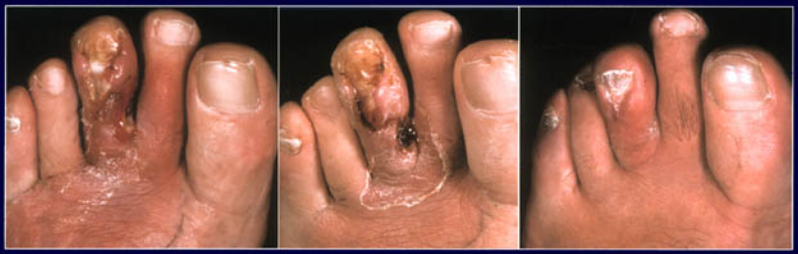
A Practical Manual of Diabetic Footcare

Michael E. Edmonds,
Alethea V.M. Foster and
Lee J. Sanders



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A PRACTICAL MANUAL OF

Diabetic Foot Care

To our families: Audrey, Stephen and
Susie Edmonds; John, Julien, William and Dennis Foster
and Debra, Rebecca, Douglas and Lauren Sanders.

A PRACTICAL MANUAL OF
Diabetic Foot Care

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We refer to patients throughout the book as 'he' simply because more men than women seem to develop diabetic foot ulcers.

This is a practical hands-on manual, uninterrupted by references. At the end of each chapter, we have given a classified reading list which should provide further information for our readers.

Prologue

Time's the king of men;
He's both their parent and he is their grave,
And gives them what he will, not what they crave.
(Pericles, Prince of Tyre, *II, iii, William Shakespeare)*

THE SCOPE OF THE PROBLEM

Diabetic foot complications are a major global public health problem. Amputation rates vary throughout the world but are always increased in people with diabetes compared to those without diabetes. Amputations are increasing in diabetic patients. Throughout the world, health-care systems, both public and private, have been unsuccessful in managing the overwhelming problems of patients suffering with diabetic foot complications. The results of this failure are shown in the following case histories, illustrated in Figs 1–5.

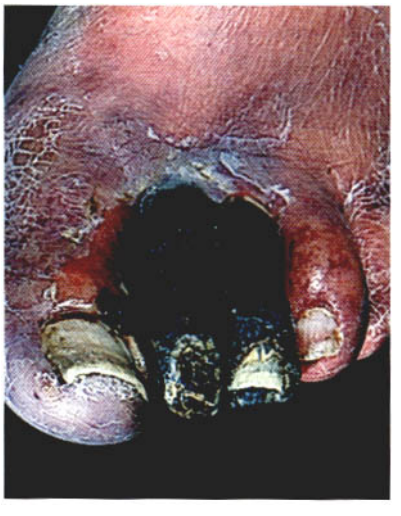

Fig. 1 Foot from the UK. This 85-year-old man with type 2 diabetes of 8 years' duration received regular dressings of his ulcerated ischaemic foot for 9 months, but was not referred until extensive gangrene had developed.

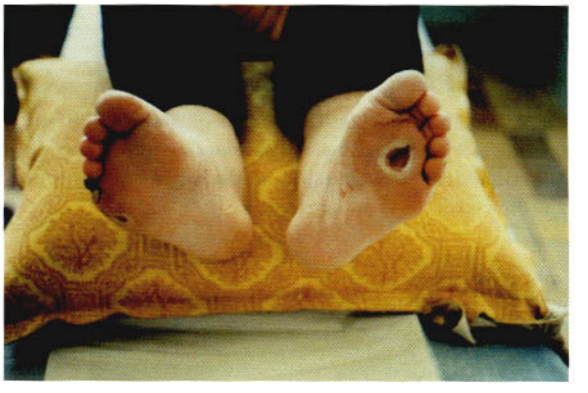

Fig. 2 Foot from Ukraine. This 48-year-old man with type 2 diabetes of 12 years' duration trod on a nail and developed severe infection with wet gangrene of the right 5th toe. He had a longstanding neuropathic ulcer of the left foot.

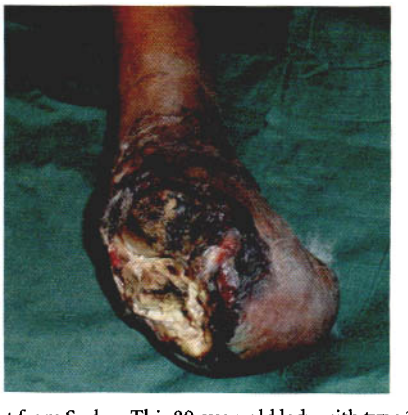

Fig. 3 Foot from Sudan. This 80-year-old lady with type 2 diabetes of 15 years' duration and neuropathic feet sustained a puncture wound through the thin sole of her sandal. She did not seek advice and developed profound sepsis.

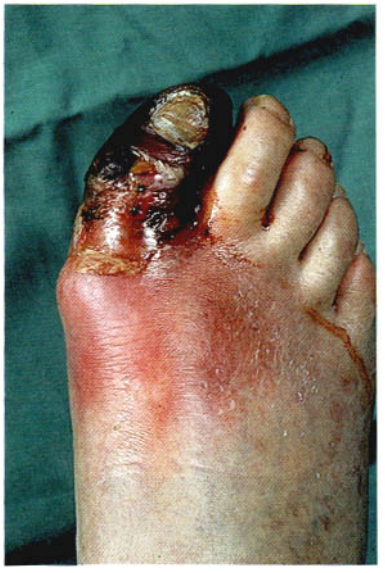

Fig. 4 Foot from the USA. This 58-year-old woman with type 2 diabetes of 9 years' duration, wore a tight shoe which rubbed a blister. She did not seek help because the blister was not painful and presented late—with gangrene.

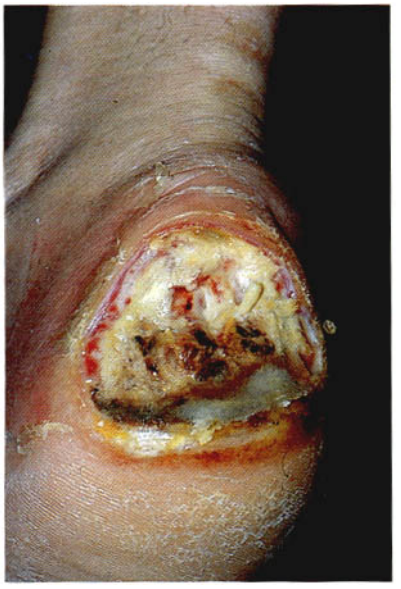

Fig. 5 Foot from Australia. This 75-year-old man with type 2 diabetes of 20 years' duration developed fissures round his heel which were a portal of entry for severe infection.

These pictures show authentic diabetic feet from five continents of the world. Foot catastrophes such as these, as Elliott Joslin pointed out, do not strike like lightning out of heaven, but are too often due to ignorance and apathy, which prevent patients from detecting problems early and seeking treatment, and which prevent health-care professionals from organizing rapid and effective care. In nearly every case there are warning signs which, if acted upon, could prevent tragedy. However, because of local barriers to effective care, patients often do not receive help in time to save their feet.

Diabetic patients in the real world are often perceived as the poor relations, the 'lepers of our time'. Indeed, diabetic foot patients have more in common with lepers than just neuropathy: in many quarters they are regarded with disgust and antipathy as dirty, smelly, 'unclean', socially unacceptable feet belonging to patients who take up hospital beds for unacceptably long periods of time.

Equally, diabetic foot patients may be regarded by inexperienced staff as 'feckless' patients, who fail to look after themselves and are directly responsible for their problems. Health-care systems are 'symptom-led' and thus fail to respond to the needs of the diabetic foot patient, who usually has neuropathy, numb feet and no complaints.

In addition, diabetic foot problems are frequently underestimated. Just as there is no such entity as 'mild' diabetes, there is no such thing as a 'trivial' lesion of the diabetic foot.

Sadly, there are many areas of the world where people with diabetes are unable to obtain good foot care, or where the provision of such care is dependent upon the patient being able to pay for it. A recent tragic case involved a diabetic man with indolent neuropathic ulceration, who amputated his own leg (using a railway line and a passing train) because he could not pay for medical care.

However, amputations are not inevitable. The aim of this book is to help readers to achieve good care for patients with diabetic foot problems and so avoid preventable amputations. Progression down the road to amputation is not inevitable and relentless. Patients can be rescued.

HISTORICAL BACKGROUND

The last century made great inroads into improving the management of diabetes. The early work of pioneers such as Nicolas Paulesco in Rumania and Georg Zuelzer in Germany culminated in the work of Banting, Best, Collip and Macleod in Canada who produced a pancreatic extract which was used successfully in patients and ended the

inevitable 'death sentence' hitherto attached to a diagnosis of type 1 diabetes. Insulin became widely available, and the subsequent development of oral hypoglycaemic agents and blood glucose monitoring also led to improved outcomes for type 2 patients. However, many diabetic patients lived longer only to develop diabetic complications, including peripheral neuropathy, peripheral vascular disease, ulceration, foot sepsis and gangrene.

The work of Elliott Joslin (USA) and R.D. Lawrence (UK) during the first half of the twentieth century was concerned with all aspects of diabetes management including the management of the foot. Since then there has been activity on all continents to attempt to reduce amputations and improve outcomes in diabetic foot patients, which have been particularly evident over the past 15 years.

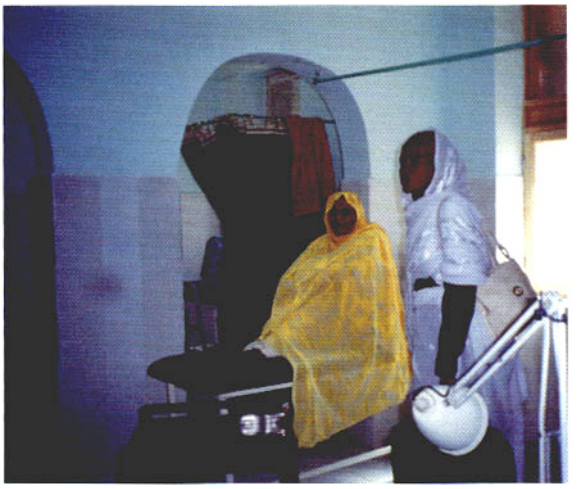

Fig. 7 The Khartoum Diabetic Foot Clinic.

ADVANCES IN DIABETIC FOOT CARE

The diabetic foot has become a major area of interest, and insight has been gained into the reasons why diabetic feet go wrong and the ways in which patients can be helped. Of all the complications of diabetes, the diabetic foot is probably the easiest to prevent and treat.

The groundswell of interest in the diabetic foot surged in the 1980s, and developments in foot care included the setting up of multidisciplinary diabetic foot clinics (Fig. 6) and the pioneering educational work of Jean Philippe Assal in Geneva, Switzerland. Paul Brand, Frank Tovey and Grace Warren worked in India as medical missionaries with leprosy patients and subsequently applied their knowledge to the management of diabetic neuropathic patients, spreading the word to Carville, USA, Basingstoke, UK, and Sydney, Australia. The popular biennial Malvern diabetic foot conferences began in 1986, and in 1991 Karel Bakker of The Netherlands established the regular International Symposia on the Diabetic Foot at Noordwijkerhout in The Netherlands. Bakker's work led to the establishment of International Working Group of the Diabetic Foot, which produced the *International Consensus on the Diabetic Foot*, published in 1999 (and now translated into 20 languages). Lee Sanders, who has contributed the chapter on surgery to this book, was the first podiatrist to be elected President for Healthcare and Education of the American Diabetic Association.

Fig. 6 International visitors at the King's Diabetic Foot Clinic: left to right, Dr Kamenov (Bulgaria), the Authors, Dr Harkless (USA) and Dr Plamen (Bulgaria).

Professor Mohammed Rasheid, a surgeon, established one of the first African diabetic foot clinics in Khartoum, Sudan, in 1998 (Fig. 7). Dr Hermelinda Pedrosa in Brazil has organized a national programme of diabetic foot care. Dr Theresa Que established the first diabetic foot clinic in The Philippines. The fall of the 'Iron Curtain' across Eastern Europe in the 1990s enabled the setting up of diabetic foot clinics in many countries including East Germany (Fig. 8) Russia, Romania and Lithuania (Fig. 9) The late Jacquie Lloyd Roberts, a UK podiatrist, established a successful chain of diabetic foot clinics in Ukraine before her untimely death.

The work of diabetic foot clinics, operating in different parts of the world, in very different conditions, has clearly demonstrated that outcomes for diabetic foot patients can be improved when dedicated and enthusiastic clinicians organize a multidisciplinary diabetic foot service. With a flexible approach, most problems can be overcome.

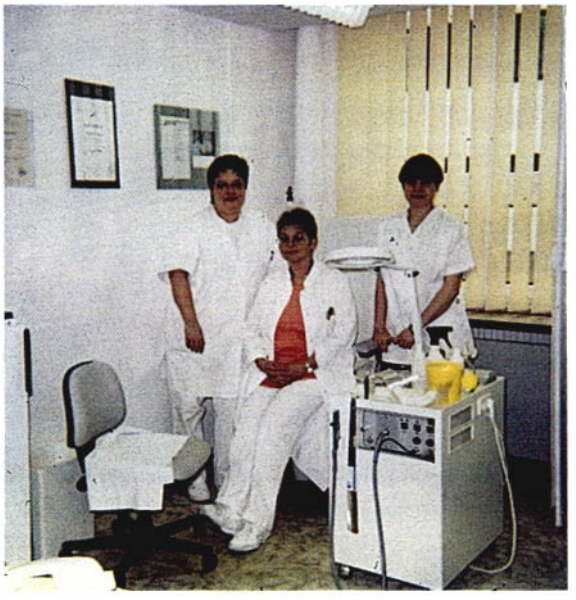

Fig. 8 Foot clinic in Magdeborg, East Germany—Dr Karola Zemlin's team.

Fig. 9 Foot care nurses in Kaunas who learned foot care from an English podiatrist, Abigail Clarke.

If key members of the team, such as podiatrists, are not available in certain countries, then doctors or nurses can take on many aspects of the role of the podiatrist. Indeed, we have learnt from the work of Dr Grace Warren with Hansen's disease patients that neuropathic patients and their families can be taught safe self-care techniques for removing callus to prevent ulceration if no other help is available.

One of the most important messages from these workers is that successful interventions in the real world do not depend on the possession of high-technology

Fig. 10 International consensus and practical guidelines on the diabetic foot.

equipment and vast financial budgets. Barriers to care which at first glance appear to be insurmountable can usually be overcome if we learn lessons from our own and other people's experiences, and, in the words of E. M. Forster, 'only connect' with each other.

'Experts' are sometimes called in to help to set up systems for managing the diabetic foot. However, experts are often outsiders from other regions or countries, who may lack information about local conditions and should refrain from being too dictatorial to local practitioners who may have expert and first-hand knowledge of local conditions and problems. While we always welcome visitors from abroad to our foot clinics, we try to avoid being too dogmatic about how they should manage feet back home: they observe what we do and extract what is relevant to their own situation but should not regard our messages as being cast in stone.

There is a dearth of evidence for treatments applied to the diabetic feet. One of the problems encountered in developing guidelines for management of the diabetic foot is that evidence is often lacking, and rarely comes from large, properly conducted, randomized controlled trials. Our recommendations are based either upon first-hand experience gained over the past 22 years working in the outpatient diabetic foot clinic and on the wards or on the published work of other groups, throughout the world.

In the spirit of the International Consensus on the Diabetic Foot (Fig. 10), we have tried to make this book relevant to all practitioners who want to set up a diabetic foot service, no matter where or under whatever conditions they labour: we hope that there is something here for everybody.

These are heady times for devotees of the diabetic foot, and we hope and believe that a new generation of young and enthusiastic practitioners will be there to take up the baton for the diabetic foot in the future. We hope that they will find this book useful and practical.

PRACTICE POINTS

- Throughout the world, health-care systems are symptom-led and they often fail to respond to the needs of the diabetic foot patient
- Diabetic foot problems are frequently underestimated: there is no such thing as a trivial lesion of the diabetic foot
- Diabetic foot patients are unfairly regarded as 'feckless'
- Increased interest in the diabetic foot over the last 25 years has resulted in major advances in the care of the diabetic foot patient
- The multidisciplinary diabetic foot service has been developed as a successful model of care throughout the world.

FURTHER READING

The scope of the problem

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1 Introduction

Time is the nurse and breeder of all good.
Hope is a lover's staff; walk hence with that,
And manage it against despairing thoughts.
(The Two Gentlemen of Verona, *III, i, William Shakespeare)*

In this book, the fundamental approach to the diabetic foot is built on a simple staging system which has been developed to provide a framework for diagnosis and management.

There are four main themes in this chapter.

- Practical assessment consisting of history, examination and investigations
- Basic classification of the diabetic foot, into the neuropathic and neuroischaemic foot
- Simple staging system, describing six specific stages in the natural history of the diabetic foot (Table 1.1)
- Multidisciplinary management plan for each stage.

This overall approach to the diabetic foot begins with a simple assessment to enable the practitioner to make a basic classification and staging. The practitioner can place the foot into each stage by means of a clinical assessment.

Having then placed the foot in a particular stage, investigations will be needed to assess severity so as to determine treatment. The book contains sufficient easily accessible information to enable the practitioner to make rapid, effective decisions which will detect problems early, organize rapid treatment and prevent deterioration and progression.

Table 1.1 Stages of the diabetic foot

Stage 1	Normal foot
Stage 2	High-risk foot
Stage 3	Ulcerated foot
Stage 4	Infected foot
Stage 5	Necrotic foot
Stage 6	Unsalvageable foot

PRACTICAL ASSESSMENT

This can be divided into three parts:

- History
- Examination
- Investigations.

History

Every attempt should be made to encourage the patient to be open and non-defensive. The history can be divided into the following sections:

- Presenting complaint
- Past foot history
- Diabetic history
- Past medical history
- Family history
- Drug history
- Psychosocial history.

Presenting complaint

Be aware that some patients may be asymptomatic due to neuropathy.

The presenting complaint is usually one or more of the following:

- Skin breakdown
- Swelling
- Colour change
- Pain.

For skin breakdown, swelling and colour change or any other presenting complaints, the following questions may be helpful:

- Where is the problem?
- When did it start?

- How did it start?
- What makes it better?
- What makes it worse?
- How has it been treated?

As regards pain, this may be a specific complaint alone or it may accompany the above problems.

Pain may arise locally or it may be diffuse. Local sources may originate from bone, joint and soft tissue including skin and subcutaneous tissue. Generalized pain in both feet suggests neuropathy. Diffuse pain in a single foot suggests ischaemia. However, pain in the ischaemic foot should not always be blamed on reduced arterial perfusion because it may be caused by infection. In the neuropathic foot, severe infections can still cause pain, particularly throbbing pain. Pain around an ulcer suggests infection or ischaemia. The following questions should be asked about pain:

- When did it start?
- How did it start?
- Was there an injury?
- Where is the pain?
- What is its nature?
- What aggravates the pain?
- What relieves the pain?
- When does it occur?
- Is it related to time of day or activity?
- What treatments have been given so far?

Clinical tips to diagnose pain due to neuropathy and ischaemia are shown in Table 1.2.

Table 1.2 Clinical tips on pain

Pain due to neuropathy

- Burning pain with contact discomfort in both feet and lower legs which may also involve the thighs
- Sharp shooting (lancinating) or lightning pains like electric shocks, lasting a few seconds
- Pain relieved by cold
- Pain worse during periods of rest
- Unilateral burning pain in the leg with muscle wasting suggests a focal neuropathy, commonly a femoral neuropathy

Pain due to ischaemia

- Persistent pain, worse on elevation and relieved by dependency (hanging the leg out of bed)
- Pain in the calf on exercise relieved by rest (claudication). However, claudication is often absent in ischaemia because of concurrent neuropathy and the distal distribution of the arterial disease
- Feet with severe ischaemia may have little pain because of neuropathy

Patients may not complain of pain itself but of other abnormal sensations which would suggest neuropathy.

- Pins and needles (paraesthesiae)
- Unpleasant tingling (dysaesthesiae)
- Tightness (as if a constricting band is around the foot)
- Cold
- Heaviness
- Numbness ('my feet feel as if they don't belong to me').

After discussing the presenting complaint, the rest of the history is devoted to gathering important relevant information about the patient to aid diagnosis and management. This information can be acquired from various sources including direct questioning of the patient, the patient's medical notes and the referral letter.

Past foot history

- Previous ulcers and treatment
- Amputations:
  Major
  Minor
- Peripheral angioplasties
- Peripheral arterial bypasses.

Diabetic history

- Type of diabetes
- Duration of diabetes
- Treatment of diabetes:
  Insulin
  Oral hypoglycaemics.

Complications of diabetes

Retinopathy

- Background
- Proliferative
- Previous laser therapy
- Vitrectomy
- Cataract.

Nephropathy

- Proteinuria
- Severe renal impairment (creatinine > 250 μmol/L, 2.83 mg/dL)
- Renal replacement therapy:
  Continuous ambulatory peritoneal dialysis (CAPD)
  Haemodialysis
  Renal transplant.

Cardiovascular

- Angina
- Heart failure

- Myocardial infarction
- Coronary artery angioplasty
- Coronary artery bypass.

Cerebrovascular
- Transient ischaemic attack
- Stroke.

Past medical history
- Serious illness (e.g. cancer, rheumatoid arthritis, etc.)
- Accidents
- Injuries
- Hospital admissions
- Operations.

Drug history
- Present medication
- Known allergies.

Family history
- Diabetes
- Other serious illness
- Cause of death of near relatives.

Psychosocial history
- Occupation
- Number of cigarettes smoked per day
- Number of units of alcohol per day
- Psychiatric illness
- Home circumstances:
  Type of accommodation
  Lives alone
  Lives with friends or relatives.

Examination

There is a need for sensitivity on the part of the examiner. Many patients will be fearful and anxious at their first visit. If, rarely, they have ischaemia but no neuropathy, or they have a severely infected foot, then they will be afraid that the examination will be painful. Other patients may be embarrassed about their feet, or may have very sensitive and ticklish feet. Before the feet are handled the patient should be reassured that the examination will not be painful and that everything will be explained. The feet should be grasped gently but firmly, and poking, prodding and tickling should be avoided. The toes should be separated gently: if they are pulled apart violently the skin may split.

The examination should be performed systematically. It consists of five parts:

- Inspection
- Palpation
- Neurological assessment
- Footwear assessment
- General examination.

Inspection
The feet should be fully examined in a systematic fashion: first the right and then the left, including dorsum, sole, medial border, lateral border, back of the heel, malleoli and interdigital areas, with a full assessment of the following:
- Skin
- Callus
- Nails
- Swelling
- Deformity
- Limited joint mobility
- Colour
- Necrosis.

Skin
The general features of the skin should be assessed, especially looking for signs of skin breakdown.

In the neuropathic foot, the skin is dry and fissured and prominent dilated veins secondary to autonomic neuropathy may be visible. Hair loss can be a sign of neuropathy as well as ischaemia. Atrophy of the subcutaneous layer with a thin, shiny, wrinkled skin may indicate ischaemia.

The classical sign of skin breakdown is the foot ulcer. Ulcer assessment is described in Chapter 4. Abrasions, bullae and fissures also represent breakdown of the skin. Bullae are often the first sign of skin breakdown in the ischaemic foot. They are also a feature of fungal skin infections (tinea pedis), as is webspace maceration. Dry skin around the heel will form deep fissures unless an emollient is applied regularly (Fig. 1.1).

Look for other skin lesions, on the leg as well as the foot, including:
- Necrobiosis lipoidica diabeticorum
- Shin spots (diabetic dermopathy).

Necrobiosis lipoidica diabeticorum (NLD) is characterized as well-circumscribed red papules that extend radially with waxy atrophic telangiectatic centres (Fig. 1.2a,b). NLD evolves to ulceration in about one-third of cases.

The round or oval macular hyperpigmented lesions of diabetic dermopathy are found in the anterior tibial region.

As well as skin lesions specific to the diabetic foot, it is important to recognize inflammatory skin disease such as psoriasis, eczema and dermatitis, which also occur in

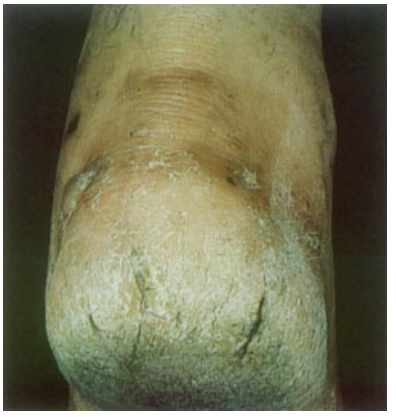


Fig. 1.1 Fissures are a portal of entry for infection and can lead to severe ulceration.

non-diabetic patients but may complicate the diabetic foot and leg.

Corns and callus

These are thickened areas of keratosis which develop at sites of high pressure and friction (Fig. 1.3). Corns are discrete areas, usually not more than 1 cm in diameter, and can extend to a depth of several millimetres. Callus forms diffuse plaques. Neither should be allowed to become excessive as this can be a forerunner of ulceration (usually in the presence of neuropathy). Haemorrhage within callus is an important precursor of ulceration.

Nails

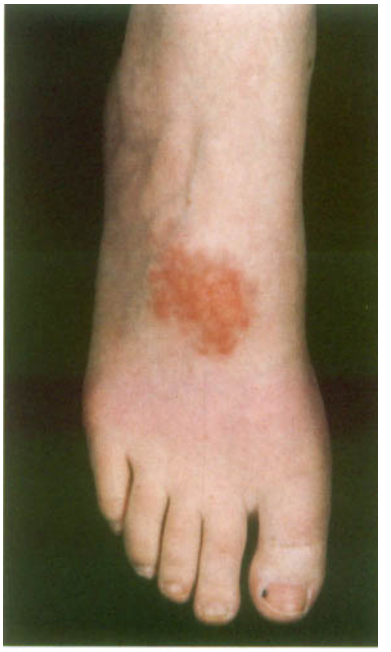
It is important to inspect the nails closely as the nail bed and periungual tissues may become the site of ulceration. The following should be assessed:

- Structure of the nails
- Colour of the nail bed
- Abnormalities under the nail
- Signs of nail infections.

Structure of the nails

Thickened nails are common in the population at large. If the shoes press on the nails they may cause bleeding under the nail. Eventually this may lead to ulceration. Atrophic nails may be present in patients with neuropathy and ischaemia.

Ingrowing toe nail (onychocryptosis) arises when the nail plate is excessively wide and thin, or develops a convex deformity, putting pressure on the tissues at the nail edge. Callus builds up in response to pressure and inflammation. Eventually, usually after incorrect nail cutting or trauma, the nail penetrates the flesh.



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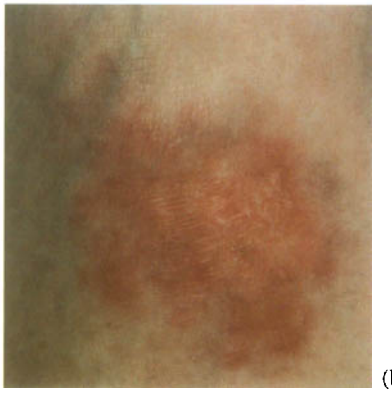


(b)

Fig. 1.2 (a) Necrobiosis lipoidica diabeticorum (NLD) on dorsum of foot and (b) close up of NLD.

Colour of nail bed

Red, brown or black discolouration of the nails may indicate subungual haematoma. The cause may be acute trauma or chronic trauma such as pressure from ill-fitting shoes (Fig. 1.4a).

In acute ischaemia the nail beds are very pale (Fig. 1.4b).

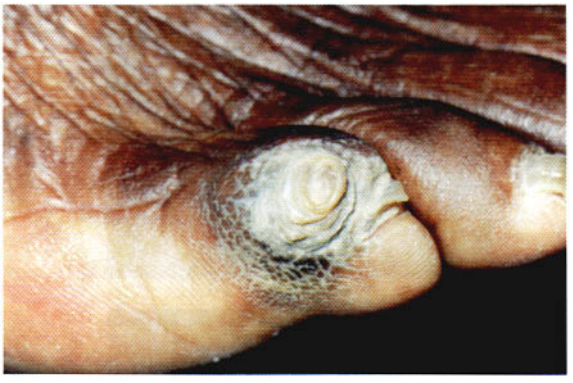
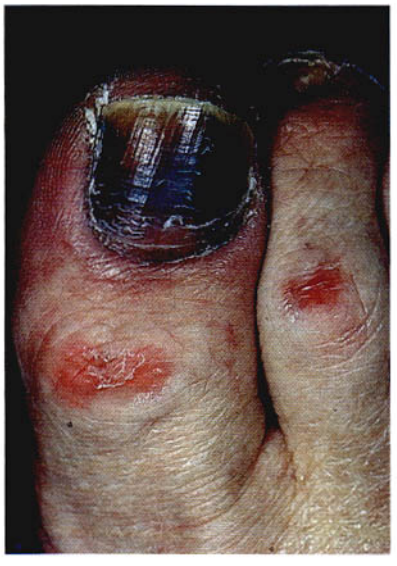


Fig. 1.3 Corn on the 5th toe.



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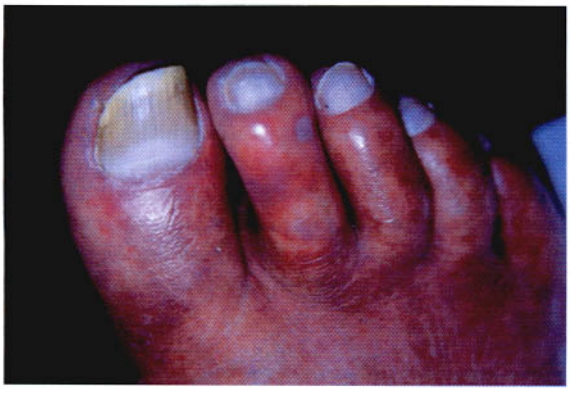


(b)

Fig. 1.4 (a) Subungual haematoma and red marks on toes resulting from wearing tight shoes. (b) Acute ischaemia—pale nail beds.

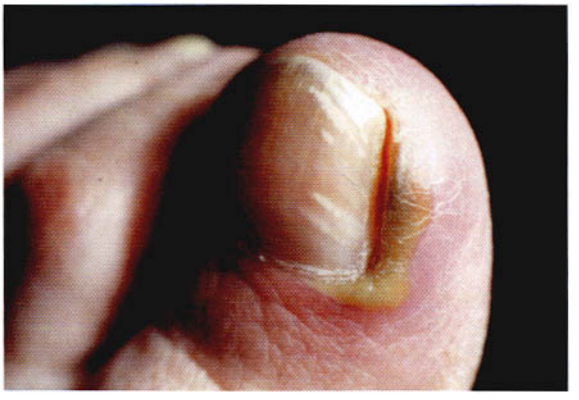


Fig. 1.5 Pressure on the sulcus from a convex nail has resulted in inflammation with secondary infection.

Abnormalities under the nail

Discharge of fluid from beneath or around the nail, and any maceration or softness of the nail plate, may indicate the presence of a subungual ulcer or infection.

Nail infections

Fungal infection of the nail usually invades the nail plate dorsally causing onycholysis. The hallux is the most common nail affected. Infection starts in one corner and over a period of years it spreads to involve the entire toe nail and may affect other nails.

Paronychia is associated with a nail that has a convex nail bed with tendency to incurve in the corners. Repetitive pressure in the insensitive foot can cause repetitive microtrauma in the nail groove, causing the nail to act as a foreign body creating a foreign body inflammatory response with secondary inflammation and localized infection (Fig. 1.5).

Swelling

Swelling of the foot is a major factor predisposing to ulceration, and often exacerbates a tight fit inside poorly fitting shoes. It also impedes healing of established ulcers. Swelling may be bilateral or unilateral. It may involve the foot or be limited to the toes.

Causes of bilateral foot swelling include:

- Cardiac failure
- Renal impairment secondary to diabetic nephropathy
- Chronic venous insufficiency (sometimes unilateral)
- Rarely, neuropathic oedema secondary to diabetic neuropathy, when it is related to increased arterial blood flow and arteriovenous shunting
- Primary lymphoedema

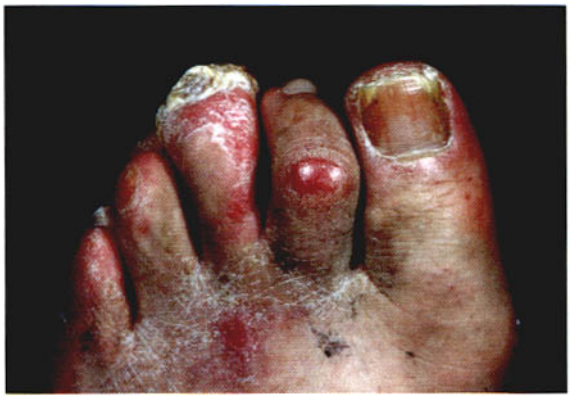


Fig. 1.6 Gout with tophi on second toe.

- Severe ischaemia associated with dependency (often unilateral).

Causes of unilateral foot swelling are usually associated with local pathology in the foot or leg. These include:

- Infection, when it is usually associated with erythema and a break in the skin
- Charcot foot (a unilateral hot, red, swollen foot; sometimes the swelling can extend to the knee)
- Gout, which may also present as a hot, red, swollen foot
- Trauma, fracture, muscle or tendon rupture, often associated with bruising
- Deep vein thrombosis
- Venous insufficiency
- Secondary lymphoedema commonly due to malignancy
- Common peroneal nerve palsy
- Localized collection of blood or pus in the foot, which may present as a fluctuant swelling
- Revascularization of a limb.

Swelling of the toe can be due to:

- Trauma
- Fracture
- Soft tissue infection
- Osteomyelitis
- Gout (Fig. 1.6)
- Charcot toe.

Deformity

Common deformities include:

- Pes cavus
- Fibrofatty padding depletion (FFPD)
- Hammer toes
- Claw toes
- Hallux valgus
- Charcot foot
- Deformities related to previous trauma and surgery.

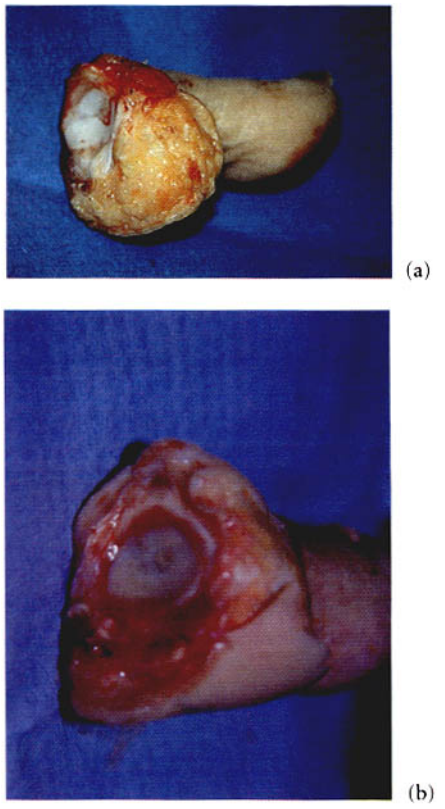


Fig. 1.7 (a) Amputation specimen from 29-year-old non-diabetic patient showing thick fibrofatty padding. (b) Amputation specimen from 29-year-old diabetic patient with history of neuropathy and ulceration showing great reduction in fibrofatty padding.

Pes cavus

Normally the dorsum of the foot is domed due to the medial longitudinal arch, which extends between the first metatarsal head and the calcaneus. When it is abnormally high, the deformity is called pes cavus and leads to reduction of the area of the foot in contact with the ground during walking. Resulting abnormal distribution of pressure leads to excessive callus formation under the metatarsal heads. This deformity is a sign of a motor neuropathy but may be idiopathic. It is often associated with clawing of lesser toes or a trigger first toe.

Fibrofatty padding depletion (FFPD)

A common complication is reduction of the thickness of the fibrofatty padding over the metatarsal heads (Fig. 1.7a,b).

Normal feet contain cushions of fibrofatty padding over the metatarsal heads which absorb plantar pressures.

In diabetic neuropathy the fibrofatty padding may be pushed forward or depleted by previous ulceration, rendering the plantar metatarsophalangeal area prone to ulceration.

Hammer toe
A hammer toe is a flexible or rigid deformity characterized by buckling of the toe. The toe takes on the configuration of a swan's neck. In people with diabetic neuropathy, hammer toes are commonly caused by weakness of the small intrinsic muscles (interossei and lumbricals) of the foot, which can no longer stabilize the toes on the ground. Muscle imbalance results in the affected toes sitting slightly back and up on the metatarsal head. This deformity results in increased pressure over the metatarsal head, over the prominent interphalangeal joint and at the tip of the toe.

Claw toes
Claw toes are similar to hammer toes, but with more buckling and greater deformity. There is fixed flexion deformity at the interphalangeal joint, associated with callus and ulceration of the apex and dorsal aspect of the interphalangeal joint. Although claw toes may be related to neuropathy, they are often unrelated, especially when the clawing is unilateral and associated with trauma or surgery of the forefoot. Claw toes may rarely result from acute rupture of the plantar fascia.

Hallux valgus
Hallux valgus is a deformity of the first metatarsophalangeal joint with lateral deviation of the hallux and a medial prominence on the margin of the foot. This site is particularly vulnerable in the neuroischaemic foot and frequently breaks down under pressure from a tight shoe.

Charcot foot
Bone and joint damage in the tarsometatarsal joints and mid-tarsal joints leads to two classical deformities: the rockerbottom deformity, in which there is displacement and subluxation of the tarsus downwards, and the medial convexity, which results from displacement of the talonavicular joint or from tarsometatarsal dislocation. Both are often associated with a bony prominence which is very prone to ulceration and healing is notoriously difficult.

When the ankle and subtalar joints are involved, instability of the hindfoot can result.

Deformities related to previous trauma and surgery
Deformities of the hip and fractures of the tibia or fibula lead to shortening of the leg and abnormal gait, which predisposes to foot ulceration. Ray amputations remove the toe together with part of the metatarsal. They are usually very successful but disturb the biomechanics of the foot leading to high pressure under the adjacent metatarsal heads. After amputation of a toe, deformities are often seen in adjoining toes.

Limited joint mobility (including hallux rigidus)
Limited joint mobility can affect the feet as well as the hands. The range of motion is diminished at the subtalar and first metatarsophalangeal joints. Limited joint mobility of the first metatarsophalangeal joint results in loss of dorsiflexion and excessive forces on the plantar surface of the first toe, causing callus formation and ulceration. It is commonly seen in barefooted and sandal-wearing populations.

Colour
It is important to observe the colour of the foot including the toes. Colour changes may be localized or diffuse. Common colour changes are red, blue, white or black.

Causes of the red foot
- Cellulitis
- Critical ischaemia, especially on dependency (dependent rubor)
- Charcot foot
- Gout
- Burn or scald.

Causes of the red toe
- Cellulitis
- Osteomyelitis
- Ischaemia
- Gout
- Chilblains
- Dermatitis/eczema.

Causes of the blue foot
- Cardiac failure
- Chronic pulmonary disease
- Venous insufficency (often with brownish pigmentation —haemosiderosis).

Causes of the blue toe
- Severe infection
- Ischaemia.

The foot may have a pale white appearance in severe ischaemia, especially on elevation. In acute ischaemia, the foot is pale, often with purplish mottling. The cause of black appearances are discussed under necrosis.

Necrosis

Areas of necrosis and gangrene can be identified by the presence of black or brown devitalized tissue. Such tissue may be wet (usually related to infection) or dry.

Causes of the black toe

- Severe chronic ischaemia
- Acute ischaemia
- Emboli
- Bruise
- Blood blister
- Shoe dye
- Application of henna
- Tumour (melanoma).

Palpation

Palpation should take place to assess:

- Pulses
- Temperature of the foot
- Oedema
- Crepitus.

Pulses

The most important manoeuvre to detect ischaemia is the palpation of foot pulses, an examination which is often undervalued.

- The dorsalis pedis pulse is palpated, using the index, middle and ring fingertips together, lateral to the extensor hallucis longus tendon on the dorsum of the foot (Fig. 1.8)
- The posterior tibial pulse is palpated below and behind the medial malleolus (Fig. 1.9).

If either of these foot pulses can be felt then it is highly unlikely that there is significant ischaemia in the foot.

If both pulses are absent, a full examination should include palpation for popliteal and femoral pulses.

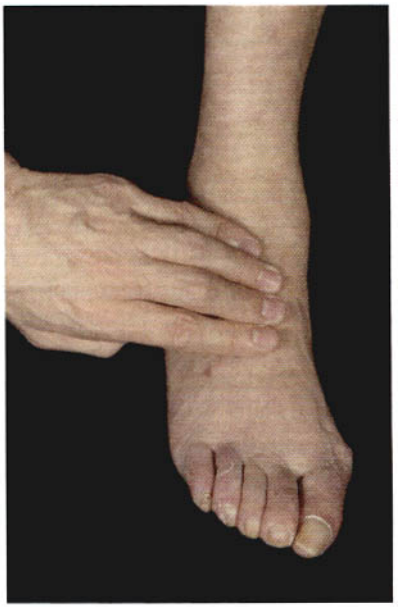


Fig. 1.8 Palpation of the dorsalis pedis pulse.

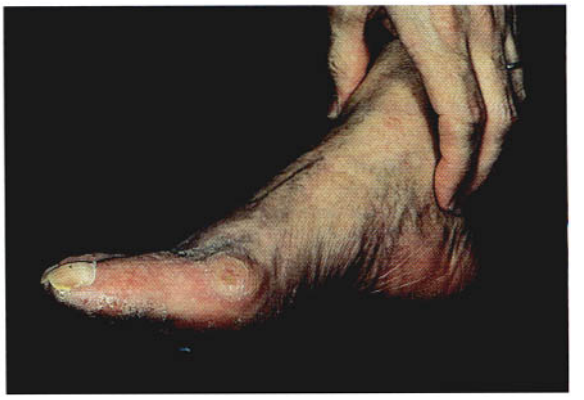


Fig. 1.9 Palpating the posterior tibial pulse.

Temperature of the foot

Skin temperature is compared between both feet with the back of the examining hand. Warm areas or hot spots indicate inflammation which may be due to infection, fracture, Charcot's osteoarthropathy or soft tissue trauma. Unilateral pedal temperature increase, especially in the absence of ulceration, is best presumed to be Charcot's osteoarthropathy.

The temperature gradient is checked by using the back of the hands and gently moving them from the pretibial region of the leg distally over the dorsum of the foot to the toes while keeping in contact with the patient's skin. An asymmetric gradient may indicate either unilateral ischaemia on the colder side or unilateral inflammatory response such as Charcot's osteoarthropathy or infection on the warmer side.

In the neuroischaemic foot, coexisting autonomic neuropathy may keep the foot relatively warm, although an ice-cold foot is indicative of acute ischaemia.

Causes of the hot foot

- Cellulitis
- Charcot foot
- Gout
- Venous insufficiency
- Deep vein thrombosis.

Causes of the cold foot
- Chronic ischaemia
- Acute ischaemia
- Cardiac failure.

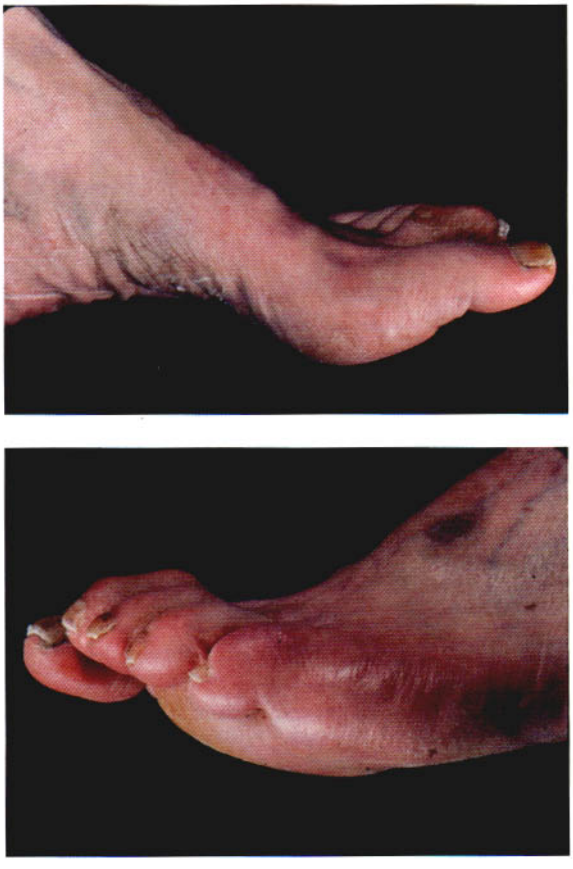
Oedema
Oedema already suspected on inspection can be confirmed by gentle digital pressure applied for a few seconds.

Crepitus
Very occasionally palpation may reveal gas in tissues as a fine crackling sensation.

Neurological assessment
Simple inspection will usually reveal signs of motor and autonomic neuropathy but sensory neuropathy must be detected by a sensory screening test or a simple sensory examination.

Motor neuropathy
The classical sign of a motor neuropathy is a high medial longitudinal arch, leading to prominent metatarsal heads and pressure points over the plantar forefoot (Fig. 1.10a,b).

Complicated assessment of motor power in the foot or leg is not usually necessary, but it is advisable to test dorsiflexion of the foot to detect a foot drop secondary to a common peroneal nerve palsy, which is usually unilateral and will affect the patient's gait. If painful mononeuropathy is suspected from the history, a more detailed neurological examination is indicated to rule out compressive lesions of nerve roots supplying the lower limb—see under Painful neuropathy in Chapter 3.


(a)

(b)

Fig. 1.10 (a) Neuropathic foot with high medial longitudinal arch. In severe cases, pressure points develop over the apices and dorsal interphalangeal joints of claw toes. (b) Claw toes in neuropathic foot.

Autonomic neuropathy
Signs of an autonomic neuropathy include a dry skin with fissuring and distended veins over the dorsum of the foot.

The dry skin is secondary to decreased sweating. The sweating loss normally occurs in a stocking distribution, which can extend up to the knee. The distended veins are secondary to arteriovenous shunting associated with autonomic neuropathy (Fig. 1.11).

Sensory neuropathy
An important indication of neuropathy will be a patient who has no pain even when significant foot lesions are present. Painless ulceration is definite evidence of a peripheral neuropathy. It is important to detect patients who have sufficient neuropathy to render them susceptible to foot ulceration. This can be carried out using a monofilament which, when applied perpendicular to the foot, buckles at a given force of 10 g. Ability to feel that level of pressure provides protective sensation against foot ulceration. It is helpful first to demonstrate the technique on the patient's forearm.

The number of sites used varies according to different protocols. Sites examined include the plantar aspects of the first toe, the first, third and fifth metatarsal heads, the plantar surface of the heel and the dorsum of the foot. The filament should not be applied at any site until callus has been removed. If the patient cannot feel the filament at any of the tested areas, then protective pain sensation is lost, indicating susceptibility to foot ulceration (Fig. 1.12). The 10-g monofilament may become overstrained and inaccurate after use on numerous occasions and should be replaced regularly. A recent study has assessed differences in the performance of commercially available 10-g

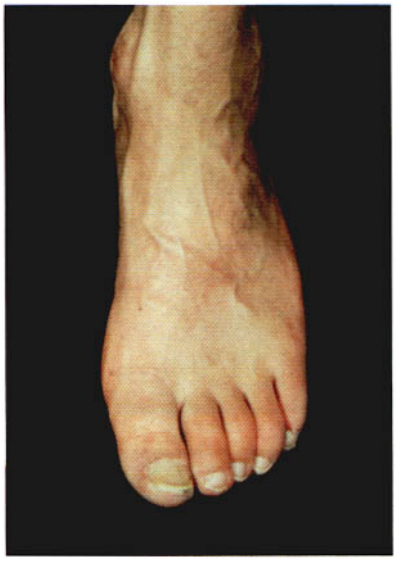


Fig. 1.11 Distended veins secondary to autonomic neuropathy.

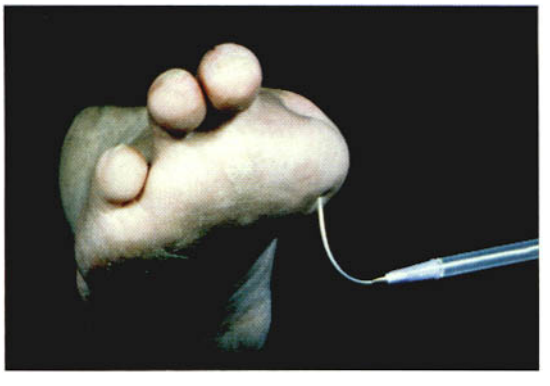


Fig. 1.12 A monofilament is applied perpendicular to the foot and pressed until it buckles at a given force of 10 g.

monofilaments. Monofilaments were tested using a calibrated load cell. Each monofilament was subjected to 10 mechanical bucklings of 10 mm while the load cell detected the maximal buckling force. Longevity testing was performed on a subset of the monofilaments by subjecting them to continuous compression until the buckling force was less than 9 g. Longevity and recovery testing suggest that each monofilament would survive usage on 10 patients before needing a recovery time of 24 h before further usage.

If filaments are not available, then a simple clinical examination detecting sensation to light touch using a cotton wisp and vibration using a 128-Hz tuning fork will suffice. It is best to compare a proximal site with a distal site to confirm a symmetrical stocking-like distribution of the neuropathy, and to avoid the use of 'pin-prick' to detect sensory loss.

Other useful but simple and practical tests for detecting neuropathy, if health-care practitioners have no access to formal equipment, include the Achilles tendon pinch, and the application of vertical pressure onto the nail plate. In practice, any patient who walks on a foot with ulceration or heavy plantar callus without concern has significant neuropathy.

Footwear assessment

It is important to examine both shoes and socks.

Examination of patient's footwear

- Is the shoe long enough?
- Is the toe box broad and deep enough?
- Are the heels low?
- Does the shoe fasten with a lace or strap?

Slip-ons are unsuitable for everyday wear.

- Is the sole thick enough to provide protection from puncture wounds?
- Is the shoe lining worn, with rough areas that may prove irritating and warrant replacement?
- Are there foreign bodies within the shoes?
- Is there excessive wear under hallux suggesting a hallux rigidus?
- Is there wear across whole of tread suggesting pes cavus?

Examination of patient's socks

- Are the socks large enough?
- Are the seams too prominent?
- Is there a tight band at the top?
- Are the socks in good repair—no holes or lumpy darns?
- Are the socks made of absorbent material?
- Are the socks very thick, taking up too much space in the shoe?

General examination

As part of the diabetic foot assessment and indeed the diabetic assessment all patients should have a physical examination including the following systems:

- Cardiovascular
- Respiratory
- Abdomen
- Eyes:
  Visual acuity
  Fundi.

(A patient lacking necessary visual acuity to give himself a

Fig. 1.13 The neurothesiometer.

daily foot examination is a patient at risk, and his family or caregiver should help him.)

Investigations

Investigations include:

- Neurological
- Vascular
- Skin temperature
- Laboratory
- Radiological
- Foot pressures.

Neurological

The degree of neuropathy can be quantified by the use of the biothesiometer or the more recently available neurothesiometer (Fig. 1.13). Both are devices which, when applied to the foot, deliver a vibratory stimulus, which increases as the voltage is raised. The vibration threshold increases with age, and for practical purposes, any patient unable to feel a vibratory stimulus of 25 volts is at risk of ulceration.

A small number of patients have a small-fibre neuropathy with impaired pain and temperature perception but with intact touch and vibration. They are prone to ulceration and thermal traumas but test normally with filaments and biothesiometer, and a clinical assessment of light touch and vibration is normal. As yet, there is no simple inexpensive method of detecting and quantifying small-fibre neuropathy. However, a simple temperature assessment of cold sensation can be made by placing a cold tuning fork on the patient's foot and leg.

Vascular

A small hand-held Doppler can be used to quantify the vascular status.

Used together with a sphygmomanometer, the brachial systolic pressure and ankle systolic pressure can be measured, and the pressure index, which is the ratio of ankle systolic pressure to brachial systolic pressure, can be calculated. In normal subjects, the pressure index is usually > 1, but in the presence of ischaemia is < 1. Thus, absent pulses and a pressure index of < 1 confirms ischaemia. Conversely, the presence of pulses and a pressure index of > 1 rules out ischaemia and this has important implications for management, namely that macrovascular disease is not an important factor and further vascular investigations are not necessary.

Many diabetic patients have medial arterial calcification, giving an artificially elevated systolic pressure, even in the presence of ischaemia. It is thus difficult to assess the diabetic foot when the pulses are not palpable, but the pressure index is > 1. There are two explanations.

- The examiner may have missed the pulses, particularly in an oedematous foot, and should go back to palpate the foot after the arteries have been located by Doppler ultrasound
- If the pulses remain impalpable, then ischaemia probably exists in the presence of medial wall calcification. It is then necessary to use other methods to assess flow in the arteries of the foot, such as examining the pattern of the Doppler arterial waveform or measuring transcutaneous oxygen tension or toe systolic pressures. Furthermore, absence of foot pulses would be an indication to investigate popliteal and femoral arteries.

Skin temperature

It is helpful to follow-up the clinical assessment of skin temperature with the use of a digital skin thermometer. An infrared thermometer is ideal and skin temperatures are compared between similar areas on each foot (Fig. 1.14). This is particularly helpful in the management of the Charcot foot.

Laboratory

Laboratory investigations are determined by clinical findings, but the following investigations are useful as a baseline in most patients:

- Full blood count (to detect anaemia or polycythaemia)
- Serum electrolytes, urea and creatinine (to assess baseline renal function)
- Serum bilirubin, alkaline phosphatase, gamma glutamyl transferase, aspartate transaminase (to assess baseline liver function)

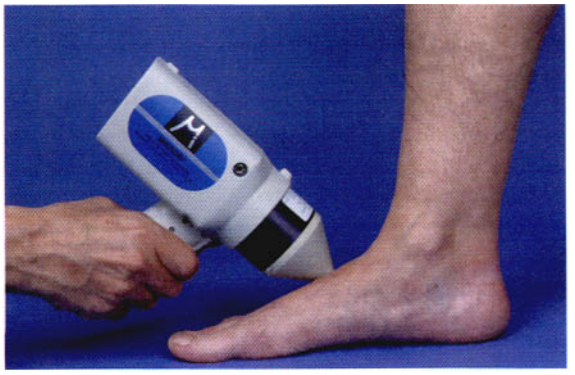


Fig. 1.14 Digital skin thermometer.

- Blood glucose and HbA_{1c} (to assess diabetic control)
- Serum cholesterol and triglycerides (to assess arterial disease risk factors).

Radiological

These will be determined by the clinical presentation, and may not always be necessary. However, in most cases, an X-ray of the foot will be required to detect:

- Osteomyelitis
- Fracture
- Charcot foot
- Gas in soft tissues
- Foreign body.

Foot pressures

These techniques measure the pressure distribution on the plantar surface of the foot. There are two main methods: 'out-of-shoe'. and 'in-shoe'. The introduction of the optical pedobarograph considerably improved the accuracy of out-of-shoe pressure measurements. Developments in computer technology have led to microprocessor-like recording devices to quantify in-shoe foot pressures and these include the EMED system and the F Scan system. These systems have the possibility of identifying patients at risk of plantar neuropathic ulceration and give a basis for the implementation of footwear adjustments or surgical intervention. We have found that they are also useful educational tools as described in Chapter 3.

CLASSIFICATION AND STAGING

After completing this basic assessment, it will now be possible to classify the diabetic foot. For practical purposes, the diabetic foot can be divided into two distinct entities: the neuropathic foot and the neuroischaemic foot. Neuropathy is nearly always found in association with ischaemia, so the ischaemic foot is best called the neuroischaemic foot. In rare cases the foot may clinically be ischaemic without signs of neuropathy, but in practice, the diabetic ischaemic foot is treated in the same way as the neuroischaemic foot, and thus, we have continued with the two main divisions.

It is essential to classify the diabetic foot by differentiating between the neuropathic and the neuroischaemic foot as their management will differ in many respects. Usually there will be no doubt as to which category the foot should be placed in. However, if the examiner has any doubt as to the correct classification, then the foot should be regarded as neuroischaemic, because if a neuroischaemic foot is wrongly classified as neuropathic, with resulting failure to do further tests to confirm ischaemia and adapt the care plan accordingly, this may lead to preventable catastrophe and loss of the foot.

Neuropathic foot

- The neuropathic foot is a warm, well-perfused foot with bounding pulses and distended dorsal veins due to arteriovenous shunting
- Sweating is diminished so skin and any callus tend to be hard and dry and prone to fissuring
- Toes are flexed and the arch of the foot may be raised
- Ulceration commonly develops on the sole of the foot, associated with neglected callus and high plantar pressures
- Despite the good circulation, necrosis can develop secondary to severe infection
- The neuropathic foot is also prone to bone and joint problems which we refer to as Charcot's osteoarthropathy.

Neuroischaemic foot

- The neuroischaemic foot is a cool, pulseless foot with poor perfusion and almost invariably also has neuropathy
- The colour of the severely ischaemic foot can be a deceptively healthy pink or red caused by dilatation of capillaries in an attempt to improve perfusion
- The neuroischaemic foot may be complicated by swelling, often secondary to cardiac failure or renal impairment
- Ischaemic ulcers are commonly seen around the edges of the foot, including the apices of the toes and the back of the heel, and are associated with trauma or wearing unsuitable shoes (Fig. 1.15)

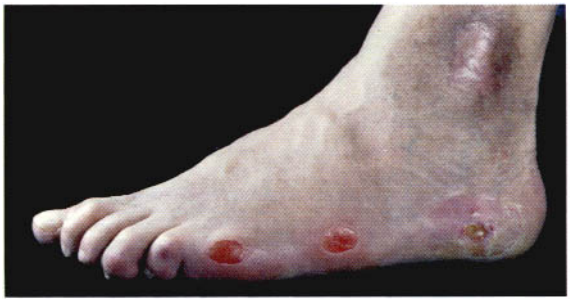


Fig. 1.15 Neuroischaemic foot with ulceration on the margins..

- The neuroischaemic foot develops necrosis in the presence of infection or if tissue perfusion is critically diminished
- Even if neuropathy is present and plantar pressures are high, plantar ulceration is rare. This is probably because the foot does not develop heavy callus, which requires good blood flow.

After classification of the diabetic foot, it is necessary to make the appropriate staging in its natural history.

The natural history of the diabetic foot can be divided into six stages as shown:

- Stage l: Normal foot
- Stage 2: High-risk foot
- Stage 3: Ulcerated foot
- Stage 4: Infected foot
- Stage 5: Necrotic foot
- Stage 6: Unsalvageable foot.

The simple staging system covers the entire spectrum of diabetic foot disease but it emphasizes the development of the foot ulcer in stage 3 as a pivotal event demanding urgent and aggressive management. However, each stage demands specific treatment.

Other classifications of the diabetic foot such as the Wagner system, the University of Texas system and the Nottingham S(AD)SAD system are essentially classifications of ulcers and do not cover the whole natural history of the diabetic foot.

The simple staging system used in this book has been created to allow all practitioners, whether experienced in diabetic foot care or not, to make an initial assessment of the diabetic foot at whatever stage in the natural history it might be. The stage sets the place in the natural history and also determines treatment. The aim is to keep all diabetic feet at as low a stage as possible.

Stage 1

At this stage, the patient does not have the risk factors of neuropathy, ischaemia, deformity, callus and swelling rendering him vulnerable to foot ulcers. The normal foot is characterized by no symptoms, including no pain, and examination is normal.

Stage 2

The patient has developed one or more of the risk factors for foot ulceration including neuropathy, ischaemia, deformity, callus and swelling. These risk factors need addressing to reduce susceptibility to ulceration.

Patients without active foot ulceration but a history of ulceration should be regarded as very high risk.

Within stage 2 there are specific conditions which are non-ulcerative but require treatment. These include:

- Severe chronic ischaemia
- Acute ischaemia.

There are also specific complications of neuropathy:

- Neuropathic fractures
- Charcot's osteoarthropathy
- Painful neuropathy.

Stage 3

The foot has a skin breakdown. Although this is usually an ulcer, it is important not to underestimate some apparently minor injuries such as blisters, skin fissures or grazes, all of which have a propensity to become ulcers if they are not treated correctly and fail to heal quickly. Ulceration is usually on the plantar surface in the neuropathic foot (Fig. 1.16) and on the margin in the neuroischaemic foot (Fig. 1.17).

Stage 4

The foot has developed infection with the presence of purulent discharge or cellulitis which can complicate both the neuropathic foot (Fig. 1.18) and the neuroischaemic foot (Fig. 1.19).

Stage 5

Necrosis has supervened. In the neuropathic foot, infection is usually the cause. In the neuroischaemic foot, infection is still the most common reason for tissue destruction although ischaemia contributes (Fig. 1.20). In some cases ischaemia alone can lead to necrosis of a previously intact foot, with slow onset of dry necrosis and necrotic toes which appear shrivelled (Fig. 1.21).

Stage 6

The foot cannot be saved and will need a major amputation.

Reasons for major amputation:

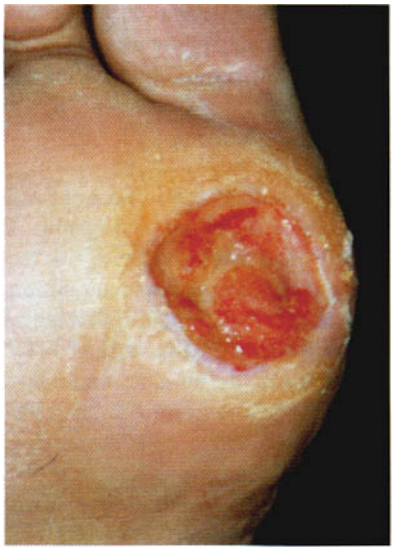


Fig. 1.16 Neuropathic ulcer on the plantar surface.

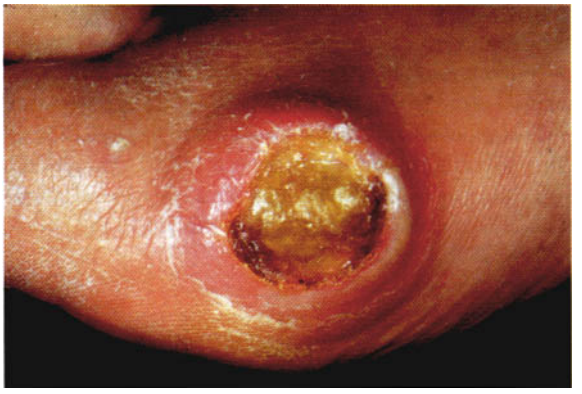


Fig. 1.17 Ischaemic ulcer on the medial border of the first metatarsophalangeal joint.

- Extensive necrosis which has destroyed the foot
- Severe infection which puts the life at risk
- Agonizing ischaemic pain which cannot be relieved.

MULTIDISCIPLINARY MANAGEMENT

The aim in managing diabetic foot problems is always to keep the patient at as low a stage as possible. At each stage of the diabetic foot it is necessary to take control of the foot to prevent further progression and management will be considered under the headings shown in Table 1.3.

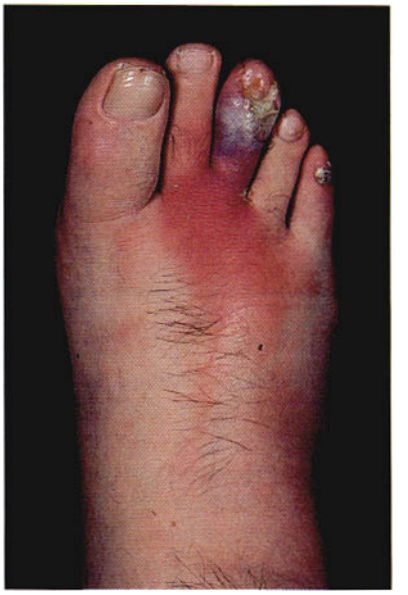


Fig. 1.18 Severe sepsis in a neuropathic foot with oedema, cellulitis, lymphangitis and a bluish tinge to the 3rd toe. Without urgent treatment, necrosis will supervene.

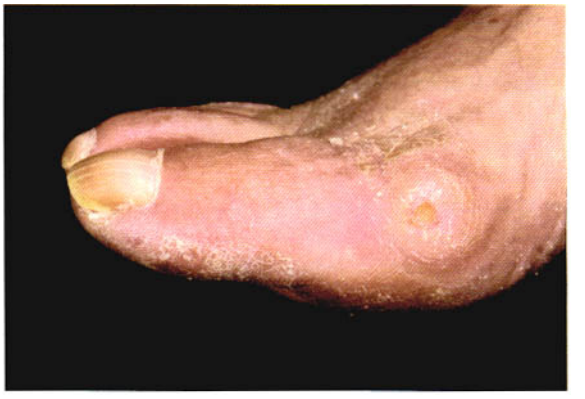


Fig. 1.19 Cellulitis in a neuroischaemic foot with ulcer that is a portal of entry for infection.

Table 1.3 Multidisciplinary management

Mechanical control
Wound control
Microbiological control
Vascular control
Metabolic control
Educational control

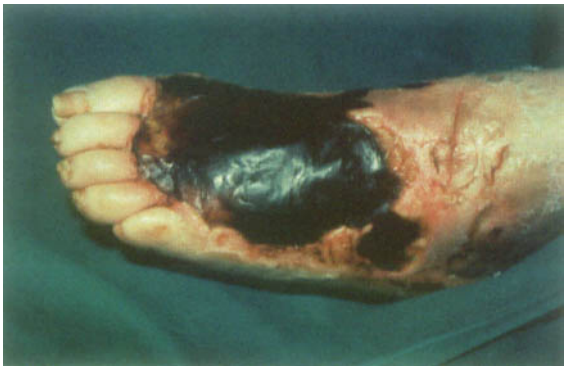


Fig. 1.20 Infection leading to necrosis on the dorsum of the neuroischaemic foot.

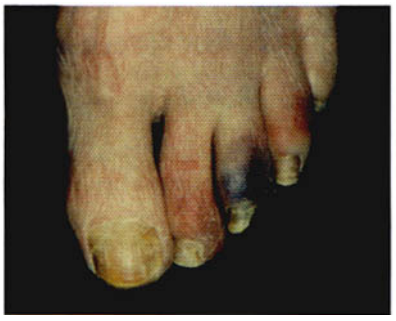


Fig. 1.21 Dry necrosis in a neuroischaemic foot.

When the examination reveals a foot at stage 3, 4 or 5 there is a need for a great sense of urgency: treatment should begin without delay. No one person can take control of the diabetic foot. Successful management needs the expertise of a multidisciplinary team including the following:

- Podiatrist
- Physician
- Nurse
- Orthotist
- Radiologist
- Surgeon.

It is helpful if the multidisciplinary team works closely together, within the focus of a diabetic foot clinic, which ideally is situated in a hospital. The team should meet regularly for ward rounds and X-ray conferences. Each team member should be available quickly in an emergency. Some roles may overlap, depending on local expertise and interest. Patients in stage 3–5 are best seen in the multidisciplinary foot service, which takes early referrals from a primary care service. Patients in stages 1 and 2 can be seen in primary care: however, there should be very rapid referral pathways between the primary care service and the hospital multidisciplinary foot service. There should be defined pathways and timescales for the treatment and follow-up of all patients after the feet have been classified and staged.

- Stage 1—Annual review with basic foot education
- Stage 2—It is difficult to stratify the risk of ulceration within this group. Any patient with one or more of the following—neuropathy, ischaemia, deformity, callus, swelling—should be referred for education and podiatry, receiving 3-monthly or more frequent treatment.
  Patients with specific problems will need the following referrals:
  Severe chronic ischaemia to diabetic foot clinic or vascular clinic within 1 week Acute ischaemia to diabetic foot clinic or vascular clinic same day
  Any neuropathic fracture/Charcot's osteoarthropathy to diabetic foot clinic within 24 h
  Painful neuropathy to diabetic foot clinic within 2 weeks
- Stage 3—to diabetic foot clinic within 1 week. Maximum follow-up period 2 weeks
- Stage 4—to diabetic foot clinic same day (may need admission for intravenous antibiotics or outpatient treatment with oral or intramuscular antibiotics). Maximum follow-up period 1 week
- Stage 5—to diabetic foot clinic same day for admission: after discharge, maximum follow-up period 1 week until any remaining necrosis is dry and well demarcated, then 2-weekly until fully healed
- Stage 6—to diabetic foot clinic same day for admission. Remaining foot should be inspected daily during perioperative and rehabilitation period. After discharge from hospital, should be followed up with maximum interval between treatments of 6 weeks.

PRACTICE POINTS

- The basic approach to the diabetic foot is assessment, classification, staging and multidisciplinary management
- Diabetic feet can be classified into neuropathic and neuroischaemic feet
- The natural history of the diabetic foot falls into six stages: normal, high risk, ulcerated, infected, necrotic and unsalvageable
- Multidisciplinary management consists of mechanical, wound, microbiological, vascular, metabolic and educational control

- The multidisciplinary foot care service should include podiatrist, physician, orthotist, nurse, radiologist and surgeon.

FURTHER READING

Practical assessment

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2 Stage 1: the normal foot

Leave gourmandising; know the grave doth gape
For thee thrice wider than for other men.
(2 Henry IV, *II, v, William Shakespeare)*

PRESENTATION AND DIAGNOSIS

Stage 1 is the normal foot. By definition, it does not have the risk factors for ulceration, namely neuropathy, ischaemia, deformity, callus and swelling. The diagnosis of stage 1 is made by excluding these factors.

A full foot assessment consisting of history, examination and simple investigations is described in the introduction.

Ideally this should be carried out on the first presentation of a patient. The assessment will need to be adapted when it is used for a screening examination as part of the annual review.

Foot screening may be carried out by any suitably trained health-care professional.

The screening assessment consists of four parts:

- Enquiry for past or present ulceration
- Testing for neuropathy with 10-g monofilament
- Palpation of foot pulses to detect ischaemia
- Inspection of the foot to look for the following abnormalities:
  Deformity
  Callus
  Swelling
  Ulceration
  Signs of infection
  Necrosis.

Patients with none of these problems are classified as stage 1. However, the screening of stage 1 patients should be repeated at yearly intervals to ensure that patients who develop risk factors are restaged and offered treatment as appropriate.

MANAGEMENT

The aim of management is to ensure that:

- Patients do not develop risk factors for diabetic foot ulceration
- If risk factors do develop, they are detected early and patients placed in stage 2
- Common foot problems that can occur in the general population are efficiently treated and do not lead to tissue breakdown even in the absence of neuropathy and vascular disease.

The following components of multidisciplinary management are important for stage 1 patients.

Mechanical control

- To encourage the use of suitable footwear, discourage inadequate footwear and thus prevent subsequent deformity and callus formation
- To keep the foot intact by treatment of non-ulcerative pathologies. There is no such thing as a trivial lesion of the diabetic foot; all foot problems need early diagnosis and appropriate intervention.

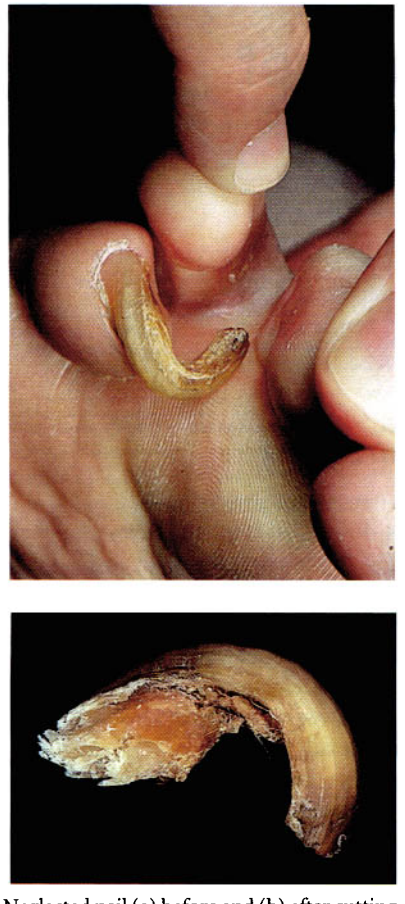
Metabolic control

To prevent or delay the development of neuropathy, microvascular and macrovascular complications

Educational control

- To encourage healthy foot care/footwear habits and detect ignorance or non-compliance early
- To make provision for intellectual deficit and psychological and social problems. Behaviour modification is an important component of care (Fig. 2.1a,b).

Because stage 1 patients have no ulcers, infection, gangrene or ischaemia, there is no need for wound, microbiological or vascular control to be addressed.



(a)

(b)

Fig. 2.1 Neglected nail (a) before and (b) after cutting.

Mechanical control

Mechanical control is achieved by wearing the correct footwear and also by the recognition and treatment of common foot problems.

Footwear

Advice on buying shoes

Stage 1 patients may obtain their shoes from shoe shops or mail-order catalogues, though it is probably best for the foot to be measured and the shoes sized and tried on, or bought 'on approval' and checked by a health-care professional. Staff of shoe shops can be taught which footwear is suitable for diabetic feet. Normal feet swell when the patient has been on his feet a lot, so shoes are best bought towards the end of the day.

For everyday wear, house shoes and for when the patient is on his feet a lot, selection should be made according to the following principles (Fig. 2.2):

Fig. 2.2 A high-street man's shoe.

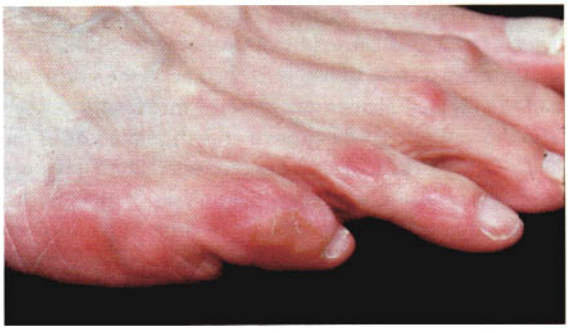
- The shoe should be 'foot-shaped'
- Toe box is roomy to avoid pressure on toes and borders of foot
- Heel cup should fit snugly
- Heels should be low (under 5 cm high)
- Shoe lining should be smooth
- Shoe should fasten with lace or strap to hold foot back in shoe
- Court or slip-on shoes should be avoided except for special occasions
- Trainers are useful if they are sufficiently long, broad and deep, with cushioned soles and a built-in rocker, and are worn with the laces fastened. However, there are many inadequate trainers and deck shoes on sale which have thin soles, a lack of cushioning, and no rocker built in
- Wearing socks reduces friction within shoes
- Socks should be non-constricting with no tight band around ankle or calf
- Socks with prominent seams should be worn inside-out
- Socks should be made of absorbent materials such as cotton
- If shoes cause pain, callus, red marks or blisters then they do not fit properly and should be discarded (Fig. 2.3a,b)
- In hot climates, sandals may be worn: however, they give little protection against trauma and the foot is not held firmly in place, resulting in excessive shear.

Wearing 'good' shoes will prevent or delay the onset of footwear-related deformity, and prevent callus from developing by reducing the mechanical forces applied to the feet.

Features of a bad shoe include:

- Slip on style, which causes pressure on the forefoot
- High heels, which reduce the range of toe dorsiflexion and can lead to hallux rigidus
- Thin-soled shoes, which make the foot sensitive to any unevenness of the ground.

(a)



(b)

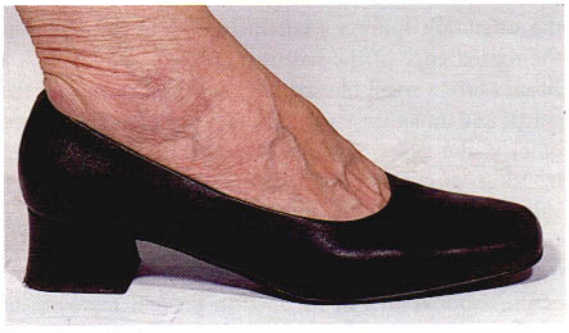


Fig. 2.3 (a) Red marks on toes followed (b) wearing unsuitable shoes with no proper fastening and narrow toe box.

If shoes are the wrong size or wrong style, they can cause permanent damage to the feet, resulting in deformity and callus.

Common foot problems

In maintaining mechanical control, it is important to diagnose foot problems including:

- Nail problems
- Fungal infections
- Fissures
- Verrucae
- Bullae
- Bullosis diabeticorum
- Chilblains
- Malignancy
- Inflammatory skin diseases
- Hyperhydrosis
- Insect bites
- Traumas
- Fractures.

Nail problems

Cutting nails may present problems. However, many people in stage 1 will be able to cut their own nails (Table 2.1).

Table 2.1 Patients who can safely cut their own toe nails

Patients who can safely cut their own toe nails
Have pain-free normal nails with no pathology
Can see feet clearly
Can reach feet
Have been taught correct nail cutting techniques

They should be taught the correct techniques for cutting normal nails as follows:

- Nails should be cut straight across or in a gentle curve
- The corners should not be cut out
- The nail plate should not be cut in one piece: a gentle 'nibbling' technique should be used
- The nail should not be cut so short that the seal between nail and nail bed is broken
- The nails should not be left so long that they can catch on the socks, risking trauma
- The nails should be cut regularly
- The nails should be cut after the bath, when the nail plate will be softer and more flexible and easy to cut
- If nail cutting is difficult or painful, patients should seek professional help.

There is no reason why any health-care professional should not cut normal nails in diabetic stage 1 feet, but proper nail nippers should be provided for staff, who should be taught correct techniques as explained above.

Onychauxis

Onychauxis is thickening of the nail without deformity, and follows an insult to the nail bed. Regular filing will reduce the thickness of the nail. Without regular reduction onychogryphosis will develop.

Onychogryphosis (ram's horn nail)

This is thickening of the nail with deformity. The cause is an insult to the nail bed. Treatment can be palliative or surgical. Palliative treatment consists of regular reduction of excessive thickness of the nail plate at 3-monthly treatment intervals.

If only one nail is affected and the patient dislikes the need for regular treatment, the nail plate can be removed under ring block local anaesthesia. If the exposed nail bed is treated with topical phenol the nail will be replaced by a fibrous plate which does not need regular reduction and has a cosmetically acceptable appearance. However, this procedure is invasive and should not be carried out on ischaemic feet.

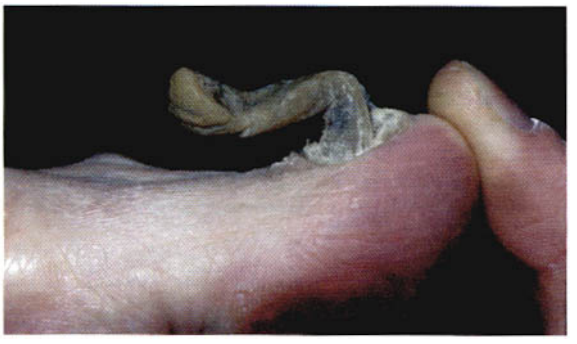


Fig. 2.4 Onychogryphosis.

CASE STUDY

Onychogryphosis treated with palliative care

(Fig. 2.4)

A 73-year-old man with type 2 diabetes of 1 year's duration complained of deformity of his left first toe nail of many years' duration (he had dropped an ammunition box on it). He did not like the appearance of the nail and said that it wore holes in his socks. He was asked to attend the diabetic foot clinic at 3-monthly intervals and the thickened nail was reduced with scalpel and file. He had no further complaints.

Key points

- Cutting and thinning gryphotic nails can avoid surgery in patients who are prepared to attend regularly
- Reduction of thickened nails improves the cosmetic appearance to near-normality.

Onychocryptosis (ingrowing toe nail)

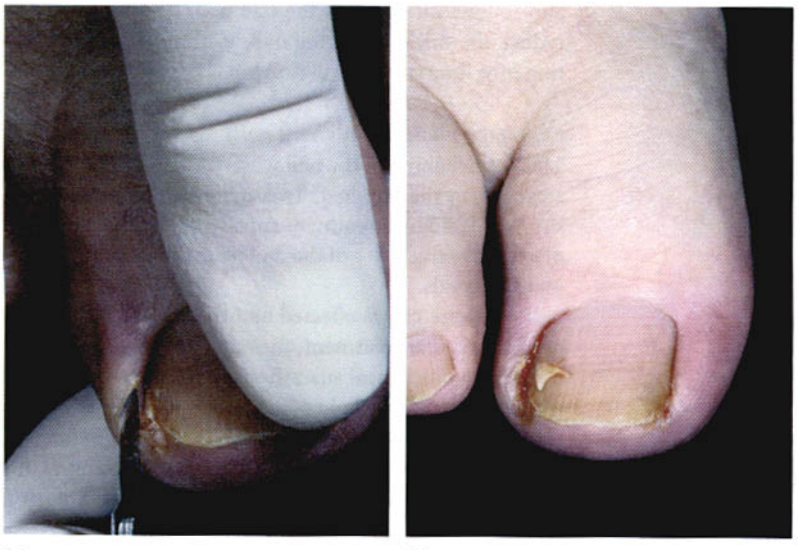
This is frequently caused by improper nail-cutting technique, when a spike of nail is left behind at the side of the nail. As the nail plate grows forward the spike is pushed into the nail sulcus (the groove of flesh at the side of the nail). Other causes of onychocryptosis include pressure on the side of the nail from tight shoes or tight socks, antithrombotic stockings or support hose, or a trauma to the side of the nail, as when the toe is stubbed. Some teenagers with a very thin nail plate and a fleshy nail sulcus are particularly prone to onychocryptosis.

Treatment of onychocryptosis involves removal of the offending splinter (nail spicule) (Fig. 2.5a,b), and the ragged edge of the nail is then filed smooth with a Black's file (a small file specially designed to fit into the sulcus and under the nail). Unless the splinter is removed quickly, the spike of nail will penetrate the flesh, and in these circumstances infection rapidly supervenes. Where onychocryptosis is recurring or chronic it can be treated very successfully with partial nail avulsion under local anaesthetic (without adrenaline). Phenolization of the exposed area of nail bed to prevent regrowth of the troublesome side of the nail prevents recurrence. This procedure should not be performed on ischaemic feet.

CASE STUDY

Acute onychocryptosis

A 26-year-old man with type 1 diabetes of 20 years' duration had acute onychocryptosis (Fig. 2.6a) which had not



(a) (b)

Fig. 2.5 (a) Onychocryptosis: a spike of nail has been left behind after cutting, and (b) the offending spike has been removed and lies on the nail plate.

responded to palliative care and underwent total nail avulsion. His toe healed in 5 weeks (Fig. 2.6b–d).

Key points

- For acute onychocryptosis which does not respond to palliative care, surgery can be a permanent solution
- A partial or total nail avulsion with phenolization can take several weeks to heal.

Involuted toe nail

This is excessive lateral curvature of the nail plate. If epithelial cells become trapped as they are shed in the

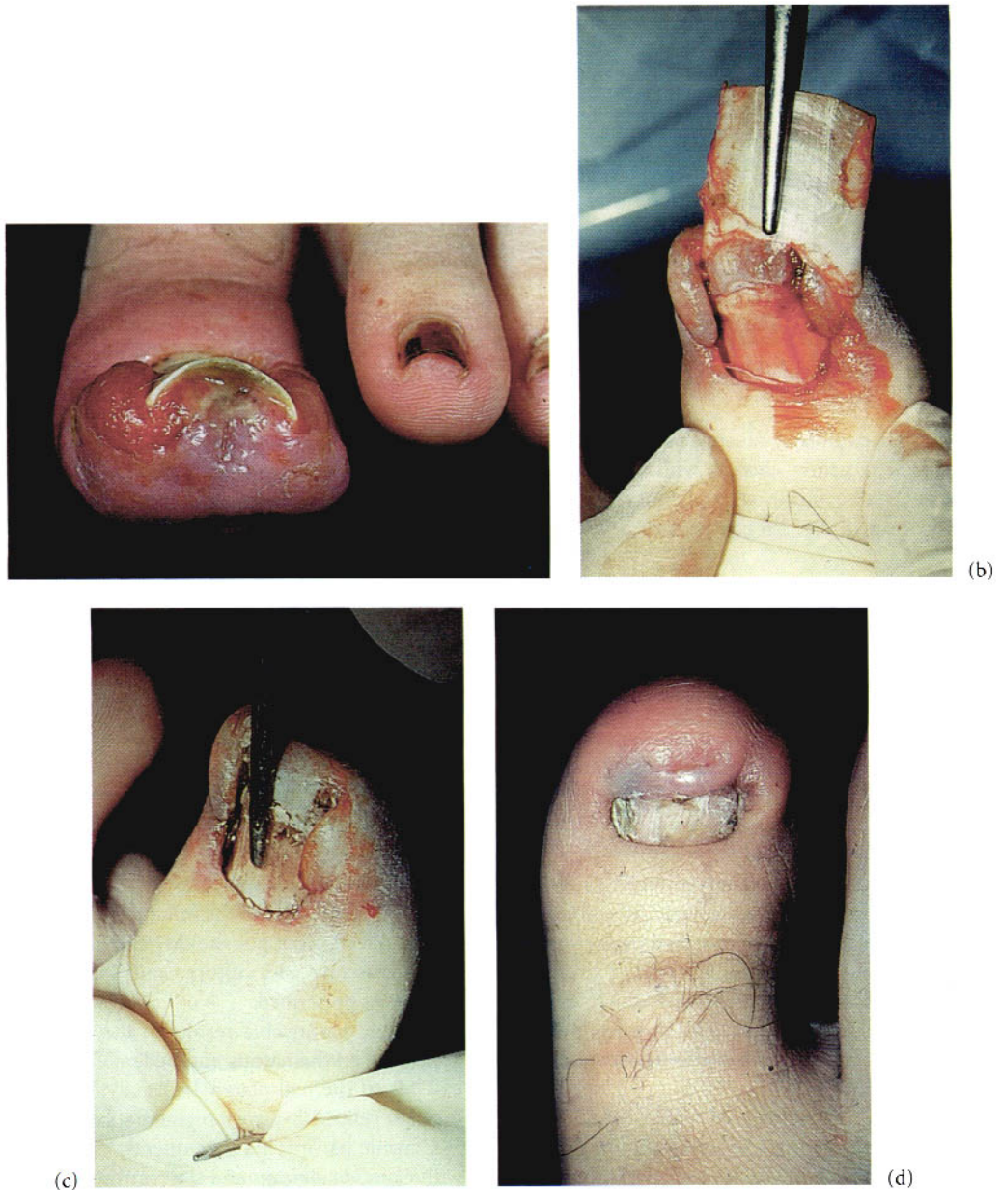


Fig. 2.6 (a) Acute onychocryptosis. (b) The nail plate is lifted off the nail bed with artery forceps. A tourniquet ensures a bloodless field. (c) Phenol is applied to the nail bed to prevent regrowth. (d) A fibrous plate has replaced the troublesome nail 6 months later.

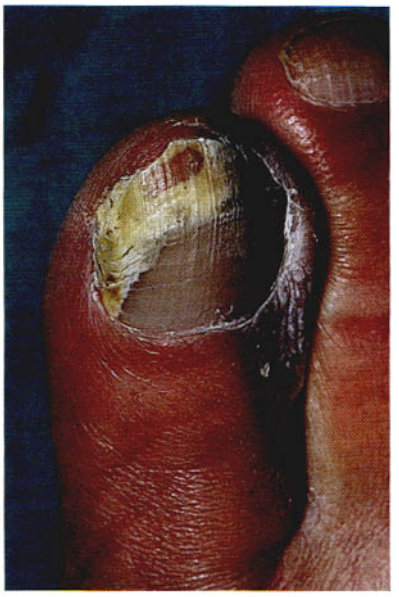


Fig. 2.7 Distal and lateral onychomycosis.

sulcus they can accumulate, causing pain and pressure. The nail sulcus is gently cleared with a Black's file. In severe cases, nail avulsion with phenolization of the nail bed, as shown in Fig. 2.6 can provide a permanent solution.

Nail infections

Onychomycosis (fungal nail). When fungal infection invades the nail plate it first causes white or yellowish discolouration of a patch of nail, which subsequently becomes thickened and friable. The majority of infections are caused by moulds called dermatophytes, or by yeasts, notably *Candida albicans.* There are four distinct types of onychomycosis:

- Distal and lateral subungual onychomycosis, which affects toe nails twice as commonly as finger nails. This is commonly caused by dermatophyte infection. The nail becomes detached from the bed (onycholysis) changing to a creamy white opaque colour (Fig. 2.7)
- Proximal subungual onychomycosis which is secondary to chronic paronychia caused by infection with yeasts of *Candida* species and is often associated with interdigital candidiasis
- Superficial white onychomycosis which is caused by *Trichophyton mentagrophytes* and is relatively uncommon
- Total dystrophic onychomycosis where the whole of the nail plate is destroyed is a consequence of any of the first three types.

Onychomycosis can cause chronic pain, physical disability and secondary bacterial infection. Eradication of the fungal infection is not easy, and some patients will opt for palliative care if they are not concerned by the cosmetic effect and the infection is not spreading.

Diagnosis can be confirmed by culture of nail clippings taken from the most proximal part of the affected nail to obtain crumbly material: however, many infections are treated without laboratory confirmation. Treatment can be palliative or active. Active treatment involves topical or systemic agents.

Palliative care involves regular debulking and thinning of the nail which can be done with a scalpel by a podiatrist. This approach is usually sufficient for most fungal nail infections and active treatment should only be considered when the infection is causing unpleasant symptoms or distress.

For topical treatment the thickness of the nail is reduced with a scalpel and an antifungal agent applied directly to the remnants. Agents available include topical amorolfine nail lacquer and strong iodine BP. Treatment should continue until a new nail has formed, which may take up to 12 months.

If systemic treatment is undertaken it is important to be aware of the possible side-effects of therapy.

- Terbinafine 250 mg daily for 3 months is the drug of choice for fungal nail infections. Rarely, it can cause liver toxicity
- Itraconazole 200 mg daily for 3 months or as Sporanox Pulse 200 mg bd for 7 days and subsequent courses repeated after a 21-day interval. Itraconazole has been associated with liver damage and should not be given to patients with a history of liver disease.

Inflammation of the nail fold. Inflammation of the nail fold, or paronychia, can be acute or chronic.

Acute paronychia is due to bacterial infection, is painful, points and discharges pus. If the margin of the nail plate is pressing on the inflamed area it should be cut back. Collections of pus should be drained. A swab is sent for microscopy and culture, and appropriate systemic antibiotics prescribed.

Chronic paronychia results in the periungual tissues appearing erythematous and oedematous. The infection extends to the nail plate which may develop yellowish-green or yellowish-brown pigmentation.

Chronic paronychia is frequently caused by infection with *Candida albicans* and the treatment is with terbinafine or itraconazole, as described above. The feet should be kept dry.

Lesions under the nail
These can be due to:
- Haematoma
- Necrosis
- Melanoma
- Exostosis.

Subungual haematoma. This follows a trauma to the nail, when blood collects under the nail plate causing red, purple or black discolouration. Pain can be agonizing. The patient should be reassured that drainage will relieve pain and reduce damage to the nail bed. The blood is evacuated through a small hole made in the nail plate by paring with a scalpel or with a chiropodist's nail drill. If the nail plate is loose it should be cut back as far as possible to prevent the loose area from catching on the hose and causing further injury, and also to assist inspection of the nail bed.

Subungual necrosis. This can be due to trauma, infection or hydrostatic pressure from a haematoma under the nail bed which is not evacuated in time to prevent local ischaemia and tissue death.

CASE STUDY
Subungual necrosis

A 79-year-old patient with type 2 diabetes of 7 years' duration dropped the family bible on his left great hallux. The toe was exquisitely painful and rapidly developed discolouration beneath the nail plate. After 4 days he visited his general practitioner who diagnosed a subungual haematoma and referred him to the diabetic foot clinic and he was seen the same day (Fig. 2.8a,b). The nail plate was cut back to reveal an area of necrosis involving the nail bed. Differential diagnosis was necrosis caused by infection or purely by hydrostatic pressure from a collection of blood under the nail plate. Systemic antibiotics were prescribed. The necrotic area gradually dried, demarcated and healed. When the new nail plate grew back it was onychogryphotic.

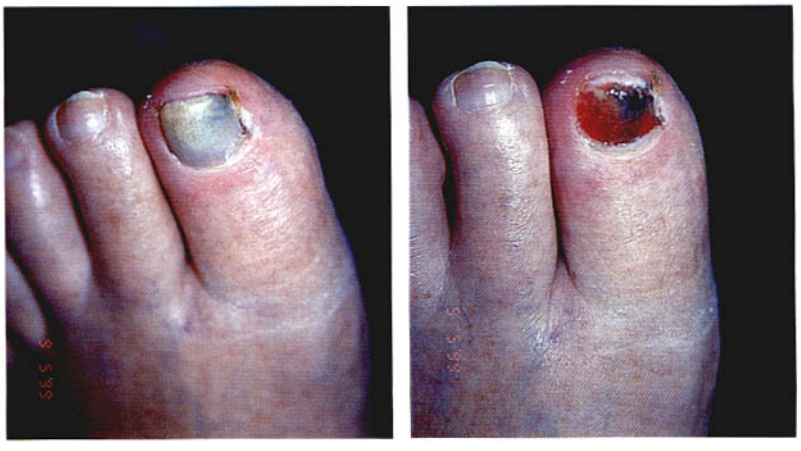
Key points
- It is impossible to assess a nail bed lesion properly without removing the overlying nail plate
- All painful subungual haematomas should be drained without delay by cutting back the nail plate.

Subungual melanoma. Malignant melanoma may also present as a discoloured area under the nail plate. Irregular discolouration of the nail bed and plate and progressive destruction of nail are seen. Some melanomas are not associated with pigment (Fig. 2.9).

Patients should be referred urgently to the dermatologist.

Subungual exostosis. An acutely painful cherry red spot develops under the nail plate. A lateral X-ray reveals bony outgrowth of the distal phalanx. The treatment is surgical.

Fungal infections (tinea pedis)
These can present in several ways:
- Dry, scaly plantar, often in a 'moccasin-like' distribution
- Acute vesicular
- Interdigital, with moist, cracked areas which may be sore, itchy and sometimes malodorous, and are associated with whitish, rubbery, macerated skin, and can undergo erosion.



(a) (b)

Fig. 2.8 (a) Apparent subungual haematoma. (b) The nail bed is necrotic.

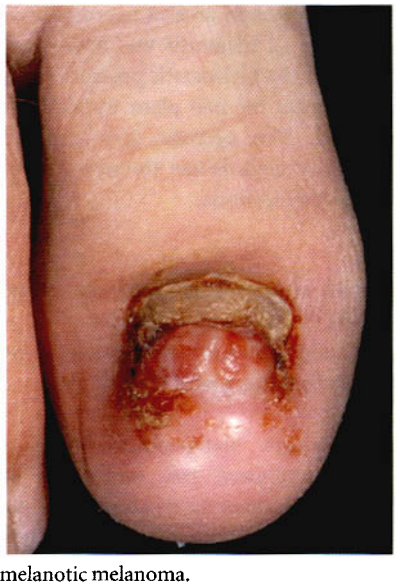


Fig. 2.9 Amelanotic melanoma.

Scrapings can be taken and sent to the laboratory for identification of the infective organism but usually a clinical diagnosis is made.

Treatment of tinea pedis

Canesten spray (clotrimazole 1% in isopropyl alcohol) applied topically is best for interdigital areas. For other parts of the foot Canesten cream can be applied. Treatment should be continued for at least 2 weeks after resolution of symptoms to avoid relapse. Whitfield's ointment is an old-fashioned but useful remedy: different vehicles in the formulation are chosen according to climate and geographical area. Tinactin (tolnaftate) and Mycil (chlorphenesin) can be bought over the counter in formulations including powder and cream.

Patients with fungal infections should receive precise instructions regarding duration of therapy and preventive measures for the future.

CASE STUDY
Interdigital tinea pedis

A 37-year-old woman with type 2 diabetes of 2 years' duration complained of severe itching between the 4th and 5th toes of her right foot. She had purchased a proprietary powder and a cream for athlete's foot and had used them for 2 weeks with no improvement. She said that the cream 'got all over the place' and the powder formed lumps which made her toes all the more sore.

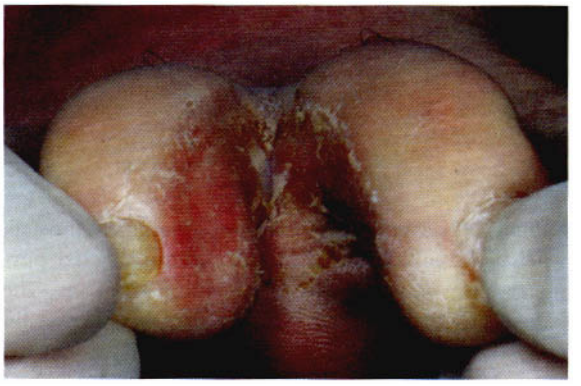


Fig. 2.10 An area of thickened rubbery white skin between the toes (tinea pedis).

Between her toes was an area of thickened rubbery white skin (Fig. 2.10) which was gently debrided away: the underlying tissue was intact, but macerated and inflamed. We prescribed Canesten spray and advised her to desist from using her proprietary remedies. When she returned to the clinic 3 weeks later the foot was healed and the problem did not recur.

Key point

- For interdigital application a spray may be more effective than a cream or powder.

CASE STUDY
Recurring tinea pedis

A 44-year-old woman with type 2 diabetes of 6 years' duration developed itchy vesicles on the border of her foot associated with dry skin, desquamation and pruritus (Fig. 2.11). We prescribed Whitfield's ointment which she

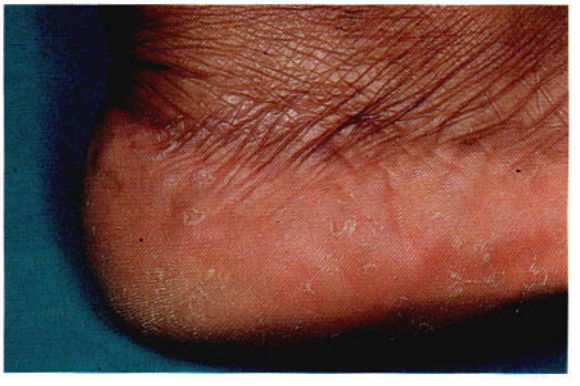


Fig. 2.11 Vesicular tinea pedis.

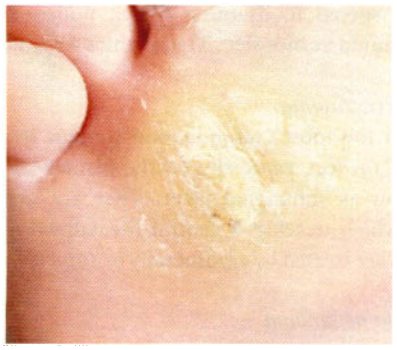


Fig. 2.12 Verruca.

used for 1 week. Three weeks later the problem recurred. She was advised to continue using the Whitfield's ointment until 2 weeks after the symptoms had resolved, and then to apply surgical spirit to the previously affected areas after washing them daily and drying them carefully. She was also advised to wear clean socks every day and dust her feet and interdigital areas with Mycil powder. The problem did not recur.

Key points

- Treatment of fungal infections should be continued for 2 weeks after symptoms have resolved
- Prophylaxis with Mycil may be necessary to prevent recurrence.

Fissures

Fissures are moist or dry cracks in epidermis at sites where skin is under tension. Deep fissures may involve dermis.

Fissures can occur in dry skin, when the treatment is an emollient, such as E45 cream, olive oil or coco butter, or in wet skin, where an astringent or antiperspirant such as aluminium chloride is helpful.

Verrucae

Warts may occur anywhere on the foot and may be single or multiple, and appear as round flattened papules or plaques. They are whitish or grey in colour with a rough surface (Fig. 2.12).

If they are on the plantar surface and thus subjected to pressure from walking, they may be difficult to distinguish from corns. However, warts are painful when they are squeezed while corns are painful when they are pressed. Skin striations are interrupted by warts but not by corns. Removal of a verruca by scalpel debridement reveals tiny reddish brown dots. Dots are not visible following removal of corns.

Small speckles of black (thrombosed blood vessels) can be a sign that the verruca is resolving spontaneously.

Accumulation of hyperkeratosis may cause pain on walking: excess keratin can be pared with a scalpel by the podiatrist or the patient may use a pumice stone. However, warts do not need to be treated unless they are painful or spreading: most will resolve within 2 years without treatment.

Some swimming pools require people with verrucae to wear verruca socks to avoid cross-infection.

The recommended treatment for ablation of painful or spreading verrucae in people with diabetes is cryotherapy with liquid nitrogen. The resulting breakdown of tissue should be kept clean and covered with a dressing. However, treatment with liquid nitrogen can cause severe pain and ulceration and should only be used on stage 1 feet. Treatment with strong acids or silver nitrate is not recommended in diabetic patients.

Sometimes surgical treatment with excision of the wart is required.

Patients should be warned not to self-diagnose or self-treat warts. Some foot malignancies present as wart-like lesions.

Bullae (blisters)

These are superficial accumulations of clear fluid within or under the epidermis which develop following trauma to the skin. Common causes include unsuitable shoes, failure to wear socks and walking in wet footwear. Pedal bullae are sometimes associated with hypoglycaemic episodes.

Several serious lesions, including early neuropathic and early ischaemic ulcers, pressure ulcers, burns, puncture wounds and infections complicating ulceration, may first present as a bulla.

Unless bullae are small, superficial and containing clear fluid, they should be regarded as stage 3 lesions.

Small, flaccid bullae can be cleaned and covered with a sterile non-adherent dressing. Large bullae (over 1 cm in diameter) and all tense bullae should be lanced with a scalpel and drained before dressing. Aspiration with a syringe is less useful because the hole frequently seals, fluid accumulates again and unrelieved hydrostatic pressure causes extension of the blister.

The cause of blisters should always be ascertained and addressed.

Bullosis diabeticorum

This is a rare condition where diabetic patients present with intraepidermal blisters which are not associated with

trauma and heal without scarring. Treatment of bullosis diabeticorum is as for bullae (see above).

Chilblains (perniosis)

These are localized inflammatory lesions, provoked by cold and injudicious reheating. Chilblains are frequently found on the toes. Initially they are white due to vasoconstriction, but usually present as dusky red swellings which are intensely pruritic in the acute stages. When they become chronic they present as purplish lesions. They are best managed by taking preventive measures. Patients should avoid standing in the cold and damp, and should refrain from toasting their toes in front of the fire when their feet are chilled. In the cyanotic phase compound tincture of benzoin BP may be used.

Malignancy

Although skin malignancy is rare in the foot, the diagnosis should be considered, especially in unusual and non-responsive conditions.

It is important to be aware that squamous cell carcinoma, malignant melanoma and, very rarely, basal cell carcinoma, may present in the foot.

Squamous cell carcinoma

It may present as a reddish plaque, warty lesion, nodule or as an ulcer with undermined ragged edges. Squamous cell carcinomas are likely to arise at the site of a scar or an existing lesion such as venous ulcer or subungual ulcer.

Most squamous cell carcinomas are locally aggressive and can spread to draining lymph nodes. Suspicious lesions should be biopsied. Treatment is surgical excision.

Basal cell carcinoma

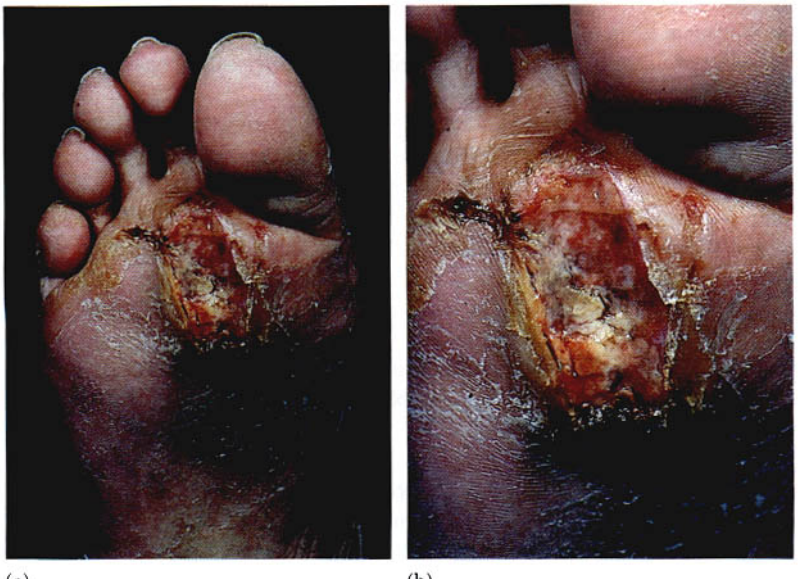
Although the most common skin tumour overall, it is very rare on feet, especially on the plantar surface, and even rarer as subungual ulcer. They are small pearly lesions which develop a central breakdown and rolled edge and are treated by cryotherapy or surgery.

Malignant melanoma

Malignant melanomas arise from existing moles or spontaneously from no apparent pre-existing lesion. Some melanomas produce little or no pigmentation and are termed hypo- or amelanotic melanomas. Melanomas are usually pigmented lesions on the foot which are irregular in outline and border, and variable in colour. They may be painful. It is wise to refer without delay to the dermatologist all pigmented lesions that develop *de novo*, change in size, shape or colour, or develop inflammation, ulceration and bleeding.

CASE STUDY
Squamous cell carcinoma

A 50-year-old female with undiagnosed type 2 diabetes, applied Bazooker, a proprietary wart remedy, to a small brown tender papule over her right third metatarsal head. Within a few days she developed a cutaneous erosion which failed to heal for 9 months and became increasingly painful (Fig. 2.13a,b). Diabetes was diagnosed by her



(a) (b)

Fig. 2.13 (a) Ulcer following application of a proprietary wart remedy which proved to be a squamous cell carcinoma. (b) Close-up of lesion.

general practitioner and she was referred to the diabetic foot clinic. She was referred on to the dermatologist because her plantar ulcer was unusually painful. The ulcer was biopsied and proved to be a squamous cell carcinoma. She underwent wide excision, but already had pulmonary, pelvic and lymph node metastases. She underwent chemotherapy and radiotherapy.

Key points

- It is important to have a high index of suspicion for plantar lesions which initially appear as papules and then break down to ulcers
- A full history should be taken of all foot lesions
- Diagnosis of skin lesions that have been treated with proprietary remedies is difficult as the morphology may be altered by the topical application of acids
- Lesions with abnormal appearance should be referred to the dermatologist without delay.

Inflammatory skin diseases

Practitioners treating diabetic patients may encounter dermatological conditions that first manifest themselves on the foot, including:

- Dermatitis/eczema
- Psoriasis
- Purpura.

It is important to be aware of these conditions so that an early diagnosis can be made and appropriate referral to dermatology arranged.

Dermatitis/eczema

Dermatitis is an inflammatory skin disease caused by different factors. Eczema and dermatitis are essentially interchangeable terms. Acute dermatitis presents as redness and scaling with vesiculation. Chronic dermatitis is recognized by thickening of the skin and excoriation. Dry, fissured, scaly lesions are treated with bland emollients.

Contact dermatitis, in which there is a hypersensitivity reaction to specific allergens, is a notable manifestation of dermatitis of the feet. Some patients become sensitized to shoes or socks or common household products (Fig. 2.14). Contact dermatitis can also be caused by an inflammatory reaction to dressings (Fig. 2.15).

Where possible the cause of contact dermatitis should be established and removed.

Varicose eczema is associated with venous hypertension. There may be a history of varicose veins or deep vein thrombosis, and haemosiderin deposition leading to brown discolouration of patches of skin. Areas of varicose eczema may break down and develop into venous leg ulcers.

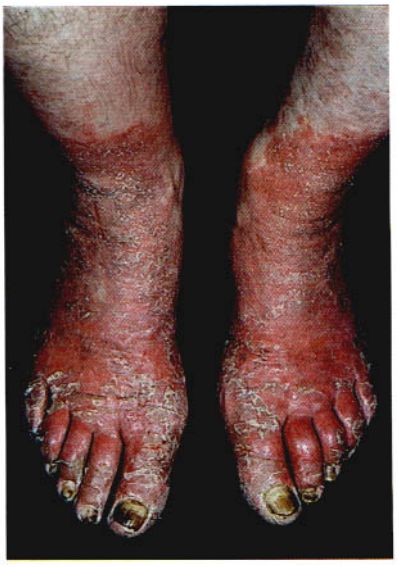


Fig. 2.14 This patient became sensitized to his socks.

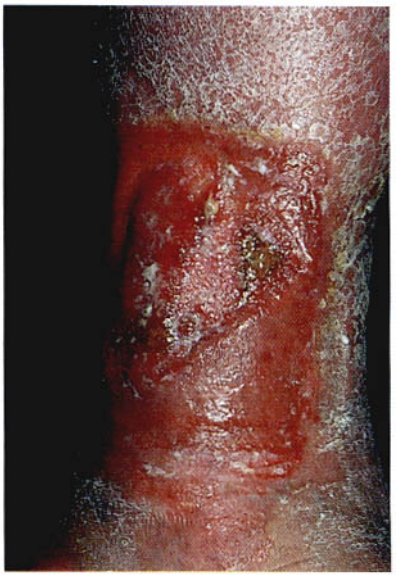


Fig. 2.15 Dermatitis caused by allergy to a dressing.

Psoriasis

Psoriasis usually affects the sole rather than the dorsum of the foot, with epidermal thickening and erythematous scaling lesions (Fig. 2.16). Pustular psoriasis presents with recurrent crops of sterile pustules with erythema distributed on the sole and lateral border of the foot. Psoriasis is associated with nail lesions including lifting of nail plate with onycholysis.

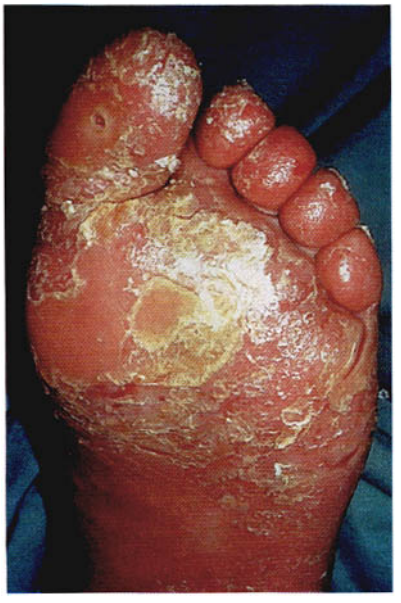


Fig. 2.16 Psoriasis.

Purpura

Purpura is caused by a disorder of the blood, such as platelet abnormalities or an abnormality of the blood vessels. Purpuric lesions do not blanch on pressure. When purpura occurs on the legs and feet the most likely causes are platelet disorders, Henoch–Schönlein purpura or meningococcaemia.

Hyperhydrosis

Hyperhydrosis is excessive sweating of the feet, and may be a particular problem in patients who live in tropical climates with high humidity. The skin becomes white, macerated and rubbery in texture and prone to blistering and fungal infections. It may be due to hyperthyroidism or anxiety.

The following procedures may help:

- Patients should avoid closed-in shoes made of plastic or other synthetic materials. Trainer-style shoes may exacerbate the problem
- Instead, patients should wear shoes made of leather or modern materials which can 'breathe' and allow moisture to evaporate
- Shoes should be changed regularly
- Insoles should be removed at the end of the day to dry out
- Absorbent cotton or acrylic fibre socks should be worn
- The patient should wear clean socks every day
- The feet should be washed every day, dried carefully and swabbed with surgical spirit including the interdigital area
- Talcum powder should be used in moderation.

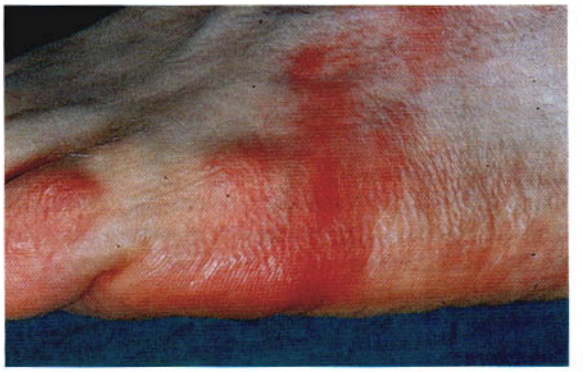
Insect bites

Insect bites can cause unpleasant cutaneous reactions.

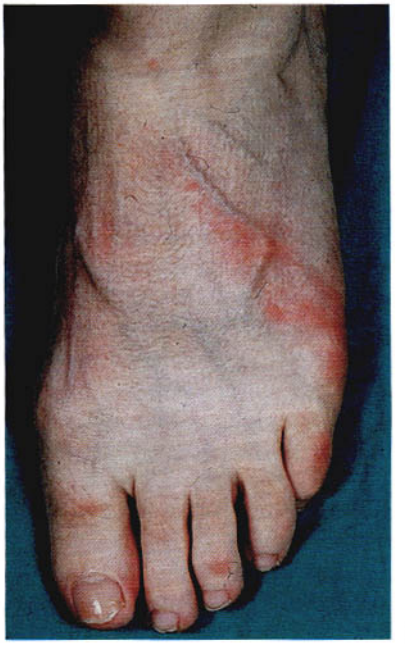
CASE STUDY

Insect bite (Fig. 2.17a,b)

A 35-year-old woman with type 1 diabetes of 9 years' duration, no neuropathy or ischaemia, and poorly



(a)



(b)

Fig. 2.17 (a) Insect bite on lateral border. (b) Spreading lymphangitis.

controlled diabetes, was bitten on the lateral border of her left foot by a mosquito. The lesion was intensely pruritic and she scratched the foot. The next day she had developed swelling of the foot and cellulitis and lymphangitis spreading up the foot. She was treated with antihistamine and systemic antibiotics and the foot improved after 3 days.

Key points

- Insect bites may cause severe cutaneous reactions, leading to severe swelling and erythema of the foot
- Patients should be warned to use mosquito repellent and cover up if sitting outside in summer in areas where biting or stinging insects are troublesome.

Traumas

Superficial traumas to the feet are extremely common, particularly if patients walk barefoot or wear unsuitable shoes. The cause of the trauma should always be identified to prevent recurrence.

Superficial cuts or grazes may be cleaned with normal saline. Lesions should then be covered with a sterile dressing or plaster. All diabetic patients should be advised to keep a first aid box at home to treat accidental injuries.

Fractures

These follow a significant trauma to the stage 1 foot, and are usually very painful and associated with severe bruising. A commonly seen fracture in patients who walk barefoot is fracture of the 5th toe caused by catching the toe on a piece of furniture and everting the toe forcefully. Intra-articular fracture of the hallux can lead to hallux rigidus, with subsequent overloading of the plantar surface and development of callus and ulceration.

Patients in stage 1 have protective pain sensation, and fractures are treated as for non-diabetic patients. Minor fractures of a toe are usually treated by using the adjoining toe as a splint and strapping the two toes together. Metatarsal fractures may be treated in a cast or brace.

Metabolic control (Fig. 2.18)

This should follow principles of modern diabetic management. Tight control of blood glucose, blood pressure, blood cholesterol and triglycerides, as well as stopping smoking and giving antiplatelet therapy when indicated, is extremely important at stage 1 in order to preserve neurological and cardiovascular function.

Hypoglycaemia is an important metabolic complication of diabetic treatment. It is defined as blood glucose less than 3.5 mmol/L (63 mg/dL). The incidence of hypoglycaemia is 10% per year in type 1 diabetes on twice daily insulin and 30% in those with multiple injections. There is less risk in type 2 diabetes: 0.5% per year if taking sulphonylureas and 2–3% in those taking insulin. All health-care professionals managing diabetic foot patients should be confident in diagnosing and treating hypoglycaemia.

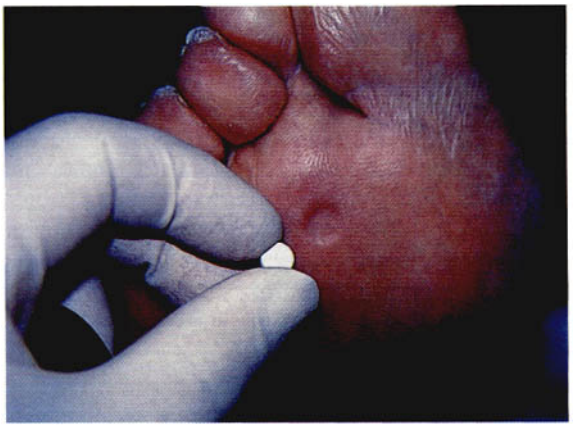


Fig. 2.18 This oral hypoglycaemic agent was found within the patient's shoe at annual review!

Blood glucose control

The results of the Diabetes Control and Complications Trial (DCCT) and the UK Prospective Diabetes Study demonstrated the value of tight control of blood glucose with sustained decreased rates of retinopathy, nephropathy and neuropathy. Treatment regimens that reduced average HbA_{1c} to ~7% (~1% above the upper limits of normal) were associated with fewer long-term microvascular complications.

In DCCT, people with type 1 diabetes who achieved near-normal glycaemic control experienced a 69% reduction in subclinical neuropathy and a 57% reduction in clinical neuropathy, as compared with the control subjects who received the usual treatment and who had higher levels of glycaemia.

The value of tight blood glucose control in preventing macrovascular complications is not yet firmly established, but lowering HbA_{1c} may reduce the risk of myocardial infarction and cardiovascular death.

Poorly controlled stage 1 patients may be more prone to develop sepsis than well-controlled patients.

Patients may injure their feet if they become hypoglycaemic. One of our patients damaged his feet on three separate occasions during severe hypoglycaemic episodes.

Blood pressure control

Hypertension (blood pressure greater than 140/90 mmHg) is a common comorbidity of diabetes, affecting 20–60% of people with diabetes, depending on age, obesity and ethnicity. Hypertension is also a major risk factor for cardiovascular disease and microvascular complications such as retinopathy and nephropathy. In type 1 diabetes, hypertension is often the result of underlying nephropathy. In type 2 diabetes, hypertension is likely to be present as part of the metabolic syndrome (i.e. obesity, hyperglycaemia, dyslipidaemia) that is accompanied by high rates of cardiovascular disease.

Randomized clinical trials have demonstrated the clear benefit of lowering blood pressure to below 140 mmHg systolic and 80 mmHg diastolic in people with diabetes. Thus target blood pressure should be lower than 140/80 mmHg. If the patient has microalbuminuria then the aim is for 130/80 mmHg.

Blood lipid control

Patients with diabetes have an increased prevalence of lipid abnormalities which contribute to high rates of cardiovascular disease, especially in type 2 diabetes. Lipid management aimed at lowering low-density lipoprotein (LDL) cholesterol, raising high-density liproprotein (HDL) cholesterol and lowering triglycerides has been shown to reduce macrovascular disease and mortality in patients with type 2 diabetes, particularly those who have had prior cardiovascular events. Reduction of saturated fat and cholesterol intake, weight loss and increased physical activity has been shown to improve the lipid profile in patients with diabetes.

Patients who do not achieve lipid goals with lifestyle modifications require pharmacological therapy. Statins should be used as first-line pharmacological therapy for LDL lowering. High serum triglycerides should be treated with improved glycaemic control and if this is not successful high-dose statins or fibrates.

Reduction of smoking

Smoking is a very significant risk factor for peripheral vascular disease. Clear, unequivocal advice should be given to stop smoking, but it is a very difficult habit to break.

Enrolment in a smokers' clinic programme may help the patient to give up. Health-care professionals cannot afford to ignore the problem of smoking in diabetic patients and should strongly encourage them to stop. Treatment should still be offered to patients who continue to smoke, but advice to stop or reduce the amount of tobacco consumed should be frequent and unequivocal.

Antiplatelet therapy

Diabetes may be considered as a hypercoagulable state. Daily intake of aspirin has reduced mortality in patients with diabetes and coronary artery disease. Dosages used in most clinical trials ranged from 75 to 325 mg/day. All patients with any degree of cardiovascular risk should take daily aspirin. If aspirin cannot be tolerated, then clopidogrel 75 mg daily should be prescribed.

Hypoglycaemia

Health-care professionals looking after diabetic foot patients should be aware of the symptoms and signs of hypoglycaemia. Capillary blood glucose measurement should be available as well as first aid treatment. A stock of glucose drink and biscuits should be available together with Hypostop Gel, glucagon and intravenous glucose.

Patients with foot problems should be aware that their treatment and investigations may be time consuming and should bring snacks or sandwiches.

Warning signs of hypoglycaemia are due to sympathetic overactivity and cerebral impairment because of reduced glucose availability.

Sympathetic overactivity

- Trembling
- Paraesthesiae around the mouth
- Shakiness
- Anxiety
- Hunger
- Sweating.

Cerebral dysfunction

- Confusion or altered behaviour
- Slurred speech
- Irritability
- Loss of consciousness.

Hypoglycaemic unawareness

Sympathetic responses decrease with increasing duration of diabetes, and patients may become unaware that they are hypoglycaemic: they develop hypoglycaemic unawareness. It occurs in 25% of patients with type 1 diabetes and in about 50% of patients with type 1 diabetes for more than 20 years. There is a change in the glucose threshold for activation of physiological responses to low glucose. The threshold is reduced to 2.5 mmol/L (45 mg/dL) instead of 4.0 mmol/L (72 mg/dL). Warning signs develop late and the brain does not recognize them because cognitive function diminishes below 3.0 mmol/L (54 mg/dL).

Protocol for treating hypoglycaemia within the diabetic foot clinic

Non-medical reception staff should be trained to keep a close eye on patients who are waiting for treatment, and organize sandwiches, drinks or lunch as appropriate.

Reception staff should be familiar with signs and symptoms of hypoglycaemia and call a health-care professional if they suspect hypoglycaemia.

If the patient reports symptoms of hypoglycaemia or staff suspect hypoglycaemia:

- A capillary blood glucose should be measured
- If below 3.5 mmol/L (63 mg/dL), hypoglycaemia is confirmed
- If the patient is not drowsy and can swallow, give 130 mL Lucozade or 200 mL fresh orange juice
- On recovery give 20 g of starchy carbohydrate such as a slice of bread or two digestive biscuits
- If patient cannot swallow, give Hypostop Gel around gums or glucagon 1 mg intramuscularly
- Check capillary blood glucose again after 15 min
- Give another glass of Lucozade if blood glucose is still below 3.5 mmol/L (63 mg/dL)
- If very drowsy give 75 mL of 20% glucose intravenously into a large vein through a large-gauge needle or give 1 mg glucagon intramuscularly
- Patient should not be left alone
- Check blood glucose again before they go home. If the patient has become hypoglycaemic from sulphonylurea therapy he should be admitted for at least 24 h and may need intravenous glucose therapy (5–20% as required)
- Give advice on hypoglycaemia prevention.

Educational control

General principles

Practical care of the stage 1 foot should involve a considerable amount of patient self-management education. All diabetic patients need to know, as an absolute minimum:

- What constitutes good foot care
- What is suitable footwear
- What to do and where to go if they develop a foot problem
- Simple first aid self-treatment
- The importance of the annual review examination.

There are different modes of communication in the education of patients. These include one-to-one discussion during the foot treatment, group discussions and distribution of written material. It is also possible to ask patients to advise and help other patients. Different patients respond to different modalities and different approaches: there is no one correct way of approaching education.

It is never too early to teach patients good foot care and footwear habits, and address barriers to care in order to gain long-term improvements in outcomes. However newly diagnosed patients may feel overwhelmed and inundated with information. For this reason, it may be best to delay foot care and footwear education for a few weeks, until the patient is feeling less upset and confused, and has digested the myriad of other information he has been given. However, the feet should always be checked at diagnosis since some type 2 patients, undiagnosed for some time, may already have diabetic foot problems. Diabetes foot care education should always be individualized and tailored according to a specific patient's needs, lifestyle and psyche, and frequently adapted as necessary. Explanations should be given as to the reasons for requests made to the patient to adapt his lifestyle. It is cruel to tell a young, fashion-conscious diabetic patient that she should spend the rest of her life in lace-up shoes, or the parents of a small diabetic child that he should never go barefoot. As in all areas of diabetes education, compromises sometimes have to be agreed upon. Advice should be practical and relevant to the patient's lifestyle. However, if the patient's behaviour is likely to lead to future problems he should be told.

Patients with a history of severe diabetic complications in near relatives, some of whom may have lost a limb, may be particularly vulnerable, both because of genetic factors and because of fear and loss of hope which makes them deny the efficacy of preventive treatment and fail to appreciate the long-term rewards of good control and foot care. These patients need extra support and education. For some patients the complications of diabetes seem unavoidable. They believe that no matter what they do, the outcome will be the same—amputation. This hopeless attitude needs to be corrected by education.

The approach must always be flexible. Some patients want a lot of information in order to feel safe: others will 'switch off' if given more than very simple basic information. Verbal education should be issued in small 'digestible' chunks, and reinforced by written material, including diagrams and pictures. Some patients will wish to have quite extensive information about the foot in diabetes and these stage 1 patients can be taught about neuropathy and ischaemia, the implications of these conditions, the ways in which their onset can be delayed or prevented and the terminology used to describe diabetic foot problems. Verbal education should be reinforced with written education and vice versa.

If group education sessions are held, the patients' families should be invited to attend and everybody should be

taught how to check the feet. Group education may help lonely or isolated people with diabetes, especially the newly diagnosed. We also ask patients to remove their shoes at education group sessions.

Some clinics have used patients as educators and we have found this approach helpful. Reformed reprobates can have great insight into barriers to care! Some of our patients have worked as volunteers, giving advice and encouragement to other patients. We also find that our 'open plan' clinic, where individual treatment areas are not walled or curtained off, enables patients and health-care professionals to learn from each other.

CASE STUDY
Poorly controlled diabetes

A 17-year-old girl with type 1 diabetes of 4 years' duration was referred to the diabetic foot clinic for education. A paternal uncle had type 1 diabetes and neuropathic ulceration. Her background was a chaotic one of great poverty and social deprivation with a history of truanting and running away from home. Her HbA_{1c} was 14%. She had frequent admissions to hospital for ketotic episodes and traumatic lesions to her heels and her navel which became infected. She was educated in foot care and footwear but continued to wear unsuitable shoes; she also frequently missed appointments at the diabetic clinic and diabetic foot clinic. However, she agreed to attend the clinic in emergency and to take antibiotics if her foot lesions became infected. She had no more admissions for foot problems, but subsequently developed severe neuropathy, proliferative retinopathy and end-stage renal failure and is currently on dialysis.

Key points
- A flexible, friendly approach in education is important to achieve cooperation and a positive outcome
- Sympathetic support and a safe haven may encourage patients to seek help early and avoid hospital admissions for foot problems.

Educational programmes for stage 1 patients should be based on the following.

Routine foot care
- Wash your feet every day in the bath or shower. Dry gently between toes
- Use mild, domestic soap and rinse the feet well after washing (when cleaning the area between the toes try not to jerk them apart: this can split the skin and make a sore place)
- Do not soak feet for too long: this makes them drier
- If skin is dry, rub in some moisturizing cream (hand cream, peanut oil, olive oil, coco butter are all good) into dry areas, but not between the toes
- If feet are moist and sweaty, apply surgical spirit to them twice daily, including area between toes
- Avoid walking barefoot
- Apart from simple first aid to injuries, do not try to treat foot problems yourself
- Do not buy proprietary remedies from the chemist
- If you want to try 'alternative medicines' discuss this with your health-care team first.

Daily foot check
Get into the habit of keeping a close eye on your feet and nails.

Remember the four danger signs of foot problems:
- Redness
- Swelling
- Pain or throbbing
- Breaks in the skin.

If any of these develop you should seek help via your diabetic foot service, which will have made clear the procedure for obtaining urgent help.

Check feet regularly for unusual lumps, bumps, splits, cracks, athlete's foot, rashes or verrucae (warts). If you find them seek help as above.

Remember
- That neglected callus can cause ulceration
- That if your foot changes shape or becomes infected you need help
- Check shoes daily for penetrating nails or sharp objects
- Check your feet every day for these danger signs or for anything else unusual about your feet
- If you have difficulty getting down to your feet, you can use a mirror to see the soles
- Ask your family to help you to check your feet if you have problems with your eyesight
- Always ask for advice if you notice anything unusual about your feet.

Nail care
It is quite safe for you to cut your own toe nails if:
- Nails are a normal shape and free of pain
- You can see your feet clearly
- You can reach your feet easily
- You have been taught correct nail cutting techniques.

Correct nail cutting techniques

- Cut nails after bath or shower: the water will soften them and make it easier
- Special nail nippers are better than ordinary scissors
- Do not cut a nail in one piece: nibble away at it. File your nails to reduce length and thickness. Rub the file in one direction only, towards the end of your toe, to avoid rocking the nail
- Do not let your toe nails grow beyond the length of the toe: they may catch on shoes and socks and become damaged
- Do not cut your nails too short: if you break the seal between the nail and the flesh you may get an infection
- Never dig or cut down the side of your nails: you risk leaving a spike of nail behind and causing an ingrowing toe nail
- Use a soft nail brush in the bath or shower to remove dirt or debris from around or under nails
- Nails need regular cutting: even normal nails will give trouble if they become too long
- If you have problems getting down to your feet or cannot see clearly, please ask for help cutting your nails from a family member or friend
- If your nails are painful or an unusual shape, change colour, or become thickened or ingrowing, or if you injure your nails, seek help from a health-care professional.

First aid information

Keep a small first aid box at home (and also take it on holiday) containing:

- Sterile dressings
- Plasters
- Bandages
- Hypoallergenic tape (sticky tape with special backing to prevent skin irritation)
- Antiseptic cream
- Tweezers
- Scissors.

Any wound which breaks the skin should be carefully cleaned under a running tap or with saline solution. (Make your own saline solution with half a pint of previously boiled water and two teaspoons of salt.)

Liquid antiseptics should be diluted according to the manufacturer's instructions before applying them to a sore place. Never apply full-strength antiseptic to your foot: it will burn the flesh. Then tape or bandage a clean dressing over the wound.

If the following occurs:

- Wound does not heal within a few days
- Foot becomes painful
- Foot becomes swollen
- Foot becomes red

seek help from your health-care professional as your foot may be infected.

Never stop taking your diabetic medication, even if your blood glucose is high and you cannot eat.

Footwear guide

- Choose a shoe which is long, broad and deep. There should be half an inch space between the end of the foot and the end of the shoe
- Shoes which fasten with a lace or strap are better than slip-ons
- Heels should be under 5 cm high
- The inner lining should be smooth
- Buy shoes in the afternoon—feet swell during the day
- Wear stockings or socks to avoid blisters
- If socks have prominent seams wear them inside out
- Break shoes in gently.

PRACTICE POINTS

- All diabetic patients should have their feet screened annually
- Diabetic patients should be advised to wear suitable footwear so as to prevent subsequent deformity
- Common foot problems should be diagnosed early and treated appropriately
- It is important to achieve good metabolic care with control of blood glucose, blood pressure, blood lipids, smoking cessation and taking antiplatelet therapy when indicated
- All diabetic patients should be educated in good foot care.

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Mechanical control

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3 Stage 2: the high-risk foot

Fear no more the heat o' th' sun . . .
Golden lads and girls all must,
As chimney sweepers, come to dust.
(Cymbeline, IV, ii, William Shakespeare)

PRESENTATION AND DIAGNOSIS

The diabetic foot enters stage 2 when it has developed one or more of the following risk factors for ulceration: neuropathy, ischaemia, deformity, swelling and callus.

These risk factors may not cause symptoms. Patients do not, thus, report problems. It is therefore important to screen patients at the annual review, which is an important part of diabetic foot care. A recent evaluation of a diabetic foot screening programme showed that it could prevent major amputations. A large randomized controlled trial of 2001 diabetic patients compared outcomes in 1001 patients who were screened for risk factors and 1000 in the control group who were not screened. The people at risk of foot ulceration in the screened group were treated with podiatry and education. At the end of 2 years there was only one major amputation in the screened group compared with 12 in the control group, which is a significant difference.

Neuropathy and ischaemia are the two most important risk factors for the diabetic foot. Deformity, swelling and callus do not commonly lead to ulceration in patients with intact protective pain sensation and a good blood supply, but when they are found in combination with neuropathy or ischaemia they significantly increase the risk of ulceration. There are other factors which increase risk, including diabetic complications, medical conditions and social problems:

- Poor vision
- Old age
- Social isolation—'lack of social connectedness'
- Poverty
- Ignorance
- Intellectual deficit
- Concurrent psychiatric illness
- Obesity.

Every diabetic foot at stage 2 will be classified as neuropathic or neuroischaemic. It is necessary to emphasize the great divide between the neuropathic foot, which lacks protective pain sensation but has a good blood supply, and the neuroischaemic foot with a combination of neuropathy and ischaemia, because the treatment will be different in the two groups.

This chapter considers (under 'Vascular control') two non-ulcerative complications of the neuroischaemic foot:

- Severe chronic ischaemia
- Acute ischaemia.

It also discusses three specific but non-ulcerative complications of the neuropathic foot:

- Painful neuropathy
- Neuropathic fractures
- Charcot's osteoarthropathy.

MANAGEMENT

Stage 2 feet require multidisciplinary care.

The following components of multidisciplinary care are important at stage 2

- Mechanical control
- Vascular control
- Metabolic control
- Educational control.

Wound control and microbiological control are not needed as the feet have intact skin.

Mechanical control

To maintain mechanical control, deformity must be accommodated by footwear and callus, dry skin and

fissures treated. Common non-diabetic foot problems, already described in stage 1, will also occur in stage 2 feet and need management as described in Chapter 2.

Footwear

Deformities in the neuropathic foot may include a raised medial longitudinal arch leading to high pressure points on the sole of the foot, which develops callus and ulceration unless protected by a special insole. The insole will usually need to be accommodated in a bespoke shoe.

The neuroischaemic foot is prone to develop ulcers upon its margins, often over the side of the 1st and 5th metatarsal heads, and 5th metatarsal base. Patients should be advised to wear a sufficiently wide and deep shoe to protect the vulnerable margins of the foot.

The overall approach is to try to accommodate these deformities in properly fitting shoes. The role of prophylactic surgery is discussed in Chapter 8.

For the multidisciplinary management of deformity, close liaison is necessary between physican, podiatrist and orthotist in the provision of shoes and insoles. Some deformities may be accommodated in high-street footwear. Patients with major deformities whose shoes cause red pressure marks or callus will need either footwear adjustments or special shoes.

An outline of the shoes and insoles available is given below, and a further discussion of footwear is given in Chapter 4.

Types of shoe

There are five main categories of shoe.

- Sensible shoes of a correct size and style from the high-street shoe shop. Once patients are high risk they should not wear mail-order shoes unless they can obtain them on a sale or return basis and have them checked by a health-care professional. Athletic shoes (trainers) are a reasonable choice for most patients. Patients who have neuropathy and don't know it will be accustomed to the tactile sensation of having shoes on their feet and may progressively buy tighter-fitting shoes to reproduce that sensation. This habit can cause pressure necrosis—patients with neuropathy should be warned to avoid tight shoes, and the implications of sensory loss should be emphasized. High-street shoes are not able to accommodate significant deformity which needs to be housed within stock shoes, modular shoes or bespoke shoes
- Ready-made, off-the-shelf, stock shoes (Fig. 3.1). These are made with extra depth and width, and without prominent seams. They usually have a low opening, are fully lined and contain a built-in rocker sole and flat-bed insoles made of microcellular rubber. The insoles can be replaced with bespoke insoles. Stretching specific areas, or making 'balloon patches' to accommodate single small deformities may also render off-the-shelf shoes suitable
- Modular shoes, which are the stage between ready-made and bespoke shoes. The orthotist carries out a trial fit using the standard stock shoe and then details a number of fixed modifications to be carried out
- Customized or bespoke shoes. These accommodate the shape of the foot which cannot be fitted within stock or modular shoes. The more abnormal the foot shape the greater the need for bespoke shoes. These may also be necessary if previous ulceration has resulted in scarring, depletion of fibrofatty padding under the metatarsal heads, or bound down plantar tissues leading to high plantar pressures. Bespoke shoes can house moulded insoles which redistribute high plantar pressures in the neuropathic foot
- Temporary ready-made shoes (for ulcerated feet) that can accommodate dressings. They are usually fitted with flat-bed insoles but a moulded insole can be inserted.

Fig. 3.1 Extra-depth stock shoe.

General principles of prescription of stock and bespoke shoes

- The patient's choice of material, colour and style should always be respected as far as possible
- For young patients, shoes made in a trainer style are often acceptable
- Patients need shoes for inside and outside, and may need bespoke shoes or boots for work, sometimes with steel toecaps. Agricultural labourers may develop prob-

lems if they wear Wellington boots for long periods in wet climates. In tropical climates agricultural labourers are at risk of deep fungal infections without protective footwear
- Shoe soles should be thick enough to prevent puncture by nails or thorns
- Shoe fastenings should be adjustable to accommodate swelling. Patients should be taught to rest the heel of the shoe on the ground and move the foot well back in the shoe before doing up the laces
- Shoes should be checked at every clinic visit and reassessed frequently for excessive wear and the changing needs of patients
- Patients should have at least two pairs so that they are never without a pair of wearable shoes
- Each pair of shoes should, if possible, not be worn for more than 2 consecutive days at a time
- Patients should wear special shoes at all times except for bed and bathing. They should not 'keep special shoes for best'—they are for everyday use. Slippers should not be worn round the house
- Shoes which become worn down should be brought in for early repair and the orthotist should supervise repair of bespoke shoes
- There is need for regular review of footwear. If foot biomechanics are abnormal there may be uneven patterns of wear which can rapidly render the shoe likely to cause problems
- New shoes, and repaired shoes, whether bespoke, stock or from high-street stores, should be checked before the patient wears them for the first time. They should never be posted to the patient without a final fitting and check and if new problems develop the patient should be seen without delay.

Insoles

Insoles are made from a variety of polyethylene foams, microcellular rubbers and ethyl vinyl acetate foams, and can be flat-bed (usually one layer, provided in stock shoes) or moulded. Moulded insoles are usually made from two or three layers of differing densities.

Insoles are used to reduce or redistribute areas of high pressure, friction and shear in the following ways:
- By loading areas of the sole which are not normally in contact with the ground, such as the medial longitudinal arch, a total contact effect can be achieved, relieving local areas of high pressure
- By extending the insole up the sides of the foot, a cradle effect will reduce friction (so-called cradled insoles)
- Under particularly high-pressure areas, such as prominent metatarsal heads, areas of the insole can be excavated out to form a 'sink'
- Extra cushioning can be used to compensate for reduced fibrofatty padding over the metatarsal heads
- A metatarsal bar or dome can be incorporated, applying pressure behind the metatarsal heads to bring the toes down
- Silicone gel plantar inserts can be used to reduce shear and this material also comes in the form of heel cups, flat-bed insoles and sleeves for individual toes.

All insoles should be regularly checked for 'bottoming-out' or excessive wear.

CASE STUDY
Nail in shoe

A 47-year-old man with type 2 diabetes of 9 years' duration and previous history of neuropathic ulcer, attended the orthotist for review 1 month after receiving new shoes because he was developing new callus in his mid-tarsal area where he had never previously had a problem. When the orthotist removed the ethyl vinyl acetate insole he found that a large nail had punctured the sole of the shoe (Fig. 3.2). Fortunately the insole was thick enough to prevent penetration of the skin, but the increased pressure had led to callus formation.

Key points
- Patients who check their feet and report any new problems at an early stage can abort problems
- A thick insole can prevent puncture wounds
- Patients with neuropathy are wise to check the soles of their shoes for penetrating nails.

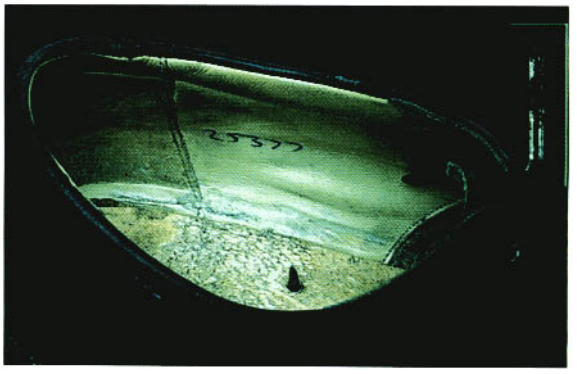


Fig. 3.2 Nail has penetrated the sole of the shoe—the insole has been removed to reveal the nail.

Fig. 3.3 The patient is wearing his favourite trainers on which a pair of bespoke trainers have been modelled, which are deep enough to contain a cradled insole.

Persuading patients to wear suitable shoes

There is a need for compromise, flexibility and imagination in footwear provision and education. Young and fashion conscious patients will want to look and dress the same as their peers, and use of trainers and Doc Marten's boots will make them feel less self-conscious. Where bespoke shoes are essential, they can be made in the above styles (Fig. 3.3).

If patients are reluctant to wear special shoes, then in-shoe pressure measurements can demonstrate that areas of the sole are at risk of ulceration because of high pressures.

CASE STUDY
High-tech education

A 25-year-old woman with type 1 diabetes of 10 years' duration and a history of neuropathic ulceration, was very reluctant to wear bespoke shoes and denied that they would help to prevent ulceration. We explained the association between high plantar pressures, callus and ulceration and then measured plantar pressures when she was wearing her own shoes and when she was wearing trainer-style shoes (Fig. 3.4a,b).

Once she saw the reduction of high pressures in the trainer-style shoes she agreed to wear them.

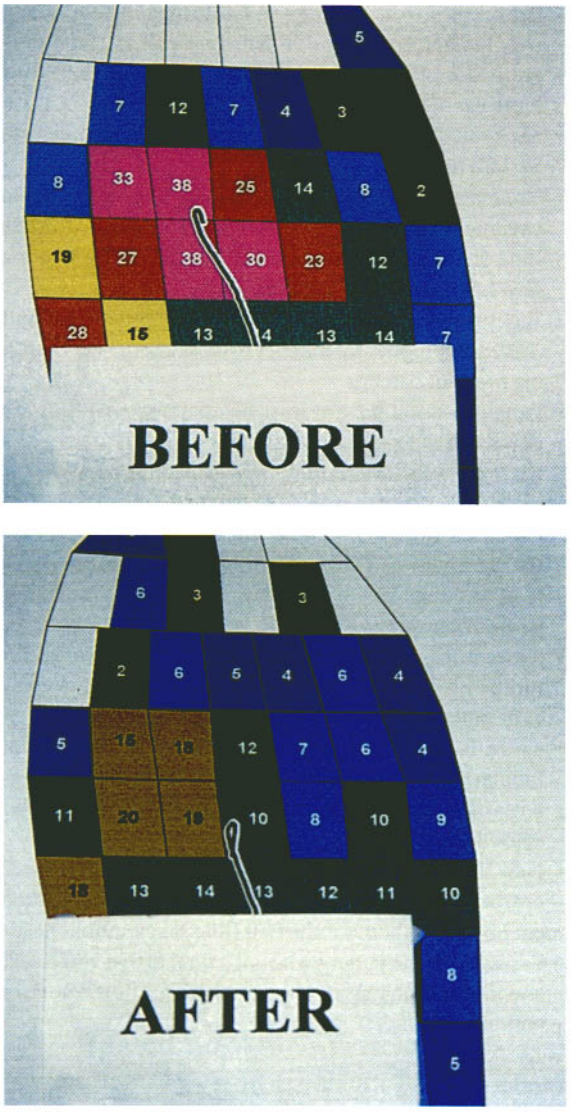


Fig. 3.4 (a) Forefoot pressure measurements using EMED system reveal red areas of unacceptably high pressure in patient's own shoes. (b) The areas of high pressure are eliminated in a trainer-style shoe.

Key points

- In-shoe pressure measurement systems are invaluable in demonstrating the reduction of forefoot pressures in special shoes
- When dangerously high pressures are demonstrated to patients they are more likely to agree to change their style of footwear
- When high-tech apparatus is not available, using low-tech educational messages is also effective. An outline of the patient's foot can be traced on paper, cut out and fitted into the patient's shoe: if it will not fit in the shoe, the patient can see clearly that the shoe is too small.

Patients' reasons for refusing to wear special shoes

- Too ugly and unfashionable
- Too heavy

- 'Look like hospital shoes'
- Unacceptable appearance
- High cost
- Excessive time to receive shoes
- Limited colours, styles, materials and durability.

It is better to compromise on design and issue a pair of shoes which are suboptimal but will be worn by the patient than to issue 'optimal' shoes which stay in the back of the wardrobe.

What to do if patients refuse to wear suitable shoes

- Try to limit the amount of walking patients do in unsuitable shoes
- If patients wear slip-on shoes, the heels should be as low as possible to avoid weight being thrown forward onto the metatarsal heads. If patients are accustomed to wearing high heels and agree to adopt a lower style, the heels should be lowered gradually to avoid damage to the Achilles tendon
- If shoes are particularly damaging, try to persuade patients to accept an alternative style
- Ascertain reasons for non-acceptance. Are they spurious or sound?
- Ask another patient who wears special shoes, of similar age, background and lifestyle, to talk to the patient
- Re-educate the patient and explain the importance of special shoes. Try to find a compromise or alternative in every case.

Management of specific deformities

The management of the following deformities will be discussed: claw toes, prominent metatarsal heads, fibrofatty padding depletion (FFPD), hallux rigidus/limitus, hallux valgus and foot drop.

Claw toes

They should be accommodated in shoes with a wide, deep toe box to reduce pressure on the dorsum of the toes. This may be achieved in high-street shoes, but often extra-depth shoes or bespoke shoes are needed. If the deformity is not 'fixed' a silicone rubber device (toe prop) can correct toe position and off-load the apices.

Prominent metatarsal heads

These can be accommodated in an extra-depth stock shoe with a cushioning insole. However, where the medial longitudinal arch is high and the metatarsal heads are extremely prominent, a cradled insole with sinks and bespoke shoes will be needed.

FFPD

Where fibrofatty padding is absent or greatly diminished, a cushioned insole, or felt padding can reduce plantar pressures. Patches of silicone gel can be applied over the metatarsal heads. If FFPD is associated with raised arch and clawed toes, a cradled insole in a bespoke shoe will be needed.

Hallux rigidus/limitus

A rocker sole can be applied to the sole of the shoe by an orthotist to reduce pressure at the end of the walking cycle when the toe leaves the ground. This condition may require surgical correction. Callus should be regularly debrided.

Hallux valgus

Extra-width stock shoes or bespoke shoes will be needed.

Foot drop

Foot drop can be accommodated by an ankle–foot orthosis (AFO) which can be of the traditional metal and leather calliper type or the more recent thermoplastic type. Sprung joints to assist dorsiflexion may also be added but will make the AFO heavier.

Management of corns, callus and fissures

Corns

Corns should be removed by sharp debridement with a scalpel. Interdigital corns may be moist and grasping them with a forceps helps maintain tissue tension. Felt padding may be used to deflect pressure but needs to be checked regularly.

CASE STUDY

Problems with felt padding

In this early case from the mid-1980s an 80-year-old woman with type 2 diabetes of 20 years' duration developed a corn over her left 1st metatarsal head. She attended a podiatrist who debrided the corn, applied a felt pad to deflect pressure, and told the patient on no account to remove the pad for 3 weeks. After 1 week the foot became painful but the patient refused to allow the pad to be removed. Three days later she was taken to casualty by her daughter. Deep necrosis had developed under the felt pad and she underwent extensive soft tissue debridement and amputation of the first ray. The foot healed in 9 months.

Key points

- Opaque coverings or padding should be lifted regularly to inspect feet which lack protective pain sensation

- Unequivocal advice should be given to patients to seek help immediately if their feet deteriorate
- For many years we refrained from using adhesive felt padding on high-risk diabetic feet. We now use felt padding as a temporary measure while insoles are awaited, but always advise the patient to lift the padding and check beneath it at regular intervals and review the patient frequently.

Callus

Plantar callus is a characteristic feature of the neuropathic foot and its potential for causing ulcers should never be underestimated. Callus concentrates pressure on the plantar aspect and increases the risk of ulceration more than 11-fold. Callus is the most important preulcerative lesion in the stage 2 foot. On the neuropathic foot it is usually hard and dry because of reduced sweating due to autonomic neuropathy. When neglected and allowed to accumulate, it causes pressure necrosis and ulceration of the underlying tissues. Good blood flow is probably necessary for exuberant callus formation.

Callus also develops on the neuroischaemic foot, but it is thin and 'glassy' and rarely causes ulceration. We do not recommend that areas of thin glassy callus on ischaemic feet be debrided unless they develop rough areas which can catch on clothing, are causing pain or develop signs of underlying problems. The practitioner must be aware that the layer of callus may be very thin, that the texture of ischaemic callus is glazed and slippery and that without great care the scalpel blade may slip. Callus in nail sulci should also be cleared with great care when patients are ischaemic. It is very important not to traumatize the ischaemic foot: underoperating should be the rule.

Preulcerative callus

Clear warning signs become apparent when callus becomes too thick and ulceration is imminent. These include:

- Callus with small speckles of blood within it where individual capillaries are damaged by pressure and begin to leak
- A deeper layer of white, macerated callus within callus (Fig. 3.5) only exposed by sharp debridement of the superficial layers
- An intraepidermal bulla full of clear fluid, but the underlying tissue is intact.

Emergency treatment to remove callus and reduce the excessive mechanical forces by means of footwear adaptations should be undertaken without delay.

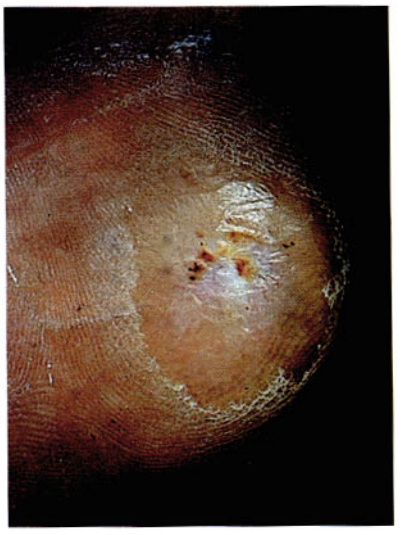


Fig. 3.5 Callus debrided to expose white macerated layer and speckles of blood.

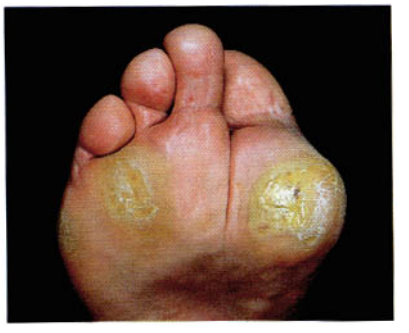


Fig. 3.6 Heavy callus containing speckles of blood indicates a preulcerative state.

CASE STUDY
Preulcerative callus

A 50-year-old man with type 1 diabetes of 30 years' duration underwent amputation of the second ray of his right foot for wet gangrene. At discharge from hospital he was reluctant to wear special shoes. After the foot healed he developed heavy callus over his 1st and 4th metatarsal heads. Speckles of blood within the callus indicated a preulcerative state (Fig. 3.6). He agreed to wear bespoke shoes after the significance of the blood within the callus was explained. The orthotists supplied cradled insoles with poron sinks in bespoke shoes, callus was debrided at monthly intervals, and he did not develop an ulcer.

Key points

- Speckles of blood within callus indicate that an ulcer is imminent
- Patients with ray amputations should be strongly advised to accept bespoke shoes and taught to return immediately if speckles of blood are observed within their callus
- Regular debridement of callus and bespoke shoes can prevent neuropathic ulcers.

Podiatric removal of callus

- Callus should be removed with a scalpel held by the operator's dominant hand
- The fingers of the operator's other hand should stretch the tissue being operated on to maintain good skin tension and ensure even removal of callus
- The foot should not be soaked prior to callus removal by scalpel debridement. The operator needs visual and tactile clues to guide him as to the amount of callus that can be safely removed. Callus contains more moisture in its deeper levels, closer to the epidermodermal junction, and if the skin and callus are macerated by soaking then valuable tactile clues as to the depth the operator has reached are lost
- Patients who develop callus on pressure points need regular treatment and careful follow-up if ulceration is to be prevented
- Speed of regrowth of callus varies and treatment periods must be individually planned
- Formation of callus is a warning that dangerously high mechanical forces are acting on the stage 2 foot, and every effort should be made to achieve effective mechanical control through footwear adaptation and lifestyle changes
- Patients should be taught the danger signs that callus is becoming preulcerative
- Patients who fail to keep an appointment for callus removal should be recalled.

In countries where there is no tradition of podiatry, nurses and physicians can learn podiatric techniques (Fig. 3.7). To learn precision and gain manual dexterity they should try first to remove the skin from an orange without removing any white pith, and practice paring wax from a candle. The first 'patients' they remove callus from should be family or colleagues, and they can then move on to practising on non-diabetic patients with no risk factors before they treat high-risk patients.

Removal of callus by patients' carers

Patients should never cut their callus off, or use proprietary corn or callus removers. These contain strong acids and can damage the skin, allowing infection to enter the foot.

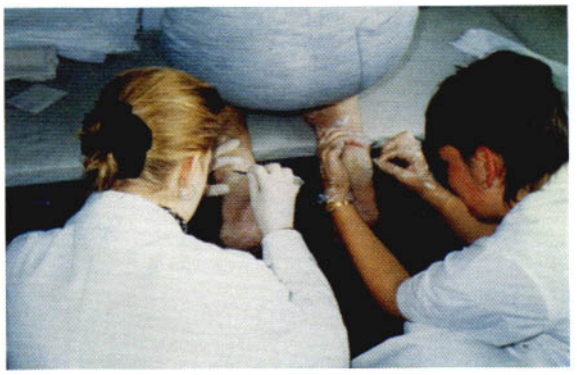


Fig. 3.7 Lithuanian nurse learns to debride a heel with an English podiatrist.

For diabetic foot patients who are unable to reach a health-care professional, the following procedure can be undertaken by a carer, family member or friend to reduce the callus.

- Soak the foot in a bowl of warm water containing a handful of salt for 15 min
- Rub a pumice stone or a piece of Scotchbrite (nylon kitchen scouring pad) over the area of callus to reduce the thickness
- Tape a piece of clean gauze over the area and keep it covered, and walk as little as possible for 48 h
- Observe the foot carefully for discharge or tissue breakdown. If these develop the foot should be shown to a health-care professional as soon as possible.

The authors are aware that the above advice is controversial. It is delivered in the belief that preventive foot care delivered by people who have been taught safe techniques and know the importance of not breaking the skin of the high-risk foot is better than no foot care at all.

In addition, correct nail cutting techniques (see Chapter 2) should be taught to stage 2 patients and their families if they live in isolated areas and are unable to obtain sufficiently frequent foot care from professionals.

Fissures

Fissures are a complication of dry neuropathic skin. Regular application of emollient helps to prevent fissures. The edges of deep fissures should be cleared of callus and the crevice can be held together with Steri-strips to speed healing (Fig. 3.8a–c). Patients who are prone to heel fissures should avoid backless footwear.

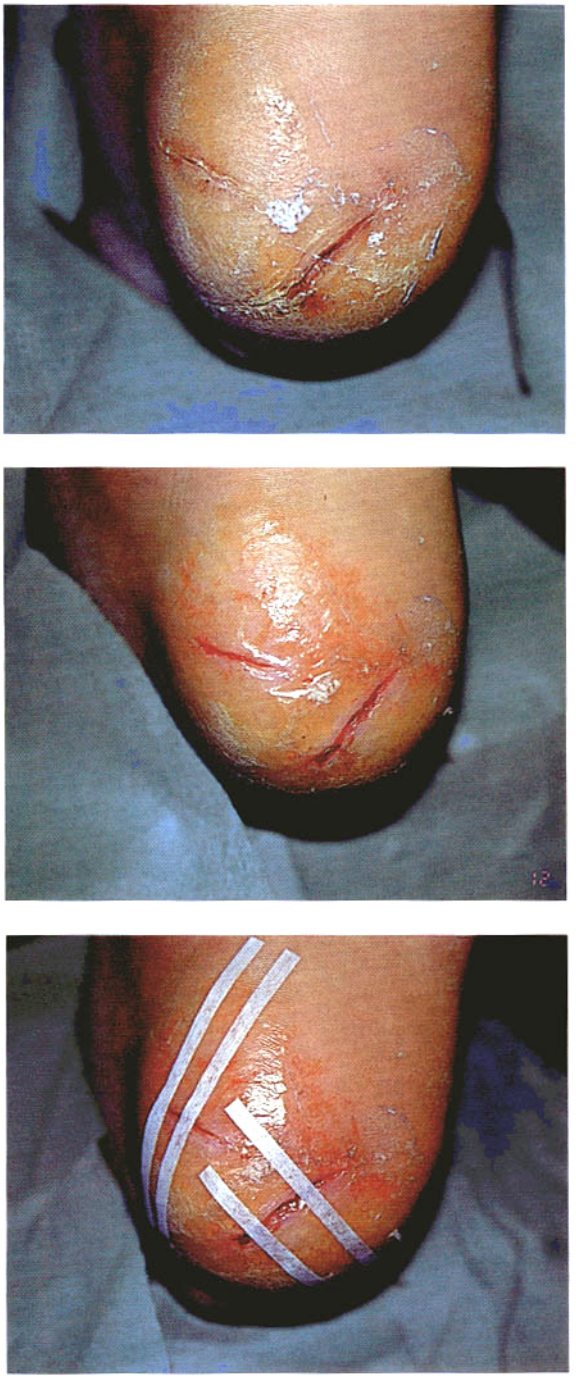


Fig. 3.8 (a) Deep fissures before debridement. (b) The edges of the fissures have been cleared of callus. (c) The edges of the fissures are held together with Steri-strips.

Other common foot disorders and their management are described in Chapter 2.

Vascular control

The majority of patients will be asymptomatic and ischaemia will be diagnosed on screening examination. Patients with absent foot pulses should have their pressure index measured to confirm ischaemia and to provide a baseline, so that subsequent deterioration can be detected before the patient presents with irreversible lesions.

Podiatrists should always ascertain their patient's vascular status before they cut the toe nails or remove callus, since any injury to the neuroischaemic foot can result in ulceration.

All diabetic patients with evidence of peripheral vascular disease may benefit from antiplatelet agents: 75 mg aspirin daily, or if this cannot be tolerated, clopidogrel 75 mg daily.

Diabetic patients with peripheral vascular disease should also be given statin therapy.

The Heart Protection Study has shown that simvastatin reduced the rate of major vascular events in a wide range of high-risk patients including those with peripheral arterial disease or diabetes.

Patients who are above 55 years and have peripheral vascular disease should also benefit from an angiotensin-converting enzyme (ACE) inhibitor to prevent further vascular episodes (as indicated by the Heart Outcomes Prevention Evaluation (HOPE) and microHOPE study).

If symptoms do develop in the foot with ischaemia, there are three main clinical presentations:

- Intermittent claudication
- Severe chronic ischaemia with or without rest pain
- Acute ischaemia.

Intermittent claudication

The classical site of claudication is the calf, although it may occur in the thigh and buttocks in aortoiliac disease. Claudication is less common in diabetic patients compared with non-diabetic patients because of peripheral neuropathy and the very distal site of atherosclerosis in the tibial vessels of the diabetic leg.

Patients with claudication should enter an exercise programme. All patients with claudication should be referred to the vascular surgeons but operative intervention is required in only 1% of diabetic patients per year. Pharmacological treatment with cilostazol can now be prescribed at a dosage of 100 mg twice daily.

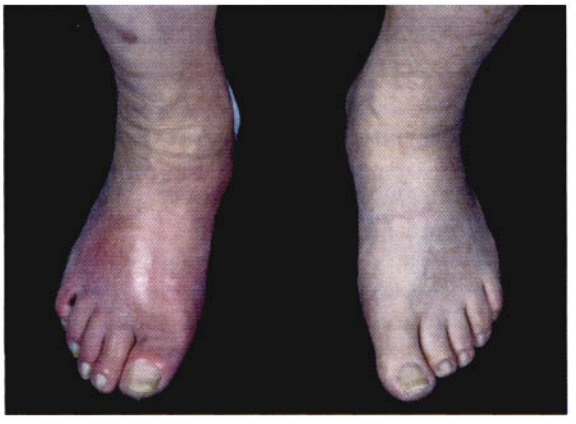


Fig. 3.9 Pink painful ischaemic right foot.

Severe chronic ischaemia

With increasing severity of occlusive arterial disease, patients may develop a pink, painful pulseless foot (Fig. 3.9). The colour of the skin is a strikingly bright pink and the foot is cold.

The amount of pain will be related to the severity of the disease and the degree of peripheral neuropathy. When neuropathy is mild, patients will have classical rest pain, which is a constant pain, often worse at night and relieved by hanging the leg down outside the bed at night. It is important not to mistake the pink painful ischaemic foot for an infected cellulitic foot. The pink painful ischaemic foot is usually cool and the infected cellulitic foot is usually hot. If the leg is elevated the pinkness of ischaemia will fade while erythema of cellulitis will remain.

The pink painful ischaemic foot is an indication of severe arterial disease and urgent vascular investigations will be necessary with a view to vascular intervention. The pressure index will nearly always be less than 0.5, although medial calcification may give an erroneously high value. It is wise to proceed to further investigations including transcutaneous oxygen tension and toe pressure measurements. A level below 30 mmHg confirms severe ischaemia in both tests. Pain control is important. (For further discussion of these investigations, vascular intervention and pain control in the foot with severe ischaemia, see Chapter 4.)

Vascular intervention may not always be possible. If it is possible to control pain, then a trial of conservative treatment may be attempted instead of immediately performing a major amputation.

CASE STUDY
Severe ischaemia

A 57-year-old man with type 2 diabetes of 7 years' duration underwent right femoral popliteal bypass. Eleven years later he flew to Canada. During the return flight he developed pain and numbness in his right foot and leg. He visited his local casualty department. A diagnosis of ischaemia was made but the severity was not appreciated and the patient was sent home. Next day he came to the diabetic foot clinic. The foot and lower leg were bright pink, icy cold and pulseless, with swollen toes and severe pain around the 1st metatarsophalangeal joint. Doppler studies could not detect pulsatile flow in the foot, and transcutaneous oxygen tension was 3 mmHg in the right foot and 50 mmHg in the left foot. He underwent an angiogram which showed that the graft was not patent. Further vascular intervention was not possible.

He developed necrosis on the medial aspect of the 1st metatarsophalangeal joint, and the lateral border of the foot. He also developed severe rest pain which was controlled with morphine sulphate. He refused a major amputation, and elected to undergo conservative treatment of his ischaemic foot in the diabetic foot clinic. There was a slow but gradual improvement leading to complete healing of the foot after 4 years. However, during this time he had five further admissions for infections. He had an initial period of 5 months off work but was then able to remain in full-time work.

Ten years later he still has two legs.

Key points

- Severely ischaemic feet in the diabetic patient may be difficult to diagnose because rest pain may not be as severe as in non-diabetic patients without neuropathy
- Severely ischaemic feet can sometimes survive without revascularization
- Close follow-up care from a multidisciplinary diabetic foot clinic is essential
- Healing can take years.

Acute ischaemia

A sudden occlusion of a major artery, usually popliteal or superficial femoral, will result in a pale, painful cold foot with purplish mottling. Initially the skin is intact, but if treatment is delayed digital necrosis will develop. (Management of necrosis is described in Chapter 6.)

Acute ischaemia is a rare complication of the stage 2 diabetic foot and can present very suddenly in:

- Patients with no previous history of vascular problems
- Patients with a history of steadily deteriorating chronic ischaemia
- Patients who have previously had peripheral arterial bypass which occludes or angioplasty with recurrence of stenosis or occlusion.

Unless the patient is profoundly neuropathic he will complain of sudden onset of pain in the leg and foot. If a hand is run down the leg a 'cut-off' point will be found where the temperature of the skin suddenly decreases.

Symptoms may include:

- Pain
- Numbness
- Paraesthesiae
- Weakness.

Signs are:

- Pallor
- Blueish-grey discolouration with mottling or 'bruised' appearance
- Paralysis.

Acute ischaemia is a clinical emergency associated with severe morbidity and mortality. If the leg is to be saved it is necessary to reperfuse it as a matter of urgency. Immediate vascular intervention is needed.

Metabolic control

Even though neuropathy or ischaemia may now be present, progression may be checked by tight control of blood glucose, blood pressure and blood lipids, and stopping smoking.

Swelling may complicate both the neuropathic and the neuroischaemic foot and it is an important factor predisposing to ulceration. Its main cause will be impaired cardiac and renal function which should be investigated and then treated accordingly. With deteriorating renal function swelling of the feet becomes a crucial factor leading to the intractable ulceration of end-stage renal failure. (See Chapter 6.)

When patients go on to haemodialysis the swelling can vary throughout the week according to the time of dialysis and it is important for the footwear to be adjustable, to accommodate fluctuant swelling.

Venous insufficiency can cause swelling of the leg and foot and should be investigated with duplex scanning, treated with support hose and referred for a vascular opinion as to the need for vein surgery.

Neuropathic oedema may respond to ephedrine starting at a dose of 10 mg tds but this may need to be increased up to 30–60 mg tds.

Educational control

General principles

- 'One-to-one' education can be carried out during the routine appointment by the podiatrist while the feet are treated
- 'Little and often' is the rule. When long 'lectures' are delivered, patients 'switch off'!
- Always seize the opportunity to get a point across or to ask a question which might reveal danger. Patients who volunteer that their vision is poor will need help with the foot check
- Changes in the patient's lifestyle (an impending holiday or trip, or a change of occupation) may indicate the need for extra education
- The diabetic foot service should get to know their patients well and be aware of changes going on in their lives which might have adverse effects on their feet
- Education programmes should be flexible, sensitive and individualized
- All stage 2 patients should be taught the danger signs of actual or impending foot problems and should know what to do and where to go to get rapid help, as described for stage 1 patients.

Above all, it is important to educate stage 2 patients in trauma prevention programmes so as to avoid ulceration and entering stage 3.

Psychological factors should also be specifically addressed in stage 2 patients.

Trauma prevention programmes

Trauma is the precursor of many ulcers, fractures and Charcot's osteoarthropathy. A special trauma prevention programme is needed for the successful management of stage 2 patients.

Causes of trauma

- Wearing of unsuitable footwear which rubs blisters and sores
- Wearing of 'thong' sandals
- Walking barefoot
- Foreign bodies within the footwear (patients should be taught to shake out their shoes before donning them and to check the inside for rough places or ruckled insoles). One of our patients developed severe ulceration when her small son dropped a Lego brick into her shoe: we found the brick later, deep within an ulcer!
- Nail penetration: one patient travelled from India back to England bringing back a tintack deeply embedded in his foot

- Burns from hot bath and shower water. Patients should check temperature of water with elbow or bath thermometer
- 'Toasting toes' in front of fires or fan heaters
- Falling asleep in front of the fire
- Walking on hot sand
- Spilling boiling water
- Falling asleep on the beach (sunburn)
- Frostbite. One of our patients suffered frostbite after being found lying unconscious in the snow.

Another patient with frostbite was a butcher with profound neuropathy who worked within a deep freeze room.

CASE STUDY
Burnt feet

A 52-year-old man with type 2 diabetes of 20 years' duration and profound neuropathy originally attended the diabetic foot clinic with neuropathic ulceration. This healed with regular debridement and special shoes. He was taught to check his feet every day, was careful to avoid trauma and had been ulcer free for 7 years. He attended the diabetic foot clinic at 3-monthly intervals for routine foot care and education.

He went on a business trip to Scotland and stayed in a hotel. During the night he was woken by a strong smell of cooking meat in his bedroom. On investigation he found a hot water pipe running along the wall by his bed, against which his foot had pressed in his sleep. He flew back to London the next morning for an emergency foot clinic appointment. He had a deep burn across the plantar surface of his forefoot which was cellulitic and discharging pus. He was admitted to hospital the same day for intravenous antibiotics. The burn gradually took on the appearance of a neuropathic ulcer, surrounded by heavy callus which needed regular debriding. Even when he was non-weightbearing, callus continued to develop. The foot healed in 17 weeks.

Key points

- No matter how careful patients who lack protective pain sensation are, they will sometimes injure themselves
- Patients in unfamiliar places are particularly vulnerable
- Patients should be specifically warned about the dangers of hot water pipes and radiators on bedroom walls
- Burns are hard to assess in neuropathic patients because all burns (not just third-degree burns) are painless (see Chapter 4).

Traditional remedies and alternative medicine

Some traditional remedies may be hazardous, in particular for feet with neuropathy. We have seen:

- Severe burns following treatment by a traditional Chinese physician who heated eggs in a pan until they were black and charred and slapped them directly onto the foot 'to draw the edges of the ulcer together'
- Maceration and infection following topical papaya ointment application
- 'Animal wool' wrapped around the toes to relieve pressure on interdigital corns, which absorbed perspiration, shrank and constricted the blood supply.

Patients and providers should be wary of unproved therapies.

The internet is full of details of folk remedies and new treatments. We ask patients to discuss the use of these remedies with us first so that we can establish their merits or dangers.

When traditional medicines prevent patients from seeking professional help their use is particularly dangerous.

Proprietary remedies can also be dangerous for the complicated diabetic foot. Many corn and callus removers contain salicylic acid. Many proprietary remedies contain equivocal advice which may be regarded by the patient as an indication that the products are safe. Warnings that patients with diabetes or peripheral vascular disease should not use them are frequently in a very small type which the patient with eye problems may be unable to read.

No matter how careful patients with neuropathy are, problems will sometimes occur. Without protective pain sensation it is extremely difficult to avoid and detect trauma, even if the patient takes great care.

Individualized trauma prevention programmes should take into account personal lifestyle and holiday and occupational activities.

Personal lifestyle

Patients in stage 2 are usually advised to avoid walking barefoot but if this is not possible the following precautions may help:

- Walking barefoot at temple or on pilgrimage to Mecca will be safer at dawn and dusk than in the heat of the day
- Grouting of floor tiles should be smooth
- Patients should clear up spillages immediately so they do not step on spilt uncooked rice or lentils or broken dropped objects.

Holiday foot care

Patients who are in unfamiliar places are at particular risk.

We remember a patient whose toes were crushed when a bus driver in Greece unfolded a seat onto his foot trapping his toes, but he was unaware of this until he tried to get up at the end of his 50-mile journey.

Trauma occurrence is particularly common in people on holiday. The reasons include the following:

- Unfamiliar environment exacerbates problems caused by poor vision or unsteady gait
- Lack of easy access to professional help
- Holidays regarded as a carefree time, when one can escape from the usual pressures
- Usual shoes discarded
- Increased alcohol consumption.

Dangers of the beach include:

- Cuts from sharp rocks, sea shells and broken glass
- Abrasions from sharp corals and sand or putting shoes on sandy feet
- Puncture wounds
- Burns from hot sand
- Not drying feet properly, leaving skin soggy and susceptible to trauma
- Insect bites and stings
- Sunburn.

CASE STUDY
Burn on the beach

A 62-year-old lady with type 1 diabetes of 40 years' duration, retinopathy and neuropathy went on holiday to Blackpool, removed her shoes and socks and sat in a deckchair on the beach for 3 h. Her head and torso were shaded by an umbrella but her feet and legs were exposed to the sun. She suffered a full-thickness burn on the dorsum of her right foot (Fig. 3.10). She was admitted to hospital for debridement and skin grafting and the foot healed in 6 weeks.

Key points

- Diabetic patients who go on holiday are vulnerable to injury. Thermal injuries are common and patients need to protect themselves from the sun
- Every diabetic foot service should have a special holiday foot care programme.

Dangers of airports include:

- Carrying heavy luggage which puts extra pressure on feet
- Airport trolleys which may run into or over the feet or lower leg
- Other passengers in a hurry who may step on toes
- Patients losing control of the situation in airports: they cannot limit their walking, or predict whether it will be a long walk to the gates.

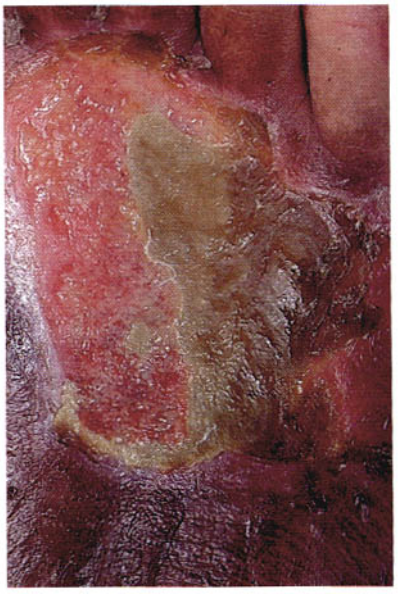


Fig. 3.10 Third-degree burn on the dorsum of the foot.

Patients who fly should:

- Walk up and down the gangway to reduce the likelihood of severe oedema or a deep vein thrombosis
- Wear adjustable footwear
- Avoid dehydration: drink plenty of water, avoid tea, coffee, alcohol and fizzy drinks.

We advise patients with a previous history of severe foot problems to organize a wheelchair at each end of the flight, and to allow plenty of time for embarkation and disembarkation.

General safety rules for patients on holiday include:

- No brand new shoes
- Wear socks
- If skin becomes dry use emollient
- Seek help from a local health-care professional if problems arise
- Telephone home foot care service for advice if necessary
- Always take out health insurance
- Take a first aid kit
- Avoid sunburn
- Wear plastic sandals on the beach and in the sea.

Occupational factors

Neuropathic patients may develop foot problems associated with jobs entailing much walking, and will some-

times need to seek desk jobs. Neuropathic patients whose job entails standing for hours in a factory in front of a machine will need to shift their stance from time to time and can be advised to set their watch beeper to go off at every quarter hour, whereupon they should 'stamp their feet about'. (They should probably explain to colleagues why they are doing this!)

Making patients feel safe

It is very frightening for high-risk patients if they sustain a trauma and cannot get immediate help without working their way through bureaucratic barriers and delays.

Many patients say that they only feel safe when they know that if they find a foot problem they can come to the diabetic foot service immediately for treatment and advice. The foot clinic should be a safe haven for people with diabetes.

Addressing psychological factors

Educators dealing with diabetic foot patients should be aware of the importance of psychological factors.

Diabetes arouses strong emotions of anger, fear and denial which can be barriers to successful management. Newly diagnosed patients are inundated with advice from health-care professionals and from well-meaning friends. Some advice may be inaccurate or unnecessarily negative. Patients may deny that diabetes or diabetic feet are a problem. On the internet a vast amount of unrefereed information is available which may give patients unrealistic expectations of the future.

In devising education programmes, the clinician should be aware of the psychological importance of the loss of protective pain sensation.

Protective pain sensation keeps behaviour within certain constraints: as we grow up we learn to avoid obvious noxious stimuli because of the unpleasant sensations associated with them. This reinforces safe behaviour, and less obvious stimuli will soon be detected because they give rise to discomfort, but it seems that this pathway needs constant reinforcement. When sensation is lost, behaviour can become reckless and hazardous.

Lack of touch and lack of pain perception have profound effects on the patient's body image and awareness of the physical boundaries of self in the following ways:

- Patients may feel that their neuropathic feet are no longer a part of them, and may ignore them and fail to look after them
- If ulcers or other problems develop, the patient is also likely to neglect them because they are not painful (and we have all been brought up to believe that if there is no pain the problem is not serious)
- Patients may therefore not perceive themselves to be at risk.

Psychological factors can worsen outcomes because they:

- Prevent a patient from understanding his foot problems
- Make him underestimate the risks he faces
- Make him refuse treatment or neglect to follow advice. Work with paraplegic patients has indicated that patients who do not perceive themselves to be at risk are more likely to neglect themselves and more likely to develop foot problems. It is essential that patients are made aware that they are at risk.

Management of patients with psychological problems

The health-care professional should always:

- Tell the truth to patients
- Avoid deliberately frightening patients (unless there is an urgent need to change their behaviour, for example when a very sick patient wishes to self-discharge from hospital or refuses a life-saving operation)
- Issue realistic advice. It is well worth taking time to explain to patients that they lack protective pain sensation, so that they understand the practical implications of their inability to feel a 10-g monofilament. In theory lack of protective pain sensation can be compensated for if patients are made aware that they will not feel pain, and taught trauma prevention and the need for regular foot inspections.

There are some patients who, despite regular and frequent education, appear to be unable to care for themselves or to take responsibility for their feet. They are labelled 'difficult patients'. They often have associated problems including:

- Old age
- Concurrent medical conditions
- Poverty
- Intellectual deficit.

Once such patients are identified it may be necessary to provide:

- Frequent treatment
- Home contact (telephone calls and postcard reminders) between appointments to ensure that all is well
- 'Conspiracy' with the community nurses or other carers to organize regular foot checks at home.

Some patients learn a sharp lesson the first time they develop a serious problem and never let it happen again, but others seem to be incapable of learning from previous experiences.

Diabetes coupled with renal disease may make patients even more reckless. There is a singularly high incidence of trauma in renal transplant patients.

SPECIFIC COMPLICATIONS OF NEUROPATHY

Painful neuropathy

The most common presentation is that of a bilateral painful peripheral neuropathy. However, pain can also be unilateral, when it is usually secondary to a focal neuropathy or mononeuritis.

Peripheral painful neuropathy

Presentation

This is a singularly disagreeable complication of diabetes. Patients in severe pain who cannot sleep become profoundly disturbed, confused, depressed and unwell. Relentless, burning pain and contact dysaesthesia make patients extremely miserable. It is particularly difficult to bear because apart from the pain there are often no signs which make it obvious that something is wrong. Some patients have described severe painful neuropathy after starting insulin, a so-called insulin neuritis, but the symptoms gradually improve if tight control is maintained.

Some health-care practitioners are unsympathetic because of the absence of signs.

Some patients with painful neuropathy lose weight. Often the first sign that they are entering the recovery phase is that they begin to gain weight again.

The pain may be sharp, shooting, stabbing or burning. There may be paraesthesiae or deep muscular aching pain, restless legs, cramps and/or sensation of cold.

Distribution of pain is usually in both feet extending into the lower legs in a stocking distribution. One limb may be slightly worse than the other. However, unilateral pain suggests either a diabetic mononeuropathy, such as femoral neuropathy, or nerve root pain due to compression such as in a prolapsed intervertebral disc.

Painful neuropathy is worse in bed at night, and when the feet and legs are in contact with clothes, including bedclothes.

Examination

On examination the feet and legs may be normal, although some patients may show a stocking distribution of loss of sensation. There may be an increased sensation to a normal stimulus such as light touch and this is termed allodynia or contact hypersensitivity. When there is increased sensation to normal painful stimuli such as pinprick, this is referred to as hyperalgesia.

When the pain is unilateral it is important to look for focal signs of root or nerve pathology, for example focal muscular weakness or unilateral loss of reflexes.

It is important to distinguish painful neuropathy from ischaemic rest pain. Rest pain is relieved by dependency but painful neuropathy is not affected by position change. In limbs with painful neuropathy, pulses are usually present. However, in the limb with rest pain there are definite signs of ischaemia, with absent pulses.

Investigations

Patients suspected of having peripheral painful neuropathy should have:

- Full blood count
- Urea/creatinine/electrolytes
- Liver function tests
- Protein electrophoresis
- Serum B_{12} levels.

Other causes of painful neuropathy include B_{12} deficiency, renal failure, alcoholic neuropathy associated with liver dysfunction and myeloma.

Vascular investigations should be carried out to rule out an ischaemic cause of the pain. In painful neuropathy, transcutaneous oxygen tension is greater than 30 mmHg and in the ischaemic limb, transcutaneous oxygen tension is usually less than 30 mmHg.

Management

The first important step is to reassure the patient that he will get better. Painful neuropathy nearly always resolves within 2 years although the pain may be replaced by numbness. The patient should be told that there is no danger of amputation.

The next step is to explore the many treatment options and try to find something to tide the patient over the next months before the pain resolves. If one treatment does not work there will be others to be tried. Reassurance, sympathy and tender loving care alone will help considerably: the knowledge that the pain will not last forever and that every attempt will be made to relieve it are also helpful. Frequent appointments should be made for review and assessment so that patients do not feel abandoned. If the patient can be helped to sleep he will feel better.

Simple measures may help. Pain may be relieved by cold: a basin of cool water may be kept beside the bed. One of our patients purchased a small refrigerator, kept it at the end of the bed, and inserted his feet when woken by pain! One patient wore a pair of his wife's tights under his

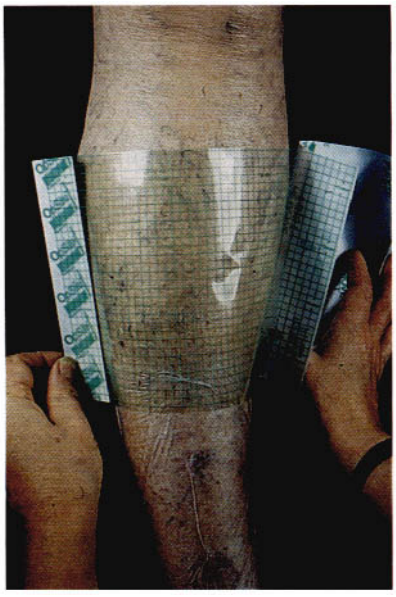


Fig. 3.11 OpSite film is applied to the skin of a patient with painful neuropathy.

trousers: another patient wore silk pyjamas underneath his city suit. A bed cradle can be used to prevent the bedclothes from touching the lower limb.

Treatment can be divided into four modalities:

- Topical therapy
- Glycaemic control
- Drug therapy
- Physical treatment.

Topical therapy

OpSite film stuck directly onto the skin and OpSite spray may help to relieve burning pain and contact dysaesthesia (Fig. 3.11).

Capsaicin is applied as a cream, and depletes peripheral pain fibres of substance P thereby blocking transmission of painful signals from the periphery to central neurones. It should be applied sparingly in a thin layer four times daily to the affected area. It should only be used on intact skin. Burning or tingling may occur in the first 2 weeks but the patient should be encouraged to persist. Patients should wash their hands immediately after use. The analgesic effect may take 6 weeks to develop.

Glycaemic control

Hyperglycaemia is known to lower the threshold to pain and in general diabetic control is usually poor in patients with painful neuropathy. It is important to improve control. In patients with type 2 diabetes, oral hypoglycaemic therapy should be optimized, but if this fails then insulin therapy should be started.

Drug therapy

This consists of analgesics, hypnotics, tricyclic antidepressants, anticonvulsants and antiarrythmics.

Analgesics. Initial therapy should be simple analgesics such as aspirin or paracetamol. If necessary, stronger analgesics may be given, such as dihydrocodeine or tramadol.

Hypnotics. Hypnotics are extremely helpful as loss of sleep makes the patient more sensitive to pain and induces depression.

Tricyclic antidepressants. Imipramine or amitriptyline may relieve burning pain. It is best to commence at a low dosage at night to avoid the side-effect of postural hypotension. The dose of amitriptyline should be 25 mg initially and this should be increased to 150 mg over a period of 16–20 weeks as long as serious side-effects do not occur. Mild side-effects include drowsiness, constipation, blurred vision, dry mouth and sweating. However, these symptoms should not persist on continuing therapy. If they do, the dose should be reduced. If the patient has difficulty in passing urine or becomes confused or develops palpitations then the medication should be stopped immediately by the patient, who should be reviewed urgently.

Anticonvulsants. These include gabapentin, phenytoin, carbamezapine, sodium valproate and lamotrigine. Gabapentin is commonly used and the starting dose is 300 mg on the first day, 600 mg on the second and 900 mg on the third day. Thereafter the dose should be increased to 1800 mg daily. It is usually well tolerated and initial drowsiness resolves.

Carbamezepine can be started at 100 mg once or twice daily and increased up to the maximum tolerated dosage (usually 800–1000 mg/day). Valproate (100–500 mg 1–3 times daily) and phenytoin (100–400 mg 1–2 times daily) may be helpful.

Antiarrhythmic drugs. These are rarely used.

Lidocaine can be given by intermittent intravenous infusion and may provide relief for several days. Mexiletine often has unacceptable side-effects although dosages of 450 mg/day significantly reduce burning pain and paraesthesiae without causing cardiovascular side-effects.

Anecdotally, patients have reported that cannabis helps.

Physical methods

Transcutaneous electrical nerve stimulation (TENS) can be used to block the pain. Electrodes are placed on either side of the painful area. Acupuncture has anecdotally been reported as useful. In very severe cases, spinal cord stimulation with implanted spinal electrodes has been used.

Focal neuropathy

The commonest focal neuropathy is femoral neuropathy but it can also present as a truncal radiculopathy on the thorax or abdomen.

Femoral neuropathy is associated with:

- Pain in thigh
- Weakness of quadriceps
- Difficulty in walking
- Weight loss.

It resolves in 18 months.

Truncal neuropathy involves the thorax or abdomen. It is associated with unilateral sensory loss, muscle weakness, which leads to a laxity and bulging of the abdominal muscles, and weight loss. Patients are often thought to have a malignancy because of the weight loss and the impression of an abdominal mass. Pain usually resolves within 9–12 months.

Neuropathic fractures

Presentation

The usual presentation is of a red, hot, swollen foot with relatively little pain. There may be a history of trauma. Commonest sites of fracture include metatarsal head, shaft or base or phalanx of the toe (Fig. 3.12). Neuropathic fracture can also occur in the calcaneum when it is often associated with avulsion of the posterior tubercle (calcaneal insufficiency avulsion fracture). In patients with severe neuropathy, particularly type 1 diabetic patients, neuropathic fractures of the tibia and fibula can also occur.

A fracture may be the initial bony change in the development of a Charcot foot (Fig. 3.13a,b).

A great pitfall in the diagnosis of neuropathic fractures is that the initial X-ray may be normal. A technetium methylene diphosphonate (MDP) bone scan is advisable if fracture is suspected.

Management

All neuropathic fractures should be treated promptly in conjunction with orthopaedic surgeons. Fractures that develop Charcot changes are associated with the greatest overall delay in diagnosis and management. Fractures should be treated with non-weightbearing and plaster immobilization, supported by crutches and/or wheelchair. Fractures do not heal at the same rate as in non-diabetic patients and plaster casts should therefore be continued until healing is confirmed by X-ray.

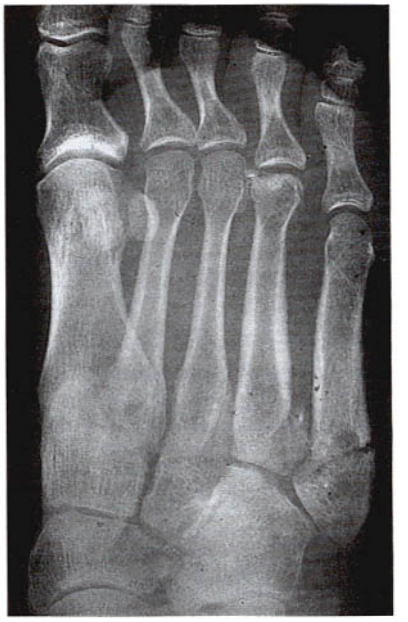


Fig. 3.12 Fractured styloid process at base of 5th metatarsal.

In our experience, fractures in stage 2 and higher stage diabetic feet take two or three times as long to heal as they do in low-risk or normal feet. Many health-care professionals appear to be unaware of this.

Many neuropathic patients have osteopenic bones and are prone to fracture bones after minimal trauma. Because of the lack of symptoms, fractures are frequently only detected after a routine X-ray.

Treatment of specific fractures

Toe fractures

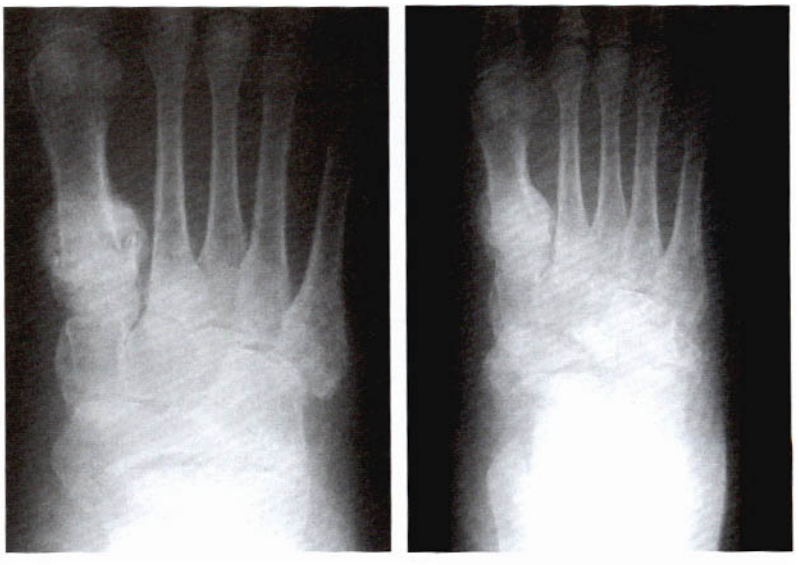
The toe is strapped to its neighbour for a splinting effect.

Metatarsal fractures

The patient is put into a below-knee plaster cast until X-rays confirm healing, which may take up to 6 months. In some cases full union is not attained. However, at this stage the initial swelling has usually resolved and patients are mobilized slowly out of the cast.

Calcaneal fractures

The limb should be immobilized in a below-knee plaster cast until radiological healing has occurred.



(a) (b)

Fig. 3.13 (a) Fractured base of first metatarsal. (b) The development of a Charcot foot with disorganization of the tarsus and fragmented navicular and cuboid.

Tibial and fibular fractures
These fractures can be treated either by open reduction and internal fixation or by cast immobilization.

CASE STUDY
Neuropathic avulsion fracture of the calcaneum

A 45-year-old woman with type 1 diabetes of 29 years' duration, developed a red, hot swollen foot 2 weeks after a fall.

Acute Charcot's osteoarthropathy of the mid-foot was diagnosed and she wore a total-contact cast for 7 months. When the foot had settled down with resolution of redness, warmth and swelling she was given a removable cast and advised to limit her walking to a few steps a day. Christmas was coming, and she went out to do her Christmas present shopping without her cast. Next morning the foot was hot, swollen and bruised and she came to the clinic. X-ray revealed that the entire posterior pillar of the calcaneum had been avulsed from the main body of the bone and was dangling from the Achilles tendon.

She was conservatively treated in a plaster cast applied to a plantar flexed foot, and subsequently in a bespoke boot: the foot remained ulcer free and the fracture healed.

Key points
- Rapid mobilization after Charcot's osteoarthropathy can lead to relapse
- We give very precise advice regarding timing of rehabilitation process and dangers of trying to speed the process.

CASE STUDY
Neuropathic fracture of the tibia

A 50-year-old woman with type 1 diabetes of 35 years' duration attempted to board a bus and missed her step and fell to the ground. She sustained a spiral fracture of the tibia and was treated conservatively with a below-knee plaster cast (Fig. 3.14a). However she developed Charcot changes at the ankle joint (Fig. 3.14b).

Key points
- Diabetic patients with type 1 diabetes have osteopenia and are susceptible to fractures
- Fractures can precipitate Charcot's osteoarthropathy.

Charcot's osteoarthropathy

This is an acute osteoarthropathy, with bone and joint destruction, that occurs in the neuropathic foot. Rarely, in diabetes, it can also affect the knee. Patients who develop Charcot's osteoarthropathy usually have evidence of a peripheral neuropathy, autonomic neuropathy and a good blood supply to the lower limb. Patients may have symptoms of autonomic neuropathy such as gastroparesis, diabetic diarrhoea, gustatory sweating or postural

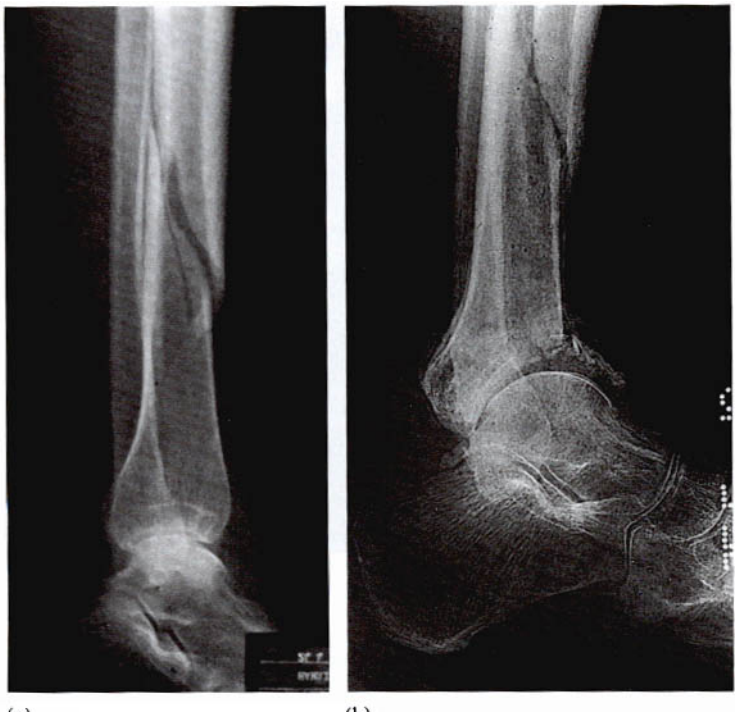


Fig. 3.14 (a) Spiral fracture of the tibia. (b) Charcot changes at the ankle joint following spiral fracture of the tibia.

hypotension. Diabetic patients with renal transplants have an increased risk of Charcot's osteoarthropathy.

Charcot's osteoarthropathy can be divided into three phases:

- Acute onset
- Bony destruction/deformity
- Radiological consolidation and stabilization.

Acute onset

There is unilateral erythema and oedema (Fig. 3.15). The foot is at least 2°C hotter than the contralateral foot and the difference may be as great as 10°C. This may be measured with an infrared skin thermometer. There may be a history of minor trauma such as tripping, twisting the ankle or walking over rough surfaces such as cobbles. Charcot's osteoarthropathy may follow injudicious mobilization after surgery, a period of bed rest or casting.

About 30% of patients complain of pain or discomfort. Rarely, pain may be very severe.

X-ray at this time may be normal. However, a technetium MDP bone scan will detect early evidence of bony destruction (Fig. 3.16). Early diagnosis is essential.

Patients awaiting bone scan should be treated as if the diagnosis has been confirmed. Although patients with an early injury may appear to be developing Charcot's osteoarthropathy, it is not possible yet to differentiate between those who have a soft tissue injury only and those who will develop extensive bony destruction. For this reason, all patients in stage 2 with a history of trauma, redness, warmth and oedema should be treated with a total-

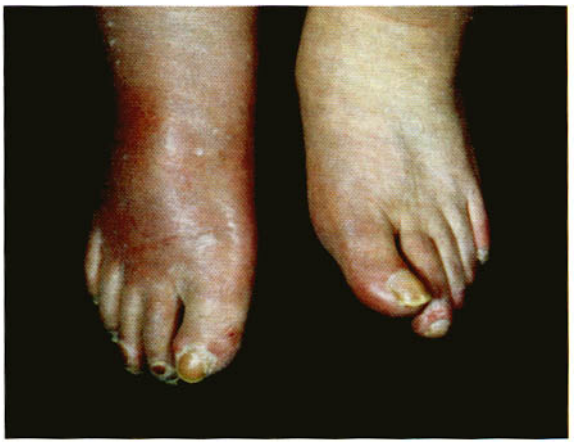


Fig. 3.15 Unilateral oedema and erythema in acute onset Charcot's osteoarthropathy.

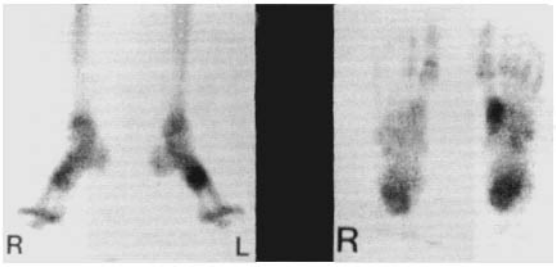


Fig. 3.16 MDP bone scan showing increased uptake at the base of the 1st metatarsal of the left foot, indicating early bony damage despite normal X-ray.

contact cast, which is described in full in Chapter 4. If the problem is not a Charcot's osteoarthropathy, but a simple sprain, it will resolve rapidly.

CASE STUDY

Differential diagnosis of acute Charcot's osteoarthropathy

A 65-year-old man with type 2 diabetes of 21 years' duration developed a hot, red, swollen left foot, after tripping in the street. On examination the foot was hot and swollen, particularly over the lateral aspect of the ankle. However, pulses were not palpable and the pressure index was 0.6. X-ray was normal. The differential diagnosis was an acute Charcot's osteoarthropathy or a soft tissue injury. Charcot's osteoarthropathy was thought to be less likely in view of the moderate ischaemia: however, it could not be excluded. The foot was put in a cast until an MDP bone scan was performed. This showed a diffuse uptake around the left ankle but no focal bony change, indicating a soft tissue injury (Fig. 3.17a,b). The patient was mobilized without a cast and the swelling gradually resolved.

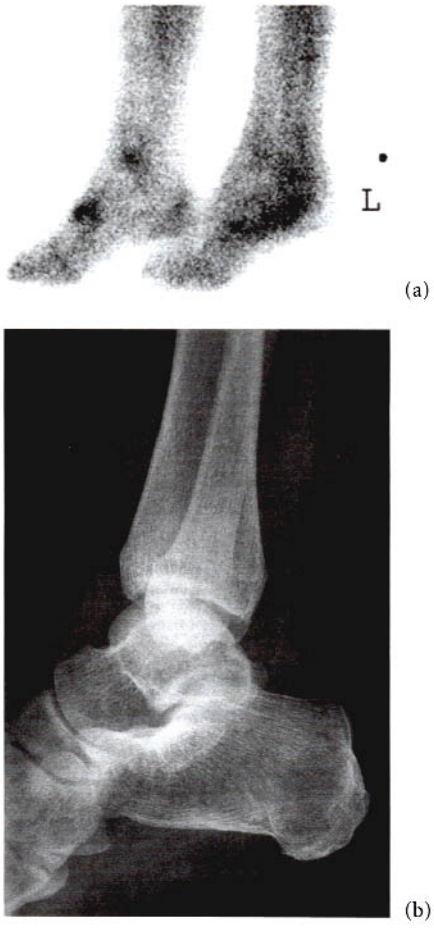


Fig. 3.17 (a) MDP bone scan showing diffuse uptake around the left ankle. (b) Normal X-ray of left ankle.

Key points

- Patients presenting with a hot swollen foot should be regarded as having a Charcot's osteoarthropathy until proved otherwise
- Until an MDP bone scan can be performed, the foot should be casted
- The MDP bone scan can differentiate between soft tissue and bony damage.

Differential diagnosis of acute onset Charcot's osteoarthropathy

It is important to differentiate between the red, hot, swollen appearance of Charcot's osteoarthropathy and the red, hot swollen cellulitic foot. Cellulitis is more likely in the presence of an ulcer which may show typical signs of infection. Infection severe enough to cause generalized redness, warmth and swelling will usually cause local signs such as discolouration of the bed of the wound, and discharge from the ulcer. The swelling of Charcot's osteoarthropathy responds more rapidly to elevation than does that of the infected foot. Infection and Charcot's osteoarthropathy can sometimes be present concurrently in the same foot. If in doubt, treat for both.

Gout and deep vein thrombosis may also masquerade as Charcot's osteoarthropathy but can be excluded by measurement of serum urate and duplex vein scan.

CASE STUDY
Acute onset Charcot's osteoarthropathy

A 64-year-old type 2 diabetic patient of 26 years' duration telephoned the foot clinic complaining of a swollen foot. He attended the next day and his foot was red, swollen and 3°C hotter than the contralateral foot. X-ray was normal. A total-contact cast was applied which was removed 5 days later in order for a technetium MDP bone scan to be performed. The bone scan showed increased uptake around the navicular. The total-contact cast was reapplied and he remained in a cast for 6 months, when the foot was within 1°C of the remaining foot, and with minimal deformity.

Key points

- If Charcot's osteoarthropathy is diagnosed early there may be no X-ray changes
- If Charcot's osteoarthropathy is suspected and the X-ray is normal, the foot is put into a cast until an urgent MDP bone scan can be performed.

Treatment of acute Charcot's osteoarthropathy

Initially the foot is immobilized in a non-weightbearing plaster cast. The cast is checked after 1 week, and replaced if it has become loose due to reduction of oedema, regularly checked and replaced as necessary. After 1 month the patient may walk for brief periods, using crutches and being encouraged to keep his weightbearing to a minimum. The casting is continued until the swelling has resolved and the temperature of the affected foot is within 2°C of the contralateral foot.

An alternative treatment is the Aircast, a prefabricated walking cast, described in Chapter 4. A moulded insole should replace the standard insole provided with the cast by the manufacturer.

A recent randomized controlled study of a single 90 mg pamidronate infusion has shown that markers of bone turnover and skin temperature decreased in both treated and untreated subjects, although to a greater degree in the treated subjects.

Bony destruction/deformity

If treatment is given early in acute Charcot's osteoarthropathy, it should help to prevent the second phase, that of bony destruction and deformity.

Once the foot becomes deformed or shows X-ray changes, it has entered the bony destructive phase. Clinical signs are swelling, warmth, a temperature 2°C greater than the contralateral foot and deformities which can present in the forefoot, mid-foot, hindfoot and ankle. An early sign on X-ray is widening of the space between the bases of the 1st and 2nd metatarsals (Fig. 3.18) Advanced changes show fragmentation, fracture, new bone formation, subluxation and dislocation. These changes often develop very rapidly, within a few weeks of the onset, and sometimes within a few days.

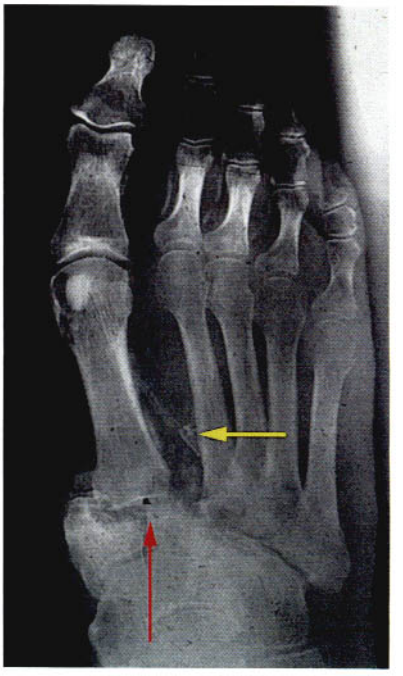


Fig. 3.18 There is a Lisfranc's tarsometatarsal joint dislocation (red arrow) with metatarsal bases shifted laterally. Yellow arrow shows vascular calcification.

CASE STUDY
Acute Charcot's osteoarthropathy with rapid onset of bony destruction and deformity

A 46-year-old man with type 1 diabetes of 33 years' duration, end-stage renal failure treated by renal transplantation and severe neuropathy, received regular foot checks under a renal foot study protocol. Three days before he went on holiday to the Channel Islands his feet were routinely checked and nothing abnormal was discerned. Two weeks later he came to the clinic on his return from holiday to report that his foot was 'a little swollen'. He reported no trauma to the foot, but had been walking more than usual on cobbled pavements. The foot was red, 5°C hotter than the contralateral foot and very

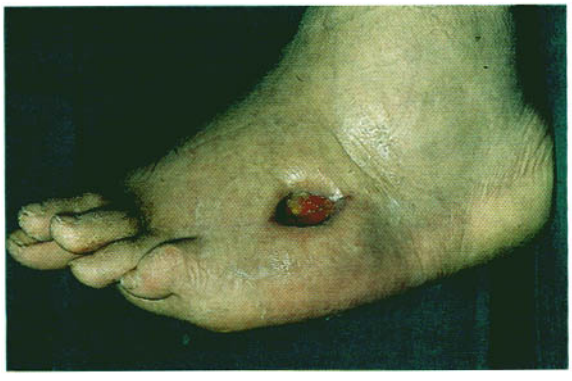


Fig. 3.19 The Charcot foot with dorsal swelling and a healing surgical wound where infection was drained.

swollen. X-ray revealed a Lisfranc's fracture-dislocation and he developed a rockerbottom foot. He was treated in a total-contact plaster cast for 6 months following which he wore bespoke boots to accommodate his deformity. The foot remained ulcer free apart from one episode of sepsis which was precipitated by dropping a heavy object on the foot. This led to a break in the skin with resulting infection on the dorsum of the foot which needed surgical drainage (Fig. 3.19).

Key points

- Charcot's osteoarthropathy can develop with great rapidity
- Severe injuries normally associated with gross trauma can develop in high-risk patients simply through walking or unperceived trauma
- Renal transplant patients have a very high risk of Charcot's osteoarthropathy
- Deformed feet can be successfully accommodated in footwear.

Bone and joint destruction can occur in any part of the foot or ankle, but the common presentations can be divided into forefoot, mid-foot and hindfoot. The forefoot involves the metatarsophalangeal and interphalangeal joints. The mid-foot involves the tarsometatarsal joints and the hindfoot includes the ankle and subtalar joints. Each region of the foot has specific clinical presentations of bony destruction or deformity.

Forefoot

This presents with generalized swelling of the forefoot and osteonecrosis of the metatarsal heads. It is rare for a significant structural deformity to develop in forefoot Charcot's osteoarthropathy. The resorption of the distal metatarsal bones giving 'sucked candy' appearances is in our experience usually associated with chronic ulceration and infection.

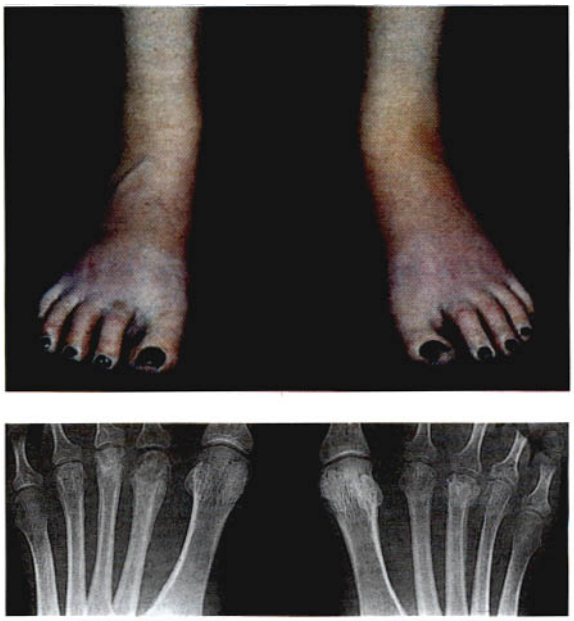
CASE STUDY

Acute Charcot's osteoarthropathy of the forefoot

A 21-year-old woman with type 1 diabetes of 15 years' duration developed painless swelling of both forefeet (Fig. 3.20a). There was no evidence of ulceration. X-ray revealed fragmentation and lucency of the 2nd, 3rd and 4th metatarsal heads of both feet (Fig. 3.20b). The patient was supplied with a wheelchair and underwent strict non-weightbearing for 4 weeks. The oedema gradually resolved. Deformity did not develop and the radiological changes stabilized.

Key points

- The radiological changes of osteonecrosis are typical of forefoot osteoarthropathy in the absence of foot ulceration and sepsis when sucked sugar candy appearances may be noted
- Charcot's osteoarthropathy may present spontaneously with no history of trauma



(a)

(b)

Fig. 3.20 (a) Swelling of the forefeet. (b) X-ray of both feet showing fragmentation and lucency of the 2nd, 3rd and 4th metatarsal heads.

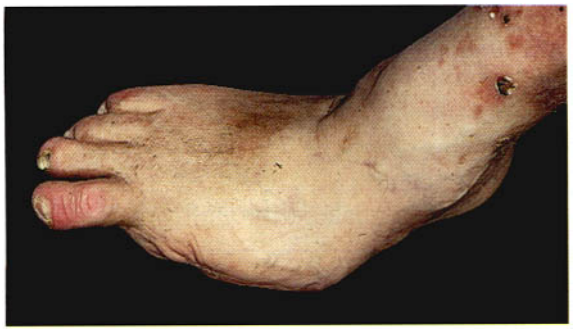


Fig. 3.21 Medial convexity of mid-foot developing after amputation of 1st toe.

- Young type 1 diabetic women are known to have osteopenia and are susceptible to fractures, including foot fractures.

Mid-foot

This is the commonest site of presentation of Charcot's osteoarthropathy and it is recognized clinically by the rockerbottom deformity and the medial convexity.

The medial convexity is associated with the classical Lisfranc's tarsometatarsal fracture-dislocation (Fig. 3.21) and also with talonavicular dislocation.

The rockerbottom deformity develops when there is disintegration and displacement of the cuneiforms or the proximal tarsal bones, resulting in collapse of the mid-foot. Rockerbottom deformity is frequently associated with plantar ulceration (Fig. 3.22).

Hindfoot

The early presentation is of a swollen ankle. Later, there is severe structural deformity and instability of the ankle joint. This can lead to a flail ankle on which it is impossible to walk. Ulceration can often develop over the malleoli.

Treatment of bone destruction/deformity

The aim of treatment is immobilization in a plaster cast (see Chapter 4) until there is no longer evidence on X-ray of continuing bone destruction, and the foot temperature is within 2°C of the contralateral foot, which can be measured with an infrared thermometer. When this is achieved, the foot has reached the stabilization phase.

Deformity in a Charcot's osteoarthropathy can predispose to ulceration, particularly on the plantar surface of the rockerbottom deformity. It may also occur on the medial convexity. These ulcers may become infected and lead to osteomyelitis. This may be difficult to distinguish from neuropathic bone and joint changes, as on X-ray, bone scan or magnetic resonance imaging, appearance is similar.

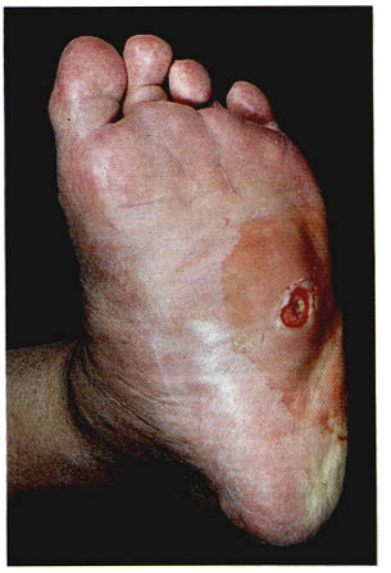


Fig. 3.22 Rockerbottom deformity of the mid-foot with early breakdown.

Radiological consolidation and stabilization

The foot is no longer warm and red. There may still be oedema but the difference in skin temperature between the feet is less than 2°C. The X-ray shows fracture healing, sclerosis and bone remodelling. The average amount of time spent in a cast by diabetic patients with Charcot's osteoarthropathy is 6 months but some patients may need a cast for over a year.

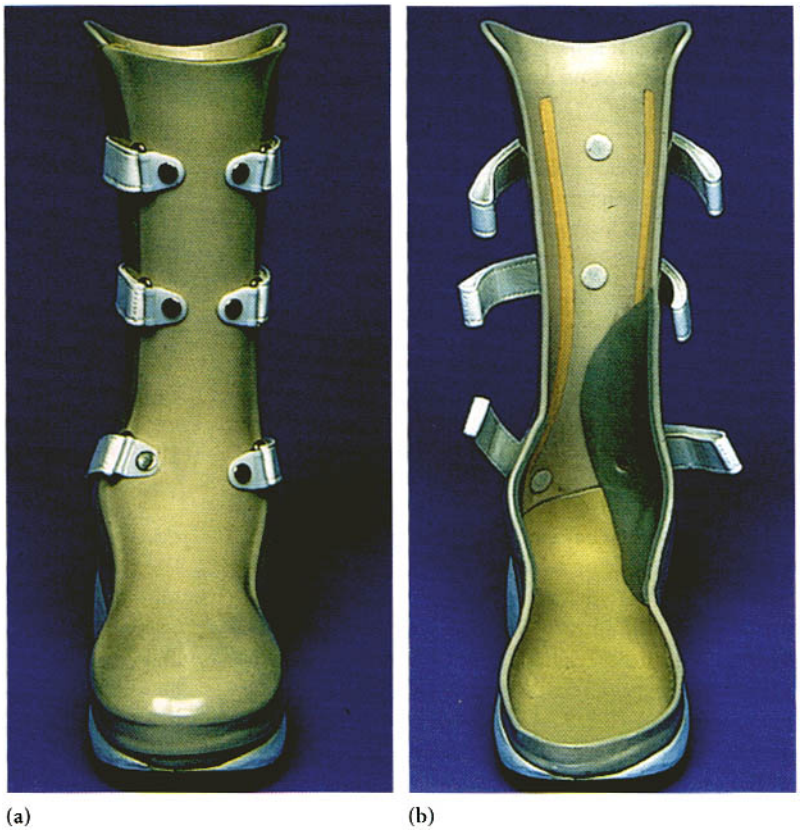
Rehabilitation is always necessary after a long period in a cast.

Forefoot

This usually stabilizes without bony deformity but patients may need moulded insoles in bespoke shoes.

Mid-foot

When the mid-foot has stabilized, the patient can now progress from a total-contact cast to a bivalved cast or Aircast (see Chapter 4) fitted with a cradled moulded insole. When the patient comes out of the cast there will be wasting of the calf muscles and joint stiffness. The physiotherapist must be aware of the dangers of reactivating the bony destruction phase by excessively rapid



(a) (b)

Fig. 3.23 (a) Anterior view of the CROW (with front piece). (b) Anterior view of the CROW with removal of the front piece to show interior. Extra internal padding (blue) has been added to cushion the vulnerable medial malleolar area.

mobilization or protracted weightbearing in the early stages of rehabilitation.

Too rapid mobilization can be disastrous, resulting in further bone and joint damage. Extremely careful rehabilitation should be the rule, beginning with just a few short steps in the new footwear. The patient rests for the remainder of the day and monitors the foot. If there is no increase in warmth, swelling and redness then he can walk a few more steps the next day, and very carefully build up to a reasonable amount of walking.

Finally, the patient may progress to bespoke footwear with moulded insoles. The rockerbottom foot with plantar bony prominence is a site of very high pressure. Regular reduction of callus can prevent ulceration. If ulceration does occur, an ostectomy (see Chapter 8) may be needed. If stabilization cannot be achieved by conservative means then it is possible to carry out operative procedures in the mid-foot.

Hindfoot

Hindfoot Charcot's osteoarthropathy may be difficult to stabilize. An attempt may be made with total-contact casting. The cast is used during the phases of acute onset and bony destruction, to reduce oedema and halt progressive bony changes and deformity. When these phases are over, in cases where the ankle has become unstable, or deformity of the hindfoot is difficult to accommodate in boots or shoes, a Charcot restraint orthotic walker (CROW) is useful, followed by an ankle–foot orthosis (AFO) with bespoke footwear.

The CROW

This is a bespoke bivalved total-contact device which externally fixates the ankle (Fig. 3.23a,b). Extra internal padding has been added to cushion the vulnerable medial malleolar area. The yellow corrugations contain ethyl vinyl acetate (EVA) to strengthen the device without increasing the bulk. All internal metal rivets have extra padding. The rigid, durable outer shell is constructed out of polypropylene and is lined with EVA. There is a bespoke moulded insole to accommodate any existing deformity and to redistribute plantar pressures. A

rockerbottom, crepe sole is attached to facilitate roll-off during walking. It is used after swelling is controlled and progressive destruction halted by total-contact casting.

AFO

The AFO is a device used to stabilize the foot and ankle. There are two main forms of AFO, the traditional conventional metal and leather calliper and the newer thermoplastic types which are more cosmetic.

CASE STUDY

Conservative management of Charcot's osteoarthropathy of the hindfoot

A 46-year-old male with type 1 diabetes of 40 years' duration presented with bilateral Charcot's osteoarthropathy. He was referred from a clinic 80 miles away and had been advised to have a right below-knee amputation. The left foot had stable mid-foot Charcot's osteoarthropathy with rockerbottom deformity: the right foot was hot with unstable hindfoot Charcot's osteoarthropathy with lateral talotibiofibular displacement. The Charcot's osteoarthropathy was diagnosed 3 years previously following a first ray amputation (Fig. 3.24). In view of the considerable distance he had to travel it was decided that casting him would be unwise. As an alternative, the treatment plan was to try to achieve stability by providing a CROW. After 5 weeks in the CROW, swelling had improved and the skin temperature difference between the two feet was less than 2°C. An AFO was manufactured containing a moulded EVA insole (Fig. 3.25a,b). The patient was advised to take 5–10 steps daily for the first week and then at each week to double the steps taken. At 10 months he was taking up to 50 steps daily and the temperature difference was still less than 2°C. At this stage there was clinical and radiological evidence that the hindfoot had stabilized. He remained in the AFO for his long-term orthotic prescription. There was no relapse and he has remained ulcer free.

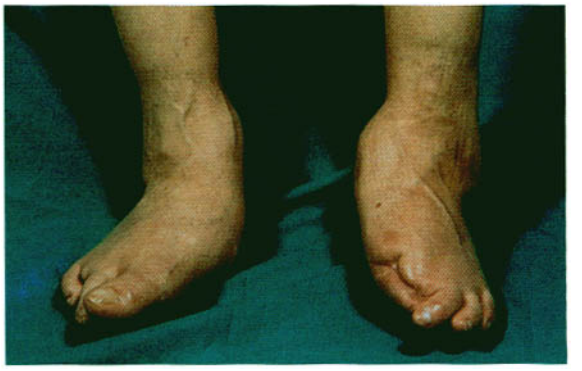
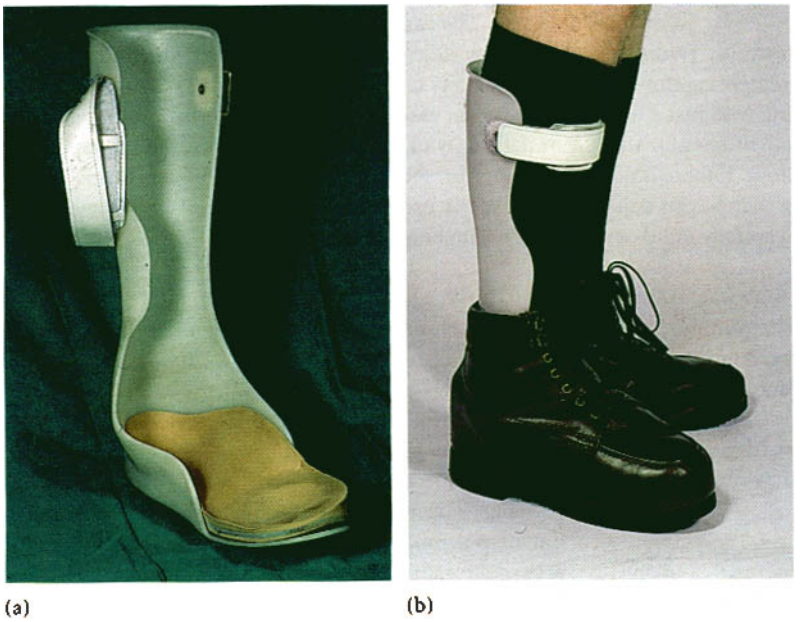


Fig. 3.24 Right foot shows stable mid-foot Charcot's osteoarthropathy; left foot shows unstable hindfoot Charcot's osteoarthropathy and first ray amputation.



(a) (b)

Fig. 3.25 (a) Ankle–foot orthosis (AFO) containing a moulded ethyl vinyl acetate insole. (b) Patient wearing AFO in bespoke boot.

Key points

- Charcot's osteoarthropathy can develop after a first ray amputation or other surgical procedure
- Casting should not be undertaken if patients cannot return to the casting clinic quickly when problems arise
- Unstable hindfoot Charcot's osteoarthropathy can be stabilized, in some cases, by conservative means using a CROW, and maintained long term in an AFO and bespoke footwear.

If conservative care is unsuccessful, and the patient has a 'flail ankle', which is often associated with intractable ulceration over the malleoli, then internal stabilization may be necessary, and this is discussed in Chapter 8.

Following reconstructive ankle surgery, the lower limb is usually managed in a cast for several months and then a CROW is supplied to maintain the stability of the hindfoot. When all is stabilized, temperatures have stayed down and patient is fully ambulant, an AFO provides long-term stability.

CASE STUDY

Surgical reconstruction of Charcot's osteoarthropathy of the hindfoot

A 61-year-old lady with type 1 diabetes of 40 years' duration developed a hot, red, swollen foot and ankle, and Charcot's osteoarthropathy was diagnosed. She was unwilling to wear a total-contact cast, but agreed to wear an Aircast. One month later she attended a wedding and discarded the Aircast for 1 day. She returned to the foot clinic the following week with an unstable flail ankle. She underwent surgical reconstruction (Fig. 3.26) and returned to the foot clinic in a non-weightbearing plaster cast which she wore for 4 months, using a knee scooter to off-load the foot. A CROW was made and she began to remobilize. She developed a swelling on the front of her ankle where a screw from her internal fixation metal plate had worked loose, and the screw was surgically removed. The joint remained stable and an AFO was made for her long-term management with bespoke footwear and insoles. She did not relapse and remained ulcer free.

Key points

- Reconstructive surgery can solve the problem of the unstable Charcot's osteoarthropathy of the ankle
- In the rehabilitation phase CROWs achieve stability and AFOs maintain stability
- The problem with removable casts during the phase of bony destruction is that some patients may remove them.

Fig. 3.26 X-ray of reconstructed Charcot hindfoot and ankle (courtesy of Dr M Myerson).

Follow-up

Patients need follow-up in a multidisciplinary diabetic foot service. Occasionally there may be a relapse in an already established but stabilized foot with Charcot's osteoarthropathy. This will present with erythema, swelling and warmth. The patient should be treated as if he was again in the acute phase.

Many patients will eventually develop Charcot's osteoarthropathy in both feet. All patients with Charcot's osteoarthropathy should therefore be taught to check their feet and ankles regularly for hot spots and danger signs.

Charcot's osteoarthropathy of the knee is rarely seen in diabetic patients: however, the authors have seen four cases in the knee and all patients previously had Charcot's osteoarthropathy of the foot.

Education for patients with Charcot's osteoarthropathy

Patients should be warned of the dangers of unprotected walking. Even one step without cast or brace can injure the foot irrevocably. They should be told always to wear the cast or walker, even in bed at night, otherwise the temptation to go to the lavatory in the middle of the night without bothering to don it may be too great. Patients should walk as little as possible: the more the foot is rested

and elevated the sooner it will recover. Patients should be advised to borrow or hire a folding wheelchair so that they can get out without overloading the foot. The foot should be elevated whenever possible, so the wheelchair should be fitted with an extending plank on which to support the leg.

The clinician should never underestimate how difficult a diagnosis of acute Charcot's osteoarthropathy is for patients. Following advice and keeping off the foot is certain to have profound effects on their daily life, at home and at work, socially and emotionally.

During the rehabilitation period the patient will be under particularly great stress, when he knows the foot is recovering and the urge to get back to a normal life will become very strong. This is a dangerous period since failure to rehabilitate slowly and gently can trigger the destructive phase into action again.

PRACTICE POINTS

- The majority of deformities in stage 2 can be accommodated in special footwear
- Callus is an important precursor of ulceration and should be treated aggressively, especially in the neuropathic foot
- The cold, pink, painful foot is an indication of severe ischaemia and requires urgent vascular intervention
- Patients must understand the implications of lack of protective pain sensation
- Education in trauma prevention is important
- Patients with painful neuropathy will need therapy of various modalities which should be delivered in a supportive environment
- Early diagnosis, immediate cast immobilization and careful rehabilitation are important for successful management of Charcot's osteoarthropathy.

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4 Stage 3: the ulcerated foot

A scratch . . . tis enough . . . t'will serve.
Ask for me tomorrow, and you shall find me a grave man.
(Romeo and Juliet, *III, i, William Shakespeare)*

PRESENTATION AND DIAGNOSIS

Stage 3 represents skin breakdown and ulceration. This is the pivotal point in the natural history of the diabetic foot. Throughout their lifetime 15% of patients will develop ulceration; 85% of amputations result from non-healing ulcers. Every break in the skin of the diabetic foot is a portal of entry for bacteria and has the potential for disaster. Feet at stage 3, both neuropathic and neuroischaemic, therefore need very rapid and active treatment.

CLASSIFICATION

The simple staging system differentiates between ulcers on neuropathic feet and ulcers on ischaemic feet. The basis of this classification is the presence or absence of ischaemia in the common background of neuropathy. The majority of ischaemic feet in diabetes will also have neuropathy and therefore we describe the ischaemic foot as neuroischaemic. However, there may be some ischaemic feet with minimal or no neuropathy and the ulcers in these feet are perhaps more accurately called ischaemic. For simplicity, we shall refer to them as neuroischaemic ulcers.

The simple staging system does not classify ulcers according to depth. It is difficult to assess the depth of an ulcer accurately, except where it is clear that it probes to bone, suggesting infection, which already has its own separate stage within the system. Describing ulcers as superficial can lead to false reassurance, as any ulcer, even a small shallow one, can deteriorate and become very serious in a short space of time. It is dangerous for diabetic patients or their carers ever to regard small superficial lesions as trivial because the destructive potential of a break in the skin cannot be accurately assessed by measuring its depth.

Although ulceration can present in many different ways, we believe the most relevant classification of ulcers is the simple division of ulcers on neuropathic feet or ulcers on neuroischaemic feet, as such a division has the most relevant and important implications for treatment. However, it is also important to recognize specific categories of ulcer within this simple classification.

Specific categories of ulcer

These include:
- Decubitus heel ulcers caused by unrelieved pressure
- Ulcers of Charcot's osteoarthropathy associated with rockerbottom deformity, medial convexity and hindfoot deformity
- Ulcers over the Achilles tendon
- Puncture wounds caused by standing on sharp objects
- Traumatic wounds, including burns
- Artefactual (factitial) ulcers caused deliberately by the patient
- Malignant ulcers.

The main part of this chapter will concentrate on the presentation and management of neuropathic and neuroischaemic ulcers, describing the multidisciplinary approach, with mechanical, wound, vascular, microbiological, metabolic and educational control. The chapter will finish with a discussion of the presentation and management of the specific categories of ulcers described above.

Neuropathic ulcer

Neuropathic ulcers are commonly found at the apices of the toes and on the plantar aspect of prominent metatarsal heads. Callus forms in these areas in response to high vertical pressures. This leads to inflammation,

further callus formation and tissue breakdown. Failure to remove callus regularly leads to ulceration. The neuropathic ulcer is usually painless. Pain associated with neuropathic ulceration may be the first symptom of infection.

Neuroischaemic ulcer

Ulceration in the neuroischaemic foot usually occurs on the margins of the foot. The first sign of ulceration is a red mark which blisters and then develops into a shallow ulcer with a base of sparse pale granulations or yellowish closely adherent slough. In ischaemia, there is often a halo of erythema around the ulcer where local blood vessels have dilated in an attempt to improve perfusion of the area (Fig. 4.1).

Pain associated with a neuroischaemic ulcer may be due to the ischaemia itself or to infection. The degree of pain will depend on the severity of concomitant neuropathy.

It is now necessary to classify the ulcer as neuropathic or neuroischaemic. This is achieved by:

- Taking a history
- Examination of the foot and the ulcer
- Investigations, including neurological and vascular status.

Taking a history

A short history of the ulcer should be taken to ascertain its cause, duration and previous treatment.

Examination

Foot examination should be carried out as described in the introduction. The ulcer should then be examined noting:

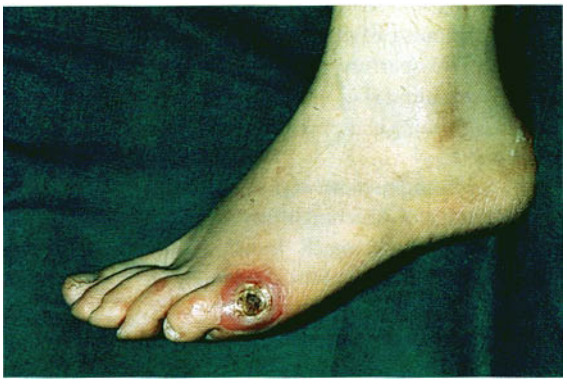


Fig. 4.1 An ischaemic ulcer on the margin of the foot with a halo of erythema.

- Site
- Size
- Appearance of the ulcer and surrounding tissues
- Discharge
- Associated swelling
- Tenderness
- Smell
- 'Probe-ability'.

The implications of these are discussed below.

Site

Ulcers on the plantar surface are usually neuropathic and ulcers on the margins of the foot are usually neuroischaemic. However ulcers can appear in other sites.

Interdigital ulcers can be caused by the toes being squeezed together in tight, ill-fitting shoes in both neuropathic and neuroischaemic feet.

Ulceration on the dorsal aspect of the toes is often associated with pressure from tight shoes in either class of foot.

Ulcers on the plantar aspect of the heel are usually caused by acute trauma, particularly treading on foreign bodies. Indeed, trauma can cause ulceration at any site in either class of foot.

Size

Any break in the skin, however small, can lead to disaster. However, larger ulcers usually take longer to heal.

Appearance of the ulcer and surrounding tissues

The colour of the base of the ulcer is important. A pink, clean, glistening ulcer bed is healthy. Variation in the colour of the ulcer bed may be significant: for example, a wound bed which is largely pink but has an area of grey discolouration often overlies a sinus. Many poorly perfused ischaemic wound beds are grey or yellow and sloughy. Moist green or yellow slough indicates infection and patients should be managed as described in Chapter 5.

Black tissue indicates necrosis and the foot should be regarded as at stage 5 and managed accordingly.

Neuropathic ulcers are usually surrounded by callus, which is often white and rubbery because it is macerated by discharge from the ulcer. The neuropathic ulcer may be almost completely covered over by callus.

Neuroischaemic ulcers may be surrounded by a small halo of thin, glassy callus.

Diffuse redness of the surrounding tissues may indicate infection in both neuropathic and neuroischaemic feet, especially if this is associated with swelling and purulent

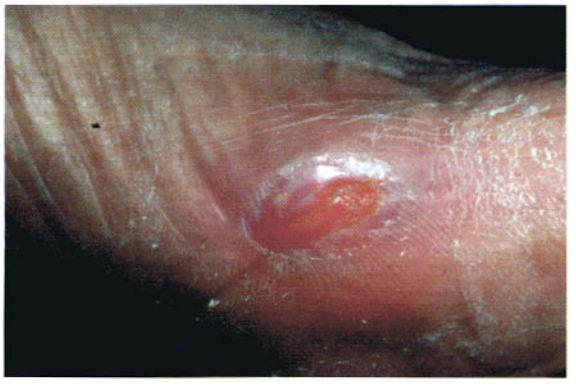


Fig. 4.2 This healing ulcer is surrounded by an area of shiny new pink and white epithelium.

discharge. A purplish colour indicates reduced oxygen supply to the tissues: this may result from ischaemia or severe infection or both.

Healing ulcers are surrounded by an area of shiny new pink and white epithelium (Fig. 4.2).

Discharge

Purulent discharge is indicative of infection. Clear or yellow-tinged viscous bubbly discharge from an ulcer which probes to bone may be synovial fluid indicating involvement of the joint. Increased amounts of clear discharge may be an early indication of infection.

Swelling

Swelling around an ulcer is usually suggestive of infection but may be related to ischaemia.

Tenderness

This should be elicited by gentle palpation and may be due either to infection or to ischaemia.

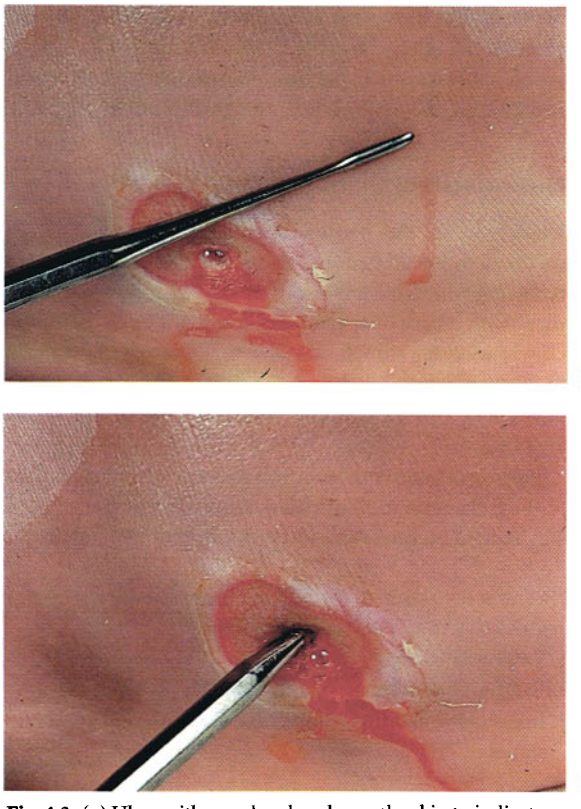
Smell

Any smell associated with an ulcer is suggestive of infection.

Probe-ability of the ulcer

The depth and dimensions of the ulcer are determined by probing, which is an important part of the examination. Probing reveals the presence of:

- Undermined edges where the probe can be passed from the ulcer under surrounding intact skin (Fig. 4.3a,b)
- Sinuses when the probe can be inserted deeper than in other areas of the ulcer bed and may reach tendon or bone. A sinus may not be immediately apparent, but may be sometimes disclosed by probing suspicious areas of the ulcer, which are a different colour to the remainder of the ulcer bed. These areas may also be less firm and resilient.



(a)

(b)

Fig. 4.3 (a) Ulcer with a probe placed over the skin to indicate position of sinus. (b) Probe inserted into sinus of same ulcer.

Some ulcers appear to pout, or stand proud of the surrounding epithelium. If such an ulcer is producing copious amounts of discharge there will often be a sinus present. The aperture of the sinus may be slit-shaped rather than round (Fig. 4.4).

When probing the practitioner should determine the following:

- The depth and breadth of the ulcer
- The depth and direction of any sinus
- Does the probe reach bone? If so, this suggests osteomyelitis and the foot is at stage 4.

Investigations

These should include neurological, vascular, laboratory and radiological investigations as described in Chapter 1.

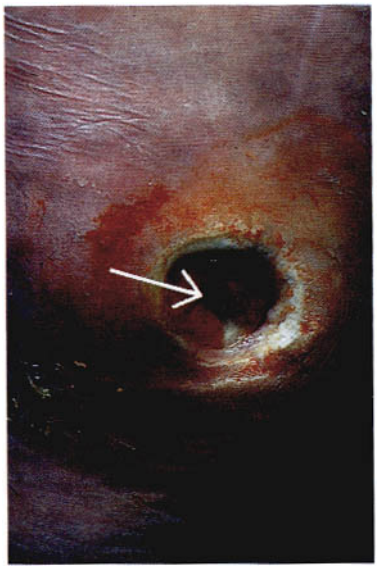


Fig. 4.4 This deep sinus has a slit-shaped aperture.

While it is not necessary to X-ray every stage 3 foot with a presenting ulcer, it may be advisable to do so in the following circumstances:

- When the history suggests that the patient may have trodden on a foreign body
- When the ulcer probes to bone, clinically suggesting osteomyelitis
- If there are clinical signs of infection
- When there is unexplained pain or swelling which may be related to neuropathic fracture or Charcot's osteoarthropathy
- When the ulcer has been present for longer than 1 month.

Radiological studies of diabetic feet have revealed a high prevalence of fractures in neuropathic patients and most of these had not been previously diagnosed.

Having completed the history, examination and investigations, it is possible to make a firm diagnosis of ulceration either in the neuropathic foot or the neuroischaemic foot. The characteristic features are described in Table 4.1.

Follow-up of ulcerated feet

In the short term, the ulcer will need close review at each visit. Signs of progress or deterioration should be looked for.

Signs that an ulcer is healing

- Ulcer bed becomes pink
- Ulcer bed becomes shiny with glistening granulations
- Dimensions of ulcer decrease with new epithelium around the edges
- Ulcer bed becomes drier with less discharge
- Swelling diminishes
- Ulcer becomes less painful
- Smell ceases
- There is no undermining
- Sinus depth reduces.

Table 4.1 Clinical features of neuropathic and neuroischaemic foot

Neuropathic foot	Neuroischaemic foot
Ulcer on plantar surface	Ulcer on margins
Heavy callus	Thin glassy callus or no callus
Warm foot	Cold foot
Pulses present	Pulses absent
$TcPo_2$ above 30 mmHg	$TcPo_2$ below 30 mmHg

Signs of deterioration

- Colour of ulcer bed changes
- Matt surface develops over ulcer bed
- Dimensions of ulcer become static or increase
- Ulcer becomes moist with increased discharge
- Local swelling develops or increases
- Foot becomes painful
- Malodour develops
- Ulcer becomes undermined.

In the long term, it should be remembered that patients with neuropathic feet may eventually develop ischaemia, and the classification should be repeated at every annual review.

MANAGEMENT

All of these components of multidisciplinary management are important in stage 3:

- Mechanical control
- Wound control
- Vascular control
- Microbiological control
- Metabolic control
- Educational control.

The aim is to heal ulcers within the first 6 weeks of their development. This is the time for aggressive management and is a window of opportunity that should be taken seriously. All diabetic foot ulcers should be referred for multidisciplinary care without delay so that the opportunity of

early healing is not wasted. The ulcer is a pivotal event on the road to amputation, and the diabetic patient with an ulcer on the foot is at great risk of infection, gangrene and loss of the leg. Because it is difficult to predict which ulcers will do well and which will do catastrophically, it is essential to organize optimal care for all ulcers.

When ulcers are healed, the foot is treated as stage 2 to prevent recurrence.

Neuroischaemic ulcers may be painful and thus pain control will also be considered.

Mechanical control

Ideally, ulcers must be managed with rest and avoidance of all pressure. However, total non-weightbearing is rarely practical and is difficult to achieve. For these reasons, ambulatory methods of achieving mechanical control are best.

In the neuropathic foot, the overall aim is to redistribute plantar pressures evenly, thus avoiding areas of high pressure which will prevent or delay healing.

In the neuroischaemic foot, the aim is to protect the vulnerable margins of the foot.

Thus, mechanical control will be considered separately in the neuropathic and the neuroischaemic foot.

Neuropathic foot

The most efficient way to redistribute plantar pressure is by the immediate application of some form of cast. If casting techniques are not available, temporary ready-made shoes with a cushioning insole can be supplied to off-load the site of ulceration. Alternatively, weight-relief shoes can be supplied, and felt padding can also be used. Additional off-loading measures are crutches, wheelchairs and Zimmer frames.

Moulded insoles in bespoke shoes are sometimes used to treat ulcers. However, shoes with insoles are not an efficient way to off-load diabetic foot ulcers. Their main function is to prevent recurrence.

Casts

Various casts are available and their use is governed by local experience and expertise. Techniques include:

- Total-contact cast
- Scotchcast boot
- Aircast (walking brace).

Total-contact cast

The total-contact cast (Fig. 4.5) is an extremely efficient method of redistributing pressure from the plantar surface of the foot. However, it is not without its complications, and should be reserved for patients whose ulcers have not responded to other treatments. It is a close fitting plaster of Paris and fibreglass cast, applied over minimal padding as follows.

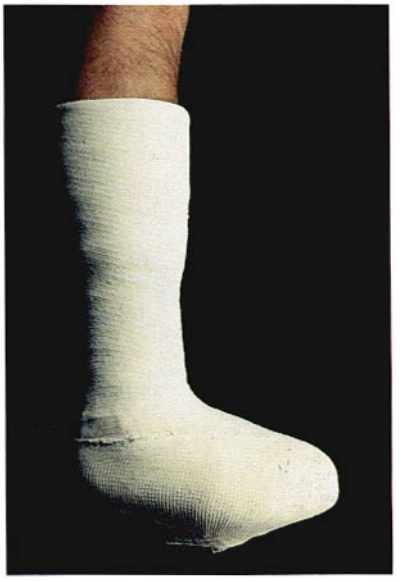


Fig. 4.5 The total-contact cast.

The casting procedure is as follows:

- When patients attend the casting clinic, they wear shorts or tracksuit bottoms otherwise they will need to unpick trouser seams
- A layer of stockinette is applied to the patient's lower leg. The length of the stockinette is twice the distance from knee to tips of toes (two-and-a-half times if the leg is plump) and the excess should be gathered up over the knee. When the cast is finished the excess stockinette is brought down to cover the cast and protect the contralateral leg from rubs
- The foot is held in a plantigrade position and any excessive creases in the stockinette around the ankle area are cut out and taped flat
- Small pieces of cast padding are inserted between the toes to keep them apart. The distal end of the stockinette is taped together
- A strip of 5 mm felt padding is applied over the tibial crest and circles of felt are applied over each malleolus and any other bony prominences, which may include the tuberosity of the navicular and the base of the 5th metatarsal

- A double layer of cast padding is applied to the entire lower limb, with three extra layers at the proximal end of the cast and three extra layers over the toes
- Fibreglass tape is applied around the foot and leg, starting at the top of the leg, 3 cm below the cast padding. The layer of tape is rubbed gently to accommodate the contours of the foot and leg. At least three layers of tape will be needed, and heavy patients will need up to six layers. Patients should be told not to touch wet fibreglass which can cause skin irritation
- The excess stockinette which was gathered up over the knee when casting commenced is rolled down to cover the outer fibreglass layer. Rubbing the stockinette provides a smooth finish
- Casts should be replaced after 1 week if reduction of oedema renders them loose
- The maximum period a cast should be left on without renewing is 1 month
- An emergency cast removal service should be available for patients if they develop problems with the cast.

The most common mistakes made when applying casts are:

- Cast comes too high up the leg, so that when the knee is bent the cast presses on the back of the thigh
- Cast is wrapped too tightly around the toes and border of the forefoot, causing pressure lesions
- Rough fibreglass outside layer is not covered with stockinette and rubs occur on the contralateral limb
- Cast is too lightweight for heavier patients and collapses
- Fibreglass is dented by pressure from finger tips—cast should be handled with the flat of the hands
- Foot is insufficiently dorsiflexed. If cast is applied to a rigid plantar flexed foot there should be compensatory building up of the heel area of the cast.

Advantages of the cast include:

- Redistributes pressure very evenly over the sole; 30% of pressure is transferred further up the leg in a 'coning' effect
- Enforces compliance—the patient cannot remove it
- 'Ball and chain'—patients walk less in a heavy cast
- Reduces oedema
- Ulcers heal very quickly—mean healing time of 6 weeks.

Disadvantages of the cast include:

- Cannot be removed so ulcer progress cannot be checked daily
- Heavy and reduces mobility
- Patient may not drive a car in a cast unless fitted with suitable controls and with permission from insurers
- A few patients develop 'cast phobia' and will not wear them. One of our patients borrowed her neighbour's electric saw and removed her cast (but fortunately not her leg). Another used a claw hammer to bash the cast off and sustained trauma to her leg
- Iatrogenic lesions (rubs, pressure sores, infections) may be undetected
- Leg may develop 'cast disease' from prolonged immobilization, e.g. muscle wasting, weakness and osteopenia
- Leg length disparity may cause discomfort and problems with knee, hip and spine (this can be prevented with a shoe raise on the contralateral side)
- Danger of fracture and the development of a Charcot foot when cast discontinued without careful rehabilitation
- Frail patients may suffer falls
- Casts are unsuitable for patients with ulcers or skin eruptions on the leg
- Problems may also arise if patients fail to care for cast.

CASE STUDY

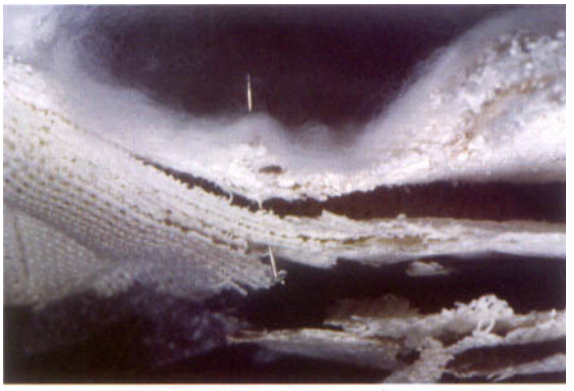
Problems with total-contact cast

A 63-year-old female with type 1 diabetes mellitus of 20 years' duration, developed an acute Charcot's osteoarthropathy which was treated in a total-contact cast. She was a very successful milliner who was currently making hats for Royal Ascot Races, and was working from a studio at home with a team of assistants. She failed to attend for her 1-week cast check. We telephoned her and she said that the cast was fine but she was frantically busy making hats and really did not want to come in. We persuaded her to attend. When she came the following day she looked tired and unwell, and the cast was in poor condition with dehiscence of the lamina. Before the cast was removed she volunteered that she 'might have dropped some pins down it'. On cast removal she had a deep necrotic ulcer on the plantar surface of the foot caused by a pin which had penetrated the inner layers of the cast (Fig. 4.6a,b) and punctured her foot. She was admitted to hospital the same day (she booked a private room and continued to direct her team preparing for Ascot over the telephone). She underwent surgical debridement and the foot healed in 2 months.

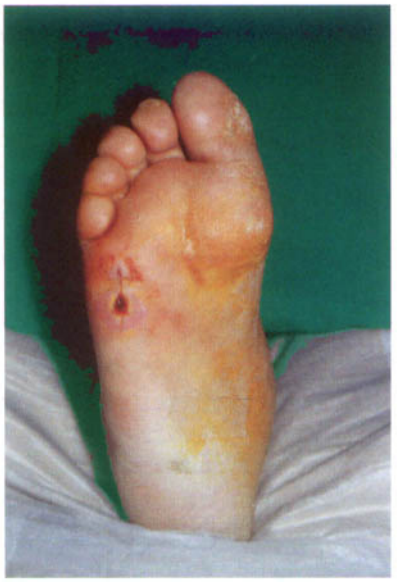
Key points

- Regular cast inspections are essential
- A patient's report on the condition of the cast may be unreliable
- A patient's occupation may be hazardous without special precautions.

Rarely a total-contact cast causes an eczematous eruption.



(a)



(b)

Fig. 4.6 (a) Cast in poor condition with pin protruding. (b) Site of ulcer on plantar surface of 5th ray indicating area where pin penetrated the foot.

CASE STUDY

Eczematous eruption within cast

A 42-year-old neuropathic man with type 1 diabetes of 40 years' duration was given a total-contact cast for acute Charcot's osteoarthropathy. After 3 weeks he developed an eczematous eruption of the whole area covered by cast and some areas on the other leg and arms.

He underwent patch testing by dermatologists, including testing to epoxy resins. These tests were all entirely negative, making a contact eczema rather unlikely, although it is possible that he was allergic to another component of his plaster.

Our solution was to enclose the cast, inside and out, with three layers of stockinette to reduce any possibility of direct contact of the patient's skin with any other component of the cast, and his skin condition improved.

Key points

- Eruptions beneath the cast may be due to allergy to cast components
- A cast with extra protective layers of stockinette may be useful when patients develop eruptions under the cast
- Patch testing may be helpful.

Rehabilitation programme. Once the ulcer is healed, the patient should be assessed for moulded insoles and bespoke shoes.

He should remain in the cast until the new shoes are ready.

It is helpful if the orthotist attends the casting clinic so that footwear preparations can be underway during the last weeks of healing and unnecessary delays are avoided.

When the patient has been healed for 1 month, the rehabilitation programme can commence, as follows:

- The cast should be bivalved and made removable by cutting out the front, padding the raw edges of both pieces with Elastoplast, and holding the two pieces in place with Velcro strapping or bandage. The padding should be incorporated into the cast
- The patient may walk for five steps without the cast on the first day of rehabilitation, wearing his new shoes and walking within the house. He should then replace the cast
- The foot should be checked for red marks on the following day. If all is well, the patient can walk for 10 steps without the cast
- Very gradually he can build up the amount of walking he does, within the house at first and then outside, but always wearing the special shoes and checking the foot every day for problems
- If blistering or ulceration occur he should return to the foot clinic at once
- Patients may require physiotherapy to build up wasted muscles.

Education for patients in casts. It is dangerous to enclose an insensitive leg and foot in a closed cast without careful education of the patient as follows.

- Keep your cast clean and dry. Walk as little as possible. If you go out in wet weather cover the cast with a plastic bag or cling film, but remove these as soon as you are under cover again

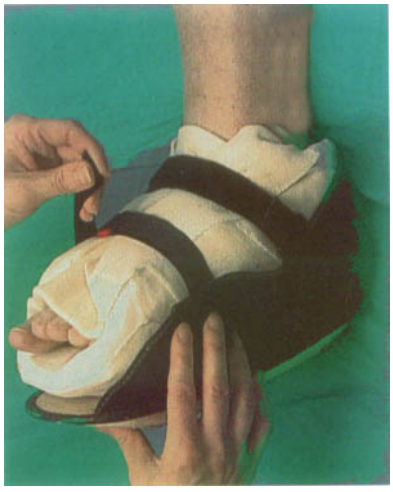


Fig. 4.7 The Scotchcast boot.

- You can obtain a plastic cast protector for bathing and showering
- Check your cast every morning and evening for:
  Cracks
  Soft areas
  Dirty areas
  Stained areas (where blood or pus has come through the cast from the inside)
  Wet areas
  Bad-smelling areas
  If any of these occur, please telephone the diabetic foot service immediately and come in for a cast check
- Check your temperature with a clinical thermometer every morning and evening. If it rises above 37.5°C, ring the clinic and come in for a check
- Check your blood glucose every morning and evening. If it rises above 15 mmol/L (270 mg/dL) please ring the clinic and come in for a check
- If you feel unwell, tired, hot, achy, shivery or have flu-like symptoms, please ring the clinic and come in for a check
- Never poke or pour anything down your cast
- Never try to remove the cast yourself
- Do not go a long distance from home
- Do not fly as there is increased danger of deep vein thrombosis.

'Scotchcast' boot (Fig. 4.7)

Not every patient is suitable for total-contact casting, and the Scotchcast boot is a useful alternative. It can also be used for patients with neuroischaemic foot or cast phobia.

It is a simple removable boot made of stockinette, cast padding, felt and fibreglass tape. Originally the fibreglass tape used was of the Scotchcast brand name: other fibreglass casting tape can, however, also be used. The boot is effective in reducing pressure on the plantar surface and the margins of the foot. It is made as follows:

- A layer of stockinette is applied to the lower limb from mid-calf to 10 cm distal to the toes
- One piece of 7-mm felt is applied to the sole of the foot extending to the tips of the toes, 5 cm up the back of the heel and 2.5 cm up each side of the foot. Lateral borders should not be higher than the top of the foot. Triangles are cut out of the felt so that it sits snugly around the heel
- Cast padding is wrapped loosely around the foot over the felt
- Three strips of fibreglass tape are cut and overlapped longitudinally so that they cover the sole of the foot. More fibreglass tape is then wrapped around the foot over the strips, keeping well within the area covered by cast padding
- The fibreglass is trimmed away below the malleoli, round the back of the heel and along the sides of the foot below the level of the felt. The fibreglass covering the dorsum of the foot is lifted away, leaving behind as much cast padding as possible. Any sharp corners of fibreglass are rounded off
- The stockinette is folded back over the foot from each end
- The entire boot is wrapped round with Elastoplast tape.

At this stage the boot cannot be removed by the patient. If a removable boot is wanted, the procedure is as follows:

- The dorsal area of the boot is cut open from toes to ankle, through all the layers of cast padding and stockinette, and the boot is removed
- The raw edges where the cut was made are sealed with Elastoplast
- A felt tongue is made as extra protection to the dorsum of the foot to avoid rubbing by the straps
- Ready-made straps or Velcro fastenings over the mid-foot and high on the foot should be adjustable to accommodate oedema
- A large sock worn over the cast offers extra protection to the toes. The boot can be worn inside a cast sandal.

Removable cast walkers

There are several models of removable cast walker. We have first-hand experience with the prefabricated pneumatic walking brace called Aircast (Fig. 4.8).

The Aircast is a bivalved device and the two halves are joined together with strapping. The cast is lined with four

Fig. 4.8 Orthowedge shoe to off-load the forefoot.

air chambers which are inflated with a hand pump to ensure a snug fit. Care must be taken that the cast does not impinge upon the margins of the foot. The Aircast has a rocker sole.

Flat-bed insoles are supplied with the Aircast although they can be replaced by bespoke moulded insoles.

Advantages of the Aircast include:

- Practitioners can view the wound and inspect the foot. This is important for practitioners who fear iatrogenic lesions or undetected infections
- Avoids the labour-intensive programme required for plaster casting
- Cast provides an immediate off-loading device
- Cast fits either foot and can be retained for future problems after one episode of ulceration is healed.

Disadvantages of the Aircast include:

- It will not accommodate severe deformity
- The cast is easily removable which renders it unsuitable for some patients who may use it only intermittently
- Aircasts should not be issued to very frail and unsteady patients who might fall and injure themselves.

CASE STUDY
Fall in an Aircast

A 40-year-old male with type 1 diabetes of 30 years' duration, proliferative retinopathy treated with laser photocoagulation, sensory neuropathy and autonomic neuropathy including postural hypotension, developed an acute right mid-foot Charcot's osteoarthropathy. Because of a previous episode of severe sepsis he was reluctant to wear a total-contact cast, but agreed to wear a removable Aircast. He suffered a fall in his home where the kitchen floor was covered with shiny linoleum and X-ray revealed a fracture of the tibial plateau which progressed to a Charcot's osteoarthropathy of the knee. A plaster of Paris cylinder was applied for 6 months, followed with a knee brace.

Key points

- Postural hypotension coupled with impaired proprioception makes patients unstable
- Unstable patients risk falls if they walk on shiny surfaces in a cast walker. However, this drawback also applies to total-contact casts and 'Scotchcast' boots
- Charcot's osteoarthropathy of the knee is rare. The authors have seen four cases, all of whom had previous Charcot's osteoarthropathy of mid-foot or hindfoot on the same side as the affected knee.

Temporary ready-made shoes

When it is not possible to provide a cast, ready-made temporary shoes with cushioning insoles that can accommodate dressings, or weight-relief shoes are helpful.

Dressing shoes

- The Darco shoe provides closed toe protection and room for bulky dressings. It can also be fitted with a Plastazote or bespoke moulded insole
- The Dru shoe is a Plastazote shoe which has two removable Plastazote insoles of medium and low density which mould to heat or pressure for a customized fit. It has Velcro fastening and can accommodate wound dressings.

Weight-relief shoes

- Orthowedge: this shoe off-loads pressure from the metatarsal head and toes using a rockerbottom wedge design
- Forefoot relief shoe: this transfers weight from forefoot to hindfoot with 10 degrees of dorsiflexion built into the shoe. A semi-rigid heel counter provides stability
- Heel relief shoe: this eliminates weightbearing on the posterior end of the foot, which is put into a plantar flex

mode to facilitate off-loading of the heel area. Weight is transferred from heel to mid-foot and forefoot

- Half shoe: the front area of the sole is 'cut away' to relieve pressure on the forefoot for forefoot ulceration and the posterior area of the sole is 'cut away' to relieve hindfoot pressure if the ulcer is on the heel. They are available off the shelf but can be customized.

Felt padding

Felt padding may be used to divert pressure from ulcers but can prevent complications from being detected if the padding covers a large area. Felt padding should be lifted regularly and should not be used as a substitute for good footwear and insoles.

The felt used is made of sheep's wool with a hypoallergenic adhesive backing, and is similar to the felt used within total-contact casts and Scotchcast boots.

Crutches

Young and active patients with neuropathic ulcers may do well with crutches as an adjunct to other pressure-relieving techniques. However, patients with impaired joint position sense or postural hypotension may be unsteady on crutches. Many patients with diabetes of long standing, especially the elderly, do very poorly with crutches and are prone to falls. It is important to check for Romberg's sign, before dispensing crutches. When asked to stand with a narrow base of support and then to close their eyes neuropathic patients may lose their sense of balance. If this is so, then the test is positive and the patient should not be given crutches. Patients with neuropathy of the hands or Dupuytren's contracture may find hand-held crutches difficult to manage. Untrained patients can risk falls especially on stairs, so crutches should always be fitted by a health-care professional. Care should be taken to avoid nerve compression injuries to the arms, to which diabetic patients may be particularly susceptible.

Patellar tendon weight-relieving orthosis

In some patients a total non-weightbearing patellar tibial brace allows patients a moderate degree of mobility while still allowing non-weightbearing status to the limb.

Pressure relief ankle–foot orthosis (PRAFO)

This is a custom-fitted AFO suitable for the treatment of decubitus heel ulcers. It is discussed fully under special ulcers.

Ankle–foot orthosis (AFO)

The AFO is a device used to stabilize the foot and ankle. There are two main forms of AFO, the traditional conventional metal and leather calliper and the newer thermoplastic types which are more cosmetic. This bespoke device is moulded to a plaster cast of the limb.

Walking stick

These must be the correct length and should have a non-slip cover on the end.

Wheelchairs

A wheelchair which is used constantly is the best off-loading device of all. Self-drive models are better than chairs which can only be pushed. An extending platform can be attached to elevate the foot. A lightweight folding wheelchair can be of great help in achieving maximum off-loading and still enabling the patient to carry on with social activities. However, many patients become depressed at the idea of having a wheelchair and feel there is a stigma attached to their use.

Zimmer frame

These are exceedingly useful stability and off-loading aids: unfortunately they are associated with elderly and frail patients and some young patients will not use them. However, many patients who cannot cope with crutches find that a Zimmer frame is more helpful, being lightweight and providing more stability.

CASE STUDY
Adaptation of a Zimmer frame

A 54-year-old lady with type 2 diabetes of 2 years' known duration, developed a plantar ulcer over a rockerbottom deformity resulting from a mid-foot Charcot's osteoarthropathy and was admitted to hospital for surgical debridement. The resulting defect measured 6 by 4 cm. She was very anxious to return home without delay. She was given a Zimmer frame and a wheelchair, and discharged home with strict instructions to rest the foot. She arrived at the diabetic foot clinic for her first postdischarge visit with a simple adaptation of the Zimmer frame made by her husband. A plumber, he had used bent copper tubing to support a cushioned plywood shelf on which she rested the knee on the side of the ulcerated foot, so as to ensure that the foot was kept off the ground. When congratulated, he modestly said that he'd only done it so that she could do the washing up!

Moulded insoles

These are mainly used to prevent recurrence of ulcers. They are designed to redistribute weightbearing away from vulnerable pressure areas and at the same time provide a

suitable cushioning and total contact with the sole. These insoles may occupy too much space for them to be accommodated in anything but bespoke shoes, although an extra-depth stock shoe can sometimes accommodate the insole if the foot itself has a reasonably normal shape.

A plaster cast is taken of the foot to represent the overall contours including the sole. The cast is filled with a foam to make a last, over which the insoles are moulded, and shoes constructed.

A variety of polyethylene foams, microcellular rubbers and ethyl vinyl acetate (EVA) foams are used to construct moulded insoles which are usually made of two or three layers of different densities, with the most compressible at the foot–insole interface.

Materials

Closed cell polyethylene foam, e.g. Plastazote:

- Easily mouldable
- Cushions
- Bottoms out.

Open cell polyurethane, microcellular rubber, e.g. Poron, or closed cell, rubber-like polymers, e.g. Neoprene:

- Not mouldable
- Excellent shock absorption
- Good long-term resilience, and will not bottom out.

EVAs, e.g. Nora or Evalon range of different densities of EVAs:

- Mouldable
- Resilient
- Elastic.

Design

Various designs of moulded insoles are in use, including a design that forms a cradle by extending the insole up the sides of the foot.

Recently, EVA insoles have been used. These insoles have a top layer of low-density EVA for cushioning, followed by two to four layers of medium-density EVA and a base layer of high-density EVA, with the most dense and rigid layer acting as a cradle. Under particularly high-pressure areas, areas of the insole can be excavated out to form a 'sink' which is filled in with pressure-relieving material (Fig. 4.9).

Alternatively, the Tovey insole uses a high-density Plastazote material for the cradle. Pressure areas are marked on this and these areas are cut away to be filled in with Neoprene cushioning. The whole cradle is covered with an upper layer of Neoprene cushioning.

Composite moulded insoles can also be made, with an upper layer of low-density Plastazote and a lower layer of polyurethane rubber.

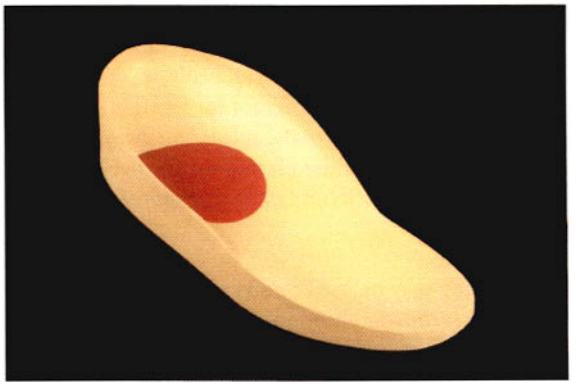


Fig. 4.9 Cradled insole with excavated sink filled with Neoprene over the mid-foot to accommodate plantar deformity.

Bespoke shoes

This footwear is custom-made for the patient and involves the production of a last which is specific to that individual's foot. Bespoke shoes can house cradled insoles. The shoes may be adapted to redistribute pressures further, and this can be carried out with rocker soles and metatarsal bars.

When first issued these shoes will need to be worn for short periods only. Their main role is prevention of recurrence.

CASE STUDY
Footwear-related blisters

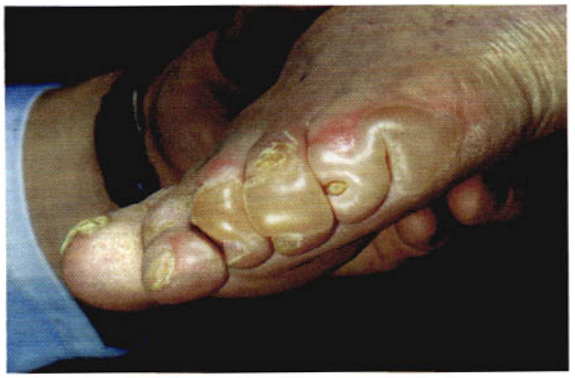
A 72-year-old lady with type 2 diabetes of 10 years' duration, with a rockerbottom Charcot foot treated in a total-contact cast, was issued with a new pair of special shoes. She was instructed to wear them for only 5 min/day at first, but went shopping and wore them for 2 h. She developed blisters on the side of her foot with a halo of erythema (Fig. 4.10a). The blisters were opened and drained in the diabetic foot clinic. Sterile dressings were applied and lifted every day to check for deterioration. The blisters were dry and healing well within 2 weeks (Fig. 4.10b) and fully healed within 2 months.

Key points

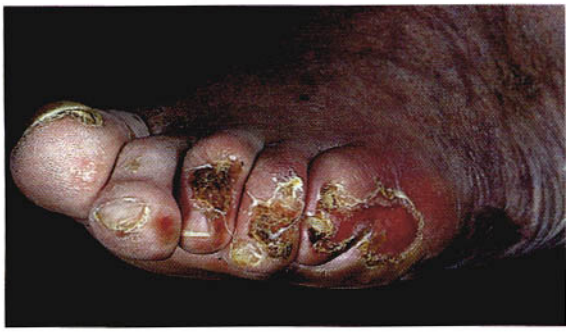
- Patients should receive very careful education about the timescale for rehabilitation
- New shoes should only be worn for periods of a few minutes per day, building up gradually to full-time use.

Neuroischaemic foot

Ulcers in neuroischaemic feet usually develop around the margins of the foot. Revascularization is the definitive



(a)



(b)

Fig. 4.10 (a) Bullae over lesser toes caused by footwear. (b) The bullae are dry and healing well after 2 weeks.

treatment, although it is still important to off-load the ulcer. A high-street shoe that is sufficiently long, broad and deep, and fastens with a lace or strap high on the foot, may be all that is needed to protect the vulnerable margins of the foot and allow healing. Neuroischaemic ulcers may be prevented from healing because the patient wears tight shoes or slip-on styles.

It may be necessary to provide special footwear. In the first instance, a temporary shoe such as a Darco may be used if dressings are bulky. Alternatively, a ready-made stock shoe which is wide fitting may be suitable. The Scotchcast boot may also be used, when ulcers are large, for postsurgical feet or for feet which fail to respond to treatment in footwear. Crutches and Zimmer frames may be useful for the neuroischaemic patient as described above for the neuropathic patient.

Wound control

Wound control of the neuropathic and neuroischaemic ulcer is based upon:

- Sharp debridement of the ulcer
- Dressings
- Advanced wound healing products
- Supplementary wound healing techniques
- Topical therapy.

Sharp debridement of the ulcer

We prefer sharp debridement as the ideal method. We do not favour autolytic, enzymatic or chemical debriding techniques.

Debridement of the neuropathic ulcer

Debridement is the most important part of wound control. It is not possible to assess a neuropathic ulcer properly without removing all associated callus. Sometimes the presence of the ulcer will only be determined following removal of callus which exposes a hitherto unsuspected lesion.

If an ulcer is suspected the patient should always be warned before the overlying callus is removed because he may otherwise feel that it was the callus removal that caused the ulcer.

Rationale

- Removes callus, thus lowering plantar pressures
- Enables the true dimensions of the ulcer to be seen
- Stimulates ulcer healing
- Removes any physical barrier to growth of new epithelium across the ulcer from the margin
- Prevents callus from sealing off an ulcer, which would prevent drainage and promote infection
- Enables drainage of exudate and removal of dead tissue (this renders infection less likely by reducing bacterial load and removing material which is a suitable growth medium for bacteria)
- Enables a deep swab or deep tissue to be taken for microscopy and culture
- Encourages healing by converting a chronic ulcer into an acute ulcer.

The debridement procedure

- Remove all callus surrounding the ulcer with a sterile scalpel
- When debriding the ulcer bed, work from the middle outwards: this carries debris and bacteria away from the ulcer bed
- Cut away all slough and non-viable tissue. It is helpful to grip the material that is to be cut away with a pair of forceps and to apply gentle traction so that the material to be cut is under tension. It is difficult to remove macerated callus or slough evenly and precisely, unless tension is applied (Fig. 4.11a–d)
- The forceps are additionally useful as one arm can

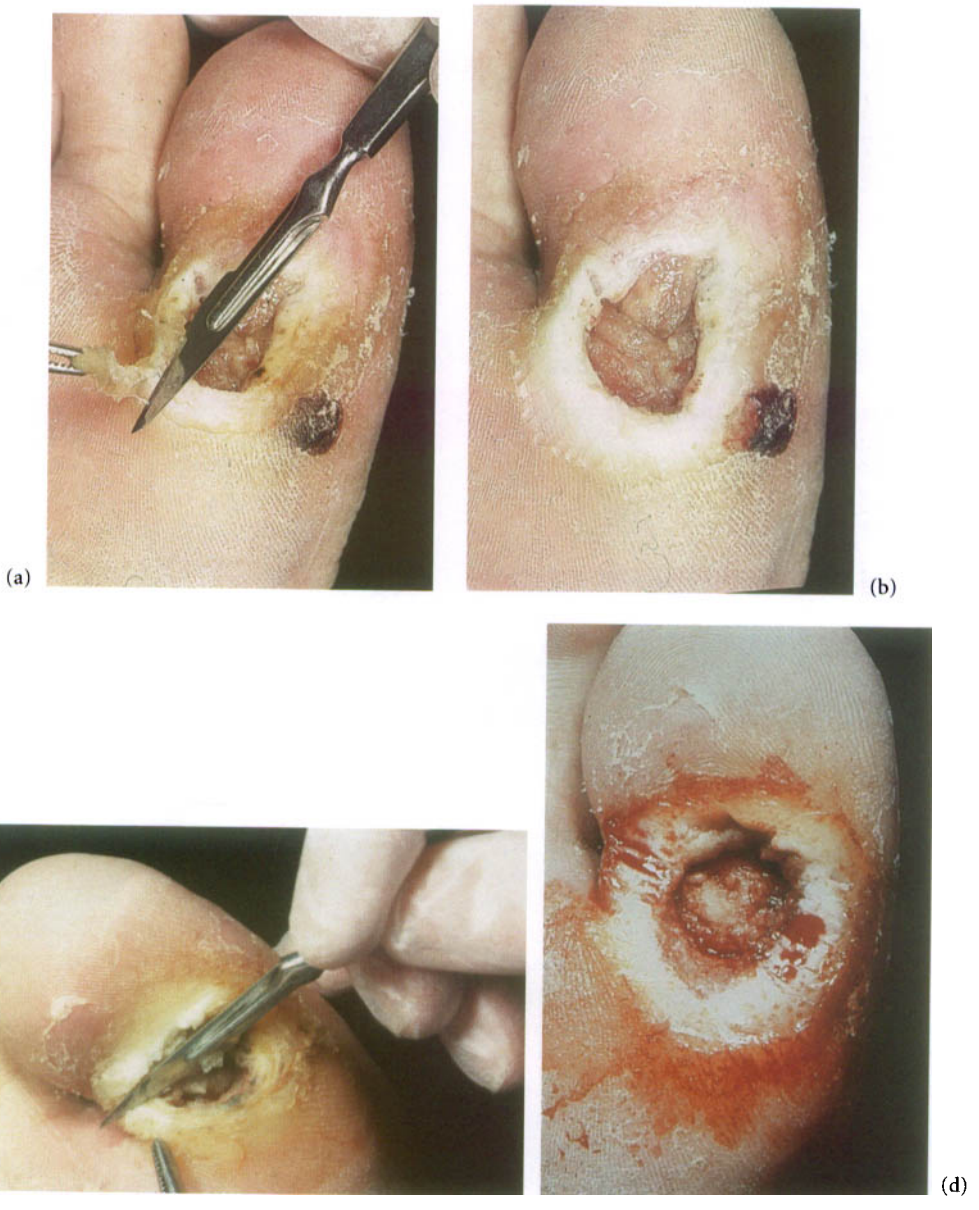


Fig. 4.11 (a) Neuropathic ulcer surrounded by callus. Callus is grasped with forceps and cut with a scalpel. (b) Neuropathic ulcer after debridement. (c) Further neuropathic ulcer. Gentle tension is applied with the forceps to keep callus taut while it is being cut. (d) Neuropathic ulcer has been debrided down to healthy bleeding tissue.

be used as a probe to explore the dimensions of the ulcer

- Probe ulcer. If the probe reaches bone this is suggestive of osteomyelitis and the foot should be regarded as a stage 4 foot
- Clean ulcer with sterile normal saline
- Take deep swab/tissue samples (see Chapter 5 for details) and send for culture without delay
- Apply sterile dressing held in place with light bandage, which should not be wrapped too tightly or encircle the toe
- Review at regular intervals (ideally weekly) and repeat the procedure

- Always ensure the patient is reminded that if problems develop he should return to the clinic immediately.

Control of bleeding

Neuropathic feet can bleed with considerable exuberance. Have gauze swabs and calcium alginate to hand. Most bleeding will stop with pressure. Apply pressure over a swab damped with saline as when the swab is removed it will be less likely to dislodge the clot if it is damp. When debriding around an ulcer apply digital pressure locally to a bleeding point while still continuing to debride with the other hand, or apply a damp swab under pressure from bandage or tape to free both the operator's hands for further debridement.

When patients go home after bleeding is staunched, they can be given extra gauze and plaster to apply on top of the clinic dressing if bleeding strikes through.

If patients are prone to bleeding after debridement they should rest and elevate the foot at home for the next 24 h.

Before debriding the neuropathic foot, ask the patient whether he is on warfarin or heparin or has any bleeding disorder, in which case debridement should be cautious.

CASE STUDY
Bleeding after debridement

An 89-year-old man with type 2 diabetes of 29 years' duration was taking warfarin. He had dry necrosis of the apex of his right hallux and regularly underwent gentle debridement in the diabetic foot clinic. On one occasion, on the day following debridement, his family noticed that blood was seeping through the dressings on his hallux. Despite rest and elevation the foot continued to bleed and the patient attended casualty. The bleeding was staunched by applying a calcium alginate dressing with digital pressure and elevation of the limb.

Key points

- Some feet bleed with alarming rapidity when debrided
- Application of topical calcium alginate dressing assists clotting
- In severe bleeding the limb should be elevated and a pressure pack bandaged on
- No patient should be sent home until bleeding is under control
- Patients should be warned that if the foot starts to bleed again when they arrive home after a debridement, they should not remove the clinic dressings but should elevate the foot, bandage additional dressing material over existing dressings, and seek medical help if the bleeding does not stop.

One of our patients developed severe bleeding at home after walking barefooted to the bathroom at night unprotected except for his ulcer dressing, and needed hospital treatment. Some neuropathic patients develop calcification of veins and arteries in the lower limb, and the pressures of walking unprotected had fractured a small calcified vein in his foot.

Debridement of the neuroischaemic ulcer

Rationale

- Enables the true dimensions of the ulcer to be seen
- Drainage of exudate and removal of dead tissue renders infection less likely
- Enables a deep swab to be taken for culture
- Removal of the surrounding halo of glassy callus (if present) prevents it from catching on the dressing and causing tissue trauma.

The debridement procedure

- Vascular status should be quantified by measuring the pressure index before debridement because only very cautious debridement should be performed if the pressure index is below 0.5
- Some neuroischaemic ulcers develop a halo of thin glassy callus which dries out, becomes hard and curls up. It is necessary to smooth off these rough areas with the scalpel as they can both catch on dressings and cause trauma to underlying tissues
- If a subungual ulcer beneath a thickened toe nail is suspected (revealed by pain, discolouration or maceration of the nail plate or exudate escaping from the periungual tissues) the nail should be very gently cut back, or layers of nail can be pared away with a scalpel to expose or drain the ulcer
- If the foot is very sensitive it may be necessary to anchor the area of tissue being debrided with forceps so that tissue can be cut away without pulling on the wound bed. This also avoids painful dragging of the scalpel blade through slack tissues. It is very important to reassure the patient that the operator will stop if debridement is painful
- It is very unwise to inject local anaesthetic into an ischaemic foot
- If a neuroischaemic ulcer bed is proud of the surrounding tissues and surrounded by thin, tightly bound down glassy callus which you want to remove, you are less likely to cut the patient if you work outwards from the centre of the ulcer towards its margin.

Dressings

Diabetic foot ulcers are chronic wounds and we believe

that they should be kept dry. The value of moist wound healing has only been demonstrated on acute wounds. Sterile, non-adherent dressings should cover all open diabetic foot lesions at all times except when they are inspected and debrided.

The rationale for keeping ulcers covered is to:

- Protect the wound from noxious stimuli
- Prevent infestation with insects
- Keep the wound warm
- Protect the wound from mechanical traumas
- Reduce the likelihood of infection.

There is no robust evidence from sufficiently large controlled studies that any one dressing is better for the diabetic foot than any other. However, the following properties are useful:

- Easy to lift
- Able to accommodate pressures of walking without disintegrating
- Absorbent.

Dressings should be lifted every day for wound inspection if possible, because when patients lack protective pain sensation the only signs of infection may be visual. For this reason, dressings which need to be left on a wound for several days to achieve their best effect may be inappropriate for diabetic feet.

In our foot clinic we frequently see the dire results of failure to detect wound deterioration early because the signs were masked by a dressing in a patient who lacked protective pain sensation. Where it is not possible to inspect the wound, any exposed adjoining areas of the foot and leg should be checked for:

- Colour change
- Swelling
- Change in temperature.

These can be signs either of uncontrolled infection or of worsening ischaemia which need urgent action.

Fever or hyperglycaemia should also be looked for.

Types of dressings used in diabetic foot patients and their relevant features

Films

- Clear, so wound inspection can be achieved without disturbing the wound
- Cannot absorb exudate which will collect under the film and form a blister and may irritate underlying tissue. Therefore contraindicated in exuding wounds
- Should never be used on infected or necrotic wounds. When used on dry necrosis will cause maceration and possibly promote infection
- Rarely appropriate for diabetic feet.

Foams

- Very absorbent
- Cushioning effect
- Bulky—may need specially roomy shoe to accommodate
- Widely used for diabetic feet.

Hydrocolloids

- Can be used only in patients with protective pain sensation
- Patient can bathe and shower
- Designed to be left on for several days. Daily changing prevents dressing from acting optimally.

Alginates

- Only to be used on moist exuding wounds
- Daily removal is time consuming
- Drying out of dressing may prevent wound drainage
- Calcium alginate is a good haemostatic agent
- Do not use on infected or necrotic wounds.

Hydrogels

- Promote autolysis and therefore promote debridement by apparently rehydrating the wound
- As we do not favour moist wound healing in the diabetic foot we see no strong indication for hydrogel therapy.

Hydrophilic fibre dressings

- Non-adherent, absorbent, fibrous dressings
- Soft and absorbent
- Conform to the wound.

Other dressings

Simple non-adherent dressings may be useful. Saline soaked gauze is widely used throughout the world.

Fastening dressings

Hypoallergenic tape and tubular bandages are useful. Conventional bandaging may cause excessive tightness. Only small amounts of tape should be applied to the skin. Encircling the entire toe with tape should be avoided in case it swells. It is important to issue precise requests to patients and nurses about techniques for holding dressings in place.

CASE STUDY

Improper use of a rubber band

A 25-year-old man with type 1 diabetes of 15 years' duration presented with a painful nail sulcus and underwent removal of a spike of nail. The toe was dressed with

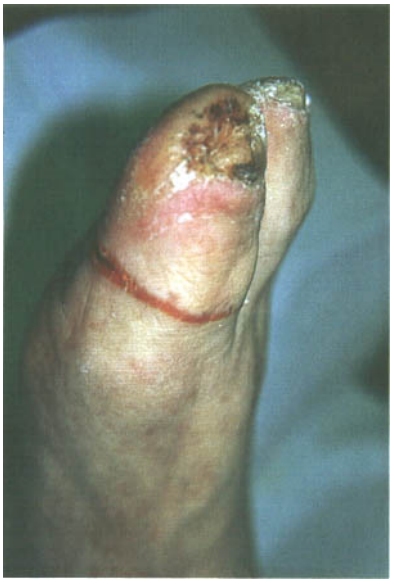


Fig. 4.12 A ring of superficial necrosis around the toe following use of a tight rubber band to hold a dressing on the toe.

Melolin and Tubegauz and he was advised to attend his practice nurse for dressings and return to the diabetic foot service in 2 weeks. At his next appointment we found a ring of superficial necrosis around the base of the toe (Fig. 4.12). On interrogation as to the cause of this, he blushingly admitted that he had not attended the nurse, but had applied dressings from his home first aid kit held in place by a rubber band around the toe.

Key points

- Patients should be given advice regarding application and fixing of dressings
- Tight bandages and ligatures encircling a digit should be avoided.

Advanced wound healing products

There may be intrinsic defects in ulcer healing in the diabetic patient. There is impairment of fibroblast function, deficiency in growth factors and abnormalities of the extracellular matrix. Initially it is important to try to achieve healing by standard techniques such as casting and debridement. However, an ulcer that does not heal is a risk for infection in the diabetic patient and advanced wound healing products should be considered. However, some of the products listed below are so expensive that the cost will be prohibitive for many practitioners.

Dermagraft

- Dermagraft is bioengineered living human dermis manufactured by seeding dermal fibroblasts onto a three-dimensional bioabsorbable scaffold
- A randomized controlled multicentre study demonstrated that 50.8% of the Dermagraft group experienced complete wound closure which was significantly greater than in the controls, of whom 31.7% had complete closure
- Must be stored at −80°C. Prior to application it is thawed, warmed and rinsed with sterile saline.

Apligraf

- Apligraf is a bioengineered bilayered skin substitute consisting of human fibroblasts embedded in type 1 bovine collagen and covered by human keratinocytes
- A randomized controlled study demonstrated a healing rate of 56% for those who were treated by application of Apligraf and standard wound care; this was significantly greater than 39% healing rate for patients who received only standard wound care
- It is delivered in a ready-to-use form on nutrient agarose in a sealed plastic bag in a disposable, battery-powered incubator.

Regranex

- This is platelet-derived growth factor which stimulates chemotaxis and mitogenesis of neutrophils, fibroblasts and monocytes
- A pivotal study in 382 patients demonstrated that Regranex gel 100 μg/g healed 50% of chronic diabetic ulcers which was significantly greater than 35% healed with placebo gel
- It presents as a tube of gel which is stored in a domestic refrigerator and applied every day.

Promogran

- Promogran consists of oxidized regenerated cellulose and collagen
- It inhibits proteases in the wound and protects endogenous growth factors
- In a 12-week study of 184 patients, 37% of Promogran-treated patients healed compared with 28% of saline gauze-treated patients but this did not reach a significant difference.

Hyaff

- Hyaff is a fibrous ester of hyaluronic acid which is a polysaccharide that is integral to the extracellular matrix and controls extracellular matrix hydration and osmoregulation

- When Hyaff is applied to the wound, hyaluronic acid is released
- Pilot studies in diabetic patients have shown promising results in the treatment of neuropathic foot ulcers, especially with sinuses.

Supplementary wound healing techniques

Several supplementary wound healing techniques have been used in diabetic foot ulcers including the following.

Skin grafts

- To speed healing of large, clean ulcers with a granulating wound bed, a split-skin graft may be harvested from the patient and applied to the ulcer
- If the donor site is chosen from an area within the distribution of the neuropathy, local anaesthetic infiltration of the donor site with a spinal needle will provide sufficient analgesia
- A general anaesthetic can thus be avoided and a donor site from within the distribution of the neuropathy will be less painful
- Careful follow-up of plantar skin grafts will be needed as they often develop callus and if this is allowed to accumulate it will result in neuropathic ulceration.

CASE STUDY

Skin graft

A 54-year-old woman with type 2 diabetes of 7 years' duration developed a plantar corn over her 1st metatarsal head, and applied a proprietary corn cure containing salicylic acid, purchased over the counter from her local pharmacy. She presented at casualty 1 week later with an infected acid burn causing a large tissue defect. She was given intravenous antibiotics and underwent surgical debridement. Two weeks later when the wound had a good bed of granulations and was ready to be closed, the surgeon infiltrated an area on the calf of her leg with local anaesthetic using a long spinal needle, and harvested a split thickness skin graft. This was applied to the wound and healed in 1 month (Fig. 4.13). The patient had no postoperative pain from the donor site which was within the distribution of her neuropathy. She had regular debridement of callus and special shoes to prevent the graft from breaking down.

Key points

- Corn cures can cause severe ulceration
- When repairing plantar defects, split thickness grafts harvested from within the area affected by neuropathy reduce postoperative donor site pain

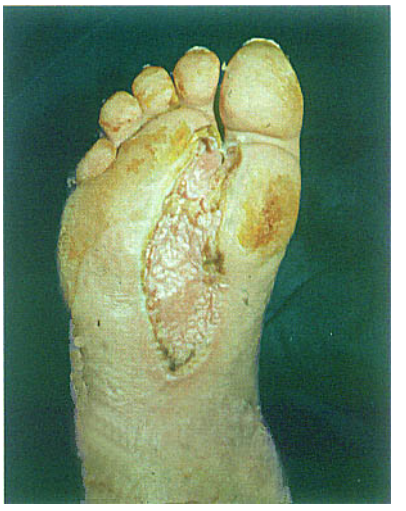


Fig. 4.13 Healed plantar skin graft with callus formation which needed regular debridement.

- Plantar skin grafts on weightbearing areas are prone to develop callus and patients should see a podiatrist.

Vacuum-assisted closure (VAC)

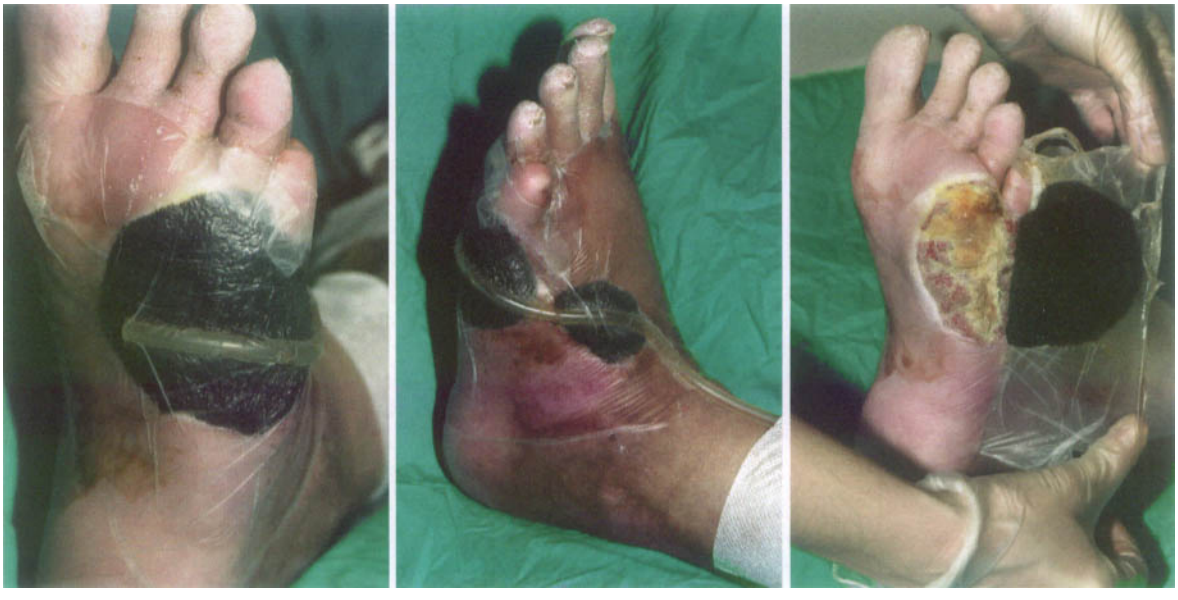
This is topical negative pressure therapy and can be used to achieve closure of diabetic foot wounds.

The pump applies subatmospheric pressure, through a tube and foam sponge applied to the ulcer over a dressing and sealed in place with a plastic film. The dressing is replaced every 2–3 days (Fig. 4.14a–e).

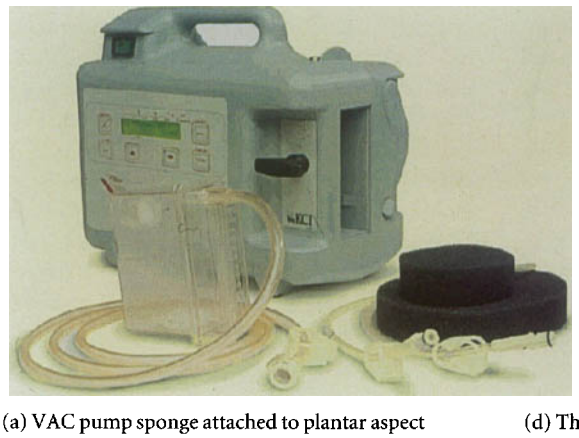
- Negative pressure improves the dermal blood supply, and stimulates granulation which can form over bone and tendon. It reduces bacterial colonization and diminishes oedema and interstitial fluid
- The course of treatment is usually a period of 7–10 days. The effect may wear off after 3 days but if the VAC pump is removed and then replaced after a further day this restores the effect. Excessive pain may prohibit the use of this technique even in diabetic patients with neuropathy.

Larva therapy (maggots)

- The larvae of the green bottle fly *Lucilia sericata* are used to debride ulcers, especially in the neuroischaemic foot
- This results in relatively rapid atraumatic physical removal of necrotic material
- Larvae also produce secretions that have antimicrobial activity against Gram-positive cocci including methicillin-resistant *Staphylococcus aureus* (MRSA)
- Randomized clinical trials are awaited.



(a) (b) (c)



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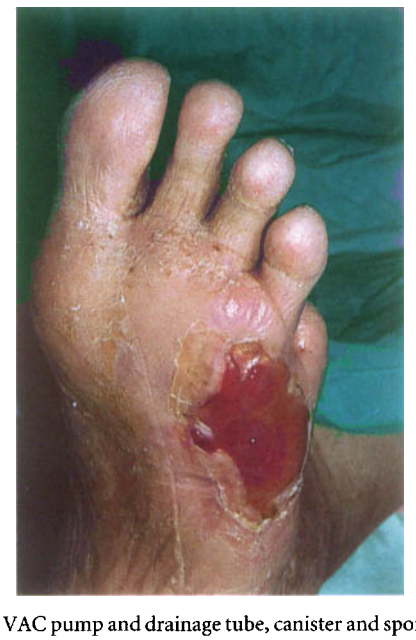


(e)

Fig. 4.14 (a) VAC pump sponge attached to plantar aspect of foot. (b) VAC pump sponge also attached to dorsolateral aspect of foot. (c) Pump sponge being removed from foot. (d) The VAC pump and drainage tube, canister and sponges. (e) Ulcer healing after 10 days VAC therapy.

Hyperbaric oxygen

- Recent studies involving relatively small groups of patients have shown that hyperbaric oxygen accelerates the healing of ischaemic diabetic foot ulcers
- Adjunctive systemic hyperbaric oxygen therapy also reduced the number of major amputations in ischaemic diabetic feet in a randomized study compared with controls

- It is reasonable to use hyperbaric oxygen as an adjunctive treatment in severe or life-threatening wounds which have not responded to other treatments.

Topical therapy

This consist of cleansing agents and antimicrobials.

Cleansing agents

- Saline: we use saline as a wound cleansing agent. It does not interfere with microbiological samples and is not damaging to granulating tissue
- Cetrimide-based cleansing agents are not recommended because of their cytotoxic action which impedes wound healing.

Antimicrobials

- Iodine is effective against a wide spectrum of organisms and comes in a variety of formulations including solutions, alcoholic tinctures, powder sprays and impregnated dressings. At high concentrations it can be toxic to human cells but bacteria are more sensitive to these effects than human cells such as fibroblasts, and thus it is believed that iodine may be useful for antisepsis without impairing wound healing. At present two types of iodine are available, povidone-iodine and cadexomer-iodine. Povidone-iodine is effective in antibacterial prophylaxis in burn patients but the evidence of its efficacy in other wound types is awaited. Cadexomer-iodine consists of microspheres formed from a three-dimensional lattice of cross-linked starch chains (cadexomers) and has been used with success in the diabetic foot ulcer
- Hypochlorite is most useful in sloughy wounds which are infected and therefore is more appropriate for ulcers in stage 4
- Silver compounds: silver sulfadiazine has been used in antibacterial prophylaxis in wounds and in skin graft donor sites. It is possible that silver may be useful as prophylaxis in diabetic foot ulcers. Recently, silver has been impregnated into dressings. *In vitro* it is effective in killing *Staphylococcus aureus*, including MRSA, and *Pseudomonas* species
- Mupirocin is active against Gram-positive infections including MRSA. To avoid the development of resistance, mupirocin should not be used for longer than 10 days and should not be regarded as a prophylactic.

Microbiological control

Now that the skin is broken, the stage 3 patient is at great risk of infection because there is now a clear portal of entry for invading bacteria to enter the foot. Additionally, in the presence of neuropathy and ischaemia, the inflammatory response is impaired. The patient lacks protective pain sensation which would otherwise automatically force him to detect the problem and rest the foot.

Bacterial growth in ulcers impedes the wound healing rate. Quantitative microbiology has shown that with increased bacterial load, wound healing slows. There is a complex host–bacteria relationship. Many wounds are colonized with a stable bacterial population. If the bacterial burden increases there will be bacterial imbalance which may show itself as increased exudate before frank infection develops. The crucial problem is when to intervene with antibiotics.

Uniform agreed practice on the role of antibiotics in the clinically uninfected stage 3 diabetic foot, where no signs or symptoms of infection are present, has not been established. Over many years of experience managing the diabetic foot, we have established the following plan for neuropathic and neuroischaemic ulcers, which has achieved significant reductions in amputations.

Our approach is also based on the results of a study of 64 diabetic patients with clean foot ulcers who were randomized into two groups: 32 patients receiving oral antibiotics and 32 patients who did not. In the non-antibiotic group 15 patients developed clinical infection compared with none in the antibiotic group, which was a highly significant difference. In the 32 patients who did not receive antibiotics there were 12 with neuroischaemic ulcers and 20 with neuropathic ulcers: 8/12 neuroischaemic ulcers developed infection which was significantly greater than 7/20 neuropathic ulcers. This indicated that neuroischaemic ulcers were more likely to develop clinical infection. Furthermore, 11 of the 15 patients who developed clinical infection had positive ulcer swabs taken at their last clinic visit compared with only one positive swab taken at the prior visit in the 17 patients who did not develop clinical infection. This indicated that properly taken ulcer swabs can be a useful indicator of impending infection.

From this study we concluded that patients with diabetes and clean ulcers associated with peripheral vascular disease and positive ulcer swabs should be considered for early antibiotic treatment. We therefore pay close attention to the results of properly taken ulcer swabs, taken at the first clinic visit and at subsequent visits. Some authors question the value of performing swab cultures in the absence of signs or symptoms of infection, or of ever prescribing antibiotics based on positive wound cultures alone in the absence of signs or symptoms of infection. In non-diabetic patients this approach is probably reasonable.

However, we feel that in dealing with high-risk diabetic feet, failure to take swabs deprives the clinician of advance warning that infection may be imminent. Infection is alarmingly common in high-risk diabetic feet, and signs and symptoms of infection are frequently absent or greatly reduced. We believe that properly taken swabs are useful.

All swabs taken from diabetic foot ulcers should be deep swabs taken after debridement has been carried out. We accept that currettings and tissue from the base of the ulcer may be a more acceptable microbiological sample compared with the deep ulcer swab and we do send such samples instead of a ulcer swab where possible. However, in many cases, particularly in the neuroischaemic foot, it is not possible to safely and painlessly obtain such samples and the deep wound swab is then the only alternative.

Neuropathic ulcers

Our approach to neuropathic ulcers is as follows.

At the first visit, if there is no cellulitis, discharge or probing to bone, the foot is deemed to be at stage 3. Debridement, cleansing with saline, application of dressings and daily inspection will suffice. The patient is reviewed at, preferably, 1 week or less, together with the result of the deep ulcer swab or tissue culture. If the neuropathic ulcer shows no sign of infection and the swab is negative, treatment is continued without antibiotics. If the ulcer has a positive swab, the patient is treated with the appropriate antibiotic, according to antibiotic sensitivities until the repeat swab, taken at weekly intervals, is negative. It is accepted that some organisms isolated from the swab may be commensals; however, if there are Gram-positive organisms or anaerobes or a pure growth of Gram-negative organisms we regard this as a significant result and microbiological evidence of infection. It was shown in a study of the bacterial population of chronic crural ulcers that a swab should be obtained routinely from patients with diabetic ulcers as 70% of diabetic ulcers with a positive swab developed clinical infection.

Neuroischaemic ulcers

We prescribe antibiotics more readily for the neuroischaemic foot because untreated infections in neuroischaemic feet lead rapidly to extensive necrosis, destruction of the foot and major amputation.

- At the first visit, if the ulcer is superficial, we prescribe oral amoxicillin 500 mg tds and flucloxacillin 500 mg qds. (If the patient is penicillin allergic, we prescribe erythromycin 500 mg qds or cefadroxil 1 g bd.) If the ulcer is deep, extending to subcutaneous tissues, we add trimethoprim 200 mg bd or ciprofloxacin 500 mg bd, and metronidazole 400 mg tds to cover Gram-negative and anaerobic organisms that may be present in the deep tissues. If, on review, the neuroischaemic ulcer shows no signs of infection and the swab is negative, antibiotics may be stopped. However, in cases of severe ischaemia (pressure index < 0.5) we may continue antibiotics until the ulcer is healed. If the neuroischaemic ulcer has a positive swab, the patient is treated with the appropriate antibiotic, according to antibiotic sensitivities until the repeat swab, taken at weekly intervals, is negative
- At every patient visit, examination for local signs of infection, cellulitis or osteomyelitis is performed. If these are found, action, including antibiotic therapy, is taken as described in Chapter 5.

Vascular control

If ulcers in the neuroischaemic foot fail to heal despite optimal treatment, the reason may be ischaemia. Atherosclerotic lesions commonly occur in the tibial arteries but also occur in the popliteal and femoral arteries, with the iliacs rarely involved. A careful vascular assessment is necessary, to determine the degree of ischaemia and to decide when to perform invasive investigations with a view to revascularization.

Initially the ankle brachial pressure index should be measured, supplemented by assessment of the Doppler waveform. Further tests such as measurement of transcutaneous oxygen tension and toe pressure may be helpful in deciding whether to pursue invasive arterial investigations with a view to vascular intervention.

Pressure index

The pressure index is widely criticized because, when the arteries are calcified, it may be artificially raised. However, we feel that it is very relevant to the investigation of the diabetic foot as long as the potential difficulties of its interpretation are understood.

If the pressure index is 0.5 then it is truly low, and indicates severe ischaemia whether the arteries are calcified or not. Indeed, if the artery is calcified the true pressure index may be even lower and even more urgent action is required.

Difficulty comes at pressure indices of 0.5 and above, when one should always pay attention to the Doppler waveform, either in audible or visible form. The normal waveform is pulsatile with a positive forward flow in systole followed by a short reverse flow and a further forward flow in diastole, but in the presence of arterial narrowing the waveform shows a reduced forward flow and is described as 'damped' (Fig. 4.15a,b).

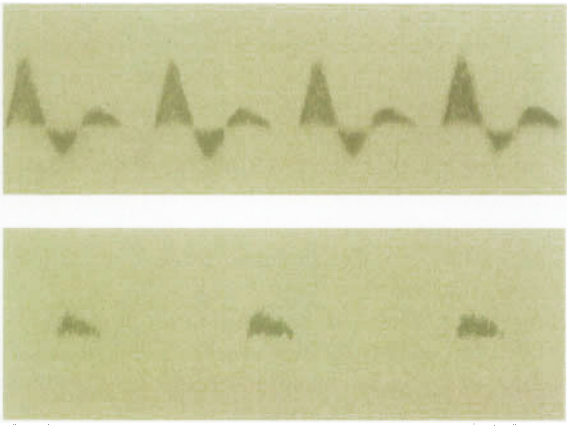


Fig. 4.15 (a) Doppler waveform from normal foot showing normal triphasic pattern. (b) Doppler waveform from neuroischaemic foot showing damped pattern.

Transcutaneous oxygen tension ($TcPo_2$)

This measurement is a non-invasive method for monitoring arterial oxygen tension and reflects local arterial perfusion pressure. A heated oxygen sensitive probe is placed on the dorsum of the foot. Normal $TcPo_2$ is greater than 40 mmHg. A level below 30 mmHg indicates severe ischaemia and indicates the need for further vascular assessment such as non-invasive angiography. However, levels can be falsely lowered by oedema and cellulitis.

Values in the foot can also be compared with those on the chest wall to derive a regional perfusion index, which is independent of fluctuations in systemic oxygen delivery, although commonly, the absolute pressure on the dorsum of the foot is used.

Toe pressures

Toe pressures can be measured using a special cuff and photoplethysmography. We regard a toe pressure of 30 mmHg or below as an indication of severe ischaemia and, in the presence of ulceration, requiring further investigations.

Angiography (Fig. 4.16)

If an ulcer has not progressed despite optimal treatment and ankle brachial pressure index is less than 0.5 or the Doppler waveform is damped and either $TcPo_2$ is less than 30 mmHg or toe pressure is less than 30 mmHg, then angiography should ideally be carried out. It is difficult to state when intervention should be carried out as this varies from patient to patient. However, the recommendations of the American Diabetes Association state that

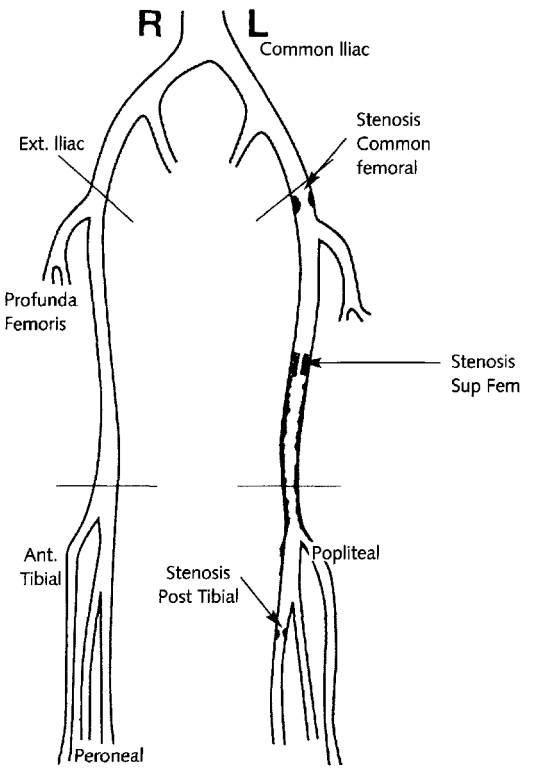


Fig. 4.16 Print-out of Duplex examination showing stenoses in the left common femoral artery, superficial femoral artery and posterior tibial artery.

further investigations should be started after 4 weeks with standard treatment.

Non-invasive procedures should be used first to examine the peripheral circulation and to detect areas of stenosis and occlusion. Transfemoral angiography with angioplasty can then be used as a definitive localized invasive procedure.

Duplex angiography

Angiography can be carried out by an examination which combines the features of Doppler waveform analysis with ultrasound imaging to produce a picture of arterial flow dynamics and morphology.

Magnetic resonance angiography (MRA)

This technique uses magnetic resonance imaging to delineate arterial flow and disease.

There are two techniques of MRA:

- Time of flight MRA is dependent on a non-contrast enhanced flow-sensitive magnetic resonance sequence.

Fig. 4.17 Magnetic resonance angiography (MRA) showing multiple areas of stenosis and occlusion through the right and left superficial femoral arteries and in the left popliteal artery. Courtesy of Dr Paul Sidhu.

It is slow, lasting up to 2 h, to cover the area from the bifurcation of the aorta to the distal lower extremity

- Gadolinium-enhanced MRA involves an intravenous injection of gadolinium contrast and a fast imaging that follows the passage of the contrast bolus through the arteries. It is much less nephrotoxic than conventional contrast (Fig. 4.17).

The advantages of MRA against conventional transfemoral angiography are that there is no need for an intra-arterial catheter and nephrotoxic contrast can be avoided.

Having carried out these non-invasive procedures to map out the arterial system, it is now possible to focus treatment by performing transfemoral angiography, together with digital subtraction angiography, followed by angioplasty or arterial bypass.

Angioplasty

Angioplasty is tolerated well and can be carried out in very elderly patients. In suitable patients it is now possible to carry out transfemoral angiography and angioplasty as an outpatient procedure. A small-bore needle is used, and after the procedure the puncture site is closed by a special 'Perclose' technique which is a suture-mediated closure of the arterial access site. This allows the patient to sit up after 2 h and to walk in 4 h.

Contraindications for day-case angiography include:

- Myocardial infarction/cerebrovascular event within last 6 months
- Severe cardiac/respiratory disease
- Major surgery within last month
- Warfarin therapy
- Renal failure.

Preparations for transfemoral angiography and angioplasty

Patients taking metformin should stop this 2 days before the procedure and restart 2 days after, or when, renal function returns to normal.

Insulin-dependent patients are placed first on the list in outpatient angiography and have their insulin after the procedure is finished.

It is important to keep the patient well hydrated.

Pre- and perioperative dopamine is no longer used.

Further details of MRA are discussed in Chapters 5 and 6.

Angioplasty is possible at several levels of the leg arterial system to obtain straight line flow to the foot. It is indicated for the treatment of isolated or multiple stenoses as well as short segment occlusions less than 10 cm in length in iliac, femoral and tibial arteries.

The use of angioplasty in this way has led to favourable outcomes. Complications are few and trash foot, caused by emboli to the distal circulation, is very rare after distal angioplasty.

CASE STUDY

Failed healing solved by angioplasty

A 69-year-old woman with type 2 diabetes for 12 years and end-stage renal failure treated by haemodialysis was referred to the foot clinic with a cold red ischaemic left foot (Fig. 4.18). She was referred to the vascular laboratory: they were unable to measure her brachial pressure due to fistulas in both arms. However her tibial vessels were monophasic pulsatile with high diastolic flow. She did not want further vascular investigations at this point. However, she returned to the foot clinic 2 weeks later with a sloughy ulcer deep to bone on the apex of her left hallux, a deep subungual ulcer on her left 3rd toe and a sloughy ulcer on her left heel. TcPo_2 tension of the right foot was 20 mmHg. She underwent duplex angiography followed by transfemoral angiography and angioplasty of an occlusion of the superficial femoral artery and stenosis of the anterior tibial artery. This achieved straight line arterial flow to her foot and her ulcers healed in 10 months.

Key points

- Angioplasty is the first-line treatment for peripheral

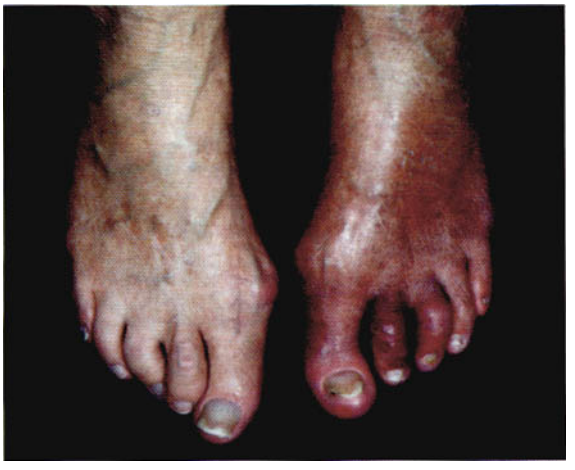


Fig. 4.18 The left foot is cold, red and ischaemic at presentation.

arterial disease in the diabetic limb, where the intention is to obtain straight line arterial flow to the foot
- Measuring the pressure index may be impossible in patients on haemodialysis with fistulas
- $TcPo_2$ is a useful alternative method of quantitating ischaemia in these circumstances.

CASE STUDY
Angioplasty in the elderly

A 90-year-old woman with type 2 diabetes of 20 years' duration developed a small painful ulcer on the lateral border of her right heel (Fig. 4.19). The foot was pulseless with a pressure index of 0.5. Pain kept her awake at night. She underwent angioplasty of a short stenosis of the superficial femoral artery, the pain improved and the foot healed in 9 weeks.

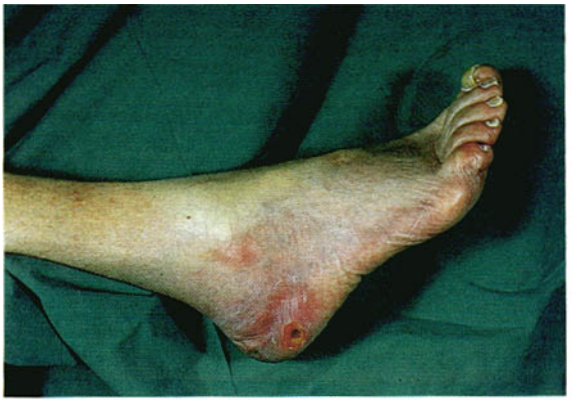


Fig. 4.19 This small painful ischaemic ulcer on the heel of a 90-year-old woman healed in 9 weeks following angioplasty.

Key points
- Advanced age is not a contraindication for angioplasty
- Patients with rest pain should be referred for vascular assessment without delay.

Arterial bypass
If lesions are too widespread for angioplasty, then arterial bypass may be considered. However, this is a major, sometimes lengthy, operation, and is not without risk. It is more often reserved to treat severe tissue destruction which cannot be managed without the restoration of pulsatile blood flow to the foot (see Chapter 6). However, in some patients, angioplasty may not be technically feasible and then arterial bypass should be considered if the ulcer does not respond to consevative treatment.

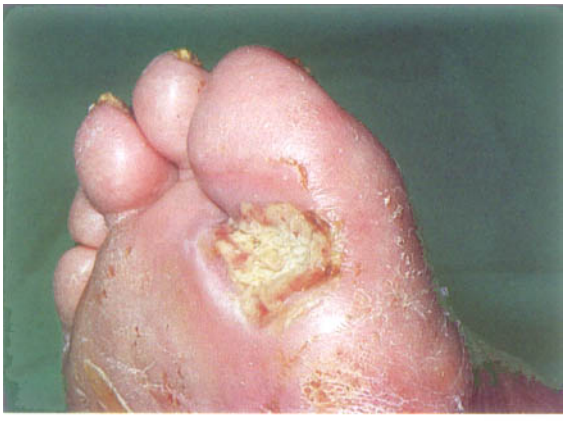
CASE STUDY
Distal bypass for ischaemic ulcer

A 62-year-old lady with type 1 diabetes of 12 years' duration wore tight shoes and developed an ulcer on the plantar surface of her forefoot after a nail penetrated her shoe. Despite regular podiatry, special shoes and antibiotics the ulcer failed to heal for 7 weeks and became larger (Fig. 4.20a). Her feet were pulseless and her pressure index was 0.4. She did not suffer from intermittent claudication or rest pain. Angiography showed a 10-cm occlusion of the superficial femoral artery and advanced disease of the proximal tibial arteries with reforming of the anterior tibial artery in the lower leg. She underwent femoral/anterior tibial bypass. The ulcer healed in 14 weeks (Fig. 4.20b).

Key points
- Patients with ischaemic ulceration which fails to heal or deteriorates should undergo angiography with a view to angioplasty or arterial bypass
- When angioplasty is not technically possible, arterial bypass should be considered if there is a suitable run-off vessel in the lower leg
- Intermittent claudication and rest pain are often absent in the severely ischaemic diabetic limb.

Pain control
In general, the neuroischaemic foot is not painful: however, in a few cases when neuropathy is mild the patient suffers pain from the ulcer as well as rest pain in the

(a)



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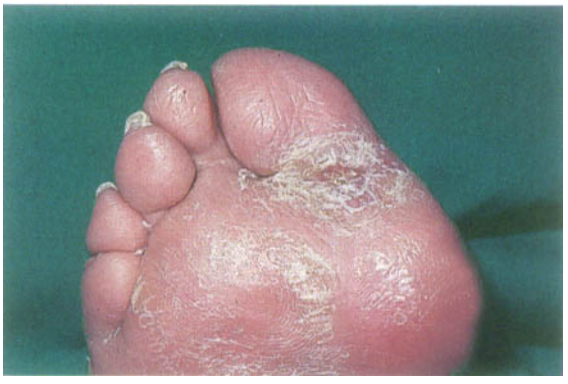


Fig. 4.20 (a) Large ischaemic ulcer, previously a penetrating injury, which failed to heal despite conservative care. (b) The same ulcer has healed in 14 weeks following femoral/anterior tibial bypass.

foot, particularly at night. It is important to control this pain.

- An opioid such as dihydrocodeine, alone (30 mg every 4–6 h) or in combination with a non-opioid analgesic, e.g. co-dydramol (dihydrocodeine 10 mg, paracetamol 500 mg, two tablets every 4–6 h) may be useful in moderate pain
- Tramadol (50–100 mg 4–6 hourly) is an opioid derivative, which is often less sedating and less constipating than codeine
- Tricyclic antidepressants, for example dothiepin 50–100 mg at night, are useful at relieving rest pain in bed
- When pain is severe, it is important to give regular morphine therapy. Initially it is best to start with a short-acting preparation taken 4-hourly, and the patient can quickly titrate the dose necessary to relieve pain. After this, it is possible to advance to the modified slow-release preparations devised for twice-daily administration such as 10–20 mg every 12 h, if no other analgesic or paracetamol has been previously prescribed. However, if it is replacing a weaker opioid analgesic, for example co-dydramol, the initial dose should be 20–30 mg every 12 h. The doses should be gradually increased but the frequency kept at every 12 h. If breakthrough pain occurs between the 12-hourly doses, then morphine, as oral solution (Oramorph 5–20 mg every 4 h) or standard formulation tablets such as Sevredol 10–50 mg every 4 h, can be given
- Beware of respiratory depression, to which diabetic patients with autonomic neuropathy can be susceptible. One of our patients with severe autonomic neuropathy underwent marked respiratory depression when she took dihydrocodeine phosphate and almost stopped breathing
- Chemical sympathectomy by paravertebral injection of phenol is also used to relieve rest pain, although it does not increase peripheral blood flow.

Opiates will be retained by patients in renal failure, often leading to drowsiness, and the dose will need to be suitably reduced.

Metabolic control

Systemic, metabolic or nutritional disturbances retard healing. It is important to control blood glucose, blood pressure and lipids, and ask the patient to stop smoking. Patients with neuroischaemic ulcers should be on statin therapy as well as antiplatelet therapy. Diabetic patients who are above 55 years and have peripheral vascular disease should also benefit from an angiotensin-converting enzyme (ACE) inhibitor to prevent further vascular episodes.

The influence of blood glucose on wound healing is controversial. Diabetes is related to faulty wound healing, but this may be because of associated factors such as infection and macrovascular disease. Nevertheless, it is important to achieve good blood glucose control. In the patient with type 2 diabetes, oral hypoglycaemic therapy will need to be optimized, but if this is not successful then insulin should be started.

When managing hypertension in the presence of leg ischaemia it is important to achieve a fine balance between maintaining a pressure that improves perfusion of the ischaemic limb while reducing the blood pressure enough to limit the risk of cardiovascular complications. However, β-blockers are not contraindicated in the neuroischaemic foot.

Many patients will have evidence of cardiac failure. Aggressive treatment will improve tissue perfusion and also reduce swelling of the feet.

Renal impairment may also be present, and optimum treatment is essential to avoid lower limb swelling.

Educational control

The care of ulcers in diabetic patients is fraught with problems. It is easy for health-care professionals to issue instructions to rest, take time off work, wear special shoes or casts and follow advice to the letter. In practice, many patients are unwilling or unable to follow advice.

People who live entirely on their own need to stand and walk to obtain the basic necessities of life.

Where the patient lives on his own, has neuropathy and sometimes poor vision as well, it can be very difficult to detect deterioration early, or to do dressings properly.

Patients who lack protective pain sensation need to know that foot ulcers are a serious problem but that they will heal with optimal care. The following educational material—aimed directly at patients—seeks to clarify common misconceptions about ulcers and their management.

Education for patients with foot ulcers

Are foot ulcers a serious health problem?

Diabetic foot ulcers are a very serious health problem. It is essential to treat them quickly. They may not hurt, which makes them even more serious because you may be tempted to stick a plaster on and forget about them.

How are ulcers caused?

Any injury to your feet, no matter how tiny, may lead to an ulcer. If you have neuropathy it is easy to injure your foot without noticing. If you have a poor blood supply to your feet then ulcers can develop for no obvious reason. It is often nobody's fault that you have an ulcer: not yours and not the fault of the last person who treated the foot before you had the ulcer. People with ulcers sometimes feel angry and afraid and look for someone to blame. It is better to avoid these negative feelings.

Many ulcers develop when patches of hard skin or callus on the foot become too thick, or when skin breaks down under a thickened toe nail. Often it is only when the hard skin is removed that the ulcer can be seen. The ulcer is not the fault of the person who removed the hard skin. It was there before. It is nobody's fault.

Other ulcers are due to carelessness. If people with numb feet and a poor blood supply walk barefoot, wear unsuitable shoes or fail to follow foot care advice then they will be very likely to develop ulcers.

How will I know if I have an ulcer?

You should check your feet every day for ulcers. You may feel no pain to warn you that you have a problem. You may see a discoloured area, a break in the skin or a sore place. You may see blood or pus, or notice that your sock is wet from discharge. If you cannot see your feet clearly you should ask for help checking them. A mirror is helpful for seeing the soles of your feet.

How can a painless ulcer be a big problem?

Any injury to the foot is difficult to heal unless you rest it. If the wound is not painful then you will be tempted to carry on with life as normal. If you do this there is a great risk that the ulcer will fail to heal and will gradually get bigger. The longer an ulcer has been present the harder it will be to heal it, so it is essential to catch ulcers early and treat them quickly and effectively.

Why are foot ulcers dangerous?

Germs can enter your foot through any break in the skin such as an ulcer, a split, a graze, a blister or a sore place and cause infection. If you have neuropathy (nerve damage) you will not feel the infection as it takes hold. If the blood supply to your foot is poor you will be unable to fight the infection. Untreated infection can destroy a diabetic foot.

What must I do to heal my foot quickly?

- You should ask for help (the same day) from your doctor, hospital or diabetic foot service
- Clean the ulcer with saline
- Cover the ulcer with a sterile dressing held in place with a light bandage
- Ask for help dressing the ulcer from your community nurse if you or your family have difficulty with this
- Keep right off the ulcer. Use crutches or a wheelchair and try not to put your foot to the ground
- Keep your foot up on a stool and cushion, sofa or bed
- Stay home from work or work from home if possible
- Do not go away on holiday with an open ulcer
- If you are offered special shoes or plaster cast treatment, remember that these heal ulcers very successfully.

Remember that a few days off work can heal an ulcer and prevent months of misery and long stays at home or in hospital.

If ulcers are healed quickly there will be less scarring and you will be far less likely to develop another ulcer in the future.

What other treatment will my ulcer need?

Ulcers are often surrounded or covered with dead tissue, callus and debris. If these are allowed to gather around the ulcer they will prevent it from healing and may make the ulcer get worse. The ulcer should be cleaned by cutting away all the unwanted tissue. It may be necessary to clean up the edges until they bleed. This sounds drastic and frightening but it is usually quite painless and is essential treatment if you want your ulcer to heal quickly. This treatment is called 'debridement'. Never try to debride your ulcer yourself: ask your podiatrist or doctor for help.

Another aspect of ulcer treatment is preventing pressure on the ulcer. This can be achieved by resting and elevating the foot, by using crutches or a wheelchair, or by wearing special footwear or casts as advised by the foot clinic.

How long do ulcers take to heal?

In a patient who attends a well-organized, multidisciplinary diabetic foot clinic and follows advice, diabetic foot ulcers should heal within 3 months.

What should I do if my ulcer fails to heal?

- Tell the foot clinic you are concerned and ask them why they think the ulcer is not healing
- Ask them if a second opinion might help
- Ensure you are being treated by an experienced multidisciplinary team
- Follow their advice explicitly
- If any aspects of their advice are impossible for you to follow, tell them and ask for advice. There may be an alternative approach which will suit you better.

What short-term lifestyle changes would help?

- Ask your family or friends for help. People like to rally round in times of trouble, and you can repay them later. If you live alone you can ask your doctor or nurse to organize help
- Let your usual high standards slip a bit: do less
- Prioritize! Leave the housework and let the dust gather or get help with cleaning: it'll all be the same in a hundred years!
- Don't run around helping other people: they should be looking after you
- Don't stand at the stove: use quick and easy recipes or use ready-cooked or take-away food. Cooking can be hard on the feet. Make the family do the washing up or use disposable plates
- Fight the boredom factor: think about things you can do while you are sitting or lying down and ways of getting out and about without overloading your foot. Get your friends to come to you instead of going to them—and ask them to bring the refreshments with them! Get a wheelchair so that you can still go out without putting pressure on your foot ulcer
- Don't feel guilty. You are keeping off your feet in order to avoid future problems. Be sure that everyone knows that. If anyone criticizes you for being lazy ask him to talk to the diabetic foot service
- Keep your foot clinic appointments without fail. If you miss one, ask for another without delay.

What should I do if my foot ulcers keep coming back?

If you get recurrent ulcers, the following long-term lifestyle changes would help:

- Try to avoid jobs which involve long hours of walking or standing
- Work as close to home as possible: long journeys are bad for feet
- Get your car adapted to hand controls instead of foot pedals. Use an automatic.

Should I drive with an ulcer?

Driving with an ulcer is better than walking, but not so good as resting.

What should I do if it is essential to drive?

If you need to drive you should:

- Stop frequently to rest and elevate the feet
- Avoid emergency stops!
- Consider having hand controls fitted to your car
- Enquire about a different design of foot pedal which will not pressurize the ulcerated area
- Always wear your hospital shoes for driving. (If shoes are large and bulky, the control pedals of the car may need to be adjusted or changed.)

What else should I be doing to help my foot heal and stay healed?

- If you enjoy sports, try to choose something like swimming which off-loads your feet
- Avoid situations where you are out of control. For example, if you go on holiday after your ulcer has healed then allow plenty of time at the airport, and organize a wheelchair if you have a history of recurring ulceration. If you go on a guided tour, ensure it will not involve long walks over rough ground
- Walk as little as possible
- Try to lose weight if you are overweight
- Be on the lookout for potential problems that have caused ulcers in the past or might cause a future problem.

Radiators or hot pipes by the bed can burn your feet, worn-out shoes can rub sores and walking barefoot can injure you. Try to remember the hidden dangers in day-to-day life and think hard about how to avoid them
- Every time you injure yourself and cause an ulcer, try to work out the reason and take steps to prevent it in future. It sounds boring, but it isn't nearly as tedious as having foot ulcers
- Be prepared to drop everything and come to the foot clinic at the first sign of a foot problem.

Can I go on holiday with an ulcer?

Ideally you should not go on holiday until
- The ulcer has healed and remained healed for at least a month
- Your footwear is adjusted
- Your footwear has been worn in and is trouble free
- You have received holiday foot care advice
- You know what to do and where to go if you get a foot problem on holiday.

What should I do if my own doctor or nurse wants to change the treatment?

Always ask them to discuss your treatment with the diabetic foot service.

Specific categories of ulcer

The rest of this chapter discusses the presentation and management of other categories of ulcer.

Bullae

Bullae are superficial fluid-filled sacs which develop when the skin is traumatized (Fig. 4.21).

In acute vesicular tinea pedis, vesicles may become confluent and form large bullae.

Large bullae may form when there is massive lower limb oedema, often secondary to cardiac or renal failure (Fig. 4.22).

In the neuropathic foot the first presentation of an ulcer is frequently as a blister under callus which should be drained and debrided (Fig. 4.23a–c).

Ischaemic ulcers on the margin of the foot can also begin with a bulla (Fig. 4.24).

When assessing a bulla the following should be ascertained:
- Is it tense or flaccid?
- What colour is it?
- What does it contain?
- What was the probable cause?

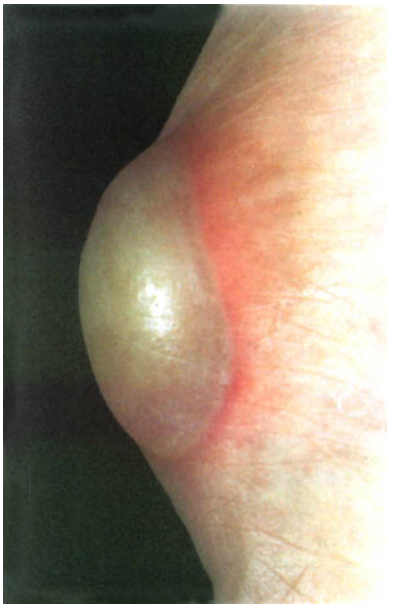


Fig. 4.21 This large bulla developed on the medial border of the 1st metatarsophalangeal joint associated with a hallux valgus deformity which was not accommodated in sufficiently wide shoes.

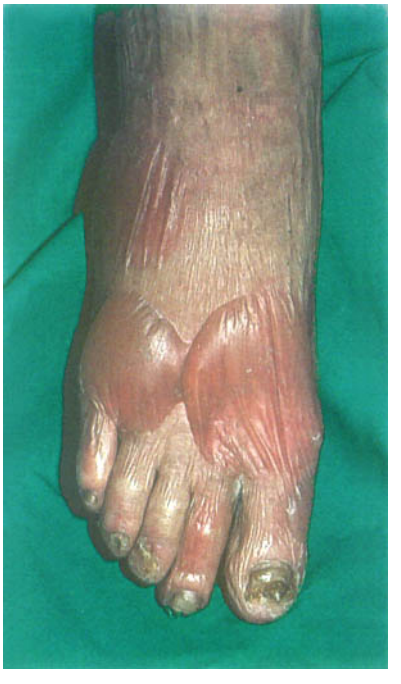
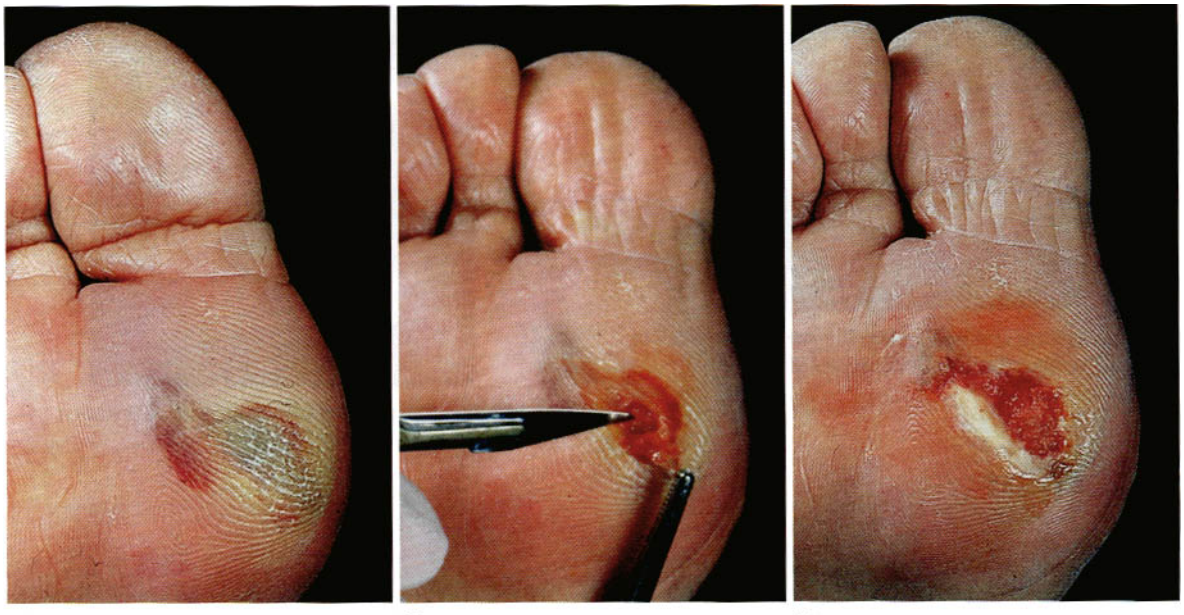


Fig. 4.22 Bulla secondary to severe cardiac failure.



(a) (b) (c)

Fig. 4.23 (a) A blister has formed under callus over the first metatarsal head. (b) The roof of the blister is grasped in forceps and cut away with a scalpel together with associated callus. (c) After debridement the base of the neuropathic ulcer is revealed.

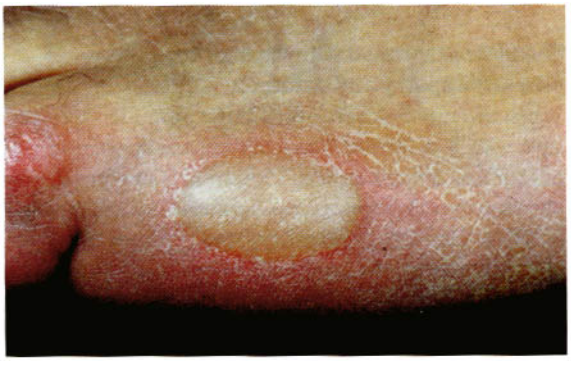


Fig. 4.24 The first presentation of this lesion was as a marginated bulla over the lateral border of the foot but it soon developed into an ischaemic ulcer.

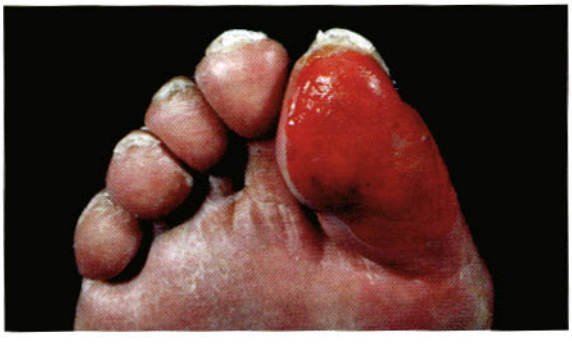


Fig. 4.25 A bulla has been drained and de-roofed for inspection of its floor.

There are two schools of thought on the wisdom of de-roofing blisters. Our practice is to drain tense bullae and all bullae more than 1 cm in diameter (Fig. 4.25).

Decubitus ulcers

Decubitus ulcers, which develop when the foot is exposed to unrelieved pressure, are common on the diabetic foot and especially on the heel. Patients who have been ill or immobilized are particularly vulnerable. Decubitus ulcers can develop in a short time.

Contributing causes include:

- Rough sheets
- Moisture
- Foreign bodies in the bed (biscuit crumbs, etc.)
- Patients attempting to move in the bed by putting excessive pressure on the heels
- Sliding down the bed so that feet are in contact with bed end.

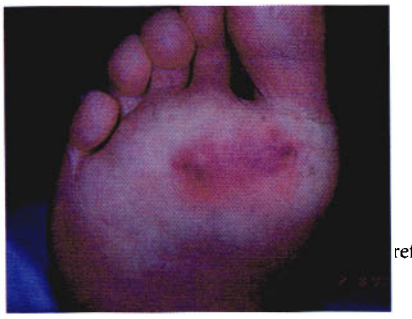


Fig. 4.26 A
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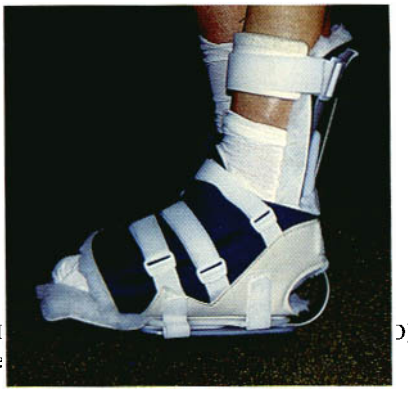


Fig. 4.27 T
relieves pre
D)

CASE STUDY
Decubitus ulcer

A 93-year-old lady with type 2 diabetes of 10 years' duration, was admitted to hospital following a stroke. On admission her feet were intact. She slid down the bed and was found with her foot jammed against the footboard. A purple area developed where the foot had been in contact with the footboard (Fig. 4.26). The footboard was removed and the purple area did not deteriorate further.

Key points

- Elderly immobile frail patients are very vulnerable
- Remove footboards from the bed of frail, immobile patients who slide down the bed.

Management of heel ulcer

The first sign of a heel ulcer is localized erythema. If pressure is not relieved a 'blister' will develop, which fills first with clear fluid and subsequently with serosanguineous fluid. The base of the blister becomes blue and then black. If pressure is not relieved then deep necrosis may develop. Tense heel blisters should be opened and drained using aseptic precautions.

Urgent pressure relief should be organized. This can be achieved by:

- Regular turning and repositioning of immobile patients to relieve continuous local ischaemia over pressure points
- The pressure relief ankle–foot orthosis (PRAFO) has a washable fleeced liner with an aluminium and polypropylene adjustable frame and a non-slip walking Neoprene base. The patient can wear this orthosis both lying down and walking to avoid pressure on the back of the heel (Fig. 4.27)
- Foam wedges are traditionally used to protect the heels. Heel-protector rings and special heel-relieving splints are available which suspend the heel to protect against further breakdown and allow the ulcer to drain.

Heel ulcers on the neuropathic foot can be healed with a total-contact cast (Fig. 4.28a,b).

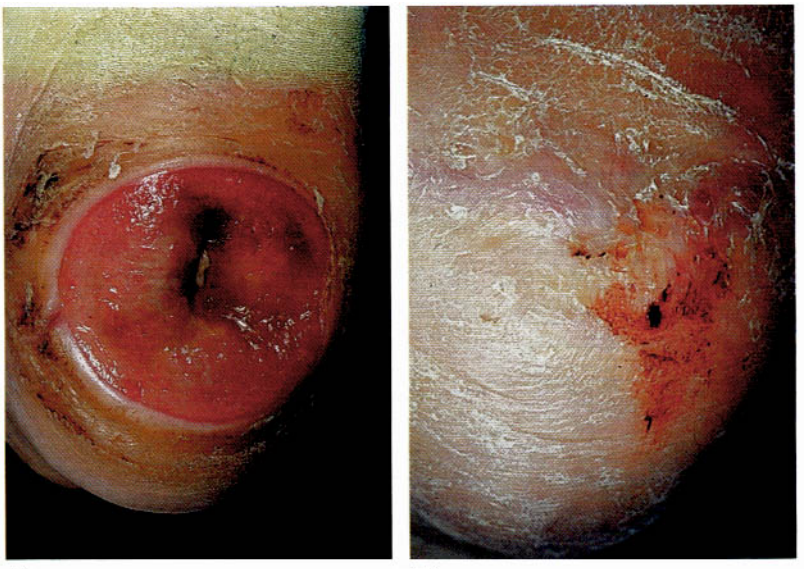
The ulcer associated with Charcot's osteoarthropathy and deformity

When ulceration occurs over a bony prominence associated with a deformed Charcot's osteoarthropathy, it is notoriously difficult to heal. Ulceration is a frequent complication of rockerbottom, medial convexity and unstable hindfoot Charcot's osteoarthropathy.

Patients with bony prominences on deformed feet with Charcot's osteoarthropathy need regular removal of callus to prevent ulceration. We use the total-contact cast to treat most of these ulcers (Fig. 4.29a,b).

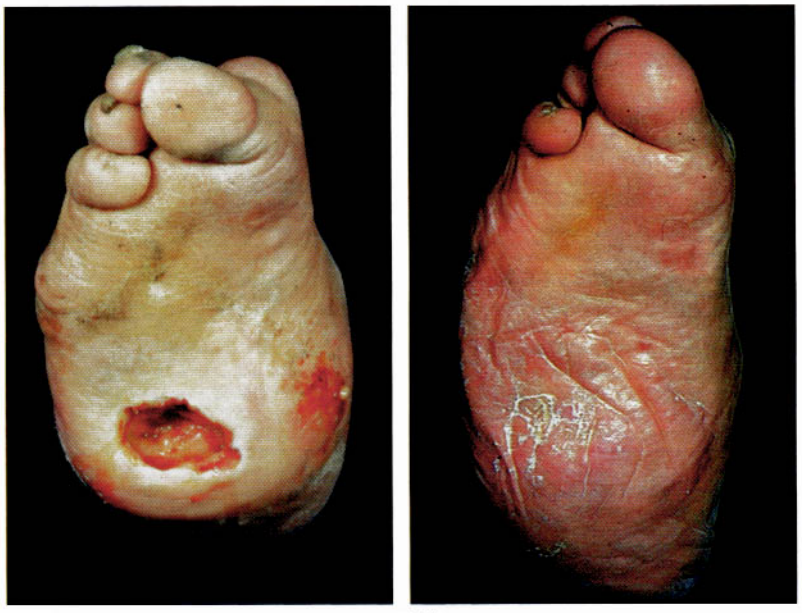
In addition, we have used advanced wound healing products including Dermagraft, Apligraf and Hyaff to promote healing of ulceration over tarsal deformities (Fig. 4.30a,b).

However, conservative measures may fail. If management in a cast does not achieve healing within 3 months, surgery should be considered. Exostectomy is indicated for the mid-foot Charcot's osteoarthropathy and internal stabilization of the ankle for the hindfoot ulcer which is not healing. The patient shown in Fig. 4.31a,b developed indolent ulceration over an unstable ankle with Charcot's osteoarthropathy of the hindfoot, but healed after he underwent internal fixation.



(a) (b)

Fig. 4.28 (a) This large heel ulcer had been present for 3 years. (b) The same ulcer healed in 9 weeks after a total-contact cast was applied.



(a) (b)

Fig. 4.29 (a) This foot with Charcot's osteoarthropathy and rockerbottom deformity developed an indolent plantar ulcer. When this picture was taken the ulcer had been present for 6 years. (b) The same foot healed in 12 months. The patient had remained at work throughout the casting period and was very active, working as an architect and inspecting building sites.

CASE STUDY

Ulceration of Charcot's osteoarthropathy of the mid-foot

A 54-year-old man with type 2 diabetes mellitus of 12 years' duration and a mid-foot Charcot's osteoarthropathy, developed an ulcer over the bony prominence on the plantar surface of his rockerbottom deformity which remained unhealed for 7 years. He was referred to the diabetic foot clinic and was treated with a total-contact cast and Dermagraft. The foot healed in 7 months and has remained healed (Fig. 4.32a,b).

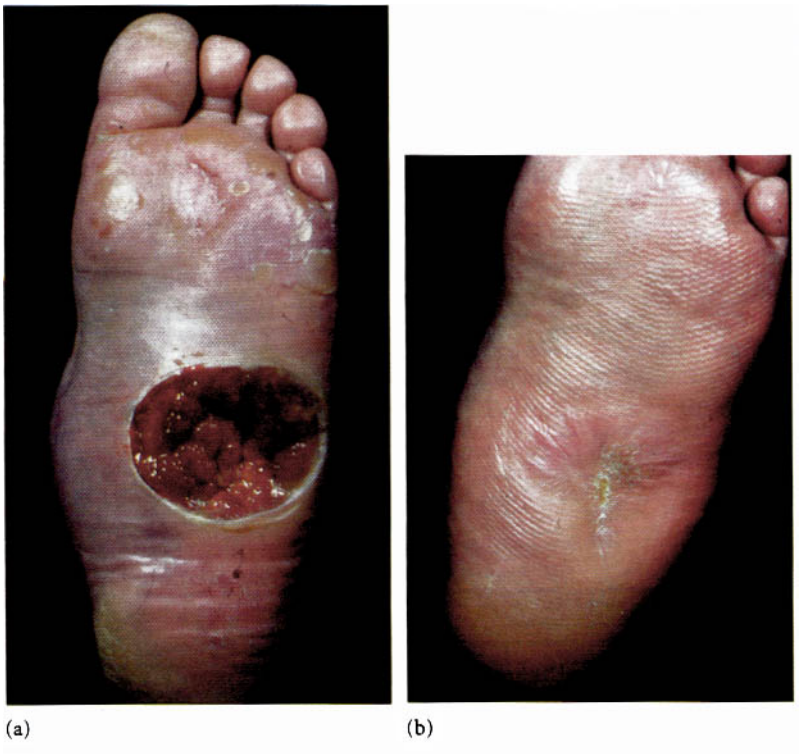


Fig. 4.30 (a) Indolent ulcer under a rockerbottom deformity associated with Charcot's osteoarthropathy of the mid-foot which had been present over 6 years. (b) The same ulcer healed in 30 months after being initially treated with Dermagraft.

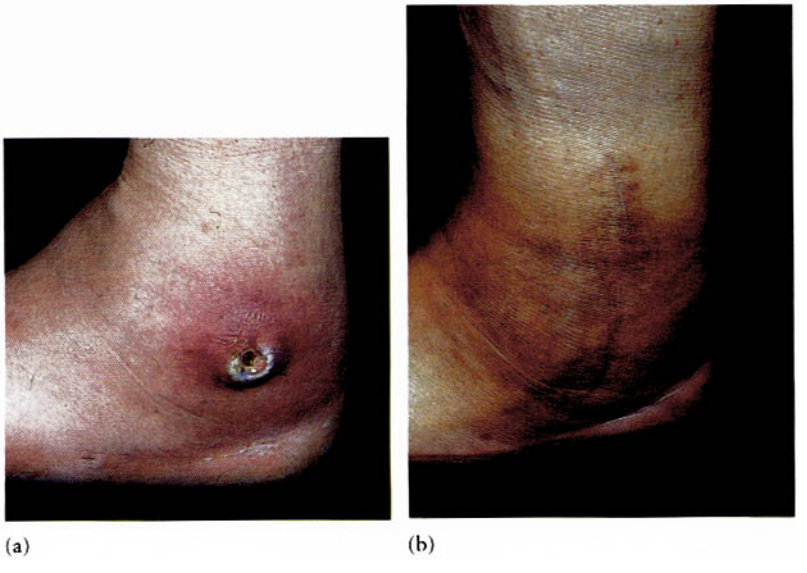
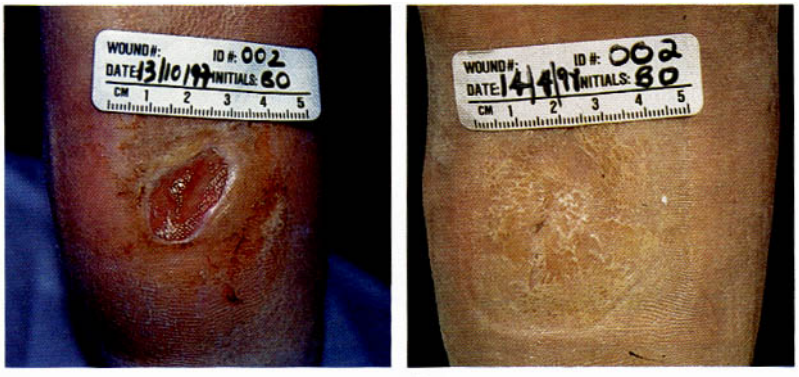


Fig. 4.31 (a) Indolent ulceration over the lateral malleolus in an unstable ankle with Charcot's osteoarthropathy of the hindfoot. (b) The ulcer has healed following internal fixation to stabilize the ankle. The healed surgical wound can be seen.

Key points

- Patients who develop callus over a rockerbottom deformity need regular podiatry to debride callus
- Ulcers associated with deformity of Charcot's osteoarthropathy should be treated with a total-contact cast
- Advanced wound products may be helpful in conjunction with a cast
- Surgical removal of the bony prominence is a simple procedure which can achieve healing.



(a) (b)

Fig. 4.32 (a) An ulcer over a bony prominence on a rockerbottom foot with Charcot's osteoarthropathy of the mid-foot. (b) The foot healed after 6 months with Dermagraft and off-loading.

Ulcers over the Achilles tendon

This is another notoriously difficult site to heal. It is an unusual site for ulceration and is usually triggered by unsuitable footwear or is a pressure lesion in an immobile patient. When tendon is exposed in the base of the ulcer the advice of a surgeon should be sought. Our colleague, E. Maelor Thomas, a founder member of our foot clinic and an orthopaedic surgeon, always said that dead tendon was the worst kind of sequestrum and should always be excised from ulcers.

We have used Hyaff to encourage granulation over healthy tendon, and have applied Apligraf to close a clean ulcer overlying a tendon.

Traumatic wounds

Puncture wounds

Puncture wounds (Fig. 4.33) are potentially serious injuries that may first appear as trivial superficial pinpoint wounds. These injuries are caused by a foreign body that may have penetrated the footwear as well as the foot. The foreign body (nail, needle, staple, glass, wooden splinter or other object) may inoculate bacteria into the wound resulting in severe infection. Some of the most serious foot infections we have seen have followed puncture wounds. In some cases the initial wound has fully healed but within a few days the 'volcano' has erupted with severe tissue destruction.

Treatment of puncture wounds is as follows:

- If possible, the foreign body should be inspected to see whether it is intact, or whether a piece has broken off which might remain in the foot
- The foot should be X-rayed and a radio-opaque foreign body sought. Even clear glass will usually show up
- The wound should be probed and irrigated, although this may be difficult when the portal of entry is small. In neuropathic patients it may be possible to enlarge the wound to ensure thorough cleaning. Local anaesthesia may be required for sensate individuals
- Wide-spectrum antibiotics should be prescribed
- The wound should be inspected at frequent intervals
- The patient should be asked to report any pain, swelling, discomfort or systemic signs or symptoms immediately
- Tetanus prophylaxis may be needed.

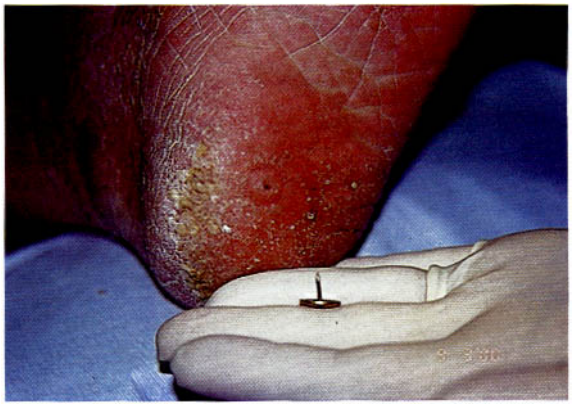
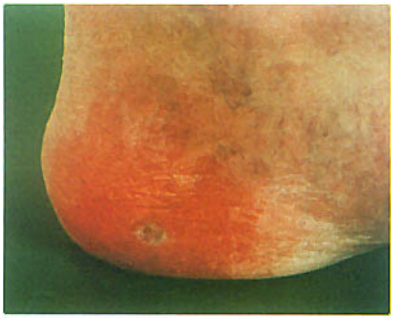


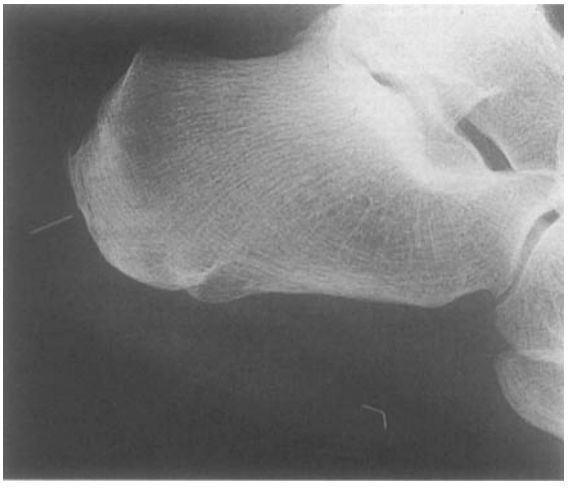
Fig. 4.33 This patient arrived at the foot clinic with a drawing pin still in her foot. The puncture wound can be clearly seen.

CASE STUDY
Foreign body in foot

A 68-year-old woman with insulin-treated type 2 diabetes of 20 years' duration complained of pain on the back of the left heel and a superficial ulcer surrounded by a halo of erythema (Fig. 4.34a). She was unaware of the cause of the ulcer. An X-ray showed two clipped-off insulin needles in the soft tissues of her heel (Fig. 4.34b). She had previously



(a)



(b)

Fig. 4.34 (a) The superficial ulcer of unknown aetiology surrounded by a halo of erythema. (b) X-ray reveals two clipped-off insulin needles embedded in the soft tissues.

had an education session with the diabetes specialist nurse who emphasized the need for safe disposal of used needles and syringes. The patient had decided to clip off the ends of needles so that they could not be harmful and used a pair of wire cutters from her husband's tool box to do this. Her eyesight was poor and some of the clipped-off needles were dropped on to her bedroom carpet which had a long shag pile.

She was seen by the surgeons who advised not to remove the needles. She was prescribed antibiotics and the ulcer healed in 4 weeks. She underwent further education on safe disposal of needles and syringes, and avoiding barefoot walking, and disposed of her shag pile carpet.

Key points

- Feet with ulcers of unknown cause should be X-rayed
- High-risk patients should not walk barefoot, even in the bedroom
- Ascertaining causes of ulceration can prevent recurrences.

Thermal trauma including burns

Severe tissue damage and ulceration can be caused by thermal traumas.

CASE STUDY
Burns

A 25-year-old male patient with type 1 diabetes mellitus of 14 years' duration, profound neuropathy and end-stage renal failure treated with dialysis, slept in a bed next to a central heating radiator. During the night, in his sleep, his leg slipped against the radiator. He sustained full-thickness burns to his leg, but only attended the diabetic foot clinic when these became malodorous. There was a moist leathery eschar with purulent discharge. He was admitted to hospital for intravenous antibiotics. The burns were surgically debrided and split-skin grafts applied from a donor site on his thigh. He healed in 5 months.

Key points

- We warn patients to position beds away from radiators
- The home environment should be adapted when patients develop neuropathy
- Full-thickness burns need skin grafting and specialist surgical care.

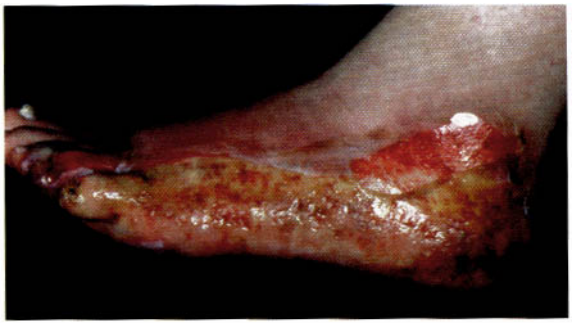
CASE STUDY
An unusual case of burns

A 48-year-old man with type 2 diabetes of 11 years' duration and profound neuropathy visited his local pub. The next morning when he checked his feet he found blisters on the apices of his left 1st and 2nd toes and came to the foot clinic. We felt the lesions looked like burns and asked him if he could possibly have burned his feet, but he denied this possibility. However, on his next visit to the pub his landlord told him that a heated foot rail had been fitted alongside the bar for his customers' comfort on cold winter nights.

Key points

- No matter how careful patients with neuropathy are, trauma is sometimes almost impossible to avoid
- Patients should be asked actively to explore how they were injured
- We warn patients about the local pub with red hot foot rails!

Superficial burns usually present as erythema or bullae and can be treated conservatively. Deep burns, which



foot were caused when the patient spilled boiling water on her foot. They were full-thickness burns which required specialist treatment from a burns unit.

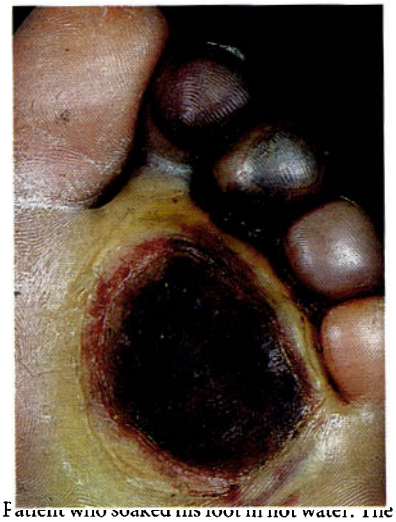


Fig. 4.36 Patient who soaked his foot in hot water. The resulting burns became secondarily infected leading to gangrene of the toes.

manifest themselves by white, devitalized tissue (Fig. 4.35) and subsequent eschar formation, need specialized care from a burns unit, as do all burns which are unhealed after 3 weeks. It can be difficult to assess the severity of recent burns to the diabetic foot since lack of pain may be due to diabetic neuropathy rather than full-thickness damage.

Recent burns should be seen at 48-h intervals until their depth is established.

Burns are common in neuropathic patients and the cause should always be established and action taken to prevent repetition. Infection is a serious complication of burns in the diabetic foot and prophylactic antibiotics may need to be given (Fig. 4.36).

Partial thickness burns are allowed to heal by secondary intention, as are some small full-thickness burns. Extensive full-thickness wounds need skin grafting.

Chemical burns may originate from proprietary remedies, including:

- Corn and callus removers, which contain strong acids or caustics
- Undiluted antiseptics applied directly to wounds
- Contact with noxious chemicals.

As with thermal burns, these can be difficult to assess. When corn cures have been used, as much macerated, acid-loaded callus as possible should be sharp debrided. Chemical burns should be cleansed with saline, covered with a dry dressing and reviewed within 48 h. Extensive burns or burns when tissue devitalization is obvious at first presentation should be referred to a specialist burns unit.

Zoophilic traumas

- Bites and scratches from domestic animals such as dogs and cats are common. Animal bites need tetanus prophylaxis. Deep bites may need surgical debridement
- Rat bites can be a problem. Paul Brand recommends that neuropathic patients keep a cat and avoid eating in bed in case crumbs attract rats
- Insect stings.

CASE STUDY

Severe reaction following insect sting (Fig. 4.37)

A 54-year-old male with type 2 diabetes of 8 years' duration, and a previous history of two episodes of cellulitis to his legs, suffered a painful sting from a large unidentified flying insect when on holiday in Spain. Within 1 h he felt cold and was 'shaking'. The following day he flew home. The leg deteriorated and developed blistering and ulceration. He attended his local accident and emergency department. He was given intravenous amoxicillin and flucloxacillin as an inpatient and discharged after 9 days (Fig. 4.37). The leg healed in 8 weeks. He was referred to the diabetic foot clinic because he was concerned about residual oedema and pigmentation. A duplex venogram was performed which revealed a deep vein thrombosis of the posterior tibial vein and he was anticoagulated.

Key points

- Insect stings can produce intensive erythema and oedema in the diabetic lower limb with local histamine release
- Long term there can be severe postinflammatory hyperpigmentation

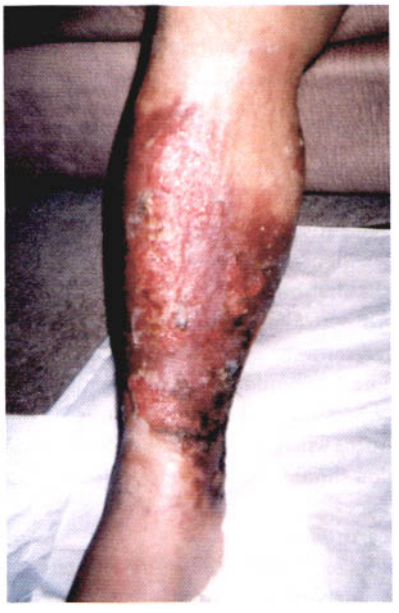


Fig. 4.37 Severe reaction following insect sting showing extent of inflammatory response.

- When swelling of the leg is persistent, it is wise to investigate for deep vein thrombosis.

Iatrogenic lesions

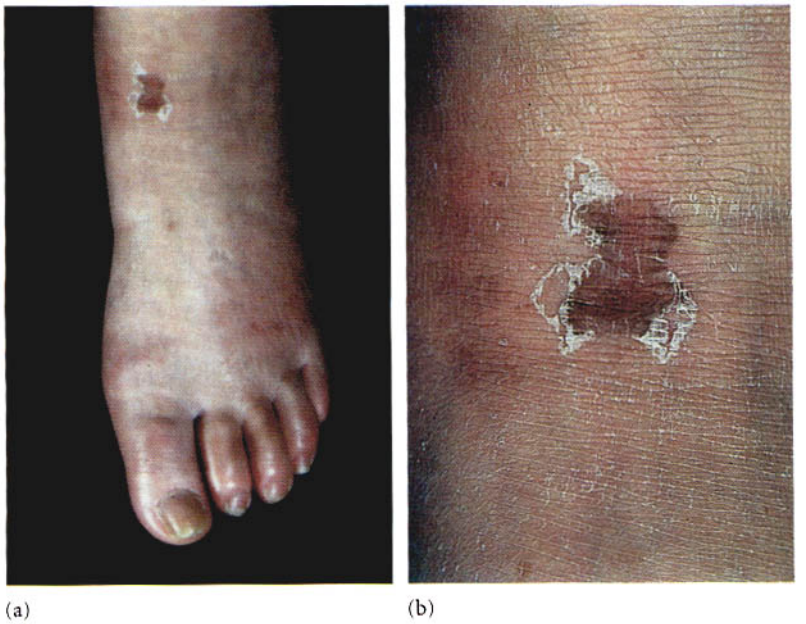
Common examples include:

- Tape applied to atrophic skin and ripped off
- Tight bandages. We have seen a 53-year-old woman with type 2 diabetes mellitus of 13 years' duration and oedematous feet, who sustained a burn to the dorsum of the foot. She applied a sterile dressing held in place by a bandage which completely encircled the foot and ankle, and made an appointment to be seen at the diabetic foot clinic. When the bandage was removed she had developed superficial necrosis from an over-tight bandage and fluctuant oedema. The bandage was replaced with a light tubular bandage and the foot healed in 2 weeks
- A further patient with cardiac failure developed a lesion in tight bandages (Fig. 4.38)
- Bulky dressings taking up too much space in unadjusted footwear
- Injection of cortisone into painful heels. Cortisone injection can lead to ulceration and full-thickness necrosis which will need surgical debridement. We have seen a case of extensive calcification of the soft tissues of the heel associated with pain and ulceration following a cortisone injection into the heel.

Artefactual ulcers

Some patients cause ulcers by pulling skin off their feet (Fig. 4.39) or applying noxious substances, or prevent ulcers from healing.

The patients we have seen have been young and mostly female, and have suffered in the past from eating disorders or 'brittle' diabetes.



(a) (b)

Fig. 4.38 (a) This patient developed necrosis on the front of the ankle from a bandage which became too tight when her oedema increased. (b) A close-up view of the iatrogenic lesion shown in (a). The conventional bandage was replaced with light tubular bandage which could accommodate her oedema.

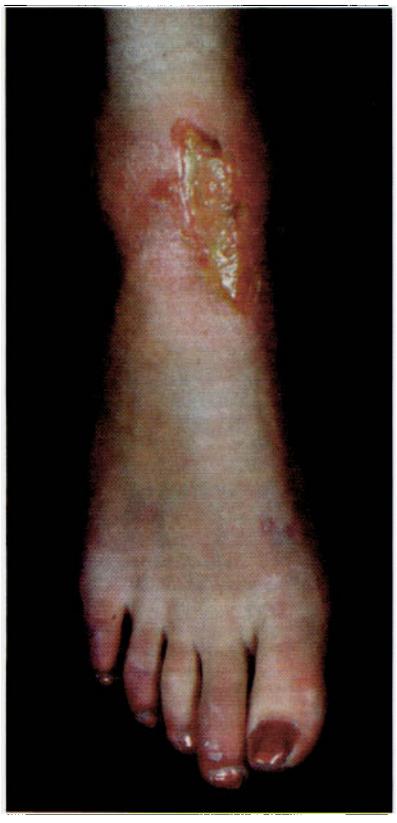


Fig. 4.39 This girl with peripheral neuropathy scratched the skin off her ankle and kept the wound open for 7 weeks.

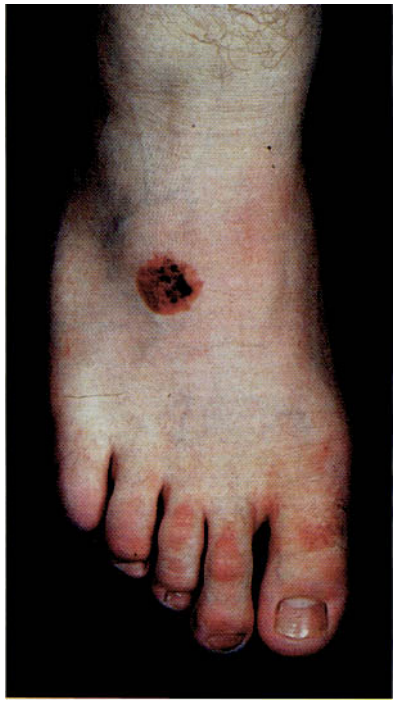


Fig. 4.40 Ulcer on the dorsum of the foot. Note the straight edge of the dark portion of the wound bed. Straight edges can be indicative of artefactual ulcer.

CASE STUDY
Artefactual ulcer

A 23-year-old woman with type 1 'brittle' diabetes of 12 years' duration, underwent a sural nerve biopsy which failed to heal for 3 months, but healed in 1 week under a tamper-proof dressing. She returned to clinic 3 months later with an ulcer on the dorsum of her foot. Parts of the ulcer bed had unusual morphometry with straight edges as shown in Fig. 4.40. It was thought that she might have an allergy to the dressing used and she was referred to the dermatologists. They did a patch test, tested the pH of the lesion on her foot and concluded that she had dipped a dressing in caustic lavatory cleaner and applied it to the foot. Following this episode she attended clinics at another hospital, had numerous admissions for infected skin lesions, developed end-stage renal failure and died aged 28 from sepsis.

Key points

- Morphologically unusual ulcers may be associated with artefactual disorder
- We consider the diagnosis of artefactual disorder in unusual ulcers and liaise with dermatologists and psychiatrists as necessary
- We do not 'confront' patients when artefactual disorder is suspected.

Many such patients have numerous admissions with severe foot infections or ketotic episodes related to foot infections. This is a difficult condition to treat and involvement of a psychiatrist with a special interest in artefactual disorders may be useful. Patients who cause their own ulcers or deliberately prevent their ulcers from healing are aware of what they are doing but have little insight into their motives. They can injure themselves with impunity because of their neuropathy (Fig. 4.41). Tamper-free dressings or unremovable casts probably provide the best means of achieving short-term healing, but the long-term outlook is poor. Confrontation and admonishment are unhelpful, as the patient will just go elsewhere for his care and will be at increased risk with

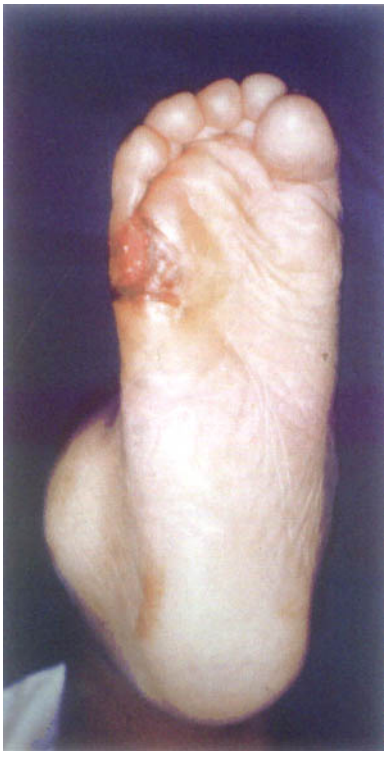


Fig. 4.41 This girl pulled skin off her foot and caused an ulcer which remained open for 3 years. It healed in 3 weeks when a tamper-free total-contact cast was applied.

clinicians who are not aware of what is going on. Many of these patients are profoundly hypochondriac and only feel safe and in control when they are causing the foot problem.

We have also seen a 41-year-old patient with 'brittle' type 1 diabetes mellitus of 24 years' duration, who had numerous hospital admissions for infected ulcers of the feet and legs. On one occasion his leg ulcers were grafted using split-skin grafts and the evening before his proposed discharge from hospital, the dressings were removed and the skin grafts scratched off. On two occasions during that admission, glass was broken on the ward and found in his foot ulcers. He was discharged, attended another hospital, and underwent bilateral below-knee amputations within 6 months.

Malignant ulcers

We have seen a number of cases of malignant tumours masquerading as diabetic foot ulcers. Several required more than one biopsy to confirm the diagnosis.

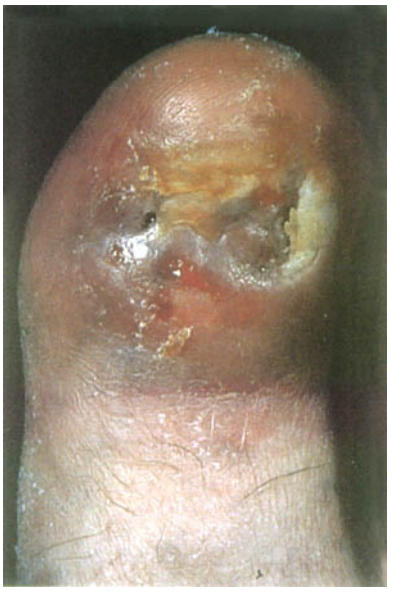


Fig. 4.42 This subungual lesion, shown 1 week following biopsy, was a squamous cell carcinoma.

'Cauliflower' appearance and development within a scar were common factors. We have also seen amelanotic malignant melanoma masquerading as subungual ulceration and basal cell and squamous cell carcinomas which were thought to be plantar warts.

Pigmented lesions which enlarge and develop satellite lesions, an irregular border, erosions or ulceration should be seen urgently by the dermatologist (Fig. 4.42). These lesions could be malignant melanoma: the lower leg is a common site. Some melanomas are not associated with pigment.

Squamous cell carcinoma, and rarely a basal cell carcinoma, may develop in an indolent diabetic foot ulcer or scar from previous ulcer or surgery.

CASE STUDY
Malignant melanoma

A 78-year-old man with type 2 diabetes of 5 years' duration was referred with a discharging subungual ulcer on his right hallux which had been present for 8 years. Pedal pulses were palpable. The footwear was narrow and insufficiently roomy, and he was asked to purchase shoes with a deep toe box which would not cause pressure on the nails.

The toe nail was cut back. The patient wore suitable shoes, and the ulcer improved, with less discharge, but

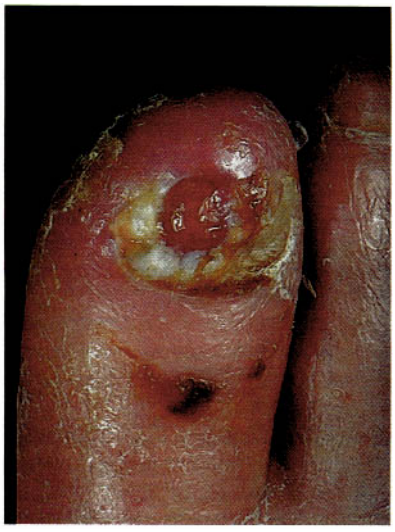


Fig. 4.43 This subungual ulcer failed to heal and was referred to the dermatologists. Biopsy revealed an amelanotic malignant melanoma.

failed to heal completely. An X-ray showed no signs of osteomyelitis.

When the foot failed to heal after 3 months, the patient was referred to dermatology for an opinion (Fig. 4.43). Although the dermatologists were not really suspicious that there was any neoplasia present because the area was not elevated, they felt that it would be wise to check, and a biopsy was arranged. A malignant melanoma was diagnosed and the toe was amputated. The patient is alive and well 3 years later and has regular follow-up appointments with the dermatologists.

Key points

- Ulcers which fail to heal after full treatment should be regarded as suspicious lesions and referred to the dermatologists for biopsy
- Malignant melanomas may be amelanotic
- Malignant lesions may not be elevated.

PRACTICE POINTS

- Ulcers need mechanical, wound, vascular, microbiological, metabolic and educational control
- Mechanical control consists of total-contact casts, Scotchcast boots, crutches, wheelchairs, Aircasts, PRAFOs, custom-made walking AFOs, rest and elevation as appropriate
- Wound control involves sharp debridement and dressings and may need advanced wound healing products or supplementary treatments
- To obtain vascular control it is necessary to assess the vascular status of all ulcerated feet. Neuroischaemic feet may need vascular intervention
- To achieve microbiological control, neuroischaemic feet, burns and puncture wounds will need early antibiotic therapy
- Optimizing control of blood glucose, blood pressure and blood lipids and helping patients to stop smoking will achieve good metabolic control
- Educational control involves teaching patients the need for rest, debridement, regular dressings and early reporting of problems, and explaining how lifestyle changes can prevent relapse.

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5 Stage 4: the infected foot

It will but skin and film the ulcerous place
Whiles rank corruption, mining all within,
Infects unseen . . .
(Hamlet, III, iv, William Shakespeare)

PRESENTATION AND DIAGNOSIS

When feet reach stage 4, they have developed infection. This is a highly significant staging post on the road to amputation. Although amputation may result from severe ischaemia or gross deformity of Charcot's osteoarthropathy, this is rare, and infection is usually the final common pathway to amputation.

More people undergo major amputation because of combined diabetes and infection than for all other causes. In this chapter we describe how infected diabetic feet are managed at King's College Hospital, London, both in the outpatient diabetic foot clinic and on the wards.

Diabetic foot patients with diffuse spreading cellulitis and extensive soft tissue infection are often in immediate danger of losing life and limb. We discuss the polymicrobial organisms associated with deep wound infections and detail the meticulous care these patients need. We have tried to illustrate these points with very detailed case studies. We hope that we have outlined a good case for multidisciplinary care, with daily input from medical and surgical teams for inpatients with severe infections.

Our aim has always been to devise a practical approach which can diagnose infections early, treat them rapidly and aggressively, and thus prevent amputations. We are guided in our decision making by a combination of the signs or symptoms of infection, the results of properly taken wound swabs and tissue cultures, and our knowledge of individual patients. Our guiding principle is that we do not forget that 85% of major amputations in people with diabetes begin with a 'clean' ulcer.

Spectrum of clinical presentations

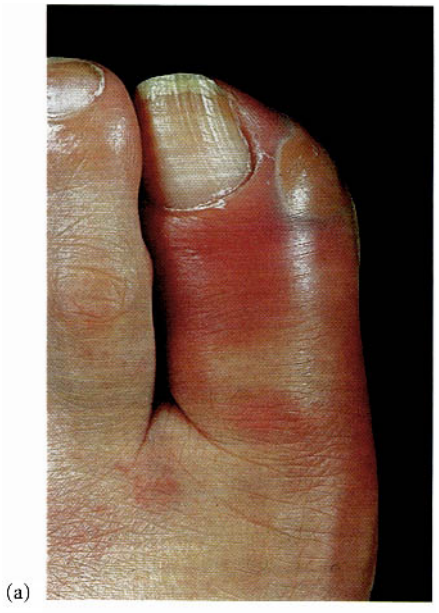
The early warning signs of infection in the diabetic foot patient may be very subtle and masked by neuropathy or ischaemia, either or both of which may prevent Galen's classical signs and symptoms of rubor, calor, dolor and loss of function from being evident. Symptoms of wound infection may be diminished in diabetic foot patients. Patients with retinopathy may not see the erythema of cellulitis.

CASE STUDY
Local signs of infection not noted by patient

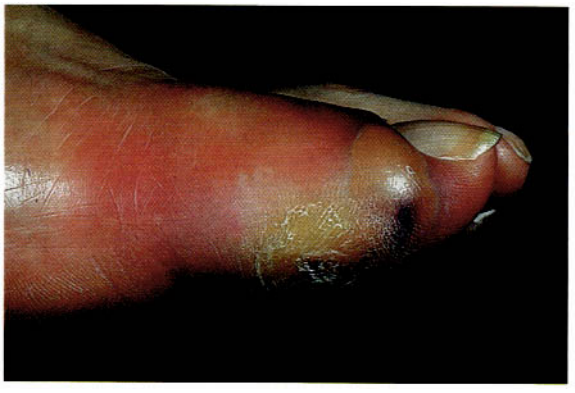
A 53-year-old lady with type 1 diabetes of 25 years' duration, proliferative retinopathy with reduced vision, peripheral neuropathy and hallux rigidus developed a neuropathic ulcer under callus on the plantar surface of her right hallux. She was warned of the usual danger signs of deterioration (redness, warmth, swelling, pain, purulent discharge) but did not return to clinic until her routine appointment. Callus had grown over the ulcer preventing drainage and the toe had become cellulitic (Fig. 5.1a,b). Callus was debrided and pus drained (Fig. 5.1c). A deep wound swab was taken and oral amoxicillin 500 mg tds and flucloxacillin 500 mg qds were prescribed. She was reviewed the next day. The toe had not improved and she was admitted for bed rest and intravenous antibiotics according to our protocol, namely amoxicillin, 500 mg tds, flucloxacillin 500 mg qds, metronidazole 400 mg tds and ceftazidime 1 g tds. The swab taken at her outpatient clinic visit grew *Staphylococcus aureus* and *Streptococcus* group B. The metronidazole and ceftazidime were stopped when this result became available. She was discharged after 4 days and the ulcer healed in 6 weeks.

Key points

- Patients with impaired vision and neuropathy cannot be relied upon to detect signs of infection such as cellulitis



(a)



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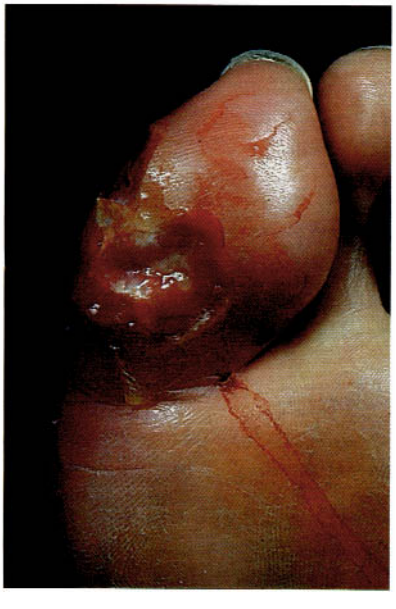


(c)

Fig. 5.1 (a) Cellulitis of the right hallux. (b) Cellulitis of the right hallux and a collection of pus under callus. (c) Callus debrided and pus drained.

- Lack of discharge does not necessarily mean that an ulcer is healed: it can indicate that callus has sealed it off, preventing drainage. If this happens the foot can deteriorate rapidly
- *Staphylococcus aureus* and *Streptococcus* group B in combination can act synergistically to produce a rapidly spreading infection.

CASE STUDY

Asymptomatic foot infection under a total-contact cast

A 45-year-old man with type 1 diabetes of 35 years' duration who had previously undergone amputation of all the toes of his left foot, had a deep plantar neuropathic ulcer and was fitted with a total-contact cast. He was given oral amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 400 mg tds and ciprofloxacin 500 mg bd but did not take them. When he returned after 1 week for a routine cast check, the cast was removed to expose a cellulitic foot and ankle. There was an area of oedema and crepitus behind the lateral malleolus and X-ray revealed gas in the soft tissues. He was apyrexial and normoglycaemic. He was admitted, underwent operative debridement of the ankle region and given amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds intravenously. Tissue debridings were sent from theatre for culture and grew anaerobic *Streptococcus* which was sensitive to metronidazole. The ankle healed in 5 weeks. During the period of bed rest in hospital the plantar ulcer also healed.

Key points

- Infection in the diabetic foot can present without pain, fever or hyperglycaemia
- Infection can develop within a total-contact cast without warning signs or symptoms

- Infections that occur within a total-contact cast should be treated promptly and aggressively to prevent serious sequelae
- Gas in the soft tissues is not necessarily caused by *Clostridium perfringens* but by other anaerobes and Gram-negative bacteria.

CASE STUDY
Pain as the sole early warning of infection

An 82-year-old woman with type 2 diabetes of 38 years' duration, profound peripheral neuropathy and a previous history of neuropathic ulceration, complained of pain in her hallux at a routine foot clinic appointment. There was no history of trauma. On visual examination and palpation, nothing abnormal was detected, X-ray was unremarkable, and she was apyrexial. She was Afro-Caribbean with heavy pigmentation. She was advised to keep a close eye on the toe and return immediately if it deteriorated and to return in 48 h for review. When she came back 2 days later she had an infected ulcer on the apex of the toe, severe unilateral oedema and cellulitis spreading up the leg (Fig. 5.2). She was admitted to hospital and given intravenous amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. The toe healed in 2 weeks.

Key points

- Pain may have been the first symptom of infection in this neuropathic foot
- Elderly neuropathic patients complaining of new pain of unknown aetiology with no other clinical signs or symptoms should be rechecked within 48 h
- It is difficult to detect cellulitis in pigmented skin.

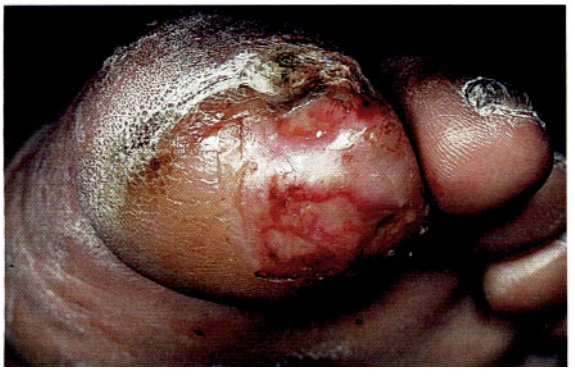


Fig. 5.2 Ulceration at the apex of the hallux with cellulitis.

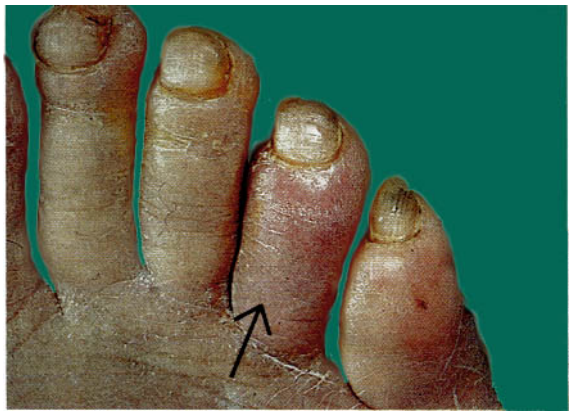


Fig. 5.3 The first sign of infection: 4th toe becomes slightly pink.

Early warning signs of infection and signs of deterioration should be searched for with great assiduity in all diabetic foot patients (Fig. 5.3), especially those with breaks in the skin. Some infected breaks in the skin will be obvious; others will only make their presence known by:

- Discharge or exudates which collect under callus or skin and present as a blister
- Colour changes under callus or nail plate
- Pain or discomfort
- Swelling
- Warmth
- Erythema.

We believe that if the practitioner waits for florid signs of infection to develop then valuable time is lost. We act upon the early signs of infection.

CASE STUDY
Delayed presentation of infection masked by callus

A 72-year-old woman with type 2 diabetes of 20 years' duration and peripheral neuropathy developed 'a dark spot' on the apex of her right 3rd toe and applied sterile gauze which was replaced at weekly intervals. The toe did not improve and regular dressings were continued for several months until her daughter noticed that the toe had become pink, and brought her up to the diabetic foot clinic. Her pedal pulses were strong and bounding. A plaque of callus covered the entire apex of the pink toe adjacent to the nail, which was thickened and difficult to distinguish from the callus. The callus was debrided with a scalpel to reveal an abscess cavity extending to the nail bed and the proximal phalanx was exposed. A speci-

men of pus grew *Staphylococcus aureus*. She was admitted to hospital and given intravenously amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. When the result of the culture was available this was reduced to flucloxacillin only. The foot healed in 1 week. She was followed up by the diabetic foot service and the problem did not recur.

Key points

- Callus may conceal deep infection which is producing very few physical signs of inflammation
- The well-perfused infected neuropathic foot responds well to debridement and drainage, pressure relief and appropriate antibiotics
- Patients with a history of ulceration and infection developing under callus need follow-up care to prevent recurrence.

The most common manifestation of infection is cellulitis, usually secondary to an ulcer, and presenting as a redness or erythema.

However, this stage covers a spectrum of presentations under the general chapter heading of infection, ranging from local infection of the ulcer to spreading sepsis, sloughing of soft tissue and finally, vascular compromise of the skin secondary to sepsis, seen as a blue discolouration. This spectrum occurs in both neuropathic and neuroischaemic feet. In the presence of neuropathy and ischaemia, signs of inflammation are often diminished, yet the final pathway of infection is to overwhelming destruction of the foot. It is thus important to treat even the initial stages with considerable respect.

Infection in the diabetic foot is a protean entity, but may be classified as follows:

- *Ulcer with local signs of infection*
- *Ulcer with surrounding erythema*
- *Ulcer with diffuse spreading erythema*
- *Ulcer with extensive deep soft tissue infection*
- *Ulcer with extensive erythema and with blue/purple/black discolouration of surrounding tissues.*

Any break in the skin can lead to cellulitis and occasionally cellulitis is found without an obvious break in the skin being present.

Any of these presentations may be complicated by underlying osteomyelitis. Clinically, osteomyelitis may be suspected when a sterile probe inserted into the base of the ulcer penetrates to bone. This may happen in an apparently clean, uninfected ulcer, but osteomyelitis must still be suspected. X-ray and magnetic resonance imaging (MRI) may be helpful in the diagnosis of osteomyelitis, which may be confirmed by bone biopsy and a positive bone culture.

Any of the above presentations may be accompanied by systemic signs.

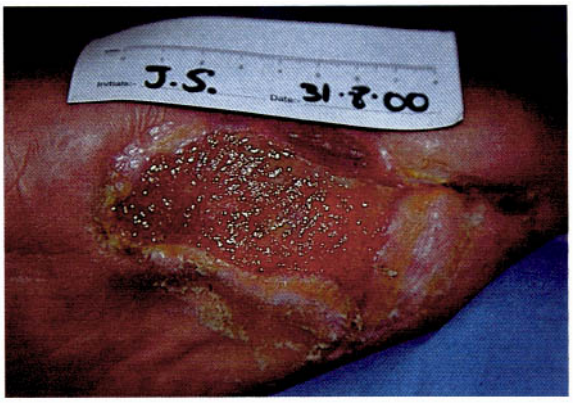


Fig. 5.4 The granulation tissue in this large ulcer has become friable and bleeds easily. This is an early sign of infection.

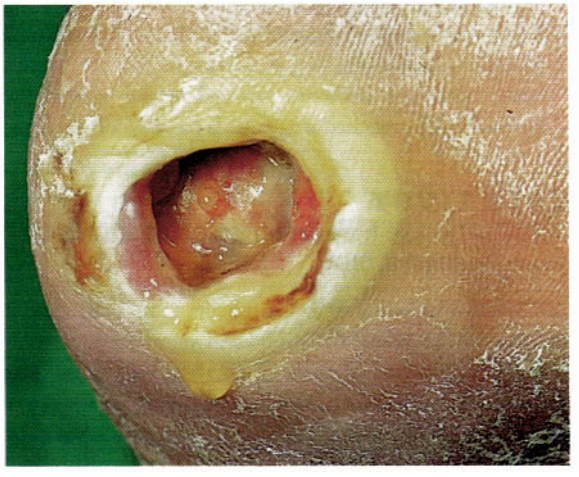


Fig. 5.5 Exudate is dripping out of this neuropathic ulcer.

Ulcer with local signs of infection

Local signs that an ulcer has become infected include any or all of the following:

- Pain
- Base of the ulcer changes from healthy pink granulations to yellowish or grey tissue
- Increased friability of granulation tissue (Fig. 5.4)
- Increased amount of exudate (Fig. 5.5)
- Exudate changes from clear to purulent

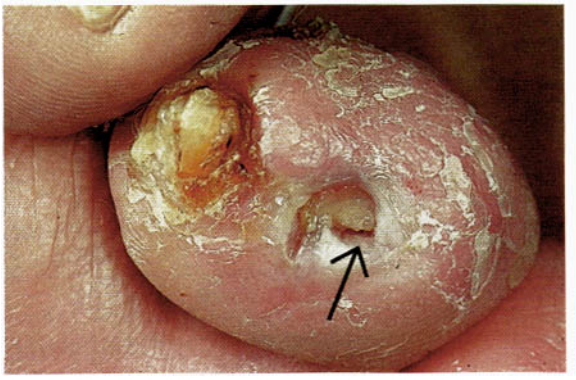


Fig. 5.6 A deep sinus has appeared in the base of this ulcer.

- Unpleasant smell
- Sinuses develop in an ulcer (Fig. 5.6)
- Edges may become undermined so that a probe can be passed under the skin
- Bone or tendon becomes exposed in the base of the ulcer or can be reached if a probe is passed along a sinus.

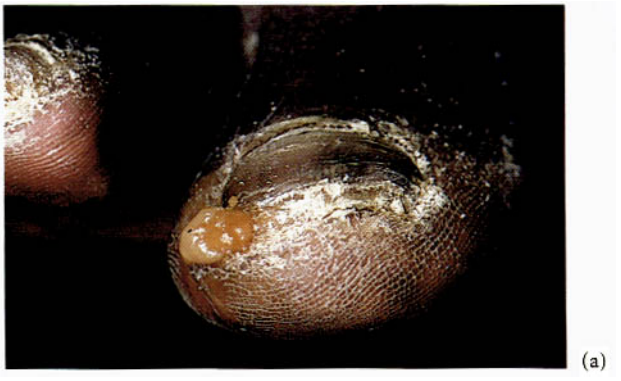
CASE STUDY

Pain as the sole manifestation of infection in a neuroischaemic foot

A 77-year-old blind Afro-Caribbean man with type 2 diabetes of 22 years' duration, and peripheral vascular disease complained of pain in his right hallux and was brought to the foot clinic the same day (Fig. 5.7a). There was no swelling or cellulitis but pain was exacerbated by gentle pressure on the nail plate and a small area of nail plate close to the medial sulcus was very gently pared away to expose a small abscess under the nail which was drained (Fig. 5.7b). A deep swab was sent for culture and the abscess cavity was irrigated with normal saline and dressed with Melolin and Tubegauz; amoxicillin 500 mg tds and flucloxacillin 500 mg qds were prescribed. The wound swab grew *Staphylococcus aureus* and *Streptococcus* group B. The toe healed in 1 month.

Key points

- Pain may be the sole manifestation of infection in the diabetic neuroischaemic foot and is not always due to ischaemia
- Pain from infection under the nail is exacerbated by gentle pressure on the nail plate
- A podiatrist can achieve successful drainage of infection without causing further trauma to the ischaemic toe



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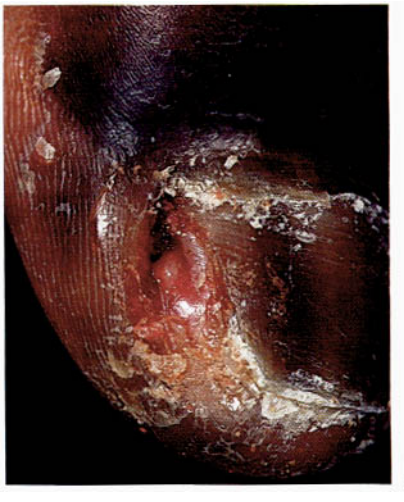


(b)

Fig. 5.7 (a) This patient complained of pain on the medial side of his toe. There was no swelling or cellulitis. The nail has been gently pared back and a small abscess is draining. (b) Abscess site post drainage.

- Infection in the neuroischaemic foot may not be associated with swelling
- A heavily pigmented skin makes cellulitis difficult to detect.

Ulcer with surrounding erythema

There will usually be local signs of infection as described above. There is a localized erythema, warmth and swelling usually associated with ulceration, although the portal of entry of infection may be a corn, callus, blister, fissure or any other skin break (Fig. 5.8).

In the darkly pigmented foot, cellulitis can be difficult to detect, but careful comparison with the other foot may reveal a tawny hue.

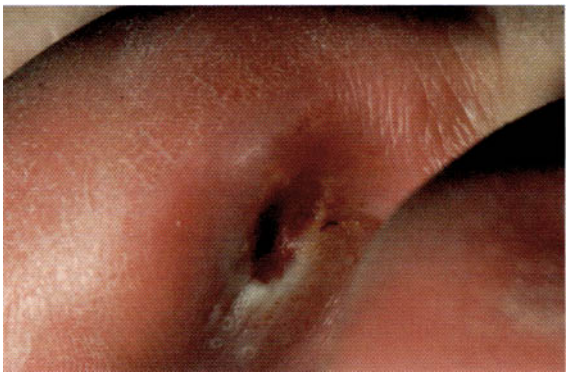


Fig. 5.8 This interdigital ulcer has associated local erythema.

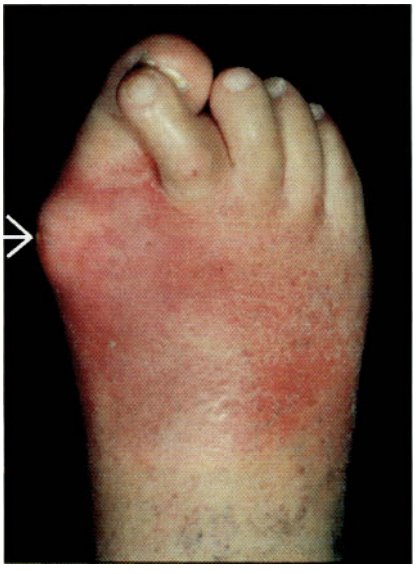


Fig. 5.9 There is diffuse cellulitis spreading over the dorsum of the foot from an ischaemic ulcer on the medial border (arrow).

Ulcer with diffuse spreading erythema

There is an intense widespread erythema and swelling often with lymphangitis, and regional lymphadenitis (Fig. 5.9).

Ulcer with extensive deep soft tissue infection

This involves the skin and subcutaneous tissues. This is a severe infection involving the deep soft tissues of the foot. In the presence of neuropathy, pain and throbbing may be absent, but if present this is a danger sign, usually indicating serious infection with pus within the tissues. Palpation may reveal fluctuance, suggesting abscess formation. There may be bulging of the plantar surface of the foot.

Discrete abscesses are relatively uncommon in the infected diabetic foot. Often there is a generalized sloughing of the ulcer and surrounding subcutaneous tissues which eventually liquefy and disintegrate. A probe can often be inserted deep into the foot. These infections are often polymicrobial and both Gram-positive and Gram-negative organisms are present together with anaerobes.

CASE STUDY

Hidden depths—unsuspected soft tissue infection complicating apparently superficial heel ulceration under callus

A 56-year-old man with type 2 diabetes of 12 years' duration and peripheral neuropathy trod on a nail while walking barefoot. The wound healed after 6 days, but the heel developed a callus which became painful after 2 weeks so he sought advice from the diabetic foot service. The callus was debrided and the underlying skin appeared to show superficial ulceration only (Fig. 5.10a). However, when the heel was palpated the patient complained of pain, and careful inspection revealed a deep sinus from which a bead of pus could be expressed (Fig. 5.10b). He was admitted for intravenous antibiotics, a surgical opinion was sought and he underwent extensive operative debridement of infected sloughy tissue the same day (Fig. 5.10c). The large residual defect healed after 7 months (Fig. 5.10d).

Key points

- Puncture wounds can lead to deep infections
- Pain on palpation in the ulcerated neuropathic foot is often a symptom of severe underlying infection
- Careful palpation may express pus and reveal a deep sinus
- Puncture wounds may be complicated by cellulitis and lead to this presentation. Bacteria are inoculated at the base of the puncture wound and then track back towards the surface of the skin, with infection eventually manifesting itself as a cellulitis and extensive deep soft tissue infection.

Ulcer with extensive erythema and with blue/purple/black discolouration of surrounding tissues

Infection can also present as a bluish-purple discolouration when there is inadequate supply of oxygen to the soft tissues.

This is caused by increased metabolic demands of infection and a reduction of blood flow to the skin,

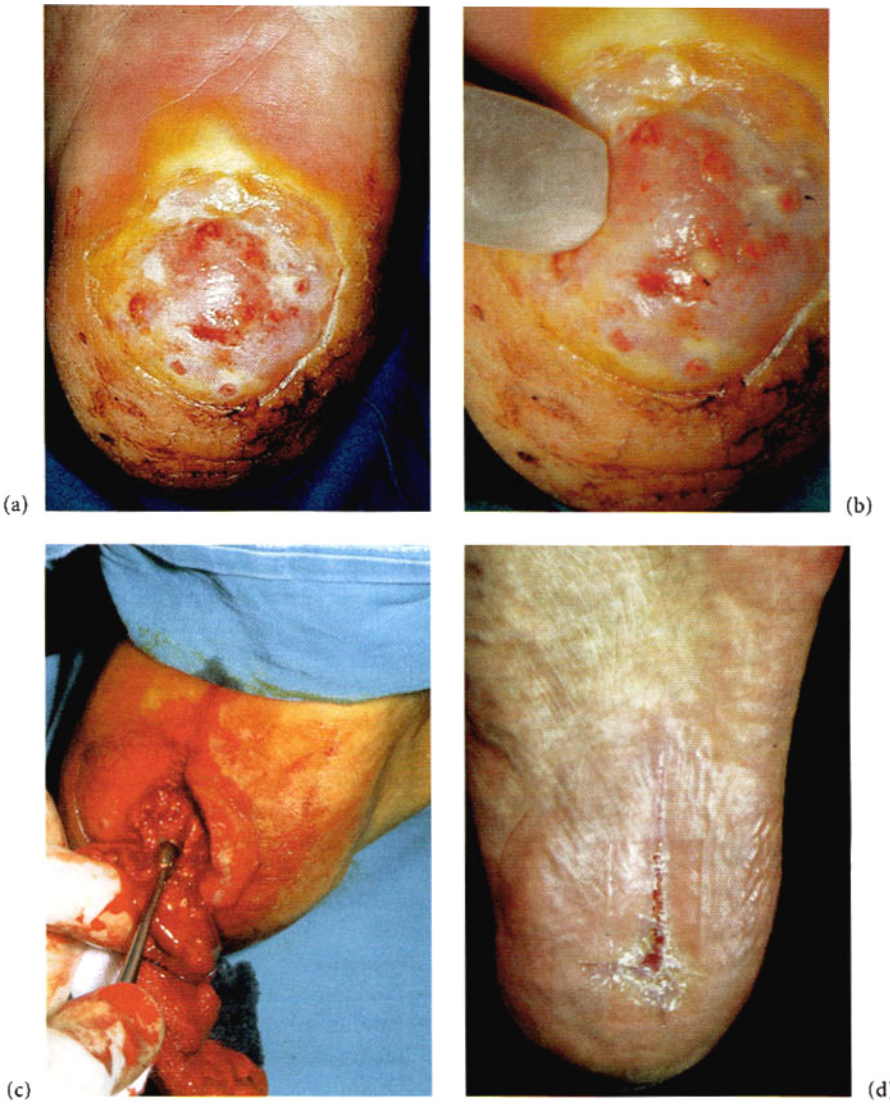
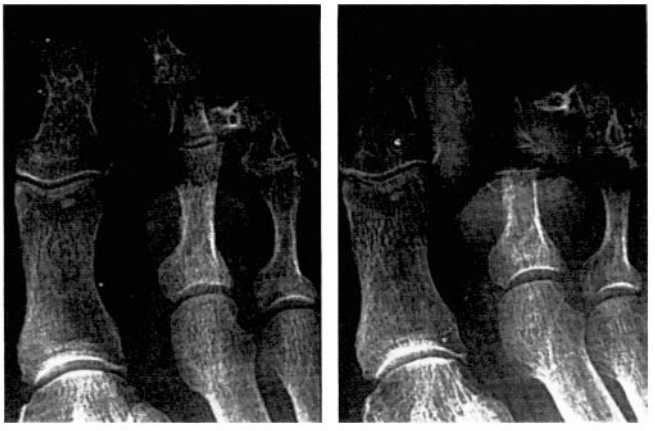


Fig. 5.10 (a) Callus has been debrided and ulceration appears to be shallow. (b) Palpation extrudes a bead of pus from deep in the heel. (c) Surgical debridement of infected tissues. (d) The foot healed after surgical debridement.

secondary to a septic vasculitis of the cutaneous circulation. Blue discolouration can occur in both the neuropathic and also the neuroischaemic foot, particularly in the toes, and in the neuroischaemic foot must not be automatically attributed to worsening atherosclerosis.

In very severe cases of cellulitis, bluish-purple or black discolouration of the skin develops with blistering. Purple blebs may indicate subcutaneous necrosis. A probe can often be inserted deep into the foot.

Systemic symptoms and signs may be present in the patient whose foot has extensive diffuse cellulitis, deep soft tissue infection or blue discolouration. However, systemic signs and symptoms are notoriously absent in many severe infections of the diabetic foot. Among patients hospitalized for severe infections only 12–35% have significant fever and only 50% of episodes of severe cellulitis will provoke a fever or leucocytosis. However, when a fever is present it usually indicates a severe



(a) (b)

Fig. 5.11 (a) The X-ray shows intact distal phalanx of the 2nd toe. (b) Destruction of the distal phalanx 2 weeks later indicative of osteomyelitis.

infection. The deep spaces of the foot are usually involved with tissue necrosis, severe cellulitis or bacteraemia.

Other warning signs of severe infection which may or may not be present include:

- An unusually drowsy patient
- A patient who is shivering.

Severe subcutaneous infection by Gram-negative and anaerobic organisms produces gas which may be detected by palpating crepitus on the lower limb and can be seen on X-ray. The presence of gas does not automatically mean that the classical gas gangrene organism *Clostridium perfringens* is present. The most common cause is either Gram-negative organisms or other anaerobes.

Any of the above five presentations may be complicated by underlying osteomyelitis.

Osteomyelitis

- Osteomyelitis should be suspected when a sterile probe, inserted into the base of an ulcer, penetrates to bone. Usually osteomyelitis will present in association with soft tissue infection. Occasionally it may present as probing to bone in an apparently clean, non-infected ulcer
- It may present as obvious fragmentation of the bone within the ulcer bed which is easily visible
- It is most commonly diagnosed on X-ray (Fig. 5.11a,b). Loss of cortex, fragmentation and bony destruction on X-ray are signs of osteomyelitis
- Chronic osteomyelitis of a toe has a swollen, red, sausage-like appearance (Fig. 5.12).

Rarely, we have seen metastatic infection from a primary infection in the foot.

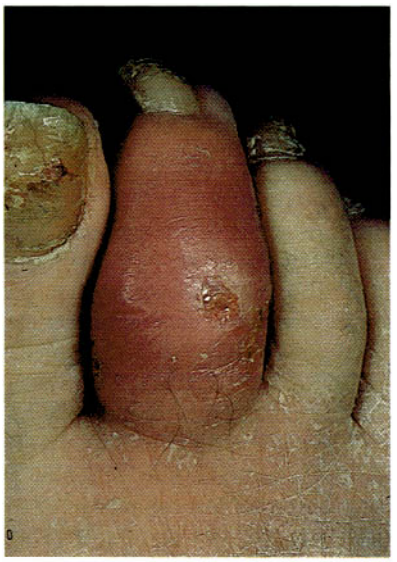


Fig. 5.12 'Sausage toe' with ulceration and cellulitis indicates osteomyelitis.

CASE STUDY

Bacterial discitis of the lumbar spine as a complication of longstanding neuropathic ulceration in a foot with Charcot's osteoarthropathy

A 55-year-old man with insulin-treated type 2 diabetes of 15 years' duration, peripheral neuropathy and impaired liver function due to previous hepatitis B infection presented late with acute Charcot's osteoarthropathy of

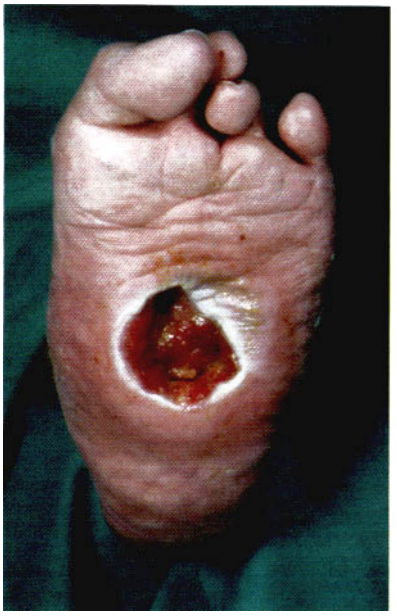


Fig. 5.13 The patient has undergone surgical debridement of his infected foot with rockerbottom deformity and plantar ulceration.

the right foot. Two years subsequently he developed neuropathic ulceration of the left 2nd toe. The ulcer became infected and he was admitted to hospital for intravenous antibiotics and underwent a ray amputation. He was discharged after 3 weeks. His left foot became red, swollen and warm, and Charcot's osteoarthropathy was diagnosed. He declined a total-contact cast but said that he would rest the foot as much as possible. (Two nights later one of the authors saw him in Central London at a Promenade Concert hiding behind a pillar so as not to be seen and reprimanded for not resting!) He developed a rockerbottom deformity with a neuropathic ulcer over the bony prominence, and re-presented with severe infection and spreading cellulitis. He was admitted for intravenous antibiotics and surgical debridement of the foot, with removal of infected soft tissue (Fig. 5.13). Three months later, he complained of severe pain in the region of his lumbar spine. An MRI scan revealed infection of the disc between the 4th and 5th lumbar vertebrae, which was treated with long-term antibiotics and resolved.

Key points

- Metastatic infection can develop from a primary infection in the foot
- Presentation of metastatic infection may be delayed.

Differential diagnosis of the cellulitic foot

Charcot's osteoarthropathy and cellulitis

It is important to distinguish between the red hot swollen foot of Charcot's osteoarthropathy and the red hot swollen cellulitic foot (see p. 53). Infection and Charcot's osteoarthropathy can sometimes be present together in the same foot. If in doubt, treat for both.

CASE STUDY

Relapse of Charcot's osteoarthropathy or infection?

A 60-year-old type I diabetic of 42 years' duration who had bilateral Charcot's osteoarthropathy affecting both feet and 12 years' previous history of ulcers and infections, was referred to the foot clinic with a hot, swollen left ankle and erythema over the medial malleolus. Both her feet were intact. The left foot was very painful on weightbearing. A provisional diagnosis of infection was made although we could not be sure that this was not a relapse of Charcot's osteoarthropathy. She was given intravenous vancomycin 1 g bd, ceftazidime 1 g tds, metronidazole 500 mg tds and oral fucidin 500 mg tds as she had recently had an MRSA infection. The ankle initially appeared to settle, but after 3 days she developed severe pain in the left foot and ankle at rest, with a fever of 39°C and rigors. She went to theatre and an abscess communicating with the subtalar joint was drained. A swab showed pus cells but no growth. She healed in 4 months, but came back to the foot clinic again with a severely infected toe on her other foot after 2 weeks.

Key points

- Patients with Charcot's osteoarthropathy can occasionally relapse with increased swelling and warmth of the involved foot
- It is difficult to distinguish between relapsed Charcot's osteoarthropathy and infection in an intact foot
- Clinical progress needs to be closely monitored with daily review for specific signs of infection.

Ischaemia and cellulitis

In the neuroischaemic foot it may be difficult to differentiate between the erythema of cellulitis and the redness of ischaemia. It is helpful to elevate the leg. The redness of ischaemia is usually cold and is most marked on dependency: it will disappear upon elevation of the limb, whereas cellulitis will remain. The erythema associated with inflammation is warm, although a very ischaemic foot may become deceptively warm when it is infected.

Erythema also occurs secondary to traumas, including insect stings, where it is associated with histamine release.

Investigations

Microbiological investigations

We believe that it is important to make a microbiological diagnosis and ascertain the organisms that are responsible for the infection. This involves either taking a deep ulcer swab or curettings, or tissue scrapings after debridement.

How to take a deep ulcer swab/curettings:

- The ulcer is debrided of surrounding callus and superficial slough
- The ulcer is washed out with sterile normal saline
- The base of the wound is then curetted (or scraped with a scalpel blade) and the curettings/scrapings sent for culture without delay. If curetting is not possible then a deep swab is taken from the base of the ulcer.

If the patient subsequently undergoes operative debridement then deep infected tissue rather than a swab should be sent to the laboratory.

Bone fragments removed by debridement should be sent for culture.

Blood cultures should also be taken if there is fever and systemic toxicity.

Laboratory investigations

Blood should be taken for:

- Full blood count
- Urea, creatinine and electrolytes
- Liver function tests
- C-reactive protein (CRP).

Although the white blood count is only raised in 50% of infections, we find that the CRP is nearly always elevated in diabetic foot infections and is a valuable guide to the severity of infection and a useful way of judging a positive response to treatment.

Radiological investigations

With all the above presentations of infection, it is important to X-ray the foot to detect

- Signs of osteomyelitis
- Gas in the deep tissues
- Radio-opaque foreign body.

In the initial stages of osteomyelitis, X-ray may be normal. Signs of osteomyelitis such as localized loss of bone density or cortical outline may not be apparent for at least 14 days.

Magnetic resonance imaging (MRI) may be useful to look for the presence of osteomyelitis and also to detect collections of fluid in the foot. Intravenous injection of gadolinium-containing contrast agent heightens the sensitivity of the diagnosis of these clinical features. The sequences employed in MRI for detection of soft tissue and bony abnormalities include T1, short tau inversion recovery (STIR) and postgadolinium with fat suppression.

T1 sequence

This shows pathology in specific anatomy. Bone marrow which is normal is bright, and abnormal marrow is dark.

STIR sequence

Normal marrow is dark. Abnormal marrow is bright. Fluid collections become bright.

Postgadolinium with fat suppression sequence

Gadolinium concentrates in areas of inflammation and results in a hyperintense signal on T1 images. As fat is hyperintense on T1 sequence, images are acquired using a fat suppression technique. Normal marrow in the foot is predominantly composed of fat. Thus it is hypointense on the fat-suppressed images. Any bright or high signal after injection of gadolinium with fat suppression technique applied represents a focus of inflammation.

The main MRI finding in osteomyelitis is an abnormal marrow signal which is dark on T1 images and bright on STIR images. After injection of gadolinium the abnormal marrow enhances as shown by a bright focus on the fat suppressed T1 images. Further signs of osteomyelitis include cortical disruption and periosteal reaction. An MRI which is negative excludes osteomyelitis.

An abscess presents as a focal low T1 signal but a high STIR lesion. Following the injection of gadolinium on fat suppressed T1 images there is a low signal in the centre of the abscess.

However, MRI has notable limitations. MRI of the septic diabetic foot can show a number of false positive diagnoses. An abnormal marrow signal can be seen with Charcot's osteoarthropathy and fractures. The acute phase of Charcot's osteoarthropathy may show the same enhancing marrow oedema that is impossible to separate from osteomyelitis.

CASE STUDY

Diabetic foot infection and MRI

A 62-year-old patient with type 2 diabetes of 3 years' duration developed an ulcer of the right 1st toe which had been present for 6 months when first seen in the foot clinic. He then had cellulitis and a markedly swollen 1st

Fig. 5.14 (a) T1 sequence shows reduced signal in 1st metatarsal head compared with other metatarsal heads. (b) Increased uptake on STIR sequence in 1st metatarsal head compared with other metatarsal heads. (Courtesy of Dr David Elias.)

toe. X-ray was normal. Deep wound swab revealed *Streptococcus* group B. He was treated with amoxicillin 500 mg tds and gentamicin 5 mg/kg daily, both intravenously. The temperature resolved but the cellulitis was slow to improve. Because of the persistent swelling of the right 1st ray, the patient underwent an MRI to assess the presence of osteomyelitis and a possible collection of fluid. The images were T1, and STIR and T1 postgadolinium with fat suppression to assess presence of fluid collections. There was oedema within the marrow of the head of the 1st metatarsal. There was cortical loss on its volar aspect and enhancement of the marrow in this region (Fig. 5.14a,b). This indicated osteomyelitis of the metatarsal head.

There was a small collection of fluid between the extensor hallucis longus tendon and the metatarsophalangeal joint/proximal phalanx of the big toe (Fig. 5.15a–c). The decision to debride the foot and carry out a 1st ray amputation was difficult but in view of the resolution of his fever and the improvement in the soft tissues clinically, intravenous antibiotic therapy was continued and the patient's ulcer eventually healed without surgery (Fig. 5.15d).

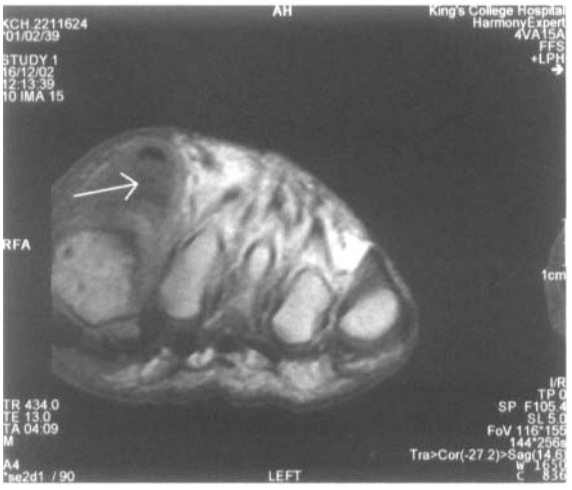
Key points

- It is difficult to diagnose osteomyelitis in the cellulitic foot as the X-ray is often initially normal
- MRI can indicate signs of marrow oedema which suggest osteomyelitis
- Group B *Streptococcus* is an important pathogen in the diabetic foot. It may need aggressive treatment with high-dose penicillin therapy such as amoxicillin 1 g tds. In severe cases, gentamicin 5 mg/kg daily can be added for synergy
- Osteomyelitis may be managed conservatively, especially when the soft tissue sepsis is responding to intravenous antibiotic therapy.

CASE STUDY

Extensive deep soft tissue infection with abscess revealed by MRI

A 65-year-old man with type 2 diabetes for 10 years tripped and fell on the pavement. He had no pain at the time but he noticed swelling the next day and was sent for an X-ray by his general practitioner. The X-ray revealed that he had fractured the necks of his left 2nd, 3rd and 4th metatarsals and he was treated with a below-knee walking plaster. He sustained ulceration on the plantar surface of the foot over the 2nd and 3rd metatarsal heads and the cast was removed. He was referred to the diabetic foot clinic. The left foot was swollen and cellulitic and he had rigors. Clinically there was no obvious abscess, but he had a fever. A deep wound swab grew *Staphylococcus aureus.* He was treated with intravenous amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. His left foot remained oedematous with pus discharging from the plantar lesion. He became increasingly unwell and went into renal failure. An MRI then showed an inflammatory mass with communication through to the plantar surface of the foot at metatarsophalangeal joint level. On the dorsal aspect of the foot a fluid collection was seen suggesting an abscess (Fig. 5.16a–c).



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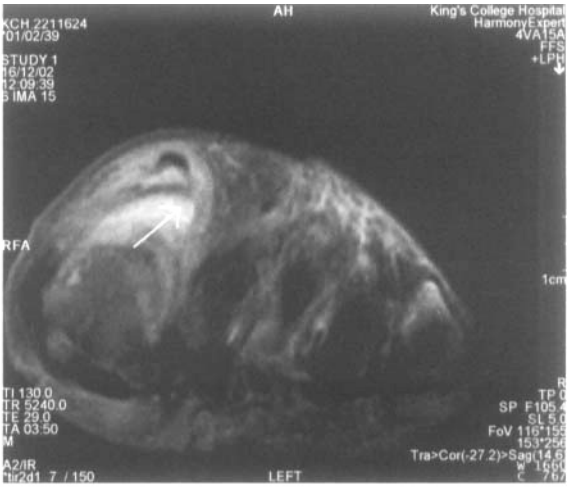

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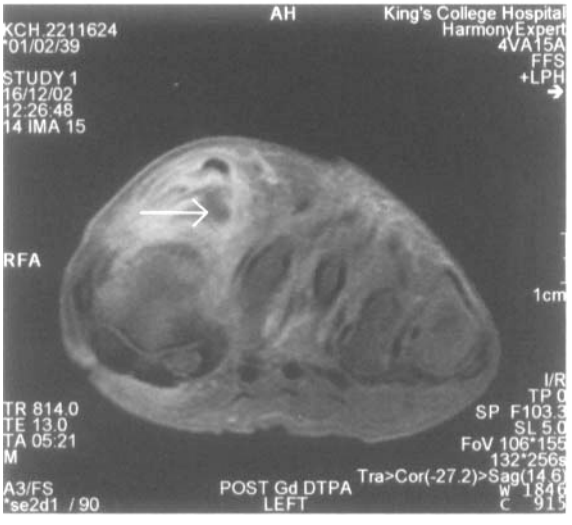

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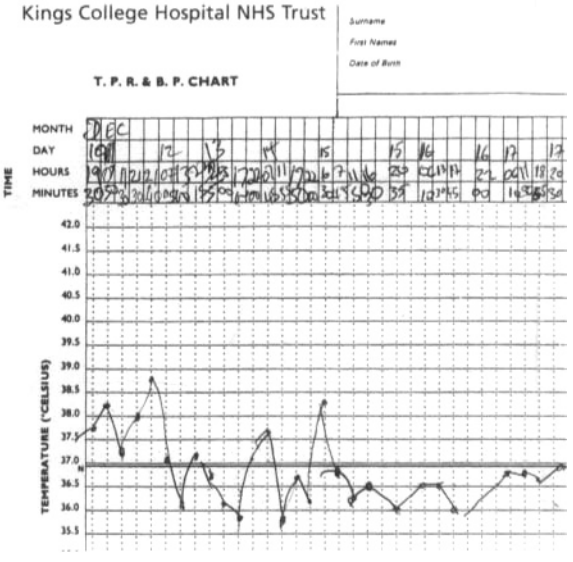


(d)

Fig. 5.15 (a) MRI T1 sequence shows normal uptake in soft tissues under extensor hallucis longus tendon. (b) Increased uptake on STIR sequence. (c) A small collection of fluid under the extensor hallucis longus tendon (post gadolinium). (d) Temperature chart showing resolution of fever.

He underwent surgical debridement and 20 mL of pus was drained from the foot. Postoperatively he needed haemodialysis but the swelling of the foot gradually improved, and the ulcer healed. His renal function improved and he came off dialysis.

Key points

- Deep infection with soft tissue involvement can be associated with severe systemic upset and in some cases with renal failure
- The decision to carry out surgical debridement of a diabetic foot is a difficult one. It is often made on clinical grounds alone. The main indications are an abscess or extensive sloughing of subcutaneous tissue
- An MRI may be helpful in locating fluid collections.

MANAGEMENT OF INFECTION

Infection in the diabetic foot needs full multidisciplinary treatment. It is vital to achieve:

- Microbiological control
- Wound control

Fig. 5.16 (a) T1 sequence shows inflammatory mass but normal uptake in dorsum of foot. (b) High uptake in the dorsum on STIR sequence. (c) Collection of fluid on the dorsum (post gadolinium). (With thanks to Dr David Elias and Dr Huw Walters.)

- Vascular control
- Mechanical control
- Metabolic control
- Educational control.

Microbiological control

The aim of this section is to enable clinicians to make rapid and effective decisions regarding the management of infection in the diabetic neuropathic and neuroischaemic foot, and to control it rapidly.

The key questions that need to be answered for each individual patient with one of the presentations above are as follows:

- Which antibiotics should be prescribed?
- Will antibiotics alone control the infection or will surgery also be necessary?

Principles of antibiotic treatment

- The microbiology of the diabetic foot is unique. Infection can be caused by Gram-positive aerobic, and Gram-negative aerobic and anaerobic bacteria, singly or in combination (Table 5.1)
- As there may be a poor immune response of the diabetic patient, even bacteria normally regarded as skin commensals may cause severe tissue damage. This includes Gram-negative organisms such as *Citrobacter*, *Serratia*, *Pseudomonas* and *Acinetobacter*. When Gram-negative

Table 5.1 Bacteria isolated from the diabetic foot

Gram positive	**Gram negative**
Staphylococcus aureus	*Klebsiella*
Streptococcus	*Escherichia coli*
Enterococcus	*Enterobacter*
	Pseudomonas
Anaerobes	*Citrobacter*
Bacteroides	*Morganella morganii*
Clostridium	*Serratia*
Peptostreptococcus	*Acinetobacter*
Peptococcus	*Proteus*

bacteria are isolated from a deep ulcer swab or curettings they should not, therefore, be regarded as automatically insignificant

- When a positive culture is found, it is then possible to focus antibiotic therapy according to sensitivities of the bacteria cultured (Table 5.2)
- However, at initial presentation we believe that it is important to prescribe a wide spectrum of antibiotics for three reasons:
  (a) it is impossible to predict the number and type of organisms from the clinical presentation
  (b) there is no way of predicting who will develop a rapidly ascending infection which becomes limb-threatening and even life-threatening
  (c) diabetic patients are immunosuppressed. The neuropathy and ischaemia of the diabetic foot reduces the local resistance to invading bacteria. As Louis Pasteur said: 'The germ is nothing. It is the terrain in which it grows that is everything'.

Duration of antibiotic therapy will depend on the clinical progress of the foot and ulcer, tissues involved, severity of the initial infection and also on individual factors relating to the patient.

The route by which therapy is given will depend on the severity of the infection. Intravenous therapy is required for serious infections. Intestinal absorption is unreliable in these circumstances.

Staphylococcus aureus

This is the commonest pathogen in the diabetic foot. Flucloxacillin is the ideal treatment. Clindamycin can also be used but beware of antibiotic-induced colitis especially in the elderly and postoperative patients. Erythromycin may increase the risk of myositis from statin therapy. When taking erythromycin, patients should be advised to stop their statin therapy temporarily.

Rifampicin and fucidin are also good antistaphylococcal agents, but they should not be given alone as resistance will develop rapidly. They should each be accompanied by a further antistaphylococcal agent.

Methicillin-resistant Staphylococcus aureus (MRSA)

MRSA can cause serious infections and in these circumstances give vancomycin 1 g IV bd, dosage to be adjusted according to serum levels, or teicoplanin 400 mg IV 12-hourly for three doses and then 400 mg daily. These antibiotics may need to be accompanied by either sodium fusidate 500 mg tds or rifampicin 300 mg tds orally (according to sensitivities).

If MRSA is isolated with moderate signs of infection, oral therapy can be given with two of the following: sodium fusidate 500 mg tds, rifampicin 300 mg tds, trimethoprim 200 mg bd or doxycycline 100 mg daily, according to sensitivities.

Streptococcus group A, B, C, E, F and G

Streptococcus group B is the commonest and can cause severe infection although A, C, E, F and G can infect the foot.

The streptococci milleri group of organisms related to *Streptococcus* group F can cause abcesses in the foot.

Streptococci can be treated with amoxicillin. Clindamycin, rifampicin and erythromycin are also active against streptococci.

Enterococcus

Enterococcus faecalis is rarely pathogenic. It may be selected out by cephalosporin treatment. If it is causing definite infection then treat with amoxicillin. *Enterococcus faecium* may need vancomycin.

Anaerobes

These are commonly found in deep infections but anaerobes are also a feature of many chronic wounds even when they are superficial.

Metronidazole is the treatment of choice. Clindamycin and augmentin (amoxicillin/clavulanic acid) also have antianaerobic activity. Meropenem intravenously is also active against anaerobes.

Gram-negative organisms

Klebsiella, *Escherichia coli*, *Proteus*, *Enterobacter*, *Citrobacter*, *Serratia*, *Pseudomonas* and *Acinetobacter* and other Gram-negative bacteria can be definitely pathogenic in the diabetic foot especially when they are in a pure growth or as part of a polymicrobial deep infection.

Table 5.2 Antibiotics for treating the infected foot

	Antibiotic treatment	
Microorganism	Oral	IV
Staphylococcus aureus	Flucloxacillin 500 mg qds Sodium fusidate 500 mg tds Clindamycin 300 mg tds Rifampicin 300 mg tds	Flucloxacillin 500 mg qds Gentamicin 5 mg/kg/day (according to levels) Clindamycin 150–600 mg qds
Methicillin-resistant *Staphylococcus aureus* (MRSA)	Sodium fusidate 500 mg tds Trimethoprim 200 mg bd Rifampicin 300 mg tds Doxycycline 100 mg daily Linezolid 600 mg bd	Vancomycin 1 g bd (according to levels) Teicoplanin 400 mg daily Linezolid 600 mg bd
Streptococcus	Amoxicillin 500 mg tds Flucloxacillin 500 mg qds Clindamycin 300 mg tds Erythromycin 500 mg qds	Amoxicillin 500 mg tds Clindamycin 150–600 mg qds
Enterococcus	Amoxicillin 500 mg tds	Amoxicillin 500 mg tds Vancomycin 1 g bd (according to levels)
Anaerobes	Metronidazole 400 mg tds Clindamycin 300 mg tds	Metronidazole 500 mg tds Clindamycin 150–600 mg qds
Coliforms (*E. coli*, *Proteus*, *Klebsiella*, *Enterobacter*)	Ciprofloxacin 500 mg bd Cefadroxil 1 g bd Trimethoprim 200 mg bd	Ciprofloxacin 200 mg bd Ceftazidime 1–2 g tds Ceftriaxone 1–2 g daily Gentamicin 5 mg/kg/day (according to levels) Piperacillin–tazobactam 4.5 g tds Meropenem 500 mg to 1 g tds Ticarcillin/clavulanate 3.2 g tds
Pseudomonas	Ciprofloxacin 500 mg bd	Ceftazidime 1–2 g tds Gentamicin 5 mg/kg/day (according to levels) Piperacillin–tazobactam 4.5 g tds Meropenem 500 mg to 1 g tds Ticarcillin/clavulanate 3.2 g tds

Intramuscular antibiotics.
Ceftriaxone 1 g daily IM to treat Gram-positives and Gram-negatives.
Teicoplanin 400 mg daily IM to treat Gram-positives including MRSA.
Teicoplanin initially should be given as 400 mg 12 hourly for 3 doses, as a loading regimen.
Imipenem w. cilastatin 500 mg bd IM to treat Gram-positives, Gram-negatives and anaerobes.
First dose of doxycycline should be 200 mg.
bd, twice daily; tds, three times daily; qds, four times daily.

Oral agents are ciprofloxacin and trimethoprim. Parenteral agents include ceftazidime, aminoglycosides, meropenem and piperacillin/tazobactam, and ticarcillin/clavulanate.

Pseudomonas

Pseudomonas can be responsible for definite tissue damage.

It may be sensitive to ciprofloxacin as an oral agent. Otherwise parenteral therapy is necessary and includes

ceftazidime, aminoglycosides, meropenem, piperacillin/tazobactam, and ticarcillin/clavulanate.

Antibiotic adjustments for patients in renal failure

(Table 5.3)

Renal impairment is common in diabetic foot patients, and commonly used drugs can give rise to problems in diabetic patients with reduced renal function for the following reasons:

- Failure to excrete a drug or its metabolites may lead to toxicity
- Sensitivity to some drugs is increased even if elimination is unimpaired
- Many side-effects are poorly tolerated by people in end-stage renal failure
- Some drugs cease to be effective when renal function is impaired.

Many of these problems can be avoided by reducing the dose or by using alternative drugs.

The level of renal function below which a dose must be reduced depends on whether the drug is eliminated entirely by renal excretion or is partly metabolized, and on how toxic it is.

For many drugs with only minor or no dose-related side-effects, very precise modification of the dose regimen is unnecessary. If even minor renal impairment is considered likely on clinical grounds, renal function should be checked before prescribing antibiotics.

Indications for surgery

Antibiotics alone may be unable to control infection and it is necessary to decide whether adjunctive surgery is necessary. In severe episodes of cellulitis, the ulcer may be complicated by extensive infected subcutaneous soft tissue. At this point, the tissue is not frankly necrotic but has started to break down and liquefy. It is best for this tissue to be removed operatively. The definite indications for urgent surgical intervention are:

- A large area of infected sloughy tissue
- Localized fluctuance and expression of pus
- Crepitus with gas in the soft tissues on X-ray
- Blue or purplish discolouration of the skin.

Management of the five presentations of infection

Treatment is discussed for the five presentations of infection, in neuropathic feet and in neuroischaemic feet, both as initial treatment and follow-up.

Infection in the neuroischaemic foot is often more serious than in the neuropathic foot which has a good arterial blood supply. We regard a positive ulcer swab in a neuroischaemic foot as having serious implications, and this influences antibiotic policy.

Ulcer with local signs of infection

Neuropathic feet

We give amoxicillin 500 mg tds, flucloxacillin 500 mg qds and metronidazole 400 mg tds because streptococci, staphylococci and anaerobes are the most likely organisms. We believe that anaerobes are a common feature of superficial as well as deep infections, but may not always be isolated because of restriction on the length of time of incubation of cultures. We avoid the use of clindamycin in local infections because it has serious side-effects, the most alarming toxic effect being antibiotic-associated colitis which may be fatal. Although this can occur with most antibacterials it is more frequently seen with clindamycin.

When the ulcer extends to fascia or tendon we add either trimethoprim 200 mg bd or ciprofloxacin 500 mg bd to cover Gram negatives. However, strictly, the infection is now classified as a deep infection (see below). We do not routinely use augmentin as the risk of acute liver toxicity is six times greater with augmentin than with amoxicillin.

If the patient is allergic to penicillin, we substitute erythromycin 500 mg qds for amoxicillin.

We send a deep swab or curettings for culture. It is important to know the organisms that are causing the infection so that antibiotics can be used accurately to target the causative organisms. It is said by some that it is not important to know the organisms as there will usually be a good response to antibiotic therapy. However, if there is not a good response and the patient deteriorates and no swab has been taken, it is then difficult to be accurate in antibiotic therapy. Meanwhile, time has been lost while the patient continues to deteriorate.

Follow-up plan. If no signs of infection and no organisms are isolated, we stop antibiotics.

If no signs of infection are present but organisms are isolated, we focus antibiotics and review the patient in 1 week.

If signs of infection are present but no organisms are isolated, we continue antibiotics as above.

If signs of infection are present and organisms are isolated, we focus antibiotics according to sensitivities.

If MRSA is grown, but there are no signs of infection we use topical mupirocin 2% ointment if sensitive. Patients should undergo an MRSA eradication protocol to remove it from carrier sites (Table 5.4). If MRSA is isolated with signs of infection, oral therapy with two of the following should be given: sodium fusidate 500 mg tds, rifampicin

Table 5.3 Antibiotic dosage in renal failure. (HD, haemodialysis; CAPD, continuous ambulatory peritoneal dialysis; CAVH, continuous arteriovenous haemofiltration; Cr, creatinine.)

Antibiotic	Dose for normal renal function	Mild impairment (serum Cr 120–200 μmol/L)	Moderate impairment (serum Cr 200–400 μmol/L)	Severe impairment (serum Cr > 400 μmol/L)
Amikacin	iv 7.5 mg/kg/bd	Give 7.5 mg/kg daily redose < 5 mg/L	Give 7.5 mg/kg daily redose < 5 mg/L	Give 7.5 mg/kg daily redose < 5 mg/L
Amoxicillin	iv/po 500 mg tds	No change	No change	500 mg bd
Benzylpenicillin	iv 1.2 g qds	No change	75% of normal dose	Maximum 3.6 g daily
Cefadroxil	po 0.5–1 g bd	0.5–1 g bd	0.5–1 g od	0.5–1 g od
Ceftazidime	iv 1–2 g tds	1 g 12 hrly	0.5–1 g 24 hrly	0.5–1 g 48 hrly
Ceftriaxone	iv 1–4 g daily	No adjustment	No adjustment	1–2 g od
Cefuroxime	iv 750 mg to 1.5 g tds	No adjustment	750 mg to 1.5 g bd	750 mg bd
Ciprofloxacin	iv 100–400 mg bd po 250–750 mg bd	No adjustment po max 500 mg bd	iv 100–200 mg bd po max 500 mg bd	iv 100–200 mg bd po max 500 mg bd
Clindamycin	iv 150–600 mg qds po 150–450 mg qds	No adjustment No adjustment	No adjustment No adjustment	No adjustment No adjustment
Doxycycline	po First day 200 mg then 100 mg daily	No adjustment	No adjustment	No adjustment
Erythromycin	iv 500 mg qds po 500 mg qds	No adjustment	No adjustment	iv 500 mg tds po 500 mg tds
Flucloxacillin	iv 500 mg qds po 500 mg qds	No adjustment No adjustment	No adjustment No adjustment	500 mg tds 500 mg tds
Gentamicin	iv 5 mg/kg once daily	1.5–2 mg/kg redose < 1 mg/L	1.5–2 mg/kg redose < 1 mg/L	1.5–2 mg/kg redose < 1 mg/L
Meropenem	iv 500 mg to 1 g tds	500 mg to 1 g 12 hrly	250–500 mg 12 hrly	250–500 mg 24 hrly
Metronidazole	iv 500 mg tds po 400 mg tds	No adjustment No adjustment	No adjustment No adjustment	No adjustment No adjustment
Rifampicin	po 600 mg bd	No adjustment	No adjustment	No adjustment
Sodium fusidate	iv 500 mg tds po 500 mg tds	No adjustment No adjustment	No adjustment No adjustment	No adjustment No adjustment
Tazocin	iv 2.25–4.5 g tds	2.25–4.5 g tds	2.25–4.5 g bd	2.25–4.5 g bd
Teicoplanin	iv load with 400 mg 12 hrly iv then 400 mg daily po 100–200 mg bd	Load first then 200 mg once daily 200 mg bd then half dose	Load first then 200 mg alt days normal for 3 days then half dose	Load first then 200 mg 3x/week Half normal dose
Trimethoprim				
Vancomycin	iv 1 g bd trough < 10 mg/L	Give 1 g redose when level < 10 mg/L	Give 1 g redose when level < 10 mg/L	Give 1 g redose when level < 10 mg/L

Antibiotic	HD	CAPD	CAVH
Amikacin	Give 7.5 mg/kg daily redose < 5 mg/L	Give 7.5 mg/kg daily redose < 5 mg/L	Give 7.5 mg/kg daily redose < 5 mg/L
Amoxicillin	500 mg bd	500 mg bd	500 mg tds
Benzylpenicillin	Maximum 3.6 g daily	Maximum 3.6 g daily	50% of normal dose
Cefadroxil	0.5–1 g od	0.5–1 g od	Not applicable
Ceftazidime	0.5–1 g 48 hrly	0.5–1 g 48 hrly	1–2 g od
Ceftriaxone	1–2 g od	1–2 g od	1–2 g od
Cefuroxime	750 mg bd	750 mg bd	750 mg bd
Ciprofloxacin	iv 100–200 mg bd po max 500 mg bd	iv 100–200 mg bd po max 500 mg bd	iv 200 mg bd po max 500 mg bd
Clindamycin	No adjustment	No adjustment	No adjustment
Doxycycline	No adjustment	No adjustment	No adjustment

Cont'd.

Table 5.3 *(cont'd)*

Antibiotic	HD	CAPD	CAVH
Erythromycin	iv 500 mg tds po 500 mg tds	iv 500 mg tds po 500 mg tds	iv 500 mg tds po 500 mg tds
Flucloxacillin	500 mg tds	500 mg tds	500 mg qds
Gentamicin	1.5–2 mg/kg redose < 1 mg/L	1.5–2 mg/kg redose < 1 mg/L	1.5–2 mg/kg redose < 1 mg/L
Meropenem	250–500 mg 24 hrly	250–500 mg 24 hrly	250–500 mg 12 hrly
Metronidazole	No adjustment	No adjustment	No adjustment
Rifampicin	No adjustment	No adjustment	No adjustment
Sodium fusidate	No adjustment	No adjustment	No adjustment
Tazocin	2.25–4.5 g bd	2.25–4.5 g bd	2.25–4.5 g tds
Teicoplanin	Load first then 200 mg 3×/week	Load first then 200 mg 3×/week	Load first then 200 mg alt. days
Trimethoprim	Half normal dose	Half normal dose	Half normal dose
Vancomycin	Give 1 g redose when level < 10 mg/L	Give 1 g redose when level < 10 mg/L	Give 1 g redose when level < 10 mg/L

HD, haemodialysis; CAPD, continuous ambulatory peritoneal dialysis; CAVH, continuous arteriovenous haemofiltration; Cr, creatinine.

Table 5.4 MRSA screening protocol

Barrier nurse
Topical protocol for 5 days
Mupirocin 2% nasal ointment every 8 h
Tricolsan 2% liquid soap for body and hair washing once daily
2 days protocol free
Repeat swabs 3 times at 48-h intervals
If MRSA negative, no further treatment necessary
If MRSA positive, re-treat with 5 days topical protocol and repeat swabs at 48-h intervals until 3 sets of negative swabs are obtained

300 mg tds, trimethoprim 200 mg bd or doxycycline 100 mg daily, according to sensitivities.

In this follow-up plan, the difficult decision is when there are no signs of infection present but organisms are isolated.

Some organisms may be commensals; however, if there are Gram-positive organisms or anaerobes or a pure growth of Gram-negative organisms we regard this as a significant result and microbiological evidence of infection.

Neuroischaemic feet

Regardless of the depth of the wound we aim to cover aerobic Gram-positive, aerobic Gram-negative and anaerobic infections, and thus we give amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 400 mg tds and trimethoprim 200 mg bd or ciprofloxacin 500 mg bd. If the patient is allergic to penicillin, we substitute erythromycin 500 mg qds for amoxicillin and flucloxacillin.

We use this therapy for both superficial and deep infections because Gram-negative organisms may be a feature of infections in the ischaemic foot whether the ulcer is superficial or deep.

We send deep swabs or curettings (where possible) for the reasons already described above.

Follow-up plan. If there are no signs of infection and no organisms are grown, we consider stopping antibiotics. However, if the patient is severely ischaemic, with pressure index below 0.5, we continue antibiotics until the ulcer is healed.

If there are no signs of infection but organisms are present, we focus the antibiotics according to sensitivities.

If signs of infection are present but no organisms are grown, we continue to give broad-spectrum antibiotics as above.

If signs of infection are still present and organisms are grown, we focus the antibiotics according to sensitivities.

If MRSA is isolated with signs of infection, oral therapy with two of the following is given: sodium fusidate

500 mg tds, rifampicin 300 mg tds, trimethoprim 200 mg bd or doxycycline 100 mg daily, according to sensitivities.

Ulcer with surrounding erythema

The antibiotic management and follow-up plan is the same as for the ulcer with local infection. The erythema should be traced to allow early recognition of extension of erythema.

Ulcer with diffuse spreading erythema

Neuropathic feet and neuroischaemic feet

These patients need parenteral therapy. Ideally these patients are admitted to hospital and given intravenous antibiotic therapy as follows: amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. For patients who are allergic to penicillin we substitute erythromycin 500 mg qds for amoxicillin and flucloxacillin. We use this regime because these infections are often polymicrobial.

If intravenous therapy is not possible then a useful therapy is intramuscular ceftriaxone 1 g in 3.5 mL of 1% lignocaine (lidocaine) once daily together with metronidazole 1 g rectally tds. Ceftriaxone is a useful wide-spectrum antibiotic with a prolonged period of activity making it suitable for once a day administration either intramuscularly or intravenously. This regime is suitable for neuropathic and neuroischaemic patients, but anticoagulant therapy is a contraindication to intramuscular injections.

Other parenteral therapies which are effective include:

- Ciprofloxacin and clindamycin
- Ticarcillin/clavulanate
- Piperacillin/tazobactam (see Table 5.2).

Follow-up plan

The infected foot should be inspected daily to gauge the initial response to antibiotic therapy.

Appropriate antibiotics should be selected when sensitivities are available.

- If no organisms are isolated and yet the foot remains severely cellulitic, then a repeat deep swab or tissue should be taken but the quadruple antibiotic therapy as above should be continued
- If MRSA is isolated, give vancomycin 1 g IV bd, dosage to be adjusted according to serum levels, or teicoplanin 400 mg IV 12-hourly for three doses and then 400 mg daily. These antibiotics may need to be accompanied by either sodium fusidate 500 mg tds or rifampicin 300 mg tds orally (according to sensitivities)
- Intravenous antibiotic therapy can be changed to the appropriate oral therapy when the signs of cellulitis have resolved
- Patients should be followed up weekly in the diabetic foot clinic and antibiotic therapy adjusted as described above.

CASE STUDY

Ulcer with diffuse spreading erythema in neuropathic foot

A 44-year-old man with type 1 diabetes of 29 years' duration and severe peripheral neuropathy who was not previously known to the diabetic foot clinic was admitted to hospital with an infected left 3rd toe and extensive oedema and diffuse cellulitis extending up the leg. His pedal pulses were bounding. He had peripheral neuropathy. He had a mild fever 37.5°C. The 3rd toe was sloughy with two ulcers which probed to bone (Fig. 5.17a). X-ray was normal. Day 1 C-reactive protein (CRP) was 106 mg/L. Initially he was given amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 400 mg tds and ceftazidime 1 g tds intravenously. By day 3 toe discolouration was marked (Fig. 5.17b). On day 3 the CRP was still raised at 105 mg/L. A swab grew *Streptococcus* group B and mixed anaerobes. His antibiotic therapy was changed to gentamicin 5 mg/kg daily with amoxicillin 1 g tds and metronidazole 500 mg tds intravenously. By day 5 he showed improvement and his CRP had fallen to 60 mg/L. By day 7 there was further improvement with resolution of bluish discolouration and the CRP was 36 mg/L (Fig. 5.17c). X-ray showed that there was destruction of the proximal interphalangeal joint of the 3rd toe and of the periarticular phalanges (Fig. 5.17d). However, clinically he continued to improve and eventually healed after 16 weeks (Fig. 5.17e). After the toe healed, he failed to attend regular follow-up appointments, but did attend as an emergency when he spilled boiling water on his feet. The resulting burns were superficial and healed quickly, after which he was lost to follow-up again.

Key points

- Serial CRP measurements are usually a useful indicator of progress of diabetic foot infections
- Serious infections often involve more than one organism
- The initial microbiological sample which is either curettings or a deep wound swab is extremely important in the management of diabetic foot infections

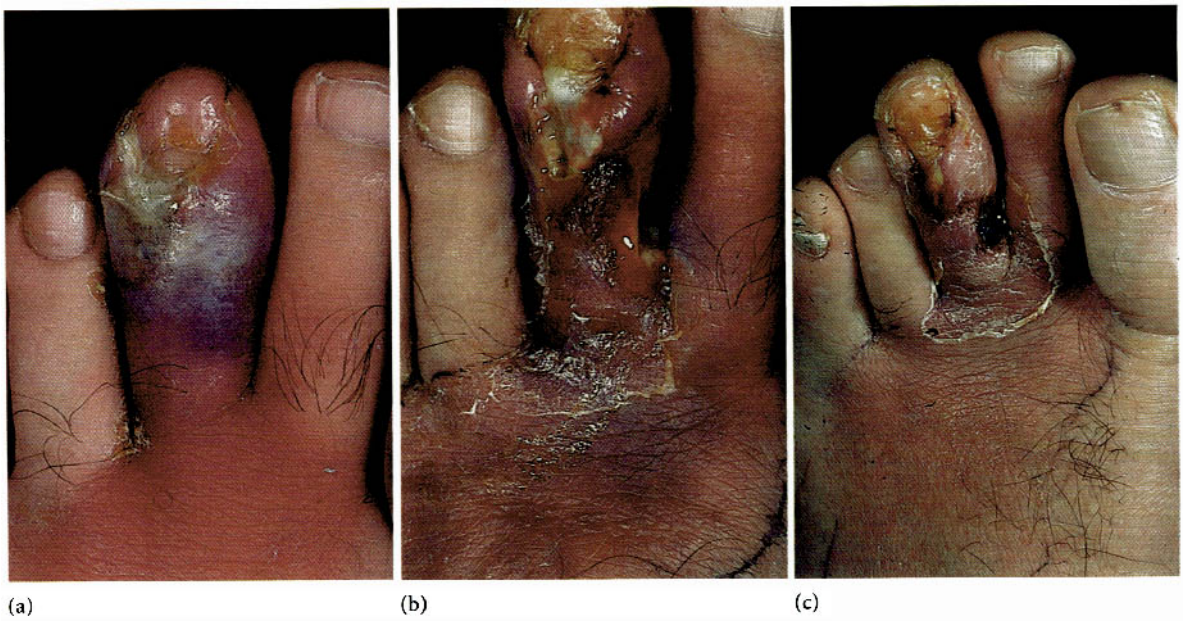


Fig. 5.17 (a) On day 1 the patient's CRP was 106 mg/L. The toe was infected and there was severe cellulitis. (b) On day 3 the CRP was still raised at 105 mg/L and the discolouration of the toe was present. The cellulitis had not resolved. (c) Cellulitis is resolving, bluish discolouration is fading, CRP is 36 mg/L. We noted scaling and desquamation of the previously intensely cellulitic area. (d) Destruction of proximal interphalangeal joint of 3rd toe. (e) The foot healed in 16 weeks.

- *Streptococcus* group B is an important pathogen of the diabetic foot
- Antibiotic therapy needs to be focused towards the organisms isolated on initial culture
- Bone and joint destruction may be treated conservatively with antibiotics as long as soft tissue infection is resolving.

CASE STUDY

Care of a neuroischaemic ulcer with diffuse spreading erythema

A 79-year-old man with type 2 diabetes of 5 years' duration with hypertension and steroid-treated polymyalgia

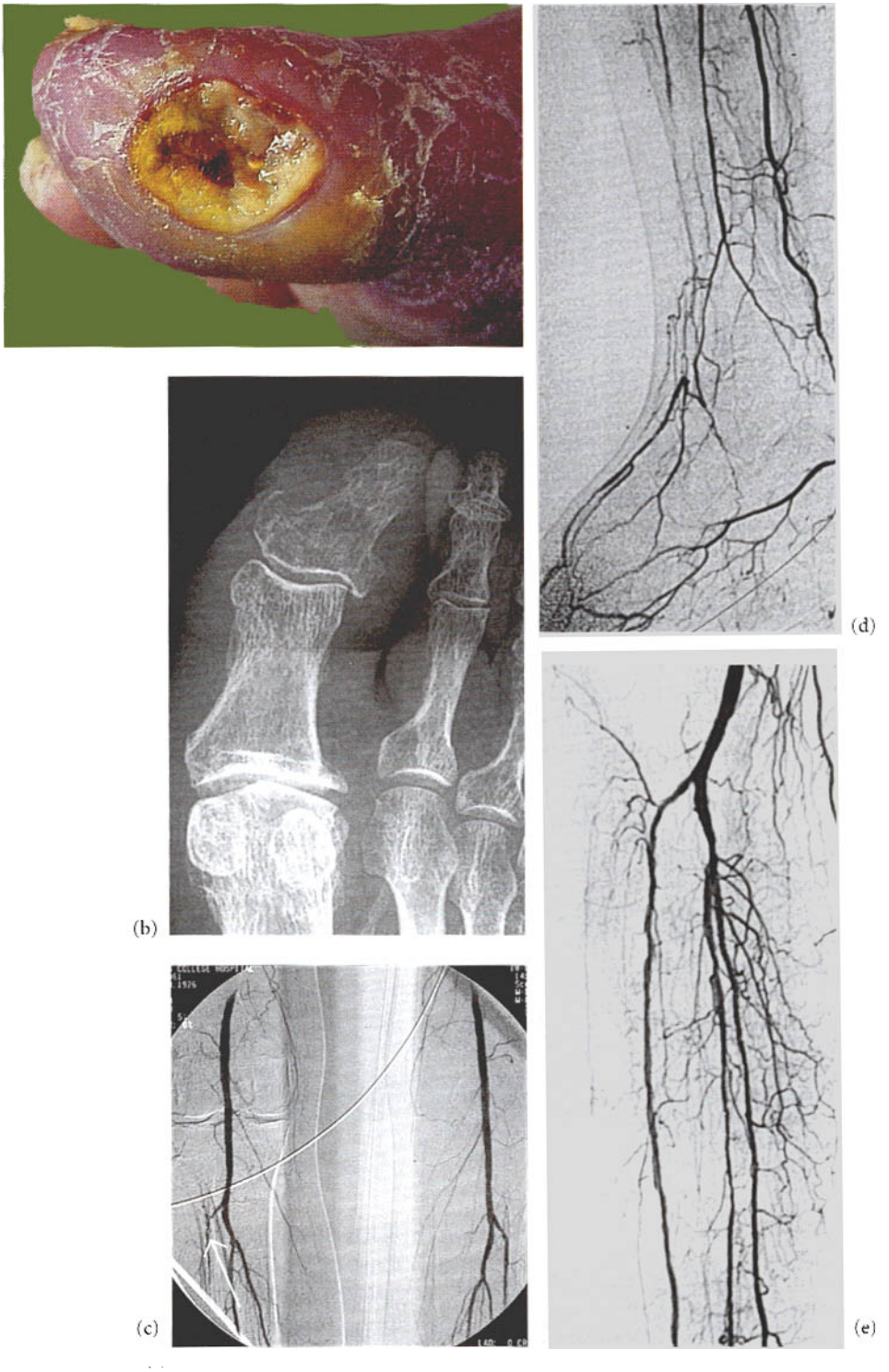


Fig. 5.18 (a) Ulcer with cellulitis on toe spreading to the foot. (b) X-ray was initially normal. (c) Stenosis at origin of anterior tibial artery. (d) Occlusions of anterior tibial artery. (e) Angioplasty of anterior tibial artery.

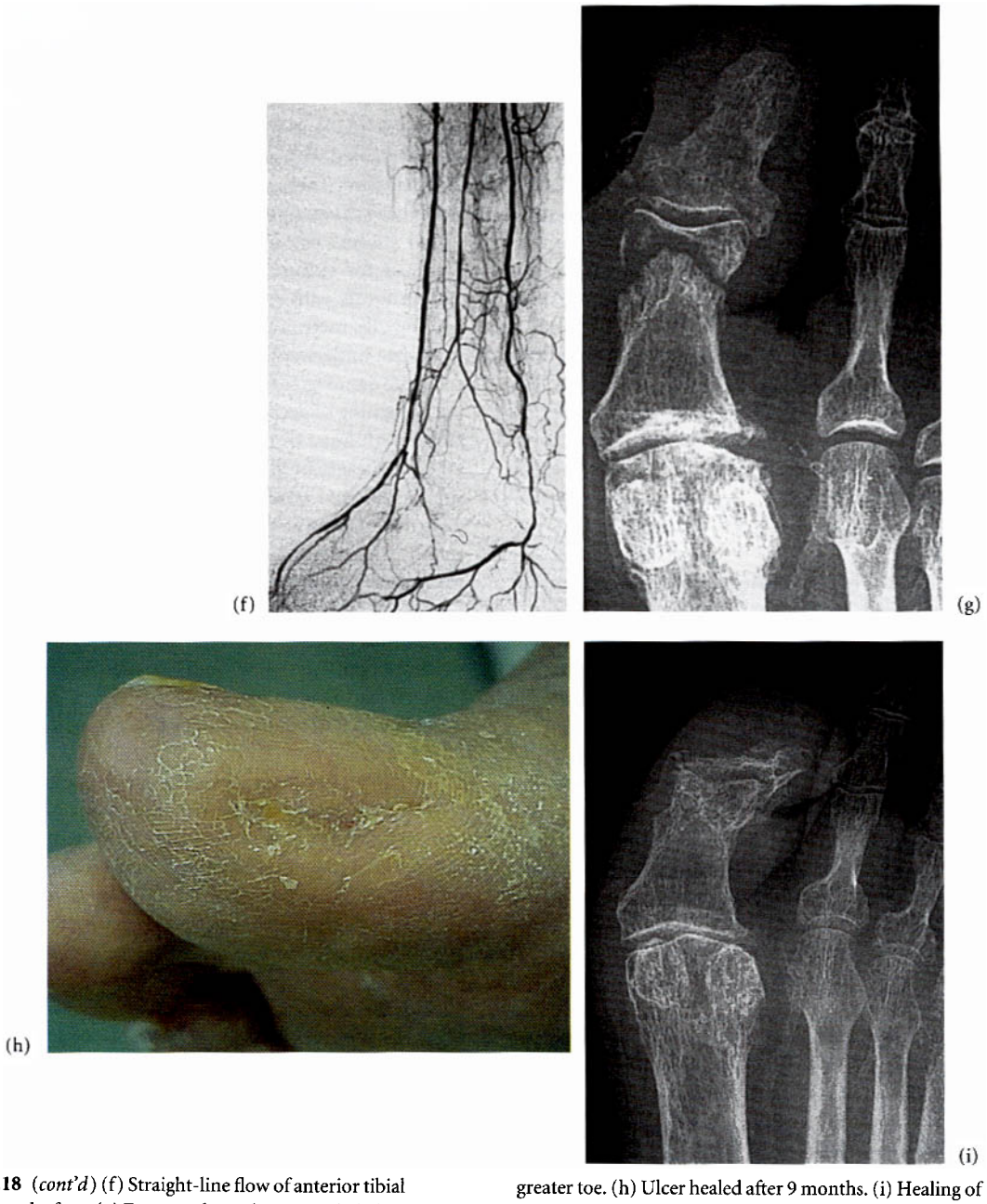


Fig. 5.18 (*cont'd*) (f) Straight-line flow of anterior tibial artery to the foot. (g) Fracture through proximal phalanx of greater toe. (h) Ulcer healed after 9 months. (i) Healing of fracture.

rheumatica was referred from his local hospital with neuro-ischaemic ulceration of his right hallux and diffuse spreading cellulitis involving the forefoot (Fig. 5.18a). The ulcer had been present for 3 weeks, had first appeared to be a blood blister and had a moist sloughy base. A probe inserted into the ulcer touched bone. X-ray was initially normal (Fig. 5.18b). He was treated with amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. Tissue was sent for culture and grew MRSA and mixed anaerobes. Antibiotic

therapy was changed to vancomycin 1 g bd and metronidazole was continued. Pedal pulses were impalpable and transcutaneous oxygen tension was 31 mmHg. He underwent digital subtraction angiography which showed stenosis and occlusions of the anterior tibial artery (Fig. 5.18c,d). He underwent angioplasty, dilating stenoses and occlusions along the anterior tibial artery on to the dorsalis pedis artery (Fig. 5.18e,f) following which his transcutaneous oxygen tension rose from 31 to 47 mmHg. Repeat X-ray showed fracture through the proximal phalanx of the greater toe (Fig. 5.18g). But as the ulcer was continuing to improve and soft tissue infection was resolving he continued on conservative therapy with Hyaff applied to the ulcer and a 'Scotchcast' boot. The ulcer healed after 9 months (Fig. 5.18h) and there was reasonable resolution of the bony changes to the proximal phalanx although there was resorption of bone of the distal phalanx (Fig. 5.18i).

Key points

- Angioplasty can be a valuable adjunct to treatment for infection in the neuroischaemic foot
- Even if the improved blood supply is temporary and the artery restenoses after a few weeks or months, the management of infection will have benefited from the increased perfusion
- Initial culture was crucial in this case indicating MRSA which necessitated a change in antibiotic treatment
- Osteomyelitis can sometimes be treated conservatively.

Ulcer with extensive deep soft tissue infection

Neuropathic feet and neuroischaemic feet. The antibiotic treatment is the same as for extensive cellulitis, except that intramuscular therapy with ceftriaxone is probably not sufficient therapy. Every attempt should be made to give intravenous therapy.

If the patient shows signs of systemic sepsis such as systolic pressure < 100 mmHg or tachycardia > 125/min, then it may be advisable to give also a *stat.* dose of gentamicin 5 mg/kg/day. It has a wide spectrum of action against Gram-positive and Gram-negative organisms and it has a significant postantibiotic effect for 24 h.

However, patients in this group may well need surgical debridement as well as intravenous antibiotic therapy.

The follow-up plan is the same as described for extensive diffuse erythema or cellulitis.

Clinical and microbiological response rates have been similar in trials of various antibiotics and no single agent or combination of agents has emerged as most effective.

CASE STUDY

Neuropathic ulcer with extensive sloughing of subcutaneous tissue

A 68-year-old man with type 2 diabetes of 15 years' duration presented with a swollen left foot which was brawny and cellulitic. There was a deep ulcer over the 4th metatarsal head discharging pus (Fig. 5.19a). It had started as a blister 4 weeks previously. Pulses were bounding. Tissue was sent for culture and he was admitted and treated intravenously with amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds. He underwent operative surgical debridement on the same day (Fig. 5.19b). There was extensive subcutaneous sloughing of deep tissue down to bone in the forefoot. Culture of tissue from the diabetic foot clinic and tissue taken at surgery both grew *Staphylococcus aureus*, *Proteus* spp. and mixed anaerobes. The initial antibiotic regime was continued to eradicate the above organisms until the cellulitis had settled. He made a good recovery and the wound healed within 10 weeks (Fig. 5.19c).

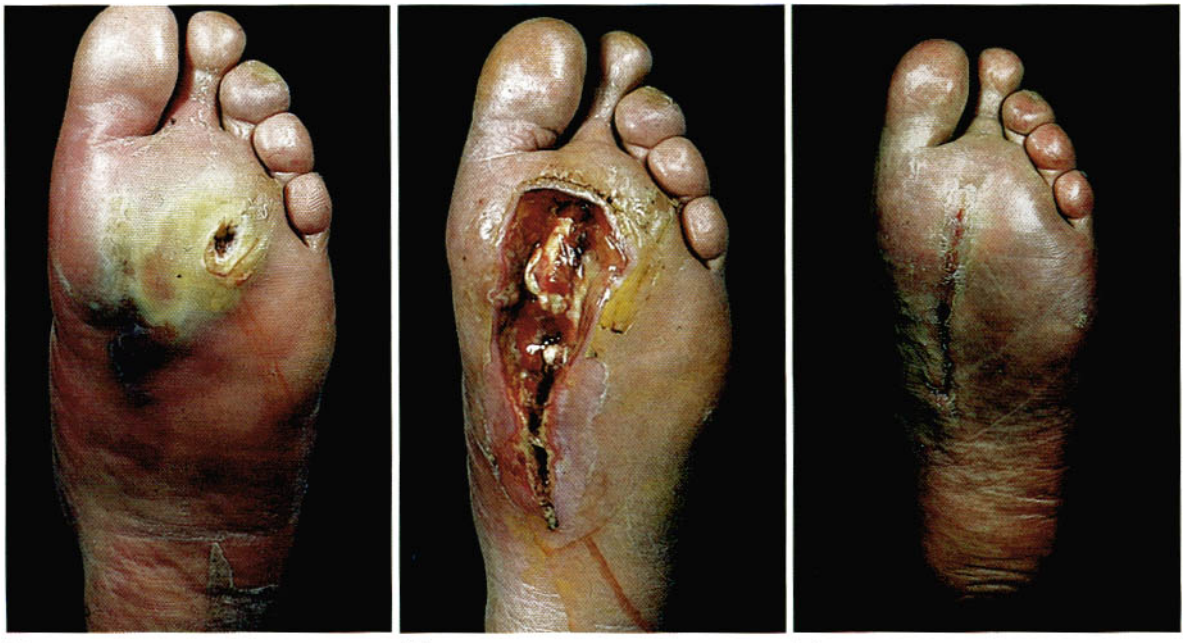
Key points

- The patient presented with an extensive deep lesion which had simply started as a blister 4 weeks previously and had recently developed a rapidly extending infection
- The patient had been previously given flucloxacillin but the tissue grew *Proteus* and mixed anaerobes which would not have been sensitive to flucloxacillin
- This was a polymicrobial infection which needed combined antibiotic therapy
- There was extensive soft tissue destruction and the patient required urgent operative surgical debridement.

Ulcer with extensive erythema and with blue/purple/black discolouration of surrounding tissues

Neuropathic feet and neuroischaemic feet. The antibiotic therapy and follow-up plan is the same as above but surgery should be considered urgently. If the blue or purple discolouration is noted and treated promptly then it may resolve and the skin return to a normal colour. However, if intervention is late the blue discolouration indicating compromised oxygen supply to the skin will lead to necrosis and the foot moves to stage 5.

It is important to explore the possibility of revascularization in the infected neuroischaemic foot. Improvement of perfusion will not only help to control infection, but also promote healing of wounds if operative debridement is necessary.



(a) (b) (c)

Fig. 5.19 (a) Deep ulcer with subcutaneous sloughing visible. (b) Extent of debridement necessary to remove all necrotic tissue down to healthy bleeding tissue. (c) The wound is healed at 10 weeks.

CASE STUDY

Blue discolouration in a neuroischaemic foot

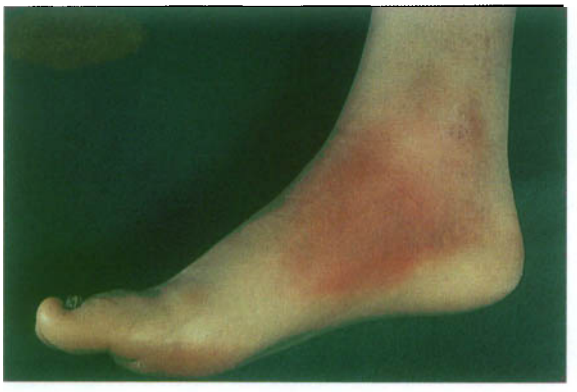
A 48-year-old man with type 1 diabetes mellitus of 20 years' duration, peripheral neuropathy, background retinopathy and no proteinuria presented with a 1-week history of malaise, high blood glucose and a 2-day history of discomfort and redness of the right foot. There was no history of trauma. There was an area of erythema over the dorsum and both medial and lateral aspects of the right foot, which was also oedematous. There was no break in the skin (Fig. 5.20a).

Body temperature was 39.2°C, pulse 104 regular, foot pulses absent, chest clear, abdomen normal. Haemoglobin 11.4 g/dL, WBC 17.3 10^9/L, erythrocyte sedimentation rate (ESR) 105, HbA_1 13% and glucose 18.6 mmol/L (335 mg/dL). X-ray of the foot was normal. Blood cultures showed no growth. There was no open lesion to take a swab.

Doppler studies in both legs showed a very high ankle/brachial pressure index (above 1.5) indicative of calcification. The foot artery waveforms were damped, indicating reduced blood flow. A clinical diagnosis of cellulitis in a neuroischaemic foot was made. The other much less likely diagnosis was an acute onset of Charcot's osteoarthropathy. However, it is unusual for Charcot's osteoarthropathy to be associated with such a high body temperature, which is much more suggestive of sepsis.

He was treated with quadruple intravenous antibiotic therapy (amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds) to provide a wide spectrum cover and commenced on sliding scale intravenous insulin. By the next day, body temperature had fallen to 37.2°C but there was no improvement in the cellulitis. There were spikes of fever on the evening of the second and third days (Fig. 5.20b). The area of cellulitis did not regress, but there was no evidence of a collection of pus. A surgical opinion was obtained and confirmed that there was no indication for surgery.

On the fourth day of the admission the patient was still pyrexial. He also had a rigor. The ceftazidime was withdrawn and intravenous gentamicin 80 mg tds started. (This was before once-daily dosaging had come in.) The most common organism isolated in diabetic foot infections is *Staphylococcus aureus* and gentamicin is active against *Staphylococcus* as well as providing Gram-negative cover.



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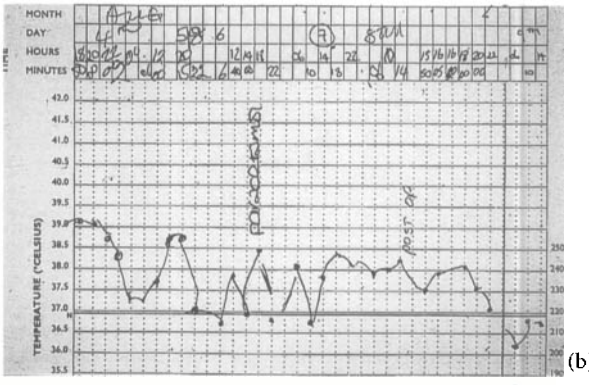

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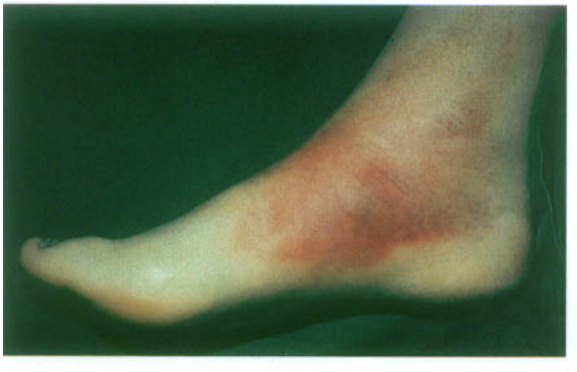

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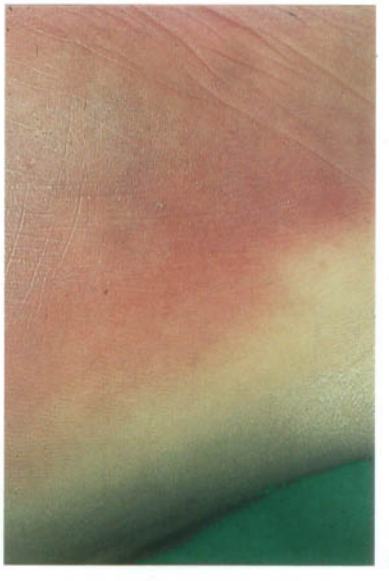

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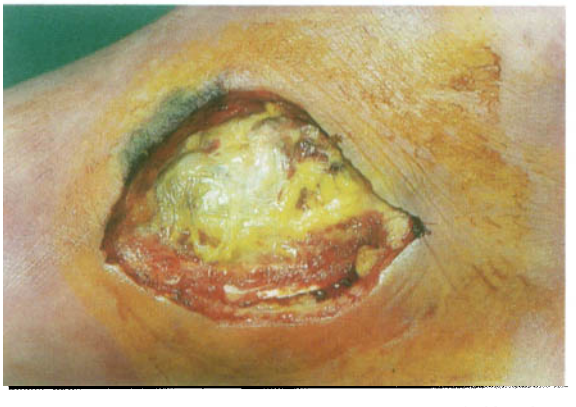

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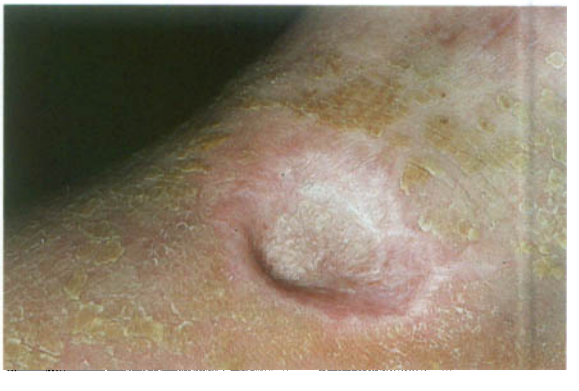


(f)

Fig. 5.20 (a) Area of redness and oedema on the medial border of the foot at presentation. (b) The temperature chart shows spikes of fever over the first 5 days with resolution of fever after surgery. (c) A patch of purplish discolouration has appeared within the cellulitis. (d) Close-up view of purple discolouration. (e) Area of debridement of purple area, showing removal of necrotic fascia. There is a small area of purple discolouration in the skin margin of the upper aspect of the wound which was debrided in a bedside procedure. (f) The wound healed in 2 months after split skin graft.

On the fifth day the patient remained pyrexial and within the area of erythema a patch of purplish discolouration was noted (Fig. 5.20c,d). In very severe cases of cellulitis, bluish-purplish discolouration of the skin indicates subcutaneous necrosis. The patient underwent surgical debridement of the area of discolouration. Subcutaneous necrosis and pus were noted. There was a wide excision of an ellipse of skin and subcutaneous tissue. Histology showed fibrous connective tissue, fat and necrotic debris showing extensive necrosis with haemorrhage.

Tissue and pus were sent for culture. Both grew *Staphylococcus aureus*. Antibiotic therapy specifically for *Staphylococcus aureus* was prescribed including rifampicin 600 mg bd, flucloxacillin and gentamicin. One day after the debridement an area of skin at the margin of the wound developed blue discolouration (Fig. 5.20e) and was debrided in a bedside procedure. The patient's temperature resolved within 2 days.

In view of the Doppler studies, the patient underwent a transfemoral angiogram which showed a 90% stenosis just distal to the origin of the right posterior tibial artery with a diffusely diseased common femoral artery. He underwent angioplasty of the posterior tibial artery 10 days after the surgical debridement. Finally a split-skin graft was applied and the wound healed in 2 months (Fig. 5.20f).

Key points

- The patient had been feeling generally ill with 'flu-like symptoms': this may be the first sign of a foot infection
- Severe infection can develop in an apparently intact foot
- Patients need close daily supervision with input from medical and surgical teams
- If fever does not resolve then further intervention is necessary
- Following surgical debridement, wounds in patients with subcutaneous necrosis should be inspected every day to detect any extension of the necrotic area. Any area developing blue discolouration of the skin should be excised.

Osteomyelitis

Osteomyelitis can complicate any of the above infective presentations.

Initial treatment

- Usually antibiotics will be given for the associated soft tissue infection as above
- When soft tissue infection is not present but a diagnosis of osteomyelitis is made clinically, an empirical regime with good bone penetration should be given such as rifampicin 300 mg tds and ciprofloxacin 500 mg bd
- On review, antibiotic selection is guided by the results of deep swabs or bone culture. Some centres base their antibiotic selection on bone culture results alone and do not believe that sinus tract or ulcer culture swabs are reliable. It is useful to choose antibiotics with good bone penetration, such as sodium fusidate 500 mg tds, rifampicin 300 mg tds, clindamycin 300 mg tds and ciprofloxacin 500 mg bd.

Follow-up plan

- Oral antibiotics should be given for at least 12 weeks. Parenteral therapy has in the past been given for 4–6 weeks followed by oral therapy. However, it may be possible to limit the parenteral therapy to 2 weeks and follow this with appropriate oral antibiotics (if the infected bone is resected then a shorter course of antibiotics such as 4 weeks may be necessary). Conservative therapy is often successful, and is associated with resolution of cellulitis and healing of the ulcer
- However, if, after 3 months treatment, the ulcer persists, with continued probing to bone which is fragmented on X-ray, we favour resection of the underlying bone, which may entail toe amputation or removal of the metatarsal head.

CASE STUDY

Conservative treatment of osteomyelitis

A 76-year-old woman with type 2 diabetes of 10 years' duration was referred to the diabetic foot clinic by casualty. She had an erythematous right 2nd toe with fusiform swelling (sausage toe) and a sloughy ulcer with a draining sinus which probed to bone (Fig. 5.21). Cellulitis extended onto the dorsum of the foot. She had been aware of redness and swelling of the toe for 3 days and the foot had begun to throb over the previous 24 h.

Pedal pulses were absent. She was sent for X-ray (unremarkable), and for vascular assessment which showed monophasic pulsatile waveforms and elevated indices due to arterial calcification. A deep swab was sent for culture and grew *Staphylococcus aureus*. The ulcer was debrided and dressed with a foam dressing. Quadruple antibiotics were prescribed initially: oral amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 400 mg tds and ciprofloxacin 500 mg bd; and then narrowed down to fucidin 500 mg tds and flucloxacillin 500 mg qds. Although repeat X-ray after 2 weeks showed lucency of the terminal phalanx compatible with osteomyelitis, the ulcer healed

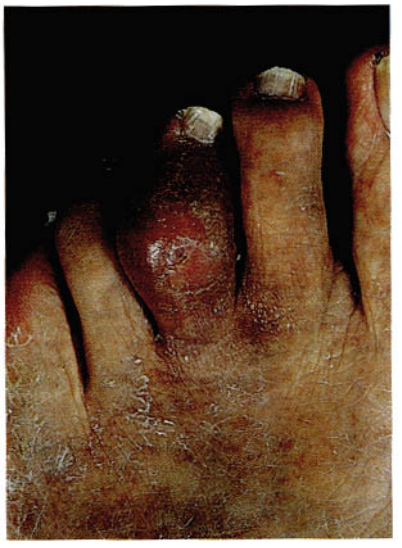


Fig. 5.21 Sausage toe with small ulcer on the dorsum.

in 3 weeks. The fusiform swelling remained but the erythema resolved after 3 months.

Key points

- Fusiform swelling (sausage toe) and erythema are frequently associated with osteomyelitis and X-rays are needed to confirm the extent of the infection in the bone and monitor progress
- Erythema may take several weeks to settle if infection was severe
- 'Sausage toe' may resolve with antibiotics: surgery is not always necessary for osteomyelitis
- In some centres osteomyelitis is treated by definitive bone resection followed by 4–6 weeks of parenteral or equivalent therapy.

Adjuvant therapy

Additional measures may improve resolution of infection. Granulocyte colony stimulating factor (GCSF) may improve outcome. Hyperbaric oxygen reduces the rate of amputations. Both need further study.

Wound control

Diabetic foot infections are almost always more extensive than would appear from initial examination and surface appearance. It is wise to perform an initial debridement in the diabetic foot clinic so that the true dimensions of the lesion can be revealed and samples obtained for culture. Often callus may be overlying the ulcer and this must be removed to reveal the extent of the underlying ulcer and allow drainage of pus and removal of infected sloughy tissue.

Wound care of ulcers with local signs of infection and ulcers with surrounding erythema

Ulcers with local signs of infection and ulcers with associated cellulitis should have podiatric debridement as described below.

Podiatric debridement of neuropathic ulcers

- All callus surrounding the wound is removed by sharp debridement
- If the ulcer is subungual, overlying nail is cut back to expose the base of the ulcer
- Undermined areas detected by probing are cut out
- Sloughy or discoloured areas of the wound bed are sharp debrided down to healthy bleeding tissue. Local discolouration of the wound bed is often a marker for an underlying track or fluctuant area containing pus. Sometimes the track has not yet broken through to the surface and the only indication of its presence is that the tissue of the wound bed which overlies the track may be a different colour, often greyish or purple. The discoloured area should be debrided away using scalpel and forceps to explore the underlying area
- Where a wound sinus is present and is very small it may be enlarged with a scalpel in order to aid drainage and enable tissue to be taken. The sinus may appear as an obvious hole in the wound bed or as a tiny slit, which is easily overlooked
- Sometimes the edges of the slit are pouting, the ulcer is very wet, and on palpation, pus or serous fluid emerges from the sinus. Fluctuant areas are drained. If pus is present it is collected in a syringe or small sterile pot for culture
- The ulcer is cleaned with normal saline. Curettings or deep swab are taken from the base of the ulcer and sent to the laboratory for culture
- Extent of cellulitis is marked with a spirit-based fibre pen so that any extension can be noted next time the dressing is lifted
- A sterile, absorbent, easily lifted dressing held in place with a light tubular bandage is applied
- The patient is instructed not to walk but to rest and elevate the foot and use crutches or wheelchair
- The dressing is lifted every day for wound inspection and to check the extent of the cellulitis. Callus or slough

which reforms is sharp debrided at frequent intervals (not longer than 1 week).

Podiatric debridement of neuroischaemic feet

Neuroischaemic feet with signs of local infection or local cellulitis are treated as above but with some important differences which are described below.

- Debridement is far less aggressive than for the neuropathic foot
- Accretions of slough in the wound bed are gently debrided with scalpel and forceps but great care is taken not to damage viable tissue
- If deep sinuses are located by probing, they should not be enlarged unless there is a very obviously fluctuant area associated with the sinus. In these circumstances the advantages of draining pus outweigh the danger of damaging ischaemic tissues
- Undermined edges are not removed
- If a halo of very thin dry callus develops around the ulcer it is very carefully debrided
- If the ulcer is subungual, overlying nail is very gently pared away so that the ulcer can drain
- The patient is sent to the hospital immediately if the foot deteriorates.

Dressing regimes for ulcers with local signs of infection or surrounding erythema

Regular dressing changes are important for all infected diabetic foot ulcers. Exudate may cause maceration and irritation of the tissues surrounding the ulcer. At dressing change, infected ulcers and surrounding areas should be carefully cleansed with saline and dried before a fresh dressing is applied. Simple, non-adherent dressings which are easily lifted are best. Any dressing which might prevent the free flow of exudate from an infected foot, or clog up a discharging sinus, should be avoided. Dressings should always be changed before 'strike-through' occurs.

CASE STUDY

Infection masked by irregular dressing changes

An 84-year-old man with type 2 diabetes of 12 years' duration, a previous above-knee amputation and a neuroischaemic foot (Fig. 5.22) developed a shallow ischaemic ulcer on his 2nd toe and a dressing was applied and changed at weekly intervals. Between dressing changes the ulcer deteriorated and became sloughy and deep with associated cellulitis, although the patient felt no pain. He was admitted to hospital for intravenous antibiotics.

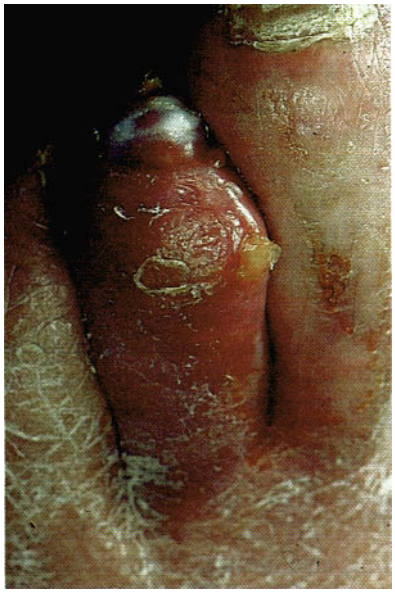


Fig. 5.22 Cellulitis and sloughy ulcer on dorsum of 2nd toe.

Vascular intervention was not feasible. The ulcer took 13 months to heal.

Key points

- Patients who lack protective pain sensation should have regular and frequent wound inspections
- We avoid using dressings which cannot be lifted frequently on patients without protective pain sensation
- Healing of previously infected lesions is very protracted in neuroischaemic patients.

Wound care of ulcers with diffuse spreading erythema, ulcers with extensive deep soft tissue infection and ulcers with extensive erythema and blue/purple/black discolouration of surrounding tissues

Diffuse spreading cellulitis should respond to intravenous antibiotics, but the patient needs daily review to detect evidence of spreading infection. An outline of the area of cellulitis may be drawn on the foot with a spirit-based pen so that extension of the cellulitic area can be detected quickly.

In extensive deep soft tissue infection and cellulitis with blue-black discolouration, the ulcer may be complicated by sloughy infected soft tissue. It is best for this tissue to be removed operatively.

The definite indications for urgent surgical intervention (as described above on p. 117) are:

- A large area of infected sloughy tissue
- Localized fluctuance and expression of pus
- Crepitus with gas in the soft tissues on X-ray
- Purplish discolouration of the skin, indicating subcutaneous necrosis.

We have repeated these indications here as we believe that they are so important. In these circumstances surgical debridement is always necessary in either neuropathic and neuroischaemic feet. However, if the foot is neuroischaemic, surgical debridement needs to be accompanied by an assessment of the arterial perfusion to the foot to evaluate the healing potential of surgical wounds. These patients will need timely vascular intervestigation.

CASE STUDY

Extensive deep soft tissue infection secondary to interdigital tinea

A 43-year-old man with type 2 diabetes of 2 years' duration was admitted via casualty with an infected neuropathic left foot with cellulitis, oedema and a purple patch on the dorsum of the foot. He was apyrexial. The dorsum of his foot was fluctuant and he was taken to theatre and underwent incision and drainage of an abscess. The pus from the abscess grew *Staphylococcus aureus* and he was treated with flucloxacillin 500 mg qds. The wound was not sutured but left open to heal by secondary intention. The original portal of entry was thought to be a webspace infected with tinea pedis. The foot healed in 9 weeks. He was issued with two pairs of bespoke trainer-style shoes and remained healed.

Key points

- Toe web infections with tinea pedis can be the portal of entry for deep soft tissue infections
- If infected neuropathic feet undergo rapid surgical drainage they heal quickly
- Infected feet with fluctuant areas should undergo surgical drainage as soon as possible to avoid the development of necrosis.

CASE STUDY

Ischaemic foot complicated by extensive deep soft tissue infection needing a wide excision

A 73-year-old Afro-Caribbean patient with type 2 diabetes of 7 years' duration, attended the diabetic foot clinic at 2-monthly intervals for nail care. He had no excessive callus formation requiring debridement. His daughter brought him to the clinic as an emergency: he was unable to put his shoe on because his foot was swollen. He felt no pain in the foot and was apyrexial. We found an ischaemic ulcer on his 5th toe which had not been present at his previous visit to the diabetic foot clinic, and brawny oedema of the dorsum of the foot (Fig. 5.23a). He was admitted to hospital for intravenous antibiotics (amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 500 mg tds and ceftazidime 1 g tds) and underwent amputation of the 4th and 5th toes and extensive debridement (Fig. 5.23b) followed by split-skin grafting and a distal bypass. The weightbearing area of the foot was greatly reduced. After the successful distal bypass had improved perfusion of the foot he began to develop plantar callus over his metatarsal heads and his wounds also developed callus and took on the appearance of neuropathic ulceration.

The foot failed to heal initially despite provision of regular debridement and bespoke footwear. The foot finally healed after the orthotist manufactured a patellar-tendon bearing weight-relieving orthosis (Fig. 5.23c).

Key points

- Extensive tissue destruction was associated with 5th toe ulcer and cellulitis needing a wide excision
- Heavy pigmentation can prevent detection of cellulitis
- The clue to infection was the swelling of the foot
- When blood flow is restricted, the ischaemic foot does not produce heavy callus in response to mechanical forces of friction and shear. However, reconstructed neuroischaemic patients may have blood flow sufficient to grow callus and hence be prone to plantar neuropathic ulceration
- If footwear does not off-load a foot effectively a patellar-tendon bearing orthosis may be helpful
- Patients who have undergone a successful bypass are observed carefully for plantar callus regrowth
- They receive regular podiatry and orthotic assessment for plantar off-loading where necessary.

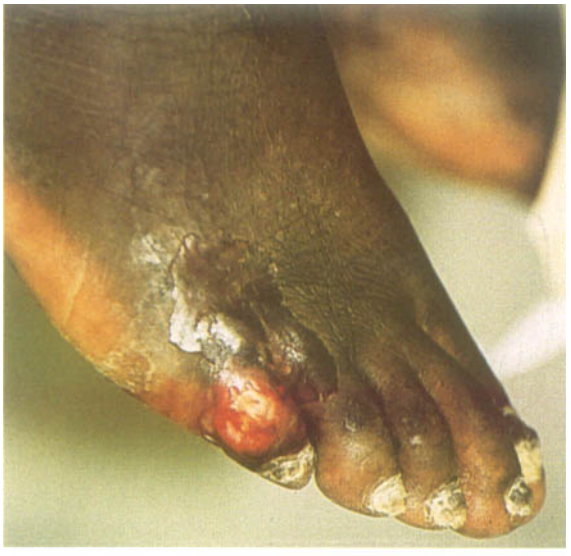
Preoperative preparation for neuropathic and neuroischaemic patients needing surgical debridement

On admission, these patients should be regarded as medical and surgical emergencies.

Preparation for surgery

The following investigations should be carried out:

- Full blood count and typing
- Serum electrolytes and creatinine
- Blood glucose



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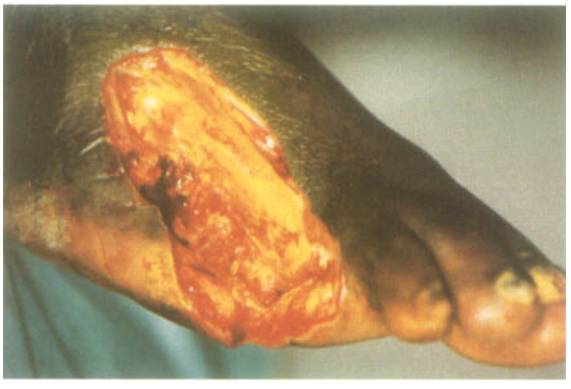

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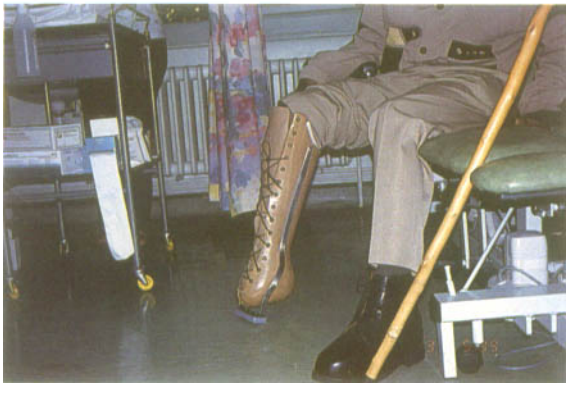


(c)

Fig. 5.23 (a) Small ulcer on 5th toe with associated blistering. (Photograph by Mark O'Brien.) (b) Extensive debridement. (Photograph by Mark O'Brien.) (c) The patient is wearing a patellar-tendon bearing weight-relieving orthosis.

Table 5.5 Insulin sliding scale. Adjust volume of fluids according to clinical state of patient

50 units soluble human insulin in 50 mL 0.9% sodium chloride	
Blood glucose (mmol/L)	**Infusion rate** (units/h)
< 4	0.5
4.1–9	1
9.1–15	2
15.1–19.9	4
> 20	Review and call doctor
Fluids	
If blood glucose > 15 mmol/L give sodium chloride 0.9%	
If blood glucose ≤ 15 mmol/L give glucose 5%	

- Liver function tests
- Electrocardiogram (ECG)
- Chest X-ray.

Patients should be medically stable prior to surgery.

Hyperglycaemia is usually present and patients with both type 1 and type 2 diabetes should be treated with an insulin sliding scale (Table 5.5).

An insulin sliding scale and intravenous fluids should be started. If patients have nephropathy there may be abnormal fluid retention and electrolyte disturbances which should be considered when prescribing the intravenous therapy. Basic cardiovascular risk should be assessed from the history, physical examination and simple investigations such as ECG and chest X-ray. Enquiry for myocardial infarction, angina, coronary artery bypass and congestive cardiac failure should be made. The high-risk patient will need close cardiovascular monitoring, and the anaesthetic technique can also be varied according to the risk of the patient.

For high-risk patients, perioperative use of β-blockers is now established, and this is discussed further in Chapter 6 where vascular surgery is covered. The anaesthetist must be aware that virtually all of these patients will have autonomic as well as peripheral neuropathy, and respiratory reflexes may be diminished. Postoperative respiratory arrests have been reported. Careful anaesthetic attention, particularly in the recovery room, is necessary.

Rarely type 1 diabetic patients may present with diabetic ketoacidosis complicating their diabetic foot infection. This should be treated before the patient goes to the operating theatre.

Emergency surgery to the foot usually consists of debridement or minor amputation. It is often difficult to assess how much debridement will be necessary and in

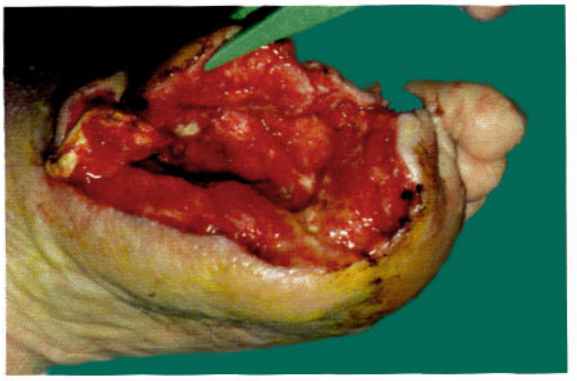


Fig. 5.24 All necrotic tissue has been removed down to healthy bleeding tissue.

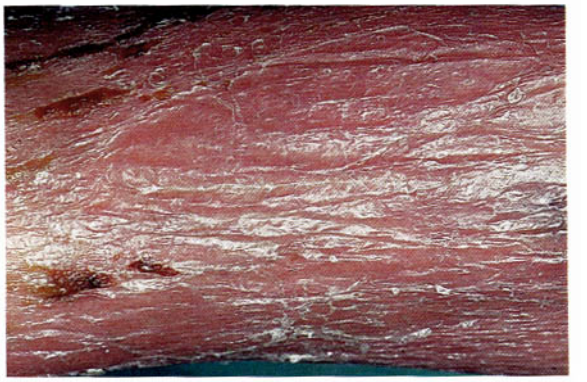


Fig. 5.25 Wrinkling of skin indicating resolution of oedema.

some cases debridement may need to be accompanied by amputation of a toe or ray. Consent for these procedures should therefore be obtained prior to operation.

The anaesthetist should understand that debridement of the foot is not a rapid procedure such as a usual incision and drainage for abscess, and therefore should anticipate at least 40 min operating time.

During surgery

- It is important that a meticulous wound exploration is carried out, with removal of infected sloughy tissue and laying open of all sinuses. It is rare to find a well-defined abscess
- The usual presentation is of heavily infected sloughy, grey tissue which needs to be removed down to healthy, bleeding tissue
- All dead tendon and necrotic tissue should be removed (Fig. 5.24). Wide excision is necessary: small incisions with drains should be avoided
- Fragmented infected and non-bleeding bone should be removed
- Deep infected tissue should be sent urgently to the microbiology laboratory
- The wound should not be sutured but left to heal by secondary intention.

After surgery

- Continue the insulin pump postoperatively until infection is resolving. Then transfer to short-acting insulin three times daily with long-acting insulin at night
- Wound irrigation with a sodium hypochlorite solution (2% Milton) may be useful for the sloughy neuropathic foot. A 1 in 50 dilution of the concentrated 1% weight in volume solution of sodium hypochlorite is made by adding 20 mL of concentrated sodium hypochlorite to 980 mL of sterile water. Approximately 300–400 mL are irrigated through the wound, making sure to swab the edges of the wound and surrounding skin with normal saline at the end of the procedure to prevent skin drying and irritation. Milton irrigation should be stopped when the wound is no longer sloughy or infected (usually within 5 days)
- We find a simple dressing regime of non-adherent dressing bulked out with gauze and held in place by a tubular bandage is best. It is very quick and simple to lift, and enables regular and frequent inspections to be done.

Signs that the foot is improving include:

- Decrease in erythema
- Pink wound
- Reduction of swelling by comparison with the other foot
- Wrinkles will be present in the skin of the foot where oedema has reduced (Fig. 5.25)
- The skin surrounding the previously infected ulcer begins to desquamate or shed and become flaky
- Discharge reduces
- Smell ceases
- Pain (if present) improves.

When there is an initial fever preoperatively, the patient's temperature is a useful indication of his progress. A steady fall in temperature is expected over the subsequent 3–4 days. If this does not occur, then uncontrolled infection should be suspected. At operation, it is sometimes difficult to remove all infected tissue and a further operative debridement may sometimes be necessary to remove remaining necrotic tissue and control infection. Deep wound swabs or tissue are taken twice weekly to assess the eradication of organisms.

- After surgery, the edges of the wound are debrided every 3 days and all callus, slough and non-viable tissue are removed. The wound is kept open and draining to heal from the base
- Patients will need bed rest and it is wise to give prophylactic subcutaneous heparin. Antithrombotic stockings should not be used on neuroischaemic feet. If they are used on neuropathic feet, they should be folded back from the toe nails to avoid pressure on the nail sulcus
- Patients who have had toe or ray amputations need careful mobilization and rehabilitation. Too rapid mobilization can provoke Charcot's osteoarthropathy.

CASE STUDY

Late presentation of Charcot's osteoarthropathy of the mid-foot after hospital admission and surgical debridement of infected diabetic foot

A 71-year-old man with previously undiagnosed type 2 diabetes was admitted to hospital with severe infection of a neuropathic foot together with proliferative retinopathy and nephropathy. Following surgical debridement and minor amputation of the first toe, the foot healed in 4 weeks. On discharge from hospital he failed a follow-up appointment and returned to the foot clinic 1 month later with an acute Charcot's osteoarthropathy, established medial convexity deformity and a neuropathic ulcer over the bony prominence.

Key points

- Charcot's osteoarthropathy can be precipitated by surgical trauma, and in particular minor amputations
- Patients who have minor amputations should be mobilized very carefully
- For detailed descriptions of surgical procedures see Chapter 8.

Vascular control

It is important to explore the possibility of revascularization in the infected neuroischaemic foot. Improvement of perfusion will not only help to control infection, but will also promote healing of the wound if operative debridement is necessary.

Angiography

Initially, non-invasive angiography should be carried out. This is either duplex angiography or magnetic resonance angiography (MRA), which should be carried out urgently to detect the presence of stenoses or occlusions which may be amenable to angioplasty or bypass.

Duplex angiography

Duplex angiography is proficient at looking at the femoral and popliteal arteries but it is sometimes difficult to obtain good views of the infrapopliteal arteries and foot arteries because of the excessive calcification in the diabetic patient.

Magnetic resonance angiography (MRA)

MRA is useful

- When femoral access is difficult
- When there is severely impaired renal function
- For delineating the aortoiliac circulation
- For demonstrating infrapopliteal vessels.

Angioplasty

When non-invasive angiography has identified the areas of occlusive disease, then angioplasty can be carried out as an invasive procedure. Angioplasty is indicated in the treatment of single or multiple stenoses or short segment occlusions of less than 10 cm. The aim is to improve the arterial circulation, achieve straight-line flow to the foot and bring about an increased blood supply to the site of ulceration and infection (Fig. 5.26a–c). Although the foot pulses may not be restored, there is usually a notable increase in the transcutaneous oxygen tension.

Angioplasty can be performed in two ways: conventional transluminal angioplasty using balloon catheters inserted through the true arterial lumen and more recently subintimal angioplasty with the catheters inserted into the subintimal plane.

Angiography and angioplasty are safe procedures with few complications so long as appropriate precautions are taken. Investigations should include:

- A recent full blood count, including a platelet count
- Blood coagulation indices
- Serum electrolytes and creatinine
- Blood grouping.

The patient should not be dehydrated. Start an insulin sliding scale together with intravenous fluids before the procedure. Dopamine is no longer used to protect kidney function but it is important to keep the patient hydrated with intravenous fluids prior to the procedure. Patients with impaired renal function should be prescribed acetyl cysteine 600 mg bd 24 h before the procedure and on the day of the procedure.

Fig. 5.26 (a) Critical stenosis of the anterior tibial artery 3 cm from its margin. (b) Angioplasty has relieved stenosis of anterior tibial artery. (c) Straight-line flow to the foot has been achieved. (Courtesy of Dr Paul Sidhu.)

If the patient has previously been on warfarin then this must be stopped at least 3 days prior to the procedure and the patient changed to intravenous heparin. The heparin infusion is stopped 2 h before the procedure. Metformin should be stopped 2 days before the procedure and restarted 2 days after the procedure.

Postangiography, patients should be monitored closely, recording blood pressure and pulse. There is a possibility of bleeding from the femoral artery injection site, which may not be immediately apparent. The pulse rate may not respond in the usual way to loss of intravascular volume because of autonomic neuropathy: therefore tachycardia may not be present. However, blood pressure will drop and an urgent blood count together with cross-matching for at least 6 pints of blood should be performed. Full resuscitation should immediately be carried out with haemodynamic monitoring. Most cases of bleeding resolve with blood replacement and tamponade themselves off spontaneously. A mass may become palpable in the lower abdomen but this may not appear until 5–6 h after the procedure.

Continued hypotension despite adequate blood replacement is an indication for surgical exploration.

Postangioplasty it may be advisable to give heparin if a long occlusion has been treated. It is best to give unfractionated heparin intravenously. If there is any postprocedure bleeding over the subsequent few hours from the femoral artery injection site, this can be exacerbated by heparin. Unfractionated heparin can be easily stopped if given intravenously: however, low molecular weight

heparin given subcutaneously is difficult to reverse and we have had one case of severe postprocedure bleeding in these circumstances.

In patients with impaired renal function, serum creatinine may rise after the procedure, and should be checked after 48 h. Usually there is a mild rise of serum creatinine of 50 mmol/L (0.57 mg/dL) and this falls gradually over the subsequent 10 days, but should be carefully monitored.

In some cases renal impairment may be severe and renal support in the form of dialysis needed.

Arterial bypass

If angioplasty is not possible because of long arterial occlusions, bypass should be considered.

If the infection is responding to conservative treatment, with resolution of cellulitis on intravenous antibiotics, then bypass, with its inherent risks, is probably not indicated. However, if operative debridement is necessary with amputation of a toe or ray, then arterial bypass may be necessary to achieve full wound healing.

Mechanical control

Patients with extensive cellulitis should not walk. They should be on bed rest and use crutches, Zimmer frame or wheelchair for trips to the bathroom. Every step taken will spread infection.

Heels must be protected with a pillow/foam wedge under the calves to keep the patient's heels clear of the mattress when he is in bed. Special mattresses should be provided to prevent decubitus ulcers. If he slides down the bed he risks pressure lesions from the bed end.

Long periods on the operating table can lead to heel blistering and heel necrosis, and patients who go to theatre should have their heels regularly protected.

We sometimes off-load postoperative wounds in the neuropathic foot with a removable bivalved cast or windowed cast which enable wound inspection.

After operative debridement in the neuroischaemic foot, non-weightbearing is advised until the wound is healed. Patients with infected neuroischaemic feet may wear a 'Scotchcast' boot in bed to protect the heel.

Pressure-relieving ankle–foot orthoses (PRAFOs) are helpful heel off-loaders in both classes of foot.

Metabolic control

It is important to make sure that there are no systemic, metabolic or nutritional disturbances to impair the response to infection and retard healing of wounds.

In severe infections, considerable metabolic decompensation may occur. Full resuscitation is urgently required with intravenous fluids and intravenous insulin sliding scale which is often necessary to achieve good blood glucose control whilst the patient is infected. This is followed by a basal-bolus regimen of three times a day short-acting insulin before meals and long-acting insulin at night. Total dose of insulin (units) = 0.5 × body weight (kg), split 2/3 as bolus and 1/3 as basal.

These are complex patients, and cardiac and renal function should be assessed. Echocardiography will identify patients with left ventricular dysfunction. This is expressed as the ejection fraction and a value less than 35% increases the risk of non-cardiac surgery. Close observation and monitoring of these systems is essential to maintain correct electrolyte and fluid balance, especially postoperatively.

Neuroischaemic patients should be regularly taking statins, angiotensin-converting enzyme (ACE) inhibitors and antiplatelet agents and these should be continued if the patient is admitted to hospital. Aspirin should not be stopped before angiography or angioplasty although if the patient is taking aspirin and clopidogrel, the latter should be stopped.

High blood glucose is associated with reduced white cell function, which improves when the blood glucose is lowered.

Educational control

Patients who develop severe infection and present late may have other problems which predispose them towards neglecting infections and failing to accept care. These include:

- Drug addiction
- Psychological problems.

Drug addiction

Some of the worst neglected foot infections we have seen were in the neuropathic feet of young diabetic patients who were addicted to drugs.

CASE STUDY

Early discharge without accepting treatment by a young diabetic patient addicted to crack cocaine

A 28-year-old man with type 1 diabetes mellitus for 18 years attended the casualty department complaining of a painful foot. He was well known to the hospital and was addicted to crack cocaine. He had severe infection of

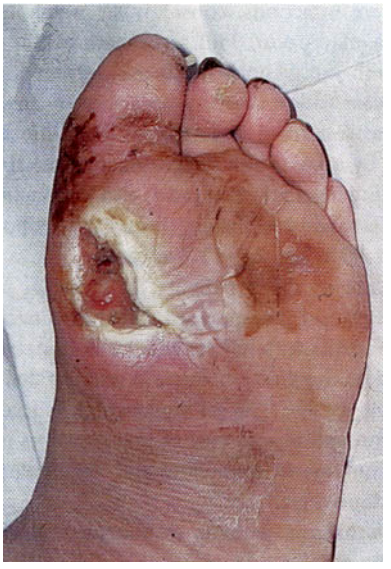


Fig. 5.27 Ulcer over 1st metatarsal head with surrounding cellulitis.

the left hallux, and deep, infected ulcers over both 1st metatarsal heads (Fig. 5.27). He was admitted to the ward for intravenous antibiotics and possible surgical debridement but walked off the ward 2 h later before treatment was started and was lost to follow-up. Three weeks later he presented again at casualty and accepted admission. He explained that he was a crack cocaine addict and had self-discharged because he was fleeing from his dealer to whom he owed money. In the meantime he had accepted treatment from another hospital two hundred miles away where he received a short course of intravenous antibiotics. Again he self-discharged when his supply of drugs ran out. Back at home he did not seek further treatment until he developed wet necrosis of the hallux and the pain in his foot became extremely severe.

He underwent extensive surgical debridement of the 1st ray and amputation of the hallux. The foot was slow to heal, and he frequently left the ward for periods of several hours without saying where he was going and a cast with a window over the ulcer was applied to protect his foot. Nursing staff on the ward found him a very difficult patient who would not follow advice. He discharged himself after 8 weeks, and kept two follow-up appointments in the casting clinic but was then lost to follow-up.

Key points

- Drug addiction is an enormous barrier to care. Patients are frequently non-compliant with erratic attendances. The door should always be left open to these patients
- Addicts are frequently unable to attend clinic regularly, to follow the treatment regimes suggested, to control their diabetes adequately or to attend early when problems arise
- Refusal to enter hospital and self-discharge from hospital against medical advice are also common in these very challenging patients.

Psychological problems

Concurrent psychological problems are also formidable barriers to care.

Figure 5.28a,b shows the feet of a middle-aged depressed man who lived alone and neglected his feet. His right foot developed ulceration over the dorsum of the 2nd toe which was complicated by cellulitis. He had peripheral neuropathy with marked clawing of the toes. He wore no socks, the ulcer was not dressed and his shoes were too tight.

It is important for all patients and their families to understand the dangers of untreated foot infections, that signs and symptoms are often diminished and that it is essential to check the foot regularly to detect deterioration. We use a question and answer sheet to educate patients as follows.

Why is foot infection a dangerous complication of diabetes?

In a diabetic foot, the usual warning signs of infection may be absent or greatly reduced, particularly if you have poorly controlled diabetes, neuropathy or poor blood supply to your feet. If you do not know you have an infection and you continue to walk then infection will be pumped through the foot with every step you take. You can become seriously ill very quickly.

Wouldn't I know there was a serious problem because my foot would hurt?

Infection in diabetic feet is not always painful, but it can quietly destroy the foot. If you wait for pain or other symptoms to develop, the foot infection may need many weeks of treatment and it may even be too late to save the foot.

How can I recognize an infection early?

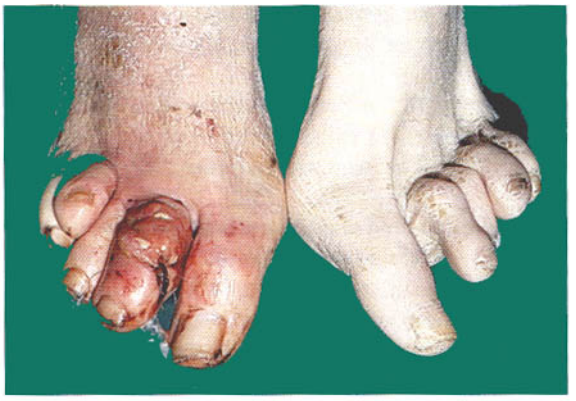
Your feet should be carefully checked every day for signs of infection.

What are the signs of infection?

You should watch out for:

- Swelling of a foot or part of a foot. Compare your two feet: are they the same size?

(a)



(b)

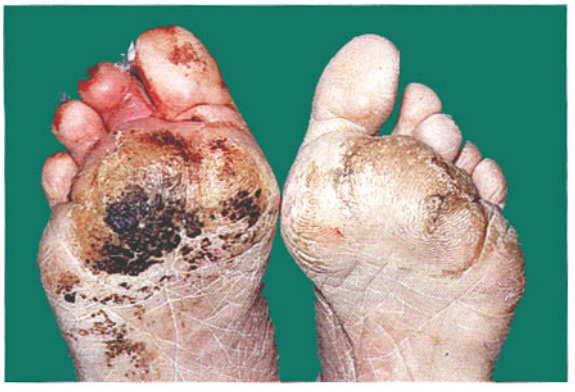


Fig. 5.28 (a) Dorsal view of both feet shows severe clawing of toes, ulceration and cellulitis. (b) Plantar view of the same pair of feet shows neglected callus and accumulated crusted exudates.

- Colour change. Look for red patches or streaks spreading up the foot and leg, or patches of blue, purple or black near the ulcer
- Collection of fluid under the skin which may look like a clear blister, a blood blister or a blister filled with pus. These may also develop under areas of hard skin
- Pain or throbbing in the foot
- Foot develops a hot spot
- Pus or watery discharge or blood leaking from any part of the foot
- The foot smells strongly
- Your body temperature rises above 37.5°C
- Your diabetes goes out of control for no obvious reason
- You shiver and shake
- You feel very cold, or burning hot
- You feel tired, sleepy, weak or unwell with loss of appetite and 'flu-like symptoms'.

What shall I do if I can't check my own feet for these danger signs?

If it is difficult for you to see your feet clearly or to get down to your feet, then please tell the foot clinic.

Remember that symptoms that feel like flu may be due to a foot infection. Always check both your feet if you are unwell. If you call the doctor in, ask him to check your feet.

What should I do if I find a danger sign?

- Seek help the same day from your diabetic foot service or emergency department
- Do not walk on the infected foot: every step you take will pump infection through the foot and leg
- Until you get to hospital, lie with your foot up on a bed or sofa
- Do not wait until tomorrow: the worst infections are the ones which are not caught early.

Must I go at once?

Even if you have an appointment with your GP or foot clinic very soon you should never wait until then: always act the same day.

Remember that the earlier in the day that you come to clinic as an emergency, the easier it is to sort out your problem. It is difficult to get any necessary tests and investigations performed late in the day. If you need to be admitted into hospital, and beds are in short supply, then the earlier the hunt for a bed is started the sooner your treatment can begin.

Is my foot really so important? Even if it doesn't hurt?

Please always give your foot a high priority. It may not be painful, but it is very important to start treatment as soon as possible.

What if I don't like hospital?

You may be afraid of being taken into hospital, but one thing is sure: if you delay then any hospital stay will be for much longer.

Isn't it unfair for me to get extra appointments when other people need help too and the foot service is busy?

Never delay coming to the foot clinic as an emergency because you feel it is unfair for you to receive extra appointments over and above your routine ones, or because you know the staff are very busy. Good diabetic foot services always run an emergency service in order to catch problems early. Their workload allows for emergency visits. If you do not play your part in this system and your foot is badly damaged by the delay, then the result will be extra work and trouble for everybody, especially you.

Isn't it unwise to take antibiotics for long periods?
Some patients worry about taking antibiotics for long periods. But if diabetic foot ulcers become badly infected the leg is at risk, which is why antibiotics are often prescribed earlier, and for longer periods, than in a patient who is not diabetic.

Didn't I read in the paper that antibiotics cause superbugs which are resistant?
Some patients worry because they read in the papers that taking too many antibiotics can cause germs to develop drug resistance, so that antibiotics will not work for them in the future. The most common cause of this problem is when patients do not complete a course of antibiotics. You can help prevent superbugs from emerging by never stopping your tablets without consulting your doctor. Always take your antibiotics with you wherever you go so that you do not miss a dose.

Do antibiotics cause side-effects?
Some antibiotics can cause side-effects. If you ever develop new symptoms when taking antibiotics you should rapidly contact the diabetic foot clinic and ask for advice. However, you can reduce the likelihood of problems by following precise instructions on the medication, washing down tablets with plenty of water and eating live yoghurt.

How does yoghurt help?
The reason for this is that your bowel (intestines, gut) is full of harmless microorganisms which help you to digest your food. The antibiotics kill the harmful germs in your foot, but also kill the good microorganisms in your bowel. If you eat live yoghurt you will replace the good microorganisms. Below is more information about side-effects.

Possible side-effects from antibiotics
Side-effects: what to do
- Nausea and indigestion: take tablets with lots of water and live yoghurt

If any of the following problems arise, stop the antibiotics and contact the diabetic foot service:
- Vomiting
- Diarrhoea
- Rash
- Severe itching
- Hallucinations
- Hypoglycaemia.

Except in the circumstances explained above you should never stop taking your antibiotics without checking first with the foot clinic. If you stop a course part-way through it may make the germs resistant to the antibiotic so that the antibiotic no longer works for you—or for other patients.

Remember that any other symptoms you develop may have nothing to do with the antibiotics you are taking. Always check it out with your foot clinic.

What else do I need to do if I am taking antibiotics?
Keep a precise record of all the medication you are taking and bring it with you to the foot clinic every time you come. It is very difficult for the clinic to plan your treatment when they do not know what other medication you are taking.

If you see another doctor who stops your foot clinic medication, or prescribes new medication ask him to speak to the clinic. You should also let the clinic know if any new health problems are diagnosed.

How should I look after an infected ulcer?
- Do keep your foot out of the bath or shower unless it is covered with a plastic cast protector. This protects the wound from damage, and protects other people from your infection. If infected wounds are left uncovered they are very attractive to flies, dogs and cats
- Do ensure the dressing is changed at least once a day, and the wound is washed with saline and dried carefully. Ask for help if you cannot look after the wound yourself
- If fluid strikes through the dressing so that you can see it, then it is time for the dressing to be changed
- Do not use a dressing which is designed to be left on the foot for several days. This is very dangerous for diabetic feet which lack protective pain sensation and have an infection because the infection can spread under the area covered by the dressing and you will not know that this is happening
- Do not change the foot clinic treatment regime without consulting them first. If you have a nurse coming to your home to do the dressings and she wants to change the type of dressing used, then ask her to discuss this with the foot clinic. If your doctor changes the treatment, let the diabetic foot clinic know.

Can I use alternative medicines or folk remedies?
It is unwise to use alternative medicine, folk remedies or over-the-counter treatments for an infected diabetic foot. Always check with your foot clinic before doing this.

Is it all right to soak my foot in salty water?
If you want to do this then always check the temperature

of the water first. It should be under 43°C. Use a bath thermometer to measure the temperature.

Can I put a hot poultice on my foot to draw out the infection?
No. You risk severe burns.

Anything else I should be doing?
Don't walk. Use crutches or a wheelchair if you have to move. Every step you take will spread infection along your foot. Resting your foot is essential. Every step you take will make the infection worse.

PRACTICE POINTS

- Signs and symptoms of foot infection are diminished in diabetic patients
- Microbiological investigation is essential for all infected diabetic feet
- Severe infections need intravenous antibiotic therapy and urgent assessment of the need for surgical drainage and debridement
- Infected neuroischaemic feet need vascular assessment and intervention where appropriate
- Patients with ulceration should be taught the danger signs of infection
- Without urgent treatment, severe infections will progress to necrosis.

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6 Stage 5: the necrotic foot

How poor are they that have not patience!
What wound did ever heal but by degrees?
(Othello, II, iii, William Shakespeare)

PRESENTATION AND DIAGNOSIS

This stage is characterized by the presence of necrosis (gangrene) which has grave implications, threatening the loss of the limb. Necrosis can involve skin, subcutaneous and fascial layers. In lightly pigmented skin it is easily evident but in the subcutaneous and fascial layers it is not so apparent. Furthermore, the extent of necrosis may be difficult to determine: often the bluish-black discolouration of skin is just the 'tip of an iceberg' of massive necrosis.

Other conditions may masquerade as necrosis (see Chapter 1). Purplish-black or brown discolouration of the skin also occurs after bruising and is sometimes difficult to differentiate from early necrosis associated with a history of trauma. A superficial collection of dried blood within a blister or tracking under the skin can give part of a foot a black and leathery appearance. Cyanosis of toes and feet is seen in severe cardiac and respiratory failure.

Shoe dye and the application of topical henna will result in black or brown discolouration of the skin.

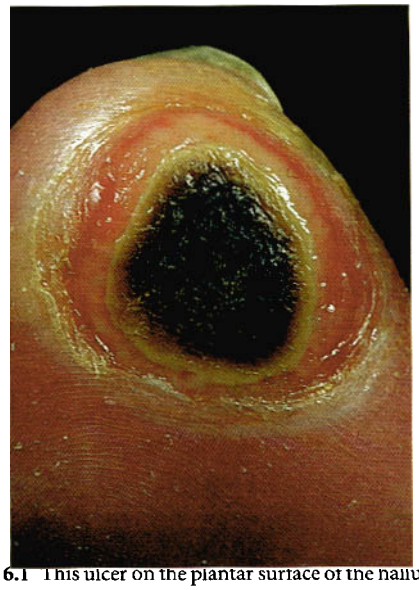


Fig. 6.1 This ulcer on the plantar surface of the hallux changed its colour from healthy pink to black over a 4-day period.

Early signs of necrosis

The signs that part of a foot is becoming necrotic may be subtle in the early stages, and may mimic bruising or chilblains. A careful search should be made for early signs:

- A toe which is developing a blue or purple tinge, having been previously pink because of infection or ischaemia
- Toes which have become very pale in comparison with their fellows
- An ulcer which has changed its colour from healthy shiny pink granulations to grey, purple or black or its texture from a smooth to a matt surface (Fig. 6.1).

Causes of necrosis

Necrosis can be due to infection, when it is usually wet, or to occlusive macrovascular disease of the arteries of the leg, when it is usually dry. Necrosis is not, as previously thought, due to a microangiopathic arteriolar occlusive disease, or so-called small vessel disease. Health-care professionals working with diabetic foot disease should avoid using this term, which is imprecise and may lead to therapeutic nihilism.

Digital necrosis is common in patients with renal impairment, particularly those with end-stage renal failure, even though they are treated with dialysis. Patients

with severe renal impairment also have a propensity to develop dry necrosis, sometimes in the presence of palpable pedal pulses and in the absence of infection. A further discussion of foot disease in the diabetic patient with renal impairment is given below.

Wet necrosis

Wet necrosis is secondary to a septic vasculitis associated with severe soft tissue infection and ulceration, and is the commonest type of necrosis in the diabetic foot. However, there is often a delay in presentation of the patient.

CASE STUDY

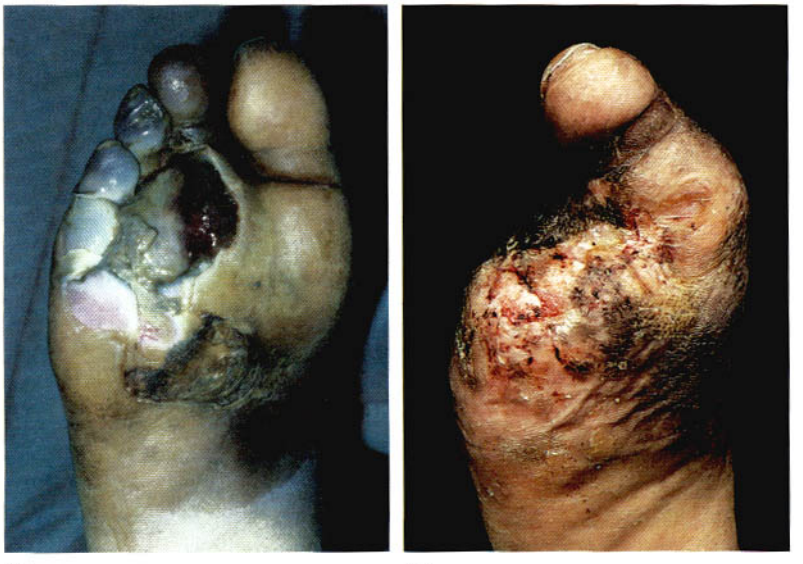
Puncture wound with delay in presentation of wet necrotic foot

A 38-year-old male Afro-Caribbean patient with type 2 diabetes mellitus of 2 years' known duration, and a body mass index of 31, who had been lost to follow-up after his initial diagnosis, trod on a tin tack. This penetrated the sole of his shoe during his work as a school caretaker. He visited his general practitioner who prescribed a 5-day course of 250 mg amoxicillin tds. After 5 days the patient thought the foot had healed.

Two weeks later, the patient's girlfriend noted greenish discolouration of the sole of his foot and insisted that he attend the hospital casualty department. The plantar surface of the forefoot was bulging, there was deep infection involving the 3rd, 4th and 5th webspaces and the 2nd, 3rd, 4th and 5th toes were necrotic, although this was not immediately apparent because the dorsum of the foot was heavily pigmented (Fig. 6.2a). He was admitted to hospital for intravenous antibiotics and surgical debridement, and four toes and their adjoining metatarsal heads were removed. Once the infection was controlled he insisted on returning to work. He was treated in a total-contact cast, and healed in 6 months (Fig. 6.2b). He was physically very active and his casts needed additional strengthening and very frequent replacement. On one occasion, the day after his cast had been applied, he went for a very muddy walk on Dartmoor, a national park renowned for its bogs and rocks, in his cast and returned to the clinic with the cast in tatters. He developed end-stage renal failure but continued to work and to lead an active life and while on holiday in Spain dialysed in the local restaurant by hanging his CAPD bag from a hat stand. Two years after his admission for the foot problem he was found dead in bed from a myocardial infarction.

Key points

- Untreated infection can rapidly lead to necrosis
- Puncture wounds should be followed up very carefully as signs of infection will only become apparent when they have spread from the deep tissues to the superficial structures
- Bulging of the plantar surface indicates deep infection with collection of pus which needs drainage
- In the neuropathic foot, extensive necrosis can be



(a) (b)

Fig. 6.2 (a) Plantar view of infection following a puncture wound which has led to wet necrosis of the forefoot requiring amputation of four toes and their adjoining metatarsal heads. (b) Full healing of the very large postsurgical tissue defect took 6 months. He wore a total-contact cast to off-load the wound.

successfully treated by surgical debridement with eventual complete healing
- Physically active and heavy patients need extra-strong casts.

CASE STUDY
Delay in presentation due to confusion between blood blister and wet necrosis

A 67-year-old man with type 2 diabetes which had not previously been diagnosed, developed what he took to be a blood blister on his left hallux. The lesion was not painful, he felt well, and he did not seek treatment until he noticed an unpleasant odour and went to casualty. The toe was infected and necrotic. Pedal pulses were bounding. His vibration perception threshold was 45 volts (Fig. 6.3). He underwent amputation of the hallux and the foot healed in 4 months. Three weeks after he was discharged he re-presented at the diabetic foot clinic with a rocker-bottom Charcot foot. This was treated in a total-contact cast for 6 months and there was no progression of the deformity.

Key points
- Necrosis may be mistaken for blood blisters by patients and health-care professionals
- In patients with neuropathy, necrosis may not be painful
- Patients with neuropathy who undergo surgery for necrosis may develop Charcot's osteoarthropathy
- Patients with type 2 diabetes may have undiagnosed diabetes for many years and develop severe complications including neuropathy and foot ulceration by the time of diagnosis
- In wet necrosis, the tissues are grey or black, moist and often malodorous. Adjoining tissues are infected and pus may discharge from an ulcerated area between necrosis and viable tissue. There may be no clear demarcation line between necrosis and viable tissue and it may be difficult to predict exactly which areas of tissue are viable until debridement has been performed
- Once infection is established, necrosis can develop within a few hours.

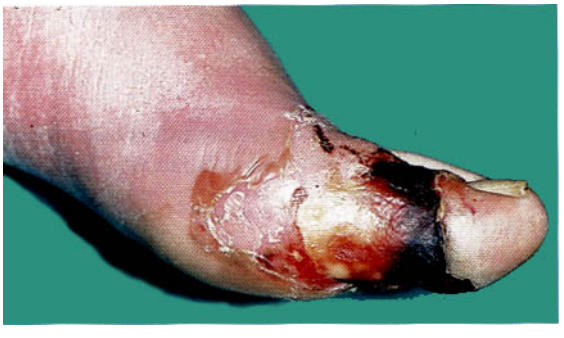


Fig. 6.3 This necrotic toe in a previously undiagnosed diabetic patient was thought by him to be a blood blister.

CASE STUDY
Wet necrosis with rapid onset

A 73-year-old Afro-Caribbean woman with type 2 diabetes of 30 years' duration, peripheral vascular disease and a previous below-knee amputation attended the diabetic foot clinic with a 2-cm broken blister on her left heel. She was obese and confined to a wheelchair. She did not want to take antibiotics and said she would prefer not to have visits from the district nursing service as her daughter, with whom she lived, would look after the foot. Her daughter was carefully taught to clean and dress the foot, and advised to check it every day. We emphasized the need for immediate return to the diabetic foot clinic if any deterioration occurred. The patient returned to clinic 1 week later, for her routine appointment, with a discharging, malodorous ulcer and extensive deep necrosis. She was admitted to hospital and given intravenous antibiotics. She had angiography but there was severe infrapopliteal disease and no distal arterial run-off. No vascular intervention was feasible and she underwent a second below-knee amputation.

Key points
- Ischaemic foot ulcers can become infected rapidly and deteriorate to wet necrosis with alarming rapidity
- Detection of deterioration was rendered more difficult because the patient had very heavily pigmented skin and did not want home nursing visits
- We encourage our patients and their families to be 'critically observant'. At the first appearance of a break in the skin, no matter how small and shallow, they are advised to seek help the same day.

Dry necrosis

Dry necrosis is hard, blackened, leathery, mummified tissue. There is usually a clear demarcation line between necrosis and viable tissue.

Dry necrosis is the result of severe ischaemia, secondary to poor tissue perfusion from atherosclerotic narrowing of the arteries of the leg, often complicated by thrombus and occasionally, emboli.

Necrosis in the neuropathic foot

In the neuropathic foot, the first presentation of necrosis is almost invariably of wet necrosis, and this is caused when infection complicates an ulcer, leading to a septic arteritis of the digital and small arteries of the foot. The walls of these arteries are infiltrated by polymorphonuclear leukocytes leading to occlusion of the lumen by septic thrombus. This leads to the so-called 'diabetic gangrene' where a toe become blue and subsequently black and necrosed, while a few centimetres proximally a bounding pedal pulse can often be palpated. It is this presentation which probably gave rise to the myth of diabetic gangrene being caused by 'small vessel disease'. Sometimes the portal of entry for the infection which damages the digital arteries is on the same toe, but it may be proximal and is sometimes several centimetres away from the affected toe.

CASE STUDY
Septic arteritis and wet necrosis in a neuropathic foot

A 72-year-old man with type 2 diabetes of 11 years' duration and peripheral neuropathy developed a neuropathic plantar ulcer over his 4th metatarsal head. After 3 weeks the foot became swollen with purulent discharge and he was systemically unwell. He was admitted to hospital and given amoxicillin 500 mg tds, flucloxacillin 500 mg qds and metronidazole 500 mg tds intravenously. An ulcer swab grew *Staphylococcus aureus* and *Streptococcus* group B and mixed anaerobes.

The plantar ulcer was the only break in the skin. Within 24 h it was observed that the 4th toe was turning blue (Fig. 6.4a). The patient underwent a double ray amputation. The amputation specimen was taken for histological examination which revealed septic arteritis of both digital arteries (Fig. 6.4b). The lumen of each artery was almost totally occluded by septic thrombus. The foot healed in 8 weeks (Fig. 6.4c).

Key points

- When necrosis develops in the neuropathic foot, it is usually due to infection and presents as wet necrosis
- Forefoot plantar ulceration can lead to webspace infection with septic arteritis of the digital circulation
- Septic arteritis leads to gangrene even in the well-perfused neuropathic foot
- Early treatment of septic arteritis may salvage the affected toe.

Necrosis in the neuroischaemic foot

Both wet and dry necrosis can occur in the neuroischaemic foot.

Wet necrosis

Wet necrosis is caused by a septic arteritis, secondary to soft tissue infection and ulceration as in the neuropathic foot. However, in the neuroischaemic foot, reduced arterial perfusion to the foot resulting from atherosclerotic occlusive disease of the leg arteries is an important predisposing factor. When wet and infected necrosis is successfully treated with antibiotics it will dessicate and become dry necrosis.

CASE STUDY
Necrosis from *Pseudomonas* infection

A 77-year-old man with type 2 diabetes of 12 years' duration developed ulceration on the lateral aspect of his right 5th metatarsal head. His feet were neuroischaemic. There was a sudden deterioration with a mild fever and spreading wet necrosis (Fig. 6.5a). A tissue sample was taken for culture which revealed a pure heavy growth of *Pseudomonas*. The patient was treated with antipseudomonal therapy in the form of ceftazidime 1 g tds and the necrosis which had previously been wet became dry and the fever resolved (Fig. 6.5b,c).

Key points

- Gram-negative organisms including *Pseudomonas*, *Citrobacter*, *Serratia* and *Acinetobacter* can cause infection and necrosis in the diabetic foot
- It is important to detect such organisms by collecting a tissue specimen, if possible, or a deep ulcer swab
- Spreading necrosis in the neuroischaemic foot may be due to infection and not to increasing ischaemia and should be treated with antibiotic therapy and, if indicated, surgical debridement.

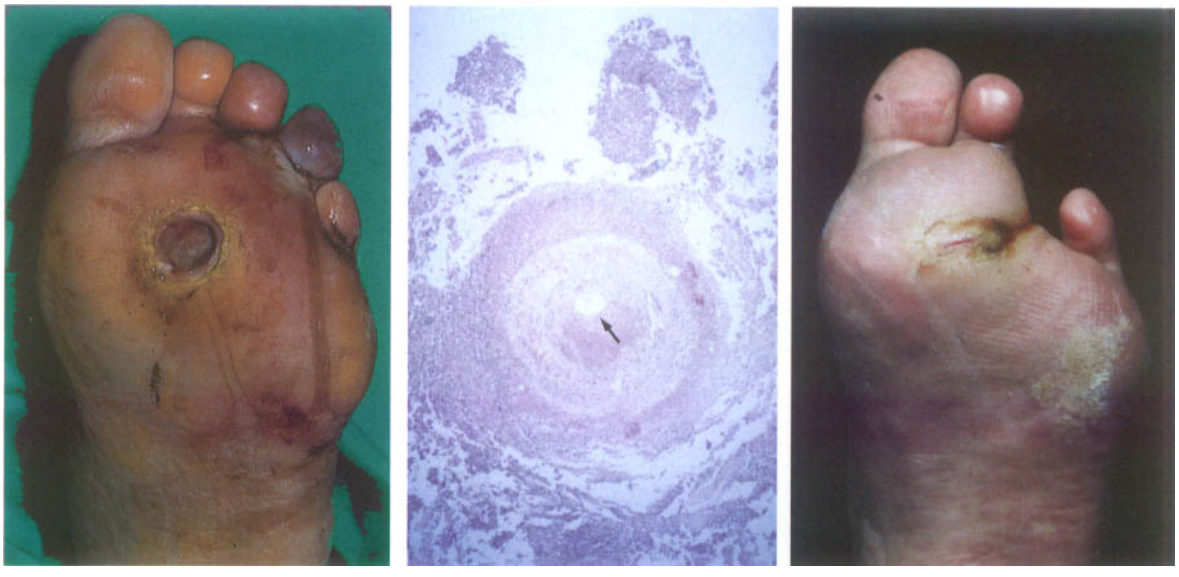
Dry necrosis

Dry necrosis is secondary to a severe reduction in arterial perfusion and occurs in three circumstances:

- Severe chronic ischaemia
- Acute ischaemia
- Emboli to the toes.

Severe chronic ischaemia

Peripheral arterial disease usually progresses slowly in the diabetic patient, but eventually a severe reduction in



(a) (b) (c)

Fig. 6.4 (a) Neuropathic foot with infected plantar ulcer. The 4th toe has turned blue because septic arteritis has led to occlusion of both digital arteries. The 3rd toe is changing colour. (b) Septic arteritis. Cross-section of digital artery showing lumen almost totally occluded by septic thrombus. (c) Foot healed after amputation of 3rd and 4th rays.

arterial perfusion results in vascular compromise of the skin. This is often precipitated by minor trauma, leading to a cold, blue toe which usually becomes necrotic unless the foot is revascularized. Many diabetic feet with a very low pressure index do well until the skin is breached by an injury. Inflammation and successful healing make increased vascular demands which the ischaemic foot is unable to fulfil.

Many diabetic neuroischaemic patients never complain of intermittent claudication or rest pain. If the patient has concurrent retinopathy with severe visual impairment he will frequently be unaware of ulcers or necrosis. The name 'eye–foot syndrome' has been attached to cases of middle-aged or elderly men who lived alone, had undiagnosed diabetes leading to retinopathy and neuropathy, and presented late with necrosis of the feet.

Acute ischaemia

Blue discolouration leading to necrosis of the toes is also seen in acute ischaemia, which is usually caused either by thrombosis complicating an atherosclerotic narrowing in the superficial femoral or popliteal artery or emboli from proximal atherosclerotic plaques to the femoral or popliteal arteries.

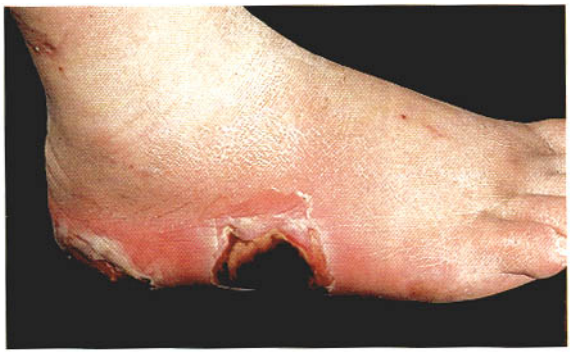
Acute ischaemia presents as a sudden onset of pain in the leg associated with pallor and coldness of the foot, quickly followed by mottling and slaty grey discolouration, and pallor of the nail beds. The diabetic patient may not experience paraesthesiae because of an existing sensory neuropathy, which also reduces the severity of ischaemic pain and may delay presentation. Some patients may complain of extreme weakness of the affected limb but not of pain.

CASE STUDY

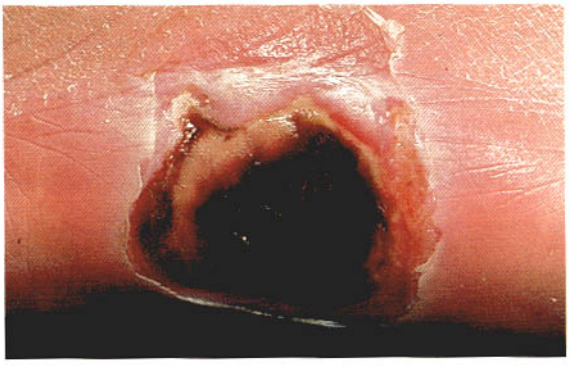
Necrosis following acute ischaemia

A 74-year-old lady with type 2 diabetes of 32 years' duration, developed acute ischaemia of her right leg (Fig. 6.6a). Postoperatively she developed blistering on the dorsum of the foot and discolouration of the tips of three toes, but the foot was well perfused and the graft was patent (Fig. 6.6b–d). She was treated conservatively with systemic antibiotics to control and eradicate infection and gentle debridement of the demarcation lines between gangrene and viable tissue. She had regular graft surveillance. After 7 months the necrotic areas autoamputated and the foot healed.

(a)



(b)



(c)

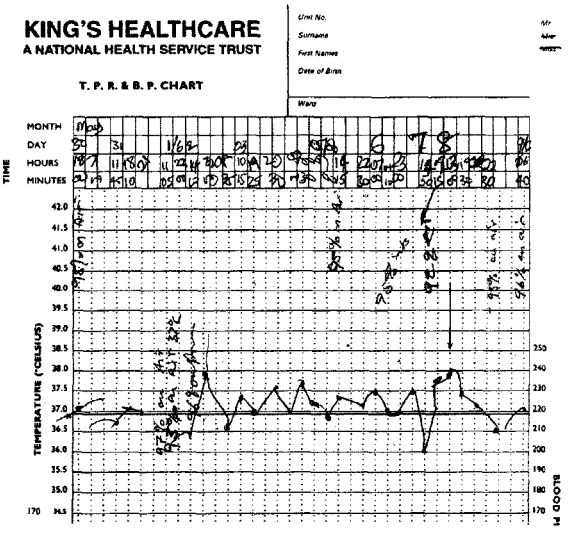


Fig. 6.5 (a) The foot has developed spreading necrosis. (b) Close-up view of foot. (c) The patient had fever whilst the necrosis was spreading and this resolved when ceftazidime as antipseudomonal treatment was started.

Key points

- After an episode of acute ischaemia patients may develop areas of necrosis even following successful revascularization
- Such necrosis is related to the markedly reduced perfusion during the acute ischaemic episode
- These areas of necrosis are usually dry but may become wet if they become infected
- Such necrosis can be treated conservatively
- If necrosis develops after an arterial bypass, it is important to check perfusion of the foot immediately to ensure that the graft is still patent.

Emboli to the toes

Another cause of necrosis, particularly to the toe, is the passage of emboli to the digital circulation often originating from atherosclerotic plaques in the aorta and leg arteries.

'Showers of emboli' may originate from plaques in the aortoiliac and the superficial femoral arteries. The plaques are usually irregular or ulcerated and covered with debris particularly in the aorta. The emboli lead to cool painful cyanotic toes and the development of areas of necrosis at the tips of the toes, which generally heal without the need for amputation (Fig. 6.7). These patients may present with palpable pedal pulses.

Emboli may also occur as a complication of invasive angiographic procedures. Emboli may also originate from the heart. Cholesterol emboli may also be related to warfarin therapy.

The initial sign of emboli may be bluish or purple discolouration which is quite well demarcated but which quickly proceeds to necrosis. If it escapes infection it will dry out and mummify.

If patients with emboli have minimal or no neuropathy the foot is extremely painful.

CASE STUDY

Emboli, minimal neuropathy and severe localized pain

A 51-year-old woman with type 2 diabetes of 7 years' duration was referred to the diabetic foot clinic by the vascular surgeon who had diagnosed peripheral embolic disease. She had very discrete areas of non-blanching blue discolouration on the tips of her right 3rd and 5th toes (Fig. 6.8). Her pressure index was 0.5 and her vibration perception threshold was 20 volts.

Angiography had shown multiple stenoses of the right superficial femoral artery and she was due for angioplasty. Aspirin and dipyridamole had been prescribed by the

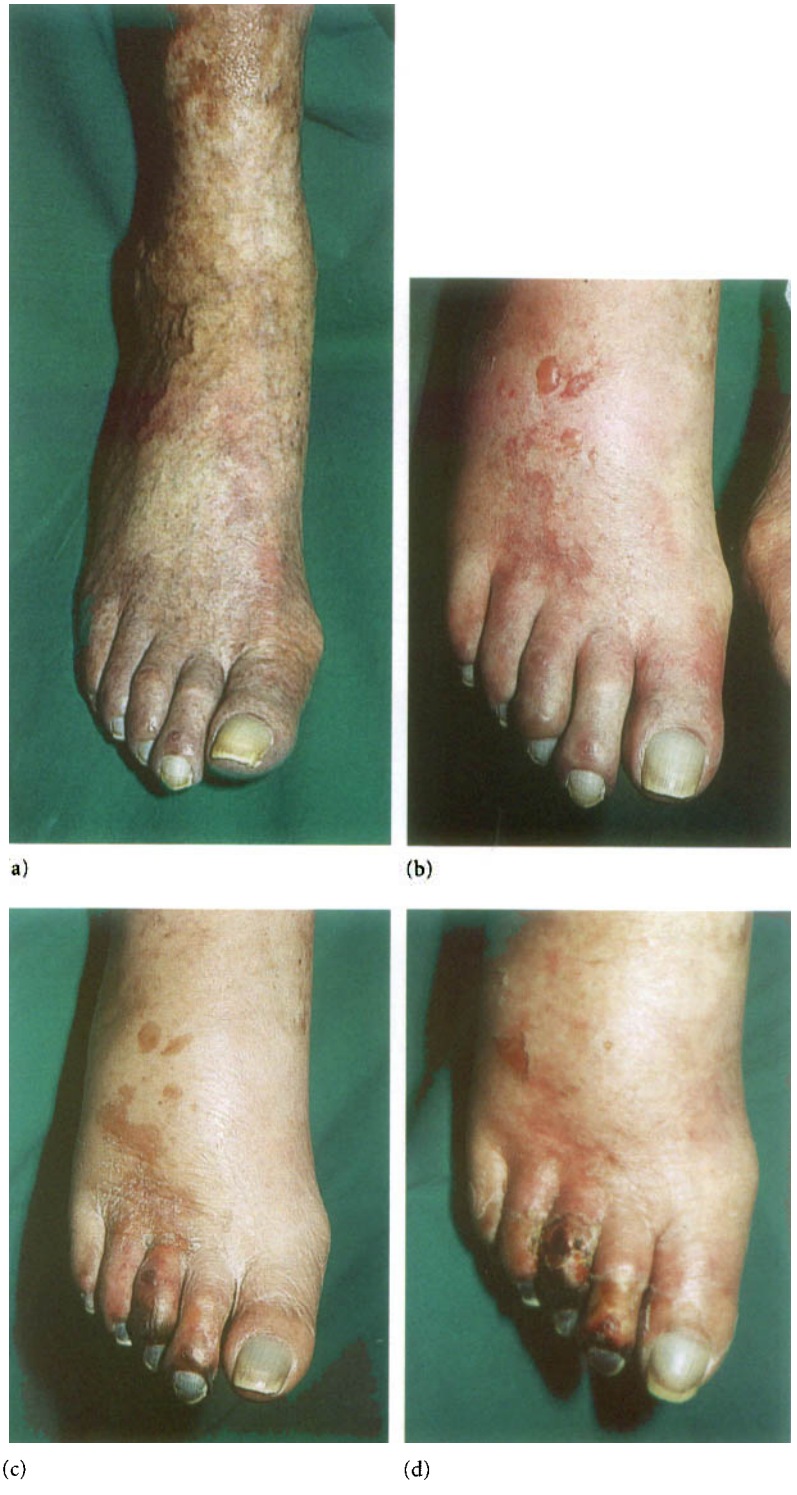


Fig. 6.6 (a) Acute ischaemia: the foot is grey, pallid and cold and needed distal bypass for limb salvage. (b) Two days after her distal bypass the foot has developed small blisters on the dorsum and the tips of the toes are discoloured. (c) Two weeks later the discoloured areas were necrotic. (d) One month after the discolouration was first noted the necrosis demarcated and stopped extending.

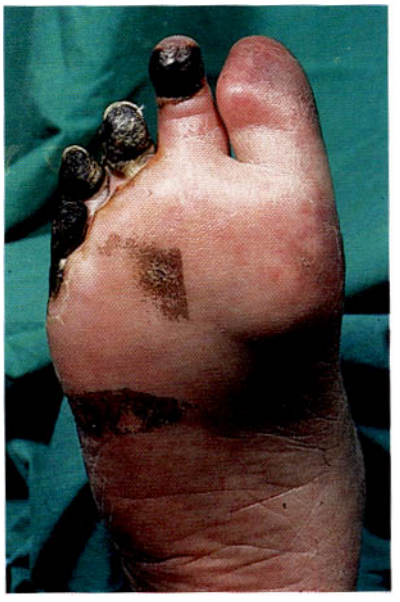


Fig. 6.7 A shower of emboli has led to necrosis of four toes.

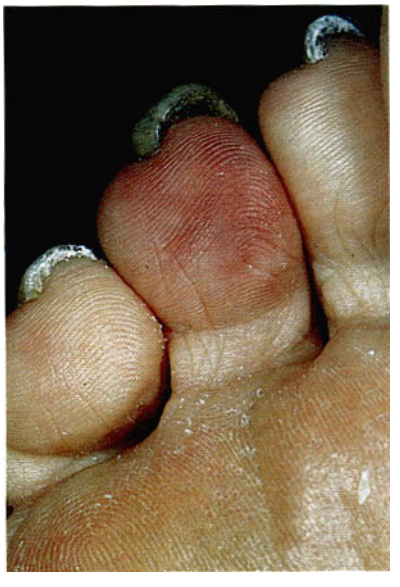


Fig. 6.8 Discrete areas of non-blanching blue discolouration on the tip of the right 3rd toe. This toe was so painful that she would not allow us to cut the nail.

vascular surgeons. She had neglected toenails which were cut except for the right 3rd which she could not bear to be touched because it was so painful. She underwent angioplasty with almost immediate improvement in her pain and her pressure index rose to 0.9. The blue areas developed superficial necrosis which healed over the next 3 months.

Key points

- Peripheral emboli present as areas of discrete bluish discolouration in one or more toes
- The origin of the emboli in this case was atherosclerotic plaques in the superficial femoral artery
- Such patients with peripheral embolic disease should receive antiplatelet therapy and vascular intervention if there is proximal arterial occlusive disease.

CASE STUDY
Emboli and palpable pulses

A 64-year-old man with type 2 diabetes of 9 years' duration was referred to the diabetic foot clinic by his general practitioner with a painful blue left hallux which did not blanch when digital pressure was applied. He worked as a porter in a meat market and was a heavy smoker who drank up to 14 units of alcohol per day. He denied any trauma to the foot, and his pedal pulses were palpable. X-ray of his foot and ultrasound of his abdomen including the aorta were normal. He was in sinus rhythm and his electrocardiogram (ECG) and echocardiogram were normal.

The toe became necrotic but was not amputated. At first it was believed that the necrosis was full thickness and that the toe would autoamputate through the interphalangeal joint, but the necrosis was more superficial than first thought. The toe was treated conservatively with outpatient debridement by the podiatrists and systemic antibiotics and aspirin, and healed in 1 year. He continued to smoke heavily despite being referred to the smoking cessation clinic.

Key points

- The source of the emboli was not clearly established. It is likely that emboli arose from the aorta or the proximal circulation of the lower limb. There was no evidence of aortic aneurysm
- Healing of necrotic digits secondary to emboli is usually protracted
- It is important to prevent infection in the necrotic toes
- Antiplatelet therapy should be given to patients with suspected emboli.

Necrosis and renal impairment

Patients with advanced diabetic nephropathy or end-

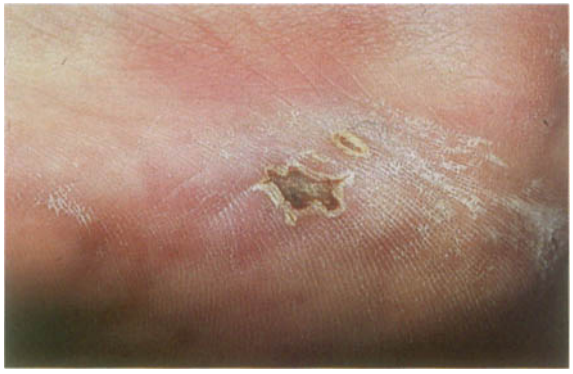


Fig. 6.9 A small split in dry skin on the border of the foot of a patient in end-stage renal failure treated with haemodialysis is becoming necrotic.

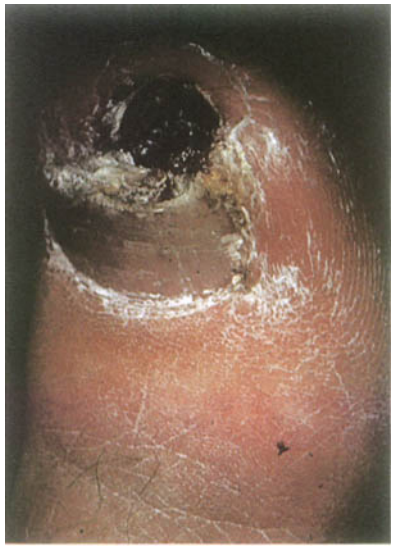


Fig. 6.10 This patch of necrosis developed on the apex of the 1st toe of a patient in end-stage renal failure treated by renal transplantation. Her pedal pulses were palpable. The patch of necrosis began as a small crack in the nail sulcus and spread very slowly to involve most of the toe, which was amputated because of severe pain. She smoked 25 cigarettes a day.

stage renal failure have an increased propensity to develop necrosis. Most have anaemia, neuropathy (which may be aggravated by uraemia) and arterial calcification. In addition, the atherosclerotic process is accelerated. The reasons for this propensity of diabetic renal patients to develop necrosis are not entirely clear.

Necrosis can occur in diabetic renal patients with palpable pulses in the absence of severe peripheral arterial disease and in the absence of infection. An apparently small and trivial trauma such as a small split in dry skin (Fig. 6.9) or a tight nail sulcus will frequently lead to necrosis which then spreads (Fig. 6.10). Necrotic lesions often become rapidly infected in diabetic patients with renal failure.

Traumatic injuries are very common in diabetic patients in end-stage renal failure (Fig. 6.11): this may be because the soft tissues of the foot are more easily damaged in end-stage renal failure, or because patients with the heavy burden of managing to cope with diabetes and renal problems become more careless about looking after their feet.

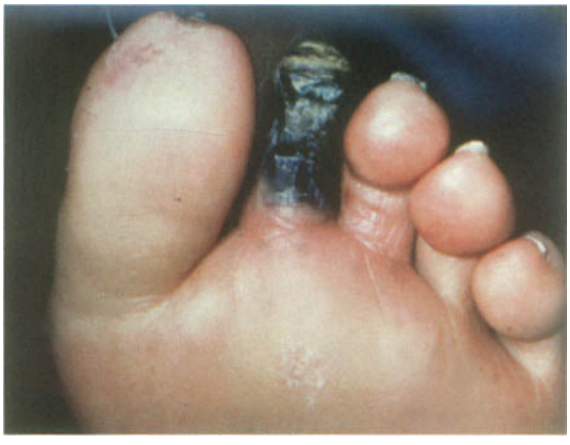


Fig. 6.11 This patient in end-stage renal failure treated with renal transplantation dropped an object on her toe and developed dry necrosis. The toe autoamputated after 13 months.

CASE STUDY

'Feckless' patient with end-stage renal failure

A 44-year-old woman with type 1 diabetes of 26 years' duration, proliferative retinopathy, profound neuropathy and end-stage renal failure treated by renal transplant had her feet checked at monthly intervals at the renal unit as part of a research protocol. Her foot pulses were palpable. She was educated in foot care, foot inspections and early reporting of any problems. However, during a 3-year period she suffered nine separate episodes of foot trauma, none of which she reported early: they were detected at her renal unit appointment. Causes of trauma included blisters from ill-fitting shoes, picking at dry skin, pulling off pieces of nail and being 'trodden on by a baby'. In the last episode she stubbed her toe while walking barefoot, did not report the injury and presented late to the renal unit with spreading cellulitis, wet necrosis and

septicaemia. She was resuscitated and treated with intravenous antibiotics and underwent 1st ray amputation to remove the source of her sepsis. Despite this, her septicaemia progressed and became overwhelming and she suffered a cardiac arrest and could not be resuscitated.

Key points

- Diabetic renal patients are susceptible to frequent and repeated traumas
- They often present late
- Infection in the renal transplant patient, in the presence of immunosuppressive treatment, can rapidly become overwhelming.

CASE STUDY

Iatrogenic necrosis in a renal foot

A 64-year-old man with type 2 diabetes of 16 years' duration, retinopathy, peripheral neuropathy, peripheral vascular disease and end-stage renal disease treated by renal transplantation. He had bilateral peripheral vascular disease and necrosis of the apices of the toes of his right foot, but his left foot was intact. The necrosis had started spontaneously but was slow to resolve and he was admitted for an angiogram. During the procedure he was thought to be hypoglycaemic, and a capillary blood sample was obtained by pricking his left second toe. Within 24 h the toe turned blue and subsequently developed full-thickness necrosis which gradually spread up the foot until it affected the entire forefoot. He refused partial amputation or major amputation, and the foot was regularly debrided (Fig. 6.12). Antibiotics were administered to treat episodes of infection and the necrotic areas remained dry and eventually separated after 2 years. The foot remained healed until he died of a myocardial infarction 6 months later.

Key points

- Blood samples should never be taken from the toes of a diabetic neuroischaemic foot
- Patients in renal failure are particularly prone to develop necrosis following an apparently trivial injury
- Once established in the toe, necrosis may spread rapidly in the forefoot.

MANAGEMENT

Whether the stage 5 foot is neuropathic or neuroischaemic it should always be regarded as a clinical emergency which should be seen by the diabetic foot service without delay and preferably the same day that it is noticed.

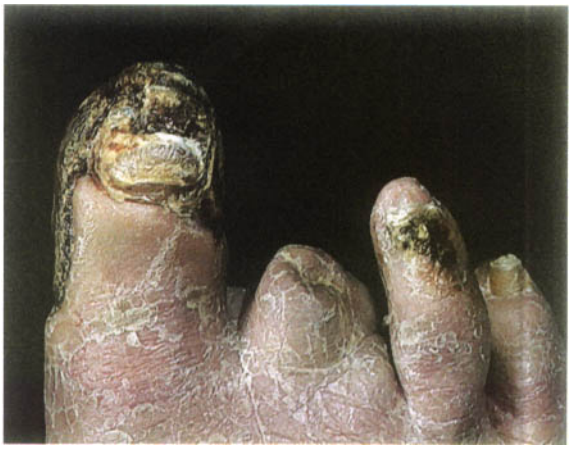


Fig. 6.12 Dry necrosis which began when the 2nd toe was pricked to obtain a blood sample. The 2nd toe has already autoamputated.

Patients with necrosis should not be treated solely in the community, by individual health-care practitioners: this is unfair both to patients and health-care professionals. However, most patients will need follow-up wound care from the community nursing team as well as the hospital.

It is important for health-care practitioners and patients to be aware that necrosis does not automatically progress to major amputation. Necrosis can often be successfully treated. However, each class of foot requires a different approach to the management of necrosis.

In the neuropathic foot, wet gangrene due to infection can be treated with intravenous antibiotics and surgery.

In the neuroischaemic foot, this approach may also be used, but when the foot is very ischaemic, revascularization should be performed if feasible. If vascular intervention is not possible, surgery should be avoided if possible, and intravenous antibiotics may be used to convert wet necrosis to dry necrosis.

Dry necrosis in the neuroischaemic foot can be successfully managed with revascularization of the foot and amputation. If vascular intervention is impossible, some cases of dry necrosis will do well and autoamputate with conservative care alone.

Patients should be admitted immediately for urgent investigations and multidisciplinary management. It is important to achieve:

- Wound control
- Microbiological control
- Vascular control

- Mechanical control
- Metabolic control
- Educational control.

Wound control

Feet at stage 5 must always be classified as neuropathic or neuroischaemic because the treatment offered will differ according to the vascular status of the foot, and treatment decisions need to be made very quickly if the foot is to be saved.

Neuropathic foot

In the neuropathic foot, operative debridement is almost always indicated for wet gangrene. The main principle of treatment is surgical removal of the necrotic tissue, which may include toe or ray amputation (removal of toe together with part of the metatarsal) or, rarely, transmetatarsal amputation.

Although necrosis in the diabetic foot may not be associated with a definite collection of pus, the necrotic tissue still needs to be removed.

CASE STUDY

Trauma, infection, necrosis and ray amputation

A 56-year-old diabetic man with type 1 diabetes of 31 years' duration and peripheral neuropathy stubbed his left hallux when walking barefoot. He was aware that the nail was damaged but felt no pain and assumed the injury was trivial. He denied ever receiving foot care education and had not attended the diabetic foot clinic. One week later he attended casualty with a necrotic hallux and cellulitis spreading up the foot (Fig. 6.13). Pedal pulses were bounding. Intravenous antibiotics were administered and he went to theatre within 24 h and underwent amputation of the first ray. There was no collection of pus, but extensive sloughy tissue. The foot healed in 10 weeks.

Key points

- In the neuropathic foot, there is good arterial circulation and the treatment of choice of wet necrosis is surgical removal
- The postoperative wound in the neuropathic foot heals as long as infection is controlled
- Diabetic neuropathic patients who are ignorant of foot care are extremely vulnerable
- We give them education and frequent follow-up appointments.

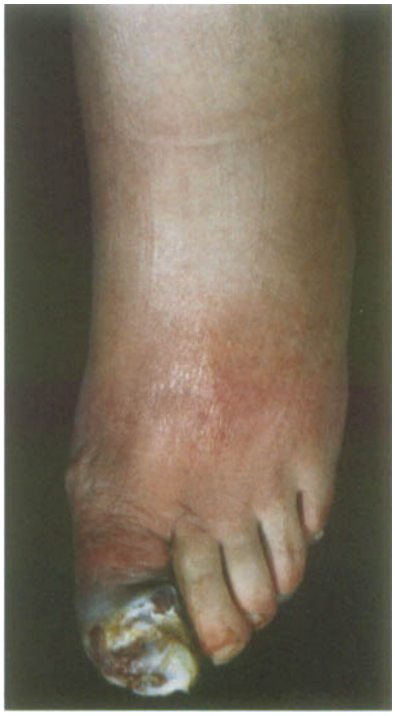


Fig. 6.13 Wet necrosis of the hallux with cellulitis spreading up the foot. There was no collection of pus but extensive sloughy tissue was present and he underwent amputation of the 1st ray.

Very occasionally, patients with neuropathic feet may not be suitable for or refuse operation, and the aim would then be to convert wet gangrene into dry by conservative treatment and intravenous antibiotics and allow autoamputation, where after a number of weeks or months the toe 'drops off' leaving a healed stump. Many debridements of necrotic tissue can be performed in the outpatient clinic by podiatrists. Diabetic patients do not require local anaesthetic by virtue of their neuropathy and the fact that the tissue being removed is not living and therefore insensitive.

Neuroischaemic foot

In the neuroischaemic foot, wet necrosis should also be removed when it is associated with severe spreading sepsis. This should be done whether pus is present or not. However, where necrosis is limited to one or two toes in the neuroischaemic foot we avoid surgery where possible until vascular intervention has been achieved. If angioplasty or arterial bypass is not possible, then a decision must be made either to amputate the toes in the presence of ischaemia or allow the toes, if infection is controlled, to

convert to dry necrosis and autoamputate. Sometimes this decision can be a difficult one. Surgical amputation leaves a large tissue defect which, in the neuroischaemic foot, may never heal. However, a transcutaneous oxygen tension of greater than 30 mmHg on the dorsum of the foot indicates a reasonable chance of healing. Autoamputation is a process which takes many months and there is always a danger that the foot may become infected if the necrotic toe is left to autoamputate.

Techniques to treat necrosis

- Outpatient debridement
- Operative surgical debridement
- Facilitated autoamputation
- Larva therapy.

Outpatient debridement

The rationale for outpatient debriding of necrosis is as follows:

- Removes wet necrosis, which is an excellent culture medium for microorganisms, thus rendering infection less likely (Figs 6.14, 6.15). Debridement enables inspection of the underlying tissues: are there pockets of pus, are the tissues well perfused, is there healthy granulating tissue underlying necrosis?

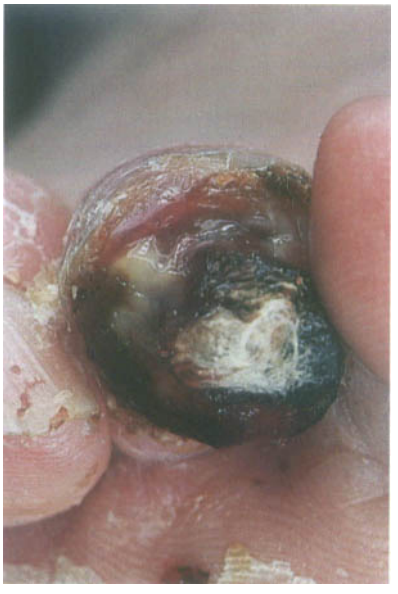


Fig. 6.14 This necrotic toe is exuding pus from along the demarcation line between necrosis and viable tissue and needs to be debrided.

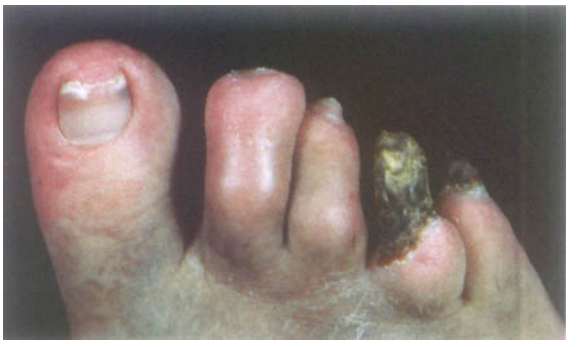


Fig. 6.15 This 4th toe is dry and well demarcated and has been debrided at 2-weekly intervals by the podiatrist—note the beautifully clean demarcation line between necrosis and viable tissue.

- Speeds healing by converting the lesion into an acute wound
- Removes a physical barrier from the edge of the wound, enabling new epithelium to grow across more easily
- The necrosis removed can be sent to the laboratory for culture and sensitivities
- Enables the true dimensions of the lesion to be seen, and in particular the depth (Fig. 6.16a,b).

The operator works from proximal to distal, away from the demarcation line, to avoid cutting into viable tisse.

Wet necrosis is grasped in forceps and gentle traction is applied so that the tissue being cut is taut, which enables greater precision (Fig. 6.17a–c).

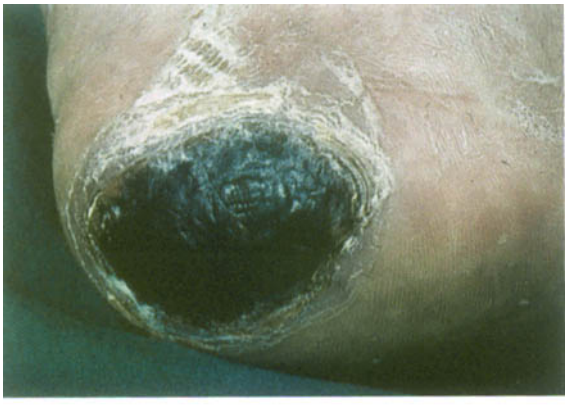
Heaped up material along the demarcation line is removed.

When necrotic material is sent for culture the surface material is first debrided away and the tissue sample is taken from deeper areas.

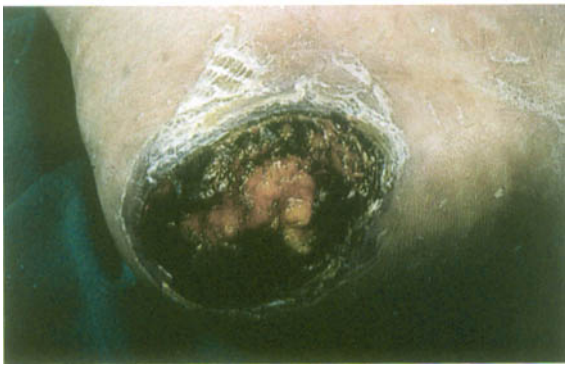
Haemodialysis patients, who undergo rapid haemodynamic changes on dialysis, are heparinized to prevent clotting of the access graft, so ulcerated and necrotic lesions may bleed on debridement around the time of dialysis.

Operative surgical debridement (neuropathic and neuroischaemic foot)

Surgical debridement or amputation should be considered if the necrotic toe or any other area of necrosis is painful or if the circulation is not severely impaired, that is, a pressure index above 0.5 or a transcutaneous oxygen tension above 30 mmHg. Postoperatively there may be a considerable tissue deficit with exposure of bone or tendon. Such deficits may be repaired by plastic reconstructive surgery.



(a)



(b)

Fig. 6.16 (a) This patient has developed dry necrosis on the lateral border of the heel but the depth of the necrosis is not clear. (b) An area of necrosis has been sharp debrided to reveal the true depth of the necrosis, but viable tissue has not been injured. In this case necrosis is superficial and can be treated by regular podiatric debridement.

Before surgery

The preparation and principles of operative debridement are similar to that described in stage 4. Patients will need:

- Full blood count and typing
- Serum electrolytes and creatinine
- Blood glucose
- Liver function tests
- Chest X-ray
- ECG.

Consent should be obtained for the most extensive debridement anticipated, including digital or ray amputation.

During surgery

It is important to remove all necrotic tissue, down to bleeding tissue, as well as opening up all sinuses. Deep necrotic tissue should be sent for culture immediately. Wounds should not be sutured. A foot with a large gaping wound following extensive tissue removal may be lightly held together by winding long strips of paraffin gauze around the foot: however, the strips should be cut through to accommodate swelling and must not prevent draining of exudate.

After surgery

In the neuropathic foot, irrigation with 2% Milton (1 in 50 dilution—see Chapter 5) may be useful for 5 days. Any Milton solution in contact with the skin should carefully be rinsed off as it has an extremely drying effect. After rinsing, emollient cream should be applied to intact skin.

Ischaemic wounds are extremely slow to heal even after revascularization, and wound care needs to continue on an outpatient basis in the diabetic foot clinic. Some feet take many months, or even years, to heal, but with patience outcomes may be surprisingly good. Even if healing is never achieved many patients prefer to live with an ulcerated foot than to undergo amputation.

Repair of tissue deficits

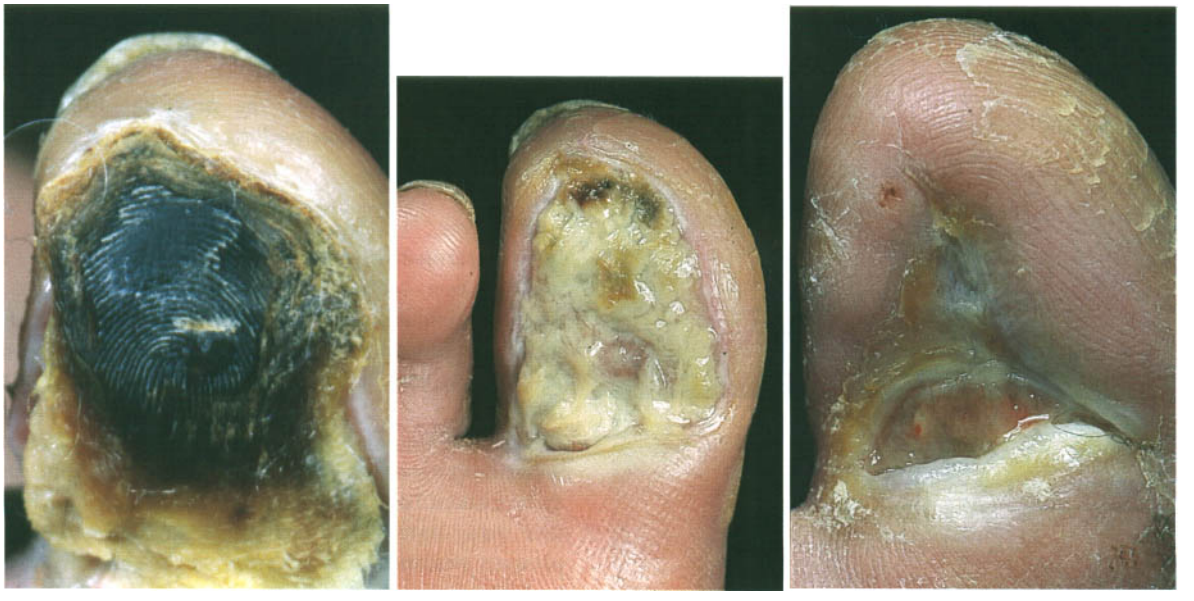
Debridement of necrotic lesions of the foot often leads to severe tissue deficits. Management of these soft tissue deficits is complex and skin grafts, local flaps and free tissue transfer have been used. Free tissue transfer is usually carried out for limb salvage and combined with arterial reconstruction in the ischaemic limb. Donor tissue from above the waist is usually used, particularly muscle flaps from the rectus abdominis or latissimus dorsi.

In a free tissue transfer, the arteriovenous pedicle accompanies the transferred tissue and is anastomosed to recipient vessels. A pedal or tibial vessel, which is either a bypass graft or a native revascularized artery, serves as the inflow tract for the free flap which is anastomosed using microsurgical techniques.

CASE STUDY

'Gas gangrene' diagnosed from culture of tissue

A 65-year-old man with type 2 diabetes of 23 years' duration and chronic ischaemia developed four necrotic toes following an episode of infection which was treated in hospital with intravenous antibiotics. Vascular intervention was not feasible and the toes were treated conservatively, with treatment consisting of pain control with liberal analgesia, oedema control with diuretics, infection control with oral antibiotics, and wound control with



(a) (b) (c)

Fig. 6.17 (a) The proximal portion of this necrotic ulcer was wet and discharging pus. (b) The same ulcer 2 weeks later after debridement of wet necrosis. (c) 10 weeks after the first debridement the toe is healing well.

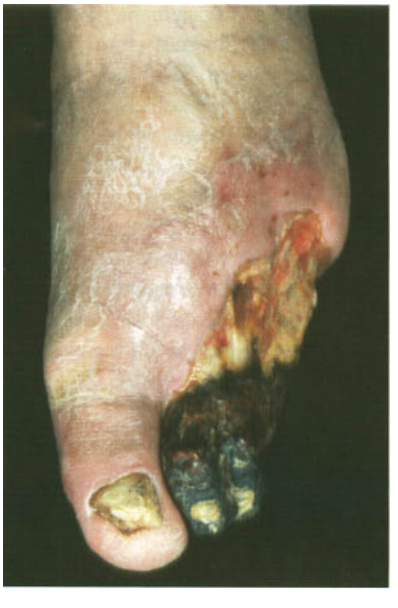


Fig. 6.18 *Clostridium perfringens*. A tissue sample was sent for culture from the necrotic area at the base of the 3rd toe.

regular debridement along the demarcation line between necrosis and viable tissue.

After 8 weeks (Fig. 6.18), a tissue sample was sent for culture and grew *Clostridium perfringens* from tissue and not from the swab. We recalled the patient to clinic: he had an X-ray which revealed gas in the tissues. He was admitted, underwent surgical debridement and was given benzylpenicillin 1.2 g qds intravenously. He was discharged after 1 week. Over the next 8 months the gangrene auto-amputated and the foot healed.

Key points

- Diabetic patients with necrosis in the foot require close follow-up in the diabetic foot clinic and intense surveillance to detect infection
- Without regular microbiological investigations this serious infection would not have been diagnosed early
- *Clostridium perfringens* is best treated by high-dose intravenous penicillin.

CASE STUDY

Ray amputation in a neuropathic foot and its postoperative care

A 58-year-old man with type 2 diabetes of 10 years' duration, presented at casualty with a cellulitic right foot and blue 5th toe (Fig. 6.19). Pedal pulses were bounding and he did not complain of pain. He was admitted for intravenous antibiotics and a ray amputation was performed to remove all necrotic and sloughy tissue. When the dressing was 'taken down' after 48 h the wound bed was grey

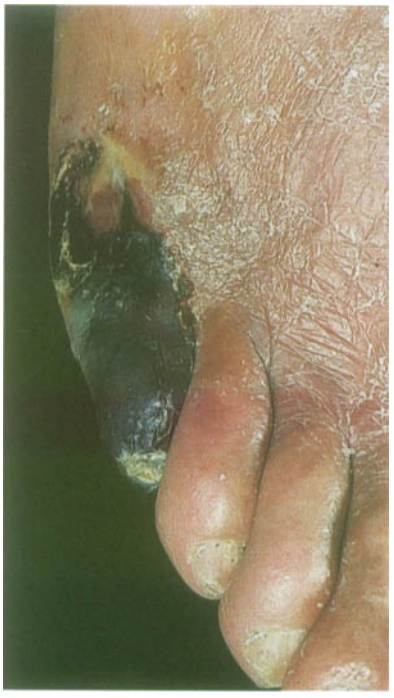


Fig. 6.19 This neuropathic patient developed a blue toe 'out of the blue', as the patient said. There was no history of trauma but a small tissue defect which was painless, and which was the portal of entry for infection.

and sloughy. The wound was irrigated with 2% Milton for 4 days until a wound bed of pink, healthy granulations was present, after which it was cleansed with saline and dressed with a foam dressing. He was discharged after 3 weeks. The foot healed in 6 weeks with minimal scarring; he received follow-up care in the diabetic foot clinic, and ulceration did not recur.

Key points

- Digital necrosis in the neuropathic limb is best treated by toe or ray amputation
- At surgery all necrotic tissue should be removed but sometimes it is difficult to be sure about the viability of all tissue at the operation site
- Thus at the first postoperative dressing, the wound may still contain necrotic and sloughy tissue
- Patients are sometimes taken back to operating theatre for the first change in dressings when further operative debridement can be carried out if necessary
- When postoperative wounds in neuropathic feet are sloughy, Milton irrigation can be used until the wound bed shows healthy granulations
- Milton irrigation does not appear to impair healing of sloughy wounds.

CASE STUDY

Toe amputation in a neuroischaemic foot

A 57-year-old man with type 2 diabetes of 6 years' duration developed a blister on his right 5th toe from a shoe rub. After 4 days the foot became painful and discoloured and he attended casualty. His pedal pulses were impalpable. He had wet necrosis of the 5th toe, oedema, cellulitis and lymphangitis spreading up the foot (Fig. 6.20a,b). He was admitted to hospital and given amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole 400 g tds and ceftazidime 1 g tds intravenously. He underwent angiography and angioplasty of occlusion of the anterior tibial artery and the toe was amputated. The foot healed in 8 weeks.

Key points

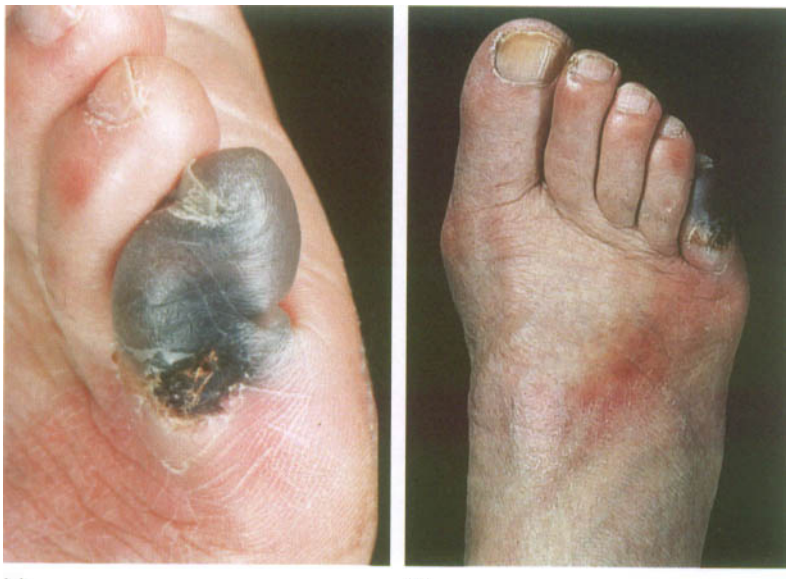
- In cases when the limb is not immediately threatened, and the necrosis is limited to one or two toes, it may be possible to control infection with intravenous antibiotics and proceed to urgent angiography and revascularization
- Angioplasty may improve the arterial circulation to allow healing of a toe or ray amputation
- If angioplasty is not possible then arterial bypass should be considered and the toe or ray amputation can be performed at the same time as bypass.

CASE STUDY

Large tissue deficit in a neuroischaemic foot secondary to infection needing distal arterial bypass

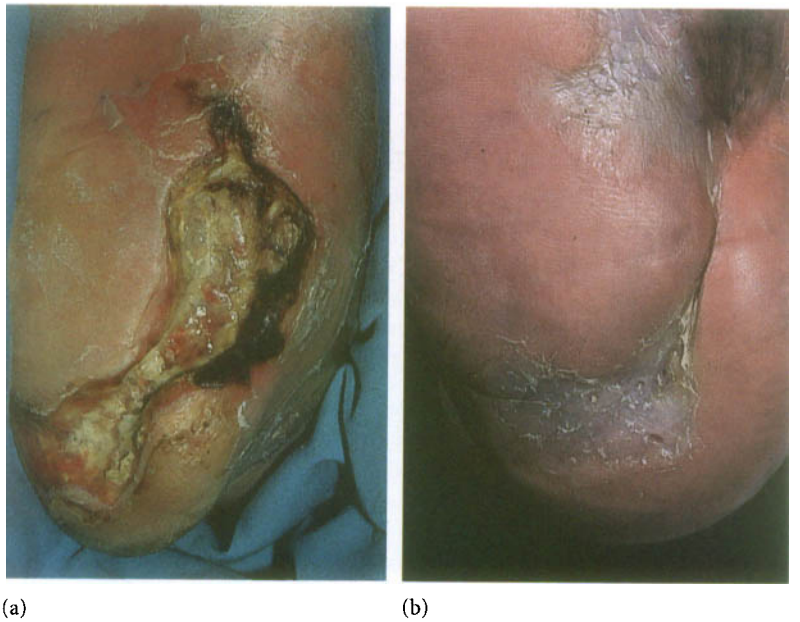
A 43-year-old male with type 1 diabetes of 27 years' duration, with peripheral and autonomic neuropathy, was referred with indolent neuropathic ulceration complicated by local cellulitis over the left 5th metatarsal head. His pedal pulses were palpable. He was treated with oral amoxicillin 500 mg tds and flucloxacillin 500 mg qds and outpatient debridement. His deep wound swab had grown *Staphylococcus aureus* and *Streptococcus* group G. The cellulitis resolved and he was given a total-contact cast. The ulcer healed after 8 weeks and he was given bespoke shoes with cradled insoles.

Two years later he was admitted with an infected ulcer on the plantar surface of the right heel where he had pulled off a piece of loose skin (Fig. 6.21a). His pedal pulses were now impalpable. He was admitted and given intravenous vancomycin 1 g bd, ceftriaxone 1 g bd and



(a) (b)

Fig. 6.20 (a) Wet necrosis of the 5th toe. (b) Lymphangitis spreading up the same foot.



(a) (b)

Fig. 6.21 (a) Infected ulcer on plantar surface of heel. (b) The ulcer has healed following application of Apligraf.

metronidazole 400 mg tds. He had recently had a methicillin-resistant *Staphylococcus aureus* (MRSA) infection on the contralateral foot. Angiography showed occlusions of the right common iliac artery and superficial femoral artery. It was planned to perform an angioplasty of the right common iliac and right external iliac as a crossover procedure via the left femoral artery. However, whilst awaiting this the patient developed a critically ischaemic right foot. Urgent angiogram at this time showed thrombus in the right superficial femoral artery. He underwent

thrombolysis. A check angiogram showed a patent superficial femoral artery, but significant stenoses in the popliteal artery with a good two-vessel run-off. He underwent a popliteal angioplasty. Following this, transcutaneous oxygen tension was 57 mmHg on the chest and 69 mmHg on the dorsum of the right foot. This was deemed adequate perfusion and no further vascular intervention was attempted. The ulceration on the right heel remained clean and a vacuum-assisted closure (VAC) pump was added to improve granulation.

The patient was followed up in the diabetic foot clinic and the ulcer had almost healed after 4 months. Despite careful education about the danger signs of deterioration he then presented very late with chills, sweating, infection of the heel ulcer and blue discolouration of the medial aspect of the right foot. He had spreading cellulitis and a 2-cm area of necrosis on the medial aspect of the right foot. Doppler waveforms were monophasic and damped. The working diagnosis was that he had mid-foot sepsis likely to be tracking from the ulcer and he underwent operative surgical debridement. There was a track leading from the heel ulcer along the extensor tendons to the mid-foot and this was laid open. All dead and infected tissue was excised back to bleeding tissue. Deep tissue culture revealed MRSA and mixed anaerobes. Clinically, he was septic and was treated with vancomycin 1 g bd, rifampicin 600 mg bd (as an adjunctive treatment for MRSA), metronidazole 500 mg tds and Milton irrigation to the wound. In view of his sepsis he was also given gentamicin 5 mg/kg daily. Angiography showed further stenoses of the superficial femoral and popliteal artery.

Initially it was planned to carry out an angioplasty, but it was decided that distal bypass surgery had the best chance of restoring the pulsatile blood flow which was necessary to heal his large tissue deficit. He underwent a distal bypass from the right common femoral artery to the anterior tibial artery with a reversed lower saphenous vein. The reversed lower saphenous vein was tunnelled laterally to the knee subcutaneously. There was a good quality common femoral artery and a non-calcified anterior tibial artery in mid-shin of good calibre. The bypass was successful but the plantar wound was slow to heal.

His postoperative course was stormy. He developed a fever with productive cough associated with rigors and vomiting. He was treated with vancomycin 1 g bd and meropenem 1 g tds intravenously empirically and improved. He complained of back pain and X-ray showed evidence of vertebral collapse. He had magnetic resonance imaging (MRI) of his spine as a metastatic abscess was suspected. However, the MRI showed changes consistent with a haemangioma but this did not require neurosurgical intervention. An application of Apligraf finally healed the ulcer (Fig. 6.21b).

During this admission his wife visited the hospital every day, arriving early in the afternoon and leaving late in the evening. She took a great interest in his care and learned to clean and dress the foot. She was taught the danger signs of deterioration. Subsequently he never again presented late with severe infection because his wife checked his feet every day and brought him to the diabetic foot clinic at the first sign of a break in the skin.

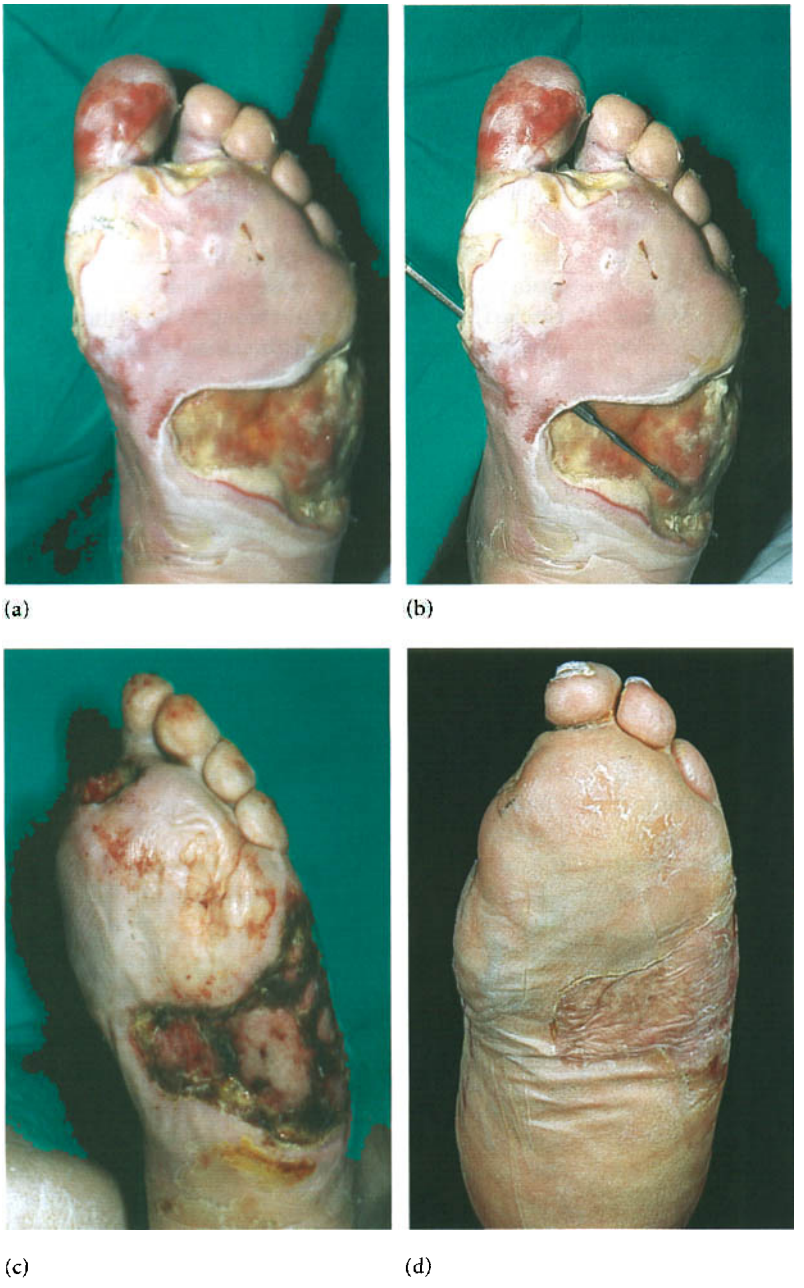
Key points

- Patients admitted with severe infection need intense multidisciplinary care and frequent medical and surgical review
- Surgical debridement was needed on his second admission because of necrosis secondary to infection
- An arterial bypass was needed to restore pulsatile flow to heal a large tissue defect. The increase in blood flow after angioplasty may not have been sufficient
- The family of vulnerable patients should be taught to check the feet daily and respond rapidly at the first sign of a break in the skin
- Neuropathic feet eventually become neuroischaemic but this will need to be diagnosed by examination of the foot pulses at every visit
- Patients with neuroischaemic foot ulceration need long-term follow-up in a multidisciplinary diabetic foot service.

CASE STUDY

Necrotizing fasciitis in a previously neuropathic foot that had become neuroischaemic

A 67-year-old man with type 2 diabetes of 22 years' duration and a previous history of frequent neuropathic ulceration, developed fever, rigors, fatigue and malaise, and asked his general practitioner to visit him. Influenza was diagnosed and paracetamol was prescribed. The general practitioner called again, 2 days later, when the patient had not improved, and told him to continue taking paracetamol. Two days later the patient's wife became aware of an unusual odour, checked her husband's feet, and found that his hallux was purple and a large ulcer had developed on the side of his foot. Same day admission was arranged. The patient had extensive wet gangrene and necrotizing fasciitis of the plantar aspect of the foot (Fig. 6.22a,b). It was noted that the foot pulses were not palpable and



(a) (b) (c) (d)

Fig. 6.22 (a) The hallux is red and purple at its tip. There is a large area of wet necrosis and sloughing of tissue on the lateral border of his foot. (b) It is possible to pass a probe from the medial to the lateral border of the foot. (c) The large tissue defect has been surgically debrided and received a split-skin graft which has taken: the hallux has been amputated. (d) The 2nd toe has also developed necrosis and autoamputated. The foot has healed.

Doppler studies disclosed that the patient, previously neuropathic, had become neuroischaemic.

Extensive debridement of infected soft tissue and amputation of the hallux was performed as an emergency procedure leaving him with a large tissue deficit. *Staphylococcus aureus* and *Pseudomonas* was grown from the tissue. He was treated with ceftazidime 1 g tds and flucloxacillin 500 mg qds. He underwent a distal arterial bypass. His postoperative progress was slow and the tissue defect was covered with a split-skin graft (Fig. 6.22c,d). After

discharge he attended the diabetic foot clinic at monthly intervals for sharp debridement of callus from the skin graft.

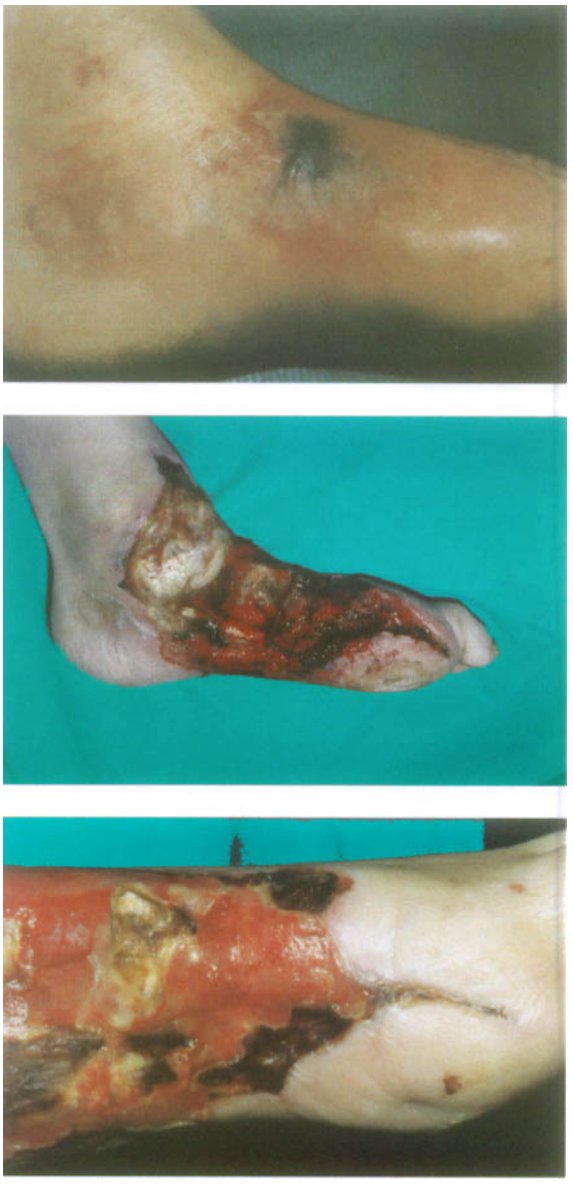
Key points

- Diabetic foot infections may masquerade as influenza
- Patients who cancel their appointments because of 'flu-like symptoms' should have their feet checked
- Our education programme specifically warns patients that symptoms of flu may be caused by foot infections
- Soft tissue destruction in the neuroischaemic foot is usually due to infection
- Always be on the outlook for neuropathic feet that have become neuroischaemic
- Extensive tissue loss in the neuroischaemic foot should be managed ideally with an arterial bypass.

CASE STUDY

Extensive debridement of severe sepsis in a neuroischaemic foot

A 66-year-old man with type 2 diabetes of 10 years' duration went to casualty complaining of a swollen foot with a small purple area on the medial border. His foot pulses were impalpable and his pressure index was 0.7. He was unwell with pyrexia of 39°C and had rigors. He was taken to operating theatre for debridement. Although the area of non-viable tissue appeared to be not more than 3 cm in diameter (Fig. 6.23a), surgical debridement revealed very extensive tissue destruction involving subcutaneous tissues and bones. He underwent excision of the 1st toe, 1st metatarsal, medial cuneiform and navicular, and large areas of skin and soft tissue. Apparently healthy overlying skin covered a layer of necrosis which had tracked between skin and subcutaneous tissues like the filling of a sandwich. One week later the wound was not granulating (Fig. 6.23b). He did not want a bypass but agreed to undergo angioplasty of stenoses in his popliteal artery and tibioperoneal trunk, after which the wound granulated well (Fig. 6.23c). He was discharged after 3 months for shared care of his healing wound between community nurses and the diabetic foot clinic. However, he died of a myocardial infarct the same day.



(a)

(b)

(c)

Fig. 6.23 (a) Cutaneous necrosis on the medial surface of foot indicating severe underlying tissue destruction. (b) One week postoperative this wound is not granulating. (c) Following angioplasty the wound is granulating well 6 weeks later.

Key points

- Surface appearances can be very deceptive: the visible area of discolouration is usually just the tip of the iceberg
- Apparently healthy skin can cover extensive necrosis
- It is often only when surgical debridement is performed that the true extent of necrosis is understood
- If the pressure index is low and the wound is not granulating vascular intervention shoud be carried out
- It may take several weeks for the optimal effects of an angioplasty to take effect and granulation of the wound to take place.

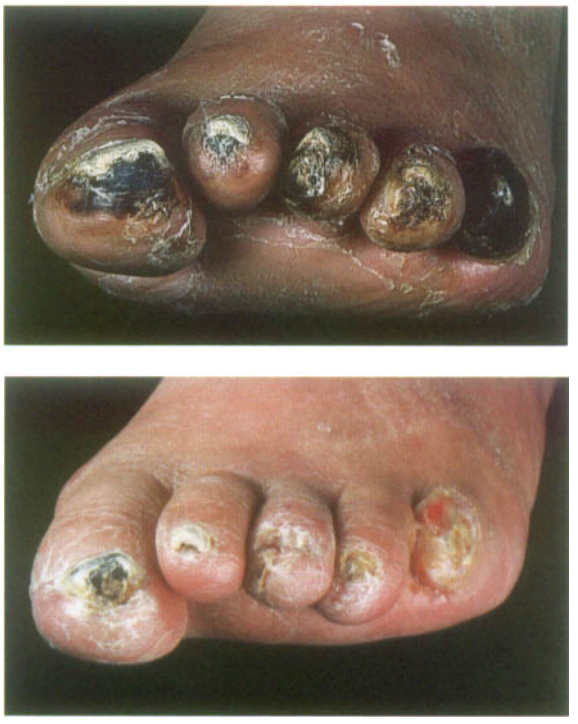


Fig. 6.24 (a) This lady with a necrotic 5th toe and necrotic apices of the 1st, 3rd and 4th toes underwent podiatric debridement. (b) The same foot 6 weeks later after regular 2-weekly debridements by the podiatrist.

Facilitated autoamputation

Careful sharp debridement is performed along the demarcation line between necrosis and viable tissue to debulk dead tissue, drain pockets of pus and prevent accumulation of debris (Fig. 6.24a,b). Scalpel and forceps are used: if necrotic material is moist then traction should be applied with the forceps to enable precise cutting with the scalpel. If tension is not applied it will be impossible to clear away the moist necrosis and the operator is in danger of cutting the patient.

If areas of the necrotic toe are moist and the necrosis is full thickness (deep to bone) then the necrotic portion may be best removed by amputating it through the interphalangeal joint distal to the demarcation line between necrosis and viable tissue. The removed necrotic apex can be sent to the laboratory for culture. Once this procedure has been performed it is easier to debride necrotic material from around the stump without damaging viable tissue. The patient should always be warned and consulted if this procedure is to be attempted.

We remember one patient who watched with interest while his necrotic toe was amputated and asked if he could take it home. When asked why he wanted it he explained that he was meeting friends at the pub that evening and intended to drop the toe into someone's beer. The diabetic foot clinic staff confiscated the offending toe!

CASE STUDY

Autoamputation of a neuroischaemic necrotic digit

A 65-year-old man with type 2 diabetes of 12 years' duration was referred to the diabetic foot clinic with a dry necrotic 4th toe (Fig. 6.25a). He was admitted for vascular assessment but no intervention was possible so the necrosis was treated conservatively in the belief that surgical removal would leave a large defect which would be difficult to heal in an ischaemic foot. His transcutaneous oxygen tension was 25 mmHg. He was given oral antibiotics, regular debridement along the demarcation line between gangrene and viable tissue and insulin for optimal control of his diabetes. The toes were dressed with Melolin as a non-stick dressing and pieces of gauze were placed between them to separate the gangrenous toe from its neighbours, and the community nurses visited him every day.

After 5 months the gangrenous toe autoamputated to reveal a healed stump (Fig. 6.25b).

Key points

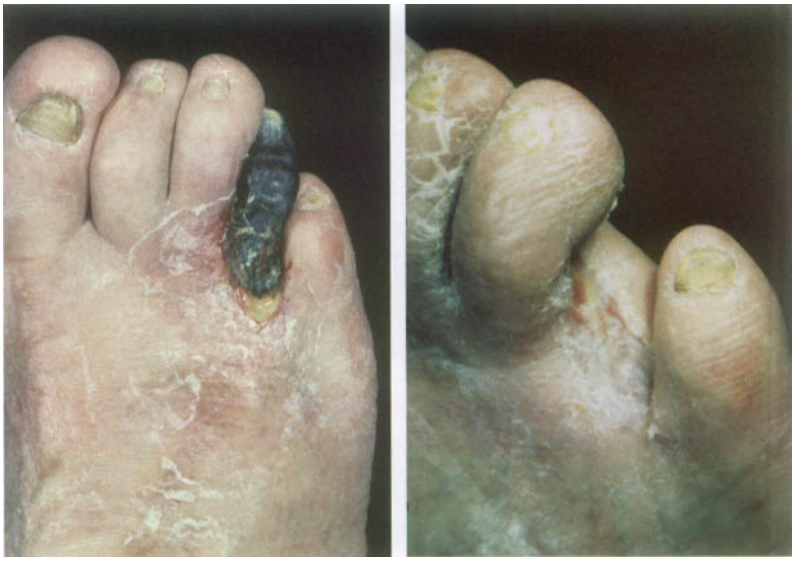
- When it is not possible to revascularize the limb, necrotic toes may be managed conservatively by autoamputation
- We felt that the existing arterial perfusion, as reflected in the transcutaneous oxygen tension of 25 mmHg, was sufficient to allow successful autoamputation but might not enable a surgical amputation wound to heal.

Larva therapy

Maggot therapy can be used to debride necrotic tissue and slough in stage 5 feet. The larvae used are those of the green bottle fly.

Medical maggots of *Lucilia sericata* (the larvae of the green bottle fly) may assist in the eradication of infection by ingesting and digesting bacteria, including MRSA, and infected, sloughy tissue. Because they feed on dead flesh and not on living flesh they are sometimes used to debride infected slough or necrotic tissue from ulcers, particularly in the neuroischaemic foot.

The maggots should be well contained within special bags provided by the 'maggot farm'. These are used to



(a) (b)

Fig. 6.25 (a) Necrotic neuroischaemic toe. (b) Autoamputation: the toe has dropped off to reveal a healed stump.

enclose the foot or part of the foot. The bag should be covered with dressings to help absorb exudate, but the maggots may drown or suffocate unless dressings are loosely applied.

Patients should be on bed rest. If the patient is allowed to walk the maggots may be crushed.

The normal skin around the wound should be masked with zinc oxide bandage to prevent the skin being affected by digestive enzymes produced by the maggots which will otherwise make it red and raw.

Copious amounts of thin discharge, which is often a rusty brown colour, and a 'fusty' smell are associated with wounds containing maggots, and patients and other members of staff should be forewarned about this.

Most patients are not bothered by the movements of the maggots within the wound, but ischaemic patients occasionally complain of increased pain during maggot therapy.

The maggots can be irrigated out of the wound on the fourth day.

CASE STUDY
Larva therapy

A 52-year-old patient with type 1 diabetes mellitus for 30 years, retinopathy, nephropathy, peripheral neuropathy and peripheral vascular disease underwent amputation of his left hallux following an episode of infection. The skin defect 2 months after surgery contained abundant sloughy necrosis which was very difficult to grasp with forceps and to sharp debride with a scalpel. Immature sterile larvae of the green bottle fly were obtained from a medical maggot farm and applied to the wound, which was then covered with gauze moistened with saline. The skin adjoining the wound was masked with zinc oxide bandage to prevent irritation from enzymes produced by the maggots and the foot and lower leg were enclosed within a nylon mesh bag to prevent the maggots from escaping on to the ward.

The foot was checked every day and the overlying saline gauze replaced if it became soiled. By day 2 the maggots were very active in the wound. By day 3, copious amounts of thin, light reddish-brown exudate was being produced and the absorbent dressings were replaced with fresh ones. The maggots were still very active. On day 4 a 'honeycomb' effect was seen; the maggots were less active and their heads were down feeding at the wound interface. On day 5 the maggots had stopped feeding and the 'honeycomb' effect was no longer seen. The maggots were then irrigated out of the wound using a forceful jet of normal saline squeezed from a litre bag.

All the slough had been eaten by the maggots. The wound bed was mostly healthy with one small sinus deep to bone, the presence of which had not been apparent before. The true dimensions of the lesion could now be seen. The foot healed in 4 months.

Key points

- When slough is slippery and slimy, maggot debridement may be easier than sharp debridement with forceps and scalpel
- Maggots may reveal hidden depths to wounds
- Maggots can remain in the wound until they have finished feeding, but should be inspected regularly and will usually be removed on the fourth day
- Where podiatrists are unavailable, maggots may be useful for debriding infected or ischaemic wounds.

Dressing and cleaning the necrotic foot

Simple, dry, non-adherent dressings should be used. The aim is always to keep the necrosis dry and well demarcated, and to inspect the foot on a daily basis so that any new infection can be detected and treated appropriately.

It is important to separate necrotic toes from their neighbours. Dry sterile dressings are placed between the toes to keep them apart because if necrosis is in direct contact with viable tissue it can become moist, develop infection and spread to adjoining, previously healthy, tissue. Areas of the foot which are not involved with necrosis can be washed every day with warm water and mild soap, and dried carefully. Necrotic areas should be kept dry and covered with dressings.

Many chemists sell 'cast protectors' which are strong plastic bags in the shape of a leg that keep casts or bandages dry so that patients with necrosis may bathe or shower.

Necrotic toes should be kept dry.

CASE STUDY

Importance of keeping necrosis dry

A 76-year-old woman with type 2 diabetes of 7 years' duration, developed necrosis of her right 4th toe. She was neuroischaemic with a pressure index of 0.4, and no vascular intervention was possible. The necrosis was treated conservatively and her daughter was asked to keep the toe dry and to ensure that dressings were applied to separate the necrotic toe from its neighbours. However, her daughter allowed her to have a bath, and did not separate the toes with dressings. When she attended the diabetic foot clinic 1 week later, the necrosis was moist and necrosis was also present in an adjoining toe where it had been in contact with the necrotic toe (Fig. 6.26).

She was admitted to hospital and received intravenous antibiotics. The pressure index had not fallen. The necrosis became dry and well demarcated again and spread no further. Seven months later the toe separated to reveal a healed stump.

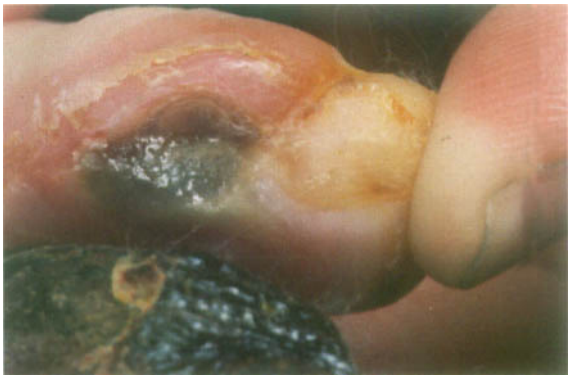


Fig. 6.26 No dressing was placed between the toes and necrosis has spread to the portion of the previously healthy toe which was in contact with the necrotic toe. A necrotic toe should always be separated from its healthy neighbours by a dry dressing placed between the toes.

Key points

- Patients should not immerse their necrotic toes in the bath. Moistening necrosis may encourage infection
- Furthermore, if a necrotic toe is in direct contact with a viable toe, the necrotic toe may 'absorb sweat' and become moist. The area of moist necrosis is an excellent culture medium for bacteria which then spread from the necrotic toe to the adjoining toe
- If the interdigital area is macerated it is no longer a barrier to bacteria which may enter the viable toe and cause ulceration and necrosis
- Neuroischaemic feet with dry necrosis may remain at stage 5 for many months until the necrotic toe drops off to reveal a healed stump.

Microbiological control

Wet necrosis

The microbiological principles of managing wet necrosis are similar to those for the management of infection of the foot with extensive soft tissue infection or the foot with blue discolouration as described in Chapter 5. When the patient initially presents, deep wound swabs and tissue specimens are sent off for microbiology. Deep tissue taken at operative debridement must also go for culture.

Intravenous antibiotic therapy

Both neuropathic and neuroischaemic patients need parenteral therapy. They are admitted to hospital and given intravenous antibiotic therapy as follows: amoxicillin 500 mg tds, flucloxacillin 500 mg qds, metronidazole

500 mg tds and ceftazidime 1 g tds. For patients who are allergic to penicillin we substitute erythromycin 500 mg qds, vancomycin 1 g bd or clindamycin 600 mg qds for amoxicillin and flucloxacillin. We use this regime because these infections are often polymicrobial. Other regimes are in use including:

- Ciprofloxacin with clindamycin
- Piperacillin/tazobactam
- Ampicillin/sulbactam
- Ticarcillin/clavulanate
- Meropenem.

Intravenous antibiotics can be replaced with oral therapy after operative debridement and when infection is controlled. On discharge from hospital, oral antibiotics are continued and reviewed regularly in the diabetic foot clinic. When the wound is granulating well and swabs are negative then the antibiotics are stopped.

It is often difficult to have all wet necrosis removed surgically. The foot may be ischaemic, and the patient may not be fit for an operation. In these circumstances we believe that it is best to leave the necrosis and convert wet necrosis to dry necrosis using antibiotics and allow it to separate. The presence of necrosis will increase the risk of infection: however, these patients are under very close surveillance and early signs of wet necrosis are carefully sought for.

Dry necrosis

When dry necrosis develops secondary to severe ischaemia, antibiotics should be prescribed if discharge develops, or the deep wound swab or tissue culture is positive, and continued until there is no evidence of clinical or microbiological infection.

When toes have gone from wet to dry necrosis and are allowed to autoamputate, antibiotics should only be stopped if the necrosis is dry and mummified, the foot is entirely pain free, there is no discharge exuding from the demarcation line, and swabs are negative.

In severely ischaemic feet (pressure index < 0.5) antibiotics may sometimes be continued until healing.

Daily inspection is essential. Regular deep swabs and tissue should be sent for culture and antibiotics should be restarted if the demarcation line becomes moist, the foot becomes painful, or swabs or tissue cultures grow bacteria.

Vascular control

All neuroischaemic feet that present with necrosis must have Doppler studies to confirm ischaemia. This should be followed by non-invasive investigations as described in Chapter 3.

The patient can have either duplex angiography or magnetic resonance angiography to show stenoses or occlusions of the arteries of the leg, particularly in the tibial arteries (Fig. 6.27a,b).

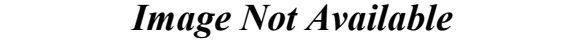


Fig. 6.27 (a) There is diffuse atheromatous disease of both superficial femoral arteries with an area of focal narrowing at the mid-level on the right. Both popliteal arteries are severely diseased. (b) On the right there is a single vessel, the anterior tibial, extending to the level of the plantar arch and it is severely diseased in the proximal aspect. There is reconstitution of posterior tibial at ankle level. On the left there is an occlusion of the tibioperoneal trunk with collaterals filling the more distal anterior and posterior tibial arteries. (Courtesy of Dr Huw Walters.)

Having diagnosed the site of disease, then revascularization can be planned.

In wet necrosis, revascularization is necessary to heal the tissue deficit after operative debridement. In dry necrosis, which occurs in the background of severe arterial disease, revascularization is necessary to maintain the viability of the limb.

Angioplasty

In some patients, increased perfusion following angioplasty may be useful and this will result in an improvement in the ischaemic wound. Indeed, this is often the only interventional procedure that can be performed as the patient may be too frail to undergo peripheral vascular surgery.

CASE STUDY

Angioplasty and delayed healing until removal of sequestrum in a neuroischaemic foot

A 91-year-old lady with type 2 diabetes of 20 years' duration who lived in a nursing home was referred as an emergency with a painful oedematous, ischaemic left foot complicated by severe cellulitis and lymphangitis and a malodorous ulcer on the apex of her 2nd toe. A swab grew mixed coliforms. The 5th toe had a bluish tinge. Her Doppler waveforms were severely damped and her pressure index was 0.3. She was admitted for wide-spectrum intravenous antibiotics and underwent angioplasty of the superficial femoral artery and anterior tibial artery. Within 3 days of the procedure the pain had greatly diminished, but the blue area of the toe was necrotic. She was followed up in the diabetic foot clinic and was given extra-depth shoes and regular debridement every 2 weeks. After 5 months an area of loose bone was evident in the wound. The bone came away to reveal complete healing of the wound after 10 months.

Key points

- Advanced age is not a contraindication to vascular intervention
- Angioplasty can be routinely and safely carried out in elderly patients who might not be fit for vascular surgery
- Improved pain is a good mark of clinical progress
- Bony sequestrum in a wound delays healing and should be removed.

Arterial bypass

Angioplasty rarely restores pulsatile blood flow, however, unless a very significant localized stenosis in iliac or femoral arteries has been successfully dilated. When the limb is severely ischaemic and there is considerable tissue deficit, it is necessary to restore pulsatile blood flow. This is best achieved by arterial bypass.

Preoperative assessment

Patients will have cardiovascular disease and need preoperative assessment to maximize cardiorespiratory status. Cardiovascular risk should be carefully assessed from the history, physical examination, functional capacity and resting ECG. An estimation of low risk vs. high risk can be made from these assessments.

When patients are of an intermediate risk then extra preoperative tests may be needed, including echocardiography, which will identify patients with left ventricular dysfunction. A low ejection fraction (less than 35%) increases the risk of non-cardiac surgery.

A useful non-invasive evaluation is perfusion nuclear imaging with thallium. Risk is increased if two or more reversible perfusion defects are present on thallium imaging. For patients with diabetes the most significant independent predictors of postoperative death are advanced age, resting electrocardiographic abnormalities and abnormalities revealed on thallium imaging.

Perioperative care

The perioperative use of β-blockers, particularly bisoprolol, is now established in patients undergoing vascular surgery. Studies using thallium reperfusion imaging as an indicator of abnormal cardiac function have shown a significant reduction in perioperative and in-hospital mortality after vascular surgery in patients randomized to bisoprolol compared with placebo. This persisted throughout the treatment period of 6 months.

Peripheral arterial disease is common in the tibial arteries, and distal bypass with autologous vein has become an established method of revascularization. A conduit is fashioned from either the femoral or popliteal artery down to a tibial artery in the lower leg, or the dorsalis pedis artery on the dorsum of the foot.

Patency rates and limb salvage rates after revascularization do not differ between diabetic patients and non-diabetic patients, and a more aggressive approach to such revascularization procedures should be promoted. Arterial bypass can be successfully carried out in patients with severe renal impairment and this should not be a contraindication.

Postoperative care

Postoperatively, the leg has wounds both where the graft has been inserted and from where the vein has been harvested. Wounds overlying the arterial graft must be kept

free from infection otherwise the graft will block. Such wounds need regular cleaning and covering with dry sterile dressings, and any associated necrotic tissue which becomes bulky or moist should be gently debrided. Postoperative oedema is common and treatment with elevation is important. The patient should enter a graft surveillance programme.

CASE STUDY
Complicated leg wounds following distal bypass surgery

A 76-year-old lady with type 2 diabetes of 30 years' duration underwent a distal bypass for critical ischaemia. Three days later the proximal area of her leg wound developed bluish discolouration (Fig. 6.28a). The next day it began to break down (Fig. 6.28b) and then dehisced, revealing an area of yellow slough (Fig. 6.28c). Wide-spectrum antibiotics were prescribed. The area dried out and formed a dark brown eschar which stood proud of the area of skin. The vascular surgeon agreed that if debris accumulated in this area it should be gently debrided in the diabetic foot clinic and this was done weekly on three occasions.

When she next came to the foot diabetic clinic for her routine weekly appointment, and the dressing was taken down, the eschar on the leg was seen to be gently vibrating at the same rate as the patient's pulse. The patient was taken to theatre for an emergency procedure. The artery underlying the eschar ruptured as she was being lifted onto the operating table: even though the surgeons were fully prepared there was considerable blood loss. The artery was repaired and the leg healed in 4 months (Fig. 6.28d).

Key points

- Pulsating wounds of distal bypass surgery should be referred as an emergency to the vascular surgeon
- Patients whose bypass leg wounds become infected are at great risk of losing the graft
- Outpatient debridement of leg wounds should be performed with great caution
- Leg wounds from distal bypasses should be inspected weekly until fully healed.

CASE STUDY
Septic arteritis, double ray amputation, distal bypass and skin grafting

A 71-year-old diabetic man with type 2 diabetes diagnosed when he attended casualty with gross foot sepsis, neuropathy and peripheral vascular disease, underwent a double ray amputation for immediate limb salvage (Fig. 6.29a,b). This was followed by a distal arterial bypass. One week later the foot wound was debrided in the diabetic foot clinic and a split-skin graft was applied 2 days later in theatre (Fig. 6.29c) from a donor site on the patient's calf which was infiltrated with local anaesthetic through a spinal needle. He did not complain of pain from the donor site which was within the distribution of his neuropathy. On discharge he agreed to use a wheelchair but declined antibiotics and had a subsequent admission for infection which resulted in amputation of the 5th toe. The foot healed in 1 year (Fig. 6.29d).

Key points

- Infection is often responsible for tissue damage in the neuroischaemic foot and surgical debridement as an emergency may be necessary to remove necrosis
- Urgent vascular investigations should then take place to prepare for revascularization
- Wound closure may be achieved with skin grafts
- Skin grafts can be taken from within the distribution of the neuropathy to avoid general anaesthetic and postoperative pain
- Infection will destroy a skin graft and delay wound healing
- Following bypass or angioplasty, regular vascular review is essential to detect deterioration early.

Investigation of patients with emboli

When dry necrosis is secondary to emboli, a possible source should be investigated, and therefore the following investigations should be performed:

- ECG to detect atrial fibrillation or recent myocardial infarct
- Echocardiogram to detect the presence of valvular disease or thrombus in the left ventricle
- Ultrasound of abdomen to detect aortic aneurysm
- Duplex angiography of the lower limbs to detect atherosclerotic plaque in iliac or femoral arteries.

Having located the source of the emboli, appropriate treatment can be given. In general, antiplatelet therapy with aspirin is the usual treatment.

Mechanical control

During the peri- and postoperative period, bed rest is essential with elevation of the limb to relieve oedema and afford heel protection. Prophylaxis of deep vein thrombosis should be carried out using a low molecular weight heparin subcutaneously daily. Low molecular weight

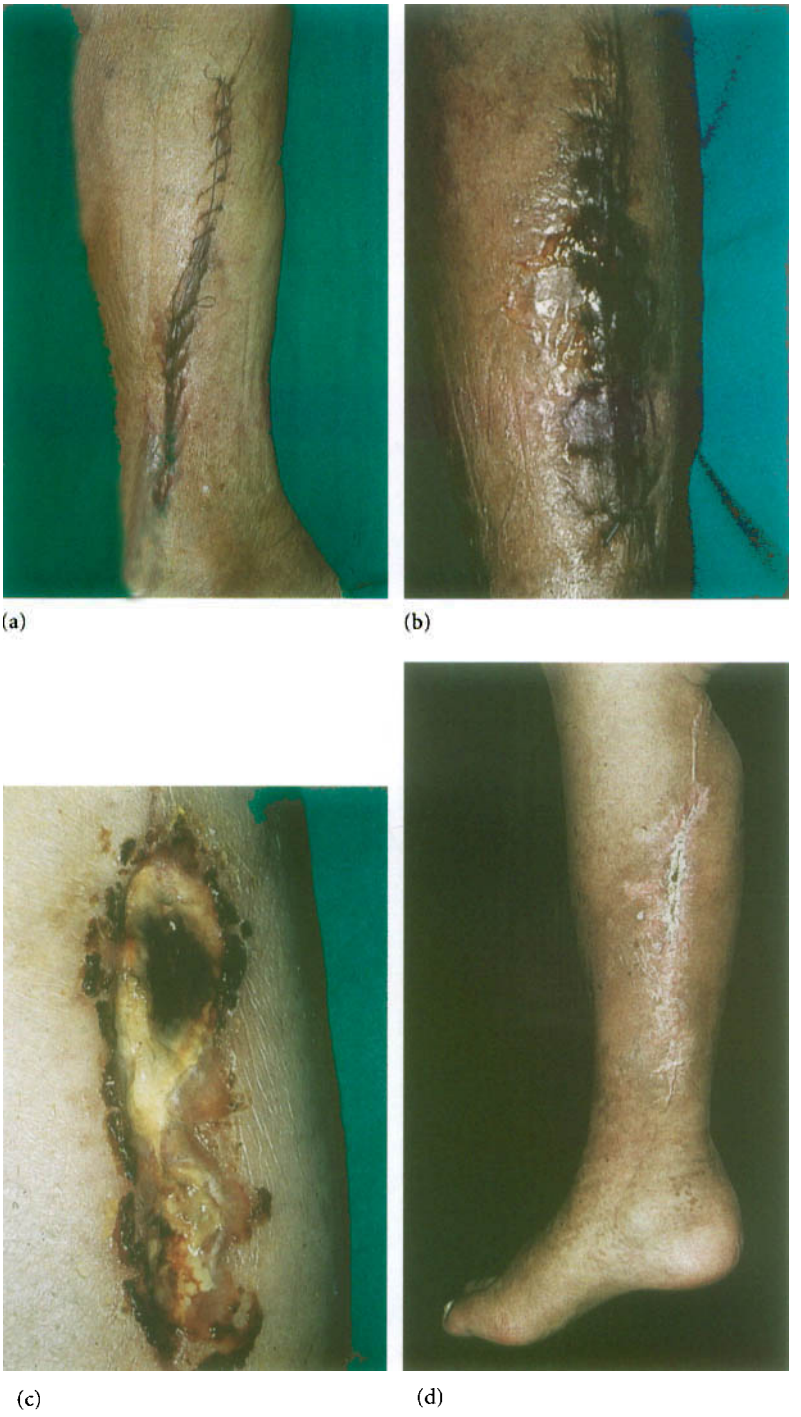


Fig. 6.28 (a) The wound has developed bluish discolouration. (b) The wound is breaking down. (c) The wound is sloughy and necrotic. (d) After 4 months the leg is fully healed and the bypass is still working.

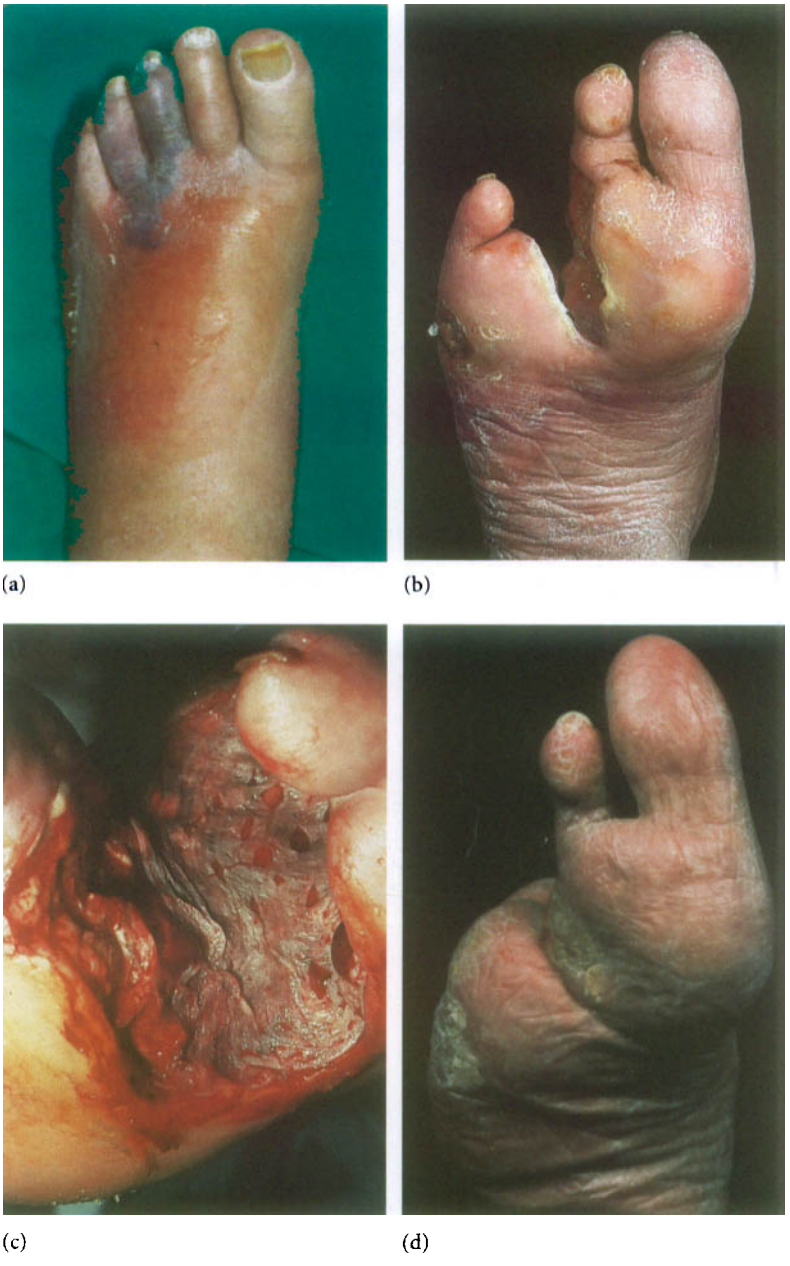


Fig. 6.29 (a) Severe sepsis with bluish discolouration of 3rd and 4th toes secondary to septic arteritis. (b) The patient underwent amputation of the 3rd and 4th rays. (c) A split-skin graft is applied to the tissue defect. (d) Foot has healed.

heparin is as effective and as safe as unfractionated heparin in the prevention of venous thromboembolism. The standard prophylactic regimen does not require monitoring.

In the neuropathic foot, non-weightbearing is advisable initially and then off-loading of the healing postoperative wound may be achieved by casting techniques.

After operative debridement in the neuroischaemic foot, especially when revascularization has not been possible, non-weightbearing is advised until the wound is healed.

If necrosis is to be treated conservatively, by autoamputation, which can take several months, then the patient needs a wide-fitting shoe such as a Dru shoe to accommodate foot and dressings, or a 'Scotchcast' boot.

Patients should walk as little as possible.

Metabolic control

When patients present with necrosis, in the background of severe infection or ischaemia, they may be very ill, and will need close metabolic and haemodynamic monitoring. Considerable metabolic decompensation may occur, and full resuscitation is required with intravenous fluids and intravenous insulin sliding scale which is often necessary to achieve good blood glucose control whilst the patient is infected or the leg severely ischaemic. High glucose levels are associated with postoperative infections in the leg as well as infections of the urinary tract and the respiratory system.

Patients will often have cardiac and renal impairment, which will need careful monitoring to optimize the regulation of fluid balance, so as to avoid hypotension from underperfusion and hypertension and peripheral oedema from overperfusion. Oedema is a potent cause of impaired wound healing. Many patients have autonomic neuropathy which may contribute to impaired blood pressure control and more frequent cardiac arrhythmias.

Nutritional impairment is denoted by a serum albumen of < 3.5 g/L and a total lymphocyte count of less than 1.5×10^9/L. A high-calorie diet should be instituted. A minimum of 1800 calories per day should be ingested to avoid the negative nitrogen balance that could accompany the depletion of protein stores.

Educational control

Gangrene can develop with alarming rapidity in the diabetic foot.

There have been cases of carers of high-risk diabetic patients being accused of neglecting their charge, when necrosis has developed rapidly and the patient has then been assessed by an inexperienced practitioner who does not realize how quickly necrosis can develop and has accused the carer of taking no action to help the patient 'who must have had gangrene for several weeks'. Healthcare professionals and patients should be aware that necrosis can develop very quickly. However, a recent case of 'gangrene' at King's College Hospital, London, was not all it seemed (see below).

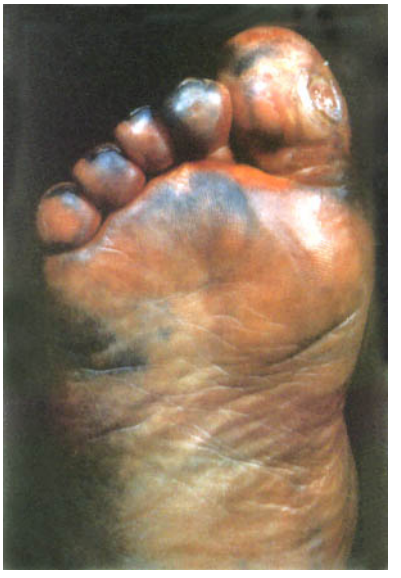


Fig. 6.30 Henna masquerading as necrosis.

CASE STUDY
Henna masquerading as necrosis

A 64-year-old lady from North Africa with type 2 diabetes of 12 years' duration and peripheral neuropathy was admitted to hospital with severe sepsis of her left foot and a neuropathic ulcer on the apex of her left 1st toe. Her pedal pulses were bounding. She was extremely reluctant to come into hospital saying that she preferred traditional remedies. However, she agreed to have intravenous antibiotics and callus debrided from around the ulcer but she refused to have a dressing put on the foot.

Forty-eight hours later the nurses noticed that her entire forefoot had turned black and the foot clinic team was telephoned from the ward and told that she had 'spreading gangrene' (Fig. 6.30). The patient's daughter had visited the previous evening bringing with her some henna which is a traditional wound-healing folk remedy in North Africa. Unknown to the nurses she had made up a paste of henna and water and put it on her foot, which was the cause of the black staining. It was now difficult to assess the foot since the cellulitis and the colour of the wound bed were changed and masked by the henna, and she agreed not to repeat the process. The swelling associated with the infection reduced and the ulcer became smaller and dryer.

She remained in hospital for a further 10 days, during which time she applied table salt to the wound bed four times a day with her meals, and she continued this prac-

tice after discharge. The ulcer healed in 6 weeks and she agreed to attend regularly for callus removal and did so until she died of a myocardial infarct 2 years later.

Key points

- All that blackens is not gangrene!
- Gangrene cannot be diagnosed from a telephone call
- Immediate direct inspection of the foot is necessary
- We discuss folk remedies with our patients and try to be aware of local traditions
- Topical applications such as henna, which stain the wound bed and surrounding tissue, render wound assessment difficult.

Fear of gangrene

Some patients and their families find necrotic feet deeply upsetting. The use of the word 'gangrene' can distress and frighten some patients. It should be explained that just because a small area of the foot has developed necrosis it does not mean that the whole foot will be destroyed or that amputation is inevitable.

The health-care practitioner should never express distaste or disgust. If he does not know the patient well, then before the foot is uncovered he should ask whether the patient has seen it. This is particularly important when the foot is first 'taken down' after surgery, because the sight of missing toes can be very upsetting and shocking.

After an amputation some patients do not want to inspect the foot at all until it is fully healed.

Patients who develop necrosis are often deeply fearful of the future. They need careful education including reassurance that much can be done to help them. Avoiding the words 'gangrene' or 'necrosis', and hiding the foot away under a dressing, can be a form of escapism which does little to address the patient's fears. We believe that practical and straightforward explanations are best.

The following educational material is in the form of commonly asked questions and answers.

If I have gangrene will I lose my leg?

Many people find gangrene a frightening word. This may be because people remember hearing about World War I and how many soldiers in the trenches developed gas gangrene which destroyed their legs and often killed them too. In fact, gangrene in the diabetic foot, although a serious problem, will not always lead to loss of the leg. In many cases the damage can be limited to loss of a small area of the skin of the foot, which will heal completely in the end leaving only a scar.

What does the word gangrene mean?

Gangrene means tissue death. There are two kinds of gangrene. One—wet gangrene—is caused by infection. The other type of gangrene—dry gangrene—is caused by a poor blood supply. When not enough blood reaches a part of the foot, the skin and flesh may die and change colour to brown or black. This is gangrene, also called necrosis.

How would I know if I was getting gangrene?

The first signs of gangrene may be that an area of the foot changes colour. It may or may not be painful. Part of the foot usually develops a bluish or purple colour.

What should I do if I think I might be getting gangrene?

If you spot it early and seek treatment immediately it is often possible to treat the infection or poor blood supply and save the foot.

The worst thing you can do in these circumstances is to ignore the problem in the hope that it will go away and get better by itself. This is unlikely to happen. Go to your diabetic foot service immediately. Do not delay.

How is gangrene treated?

There are several different treatment programmes for patients with gangrene.

If the problem is due to infection, you should be admitted to hospital and given strong antibiotics through a vein in your arm. Later the black area of your foot may be taken off in the operating theatre. This will depend on how good the blood supply to your foot is. If blood flow is good then the foot should heal quickly.

If the problem is due to infection combined with a poor blood supply then you will need antibiotics but it may also be necessary and possible to improve your blood supply to the foot.

How could my blood supply be improved?

With an angioplasty. Angioplasty involves stretching a narrow blood vessel to enable more blood to pass down it.

Are there any other ways to get more blood down to the foot?

Another possibility is a bypass operation. Bypass involves attaching a new piece of blood vessel to bypass any blockage in the blood supply. If a bypass procedure is successful it may be possible to amputate the gangrenous part of your foot and achieve quick healing.

A third possibility will apply if the blood supply to your foot cannot be improved by angioplasty or bypass. Once

any infection is under control the gangrenous area of your foot will dry out and drop off. This process is called autoamputation. It will take several months, and a lot of patience, before this happens.

What footwear can I wear if I have gangrene?
Until your foot is completely healed it will probably be necessary for you to wear a special shoe or cast boot to avoid any pressure on the gangrenous areas.

Will my black toe just fall off?
If your black toe becomes very loose and you are afraid of it coming off in bed, then it may be possible for the foot clinic to remove it painlessly.

Should my foot be dressed and bandaged?
Your gangrenous foot should be covered with dressings at all times. Animals and flies find gangrenous tissue very attractive.

How can I tell if my gangrenous foot is doing well?
If germs infect your gangrenous foot you will find that the black areas become wet and smell bad, and may become light grey or whiteish in colour. If this happens you should seek help from the foot clinic without delay.

Important signs that a gangrenous foot is not doing well are as follows:

- Foot becomes wet
- Foot changes colour from black or dark brown to grey
- Foot discharges fluid
- Foot swells
- Red area develops around line between gangrene and normal tissue
- Red mark spreads up leg
- Foot smells bad
- Leg or foot become more painful.

If you notice changes, go to the diabetic foot service at once.

What kind of dressings should be on my foot?

- You should avoid moist dressings at all costs. If gangrene becomes wet it is an excellent growth medium for bacteria and infection is likely
- Use dressings to separate black toes from their healthy neighbours, lest the problem spread from one to the other
- Avoid 'specialist' dressings and treatments like hydrocolloids, alginates, wet to dry dressings and whirlpool
- If you receive visits from a community nurse who wants to change your dressing regime, ask her to contact the diabetic foot clinic service first.

Can I bathe or shower?
Keep gangrenous toes out of the bath or shower. If gangrene becomes wet it is likely to become infected. It is possible to purchase a 'cast protector' which is a strong plastic bag shaped like a leg. You can use this to cover your foot and keep it dry while the rest of your body is bathed and showered.

How often should my foot be checked and why?
Your gangrenous foot should be checked every day for signs that it is getting worse. If this happens, talk to the diabetic foot service the same day. If it happens at a weekend then go to the casualty department at your local hospital and ask to be seen by the diabetic team. You cannot afford to wait even one day if a gangrenous foot is going wrong: taking immediate action can save your foot.

Will I need regular appointments at the foot service?
Regular treatment is very important. When you see the podiatrist or doctor at the diabetic foot service he may use a scalpel to cut small pieces of dead tissue off your foot.

Why will he do that?
This procedure, called debridement, helps healing.

How does it help healing?
The less dead tissue there is around, the fewer germs will be on your foot. Dead tissue in direct contact with healthy tissue can cause problems, so as much dead tissue as possible will be removed when you come to clinic.

Won't it hurt?
This procedure should not be painful as the only tissue being removed is already dead. However, if it causes you discomfort you should always say so.

Why do hospitals behave as if it is their diabetes and their foot and not mine?
You may sometimes feel as if your diabetes and your foot no longer belong to you, and have passed into the possession of the eager group of people—the team of the diabetic foot service—who see you every time you come to clinic. Never forget—it is your foot, and you are the one who should decide what happens. You should always be told what is going to be done to you, and why, and what will happen next.

What if I don't understand what they want to do?
If you do not understand, then ask for things to be explained again. It is essential that you are aware of what is

being done and take an interest. Even if you trust your foot clinic team, you should still take an interest, and try to understand why the gangrene developed, what, if anything, has gone wrong in the past and the ways that future trouble can be prevented.

What should I do if I'm away from home and my foot gets worse?

If you are away from home and your foot gives trouble you should seek treatment at the nearest hospital.

What should I do if I am admitted to another hospital or receive treatment elsewhere?

It is very important, in these circumstances, that the diabetic foot service is informed about what is happening. If you are under the care of less experienced people they should usually welcome input from the diabetic foot clinic.

PRACTICE POINTS

- Necrosis does not automatically lead to amputation
- Necrosis can be divided into wet necrosis and dry necrosis
- Wet necrosis in neuropathic feet needs intravenous antibiotics and surgical debridement
- Wet necrosis in neuroischaemic feet needs intravenous antibiotics, surgical debridement and vascular reconstruction
- Dry necrosis in neuroischaemic feet needs vascular reconstruction and amputation or outpatient debridement and autoamputation
- Renal patients are particularly prone to develop necrosis
- When necrosis become wet, smelly, painful or spreading then patients should seek help urgently.

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7 Stage 6: the unsalvageable foot

Cease to lament for that thou canst not help
And study help for that which thou lament'st.
(The Two Gentlemen of Verona, *III, i, William Shakespeare)*

PRESENTATION AND MANAGEMENT

Major amputation is sometimes inevitable, particularly in neuroischaemic patients. Rehabilitation of the diabetic amputee is extremely difficult and is characterized by long stays in hospital. Major amputation therefore must not be taken lightly.

Morbidity and mortality associated with major amputation in diabetes are very high. Without optimal care, within 3 years half of diabetic major amputees will be dead and of the survivors, half will have lost their remaining leg. Survival of above-knee amputees is significantly less than below-knee amputees, and relative mortality is higher for females than for males.

Reasons for major amputation

Diabetic patients who present with extensive ulcers on their feet are sometimes offered early amputation as 'the one sure way of sorting out the problem permanently', on the basis that such an operation is likely to be inevitable at some time in the future. This approach may be useful for young, otherwise healthy, non-diabetic patients incapacitated by pain or a useless limb, whose other limb is normal. However, major amputation does not guarantee an ulcer-free existence for the diabetic patient, and non-healing ulcer alone should not normally be an indication for major amputation.

Major amputation is usually carried out for the neuroischaemic foot and should be rare in the neuropathic foot. Major amputation in the neuroischaemic foot is necessary in the following circumstances:

- When overwhelming infection has destroyed the foot and threatens the patient's life
- When there is severe ischaemia with rest pain that cannot be controlled
- When extensive necrosis secondary to a major arterial occlusion has destroyed the foot.

CASE STUDY

Overwhelming necrosis after patient lost to follow-up

A 73-year-old man with type 2 diabetes of 25 years' duration, peripheral neuropathy, peripheral vascular disease and previous amputation of his 2nd toe for osteomyelitis, failed to attend follow-up appointments in the diabetic foot clinic. He lived alone and turned away ambulance transport, despite frequent reminders and notification of his general practitioner, who arranged weekly visits by the district nurses. His forefoot changed colour: it was initially blue and then became black but because the patient did not complain of pain no help was sought. After 5 weeks the discolouration spread up the foot. He was admitted to hospital with wet gangrene. The foot was already destroyed at presentation (Fig. 7.1). He underwent a below-knee amputation.

Key points

- Necrosis is often painless in the diabetic foot
- If dry necrosis becomes infected, wet necrosis can supervene and destroy the foot in a short time
- Regular follow-up is crucially important for high-risk diabetic patients with foot problems
- We become alarmed if high-risk patients fail to keep appointments and try to arrange for regular inspections of the feet.

CASE STUDY

Overwhelming necrosis following arterial occlusion

An 80-year-old woman with type 2 diabetes mellitus was admitted to hospital following a stroke and discharged home under the care of the general practitioner. The right foot became discoloured but she felt no pain and it was not until overwhelming necrosis of the right foot and

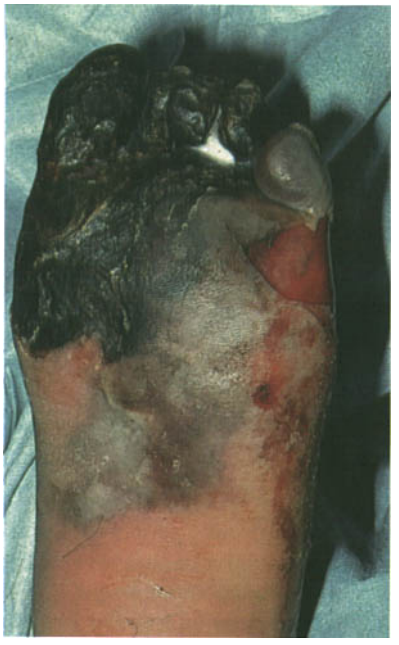

Fig. 7.1 Overwhelming necrosis: this foot was already destroyed at presentation.

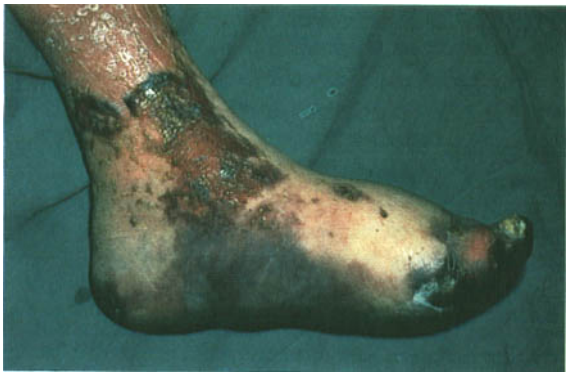

Fig. 7.2 Necrosis spreading to the leg after a major arterial occlusion.

lower limb had developed that she showed her leg to the district nurses (Fig. 7.2). When she was admitted to hospital she was moribund. She had necrosis spreading to the lower leg which was icy cold. Clinically, this indicated a major arterial occlusion. She underwent palliative care and died shortly afterwards.

Key points

- If neuropathy is profound even occlusion of a major vessel will not be painful.
- Patients who have had a stroke may have abnormal sensations and fail to complain.
- Vulnerable patients in the community should have their feet checked regularly by health-care professionals.

Major amputation in a neuropathic foot should be a very rare event and necessary only when:

- Infection has irretrievably destroyed the foot
- Charcot's osteoarthropathy has destroyed the ankle joint, attempts at external stabilization have been unsuccessful and internal fixation is not possible.

The decision to amputate

When a major amputation is being considered the following factors should be addressed by the multidisciplinary team:

- Social factors: some practitioners believe that patients who face many weeks or months of treatment should be offered a major amputation as a serious treatment option and that amputation can often be viewed very positively. Elliott Joslin felt that if life expectancy was very limited it could not be regarded as a success if the patient spent much of his remaining days in hospital to save his leg
- Emotional factors: many patients and their families react with horror to the idea of a major amputation. Depression after amputation is common
- Financial factors: diabetic foot patients may be regarded as 'expensive' patients in terms of:
  - Number of bed days occupied
  - Consumption of expensive antibiotics
  - Costly interventions.

  However, major amputation is not cheap and involves:
  - Accumulated costs of rehabilitation
  - Prosthetics service
  - Loss of earnings
  - Costs of special services
- Functional factors: in elderly, frail diabetic patients the functional results of amputation are usually poor: many patients do not walk again and never return to independent living.

There should be clear criteria for amputation as described above, and the decision should be made by a multidisciplinary team together with the patient and his family. Sometimes the decision is not an easy one.

CASE STUDY
Problems with 'the good leg'

A 39-year-old male with type 1 diabetes of 27 years' duration and end-stage renal failure treated by continuous

ambulatory peritoneal dialysis had a history of bilateral neuropathic ulceration and underwent amputation of left 3rd, 4th and 5th toes. The foot healed and he was issued with an orthotic walker. The foot remained intact for most of the time, but broke down if he had to run for a bus or walked more than usual. He was desperate to wear 'normal' footwear. He was placed on the waiting list for a joint renal/pancreas transplant but had been told that if he had ulceration the transplant could not be carried out. He had two young children and deeply resented being unable to run and walk freely and participate in sports. His orthotist referred him to the rehabilitation team who advised him to consider a below-knee amputation since he was a comparatively young man and would do well with a modern prosthesis.

When the patient discussed this proposal with the multidisciplinary diabetic foot team they perceived major amputation far less positively. They pointed out to the patient that with a prosthetic limb his remaining foot, with a previous history of ulceration would be overloaded and that a major amputation could not guarantee that he would remain free from ulceration. Nonetheless he made the decision to ask for a major amputation and was put on the waiting list for an elective below-knee amputation.

One month later he attended at the diabetic foot clinic as an emergency, complaining of pain and numbness in the right foot (the 'good' foot) which had been present for several days. The foot pulses were impalpable, and the leg and foot were mottled and grey and cold from mid-calf downwards. He developed necrosis of his medial longitudinal arch and hallux which slowly extended to involve three of the lesser toes and the heel. The angiogram showed occlusion of the mid and distal popliteal artery with faint filling of the anterior tibial artery and no plantar arch was seen. The popliteal artery was angioplastied, which allowed increased flow through the popliteal artery, anterior tibial artery and some flow into the plantar vessels. He has subsequently had repeat angioplasty of the popliteal and superficial femoral arteries at 3 and 4 months after presentation, and also underwent surgical debridement of necrotic tissues. He has cancelled amputation of the left leg.

Key points

- Every foot is a precious commodity which should be preserved if at all possible because of future risks to the other foot
- Angioplasty can be a successful treatment even for occlusion of the popliteal artery
- Angioplasty can be repeated to maintain the patency of the artery.

Choice of level of amputation

The level of amputation should be carefully considered to ensure that there is sufficient perfusion to achieve wound healing. When possible, a below-knee amputation should be carried out to conserve the knee joint and aid the fitting of a prosthesis. Preserving the knee joint lowers the energy expenditure necessary for walking. The cardiovascular cost for walking and foot plantar pressures in the opposite limb both increase in direct proportion as the amputation becomes more proximal.

The aims are:

- To keep the amputation as distal as possible
- To amputate above painful, cold, pale discoloured tissue
- To amputate below warm pink well-perfused tissue.

About one-fifth of transtibial amputations and knee disarticulation amputations undergo revision surgery to proximal amputation due to healing complications.

Preoperative care

The following points should be recognized:

- Admission to hospital is always an anxious time, especially for patients fearing or facing a major amputation
- When patients are worried and anxious they may not retain information
- Information should be repeated several times and reinforced with the written word
- Patients like to feel that their limb is valuable, and that initial investigations and interventions are made in an effort to try and save the limb
- Patients want to know the reason why the leg needs to be amputated
- If patients are ill and toxic they may not comprehend what is happening
- Patients who are facing amputation should be given time to come to terms with it wherever possible
- Detailed explanations of all procedures should be given. The level of amputation should be explained and the intended site of amputation should be touched with the health-care professional's hand to demonstrate the level. The patient should be told that the wound will be covered with skin
- The effects of a general anaesthetic should be explained to the patient and family
- The stress of amputation should not be underestimated. If the operation is delayed or cancelled for any reason the patient and relatives should be informed immediately.

Physical assessment of the patient is important.

Cardiorespiratory status and metabolic control should be optimized. Patients should be encouraged to cease smoking. Malnutrition increases the risk of delayed wound healing. Weight loss and diminished appetite are common and patients should be seen by the dietitian.

Antibiotic prophylaxis should be used.

Once major amputation is planned a lumbar epidural block with bupivicaine can be started 48 h beforehand to relieve postoperative pain.

Perioperative care

A major amputation will put the remaining foot at great risk of ulceration.

The heel of the surviving foot should be protected on the operating table and postoperatively. One of our surgeons always wrapped several layers of thick cotton wadding (Gamgee, Robinson) around the heel of the contralateral foot to avoid pressure on the heel during the amputation.

Drains are advisable for amputations, as blood clots are a good culture medium for bacteria.

A rigid dressing applied in below-knee amputations from the end of the stump to the mid-thigh, in full extension, will reduce oedema, protect the wound and limb against trauma, and prevent knee contractures during the first 3 postoperative weeks.

Postoperative care

Some patients return from theatre thinking that they still have their leg as they can still 'feel' it. Without reminders they may get out of bed and try to 'stand on two feet' resulting in a fall and possible injury to the stump or the other foot.

Phantom sensation gradually decreases and may telescope so that the patient feels his foot at his thigh.

During the postoperative period patients who have lost a limb often describe similar feelings to those described by people who have just had a chronic disease such as diabetes diagnosed, or people who have undergone a bereavement.

There may be physical sensations including:

- Fatigue
- Helplessness, muscle weakness, lack of energy
- Feeling of hollowness in stomach
- Tightness in chest and throat, and breathlessness
- Oversensitivity to noises
- Dry mouth.

Common emotional reactions include:

- Sense of unreality and light-headedness
- Feeling of observing oneself from outside
- Disbelief, confusion, hallucinations, sleep disturbance, dreams
- Preoccupation
- Sense of the presence of the lost limb
- Absent mindedness
- Sighing, crying.

Other feelings may include:

- Despair
- Sadness
- Anger and frustration
- Guilt and self-reproach
- Anxiety
- Loneliness
- Shock
- Relief.

The initial phase involves shock and disbelief, which is often followed by feelings of sadness, despair, anxiety and sometimes anger or pining. Although these feelings usually decrease as patients start to recover or adapt, individual reactions vary. It is important for patients and their families to be aware of these effects.

In the early phase, patients may feel emotionally numb, and may need help with making the simplest decisions.

Common social reactions include:

- Social withdrawal
- Avoiding reminders of the amputation.

Gradually the patient enters the recovery phase. In order to optimize recovery it is necessary for all healthcare professionals to:

- Listen and give the patient time
- Acknowledge their special loss
- Acknowledge any feeling, especially negative ones such as anger, and offer reassurance that these are normal and to be expected
- Be prepared to handle the stump and encourage spouse or family to do likewise
- Be aware that some patients hate the word 'stump'
- Be aware that the first dressing change is particularly frightening. Comments about the state of the wound should be true and tactfully presented, avoiding negative facial disgust or inappropriate belittling comments
- Reassure the patient that any stump pain should settle gradually as oedema and inflammation settle and should be reduced by regularly prescribed analgesia.

The relatives of patients also need attention and sympathy. The psychological effect of amputation on the patient's relatives may be profound. We remember the wife of a major amputee who refused to have a ramp fitted to the

house because she said it would spoil the look of her home. We are not sure whether she meant that ramps were unaesthetically pleasing, whether she felt there was a stigma attached to a house with a ramp, or whether she simply enjoyed being able to control their exits and their entrances. We were also unclear about the motives of another patient's wife who refused to allow him to be fitted with a prosthesis after his below-knee amputation. She said the reason was that she was afraid that he would fall over and hurt himself, but the patient himself believed that she enjoyed being in control.

Amputation wounds are often slow to heal in neuroischaemic patients. Infection should be treated aggressively and the vacuum-assisted closure (VAC) pump may be useful. However, if there is poor arterial perfusion to the below-knee wound it may be necessary to convert it into an above-knee amputation.

Shortly after the amputation has been performed, oedema of the stump can be a problem, and JUZO socks, which are compression stump shrinkers, provide good oedema control both in the acute stages, and long term. They are only provided after the stitches have been removed as they can otherwise drag on the wound edges and cause dehiscence. The stump is measured and the sock supplied between days 5 and 10 postamputation.

Initially, the sock is applied for 10 min only and the stump is then inspected for problems including colour change and breaks in the skin.

Once healed, the stump should be inspected daily for skin breakdown, which should be cleaned, dressed and off-loaded until complete healing is achieved.

Mobility aids

Before the definitive prosthesis is issued, some patients may be suitable for mobility aids.

The amputee mobility aid (AMA) is suitable for below-knee and through-knee amputees only. The stump is supported and stabilized by an inflatable bag, which also assists in reducing oedema. It is a physical and psychological boost to get the patient on his feet early. It has a knee joint.

The pneumatic postamputation mobility aid (PPAM aid) has an inflatable socket and is suitable for above-knee, through-knee and below-knee amputees.

The skin should always be checked before and after use of mobility aids.

When the amputation stump is slow to heal, special prostheses can be used to facilitate weightbearing and reduce pressure on the stump (Fig. 7.3).

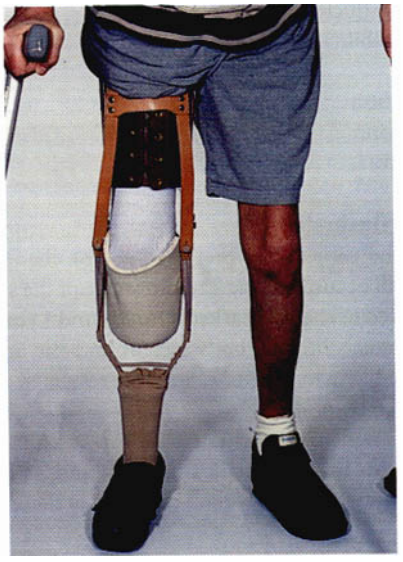

Fig. 7.3 This patient is wearing an interim prosthesis with felt cup, corset and metal shin.

The definitive prosthesis

The standard prosthesis contains a stump sheath worn inside a customized thermoplastic socket. This is then fitted onto a modular prosthesis. The shank of the prosthesis articulates with a prosthetic foot that is matched to the patient's physique and functional requirements.

Putting the definitive prosthesis on and off may be difficult if hands are neuropathic and eyesight is poor, and visual inspection of the stump may be difficult. Velcro straps are useful in the patient with neuropathy and poor hand function to aid donning and doffing of the prosthesis. If skin is atrophic and circulation is reduced, stasis dermatitis may be a problem, and the skin is easily injured.

Rehabilitation

The key for success in dealing with the stage 6 patient is careful follow-up. With good planning and follow-up care after a major amputation, the stump will heal but it is also important to ensure that it will remain intact. Healthcare professionals in the diabetic foot service should be aware of the presentation and management of problems with the amputated stump. Often they will be treating the remaining foot of their patients but be asked to advise on problems with the stump of the contralateral leg.

Problems with the stump include:

- Volume fluctuation
- Susceptibility to trauma
- Callus
- Ulceration
- Infection.
- Ischaemia.

Volume fluctuation

It is in the early stages that the greatest changes in the stump will occur, and the size and shape of the stump can be expected to change markedly for around 1 year. During the amputee's life, his body weight and the amount he walks are likely to fluctuate, and this results in alteration of the stump volume. Cardiac and renal disease can lead to stump oedema. Fluctuating stump volume is a particular problem in diabetic amputees, and patients with neuropathic stumps may have difficulty in detecting it.

Susceptibility to trauma

Amputees are vulnerable to falls, particularly when not wearing the prosthesis, and may fracture the stump.

CASE STUDY
Fractured stump

A 62-year-old woman with type 2 diabetes of 18 years' duration underwent a right below-knee amputation after developing a deep infection of her neuropathic foot. She lived abroad and had no rehabilitation, but 4 months later she was visiting her daughter in the UK, developed neuropathic ulceration of the left hallux and was referred to the diabetic foot clinic as an emergency.

At her second visit she mentioned that she had fallen and injured her stump when transferring from a taxi to her wheelchair. She was wearing a silicone sock on her stump at the time. She felt no pain in her stump after the fall, but 3 days later she developed severe pain in the stump which kept her awake at night and was described as 'aching' and 'spasmodic'. On examination there was minimal swelling, no haematoma and no break in the skin of the stump. X-ray revealed a fracture through the tibia extending to the resected surface (Fig. 7.4). She was referred to the fracture clinic and a bivalved stump splint was applied.

Key points

- In a neuropathic stump, signs and symptoms of fracture may be minimal or delayed
- Neuropathic stumps should be X-rayed following trauma.

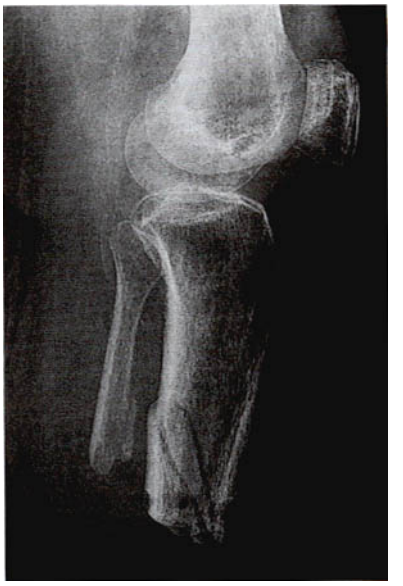

Fig. 7.4 Fracture of tibia of below-knee stump.

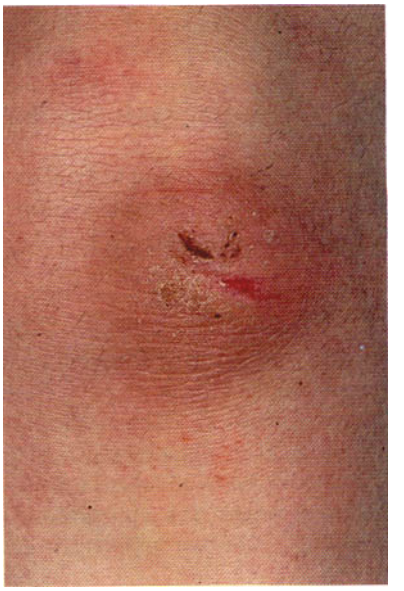

Fig. 7.5 Callus on a stump.

Callus

Patients with neuropathic stumps are prone to develop callus. If this is not debrided it will lead to ulceration (Fig. 7.5).

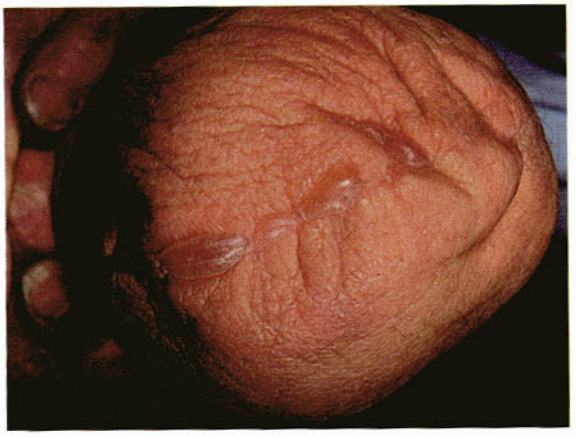

Fig. 7.6 This neuropathic stump developed blistering along the amputation scar.

Ulceration

This often begins with a blister which may be caused by friction or pressure within the prosthesis socket. All tissue breakdowns should be cleaned with saline and covered with a dressing. If possible the patient should not wear his prosthesis, and should seek urgent review of the fit (Fig. 7.6).

Infection

There is a great risk of ulcers on the stump becoming infected, and we have seen a case of below-knee amputation being converted to above-knee amputation because of infection which occurred 4 years after the first amputation. Management is debridement, pressure relief and antibiotics.

Ischaemia

Ischaemia can present as pain, often associated with erythema and a cold stump. Figure 7.7a,b shows the stump of an ischaemic below-knee amputation which has become painful and very red, and shows early signs of blistering. This was due to increasingly severe arterial disease and the patient was converted from a below-knee to an above-knee amputation.

Care of the remaining limb

After amputation, the value of the remaining limb should not be underestimated: even if the patient never walks again he will need his leg to transfer from chair to bed and lavatory, and thus maintain a little independence.

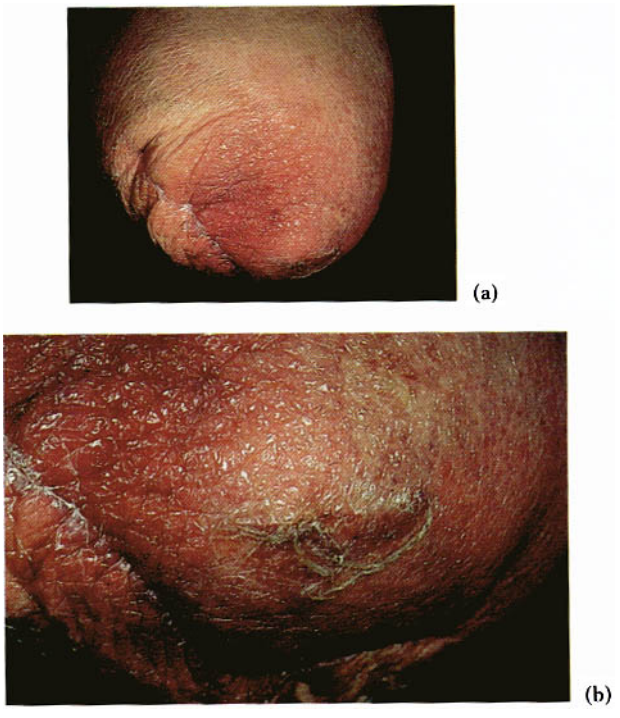

Fig. 7.7 (a) A painful red ischaemic stump. (b) Close-up view shows early signs of blistering. She went from a below-knee amputation to an above-knee amputation.

CASE STUDY
Gangrenous heel

A 78-year-old man with type 2 diabetes of 9 years' duration and peripheral vascular disease treated with left distal bypass presented late with infection of the left foot which resulted in overwhelming necrosis. He was ill and toxic and underwent an above-knee amputation of his necrotic left leg. Four days later a blister was noted on his right heel which became infected and necrotic. He was given antibiotics to control infection. The necrosis dried out and became well demarcated from surrounding tissue. He underwent angiography and distal bypass to the right leg. A pressure-relieving ankle–foot orthosis (PRAFO) was issued. The foot healed in 6 months.

Key points

- Heel protection is essential for diabetic patients during the perioperative and postoperative period
- Dry necrosis of the heel can be treated by gentle debridement and does not necessarily need operative surgical debridement

- There should be close liaison between the diabetic foot service and the rehabilitation team
- The remaining foot will be at risk of overloading, and should be carefully protected during the perioperative and postoperative period. The remaining foot also needs careful attention.

Diabetic amputees should:

- Not attempt to cut their own toe nails
- Check the foot and stump every day
- Report problems immediately
- See a podiatrist regularly.

Rehabilitation physiotherapists, prosthetists, orthotists and ward staff must understand the need to avoid trauma to the remaining foot at all costs. Major amputees are among the most high risk of all diabetic foot patients. Even with optimal foot care, foot problems occur in many major amputees, and unless they are detected early and aggressively treated by a multidisciplinary team, the outlook will be very poor.

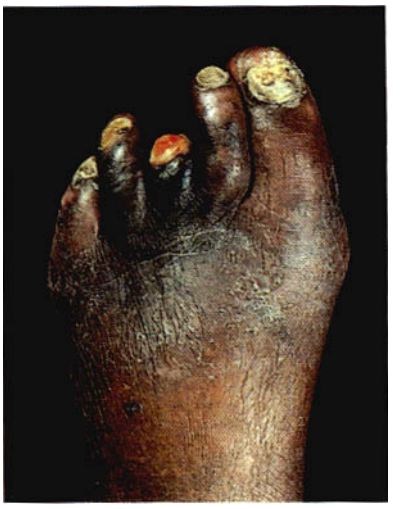

Fig. 7.8 Ischaemic ulceration in a foot with severe rest pain which led to above-knee amputation.

Living with an amputation

Patients who have undergone a major amputation face the major frustrations of losing independence and being wheelchair bound. Practical help and information should always be available. Very simple advice can help with day-to-day activities, such as drink holders and trays which clamp to wheelchairs, cordless telephones and self-propelled wheelchairs if vision and manual control are adequate. Some of our amputated patients use electric wheelchairs and buggies very successfully, and one patient uses a small four-wheel drive vehicle to get into the countryside and covers very rough ground. Compact folding wheelchairs enable patients to get out by car. Manual or automatic controls for cars can enable patients to drive. Ramps and disabled lavatories foster independence.

It is important for health-care professionals to promote patient independence as far as possible. Major amputees in wheelchairs should not be moved without their permission, but should be encouraged to decide where they go and what happens to them wherever possible.

It is distressing for patients not to be offered a prosthetic limb and they may feel that it is because they have been written off, or that they do not have long to live.

CASE STUDY

Amputation for uncontrolled pain and coming to terms with limitations

An 83-year-old woman with type 2 diabetes of 14 years' duration with a previous history of stroke, who was already wheelchair bound, presented with ischaemic ulceration and severe rest pain (Fig. 7.8). She had had transfemoral angiography and this had shown severe infrapopliteal disease with no recognizable main artery below the knee. Furthermore the arterial circulation of the foot was extremely poor with no plantar arch present. Neither angioplasty nor bypass was possible. She did not want an amputation and her ischaemic ulceration was treated conservatively. However, despite opiate analgesia it was not possible to control her pain and for this reason she underwent above-knee amputation.

Postoperatively, she was happy that she no longer had pain, and she was rehabilitated to return to her wheelchair lifestyle. She attended the diabetic foot clinic for care of the remaining foot. Three months later we noticed that she was depressed and she confided that she would have liked to have tried a prosthesis. The physiotherapists agreed to see her in the rehabilitation gym, and to let her try a pneumatic postamputation walking aid (PPAM aid) and discuss having a cosmetic prosthesis. After two sessions she came to terms with the fact that a prosthesis would not help her and declined further treatment. However, she said that she felt much happier because, as she told us, 'I was allowed to try instead of being written off'.

Key points

- Uncontrolled rest pain in the presence of unreconstructable arterial disease is an important reason for a major amputation
- Patients may resent not being offered the chance to try a prosthesis

- Help and advice on all aspects of living with an amputation are available from national organizations such as the Disabled Living Foundation in the UK and the Amputee Coalition of America.

Leaving hospital

When the time comes for the patient to leave hospital the following should be addressed:

- A wheelchair should be provided
- Wheelchair accessible accommodation should be available
- Patient is able to transfer
- Patient is managing his own programme of exercises
- Patient is medically stable
- Follow-up care has been organized.

Ideally the patient should be capable of:

- Putting on and taking off the prosthesis independently
- Walking with the prosthesis
- Moving up and down stairs
- Walking outside
- Getting on and off the floor independently.

Phantom limb, phantom pain and residual pain

Phantom limb is sensation felt at the amputated site which is not painful. Phantom pain is pain felt at the amputated site. Residual pain is stump pain felt at the site of the surgical incision.

Pain felt when wearing a prosthesis could be due to:

- Socket fitting problems
- Inappropriate prosthetic device
- Low pain threshold
- Pressure sores.

Postamputation pain is more likely if the patient has been in pain for a long time before the amputation.

Management

Treatment of phantom limb pain includes:

- Percussion therapy
- Transcutaneous electrical nerve stimulation
- Drug therapy—tricyclic antidepressants
- Ultrasound.

The aftermath of amputation: advice to health-care professionals

When catastrophes happen and patients lose a leg because of diabetic foot complications, then a storm of strong emotions, including fear and anger, is often aroused in the patient and his relatives. They may seek a scapegoat—someone to blame for the amputation—as if apportioning guilt makes them feel safer because they can then deny that a similar disaster could happen to the remaining leg. Unfortunately, it is often the last person who saw or treated the foot who is blamed for the catastrophe, and the sins they are accused of may be sins of commission or sins of omission.

When patients die after an amputation, their grieving relatives may similarly look for someone to blame. If patients and practitioners do not know each other well and treatments are not explained, problems of communication are more likely to develop.

Because diabetic feet can go wrong with alarming rapidity and the triggering factors may not always be clear, practitioners are very vulnerable to criticism. We recommend the following precautions:

- Wherever possible, practitioners should not attempt to treat high-risk diabetic foot patients in isolation
- Full and careful record keeping is mandatory
- When things are going badly, patients and their families should be forewarned.

Podiatrists are particularly vulnerable to false accusations because they often work alone and patients may not understand their scope of practice. Reasons for callus removal, cutting back nails and ulcer debridement should be explained clearly. Unproven therapies should be approached with caution.

PRACTICE POINTS

- The main causes of amputation are severe uncontrolled pain, or major tissue deficit secondary to ischaemia or infection
- Amputees need heel protection during the perioperative and postoperative periods. Physiotherapists, prosthetists and all members of the rehabilitation team should be aware of the vulnerability of the remaining foot
- The stump of the diabetic amputated limb is susceptible to trauma, ulceration, infection and ischaemia which demand urgent assessment and management
- The remaining foot should receive intensive multidisciplinary care.

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8 Surgical approach to the diabetic foot

This fester'd joint cut off, the rest rests sound;
This, let alone, will all the rest confound.
*(*Richard II, *V, iii, William Shakespeare)*

Diabetic gangrene is preventable in the overwhelming majority of cases . . . Hopefulness in the treatment of gangrene is possible. It comes first of all from the knowledge that the majority of the cases are needless and occur in those patients who have not been trained in the care of their feet or who have not followed training . . . prompt treatment of the infection might have prevented the gangrene.
(Elliott P. Joslin, MD, The menace of diabetic gangrene, N Engl J Med, *1934)*

INTRODUCTION

The discovery and commercial production of insulin in the early 1920s were seminal developments in the treatment of diabetes that allowed people affected by this disease to live an almost normal life. Although insulin commuted the death sentence attributed to diabetes, it was soon recognized that it was not a cure. As people affected by this disease lived longer, they began to experience serious complications including blindness, kidney failure, heart disease, stroke and amputations. In 1934, the American diabetes specialist Elliott P. Joslin remarked that following the introduction of insulin, mortality from diabetic coma had fallen significantly from 60% to 5%. Yet, deaths from diabetic gangrene had risen significantly. Joslin alleged that the reason for this complication was that physicians were not aggressive enough in their treatment of diabetes. He firmly believed that gangrene and amputations were preventable. He noted that there was almost always a history of injury to the foot that preceded the development of gangrene and led to amputation. He observed that burns and shoes were responsible for the most common injuries. Joslin's remedy was a team approach to diabetes care that emphasized patient education in foot care, dietary therapy, exercise, prompt treatment of foot infections and specialized surgical care. Joslin stressed the importance of cleanliness, daily foot inspection for early signs of trouble and preventive foot care. These recommendations are just as relevant today and are the foundation of diabetes foot care and self-management education.

Surgical management of the diabetic foot plays an integral role in the prevention and management of limb-threatening complications for people with diabetes. The objective of this chapter is to provide clinicians with a comprehensive and practical discussion of surgical management, as part of a team approach to care for patients with diabetes. It should be understood at the outset that early intervention provides the best chance of surgical cure and limb salvage. The goal is to preserve a functional plantigrade foot, and to prevent major amputation. In the words of the Roman poet Ovid, 'Stop it at the start: it's late for medicine to be prepared when disease has grown strong through long delays' (Ovid, 43BCE–18CE, *Remedia Amoris*, line 91).

For the purpose of this discussion, surgery of the diabetic foot will be stratified into three broad categories including:

- Elective surgical procedures
- Prophylactic surgical procedures
- Emergent (emergency) surgical procedures.

Elective surgical procedures

Elective surgery includes procedures that are advantageous to the patient but not urgent. For example, correction of a painful bunion or hammer toe in a stage 1 patient (with protective pain sensation, adequate perfusion and well-controlled diabetes) is considered elective. Yet, surgical correction of these same deformities is considered prophylactic surgery when the patient is neuropathic or neuroischaemic and the condition places the foot at risk for ulceration, infection and amputation. The patient's risk of developing a foot ulcer, as well as the patient's healing potential (vascular status) will determine whether a procedure is advisable or not. An important caveat is that patients must have adequate distal perfusion for surgical wounds to heal.

Elective surgery for the well-controlled, low-risk diabetic at stage 1 should be considered the same as for any other healthy patient. The surgical procedures as well as their risks and benefits are essentially the same. A discussion of these procedures is beyond the scope of this chapter, and the reader is referred to standard texts on foot surgery. Instead, the focus of this chapter is on prophylactic and emergent surgical procedures performed on the high-risk diabetic foot.

Prophylactic surgical procedures

Prophylactic surgery includes procedures which are necessary to prevent further compromise of the foot: for example, a patient with chronic recurrent ulceration beneath the hallux, who has a limitation of motion at the 1st metatarsophalangeal joint. The pathomechanical aetiology of this lesion, in an insensate patient, is hallux limitus or rigidus. Unless this condition is corrected the ulcer will never be completely resolved. Another example is the patient with a stable Charcot foot, with residual deformity, that cannot be accommodated by a shoe or brace. The deformity presents a serious and predictable risk for breakdown of the skin. We know that if this condition is not corrected, that shear stress and vertical forces on the skin will result in ulceration. Chronic, recurrent ulceration, with infection, will ultimately result in extension of the infection to bone (osteomyelitis), and amputation. Although these conditions are not immediately limb threatening, their natural history reveals that it is more likely than not that they will eventually become emergent conditions.

Emergent surgical procedures

Emergent surgery includes conditions that require immediate surgical intervention. These patients generally present to the emergency room/casualty department with serious foot infections. It is important to emphasize that signs of systemic toxicity are not always present and clinical findings may be subtle. Patients may or may not be febrile; they may or may not have an elevated white blood cell count: however, their diabetes is most often out of control. These patients require immediate hospitalization and work-up for infection and surgery. Depending upon the presentation, surgical treatment may include: incision and drainage of pus, exploration of wounds, debridement of necrotic soft tissue and bone, revascularization and local amputation of the foot. The urgency and aggressiveness of surgical care is determined by the nature of the presentation and by the clinician's familiarity with the diabetic foot. Limb-threatening conditions in seriously ill patients require immediate medical and surgical triage.

PRINCIPLES OF SURGICAL MANAGEMENT

A team approach to the medical care of patients with diabetes is necessary for successful surgical management. Prior to surgical intervention, patients require thorough preoperative medical assessment and aggressive management of their diabetes and comorbid conditions. Prompt attention must be directed to cardiovascular, renal, peripheral vascular and infectious disease issues. There is also a need to assess the patient's nutritional status and requirements for help from the dietitian. Wounds will not heal without adequate nutrition, nor will they heal with insufficient distal perfusion. Clinicians should be well trained to recognize emergency diabetic foot problems, and to distinguish immediately between limb-threatening and non-limb-threatening presentations. Successful surgical intervention demands timely drainage of infection and debridement of necrotic, bacteria-laden tissues. The risk of tissue loss and amputation is increased with inadequate antibiotic coverage and long delays in providing surgical care. It is important to emphasize that antibiotics alone are not sufficient for the management of most diabetic foot infections. Infected wounds must be incised and dependent drainage established. Postoperatively patients must be followed closely, with lifetime surveillance in the diabetic foot clinic. Appropriate footwear and preventive services are required.

Principles of surgical management include:

- Prompt detection and intervention
- Preoperative medical work-up and clearance for surgery
- Medical management of diabetes and comorbid conditions
- Targeted antibiotic coverage of infection
- Vascular work-up
- Consultations:
  Foot and ankle surgeon
  Infectious disease specialist
  Vascular surgeon
  Diabetologist
  Prosthetist/orthotist
  Physiotherapist
- Wound care and dressings
- Postsurgical surveillance
- Podiatric care, footwear and orthoses.

GOALS OF DIABETIC FOOT SURGERY

- Establish dependent drainage
- Remove bacteria-laden necrotic soft tissues
- Remove infected/necrotic bone
- Correct deformity
- Reduce risk of ulceration or amputation
- Restore stability and alignment
- Preserve function
- Achieve a cosmetically acceptable result
- Prevent major amputation of the leg.

SURGICAL PROCEDURES

Incision and drainage

Incision and drainage is the basic tenet of treatment for nearly all infections of the diabetic foot. Streptococcal cellulitis is an exception to this rule. Initial drainage of an abscess can be performed in the emergency department or at the bedside, under local field block or regional ankle block anaesthesia (Fig. 8.1a,b). Drainage means opening up all collections of pus (abscesses), with gentle probing of the superficial and deep tissues for sinus tracts. If present, sinus tracts need to be laid open. Bacteria-laden necrotic tissues are debrided and dependent drainage is established. Sometimes amputation of a toe(s) or ray(s) may be necessary to establish drainage: however, this is best done in the operating theatre. In severe limb-threatening situations, guillotine amputation of the foot may be necessary to stem systemic toxicity. It is important to emphasize that medical treatment of infection, solely with antibiotics, is insufficient to resolve the majority of diabetic foot infections. In the simplest and most common scenario, surgical debridement of ulcers is the mainstay of treatment. Salvage of the diabetic foot may require aggressive debridement and revascularization.

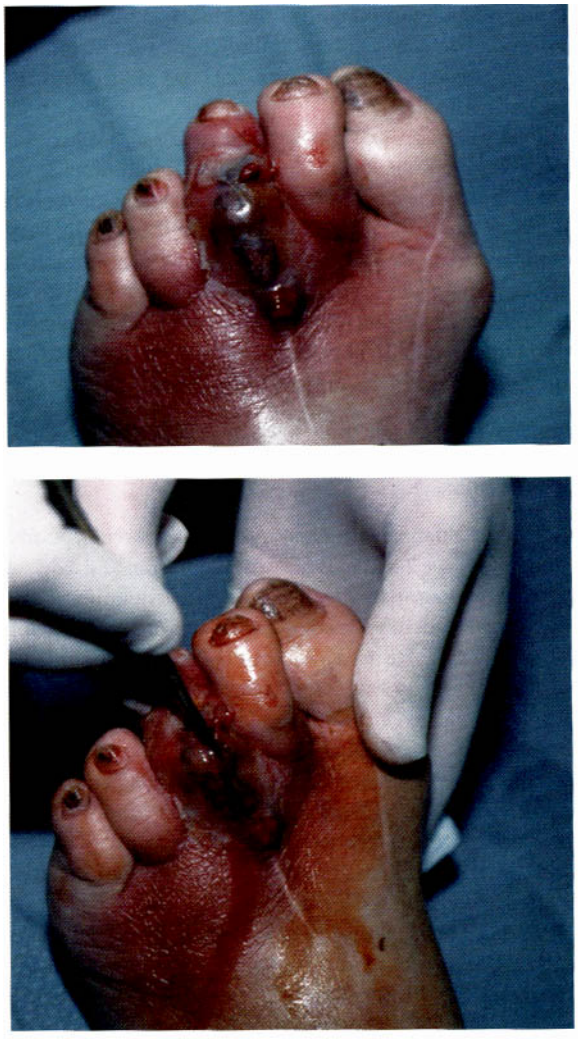


Fig. 8.1 (a) Abscess on 3rd toe with cellulitis, left foot. (b) Incision and drainage of the abscess, at the bedside.

Gas in the soft tissues is a serious finding requiring an immediate trip to the operating theatre for open drainage of all infected spaces, and intravenous broad-spectrum antibiotics. This presentation is characterized clinically by crepitus, a crackling sensation noted on palpation of the affected soft tissues. This finding is confirmed on

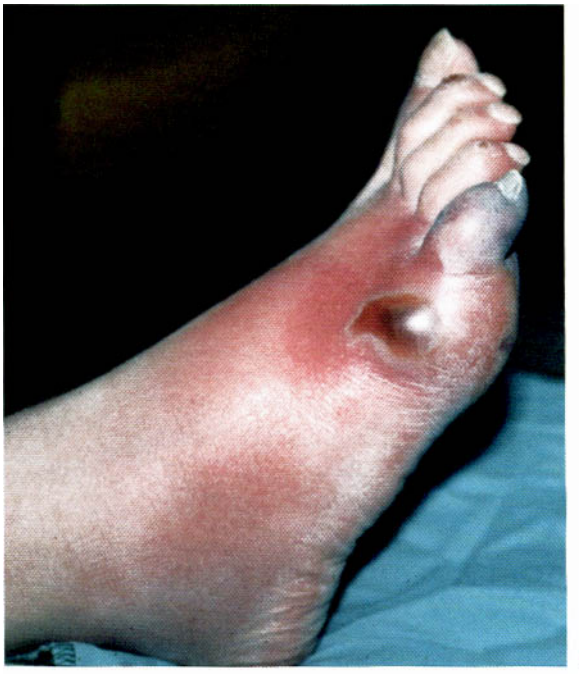
radiographic examination of the foot. Gas formation by infecting bacteria is common in diabetic foot infections, and is caused by both clostridial and non-clostridial organisms.

One or more incisions may be necessary. Whenever possible, incisions should be directed longitudinally on the foot, so as to avoid the neurovascular structures. It is important to inspect the foot for involvement of the deep compartments, as well as to look for infection that tracks along fascial planes and tendon sheaths. Several trips to the operating theatre may be required. It is incumbent upon the surgeon to plan the incisions with regard to foot function and ultimate surgical repair.

CASE STUDY
Infection

A 53-year-old man with a history of schizophrenia, poorly controlled type 2 diabetes of 9 years' duration and a dense peripheral neuropathy with sensory loss extending above the ankle, had developed his own unique method for sensory testing. He used a lit cigarette to establish the level of sensory loss on his lower legs and had several circular scars and burns in various stages of healing. He was followed for routine care in the diabetic foot clinic, for treatment of an intractable plantar keratosis beneath his right 5th metatarsal head. Having missed his last scheduled appointment, the patient finally returned to clinic with the chief complaint of pain in his right foot that had started 2 weeks before.

Physical examination revealed swelling, cellulitis and increased skin temperature of the forefoot with an abscess overlying the 5th metatarsophalangeal joint (Fig. 8.2a). Inspection of the plantar aspect of the foot revealed a thick callus, with haemorrhage, beneath the 5th metatarsal head. The 5th toe appeared bluish-black. Laboratory studies revealed an elevated white blood cell count, 17 000/μL, and elevated fasting serum glucose, 203 mg/dL (11.3 mmol/L). Radiographic evaluation revealed subluxation of the 5th metatarsophalangeal joint. The patient was admitted to the hospital for surgical management and intravenous antibiotics. At the bedside, the abscess was incised and drained, revealing a purulent-sanguineous discharge (Fig. 8.2b). Using a sterile probe, the dorsal wound was found to communicate with the plantar aspect of the joint, and exited through the bottom of the foot. Wound cultures revealed a single organism, *Staphylococcus aureus*, sensitive to penicillinase-resistant penicillins. Five days following hospital admission, the patient's white blood cell count was 12 600/μL, dry gangrene had clearly



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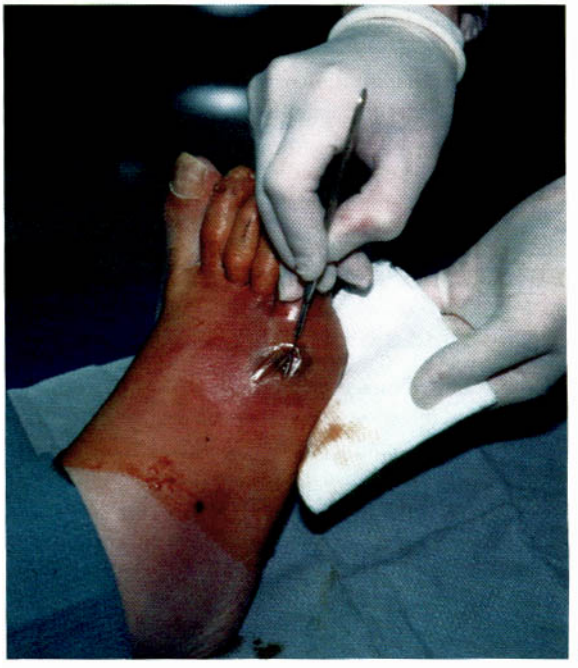


(b)

Fig. 8.2 (a) Abscess with cellulitis, right foot. Tense bulla overlying the 5th metatarsophalangeal joint. (b) Incision and drainage, at the bedside.

demarcated at the base of the 5th toe, and the cellulitis and swelling had subsided. Fifth ray amputation was advised: however, the patient did not immediately consent to surgery. The surgical management will be continued later in the section covering amputations and 5th ray resections.

CASE STUDY
Limb-threatening deep plantar space infection

A 69-year-old Afro-American man with type 2 diabetes of 22 years' duration and peripheral vascular disease, presented to the diabetic foot clinic with a limb-threatening deep plantar space infection of his left foot. Physical examination revealed an erythematous, swollen foot with fluctuance in the medial arch. There was a full-thickness ulcer noted beneath the 1st metatarsal head, with pus present in the wound. The ulcer probed to bone. He was febrile with an elevated WBC count and elevated serum glucose 400 mg/dL (22.2 mmol/L). The patient was admitted to hospital, and preoperative laboratory testing, chest X-ray, electrocardiogram and medical consultation were obtained. Empiric intravenous antibiotic therapy was initiated and the patient was taken to the operating theatre the same evening for extensive incision and drainage of his foot. The incision extended from the 1st metatarsal head to his ankle (Fig. 8.3).

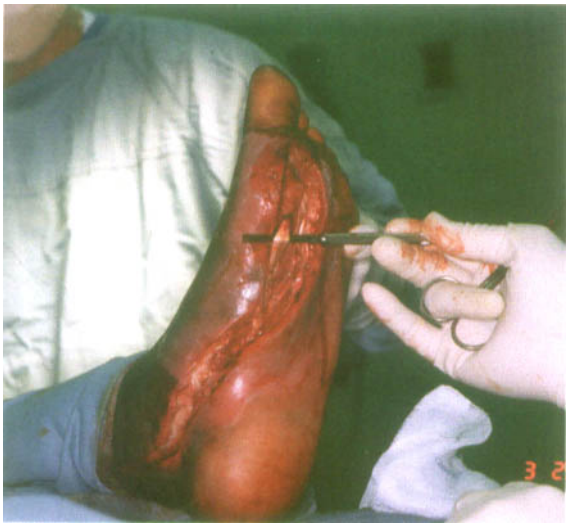


Fig. 8.3 Deep plantar space infection, left foot. Incision and drainage performed in the operating room. The flexor hallucis longus tendon is visible in the wound.

Hammer toe correction

Patients with diabetes often develop one or more digital contractures. Flexion contracture of the toes develops as a result of biomechanical imbalance between the long flexor and extensor tendons to the toe. The intrinsic muscles of the foot, the interossei and lumbricales, function to stabilize the toes on the weightbearing surface. Weakness of these muscles, secondary to motor neuropathy, results in the development of hammer toes and, in some cases, claw toes.

Hammer toe

A hammer toe is characterized by hyperextension of the toe at the metatarsophalangeal joint, and flexion contracture of the toe at the proximal interphalangeal joint. The resulting deformity, like a swan's neck, results in retrograde force on the metatarsal head, causing increased plantar pressure, metatarsalgia, callus formation and eventually ulceration. Friction and pressure caused by the shoe on a prominent proximal interphalangeal joint, results in the development of a corn and, eventually, an ulcer. Pressure at the tip of the flexed toe may result in a distal corn and eventually an ulcer (Fig. 8.4a).

Claw toes

Claw toes are characterized by flexion contracture at both the proximal interphalangeal joint and distal interphalangeal joint. Claw toes are often associated with cavus (high arched) feet (Fig. 8.4b).

Mallet toe

Mallet toe is a digital deformity characterized by flexion at the distal joint. Risk of ulceration is associated with each of these three digital deformities, at the tips of the toes and over the prominent interphalangeal joints.

Surgical correction of digital deformities may be indicated in high-risk patients with loss of protective sensation. Surgical intervention becomes a more pressing issue once the patient has developed ulceration and/or infection. Chronic, recurrent plantar ulceration beneath a metatarsal head may require correction of the digital deformity to relieve pressure beneath the metatarsal head (Fig. 8.4c).

Proximal interphalangeal joint arthroplasty

Proximal interphalangeal joint arthroplasty is indicated for correction of rigidly contracted hammer toes with

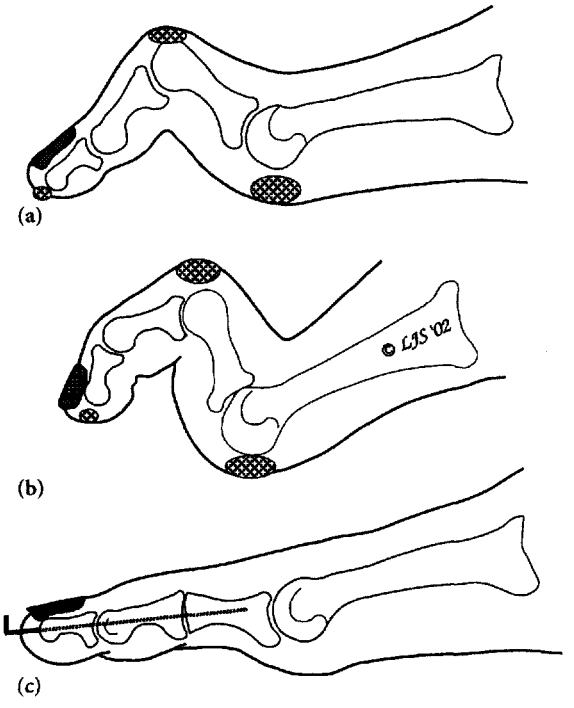


Fig. 8.4 Digital deformities. Areas of friction and elevated pressure are identified beneath the metatarsal head, over the prominent proximal interphalangeal joint, and at the tip of the toe. (a) Hammer toe. (b) Claw toe. (c) Surgical correction of hammer toe deformity with proximal interphalangeal joint arthroplasty and internal fixation with a Kirschner wire.

or without ulceration at the tip of the toe, or over the prominent proximal interphalangeal joint (Fig. 8.5a).

Technique

The procedure is performed in the operating theatre under local anaesthesia and sedation, with an ankle tourniquet for haemostasis. If an ulcer or corn is present over the proximal interphalangeal joint, it is excised, using two converging semi-elliptical incisions. The incisions are carried down to the subcutaneous tissue and the ulcer is excised. The extensor tendon is exposed (Fig. 8.5b) and transected at the level of the interphalangeal joint. The tendon is retracted proximally exposing the head and neck of the proximal phalanx. The medial and lateral collateral ligaments are freed using a Beaver No. 64 mini-blade or scalpel with a No. 15 blade. The head of the proximal phalanx is then transected at the surgical neck using a double action bone cutting forceps or power saw (Fig. 8.5c). The bone is examined for evidence of osteomyelitis which, if present, would dictate removal of additional bone. The extensor tendon is then repaired with absorbable sutures at the level of the proximal interphalangeal joint. If there is evidence of extension of the toe at the metatarsophalangeal joint, then the extensor tendon can be lengthened or transected, followed by a dorsal transverse metatarsophalangeal capsulotomy. A McGlamry elevator is helpful to release adhesions of the plantar structures. If the metatarsophalangeal joint contracture has not been corrected, then partial metatarsal head resection may be required. In the presence of infection the wound should be packed open, with a return to the operating theatre for delayed primary closure within a week. For clean wounds, the skin is closed with 5–0 nylon simple interrupted sutures (Fig. 8.5d) The use of a 0.45 Kirschner wire, placed across the joint, is optional. When used to maintain the corrected position of the toe, the wire is placed in a retrograde fashion, from the proximal interphalangeal joint, out through the tip of the toe. It is then driven back into the proximal phalanx. It is wise to reserve the use of internal fixation for clean, elective cases.

Dressings and postoperative care

Dressings consist of non-adherent fine mesh gauze (petrolatum, 3% Xeroform or Adaptic) and a dry sterile gauze bandage with the toe splinted in its corrected position. A surgical shoe is dispensed and the patient is able to ambulate as tolerated. The first postoperative dressing change is within 1 week. Sutures are removed in 10–14 days, and if a Kirschner wire was used it is removed at 3 weeks. The patient is able to return to a roomy shoe with a broad toe box after 3–6 weeks, as oedema resolves.

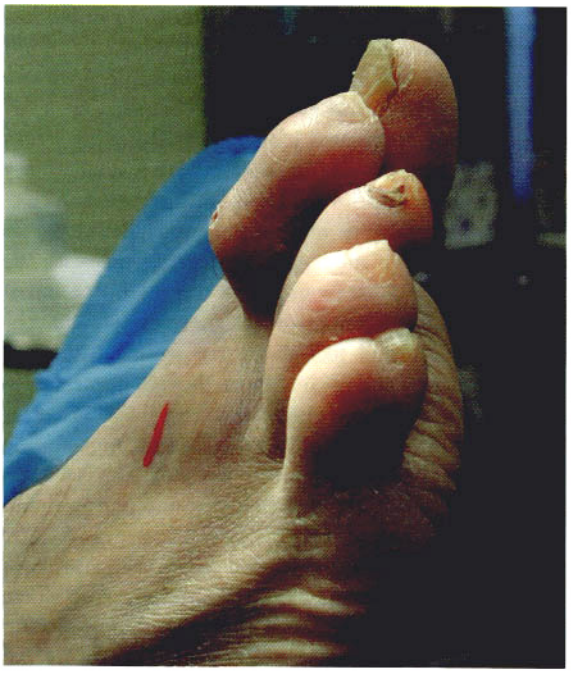
CASE STUDY

Rigid hammer toe deformity

A 64-year-old man, a retired pilot, with type 2 diabetes of 16 years' duration, was followed regularly in the diabetic foot clinic for treatment of a rigid hammer toe deformity of his right 2nd toe, with recurrent ulceration over the proximal interphalangeal joint. The patient underwent an elective proximal interphalangeal joint arthroplasty, with lengthening of the extensor hallucis longus tendon and dorsal capsulotomy of the metatarsophalangeal joint. A Kirschner wire was not used in this case, and the toe was splinted in its corrected alignment. The postoperative course was uncomplicated.

Mallet toe correction—distal interphalangeal joint arthroplasty

Mallet toe correction is indicated for lesions that develop at the tip of the toe. In the presence of mallet toe deform-



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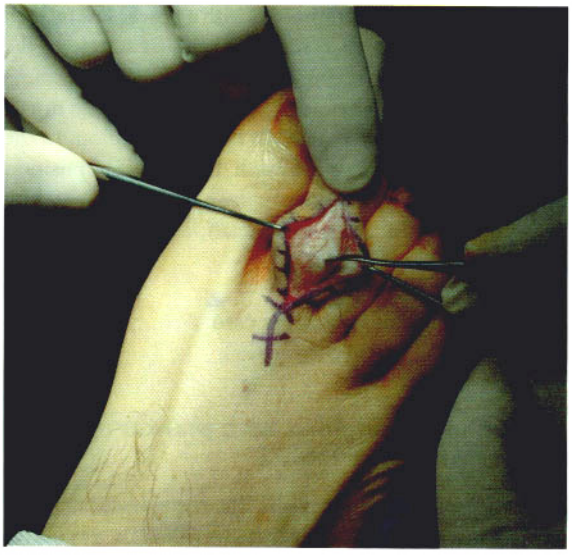

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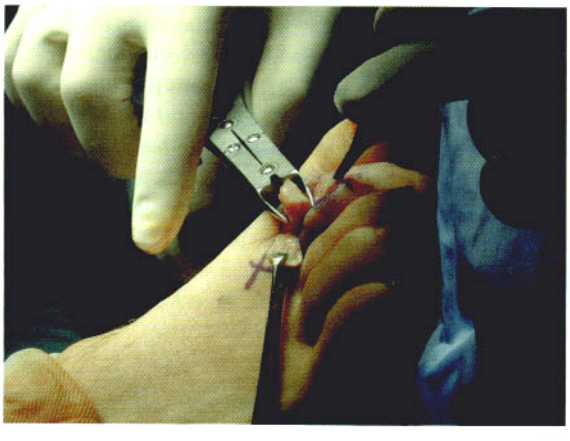

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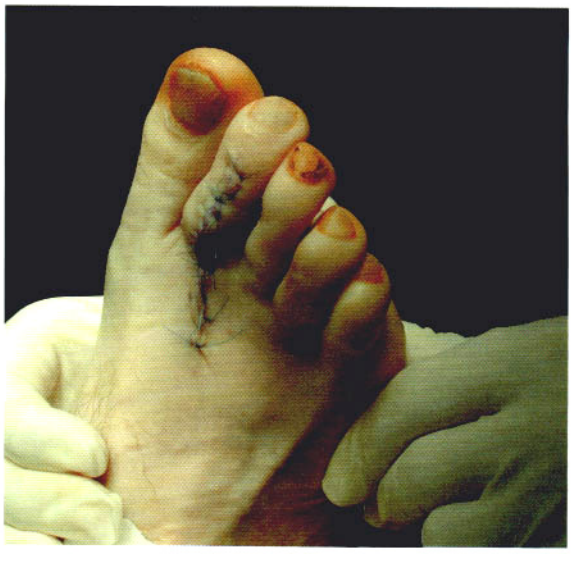


(d)

Fig. 8.5 Surgical correction of hammer toe deformity. (a) Preoperative appearance of a rigid hammer toe, 2nd toe, right foot. Notice the very prominent deformity at the proximal interphalangeal joint. (b) The extensor digitorum longus tendon and joint capsule are identified overlying the proximal interphalangeal joint. (c) Removal of the head of the proximal phalanx with a double action bone cutting forceps. (d) Immediate postoperative appearance.

ity the tip of the toe is traumatized with every step that the patient takes. The initial lesion is a callus, which eventually progresses to a preulcerative lesion (haemorrhage within the callus) and then to a full-thickness ulcer that may probe to bone.

Technique

The procedure is performed in the operating theatre under local anaesthesia, with a Penrose drain applied as a tourniquet at the base of the toe. No tourniquet is used if there is a question of vascular compromise. Two semi-elliptical incisions are made in a transverse manner over the distal interphalangeal joint of the toe. The incisions are carried down through the skin, the extensor tendon and joint capsule, and these structures are removed. The interphalangeal joint is identified and the collateral ligaments are severed using a Beaver No. 64 mini-blade. The blade is kept close to bone at all times. The distal aspect of

the middle phalanx is transected with a power saw or with a bone-cutting forceps. It may be necessary to release the long flexor tendon, and this can be done through the same dorsal incision. The deformity is reduced and the dorsal capsule and skin are repaired in the usual fashion. In the absence of ulceration or infection, the corrected position of the toe is maintained by placing a 0.45 Kirschner wire across the joint in a retrograde manner, as described for hammer toe correction.

Dressings and postoperative care

Same as for hammer toe correction.

Hallux limitus/rigidus with plantar ulceration of the hallux

Limited joint mobility of the 1st metatarsophalangeal joint, with decreased range of dorsiflexion, results in elevated plantar pressure beneath the hallux. Repetitive moderate stress on the skin is observed clinically by the formation of a callus beneath (or plantar medial to) the hallux interphalangeal joint (Fig. 8.6). Haemorrhage within the callus represents a preulcerative condition that requires regular debridement and footwear modification. The natural history for hallux interphalangeal joint lesions is for the preulcerative condition to progress to a full-thickness ulcer and eventually to amputation. Correlation between elevated plantar pressure and a lesion beneath the hallux can be demonstrated qualitatively using a Harris footprint mat, or quantified using an electronic gait platform or in-shoe measuring device. Location of the peak plantar pressure corresponds with the location of the callus or ulcer. All too often, the initial treatment for this condition totally ignores the pathomechanical aetiology and focuses on the wound. Although local wound care and off-loading of the foot may result in healing of the ulcer, this outcome is short lived. The ulcer inevitably recurs and becomes a chronic non-healing wound. The obvious risk, for a diabetic patient, is wound infection, extension of infection to bone and eventual amputation of the hallux.

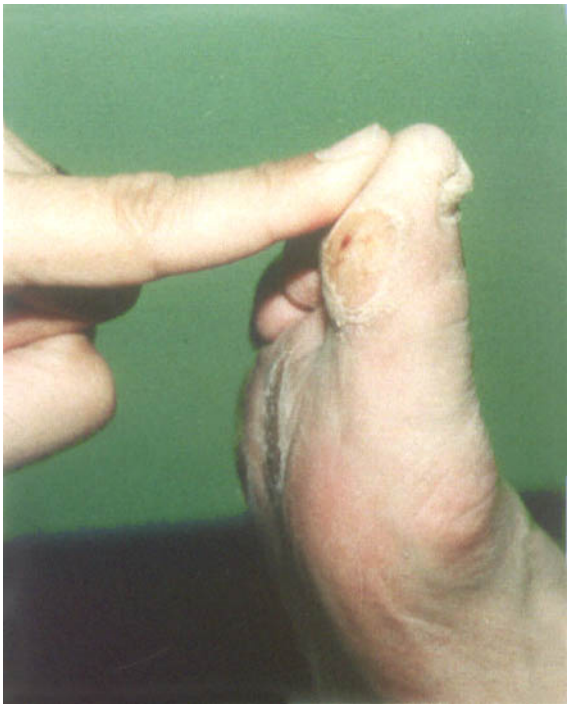


Fig. 8.6 Hallux limitus. There is a callus with preulcerative changes on the plantar medial aspect of the great toe.

Surgical treatment for hallux interphalangeal joint ulcers is directed at increasing the dorsiflectory range of motion of the hallux. Some authors advocate hallux interphalangeal joint arthroplasty and report an overall success rate of 91%, with only minor complications. Most authors however, advocate a procedure, to increase dorsiflectory range of motion, at the level of the 1st metatarsophalangeal joint. Keller arthroplasty is the procedure most often cited for surgical correction of recalcitrant interphalangeal joint ulceration of the hallux in adults. Downs reported successful results of this procedure in a series of patients ranging in age from 30 to 65 years. Postoperatively all ulcers healed promptly with no recurrence at follow-up after 2–5 years. Dannels reported similar results in a series of Native American Indians with diabetes, with an age range of 39–58 years.

Keller resectional arthroplasty of the 1st metatarsophalangeal joint

Technique

This procedure can be performed under regional ankle block anaesthesia, with an ankle tourniquet. A dorsal longitudinal incision is made over the 1st metatarsophalangeal joint just medial to the extensor hallucis longus tendon. The incision starts at the neck of the proximal phalanx and extends ~ 2 cm proximal to the metatarsal head. Skin hooks are used to retract the skin edges, small bleeders are clamped and bovied, and the incision is then carried deep through the capsule down to bone. Subperiosteal dissection is carried out over the proximal phalanx. The joint capsule is reflected, allowing direct visualization

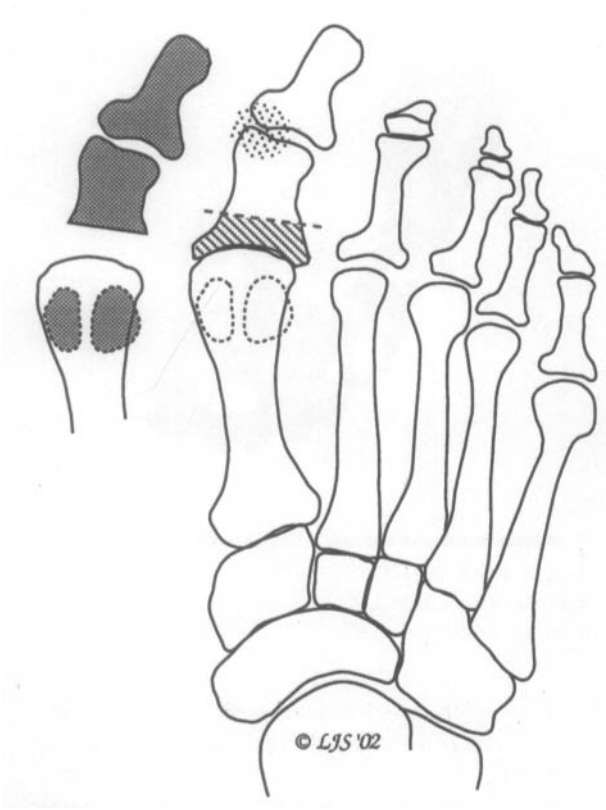


Fig. 8.7 Keller resectional arthroplasty of the 1st metatarsophalangeal joint, before and after removal of the proximal one-third of the base of the proximal phalanx.

of the metatarsophalangeal joint. The collateral ligaments are cut using a Beaver No. 64 mini-blade or No. 15 blade and the proximal one-quarter to one-third of the proximal phalanx is transected, perpendicular to the long axis of the phalanx, with a power saw (Figs 8.7, 8.8). The most difficult part of this procedure is removing the phalangeal base. The bone is grasped with a bone clamp and the intrinsic muscle attachments, for the flexor hallucis brevis and the adductor hallucis, are carefully freed using a Beaver No. 64 mini-blade. Care must be taken to avoid cutting the flexor hallucis longus tendon. The wound is irrigated with normal sterile saline and a piece of Gelfoam® sponge (haemostatic absorbable gelatin) is placed in the void created by removal of the phalangeal base. The joint capsule is closed with 3–0 absorbable sutures in a simple interrupted fashion. If possible, the capsule should be purse stringed, interposing soft tissue between the metatarsal head and the phalangeal base. The skin is closed with a 4–0 absorbable suture in a running subcuticular fashion and Steri-Strips® are placed across the incision.

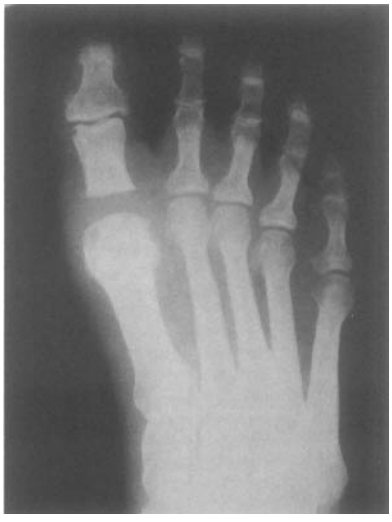


Fig. 8.8 Postoperative anteroposterior radiograph reveals resection of the proximal one-third of the phalangeal base of the hallux.

The use of one or two Kirschner wires to maintain the hallux position is at the surgeon's discretion. I have not found it necessary to use Kirschner wires, and prefer splinting, with early passive range of motion exercises to maintain hallux dorsiflexion.

Dressings and postoperative care

Dressings consist of non-adherent fine mesh gauze (petrolatum, 3% Xeroform™ or Adaptic™), and a fluffy dry sterile compression gauze bandage, with the hallux splinted in its corrected position. A surgical shoe is dispensed. The patient is instructed to rest at home, remain non-weightbearing and to elevate his feet for 48 h. He is then allowed partial weightbearing in a surgical shoe with crutches or a walker. The first postoperative dressing change is within 1 week. Dressings are changed weekly for 3–4 weeks postoperatively. Sutures are removed in 14–21 days, and the patient is allowed to return to a roomy shoe with a broad toe box in 3–6 weeks.

Advantages

- Performed under local anaesthesia with minimal risk
- Minimally debilitating
- Increases range of motion at the metatarsophalangeal joint
- Decompresses the hallux interphalangeal joint ulcer and allows for rapid healing
- Patients may begin protected weightbearing immediately.

Disadvantages

- Reduction in plantar flexion strength of the great toe
- Loss of toe purchase
- Weakness during the toe-off phase of gait
- Shortening of the great toe.

Complications

- Postoperative infection
- Development of lesser metatarsalgia
- Cock-up deformity of the hallux
- Digital fracture(s)
- Charcot's osteoarthropathy (rarely).

CASE STUDY

Hallux limitus

A 46-year-old man with type 2 diabetes of 12 years' duration, documented peripheral neuropathy with loss of protective sensation and history of a chronic non-healing ulcer on the plantar medial aspect of his right hallux interphalangeal joint, had limited joint mobility in the 1st metatarsophalangeal joint, with approximately 10° of hallux dorsiflexion (Fig. 8.9). Quantitative plantar pressure measurements revealed markedly elevated peak plantar pressure, 95 N/cm^2, beneath the great toe (Fig. 8.10). The maximum peak pressure corresponded to the precise location of his ulcer. Radiographs revealed no evidence of osteomyelitis. Conservative treatment consisted of local wound care, total-contact casting, a walking brace and extra-depth shoes with total-contact orthoses. Over a period of several months the ulcer showed some improvement with off-loading: however, it never completely healed. The ulcer became infected on two occasions with *Staphylococcus aureus*. The infections resolved promptly with oral antibiotics.

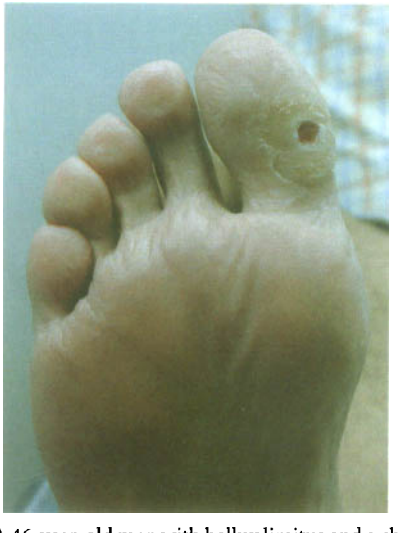


Fig. 8.9 A 46-year-old man with hallux limitus and a chronic ulcer beneath the hallux interphalangeal joint, right foot.

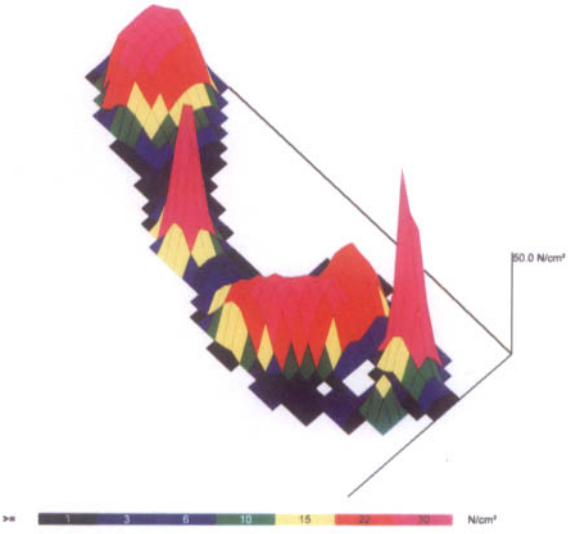


Fig. 8.10 EMED three-dimensional display of peak plantar pressures beneath the right foot. The peak pressure beneath the hallux was 95 N/cm^2.

It became clear that the ulcer would not heal without surgical intervention, and that he was at high-risk for future infections and amputation of his hallux. Keller resectional arthroplasty of the 1st metatarsophalangeal joint was proposed as a salvage procedure and the patient consented. Within 4 weeks following the procedure, the hallux ulcer was completely healed. A complication occurred on postoperative day 18, with development of pain and swelling at the base of the right 2nd toe. The patient indicated that his foot began to hurt as he was walking to his car. X-rays revealed a non-displaced fracture of the proximal phalangeal base of the 2nd toe. The 2nd and 3rd toes were splinted, the patient was placed in a walking brace, and the foot healed without further complications.

Sesamoidectomy

Sesamoidectomy is indicated for the treatment of a discrete intractable lesion, beneath the 1st metatarsal head, that fails to heal or remain healed with a conservative approach to treatment (local wound care, total-contact casting or attempts to off-load the forefoot with orthoses

and custom footwear). Sesamoidectomy is also indicated for the curative treatment of osteomyelitis of the sesamoid bone. This procedure is appropriate for the treatment of neuropathic patients with evidence of increased plantar pressure beneath the 1st metatarsal head. Weightbearing radiographs taken with a radio-opaque open circle marker placed over the ulcer will confirm the relationship between an enlarged or arthritic sesamoid bone and the plantar lesion. There may also be evidence of a plantar flexed 1st metatarsal associated with a cavus foot deformity. Excision of the tibial sesamoid, fibular sesamoid or both sesamoids may be indicated.

Technique

Surgical approach to the tibial sesamoid can be either medial or plantar. A low medial longitudinal incision is centred over the 1st metatarsophalangeal joint between the dorsal and plantar cutaneous nerves to the hallux. This incision is deepened to the level of the joint capsule and the capsule is incised in the same plane. The tibial (medial) sesamoid is visualized within the joint capsule beneath the metatarsal head. The capsule is grasped with a clamp and the sesamoid is shelled out with a Beaver No. 64 mini-blade or No. 15 scalpel blade. The fibular (lateral) sesamoid is more difficult to reach through a medial incision, and may be more accessible from a dorsal longitudinal approach, over the first webspace. This is a reasonable approach if the sesamoid is located in the intermetatarsal space.

A plantar approach is indicated for excision of the ulcer, and allows for direct visualization of both sesamoid bones.

Dressings and postoperative care

The decision to close the wound primarily or to pack it open will vary with each case. Infected or contaminated wounds should be packed open, and either allowed to heal by secondary intention or brought back to the operating theatre for delayed wound closure. Dressings consist of a dry sterile compression gauze bandage with the hallux splinted in its proper alignment. The patient is instructed to rest at home, remain non-weightbearing and elevate his feet for 48 h. If the wound was packed open, the dressing is changed on the first or second postoperative day. If possible, arrangements should be made for a visiting nurse to perform the necessary daily dressing changes. The patient is then allowed limited protected weightbearing in a walking brace. The first postoperative visit is scheduled within 1 week with weekly visits scheduled until the wound is healed. Once healed, the patient will require therapeutic shoes and insoles.

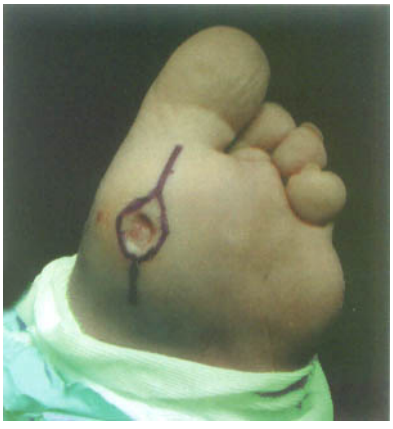


Fig. 8.11 Preoperative appearance of a chronic ulcer beneath the tibial (medial) sesamoid, left foot.

Advantages

- The procedure is performed under local anaesthesia with minimal risk
- Surgical simplicity
- Low morbidity.

Disadvantages

- Possibility of developing a hallux hammer toe or hallux abducto valgus deformity.

Complications

- Infection, wound dehiscence and hallux hammer toe.

CASE STUDY
Removal of sesamoids

A 69-year-old man with type 2 diabetes of 22 years' duration and peripheral neuropathy had a history of chronic full-thickness ulceration beneath his left 1st metatarsal head. The ulcer did not probe to bone (Fig. 8.11). Radiographs revealed a hypertrophic tibial sesamoid (Fig. 8.12). Conservative treatment was employed for 4 months with no improvement in his condition. The patient was offered the option of surgical treatment and he consented. In this case, the plantar ulcer was excised and both medial and lateral sesamoids were removed (Fig. 8.13a,b). The wound was packed open and allowed to heal by secondary intention.

Lesser metatarsal osteotomy

Dorsiflectory metatarsal osteotomies are performed for the treatment of lesser metatarsalgia, most often for

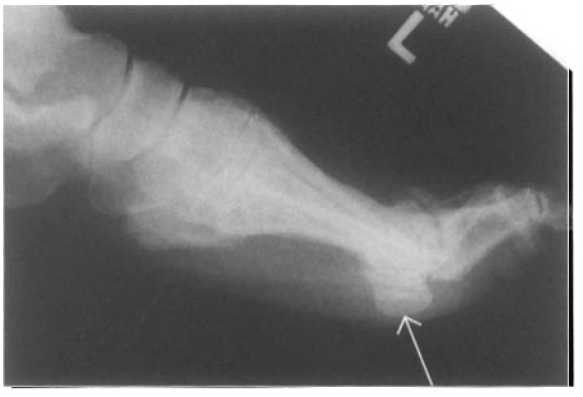


Fig. 8.12 Lateral radiograph of the left foot, reveals a hypertrophic tibial sesamoid. The ulcer was located beneath the sesamoid.

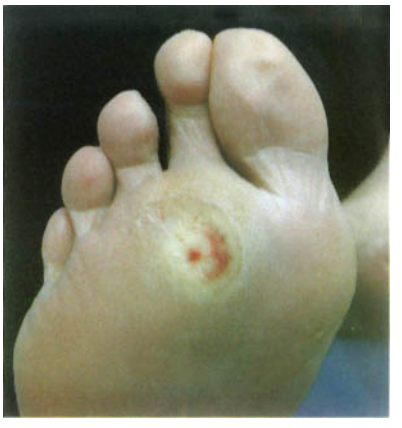


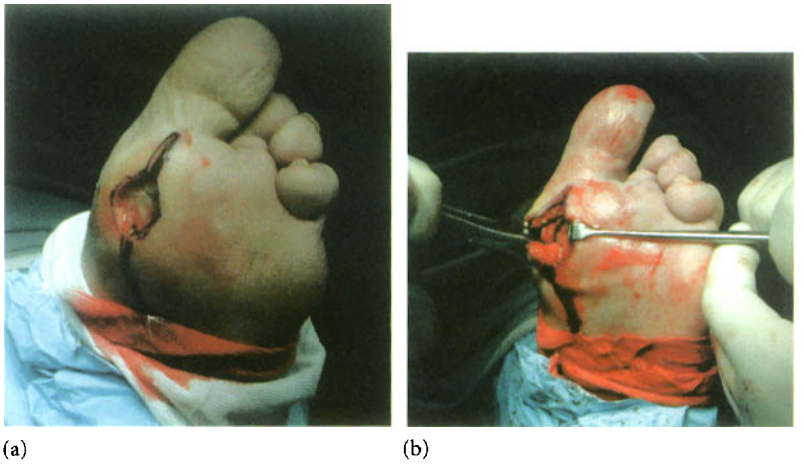
Fig. 8.14 Chronic intractable plantar keratosis beneath the 2nd metatarsal head. The callus has been debrided, revealing preulcerative haemorrhage within the skin. This is an indication for lesser metatarsal osteotomy.

intractable plantar keratoses (IPKs), when non-surgical methods have failed. These procedures are controversial and are often plagued by postoperative complications such as transfer lesions, non-union or malunion, and floating toes. Infection and screw failure have also been reported. Caution should be exercised when considering these procedures for neuropathic individuals. Metatarsal osteotomy is not advised in the presence of infection or full-thickness ulceration beneath the metatarsal head.

Figure 8.14 illustrates a chronic IPK with preulcerative haemorrhage within the callus and a long 2nd metatarsal. This is an appropriate indication for lesser metatarsal osteotomy. Multiple lesser metatarsal osteotomies have been described to include transverse, oblique and closing wedge procedures. Two of the more popular distal procedures are the vertical chevron metatarsal osteotomy and the Weil shortening osteotomy. Both procedures require internal fixation.

The Weil osteotomy

The Weil shortening osteotomy is a distal lesser metatarsal procedure, designed to shorten one or more of the central metatarsals (2nd, 3rd and sometimes the 4th) without elevating or depressing the metatarsal head. The head moves proximal to the existing plantar callus, and decompresses the metatarsophalangeal joint. The procedure should be reserved for cases that fail conservative treatment, and only used in cases where the affected metatarsals are comparatively long. Complications are



(a) (b)

Fig. 8.13 Sesamoidectomy. (a) Intraoperative photograph, the ulcer has been excised. (b) The hypertrophic tibial (medial) sesamoid has been grasped with a bone clamp and is being removed from the wound.

similar to those associated with other metatarsal osteotomies.

Technique

The procedure is performed in the operating theatre under local anaesthesia with an ankle tourniquet. A dorsal longitudinal incision is made over the metatarsophalangeal joint and then deepened to the joint capsule. The capsule is dissected between the extensor digitorum longus and the extensor digitorum brevis. The capsule is reflected, allowing for release of the collateral ligaments. Two small Hohmann retractors are inserted under the metatarsal neck to provide sufficient exposure to the metatarsal head. The toe is plantar flexed and the osteotomy is performed with a long, thin sagittal saw blade. The osteotomy cut begins at the distal dorsal edge of the articular cartilage and is directed proximally, oblique to the metatarsal shaft, and as parallel as possible to the sole of the foot. The distal fragment is displaced proximally, 3–5 mm, and fixed with a single self-drilling, self-tapping partially threaded 2.0 mm screw. The screw is directed from dorsal-proximal to plantar-distal. The bone peak is then resected with a rongeur and smoothed with a burr (Fig. 8.15a–c). The joint capsule is closed with 3–0 absorbable (Dexon or Vicryl) sutures and the skin is closed with 4–0 absorbable subcuticular sutures, or nylon simple interrupted sutures.

Dressings and postoperative care

Standard dressings are utilized. The patient is allowed to ambulate with crutches and partial weightbearing, in a surgical shoe. Sutures are removed in 10–14 days and patients can return to their normal footwear in 2–4 weeks, as dictated by the clinical course.

Advantages

- Simple and reliable procedure
- Stability of the osteotomy with a large area of bone to bone contact
- Pressure relief beneath the metatarsal head
- Helpful for reduction of dorsally dislocated metatarsophalangeal joints
- Pressure relief beneath the metatarsal head
- Early return to weightbearing.

Complications

- Transfer lesions
- Recurrent symptomatic plantar keratosis
- Infection
- Floating and stiff toes.

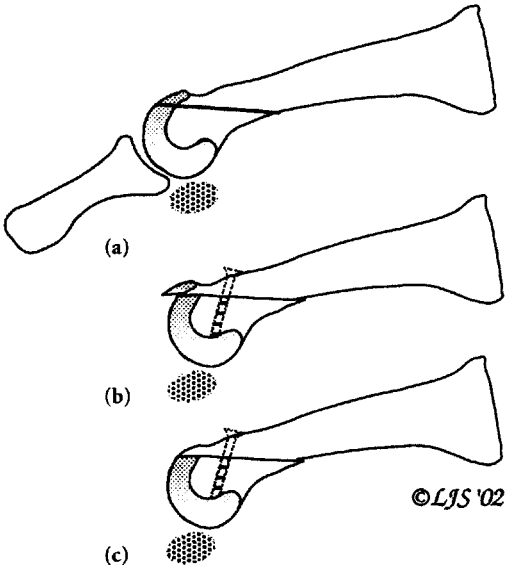


Fig. 8.15 The Weil lesser metatarsal shortening osteotomy. (a) The lesser toe is plantarflexed, and the oblique osteotomy cut begins at the distal dorsal edge of the articular cartilage. (b) Proximal displacement of the metatarsal head, approximately 3–5 mm. Note that the head of the metatarsal is now proximal to the plantar callus. Fixation is with a single 2.0 mm screw. (c) Resection and smoothing of the bone peak.

Metatarsal head resection (Fig. 8.16)

Resection of a single metatarsal head is indicated for one of the following reasons, when non-surgical methods fail to achieve the desired result:

- For removal of infected bone, in the case of chronic osteomyelitis
- For decompression of a plantar ulcer, as an alternative to metatarsal osteotomy, to facilitate wound healing
- For deformity of the 5th metatarsal with painful callus on the plantar or lateral aspect of the metatarsal head.

Resection of the metatarsal head can be performed through a dorsal incision over the metatarsophalangeal joint, or in the presence of a deep plantar ulcer, the ulcer and the metatarsal head can both be excised through a plantar approach.

Fifth metatarsal head resection

This procedure is well suited for older sedentary individuals, and for patients with osteopenia or osteomyelitis of the metatarsal head, where a transpositional osteotomy is not appropriate. Although transfer lesions (callus or ulcer) have been reported to occur beneath adjacent

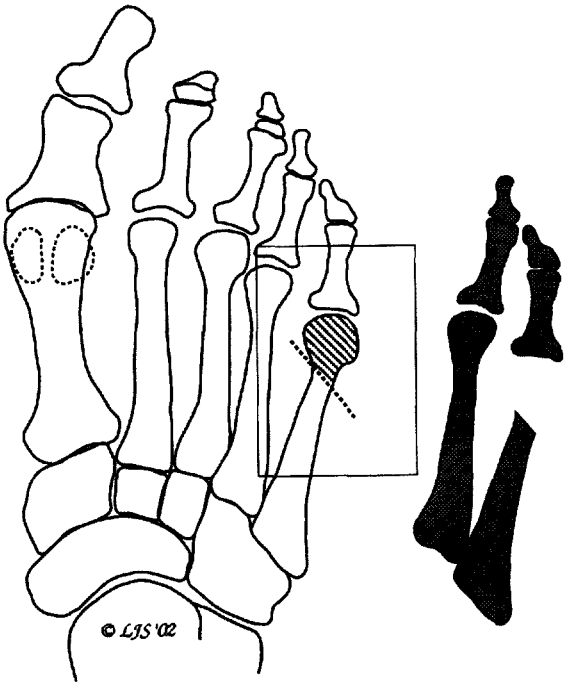


Fig. 8.16 Technique for 5th metatarsal head resection. Before and after removal of the metatarsal head. Note that the osteotomy is angled in an oblique manner at the surgical neck of the metatarsal.

metatarsal heads, following metatarsal head resections, this is not a frequent problem with 5th metatarsal head resections.

Technique

Fifth metatarsal head resection is performed under local anaesthesia with IV sedation. An ankle tourniquet is used for haemostasis. A 4-cm dorsal longitudinal incision is made over the 5th metatarsophalangeal joint and shaft, just lateral to the extensor digitorum longus tendon. The incision is carried down to fascia, the skin edges are retracted, and the incision is then continued through joint capsule and deep to the periosteum. The joint is visualized, collateral ligaments are cut with a Beaver miniblade, and the metatarsal is cut in an oblique manner, at the surgical neck, from distal-medial to proximal-lateral. The metatarsal head is removed, and the wound is irrigated. Gelfoam® is placed in the void, and the capsule is closed with 3–0 absorbable sutures in a simple interrupted fashion. The skin is closed with 4–0 nylon sutures, in a simple interrupted and horizontal mattress fashion. Drains are generally not necessary.

Dressings and postoperative care

Dressings consist of non-adherent fine mesh gauze (petrolatum, 3% Xeroform™ or Adaptic™), and a fluffy dry sterile compression gauze bandage. A surgical shoe is dispensed. The patient is instructed to rest at home, remain non-weightbearing and to elevate his feet for 48 h. He is then allowed partial weightbearing in a surgical shoe with crutches or a walker. The first postoperative dressing change is scheduled within 1 week. Dressings are changed weekly for 3–4 weeks postoperatively. Sutures are removed in 14–21 days, and the patient is allowed to return to a roomy shoe with a broad toe box in 3–4 weeks.

Advantages

- Can be performed under local anaesthesia with minimal risk
- Simple procedure
- Can be used for osteomyelitis
- Can be closed primarily
- Rapid return to weightbearing
- Limited disability, and rapid recovery.

Disadvantages

- Possibility of a transfer lesion (callus or ulcer).

Complications

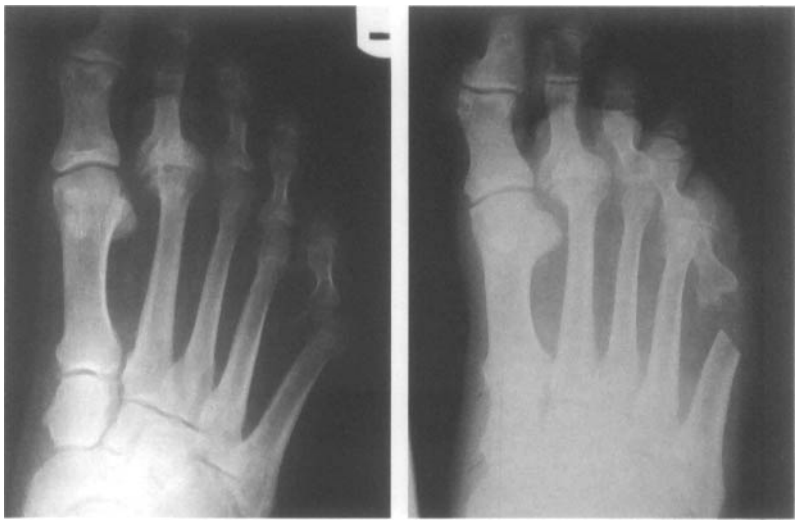
- Postoperative infection
- Delayed healing
- Regrowth of the transected metatarsal with recurrence of the lesion.

CASE STUDY
5th metatarsal head resection

An 80-year-old active man with peripheral neuropathy and loss of protective sensation presented to clinic with a prominent, painful tailor's bunion that could not be satisfactorily accommodated by footwear. The patient had a 5th metatarsal head resection performed 2 years earlier, for correction of a similar condition affecting his right foot. He was very satisfied with the results and returned for surgical correction of his left foot. The surgical procedure and postoperative course were uneventful. The surgical outcome was excellent (Fig. 8.17a,b).

Achilles tendon lengthening

Increased pressure on the plantar aspect of the forefoot has been shown to be associated with limited joint mobility and with equinus deformity of the ankle. In the



(a) (b)

Fig. 8.17 (a) Preoperative anteroposterior radiograph reveals deformity of the right 5th metatarsophalangeal joint with lateral bowing and prominence of the 5th metatarsal head. (b) Postoperative anteroposterior radiograph reveals resection of the 5th metatarsal head.

presence of peripheral neuropathy, elevated pressure beneath one or more metatarsal heads can result in the development of ulceration. Ankle equinus may also contribute to the development of Charcot's osteoarthropathy with collapse of the mid-foot or avulsion fracture of the posterior process of the calcaneus. Armstrong and coworkers, at the University of Texas Health Science Centre at San Antonio, reported on a study to determine the degree to which pressure on the plantar aspect of the forefoot is reduced following percutaneous lengthening of the Achilles tendon in high-risk subjects with diabetes. They demonstrated that peak plantar forefoot pressures were reduced by approximately 27% following percutaneous Achilles tendon lengthening. These authors suggest that lengthening of the Achilles tendon, in high-risk patients with diabetes, may decrease the likelihood of ulceration and may increase the efficacy of pressure-reduction modalities such as casts or braces. In fact, this procedure facilitates the healing of recalcitrant forefoot plantar ulcers.

CASE STUDY

Percutaneous lengthening of Achilles tendon

A 55-year-old African-American male with poorly controlled type 2 diabetes (HbA_{1c} = 10.6%) of 8 years' duration, and dense peripheral neuropathy was seen regularly in the diabetic foot clinic for treatment of a chronic nonhealing full-thickness ulcer located beneath the 2nd and 3rd metatarsal heads of his left foot. The ulcer did not probe to bone. Treatment consisted of surgical wound debridement, total-contact casting, walking brace and a variety of topical wound healing agents.

Diagnostic studies

- Serial X-rays were negative for osteomyelitis: however, they revealed a long 3rd metatarsal
- MRI was unremarkable
- Non-invasive vascular studies were normal with evidence of strong pedal pulses
- EMED plantar pressure measurements revealed markedly elevated peak plantar pressure, 117 N/cm^2, located beneath the 2nd to 3rd metatarsal heads, right foot
- Arthrometric evaluation revealed ankle joint dorsiflexion was –6° with the knee extended, –2° with the knee flexed on the right ankle.

The proposed treatment for this patient was a percutaneous lengthening of the right Achilles tendon, by triple hemisection, under local anaesthesia. The patient consented to this minimally invasive procedure. Within 3–4 weeks the plantar ulcer was completely healed and has remained healed for the last 3 years. Peak pressure at the site of ulcer was significantly reduced to 42 N/cm^2. The surgical technique is described below.

Percutaneous Achilles tendon lengthening—triple hemisection

Technique

This procedure is indicated for correction of mild to moderate gastrocnemius-soleus ankle equinus. Three

Fig. 8.18 (a) Percutaneous lengthening of the Achilles tendon—triple hemisection. Proposed cuts in tendon. From Sanders (1997) with permission from Elsevier Science. (b) Dorsiflexion of foot after triple hemisection.

hemisections of the tendon are performed, two medial and one lateral. The distance between the hemisections is determined by the overall size of the tendon and the amount of lengthening desired. An alternative procedure, attributed to Hoke, incorporates two posterior and one anterior hemisections of the Achilles tendon, and is performed in the frontal plane through a medial approach.

The patient is placed in a prone position on the operating room table. Local anaesthesia is infiltrated just above the Achilles tendon on the back of the leg. A tourniquet is not required for this procedure. The surgeon stands at the end of the operating table facing the foot, which hangs over the end of the table. The plantar surface of the foot is placed against the abdomen of the surgeon and gently dorsiflexed while palpating the Achilles tendon. A skin marker is used to define the borders of the tendon, from its insertion into the calcaneus, to its proximal myotendinous junction. The proposed cuts in the tendon are drawn on the skin, as shown in Fig. 8.18a. These marks help the surgeon to remember the direction of the cuts. The distal cut is made 1.0–2.5 cm superior to the tendon's insertion into the calcaneal tuberosity. A Beaver No. 64 mini-blade is introduced through the skin and tendon in a perpendicular manner, bisecting the tendon. The tendon is then lifted away from the leg, and the blade turned medially. Hemisection of the tendon is accomplished by gently working the blade against the tendon until its fibres are completely cut. When satisfactorily performed a gap can be palpated in the tendon. Avoid forcefully pushing the blade against the tendon as this may result in tenotomy, with rapid loss of resistance, followed by uncontrolled movement of the blade and subsequent laceration of the skin or the surgeon's finger. This procedure is repeated in the opposite direction, 2.5–4.0 cm more proximally, and then again 2.5–4.0 cm more proximal to the second cut. The foot is then firmly dorsiflexed to an angle greater than 90°, generally 5° above neutral (Fig. 8.18b). Overcorrection should be avoided, as this may lead to rupture of the tendon or a calcaneus deformity. The stab wounds are generally so small that they do not require sutures. However, if desired, a single interrupted 5–0 nylon suture can be used.

Dressings and postoperative care

Dressings consist of non-adherent fine mesh gauze (petrolatum, 3% Xeroform™ or Adaptic™), and dry sterile gauze dressing. A well-padded plaster splint is applied to immobilize the foot and ankle, and to maintain the ankle in approximately 5° of dorsiflexion. At the first dressing change, the patient is placed in either a short-leg walking cast or a walking brace for 6 weeks. The decision to cast or brace should be determined on an individual basis, based upon the surgeon's assessment of patient compliance.

Advantages

- Simple to perform
- Minimally invasive procedure
- Effective for relieving elevated forefoot plantar pressures.

Disadvantages

- Over lengthening of the tendon can result in a calcaneus gait.

Complications

- Tendon rupture
- Infection
- Development of a plantar heel ulcer
- In cases with moderate to severe shortening of the Achilles tendon, open lengthening of the tendon may be necessary.

Partial calcanectomy

Partial calcanectomy is indicated for the surgical management of large non-healing wounds located over the heel,

with or without osteomyelitis. These wounds are typically chronic decubitus ulcers located on the posterior aspect of the heel, or neuropathic ulcers on the plantar surface of the heel. Regardless of the aetiology, heel ulcers are often unresponsive to conservative therapy and are frustrating to treat. Partial calcanectomy is a viable alternative to below-knee amputation for these patients, provided that they have adequate distal perfusion. The procedure eradicates infection and achieves wound closure and limb preservation. Patients who are ambulatory before surgery are generally able to resume the same level of function postoperatively. Smith and coworkers proposed the following preoperative criteria for performing a partial calcanectomy:

- Ankle–brachial index > 0.45
- Transcutaneous oxygen tension ($TcPo_2$) > 28 mmHg
- Serum albumin > 3.0 g/dL
- Total lymphocyte count of more than 1500.

Advantages

- Simple procedure
- Low morbidity
- Rapid convalescence
- Good functional results.

Disadvantages

- None. This is a salvage procedure.

Complications

- Infection
- Delayed healing
- Failure to heal.

CASE STUDY

Partial calcanectomy

A 59-year-old Caucasian male with type 2 diabetes of 7 years' duration and history of multiple foot surgeries for the treatment of infected neuropathic plantar ulcers underwent a successful transmetatarsal amputation of his right foot, 2 years earlier. He had recently been treated for a full-thickness ulcer beneath his 4th metatarsal head. This lesion was unresponsive to conservative care, and an Achilles tendon lengthening procedure was performed. The forefoot ulcer healed quickly following the tendon lengthening: however, the patient went on to rupture his tendon and developed a calcaneus gait. The result of elevated pressure beneath his heel was the development of a chronic full-thickness ulcer, approximately 3.0 × 2.0 cm (Fig. 8.19). Although the patient had adequate distal perfusion, the ulcer failed to heal with conservative therapy;

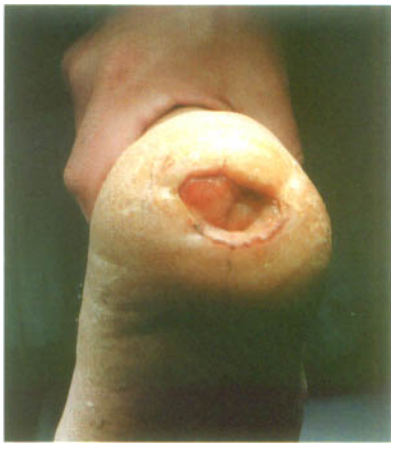


Fig. 8.19 Full-thickness neuropathic ulcer on the plantar aspect of the right heel, with osteomyelitis of the calcaneus.

total-contact casting, local wound care and antibiotics. Osteomyelitis was suspected. Partial calcanectomy, a salvage procedure, was offered to the patient as an alternative to below-knee amputation and he consented to the operation.

Technique

The procedure is performed under spinal anaesthesia using a thigh tourniquet for haemostasis. The patient is placed in a prone position on the operating theatre table. Two converging semi-elliptical incisions are made surrounding the ulcer. The incisions are carried deep to bone, and the ulcer is completely excised. The incision is extended proximally, to expose the posterior aspect of the calcaneus, and deepened to the fascia overlying the Achilles tendon (Fig. 8.20). A No. 15 blade is used to transect the tendon at its insertion on the posterior tubercle of the heel. The tendon is dissected free, grasped with an Allis clamp and reflected out of the wound. Dissection is then directed close to bone, exposing the body of the calcaneus. In the case cited above, the posterior aspect of the heel was resected, using a sagittal saw, in a plane entering the posterior superior aspect of the calcaneus and exiting plantarly at the insertion of the plantar fascia. Inspection of the residual calcaneus revealed healthy cancellous bone. The wound was thoroughly irrigated using 2 L of normal sterile saline solution containing an antibiotic. Three drill holes were then made in the posterosuperior aspect of the calcaneus, for reattachment of the Achilles tendon, using 3–0 Ethibond non-absorbable sutures. A tube low suction (TLS) drain was inserted, exiting through the lateral aspect of the heel. The deep tissues were closed with 2–0 absorbable sutures. The skin was closed using a

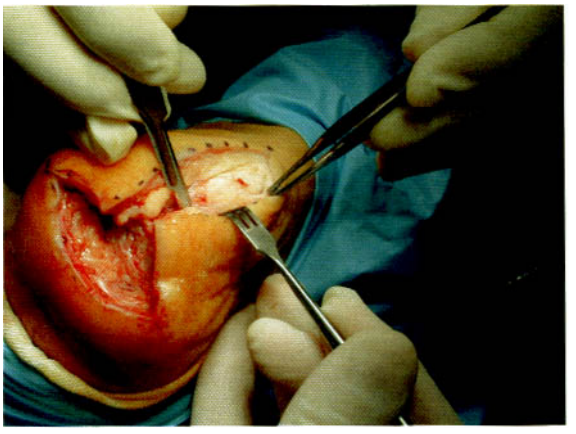


Fig. 8.20 The heel ulcer has been excised and the incision extends proximally over the Achilles tendon.

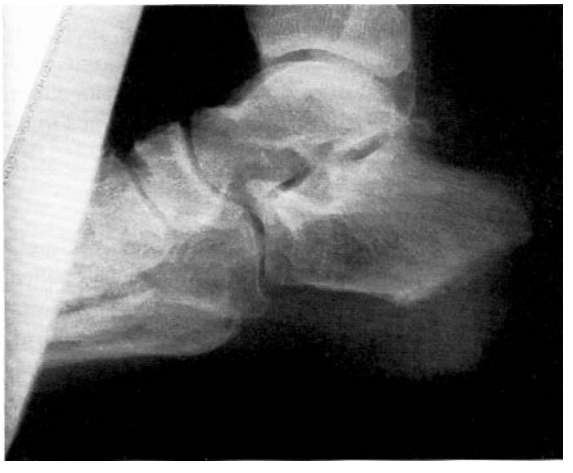


Fig. 8.21 Partial calcanectomy of the right foot. Postoperative lateral radiograph of the same patient shown in Figs 8.19 and 8.20.

combination of 3–0 nylon vertical and horizontal mattress sutures, and simple interrupted sutures. (*Caution*—infected wounds should be packed open and allowed to heal by secondary intention, or brought back to the operating theatre for delayed wound closure.) Figure 8.21 is the postoperative lateral radiograph, which reveals the amount of bone that was removed.

Dressings and postoperative care

Dressings consist of non-adherent fine mesh gauze (petrolatum, 3% Xeroform™ or Adaptic™), placed on the suture line, a bulky gauze fluff dressing and additional padding for protection of the heel and lateral border of the foot. Dressings are held in place by gauze bandage. A well-padded plaster splint is applied to immobilize the foot and ankle. Drains are generally removed after 48 h. Moderate bleeding from the cut cancellous bone is to be expected, especially over the first 12–24 h, and dressings may need to be reinforced with additional absorbent material. After 7 days, a well-padded short-leg non-weightbearing cast is applied. The cast is changed at 2-week intervals for inspection of the wounds. Sutures are left in place for 3–4 weeks. Once the skin is healed, the patient is placed in a walking brace for 4 weeks and is then allowed to ambulate in a therapeutic shoe with an ankle–foot orthosis (AFO).

AMPUTATIONS OF THE FOOT

Amputations of the foot can be divided into non-emergency and emergency procedures. Non-emergency amputations allow some flexibility in the creation of skin flaps, selection of level and wound closure. These cases generally include neuropathic feet that are structurally or functionally impaired, with satisfactory circulation and controlled infection. They are characterized by moderate to severe forefoot deformities with associated chronic non-healing ulcers that are recalcitrant to conservative medical and surgical management. In some cases, the presenting deformities are the residua of prior infection, tissue necrosis and chronic non-healing wounds.

Emergency amputations include those performed for gangrene, severe soft tissue infection, osteomyelitis, peripheral vascular disease, tumours or trauma. The main consideration in determining the level of amputation in these cases is the extent of healthy tissue. When infection is the primary issue, an open or guillotine amputation may be necessary. In most cases, adequacy of blood supply to the foot ultimately determines the level at which successful amputation can be performed. Although non-invasive laboratory methods have been proposed for evaluating wound healing potential, clinical experience and judgement are most often relied upon.

Preoperative physical examination should include a quantitative assessment of ankle joint dorsiflexion. Contracture of the Achilles tendon is generally more apparent prior to amputation of the forefoot, and suggests the need for tendon lengthening at the time of amputation. The procedure is performed as necessary, in the presence of equinus or excessive spasticity. In many cases equinovarus deformity is a complication of Lisfranc and Chopart amputations. A longitudinal open procedure or percutaneous approach can be utilized according to the surgeon's preference.

Wound healing criteria for amputation surgery

Measurements of $TcPo_2$ are useful to predict accurately the presence of critical vascular disease and the success of major or minor amputations. $TcPo_2$ levels ≥ 30 mmHg, bode well for healing of a forefoot amputation, and are more accurate predictors than a palpable pedal pulse. $TcPo_2$ levels < 30 mmHg indicate significant vascular disease and foreshadow wound healing failure and amputation. These patients require well-timed vascular surgery consultation, arteriography and revascularization.

Toe pressures, measured by photoplethysmography, are helpful to predict the healing potential of primary forefoot amputations. In a retrospective study of 136 amputations, Vitti and coworkers observed that healing occurred in all diabetics with preoperative toe pressures > 68 mmHg. Other preoperative criteria for wound healing include: an ankle–brachial index > 0.50, serum albumin > 3.0 g/dL, serum protein > 6.0 g/dL and total lymphocyte count > 1500. Patients should be medically stable, with diabetes and infection under control.

It is important to note that the preoperative wound healing criteria listed above are only guidelines. The most important factors are individualized patient assessment and clinical experience.

Selection of anaesthesia

Amputations at the level of the forefoot or mid-foot can be performed safely under regional ankle block anaesthesia, or if desired under spinal or epidural anaesthesia. In a series of consecutive transmetatarsal or mid-foot amputations performed by the author, an ankle block was administered in 83% of the cases. Patients receiving regional ankle block anaesthesia with intravenous sedation tolerate surgery well, with minimal anaesthesia risk.

Digital amputations

Digital amputation is indicated in the presence of fixed digital deformity, osteomyelitis, septic arthritis or recurrent ulcers over the interphalangeal joint or distal aspect of the toe. Neuroischaemic patients may present with severe necrotic ulceration of the distal third of the toe. In yet another scenario, patients undergoing invasive cardiovascular procedures may develop a shower of cholesterol emboli to their toes, with resultant gangrenous changes involving the tips of their toes.

A common clinical scenario is the neuropathic patient, with a hammer toe deformity, who presents with a superficial ulcer over the proximal interphalangeal joint of the 2nd or 3rd toe. This may have started as a 'simple' corn. Local wound care is initiated: however, the ulcer is refractory to treatment. The toe becomes infected, very swollen (sausage-like), and shortly thereafter, the head of the proximal phalanx is visible in the wound. There is little recourse but to amputate the entire toe.

In some cases, when the lesion is limited to the distal portion of the toe, a terminal amputation can be performed at the distal joint. Amputation of a single lesser toe generally causes very little disability. However, amputation of the great toe with its metatarsal head alters weightbearing and increases the vulnerability of the remaining toes.

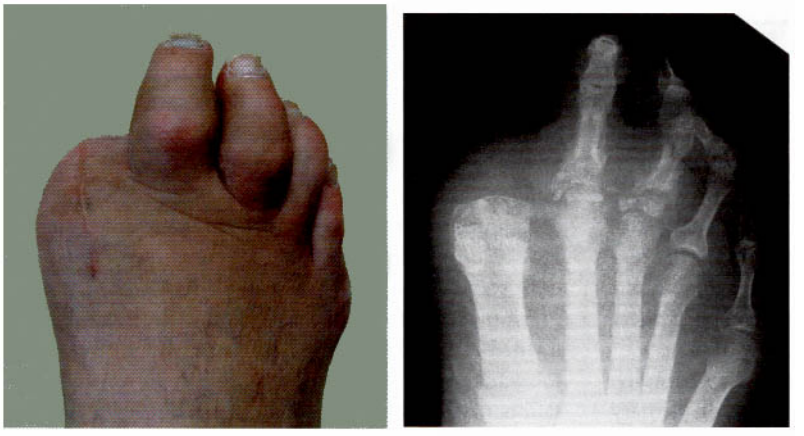
When performing digital amputations the surgeon can be creative with skin closure. Medial and lateral skin flaps are most often employed because they protect the neurovascular structures on either side of the toe. However, dorsal and plantar flaps are also acceptable. The length of the flaps can also vary, with one flap being longer than the other. This is an important consideration for wound closure, when there has been skin loss due to infection or necrosis.

Partial digital amputation of the hallux

Distal amputation of the hallux, sometimes referred to as a terminal Symes amputation, is indicated for lesions of the distal toe or nail bed, e.g. osteomyelitis of the distal phalangeal tuft, ulceration of the nail bed or tumour. The procedure employs either resection of the tuft of the distal phalanx or disarticulation of the toe at the interphalangeal joint. This procedure preserves acceptable length and function of the hallux. A similar surgical approach can be modified for the lesser toes.

Technique

The procedure for amputation through the hallux interphalangeal joint is performed in the operating theatre under local anaesthesia and sedation, with a Penrose drain applied as a tourniquet around the base of the great toe. A long plantar and short dorsal skin flap is fashioned. The transverse dorsal skin incision is made, proximal to the posterior nail fold, at the level of the interphalangeal joint. The incision extends from medial to lateral and is then directed distally around the end of the toe, to form a long plantar flap. The toe is disarticulated at the interphalangeal joint and all tissues are excised (nail plate, nail bed,



(a) (b)

Fig. 8.22 Hallux amputation in a neuropathic patient. (a) Clinical appearance 5 months following disarticulation of the hallux at the metatarsophalangeal joint, with medial and lateral skin flaps for closure. There is moderate to severe swelling of the 2nd and 3rd toes. (b) Anteroposterior radiograph reveals pathological fractures of the proximal phalanges of the 2nd and 3rd toes.

nail matrix and distal phalanx). A long plantar flap is fashioned (trimmed to fit), and sutured without tension to the short dorsal flap with 4–0 nylon simple interrupted sutures. If the toe is infected at the time of surgery, the wound should be left open or very loosely approximated. The patient can then be brought back to the operating theatre for delayed wound closure when the infection is resolved.

Dressings and postoperative care

Dressings consist of non-adherent fine mesh gauze (petrolatum, 3% Xeroform™ or Adaptic™), and a fluffy dry sterile compression gauze bandage. A surgical shoe is dispensed. The patient is instructed to rest at home, remain non-weightbearing and to elevate his feet for 48 h. He is then allowed partial weightbearing in a surgical shoe with crutches or a walker. The first postoperative dressing change is scheduled within 1 week. Dressings are changed weekly for 3–4 weeks postoperatively. Sutures are removed in 14–21 days, and the patient is allowed to return to his regular footwear as soon as the operative site is completely healed and swelling has subsided.

Amputation of the lesser toes can be performed by a transphalangeal approach or by disarticulating the toe at the metatarsophalangeal joint. Some authors have suggested leaving a 'button' of proximal phalanx intact over the metatarsal head. However, there is a need for caution. The residual portion of the proximal phalanx can cause discomfort and become a future site of ulceration. I have found that transphalangeal amputations often result in dorsiflexion of the residual phalangeal stump, with irritation of the skin over the prominent bone. For this reason I disarticulate the toe at the metatarsophalangeal joint.

Hallux amputation (Fig. 8.22a,b)

Amputation of the great toe invariably results in biomechanical dysfunction of the foot. The degree to which this occurs depends upon whether or not a portion of the 1st metatarsal has also been removed. The loss of propulsive function is not detrimental to neuropathic patients who already have an apropulsive gait. Of greater concern, however, are the following postoperative sequelae:

- Compensatory flexion contracture of the 2nd toe
- Ulceration at the tip of the 2nd toe
- Lesser metatarsalgia
- Pathologic fractures
- Ulceration beneath the 1st or 2nd metatarsal heads (Fig. 8.23).

These complications may eventually result in a more proximal amputation.

Technique

Caution—amputation of the hallux is deceptively simple. Surgeons often find that, once the hallux is amputated, they are unable to close the surgical wound without removing the metatarsal head and a portion of the shaft. This will always occur when using a racquet incision encircling the base of the great toe. The dilemma is how to prevent this from happening. The approach, when feasible, should be to develop a long medial or lateral skin flap that will allow for closure over the disarticulated 1st metatarsal head.

Ray amputations

A ray resection consists of excision of a toe and its corres-

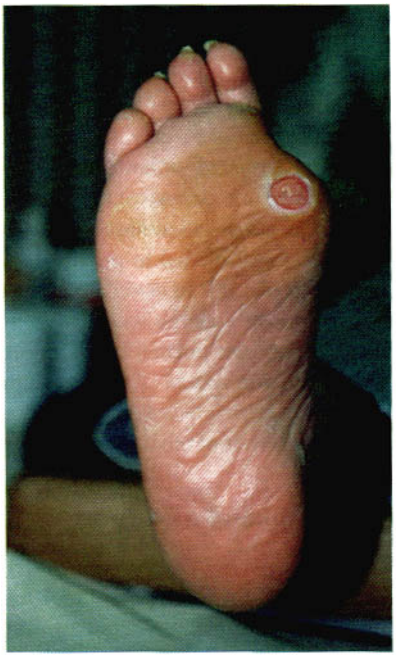


Fig. 8.23 A full-thickness neuropathic plantar ulcer developed beneath the 1st metatarsal, as a complication of amputation of the hallux with resection of the 1st metatarsal head.

ponding metatarsal. The most frequent complication of a ray resection is transfer ulceration. The highest success appears to be with resection of a central ray (2nd or 3rd), or 5th ray. Amputation of the hallux and 1st metatarsal frequently results in imbalance of the medial column of the foot with a poor functional outcome. Therefore, it is very important to preserve 1st metatarsal shaft length whenever possible.

Amputation of the 5th ray alone is indicated when infection and necrosis involve the 5th toe and/or the skin over the metatarsophalangeal joint. This can develop in neuroischaemic patients from unremitting pressure, caused by a tight shoe or bandage, over the lateral aspect of the 5th metatarsal head. In neuropathic individuals, repetitive moderate stress on the skin beneath a prominent 5th metatarsal head will eventually result in callus formation, ulceration and infection. The primary objective of this procedure is to achieve adequate resection of the infected or necrotic tissues, in order to create a wound that can be closed without tension. The configuration of the skin incision is determined by the extent of the infected necrotic tissues to be excised. Whenever possible, the 5th metatarsal base should be preserved together with its muscle attachments for the peroneus brevis and tertius.

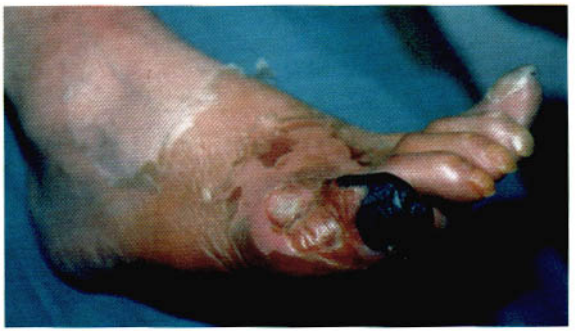


Fig. 8.24 Same patient as in Fig. 8.2. Resolving infection with dry gangrene demarcated at the base of the right 5th toe.

This is important for the prevention of varus deformity of the foot. Varus deformity occurs when inversion of the foot is left unopposed. Following ray resection, part or all of the incision may be left open, with the patient returning to the operating theatre for delayed primary closure. Ray resections are sometimes performed as an initial incision and drainage procedure to control infection prior to a more definitive amputation.

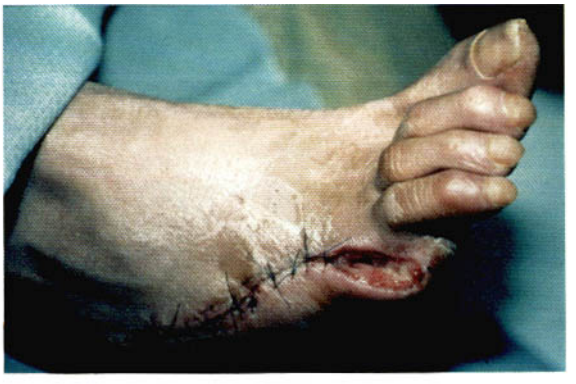
CASE STUDY

Ray resection to control infection with delayed primary closure

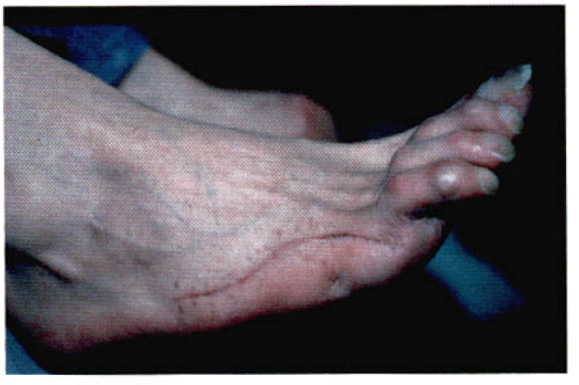
A 53-year-old man with poorly controlled type 2 diabetes of 9 years' duration was admitted to hospital with the provisional diagnosis of cellulitis of the right foot and infected abscess of the 5th metatarsophalangeal joint. Initial surgical treatment was incision and drainage of the abscess. Dry gangrene demarcated at the base of his 5th toe (Fig. 8.24). Although the patient initially refused further surgical treatment, he eventually changed his mind and consented to surgery. He was taken to the operating theatre where, under regional ankle block anaesthesia and sedation, his right 5th toe was amputated together with the distal two-thirds of the 5th metatarsal. The proximal portion of the wound was closed with 3–0 nylon simple interrupted sutures, and the distal portion was packed open. One week later, the patient returned to the operating room for delayed primary closure of the wound (Fig. 8.25a–c).

Technique

The 5th ray is amputated through a dorsolateral approach, with a racquet incision encircling the 5th toe. The toe is disarticulated at the metatarsophalangeal joint, and all necrotic tissues are excised. The incision is then extended



(a)



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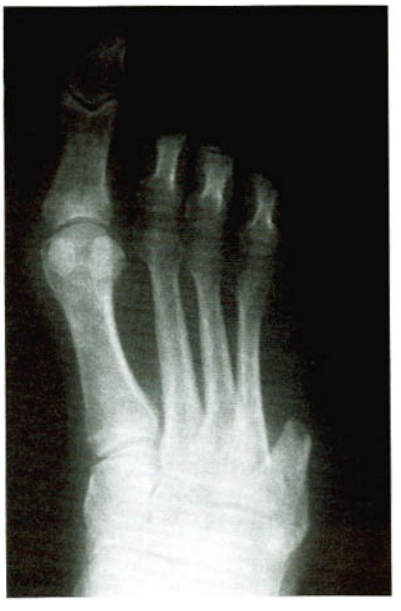

(c)

Fig. 8.25 Fifth ray amputation. (a) Partial amputation of the right 5th ray. The distal portion of the wound was left open. (b) Appearance of the right foot healed after delayed wound closure. (c) Anteroposterior radiograph reveals resection of the distal two-thirds of the 5th metatarsal.

proximally, in a curvilinear fashion over the 5th metatarsal shaft, to the level of the base. Dissection is kept close to the bone. The soft tissues are retracted using blunt Senn retractors. The exact amount of bone to be removed is determined, at the time of surgery, by how well the skin edges can be approximated without tension. Removing more bone may facilitate closure of the wound. The 5th metatarsal shaft is cut in an oblique manner, using a sagittal saw, from distal-medial to proximal-lateral. The reason for this angled cut is to avoid a bony prominence that could cause pressure on the skin. The wound is then thoroughly irrigated with normal sterile saline solution using a pulsed irrigation system. The decision to close the wound, or to pack it open is based upon the appearance of the wound and the results of reliable wound cultures.

Transmetatarsal and mid-foot amputations

Amputations through the forefoot and mid-foot include the transmetatarsal, Lisfranc and Chopart amputations. The Lisfranc amputation is performed at the tarsometatarsal joints and the Chopart amputation is performed at the mid-tarsal joints (Fig. 8.26). Mid-foot amputations frequently develop equinovarus deformity, which requires Achilles tendon lengthening or tenotomy. When preoperative criteria are met, healing occurs in > 80% of transmetatarsal and mid-foot amputations.

Transmetatarsal amputation

Indications

The indications and technique for performing a transmetatarsal amputation have changed very little since McKittrick and Warren initially described them in the 1940s and 1950s. They proposed three basic criteria, and I have added a fourth:

- Gangrene of one or more toes, without entering on to the foot
- Stabilized infection or open wound involving the distal portion of the foot
- An infected lesion in a neuropathic foot
- Moderate to severe forefoot deformity.

Careful preoperative preparation is necessary with drainage of infection, culture-directed antibiotics and daily wound care. Successful amputation requires attention to detail, careful planning of skin flaps, atraumatic operative technique and, whenever possible, primary closure of the wound. The use of an ankle or thigh tourniquet is desirable: however, this is at the discretion of the surgeon. Relative contraindications to the use of a tourniquet include ischaemia or recent lower extremity revascular-

Fig. 8.26 Transmetatarsal and mid-foot amputations. Three levels of amputation. From Sanders (1997) with permission from Elsevier Science.

ization. A bloodless field enables the surgeon to work more efficiently and saves operating time. However, prior to closing the wound, the tourniquet must be released and all bleeders ligated or coagulated. Meticulous haemostasis is required to prevent blood loss and haematoma formation. If ankle equinus is noted, it should be corrected at the same time as the transmetatarsal amputation.

Technique

The patient is placed in a supine position with the foot and lower half of the leg prepped and draped in the usual manner. Bony landmarks are identified for the 1st and 5th metatarsal heads and bases. The desired level of bone resection is determined, e.g. mid-shaft level, and then using a skin marker a line is drawn across the dorsum of the foot from mid-shaft of the 1st metatarsal to mid-shaft of the 5th metatarsal. Lines are then extended distally, along the 1st and 5th metatarsal shafts, to the bases of the hallux and 5th toe, and then curved across the plantar skin just proximal to the sulcus of the toes. This approach will create a short dorsal and long plantar flap.

Starting anteromedially at the 1st metatarsal shaft, the knife is held perpendicular to the skin and an incision is made through the skin, across the dorsum of the foot, ending at the previously determined level on the 5th metatarsal shaft. The dorsal incision is deepened to expose the long extensor tendons. Vessels are identified, ligated or electrocoagulated. The incision is then carried down to bone. Prior to transection of the metatarsals, an osteotome or key elevator is directed distally away from the dorsal skin incision, to reflect the soft tissues and periosteum. The dorsal flap should not be undermined.

Incisions are then carried distally toward the toes and then across the plantar aspect of the foot, developing a long thick myocutaneous flap. The plantar flap is retracted using rake retractors. It is important to keep the dissection close to the metatarsal shafts, thereby creating a thick viable plantar flap. The plantar flap is reflected proximally to the intended level of bone resection. The metatarsal bones are then cut transversely, with a power saw, at the level of the dorsal skin flap. The cuts are angled slightly from dorsal-distal to plantar-proximal. The 1st and 5th metatarsals are bevelled medially and laterally to prevent focal points of pressure. The distal foot is grasped securely with a small bone clamp, and then removed by sharp dissection.

The plantar flap is inspected, and debrided as necessary. Exposed flexor tendons should be grasped without tension and excised. Antibiotic solution is used to irrigate the wound copiously. The plantar flap is then brought up over the resected metatarsals and approximated with the dorsal flap. If the plantar flap is too long, redundant skin should be remodelled. The flap should be carefully marked with a skin marker and excess skin removed. Accurate trimming of the skin is accomplished by placing several Allis tissue forceps on the edge of the skin to be excised. The surgical assistant holds the forceps with gentle tension, and a fresh blade is used to trim the excess skin. Placing a malleable retractor beneath the plantar flap, while it rests on the dorsum of the foot, provides a firm supporting surface to cut on.

The tourniquet is deflated prior to wound closure, and bleeders are ligated or coagulated. Some oozing of blood from the transected bone marrow and from muscle is to be expected. The skin flaps are approximated without tension and secured with a few simple interrupted, subcutaneous 3–0 absorbable sutures. A TLS drain is placed in the wound, exiting the skin on the dorsolateral aspect of the foot. Skin flaps are carefully positioned and secured with 4–0 nylon sutures in a simple interrupted fashion, or with stainless steel staples. The technique for performing a transmetatarsal amputation is illustrated in Fig. 8.27a–f.

Dressings and postoperative care

Dressings consist of non-adherent fine mesh gauze (petrolatum, 3% Xeroform™ or Adaptic™), placed on the suture line, wide gauze sponges (4 × 8s, with a long dorsal and plantar tail, secured around the stump) and padding for protection of the heel and lateral border of the foot. Dressings are held in place by 1.2-m gauze bandage. A well-padded plaster splint is applied to immobilize the foot and ankle. Drains are generally left in place for 48 h. Normal sterile saline moist-to-dry dressings are applied when there is persistent drainage from the wound or

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Fig. 8.27 Technique for performing a transmetatarsal amputation. (a) Incision with creation of a long plantar flap. (b) Plantar view. The plantar incision is made just proximal to the sulcus of the toes. The dotted line represents the dorsal skin incision and its relationship to the metatarsal shafts. (c) Removal of the forefoot, leaving a thick myocutaneous plantar flap. (d) Resection of the metatarsals. (e, f) The plantar flap has been remodelled, approximated with the dorsal skin flap and sutured in place. The lateral view depicts how the metatarsals have been cut, from dorsal-distal to plantar-proximal. The plantar view (arrows) illustrates how the cuts are bevelled on the 1st and 5th metatarsal stumps. From Sanders (1997) with permission from Elsevier Science.

when portions of the wound remain open. Sutures are removed after 14–21 days, and the patient is then placed in a short-leg non-weightbearing fibreglass cast for an additional 3–4 weeks. The patient is then encouraged to return gradually to full weightbearing, in therapeutic low quarter depth inlay footwear. Shoe modifications include a stump filler and stiffened outer sole.

Fig. 8.28 Dry gangrene of the hallux, 2nd and 3rd toes. From Sanders (1997) with permission from Elsevier Science.

CASE STUDY
Transmetatarsal amputation

A 56-year-old man with type 1 diabetes and schizophrenia, was referred to the diabetic foot clinic for dry gangrene of his hallux, 2nd and 3rd toes (Fig. 8.28). The patient was new to our clinic, having been transferred from another hospital. The patient's medical history was quite remarkable: he had recently been very ill, in ketoacidosis, with a blood sugar level > 1200 mg/dL (66.7 mmol/L). The gangrenous changes in his toes developed during this episode, and appear to be related to a shower of emboli to his toes. Pedal pulses were present and Doppler studies were otherwise normal. He underwent a successful transmetatarsal amputation with an unremarkable postoperative course (Fig. 8.29a). A custom-made orthosis with a stump filler was provided for the patient for use in extra-depth therapeutic shoes (Fig. 8.29b). The patient is very satisfied with the results of his amputation and ambulates normally without a limp. This transmetatarsal amputation has been durable with no further complications for more than 15 years.

CASE STUDY
Transmetatarsal amputation with long laterally based plantar flap

A 66-year-old man with type 2 diabetes of 30 years' dura-

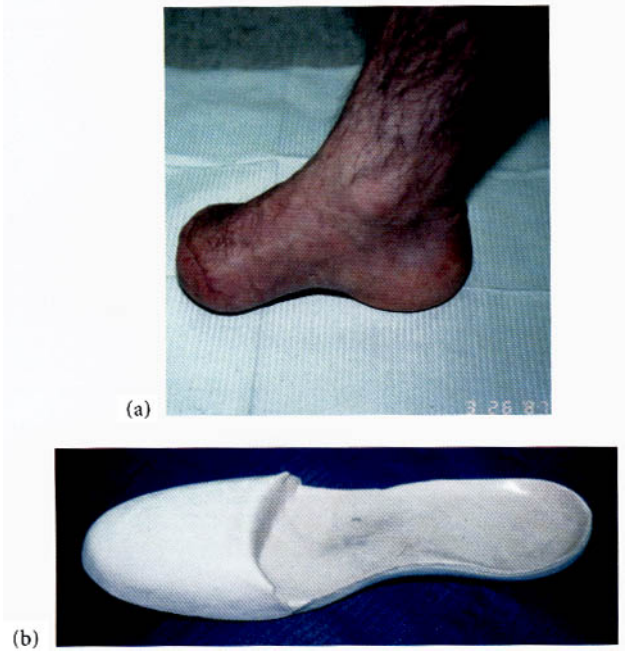


Fig. 8.29 Transmetatarsal amputation. (a) Postoperative appearance of the foot. (b) Custom-made orthosis, for use in extra-depth shoes.

tion and severe occlusive peripheral vascular disease had a chronic non-healing ulcer affecting his right great toe (Fig. 8.30a). The ulcer became infected and the patient developed dry gangrene of the hallux with cellulitis of the foot. The gangrenous right hallux was amputated, and 3 days later the patient underwent a right femoral to dorsalis pedis bypass graft. The amputation site remained dry and necrotic with no evidence of healthy granulation tissue. We believed that the wound would not heal, and 1 week later, the patient was brought back to the operating room for a definitive transmetatarsal amputation (Fig. 8.30b,c). The procedure was performed under spinal anaesthesia, without a tourniquet.

This was a challenging case that stretched the indications and limits for a transmetatarsal amputation. The success of a transmetatarsal amputation depends upon the presence of healthy plantar skin, for the creation of a plantar flap. In this case, gangrene and tissue loss extended on to the plantar skin, effectively narrowing the plantar flap. Preparing the flap in a normal manner would have left a large uncovered defect on the medial aspect of the amputation stump. To remedy this, we developed a long laterally based plantar flap, which was rotated medially to cover the wound. The operation was successful and the patient has a functional, durable foot that has remained lesion free for the past 3 years (Fig. 8.30d).

Transmetatarsal amputation with excision of plantar ulcer

Chronic non-healing neuropathic plantar ulceration is often associated with the complications of soft tissue infection and osteomyelitis. Cases which are refractory to conservative care may benefit from a modified transmetatarsal amputation with excision of a triangular wedge of skin from the plantar flap. I have also employed this technique, in the absence of a plantar ulcer, to remodel excessively broad plantar flaps, thereby avoiding redundant skin and unsightly dog-ears.

Technique

Following a standard transmetatarsal amputation procedure, the plantar flap is revised as illustrated in Fig. 8.31a–e. The ulcer is completely enclosed in a triangle with its apex located proximally. Several Allis tissue forceps are applied to the distal flap and the wedge of skin is excised. It should be emphasized that the Allis clamps are only applied to skin which is to be excised. A wide malleable retractor, placed beneath the flap, provides a firm supporting surface for the excision. The two segments of the plantar flap are then approximated with absorbable simple interrupted sutures placed within the wound, and 4–0 nylon in the skin. The dorsal and plantar flaps are closed, over a TLS drain, in the usual manner (Fig. 8.32a,b). Dressings, posterior splint and cast are applied as for a basic transmetatarsal amputation.

Open transmetatarsal amputation

Extensive forefoot infection or gangrene that extends on to the plantar skin may preclude a standard forefoot or mid-foot amputation. In these cases, an open or guillotine amputation performed at the mid-metatarsal level may be required. Guillotine amputations have a major disadvantage, in that they require extensive revision. A better alternative is to fashion flaps in the usual manner but to leave the wound open, with the intent to perform a delayed primary closure. The main disadvantage of open procedures is the prolonged length of time for healing, and the need for frequent dressing changes and debridement. Ideally, the wound will form a healthy granulation tissue base that can support a split-thickness skin graft or healing by secondary intention.

Amputations through the mid-foot

Lisfranc and Chopart amputations are frequently

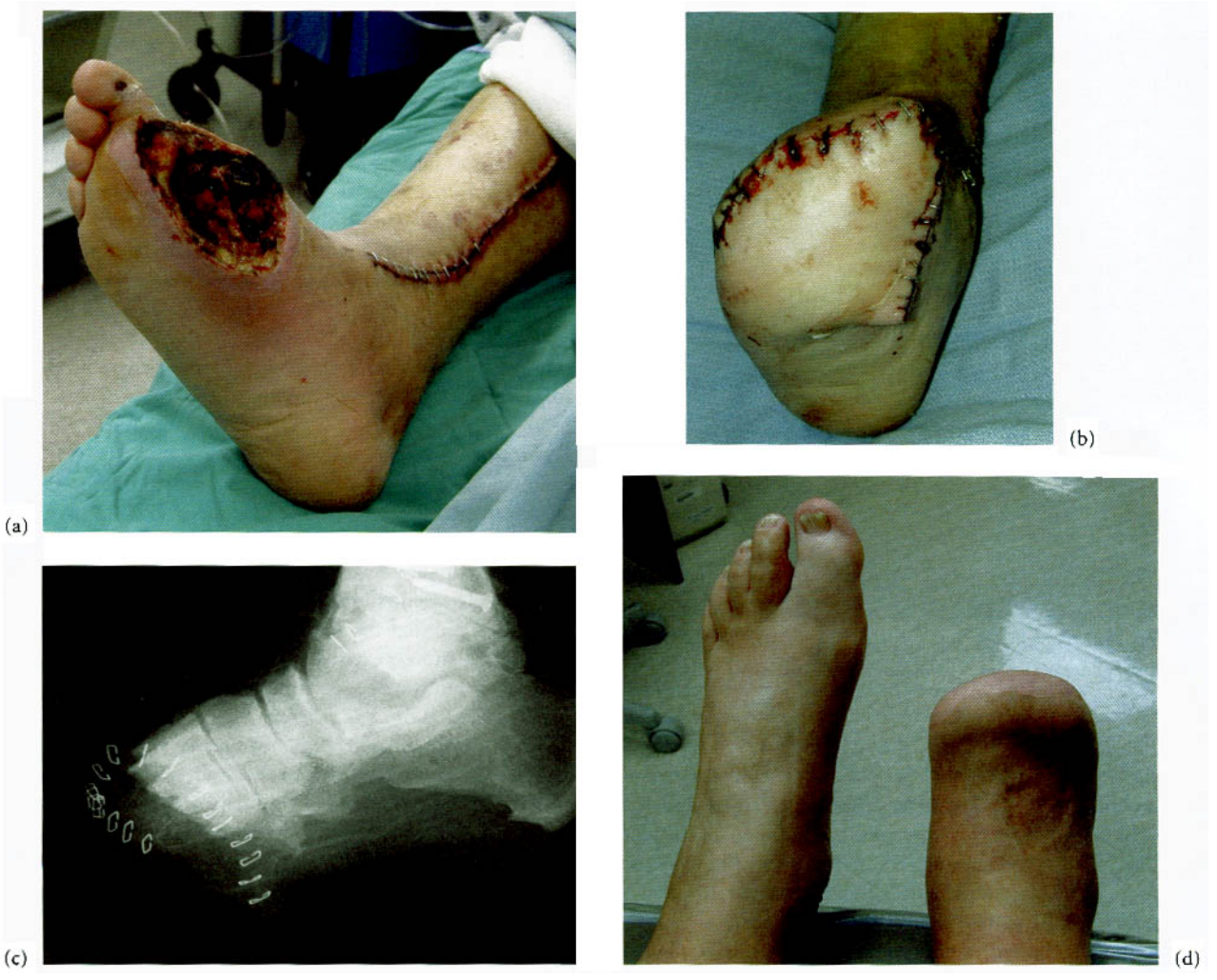


Fig. 8.30 Transmetatarsal amputation. (a) Preoperative appearance of the right foot with a large necrotic wound at the site of a failed hallux amputation. (b) The plantar flap has been rotated medially to achieve closure of the surgical wound. (c) Lateral radiograph reveals the level of amputation. Notice the angled cuts of the metatarsals. Stainless steel staples were used to close the wound. (d) Healed transmetatarsal amputation right foot.

complicated by the development of equinus deformity. Equinovarus deformity is associated with Lisfranc disarticulation. Amputation at the tarsometatarsal joints appears to be the most proximal level that allows for satisfactory function of the foot. For surgery to work at this level, care must be taken to preserve the base of the 5th metatarsal with its tendinous attachments, for eversion of the foot. The Achilles tendon should be lengthened, as necessary.

Chopart's mid-tarsal joint amputation has the advantage of producing less limb shortening than a Syme's procedure because the talus and calcaneus are retained. However, complications are commonly reported with the Chopart amputation. Severe equinus deformity develops due to loss of the tibialis anterior, long extensor and peroneal tendons, with resultant failure to balance the force of the triceps surae. The resulting foot is short with a very small weightbearing surface, and is at increased risk of further breakdown. Some authors advise reattachment of the tibialis anterior to the talus to prevent equinus deformity of the hindfoot. However, long-term results demonstrate inevitable development of equinus deformity, even with tenotomy of the Achilles tendon.

Fig. 8.31 Technique for modified transmetatarsal amputation with excision of a plantar ulcer. (a) A triangular wedge is drawn on the skin, enclosing the plantar ulcer. (b) The forefoot has been amputated and the triangular wedge of skin has been excised. (c) Plantar flap prior to approximation of the two segments of skin. (d) Plantar flap has been repaired with simple interrupted sutures. (e) Completed repair with approximation of the dorsal and plantar skin flaps. From Sanders (1997) with permission from Elsevier Science.

Fig. 8.32 Transmetatarsal amputation with excision of plantar ulcer. (a) Intraoperative view of the right foot, with the skin marked for excision of the plantar ulcer and creation of the plantar flap. (b) Completed transmetatarsal amputation with repair of the plantar flap. From Sanders (1997) with permission from Elsevier Science.

Modified Lisfranc amputation

Modifications of the Lisfranc amputation include preservation of the 5th metatarsal base, and the 2nd metatarsal base, in its intercuneiform mortise.

Technique

The patient is placed in a supine position with the foot and lower half of the leg prepared and draped in the usual manner. This procedure is performed in a manner similar to the transmetatarsal amputation, with the development of a longer plantar flap and short dorsal flap. The dorsal skin incision is made just distal to the 1st metatarsocuneiform joint and carried across the dorsum of the foot, ending just distal to the 5th metatarsal base. Occasionally it may be necessary to develop a longer dorsal flap to compensate for devitalized plantar skin. The medial and lateral incisions are carried distally along the metatarsal shafts to the necks of the metatarsals and then curved plantarly across the ball of the foot. The plantar flap is developed to the intended level of disarticulation.

The 1st metatarsal base is disarticulated from the medial cuneiform. Using a power saw, and working from medial to lateral, the 2nd metatarsal is transected at the level of the 1st and 3rd cuneiforms, leaving its base intact in the intercuneiform mortise. The 3rd and 4th metatarsals are then disarticulated, followed by transection of the 5th metatarsal, just distal to its base. Although the 5th metatarsal base will leave a prominence of bone, this generally does not cause a problem. Wound closure is performed in the same manner as described above for a transmetatarsal amputation. Dressings and postoperative care are also the same. High-top shoes or chukka boots with a stump filler and mild rocker sole are well suited for this level of mid-foot amputation.

CASE STUDY
Lisfranc amputation

A 50-year-old man with a history of IV drug abuse and type 2 diabetes underwent amputation of his right 2nd toe and was referred to us for surgical management of his infected right foot. Examination revealed several draining ulcers and sinus tracts, extending from the site of his amputated 2nd toe, to beneath the 2nd and 3rd metatarsal heads and into the central plantar space (Fig. 8.33a). Radiographs revealed osteolytic changes in the 2nd and 3rd metatarsals consistent with osteomyelitis. The patient was given the options of a partial foot amputation or below-knee amputation, and he chose to preserve his leg. He was taken to the operating theatre where under spinal anaesthesia and ankle tourniquet, he underwent a Lisfranc amputation of his right foot (Fig. 8.33b). The technical difficulty in this case was related to the poor condition of the plantar skin. We were unable to fashion a healthy long plantar flap. Closure was accomplished by creating a slightly longer dorsal flap. The surgical wound healed satisfactorily (Fig. 8.33c,d). The Achilles tendon was not lengthened in this case, and a mild equinovarus deformity developed. The right foot held up well, for approximately 9 years, until the patient developed a new ulcer and recurrent infections. He unfortunately went on to a below-knee amputation.

Chopart amputation

Technique

The incision for a Chopart amputation starts medially, at the level just proximal to the navicular tuberosity and extends over the dorsum of the foot to a point midway between the 5th metatarsal base and the lateral malleolus. Medial and lateral incisions are then carried distally, over the 1st and 5th metatarsal shafts. At mid-shaft, the incision is curved down across the sole to fashion a plantar flap. The plantar flap is developed in a careful manner, using rakes for retraction, to the level of the mid-tarsal joint. The ligaments around the talonavicular and calcaneocuboid joints are divided. A suture is placed in the end of the tendon of the tibialis anterior. Soft tissue attachments are sharply dissected free from the foot, and it is disarticulated from the rearfoot. A drill hole is made in the talus for attachment of the tibialis anterior. The plantar flap is trimmed to size. Tourniquet is released and bleeders ligated or electrocoagulated. The skin flaps are then approximated, over a drain. The Achilles tendon is tenotomized. Standard dressings and splints are applied as for a transmetatarsal amputation.

Early consultation with an orthotist/prosthetist is advised for fabrication of an appropriate AFO or prosthesis.

CASE STUDY
Chopart's amputation

A 54-year-old man with type 2 diabetes of 17 years' duration and Charcot's arthropathy had chronic ulceration beneath the calcaneocuboid joint of his left foot. He also had nephropathy, peripheral vascular disease, retinopathy, neuropathy, congestive heart failure, hypertension and cardiovascular disease. He presented at accident and emergency with fever, rigors and a grossly infected left foot. Radiographs and clinical examination confirmed gas in the soft tissues on the dorsum of his foot, and over the 1st metatarsal to the level of the medial cuneiform. On admission his glucose was 398 mg/dL (22.1 mmol/L), white blood cell count was 18 300/μL, with 96% granulocytes. The patient was diagnosed with gas gangrene, a limb-threatening infection, and was immediately taken to the operating room for a guillotine amputation of his forefoot, under general anaesthesia. The surgical wound was left open. Intraoperative wound cultures revealed anaerobic Gram-positive cocci, *Peptostreptococcus magnus* and *Peptostreptococcus asaccharolyticus*. Blood cultures also grew *Peptostreptococcus magnus*. In consultation with the infectious disease specialist, he was placed on intravenous piperacillin sodium/tazobactam sodium and clindamycin. Daily dressing changes were performed, until the wound was clean and free from infection. The patient was given the options of a Chopart's amputation, to salvage a portion of his foot, or a below-knee amputation. He was opposed to losing his leg and chose the first option.

The patient returned to the operating theatre where under spinal anaesthesia, and a thigh tourniquet, he

Fig. 8.33 Lisfranc amputation. (a) Initial presentation with a non-healing wound at the site of prior amputation of the 2nd toe, right foot. (b) Multiple draining plantar ulcers with sinus tracts. (c) Completed repair with a long dorsal flap and short plantar flap. (d) Healed Lisfranc amputation right foot, compared to transmetatarsal amputation of the left foot. Part (d) from Sanders (1997) with permission from Elsevier Science.

underwent a Chopart's amputation of his left foot. In an effort to prevent the development of ankle equinus, the tibialis anterior and extensor digitorum longus tendons were attached to the residual neck of the talus. In addition, the Achilles tendon was tenotomized. A small split-thickness skin graft was applied to the wound for coverage. The operative sites were bandaged and the foot was placed in a well-padded posterior splint. The patient was seen by an orthotist/prosthetist who provided a temporary clamshell total-contact cast.

In spite of our efforts to prevent the development of ankle equinus, this occurred, and was complicated by the

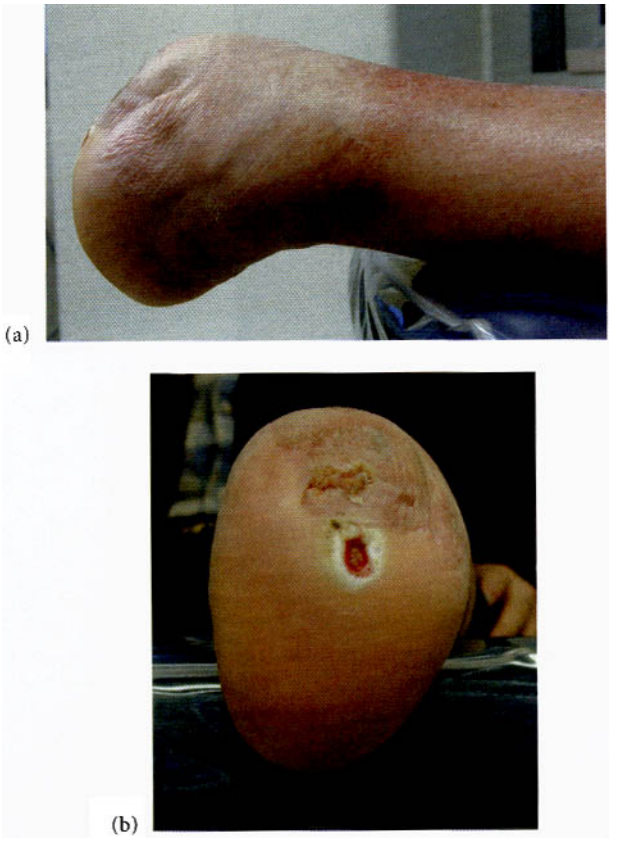


Fig. 8.34 (a) Chopart's amputation left foot. (b) Plantar ulcer.

development of ulceration. The patient ambulates well, and appears to be satisfied with the outcome of his surgery (Fig. 8.34a,b).

Outcomes of transmetatarsal and mid-foot amputations

Of the three levels of amputation discussed in this chapter, the transmetatarsal amputation is the most successful with respect to functional outcomes, patient satisfaction and long-term results. Transmetatarsal amputation preserves foot function, is cosmetically acceptable, does not require a prosthesis and enables fitting with commercially available footwear. Amputations performed at the tarsometatarsal and mid-tarsal joint levels frequently result in deformity and difficulty fitting shoes. Limb salvage can be achieved, with functional outcomes, by the motivated patient and knowledgeable surgeon with the use of these procedures.

SURGICAL MANAGEMENT OF THE CHARCOT FOOT

Most authors agree that non-operative treatment is the standard for the majority of patients with Charcot's osteoarthropathy of the foot and ankle. However, surgical intervention is indicated when deformity is severe, the foot/ankle are unstable or weightbearing is difficult. Careful patient selection and timing of operative treatment are critical for successful outcomes. Unstable joints and deformities which predispose to shearing stress and ulceration can be corrected, but only after the acute inflammatory phase (stage of development) has subsided. It appears that operative intervention during the acute phase of Charcot's joint disease contributes to further destruction and is destined to fail.

Indications and criteria

Instability, deformity, chronic ulceration and progressive joint destruction, despite rest and immobilization, are the primary indications for surgical intervention in diabetic individuals with Charcot's joint disease.

The patient's age, physical condition, compliance and comorbidities must also be considered in the surgical decision-making process. The benefits of surgery must be weighed carefully against the possible risks and complications. A simple ostectomy or limited arthrodesis may be all that is required. Contraindications to surgical management of the Charcot foot include infection, ischaemia, active bone disease, poorly controlled diabetes mellitus, a medically unstable patient and a history of poor compliance.

Criteria for surgical intervention

- Foot/ankle deformity
- Instability
- Ulceration
- Adequate circulation
- Medically stable
- Infection controlled.

Surgical procedures for the Charcot foot

Ostectomy

The most commonly employed procedure for the treatment of chronic neuropathic ulceration involves excision of bony prominences through either a plantar, medial or lateral approach. Decompression of the ulcer may be sufficient to prevent recurrence even when there is

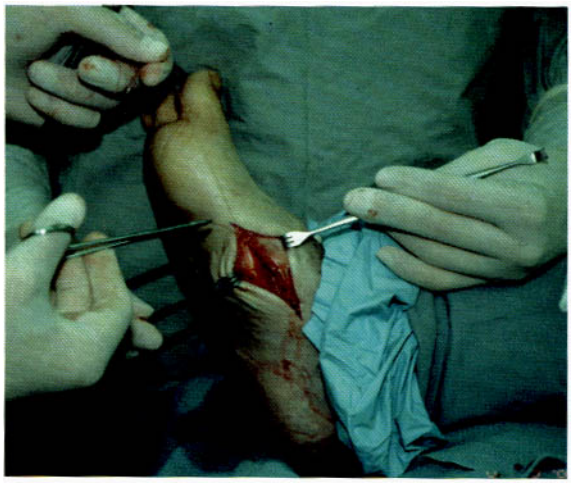
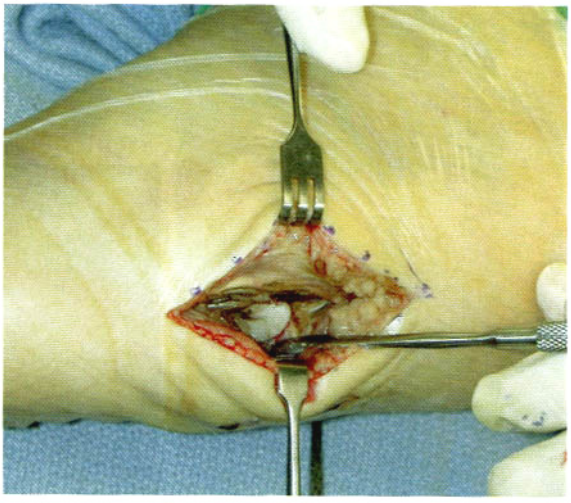


Fig. 8.35 Medial approach for ostectomy of the medial cuneiform.

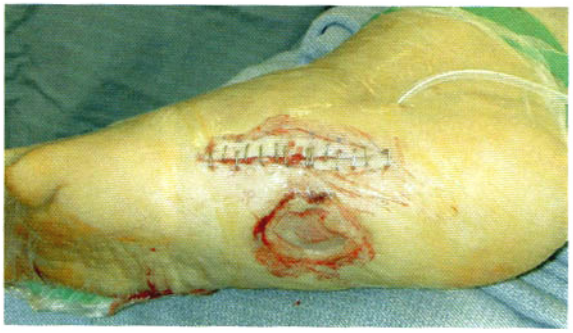
residual deformity of the foot. Excision of the ulcer, resection of underlying bony prominence, with primary or secondary closure of the wound, is a reasonable method of treatment associated with minimal morbidity.

Technique

The surgical approach to ostectomy can be direct or indirect, and is determined by the location of deformity and condition of the skin. Bone can be resected through a plantar approach: however, it is easier to do this through either a medial (Fig. 8.35) or lateral (Fig. 8.36a,b) approach. Excision of the ulcer with resection of bone provides a quick fix for chronic plantar ulceration: however, recurrence is common.



(a)



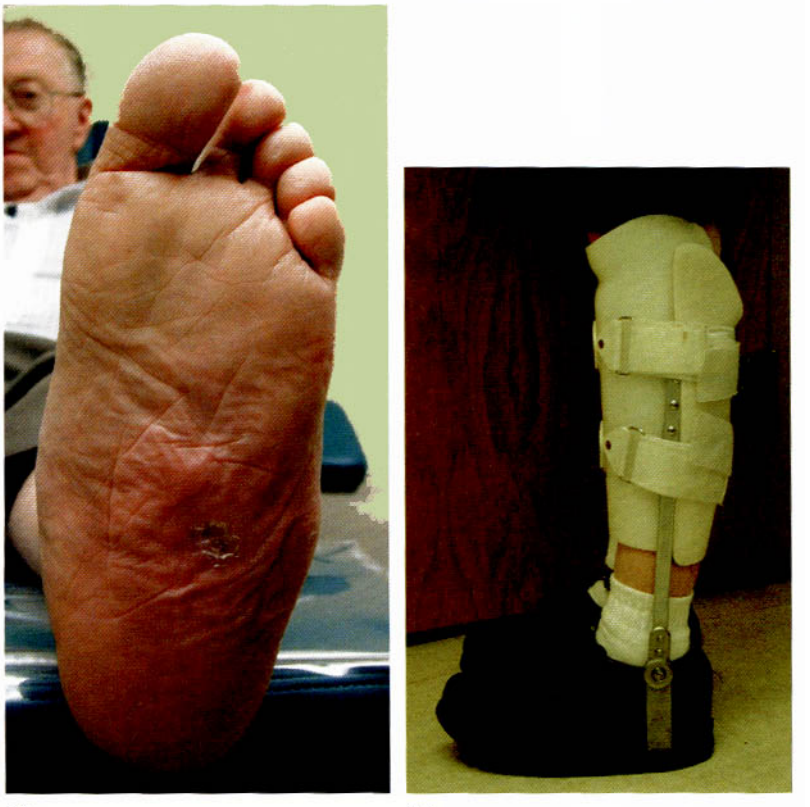
(b)

Fig. 8.36 Lateral approach for ostectomy of the cuboid. (a) Incision on the lateral border of the left foot. The articular surface of the cuboid is visible in the wound. (b) A large plantar ulcer is seen below the incision.

CASE STUDY

Percutaneous lengthening of Achilles tendon with ostectomy of cuboid

A 63-year-old man with type 2 diabetes of 12 years' duration presented to the diabetic foot clinic with a chief complaint of pain, redness and swelling of his left foot. This condition began 1 week earlier with sudden onset and with no history of injury. Physical examination revealed instability of the mid-foot, ankle equinus, bounding pedal pulses, absent deep tendon reflexes at the ankle and loss of protective sensation. He was unable to perceive the Semmes–Weinstein 6.10 (90 g) monofilament. Radiographs revealed fracture dislocation of the tarsometatarsal joints (Lisfranc's joint). The patient was afebrile with no elevation in his WBC count. His diabetes was very poorly controlled as evidenced by a markedly elevated HbA_{1c} of 12.2%. Initial treatment consisted of a well-padded compression dressing and elevation of the limb. The patient was then immobilized in a non-weightbearing cast, which was changed every 3 weeks for 2.5 months. During a cast change, at 12 weeks postimmobilization, an ulcer was noted at the apex of his collapsed mid-foot (rockerbottom deformity). Radiographs revealed that the ulcer was located directly beneath the cuboid, which was plantarly displaced. The surgical treatment for this patient included percutaneous lengthening of the Achilles tendon, with ostectomy of the cuboid performed through a lateral approach. Intraoperative and postoperative photographs illustrate the surgical approach (Fig. 8.36a,b). His postoperative course was uncomplicated and his plantar ulcer



(a) (b)

Fig. 8.37 The same patient as in Fig. 8.36(a,b). (a) The foot is well healed. (b) Patella tendon bearing brace with custom-moulded shoes and rocker soles.

healed well. He wears custom-moulded shoes and a patella-tendon bearing brace. Although there is some residual deformity, the patient remains lesion free (Fig. 8.37a,b).

Arthrodesis

Mid-foot and hindfoot arthrodesis of neuropathic joints should be considered salvage procedures, as they are technically demanding and frequently associated with complications. Recent reports have been encouraging with respect to satisfactory outcomes of these procedures as an alternative to amputation of the limb. However, there is still a need for caution, as surgical complication rates remain high. Stabilization of the medial column of the foot is crucial to the success of mid-foot arthrodesis.

Factors leading to successful arthrodesis include the preoperative condition of the foot, control of infection, operative technique and postoperative management. In general, no patient should be considered for surgery until the acute arthropathy has subsided. The precise timing for this has not been quantified. Regardless of which joints are fused, basic surgical techniques remain the same.

Technique for successful arthrodesis

- Thorough removal of all cartilage and detritus
- Careful removal of sclerotic bone down to healthy bleeding bone
- Meticulous fashioning of congruent bone surfaces for apposition
- Rigid fixation of bone.

Modern surgical techniques for internal and external fixation have greatly increased the chances for successful outcomes. Of equal importance is the necessity for prolonged postoperative immobilization, often two to three times longer than that required for a patient without neuropathic bone disease. Patients must remain non-weightbearing, and require physiotherapy and rehabilitation, with a gradual return to protected weightbearing.

The following case is a striking example of acute Charcot's arthropathy, with fracture dislocation of the mid-foot, that required realignment arthrodesis with fusion of the medial column of the foot.

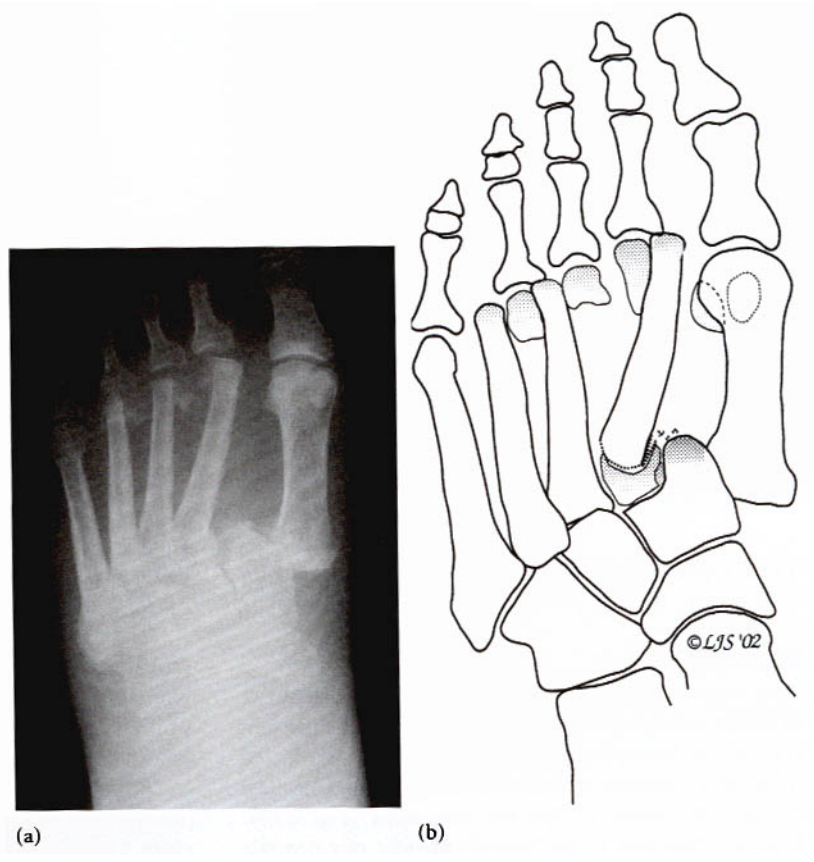


Fig. 8.38 Acute Charcot's arthropathy of the left foot. (a) Anteroposterior radiograph reveals fracture-dislocations of the tarsometatarsal joints (Lisfranc's joint), with medial dislocation of the 1st metatarsocuneiform joint, and dorsally displaced fractures of the 2nd, 3rd and 4th metatarsal heads. (b) Illustration of the radiographic findings.

CASE STUDY

Open reduction and rigid internal fixation of mid-foot fracture-dislocations

A 53-year-old Caucasian, male, janitorial worker, with type 2 diabetes of 6 years' duration presented to the diabetic foot clinic with the chief complaint of sudden and unexpected swelling of his left foot. There was no history of injury, ankle sprain, tripping or falling. Physical examination revealed moderate to severe redness, swelling and elevated skin temperature, approximately 4°C, of the left foot and ankle. Swelling extended up the leg to the knee. The medial column of the foot was unstable. Pedal pulses were present and there were no breaks in the skin. The patient had a dense peripheral neuropathy with loss of protective sensation, absent deep tendon reflexes at the ankle and elevated vibratory perception threshold > 45. The patient's body mass index was 35 kg/m^2. Laboratory data revealed an elevated HbA_{1c} 8.0%, mild anaemia, and normal WBC count. Radiographs revealed fracture-dislocation of Lisfranc's joint, with medial dislocation of the 1st metatarsal, and fracture dislocation of the 2nd metatarsocuneiform joint. In addition, there were dorsally displaced fractures of the 2nd, 3rd and 4th metatarsal heads (Fig. 8.38a,b).

He was initially placed in a well-padded Jones compression dressing and admitted to the medical centre for bed rest and elevation of his left lower extremity. Within 1 week, the swelling began to subside and he was placed in a short-leg non-weightbearing cast. The cast was changed 2 weeks later. Interim evaluation at day 21 revealed resolution of the swelling, redness and elevated skin temperature. The medial column of the foot (1st ray) remained unstable. At this time the process was considered to be subacute, and surgical intervention was advised. The following criteria for surgical intervention were met:

- Foot deformity/instability
- Adequate circulation
- Medically stable
- No evidence of infection.

The patient underwent successful open reduction and rigid internal fixation of his mid-foot fracture-dislocations,

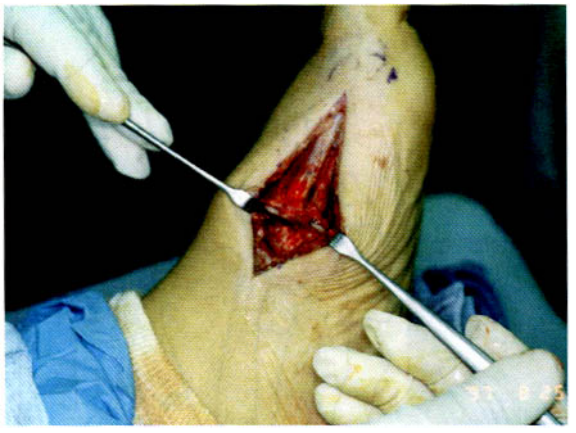


Fig. 8.39 Intraoperative view showing dislocation of the 1st metatarsocuneiform joint.

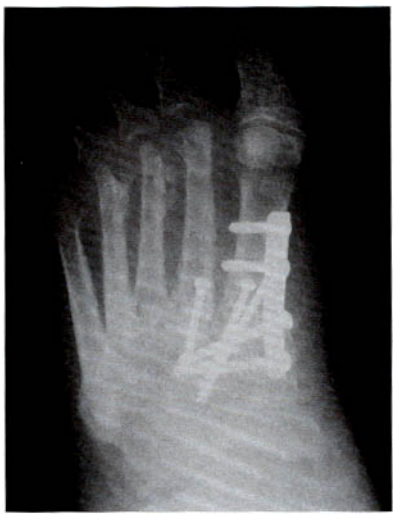


Fig. 8.40 Anteroposterior radiograph reveals satisfactory postoperative realignment of the tarsometatarsal joints.

under spinal anaesthesia, using a thigh tourniquet for haemostasis.

Technique

An 8-cm linear incision was made over the medial aspect of the 1st metatarsal and medial cuneiform. The incision was carried deep to bone and all soft tissues were reflected from the cuneiform and metatarsal, revealing complete medial dislocation of the 1st metatarsal (Fig. 8.39). Manual reduction of the dislocation was not possible because of soft tissue interposed in the joint. A sagittal power saw was used to resect a wafer of bone from the base of the 1st metatarsal, and to remove the articular cartilage from the medial cuneiform. The metatarsal was then easily relocated and fixed with two 4.0 mm cannulated screws, under C-arm fluoroscopy. A four-hole, 1/3 tubular plate was then placed across the 1st metatarsocuneiform articulation and secured with four 3.5 mm cortical screws. Attention was then directed to the fracture-dislocation of the 2nd metatarsocuneiform joint, where a 6-cm linear incision was made over the dorsum of the foot. The base of the metatarsal was resected and the cartilage removed from the intermediate cuneiform. The 2nd metatarsal was placed in proper alignment, secured with a K-wire, and fixed with a 4.0 cannulated screw. Wounds were irrigated thoroughly with normal sterile saline solution, and two closed suction drains were inserted. Deep closure of the soft tissues was obtained using 3–0 absorbable sutures, and the skin was closed with stainless steel staples. Postoperative radiographs revealed satisfactory realignment of Lisfranc's joint (Fig. 8.40).

Dressings and postoperative care

Non-adherent petrolatum gauze was applied to the wounds and covered with a fluffy compression dressing, followed by a plaster splint.

The patient was immobilized in fibreglass non-weightbearing casts for a period of 3 months. Casts were changed every 3 weeks. The patient was then placed in a walking brace for 1 month, and allowed to gradually return to full weightbearing. Custom-moulded shoes were provided. The postoperative recovery for this patient was essentially uncomplicated. The patient remained ambulatory and lived 4 more years, eventually succumbing to cardiovascular and renal complications.

Ankle arthrodesis

Arthrodesis for severe ankle deformity and instability has traditionally been reported to have a high incidence of non-union and pseudoarthrosis in patients with neuroarthropathy. Recent reports however, are more encouraging, with authors reporting success rates ranging from 66 to 100%. In cases where solid ankle fusion is not achieved, there may still be an acceptable outcome with fibrous ankylosis, when the foot is satisfactorily aligned beneath the leg. Failure to obtain fusion may be due to postoperative infection, deficiency of the arthrodesis, refracture through the site of fusion or hardware failure. The following case illustrates the surgical treatment for severe destruction and instability of the ankle in a patient whose deformity could not be braced or accommodated in custom footwear.

The surgical procedure was a tibiocalcaneal fusion with autogenous bone graft and intramedullary nailing. The goal of surgery was to realign the calcaneus under the tibia, in a plantigrade weightbearing position.

CASE STUDY

Tibiocalcaneal fusion with autogenous bone graft and intramedullary nailing

A 74-year-old gentleman, with type 2 diabetes of 17 years' duration, presented for initial consultation to the diabetic foot clinic with the chief complaint of swelling and deformity of his left ankle that developed suddenly and unexpectedly following an ankle sprain 6 months earlier. The patient had previously been treated elsewhere with a walking brace. Physical examination revealed marked deformity of the left ankle, characterized by swelling and displacement of the foot lateral to the leg. Pitting oedema extended up the leg to the level of the knee. There was a shallow ulcer 1.0 cm diameter on the medial malleolus that appeared to be caused by the brace. Skin temperature was elevated over the entire limb. Neurological examination revealed absent deep tendon reflexes at the ankle. Vibratory sensation was diminished below the knee, and absent below the malleoli. Loss of protective sensation was noted, with the patient unable to feel the Semmes–Weinstein 6.10 (90 g) monofilament. Pedal pulses were not palpable due to the swelling of the foot and ankle. Initial conservative management consisted of cast immobilization and non-weightbearing. Despite these efforts,

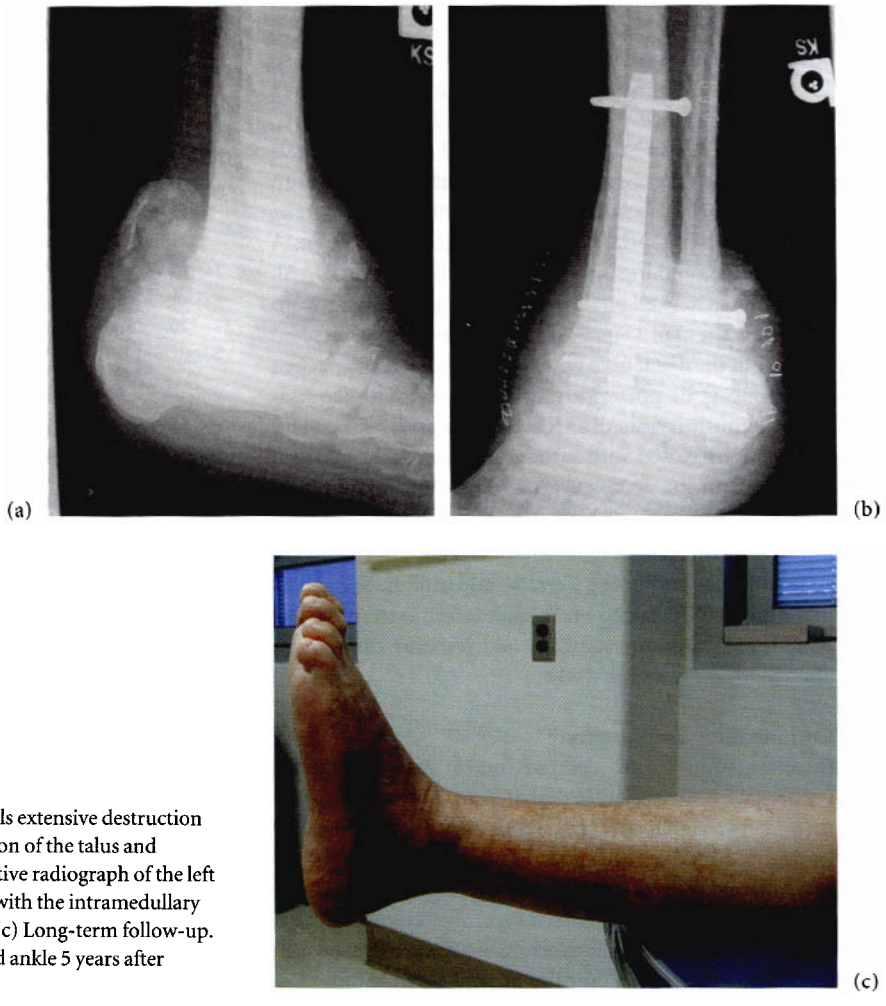


Fig. 8.41 (a) Lateral radiograph reveals extensive destruction of the left ankle joint with disintegration of the talus and fragmentation of bone. (b) Postoperative radiograph of the left ankle following tibiocalcaneal fusion with the intramedullary nail and interlocking screws in place. (c) Long-term follow-up. Clinical appearance of the left foot and ankle 5 years after surgery.

over the course of several months, ankle deformity and instability progressed, with disintegration of the talus (Fig. 8.41a). The patient was given the option of a salvage procedure, a tibiocalcaneal fusion vs. major amputation of the leg, and he opted for ankle fusion in the hope of saving his leg.

Technique

A standard medial incision was made over the medial malleolus, under fluoroscopic guidance, and the distal medial aspect of the tibia was exposed. The entire talus was noted to be destroyed and the foot was dislocated lateral to the leg. The distal tibia was then exposed subperiosteally and circumferentially and the bone was cut parallel to the standing alignment. A small portion of the talus was present laterally and this was planed down flat. Detritus was removed from the ankle. A filet guide pin was placed through the centre of the heel up through the calcaneus and up through the centre of the tibia. Sequential reamers were then utilized and reamed up to 12.5 mm. An 11 mm × 15 cm intramedullary nail was then selected and placed without difficulty. Two interlocking screws were placed in the calcaneus and then the nail was impacted into the distal tibia. The rotation was set with the tibial tubercle and then distal tibial interlocking screws were placed sequentially 70 mm, 70 mm and 70 mm. A 45-mm cross-linking screw was placed proximally. All screws were checked on the radiograph and were noted to be within the nail (Fig. 8.41b). The previously resected bone from the distal tibia was then morselized and packed with bone graft posteriorly and laterally. The wounds were copiously irrigated, a Hemovac drain was placed and wounds were closed with sutures and staples. The patient recovered on the acute surgical service for 1 week, was placed in a short-leg cast and was then transferred to the physical medicine and rehabilitation inpatient service for generalized conditioning exercise and ambulation training, non-weightbearing on the left lower extremity. The patient recovered uneventfully and remains ambulatory after nearly 5 years (Fig. 8.41c).

Complications of surgery

Infection, non-union, delayed union, pseudoarthrosis, progressive bone and joint destruction, pathological fractures, hardware failure, recurrent deformity and delayed wound healing have all been reported following surgery on patients with Charcot's joint disease. Attention to detail, targeted antibiotic coverage, meticulous surgical technique and postoperative non-weightbearing immobilization will help to minimize these complications.

ACKNOWLEDGEMENTS

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FURTHER READING

The diabetic foot

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Index

Page numbers in *italics* refer to figures; those in **bold** to tables. Index entries are arranged in letter-by-letter alphabetical order.