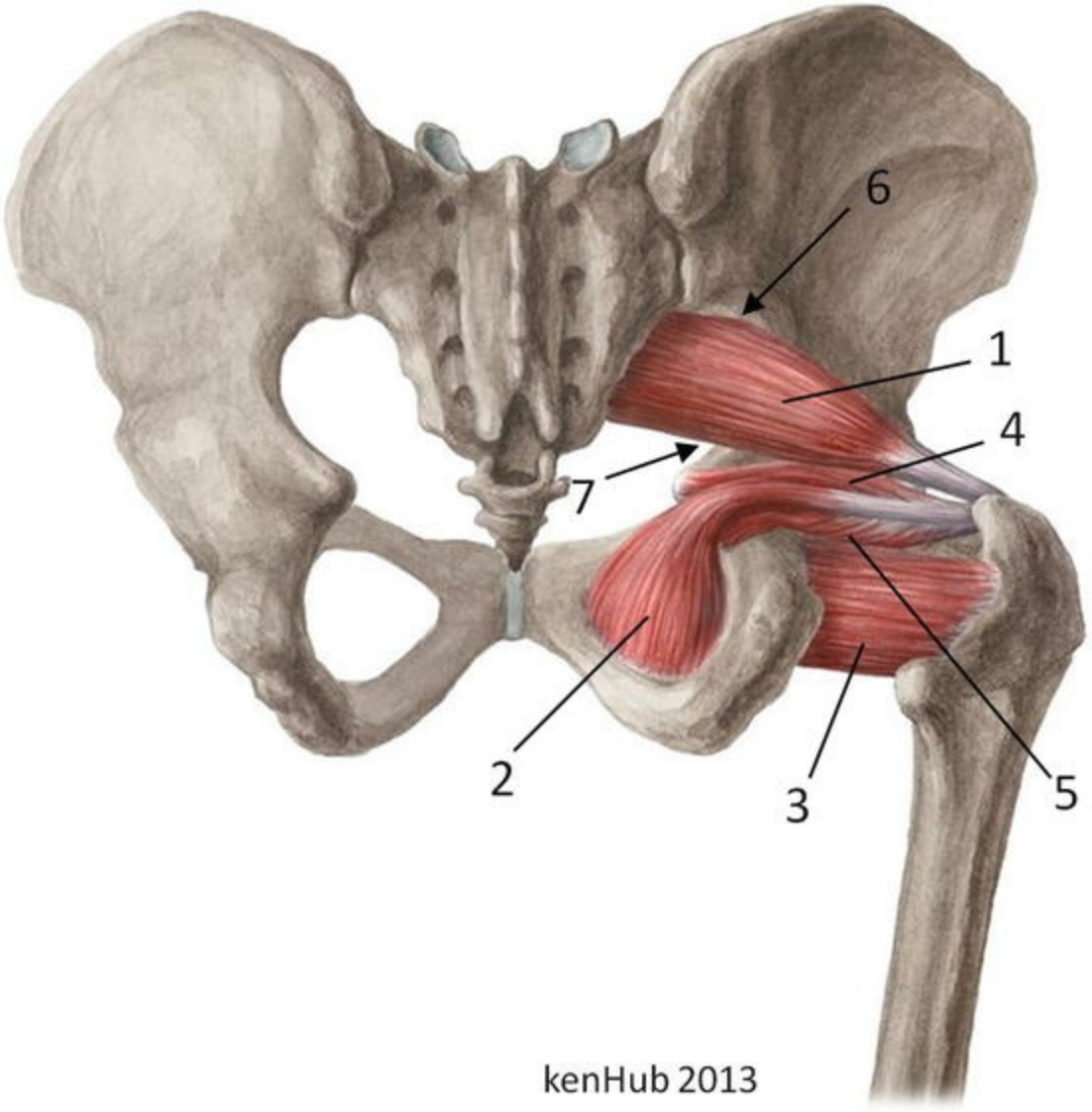

Fig. 18.2 Posterior view of the piriformis (*1*), internal obturator (*2*), quadratus femoris (*3*), and superior (*4*) and inferior gemellus (*5*) muscles. The piriformis is bounded above by the suprapiriform foramen (*6*) and below by the infrapiriform foramen (*7*), through which the sciatic nerve usually passes

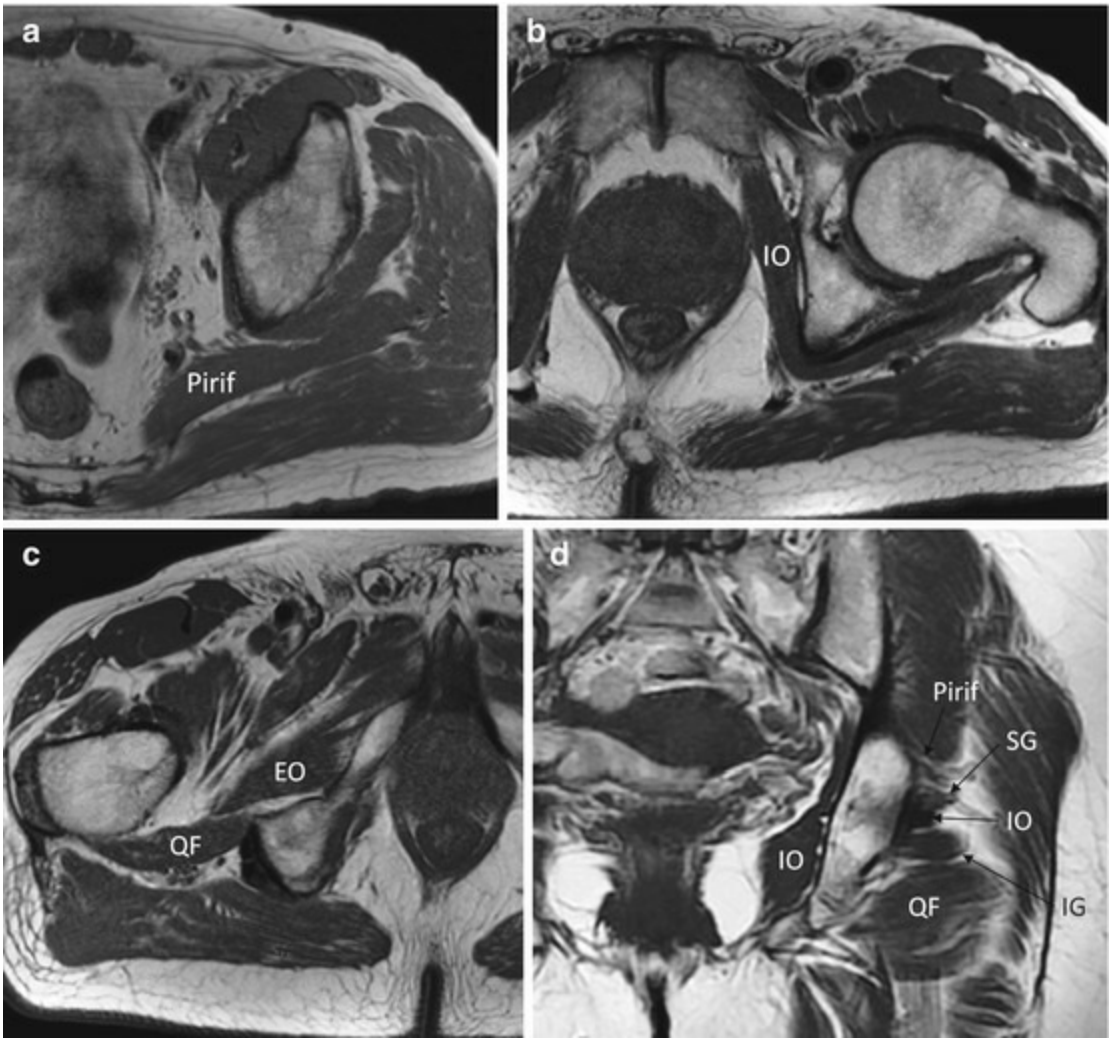

Fig. 18.3 MRI anatomy of the short external rotator muscles of the hip. (**a**) Axial T1-weighted MRI of the piriformis muscle (*Pirif.*), (**b**) the internal obturator (*IO*), (**c**) and the quadratus femoris muscle (*QF*) and external obturator (*EO*), (**d**) Coronal T1-weighted MRI of all the previous muscles (except the external obturator) but also of the inferior and superior gemellus muscles (*IG* and *SG*)

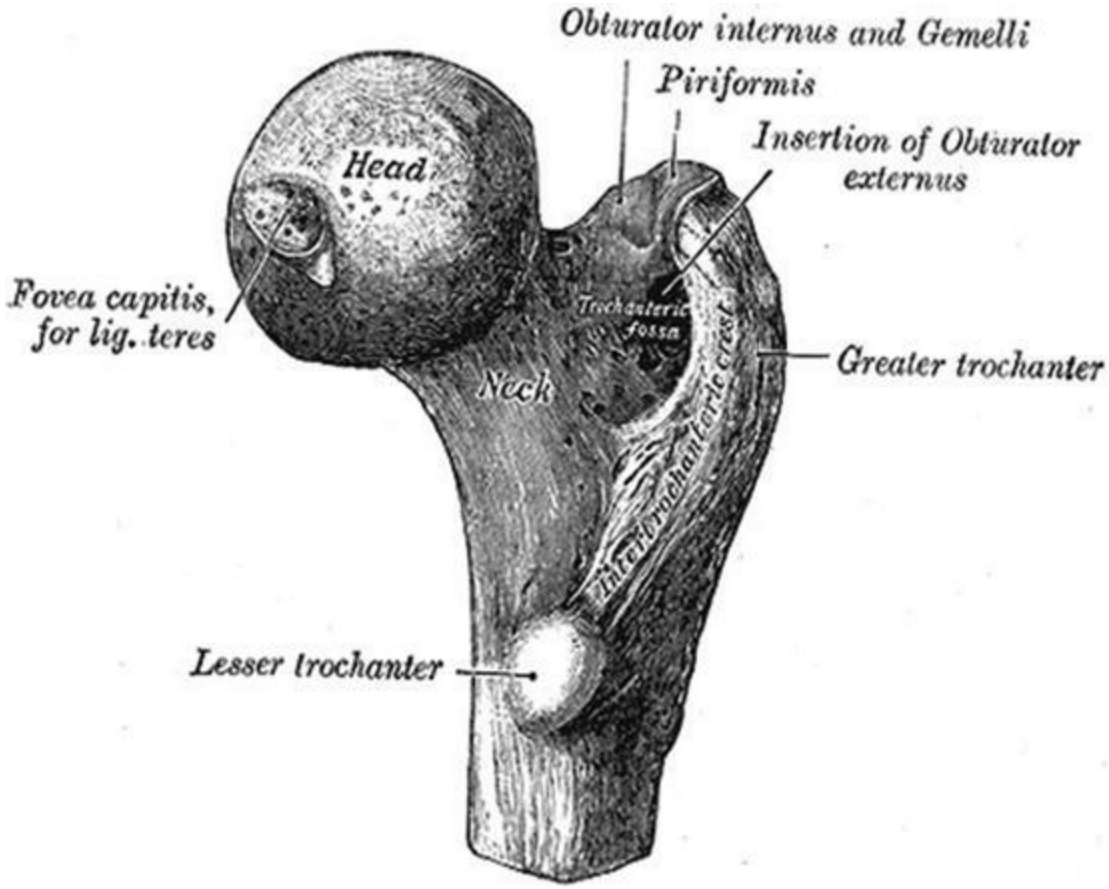

Fig. 18.4 Internal view of the greater trochanter with the insertion sites of the short external rotator muscles

There are numerous anatomical variants around the greater sciatic notch, and these explain some of the impingements with the sciatic nerve and the repercussions of some injuries to it.

Innervation: this muscle is innervated by branches of the sacral plexus (1st and 2nd sacral nerves).

Function: action of external rotation of the leg, but also abduction when the leg is flexed. While walking, it goes through successive phases of contraction and stretching, harmonizing, and synchronizing movement of the sacrum relative to the iliac bone, thus preventing overuse of the sacroiliac joints.

18.2.2 The Quadratus Femoris

Origin: it arises at the lateral surface of the ischial tuberosity of the hip bone (Figs. 18.2 and 18.5).

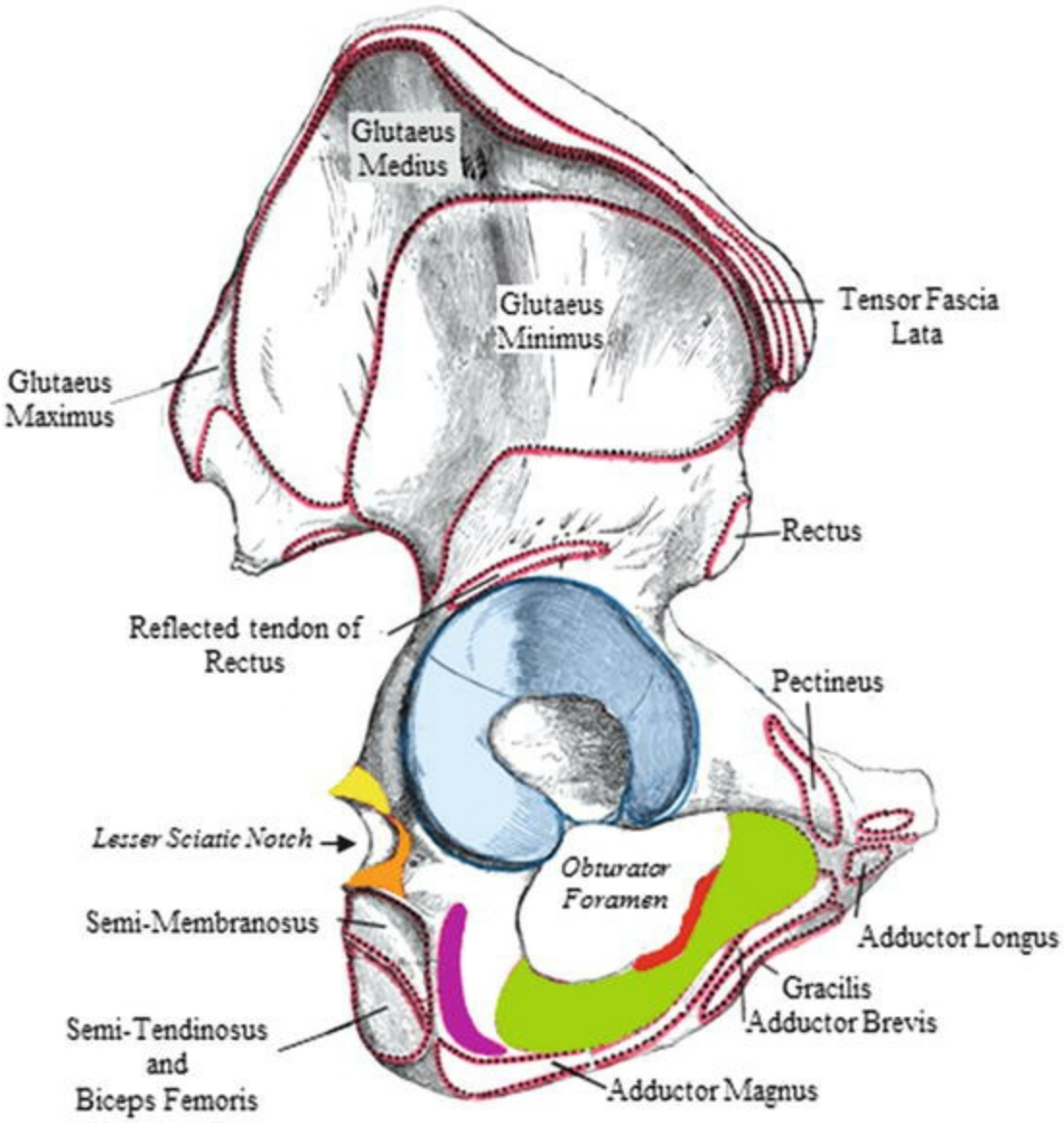

Fig. 18.5 External view of the coxal bone and the insertion sites of the quadratus femoris muscle (*violet*), superior (*yellow*) and inferior (*orange*) gemellus muscles, and the external obturator muscle (*green*)

Course: quadrilateral-shaped muscle placed transversely and horizontally (Fig. 18.3).

Insertion: it is inserted into the quadrate tubercle of the intertrochanteric crest (Fig. 18.4).

Innervation: Nerve to the quadratus femoris (collateral of the sacral plexus, S1 and S2).

Action: it is an external rotator and accessory adductor.

18.2.3 The External Obturator

Origin: it arises from the outer surface of the hip bone, on the anterior rim of the obturator foramen and external surface of the obturator membrane (Fig. 18.5).

Course: this triangular muscle has a medially oriented base and passes obliquely by the inferior and then posterior surfaces of the femoral neck, then dorsally in the external obturator groove, which is marked obliquely on the posterior and then postero-supero-external surfaces of the femoral neck (Figs. 18.2, 18.3, and 18.6).

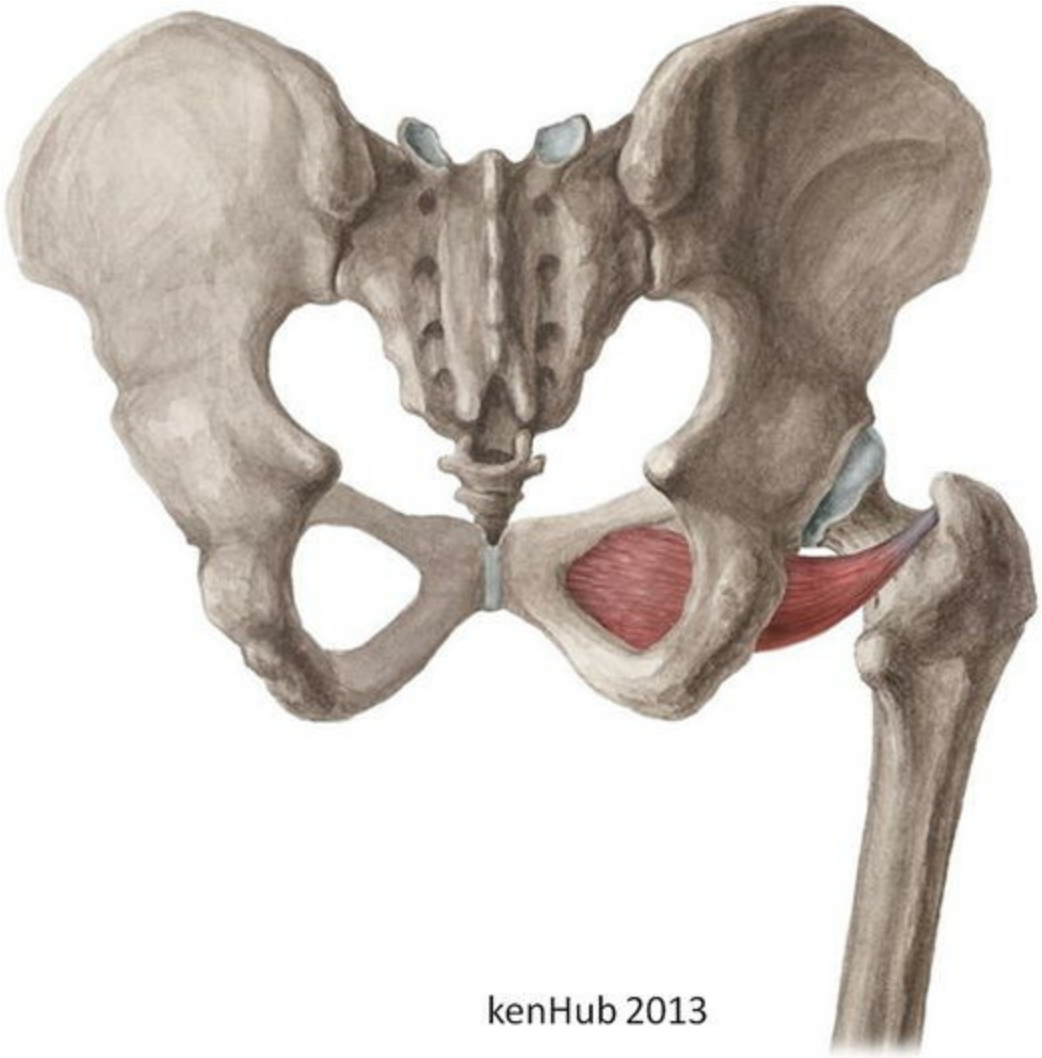

Fig. 18.6 Posterior view of the external obturator muscle

Insertion: it arrives at the trochanteric fossa, where it inserts into the digital fossa (Fig. 18.4).

Innervation: it is innervated by the obturator nerve, the terminal branch of the lumbar plexus.

Action: it is an external rotator and accessory adductor.

18.2.4 The Internal Obturator

Origin: it arises from the internal surface of the hip bone, and more specifically, from the rim of the obturator foramen and the internal surface of the obturator membrane

(Figs. 18.2 and 18.3).

Course: the muscle body is triangular with a base oriented medially and ventrally. Its path is first oblique, dorsal and lateral, until it reaches the lesser sciatic foramen, which it crosses, pressing against the lesser sciatic notch, through a bursa. It then changes direction and becomes transversal, passing to the dorsal surface of the hip joint.

Insertion: it inserts into the medial surface of the greater trochanter, in the trochanteric fossa, ventral to the digital fossa (imprinted above the digital fossa) (Fig. 18.4).

Innervation: it is innervated by the nerve to the internal obturator muscle, a collateral branch of the sacral plexus (S1 and S2).

Action: it is an external rotator and accessory abductor of the thigh in a flexed position at the level of the hip joint.

18.2.5 The Gemellus Muscles

There are two: the superior gemellus and the inferior gemellus. They surround the terminal portion of the internal obturator muscle (Figs. 18.2 and 18.3).

Origins: – superior gemellus: ischial spine (Fig. 18.5).

– inferior gemellus: arises at the superior part of the ischial tuberosity, immediately above and outside of the sacrotuberous ligament (also called the great or posterior sacrosciatic ligament) (Fig. 18.5).

Course: each is on opposites sides and a satellite of the internal obturator muscle, following its course outside the pelvis (Figs. 18.2 and 18.3).

Termination: on the terminal tendon of the internal obturator muscle; they do not have their own terminal insertions.

Innervation: superior gemellus muscle: nerve of the internal obturator muscle
inferior gemellus muscle: nerve of the quadratus femoris muscle

Action: identical to that of the internal obturator muscle.

18.3 Traumatic Injuries to the Piriformis Muscle

The piriformis syndrome has been regularly reported in the literature [3, 4] for many years. It results from an impingement of this muscle with the sciatic nerve, when both anatomical structures are normal but also when they are anatomical variants [5]. On the other hand, traumatic injuries of this muscle appear to be reported only sparsely in the literature [6].

18.3.1 Injury Mechanism

The most frequent mechanisms of injury: direct contact or a strong, resisted contraction of the muscle during a movement of sometimes limited amplitude but using a great deal

of energy.

- internal rotation: repeated sudden movements and/or sudden tensing,
- adduction: sudden adduction movement, with the thigh flexed relative to the pelvis.

Traumatic injuries affect the muscle belly, which has a T2-weighted hyperintense signal on MRI (Fig. 18.7) — more or less intense, with more or less substantial disorganization of the muscle fibers depending on the extent of the tears. These images are nonetheless not specific.

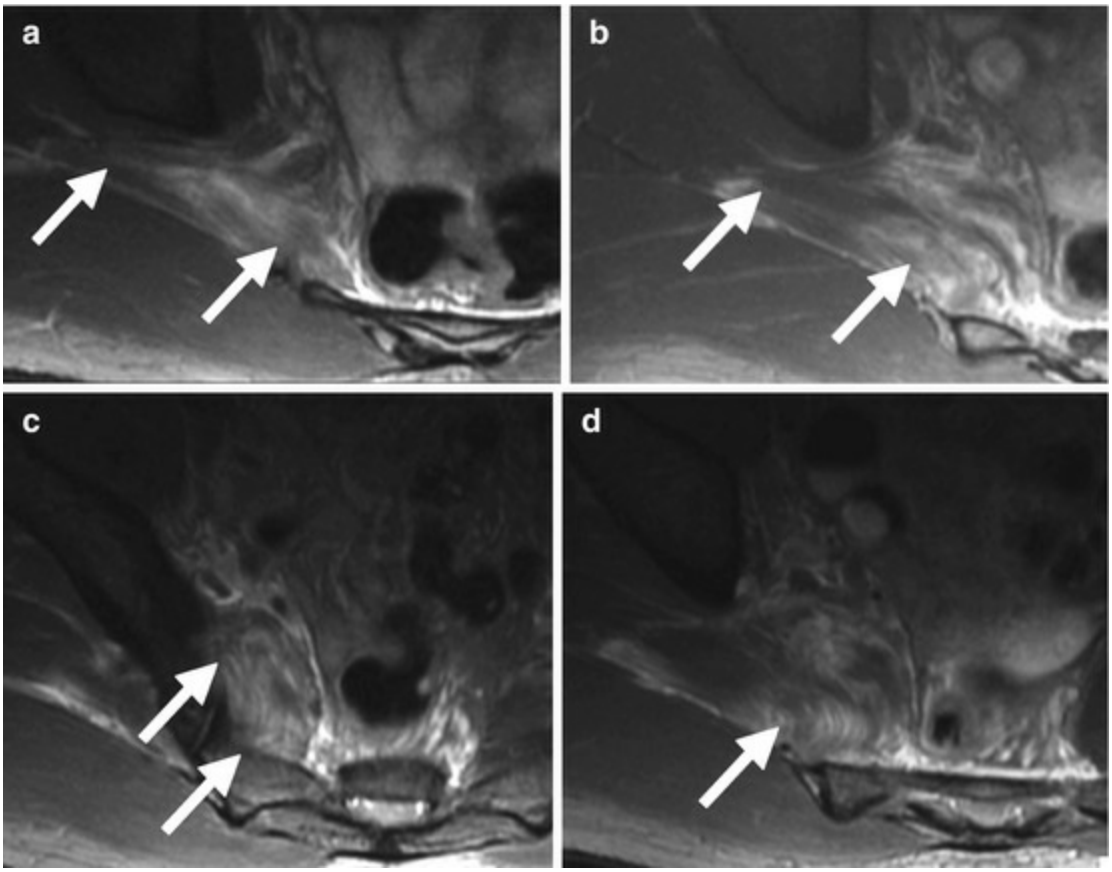

Fig. 18.7 A 25-year-old professional rugby player with a traumatic piriformis muscle injury. Axial T2-weighted fat-suppressed MRI at different levels (**a**–**d**) of the piriformis muscle (*arrows*) showing an area of hyperintense focus in the piriformis muscle belly with partial disorganization of the muscle fibers (Reproduced with the permission of Drs Alain Silvestre and Philippe Meyer, Center of Osteoarticular Imaging, Sports Clinic, Mérignac, France)

18.3.2 Traumatizing Activities

Bicycling: the problems are related to the positions cyclists use to try to improve hamstring efficiency (by riding "off the saddle" or "on the nose"). The settings used on the bicycle — especially high gears — can increase constraints on the piriformis. This piriformis pain is associated with pudendal neuralgia in 30 % of cases.

Running: the natural attitude of the legs in medial rotation during forward steps

leads to reflex contraction of the pirformis in eccentric work to maintain the leg in neutral rotation. The repetition of this phenomenon at each step can cause overuse and injury.

Asymmetric sports: these require positions in which the leg is in lateral rotation, either repeated or permanent. They lead to overuse of these short external rotator muscles, principally the piriformis (e.g., hockey, tennis, fencing).

These activities tend to lead to compensatory hypertrophy of the piriformis, especially when the patient already has morphostatic disorders (such as limb length discrepancy [7], overpronation, hip flexion contracture, or hyperlordosis).

This hypertrophy causes discomfort and local impingement although it is not strictly a traumatic injury of the muscle. Such traumatic injury occurs in a wide variety of contexts that have in common the sudden tensing of the muscle according to one of the mechanisms mentioned above.

18.3.3 Clinical Signs

The clinical signs described hereafter are those we generally look for in piriformis syndrome. In cases of traumatic muscle injury, their expression may of course be incomplete and much more rudimentary. Depending on the repercussions of the traumatic injury on adjacent structures and especially the sciatic nerve, this clinical picture may be modified by the association of neurological signs [3].

Inspection

With the subject standing or in dorsal decubitus position, the lower limb is seen to be rotated externally on the affected side.

Palpation

During examination, with the patient in lateral or ventral decubitus position, one can find:

- Pain on palpation of the muscle midway between the greater trochanter and the lateral edge of the sacrum
- Possible induration of the muscle cord, corresponding to a muscle contracture
- Pain has been described on pelvic examination, as has dyspareunia (which may be explained by the proximity of the pudendal nerve passing through the infrapiriformis foramen).

It must nonetheless be noted that even in normal subjects, strong palpation of the region of the greater sciatic notch may elicit sensitivity or even pain without any underlying anomaly.

Mobilization (Fig. 18.8)

- Pain on internal rotation of the hip
- Reduced internal rotation of the hip
- Pain and limitation of the internal rotation or extension of the hip
- The Pace and Nagle maneuvers:
- seated position
- resisted hip abduction
- Beatty maneuver:
- Lateral lying position on asymptomatic side
- Foot of injured limb behind the popliteal fossa of the uninjured limb
- Hip flexed, adduction-internal rotation
- Raise and maintain knee in this position with or without resistance

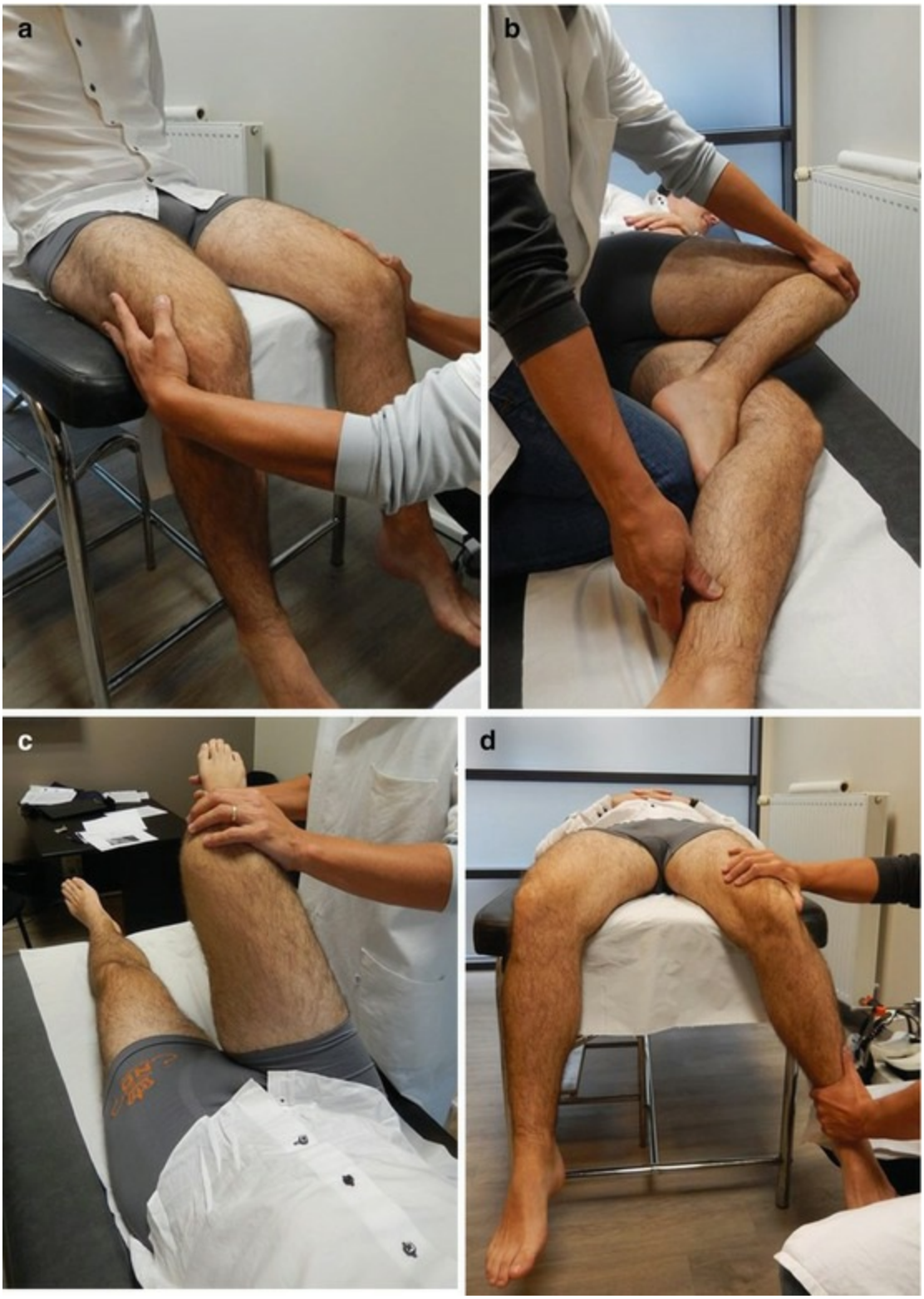

Fig. 18.8 Examples of maneuvers for clinical exploration of a piriformis muscle injury. (**a**) The Pace and Nagle maneuvers: patient in seated position, with a resisted hip abduction. (**b**) Beatty maneuver: patient in lateral lying position, injured side up, superior foot behind inferior popliteal fossa of the uninjured limb, and hip in a position of flexion-adduction-internal rotation. Raise and maintain the knee in this position with or without resistance. (**c**) Freiberg maneuver: patient in dorsal decubitus position. Bring the hip to a position of flexion-adduction-medial rotation. (**d**) Pain and limitation of internal rotation or extension of the hip

18.4 Traumatic Injuries to the Quadratus Femoris Muscle

18.4.1 Circumstances of Onset and Promoting Factors

Two different circumstances are found.

In the first situation, which appears more frequently in our experience, the injury appears to evolve in stages with subacute episodes (that create a true ischiofemoral impingement) until a more marked traumatic episode that causes a significant increase in pain and leads to consultation [8–10]. Compared with injuries of the other short external rotator muscles, these occur more frequently among elite young athletes (17–28 years), or even very young athletes (starting at 12), who train very regularly, sometimes essentially professionally or semiprofessionally. These injuries often occur during intense sports activity or advanced training for competitive events.

On MRI of these athletes, these injuries sometimes appear to be bilateral (approximately 1/3 of the cases in our experience, less in the literature), although the contralateral images are often only slightly symptomatic. This bilaterality tends to suggest an impingement mechanism (specifically, ischiofemoral impingement) rather than pure acute trauma [9].

The literature shows a very substantial predominance of women (6 women to every man) [11]. These young adults often have an acquired or constitutional shortening of the distance between the shorter lesser trochanter and the ischial tuberosity that promotes this impingement [10].

The younger subjects (around 12 years) are often dancers who have recently increased the intensity and rate of their training and physical constraints quite substantially. This results in a mismatch between the muscle volume and the diameter of the ischiofemoral space. Because this diameter increases progressively with growth, the occurrence of an injury of this type does not appear harmful for their future in dance (or other sports activity).

In the second situation, much less frequent in our experience, we find a single traumatic episode in a completely asymptomatic patient, often not at all or only slightly athletic and often older than 40 years. These too are predominantly women, but we have not noticed bilateral injuries in this context.

Some anatomical factors seem to favor this type of injury: a short femoral neck, an open angle between the pubic rami, external rotation of the ischial tuberosities with lateralized hamstring insertion (with or without enthesopathy, which would also result in increasing tendon volume), reducing by the same amount the diameter of the ischiofemoral space.

18.4.2 Injury Mechanism

In the literature and in our experience, these have essentially involved partial muscle

tears (only one case of complete avulsion reported in the literature) of the quadratus femoris muscle, located between the two bone insertion points, in the muscle belly (Fig. 18.9). This muscle belly seems to be crushed between the ischial tuberosity and the lesser trochanter.

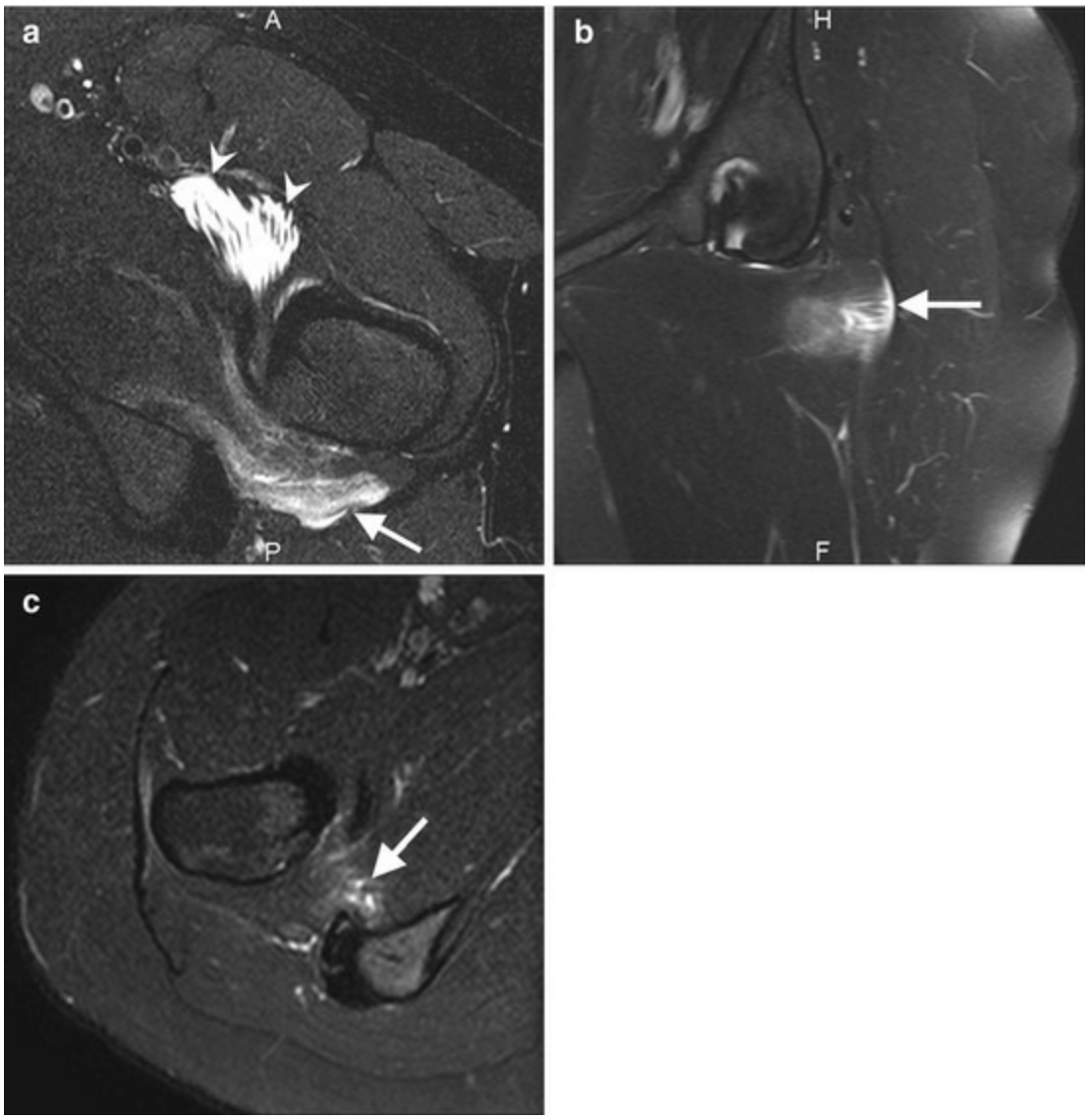

Fig. 18.9 Traumatic injury of the quadratus femoris muscle. A 26-year-old female dancer. (**a**) Axial and (**b**) coronal fat-suppressed T2-weighted MRI show a frank partial tear (*white arrow*). There is also a partial tear of the iliopsoas muscle (*white arrowheads* on **a**). (**c**) A 23-year-old female dancer. Axial fat-suppressed T2-weighted MRI shows a moderate partial tear (*white arrow*)

Two types of movement appear to be involved:

- a movement of pure internal rotation of the leg, causing a rupture by abrupt extension of the muscle.
- hyperabduction of the flexed thigh (e.g., falling while performing a frontal split), also leading to abrupt extension of the muscle.

- a movement of external rotation with a slight adduction, causing reduction of the ischial tunnel and a more or less acute hamstring impingement.

18.4.3 Traumatic Activities

The circumstances promoting traumatic mechanisms occur most frequently during some sports activities (such as dancing, fencing, martial arts, tennis, and badminton) in which rapid (or abrupt) movements of limb rotation are frequent. These circumstances also occur during some forced or resisted movements outside of sports. Accordingly, we have seen a quadratus femoris injury in a 50-year-old woman after several forceful efforts to pull the rope starter of her lawn mower.

18.4.4 Clinical Signs

On inspection, the pain is situated in the inferior portion of the buttocks, with little irradiation. It is sometimes possible to show sensation of abrupt jerking during a voluntary weight-bearing movement combining internal rotation of the lower limb and a slight pelvic tilt that reduces the diameter of the ischiofemoral space.

Clinical examination finds indeterminate and nonspecific pain in the buttocks during resisted external rotation of the leg.

In the case of marked injury to the quadratus femoris muscle, sciatic nerve irritation can cause irradiating sciatica-type pain [12].

18.5 Traumatic Injuries to the Internal and External Obturator and Inferior and Superior Gemellus Muscles

Traumatic injuries to these muscles appear to be rare, as shown by the sparseness of the literature about obturator muscle injuries [13, 14] and the absence of reports about gemellus muscle trauma. MRI shows nothing specific compared with other muscle injuries; there is still an area of T2-weighted hyperintense focus (Fig. 18.10a, b), the extent and intensity of which depend on the severity of the tear (Fig. 18.10c).

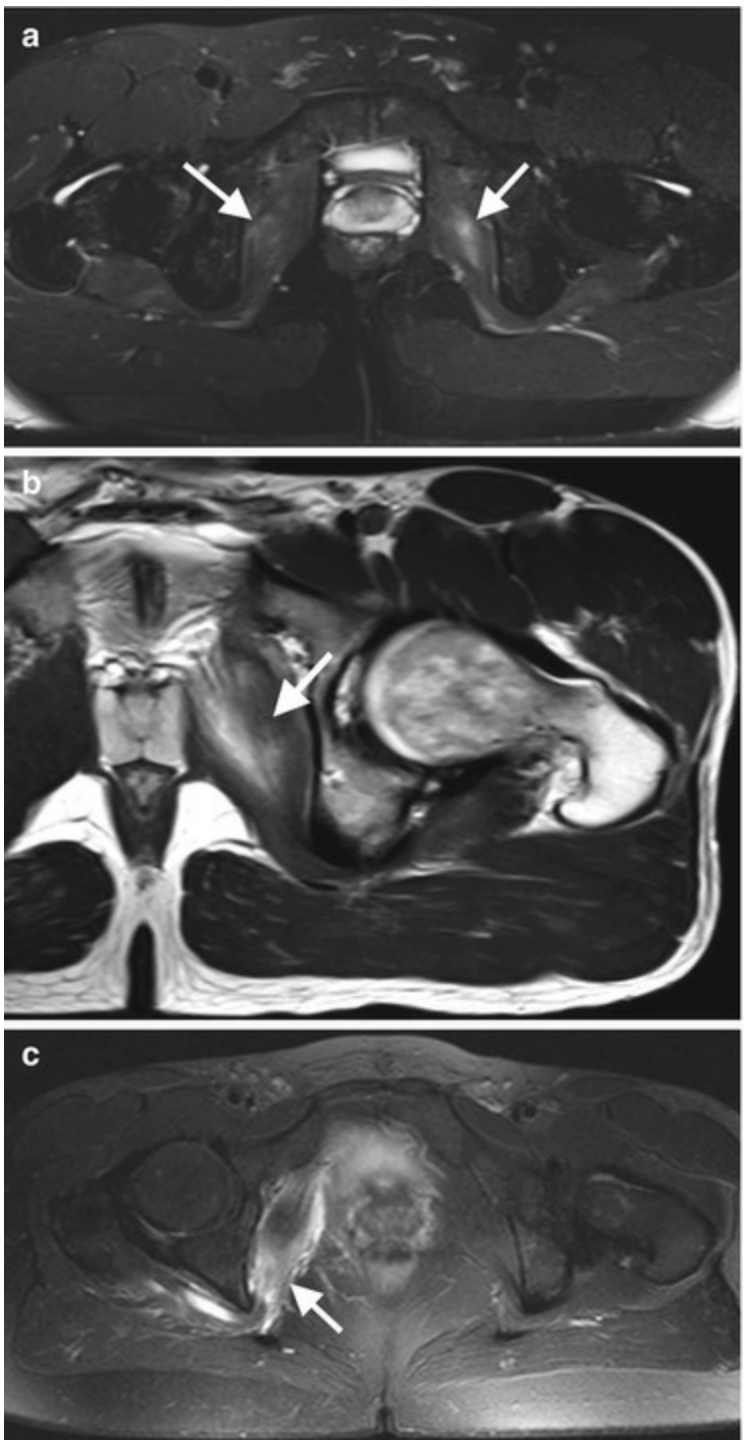

Fig. 18.10 Traumatic injury to the internal obturator muscle. (**a**) A 20-year-old man. Axial fat-suppressed T2-weighted MRI shows bilateral partial tears of the internal obturator muscle (*white arrow*), slight on the right and more pronounced on the left. (**b**) A 22-year-old man. Axial T2-weighted MRI shows a partial central tear (*white arrow*) of the left internal obturator muscle. (**c**) A 27-year-old woman. Axial fat-suppressed T2-weighted MRI shows a pronounced partial tear (*white arrow*) of the superior right internal obturator muscle belly

18.5.1 Injury Mechanism

The mechanisms for this injury are not well known but appear to be the same as for the

preceding muscles: forceful resisted muscle contraction during a movement of variable amplitude during the rotation of the trunk over the femur.

- internal rotation: repeated abrupt movements and/or sudden tensing,
- adduction: abrupt adduction movement, with the thigh flexed relative to the pelvis.

One bilateral injury has been reported [14].

18.5.2 Traumatic Activities

Football (soccer) appears to be the principal activity leading to traumatic injuries of the obturator muscles (Fig. 18.11). These injuries as well as those to the gemellus muscles also occur while dancing or playing tennis. This list is not exhaustive because any activity involving forceful rotations of the pelvis and/or lower limbs can occasion this type of injury in some circumstances (our personal series includes one case of a long jumper with such an injury on landing).

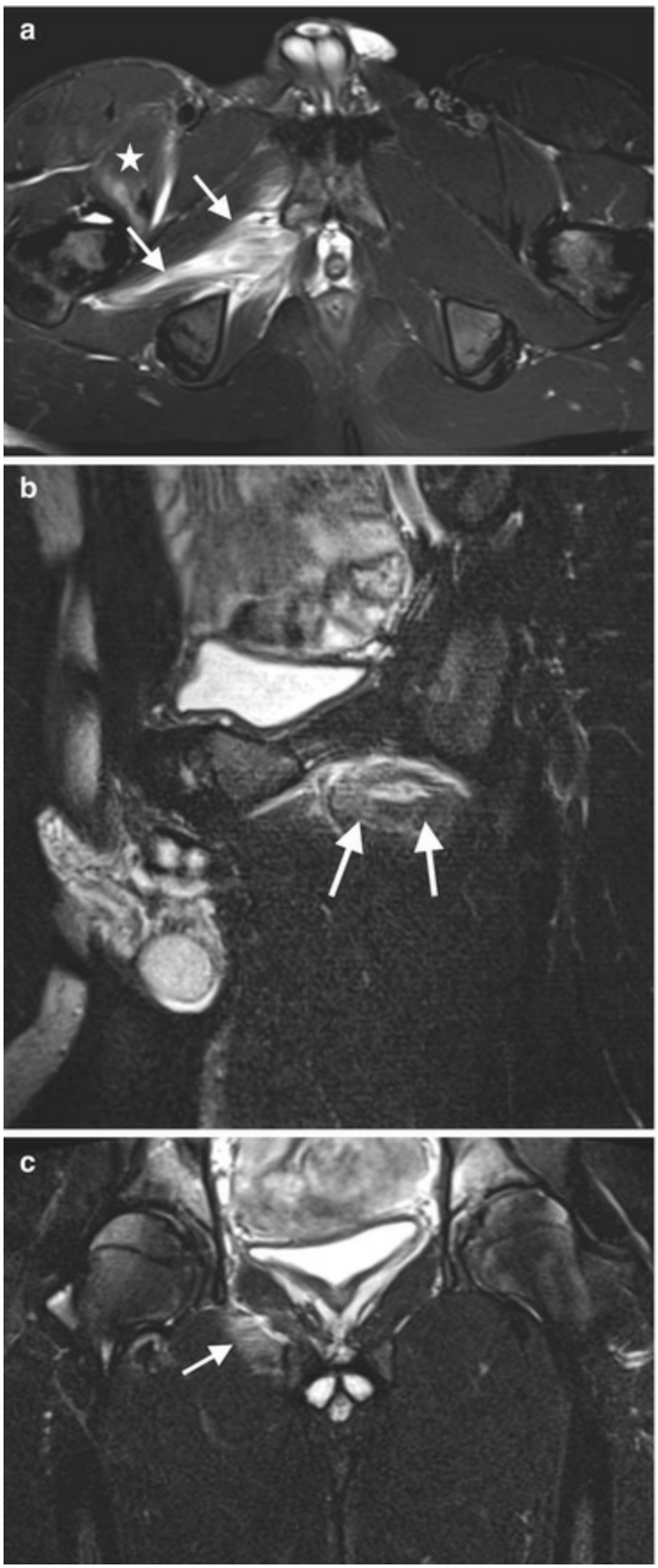

Fig. 18.11 A 24-year-old football (soccer) player with a traumatic injury of the external obturator muscle. (**a**) Axial,

(**b**) sagittal, and (**c**) coronal fat-suppressed T2-weighted MRI show partial tears of the deep aspect of the right external obturator muscle (*white arrow*) associated with mild distal tear of the ipsilateral iliopsoas (*white star* on image **a**)

18.5.3 Clinical Signs

On inspection, indeterminate pain can be noted, projecting from the hip and adductors. Patients can adopt a spontaneous position in external rotation with partial flexion of the thigh, as they can for the piriformis.

On clinical examination, there is most often no pain on palpation at any point, but pain can be provoked on mobilization during movements:

- of active, resisted, and passive external and internal rotation (all muscles)
- of resisted abduction when the thigh is flexed (internal obturator and gemellus)
- active and resisted adduction (external obturator)

Deep pain is possible during digital vaginal and rectal examination in cases of injuries to the internal obturator.

Sometimes, pain is found in the region of the pudendal nerve when irritated during a superficial injury of the internal obturator muscle. The literature also contains several cases of sciatica associated with injuries of the internal obturator [15].

18.6 Differential Diagnosis

The clinical signs of traumatic injuries to the short external rotator muscles of the hip are nonspecific. Because these injuries are rare, their discovery is more of a diagnosis of exclusion than a working diagnosis.

Among the injuries that could cause very similar symptoms are (non-exhaustive list):

- posterior acetabular lip injury
- posterior bursitis
- sciatica truncated by standard nerve root impingement or other neurologic problem [16–18]
- sacroiliitis
- traumatic injuries of the gluteus medius or maximus.
- injuries of the hamstring tendons
- bone injuries: fracture, fatigue fracture (Fig. 18.12a), tumor (Fig. 18.12b), which can have a misleading onset, mimicking a purely traumatic injury, and an

inflammatory intraosseous bone lesion (Fig. 18.12c).

- injuries of the short external rotator muscles of the hip, caused neither by trauma nor impingement: infectious myositis [19] or tumor (benign or malignant).

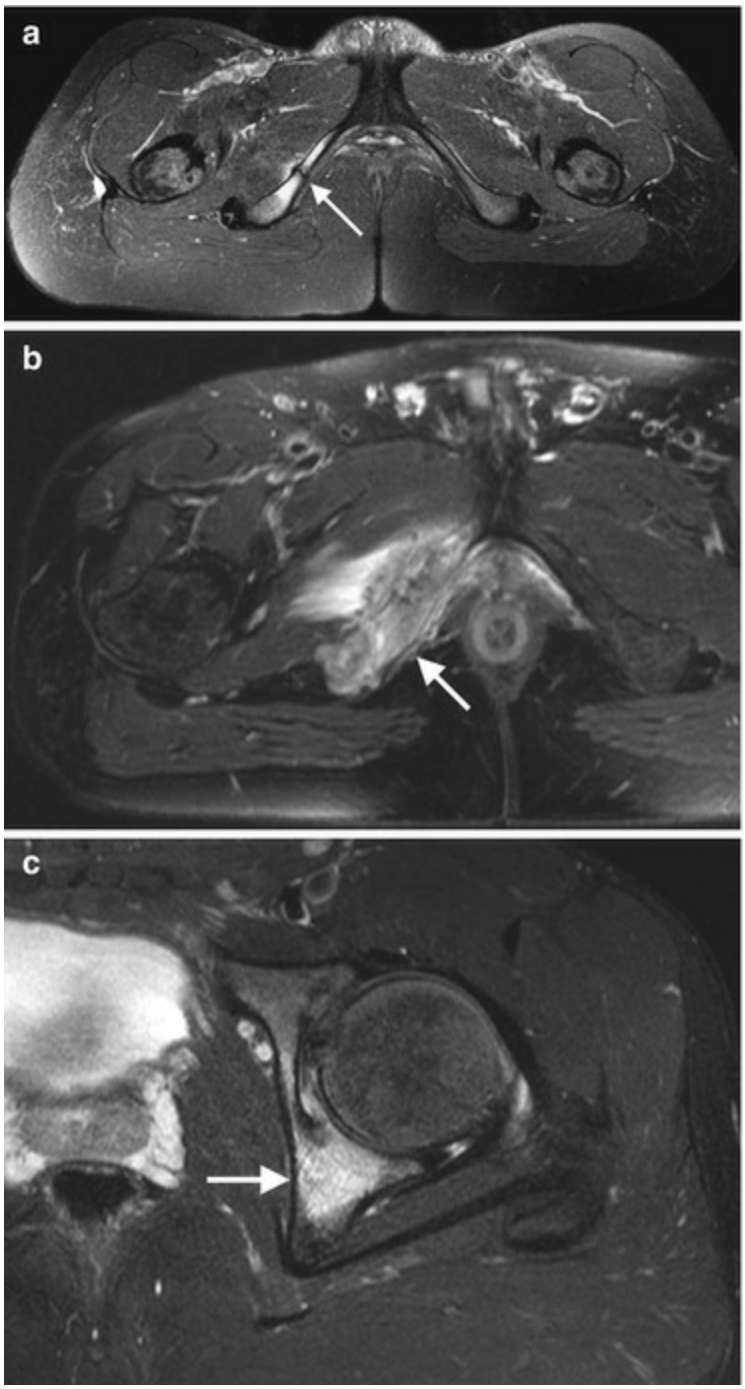

Fig. 18.12 Examples of differential diagnoses of pain that may suggest damage to a short external rotator muscle of the hip on these axial fat-suppressed T2-weighted MRI. (**a**) A 34-year-old woman triathlete with onset of pain during competition. Fatigue fracture of the right ischiopubic ramus (*white arrow*). (**b**) A 45-year-old male runner, with a pathological fracture of the ischiopubic ramus (*white arrow*) on a metastasis of prostate cancer. (**c**) A 17-year-old football (soccer) player with aseptic acetabular osteomyelitis (*white arrow*)

An imaging work-up is therefore recommended for all pain in the buttocks region that appears to be of traumatic origin and does not resolve spontaneously in the days that follow.

18.7 Conclusion

In this chapter, we described the different short external rotator muscles of the hip and showed examples of traumatic injuries and discussed some particularities of the morphology, context of occurrence, and symptoms of these injuries. Traumatic injuries to the short external rotator muscles of the hip occur mainly in young athletes and are often difficult to diagnose clinically. Moreover, the injury mechanisms at their origin are quite nonspecific and differential diagnoses are broad. MRI is an essential tool for the injury assessment because several different muscles of the pelvic girdle may be damaged simultaneously.

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19. Gluteus Maximus and Surrounding Muscles Injuries

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Abstract
Although rare, sports related injuries to the gluteus maximus (GM) and adjacent structures can cause pain and disability in athletes. Trauma, impingement with adjacent bony structures and overuse are among the most common etiologies for pathologic processes at this region. MRI is the modality of choice for imaging of gluteal region, which has a complex anatomy and physiology. Multiple pathologic processes take place at the GM and surroundings and knowledge of the imaging aspects of each of these conditions is important for patient management. In this chapter, after a brief review of key anatomical and physiologic aspects, the imaging findings of various sport related injuries of the gluteal region are presented.

19.1 Introduction

Although rare, sports related injuries to the gluteus maximus (GM) and adjacent structures can cause important pain and disability in athletes both professional and recreational. Trauma, impingement with adjacent bony structures and overuse are among the most common etiologies for pathologic processes at this region.

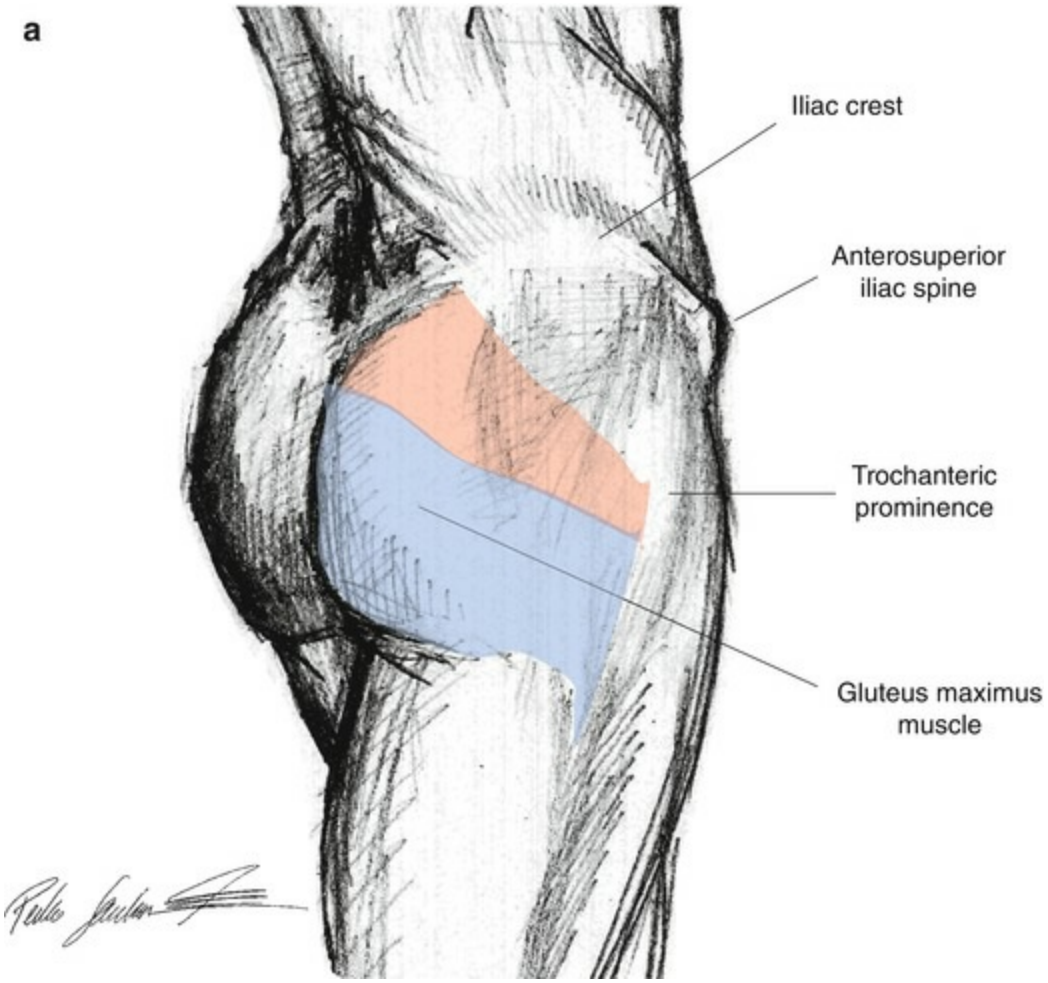
Imaging is paramount for the etiological diagnosis of musculoskeletal pathology at this region. MRI is the most commonly used method for this evaluation due to its exquisite soft tissue contrast and ability to demonstrate in detail the anatomy of the GM and its surroundings. The imaging diagnosis of gluteal pathology is not always easy

since some conditions are dynamic in nature and may have a nonspecific imaging appearance. In this chapter, after a brief review of key anatomical and physiologic aspects, the imaging aspects of various sport related diseases of the gluteal region are presented.

19.2 Anatomy and Physiology [1, 2]

The gluteal region is tightly enveloped by the deep muscular fascia of the lower limb, which encloses three sub-compartments: The fascia lata tensor compartment, the gluteus medius and minimus compartment and finally the gluteus maximus compartment [3]. The GM is a large quadrilateral muscle that alone covers the entire gluteal region. It has a coarse fasciculated structure, is irrigated by the superior and inferior gluteal arteries and is innervated by the inferior gluteal nerve (L5, S1, S2).

Two functional units with different origins can be described for this muscle. The superior portion originates at the posterior part of the iliac fossa; above the center of rotation of the hip it functions as a primary hip abductor. The inferior portion of the GM muscle arises from the inferior sacrum, sacroiliac and iliotuberal ligaments; below the center of rotation of the hip it acts as a hip extensor [4]. The GM also has a dual insertion: A deep insertion at the lateral branch of the trifurcation of the linea aspera (gluteal tuberosity) and a superficial insertion to the ilio-tibial band (ITB) and fascia lata, which lies over the thick portion of the gluteus medius tendon (Fig. 19.1). This superficial insertion explains the support to the knee extension offered by the gluteus maximus.


a
Iliac crest
Anterosuperior iliac spine
Trochanteric prominence
Gluteus maximus muscle

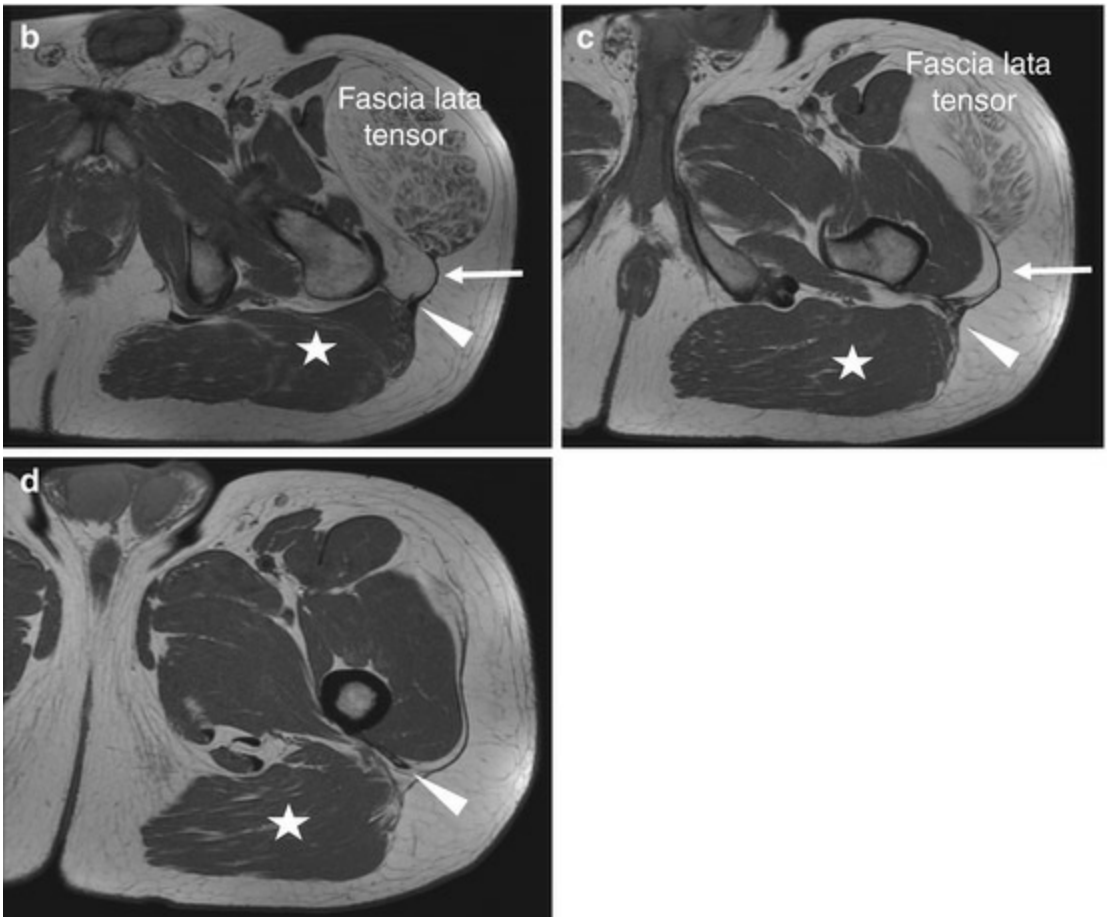


Fig. 19.1 Anatomy of the gluteus maximus. (**a**) Schematic drawing shows the topographic anatomy of the gluteal region. The superior and inferior functional units of the gluteus maximus are depicted in *red* and *blue* respectively. (**b–d**) A 45-year-old man with a soft mass at the proximal thigh. Axial T1-weighted MRI depict the gluteus maximus muscle (*star*) from proximal to distal. An infiltrative lipoma is seen at the fascia lata tensor muscle and at the peri-trochanteric region, facilitating the analysis of the gluteus maximus tendon insertion. (**b**) The superficial insertion of the gluteus maximus to the ITB is seen at the level of the greater trochanter (*arrow*). Note the physiological thickening at the myotendinous junction of the insertion (*arrowhead*). (**c**) Origin of the fibers of the deep insertion of the gluteus maximus (*arrowhead*) at the sub-trochanteric level. The ITB is indicated by the *arrow*. (**d**) Insertion of the deep fibers of the gluteus maximus tendon at the linea aspera of the femur (*arrowhead*)

The so called fascia lata or deep fascia of the inferior limb envelops and is intimately related with the thigh muscles. It originates from the iliac crest, sacrum, coccyx and the superior ilio-pubic branch. The ITB represents a thickening at the lateral portion of the fascia lata that originates at the postero-lateral portion of the superior iliac spine and spans continuously until its insertion at the tibial Gerdy tubercle. During the hip flexion and extension cycle the myotendinous junction of the superficial GM insertion and the ITB run over the greater trochanter of the femur. In full hip flexion it is in its anterior-most position, conversely in extension it is located posteriorly with respect to the greater trochanter. This movement is facilitated by the trochanteric bursa that is interposed between the medius and minimus gluteal tendons and the ITB, covering the lateral and posterior trochanteric facets [5]. The relation of these structures

with the greater femoral trochanter is also influenced by the actions of the muscles surrounding the ITB: the GM, tensor of the fascia lata and quadriceps.

19.3 Traumatic Injuries

19.3.1 Direct Trauma

Due to its superficial position direct trauma to the gluteal region may lead to GM injuries. The most frequent type of lesion found in this context is a muscle contusion, which is characterized by a hyperintensity of the muscle fibers on T2-weighted sequences with irregular margins. The deep muscle fibers are preferentially injured, which helps in the differential diagnosis with muscle sprains which tend to be superficial [6]. GM sprains are rare and the literature is scarce on this subject.

Gluteal region compartment syndromes have also been described following trauma or surgery. Patients usually present with buttock swelling and tenderness. Sciatica is described in about half of the patients. Gluteal compartment syndrome can be caused by intra muscular hematomas or any space-occupying mass originating in any of the gluteal compartments (Fig. 19.2). Additionally, lesions to the superior gluteal artery secondary to acetabular fracture or hip dislocation can also cause a gluteal compartment syndrome [7].

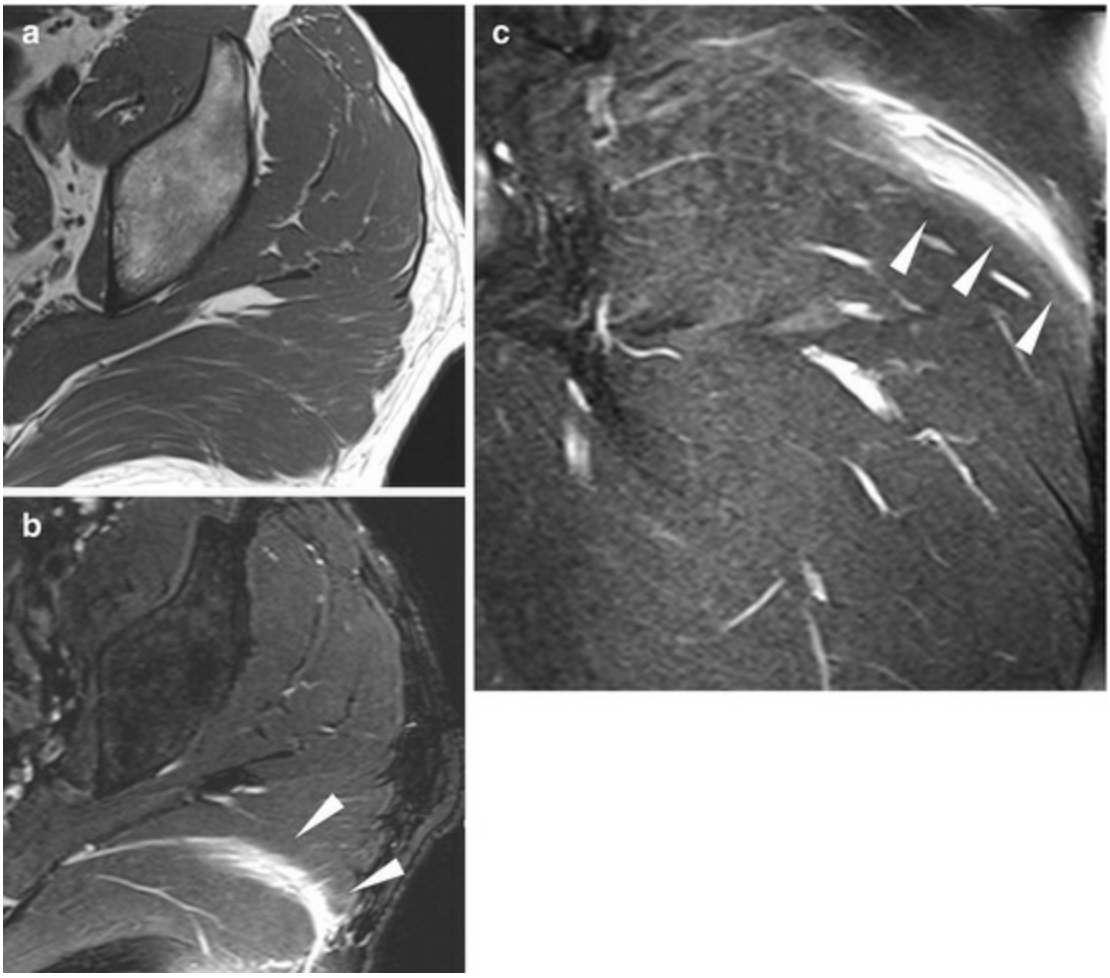


Fig. 19.2 A 30-year-old male athlete with buttock pain after trauma. (**a**) Axial T1-weighted MRI shows no abnormality of the gluteus maximus muscle. (**b**) Axial fat-suppressed T2-weighted MRI demonstrates a crescent shaped hyperintensity of the gluteus maximus muscle, associated with a small intra-muscular fluid collection (*arrowheads*). (**c**) Coronal fat-suppressed T2-weighted MRI demonstrates similar findings and distortion of the muscle fiber architecture (*arrowheads*). These findings are evocative of a grade 2 muscle sprain with intra muscular hematoma of the gluteus maximus

Sustained compression to the gluteal muscles can lead to compressive myopathy, probably related to ischemia [8]. This type of injury can be seen in patients with prolonged immobilization and presents as a signal hyperintensity of the muscular fibers that does not respect anatomic boundaries. It can be seen close to the bony prominences of the hip, such as adjacent to the greater trochanter, where compressive forces are maximal (Fig. 19.3). Compressive myopathy usually causes local pain and discomfort but regresses spontaneously. Compartment syndrome has been described in extensive cases [9].

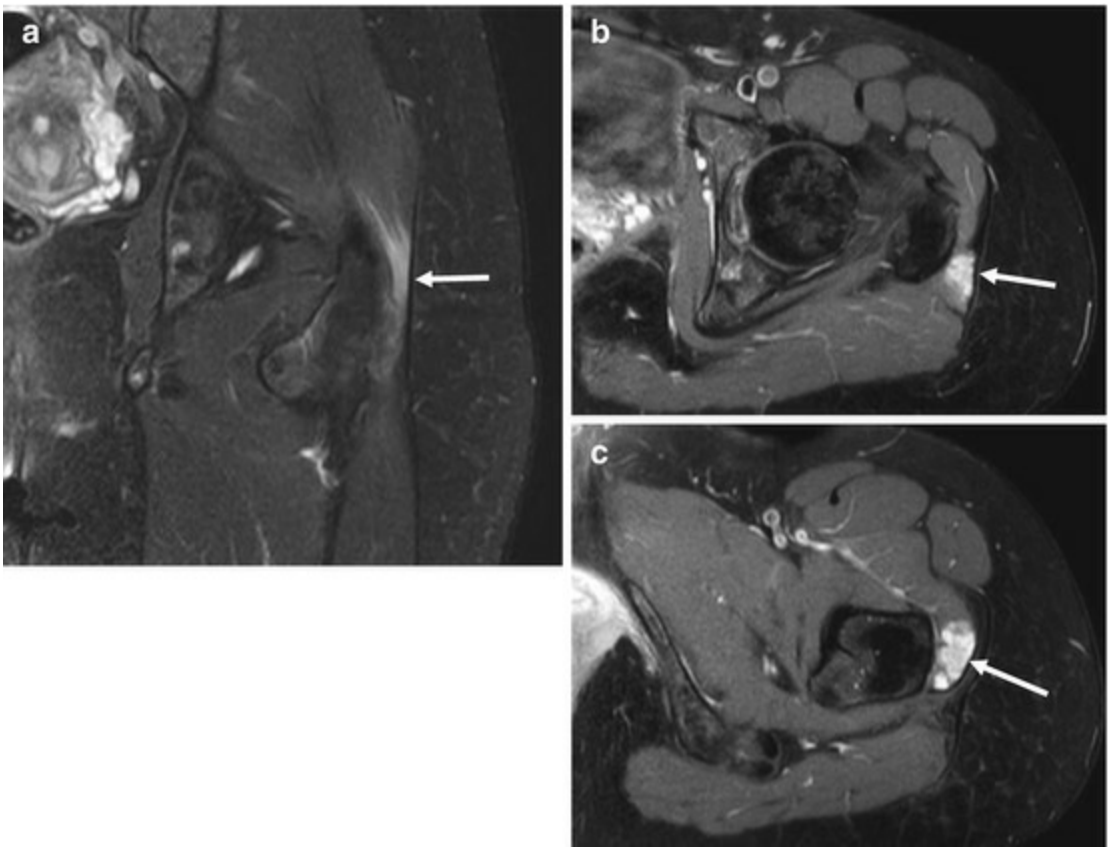
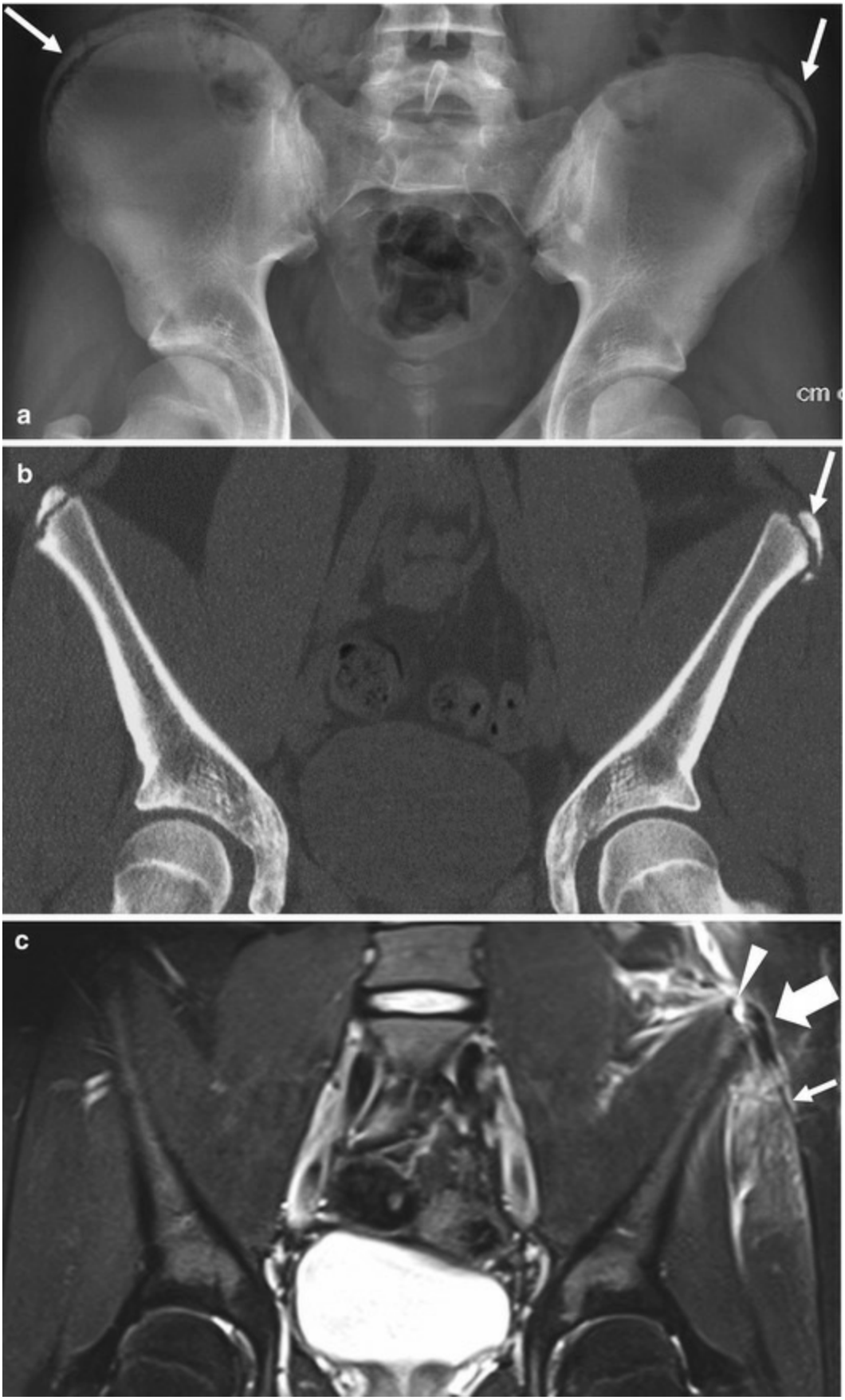


Fig. 19.3 A 30-year-old woman presenting with peri-trochanteric pain after a long time in a hammock. Coronal (**a**) and axial (**b**) fat-suppressed T2-weighted MRI show intra muscular focal areas of T2-hyperintensity at the gluteus maximus around the greater trochanter. Similar findings are present at the proximal portion of the vastus lateralis muscle (*arrow*, **c**). These findings are compatible with compressive myopathy of the gluteus maximus and vastus lateralis

19.3.2 Avulsion Injuries

The iliac crest is the most frequent site of avulsion injuries at the gluteal region. This type of lesion usually occurs in children and young adults. Although it can be secondary to acute trauma it is most commonly seen in association with micro-trauma and overuse. The anterior portion of the iliac crest adjacent to the insertion of the tensor of the fascia lata insertion and the ITB is the more frequently injured. An adapted MRI protocol with a field of view depicting the whole extension of the iliac crest is important for the diagnosis of these lesions. In acute injuries MRI shows typically edema and inflammatory changes at the tendinous insertions to the iliac crest, associated with reactive bone marrow edema-like changes to the iliac bone (Fig. 19.4). In some patients imaging findings can be discrete and a correlation with clinical findings is crucial. It is important to access the displacement of the iliac crest ossification center, for large displacements bear a worse prognosis. Sub-acute or chronic avulsions may have a bizarre appearance, with prominent bone proliferation, which should not be mistaken for bone or peri-osseous tumors. Finally, GM avulsions have been described after

direct trauma but are exceedingly rare and are usually associated with other serious injuries such as GM degloving [10].


a
b
c

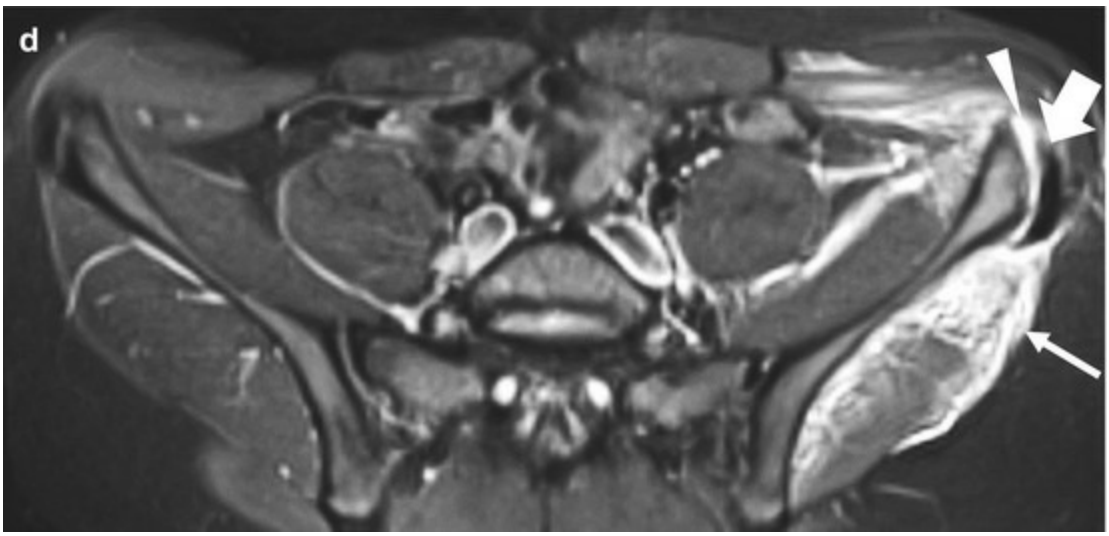


Fig. 19.4 An 18-year-old male athlete with post traumatic pain at the left iliac crest. (**a**) Anteroposterior radiograph demonstrates a bilateral irregularity and sclerosis of the iliac crest ossification centers (*arrows*). (**b**) Coronal CT reformatted image shows a discrete slipping of the left iliac crest ossification center *(arrowhead*). (**c**) Coronal and (**d**) axial fat-suppressed T2-weighted MRI show the displacement of the iliac crest ossification center (*large arrow*) with a fluid collection at the level of the synchondrosis (*arrowhead*). There is a marked signal hyperintensity of the gluteus maximus muscle fibers and the surrounding soft tissues (*thin arrow*)

19.3.3 Contractures

GM contractures can occur after direct trauma, repetitive gluteal injections or can be idiopathic. They are related to fibrous scarring of the muscle fibers and may lead to gait disturbances, pain, stiffness and anatomical deformity. MRI demonstrates a heterogeneous fibrous mass, with hypointense areas on T1- and T2-weighted images that may be associated with various degrees of muscle atrophy. Long standing contractures may be associated with bone remodeling at the posterior ilium, which can be identified on cross sectional imaging methods or conventional radiographs as an iliac hyperdense line parallel to the sacro-iliac joint [11]. Surgical release is the treatment of choice with very good results [12].

19.3.4 Morell Lavallée Effusion

Morell Lavallée lesions are characterized by a sero-hematic collection at the interface between the deep lower limb fascia and the hypodermis after traumatic injuries with shear stress to the skin. This type of lesion has been termed a closed degloving injury and is frequent at the gluteal region where the vascularization of the hypodermis is particularly rich [13]. The site of the lesion is the most important diagnostic clue on MRI, which usually demonstrates a well-defined fluid collection adjacent to the fascia lata. The signal intensity of the fluid varies depending on the age of the lesion and the stage of hemoglobin degradation. One interesting finding is a rim of signal hypointensity at the periphery of the lesion related to hemosiderin deposits, which is particularly

evocative in association with a hypersignal intensity of the fluid in T1-weighted images (methemoglobin) (Fig. 19.5). Morell Lavallée lesions regress spontaneously in about 50 % of cases. Chronic lesions can be heterogenous with internal calcifications. Care should be taken not to confound these lesions with soft tissue masses of other origins, and an active search for an ancient trauma is warranted [14].

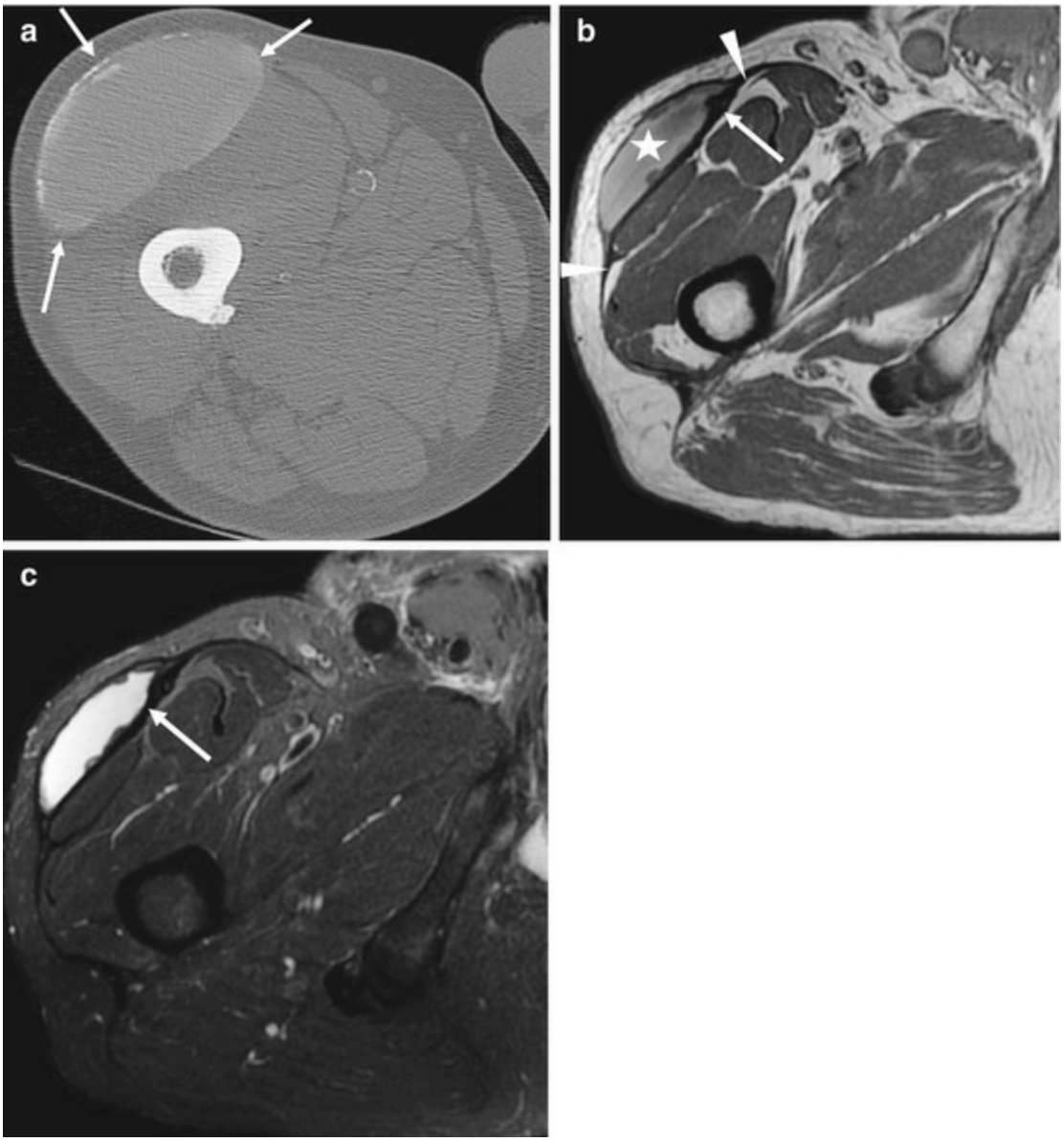


Fig. 19.5 A 70-year-old man with a history of major trauma 30 years ago, evaluated for a partially calcified mass incidentally discovered on a pelvic radiograph (not shown). (**a**) Axial CT image demonstrates a bi-concave, superficial, spontaneously hyperdense mass at the proximal thigh (*arrows*). Note that the lesion is located at the level of the deep aponeurosis of the thigh (fascia lata) and that it presents a peripheral rim of calcifications. Axial (**b**) T1- and (**c**) fat-suppressed T2-weighted MRI confirm the location of the mass adjacent to the fascia lata (*arrowheads*). The lesion is spontaneously hyperintense on T1 (*star*) and presents a thick low-signal intensity capsule in both sequences (*arrow*). These findings confirm the diagnosis of a chronic Morel Lavallée lesion of the proximal thigh

The treatment of Morel Lavallée effusions can be conservative or surgical.

Outcomes are better with surgical treatment but there is no consensus in the literature on which is the best technique. Surgical interventions with dead space closure techniques are preferred. In patients with chronic lesions with a well formed fibrous capsule around the lesion a complete resection of the lesion can be attempted. Morel Lavallée lesions can recur and serious surgical complications have been reported (infection, skin necrosis, delayed wound closure).

19.4 Peri-Trochanteric Pain Syndrome

19.4.1 Lateral Snapping Hip

Snapping hip syndrome (coxa saltans) is characterized by a painful audible or palpable snap or popping at the peri-trochanteric region during hip flexion-extension. Snapping hip syndrome has multiple possible etiologies both intra and extra articular. Lateral snapping is probably the most frequent type [15]. The passage of the ITB and the myotendinous junction of the GM over the greater trochanter creates this phenomenon, which can be completely asymptomatic or can cause pain, pseudo-instability and locking sensations leading to significant disability [16].

Multiple factors predispose to lateral hip snapping:

– Morphologic factors such as a small femoral neck angle (coxa vara), which increases the lateral projection of the greater trochanter.

– Increases in the anteversion of the femoral head, tibial internal rotation, foot pronation and also a shorter contra-lateral limb all lead to an excess in tension on the ITB [17].

– Morphologic anomalies at the surface of the greater trochanter (exostosis, fracture malunion, pseudarthrosis)

– Atrophy of the GM and fascia lata tensor leads to abnormal motion paths of the ITB over the greater trochanter during hip motion. A loss of 16 % of the eccentric contraction force in abduction was noted in a patient with lateral hip snapping compared to an age-matched control group [18].

– Anatomic variants of the ITB and the myotendinous junction of the GM have been described. Accessory ITB, thickening and anatomic variants of the GM superficial attachment (sickle deformity) to the ITB have been described [19] (Fig. 19.6).

– Strenuous physical activities especially those that require extremes of hip joint amplitude can increase significantly the mechanical stress at the peri-trochanteric region.

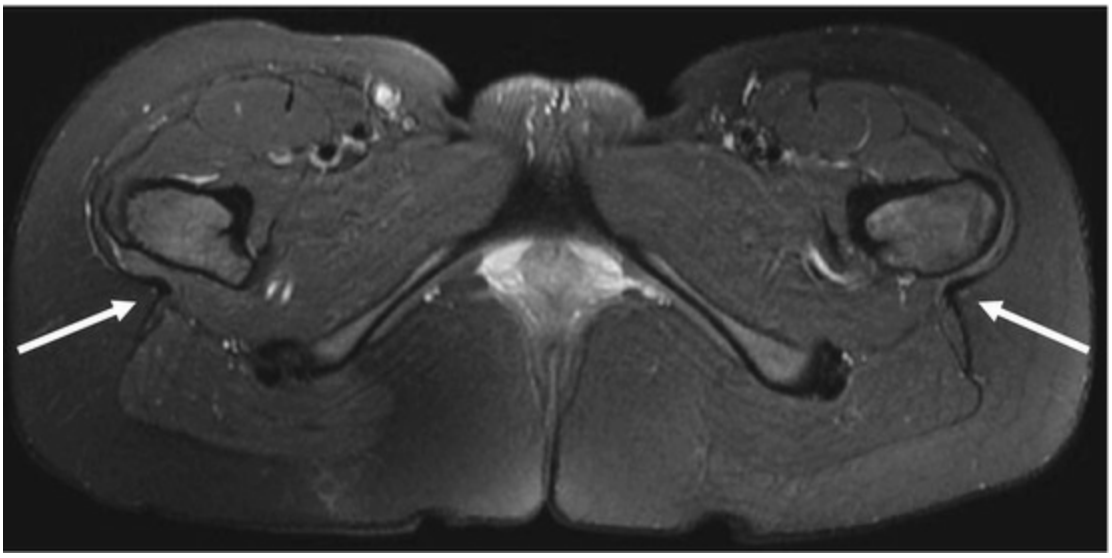


Fig. 19.6 A14-year-old girl with bilateral hip snapping, painful on the left. A sickle deformity of the gluteus maximus tendons, characterized by a deep location of the myotendinous junction posterior to the great trochanter is seen bilaterally on this axial fat-suppressed T2-weighted MRI (*arrows*)

Imaging is important in cases of lateral snapping hip because it helps confirm the diagnosis, and identifies its origin and associated morphologic findings. When a surgical treatment is proposed some form of pre-operative imaging is mandatory. Snapping syndromes are dynamic in nature. Hence the diagnosis on static imaging methods must rely on indirect signs. Conventional MR protocols can accurately depict the ITB and the myotendinous junction of the GM at the level of the greater trochanter, demonstrating abnormal signal, thickening and irregularity of the ITB and GM insertion. Inflammatory type signal hyperintensity around the ITB and peri-trochanteric bursitis can be demonstrated on fluid sensitive sequences. The absence of these findings by no means excludes a snapping hip syndrome.

Dynamic imaging methods are superior for the diagnosis of snapping phenomena [20]. The use of dynamic ultrasound is widely described in the literature [21–23]. Dynamic MRI can be used for the evaluation of lateral snapping hip syndrome. Balanced non-spoiled gradient-echo sequences (e.g. FIESTA, True FISP, Balanced FFE) allow visualization of a sudden anterior displacement of the ITB and GM myotendinous junction over the greater trochanter during hip flexion and extension (Fig. 19.7). The low spatial resolution of dynamic MRI sequences and limited amplitude of joint motion inside the gantry are important limitations of this technique [20, 24].

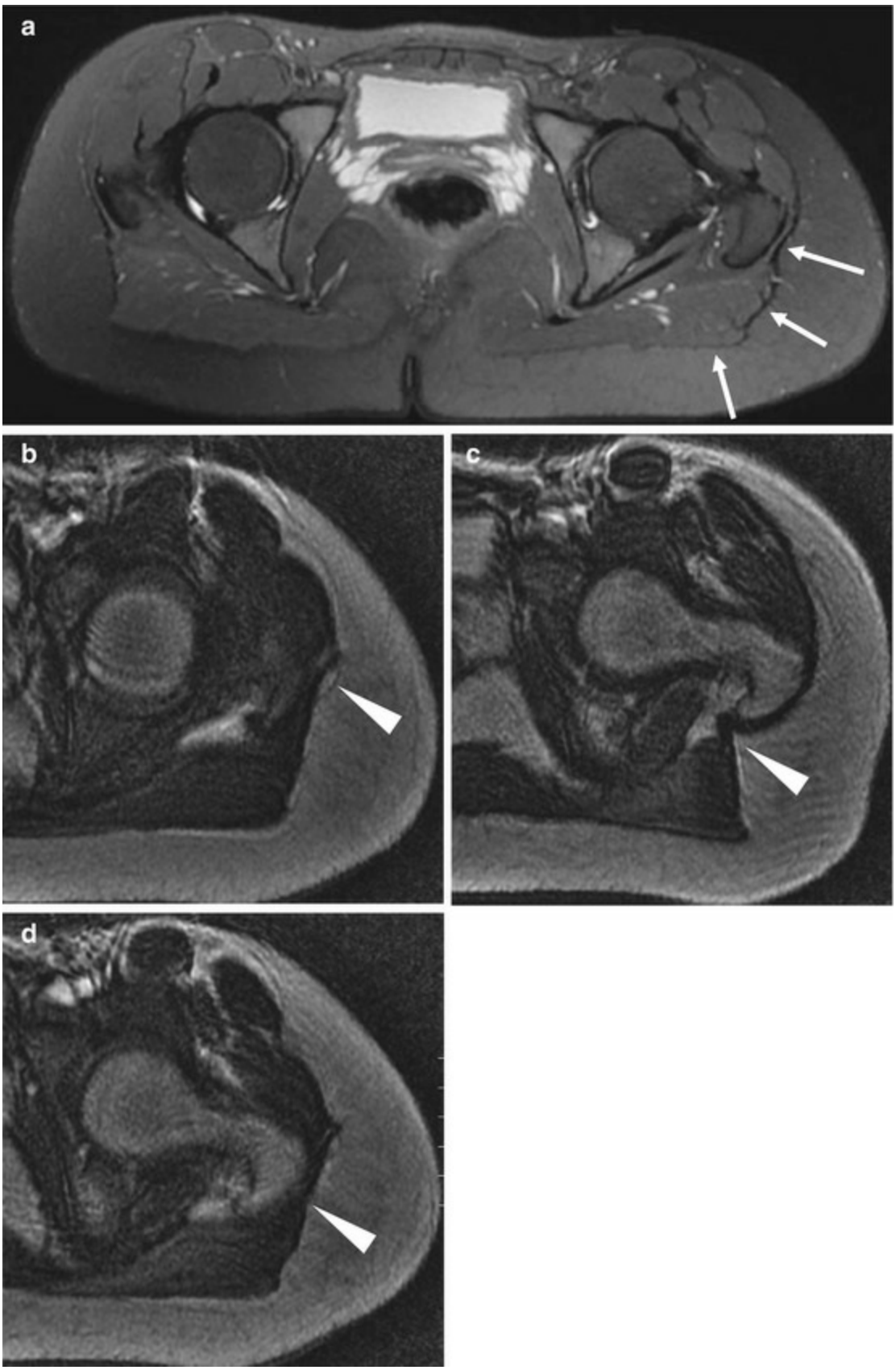


Fig. 19.7 A 21-year-old woman with a history of infantile cerebral palsy presenting with blockage and a snapping sensation of the left hip. (**a**) Static fat-suppressed axial T2-weighted MRI at the level of the greater trochanter shows a normal aspect of the gluteus maximus (GM) muscle and superficial insertion to the iliotibial band (ITB, *arrows*). (**b–**

d) Dynamic steady-state gradient-echo (FIESTA) MRI of the same patient during hip flexion-extension cycle show the position of the myotendinous junction of the GM is depicted by the *arrowhead*. At some point during flexion the GM myotendinous junction and the ITB are displaced abruptly behind the greater trochanter (**c**) with an accompanying painful palpable snap and a block in hip extension. With the continuation of the hip extension there is a sudden release with a normal position of the GM myotendinous junction (**d**)

The trophicity of the GM and fascia lata tensor muscles should be also taken in consideration. Asymmetries in volume and the presence of fatty atrophy in these muscles can predispose to lateral hip snapping. MRI can perform a full diagnostic evaluation in patients with snapping hip syndromes, allowing a confident identification of intra and extra-articular etiologies.

19.4.2 Trochanteric Bursitis

Trochanteric bursitis has multiple causes. The bursa can be directly affected (chronic microtrauma, regional muscle dysfunction) or secondarily to tendinopathy of the gluteus medius and minimus tendons due to chronic compression by the ITB [25]. Trochanteric bursitis can also be associated with excessive friction between ITB and the greater trochanter. This condition is more frequent in females, which is thought to be related to a wider gynecoid pelvis, which increases the mechanical stress over the gluteal tendons and the ITB.

MRI depicts a fluid collection and inflammatory type changes of the surrounding soft tissues adjacent to ITB on fluid-sensitive sequences and after the injection of gadolinium (Fig. 19.8). A distended bursa may extend posteriorly around the posterior facet and in some cases around the ITB. MRI should always be correlated with clinical and physical exam findings since signal anomalies in the peri-trochanteric region can be present in up to 60 % of asymptomatic patients [26, 27].

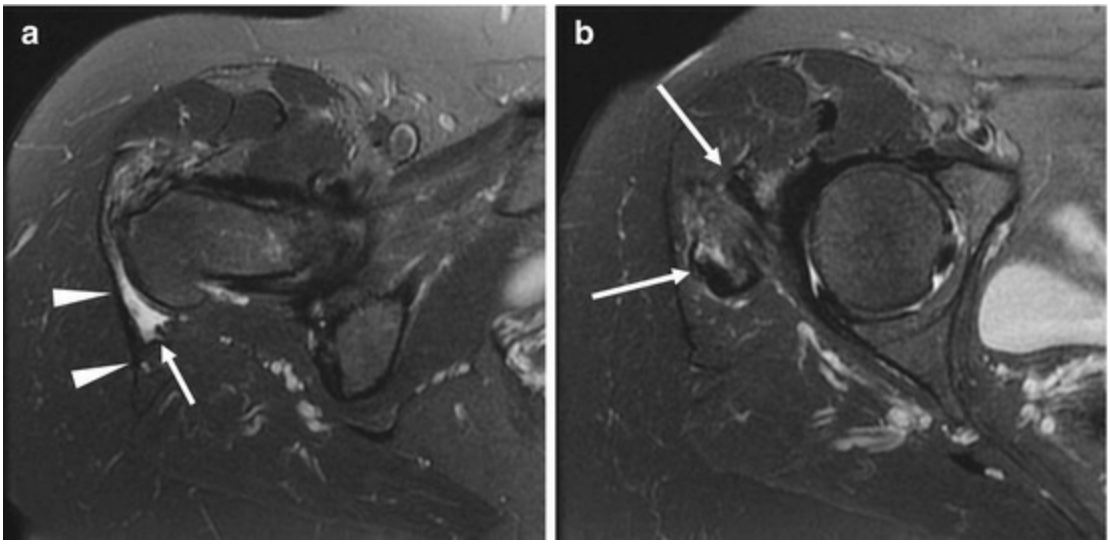


Fig. 19.8 A 65-year-old woman with chronic hip pain during physical activities (climbing stairs). (**a**) Axial fat-

suppressed T2-weighted MRI demonstrates an effusion of the trochanteric bursa that presents thickened walls and signs of synovial hypertrophy (*arrow*). There is a posterior extension of the bursa around the great trochanter. Note the overlying position of the iliotibial band and the myotendinous junction of the gluteus maximus (*arrowheads*). (**b**) Axial fat-suppressed T2-weighted MRI of the same patient demonstrates the associated tendinopathy of the gluteus medius and minimus tendons (*arrows*)

19.4.3 Differential Diagnosis

Greater trochanteric pain syndrome is extremely common and is estimated to affect 10–25 % of the population of industrialized countries [28]. There are many causes of peri-trochanteric pain, and a full review of the subject would be beyond the scope of this chapter. Gluteus medius and minimus tendinopathy and bursitis are important to consider when evaluating patients with peri-trochanteric pain. This condition is characterized by an inflammatory type hyperintensity surrounding the gluteus medius and minimus tendons, which may be thickened and hyperintense. Effusion at the gluteus medius and minimus bursae is frequently associated. Tendon ruptures are rare but can occur. Intra-articular pain can radiate to the peri-trochanteric region and should be thoroughly searched for in cases of unexplained peri-trochanteric pain. Greater trochanteric pain syndrome is also associated with lumbar pain, osteoarthritis and obesity [25].

19.5 Gluteus Maximus Tendon Pathology

Intrinsic GM tendon pathology is rare. Crystal deposition disease is probably the most frequent abnormality at this region. GM calcific tendinitis is an example of a common condition in an atypical place, which may lead to diagnostic difficulties. Hydroxyapatite deposition may lead to a prominent inflammatory reaction and contrast enhancement at the deep insertion of the GM tendon, which is associated with amorphous and low density calcification. GM calcific tendinitis may be associated with reactive bone marrow edema pattern of the femur and cortical erosions [29]. Plain film or CT may help in the identification of hydroxyapatite deposits, which are sometimes hard to see on MRI. There are no reports in the literature describing overuse or microtraumatic GM tendinopathy, which is probably extremely rare (Fig. 19.9).

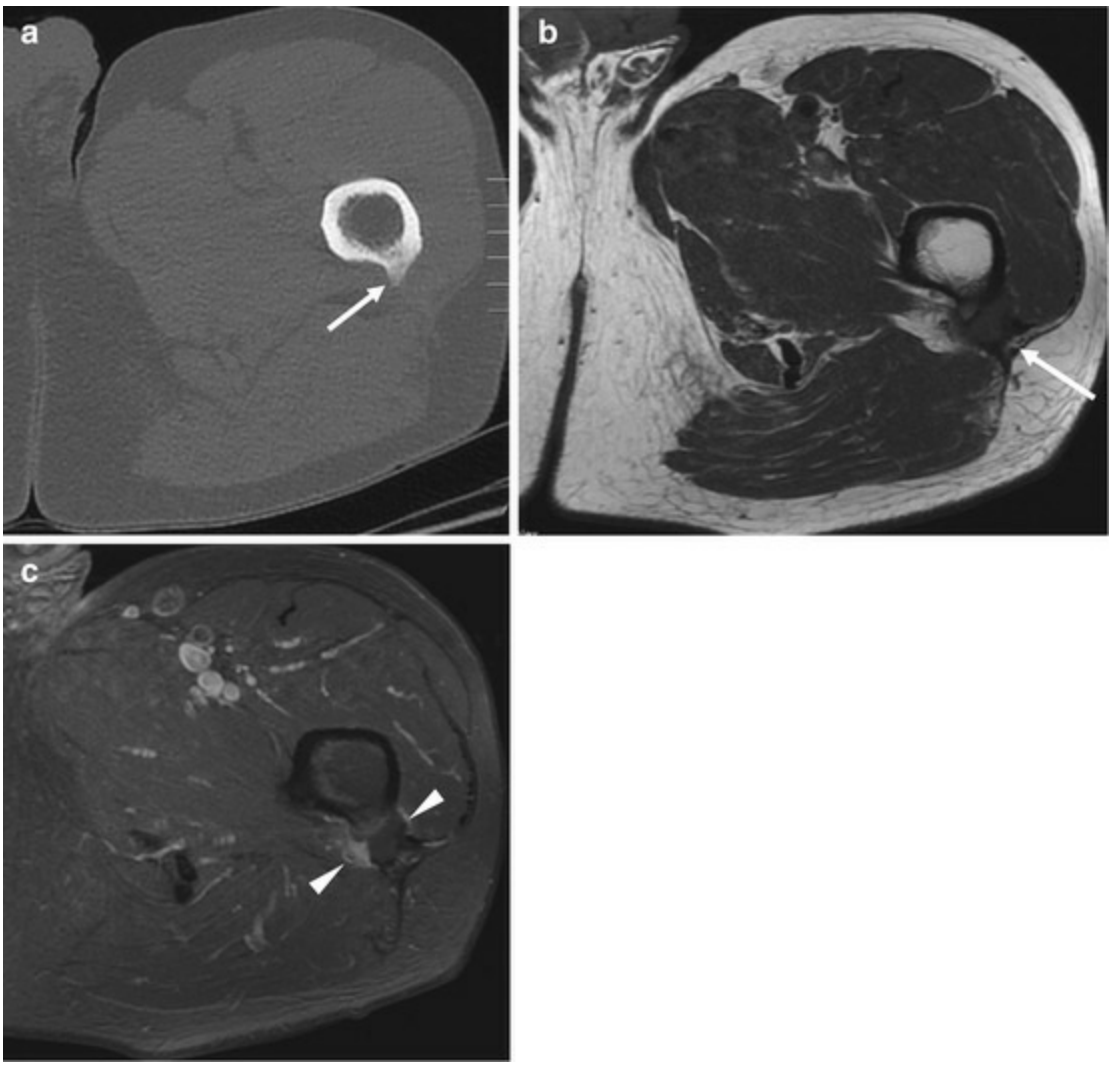


Fig. 19.9 A 70-year-old man with a painful cortical irregularity of the posterior femur. (**a**) Axial CT image shows a discrete rarefaction of the posterior femoral cortex, associated with a enthesophyte formation (*arrow*). (**b**) Axial T1-weighted MRI shows marked thickening of the deep insertion of the gluteus maximus tendon at the femur (*arrow*). (**c**) Axial fat-suppressed contras-enhanced T1-weighted MRI shows the enhancement adjacent to the enthesophyte (*arrowheads*). These findings are evocative of an inflammatory gluteal tendinopathy

Key Points

– The GM muscle has two functional units and two insertions, at the posterior femur and at the ITB.

– Traumatic lesions at the gluteal region are relatively rare but muscle sprains, compressive myopathy can occur.

– Morel Lavallée lesions should be evaluated for the presence of a fibrous capsule containing hemosiderin deposits and should be considered in the differential diagnosis of soft tissue masses.

– The use of dynamic imaging is important for the diagnosis of snapping hip

syndrome.

– Inflammatory reaction adjacent to the ITB and GM myotendinous junction, along with variations in the morphology of these structures, are associated with lateral hip snapping.

– Signal anomalies at the level of the trochanteric bursa are frequent in asymptomatic patients.

19.6 Conclusion

MRI is a complete method for the evaluation of gluteal region, which has a complex anatomy and physiology. Multiple pathologic processes take place at the GM and surroundings and knowledge of the imaging aspects of each of these conditions is important for image interpretation. Finally, in the context of snapping hip syndromes the role of dynamic acquisitions should be emphasized since they offer a real diagnostic advantage over static imaging.

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20. Plantaris Muscle Injuries

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Abstract
The plantaris muscle is a small vestigial structure and one of the superficial muscles of the posterior compartment of the calf. It is thought to be present in 80–90 % of the population and of questionable physiological importance. Despite this, injuries to the plantaris are not an uncommon cause of patient pain, and can present a diagnostic challenge clinically, making an understanding of plantaris anatomy and related pathology important to the clinician and radiologist. This chapter will focus on the anatomy, imaging and clinical findings of plantaris muscle pathology and treatment.

20.1 Introduction

The plantaris muscle is a small, thought to be an accessory vestigial muscle [1], that is present in 80–90 % of the population. In athletes the plantaris muscle belly can be involved clinically as a part of undetermined pain in the popliteal fossa or may be associated more distally with chronic Achilles tendon pain [2, 3]. Isolated ruptures of the plantaris are infrequently described and although the incidence rate is not known they are suspected to be underreported [4, 5].

Pathology of the plantaris muscle and tendon is an important differential

consideration in posterior calf pain. It is not known to what extent the diagnosis is missed by refraining from imaging. It is possible that a rupture of the plantaris tendon can be clinically diagnosed incorrectly as an acute exacerbation of Achilles tendinopathy or a (partial) acute Achilles tendon rupture. At one time there was controversy in the literature over whether there was really such a thing as an injury of the plantaris. With modern imaging and surgical documentation, however, their existence has been proved [6, 7]. Still, they remain less common than gastrocnemius injuries [8].

20.2 Anatomy

The small muscle belly of the plantaris is only about one-third the length of the belly of the gastrocnemius and less than 3 % of the mass of the gastrocnemius [9]. The muscle has been reported to range from 7 to 13 cm in length with much variation in size and form when present [10].

It is the vestigial component of the triceps surae complex as it co-functions with the soleus and gastrocnemius muscles in the superficial posterior compartment of the calf.

The plantaris and its long tendon courses between the gastrocnemius and the soleus (Fig. 20.1) and the three collectively are referred to as the triceps surae.

Fig. 20.1 Schematic drawing of the posterior view of the lower leg illustrates the plantaris muscle

The long tendon is commonly harvested by orthopedic surgeons as an autograft for ligament and tendon reconstructions, demonstrating that function is maintained despite the absence of the tendon [7]. In cadaveric dissection, the plantaris has been referred to as the "freshman's nerve", because its long, slender-white tendon can be mistaken for a nerve [10].

The plantaris has been shown to vary in its origin and insertion [1]. In most cases the muscle is found to originate from the supracondylar ridge of the lateral femoral condyle, coursing medially down the leg to insert on the calcaneus, just medial to the Achilles [7, 11–13].

At the proximal third of the leg, the muscle belly is found between the popliteus muscle anteriorly, and the lateral head of the gastrocnemius muscle posteriorly. The

myotendinous junction occurs approximately at the level of the origin of the soleus muscle from the tibia in the proximal portion of the lower leg [7]. The long thin tendon forms part of the medial border of the muscle belly as it courses between the medial head of the gastrocnemius and the soleus muscles in the midportion of the calf [14].

Anatomic studies have demonstrated that the calcaneal insertion may occur independently of the Achilles tendon and this could explain why the plantaris tendon often remains intact when the Achilles tendon ruptures [11]. Neural innervation is provided by the tibial nerve (S1, S2), common to all three muscles of the triceps surae group.

In terms of function, the plantaris is in a position to act with the gastrocnemius as either a flexor of the knee or a plantarflexor of the ankle; However, its motor functions are trivial. It appears to be a highly specialized sensory muscle and is considered to be an organ of proprioceptive function for the more powerful plantarflexors as it contains a high density of muscle spindles [10].

20.3 Pathology

Despite its small size, injuries to the plantaris are relatively common [11] and have been termed “tennis leg”. The pathogenesis of tennis leg, and the existence of an isolated rupture of the plantaris muscle as the cause, has been debated since it was initially described by Powell in 1883 [15]. Since then the term “tennis leg” has been used, but it has been a source of debate in the literature [12, 16–19].

For years, the condition was believed to be a rupture of the plantaris tendon at the medial aspect of the calf. Other authors have implicated a tear of the medial head of the gastrocnemius, soleus, plantaris or a combination thereof [12, 20, 21, 22] and some still question the role of the plantaris tendon in tennis leg [12, 20]; Arner and Lindholm [23] in 1958, Miller [18] in 1977 and Severance and Bassett [12] in 1982, all concluded that isolated rupture of the plantaris was not the cause of this clinical condition. Surgically confirmed cases of ruptures of the plantaris tendon or musculotendinous junction have been reported in conjunction with this clinical diagnosis [6, 19].

A review of the literature demonstrates that injury to the plantaris muscle either on its own, or in combination with injury to the gastrocnemius or soleus, can represent the clinical condition known as tennis leg [24].

Plantaris injuries are not as common as injuries of the gastrocnemius, which is known to be particularly vulnerable due to its superficial location that spans two joints (the knee and the ankle) and its composition of type IIb muscle fibers [17, 25]. A study by Koulouris et al. in 2002 with MRI found that the medial head of the gastrocnemius is the most commonly injured muscle of the calf, closely followed by the soleus [26].

The plantaris is injured most frequently during running or jumping and usually as the result of an eccentric load placed across a forced dorsiflexed ankle with the knee in an

extended position [20, 27]. It has been established with MRI, ultrasound and surgical exploration that injuries to the plantaris may in fact occur in isolation [6, 7, 11, 28] or in association with traumatic tears of the anterior cruciate ligament, arcuate ligament complex, and posterolateral corner muscles (lateral head of the gastrocnemius and popliteus) [7]. A posterior compartment syndrome, which requires surgical decompression, is a potential complication of plantaris or medial gastrocnemius ruptures.

20.4 Clinical Presentation

Even though the injury is the result of an indirect mechanism, patients report feeling as though they had been kicked in the back of the leg, and often report a "pop" in the calf [29].

Depending on its severity, the calf pain may cause the athlete to stop playing, or not. The pain usually becomes more severe with rest or on the next day [30]. Swelling may accompany the pain and may extend down to the ankle. Any attempt at active or passive dorsiflexion, and resisted plantarflexion will elicit severe pain [29, 30].

Deep venous thrombosis and a ruptured Baker cyst should be kept in mind in the differential clinical considerations when evaluating a patient with a probable plantaris injury [11, 14, 20].

20.5 Imaging

Magnetic resonance imaging (MRI) and ultrasound can be used in the diagnostic evaluation of patients with the clinical diagnosis of posterior lower leg pain. Ultrasound is less expensive, but operator dependent. However, ultrasound is more frequently used in the initial evaluation of suspected deep venous thrombosis and should be considered in the differential consideration of a patient with posterior calf pain. Imaging will help the clinician confirm the clinical diagnosis and exclude other etiologies (Figs. 20.2 and 20.3).

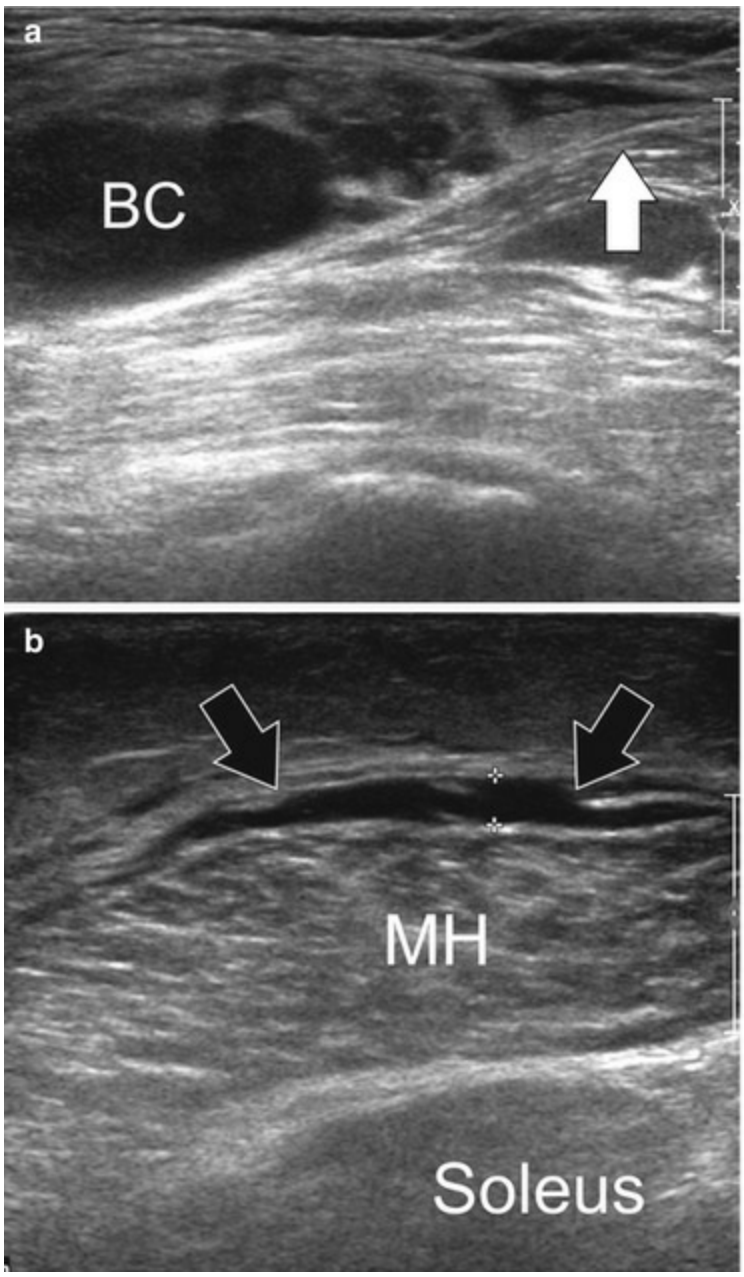

Fig. 20.2 (**a**) Longitudinal ultrasound obtained over the medial popliteal space shows a Baker cyst (*BC*). Note the pointed appearance (*arrow*) of the inferior part of the cyst due to recent rupture. (**b**) Transverse ultrasound obtained over the middle part of the medial head of the gastrocnemius muscle (*MH*) shows a fluid accumulation (*arrows*) located inside the subcutaneous tissue of the medial calf

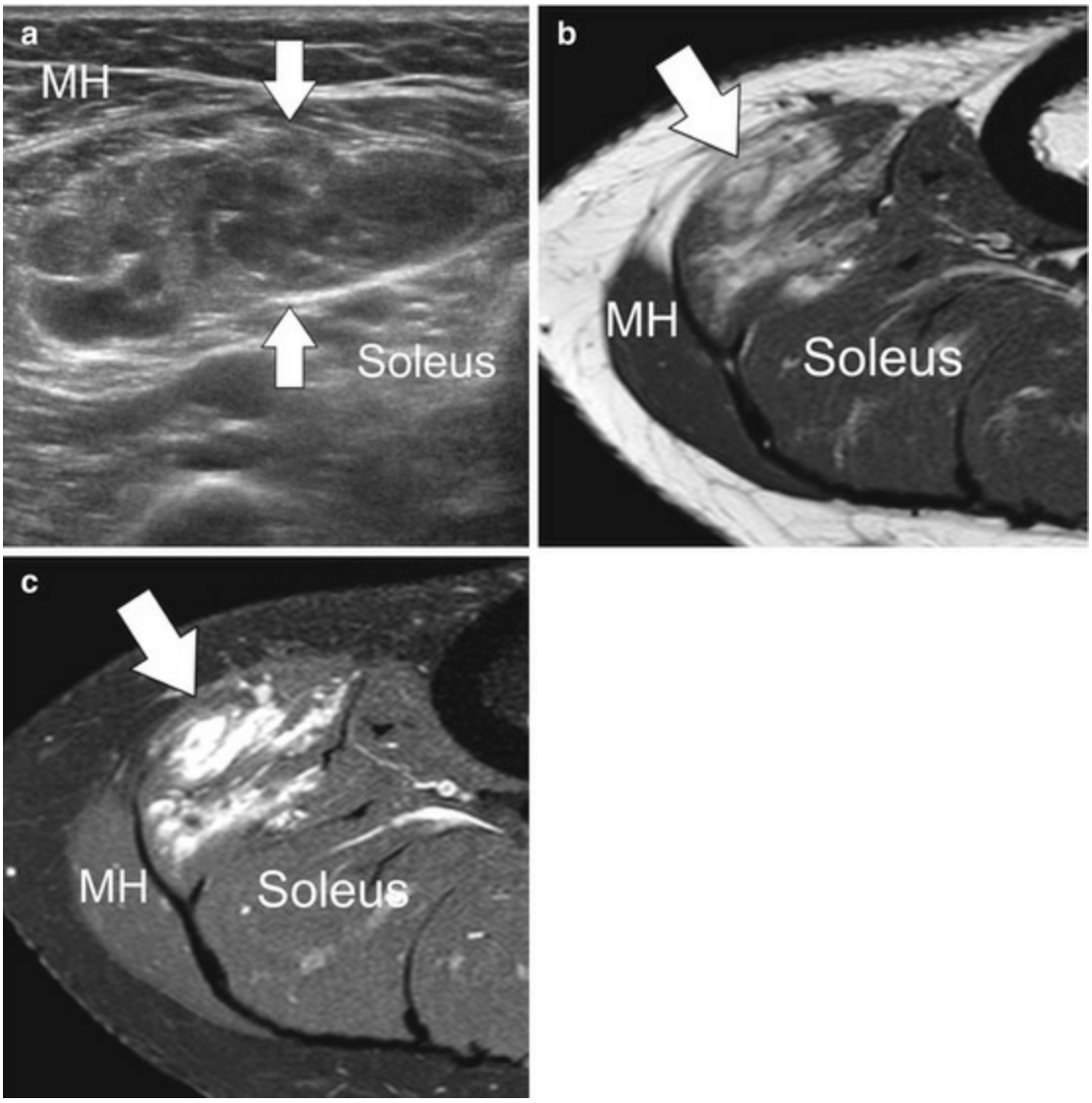

Fig. 20.3 (**a**) Transverse ultrasound obtained over the medial part of the soleus muscle shows an intramuscular mass (*arrows*) of mixed echogenicity that was a hemangioma. Corresponding axial T1- (**b**) and fat-suppressed contrast-enhanced T1-weighted (**c**) MRI confirm the ultrasound findings. *MH* medial head of the gastrocnemius muscle

On ultrasound, the muscle belly of the plantaris muscle is initially located in the proximal calf region using a transverse scan. It is seen as a triangular structure having the soleus muscle as its base and the medial and lateral bellies of the gastrocnemius as it sides [14].

While scanning with the transducer in a transverse plane it is possible to follow the muscle from its most proximal attachment on the lateral femoral condyle, down to the myotendinous junction at the level of the fibular head. Since the tendon forms the medial border of the belly, it will be seen in the transverse plane as a subtle thickening at the medial corner of the triangular muscle belly. The tendon is generally not visualized well in the transverse plane, distal to the myotendinous junction [14].

The fiber orientation of the plantaris muscle belly and tendon is best visualized by

rotating the transducer longitudinally along the long axis of the muscle. In most cases, the fibrillar pattern of both the muscle and tendon is well depicted [14] (Fig. 20.4).

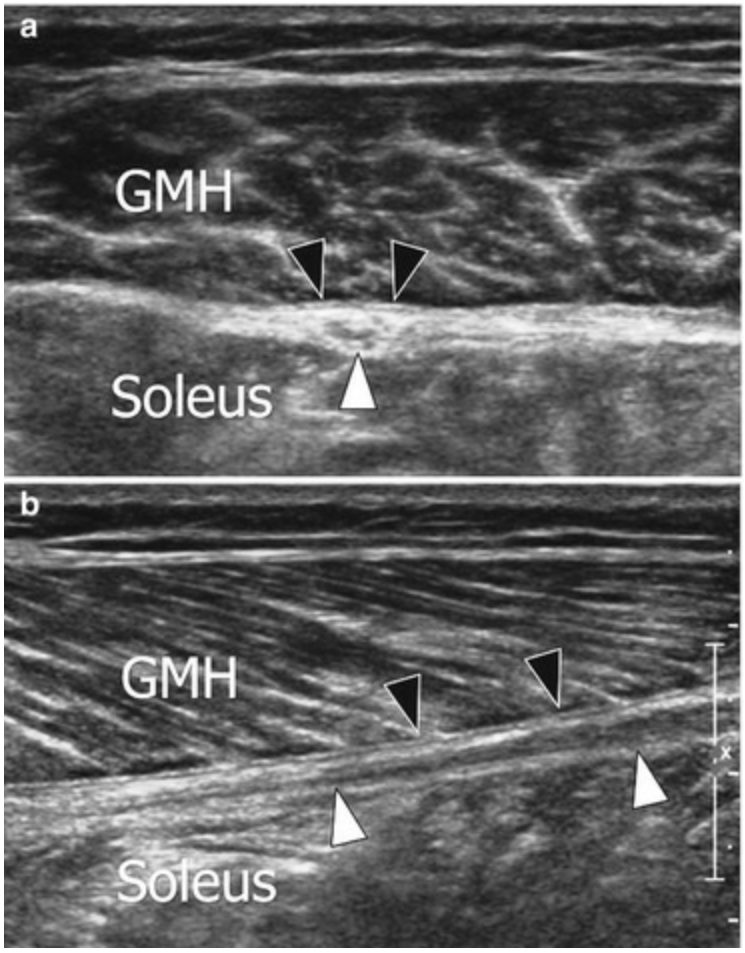

Fig. 20.4 (**a**) Transverse and (**b**) longitudinal ultrasound images demonstrate black arrowheads represent the anterior aponeurosis of the medial head and the white arrowheads depict the normal plantaris tendon. *GMH* medial head of the gastrocnemius muscle

As noted earlier, tennis leg can be a tear of the medial head of the gastrocnemius (Fig. 20.5), soleus, plantaris or a combination thereof (Fig. 20.6). Imaging will help define the extent of injury and potentially exclude more serious conditions such as deep venous thrombosis [11, 29] (Fig. 20.7). Tears of the plantaris most often occur at the level of the myotendinous junction, although there have been reports of isolated midsubstance tears [35] (Fig. 20.8).

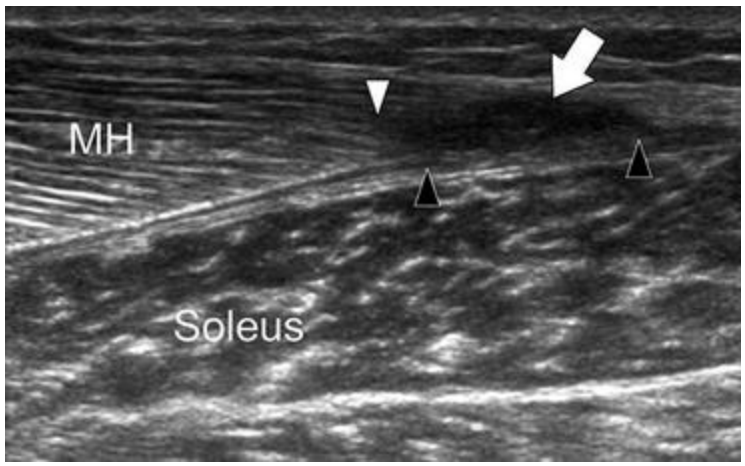

Fig. 20.5 Longitudinal ultrasound obtained over the medial head of the gastrocnemius muscle (*MH*) shows a myotendinous avulsion (*arrow*) of the inferior part of the medial head of the gastrocnemius muscle. Note interruption of the fibroadipose septa (*white arrowhead*) as well as of the distal aponeurosis (*black arrowheads*) of the medial head. No local hematoma can be detected. The proximal part of the medial head and the soleus muscle are normal

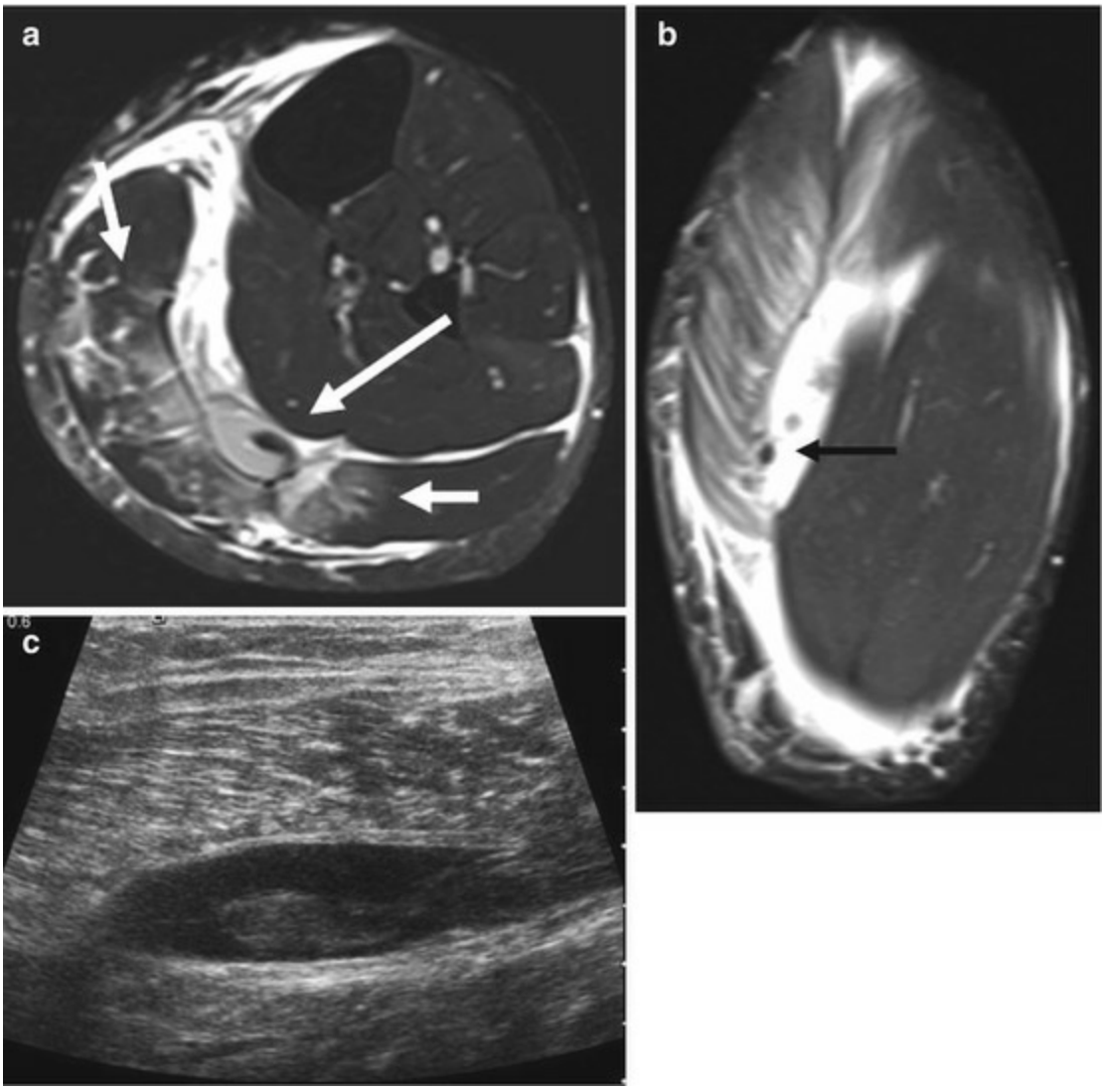

Fig. 20.6 (**a**) Axial fat-suppressed T2-weighted MRI demonstrates fluid between the soleus muscle and the medial head of the gastrocnemius muscle. Irregular low signal intensity within the fluid collection represents the remnant of the ruptured plantaris tendon (*long white arrow*). Feathery edema involving both medial and lateral heads of the gastrocnemius is compatible with strain (*short white arrows*). (**b**) Coronal fat-suppressed T2-weighted MRI again demonstrates fluid between the soleus muscle and the medial head of the gastrocnemius muscle and irregular low signal intensity within the fluid collection that represents the remnant of the ruptured plantaris tendon (*arrow*). (**c**) Longitudinal ultrasound demonstrates the same

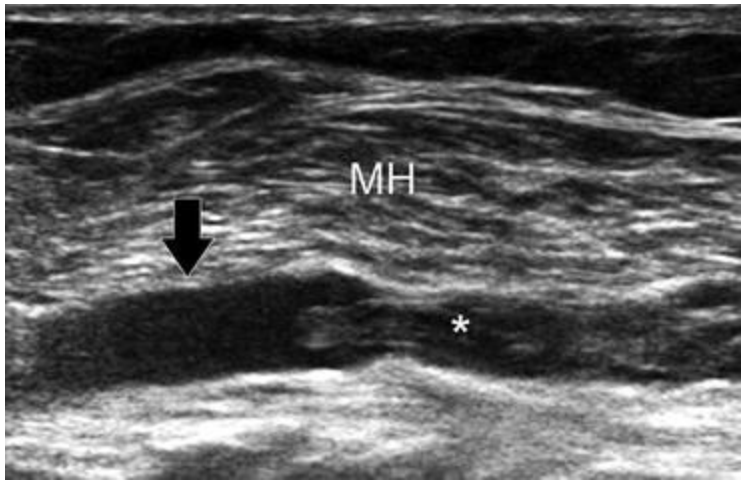

Fig. 20.7 Longitudinal ultrasound obtained over the medial head of the gastrocnemius muscle (*MH*) demonstrates echogenic thrombus (*asterisk*s) within an intramuscular vein. The proximal part of the vein (*arrow*) is filled by normal appearing anechoic blood

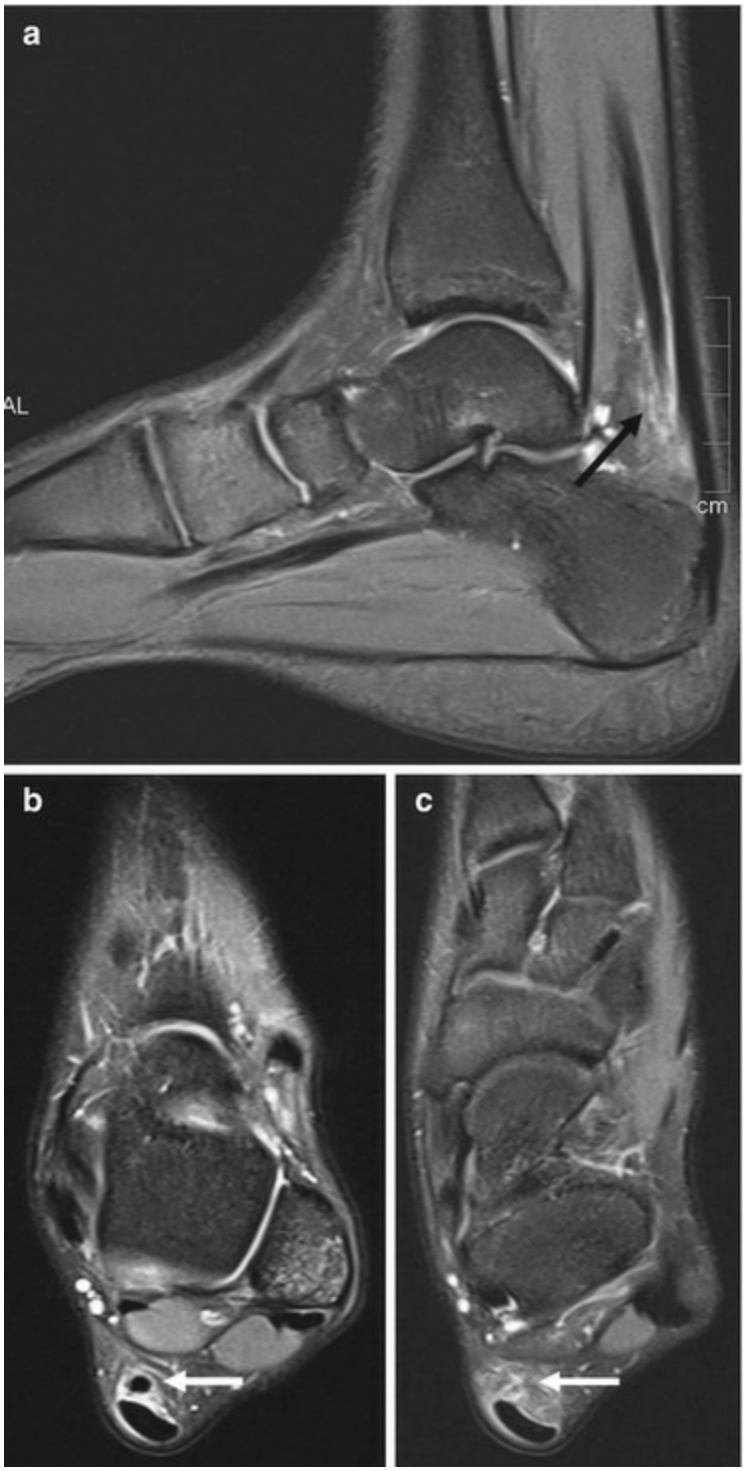

Fig. 20.8 (**a**) Sagittal fat-suppressed T2-weighted MRI demonstrates complete tear of the plantaris tendon distally with some retraction (*arrow*). (**b**, **c**) Sequential axial fat-suppressed T2-weighted MRI again demonstrate complete tear of the plantaris tendon distally with thickened retracted tendon (*white arrow*)

A rupture of the plantaris will show discontinuity of the muscle or tendon on longitudinal scanning. Fluid usually accumulates in an elliptical configuration between the medial head of the gastrocnemius and the soleus muscle bellies, along the course of the plantaris [31] (Fig. 20.9).

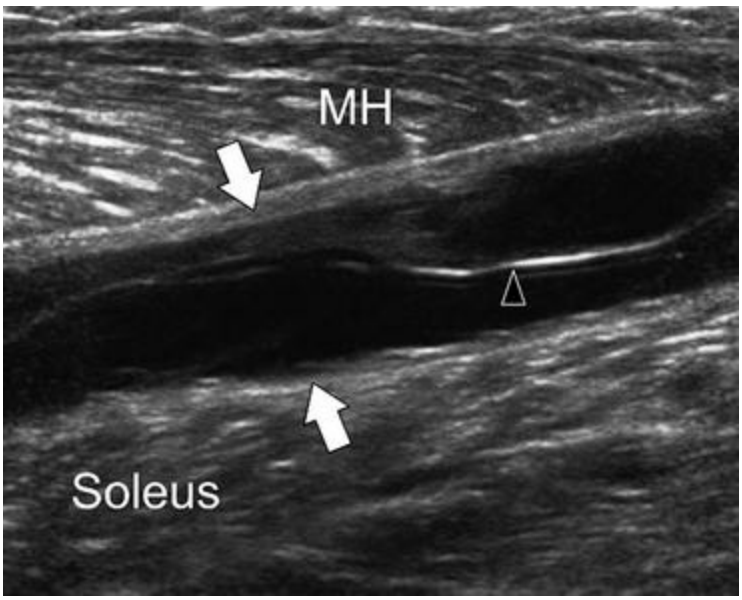

Fig. 20.9 Longitudinal ultrasound image obtained over the medial head of the gastrocnemius muscle shows a large hematoma (*arrows*) located between the inferior part of the medial head of the gastrocnemius muscle (*MH*) and the soleus muscle due to distal avulsion of the medial head. Ultrasound shows a hyperechoic structure located inside the hematoma probably corresponding to the torn plantaris tendon (*arrowhead*)

Visualization of the torn plantaris tendon is important, since fluid between aponeuroses of the soleus muscle and medial gastrocnemius muscle is non-specific, and can be seen with medial gastrocnemius muscle injury [21, 25, 32], plantaris tendon rupture especially at the level of the muscle belly or musculotendinous junction [7, 9, 25], or a ruptured popliteal cyst.

The fluid collection has been considered likely to represent secondary hematoma [21]; however, Guillodo et al. noted that a fluid collection in this location in patients with tennis leg is not hemorrhagic in origin but more likely pseudocystic [32].

Some have associated the fluid collection with plantaris pathology even when no muscle or tendon tear is seen. Using MRI, Helms et al. demonstrated that fluid may be associated with plantaris strains [7] and Leekham et al. concluded that when such fluid collections are seen in patients with a strong clinical suspicion for plantaris injury, a diagnosis of plantaris strain is appropriate even if no tear is seen [7, 14].

In a study by Delgado et al., fluid between the medial head of the gastrocnemius and soleus was a common finding in patients with rupture of the medial head of the gastrocnemius muscle, occurring in more than 50 % [11]. They believed that a hematoma would be unlikely to occur with an isolated disruption of the plantaris tendon, because, like all tendons, it is an avascular structure; it was more probable that a hematoma would develop with a rupture of the more vascular medial head of the gastrocnemius muscle. To add to this, Bianchi et al. reported it difficult to depict small ruptures in the anteromedial portion of the medial head of the gastrocnemius muscle with ultrasound [21].

Modern high-field MRI with its high spatial resolution is an excellent diagnostic modality to evaluate for suspected muscle pathology and the extent of injury and if there is any other associated pathology. The appearance on MRI depends on the severity of the muscle strain. As with other muscle pathologies, an area of abnormal T2 prolongation may be seen both within, and adjacent to the muscular fibers of the plantaris muscle at the level of the knee joint or at the myotendinous junction. In more proximal injuries to the plantaris muscle, plantaris strain can be seen in association with anterior cruciate and arcuate ligament injury [7].

With complete plantaris rupture, which usually occurs at the myotendinous junction, there will be proximal retraction of the muscle, which can appear as a mass located between the popliteus tendon and the lateral head of the gastrocnemius. This mass-like lesion will demonstrate high signal intensity on T2-weighted and fat-suppressed images in the acute setting unless it is ruptured completely through the mid-substance of the tendon [7]. In the acute setting, an intermuscular fluid collection between the soleus muscle and the medial head of the gastrocnemius may also be seen as described above (Fig. 20.10).

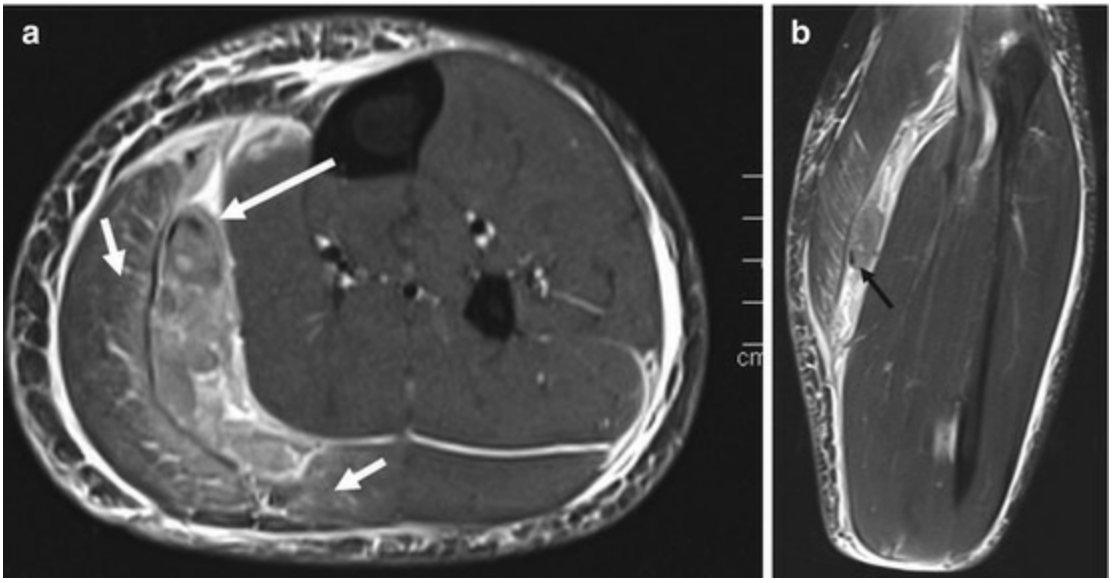

Fig. 20.10 (**a**) Axial fat-suppressed T2-weighted MRI demonstrates fluid between the soleus muscle and the medial head of the gastrocnemius muscle. Irregular low signal intensity within the fluid collection represents the remnant of the ruptured plantaris tendon (long arrow). Feathery edema involving both medial and lateral heads of the gastrocnemius is compatible with strain (*short arrows*). (**b**) Coronal fat-suppressed T2-weighted MRI again demonstrates fluid between the soleus muscle and the medial head of the gastrocnemius muscle and irregular low signal intensity within the fluid collection that represents the remnant of the ruptured plantaris tendon (*long arrow*)

20.6 Treatment

Although no studies have looked specifically at the treatment of plantaris injury [24], literature on non-specific tennis leg has clearly shown that conservative therapy is

effective [11, 18, 24, 27, 29] and that permanent disability rarely results [27]. A properly executed non-operative treatment will result in a good outcome in virtually all cases of plantaris muscle injury [33].

The treatment of plantaris injuries is usually conservative, involving the RICE–rest, ice, compression, elevation–protocol [24]. Although, there is no direct evidence for this protocol, there is scientific evidence for the appropriateness of its components [33].

The recommended period of immobilization is short (1–3 days, depending on the extent of the injury) with the ankle in a neutral, or slightly dorsiflexed position while maintaining the knee in a straightened position [24].

Following the period of immobilization a gradual progression of passive, active, and resisted movements may begin although with care that it occurs within the limits of tissue tolerance [24]. It has been demonstrated that early mobilization is important for healing by creating more rapid and intensive capillary in-growth, better regeneration of the muscle fibers, more parallel orientation of the regenerating myofibers, and a faster recovery of biomechanical strength [24, 33, 34].

Surgical treatment (fasciotomy) is indicated in situations when the injury is complicated by posterior compartment syndrome because of the swelling and hematoma formation associated with a rupture or tear [7, 11].

20.7 Conclusion

Despite the plantaris being a small vestigial muscle of limited motor function, it is important to understand and keep plantaris injuries in mind when coming up with differential diagnoses of the painful calf. Modern imaging helps play a role in confirming the clinical suspicion, determining the extent of injury and what other surrounding structures are involved. In addition, imaging helps exclude other potential causes of posterior calf pain. The treatment of plantaris muscle or tendon injuries is typically conservative.

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21. Leg Posterior Muscle Compartment Injuries

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Abstract
In this chapter we describe the anatomy of the posterior compartment muscle groups in the calf, including the gastrocnemius, soleus, plantaris, popliteus and others. The anatomy, pathophysiology, clinical signs, and imaging findings of each muscle are explained. Posterior calf pain is a frequently encountered clinical symptom in practice. Owing to its accessibility and relative low cost, ultrasound is the first step for imaging evaluation. For professional athletes, however, MRI remains the "gold standard" for imaging of muscle injuries.

21.1 Introduction

The calf is separated into two muscular anatomic areas. The deep compartment of the posterior calf is composed of the hallucis flexor muscle, the common toe flexor muscle and posterior tibial muscle. The posterior compartment (superficial) is composed of four muscles: the medial and lateral gastrocnemius, the soleus, and the plantaris muscle usually called the triceps surae muscle. Except for the soleus, these muscles are biarticular. The distal insertion of the gastrocnemius muscle and soleus compose the Achilles tendon [1]. The mechanical action of the triceps surae enables walking and maintaining vertical posture. The classic injury of the surae triceps, "tennis leg",

involves the distal medial gastrocnemial myotendinous junction.

21.2 Gastrocnemius Muscles

21.2.1 Anatomy

The medial head of the gastrocnemius arises from the posterior surface of the femur, behind the medial supracondylar ridge and adductor tubercle. The lateral head arises from the lateral surface of the lateral condyle of the femur, proximal and posterior to the lateral epicondyle, and attaches above the knee to the posterior aspect of the medial and lateral femoral condyles. Each of these heads is flat. Gastrocnemius muscles have additional proximal attachments from the posterior capsule of the knee joint and from the oblique popliteal ligament. The medial head is broader and thicker than the lateral head.

The proximal tendons of the lateral and medial head fuse posteriorly and compose the posterior aponeurosis of the gastrocnemius belly. The anterior aponeurosis of the gastrocnemius belly thickens gradually and forms the distal myotendinous junction and the distal tendon. This distal gastrocnemius tendon is separated from the soleus tendon by a bursa. Then the tendons fuse to form the Achilles tendon [2].

The distal pattern of the gastrocnemius belly is unipennate, regular and homogenous, with fibers oriented anteriorly-inferiorly which can facilitate imaging analysis, in particular by ultrasound.

21.2.2 Tennis Leg

21.2.2.1 Pathophysiology

Myotendinous strains of the distal gastrocnemius are the most frequent injuries detected in the posterior calf. They affect mostly the medial gastrocnemius (60 %). The high frequency is due to the biarticular muscle pattern, and the predominance of IIb muscle fibers (fast action fibers) [1, 3].

Most injuries occur during sudden muscle lengthening against a contracting muscle, referred to as eccentric contraction. Injuries due to eccentric contraction damage the muscle more severely than those related to concentric contraction and mainly affect type II muscle fibers. [4–6]. Furthermore the medial head is more commonly injured than the lateral head, as it has been shown to be more active (Accessory function of internal rotational and adductor of Achilles complex) [7, 8].

The severity of the injury depends on three parameters: the degree of stretching, and the velocity and force of the trauma [9, 10].

This injury has been described in young athletes during periods of heavy exercise (such as basketball, running, or severe stretching) and in more elderly patients during

simple daily movements [5].

21.2.2.2 Clinical Signs

Patients feel a sudden onset of pain with dramatic loss of function and cessation of sport activity. Focal swelling and ecchymosis of the calf can appear during the following 24 h [11].

21.2.2.3 Imaging Diagnosis

Owing to its wide accessibility and low cost, ultrasound examination comes first. The best time for ultrasound is between two and 72 h after the trauma [8, 12]. Ultrasound usually shows, on sagittal view, a loss of striated muscle structure, replaced by a heterogeneous hypo- or anechoic area due to a gap in the muscle fiber, with proximal retraction of the torn muscular fibers. This injury is usually located at the distal muscle fibers close to the myo-aponeurotic junction. A hyperechoic lesion can be noted in low grade injuries [13, 14].

If the lesion seems large on sagittal view, a complementary axial view allows differentiation of partial tears from complete ruptures (grade 2 vs 3).

Posteromedial lesions of the medial gastrocnemius can be underdiagnosed. It is important therefore to closely examine the innermost part of the muscle.

Inter-aponeurotic hematoma is frequently associated with large tears. Delgado indicated that sometimes this collection can occur without evidence of rupture of muscle components of the triceps surae (Fig. 21.1) [1].

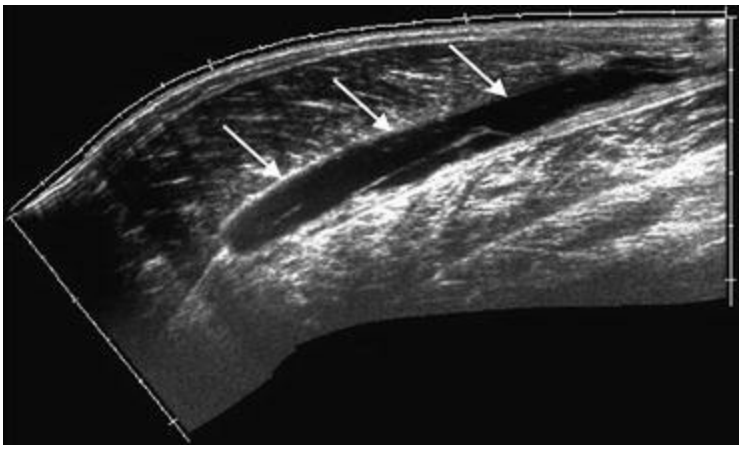

Fig. 21.1 A 20-year-old male tennis player with acute medial calf pain. Longitudinal ultrasound image reveals a large hematoma (*arrows*) between the soleus and the medial gastrocnemius aponeurosis with distal myoaponeurotic injury of medial gastrocnemius

Large muscle lesions, inter-aponeurotic collections or muscle retraction suggest a poor prognosis.

With an acute large inter-aponeurotic collection, percutaneous needle aspiration will help aponeurotic scar adhesion. If the collection recurs, the treatment can be repeated during the first 3 weeks of convalescence. If the hematoma has coagulated ultrasound guidance is recommended, between the seventh and 21st day after injury, to verify the potential secondary liquefaction.

Its availability, low cost, ease of use and sensitivity make ultrasound superior to MRI for initial diagnosis and follow-up of lesions. MRI is usually recommended only for high-level athletes (Fig. 21.2).

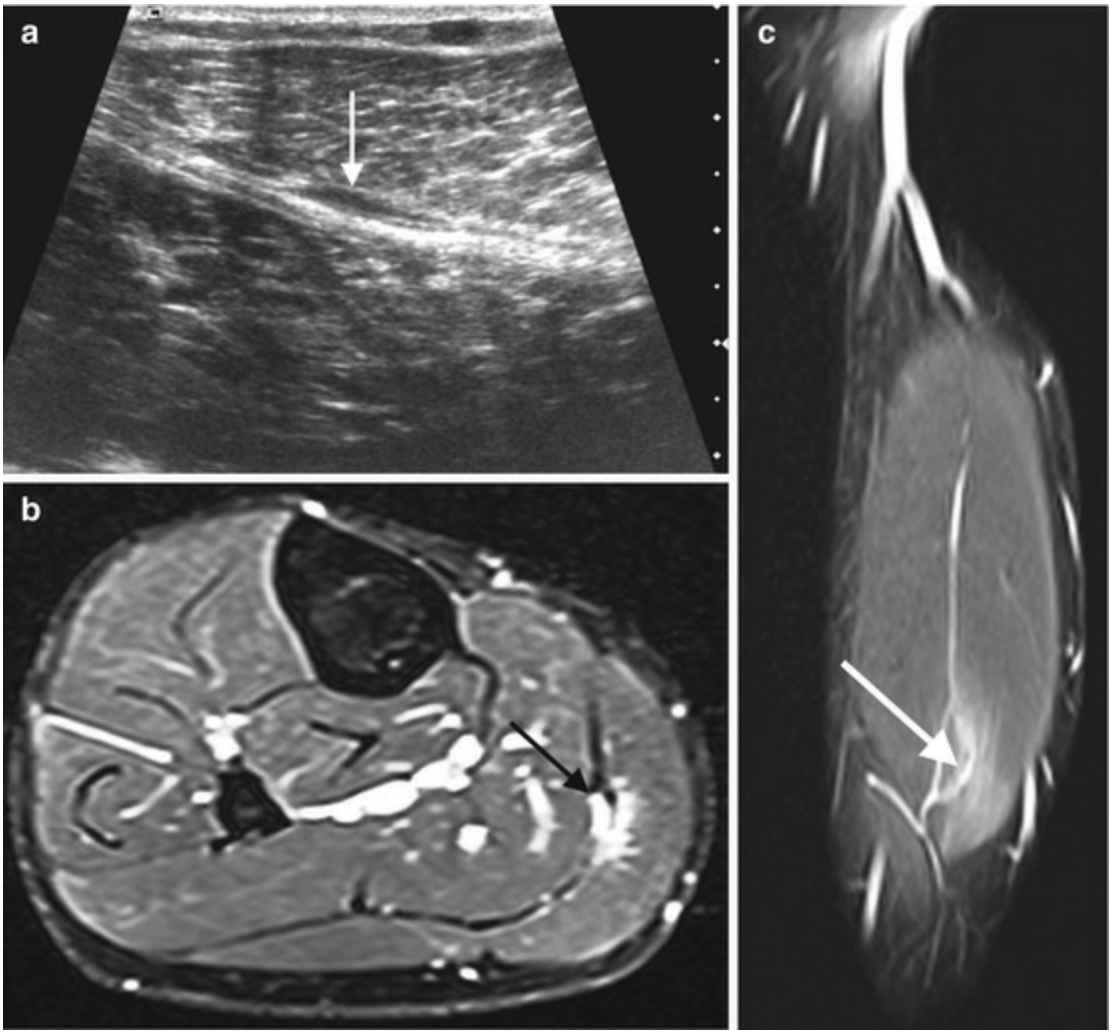

Fig. 21.2 A 28-year-old athlete with acute medial calf pain. (**a**) Longitudinal ultrasound image shows a focal aponeurotic thickening of the medial gastrocnemius (*arrow*). Coronal (**b**) and axial (**c**) fat-suppressed T2-weighted MRI confirm grade 1 medial gastrocnemius aponeurotic injury (*arrow*)

Fat saturated T2 sequences reveal intramuscular and aponeurotic high signal intensities that indicate injury. The inter-aponeurosis collection can be measured precisely, and other associated muscular lesions can be detected. Koulouris found that in a third of these patients, dual site injuries involve the medial gastrocnemius and the

soleus muscle (Fig. 21.3) [15].

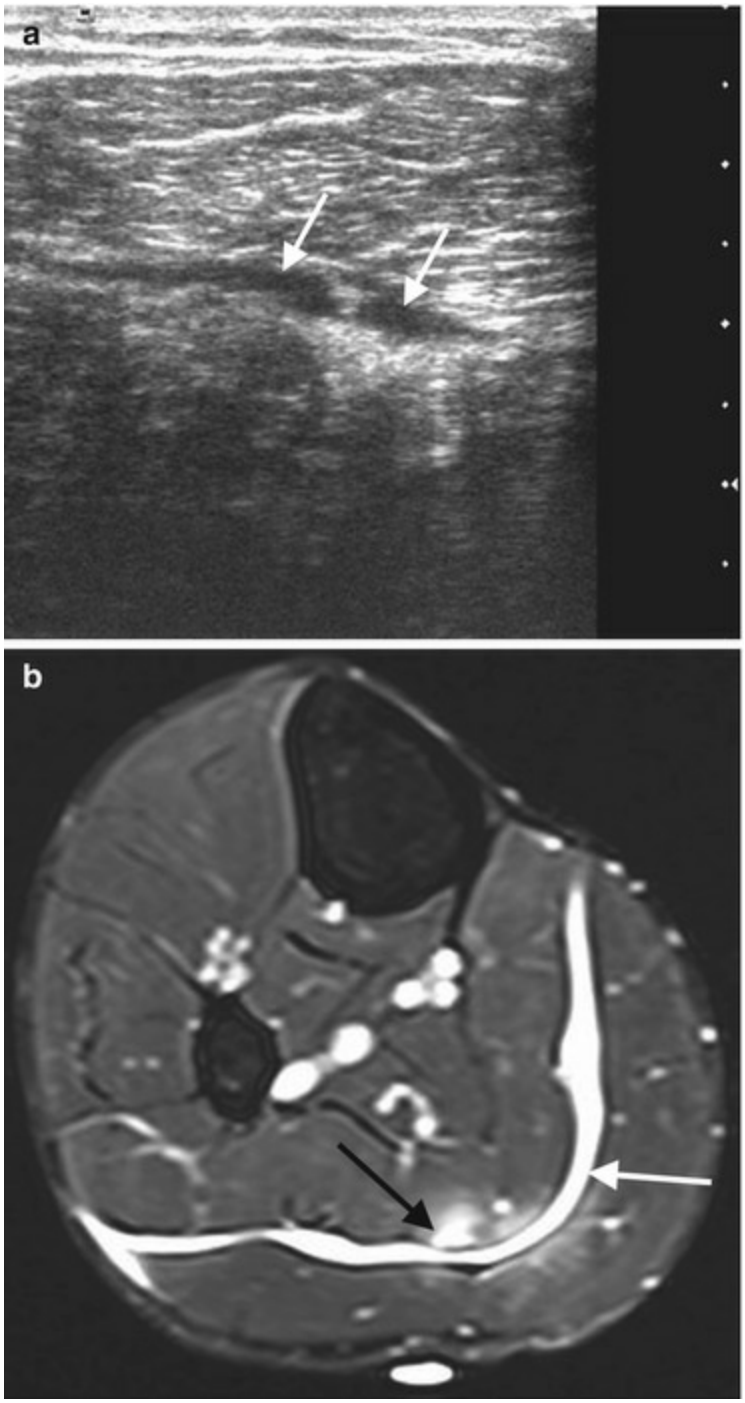

Fig. 21.3 A 26-year-old man with acute right calf pain. Transverse ultrasound image (**a**) and axial fat-suppressed T2-weighted MRI (**b**) show musculo-aponeurotic injury of the medial gastrocnemius-soleus muscles complex (*black arrow*) with edema at the musculofascial junction of the soleus and hematoma (*white arrows*)

21.2.2.4 Other Medial Gastrocnemius Muscle Injuries

Injury to the proximal myotendinous junction of the gastrocnemius posterior to the knee can occur but it is an unusual clinical entity and its imaging appearance is rarely

reported. It seems to be related to knee instability injury with posterior angle injury. The clinical significance of proximal injuries is also unclear aside from localization of pain to the knee instead of the midcalf, which can alter the differential diagnosis [11].

Bony avulsion of the proximal insertion is an unusual diagnosis found in young athletes [16].

21.2.2.5 Lateral Gastrocnemius Lesion

Lateral gastrocnemius muscle lesions are an uncommon occurrence described as symmetric lateral tennis leg. The injury is most frequently located on the myo-aponeurotic junction of the distal fibers. This diagnosis presents no specific imaging findings.

21.2.3 Soleus Muscle

21.2.3.1 Anatomy

The soleus is broad and bulky, located superficially to muscles of the deep posterior compartment (flexor hallucis longus muscles, tibialis posterior, and flexor digitorum longus,) and deeply to the gastrocnemius and plantaris.

The soleus has two distinct proximal tendinous fibular and tibial origins. The fibular arises on the posterior aspect of the head. The tibial origin is at the inferior border of a tibial bony ridge called the tibial soleal line [2, 17].

Both proximal tendinous fibers merge to form the tendinous arch of the soleus, and extend proximally into the soleus belly to form a tendinous lamina called the intramuscular aponeurosis of the soleus. The soleus muscular fiber originates proximally on the latter and extends inferiorly, forming a bipennate pattern. The angle of pennation seems to be sex specific, with higher angle pennation in the male soleus muscle [18].

The majority of soleus fibers originate on the posterior aspect of the intramuscular aponeurosis and converge downward to finish on the anterior aspect and on the borders of a new tendinous lamina, the insertion lamina.

The anterior muscle fibers originate on the anterior aspect of the intramuscular aponeurosis and form distally a central intramuscular tendon.

In the distal segment of the soleus muscle two medial and a posterior sagittal intramuscular aponeurotic band converge with the insertion lamina [8, 19, 20]

This insertion lamina, central muscular tendon and sagittal and medial aponeurosis converge and fuse with the tendinous lamina issuing from the gastrocnemius muscle to form the Achilles tendon.

21.2.3.2 Pathophysiology

The role of the soleus muscle is to maintain posture and to assist low energy activities like walking. Moreover the soleus is the main plantar-flexor muscle of the foot and the most powerful muscle of those crossing the ankle.

The lesser susceptibility of soleus to musculotendinous lesions is due to its monoarticular pattern and its composition (96 % of soleus muscle fibers are type I) [21].

Soleus injuries occur when the ankle is passively dorsiflexed while the knee is flexed.

Balius in a recent work revealed the propensity of proximal strains to occur along the medial side, probably due to the increased length of the intramuscular tendon and a larger musculotendinous junction. It can also be explained by the fact that during a plantar-flexor movement the medial head provides 71 % of its force, while the lateral head contributes only a small part [21, 22].

21.2.3.3 Clinical Signs

Soleus injuries usually appear as a moderate diffuse pain with subacute presentation, resembling delayed onset muscle soreness, probably because the soleus is composed predominantly of type I fibers. The difficulty of distinguishing among gastrocnemius, soleus, plantaris, and proximal Achilles tendon injuries and soleus lesions is usually underestimated.

Furthermore, under-appreciation of soleus strains has likely played a role in the apparently decreased incidence of soleus injuries, which can be attributed in part to the traditional role that ultrasound has played as the imaging modality of choice. Owing to the gradual onset of the injury and the fact that it is usually well tolerated, it is felt that the frequency of soleus lesions is underestimated. Soleus injuries are easily detected on MRI [15, 21].

21.2.3.4 Imaging Diagnosis

Soleus injury semiology and staging is overall the same as for gastrocnemius strains. On the other hand soleus injuries seem to be frequently underdiagnosed on ultrasound. Subtle grade 2 strains, where minimal macroscopic fiber disruption may be the only evident imaging feature (and where hemorrhage is absent), may be missed on ultrasound. Given the deeper location of the soleus muscle, resulting in relatively poorer image quality, and the anatomic complexity, it is likely that low grade and therefore subtle strains are missed, with only higher grade injuries visualized [18, 23].

As Balius noted in a recent study, the variability of injury localization makes ultrasound difficult. Nevertheless strains of the proximal medial musculotendinous junction are the most common type of soleus muscle injuries (56.4 % of all cases) [21, 22].

During ultrasound examination special attention should be given to detection of architectural anomalies of the central muscular and peripheral myo-aponeurosis junction. An isolated collection located in the inter-aponeurosis soleus-gastrocnemius space suggests careful examination of the soleus [1].

Thus, MRI is the best method to investigate injuries of the soleus (Fig. 21.4). Koulouris showed that the high prevalence of soleus injuries on MRI and the frequent association between different calf muscle injuries suggests that many cases are probably missed on ultrasound (Figs. 21.5 and 21.6) [15, 24].

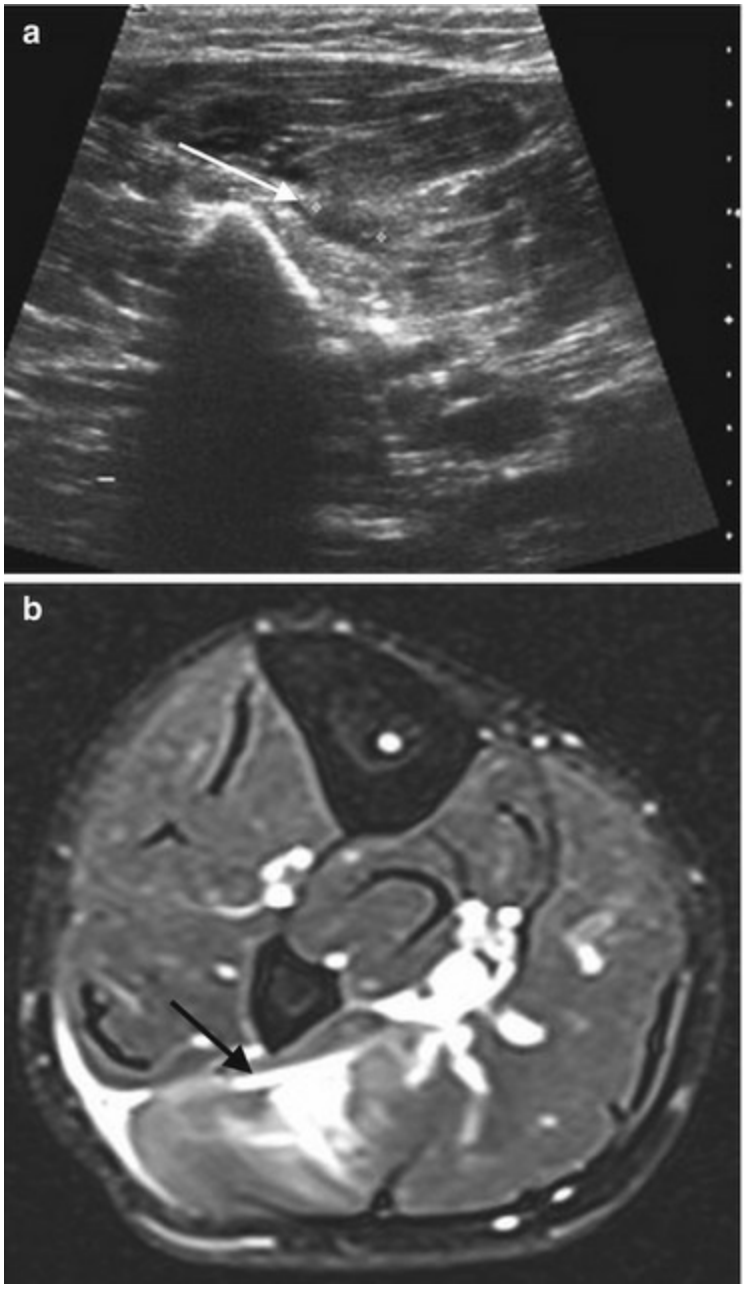

Fig. 21.4 A 26-year-old man with moderate right calf pain. Transverse ultrasound image (**a**) shows a 5 mm hypoechoic area in the soleus muscle (*arrow*). (**b**) Axial fat-suppressed T2-weighted MRI confirms grade 2 soleus muscle injury (*arrow*)

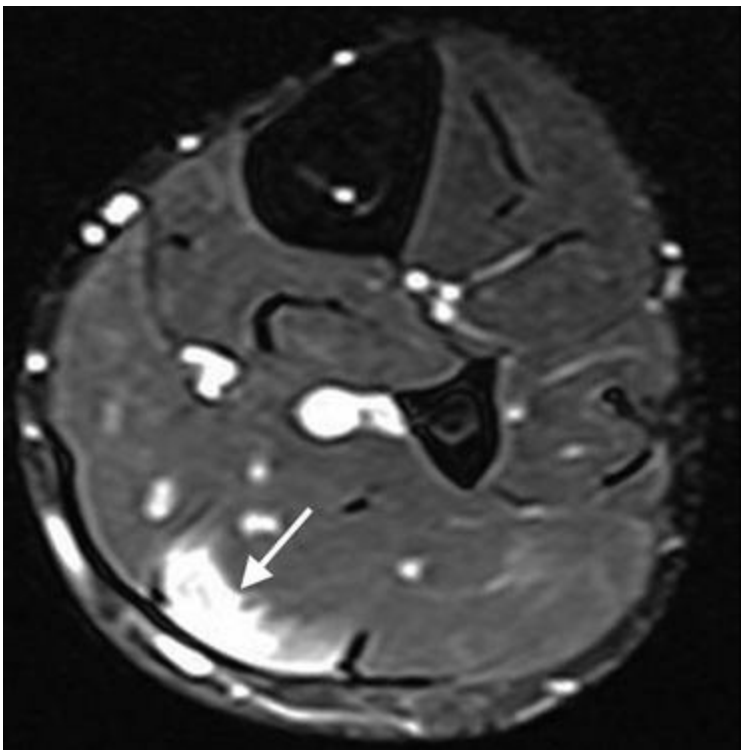

Fig. 21.5 A 23-year-old man with acute left calf pain that started during a soccer match. Axial fat-suppressed T2-weighted MRI shows grade 2 injury soleus muscle and grade 1 injury of the lateral gastrocnemius muscle (*arrow*)

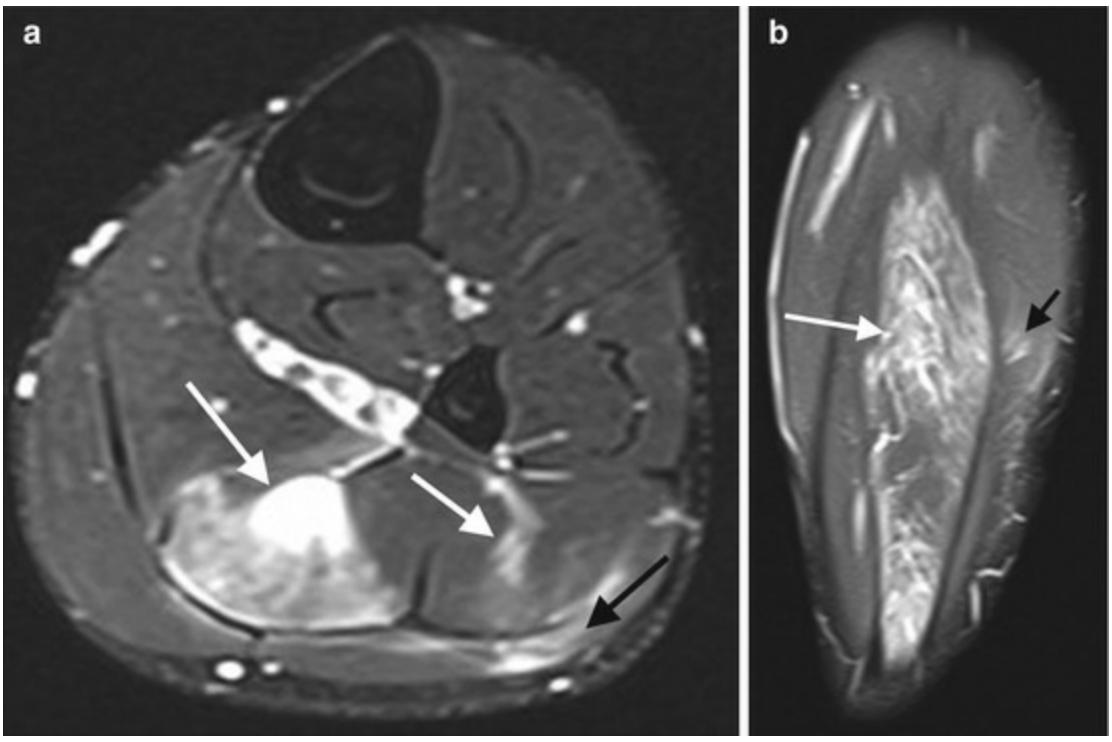

Fig. 21.6 A 24-year-old athlete with acute calf pain. Axial (**a**) and coronal (**b**) fat-suppressed T2-weighted MRI show a bifocal musculo-aponeurotic strain of the soleus (grade 2, *white arrows*) and lateral gastrocnemius muscles (*black arrow*)

Owing to the gradual onset of the injury and the fact that it is usually well tolerated, the frequency of soleus lesions is probably underestimated. Although soleus injuries are easily detected on MRI, owing to their good prognosis and the expense of MRI it is usually recommended only for high-level athletes

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21.3 Plantaris Muscle

21.3.1 Anatomy

The plantaris muscle is the smallest muscle of the calf. It is missing in as much as 7–20 % of the normal population [25].

The plantaris arises from the supracondylar lateral area of the femur, with a short muscle body located between the aponeurosis of the soleus and lateral gastrocnemius, continues by a thin tendon distally located at the anteromedial border of the Achilles tendon and inserts into the flexor retinaculum or calcaneus. Its distal insertion is separated from the Achilles tendon.

In relation to its small size the mechanical role of the plantaris muscle (flexor of the knee, plantar flexor of ankle) is weak. Nevertheless itis an agonist of the anterior cruciate ligament and participates at the posterolateral complex of the knee.

21.3.1.1 Normal Imaging

Ultrasound examination shows a fibrillar structure, with a hypoechoic belly muscle. The tendon appears in the center of the muscle belly as a hyperechoic structure with a myotendinous junction situated at the proximal one third of the calf [14, 26, 27].

On axial images the proximal part of the plantaris muscle tendon appears as a round structure between the soleus and lateral gastrocnemius belly. The distal part of the tendon is less well defined due to the hyperechoic fat environment [28].

This fat environment provides good contrast on MRI axial T1 and T2 images without fat saturation. The tendon appears as a rounded structure with well-defined borders in contact with the anteromedial Achilles tendon border.

21.3.1.2 Pathophysiology

Lesions are most common in the myotendinous junction of the plantaris, arising during eccentric contractions. Ruptures of the distal tendon or proximal belly lesions have also been reported [29, 30].

The distal tendon and myotendinous lesion seem to appear in the same biomechanical situation as tennis leg. On the other hand lesions of the proximal belly correlate with anterior cruciate ligament rupture and lesions of the posterolateral corner [1, 31–34].

21.3.1.3 Clinical Signs

Patients usually describe a strong acute pain of the upper calf or popliteal area that mimics an Achilles tendon injury with preserved plantar flexion and negative Thompson test.

The main diagnoses are Achilles tendon rupture, tennis leg, thrombophlebitis, and acute Baker cyst rupture. Acute lesions of the plantaris muscle itself and medial gastrocnemius are rare.

21.3.1.4 Imaging Diagnosis

Usually tears of the plantaris tendon are located on average 5 cm above its distal insertion and appear as a short discontinuity of the tendon (about 1 cm) (Fig. 21.7) [28].

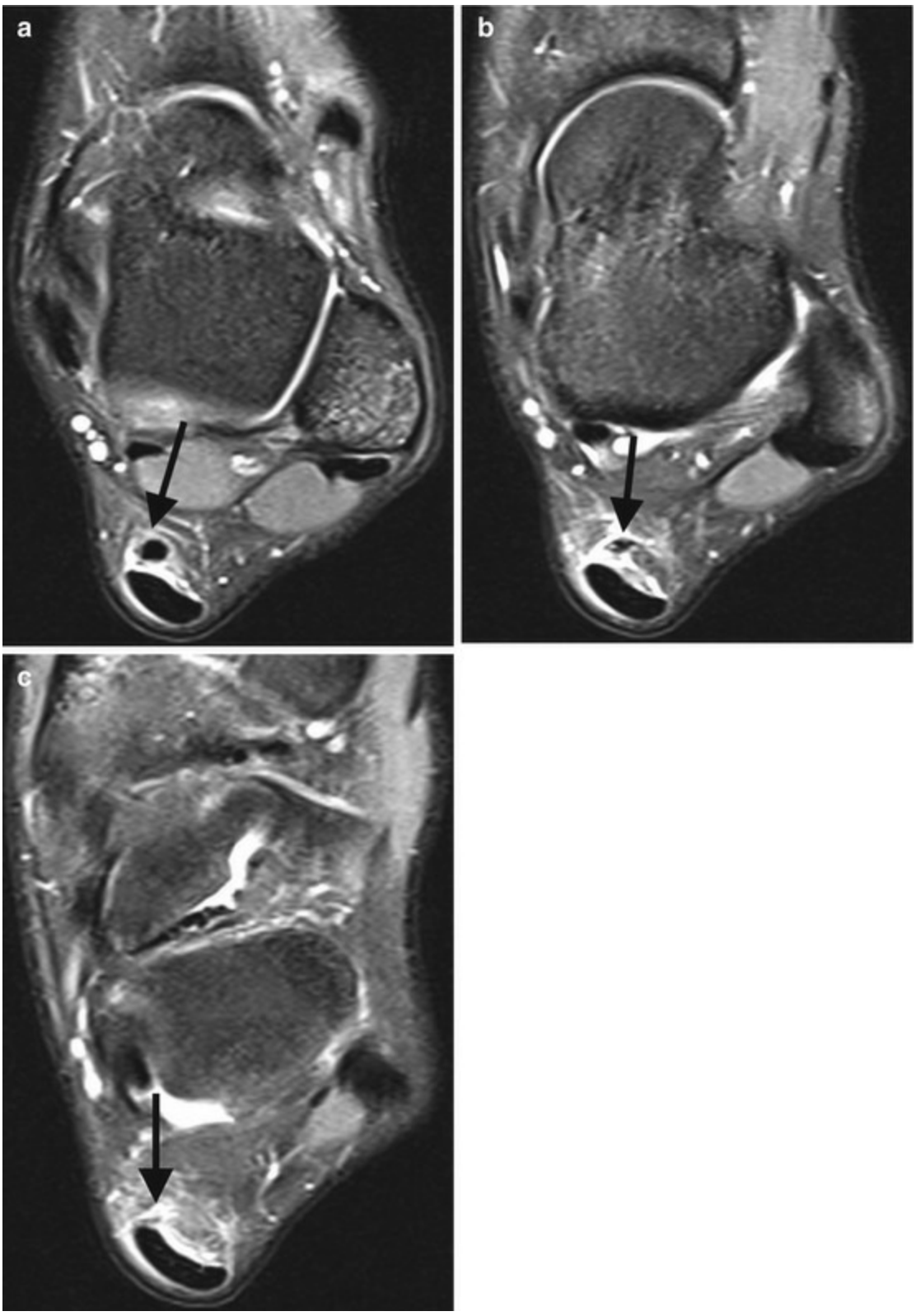

Fig. 21.7 A 15-year-old athlete with acute Achilles tendon pain and an isolated tear of the plantaris muscle tendon. (**a**–**c**) Axial fat-suppressed T2-weighted MRI show peritendinous Achilles edema with discontinuity of the distal segment of plantaris muscle tendon (*arrow*)

The diagnosis on ultrasound is based on the disappearance of the fibrillar structure

of plantaris tendon and its replacement by a hypoechoic swollen heterogeneous area. The end of the proximal tendon appears thickened. A thin hypoechoic liquid area can be seen between the aponeurosis of the medial gastrocnemius and soleus

MRI axial sequences confirm the discontinuity of the tendon with a focal hypersignal T2 area best seen on T2 images with fat saturation. On ultrasound, the proximal end of the tendon can appear thickened. Edema of the muscular body can be associated with a myotendinous lesion (Fig. 21.8) [26, 27, 34–38].

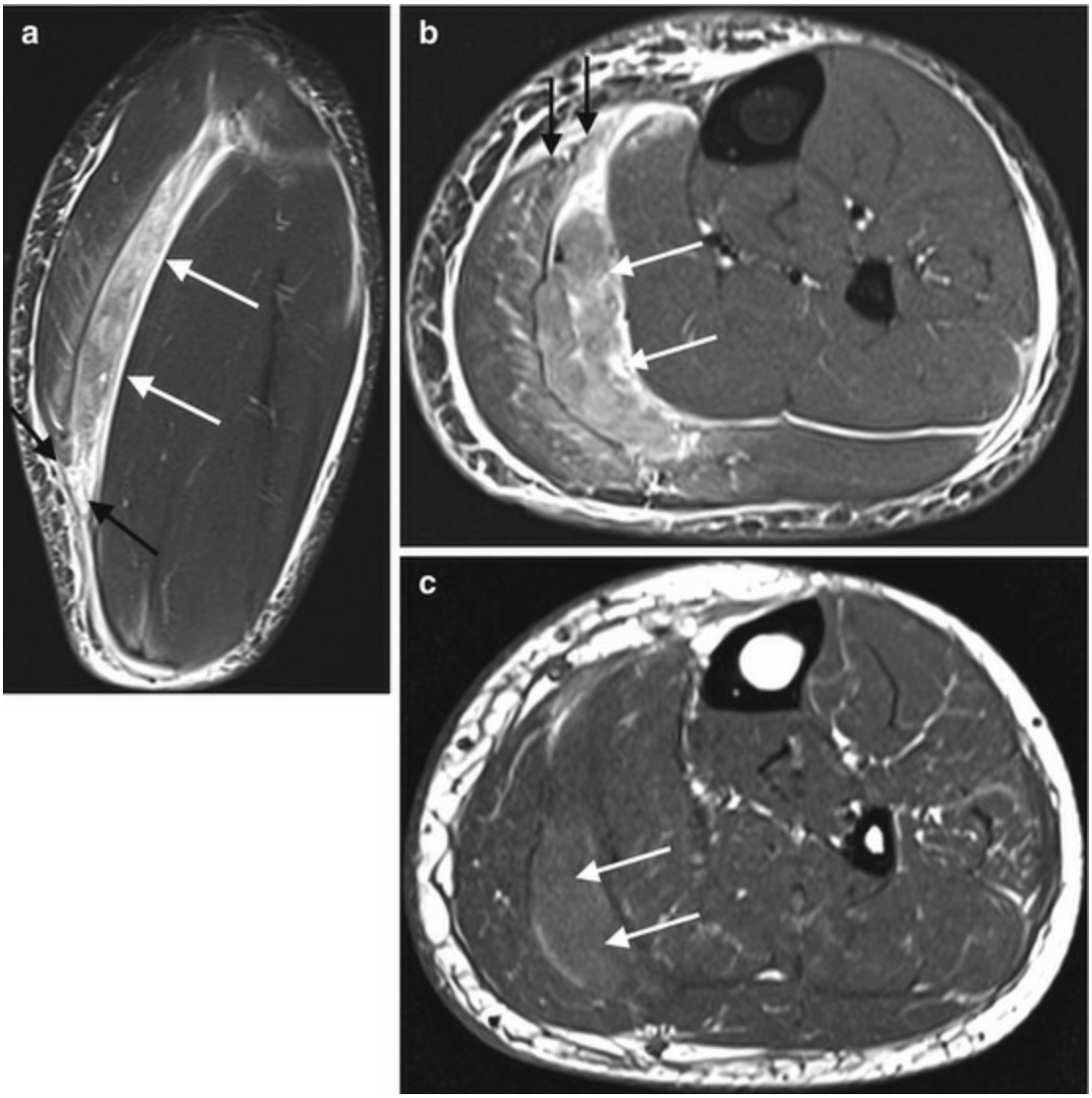

Fig. 21.8 A 42-year-old male athlete with subacute left calf pain. Sagittal (**a**) and axial (**b**) fat-suppressed T2- and axial T1-weighted (**c**) MRI reveal left acute plantaris muscle injury (grade 2, *black arrows*) with aponeurotic hematoma (*white arrows*)

21.4 Popliteus Muscle

The popliteus muscle originates at the posteromedial aspect of the proximal tibial

metaphysis and can have several attachments, but inserts primarily on the lateral aspect of the femoral condyle. The popliteus muscle is an internal rotator of the tibia on the femur and assists in flexion of the knee. It is an important stabilizer of the posterolateral corner of the knee and prevents forward translation of the femur on the tibia [39].

The overwhelming majority of injuries to the popliteus muscle occur in the muscle belly and myotendinous junction rather than its insertion, although this can occur [32]. The mechanism of injury is thought to be from a direct blow to the anteromedial aspect of the proximal tibia as the knee is hyperextended. Without contact, injury can occur with external rotation and hyperextension.

Most injuries to the popliteus are found in conjunction with injuries to other structures in the knee, most commonly the anterior cruciate ligament, with associated injuries to the posterior cruciate ligament, menisci, or collateral ligaments also reported. A small minority of popliteus injuries occur in isolation [40, 41].

Tears range from partial interstitial to complete rupture. MRI will reveal enlargement of the muscle with increased signal on T2-weighted images. With complete rupture, there will be retraction and clumping of the muscle and possible formation of a hematoma in the proximal calf. In this situation, a hematoma can compress the neurovascular bundle in the proximal calf, causing temporary compromise of the posterior tibial nerve. Recently a case of popliteus strain with muscle edema and enlargement resulted in a permanent partial deficit of the tibial nerve [41, 42].

21.5 Other Calf Muscle Injuries

The flexor hallucis longus, flexor digitorum longus and tibialis posterior compose the deep compartment of the calf. Calf injury of these muscles is unusual due to their mono articular pattern and relatively minor biomechanical role. Koulouris reported one case of an injury involving the soleus and flexor hallucis [15]. This muscle is rarely of concern.

21.6 Differential Diagnostic and Complications of Calf Injury

21.6.1 Popliteal Cyst Rupture

An acute rupture of a popliteal cyst can mimic a muscular injury of the calf, with sharp posterior or posteromedial pain, swelling and sometimes redness. Ultrasound shows a poorly-defined popliteal cyst between the semimembranosus and proximal medial gastrocnemius tendon with fluid diffusion in the posterior subcutaneous fat planes and in the fascial planes between the soleus and gastrocnemius muscles, without muscle anomaly [8].

21.6.2 Deep Venous Thrombosis

Traumatic or atraumatic calf pain requires Doppler ultrasound to eliminate the possibility of deep venous thrombosis, which can mimic calf muscle injury. On the other hand calf muscle injuries decrease venous return and favor deep venous thrombosis; thus gastrocnemian venous thrombosis secondary to myoaponeurotic disjunction is frequent.

21.6.3 DOMS

Muscle edema may occur as a result of exercise. Post-exercise imaging of muscle demonstrates increased extracellular fluid and T2 signal and is referred to as exercise enhancement. Delayed onset muscle soreness (DOMS) is a clinical entity of muscle soreness usually due to eccentric contractions [43, 44].

The appearance on MRI is similar to a low-grade muscle injury with diffuse increased T2 signal of muscle. Because it looks so much like a muscle strain it is often diagnosed clinically. Severe DOMS can progress to muscle necrosis with marked increased signal on fluid-sensitive sequences and increased CPK in blood [45].

21.6.4 Neurogenic Muscle Hypertrophy

Neurogenic muscle hypertrophy is usually related to muscle denervation with a previous history of low back pain and sciatica due to lumbar stenosis, disk herniation and, rarely, radiation therapy or trauma. The symptoms of neurogenic muscle hypertrophy are commonly observed in men aged 30–60, with painful enlargement of the calf [46].

21.6.5 Compartment Syndrome

Acute compartment syndrome occurs in the calf following muscle injury, hematoma, or fracture [24, 47]. Nerve and muscle ischemia is secondary to increased pressure within fascial compartments that leads to reduced capillary circulation. MRI may be used to evaluate the extent of involvement and to demonstrate increased T2 signal with muscle enlargement or edema [48]. Clinical diagnosis is confirmed with compartment pressure measurement.

In chronic compartment syndrome MRI after exercise may reveal increased T2 signal in the muscle compartment [49, 50]. Direct pressure measurement of the compartment is a complementary exam that can confirm the diagnosis [51].

21.7 Chronic Post Traumatic Lesion

Three types of chronic lesion can be ascribed to anomalies of the healing process.

21.7.1 Fibrotic Granuloma

Fibrotic granuloma appear hyperechoic and poorly-delineated, without posterior absorption and with loss of normal muscle pattern. They can be located, without Doppler, close to the myo-aponeurotic junction.

A dynamic study should be performed to detect local adherence. Note that it is important to describe very carefully the relationship between the fibrotic granuloma and the adjacent peripheral nerve.

The detection of multiple intralesional Doppler signals corresponds to subacute injury and can be called active fibrosis (Fig. 21.9) [8, 9].

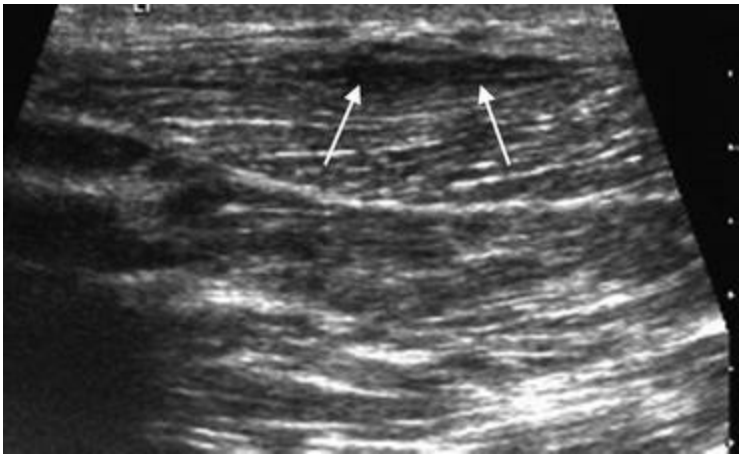

Fig. 21.9 A 21-year-old athlete with right tennis leg symptoms 1 month after a gastrocnemius strain. Sagittal ultrasound image shows heterogeneous aponeurotic thickening consistent with fibrotic granuloma (*arrows*)

21.7.2 Muscular Calcification

Hyperechoic lesions with posterior acoustic absorption may be seen during muscular ultrasound monitoring.

Two types can be described: calcified scar fibrosis and myositis ossificans.

- The first corresponds to an abnormal repair process thought to be associated with high grade muscular lesions and large hematomas. This anomaly is a fixed scar state and can be painful during muscle use.
- Acute painful swollen muscles with inflammatory clinical presentation is more typical of the first phase of myositis ossificans. This entity corresponds to a reaction of interstitial conjunctive tissue of muscle with heterotopic bony and cartilage growth. Thin muscular calcification can be detected with CT as soon as 2 weeks after the first clinical sign. Symptoms regress gradually while intramuscular ossification with well-defined bony margins appears. Spontaneous fusion with adjacent bone can be observed during the first year.

21.7.3 Encysted Hematoma

Delineated by a sclerotic rim, encysted hematomas appear as a hypoechoic fluid intramuscular or aponeurotic collection.

21.8 Conclusion

Posterior calf pain is frequently presented in clinical practice. The physician must be aware of the difficulty of making a diagnosis due to the low specificity of the clinical signs and possible differential diagnoses. Owing to its accessibility and low cost, ultrasound is the first step for imaging evaluation. Nevertheless, for professional athletes MRI remains the “gold standard” in particular to detect soleus or plantaris muscle lesions that can be underdiagnosed with ultrasound, and are often associated with lesions of the gastrocnemius.

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Part V
Clinical Cases

Bernard Roger, Ali Guermazi and Abdalla Skaf (eds.), Muscle Injuries in Sport Athletes, Sports and Traumatology,
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22. Muscular Endometriosis

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Abstract
Ectopic endometriosis is the presence of endometrial tissue outside the uterine cavity with proliferative activity and bleeding and/or fibrosis. Its prevalence is estimated at 5–10 % of the general population, higher in Caucasian women 20–40 years old (Cornilie et al. Fertil Steril 53:978–983, 1990; Eskenasi et al. Obstet Gynecol Clin North Am 24:235–257, 1997).

Muscular and subcutaneous endometriosis are the most frequent extra-pelvic manifestations of endometriosis; metastasis is possible but no prevalence has been clearly described in the literature on patients with pelvic endometriosis. Muscular endometriosis can be spontaneously present in any muscle but has been described particularly in the anterior abdominal wall, especially in the rectus abdominis (Calo et al. Ann Ital Chir, 2012). It can occur spontaneously or secondarily to laparoscopic surgery(Dallaudiere et al. Diagn Interv Imaging 94:263–280, 2013). However, in the literature, all but two cases reported endometriosis in the parietal wall, with one in the pyramidal muscle and one in the adductor compartment(Dominguez-Paez et al. Neurocirugia (Astur) 23:170–174, 2012; Fambrini et al. J Minim Invasive Gynecol 17:258–261, 2010).

22.1 Background

Ectopic endometriosis is the presence of endometrial tissue outside the uterine cavity with proliferative activity and bleeding and/or fibrosis. Its prevalence is estimated at 5–

10 % of the general population, higher in Caucasian women 20–40 years old [1, 2].

Muscular and subcutaneous endometriosis are the most frequent extra-pelvic manifestations of endometriosis; metastasis is possible but no prevalence has been clearly described in the literature on patients with pelvic endometriosis. Muscular endometriosis can be spontaneously present in any muscle but has been described particularly in the anterior abdominal wall, especially in the rectus abdominis [3]. It can occur spontaneously or secondarily to laparoscopic surgery [4]. However, in the literature, all but two cases reported endometriosis in the parietal wall, with one in the pyramidal muscle and one in the adductor compartment [5, 6].

22.2 Cases

Case 1

A 42-year-old woman with chronic cyclic groin pain during menstruation: Parietal endometrioid of the left rectus abdominis muscle.

MRI shows recent hemorrhagic changes in the rectus abdominis (arrow) with high signal intensity on T1-weighted images (Fig. 22.1a), heterogeneous signal on T2-weighted (Fig. 22.1b) and STIR (Fig. 22.1c) images and significant enhancement after gadolinium injection (Fig. 22.1d).

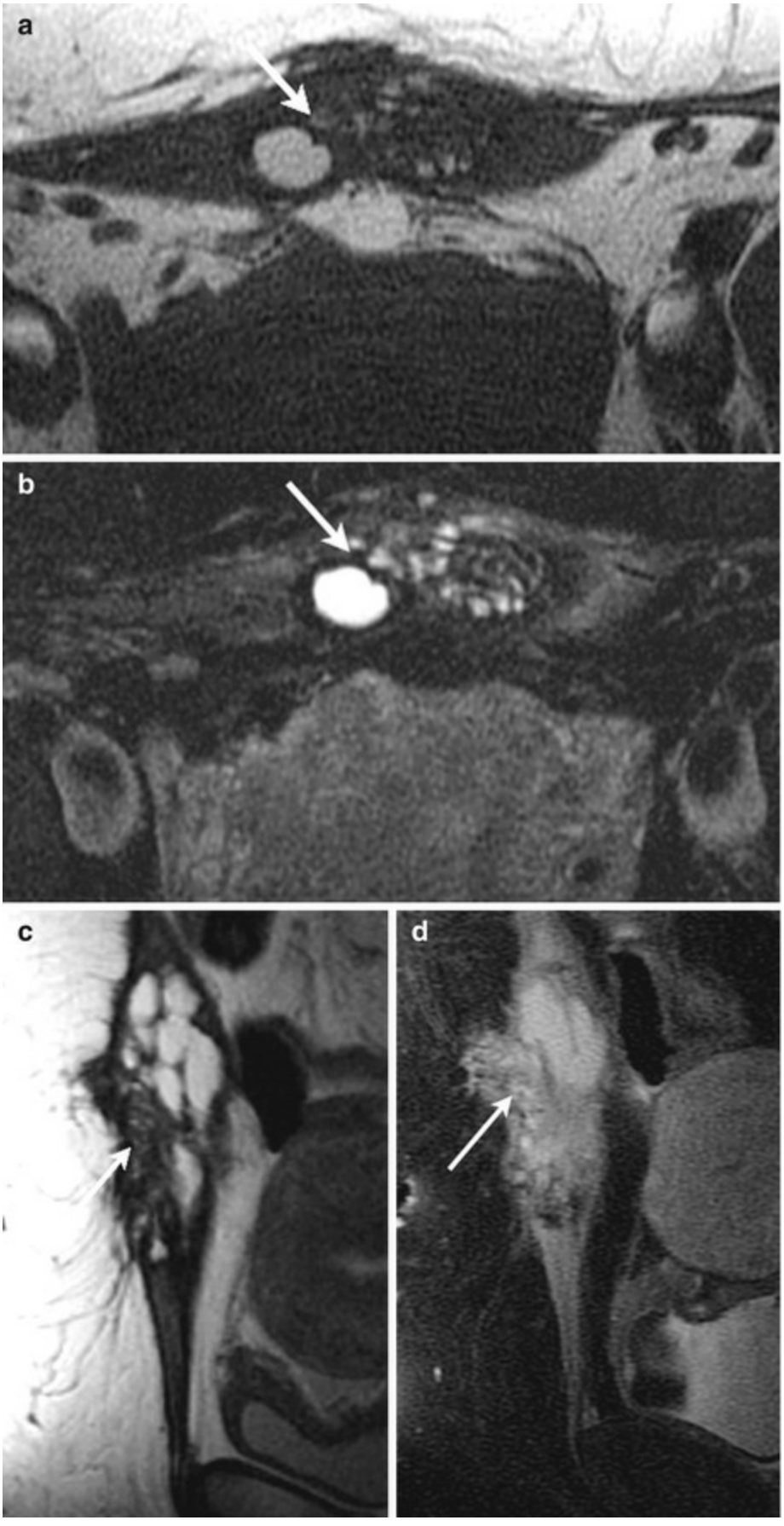
a
b
c
d

Fig. 22.1 A 42-year-old woman with chronic cyclic groin pain during menstruation: Parietal endometrioid of the left rectus abdominis muscle. MRI shows recent hemorrhagic changes in the rectus abdominis (arrow) with high signal intensity on T1-weighted images (a), heterogeneous signal on T2-weighted (b) and STIR (c) images and significant enhancement after gadolinium injection (d).

Case 2

A 35-year-old woman with a parietal mass: Parietal endometrioid in the right rectus abdominis muscle.

MRI shows recent hemorrhagic changes in subcutaneous tissue and the abdominal wall (arrow) with low signal intensity on T1-weighted images (Fig. 22.2a), heterogeneous signal on T2-weighted images (Fig. 22.2b) and significant enhancement after gadolinium injection (Fig. 22.2c).

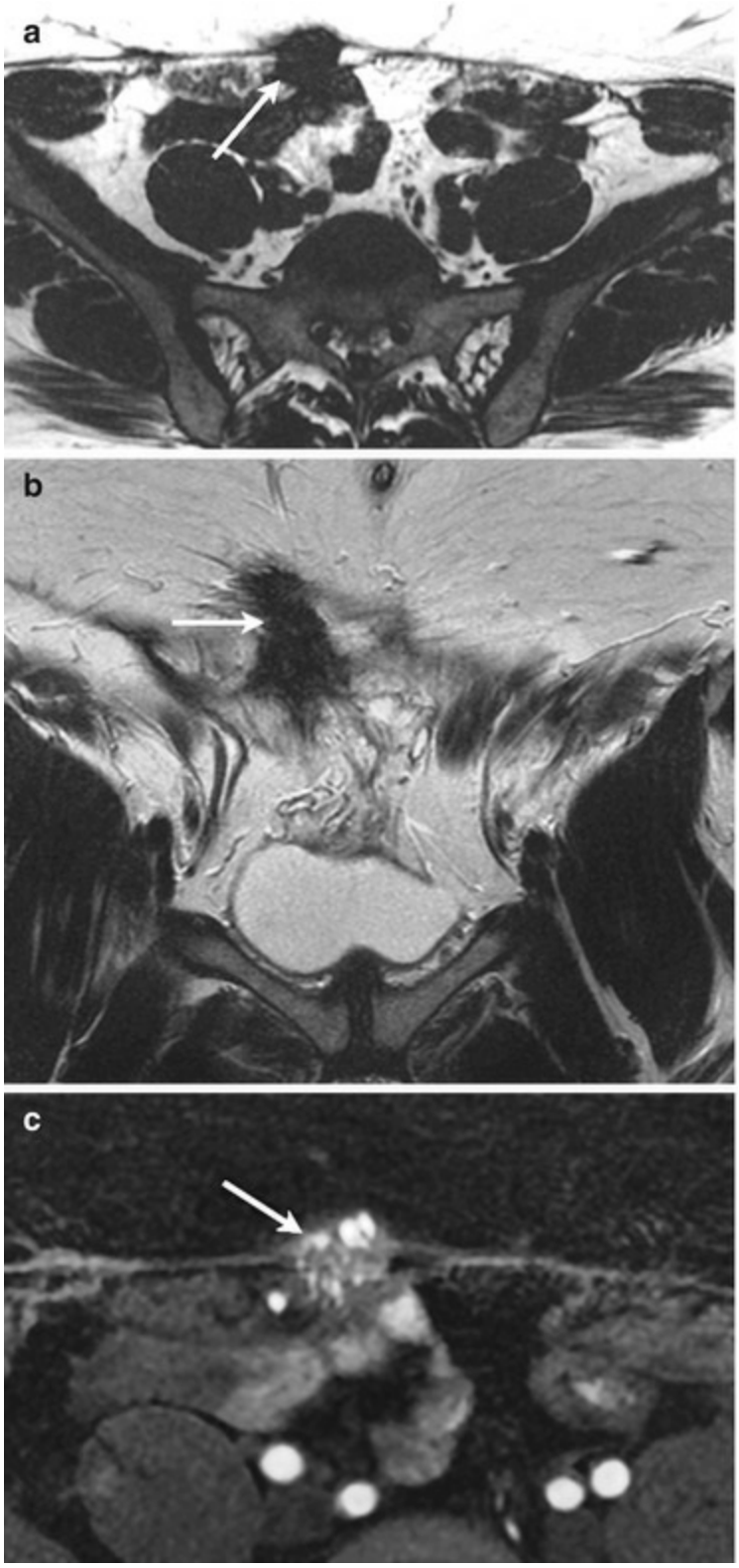

Fig. 22.2 A 35-year-old woman with a parietal mass: Parietal endometrioid in the right rectus abdominis muscle. MRI shows recent hemorrhagic changes in subcutaneous tissue and the abdominal wall (arrow) with low signal intensity on T1-weighted images (a), heterogeneous signal on T2-weighted images (b) and significant enhancement after gadolinium injection (c).

22.3 Discussion and Evaluation

Clinical examination is difficult with groin pain and endometriosis should always be considered if there is a history of chronic cyclic groin pain during menstruation. Painful nodules are due to destruction of the endometrial cells [4]. No nervous impingement has been described in the literature. Medical imaging, in particular MRI, may help in the diagnosis of extra-pelvic endometriosis in the groin muscles [7].

Ultrasound and CT are not specifically used for the diagnosis of muscular endometriosis in clinical practice. However, on CT signs of endometriosis are highly polymorphic, with generally a heterogeneous mass riding on muscle and the skin with or without hyperemia; or an isodense mass with or without contrast enhancement.

MRI allows the definitive diagnosis. On MRI, endometriosis lesions are also very polymorphic and are characterized by variable contents of hemorrhagic and/or fibrous lesions [8].

Hyperintense signal on T1-weighted sequences is noted if there is evidence of recent hemoglobin degradation; hypointense signal on T1-weighted sequences occurs in old bleeding lesions. With T2-weighted sequences, signal is quite variable, generally hypointense.

With strictly fibrous lesions, the site of the endometriosis appears hypointense on T1-and T2-weighted sequences. While the use of contrast enhancement is common for imaging of endometriosis, intravenous injection is usually not necessary. The evolution of endometriosis lesions also varies. It often involves degrading hemoglobin, which goes from hypersignal to hyposignal on T1-weighted sequences, with varying signal on T2-weighted sequences, but diagnosis can be tricky because of iterative bleeding [9]. Ultrasound usually shows an ill-defined, inhomogeneous hyporeflective mass with peripheral vascularization on color Doppler.

Hematoma and desmoid tumor represent the main differential diagnosis because of the form, the polymorphism of the lesions and their location but medical history guides the final diagnosis. Suture granuloma, lymphadenopathy, abscess, incisional hernia, primary or metastatic cancer, lymphoma, lipoma, sarcoma, and subcutaneous or sebaceous cysts can also be evoked [3]. Anatomopathology is not generally given precedence over clinical history when making the diagnosis, except in cases of atypical presentation. If the diagnosis is unclear, histology will reveal cylindrical glandular epithelium and endometrial stroma with reactional inflammatory proliferation (smooth muscle cells and fibroblasts) in endometriosis lesions [4].

Treatment depends on the severity of symptoms. Estrogen-progestin contraception, progestins, GnRH agonists, or non-steroidal anti-inflammatory drugs can be used but danazol remains the medical treatment of choice: it acts on the ovary by blocking the pituitary gonadotropic function. If medical treatment fails, surgery may be considered [10].

22.4 Conclusion

Endometriosis lesions are widely polymorphic with many possible locations but the rectus abdominis is the predominant site and should be considered first in female patients presenting with chronic pelvic groin pain with normal symphysis pubis, adductors and hernial orifice.

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23. Post-traumatic Myositis Ossificans

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23.1 Background

Post-traumatic myositis ossificans (MO) is a benign, heterotrophic ossification in muscle due to trauma of the muscle in the upper and lower limbs e.g. quadriceps and brachialis. Earlier studies suggest that the incidence of MO after a quadriceps contusion was between 9 [1] and 20 % [2]. Ryan et al. [1] suggested that patients with a quadriceps contusion with knee flexion of less than 120°, a previous quadriceps contusion, a sympathetic knee effusion, a football injury and a delay in treatment of more than 3 days have a higher risk of developing MO. The diagnosis can often be difficult as the histopathology and radiological characteristics of MO may be mistaken for those of benign or malignant tumour [3, 4]. It is usually treated conservatively with resumption of normal function [1, 2].

23.2 Case Description

A 15-year-old-male soccer player initially had a contusion to the left vastus intermedius muscle secondary to direct kick. Initial axial ultrasound (Fig. 23.1) showed contusion to the left vastus intermedius (arrows). Patient initially responded well to RICE treatment, but there was a secondary increase of pain and swelling at day 8, which halted exercise therapy and prompted additional imaging. Lateral radiograph of the left thigh (Fig. 23.2a) showed a subtle increase in density to the left quadriceps muscle (arrows). Axial ultrasound image (Fig. 23.2b) showed an increase in size of the muscle contusion

(arrows). Sagittal fat-suppressed T2-weighted MRI (Fig. 23.2c) showed diffuse edema and multiple heterogeneous collections within the vastus intermedius muscle (arrows). A repeat ultrasound examination was performed at 4 weeks from the initial trauma. Axial (Fig. 23.3a) and sagittal (Fig. 23.3b) ultrasound images showed extensive hyperechoic rim with posterior shadowing (arrows) related to ossification at the expected position of the muscle contusion, in keeping with myositis ossificans. Repeat lateral radiograph of the left thigh at 6 weeks (Fig. 23.4) and 14 weeks (Fig. 23.5) showed extensive ossification of the vastus intermedius measuring approximately 14 cm length, predominantly peripheral at 6 weeks, and more compact at 14 weeks.

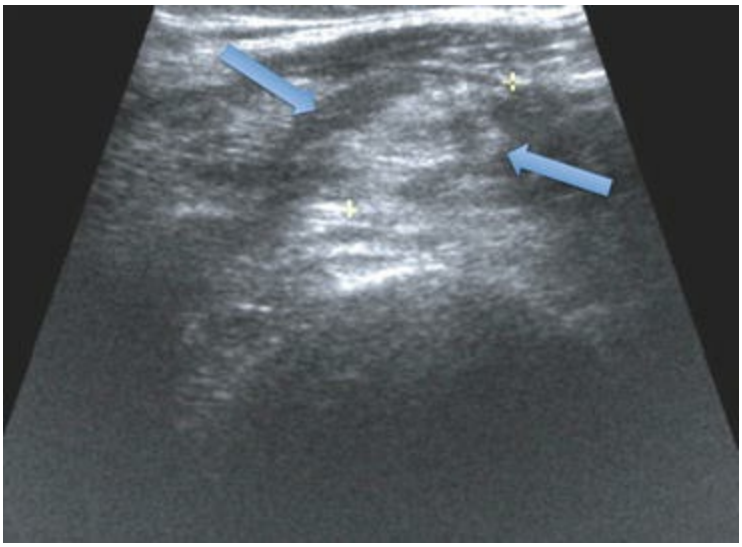

Fig. 23.1 15-year-old-male soccer player 8 days after direct contusion on the thigh. Axial ultrasound shows an ill-defined area of hyperechogenicity (*arrows*) within the vastus intermedius muscle consistent with contusion

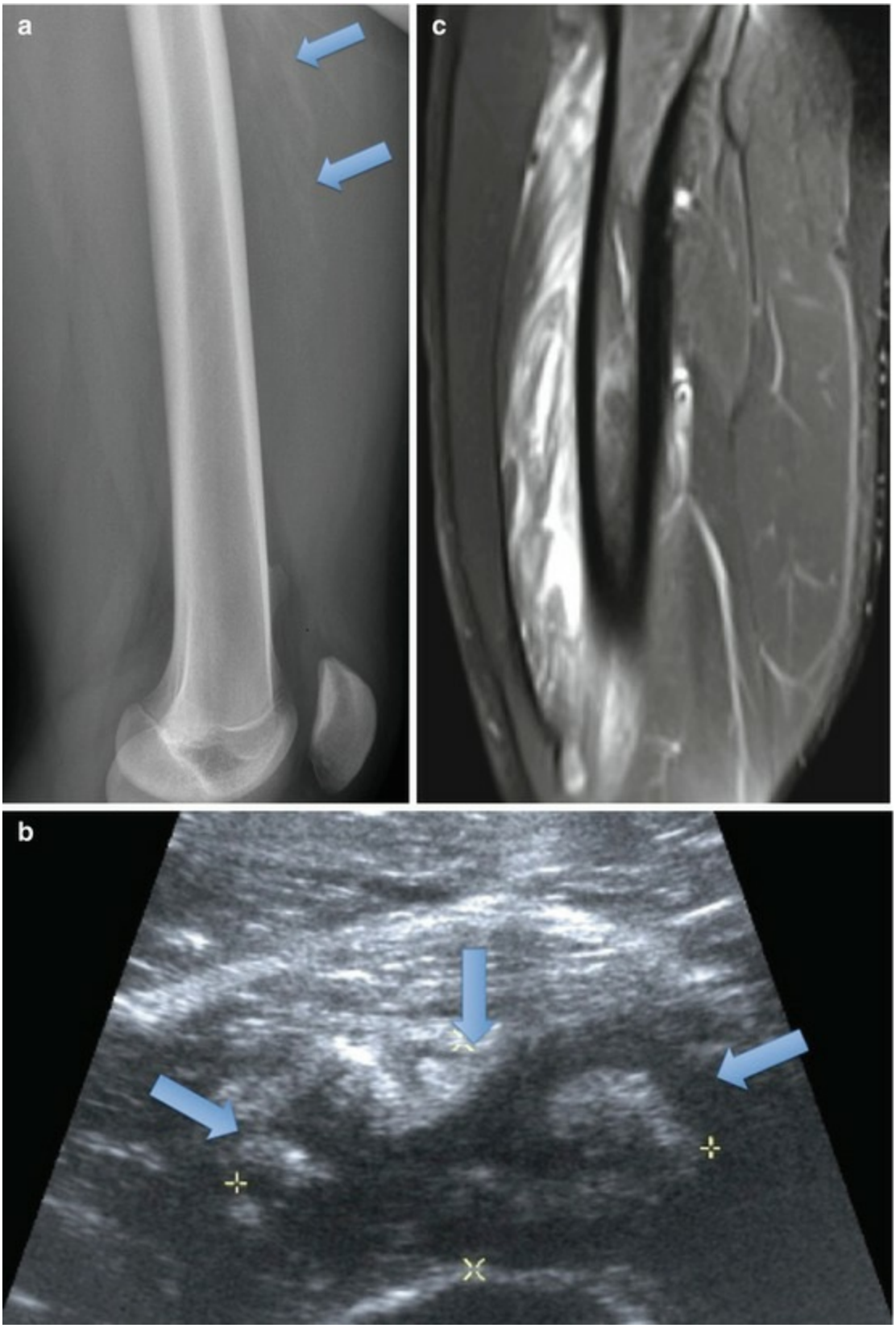

Fig. 23.2 15-year-old-male soccer player 8 days after direct contusion on the thigh. (**a**) Lateral radiograph of the left thigh shows a subtle laminar increased density (*arrows*) in the left vastus intermedius; (**b**) Axial ultrasound image shows irregular outlined muscle lacerations separated by hypoechoic fluid (*arrows*), consistent with muscle contusion and areas of haematoma. (**c**) Sagittal fat-suppressed T2-weighted MRI shows diffuse muscle edema and multiple

heterogeneous fluid collections within the vastus intermedius muscle

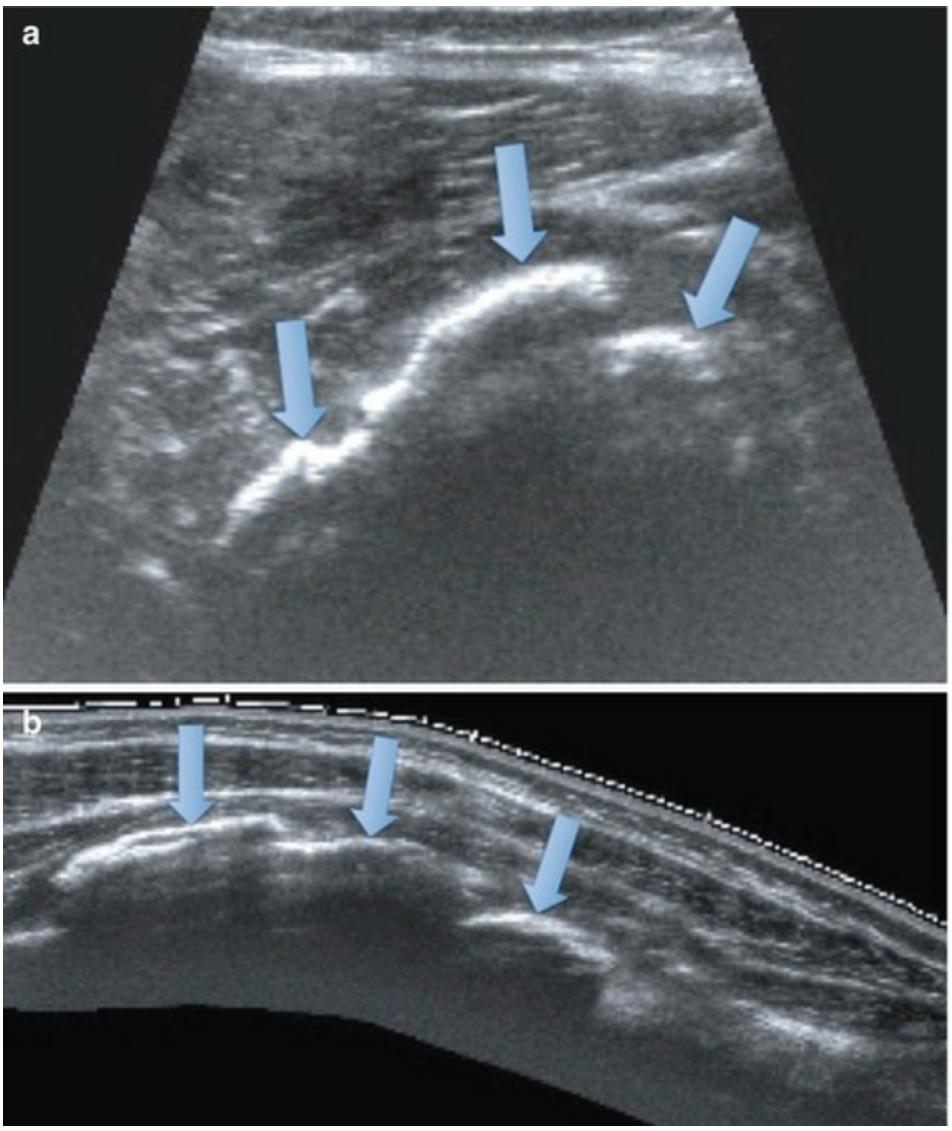

Fig. 23.3 (**a**, **b**) 15-year-old-male soccer player 4 weeks after direct contusion on the thigh Axial and sagittal ultrasound images showed extensive hyperechoic rim (*arrows*) with posterior shadowing related to ossification in vastus intermedius muscle in keeping with myositis ossificans

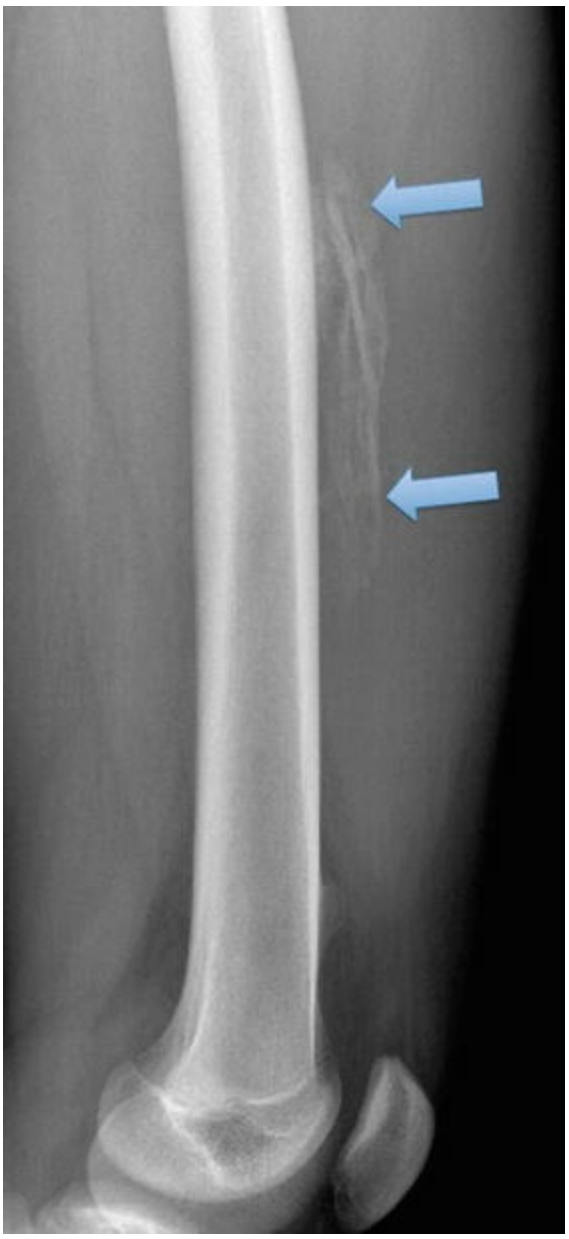

Fig. 23.4 15-year-old-male soccer player 4 weeks after direct contusion on the thigh Lateral radiograph of the left thigh shows extensive ossification (*arrows*) of the vastus intermedius

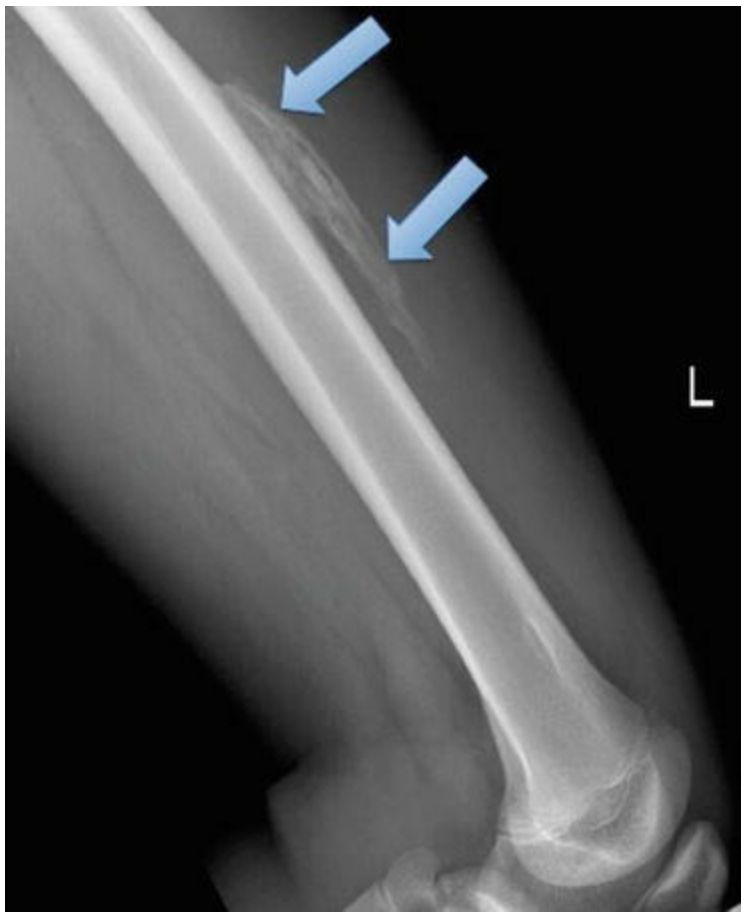

Fig. 23.5 15-year-old-male soccer player 14 weeks after direct contusion on the thigh. Lateral radiograph of the left thigh shows extensive ossification (*arrows*) of the vastus intermedius

At week 8, he underwent five weekly sessions of radial extracorporeal shockwave therapy (ESWT) of 100 impulses at 2.5–3.5 bar over every square centimeter of the mass using the Storz Medical MASTERPULS® MP100. The player felt less pain and his range of motion improved from 90 to 110° after two sessions of ESWT. He then started gradual active and passive exercises and intermittent ultrasound therapy. The swelling and pain gradually subsided and the range of motion increased as he underwent the later three sessions of ESWT. At 12 weeks, his knee could move fully and he could start jogging. He was allowed more sports specific exercises and gradually returned to normal training by the 16th week.

23.3 Discussion

In our patient, the history of direct trauma, a quadriceps contusion and knee effusion raised the suspicion of MO. Initial X -ray showed subtle changes and discrete ossification was readily diagnosed only after 4–6 weeks from the initial event. Early ultrasound scans showed soft tissue mass with no zonal demarcation or calcification.

Only the repeat ultrasound showed the increasingly reflective peripheral rim due to increasing ossification [5].

The initial appearance of MO at MRI can be confused with soft tissue malignancy like sarcomas. Acute lesions show a low or intermediate signal on T1 images that may displace fat planes and high signal on T2 with a peripheral rim of low T2 signal and much surrounding edema, often resembling sarcoma at this stage. As the lesion matures, the center usually demonstrates high signal on T1 images, in keeping with marrow, and a low-signal rim on all sequences as cortex develops around the margin [4, 6, 7].

Histopathology from fine needle aspiration cytology would require an experienced cytopathologist and proper sampling techniques [3, 4]. In our case, fine needle aspiration was not done as a combination of history of previous trauma, pain and radiological findings were adequate to arrive at the diagnosis of MO.

MO symptoms gradually subside after 6–8 weeks with rest. The swelling has been reported to be self-limiting but sometimes there can be residual hard swelling in the muscle. Usually return to competitive sports is only possible after 4–6 months [1, 2].

ESWT was suggested to be useful for symptom relief without having any significant effect on ossification in MO [8]. There are no randomized, controlled studies to document the efficacy of NSAIDs in the prevention or treatment of myositis ossificans after sports-related muscle contusion. Large lesions causing nerve impingement or severe limitation of movement could be excised surgically. More studies need to be done on prevention of myositis ossificans and possible early detection and treatment strategies which could potentially shorten the recovery time.

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24. Posterior Impingement of the Ankle: "Can There Also Be a Tendinous Entity?"

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Abstract
Posterior impingement in the ankle refers to a mechanical conflict on the back side of the ankle. This pathology is commonly seen in specific sports such as football, ballet, acro gymnastics and high jumping where hyperplantar flexion of the ankle is required. Posterior impingement can present in an acute or a chronic fashion. Posterior ankle impingement syndrome is also referred to as "os trigonum syndrome" since the posterior impingement is often associated with a prominent os trigonum (unfused posterolateral talar process). Pain results from impaction between the posterior tibial plafond and the os trigonum or posterior calcaneal process, or as a result of soft tissue compression between the two opposing osseous structures. This chapter provides an overview of the relevant terminology, functional anatomy of the posterior ankle, etiology of posterior ankle impingement, clinical and diagnostic features, historical background of this pathologic entity, and treatment strategies.

24.1 Introduction

Posterior impingement in the ankle refers to a mechanical conflict on the back side of the ankle. It occurs in specific sports such as football, ballet, acro gymnastics and high jumping where hyperplantar flexion of the ankle is a must. Posterior impingement can present in an acute or a chronic fashion, and includes about 4 % of all ankle injuries during explosive sports performance. In fact, a recent Aspetar epidemiological study –

that looked at the incidence of ankle lesions in football (soccer) in Qatar/Asia – shows that up to 14 % of all football injuries are ankle-related [1].

Injury incidence reports from FIFA show that the ankle is the third most affected joint in football during play, after the thigh and the knee [2].

Posterior ankle impingement syndrome is also referred to as “os trigonum syndrome” since the posterior impingement is often associated with a prominent os trigonum (unfused posterolateral talar process). Pain results from impaction between the posterior tibial plafond and the os trigonum or posterior calcaneal process, or as a result of soft tissue compression between the two opposing osseous structures. In addition, football slide tackles from behind that result in acute trauma to the posterolateral talar process predispose the footballer to posterior ankle impingement syndrome.

24.2 Terminology

The posterior process of the talus articulates with the tibia superiorly and contributes significantly to the subtalar articulation inferiorly at the posterior facet. It consists of a more prominent lateral (Steida process) and a medial tubercle with the flexor hallucis longus tendon running in the sulcus between them. Fractures of either tubercle or the complete posterior process, although rare, have been described and have been associated with sports injuries, including football. It is common to misdiagnose these as ankle sprains initially, while the normal accessory os trigonum just posterior to the lateral tubercle can be mistaken for a fracture. In individuals that have an accessory os trigonum, a fracture of the latter, or more commonly an injury to its synchondrosis with the lateral tubercle, can occur after injury and can lead to subsequent “os trigonum syndrome” [3, 4].

24.3 Functional Anatomy

With arthroscopy of the ankle there is significant risk of complications which can be prevented or decreased only by profound familiarity with the anatomy of the region. Adequate knowledge of the anatomy of the joint to be treated is absolutely necessary to avoid confusion and serious technical errors, and should cover not only the most common anatomic configurations (extra-articular and intra-articular) in statistical terms but also the possible anatomic variations (Fig. 24.1) [4]

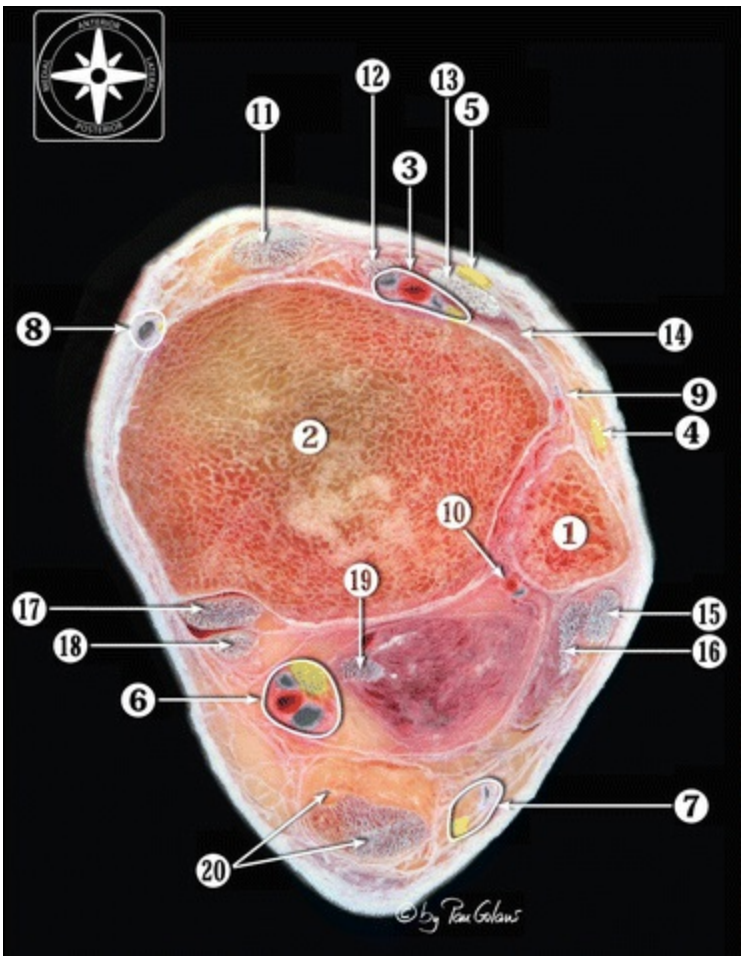

Fig. 24.1 Transverse section at the level of the tibiofibular syndesmosis showing important structures susceptible to injury during ankle arthroscopy. *1* lateral malleolus, *2* tibia, *3* anterior neurovascular bundle (deep peroneal nerve and anterior tibial artery and veins), *4* intermediate dorsal cutaneous nerve (lateral branch of the superficial peroneal nerve), *5* medial dorsal cutaneous nerve (medial branch of the superficial peroneal nerve), *6* posterior neurovascular bundle (posterior tibial nerve and posterior tibial artery and veins), *7* sural nerve and small saphenous vein, *8* saphenous nerve and great saphenous vein, *9* anterior peroneal artery, *10* posterior peroneal artery, *11* tibialis anterior tendon, *12* extensor hallucis longus tendon, *13* extensor digitorum longus tendon, *14* peroneus tertius muscle belly, *15* peroneus brevis longus, *16* peroneus brevis tendon, *17* tibialis posterior tendon, *18* flexor digitorum longus tendon, *19* flexor hallucis tendon (musculotendinous), *20* calcaneal and plantaris tendons (Drawing courtesy of Pau Golano)

The main anatomical structure for orientation and to determine the safe working area is the flexor hallucis longus tendon (FHL). Just medial to this tendon runs the posterior neurovascular bundle (tibial nerve and posterior tibial artery and veins). Posterior ankle arthroscopy should therefore routinely be performed lateral to the FHL tendon.

Proper positioning of the ankle and the hallux results in better visualization of the tendinous portion of the FHL muscle and avoids unnecessary resection of some of the muscle fibers that reach the lateral tendinous border in a semipeniform morphology. Plantar flexion of the ankle or hallux flexion facilitates visualization of the FHL tendon proximal to the lateral talar process.

The posterior ankle ligaments are also important for orientation during the posterior

ankle arthroscopy. These ligaments include the posterior talofibular ligament, the posterior intermalleolar ligament, also called the tibial slip in the arthroscopic literature, and the posterior tibiofibular ligament which is composed of a superficial and deep component or transverse ligament (Fig. 24.2).

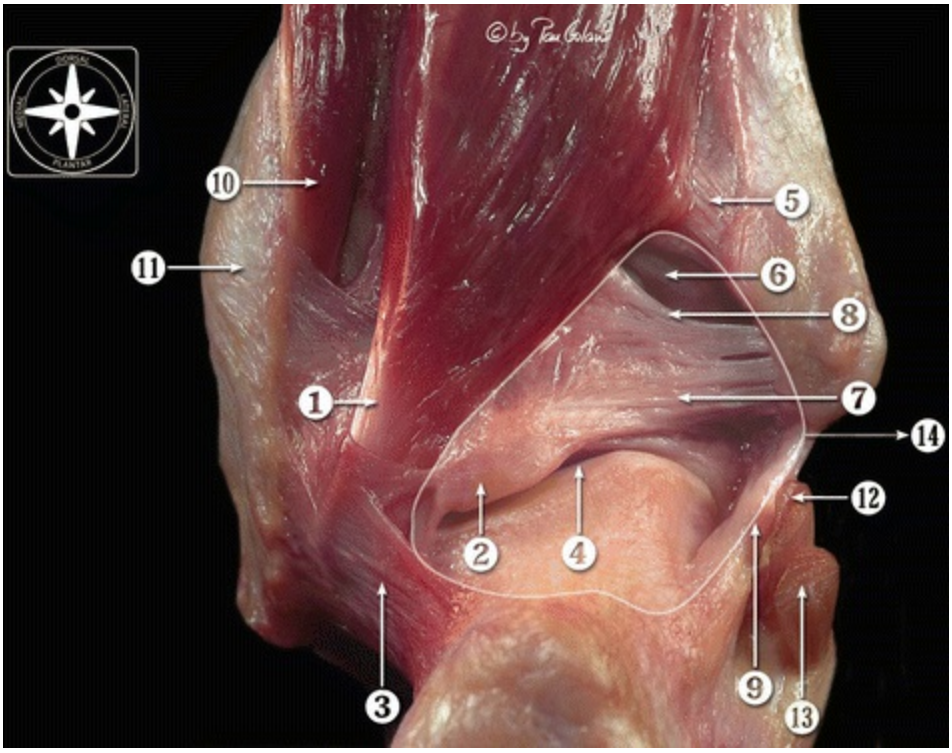

Fig. 24.2 Posterior view of the anatomical dissection of something showing the boundaries and principal anatomical details to be recognized during posterior ankle arthroscopy. The neurovascular structures were removed. *1* flexor hallucis longus tendon and muscle belly, *2* lateral talar process, *3* flexor hallucis longus retinaculum, *4* subtalar joint line, *5* superficial component of the posterior tibiofibular ligament, *6* deep component of the posterior tibiofibular ligament or transverse ligament, *7* posterior talofibular ligament, *8* posterior intermalleolar ligament or tibial slip, *9* calcaneofibular ligament, *10* flexor digitorum longus tendon and muscle belly, *11* tibialis posterior tendon covered by the flexor retinaculum, *12* peroneal brevis tendon (cut). *13* peroneal longus tendon, *14* boundaries of the safe anterior working area (Drawing courtesy of Pau Golano)

When the posterior ankle compartment is visualized arthroscopically, the location of the FHL tendon should be determined first. Then the detailed anatomy of the posterior ankle can be identified more carefully.

The posterior talofibular ligament, a component of the lateral collateral ligament, originates from the malleolar fossa, located on the medial surface of the lateral malleolus, coursing almost horizontally to insert in the posterolateral surface of the talus. This ligament is also an important reference in posterior ankle arthroscopy. Its location is important to identify the site of the subtalar and talocrural working areas (Fig. 24.3). The posterior subtalar recess is plantar to this ligament and the talocrural joint is located dorsally [5–10].

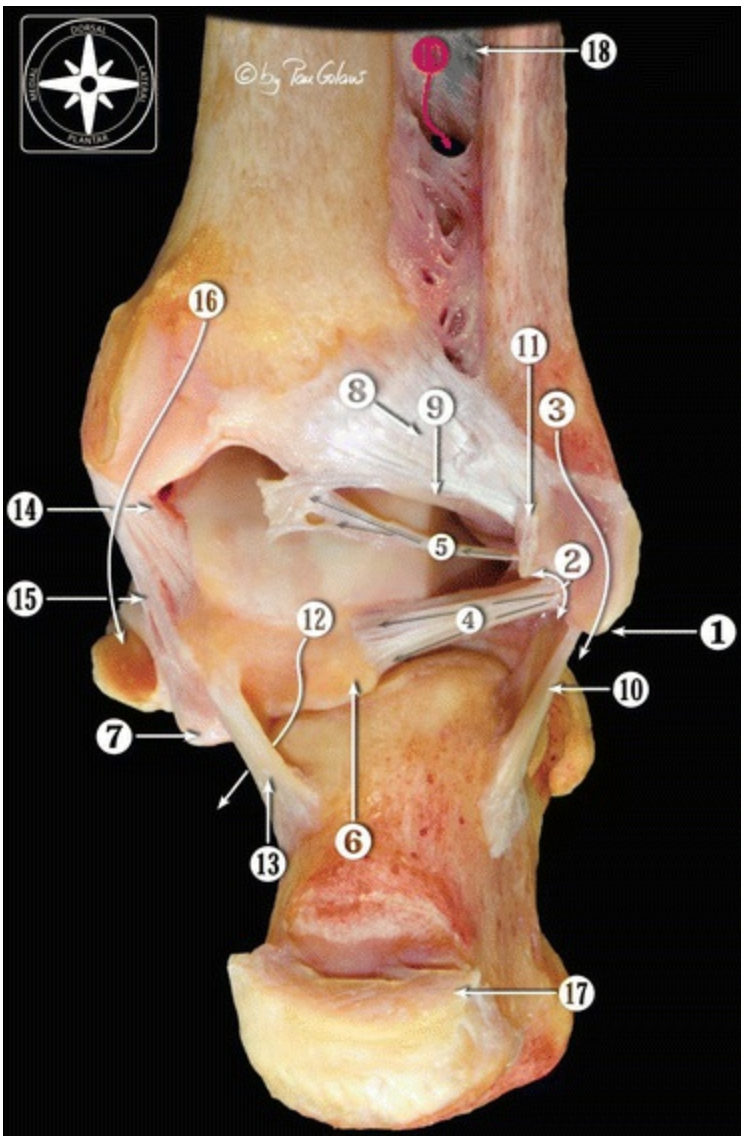

Fig. 24.3 Posterior view of the anatomical dissection of ankle ligaments (dorsal flexion). The capsule was removed. *1* lateral malleolus, *2* malleolar fossa, *3* peroneal groove of the fibula and peroneal tendons traject, *4* posterior talofibular ligament, *5* posterior intermalleolar ligament or tibial slip (capsular insertion cut), *6* lateral talar process, *7* medial talar process, *8* superficial component of the posterior tibiofibular ligament, *9* deep component of the posterior tibiofibular ligament or transverse ligament, *10* calcaneofibular ligament, *11* malleolar insertion of fibulotalocalcaneal ligament or Rouvière and Canela-Lazaro ligament (cut), *12* tunnel for flexor hallucis longus tendon, *13* flexor hallucis longus retinaculum, *14* deep posterior tibiotalar ligament of the medial collateral ligament (deep layer), *15* tibiocalcaneal ligament of the medial collateral ligament (superficial layer), *16* tibialis posterior tendon (cut) and tendon traject, *17* calcaneal or Achilles tendon (cut), *18* interosseous membrane, 19 foramen in the interosseous membrane for the anterior peroneal artery (Drawing courtesy of Pau Golano)

24.4 Etiology

Posterior ankle impingement syndrome is a clinical pain syndrome that reflects the most common cause of posterior ankle pain. It can be provoked by a forced hyperplantar flexion movement of the ankle [11–13]. In the event of a soft tissue or bony posterior

impingement of the ankle, plantar flexion induces conflict between the posterior malleolus of the distal tibia and the postero-superior calcaneal bone. A bony prominent posterior process of the ankle occurs in almost 7 % of the sports population and can present as a hypertrophic posterior talar process or as an os trigonum. Although apparent posterior bony prominences caused by acute or repetitive overload (micro-) trauma can induce posterior ankle pain, they are not necessarily associated with the posterior ankle impingement syndrome.

Soft tissue impingement in the posterior ankle region can also occur and is frequently disregarded. It is triggered mainly by hypertrophic FHL musculo-tendinous tissue, additional tendinous or doubled tendon structures and post traumatic scarification [9].

Since an acute forced hyperplantar flexion movement on the ankle or a repetitive overload induces the bony or soft-tissue conflict in the posteriorly located components of the ankle joint, these lesions are seen mainly in a sport specific population. The classical example of repetitive overload is seen in ballet dancers, where the forced plantar flexion during “en pointe” and “demi pointe” positioning induces repetitive impingement on the posteriorly located soft tissue components. Other types of sports that often lead to the posterior ankle impingement syndrome include football, swimming, cycling and any other sport in which the mechanism of injury is a repetitive forced plantar flexion or an acute setting (for example during a blocked kicking action in football). If the lesion occurs due to compression of the os trigonum between the distal tibia and calcaneal bone, it can lead to displacement of the os trigonum, disabling soft tissue inflammatory processes, or even fractures of the processus posterior tali or distal tibia (Fig. 24.4) [9].

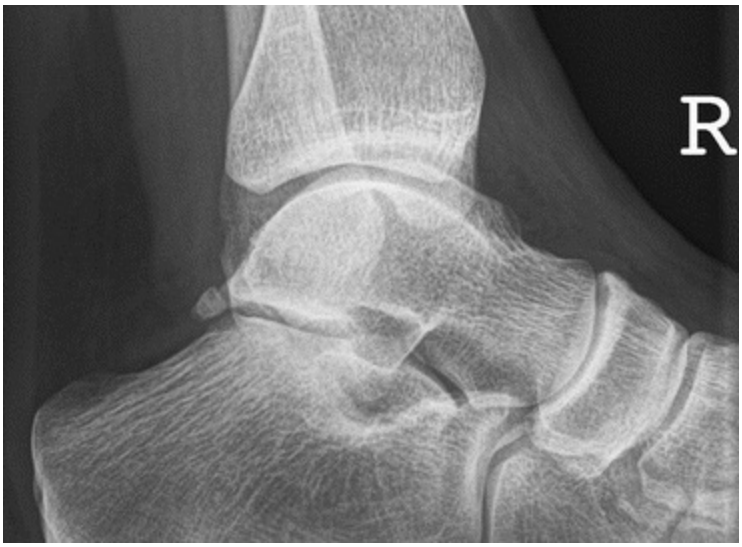

Fig. 24.4 Lateral radiograph of the right ankle shows the os trigonum, a possible bony impingement trigger in the ankle

24.5 Clinical and Diagnostic Features

Clinically, posterior impingement can be much more difficult to detect and diagnose than other types of ankle impingement because the affected structures lie much deeper, and it can be mimicked by or coexist with other disease processes such as peroneal tendinopathy, retrocalcaneal bursitis, osteochondral lesions of the posterior talar dome, Achilles tendinopathy, flexor halluces longus tendinopathy or tenosynovitis, posterior tibial osteochondral injuries, tarsal tunnel compression, tarsal coalition, and Haglund deformity. Patients will complain of chronic deep posterior ankle pain that is worsened with push-off activities such as jumping. Physical examination includes palpation over the posterolateral and posteromedial process. Patients that suffer from posterior ankle impingement present with a posteriorly localized ankle pain during a (forced) plantar flexion movement. The posterior ankle impingement test is a pathognomonic test to identify the clinical diagnosis of posterior ankle impingement. In the test, the ankle is passively and quickly forced from neutral to hyperplantar flexion position; if the patient encounters suddenly recognizable posteriorly located ankle pain the diagnosis is confirmed. To increase compression on the posterolateral structures of the ankle, plantar flexion, external rotation and eversion movements can be applied during clinical testing.

Since the neurovascular structures and tendons are localized in the posteromedial region of the ankle, this area is not always easily palpated compared to the clinical examination of the posterolateral part of the ankle [7].

Diagnosis can be confirmed with significant reduction in pain following injection of an anesthetic into the posterolateral capsule of the tibiotalar joint. MRI is useful for more accurately identifying the anatomic site of abnormality, as well as revealing coexisting pathologies. Fortunately, rest is often adequate therapy regardless of whether the symptoms are acute or chronic. When non-operative measures have failed, open or arthroscopic removal of the os can quickly return the footballer to play. Calder et al. demonstrated the effectiveness of posterior ankle arthroscopy in the treatment of posterior ankle impingement syndrome in the elite footballer, with return to training expected at an average of 5 weeks.

24.6 Fractures

24.6.1 Fractures of the Lateral Tubercle and OS Trigonum Complex

The lateral tubercle serves as an attachment to the talocalcaneal and the posterior talofibular ligament. An avulsion type (Shepherd) fracture may be the result of a plantarflexion and inversion force, while a compression type fracture is usually the result of the posterior process being squeezed between the posterior tibia and calcaneus

in extreme plantarflexion. The same mechanisms can lead to injuries to the os trigonum in individuals that have it. Athletes will usually present with pain and swelling in the posterolateral ankle. They will often ascribe the onset of symptoms to kicking the ball, and will have tenderness to deep palpation in the area between the Achilles tendon and the lateral malleolus, and exacerbation of symptoms on plantar flexion of the ankle; compression of the fracture by the FHL by dorsiflexion of the great toe can also be occasionally seen. A plain lateral radiograph provides a good view of the lateral tubercle. Care must be taken when reviewing the radiograph so as not to consider a fracture of the lateral tubercle as a normal os trigonum. The latter when present usually has a smooth cortical rim, compared to an irregular outline in the case of a fracture of the tubercle. Fractures of the os trigonum can also be found, but are rare. A fine-cut CT scan is useful to identify fractures and assess displacement, but an MRI scan is also advisable to identify possible bone bruising or injury to the synchondrosis between the os trigonum and the lateral tubercle [13].

24.6.2 Fractures of the Medial Tubercle

Fractures of the medial tubercle are rare. They can occur due to avulsion by the posterior talotibial ligament (posterior aspect of the deltoid ligament) by dorsiflexion and eversion (Cedell fractures), or by direct compression of the process as above or with impingement of the sustentaculum tali in supination. In contrast to lateral tubercle injuries, pain and swelling is usually present between the Achilles tendon and the medial malleolus, but there may be limited pain on walking or moving the ankle. It is difficult to visualize fractures of the medial tubercle on plain AP and lateral radiographs, and it has been suggested that the addition of two oblique views at 45–70° of external rotation may significantly aid detection before resorting to CT or MRI [13].

24.6.3 Fractures of the Entire Posterior Process

Fractures of the entire posterior process of the talus are rare, but are significant in that they can affect two articulations, the tibiotalar and the subtalar, with the posterior process contributing a quarter of the articulation with the posterior facet of the calcaneus. Fractures can occur due to direct trauma or compression between the posterior tibia and calcaneus with extreme plantar flexion and tend to be associated with high energy injuries. An inversion component to the mechanism has also been suggested, as well as injuries occurring with the foot in inversion and plantarflexion. Plain radiographs usually demonstrate these more obvious injuries, but fractures can often be missed unless there is a high index of suspicion. It is important to look for associated injuries, such as subtalar dislocation which has been described, and it is felt that CT imaging is essential to identify the fracture, assess its extent and displacement, exclude other injuries and identify the presence of small fragments (more than 1–2 mm)

in either articulation that may not be obvious on plain films. Early diagnosis and management is paramount for these injuries as delay in diagnosis appears to be detrimental to the outcome. Avulsion fractures of the tubercles are commonly extra-articular and can be treated non-operatively if undisplaced or minimally displaced, using a non-weight bearing below-knee cast in neutral or 15° equinus for 4–6 week, followed by mobilization and weight-bearing as tolerated. Nevertheless, in one series, two thirds of patients continued to have pain and less than 10 % responded to further immobilization and steroid injection. If the fracture site continues to be painful, proprioceptive and muscle-strengthening exercise with custom made orthotics can help with the resultant posteromedial pain, but late surgical excision (at 4–6 months) can provide good results and return to play in the majority of patients. Although fractures of the os trigonum are rare, treatment in a below-knee cast for 3 weeks has been shown to be successful with resolution of symptoms at 6 weeks; symptomatic non-unions have been reported which responded well with excision of the os trigonum. From the results above, it is felt that if the fragment is less than 2 mm and/or interferes with the articulation, operative fixation would be preferable. Significant displacement of the fracture or extension into the talar body would also benefit from surgery to avoid painful nonunion, tarsal tunnel syndrome, posterior impingement of the FHL and degenerative changes due to the intra-articular displacement or the presence of small displaced fragment (s) trapped in the joint. Fractures of the entire process, although rare, do not appear to do well with non-operative treatment with high incidence of nonunion and early degenerative changes, but in the reported series of four patients there was significant delay in diagnosis and early unprotected weight-bearing was allowed.

Open reduction and internal fixation has been recommended and fixation with K-wires or inter-fragmentary compression screws, with cannulated headless compression screws have shown satisfactory results. Both the posterolateral and posteromedial approach may be used. In the former the sural nerve is at risk, while others use the posteromedial approach, protecting the neurovascular bundle in the tibial tunnel which may provide good access to the fracture as well as the ankle and subtalar joints. Use of a medial malleolar osteotomy has been described, but it is felt that it may cause unnecessary injury to the distal tibia. Late fixation or excision can also be approached surgically in a similar manner, while arthroscopic excision is an option for those experienced with posterior ankle arthroscopy. In cases of late fixation, 6 weeks in a non-weight bearing cast is recommended, while excision and early mobilization at 7–10 days are recommended to minimize stiffness once soft tissue healing is achieved [13].

24.7 Historical Background

In the early 1930's –mainly because of its anatomic features – the ankle joint was found unsuitable for arthroscopy [5]. Forty years later, in 1970 Tagaki and later Watanabe

made considerable contributions to the arthroscopic surgery of the ankle, and the latter published a series of 28 ankle arthroscopies in 1972 [14]. Since then, numerous publications have followed.

Over the last three decades, arthroscopy of the ankle joint has become a standard and important procedure, with numerous indications for both anterior and posterior intra-articular pathology, as well for tendinous problems around the ankle.

The advantages of arthroscopy of the ankle are the direct visualization of the structures, improved assessment of the articular cartilage, faster rehabilitation and earlier resumption of activity.

Although there is less reason to perform diagnostic arthroscopy now because imaging technology has improved so much, the lack of direct access to the ankle and the nature and deep location of its hindfoot structures mean that problems in the posterior ankle still pose diagnostic and therapeutic challenges.

Historically, the hindfoot was approached by a three-portal technique: the anteromedial, anterolateral and posterolateral portals, with the patient in the supine position. It is known that the traditional posteromedial portal is associated with potential damage to the tibial nerve, the posterior tibial artery and its surrounding tendons locally. Therefore, a two-portal endoscopic technique was introduced in 2000 and since then, has shown to give excellent access to the posterior ankle compartment, the subtalar joint and the surrounding extra-articular posterior ankle structures (4).

24.8 Surgical Procedure

Hindfoot endoscopy enables the surgeon to more easily assess the posterior ankle compartment. The main indications for posterior ankle arthroscopy are treatment of an os trigonum and FHL pathology [6, 8–11]. Nowadays however, numerous ankle pathologies in athletes can be treated through this minimally invasive technique and new indications are being added.

The patient is prone with a tourniquet above the knee at the affected side, which should be carefully marked preoperatively. The affected ankle is positioned just over the edge of the operating table and is supported to allow free ankle movement (Fig. 24.5a).

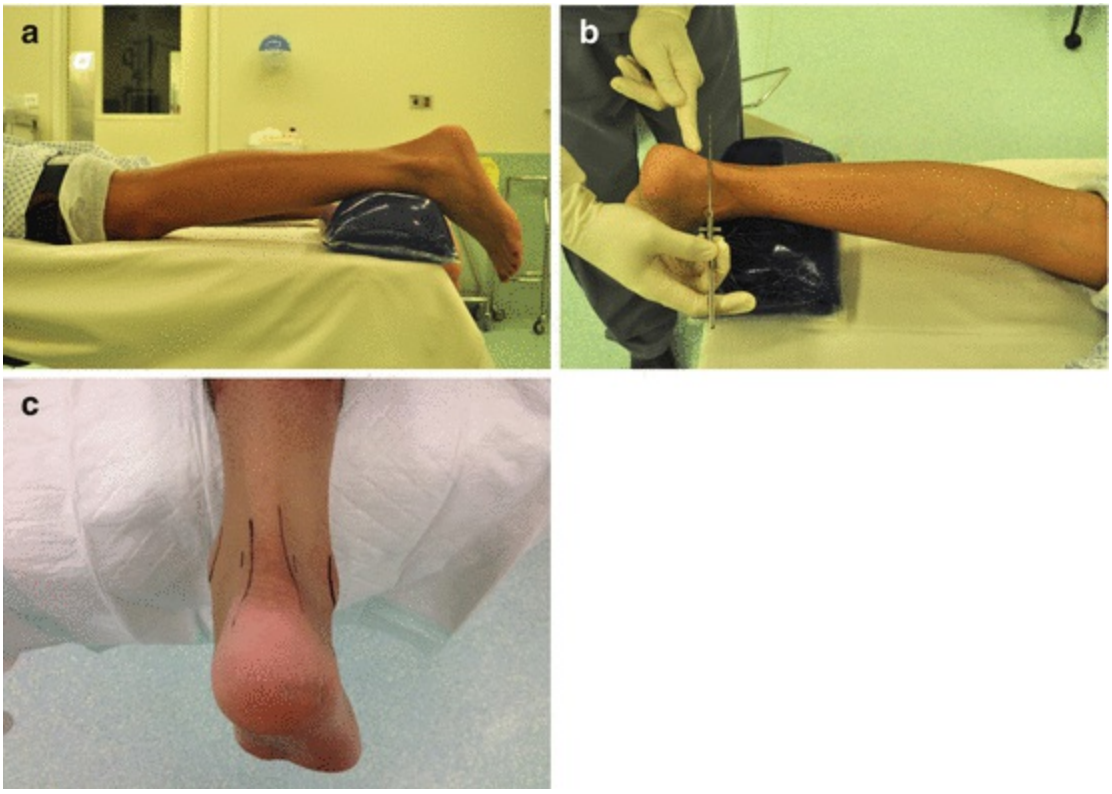

Fig. 24.5 Positioning of the patient for left ankle posterior arthroscopy: (**a**), portal preparation in left ankle posterior arthroscopy (**b**), and the preoperative setup in positioning and portal preparation for posterior ankle arthroscopy (**c**)

The anatomical landmarks for portal placement are the sole of the foot, the lateral malleolus, and the medial and lateral borders of the Achilles tendon. With the ankle in the neutral position (90°), a straight line, parallel to the sole of the foot, is drawn from the tip of the lateral malleolus to the Achilles tendon and is extended over the Achilles tendon to the medial side. The posterolateral portal is located just proximal to and 5 mm anterior to the intersection of the straight line with the lateral border of the Achilles tendon (Fig. 24.5b).

The posteromedial portal is located at the same level as the posterolateral portal, but on the medial side of the Achilles tendon (Fig. 24.5c).

Before addressing any pathology, the FHL tendon should be localized just medially to the posterior neurovascular bundle [7]. The FHL tendon determines the working area which is basically only lateral to the tendon (Fig. 24.6). Once this working area is determined the whole spectrum of posterior pathology can be treated supero-inferiorly from the tibiotalar over the subtalar joint towards the Achilles tendon insertion and mediolaterally from the tarsal tunnel release toward the peroneal tendons.

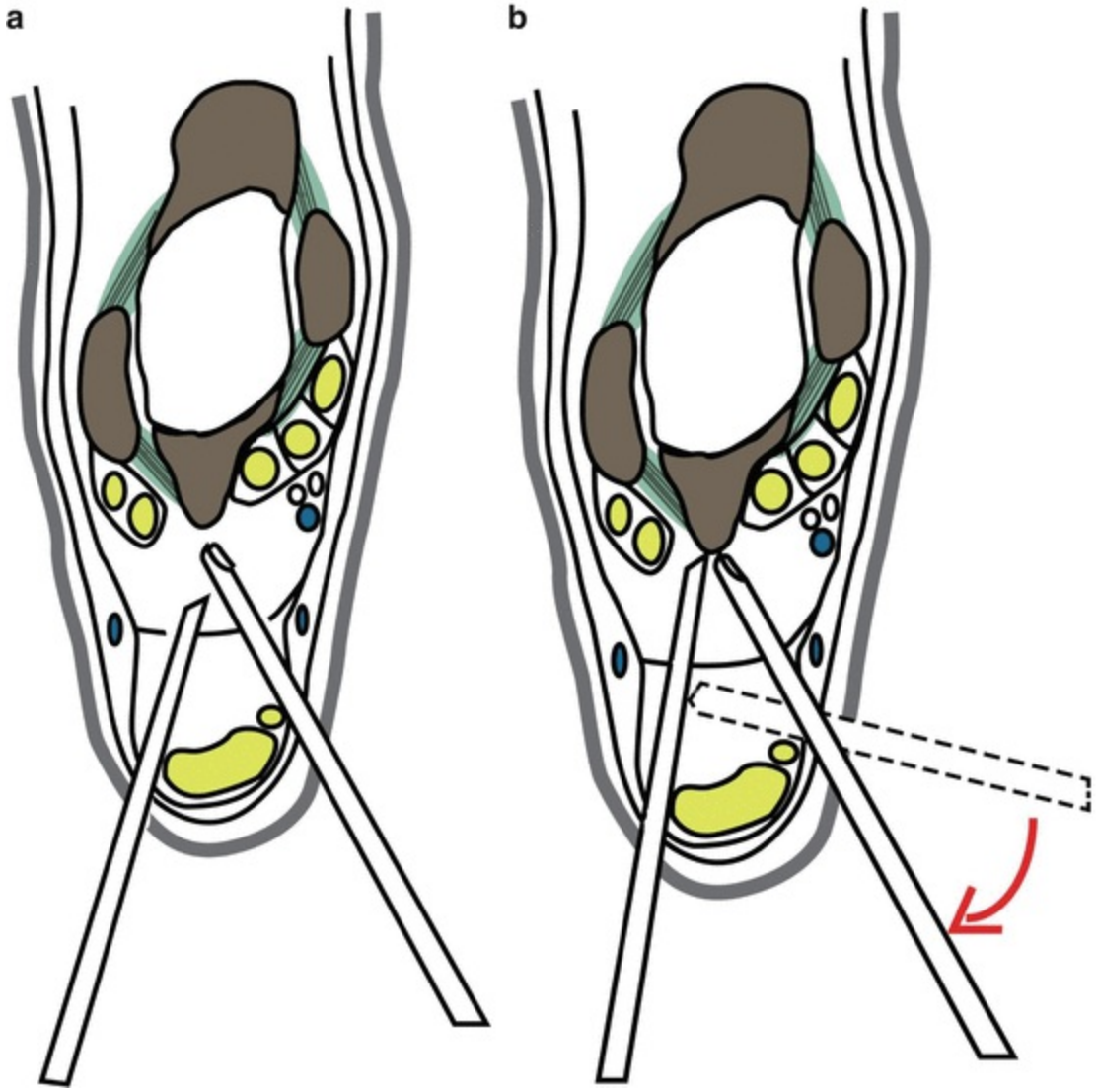

Fig. 24.6 Determining the working area for arthroscopy in posterior ankle pathology. During insertion, the arthroscope is aimed towards the first foot web space. This enables the surgeon to determine a safe working area (**a**). After insertion, the arthroscope is turned horizontally to broaden the working area after identification of the flexor hallucis longus (**b**)

Now the pathology can be addressed, ranging from debridement of soft tissue to the removal of an os trigonum or the release of the FHL tendon from its adjacent structures. Also – and more specifically –groove deepening in case of recurrent peroneal tendon dislocation, an endoscopic tarsal tunnel release, addressing a Cedell fracture or prominent posteromedial talar tubercle and treating a posteromedial talar dome lesion are now within the scope of this treatment.

Hindfoot endoscopy can be also used for the treatment of talar body fractures (Fig. 24.7), intraosseous talar cysts (that are localized posteriorly in the ankle) and as well for pigmented villonodular synovitis. This is a condition that can be localized in the posterior ankle compartment and can invade the whole posterior part of the talus, extending proximally up to the FHL tendon sheath (Fig. 24.8) [9]. Furthermore, Achilles

tendinopathy/denervation (Fig. 24.9) and Haglund syndrome pathology in the ankle can nowadays also successfully be addressed by the posterior minimal invasive 2-portal endoscopic technique in athletes. This condition requires, though, a more distally aimed two incision technique that covers the pathology all the way up to the Achilles tendon insertion (Fig. 24.10).

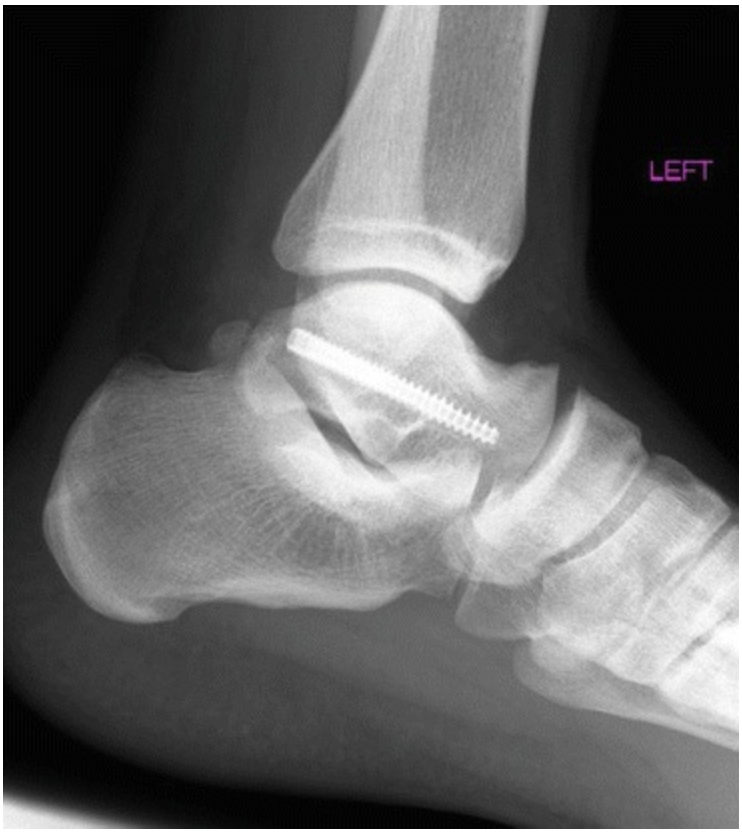

Fig. 24.7 Lateral radiograph of the ankle shows posterior arthroscopy-assisted talar body fracture treatment with a compression screw in a displaced stress fracture case

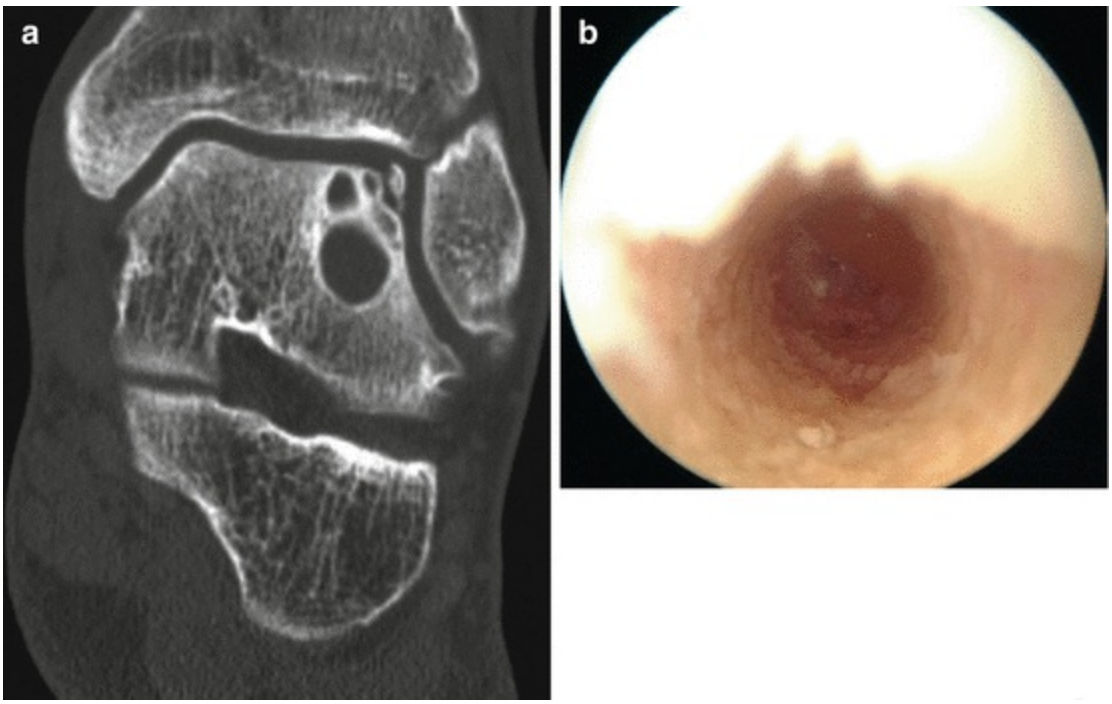

Fig. 24.8 Coronal reformatted CT image of the left ankle (**a**) shows subchondral talar cyst. Intra-osseous view of a talar cyst during ankle arthroscopy (**b**)

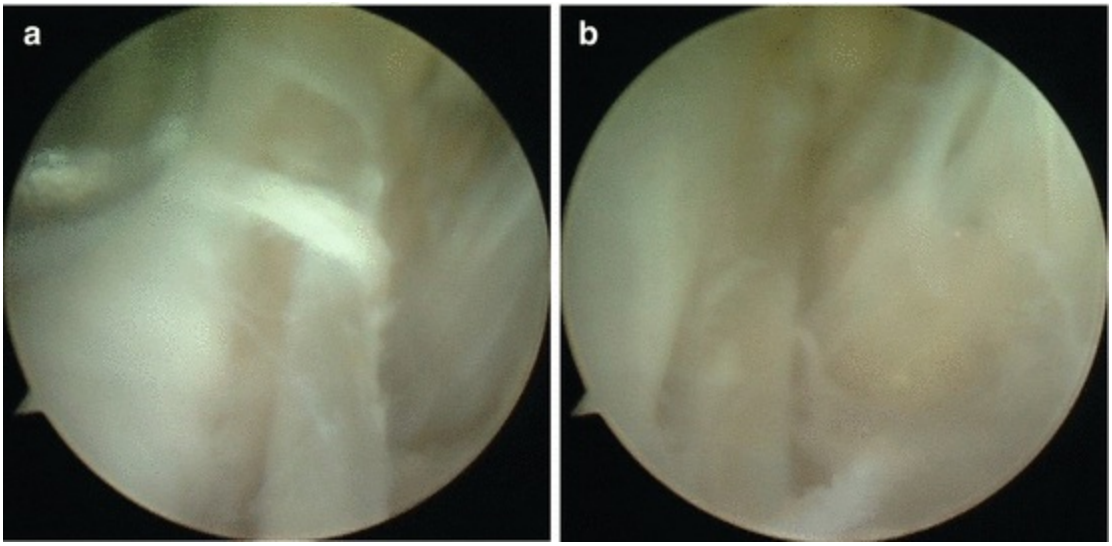

Fig. 24.9 Achilles tendon endoscopic picture with full view of the tendinopathy adhesions between the plantaris and the Achilles tendon (**a**). Achilles tendon endoscopic picture after release of the adhesions between the plantaris and the Achilles tendon (**b**)

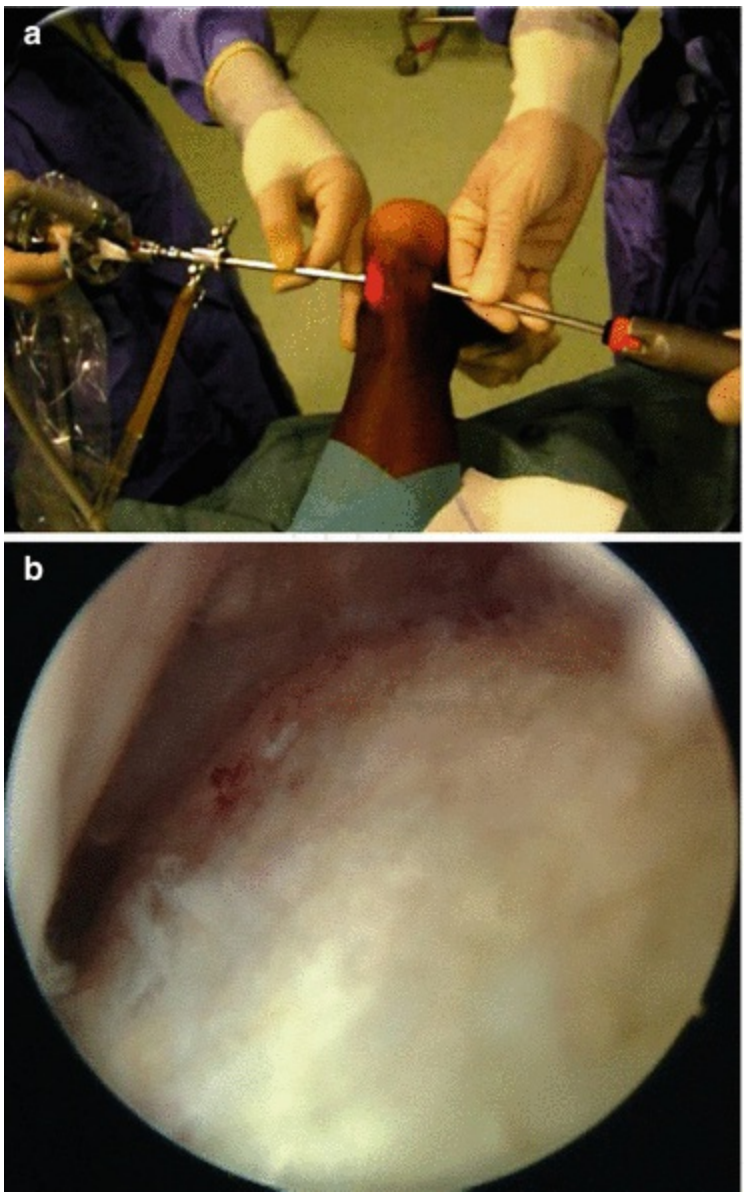

Fig. 24.10 The picture shows endoscopic Haglund's syndrome treatment with two more distally localized portals (**a**). Endoscopic view on the distal bony Achilles tendon insertion after removal of the bony prominences (**b**)

Significant advantages of this method include lower morbidity, shorter postoperative hospitalization time and quicker return to full sports. Hindfoot endoscopy is a safe and effective method for treating posterior talar cystic lesions and is an attractive alternative to open surgery for experienced arthroscopic surgeons. The most influential indication for posterior ankle arthroscopy remains the treatment of os trigonum and FHL release (Fig. 24.11). New indications continue to be found for this technique that stretch the boundaries in treating posterior ankle problems. These newest are posterior facet subtalar fusion (Fig. 24.12) and posterior tibiotalar fusion (Fig. 24.13) and they clearly show that we have not yet reached the limits of the posterior approach technique.

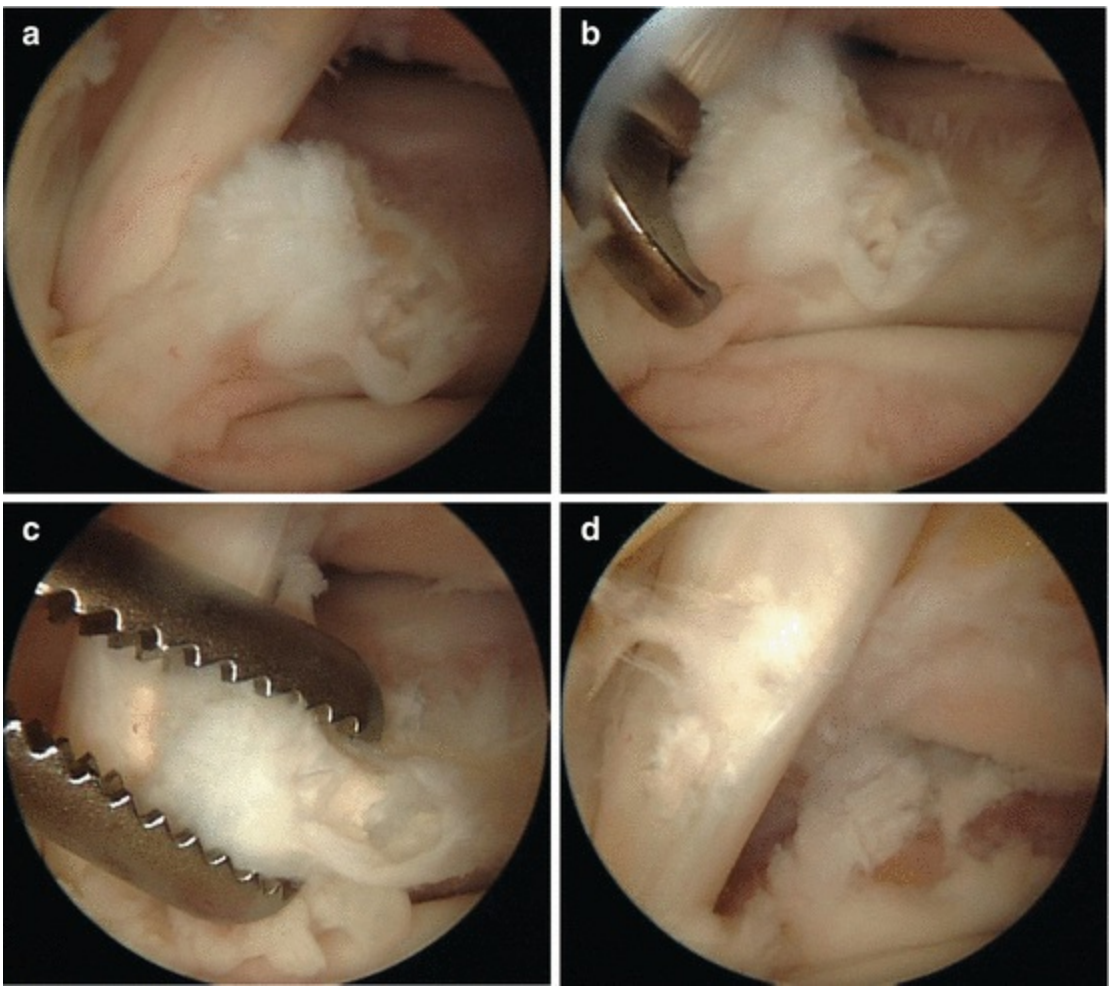

Fig. 24.11 Posterior ankle arthroscopy shows the four step decompression of the flexor hallucis longus from an os trigonum (**a**–**d**) (Note the impingement signs on the flexor hallucis longus after os trigonum decompression)

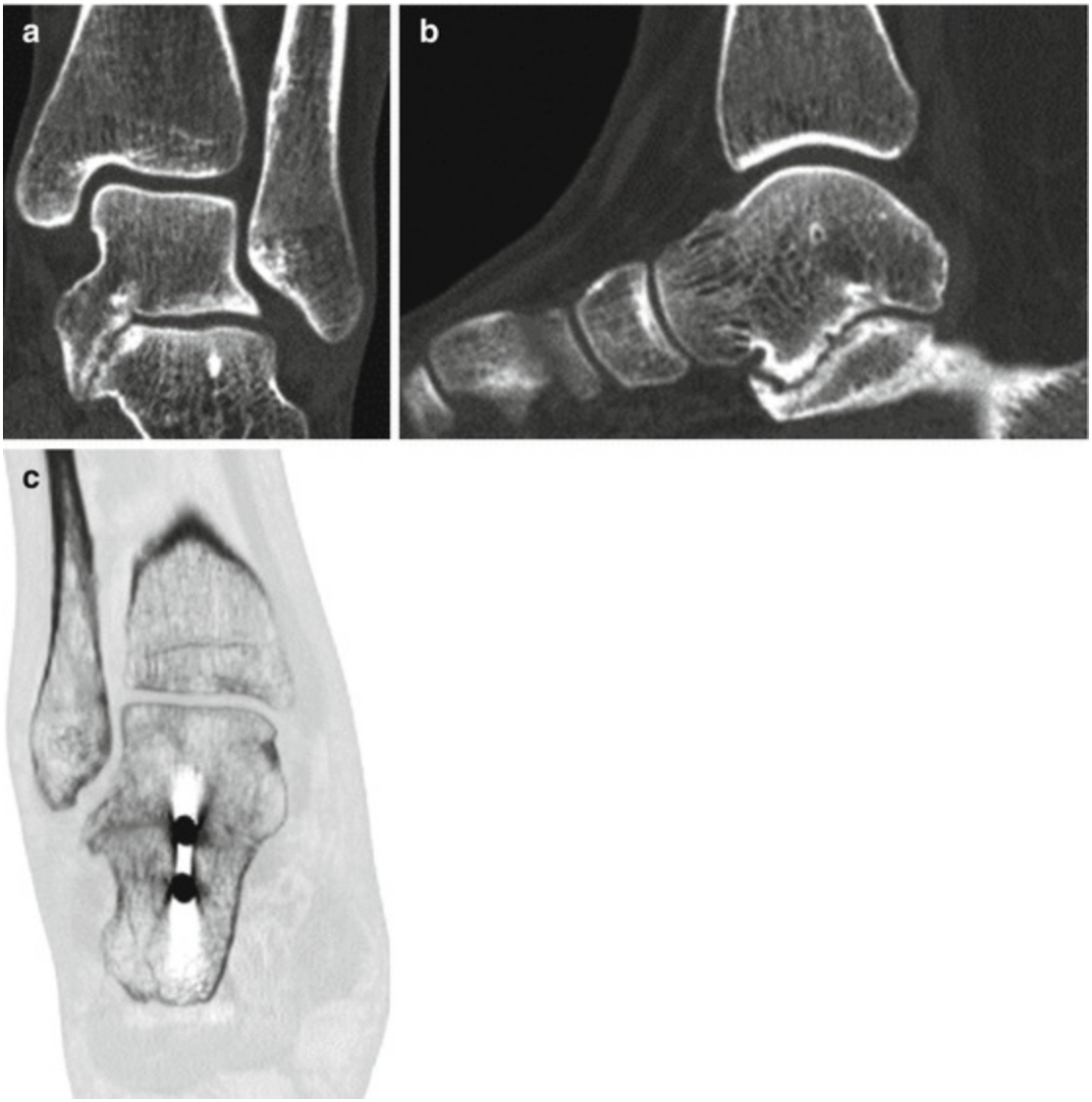

Fig. 24.12 Coronal (**a**) and sagittal (**b**) reformatted CT images of the left ankle of a patient with a medial talocalcaneal ankle joint coalition with indications for an arthroscopic subtalar posterior facet ankle joint fusion. Seven weeks after an arthroscopic subtalar fusion, coronal reformatted CT image (**c**) already shows consolidation of the fused parts

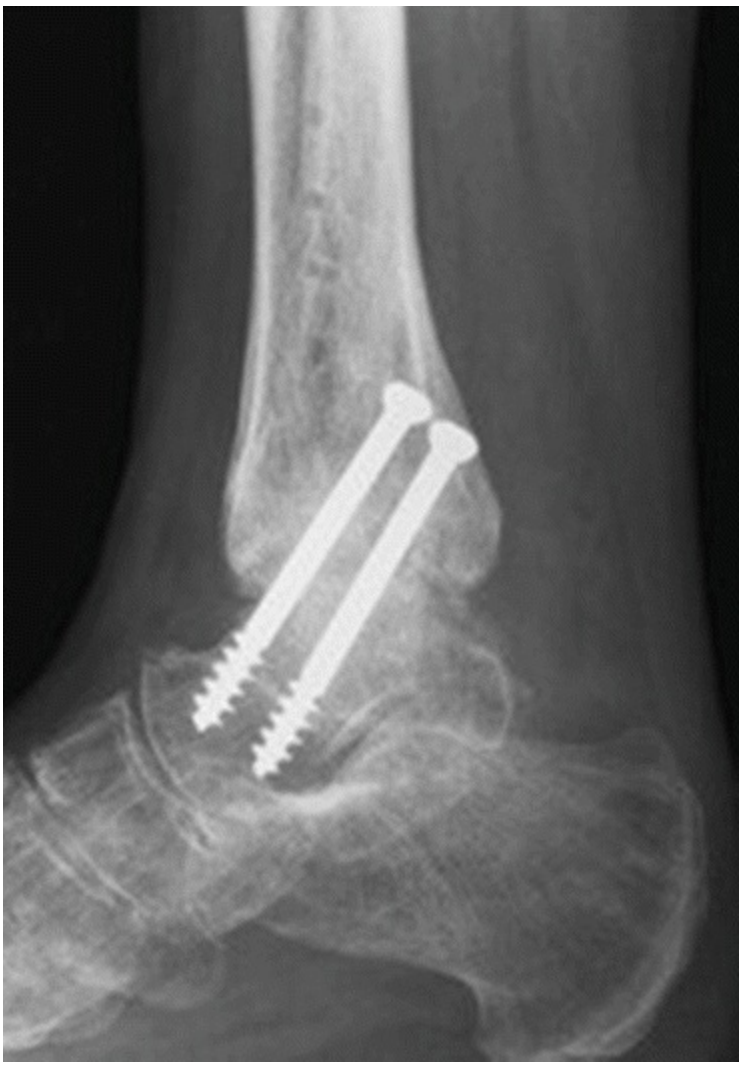

Fig. 24.13 Lateral radiograph of the ankle shows a tibiotalar fusion after a posterior arthroscopic procedure

24.9 Conclusion

Posterior ankle arthroscopy is a challenging, but safe, reliable and effective technique in the treatment of posterior ankle impingement. Due to the improved functional outcome after surgery and quicker rehabilitation time, athletes can hugely benefit from this technique. The initial indications include pathology of the flexor hallucis longus and os trigonum. Nowadays however the technique is used – with or without an additional portal – for an increasing amount of posterior ankle pathologies. Studies are now underway to assess the value of the new indications and they have already shown that the arthroscopic posterior technique in posterior ankle pathology has not yet reached its limits. Arthroscopy is frequently but erroneously believed to address only bony problems in the ankle. Many soft tissue problems (especially FHL) arise in clinical posterior impingement problems over the ankle. They are a common trigger to this pathology and should always be considered and addressed.

24.9.1 How to Diagnose Posterior Impingement of the Ankle

- Ask the patient about sport-specific repetitive ankle movements
- Perform a hyperplantar flexion movement of the ankle
- Look for palpatory pain along the course of the flexor hallucis longus

24.9.2 How to Treat Posterior Impingement of the Ankle

- Perform a diagnostic injection
- Start with the standardized 2-portal technique after initial cadaveric training
- Search for the flexor hallucis longus tendon and the posterior talofibular ligament as primary anatomical landmarks

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25. Ischiofemoral Impingement

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Abstract and Background
Ischiofemoral impingement is a rare cause of hip pain, yet it should be considered whenever a patient complains of a snapping hip. We report a case of a soccer player who described progressive onset of a hip pain without trauma. Results of the physical examination and tests of the range of motion of the hip were normal. There were no findings on X-rays and ultrasound. MRI, however, revealed an area of high signal intensity on T2-weighted images which lead to the diagnosis of ischiofemoral impingement. Medical treatment including physiotherapy, stretching and corticosteroid injection under sonographic guidance provided relief from pain and allowed the player to return to the game.

25.1 Case Description

A 22-year-old professional soccer player (goalkeeper) was referred for management of hip pain which began several weeks before. The pain increased slightly and lead to a halt in activity. There was no snapping. On physical examination, posterior pain during hip range of motion was encountered.

Plain radiographs and ultrasound did not reveal any abnormality. MRI showed an area within the quadratus femoris muscle of high-signal intensity on T2-weighted fat-suppressed images but not on T1-weighted images. Gadolinium intravenous injection

revealed peripheral enhancement.

Treatment included physiotherapy, hip abductor stretching and corticosteroid injection under ultrasound guidance. Return to previous activities occurred 4 weeks after the initial visit (Fig. 25.1).

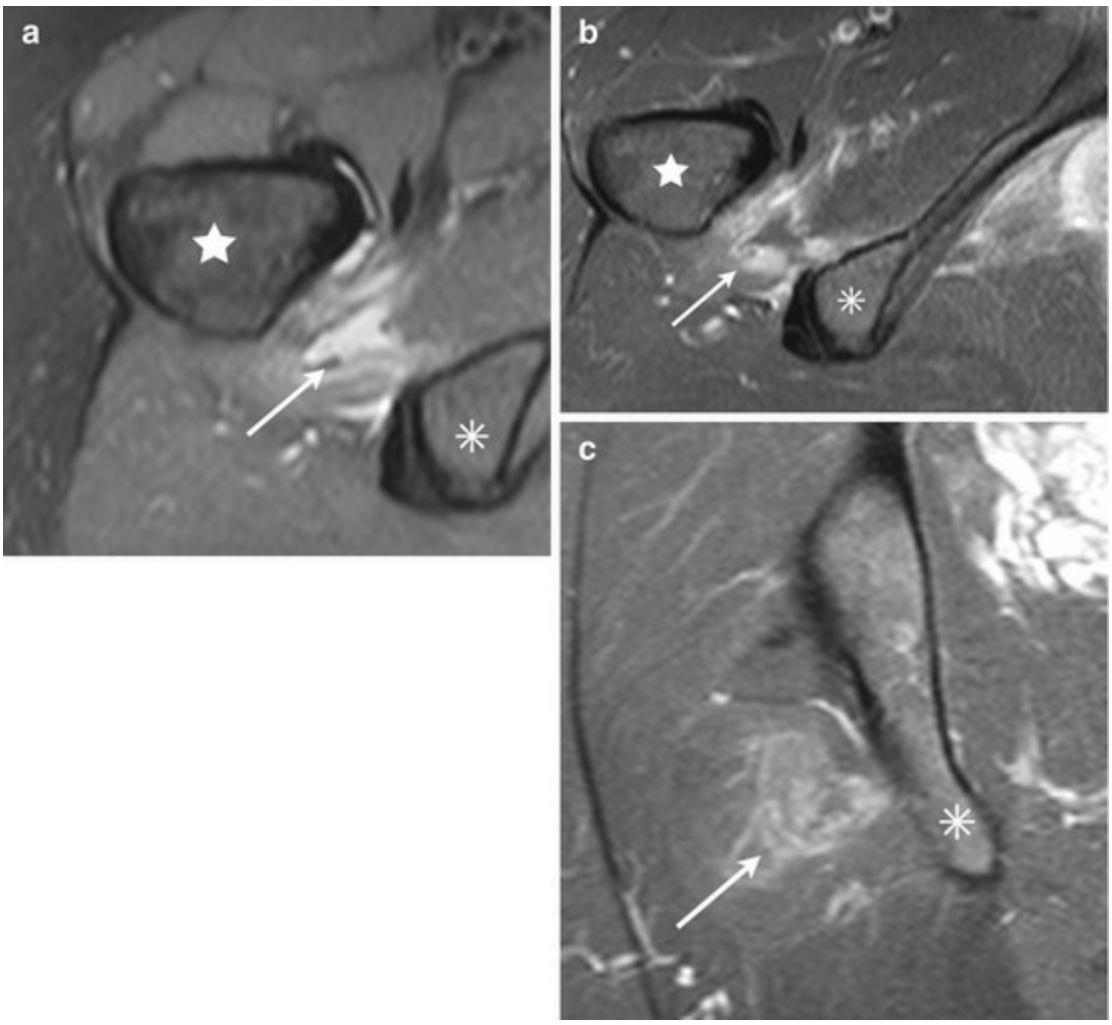

Fig. 25.1 Axial fat-suppressed T2- (**a**), axial contrast-enhanced fat-suppressed T1- (**b**) and coronal fat-suppression T2-weighted (**c**) MRI show an area of high-signal intensity within the quadratus femoris (*white arrow*) and peripheral enhancement after gadolinium injection. Lesser trochanter–*star*; ischial tuberosity–*asterisk*

25.2 Discussion and Evaluation

Ischiofemoral impingement is mainly congenital although it was first described in patients with total hip arthroplasty or femoral osteotomy by Johnson in 1977 [1]. Other studies have shown that the impingement occurs in athletes and non-athletes and is more common in women than in men. It occurs mostly in the fifth decade and is bilateral in one third of patients [2–4].

Ischiofemoral narrowing is the main cause of impingement and may be result from

trauma or tumoral disorders. Nevertheless, a case of a 17-year-old girl with post-traumatic ischiofemoral narrowing without any fracture but with an audible snapping was reported by Ali et al.; she required resection of the lesser trochanter [5]. A traumatic cause may not be uncovered; it is important to remember that the onset of symptoms is usually progressive and has a positional cause [6, 7] (Fig. 25.2).

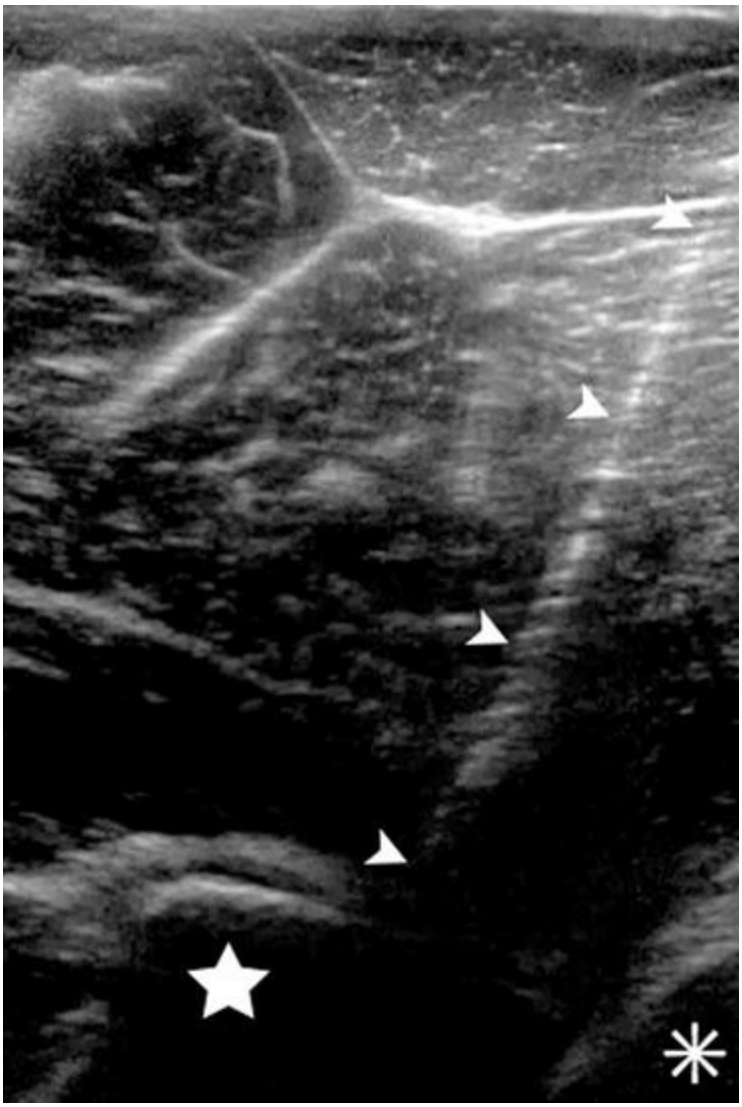

Fig. 25.2 Ultrasound guidance allows monitoring of the position of the needle (*arrowheads*) and corticosteroid injection within the quadratus femoris. Lesser trochanter–*star*; ischial tuberosity–*asterisk*

We report a case of ischiofemoral impingement in its classical presentation. Ischiofemoral narrowing leads to compression of the quadratus femoris muscle and progressive onset of hip pain. Snapping is a less common finding and was absent in this case. Clinical examination is often normal although pain may be reproduced by a combination of extension, adduction and external rotation of the hip [1, 4].

X-rays are often normal or may show secondary causes such as post-traumatic deformities or tumors involving the lesser trochanter or the ischium [8]. Sclerosis or

cystic changes of the lesser trochanter have also been described [9]. Ischiofemoral narrowing cannot be assessed on radiographs.

CT or MRI are more reliable to assess the distance between the lesser trochanter and the ischial tuberosity. Torriani et al. showed that the mean distance was 13 +/- 5 mm in patients with ischiofemoral impingement and 23 +/- 8 mm in a control group [10]. Care should be taken when assessing this measurement because it depends on the degree of the internal or external rotation of the hip. A study on MRI assessment of ischiofemoral impingement by Tosun et al. showed that the ischiofemoral space (distance between the lateral cortex of the ischial tuberosity and medial cortex of the lesser trochanter), quadratus femoris space and muscle volume values of the patient group were significantly lower than those of controls. Measurements of the hamstring tendon area and inclination angle (angle between the long axis of the femoral neck and the long axis of the femoral shaft on coronal T1-weighted images) in the patient group were also significantly higher than in controls. The quadratus femoris muscle fatty replacement grades were significantly higher in the patient group than in the control group [2].

MRI is the most useful imaging modality to diagnose ischiofemoral impingement because it allows assessment of the quadratus femoris within the ischiofemoral space. This muscle emerges from the lateral edge of the ischial tuberosity, proximal to the hamstring tendons and inserts on the intertrochanteric crest of the proximal femur. It is innervated by a small branch of the sacral plexus [11]. Its proximity with this latter may explain why ischiofemoral impingement can cause pain with distal radiation.

The quadratus femoris acts as an external rotator of the hip. Degenerative changes occur in the muscle but are not associated with the size of the ischiofemoral space [12].

With ischiofemoral impingement, the most common finding is diffuse edema within the muscle which is best seen on T2-weighted images. Others signs include bursa-like high signal intensity, fatty replacement or muscle atrophy [13].

The main differential diagnosis includes muscle strain which occurs mainly at the myotendinous junction and which usually has a traumatic origin [11]. Osteoarthritis or osteonecrosis of the hip and femoroacetabular impingement can also be considered. Lumbosacral radiculopathy or piriformis syndrome may also cause hip pain with radiating lower-limb pain.

Treatment usually includes rest, nonsteroidal anti-inflammatory drugs and corticosteroid injections under sonographic guidance. The goal of an exercise program is to stretch hip muscles to increase the range of motion in the hip joint. Lack of pain relief may lead to resection of the lesser trochanter [5].

25.3 Conclusion

Ischiofemoral impingement should be considered in patients who have chronic hip pain

radiating distally and a deep snapping sensation. X-rays may exclude classical hip disorders such as osteoarthritis, osteonecrosis or femoroacetabular impingement. MRI is the modality of choice: it shows edema within the quadratus femoris and narrowing of the ischiofemoral space. Treatment is usually conservative but may require surgical resection of the lesser trochanter

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26. Occult Muscular Vein Thrombosis as a Consequence of Prior Muscle Strain

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Abstract
Muscle strains are common and are usually assessed by ultrasound or MRI. Complications of strains include re-injury, chronic pain due to fibrous or calcific scar, muscle herniation and thrombophlebitis. We present a case of an athlete presenting thrombosis within the soleus and the medial head of the gastrocnemius veins following previous history of a strain involving the medial head of the medial gastrocnemius.

26.1 Clinical History

A 30-year-old man had an indirect trauma on the left calf when playing soccer and initial ultrasound examination revealed a grade 3 strain of the medial head of the gastrocnemius, also called "tennis-leg". Three weeks after initial trauma, calf pain increased which led to an MRI. This latter showed a mild high-signal area at the deep aspect of the medial head of the gastrocnemius. It also revealed abnormalities within the veins of the soleus which were enlarged and have hypointense central signal and peripheric hyperintense signal (Figure 26.1). Ultrasound confirmed the diagnosis of muscular vein thrombosis showing echoic thrombus within the veins of the soleus which were not compressible and absence of signal at color Doppler (Figure 26.2). Pain relief was obtained 1 week after the diagnosis of muscular phlebitis and return to previous activities was possible 2 months later.

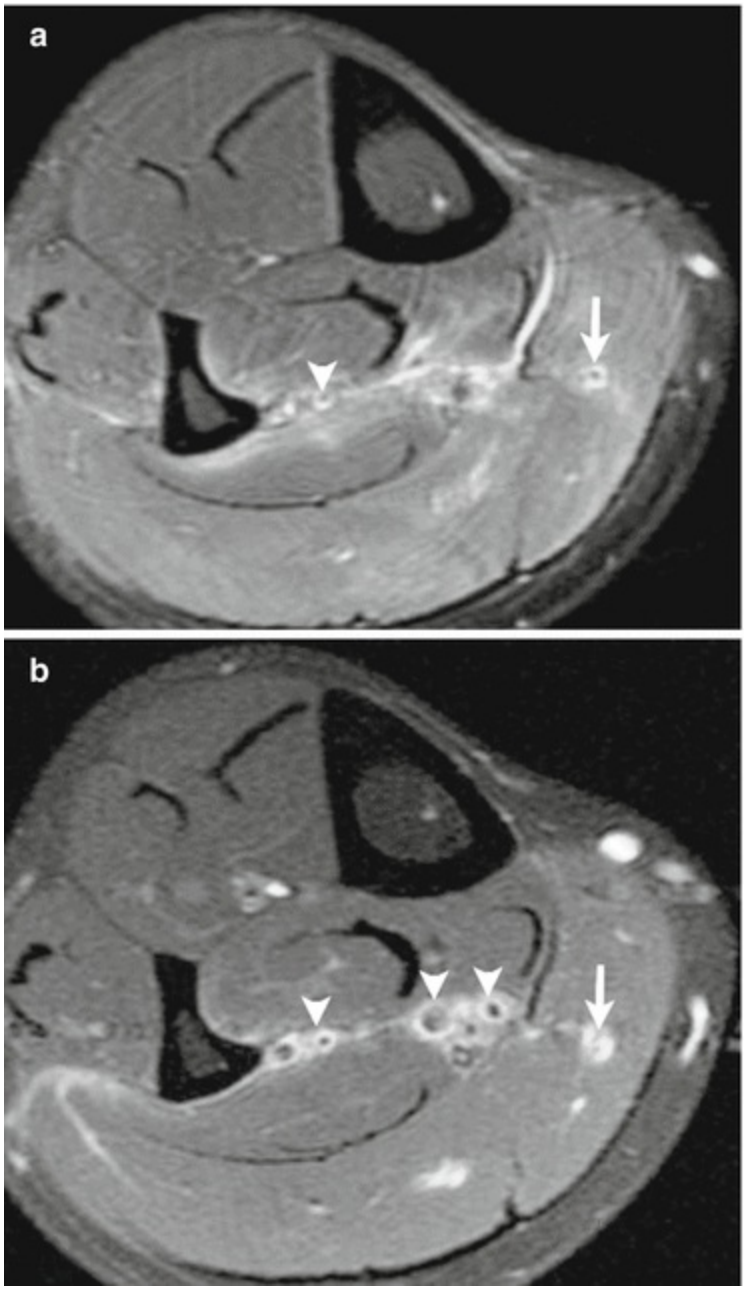

Fig. 26.1 (**a**, **b**) Axial fat-suppressed T2-weighted MRI show thrombi within the veins of soleus (*arrowheads*) and medial head of gastrocnemius muscles (*arrow*)

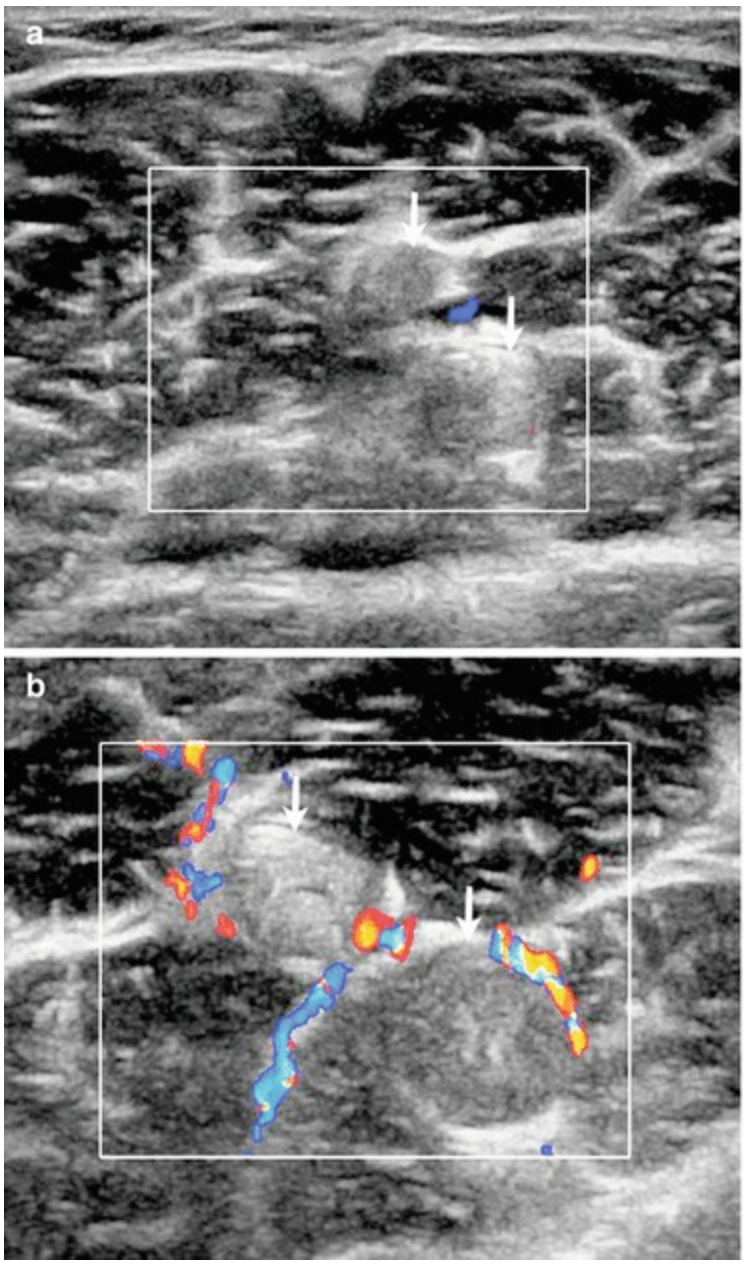

Fig. 26.2 (**a**, **b**) Transverse Doppler ultrasound show echoic thrombi (*arrows*) within the veins of the medial head of the gastrocnemius muscle

26.2 Discussion

It is reported that calf vein thrombosis affects 10 % of patients with a previous tennis leg lesion [1].

The thrombosis is mainly due to lack of calf muscular contracture and rest during healing but the possibility of direct trauma to the veins should be kept in mind [2, 3].

Intramuscular veins of each head of the gastrocnemius consist of two veins running close to a central artery and within a central septa. Veins appear anechoic in the transverse plane and are normally compressible with the probe.

The diagnosis is usually simple but it is necessary to allow accurate treatment and prevent complications such as post-thrombotic syndrome or pulmonary embolism. Muscular vein thrombosis may be suspected on B-mode when muscular veins are filled by a non-compressible echoic material [4, 5].

At color Doppler, there is no spontaneous flow within the veins.

Indirect signs include lack of vein compressibility in the transverse plane and increased diameter just proximal to the thrombus. It appears echoic at the acute phase and tend to become hypoechoic at the chronic phase. It is also soft initially and becomes slightly harder later [6, 7].

The height of the thrombosis is usually between 5 and 10 cm. It is important to locate the proximal border of the thrombosis: a thrombosis within the gastrocnemius veins may extend through the popliteus vein whereas a thrombosis within the soleus vein can reach the fibular and/or the tibial veins.

26.3 Conclusion

Ultrasound is the imaging modality of choice to allow the diagnosis and the follow-up of "tennis-leg" and in case of clinical suspicion of deep vein thrombosis which may occur during the healing phase.

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27. Pectoralis Major Injury

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27.1 Introduction

Ruptures of the pectoralis major occur predominantly in men aged between 20 and 40 years, and have become more common in the last decade with increasing popularity weight lifting, martial arts and gymnastics. In some series, anabolic steroids use is reported in up to 95 % of the patients [1].

The tendon usually tears at the humeral insertion or in the musculotendinous junction during eccentric contraction with the arm in external rotation and extension, as in bench press exercise. Complete ruptures are more common than partial tears and are often followed by intense pain that interrupts physical activity, followed by ecchymosis and local edema. Sometimes, an audible pop can be heard at the time of the rupture [2]. Partial tears or strains can be reported as pain or weakness at the time of the exercise and remain undiagnosed.

Magnetic resonance imaging is the imaging method of choice for pectoralis major tears, for adequate surgical planning or for diagnostic confirmation in the chronic phase, although ultrasound can be helpful in the emergency room. Accurate and early diagnosis of a pectoralis major tear is critical as surgical repair in the acute period improves the diagnosis.

27.2 Muscle Anatomy

The pectoralis major is a large, fan-shaped muscle that covers the upper chest wall anteriorly. The muscle is generally divided into two portions, the clavicular portion that

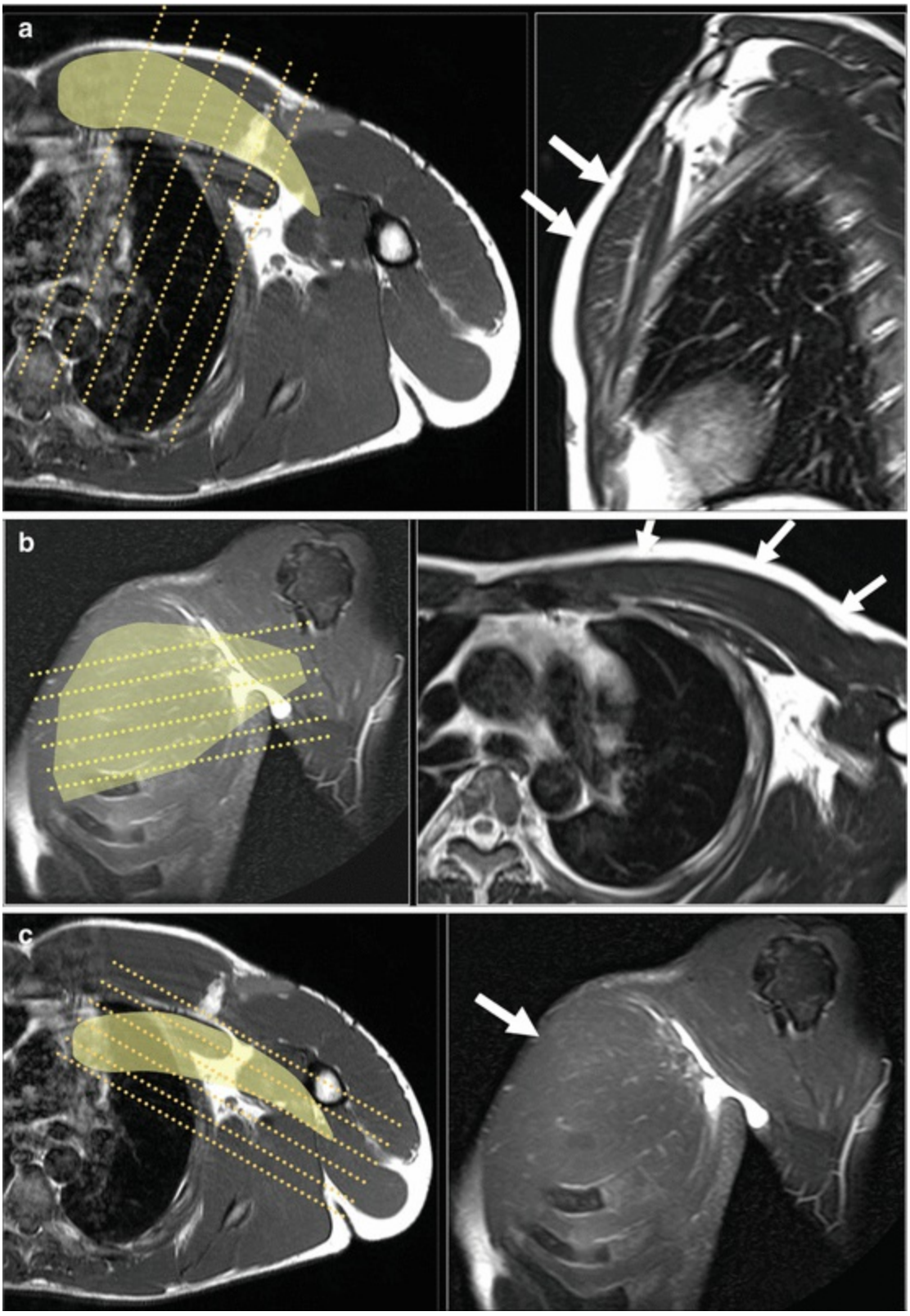
originates on the medial one half to two thirds of the clavicle, and the segmented sternocostal portion that originates at the upper two thirds of the sternum and extends to the fifth and sixth ribs and the external oblique fascia. Some authors describe the lowest portion of the muscle as a distinct abdominal laminae.

These fibers fuse to form a tendon with a bilaminar configuration with continuity between the two laminae distally at the humeral insertion. The anterior tendon is composed of fibers from the clavicular head and superior fibers of the sternocostal head, and the posterior tendon is composed of the sternocostal and inferior segments; they insert directly at the lateral edge of the bicipital groove, without twisting of the anterior tendon fibers before insertion.

The humeral footprint [3] is located in the lateral edge of the bicipital groove and measures 4.8–7.7 cm in the cephalocaudal dimension and 1.4–5.6 mm in thickness. The tendon inserts 4 cm distal to the greater tuberosity of the humerus, between the tendons of the latissimus dorsi laterally and teres major medially (a mnemonic for this pattern of insertion is “lady between two majors”).

27.3 MRI

MRI is the diagnostic study of choice when pectoralis major tears are suspected. A high field magnet (1.5–3.0 T) is indicated; a surface coil should cover the axilla and the proximal part of the arm with a large field-of-view (18–22 cm) using the humeral head and the deltoid tuberosity as anatomical landmarks. In our institution we use a standard protocol with T1-weighted images and T2-weighted images with fat suppression in the three orthogonal planes (axial, sagittal and coronal), 4–5 mm thickness and a 512 × 256 matrix (Fig. 27.1).


a
b
c

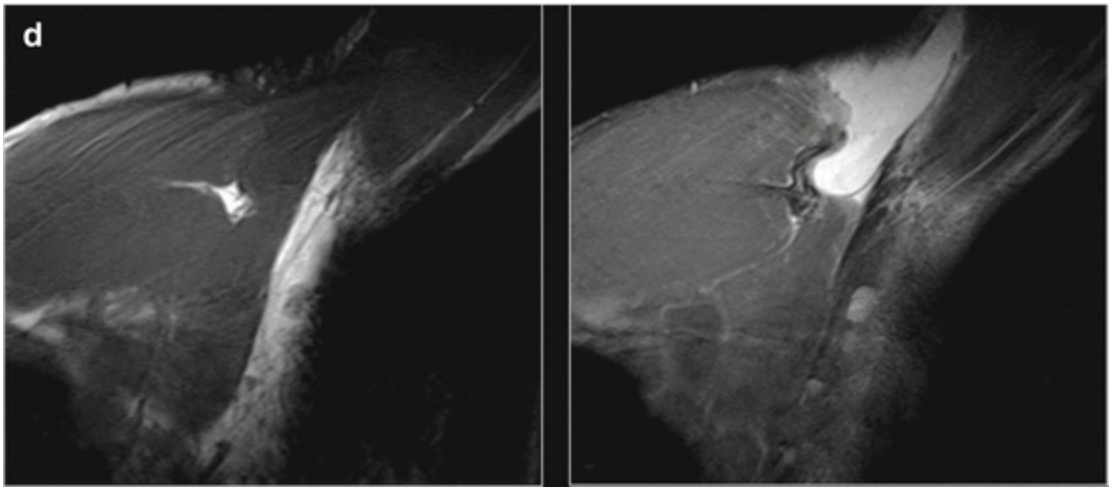


Fig. 27.1 These figures show how MRI is acquired for the pectoralis major muscle in sagittal (**a**), axial (**b**), coronal (**c**), and abduction and external rotation (ABER) (**d**) planes

An abduction and external rotation (ABER) sequence can be added to the protocol for estimating the tendinous gap in the proper anatomic axis of the tendon (Fig. 27.1d).

In 2000, Lee et al. published a cadaveric study, and described important anatomical landmarks of the pectoralis major insertion on MRI [4].

– The superior margin of the pectoralis insertion is the quadrilateral space. Another reliable landmark of the superior aspect of tendon insertion is the origin of the lateral head of the triceps muscle, identifying the tendon approximately 5–10 mm superior to the level at which the lateral head of the triceps is first identified (Fig. 27.2).

– The inferior boundary of tendon insertion is the superior aspect of the deltoid tuberosity (Fig. 27.3).

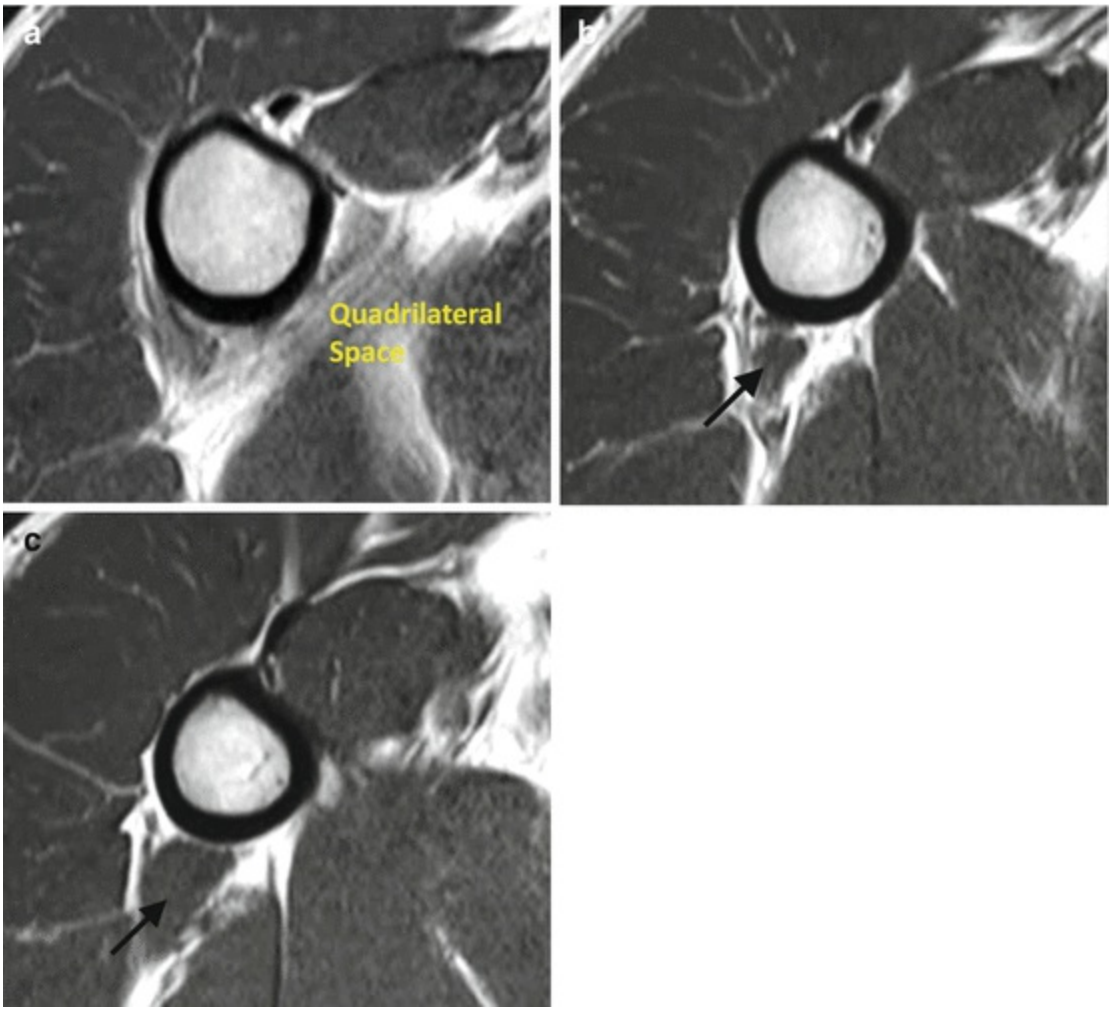


Fig. 27.2 Insertion of the pectoralis major tendon and superior anatomic landmarks. Axial T1-weighted MRI show the quadrilateral space (**a**), the origin of the lateral head of the triceps (*arrow*, **b**, **c**) and the superior aspect of the tendon insertion (**c**).

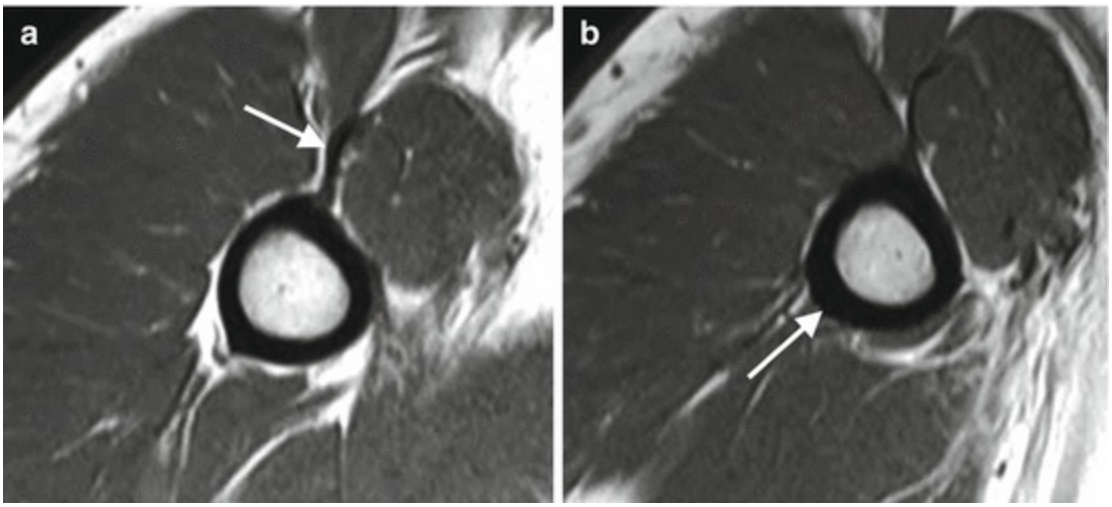


Fig. 27.3 Insertion of pectoralis major tendon and inferior anatomic landmarks. Axial T1-weighted MRI show the normal aspect of pectoralis tendon insertion (*arrow*, **a**) at the lateral edge of the intertubercular groove, and also show

the deltoid tuberosity (*arrow*, **b**) an anatomical landmark for the inferior aspect of insertion

27.4 Pectoralis Major Tears

Pectoralis major tears can be described by anatomic location as muscle origin (clavicular and sternocostal head), muscle belly, musculotendinous junction, intratendinous, humeral insertion, and bony avulsion [5–7].

27.5 Muscle Origin and Muscle Belly Tears or Sprains

Less common than other sites and usually due to direct trauma, as in an automobile accident or a blow against the chest. In general they are incomplete tears, with the muscular damage restricted to the area of direct contact and are treated conservatively.

27.5.1 Musculotendinous Junction, Intra-tendinous, Humeral Insertion and Bony Avulsion

Pectoralis tendon tears occur primarily at the tendon insertion and musculotendinous junction. Bony avulsions can occur in 2–5 % cases.

In MRI complete tears are characterized by tendon disruption and muscle fiber edema with subsequent proximal retraction, usually superficial to the biceps brachii tendon short head and coracobrachialis. A fluid collection usually is interposed between the torn tendon and the humerus (Figs. 27.4 and 27.5).

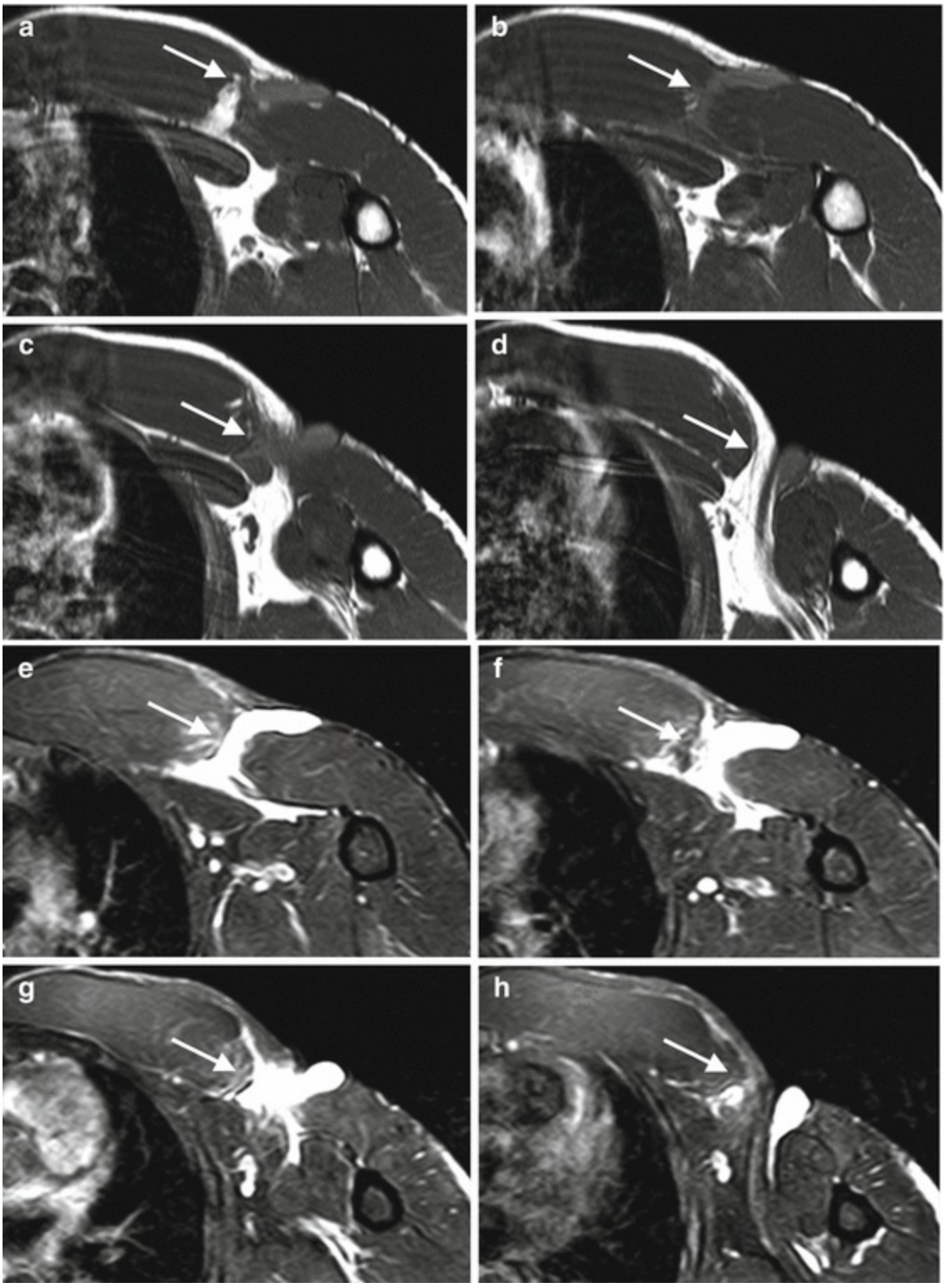


Fig. 27.4 A 32-year-old man with a complete myotendinous junction tear after bench press exercise. Axial T1-weighted MRI show a complete distal pectoralis major tear (*arrow*, **a**–**d**) retracted proximally, superficial to biceps brachii short head. Axial fat-suppressed T2-weighted MRI demonstrate the fluid collection filling the tendinous gap and muscle fiber edema (*arrow*, **e**–**h**)

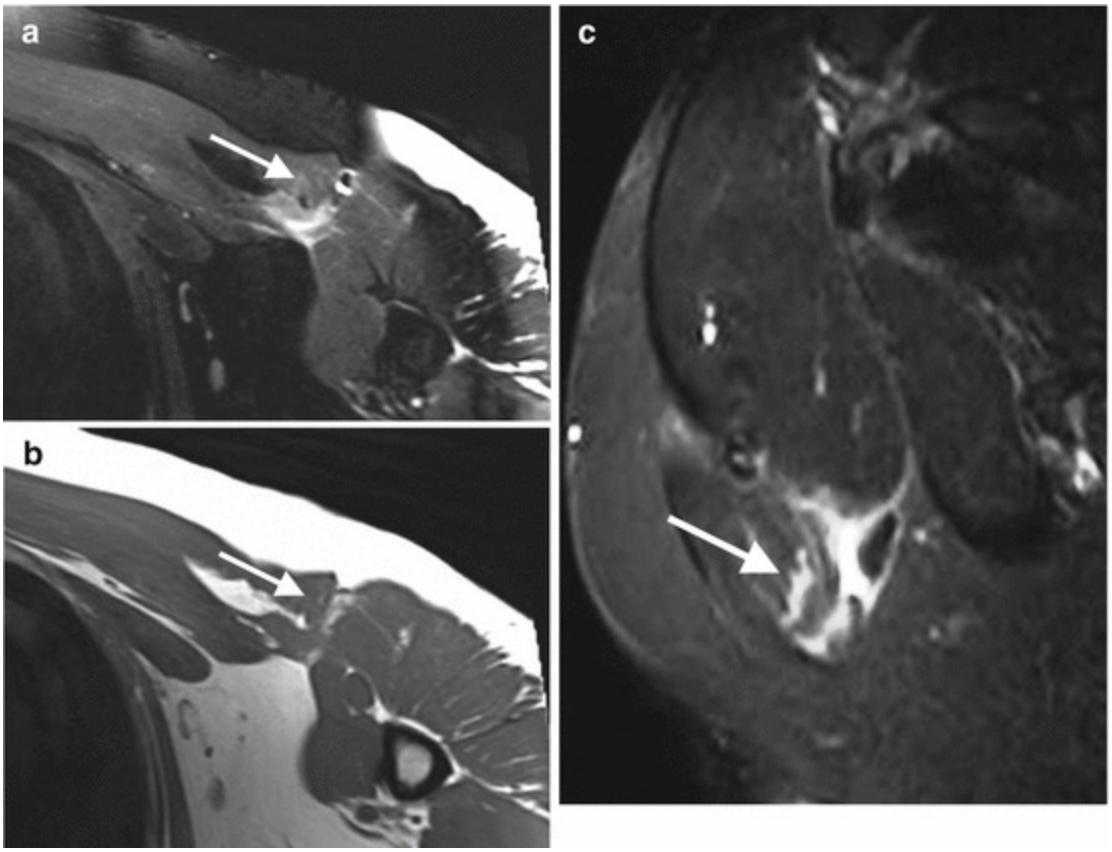


Fig. 27.5 A 39-year-old man with a complete myotendinous junction tear after bench press exercise, 1 week later. Axial fat-suppressed T2-weighted MRI shows a complete distal pectoralis major tear (*arrow*, **a**). Axial T1-weighted image shows the tendon retracted proximally, superficial to biceps brachii short head and coracobrachialis (*arrow*, **b**). Sagittal fat-suppressed T2-weighted MRI demonstrates tendon discontinuity in the distal third (*arrow*, **c**)

Incomplete tears also are better visualized on MRI. In the acute phase, the tear is characterized by edema of the tendon with focal discontinuity. In the chronic phase the diagnosis is made by focal rupture and tendon retraction or even by fibroscar thickening of the tendon (Figs. 27.6, 27.7 and 27.8). In chronic extensive ruptures a thin tendon without frank discontinuity at the humeral insertion is commonly seen that leads to aesthetic deformity of the chest wall (Fig. 27.9)

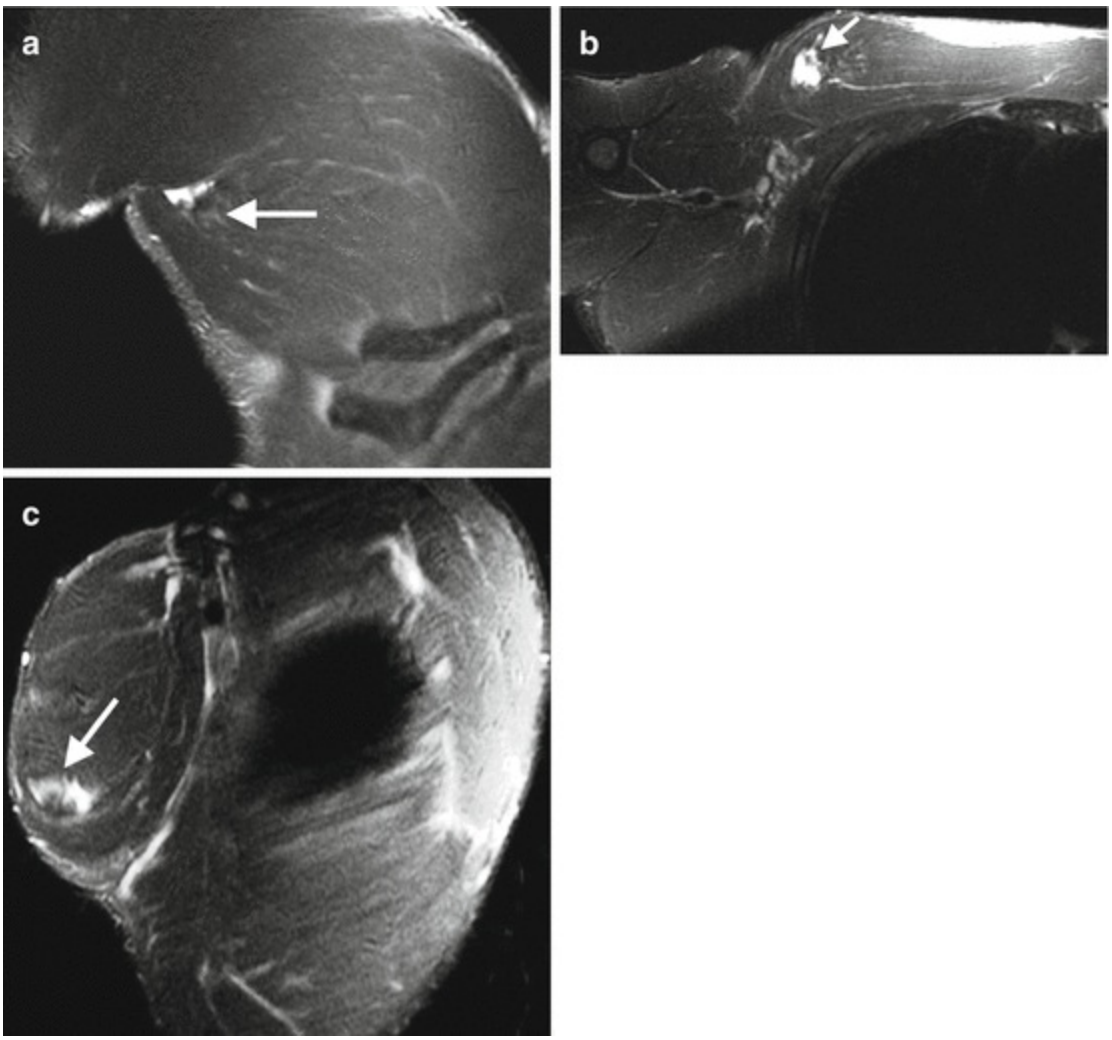


Fig. 27.6 A 26-year-old-man with a partial tear of the myotendinous junction after a jiu-jitsu blow. Coronal fat-suppressed T2-weighted MRI shows focal discontinuity of myotendinous junction (*arrow*, **a**). Axial fat-suppressed T2-weighted MRI shows tendon retraction and a small fluid collection (*arrow*, **b**). Sagittal fat-suppressed T2-weighted MRI demonstrates tendon discontinuity in the distal third (*arrow*, **c**)

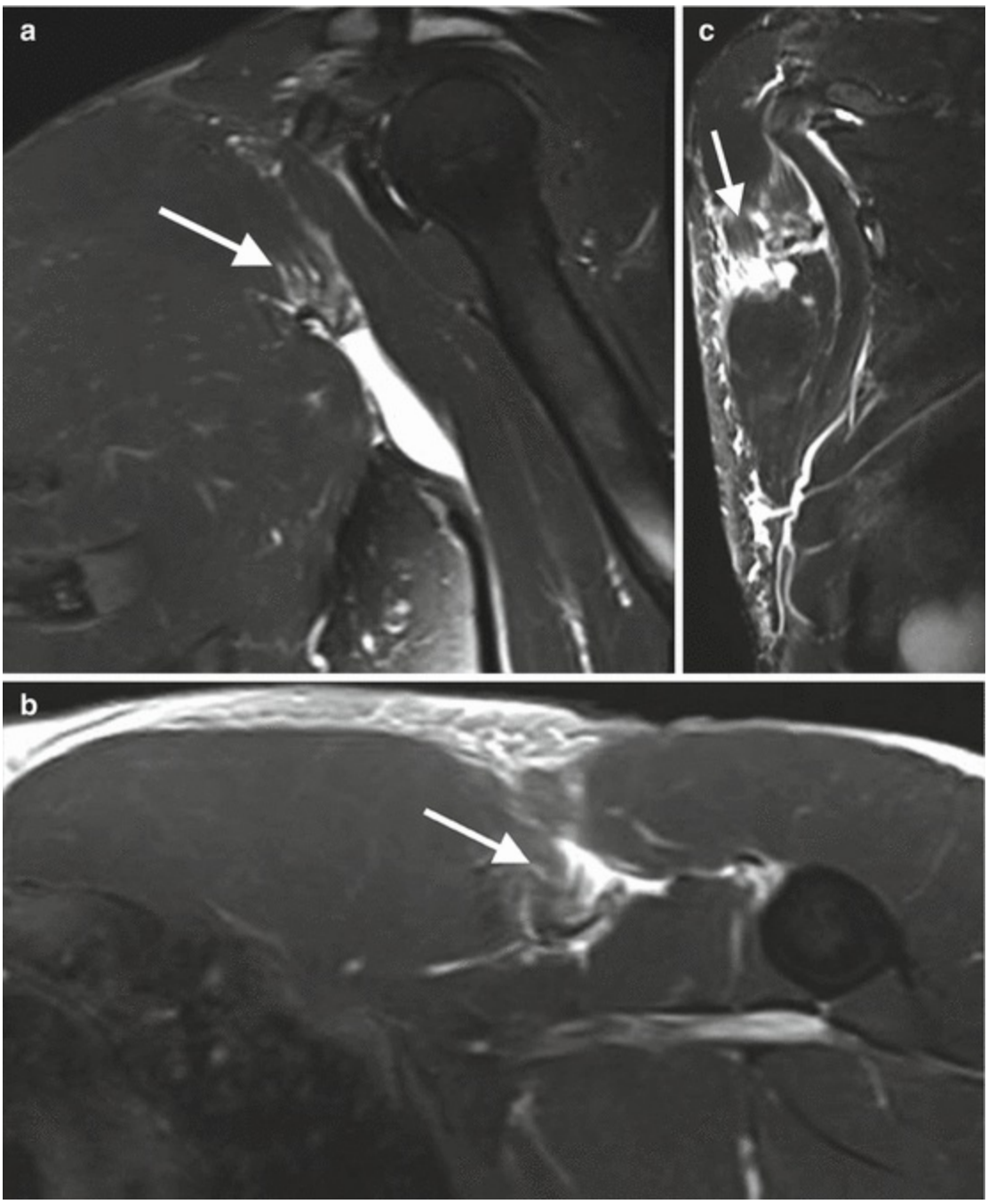


Fig. 27.7 A 23-year-old-man with complete pectoralis major tear at humeral insertion after bench press exercise. Coronal fat-suppressed T2-weighted MRI shows complete tendon retraction (*arrow*, **a**) and a fluid collection. Axial (**b**) and sagittal (**c**) fat-suppressed T2-weighted MRIs show complete discontinuity at humeral insertion (*arrow*)

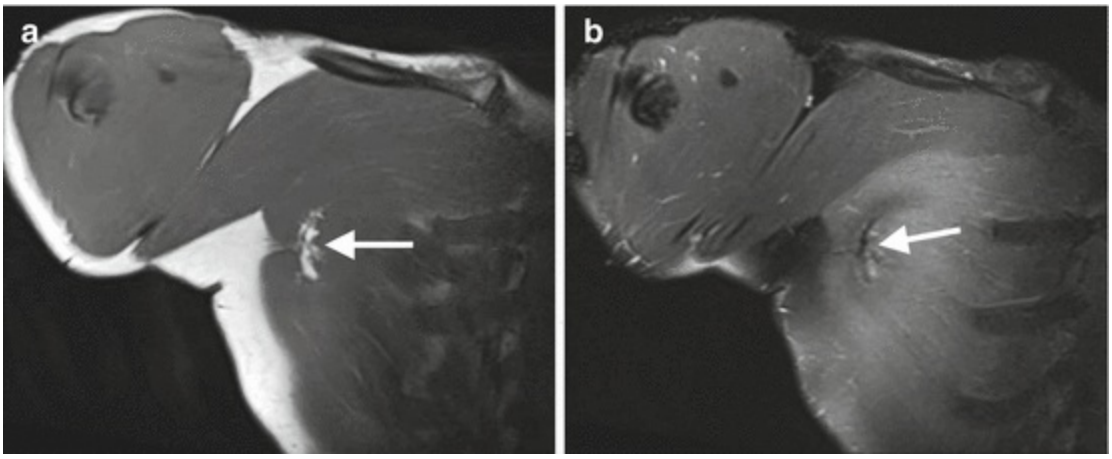


Fig. 27.8 A 37-year-old-man with chronic pain during weight lifting. Coronal T1-weighted MRI (**a**) shows focal tendon discontinuity and proximal retraction suggestive of a chronic partial tear of the musculotendinous junction (*arrow*). Coronal fat-suppressed T2-weighted MRI shows fibroscar alterations of the retracted tendon (*arrow*, **b**)

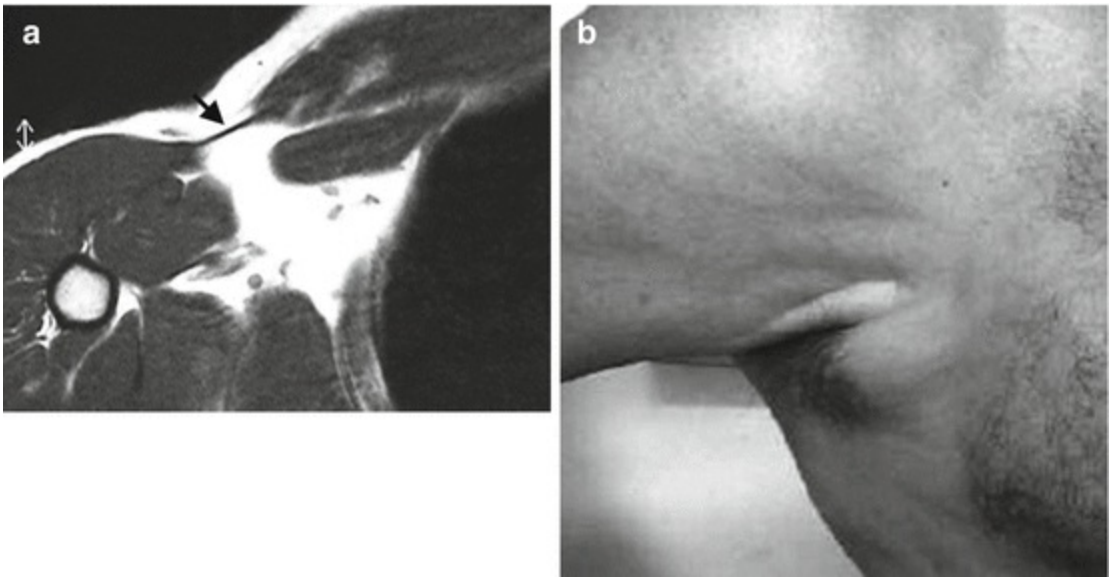


Fig. 27.9 A 38-year-old-man with a chronic rupture of the myotendinous junction. Axial T1-weighted MRI shows a thin tendon (*arrow*, **a**) without frank discontinuity, suggesting a high grade tear that leads to aesthetic deformity of the chest wall as shown in this picture (**b**)

27.6 Ultrasound

The patient is seated in front of the examiner with the arm in neutral position. The anatomical landmark for identification of the pectoralis major insertion is the bicipital groove. As we move the transducer inferiorly, the pectoralis tendon will appear like a hyperechoic and fibrillar structure that crosses the biceps tendon and inserts in the lateral edge of the groove (Fig. 27.10). Dynamic maneuvers like external rotation of the arm or active contraction of the muscle belly can be used to improve the visualization of the tendon insertion especially in partial tears without retraction of the tendon.

Comparison with the contralateral side also can be used in diagnostic doubt (Fig. 27.11) [8].

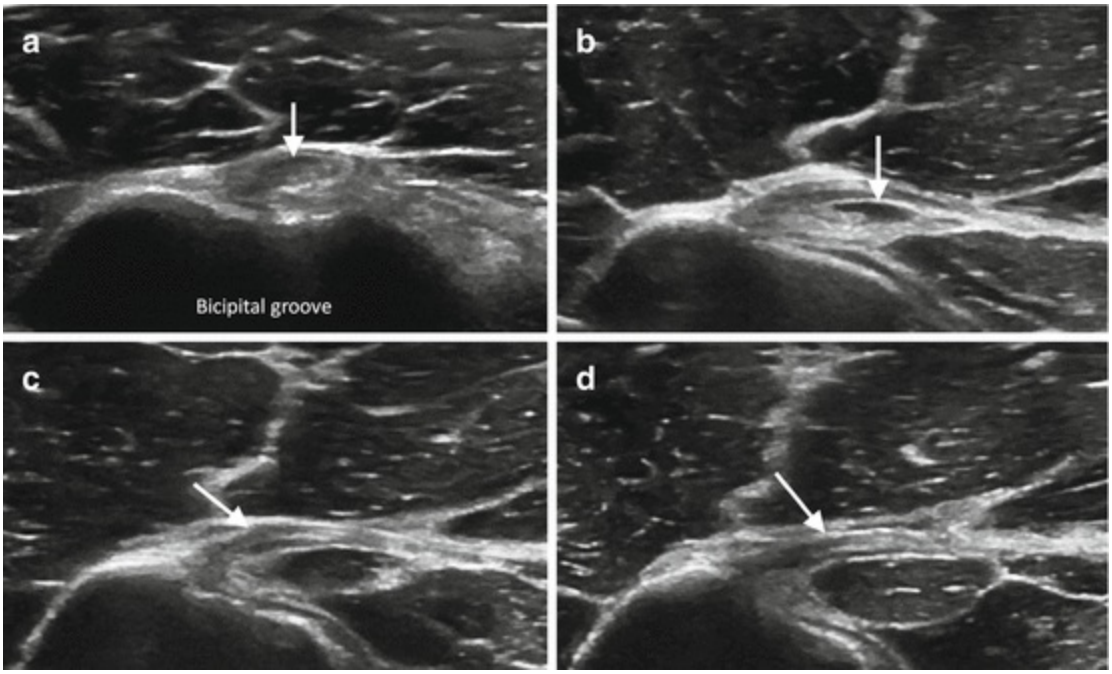


Fig. 27.10 Normal transverse ultrasound appearance of the pectoralis tendon insertion. The bicipital groove is used as an anatomical landmark (**a**, **b**). As we move the transducer inferiorly the pectoralis tendon (*arrow*) will cross the tendon biceps tendon (*arrow*) and insert in the lateral edge of the groove (**c**, **d**)

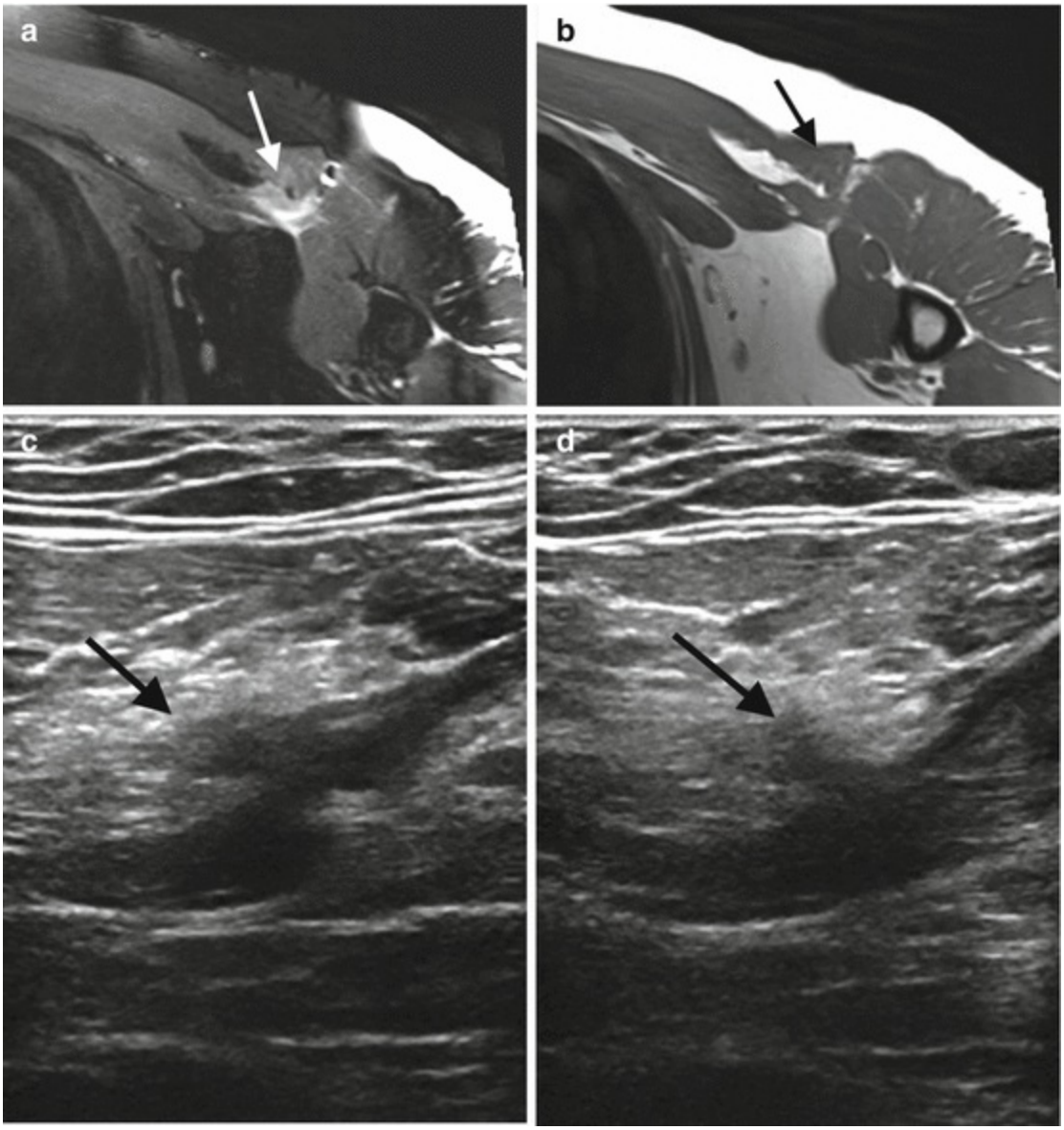
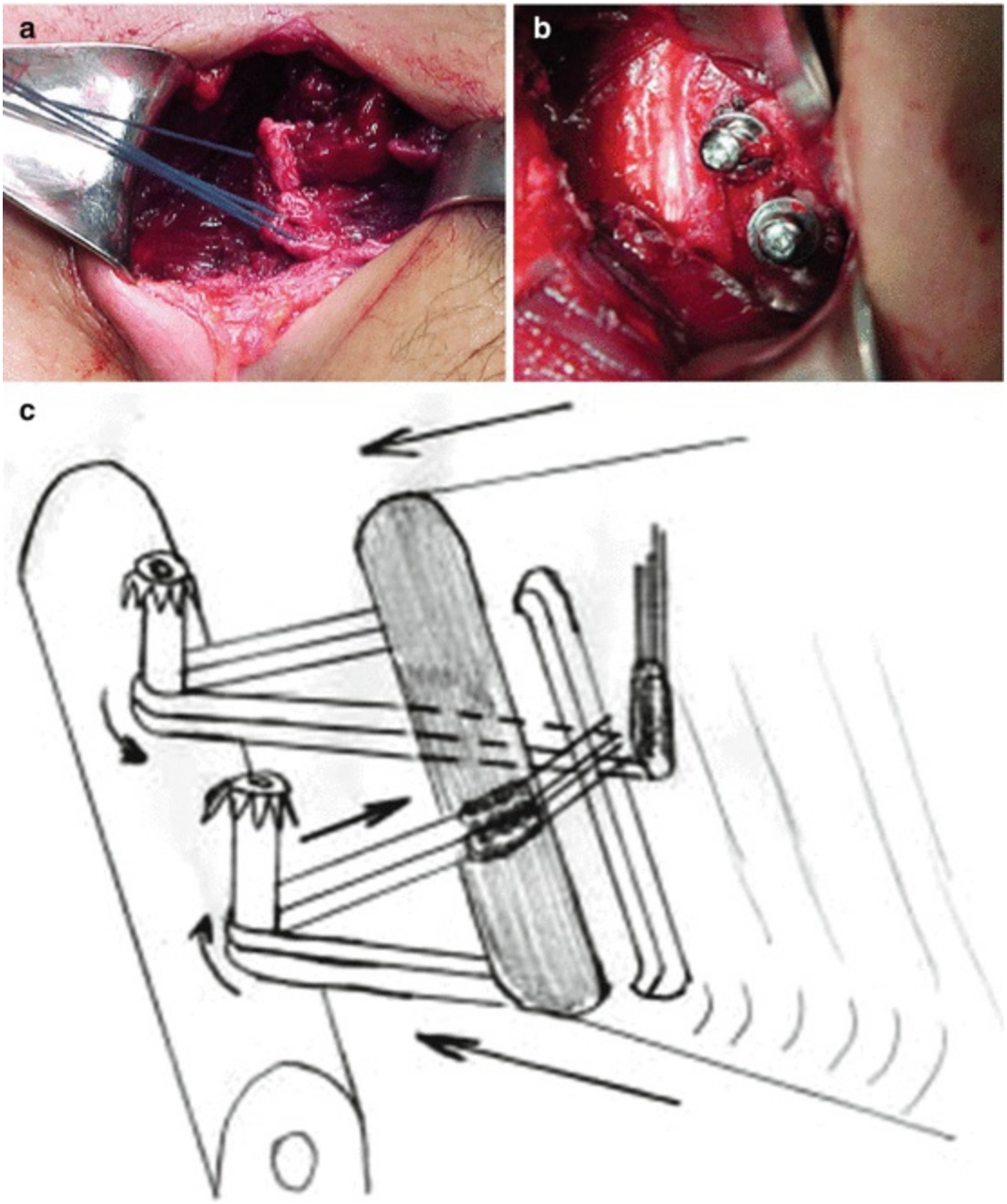


Fig. 27.11 Ultrasound appearance of a complete myotendinous junction tear. Axial fat-suppressed T2-weighted (**a**) and T1-weighted (**b**) MRI show a complete distal pectoralis major tear (*arrow*). Transverse ultrasound images (**c**, **d**) show the torn and retracted tendon at myotendinous junction. An important finding that signals a tear of the pectoralis tendon is the hypoechoic fluid collection at the musculotendinous junction or humeral insertion (*arrow*, **c**, **d**), beyond the concomitant tendon discontinuity

27.7 Postoperative Tendon

Regardless of the surgical technique used to repair the tendon (tendon to tendon, bone trough, suture anchors and cortical buttons) MRI can distinguish tendon integrity (Fig. 27.12) from scarring (Fig. 27.13) and metal artifacts (Figs. 27.14 and 27.15). It is the diagnostic method of choice for postoperative follow up [9–14].


a
b
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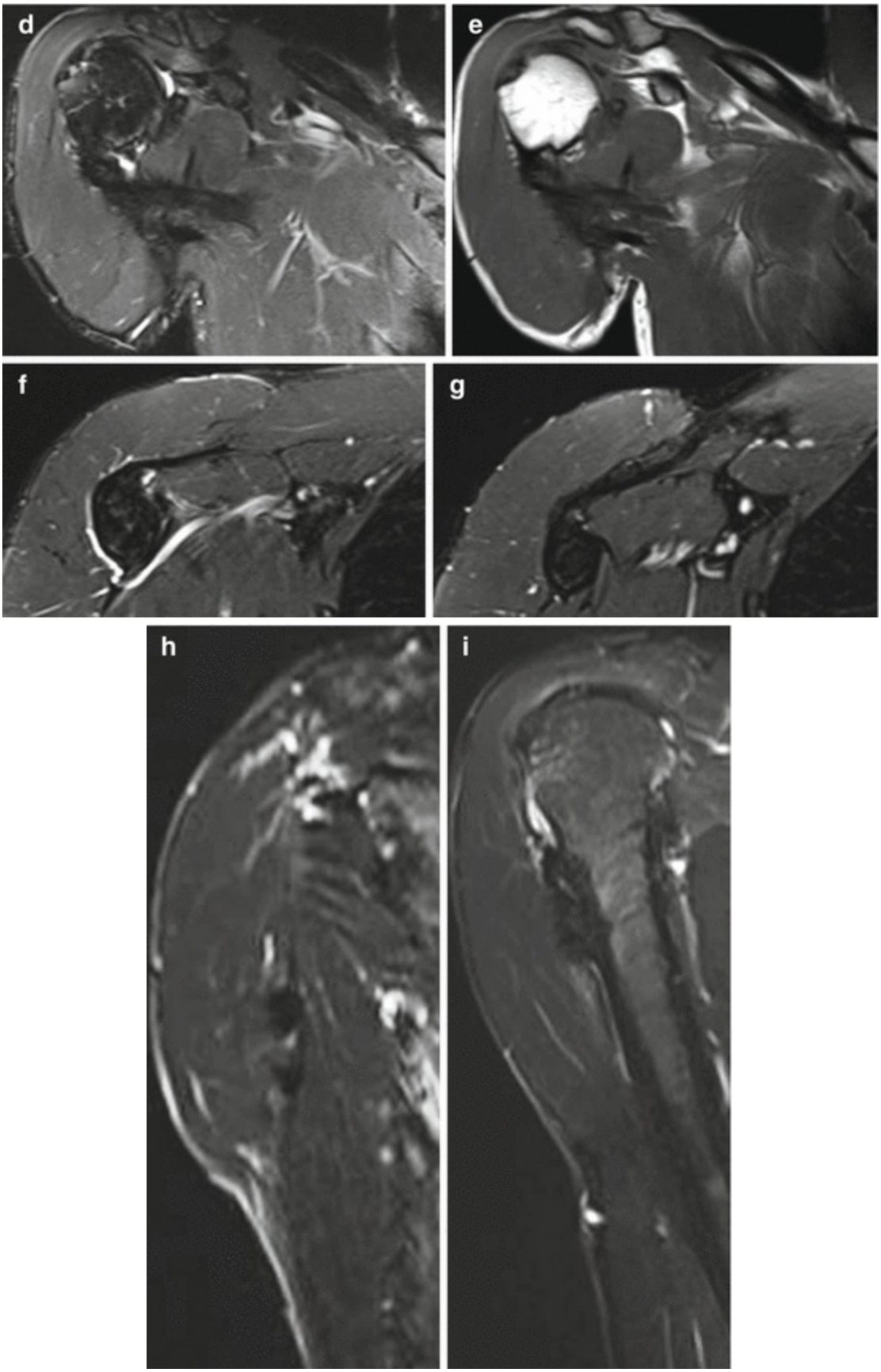


Fig. 27.12 Postoperative follow-up of pectoralis tendon repair. Preoperative images (**a**, **b**) and drawing (**c**) show the technique using screws and washers for fixation next to the proximal humerus. MRI in the three orthogonal planes

(**d**–**i**) shows good progress of the repair and tendon continuity

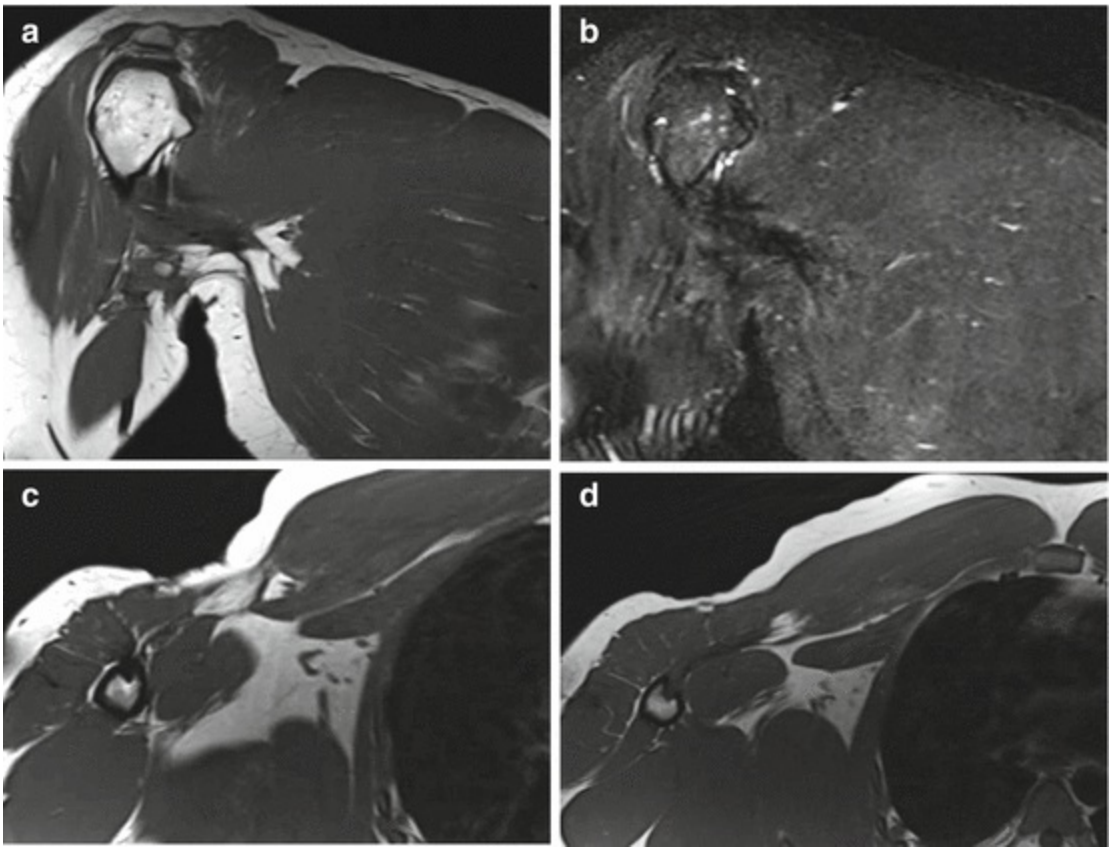


Fig. 27.13 Postoperative follow-up of pectoralis tendon repair using the bone trough technique. Coronal T1-weighted (**a**) and fat-suppressed T2-weighted (**b**) as well as axial T1-weighted MRI (**c**, **d**) show fibroscar alterations at humeral insertion

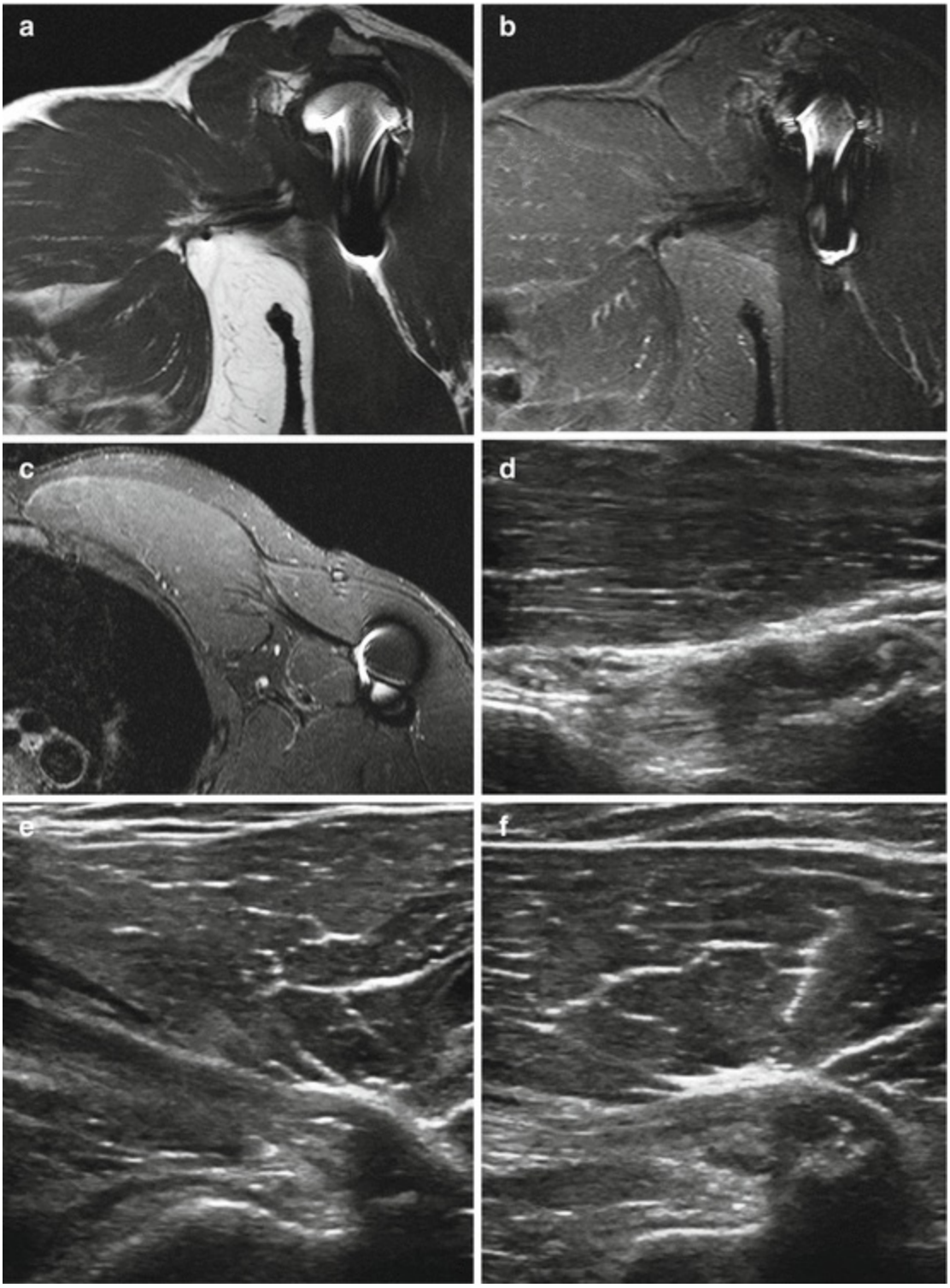
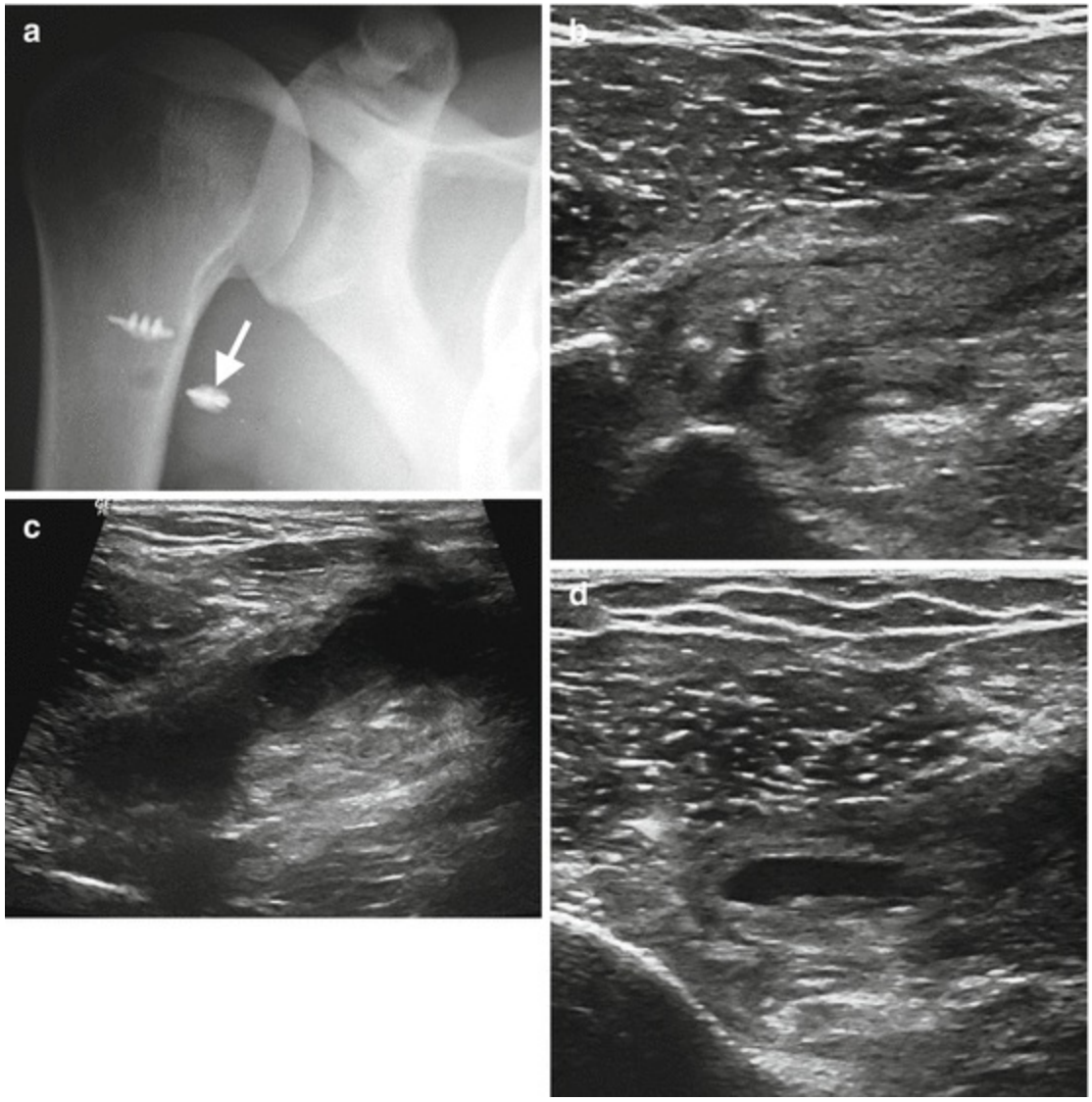


Fig. 27.14 Postoperative follow up of a pectoralis tendon repair using suture anchors. Coronal T1-weighted (**a**) and fat-suppressed T2-weighted (**b**) as well as axial fat-suppressed T2-weighted (**c**) MRI show metal artifacts at the humeral insertion with partial image distortion. Additional transverse ultrasound images (**d**–**f**) show tendon continuity


a
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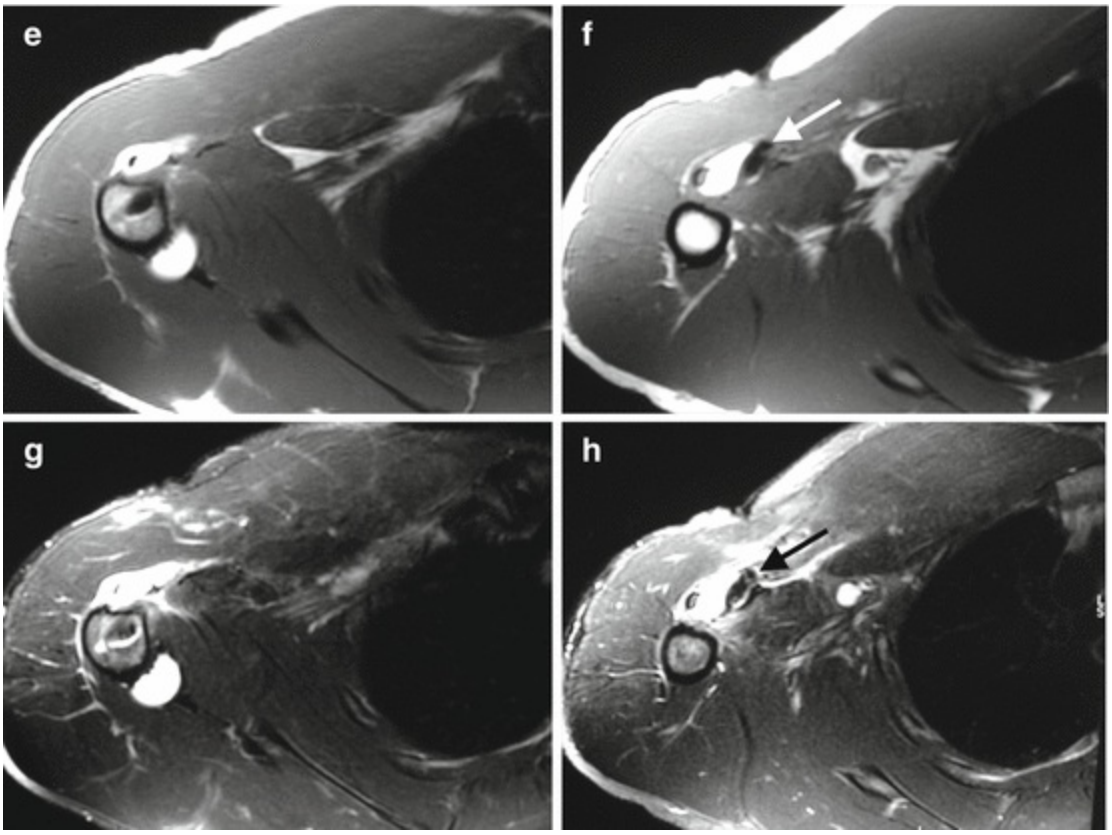

Fig. 27.15 Re-rupture of pectoralis tendon major tendon 45 days after surgical repair with surgical anchors. Anteroposterior radiograph of the shoulder (**a**) shows dislocation of the inferior anchor (*arrow*). Transverse ultrasound images (**b**–**d**) show fluid collection and hyperechogenic foci at humeral insertion. Axial proton density-weighted MRI (**e**–**h**) show the inferior anchor (*arrow*) displaced proximally and a fluid collection interposed at the humeral insertion

27.8 Differential Diagnosis

27.8.1 Calcific Tendonitis

A prevalent pathology that can mimic pectoralis tendon injuries is calcific tendonitis, which can lead to significant pain and decreased range of movement in middle aged patients. Calcification foci, typically at the tendon insertion, and clinical history suggest the diagnosis (Figs. 27.16 and 27.17).

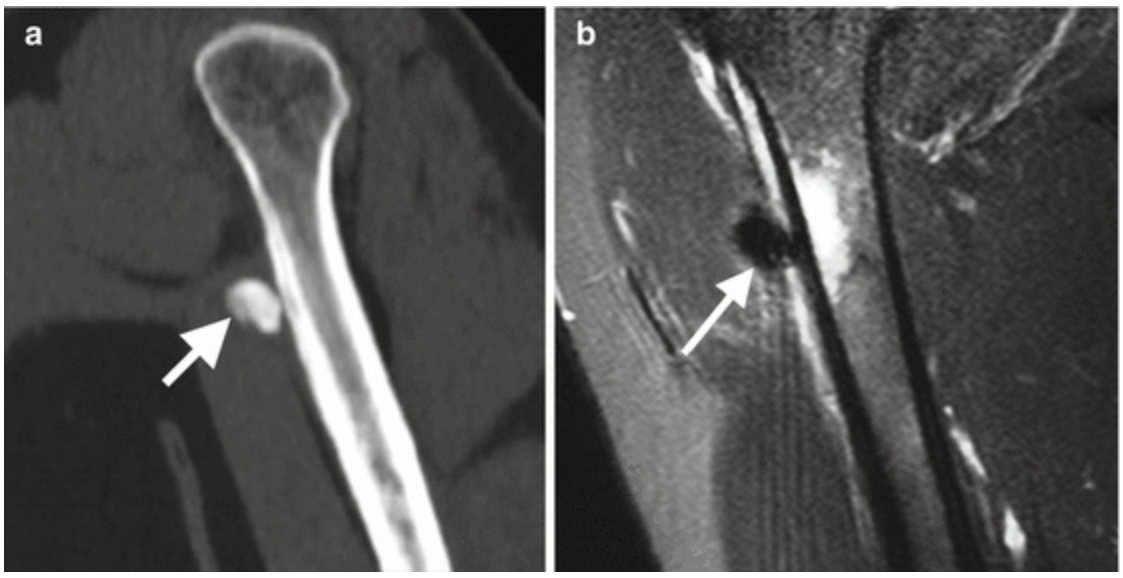

Fig. 27.16 Calcific tendinitis of the pectoralis major tendon at the humeral insertion in a 48-year-old-man with intense pain that limits daily physical activity. Coronal reformatted CT image (**a**) and coronal fat-suppressed T2-weighted MRI (**b**) show calcification (*arrow*), erosions and cortical irregularity with medullar edema

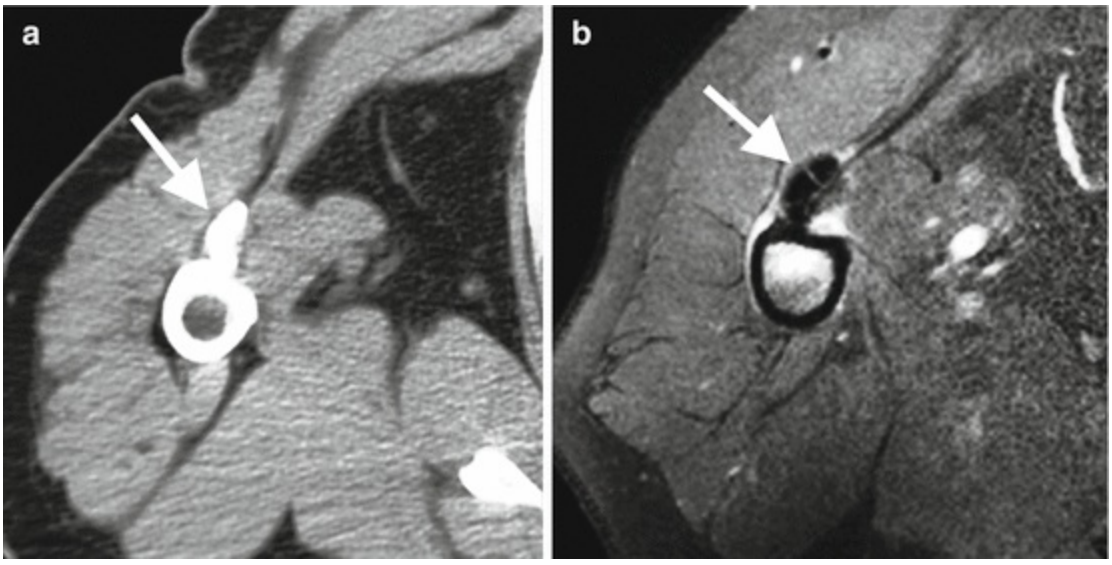

Fig. 27.17 A 53-year-old-woman with excruciating pain. Axial CT image (**a**) and axial fat-suppression T2-weighted MRI (**b**) show grouped calcification foci (*arrow*), the typical "comet-tail" calcification at the distal pectoralis major tendon and cortical irregularity

27.9 Conclusion

Diagnosis of pectoralis major tears requires adequate knowledge of the complex anatomy of the muscle by the radiologist and a high grade of suspicion by the clinician. MRI should be considered in the evaluation of these patients, with the aim of early diagnosis and, when appropriate, surgical repair.

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Part VI
Imaging Specifics

Bernard Roger, Ali Guermazi and Abdalla Skaf (eds.), Muscle Injuries in Sport Athletes, Sports and Traumatology,
DOI 10.1007/978-3-319-43344-8_28

28. Plasma Rich in Growth Factors for the Treatment of Skeletal Muscle Injury

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Abstract
Skeletal muscle tissue represents between 35 and 50 % of an adult's body weight, and it responds efficiently to changes in homeostasis. Muscle injuries are some of the most common sporting injuries and cause alterations that disrupt the force-transmission chain and result in functional impotence. Current treatments for muscle injuries have not undergone any major changes in recent years irrespective of the level of sport practiced, and their appropriate treatment remains a daunting clinical challenge. One innovative biological approach is intra-muscular injection of platelet rich plasma (PRP), which creates a suitable microenvironment to accelerate repair processes. Appropriate treatment requires an adequate diagnosis of the injury, which must include clinical history, physical examination and complementary tests. Ultrasound and magnetic resonance imaging (MRI) techniques are required for muscle injures, not only for diagnosis but also in the application of PRP that will be carried out once the type and level of injury have been defined. The chapter ends with a description of the protocol we have developed including PRP elaboration, patient preparation and PRP infiltration, indispensable factors for a speedy and successful recovery of the athlete.

28.1 Introduction

Skeletal muscle tissue in humans represents between 35 and 50 % of adult body weight, depending on age, sex, diet, and level of physical activity. Skeletal muscles are extremely plastic and dynamic organs capable of responding efficiently to changes in homeostasis. Protein synthesis and degradation are coordinated and regulated by cellular signaling pathways that are influenced by mechanical stress, physical activity, growth factors and the availability of nutrients. By converting chemical energy into mechanical force, thereby generating movement, muscles allow the body to survive and adapt to its surroundings. They are the connection between the environment and gene expression of myofibers, acting like an interface between the environment and the central nervous system. Moreover, muscles are mechanical brakes and buffers or springs, and besides their purely mechanical roles, perform other functions, such as metabolic, temperature-regulating, or endocrine roles.

Long overlooked as a non-genetic input in tissue patterning and remodeling, mechanical load or stress plays a crucial role both in the diversity of cell phenotypes and their functions found in the skeleton [36]. Mammalian muscles are made up of a purposeful-looking array of ingredients such as the cells known as muscle fibers or myofibers, which can reach up to 30 cm in length. In addition, there is a highly organized complex extracellular matrix (ECM) made up mainly of water and collagens, perlecan, laminins, tenascin C, fibronectin, entactin, several glycoproteins and proteoglycans as well as metalloproteases which account for about 1–10 % of the muscle tissue [16]. The ECM adopts a net-support configuration that confers elasticity and encompasses nerve endings, blood vessels, fibroblasts as well as adipocytes and macrophages, and forms a scaffold (endomysium) that intimately surrounds each myofibers. Included in this ECM and intimately related to the sarcolemma, there is a highly specialized interstitial connective tissue organized in a layer known as the basement membrane which is made up of laminins, collagen IV, nidogens and glycosaminoglycans. The basement membrane is closely bound to the sarcolemma by integrins and dystrophins, and is involved mainly in compartmentalization, although its molecular composition endows it with adhesive and inductive functions for a variety of cell fates [28].

The mechanical energy generated in the actin/myosin myofilaments of sarcomeres is transferred from them to the sarcolemma and then through the basement membrane to the ECM and finally to the tendon. These structures operate as the highway of mechanical energy known as mechano-transduction. In addition to the structural and mechanical support for muscle tissue, the molecular composition of the ECM is crucial for a wide range of cellular behavior such as adhesion, migration, growth and differentiation, since laminins, collagens and other proteins located in the ECM play a vital role as signaling molecules and binding sites for many cells [10], thereby inducing and organizing muscle

tissue in development and repair processes. Since virtually all the cells that make up the musculoskeletal tissues are mechano-sensitive and experience mechanical stress through the distortion of the ECM complex and respond to them by changing their cellular biochemistry and physiology (mechano-transduction and plastic adaptation), mechanical forces are required to maintain the physical integrity of anatomical structures and homeostasis of the tissues by regulating cell functions, including gene induction, protein synthesis, and cell proliferation, differentiation, growth, survival and apoptosis [20, 33]. It has in fact been understood that whereas growth factors or soluble factors drive tissue development, mechanical factors govern tissue pattern [19].

Adult skeletal muscles are made up of multinucleated myofibers (fibers) which are established during embryogenesis by the fusion of myogenic cells (myoblasts). Adult myofibers, on the other hand, show their plasticity, namely, muscle growth and repair processes (in the absence of myofibers necrosis and not involving an inflammatory response) by changing either fiber size, in response to physical activity (disuse atrophy/hypertrophy), nutrition status, inflammation, denervation and age, or fiber type (fast-to-slow or slow-to-fast switch), in response to the type of exercise and denervation, or even by forming new myofibers (or segments of them) as a result of injury or damage [9, 29]. This myofiber plasticity is influenced by the balance of protein synthesis and degradation which can cause the loss or increase of organelles and cytoplasm, and/or by the natural cell-cell fusion of myoblasts, to generate multinucleated adult skeletal myofibers which are mainly fueled by the addition of new myonuclei and myofibers involving proliferation of satellite cells [29]. However, the muscle regeneration process, defined as the formation of new myofibers (or segments of them) after necrosis has become established, deploys different patterns of tissue remodeling after injury, illustrating how muscle regeneration is an open condition-sensitive process ruled by microenvironmental cues (mainly mechanical and physical-chemical). As a paradigm for regenerative biology, skeletal muscle may be seen as tissue that conserves and shares modules of regulatory pathways and transcription factors of embryonic myogenesis and development which are redeployed for tissue repair and regeneration after muscle injury [13, 14].

28.2 Skeletal Muscle Injury and Repair Process

Muscle injuries are some of the most common sporting injuries, irrespective of the level of sport practiced, accounting for between 10 and 55 % of all such injuries and encompassing contusions, strain, and lacerations [21]. In football, for example, muscle injuries are responsible for the largest number of both training and competitive days lost. The severity of this type of injury depends on the functional inability to train and, obviously, compete; in many cases this functional loss lasts for 30–40 days.

The most common mechanism of skeletal muscle strain in elite sportsmen is the

concentric/eccentric muscle movements associated with high levels of explosive force in response to sharp changes in direction and speed. This type of muscle exercise injury is one of the physiological injury models. Biarticular muscles such as the hamstrings, rectus femoris, calf muscles, or femoral biceps are most commonly affected by muscle ruptures or tears, although the bruising and trauma resulting from direct impact of the muscle mass during activation should also be taken into account. Muscle damage, whether extrinsic (bruising) or intrinsic (strain-rupture), brings about necrosis and destruction of the constituents of muscle tissue such as sarcomeres, the sarcolemma, capillaries, or other extracellular matrix elements such as integrins and dystrophins, depending on the intensity of the force and myofibers, although the impairment of their basal lamina might be diverse in degrees. This alteration disrupts the force-transmission chain and results in a functional impotence. Furthermore, it generates a tissue necrosis area, mainly due to the mass entry of calcium into the muscle cells and subsequent activation of proteases such as calpains that break down myofibrils and other cell constituents. The resulting hematoma from the torn blood vessels fills the gap created between the already retracted myofiber stumps. The processes of defense, proliferation, regeneration, maturation and remodeling of the different cells and structures in muscle tissue slowly take over in a spatial and temporal interaction [7].

As part of the endomysium, cells, basal lamina and peripheral capillaries may present a disruption in muscle tears or bruising. Such ruptures often affect the neuromuscular junction itself, resulting in stumps of "non-innervated" muscle fibers. The most common site for muscle ruptures is the region known as the myotendinous junction (MTJ). Subsequent to a muscle rupture and regardless of the mechanisms that bring about the injury or impairment, muscle regeneration as a spontaneous event involves the launch of a series of biological programs such as local defense, myogenesis, angiogenesis, reinnervation, and remodeling, and spans several hierarchical levels from genetic through molecular and cellular levels to tissue and organ levels. From the beginning of the process, angiogenesis and neovascularization appear to be crucial in functional muscle regeneration, furnishing the new tissue with oxygen and other blood-derived cells and nutrients, at the same time removing carbon dioxide and other tissue-waste products. Innervation, another tissue process that shows an exploratory behavior, is essential for growth and maturation of newly formed myofibers. In general, reinnervation occurs at the original junctional basement membrane which is endowed with a specific memory [15] provided that the fibrotic scar tissue does not impinge on and infiltrate the basement membrane and thereby preventing the progression of axons.

These events are regulated by multiple soluble molecules including cytokines and growth factors released by several cell processes, and by biochemical and cell signaling pathways, such as Notch-1, PI3K/Akt or NF-kB [32] or those arising due to mechanical stress [9]. It is worth recalling that damaged muscle cells (myofibers) in

mammals have a poor potential to repair themselves once they have undergone necrosis, and end up generating a granulation tissue which will be replaced by a fibrotic scar tissue. Nevertheless, new muscle might form from the proliferation (regeneration) of the satellite cells. These cells are considered to be muscle stem cells, located between the surface of myofibers and the basal lamina, which can somehow redeploy as in embryo development. When the mechanical damage is less severe and the tissue impairment is reversible, in the absence of necrosis and cell death, there is merely a disruption of subcellular architectural organization, giving tissue the potential to inaugurate the repair or restoration process [15]. Under physiological conditions, muscles generate and respond very differently in terms of duration, intensity, type of muscle action, and frequency, meaning that this tissue remodels and repairs "small damage foci" by activating various basic biological programs. For example, damage to the cell membrane or sarcolemma as a result of eccentric muscle actions (simultaneous braking and stabilization, such as walking downhill for example) is repaired by the muscle cell by the gluing/fusion of vesicles obtained from the sub-sarcolemma. This type of repair occurs under the action of dysferlin [9].

28.3 Cellular and Molecular Mechanisms Regulating Muscle Repair and Regeneration

Skeletal muscle tissue responds to sports-related muscle injuries on three timescales. First, the biological defense programs are initiated immediately after the injury (0 to 2–3 days) by activation of hemostasis and the innate immune system, thereby preventing possible hemorrhage and infection as well as sealing the injured area. The pain episode entails a natural period of immobilization, hence mechanical stress should be avoided. Both platelets and macrophages in the tissue itself synthesize and release bioactive substances and growth factors such as PDGF, VEGF, HGF, TGFB, TNF and IL-6 mainly with chemotactic, migratory and satellite cell activation effects, attracting monocytes and more macrophages to the damaged area [7, 9, 31]. The contribution of cells from the innate immune system is important in this repair process as the macrophages adopt a proinflammatory phenotype in this microenvironment, thereby phagocytosing tissue debris and cleaning the necrotic zone. This phagocytosis and proteolysis is essential to ensure that subsequent repair processes commence in the rupture zone, which is currently occupied by a fibrin clot. The other organic cell defense line, namely the platelet, belongs to the hemostasis and clotting system. The hematoma organizes itself into a clot while the previous process is under way. Platelets come into contact with fibronectin and collagen from the extracellular matrix, thereby triggering the release of growth factors which, together with those released by macrophages, endothelial cells, and pericytes, stimulate tissue repair [7, 9].

By 48 h, fibrin and fibronectin have created a matrix/clot that serves as a bridge between the edges of the myofibers affected, thereby demarcating the repair site [22]. Platelets, endothelial cells, macrophages, and proliferating satellite cells all express growth factors, which in turn, stimulate other cells such as fibroblasts that initially synthesize type III collagen (type I collagen after day 5 or 6), tenascin, and fibronectin, as well as integrins that will laterally affix the broken edges to adjacent fibers. This new tissue re-establishes the bond between the ends, although the tissue formed is very fragile and somewhat elastic [21]. Angiogenesis is involved in the synthesis of new capillaries and starts with endothelial cells and pericytes induced by VEGF in this fibrous callus that now joins the ends of the various broken fibers while the matrix continues to be infiltrated with macrophages. As a result of the new microenvironment created within this callus by the activity of fibroblasts, myoblasts, and endothelial cells, these macrophages express an anti-inflammatory phenotype, releasing TGF-β and IL-10 [8].

On a longer timescale (4–15 days), and through appropriate environmental influences, adaptive plasticity enables the emergence of cell phenotypes which are apt to generate an ECM that will face external cues (mechanical load). Angiogenesis, myogenesis and reinnervation overlap with the ECM regeneration process. In other words, these events run in parallel with the synthesis of the interstitial tissue (synthesis of tenascin C, fibronectin, collagen, and other proteins) that make up what is traditionally known as the fibrous callus. Around 6 or 7 days after the injury, myogenesis is already generating differentiated myoblasts which are integrated into the fibrin matrix that penetrates and infiltrates the damaged stumps. Within the fibrin scaffold, myoblasts differentiate and fuse together to form myotubes which then evolve into the synthesis of a new cell, a process that may be somewhat slower. Moderate sustained mechanical load modulates the fusion and ensuing alignment of myoblasts into myofibers [11] and minimizes or even avoids the formation of scar tissue by inhibiting the NF-kB of fibroblasts which might promote fibrotic scar. If an early and controlled mobilization of the damaged zone containing the callus/bridge or fibrotic zone is initiated at this stage, the maturation-remodeling process can be triggered, thus resulting in a reciprocal activity among:

- The orientation of the myoblasts inside the matrix, a diversion of tenascin and fibronectin synthesis towards the synthesis of more type I collagen and its reorientation, while not blocking the presence or fusion of either myoblasts or nerve endings [23];
- The creation of lateral connections via integrins;
- Contact with new capillaries.

This is the ideal three-dimensional microenvironment to ensure that the capillary

buds join together and do not collapse. Once the clot has formed (from the initial hematoma), the integrity of the basement membrane and the 3D structure provided by the fibrin callus or matrix are essential for myogenesis and reinnervation. As a result of the growth of new axons from adjacent nerves, reinnervation and the creation of a new neuromuscular junction in the repaired or regenerated fibers may be driven by NGF and IGF-1, both of which are present in the damaged tissue and synthesized by muscle cells and fibroblasts under paracrine influence [7]. Repair of the basement membrane is the first key step in reconstruction of the neural canal (space in the fibrillar void) that ensures subsequent compartmentalization of the repair phenomena. It should also be noted that the presence of tenascin C in the extracellular matrix, the synthesis of which is induced by mechanical stress, is a prerequisite for muscle reinnervation. The path used by the axons (myelin synthesis), whose viability is enhanced by the TGF-βand fibrin network to reach the new fiber and establish a neuromuscular connection, is via the basement membrane. As a result, it is important that the newly formed granulation tissue which joins the damaged fibers together does not form a barrier to progress of the axon from neighboring nerve endings [22, 23] and does not surround them with fibrosis resulting from excess collagen synthesis or defective MMP synthesis.

During the repair process, the existence of a mechanical stimulus causes integrins to laterally bind the edges of muscle cells to the extracellular matrix via laminins, thereby preventing them from retracting [23] and contributing to the repair process. Both the gradual and controlled mechanical stimulus that induces IGF synthesis by muscle cells (by endocrine and paracrine activity) and the paracrine and autocrine synthesis of growth factors such as HGF and TGF-β by fibroblasts during the final remodeling phase appear to be essential, since both these signals may have a synergic effect on the activity of the fibroblasts that are remodeling the ECM [25] and repaired tissue. Controlled exercise helps to reorient type I collagen, thereby enhancing the penetration and alignment of myoblasts and stimulating remodeling [11, 21, 23].

In the wake of these biological processes, about 15 days after sustaining the injury the new tissue begins to acquire the order and configuration of its components, thereby recovering its function, the goal of the whole repair process. The correct coordination and spatial and time sequence of the different steps in this biological muscle repair program will be key to re-establishing the structural integrity and functional properties of the repaired tissue. However, the fibers also need an electrical stimulus during the repair process. This means that a new neuro-muscular junction containing this repaired fiber or, if applicable, the new fiber formed, must be established beforehand in the corresponding motor end-plate [9]. It is important to note that each fiber or skeletal muscle cell connects to a motor neuron at a single point known as the motor end-plate or neuromuscular junction, and this connection is essential for cell trophism and function (in contrast to cardiac muscle cells). As such, a rupture of the fiber may directly affect the neuromuscular junction or leave one of the stumps with no neural connection [22,

23]. As with the vascular system, the nervous system also exhibits an exploratory behavior by searching for, and establishing connections with muscle cells, probably while angiogenesis is underway [14].

28.4 An Innovative Biological Approach to the Treatment of Muscle Injuries: Plasma Rich in Growth Factors (PRGF-Endoret)

The appropriate treatment of muscle injuries remains a daunting clinical challenge. Since muscle tissue is a complex mechano-sensitive tissue, every pharmacological and surgical therapy should be assisted by mechano-therapy [24]. In this respect, and as a clinical application of cell mechano-transduction, a rehabilitation program which included the employment of PRGF in a synergistic manner would play a crucial role in both promoting the repair and remodeling of injured tissue.

One innovative biological approach is the application of platelet rich plasma (PRP) in intra-muscular injections. Although a universally accepted definition of PRPs in terms of platelet concentration and presence or absence of leukocytes is lacking, PRP products include plasma and twofold or more increases in platelet concentrations above baseline levels, while the concentration of leukocytes and erythrocytes varies widely [2, 12] from a complete absence to a high concentration. Plasma rich in growth factors (PRGF-Endoret) is an endogenous blood-derived product which conveys growth factors, cytokines, and morphogens contained in the platelets as well as fibrinogen and other plasmatic proteins in a biologically balanced aggregate that does not contain leukocytes. It is managed and delivered pharmacologically [2, 3]. The process of platelet activation and hydrolysis of prothrombin into thrombin is driven by the addition of calcium chloride, simultaneously causing the release of a plethora of growth factors and the polymerization of fibrin [5]. Once activated, the liquid formulation is quickly injected as a solution into muscle, and due to its local “in vitro” and gradual “in vivo” activation and homogeneous distribution through and interaction with the ECM of muscles, it is converted into a matrix-like viscous and malleable structure [5]. After the intramuscular infiltration over the injured areas, afibrin-scaffolding formed in situ as an extracellular matrix serves as a highway for mechanical energy to transit from the environment to the cell, thereby bridging cell-to-cell tissue transition, promoting multi-cellular assembly and providing mechanical support as well as endowing tissues with a suitable microenvironment for biological restoration [27]. Since they are autologous, bio-reabsorbable, bio-compatible, and free of leukocytes and red cells, PRGF scaffolds are the best tailored among all the tissue engineering materials.

Current treatments indicated for muscle injuries have not undergone any great change in recent years. Only recently, some novel techniques seem to have emerged, but

none of these have yet been supported by a body of evidence in basic science or in clinical research [18]. It is fair to say that none of the other currently available treatments has demonstrated experimental efficacy when administered either in a general or local manner, such as percutaneous infiltrations into a damaged area. These infiltrations involve a large number of molecules and substances that range from homeopathic preparations to autologous serum, including vitamin B and its derivatives. The goal of all these treatments is to improve and accelerate the process of muscle repair, and consequently, to achieve a speedy recovery of the patient to both daily and sports activities as soon as possible. A pilot study conducted in athletes who presented muscle strains showed that the patients treated with autologous serum recovered faster [35].

Concerning PRP, clinical research has not yet clarified the therapeutic effect of this treatment in muscular injuries [1]. Furthermore, the large number of PRP systems on the market and the poor standardization of its applications make difficult its understanding and hamper its correct use. So far, no clinical trials have been conducted showing the improvement of these injuries treated with PRP compared to current treatments such as physical therapy, ice or corticoid injections, and only a few clinical reports seem to shed light on the effect of PRP in muscle damage [17, 26, 34]. However, these pathologies present a high prevalence in athletes and entail lost days from competition and training, making it necessary to search for alternatives to traditional treatments. Our group has designed a rigorous and well defined protocol for the application of different PRGF-Endoret-based formulations in several pathologies, among them muscular injuries. The first results of muscle recovery after administration of growth factors and other bioactive molecules using this technology were presented at the 6th EFFORT Congress in Helsinki in June 2003 [4]. Thereafter, the application of PRGF-Endoret in this field of musculoskeletal injuries has become a standard practice in our clinic [6]. The protocol that we will describe was used by Jaadouni, who discussed in his doctoral thesis the use of PRP (PRGF-Endoret) in acute muscle injuries in athletes. A single PRGF infiltration was performed in a group of 50 patients, most of them rugby players, within the first ten days after injury. Among the results, it highlighted an average recovery of 35 days and a low rate of relapses, finding only one case (2 %), while in the literature the rate of relapse in these muscle injuries is around 30 % [30].

28.5 PRGF-Endoret Protocol Used in Muscle Injuries

Sports-related muscle injuries can be classified as acute such as muscle tears, or chronic like fibrosis and muscle cysts or seromas. To perform an appropriate medical treatment it is essential to conduct an adequate diagnosis of the injury, which has to include clinical history, physical examination and complementary tests. Ultrasound and magnetic resonance imaging (MRI) techniques are specially required in muscle injures,

not only in the diagnosis but also in the application of PRGF-Endoret that will be carried out once type and level of injury have been defined [3].

1. Thirty-six mL of peripheral venous blood is withdrawn into 9-mL tubes containing 3.8 % (wt/vol) sodium. Occasionally, due to the size of the lesion, it may be necessary to extract further amounts of blood. Blood is centrifuged at 580 g for 8 min at room temperature (PRGF-Endoret, Vitoria, Spain). The upper volume of plasma contains a similar number of platelets as peripheral blood, and it is drawn off (F1). The 2-mL plasma fraction, located just above the sedimented red blood cells, is collected in another tube without aspirating the buffy coat. This plasma contains a moderate enrichment in platelets (two to threefold the platelet count of peripheral blood) with scarce leukocytes (F2) (Fig. 28.1).

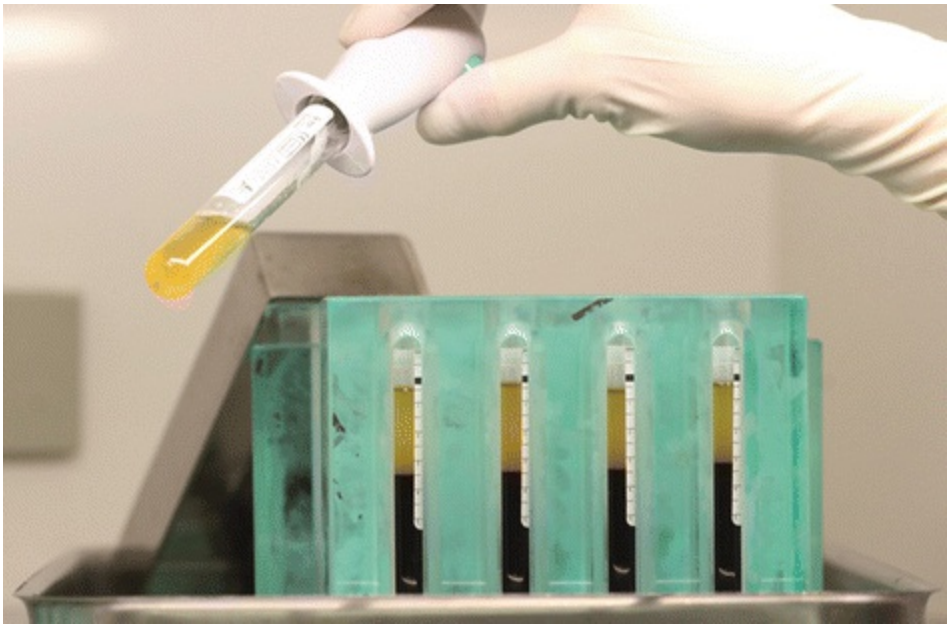

Fig. 28.1. Preparation of liquid PRGF®-Endoret®

2. With ultrasound guidance, the injury and, if applicable, the possible hematoma associated with the muscle tear (seromas and fibrosis in the case of chronic injuries), are located.

3. After locating the lesion, the area of skin to be infiltrated is prepared with a sterile field and antiseptic is applied; the area is demarcated with disposable cloths to perform ultrasound control in a comfortable way (the probe should be covered with a sterile cover) (Fig. 28.2). Next, hematoma, seroma, or cysts, if present, are then punctured/evacuated using a syringe (Fig. 28.3).

4. Once the hematoma is evacuated, F2 is activated with calcium chloride (10 % wt/vol).

5. PRGF-Endoret activated liquid formulation is injected into the injury site under ultrasound guidance (Fig. 28.4). The volume injected should be the maximum possible, depending on the size of the muscle, injury and severity. Although the amount of PRGF-Endoret infiltrated is usually 6–8 mL, volume can reach 10–15 mL.

6. When the injury site has been infiltrated, it is necessary to conduct PRGF-Endoret infiltrations into the peripheral healthy muscle surrounding the injury, including interfascicular and interfibrillar regions. With these infiltrations, satellite cells are activated and mobilized, triggering muscle reparation processes and cell signaling pathways by activating endothelial cells, macrophages, and platelets. Infiltrations adjacent to the site of injury must be carried out systematically, for instance injury/stump, proximal-stump, distal-fascia, or deep and proximal interfascicular zone (Fig. 28.5).

7. Finally, ice is applied to the infiltration area for about 10 min.

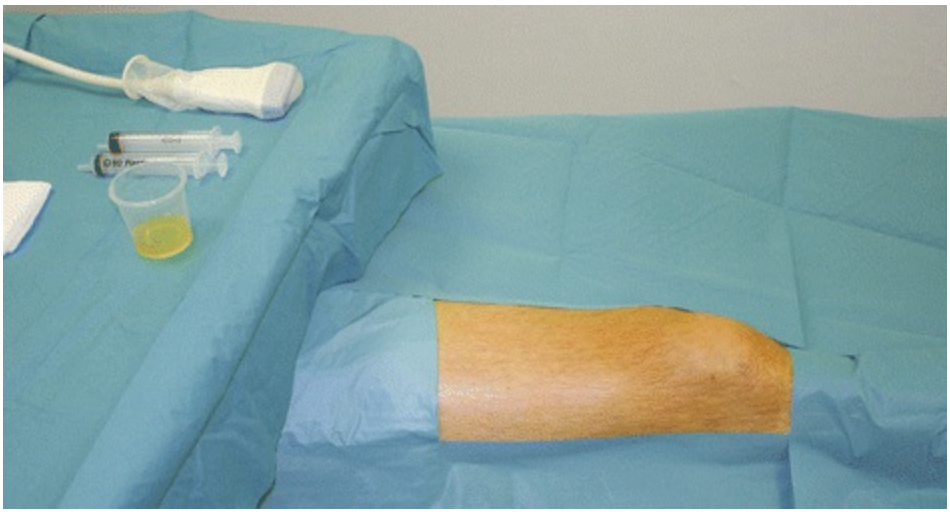

Fig. 28.2 Preparation of the sterile field required to perform ultrasound control, evacuation of hematoma if it exists, and infiltration of PRGF®-Endoret®

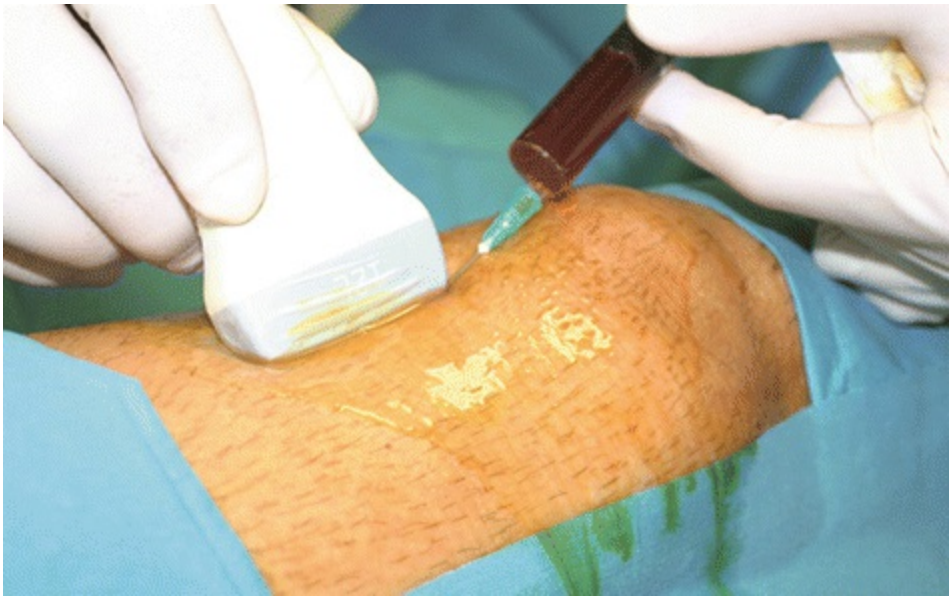

Fig. 28.3 The damaged regions are identified by ultrasound and the hematoma, if present, is then punctured and evacuated

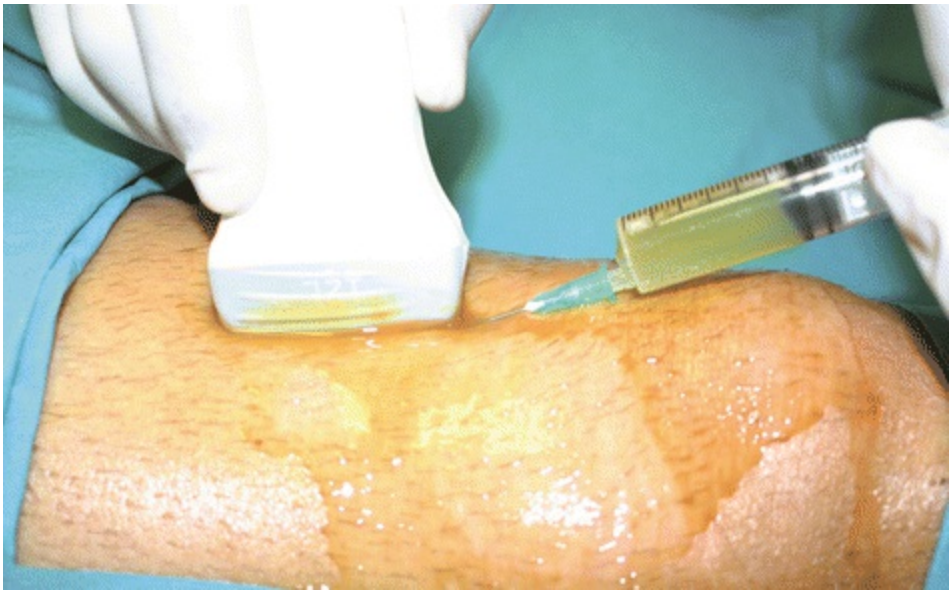

Fig. 28.4 PRGF Endoret infiltration at the site injury and into surrounding (interfascicular and interfibrillar) areas with correct orientation of the needle

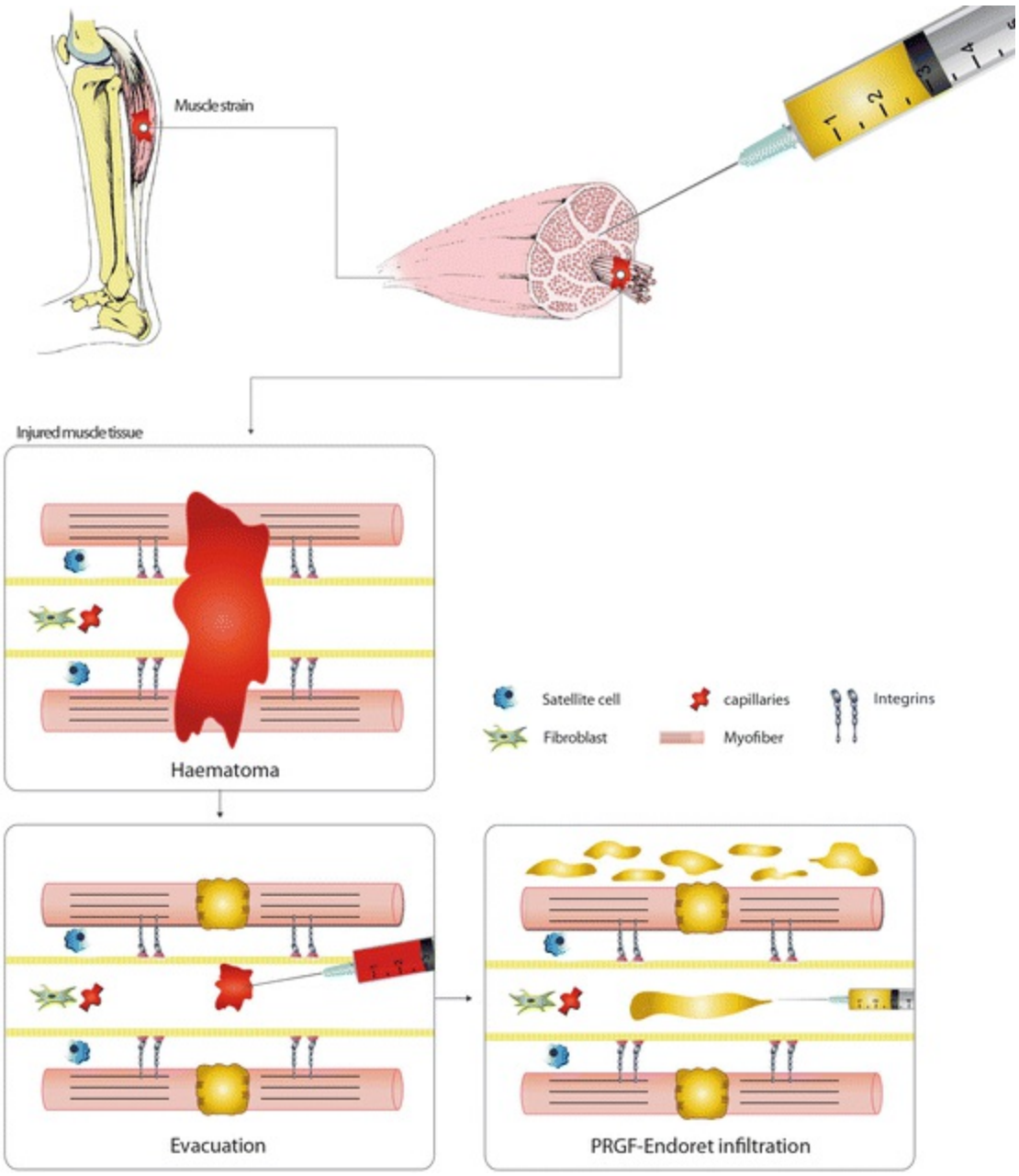

Fig. 28.5 A summary of the most important phases during percutaneous intramuscular infiltration of liquid activated PRGF®-Endoret®

Both clinical and ultrasound monitoring are performed weekly during patient follow-up to evaluate the potential need for more infiltrations. This decision is based on ultrasound images and any pain that the patient presents during this period. One or two sequential infiltrations (on a weekly basis) are usually sufficient, and more than three injections are not normally required. In addition, physiotherapy and rehabilitation treatment are mandatory since the limb has to be mobilized in an early and progressive manner. The mechanical stimulus leads to proper recovery of these patients, since it acts in a synergic ways with the biological effects of PRGF-Endoret [23, 24]. It does not, however, replace continued rehabilitation, but simply shortens the functional recovery

times and stages. Complications such as seromas, cysts, or muscle fibrosis, have to be approached based on the same principles used in acute ruptures.

28.6 Conclusion

Despite the considerable evidence indicating that growth factors and fibrin matrix are instrumental in the muscle repair and regeneration process, there is a gap between basic science and clinical assessment in the treatment of muscle injuries. This gap should be bridged by performing clinical trials to evaluate the efficacy of PRGF for shortening the healing process and avoiding relapse.

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29. Advanced Magnetic Resonance Imaging of Muscles in Sports Medicine

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Abstract
MRI is the method of choice to confirm and evaluate the extent and severity of muscle injuries in acute and chronic situations. However, sports medicine physicians frequently face in their clinical practice cases of persistent clinical symptoms and/or persistent loss of function after muscle injury, with routine imaging including MRI that is unremarkable. Moreover, function and composition of muscle tissue cannot be assessed with routine MRI. Advanced MRI techniques for muscle assessment are available and may provide information on composition, microstructure, and function of muscles or groups of muscles. To date, these techniques have been applied mainly in clinical research regarding other muscle affections such as muscular dystrophy and other myopathies. Some of these techniques are widely available in clinical scanners (T2 mapping, proton MR spectroscopy, fat-water separation techniques), whereas others require special software and hardware and are not widely available in clinical practice (diffusion-tensor imaging (DTI), phosphorus MR spectroscopy, MR elastography). In this chapter, we will discuss these advanced MRI techniques for muscle assessment and their potential applications in sports medicine.

29.1 Introduction

Magnetic resonance imaging (MRI) is considered the imaging method of reference to assess the morphology of muscles in athletes due to its ability to visualize soft tissues with excellent contrast and to provide high-resolution and multiplanar assessment of muscles, especially when traumatic lesions are clinically suspected [1, 2]. MRI is the method of choice to confirm and evaluate the extent and severity of muscle injuries [3]. Furthermore, some of the MRI morphologic features of acute muscle injury, such as the extent of injuries and the differentiation between edema and tears have proved to be related to important clinical features such as time to recovery and risk of re-injury [3–16]. Also, with chronic injuries, MRI may be useful for demonstrating scar tissue formation at the site of injury or involving other sites in the muscles, as well as focal or diffuse fat atrophy in the injured muscles, some of which may correlate with persistent clinical symptoms and loss of function. In cases of chronic injury with scar tissue formation, dynamic ultrasound assessment can be used to ensure that there is no remaining rupture of muscle fibers at the site of injury.

Sports medicine physicians, however, frequently face in their clinical practice cases of persistent clinical symptoms and/or persistent loss of function after muscle injury, with routine imaging including MRI that is unremarkable. Furthermore, for some athletes undergoing joint surgery after injury, rehabilitation and muscle strengthening of the limb in a short follow-up is not always successful and persistent loss of function can occur. Routine imaging of muscles in these cases is almost always unremarkable. The function and composition of muscle tissue simply cannot be assessed with routine MRI.

Fortunately, advanced MRI techniques for muscle assessment are available that can provide information on composition, microstructure, and function of muscles or groups of muscles. To date, these techniques have been applied mainly in clinical research regarding other muscle affections such as muscular dystrophy and other myopathies [17–25]. Some of these techniques are widely available in clinical scanners (T2 mapping, proton MR spectroscopy, fat-water separation techniques), whereas others require special software and hardware to be implemented and are not widely available in clinical practice (diffusion-tensor imaging (DTI), phosphorus MR spectroscopy, MR elastography).

These techniques have the potential to assess function, including assessment of recruitment of muscles for a given activity, as well as biological and metabolic function; composition, including assessment of early (microscopic) fat atrophy; microstructure, by evaluating the direction of muscle fibers as in tractography models after applying DTI; and elasticity (MR elastography). Below we will discuss the various advanced MRI techniques for muscle assessment and their potential applications in sports medicine.

29.2 T2 Mapping ("Functional MRI")

T2 relaxation time mapping (T2 mapping) measures the time constant of decay of the nuclear MR signal. A multi-echo spin-echo technique is used to measure T2 values. The signal intensity of each pixel is fitted to one or more decay exponentials, depending on whether more than one distribution of T2 is thought to be present within the sample. Whereas routine MRI allows a subjective (qualitative) assessment of T2 values of muscles, quantitative T2 mapping provides objective data by generating either a color or a gray-scale map representing the variations in relaxation time within tissues (Fig. 29.1).

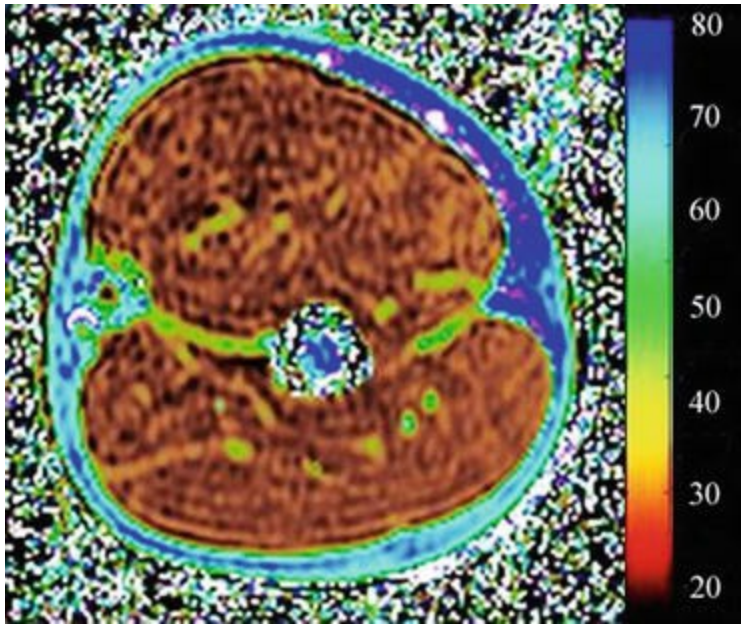


Fig. 29.1 Axial T2 color map of the middle arm in an asymptomatic volunteer. When matched to the color scale of T2 values, note that the values of the flexor and extensor muscles' fibers are lower than those of the surrounding soft-tissues containing fat, such as the superficial and deep aponeurosis, the subcutaneous fat, and the bone marrow

It is accepted that, in different tissues the rate of interactions between water and macromolecules will vary; when interactions increase, T2 decreases, when interactions decrease T2 increases. T2 mapping has been widely applied in clinical research, mainly in the assessment of hyaline articular cartilage [26–30]. In muscles, physiologic or pathologic changes in the interactions between water and macromolecules will affect the T2 relaxation times (alterations of water binding). Furthermore, the distribution of water in the intracellular and extracellular compartments of muscles directly contributes with different components to the T2 relaxation times with this technique [17]. About 80 % of the water signal from muscles is from intracellular water (contributing to a fast relaxation time – 20–40 ms), whereas around 10 % of the water signal is from extracellular water (contributing to a slow relaxation time – 150–400 ms) [31].

It has been demonstrated that the T2 relaxation time of skeletal muscles increases

during and after exercise [32–40]. The underlying mechanism for the increase is not fully understood, and probably multiple factors are involved. During exercise, contraction of muscle fibers requires energy consumption, leading to an increase in intracellular osmolites (sodium, phosphate, and lactate) responsible for an increase in intracellular osmolarity. This will cause water influx into the muscles with an increase in intracellular water, leading to an increase in T2 relaxation times [41]. Other factors are muscle perfusion [42] and a decrease in pH [43, 44]. Even though the mechanism involved in exercise-induced changes of muscles is not fully understood, the differences in T2 relaxation times between metabolically stressed muscles and less active muscles have practical applications. For specific exercises in the upper and lower limbs, as well as in the trunk, it is possible to isolate specific muscles or groups of muscles that activate during and after exercise [32, 35–38]. Sometimes, even the degree of activation (function) may be estimated as well [34]. In the literature, T2 mapping of exercised muscles is often referred as "functional MRI" or "fMRI" of muscles. Two decades ago a linear relationship between the mean muscular force produced during exercise and the T2 values of calf muscles was demonstrated; the increase in mean force correlated with the increase in T2 values [39]. The same study also tested the effect of venous occlusion on T2 values of muscles, but could not demonstrate a direct relationship between these two factors. Another study [45] showed that specific muscles around the neck demonstrated activation after specific head movements by showing an increase in T2 values, and the absolute and relative cross-sectional area of muscle exhibiting an increase in T2 values was greater when the exercise load applied was greater, providing another demonstration of a linear relationship between muscular activity/load and T2 values. T2 mapping was also shown to be correlated with quantitative measurements of muscle activity. A linear relationship between exercise intensity and T2 values was demonstrated [35], in a group of seven volunteers at rest after a series of 100 repetitions of 90-degree hip and knee flexion. Then they repeated the exercises but with a one kilogram load on the right ankle, and then they were scanned again at rest. The increase in T2 values of the right psoas muscle followed the increase in the exercise load; T2 values after the second series were greater than after the first series. A more recent study [37] assessed the correlation between total power output of muscles of the thigh during two, five, and ten sets of sprint cycling and increased T2 values of these muscles. A moderate and significant correlation was found between total power output and a linear increase in T2 values of the quadriceps. Although the thigh muscles were not uniformly activated after exercising, a linear increase in T2 values was demonstrated for the majority of them. Adams et al. [46] evaluated muscle activity in seven subjects before and after they performed five sets of concentric or eccentric arm curls with each of four resistances accounting for 40, 60, 80, and 100 % of their 10 repetition maximum for concentric curls. They did so by scanning the middle of the arm on MRI using T2 mapping, as well as by collecting surface electromyography signals

from the biceps brachii and the long head of the triceps brachii muscles. Both concentric and eccentric contractions lead to an increase in both T2 and integrated electromyography, which were strongly correlated. The relationship between T2 values and muscle work rate has also been demonstrated in previous studies. Jenner et al. [43] reported the results from a study evaluating the signal intensity of the anterior tibialis muscle on MRI during progressive ankle dorsiflexion exercise, and concluded that signal intensity was linearly correlated to exercise intensity, but not with the total work performed.

One must be careful when affirming that T2 changes may determine precisely the amount of work performed by a given muscle, which would be true if a linear relationship exists between T2 values and muscle work. By using a wide range of muscle work intensities, Cheng et al. [47] demonstrated that the increases observed in T2 values during increases in exercise intensity were nonlinear, especially at low work intensities. Fleckenstein et al. [48] showed that increases in the integrated force vs. time curve and in T2 values occurred during progressive maximal voluntary handgrip contractions, and repeated analyses of variance of such increases were statistically significant. However, they found that the increase in T2 values was likely represented by a hyperbolic curve as progressively longer bouts of contractions were applied – there was a limit in the increase of T2 – and suggested the existence of a limit in the amount of water that muscles absorb from the vasculature during exertion. Furthermore, by studying the T2 values in each pixel of muscles Prior et al. [32] showed that T2 did not vary substantially among different intensities of exercise.

There are some considerations that must be taken into account regarding T2 values and muscle activity. There is evidence that increases in T2 values in muscles during or just after exercising are related to muscle activity, and such changes have the potential to show which muscles are being recruited with specific exercises (Figs. 29.2, 29.3 and 29.4). The relationship between T2 values and muscle activity/work is not always linear. Several factors are probably involved in these relationships; the specific muscle being exercised, the intensity of the exercise/load, and the physical training level of the subjects. There is no consensus about how, when or where to apply T2 mapping to determine the work performed by a specific muscle or group of muscles.

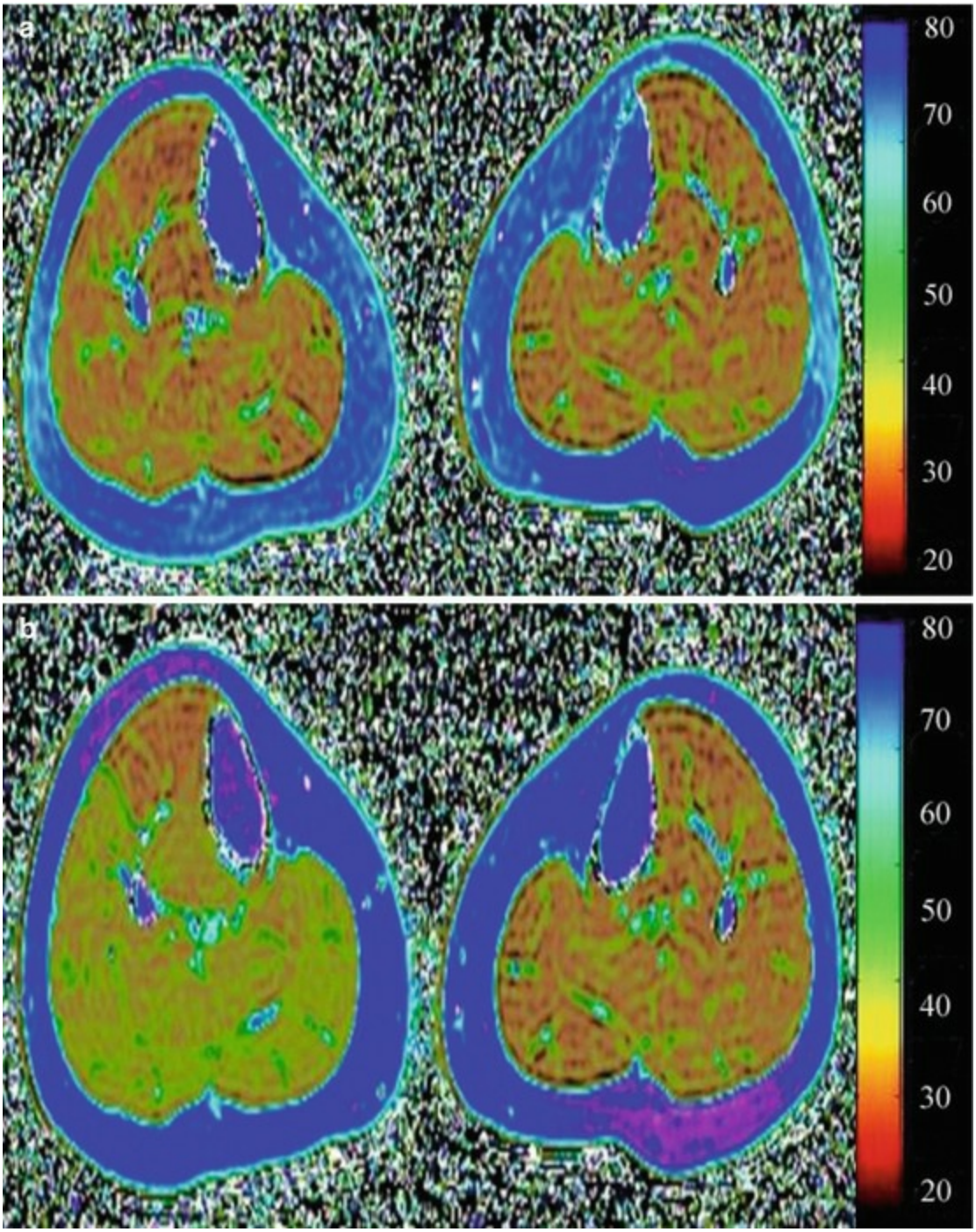


Fig. 29.2 (**a**) Axial T2 color map of themidsection of both legs in a young male volunteer before exercising. Note that the distribution of T2 values in the muscles (*colors*) are mainly symmetrical when comparing both legs. (**b**) Axial T2 color map in the same subject after four sessions of right (unilateral) plantar flexion. Note that the T2 values of the triceps surae muscle of the right leg are higher in comparison with the non-exercised contralateral leg. T2 values in other muscles of the right leg are also elevated but to a lesser degree

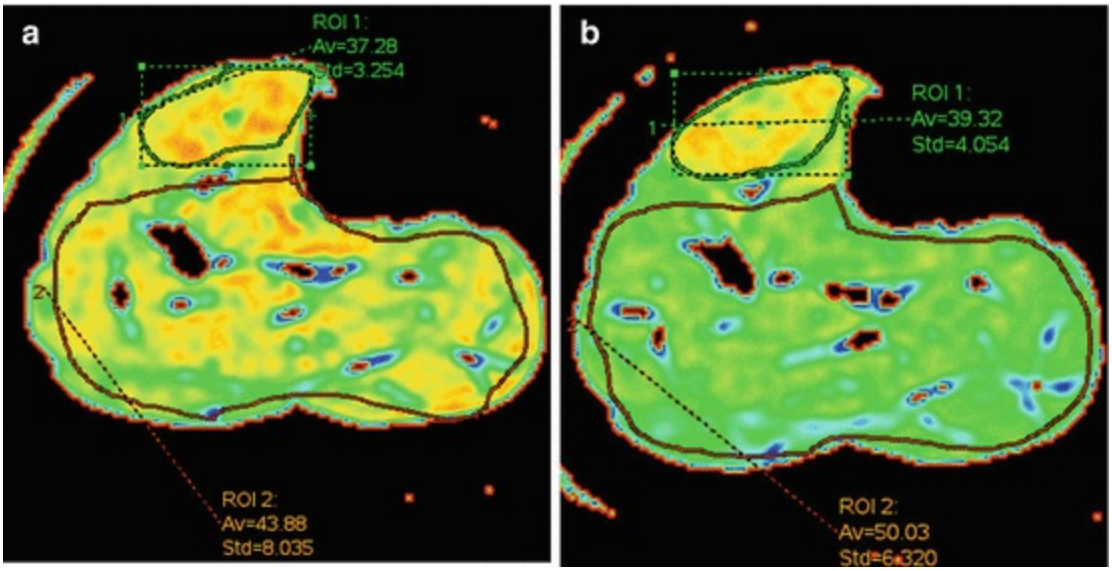
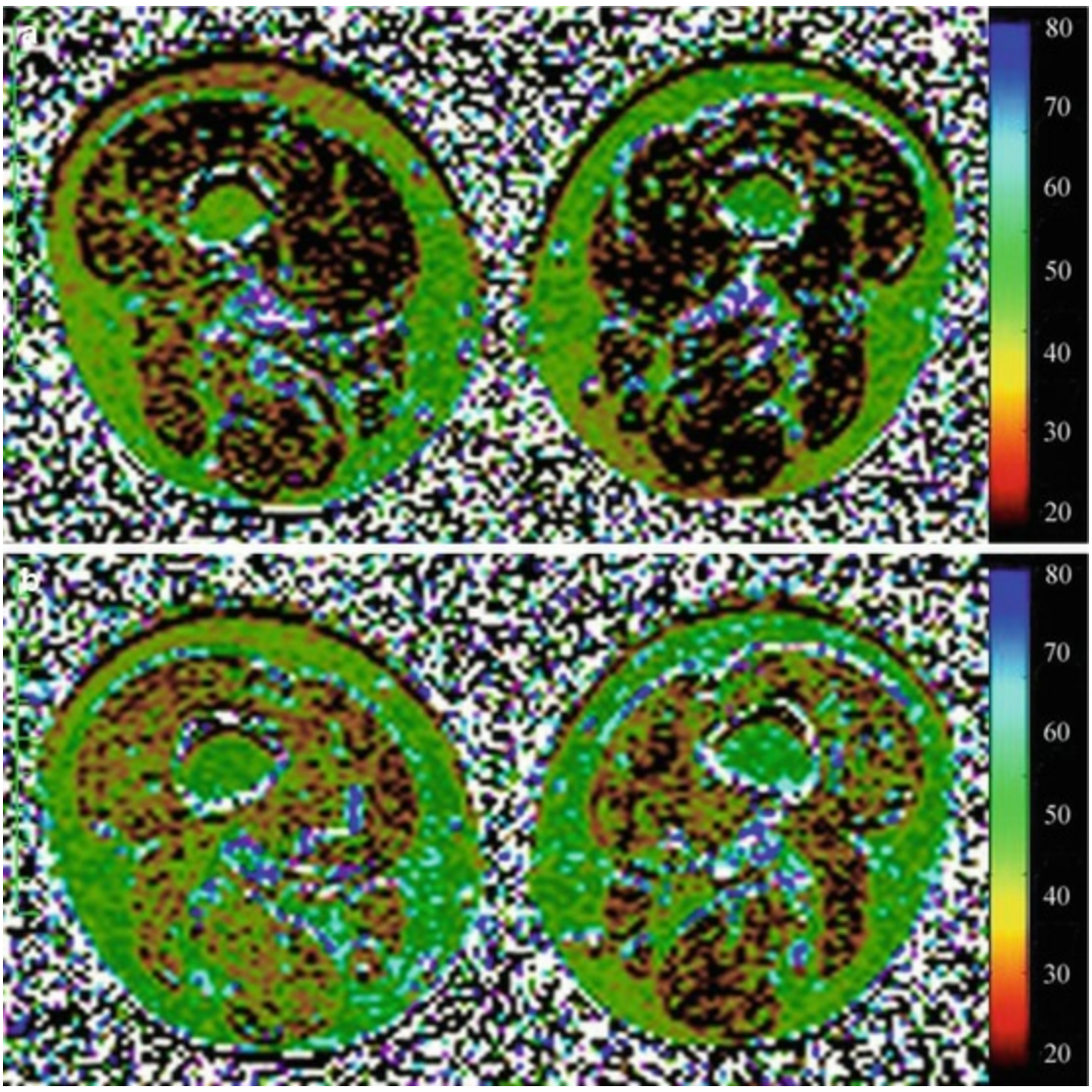


Fig. 29.3 Quantitative assessment of T2 values of muscles of the mid right leg in a young female volunteer before and after six sessions of right plantar flexion. The average of T2 values in the posterior (calf) muscles increased from pre-exercise (**a**; 43.88 ms ± 8.03) to post-exercise (**b**; 50.03 ms ± 6.32). Note also the significant change in the color-coded T2 maps of the calf muscles between A and B. There was only a slight increase in T2 values (and change in color-coded) of the extensor muscles from **a** to **b**.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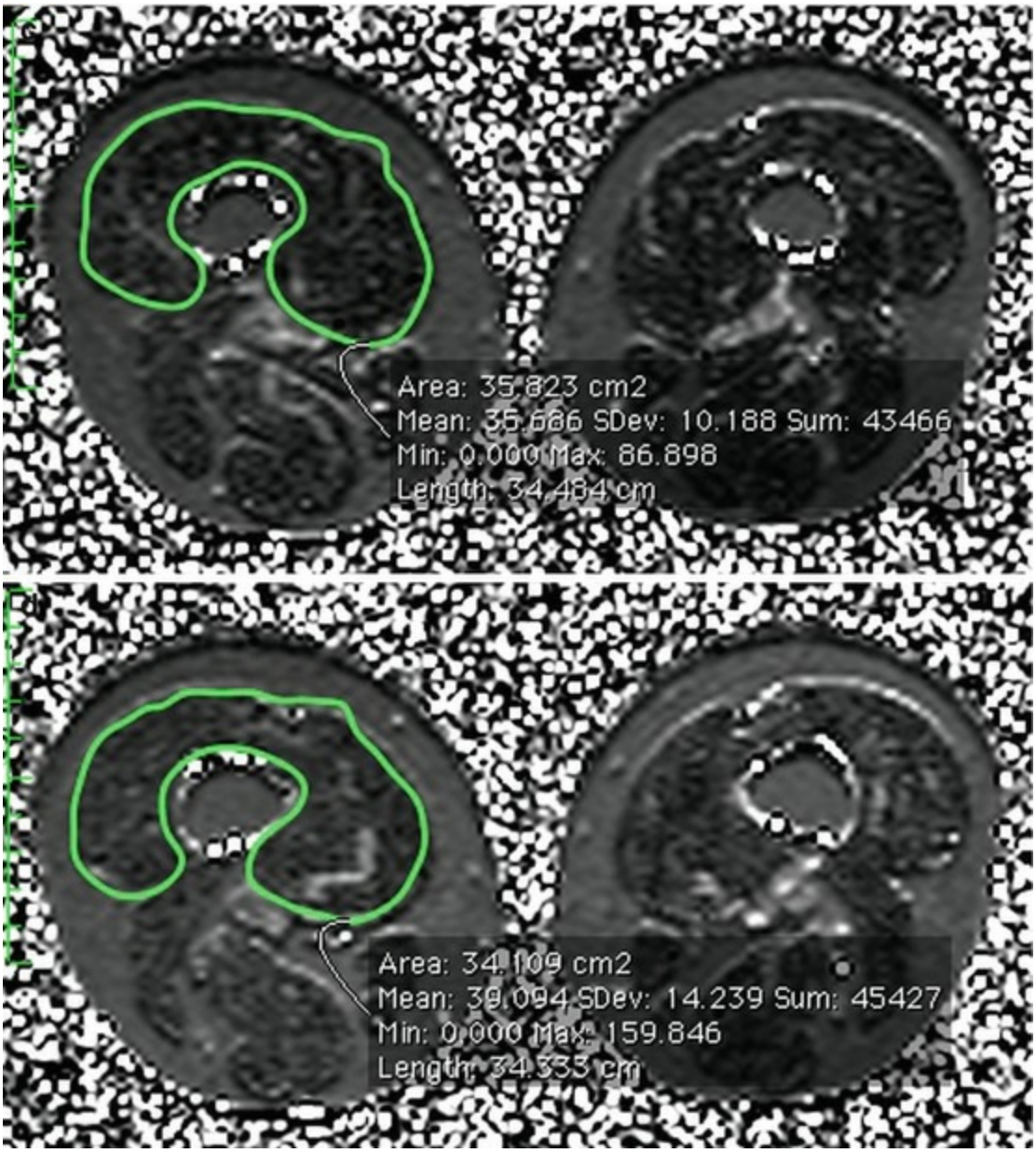
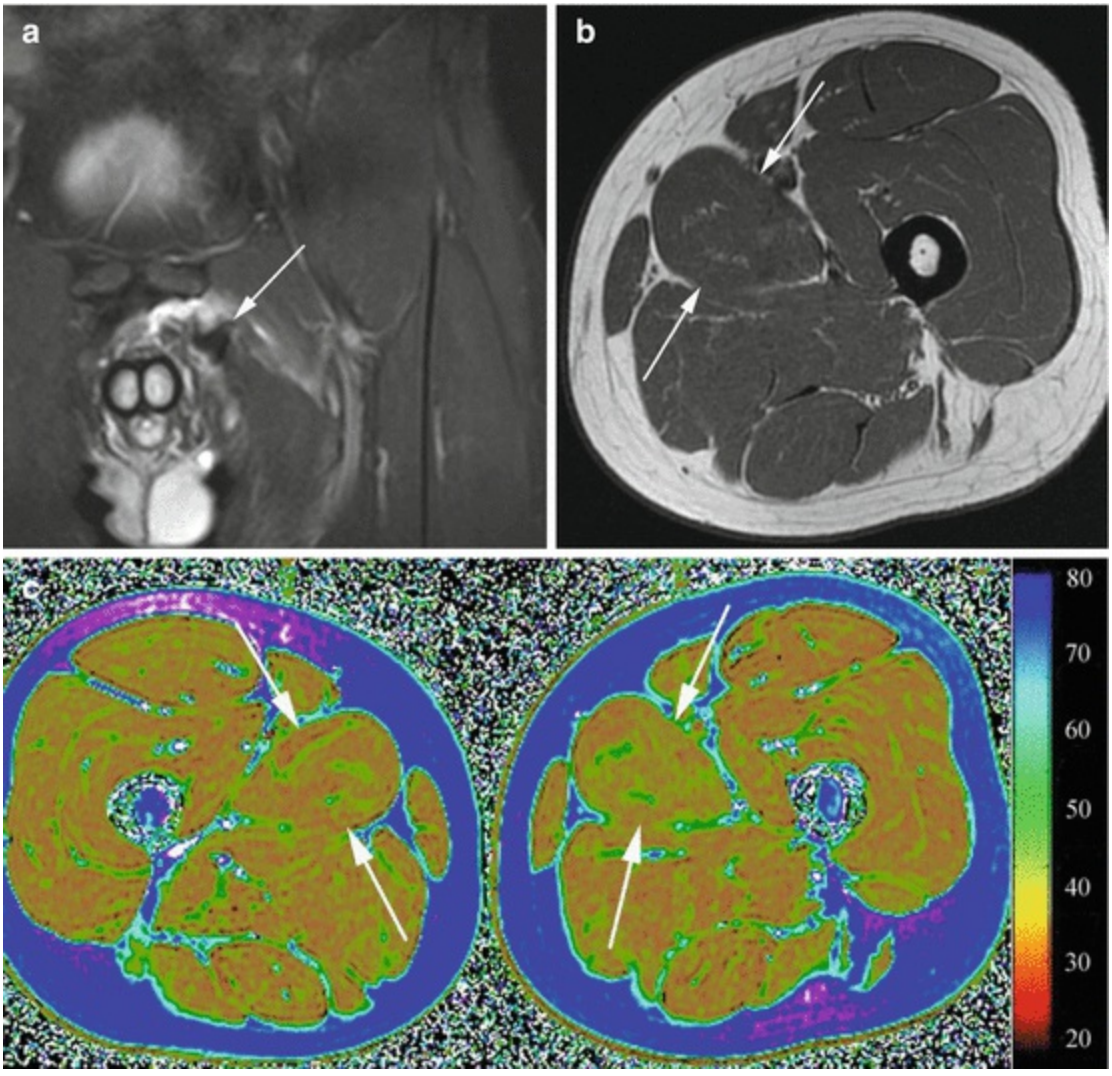


Fig. 29.4 Qualitative and quantitative assessment of T2 values of muscles of both thighs in a healthy female volunteer before and after squat exercising. Qualitative assessment using T2 color-coded maps of both thighs shows a diffuse increase in T2 values of muscles after a series of squats (**b**) when compared to T2 values before exercise (**a**). Quantitative assessment of the right quadriceps muscle showed an increase of the mean T2 values after squating (**d**; 39.1 ms ± 14.2) when compared to T2 values before exercising (**c**; 35.7 ms ± 10.2) (Courtesy of Augusto P. Baffa and Marcello H. Nogueira-Barbosa, University of São Paulo at Ribeirão Preto, Brazil)

The literature, however, does show that there is a much potential for T2 mapping in sports medicine. Quantitative T2 data may provide useful information about the capacity of a muscle (or a group of muscles) to be activated by a specific exercise aimed at that muscle. This may be true in cases of chronic muscle strain/rupture in which the muscle is still functionally impaired. Even without exercising the affected muscle, quantitative T2 mapping may be useful in demonstrating early fat atrophy when routine MRI does not

exhibit unequivocal findings of atrophy, since an increase in the fat content of muscles will increase their T2 (Fig. 29.5 and 29.6). Also, in post-operative joints for any reason, there are cases in which, a given muscle or a group of muscles of the limb affected will not recover its initial function during the rehabilitation and muscle strengthening process. In these cases routine imaging is often unremarkable, and the application of T2 mapping before and after exercise may bring useful quantitative information. Furthermore, the application of T2 mapping as a measure of muscle recruitment would be very useful in the follow-up of rehabilitation and muscle strengthening programs in athletes [33], as it could show roughly the efficacy of specific exercises in different regions of limbs and trunk, by demonstrating which muscles are being recruited. Finally, this could be a useful tool in the assessment of the effects of many orthoses available in many sports fields. Objective T2 quantitative data may be obtained from muscles by exercising a region where a specific orthosis is being applied for training and competition. All these possible applications of T2 mapping of muscles should be tested in future prospective and controlled studies.



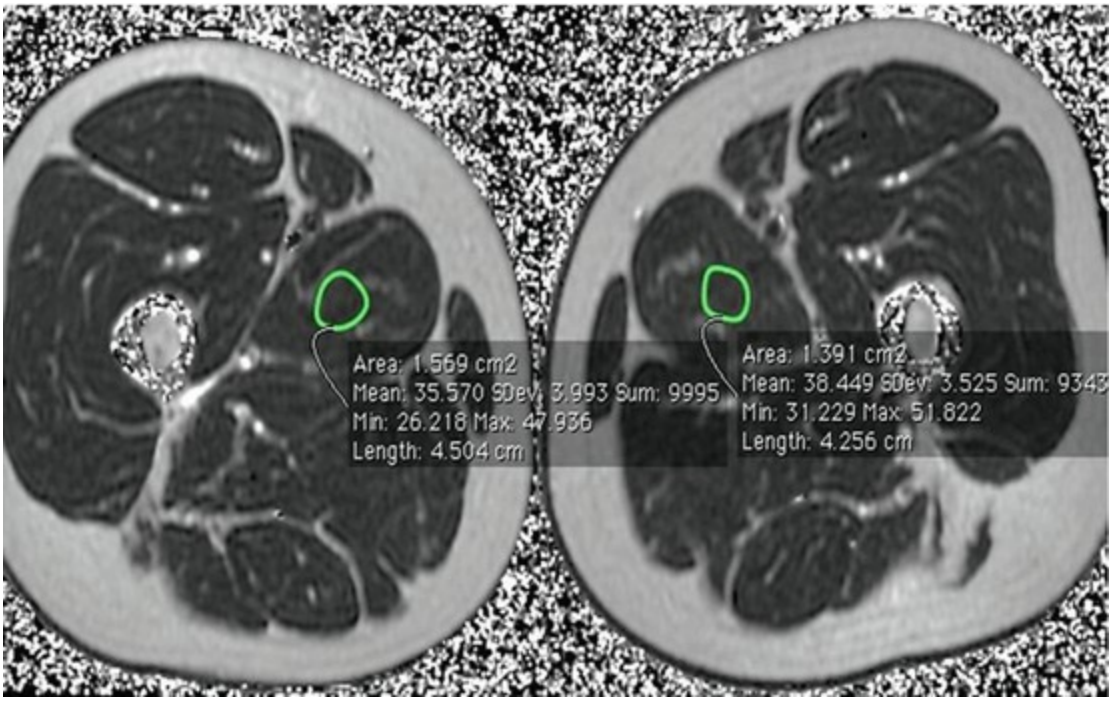


Fig. 29.5 Recreational 36-year-old soccer player with an untreated complete avulsion of the left adductor longus tendon at its pubic insertion 2 months prior, demonstrated on coronal fat-suppressed T2-weighted MRI (**a**; *arrow*). The appearance of the left adductor muscle on the axial T1-weighted MRI of the left thigh is unremarkable (**b**, *arrows*). Axial color T2 mapping of both thighs without exercise demonstrates multiple foci of increased T2 values within the left adductor muscle compared with the right side (**c**, *arrows*), possibly indicating early fat atrophy of the left adductor longus muscle. Quantitative assessment of T2 values (**d**) of both adductor muscles shows a higher T2 value on the left side than on the right (38.45 ms ± 3.52 vs. 35.57 ms ± 3.99)

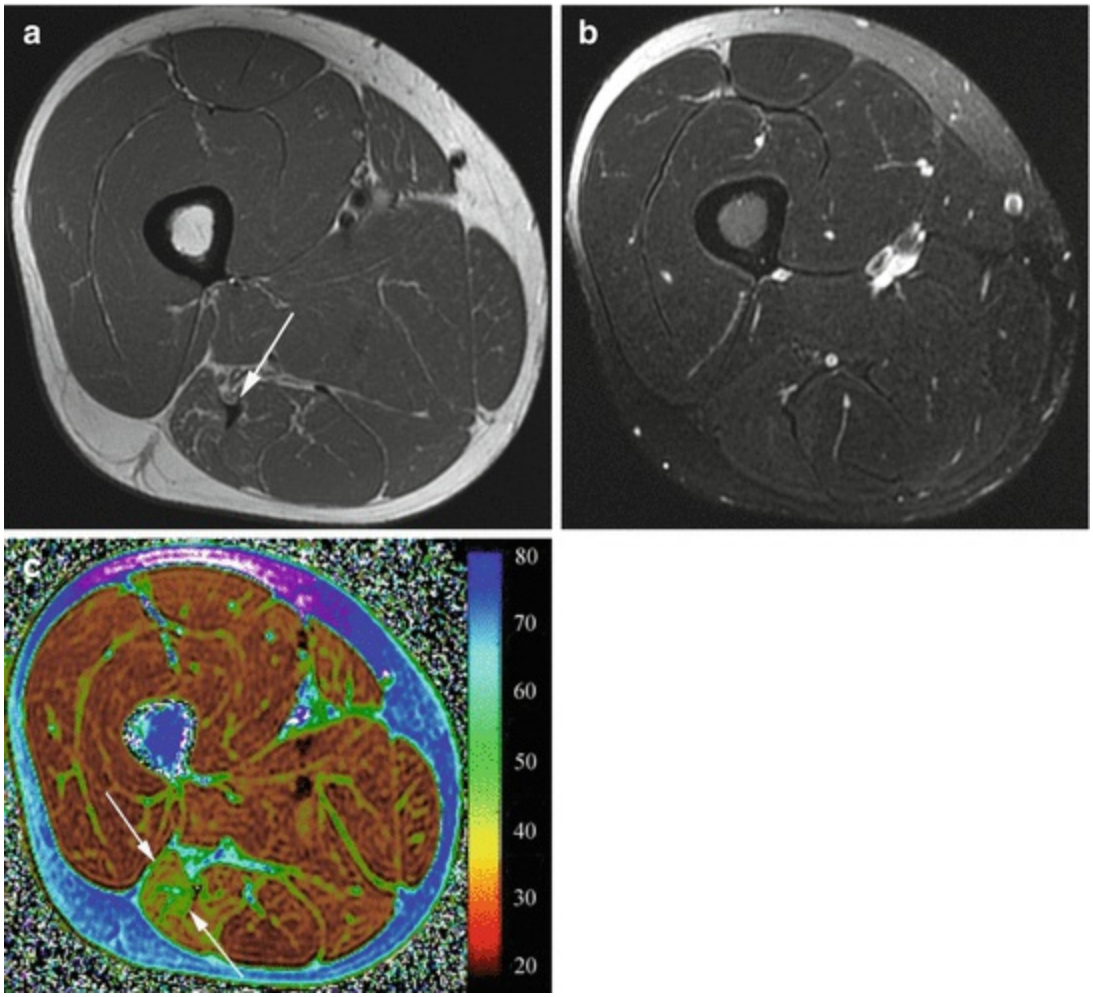


Fig. 29.6 Professional 29-year-old soccer player with a history of a strain of the long head of biceps femoris (LHBF) muscle 10 weeks prior. (**a**) Axial T1-weighed MRI shows mild chronic changes with thickening of the LHBF tendon at the proximal myotendinous junction (*arrow*), without significant changes in the adjacent muscle bulk. (**b**) Axial fat-suppressed T2-weighted MRI does not demonstrate signs of edema in the LHBF muscle. (**c**) Axial color-coded T2 map of the thigh demonstrates an increase in T2 values of the LHBF muscle (*green color*, *arrows*) when compared to other muscles in the field of view, which may be related to early fat atrophy of the muscle

29.3 Diffusion Tensor Imaging and Tractography

Diffusion tensor imaging (DTI) is an MRI technique widely applied in neuroradiology for the assessment of the orientation and integrity of white matter tracts in the brain and spinal cord [49, 50]. This technique is able to assess the anisotropy of water diffusion, and fiber tracking should be feasible using DTI, on the principle that water diffusion will be greater along the orientation of the white matter fibers than in another direction. In each imaging voxel a diffusion tensor will be reconstructed from multiple diffusion-weighted MRIs with at least six independent diffusion-encoding directions (Fig. 29.7). Once the tensor is estimated, the eigenvalues and eigenvectors provide data regarding the effective diffusivity along the orthogonal directions [51, 52]. DTI enables three-

dimensional assessment and visualization of the fiber tracts; it is known as “fiber tractography”. Because of the highly anisotropic nature of muscle tissues, DTI may also be applied in skeletal muscles [17, 51–58] to assess the integrity and the orientation of muscle fibers. Two important parameters may be calculated from the generated tensor images: the fractional anisotropy, representing a measure of how directional the water diffusion is, and the mean diffusivity, which represents the average length of water diffusion along the selected directions. The fractional anisotropy is by far the most used tensor value (Fig. 29.8). Further, information on water diffusivity may be provided when assessing the apparent diffusion coefficient of tissue water, which reflects cellular membrane integrity and microcirculation/perfusion.

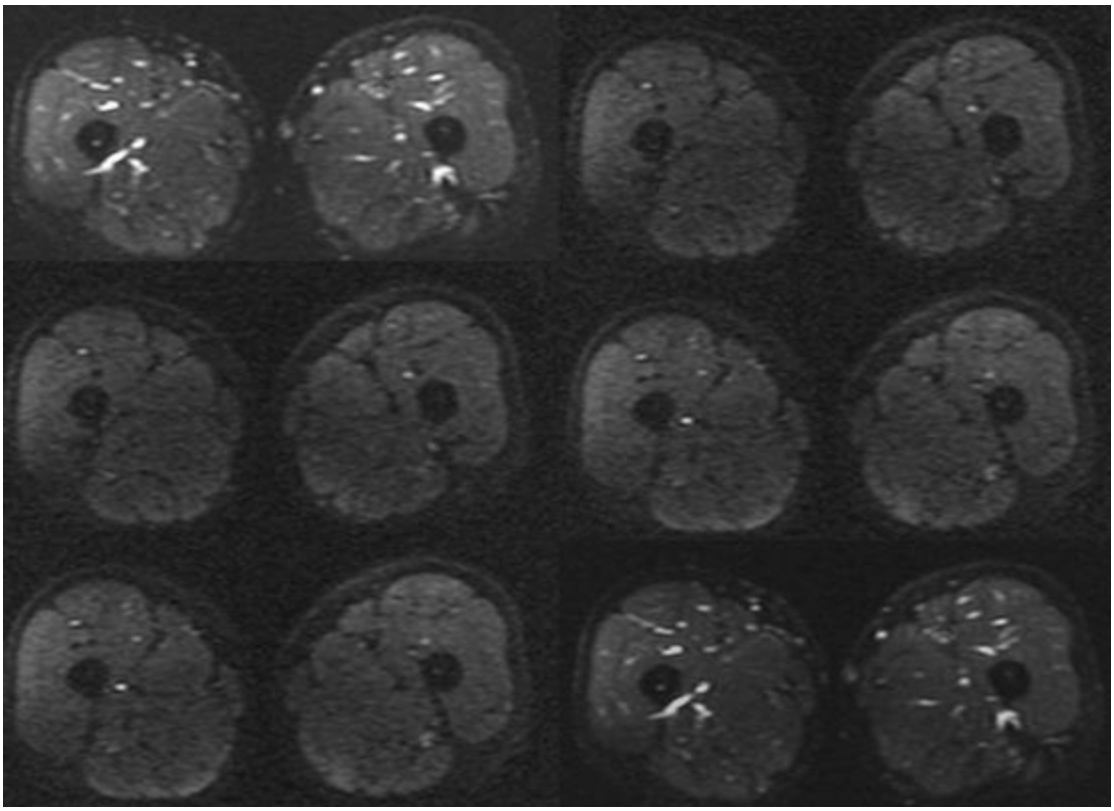


Fig. 29.7 To cover different possible directions of water molecules’ diffusion along the different orientations of fibers, multiple diffusion-weighted acquisitions (six in this example) using different and independent diffusion-encoding directions are applied, from which a diffusion tensor image will be reconstructed

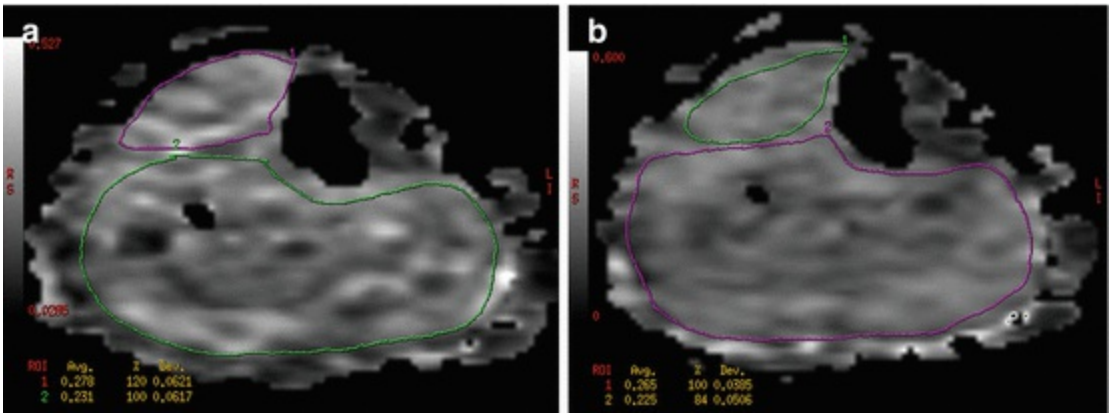


Fig. 29.8 Assessment of fractional anisotropy in a young female volunteer without muscle injury. Assessment before (**a**) and after (**b**) six sessions of plantar flexion did not show significant changes in fractional anisotropy values (how directional the water diffusivity is) in the calf muscles (0.231 ± 0.06 before and 0.225 ± 0.05 after)

Thus, DTI and fiber tractography seem to represent a powerful tool in the assessment of the skeletal muscle microstructure in different scenarios. Previous works attempted to demonstrate its usefulness in muscle microstructure assessment. By comparing patients with injured calf muscles with subjects without calf muscle injury, Zaraiskaya et al. [51] demonstrated that fractional anisotropy values were reduced in all patients when compared to controls, while apparent diffusion coefficient values were consistently higher in patients, suggesting that DTI may be useful in assessing fiber integrity and identifying injured or disrupted muscle tissue. Sinha et al. [58] assessed the feasibility of in vivo fiber tracking of the calf muscle fibers with DTI. The orientation angles of fibers in the different muscles assessed in five volunteers were mostly equivalent to those demonstrated in previous spectroscopic studies, supporting the ability of DTI in tracking muscle fiber direction. The usefulness of DTI assessment of muscles was further demonstrated in a study that aimed to create biomechanical models of the quadriceps muscles by comparing patients with chronic lateral patellar dislocation with healthy volunteers. After tracking muscle fibers from the vastus lateralis oblique and vastus medialis oblique muscles and determining the pennation angles and the orientation of resultant force vectors, the authors found that the patients had more laterally directed predicted resultant force vectors that the control group [54]. Froeling et al. [52] could reproduce in detail the complex architecture of muscle fibers of the human forearm in five healthy volunteers using DTI and applying three-dimensional fiber tractography, including a comparison between the segmented fiber tracts obtained with DTI and animations from 20 photographs taken of each isolated muscle after cadaver dissection. The potential of DTI for demonstrating muscle injury on a microscopic level (and thus assessing microstructure of muscles) was seen in a study that assessed the correlations between DTI parameters and histologic changes (Z-band disruption) before and after 300 eccentric actions of the knee extensors on an isokinetic dynamometer [53]. The authors showed that the histologic indices of damage

coincided with changes in DTI parameters (fractional anisotropy and apparent diffusion coefficient), and Z-band streaming quantified per fiber was significantly correlated with fractional anisotropy. More recently the ability of DTI to measure maximum muscle power, which is an indirect measure of fiber type distribution, was assessed in 11 soleus muscles of healthy volunteers. The study showed significant correlations between soleus muscle power and some DTI parameters such as fractional anisotropy and radial diffusivity [55].

Again here, although only few studies have explored DTI in the evaluation of muscle structure and function, we may say there is great potential for this technique in sports medicine research. One example would be where low-grade acute muscle strain is seen, without significant macroscopic fiber disruption on routine clinical imaging (ultrasound or MRI), but recovery is taking longer than expected. By assessing the microstructure of muscles (fiber integrity and orientation), DTI may be able to show that damage on a microscopic level is greater in athletes with longer recovery times than in those with shorter recovery times, even though macroscopically, in routine imaging, the severity and extent of findings do not differ between the two groups. Furthermore, it may also be useful in chronic injuries, when muscle function is not improving after rehabilitation and muscle strengthening although macroscopically, on routine imaging, the injury seems to be healed. DTI may help to identify persisting alterations in fiber orientation and integrity in these cases (Fig. 29.9). Finally, a few studies have shown that some DTI parameters are correlated with microscopic muscle disruption and with muscle power, although more studies are needed to confirm these correlations. These findings suggest the potential of this technique in tailoring rehabilitation and muscle strengthening programs for athletes, as well as in monitoring response to treatment.

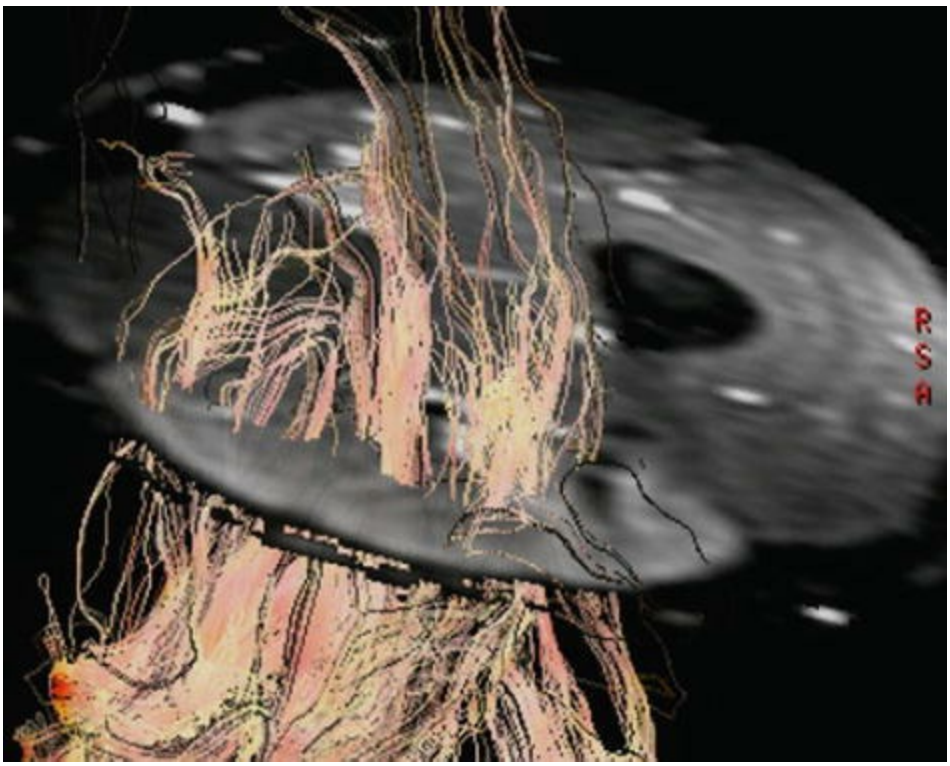


Fig. 29.9 An example of fiber tracking (tractography) after applying DTI in the mid thigh

29.4 MR Spectroscopy

Unlike most imaging techniques, MR spectroscopy (MRS) provides information regarding the biochemical composition of tissues but cannot assess anatomic structure. During MRS acquisition, the signals are processed by Fourier transformation into plots of frequency of nuclear rotation versus signal intensity. The chemical shift (variation in frequency) of different metabolites will be expressed in parts per million of the field strength (represented by the x-axis) and the (peak) signal intensity of different metabolites will be represented by the y-axis. The most widely used version, 1H-MRS, can study muscle composition by demonstrating resonances from fat, water, creatine, trimethylammonium-containing compounds, and many other metabolites [17, 59]. A few recent studies showed the potential of 1H-MRS for detecting and monitoring several metabolites, including creatine and lactate, and their relationships with different phases and intensity of muscle exercise [59–62].

Because most of the muscle metabolites relevant in energy transduction contain phosphorus, 31P-MRS is better suited to assess muscular concentrations in vivo and to monitor changes over time [63]. With 31P-MRS, only relevant muscle metabolites are visible in the spectrum of signals acquired: the most signal intensities are from phosphocreatine (PCr), inorganic phosphate (Pi), and the three phosphate groups of adenosine triphosphate (ATP), which are involved in the energy metabolism (Fig. 29.10). Other visible peaks include those from phosphomonoesters and

phosphodiesters. Although adenosine diphosphate (ADP) cannot be directly assessed by this technique, its concentration can be calculated indirectly using the creatine kinase reaction. Furthermore, 31P-MRS has the ability to measure the pH, which can be determined from the chemical shift difference between Pi and PCr. Thus, 31P-MRS can assess the concentrations of relevant metabolites involved in energy transduction and the pH of muscles, and it can be applied in dynamic studies to acquire kinetic data on glycolytic ATP synthesis, oxidative ATP synthesis, as well as intracellular pH regulation. It is a powerful tool for the assessment of muscle metabolism and mitochondrial activity, which can be studied in the sports medicine setting, especially through rest-exercise-recovery protocols [63].

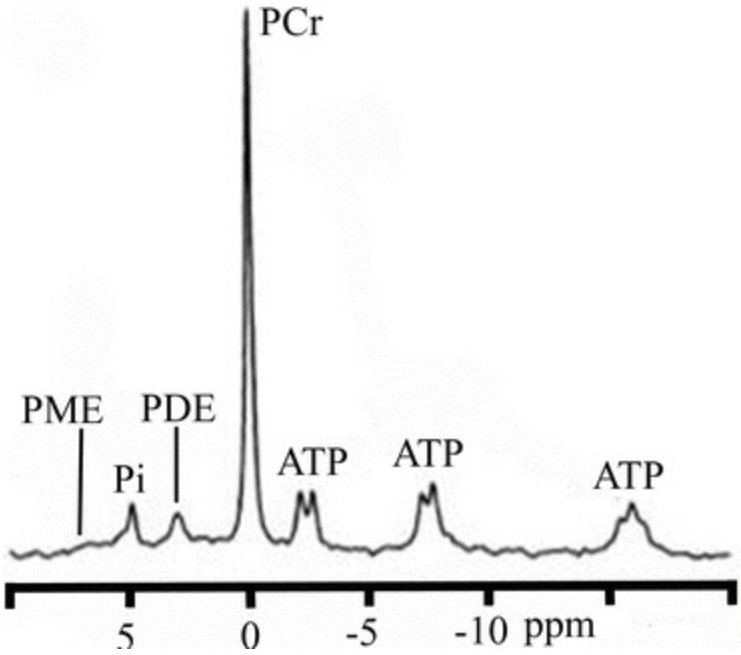


Fig. 29.10 Spectrum of (peak) signals acquired after 31P-MRS acquisition: *PCr* phosphocreatine, *Pi* inorganic phosphate, *ATP* adenosine triphosphate; *PME* phosphomonoesters, *PDE* phosphodiesters (Figure modified from [63])

By knowing the physiologic changes in muscle metabolites and pH during rest, exercise, and recovery after exercise periods, it is possible then to detect metabolic abnormalities in muscles where function and work are not optimal. This approach was used in a study evaluating muscle metabolism and force production in sprint-trained runners, endurance-trained runners, and untrained subjects, using 31P-MRS. The study demonstrated important differences in force production, aerobic and anaerobic muscle metabolism in these three distinct groups, showing different patterns of PCr breakdown and recovery [64]. A recent study evaluating the same groups of subjects using 31P-MRS also showed important differences among endurance and sprint athletes and untrained subjects by assessing PCr muscle metabolism [65]. 31P-MRS could be also applied to test different training methods aimed at improving muscle metabolism and function in a sports medicine setting. An example of such application was shown in a

study that, by using 31P-MRS to assess PCr recovery, demonstrated the efficacy of short-term high-intensity interval training in increasing the functional oxidative capacity in the quadriceps muscle [66]. The alterations in PCr, Pi, and pH were assessed in the quadriceps muscle in a study of healthy subjects using 31P-MRS during exercise above and below the "critical power", representing the highest constant work rate that could be sustained by the subject without progressive loss of homeostasis (of parameters assessed). The authors could nicely identify that during exercises above the critical power, there was continuous depletion of PCr with Pi and pH changing to values observed at the termination of high-intensity exhaustive exercises (changes related to the fatigue process) [67].

In view of this sort of research, there seems to be much potential for the use of MRS, especially 31P-MRS, in sports medicine. Unfortunately, it is not widely available, and it is both costly and time consuming, which make it difficult to apply in large groups of athletes.

29.5 Other MR Techniques

Dixon MRI was applied in studies aimed at quantifying the amount of fat in the skeletal muscle tissue [17, 68–71], which can be useful for detecting and monitoring of muscle fat-atrophy in pathologies leading to muscle dystrophy. Unlike chemical fat-saturation techniques, Dixon MRI is less susceptible to inhomogeneities of the magnetic field and provides homogeneous fat-suppressed images. Two spin-echo acquisitions are acquired for this technique, the first when the protons from water and fat are in the same phase direction (in-phase), and the second when they are in opposed phases (out-phase). The echo times chosen for both acquisitions are field strength-dependent since they are based on the chemical shift between the precessing water and fat protons. When a given voxel contains both water and fat, the signals from the two acquisitions are different: in the in-phase acquisition, the signal from that voxel will be enhanced; in the out-phase acquisition, the signal in that voxel will drop. The images from both acquisitions can be added or subtracted to create a water-only or a fat-only image [17, 72]. Dixon provides quantitative data on the fat fraction of different tissues, including muscles. Differently from the techniques already discussed in this chapter, Dixon MRI is less useful in providing data on muscle activity or function.

Iterative decomposition of water and fat with echo asymmetry and least-squares estimation, known as IDEAL [73], can also provide a quantitative assessment of muscle fat-atrophy as well as uniform fat suppression in challenging magnetic field environments by applying a three-point water-fat separation, which relies on the use of asymmetrical echoes and least-squares fitting to maximize signal to noise ratio performance (Fig. 29.11).

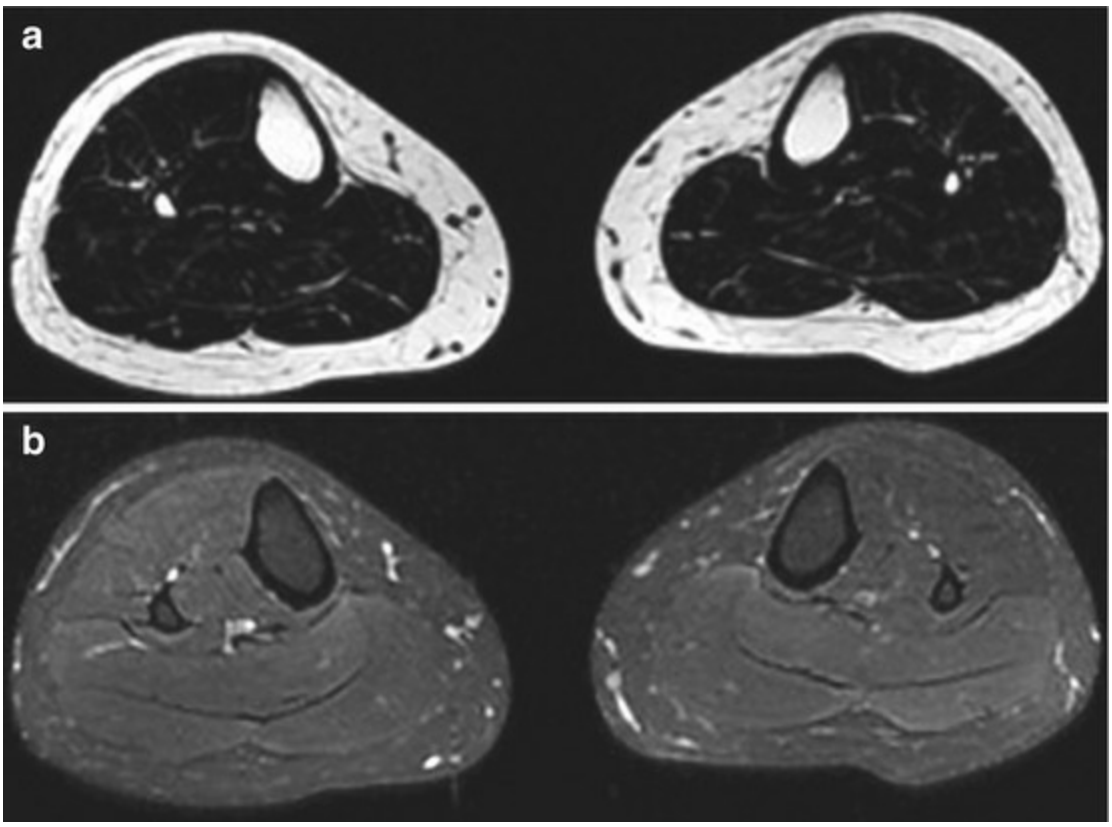


Fig. 29.11 An example of fat-water separation using the IDEAL technique, providing fat (**a**) and water (**b**) MRI of the mid legs.

In sports medicine, Dixon MRI and IDEAL could be applied whenever the assessment of the fat fraction of muscles is useful, especially in cases of the onset of muscle atrophy after traumatic muscle injury or after surgery on the limb. This could be particularly useful in cases where fat atrophy is developing in an early stage of recovery, where the qualitative assessment of muscles using routine MRI sequences will not demonstrate any morphologic change. Care must be taken, however, with assessment of muscle fat atrophy after exercise using Dixon imaging, since it has been shown that the calculated fat fraction after exercise can be underestimated [69].

MR Elastography (MRE) can assess the shear stiffness of soft tissues such as muscles, and thus their elasticity properties. An external mechanical device controlled by the MR pulses first produces continuous and harmonic shear waves in various frequencies; by applying motion-encoding gradients in conventional MR sequences, MRE then encodes the propagating shear waves produced by the external device into the phase of the MR images [74]. Muscular motion occurring in any direction can be depicted while adjusting both position and amplitude of the motion-encoding gradient. Algorithms generate quantitative maps of shear stiffness from the wave. Because the elasticity and mechanical properties of skeletal muscles may vary between muscles with normal physiological function and those with various pathologies, MRE was used in previous studies to identify differences in such properties in various pathologic

conditions in patients compared to healthy volunteers [75, 76]. The potential of this technique in some situations in sports medicine is easily imagined. A recent study of an animal model showed important differences in the stiffness of immobilized muscles compared to muscles of the contra-lateral limb, suggesting potential as a tool to guide rehabilitation [77]. Muscle stiffness was evaluated before and following eccentric exercise of calf muscles in a recent study including eight healthy subjects [78]. The authors showed an increase in stiffness after eccentric exercise with a peak occurring 48 h after exercise. Other recent studies demonstrated the ability of MRE to assess the elasticity of muscles [79, 80]. Whether the elasticity and mechanical properties of muscles as assessed using MRE are related to relevant clinical muscular functional parameters remains to be demonstrated, but it is certainly intriguing and could be very useful in sports medicine.

29.6 Conclusion

Advanced MRI techniques are available for skeletal muscle assessment and can provide information on composition, microstructure, and function of muscles or groups of muscles. These techniques have been shown to be useful for evaluating the recruitment of muscles for specific activities, for biological and metabolic muscular function, for muscle composition including the assessment of early fat atrophy, and for muscle microstructure and elasticity. These advanced techniques could be very helpful when routine imaging cannot explain the persistence of limited muscle function and/or strength after appropriate management. Their potential and their application in sports medicine is a subject wide open for further clinical research.

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30. Three-Compartment Body Composition Measurement by Dual-Energy X-Ray Absorptiometry: Use in the Prevention of Cervical Spine Trauma and in the Follow-Up of Muscular Injuries in Elite Rugby Union Players

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Abstract
Our purpose is to emphasize the use of DXA for the assessment of skeletal muscular mass in rugby players. Ideally, seasonal follow-up with DXA includes three scans during the playing season but this objective is quite difficult to reach. We studied 38 elite senior players for two seasons, but unfortunately only eight were scanned at least three times in both seasons. One player underwent MRI and DXA studies after a spinal

trauma with whole body and paraspinal three-compartmental measurements at 3 weeks. DXA is the gold standard for following fat mass and lean mass components of the players, as well as for total body and regional studies of cervical musculature. Each player gets a three compartmental study, monitored by colored visual mapping and by diagrams of follow-up over time. The lean mass/height^2 ratio and appendicular lean mass/height^2 ratio are good criteria for the assessment of lean mass. DXA is also a good tool for the prevention of injuries and for assessment of recovery after lower limb and cervical spine injuries. We suggest adding DXA to the protocol for the evaluation of capacity to play in the French Rugby Federation.

30.1 Introduction

Muscle is one of the components of performance along with bony structures, tendons, ligaments and brain. Good muscular architecture allows strength, athletic power, speed and suppleness.

Epidemiological studies have confirmed the high rank of muscular lesions among the injuries sustained by senior rugby union players, particularly in the lower limbs [1–5]. Because the recurrence rate is so high, analysis of risk factors and prevention of injuries are both good targets for study.

Detailed knowledge of total body composition is much more important than simply measuring fat with a skin fold test. DXA-calculated lean soft tissue is the "gold standard" and has been shown to closely approximate skeletal muscular mass [6].

DXA then is a good method for assessment of muscular tissue. Monitoring of the regional distribution of fat free lean mass and 11 fat mass is obviously useful for an anthropometric survey of elite rugby players [7, 8] because rugby has weight requirements that players must meet.

Our previous work was directed towards the prevention of cervical spine injuries [9, 10] and we have also studied cervical muscular structure in former rugby players [11].

So we propose to the French Rugby Federation (Fédération Française de Rugby (FFR)) a new classification – based on clinical and MRI items – for determining whether an athlete is qualified for play [12].

The actual purpose is to demonstrate the contribution of DXA to measurement of three-compartment body composition in the follow-up of rugby players and to underline the importance of lean mass in relation to muscular mass.

Distinguishing fat mass from lean mass can be done in part by "indicial and mapping" follow up with, for example, DXA, which requires very little radiation. Thus there are multiple advantages to the use of DXA scanning in sports medicine:

- Performance can be correlated with lean mass [7], because muscle generates

strength;

- Distribution of muscle mass can be analyzed according to the player's post [13];
- Recovery of lean mass after injury can be tracked [14];
- Cervical muscle mass can be followed after spinal trauma, because DXA allows paraspinal measurement;
- Results of dietary programs can be assessed [15].

30.2 Methods

30.2.1 Experimental Approach to the Problem

The Imaging Department of Medipole Garonne entered into a partnership with two elite rugby union teams, the Stade Toulousain and the Castres Olympique.

Every year we perform an MRI study of the cervical spine of new players to evaluate their risk of neurological injury during the season. Static and dynamic sagittal MRI sequences of the spine are compared with an axial evaluation of muscular structure.

For 3 years we have set a follow-up protocol for the three compartment study of body mass by DXA, ideally with three scans – pre, mid and post season.

We reserved a particular follow-up for players who gained too much weight in the interseason and for those with lower limb and spinal injuries.

30.2.2 Subjects

For this paper, only the DXA follow-up of rugby players from the Stade Toulousain rugby team was retained, because they had the most significant number of examinations. During the 2011–2012 season 21 players were studied but only 23 DXA scans were acquired, and during the 2012–2013 season there were 30 players with 52 DXA studies.

The number of players scanned was 38: 21 forwards and 17 backs.

All the athletes agreed to take part in the study, which was approved by medical staff.

The plan for three DXA scans per season quickly turned out to be a utopia.

Because of turnover on the team, only 14 players were followed for two consecutive years. Follow-up was performed for eight players investigated at least three times and on two seasons. One player was followed for more than 2 weeks after a cervical spinal trauma.

30.2.2.1 Procedures

All of our players underwent a total-body dual-energy X-ray absorptiometry (DXA) scan (Discovery W, 128 detectors, Hologic Inc. USA). Scanning and analysis accorded with the whole-body dataset from the NHANES population-based sample which provides reference values for subjects 8–85 years old and by gender and ethnicity [16].

DXA was used to measure three aspects of the whole body.

– Fat mass: percent fat (total body-fat mass ratio), fat mass/height^2 (kg/m^2), percent fat trunk/percent fat legs ratio and trunk/limb fat mass ratio.

– Lean mass: lean mass/height^2 ratio and appendicular lean mass/height^2 ratio (kg/m^2).

– Bone mass: bone mineral content (BMC) and bone mineral density (BMD).

Specific regions, the trunk, arms, legs and the cervical paraspinal muscles can also be similarly analyzed (Fig. 30.1).

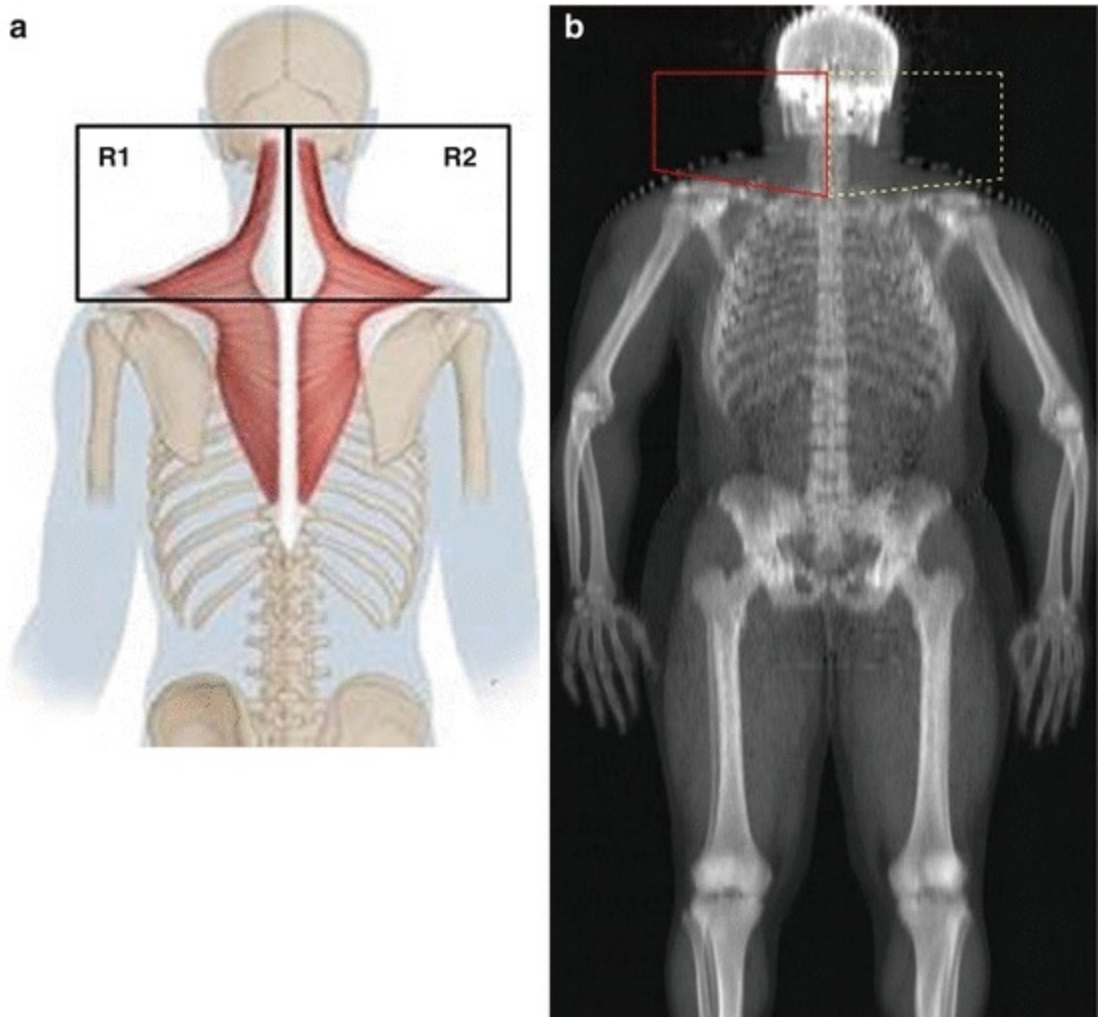


Fig. 30.1 Cervical sub-region (**a**) chosen for DXA measurements (**b**)

With DXA, the least detectable mass is 250 g. The accuracy rate is 2 % for a reproducibility of ± 200 g for the members and of ± 450 g for the thorax.

Effective X-ray dose for a whole-body scan is only 8.4 μSv.

Dynamic calibration is integrated into the measurement procedure.

DXA cannot measure hydration status but is robust with respect to variations in lean mass hydration. Hologic Inc. takes into account a constant coefficient of hydration of the lean mass of 73.2 % which little influences the measurement of fat mass and lean mass.

30.2.3 Follow-Up of the Indices (Fig. 30.2)

For each player, the three compartmental trend was monitored by colored visual mapping and by diagrams of follow-up over time.

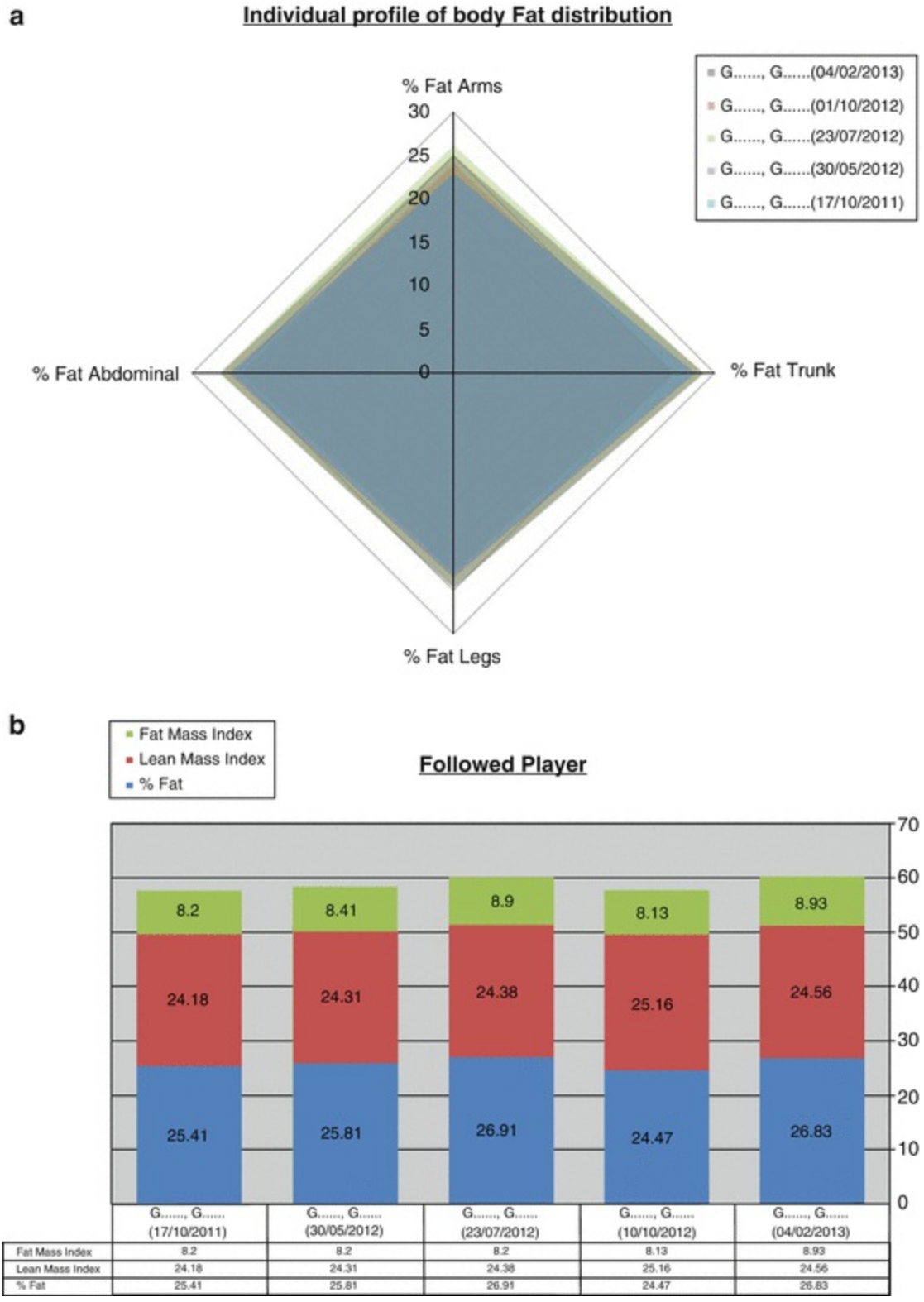


Fig. 30.2 Individual follow-up for body fat distribution (**a**) and fat mass/lean mass indices (**b**)

This type of monitoring can emphasize the variations of fat mass and lean mass in the trunk and the limbs (percent fat, fat mass ratio, lean mass ratio).

Polygonal diagrams are very attractive to show the individual profile of the player

for separation of fat mass and lean mass with four indications of percent mass for legs, arms, trunk and abdomen.

30.3 Results

Table 30.1 describes the baseline characteristics of the cohort at the start of the study for all the players: age, weight, height, BMI.

Table 30.1 Baseline characteristics for the cohort at the beginning of the study

Table 1 (n = 38)	Forwards (n = 21)	Backs (n = 17)
Characteristics		
Mean age (years)	25	26.8
Mean height (cm)	190.5	182.8
Mean weight (kg)	116.5	93.8
Mean BMI (kg/m^2)	32.2	27.9

Table 30.2 describes the ratios for all the players at the start of the study: fat mass percent, fat mass/height^2, lean mass/height^2, appendicular lean mass/height^2. SD for lean mass/height^2 is also shown by player position, i.e. forwards and backs.

Table 30.2 Baseline ratios for the cohort at the beginning of the study

Table 2 (n = 38)	Forwards (n = 21)	Backs (n = 17)
Characteristics		
Fat mass ratio (percent fat)	20.1	15.3
Fat mass/height^2	6.5	4.3
Lean mass/height^2	24.9	23.5
Appendicular lean mass/height^2	13.5	11.5

Figure 30.2 shows the indicial individual follow-up for players.

Figure 30.3 shows the case of a cervical sprain (hyperflexion) followed by MRI and DXA for 3 weeks. In that 3 weeks:

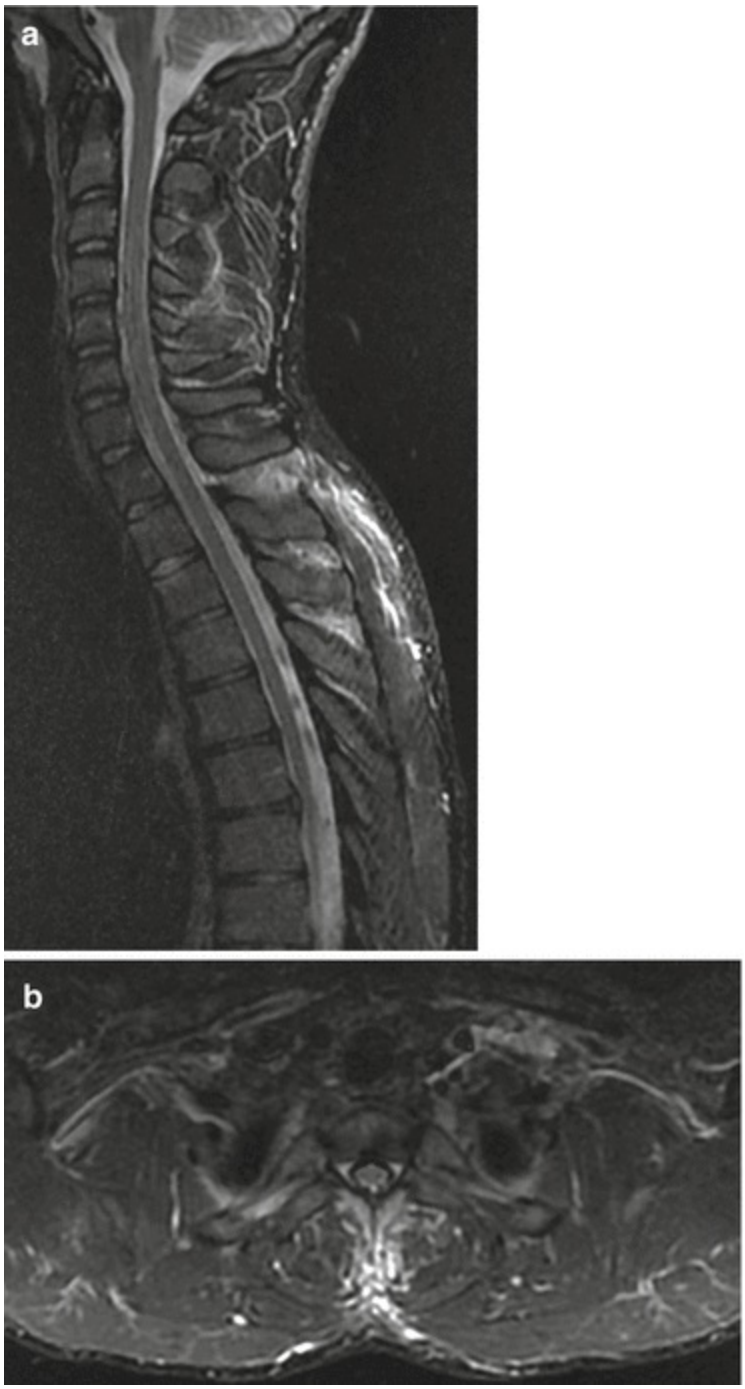
– total body lean mass + bone mass decreased 2.79 %;

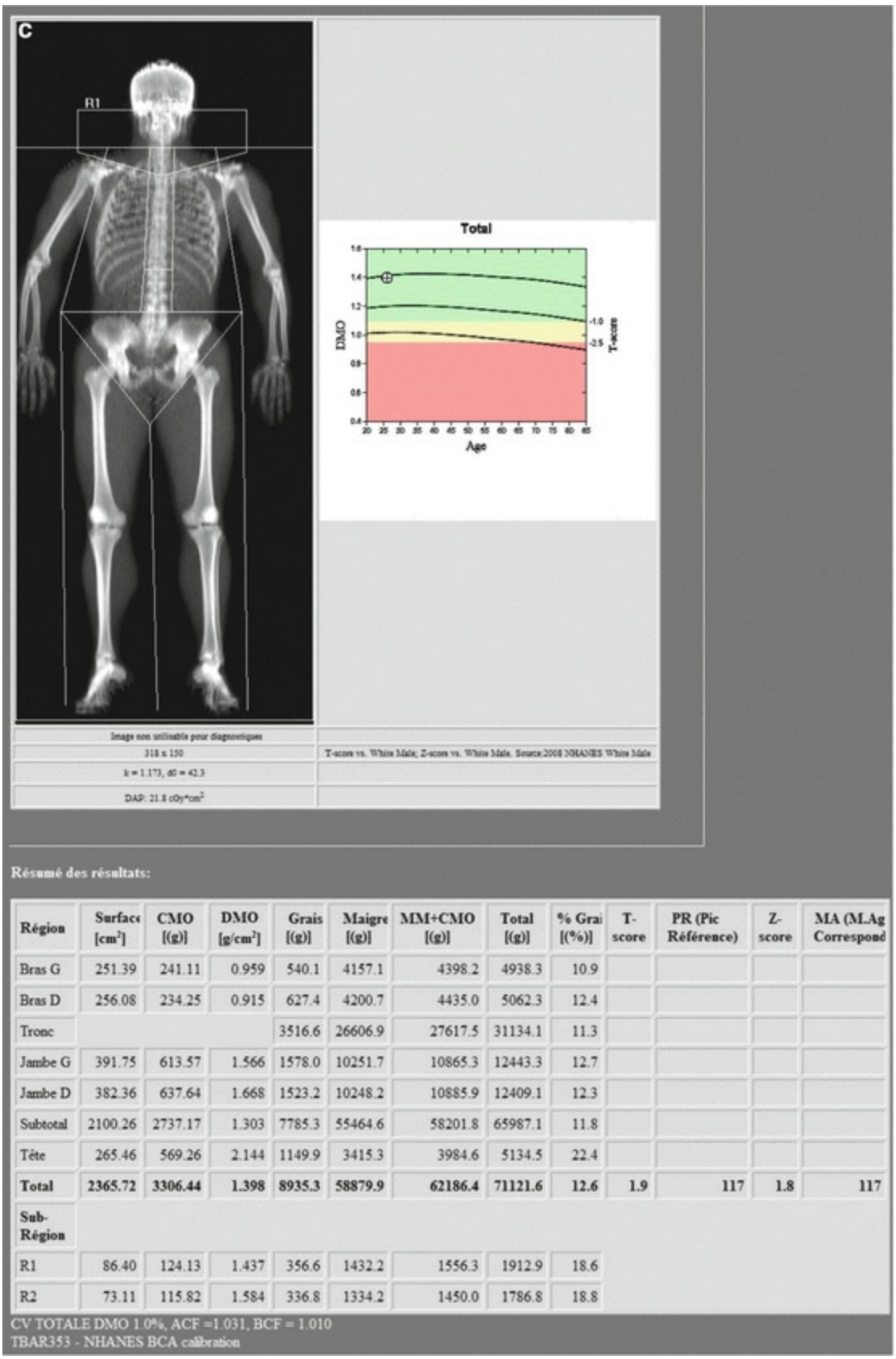
– lean mass/height^2 changed from 20.5 to 20, appendicular lean mass/height^2 changed from 10.1 to 9.75;

– total body percent fat increased from 12.6 to 12.8 (1.58 %);

– analysis of cervical sub regions showed no significant variation for percent fat

whereas the total cervical lean mass +bone mass increased 4.27 %.





Région	Surface [cm²]	CMO [(g)]	DMO [g/cm²]	Grais [(g)]	Maigre [(g)]	MM+CMO [(g)]	Total [(g)]	% Grai [(%)]	T-score	PR (Pic Référence)	Z-score	MA (M.Ag Correspond
Bras G	251.39	241.11	0.959	540.1	4157.1	4398.2	4938.3	10.9				
Bras D	256.08	234.25	0.915	627.4	4200.7	4435.0	5062.3	12.4				
Tronc				3516.6	26606.9	27617.5	31134.1	11.3				
Jambe G	391.75	613.57	1.566	1578.0	10251.7	10865.3	12443.3	12.7				
Jambe D	382.36	637.64	1.668	1523.2	10248.2	10885.9	12409.1	12.3				
Subtotal	2100.26	2737.17	1.303	7785.3	55464.6	58201.8	65987.1	11.8				
Tête	265.46	569.26	2.144	1149.9	3415.3	3984.6	5134.5	22.4				
Total	**2365.72**	**3306.44**	**1.398**	**8935.3**	**58879.9**	**62186.4**	**71121.6**	**12.6**	**1.9**	**117**	**1.8**	**117**
Sub-Région												
R1	86.40	124.13	1.437	356.6	1432.2	1556.3	1912.9	18.6				
R2	73.11	115.82	1.584	336.8	1334.2	1450.0	1786.8	18.8				

Fig. 30.3 Cervical sprain between the spinous processes of the first and second thoracic vertebrae. Sagittal (**a**) and axial (**b**) STIR MRI shows interspinous ligament tear and edema seen as a hyperintensity signal. Detailed DXA measurements with cervical sub-region R1 and R2 (**c**)

30.4 Discussion

30.4.1 Specificity of the Rugby Player

For a man of medium corpulence, the fat-mass ratio (total body fat-mass ratio) is between 15 and 20, and the lean-mass ratio between 35 and 40.

According to the data of the World Health Organization [17], the normal BMI (body mass index) is between 18.5 and 25 kg/m^2. In reality most of the rugby players have a BMI beyond 25 kg/m^2 without being overweight or obese.

Thus, we shall insist on using fat mass/height^2 and lean mass/height^2 ratios, the only indices that are really adapted to the follow-up of rugby players.

Another dilemma is the antagonism between the protecting mass, fat and lean, and the performance/weight ratio. In many sports it is considered and proved that carrying excess body fat can reduce performance because of the weight overload and slower performance. On the other hand, higher subcutaneous fat is also considered protection against injury [18]. The “airbag” represented by fat and muscles protects the human body.

A comparative analysis of mass distribution by player post is also interesting [13]. According to the literature, height, weight, percent fat mass and BMI are higher in forwards than backs. Our Tables 30.1 and 30.2 confirm this fact, and also that the ratios for fat mass and lean mass are both higher in forwards.

30.4.2 Importance of the Follow-Up in the Change of Lean-Mass Composition [14, 19, 20]

The ideal times for DXA follow-up of rugby players for our purposes would include each of the three phases of the competitive season: pre-season (August or September), mid-season (January or February), and post-season (June or July).

In reality this ideal is quite impossible because of the busy schedules of the teams.

Georgeson et al [14] studied three-compartment body changes and tried to determine their relationship to injury.

Does body composition change across a 12-month period in response to pre-season training, seasonal game play and off-season rest? Is pre-season body composition (bone, muscle and fat) related to injury incidence throughout the season? Are changes in body composition during the playing season related to incidence of injuries?

We asked these questions and found that professional rugby league players tend to lose lean mass across a playing season but regain it with pre-season training. No gain for fat mass was found in this study.

BMD increased until mid-season and decreased thereafter.

No strong relationships were detected between anthropometric characteristics and

incidence of injury. Georgeson et al however questioned whether reductions in lean mass might reduce muscle strength and endurance, speed, agility and power across the course of the season.

Harley et al [20] noticed that between the beginning and midpoint of the season, bone mass, fat mass, lean mass and per cent fat showed no significant change. BMC however increased significantly.

Between mid-season and post-season, bone mass and BMC showed no significant change, although significant changes were observed in lean mass (-1.54 %), fat mass (+4.09 %), and percent fat (+4.98 %).

The significant changes in body composition seen over the latter stages of the competitive season may have implications for performance capabilities at this important stage of competition. An increase in fat mass and decrease in lean mass may have a negative effect on the power/bone mass ratio, and therefore may be a cause for concern for playing, coaching, and medical staff. Coaching and strength and conditioning staff should prescribe appropriate training and nutritional practices with the aim of maintaining the players' optimal body composition until the conclusion of the competitive season, so that performance capabilities are maximized over the entire competition period.

Because lean mass (muscular mass) is the real provider of strength for the athlete, we think that the lean mass/height^2 ratio and the appendicular lean mass/height^2 ratio seem to be the best indices for the study of skeletal muscular mass.

The frequency of lower limb injuries is high, so we want to insist on the importance of the assessment of lower-limb volumes [21], but the comparison with upper limbs and the search for any asymmetry should also be considered

We also believe that the age-related decrease in muscle mass and function, known as sarcopenia [6] is correlated with functional limitation and frailty.

Moreover we know that satellite promyoblastic cells [22] are naturally present in all the skeletal muscles, but that their density in muscles decreases with the age.

The wounded muscle can regenerate from the easily actionable satellite cells by creating new muscular fibers. The production of cytokines facilitates the repair.

Thus, DXA can be a tool for the follow-up of muscle healing.

30.4.3 Contribution of the Lean Mass Component to BMD and Relation with Sports

So it is clear that sports favors development of lean mass but not fat mass. It has already been established that in men lean mass is a significant determinant of BMD whereas fat mass is a negligible determinant [23].

. Moreover, total body lean mass, as well as muscular strength, strongly correlated with total body mass and with the BMD and BMC of regional sites of stress such as the

spine, pelvis, and lower limbs.

Playing rugby is associated with improved physical fitness, enhanced axial and appendicular bone mass and increased bone turnover in adults [24].

These facts strongly correlate with our impressions in former rugby players, that well developed cervical muscle mass protects against pain.

Regarding prevention of injuries in rugby players we can say that muscle and bone are fully connected. Without muscle the bone is exposed to osteoporosis and fracture. Pacific Islanders, for example, have been shown to have larger bones and greater BMD than whites and greater body mass [25]. And we know that rugby players are getting bigger with a big increase in height and weight.

30.4.4 Prevention of Traumas

For all the players, and not only for front line players, and for future generations of players, a strong cervical muscular architecture is the major element of protection against serious neurological wounds.

The muscular mass must be preserved and increased to protect the cervical spine.

Exercise to strengthen the cervical muscles and increase their mass is necessary, and should be prescribed for young athletes as well as professionals.

Stade Toulousain and the French national rugby team in Marcoussis use a “scrum machine.”

Our study of former rugby players [11] indicates that professional rugby seems to induce the occurrence of degenerative cervical spine diseases. On the other hand, rugby players maintain stronger paraspinal muscular mass compared to the general population. Studies of muscular mass using MRI [11] have shown that former rugby players have a significantly greater muscular surface than control patients, regarding the longus colli and splenius capitis.

We also know that muscle strengthening programs are effective for controlling chronic neck pain [26]. Thus we think that regularly practicing sports can help to control pain, as has been demonstrated [27]. It seems equally true that the physiology of the muscle is also a factor in controlling pain, as demonstrated in gonarthrosis [28].

The use of DXA for the specific study of lumbar spine and correlated prevention for muscular and spinal injuries was considered by the medical staff of ASM Clermont Auvergne, another team in the FFR [29, 30].

We quote their conclusions with a total approval.

The DXA measures the composition of the body, bone mineral density, and spine morphology in order to determine the effects of both training and nutrition. It also helps to establish specific training programmes adapted to the spine morphology and, when necessary, to the anomaly revealed during the scan. (29)

“The early disc degeneration is a reality in combat and contact sports. Careful

clinical monitoring is necessary to detect and take care of these pathologies. But as a priority, it is necessary to integrate since the young age a program of protection of the spine and to adapt it to the growing constraints required in above age categories, depending on the level of practice and this until the age of 18 years" [30].

In the spine as well as the limbs it is necessary to respect the balance between flexors and extensors muscles.

The follow-up of fat mass and lean mass + bone mass in our only case of spinal trauma is a good example. The study of the paraspinal fat mass and lean mass components must be systematic to obtain a satisfactory statistical follow-up. This type of paraspinal study seems to us to be a new tool for the follow-up and prevention of spinal injuries in rugby.

30.4.5 Practical Applications and Conclusions

Ideal timing for DXA seasonal follow-up of rugby players ideally is during each of the three phases of the competitive season.

A good dietary maintenance program is useful, mainly at the off-season and at the beginning of the season to rebalance muscle and fat mass ratios. DXA is good tool for the monitoring of this dietary program, in close consultation with a dietitian.

We have to increase the role of DXA for the screening of elite rugby union players to improve protection of the cervical spine. Then we must add to our "cervical spine MRI protocol" [9–12] a systematic three-compartment analysis of the cervical paraspinal muscles. This "targeted" DXA study of cervical muscles is more rigorous than a simple T1-weighted MRI cervical slice with measurement of muscular surfaces.

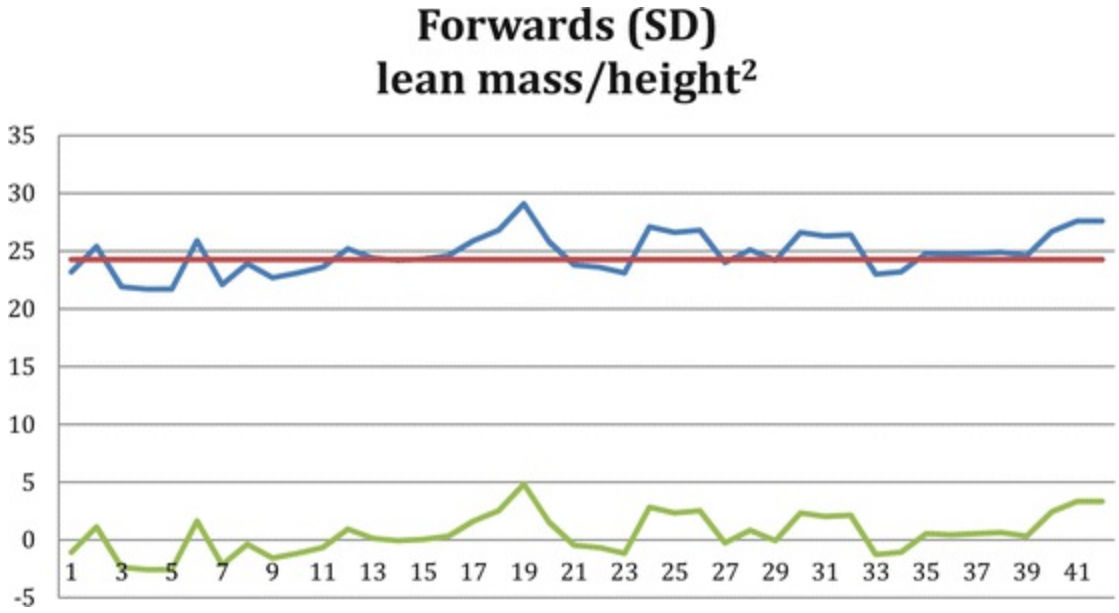
After spine trauma return to play can be monitored by clinical data, MRI sequences and localized DXA.

We must insist on the role of the lean mass indices and especially the lean mass/height^2 ratio and appendicular lean mass/height^2 ratio, for the follow-up of muscular injuries.

Although at the moment the literature is not convincing concerning the correlation between muscular injury and lean mass variations [14], we have set up our own program of follow-up of lean mass. Two main studies concerning body mass changes and rugby [14, 20] both confirm the variation of lean mass. Then DXA can help to monitor the lean mass and fat mass indices, and especially lean mass imbalance resulting from muscular injury, with the aim of managing physiotherapy and nutrition plans, and thus prepare for the optimal rehabilitation.

In the collaborative effort of the Imaging Center of Medipole Garonne, Hologic Inc. and Stephanix Inc., some advanced reporting protocols have been developed to simplify the analysis of body composition in top athletes. The objective was to create self-explaining diagrams expressing the evolution of fat and lean mass in rugby players

sorted by post while also enabling comparisons between multiple players and/or posts. The originality of this tool is that, while developed for rugby, its versatility and ease of use make it a fantastic instrument for body composition follow-up in any sport, transforming extended raw values into easy-to-read diagrams. On top of easing the reporting process, this tool also seriously improves subject compliance in either specific training or diet programs to improve overall body composition.



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