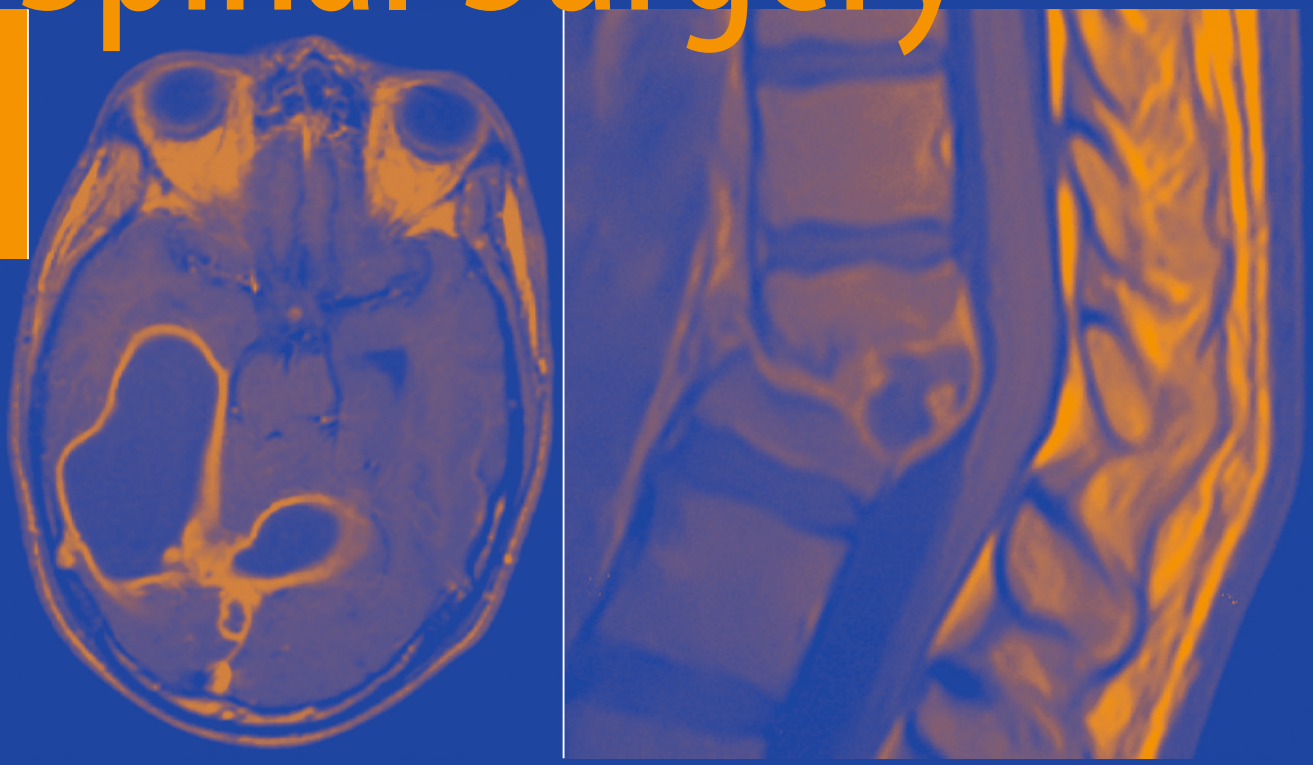
Ali Akhaddar

# Atlas of Infections in Neurosurgery and Spinal Surgery



Springer

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ISBN 978-3-319-60085-7 ISBN 978-3-319-60086-4 (eBook)
DOI 10.1007/978-3-319-60086-4

Library of Congress Control Number: 2017943166



Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

*To His Majesty Mohammed VI, King of Morocco*
*To my parents, my wife, and my children, with love*
*To my teachers, with gratitude*
*To my patients, who are my inspiration*

# Foreword I

Professor Ali Akhaddar has written a comprehensive *Atlas of Neurosurgery and Spinal Surgery Infections*. This book will become a standard reference for neurosurgeons and spinal surgeons in the future, as it is very difficult to find this information so well described in one place. I foresee it being used in all departments of neurosurgery and spinal surgery and in infectious disease specialties. As time goes on, this area of neurosurgery will receive more attention because advances in the diagnosis and treatment of CNS-associated infections will benefit from the advances in precision medicine, in which the approach to diseases is precisely matched to each person's genetics.

Dr. Akhaddar is to be complimented on undertaking this work, as infection is mostly ignored by neurosurgeons and spinal surgeons and left to our colleagues in other specialties to treat. That approach leads to the further disintegration of the specialty of neurosurgery, which encompasses the diagnosis and treatment of all diseases affecting the central nervous system, including the spine. I suggest that all neurosurgeons and spinal surgeons obtain a copy of this fine book as a standard reference. It would also be of value to have a neurosurgeon devote time to this specialty of neurosurgery during residency, as proficiency in the knowledge of infectious disease is very important in both the developing world and developed world. It would also be valuable for departments of neurosurgery to add members of the infectious diseases specialty to the department of neurosurgery. Rounds made with these specialists could add further dimension to the understanding of infectious diseases treated in other organs that would have application to neurosurgery patients.

This is an excellent work.

James I. Ausman, MD, PhD
Professor of Neurosurgery, University of California, Los Angeles (UCLA), USA
Former Chairman of Neurosurgery, Henry Ford Hospital and University of Illinois at Chicago, USA
Editor in Chief, *Surgical Neurology International*

# Foreword II

This book, written and edited by Prof. Akhaddar, is an important contribution to the improvement of the neurosurgical care of the patients. It covers an area that is not so frequently discussed in the literature. Textbooks of this format on the topic—atlas with multiple figures, illustrating the text—are essential for the education of young colleagues, especially in those parts of the world where infectious diseases present a major health problem.

Prof. Ali Akhaddar is very experienced in this field, which allows him to highlight the most important points for the reader. The textbook is very well structured: each chapter has an epidemiology and etiology, clinical presentation, imaging features, laboratory findings, treatment options, and outcome subsections. All the 30 chapters are very well illustrated. It covers not only the most frequent infections but also diseases that in the Western world are just of academic interest. However, in some parts of the world, they are not uncommon and their treatment is very challenging.

I would like to congratulate and thank Prof. Akhaddar for his efforts to prepare this textbook, which I certainly recommend not only to the young neurosurgeons but also to those that are less experienced in the field of infectious diseases.

Madjid Samii, MD
Professor of Neurosurgery
President of the International Neuroscience Institute, Hannover, Germany
Honorary President of the World Federation of Neurosurgical Societies (WFNS)

# Preface

If there's a book that you want to read, but it hasn't been written yet, then you must write it.

Toni Morrison (*Nobel Prize in Literature, 1993*)

Management of central nervous system (CNS) and spinal infections is challenging. Without early diagnosis and adequate treatment, these serious diseases can result in permanent neurologic deficits, seizure, spinal deformities, and, in severe cases, generalized sepsis and death. Modern neurosurgery and spinal surgery have received a great deal of consideration in the literature, but the topic of craniospinal infections has not attracted comparable attention. It is essential to recognize and become familiar with the variety of infectious conditions that should continue to preoccupy our surgical community, and this atlas focuses on CNS, cranial, and spinal infections from a surgical perspective. Classically, cranial and spinal infections are arranged by the anatomical location involved. The development of these diseases requires a good knowledge of the pathogens implicated, the patients' predisposing factors and comorbidities, sources of infections, and mechanisms of spread. Although the most common origin of neurosurgical infections is nonspecific bacteria, the role of other microorganisms should not be overlooked. The particular topic of postoperative cranial and spinal infections is also considered.

*Ubi pus*, *ibi evacua* ("If there's pus about, let it out.")

Today, this well-known Latin aphorism is not always appropriate for infections encountered in neurosurgery and spinal surgery: first, because of the singular anatomic composition of the CNS and its coverings; second, because of the advent of antimicrobial chemotherapy; and finally, because of the wide variety of clinical presentations and infectious lesions that affect these body areas. Presently, the spectrum of treatment goes from antibiotic therapy alone to combination with surgical drainage to more invasive surgical procedures.

CNS infections differ from those of other organ systems in many ways. The brain and spinal cord are protected from infection by the skull and the spine and are surrounded by layers of meninges, which serve as a mechanical barrier—a defense reinforced by the chemical and mechanical filtering capacities of the blood-brain barrier. The composition of the cerebrospinal fluid (CSF) makes it a very good culture medium, however, as the CNS and subarachnoid space are regarded as immunologically sequestered because of the lack of a lymphatic system.

Though many of the infectious processes that affect the CNS and the spine can result in severe sequelae and even death, the prognosis of these infections has improved significantly over the past 30 years, in great measure as a result of improved techniques to aid diagnosis, modern antibiotics and surgical procedures, and intensive care facilities. These make neurosurgical and spinal infections more challenging to manage, especially with the changing traits of many infectious diseases in the past few decades. Indeed, increases in migratory flows, refugee movements, international travel, and immunocompromising conditions have advanced the likelihood of detecting infectious diseases that are usually uncommon, especially in developed countries. In addition, the augmentation of cranial and spinal procedures worldwide has increased the relative incidence of postoperative infectious complications, even as other sources of infection have decreased. It is well recognized that severe postsurgical infection is a real

nightmare for the neurosurgeon and the spinal surgeon. In all cases, the early identification of the infectious agent and aggressive medical treatment (with or without further surgery) are decisive in achieving the best outcome.

For many scientists, old and new infections will occur in the future, as they did in the past. Meanwhile, updated information on the diagnosis and management of cranial and spinal infections is required, especially for clinicians, surgeons, neuroradiologists, and laboratories, which may be unfamiliar with the wide array of clinical presentations and neuroimaging characteristics of these diseases. Furthermore, modern diagnosing and managing of patients with infectious diseases are done largely through examination of "visual signs."

Most textbooks of neurosurgery and spinal surgery neglect the subject of infection or mention it only briefly, but infections will continue to be part of cranial and spinal practices, and we surgeons must consider this reality. Colleagues at any stage of training must also live with the consequences of cranial and spinal infections, increase experience from them, and learn the appropriate lessons. It is essential for us to recognize the variety of infectious conditions that continue to be seen in our practices.

What the mind does not know, the eyes cannot see—Johann Wolfgang von Goethe

With the introduction of the multidisciplinary and interdisciplinary team approach, a need has become evident for a timely and concise book about CNS and spinal infections from a surgical perspective. This book combines illustrations and biological, clinical, radiological, and surgical images taken from the author's extensive library (1997–2017) to provide readers with unparalleled access to a comprehensive collection of craniospinal infectious images. "One picture is worth a thousand words" (*Tess Flanders*). This atlas is designed to complement and provide a visual supplement to already existing good textbooks on CNS infections. The involvement of each lesion and area is dealt with in a brief and easy-to-comprehend manner. In a unique way, various neuroimaging and laboratory abnormalities are then linked to the clinical features, treatment procedures, and surgical views, to encourage a smooth and easy practical integration. Practicing neurosurgeons, spinal surgeons (including orthopedists), neurologists, rheumatologists, neuroradiologists, infectious disease specialists, rehabilitation physicians, microbiologists, pharmacologists, histopathologists, and other clinicians and researchers worldwide will find a comprehensive visual encyclopedia using more than 1,140 parts of figures of CNS, cranial, and spinal infections.

The 30 chapters of this book cover most common infectious conditions seen in neurosurgical and spinal practices and requiring surgical interventions. It is divided into five sections: a general introduction, craniocerebral infections, vertebromedullary infections, infections following cranial and spinal surgery, and a section describing the most important specific pathogens and other particular conditions.

The main goal is to deliver more information in less space than traditional prose. Besides documenting the work, this atlas has a teaching value. The format makes it easily accessible, as it includes a definition of each infection and its epidemiology and etiology, main clinical presentations, imaging features, laboratory findings, treatment options, and outcome information. It will help the reader in choosing the most appropriate way to manage this multipart problem. We hope this atlas will provide a timely addition to the fields of neurosurgery, spinal surgery, and infectious diseases.

Marrakech, Morocco
December 2016

Ali Akhaddar, MD, IFAANS

# Acknowledgments

The author would like to acknowledge the following individuals for their continuous support and encouragement:

- *Prof. Brigadier General Abdelkrim Mahmoudi*, *MD*. Health Inspector of the Moroccan Royal Armed Forces
- *Prof. Driss Aboutajdine*, *PhD*. Former Director of the Moroccan National Center for Scientific and Technical Research (CNRST). May God bless his soul, rest in peace
- *Prof. Mohamed Adnaoui*, *MD*. Dean of the Faculty of Medicine and Pharmacy, Mohammed V University in Rabat, Morocco
- *Prof. Colonel Major Khalid Sair*, *MD*. Chief Medical Officer of Avicenne Military Hospital, Marrakech, Morocco
- *Prof. Mohamed Boucetta*, *MD*. Mentor and Former Chairman of the Department of Neurosurgery, Mohammed V Military Teaching Hospital, Rabat, Morocco
- *Mrs. Khadija Akhaddar*, Deputy Director of Administration, CPE Aux Petits Lurons at Gatineau, Québec, Canada
- *Prof. Redouane Abouqal*, *MD*. Head of the Laboratory of Biostatistics, Clinical and Epidemiological Research (LBRCE), Faculty of Medicine and Pharmacy, Mohammed V University in Rabat, Morocco
- *Prof. Mohamed ou Ayad Kerouach*, Agricultural and Forestry Engineer, Bookseller in Rabat, and Provider/Distributor in Morocco of the main international scientific editions

The author wishes also to thank the following colleagues for providing this book with some illustrations and pictures:

- *Prof. Mohamed Boucetta*, *MD*. Former Chairman of the Department of Neurosurgery, Mohammed V Military Teaching Hospital, Rabat, Morocco
- *Prof. Mostafa Elouennass*, *MD*. Department of Microbiology, Mohammed V Military Teaching Hospital, Rabat, Morocco
- *Prof. Mohamed Mahi*, *MD* and *Prof. Hassan En-nouali*, *MD*. Department of Radiology, Mohammed V Military Teaching Hospital, Rabat, Morocco
- *Prof. Mohamed Zalagh*, *MD*. Department of Otorhinolaryngology, Mohammed V Military Teaching Hospital, Rabat, Morocco
- *Prof. Prasad Krishnan*, *MD*. Department of Neurosurgery, National Neurosciences Centre, Kolkata, West Bengal, India
- *Prof. Mohamed Lakouichmi*, *MD*. Department of Maxillofacial Surgery, Avicenne Military Hospital, Marrakech, Morocco
- *Prof. Redouane Moutaj*, *PharmD*. Department of Parasitology and Mycology, Avicenne Military Hospital, Marrakech, Morocco
- *Dr. Abderrahim El Ktaibi*, *MD* and *Prof. Issam Rharrassi*, *MD*. Department of Histopathology, Avicenne Military Hospital, Marrakech, Morocco
- *Dr. Redouane Rokhssi*, *MD* and *Prof. Nabil Hammoune*, *MD*. Department of Radiology, Avicenne Military Hospital, Marrakech, Morocco

- *Dr. Hassan Baallal*, *MD*. Department of Neurosurgery, Avicenne Military Hospital, Marrakech, Morocco
- *Dr. Abad Cherif El Asri*, *MD*. Department of Neurosurgery, 5th Military Hospital, Guelmim, Morocco

Also many thanks are due to the following for their assistance in the preparation of the text and the illustrations:

- *Mr. Saïd Mahfoud*, English Teacher, Marrakech, Morocco
- *Prof. Abderrahman Boukhira*, *PharmD*. Department of Biochemistry, Avicenne Military Hospital, Marrakech, Morocco
- *The team of Springer-Nature US* for coordinating various phases of this project, especially Richard A. Hruska (Executive Editor, New York, USA) and Lee Klein (Developmental Editor, Philadelphia, USA) and their co-workers.

Finally, the author respectfully thanks and extends deep appreciation to *Prof. James Ausman* and *Prof. Madjid Samii* for kindly agreeing to write forewords for this book.

# Contents

# Part I

# General Considerations

# Classification and Sources of Infections

# 1

Infectious diseases of the CNS are classified into diffuse and focal forms. The diffuse lesions are related to the involved compartment, such as meningitis, meningoencephalitis, encephalitis, myelitis, radiculomyelitis, or myeloencephalitis. The focal lesions mainly correspond to granulomas, suppurative collections, and cysts. Diffuse lesions are mainly encountered in neurological practice, whereas focal lesions are found in neurosurgery.

Classically, cranial and spinal infections are arranged by the anatomical location involved (Tables 1.1 and 1.2). Cranial infections include scalp abscesses, cranial osteomyelitis, intracranial infections (Fig. 1.1), and infectious suppurations of the orbit. Spinal infections include paraspinal soft tissue infections (Fig. 1.2), vertebral column and discal infections (spondylodiscitis) (Fig. 1.3), and spinal canal infections (Figs. 1.4 and 1.5). Most spinal canal infections are extradural, but less commonly, they may be intramedullar, and they can also be intradural extramedullary (called spinal subdural infections).

CNS and spinal infections can occur in healthy individuals, but some comorbidities or specific underlying conditions may be involved in developing cranial or spinal infections. Among these are the following conditions, which compromise the immune system:

- Advanced age
- Intravenous drug abuse
- Defective immune system
- HIV infection
- Use of immunosuppressive medication or long-term systemic use of corticosteroids
- Diabetes mellitus
- Organ transplantation
- Malnutrition
- Cancer

Various other predisposing causes have been suspected, such as obesity, smoking, prolonged preoperative stay, colonization with *Staphylococcus aureus*, irradiation, hemodialysis, and being a newborn.

Regarding the sources of infection, causative pathogens may reach the CNS, the cranial bones, or the spine using different likely portals of entry and mechanisms of spread, especially these most usual possibilities:

- Extension from contiguous sites of adjacent infection
- Posttraumatic or postsurgical direct inoculation
- Hematogenous dissemination from a remote source of infection

Postoperative cranial and spinal infections are serious complications after surgical interventions; they can lead to poor patient outcomes and increase health-care costs. Classically, neurosurgical interventions are divided into five categories: clean, clean with foreign body, clean-contaminated, contaminated, and dirty. Risk factors for postoperative infections after neurosurgical procedures include an ASA (American Society of Anesthesiologists) score higher than 2, postoperative monitoring of intracranial pressure, ventricular drains, CSF leak, longer duration of procedures, placement of foreign bodies, repeat or additional neurosurgical procedures, shunt infections, and emergency procedures. Foreign body materials erroneously left behind during neurosurgical or spinal interventions (retained surgical sponges, sometimes called *textilomas*) cause foreign body reaction in the contiguous tissue and may be the origin of subacute or chronic suppuration. Most postoperative cranial and spinal infections are discussed in Section IV of this book, including infectious complications with cranial and spinal implanted devices.

Medical infectious complications should be considered in our neurosurgical and spinal practices even when these adverse events are not directly associated with surgery or surgical techniques. Potential sources of infection include the CNS; the cardiovascular, pulmonary, gastrointestinal, and genitourinary systems; skin and soft tissue; bones and



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**Table 1.1** Anatomic localization of cranial infections

| Level | Anatomic space | Disease |
|---|---|---|
| **Scalp** | Scalp layers | Subcutaneous abscess |
| | | Cellulitis |
| | | Subgaleal abscess |
| | | Draining sinus tract |
| **Skull** | Bone | Osteomyelitis |
| | Extradural space | Extradural abscess |
| **Dura mater** | Subdural space | Subdural empyema |
| | Dural sinus | Dural sinus thrombophlebitis |
| **Arachnoid mater** | Arachnoid layer | Arachnoiditis |
| | | Pachymeningitis |
| | Subarachnoid space | Meningitis |
| **Pia mater/brain parenchyma** | Pia mater | Cortical venous thrombosis |
| | Brain parenchyma | Brain abscess |
| | | Encephalitis |
| | Ventricle | Ventriculitis |
| | | Pyoventriculitis |
| **Orbit** | Orbital space | Orbital infections |

**Table 1.2** Anatomic localization of spinal infections

| Level | Anatomic space | Disease |
|---|---|---|
| **Paraspinal** | Cutaneous/ subcutaneous | Abscess |
| | | Cellulitis |
| | | Draining sinus tract |
| | Paraspinal muscles | Paraspinal pyomyositis |
| **Spine** | Vertebra (bone) | Vertebral osteomyelitis |
| | Intervertebral space | Discitis |
| | Vertebra and intervertebral space | Spondylodiscitis |
| **Spinal canal** | Spinal epidural space | Spinal epidural abscess |
| | Spinal subdural space | Spinal subdural abscess (intradural extramedullary abscess) |
| | Arachnoid mater | Arachnoiditis |
| | | Pachymeningitis |
| | Subarachnoid space | Meningitis |
| | Spinal cord parenchyma | Spinal cord abscess |
| | | Myelitis |

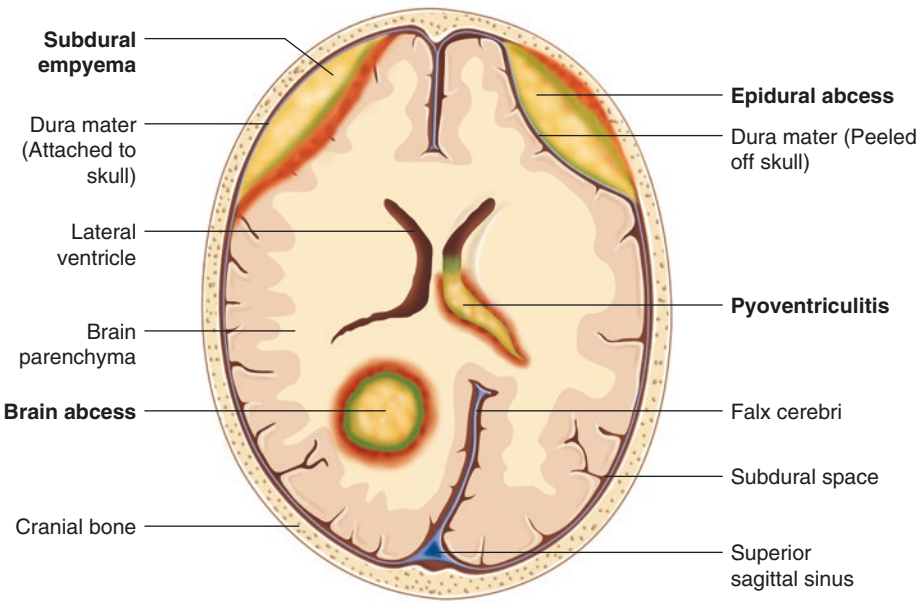


**Fig. 1.1** Different types of intracranial suppurative collections

joints and paranasal sinusitis; and bacteremia. However, wound infection, urinary tract infection, pneumonia, and intravascular catheter-related infection are the most prevalent infections in our practice and must not be ignored or missed, especially when they are regarded as "minor complications." Fever is usually related to infection, but most fevers appear within the first 72 h after neurotrauma or surgical procedures and resolve spontaneously. Table 1.3 lists the classic "five Ws" mnemonic for the evaluation of postoperative fever. Careful clinical assessment drives decision-making to optimize care of our patients.

Postoperative infectious complications can be reduced with more standardized protocols for infection prophylaxis. This topic is outside the scope of this atlas, however.

Although the most common origin of neurosurgical infections is nonspecific bacteria, the role of other microorgan-

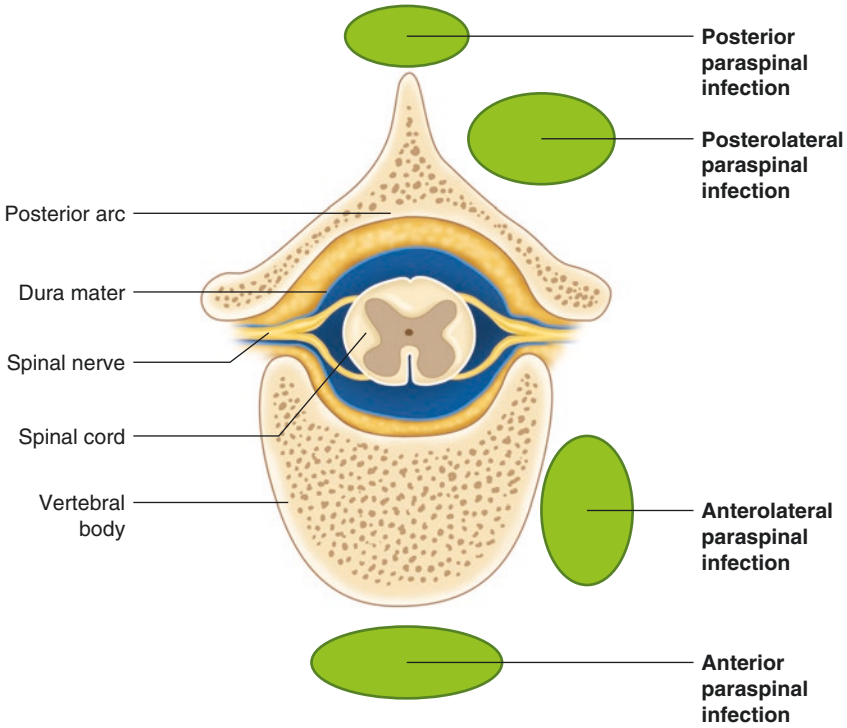


**Fig. 1.2** Topographic classification of paraspinal infections

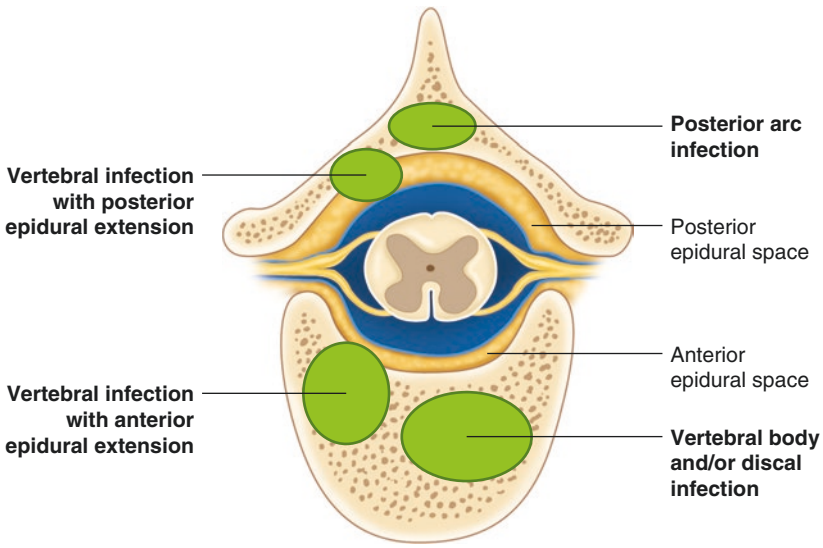


**Fig. 1.3** Topographic classification of spinal infections

isms should not be overlooked. In consideration of this fact, Section V is devoted to understand diagnostic and therapeutic approaches for other specific bacterial, fungal, and parasitic pathogens, as these pathologies have received much interest in recent years.

The neurosurgeon's role in the management of most of patients with *diffuse* lesions of the CNS is in providing tissue by biopsy and/or CSF for diagnosis, as well as surgical management of increased intracranial pressure. Regarding craniocerebral and spinal *focal* lesions, the surgeon has a number of roles:

- Obtaining tissue samples for definitive diagnosis (by stereotactic or image-guided needle biopsy)
- Aspiration/excision of the space-occupying lesions or reduction of its mass effect
- Decompressing neural structures (via craniotomy/craniectomy or laminectomy procedures)
- CSF shunting against symptomatic hydrocephalus
- Debriding infected and necrotic tissue when infection persists or worsens despite appropriate antimicrobial regimens
- Treating spinal instability and significant deformity

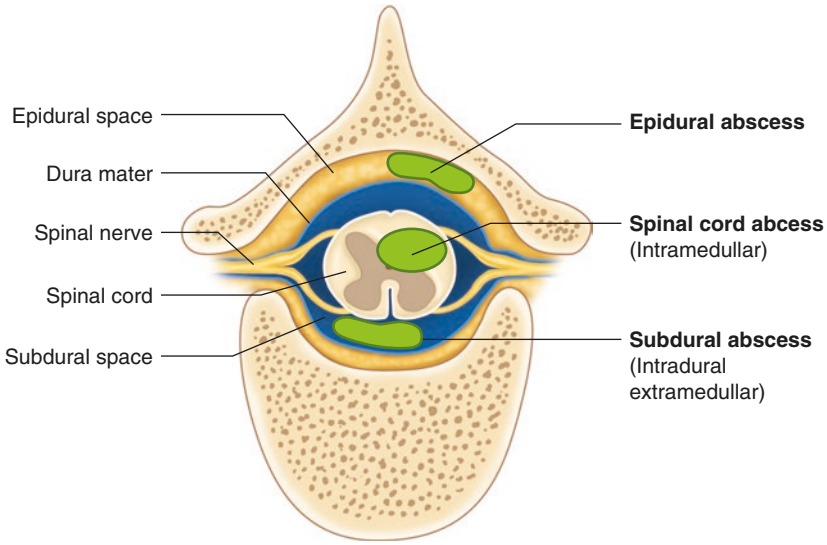


**Fig. 1.4** Topographic classification of spinal canal abscesses (transversal)

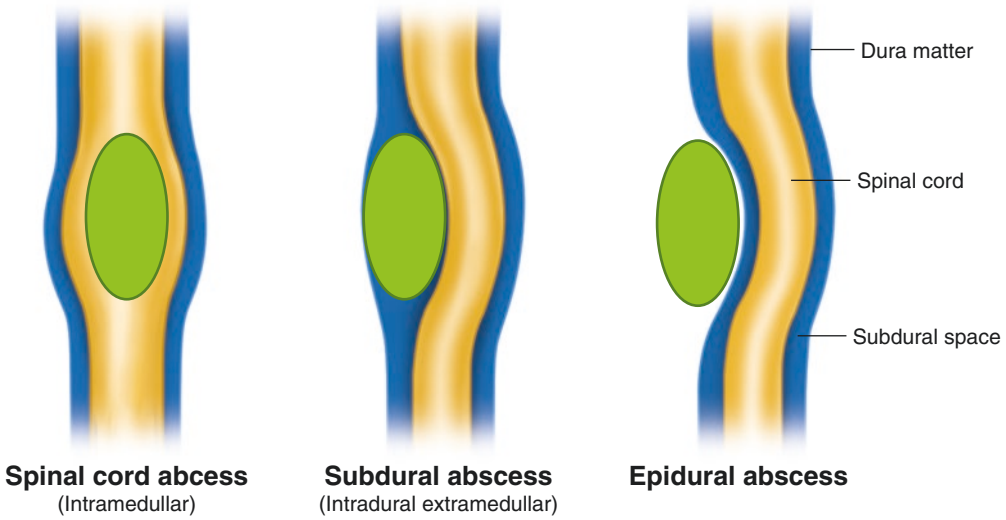


**Fig. 1.5** Topographic classification of spinal canal abscesses (longitudinal)

**Table 1.3** The "five **W**s" mnemonic of most common causes of post-operative fever in the order in which they occur

| | |
|---|---|
| **W**ind | Pneumonia |
| | Atelectasia |
| **W**ater | Urinary tract infection |
| | Intravascular line infection |
| **W**ound | Wound infection |
| **W**alk | Venous thromboembolism |
| **W**onder drugs | Drug/antibiotic fever |
| | Transfusion reaction |

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# Laboratory Collections and Sample Processing

# 2

The CNS and its coverings may be infected by various microorganisms, including nonspecific and specific bacteria, fungi, and parasites. These infecting organisms differ according to age of onset, pathogenesis, anatomic location, and underlying medical status. The rapid recognition of the pathogenic agent is crucial for the successful treatment of any infectious disease. Efforts must be undertaken to identify more and more causative pathogens. With this in mind, the correct collection and transfer of specimens are also decisive for the isolation, identification, and characterization of microorganisms that cause cranial or spinal infection. Pus, suspected fluids, infected tissue, bone, and debrided material should be sent for routine microbiological and histopathologic studies including aerobic and anaerobic bacteria, acid-fast bacillus (AFB), and fungal culture, as well as antimicrobial sensitivity testing (Figs. 2.1, 2.2, 2.3, 2.4, 2.5, 2.6, 2.7, 2.8, and 2.9).

Bacteria and fungi have characteristic appearance on a variety of culture mediums, and the final identification is based on biochemical tests (Figs. 2.10, 2.11, 2.12, 2.13, 2.14, 2.15, 2.16, 2.17, 2.18, 2.19, and 2.20). It is also important to recognize that many cranial or spinal infections are polymicrobial, and culture-negative cases are not insignificant. Molecular studies may be useful to detect organisms that are difficult or slow to grow by culture. The results of antimicrobial susceptibility testing should be combined with clinical and neuroimaging information, experience, and local epidemiological data when selecting the most appropriate anti-infectious drugs for the patient. Histopathology is also useful for recognizing some specific infections such as mycobacteria, fungi, and parasites and to rule out the presence of a compounding tumoral, granulomatous, or atypical inflammatory process.

Clinicians and biologists must work together in *perfect coordination*. To avoid loss of viability of the pathogens, clinical and surgical specimens should be obtained before delivering any antimicrobial therapy. Whenever possible, bacterial cultures should be obtained using tissue specimens rather than swabs. Moreover, cultures from the sinus tracts may be deceiving because of contamination with bacteria and/or fungi from the skin. Preferably, both culture and histological examination should be done simultaneously and transported to the laboratories immediately. Ideally, three blood cultures obtained at different times should be performed. Collecting the sample during a fever spike or a septicemic phase increases the yield.

It is important to take into consideration that many cranial and spinal infections are polymicrobial and can even consist of both anaerobic and aerobic bacteria as well as mixed fungi and bacteria or mycobacteria and pyogenic bacteria. Therefore, special anaerobic and fungi transport medium must be used without introducing air during direct injection of liquid samples. Properly gram-stained preparation and analysis are simple and fast methods to give preliminary information to the clinicians. Special suspicions about bacteria, fungi, or parasites must be communicated to the laboratory, because the diagnosis may be overlooked using routine microbiologic methods.

After isolation and identification of the infecting microorganisms, antimicrobial susceptibility testing is performed



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**Fig. 2.1** Sterile universal container for sending biopsy material, fluid, and pus specimens for culture: closed (**a**) and opened (**b**)

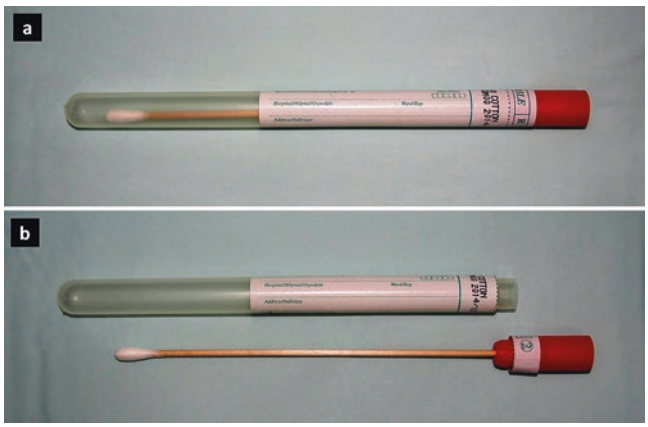



**Fig. 2.2** Medical swab and its holder for sampling and transporting bacteria and fungi for culture: closed (**a**) and opened (**b**)

**Fig. 2.3** Blood bottles with specific media for aerobic and anaerobic cultures. Samples other than blood may be received in these bottles. In the laboratory, bottles are loaded up in an automated system

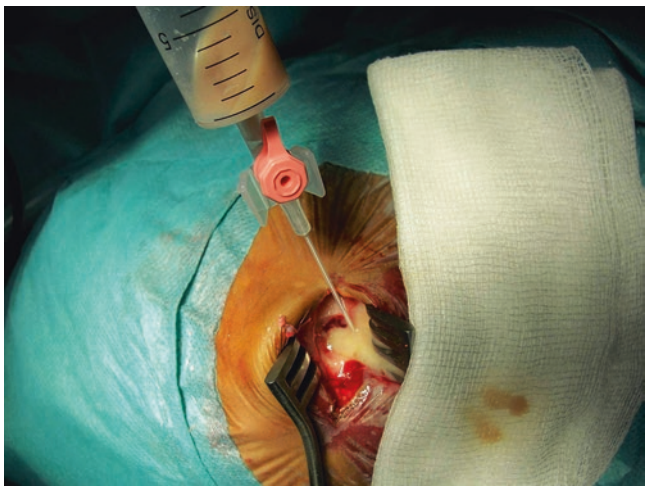


**Fig. 2.4** Operative view. Burr hole drainage of an intracranial epidural abscess

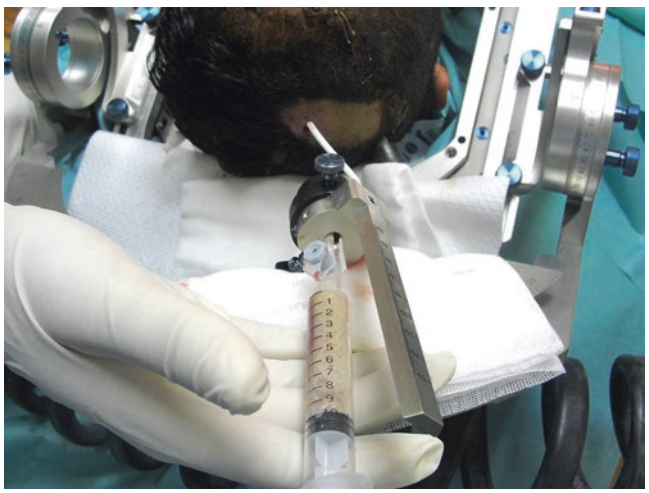


**Fig. 2.5** Operative view. Stereotactic aspiration of an intracranial subdural empyema

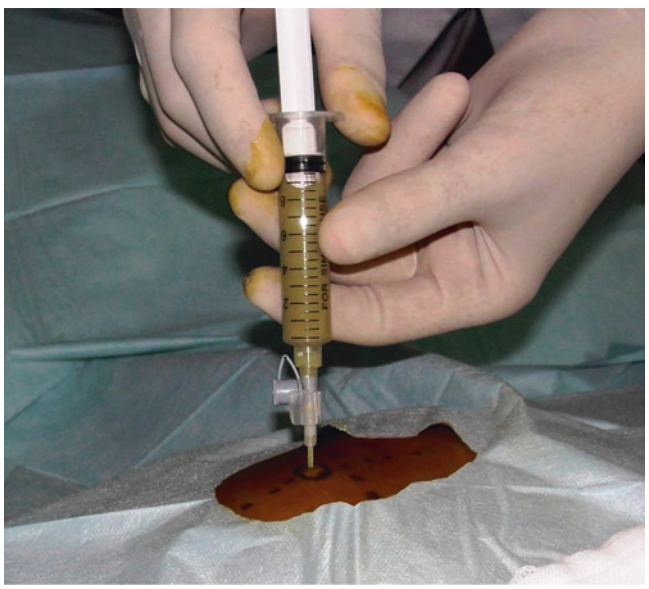


**Fig. 2.6** Percutaneous aspiration of a posterior paraspinal abscess

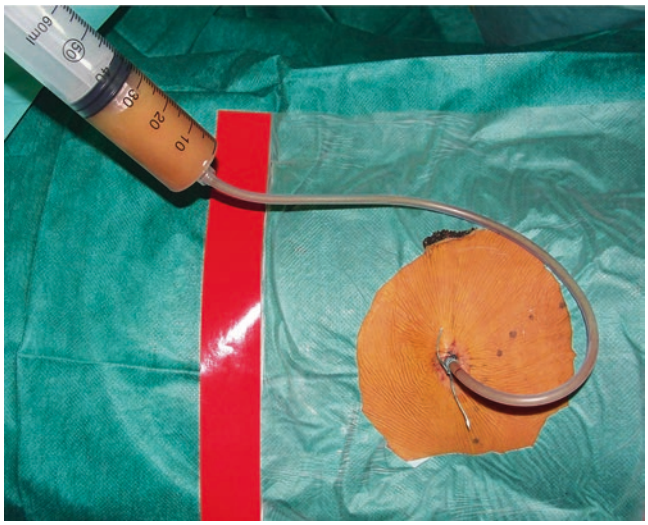


**Fig. 2.7** Posterior percutaneous aspiration with drainage of a psoas abscess in the *left* side under local anesthesia. *Mycobacterium tuberculosis* was identified in the cultured specimens

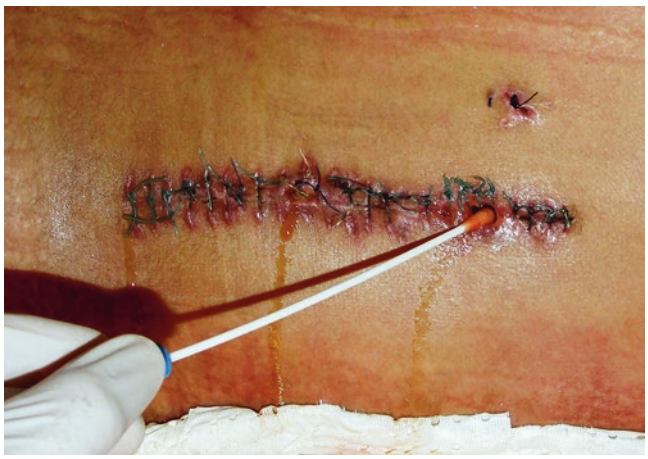


**Fig. 2.8** Postoperative infection of a surgical wound. It is important to swab the surgical wound bed

in vitro to predict in vivo success or failure of antibiotic therapy (Figs. 2.21 and 2.22). Hospital-acquired pathogens such as methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant *S. aureus* (VRSA) are increasingly resistant to multiple classic antimicrobial therapies. The choice of optimal medical therapy is determined by antimicrobial penetration to the appropriate organ and in vitro susceptibilities of the causative pathogens. Clinical and neuroimaging information, experience, and local epidemiological data should be taken into account when selecting the most appropriate antibiotic for the patient. Treatment should begin without delay after specimens have been taken, especially in patients with severe sepsis. The treatment must be adjusted once the pathogen has been identified and the antibiotic sensitivities are known.

**Fig. 2.9** Samples and purulent material are collected and transported to the laboratories in sterile universal containers and specific bottles

Screening for other potential sources of infection is important. Cultures from possible primary or secondary infectious focus should also be obtained, especially otorhinolaryngology, oral, sputum, urine, skin, and blood cultures. Unlike the other sources, pathogens that spread through the bloodstream are often monomicrobial.

Molecular studies like DNA probes or polymerase chain reaction (PCR) for genetic analysis are more sensitive than culture-dependent methods but may be less specific and are not widely available for routine diagnostic intentions.

Biopsy specimens are also helpful for histopathologic studies to rule out the presence of a compounding tumoral, granulomatous, or atypical inflammatory process. Histopathology is also very useful for recognizing some specific infections such as mycobacteria, fungi, and parasites, particularly when clinical information, biological data, and neuroimaging investigations are not conclusive (Figs. 2.23, 2.24, 2.25, 2.26, 2.27, 2.28, 2.29, 2.30, 2.31, 2.32, 2.33, 2.34, and 2.35).

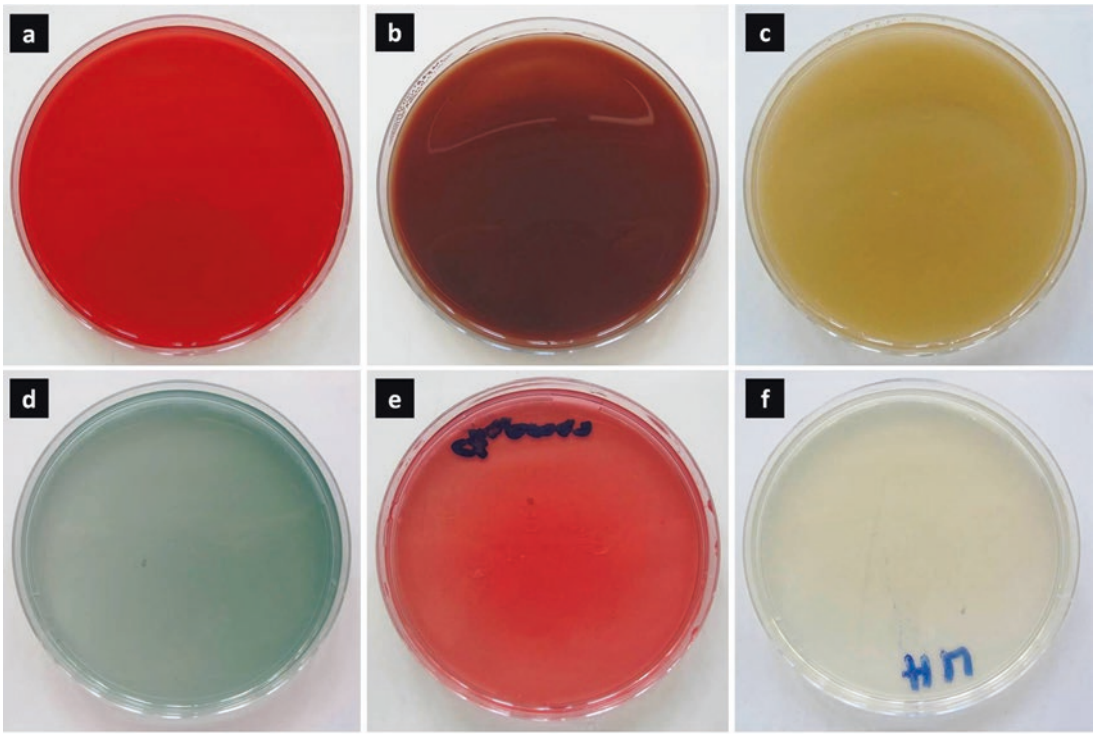



**Fig. 2.10** Different bacterial culture plates for routine bacterial cultures. Blood agar (**a**), chocolate agar + PolyViteX (**b**), chromogenic agar medium (**c**), cystine lactose electrolyte-deficient (CLED) agar (**d**), mannitol salt agar (Chapman medium) (**e**), and Mueller–Hinton (MH) agar (**f**)

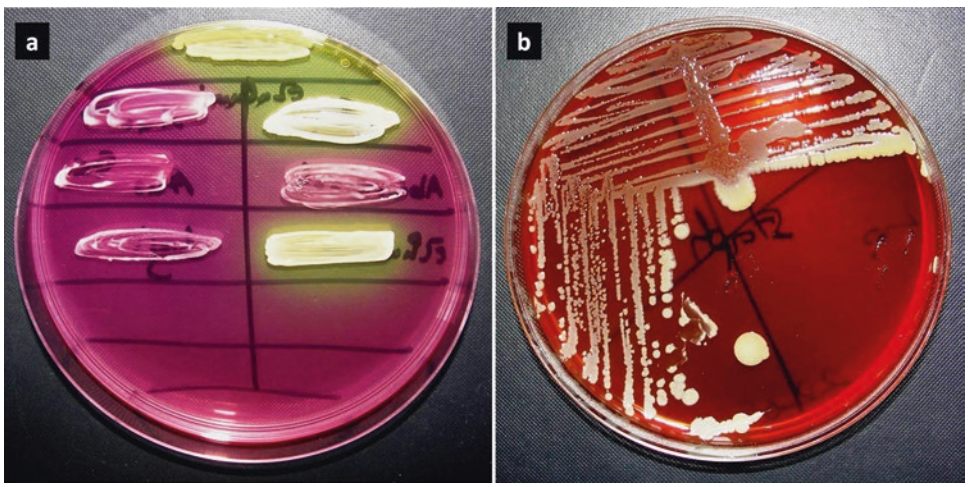



**Fig. 2.11** Culture of colonies of *Staphylococcus aureus* on mannitol salt agar (Chapman medium) (**a**) and on blood agar (**b**)

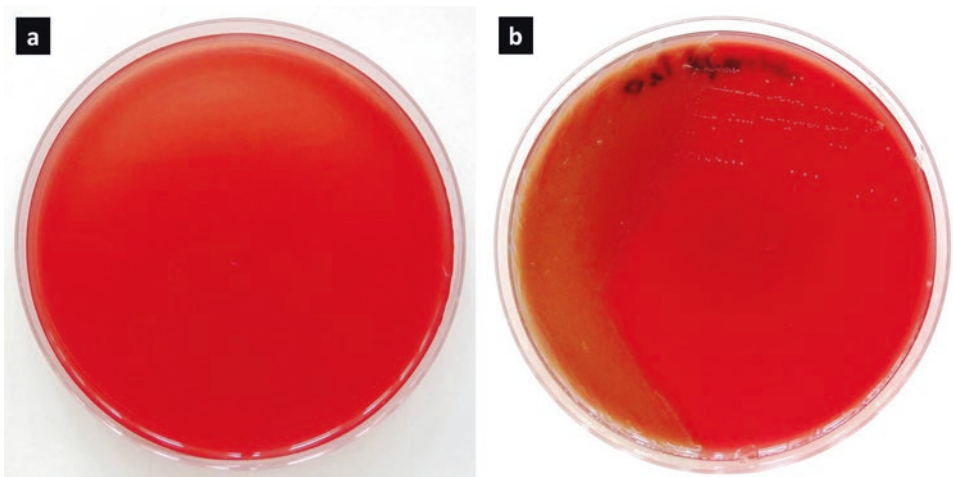



**Fig. 2.12** Blood agar plate without (**a**) and with (**b**) colonies of alpha-hemolytic streptococci

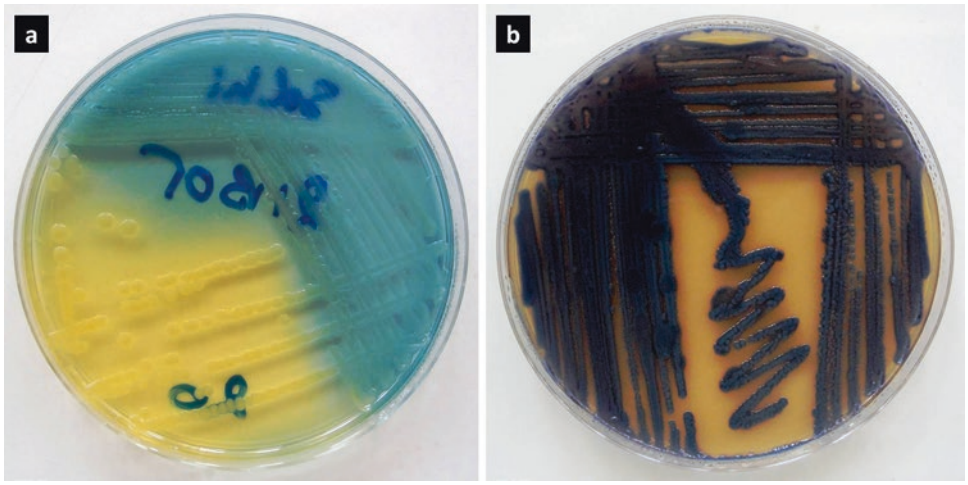



**Fig. 2.13** Culture of colonies of *Klebsiella pneumoniae* on CLED agar (**a**) and chromogenic medium (**b**)

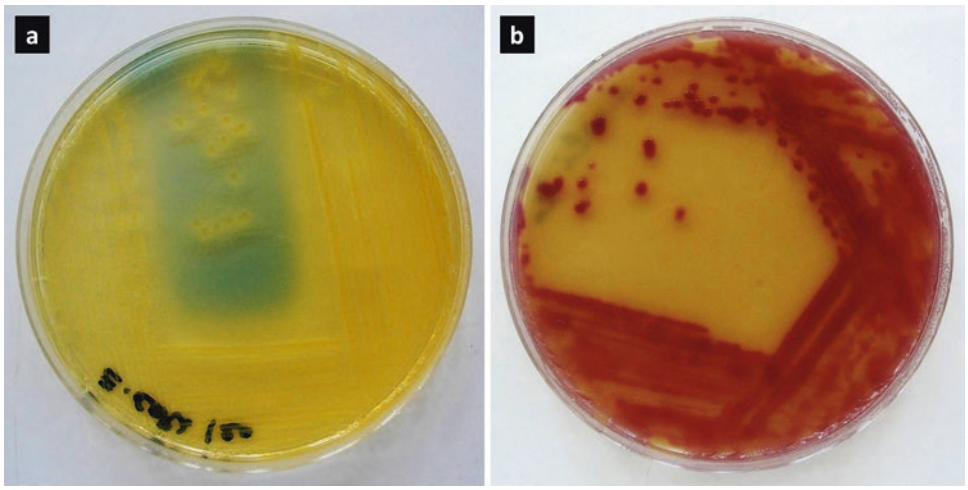



**Fig. 2.14** Culture of colonies of *Escherichia coli* on CLED agar (**a**) and chromogenic medium (**b**)

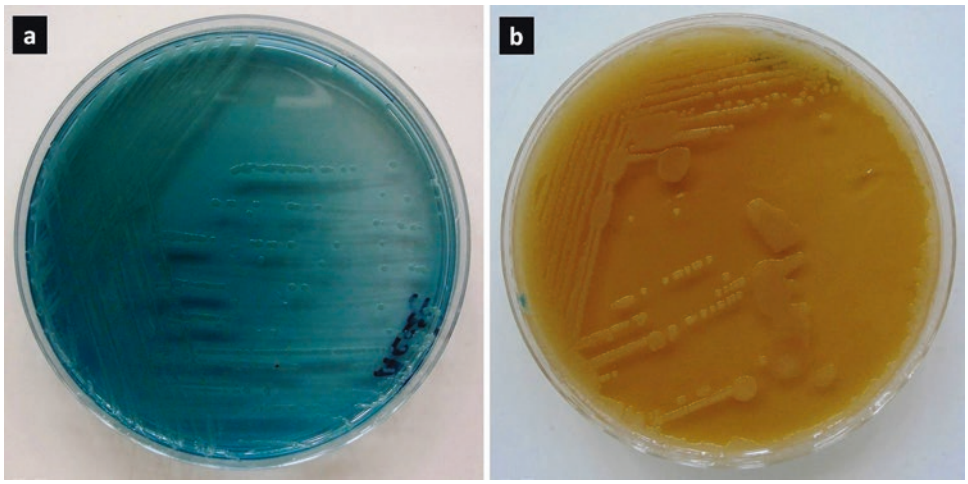



**Fig. 2.15** Culture of colonies of *Acinetobacter baumannii* on CLED agar (**a**) and chromogenic medium (**b**)

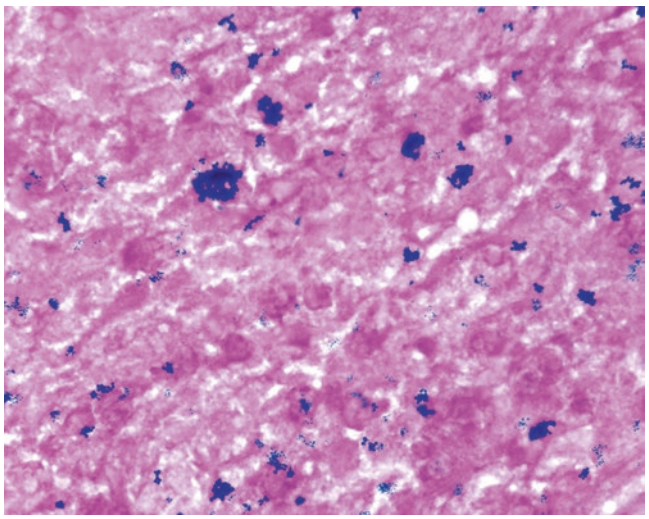


**Fig. 2.16** Gram stain (positive) of *Staphylococcus aureus* from a scalp infection

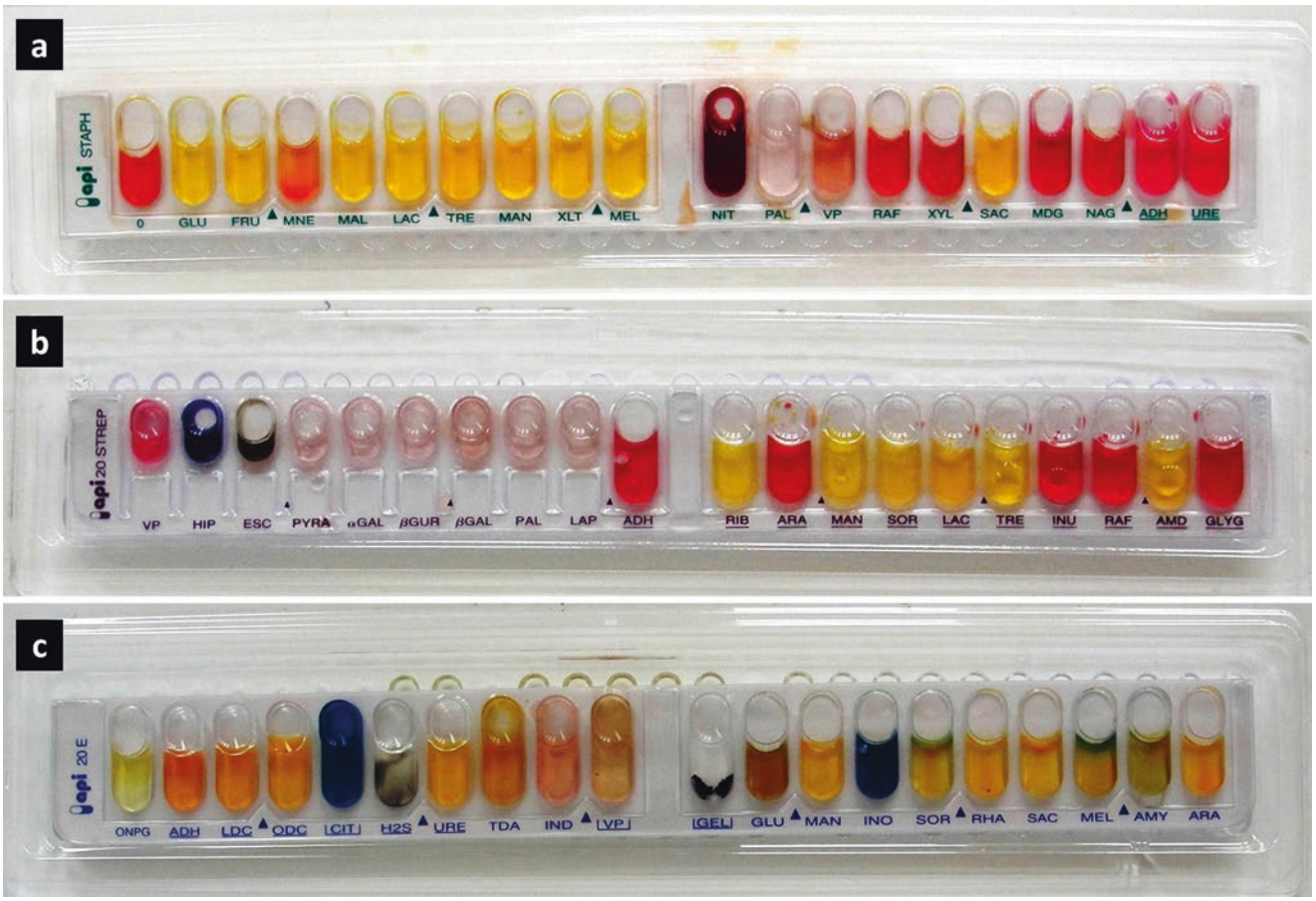



**Fig. 2.17** Bacterial identification using gallery of the analytical profile index (API®) system (bioMérieux, Inc.). This system is developed for quick identification of clinically relevant bacteria. API® Staph for *Staphylococcus* species (**a**), API® 20 Strep for *Streptococcus* species (**b**), and API® 20E for *Enterococcus* species (**c**)

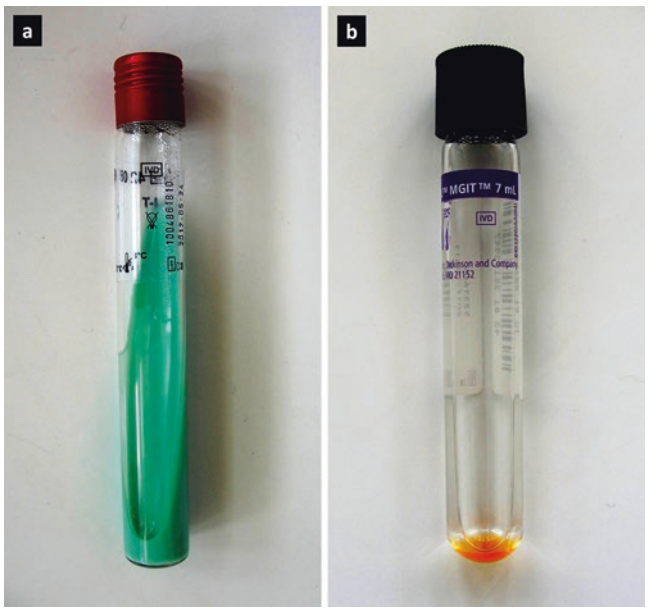



**Fig. 2.18** Lowenstein–Jensen medium slant (*solid medium*) (**a**) and Mycobacteria Growth Indicator Tube (MGIT) (*liquid medium*) (**b**) for detection of mycobacteria

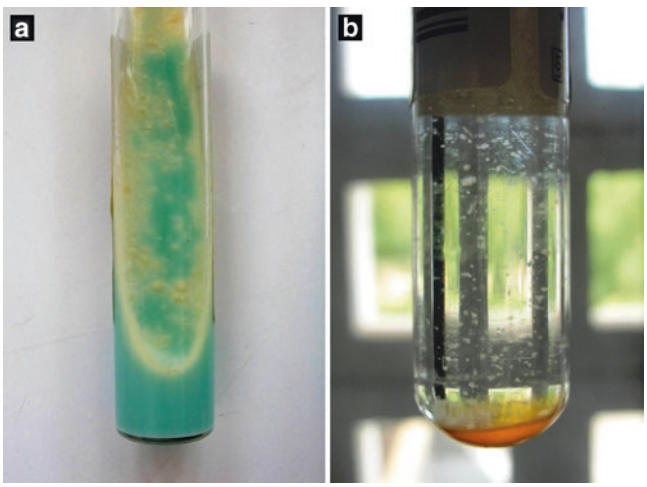



**Fig. 2.19** Growth of colonies of *Mycobacterium tuberculosis* on Lowenstein–Jensen medium (**a**). Macroscopic appearance of mycobacterial growth on MGIT (**b**)

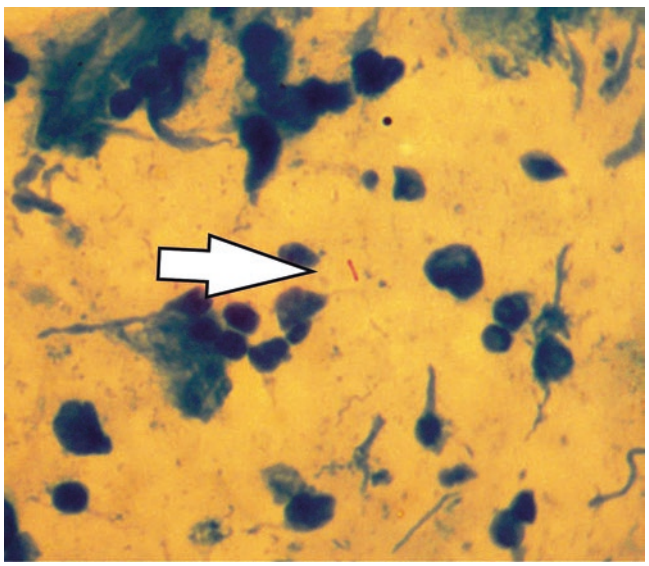


**Fig. 2.20** Positive acid-fast bacillus (AFB) (*arrow*) following Ziehl–Neelsen staining from a bone tuberculosis

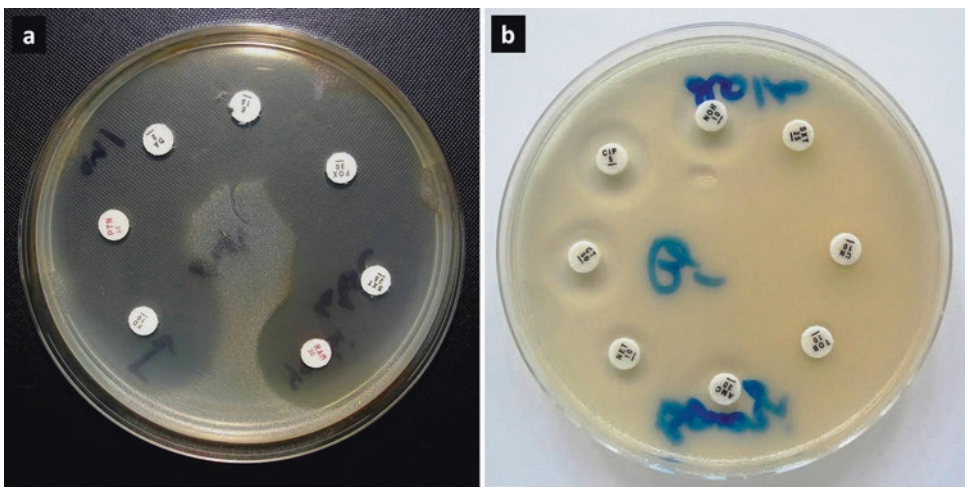



**Fig. 2.21** Antibiogram of *Staphylococcus aureus* on Mueller–Hinton agar (antibiotic sensitivity test). Areas of inhibited bacterial growth are seen around antibiotic discs. Resistance is demonstrated by no zones (or smaller zones) around the antibiotic discs. Good susceptibility of bacteria to antibiotics (**a**); drug resistance (**b**)

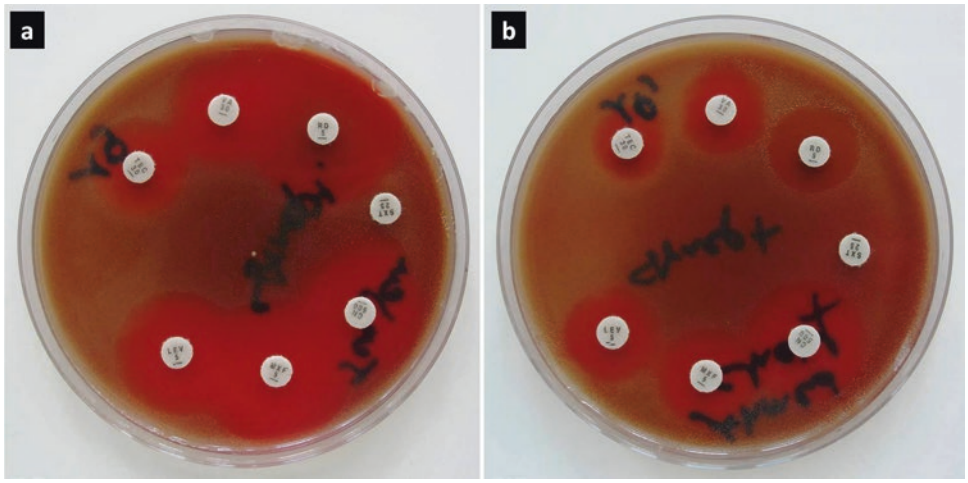



**Fig. 2.22** Sensitivity testing plate of *alpha-hemolytic streptococci* (antibiogram). Susceptibility is demonstrated by a clear zone around each antibiotic disc. Good susceptibility of bacteria to antibiotics (**a**); drug resistance (**b**)

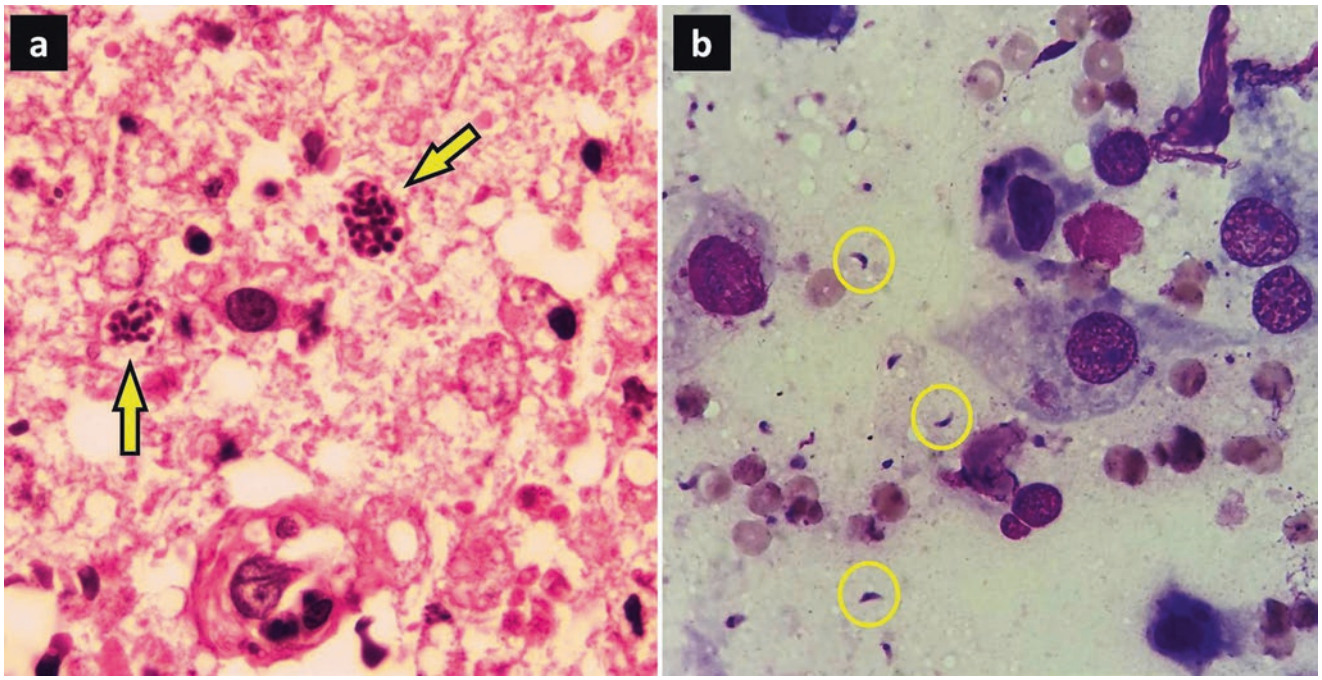



**Fig. 2.23** Toxoplasmosis obtained from a brain granuloma. Microscopy findings of *Toxoplasma gondii*. (**a**) Histopathologic features of toxoplasma encephalitis. A few toxoplasma cysts are noted (*arrows*) in this necrotic brain lesion shown at medium-power magnification (hematoxylin–eosin staining). (**b**) *Toxoplasma gondii* tachyzoites (*circles*) stained with May–Grünwald Giemsa (MGG). Medium-power magnification (Courtesy of Pr. Moutaj R. PharmD, Marrakech, Morocco)

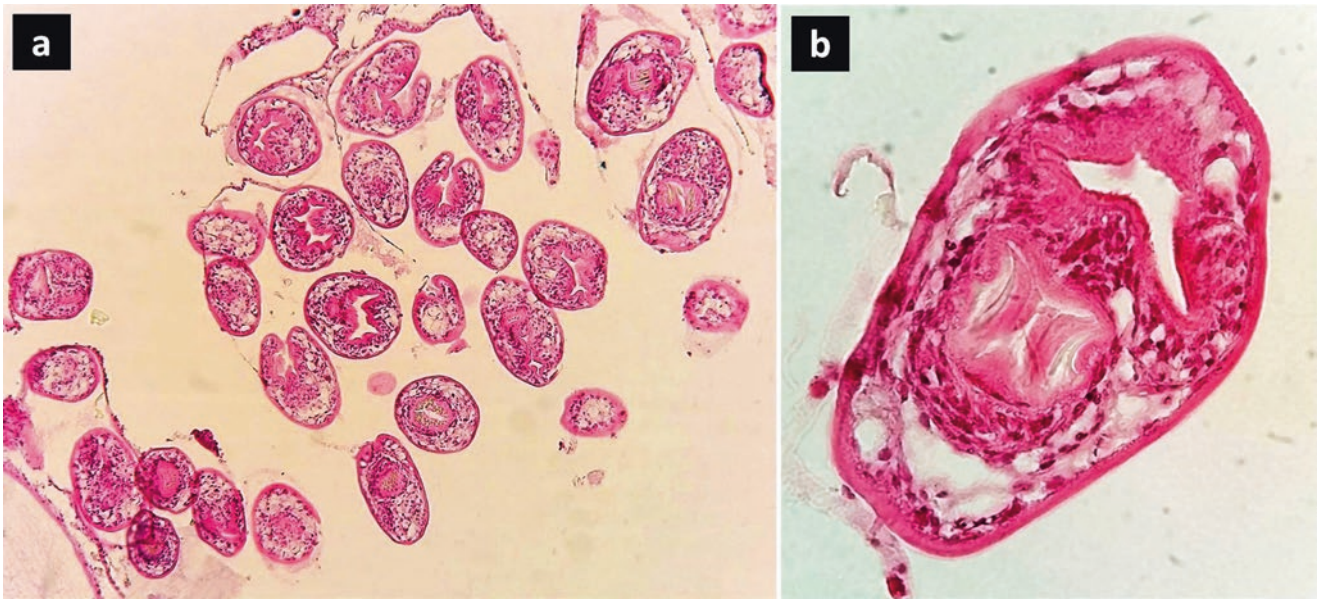



**Fig. 2.24** Hydatid disease. Various protoscoleces of *Echinococcus granulosus* (hematoxylin–eosin staining), shown at medium-power (**a**) and high-power magnification (**b**) (Courtesy of Pr. Moutaj R. PharmD)

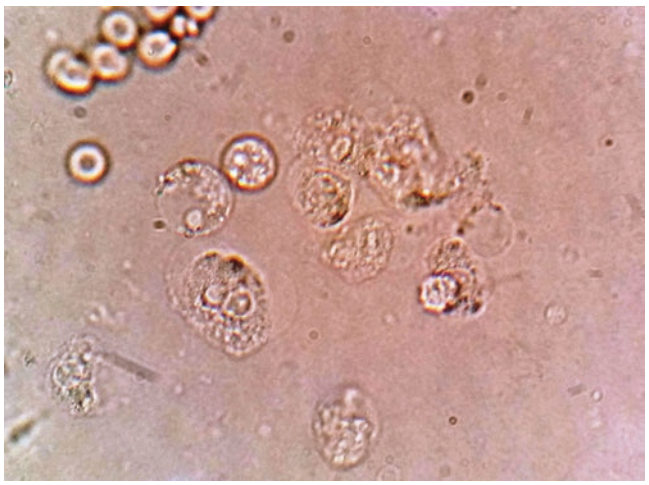


**Fig. 2.25** Amebiasis. *Entamoeba histolytica*, hematophagous forms on optic microscope (Courtesy of Pr. Moutaj R. PharmD)

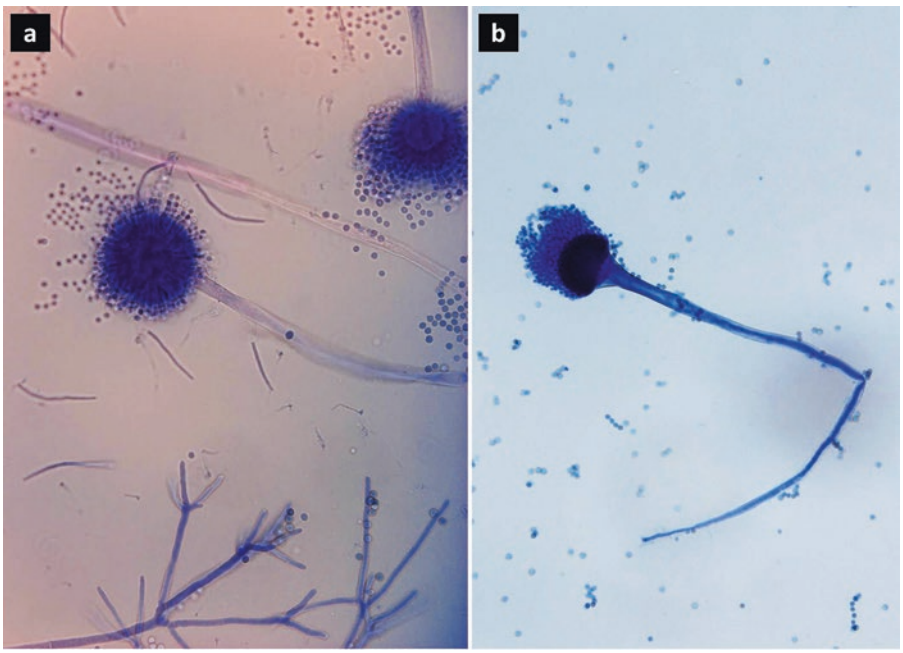



**Fig. 2.26** Aspergillosis. *Aspergillus flavus* (**a**) and *Aspergillus fumigatus* (**b**) on optic microscope (lactophenol cotton blue staining) (Courtesy of Pr. Moutaj R. PharmD)

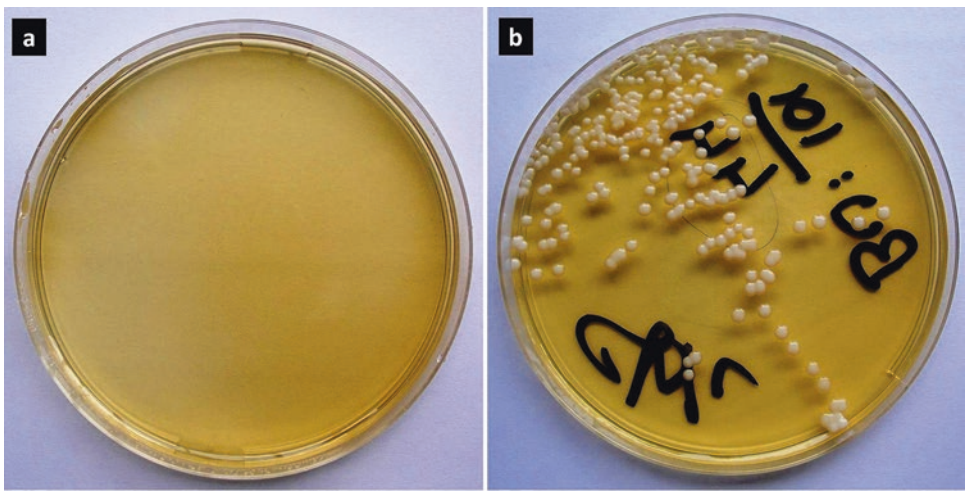



**Fig. 2.27** Candidiasis. Sabouraud agar plate without (**a**) and with (**b**) colonies of *Candida albicans*. (*Courtesy of* Pr. Moutaj R. PharmD)

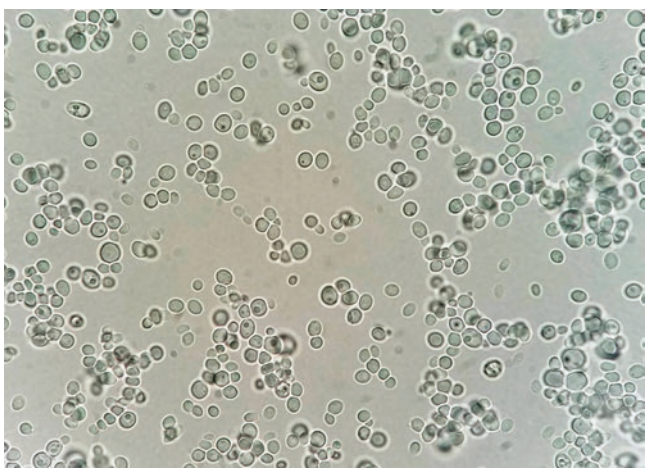


**Fig. 2.28** Candidiasis. Spores of *Candida albicans* on optic microscope (*Courtesy of* Pr. Moutaj R. PharmD)

**Fig. 2.29** Cryptococcosis. *Cryptococcus neoformans* from cerebrospinal fluid specimen on optic microscope using a China ink staining preparation (*Courtesy of* Pr. Moutaj R. PharmD)

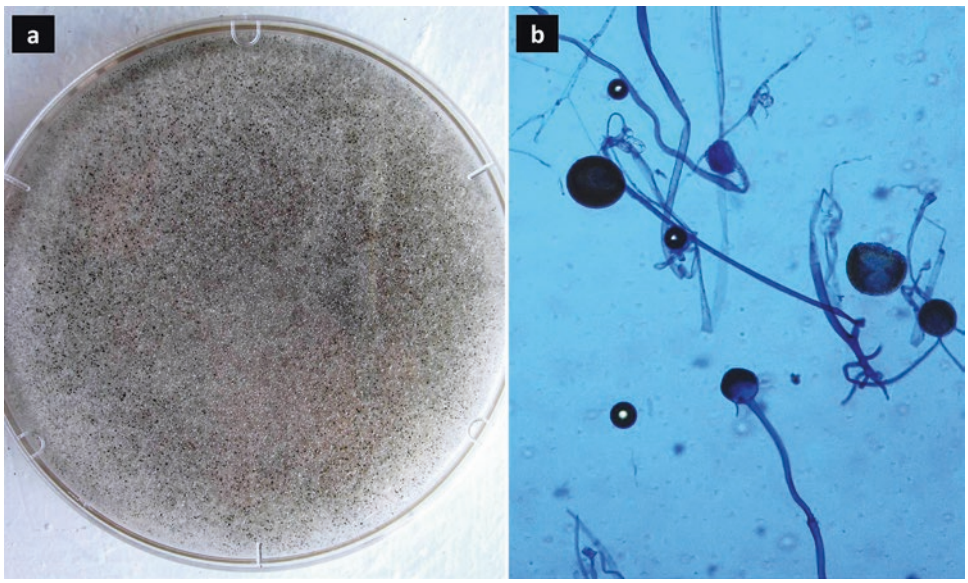



**Fig. 2.30** Mucormycosis. Morphological features of *Rhizopus oryzae* grown on Sabouraud agar: a gray hairy colony (**a**). Microscopic features of *Rhizopus oryzae* using lactophenol cotton blue staining (**b**) (Courtesy of Pr. Moutaj R. PharmD)

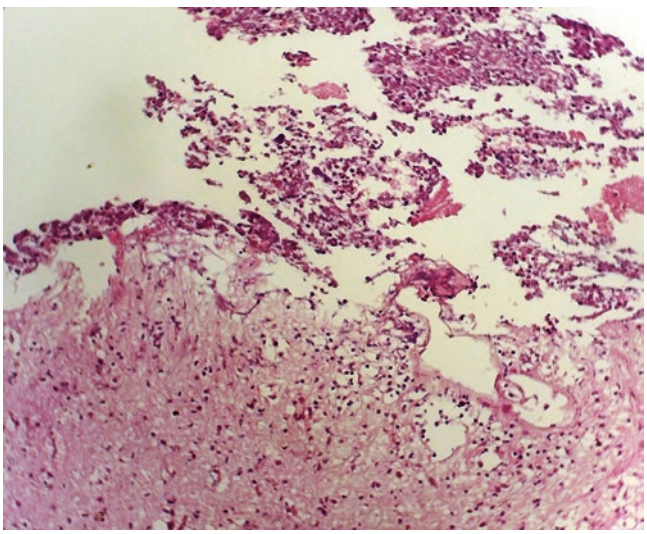


**Fig. 2.31** Brain abscess. Photomicrograph of specimen showing area of pus and necrotic debris (*top*) surrounded by inflammatory infiltrates (encephalitis) (*below*) (hematoxylin–eosin staining at medium magnification)

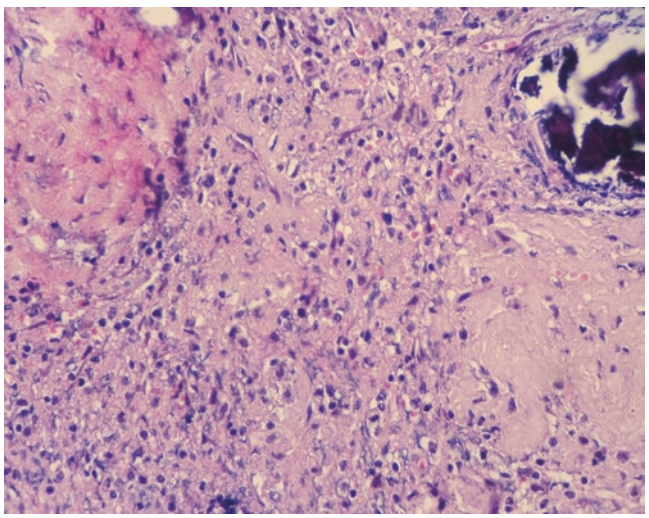
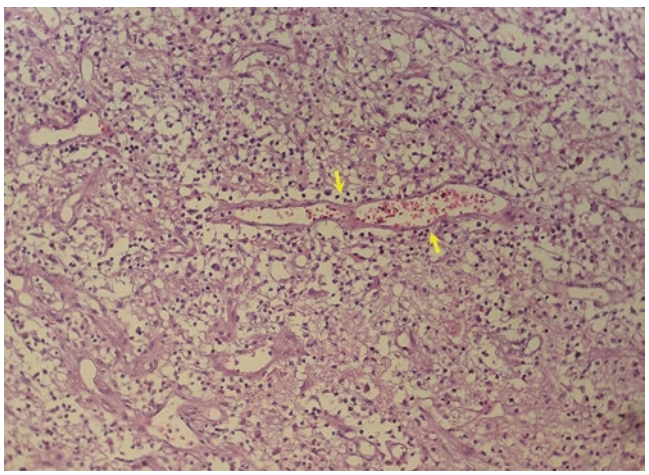


**Fig. 2.33** Histopathologic features of subacute form of spinal osteomyelitis. Bone necrosis with lymphoplasmocytic inflammatory infiltrates and neutrophilic granulocytes. High-power magnification (hematoxylin–eosin staining)

**Fig. 2.32** Brain abscess. Histopathologic features of subacute encephalitis with vasculitis (*arrows*). Medium-power magnification (hematoxylin–eosin staining)



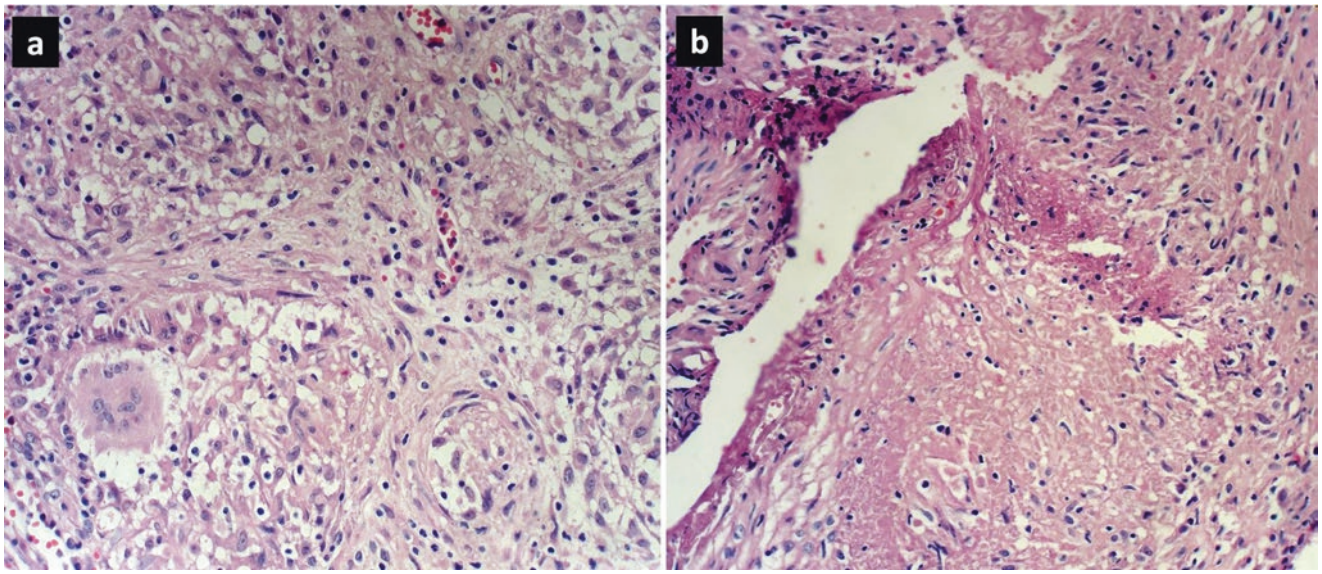



**Fig. 2.34** Histopathologic features of brain tuberculoma. Presence of epithelioid–giant cell granulomas with mononuclear cell infiltration (**a**) and caseous necrotic material (**b**) (hematoxylin–eosin staining)

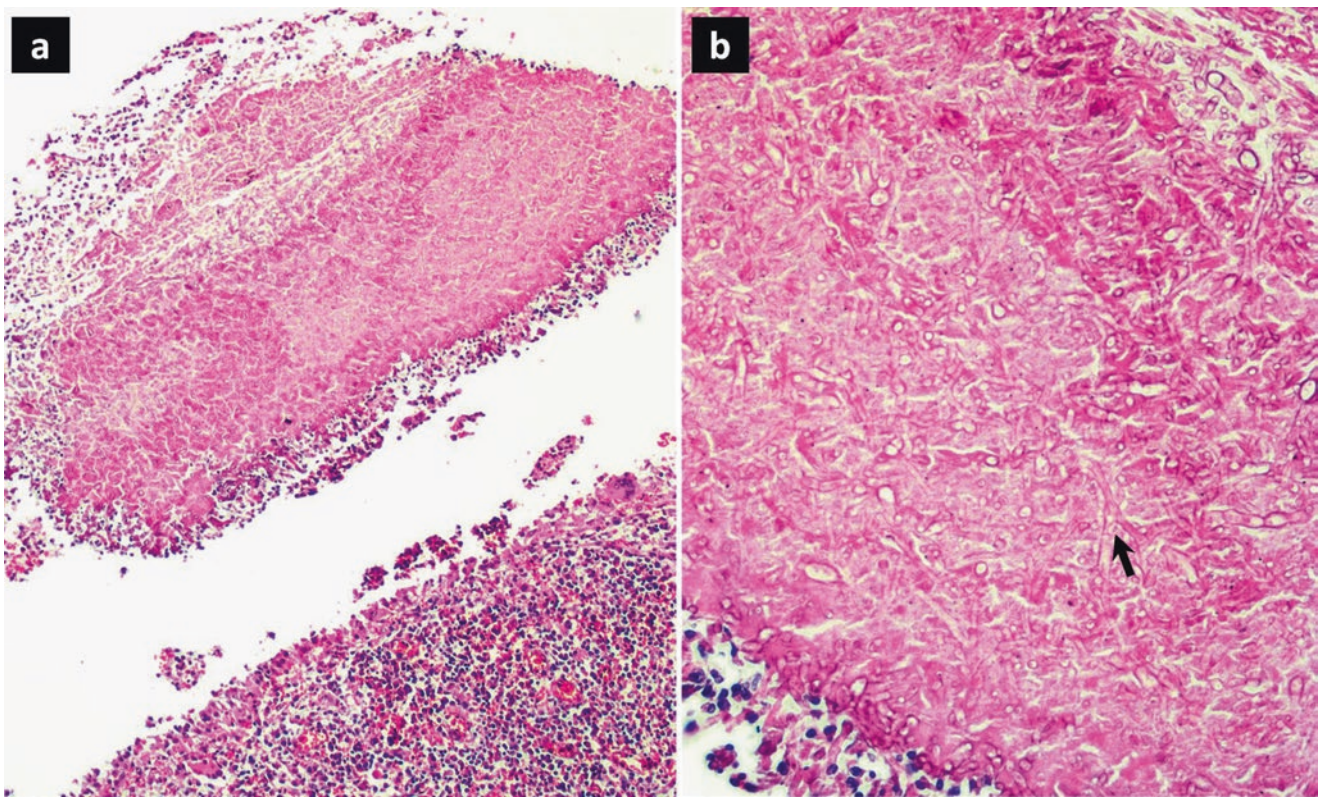



**Fig. 2.35** Histopathologic features of aspergillosis. (**a**) Photomicrograph of specimen showing the necrotic debris at low magnification. (**b**) Presence of septation and dichotomous branching at approximately 45° angle (*arrow*), consistent with *Aspergillus* species at medium-power magnification (hematoxylin–eosin staining)

## Suggested Reading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# Part II

# Infections of the Brain and Its Coverings

# Scalp Abscesses

# 3

Scalp infections are usually superficial, minor, and limited, but they can extend deeply, inducing serious complications. Tender, fluctuant swelling, erythema, and local heat are the main signs. Sometimes, pus formation and fistula with purulent discharge can be seen. CT scan, MRI, and ultrasound imaging may be used for detection and confirmation when the diagnosis is uncertain or for the evaluation of the severity of infection and its extension (especially concomitant cranial osteomyelitis). Superficial, limited infections may be treated with antibiotics alone. Extensive subgaleal abscess should be drained, followed by appropriate antimicrobial therapy (Case 3.1, Figs. 3.1, 3.2, 3.3, and 3.4). Long-term sequels are rare (especially chronic cutaneous fistula and esthetic scar).

## Epidemiology and Etiology

Infections of the scalp may involve any of its five layers (remembered as SCALP: ***s***kin, ***c***onnective tissue, ***a***poneurosis, ***l***oose connective tissue, and ***p***ericranium). Most infections are superficial, minor, and limited, as erysipelas, non-necrotizing cellulitis, impetigo, furuncles, folliculitis, and local scalp abscess, but they can extend and induce more serious complications such as necrotizing fasciitis and cranial osteomyelitis.

Scalp abscesses are uncommon purulent infectious collections involving principally the subgaleal space (under the aponeurosis or epicranium). Subgaleal abscesses may be spontaneous, owing to contiguous spread (usually from a paranasal sinus infection to a chronic scalp lesion). Hematogenous infection is rare. Acquired or iatrogenic subgaleal scalp abscess is more common, following trauma or surgery (including pin placements, fetal scalp monitors, cosmetic surgery, and craniotomy). (*See* Chap. 21, "Surgical Site Infections in Cranial Surgery.")

## Clinical Presentations

Tender, fluctuant swelling, erythema, and local heat are the main signs. Sometimes, pus formation, purulent fistula, and discharge with a foul odor can be seen (Figs. 3.5, 3.6, 3.7, and 3.8). Tenderness out of proportion to soft tissue findings suggests osteomyelitis of the skull rather than scalp abscess. (*See* Chap. 4, "Cranial Osteomyelitis.")

General and/or regional signs of infection may also be encountered, as fever, chills, fatigue, irritability, headache, and lymphadenopathy.

Clinical symptoms may be more serious (meningitis and neurologic complications) if the infection progresses into deep anatomic structures such as the cranial bone and intracranial space.

## Imaging Features

Plain radiography may show swollen extracranial tissue and associated cranial osteomyelitis.

CT scan and MRI may be used for detection and confirmation when the diagnosis is uncertain or for evaluation of the severity of infection and its extension (Figs. 3.9, 3.10, and 3.11). The appearance is that of a fluid collection with peripheral enhancement. CT scans generally show a homogenous, low-density mass with rim enhancement following contrast injection. MRI typically demonstrates a cystic mass with homogenous, low-intensity, or isointense signal on



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_3

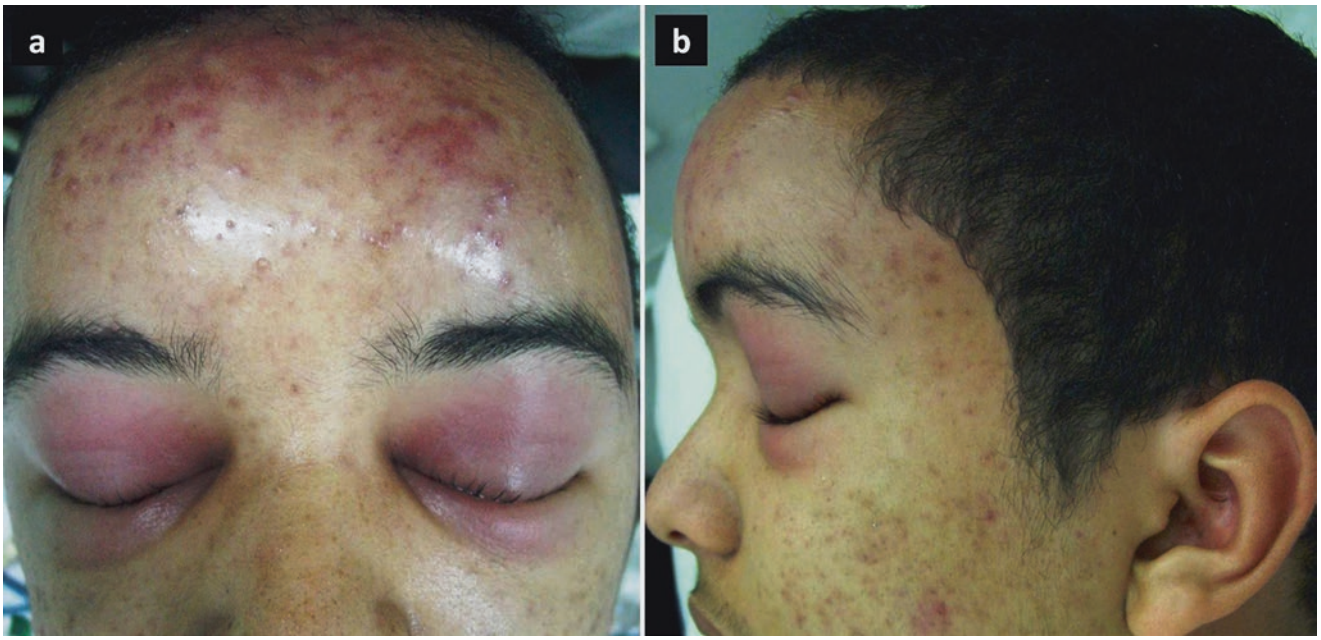


**Fig. 3.1** Case 3.1. Bifrontal tender, fluctuant swelling, facial erythema, and periorbital cellulitis in a 15-year-old boy previously treated for paranasal sinusitis. Frontal view (**a**) and lateral view (**b**)

T1-weighted images and high-intensity signals on T2-weighted images, with rim enhancement after gadolinium administration.

Ultrasound detection can help in excluding other soft tissue collections and masses. The ultrasonographic appearance is that of a heterogeneous, hypoechoic lesion contiguous to the cranial vault, occasionally with a vascular periphery.

## Laboratory Findings

An increased peripheral white blood cell count is not required for diagnosis, and inflammatory biomarkers (erythrocyte sedimentation rate and C-reactive protein level) are highly variable in their expression. The procalcitonin level is more specific and helpful in follow-up, but it lacks sensitivity.

The most common pathogens are gram-positive skin flora, especially *Staphylococcus* species (*S. aureus*) and *Streptococcus* species *(S. epidermidis).* However, gram-negative bacilli are not rare (especially *E. coli* in neonates). Polymicrobial infections may also occur, with the presence of anaerobes.

## Treatment Options

Infectious lesions confined to the skin are usually treated by dermatologists and plastic surgeons. Superficial, limited infections may be treated with antibiotics alone.

Subgaleal abscess should be drained (reopen the wound for posttraumatic suppuration). Obtain intraoperative cultures of the collection, remove the necrotic tissues, clean the region with antiseptic solutions, put in a drain, and close the wound whenever possible. Parenteral antimicrobial therapy is administered for 1–3 weeks, followed by suitable oral therapy.

Tubercular and fungal abscesses must be treated with the appropriate anti-infectious regimens.

## Outcomes

Close clinical monitoring and inspection of the infected region are important to guarantee good response to treatment. Complete clinical recovery is expected for most patients. The outcome is related to the severity of infection (especially cranial bone complications and intracranial extensions) and to the delay in diagnosis. Long-term sequels (especially chronic cutaneous fistula, local discomfort, or facial scar) are rare.

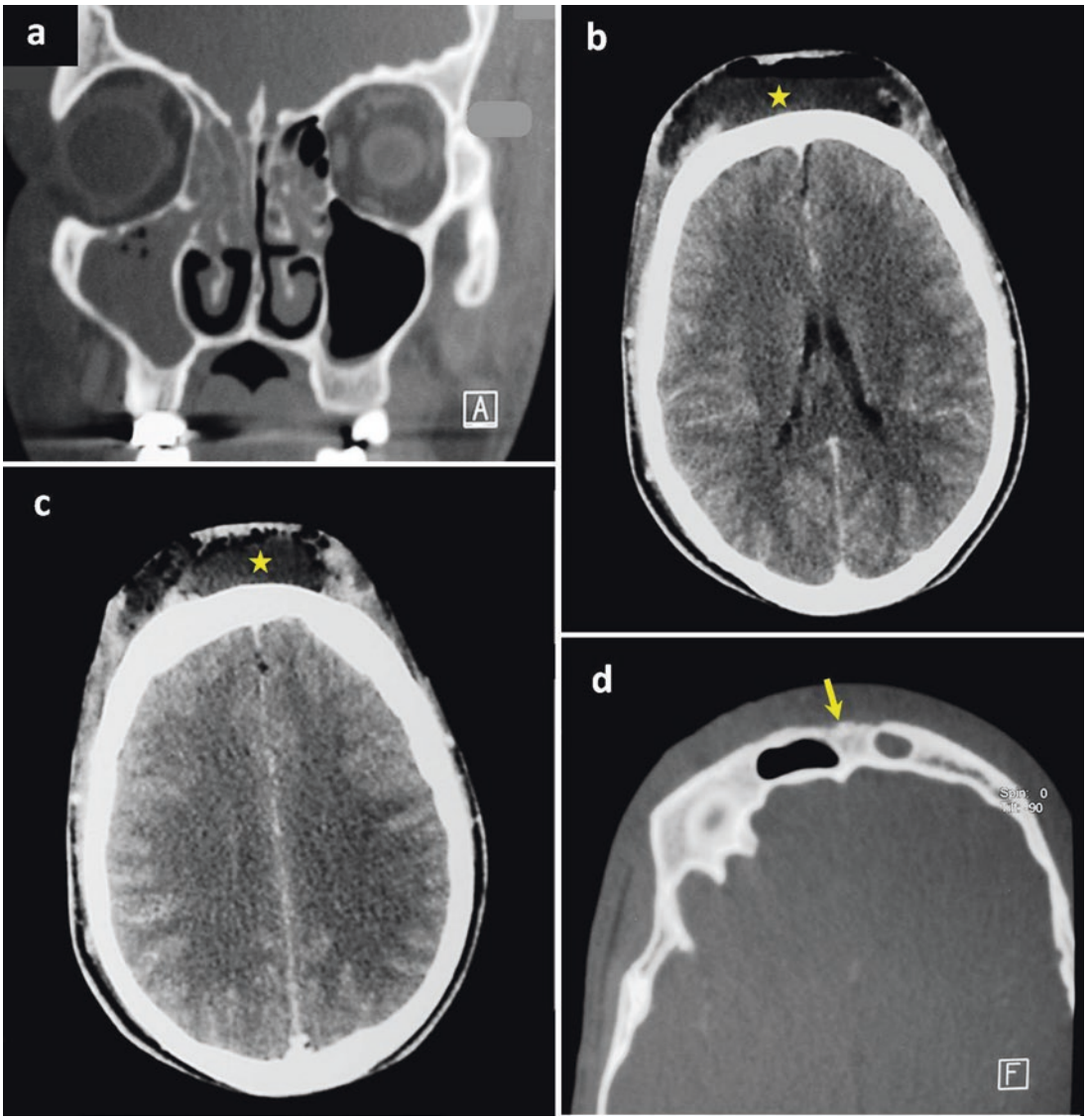


**Fig. 3.2** Case 3.1. (**a**) In the same patient, a coronal maxillofacial CT scan shows right paranasal pansinusitis. (**b**, **c**) Axial cranial CT scan after contrast administration, revealing a frontal subgaleal abscess (*star*) with bubbles of gas showing the presence of gas-forming microorganisms. (**d**) Note the erosion of the outer frontal bone table (*arrow*) on bone window CT scan

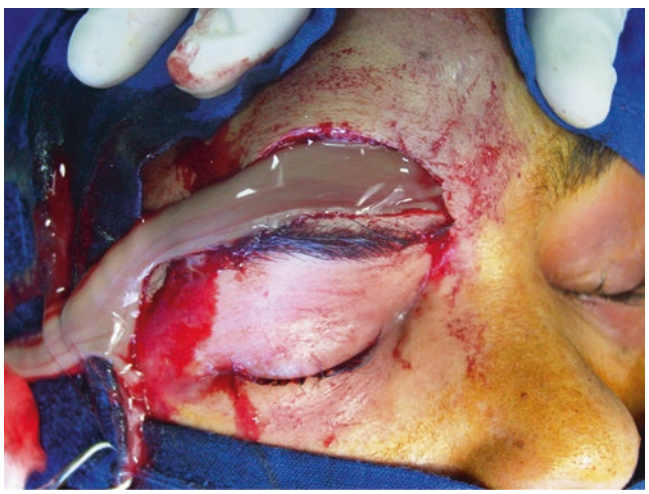

**Fig. 3.3** Case 3.1. Operative view: surgical drainage of the scalp abscess by the ENT team (right eyebrow incision)

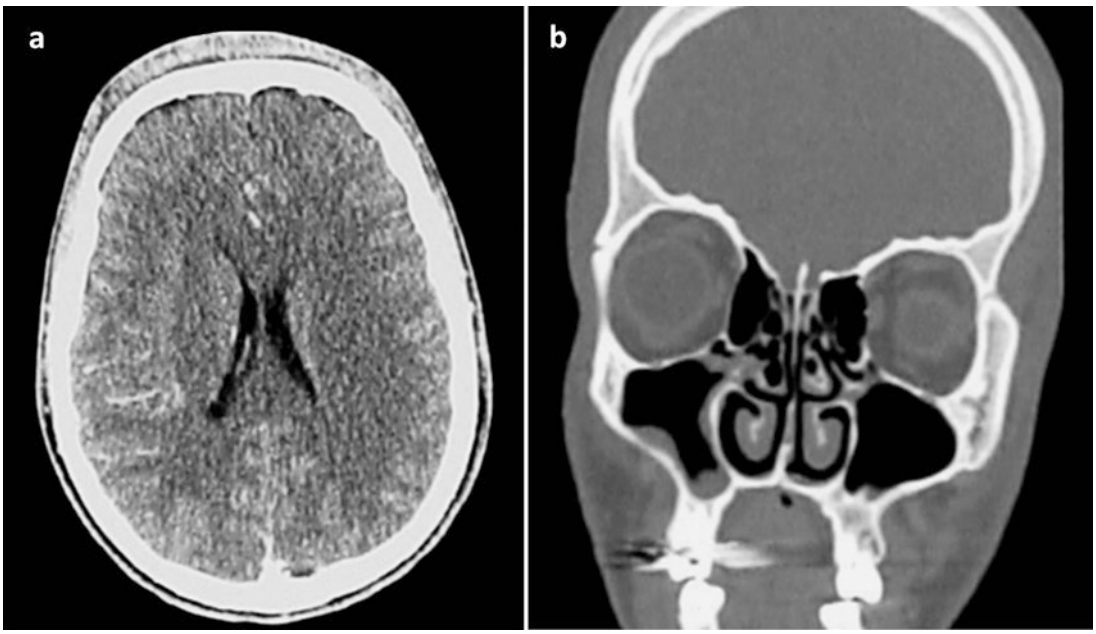


**Fig. 3.4** Case 3.1. Control CT scan 3 weeks later. The subgaleal abscess (**a**) and the paranasal sinusitis (**b**) have almost completely disappeared

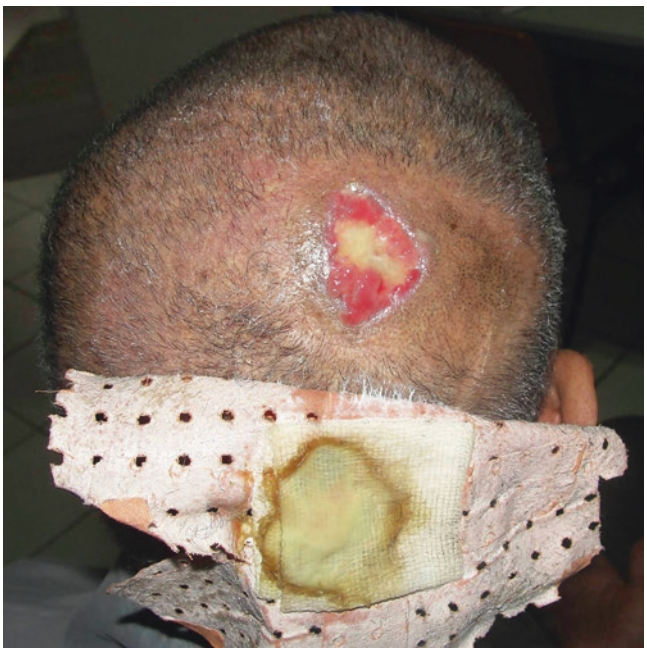

**Fig. 3.5** Infectious, chronic, non-healing scalp pressure ulcer in the right parietal area. Note the purulent dressing

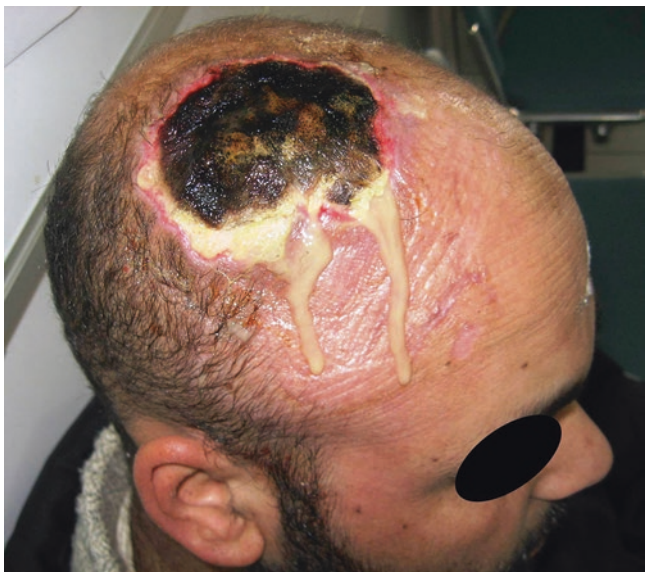

**Fig. 3.6** Posttraumatic scars in the right parietal region of the scalp, with local signs of suppuration

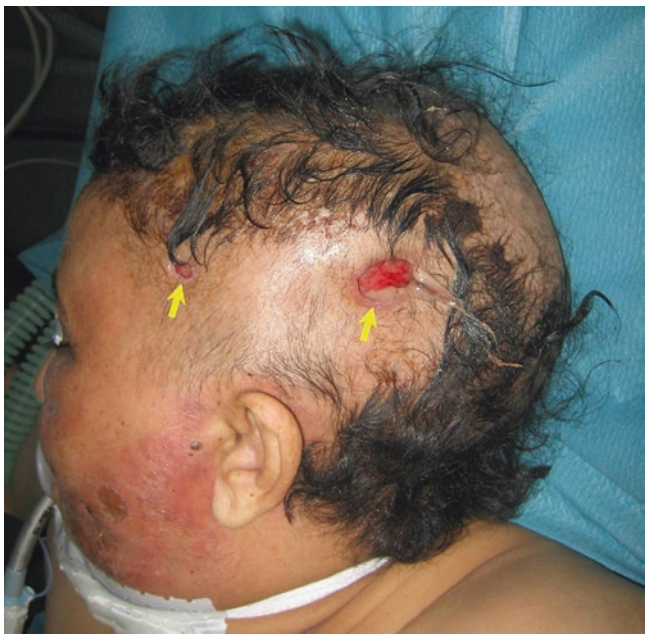

**Fig. 3.7** Two fistulous tracts (*arrows*) and discharge of purulent secretion via the breached scalp. Note the extensive facial cellulitis on the left side (Reproduced from Akhaddar (2016); with permission)

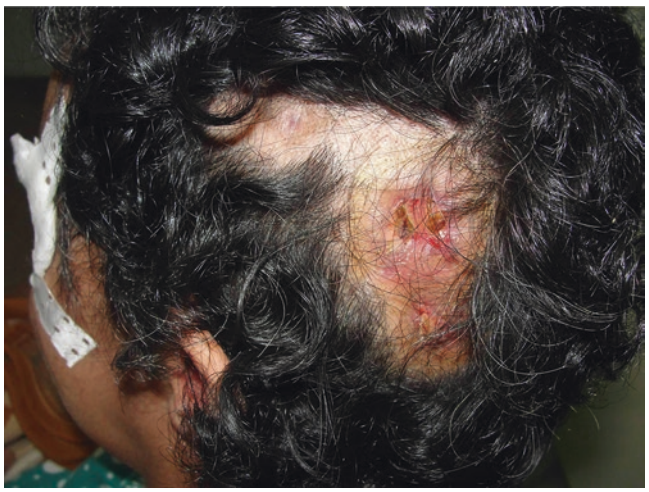

**Fig. 3.8** Chronic, erythematous, non-healing wound with local signs of suppuration located in right parietal area of the scalp

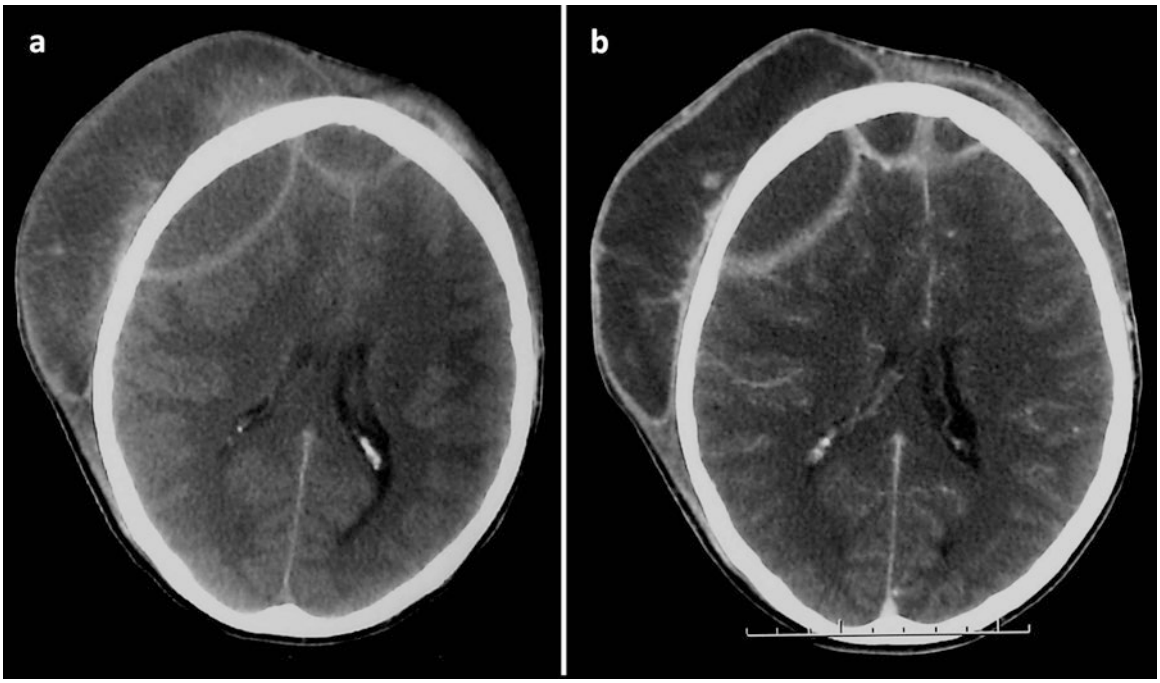


**Fig. 3.9** Axial cranial CT scan before (**a**) and after (**b**) contrast injection. Voluminous frontoparietal subgaleal abscess in a 27-year-old man who had a frontal paranasal sinusitis. Note the extensive intracranial parameningeal suppurations

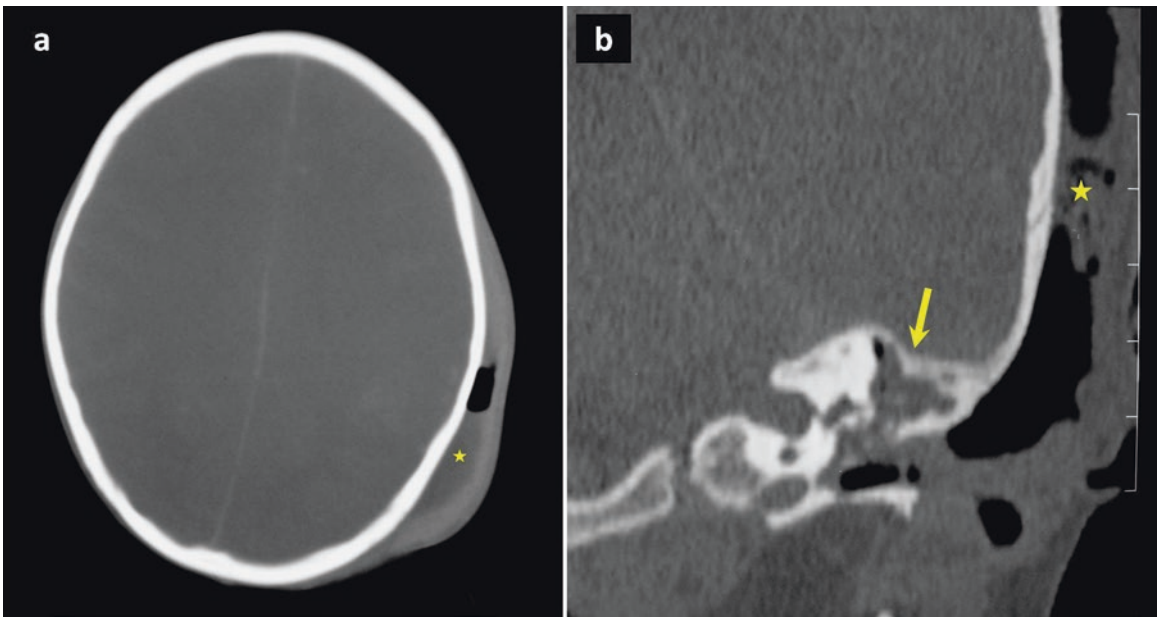


**Fig. 3.10** Axial (**a**) and coronal (**b**) cranial CT scan showing a left fronto-temporoparietal subgaleal abscess (*stars*) with ipsilateral latent otitis media (*arrow*). Note the various bubbles of gas within the extracranial abscess formation, showing the existence of gas-forming microorganisms

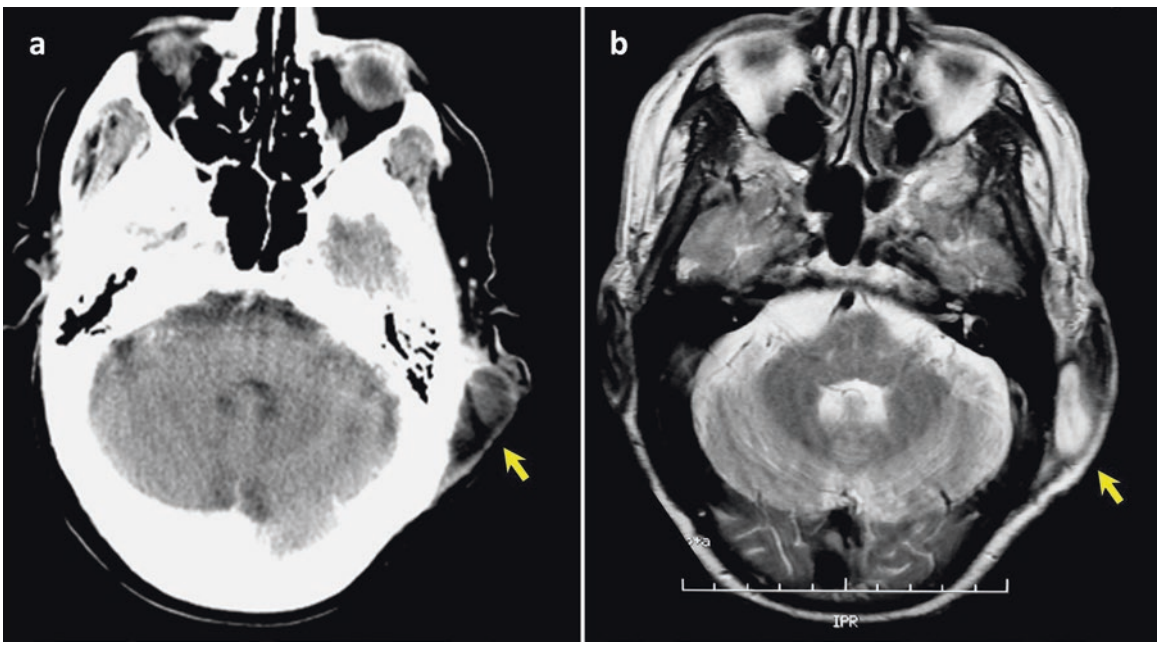


**Fig. 3.11** Axial cranial post-contrast CT scan (**a**) and T2-weighted MRI (**b**) showing an extracranial, retroauricular suppurative collection on the left side (*arrows*)

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# Cranial Osteomyelitis

# 4

Cranial osteomyelitis is an uncommon osseous infection with many different etiologies and various clinical presentations. The classic clinical picture is local, with or without general signs of infection. Seizures, signs of raised intracranial pressure, and local neurologic deficits are related to intracranial extension. Biologic parameters may be elevated (unspecific), but microbiologic and histopathologic examinations of the bone remain the gold standard for the diagnosis. Imaging studies are important tools in the initial diagnosis and in the monitoring of treatment results. Most diagnoses are delayed, needing a combination of surgical debridement, correction of the primary source of infection, and long-term use of antimicrobial agents. Complete clinical recovery is predictable for most patients. The outcome is related to the severity of infection (especially intracranial complications and skull base osteomyelitis) and to the delay in diagnosis.

## Epidemiology and Etiology

Osteomyelitis is an inflammatory condition of the bone (osteitis) and bone marrow (myelitis), which is mainly caused by pyogenic bacteria. Osteomyelitis usually begins as an acute infection, but it may change into a chronic form. The skull is an uncommon location of bony infection, which is often described in the cranial vault.

Traditionally, the disease is usually related to three main sources of infection:

- Direct extension from a contiguous site of infection
- Postsurgical or posttraumatic direct inoculation
- Hematogenous dissemination from a remote source of infection

In developed countries, postsurgical craniotomy infections remain the most common cause of cranial osteomyelitis (*see* Chap. 21), whereas in developing nations, paranasal sinusitis and scalp infections have become the predominant sources.

Classically, the neurocranium has significant proximity with extracranial structures and intracranial spaces, which largely explain the location of infection and its extension (3SO) (Fig. 4.1).

## Clinical Presentations

The classic clinical picture of cranial osteomyelitis is local with or without general signs of infection. Local signs of infection are swelling, pus formation, purulent fistula, and discharge with a foul odor. The cranial bone may be exposed. General signs of infection are represented by fever, chills, fatigue, lethargy, irritability, headache, and lymphadenopathy. Meningitis and neurologic syndromes should alert for intracranial complications.

The duration of symptoms is typically several months in chronic infections. If the duration is less than 1 month, the infection is considered acute.

## Imaging Features

CT scan findings in the acute stage of cranial osteomyelitis consist of an area of rarefaction and loss of diploic bone trabeculae; demineralization, erosion, or thinning of the cortical bone table; and extracranial and subperiosteal abscesses. In chronic form, diploic bone sclerosis and cortical bone thickening may be mixed with areas of radiolucency and interruption of cortical bone. Sequester formations are combined, with more or less extensive destruction of the skull tables.

MRI is useful in demonstrating intracranial extension, especially epidural abscess and/or subdural empyema. In the acute stage of osteomyelitis, MRI findings consist of replacement of diploic bone fat by inflammation, enlargement of diploic space and thinning of skull tables, a T2 increase in signal intensity, and T1 contrast enhancement. In chronic form, there is dissolution of cortical bone fragments with



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_4

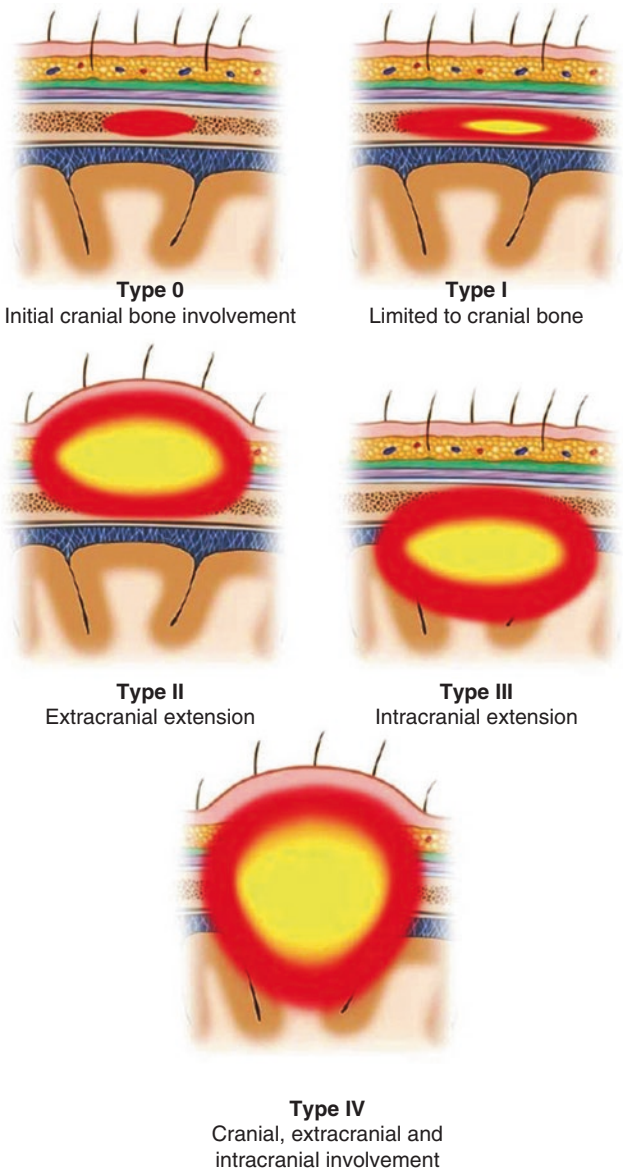


**Fig. 4.1** Staging system for skull osteomyelitis (3SO). Five main types of cranial osteomyelitis based on the location of infection and its extension (Reproduced from Akhaddar (2016); with permission)

sequester formation and soft tissue/dural gadolinium enhancement (Figs. 4.2, 4.3, 4.4, 4.5, 4.6, 4.7, and 4.8).

Bone scans using technetium-99m, gallium-67 citrate, single-photon emission computed tomography (SPECT), positron emission tomography (PET), or fluorodeoxyglucose PET (FDG-PET) are very sensitive for detecting cranial bone infection (Figs. 4.9, 4.10, 4.11, 4.12, 4.13, and 4.14). Ultrasonographic imaging of swelling may help in detecting subgaleal abscess.

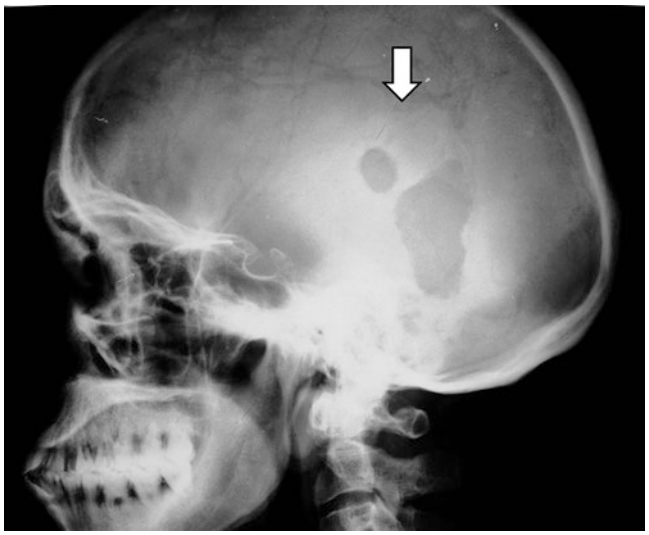

**Fig. 4.2** Lateral skull plain radiography showing temporoparietal osteolytic lesions surrounded by sclerosis (Reproduced from Akhaddar (2016); with permission)

## Laboratory Findings

Some biologic parameters such as blood leukocyte level, erythrocyte sedimentation rate, and C-reactive protein may be elevated in cases of cranial osteomyelitis, but these findings are inconstant and unspecific. Microbiologic and histopathologic examinations of the bone remain the gold standard for the diagnosis.

Blood cultures (preferably taken during a fever spike) may be helpful, especially if hematogenous spread is suspected.

Various microorganisms (bacteria, fungi, and parasites) may be responsible for the infection, particularly aerobic and anaerobic bacteria. The most common individual pathogens are *Streptococcus* species, *Staphylococcus* species, *Bacteroides* species, and *Fusobacterium* species. These microorganisms reflect the bacterial species encountered in paranasal sinus air infection and endogenous skin flora. Polymicrobial infections are not rare.

Histopathologic studies are important tools for the diagnosis (Fig. 4.15). Acute osteomyelitis shows marrow edema, bony spicules, necrosis, and acute inflammatory cells. Chronic forms demonstrate irregular fragments of devitalized bone surrounded by dense fibrous tissue. Histopathology is also helpful for diagnosing some specific osteomyelitis pathogens such as mycobacteria, fungi, and parasites (*see* Chap. 2).

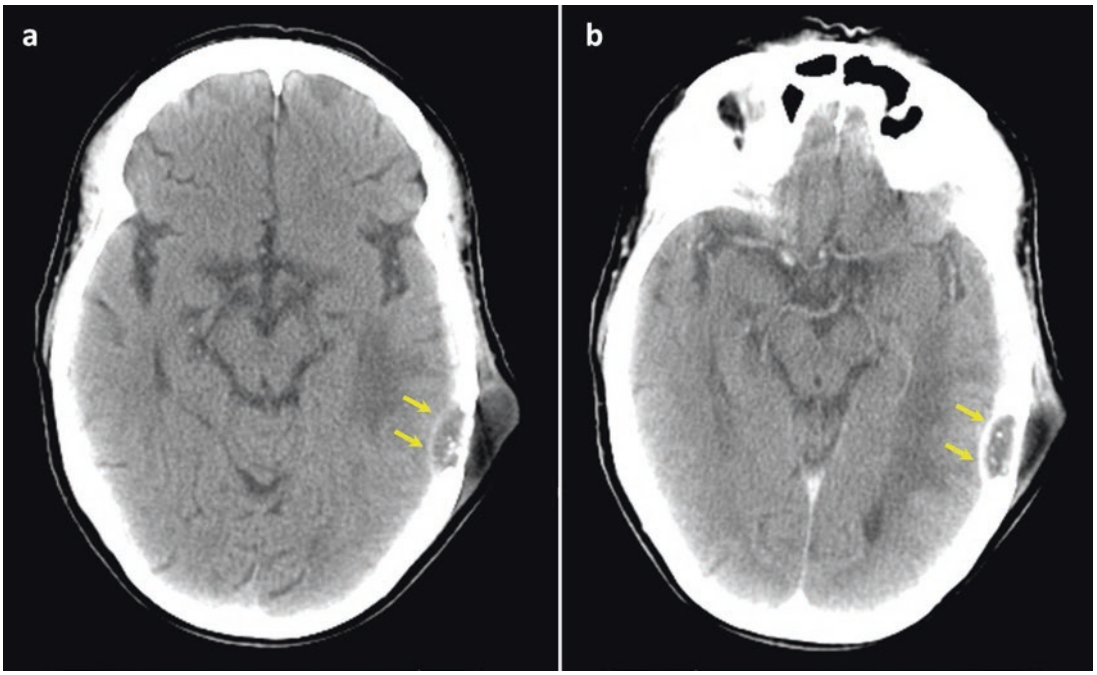


**Fig. 4.3** Axial cranial CT scan before (**a**) and after (**b**) contrast injection showing chronic osteomyelitis with temporoparietal defect (*arrows*). Note some osseous sequestra within the osteolytic lesion (Reproduced from Akhaddar (2016); with permission)

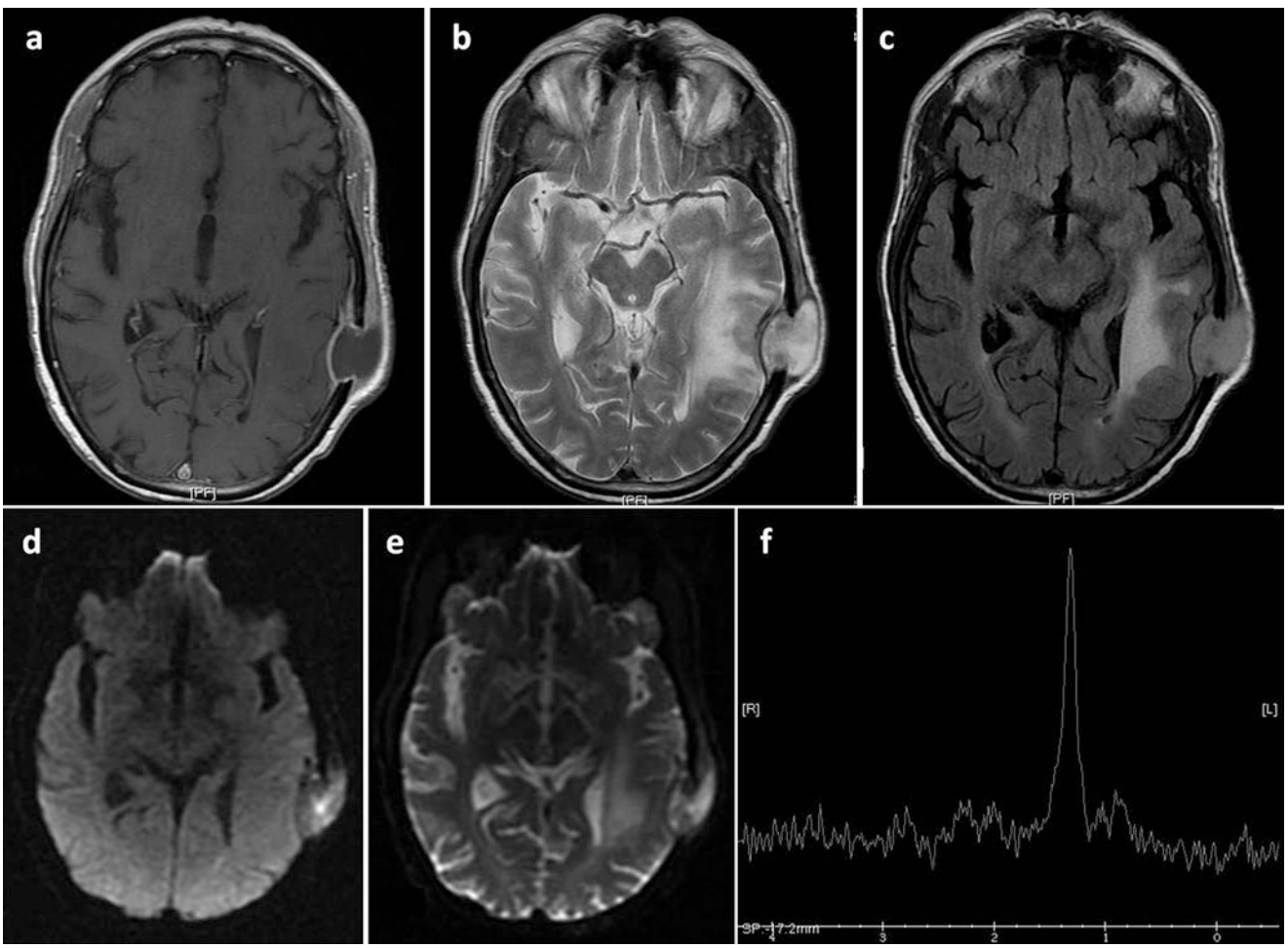


**Fig. 4.4** Chronic temporoparietal osteomyelitis. Axial cranial MRI on T1-weighted image after gadolinium administration (**a**), T2-weighted image (**b**), FLAIR sequence (**c**), diffusion-weighted image (**d**), apparent diffusion coefficient map (**e**), and spectroscopy (**f**) (Reproduced from Akhaddar (2016); with permission)

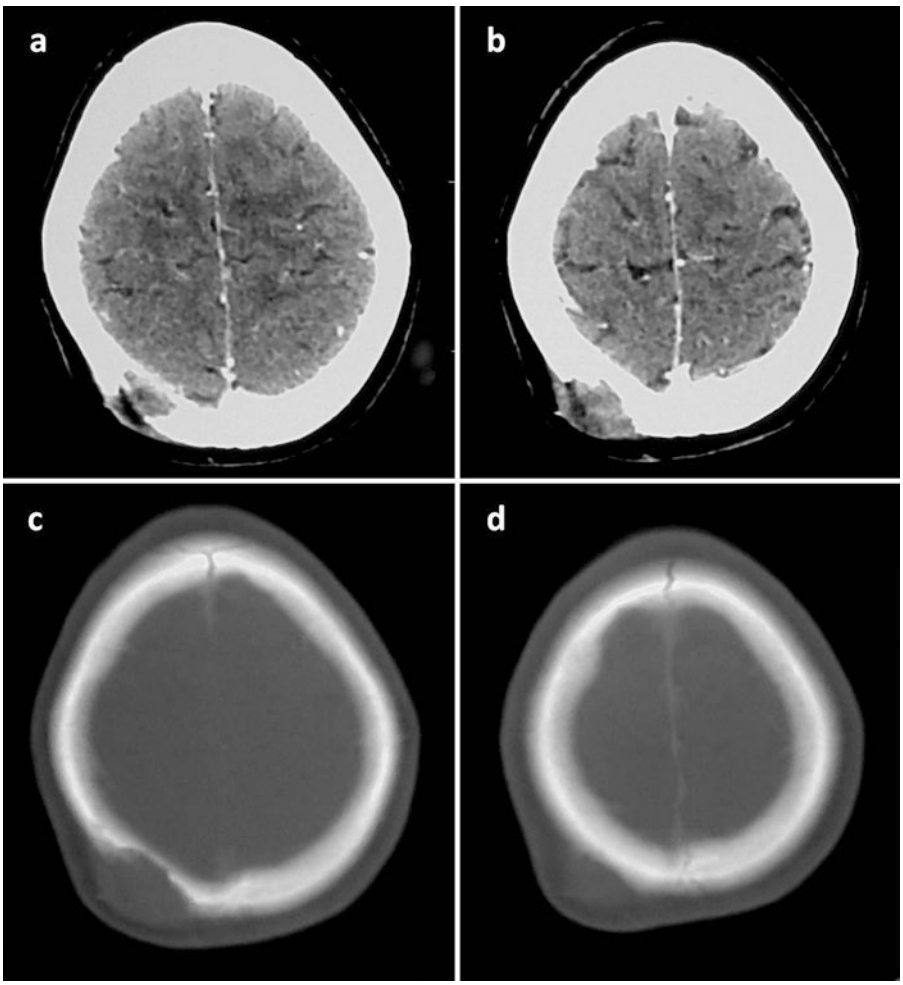


**Fig. 4.5** Axial CT scan of the skull in parenchymal (**a**, **b**) and bone (**c**, **d**) windows, showing hypodense parietal bone lesion with extracranial extension (Reproduced from Akhaddar (2016), p. 262; with permission)

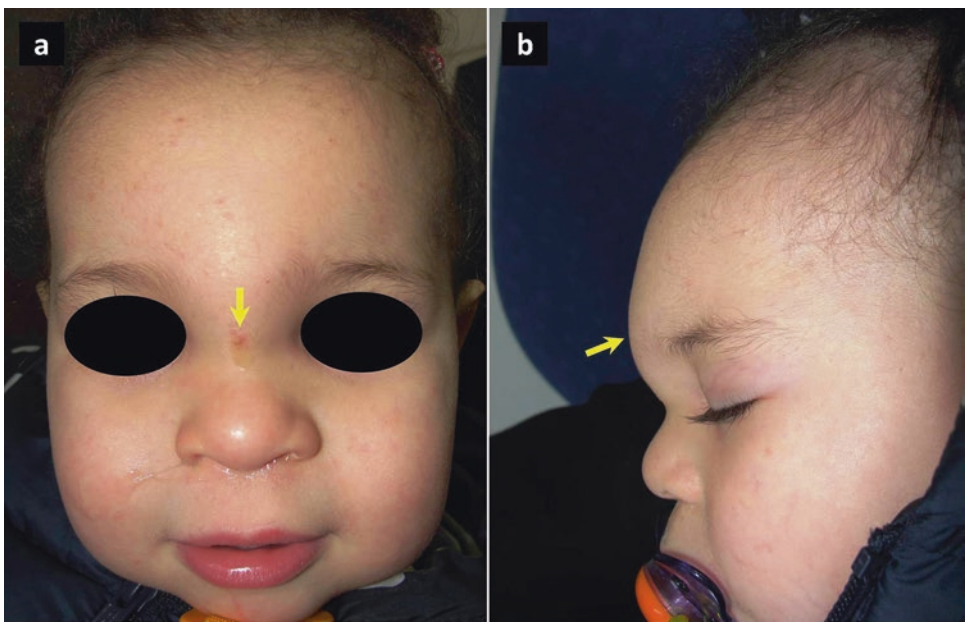


**Fig. 4.6** Case 4.1. Suppurative nasal dermal sinus tract in a 2-year-old girl (*arrow*). Frontal view (**a**) and lateral view (**b**)

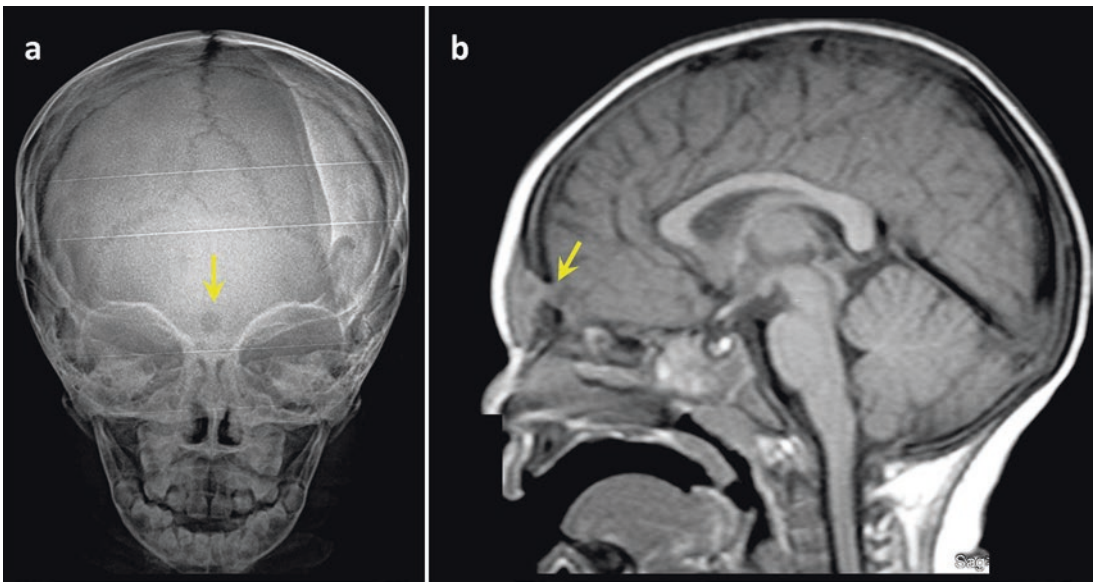


**Fig. 4.7** Case 4.1. Frontal osseous defect with osteomyelitis (*arrow*). Anteroposterior cranial radiography (**a**) and sagittal T1-weighted MRI without gadolinium injection (**b**)

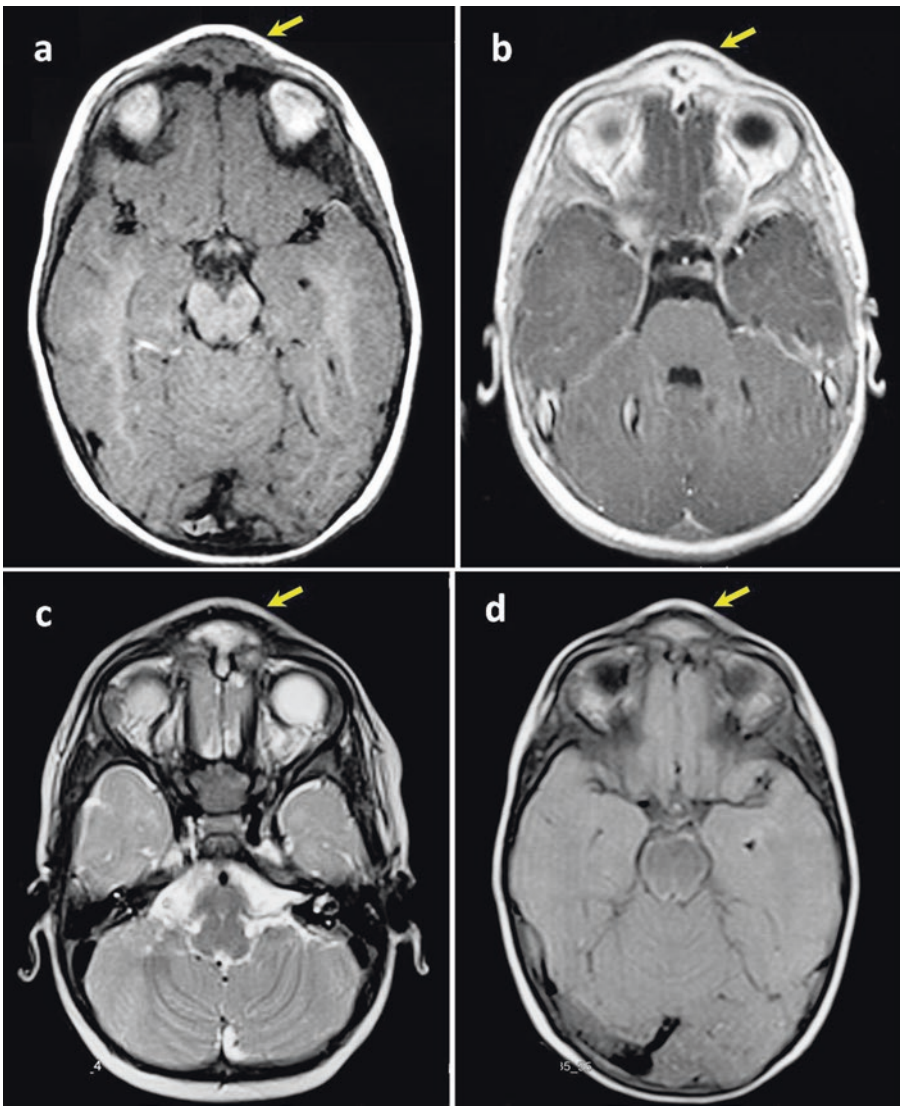


**Fig. 4.8** Case 4.1. Infected nasal dermoid (*arrow*) with frontal osteomyelitis and intracranial extension. Axial T1-weighted MRI before (**a**) and following (**b**) gadolinium injection. Axial T2-weighted MRI (**c**) and FLAIR sequence (**d**). Note the peripheral rim enhancement (**b**)

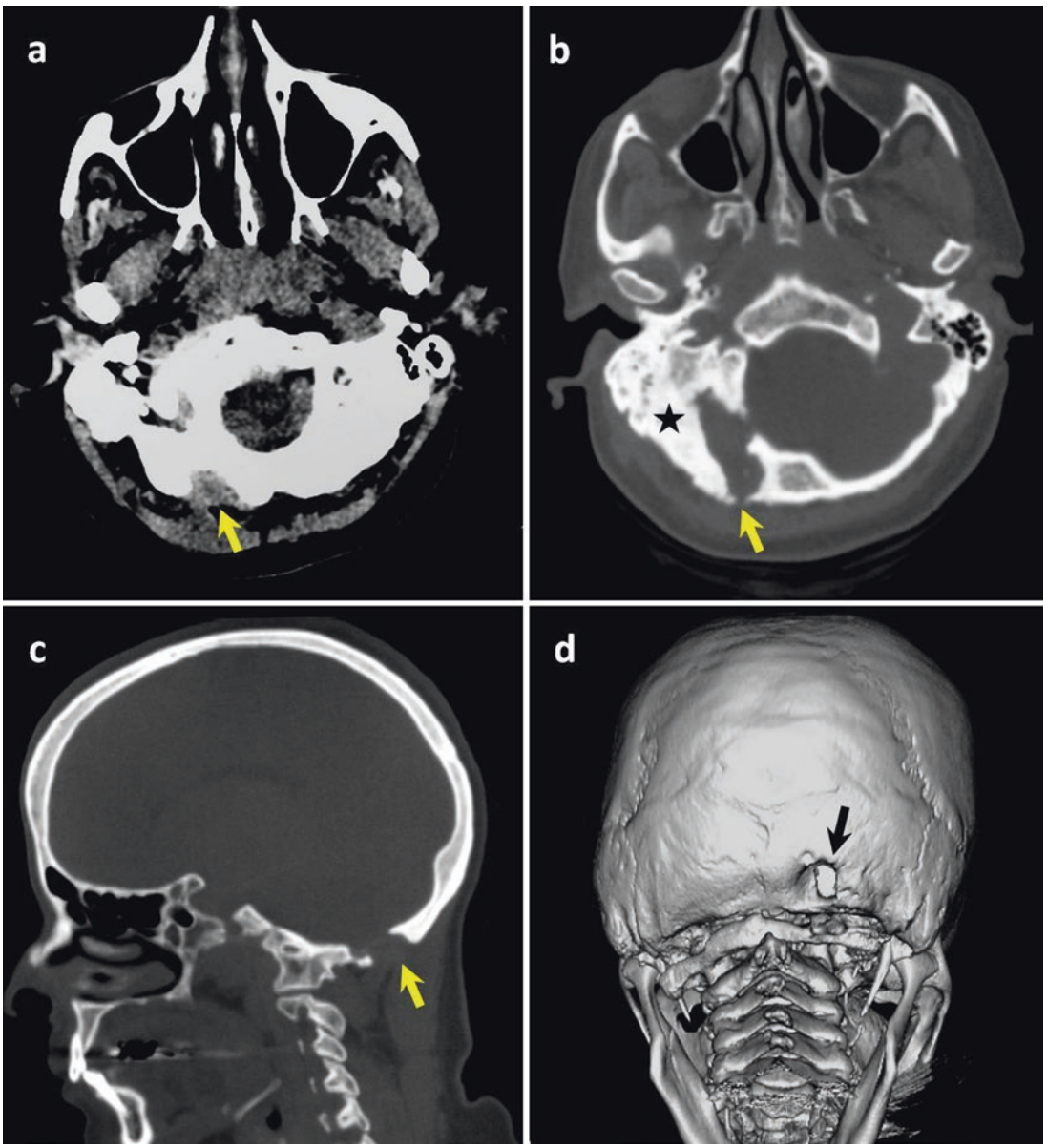


**Fig. 4.9** Case 4.2. Chronic temporo-occipital skull base osteomyelitis in a 43-year-old woman. This patient presented with an intermittent occipital subcutaneous swelling over the past 10 years, with mild headaches but without a history of otitis. Axial cranial CT scan on parenchymal (**a**) and bone (**b**) windows, sagittal view (**c**), and three-dimensional bone reconstruction (reformatted volume rendered) (**d**) showing the occipital osteolytic lesion (*arrow*) with coalescent mastoiditis (*black star*) on the *right side*

## Treatment Options

Antimicrobial therapy is generally concomitant to surgical procedures. Antibiotic therapy should include broad-spectrum antibiotic drugs against aerobic and anaerobic cocci and bacilli, with adequate penetration into the cranial bone, the meninges, and the brain parenchyma. Because of bone infection, antibiotics should be given for at least 8 weeks. Patients with skull base osteomyelitis or brain suppurations may need longer courses.

Generally, the standard empiric antibiotic regimen includes vancomycin, metronidazole, and a third-generation cephalosporin. Antistaphylococcal activity should always be considered. Treatment should be optimized according to the results of the susceptibility testing of the pathogen isolated. Tubercular and fungal infections must be treated with the appropriate anti-infectious regimens.

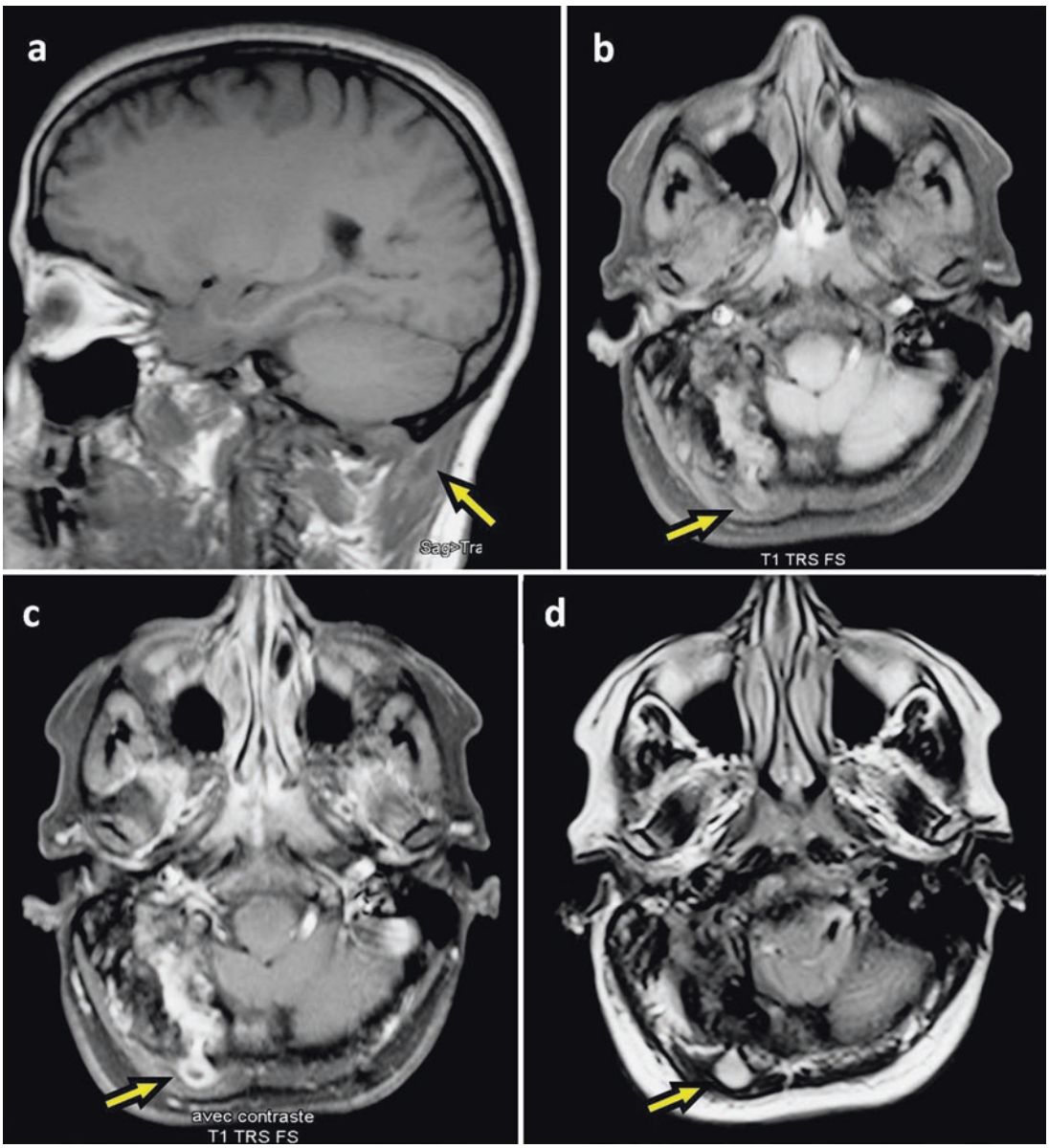


**Fig. 4.10** Case 4.2. Extracranial occipital abscess with intracranial extension and surrounded osteomyelitis. Sagittal (**a**) and axial (**b**) T1-weighted MRI without gadolinium injection. Axial enhanced T1-weighted image (**c**) and diffusion-weighted sequence (**d**) reveal the ring-enhanced extracranial abscess formation (*arrow*) with its intracranial extension through the right occipital bone defect. Note the soft tissue/dural gadolinium enhancement (**c**)

Patients with limited extracranial soft tissue infection and mild cranial osteomyelitis can be treated with antibiotics alone. Sometimes, simple cutaneous drainage or limited scalp incision is sufficient and may be used to collect specimens.

Surgical debridement remains critical to eliminate areas of necrosis and sequester. Infected bone with evidence of necrosis should be removed, followed by cranioplasty on a second intervention. An extradural closed drainage system can be used. Sometimes, a titanium mesh may be used at the time of operation. When needed, a frontal sinus may be cranialized.

Management of associated intracranial suppurative complications should be taken into consideration, as discussed in Chaps. 5, 6, and 8. Treatment of concomitant rhinosinusitis, orodental infection, or otomastoiditis is needed to eliminate the primary source of infection in many patients.

If available, adjuvant hyperbaric oxygen therapy should be considered for the treatment of refractory forms.

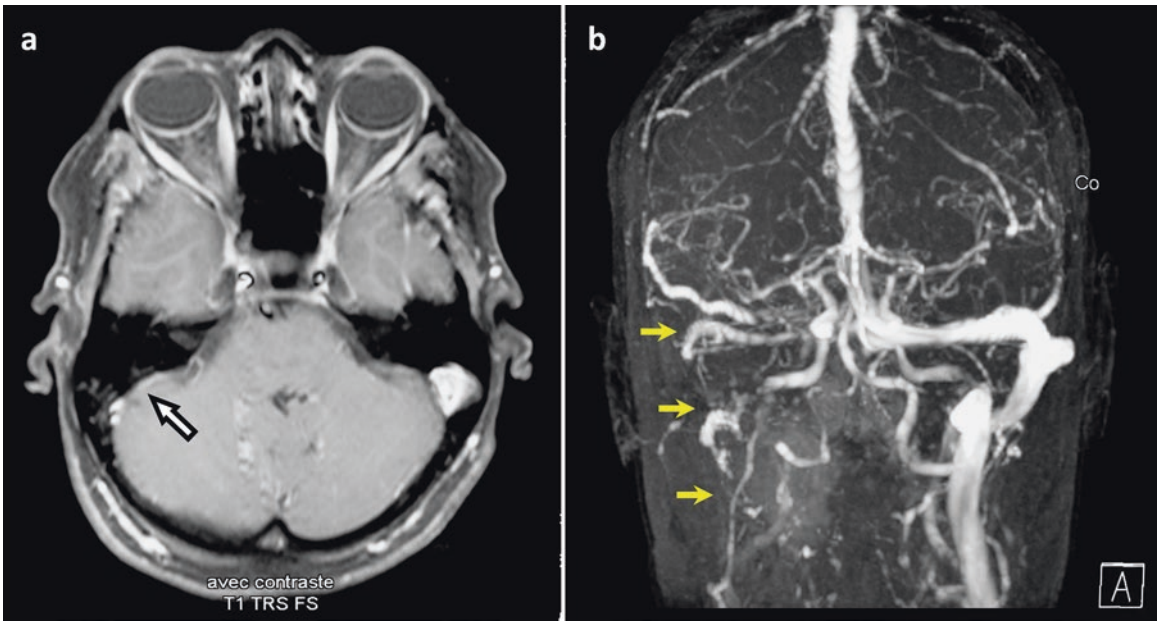


**Fig. 4.11** Case 4.2. Axial enhanced T1-weighted image (**a**) and 3D MR venography (frontal view) (**b**) showing lateral venous sinus thrombosis in the right side (*arrows*), likely due to skull base infection and mastoiditis

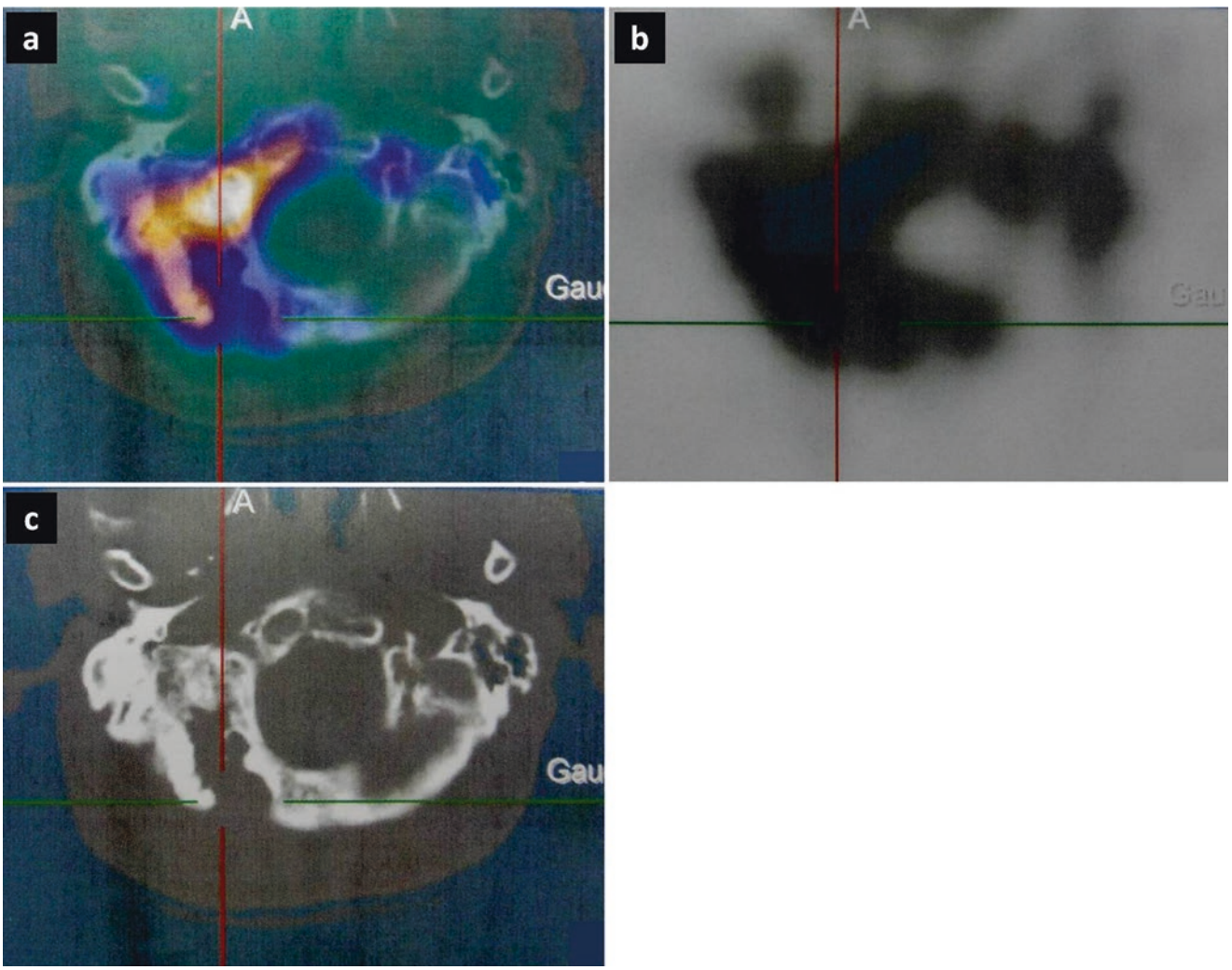


**Fig. 4.12** Case 4.2. The cranial base infection was detected more precisely on axial SPECT/CT (**a**), SPECT (**b**), and CT scan images of the head (**c**)

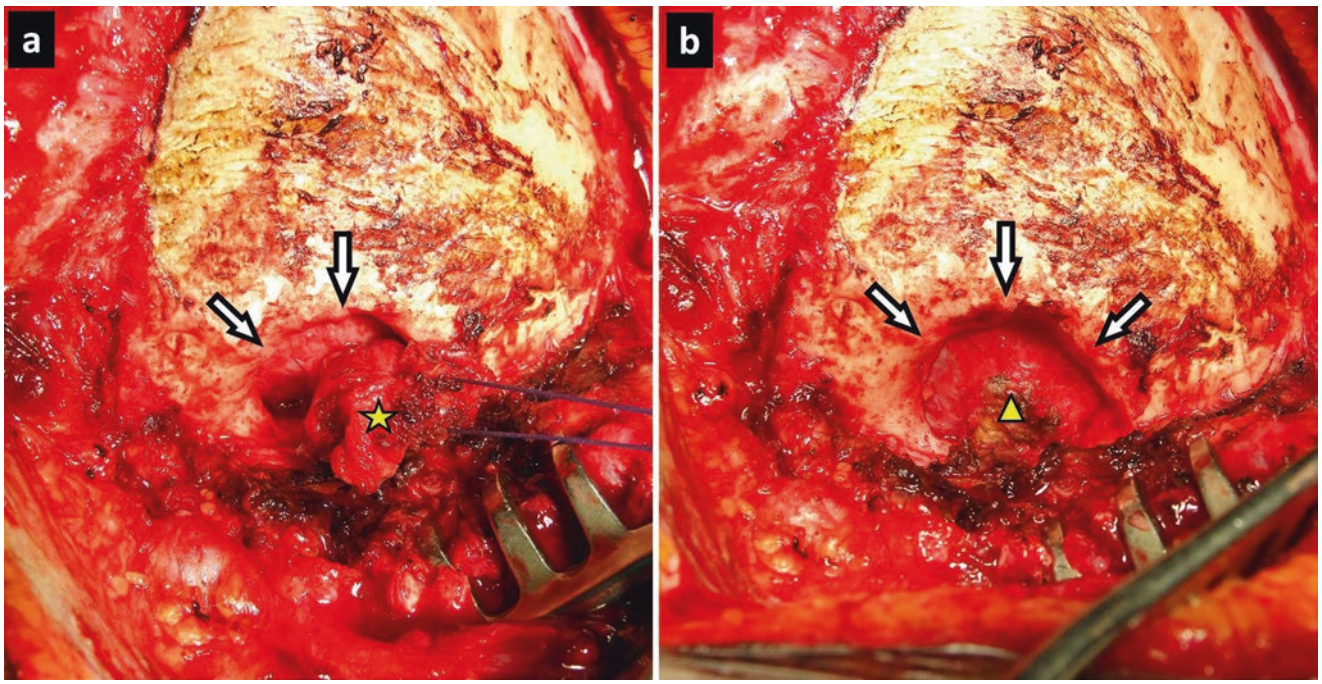


**Fig. 4.13** Case 4.2. Operative view, via a suboccipital approach (**a**, **b**). The occipital bone defect was enlarged (*arrows*), and the dense fibrous capsular abscess was removed (*star*). The abscess wall was adherent to the dura mater but without subdural extension (*triangle*) (**b**)

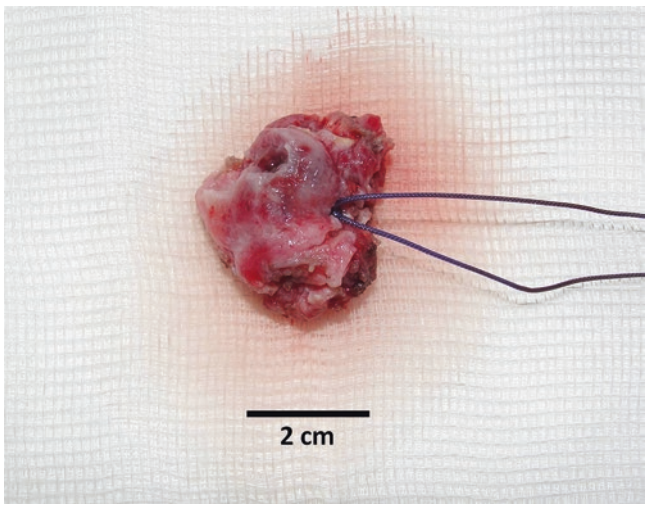


**Fig. 4.14** Case 4.2. Macroscopic appearance of the complete removed capsular abscess. The wall abscess was resected en bloc without complication. *Staphylococcus aureus* was identified in the purulent fluid

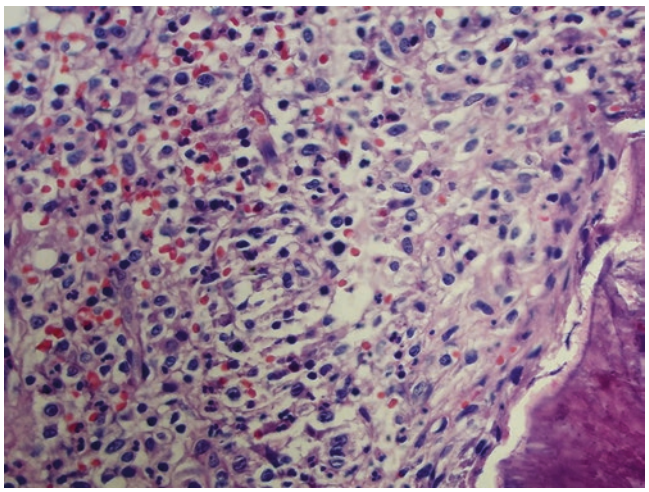

**Fig. 4.15** Photomicrograph of subacute form of cranial osteomyelitis. Bone necrosis with lymphoplasmacytic inflammatory infiltrates, together with neutrophilic granulocytes (high-power magnification, hematoxylin–eosin staining)

## Outcomes

Patients should be monitored closely (clinically, biologically, and on neuroimaging studies) for evidence of therapy success. Unlike biologic inflammatory markers, both CT scans and MRI may take months to years to return to normal following the resolution of the cranial bone infection.

Complete clinical recovery is predictable for most patients. The outcome is related to the severity of infection (especially intracranial complications and skull base osteomyelitis) and to the delay in diagnosis.

Cranial osteomyelitis is often the transitional point between a primary extracranial or otolaryngologic infection and its expected secondary intracranial extension. Osteomyelitis of the skull thus is likely to get worse when

intracranial infectious complications occur, with their potentially lethal consequences.

Long-term sequels are rare, especially chronic headache, seizure, aphasia, permanent neurologic deficit, chronic osteomyelitis, and local discomfort (esthetic scar and hypoesthesia).

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# Cranial Epidural Abscesses

# 5

Cranial epidural abscess is a purulent collection developing between the dura mater and the cranial bone. The occurrence of cranial osteomyelitis is not rare. In developed countries, postoperative craniotomy infections remain the most common cause of the disease, whereas in developing nations, otorhinolaryngologic and dental infections have become the predominant sources. Because of the relative isolation of the brain parenchyma and the insidious development of the infection, these abscesses tend to reach a large size at diagnosis. Biologic parameters may be elevated but are nonspecific. Imaging studies are important tools in the initial diagnosis (biconvex appearance) and in the monitoring of treatment results. The abscess must be evacuated to eradicate the infection. Treatment of the primary source of suppuration can also be performed in the same intervention in most patients. The dura should not be opened, to avoid any meningitis or deeper suppuration. The outcome is related to the delay in diagnosis and to the severity of infection, especially deeper intracranial complications and extensive cranial osteomyelitis.

## Epidemiology and Etiology

Cranial epidural abscess is a suppurative collection developing in the virtual space between the cranial bone and the dura mater (also known as the epidural or extradural space) (Figs. 5.1, 5.2, and 5.3). These lesions are usually well localized, with a biconvex (lentiform) shape. The adherence of the dura mater to the cranial bone can limit the expansion of the epidural abscess. The abscess may be associated with cranial bone infection (cranial osteomyelitis), but it rarely spreads into the subarachnoid space or the brain parenchyma.

Cranial epidural abscesses are the third most common localized intracranial infections, after brain abscesses and subdural empyemas. As with other suppurative intracranial collections, epidural abscesses are usually related to three main sources of infection:

- Direct extension from a contiguous site of infection
- Postsurgical or posttraumatic direct inoculation
- Hematogenous dissemination from a remote source of infection

Many patients have a history of untreated or inadequately treated paranasal sinusitis or otitis media; however, congenital dermal sinuses may be associated with extradural infection.

Epidural abscess occurs most commonly in males during the second and third decades of life, as this population has the greatest probability of developing complicated paranasal sinusitis (Figs. 5.4 and 5.5).

## Clinical Presentations

Patients present systemic signs of infections, sometimes with more localized signs related to scalp abscess and/or cranial osteomyelitis. Headache is a common symptom.

When the abscess increases in size, signs of raised intracranial pressure develop, with altered mental status and focal neurologic signs (according to the location of the disease).

The clinical presentation of epidural abscess is usually more indolent and insidious than the presentation of subdural empyema. Seizures are less common in patients with brain abscess or subdural empyema, and symptoms and signs of meningitis are rare.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_5

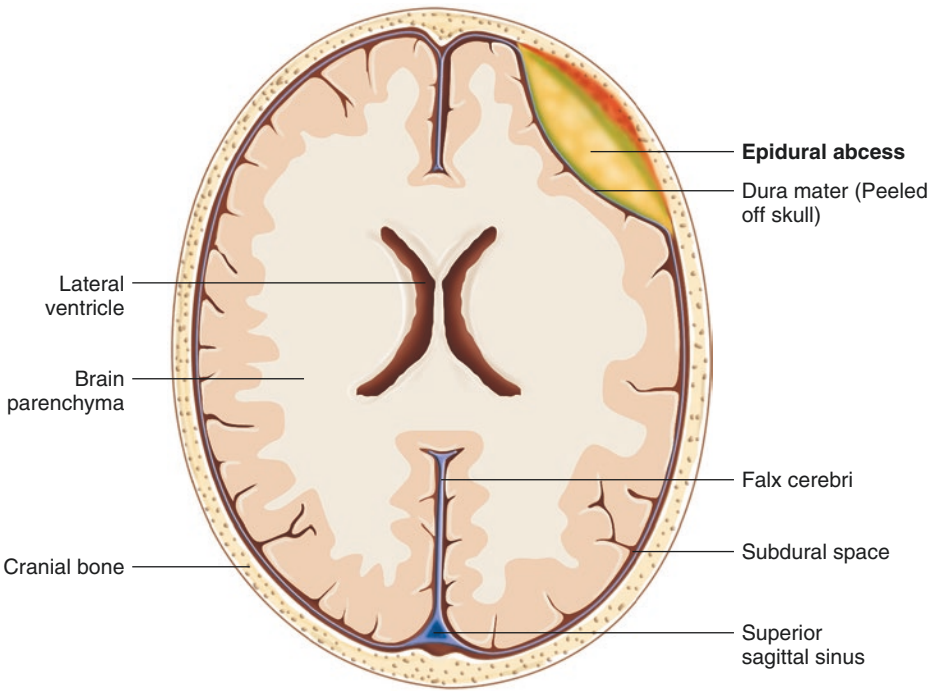


**Fig. 5.1** Localization of cranial epidural abscess (Reproduced from Esenkaya et al.; with permission)

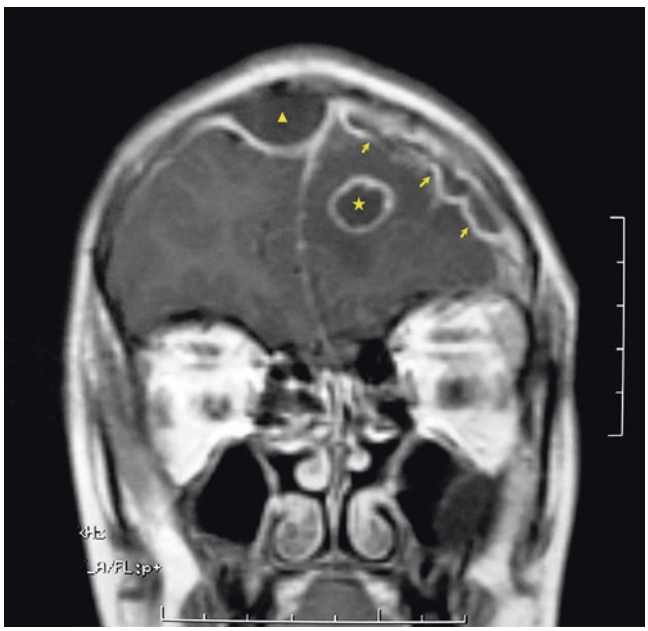

**Fig. 5.2** Coronal cranial T1-weighted MRI following gadolinium injection, showing a rare association of the three main types of intracranial suppurative collections in the same patient: brain abscess (*star*), subdural empyema (*arrows*), and epidural abscess (*triangle*) (Reproduced from Esenkaya et al.; with permission)

Symptoms and signs referred to the primary source of infection (especially ENT infections) should be considered and sought.

## Imaging Features

On CT scan, the epidural abscess appears as a poorly defined lentiform/biconvex collection with low or intermediate density. After contrast injection, the convex inner side of the low-density lesion becomes enhanced (rim enhancement) (Figs. 5.6, 5.7, and 5.8). Small collections may not be visible. Cranial epidural abscess does not significantly deform underlying brain parenchyma unless it is quite large.

On MRI, the lesion generally appears isointense or hypointense on T1-weighted images, with hyperintense patterns on T2-weighted images. High signal on diffusion-weighted imaging (DWI) indicates restricted diffusion. After gadolinium administration, there is thickened dural enhancement, which differentiates suppurative from sterile collections. Apparent diffusion coefficient (ADC) images (low signal) and MR spectroscopy (MRS) (elevated lactate) can confirm the diagnosis.

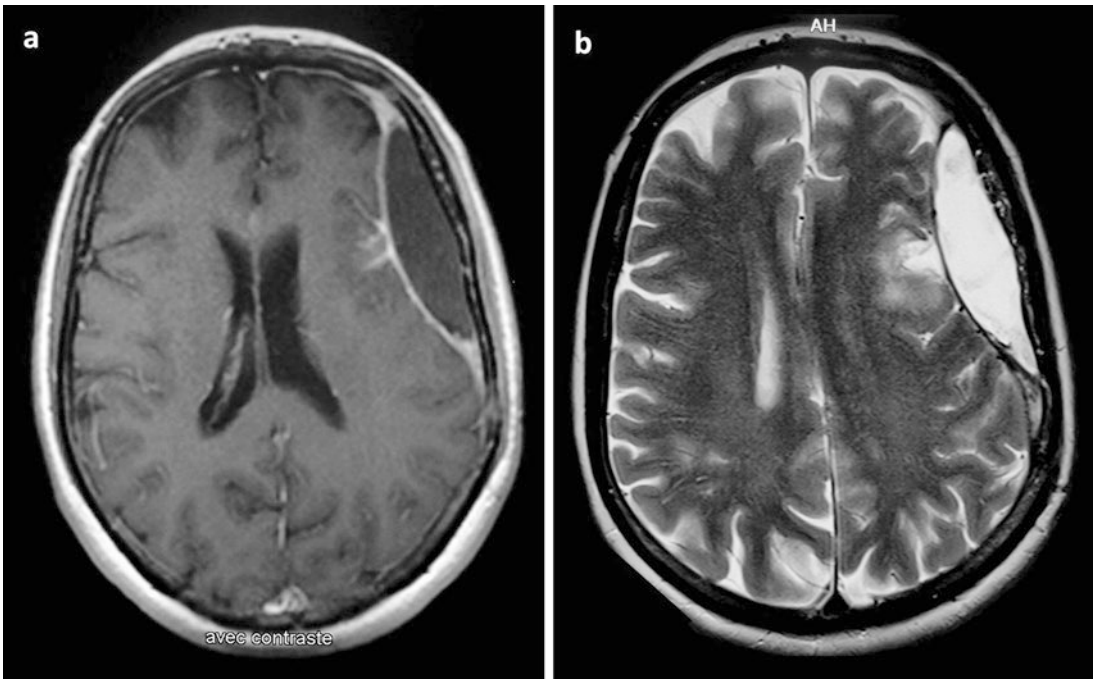


**Fig. 5.3** Large frontoparietal epidural abscess in the left side. Axial cranial post-gadolinium T1-weighted (**a**) and T2-weighted (**b**) images

## Laboratory Findings

Inflammatory parameters (C-reactive protein level, erythrocyte sedimentation rate) and blood count (leukocytosis, anemia) may help in the diagnosis but are unspecific and highly variable in their expression. Procalcitonin levels are more specific.

Aerobic and anaerobic bacteria may be responsible for the infection, often as a polymicrobial suppurative infection. The most common individual pathogens are streptococci, staphylococci, *Bacteroides* species, and *Fusobacterium* species. These microorganisms reflect the bacterial species encountered in paranasal sinus air infection. Sterile cultures are a common finding.

## Treatment Options

Medico-surgical management is needed. Antibiotic therapy should include broad-spectrum antibiotic drugs against aerobic and anaerobic cocci and bacilli, with adequate penetration of the CNS and abscess.

Generally, the standard empiric antibiotic regimen includes vancomycin, metronidazole, and a third-generation cephalosporin. Antistaphylococcal activity should always be considered. Treatment should be optimized according to the results of susceptibility testing of the pathogen isolated.

Patients with small or limited epidural abscess without neurologic complications can be treated with antibiotics only, but most cases require burr hole craniotomy for decompression, with irrigation, debridement, and drainage. The dura should not be opened, to avoid any meningitis or deeper suppuration. A large bone flap will be necessary in cases with a more solid, organized granulomatous collection or if the patient has not improved clinically despite receiving appropriate intravenous antimicrobial therapy.

In many patients, treatment of concomitant rhinosinusitis, orodental infection, or otomastoiditis is needed to eliminate the primary source of infection.

## Outcomes

A delay in surgical intervention and antibiotic therapy has been associated with higher morbidity and mortality rates. Repeat surgical procedures may be required in cases of persistent or recurrent suppurative collection.

Close clinical, biological, and neuroimaging monitoring is important to ensure adequate response to treatment (Figs. 5.9 and 5.10).

Complete clinical recovery is predictable for most patients. The outcome is related to the severity of infection (especially cranial base infections and deeper collections) and to the delay in diagnosis. Long-term sequels are rare (aphasia, seizure, permanent neurologic deficits, and chronic osteomyelitis).

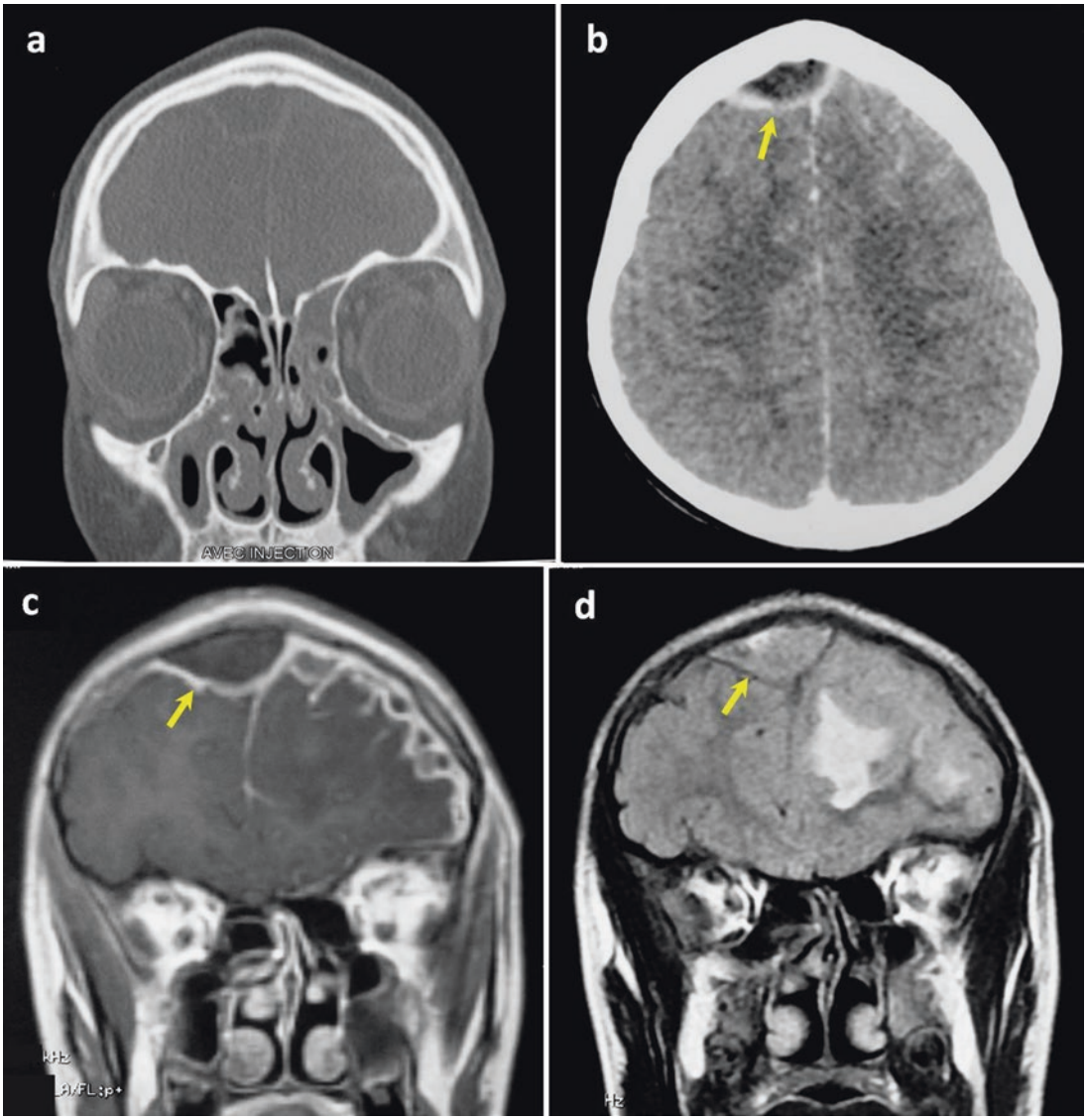


**Fig. 5.4** Case 5.1. A boy with a history of inadequately treated paranasal sinusitis who developed an epidural abscess. Coronal maxillofacial (**a**) and axial post-contrast CT scan (**b**) showing the paranasal sinusitis and the right intracranial epidural abscess with peripheric contrast enhancement (*arrow*). Coronal cranial MRIs show the epidural abscess collection (*arrows*) on post-gadolinium T1-weighted image (**c**) and a FLAIR sequence (**d**)

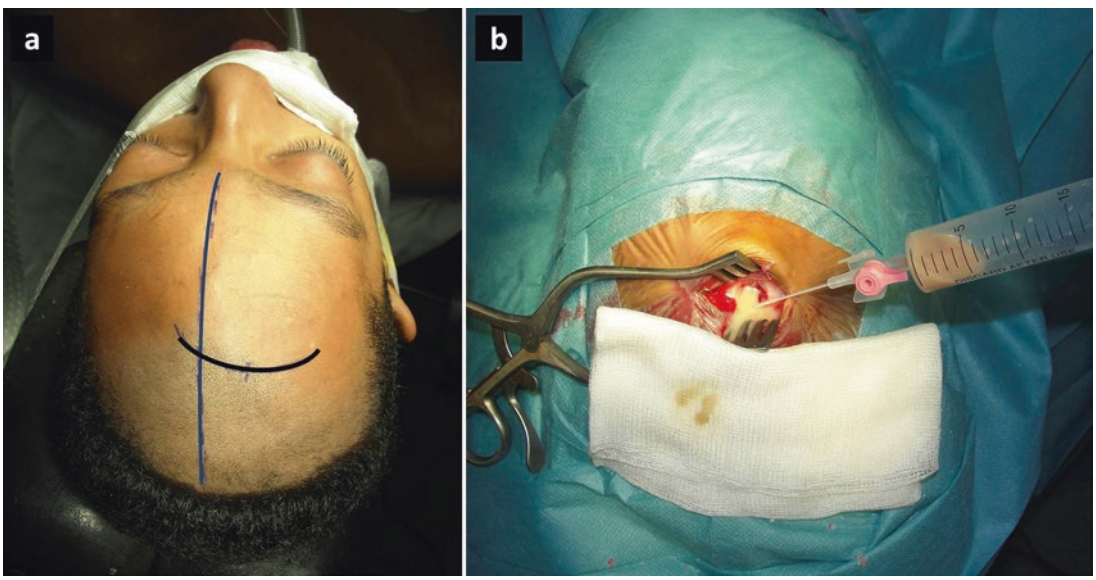


**Fig. 5.5** Case 5.1. Operative view of the limited frontal scalp incision (*black line*) (**a**) and the epidural pus aspiration through a burr hole craniotomy (**b**)

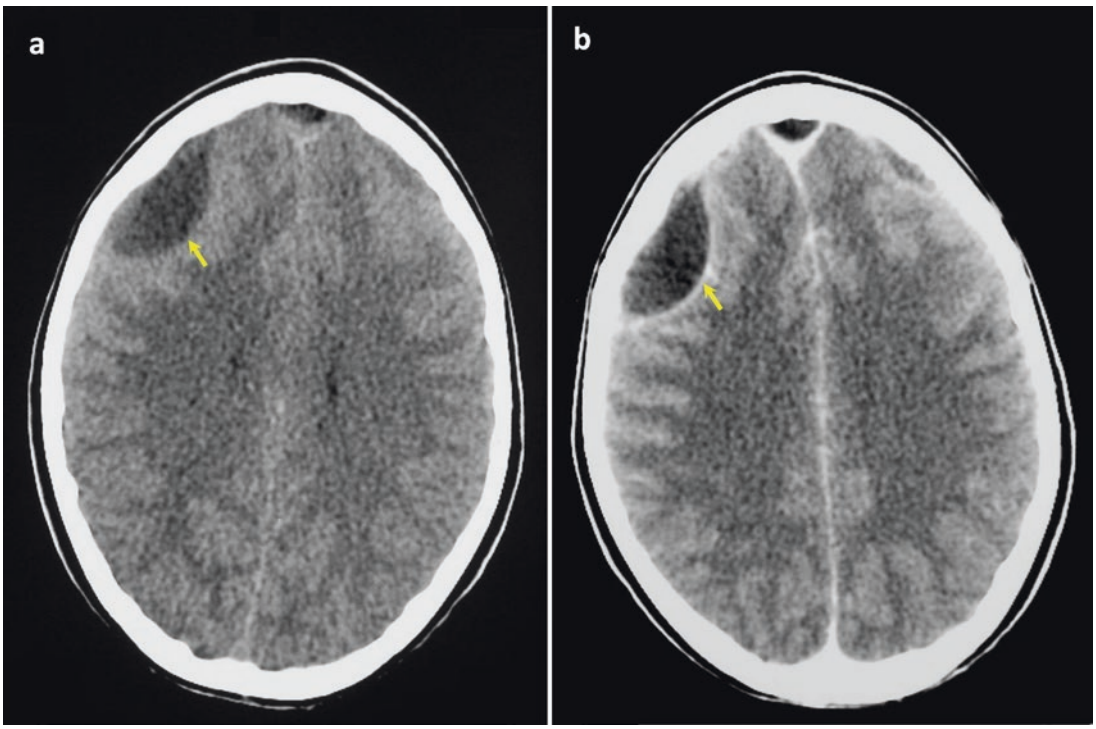


**Fig. 5.6** Case 5.2. Axial cranial CT scan before (**a**) and after (**b**) contrast administration, revealing a frontal epidural fluid collection in the right side with inner side peripheral enhancement

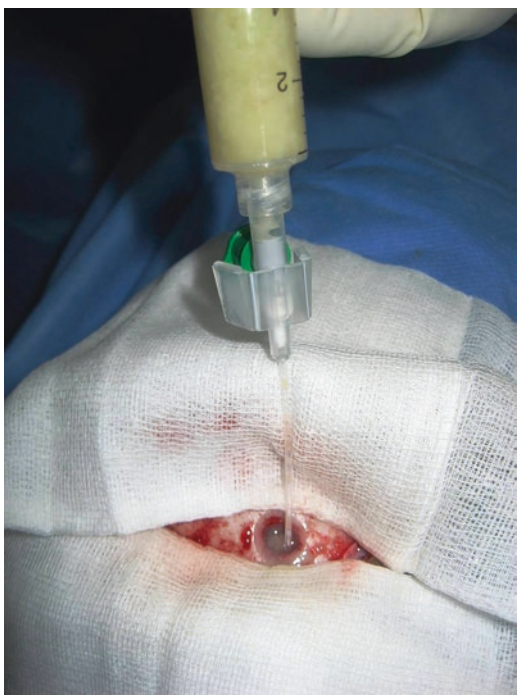

**Fig. 5.7** Case 5.2. Operative view of the surgical procedure: aspiration of the epidural pus following a frontal burr hole craniotomy

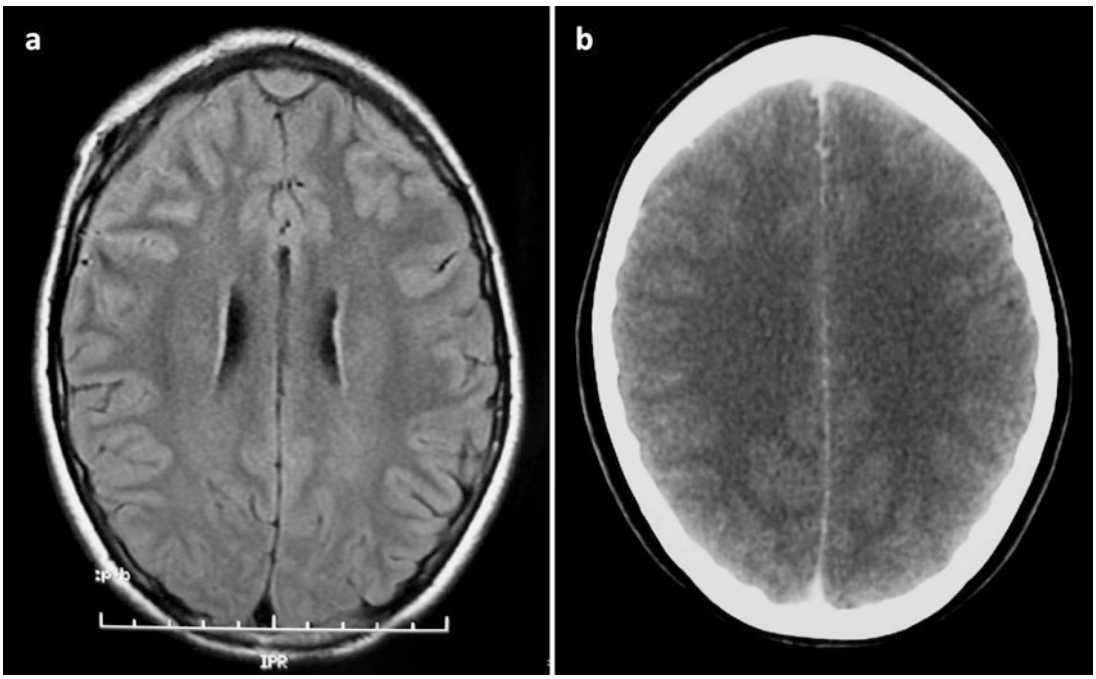


**Fig. 5.8** Case 5.2. Postoperative neuroimaging. Axial cranial FLAIR MRI (1 week later) (**a**) and post-contrast CT scan (1 month later) (**b**)

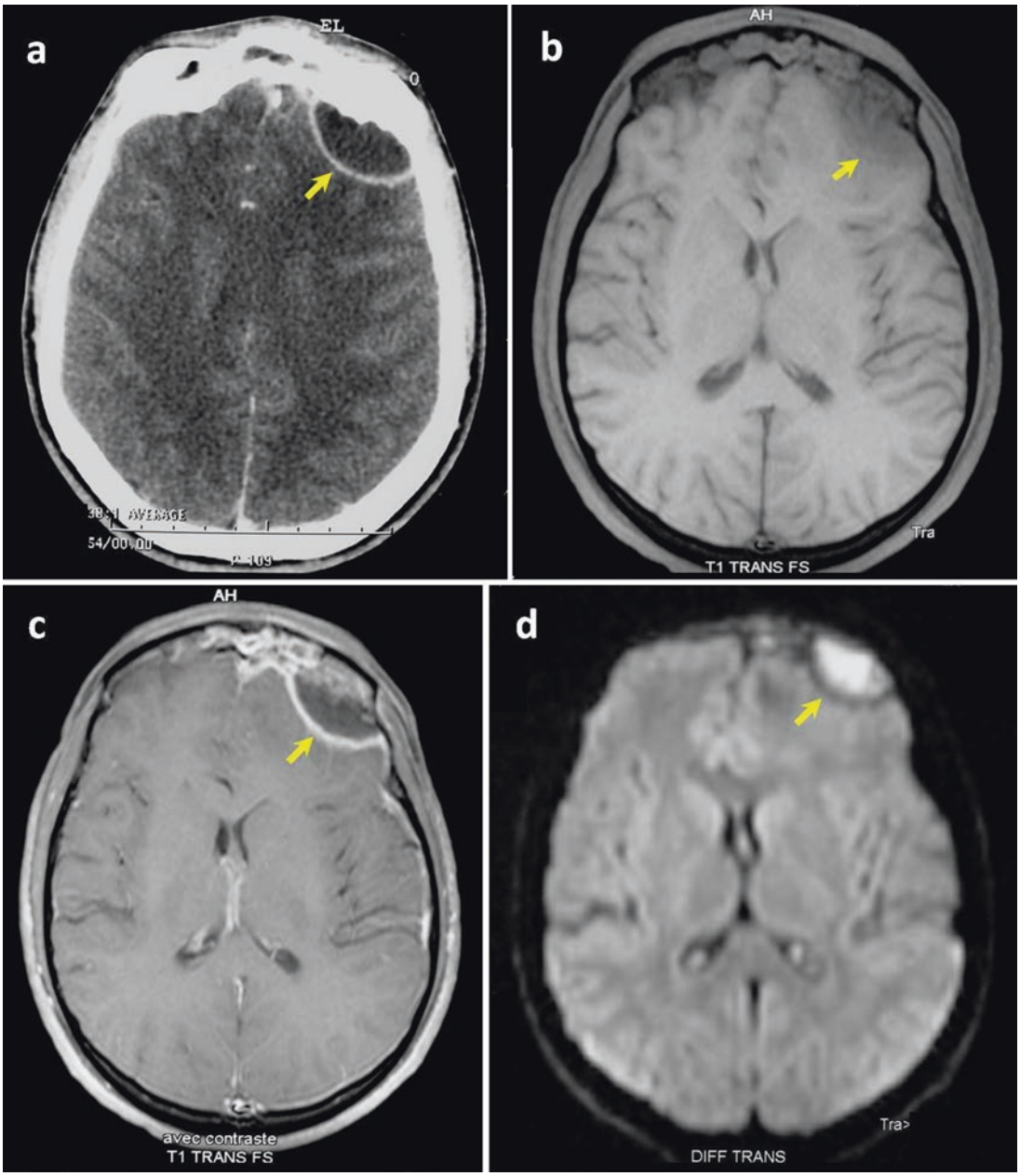


**Fig. 5.9** Case 5.3. Cranial epidural abscess (*arrow*) in a young man with untreated frontal paranasal sinusitis. Axial cranial post-contrast CT scan (**a**), T1-weighted MRI before (**b**) and following (**c**) gadolinium administration, and a diffusion-weighted image (**d**)

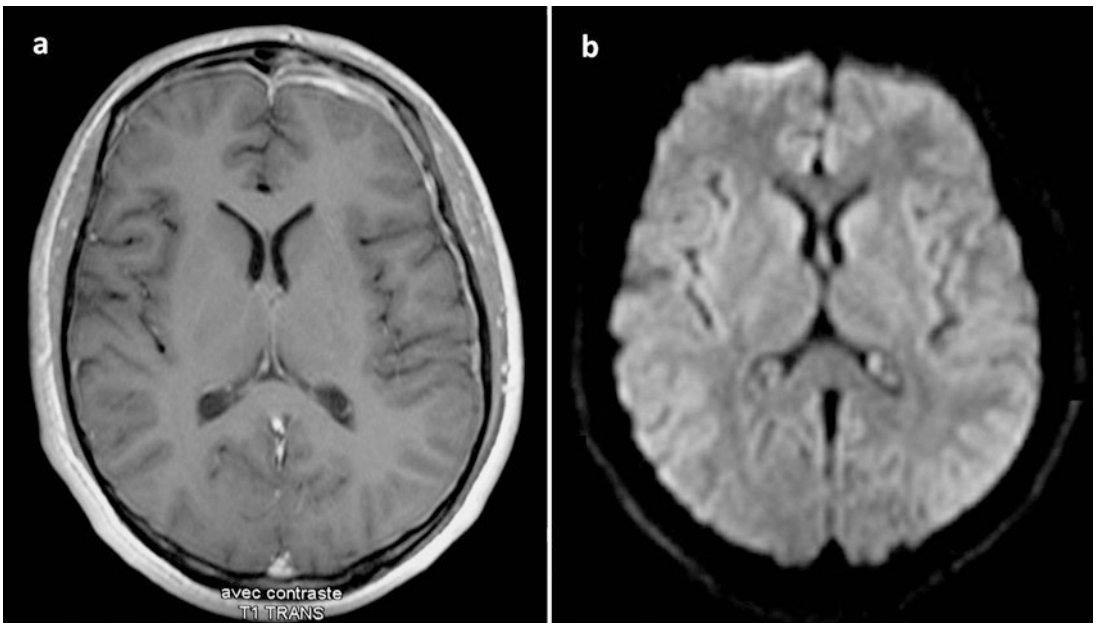


**Fig. 5.10** Case 5.3. Postoperative axial cranial T1-weighted MRI with gadolinium injection (**a**) and on diffusion-weighted sequence (**b**) 4 weeks later

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# Cranial Subdural Empyemas

# 6

Cranial subdural empyema is a purulent collection developing between the dura mater and the arachnoid membrane (Fig. 6.1). A large range of pathogens may be responsible for the infection, and their successful culture is the solution to optimizing the medical section of management. The occurrence of associated meningitis is not rare. Cranial subdural empyema should always be considered in any patient who presents with meningeal signs and a focal neurologic deficit. Biologic parameters may be elevated but are nonspecific. Imaging studies (especially MRI) are important tools in the initial diagnosis (identifying an extra-axial crescent-shaped collection) as well as in the monitoring of treatment results (Figs. 6.2 and 6.3). The antibiotic regimen is classically similar to the treatment for cerebral abscess. Subdural empyema represents a surgical emergency, so craniotomy and rapid evacuation of the purulent collection are obligatory as soon as it is diagnosed. Eradication of primary infected foci should not be missed. Subdural empyema is more fulminant and fatal than epidural abscess. Persistent seizures and residual hemiparesis are the most important sequelae. Mortality may result from associated venous infarction of the brain.

## Epidemiology and Etiology

Subdural empyema is a purulent collection in the subdural space (between the dura and the arachnoid membrane) causing inflammation and edema of the adjacent brain, septic thrombophlebitis, and venous infarction, which can rapidly expand into the subarachnoid space and the brain parenchyma.

The most common source of subdural empyema is from contiguous spread, mainly from paranasal purulent sinusitis, middle ear/mastoid air sinus infection, or odontogenic origin (Figs. 6.4 and 6.5). In the pediatric population, bacterial meninges may induce subdural empyema. This suppurative condition is rarely encountered following craniotomy or traumatic injuries, but subdural empyema secondary to chronic subdural hematoma is a well-known infectious complication (*See* Chap. 21).

Subdural empyema, like epidural abscess, occurs most usually in males during the second and third decades of life, corresponding to the population with maximum probability of developing complications of otorhinolaryngologic infections.

## Clinical Presentations

This classic fulminant infectious disease may have a more serious clinical presentation than that of cranial epidural abscess. Symptoms are due to mass effect, inflammatory involvement of the brain and meninges, and thrombophlebitis of cerebral veins and/or dural venous sinuses. Headache, fever, nuchal rigidity (meningismus), and seizure are common. Focal neurologic deficits vary according to the size and location of the disease. Cranial subdural empyema should always be considered for any patient who presents with meningeal signs and a focal neurologic deficit (Figs. 6.6 and 6.7).

Symptoms and signs referred to the primary source of infection (especially ENT infections) should be considered and sought.

## Imaging Features

CT scan findings usually demonstrate a low-density crescent-shape collection over the brain hemisphere or along the falx with internal displacement of the gray–white junction and the median structures. Loculations may also be associated. Mass effect is habitually caused more by edema than by the subdural collection itself. The peripheral margins are better demarcated after contrast injection, particularly along the medial border of the collection at the pial surface. However, subdural empyema can be missed by the CT scan, and MRI has become the best neuroimaging technique (Figs. 6.8, 6.9, 6.10, and 6.11).

On MRI, this extra-axial collection generally appears isointense to hypointense on T1-weighted images with



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_6

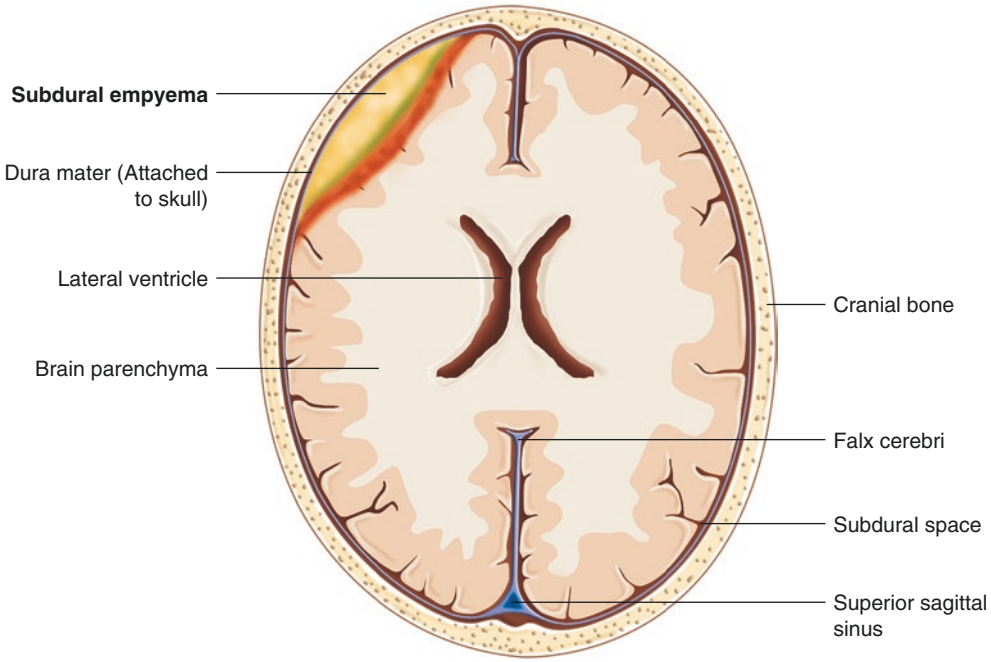


**Fig. 6.1** Localization of cranial subdural empyema

hyperintense patterns on T2-weighted images. They have high signal on diffusion-weighted imaging (DWI) indicating restricted diffusion (bright signal). After gadolinium administration, there is thickened dural enhancement which differentiates suppurative from sterile collections. Apparent diffusion coefficient (ADC) images (low signal) and MR spectroscopy (MRS) (elevated lactate) can confirm the diagnosis. Cerebral edema, encephalitis, venous infarction, mass effect, and brain abscess formation are more easily highlighted than on CT scans.

Potential cerebrovascular complications, especially dural venous thrombosis, may be seen on MR venography.

Abnormalities on electroencephalography may be localized and can help in excluding a more generalized intracranial disease like encephalitis.

## Laboratory Findings

Inflammatory parameters (C-reactive protein, erythrocyte sedimentation rate) and blood count (leukocytosis, anemia) may help in the diagnosis but are unspecific and highly variable in their expression. Procalcitonin levels seem more specific.

Subdural empyema may be bacterial (aerobic or anaerobic bacteria), fungal, or mycobacterial. (*See* the information on specific pathogens in section "Treatment Options"). It is often a polymicrobial suppurative infection. The most common individual pathogens are streptococci, staphylococci, *Bacteroides* species, and *Fusobacterium* species. These microorganisms reflect the bacterial species encountered in paranasal sinus infections. Sterile cultures are not uncommon. *Haemophilus influenzae* and *Streptococcus pneumoniae* may be responsible for infection in infants following purulent meningitis. Blood cultures are rarely positive.

Lumbar puncture is generally not recommended because of the possible occurrence of high intracranial pressure and subsequent risk of brain herniation. Organisms are usually present only in cases of meningitis.

## Treatment Options

The antibiotic regimen is classically similar to treatment for cerebral abscess, usually including a third-generation cephalosporin and metronidazole (sometimes in addition to vancomycin or amikacin) for at least 8 weeks.

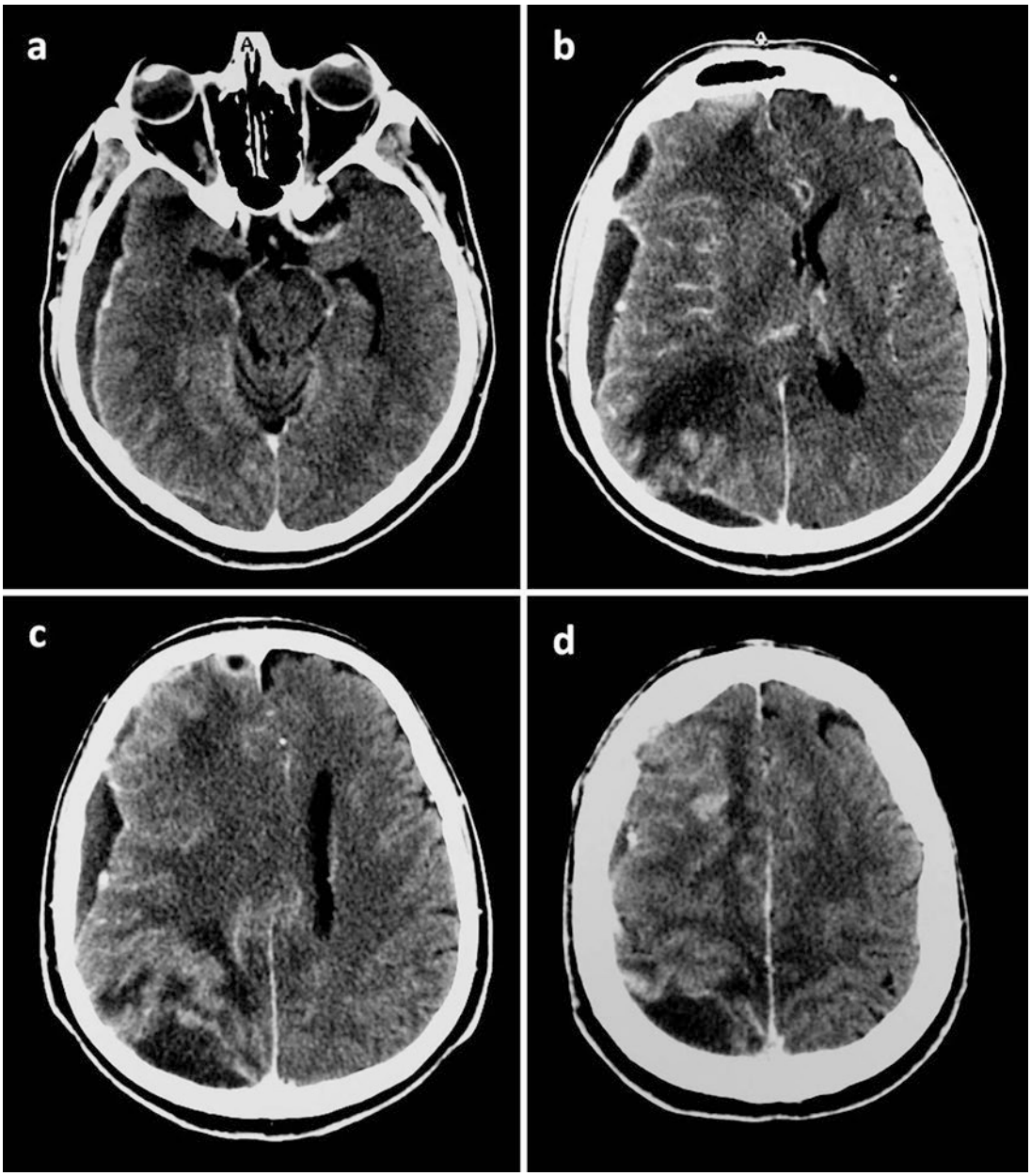


**Fig. 6.2** Case 6.1. (**a**–**d**) Axial cranial enhanced CT scans showing multiple subdural empyemas in the right side

Surgical management is indicated in almost all cases of subdural empyema for cerebral decompression, drainage of purulent material, and identification of causative bacteria. Surgical procedures include burr hole drainage, stereotactic drainage for deep-seated parafalcine or tentorial empyemas, and craniotomy for irrigation, débridement, and drainage (Figs. 6.12, 6.13, 6.14, 6.15, 6.16, and 6.17).

Purulent material tends to be in a fluid state early in the disease course, so the pus may be more amenable to burr hole drainage. With time, the suppuration may become loculated and require craniotomy. Repeat surgical procedures may be needed.

An external ventricular drain should be placed to prevent CSF obstruction in some patients with obstructive hydrocephalus. The CSF drainage will be either gradually withdrawn over time and removed or replaced later by a definitive shunt (when the CSF will be sterile).

Use of corticosteroids has been controversial; they may be beneficial in patients with secondary brain edema resulting in raised intracranial pressure.

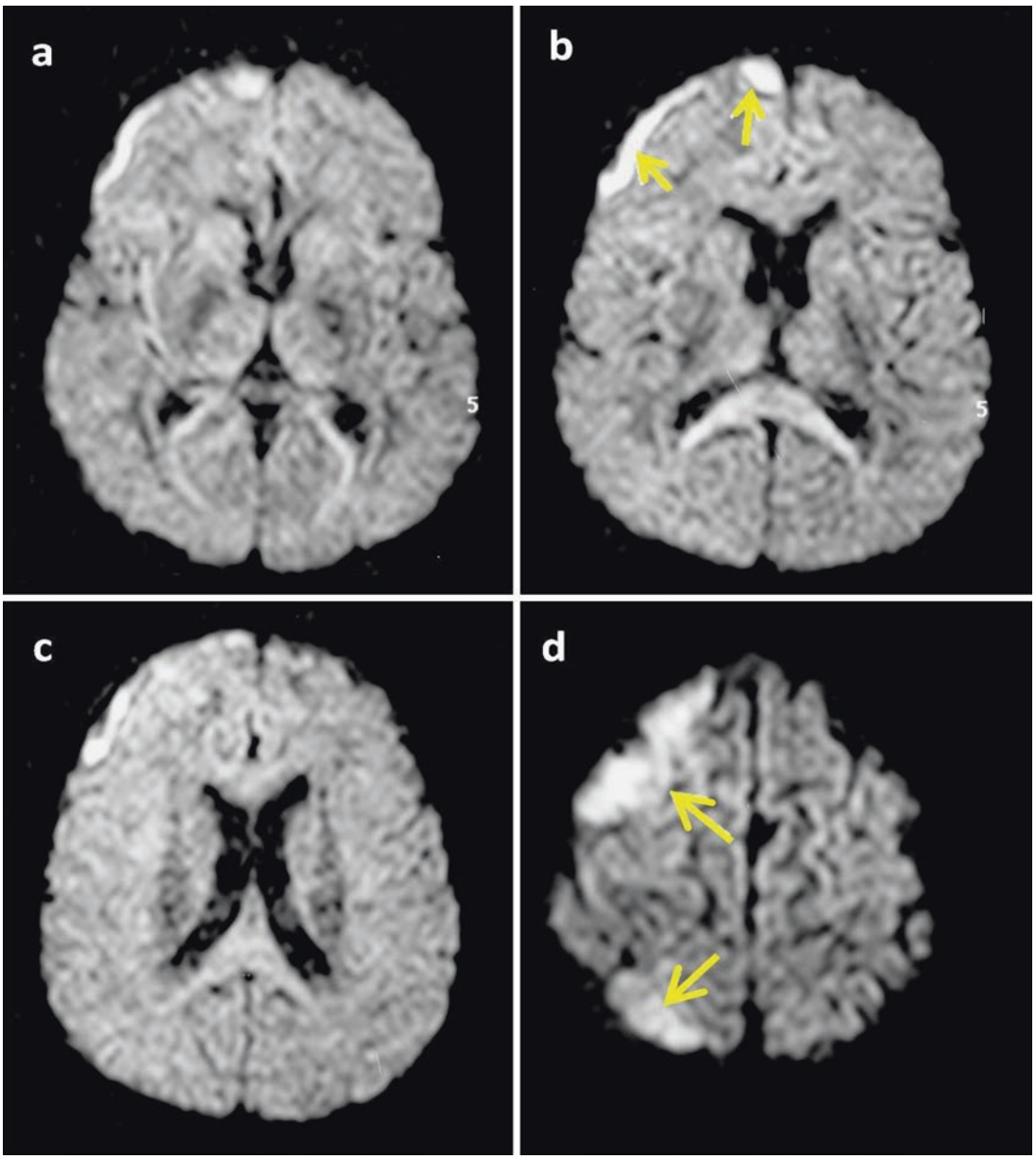


**Fig. 6.3** Case 6.1. (**a**–**d**) Axial cranial diffusion-weighted MRI. Subdural empyemas have high signal, indicating restricted diffusion (bright signal) (*arrows*)

As with other severe neuroinfections, many additional supportive therapies may be necessary in addition to initial resuscitation and management of severe infection issues. Also needed may be anticonvulsants, corticosteroids, anticoagulants, analgesics, antipyretics, treatment of any medical comorbidities, and functional rehabilitation.

## Outcomes

Subdural empyema is more fulminant and fatal than epidural abscess. Persistent seizures and residual hemiparesis are the most important sequelae. Mortality may be associated with venous infarction of the brain.

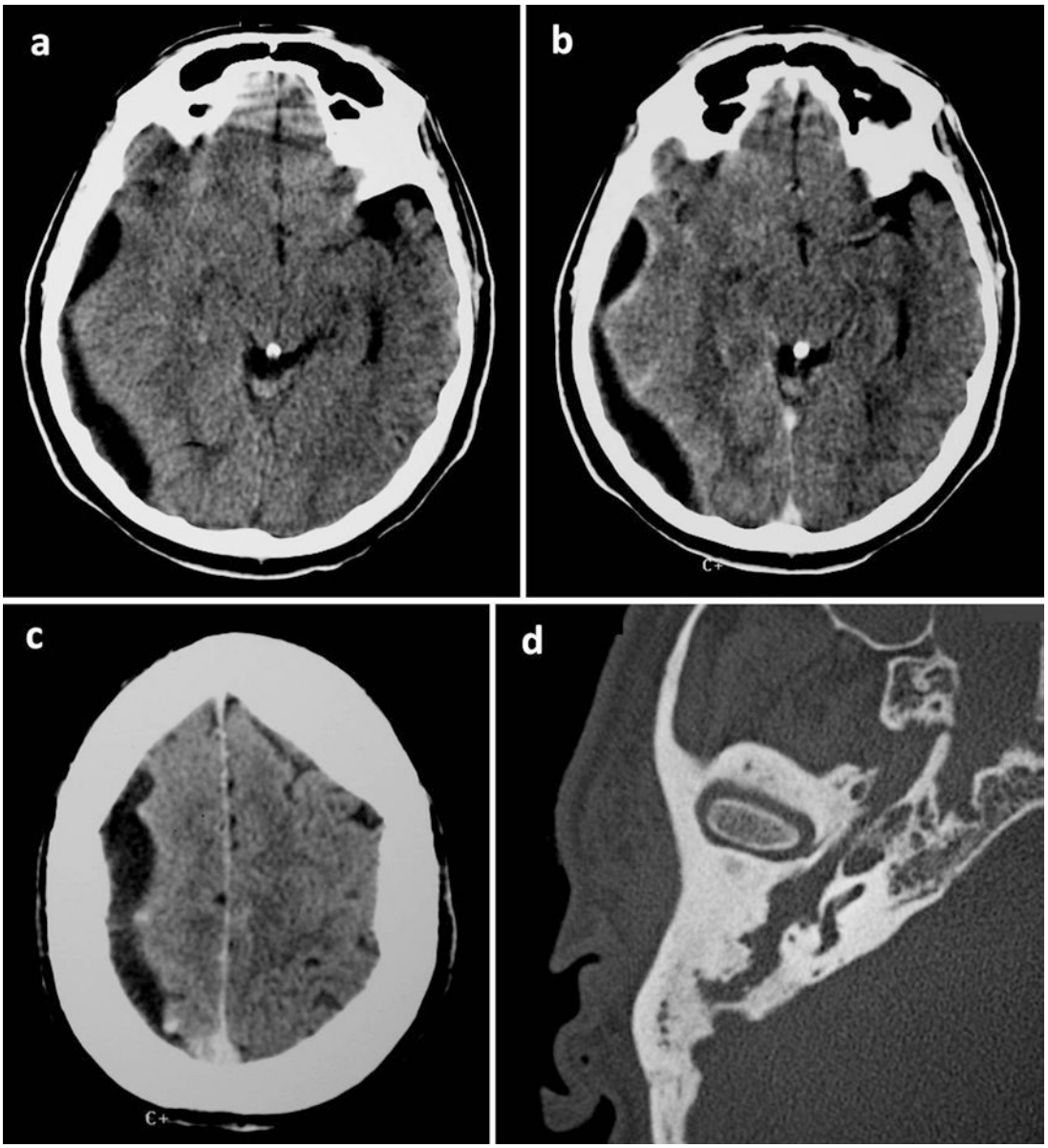


**Fig. 6.4** Case 6.2. (**a**–**c**) Axial cranial enhanced CT scans showing hemispheric subdural empyema in the right side. (**d**) This patient has also an ipsilateral chronic otitis media seen on axial cranial CT-scan on bone window

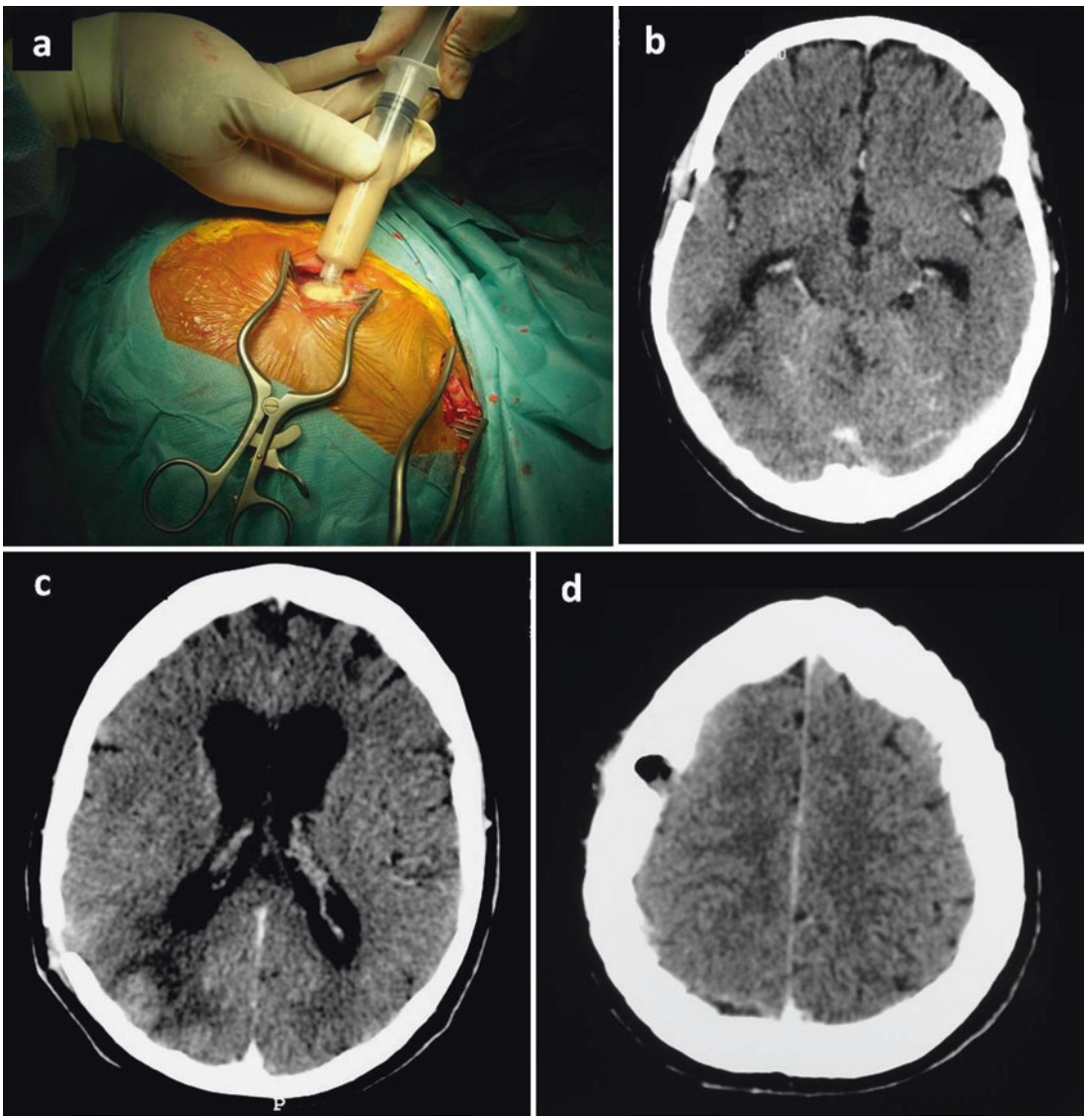


**Fig. 6.5** Case 6.2. (**a**) Operative view showing drainage of the subdural suppurative collection through two burr-hole craniotomies. (**b**–**d**) Postoperative axial cranial CT scans after contrast administration 1 month later

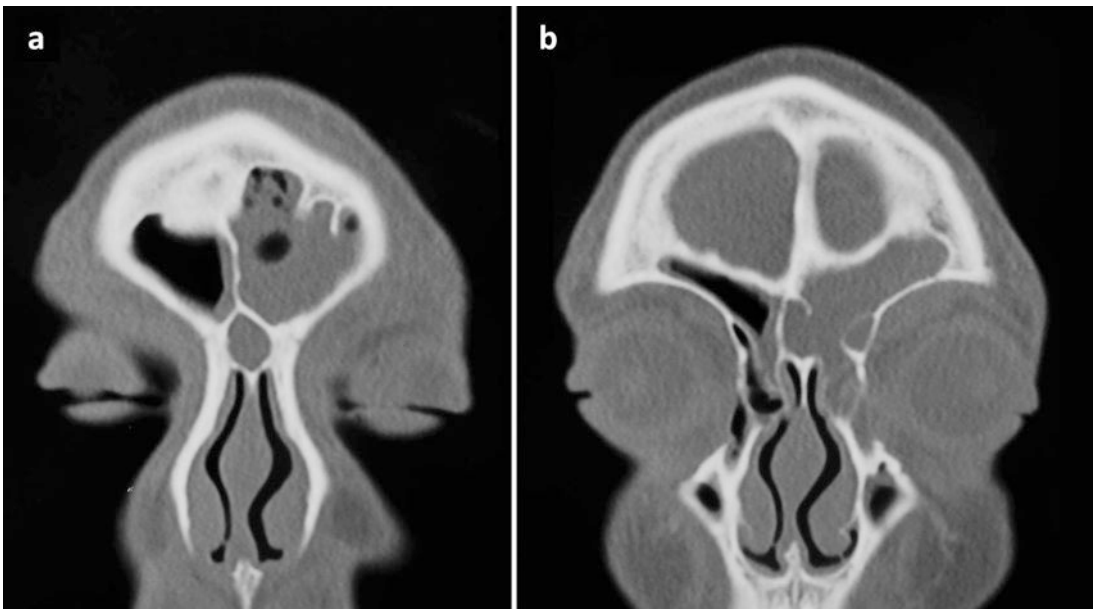


**Fig. 6.6** Case 6.3. (**a**, **b**) Coronal cranio-facial CT scan in a 24-year-old man with a long history of refractory paranasal sinusitis

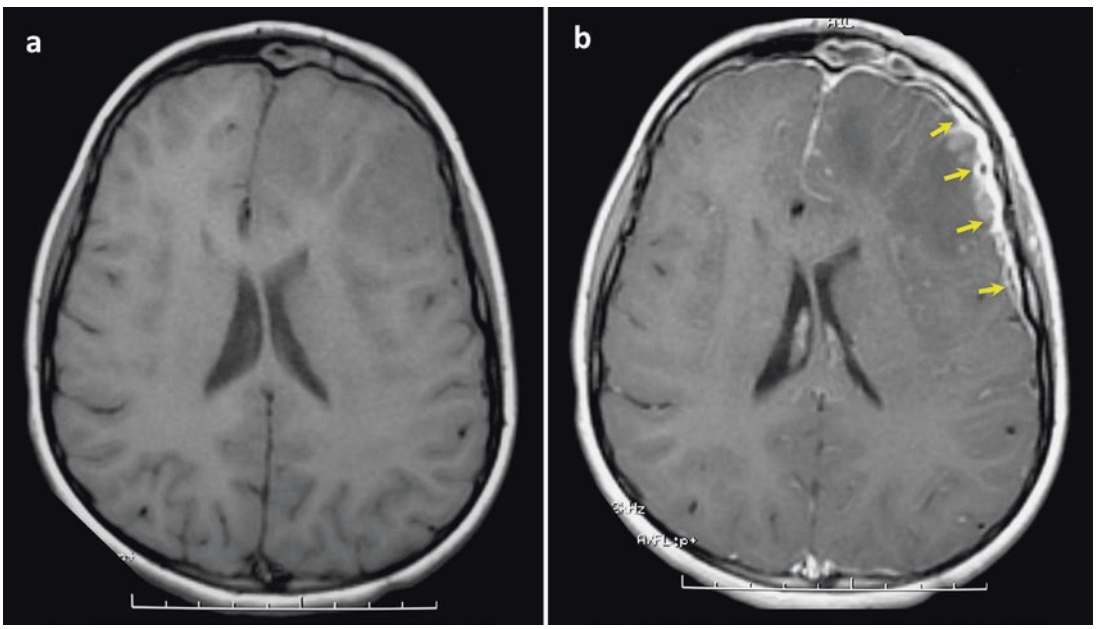


**Fig. 6.7** Case 6.3. The patient developed rapid symptoms of meningo-encephalitis. Axial cranial T1-weighted MRI before (**a**) and after (**b**) gadolinium administration reveals a small subdural empyema in the left side with features of pachymeningitis and adjacent brain edema. Note the thickened dural enhancement (*arrows*) (**b**)

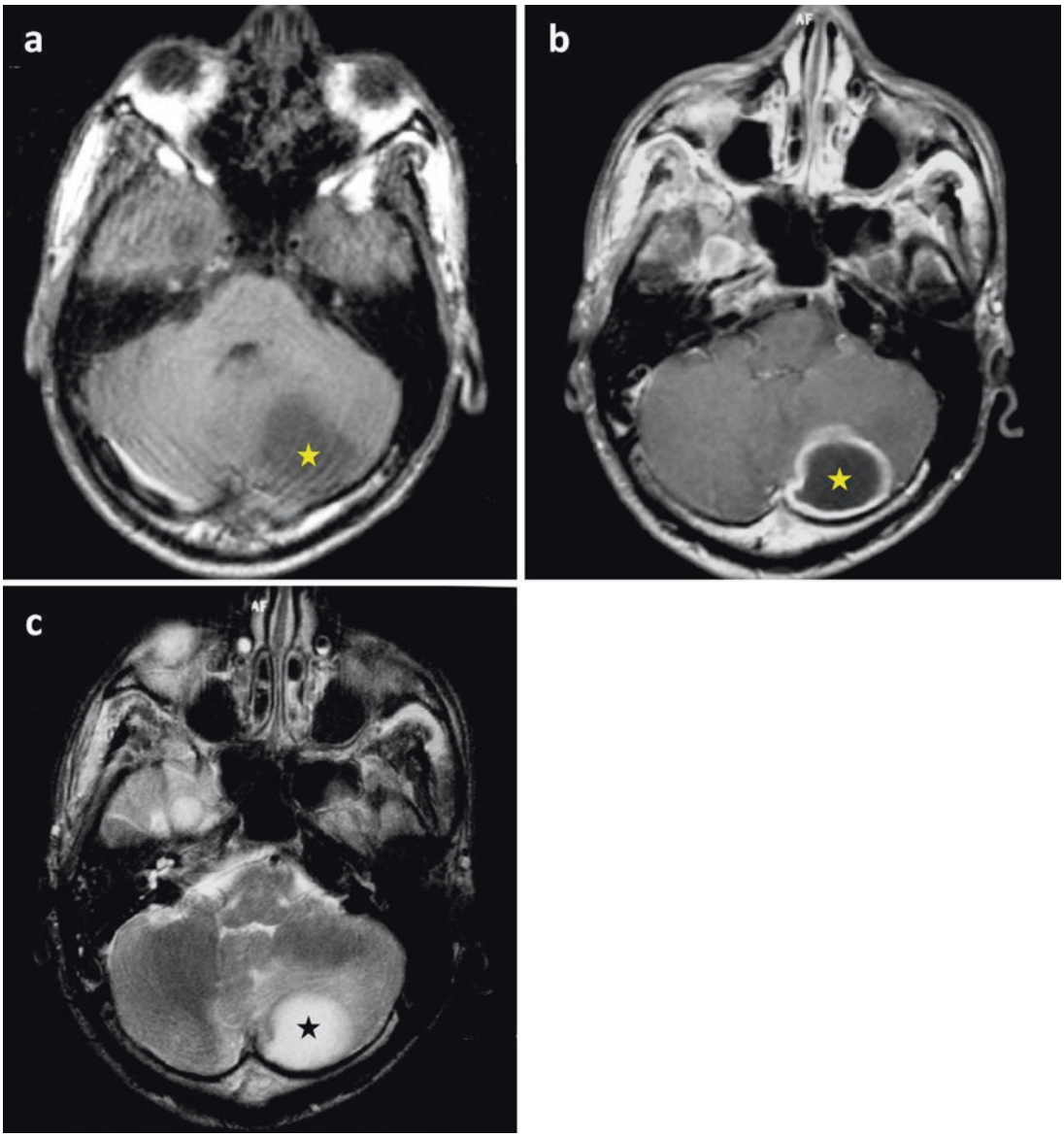


**Fig. 6.8** Axial cranial MRI showing a posterior fossa subdural empyema in the left side (cerebellar convexity) (*star*). T1-weighted images before (**a**) and after (**b**) gadolinium administration and T2-weighted image (**c**) show the empyema appearance

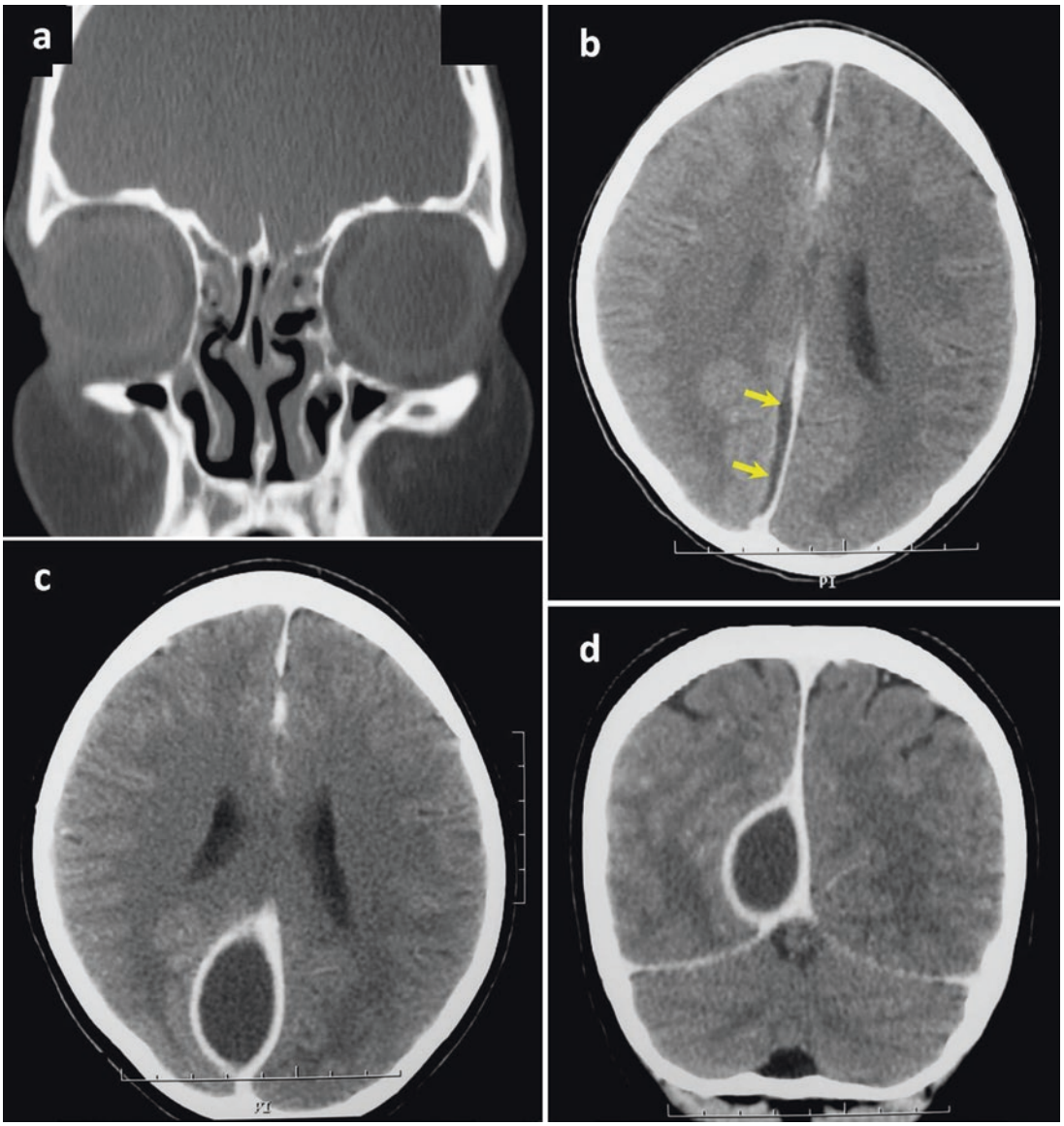


**Fig. 6.9** Interhemispheric (parafalcine) subdural empyema in a 14-year-old girl who was adequately treated for a paranasal sinusitis. Coronal cranio-facial CT scan (**a**) and axial cranial CT scan with contrast enhancement (**b**). Initially, this subdural empyema (*arrows*) was treated only with empiric broad-spectrum antibiotic therapy, but the patient's clinical symptoms worsened 1 week later. On control CT scan (**c**, **d**), the empyema increased in volume and then drained surgically

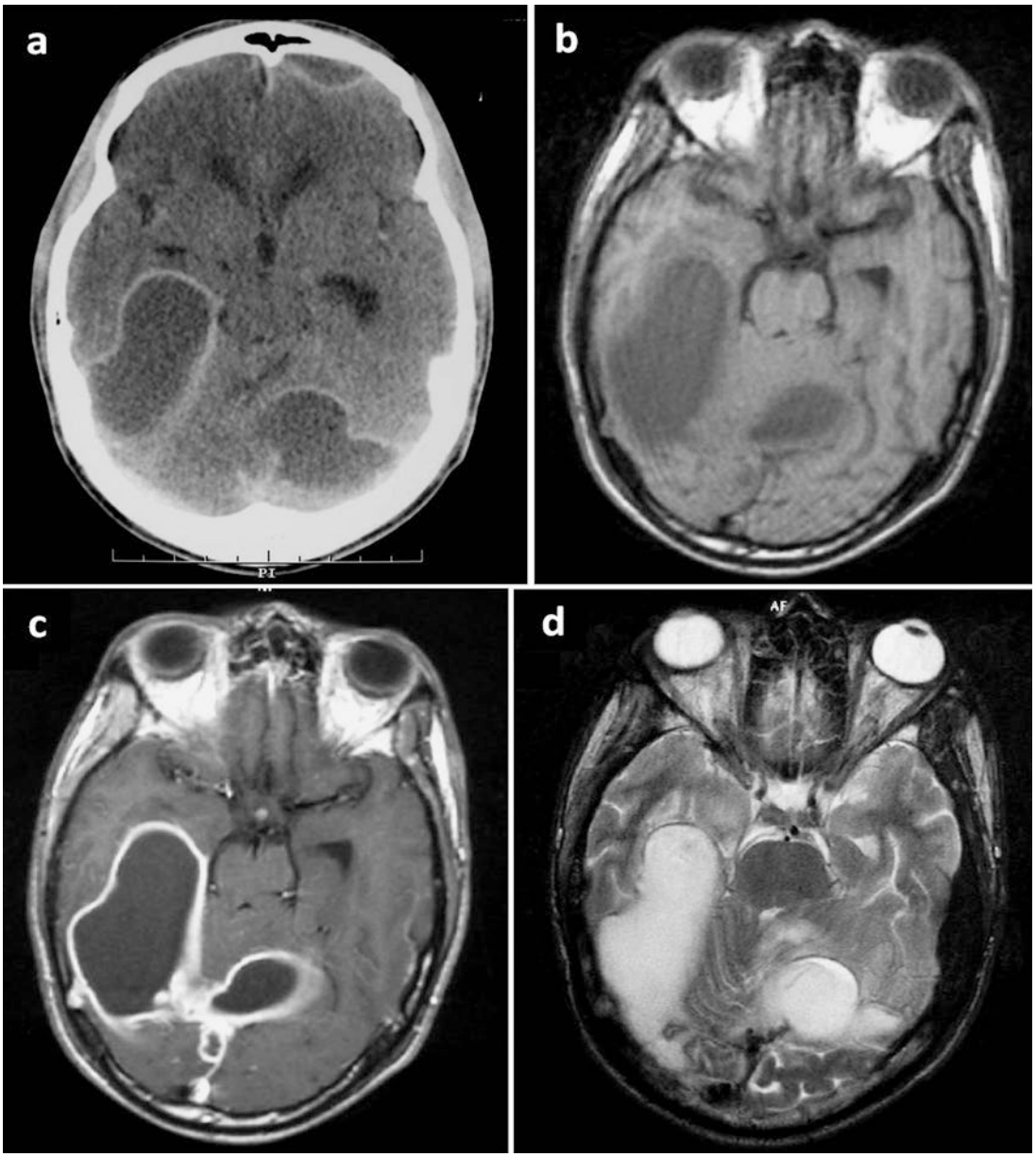


**Fig. 6.10** Case 6.4. Axial post-contrast CT scan (**a**), T1-weighted images without (**b**) and with (**c**) gadolinium injection, and a T2-weighted image (**d**) show multiple supratentorial (*right side*) and infratentorial (*left side*) subdural empyemas mimicking intraparenchymal brain abscesses

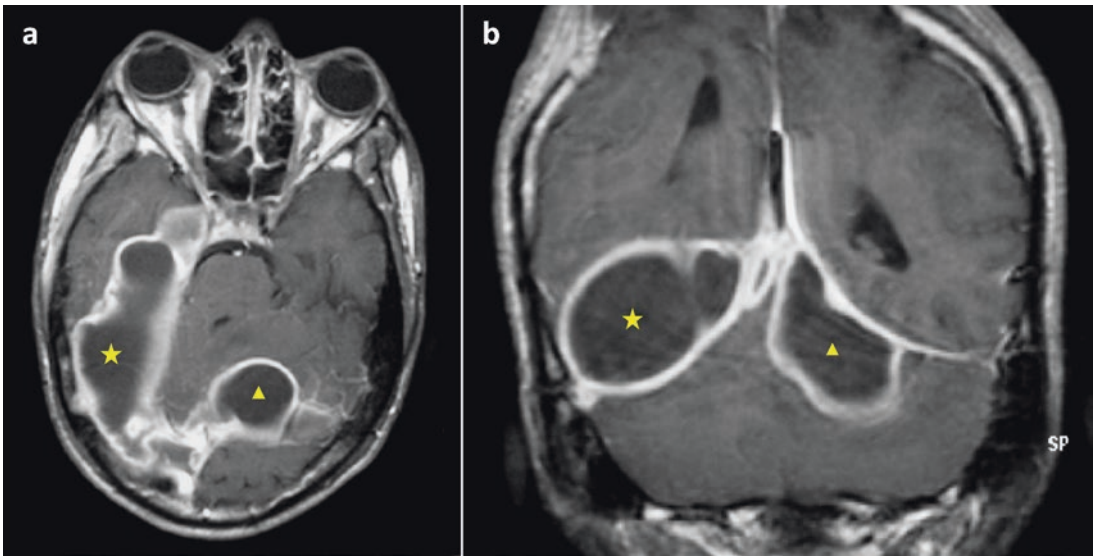


**Fig. 6.11** Case 6.4. Axial (**a**) and coronal (**b**) post-gadolinium T1-weighted MRI, showing various parafalcine, supratentorial (*star*), and infratentorial (*triangle*) subdural empyemas

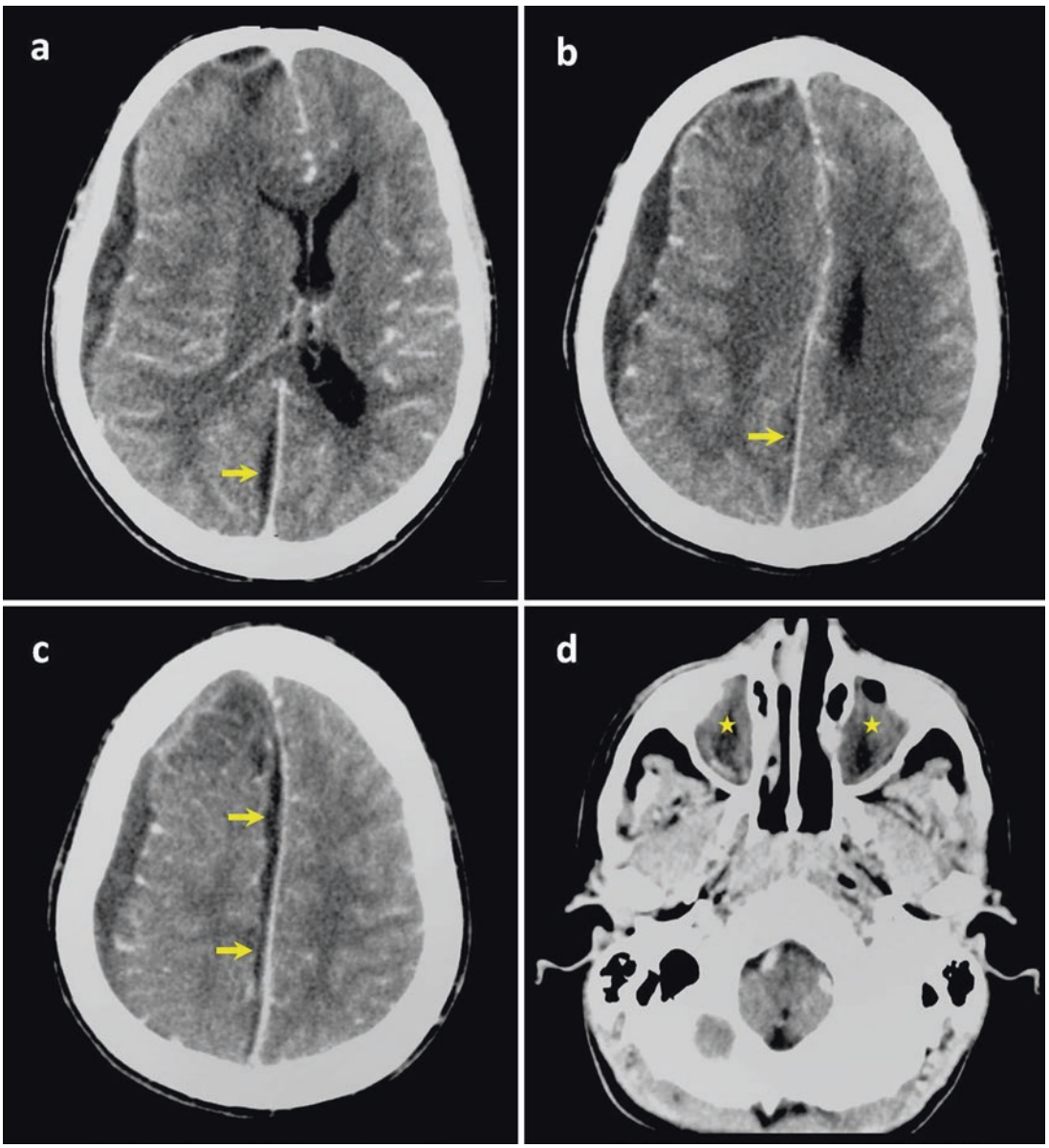


**Fig. 6.12** Case 6.5. (**a**–**d**) Axial cranial post-contrast CT scan showing extensive frontoparietal and interhemispheric subdural empyemas on the right side (*arrows*). Note bilateral maxillary sinusitis (*stars*)

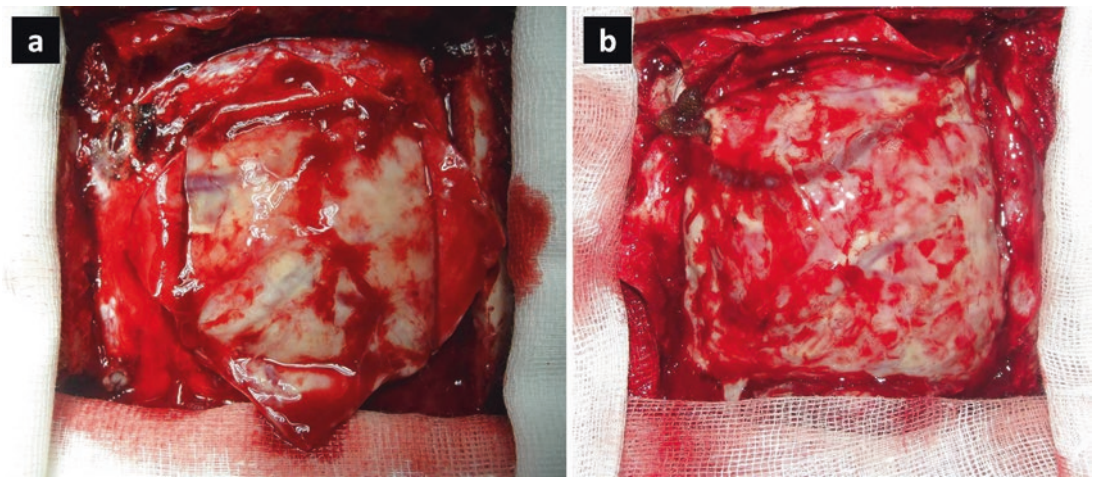


**Fig. 6.13** Case 6.5. Operative view of the subdural empyema (convexity) following dural opening (**a**) and a procedure of irrigation and gentle débridement (**b**)

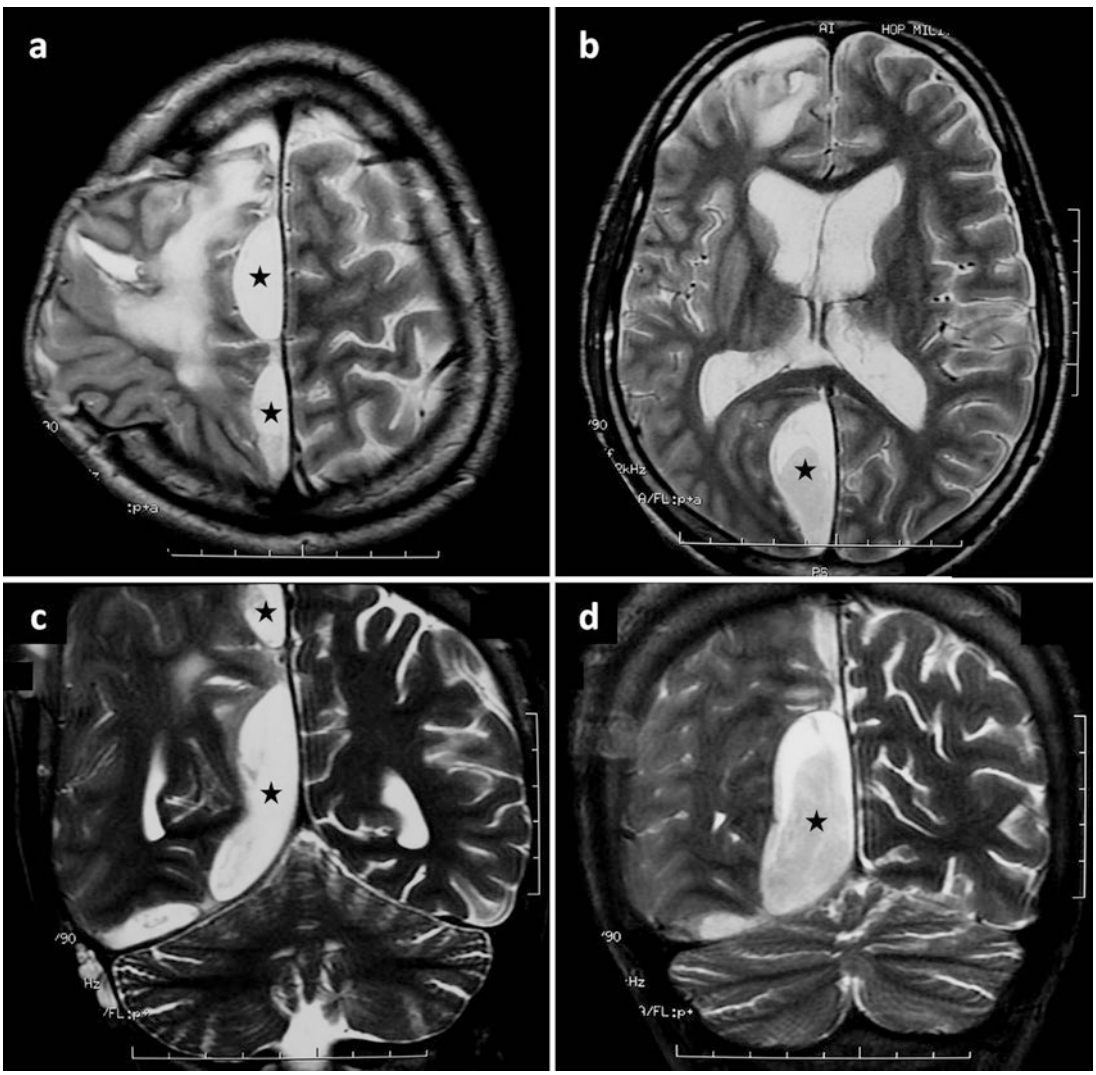


**Fig. 6.14** Case 6.6. Axial (**a**, **b**) and coronal (**c**, **d**) cranial T2-weighted images. This patient was operated on 2 weeks previously for a frontoparietal subdural empyema (craniectomy) on the right side. He then developed multiple interhemispheric subdural empyemas (*stars*)

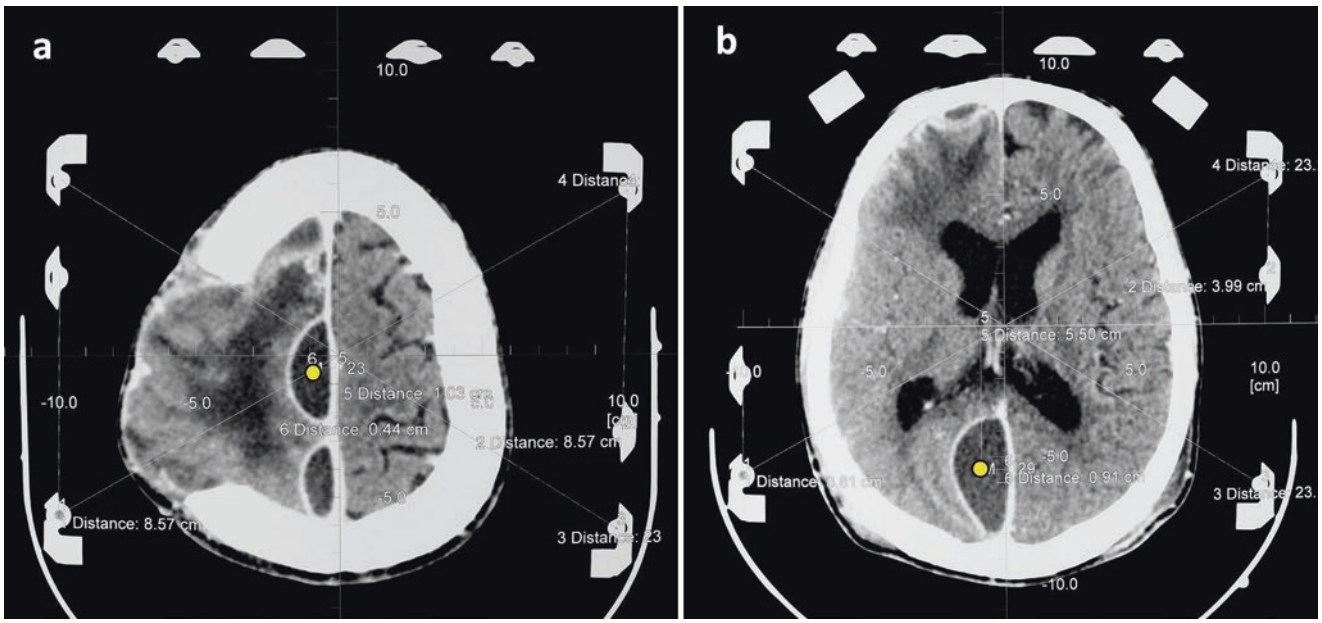


**Fig. 6.15** Case 6.6. (**a**, **b**), Axial cranial CT scans with contrast injection during preplanning procedure of stereotactic-guided aspiration/drainage of the subdural empyemas in the same patient (Radionics Cosman Roberts Wells CRW* frame). *Yellow dots* represent the targets

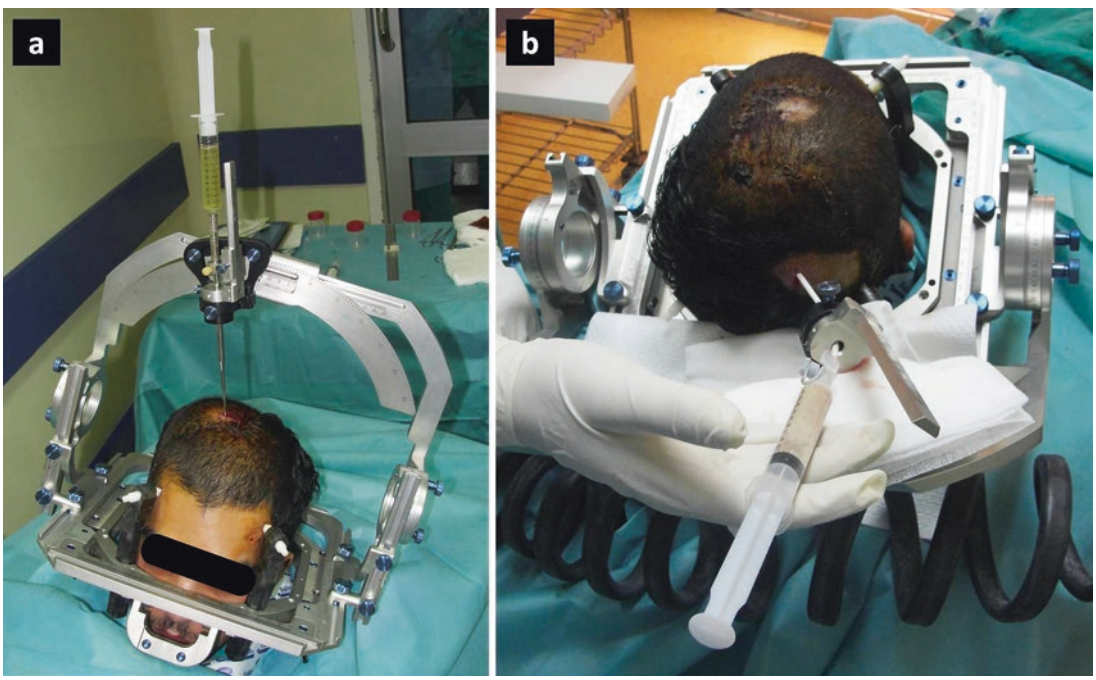


**Fig. 6.16** Case 6.6. Intraoperative pictures showing a stereotactic aspiration of both anterior (**a**) and posterior (**b**) interhemispheric subdural empyemas on the right side. Note the purulent material filling the syringe after a moderate aspiration under local anesthesia. A small, soft silicone tube was left in situ in the right parietal region (**b**)

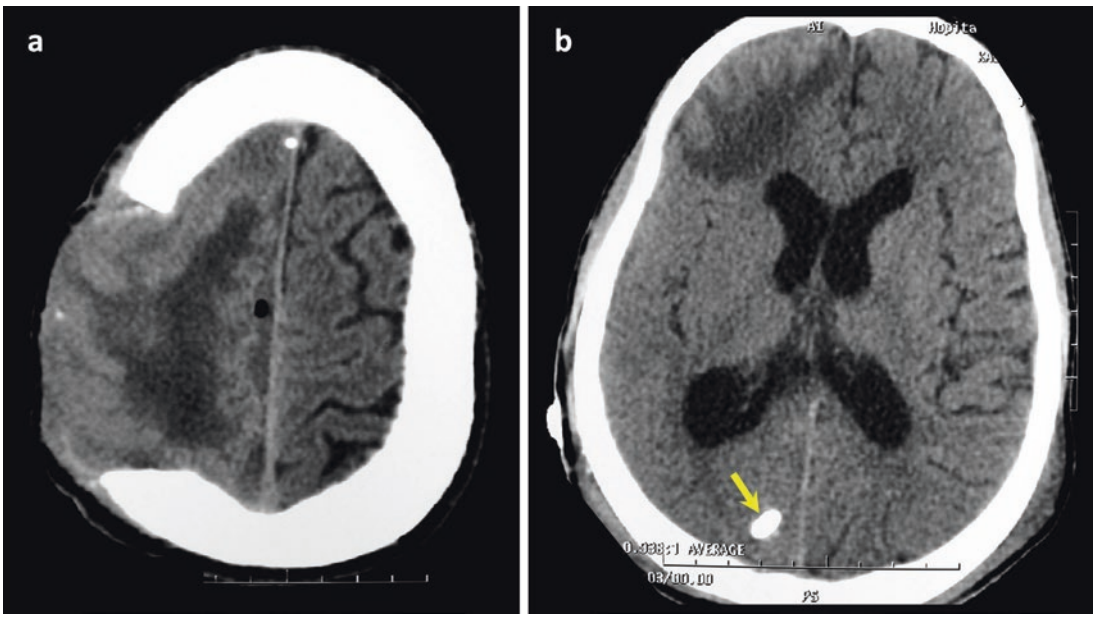


**Fig. 6.17** Case 6.6. (**a**, **b**) Postoperative cranial CT scans showing a good improvement of the suppurative collections. The drain is seen in the control CT scan (*arrow*) (**b**)

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# Posttraumatic Meningitis

7

Dural tear and cerebrospinal fluid (CSF) leakage are major risk factors for posttraumatic meningitis. Fractures involving the skull base and the paranasal sinuses have the potential for the subsequent development of a transient or persistent CSF leak. Penetrating head injuries with dural laceration are also associated with increased chances of meningitis. Classically, we distinguish early from late meningitis, with the highest incidence within 2 weeks following injury. Clinical presentations vary; most patients present with headache, fever, stiff neck, and change in mental status. Careful interrogation and clinical assessment are important for detecting a CSF fistula. CSF gram stain and cultures are essential in determining the cause of meningitis, although other laboratory values and imaging results also may be helpful. Appropriate intravenous antibiotics are started based on CSF culture and bacterial sensitivity results. Management of persistent traumatic CSF leak requires conservative measures, spinal drainage, and even surgical repair (open intracranial surgery or an endoscopic procedure) using multilayer closure. If treated promptly and vigorously, traumatic meningitis may resolve without sequelae, but if neglected, this infective complication is a significant cause of morbidity and death among patients with head injuries.

## Epidemiology and Etiology

Meningitis following traumatic head injury is unusual, with an incidence of less than 2%. This incidence is greatly increased when there is an associated CSF leak. Dural tear and CSF leakage are major risk factors for meningitis because of the passage of causative pathogens into the subarachnoid space.

Fractures involving the skull base, the orbits, and/or the paranasal sinuses have the potential for the subsequent development of a transient or persistent CSF leak. Penetrating (open) head injuries with dural laceration are also associated with increased chances of meningitis. Penetrating injury can be caused by high-velocity projectiles or by objects of lower velocity.

Meningitis can present immediately following head trauma, or it can be delayed for many months or years. In most cases, however, the diagnosis of meningitis is made within 2 weeks of the injury.

## Clinical Presentations

Signs and symptoms in traumatic meningitis vary, but most patients present with headache, fever, photophobia, neck stiffness, and change in mental status (deterioration in consciousness). Careful interrogation and clinical assessment are important for detecting a CSF fistula, indicated by the presence of active clear discharge from the nose (rhinorrhea) or ear (otorrhea) (Figs. 7.1 and 7.2).

In patients with recent head trauma, the mixture of blood and CSF may make the diagnosis difficult. CSF mixed with blood forms a “halo sign” when dripped on filter paper. However, the halo sign may be associated with false-positive results. Valsalva maneuver is a positive indicator.

In patients with severe head injuries or those with impaired consciousness, the appreciation of CSF leak and even the development of meningitis could be more challenging (Figs. 7.3 and 7.4).

## Imaging Features

Axial CT scan will often show pneumocephalus, but it is not highly sensitive in identifying the fistula site (Figs. 7.5, 7.6, and 7.7). Thin-slice CT imaging with sagittal and coronal reconstruction will often show the site of the bony defect. MRI does not delineate bony defects well within the anterior or middle cranial fossa, but MR cisternography is a highly effective test for identifying a specific CSF leak site. It often shows a T2-hyperintense column of CSF communicating between the subarachnoid space and sinuses.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_7


**Fig. 7.1** Case 7.1. (**a**) Anterior cerebrospinal fluid (*CSF*) rhinorrhea following a craniofacial injury (*arrow*). (**b**) Hemorrhagic CSF was collected and transported to the laboratories in a sterile universal container

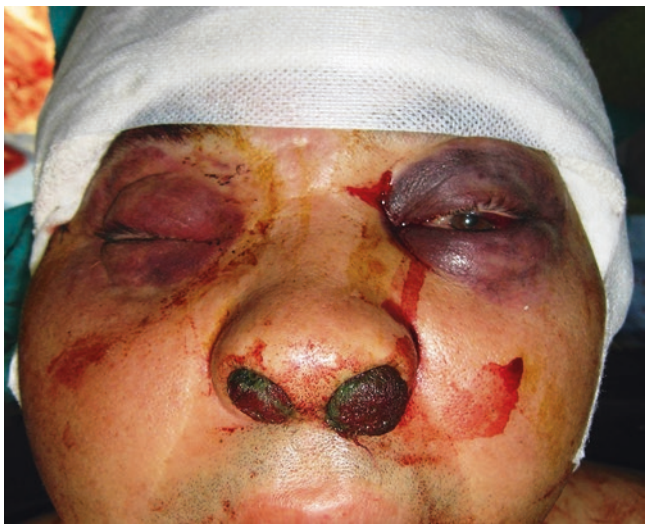

**Fig. 7.3** Case 7.2. Clinical photograph of patient with blunt craniofacial trauma. Note the periorbital ecchymosis, facial edema, and anterior nasal packing due to important epistaxis

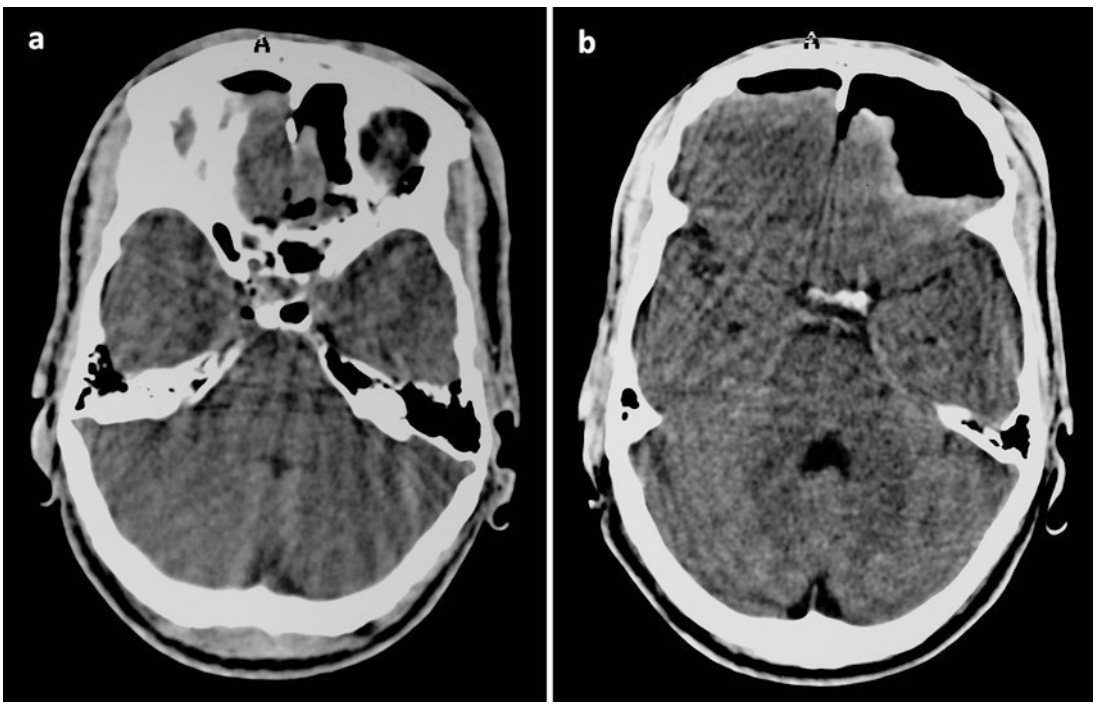


**Fig. 7.2** Case 7.1. (**a**, **b**), Axial cranial CT scans showing the presence of air in the anterior intracranial cavity (posttraumatic pneumocephalus)

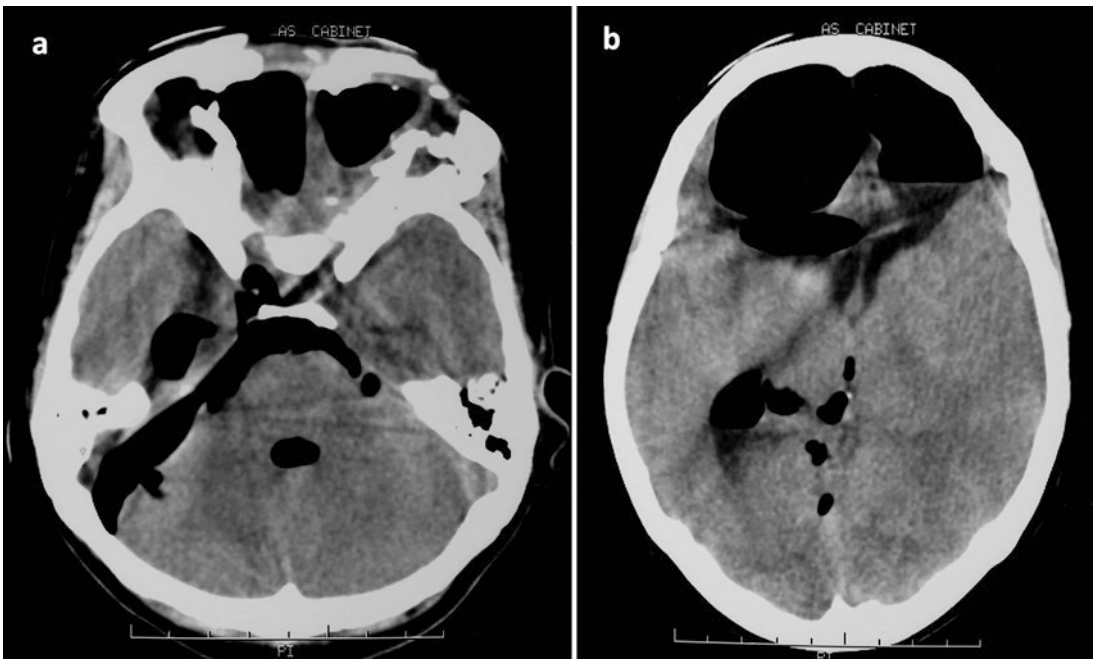


**Fig. 7.4** Case 7.2. (**a**, **b**), Axial CT scans revealing multiple cranio-orbital fractures and extensive pneumocephalus. This patient developed a post-traumatic bacterial meningitis

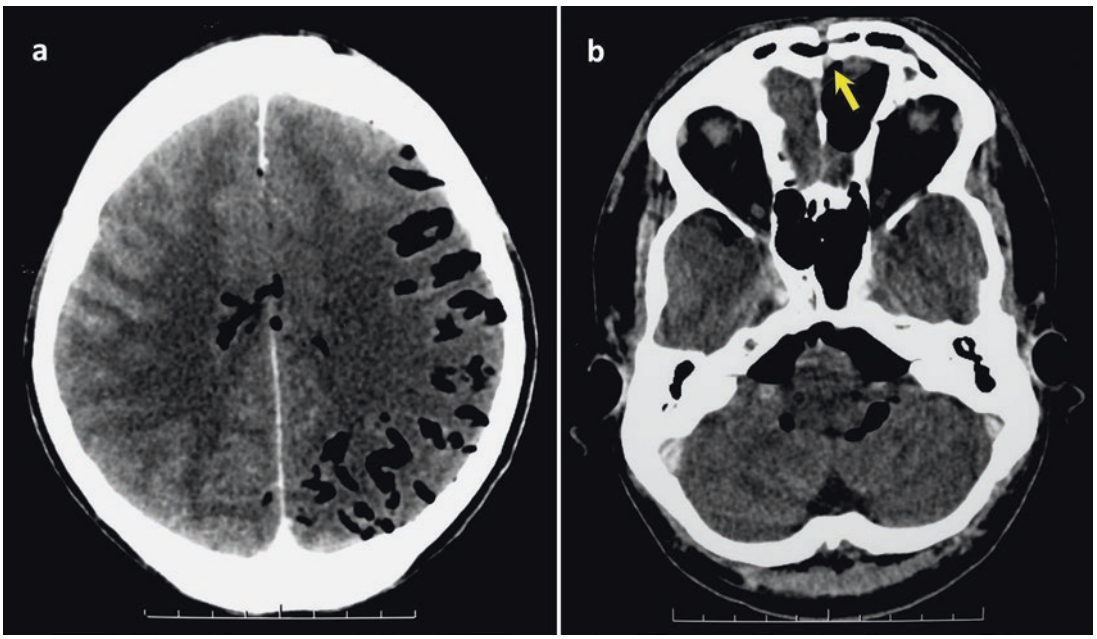


**Fig. 7.5** (**a**, **b**), Noncontrast axial CT scans in a patient with a bacterial meningitis following head injury. Pneumocephalus can be seen near the superior sagittal sinus and along the convexity. Note the frontal sinus fractures (*arrow*)

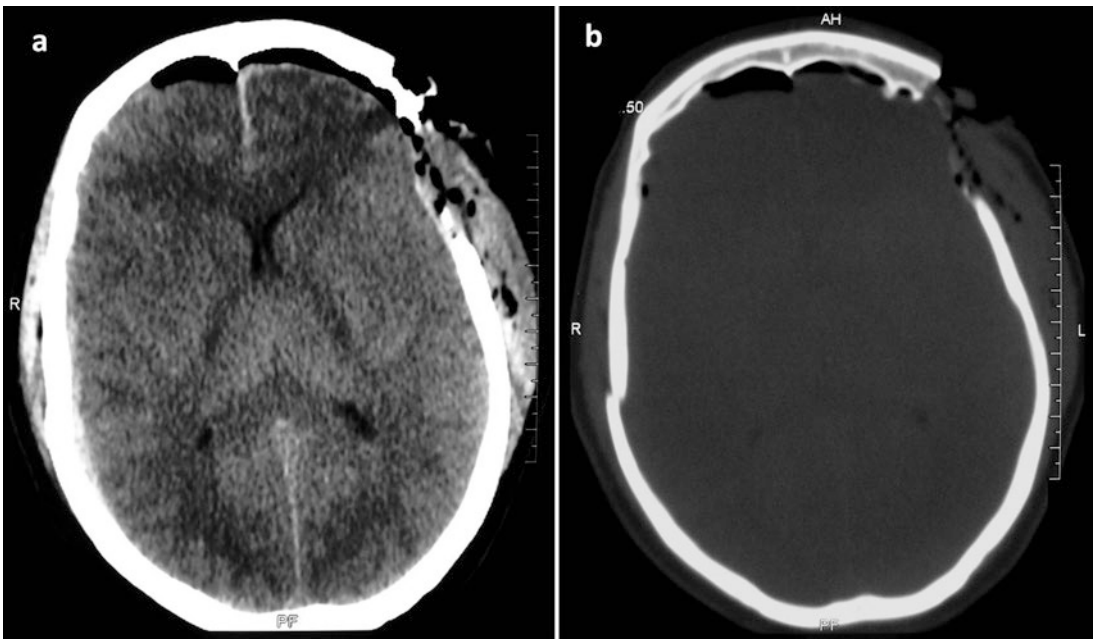


**Fig. 7.6** Axial cranial CT scan on parenchymatous (**a**) and bone (**b**) windows revealing an open skull fracture in the left frontal area. There is a posttraumatic brain edema with a pneumocephalus in the frontal convexity

CT cisternography is also a valuable modality for identifying the site of a CSF fistula. It often shows contrast extravasation through a bony defect and pooling in a paranasal sinus. Radionuclide cisternography can be used to confirm the presence of a skull base CSF leak and can aid in its localization.

## Laboratory Findings

Lumbar puncture with CSF analysis often shows increased protein and decreased glucose level. Identifying the pathogen responsible using a CSF gram stain and culture is essential in determining the cause of meningitis. *Streptococcus pneumoniae* is the most frequent offending microorganism liable for posttraumatic meningitis (50–70%). Other bacteria include *Staphylococcus aureus* and streptococcus species and gram-negative bacteria such as *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Haemophilus influenzae*, and *Neisseria meningitidis*. Polymicrobial infections involving anaerobes are common following gross contamination.

Negative CSF cultures are not rare and may be due in part to prophylactic antibiotics. Blood cultures may be helpful for documenting the existence of *S. pneumoniae*.

To verify the presence of a CSF leak, beta-2 transferrin assay and beta-trace protein are the best lab tests for identifying the presence of CSF in nasal or ear fluids.

## Treatment Options

Medical therapy should include broad-spectrum antibiotic drugs against gram-positive cocci and gram-negative bacilli with a good CSF penetration. In general, initial antibiotics include a third-generation cephalosporin combined with vancomycin. The antibiotic regimen is then tailored to the specific pathogen, once antibiotic sensitivity results are established. Antibiotic drugs must be sustained for 1–2 weeks after the CSF is sterilized.

Management of persistent traumatic CSF leak requires bed rest, hydration, medication (perhaps corticosteroids or acetazolamide), lumbar taps, spinal drainage, and even surgical repair. The defect site can be repaired either by open intracranial surgery or an endoscopic procedure of the skull base. Intraoperative use of intrathecal fluorescein can facilitate the recognition and confirmation of the fistula location. During surgery, graft material is placed to close the hole in the skull base (multilayer closure) (Figs. 7.8 and 7.9).

In some patients, CSF leak may resolve spontaneously or following treatment of meningitis, but the potential for later CSF leak and/or recurrent meningitis is increased in these cases.

Prophylactic antibiotics are not effective in preventing meningitis and may select for more virulent and more resistant microorganisms if an infection occurs.

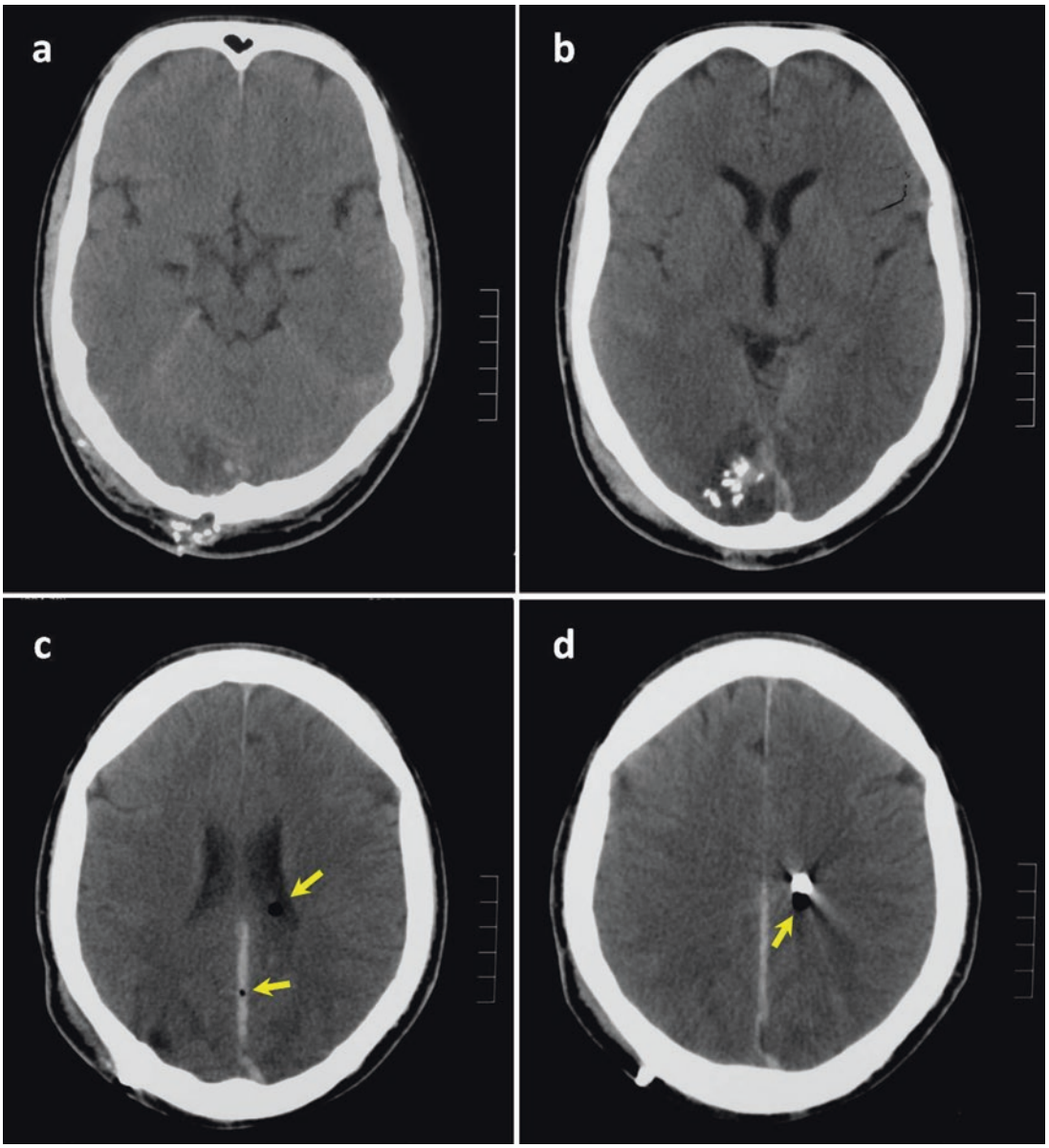


**Fig. 7.7** (**a–d**) Axial cranial CT scans in a patient following a penetrating craniocerebral injury (open head injury of the right occipital region). Note the presence of air in the brain parenchyma (*arrows*)

## Outcomes

Posttraumatic meningitis may have potential complications such as pneumonia, anosmia, seizure, mental retardation, hydrocephalus, and other important neurologic deficits. Mortality is not rare.

Patients with recurrent meningitis must be evaluated for the presence of abnormal communication with the intracranial or intraspinal content. Congenital anatomical defect should always be considered.

Prognosis depends on the state of general health, the concomitant intracranial and systemic damage, the delay in diagnosis, the patient's age, and the response to therapy.

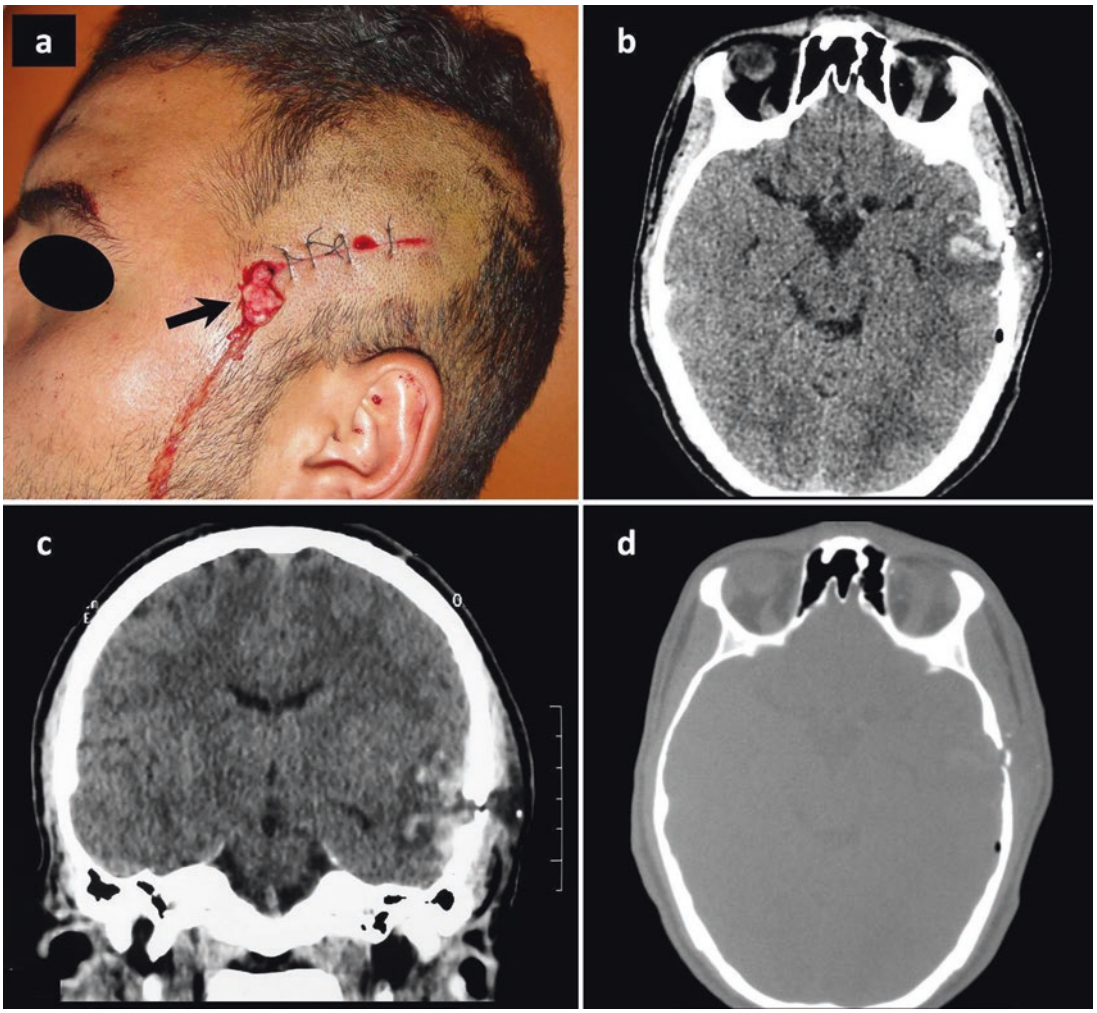


**Fig. 7.8** Case 7.3. Left temporal craniocerebral wound after assault with an axe handle. Clinical photograph (**a**) shows the temporal wound with extracranial extrusion of brain matter (*arrow*). Axial (**b**) and coronal (**c**) CT scans on parenchymatous windows. Axial CT scan on bone window (**d**)

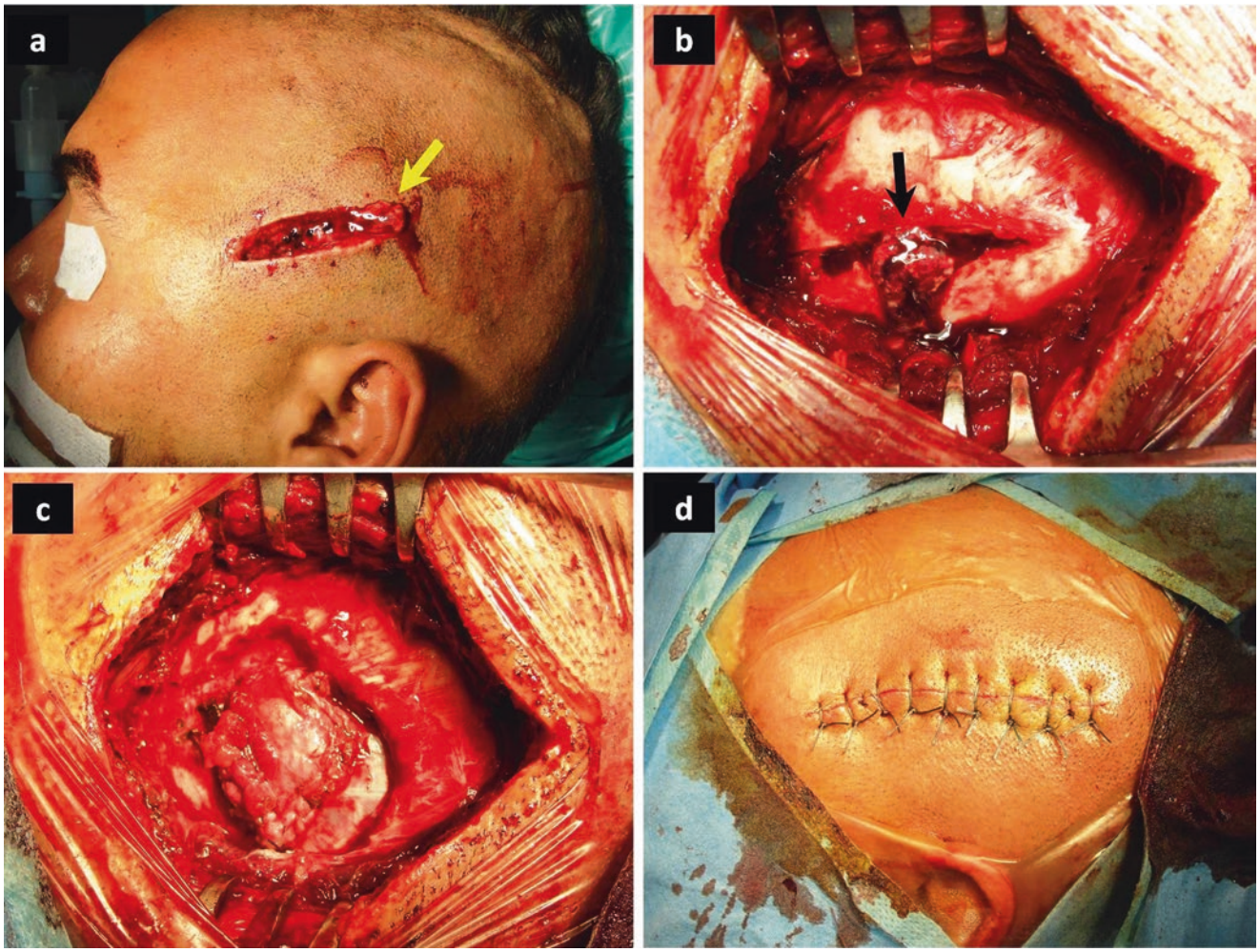


**Fig. 7.9** Case 7.3. Operative views. The wound was cleaned, enlarged, and opened (**a**, **b**). Surgery required craniectomy, debridement, evacuation of hematomas, and removal of bone fragments, obtaining hemostasis and watertight dural closure with epicranial graft (**c**, **d**)

## Suggested Reading


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# Brain Abscesses

# 8

Brain abscess is a focal purulent collection in the cerebrum, representing a serious infection of the central nervous system. The brain parenchyma may be infected by various microorganisms, principally bacteria. Infective pathogens may reach the brain in various ways, but sometimes the origin remains unknown. Most of the clinical signs are due not to the systemic manifestations of infection but to the size and location of a space-occupying lesion within the brain parenchyma and the virulence of the pathogen. Biological findings must always be interpreted in combination with clinical and imaging data. CT scans and MRI studies are important tools in the initial diagnosis and in the monitoring of treatment results. The spectrum of treatment goes from antibiotherapy alone to its combination with simple stereotactic drainage to invasive neurosurgical procedures. At present, good clinical recovery is predictable for many patients, and the mortality rate is increasingly low, but the prognosis depends on the patient's general health; the number, size, and location of the suppurative collections; the delay in diagnosis; and the response to therapy.

## Epidemiology and Etiology

Brain abscess is an intracerebral suppurative collection that represents one of the most serious infections of the central nervous system. The disease usually presents as a mass lesion causing local neurologic deficits related to the region involved. Brain abscess affects males more often than females, more commonly at ages 20–40 years.

The most common source of cerebral abscess (especially in developing countries) is from contiguous spread. Paranasal purulent sinusitis, middle-ear/mastoid air sinus infection, and odontogenic infections are the most common vectors. In hematogenous spread, the lung and the heart are the most frequent origins (lung abscess, bronchiectasis, empyema, congenital cyanotic heart disease, pulmonary arteriovenous fistulas, or endocarditis). No source can be found in 25% of cases, considered cryptogenic. Intracranial suppurations following traumatic injuries (open cranial fracture or a penetrating injury) are uncommon. Brain abscess is a rare but well-known postoperative complication following neurosurgical procedures. (*See* Chap. 21.)

Immunocompromised states (HIV infection, malignancy, transplant recipients, neutropenic states) are an increasingly important predisposing factor for the development of intracranial infections.

## Clinical Presentations

The classic presentation of brain abscess is headache, confusion, focal or generalized seizures, nausea and vomiting, and focal motor, sensory, or speech disorders. Most of the clinical signs are due not to the systemic manifestations of infection but rather to the size and the location of a space-occupying lesion within the brain parenchyma and the virulence of the pathogen. Meningeal symptoms and signs may be present when the lesion reaches the brain cortex. Fever is not constantly present. The classic triad of headache, fever, and focal neurologic deficit is uncommon.

The course of the disease can be indolent, but sometimes it may be rapidly life-threatening. The clinical presentation of brain abscess in the immunocompromised patient may be overlooked because of the diminished inflammatory response.

Obviously, the past medical history should be checked for any predisposing factors. Otorhinolaryngologic and general clinical examinations should be performed.

## Imaging Features

Neuroimaging features depend on the stages of brain abscess formation (Fig. 8.1):

- Early cerebritis (0–3 days)
- Late cerebritis (4–9 days)



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_8

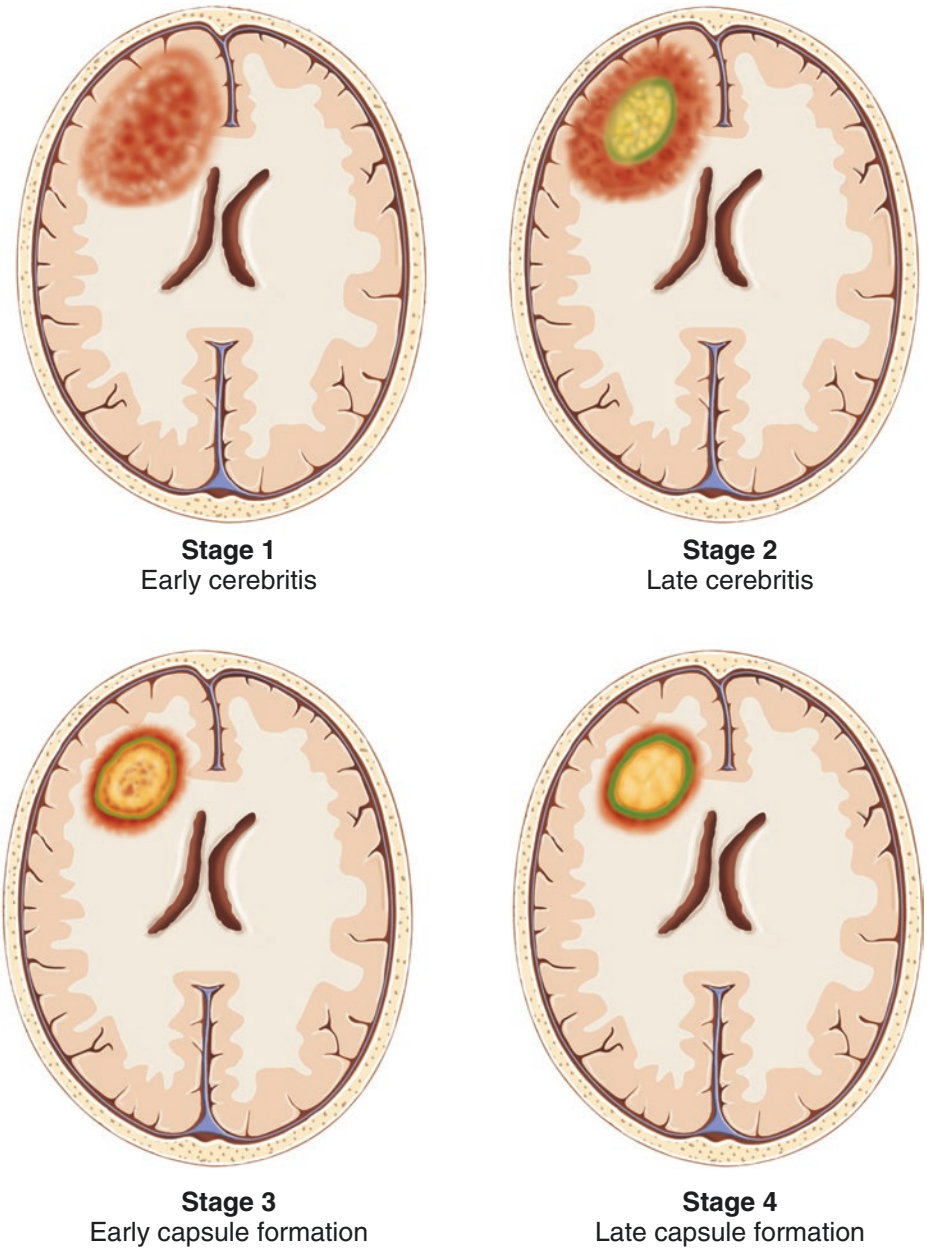


**Fig. 8.1** Different stages in the development of a brain abscess

- Early capsule (10–14 days)
- Late capsule (more than 2 weeks)

CT scanning will often demonstrate a homogenous, low-density mass with rim enhancement following contrast administration and extensive perifocal edema (Figs. 8.2, 8.3, and 8.4).

MRI is more sensitive. It typically shows a cystic mass with homogenous, low-intensity, or isointense signals on T1-weighted images and high-intensity signals on T2-weighted images, with rim enhancement after gadolinium injection (Figs. 8.5, 8.6, and 8.7).

Both CT scans and MRI can reliably distinguish brain abscess from other causes of ring-enhancing lesions. The mnemonic "MAGICAL DR" (Table 8.1) lists the most frequent ring-enhancing lesions seen on neuroimaging.

Restricted diffusion-weighted imaging (DWI) (bright signal), a diminished apparent diffusion coefficient (ADC) (low signal), and MR spectroscopy (MRS) (elevated lactate and glycerophosphocholine) often support the presence of brain abscess.

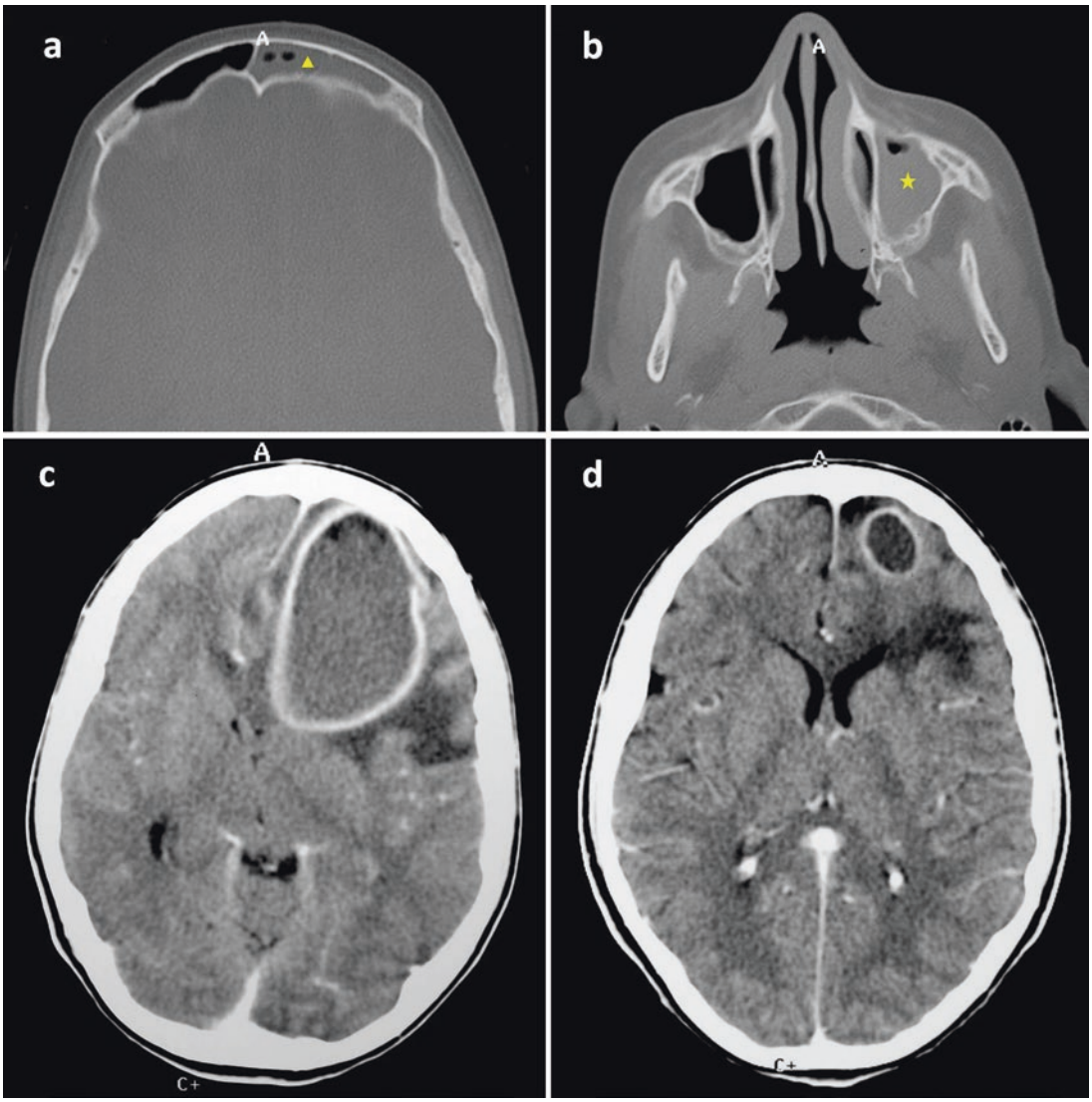


**Fig. 8.2** This 23-year-old man developed a large frontal brain abscess following inadequately treated paranasal sinusitis. Axial cranial CT scans demonstrated a frontal sinusitis (*triangle*) (**a**) and maxillary sinusitis (*star*) (**b**) on the *left* side. Preoperative (**c**) and postoperative (**d**) CT scans with contrast injection after surgical aspiration of the abscess

In addition, modern imaging may be useful in diagnosing the primary sources of craniofacial infections.

## Laboratory Findings

Brain abscess may be bacterial (aerobic or anaerobic bacteria), fungal, or mycobacterial and is often found to be polymicrobial. (*See* section "Treatment Options"). The most common individual pathogens are streptococci, staphylococci, *Bacteroides* species, and *Fusobacterium* species. These microorganisms reflect the bacterial species encountered in paranasal sinus infections. Sterile cultures are not uncommon.

Blood tests can sometimes demonstrate leukocytosis and abnormalities in some inflammatory parameters such as C-reactive protein level and erythrocyte sedimentation rate. Blood cultures are rarely positive.

Lumbar puncture is generally not recommended because of the possible occurrence of high intracranial pressure and the subsequent risk of brain herniation.

Histopathology may demonstrate necrosis and abscess wall, with infiltration by polymorphonuclear leucocytes or macrophages (Figs. 8.8, 8.9, and 8.10). Histopathology is also helpful for diagnosing some specific causative pathogens such as mycobacteria, fungi, and parasites.

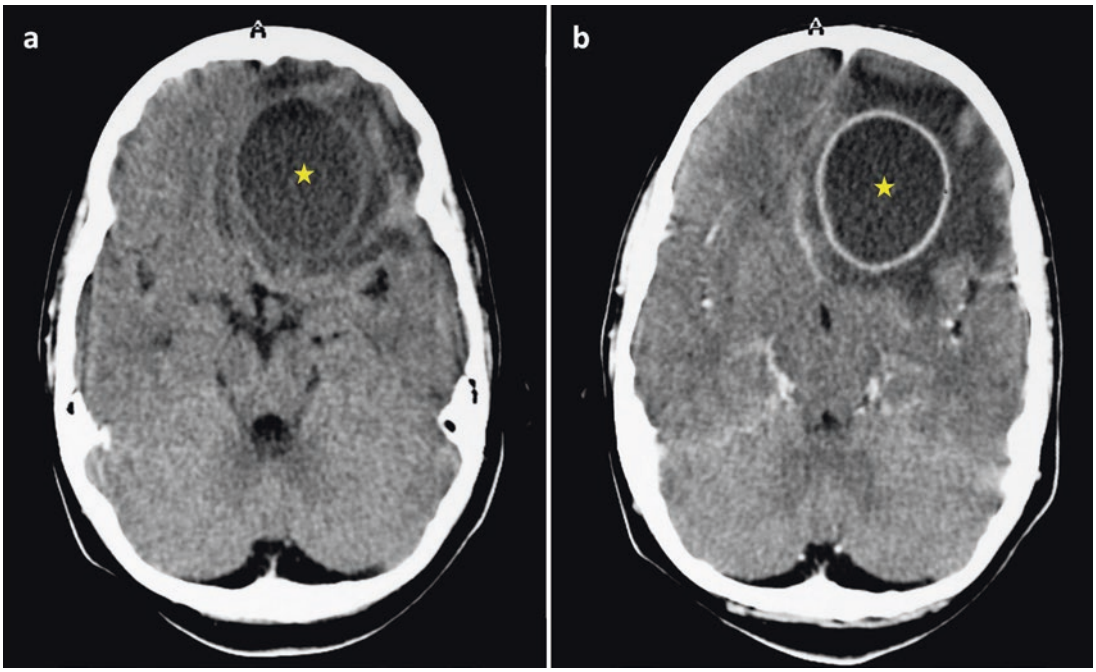


**Fig. 8.3** Axial cranial CT scan before (**a**) and after (**b**) contrast administration, showing a homogeneous, low-density frontal brain lesion (*star*) with rim enhancement and perifocal edema: a left frontal brain abscess

## Treatment Options

Medical management typically accompanies surgical treatment. Appropriate antibiotic choice should take into account likely infectious agents (aerobic and anaerobic cocci and bacilli) as well as adequate CSF penetration. The antibiotic regimen usually includes a third-generation cephalosporin and metronidazole (sometimes in addition to vancomycin or amikacin) for at least 8 weeks. Tubercular and fungal abscesses must be treated with the appropriate anti-infectious regimens.

Patients in the early stage of abscess formation (cerebritis) or with a small abscess can be treated with antibiotics alone (conservative management). Classic indications for aspiration are an abscess larger than 2 cm in diameter, a risk of intraventricular rupture, and failure of adequate antimicrobial therapy. Repeated aspiration is sometimes needed, as well as the placement of a soft catheter to allow ongoing drainage and instillation of intracavity antimicrobial drugs. Needle aspiration is more often being used instead of open surgical evacuation/excision, especially with the development of stereotactic procedures (frame or frameless neuronavigation systems). Brainstem and cerebellar abscesses are often indications for posterior fossa craniotomy decompression, however.

Open surgical excision may be the preferred initial surgical option if a foreign body or bone chips are present, if the abscess(es) are multiloculated, or if fistulous communication occurs. Intraoperative cultures for aerobic, anaerobic, and acid-fast bacilli and fungi should always be sent. The antibiotic treatment should be adapted to the specific pathogen once antimicrobial susceptibilities are established.

Use of corticosteroid has been controversial but is beneficial in patients with secondary brain edema resulting in raised intracranial pressure. Antiepileptic prophylaxis may be considered with abscesses close to epileptogenic areas.

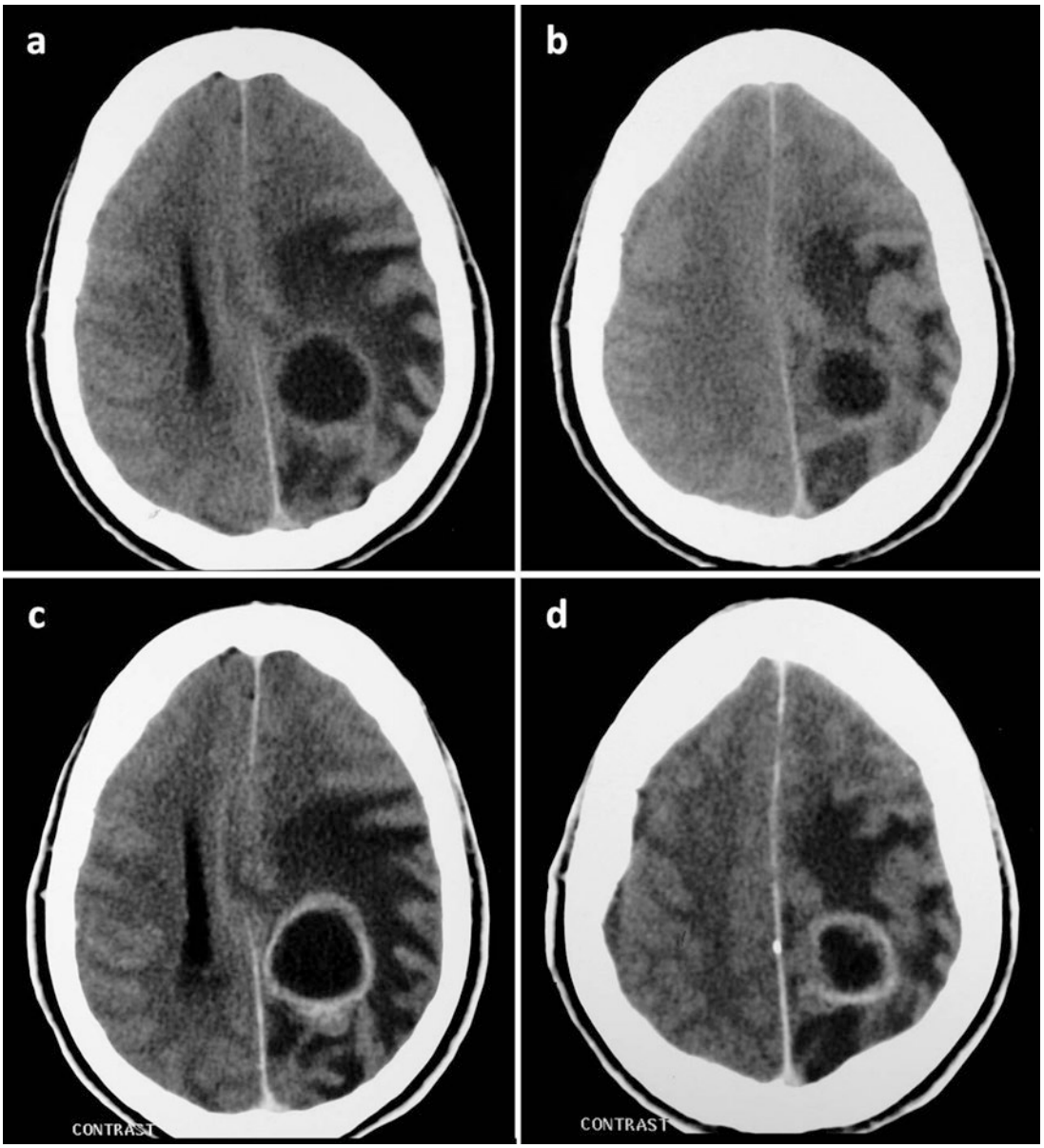


**Fig. 8.4** Axial cranial CT scans before (**a**, **b**) and after (**c**, **d**) contrast administration, showing a frontoparietal brain abscess on the *left* side, with a *thick* capsule

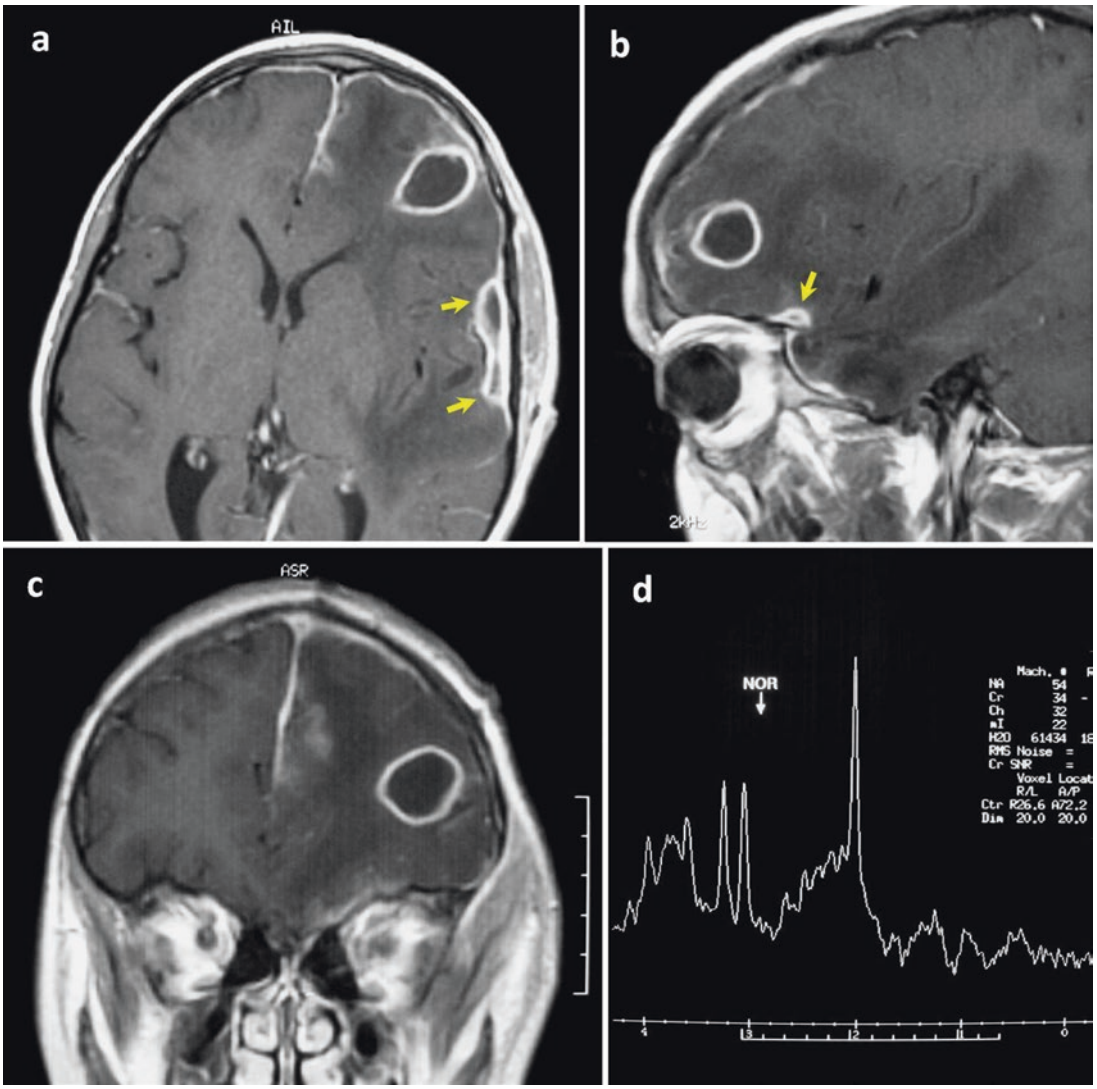


**Fig. 8.5** Cranial axial (**a**), sagittal (**b**), and coronal (**c**) post-gadolinium T1-weighted MRI and spectroscopy (**d**) showing a left frontal intraparenchymal abscess with rim enhancement. Note the associated subdural empyema (*arrows*)

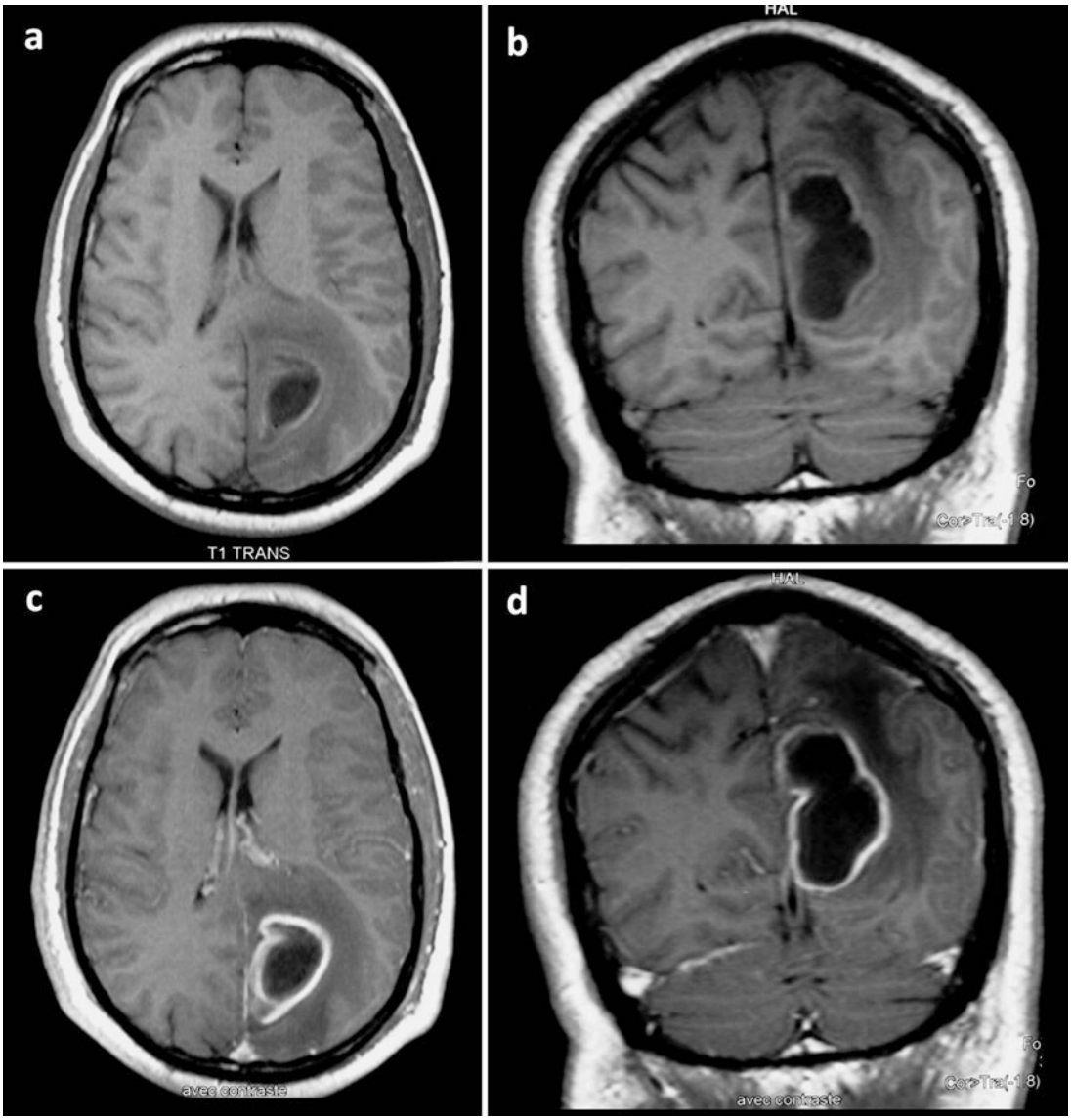


**Fig. 8.6** Case 8.1. Axial and coronal cranial T1-weighted MRI before (**a**, **b**) and after (**c**, **d**) gadolinium administration, revealing an irregular, cystic lesion located in the *left* parafalcine parieto-occipital area, with homogenous, low-intensity signal and rim enhancement

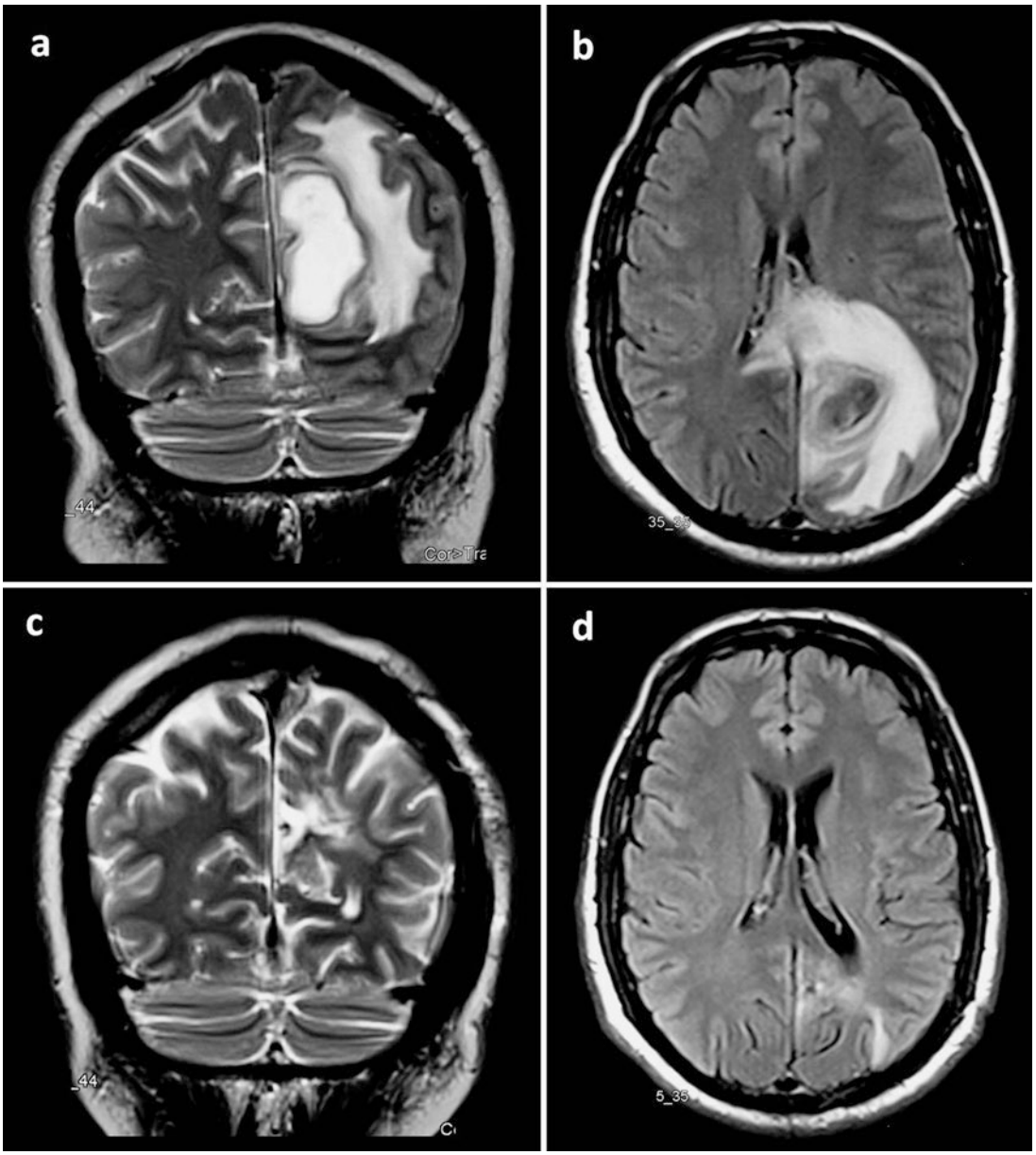


**Fig. 8.7** Case 8.1. (**a**, **b**) Coronal T2-weighted MRI and axial diffusion-weighted image before surgery, showing the intraparenchymal suppurative collection with extensive edema. (**c**, **d**) Control MRI following surgery

**Table 8.1** "MAGICAL DR" mnemonic for ring-enhancing brain lesions

| Initial | Cerebral ring-enhancing lesion |
|---|---|
| **M** | Metastasis |
| **A** | Abscess |
| **G** | Glioblastoma |
| **I** | Infarct (subacute phase) |
| **C** | Contusion |
| **A** | AIDS |
| **L** | Lymphoma |
| **D** | Demyelinating disease (tumefactive multiple sclerosis) |
| **R** | Radiation necrosis |
| | Resolving hematoma |

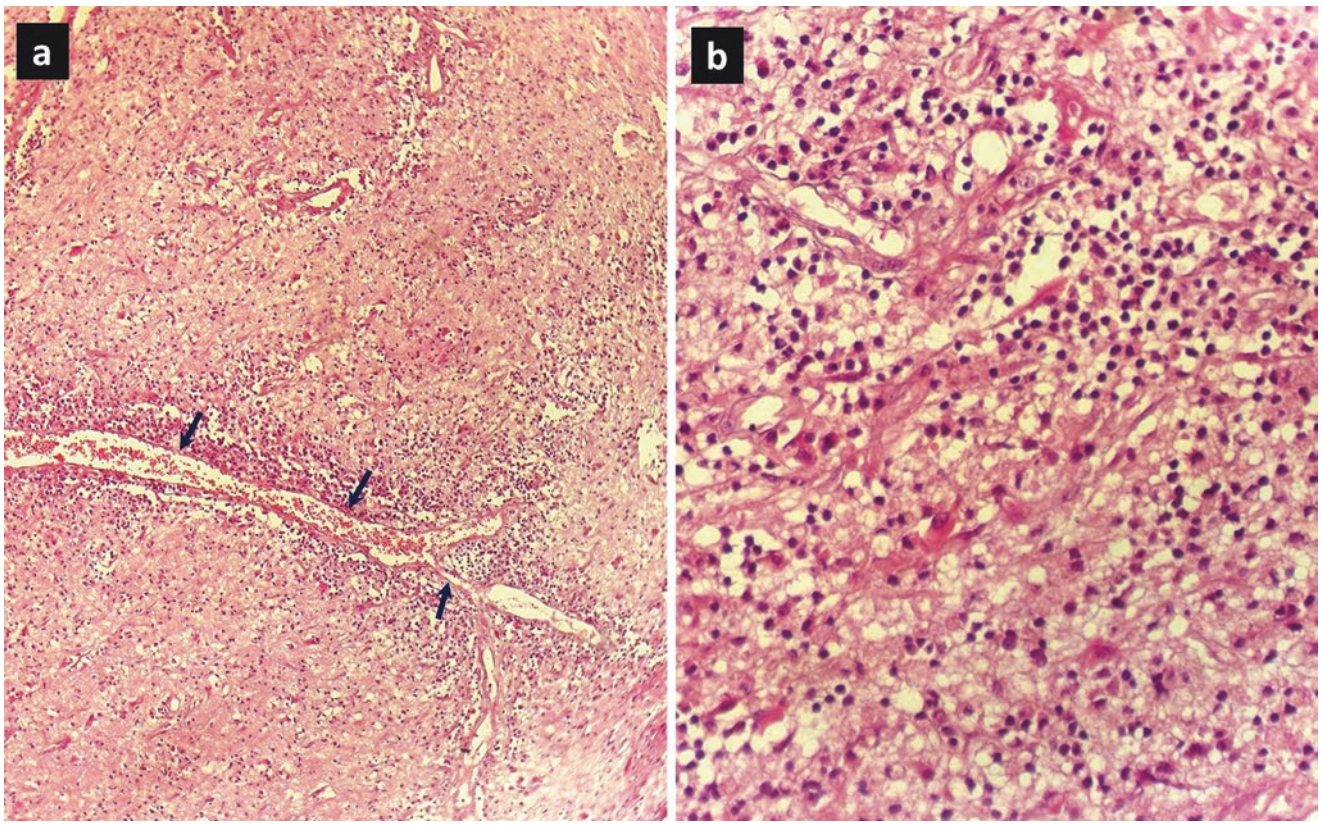


**Fig. 8.8** Case 8.1. Histopathologic features of the brain abscess, at medium-power (**a**) and high-power (**b**) magnifications. Subacute encephalitis with infiltration by polymorphonuclear leucocytes and macrophages. Note the vasculitis (*arrows*) (**a**) (hematoxylin–eosin staining)

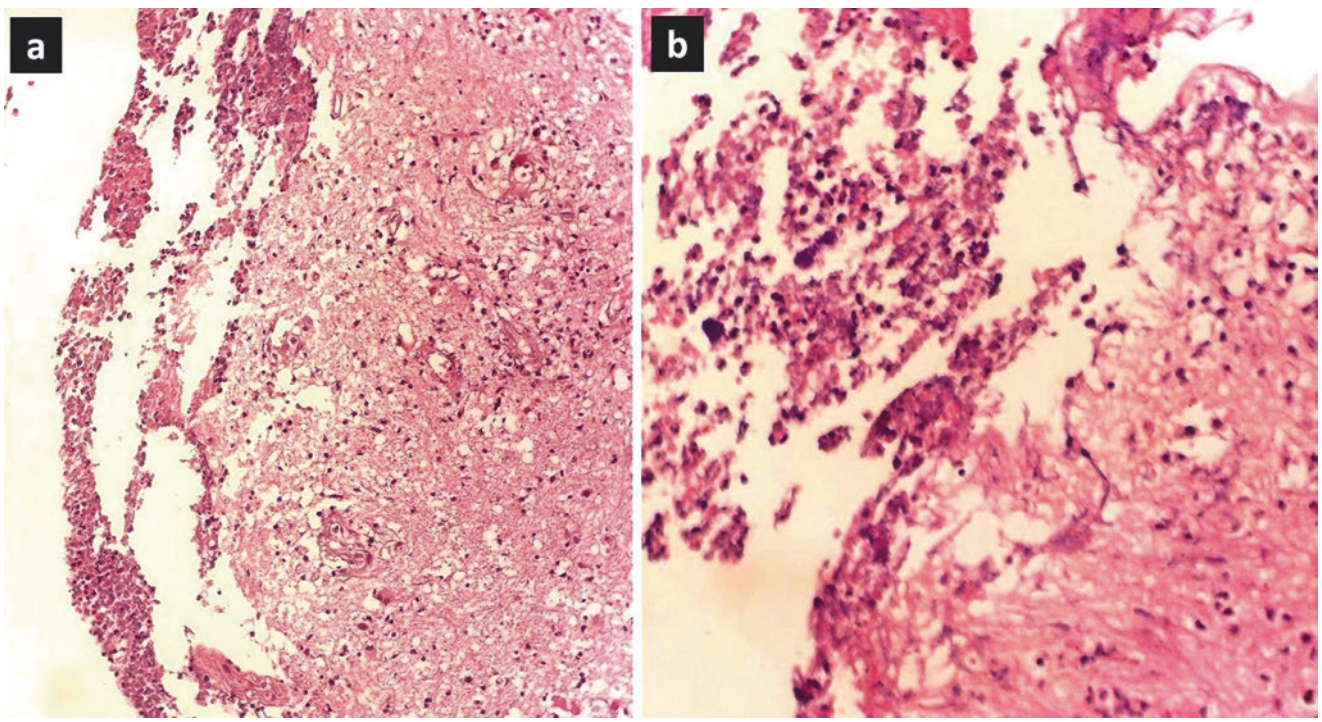


**Fig. 8.9** Case 8.1. Brain abscess. Photomicrograph of specimen showing areas of pus and necrotic debris surrounded by inflammatory infiltrates (encephalitis), at medium-power (**a**) and high-power (**b**) magnifications (hematoxylin–eosin staining)

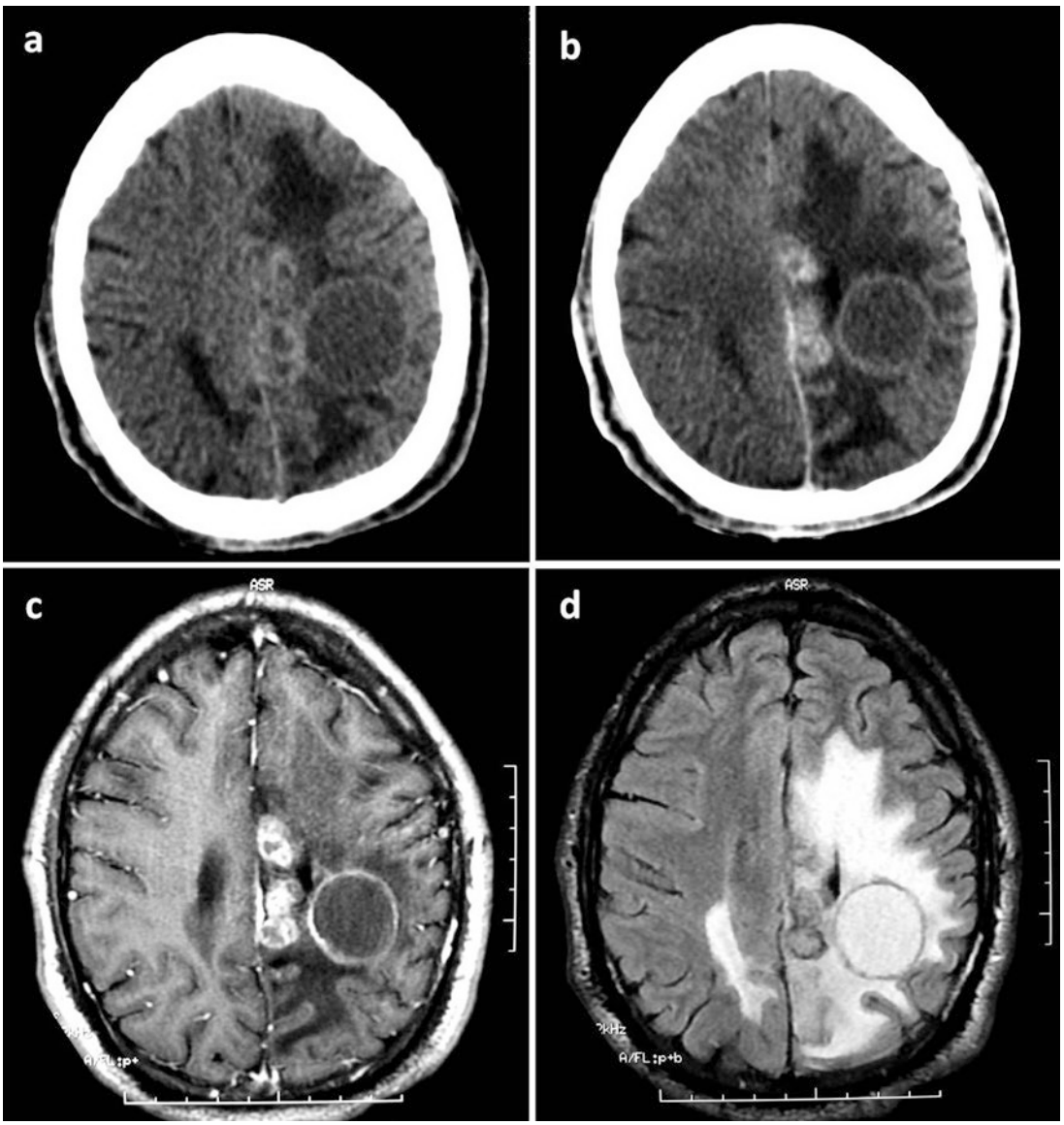


**Fig. 8.10** Axial CT scan before (**a**) and after (**b**) contrast injection, showing a left rolandic abscess with ring enhancement and adjacent granulomatous parafalcine lesions. The same lesion on T1-weighted MRI following gadolinium administration (**c**) and on FLAIR sequence (**d**)

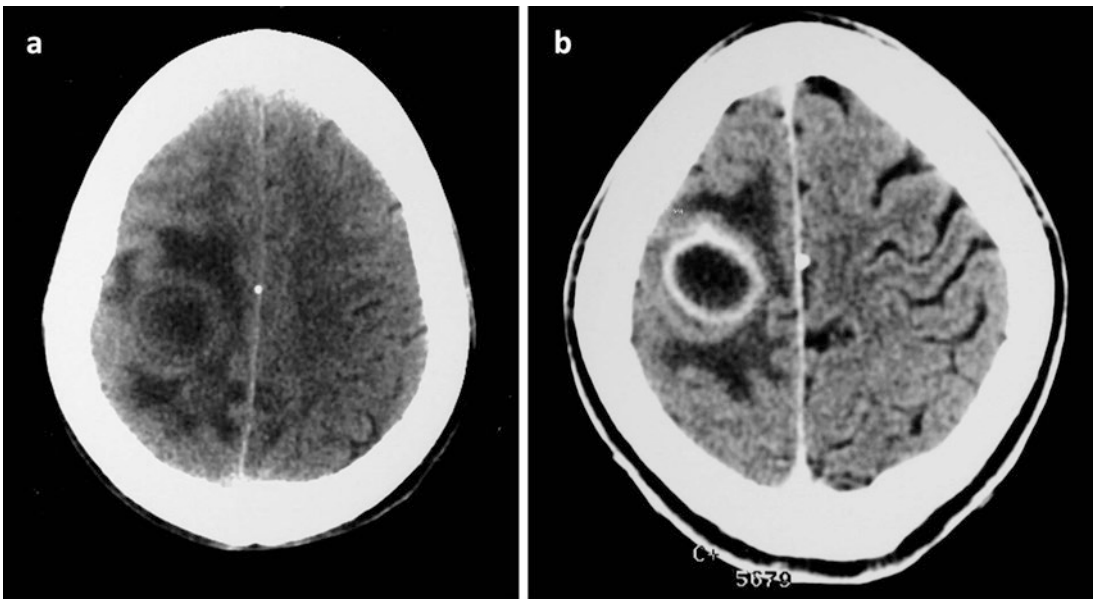


**Fig. 8.11** Case 8.2. Cranial axial CT scan before (**a**) and following (**b**) contrast injection, showing a frontal, intraparenchymal abscess located in the *right* rolandic area. Complete surgical excision was decided after failure of repeated stereotactic aspiration and adapted antibiotic therapy (recurrence)

## Outcomes

Close clinical, biological, and neuroimaging monitoring is important to ensure adequate response to treatment. Complications include seizures, ventriculitis (*see* Chap. 10), hydrocephalus, and recurrent abscesses. Recurrence is not rare (10 %); reasons include inadequate antibiotic therapy, failure to aspirate large abscesses, presence of a retained foreign body or dural fistula, and failure to eliminate primary sources of infection (Figs. 8.11 and 8.12).

At present, good clinical recovery is predictable for many patients, and the mortality rate is increasingly low. The prognosis depends on the general health and age of the patient; the number, size, and location of the suppurative collection; the delay in diagnosis; and the response to therapy. Common long-term sequels include seizures, focal neurologic deficits, and permanent alterations in mental status (Figs. 8.13, 8.14, 8.15, 8.16, 8.17, 8.18, 8.19, 8.20, 8.21, and 8.22).

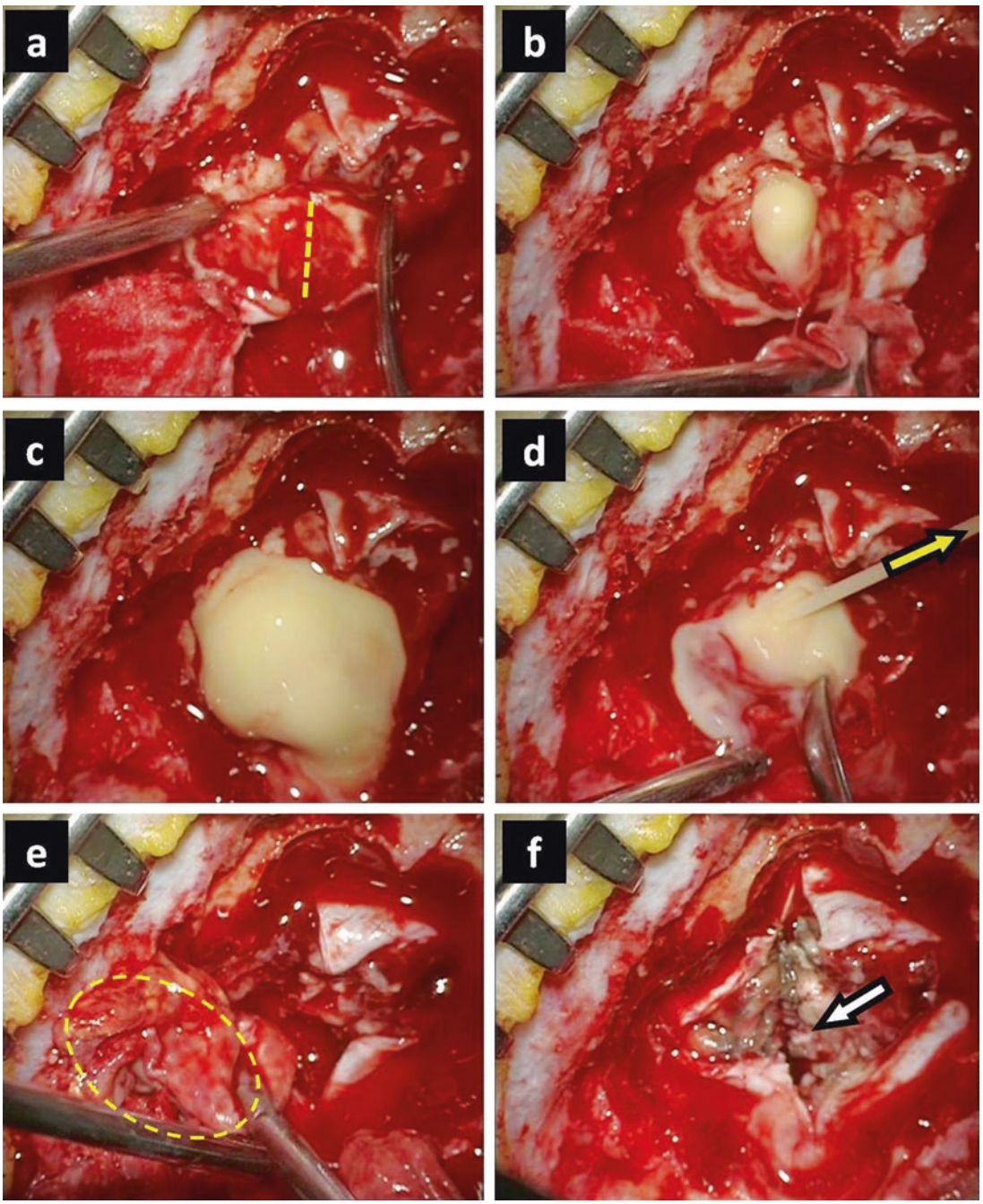


**Fig. 8.12** Case 8.2. Open surgical excision of the brain abscess. Operative views step by step. (**a**) After opening the dura, a corticectomy is performed (*dotted line*). (**b**, **c**) Release of the purulent content. (**d**) Needle aspiration (*arrow*) to obtain material for culture and to decompress the mass. (**e**) Excision of the capsule: following circumferential dissection, the abscess capsule is elevated gently and excised (*oval dotted line*) from the surrounding brain parenchyma. (**f**) Final appearance of the residual cerebral cavity (*arrow*) after gentle hemostasis

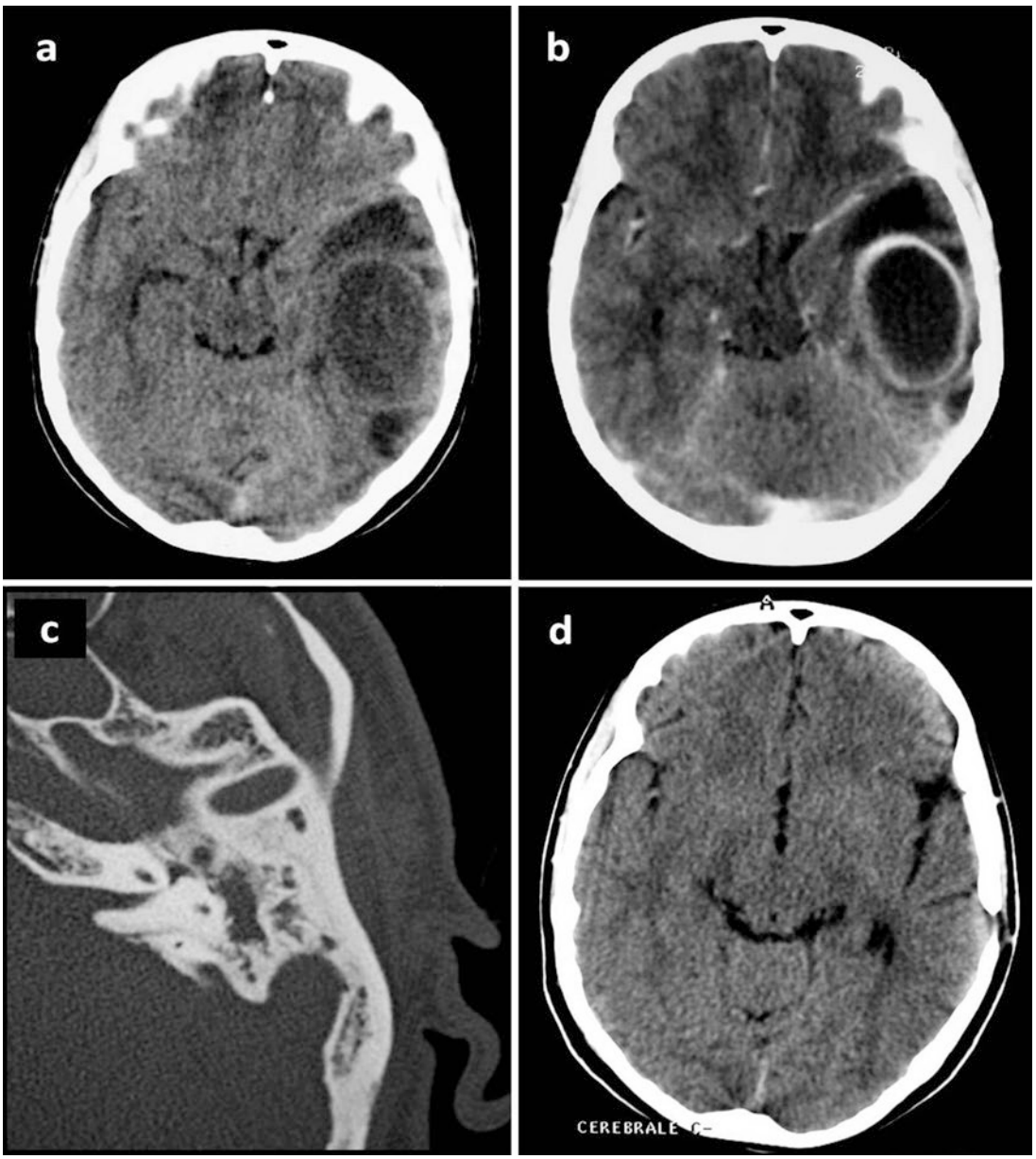


**Fig. 8.13** Left temporal brain abscess with ipsilateral otitis media. Axial CT scan without (**a**) and with (**b**) contrast administration. Axial petrous bone CT scan (**c**) showing the chronic otitis media. Postoperative post-contrast CT scan 1 month later (**d**) shows complete resolution of the abscess

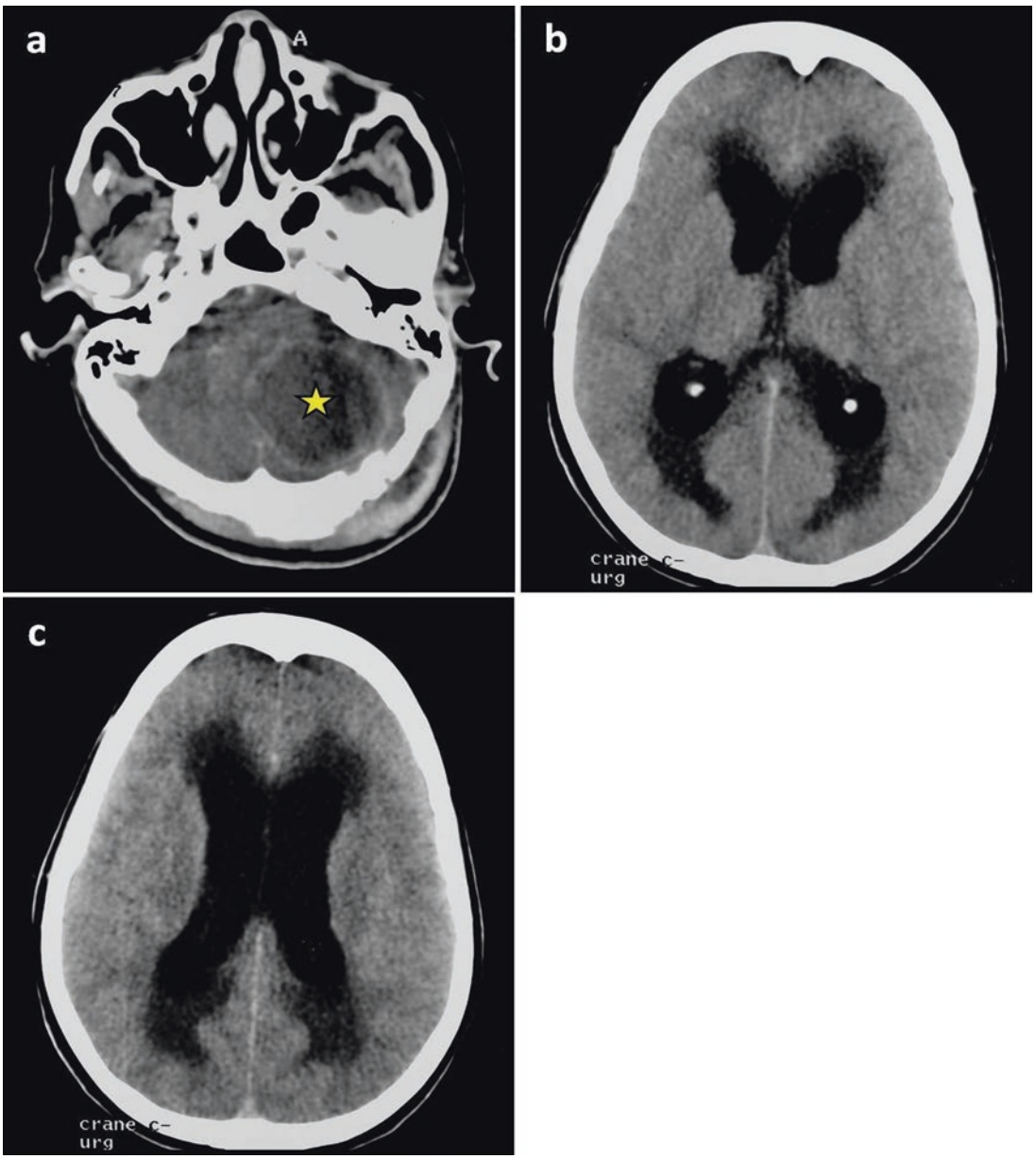


**Fig. 8.14** Axial cranial CT scan with (**a**) and without (**b**, **c**) contrast administration shows a left hemispheric cerebellar abscess (*star*) with fourth ventricular compression and supratentorial acute hydrocephalus. Note the *thin* capsule enhancement around the abscess formation (**a**)

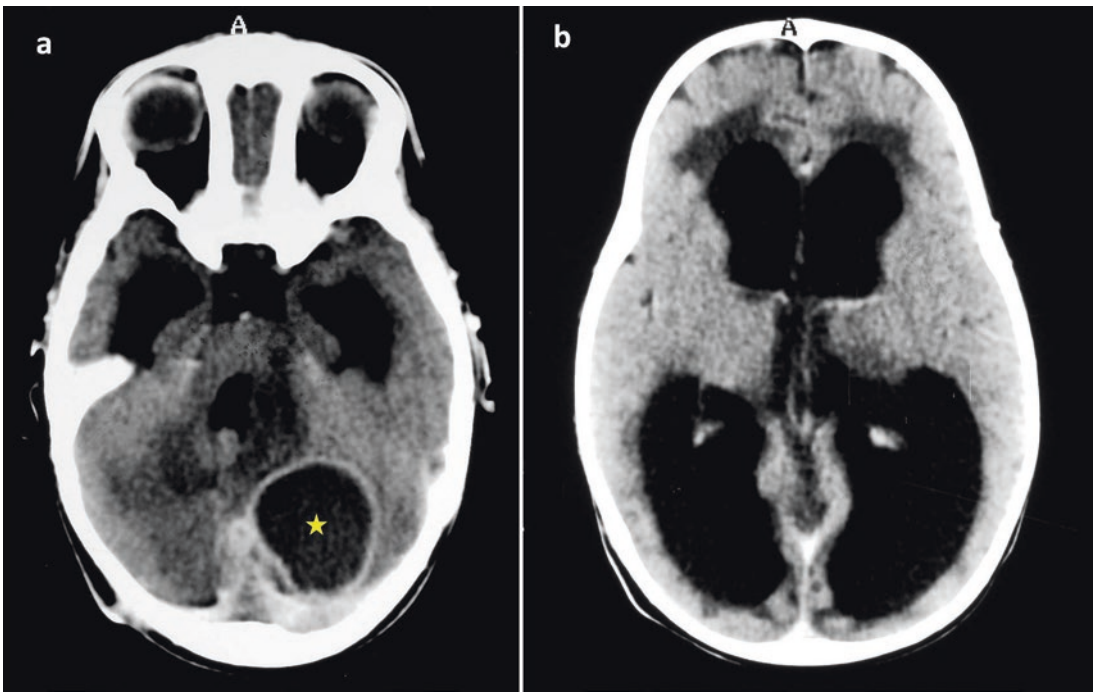


**Fig. 8.15** Axial cranial CT scan after contrast injection **a**, **b** shows posterior fossa abscess in the *left* hemispheric cerebellar area with acute hydrocephalus in a 1-year-old baby

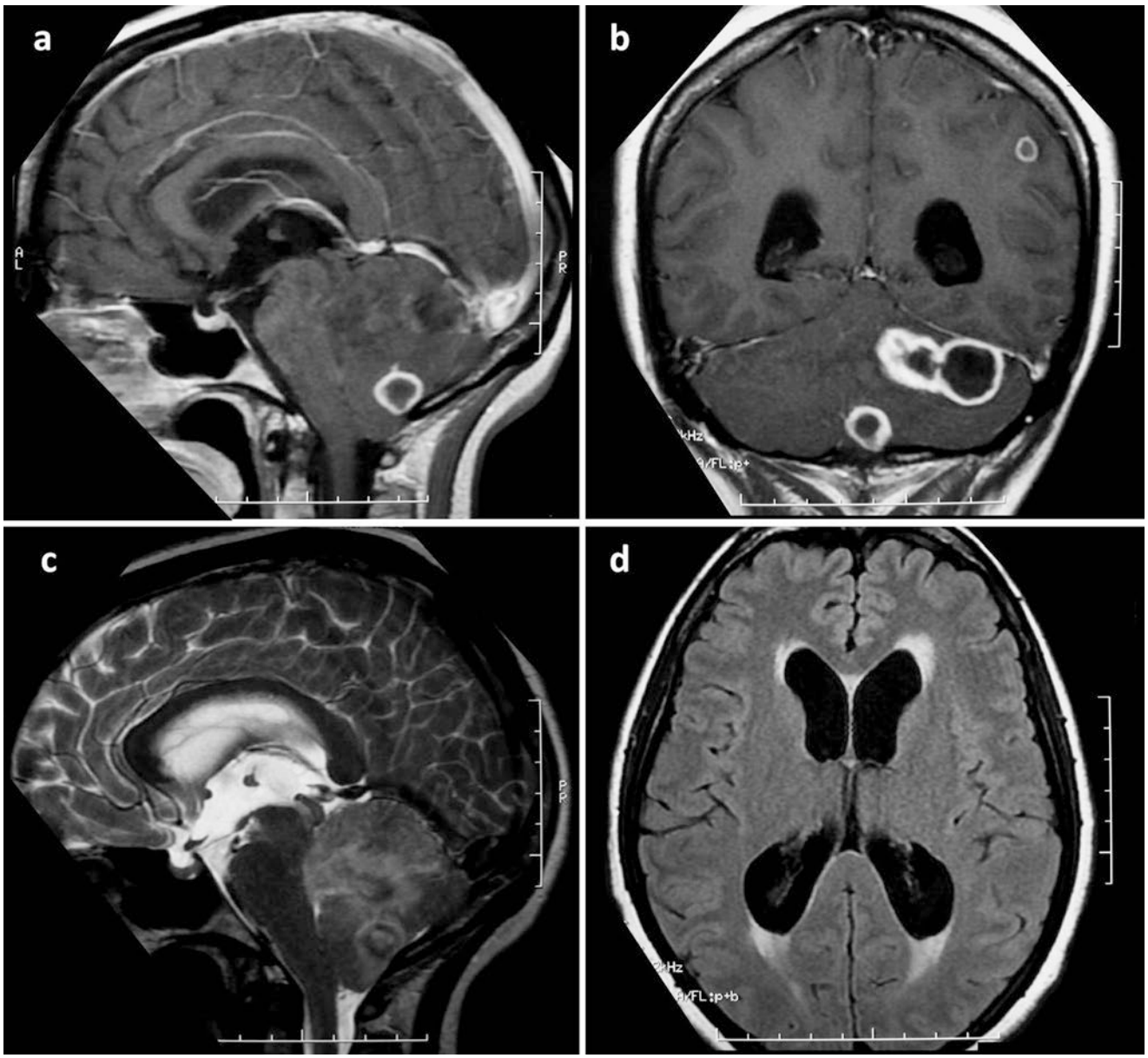


**Fig. 8.16** Sagittal (**a**) and coronal (**b**) post-gadolinium T1-weighted MRI and sagittal T2-weighted MRI (**c**) and axial FLAIR sequence (**d**) show multiple tuberculous cerebellar abscesses with supratentorial hydrocephalus

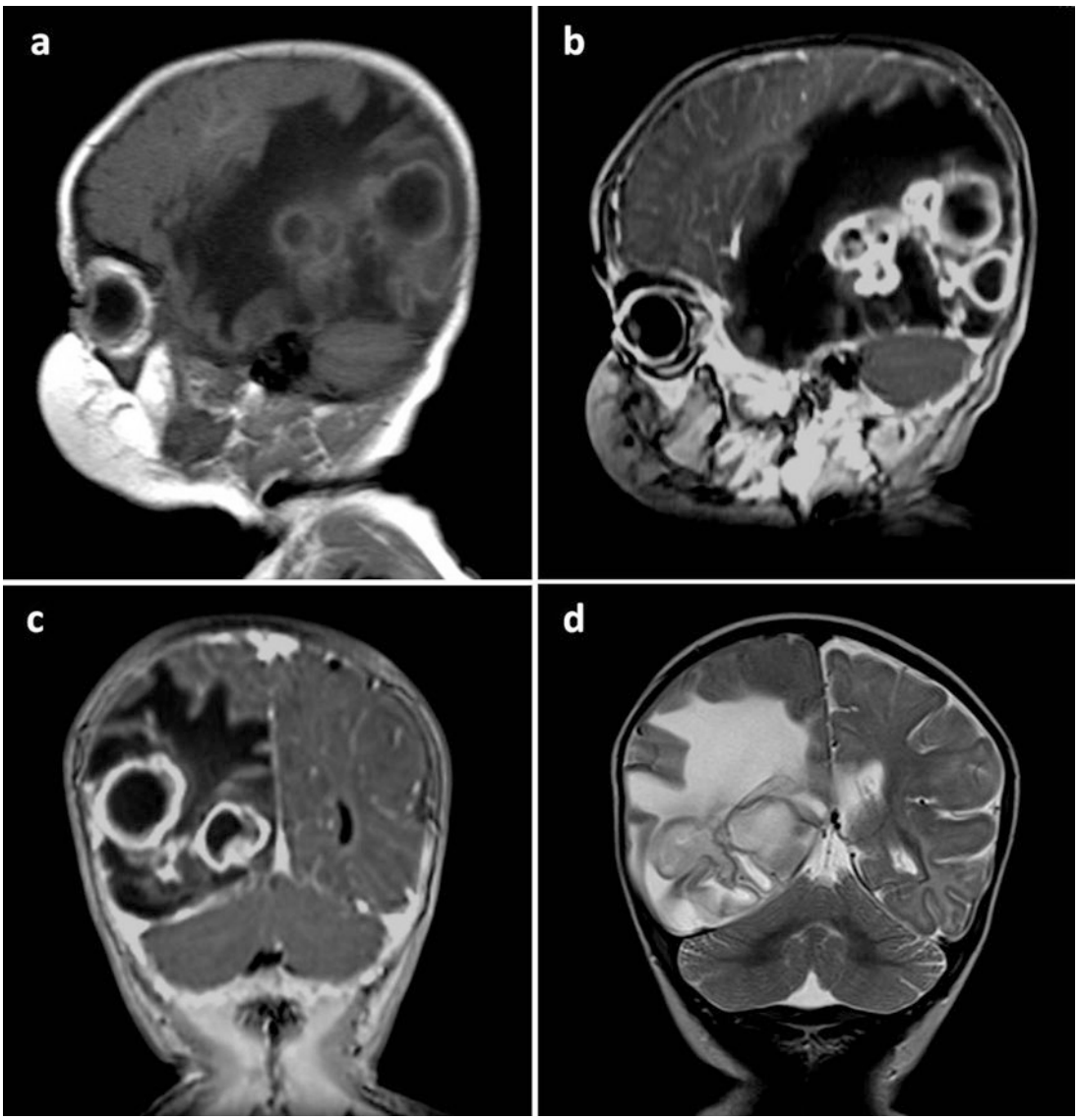


**Fig. 8.17** Case 8.3. Multiple multiloculated brain abscesses located in the *right* parieto-temporo-occipital area with important surrounding edema, seen on sagittal T1-weighted MR images before (**a**) and after (**b**) gadolinium administration and coronal post-gadolinium T1-weighted (**c**) and T2-weighted MRI (**d**)

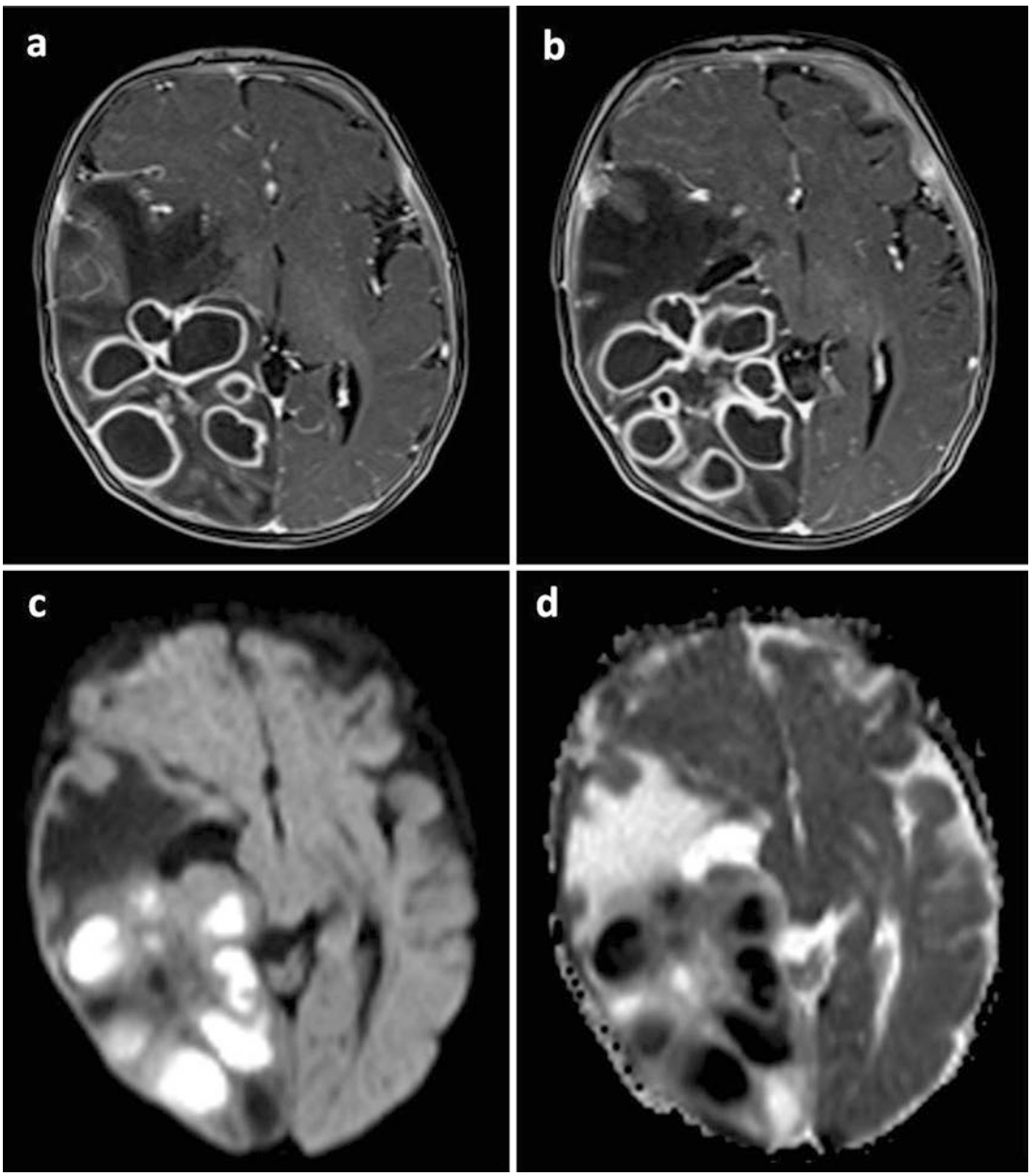


**Fig. 8.18** Case 8.3. Axial post-gadolinium T1-weighted MRI (**a**, **b**), diffusion-weighted image (DWI) (**c**), and apparent diffusion coefficient map (**d**). Note the restricted DWI (*bright signal*) (**c**) and the diminished apparent diffusion coefficient (*low signal*) (**d**)

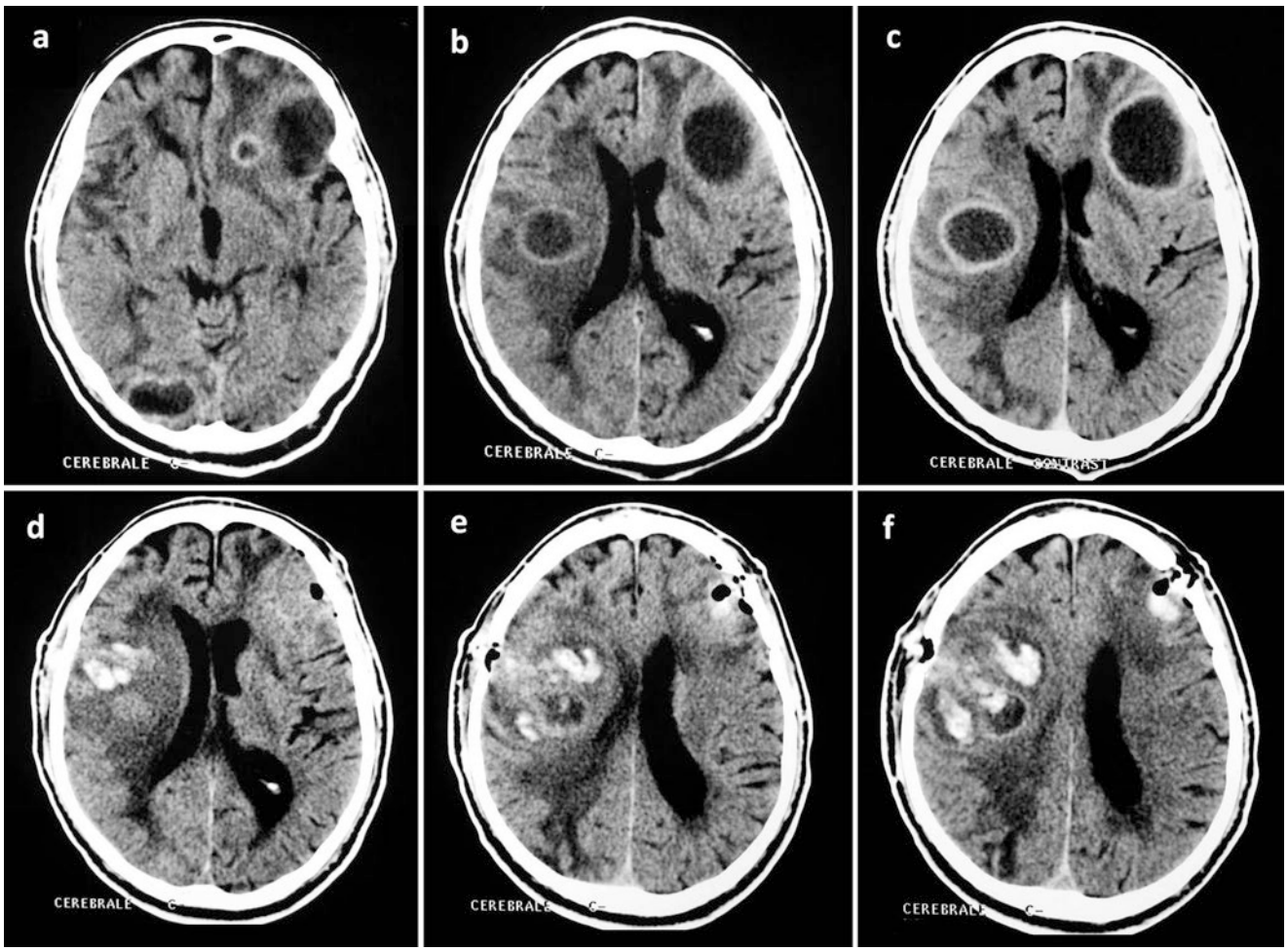


**Fig. 8.19** Multiple bacterial brain abscesses in an elderly man with complicated diabetes mellitus, seen on axial cranial CT scans before surgical aspiration (**a–c**) and immediately following it (**d–f**). Note the important postoperative bleeding within the abscess cavities

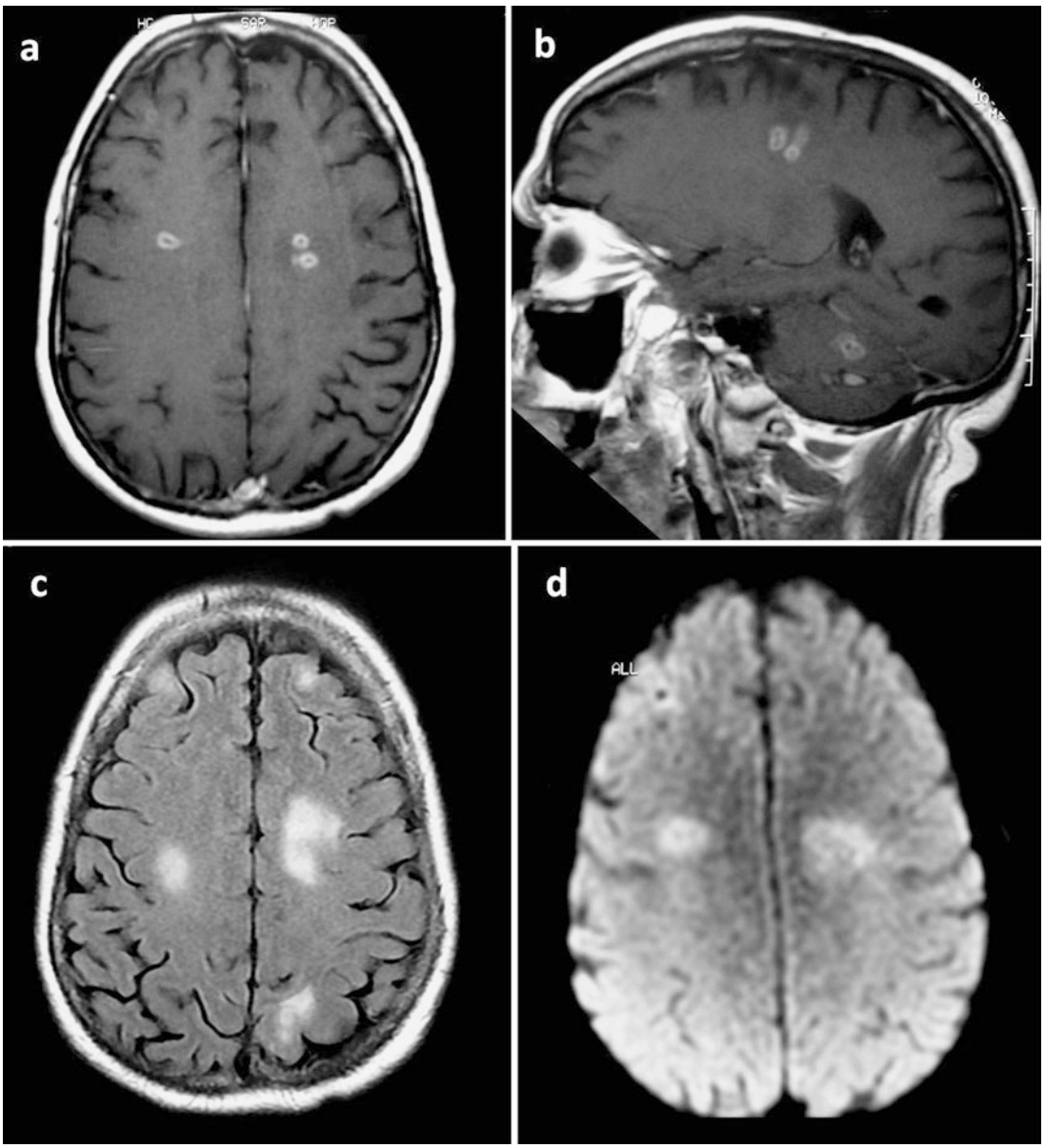


**Fig. 8.20** Multiple infratentorial and supratentorial bacterial brain abscesses on both sides (miliary), seen on axial (**a**) and sagittal (**b**) post-gadolinium T1-weighted MRI, on FLAIR sequence (**c**), and on DWI (**d**)

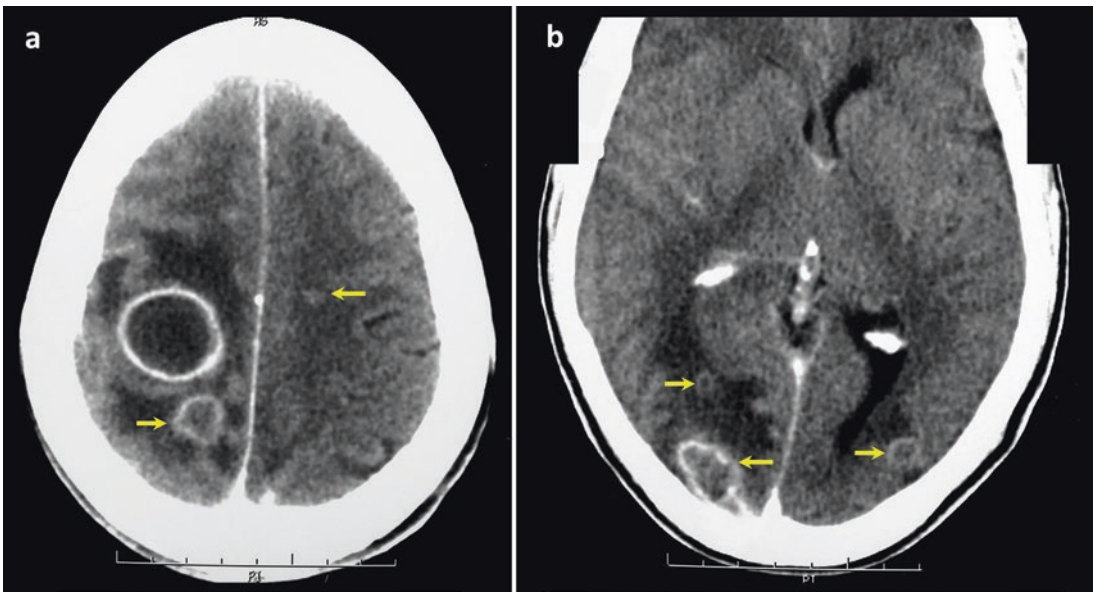


**Fig. 8.21** (**a**, **b**) Multiple brain abscesses on both sides in a patient with infectious endocarditis, seen on axial cranial CT scans following contrast injection. Note the various sizes of the abscesses (*arrows*)

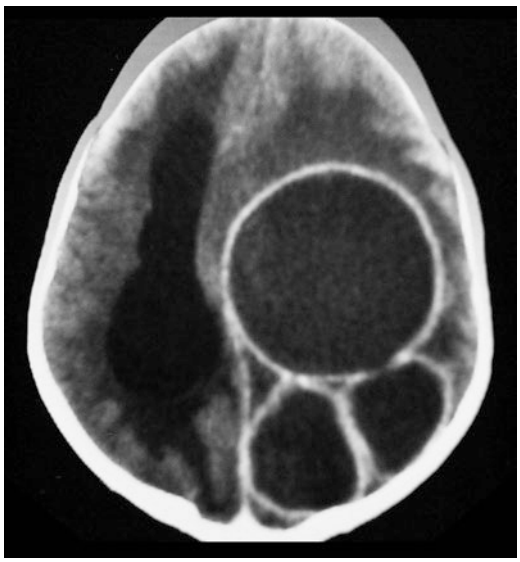

**Fig. 8.22** Axial post-contrast CT scan showing multiple large abscesses in the *left* hemispheric cerebrum in a comatose baby

## Suggested Reading


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# Infectious Encephalitis

# 9

Most cases of acute encephalitis result from primary viral infections. Among them, encephalitis due to herpes simplex virus is the most common in immunocompetent adults, in whom it causes fulminant necrotizing hemorrhagic encephalitis with a predilection for the temporal lobes. Infectious encephalitis secondary to bacteria, parasites, or fungi is less common but should be considered. Acute viral encephalitis is a medical emergency that needs prompt diagnosis and treatment, as acute forms of infectious encephalitis may progress to disability and death.

## Epidemiology and Etiology

Infectious encephalitis, also called primary encephalitis, is a diffuse inflammation of the brain resulting from direct invasion by an infectious agent. The term "meningoencephalitis" is used when the meninges are also involved. Encephalitis is classified as primary or secondary.

In secondary encephalitis (also known as post-infectious or para-infectious encephalitis), the brain may be involved indirectly during or after an infection or after the administration of a vaccine.

Encephalitis also may be acute or chronic. Most acute encephalitis involves primary viral infections (herpes simplex, varicella zoster, cytomegalovirus, Epstein–Barr virus, arboviruses, HIV, and others). Infection due to herpes simplex virus is the most common in immunocompetent adults. Acute viral encephalitis is a medical emergency that needs prompt diagnosis and treatment.

Different forms of chronic encephalitis may be seen, including progressive multifocal encephalopathy in immunodeficient adults and subacute sclerosing panencephalitis in children.

Nonviral causes of encephalitis include *Mycobacterium tuberculosis*, *Listeria monocytogenes*, *Rickettsia* species, syphilis, *Toxoplasma gondii*, *Plasmodium falciparum,* and some other parasitic and fungal infections. Noninfectious etiologies consist of Behçet's disease, vasculitis, carcinoma, and drug reactions. However, most cases of encephalitis do not have an identified etiology.

The neurosurgeon may be confronted with some patients with infectious encephalitis appearing clinically and radiologically as a mass lesion. Histological examination and CSF shunting for hydrocephalus may be needed.

## Clinical Presentation

Adult patients with acute infectious encephalitis present with acute onset of fever, headache, confusion, personality changes, and sometimes seizures. Younger children or infants may present with irritability, poor appetite, and fever. Coma appears within days. Neurological examinations usually reveal a drowsy or confused patient with or without focal neurologic deficits. Stiff neck (nuchal rigidity), due to the irritation of the meninges covering the brain, indicates that the patient has either meningitis or meningoencephalitis. These presentations may mimic bacterial meningitis or subarachnoid hemorrhage.

The clinical features in patients with chronic encephalitis may resemble those seen in acute encephalitis, but the onset is more gradual.

## Imaging Features

CT scans and especially MRI of the brain are sensitive for the diagnosis of viral encephalitis. When herpes simplex virus is the causative pathogen, edema (hypodensity) is predominantly localized in the temporal lobe, orbitofrontal lobe, and limbic system on CT scans. Diffuse edema may be associated with hemorrhagic lesions (with a poorer prognosis). Enhancement may occur in the second week. MRI will demonstrate edema as high signal on T2-weighted images, primarily within the temporal lobe, with some extension across the Sylvian fissure (the transsylvian sign). These lesions may be unilateral or bilateral (Figs. 9.1, 9.2, 9.3, 9.4, 9.5, 9.6, 9.7, 9.8, and 9.9).



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_9

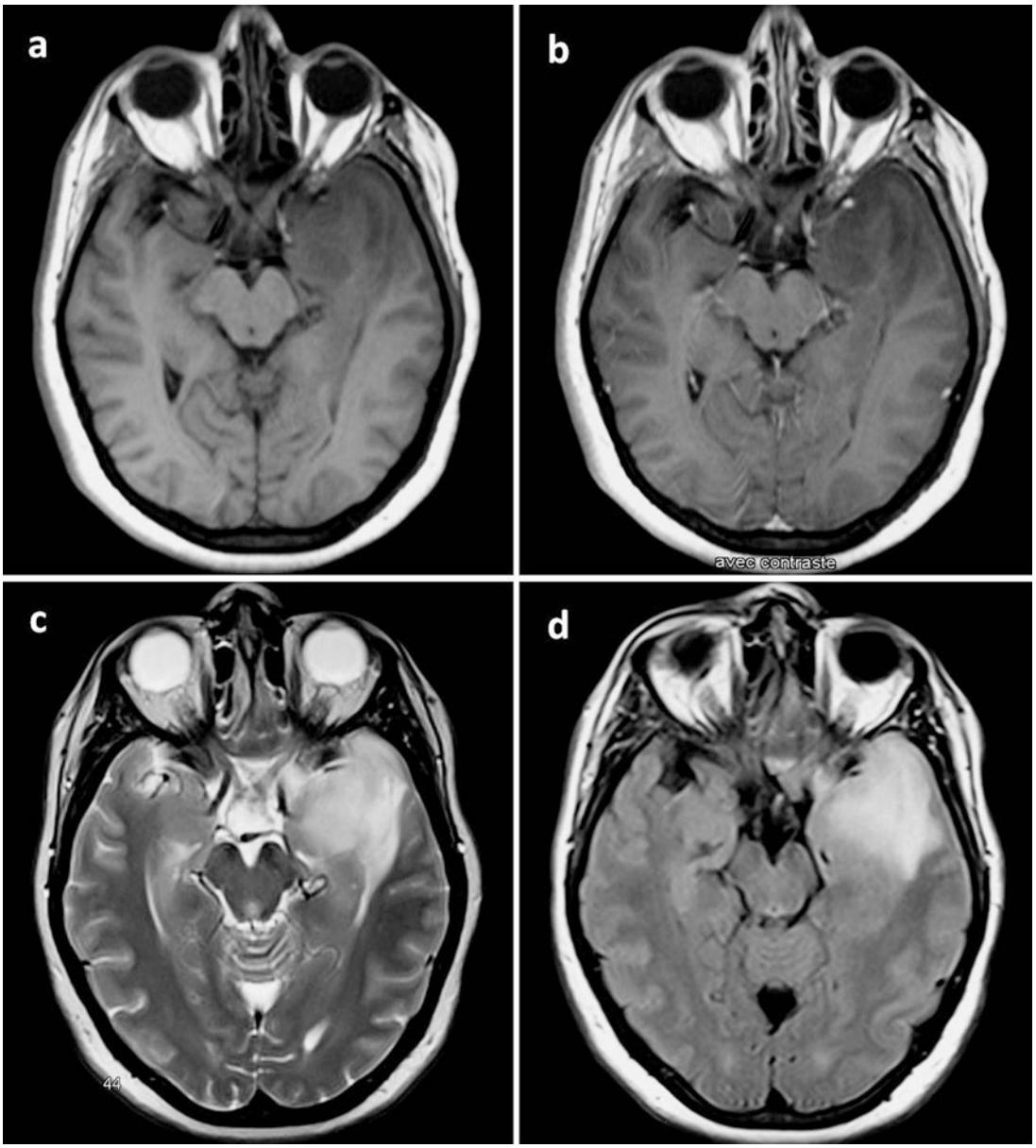


**Fig. 9.1** Axial brain T1-weighted MR images before (**a**) and after (**b**) gadolinium administration, T2-weighted image (**c**), and FLAIR sequence (**d**) showing characteristic changes in the *left* temporal lobe consistent with herpesviral encephalitis

Electroencephalography (monitoring brain activity) will produce abnormal signal in the form of periodic lateralizing epileptiform discharges.

When cerebral vasculitides are associated, appropriate neuroimaging (CT angiography, MR angiography, or conventional four-vessel angiography) is needed to quickly establish the diagnosis. The neurovascular imaging signs are those of inflammation of intracranial vessels with pathological vascular remodeling, vascular occlusion, and ischemia.

## Laboratory Findings

CSF analysis is nonspecific and often shows a picture compatible with aseptic meningitis (increased red blood cells and elevated protein levels). Low glucose levels are uncommon. Herpes simplex virus DNA can be detected in CSF by polymerase chain reaction (PCR) assay. Antibodies may appear in the CSF after 2 weeks.

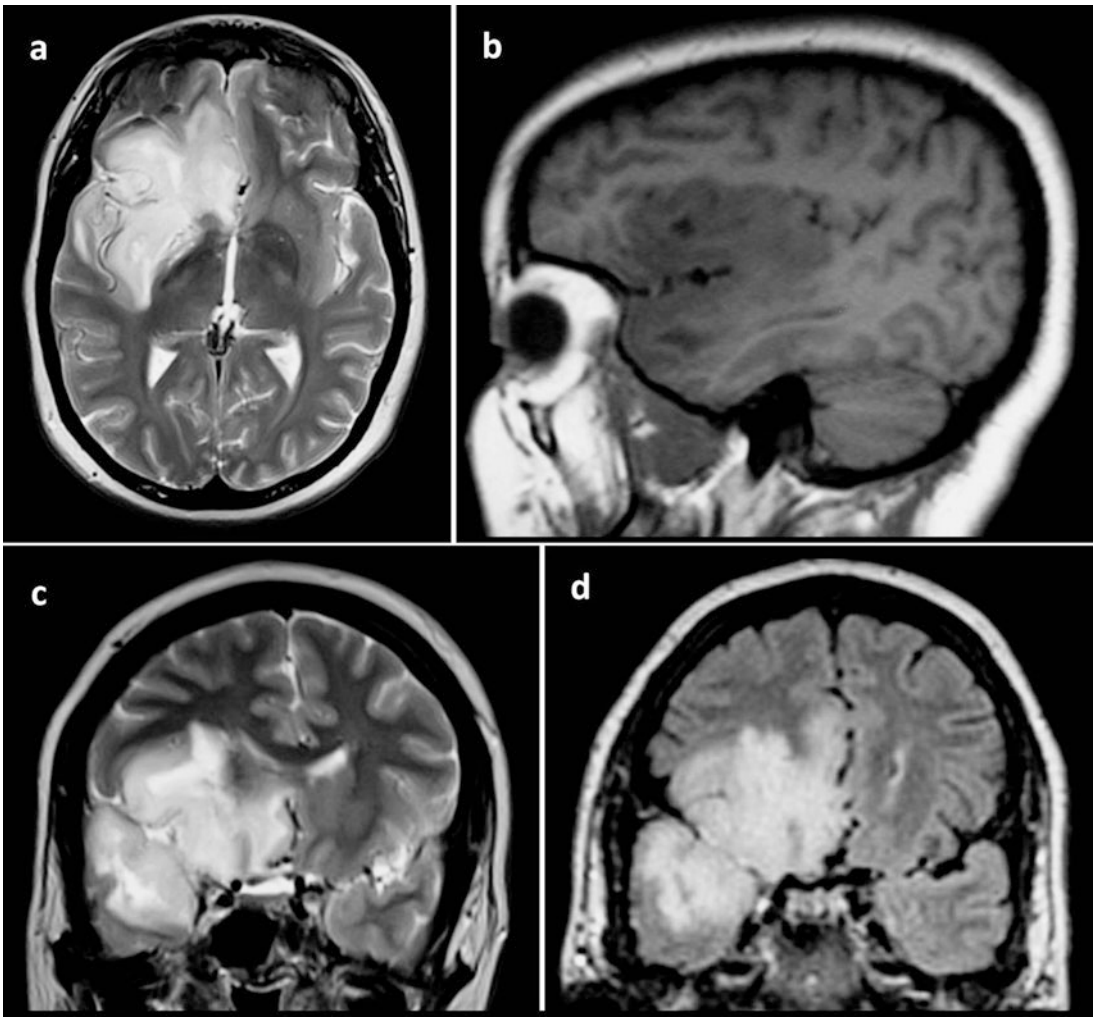


**Fig. 9.2** Temporal and frontal lobe abnormalities in a patient with herpes simplex encephalitis on the right side. T1-weighted images before (**a**) and after (**b**) gadolinium administration, T2-weighted image (**c**), and FLAIR sequence (**d**). Note the "transsylvian sign"—signal abnormality localized in the right insular area with extension across the Sylvian fissure

Brain biopsy is indicated for viral culture and histological examination. The anterior inferior temporal lobe is the preferred site for viral isolation. Other, less-specific histological findings are perivascular cuffing, lymphocytic infiltration, hemorrhagic necrosis, neuronophagia, and intranuclear inclusions.

Several other biologic tests may also be needed to help diagnose infectious encephalitis and check for an underlying bacterial, fungal, or parasitic cause.

## Treatment

Treatment is based on general supportive care. Corticosteroids, mannitol, and hyperventilation are used to reduce brain swelling. Sedatives are administered for agitation, antiepileptic drugs for seizure, and antipyretics for fever. Physical therapy is used to avoid decubitus complications.

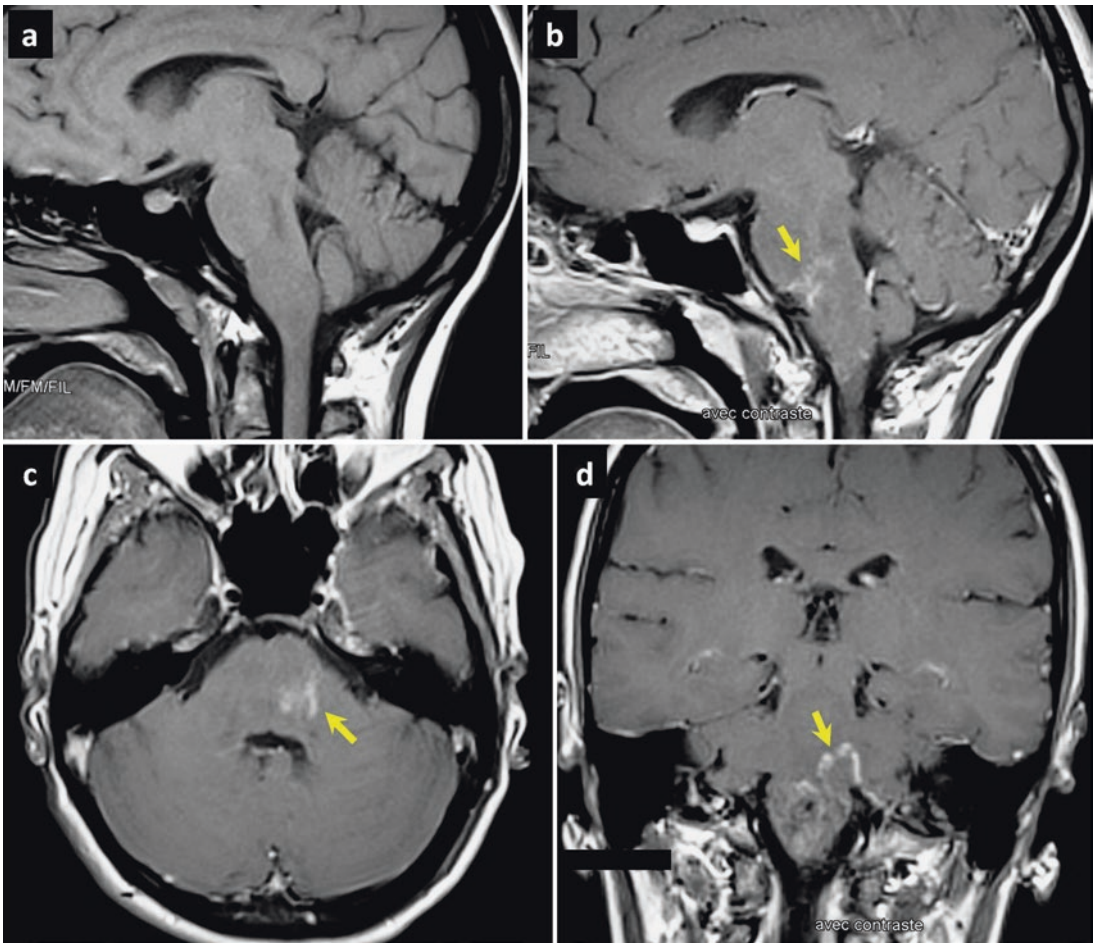


**Fig. 9.3** Case 9.1. Sagittal T1-weighted brain images before (**a**) and after (**b**) gadolinium injection, axial (**c**) and coronal (**d**) enhanced T1-weighted images showing features of brainstem encephalitis due to herpes simplex virus. Note the ponto-bulbar gadolinium enhancement (*arrow*)

Antiviral parenteral medication is appropriate when the cause is viral. Acyclovir is capable of improving patient outcome, especially in herpes simplex virus encephalitis.

Bacterial, fungal, or parasitic etiologies should be treated with the appropriate anti-infectious regimens.

The neurosurgeon's role in the management of most patients with encephalitis is in aiding diagnosis by providing tissue by biopsy and/or cerebrospinal fluid (CSF) and in the treatment and surgical management of increased intracranial pressure. Timely CSF diversion procedures for hydrocephalus may prevent long-term neurological and cognitive decline.

## Outcomes

Both acute and chronic forms of encephalitis may progress to disability and death, but acute viral encephalitis is more rapidly life-threatening than chronic encephalitis. Mortality following antiviral treatment is influenced by age, Glasgow Coma Score, and the duration of disease before therapy.

Cognitive disability, seizures, and motor deficits are common sequelae seen among survivors.

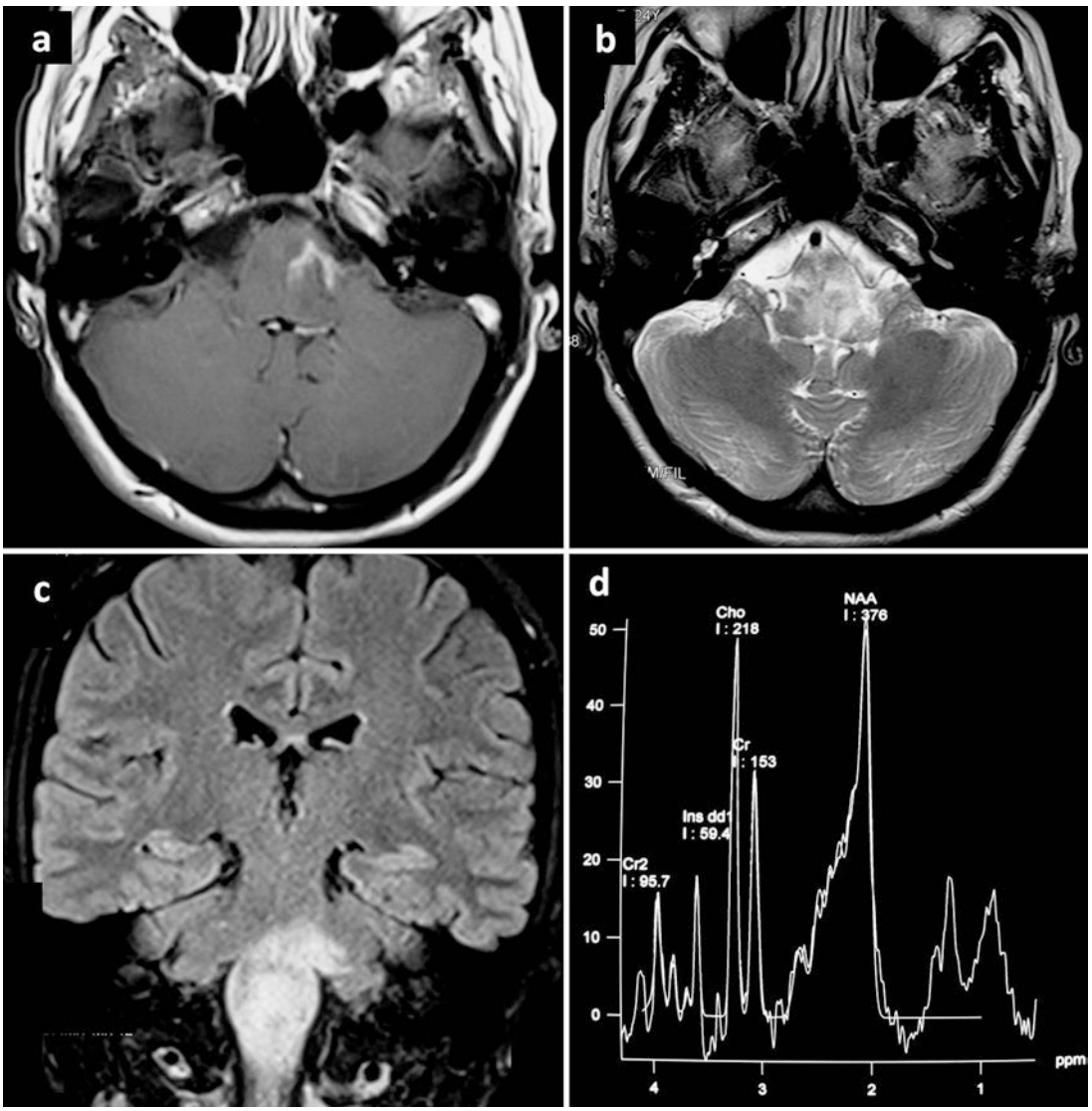


**Fig. 9.4** Case 9.1. Herpes brainstem encephalitis. Axial gadolinium-enhanced T1-weighted (**a**) and T2-weighted (**b**) images, coronal FLAIR sequence (**c**), and spectroscopy (**d**)

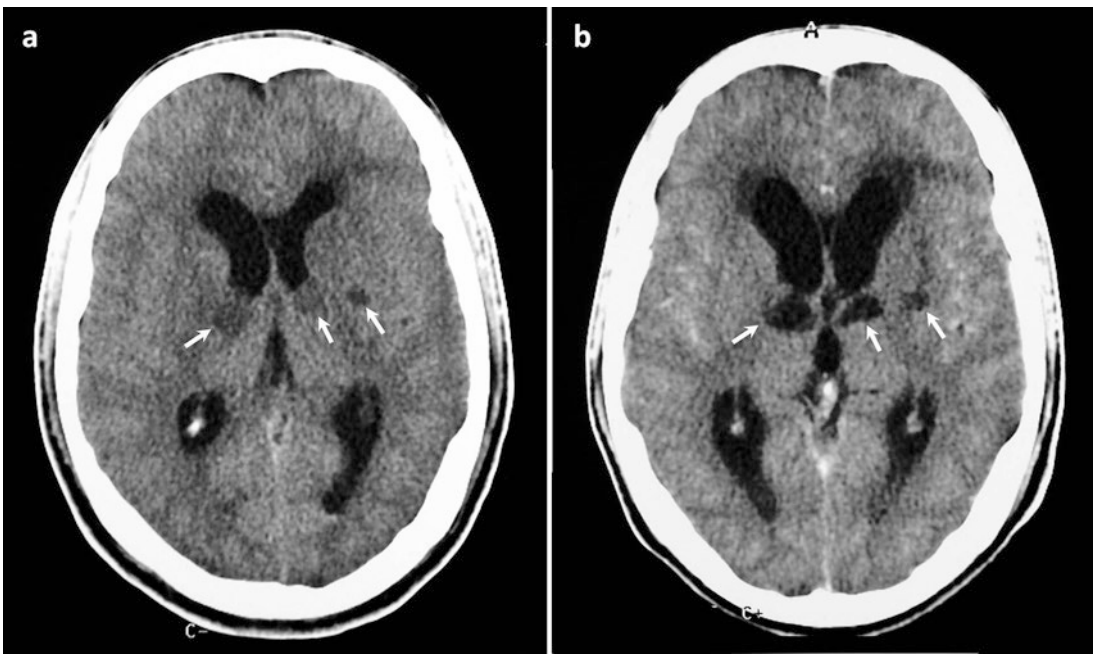


**Fig. 9.5** Case 9.2. Acute varicella zoster meningoencephalitis in a 23-year-old man who presented with acute headache, confusion, and right hemiplegia. Axial brain CT scans before (**a**) and after (**b**) contrast injection show multiple hypodense, nonenhancing focal lesions (*arrows*)

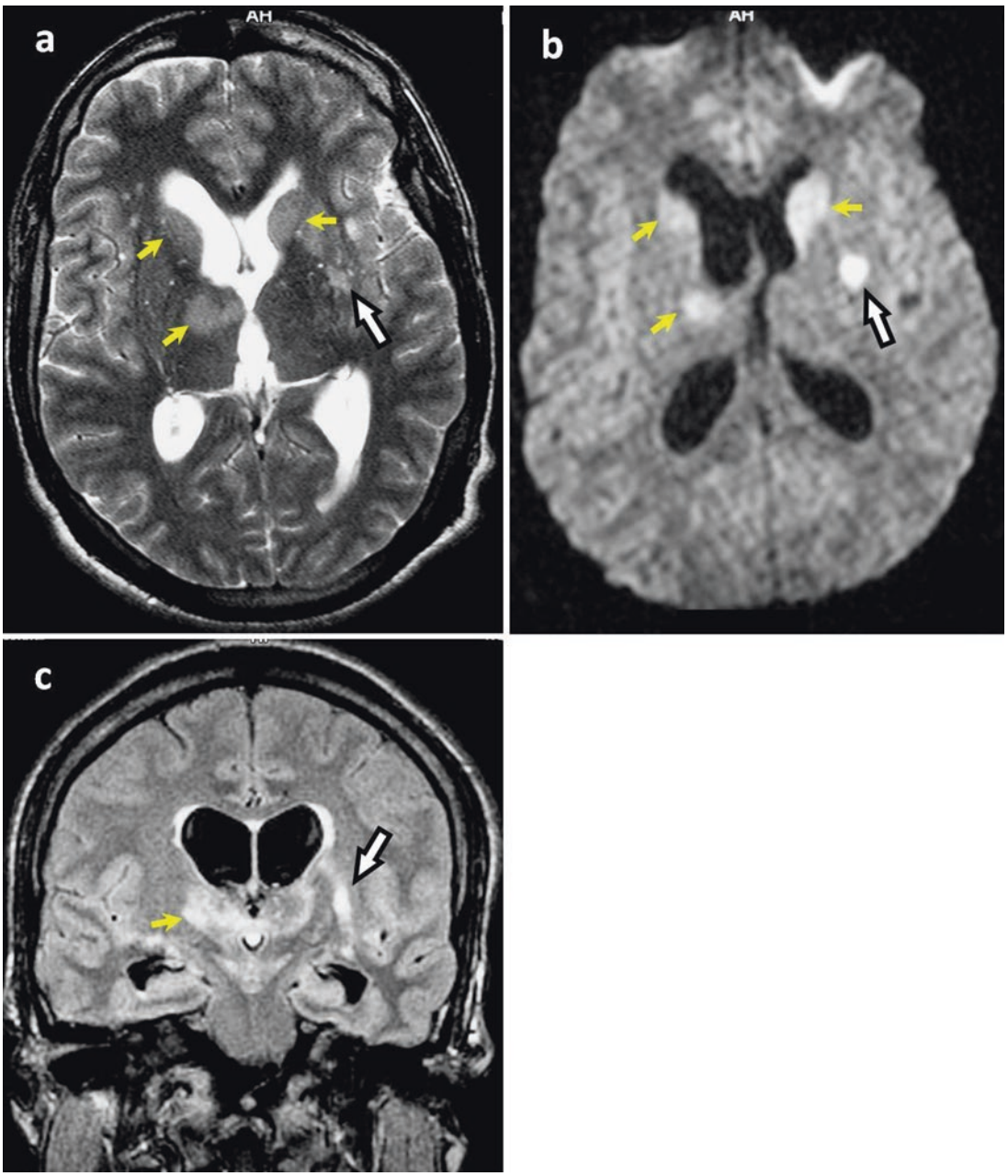


**Fig. 9.6** Case 9.2. Axial T2-weighted image (**a**), diffusion-weighted sequence (**b**), and FLAIR sequence (**c**) reveal multiple bilateral signal abnormalities located in the head of the caudate nucleus and the thalamus (*yellow arrows*). Note the left internal capsule lesion (*white arrow*), explaining the contralateral hemiplegia

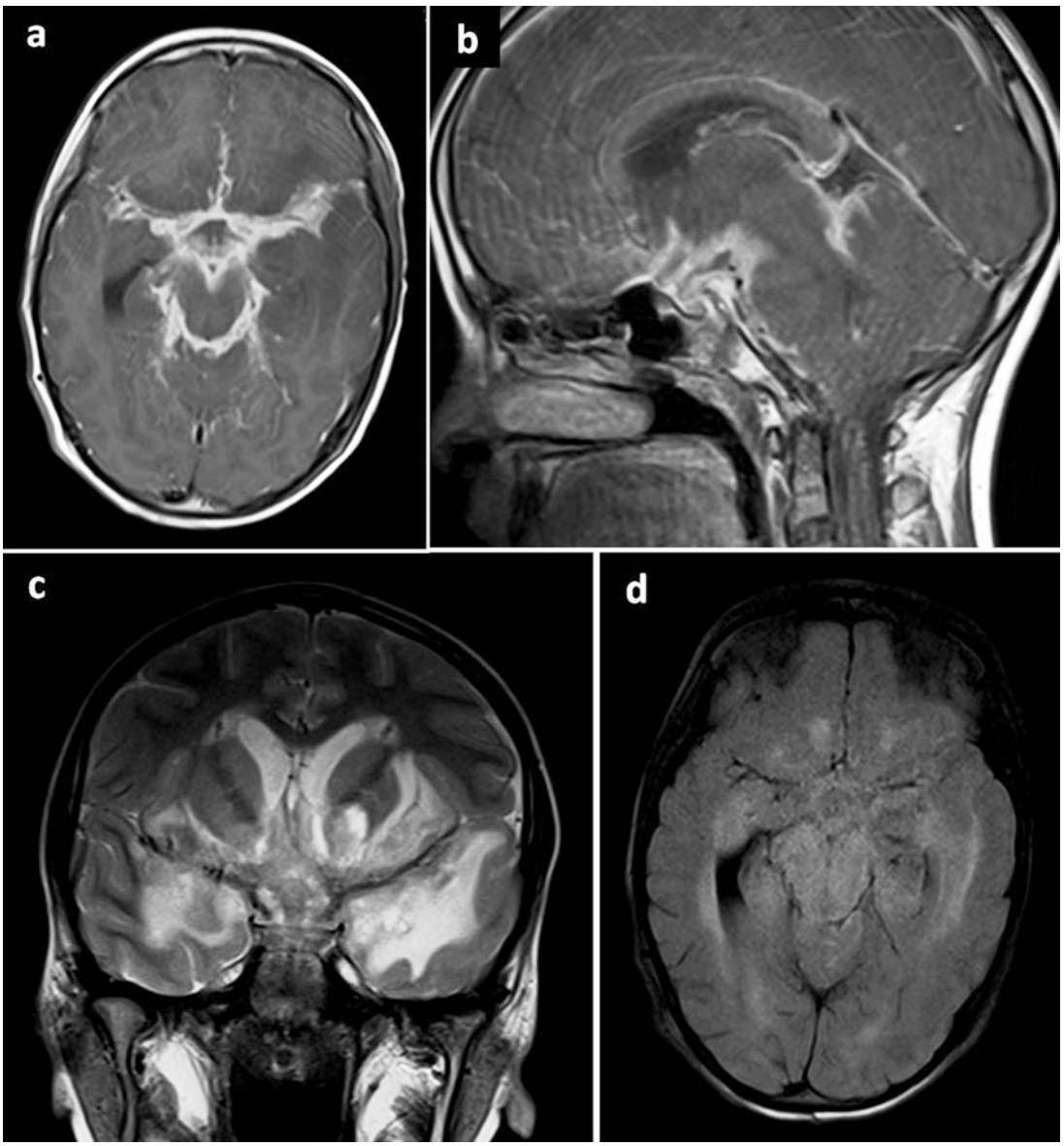


**Fig. 9.7** Tuberculous meningoencephalitis in a 12-year-old boy. Axial (**a**) and sagittal (**b**) T1-weighted images, coronal T2-weighted image (**c**), and FLAIR sequence (**d**). Note the significant basilar leptomeningeal enhancement (**a**, **b**) and the extensive adjacent parenchymal edema

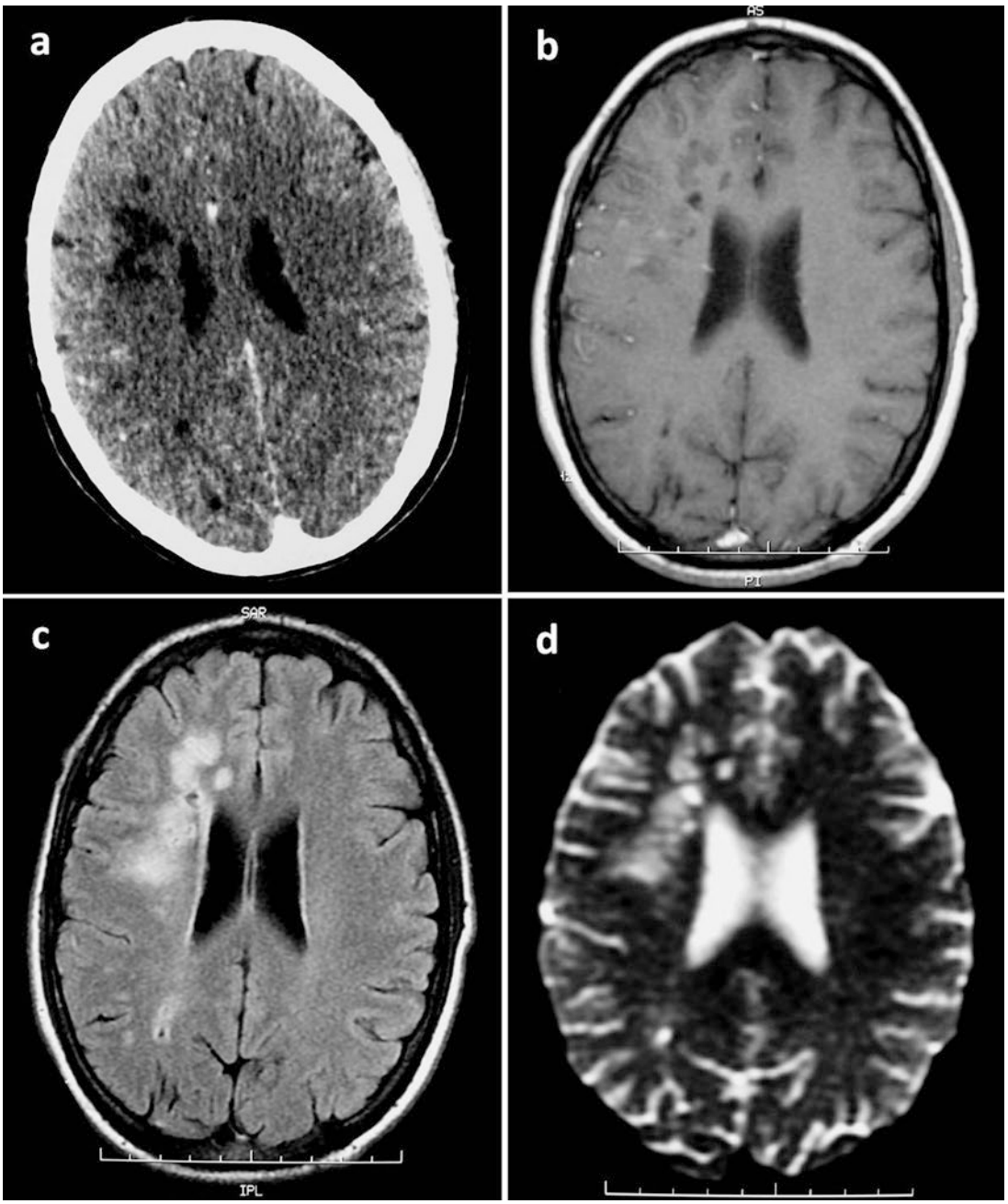


**Fig. 9.8** Axial cranial-enhanced CT scan (**a**), T1-weighted image following gadolinium injection (**b**), FLAIR sequence (**c**), and apparent diffusion coefficient map (**d**) showing neuroimaging features of focal subcortical encephalitis located in the right frontal area, with mild gadolinium enhancement

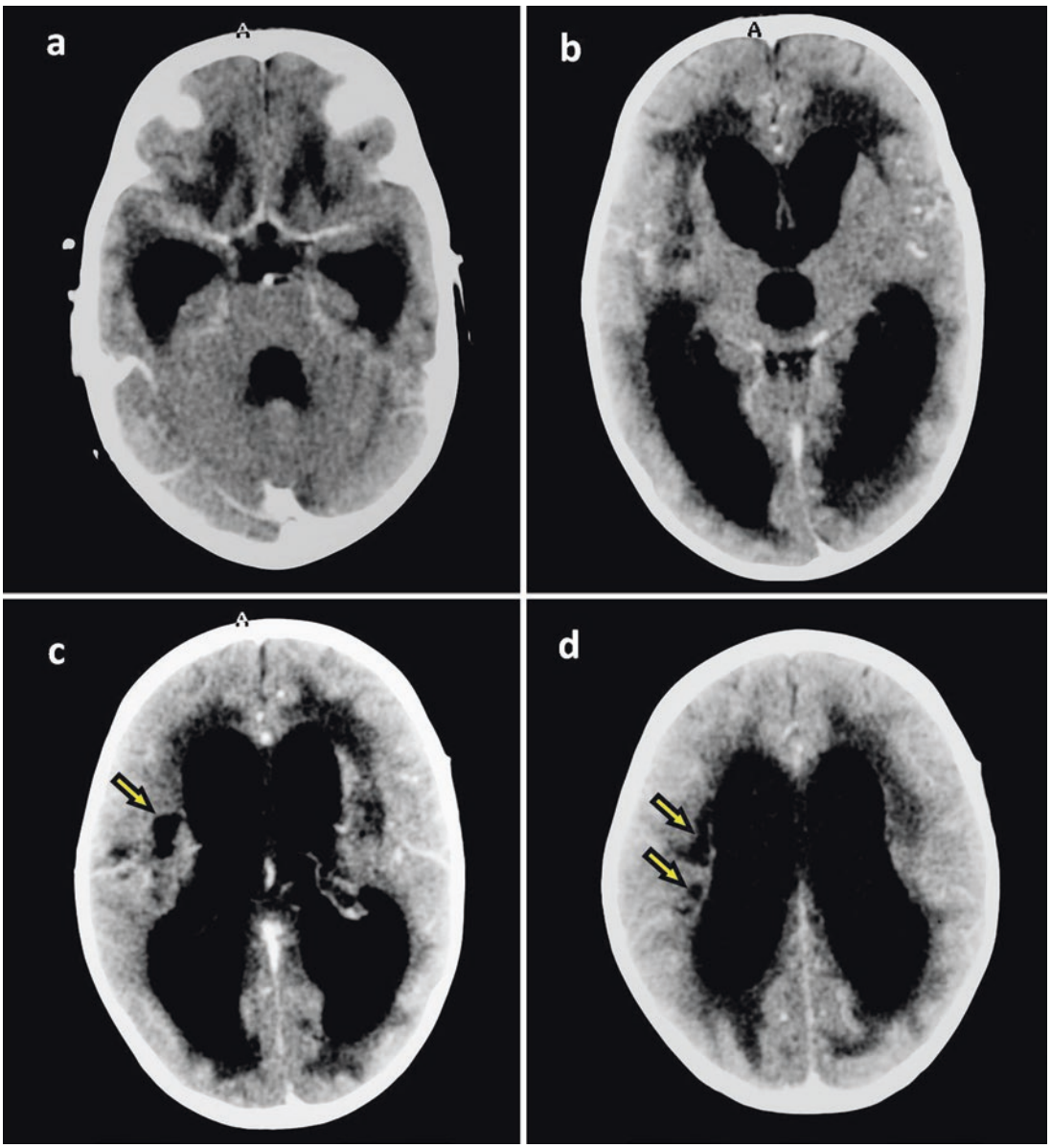


**Fig. 9.9** (**a–d**) Axial cranial CT scans after contrast injection. Tuberculous meningoencephalitis with acute hydrocephalus and multiple ischemic infarcts (*arrows*) in a 15-month-old baby

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# Pyogenic Ventriculitis

# 10

Pyogenic ventriculitis (pyoventriculitis) is characterized by the existence of suppurative fluid in the cerebral ventricular system. It may result from the rupture of a brain abscess, extension of meningitis into the ventricles, implantation of pathogens following a head injury, or a neurosurgical procedure with or without an implanted device. The typically indolent clinical course of pyoventriculitis sometimes can be rapidly life-threatening. Signs and symptoms are those of meningitis and raised intracranial pressure. Focal neurologic deficits may be present when a brain abscess is associated. Neuroimaging techniques are fundamental in the diagnosis. CT scan and especially MRI usually demonstrate intraventricular debris and pus in the cerebrospinal fluid (CSF). Other findings may include hydrocephalus, periventricular anomalies, and ventricular ependymal enhancement. CSF studies usually show a low glucose level, high protein, and pleocytosis. Isolation of the pathogenic agent and culture are essential in determining the antimicrobial therapy. When ventriculitis is unresponsive to intravenous antibiotics or if neurologic status is considered perilous, intrathecal antibiotic drugs can be administered. Concomitant brain abscess may be drained. Pyogenic ventriculitis is a potentially fatal infection that can lead to severe sequelae.

## Epidemiology and Etiology

Ventriculitis is an inflammation of the ependymal lining of the brain ventricular system. Pyogenic ventriculitis (pyoventriculitis) is characterized by the presence of suppurative fluid in the cerebral ventricles. Most cases are secondary to intraventricular rupture of a cerebral abscess (pyocephalus), direct implantation of pathogens following head injury (especially with skull base fracture and CSF leak), or a neurosurgical procedure or device (especially a ventricular catheter). Less frequently, ventriculitis may be a complication of meningitis or may occur spontaneously in immunocompromised patients.

Brain abscess can cause significant mass effect if it reaches a great volume, and a periventricular location entails the danger of intraventricular rupture and pyoventriculitis owing to the relatively poorer vascularization and capsule development from the ependymal plane.

Pyogenic ventriculitis is a rare but serious intracranial infection that classically is associated with poor outcomes and even death.

## Clinical Presentations

The clinical course of pyoventriculitis is typically indolent and nonspecific, but sometimes it may be rapidly life-threatening. Signs and symptoms include headache, fever, seizure, stiff neck, photophobia, and altered mental status, as seen in patients with meningitis. Signs of raised intracranial pressure due to hydrocephalus should be considered.

Focal neurologic deficits may be present when brain abscess is associated. Intraventricular rupture of the abscess can result in the rapid clinical deterioration of a previously stable patient. Severe headaches and signs of meningeal irritation are prominent before rupture.

## Imaging Features

Brain imaging techniques are fundamental in showing pyogenic ventriculitis. CT scans and especially MRI usually demonstrate intraventricular debris and pus in the CSF. Other findings may consist of hydrocephalus, periventricular MR anomalies (reflecting inflammatory alterations), and ventricular ependymal enhancement (Figs. 10.1, 10.2, 10.3, 10.4, and 10.5). Negative imaging may delay the diagnosis and worsen the prognosis, especially if the clinical picture is ambiguous.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_10

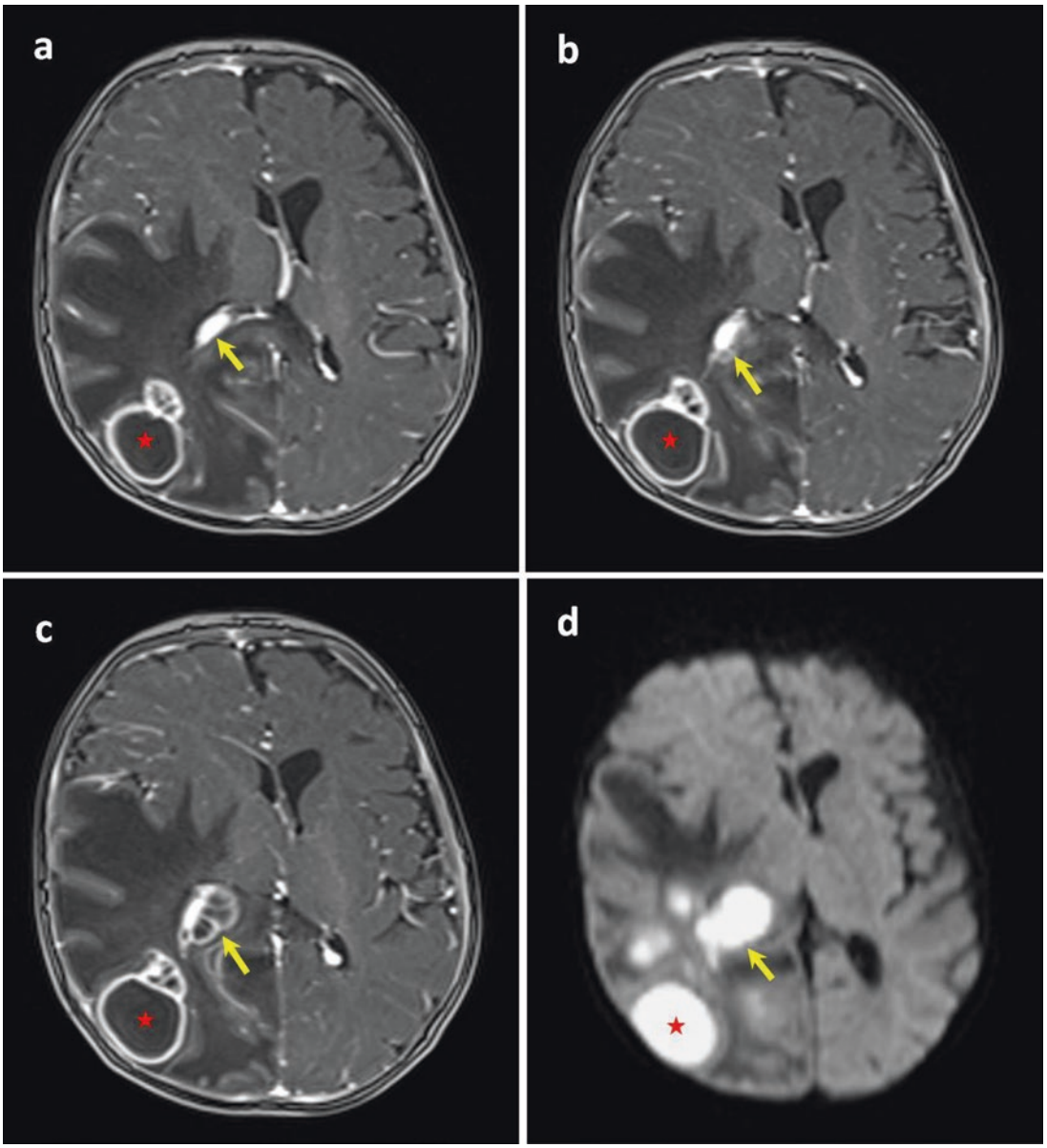


**Fig. 10.1** Brain MRI of pyoventriculitis. Axial post-contrast T1-weighted images (**a–c**) and a diffusion-weighted image (DWI) (**d**) show rupture of a right parietal brain abscess (*star*) into the adjacent lateral ventricle (*arrow*). There is peripheral rim enhancement of the intraparenchymal abscess and its intraventricular component and also linear enhancement along the right lateral ventricular wall (ependymitis) (**a–c**). The right lateral ventricular lesion is seen on DWI as marked hyperintensity (**d**)

Restricted diffusion-weighted imaging (DWI) (bright signal), periventricular high signal, and ependymal enhancement on fluid-attenuated inversion recovery (FLAIR) sequences are considered very useful in detecting pyoventriculitis. The lower apparent diffusion coefficient (ADC) values of the hyperintense lesions on DWI might suggest the presence of material with restricted water diffusion in the periventricular space.

## Laboratory Findings

CSF studies usually show a low glucose level, high protein, and pleocytosis. Identifying the pathogen responsible using a CSF gram stain and culture is essential in determining the cause. Coagulase-negative staphylococci, gram-negative bacilli, and *Staphylococcus aureus* are most often responsible

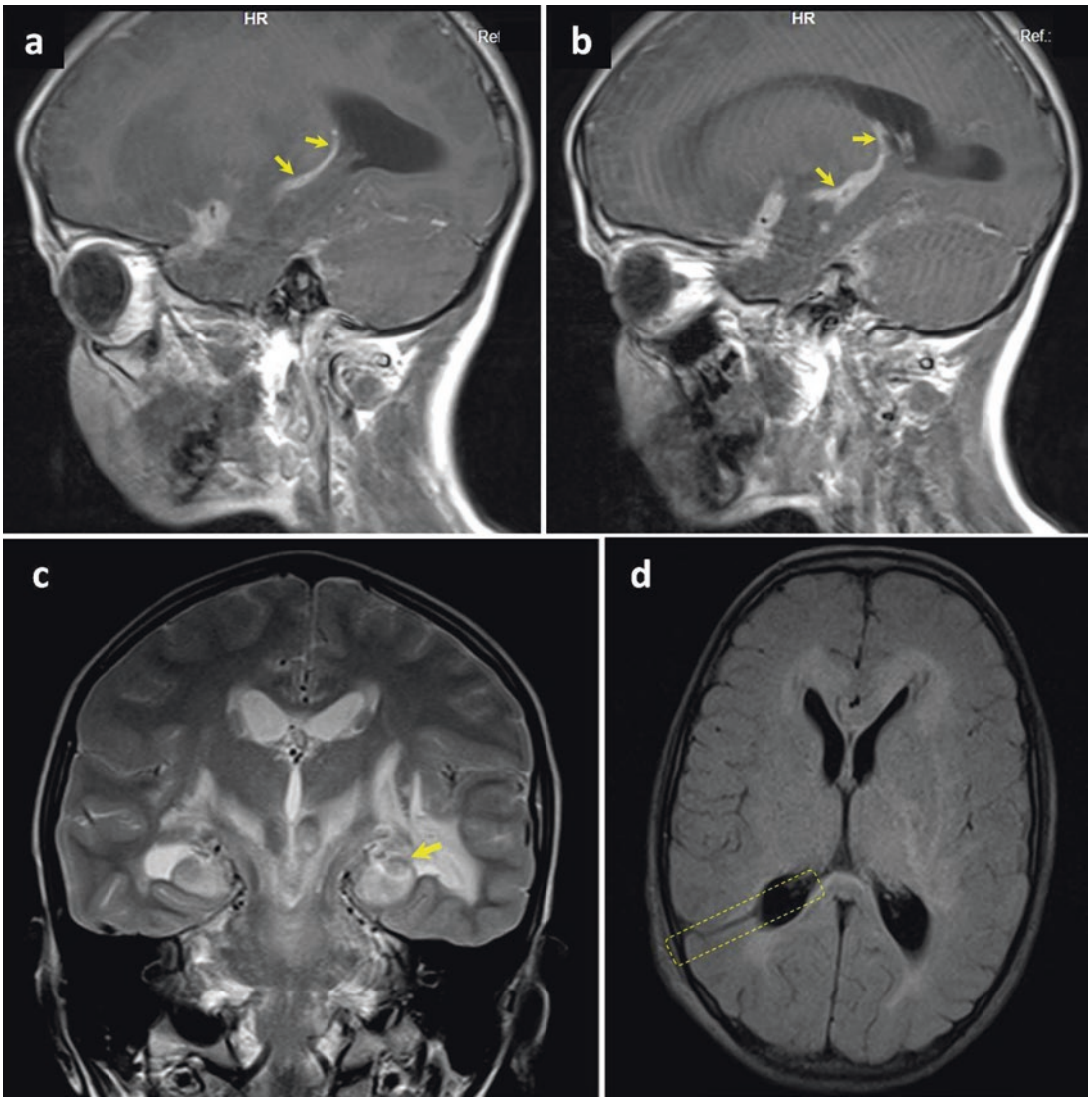


**Fig. 10.2** Tuberculous ventriculitis in a 2-year-old boy under antituberculous treatment for a meningoencephalitis, as seen on sagittal contrast-enhanced T1-weighted MRI (**a**, **b**), a coronal T2-weighted image (**c**), and a fluid-attenuated inversion recovery (FLAIR) sequence (**d**). An area of slight hypointensity is seen in the *left* temporal horn (**c**), with intraventricular and linear ependymal enhancement of the *left* temporal horn and the trigone (*arrows*). This patient developed a secondary hydrocephalus. CSF shunting was performed with a good outcome (**d**)

for ventriculitis. *Streptococcus pneumoniae* and gram-negative bacilli are often seen in ventriculitis following head injury. Gram-positive cocci and *Acinetobacter* species are common findings in catheter-related ventriculitis.

Blood tests can sometimes demonstrate leukocytosis and abnormalities in inflammatory parameters such as the C-reactive protein level and erythrocyte sedimentation rate. Blood cultures may be useful.

## Treatment Options

Vancomycin is the drug of choice for treatment of staphylococci including *S. aureus*, *S. epidermidis*, or gram-negative rods. Linezolid in combination with a third-generation cephalosporin has also been recommended, especially when the ventriculitis is caused by *Enterobacter* species or *Pseudomonas* species meningitis.

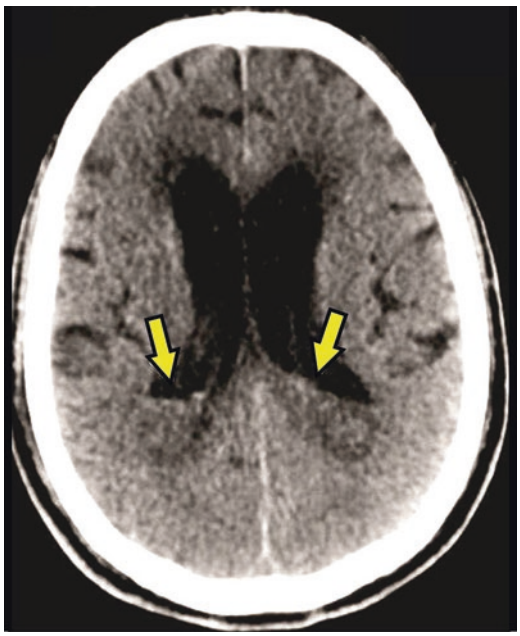

**Fig. 10.3** Cranial axial CT scan without contrast injection in a patient under antibiotic therapy for a posttraumatic meningitis. Purulent debris is lodged in the posterior aspect of the lateral ventricles (*arrows*)

When ventriculitis is unresponsive to intravenous antibiotics or if neurologic status is more perilous, intrathecal vancomycin or colimycin can be administered using an external intraventricular drain or an implanted device (like an Ommaya reservoir). Intraventricular administration of antibiotics is not recommended in neonates, however.

Concomitant brain abscess should be drained whenever possible.

As with other severe neuroinfections, many additional supportive therapies may be necessary, as well as initial resuscitation, management of severe infection issues, anticonvulsants, corticosteroids, anticoagulation, analgesics and antipyretics, treatment of any medical comorbidities, and functional rehabilitation.

## Outcomes

Pyogenic ventriculitis can lead to severe sequelae (seizures, focal neurologic deficits, and permanent alterations in mental status) and even death. Treatment should be aggressive and appropriate to achieve a favorable long-term patient outcome.

Prognosis depends on the state of general health, the concomitant CNS complications, the delay in diagnosis, the patient's age, and the response to therapy.

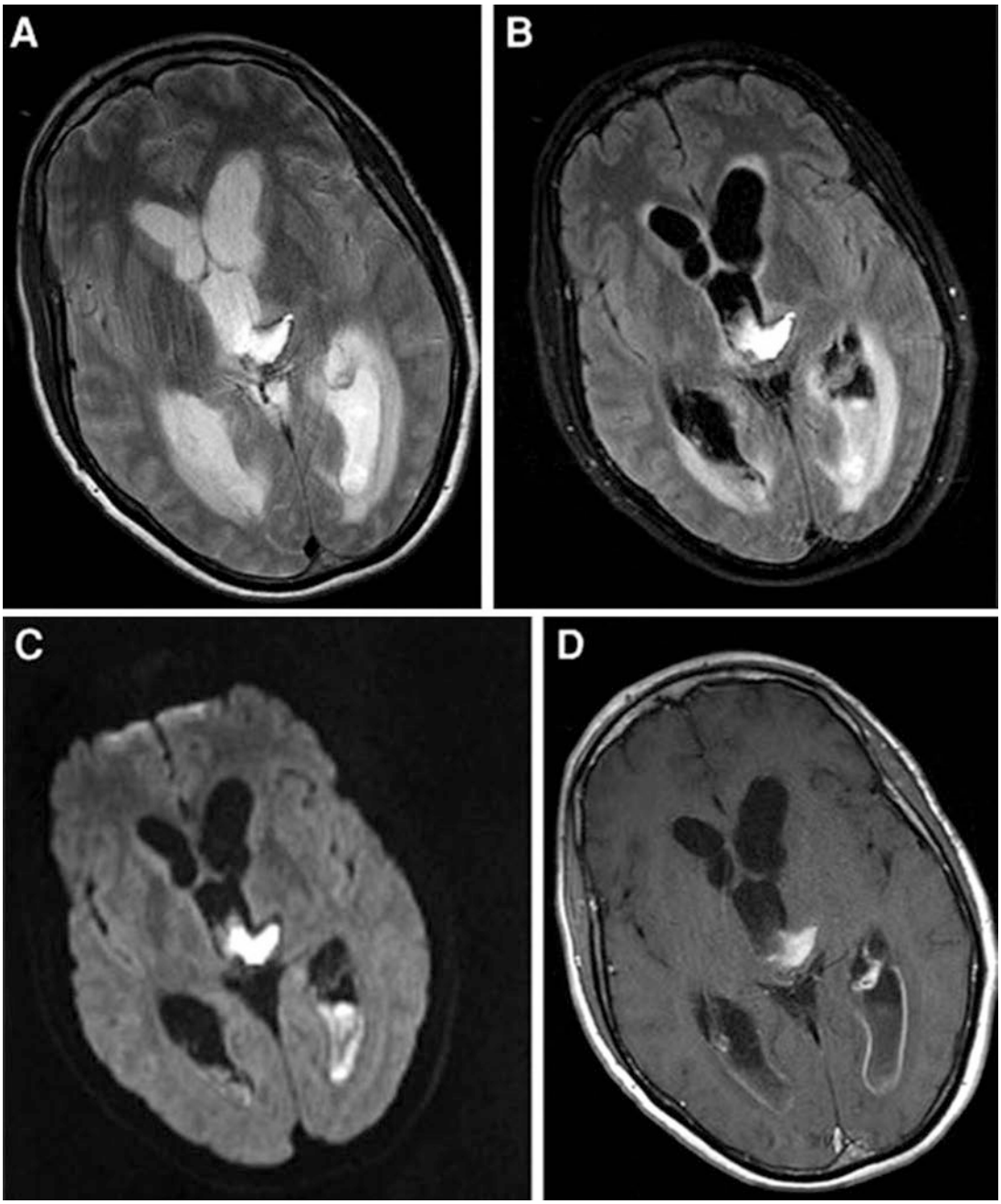


**Fig. 10.4** MRI of the brain showing a pyogenic ventriculitis. Axial T2-weighted image (**a**), FLAIR image (**b**), DWI (**c**), and contrast-enhanced T1-weighted image (**d**). The intraparenchymal hematoma in the left thalamus/mesencephalon is hyperintense on all sequences and shows diffusion restriction, consistent with clotted blood. The periventricular inflammation is easily identified on the T2-weighted (**a**) and FLAIR (**b**) images as a hyperintense halo in the subependymal white matter surrounding the occipital horns. The thick-walled ependymal enhancement in the occipital horns (**d**) indicates ependymitis. The ventricular debris in the occipital horns has high signal intensity on the FLAIR image (**b**) and shows diffusion restriction (**c**); this finding is consistent with pus (From Jorens et al. (2009); with permission)

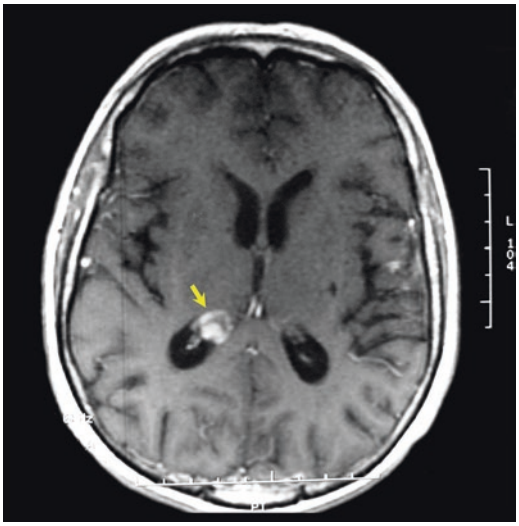

**Fig. 10.5** Axial post-gadolinium MRI of the brain showing a tuberculous granulomatous lesion in the right lateral ventricle separating the CSF anteroposteriorly (*arrow*)

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# Pituitary Abscesses

# 11

Pituitary abscess is a rare, focal purulent collection of the sellar region. A wide variety of bacterial and fungal microorganisms have been implicated. The most common clinical presentation is anterior pituitary hypofunction, followed by headache, fever, diabetes insipidus, and visual disturbances. Biologic inflammatory parameters are unspecific and should always be interpreted in combination with clinical and imaging data. MRI studies demonstrate a cystic or partially cystic mass in the sellar area with rim enhancement after gadolinium injection. Concomitant sphenoid sinusitis is suggestive, but the diagnosis of pituitary abscess is often missed prior to surgery. Adapted antibiotic therapy, complete transsphenoidal surgical drainage, and hormone replacement for hypopituitarism are the keys to treatment. Symptoms due to mass effect improve after drainage, but hypopituitarism often persists and recurrence is not rare.

## Epidemiology and Etiology

Pituitary abscess is a rare focal purulent collection of the sellar region. Spontaneous pituitary abscesses are classified as primary (in a previously normal, healthy gland) or secondary (with a preexisting lesion). Posttraumatic and postoperative (iatrogenic) abscesses are rarer. Spontaneous abscesses typically develop secondary to extension from paranasal sinus infection (especially sphenoid sinusitis) (Fig. 11.1) or from a spontaneous or traumatic cerebrospinal fluid (CSF) leak.

## Clinical Presentations

The most common clinical presentation is anterior pituitary hypofunction: weakness, anorexia, amenorrhea, and hypogonadism. Other symptoms include headache, fever, meningitis, diabetes insipidus, visual disturbances (mainly due to inflammation of the optic nerves), and altered mental status.

Otolaryngologic examination should be performed to look for a potential infectious focus.

## Imaging Features

CT scan will often demonstrate a homogenous, low-density mass with rim enhancement following contrast administration, with or without sphenoid sinusitis.

MRI studies show a cystic or partially cystic mass in the sellar area that is hypointense or isointense on T1-weighted images and hyperintense or isointense on T2-weighted images, with rim enhancement after gadolinium injection (Figs. 11.2, 11.3, and 11.4). Differential diagnosis should consider Rathke's cyst, pituitary apoplexy, craniopharyngioma, and cystic pituitary adenoma. Restricted diffusion-weighted imaging (DWI) can be useful in differentiating abscesses from other cystic pituitary lesions, but the value of DWI is somewhat inadequate along the skull base and paranasal sinuses.

The diagnosis of pituitary abscess is often missed prior to surgery. Modern imaging may be useful in diagnosing the primary sources of craniofacial infections.

## Laboratory Findings

A wide variety of bacterial and fungal microorganisms have been implicated in the formation of spontaneous pituitary abscesses. The most common infectious agents in primary abscesses are *Staphylococcus* species, *Streptococcus* species, *Pseudomonas* species, and gram-negative bacteria. *Aspergillus fumigatus* is most frequently isolated in cases of



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_11

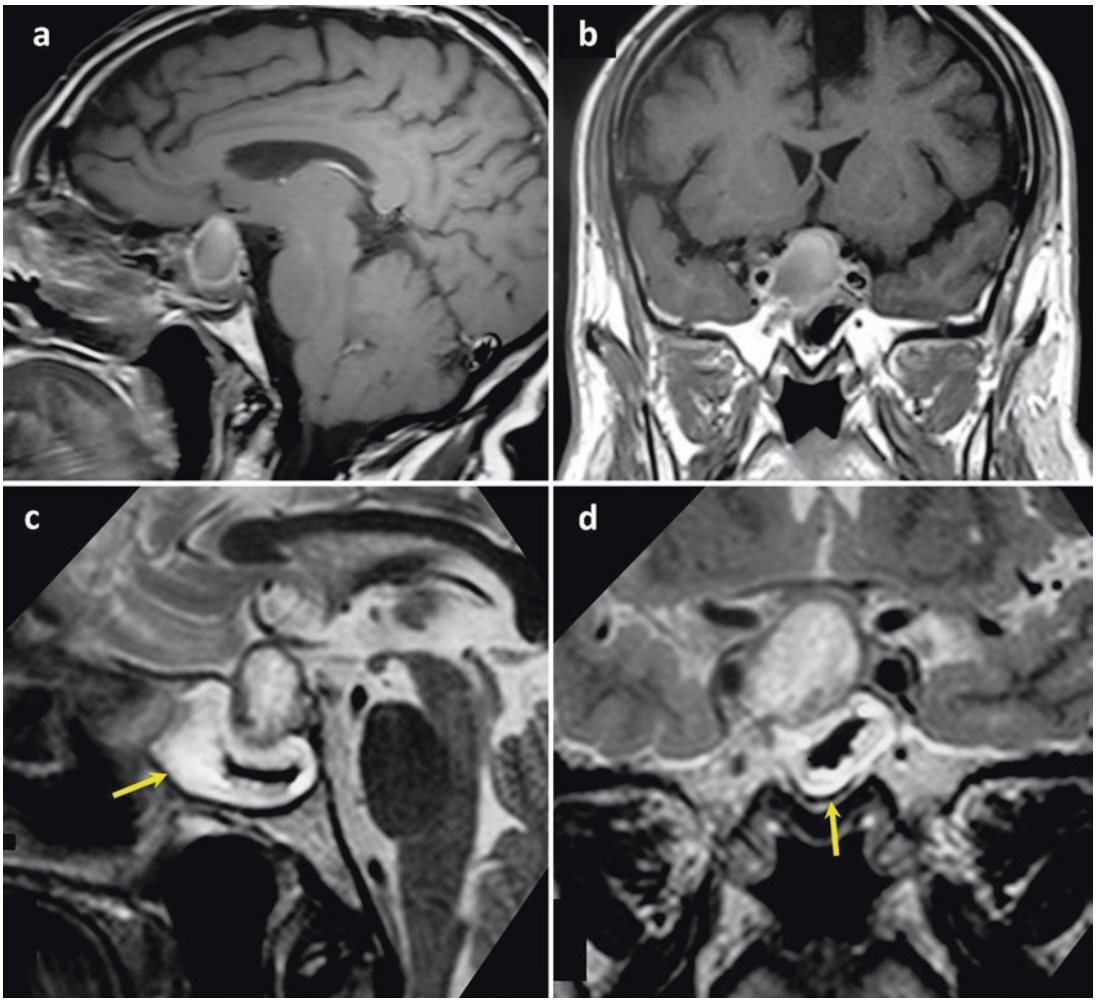


**Fig. 11.1** Spontaneous bacterial pituitary abscess with associated sphenoidal sinusitis. Sagittal (**a**) and coronal (**b**) T1-weighted MR images demonstrating an isointense lesion in the pituitary fossa, with ring enhancement after contrast injection. Sagittal (**c**) and coronal (**d**) T2-weighted images showing an intrasellar, hyperintense cystic lesion with thickened sphenoid sinus mucosa suggesting sinusitis (*arrow*). Note the marked compression of the optic chiasm

secondary abscess. Culture-negative or so-called "sterile" abscesses are not rare.

Biologic inflammatory parameters and elevation of white blood cell count are unspecific and should always be interpreted in combination with clinical and imaging data. Blood cultures are rarely positive.

Histopathology may demonstrate necrosis and abscess wall, with infiltration by polymorphonuclear leucocytes or macrophages. The possibility of secondary pituitary abscess must be always taken into consideration. Some preexisting lesions in the pituitary area may be concomitant to the pituitary abscess, like Rathke's cleft cyst, pituitary apoplexy, craniopharyngioma, and cystic pituitary adenoma (Fig. 11.5). Most secondary pituitary abscess is diagnosed by postoperative histopathologic evidence.

## Treatment Options

Transsphenoidal surgical drainage and marsupialization of the abscess wall are the usual management. The sellar floor should be reconstructed if CSF leakage occurs.

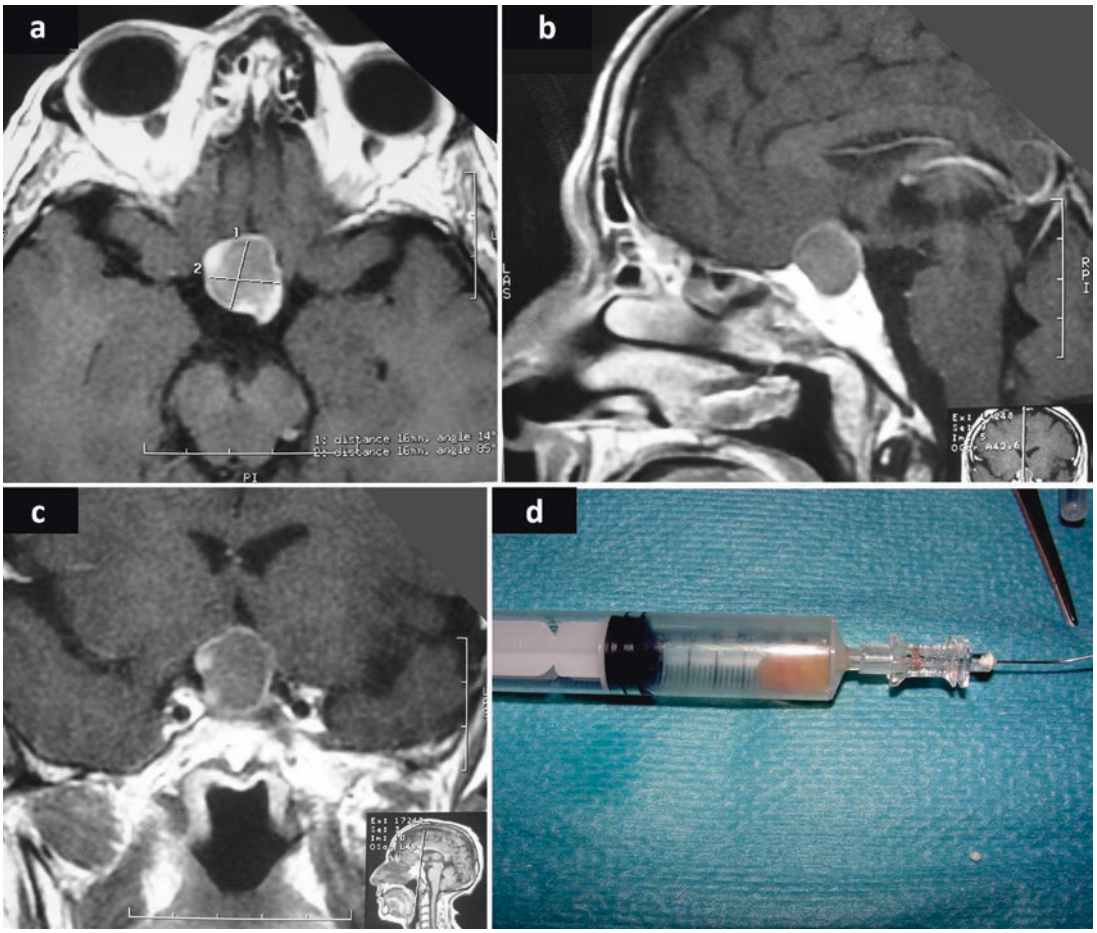


**Fig. 11.2** Axial (**a**), sagittal (**b**), and coronal (**c**) sellar post-gadolinium T1-weighted MRI showing typical features of pituitary abscess. Operative photography (**d**) showing the purulent fluid drained from the intrasellar abscess

Intraoperative cultures should always be done for aerobic and anaerobic bacteria, acid-fast bacilli, and fungi. The antibiotic drugs must be adapted to the specific pathogen once antimicrobial susceptibilities are established. Antibiotic therapy for bacterial abscesses is typically required for 6–8 weeks.

Conservative management with antibiotics alone may be suggested for small suppurative collections without any signs of complication. Surgery should be reconsidered if complications occur.

Postoperative hormone replacement should always be considered.

## Outcomes

Most symptoms due to mass effect improve following drainage, but hypopituitarism persists in many patients. Despite adequate surgical and medical therapy, recurrences may appear. Outcomes for patients with invasive fungal abscess and rhinocerebral mucormycosis are poor.

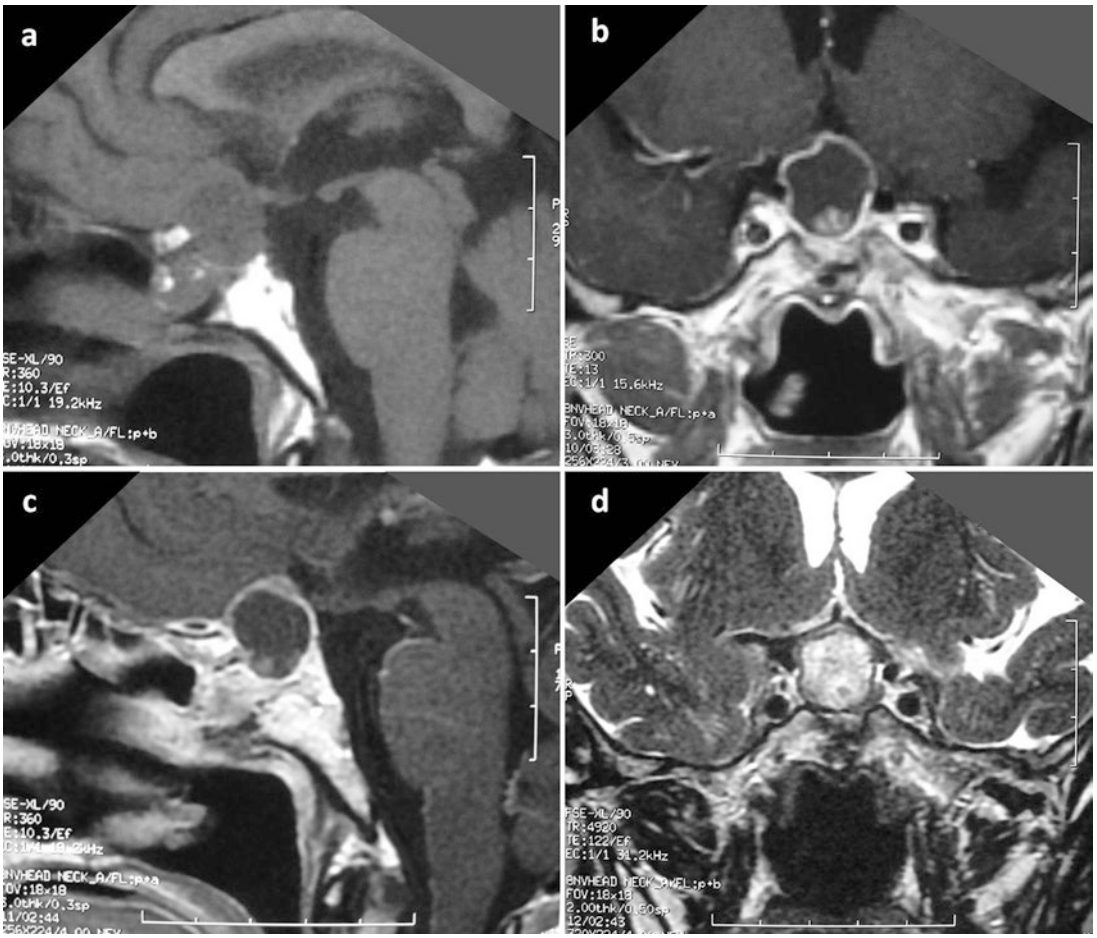


**Fig. 11.3** Postoperative bacterial pituitary abscess with suprasellar extension in a patient with a history of prior transsphenoidal surgery for a pituitary adenoma. Sagittal T1-weighted MRI without gadolinium injection (**a**), coronal (**b**) and sagittal (**c**) T1-weighted gadolinium-enhanced MRI, and coronal T2-weighted MRI (**d**)

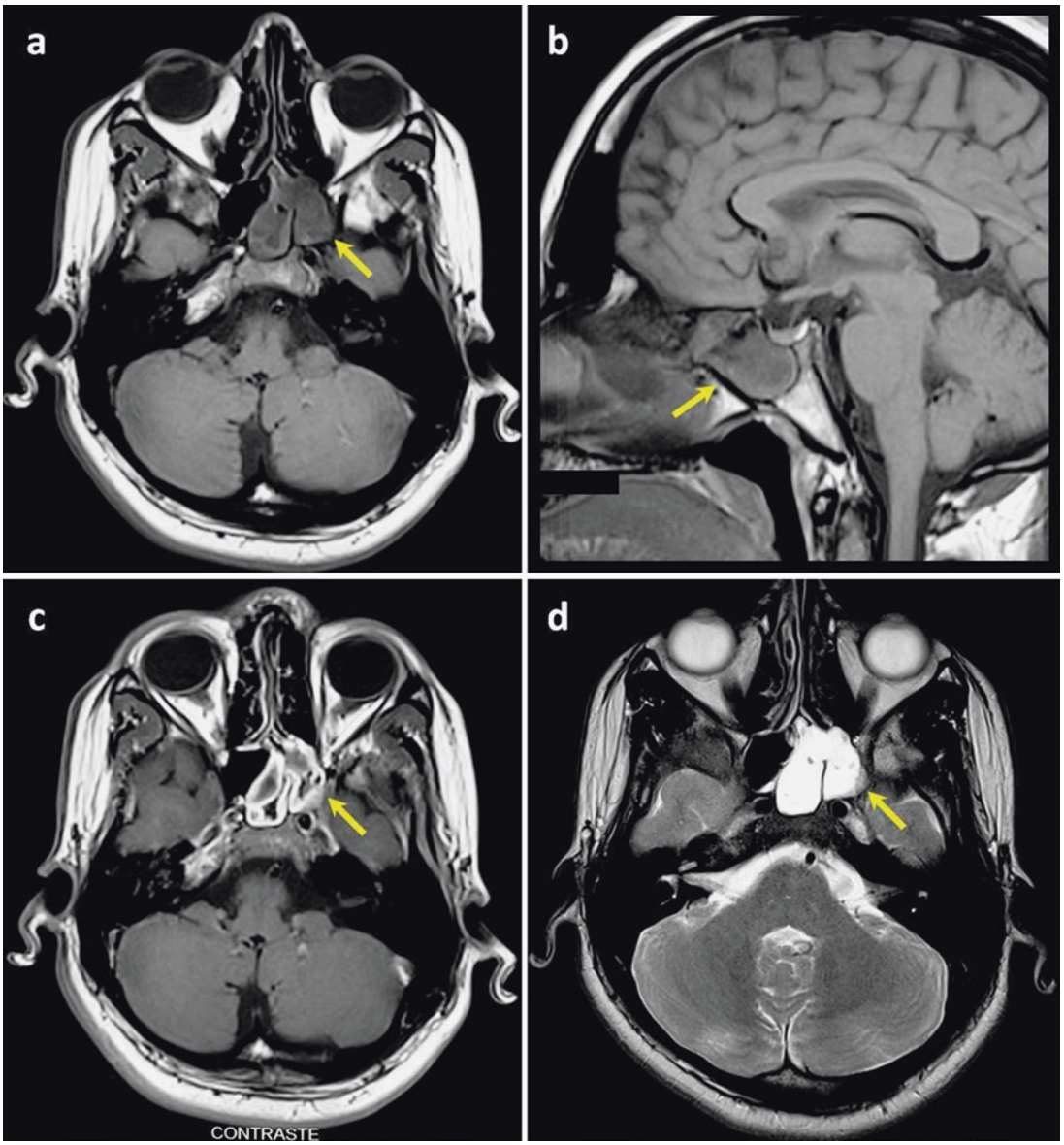


**Fig. 11.4** Axial (**a**) and sagittal (**b**) T1-weighted MRI without gadolinium administration. Axial T1-weighted gadolinium-enhanced MRI (**c**) and T2-weighted image (**d**) showing an isolated, chronic sphenoidal sinusitis in an asymptomatic patient (*arrow*)

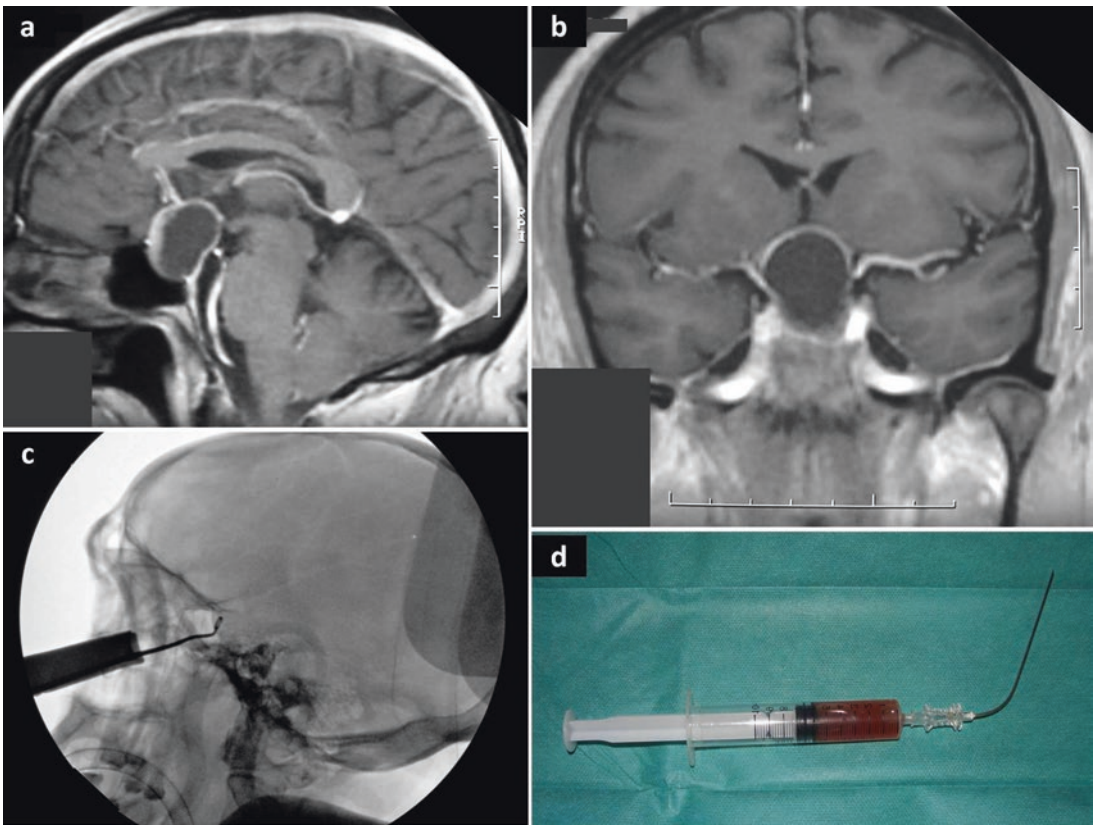


**Fig. 11.5** A cystic pituitary adenoma mimicking a pituitary abscess. (**a**, **b**) Coronal and sagittal T1-weighted MRI after gadolinium administration. (**c**) Fluoroscopic control during the transsphenoidal surgical procedure: The surgical instrument is noted passing toward the sphenoid sinus through a speculum placed in the nasal cavity. (**d**) Operative photography of the sterile fluid collection drained from the intrasellar cystic tumor

## Suggested Reading


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# Orbital Abscesses

# 12

Orbital abscess is a serious form of orbital infection. The suppurative collection may be found along the orbital walls (subperiosteal abscess) or in the extraconal and intraconal spaces (true intraorbital abscess). Infection from contiguous structures (especially paranasal sinusitis) is the most common predisposing factor (Figs.12.1, 12.2, 12.3, 12.4, and 12.5). Most patients present with painful, unilateral periorbital swelling and erythema, chemosis, proptosis, visual impairment, and ophthalmoplegia. Systemic features include fever and malaise. Fungal infections may have a more chronic, indolent presentation. Neurologic manifestations are associated with intracranial spread of the infection. CT scan remains the imaging investigation of choice for the diagnosis. A complementary MRI may be needed in cases with intracranial involvement and/or in the differential diagnosis of other orbital diseases. Ultrasonography is useful to assess the response to treatment. The most common microorganisms are those encountered in paranasal sinusitis. Fungal pathogens are rare. Orbital abscesses generally require surgical drainage (Figs. 12.6, 12.7, 12.8, 12.9, and 12.10). Conservative management consisting of intravenous antibiotics and close ophthalmological and clinical monitoring may be suggested for small suppurative collections with no signs of complication, but surgery should be reconsidered if complications arise. The outcome is generally good but is related to the severity of intracranial infections and the delay in diagnosis (potential blindness).

## Epidemiology and Etiology

Orbital infections include a wide variety of disease processes; based on the location and extension of the infection, they range from pre-septal cellulitis to cavernous sinus thrombosis. According to Chandler's classification system of orbital infections (Table 12.1), orbital abscesses correspond to Type III (subperiosteal abscess) and Type IV (true intraorbital abscess). The periosteum plays an important role against the progression of the infection to the orbital space and later to the intracranial contents.

Orbital abscess is frequently secondary to spread of infection from contiguous structures such as paranasal sinuses (especially ethmoids and maxillary sinuses), ear, teeth, face, scalp, lids, and lachrymal gland. Direct inoculation is more unusual, including fractures/trauma, orbital foreign bodies, and ocular surgeries. The indirect mode of spread is by infective thrombophlebitis via the anastomotic valveless venous system. Endogenous spread is rare and is mainly seen in immune-compromised patients.

Visual loss represents the most serious local complication and can occur due to corneal damage, persistent raised intraocular pressure, central retinal artery occlusion, or septic, inflammatory, or ischemic optic neuropathy.

All age groups may be affected, but orbital cellulitis is most commonly seen in children, and orbital abscess is more prevalent in young adults.

## Clinical Presentations

Orbital abscess is characterized by unilateral, painful periorbital swelling and edema, erythema, conjunctival chemosis, proptosis, visual impairment, and restricted ocular motility (ophthalmoplegia). Systemic features include fever and malaise. A chronic, indolent presentation can be seen in cases of fungal or tuberculous infections.

Signs and symptoms vary depending upon the size and site of the abscess, the duration of the disease, the virulence of the causative pathogens, and local or general predisposing factors. It is important to consider the patient's past medical history and predisposing factors.

The occurrence of neurologic signs and symptoms is associated with intracranial spread of the infection.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_12

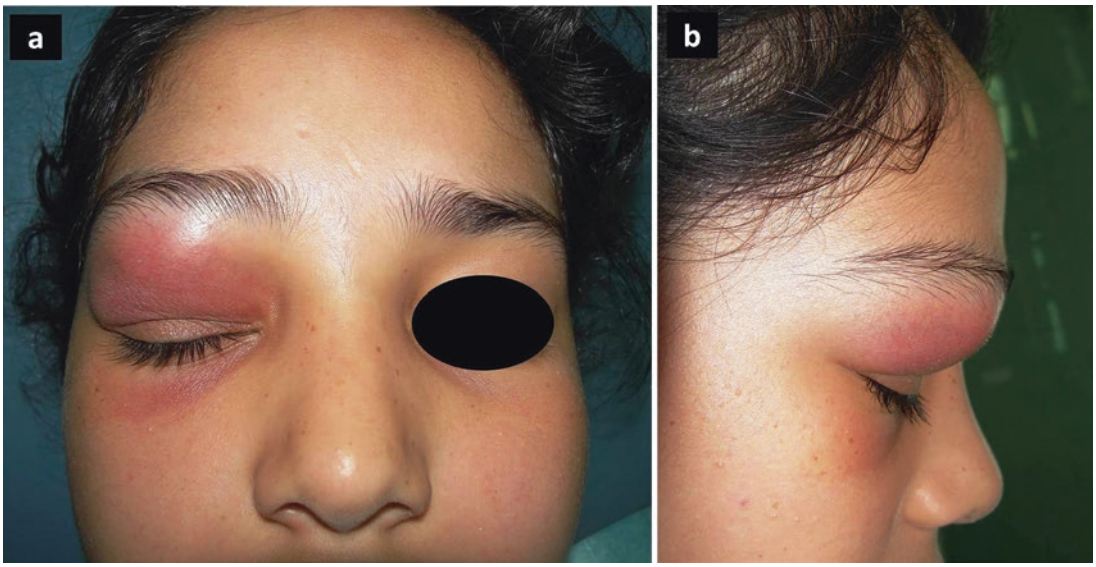


**Fig. 12.1** Case 12.1. Photograph of a 13-year-old girl who was inadequately treated for a paranasal sinusitis 2 weeks previously: frontal (**a**) and lateral (**b**) views. There is right superior eyelid edema and erythema

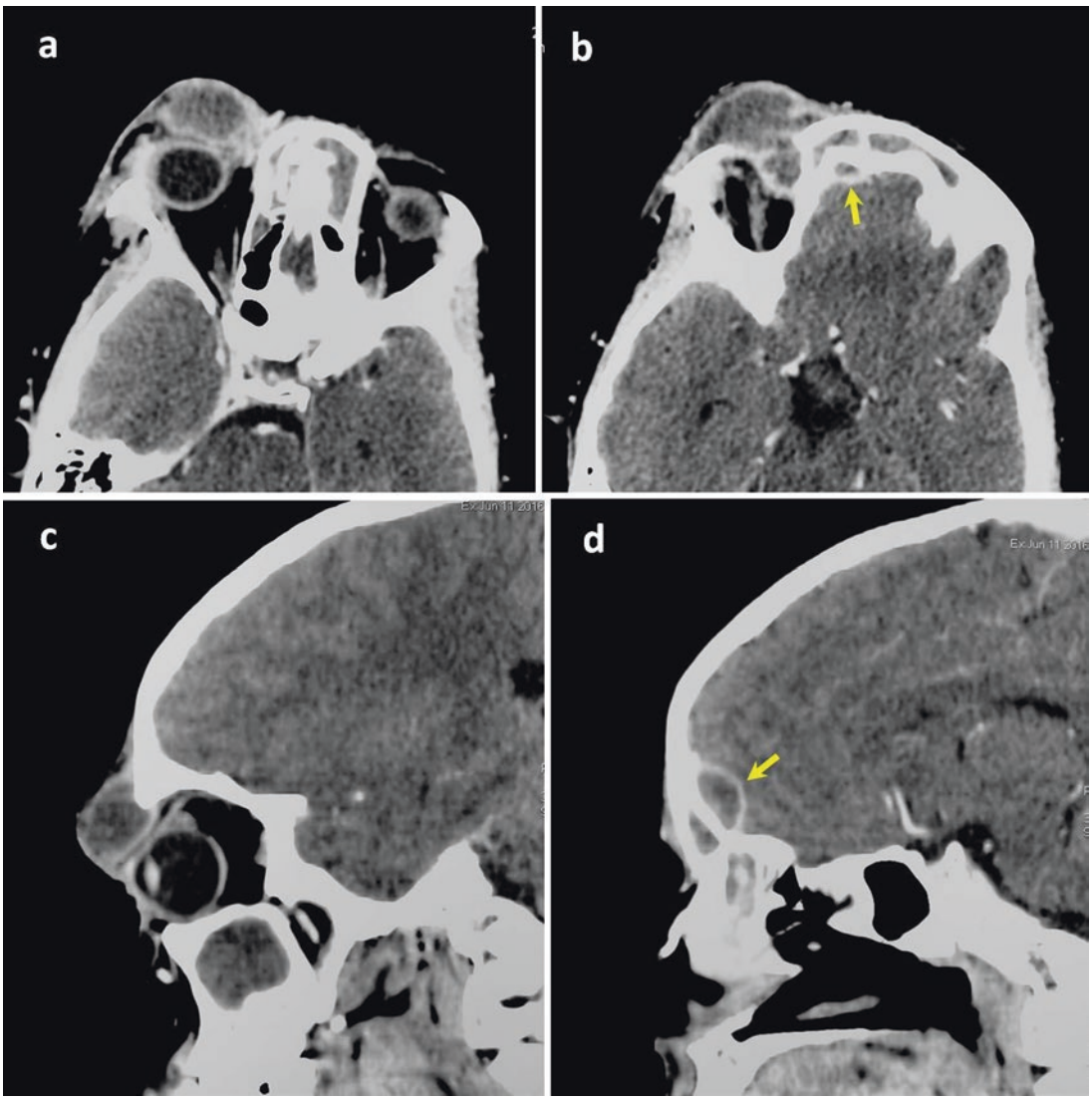


**Fig. 12.2** Case 12.1. Axial (**a**, **b**) and sagittal (**c**, **d**) cranio-orbital post-contrast CT scans showing a right superior palpebral abscess with mild orbital extension and ocular globe compression. Note the extensive paranasal sinusitis and the cranial frontal epidural abscess (*arrow*)

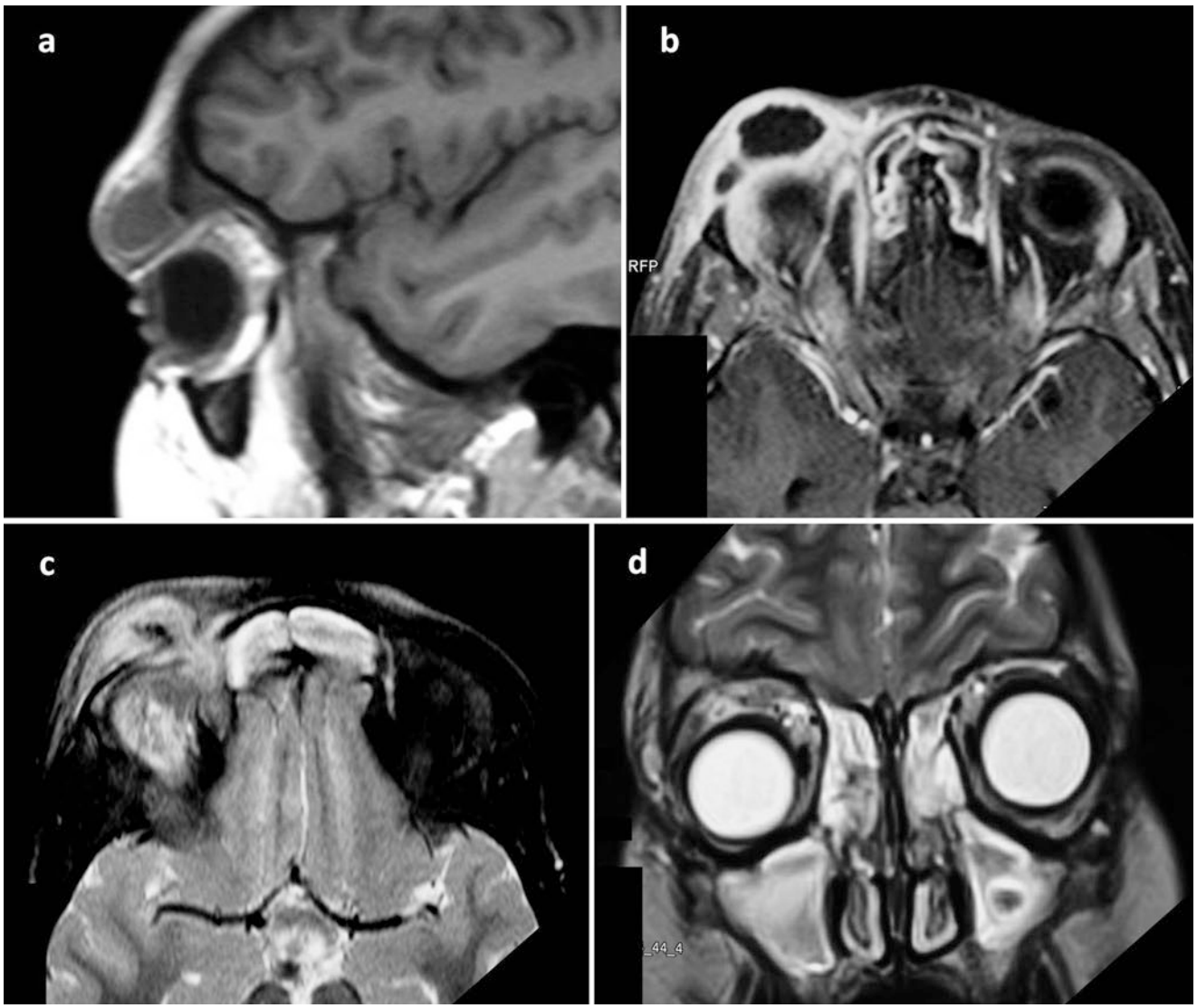


**Fig. 12.3** Case 12.1. Sagittal T1-weighted image without gadolinium injection (**a**), axial T1-weighted image with gadolinium injection (**b**); axial (**c**) and coronal (**d**) T2-weighted MRI in the same patient

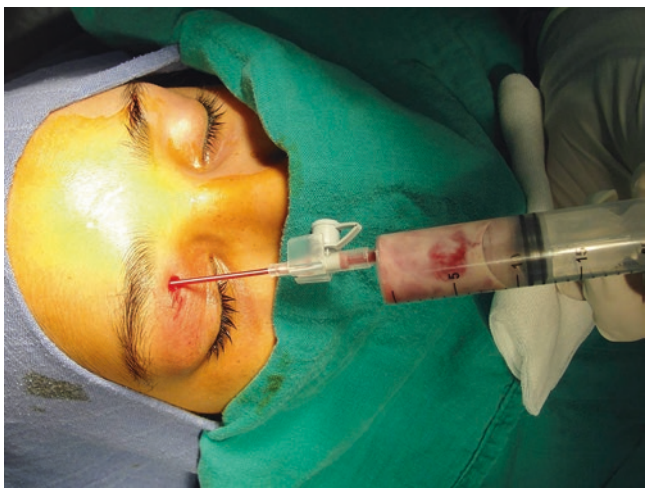

**Fig. 12.4** Case 12.1. Operative view. A thick, yellowish green fluid was percutaneously drained under local anesthesia

## Imaging Features

Craniofacial radiography may reveal paranasal sinusitis with sinus opacification, mucosal thickening, and presence of a fluid level.

On CT scan, orbital abscess is seen as a low-density collection along the orbital walls (subperiosteal abscess) or into the extraconal and intraconal spaces (true intraorbital abscess) with contrast-enhancing margin (Fig. 12.11). Whereas orbital cellulitis is characterized by elimination of orbital fat shadows, intraorbital gas or a fluid-gas level is strongly evocative of abscess formation. Associated findings on CT scan include paranasal sinusitis (especially anterior ethmoid and maxillary sinusitis), intracranial suppurative collections, cranial osteomyelitis, and cavernous sinus thrombosis.

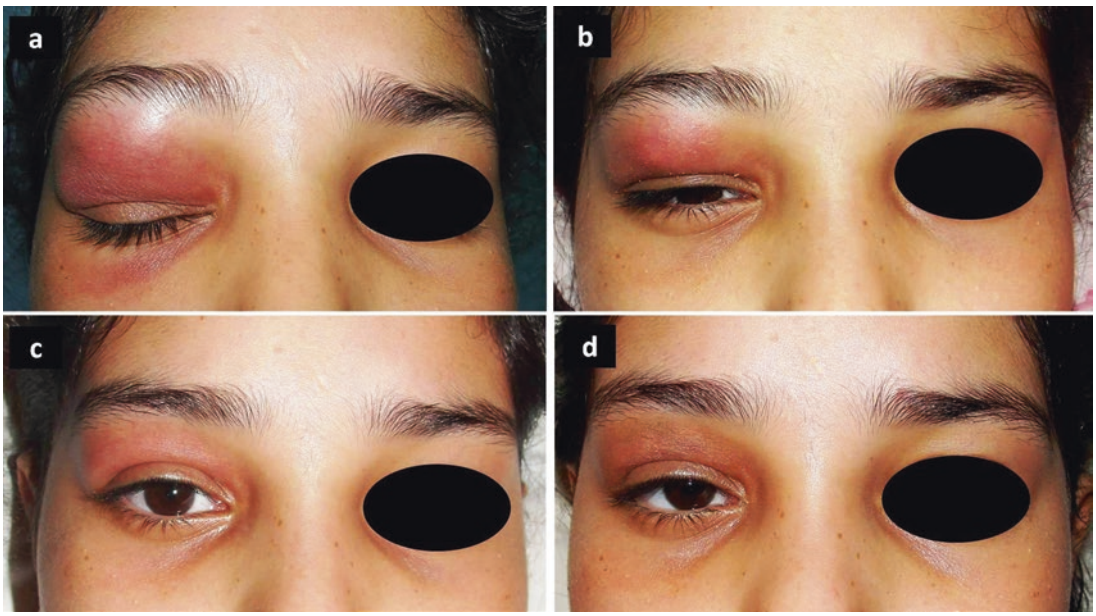


**Fig. 12.5** Case 12.1. Postoperative evolution under adapted antibiotherapy: Before treatment (**a**) and at 1 week (**b**), 2 weeks (**c**), and 4 weeks (**d**)

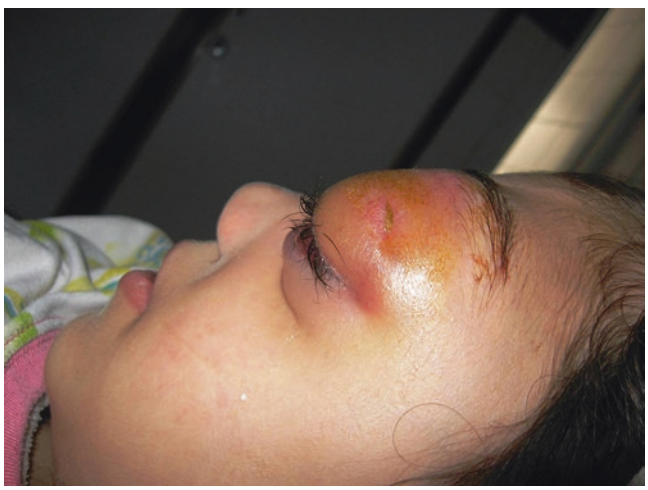

**Fig. 12.6** Case 12.2. Lateral photograph of an 11-year-old girl who was adequately treated for dental caries 2 weeks previously. There is *left* superior palpebral swelling with erythema and proptosis

Ultrasonography is useful in delineating anteriorly located orbital suppurative collections.

A complementary MRI is often necessary, particularly in cases with intracranial involvement or for the differential diagnosis of other orbital diseases.

## Laboratory Findings

Usual inflammatory parameters may help in the diagnosis but are nonspecific and highly variable in their expression.

The most common microorganisms found in orbital infections are species of *Streptococcus*, *Staphylococcus*, *Haemophilus influenzae* type b, and anaerobic bacteria, pathogens that represent the bacterial species encountered in paranasal sinusitis. Polymicrobial infections are seen more frequently in adults, but tuberculous and fungal infections (especially *Aspergillus* species and mucormycosis) are rare.

Blood cultures are rarely positive, especially if the patient has already received antibiotic drugs. *Pseudomonas aeruginosa* and *Proteus mirabilis* are reported in association with systemic bacteremia in patients with diabetes mellitus, acute leukemia, or cholecystitis. The collection and processing of samples from other potential sources of infection are important for identification of causative pathogens.

## Treatment Options

Classically, types I and II in Chandler's classification are usually treated conservatively, whereas types III, IV, and V are generally treated surgically.

Medical therapy should include broad-spectrum antibiotic drugs against aerobic and anaerobic bacteria. Initial antibiotics must consist of a third-generation cephalosporin combined with metronidazole. Depending of the source of infection and based on the drug sensitivity report, other antibiotics may be used, such as flucloxacillin, vancomycin, and/or fosfomycin. The optimal duration of antibiotic therapy varies, with 3 weeks as the minimal suggested duration. In case of fungal infection, amphotericin B or voriconazole is required.

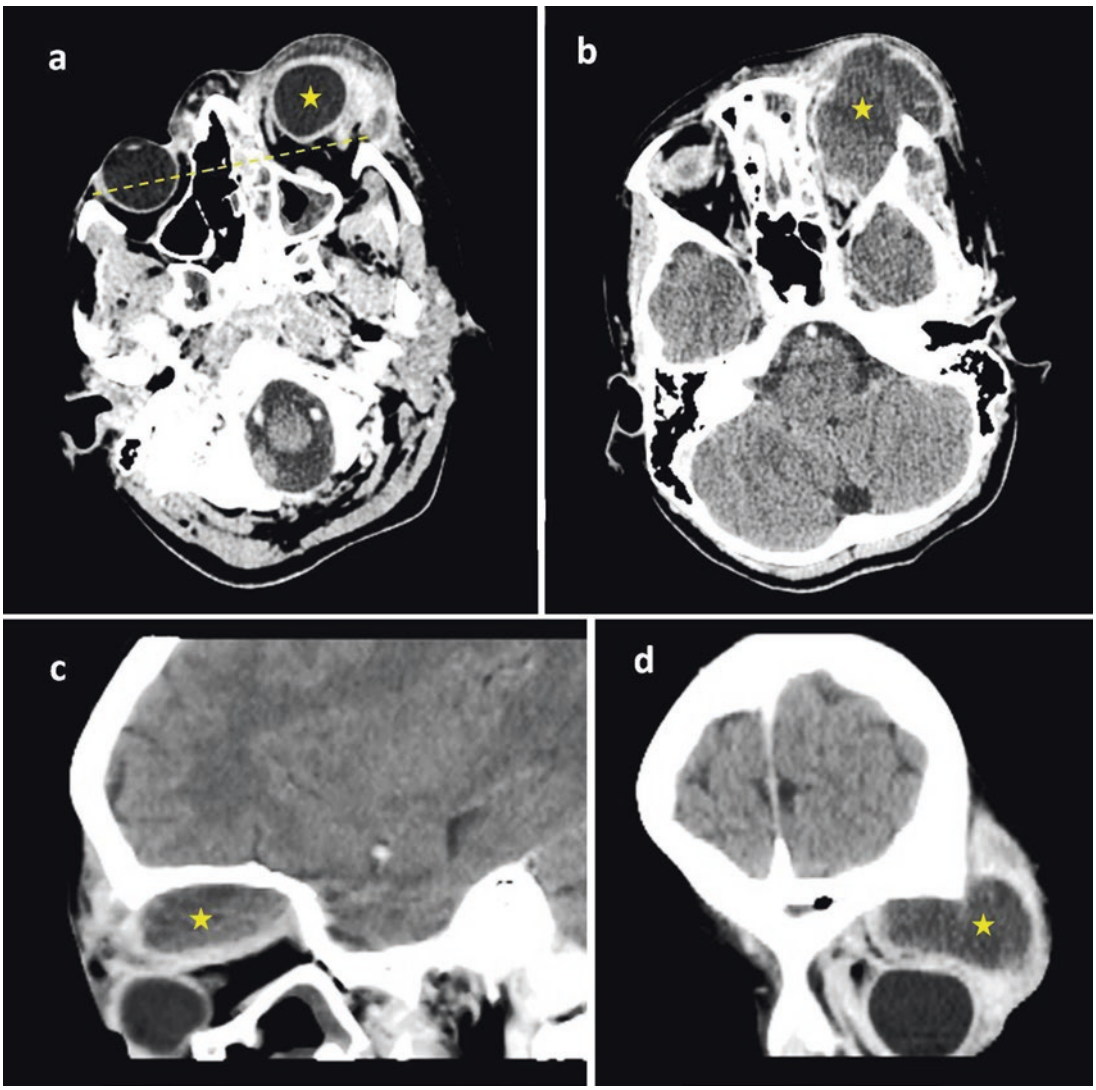


**Fig. 12.7** Case 12.2. Left orbital bacterial abscess. Cranio-orbital post-contrast CT scan: Axial (**a**, **b**), sagittal (**c**), and coronal (**d**) views. This large abscess (*star*) was associated with intraorbital cellulitis and adjacent ethmoido-maxillary paranasal sinusitis

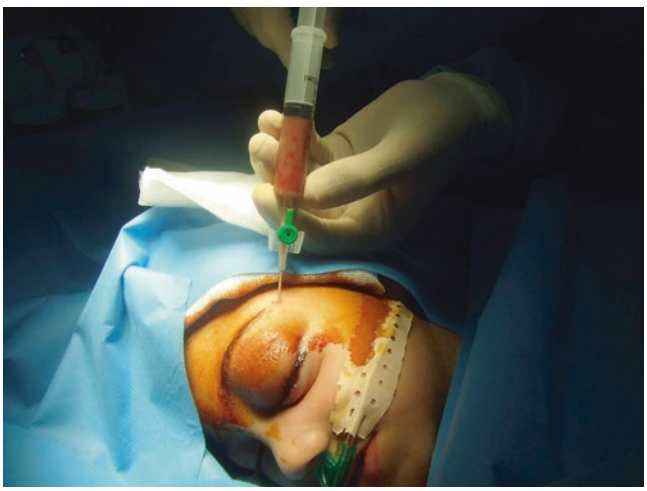

**Fig. 12.8** Case 12.2. Operative photograph. The suppurative collection was percutaneously drained under local anesthesia

Patients with orbital abscesses require surgical drainage. Conservative management consisting of intravenous antibiotics and close ophthalmological and clinical monitoring may be suggested for small suppurative collections without any signs of complication.

The most important surgical objectives are to drain the pus sufficiently, decrease intraorbital tension, and take samples for culture. Retained wood or other foreign bodies in posttrauma patients requires orbital surgery for foreign body removal. Surgical drainage is indicated in several circumstances:

- Disease progression within 48 h despite adequate antibiotic therapy
- Suspected visual compromise
- Abscess volume larger than 1,250 $mm^3$

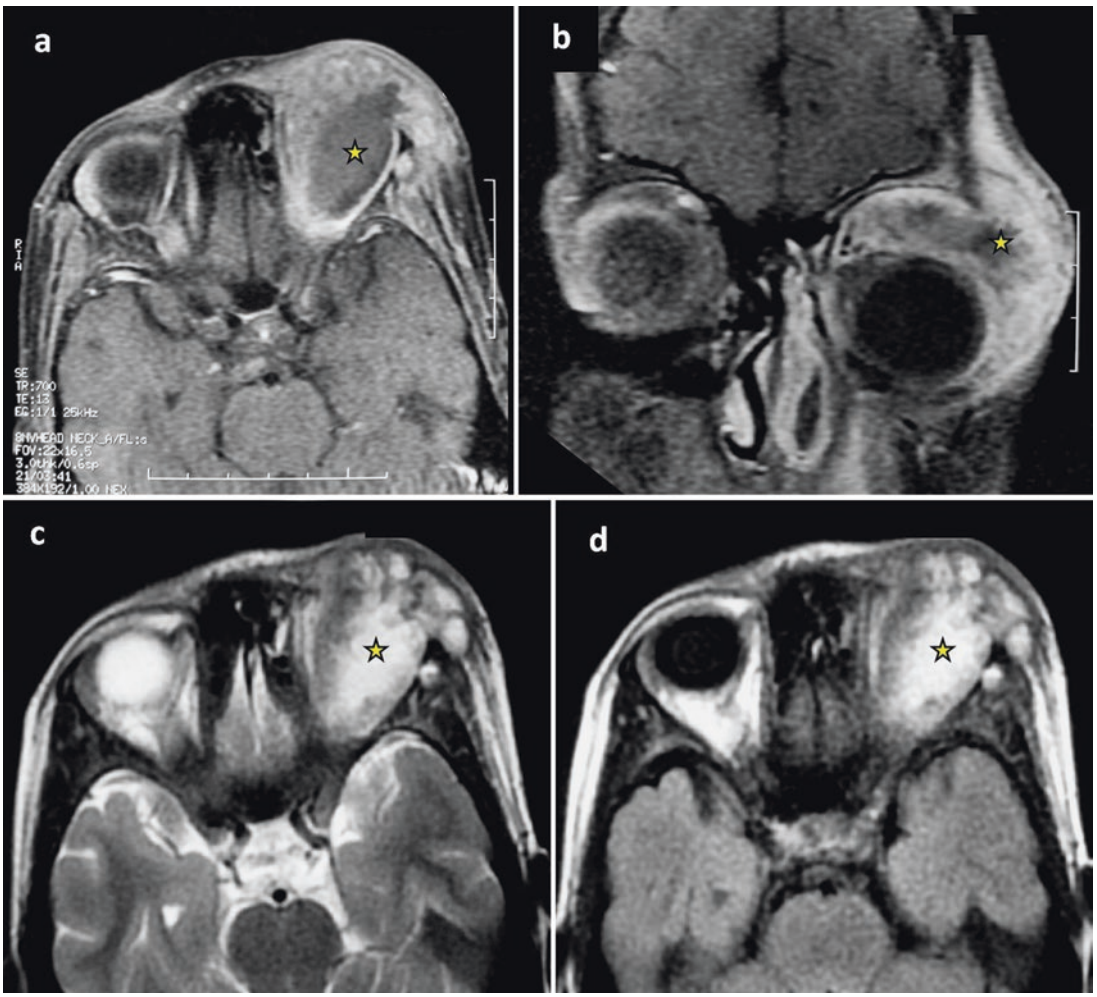


**Fig. 12.9** Case 12.2. Cranio-orbital MRI performed 2 days following the first (incomplete) drainage. Axial (**a**) and coronal (**b**) T1-weighted MRI with gadolinium administration, axial T2-weighted image (**c**), and FLAIR sequence (**d**) showing the abscess collection (*star*) with ring enhancement

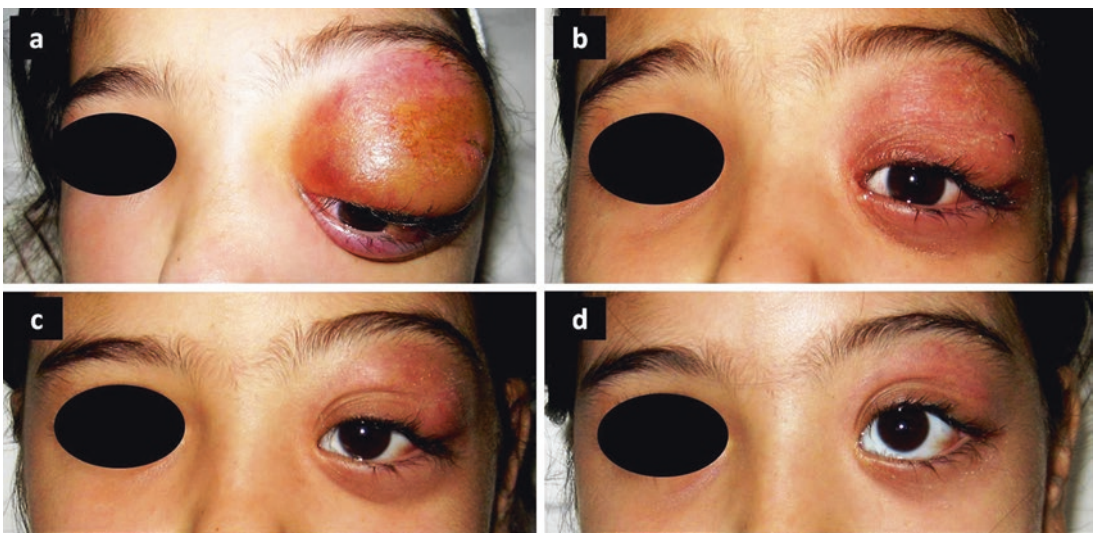


**Fig. 12.10** Case 12.2. Postoperative evolution under adapted antibiotherapy: Before treatment (**a**) and at 1 week (**b**), 2 weeks (**c**), and 4 weeks (**d**)

- Significant proptosis
- Immunosuppression

Transnasal endoscopic drainage may be suggested for patients with medial or medial-inferior orbital abscess. This mini-invasive technique also facilitates concomitant drainage of paranasal sinusitis. Craniotomy may be done in some patients with intracranial suppurations.

**Table 12.1** Location and extension of orbital infections (Chandler's classification system)

| Type | Description |
|---|---|
| I | Inflammatory edema (pre-septal cellulitis) |
| II | Orbital cellulitis (post-septal cellulitis) |
| III | Subperiosteal abscess |
| IV | Orbital abscess |
| V | Cavernous sinus thrombosis |

An integrated multidisciplinary approach (involving ophthalmologists, radiologists, otorhinolaryngologists, neurosurgeons, infectious disease specialists, and microbiologists) is the key to successfully managing this infectious disease (Fig. 12.12).

## Outcomes

Generally, visual acuity is recovered a few days after drainage, though the return of ocular motility and complete recovery may take up to 2 months.

Delay in treatment can result in blindness (loss of vision), bone involvement, cavernous sinus thrombosis, contralateral eyelid edema and ophthalmoplegia, intracranial suppuration, meningitis, septic embolism, neurologic sequelae, generalized sepsis, and even death.

Prevention of orbital infection is linked to the appropriate treatment of the conditions that may lead to this disease.

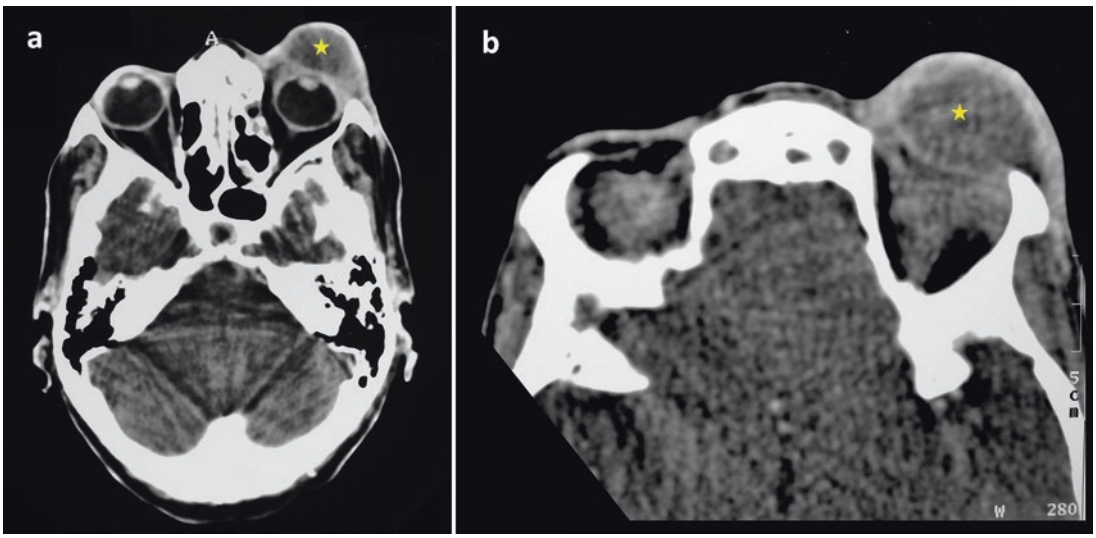


**Fig. 12.11** Left superior palpebral abscess without exophthalmos. (**a**, **b**), Axial cranio-orbital CT scans following contrast administration, showing the suppurative collection (*star*)

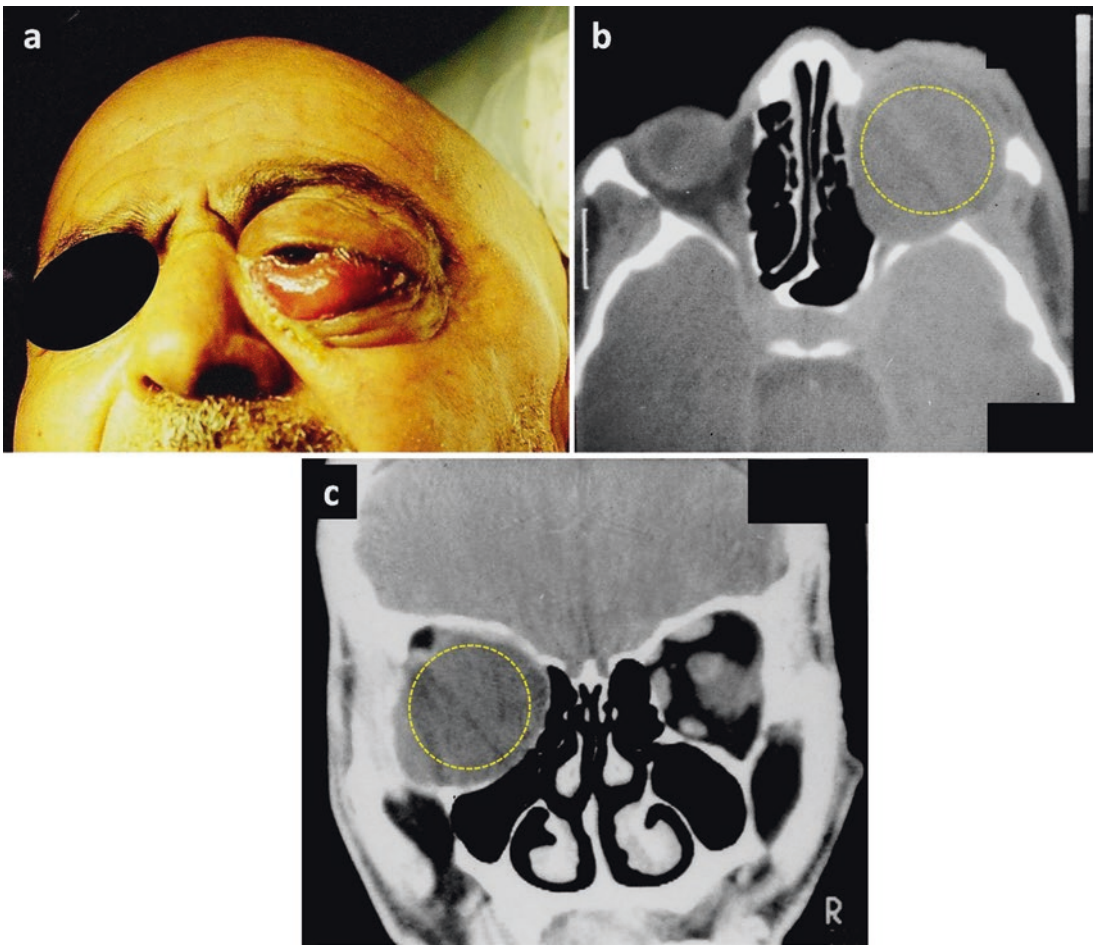


**Fig. 12.12** Clinical photograph of a patient with left orbital hydatid cyst (**a**). Axial (**b**) and coronal (**c**) cranio-orbital CT scan showing the large orbital hydatid cyst (*dotted circle*) occupying almost the whole of the orbit (*enlarged*)

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# Mucopyoceles

# 13

A mucopyocele is an infected paranasal sinus mucocele, which can extend into adjacent structures such as the orbit and/or the cranial cavity. Symptoms are due to anatomic mass effect. Systemic manifestations of infection are uncommon. Mucopyocele is best seen on CT scanning, which shows soft tissue opacification and expansion of the sinus, with thinning and loss of bone. Intravenous contrast typically shows ring enhancement. MRI is useful in identifying the relationship between the lesions and surrounding vital structures such as brain, orbit, and vascular structures. Histopathologic examination shows a cystic lesion lined with respiratory mucosa with pyogenic inflammation and suppurative necrosis. Isolation and identification of the causative bacteria is crucial for selecting the most appropriate antibiotic. Various surgical methods, including external or transnasal endoscopic approaches, are used to successfully eliminate the suppurative collection. Outcomes are usually excellent.

## Epidemiology and Etiology

A mucocele is a benign and expansile mucus-filled cystic lesion originating in the paranasal sinuses as a result of the chronic obstruction to sinus ostia. The mucus may become infected, forming a mucopyocele (an abscess-like formation with purulent material), and can extend into adjacent structures. Indeed, mucopyocele may invade the orbit, erode the skull base, and displace the ocular bulb and the frontal lobe.

The frontal and ethmoid sinuses are most commonly affected, followed by the sphenoid and maxillary sinuses. Risk factors for development of paranasal sinus mucoceles include prior surgery, trauma, sinusitis, allergy, osteoma, and radiation. Expansion of a mucocele may take place over several years, whereas it expands rapidly when associated with secondary infection leading to a mucopyocele. Adults are most often affected; mucopyocele is rarely seen in children.

## Clinical Presentations

Common clinical presentations are headache, facial swelling or deformity, dental pain, nasal obstruction, and orbital manifestations (periorbital pain, proptosis, ophthalmoplegia, diplopia, and visual loss). Systemic manifestations of infection are uncommon.

Intracranial extension by eroding the cranial bones can lead to CSF rhinorrhea, meningitis, empyema, brain abscess, raised intracranial pressure, and cranial nerve paresis.

## Imaging Features

Plain radiography of a frontoethmoidal mucopyocele shows an expanded frontal sinus with loss of the scalloped margin and translucence and depression or erosion of the supraorbital ridge. In the sphenoid, the sinus appears expanded, with elevation or erosion of the floor of the pituitary fossa.

Mucopyocele is best seen on CT scans, which show soft tissue opacification and expansion of the sinus, with thinning and loss of bone (Figs. 13.1, 13.2, 13.3, 13.4, and 13.5). There may also be evidence of bony remodeling and sclerotic margin. Intravenous contrast typically shows ring enhancement.

MRI is useful in identifying the relationship between the lesions and surrounding vital structures like brain, orbit, and vascular structures (Figs. 13.6, 13.7, 13.8, 13.9, and 13.10). The content of the mucopyocele is often hyperintense on T2-weighted images, with variable intensity on T1-weighted images. Following gadolinium injection, a layer of mucosal enhancement is seen along the periphery.

Some lesions may mimic mucopyocele. These include acute or chronic sinusitis, polyps, retention cyst, dermoids, cholesterol granuloma, and a variety of benign and malignant cystic tumors.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_13

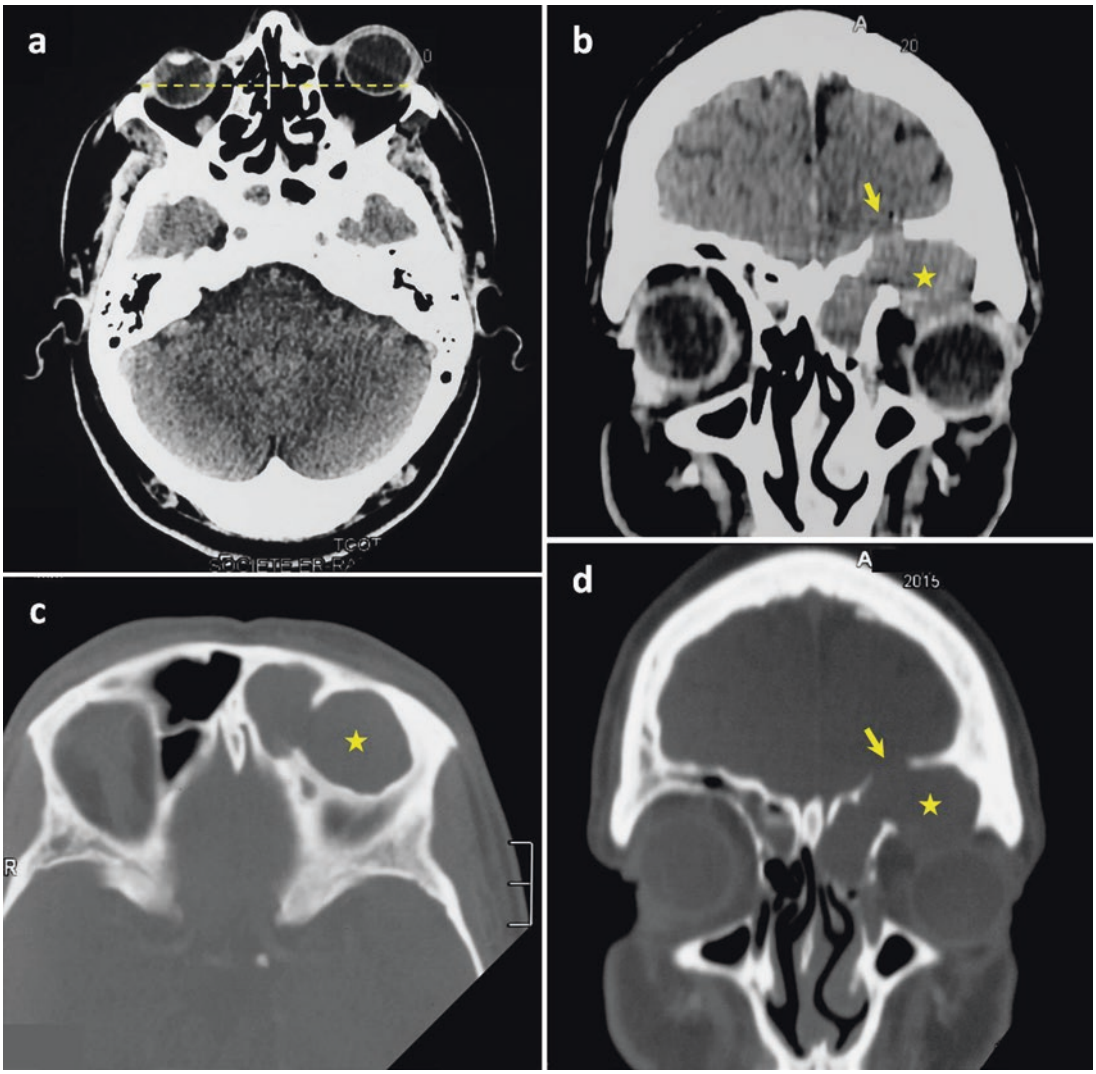


**Fig. 13.1** Case 13.1. Axial and coronal craniofacial CT scans on parenchymal (**a**, **b**) and bone (**c**, **d**) windows. There is a left frontoethmoidal mucopyocele (*star*) causing frontal sinus expansion, loss of the scalloped margin (*arrow*), and erosion of the supraorbital ridge with intraorbital extension (exophthalmos)

## Laboratory Findings

Usual biologic inflammatory parameters and elevation of white blood cell count are nonspecific and should be interpreted in combination with clinical and imaging features. Blood cultures are rarely positive.

Histopathology examination shows a cystic lesion lined with respiratory mucosa of pseudostratified columnar epithelium with pyogenic inflammation and suppurative necrosis (*see* Fig. 13.5). Cranial osteomyelitis and/or intracranial suppurative collections may be associated with extensive forms of the disease (Figs. 13.11 and 13.12).

The main aerobic bacteria are *Staphylococcus aureus*, alpha-hemolytic streptococci, *Haemophilus* species, and gram-negative bacilli. The principal anaerobes are *Peptostreptococcus* species, *Prevotella* species, *Fusobacterium* species, and *Propionibacterium acnes*.

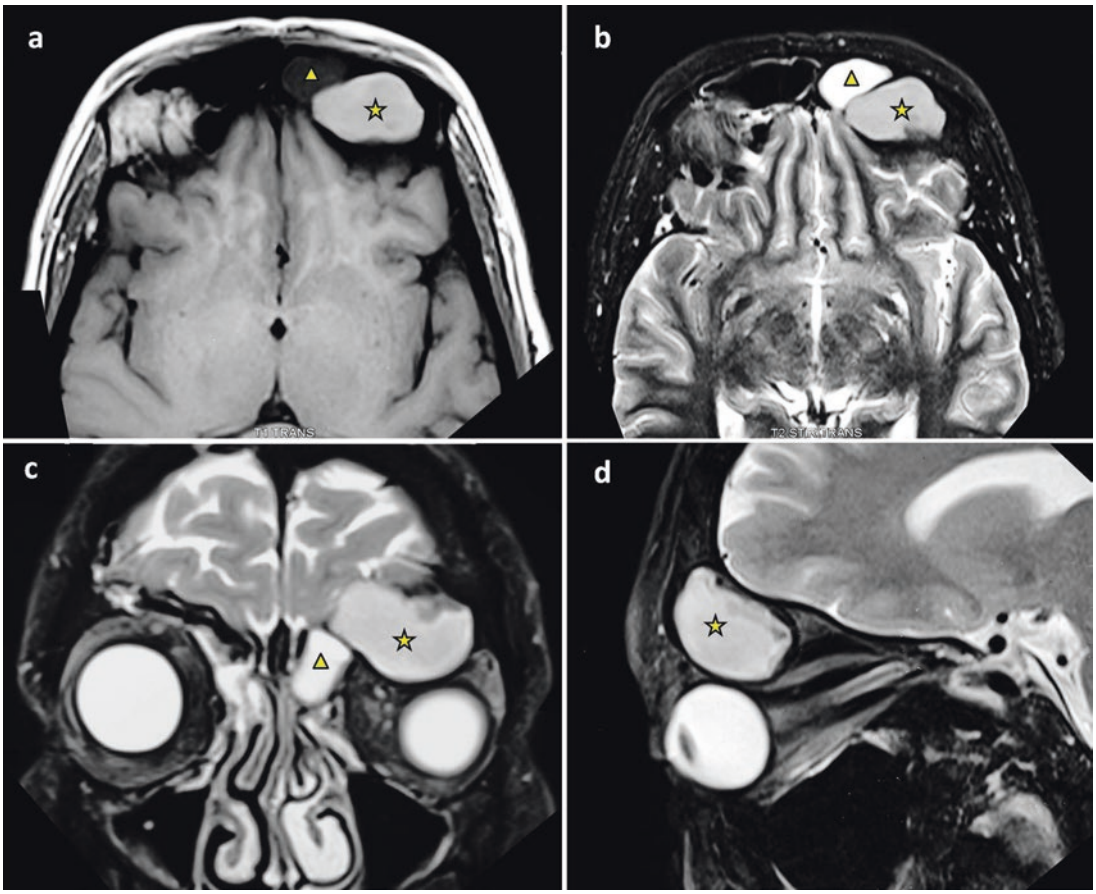


**Fig. 13.2** Case 13.1. Axial craniofacial T1-weighted MRI without gadolinium injection (**a**) and T2-weighted axial (**b**), coronal (**c**), and sagittal (**d**) MRI. The cystic lesion has two components: a mucocele (*triangle*) and a mucopyocele (*star*). The mucopyocele is slightly more hyperintense on the T1-weighted images and more hypointense on the T2-weighted images than the classic mucocele

## Treatment Options

The main principle of the surgical treatment of mucopyocele is to clear the suppurative cyst, completely remove the paranasal sinus mucosa (to avoid recurrence), and restore a plane of separation between the extracranial and intracranial space. Acceptable cosmetic results should be taken into account.

The surgical procedure ranges from mini-invasive approaches, such as transnasal endoscopic marsupialization and drainage (Figs. 13.13, 13.14, and 13.15), to more aggressive surgery like craniofacial exposure (*see also* Figs. 13.3, 13.4, 13.9) and transcranial extradural approach through a coronal flap and frontal sinus exclusion by fat tissue.

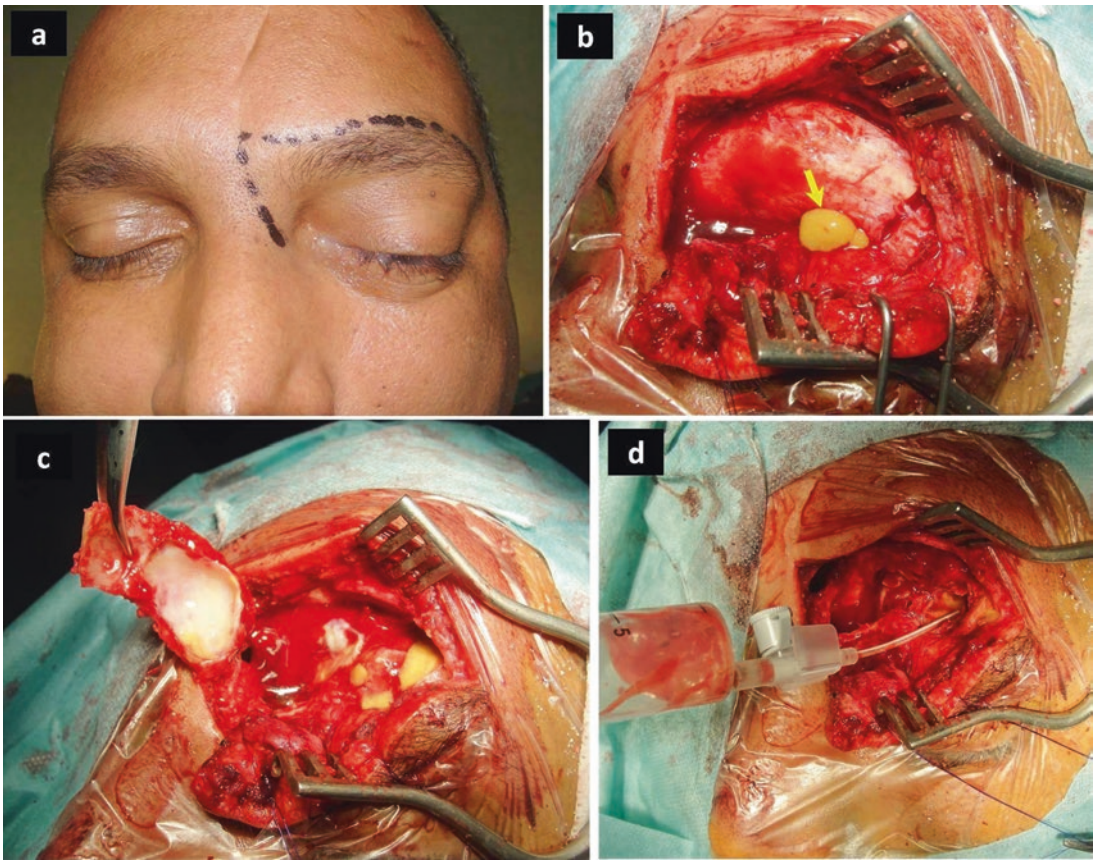


**Fig. 13.3** Case 13.1. The patient was operated on via a supraorbital limited-paralateronasal approach on the left side (**a**). The supraorbital ridge was exposed (**b**) and then cut (**c**). Note the wall hernia of the pyomucocele (*arrow*) (**b**). The cystic cloudy content was aspirated and sent for microbiological studies (**d**)

Appropriate surgical intervention and a close collaboration between the otolaryngologist, maxillofacial surgeon, ophthalmologist, and neurosurgeon are fundamental.

Intraoperative cultures for aerobic and anaerobic bacteria, fungi, and acid-fast bacilli should always be done. The antibiotic drugs should be adapted to the specific pathogen once antimicrobial susceptibilities are established. About 2 months of antibiotic therapy are typically required for bacterial abscesses.

## Outcomes

Outcomes are generally excellent, with satisfactory esthetic results. Most symptoms due to mass effect improve following drainage, and recurrence rates are low if the lesion is treated early and adequately. In more severe cases, the prognosis is related to that of intraorbital and intracranial infections.

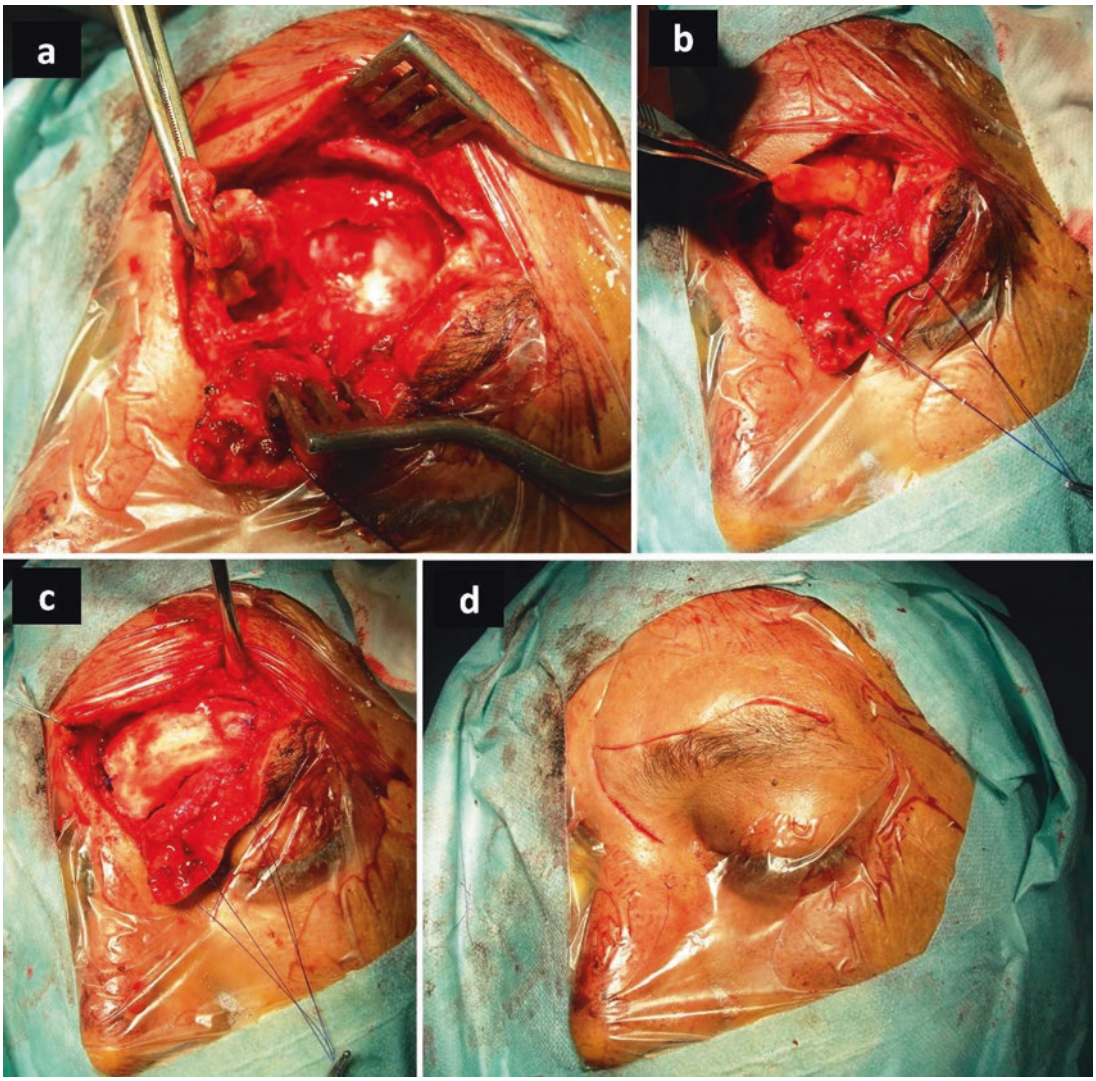


**Fig. 13.4** Case 13.1. Operative views. The cystic wall was completely removed (**a**) with placement of a piece of paraumbilical fat as an autograft (**b**). The supraorbital ridge was repositioned (**c**), and the incision was closed (**d**)

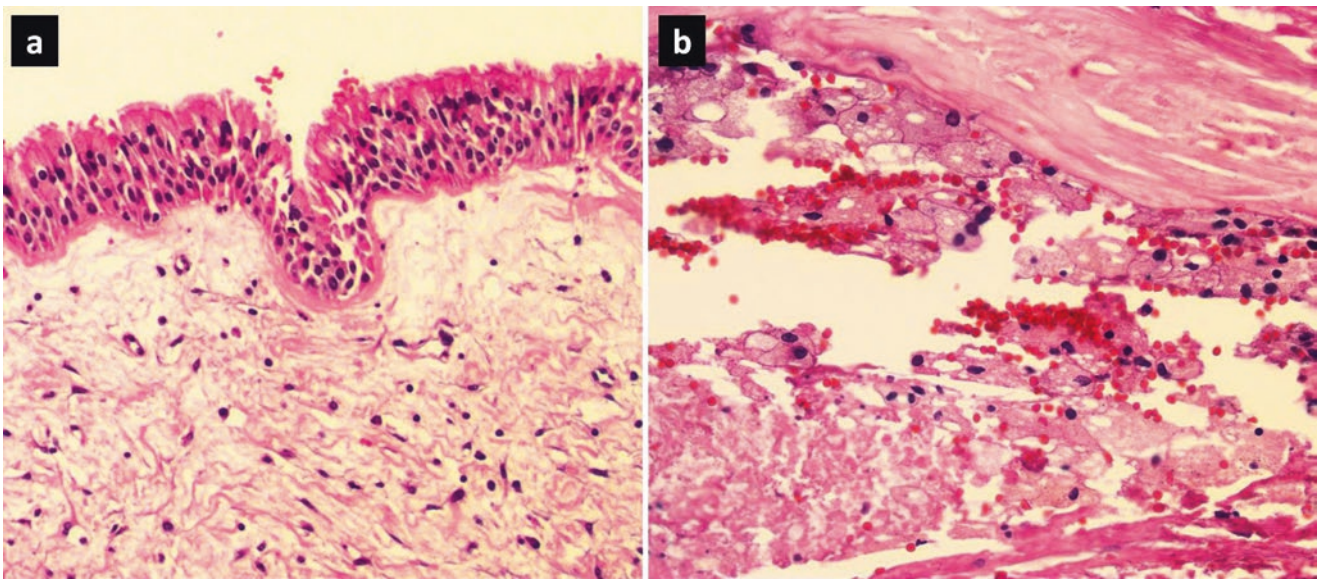


**Fig. 13.5** Case 13.1. (**a**) Photomicrograph showing a *thick*, fibrous-walled cyst lined by a ciliated respiratory mucosa (low-power magnification; hematoxylin-eosin staining). (**b**) Mucoid material with inflammatory cells and vacuolated macrophages (mucophages) (medium-power magnification; hematoxylin-eosin staining)

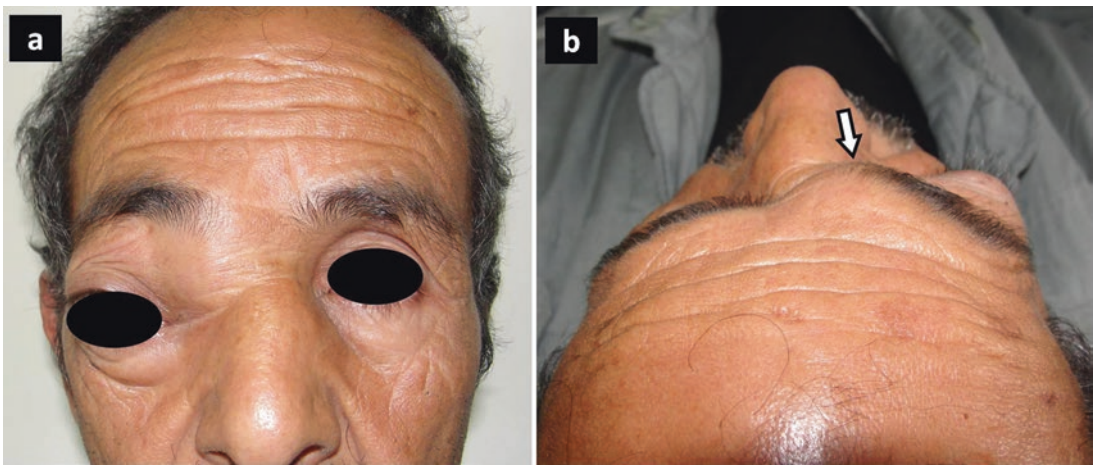


**Fig. 13.6** Case 13.2. This 55-year-old man had a chronic right proptosis with diplopia and recurrent episodes of fever. Clinical pictures show frontal (**a**) and superior (**b**) views. Note facial deformity and frontal swelling (*arrow*)

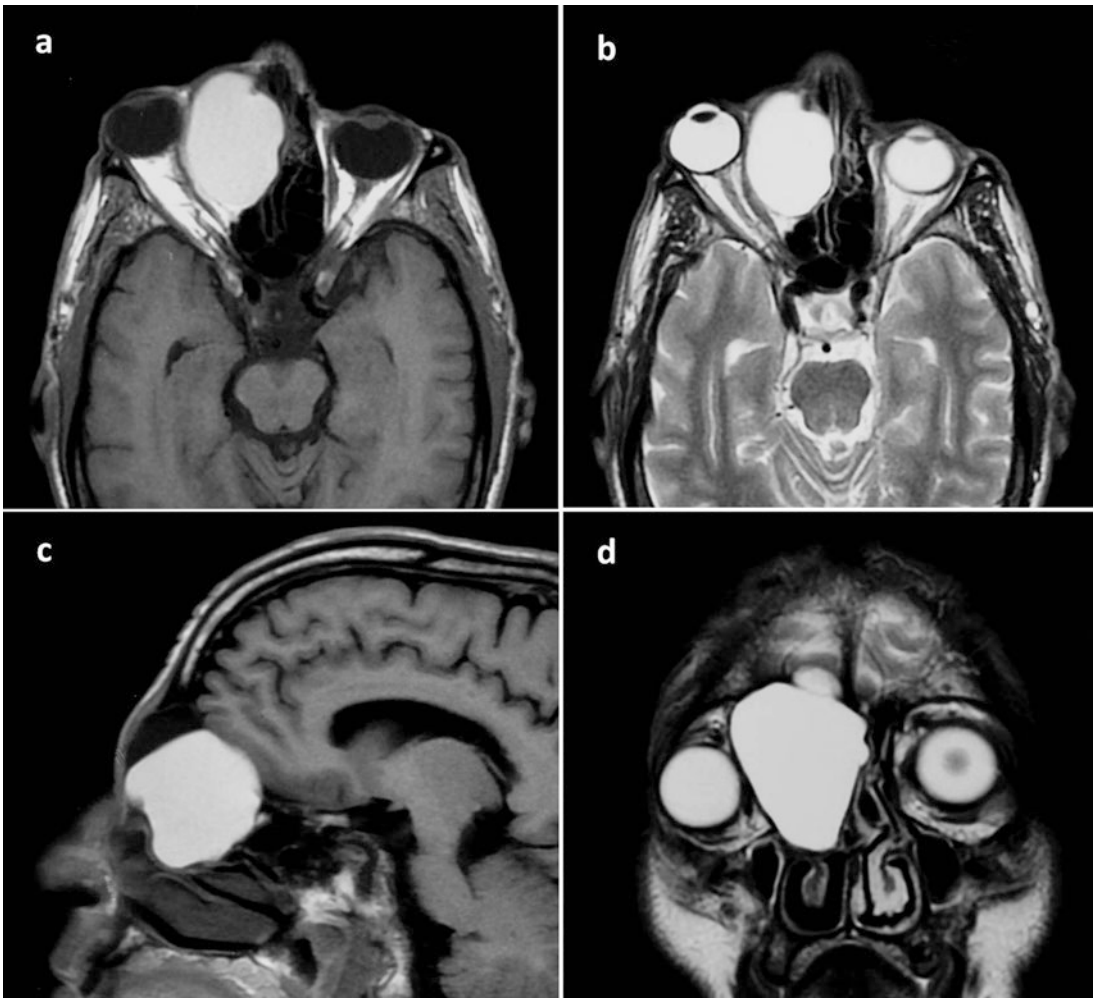


**Fig. 13.7** Case 13.2. Axial craniofacial T1-weighted (**a**) and T2-weighted (**b**) MR images. Sagittal T1-weighted (**c**) and coronal T2-weighted (**d**) MR images show the right frontoethmoidal cystic lesion with intraorbital extension

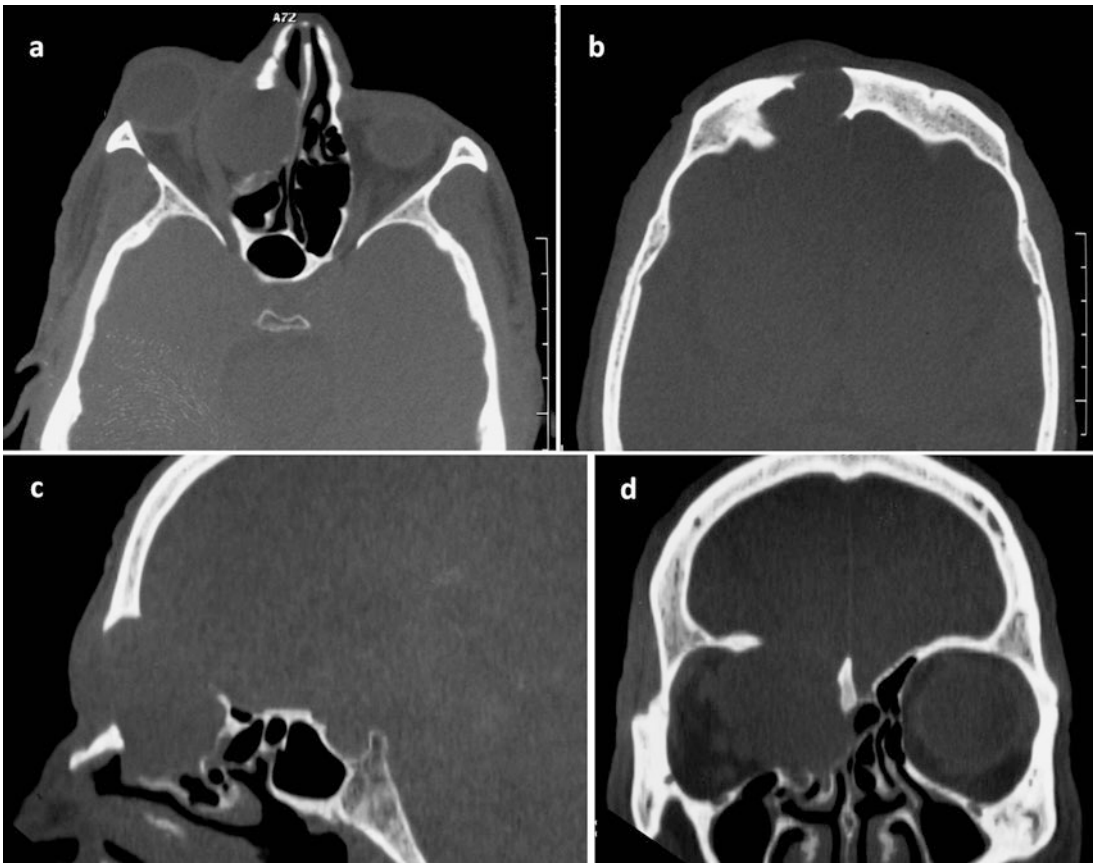


**Fig. 13.8** Case 13.2. Axial (**a**, **b**), sagittal (**c**), and coronal (**d**) craniofacial CT scans on bone windows. This cystic lesion causes thinning and loss of the surrounding bony structures, with intraorbital and intracranial extension

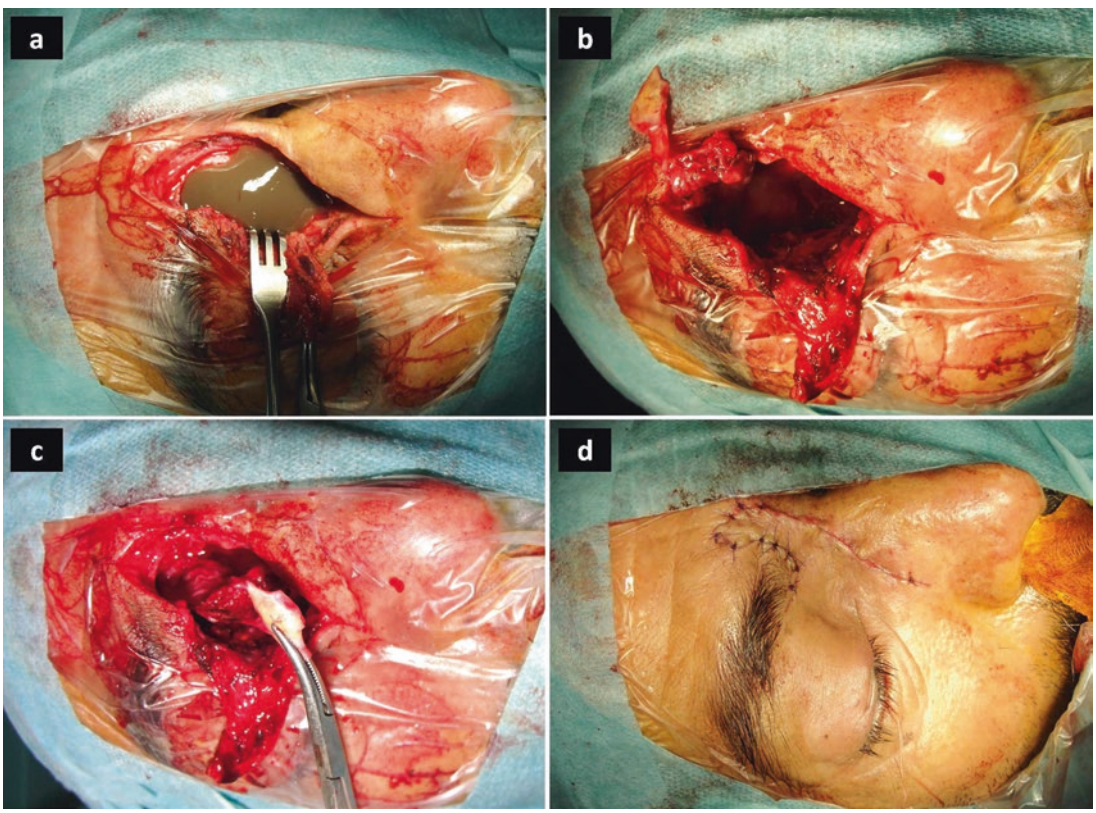


**Fig. 13.9** Case 13.2. Intraoperative views. A right paralateronasal approach was used. The *brownish* cystic content was aspirated and sent for microbiological studies (**a**). The cystic wall was completely removed (**b**, **c**), with placement of a piece of paraumbilical fat as an autograft. Then the incision was closed (**d**)

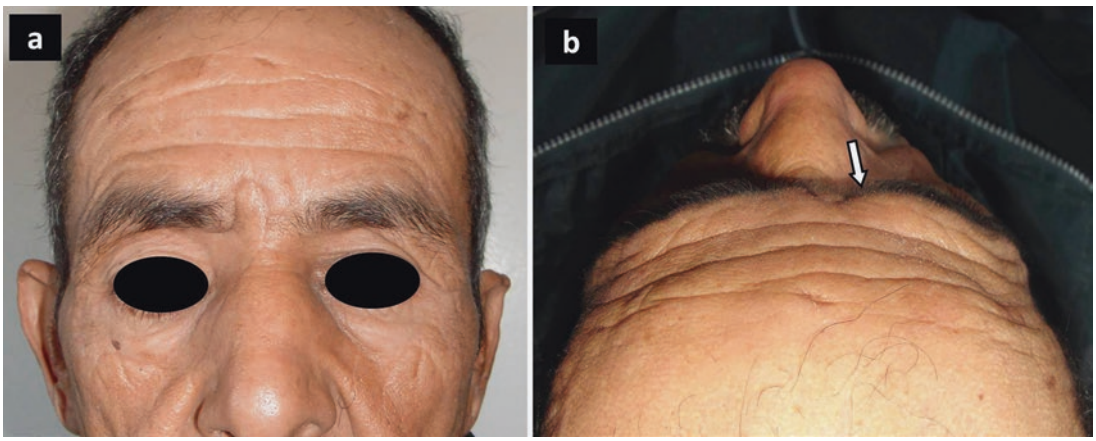


**Fig. 13.10** Case 13.2. Postoperative clinical picture 3 months later: frontal (**a**) and superior (**b**) views. The right proptosis and the frontal swelling (*arrow*) have disappeared

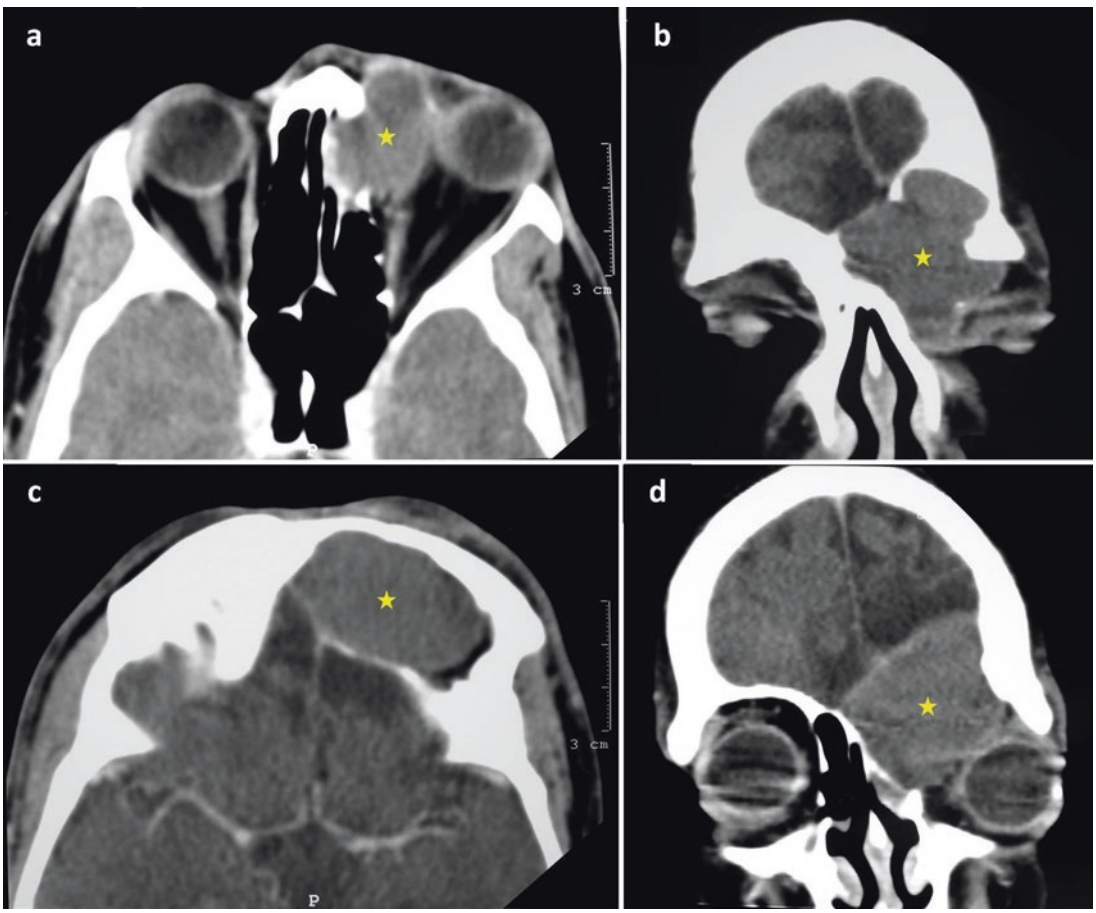


**Fig. 13.11** Case 13.3. Axial and coronal cranio-orbital CT scans before (**a**, **b**) and after (**c**, **d**) contrast administration. There is a *left* frontoethmoidal mucopyocele (*star*) causing frontal sinus expansion, loss of the scalloped margin, and erosion of the supraorbital ridge with intraorbital and intracranial extension. Intravenous contrast shows mild ring enhancement (**c**, **d**)

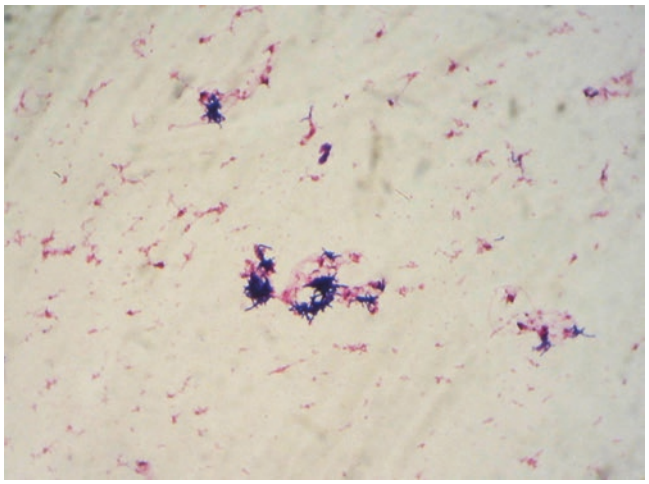

**Fig. 13.12** Case 13.3. Bacterial mucopyocele. Gram-positive filamentous bacillary forms consistent with *Actinomyces* species (gram staining)

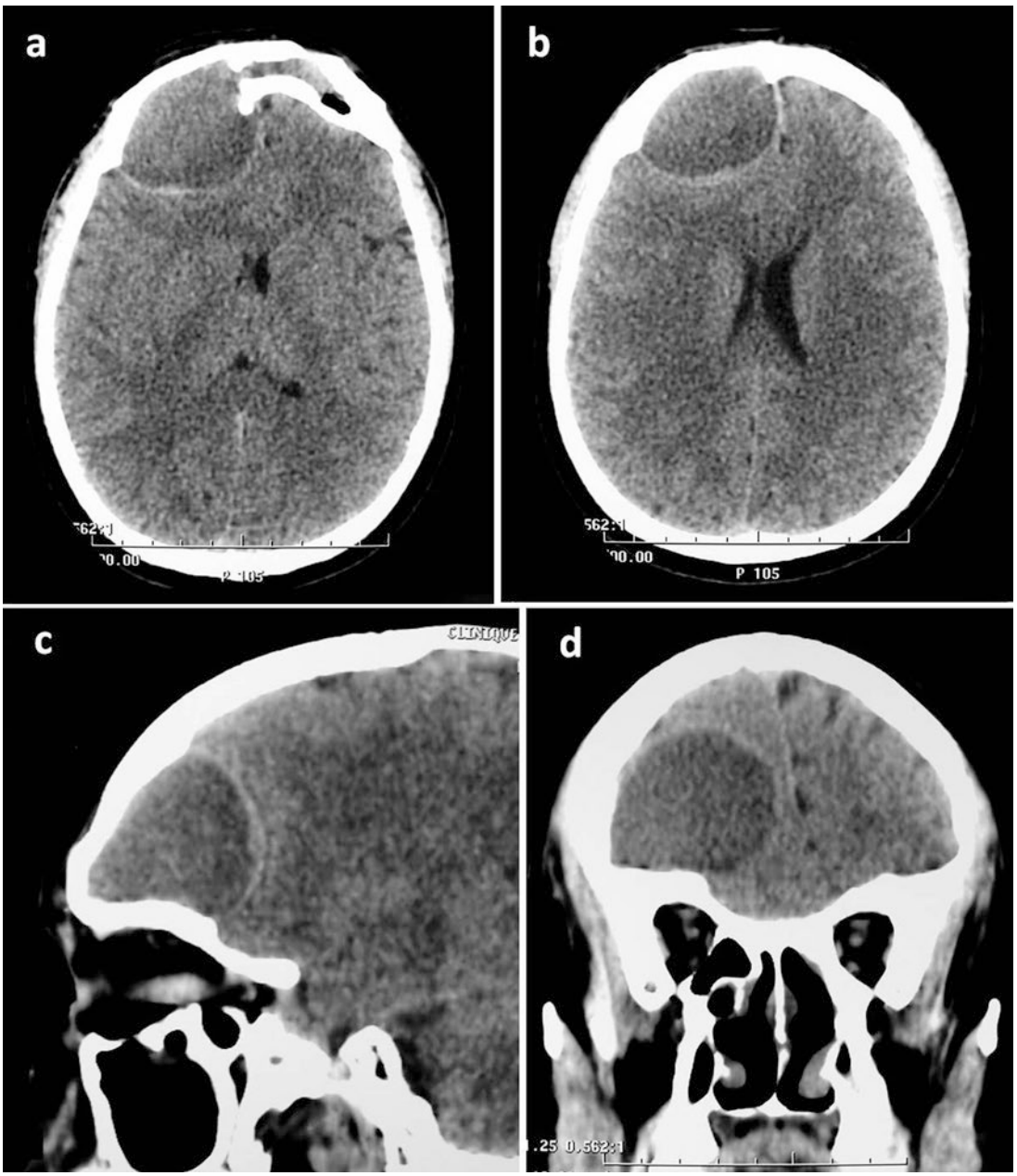


**Fig. 13.13** Case 13.4. Large frontal supraorbital, extradural pyomucocele on the right side. Axial (**a**, **b**), sagittal (**c**), and coronal (**d**) cranial CT scans without contrast injection

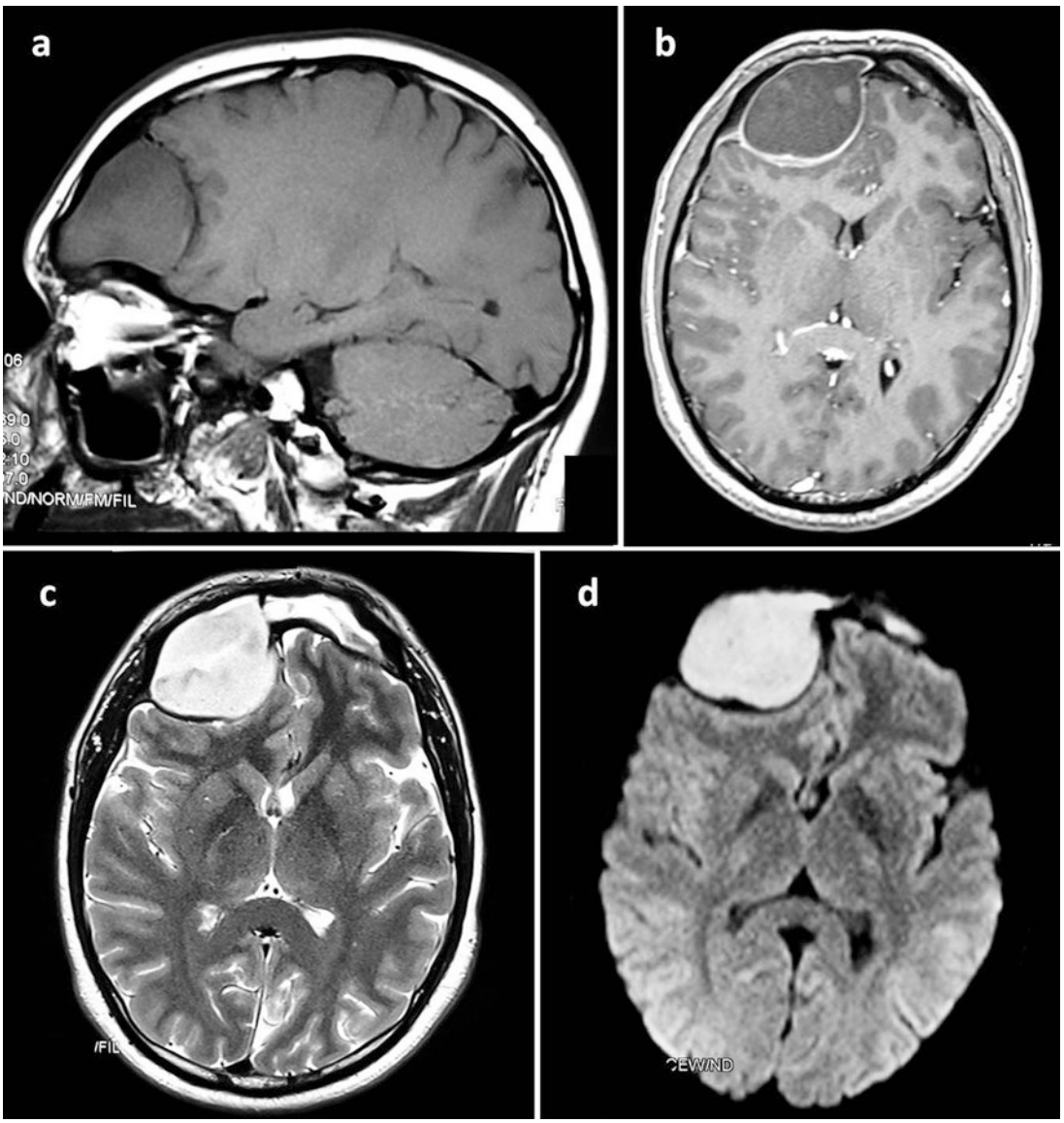


**Fig. 13.14** Case 13.4. Sagittal T1-weighted MRI without gadolinium administration (**a**), axial post-gadolinium T1-weighted image (**b**), axial T2-weighted image (**c**), and diffusion sequence (**d**) showing the cystic lesion with brain compression and peripheral enhancement (**b**)

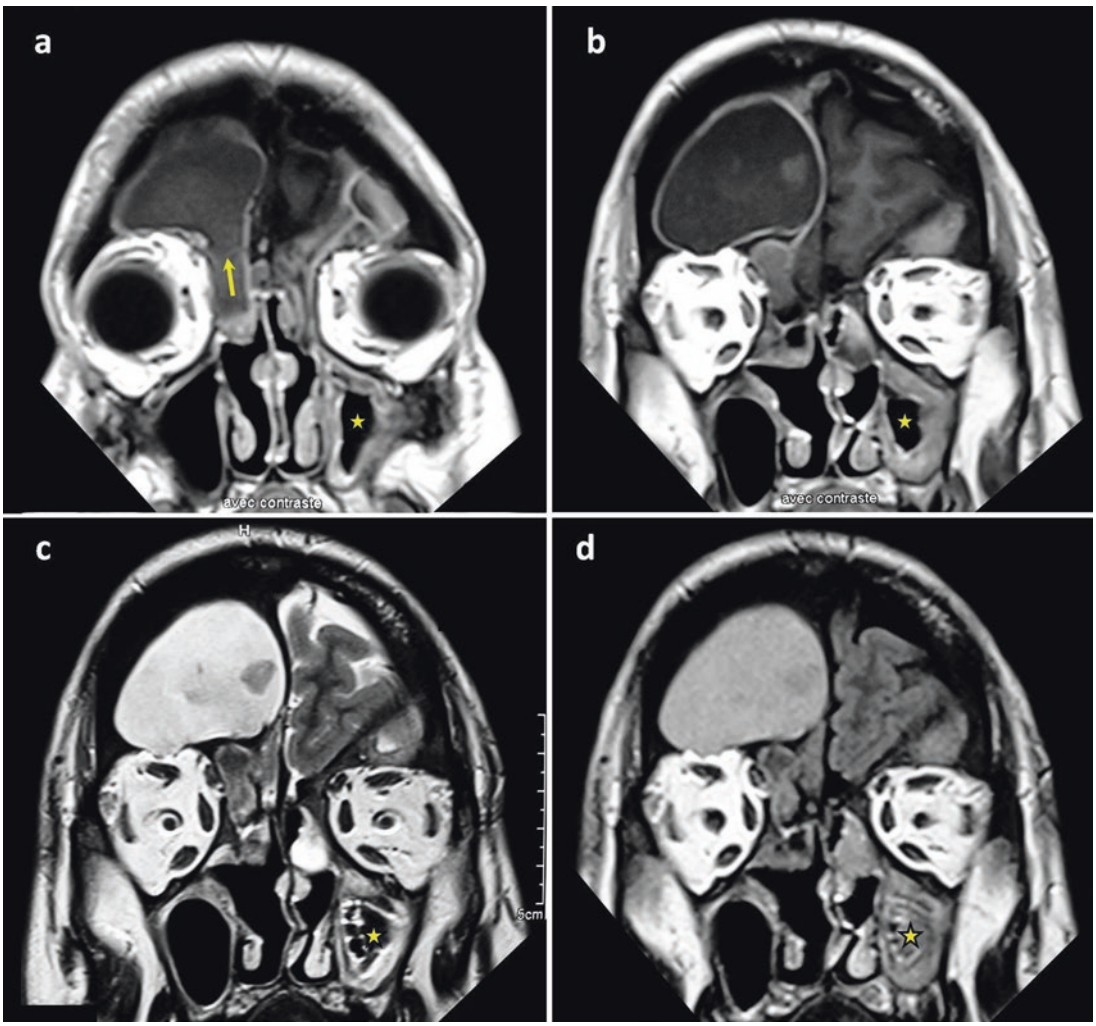


**Fig. 13.15** Case 13.4. Coronal post-gadolinium T1-weighted (**a**, **b**) and T2-weighted (**c**) images and FLAIR sequence (**d**). This frontal pyomucocele extends toward the ipsilateral ethmoidal cells (*arrow*). Note the chronic contralateral maxillary sinusitis (*star*). The patient was operated on via a transnasal endoscopic approach

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# Pott's Puffy Tumors

# 14

Pott's puffy tumor is an uncommon form of cranial osteomyelitis, mostly a complication of frontal sinusitis in an adolescent male presenting with a forehead subperiosteal abscess, frontal osteomyelitis, and possible intracranial and/or orbital extensions. The subperiosteal abscess is usually misdiagnosed as a primary scalp abscess. Diagnosis historically has been made on a clinical basis, but modern neuroimaging tools demonstrate more anatomical details, especially when intracranial or orbital complications occur. A multidisciplinary approach is needed to treat frontal sinusitis, cranial bone infection, and its intracranial complications. Antibiotic therapy should include broad-spectrum antibiotic drugs against organisms of paranasal air sinus origin. The outcome, though generally good, is related to the severity of intracranial infections and to the delay in diagnosis.

## Epidemiology and Etiology

Pott's puffy tumor, an unusual form of cranial osteomyelitis, is characterized by the association of localized forehead swelling with overlying extracranial subperiosteal abscess and underlying frontal bone infection. Erosion of the inner table of the skull results in cranial epidural abscess (Fig. 14.1). More seriously, infections may progress deeply into the subdural space (subdural empyema), the subarachnoid space (meningitis), the brain parenchyma (abscess/encephalitis), or the dural sinus (dural thrombophlebitis). Orbital involvement is not a rare complication.

Less frequently, sinogenic intracranial involvement is possible without direct erosion of the cranial bone. In this perspective, infection spreads via septic thrombophlebitis of intracranial or extracranial venous channels.

Frontal sinusitis is the main cause of Pott's puffy tumor, but it may be associated with additional acute or chronic paranasal sinusitis. Other factors are dental infection, insect bite, acupuncture, cosmetic surgery, cocaine abuse, intranasal methamphetamine use, and previous craniotomy.

Pott's puffy tumor is most common in adolescent males and occurs less often in adults and newborns.

## Clinical Presentations

The classic clinical picture is a tender forehead swelling mimicking a "unicorn appearance" with headache, fever, and rhinorrhea. Neurologic presentations should alert for intracranial complications. Fronto-cutaneous fistula can occur with purulent discharge. Orbital involvement may present with eyelid and/or periorbital edema and proptosis.

Failure or delay of symptom resolution with classic antibiotics may mask the intracranial extent of infections and neurologic symptoms and signs.

## Imaging Features

Cranial bone erosions are noticed on CT scans. Further paranasal sinusitis and orbital involvement may be associated (Figs. 14.2, 14.3, and 14.4).

MRI is useful in demonstrating intracranial and intraorbital complications, bone marrow edema, leptomeningeal enhancement, extradural and/or subdural collections, brain abscess, and sinus venous thrombosis (Fig. 14.5).

Bone scan is very sensitive for detecting cranial bone infection. Ultrasonographic imaging may help in detecting subperiosteal abscess associated with erosion of the frontal bone.

## Laboratory Findings

The usual inflammatory parameters may be useful for the diagnosis, but they are nonspecific and highly variable in their expression.

Aerobic and anaerobic bacteria can be responsible for the infection, often as a polymicrobial suppurative infection.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_14

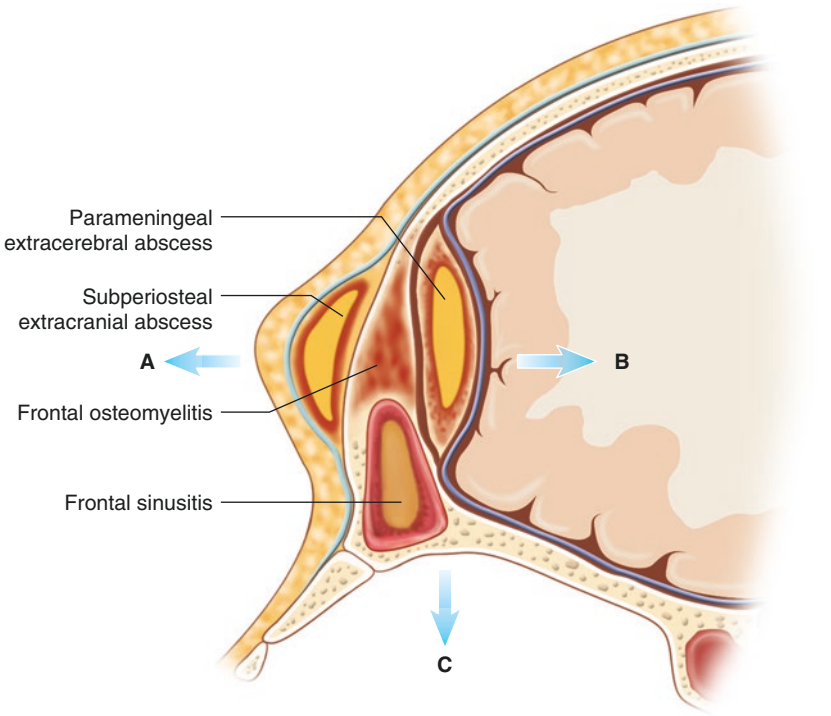


**Fig. 14.1** Main topographic complications of Pott's puffy tumor. (**a**) Extracranial extension, (**b**) intracranial extension, and (**c**) orbital extension

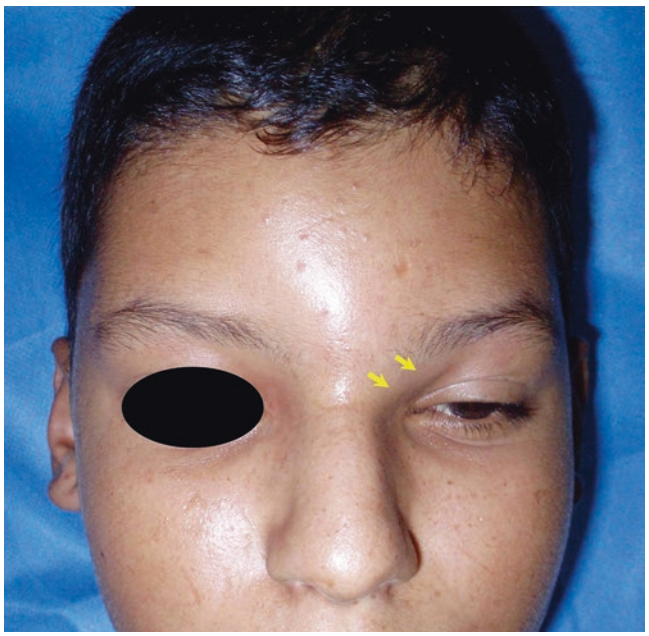

**Fig. 14.2** Case 14.1. Frontal view of a patient with forehead swelling and

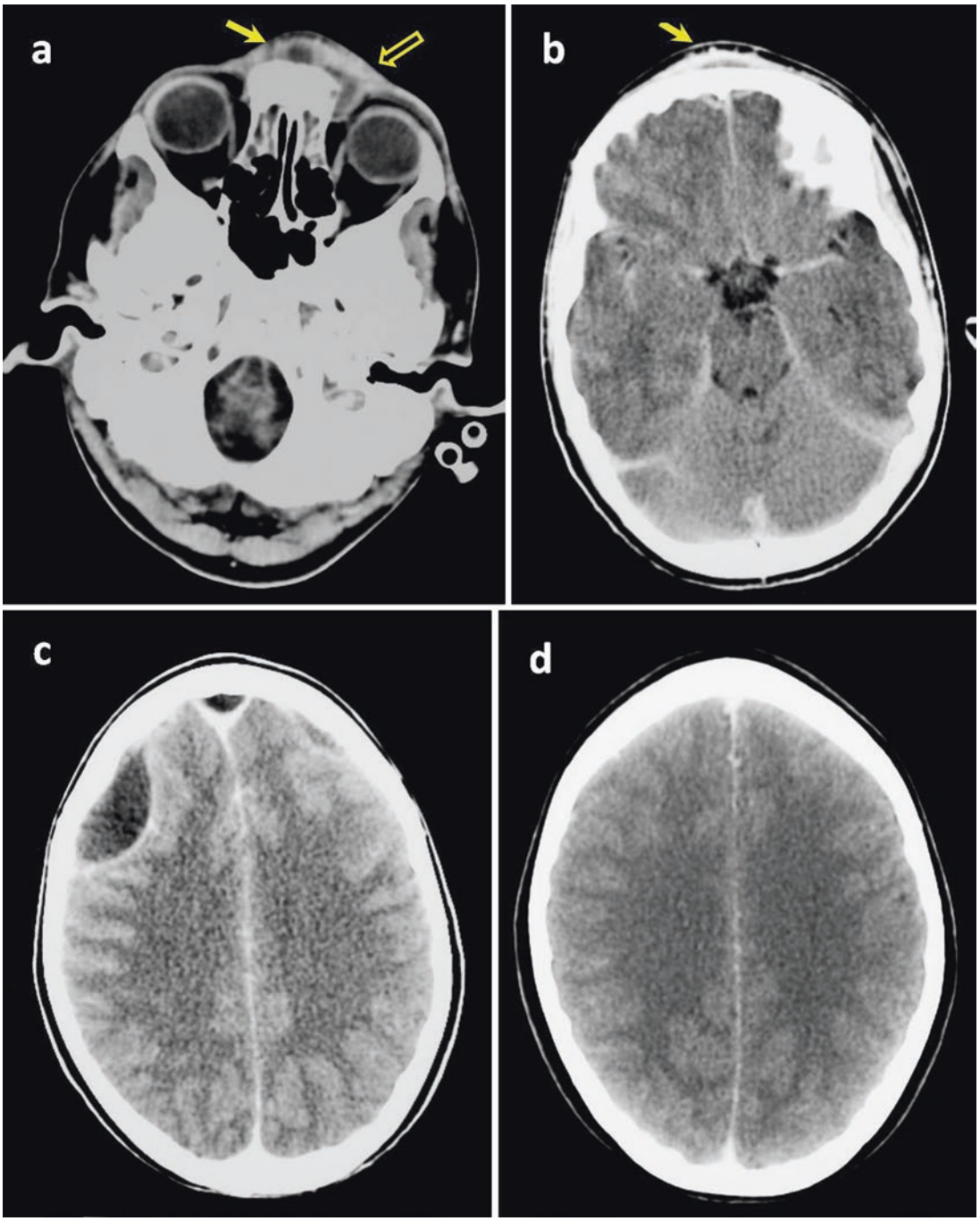


**Fig. 14.3** Case 14.1. Axial cranio-orbital post-contrast CT scans (**a–c**) showing the frontal subgaleal abscess (*solid arrow*) and its intraorbital extension (*hollow arrow*). Note the bi-frontal intracranial epidural abscesses (**c**). The right frontal extradural empyema was drained with an adapted antibiotic therapy. Control CT scan (**d**) 4 weeks later shows complete disappearance of the empyemas

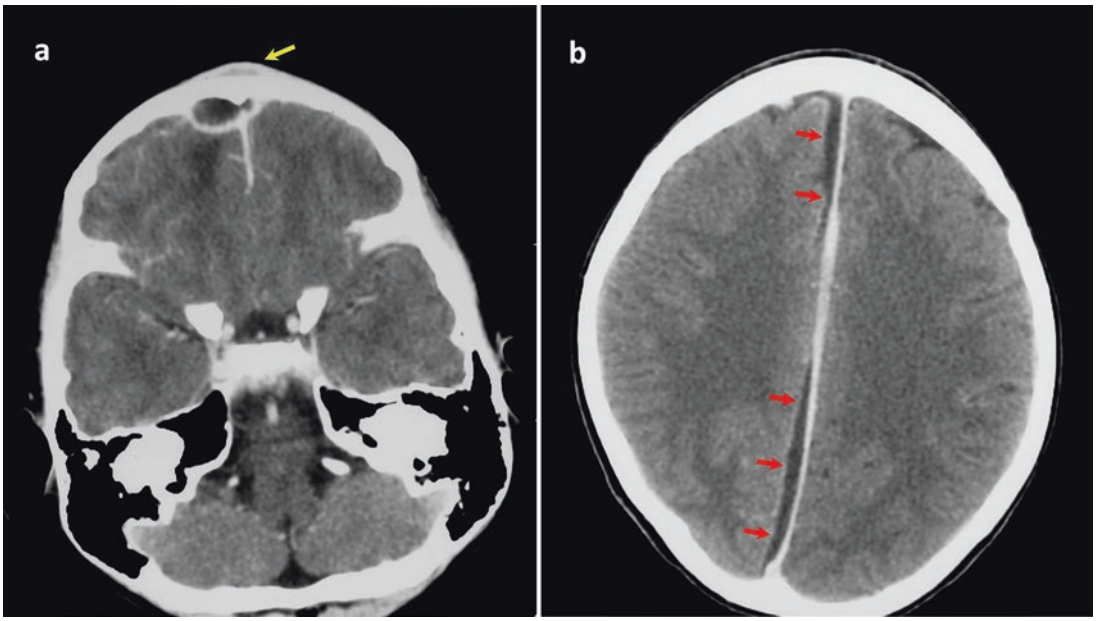


**Fig. 14.4** Axial cranial CT scan with contrast injection revealing an extracranial frontal subperiosteal abscess (*arrow*) (**a**) with associated intracranial subdural parafalcine empyema (*arrows*) (**b**)

The most common individual pathogens are streptococci, staphylococci, *Bacteroides* species, and *Fusobacterium* species. These microorganisms reflect the bacterial species encountered in air sinus infection.

Sterile cultures are common, probably because most patients receive antibiotic drugs before surgery.

## Treatment Options

Medical management typically accompanies surgical treatment. Antibiotic therapy should include broad-spectrum antibiotic drugs against aerobic and anaerobic cocci and bacilli, with adequate CNS and abscess penetration. Because of cranial bone infection, antibiotics should be given for at least 8 weeks.

Patients with a small subperiosteal abscess and/or limited epidural abscess without neurologic complications can be treated with antibiotic drugs only.

A multidisciplinary approach (neurosurgery and otorhinolaryngology) is needed to treat paranasal sinusitis, osteomyelitis, and its intracranial and encephalic complications.

Craniotomy for drainage and debridement can be done with an open or minimally invasive approach. Infected bone with evidence of necrosis should be removed, followed by cranioplasty on a second intervention. A titanium mesh may be used at the time of operation. When accessible, the posterior wall of the frontal sinus should be explored during surgery and its cranialization done if possible. The frontal sinus may be managed with trephination, obliteration, and/or exenteration. An endoscopic approach may be effective.

If available, adjuvant hyperbaric oxygen therapy should be considered for the treatment of refractory forms.

## Outcomes

Close clinical and biological monitoring and neuroimaging are important to ensure adequate response to treatment.

Complete clinical recovery is predictable for most patients. The outcome is related to the severity of infection (especially intracranial complications) and to the delay in diagnosis. Long-term sequelae are rare but may include aphasia, seizure, chronic osteomyelitis, and facial scar.

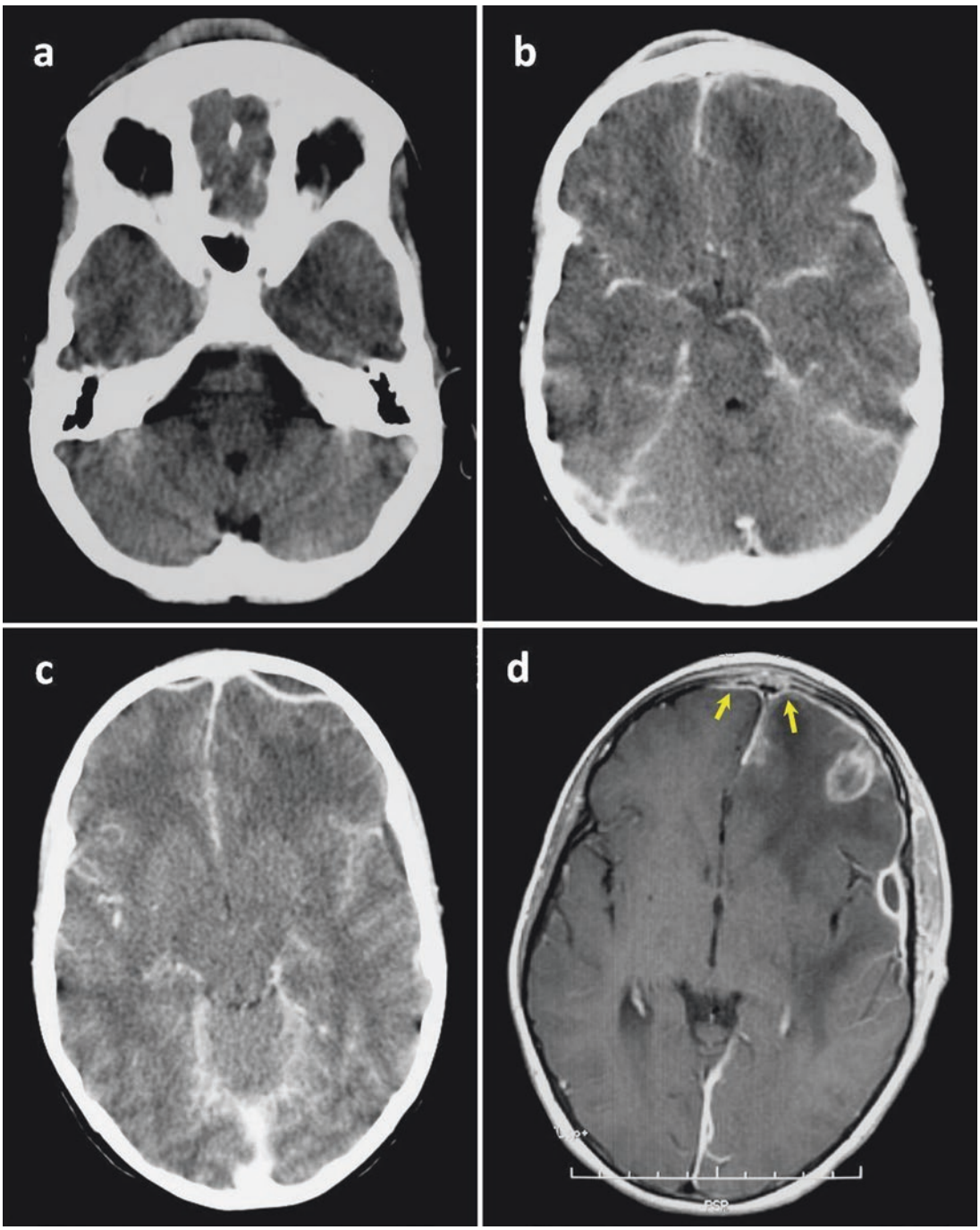


**Fig. 14.5** Axial cranial CT scan after contrast administration (**a–c**) and T1-weighted MRI with gadolinium injection (**d**) showing a Pott's puffy tumor with extracranial and intracranial suppurative collections. Note the frontal bone infection (osteomyelitis) (*arrows*)

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# Intracranial Infectious Aneurysms

# 15

Intracranial infectious aneurysms (IIAs), or mycotic aneurysms or microbial aneurysms, are rare cerebrovascular lesions that occur through microbial infection of the cerebral arterial wall. Although some cases remain asymptomatic, most symptomatic patients present with symptoms related to aneurysm rupture and to its causative origin. IIAs are typically thin-walled and friable, often with a wide or absent neck and a high tendency to rupture and bleed. Echocardiography is mandatory to look for signs of endocarditis, the most common source of the infection. Treatment is based on administration of antibiotics in combination with surgical or endovascular procedures according to the character and position of the aneurysm and the clinical condition of the patient. IIAs constitute a distinct group of potentially fatal cerebrovascular lesions. Patients with immunosuppression, those with fungal aneurysm, and those with multiple IIAs have a high mortality rate.

## Epidemiology and Etiology

Intracranial infectious aneurysms (IIAs) (also called mycotic aneurysms or microbial aneurysms) are rare cerebrovascular lesions that occur through microbial infection of the cerebral arterial wall. The expression "mycotic aneurysm" is inappropriate because it usually refers more loosely to all infectious aneurysms. The term "mycotic" intracranial aneurysm should be used to indicate only those of truly fungal etiology. Most IIAs are bacterial; fungal origin is quite rare.

Less than 5% of all intracranial aneurysms are infectious in origin, but the frequency of IIAs is increased in immunocompromised patients.

Classically, there are three primary forms of IIA: Intravascular, extravascular, and cryptogenic (Table 15.1). There is no clear sex predominance; most patients are young adults (median age, 35 years). Table 15.2 outlines some criteria for the diagnosis of clinically definite, probable, and possible IIA.

## Clinical Presentations

Cases may remain asymptomatic, but most symptomatic patients present with symptoms related to aneurysm rupture and its causative origin. The most common clinical findings are fever, headache, loss of consciousness, motor deficit, cranial nerve palsy, seizure, and meningitis. Symptoms and signs of infective endocarditis are present in more than three quarters of all patients. The mitral valve is most often involved, followed by the aortic valve.

Fungal IIA is frequently associated with intravenous drug abuse and immunocompromised conditions. These patients are normally not febrile and have a more indolent clinical presentation.

Patients with IIA occurring from an extravascular origin tend to have symptoms and signs of more local infection with unruptured aneurysms.

Screening for IIAs should be performed in patients with predisposing infectious conditions (especially potential causative etiologies).

## Imaging Features

Both CT angiography and digital subtraction angiography (DSA) are useful for screening purposes. Parenchymal changes associated with IIA are better assessed on MRI. IIAs are typically thin-walled and friable, often with a wide or absent neck and a high tendency to rupture and bleed. Intraparenchymal hemorrhage (including hemorrhagic transformation of ischemic stroke) is more common than subarachnoid hemorrhage.

Most IIAs are located in the middle, posterior, or anterior cerebral arteries. Distal arterial sites are frequent. Up to 25% of patients have multiple aneurysms. Both saccular and fusiform aneurysms are seen, without predominance.

On follow-up neuroimaging of an IIA, it is common to see a change in size or the appearance of a new aneurysm.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_15

**Table 15.1** Primary forms of infectious intracranial aneurysms

| Form | Characteristics |
|---|---|
| **Intravascular** | Endoluminal septic emboli in distal vasculature; may be multiples; mainly due to subacute bacterial endocarditis |
| **Extravascular** | Extraluminal-associated infection like meningitis, orbital cellulitis, ENT infections, cavernous sinus thrombophlebitis, or skull base osteomyelitis. Starting in the adventitia, spreading inward to the media, and finally to the intima |
| **Cryptogenic** | Presumptive diagnosis based on clinical/radiological or histological findings without obvious inflammatory process |

**Table 15.2** Kannoth's proposed criteria for diagnosis of infectious intracranial aneurysm

| Diagnostic standard | Criteria |
|---|---|
| Mandatory criterion | Aneurysm demonstrated by imaging |
| Supporting criteria | *Predisposing infection:* |
| | Infective endocarditis |
| | Meningitis |
| | Cavernous sinus thrombophlebitis |
| | Orbital cellulitis |
| | *Angiographic features:* |
| | Multiplicity |
| | Distal location |
| | Fusiform shape |
| | Change in size of aneurysm or appearance of new aneurysm on follow-up angiogram |
| | *Other features:* |
| | Younger age (<45 years) |
| | History of recent lumbar puncture |
| | Fever at presentation |
| | Intraparenchymal hemorrhage on CT/MRI |
| ***Clinically definite IIA*** | Mandatory criterion and any three of the supportive criteria are met |
| ***Clinically probable IIA*** | Mandatory criterion and any two of the supportive criteria are met |
| ***Clinically possible IIA*** | Mandatory criterion and any one of the supporting criteria are met |

IIA—infectious intracranial aneurysm
Adapted from Kannoth et al. (2009); with permission

Echocardiography is mandatory to look for signs of endocarditis. In addition, modern imaging may be useful in diagnosing the primary sources of craniofacial infections.

## Laboratory Findings

Blood cultures and cerebrospinal fluid studies may be positive in less than half of all patients.

Blood tests can demonstrate leukocytosis or an elevated erythrocyte sedimentation rate and C-reactive protein level. These biologic inflammatory parameters should be evaluated in conjunction with clinical and neuroimaging studies.

A variety of bacteria, fungi, mycobacteria, parasites, and viruses could cause IIA. The most common causative pathogens are *Streptococcus* species (especially *S. viridans*), followed by *Staphylococcus* species. *Enterococcus* species, gram-negative bacteria, and *Mycobacterium tuberculosis* are less frequent. True fungal (mycotic) microorganisms are rare (less than 5%), most often *Aspergillus fumigatus*, *Candida albicans*, or *Mucor* species. (Please refer to Chap. 29.)

Histopathology may demonstrate infiltration of the media and adventitia of the vessel wall by polymorphonuclear leucocytes. There is also important proliferation of intima and destruction of internal elastic lamina. Pathogenic agents may be identified within the vessel wall by appropriate coloration.

## Treatment Options

Treatment is based on administration of antibiotics in combination with surgical or endovascular procedures, depending on the character and position of the IIA and the clinical condition of the patient (Figs. 15.1, 15.2, 15.3, 15.4, and 15.5).

Appropriate antibiotic drugs, covering both *Streptococcus* and *Staphylococcus* species, should be started as soon as possible and continued for at least 6 weeks. Total aneurysm resolution with antibiotic therapy is possible in about 30% of cases. The unruptured IIA can be managed conservatively under an antibiotic regimen unless the aneurysm enlarges or fails to reduce in size after 6 weeks of treatment. Tubercular and fungal lesions must be treated with the appropriate anti-infectious regimens.

Initial surgical indications include intracranial hypertension (especially hematoma or acute hydrocephalus) and herniation syndromes. Delayed clipping may be done if the IIA enlarges or persists despite antibiotic therapy. With a friable aneurysm, wrapping techniques or trapping the artery parent may be an alternative.

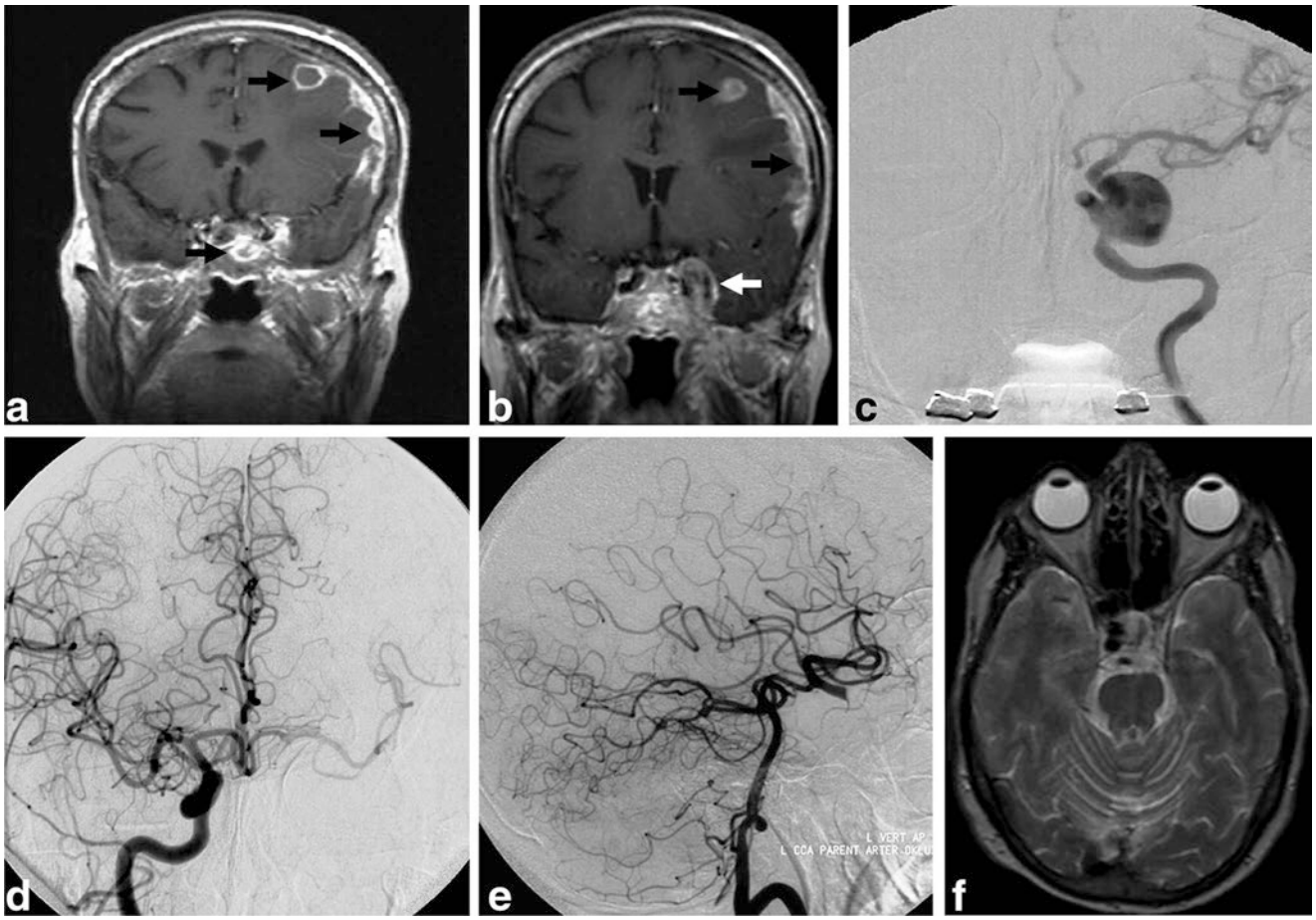


**Fig.15.1** A 62-year-old female patient with known diagnosis of mucormycosis, presenting with new-onset ptosis. (**a**, **b**) T1-weighted, contrast-enhanced MR images show widespread cortical abscess formation and sphenoid sinus lesion (*black arrows*) and signal void huge cavernous carotid artery aneurysm (*white arrow*), which was absent on MRI at the initial workup. (**c**) Left internal carotid artery (ICA) angiogram (anteroposterior view) confirms a huge cavernous carotid artery aneurysm. (**d**, **e**) Right ICA and left vertebral angiograms show occlusion of both the aneurysm and parent artery after the use of detachable balloons. Note the filling of the left middle cerebral artery via posterior and anterior communicating arteries. (**f**) Late control MRI shows absence of both aneurysm and carotid artery (Reproduced from Esenkaya et al. (2016), with permission)

Endovascular procedures have increased over the past three decades. Distal or fusiform aneurysms are treated with parent vessel occlusion (using cyanoacrylate, autologous clot, or polyvinyl alcohol microparticles). Proximal saccular aneurysms are treated by latex balloons and coils.

Patients with subacute endocarditis require heart valve replacement.

## Outcomes

IIAs constitute a distinct group of potentially fatal cerebrovascular lesions. Close clinical and biological monitoring with serial cerebral angiography is important to ensure adequate response to treatment.

The mortality rate is high in patients with immunosuppression, fungal aneurysms, or multiple IIAs.

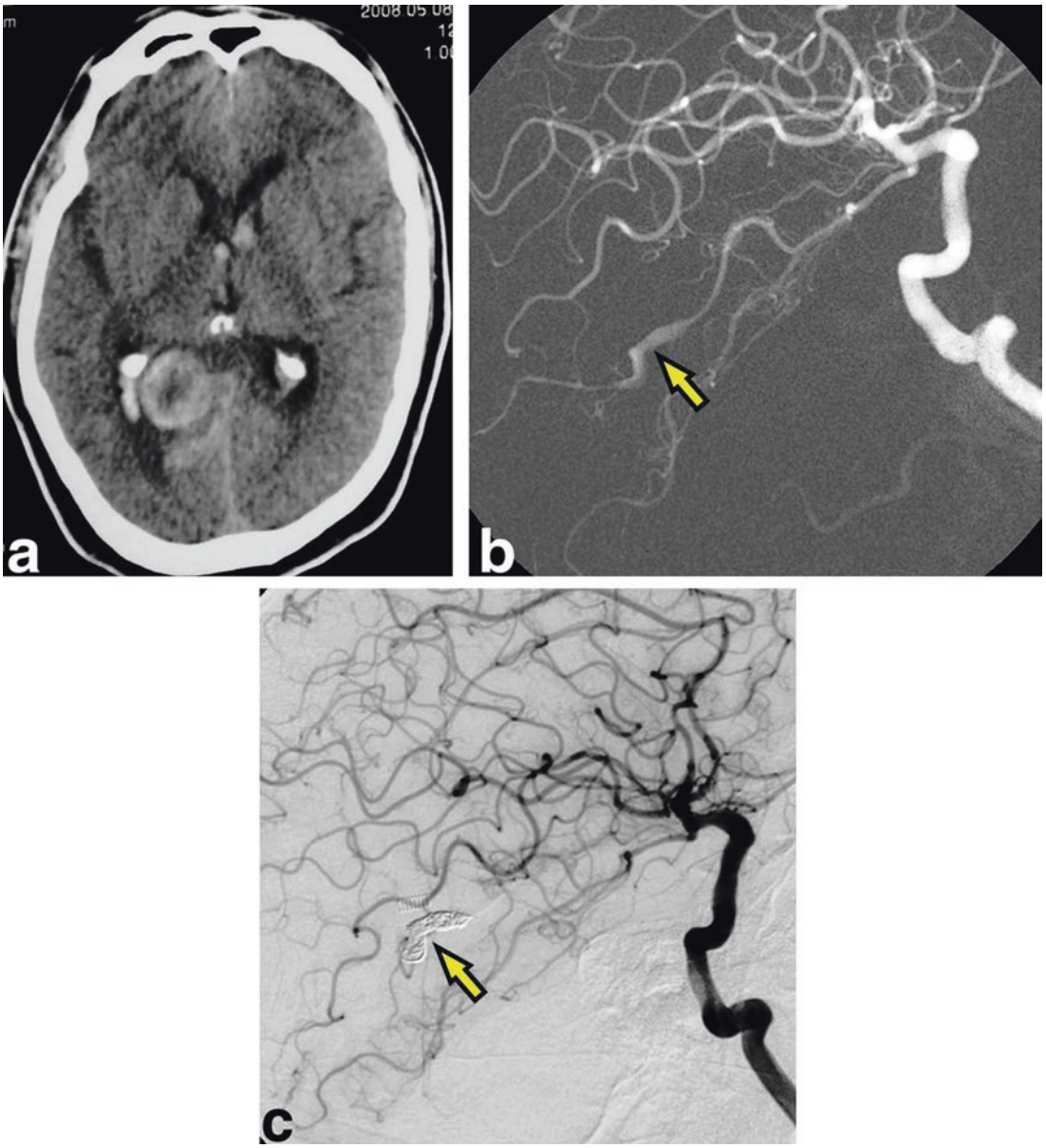


**Fig. 15.2** A 63-year-old man presented with intraparenchymal hematoma. (**a**) CT scan shows right occipitoparietal hematoma with intraventricular extension. (**b**) Right ICA lateral angiogram reveals a fusiform aneurysm at the P3 segment of the left posterior cerebral artery (*arrow*). (**c**) Final angiogram shows aneurysm and parent vessel occlusion after embolization with coils (*arrow*) (Reproduced from Esenkaya et al. (2016), with permission)

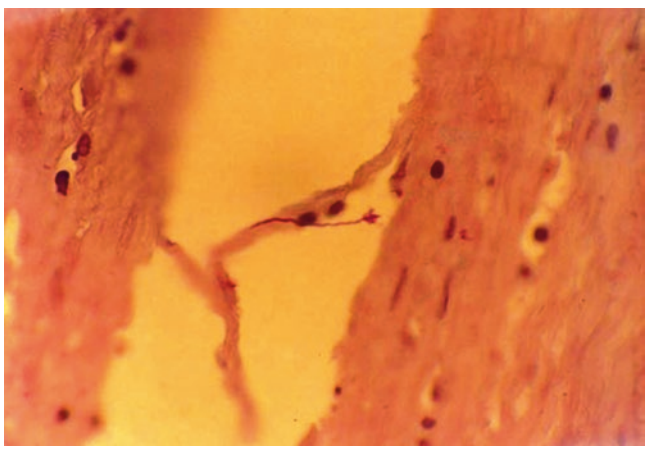

Fig. 15.3 Postmortem histological examination of the basilar artery (hematoxylin–eosin, ×1100). Detail of partially dissociated artery wall showing *Candida* hyphae (Reproduced from Marazzi et al. (2008), with permission)

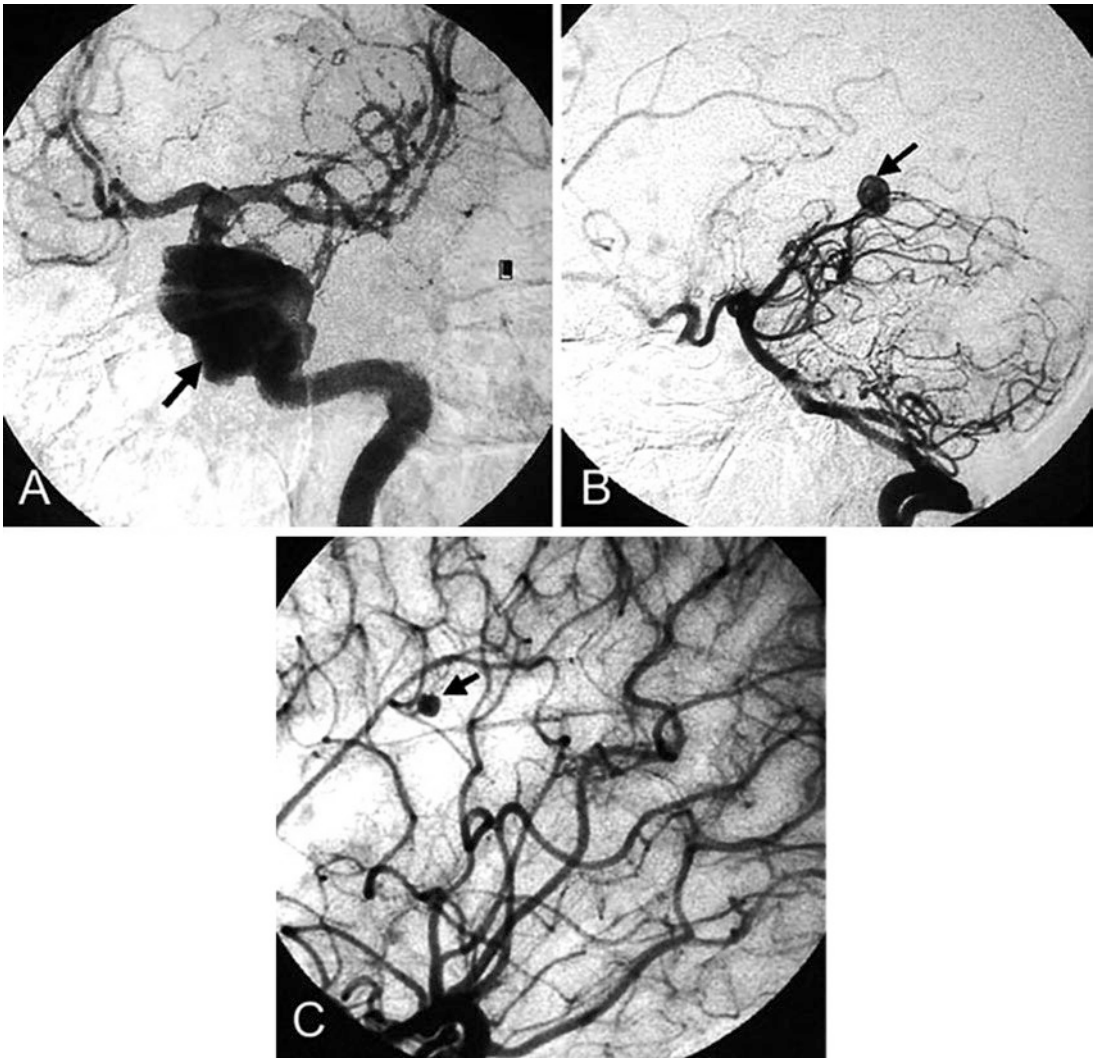


**Fig. 15.4** Angiographic appearance of microbial aneurysm (MA). (**a**) Digital subtraction angiography (DSA) of the carotid artery, showing a giant MA of the cavernous portion of the ICA (*arrow*). (**b**) DSA showing a distal MA of the posterior cerebral artery (*arrow*). (**c**) MA on a branch of the middle cerebral artery (*arrow*) (Reproduced from Kannoth et al. (2009), with permission)

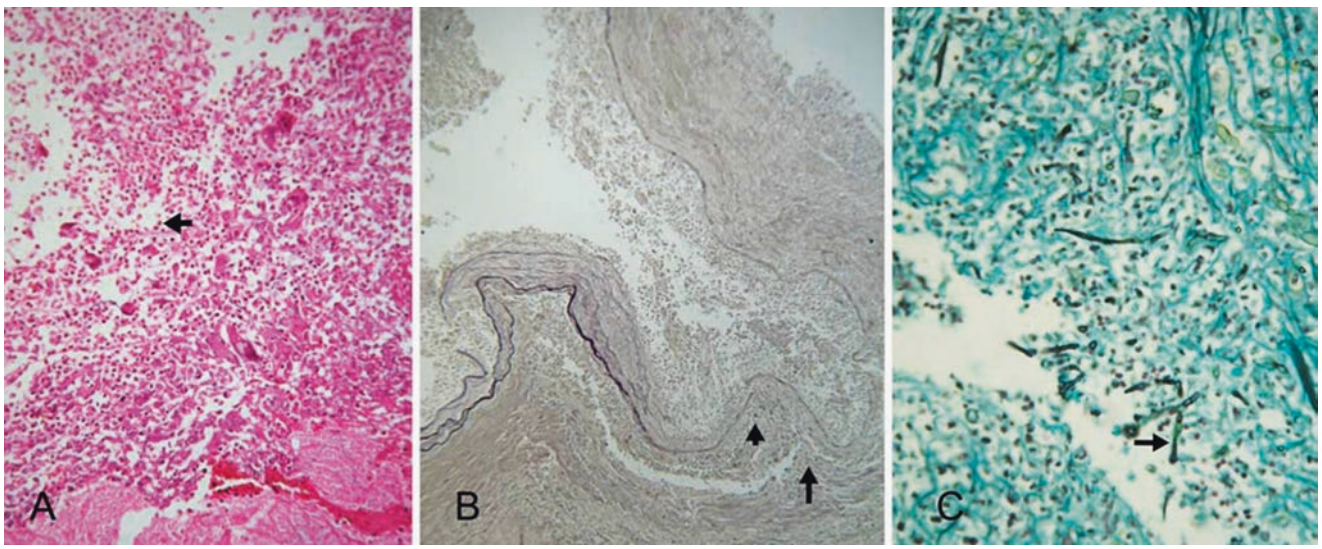


**Fig. 15.5** Histological findings in microbial aneurysm. (**a**) Photomicrograph of a section through the wall of an MA of the basilar artery, showing inflammatory cell infiltration of the wall of the MA resulting in destructive changes in the intima and media (hematoxylin–eosin, ×200). (**b**) Photomicrograph showing destruction in the continuity of internal elastic lamina resulting in aneurysm formation (elastic Van Gieson (EVG) stain, ×200). (**c**) Photomicrograph showing presence of septate and branched hyphae of *Aspergillus fumigatus* seen embedded in inflammatory exudates (Grocott–Gomori stain, ×200) (Reproduced from Kannoth et al. (2009), with permission)

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# Part III

# Infections of the Spine and Its Coverings

# Paraspinal Pyomyositis

# 16

Pyomyositis (tropical myositis) is a subacute, purulent infection of the large skeletal muscles around the pelvis and lower extremities. Paraspinal muscles are rarely involved. *Staphylococcus aureus* is the most common causative pathogen. Clinical presentations are usually nonspecific and include local and systemic signs and symptoms of infection, with limited range of spinal motion. Back pain is considered unusual. Both MRI and CT scanning can accurately identify intramuscular soft tissue abscesses. Concomitant spinal and/or intraspinal extension may be seen. Ultrasound exploration is useful for repeated monitoring of the infection site. Laboratory tests reveal nonspecific inflammation. Histological examination discloses foci of abscess surrounded by marked inflammatory granulation tissue and degenerating muscle fibers. Treatment requires appropriate antibiotics when the infection is in the pre-suppurative form and prompt surgical drainage when a suppurative collection arises. If treated adequately, most patients heal without recurrences or additional complications. Delay in diagnosis may result in osteomyelitis of adjacent spinal and pelvic bones, spinal epidural abscess, retroperitoneal abscess, metastatic infection, sepsis, and occasionally death.

## Epidemiology and Etiology

Soft tissue infections adjacent to the spine include a wide variety of presentations that are often associated with vertebral and/or discal infections. Isolated paraspinal suppurations are unusual; they may involve the skin or the paraspinal muscular groups (superficial or deep) (Fig. 16.1). The most severe form, primary paraspinal pyomyositis, is rarely seen in neurosurgical and spinal practices.

Pyomyositis is a subacute, purulent infection of the large skeletal muscles that usually manifests as single or multiple abscesses. Generally, no local or adjacent source of infection is apparent. In fact, the pathogenesis is unclear, but trauma, viral infection, and malnutrition have been implicated. It is classically a tropical disease, but it has been recognized worldwide owing to increasing numbers of cases of diabetes mellitus, HIV infection, drug abuse, and malignancy. Pyomyositis has three consecutive stages: pre-suppurative diffuse muscle infection, abscess formation, and myonecrosis with sepsis. The disease usually affects children and young adult men; it is rarely seen in older people. Common sites of infection are muscle groups around the pelvis and the lower and upper extremities. Paraspinal muscles (cervical, thoracic, lumbar, or psoas) are involved in less than 4% of cases.

Diagnosis is frequently delayed because of inexperience with the disease, the lack of specific symptoms and signs, atypical appearances, and a wide range of differential diagnosis.

## Clinical Presentations

Symptoms and signs are usually nonspecific and include fever, vague local tenderness, pain and swelling around the muscular infection site, flank pain, abdominal pain, and limited range of spinal motion, with or without scoliotic attitude. The overlying skin may be swollen, erythematous, and warm (Fig. 16.2). Back pain is considered unusual. If an iliopsoas muscle abscess is present, the patient may have a positive psoas sign (hip flexor) and pain radiating to the hip or thigh area.

Obvious systemic source of sepsis should always be considered, with screening for an infectious focus of secondary pyomyositis.

The condition is often initially misdiagnosed as muscle strain, acute myositis, contusion, hematoma, thrombophlebitis, perinephric abscess, acute appendicitis, osteomyelitis, septic arthritis, soft tissue sarcoma, cellulitis, or necrotizing fasciitis.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_16

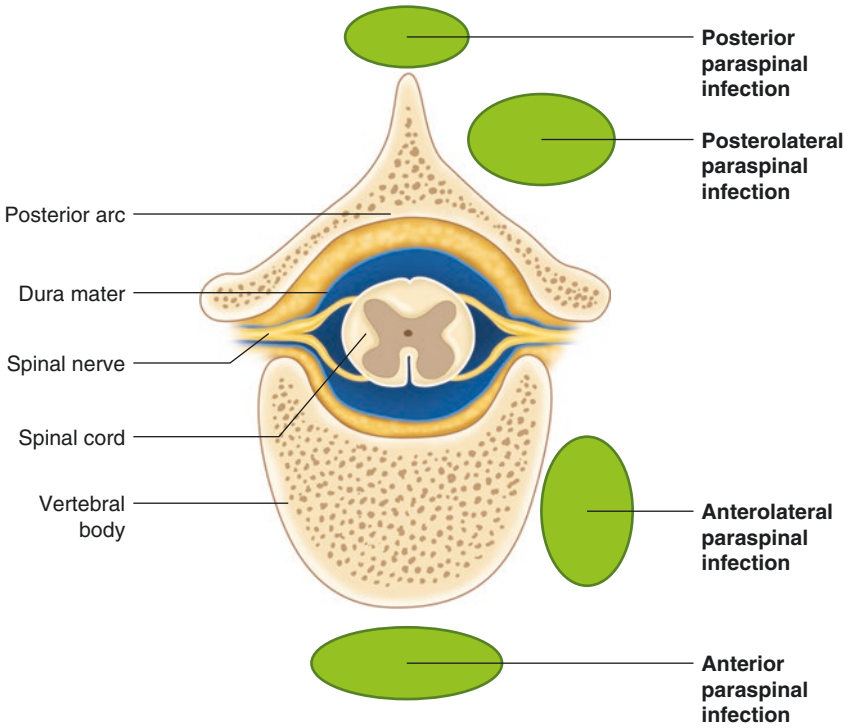


**Fig. 16.1** Topographic classification of paraspinal infections

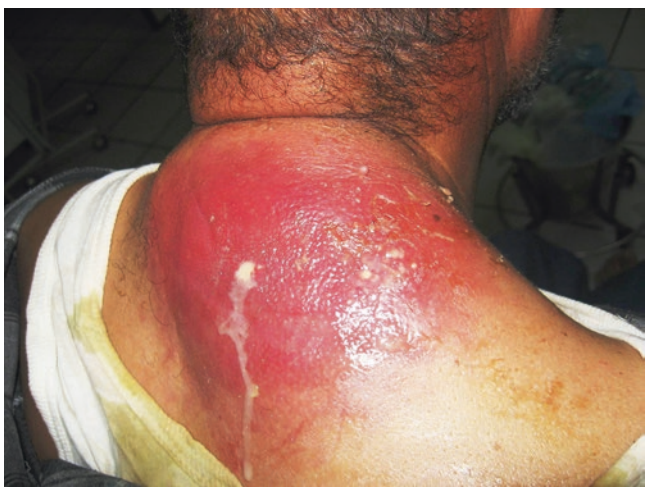

**Fig. 16.2** Superficial soft tissue infection of the posterior cervicothoracic area, associated with contiguous infection of the skin in a diabetic patient (fulminant cellulitis with suppurative fistula)

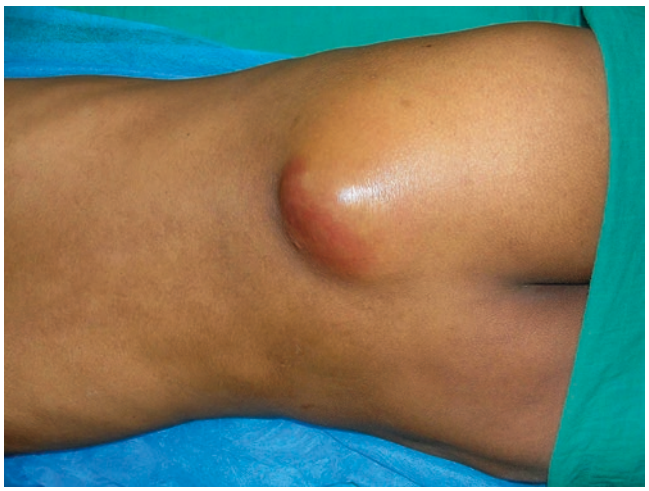

**Fig. 16.3** Case 16.1. Prominent swelling on the right side, which extends from the lumbosacral spine down to the buttock

## Imaging Features

Plain radiography has limited utility, although it can rule out a primary spinal bone lesion.

Ultrasound exploration demonstrates a bulky muscle with irregular echo-texture and a hypoechoic focal lesion. Occasionally, internal debris and air bubbles are visible. Ultrasonography is also useful for repeated monitoring of the infection site.

CT scanning provides better delineation of muscular structure and valuable data on the nature and extent of the disease. A focal area of low attenuation or gas formation may be seen, with rim enhancement after contrast medium injection. Intramuscular accumulation of fluid can be seen in the abscess stage (Figs. 16.3, 16.4, 16.5, 16.6, 16.7, 16.8, and 16.9). CT scans are also useful to exclude any bony involvement, especially spinal or pelvic osteomyelitis.

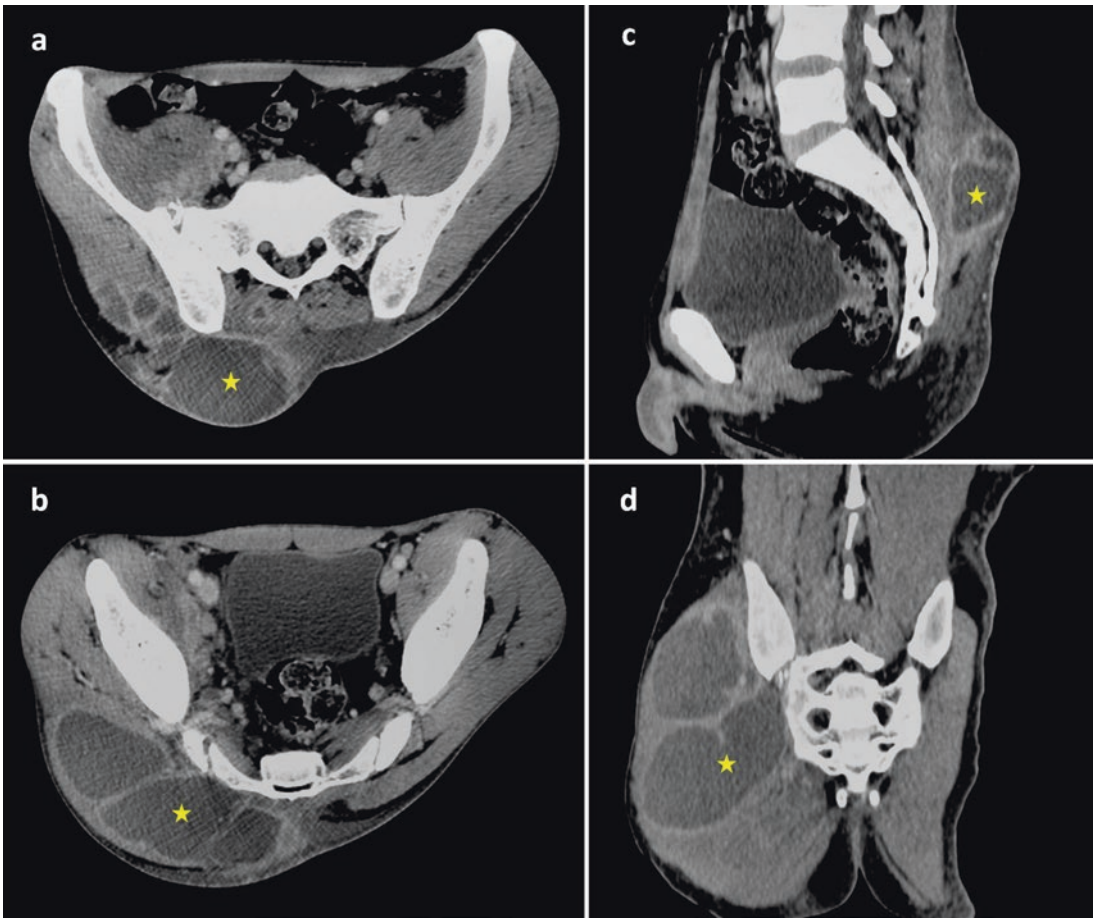


**Fig. 16.4** Case 16.1. Axial (**a**, **b**), sagittal (**c**), and coronal (**d**) CT scans with contrast administration, showing a multiloculated fluid collection (*star*) in the right gluteal musculature due to pyomyositis in a 23-year-old patient

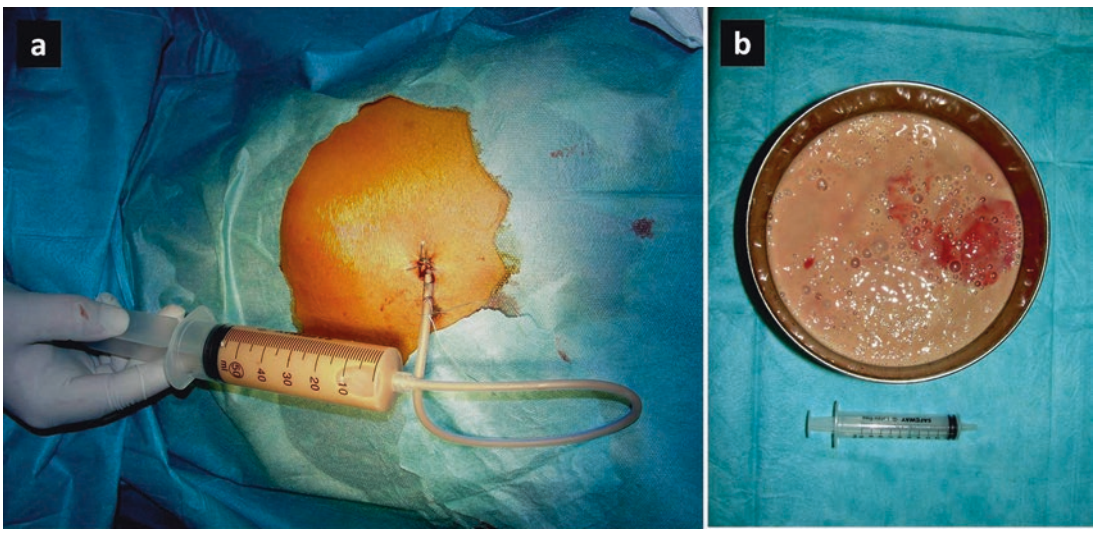


**Fig. 16.5** Case 16.1. Aspiration of the swelling showing frank pus fluid (**a**); 800 cc of purulent fluid was obtained from aspiration (**b**). *Staphylococcus aureus* was identified in the cultured specimens

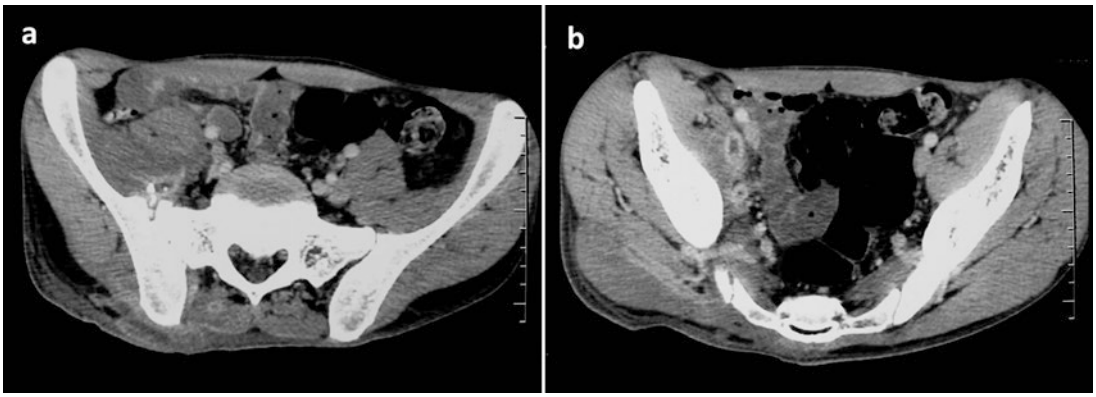


**Fig. 16.6** Case 16.1. Axial post-contrast CT scan obtained 2 weeks later, showing nearly complete resolution of the abscess (**a**, **b**)

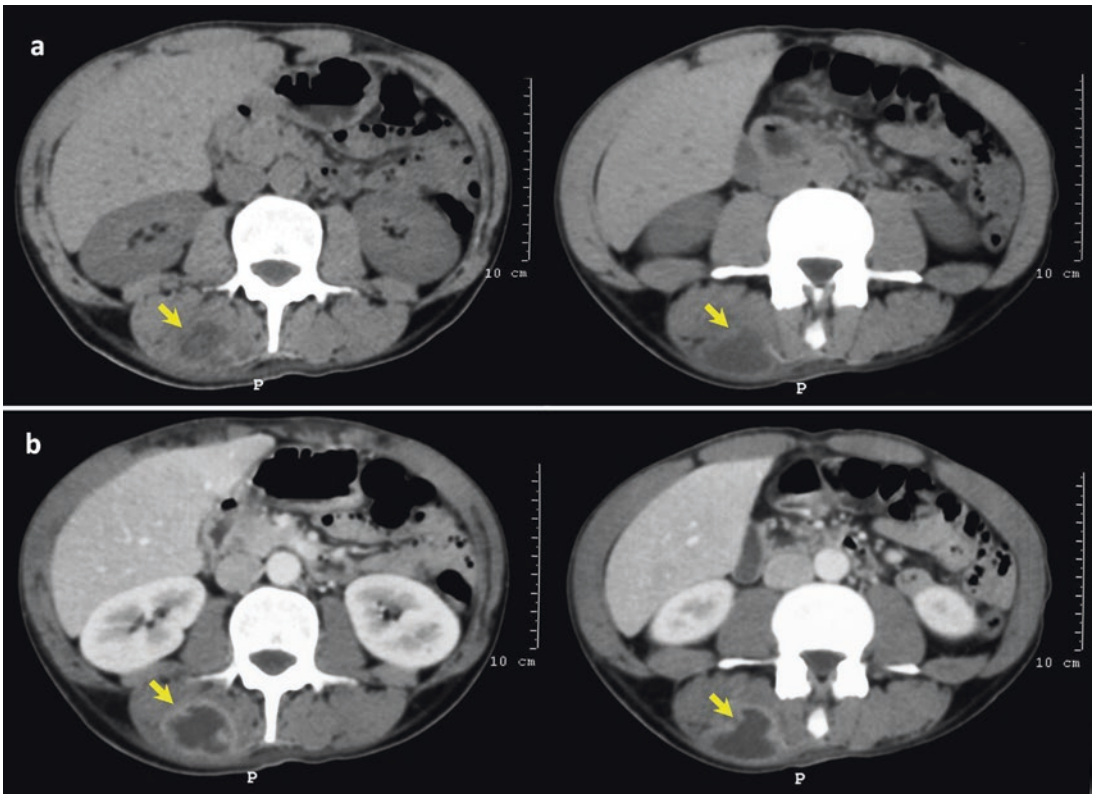


**Fig. 16.7** Abdominal axial CT scan before (**a**) and after (**b**) contrast injection, revealing a spontaneous primary pyogenic erector spinae muscle abscess (*arrow*); the pyomyositis was due to *Staphylococcus epidermidis*

MRI is more sensitive and specific and can detect affected muscles in an early stage. The suppurative collection appears hypointense or isointense on T1-weighted images and hyperintense on T2-weighted images (Figs. 16.8 and 16.10). The degree of rim enhancement depends on the extent of abscess wall and granulation tissue present. MRI is also useful in diagnosing concomitant spinal and/or intraspinal extension.

The use of 18-fluorodeoxyglucose (18-FDG) positron emission tomography–computed tomography (PET-CT) is a promising combined modality for the diagnosis.

## Laboratory Findings

Laboratory tests reveal nonspecific signs of inflammation: marked elevation of the erythrocyte sedimentation rate and C-reactive protein levels, an increased peripheral white blood

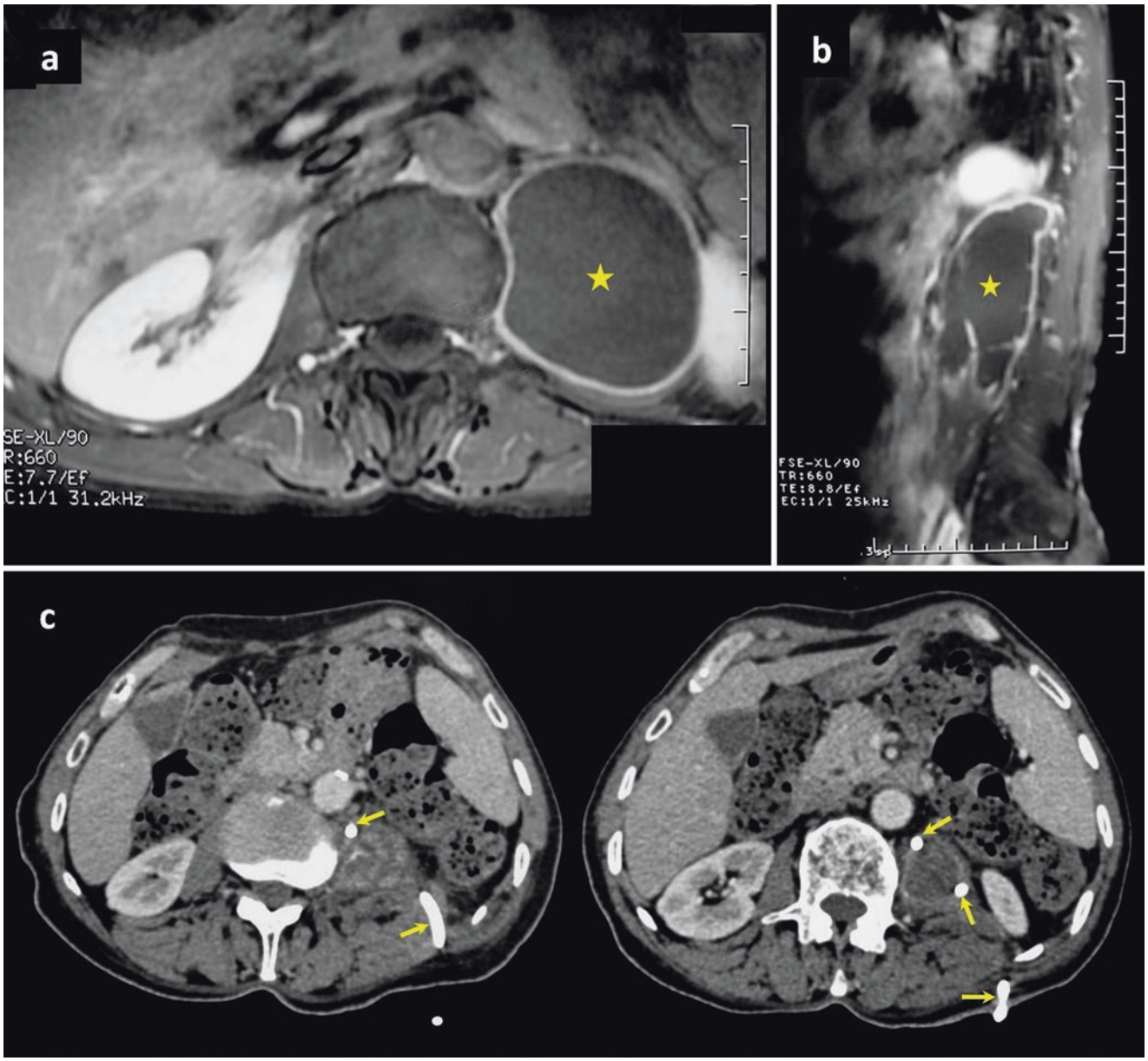


**Fig. 16.8** Axial (**a**) and sagittal (**b**) T1-weighted MR images with gadolinium administration, showing a left isolated iliopsoas pyomyositis (primary abscess) (*star*) without evidence of spinal involvement. Percutaneous abscess drainage was performed, and a drain was left in situ, as seen in the control CT scan (*arrows*) (**c**)

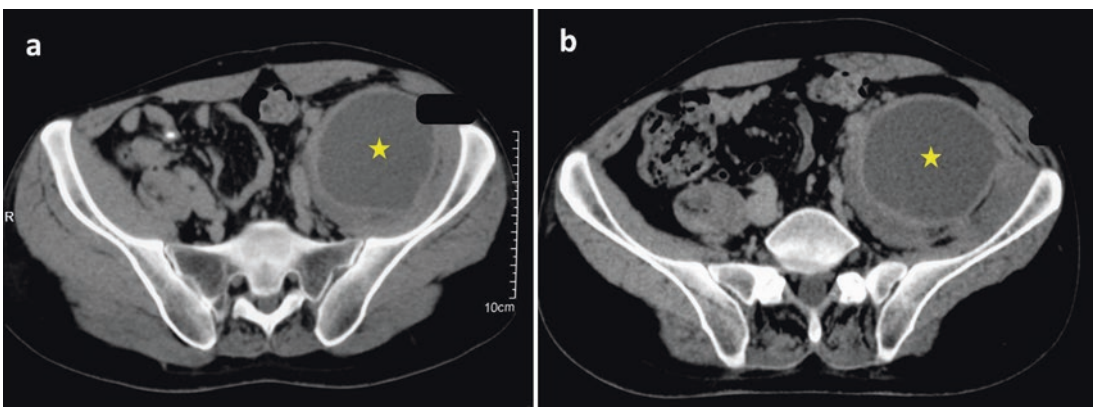


**Fig. 16.9** Axial abdominal CT scan showing an important left iliopsoas abscess (*star*) due to brucellosis (**a**, **b**)

cell count, and anemia. Eosinophilia may be seen in tropical pyomyositis. Elevation of the skeletal muscle enzyme levels is uncommon despite extensive myofibril destruction.

*Staphylococcus aureus* is the most common causative pathogen. Other microorganisms include *Streptococcus* species, *Escherichia coli*, *Salmonella* species, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Haemophilus influenzae*, and *Mycobacterium tuberculosis*. Blood cultures are inconstantly positive.

Histological examination reveals foci of abscess surrounded by marked inflammatory granulation tissue and degenerating muscle fibers.

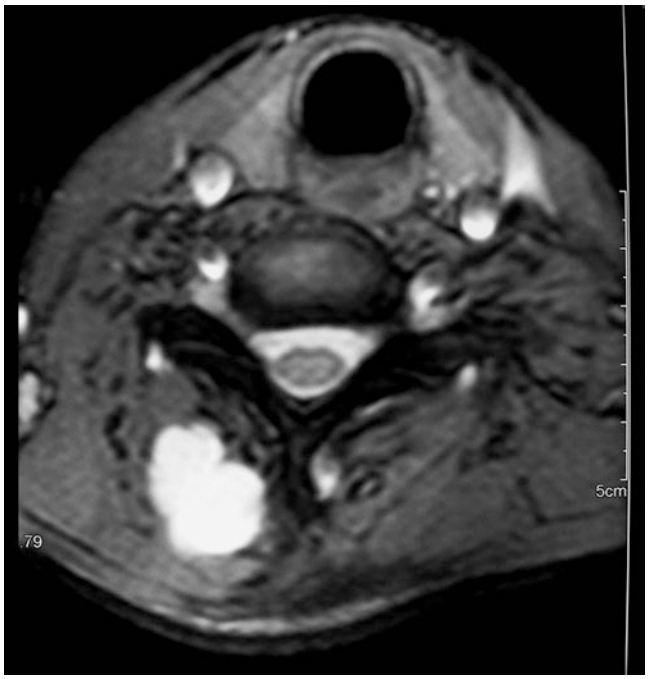


**Fig. 16.10** Axial T2-weighted MRI of the cervical spine, revealing an intramuscular paraspinal cystic lesion on the right side without bony involvement, secondary to nonpyogenic hydatidosis

## Treatment Options

Many superficial abscesses confined to the skin are treated by dermatologists. Superficial, limited infections may be treated with antibiotics alone.

Treatment of paraspinal pyomyositis depends on the stage of the disease. In the pre-suppurative stage, the lesion can be treated with antistaphylococcal antibiotics alone, with consideration of the local rate of infection with MRSA (methicillin-resistant *Staphylococcus aureus*). With abscess formation, open surgical drainage is required, followed by intravenous antibiotic drugs. Radiologic guidance may be useful for deeper abscess collections. Antibiotics given for 3–4 weeks are usually sufficient in patients without complications. Tubercular lesions must be treated with the appropriate antituberculous regimens. Sepsis and septic shock need early and aggressive resuscitation.

## Outcomes

When treated early and adequately, most patients heal without recurrences or additional complications. The possibility of an obvious systemic source of infection (multifocal infec-

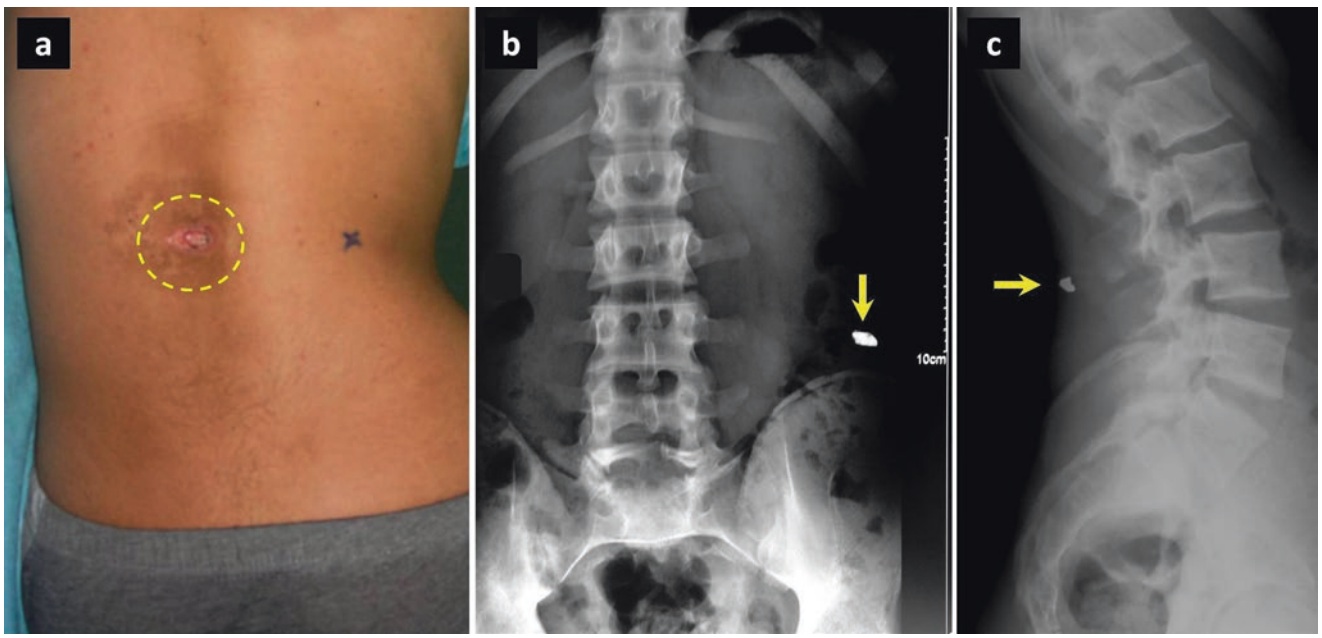


**Fig. 16.11** Case 16.2. Photograph of a patient with a chronic, superficial soft tissue abscess (*dotted circle*) in the posterior lumbar area (**a**). The patient recalls being a victim of lumbar trauma a few months previously. Plain radiographs of the lumbar spine in anteroposterior (**b**) and lateral (**c**) views show a retained metallic foreign body (*arrow*)

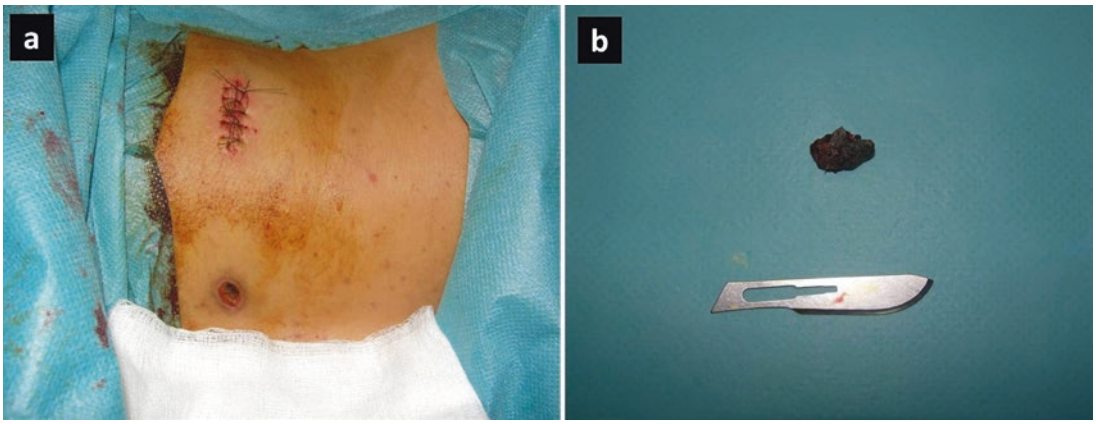


**Fig. 16.12** Case 16.2. Operative view after extraction of the metallic foreign body and debridement of the superficial abscess (**a**, **b**)

tion) should be considered if the patient does not respond adequately to the antibiotic therapy.

Delay in diagnosis may result in osteomyelitis of adjacent spinal or pelvic bones, spinal epidural abscess, retroperitoneal abscess, metastatic infection, sepsis, and occasionally death (Figs. 16.11 and 16.12).

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# Vertebral Body and Discal Infections

# 17

Spondylodiscitis, the most common form of spinal infection, may be pyogenic, granulomatous (tuberculosis, brucellosis, or fungal microorganisms), or parasitic, but the most frequent causative pathogens are *Staphylococcus aureus* and *Mycobacterium tuberculosis*. Hematogenous spread, direct open spinal trauma, and infections from adjacent areas are the three main contamination routes. Clinical symptoms (pain, fever, and spinal deformity) are highly suggestive but may be lacking. Routine laboratory investigations are not specific, except that blood cultures may be positive. MRI is the cornerstone exploration to evaluate for vertebral osteomyelitis and discitis. Particular attention should be paid to the identification of the pathogenic agent and its sensitivity to antimicrobial drugs. CT-controlled biopsy and drainage of paravertebral abscess also can aid in making the diagnosis. Most cases of spontaneous spondylodiscitis can be managed successfully with anti-infectious therapy alone. Surgery is indicated to obtain diagnostic cultures, decompress neural structures, treat spinal instability and significant deformity, and debride infected and necrotic tissue if infection persists or worsens despite appropriate antimicrobial regimens. Untreated spinal infection can result in chronic pain, spinal deformity, neurologic deficits, sepsis, and even death. Fortunately, when diagnosed early and treated adequately, the prognosis of spondylodiscitis is favorable for most patients.

## Epidemiology and Etiology

Infections of the vertebral column involve vertebral bone (*osteomyelitis*), intervertebral disc space (*discitis*), or a combination of both (*spondylodiscitis*) (Fig. 17.1). In this chapter, these three forms of vertebral column infections are grouped together under the term *spondylodiscitis* because vertebral osteomyelitis and discitis rarely occur separately. Spondylodiscitis is a serious disease that can lead to spinal deformities, segmental instabilities, or neurologic deficits. Infection may be pyogenic, granulomatous (tuberculosis, brucellosis, or fungal microorganisms), or parasitic. The three main contamination routes are hematogenous spread (arterial or venous), direct open spinal trauma, and infections from adjacent areas.

The lumbar spine (60%) and thoracic spine (30%) are the areas most often affected. Infections of the cervical or sacral segment (10%) or multilevel infections (5%) occur more rarely. Concomitant infection of the surrounded paraspinal soft tissues and/or the spinal canal structures may be seen (as in Chap. 24, "Spinal Tuberculosis") (Figs. 17.2, 17.3, 17.4, 17.5, 17.6, and 17.7). Spondylodiscitis following a surgical procedure is unusual, but it is a well-known postoperative complication (s*ee* Chap. 22).

Predisposing factors for the development of spinal infections include intravenous drug abuse, alcoholism, diabetes mellitus, immunocompromised states, chronic renal failure, cirrhosis, and some extraspinal infections (heart, urinary tract, pelvis, skin, teeth, and lungs).

Spondylodiscitis can occur at any age but is most prevalent in childhood and in people over 65 years of age, with a male predominance.

## Clinical Presentations

Signs and symptoms vary depending on the type of spinal infection and the region involved, but generally, pain is localized initially at the site of the infection. Other clinical data include fever, paraspinal muscle spasms and rigidity, spinal deformity or angled gibbosity, radicular pain, and neurologic deficits in different combinations. Obviously, the past medical history should be checked for any predisposing factors. Sensory involvement is less common than motor signs because compression is primarily anterior. Systemic findings may also be present, including chills, night sweats, weight loss, anorexia, and general malaise. More rarely, chronic vertebral spondylodiscitis may present with a cutaneous draining sinus tract.

The clinical presentation can be acute, subacute, or chronic, but the course of the disease is often long and more



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_17

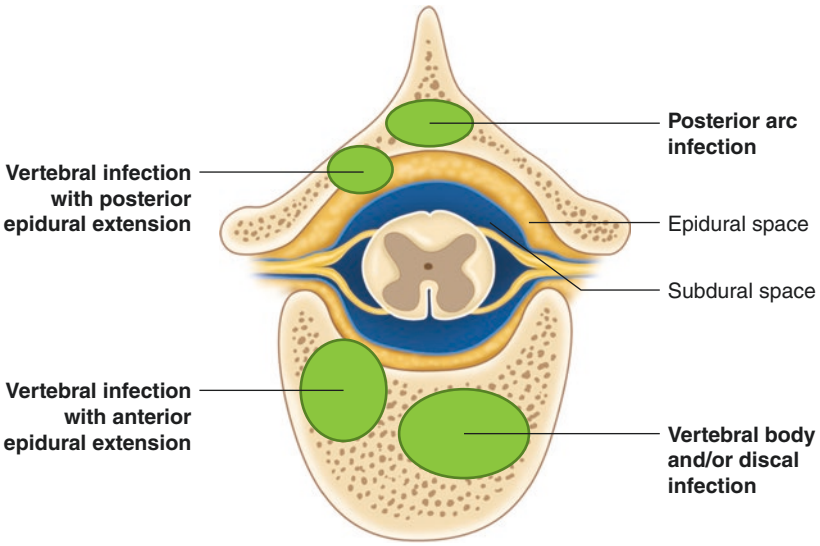


**Fig. 17.1** Topographic classification of spinal infections

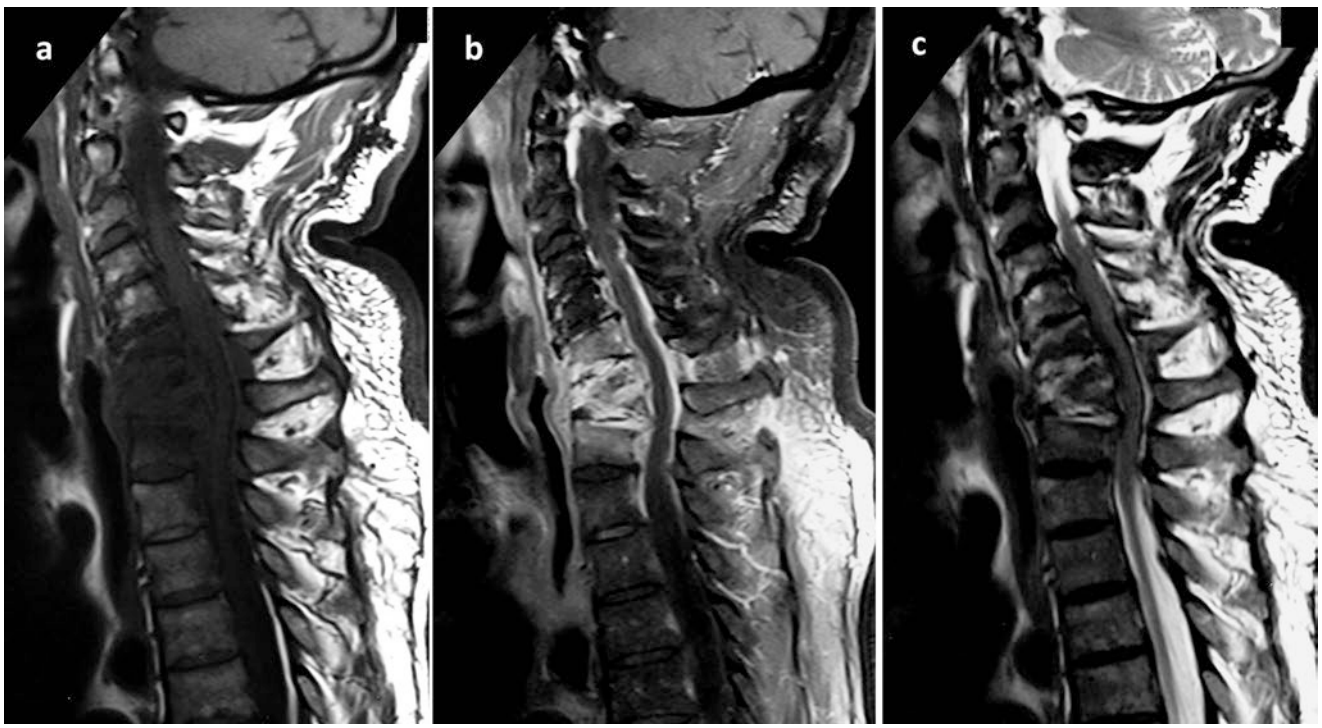


**Fig. 17.2** Case 17.1. Cervicothoracic (C6–T1) bacterial spondylodiscitis in a 62-year-old man with a history of diabetes mellitus and obesity. The involved area demonstrates hypointensity on a T1-weighted MR image (**a**), enhancement on a T1-weighted image with gadolinium (**b**), and focal hyperintensity on a T2-weighted image (**c**)

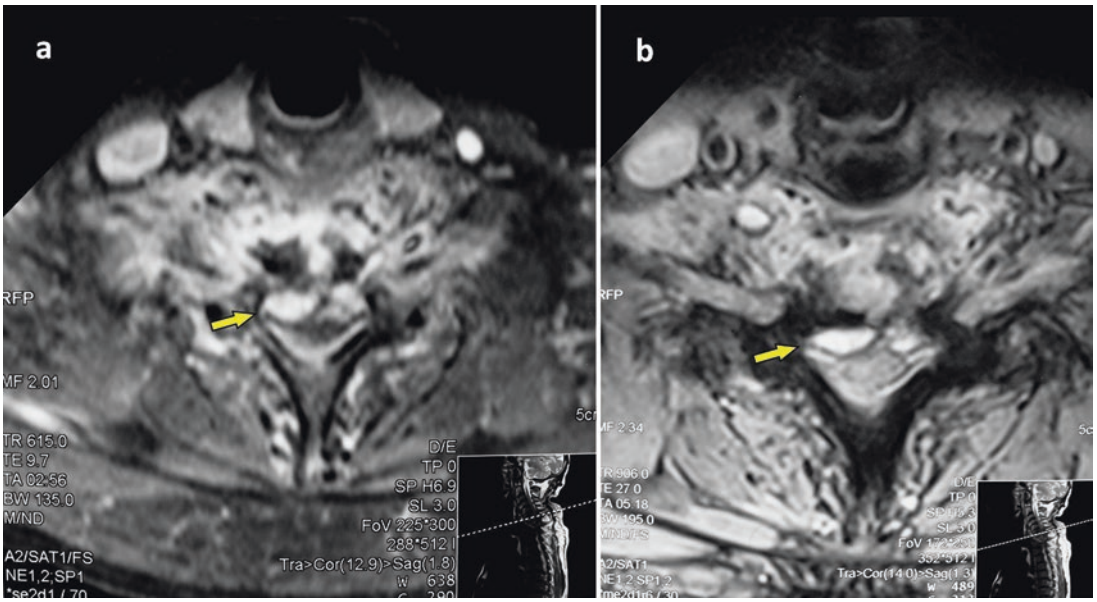


**Fig. 17.3** Case 17.1. Axial post-gadolinium T1-weighted MR image (**a**) and a T2-weighted image (**b**) revealing spinal canal involvement and an epidural abscess (*arrow*)

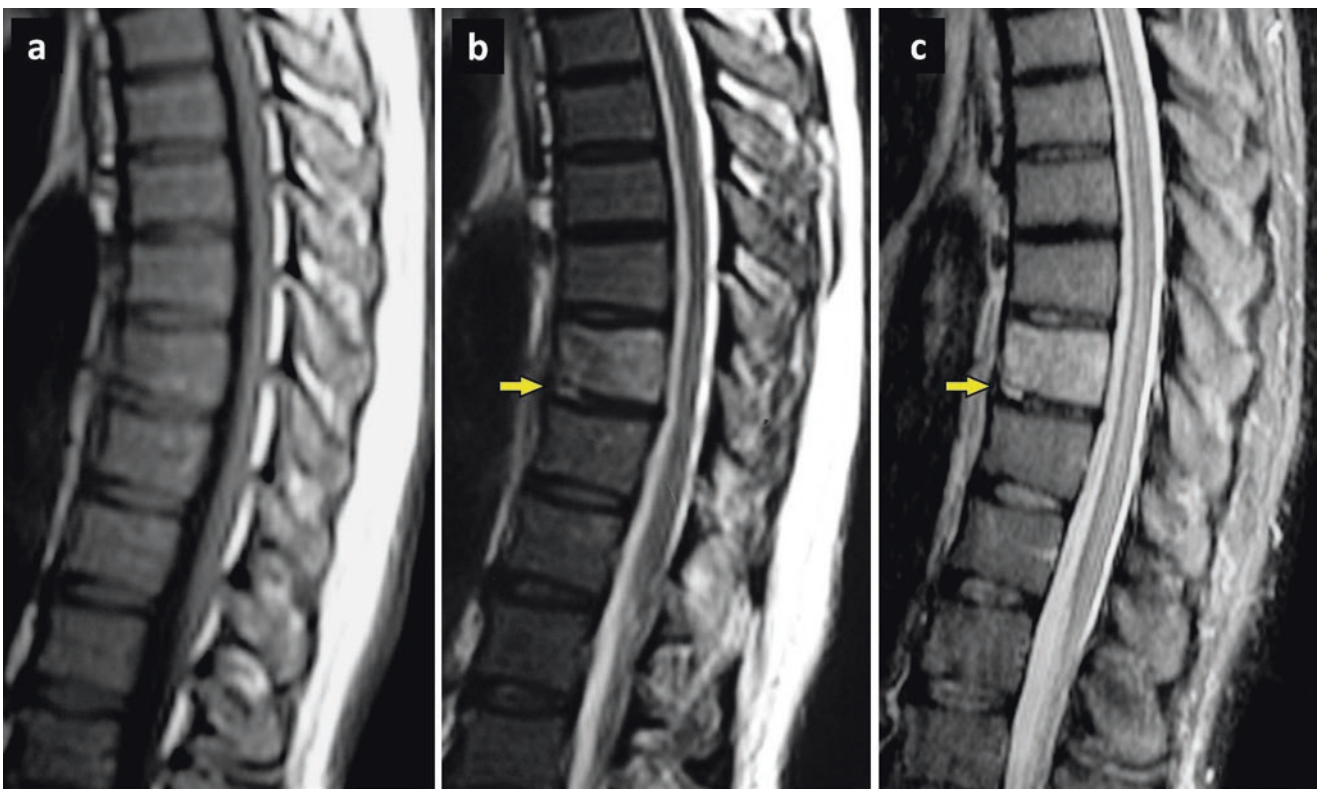


**Fig. 17.4** Case 17.2. This 62-year-old man was using corticosteroid for rheumatoid arthritis. He presented with a 2-week history of back pain and fever without neurologic symptoms. The C-reactive protein levels were high. Spinal thoracic MRI shows osteomyelitis of T10 with small involvement of the anterior part of the T10–T11 disc space (*arrow*). Sagittal T1-weighted image (**a**) without gadolinium administration, T2-weighted image (**b**), and short tau inversion recovery (STIR) sequence (**c**). Note the modification of bone marrow signal intensity (early stage) without osseous destruction (**b**, **c**). The patient's symptoms improved under empiric antibiotherapy

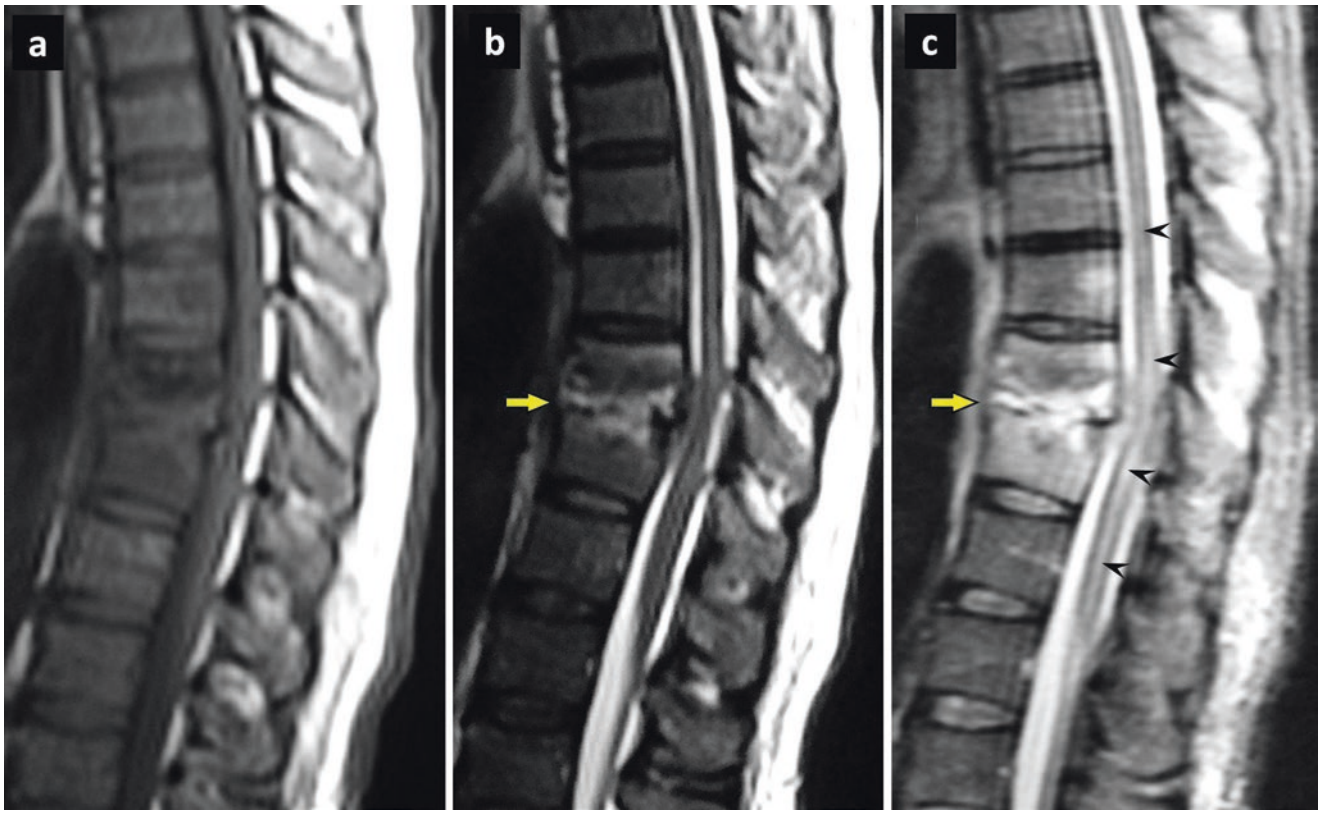


**Fig. 17.5** Case 17.2. Three months later, he presented with acute paraparesis. Spinal thoracic MRI shows features of T10–T11 spondylodiscitis (*arrow*) with spinal intracanalar extensions. Sagittal T1-weighted image (**a**) without gadolinium administration, T2-weighted image (**b**), and STIR sequence (**c**), which shows high signal intensity within the spinal cord (*black arrowheads*)

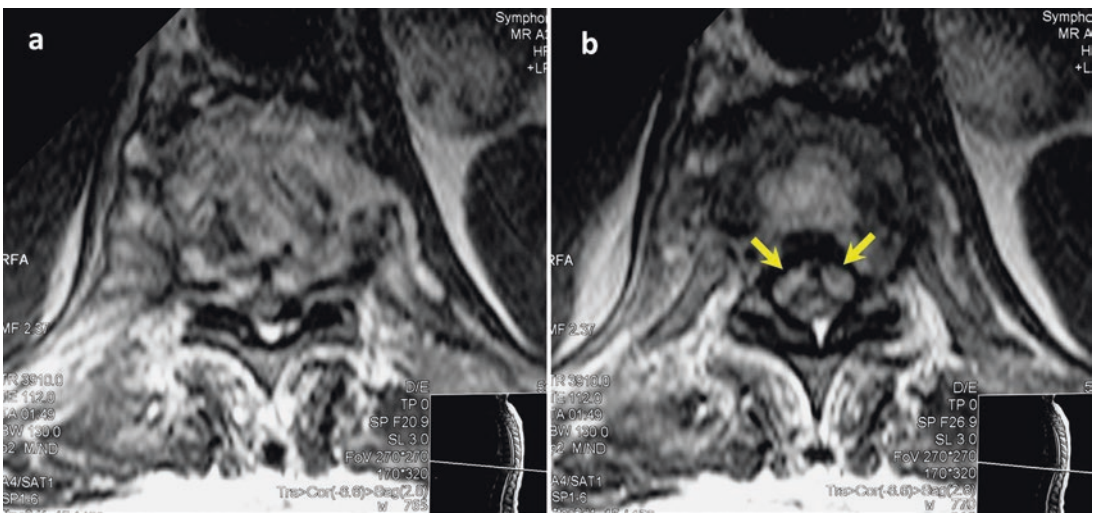


**Fig. 17.6** Case 17.2. Axial T2-weighted images (**a**, **b**), revealing the T10–T11 discitis with anterior epidural abscesses (*arrows*)

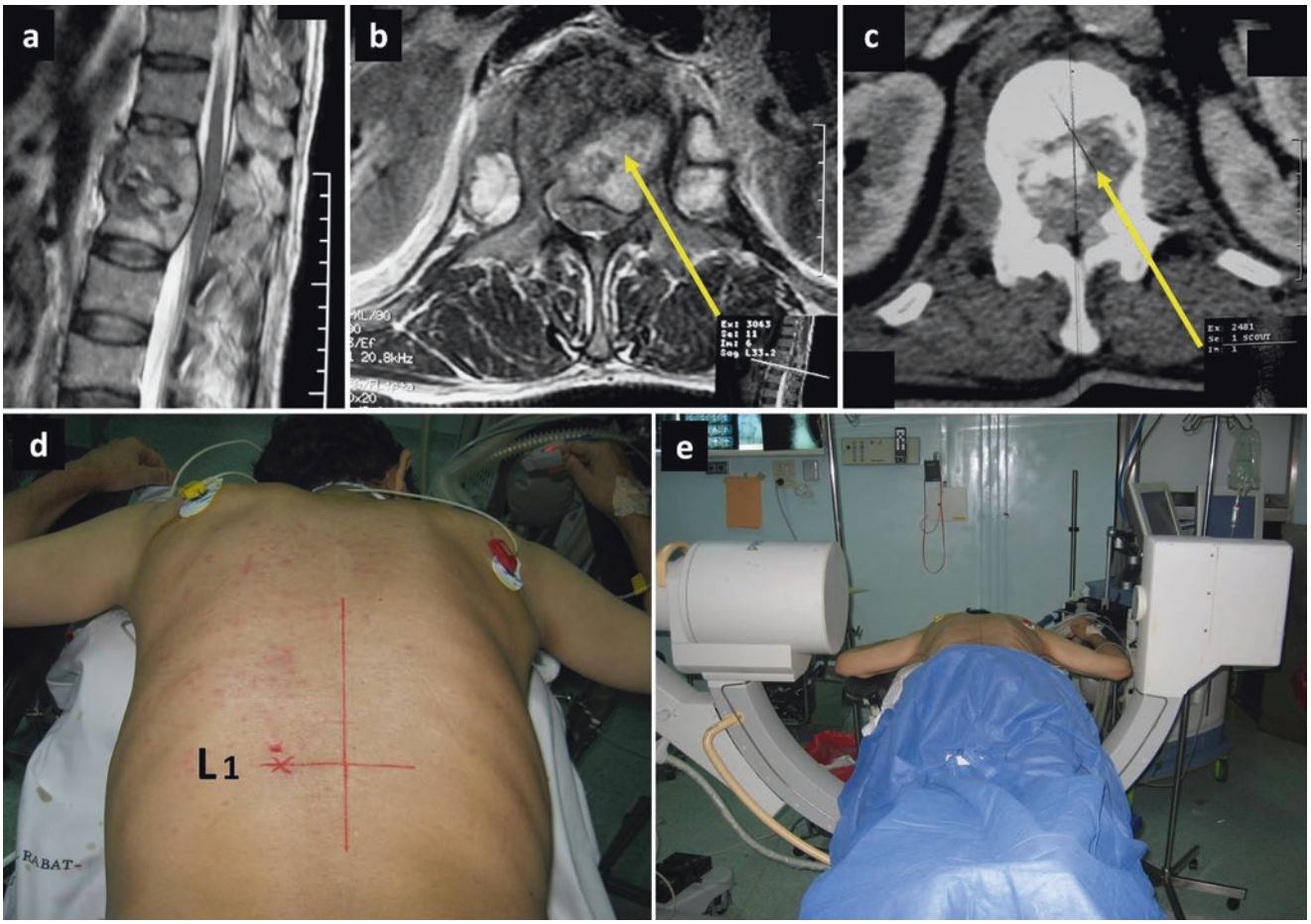


**Fig. 17.7** Thoracolumbar (T12–L1) bacterial spondylodiscitis in a 43-year-old man. Both sagittal (**a**) and axial (**b**) T2-weighted MR images reveal the lesion with conus medullaris compression and bilateral paravertebral extension (abscesses). An axial CT scan (**c**) shows vertebral bone destruction and the site of a posterior lumbar percutaneous biopsy (left side) (*arrow*) for microbiologic studies and culture, which confirmed the infection due to *Staphylococcus aureus*. The surgical procedure (percutaneous) was performed under radioscopic guidance (**d**, **e**)

indolent in granulomatous and parasitic spondylodiscitis than in pyogenic cases. Fever is less likely with granulomatous infections and is generally absent in parasitic spondylodiscitis.

## Imaging Features

Imaging studies are the mainstay to identify the location and extent of the spinal lesions. Plain radiographs may be useful as an initial screening study, but narrowing of the intervertebral disc space and erosion of adjacent end plates may not be seen because changes may take several weeks to appear. Nuclear medicine imaging explorations, particularly single-photon emission computed tomography (SPECT) combined with CT scan and fluorodeoxyglucose positron emission tomography (FDG-PET), are more sensitive in early detection of suspected spondylodiscitis before much more spinal destruction occurs.

CT scans give important information on the degree of bone destruction, which is particularly useful for surgical planning and performing image-guided percutaneous biopsies and paraspinal abscess drainage, if needed. MRI with gadolinium administration is considered the best imaging modality for diagnosis of spondylodiscitis because of its high sensitivity, specificity, and anatomical data. On T1-weighted images, the vertebral body and the narrowed intervertebral disc space have low signal intensity. After gadolinium injection, the inflammatory lesions will enhance, especially the vertebral end plates and combined abscess formations. On T2-weighted images, the bony and discal lesions appear hyperintense, owing to increased edema. Fat suppression sequences may help in the diagnosis by subtracting the high signals of bone marrow and epidural fat. MRI is also useful in demonstrating spinal canal lesions, especially associated spinal epidural abscess and spinal cord compression (Figs. 17.8, 17.9, 17.10, 17.11, 17.12, 17.13, 17.14, 17.15, and 17.16).

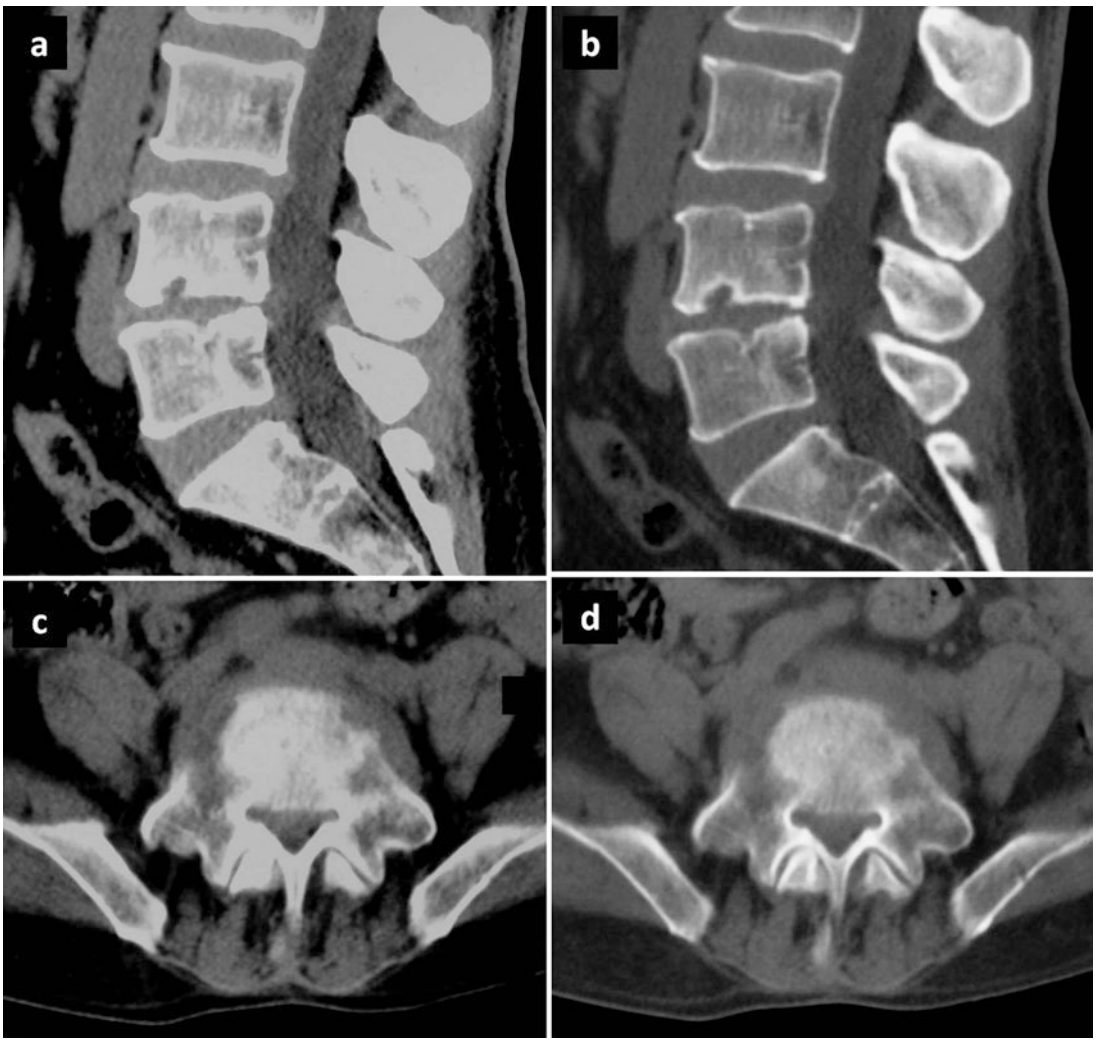


**Fig. 17.8** Case 17.3. Lumbar (L4–L5) spondylodiscitis in a 65-year-old man who presented with a 2-month history of bilateral lumbosciatalgia without fever. Both sagittal (**a**, **b**) and axial (**c**, **d**) CT scans reveal vertebral end plate destruction and narrowed disc space

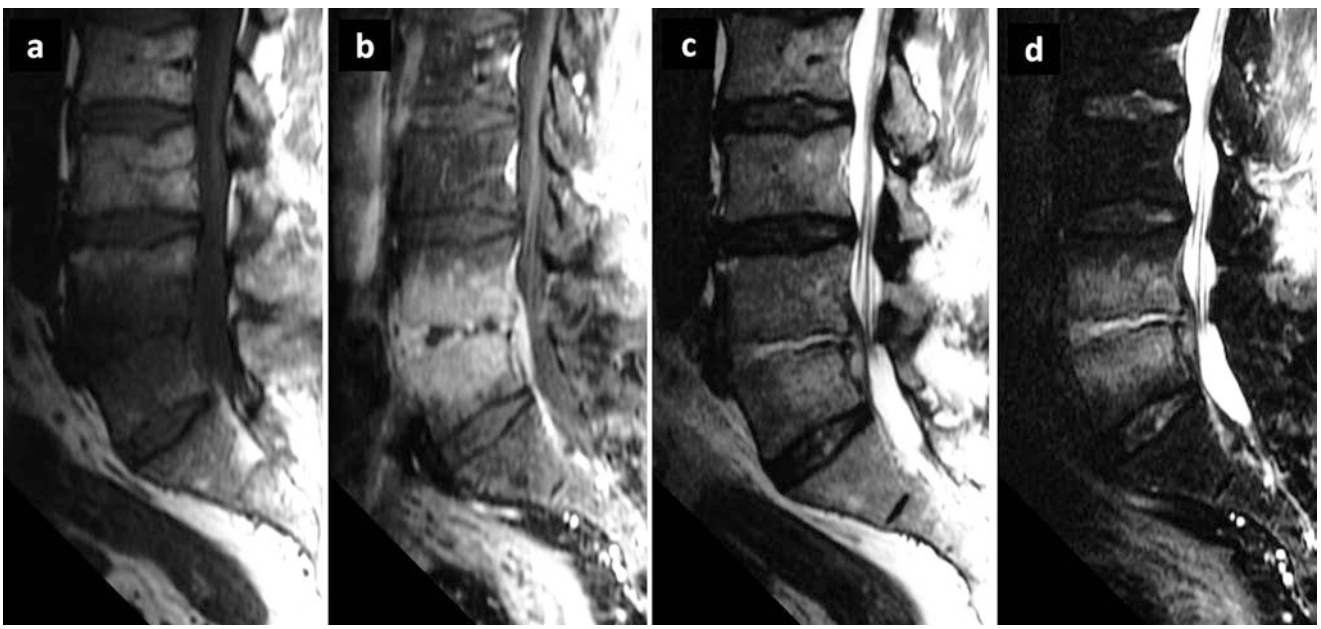


**Fig. 17.9** Case 17.3. Sagittal T1-weighted MR images before (**a**) and after (**b**) gadolinium administration, a T2-weighted image (**c**), and a STIR sequence (**d**). The lesion was highly enhanced after gadolinium injection, showing extensive adjacent edema and spinal intracanalar extension

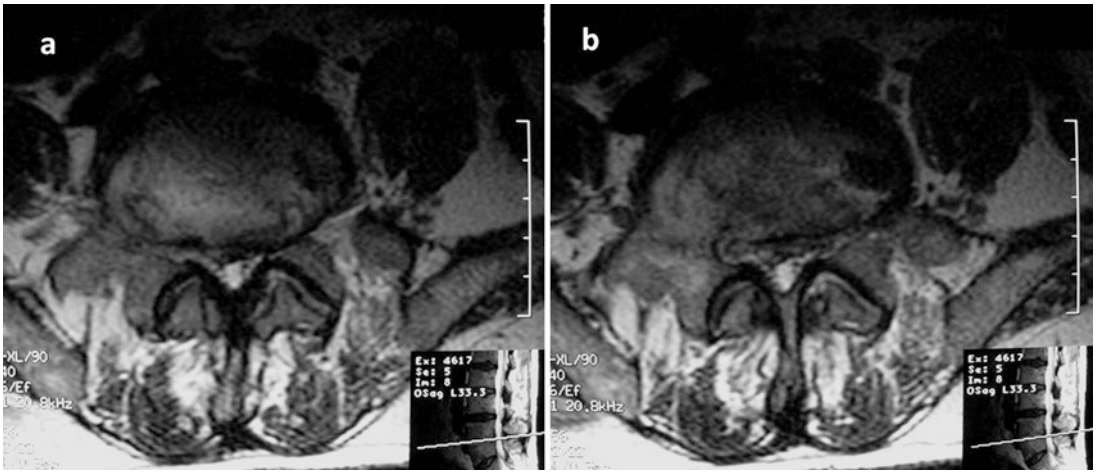


**Fig. 17.10** Case 17.3. The spinal cauda equina compression was also seen on axial T2-weighted images (**a**, **b**)

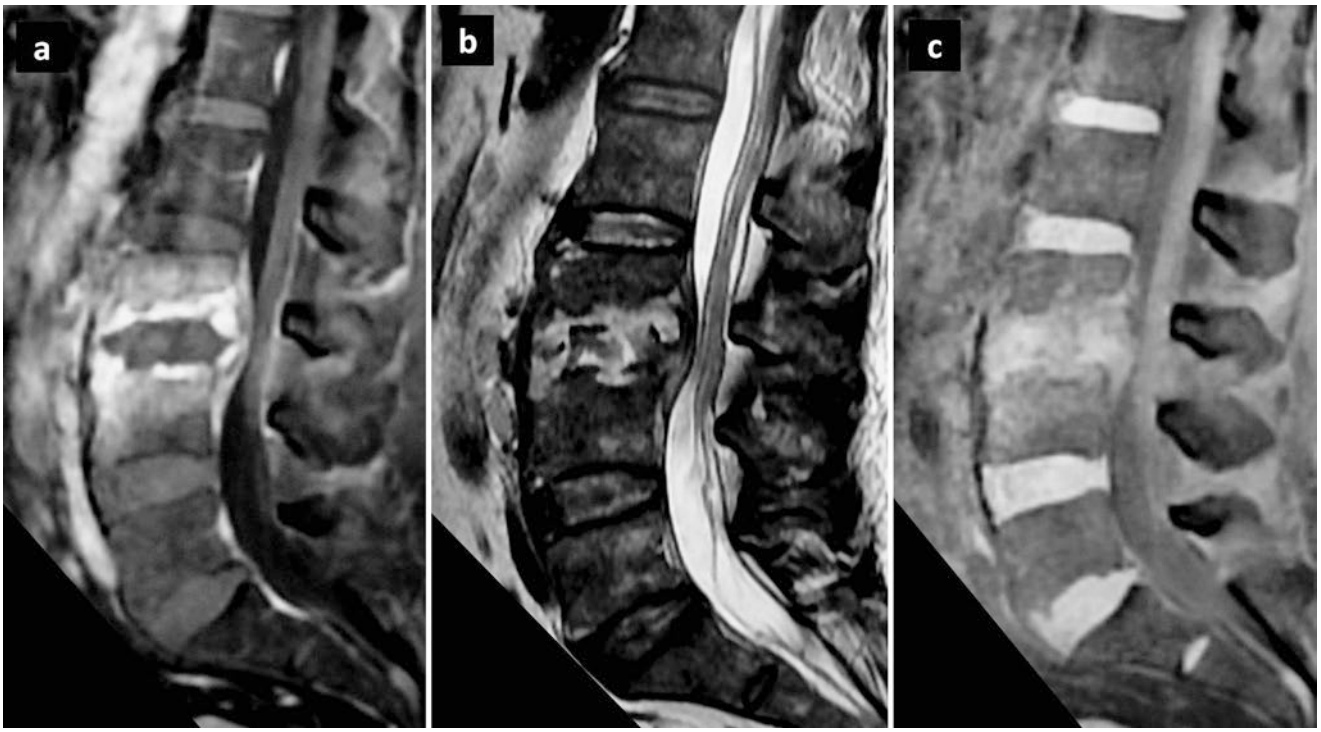


**Fig. 17.11** Case 17.4. Lumbar L3–L4 bacterial spondylodiscitis in a 54-year-old diabetic woman. Sagittal enhanced T1-weighted image (**a**), T2-weighted image (**b**), and STIR sequence (**c**)

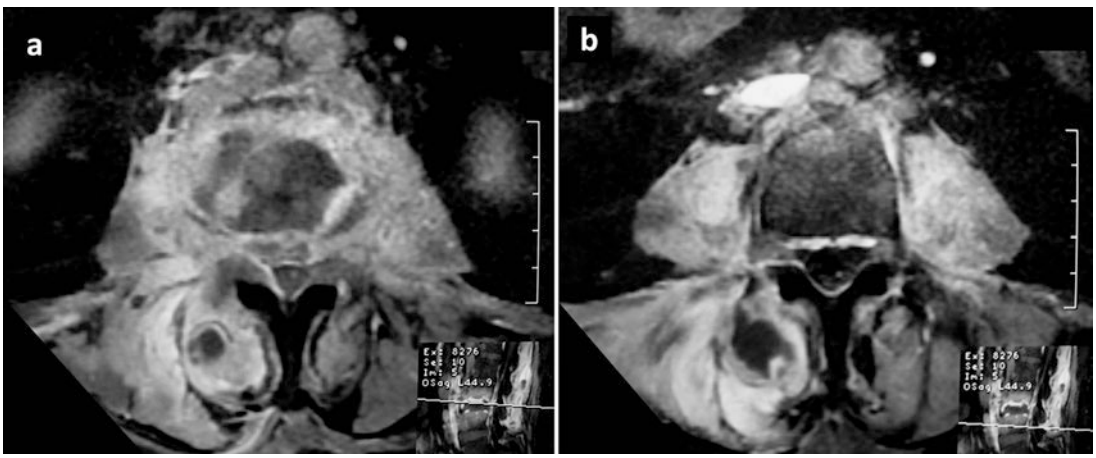


**Fig. 17.12** Case 17.4. Axial T1-weighted images following gadolinium administration show paravertebral and intracanalar extension with enhancement (**a**, **b**)

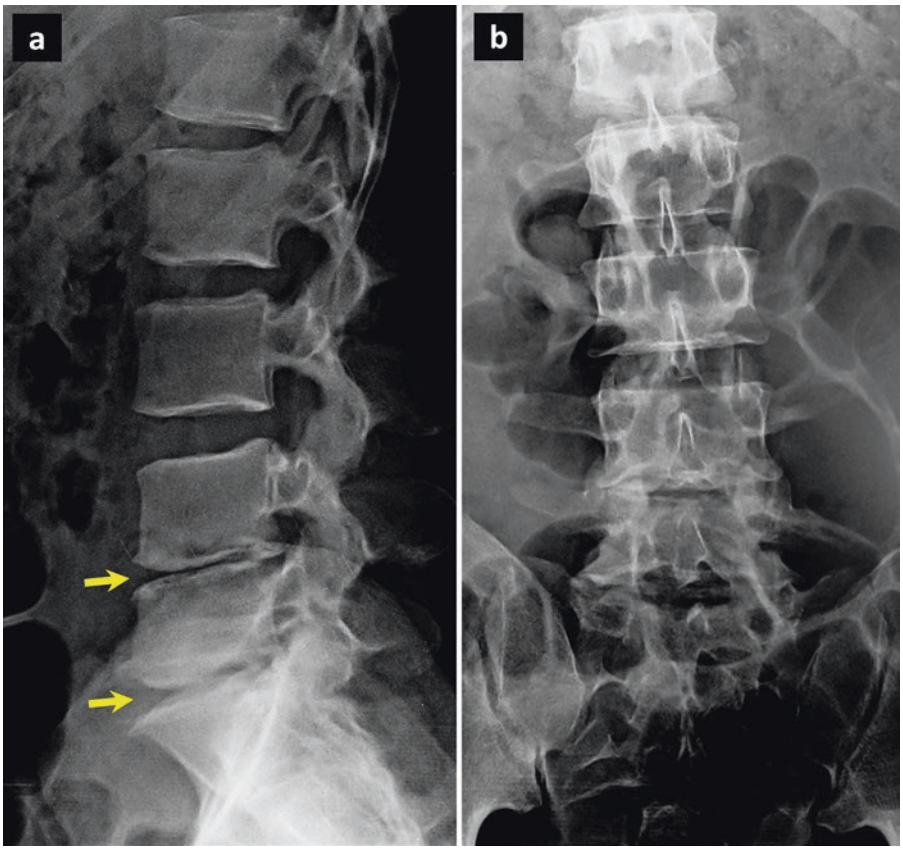


**Fig. 17.13** Case 17.5. This 36-year-old obese man had a long history of back pain. He presented with 2 weeks of right sciatica without fever. Lateral (**a**) and anteroposterior (**b**) lumbar spinal radiographs revealed narrowed disc space at the L4–L5 and L5–S1 levels (*arrows*)

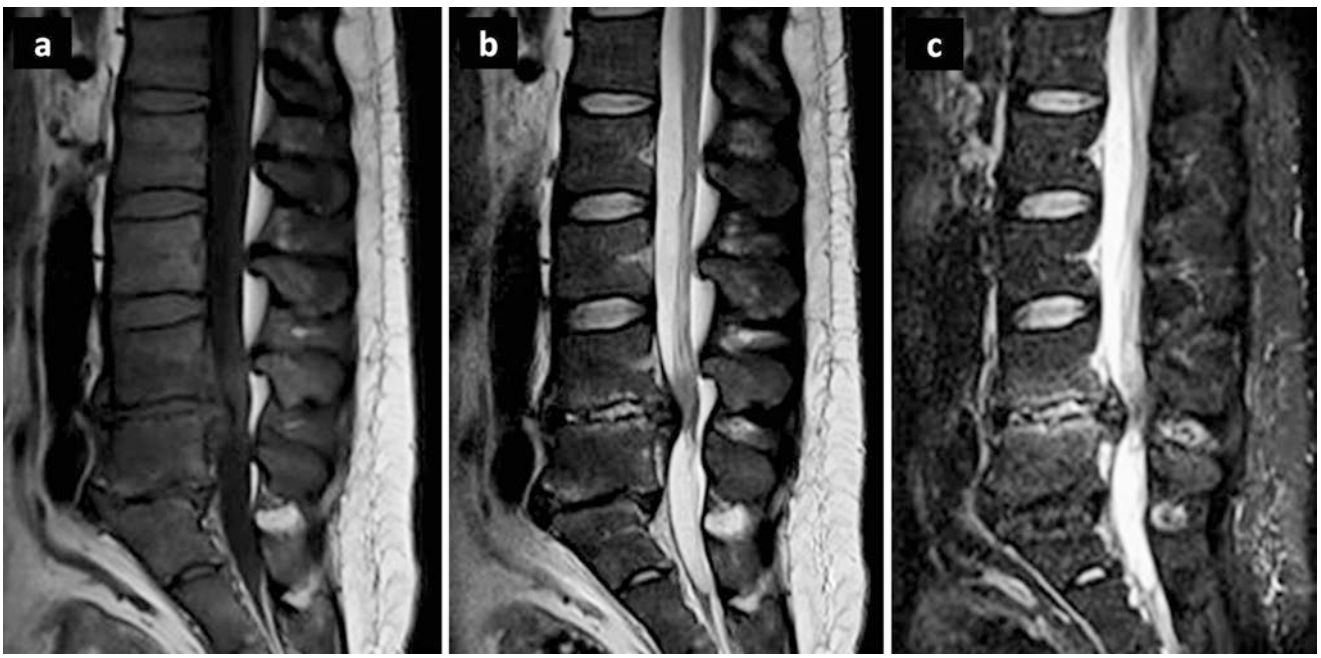


**Fig. 17.14** Case 17.5. Sagittal noncontrast T1-weighted MRI (**a**), T2-weighted MRI (**b**), and a STIR sequence (**c**) were highly suggestive for lumbar disc herniations (no bony destruction)

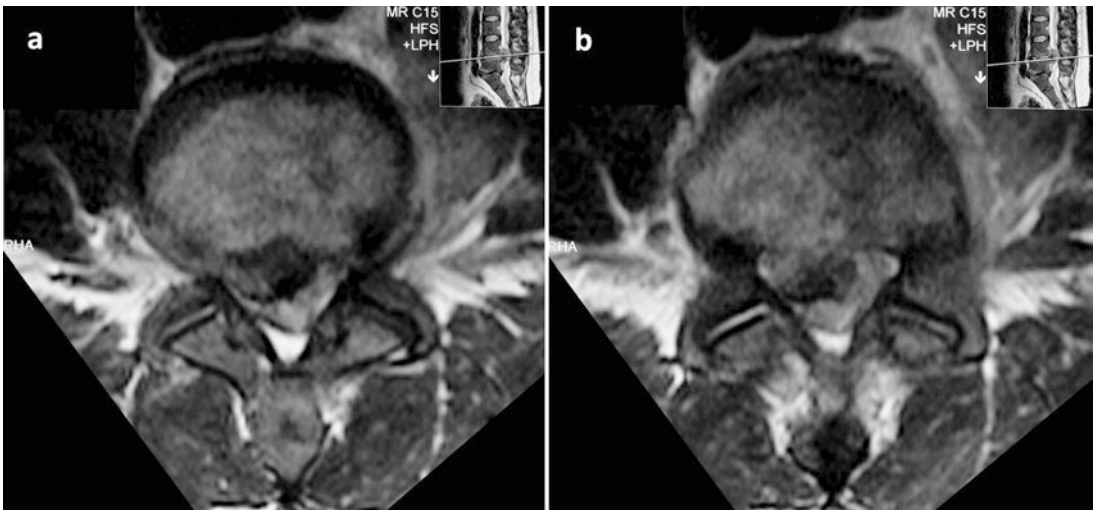


**Fig. 17.15** Case 17.5. Axial T2-weighted images (**a**, **b**) show the intracanalar extension of the lesion (largest on the right side). The patient's erythrocyte sedimentation rate and white blood cell count were normal, but C-reactive protein levels were elevated

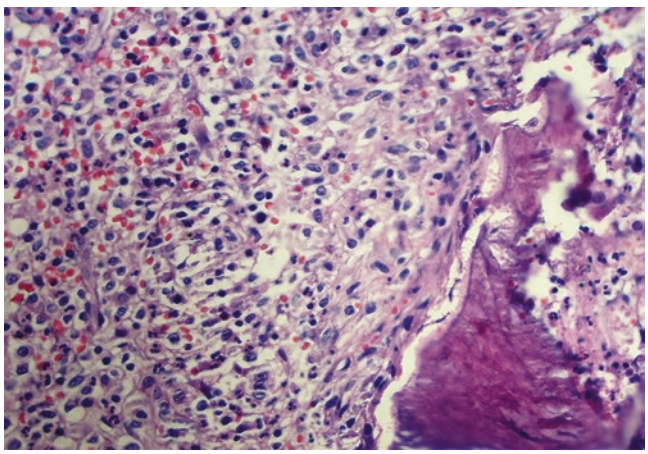

**Fig. 17.16** Case 17.5. During surgery, the disc (L4–L5) had an "unusual" friable appearance with abnormal hemorrhagic end plates; an infection due to *Staphylococcus aureus* was confirmed. Histopathologic features of the end plate reveal subacute osteomyelitis: the presence of bone necrosis with lymphoplasmocytic inflammatory infiltrates and neutrophilic granulocytes (hematoxylin–eosin staining, high-power magnification)

Many differential diagnoses should be considered, including osteoporotic fractures, degenerative changes, inflammatory spondyloarthropathies, and spinal tumors (especially metastatic neoplasms).

## Laboratory Findings

Routine laboratory investigations are not specific but may be useful in establishing the diagnosis of spondylodiscitis, especially when the clinical signs and symptoms are also not specific. Blood tests can sometimes demonstrate an elevated peripheral white blood cell count with neutrophilia and abnormal C-reactive protein (CRP) levels and erythrocyte sedimentation rate (ESR). (The ESR and CRP are more valuable in monitoring the therapy effect, however.) Procalcitonin levels seem more specific than CRP levels for pyogenic bacterial infectious stimuli. Blood cultures may be positive. Cultures from other potential sources of infection are important in identifying causative pathogens.

Spondylodiscitis may be bacterial, mycobacterial, fungal, or parasitic (*see* Section V regarding specific pathogens). The most common individual pathogen is *Staphylococcus aureus*, followed by gram-negative rods (particularly *Escherichia coli* and *Proteus* species), *Streptococcus* species, and *Enterococcus* species. *Mycobacterium tuberculosis* (*see* Chap. 24) is more common in many developing countries (especially in Southeast Asia and Africa). On the other hand, brucellosis is seen in countries around the Mediterranean Sea and is associated with animal contact or the ingestion of unpasteurized milk (Figs. 17.17, 17.18, 17.19, and 17.20). Fungal types of spondylodiscitis (like aspergillosis, blastomycosis, candidiasis, coccidioidomycosis, and histoplasmosis) are rare but are more likely to be encountered in immunocompromised populations. Although rare, hydatidosis is the most frequent parasitic spondylodiscitis (*see* Chap. 27). Polymicrobial infections are uncommon and usually result from contiguous spread.

Percutaneous CT-controlled biopsy is very helpful in making the diagnosis (up to 70% of positive results). Histopathologic studies are an important tool for diagnosing some specific infections such as mycobacteria, fungi, and parasites. Blood cultures should also be performed a few

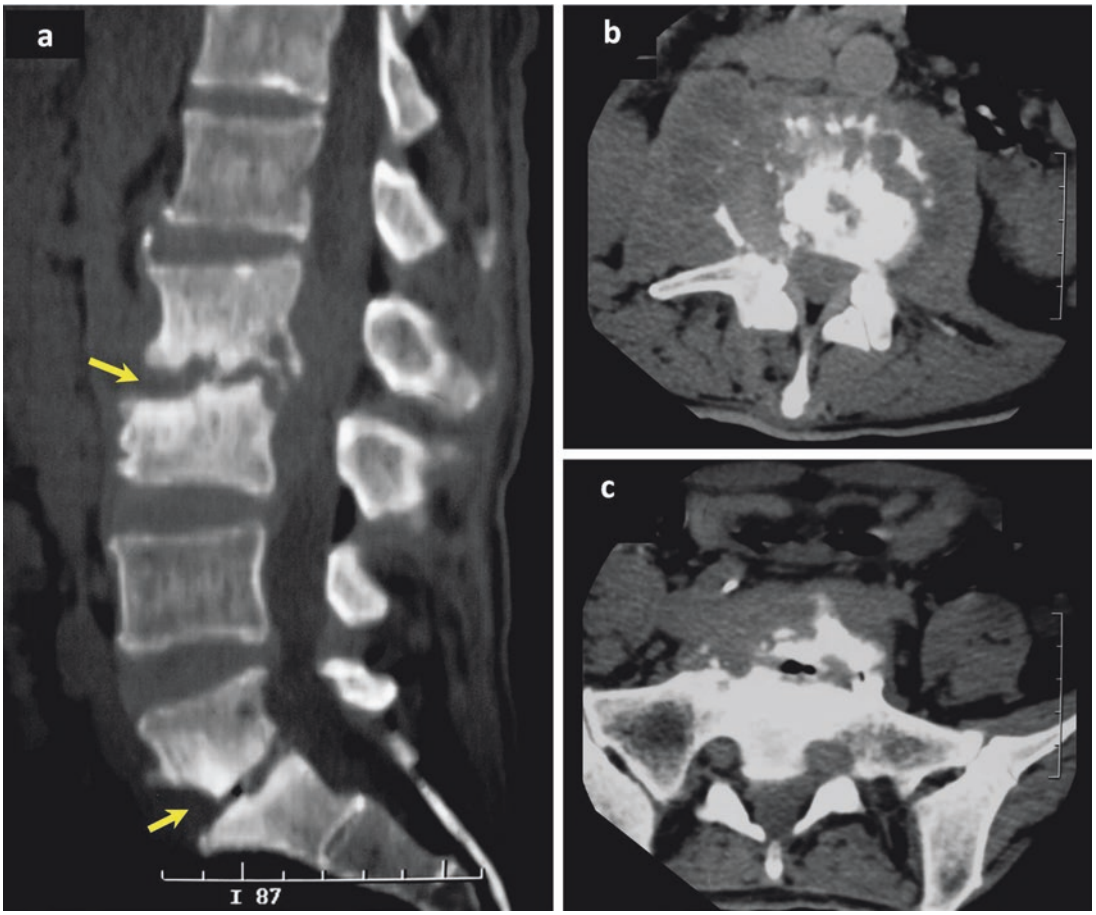


**Fig. 17.17** Case 17.6. Sagittal (**a**) and axial (**b**, **c**) lumbar CT scans on bone windows show bifocal lumbar spondylodiscitis (L2–L3 and L5–S1) (*arrows*) due to *Brucella melitensis*

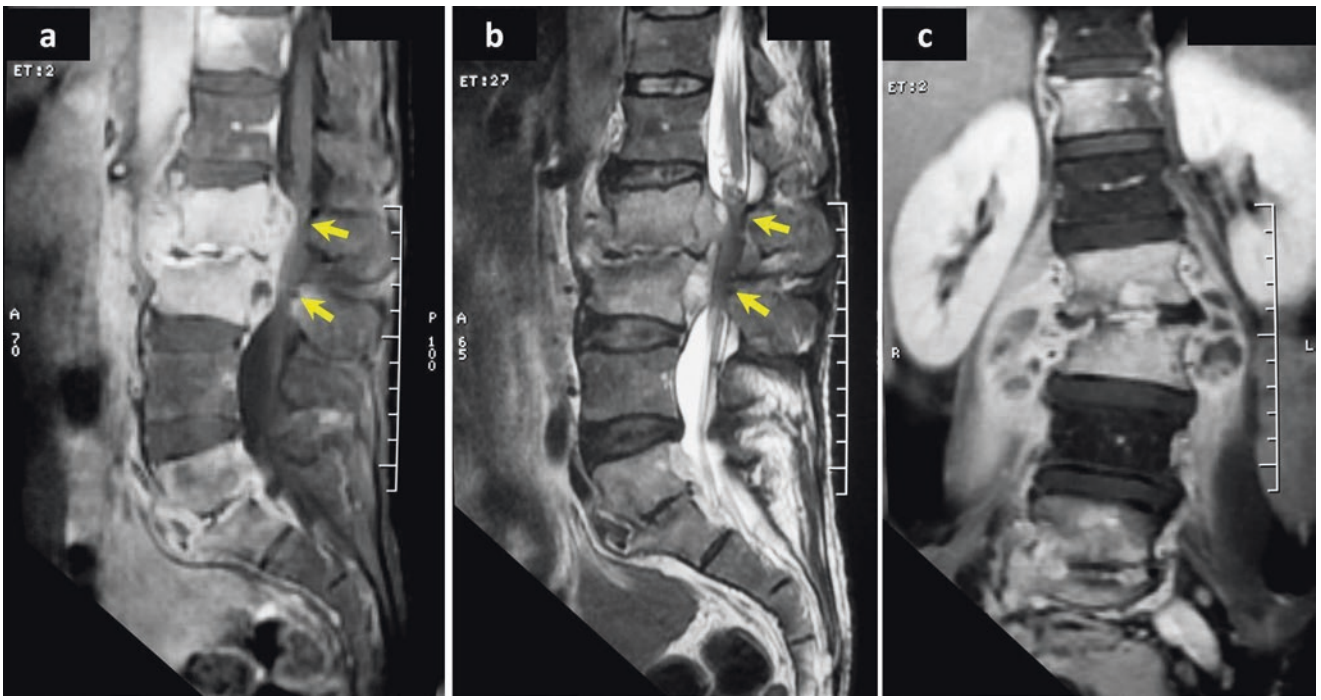


**Fig. 17.18** Case 17.6. Sagittal enhanced T1-weighted MRI (**a**), T2-weighted MRI (**b**), and a coronal T1-weighted image with gadolinium injection (**c**) show multilevel, noncontiguous spondylodiscitis with cauda equina compression (*arrows*)

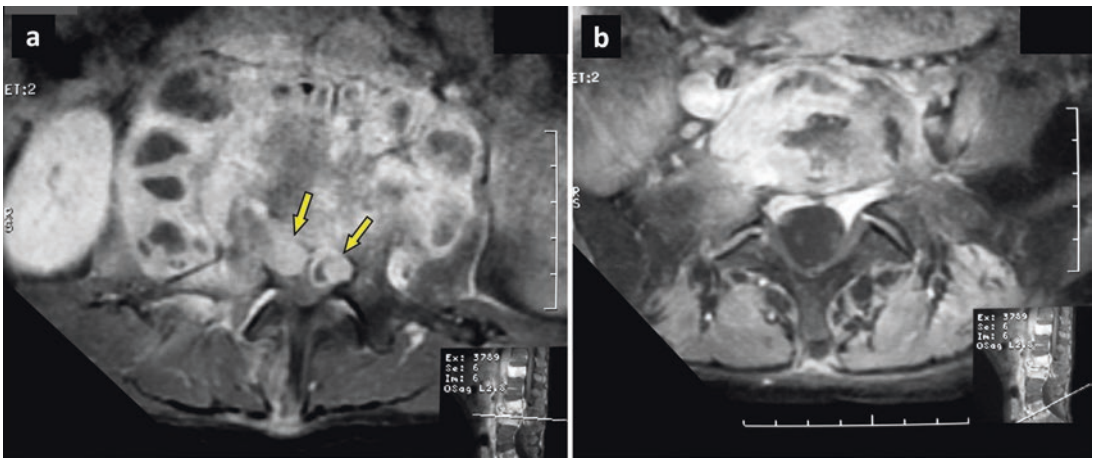


**Fig. 17.19** Case 17.6. Axial T1-weighted MRI following gadolinium injection through L2–L3 (**a**) and L5–S1 (**b**) reveal the spinal canal involvement (*arrows*) and bilateral paraspinal tissue extension

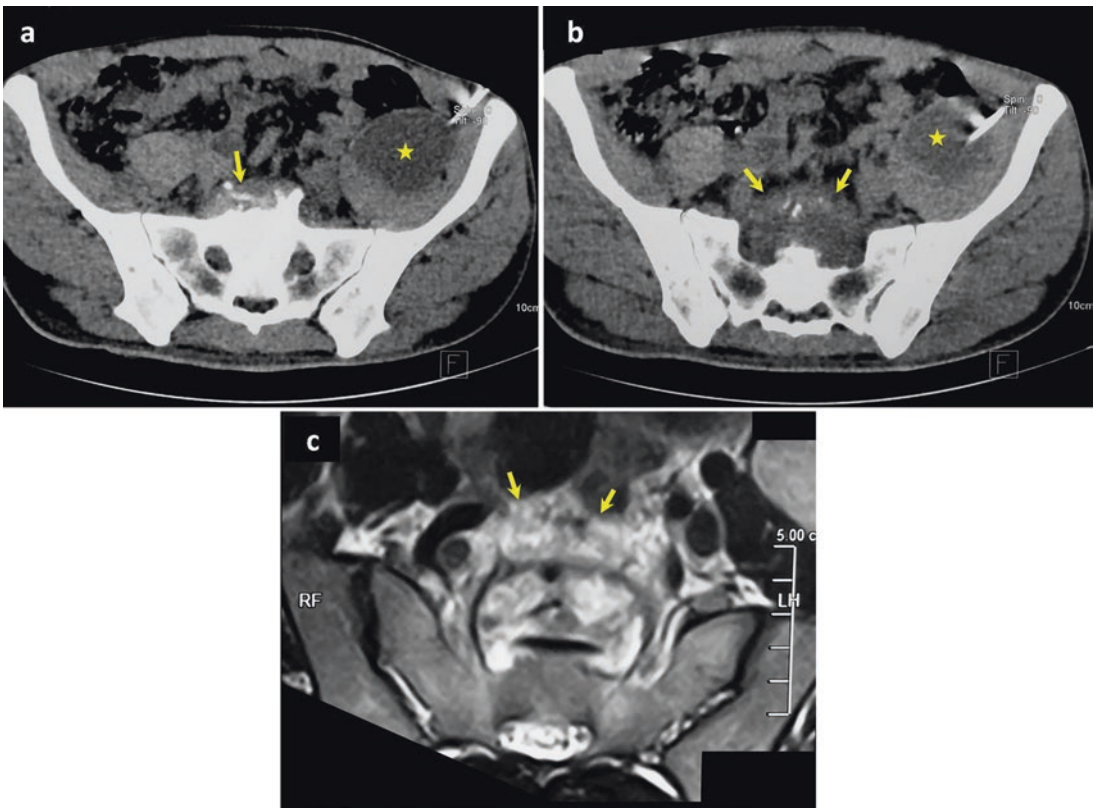


**Fig. 17.20** Brucellosis of the sacral spine. Axial post-contrast CT scan (**a**, **b**) and T2-weighted MRI (**c**). Note the anterior bone erosion of the sacrum (S2–S3) (*arrows*) and the left iliopsoas abscess (*star*)

hours after percutaneous biopsy. Open surgical biopsy is necessary if both blood cultures and percutaneous CT-guided biopsy fail to give a positive diagnosis. Serologic or antigen testing for specific bacteria, fungi, or parasitic infections may be performed in patients from areas where such diseases are endemic.

## Treatment Options

The goal of the treatment of spondylodiscitis is to eliminate infection with preservation of spinal stability and neurologic function.

Most patients require long-term intravenous antibiotic, antifungal, or antiparasitic drugs, with extended hospitalization. External immobilization (an orthosis, brace, or collar) may be recommended when there is significant pain or the potential for spine instability. If the patient is neurologically and structurally stable, antibiotic treatment should be administered after identification of the causative pathogen. Patients generally undergo antimicrobial therapy for a minimum of 6–8 weeks. The type of medication is determined on a case-by-case basis depending on the patient's underlying conditions, drug sensitivities of the responsible microorganism, and the severity of the infection. When cultures remain negative, a combination of a third-generation cephalosporin, vancomycin, and metronidazole is recommended, against the most frequently responsible bacteria.

For patients with spinal tuberculosis, the first-line regimen is a combination of isoniazid, rifampicin, pyrazinamide, and ethambutol or streptomycin for 2 months, followed by two drugs (isoniazid and rifampicin) for 6–9 months (*see* Chap. 24). Brucellosis is often treated with a combination of doxycycline and rifampicin for 6 months. Fungal infections must be treated with the appropriate anti-infectious regimens (amphotericin B or azole agents). Oral anthelmintic drugs such as albendazole and mebendazole are promising for spinal hydatidosis (*see* Chap. 27).

Surgery is indicated to obtain diagnostic cultures, decompress neural structures (spinal cord and/or spinal nerve roots), maintain or restore spinal stability, treat significant deformity, drain associated abscesses, and debride infected and necrotic tissue if infection persists or worsens despite appropriate antimicrobial drugs. Various spinal approaches—anterior, posterior, posterolateral, or combined—can be used. Despite infection, fusion and/or instrumentation (plates, screws, hooks, cages, and rods) may be employed.

Physical rehabilitation, adequate nutrition, and treatment of medical comorbidities are important to the successful treatment of spondylodiscitis.

## Outcomes

Untreated spinal infection can result in chronic pain, spinal deformity, neurologic deficits, sepsis, and even death.

Prognosis of spondylodiscitis depends on the state of general health, the extent of the local lesion, the causative pathogens, the early establishment of the diagnosis, and the response to therapy. Overall, the prognosis is usually favorable and mortality is rare. The recurrence rate is about 10%, so spontaneous spondylodiscitis requires a long follow-up period for evidence of therapy failure. The most likely sequelae associated with spinal infections include chronic pain, persistent weakness, lower extremity spasticity, sphincter dysfunction, pseudarthrosis, and chronic infection.

Mortality is often related to a missed diagnosis, coexisting comorbidities, or concomitant infections, or death may occur as a complication of residual paraplegia or tetraplegia.

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# Spinal Epidural Abscesses

# 18

In spinal epidural abscess (SEA), purulent material collects between the dura and osseous–ligamentous structures of the spine (Figs. 18.1 and 18.2). It is a rare spinal infection with increasing incidence. The main causative factors include hematogenous spread, contiguous extension, and direct inoculation. Combination with a spondylodiscitis is not rare. SEAs are more common in the elderly, especially patients with multiple comorbidities. Major risk factors include intravenous drug abuse, diabetes mellitus, chronic renal failure, alcoholism, malignancy, and other immunocompromised conditions. The most common symptoms and signs are neck or back pain, fever, and neurologic deficits, but the presentation can be ambiguous and variable, resulting in delayed diagnosis. MRI is the imaging modality of choice. The epidural collection appears isointense or hypointense on T1-weighted images with gadolinium enhancement and nonhomogeneous hyperintense on T2-weighted images. The most frequent pathogens responsible are *Staphylococcus* and *Streptococcus* species. Anaerobic and fungal infections are rare. Surgery with adjuvant antibiotics remains the optimal treatment for the neurologically symptomatic patient. Surgical therapy allows neurologic decompression, microbiological source control, and spinal stabilization, if needed. Although neurologic deficits can decrease after surgical decompression and drainage, multiple comorbidities, delayed diagnosis, and a complete motor deficit predispose to a poorer outcome.

## Epidemiology and Etiology

Spinal epidural abscess (SEA), also known as spinal extradural abscess, is a focal, purulent collection developing in the space between the spinal dura and osseous–ligamentous structures of the spine. It is a rare infection of the central nervous system, but the incidence may be increasing. SEA is more common than spinal subdural abscess and spinal cord abscess. All spinal canal infections have potential for permanent damage to the spinal cord and even sepsis.

The dura mater adheres to the posterior longitudinal ligament anteriorly, so abscess collection is unusual in the anterior part of the epidural space unless there is a concomitant infection in the vertebral body (spinal osteomyelitis), disc space (discitis), or both (spondylodiscitis). There are no anatomic limitations to extension of an SEA in the posterior and lateral epidural spaces, so abscesses tend to be more extensive. SEA rarely spreads into the subarachnoid space or the spinal cord parenchyma.

The development of this infectious disease mainly occurs as a result of hematogenous spread from a distant source of infection (urinary tract infection, pneumonia, retropharyngeal abscess, endocarditis, septic arthritis, skin infection, or intra-abdominal abscess), contiguous extension from neighboring infections (vertebral osteomyelitis and/or discitis or psoas abscess), and direct inoculation (epidural anesthesia, surgery, implanted devices, foreign bodies, lumbar puncture, penetrating injuries, and decubitus ulcer). Some cases remain cryptogenic.

Underlying diseases and risk factors identified in patients with SEA include intravenous drug use, HIV infection, diabetes mellitus, chronic renal failure, hepatic cirrhosis, alcoholism, underlying malignancy, and morbid obesity. Additionally, any immunosuppressive therapy can promote spinal canal infections.

Thoracic and lumbar levels are the most common sites. Usually, three to five vertebral levels are involved posterior to the thecal sac. SEAs are more common in elderly patients with multiple comorbidities and are relatively unusual in the pediatric population.

## Clinical Presentations

Clinical presentations of SEA are many and varied. Early findings may be subtle and are often misleading because of the patient's other problems. Symptoms usually develop through four stages, however: (1) spinal pain, (2) radicular pain, (3) muscular weakness, and (4) complete paralysis.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_18

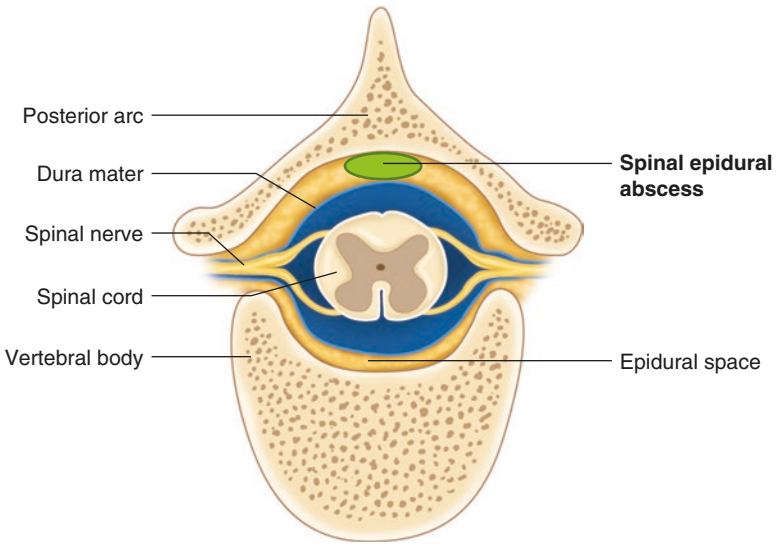


**Fig. 18.1** Localization of spinal epidural abscess (transverse view)

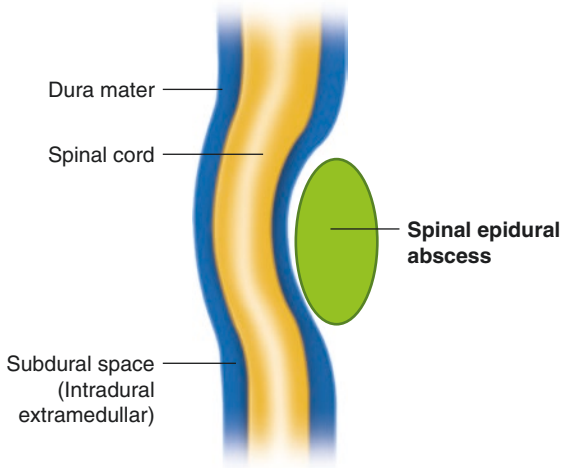


**Fig. 18.2** Localization of spinal epidural abscess (longitudinal view)

Systemic signs of infection (fever, malaise, irritability, night sweats, and headache) are common but inconstant.

The neck or back pains are severe but not always associated with local spinal tenderness. In severe forms (sepsis), fever may be the first presentation. The progression of neurologic deficits may take days or months to become clinically apparent. The complete classic triad of neurologic deficit, spinal pain, and fever is not always present.

Signs and symptoms depend on the site of the lesion, the size of the abscesses, their chronicity, and the patient's general condition. The symptoms are often nonspecific and may be very similar to those of other spinal canal infections. Parameningeal reaction and frank meningitis are rare.

An obvious etiology, including a recent history of infection or a procedure, should be considered.

## Imaging Features

Plain radiography is normal unless there is associated spondylodiscitis. A CT scan may better define the destructive bony changes, and intraspinal gas may be seen.

MRI is the imaging method of choice for confirmation of the suppurative infection and determination of its location in the spinal canal (Figs. 18.3, 18.4, 18.5, and 18.6). Typical findings appear as a hypointense or isointense epidural mass on T1-weighted images and a hyperintense epidural mass on T2-weighted images with gadolinium enhancement. Collections that are fluid and thus easily drained tend to be hyperintense on T2-weighted images and to enhance only peripherally around a central core of hypointensity on T1-weighted images. More solid collections or granulation tissue tend to enhance throughout and to appear isointense or hypointense on T2-weighted images. A fat suppression sequence may help in the diagnosis by subtracting the high signals of epidural fat and bone marrow. Combination of SEA with spondylodiscitis is not uncommon.

The most common differential diagnoses include hematoma, herniated disc, myelitis, spinal subdural abscess, and spinal epidural tumors, especially lymphoma and metastatic neoplasms.

## Laboratory Findings

The pathogens most often responsible for SEA are S*taphylococcus* species (especially *S. aureus* and *S. epidermidis*), followed by *Streptococcus* species (*S. pneumoniae* and *S. viridans*). Other bacteria include *Enterococcus* species, *Propionibacterium acnes*, and gram-negative species (*Escherichia coli*, *Pseudomonas aeruginosa*, *Salmonella* species, and *Klebsiella* species), which are sometimes found

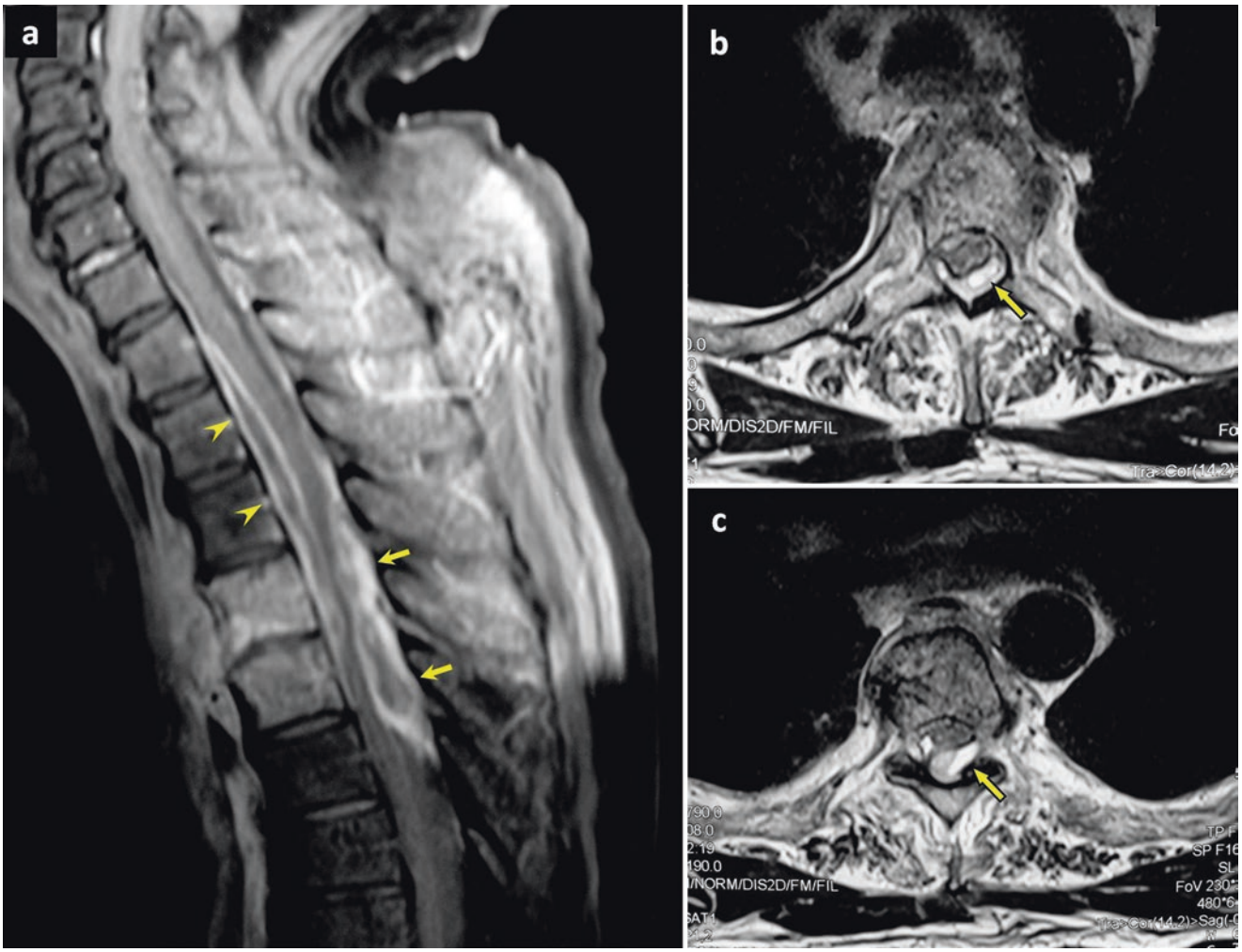


**Fig. 18.3** This 65-year-old man on hemodialysis for end-stage renal disease secondary to diabetic nephropathy was admitted with paraplegia, severe hyperglycemia, and diabetic ketoacidosis. Spinal sagittal T1-weighted MR image with gadolinium injection (**a**) and axial T2-weighted images (**b**, **c**) show a thoracic epidural abscess extending from T2 to T7 with spinal cord compression. This abscess has two components: anterior (*arrowheads*) and posterior (*arrows*)

after urinary or digestive infections. The presence of anaerobes is unusual, but multiple types of bacteria are common. *Mycobacterium tuberculosis* is relatively frequent in Asia and Africa, and tuberculous infections are being seen more regularly in developed countries as well (*see* Chap. 24). Fungal infections (candidiasis, aspergillosis, coccidioidomycosis, and blastomycosis) are uncommon. Sterile cultures of abscess material are seen in 30–50% of patients.

The usual biologic markers of acute inflammation (white blood cell count, C-reactive protein level, and erythrocyte sedimentation rate) are useful, but they are not sensitive indicators, especially in patients with chronic forms of suppuration.

Urine, sputum, and blood cultures to screen for other potential sources of infection are important. Lumbar puncture (spinal tap) is not usually performed because it does not contribute to the diagnosis and may even be hazardous, presenting a danger of introducing infected material into the subarachnoid space or causing sudden neurologic deterioration. Accompanying bacterial meningitis is very rare.

Needle biopsies may be performed under imaging guidance to provide microbiological identification of infection from disc spaces, vertebral bodies, or paraspinal fluid collections.

## Treatment Options

Classically, treatment of SEA requires surgical drainage of the abscess cavity followed by aggressive antibiotic therapy.

The surgical procedure includes decompressive laminectomy at the appropriate levels, surgical drainage of the abscess cavity, and debridement of granulation tissue. Occasionally, less invasive methods are used, like hemilaminectomy, interlaminar fenestration, or CT-guided

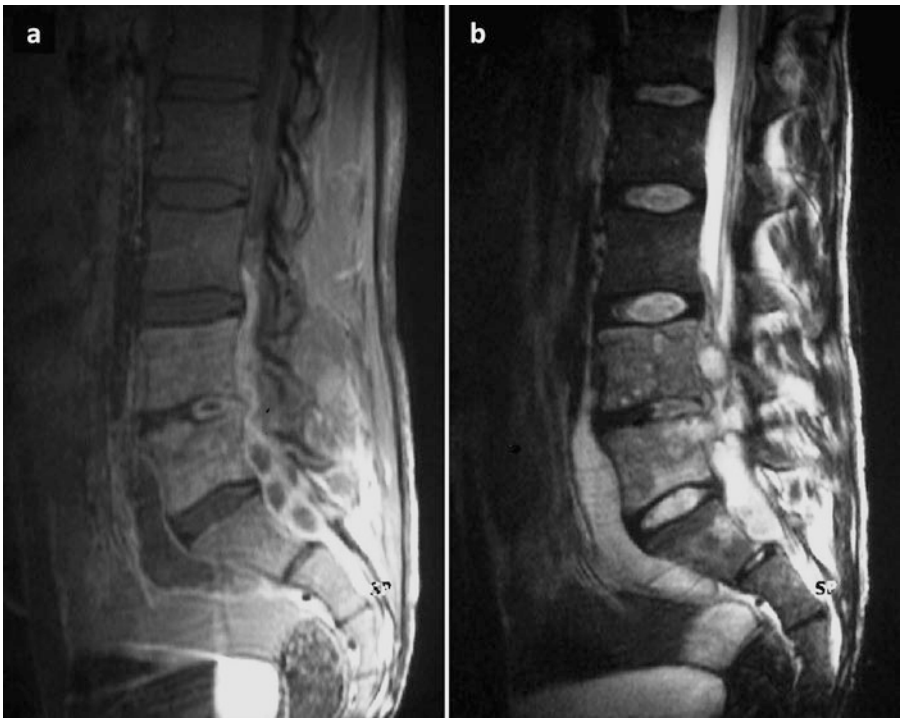


**Fig. 18.4** Case 18.1. Lumbosacral spinal sagittal post-gadolinium T1-weighted MRI (**a**) and T2-weighted MRI (**b**) showing a multiloculated, tuberculous epidural abscess extending from L3 to S1 with cauda equina compression. Note the associated L4–L5 spondylodiscitis

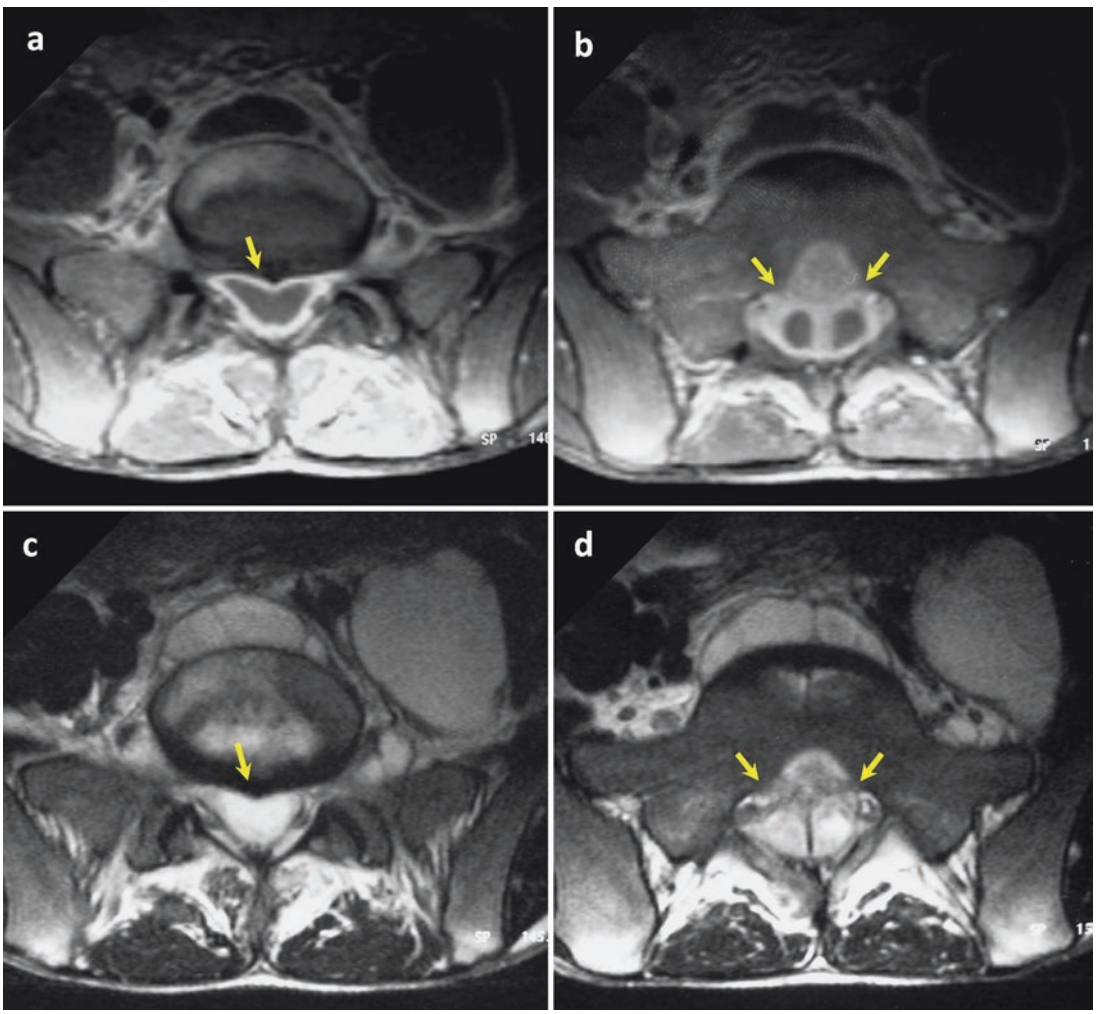


**Fig. 18.5** Case 18.1. Lumbosacral spinal axial post-gadolinium T1-weighted MRI (**a**, **b**) and T2-weighted MRI (**c**, **d**) showing the multiloculated, intracanalar spinal epidural abscess (*arrows*) with spondylodiscitis and paraspinal extension (Pott's disease)

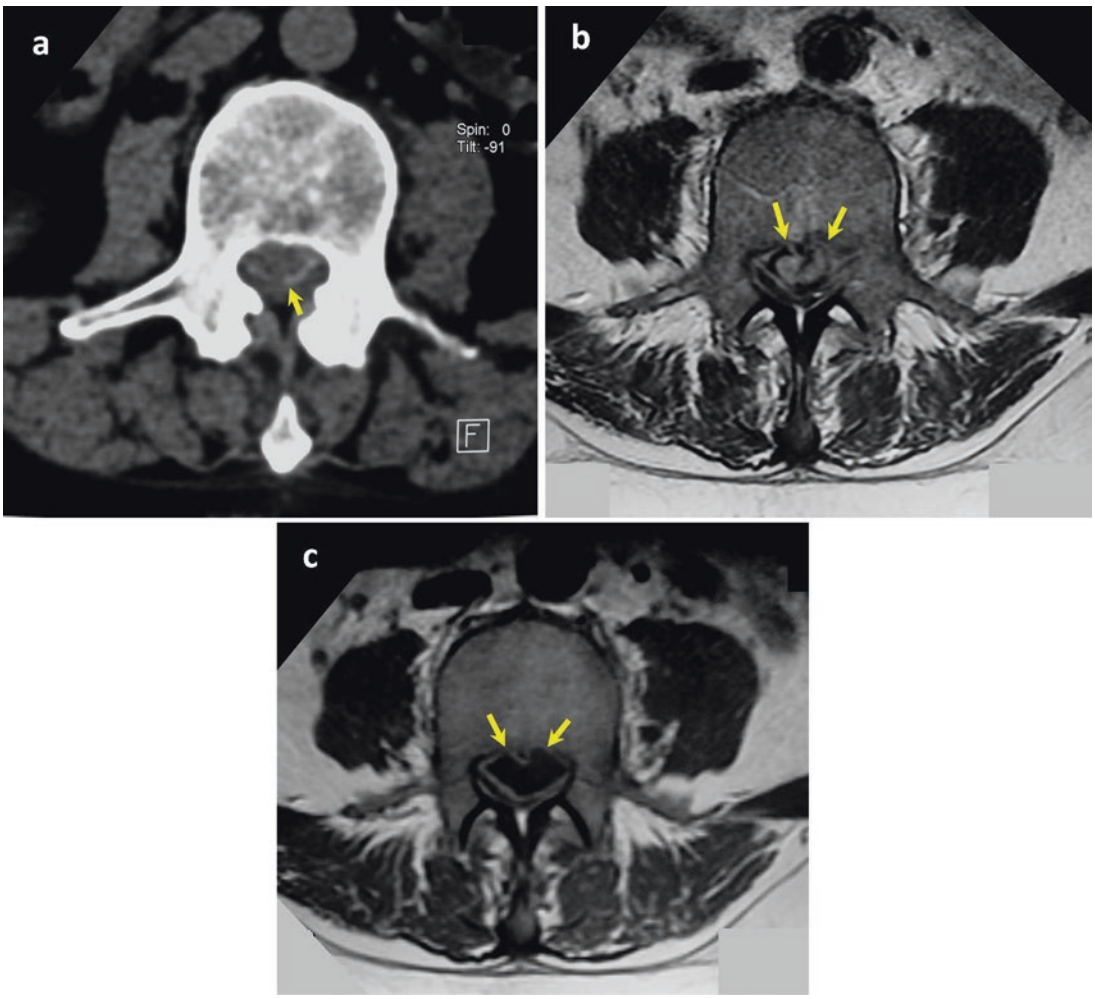


**Fig. 18.6** This 46-year-old man presented with paraparesis and a 2-month history of progressive low back pain radiating to both legs. An axial lumbar spinal CT scan (**a**), axial T2-weighted MRI (**b**), and T1-weighted MRI following gadolinium administration (**c**) reveal ring enhancement of an anterior epidural abscess at the L1 vertebral level (*arrows*) without adjacent bony lesions

needle aspiration. In patients with concomitant spondylodiscitis and significant vertebral body destruction, wide laminectomy should be used, taking care not to destabilize the spine. If potential spinal instability requires reconstruction of the spine with spinal implants, the procedure does not have contraindications regarding infection.

Medical therapy alone with immobilization (conservative management) may be appropriate for patients in the early stage of abscess formation, those with poor general condition, or those without neurologic deficits. If the diagnosis is unclear or the patient does not respond to conservative management, surgical decompression should be performed quickly.

Intravenous antibiotic therapy should be initiated as soon as possible, based on the presumed etiology of infection, and then should be tailored according to culture results and antibiotic susceptibility testing. If the organisms and source of infection are unknown (most likely *S. aureus*), empiric antibiotics (mainly third-generation cephalosporin and vancomycin) should be used.

Immunocompetent patients with little or no spondylodiscitis need a minimum of 6 weeks of antibiotic therapy. A medical cure of concomitant vertebral osteomyelitis requires a longer duration of antibiotic administration, with 6 weeks of immobilization. Tubercular and fungal abscesses must be treated with the appropriate anti-infectious regimens.

Corticosteroids may be used for a few days as an adjuvant therapy in patients with extensive secondary spinal cord edema.

## Outcomes

Response to therapy should be assessed on the basis of clinical and neurologic assessments, with biologic and MRI monitoring to document the resolution of the epidural infection.

The best predictor of outcome is the preoperative clinical and neurologic condition, the delay in diagnosis and treatment, and the response to therapy. Although neurologic

deficits may decrease after decompression and drainage of the abscess collection, multiple comorbidities and a complete motor deficit predispose to a poorer outcome.

Mortality is often related to a missed diagnosis, coexisting comorbidities, or concomitant infections, or it may be a complication of residual paraplegia or tetraplegia.

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# Spinal Subdural Abscesses

# 19

Spinal subdural abscess (or empyema) is a focal suppurative collection within the subdural space between the dura and the arachnoid layer (Figs. 19.1 and 19.2). This rare infection of the central nervous system has high neurologic morbidity and even mortality and may be seen in both children and adults. Clinically, the triad of fever, back or radicular pain, and neurologic deficits are suggestive of the disease, though chronic forms tend to have less-specific symptoms that mimic those of spinal cord tumors. MRI with gadolinium injection is the investigation of choice. *Staphylococcus aureus*, *Streptococcus* species, and *Mycobacterium tuberculosis* are the microorganisms most likely to be found. The classic treatment of spinal subdural abscesses is surgical drainage of the suppurative collection followed by the administration of adequate antibiotic drugs. Conservative management is not appropriate. Good clinical recovery is correlated with prompt diagnosis, rapid surgical decompression and drainage, and appropriate antimicrobial therapy. Common complications include paraplegia, sphincter dysfunctions, recurrence, meningitis, sepsis, and even death.

## Epidemiology and Etiology

Most spinal canal infections are extradural; less common are infections within the spinal cord, but infection can also be intradural extramedullary. Abscesses contained in the intradural extramedullary space are also called *spinal subdural abscess* or *spinal subdural empyema*.

Spinal subdural abscesses and empyemas are not well distinguished in the literature. Abscess has a capsule that separates pus from normal structures, whereas empyema is an accumulation of pus in a preexisting cavity. Spinal subdural abscess is a very rare and unpredictable form of central nervous system infection that has potential for permanent neurologic damage and even sepsis and death.

Like other kinds of spinal canal infections, the common etiologies related to the development of spinal subdural suppurations are hematogenous spread from a distant site of infection (urogenital tract, pneumonia, endocarditis, and otitis), contiguous extension (spondylodiscitis, congenital dermal sinuses, and intramedullary tumors), and direct inoculation (epidural anesthesia, intraspinal catheter, lumbar puncture, or discography), but some cases remain cryptogenic.

Predisposing factors worth consideration include alcoholism, diabetes mellitus, immunosuppressive drugs, malignancy, chronic renal failure, intravenous drug abuse, rheumatic heart valve disease, and tuberculosis.

The thoracolumbar spine is the most affected area. Concomitant spondylodiscitis is rare. Involvement of multiple levels is relatively common because of easy dissemination of the infection in the subdural space. Spinal subdural abscesses may occur at any age.

## Clinical Presentations

Clinical features of spinal subdural abscess are similar to those of spinal cord abscess. Acute forms present as acute spinal cord syndrome, mimicking acute transverse myelitis. Fever and back and/or root pain are typical presenting symptoms, in addition to progressive neurologic deficit (paraparesis/tetraparesis, sensory loss, or sphincter disturbances) below the level of the lesion. Initially, there is no spinal tenderness (unlike with spinal epidural abscess). Meningismus is a common symptom.

Chronic forms tend to have less-specific symptoms, mimicking spinal cord tumors, with a slow neurologic deficit and progressive back pain, but without fever. The signs and symptoms depend on the site of the lesion, the size and number of the abscesses, and their chronicity.

Obvious etiology, including an anatomical defect (reflecting congenital anomalies) or a recent history of infection or an invasive procedure should always be considered.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_19

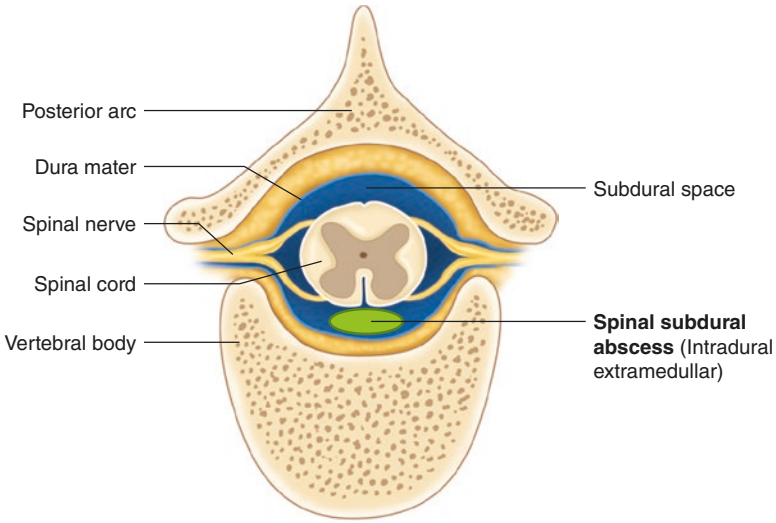


**Fig. 19.1** Localization of spinal subdural abscess (transverse view)

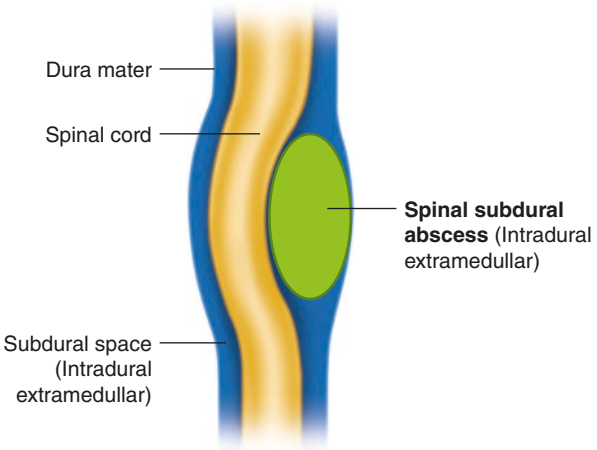


**Fig. 19.2** Localization of spinal subdural abscess (longitudinal view)

## Imaging Features

CT scans may show bubbles of air in the intradural space and rarely the appearance of spinal osteomyelitis or discitis, but MRI is the best imaging modality for exploration of spinal canal infections. MRI features of spinal subdural abscess resemble those of intracranial subdural empyemas (*see* Chap. 6). The spinal subdural abscess appears poorly delimited. On T1-weighted images, the intradural collection is isointense. On T2-weighted images, the contents of the lesion appear homogenous and hyperintense, but its capsular margins are hypointense. Gadolinium-enhanced T1-weighted images define the extent of the lesion by enhancing the margins (rim enhancement), as is typical of an abscess (Figs. 19.3, 19.4, 19.5, 19.6, 19.7, and 19.8).

Differential diagnoses include intradural extramedullary tumor (neuroma and meningioma), hematoma, inflammatory pseudotumor, pachymeningitis/arachnoiditis, and epidural abscess.

## Laboratory Findings

The microorganism most often responsible for spinal subdural abscess is *Staphylococcus aureus*, followed by *Streptococcus* species. Other bacteria include *Escherichia coli*, *Pseudomonas aeruginosa*, *Mycobacterium tuberculosis*, *Peptococcus magnus*, and *Fusobacterium* species. The presence of anaerobes is unusual, but multiple types of bacteria are common. Sterile culture with no causative pathogens may be seen.

Hyperleukocytosis and elevated inflammatory markers (C-reactive protein level and erythrocyte sedimentation rate) are common, but they are neither sensitive nor specific indicators.

Cerebrospinal fluid may be purulent, with very elevated leukocytosis counts, decreased glucose, increased protein, and a positive Gram stain.

## Treatment Options

Typically, treatment of spinal subdural abscesses is surgical drainage of the suppurative collection followed by administration of adequate antibiotics (initially using vancomycin and a third-generation cephalosporin). Generally, conservative management (antibiotic therapy) alone is not appropriate, however.

Surgical procedure includes decompressive laminectomy at the appropriate levels and surgical drainage of the abscess cavity. Ultrasound may be used as an intraoperative guide. If possible, the arachnoid must be preserved. Pus should be tested in aerobic and anaerobic cultures for antibiotic susceptibility and for *Mycobacterium tuberculosis*.

Appropriate intravenous antibiotics are used for 4–6 weeks, followed by an additional 2–3 months of oral antibiotic drugs.

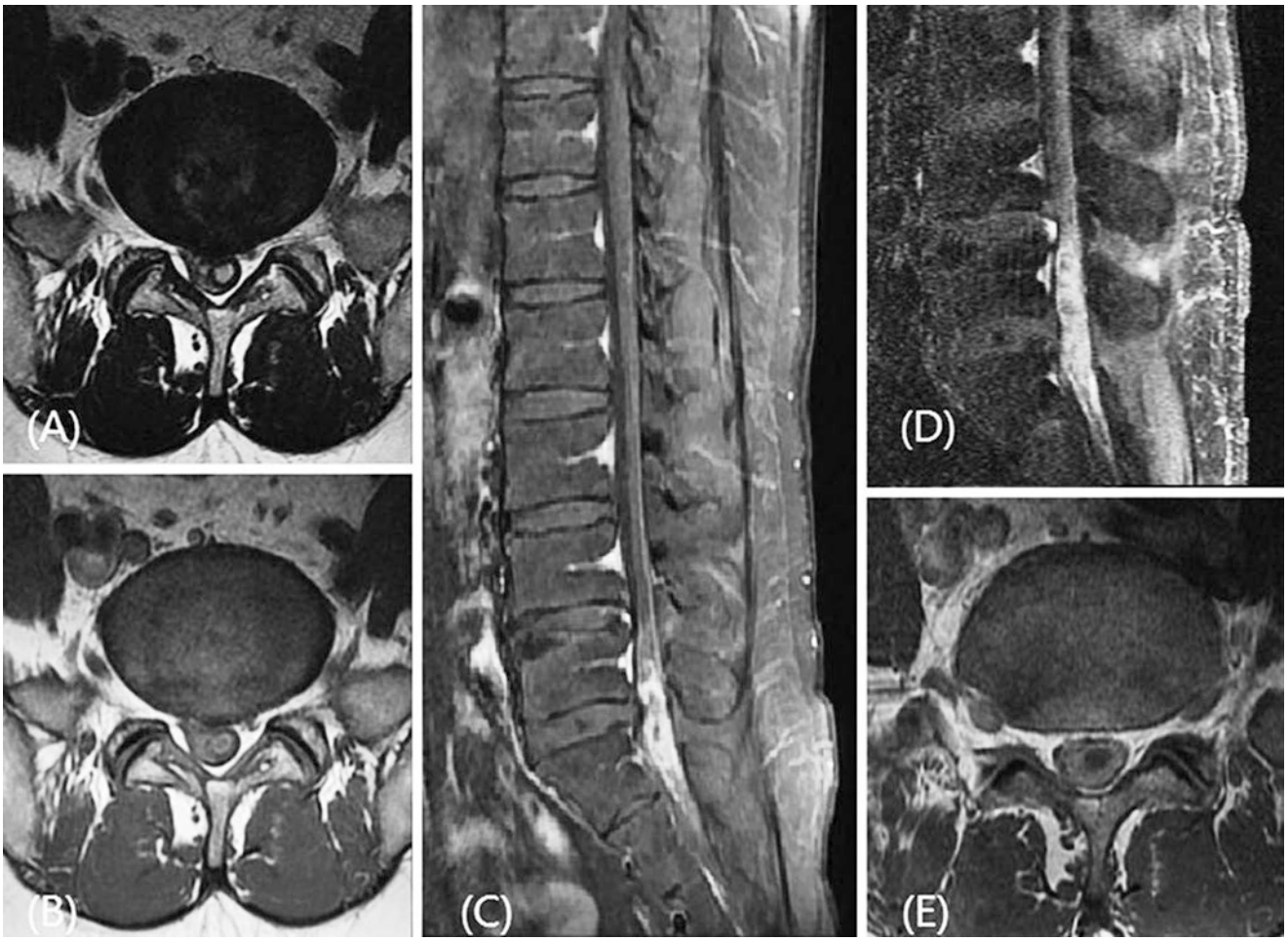


**Fig. 19.3** Case 19.1. Initial T2-weighted MR image (**a**); T1-weighted MR image (**b**); fat-suppressed, contrast-enhanced T1-weighted MR image (**c**). Contrast-enhanced MR images 1 week later (**d**, **e**) (From Lim et al. (2013), with permission)

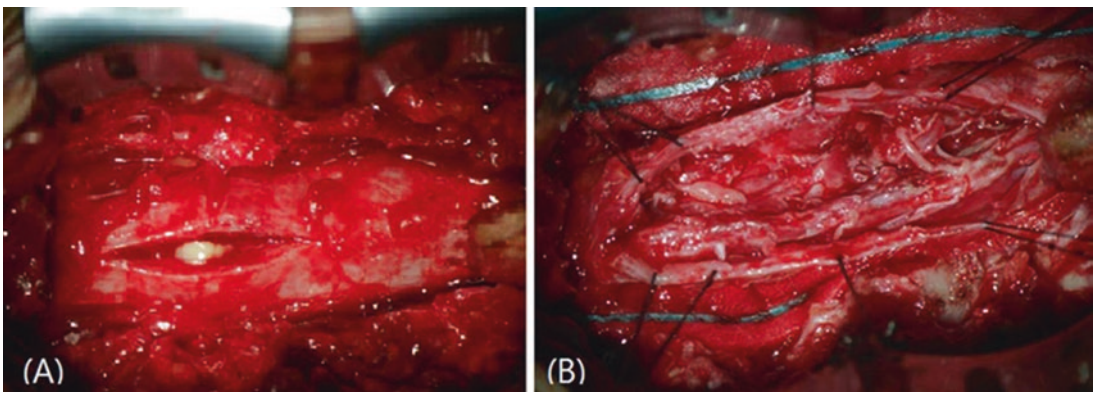


**Fig. 19.4** Case 19.1. In operation, after durotomy, pus was revealed within the dura (**a**). After abscess removal, granulation tissue and adhesion were found (**b**) (From Lim et al. (2013), with permission)

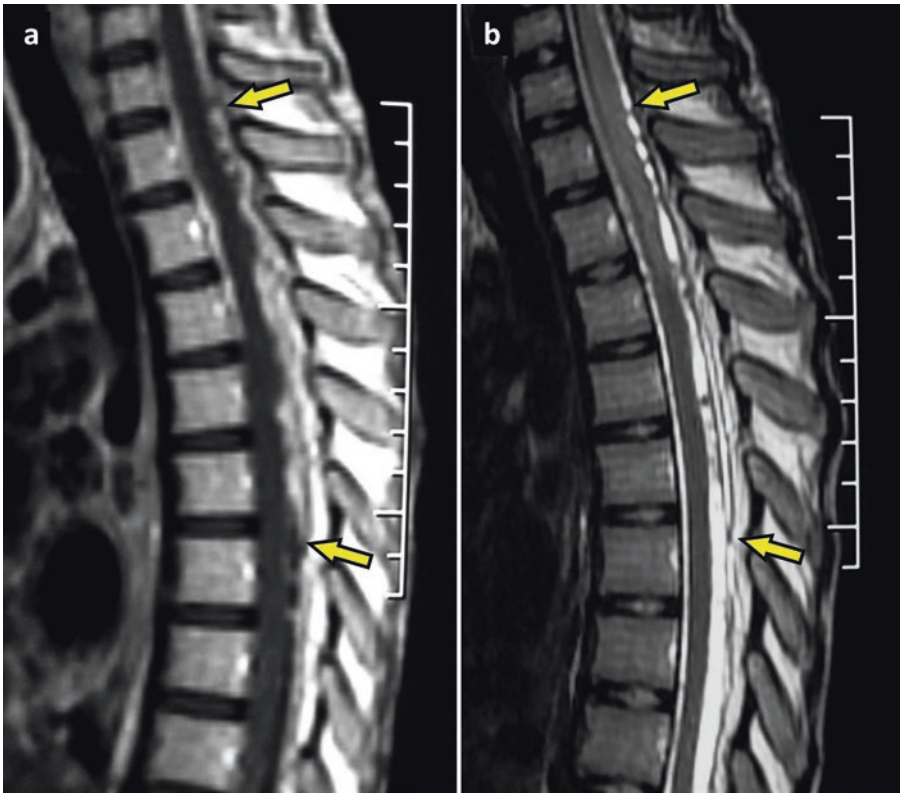


**Fig. 19.5** Sagittal T1-weighted MRI after gadolinium enhancement (**a**) and T2-weighted image (**b**) of the thoracic spine in an adolescent with tuberculous meningitis, showing extensive posterior subdural granulomatous lesions from T2 to T6 (*arrows*) with spinal cord compression

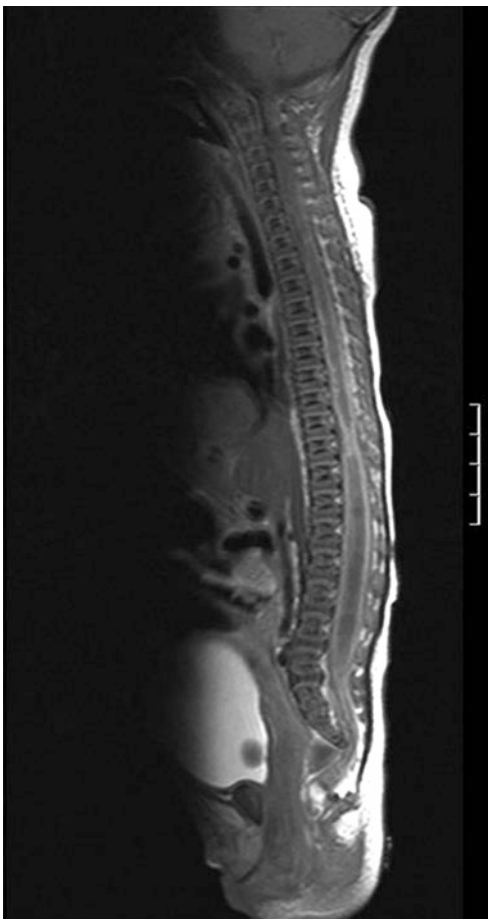

**Fig. 19.6** T1-weighted MRI demonstrating a holocord subdural abscess in a 4-week-old child with the Currarino triad (From Sandler et al. (2013), with permission)

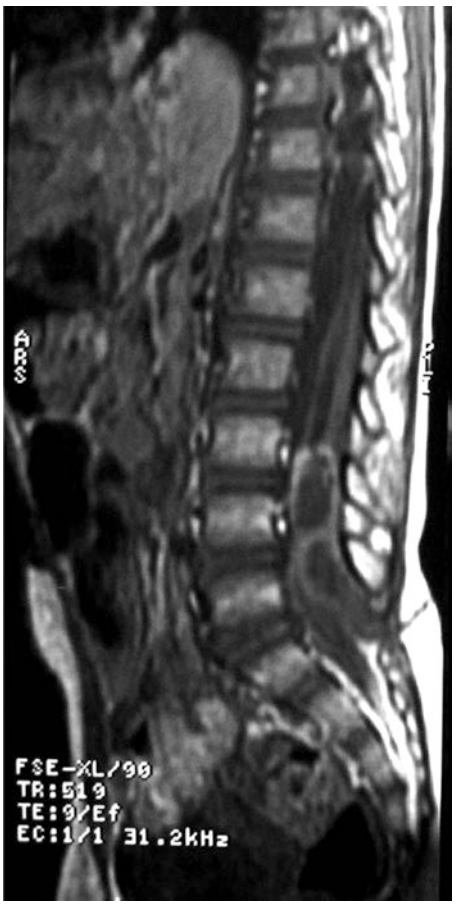


**Fig. 19.7** T1-weighted contrast-enhanced MRI of the lumbar spine in a child with a dermal sinus tract that traversed intradurally and eventually led to the formation of two ring-enhancing abscesses (From Sandler et al. (2013), with permission)

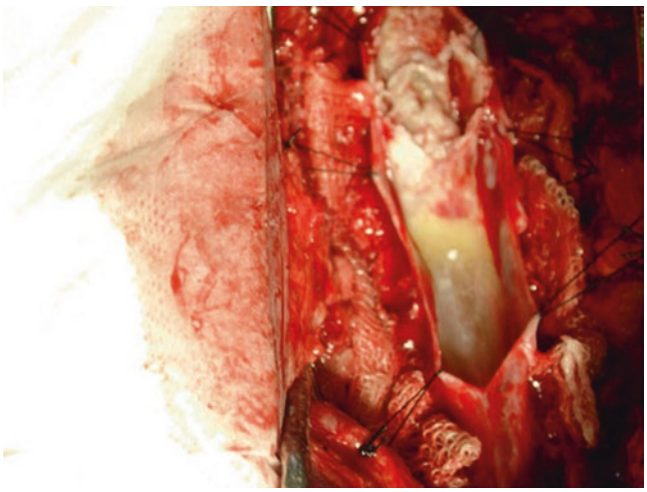

**Fig. 19.8** Intraoperative photograph of a child with tuberculosis, demonstrating an encapsulated intradural infection admixed with a more caseous material superiorly (From Sandler et al. (2013), with permission)

Tubercular and fungal abscesses must be treated with the appropriate anti-infectious regimens.

The use of corticosteroids may be suggested as an adjuvant therapy for patients with extensive secondary spinal cord edema and to prevent occlusive thrombophlebitis.

## Outcomes

Response to therapy should be evaluated on the basis of clinical and neurologic assessments, biological monitoring, and MRI studies to document the resolution of the spinal canal suppurative collection.

The neurologic outcome depends primarily on the preoperative neurologic condition of the patient, the delay in diagnosis and institution of therapy, and the response to treatment. Good clinical recovery is predictable for many patients (with motor function recovered more readily than sphincter function), and the mortality rate is low. Common complications include paraplegia, sphincter dysfunctions, recurrence, meningitis, sepsis, and even death.

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# Spinal Cord Abscesses

# 20

Spinal cord abscess is a focal suppurative collection within the spinal cord parenchyma (Figs. 20.1 and 20.2). It is a rare infection of the central nervous system with potential permanent neurologic damage. Spinal cord abscesses are more common in children, particularly those with congenital midline defects. Clinically, acute forms present as acute transverse myelitis, but chronic abscesses tend to have less-specific symptoms that mimic spinal cord tumors. MRI with gadolinium injection is the imaging technique of choice. The microorganisms most often responsible for spinal cord abscess are *Staphylococcus aureus* and *Streptococcus* species. Anaerobic and fungal infections are rare. Classically, treatment of intramedullary abscesses involves laminectomy, myelotomy, and surgical drainage of the abscess cavity, followed by the administration of appropriate antibiotics, although medical therapy alone may be appropriate in some cases. Good clinical recovery is predictable for many patients and the mortality rate is low, but the prognosis of spinal cord abscess depends on the patient's general health, the delay in diagnosis and treatment, and the response to therapy. Common complications include paraplegia, sphincter dysfunctions, recurrence, meningitis, spinal cord infarction, sepsis, and even death.

## Epidemiology and Etiology

Spinal cord abscess, also known as *intramedullary abscess*, is a focal purulent collection within the spinal cord parenchyma. This rare infection of the central nervous system can cause permanent damage of the spinal cord structures.

This infection can develop through hematogenous spread (from the urogenital tract, pneumonia, endocarditis, or otitis), by contiguous extension (spondylodiscitis, congenital dermal sinuses, or intramedullary tumors), and by direct inoculation (epidural anesthesia, lumbar puncture, or penetrating wound). Some cases remain cryptogenic.

Clinical, neuroimaging, and laboratory features depend on the stages of the formation of the spinal intramedullary abscess: The bacterial nidus is first infiltrated by polymorphonuclear leukocytes, and suppurative myelitis results (early and late stages). This myelitis is followed by the development of a necrotic center with liquefaction and a peripheral capsule.

The lower thoracic and lumbar areas are the sites of predilection (Fig. 20.3). The lesion can extend across one to six spinal segments or even more. Spinal cord abscesses are more common in male children (owing to congenital midline defects) and are rarely seen in adults.

## Clinical Presentations

Acute forms present as acute spinal cord syndrome, mimicking an acute transverse myelitis. Generally, symptoms include pain, motor and sensory deficits, urinary incontinence, fever, meningismus, and sometimes brainstem dysfunction. Chronic abscesses tend to have less-specific symptoms that may mimic spinal cord tumors, including a slowly developing neurologic deficit and gradually progressive back pain without fever. The triad of neurologic deficit, spinal pain, and fever is seen in less than one third of patients.

Signs and symptoms depend on the site of the lesion, the size and number of abscesses, and their chronicity. The symptoms of spinal cord abscess are highly similar to those of a spinal subdural abscess.

Obvious etiology, including an anatomical defect or a recent history of infection or an invasive procedure, should always be considered.

## Imaging Features

Plain radiography and CT scans are usually normal.

MRI is the imaging modality of choice for exploration of intramedullar suppurative collections. MRI features seem to be similar to the changes seen with brain abscesses



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_20

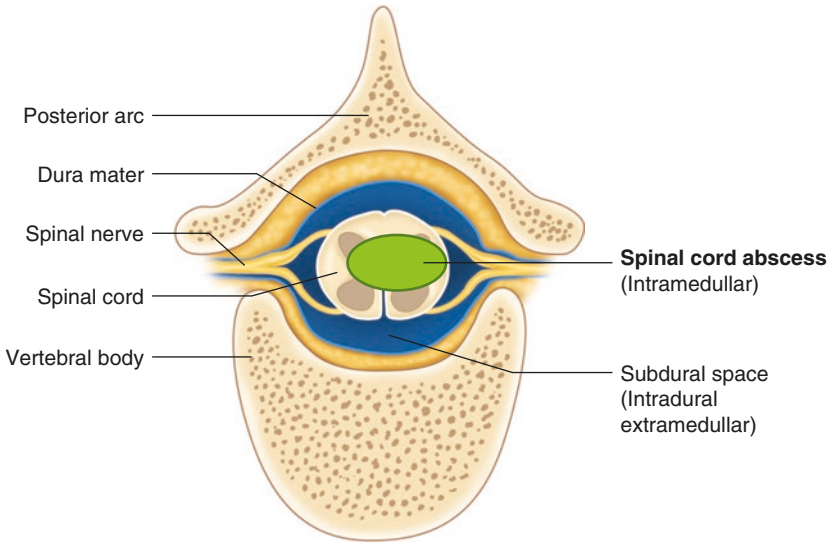


**Fig. 20.1** Localization of spinal cord abscess (*transverse view*)

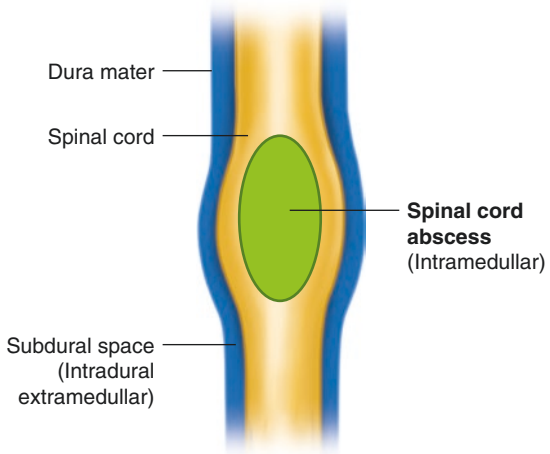


**Fig. 20.2** Localization of spinal cord abscess (*longitudinal view*)

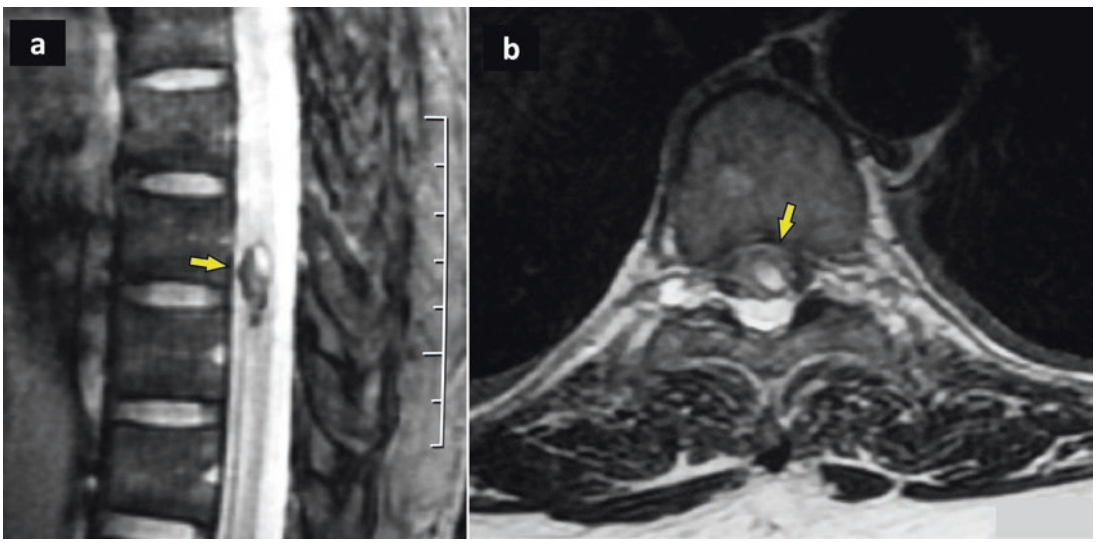


**Fig. 20.3** Sagittal (**a**) and axial (**b**) T2-weighted MRI in a diabetic patient who presented with a progressive, subfebrile paraplegia. An intramedullar granulomatous abscess is situated between the T9 and T10 vertebrae (*arrow*)

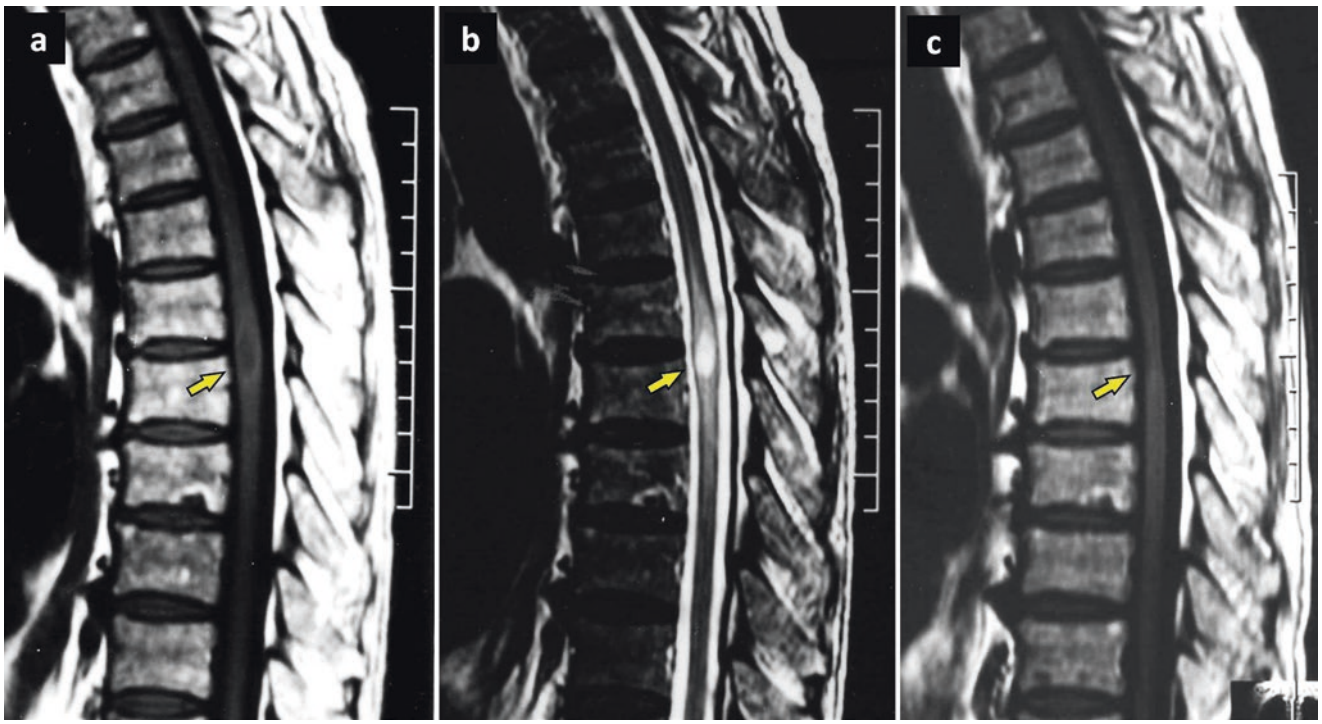


**Fig. 20.4** Sagittal T1-weighted MRI after gadolinium administration (**a**) and on T2-weighted sequence (**b**). This midthoracic, intraparenchymal spinal cord ringlike lesion (*arrow*) proved to be an abscess. The patient was treated with antibiotic therapy and corticosteroids with a good recovery. The complete resolution of the abscess is seen on post-contrast sagittal T1-weighted images performed 12 weeks later (**c**), showing moderate spinal atrophy without gadolinium enhancement (*arrow*)

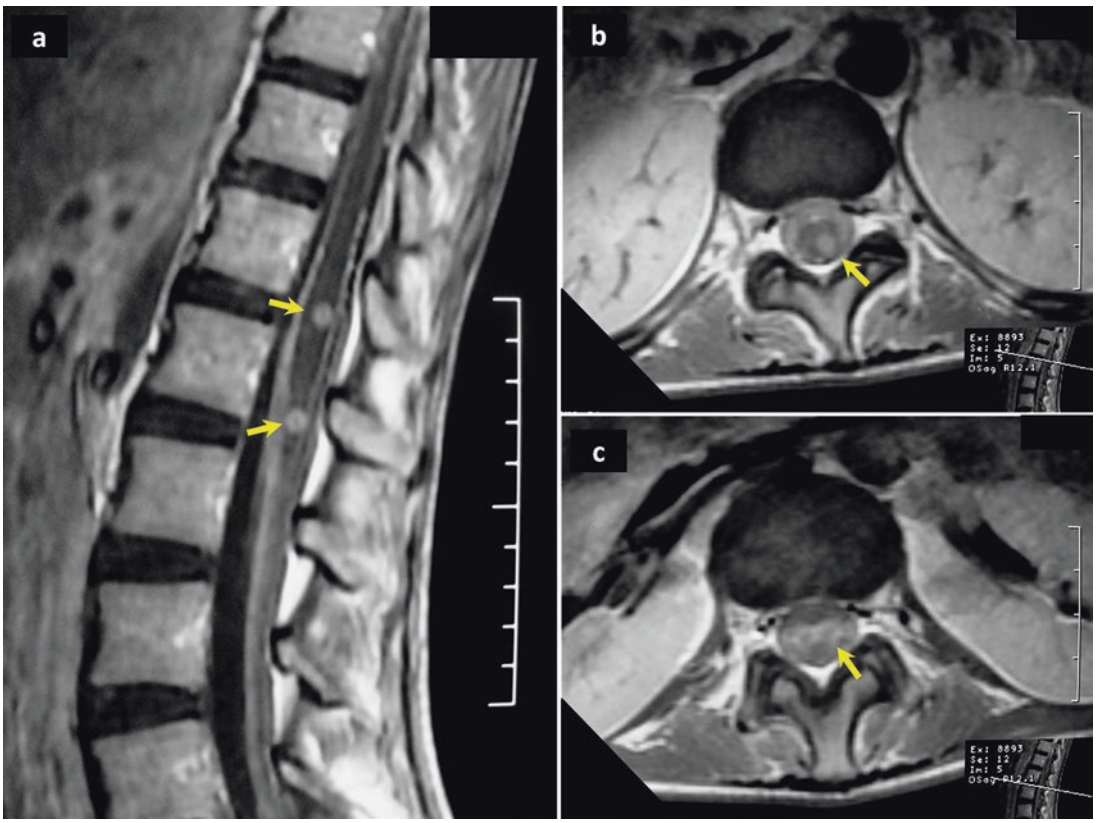


**Fig. 20.5** Sagittal (**a**) and axial (**b**, **c**) T1-weighted MRI after gadolinium showing two tuberculomas in the conus medullaris (*arrows*) in a 20-year-old woman treated for a tuberculous meningitis

(*see* Chap. 8). The spinal cord abscess appears hypointense on T1-weighted images and hyperintense on T2-weighted images. A poorly defined rim enhancement is seen on T1-weighted images following gadolinium administration (Figs. 20.4, 20.5, and 20.6). Follow-up MRI may reveal a well-defined enhancing margin with intramedullar central low signal intensity.

The purulent fluid shows high signal intensity on diffusion-weighted MR imaging (DWI) and low apparent diffusion coefficient (ADC) values that reflect decreased diffusion properties.

Screening for a congenital spinal defect (sinus tract, spina bifida, and/or a neural tube defect) is mandatory, especially in children.

The main differential diagnosis includes spinal cord ischemia, hematoma, myelitis, granuloma, multiple sclerosis, hemangioblastoma, and other primary or secondary intramedullary tumors (glioma or metastases).

## Laboratory Findings

The microorganisms most commonly responsible for spinal cord abscess are *Staphylococcus aureus* and *Streptococcus* species. Other bacteria include *Haemophilus influenzae*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Escherichia coli*, and *Mycobacterium tuberculosis*. The presence of anaerobes is unusual, but multiple types of bacteria are common, particularly in patients with a congenital sinus tract. Fungal infections (*Candida*, *Nocardia*, *Cryptococcus*, or *Aspergillus* species) are rare. More than 30% of cases have sterile cultures in which no causative pathogens are seen.

Hyperleukocytosis and elevated inflammatory markers (C-reactive protein level and erythrocyte sedimentation rate) are common, but they are not sensitive indicators, especially in patients with chronic abscesses.

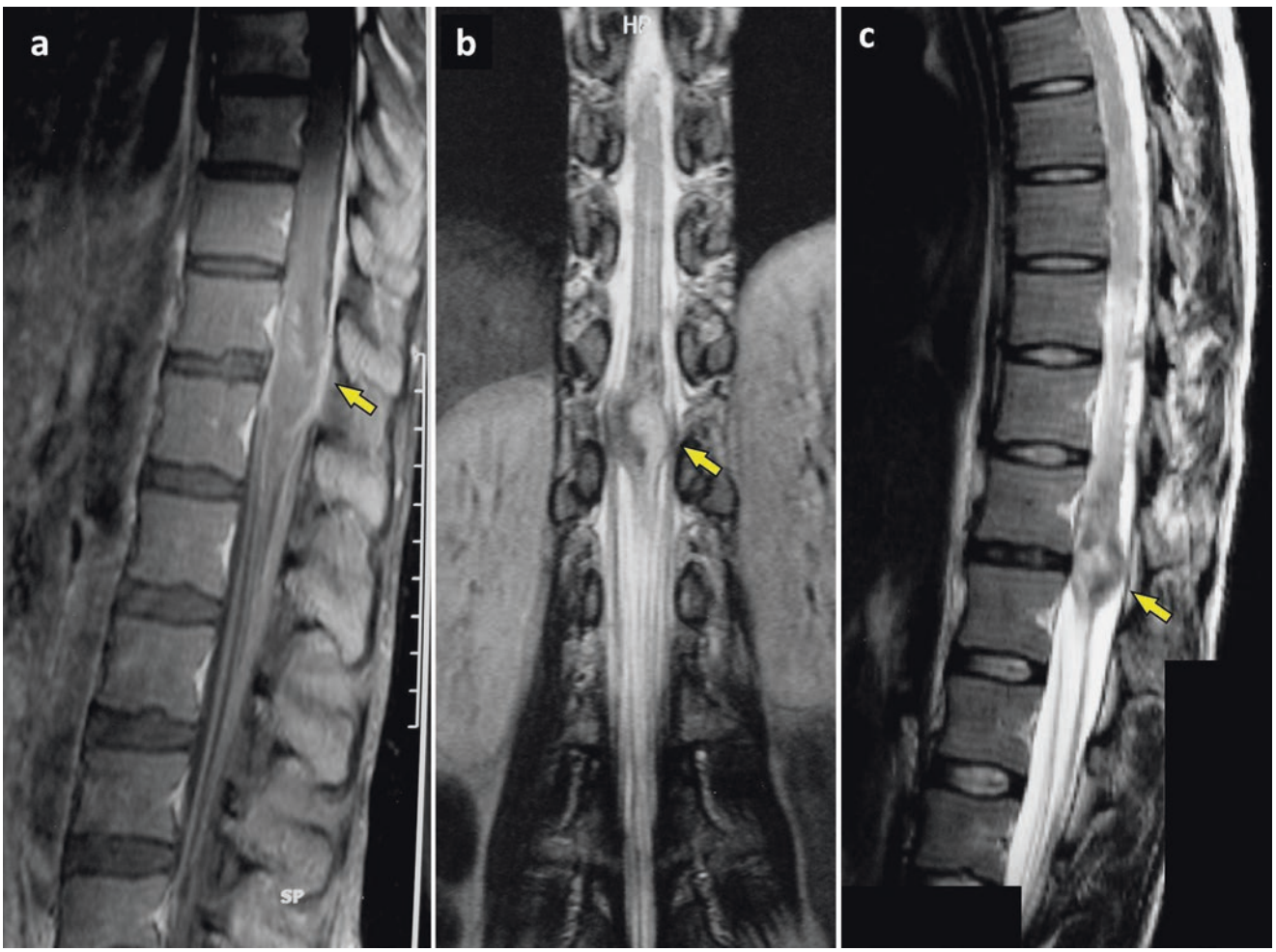


**Fig. 20.6** Early stage of infectious myelitis. Sagittal post-gadolinium T1-weighted MRI (**a**) and coronal (**b**) and sagittal (**c**) T2-weighted images. There is a high signal on the T2-weighted images (**b**, **c**), with poorly defined enhancement on the T1-weighted image (**a**) (*arrow*)

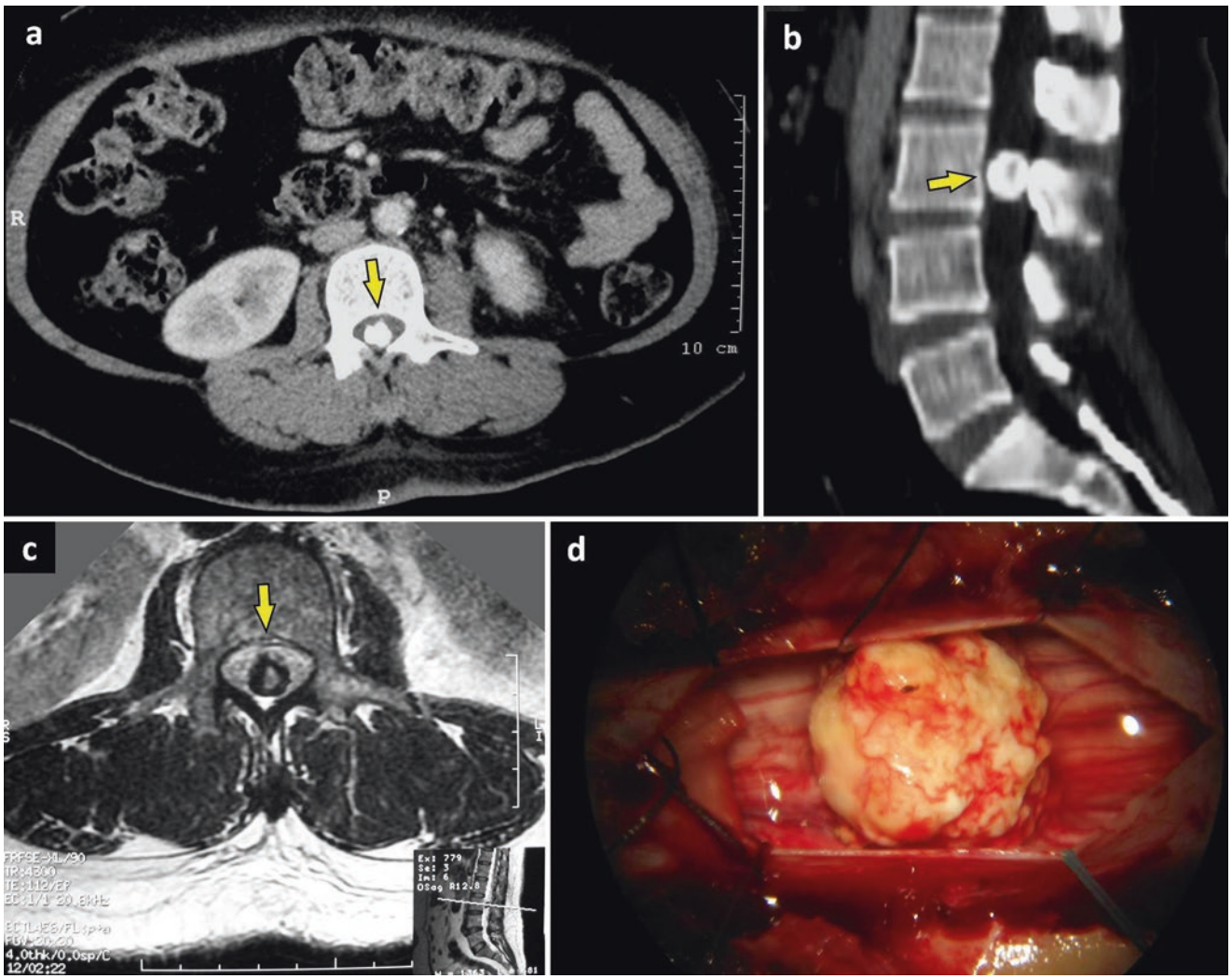


**Fig. 20.7** Axial abdominal CT scan with contrast injection (**a**), sagittal lumbar spinal CT scan on bone window (**b**), and axial T2-weighted MRI (**c**), revealing a granulomatous, calcified tuberculoma of the filum terminalis (*arrow*). Operative view of the tuberculoma under microscopic magnification (**d**)

Cerebrospinal fluid is often abnormal, with elevated leukocytosis counts, decreased glucose, increased protein, and a positive Gram stain.

## Treatment Options

The classic treatment of spinal cord abscesses involves surgical drainage of the abscess cavity and administration of appropriate antibiotics, although medical therapy alone (conservative management) may be appropriate in some cases, such as patients in the early stage of abscess formation (presuppurative myelitis), patients with a small abscess, or those without congenital neuroectodermal abnormalities. If the diagnosis is unclear, however, or if the patient does not respond to adequate conservative management, surgical decompression should be performed.

The surgical procedure includes decompressive laminectomy at the appropriate levels, midline myelotomy, and surgical drainage of the abscess cavity. Ultrasound may be used as an intraoperative guide. Surgical treatment should be adopted for patients with congenital neuroectodermal abnormalities.

A minimum of 4–6 weeks of intravenous antibiotic therapy is recommended, followed by an additional 2–3 months of oral antibiotic drugs.

Tubercular and fungal abscesses must be treated with the appropriate anti-infectious regimens (Fig. 20.7). Corticosteroids may be used as an adjuvant therapy in patients with extensive secondary spinal cord edema.

## Outcomes

Response to therapy should be evaluated on the basis of clinical and neurologic assessments, biological monitoring, and MRI studies to document the resolution of the spinal cord suppurative collection.

The neurologic outcome depends primarily on the preoperative condition of the patient, the delay in diagnosis and treatment, and the response to therapy. Good clinical recovery is predictable for many patients (with motor function recovered more readily than sphincter function), and the mortality rate is low. Common complications include paraplegia, sphincter dysfunctions, recurrence, meningitis, spinal cord infarction, sepsis, and even death.

Mortality is often related to a missed diagnosis, concomitant comorbidities, further infections in the body, or it may be a complication (e.g., decubitus) of residual paraplegia or tetraplegia.

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# Part IV

# Infections Following Cranial and Spinal Surgery

# Surgical Site Infections in Cranial Surgery

# 21

Surgical site infections (SSIs) following cranial surgery remain a serious clinical problem and major causes of morbidity and mortality. Classically, SSIs are divided into extradural and intradural infections. Risk factors can be categorized as patient related or procedure related. Clinical symptoms vary greatly depending on many features, with the most common being fever, local wound inflammation, purulent drainage, headache, mental status change, seizure, and focal neurologic deficits. CT scanning and MRI offer an interesting basis for the diagnosis of cranial and intracranial infections. Inflammatory biomarkers are highly variable in their expression in the postoperative period, with the most useful being C-reactive protein (CRP) and procalcitonin levels. When possible, CSF should be evaluated in patients with suspected intradural infections. The most common causative pathogens are quite similar, dominated by gram-positive skin flora, especially *Staphylococcus aureus*. Patients with limited infections may be managed with local measures and antimicrobial therapy alone, but these selected cases require careful monitoring and should be considered for surgery if complications arise. Abscess, empyema, and large cranial osteomyelitis require reoperation with cleaning, evacuation of fluids, debridement, removal of foreign material, drainage, and careful wound closure. Strict awareness of hygiene, with careful application of protocols, decreases SSIs. If treated rapidly and vigorously, SSIs may resolve without sequelae, but many neurologic complications may occur, especially in patients with intradural infections.

## Epidemiology and Etiology

Despite improvements in surgical practice and infection control techniques, SSIs remain an important clinical problem and a major cause of morbidity and mortality. Additionally, postoperative infections represent an important contributor to the cost of care and a truly frustrating problem for surgeons, who may be faced with patient dissatisfaction and litigation.

The incidence of SSIs in cranial surgery is reported to be less than 17% (average of 5%), depending on the type of surgical procedure, the length of postoperative follow-up, and the quality of data collection. Generally, the depth of SSI is classified as subgaleal, cranial bone, epidural, subdural, and/or intracerebral. A given layer is considered infected if there is gross purulent material in that layer intraoperatively or if cultures taken from that layer are positive.

Classically, the surgical site is divided into *extradural* (or incisional) and *intradural* (or organ-related) parts. Extradural infections include the scalp, the cranial bone, or the epidural space (Figs. 21.1, 21.2, 21.3, 21.4, 21.5, 21.6, 21.7, 21.8,

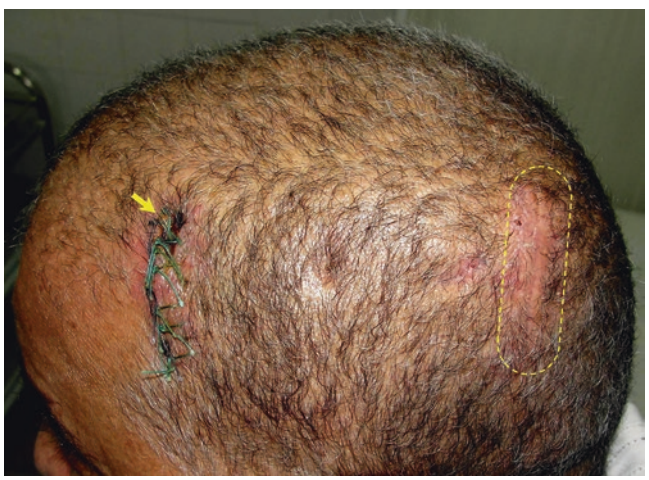

**Fig. 21.1** This diabetic patient was operated on 2 weeks previously for chronic subdural hematoma. The posterior incision healed correctly (*dotted line*), but the anterior wound presents local signs of inflammation and infection near the incision and superficial wound dehiscence with delayed healing (*arrow*)



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_21

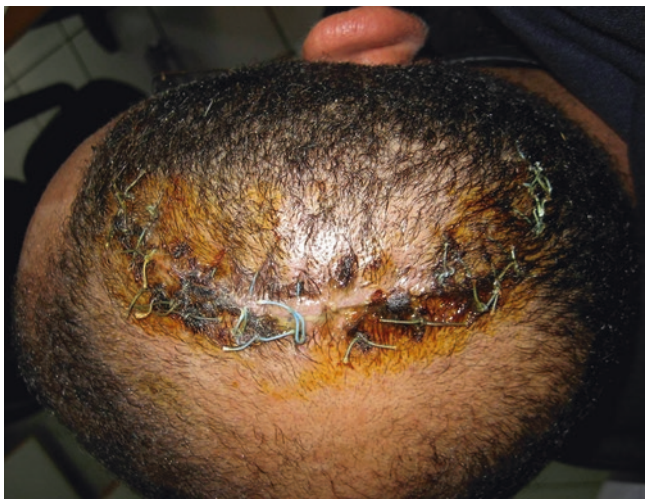

**Fig. 21.2** Surgical site infection in a 36-year-old man treated at another hospital 4 weeks before for a traumatic cranial extradural hematoma. There is a purulent wound discharge due to poor postoperative care

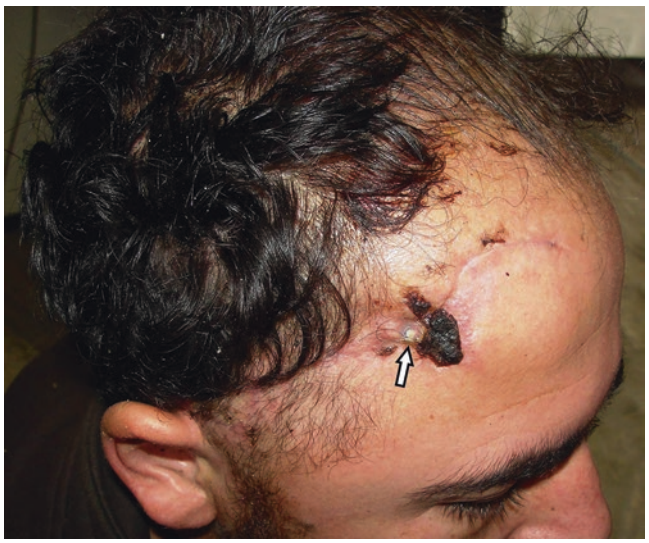

**Fig. 21.4** Postoperative purulent discharge (*arrow*) in a 41-year-old man 4 weeks after an operation for a frontal depressed skull fracture, with poor postoperative care

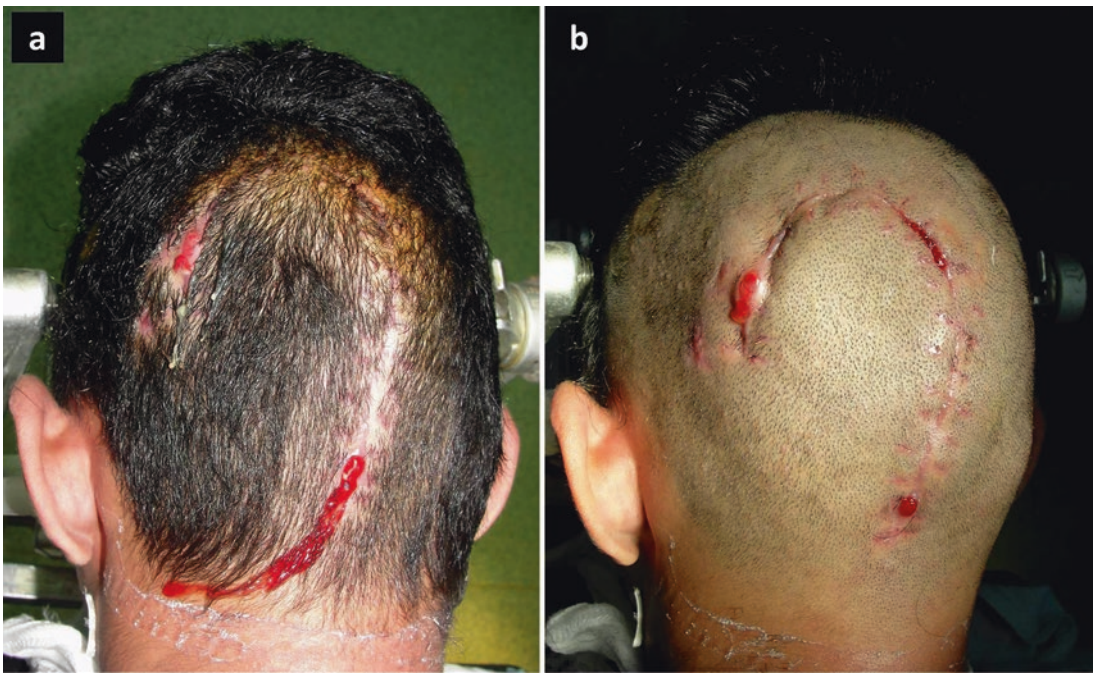


**Fig. 21.3** Wound infection with serous drainage in a 16-year-old boy 3 weeks after an operation at another institution for cranial extradural hematoma, shown before (**a**) and after (**b**) shaving

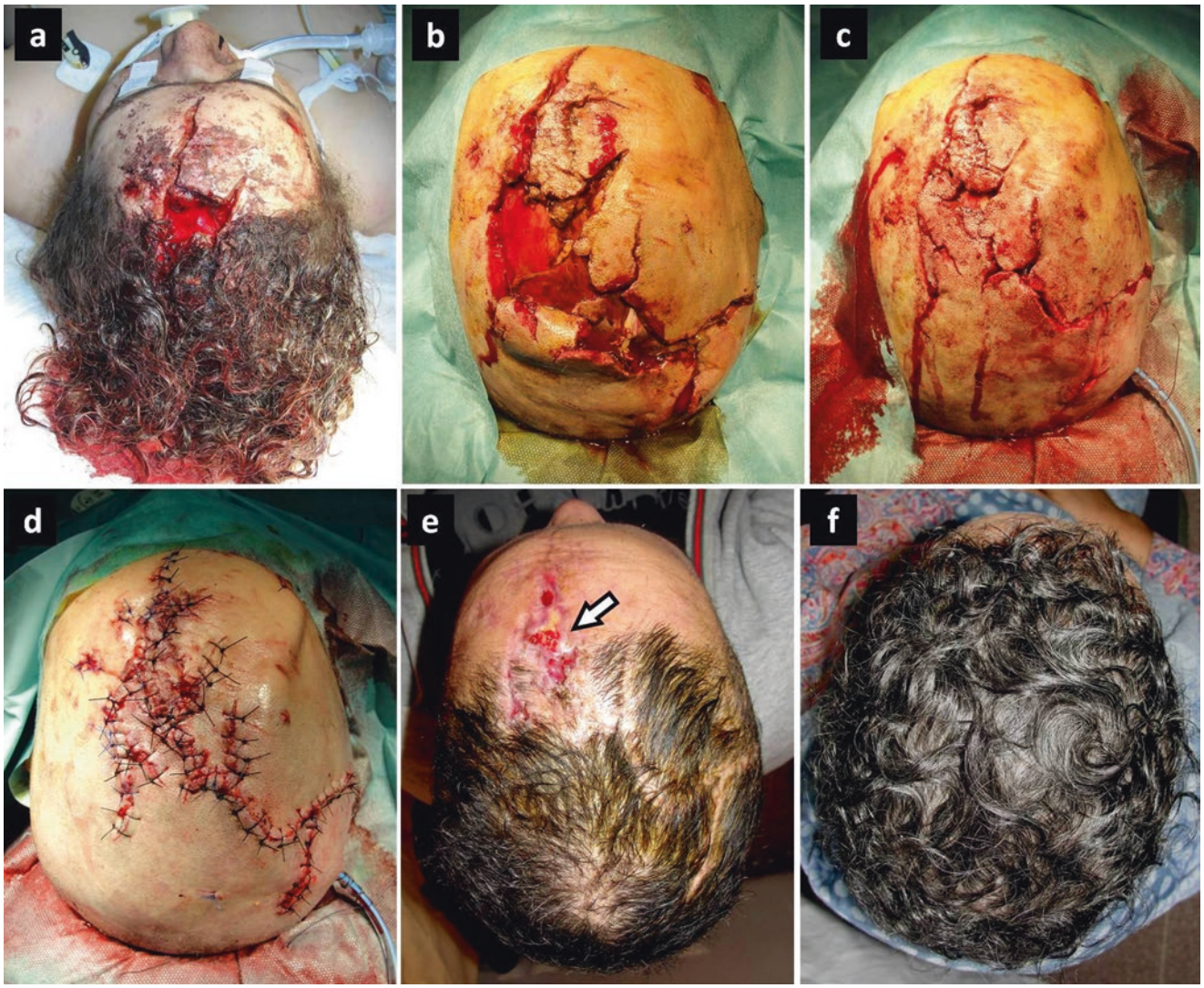


**Fig. 21.5** Posttraumatic extensive scalp laceration (**a**). Operative view after shaving and cleaning (**b**). Closing subcutaneous plane after placing a surgical drain (**c**). Superficial wound closure (**d**). Wound inflammation 5 weeks later (*arrow*) (**e**). Complete healing (4 months later) (**f**)

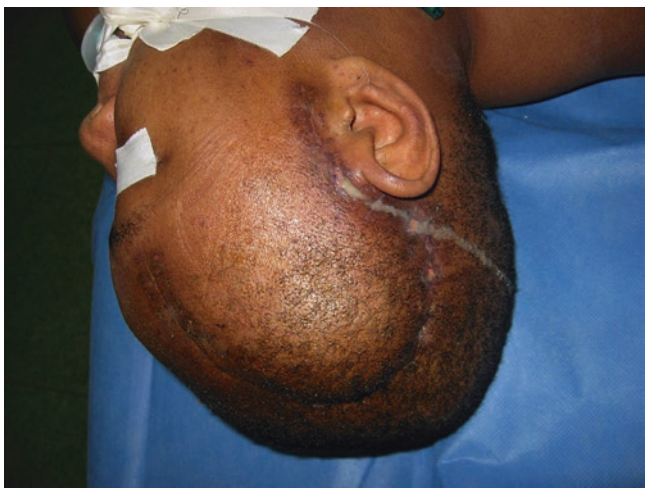

**Fig. 21.6** Case 21.1. Temporal cutaneous and deep scalp dehiscence with purulent discharge occurring 4 weeks after operation for right sphenoid wing meningioma (Reproduced from Akhaddar (2016); with permission of Springer Publishing)

21.9, 21.10, 21.11, 21.12, 21.13, 21.14, 21.15, 21.16, 21.17, and 21.18). Intradural infections can be contained in the subdural space (empyema) or an intracerebral setting (abscess), or they can be generalized (meningitis and/or ventriculitis) (Figs. 21.19,21.20, 21.21, 21.22, 21.23, 21.24, 21.25, 21.26, 21.27, and 21.28). Infection may affect a single location or a combination of locations.

Risk factors for SSI in cranial surgery can be categorized as patient related or procedure related. Patient-related risk factors include diabetes mellitus, obesity and malnutrition, tobacco use, concomitant infection at another site, prolonged preoperative hospitalization, colonization with *Staphylococcus aureus*, compromised immune function, corticosteroid use, and older age. Procedural risk factors are related to inappropriate antibiotic prophylaxis, duration of surgery longer than 4 h, implantation of foreign material, type and method of surgery, defective sterile technique, local

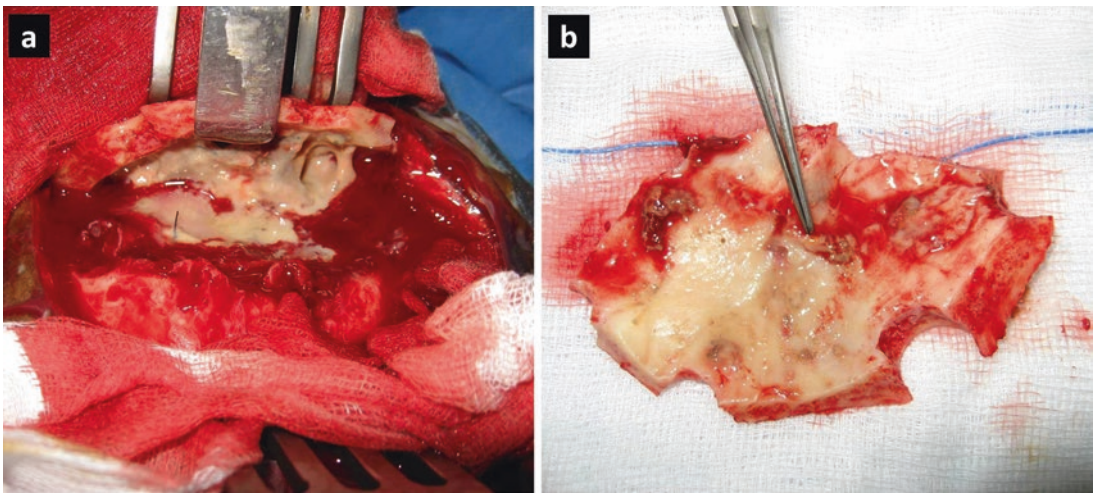


**Fig. 21.7** Case 21.1. Operative views. Presence of a large temporal epidural abscess (**a**). Note the infected cranial bone flap (**b**) (Reproduced from Akhaddar (2016); with permission of Springer Publishing)

radiation and chemotherapy, and (above all) leakage of cerebrospinal fluid (CSF). In addition, it is important to consider some postoperative conditions such as unsatisfactory local wound care and poor patient primary healing.

Superficial wound infections are the most common SSI, followed in order by meningitis, epidural and subdural empyema, bone flap osteomyelitis, and brain abscesses.

## Clinical Presentations

The latency of SSI after cranial surgery is varied. Scalp infections (median of 13 days) and meningitis or ventriculitis (median of 7 days) occur earlier than intradural purulent collections (median of 15 days), and bone flap infections (median of 27 days) appear even later.

Clinical symptoms vary greatly depending on the site of infection, the virulence of the microorganisms, the patient's age and premorbid conditions, the type of surgical procedure, and the timing of infection. The most frequent clinical findings are fever, purulent drainage, mental status change, headache, swelling, seizure, and focal neurologic deficits. Fever is an inconstant symptom, so the absence of fever cannot exclude the possibility of infection.

Postoperative extradural SSIs present principally with local signs of inflammation with or without fluid/purulent discharge, dehiscent or opened wound, or local pain and tenderness. Usually, focal neurologic deficits are absent. Manifestations of bone flap infection may be delayed.

Patients with meningitis typically present with fever, neck stiffness, and altered level of consciousness. Cases with CSF shunt infection show clinical signs of acute bacterial meningitis and sometimes symptoms of shunt malfunction or obstruction (Figs. 21.29, 21.30, 21.31, and 21.32).

Postoperative intradural suppurative collections tend to manifest by the development of new focal neurologic deficits or worsening of preexisting ones, normal or elevated temperature, and progressively impaired level of consciousness.

Some SSIs can be insidious and can cause few or no symptoms, such as occasionally only an intermittent, low-grade fever or general malaise. The surgeon should consider the possibility of infection in these patients and institute an appropriate diagnostic assessment.

Attention also should be paid to possible concomitant infection elsewhere in the body.

## Imaging Features

In the postoperative period, the most useful neuroimaging exploration is the CT scan, which offers an interesting basis for the diagnosis of cranial and intracranial infections. Usually, infections present as a fluid collection with surrounding enhancement. MRI also can provide structural as well as perfusional information in screening for these infections.

In patients with meningitis, the CT scan is usually normal, or it may demonstrate ventricular dilatation. On MRI, T1-weighted images may reveal gadolinium enhancement of the leptomeninges.

Bone flap osteomyelitis is best evaluated on CT scans. The most frequent findings are erosion and thinning of the

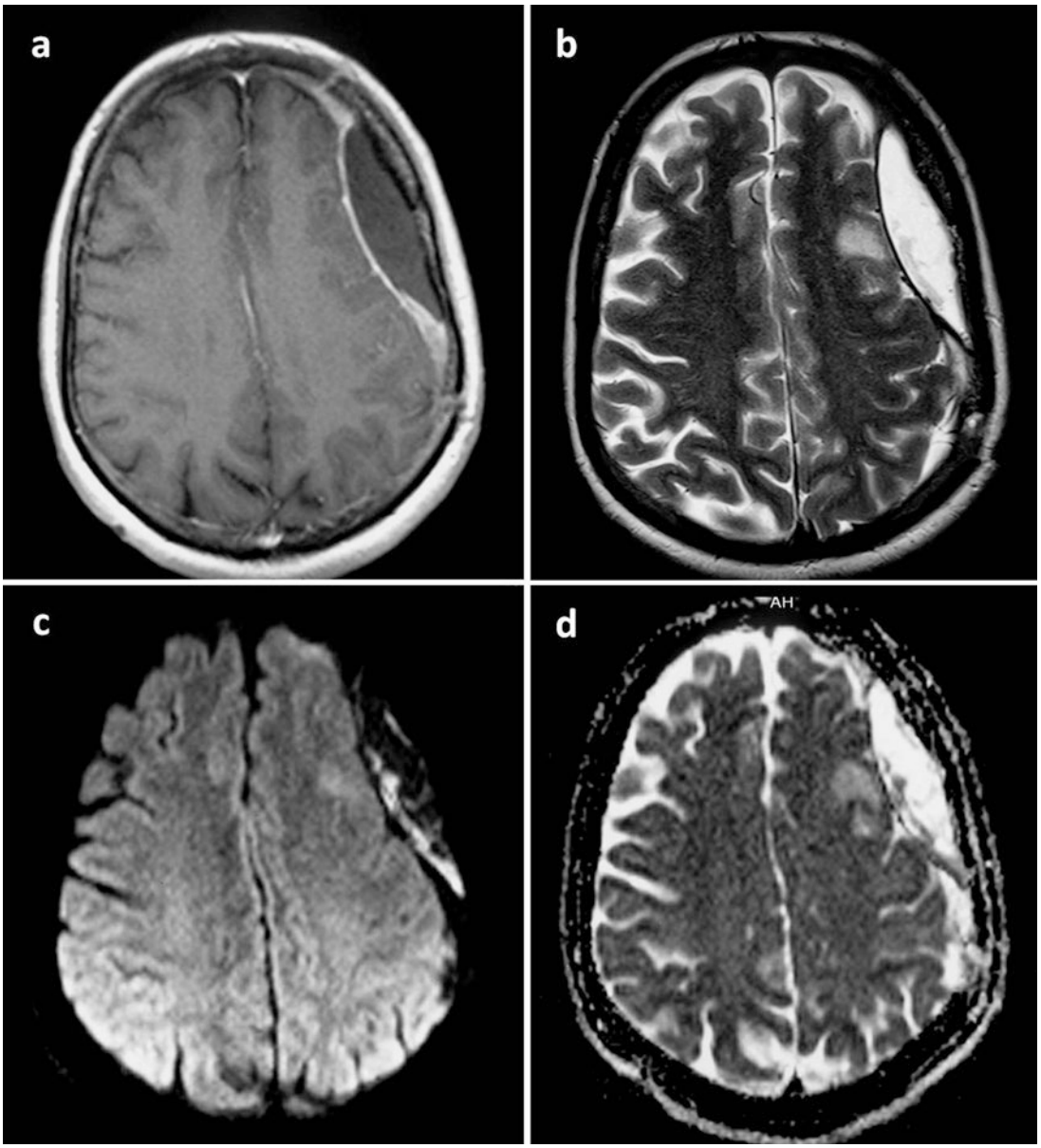


**Fig. 21.8** Case 21.2. Cranial epidural abscess occurring 2 months after complete surgical removal of an adjacent cranial vault meningioma, shown on an axial cranial-enhanced T1-weighted image (**a**), T2-weighted image (**b**), diffusion-weighted image (*DWI*) (**c**), and apparent diffusion coefficient (*ADC*) map (**d**)

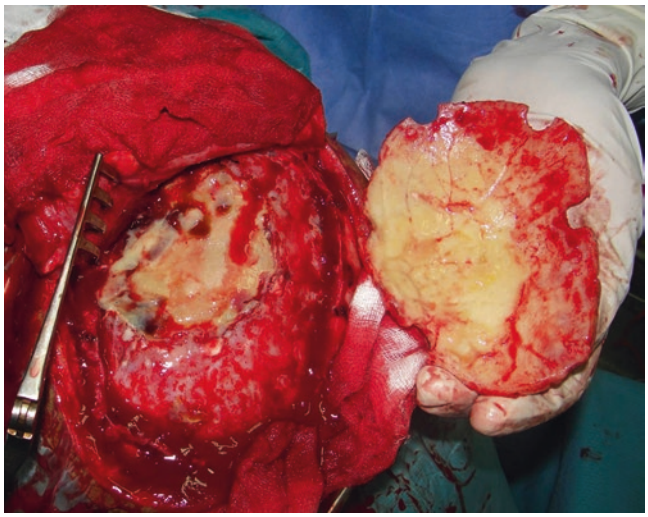

**Fig. 21.9** Case 21.2. Operative view. The wound was reopened. When the bone flap was elevated, an abundant purulent collection was observed. Yellowish pus was adherent to a Gore-Tex® dural plastia implanted in the previous operation for cranial vault meningioma (Reproduced from Akhaddar (2016), p. 195, with permission of Springer Publishing)

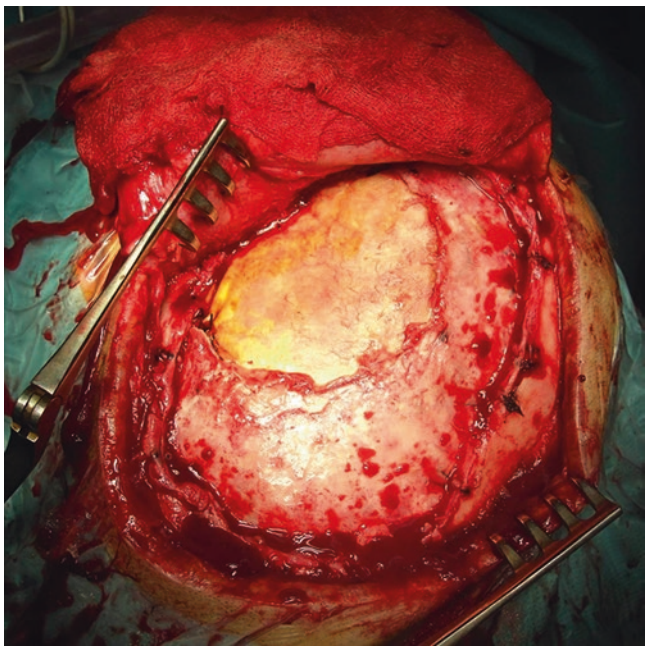

**Fig. 21.10** Case 21.2. Operative view after careful extradural drainage, debridement (soft tissues, granulations, abscesses, and bone), and washing of the surgical site with antiseptic products (Reproduced from Akhaddar (2016), p. 195, with permission of Springer Publishing)

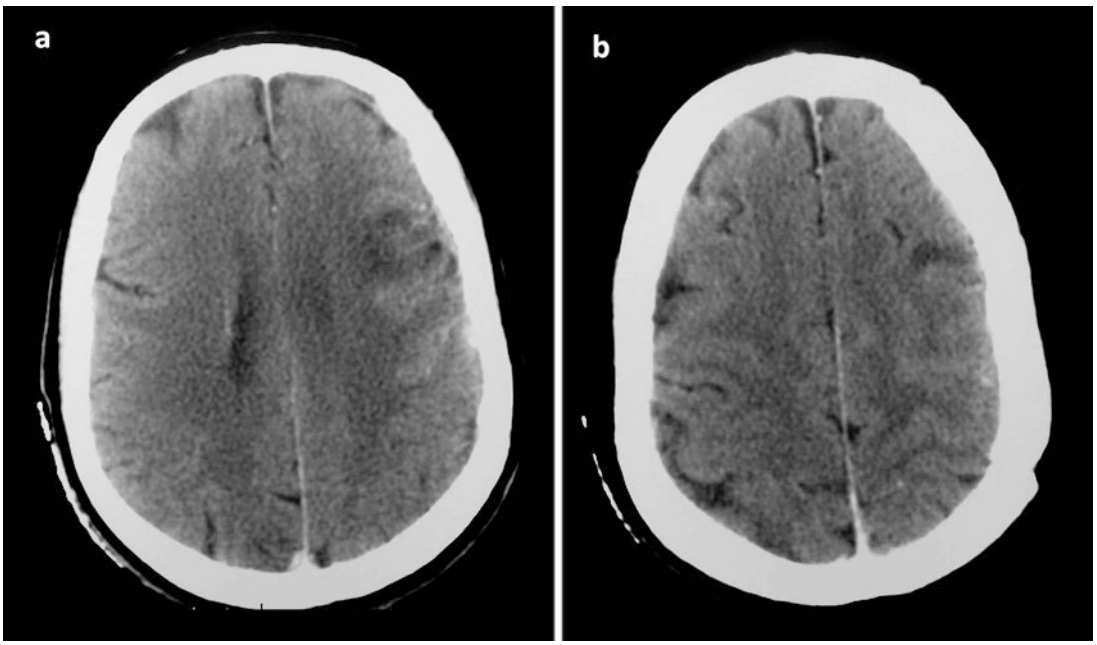


**Fig. 21.11** Case 21.2. Control axial CT scan with contrast injection (**a**, **b**), showing complete resolution of the epidural suppurative collection

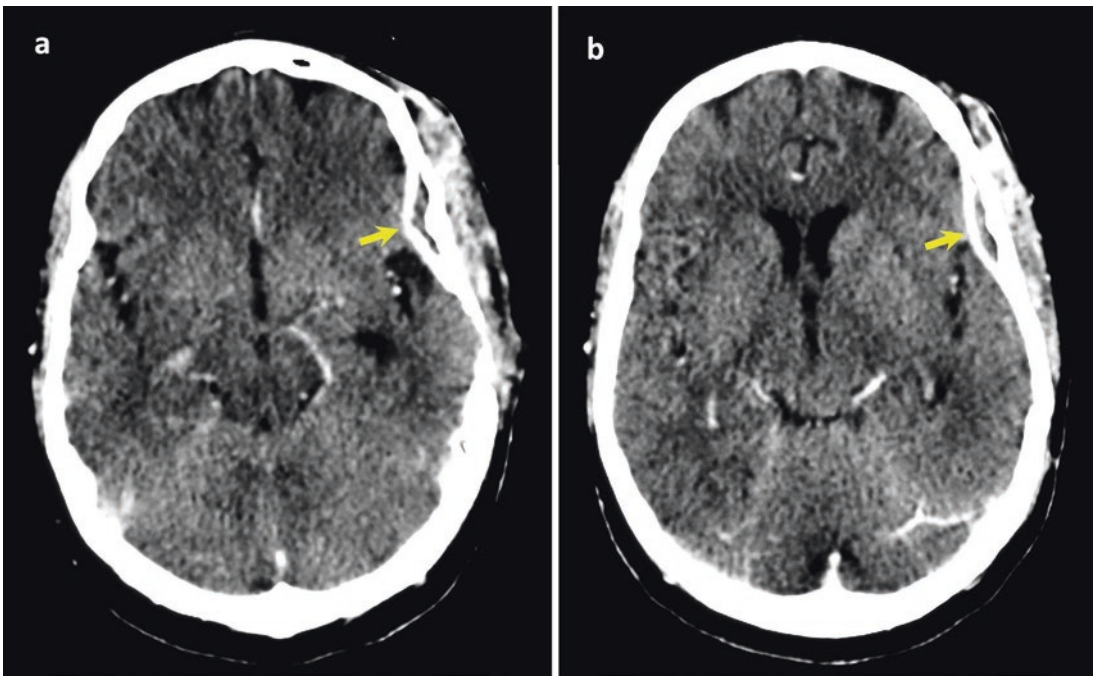


**Fig. 21.12** Case 21.3. Cranial epidural abscess occurring 4 weeks after complete surgical removal of a sphenoid wing meningioma. Axial cranial-enhanced CT scan (**a**, **b**) revealing a left frontotemporal epidural abscess formation with ring enhancement (*arrow*)

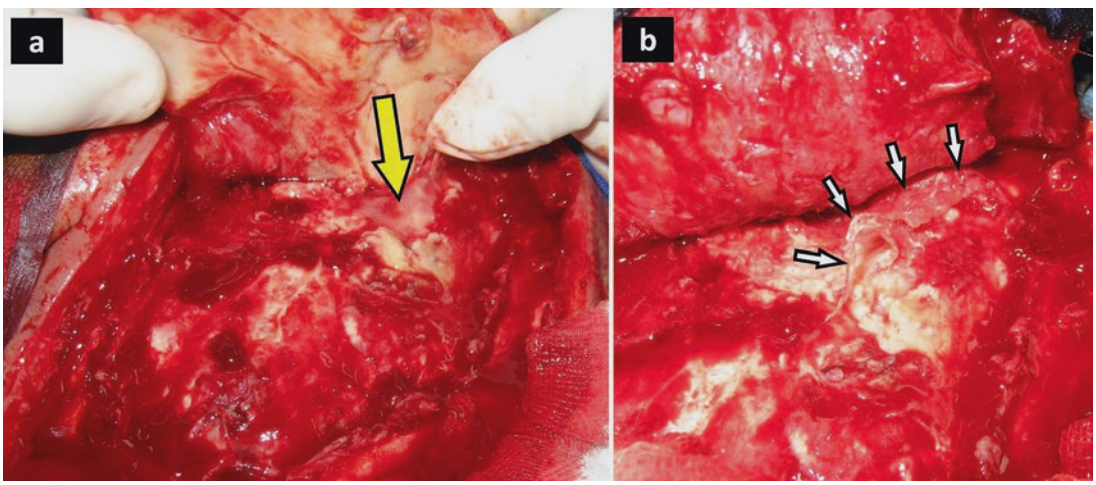


**Fig. 21.13** Case 21.3. Operative view after bone flap removal, showing the extradural suppurative collection (*arrow*) (**a**). During surgical debridement and cleaning of the surgical site infection, an epidural surgical cotton (retained foreign body textiloma) (*arrows*) was found (**b**)

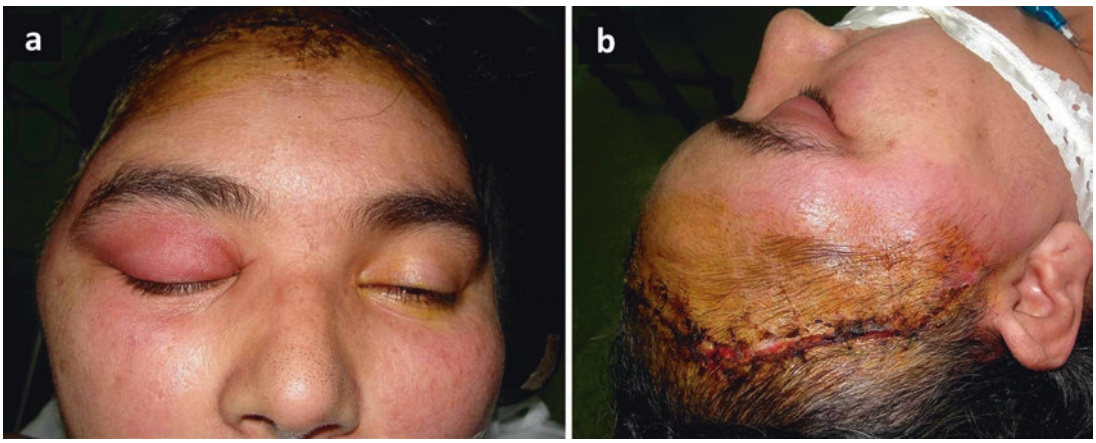


**Fig. 21.14** Case 21.4. Clinical photographs showing right frontotemporal swelling with periorbital cellulitis (**a**). Note the wound inflammation secondary to deep incisional infection (**b**) occurring 2 weeks postoperatively, following a subfrontal approach for a large olfactory groove meningioma (Reproduced from Akhaddar (2016), p. 196, with permission of Springer Publishing)

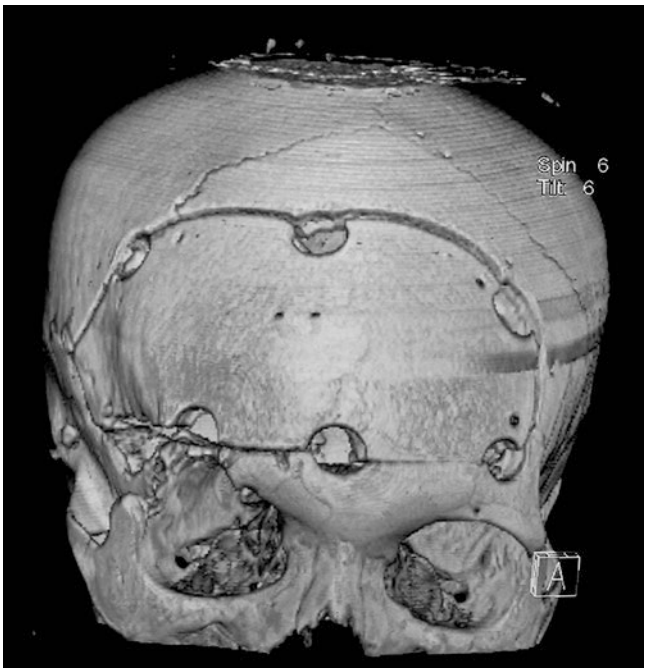


**Fig. 21.15** Case 21.4. Postoperative three-dimensional bone CT scan reconstruction (reformatted volume rendered) showing the location of the bone flap

involved bone, with surrounding areas of gas in the subgaleal and/or extradural space.

Suppurative collections have a low density on CT scan and high-intensity signal on T2-weighted MRI, with surrounding enhancement following contrast administration. Imaging of brain abscess varies with the stage of the abscess. The localized subdural empyema or brain abscess may be seen within the resection cavity. Because the subdural space and post-resection cavity may be unified, both subdural empyema and brain abscess may result together.

## Laboratory Findings

Inflammatory biomarkers are highly variable in their expression in the postoperative period. The CRP level remains the most useful postoperative parameter, especially if it remains elevated beyond the fourth postoperative day or becomes elevated again after normalization. The procalcitonin level is more specific and helpful in follow-up, but it lacks sensitivity. Nevertheless, SSI should not be excluded in a patient with no fever, normal white blood cell count, abnormal erythrocyte sedimentation rate (ESR), and a negative blood culture.

When possible, CSF should be evaluated in a patient with suspected postoperative intradural infections. Perturbation of protein/glucose levels and white blood cell count may be suggestive, but only gram staining and CSF culture confirm the diagnosis.

Microorganisms found in SSI following cranial surgery are quite similar. The most causative pathogens are gram-positive skin flora, especially *Staphylococcus aureus* and *Staphylococcus epidermidis*, but gram-negative bacteria are not unusual. Fungal infections are rare but should always be considered. Brain abscesses are often polymicrobial.

## Treatment Options

Medical treatment may start with a broad spectrum of antibiotics, with targeted treatment initiated according to the antimicrobial susceptibility testing.

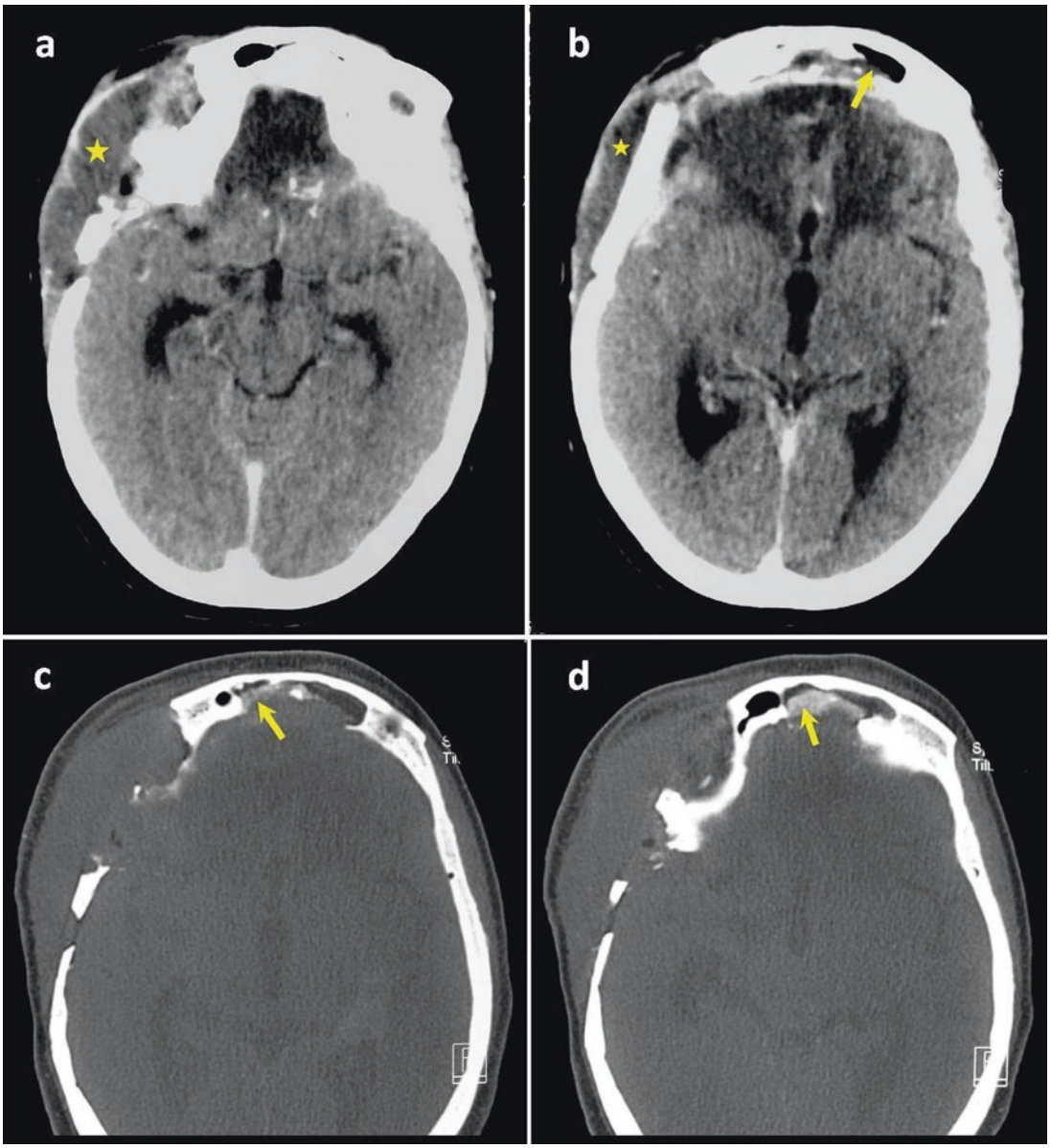


**Fig. 21.16** Case 21.4. Axial cranial CT scan after contrast injection (**a**, **b**) and with bone windows (**c**, **d**) showing the extracranial abscess formation (*star*). Note the incomplete right frontal sinus cranialization (*arrow*) (Reproduced from Akhaddar (2016), p. 197, with permission of Springer Publishing)

Patients with limited scalp infections and those with isolated meningitis or without collections on neuroimaging may be managed with local measures and antimicrobial therapy alone. These patients require careful clinical, biological, and neuroimaging monitoring, however, and should be considered for surgery if complications arise.

Scalp abscess, bone flap infection, and epidural abscess should be inspected in the operating room, with cleaning, evacuation of fluids, debridement of devitalized tissue, local antiseptic techniques, drainage, and careful wound closure. With large scalp defects, specific scalp reconstruction options may be required, to be performed by a plastic surgeon. The bone flap can be removed if needed, and a delayed cranioplasty may be achieved several months later. Hyperbaric oxygen therapy may be useful in managing refractory cases of postsurgical cranial osteomyelitis (Fig. 21.33).

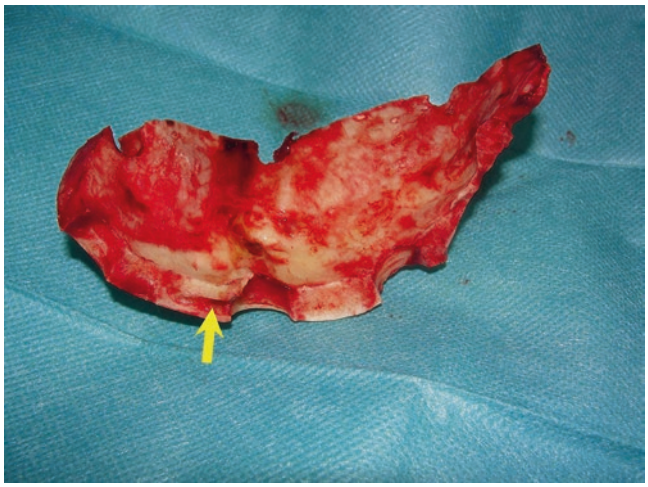

**Fig. 21.17** Case 21.4. Infected bone flap with left frontal sinus incompletely cranialized (*arrow*) (Reproduced from Akhaddar (2016), p. 198, with permission of Springer Publishing)

When subdural empyema occurs, removing the pus through a craniotomy is better than using burr holes to achieve sufficient drainage. Treatment of postoperative brain abscess (unlike spontaneous cases) requires surgical reopening of the surgical site to obtain specimens, drain the suppurative collection, and inspect and clean the resection cavity.

In some selected patients with intradural suppurative collections, medical therapy alone can be considered, particularly in cases with a small lesion, very limited infectious extension, no intracranial mass effect, no neurologic deficits, and an early favorable response to antibiotics. These patients should be followed carefully and frequently evaluated, however, with surgical intervention when necessary.

CSF fistula should be managed surgically when diagnosed to prevent any form of SSI, especially postoperative meningitis. A short-term conservative treatment with lumbar puncture or a drain may be attempted to avoid reopening the surgical site, but definitive surgical closure of the CSF leak (with fascia lata or umbilical fat) is mandatory for most patients.

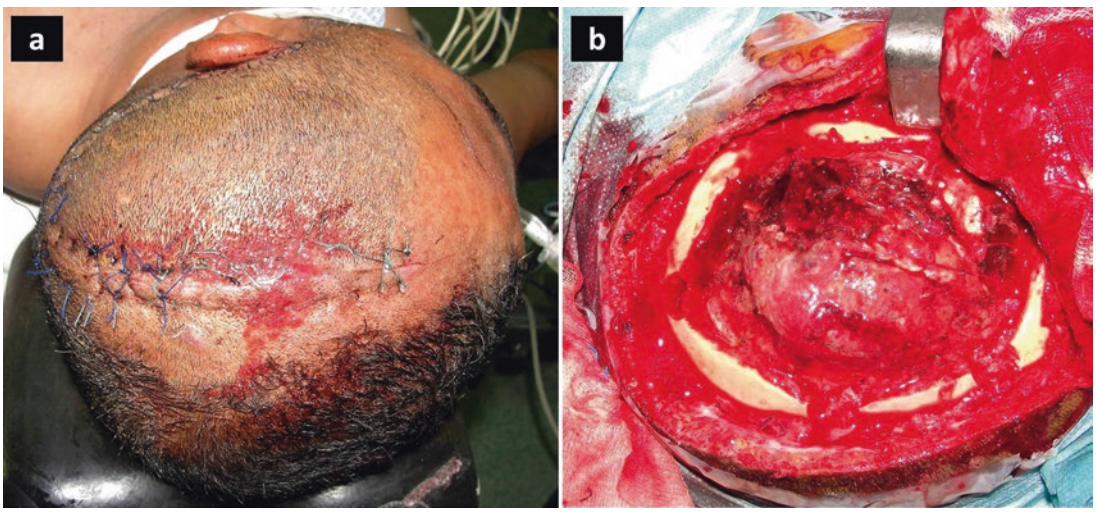


**Fig. 21.18** Surgical site infection in a 46-year-old man operated on 10 days before for a left temporoparietal cranial vault meningioma, showing a cloudy wound drainage (**a**). Operative view (**b**) shows seropurulent exudates into the epidural space before debridement and cleaning (Reproduced from Akhaddar (2016), with permission of Springer Publishing)

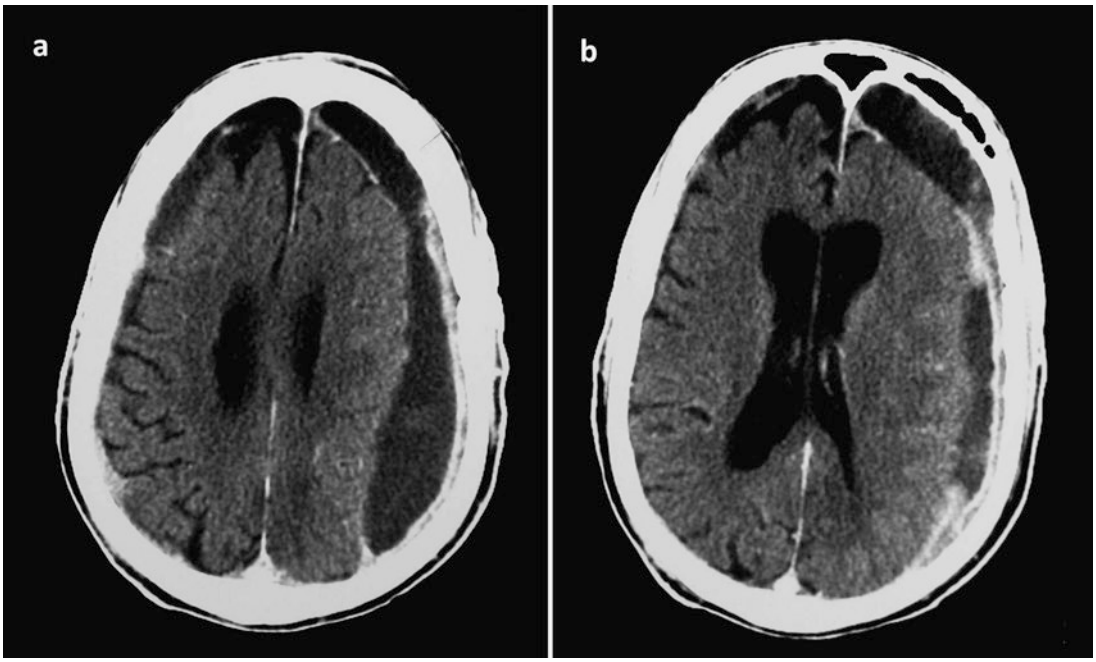


**Fig. 21.19** Case 21.5. (**a**, **b**) Postoperative subdural empyema in an 80-year-old man 4 weeks after surgery for a chronic subdural hematoma. Axial cranial CT scan with contrast injection

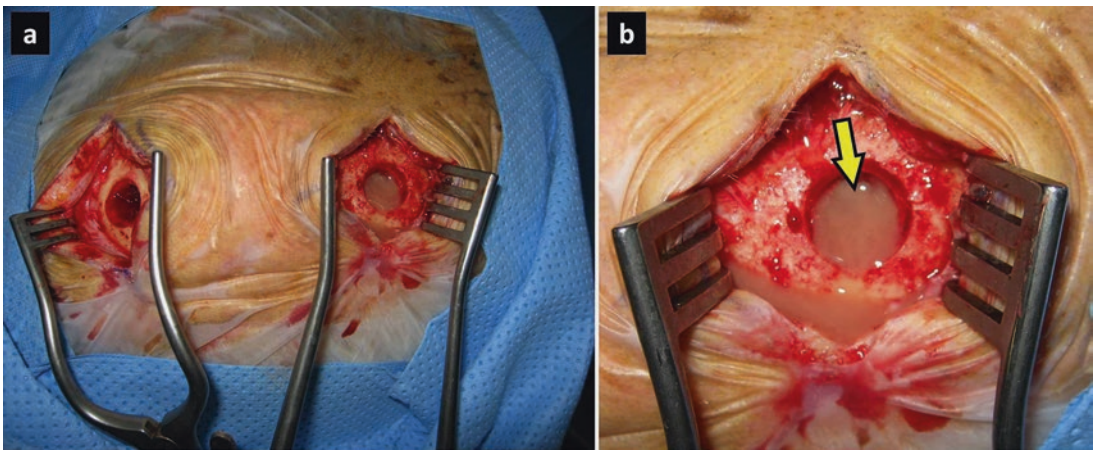


**Fig. 21.20** Case 21.5. (**a**, **b**) Operative views showing the suppurative content of the intracranial subdural collection (*arrow*)

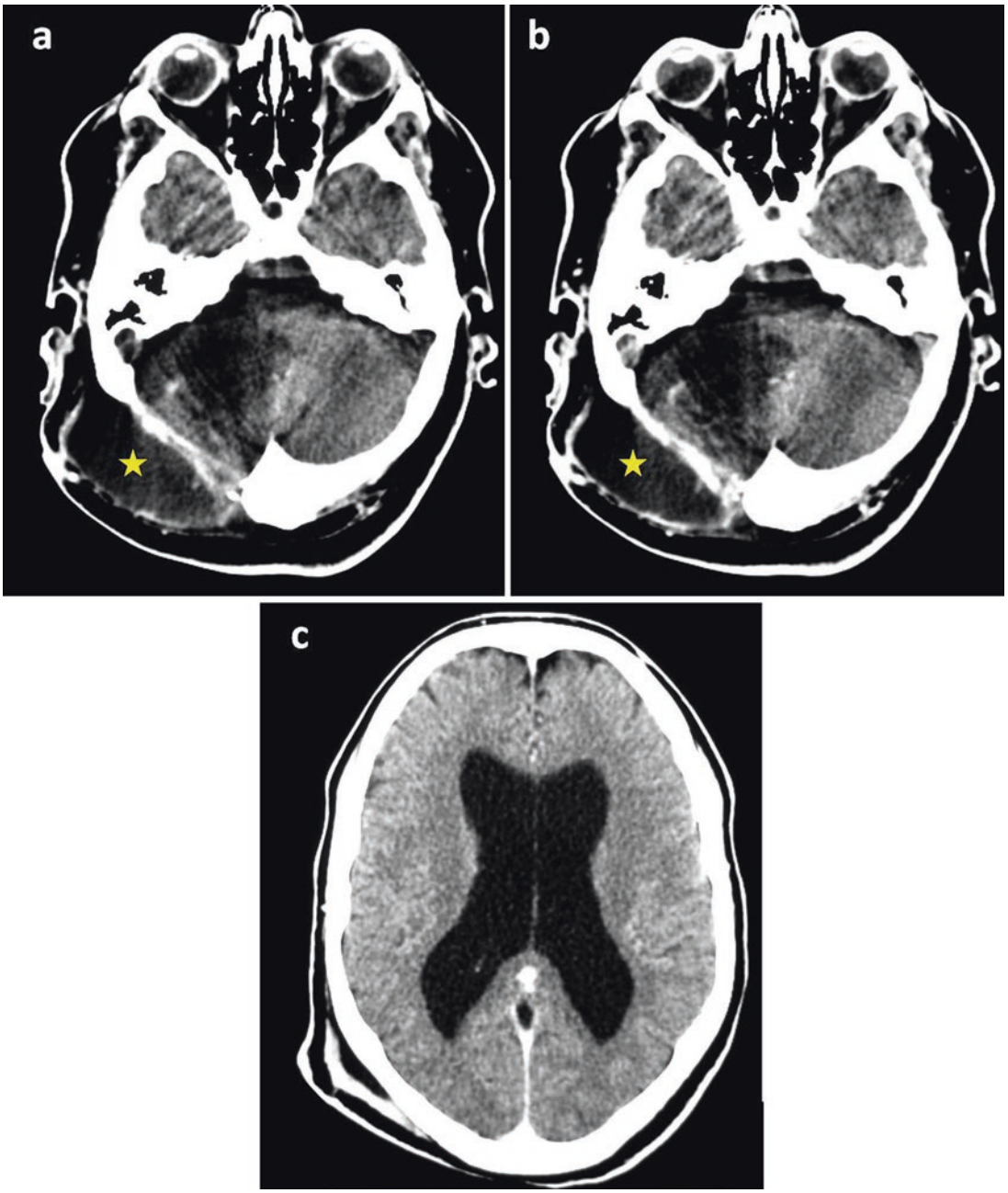


**Fig. 21.21** Axial cranial CT scan without (**a**) and with contrast administration (**b**, **c**) showing a large right suboccipital pseudomeningocele (*star*) in a patient operated on 3 weeks previously for a giant vestibular schwannoma of the cerebellopontine angle. This patient also has bacterial meningitis. Note the peripheral enhancement of the pseudomeningocele (**b**) and the concomitant hydrocephalus (**c**)

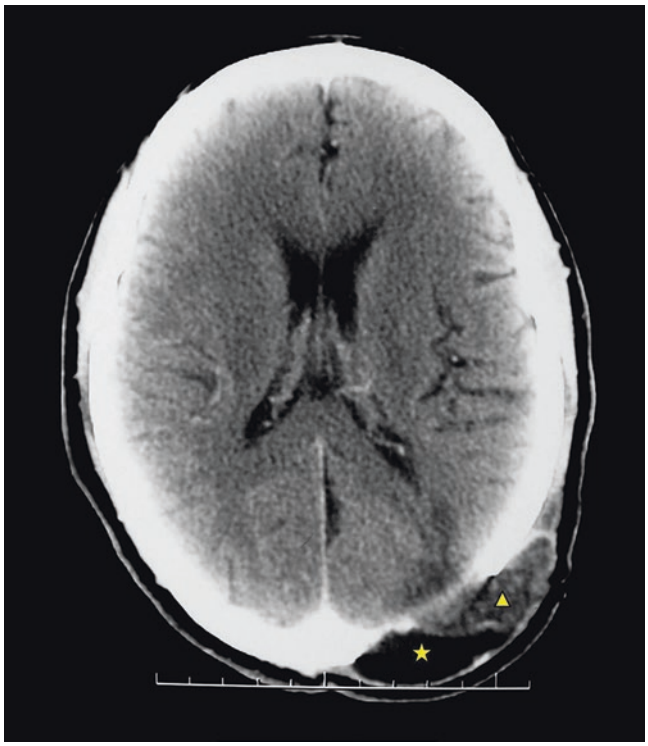

**Fig. 21.22** Left parieto-occipital postoperative infected pseudomeningocele 4 weeks after surgery for a convexity meningioma. Axial cranial-enhanced CT scan showing the pseudomeningocele with a fluid–fluid level (*triangle*/*star*) and peripheral enhancement

Management of CSF shunting infection requires surgical removal of the shunt, placement of temporary external ventricular drainage, intravenous antibiotic therapy, and implantation of a new shunt system if needed when the infection has been eradicated.

In all cases, if hardware is involved with serious infection, the foreign material should be removed during revision surgery (Figs. 21.34 and 21.35).

Supportive therapy is often needed, including fluids, analgesics, antipyretics, anticoagulation, anticonvulsants, and functional rehabilitation. Some patients with sepsis or even septic shock may require initial resuscitation with a stay in the intensive care unit for treatment.

Strict awareness of hygiene with careful application of preoperative and postoperative protocols will decrease neurosurgical site infections. The surgeon should control the general operating room situation, prepare the surgical case carefully, adhere to antiseptic principles and antibiotic prophylactic therapies, minimize tissue damage, operate in a timely fashion, and close the wound meticulously.

## Outcomes

If treated rapidly and vigorously, SSI may resolve without sequelae, but many complications may occur, depending on the type and location of infection, the age and underlying condition of the patient, the type of surgical procedure, the early diagnosis, and the response to treatment.

Inadequate or inappropriate treatment can cause recurrence. Neurologic morbidity and mortality are more likely for patients with intradural infections than for those for extradural infections. Sequels consist of persistent seizures, residual focal neurologic deficits, permanent alteration in mental status, and esthetic scars.

Outcomes after shunt infection are variable. The risk of reinfection is higher. Patients may develop polycystic and multiloculate hydrocephalus, seizures, and decreased intellectual performance (especially in children).

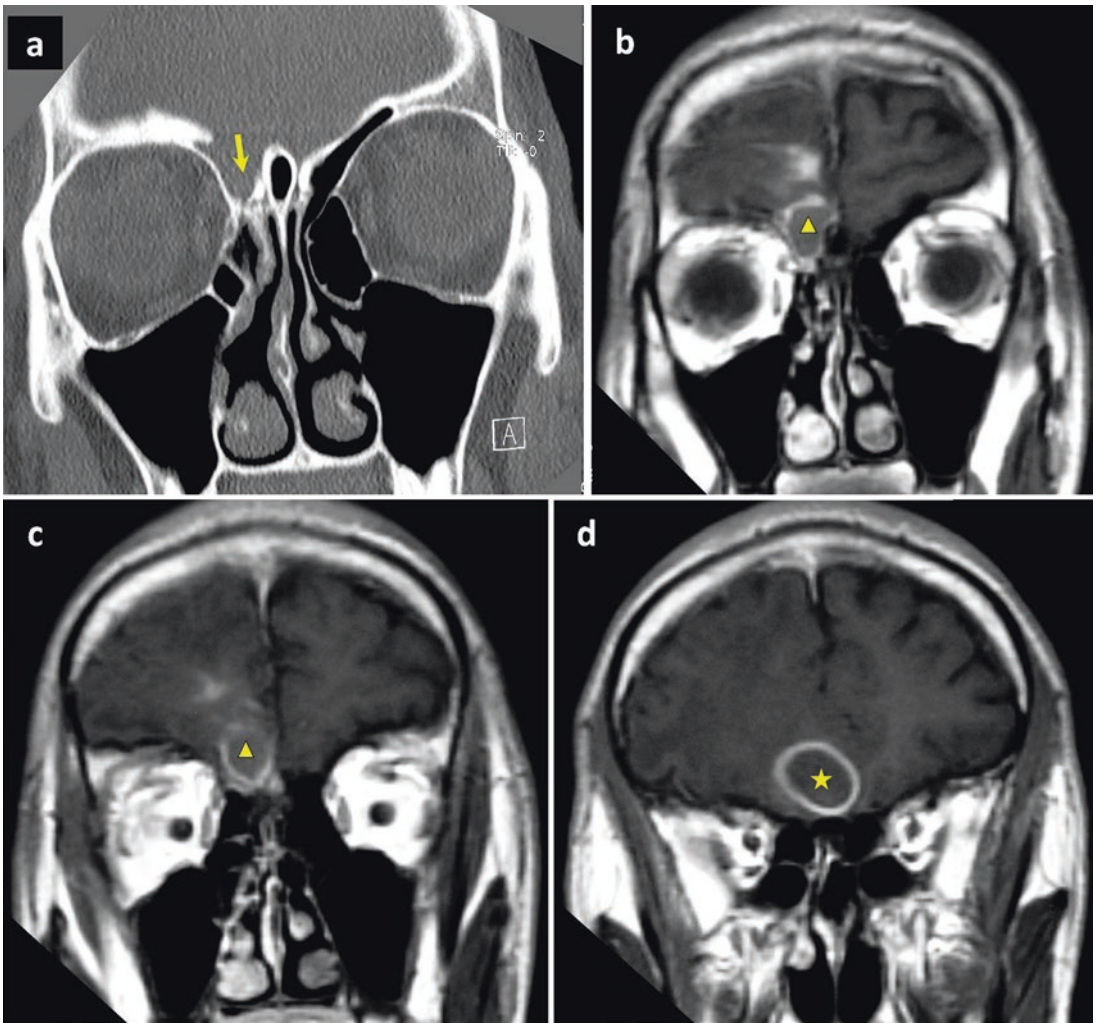


**Fig. 21.23** Case 21.6. Coronal craniofacial CT scan (**a**) and T1-weighted MR images with gadolinium injection (**b**–**d**). This patient was operated on for an anterior spontaneous CSF fistula due to a cribriform plate defect (*arrow*), using a transnasal endoscopic approach. One month later, he developed two intracranial suppurative collections: one in the adjacent subdural space (*triangle*) and one within the frontal brain parenchyma (*star*)

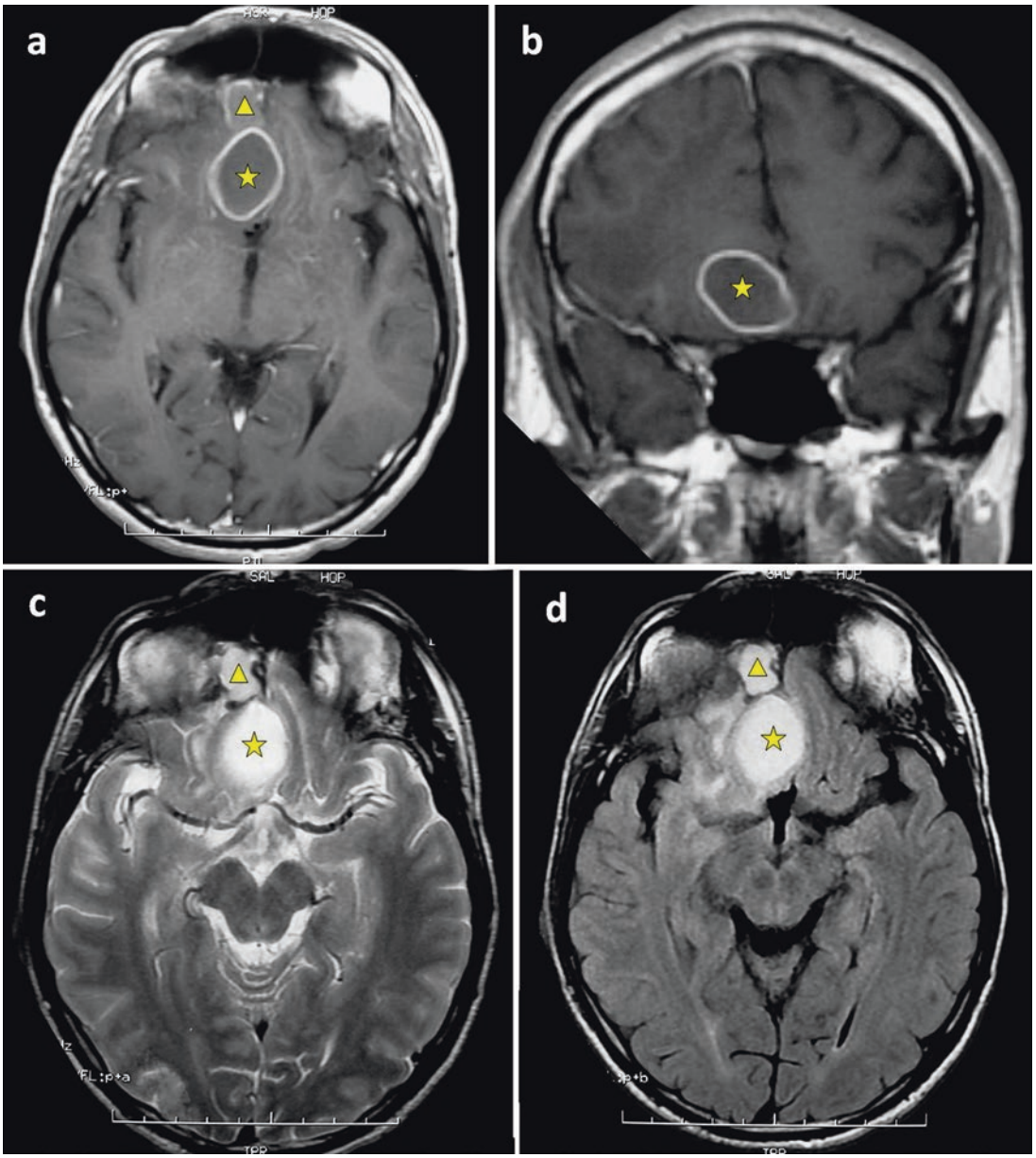


**Fig. 21.24** Case 21.6. Axial (**a**) and coronal (**b**) post-gadolinium T1-weighted images, axial T2-weighted image (**c**), and FLAIR sequence (**d**). These two abscesses (*triangle* and *star*) are isointense to hypointense on T1-weighted images and hyperintense on T2-weighted images, with ring enhancement following gadolinium injection

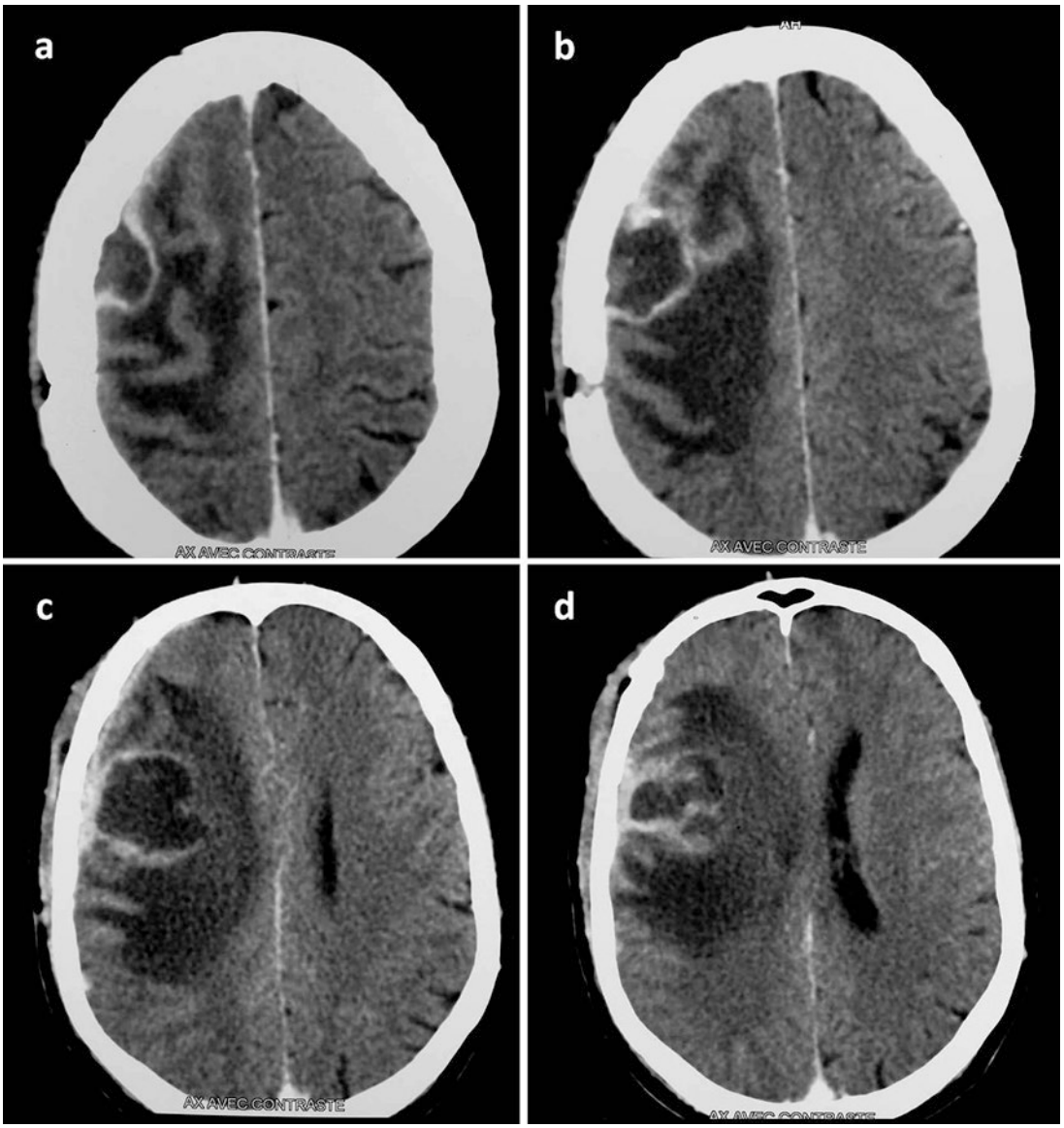


**Fig. 21.25** (**a**–**d**) Axial brain CT scan with contrast injection showing postoperative brain abscess located in the right frontal lobe in a 53-year-old man operated on 7 weeks before for an adjacent convexity meningioma

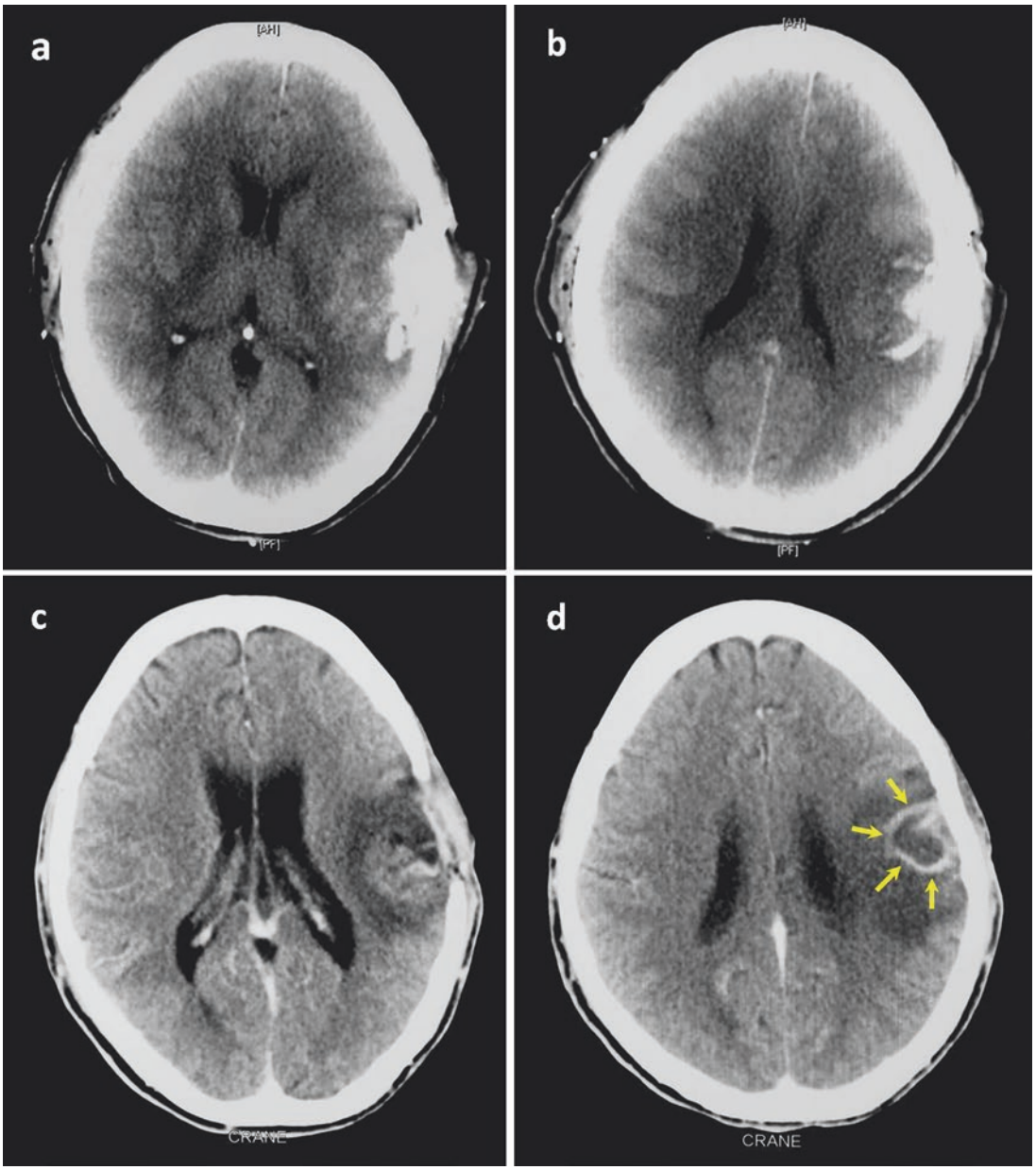


**Fig. 21.26** This 28-year-old man was operated on for an open depressed skull fracture located in the left frontoparietal area, seen on axial cranial CT scan without contrast injection (**a**, **b**). Six weeks later, he developed an adjacent intraparenchymal brain abscess. Postoperative enhanced CT scan (**c**, **d**) shows the ring-enhancing brain lesion "abscess formation" (*arrows*) with extensive perifocal edema

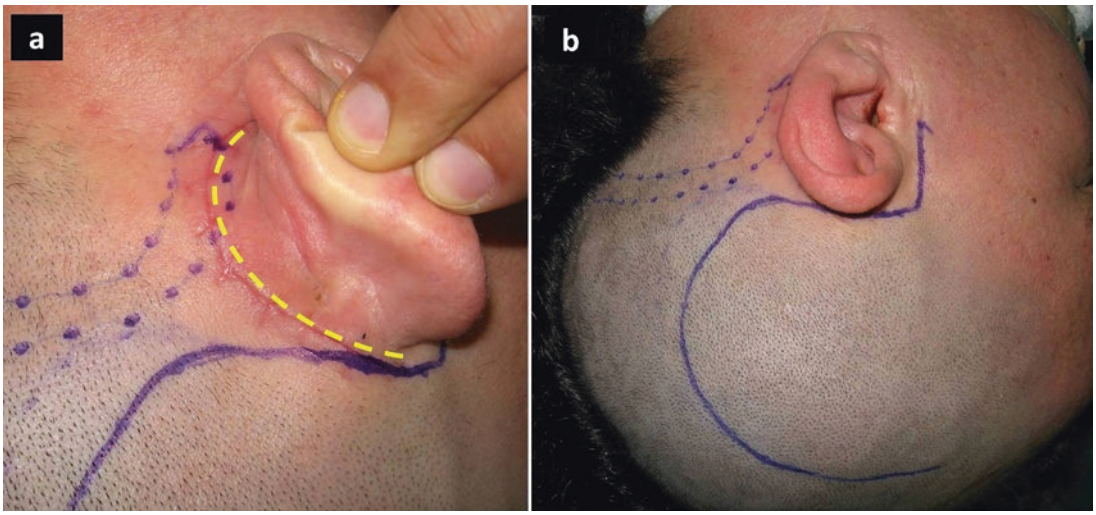


**Fig. 21.27** Case 21.7. Clinical picture of a 53-year-old woman 1 month after a cholesteatoma in the left middle ear was removed by an ENT team, using a retroauricular incision (*yellow dotted line*) (**a**). She developed a secondary CSF otorrhea with suspected meningitis. A suprapetrosal extradural approach was then planned (*purple continuous line*) to repair the dura mater (**b**)

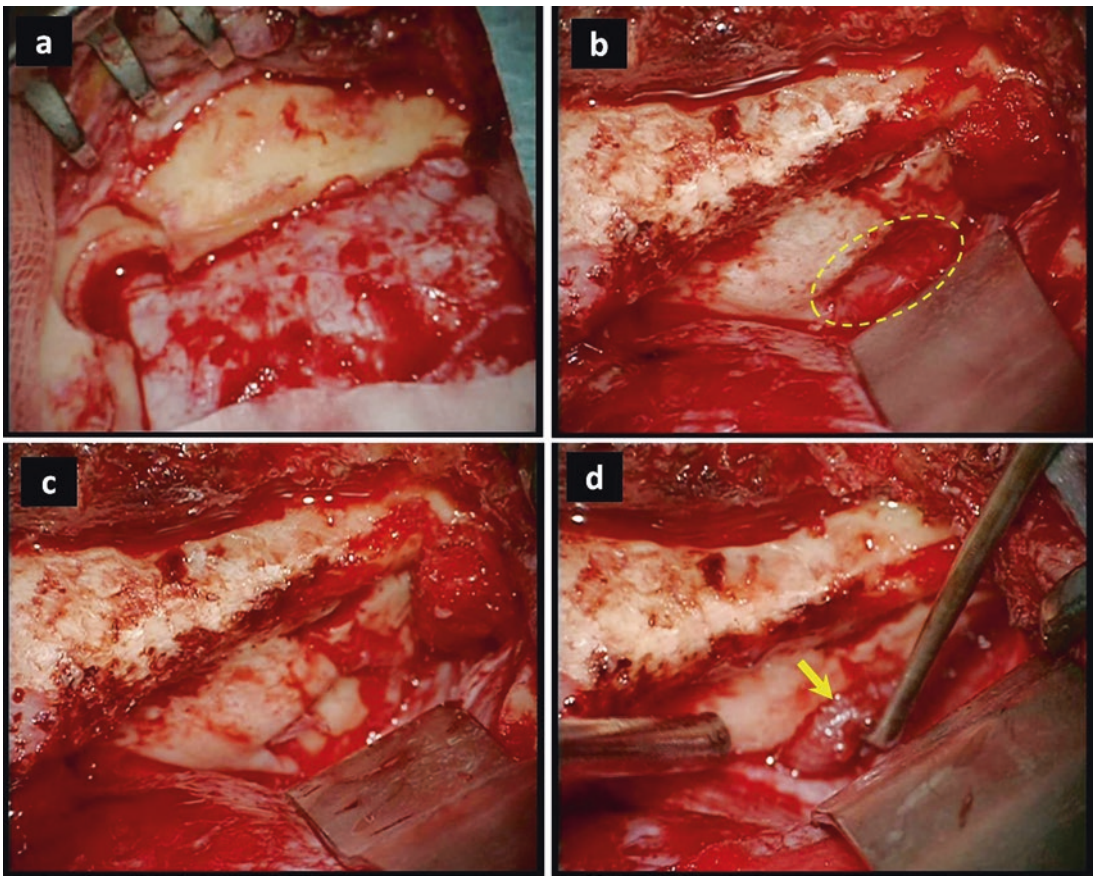


**Fig. 21.28** Case 21.7. Operative views under microscopic magnification (step-by-step neurosurgical procedure). Removing the temporal bone flap in the left side (**a**). Dissecting the dura and identifying the iatrogenic bone defect (*dotted oval*) (**b**). The osseous defect was delimited and cleaned (**c**), and a portion of temporal muscle graft was inserted inside the defect (*arrow*) (**d**). The muscle graft was secured by fibrin glue (**e**), packing of absorbable knitted fabric (Surgicel®) (**f**), and autologous bone (**g**, **h**) (multilayer closure). CSF fluid leakage was not identified intraoperatively. The patient had a good outcome

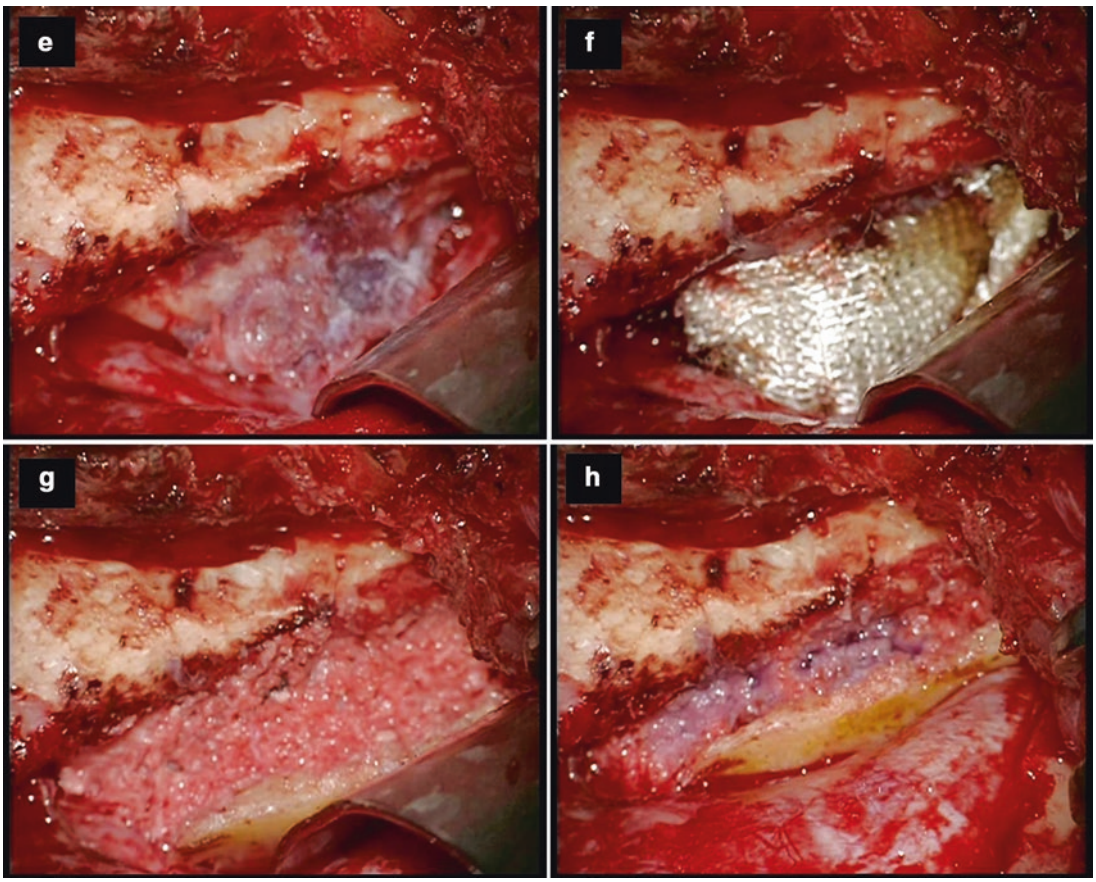


**Fig. 21.28** (continued)

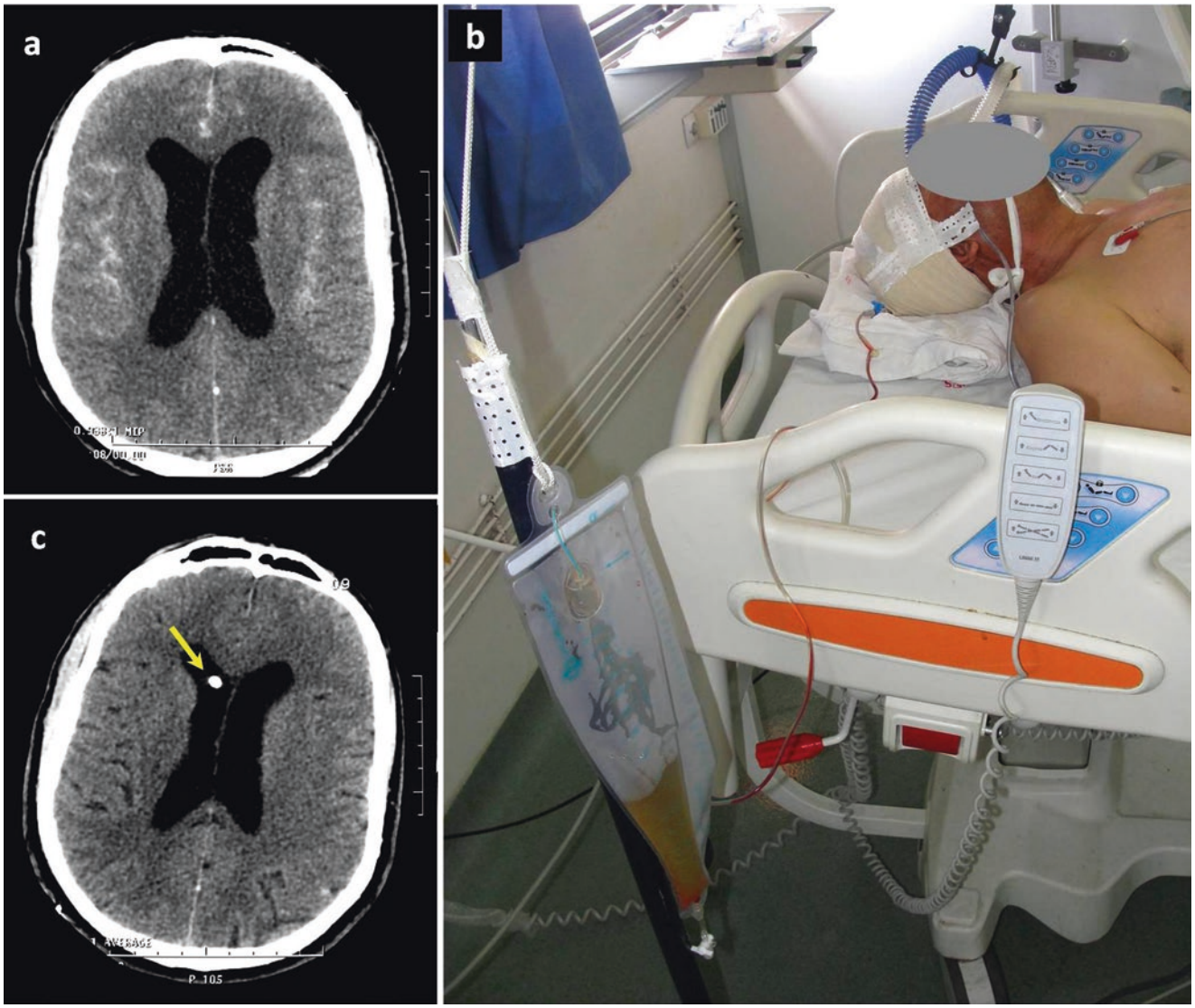


**Fig. 21.29** Axial cranial CT scan without contrast injection shows nontraumatic subarachnoid bleeding with acute hydrocephalus (**a**). Temporary external intraventricular CSF drainage was used in this patient, who developed meningitis in the seventh postoperative day (**b**). Control CT scan showing the right frontal intraventricular catheter (**c**)

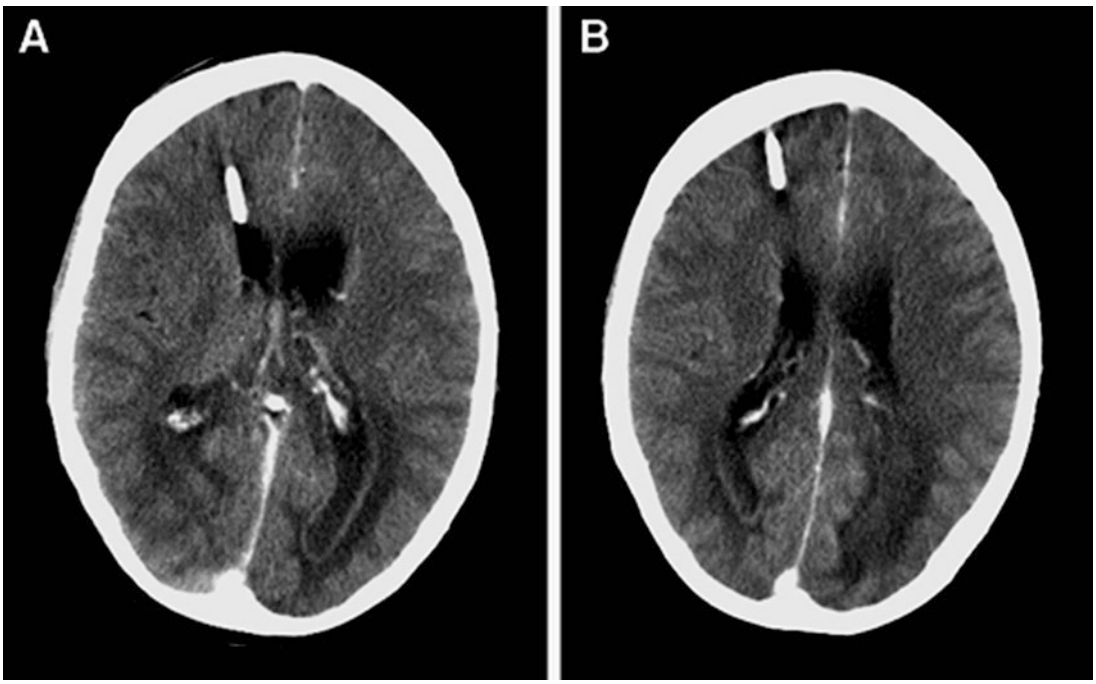


**Fig. 21.30** Contrast-enhanced CT scan of the brain on day 23 after admission for an intraparenchymal hematoma with extension of blood into the ventricles (**a**, **b**). A right frontal ventriculoperitoneal shunt is present. The ependymal lining of the occipital horns of the lateral ventricles shows thick-walled contrast enhancement, consistent with ependymitis. Confluent, hypodense margins around the ventricles indicate periventricular inflammation. In the dependent part of the occipital horns, there is intraventricular debris, which has a higher density than the CSF in the nondependent parts of lateral ventricles (From Jorens et al. (2009), with permission of Springer Publishing)

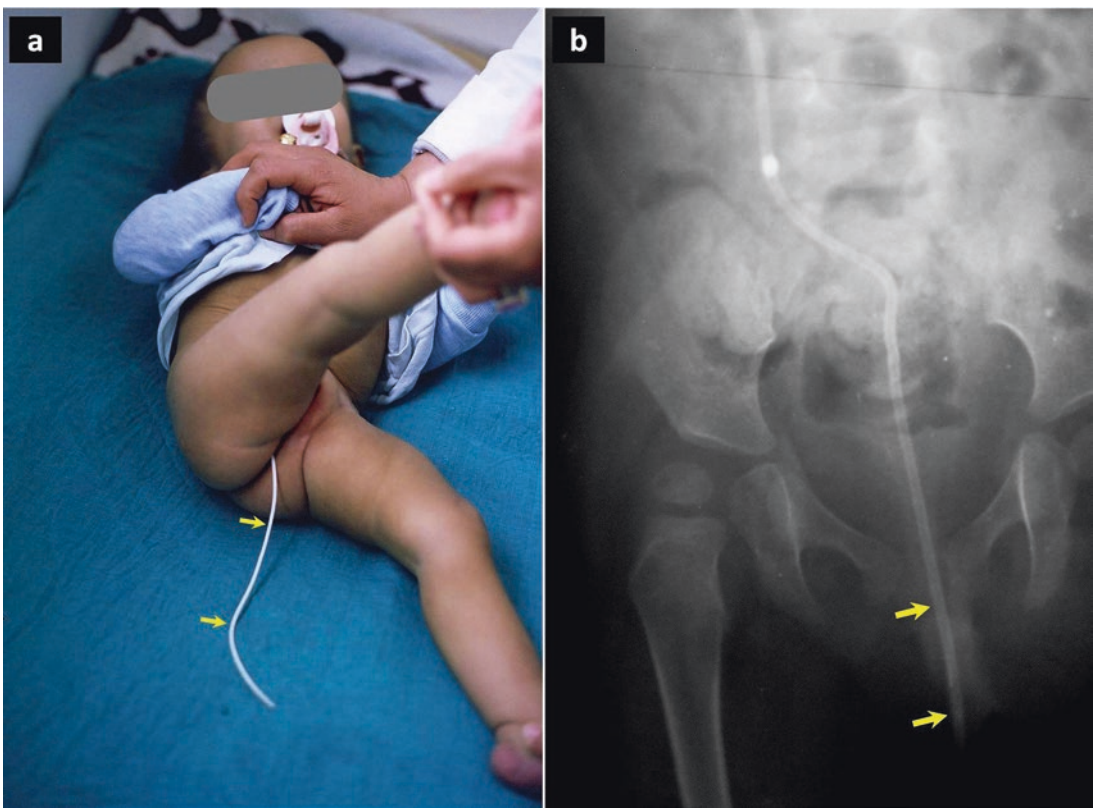


**Fig. 21.31** Extrusion of a distal shunt catheter through the anus (*arrows*) of an 18-month-old girl who presented with meningitis (**a**). The shunt had been inserted in the peritoneal cavity 6 months before. Abdominal plain radiography (**b**) shows the extraperitoneal extrusion of the peritoneal catheter (*arrows*)

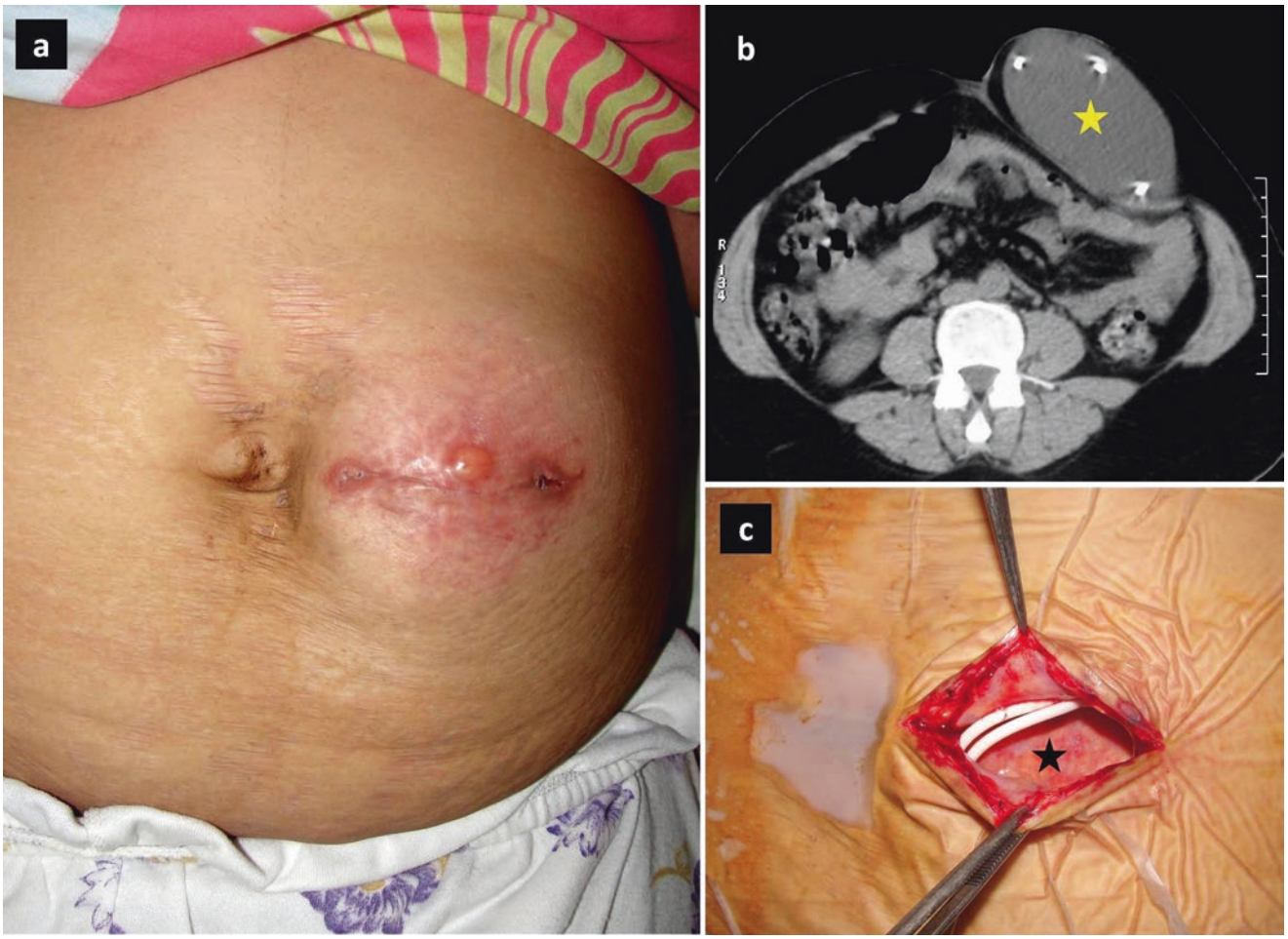


**Fig. 21.32** Erythematous paraumbilical abdominal wound with swelling (**a**). Axial abdominal CT scan showing an extraperitoneal subcutaneous pseudocyst with the distal shunt catheter in the middle of a fluid collection (**b**). Operative findings showing the subcutaneous enrolled distal catheter (**c**) within the pseudocyst formation (*star*) (Reproduced from Akhaddar (2015), p. 143, with permission)

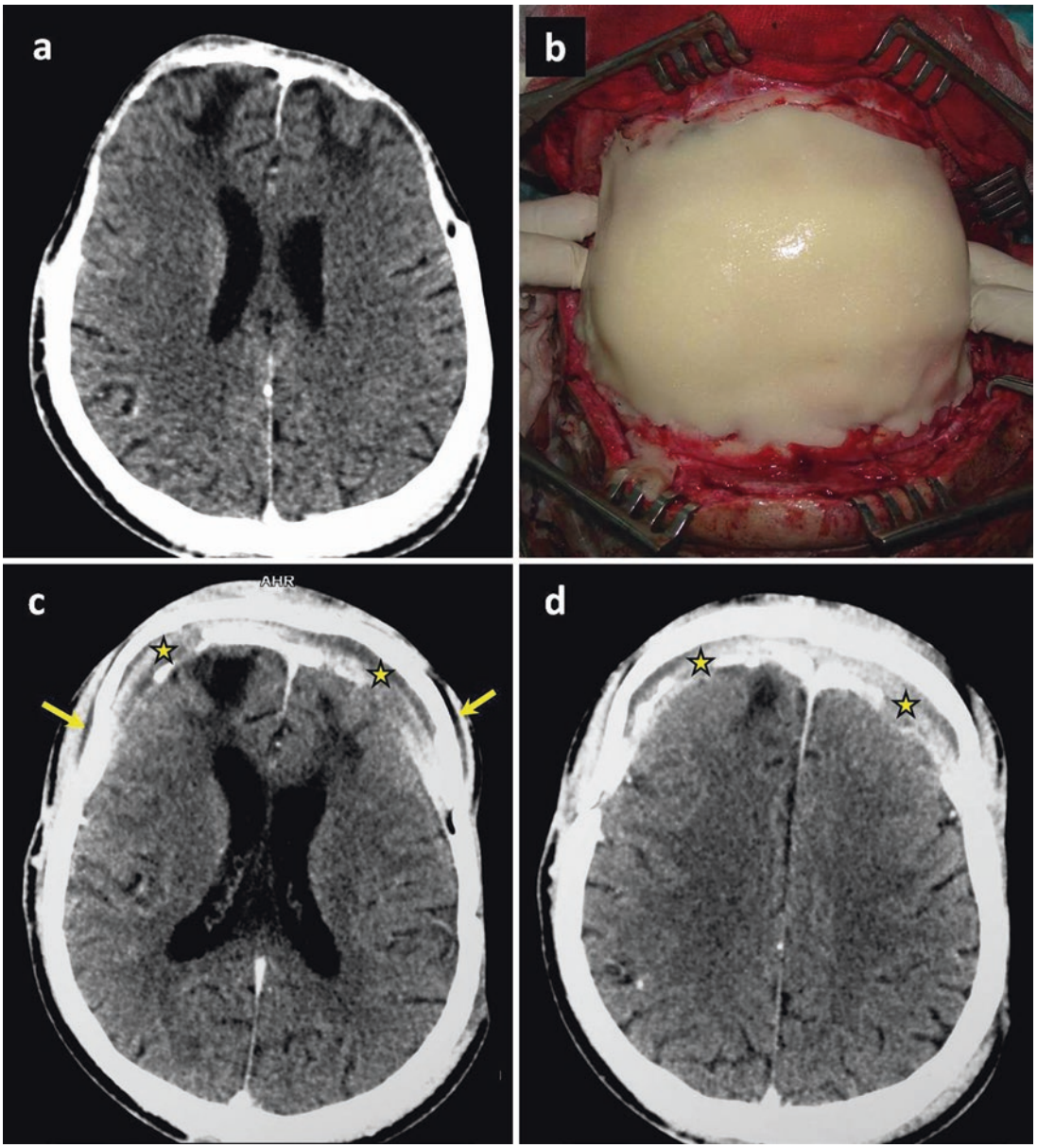


**Fig. 21.33** Axial cranial CT scan in a young patient operated on 18 months before for bifrontal decompressive craniectomy (**a**). Operative view of a frontal antibiotic-impregnated polymethyl methacrylate cranioplasty (**b**). Axial cranial-enhanced CT scans (**c**, **d**) showing extracranial suppurative collections (*arrows*) and intracranial epidural suppurative collections (*stars*) at the tenth postoperative week

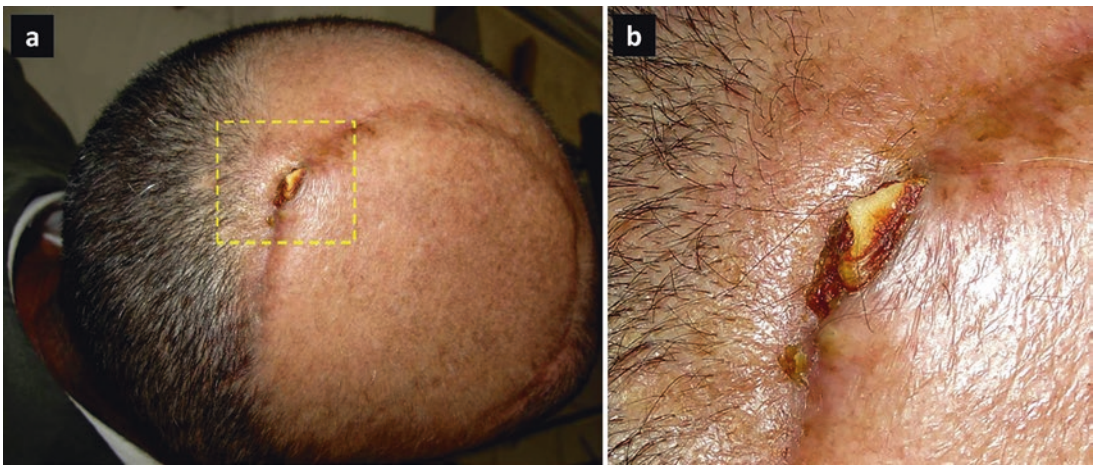


**Fig. 21.34** (**a**, **b**) Clinical pictures showing an exposed cranial acrylic plate. This patient also has contiguous cranial osteomyelitis. He was operated on 11 years before for an acute subdural hematoma, with methyl methacrylate cranioplasty

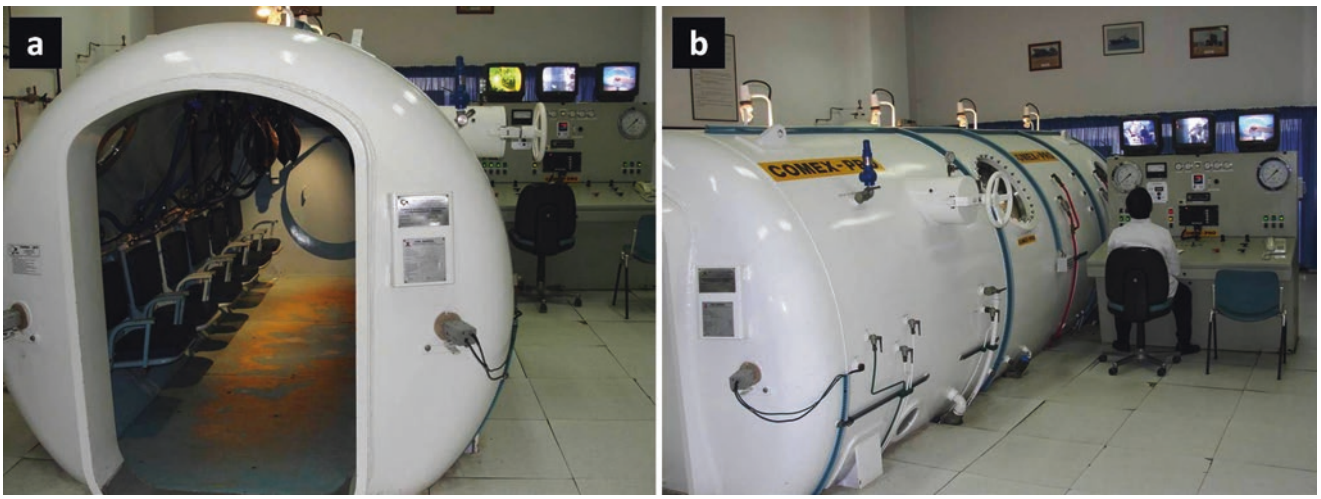


**Fig. 21.35** (**a**, **b**) Multiplace hyperbaric oxygen chamber. The chamber is pressurized with air, and the patients breathe 100% oxygen through clear hoods (Courtesy of N. El Omari, MD. Department of Hyperbaric Medicine, Mohammed V Military Teaching Hospital, Rabat, Morocco)

## Suggested Reading


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# Surgical Site Infections in Spinal Surgery

# 22

Surgical site infections (SSIs) following spinal surgery remain an important complication that requires urgent detection. Even with the use of sterile technique and antibiotic prophylaxis, a significant proportion of patients develop infection, which may require reoperation and aggressive antibiotic therapy. Classically, the SSI is divided into incisional (superficial or deep) or organ/space infections, depending on the tissue compartment concerned. Clinical symptoms vary greatly, with the most frequent being fever, spinal pain, local signs of inflammation, wound discharge, and (rarely) new neurologic deficits. CT scans and MRI offer an interesting basis for the diagnosis of spinal and intraspinal infections. Inflammatory biomarkers are highly variable in their expression; the most useful are procalcitonin and amyloid serum A levels. When possible, CSF should be assessed in cases with suspected intradural infections. Causative pathogens are most often gram-positive skin flora, particularly *Staphylococcus aureus*. Most patients with superficial wound infections and limited deeper infections are treated with local wound care and antibiotics only, but these cases require careful monitoring and should be considered for surgery if complications arise. Operative management may be indicated for drainage or dehiscence of the incision, clinical sepsis, neurologic deficits secondary to fluid collection or mass effect, a spinal or epidural abscess, or instability from bone destruction or failure of an implant or fixation. If treated quickly and vigorously, SSI may resolve without sequelae, but complications may occur, especially in patients with deep infections. Sequels consist of spinal instability and deformity, pseudarthrosis, residual neurologic deficits, and chronic spinal pain.

## Epidemiology and Etiology

SSIs following spinal surgery remain a significant cause of morbidity, prolonged hospitalization, and increased costs. Even with the use of sterile techniques and antibiotic prophylaxis, a significant proportion of patients go on to develop infection severe enough to require aggressive antibiotic therapy with or without reoperation. Postoperative infections are also a frustrating problem for surgeons faced with patient dissatisfaction and sometimes a lawsuit.

Classically, SSIs are divided into incisional (superficial or deep) infections or organ/space infections, depending on the tissue compartment concerned. A superficial incisional SSI involves the skin and/or subcutaneous tissues (Figs. 22.1, 22.2, 22.3, and 22.4). A deep incisional SSI can be contained in the fascia and muscle layers (Figs. 22.5, 22.6, 22.7, 22.8, 22.9, and 22.10). Deeper organ/space postoperative spinal infections may involve structures such as the intervertebral disc space (discitis), the vertebral bodies (spinal osteomyelitis), or the epidural space (epidural abscess) (Figs. 22.11, 22.12, 22.13, 22.14, and 22.15). Infection may affect a single location or a combination of locations.

SSI is defined as occurring within 30 days of surgery or within 12 months of placement of foreign bodies, such as spinal instrumentation. The incidence of SSI in spinal surgery is reported to be less than 14%, depending on the type of surgical procedure, the length of postoperative follow-up, and the quality of data collection. Lower rates are reported for minimally invasive approaches, anterior spinal exposures, and degenerative spine, whereas rates appear to be higher with spinal instrumentation, after complex surgical procedures, and in patients with traumatic spinal injuries or neoplastic processes.

Patient-specific risk factors for SSI include advanced age, drug and alcohol abuse, smoking, diabetes mellitus, obesity, malnutrition, long-term steroid use, and compromised immune function. Procedural risk factors are related to repeat surgery, a high number of levels fused, a posterior approach, longer operative time, blood transfusion, and increased intraoperative blood loss. Also important are postoperative circumstances such as unsatisfactory local wound care and poor patient primary healing.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_22

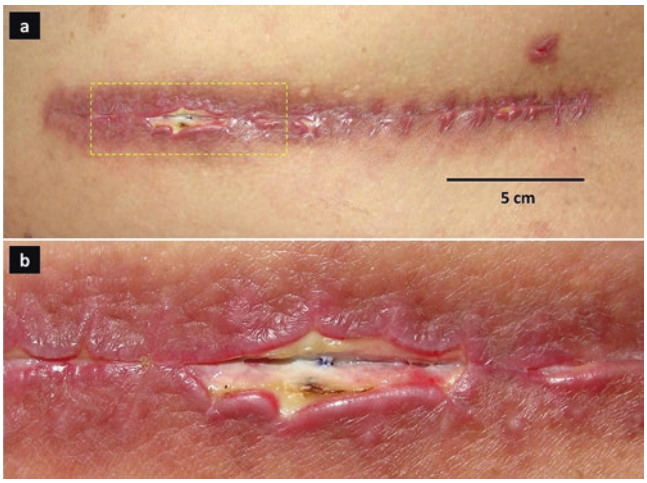


**Fig. 22.1** (**a**, **b**) This diabetic patient was operated on 3 weeks previously for dorsolumbar spinal injury. There are local signs of inflammation near the incision and superficial wound dehiscence with delayed healing

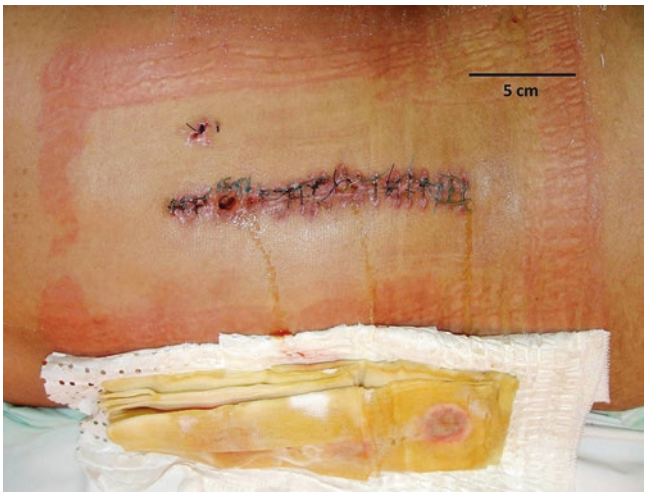


**Fig. 22.3** Case 22.1. Surgical site infection in a 16-year-old girl treated 10 days before for lumbar spinal fractures. There is delayed healing, wound dehiscence, and purulent discharge. Note the purulent dressing

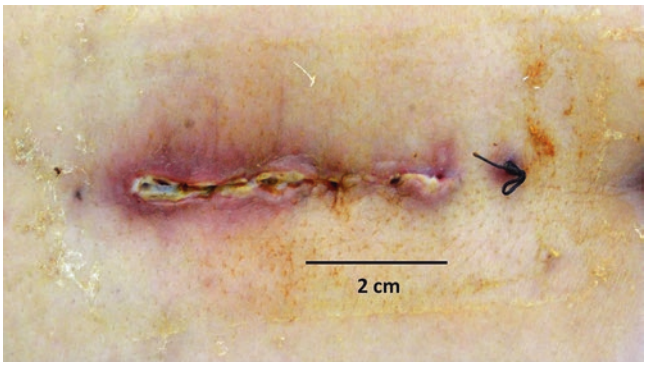


**Fig. 22.2** Postoperative wound inflammation with superficial dehiscence in a 36-year-old man operated on 10 days previously for lumbar disc herniation

## Clinical Presentations

Clinical presentations and diagnosis time vary depending on the location of infection, the causative pathogens, the patient's age and premorbid conditions, the type of surgical procedure, and the timing of infection. The most frequent clinical findings are fever, spinal pain, local signs of inflammation near the incision, wound discharge, and (rarely) new neurologic deficits. Fever is a fluctuating symptom, so the lack of fever cannot rule out the possibility of infection. Systemic findings may also be present, including chills, night sweats, anorexia, and general malaise. Some patients suffer from severe sepsis and end organ failure.

Patients with superficial incisional infections commonly present with pain and tenderness on palpation of the surgical site, local redness or swelling, and wound discharge (serosanguinous or purulent). With deep incisional infections, the presentation may be only local spinal pain and signs of inflammation, without wound dehiscence or discharge. Occasional drainage is noted, but frank sepsis is uncommon.

Cases with organ/space infections present persistent, progressive back pain at the site of operation, with limited spinal range of motion and paraspinal muscle spasms. The worsening spinal pain can radiate to the hip, leg, buttock, abdomen, or perineum but without true neurologic radiculalgia. Deep infections often lack notable superficial features.

Postoperative infections after anterior cervical procedures are rare and may be caused by pharyngeal or esophageal injuries during the surgery. In this perspective, retropharyngeal abscess is the most frequent presentation, with painful swallowing.

Focal neurologic deficits are usually absent; when they occur, epidural or rarely subdural abscesses should be suspected.

Patients with meningitis typically present with fever, neck stiffness, and altered level of consciousness. Cases with lumbar cerebrospinal fluid (CSF) shunt infection show clinical signs of acute bacterial meningitis and sometimes symptoms of shunt malfunction or obstruction.

Attention should be paid to possible concomitant infection elsewhere in the body.

## Imaging Features

Plain radiographs are the first imaging modality used when spinal infection is suspected, but negative results do not rule out infection.

Deep postoperative spinal infections should be evaluated mainly with MRI and gadolinium administration. Involved

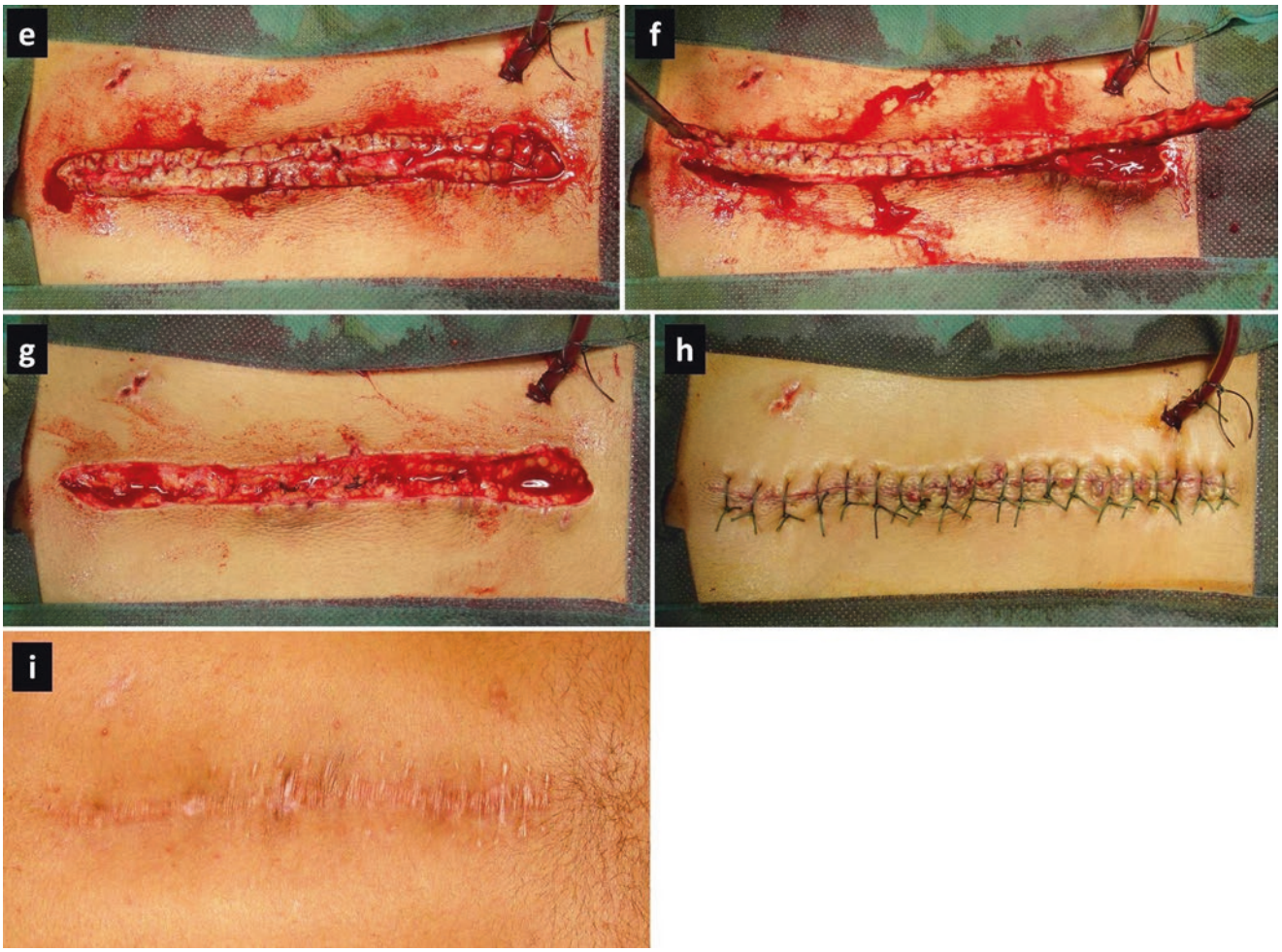


**Fig. 22.4** Case 22.1. Operative views: Wound swab procedure (**a**). Removal of surgical wires (**b**). Reopening the wound (**c**), collecting specimens (avoiding skin contamination), careful drainage and debridement, and then washing the surgical site with antiseptic products. Closing muscular fascia and subcutaneous plane (**d**). (If needed, a surgical drain may be left in place.) Removing the edges of the wound (containing purulent and nonviable skin) (**e**–**g**). Superficial wound closure (**h**). Complete healing (back photograph 3 months later) (**i**)

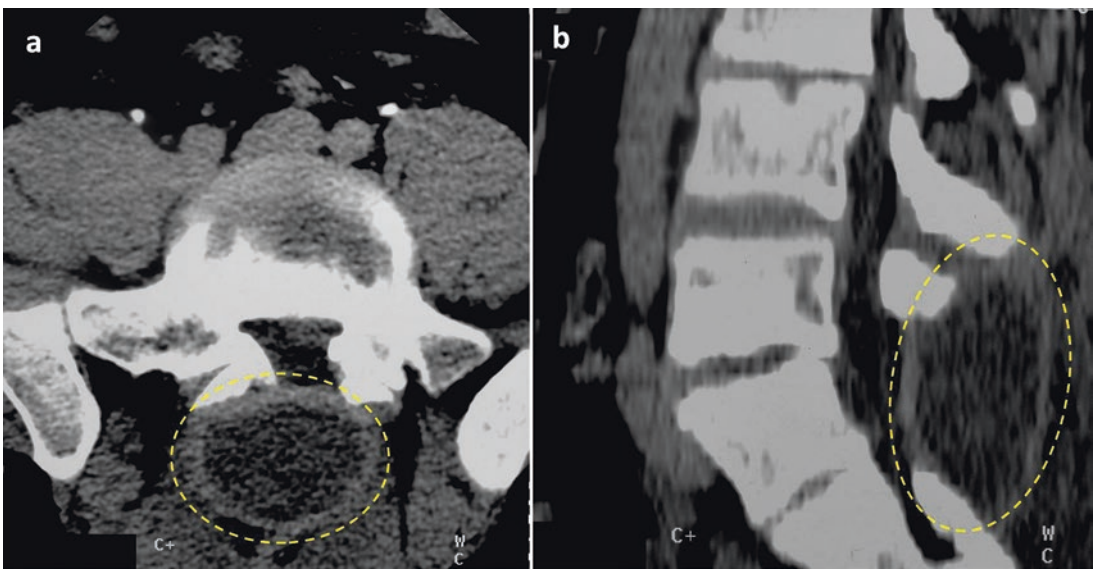


**Fig. 22.5** Axial (**a**) and sagittal (**b**) contrast-enhanced CT scan of the lumbar spine showing a postoperative posterior deep abscess collection (*dotted oval*)

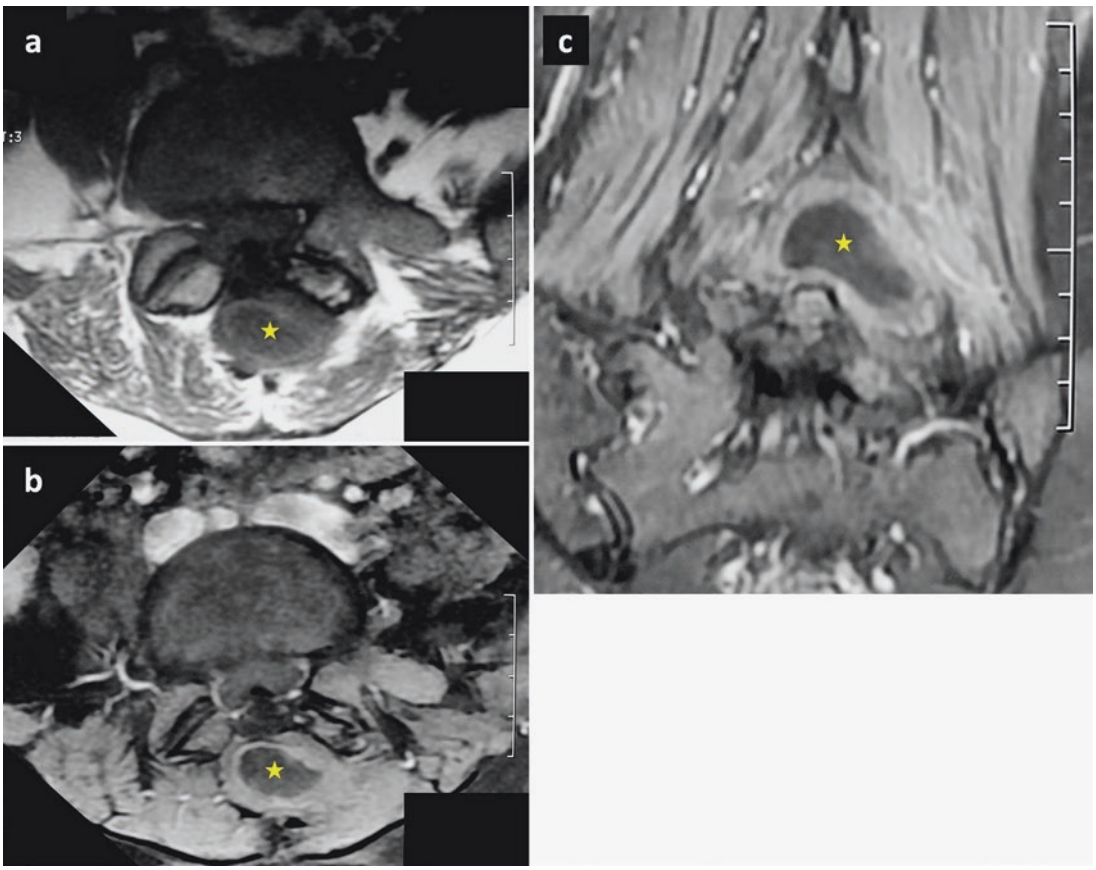


**Fig. 22.6** Postsurgical lumbar spine abscess. Axial lumbar T1-weighted images before (**a**) and after (**b**) gadolinium administration and coronal post-contrast T1-weighted image (**c**). There is a well-circumscribed, hypointense cystic lesion (*star*) with ring enhancement after gadolinium injection

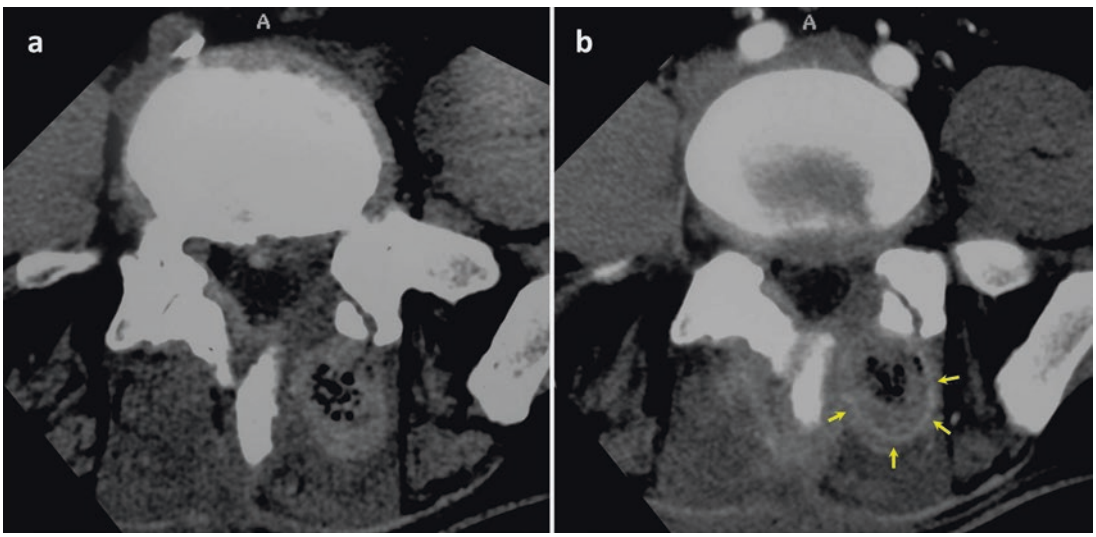


**Fig. 22.7** Case 22.2. Paraspinal textiloma after posterior lumbar surgery. Axial lumbar CT scan before (**a**) and after (**b**) contrast injection. Classic spongiform pattern of mixed mass with gas bubbles corresponding to a "textiloma." Note the ring enhancement after contrast injection (*arrows*)

tissues usually present with increased signal on T2-weighted images. Vertebral osteomyelitis and discitis have low signal intensity on T1-weighted images and high signal intensity on T2-weighted images, owing to increased edema. The inflammatory lesions will enhance following gadolinium injection, especially with involvement of the end plates and combined abscess formations.

Early in the infection process, short tau inversion recovery (STIR) sequences suggest higher signal intensity to help differentiate the infected area from the normal spine. Nuclear medicine imaging—particularly fluorodeoxyglucose positron emission tomography (FDG-PET)—is more sensitive for the early detection of suspected spondylodiscitis. MRI is also useful in demonstrating spinal canal lesions, especially associated spinal epidural abscess and spinal cord compression.

Vertebral osteomyelitis is best evaluated on CT scans. The most frequent findings are narrowing of the disc space and erosion of the adjacent end plates. The existence of haloing (lysis) around implants is suggestive of hardware failure secondary to associated infection. Suppurative collections have a low density on CT scans, with surrounding enhancement following contrast administration. Rarely, a paraspinal textiloma may be suspected when a spongiform pattern with gas bubbles is shown. CT guidance can also be used to obtain a tissue biopsy of postoperative discitis or osteomyelitis or for needle aspiration of pus from abscess cavities to obtain a microbiological diagnosis.

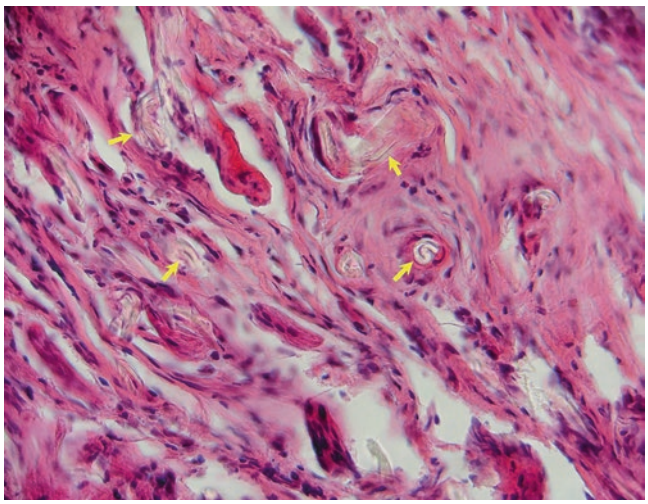

**Fig. 22.8** Case 22.2. Histopathologic features of a "textiloma": Foreign body granuloma containing polynuclear and multinucleated giant cells around the fibers cut in longitudinal and cross sections (*arrows*) (medium-power magnification, hematoxylin–eosin staining)

## Laboratory Findings

Routine inflammatory biomarkers (erythrocyte sedimentation rate, C-reactive protein, and white blood count) are highly variable in the postoperative period and are nonspecific. SSI should not be excluded in a patient with no fever, normal white blood count, abnormal ESR, and a negative blood culture. Procalcitonin levels are more specific and helpful in the monitoring of spinal infection, but they also lack sensitivity. The serum amyloid A (SAA) level is considered a better inflammatory marker in the assessment of SSI following spine surgery.

When possible, CSF should be evaluated in patients with suspected postsurgical intradural infections. Disturbance of protein/glucose levels and white blood cell count may be suggestive, but only gram staining and CSF culture will establish the diagnosis.

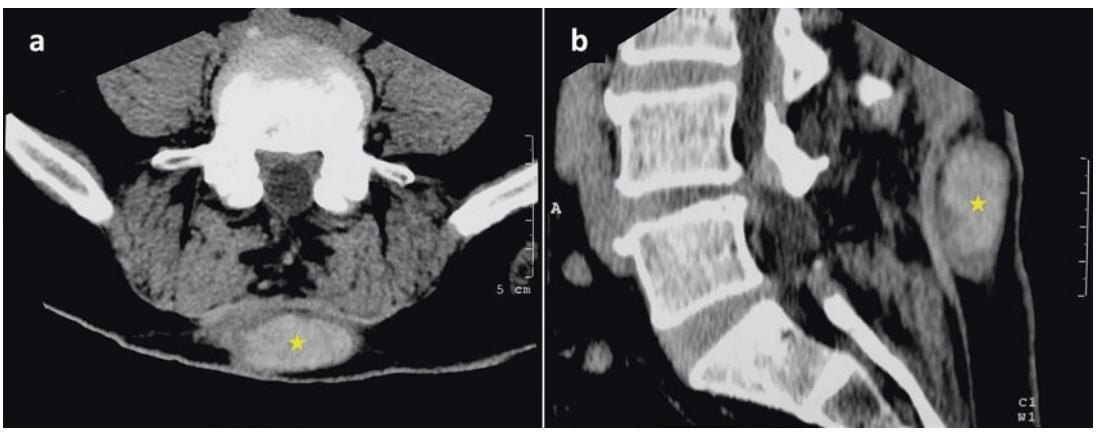


**Fig. 22.9** Axial (**a**) and sagittal (**b**) lumbar spinal CT scan. Postoperative superficial surgical site infection (subcutaneous abscess formation, *star*) following lumbar disc herniation surgery

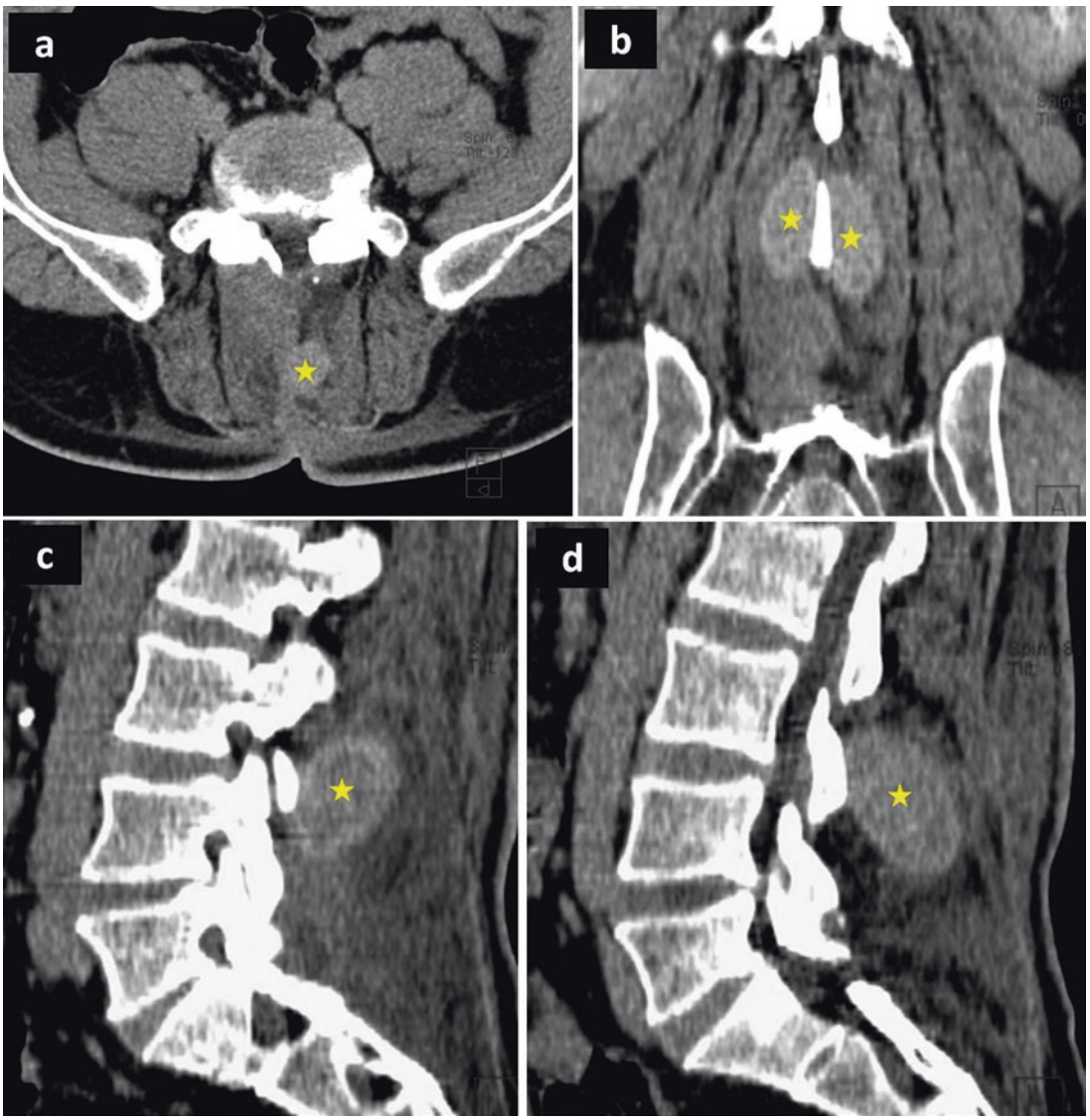


**Fig. 22.10** Axial (**a**), coronal (**b**), and sagittal (**c**, **d**) lumbar spinal CT scans showing deep surgical site infection (abscess formation on both side of the spinous process, *stars*) following posterior lumbar spine surgery

The causative pathogens most often found in SSI following spinal surgery are gram-positive skin flora, especially *Staphylococcus aureus* and *Staphylococcus epidermidis*, though *Enterococcus faecalis* and *Enterobacter cloacae* are not unusual. *Klebsiella pneumoniae*, *Escherichia coli*, *Pseudomonas aeruginosa*, and *Proteus* species are the most common gram-negative isolates from postoperative wound spinal infections. Fungal and mycobacterial infections are rare but should always be considered.

## Treatment Options

Medical therapy starts with broad-spectrum gram-positive and gram-negative coverage, which is modified on the basis of culture results and antimicrobial susceptibility testing. Patients with deep SSI are primarily treated with 3–6 weeks of parenteral antibiotics, often followed by 2–4 months of oral antibiotics. Most superficial wound infections are treated with local wound care and oral antibiotics for an average of

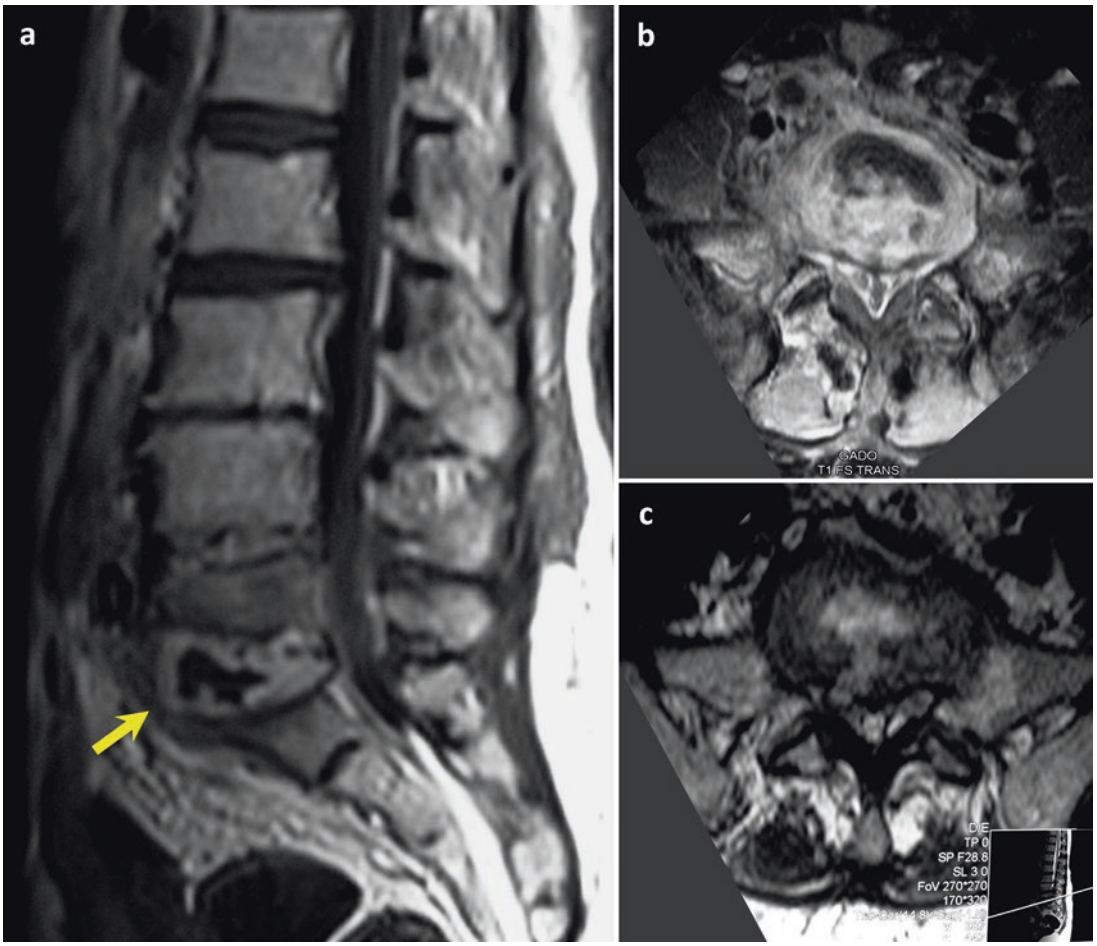


**Fig. 22.11** Sagittal (**a**) and axial (**b**) lumbar spinal T1-weighted MR images with gadolinium administration and axial T2-weighted image (**c**). Postoperative spondylodiscitis on L5–S1 level (*arrow*) 1 month following surgery for a lumbar herniated disc

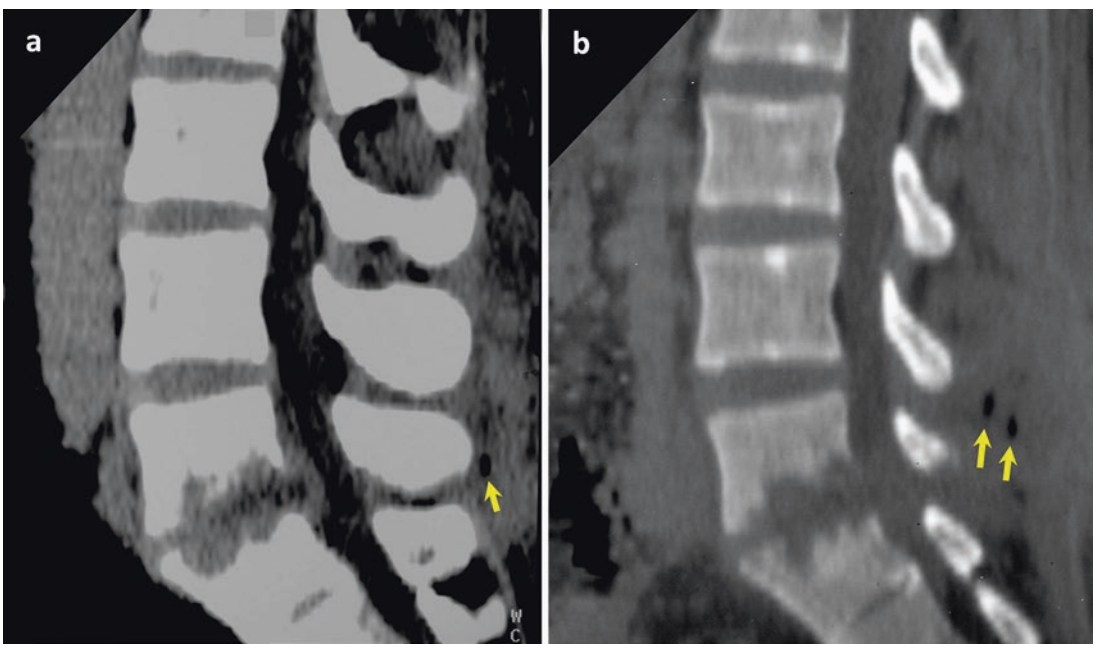


**Fig. 22.12** Case 22.3. Sagittal lumbar spinal CT scan without contrast injection on parenchymal (**a**) and bone (**b**) windows. Postoperative spondylodiscitis on L5–S1 level, shown 5 weeks after surgery for lumbar herniated disc. Note the gas bubbles along the surgical approach (*arrows*)

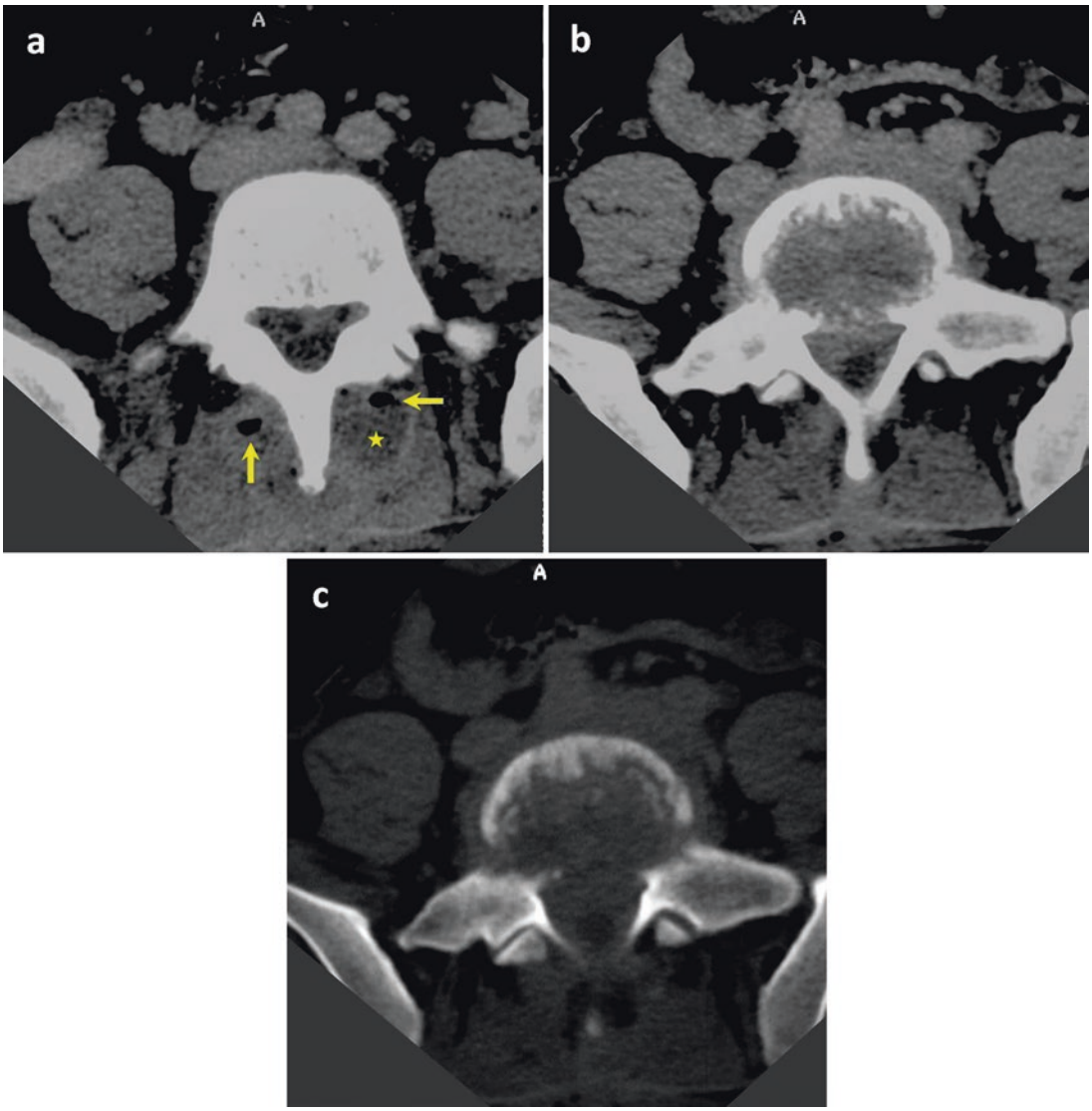


**Fig. 22.13** Case 22.3. Axial lumbar spinal CT scan without contrast injection on parenchymal (**a**, **b**) and bone (**c**) windows. There is a posterior paraspinal abscess on the left side (*star*) with bilateral air bubbles (*arrows*)

only 2–3 weeks. The exact duration of antimicrobial treatment is based on clinical, laboratory, and imaging responses. Nonoperative patients should be observed carefully, with frequent evaluation and surgical intervention when necessary. External spinal immobilization (bracing), strict bed rest, and the use of a corset during activity should be indicated for patients with deep SSI that is managed without surgery.

Operative management may be indicated for patients with drainage or dehiscence of the incision, clinical sepsis, neurologic deficits secondary to fluid collection or mass effect, a spinal or epidural abscess, and instability from bone destruction or failure of an implant or fixation. The goal of surgery is to debride all necrotic and nonviable tissue and then stabilize the spine to prevent deformity and/or neurologic injury. A surgical drain should be used as soon as possible before closure.

When postoperative infection occurs in the setting of spinal instrumentation, there is no clear consensus about whether to remove the hardware (Figs. 22.16, 22.17, 22.18, 22.19, and 22.20). Most authors have suggested retention of all stable hardware and primary replacement of instrumentation if fixation failure has occurred. Further debridements are performed if clinical evidence of uncontrolled infection continues.

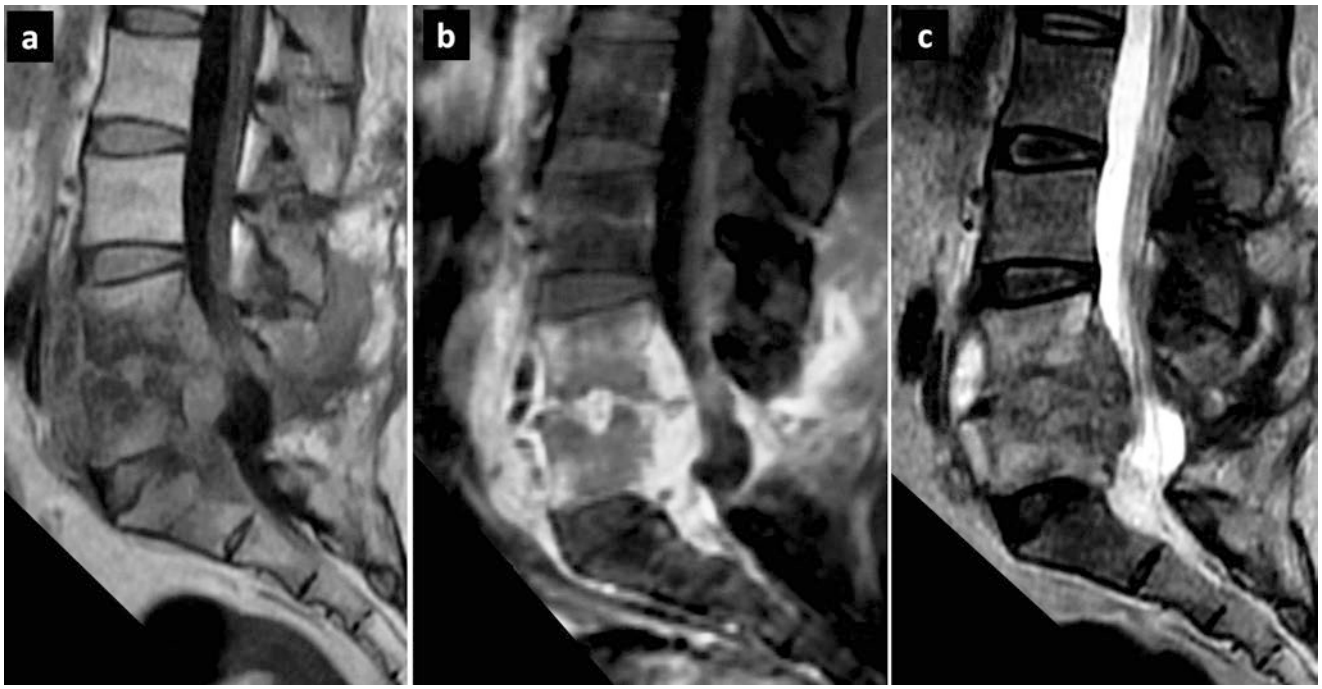


**Fig. 22.14** Case 22.4. Sagittal lumbar spinal T1-weighted MR images before (**a**) and after (**b**) gadolinium injection and T2-weighted image (**c**) showing a postsurgical L4–L5 spondylodiscitis with intracanalar and paraspinal extension

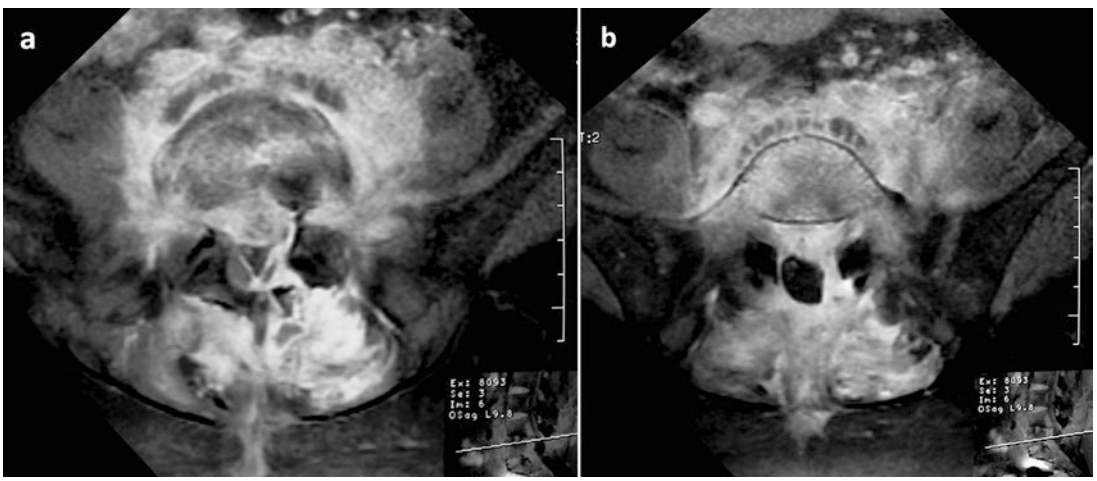


**Fig. 22.15** Case 22.4. (**a**, **b**) Axial enhanced T1-weighted images of the lumbar spine. Note the significant extensive enhancement of the inflammatory tissues following gadolinium administration

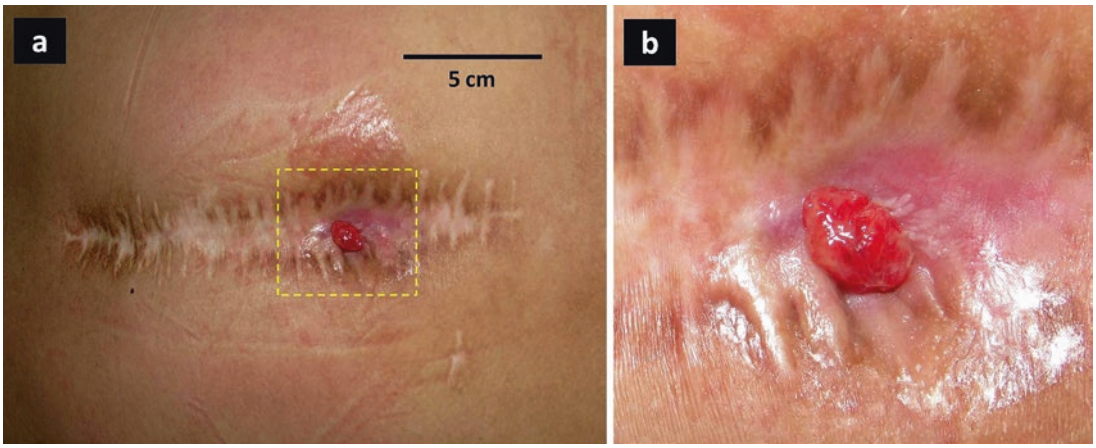


**Fig. 22.16** Case 22.5. (**a**, **b**) Recent suppurative wound fistula (intermittent purulent drainage) in a paraplegic patient 4 years after surgery for thoracic spinal cord injury with posterior instrumentation

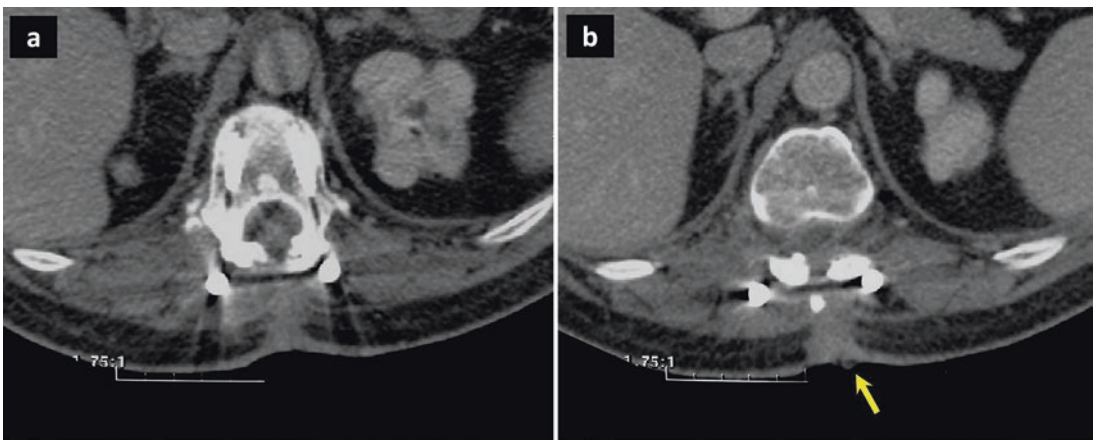


**Fig. 22.17** Case 22.5. (**a**, **b**) Axial spinal CT scan on bone windows through the T11 vertebral level, showing the location of the cutaneous fistula deeply (*arrow*)

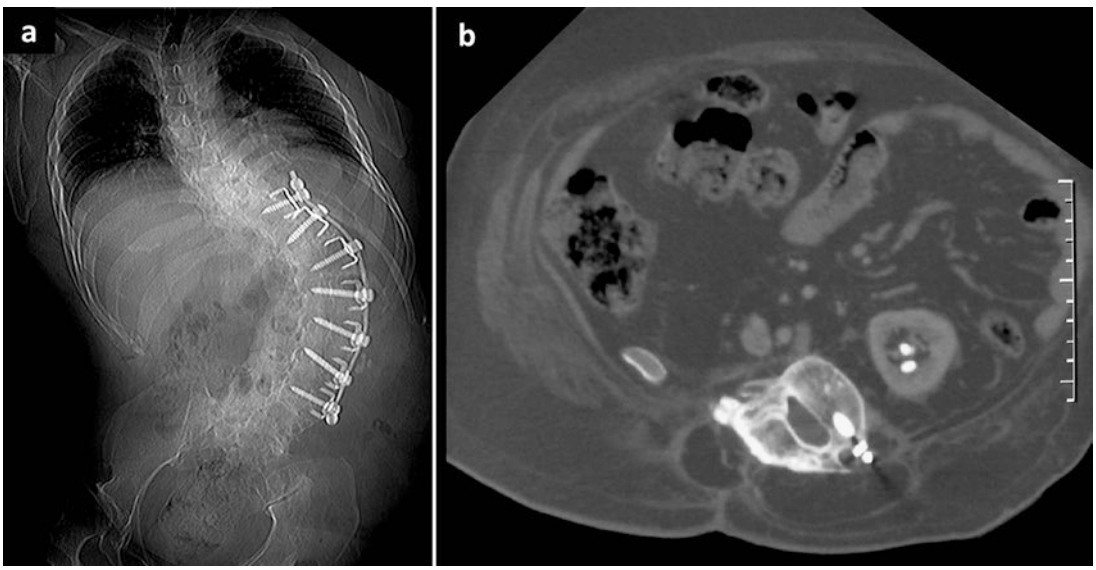


**Fig. 22.18** Case 22.6. Frontal spinal plain radiography (**a**) and axial spinal lumbar CT scan on bone window (**b**). This 48-year-old paraplegic patient was operated on more than 30 years previously for a dorsolumbar neurogenic scoliosis, using Dwyer spinal instrumentation

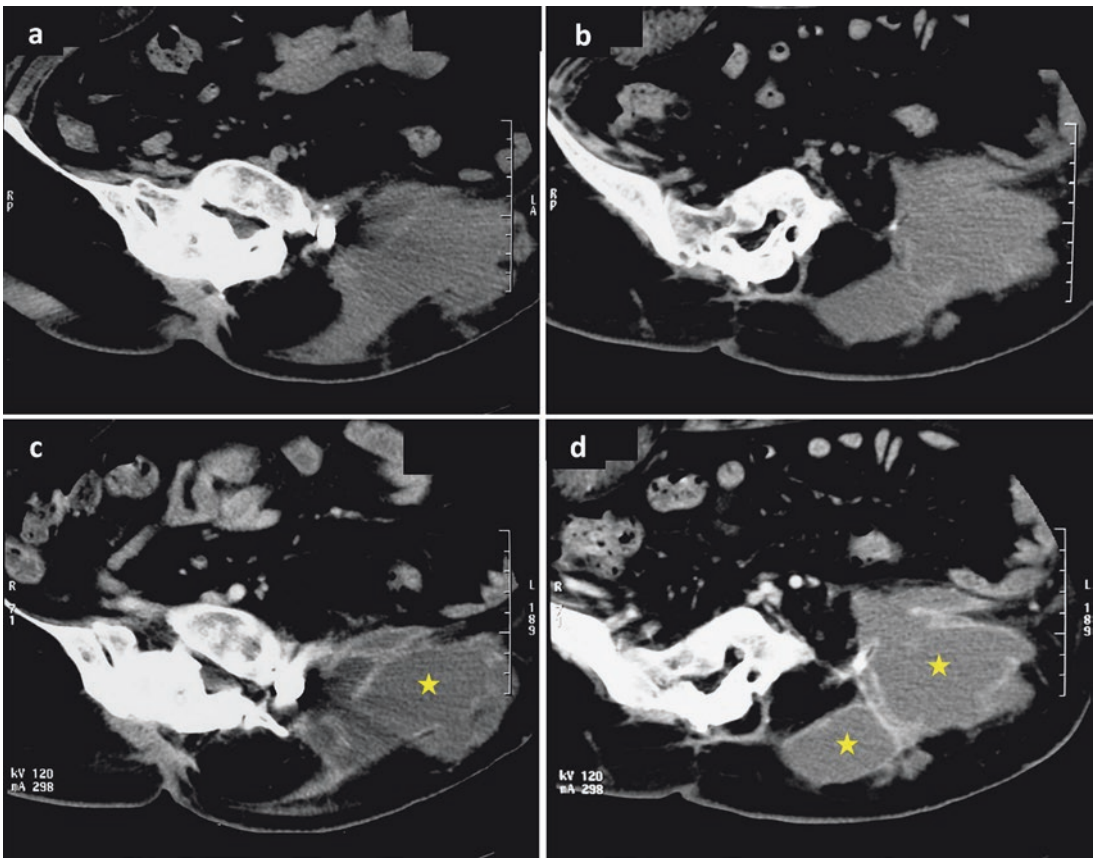


**Fig. 22.19** Case 22.6. The patient presented with a large lumbar paraspinal swelling in the left side with fever. Axial spinal lumbar CT scans before (**a**, **b**) and following (**c**, **d**) contrast administration reveal a subcutaneous, multiloculated abscess formation (*stars*), which extends deeply into the paravertebral and retroperitoneal spaces

With large wound defects, specific reconstruction performed by a plastic surgeon may be required. In some refractory cases, vacuum-assisted devices or hyperbaric oxygen therapy may be helpful.

Supportive therapy is often needed, including fluids, analgesics, antipyretics, anticoagulation, and functional rehabilitation. Some patients with sepsis and even septic shock may need initial resuscitation and time in the intensive care unit.

If a CSF fistula is diagnosed, it should be managed surgically to prevent any form of SSI, especially postoperative meningitis.

Strict awareness of hygiene with careful application of preoperative and postoperative protocols decreases spinal SSIs.

## Outcomes

If treated rapidly and vigorously, SSI may resolve without sequelae. But many complications may occur, depending on the type and location of infection, the age and the underlying condition of the patient, the type of surgical procedure, the time to diagnosis, and the response to treatment.

Inadequate or inappropriate treatment can cause recurrences and extensive infections. The rate of neurologic morbidity and mortality is higher for patients with deep infections than for those with superficial ones. The mortality rate of spinal SSI is less than 1.4%.

Sequelae consist of spinal instability and deformity, pseudarthrosis, residual neurologic deficits, and chronic spinal pain.

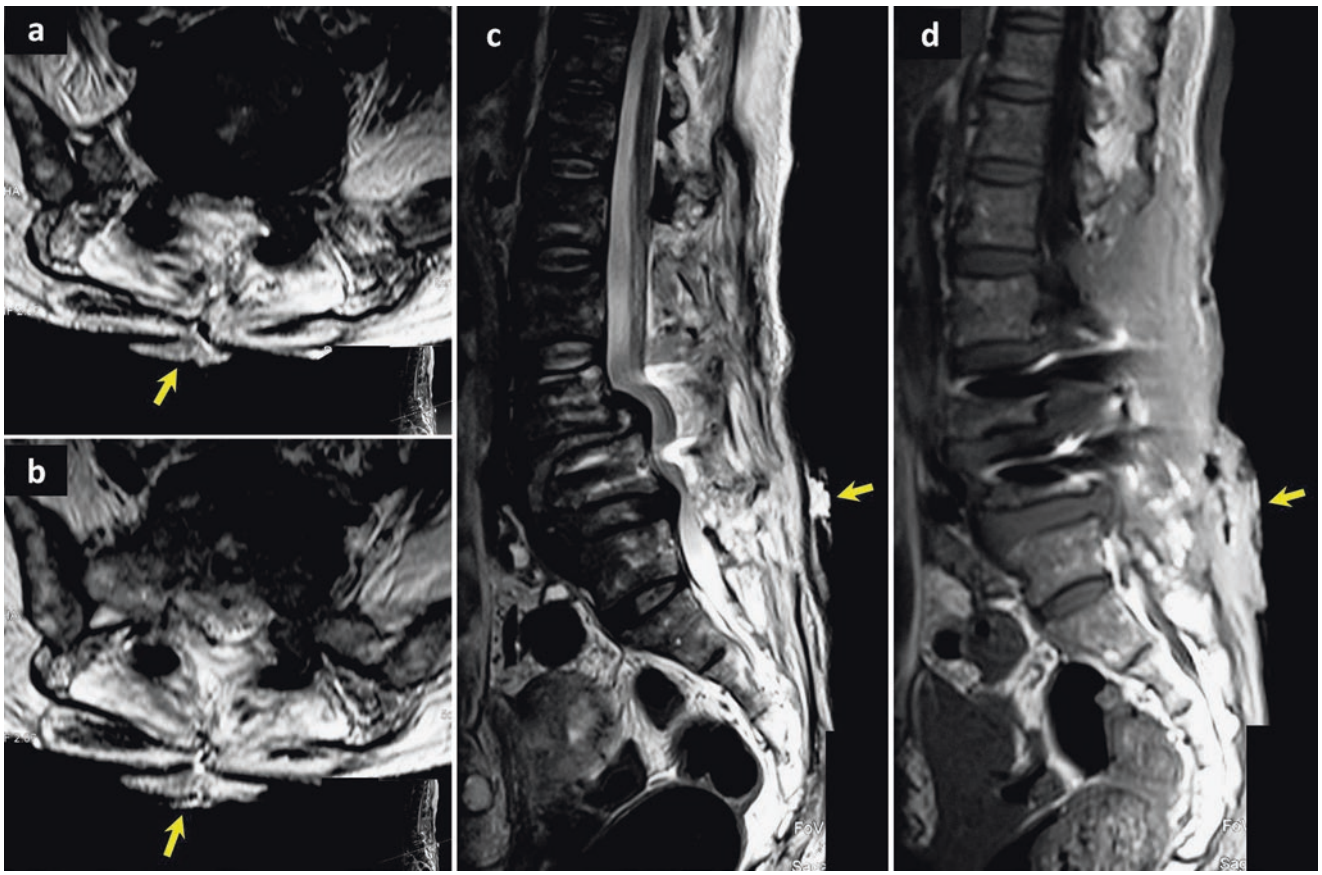


**Fig. 22.20** Axial (**a**, **b**) and sagittal (**c**) spinal lumbar T2-weighted MR images, and sagittal T1-weighted image (**d**) revealing a posterior midline cutaneous fistula (*arrow*) in a 58-year-old diabetic woman operated on 3 months previously for a spondylolisthesis using posterior spinal instrumentation at the L3–L4 vertebral levels. The fistula has occasional purulent drainage

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# Part V

## Specific Pathogens and Other Particular Conditions

# Brain Tuberculomas

# 23

Brain tuberculoma is a serious form of extrapulmonary tuberculosis. The incidence of the disease is increasing in developed nations largely because of immigration and epidemic HIV disease. Brain tuberculoma may be solitary or multiple, with or without tuberculous meningitis. Tuberculoma is often infratentorial in children and supratentorial in adults. It progresses slowly and insidiously, as a mass lesion. Dominant clinical manifestations include seizure, intracranial hypertension, and focal neurologic deficits. CT scan and MRI habitually demonstrate peripheral or uniform contrast-enhancing lesions with extensive perifocal edema. MRI spectroscopy and diffusion-weighted imaging may be helpful in making the diagnosis. Routine laboratory investigations are nonspecific. Etiological confirmation is frequently made either by demonstration of acid-fast bacilli in a pathology specimen or histological evidence of epithelioid–giant cell granulomas with caseous necrotic material. Stereotactic procedures have improved the safety of brain biopsy, and newer techniques are helpful for early and rapid diagnosis. Management of brain tuberculoma mainly involves long-term antituberculous drugs, but surgery may be required to obtain a definitive diagnosis, to relieve mass lesions or high intracranial pressure, or for CSF diversion. Most patients with a solitary tuberculoma sensitive to antituberculous therapy have a good outcome. Neurologic sequelae sometimes encountered include seizures, focal neurologic deficits, aphasia, blindness, and cognitive impairment. Drug-resistant infections are associated with a high mortality rate.

## Epidemiology and Etiology

Tuberculosis is common in many developing nations (especially in Southeast Asia and Africa), and tuberculous infections are also being seen more regularly in developed countries as a result of immigration and the growth of the immunocompromised population. About 14% of patients with new cases of tuberculosis registered worldwide are HIV positive. The causative pathogen of tuberculosis is mainly *Mycobacterium tuberculosis*, an acid-fast bacterium, which is uncommon in extrapulmonary organs.

Central nervous system (CNS) involvement (10–15% of all tuberculous infections) manifests primarily as meningitis or meningoencephalitis. Space-occupying lesions, abscesses, and especially tuberculomas are uncommon but are associated with high morbidity and mortality rates. Brain tuberculoma can be solitary or multiple and may or may not be associated with meningitis. It may occur in any part of the brain but is most likely in the posterior fossa in children and in the supratentorial compartment in adults (Figs. 23.1, 23.2, 23.3, 23.4, 23.5, 23.6, 23.7, 23.8, 23.9, 23.10, 23.11, and 23.12).

As with other forms of tuberculous disease, predisposing factors for the development of brain tuberculoma include an individual or family history of tuberculosis, poverty, malnutrition, alcoholism, drug abuse, imprisonment, diabetes mellitus, advanced age, immunosuppressive treatment, HIV infection, and chronic renal failure. Genetic factors in the bacteria and the host play a crucial role in the pathogenesis of CNS tuberculosis.

## Clinical Presentations

A previous clinical history of exposure to tuberculosis may be suggestive for the diagnosis.

Brain tuberculoma progresses slowly and insidiously, as a mass lesion. Symptoms and signs are heterogeneous, depending on the variable location, size, number, and extension of this granulomatous lesion. The most common presenting manifestation of brain tuberculoma is seizure, intracranial hypertension (increasing headache, vomiting, visual loss, diplopia, and disturbance of consciousness), and focal neurologic deficits. Other unusual clinical findings include



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_23

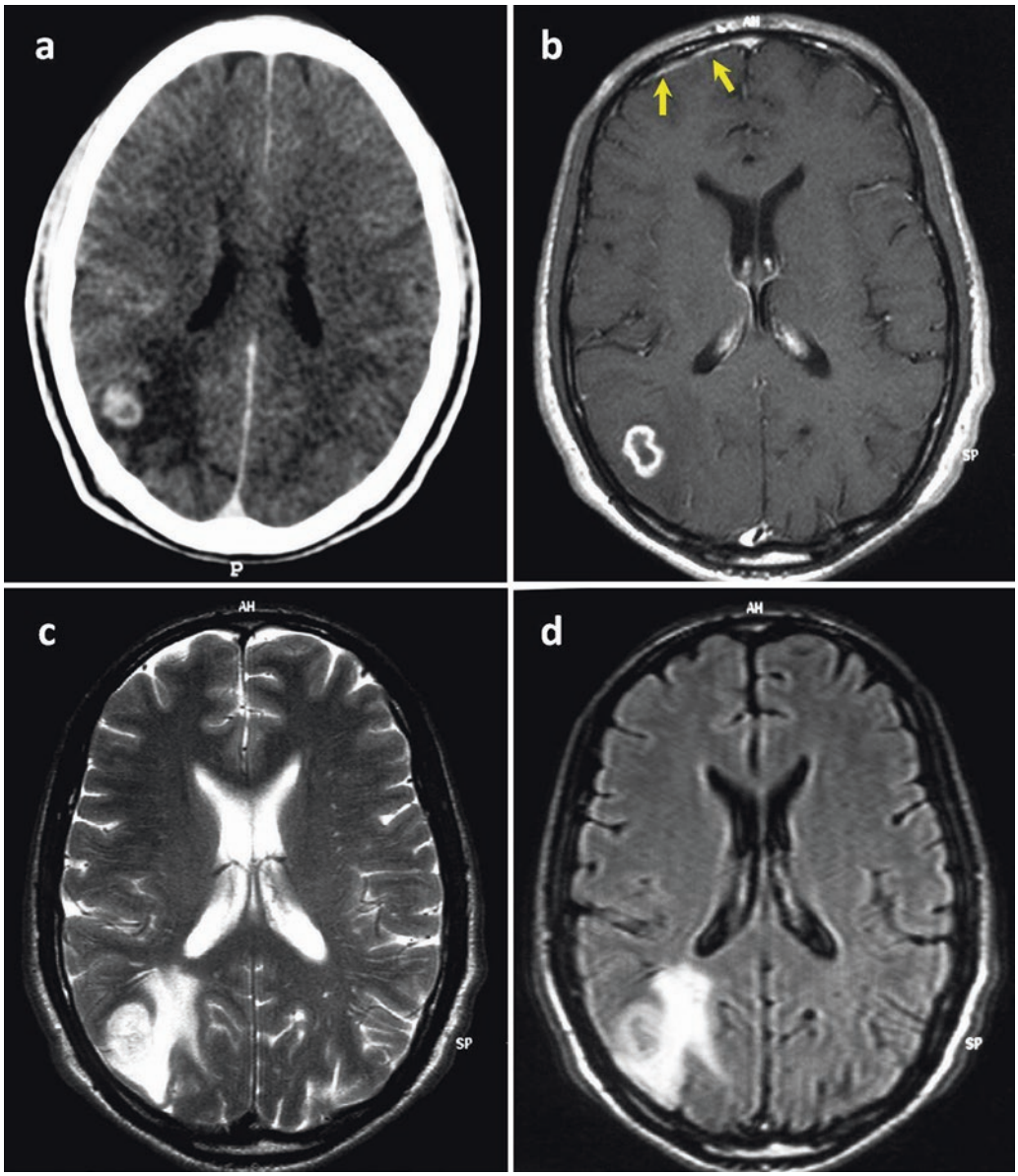


**Fig. 23.1** Case 23.1. Solitary brain tuberculoma. Axial cranial CT scan with contrast administration (**a**), post-gadolinium T1-weighted MR image (**b**), T2-weighted image (**c**), and FLAIR sequence (**d**). This granulomatous lesion was enhanced following contrast injection, with perifocal edema located in the right parietal region. Note the concomitant frontal meningeal enhancement (*arrows*)

nuchal rigidity, movement disorders, hypopituitarism, and brainstem syndrome.

Some patients may manifest systemic symptoms such as fever, chills, night sweats, anorexia, weight loss, and general malaise. A careful clinical search should be done to look for concomitant extraneural disease, especially pulmonary or lymphadenopathy tuberculosis.

The advanced stages of brain tuberculoma are characterized by deep coma, hemiplegia or paraplegia, decerebrate posturing, deterioration in vital signs, and even death. The clinical presentation in the immunocompromised patient may be overlooked because of the diminished inflammatory response.

## Imaging Features

CT scans and MRI often demonstrate the typical features of contrast-enhancing lesions with extensive perifocal edema. Indeed, tuberculoma can have a disproportionate mass effect compared with its own size, owing to significant surrounding brain edema. The contrast enhancement may be either

peripheral or uniform. MRI is more sensitive and specific than CT scans to identify small lesions and those in the infratentorial area, but the more specific "target sign" (a central nidus of calcification) is best viewed on CT scan (Figs. 23.13, 23.14, 23.15, 23.16, 23.17, and 23.18). In children, tuberculomas are generally located in the posterior fossa. Patients with concomitant tuberculous meningitis may have thick, enhancing basal exudates with infarction (especially in the internal capsule, basal ganglion, and thalamic regions) and/or ventriculomegaly.

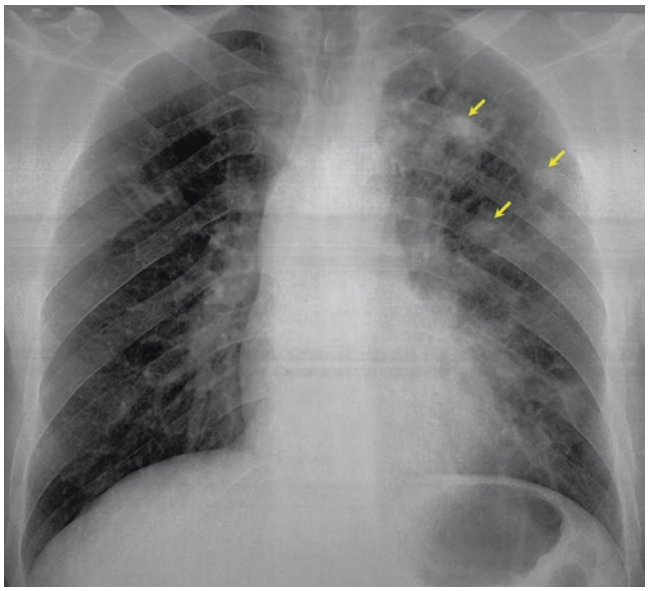

**Fig. 23.2** Case 23.1. This patient also had multiple pulmonary tuberculomas (*arrows*) on chest X-ray

Brain tuberculomas may resemble other ring-enhancing brain lesions (as listed in Table 8.1), especially pyogenic brain abscess, other granulomatous lesions, or tumors. In HIV patients, a variety of infections and malignancies that may cause brain mass lesions should be considered, including toxoplasmosis, progressive multifocal leukoencephalopathy, cryptococcosis, and lymphoma (*see* Chap. 30, especially Fig. 30.15, an image of HIV-associated cerebellar tuberculomas).

MR spectroscopy (MRS) and diffusion-weighted imaging (DWI) may be helpful in making a noninvasive diagnosis of tuberculoma. On DWI, tuberculoma appears as a lesion with low signal intensity. On MRS, tuberculomas usually show large lipid peaks with increased choline (Cho) levels and decreased levels of NAA and creatinine (Cr). A Cho/Cr ratio greater than 1 often supports the presence of brain tuberculoma. Tuberculomas also reveal an important decrease in the NAA/Cr ratio and a small decrease in the NAA/Cho ratio.

Further techniques for imaging of the chest, abdomen, and pelvis may confirm an extraneural focus of tuberculosis.

## Laboratory Findings

Routine laboratory investigations (elevated erythrocyte sedimentation rate or C-reactive protein level and leukocytosis) are nonspecific. Tuberculin skin testing is highly variable;

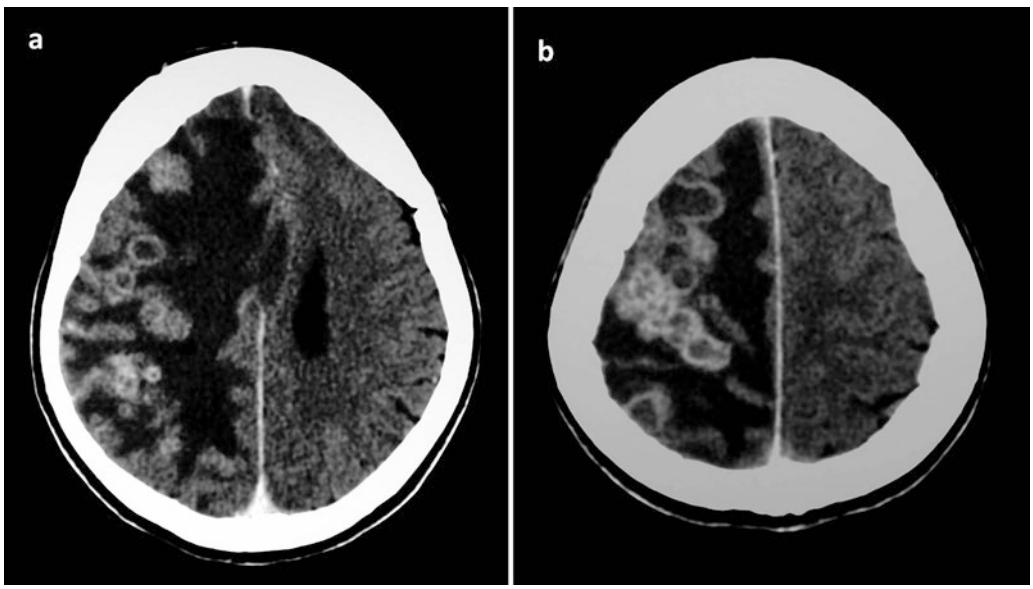


**Fig. 23.3** Case 23.2. (**a**, **b**) Axial contrast-enhanced CT scans of the brain showing multiple right frontoparietal tuberculomas with surrounding edema

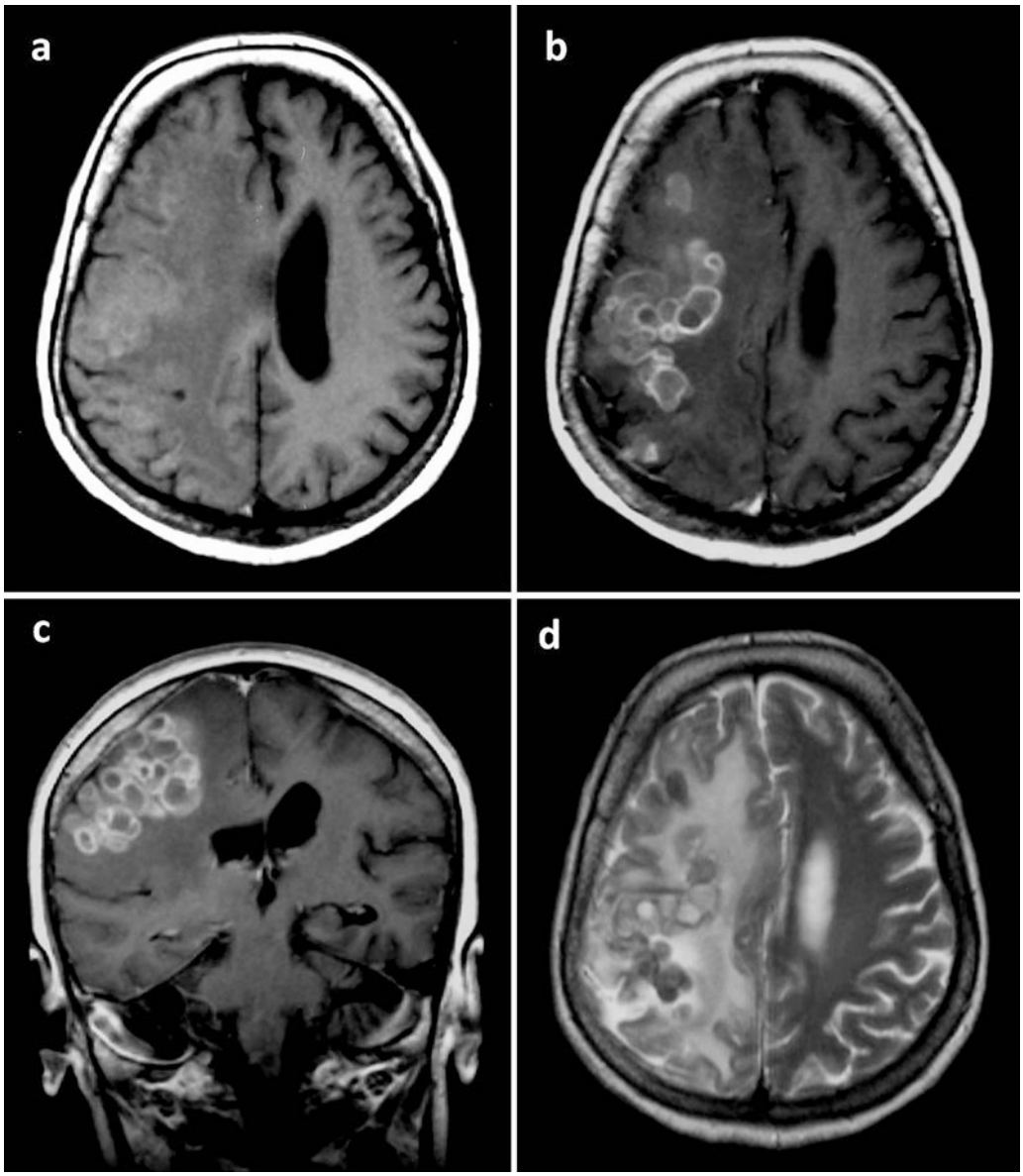


**Fig. 23.4** Case 23.2. Axial T1-weighted MR images before (**a**) and after (**b**) gadolinium administration, coronal view (**c**), and axial T2-weighted image (**d**). Showing multiple tuberculous granulomas with extensive peripheral edema and ventricular/midline mass effect

false negativity should be read with prudence in immunocompromised populations.

Cerebrospinal fluid (CSF) examination in the absence of features of tuberculous meningitis may reveal an elevated protein level in most patients, sometimes with moderate pleocytosis. However, acid-fast bacilli are less usually found in CSF, compared with those who have tuberculous meningitis. Lumbar puncture is generally not recommended in the presence of intracranial hypertension.

Cultures from other potential sources of infection (such as sputum, gastric fluid, urine, and bone marrow) may be helpful in detecting extraneural tuberculosis. Tissue examination is often required to confirm the diagnosis, and stereotactic procedures (using a frame or frameless systems) have improved the safety of brain biopsy.

Diagnosis confirmation is often made either by demonstration of acid-fast bacilli on pathological specimen or histological evidence of epithelioid–giant cell granulomas with mononuclear cell infiltration and caseous necrotic material in a biopsy specimen. Except in tuberculous brain abscess, it is difficult to find *Mycobacterium tuberculosis* on conventional microbiological methods like Ziehl–Neelsen staining. In addition, cultures on Lowenstein–Jensen media take 6–8 weeks for the growth to appear.

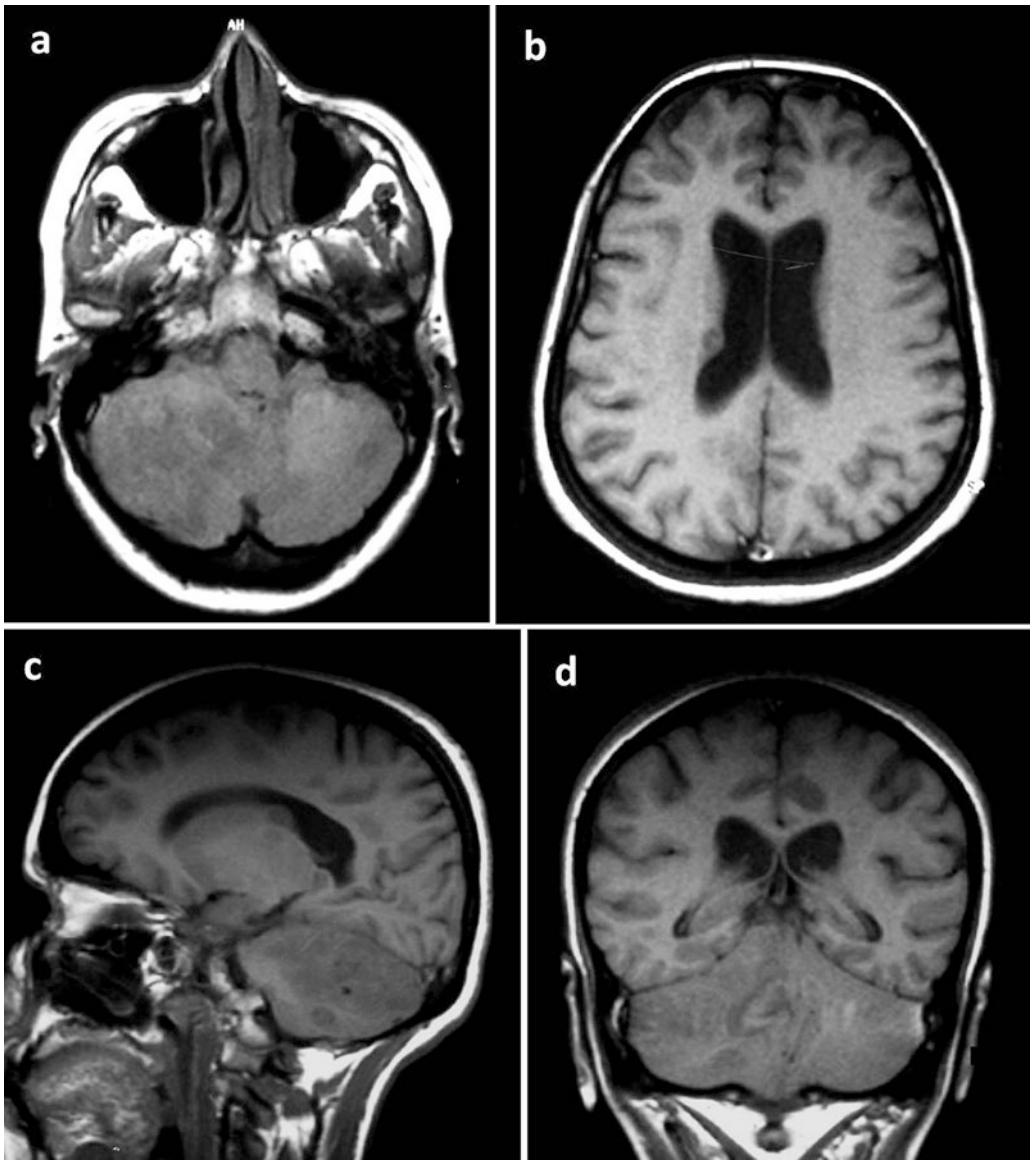


**Fig. 23.5** Case 23.3. Brain MRI, using T1-weighted images without gadolinium enhancement: axial (**a**, **b**), sagittal (**c**), and coronal (**d**) views. There are multiple infratentorial and supratentorial small lesions (tuberculomas)

DNA amplification techniques such as polymerase chain reaction (PCR) and the QuantiFERON®-TB Gold In-Tube assay have shown very promising results for the early and rapid diagnosis of CNS tuberculosis. The presence of mixed bacteria or fungi is rare but should always be considered.

## Treatment Options

Management of brain tuberculoma primarily involves medical therapy in the form of long-term antituberculous drugs. Some patients do require surgery, however.

The first-line regimen is a combination of isoniazid, rifampicin, pyrazinamide, and ethambutol, or streptomycin for 2 months, followed by two drugs (isoniazid and rifampicin) for 7–10 months. Antituberculous therapy in HIV-infected patients is the same as for uninfected patients.

In some cases, drug-related toxicity may require changes to the drugs used, and the emergence of a new strain of drug resistance should be taken into account and managed with second-line therapy (fluoroquinolone, pyrazinamide, ethionamide, or prothionamide and an injectable agent such as amikacin or capreomycin). Regular follow-up is essential for a complete cure.

Although the use of corticosteroid treatment in brain tuberculoma is controversial, steroids may be useful in cases

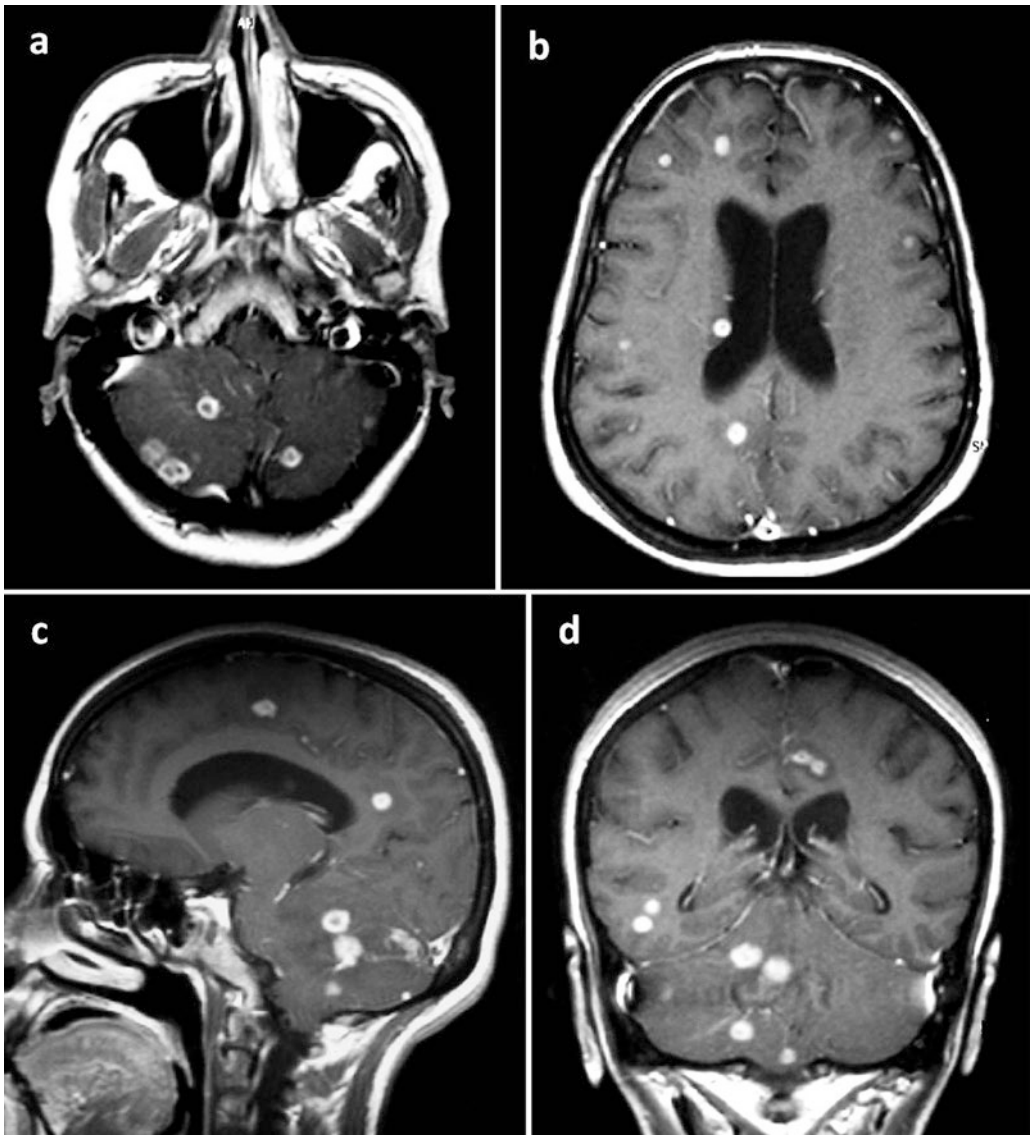


**Fig. 23.6** Case 23.3. Gadolinium-enhanced T1-weighted MRI: axial (**a**, **b**), sagittal (**c**), and coronal (**d**) views. The tuberculomas (miliary) are best seen following gadolinium injection

with raised intracranial pressure, important brain edema with mass effect, compromised mental or neurologic status, and potential life-threatening conditions such as brain herniation. Treatment duration should not exceed 8 weeks. Antiepileptic prophylaxis may be considered if lesions are close to epileptogenic areas.

Under several conditions, neurosurgical intervention may be required (Figs. 23.19, 23.20, 23.21, 23.22, and 23.23):

- To obtain a definitive diagnosis (biopsy)
- To relieve mass lesions (craniotomy and microsurgical excision)
- If medical therapy has had no success
- To manage high intracranial pressure (decompressive craniectomy)
- For CSF diversion in cases of hydrocephalus (external ventricular drainage, internal shunt placement, or endoscopic third ventriculostomy)

Physical rehabilitation, adequate nutrition, and treatment of underlying medical comorbidities are crucial to the successful treatment of CNS tuberculosis.

## Outcomes

The prognosis of brain tuberculoma depends on the patient's general health, the underlying neurologic conditions, the presence of concomitant tuberculous meningitis, the diagnosis time, and the response to antituberculous medications.

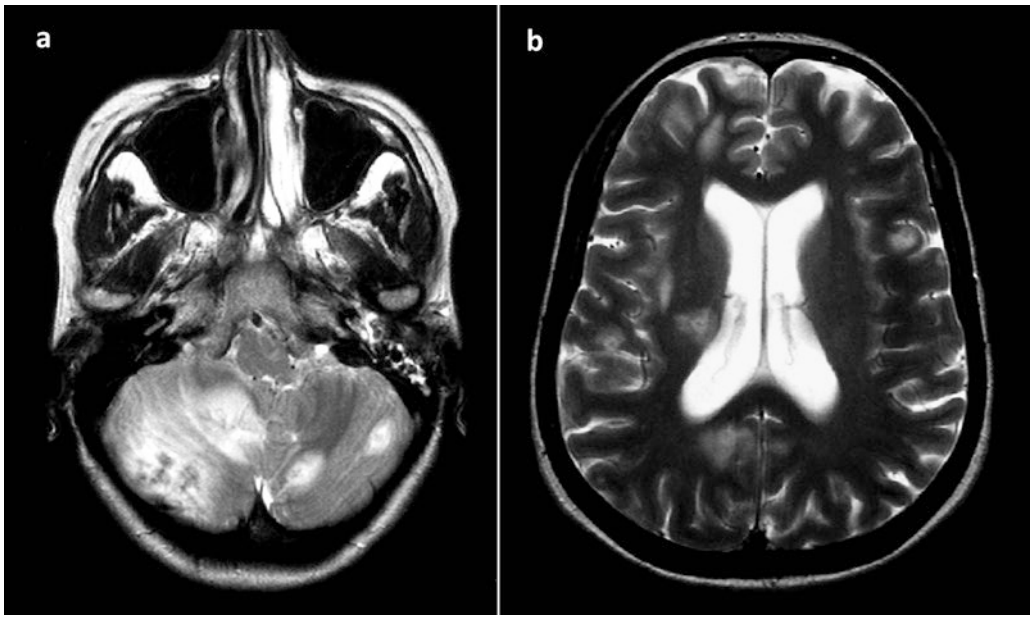


**Fig. 23.7** Case 23.3. (**a**, **b**) The same brain lesions on axial T2-weighted MRI

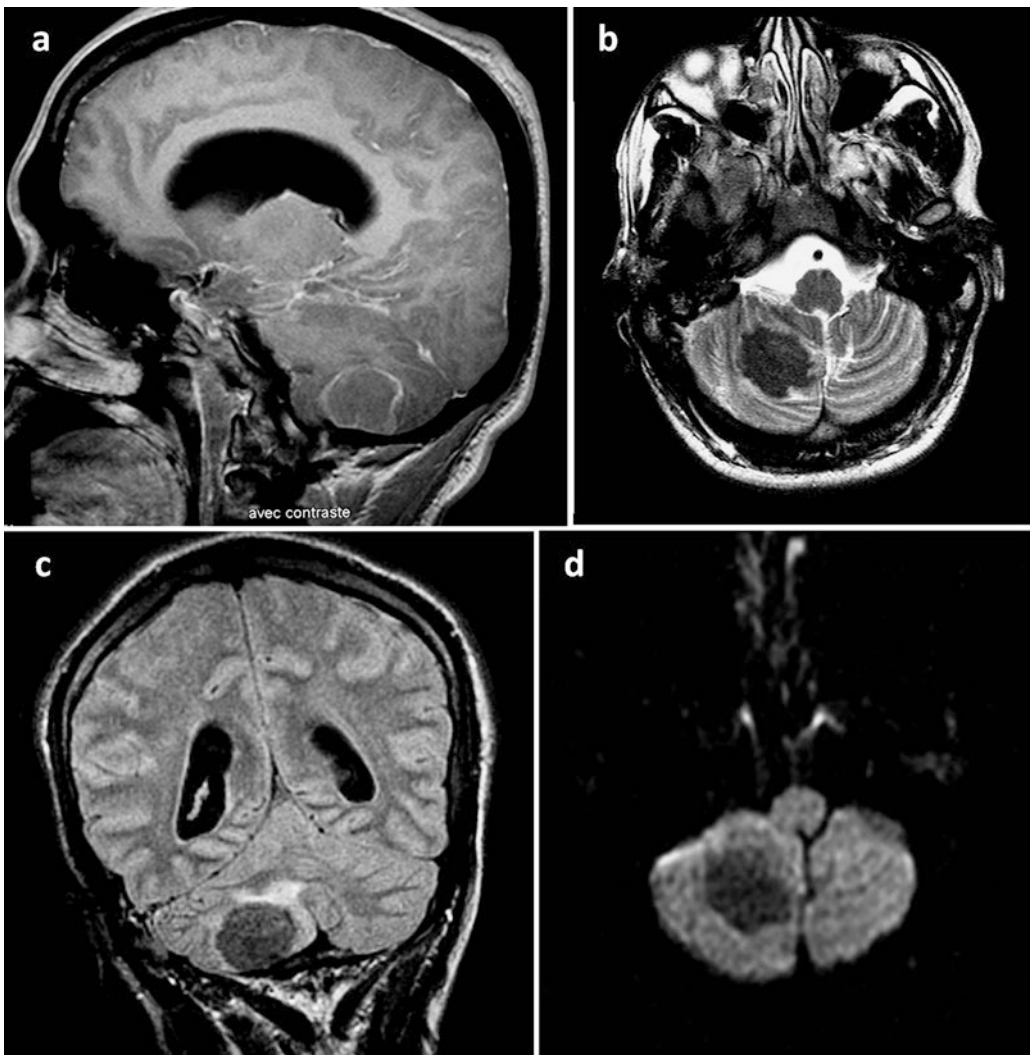


**Fig. 23.8** Case 23.4. Large solitary tuberculoma of the posterior fossa located in the right cerebellar hemisphere. Sagittal T1-weighted MR image with gadolinium injection (**a**), axial T2-weighted image (**b**), coronal FLAIR sequence (**c**), diffusion-weighted image (**d**)

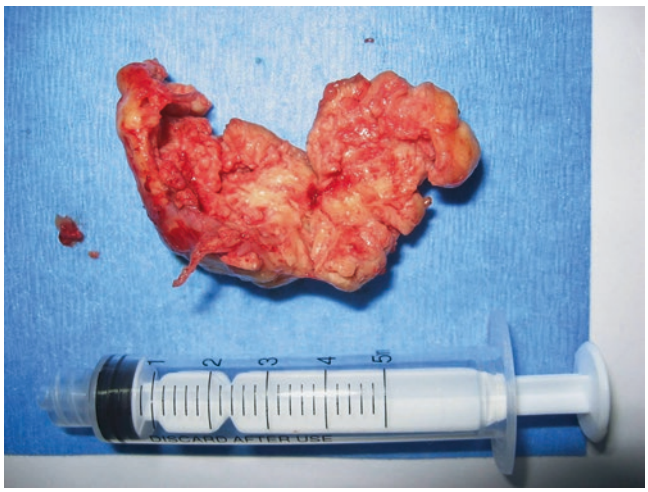

**Fig. 23.9** Case 23.4. Macroscopic appearance of the completely removed granulomatous lesion

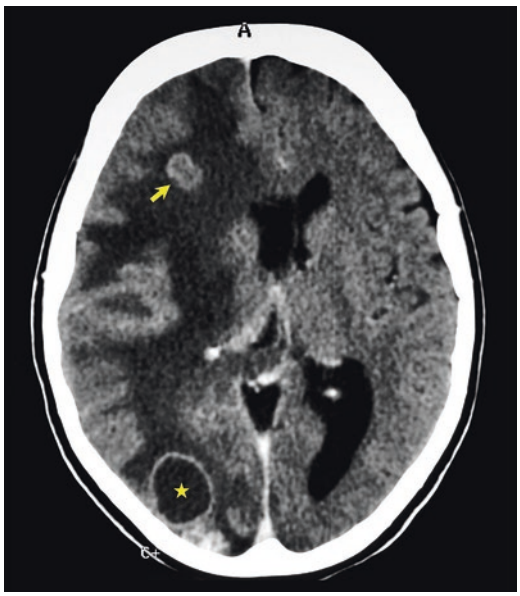


**Fig. 23.11** Case 23.5. Axial post-contrast CT scan revealing concomitant parieto-occipital ring (*star*) and frontal nodular (*arrow*) brain tuberculomas

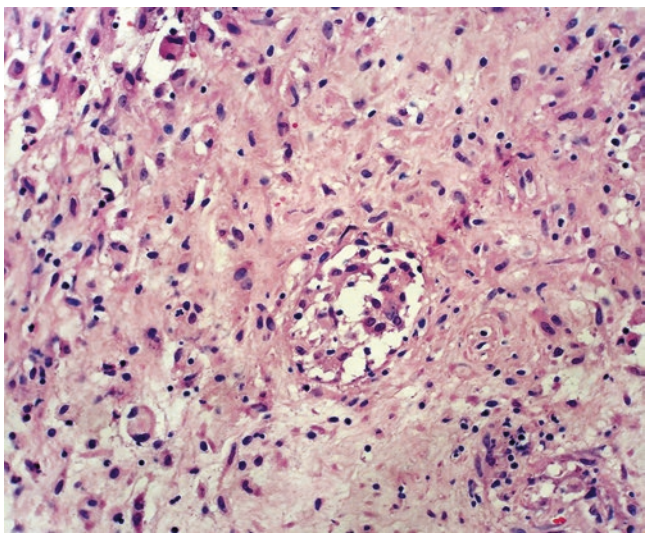

**Fig. 23.10** Case 23.4. Histopathologic features of cerebellar tuberculoma. Presence of epithelioid–giant cell granuloma with mononuclear cell infiltration (hematoxylin–eosin staining)

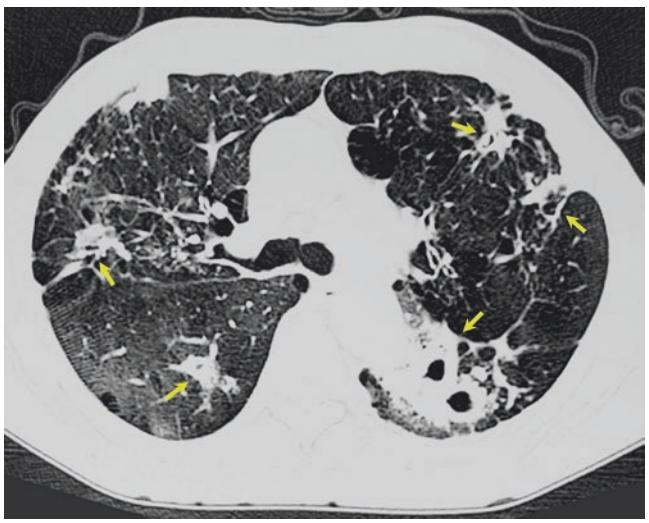

**Fig. 23.12** Case 23.5. This patient had also pulmonary lesions detected on thoracic CT scan (*arrows*)

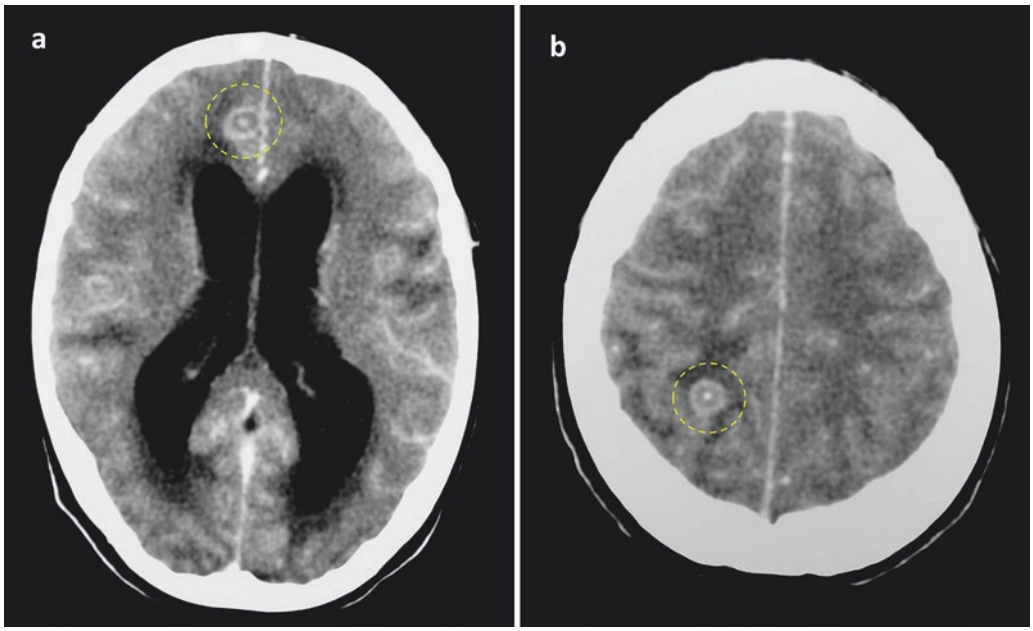


**Fig. 23.13** (**a**, **b**) Axial CT scan with contrast injection, showing brain tuberculomas with a central nidus of calcification known as a "target sign" (*dotted circle*) in a patient with a tuberculous meningoencephalitis. Note the dilated lateral ventricles (hydrocephalus)

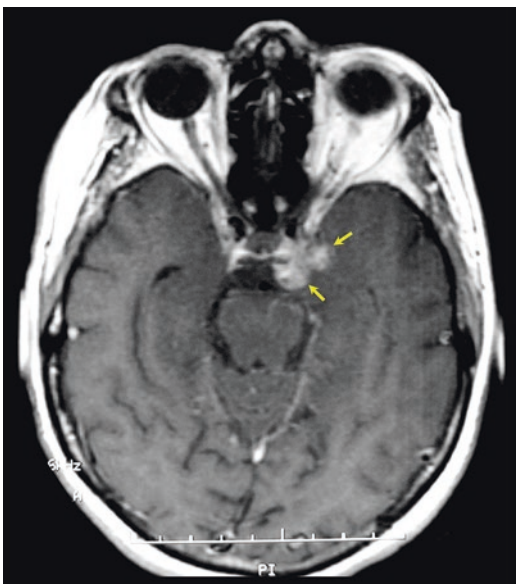

**Fig. 23.14** Optochiasmatic tuberculomas (*arrows*) on axial post-gadolinium T1-weighted MRI

Most patients with a solitary tuberculoma sensitive to first-line antituberculous therapy have a good outcome, but about 20% of treated patients experience various neurologic sequelae, including seizures, focal neurologic deficits, aphasia, blindness, or cognitive impairment. Patients with extensively drug-resistant infections have a poor prognosis.

Prevention of tuberculosis is based on generalized and controlled immunization (BCG vaccination) along with improvement of health care and the living conditions of the population.

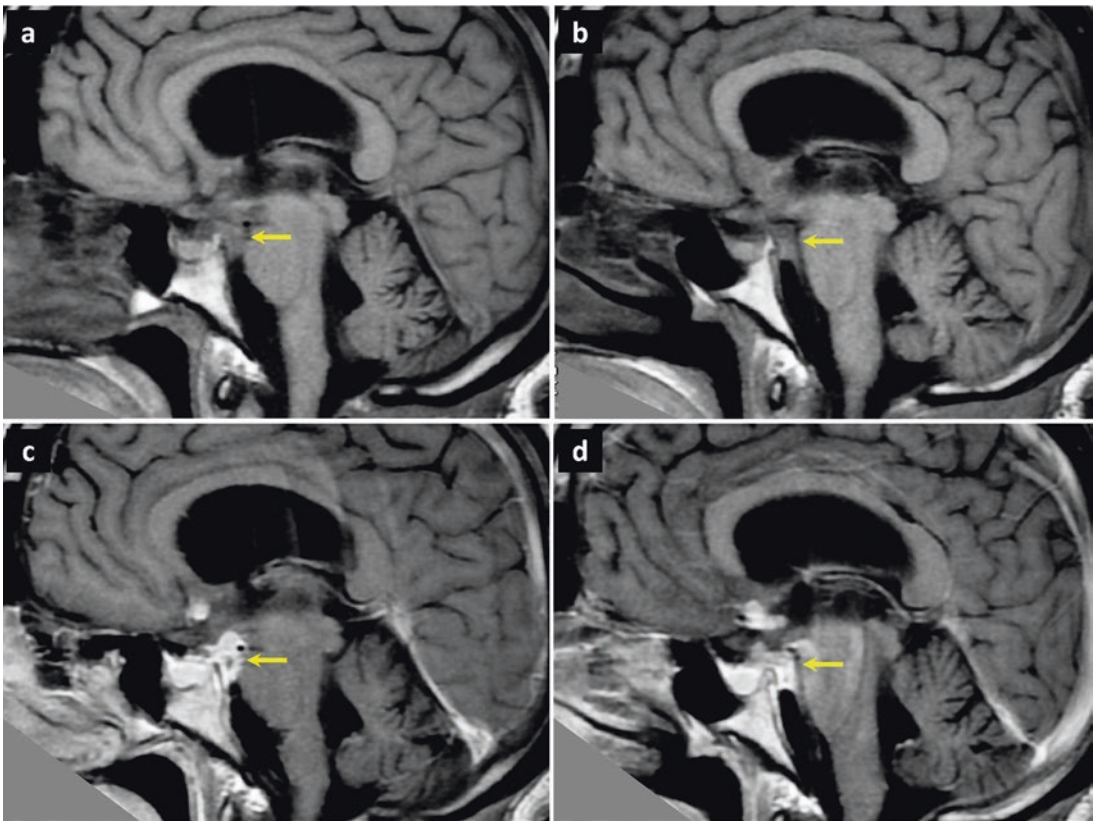


**Fig. 23.15** Case 23.6. Sagittal T1-weighted MR images without (**a**, **b**) and with (**c**, **d**) gadolinium injection, revealing multiple tuberculomas of the sellar, suprachiasmatic, retrochiasmatic, and retrosellar (*arrow*) regions

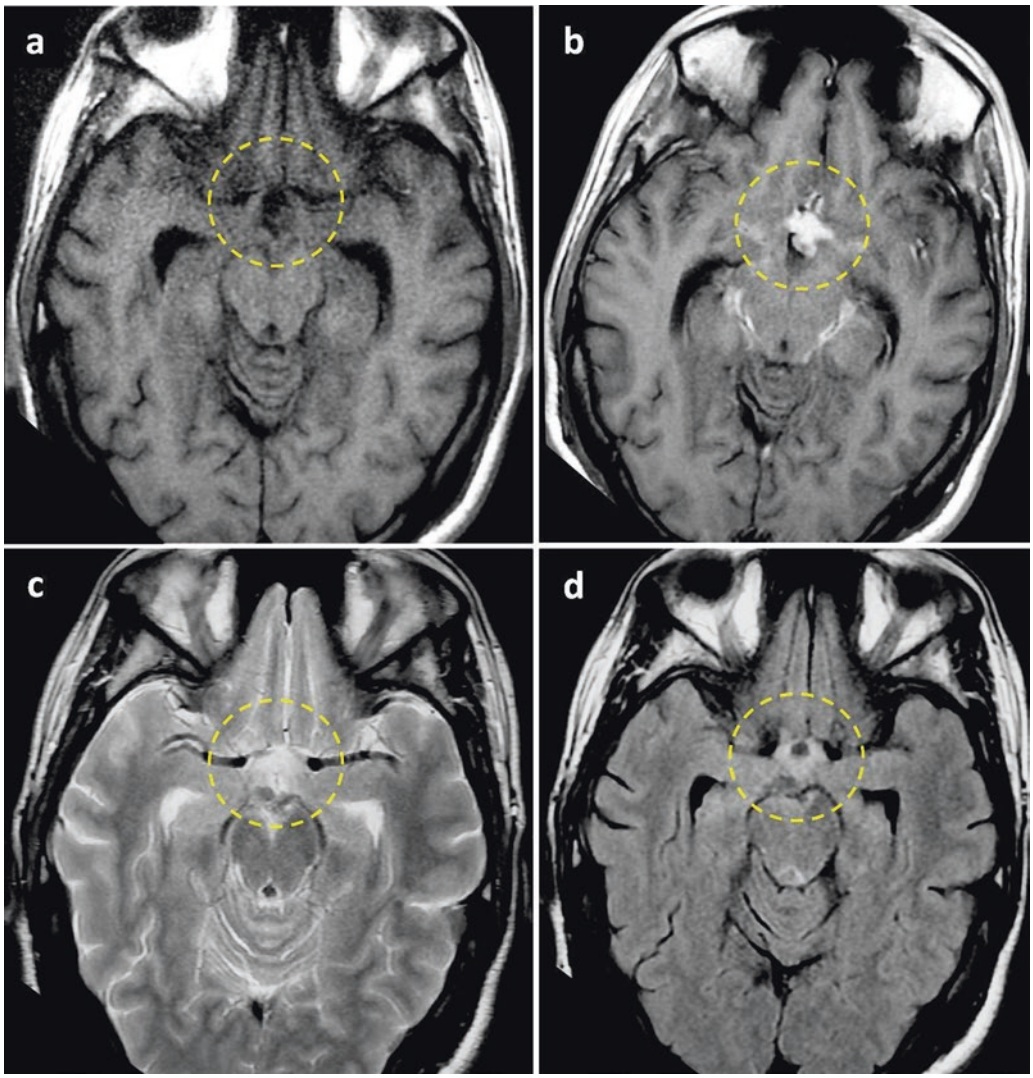


**Fig. 23.16** Case 23.6. Hypothalamic suprachiasmatic tuberculomas (*dotted circle*) on axial T1-weighted MR images before (**a**) and after (**b**) gadolinium administration, axial T2-weighted image (**c**), and FLAIR sequence (**d**)

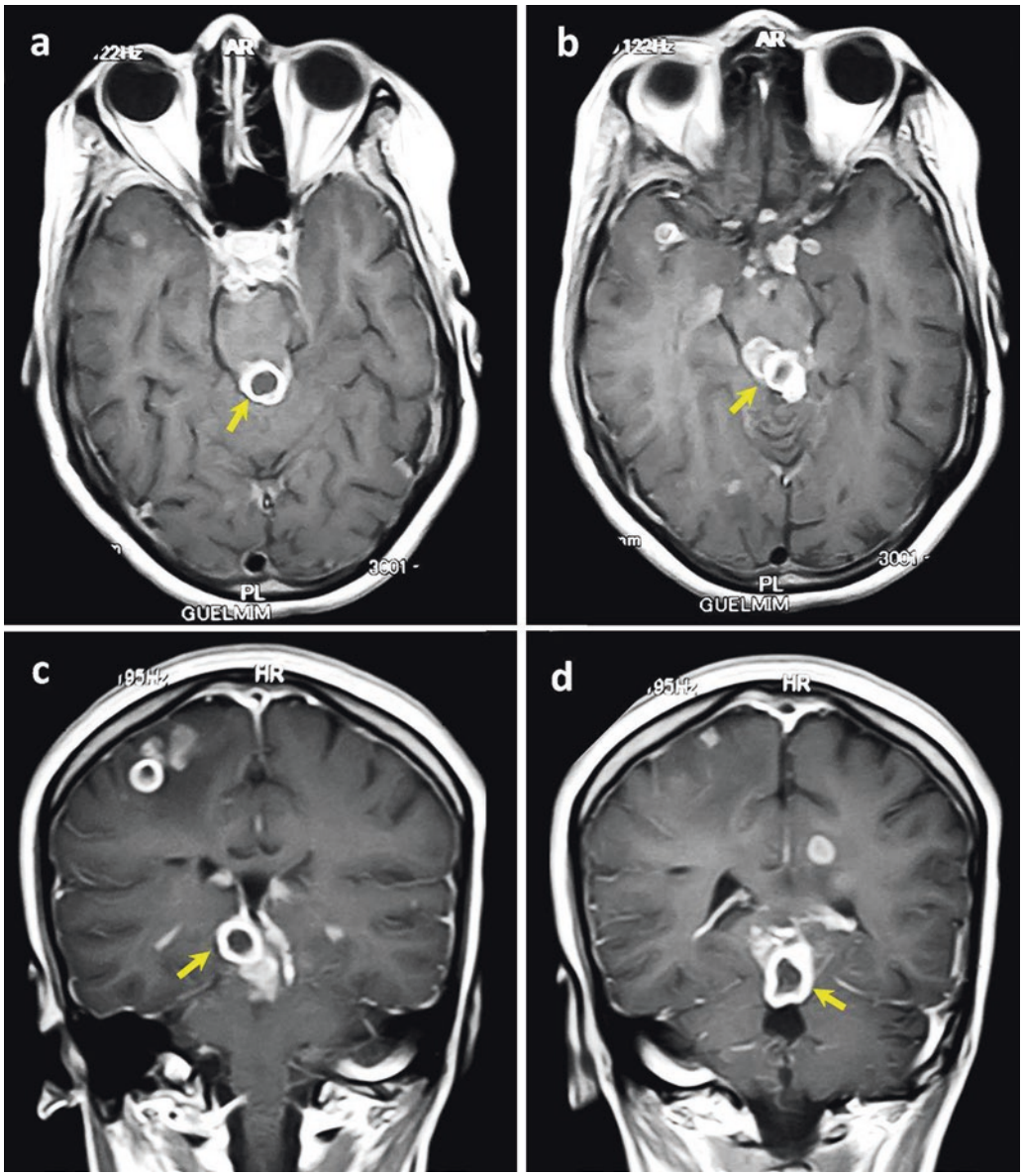


**Fig. 23.17** Axial (**a**, **b**) and coronal (**c**, **d**) post-gadolinium T1-weighted MRI showing multiple brain tuberculomas with a rare location in the pineal area (*arrow*)

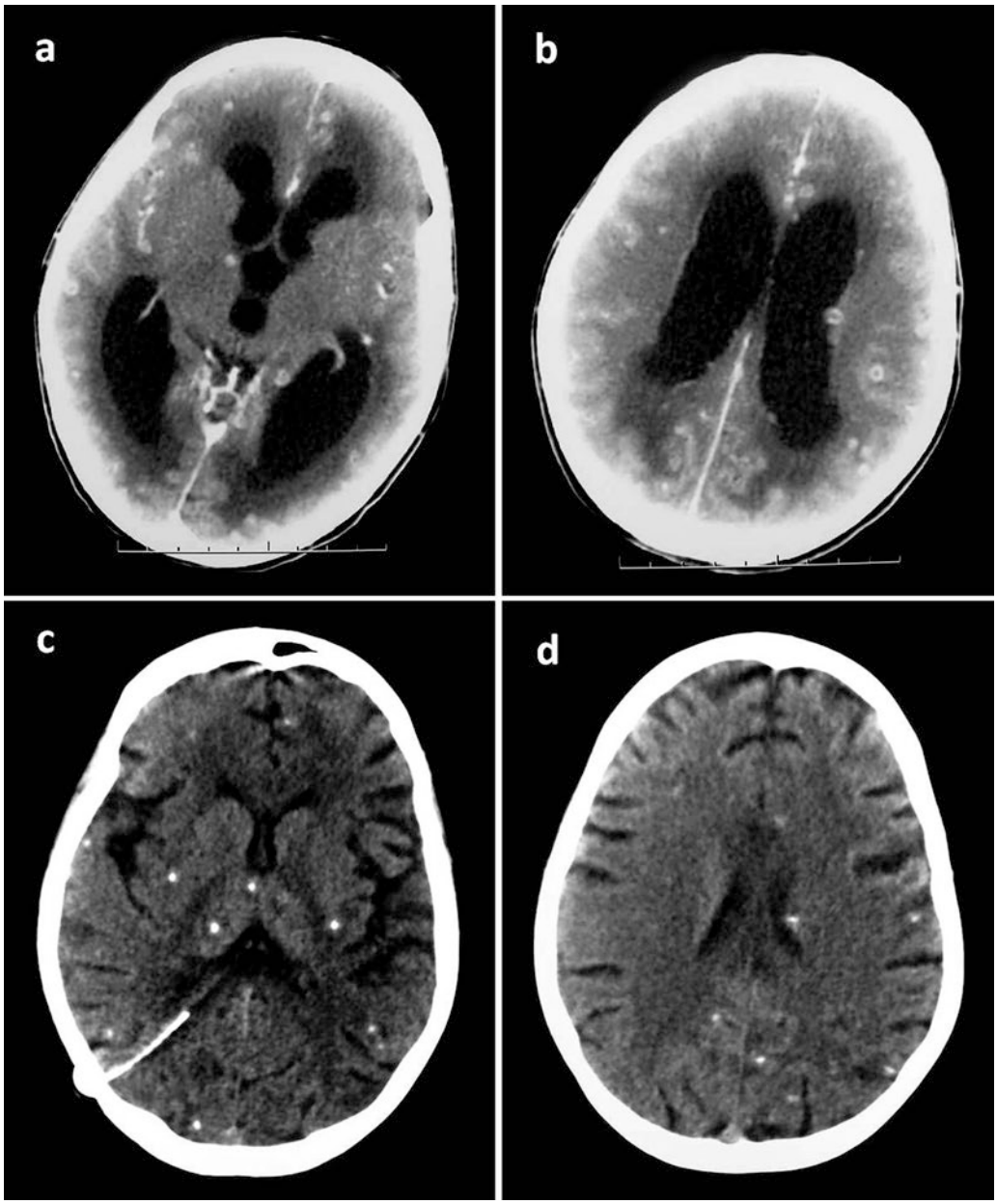


**Fig. 23.18** Axial post-contrast CT scan before (**a**, **b**) and after (**c**, **d**) ventricular shunt placement in a child who was under treatment for tuberculous meningoencephalitis and developed an acute hydrocephalus. Note disseminated (miliary) brain tuberculomas

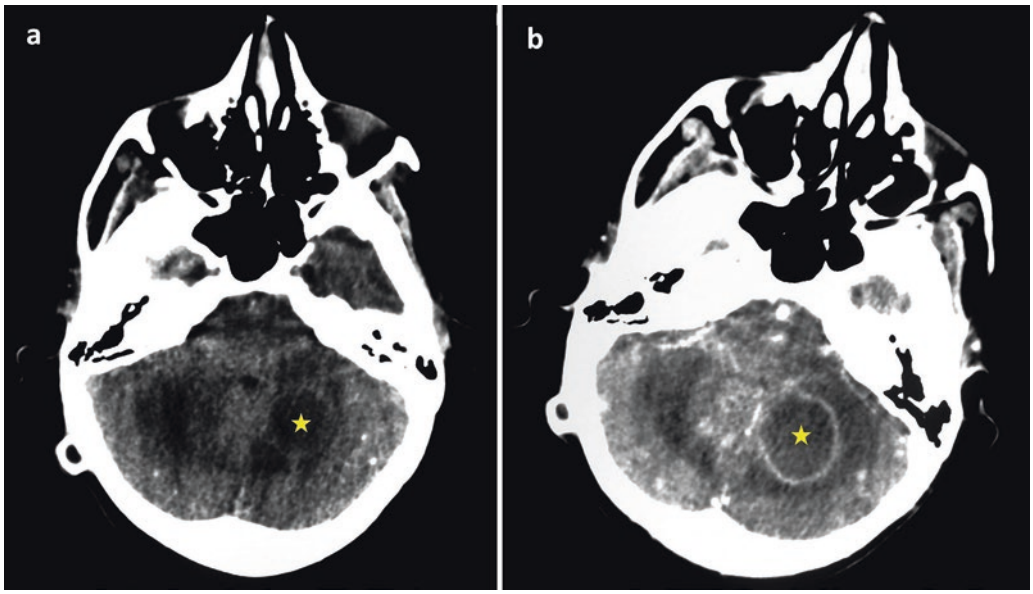


**Fig. 23.19** Case 23.7. Left hemispheric cerebellar collection (abscess) (*star*) in a patient under antituberculous therapy for meningoencephalitis, shown on axial CT scan before (**a**) and after (**b**) contrast injection. The abscess formation had a ring enhancement

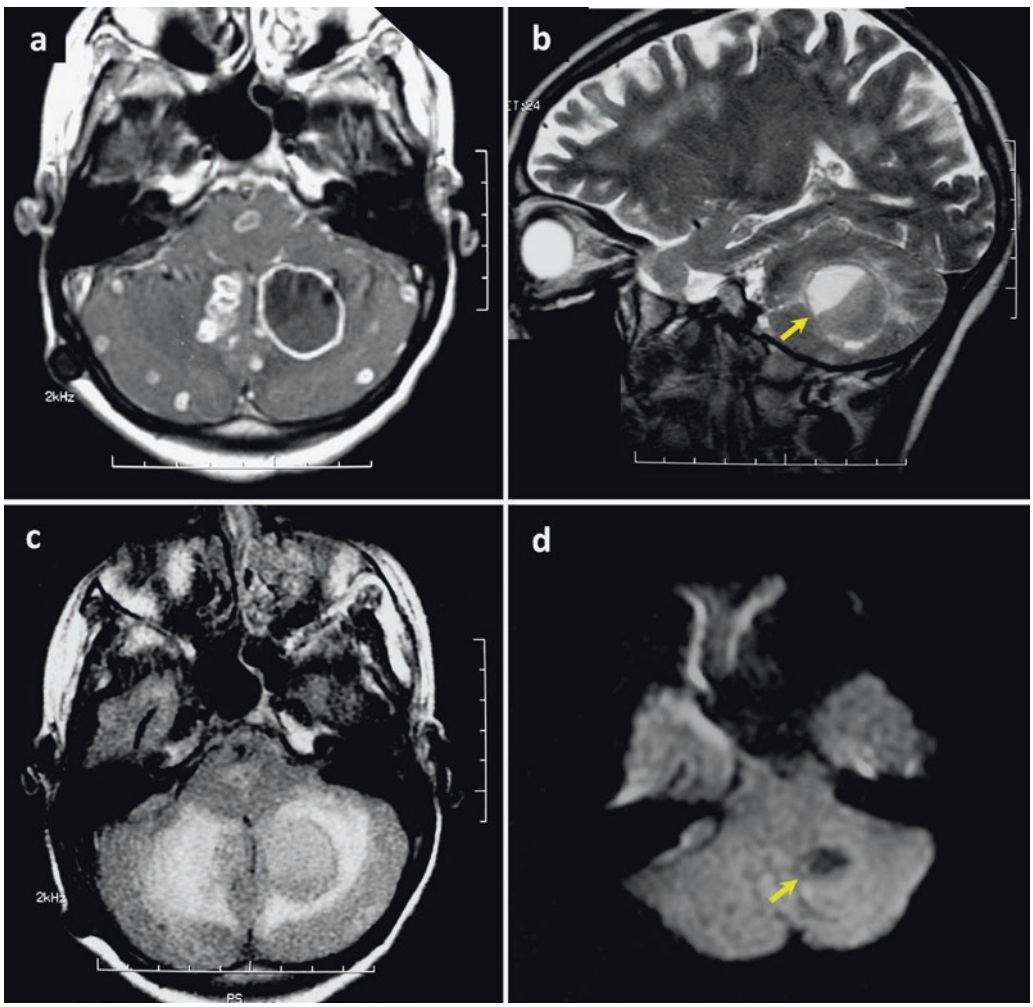


**Fig. 23.20** Case 23.7. MRI features of this tuberculous cerebellar abscess on axial-enhanced T1-weighted image (**a**), sagittal T2-weighted image (**b**), FLAIR sequence (**c**), and diffusion-weighted image (**d**). Note the fluid–fluid level of the abscess (*arrow*) (**b**, **d**)

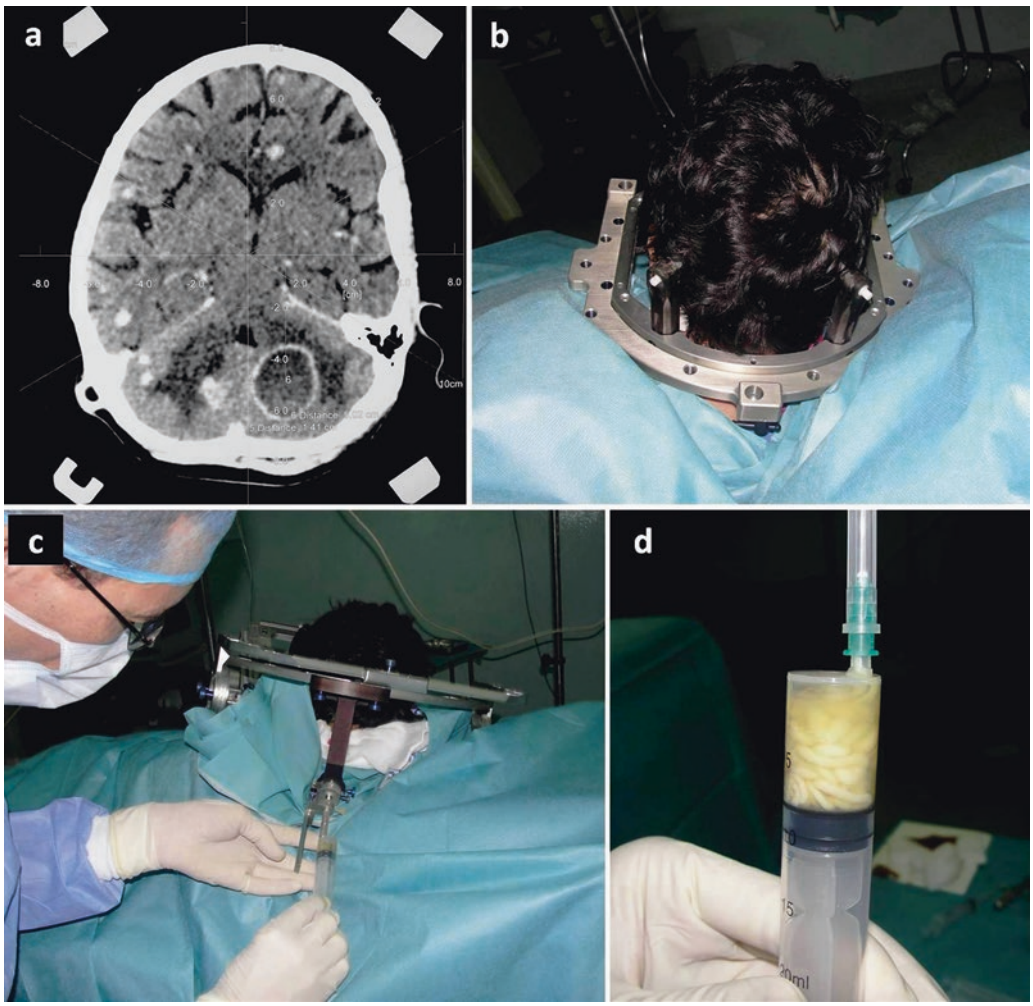


**Fig. 23.21** Case 23.7. Axial cranial CT scan with contrast injection during preplanning for stereotactic-guided aspiration of the cerebellar abscess (**a**). Radionics™ Cosman–Roberts–Wells (CRW™) frame (**b**). Operative view (percutaneous left suboccipital approach under local anesthesia): aspiration of the abscess collection under stereotactic guidance (**c**). Purulent material filling the syringe after a moderate aspiration (**d**)

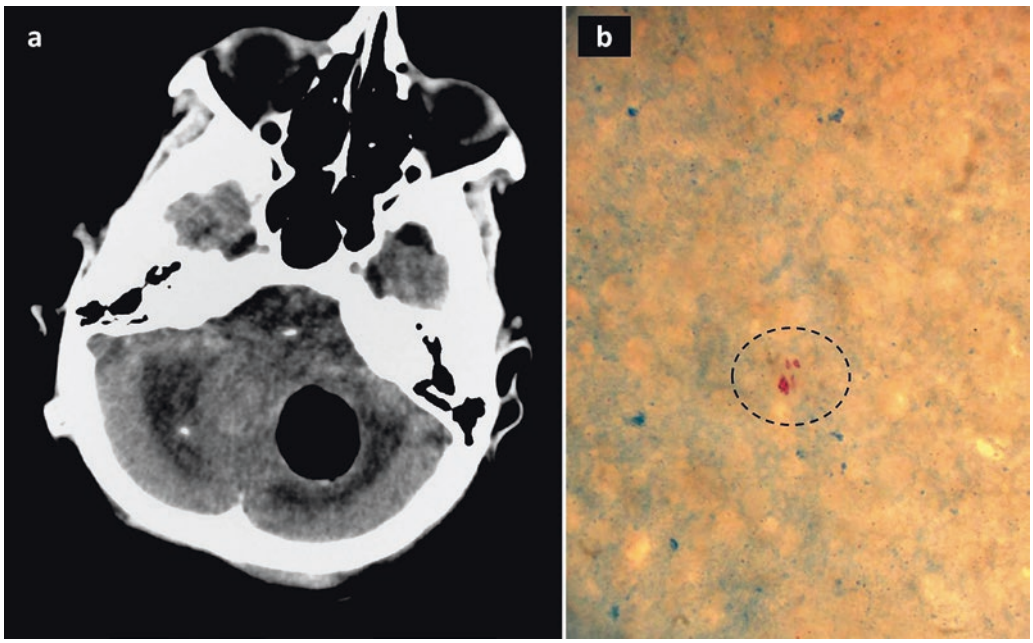


**Fig. 23.22** Case 23.7. Immediate postoperative axial CT scan showing the residual cavity of the cerebellar abscess (**a**). Positive acid-fast bacillus (*AFB*) following Ziehl–Neelsen staining in the same patient (**b**)

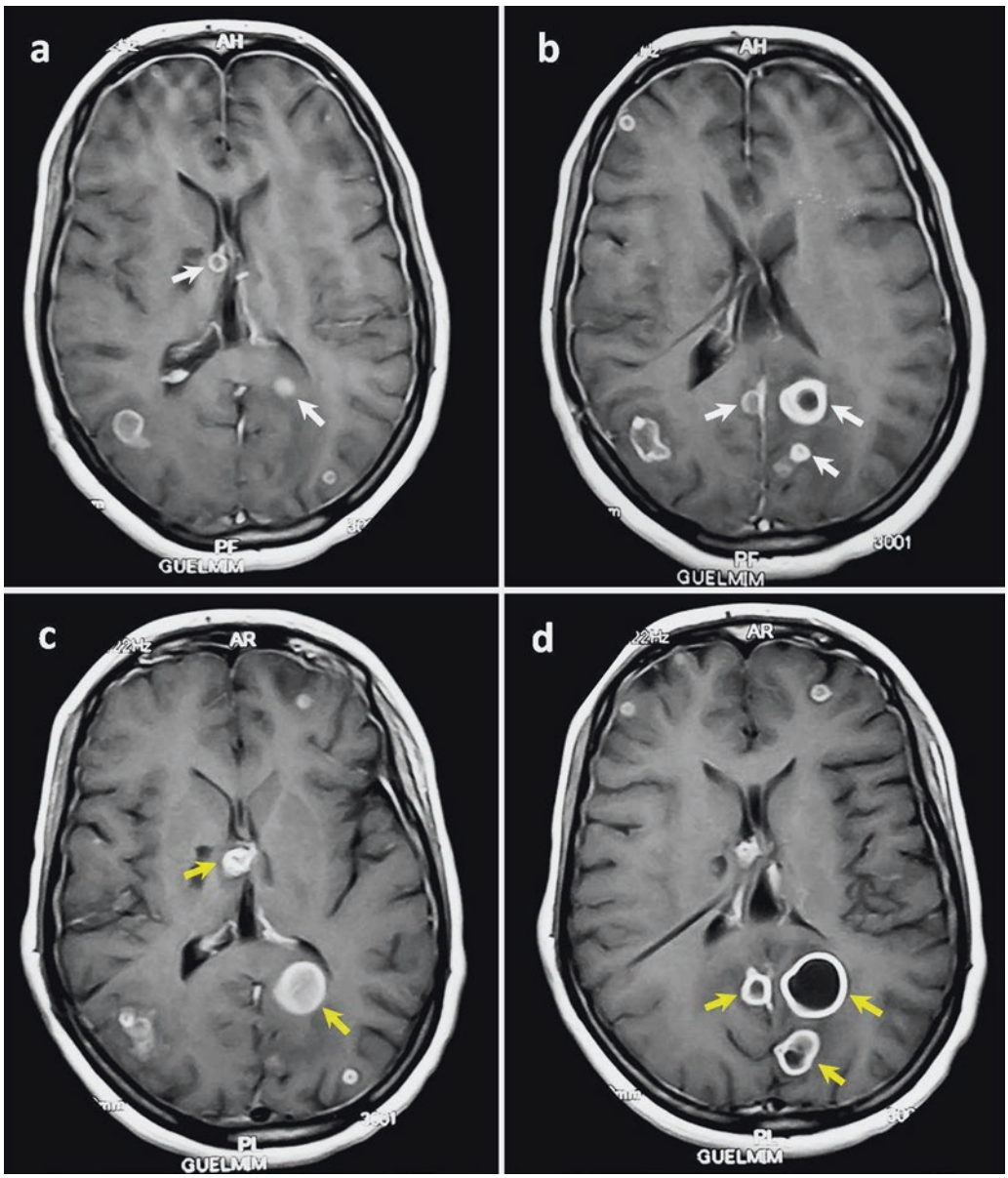


**Fig. 23.23** Multiple brain tuberculomas with paradoxical enlargement, in a 21-year-old woman treated for tuberculous meningoencephalitis, shown on initial gadolinium-enhanced, T1-weighted MRI (*white arrows*) (**a**, **b**). Despite effective treatment by antituberculosis therapy, the control MRI 4 months later (**c**, **d**) discloses paradoxical enlargement of some tuberculomas (*yellow arrows*)

## Suggested Reading


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# Spinal Tuberculosis

# 24

Spinal tuberculosis is severe and can lead to spinal deformities, segmental instabilities, or neurologic deficits. The spine is usually infected through hematogenous dissemination from a pulmonary focus. Spinal tuberculosis includes spondylodiscitis and spinal epidural, subdural, and cord lesions. Spondylodiscitis (Pott's disease) is the most frequent form (Fig. 24.1). Spinal tuberculosis progresses slowly and insidiously. Common clinical manifestations include spinal pain and tenderness, paraplegia, and spinal deformities. MRI is more sensitive than plain radiography and more specific than CT scans. Routine laboratory investigations are not specific. Etiological confirmation is made either by demonstration of *Mycobacterium tuberculosis* on pathological specimen or histological evidence of epithelioid-giant cell granulomas with caseating necrosis on the biopsy material. DNA amplification techniques and the QuantiFERON®-TB Gold In-Tube assay are helpful for early and rapid diagnosis. The primary treatment of spinal tuberculosis is chemotherapy. Surgery is indicated mainly for failure of conservative treatment, progressive neurologic deficit, and the prevention or correction of spinal deformity. When diagnosed early and treated adequately, the prognosis of tuberculous spondylodiscitis is usually favorable, particularly in patients without neurologic deficit and deformity. Patients with intradural lesions have a much poorer neurologic prognosis than those with spondylodiscitis.

## Epidemiology and Etiology

Tuberculosis continues to be a significant health problem in many developing countries and is gradually reemerging in Europe and North America, mainly owing to immigration and epidemic HIV disease. The chief causative pathogen of tuberculosis is *Mycobacterium tuberculosis*, an acid-fast bacterium that is uncommon in extrapulmonary organs.

The spine is usually infected through hematogenous dissemination from an active focus in the lungs. A wide variety of pathologic entities may be seen in spinal tuberculosis:

- Spondylodiscitis
- Spinal epidural lesion
- Spinal subdural lesion
- Spinal cord tuberculoma

The most frequent form is spondylodiscitis, also known as Pott's disease. This is a combination of vertebral osteomyelitis and discitis with paravertebral and epidural extension. On the other hand, intradural lesions are rare.

Spondylodiscitis is a serious disease that can lead to spinal deformities, segmental instabilities, or neurologic deficits. Table 24.1 outlines the mechanisms of neurologic involvement in spinal tuberculosis. Although involvement at the thoracolumbar junction is most common, any level can be affected. Two vertebrae are typically affected, with the vertebral body more commonly involved than the posterior arch. The majority of patients are young men.

Predisposing factors for the development of Pott's disease include a history of previous tuberculosis (individual or familial), poverty, malnutrition, alcoholism, drug abuse, imprisonment, diabetes mellitus, advanced age, immunosuppressive treatment, HIV infection, and chronic renal failure.

## Clinical Presentations

Spinal tuberculosis progresses slowly and insidiously, with a course that is often longer and more indolent than the usual pyogenic spondylodiscitis. Symptoms and signs may vary from simple spinal pain to complete paraplegia or tetraplegia, depending on the location, the size, and the extension of the granulomatous and/or suppurative lesions.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_24

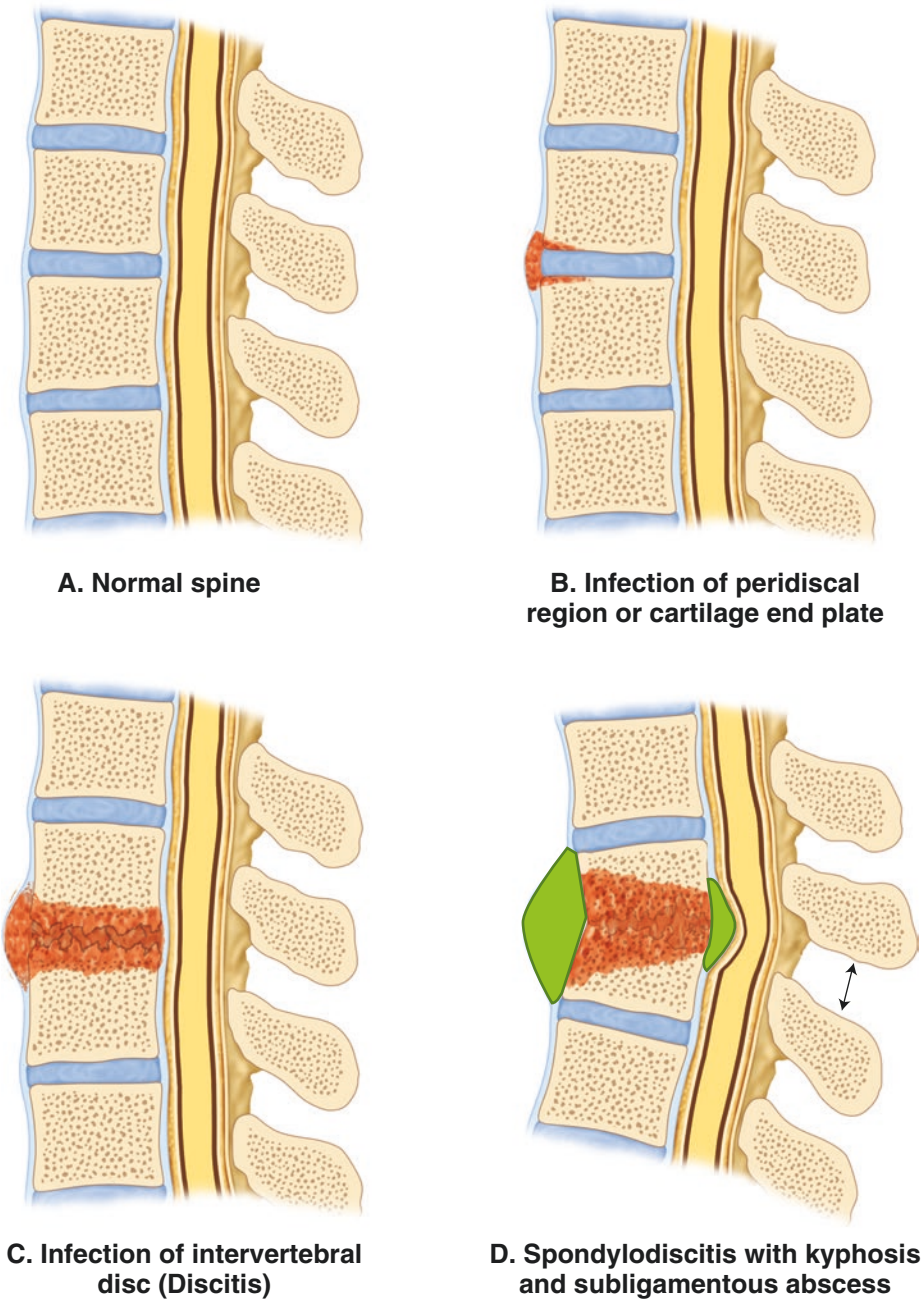



**Fig. 24.1** Classic location of spinal tuberculosis and its extension

**Table 24.1** Mechanisms of neurologic involvement in spinal tuberculosis

| Type | Mechanism |
|---|---|
| **A** | Direct mechanical pressure by the spondylodiscitis |
| **B** | Granulomatous lesions |
| **C** | Infective myelitis or radiculomyelitis |
| **D** | Infective spinal artery thrombosis |
| **E** | Arachnoiditis or pachymeningitis |

The most common presenting manifestation of tuberculous spondylodiscitis is rachialgia at the site of the infection, but systemic signs such as fever, chills, night sweats, anorexia, weight loss, and general malaise are frequent. Other clinical findings include paraspinal muscle spasms and rigidity, spinal deformity or even severely angled gibbosity, radicular pain, and neurologic deficits in different combinations.

Particular attention should be paid to associated paravertebral cold abscess in the retropharyngeal, mediastinal, psoas, or gluteal regions. Abscess formation is common, and they can grow to a very large size without pain or other signs of inflammation. More rarely, chronic vertebral spondylodiscitis may present with a cutaneous draining sinus tract. Screening of the whole body should be done to look for a concomitant infectious disease.

## Imaging Features

Imaging studies are the mainstay to identify the location and extent of the spinal lesions. MRI is a more sensitive imaging technique than X-ray and more specific than CT scanning (Figs. 24.2, 24.3, 24.4, 24.5, 24.6, 24.7, 24.8, 24.9, 24.10, 24.11, 24.12, 24.13, 24.14, 24.15, 24.16, 24.17, 24.18, 24.19, 24.20, 24.21, 24.22, 24.23, 24.24, 24.25, 24.26, 24.27, 24.28, 24.29, 24.30, 24.31, 24.32, 24.33, 24.34, 24.35, 24.36, 24.37, 24.38, 24.39, 24.40, 24.41, 24.42, 24.43, 24.44, 24.45, 24.46, and 24.47).

Plain radiographs may be useful as a first diagnostic step. The characteristic findings include rarefaction of the vertebral end plates, loss of disc height, osseous destruction, kyphosis, soft tissue abscess, and finally new bone formation and bony bridging. These lesions may take several weeks to appear. The posterior vertebral structures are long preserved. Chest X-ray should be performed, because up to 50% of patients with spinal tuberculosis have coexisting lung tuberculosis.

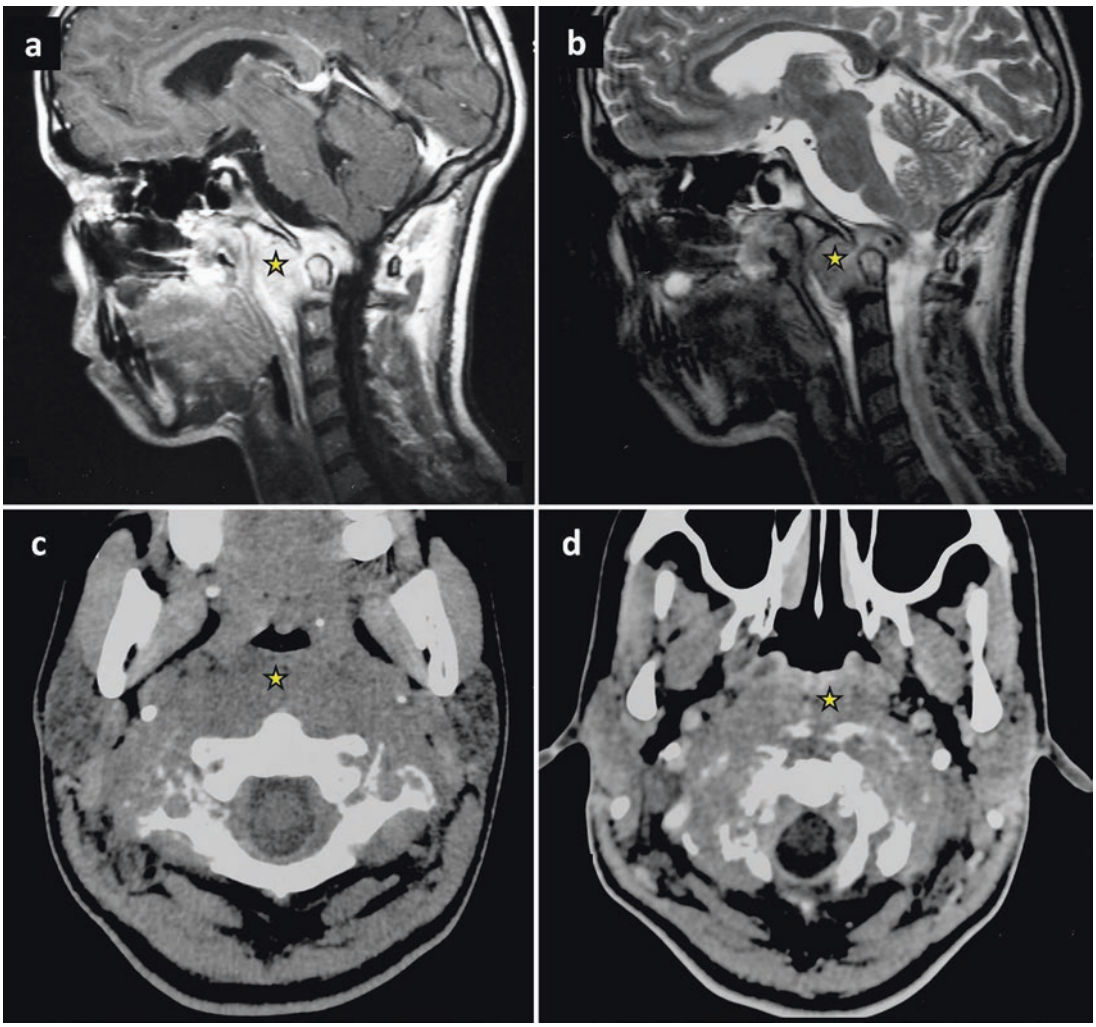



**Fig. 24.2** Tuberculous disease involving the craniocervical junction. Sagittal enhanced T1-weighted (**a**) and T2-weighted (**b**) MRI. Axial CT scan after contrast enhancement (**c**, **d**). The granulomatous lesion involves the odontoid process, the anterolateral part of C1 with retropharyngeal involvement (*star*). Note the suboccipital luxation and the foramen magnum extension

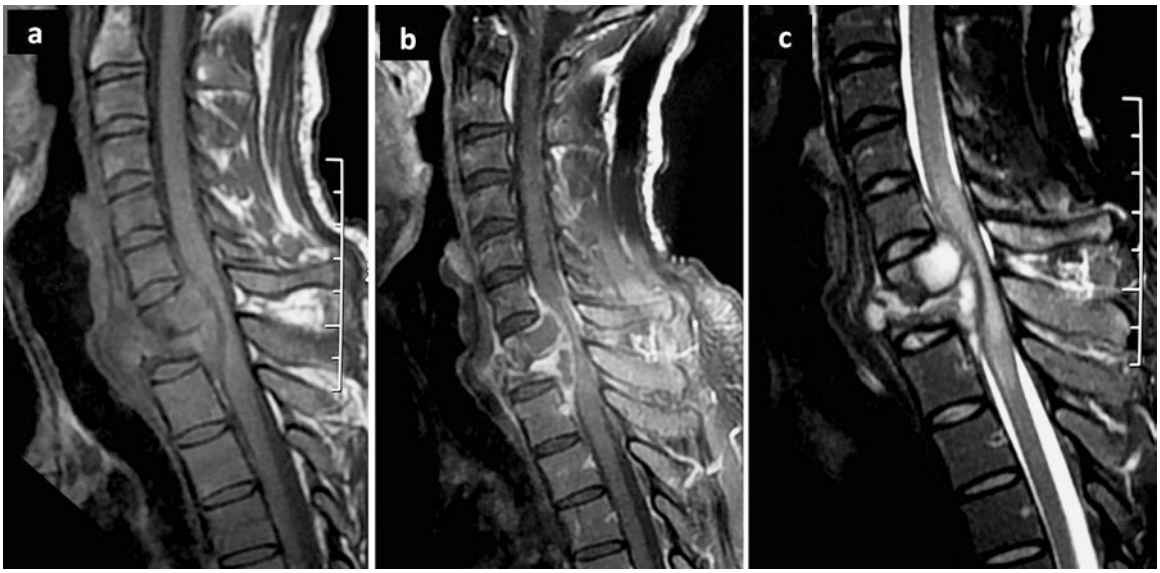



**Fig. 24.3** Case 24.1. Cervicothoracic Pott's disease. Sagittal T1-weighted images before (**a**) and after (**b**) gadolinium administration and T2-weighted image (**c**). C7 and T1 were collapsed with bone destruction, spinal subluxation, and epidural extension. Note the extensive high signal intensity within the spinal cord (**c**)

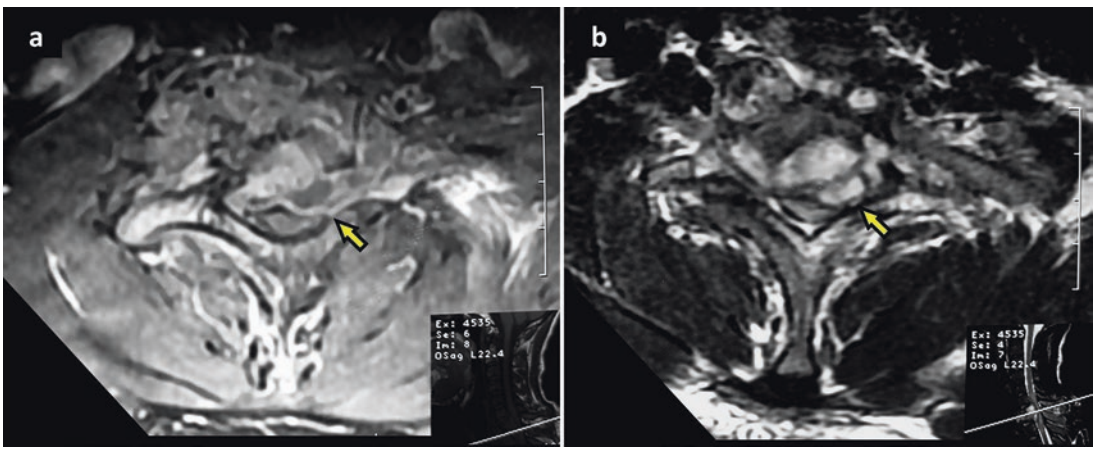



**Fig. 24.4** Case 24.1. Axial fgadolinium T1-weighted MR image (**a**) and T2-weighted image (**b**). Note the spinal cord compression caused by the epidural abscess (*arrow*)

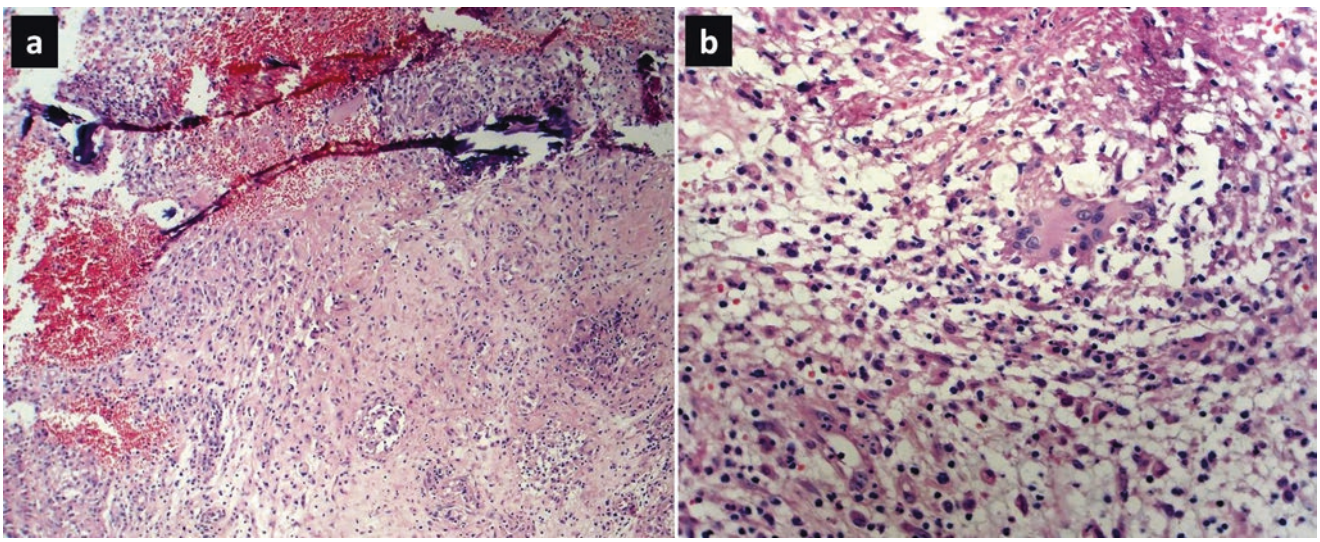



**Fig. 24.5** Histopathologic features of tuberculous spondylodiscitis, with the presence of epithelioid granulomas and giant cells with mononuclear cell infiltration. Medium-power (**a**) and high-power (**b**) magnification; hematoxylin–eosin staining

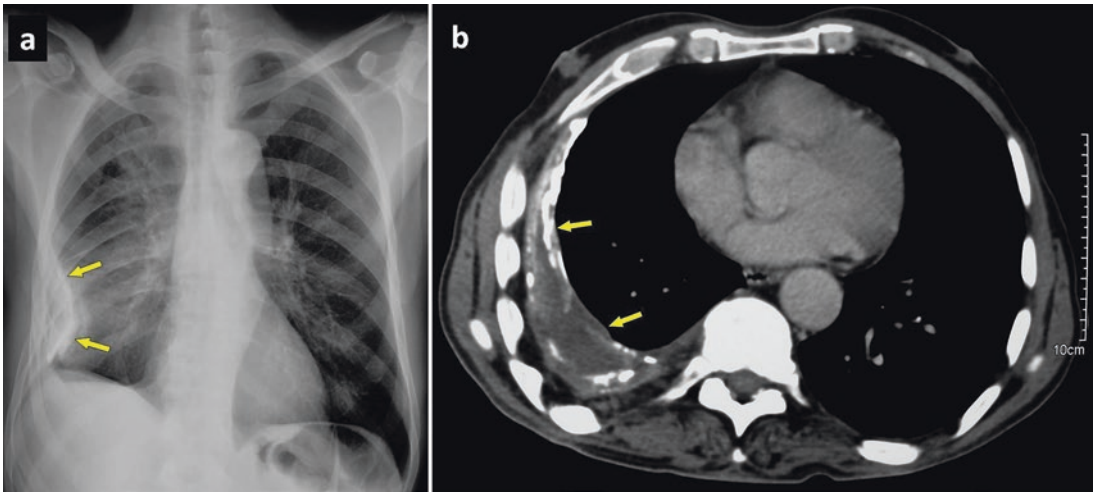



**Fig. 24.6** Case 24.2. Chest X-ray (**a**) and axial thoracic CT scan (**b**). This patient had a history of calcified right pachypleuritis (*arrows*) due to tuberculosis

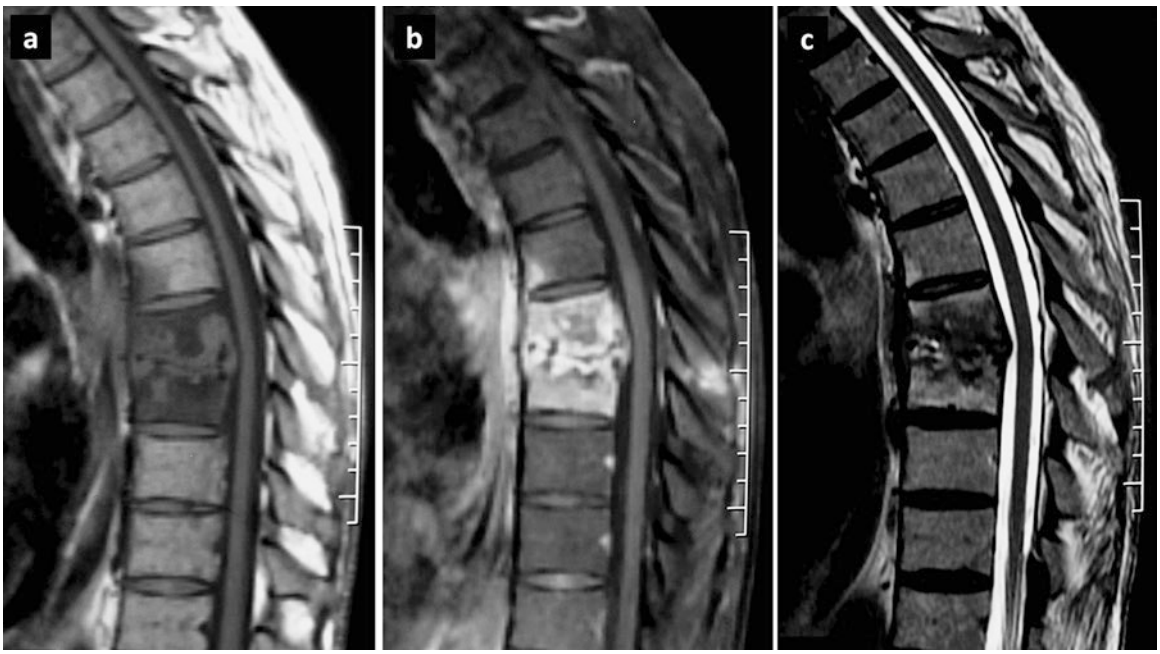



**Fig. 24.7** Case 24.2. He developed a T7–T8 spondylodiscitis without neurologic deficit. Sagittal T1-weighted MR image without (**a**) and with (**b**) gadolinium injection and T2-weighted image (**c**). Note the discal destruction and involvement of the vertebral bodies on both sides of the disc

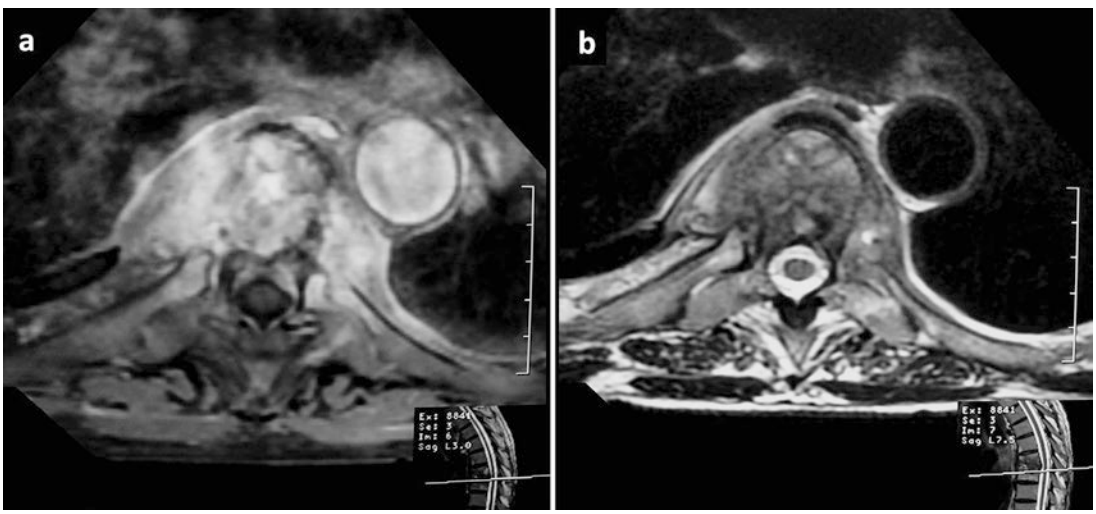



**Fig. 24.8** Case 24.2. Axial enhanced T1-weighted MR image (**a**) and T2-weighted image (**b**) showing extension of granulation tissue adjacent to the vertebral body

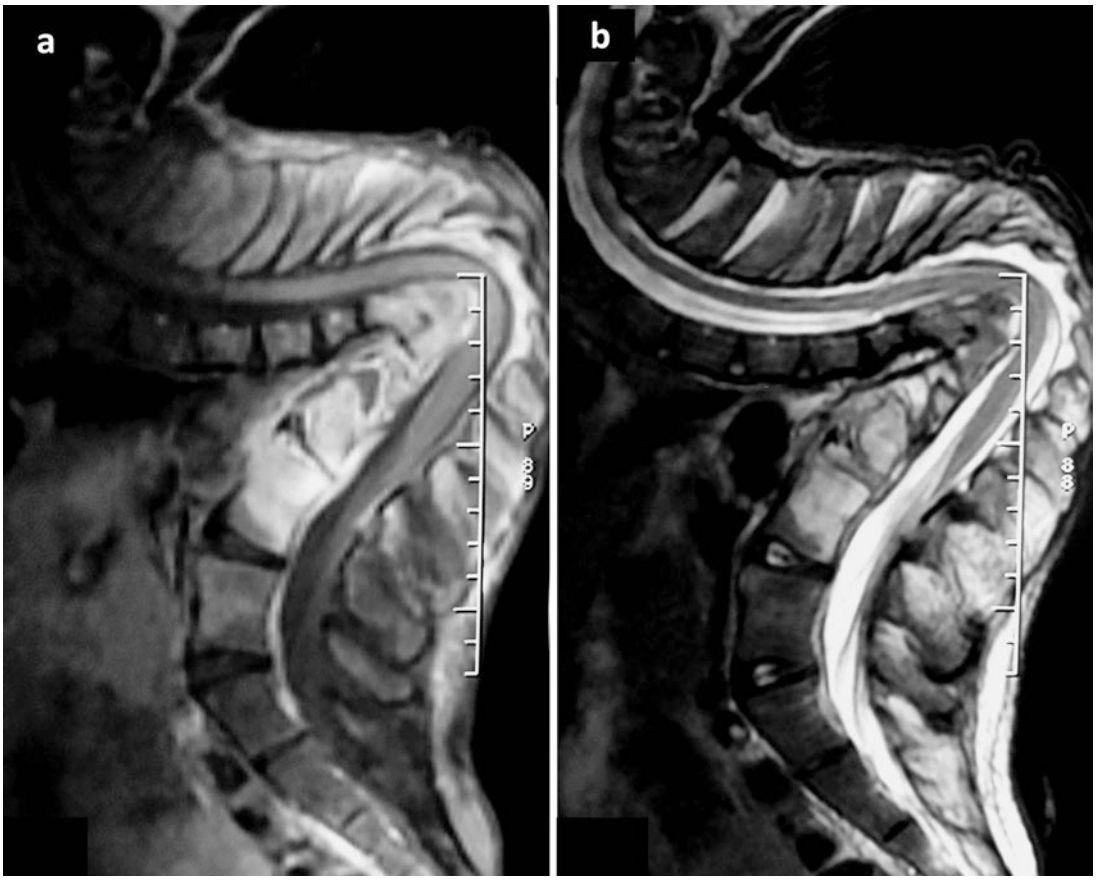



**Fig. 24.9** Case 24.3. This 26-year-old man had a history of neglected thoracic Pott's disease in his childhood. He developed a new lumbar posterior paraspinal swelling and fever without neurologic deficit. Sagittal enhanced T1-weighted (**a**) and T2-weighted (**b**) MR images showing the complex thoracic deformity (kyphoscoliosis as a sequela of previous tuberculosis) with new lumbar spondylodiscitis

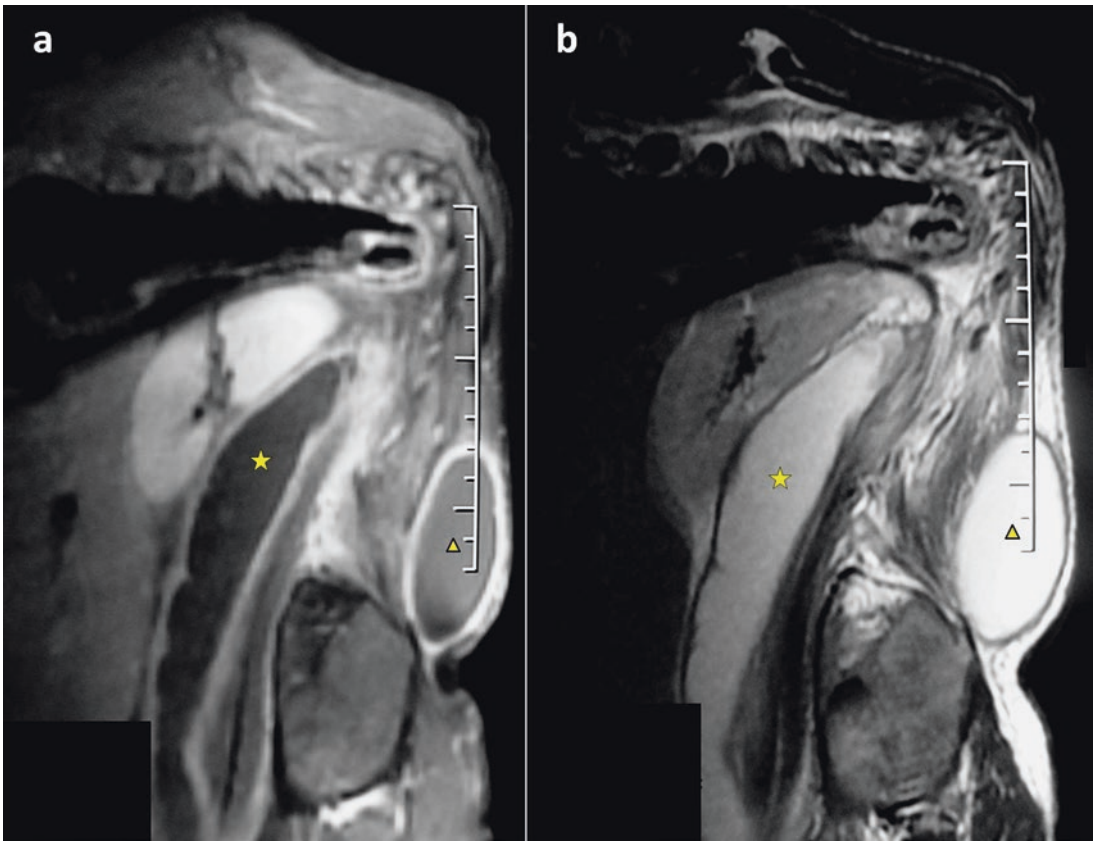



**Fig. 24.10** Case 24.3. Sagittal enhanced T1-weighted (**a**) and T2-weighted (**b**) MR images showing two large paraspinal "cold" abscesses: psoas (*star*) and lumbar subcutaneous (*triangle*)

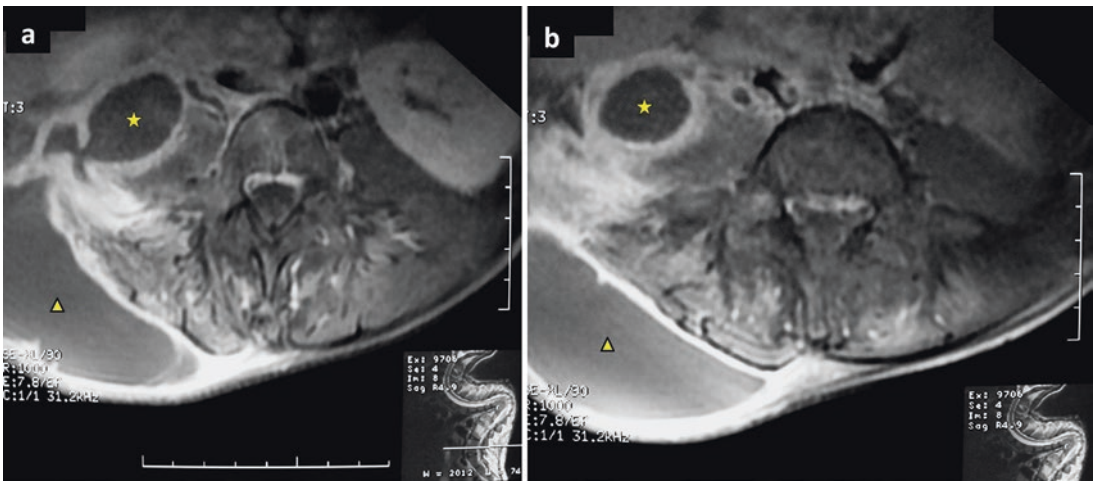



**Fig. 24.11** Case 24.3. (**a**, **b**) Axial T1-weighted images following gadolinium administration, revealing the locations of the abscess collections: psoas (*star*) and lumbar subcutaneous (*triangle*)

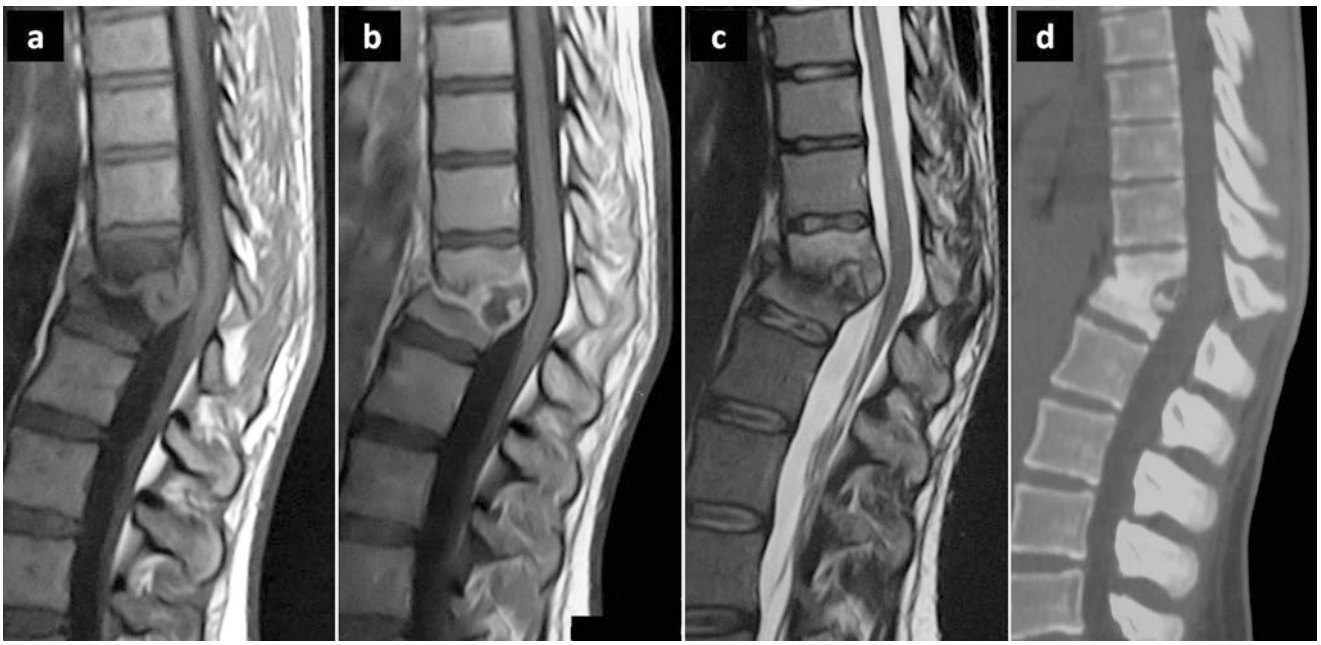



**Fig. 24.12** Case 24.4. Tuberculous spondylodiscitis at T11–T12 without neurologic deficit. Sagittal T1-weighted MR images before (**a**) and after (**b**) gadolinium injection and T2-weighted MRI (**c**). This patient was treated with antituberculosis drugs and external immobilization for 9 months. At the end of treatment, a sagittal CT scan on bone window shows vertebral body fusion (**d**)

CT scan with three-dimensional reconstruction gives important information on the degree of bone destruction, epidural extension, and soft tissue involvement, which is particularly useful for surgical planning and the performance of image-guided biopsies and drainage of paraspinal abscesses. Soft tissue calcifications are highly suggestive of Pott's disease.

MRI frequently demonstrates involvement of the vertebral bodies on both sides of the disc, discal destruction, cold abscess, vertebral collapse, and spinal deformities. In the early stages, only disc alteration with modification of bone marrow signal intensity is seen. Abscess formation and extension of granulation tissue adjacent to the vertebral body are greatly suggestive of spinal tuberculosis. MRI is also useful in detecting spinal canal lesions, especially concomitant spinal epidural lesions, intramedullary or extramedullary tuberculoma, arachnoiditis or pachymeningitis, and spinal cord edema and cavitation.

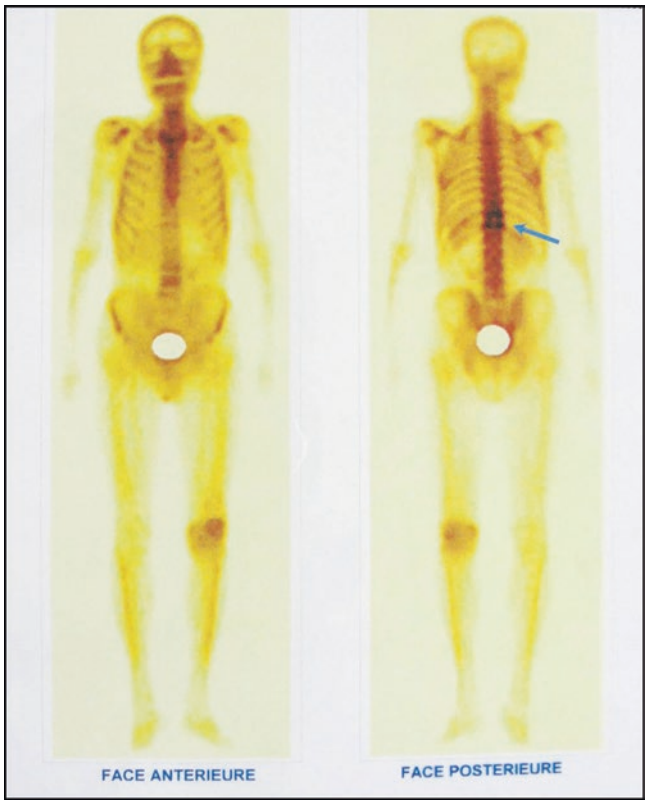
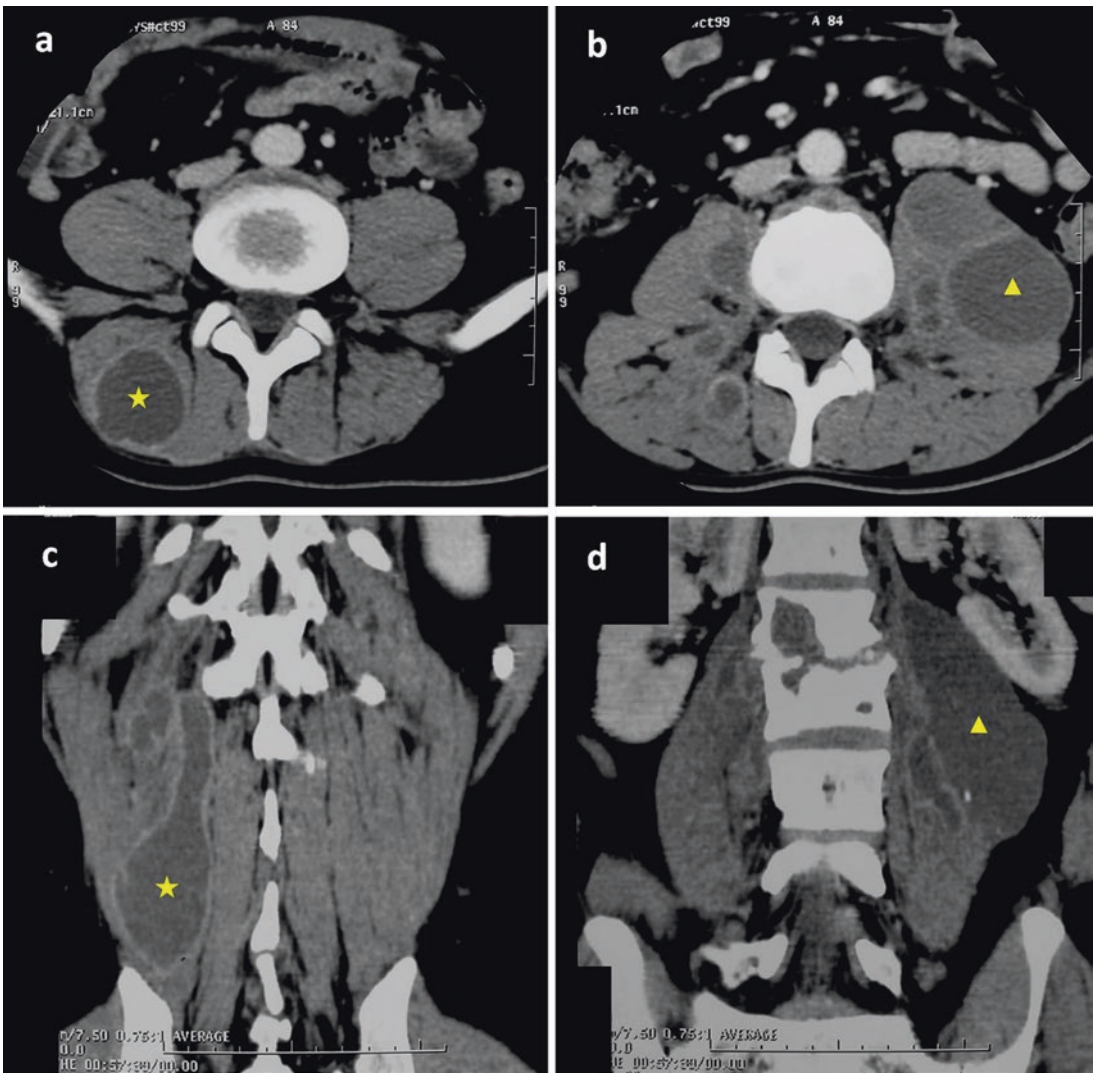



**Fig. 24.13** Case 24.4. Initial whole-body bone scintigraphy before treatment (using Tc-99m MDP bone scan) showing increased radiotracer localization in the vertebral body of T11 and T12 (*arrow*)



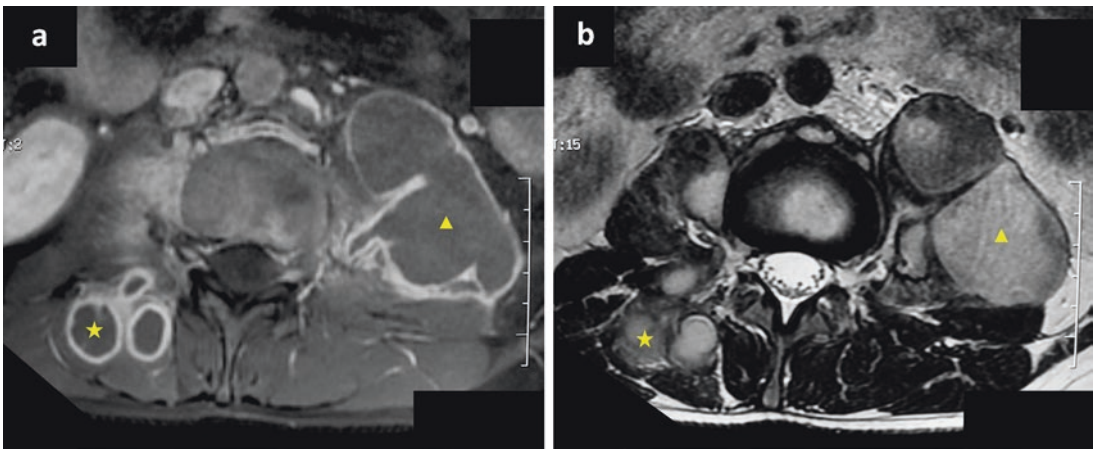



**Fig. 24.15** Case 24.5. Axial enhanced T1-weighted (**a**) and T2-weighted (**b**) MR images showing the right posterior paraspinal abscess (*star*) and the left multiloculated psoas abscess (*triangle*)

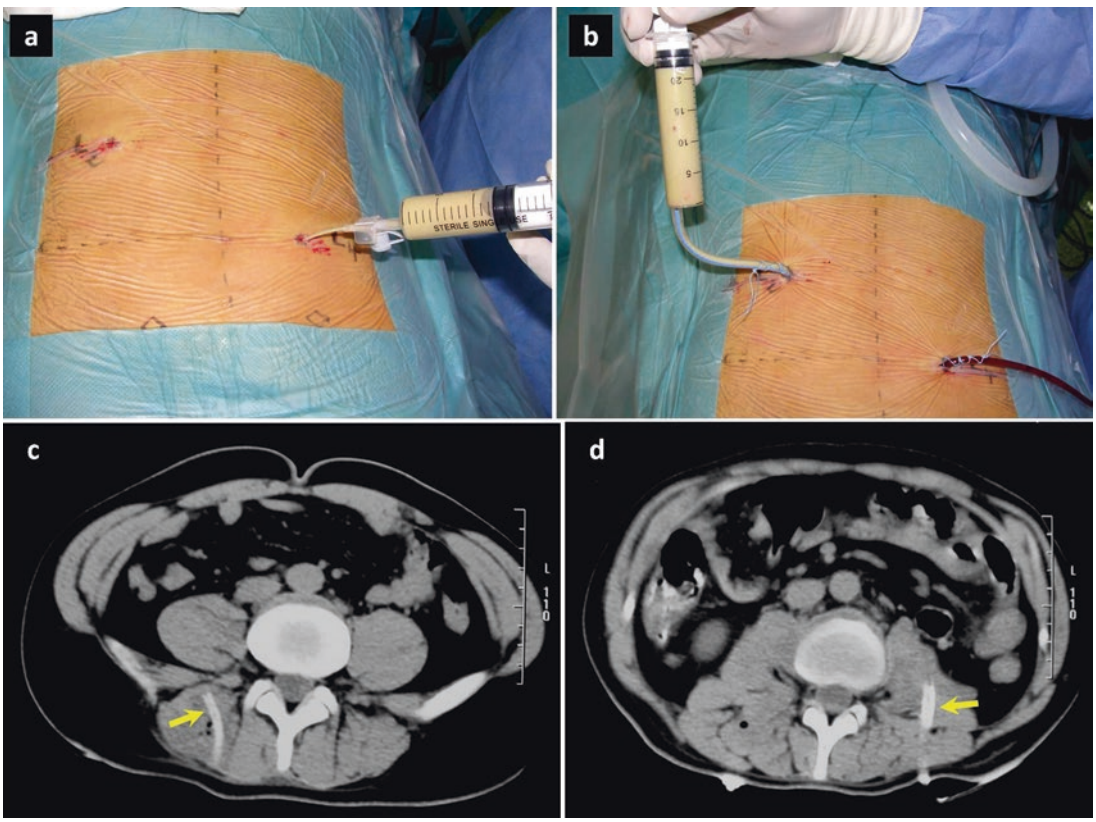



**Fig. 24.16** Case 24.5. Both abscesses were drained surgically under local anesthesia. Operative views of percutaneous aspiration/drainage of the purulent collections from the right posterior paraspinal abscess (**a**) and the left psoas abscess (**b**). Control CT scan showing the surgical drains left in situ (*arrows*) and complete resolution of the abscesses (**c, d**)

**Fig. 24.14** Case 24.5. Multiple paraspinal cold abscesses in a patient with L1–L2 Pott's disease. Axial (**a**, **b**) and coronal (**c**, **d**) post-contrast CT scans reveal a right posterior paraspinal abscess (*star*) and a left multiloculated psoas abscess (*triangle*)

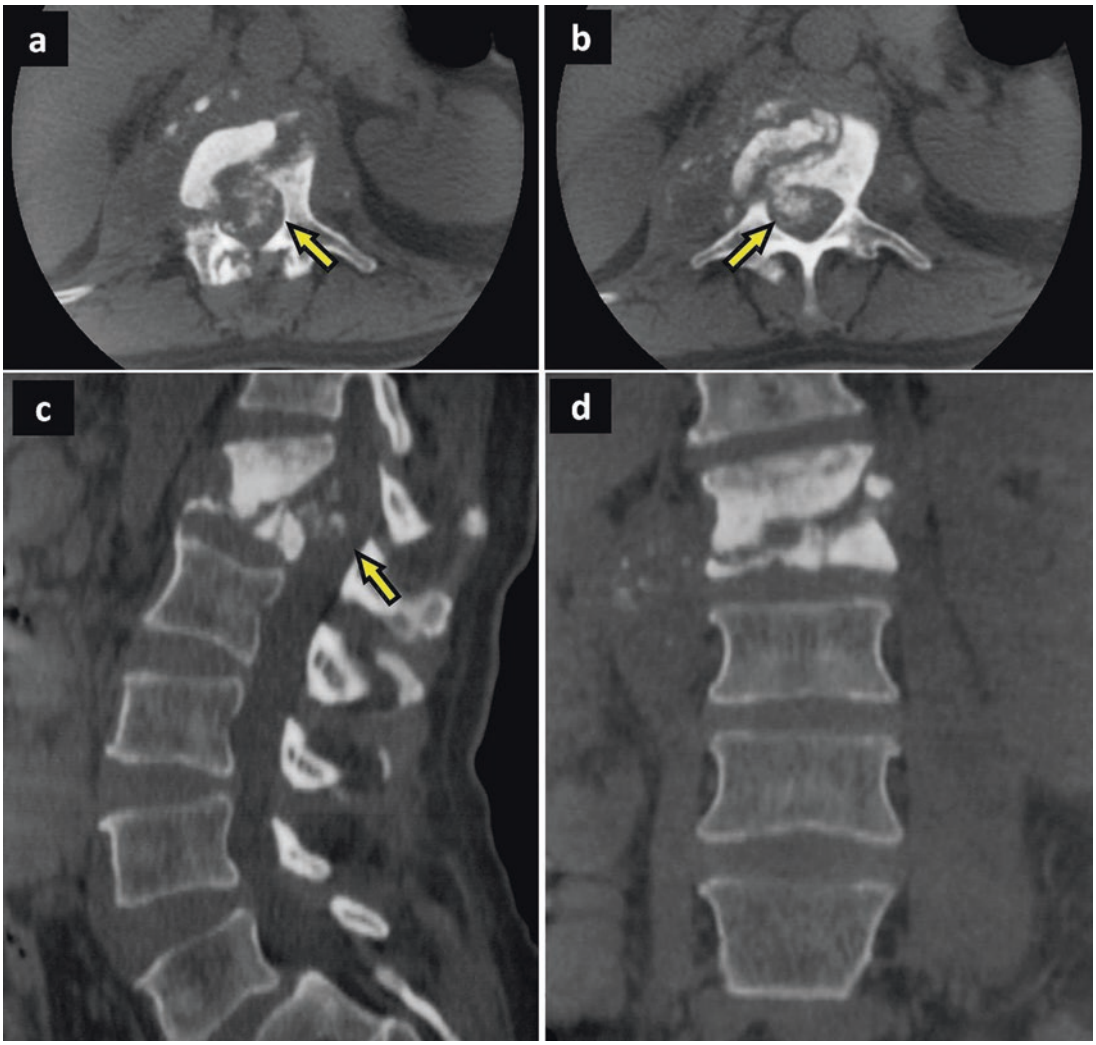
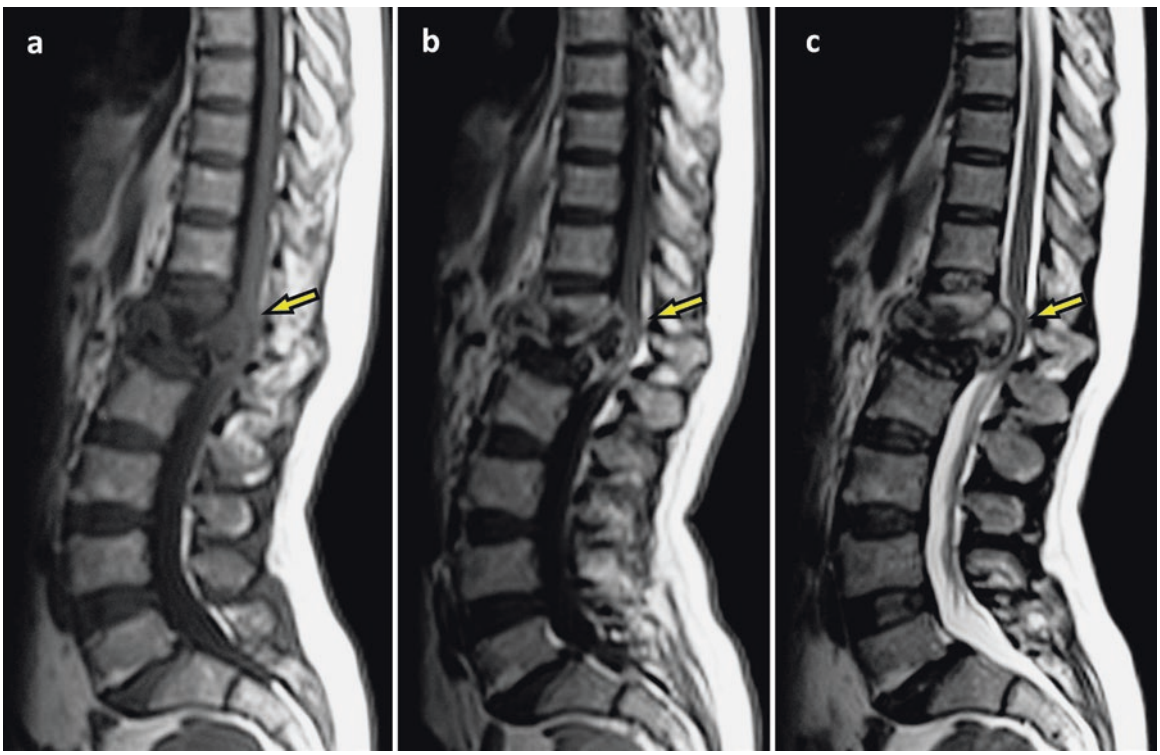



**Fig. 24.17** Case 24.6. Axial (**a**, **b**), sagittal (**c**), and coronal (**d**) spinal CT scans on bone windows, revealing vertebral body destruction and collapse of T12 and L1 with intracanalar extension. Note the paraspinal soft tissue calcifications



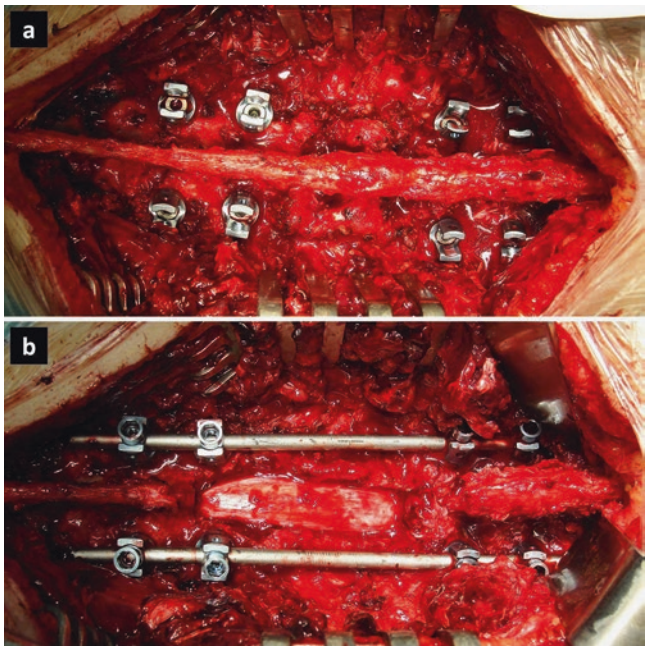



**Fig. 24.19** Case 24.6. Posterior intraoperative views: placement of pedicle screws under fluoroscopic guidance on T10, T11, L2, and L3 (**a**). Decompressive posterior laminectomy on L1–T12 with bilateral connecting rod screws (**b**)

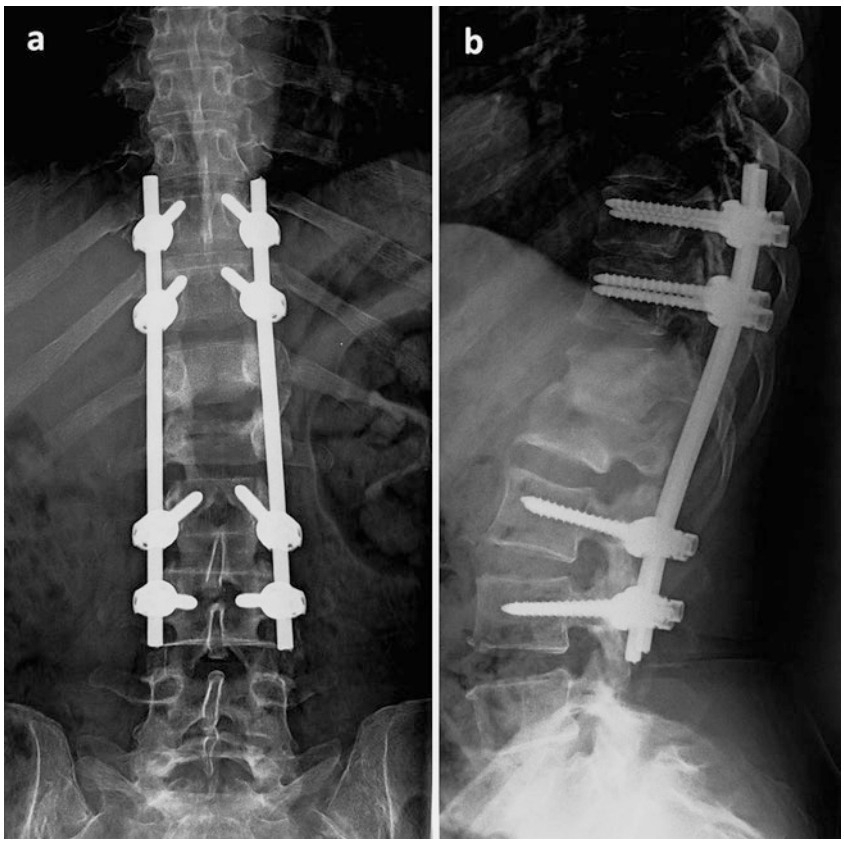



**Fig. 24.20** Case 24.6. (**a**, **b**) Postoperative spinal X-rays showing the posterior spinal instrumentation and the anterior vertebral body fusion

**Fig. 24.18** Case 24.6. Sagittal T1-weighted MR images before (**a**) and after (**b**) gadolinium administration, and T2-weighted image (**c**) showing the degree of spinal cord compression (*arrow*)

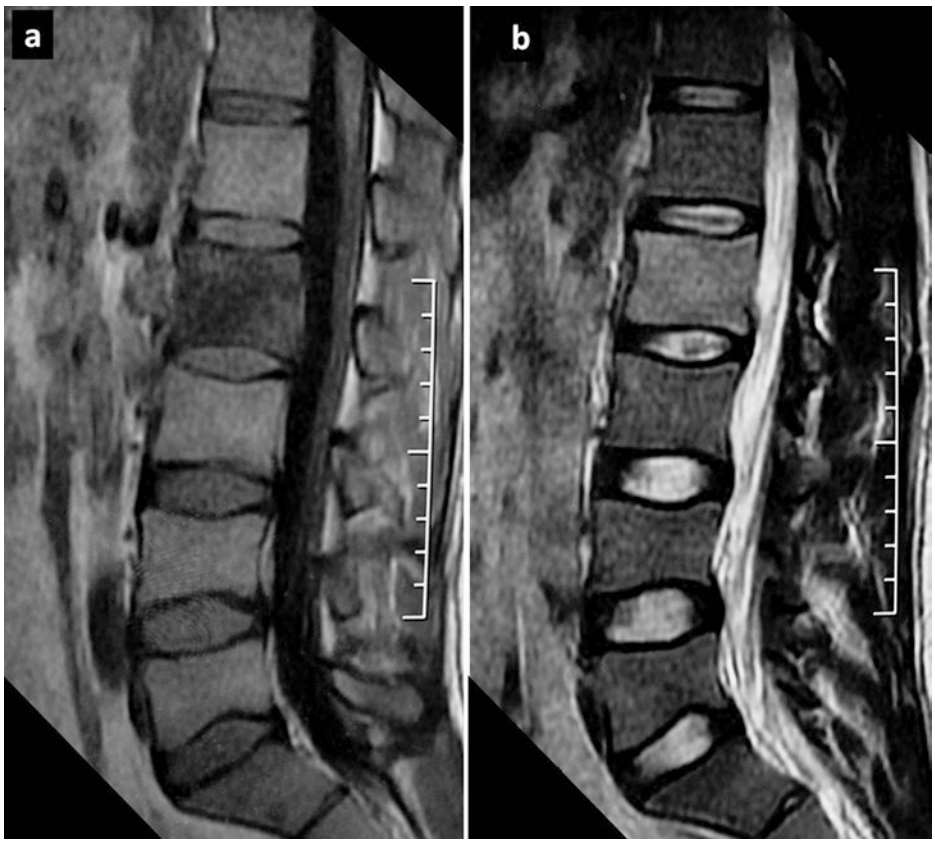



**Fig. 24.21** Case 24.7. Sagittal T1-weighted (**a**) and T2-weighted (**b**) MRI demonstrating modification of bone marrow signal intensity (early stage) of L2 without discal involvement. No diagnosis was made at this stage

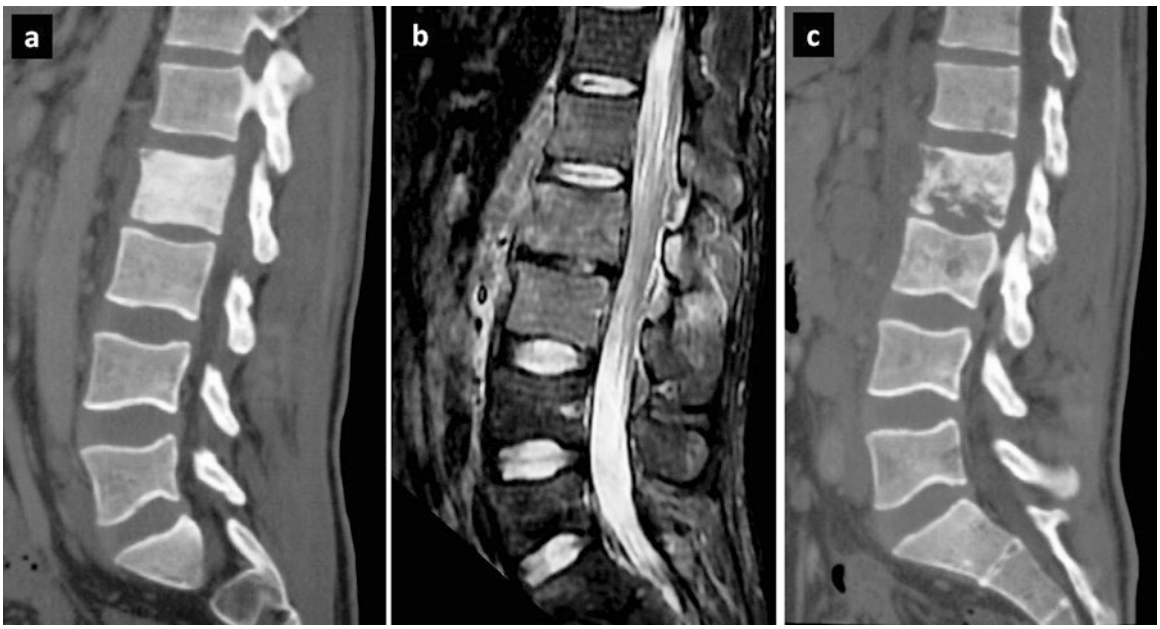



**Fig. 24.22** Case 24.7. Sagittal spinal CT scan (**a**) and T2-weighted MRI (**b**) 2 months later, showing loss of disc height with early discal alteration but without osseous destruction. A CT scan-guided biopsy was performed at this stage; the diagnosis of infectious spondylodiscitis was confirmed, but no pathogens were isolated. Despite nonspecific antibiotherapy, clinical symptoms worsened 1 month later. A new CT scan shows osseous destruction of the L2 vertebral body (**c**). The diagnosis of tuberculous disease was then made via an open surgical biopsy, and the patient had a good outcome under antituberculous therapy and external spinal immobilization

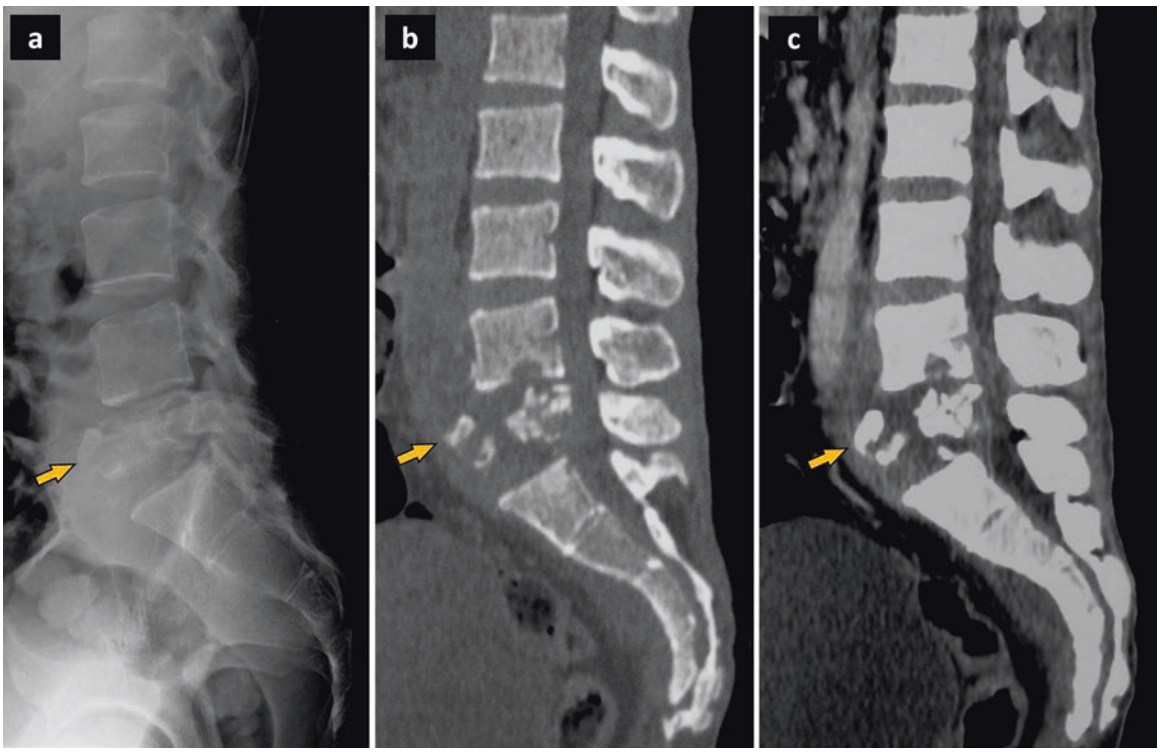



**Fig. 24.23** Case 24.8. This 41-year-old patient presented with bilateral lumbosciatalgia and signs of psoitis. Lateral plain radiography (**a**), sagittal CT scan on bone window (**b**), and post-contrast parenchymatous window (**c**) reveal anterior vertebral collapse with destruction of L5 and the inferior end plate of L4 (*arrow*)

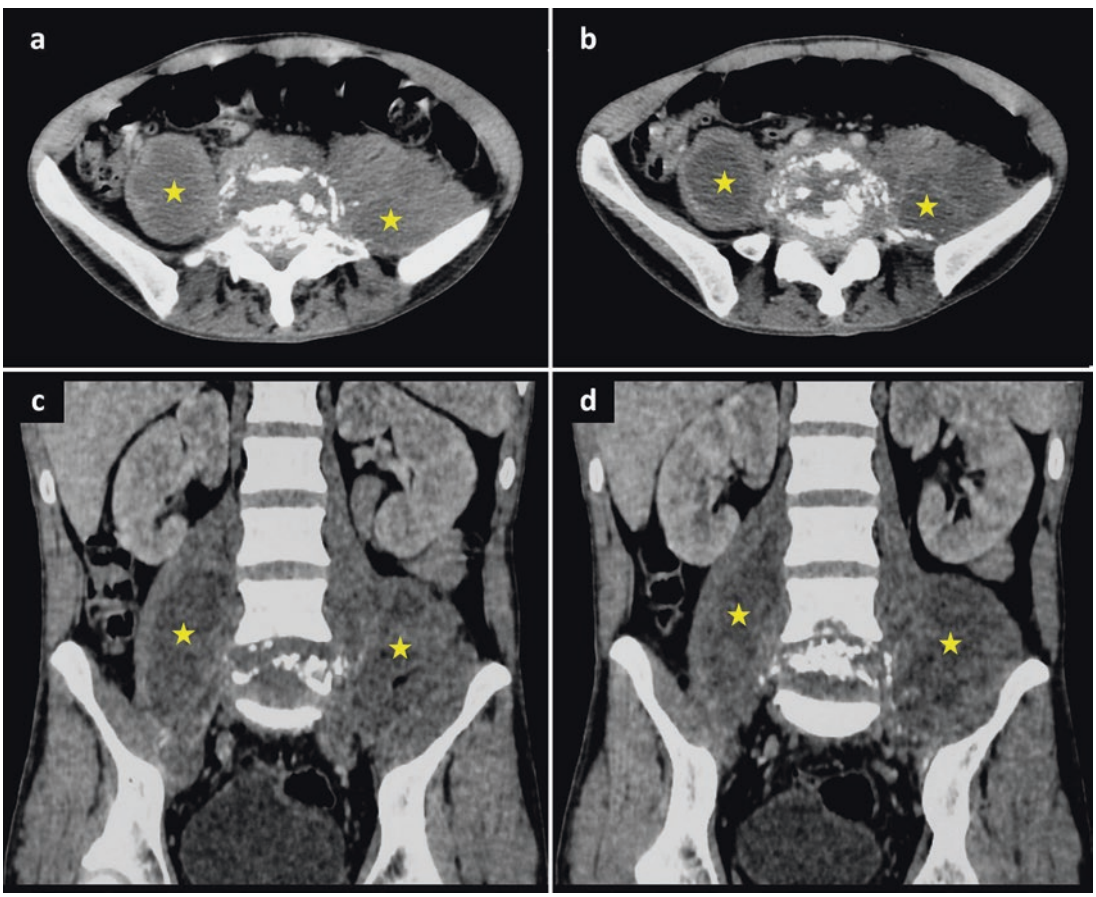



**Fig. 24.24** Case 24.8. Axial (**a**, **b**) and coronal (**c**, **d**) post-contrast CT scans reveal bilateral iliopsoas "cold" abscesses (*stars*) with peripheral enhancement

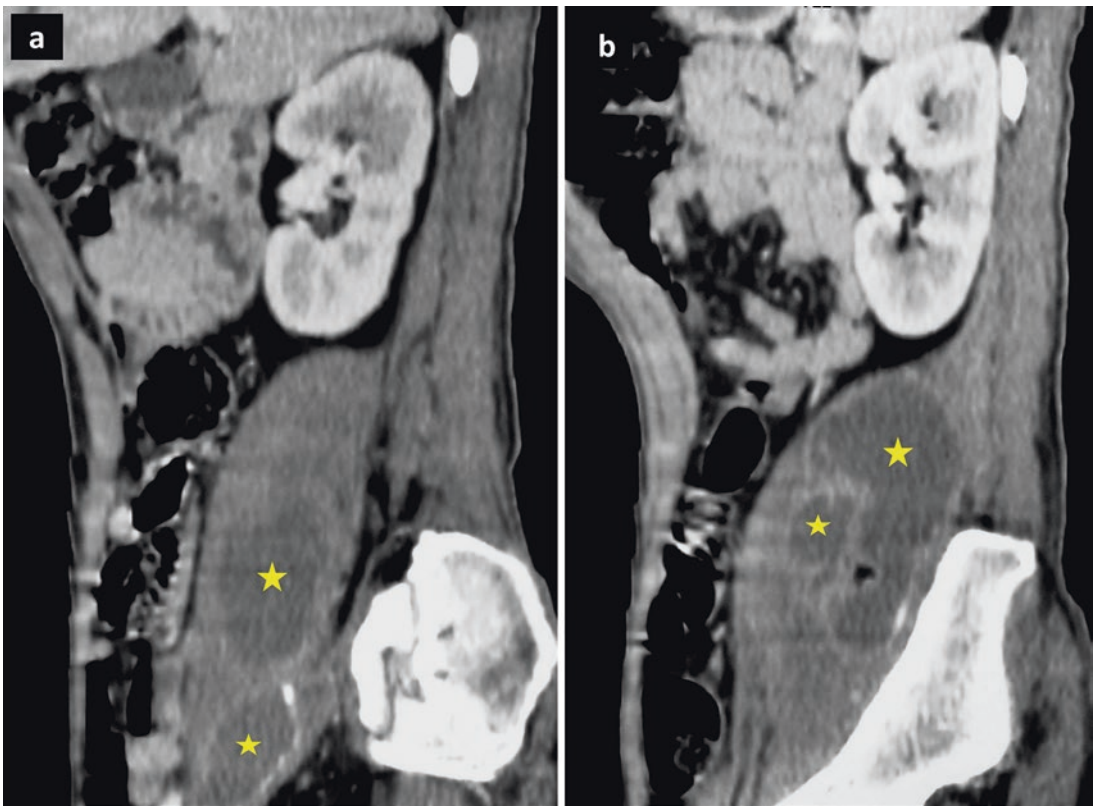



**Fig. 24.25** Case 24.8. Sagittal spinal CT scan following contrast administration, showing the right (**a**) and left (**b**) multiloculated iliopsoas abscesses (*stars*)

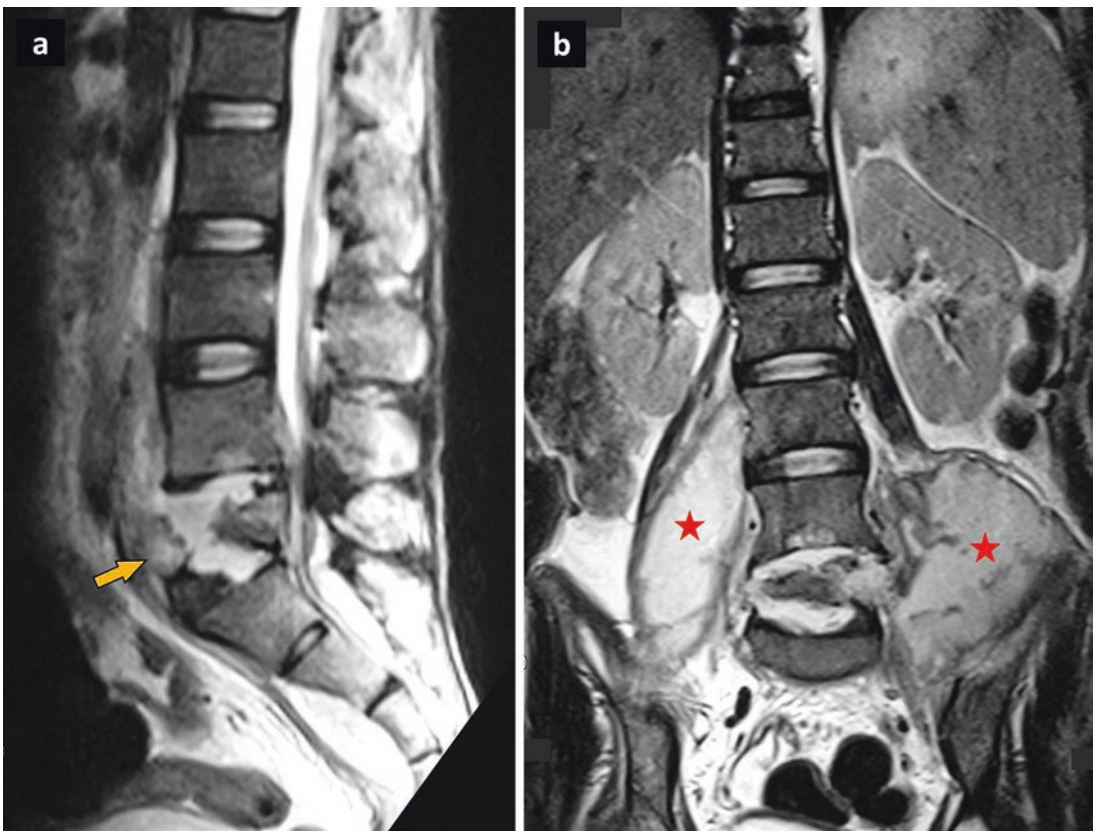



**Fig. 24.26** Case 24.8. Sagittal (**a**) and coronal (**b**) T2-weighted MRI revealing destruction of anterior part of L5 (*arrow*) with its adjacent intervertebral discs (**a**). Note the high signal intensity of the iliopsoas abscesses (*stars*) (**b**)

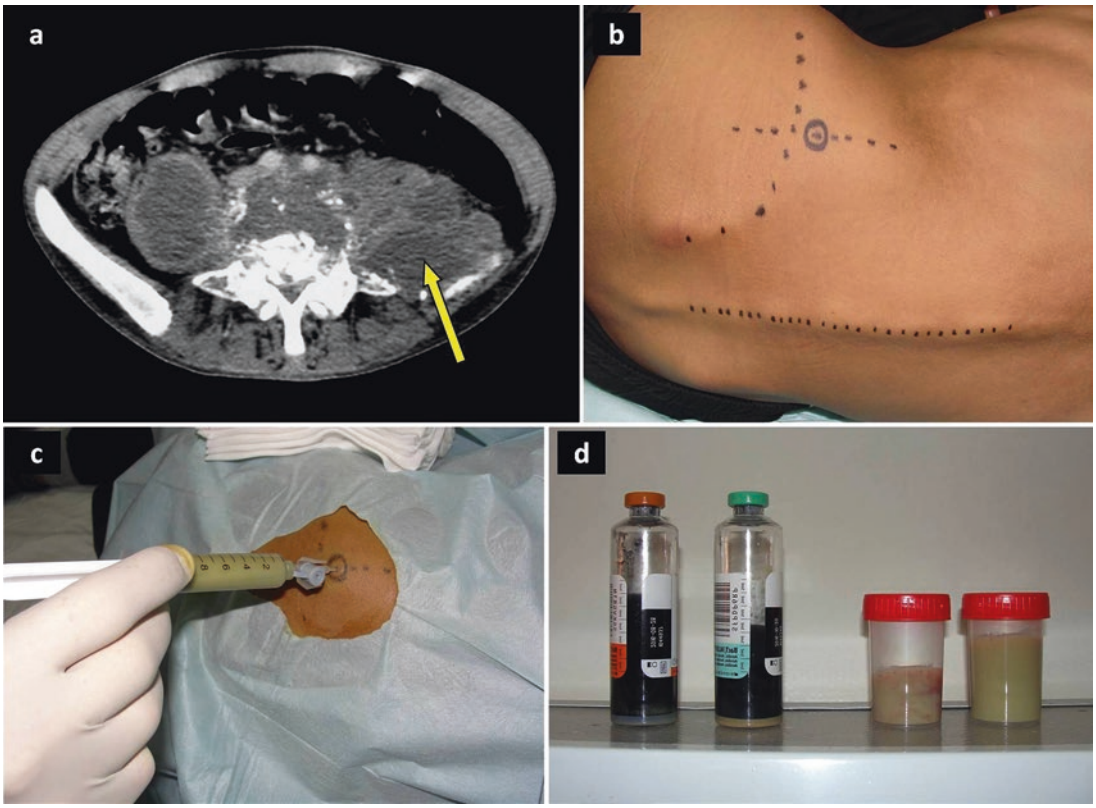



**Fig. 24.27** Case 24.8. Diagnosis was made following percutaneous aspiration and drainage of the psoas abscess in the left side. (*Mycobacterium tuberculosis* was identified by the GeneXpert MTB/RIF assay.) Axial CT scan after contrast injection (**a**), showing the left posterior approach (*arrow*). Preplanning surgical procedure with skin marks (**b**). Aspiration/drainage of the psoas abscess under local anesthesia (**c**). Samples and purulent material were collected and transported to the laboratories in sterile universal containers and specific bottles (**d**)

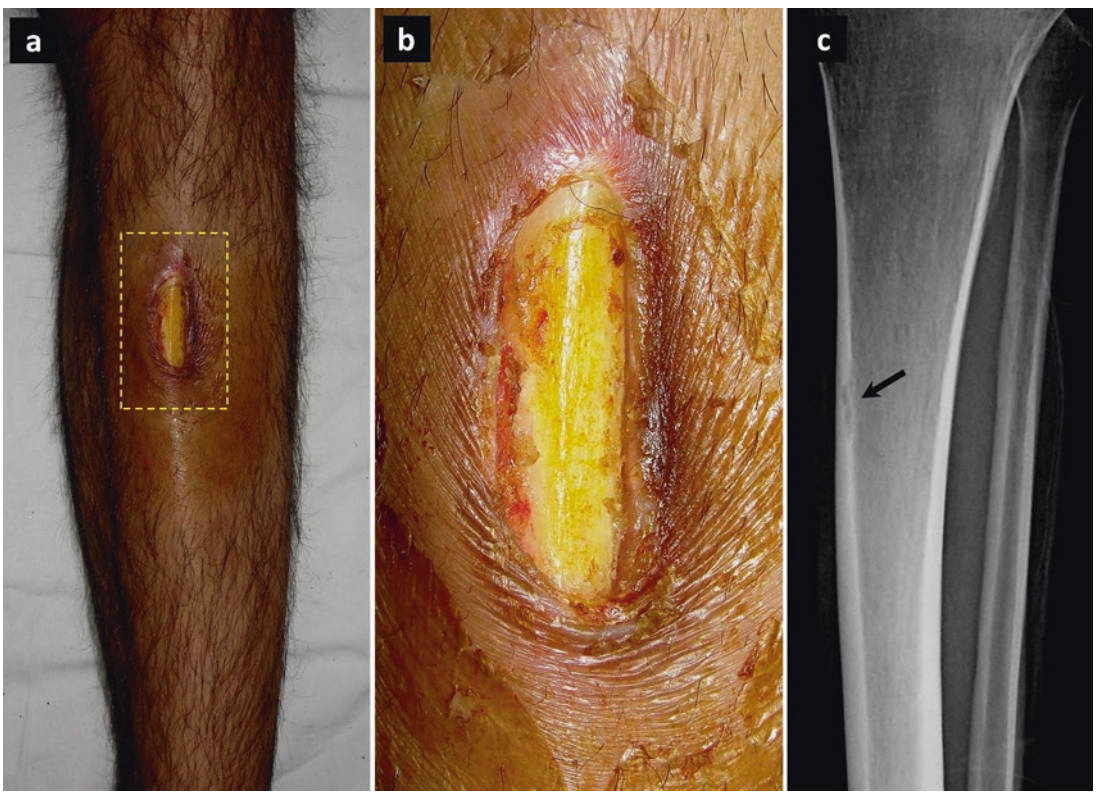



**Fig. 24.28** Case 24.8. This patient had also a chronic wound on the left leg (**a**, **b**), with the presumptive diagnosis of anterior diaphyseal tuberculosis on the tibia, shown on plain radiography (*arrow*) (**c**)

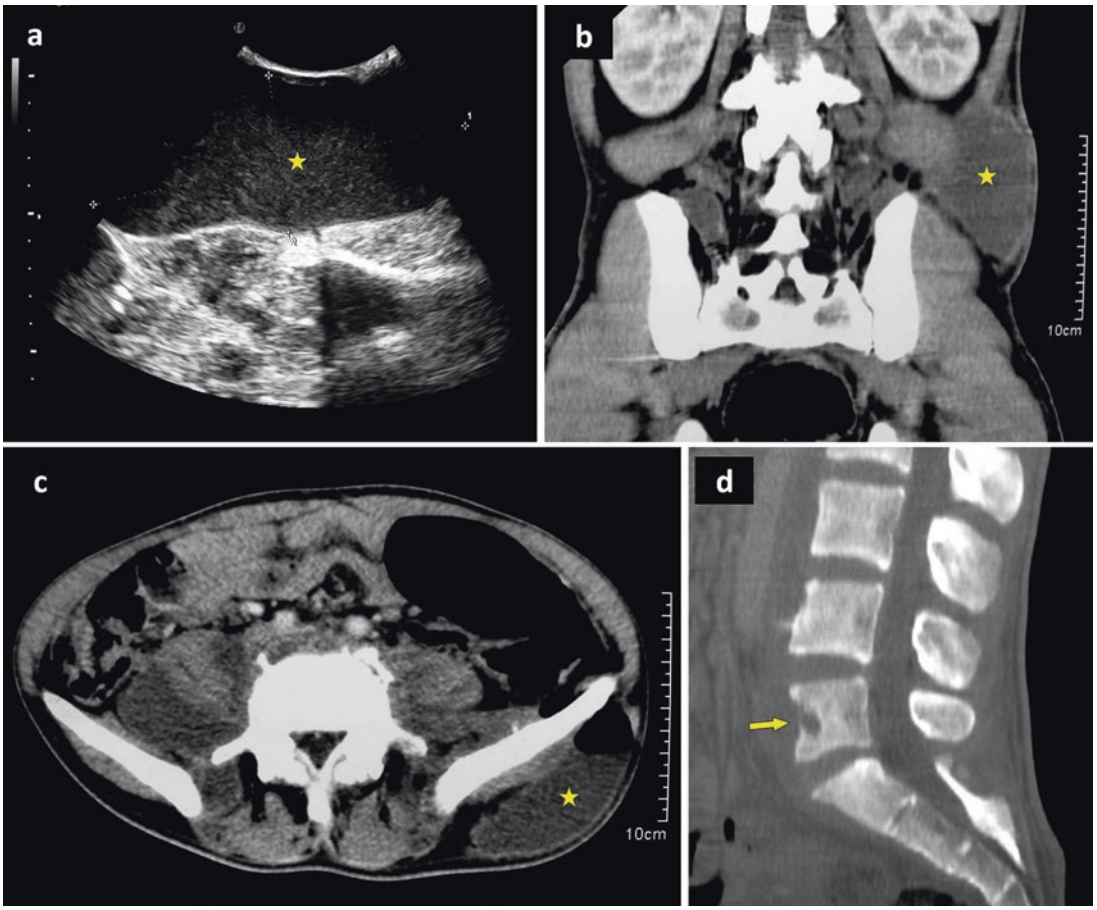



**Fig. 24.29** Case 24.9. This patient was observed and treated (with nonspecific antibiotherapy) for lumbar paraspinal abscesses. Ultrasonography of posterior lumbar paraspinal swelling in the left side (**a**), as well as coronal (**b**) and axial (**c**) abdominal CT scans and sagittal lumbar CT scan on bone window (**d**), shows a left lumbar paraspinal subcutaneous suppurative collection (*star*) and right psoas abscess. Note bone osteolysis of the anterior part of the vertebral body of L5 (*arrow*)

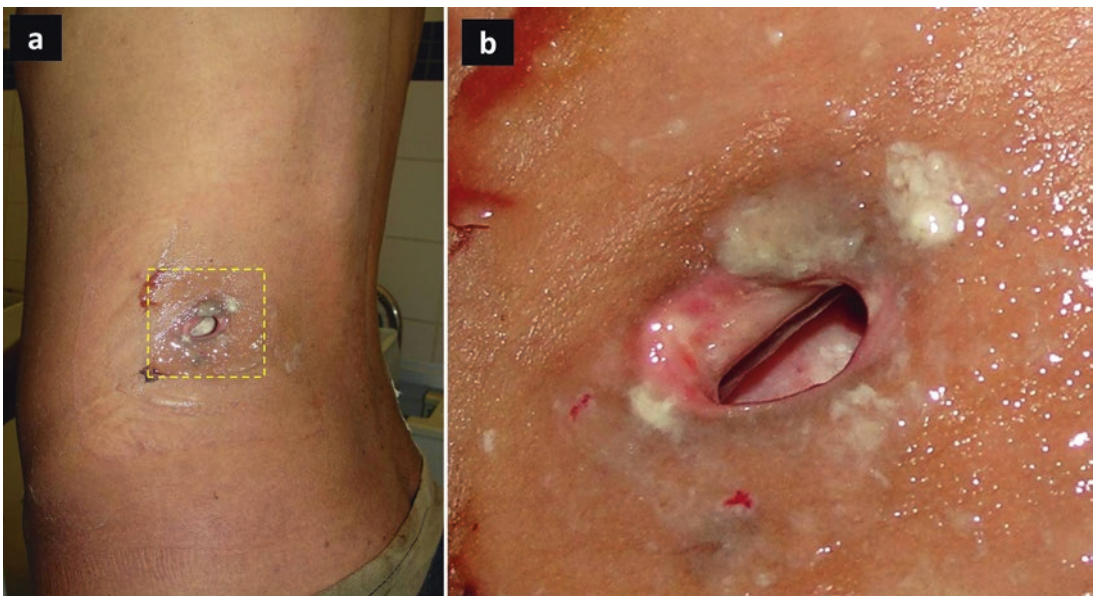



**Fig. 24.30** Case 24.9. Spontaneous fistulation of the lumbar paraspinal abscess in the left side (**a**, **b**) 1 month later. Microbiologic examination identified both *Mycobacterium tuberculosis* and *Staphylococcus aureus* (coinfectious disease)

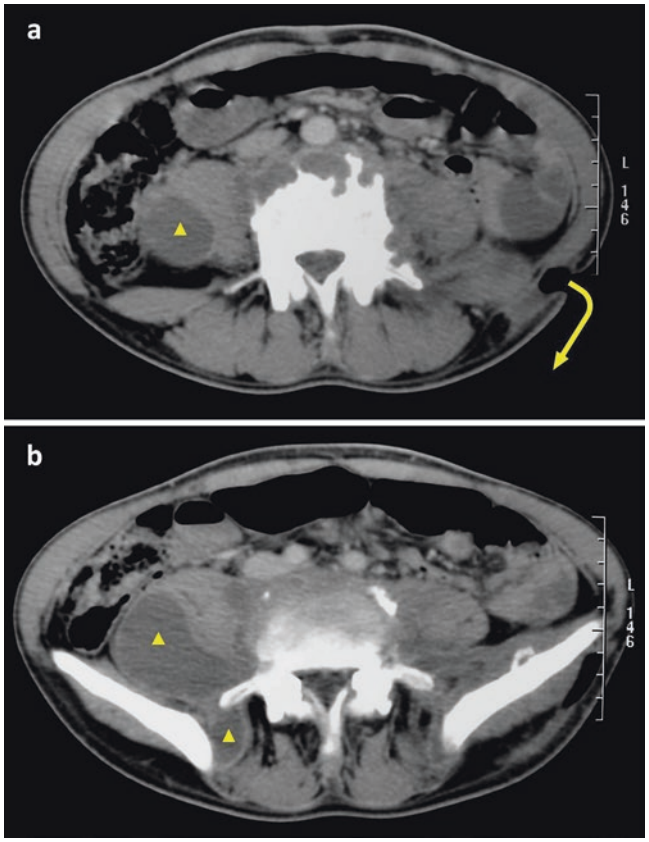



**Fig. 24.31** Case 24.9. (**a**, **b**) Axial abdominal post-contrast CT scan showing the location of the cutaneous fistula with its deep extension (*arrow*). Note the extension of the right iliopsoas abscess toward the back (*triangle*)

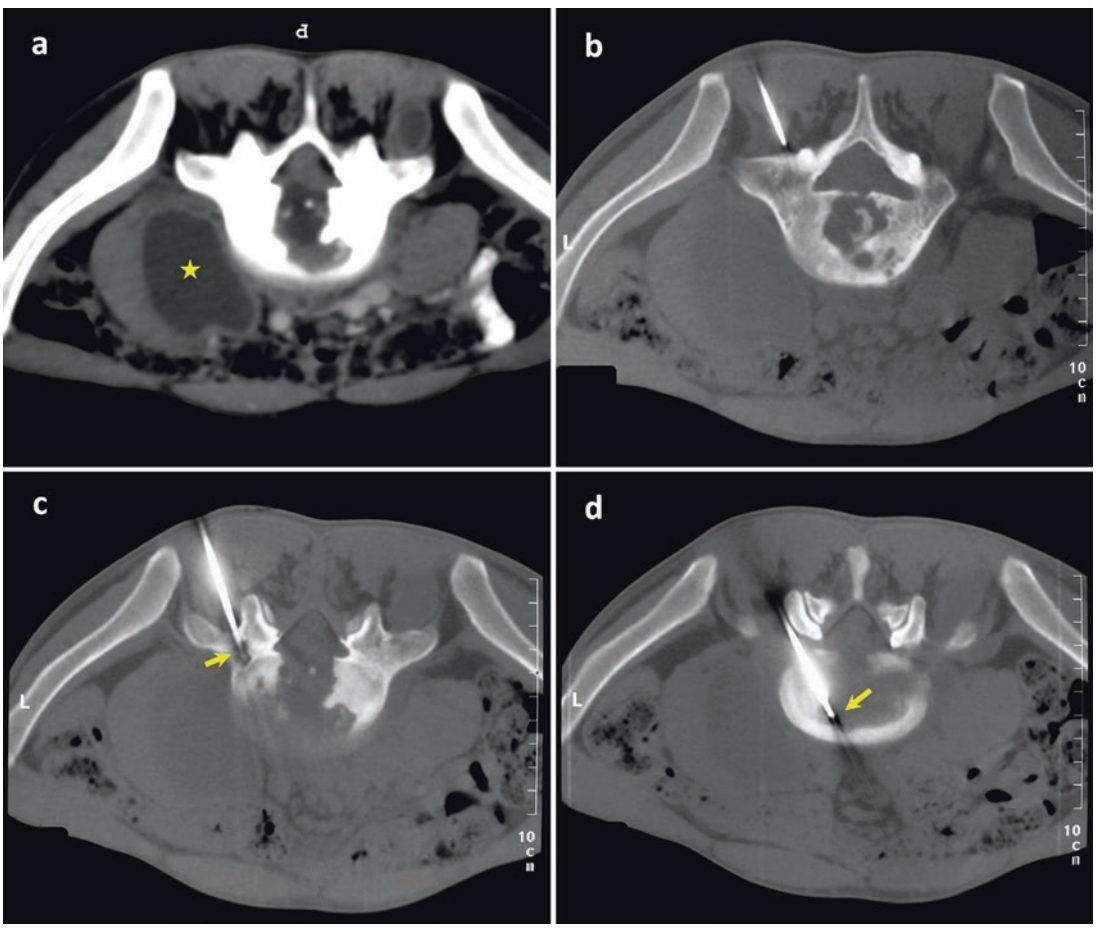



**Fig. 24.32** Case 24.10. Lumbosacral spondylodiscitis. Axial post-contrast CT scan on parenchymal window (**a**) and bone windows (**b–d**) showing the CT scan-guided procedure for biopsy (*arrow*) under local anesthesia. Note the left psoas abscess (*star*) (**a**)

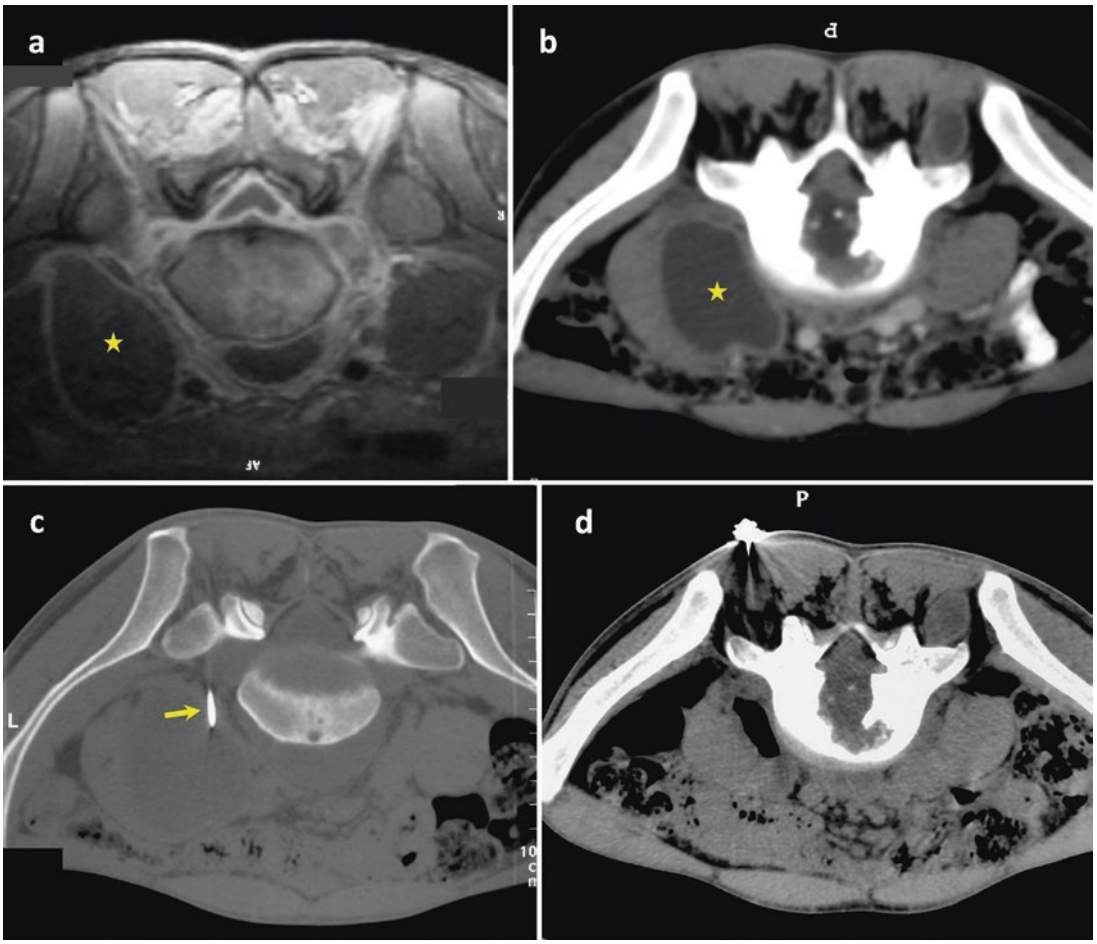



**Fig. 24.33** Case 24.10. Axial enhanced T1-weighted MRI (**a**) and post-contrast CT scan on parenchymal window (**b**) revealing the psoas abscess in the left side (*star*). Axial CT scan on bone window showing the CT scan-guided procedure for aspiration/drainage of the psoas abscess (*arrow*) (**c**). Nearly complete drainage of the abscess on control CT scan (**d**)

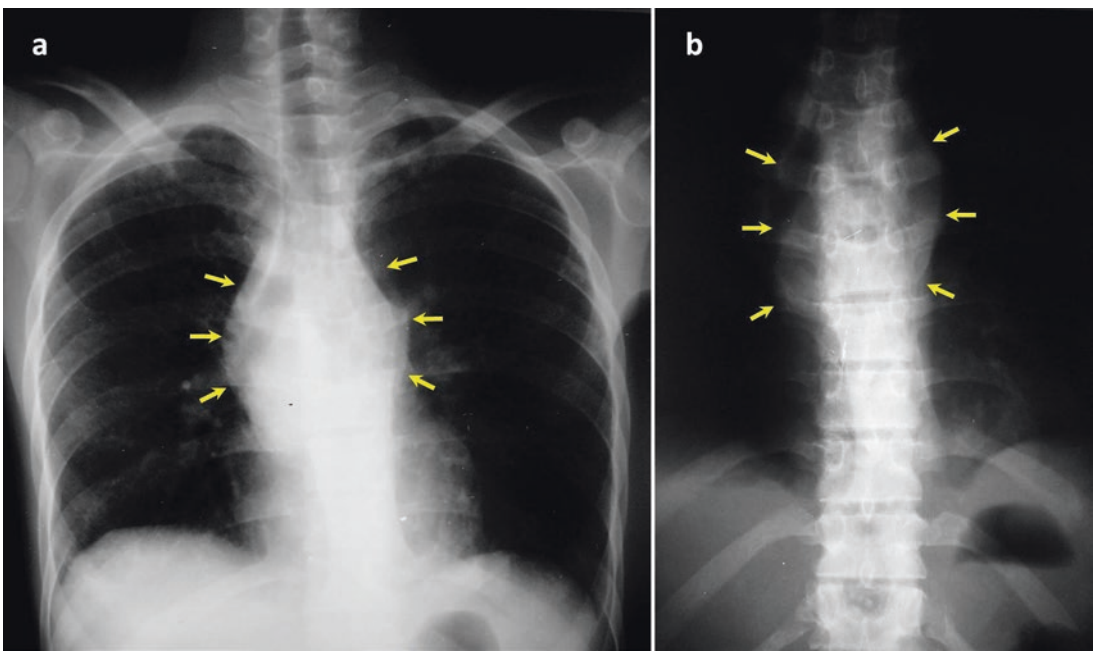



**Fig. 24.34** Case 24.11. Anteroposterior chest radiogram (**a**) and spinal thoracic radiogram (**b**) showing a fusiform or spindle-shaped radiodense shadow (*arrows*)

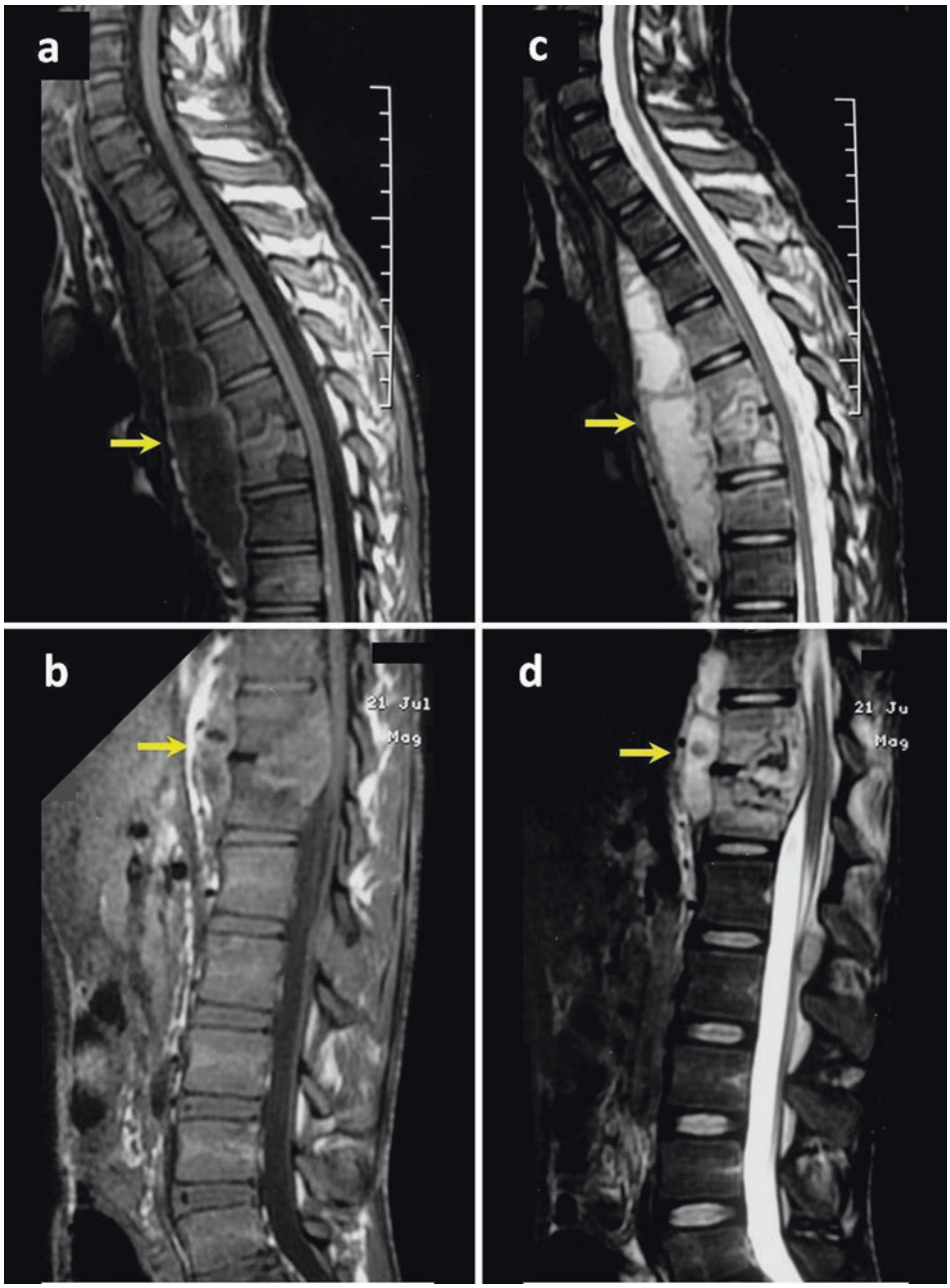



**Fig. 24.35** Case 24.11. Sagittal spinal cervicothoracic (**a**) and thoracolumbar (**b**) T1-weighted MR images and T2-weighted images (**c**, **d**) reveal bifocal, noncontiguous spondylodiscitis on the T11–T12 and T6–T7 levels. Note the adjacent prevertebral, multiloculated "cold" abscesses (*arrow*)

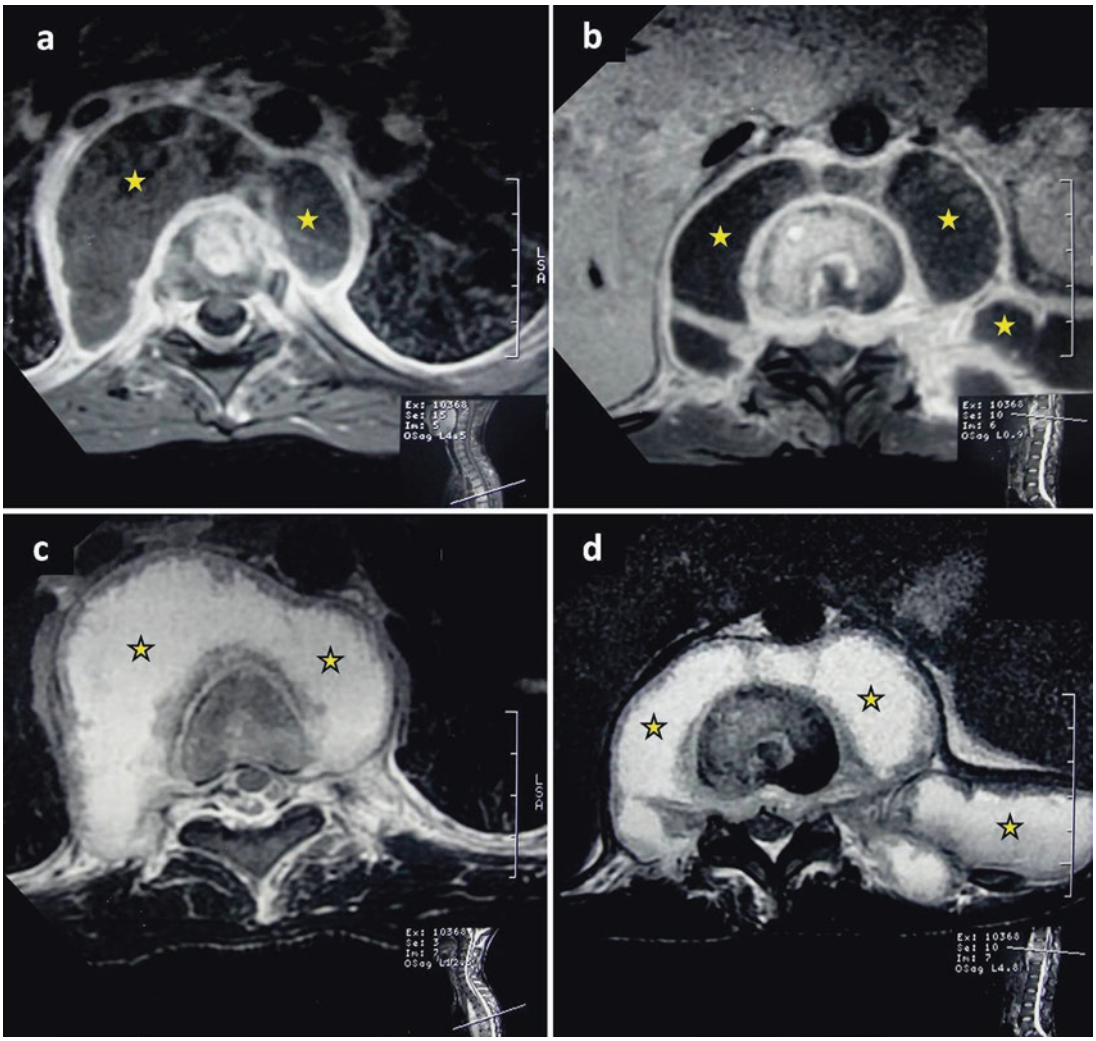



**Fig. 24.36** Case 24.11. Axial spinal enhanced T1-weighted images through the T7 (**a**) and T11 (**b**) levels, and T2-weighted images (**c**, **d**) show the paravertebral collections (*stars*)

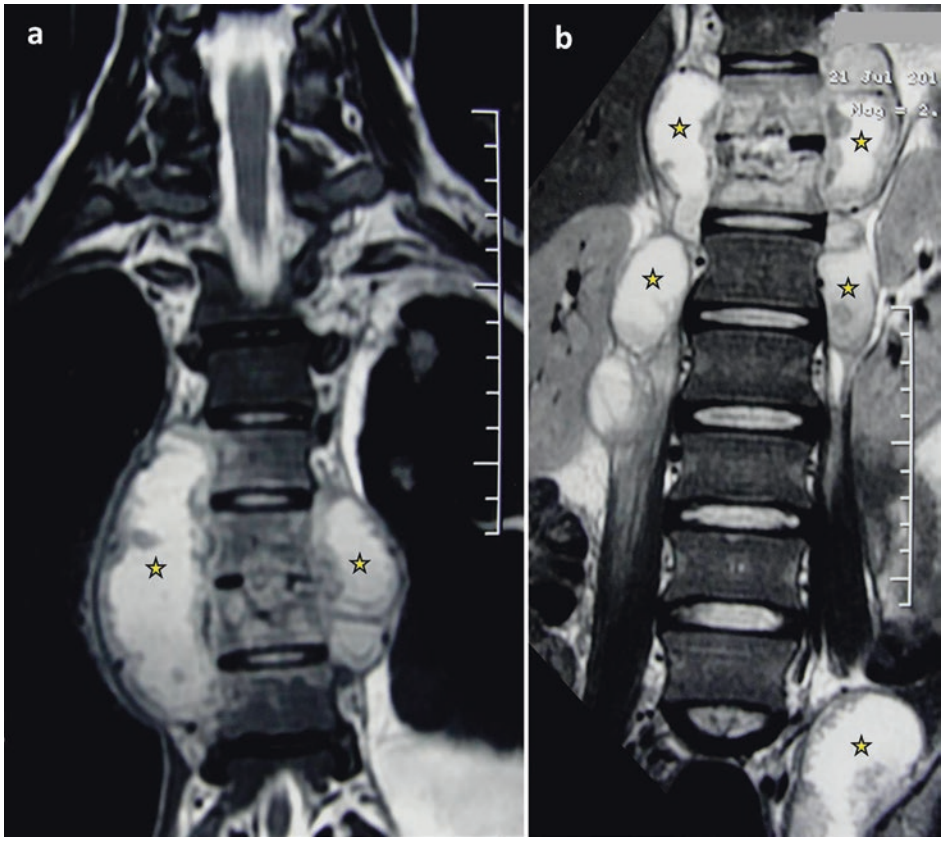



**Fig. 24.37** Case 24.11. Coronal spinal cervicothoracic (**a**) and thoracolumbar (**b**) T2-weighted MR images reveal the fusiform aspect of the paravertebral "cold" abscesses (*stars*)

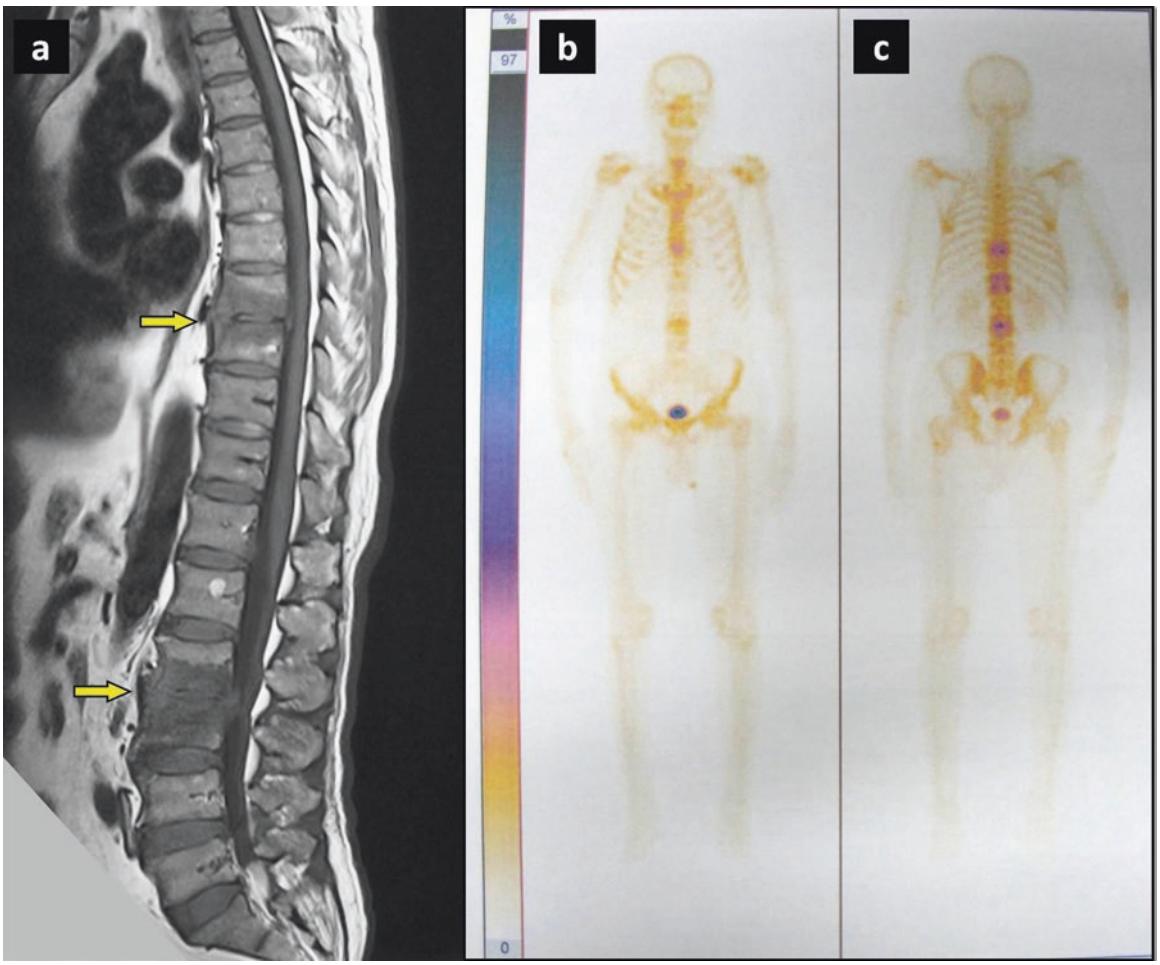



**Fig. 24.38** Case 24.12. (**a**) Sagittal spinal T1-weighted MR image showing modification of bone marrow signal intensity with vertebral body involvement at the T8–T9 and L2–L3 levels. (**b**, **c**) Whole-body bone scintigraphy using Tc-99m MDP bone scan, revealing increased radiotracer localization in the vertebral body at T8, T9, T11, T12, L2, and L3. Note that T11–T12 localization was not seen on the MRI

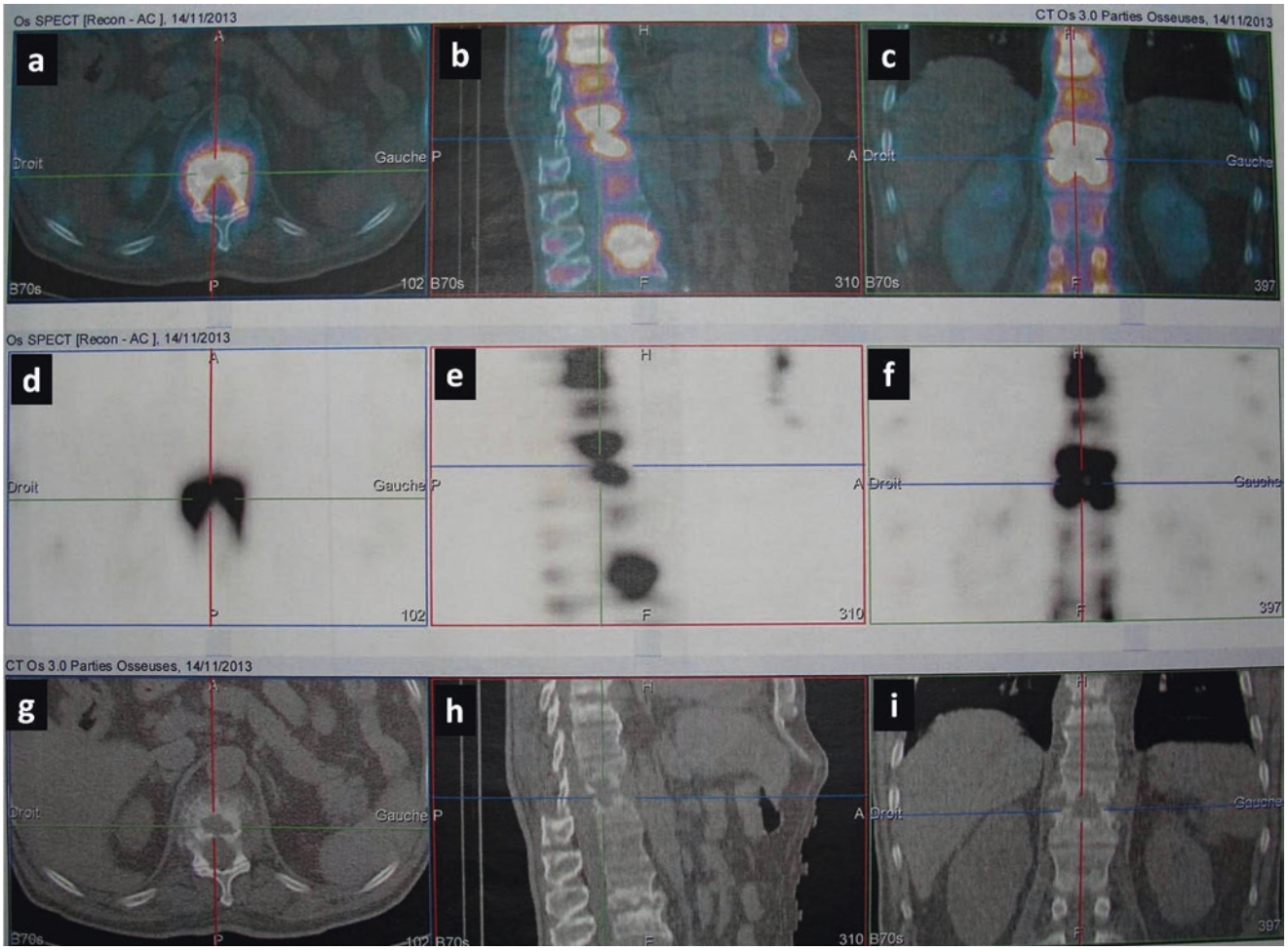



**Fig. 24.39** Case 24.12. Multifocal vertebral involvement seen on SPECT/CT (**a–c**), SPECT (**d–f**), and CT scan images (**g–i**). Prostate cancer bone metastases were suspected in this elderly man, but there was no improvement despite chemotherapy and radiation therapy

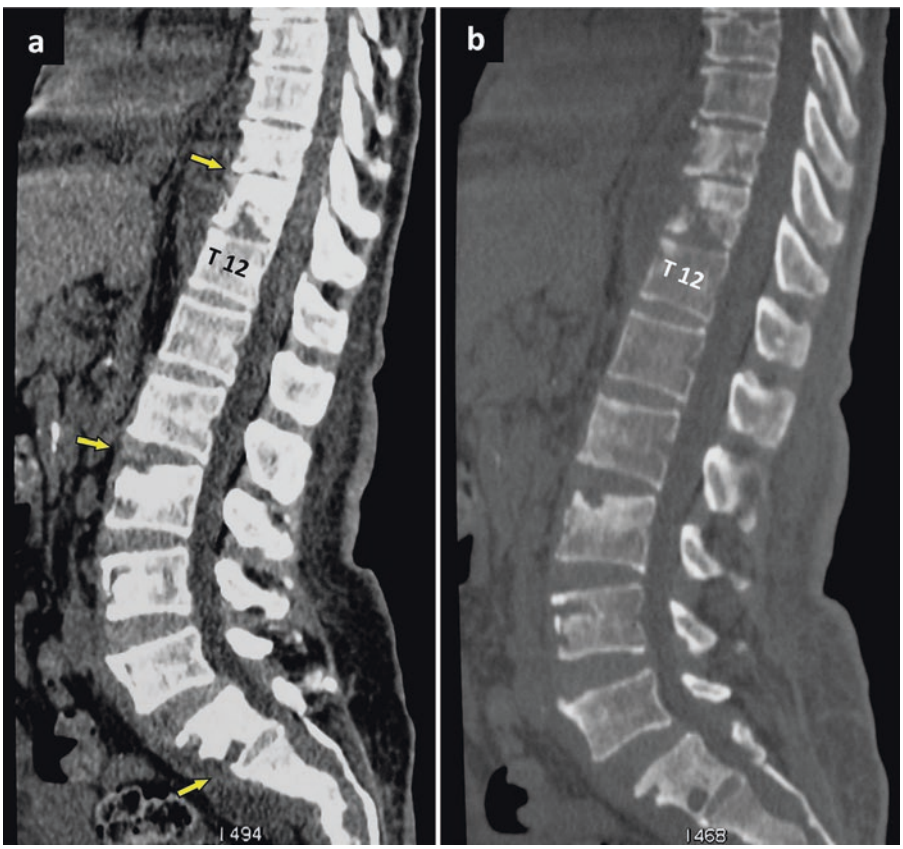



**Fig. 24.40** Case 24.12. Sagittal spinal CT scans on parenchymal window (**a**) and bone window (**b**) performed 3 months later reveal extension of the spinal lesions (*arrows*)

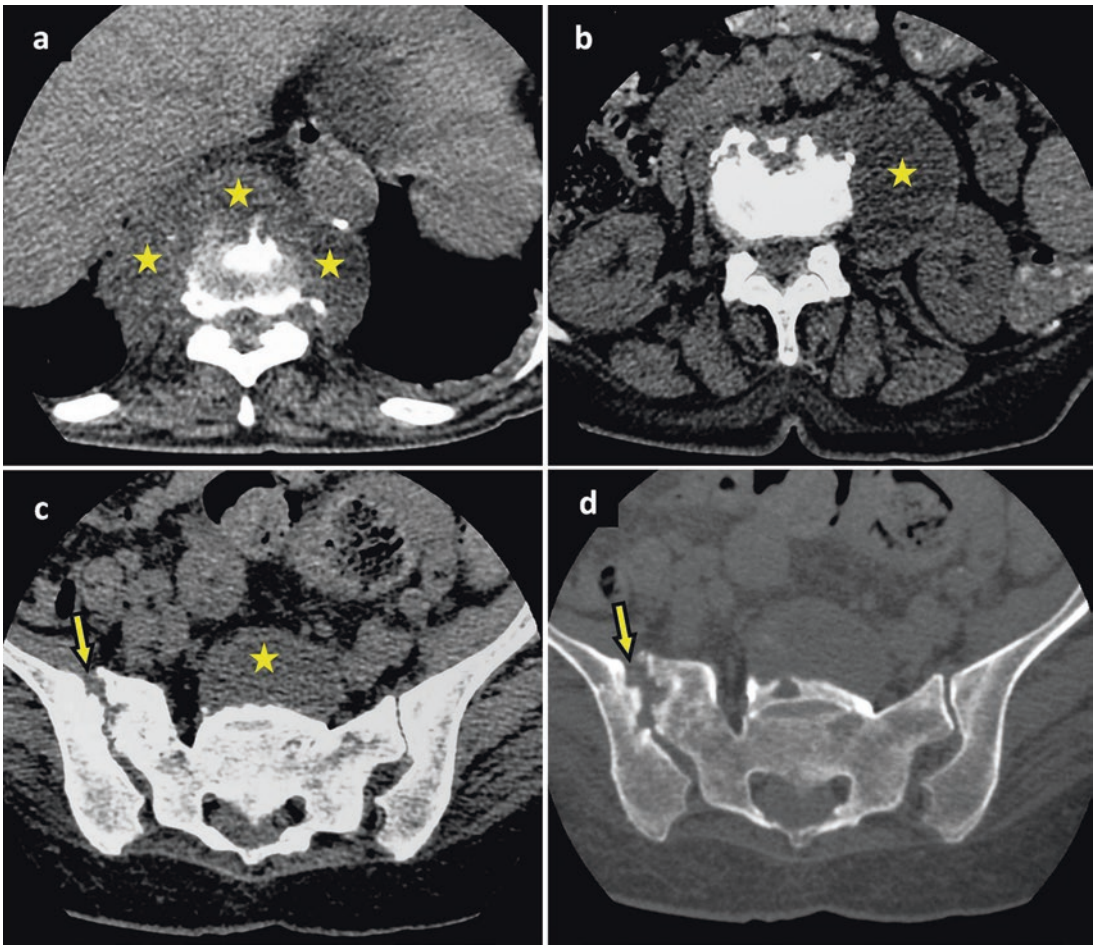



**Fig. 24.41** Case 24.12. Axial spinal CT scan through T10 (**a**), L2 (**b**), and S1 (**c**) levels, revealing vertebral body osteolysis with paravertebral collections (*stars*) and concomitant sacroiliitis in the right side (*arrow*)

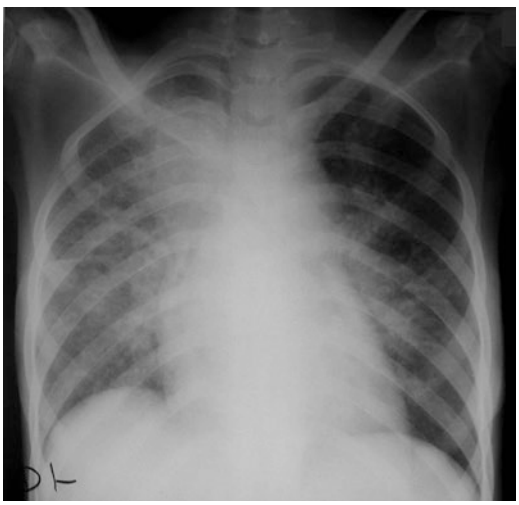


**Fig. 24.42** Case 24.12. The patient also had pulmonary involvement seen on chest radiography. He was then referred to our department due to neurologic deterioration (cauda equina syndrome). Surgery was indicated for diagnosis purposes and decompression of neural structures. The diagnosis of tuberculosis was confirmed on the basis of histopathologic examination and culture of *Mycobacterium tuberculosis*

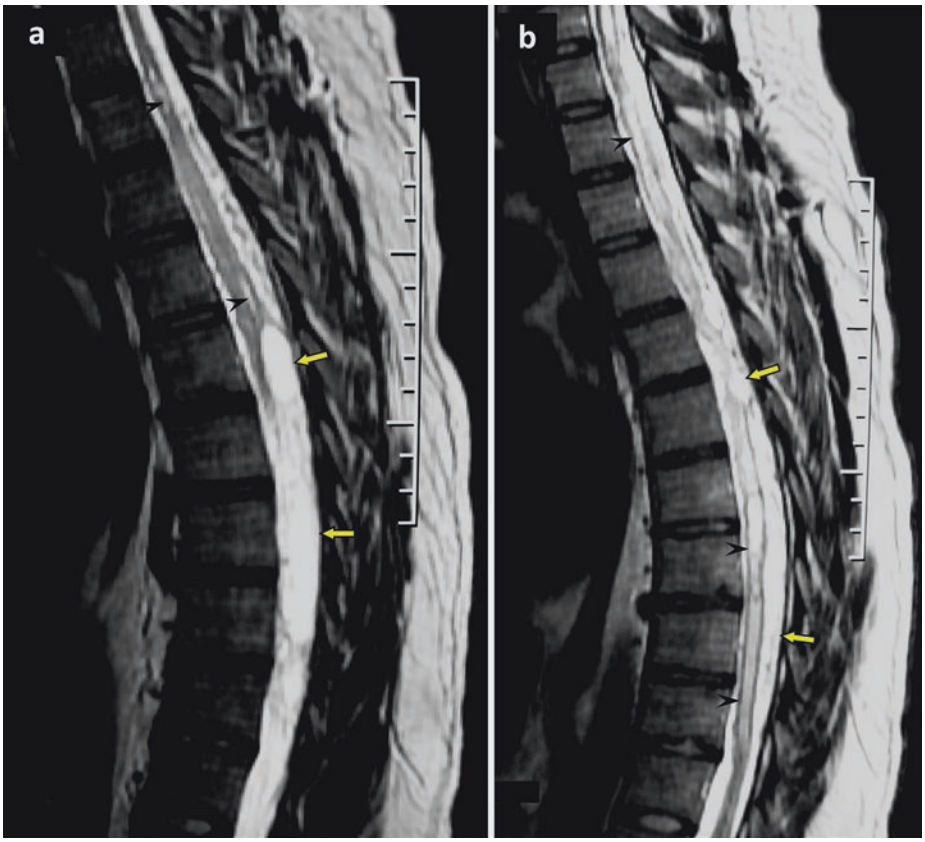



**Fig. 24.43** Case 24.13. (**a**, **b**) Spinal tuberculous arachnoiditis in a 38-year-old man with a history of tuberculous meningitis 2 years previously. Sagittal T2-weighted MRI showing multiloculated cystic lesions surrounding the thoracic spinal cord (*arrows*). Note the high signal within the spinal cord (*arrowheads*). Changes of arachnoiditis may be focal, multifocal, or diffuse

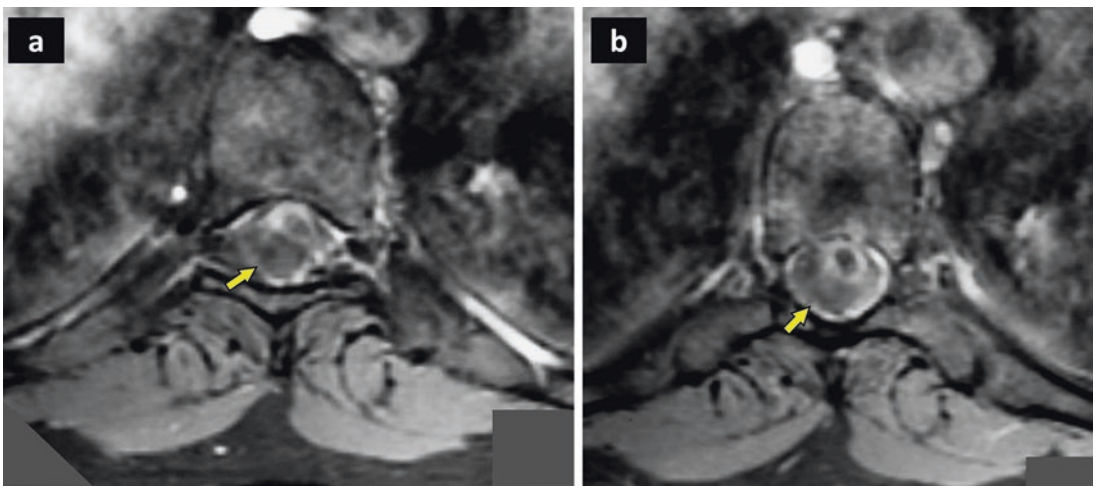



**Fig. 24.44** Case 24.13. (**a**, **b**) Axial T2-weighted MRI revealing posterior multiloculated subdural cysts (*arrow*) surrounding the spinal cord parenchyma. Frequently, there is vascular involvement with periarteritis and occlusion of small vessels. Neuronal structures are damaged by direct compression and by ischemia

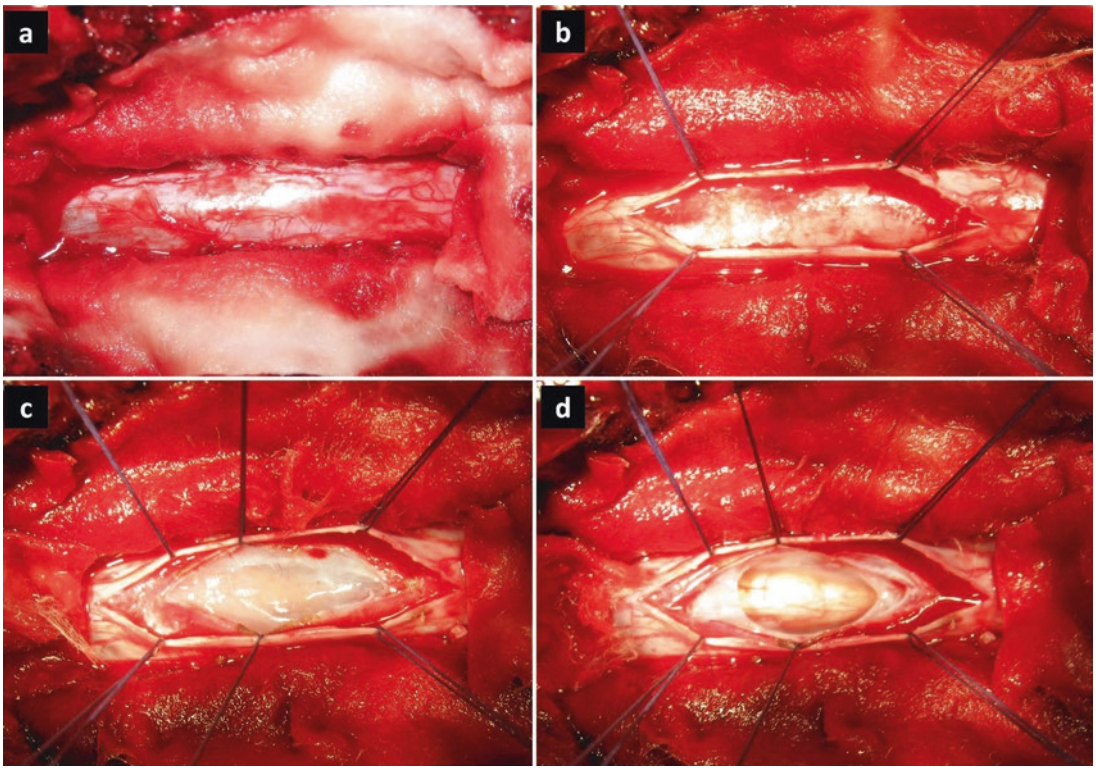



**Fig. 24.45** Case 24.13. Intraoperative views after limited posterior thoracic laminectomy (**a**–**d**). The dura mater was opened (**b**). Note the important adhesive and arachnoid thickening with fibrosis (a weblike appearance) (**c**). Spinal cord appearance following gentle arachnoid dissection (**d**)

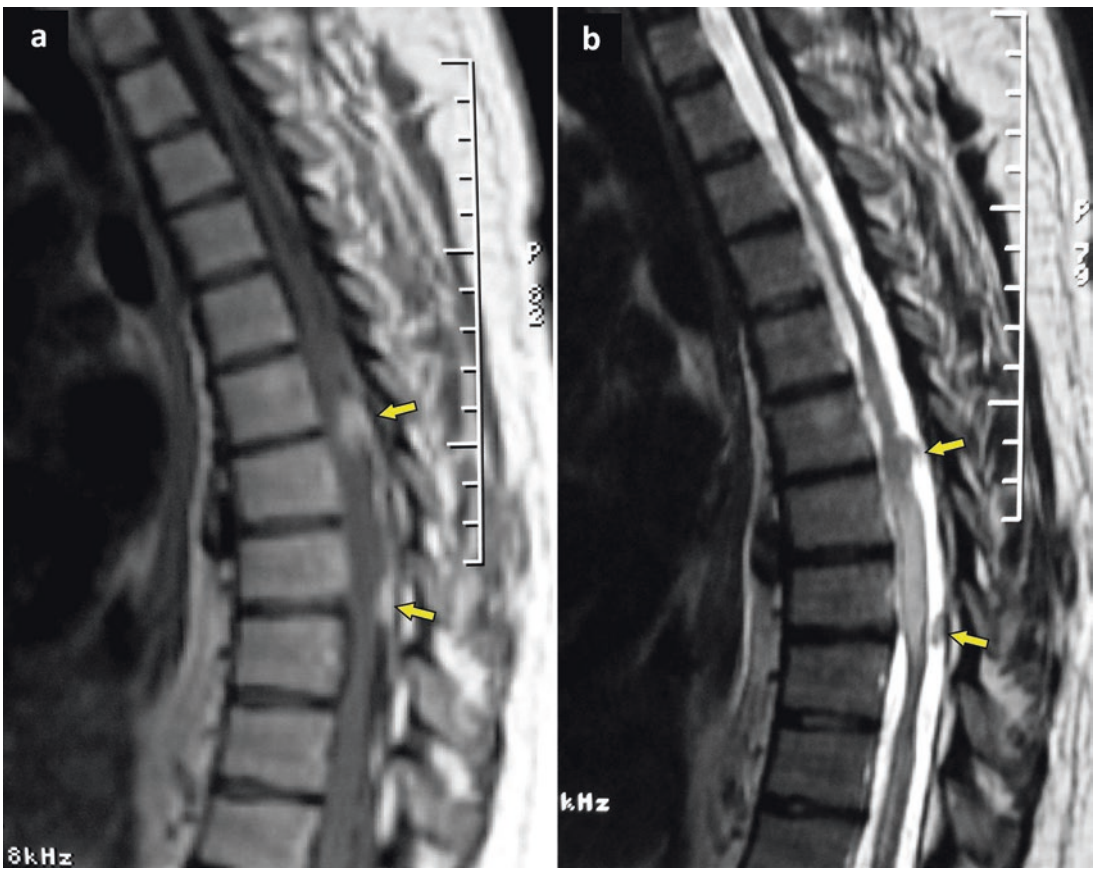



**Fig. 24.46** Case 24.14. Thoracic spinal intradural extramedullary tuberculomas. Sagittal enhanced T1-weighted MR image (**a**) and T2-weighted sequence (**b**) showing a T5–T7 myelitis with two intradural extramedullary tuberculomas on T5–T6 and T7–T8 vertebral levels (*arrows*)

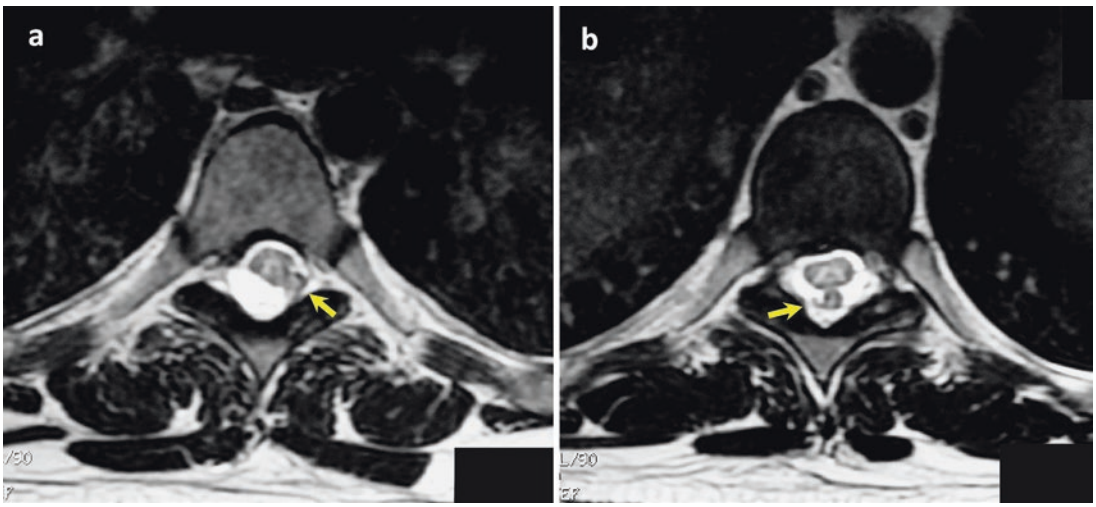



**Fig. 24.47** Case 24.14. Axial spinal T2-weighted images showing the two subdural tuberculomas (*arrows*) on the T5–T6 (**a**) and T7–T8 (**b**) vertebral levels

Nuclear medicine imaging is more sensitive in early detection of suspected spondylodiscitis before much more spinal destruction occurs. Infection usually causes a hot spot, but avascular bone fragments may produce a cold spot. Single-photon emission computed tomography (SPECT) is more accurate than conventional scintigraphy. SPECT scan can be further improved by adding CT scanning. Recently, fluorodeoxyglucose positron emission tomography (FDG-PET) has appeared as another modality with good accuracy in identifying spondylodiscitis.

Differential diagnosis includes spondylodiscitis from other infections, osteoporotic fracture, degenerative changes, inflammatory spondyloarthropathies, and spinal tumors, especially metastatic neoplasms.

## Laboratory Findings

Routine laboratory findings such as elevated erythrocyte sedimentation rate (ESR) or C-reactive protein level (CRP) or hyperleukocytosis are not specific, but ESR is more valuable in monitoring the therapy effect. Blood cultures are rarely positive. Tuberculin skin tests are extremely variable, and false negativity should be interpreted with prudence in immunocompromised populations. Cultures from other potential sources of infection (sputum, gastric fluid, and urine) may be helpful in some cases.

To prevent possible neurologic deterioration, lumbar puncture (spinal tap) is generally not recommended. Concomitant meningitis is rare.

Confirmation of the diagnosis can be made either by demonstration of acid-fast bacilli on pathological specimen or histological evidence of epithelioid–giant cell granulomas with caseating necrosis on the biopsy material. Neuroimaging-guided biopsy should be considered if other explorations fail to verify active extraneural tuberculosis. Nevertheless, it is difficult to find *Mycobacterium tuberculosis* with conventional microbiological methods like Ziehl–Neelsen staining, and it is likely to take 6–8 weeks for the growth to appear on cultures using Lowenstein–Jensen medium.

DNA amplification techniques such as polymerase chain reaction (PCR) and the QuantiFERON®-TB Gold In-Tube assay have shown very promising results for the early and rapid diagnosis of the disease. The possibility of mixed bacteria or fungi should always be considered.

## Treatment Options

The goal of the treatment of tuberculous spondylodiscitis is to eliminate infection and preserve spinal stability and neurologic function.

Patients require long-term antituberculosis chemotherapy. The first-line regimen is a combination of isoniazid, rifampicin, pyrazinamide, and ethambutol or streptomycin for 2 months, followed by two drugs (isoniazid and rifampicin) for 6–9 months. In some cases, drug-related toxicity may require the withdrawal or change of a drug. Moreover, the emergence of a new strain of drug resistance should be taken into account and managed with a second line of therapy. Regular follow-up is essential for a complete cure.

There is no definite role for corticosteroids in spinal tuberculosis, except in cases of spinal arachnoiditis, myelitis, or nonosseous spinal tuberculosis. External immobilization

(orthosis, brace, or collar) may be recommended when there is significant pain or the potential for spine instability.

There are several indications for surgery:

- Confirmation of diagnosis
- Decompression of neural structures
- Prevention or correction of spinal instability, severe kyphosis, or significant deformity
- Drainage of large concomitant abscesses
- Clinical deterioration or lack of clinical improvement

Some authors suggest that debridement of granulomatous and osteolytic lesions can help to accelerate healing.

The spinal approaches used may be anterior, posterior, posterolateral, or combined. Fusion and/or instrumentation (plates, screws, hooks, cages, and rods) may be used despite infection.

Physical rehabilitation, adequate nutrition, and treatment of medical comorbidities are essential to the successful treatment of Pott's disease.

## Outcomes

The prognosis of tuberculous spondylodiscitis depends on the patient's general health, the underlying neurologic conditions, the extent of the local lesion, the early establishment of the diagnosis, and the response to therapy. Prognosis is generally good in patients without neurologic deficit and deformity. Mortality due to Pott's disease is rare, often related to a missed diagnosis, coexisting comorbidities, or a complication of residual paraplegia or tetraplegia.

Patients with intradural lesions have a much poorer neurologic prognosis than patients with spondylodiscitis. The sequelae most likely to be associated with spinal tuberculosis include persistent weakness, lower extremity spasticity, sphincter dysfunction, and spinal deformities.

Prevention of tuberculosis is based on generalized and controlled immunization, along with improvement of health care and the living conditions of the population.

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# Neurocysticercosis

# 25

Neurocysticercosis is the most common parasitic disease of the central nervous system (CNS). It is caused by the larval form of *Taenia solium*. Within the CNS, the brain parenchyma is the most commonly involved site, followed by subarachnoid and intraventricular areas. Spinal cord localizations are rare. Signs and symptoms vary, depending on the location of cysticerci within the CNS, their number, and the host's immune response. The neuroimaging appearance of neurocysticercosis changes according to the viability of the larval cyst and the immune response. MRI is superior to CT scans for visualizing brain structures and cystic lesions, but CT scans are better for the detection of calcified lesions. Serologic tests may be helpful in making the diagnosis. Anthelmintic agents, including praziquantel and albendazole, are the mainstay of treatment for parenchymal neurocysticercosis, and steroids may help to decrease edema and prevent some complications. Surgery may be indicated for diagnosis, cystic resection, CSF shunting, or spinal decompression. The prognosis of neurocysticercosis is best for lesions in the brain parenchyma; subarachnoid and intraventricular cysts produce greater morbidity and mortality. Preventive programs may focus on avoiding parasite transmission.

## Epidemiology and Etiology

Cysticercosis, the most frequent parasitic infection of the CNS, is the leading cause of epileptic seizures in developing countries. This parasitic zoonosis is caused by the larval form of the pork tapeworm *Taenia solium.* Pigs are the intermediate host, and humans are the definitive host, though humans also may become an intermediate host by accidentally ingesting the taenia eggs (autoinfection or food/water contaminated with feces) (Fig. 25.1).

Neurocysticercosis is endemic in Latin America, the Indian subcontinent, Southern Asia, and sub-Saharan Africa, but it is rare in Islamic countries because of the proscription of eating pork. In recent years, the prevalence of the disease has increased in Europe and North America, owing to migration and travel.

Within the CNS, the brain parenchyma is the most commonly involved site (60%), followed by the subarachnoid space (30%) and the intraventricular area (15%) (Figs. 25.2, 25.3, 25.4, and 25.5). Localization in the spinal cord is rare (less than 5% of cases). Lesions in more than one compartment (mixed lesions) may be found in 25–50% of patients.

Classically, neurocysticercosis is classified into four stages: vesicular, colloidal vesicular, granular nodular, and calcified nodular (Table 25.1) Two types of cysts tend to develop in the brain: single cysts (*Cysticercus cellulosae*) or multiple cysts (*Cysticercus racemosus).*

## Clinical Presentations

Signs and symptoms of neurocysticercosis are produced by mass effect, inflammatory reaction, or obstruction of the flow of CSF. Thus, presenting symptoms are variable, depending on the location of lesions within the CNS, the number of cysticerci, and the host's immune response.

Intraparenchymal infection results in seizures, local neurologic deficits, raised intracranial pressure, and altered mental status. In the subarachnoid space, cysts may produce basal meningitis, cranial nerve palsies, vasculitis, and stroke. In the ventricles, the cystic lesion (especially a free-floating intraventricular cyst) may induce intermittent obstructive hydrocephalus. Head movement can rapidly increase the intracranial pressure (Brun's syndrome). Spinal cord involvement may cause arachnoiditis, meningitis, myelitis, or cord compression, especially in the form of thoracic spastic paraparesis with bladder dysfunction. Symptoms may also be secondary to immunologic effects of the disease.

Also to be considered is the possible occurrence of lesions in other systemic sites, especially skeletal muscles, eyes, and subcutaneous tissue.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_25

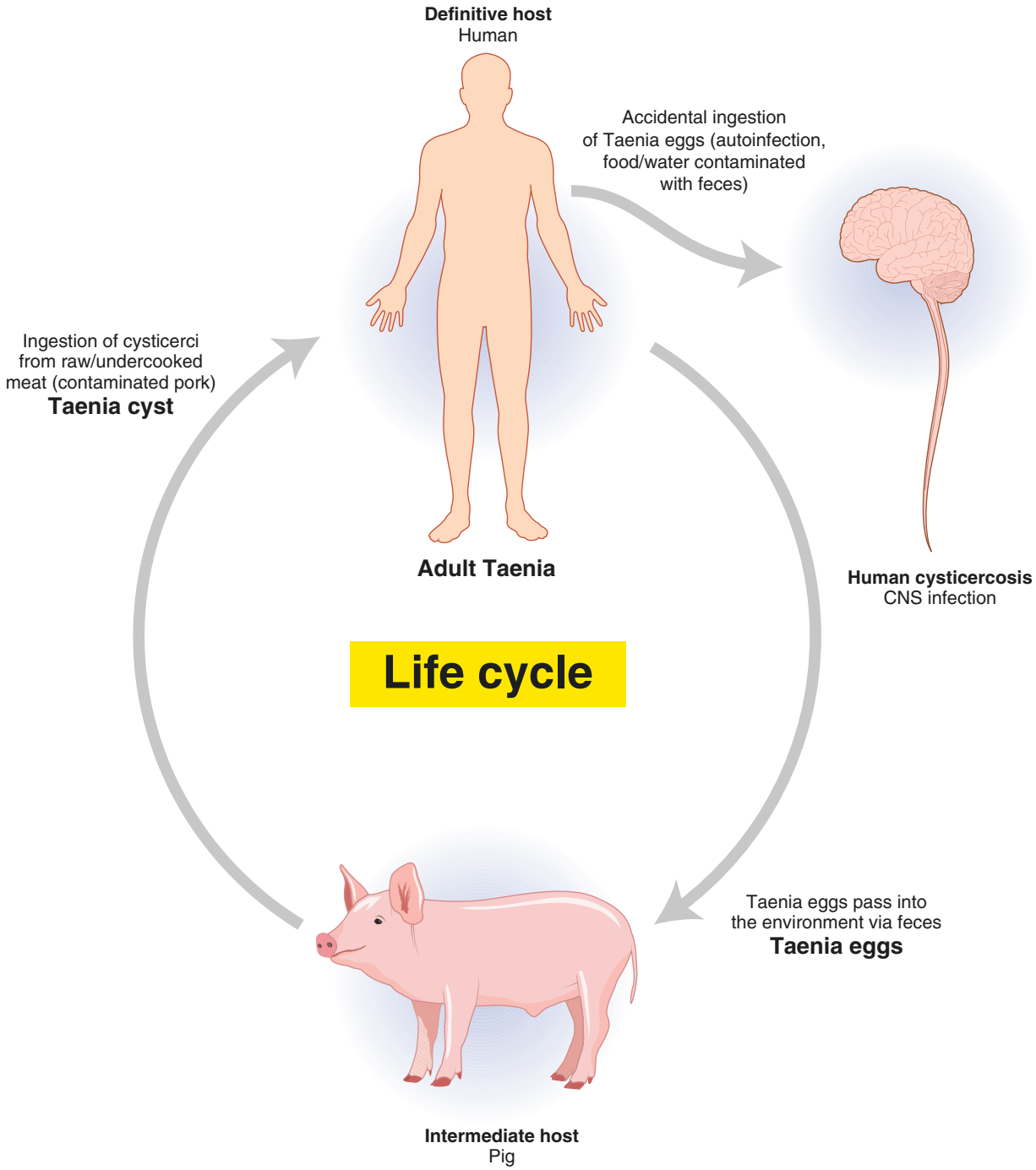


**Fig. 25.1** Life cycle of *Taenia solium*

## Imaging Features

The neuroimaging appearance of neurocysticercosis changes according to the viability of the larval cyst and the host's immune response. MRI is superior to CT scanning for visualizing brain structures, anatomy, and cystic lesions, but CT scans are the best screening procedure for the detection of calcified lesions (Figs. 25.6 and 25.7). Viable cysts are likely not to contrast-enhance without surrounding edema. Inflammation becomes visible as contrast enhancement and pericystic edema. Basal leptomeningeal enhancement also may be seen. When lesions are at the calcified stage, multiple, small, calcified nodules are found with little or no surrounding edema and without contrast enhancement.

At the vesicular and colloidal stage, an eccentric scolex may be seen within the cyst, giving the lesion a pathognomonic

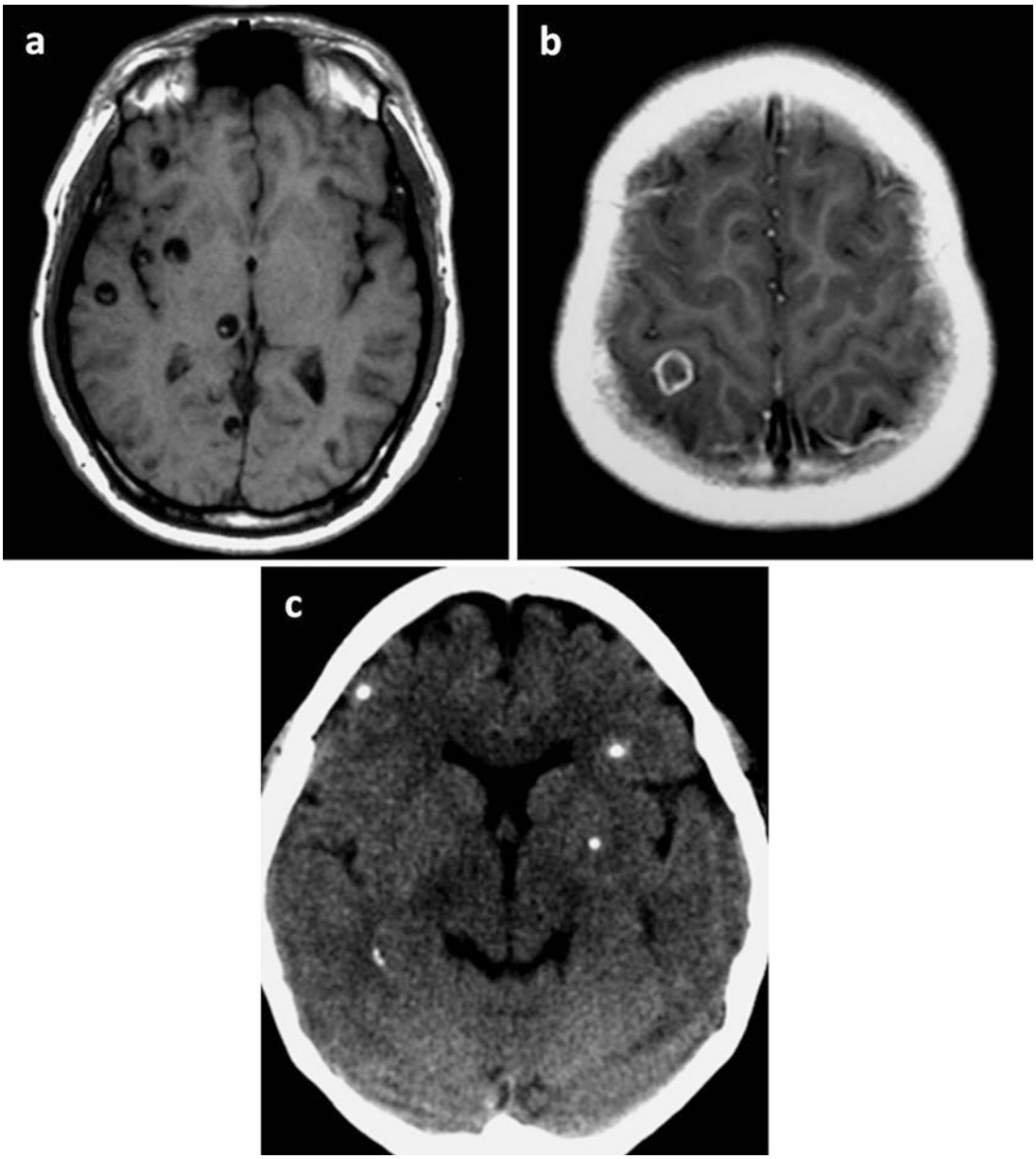


**Fig. 25.2** Imaging findings in patients with parenchymal brain cysticercosis: viable cysts showing the scolex (**a**), colloidal cyst appearing as a ring-enhancing lesion (**b**), and calcifications (**c**) (Reproduced from Del Brutto (2012))

"hole-with-dot" appearance on MRI. Spinal intradural cysticercosis is identified on MRI by findings similar to those of intracranial subarachnoid and parenchymal cysts (Fig. 25.8).

Advanced MRI sequences (diffusion-weighted imaging, diffusion-tensor imaging, susceptibility-weighted imaging, constructive interference in steady state, MR spectroscopy, and perfusion MRI) aid in better visualization of the cysts.

## Laboratory Findings

Mild peripheral eosinophilia can occur. CSF may show a lymphocytic pleocytosis, eosinophilia, and hypoglycorrhachia in cases with meningeal involvement.

Several laboratory methods were developed to detect host antibodies against circulating cysticercal antigens, but the most

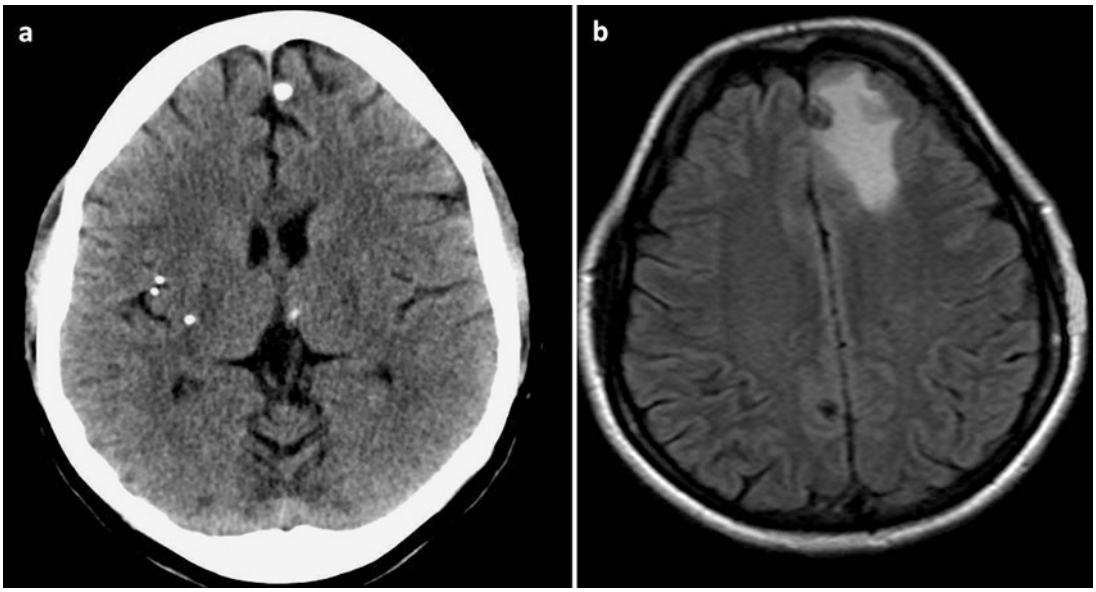


**Fig. 25.3** (**a**) Baseline CT scan demonstrating a dense calcification in the left frontal lobe, as well as other calcifications. (**b**) Fluid-attenuated inversion recovery (FLAIR) MRI sequence following a seizure, revealing perilesional edema (Reproduced from Coyle and Tanowitz (2009))

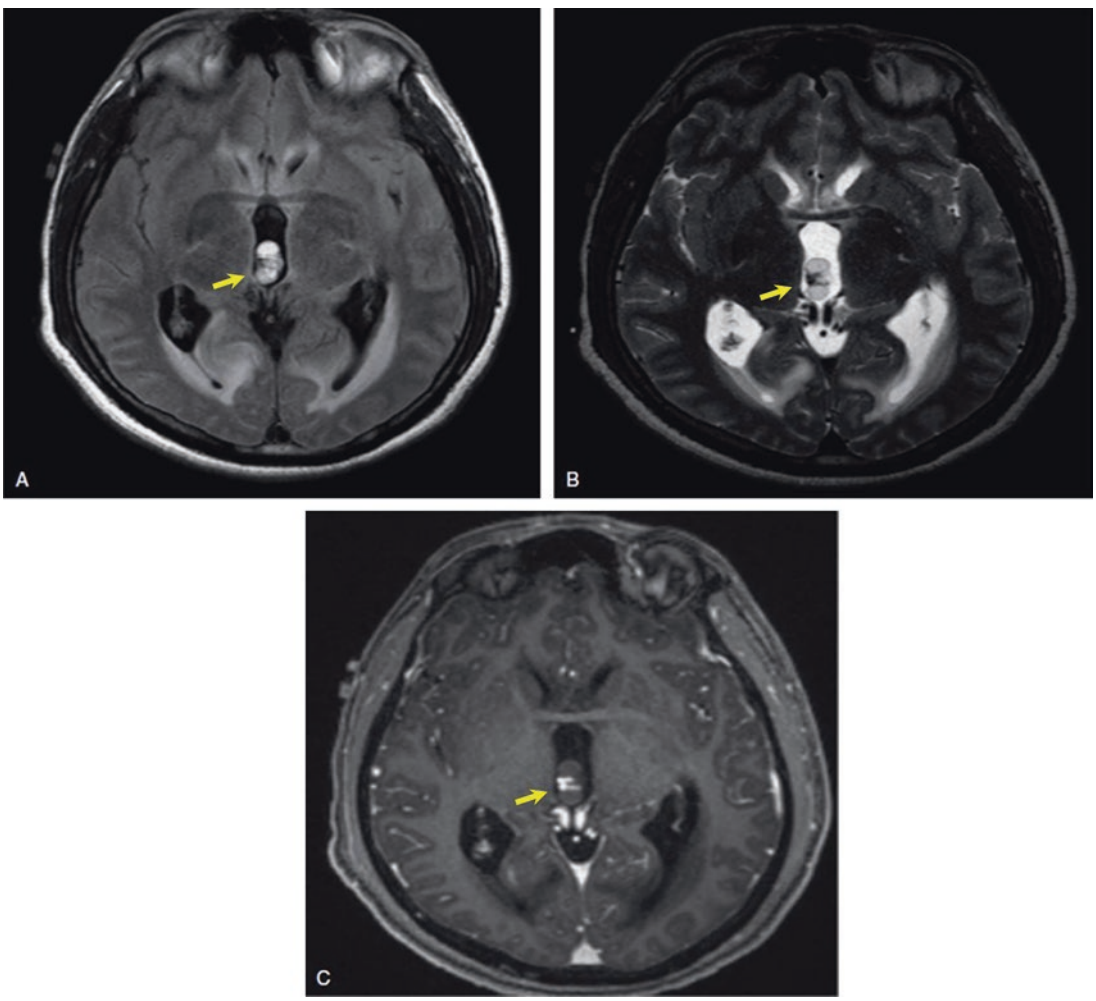


**Fig. 25.4** Intraventricular neurocysticercosis. Axial FLAIR (**a**), T2-weighted (**b**), and post-contrast T1-weighted (**c**) MR images show an intraventricular cyst causing hydrocephalus and ependymitis resulting in periventricular signal changes. The cyst (*arrow*) has different signal than CSF and shows enhancement, making it easy to see (Reproduced with permission from Aygun et al. (2013))

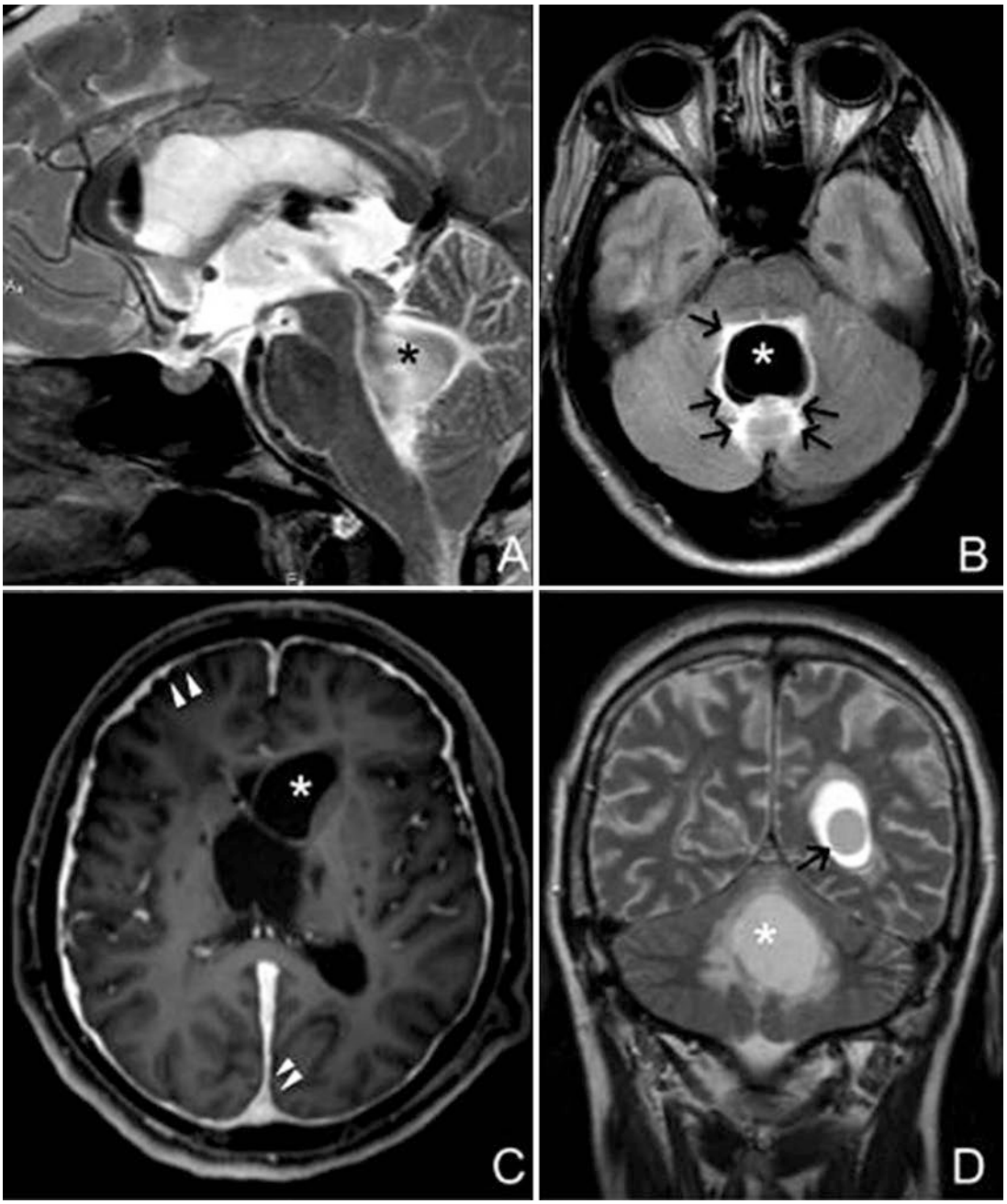


**Fig. 25.5** MRI images of cysts inside brain ventricles. The fourth ventricle is the most common site for ventricular neurocysticercosis. A large cyst (*star*) in the fourth ventricle (**a**) resulted in perilesional edema (*arrows*) in the patient's posterior fossa (**b**). The lateral ventricles are also common sites of cyst location. Meningeal enhancement (*arrowheads*) occurred in a patient with a cyst (*star*) in the left lateral ventricle (**c**). In some patients, multiple ventricles can be compromised, as illustrated by the cysts in the left lateral ventricle (*arrow*) and fourth ventricle (*star*) of this patient (**d**) (Reproduced from Bazan et al. (2016))

**Table 25.1** The stage pathologies of cysticercosis

| Stage | Form | Pathology |
|---|---|---|
| **First** | Vesicular | Cyst with clear fluid and viable larva (eccentric scolex) |
| **Second** | Colloidal vesicular | Larvae begin to degenerate and fluid becomes turbid |
| | | Perilesional edema |
| **Third** | Granular nodular | Healing stage, in which the cyst retracts |
| | | Scolex begins to calcify |
| | | Perilesional edema and sometimes necrosis |
| **Fourth** | Calcified nodular | Contraction of granulomatous lesion, with fibrosis and calcifications |
| | | No surrounding edema |

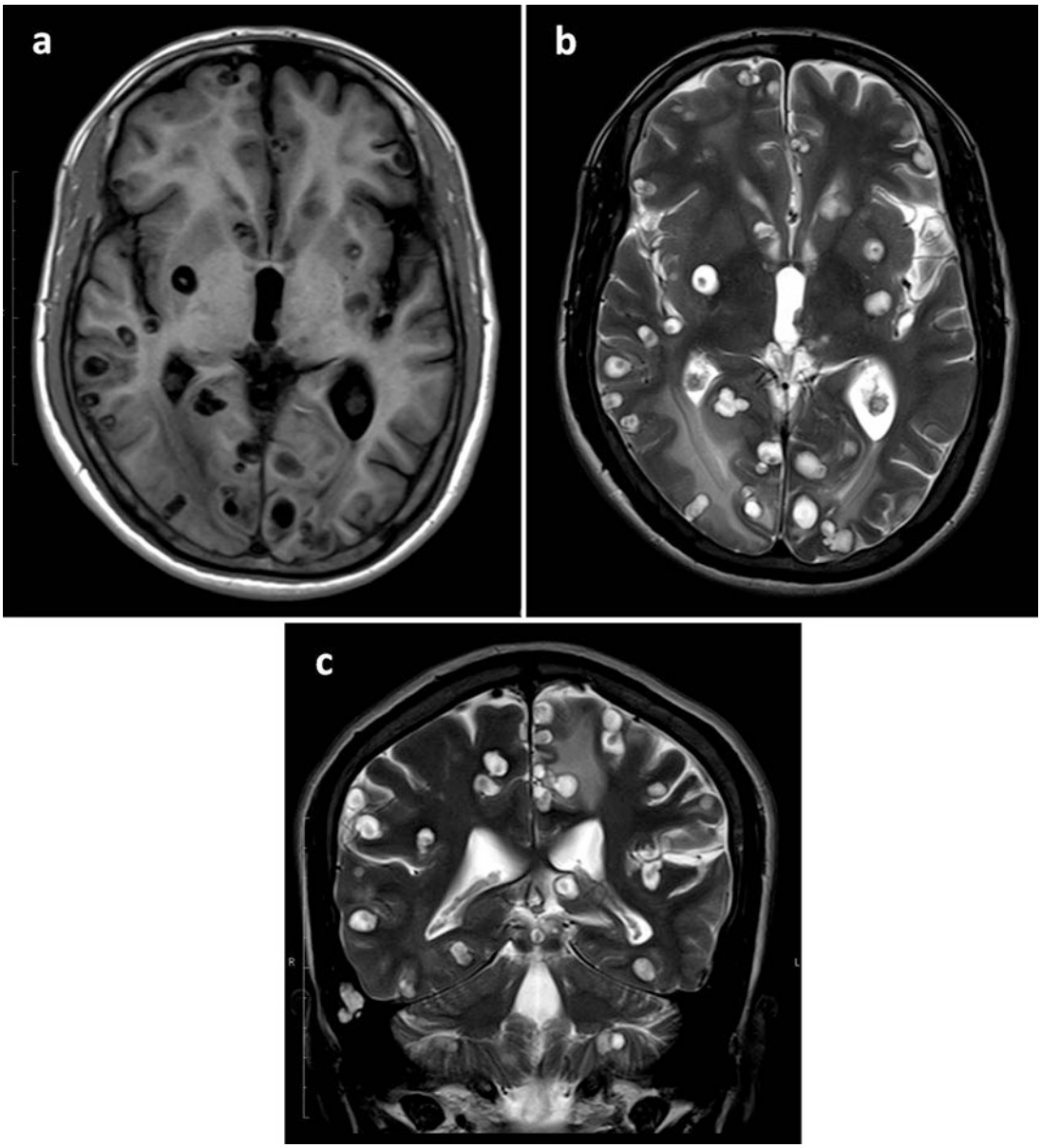


**Fig. 25.6** "Starry sky" appearance of multiple brain cysticercosis in a 42-year-old man. Axial nonenhanced T1-weighted (**a**) and T2-weighted (**b**) MRI. Coronal T2-weighted image (**c**). Note that a scolex may be seen within some cysts (Courtesy of Prasad Krishnan, MD; Kolkata, India)

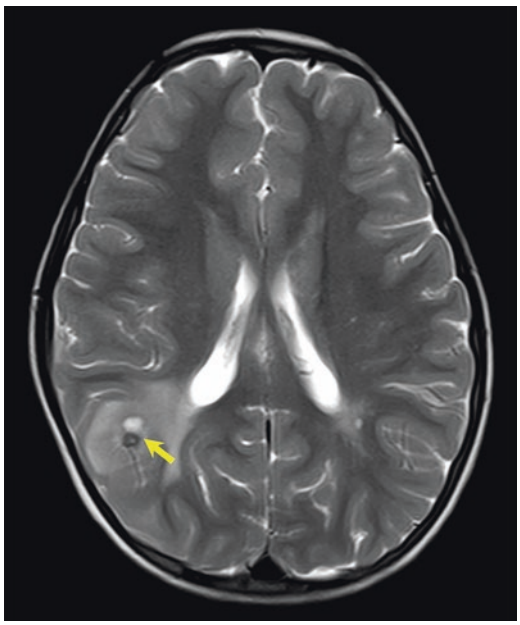

**Fig. 25.7** Cranial axial T2-weighted MRI in a 7-year-old girl showing a single cyst of *Taenia solium* located in the right brain parietal area (Courtesy of Prasad Krishnan, MD; Kolkata, India)

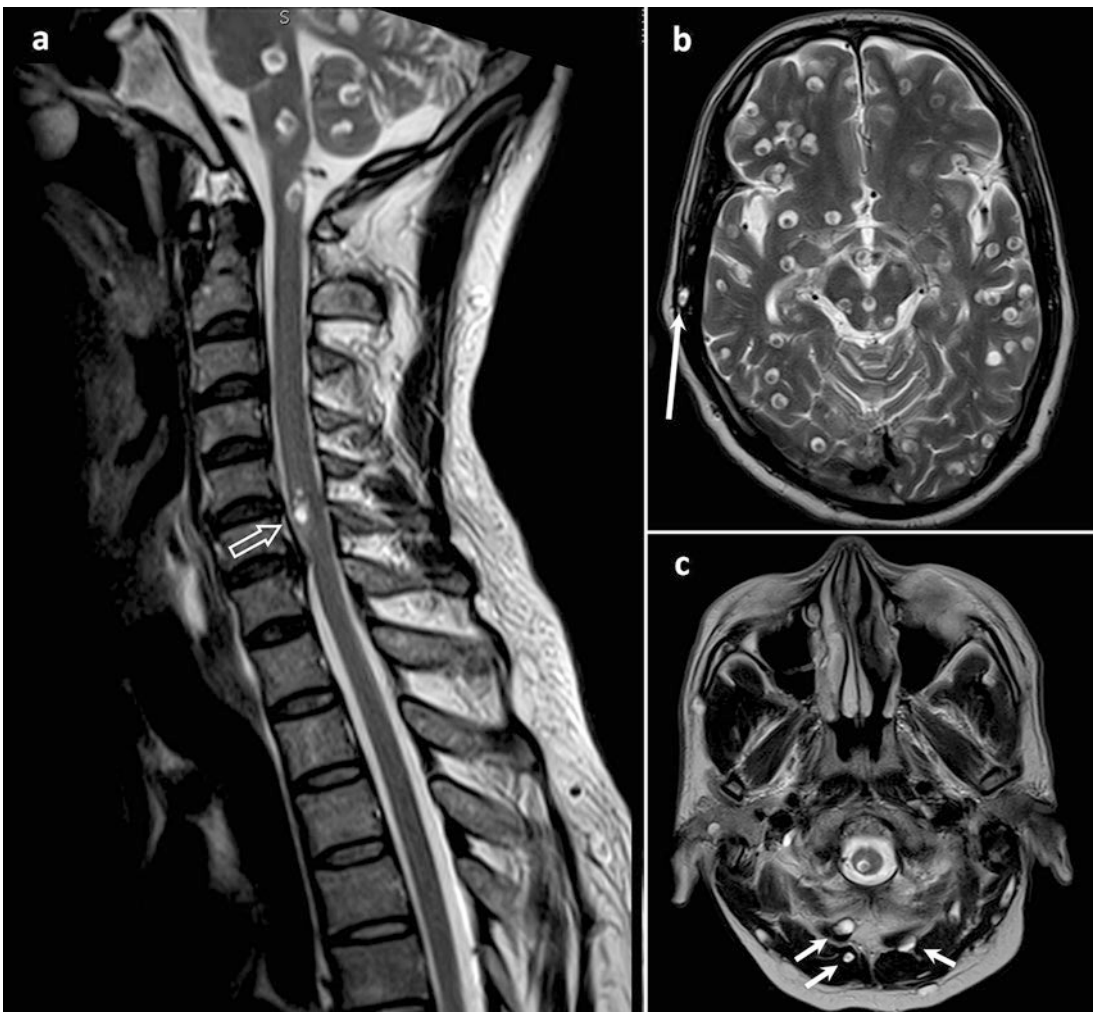


**Fig. 25.8** Extensive brain and spinal cord neurocysticercosis in a 50-year-old man, visible on sagittal (**a**) and axial (**b**, **c**) T2-weighted MR images. Note the cyst within the spinal cord (C6 vertebral level) (*outlined arrow*) and the concomitant muscular localizations (right temporal muscle and neck muscles) (*white arrows*) (Courtesy of Prasad Krishnan, MD; Kolkata, India)

effective in practice are enzyme-linked immunosorbent assay (ELISA) and electroimmunotransfer blot (EITB) assay. These tests are carried out on either blood (serum) or CSF.

Histologic demonstration of the parasites from brain stereotactic biopsy (when possible) can offer a definitive diagnosis in some confusing clinical situations. The cyst wall is distinguished microscopically by undulating, dense cuticle and focal globular formations suggesting the scolex structure.

## Treatment Options

The mainstay of treatment for parenchymal neurocysticercosis is anthelmintic therapy, including praziquantel and albendazole. Steroids may help decrease massive brain edema and prevent cerebral infarction, acute hydrocephalus, and spinal cord swelling.

Surgery occasionally may be indicated for brain biopsy (by a stereotactic procedure) or resection of intraparenchymal cystic mass lesions.

Patients with subarachnoid neurocysticercosis require months of treatment with both anthelmintic agents and steroids to avoid complications. Surgery (the resection under microscope of localized lesions) may be considered if medical treatment fails. CSF shunting is necessary for patients with symptomatic hydrocephalus, but there is a high prevalence of shunt dysfunction.

Intraventricular cysticercosis requires anthelmintic drugs, CSF shunting, and/or surgical cystic removal (particularly neuroendoscopic resection). In patients with spinal intradural lesions (subarachnoid or more rarely intramedullary), surgery should be indicated if spinal cord compression occurs.

Patients with preoperative seizures will need antiepileptic therapy for a long time.

## Outcomes

The prognosis of neurocysticercosis is best for intraparenchymal brain lesions, with a mortality rate lower than 10%. Extraparenchymal cysts, especially subarachnoid and intraventricular lesions, have a greater morbidity and mortality. Complications include chronic meningitis, hydrocephalus, dementia, vasculitis (inducing infarcts), and encephalitis. The most common sequelae are seizures, which require long-term anticonvulsive therapy.

Preventive programs may focus on avoiding parasite transmission and improving the management and vaccination of pigs.

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# Brain Hydatid Disease

# 26

Intracranial hydatidosis is a rare parasitic disease secondary to human intracranial development of the larval cysts of the tapeworm *Echinococcus granulosus*. The cyst, which is usually encephalic, spherical, solitary, subcortical, and supratentorial, is most often seen in children or young men. The most common presenting symptoms are those of intracranial hypertension, neurologic deficits, seizures, and mental changes. The cyst is well limited, with a density like CSF on CT scans and signal intensity like CSF on MRI. Surgical removal of the intact cyst is the cornerstone of treatment. The cyst is frequently delivered via a hydrostatic expulsion (Dowling's technique). Every effort should be made to deliver the intact cyst without rupture. Anthelmintic treatment may be considered to control the disease locally, avoid systemic spread, and prevent recurrence. Recovery from neurologic deficits occurs in most patients, and the mortality rate is less than 10%. Recurrence is common, nearly always owing to spillage of cystic contents at the time of removal of the original cyst or in patients with multiple cysts.

## Epidemiology and Etiology

Dogs and wild animals are the definitive host for the tapeworm *Echinococcus granulosus*, and the human acts as the intermediate host (Figs. 26.1, 26.2, and 26.3). Echinococcal infestation in humans occurs through the fecal–oral route by ingestion of food contaminated with canine feces containing the eggs of the organism.

The disease is endemic in those parts of the world where cattle and dogs are bred simultaneously (Mediterranean countries, the Middle East, Oceania, South Africa, and South America). In recent years, migration and travel have increased its prevalence in Europe and North America, where it is considered an emerging or reemerging disease.

Liver (60%) and lungs (30%) are the common sites of infestation. CNS involvement occurs in less than 3% of cases, with the brain affected in approximately 2% and the spine in less than 1% of patients infected with the parasite.

In the brain, the cysts are usually spherical, with a wall that is white, smooth, soft, and elastic. They tend to be slow growing (1–5 cm per year) and are often solitary, subcortical, and supratentorial. The ventricles, brainstem, posterior fossa, orbit, subdural and epidural areas, and skull are exceptional locations. Most patients are children or young men.

## Clinical Presentations

Symptoms, which depend on the involved area and the size of the cystic lesion, may vary from simple headache to uncal herniation. The most common presenting symptoms are headache, weakness of extremities, seizures, mental changes, and increasing head size or cranial deformities, especially in children. Symptoms and signs of raised intracranial pressure are frequent. Papilledema can lead to optic atrophy with unilateral or bilateral blindness. Irritability, change in school or job performance, and psychotic syndromes are less common.

A history of abdominal or thoracic surgery for hydatidosis and recent neurologic signs are highly suggestive of a brain hydatid cyst.

## Imaging Features

CT scan features of brain hydatidosis are characteristic: a spherical, thin-walled, well-limited, unilocular, hypodense (similar to CSF) lesion without surrounding edema (Figs. 26.4, 26.5, 26.6, 26.7, 26.8, 26.9, 26.10, 26.11, 26.12, 26.13, 26.14, 26.15, 26.16, 26.17, 26.18, and 26.19). Typically, there is no enhancement after contrast injection. Ipsilateral ventricles can be compressed, with midline shift to the contralateral hemisphere. Occasionally, one large cystic



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_26

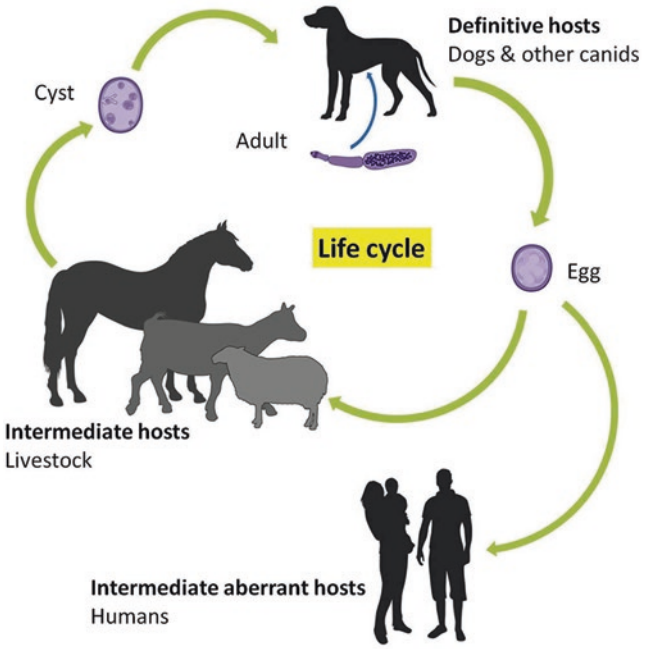


**Fig. 26.1** Schematic representation of the life cycle of *Echinococcus granulosus* (From Guisantes (2014); with permission of Springer)

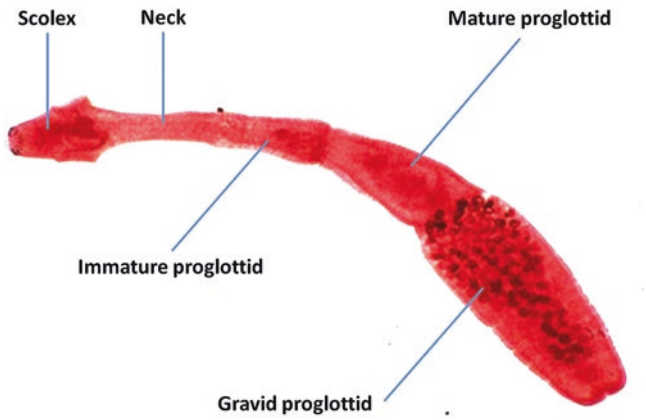


**Fig. 26.2** Adult *Taenia* of the tapeworm *Echinococcus granulosus*. The worm is about 4 mm in length and consists of a scolex (head) bearing four suckers, numerous hooks, and three proglottids. The gravid proglottid contains eggs (Courtesy of Pr. A. Dakkak, DVM, PhD; Rabat, Morocco)

lesion with internal septations suggestive of daughter cysts can be observed. Calcifications of the cyst wall are rare.

On MRI, the cyst is isointense to CSF on T1- and T2-weighted images and on fluid-attenuated inversion recovery (FLAIR) images, with a hypointense rim on T1- and T2-weighted images; perilesional edema is not present.

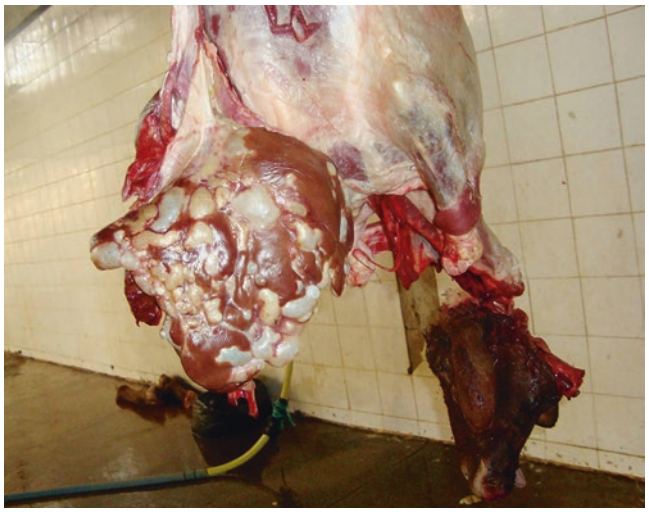

**Fig. 26.3** Multiple vesicles of *Echinococcus granulosus* infiltrating the liver parenchyma in a sheep (Courtesy of Dr. Y. Lhor, DVM, PhD; Rabat, Morocco)

Gadolinium-enhanced T1-weighted images classically show no enhancement, though an enhancing ring lesion is observed in the case of an infected cyst. Proton MR spectroscopy is used to characterize the metabolic content of the cysts.

Differential diagnoses to be considered include cystic astrocytoma, arachnoid cyst, epidermoid cyst, abscess, glio-ependymal cyst, and porencephalic cyst (Figs. 26.20 and 26.21).

## Laboratory Findings

Patients may have eosinophilic pleocytosis. Brain tissue evokes little or no immunologic reaction to hydatidosis, so serologic tests are of little value in diagnosing this condition. Complications such as rupture or infection are the most important factors influencing a positive serology.

On microscopic examination, the cyst contains germinating parasitic particles called *hydatid sand*.

## Treatment Options

Surgical removal of the intact cyst is the mainstay of treatment. Every effort should be made to prevent spillage of the cyst fluid and to remove the cyst completely.

Dowling's procedure is normally used; this consists of the spontaneous delivery of the intact encephalic cyst through a large craniotomy with a limited corticectomy. The cyst is

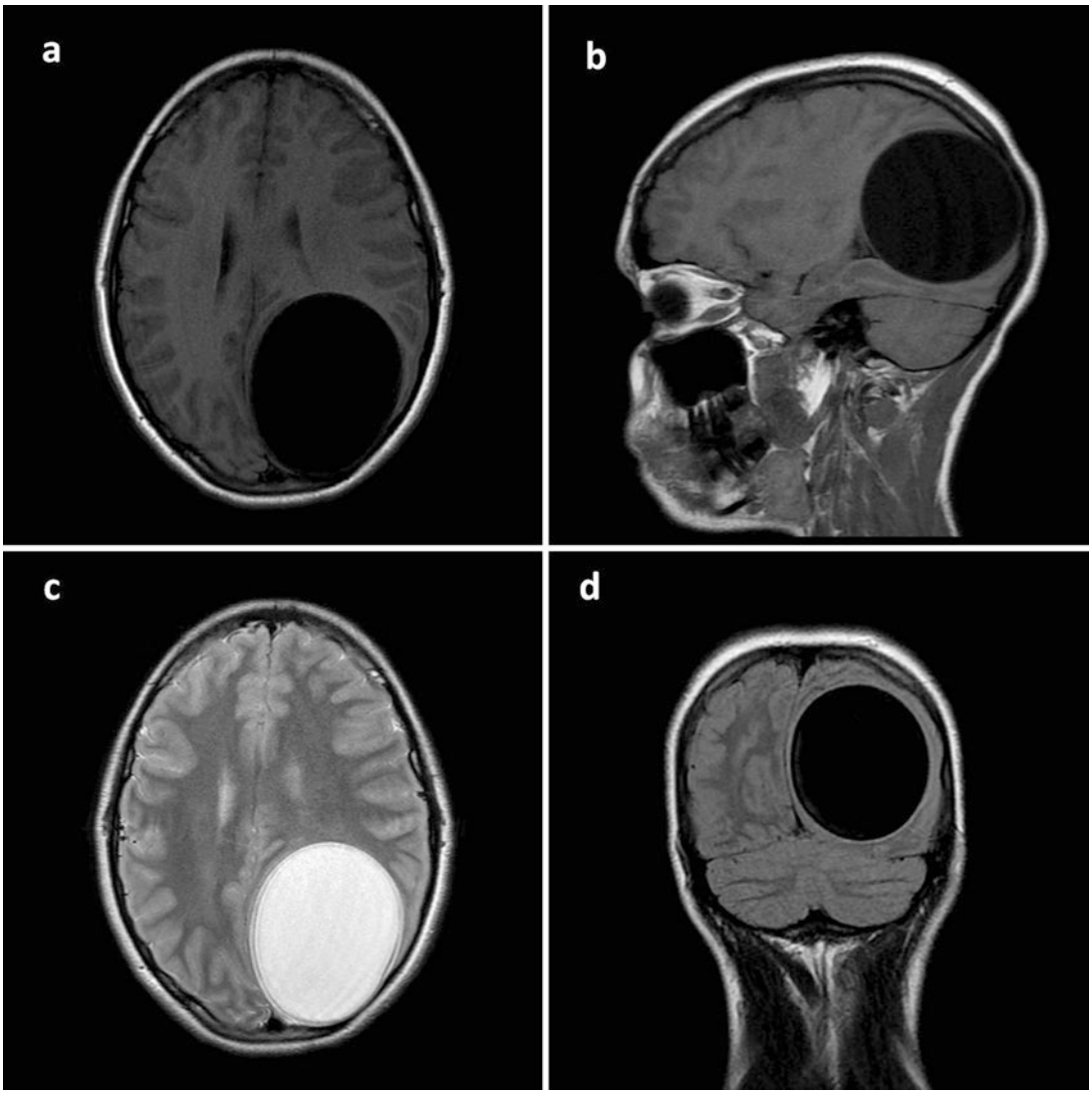


**Fig. 26.4** Case 26.1. Brain hydatid cyst. Large, well-defined unilocular cystic lesion in the left parieto-occipital region without marginal edema on axial **(a)** and sagittal (**b**) T1-weighted MRI, axial T2-weighted MRI **(c)**, and coronal fluid-attenuated inversion recovery (FLAIR) sequence (**d**)

delivered by forcing saline solution around the brain parenchyma (hydrostatic expulsion) (See also Figs. 26.7, 26.8, and 26.9). This surgical hydrodissection of the cyst/brain interface is performed by gentle irrigation with soft-tipped catheters. This technique is facilitated by the rarity of adherences between the cyst wall and the brain surface. Irrigation of the operative field with scolicidal solutions (hypertonic saline or oxygenic water) may be used to prevent local recurrence.

If accidental breakage of the cyst occurs, adjacent tissues will be contaminated, with possible future recurrence of multiple cysts. Allergic reaction (anaphylactic shock) is another complication observed when intraoperative spillage occurs.

Anthelmintic drugs (mainly albendazole and mebendazole) may be used in the perioperative period to prevent recurrences, but their efficacy in the CNS is not well established.

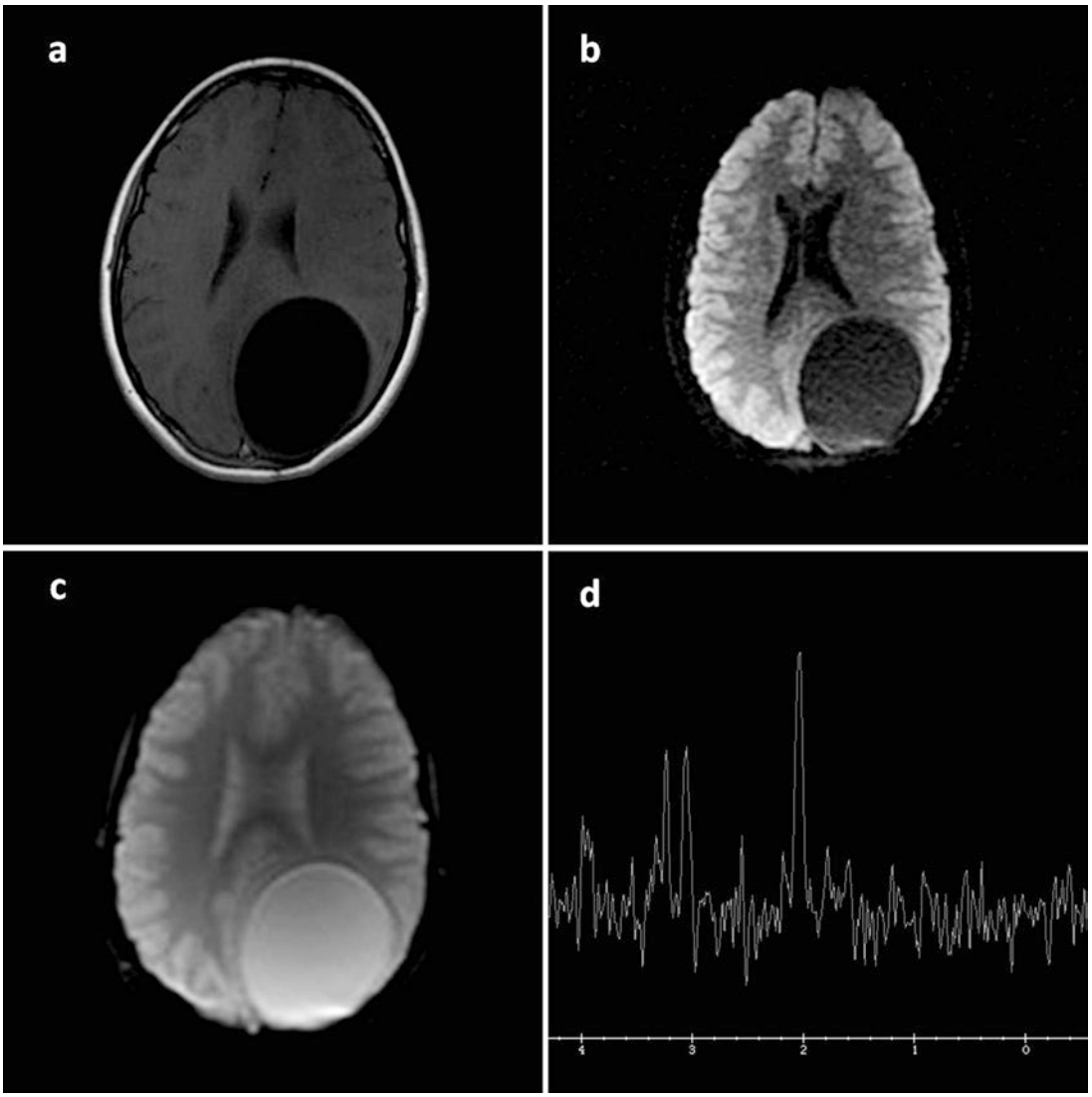


**Fig. 26.5** Case 26.1. Brain hydatid cyst on axial T1-weighted MRI (**a**), diffusion-weighted image (**b**), apparent diffusion coefficient map (**c**), and MR spectroscopy (**d**)

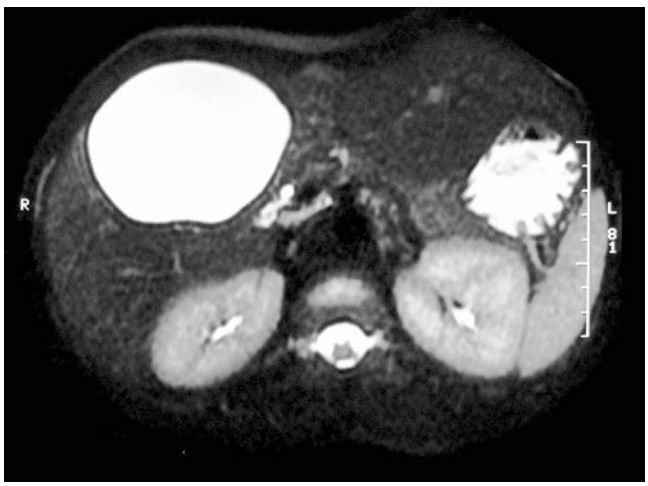


**Fig. 26.6** Case 26.1. This patient also has a solitary liver hydatid cyst. Appearance on axial abdominal T2-weighted MRI

Patients with preoperative seizures need long-term antiepileptic therapy.

## Outcomes

Most patients recover from neurologic deficits. Postoperative complications include subdural effusion, extradural hematoma, and infection, but the mortality rate is less than 10%.

Recurrence is a common complication that is nearly always due to spillage of cystic contents at the time of removal of the original cyst, or in patients with multiple cysts, so a long period of follow-up is needed.

Large preventive programs should aim to break the parasite life cycle and educate farmers in endemic areas.

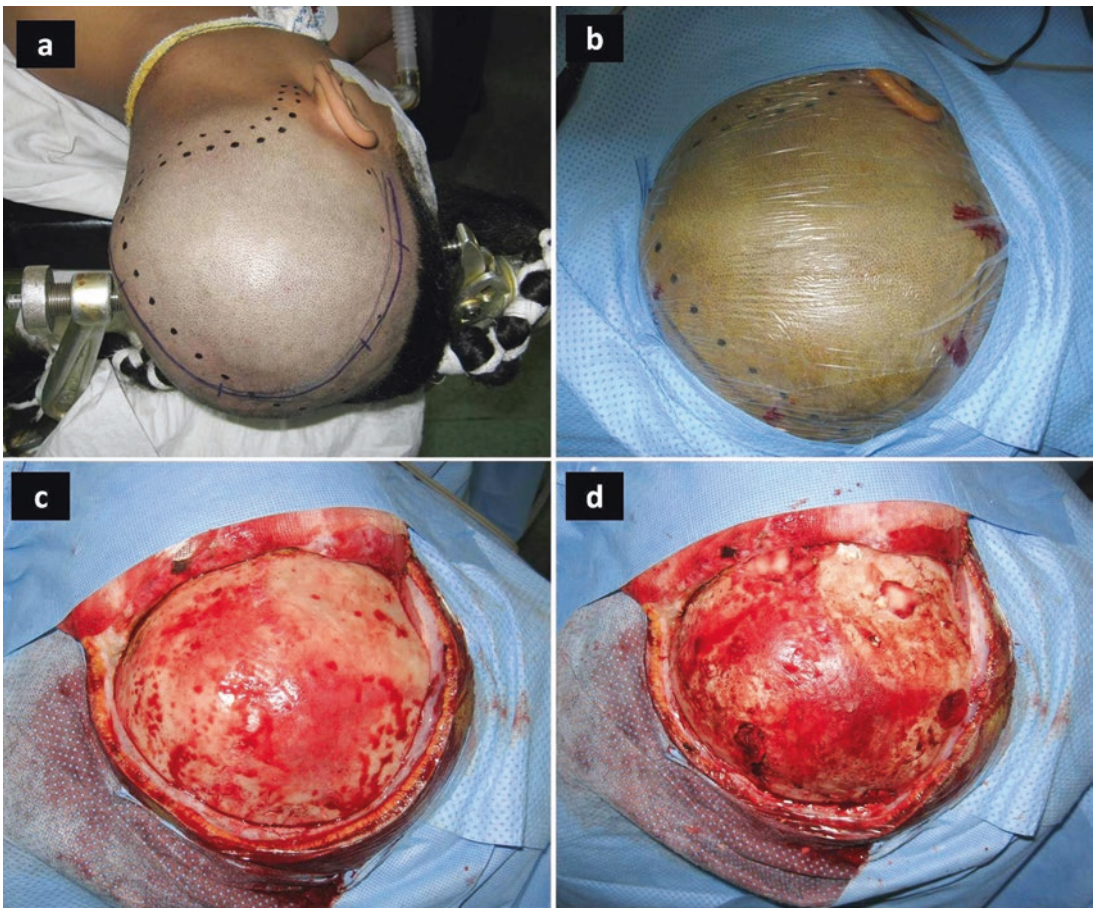


**Fig. 26.7** Case 26.1. Operative views. Patient's head positioning (**a**), the operating field sterilized and draped (**b**), a large skin flap reflected (**c**), and burr hole craniotomy performed for a large bone flap (**d**)

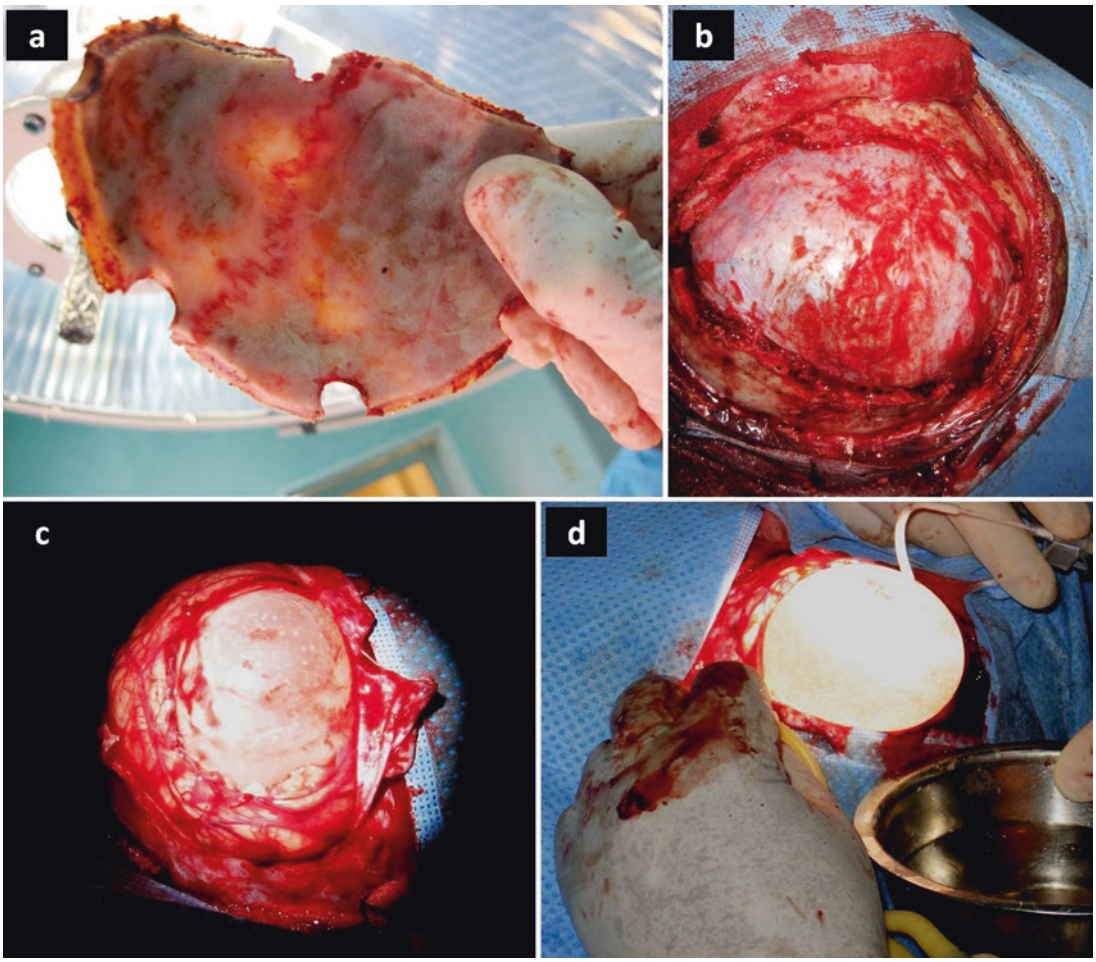


**Fig. 26.8** Case 26.1. Operative views. The large free bone flap removed (the bone is *thinned*) (**a**); the tense dura mater (**b**); the entire cystic wall exposed (**c**). Dowling's procedure: spontaneous delivery of the intact brain cyst by hydrostatic expulsion (**d**)

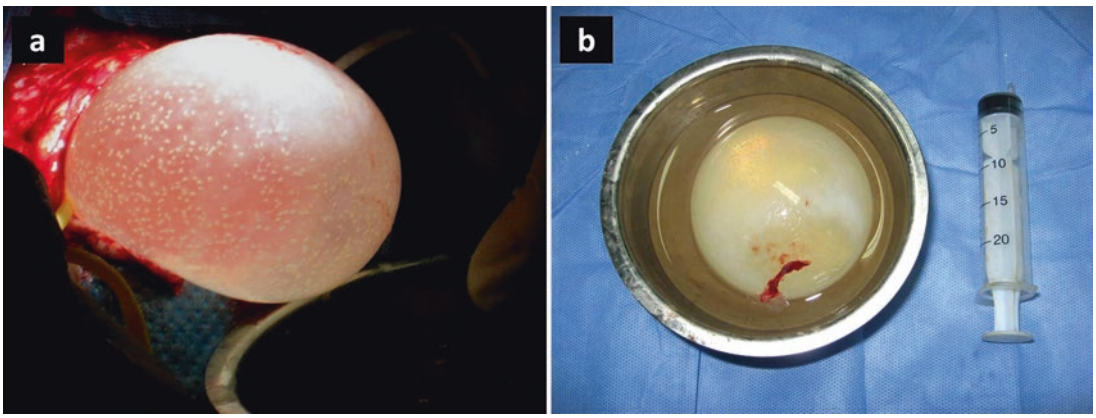


**Fig. 26.9** Case 26.1. Operative views. Progressive complete expulsion of the hydatid cyst (**a**). Intactly removed hydatid cyst in a bowl with water (**b**)

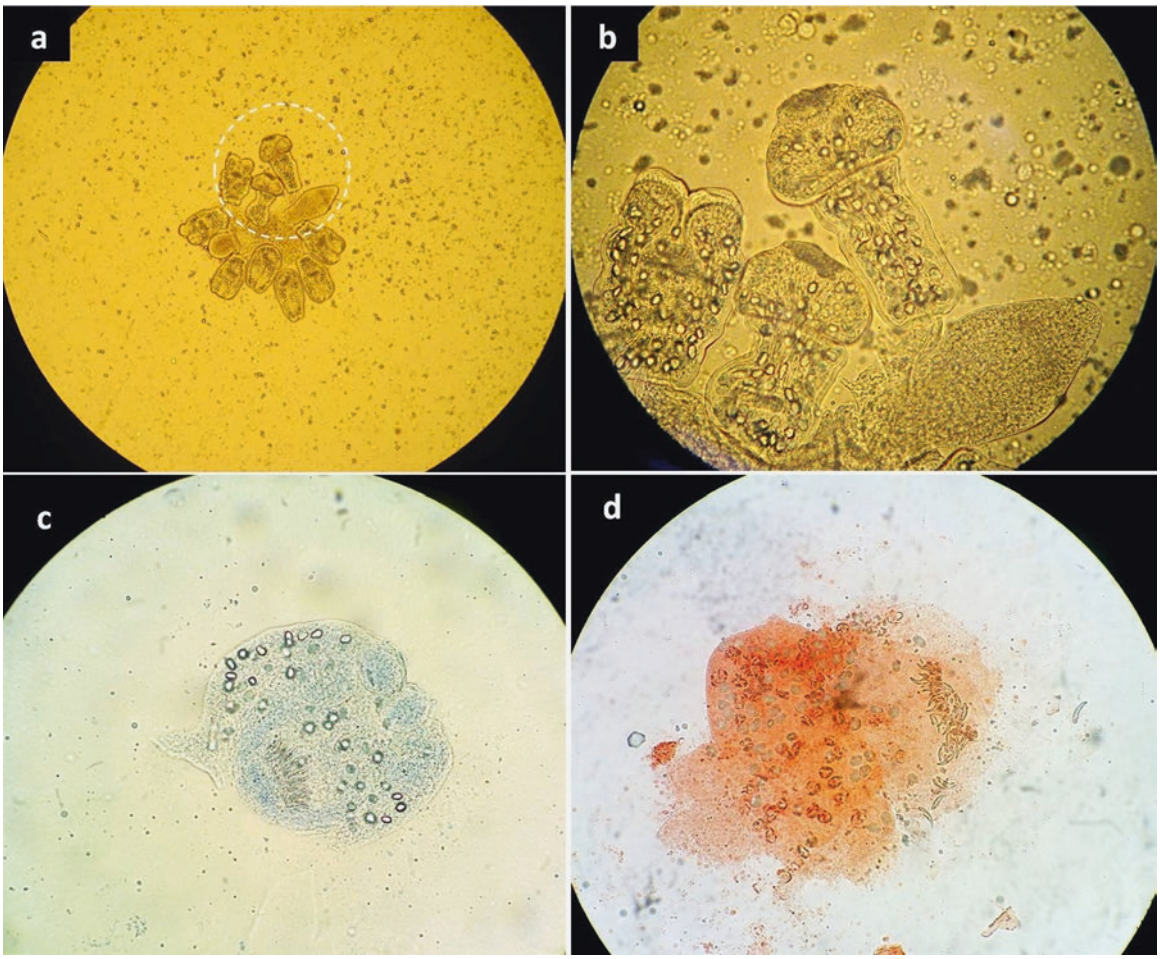


**Fig. 26.10** Case 26.1. Various protoscoleces in the brain hydatid cyst, shown at low magnification (**a**) and higher magnification (**b**). Protoscolex stained with methylene *blue* (**c**) and Congo *red* (**d**) (Courtesy of Pr. B. Lmimouni, PharmD; Rabat, Morocco)

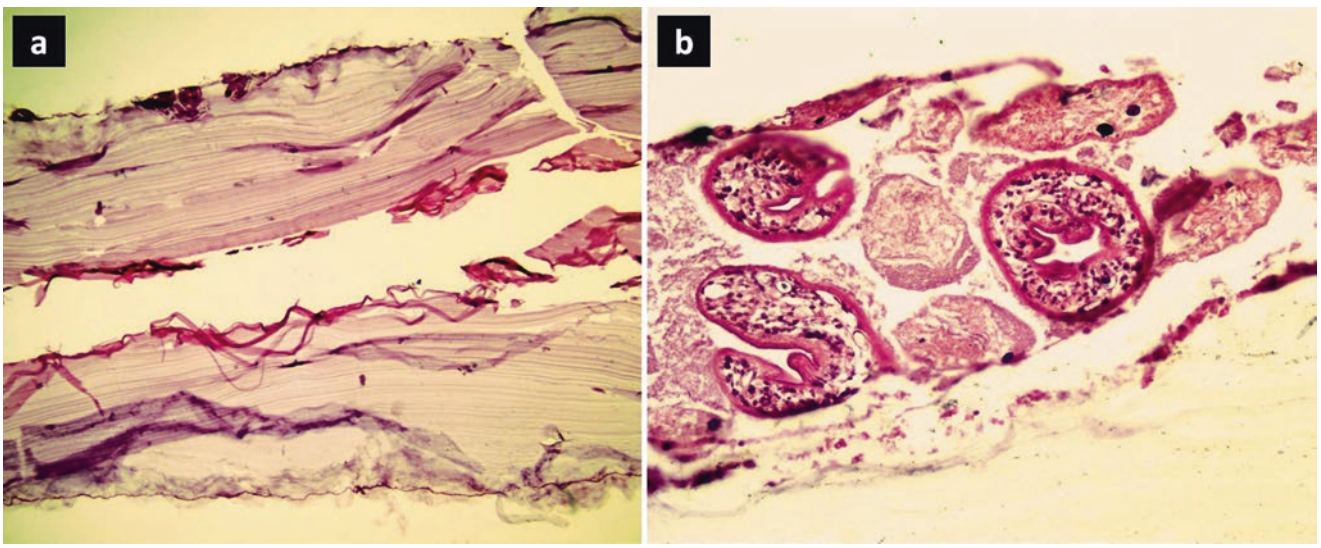


**Fig. 26.11** Histologic features of the cyst wall of *Echinococcus granulosus* (hematoxylin–eosin staining): cystic structures with laminated membranes and inner germinal layer (low-power magnification) (**a**). Photomicrograph showing various protoscoleces (medium-power magnification) (**b**)

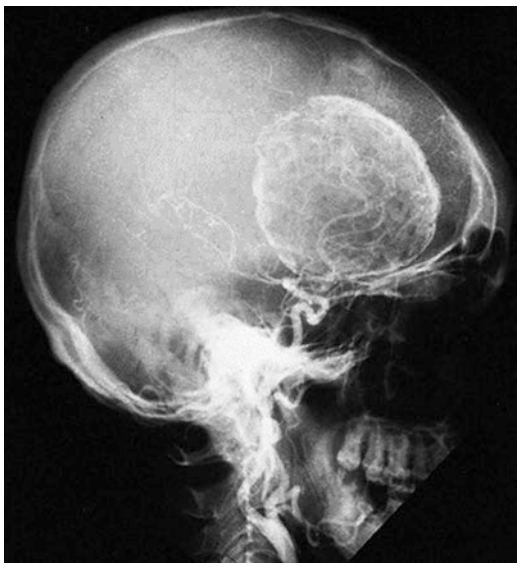

**Fig. 26.12** Lateral skull radiography showing a calcified frontal hydatid cyst (From Hossain (2014); with permission of Springer)

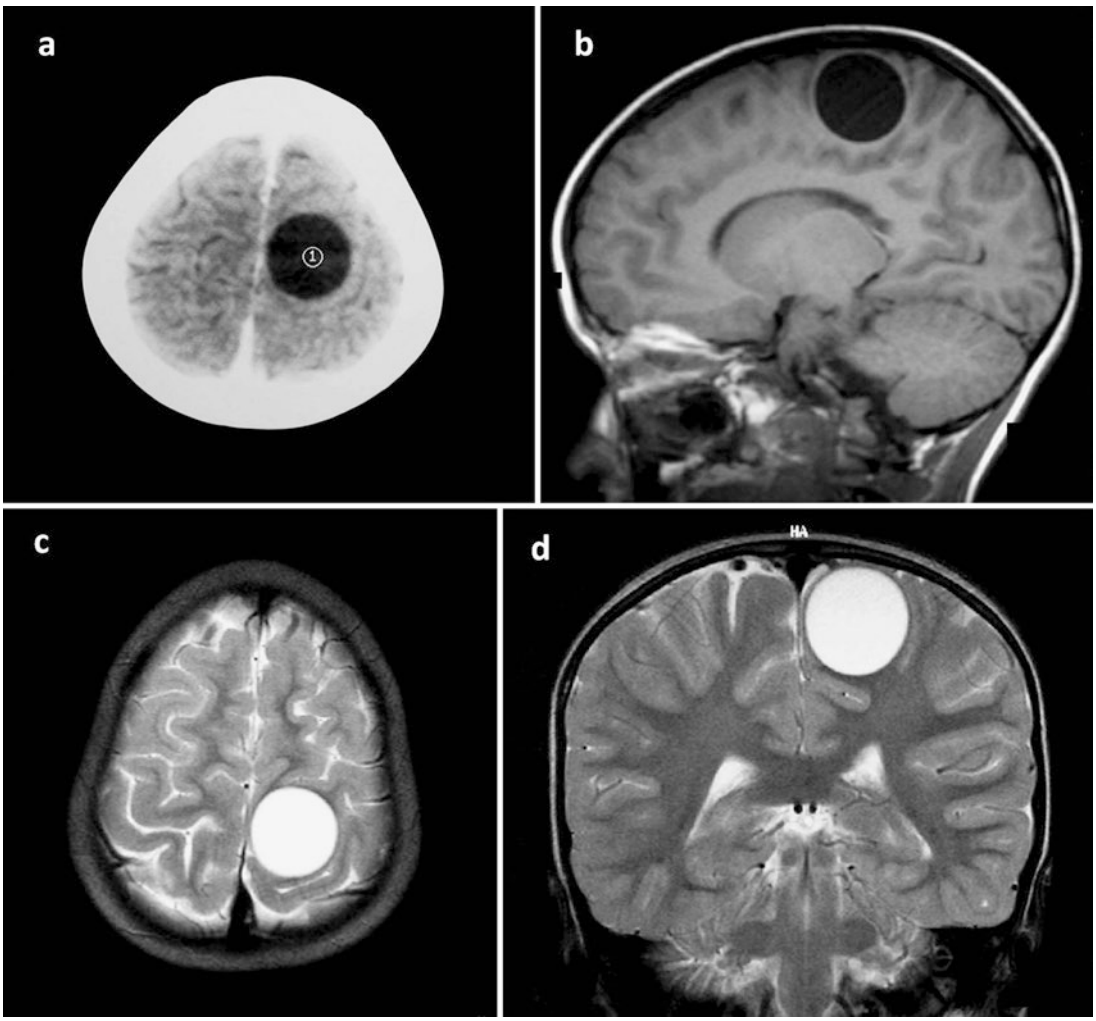


**Fig. 26.13** Brain hydatid cyst in a child. Axial CT scan (**a**), sagittal T1-weighted MRI (**b**), axial (**c**) and coronal T2-weighted MRI (**d**) showing a well-defined, solitary cystic lesion in the left fronto-rolandic area compressing the surrounding brain parenchyma without edema

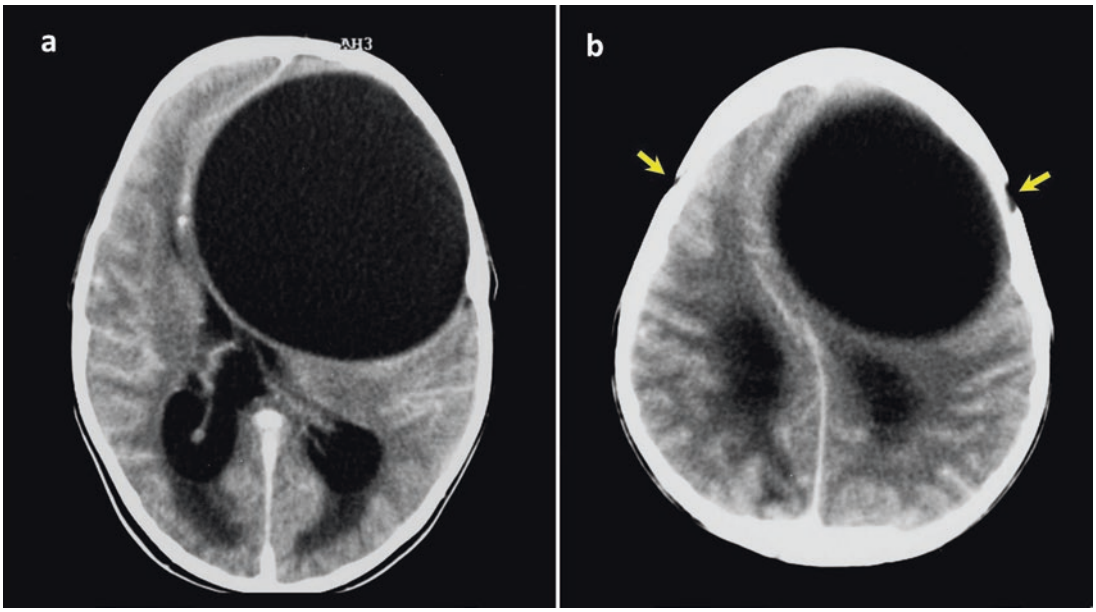


**Fig. 26.14** (**a**, **b**) Axial CT scan with contrast injection in a child, revealing a giant brain hydatid cyst compressing and causing an important shift of the midline structures. Note the disjunction of the coronal suture (*arrows*)

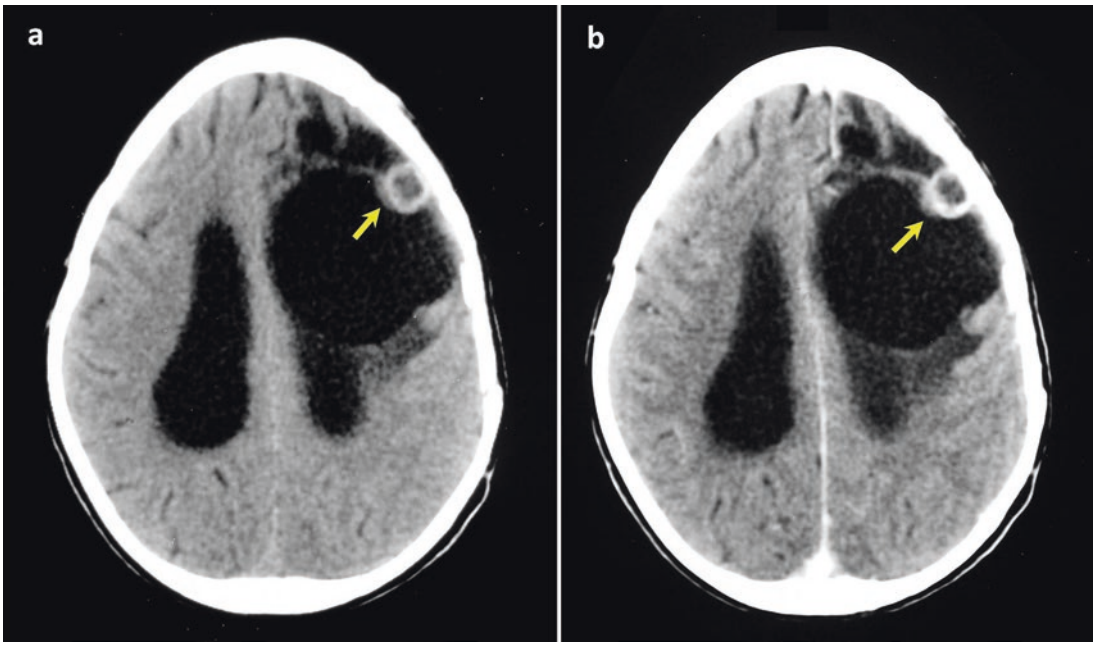


**Fig. 26.15** Axial CT scan before (**a**) and after (**b**) contrast injection 6 months after surgery for a left frontal hydatid cyst. There is a small residual cyst or postoperative recurrence (*arrows*)

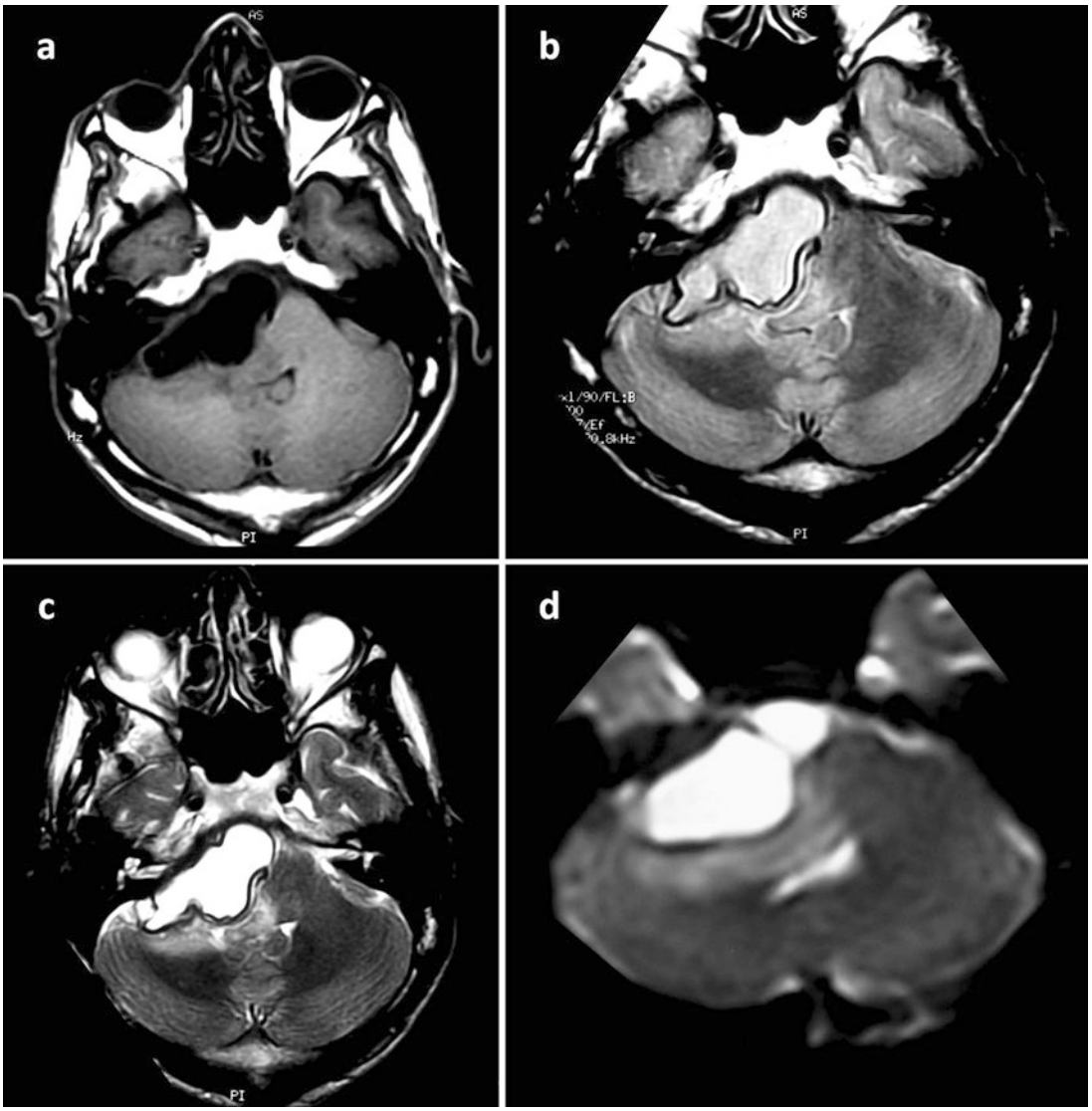


**Fig. 26.16** Case 26.2. Intracranial hydatid cyst of the right cerebellopontine angle with brainstem compression. Axial T1-weighted MRI (**a**), FLAIR sequence (**b**), T2-weighted MRI (**c**), and apparent diffusion coefficient map (**d**)

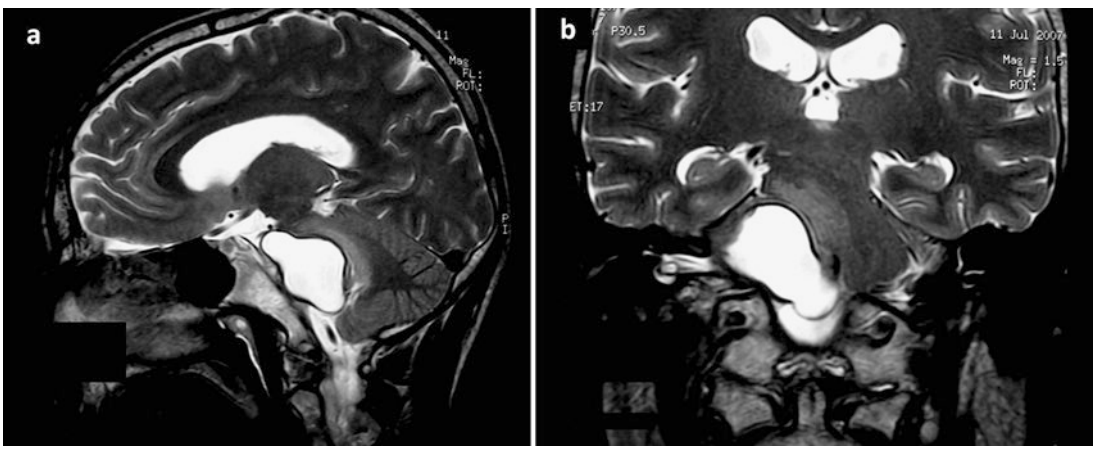


**Fig. 26.17** Case 26.2. Intracranial hydatid cyst of the right cerebellopontine angle compressing the brainstem. Sagittal (**a**) and coronal T2-weighted MRI (**b**)

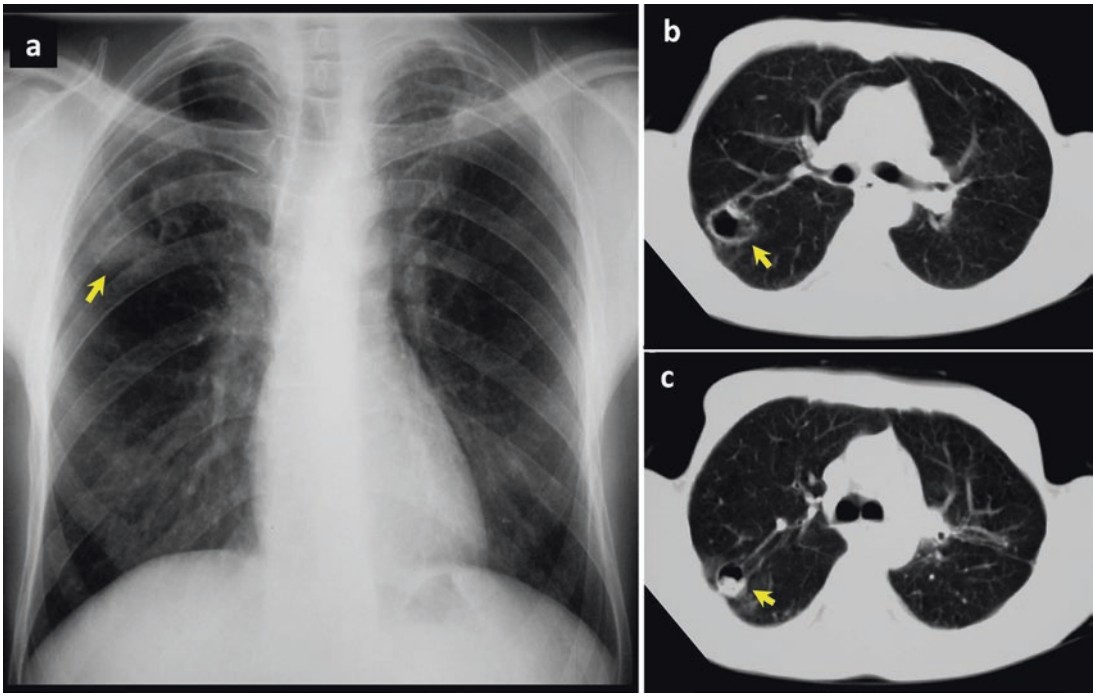


**Fig. 26.18** Case 26.2. This patient also has a solitary pulmonary hydatid cyst (*arrows*), seen on chest X-ray (**a**) and axial chest CT scan (**b**, **c**)

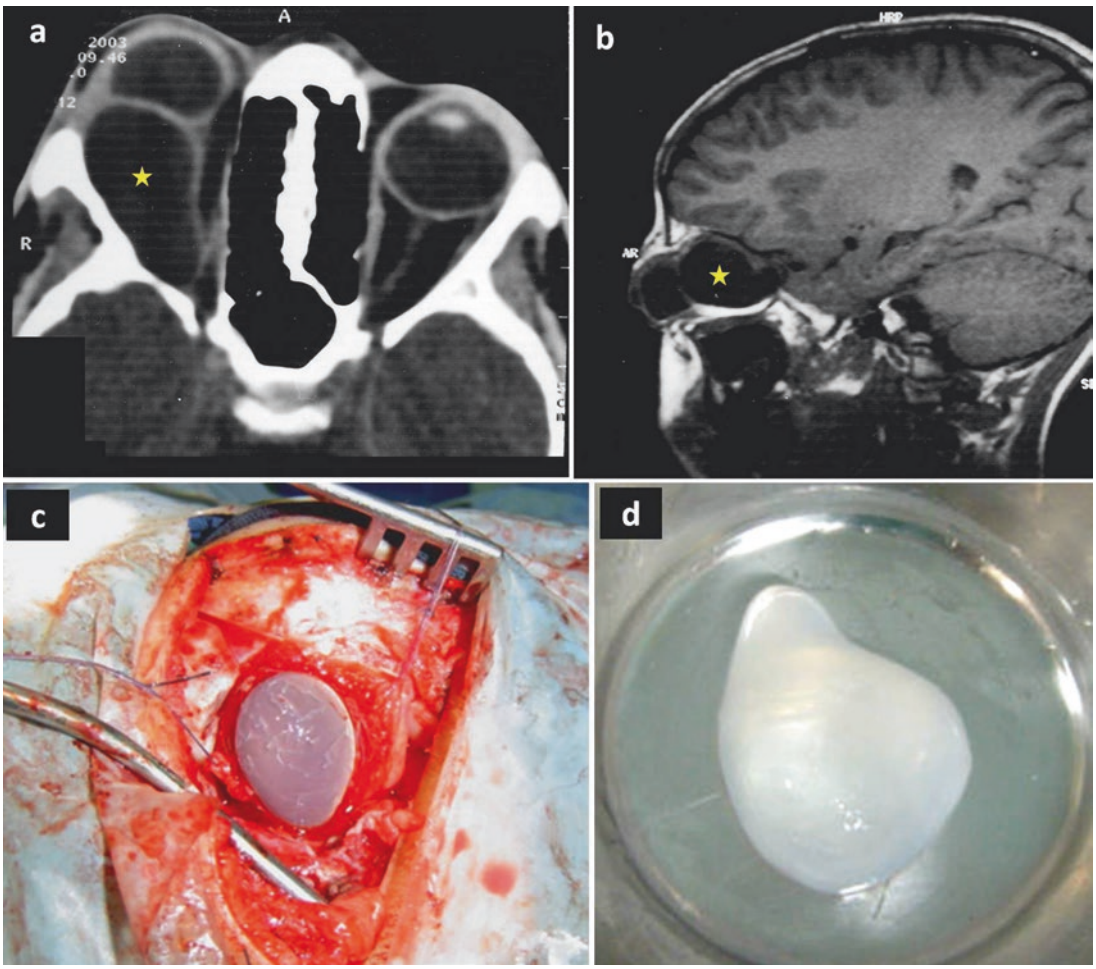


**Fig. 26.19** Hydatid cyst in the *right* orbital cavity. Axial CT scan (**a**) and sagittal T1-weighted MRI (**b**) showing a large cyst-like retrobulbar mass (*star*). Operative view of the parasitic cyst removed through a lateral orbitotomy (Kronlein approach) (**c**). The orbital hydatid cyst was extracted without rupture (**d**) (Courtesy of A. Taous, MD; Meknes, Morocco)

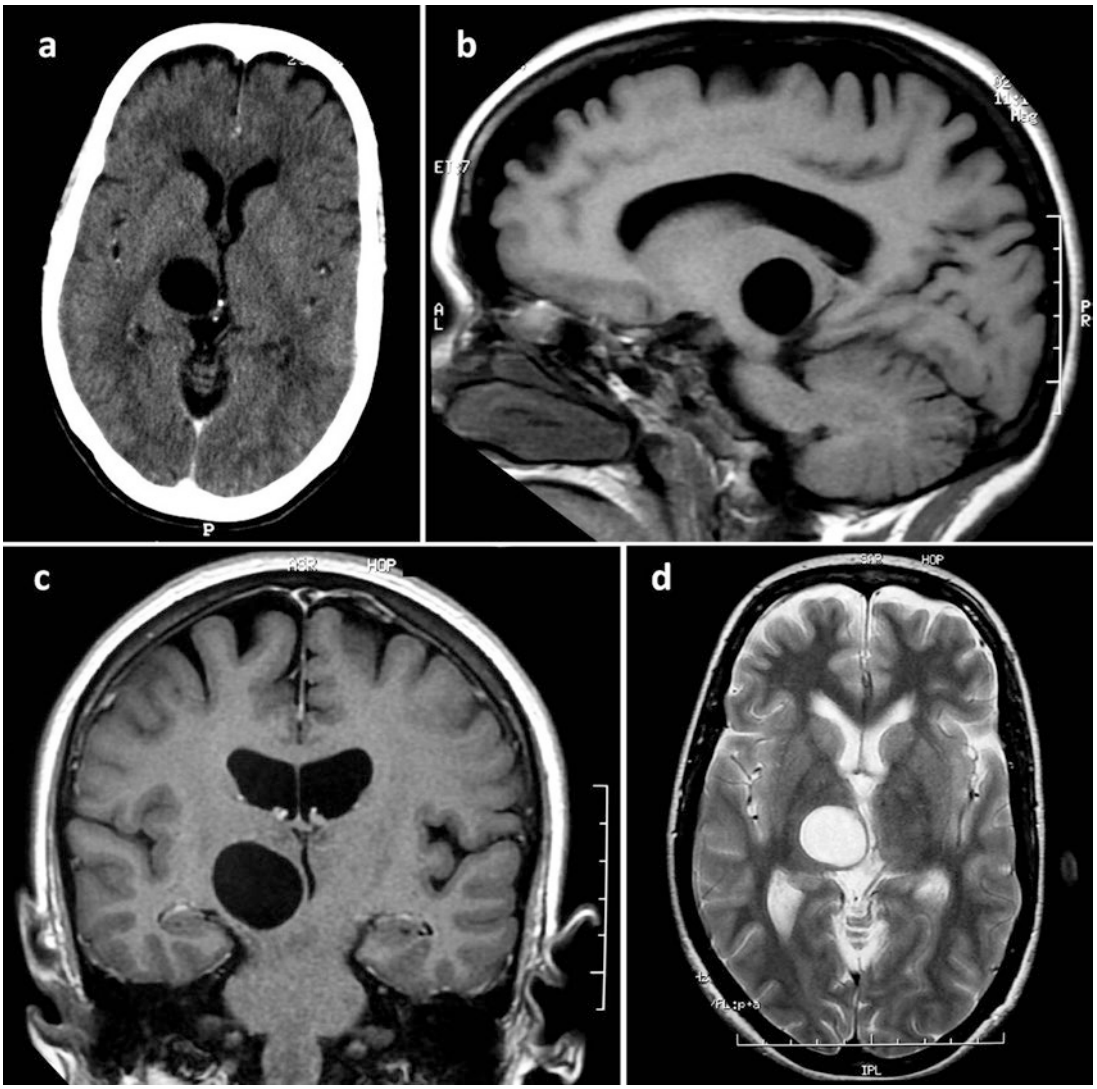


**Fig. 26.20** Thalamo-mesencephalic neuroepithelial cyst mimicking a hydatid cyst. Axial CT scan with contrast injection (**a**); sagittal (**b**) and coronal T1-weighted MRI (**c**); and axial T2-weighted MRI (**d**)

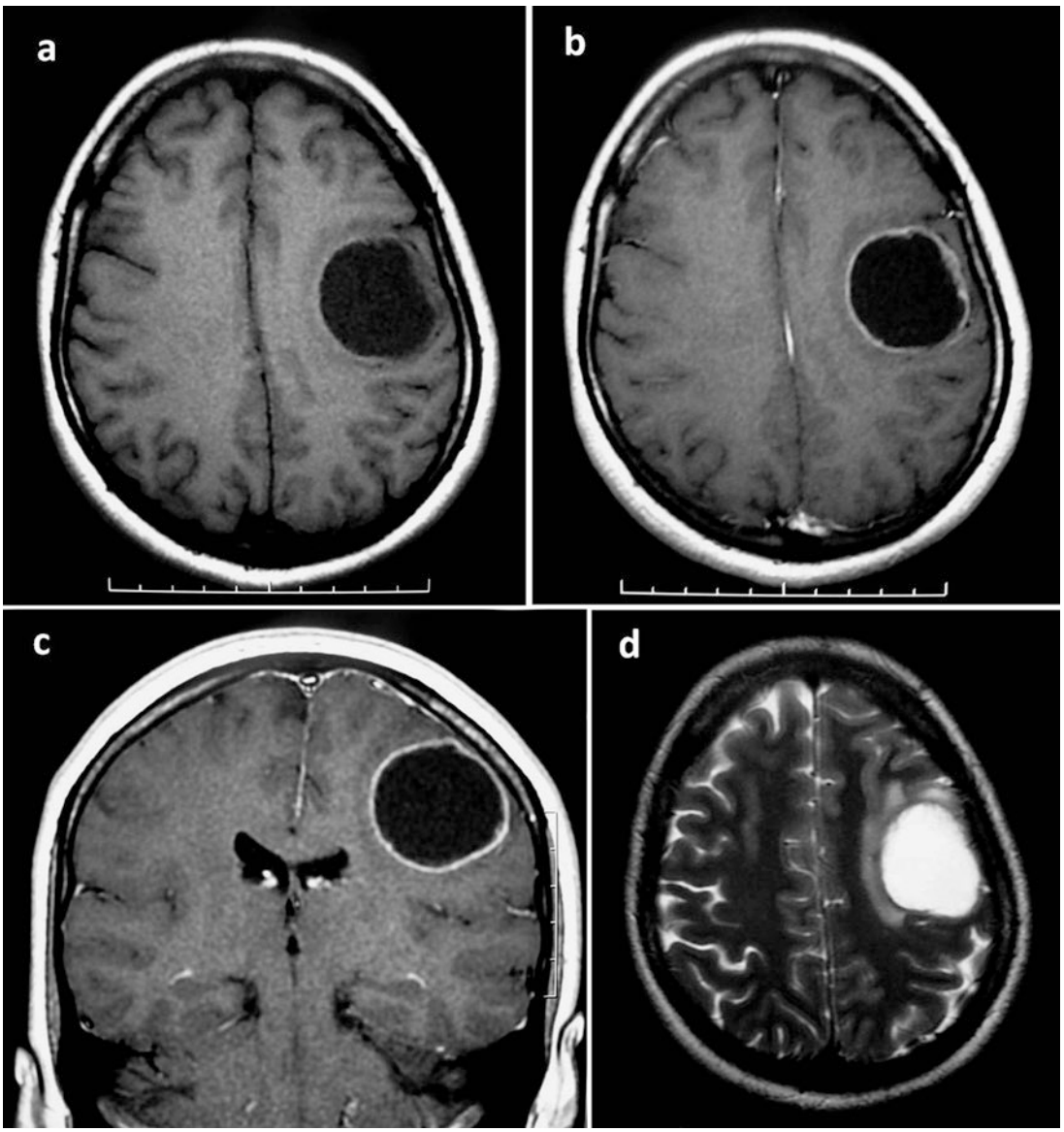


**Fig. 26.21** Prerolandic benign cystic astrocytoma in the *left* frontal area, mimicking a hydatid cyst. Axial T1-weighted MRI before (**a**) and after (**b**) gadolinium injection, coronal T1-weighted MRI after gadolinium injection (**c**), and axial T2-weighted MRI (**d**). Note the enhancing ring lesion after gadolinium administration and the mild perifocal edema

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# Spinal Hydatid Disease

# 27

Spinal hydatidosis is a rare parasitic disease secondary to human vertebral column development of the larval cysts of the tapeworm *Echinococcus granulosus*. The cyst is usually microvesicular, multiple, and invasive. Common presenting symptoms are chronic back pain, radiculopathy, lower limb weakness, sphincter disturbances, and spinal deformity. On CT scan and MRI, the cysts (with CSF-like content) are multiloculated, with irregular branching and bony erosions. The treatment objectives for spinal hydatidosis are to remove the lesions, relieve pain, avoid neurologic deficit, and preserve spinal stability, but unlike most cases of brain hydatidosis, it is almost impossible to resect the cysts en bloc without rupture. Spinal hydatidosis is highly likely to recur, leading to progressive destruction of the vertebral column and neurologic deterioration. Thus, spinal hydatidosis has a poor prognosis and is often compared to a local spinal malignancy.

## Epidemiology and Etiology

As discussed in Chap. 26, hydatidosis is caused by the larval cysts of the tapeworm *Echinococcus granulosus*, with infestation in humans occurring through the fecal–oral route by ingestion of food contaminated with canine feces containing the eggs of the organism (*see* Fig. 26.1). The disease is endemic in Mediterranean countries, the Middle East, Oceania, South Africa, and South America, but in recent years, migration and travel have increased its prevalence in Europe and North America, where it is considered an emerging or reemerging disease.

The liver (60%) and lungs (30%) are the most common sites of infestation; only 0.5–2% of cases involve the skeleton. The vertebral column is affected in less than 1% of all patients. The ratio of cases of spinal hydatid disease to disease of the brain is about 1:2. Destruction of the vertebra causes mechanical instability and secondary neurologic damage.

In the spine, the cysts are usually microvesicular, multiple, and invasive. Classically, spinal hydatidosis is classified into five types: intramedullary, intradural extramedullary, epidural, vertebral, and paravertebral (Fig. 27.1). Sometimes, *dumbbell appearance* may be seen. Dumbbell type refers to spinal hydatidosis which has both a component within the spinal canal and a component in the paravertebral space linked by hydatid cysts traversing the neural foramen. Vertebral body lesions with epidural extension are the most frequent form, in which neural, vertebral, bony, and soft tissues are involved at the same time (Figs. 27.2, 27.3, 27.4, and 27.5). On the other hand, intradural hydatidosis is rare. The most common sites occupied are the lower thoracic and lumbar segments. The majority of patients are men.

## Clinical Presentations

It is well known that spinal hydatidosis remains asymptomatic for a long time due to its slow development. Symptoms and signs depend on the area involved and on the size, number, and the extension of the cystic lesions; the effects may vary from simple rachialgia to complete paraplegia or tetraplegia.

The most common presenting symptoms are chronic back pain, radiculopathy, lower limb weakness, sphincter disturbances, and spinal deformity. Spinal injury may induce a pathologic fracture with acute neurologic symptoms. Other signs and symptoms may be related to hepatic and pulmonary involvement. A past history of hydatid disease will be suggestive.

## Imaging Features

Plain X-ray may find multiple, well-defined, osteolytic, expansile cavitatory vertebral areas ("moth-eaten" lesions) without periosteal reaction or sclerosis. A chest X-ray can show concomitant pulmonary hydatid cysts (Fig. 27.6).



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_27

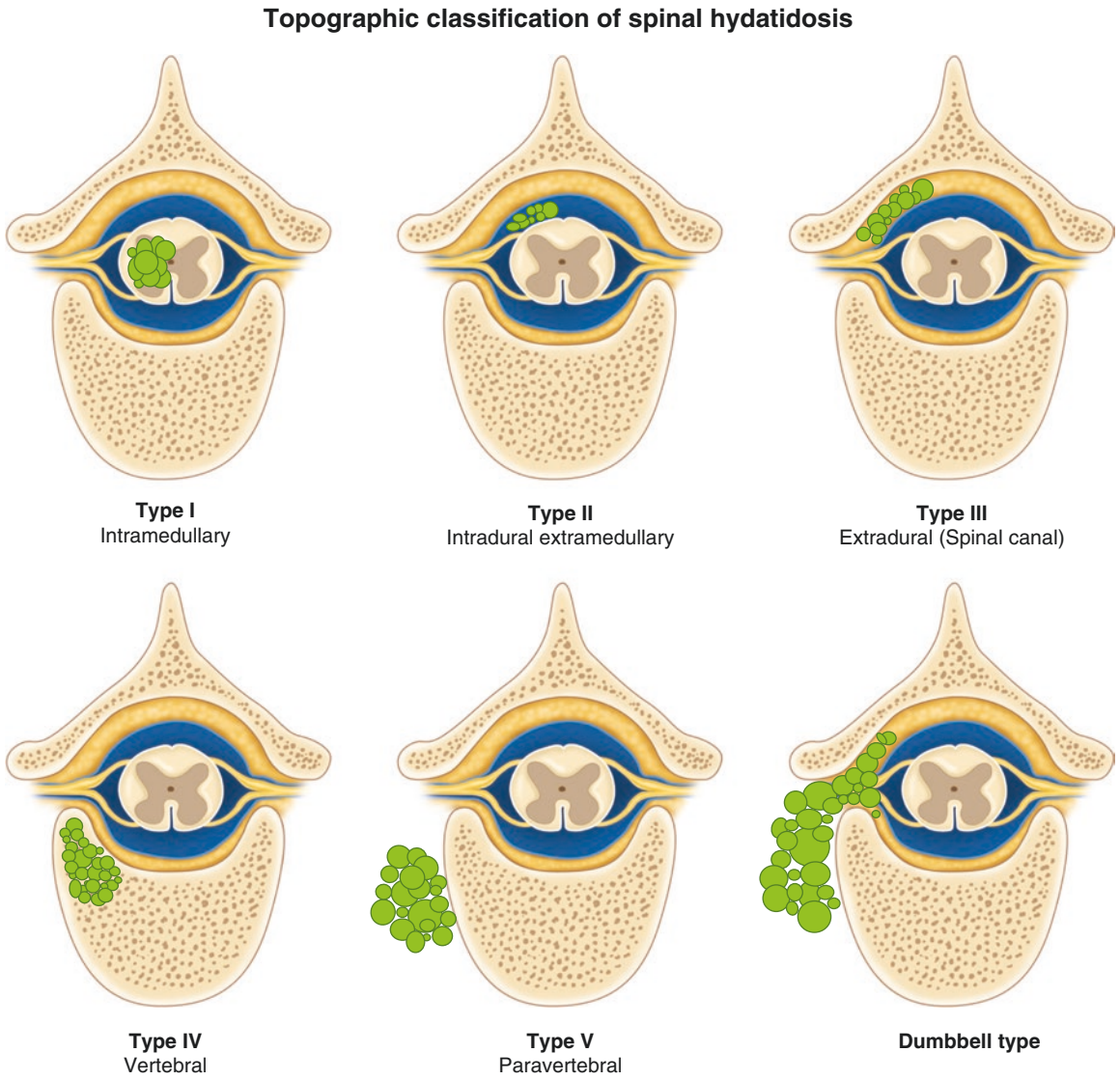


**Fig. 27.1** Topographic classification of spinal hydatidosis

CT scan reveals irregular bony erosions of the vertebral body, the posterior neural arch, and/or the ribs (Fig. 27.7). The occurrence of "classic" spherical lesions with several daughter cysts is common in the paraspinal soft tissues. Contrast enhancement is rare and is often related to bacterial coinfection.

Myelo-CT scan may be useful, showing a detailed bony resolution as well as the compression of the thecal sac. Myelography carries a risk of spreading the disease through accidental cyst puncture, however.

On MRI, the cysts appear multiloculated, with thin walls and irregular branching; they may resemble a bunch of grapes (Figs. 27.8 and 27.9). These fluid-filled lesions have a low intensity on T1-weighted images and high intensity on T2-weighted images. The modification in signal intensity may suggest loss of cyst viability. Gadolinium-enhanced T1-weighted images classically show no enhancement. Enhancing ring or granulomatous lesions are observed in the case of infected cysts. A spinal hydatid cyst in an intradural location will appear oblong, like a sausage. Diffusion-

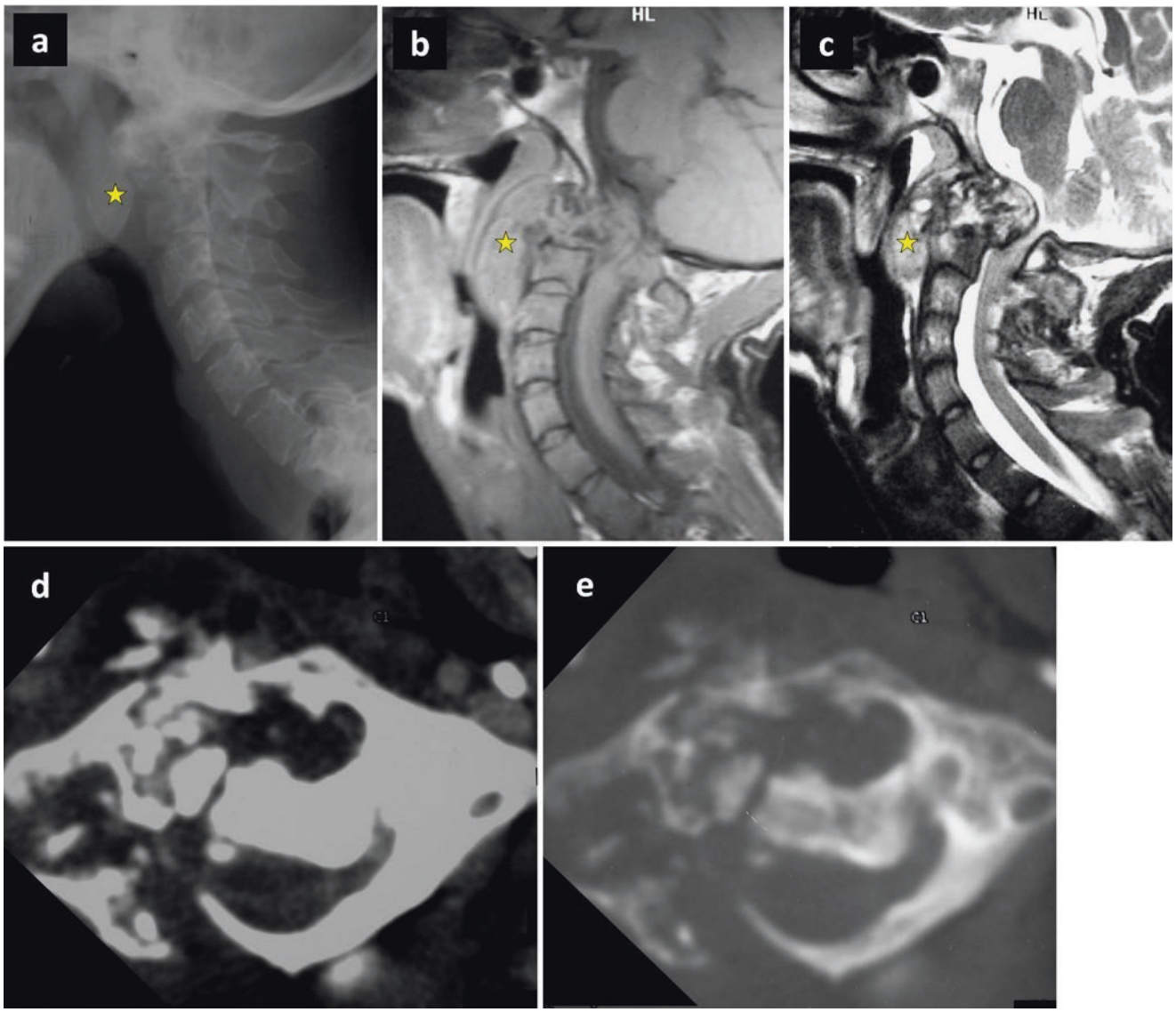


**Fig. 27.2** Hydatid disease involving the craniocervical junction. Lateral plain radiography (**a**), sagittal T1-weighted MRI (**b**), T2-weighted MRI (**c**), axial CT scan in bone window (**d**), and parenchymatous window (**e**). The cystic lesion involves the odontoid process, the body of C2, and the anterolateral part of C1 with retropharyngeal (*star*) and intracanalar extensions. Note the suboccipital luxation and the bulbar compression

weighted images can help to differentiate hydatid cysts from abscesses and other cystic lesions.

Paraspinal and associated abdominal lesions may be assessed by ultrasonography or CT scan.

Differential diagnosis can include developmental cysts (epidermoid, dermoid, teratoma, neurenteric cysts), meningocele, arachnoid cyst, Tarlov cyst, synovial cyst, hemangioma, metastasis, plasmacytoma, schwannoma, aneurysmal bone cyst, cysticercosis, and Pott's disease.

## Laboratory Findings

Negative results of serologic tests have no significance in spinal hydatidosis. A positive reaction with important eosinophilia adds a few points to the diagnosis. The existence of complications (rupture or infection) is the most important factor influencing a positive serology.

On microscopic examination, the cyst contains germinating parasitic particles called *hydatid sand*.

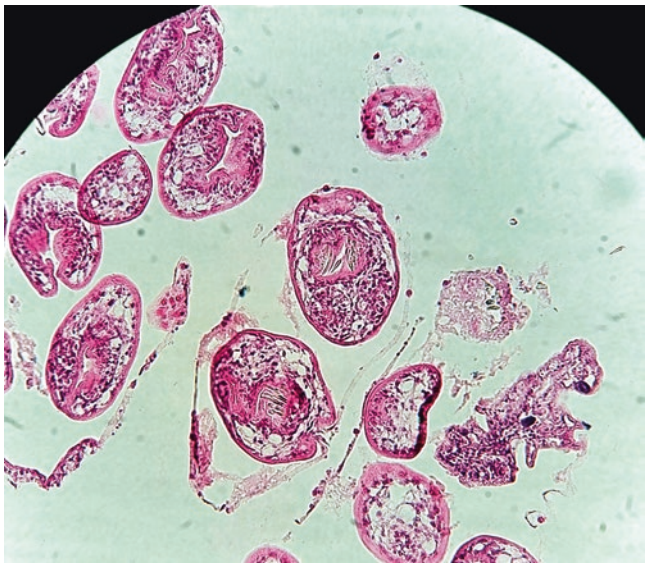

**Fig. 27.3** Photomicrograph showing various protoscoleces (medium-power magnification; periodic acid–Schiff staining) (Courtesy of Pr. R. Moutaj, PharmD; Marrakech, Morocco)

## Treatment Options

The initial treatment of choice for spinal hydatidosis is surgical neural decompression, with complete excision of the cysts, when possible. Unlike most brain hydatidosis, however, en bloc cyst resection without rupture is almost impossible. Various spinal approaches can be used, including anterior, posterior, posterolateral, or combined approaches. Spinal instability and deformity are managed when necessary to prevent potential neurologic complications. Fusion and/or instrumentation (plates, rods, and hooks) may be used regardless of infection.

Irrigation of the operative field with scolicidal solutions (hypertonic saline or oxygenic water) may be used to prevent future local recurrence. Anaphylactic reaction is another complication observed when intraoperative spillage occurs.

Anthelmintic agents (mainly albendazole and mebendazole) may be given in the perioperative period to prevent recurrences, but their efficacy is not well established.

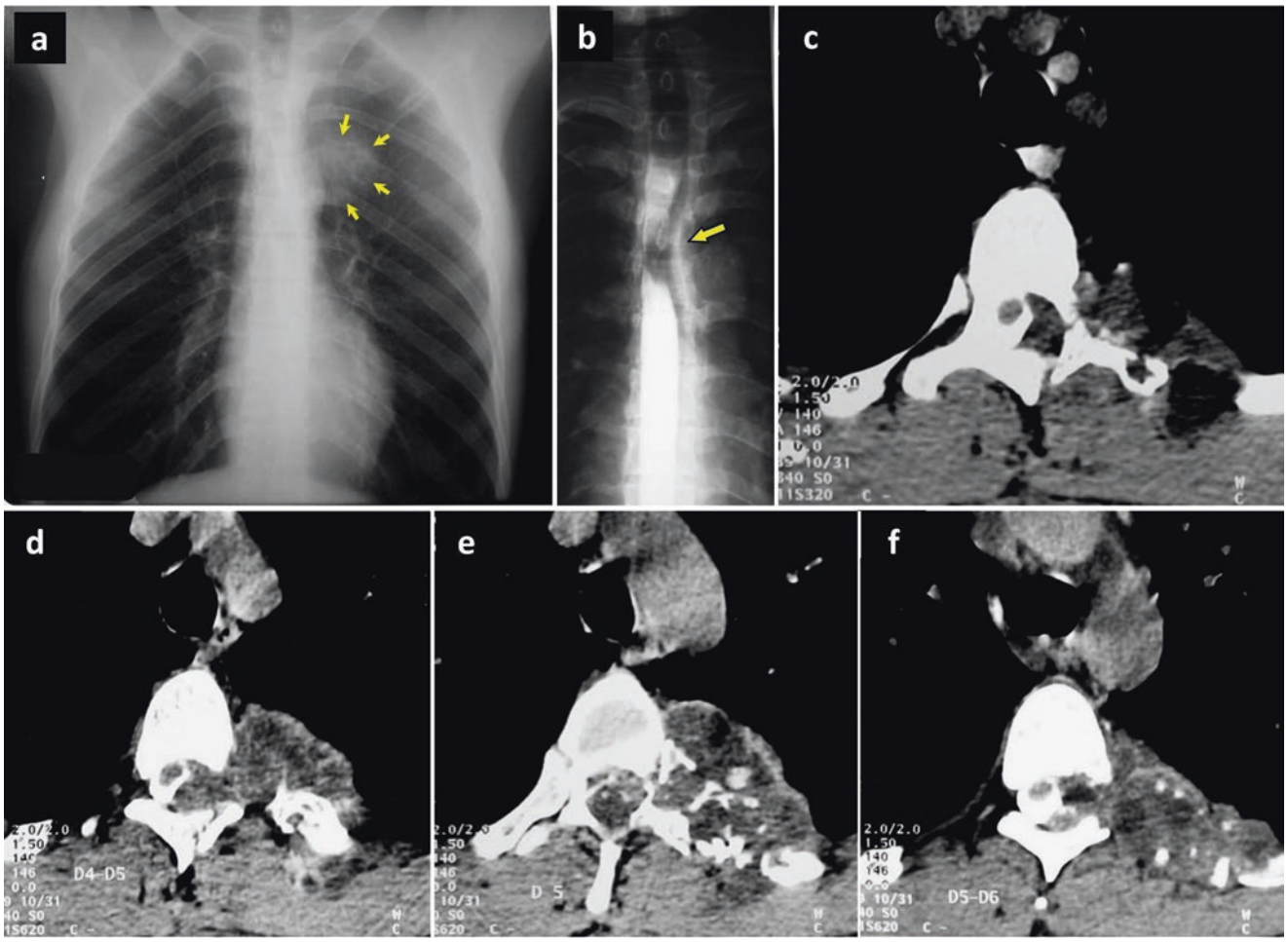


**Fig. 27.4** Intraspinal and paraspinal extradural hydatid cysts (dumbbell type) of the left thoracic region, causing thecal and spinal cord compression. Plain chest radiography (**a**), myelography (**b**), and post-myelography CT scans (**c**–**f**)

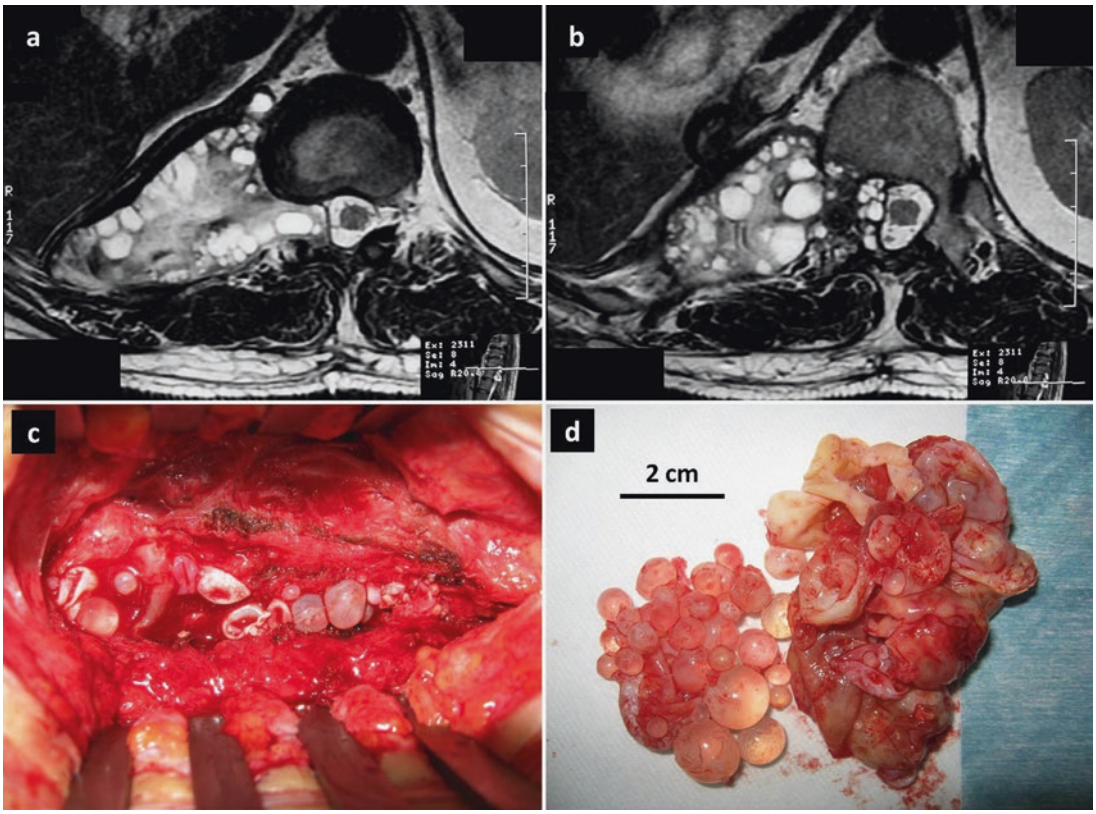


**Fig. 27.5** Intraspinal and paraspinal epidural hydatid cysts (dumbbell type) of the right thoracolumbar area, causing spinal cord compression. Axial T2-weighted MRI (**a**, **b**). Operative view after posterior dorsolumbar exposure and laminectomy, showing multivesicular hydatidosis in the epidural space (**c**). A variety of multiple hydatid cysts (**d**)

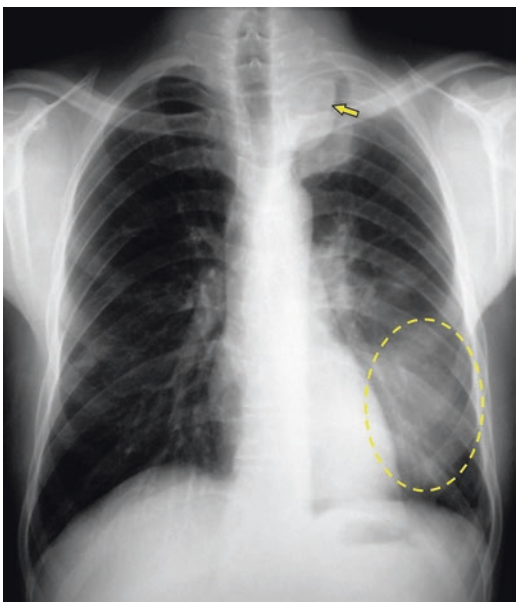

**Fig. 27.6** Case 27.1. Plain chest radiography showing multiple hydatid cysts located in the *left* thoraco-pulmonary area: *upper* thoracic paraspinal (*arrow*) and retrocardiac (*oval dotted line*)

## Outcomes

Most patients with early treatment recover from neurologic deficits, but because of the high rate of recurrence, spinal hydatidosis has a poor prognosis and is often compared to a spinal malignant disease (called "le cancer blanc"). Thus, patients require a long follow-up period, and reoperations are generally needed (Figs. 27.10, 27.11, 27.12, 27.13, 27.14, 27.15, 27.16, and 27.17).

Complications of decubitus may lead to death in a paraplegic patient. The prognosis of other hydatid lesions in the body also should be taken into account (Fig. 27.18).

Health preventive programs should be aimed at breaking the parasite life cycle and educating rural populations in endemic regions.

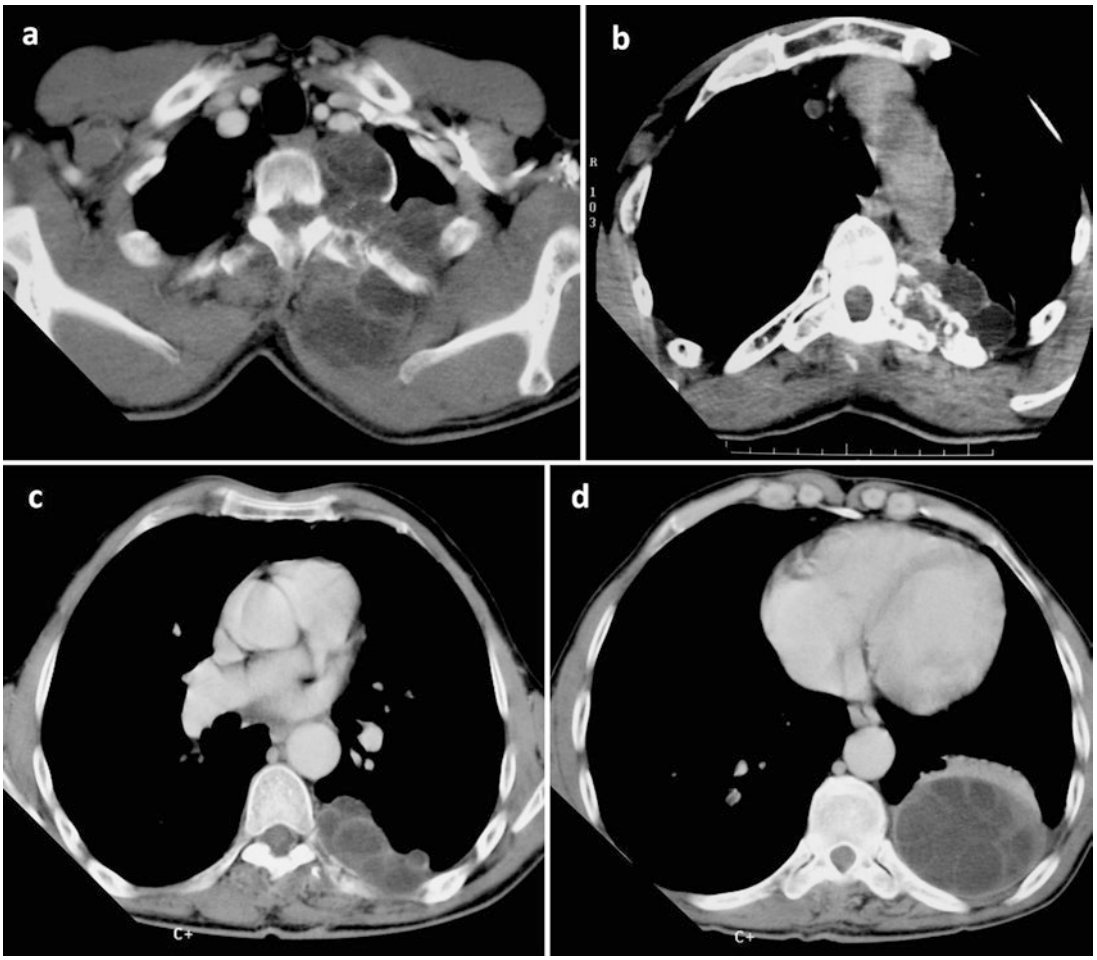


**Fig. 27.7** Case 27.1. (**a**–**d**) Axial thoracic CT scan after contrast administration, revealing hypodense, multiloculate, left thoracic paraspinal lesions with osteolysis of the posterior portion of the ribs

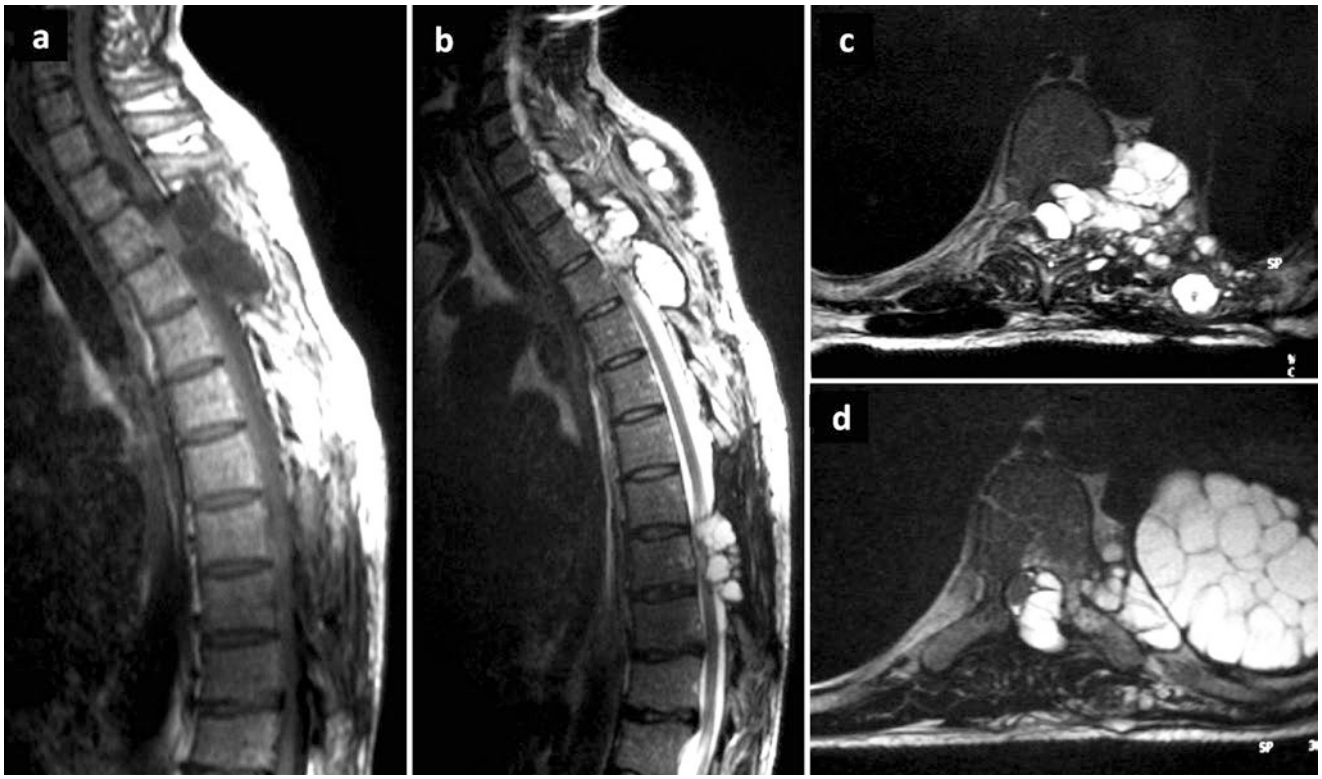


**Fig. 27.8** Case 27.1. Intraspinal cysts with spinal cord compression are better visualized on MRI. Sagittal T1-weighted MRI (**a**), T2-weighted MRI (**b**), and axial T2-weighted MRI (**c**, **d**). All the cysts are isointense to CSF

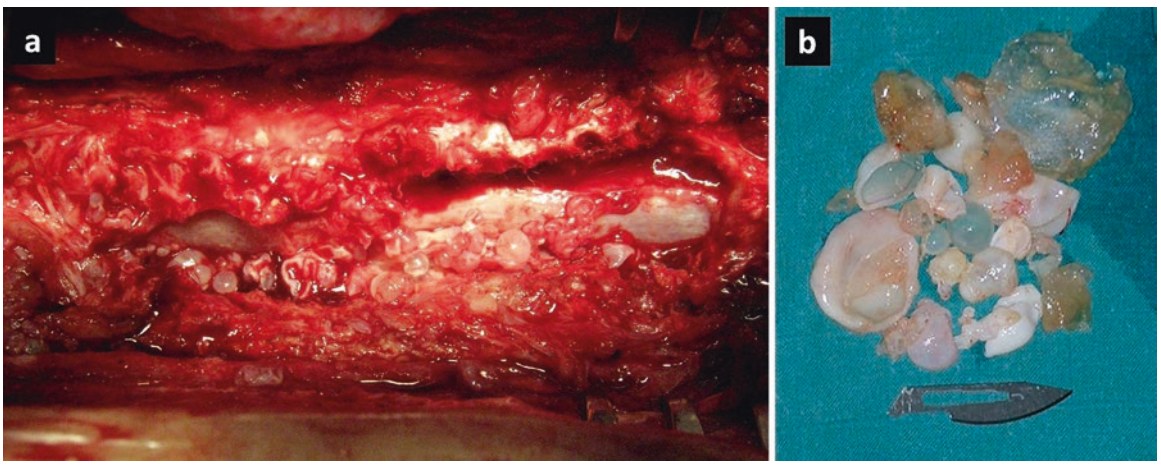


**Fig. 27.9** Case 27.1. Intraoperative view after thoracic laminectomy, showing multiple epidural cysts of *Echinococcus granulosus* (**a**). Appearance of some hydatid cysts (**b**)

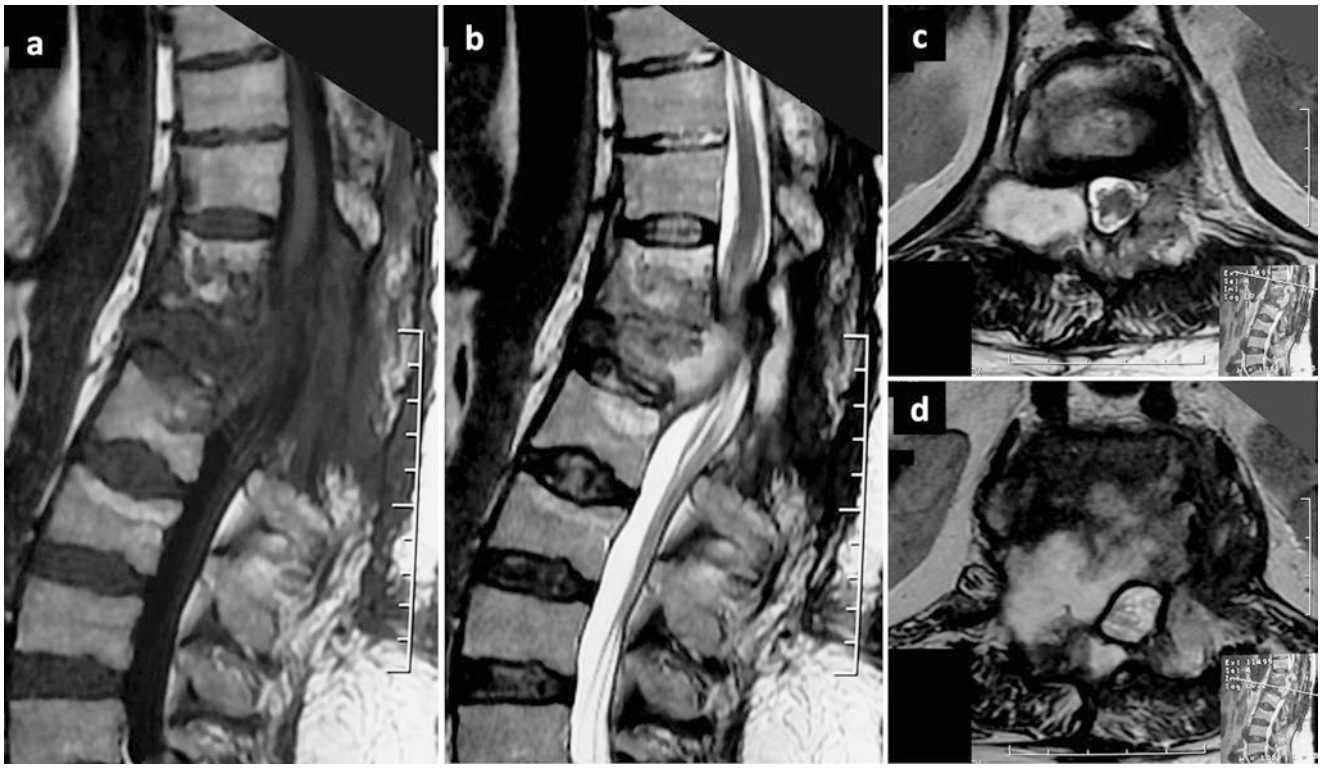


**Fig. 27.10** Sagittal T1-weighted MRI (**a**), T2-weighted MRI (**b**), and axial T2-weighted MRI (**c**, **d**) showing conus medullaris and cauda equina compression in this female patient with recurrence of disease and infected hydatid cysts 9 years after she underwent posterior laminectomy, debridement, and posterior fusion for thoracolumbar hydatid disease

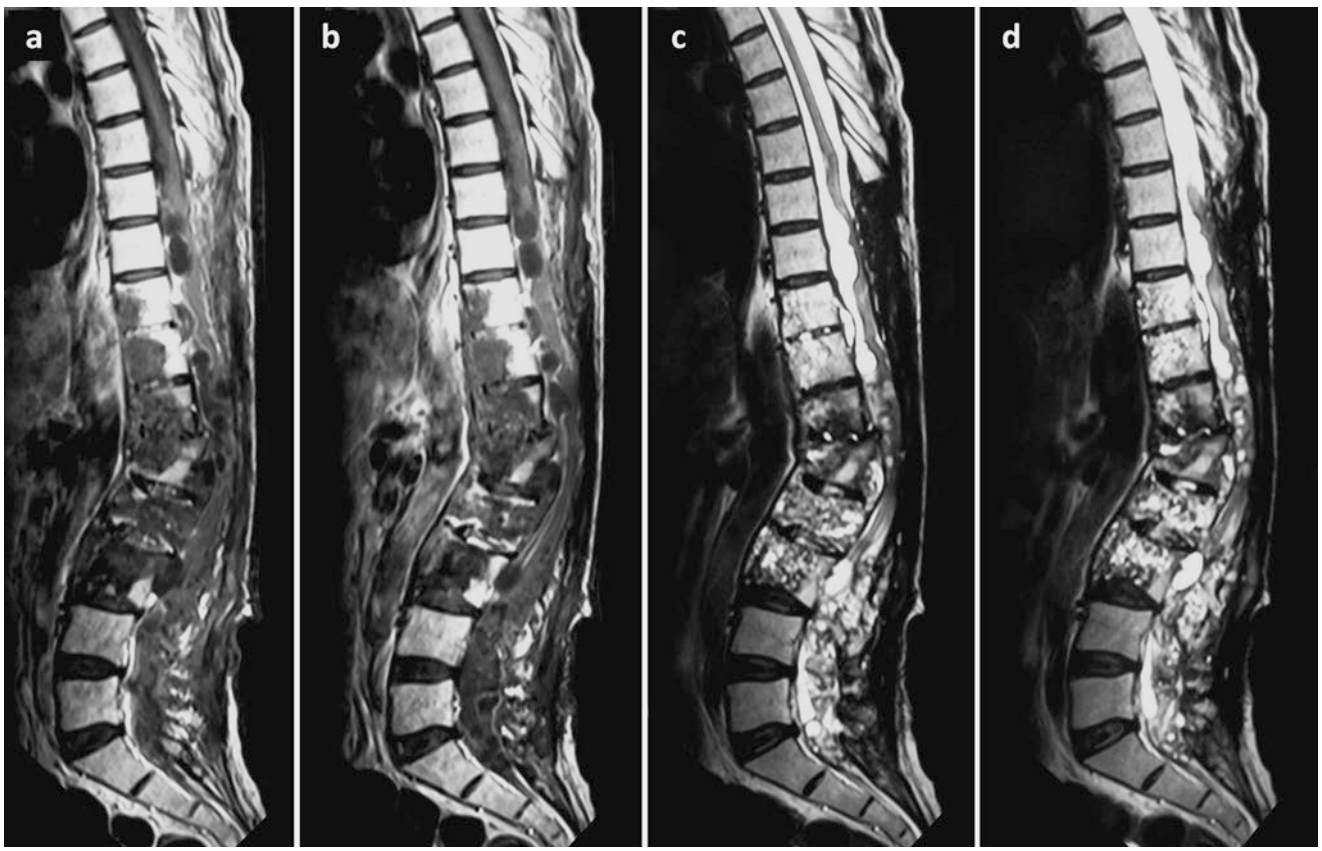


**Fig. 27.11** Extensive spinal thoracolumbar hydatid cysts involving both the epidural and the subarachnoid spaces in a patient who has undergone multiple operations. Sagittal T1-weighted MRI (**a**, **b**) and T2-weighted MRI (**c**, **d**)

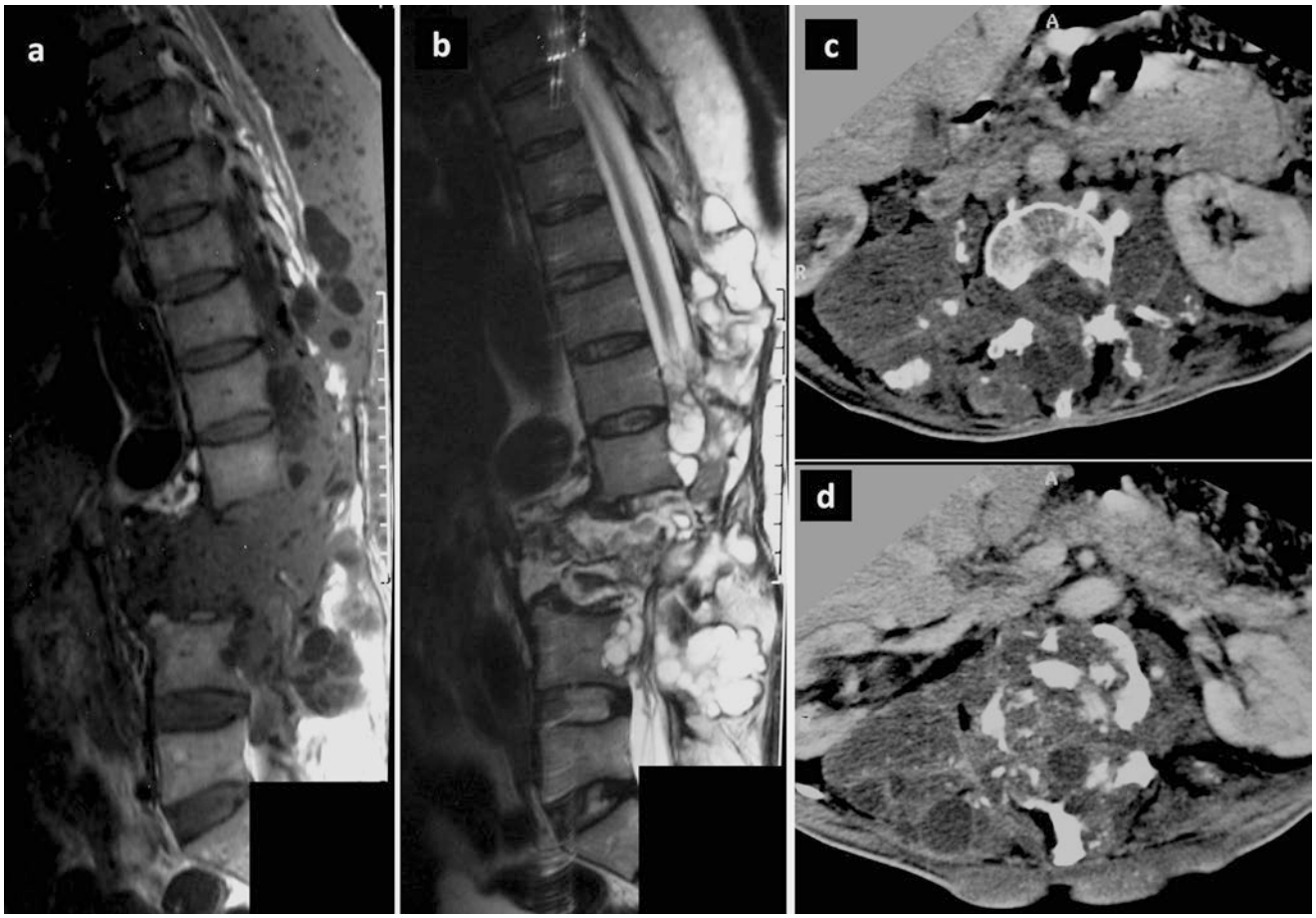


**Fig. 27.12** Sagittal T1-weighted MRI (**a**) and T2-weighted MRI (**b**) viewing complex and extensive multivesicular lesions in the spinal lumbar and thoracic regions, with important L2–L3 vertebral luxation. Axial CT scanning after contrast injection (**c**, **d**) shows bony erosions of the vertebral body and the posterior neural arch of the L2 and L3 vertebrae, with extensive intracanalar and paraspinal cystic lesions

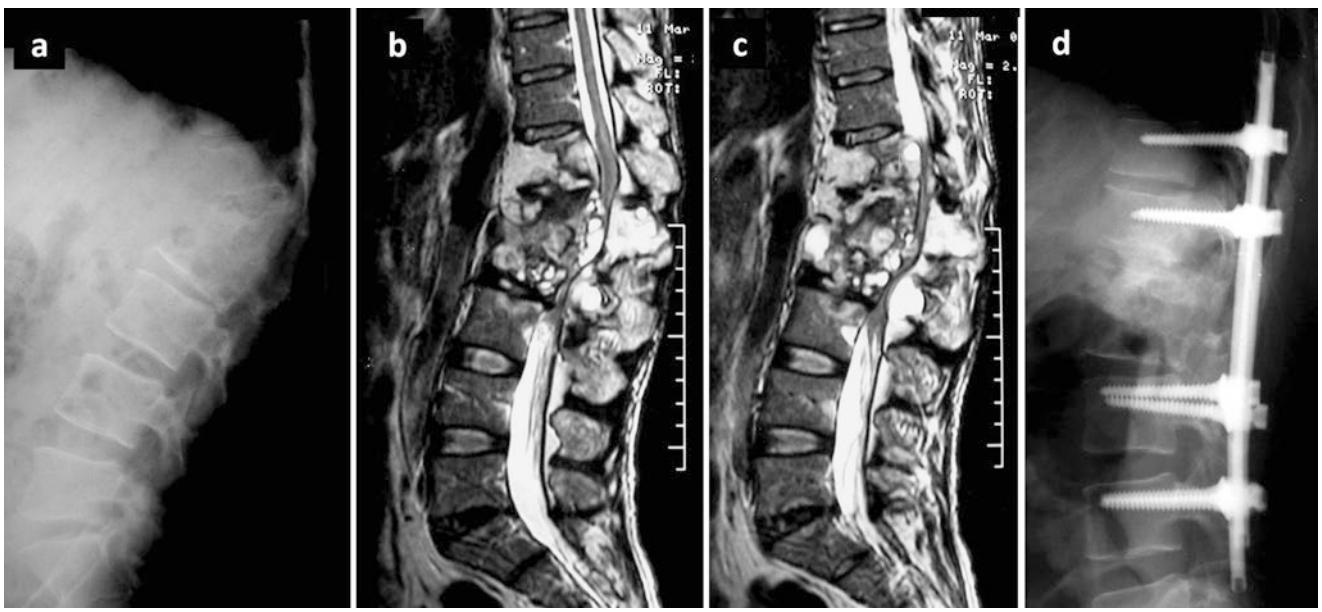


**Fig. 27.13** Lateral spinal thoracolumbar X-ray (**a**) and sagittal T2-weighted MRI (**b**, **c**) showing vertebral body osteolysis of T12–L1–L2 with spinal subluxation and spinal cord compression. Postoperative lateral view (**d**) after posterior decompression and instrumentation

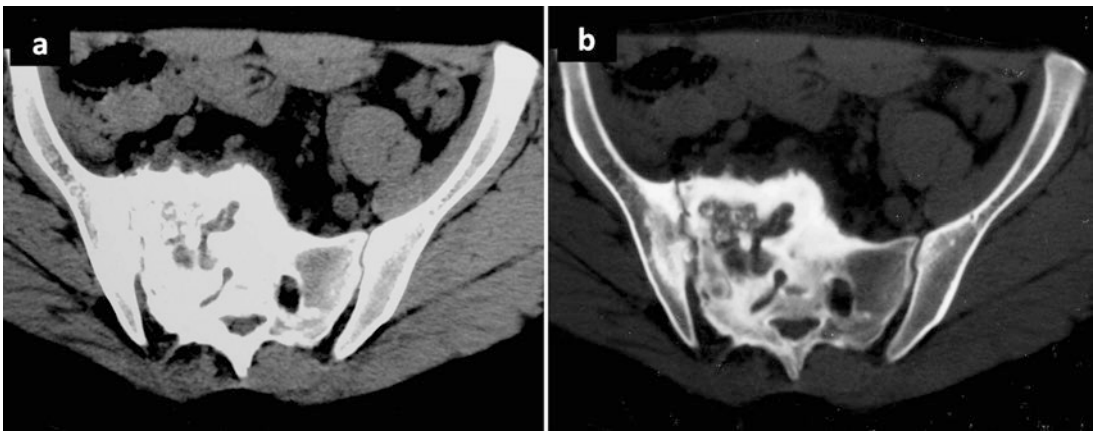


**Fig. 27.14** Hydatid cyst of the right side of the sacrum with sacroiliac involvement. Axial CT scan on parenchymal window (**a**) and bone window (**b**)

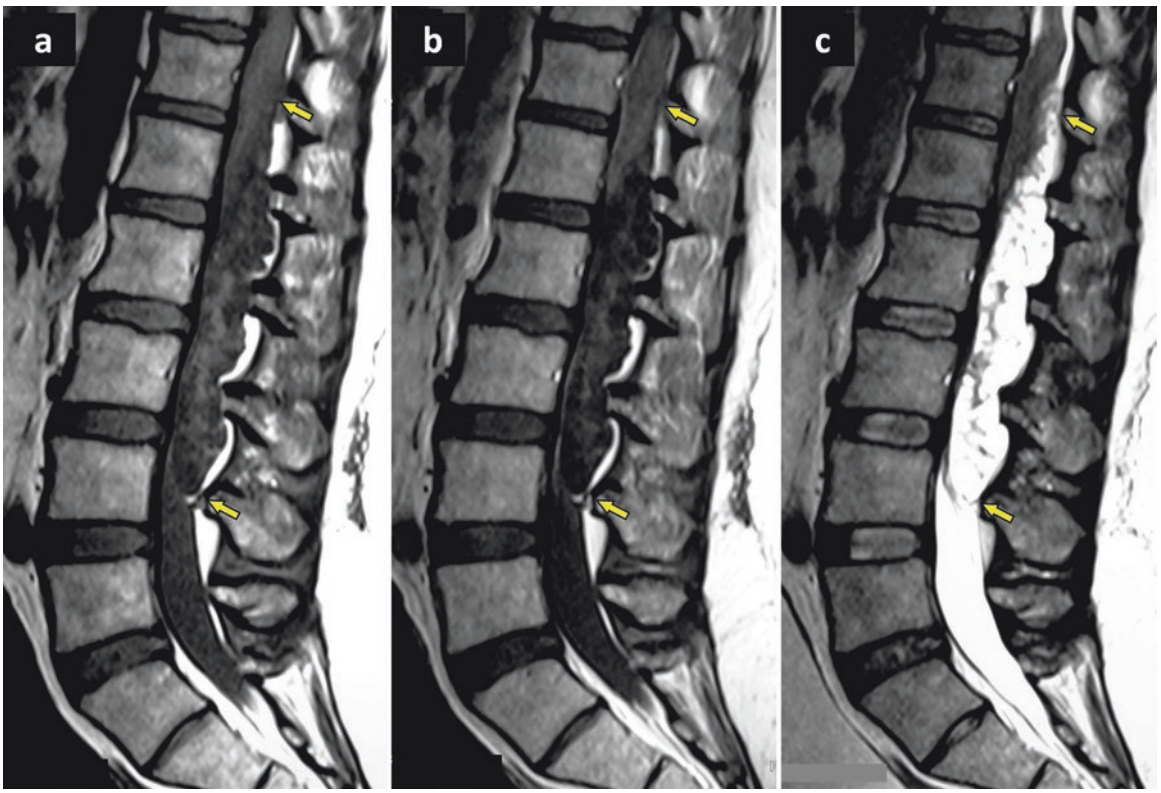


**Fig. 27.15** Case 27.2. Spinal posterior thoracolumbar multivesicular hydatid lesion from T12 to L4 (*arrows*) without bone erosion. Sagittal T1-weighted MRI before (**a**) and after (**b**) gadolinium administration, and T2-weighted MRI (**c**)

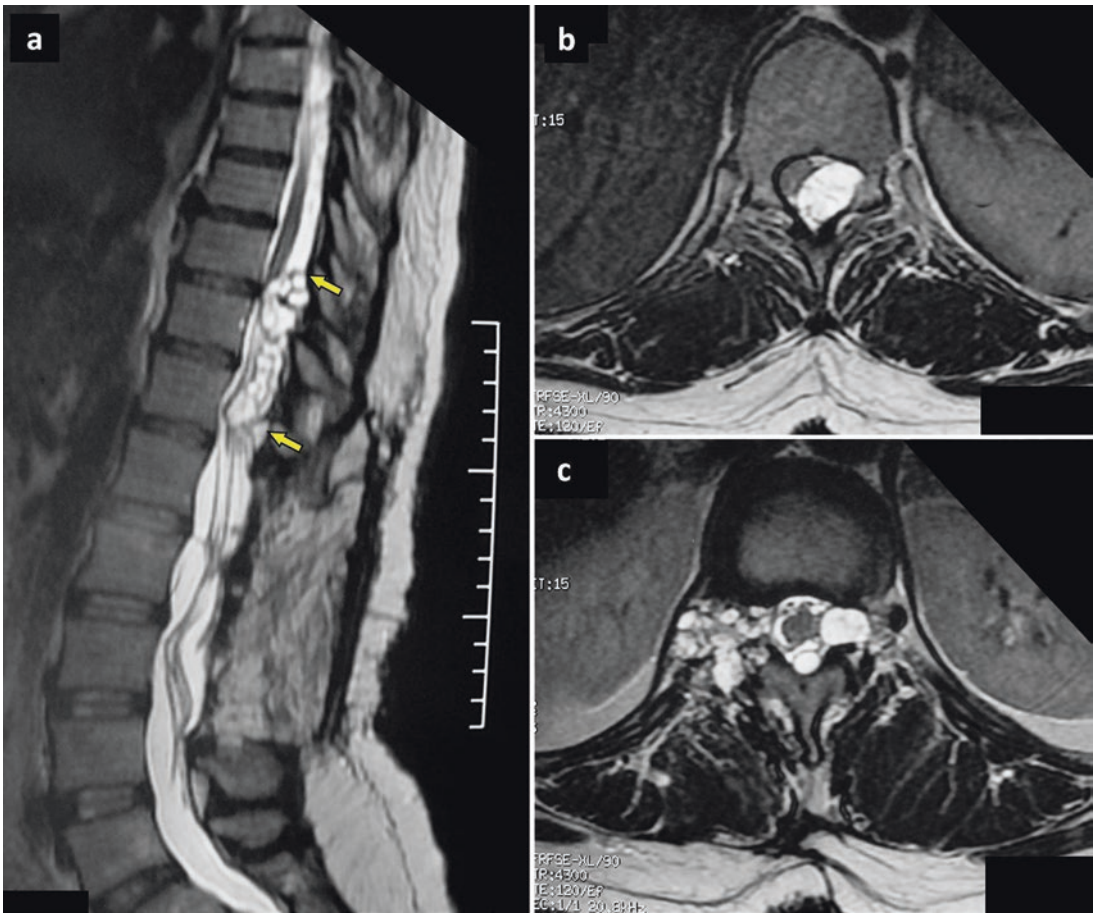


**Fig. 27.16** Case 27.2. This patient underwent posterior limited laminectomy (L3 to L1) with debridement. Unfortunately, she presented with disease recurrence 20 months later, owing to incomplete cyst resection (*arrows*), as seen on sagittal (**a**) and axial T2-weighted MRI (**b**, **c**). Laminectomy from T12 to T10 was performed, with a good recovery

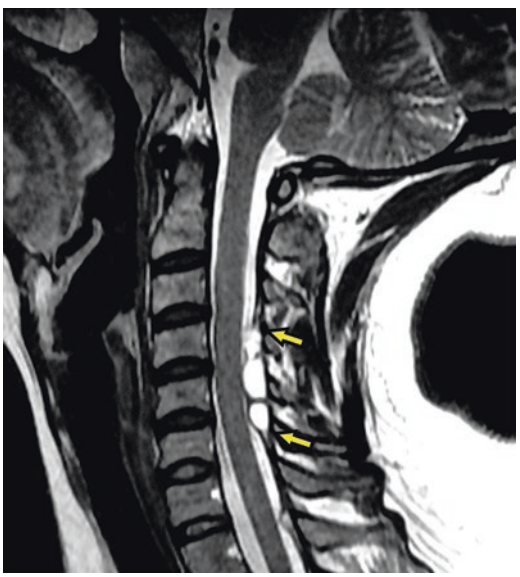

**Fig. 27.17** Sagittal T2-weighted MRI shows spinal posterior cervical epidural cystic lesions with mild spinal cord compression from C4 to C6 (*arrows*)

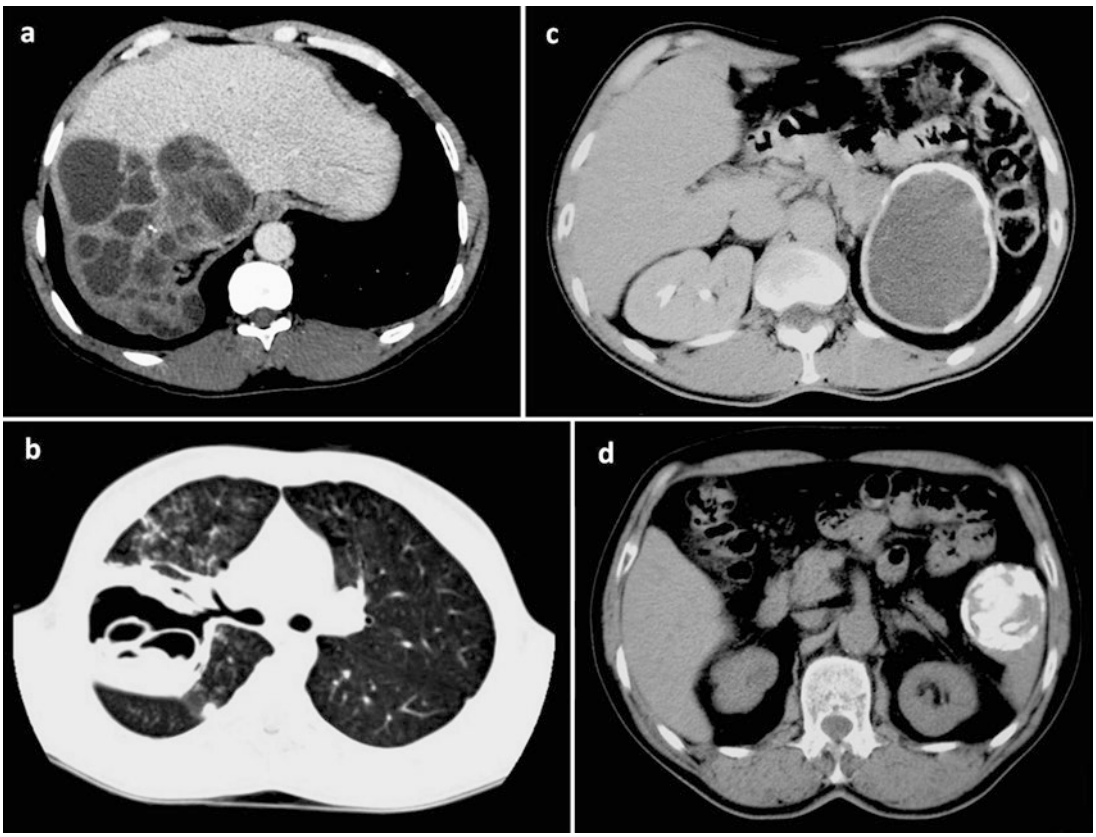


**Fig. 27.18** Axial CT scan showing hydatid disease of the liver (**a**), the left kidney (**b**), the right lung (**c**), and the spleen (calcified) (**d**)

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# Other Parasitic Infections of the Central Nervous System

# 28

Many parasitic infections involve the central nervous system. Although they are still unusual in daily practice, it is important to be aware of these emerging diseases and to think about them in the differential diagnosis of appropriate cases. Most are medically treated conditions that do not require surgical intervention. The neurosurgeon's role in the management of most patients with neuroparasitosis is in providing tissue by biopsy for diagnosis, performing cerebrospinal fluid (CSF) diversion in patients with symptomatic hydrocephalus, and sometimes addressing a cerebrospinal mass effect or spinal instability. The most frequent parasitic diseases that potentially involve neurosurgical intervention are **cysticercosis**, **toxoplasmosis**, **hydatidosis**, **amebiasis**, and **schistosomiasis.** An early diagnosis and aggressive treatment are important to achieve the best outcome possible. Control and preventive programs are needed in areas of endemicity to reduce these infectious diseases.

## Cysticercosis

Neurocysticercosis is discussed in Chap. 25.

## Toxoplasmosis

Toxoplasmosis is a worldwide parasitic infection due to the intracellular coccidian protozoan *Toxoplasma gondii*. The causative pathogen is acquired by eating inadequately cooked meat, by accidental ingestion of contaminated cat feces (contamination of water, soils, vegetables, and fruits), or via maternal transmission to her fetus. From the small intestine, propagation may occur to all organs, including the brain, heart, eye, skeletal muscle, placenta, and fetus. Although most infections are asymptomatic, there are three main types of neurotoxoplasmosis: congenital toxoplasmosis, toxoplasmosis in immunocompetent hosts, and an opportunistic infection in immunocompromised patients, including those with acquired immunodeficiency syndrome (AIDS), malignancy, or a history of organ transplantation. Chapter 30 includes information on toxoplasmosis in HIV patients and includes images of toxoplasmosis-related granulomatous lesions and encephalitis in these patients (Figs. 30.3, 30.4, 30.5, 30.6, 30.7, 30.8, 30.9, 30.10, 30.11, 30.12, and 30.13).

CNS invasion may present as a mass lesion (toxoplasmosis abscess), encephalopathy, or meningoencephalitis, which is the most frequent and often most serious manifestation of toxoplasmosis in AIDS patients. On the contrary, congenital toxoplasmosis manifests as chorioretinitis, encephalomyelitis, hydrocephalus, and microcephaly. Patients with abscess formation present with focal neurologic deficits related to the location of the lesions. Spinal cord involvement (myelitis) is uncommon.

The diagnosis is made by a combination of serologic testing (on blood, CSF, or both) and tissue biopsy. In histopathologic studies, the parasites are best seen in tissue section stained by the Giemsa technique. There are areas of tissue necrosis delimited by an important mononuclear reaction.

Neuroimaging findings are variable. The congenital form manifests in the newborn as hydrocephalus, microcephaly, and calcifications (mainly in the periventricular areas, the basal ganglia, and the subcortical regions). Toxoplasma abscesses are often bilateral and multiple. On CT scan and MRI, there are focal, rounded lesions with variable adjacent edema and ring enhancement (Figs. 28.1, 28.2, 28.3, and 28.4). Most lesions are subcortical; they are often located in the basal ganglia and corticomedullary junction. The differential diagnosis of neurotoxoplasmosis, especially in immunocompromised patients, includes lymphoma, progressive multifocal leukoencephalopathy, cytomegalovirus encephalitis, pyogenic abscess, tuberculomas, and other opportunistic infections such as fungal pathogens.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_28

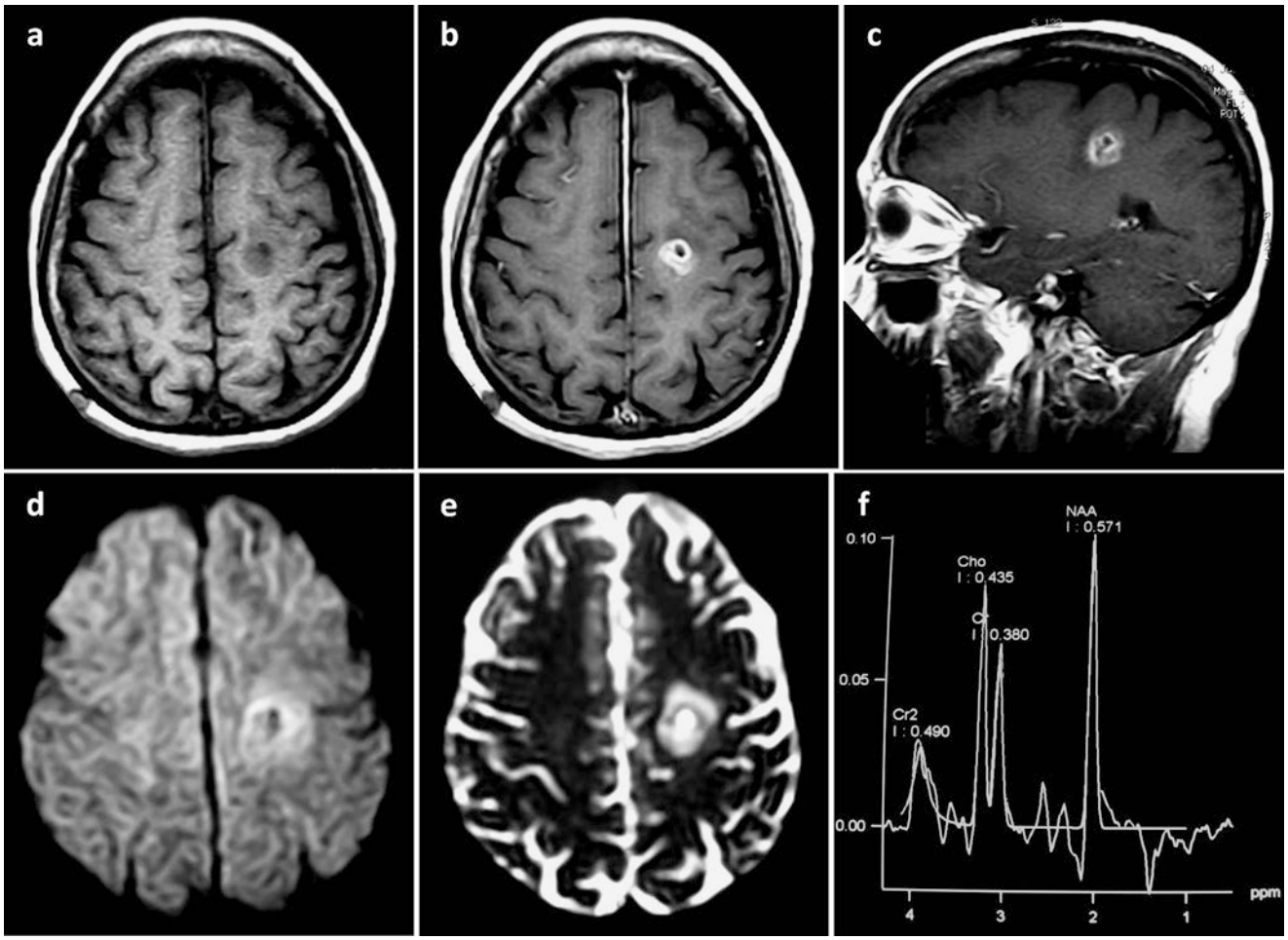


**Fig. 28.1** Brain granuloma caused by toxoplasmosis. Axial T1-weighted MR image before (**a**) and after (**b**) gadolinium administration. Sagittal enhanced T1-weighted image (**c**), diffusion-weighted image (**d**), apparent diffusion coefficient map (**e**), and MR spectroscopy (**f**). There is a solitary ring-enhancing lesion with perilesional edema in the left frontal lobe

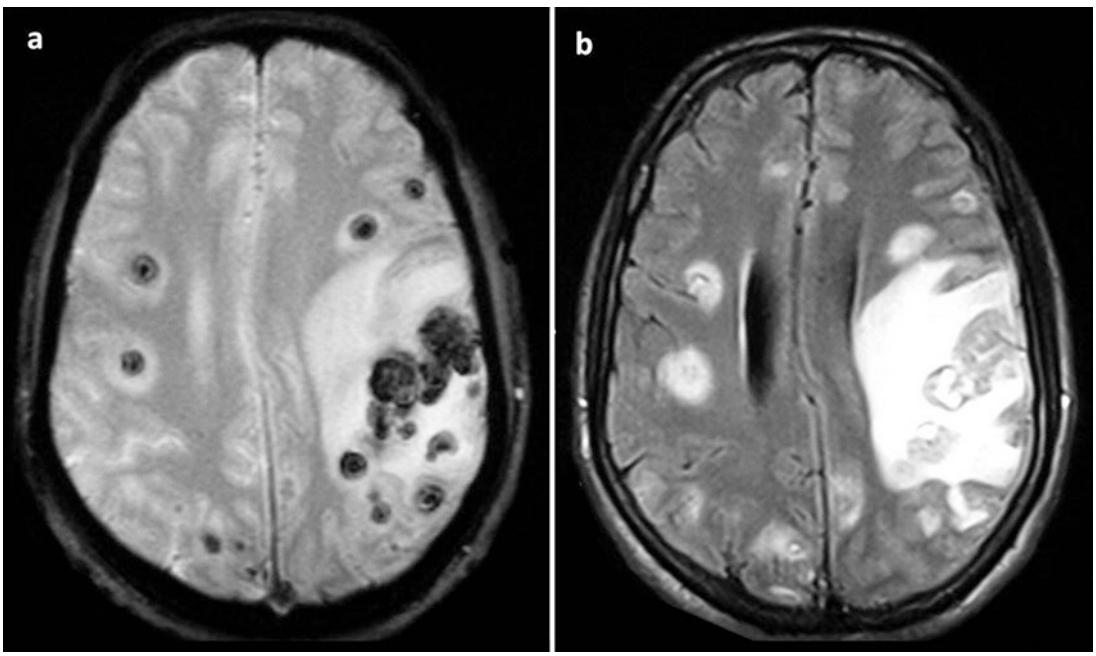


**Fig. 28.2** Multiple brain granulomatous lesions due to *Toxoplasma gondii* shown on proton density weighted image (**a**) and on a fluid-attenuated inversion recovery (FLAIR) sequence (**b**). Note the important, extensive perifocal edema

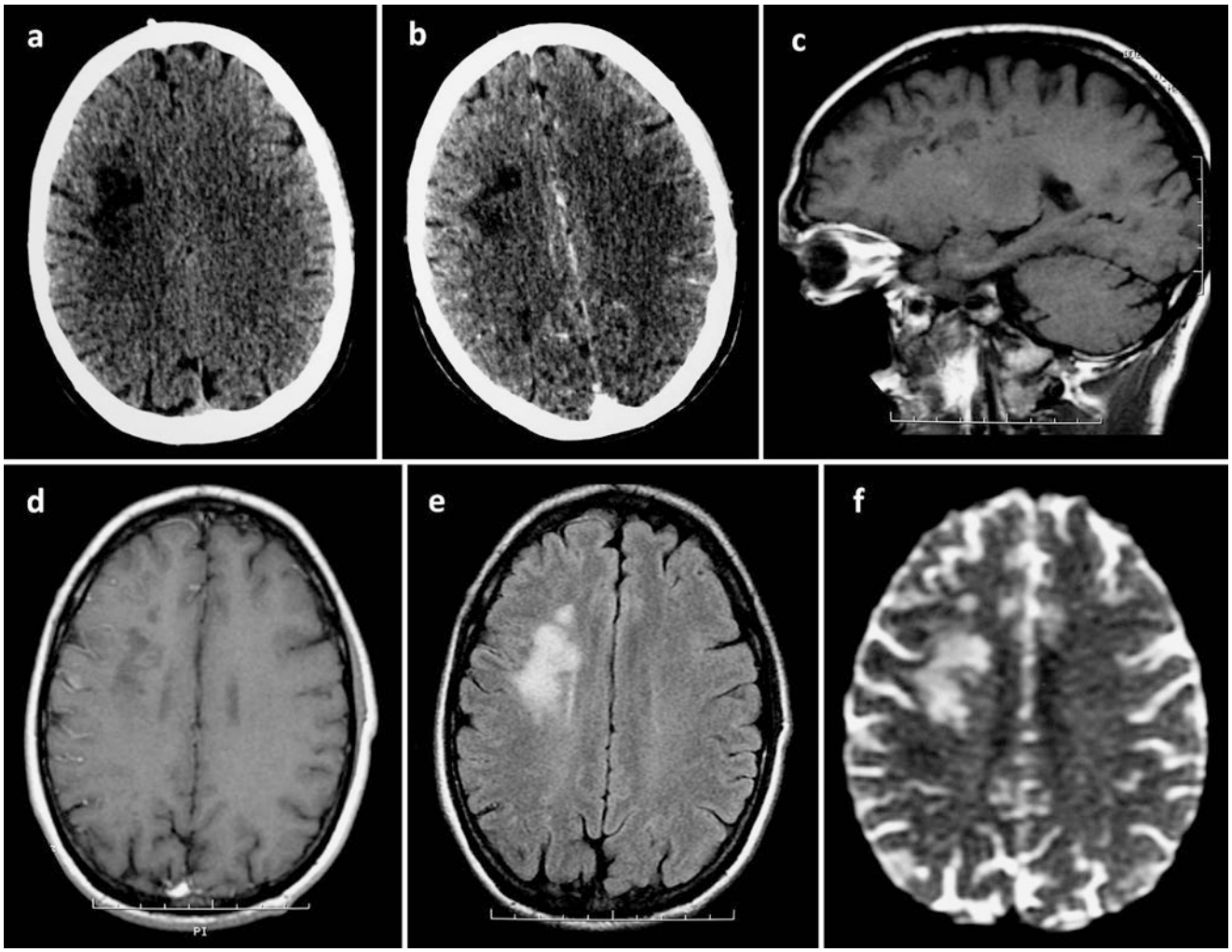


**Fig. 28.3** Localized right frontal subcortical encephalitis in a 43-year-old man who presented with epileptic seizures. The diagnosis of toxoplasmosis was made by serologic testing (on CSF). Shown are an axial cranial CT scan without (**a**) and with (**b**) contrast injection; sagittal T1-weighted image without gadolinium (**c**); an axial enhanced T1-weighted image (**d**); a FLAIR sequence (**e**); and an apparent diffusion coefficient map (**f**)

Treatment of congenital toxoplasmosis requires pyrimethamine and sulfadiazine. In patients allergic to sulfadiazine, clindamycin, atovaquone, and trimethoprim/sulfamethoxazole may be an alternative. In immunocompromised patients, folinic acid should be added to pyrimethamine/sulfadiazine for a total duration of 4–6 weeks. Spiramycin is the best treatment for pregnant women. Surgical procedures are reserved for brain lesions with significant mass effect and those that do not respond to medical therapy, as well as for biopsy for a definitive diagnosis and CSF diversion for patients with symptomatic hydrocephalus.

Hydrocephalus, permanent focal motor or sensory deficits, or seizures are the most frequent residual disabilities, especially in immunocompromised hosts. For untreated infants, congenital toxoplasmosis can cause severe neurologic damage and blindness. Neurotoxoplasmosis-related mortality is significantly high in AIDS patients. The most important factors associated with deaths and neurologic sequelae are extreme ages, poor level of consciousness, and

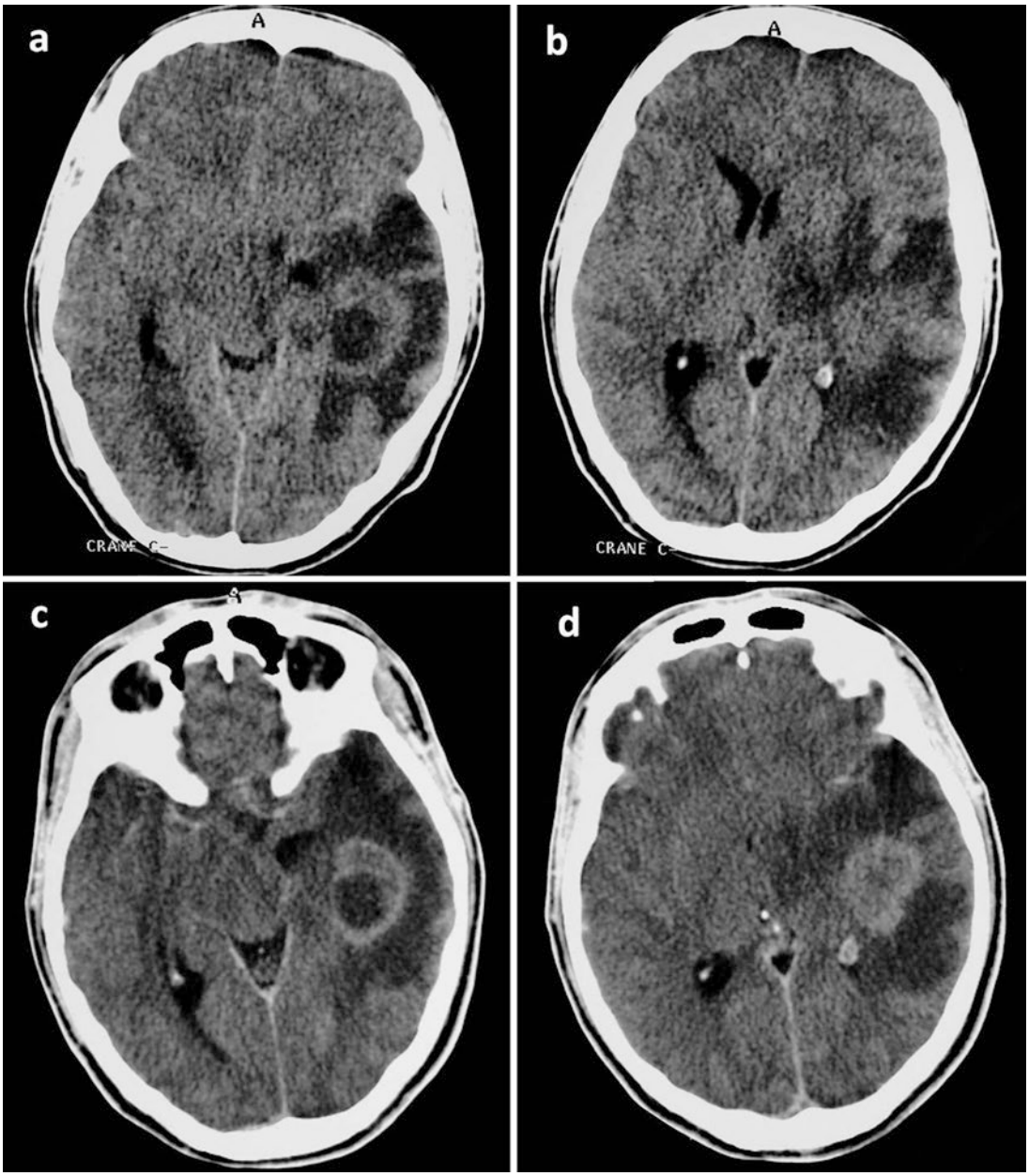


**Fig. 28.4** Toxoplasmic abscess of the left temporal lobe in a 30-year-old man who presented with behavior disorders. Axial cranial CT scans before (**a**, **b**) and after (**c**, **d**) contrast administration show a ring-enhancing, granulomatous lesion with surrounding edema

cognitive impairment. In any case, the overall prognosis depends on the severity and extent of brain involvement, in addition to underlying conditions. In the AIDS population, prophylactic treatment with trimethoprim/sulfamethoxazole is used against toxoplasma encephalitis.

## Hydatidosis

Chapters 26 and 27 cover hydatidosis of the brain and the spine.

## Amebiasis

Amebiasis is a widespread parasitic disease caused by the protozoans *Entamoeba histolytica* (see Fig. 2.25), *Naegleria fowleri*, *Acanthamoeba astronyxis*, and *Balamuthia mandrillaris*. Human contamination occurs during contact with infected water. CNS infections are rare but have life-threatening consequences. Amebiasis is most common in developing countries of the tropics. Both previously healthy and immunocompromised patients may be involved. There

are three major types of amebic infection: brain abscess (due to *E. histolytica*), primary amebic meningoencephalitis (due to *N. fowleri*), and granulomatous meningoencephalitis (due to *A. astronyxis* and *B. mandrillaris*).

In brain abscess, most symptoms result from increased intracranial pressure, local mass effect, and meningismus with or without fever. A concomitant liver abscess may be associated. *Granulomatous amebic meningoencephalitis* (GAM) is more indolent, resulting in classic subacute encephalitis with headache, fever, seizures, hemiplegia, and altered mental status. Skin lesions (purple nodules) may be found. *Primary amebic meningoencephalitis* (PAM) is characterized by change in taste or smell, with rapid progression to coma and death. There is often a recent history of swimming.

Diagnosis is done on culture, serology, immunofluorescence on CSF or brain biopsies, or more recently with polymerase chain reaction. Neuroimaging findings are nonspecific. In PAM, CT scans and MRI show nonspecific brain swelling with enhancing leptomeninges and cisterns around the anterior cranial fossa after contrast administration. In cases of GAM, neuroimaging studies detect multiple punctate, nodular, or small ring-enhancing lesions with surrounding edema, mainly in the frontal and parietal lobes and basal ganglia.

Excluding brain abscess due to *E. histolytica* (metronidazole therapy), treatment of CNS amebiasis is habitually late and nonspecific. There is no well-established treatment for CNS amebiasis. Aggressive treatment with amphotericin B, rifampin, fluconazole, pentamidine, or metronidazole is used, but the mortality rate is high. Steroids and anticonvulsant drugs can be administered if needed. Surgically, some localized lesions may be drained or excised. CSF diversion should be performed for patients with symptomatic hydrocephalus.

Except for *E. histolytica*, other amebic infections are difficult to treat and are fatal in more than 90 % of patients, even those who are immunocompetent. Long-term morbidity in survivors is usually low. Prevention of amebiasis and adequate management of dysentery is mandatory.

## Schistosomiasis

Schistosomiasis (bilharziasis) is a parasitic infection caused by the trematode platyhelminth of the genus *Schistosoma*. Five species of *Schistosoma* infect humans: *S. mansoni*, *S. haematobium*, *S. japonicum*, *S. mekongi*, and *S. intercalatum*. Schistosomiasis is the third most common parasitic disease in the world. This infection is prevalent in tropical and subtropical areas. In humans (definitive hosts), the parasite enters the body through the skin following aquatic exposure to the larval form. The transformed larvae (adult worms) reach the mesenteric veins. Larvae may also migrate to the cerebral vasculature (*S. japonicum*) and the spinal cord (*S. mansoni* and *S. haematobium*), but the CNS is affected in fewer than 5% of all cases.

There are three major types of CNS infections:

- Acute schistosomal meningoencephalitis (ASM)
- Pseudotumoral encephalic schistosomiasis (PES)
- Spinal cord schistosomiasis (SCS)

In ASM (also called Katayama fever), associated symptoms are fever, headache, seizures, visual loss, meningismus, altered mental status, and focal neurologic deficits. Hemorrhagic stroke may occur, owing to cerebral vasculitis. PES may present as a tumor-like lesion with headache, seizures, and progressive focal neurologic deficits. There are three clinical presentations of SCS: medullary, myeloradicular, and conus–cauda equina syndrome. The inferior part of the spinal cord and the conus are most frequently affected. Concomitant skin allergic symptoms, fever, hematuria, and hepatosplenomegaly may be present.

Affirmation of the diagnosis is difficult. Peripheral eosinophilia and evidence of parasitic eggs in the urine or stool are irregular. Rectal biopsy may be useful for identification of eggs. CSF examinations are nonspecific, but detection of antibodies by ELISA in samples of blood or CSF is specific. Definitive diagnosis is based on the pathological demonstration of parasitic eggs in brain or spinal cord tissue acquired at biopsy. In the brain, CT scans and MRI show nonspecific signs of edema, granulomas, tumor-like lesions, and (more rarely) intracerebral hematoma. Granulomatous lesions are most often located in the cerebellum. Spinal canal lesions appear on MRI as nonspecific focal enlargement of the cord with heterogeneous gadolinium enhancement.

Treatment is based on antischistosomal agents (praziquantel or oxamniquine) with corticosteroids. In the brain, surgical procedures should be considered if large granulomas cause mass effect, if the neurologic signs and symptoms progress despite antiparasitic drugs, or if the diagnosis is not sure. Decompressive laminectomy may be an option in some cases with spinal cord compression.

The outcome of cerebral schistosomiasis is usually good. Potential neurologic sequelae are refractory seizures and cognitive impairment, especially in children. Spinal cord forms tend to be worse. A significant degree of sensory and motor impairment is common and may require prolonged rehabilitation. Preventive programs are required in areas of endemicity to reduce this disease.

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# Fungal Infections of the Central Nervous System

# 29

Although the most common origin of central nervous system (CNS) infections is bacteria, the role of fungi should not be overlooked; these infections have received much interest in recent years. Fungal diseases have a significant morbidity and mortality. Clinical presentations may be acute and fulminant or chronic and gradually progressive. CNS involvement usually takes the form of either meningitis or intraparenchymal granuloma or abscess. Most patients are immunocompromised hosts, although immunocompetent subjects can also be infected. Aspergillosis, candidiasis, and mucormycosis are almost exclusively seen in immunocompromised or severely debilitated individuals. Cryptococcosis occurs in both previously healthy and immunocompromised patients; blastomycosis and histoplasmosis commonly concern healthy subjects. Diagnosis is often difficult and is usually made by serologic testing, histological examination, and mycologic culture. Patients may require surgical support for biopsy (definitive diagnosis), management of intracranial hypertension, spinal cord decompression, and CSF shunting. Treatment is primarily medical, however, with long-term courses of antifungal drugs and control of the underlying disease. The patient's initial clinical condition and the potential concomitant diseases are the strongest predictor of clinical outcome. This chapter focuses on the fungal infections most pertinent to neurosurgical practices: **aspergillosis**, **blastomycosis**, **candidiasis**, **cryptococcosis**, **histoplasmosis**, and **mucormycosis** (zygomycosis).

## Aspergillosis

*Aspergillus* species are ubiquitous filamentous fungi manifesting mainly in conditions of immunosuppression. Predisposing conditions include HIV infection, organ transplantation, malignancy, intravenous drug use, tuberculosis, hepatic cirrhosis, and prolonged antibiotherapy and corticosteroids. Infection of previously healthy subjects is very rare. Several species have been described as pathogens: *A. fumigatus* is the most common cause of clinical aspergillosis, followed by *A. flavus* and *A. niger* and rarely *A. terreus*. Invasive aspergillosis can occur in most organs, with the pulmonary system being the main target of this opportunistic infection. Among extrapulmonary locations, the CNS is involved in about 50 % of patients with disseminated aspergillosis. Neuroaspergillosis may occur in various forms, particularly brain abscesses, meningitis, meningoencephalitis, ventriculitis, or granuloma (aspergilloma or fungus ball), with or without concomitant paranasal sinusitis.

Clinically, CNS aspergillosis may present as an acute necrotizing infection or a chronic granulomatous process. Orbital infection may lead to proptosis, altered vision, ophthalmoplegia, and orbital apex syndrome. Vascular complications are more rare (mycotic aneurysms, hemorrhagic and cerebral infarcts). The appearance of CNS aspergillosis on CT scan or MRI is nonspecific.

The diagnosis of this fungal infection is difficult and is often made by direct examination and culture of the lesion (see Fig. 2.26) (stereotactic tissue biopsy). Histopathologic examination shows granulomatous inflammation with septated hyphae in the form of acute-angle and dichotomous branching at regular intervals (Fig. 29.1). Blood and CSF cultures are rarely positive. Detection of galactomannan or beta-glucan antigen in serum or CSF may be helpful, but the sensitivity is low.

Antifungal treatment is based on voriconazole, but itraconazole, posaconazole, and amphotericin B may be used as second-line therapy. When possible, radical surgical debridement of brain abscess should be considered. Indeed, in cases with space-occupying lesions, a neurosurgical procedure is associated with improved survival. Other surgical indications include brain biopsy, ventricular CSF shunting, and mycotic aneurysm.

The prognosis for patients with cerebral aspergillosis is poor, with a survival rate less than 5%. The most important predictive factor for a poor outcome in neuroaspergillosis is a delay in diagnosis and initiation of antifungal therapy.



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_29

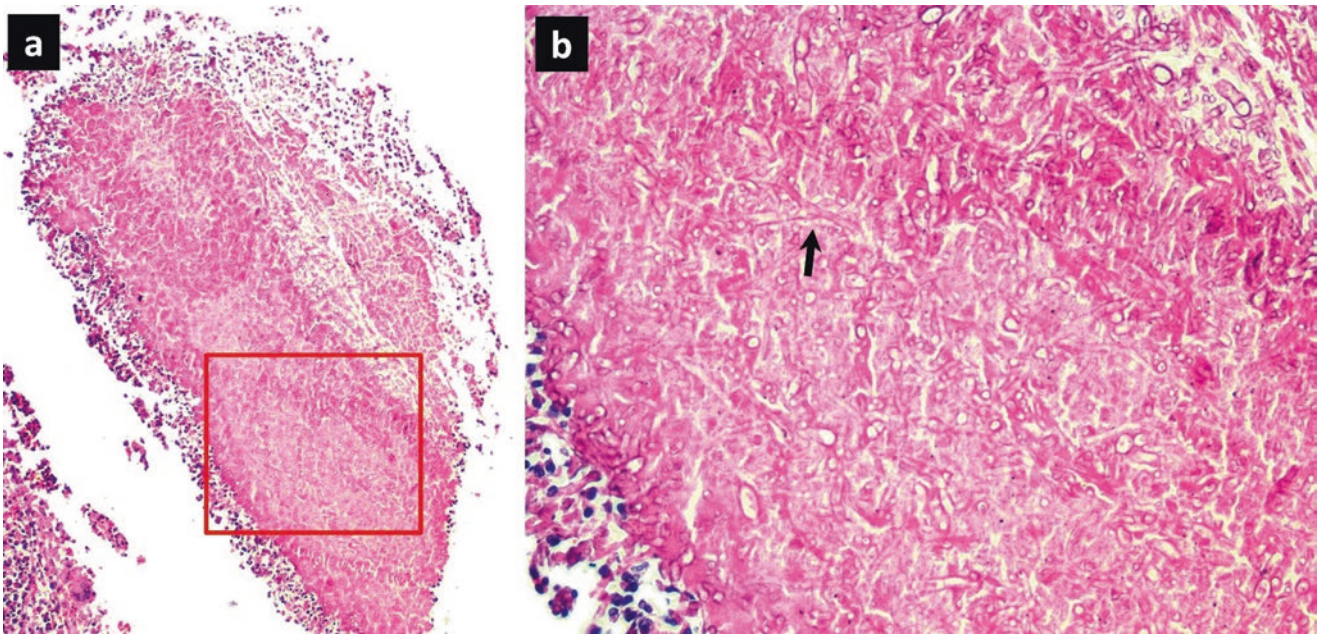


**Fig. 29.1** Photomicrograph of a specimen at low magnification showing necrotic debris (**a**). Photomicrograph at high-power magnification showing septation and dichotomous branching at approximately 45° angles (*arrow*), consistent with *Aspergillus* species (**b**) (hematoxylin–eosin staining)

## Blastomycosis

Blastomycosis, also known as Gilchrist's disease, is a fungal infection caused by the dimorphic microfungus *Blastomyces dermatitidis.* This microorganism is endemic in parts of North America and Africa. The lung and the skin are the most frequently involved organs, followed by the genitourinary tract and the skeleton. CNS infections are unusual (in less than 5% of patients with disseminated blastomycosis) and may occur following hematogenous spread or through direct extension of cranial or spinal infections. CNS blastomycosis may manifest as intracerebral granuloma (blastomycoma) or chronic meningitis with or without hydrocephalus. Direct extension can lead to the formation of a cranial or spinal epidural abscess with adjacent mass effect.

Blastomycomas may be asymptomatic or cause signs and symptoms related to their location in the CNS. On CT scan and MRI, brain blastomycomas appear as solitary or multiple granulomas, which enhance homogeneously or in a ringlike form following contrast administration. These granulomatous lesions are also surrounded by varying degrees of edema. The diagnosis of neuroblastomycosis can be difficult to obtain without a pulmonary infection or obvious localizations. In chronic meningitis, the pathogen may be easier to identify microscopically or following CSF culture.

Amphotericin B is the primary treatment for CNS blastomycosis because azole agents poorly traverse the blood–brain barrier. In the absence of CNS involvement, however, itraconazole and ketoconazole may be administered for systemic blastomycosis. The most common indications for neurosurgery in blastomycosis are to obtain material for tissue diagnosis, CSF shunting in symptomatic hydrocephalus, drainage of abscess formations, and resection of intraparenchymal granulomas causing mass effect. When osteomyelitis occurs, cranial and spinal debridement may be performed.

Overall, the prognosis is relatively favorable in immunocompetent patients, but the mortality rate can reach 50% among immunocompromised subjects.

## Candidiasis

Candidiasis is known as the most common fungal infections of the CNS, but it is rarely diagnosed before autopsy. *Candida* microorganisms are commensal in humans, habitually found in normal flora of the mouth, the skin, and the gastrointestinal and genitourinary tracts. About 50% of patients with candidemia have CNS involvement, but the mortality rate of these patients is very high (80–95%). *Candida albicans* is the most common species in the CNS. Other species include *C. glabrata*, *C. tropicalis*, *C. parapsilosis*, and *C. krusei*. In addition to CNS involvement secondary to hematogenous spread, neurocandidiasis can also occur exogenously, following surgery, trauma, or the use of shunts and catheters. Both the brain parenchyma and the meninges may be involved. Parenchymal presentations consist of multiple microabscesses and granulomas, which

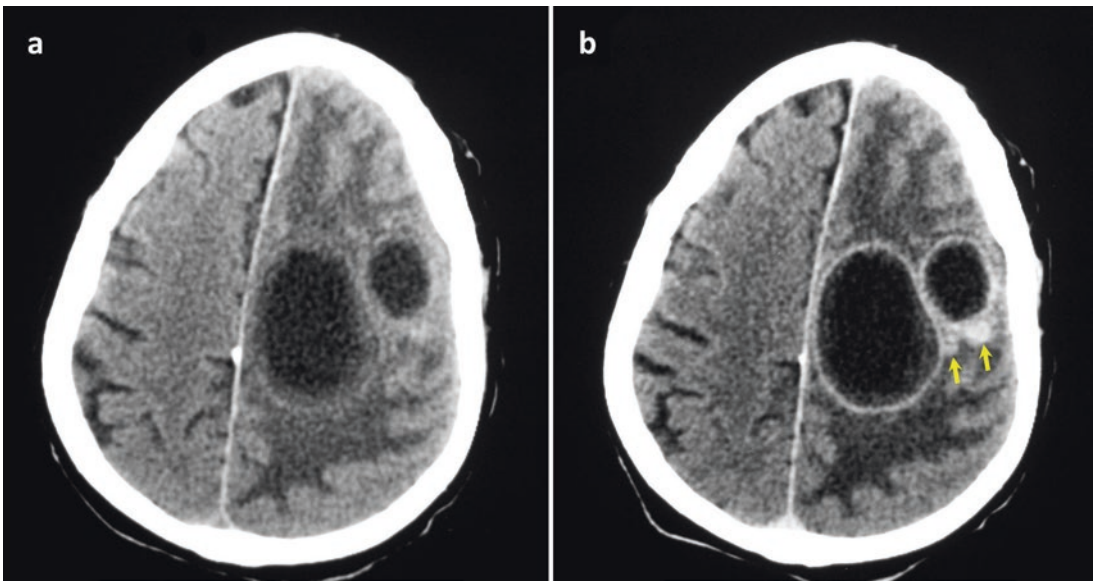


**Fig. 29.2** Case 29.1. Axial cranial CT scan before (**a**) and after (**b**) contrast administration, showing two frontal brain abscesses in the left side with ring enhancement and extensive perifocal edema. Note the adjacent granulomatous lesions (*arrows*)

are usually subcortical or in the basal ganglia and the posterior fossa (Figs. 29.2 and 29.3). Chronic meningitis, encephalitis, mycotic aneurysms, subdural empyemas, solitary or multiple abscesses, and intraventricular fungal balls can also be found (Figs. 29.4 and 29.5).

Signs and symptoms of parenchymal lesions are similar to those of a progressive encephalopathy, with rare focal neurologic deficits. When the meninges are involved, the clinical picture resembles bacterial meningitis, with fever, headache, photophobia, vomiting, and stiff neck. Ischemic or hemorrhagic strokes are rare but may be seen when vascular involvements occur.

Neuroimaging findings are variable and nonspecific, but they can assist in the diagnosis through visualization of lesions. Microabscesses are often too small to detect on MRI, with or without enhancement after gadolinium administration, but some lesions with hypersignal on T2-weighted images or on proton density can be suggestive. CSF examination can show signs of inflammation in chronic meningitis, with mild pleocytosis, high protein levels, and low glucose levels. Pathogens may be identified in CSF or after culture (see Figs. 2.27 and 2.28). Blood culture can be positive in 40–50% of patients with disseminated infection. In the absence of *Candida* identification, specific mannan antigen detection in the CSF and a polymerase chain reaction (PCR)-based test may be helpful. The diagnosis is often more delayed in cases with parenchymal lesions.

The standard treatment for CNS candidiasis includes administration of amphotericin B for several weeks, followed by fluconazole. Elimination of all infected devices is mandatory. Patients may require surgical support for management of high intracranial pressure, excision or drainage of abscesses, CSF shunting, and mycotic aneurysm. During antifungal treatment, CSF and neuroimaging examinations should be performed to follow the results of therapy.

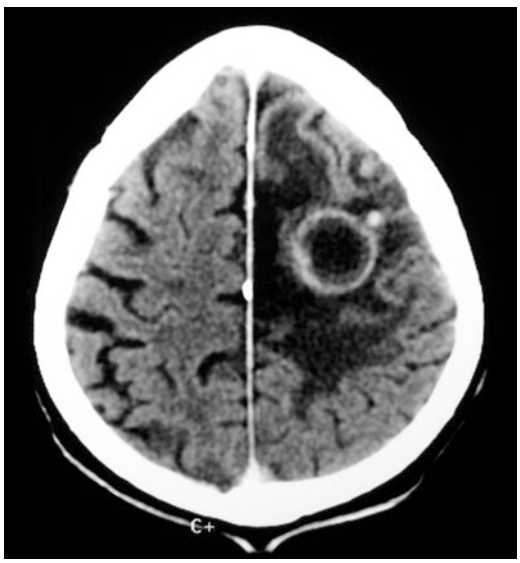


**Fig. 29.3** Case 29.1. Enhanced CT scan following stereotactic brain abscess aspiration. *Candida albicans* was identified on microbiologic study. Unfortunately, this elderly patient died from a systemic candidiasis

The mortality rate is about 80% in untreated or inadequately treated patients. The prognosis remains poor for patients with intraparenchymal candidiasis, but a good survival rate may be encountered in cases with meningitis. Despite some favorable results, neurologic sequelae such as cerebral palsy, psychomotor retardation, hydrocephalus, and blindness are common, especially in children.

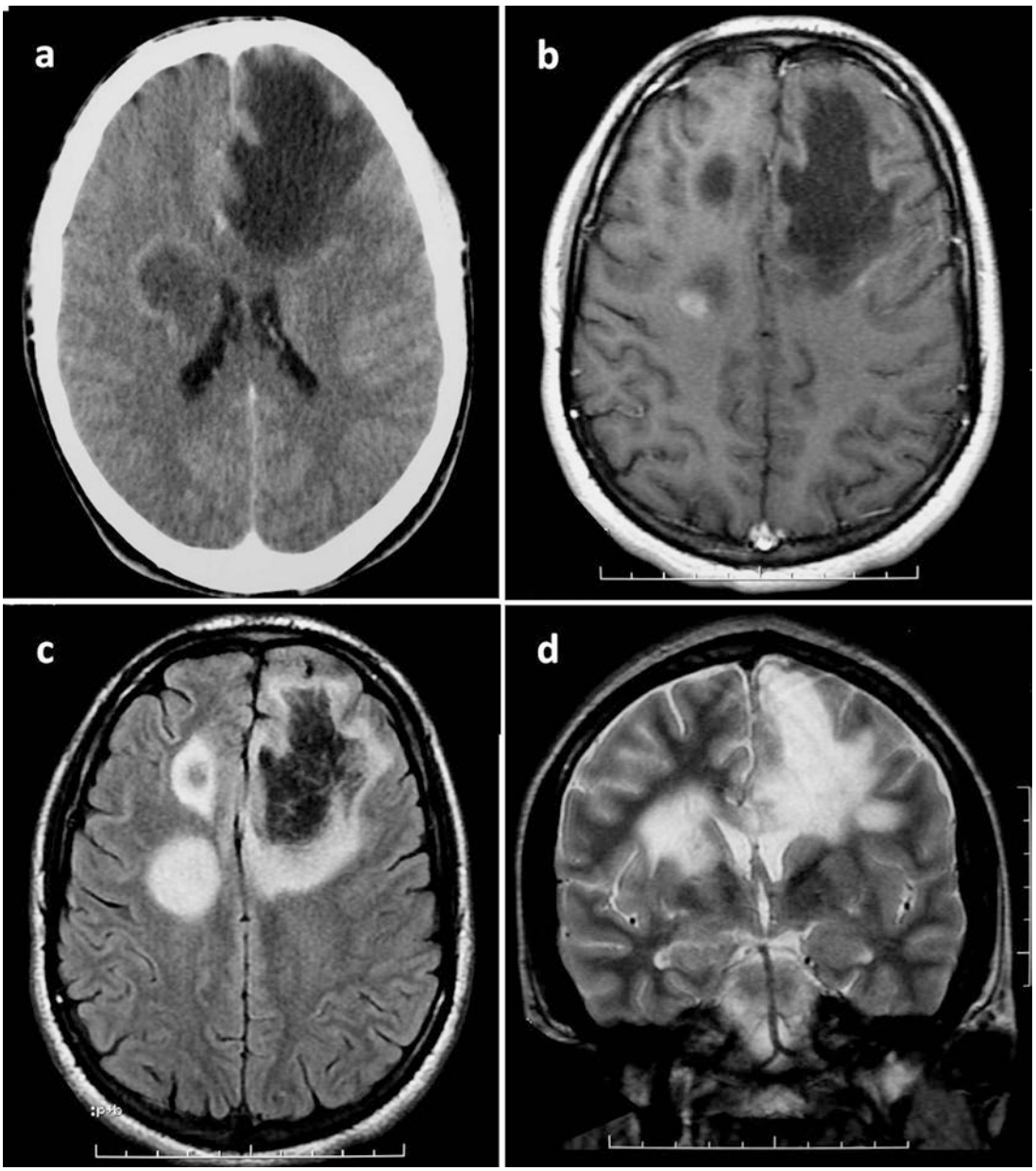


**Fig. 29.4** Case 29.2. Acute disseminated fungal encephalitis due to *Candida albicans.* Axial cranial post-contrast CT scan (**a**), enhanced T1-weighted image (**b**), fluid-attenuated inversion recovery (FLAIR) sequence (**c**), and coronal T2-weighted image (**d**)

## Cryptococcosis

Cryptococcosis is an infection caused by the encapsulated yeasts *Cryptococcus neoformans* or *Cryptococcus gattii.* These fungi are found in soil and often are associated with avian droppings or eucalyptus trees. The portal of entry of *Cryptococcus* species is the respiratory tract; it colonizes the lung, from where it disseminates systemically and sometimes to the CNS. Although most patients are immunocompromised hosts (especially HIV patients, organ transplant recipients, and those with immunosuppressive therapy or malignancy), healthy and apparently healthy subjects may also be infected. Involvement of the CNS has been reported in 30–50% of patients with systemic infection. Both cerebral parenchyma and meninges may be involved. Meningitis and meningoencephalitis are the most usual manifestations in the CNS, but occasionally a granulomatous lesion with cystic changes (cryptococcoma or mucinous pseudocyst) can develop.

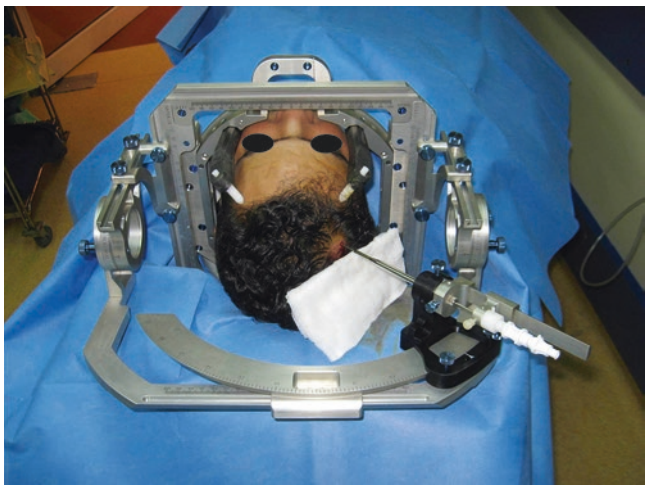

**Fig. 29.5** Case 29.2. Diagnosis of candidiasis was confirmed histopathologically following CT-guided stereotactic biopsy (right frontal lesion with peripheral enhancement) under local anesthesia, using a Radionics™ CRW™ (Cosman–Roberts–Wells) frame

Cryptococcal meningitis generally presents as severe, subacute, or chronic headache with or without fever. Vomiting, blurred vision, and confusion may also be associated. When meningoencephalitis develops, seizures, focal neurologic deficits, and consciousness disorders are added to the meningitis syndrome. Clinical pictures of intraparenchymal cryptococcomas are similar to those of any intracranial mass.

Neuroimaging data are not specific for cryptococcosis, and CT scan or MRI findings are similar to those of neurotuberculosis, such as post-contrast enhancement of the meninges, hydrocephalus, and ring-shaped mass lesions with surrounding edema (Figs. 29.6 and 29.7). Typically, most mucinous pseudocysts develop in the basal ganglia. Unspecific meningeal inflammation is found in CSF examination. Detection of cryptococcal antigen in blood and CSF may be helpful, but the diagnosis of cryptococcosis is mainly made by the identification of *Cryptococcus* organisms from pathologic sections and culture of specimens obtained through CSF or surgical biopsy (see Fig. 2.29). Blood culture may be conclusive in patients with disseminated cryptococcosis. Mucicarmine stains can help differentiate *Cryptococcus* species from other fungal infections.

The first-line therapy for neurocryptococcosis is intravenous amphotericin B and oral flucytosine. For severe cases, this first-line therapy must be followed by oral fluconazole (or perhaps itraconazole if fluconazole is not tolerated). As with other CNS fungal infections, surgery may help in obtaining material for tissue diagnosis, performing CSF shunting for symptomatic hydrocephalus (despite a potential risk of cryptococcal peritonitis), draining of abscess formations, and resection of intraparenchymal granulomas causing mass effect.

The most important prognostic factor remains the severity of the underlying disease. In treated patients, the mortality rate is generally between 30% and 60%. If untreated, CNS cryptococcosis is inescapably fatal. Neurologic sequelae including visual loss, cranial nerve palsies, neurologic deficit, or mental impairment occur in 40–50% of all treated patients, and 20–25% experience a relapsing course.

## Histoplasmosis

Histoplasmosis is caused by the pathogen *Histoplasma capsulatum*, a fungal infection endemic in North America and particularly in river valleys. This microorganism is commonly found in soil contaminated by bird or bat excretions. Both immunocompetent and immunocompromised patients can be infected. Histoplasmosis is usually limited to the lungs, but the CNS is involved in about 20% of patients with disseminated infection. Chronic meningitis is the most frequent form of CNS infection, often complicated with communicating hydrocephalus. Acute meningitis, encephalitis, multiple mass lesions of the brain or spinal cord, brain abscesses, and ischemic stroke are unusual presentations of this disease.

CNS involvement is characterized by signs and symptoms similar to those of other chronic fungal meningitis, such as headache, fever, lethargy, and altered mental status. Basilar meningitis may be associated with cranial nerve palsies, seizures, and raised intracranial pressure due to hydrocephalus. The development of granulomatous lesions (histoplasmomas) can lead to focal neurologic manifestations that depend on the location and size.

CT scan and MRI findings are unspecific. Most histoplasmomas appear as "granulomatous lesions" with a variable degree of surrounding edema and enhancement after contrast injection. Confirmation of CNS involvement is difficult even with fungal culture and histopathologic examinations of involved tissue. Detection of anti-*Histoplasma* antibody in CSF and blood, *Histoplasma* antigen in urine and blood, as well as *Histoplasma* DNA by PCR may be helpful.

Neurohistoplasmosis requires prolonged and aggressive therapy, as it has a high risk of relapse (up to 50% of treated patients). Generally, treatment is based on amphotericin B, followed with itraconazole for at least 1 year and until CSF anomalies and *Histoplasma* antigen levels are normalized. Usually, surgical intervention consists of resection of solitary histoplasmomas with parenchymal mass effect or CSF shunting for symptomatic hydrocephalus.

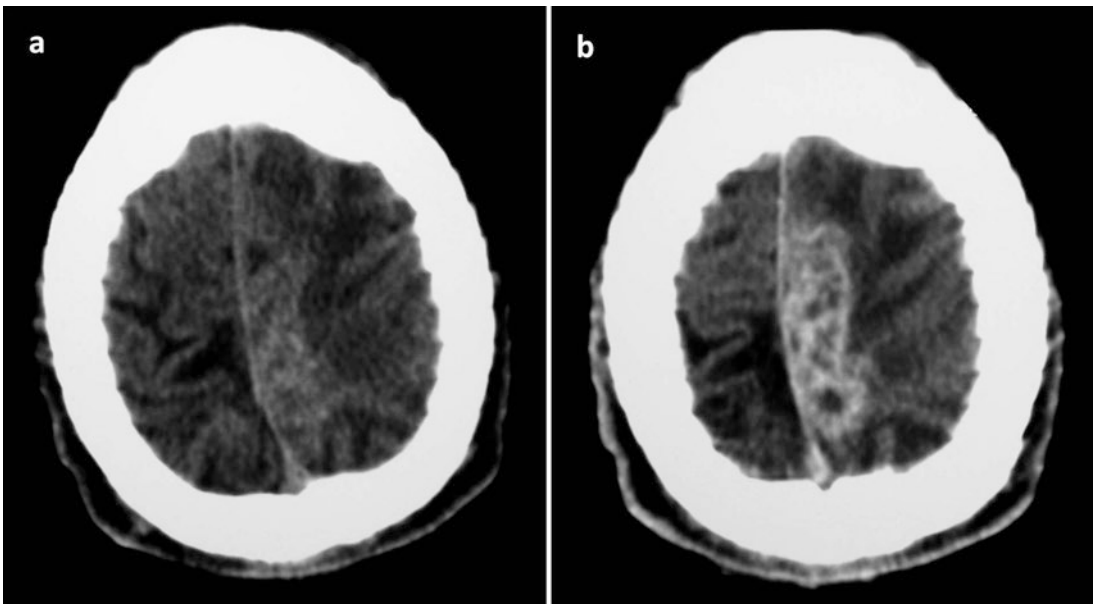


**Fig. 29.6** Case 29.3. Cranial posttraumatic parafalcine fungal granulomas located in the left frontoparietal region. The diagnosis of cryptococcosis was suspected. Axial CT scan without (**a**) and with (**b**) contrast injection

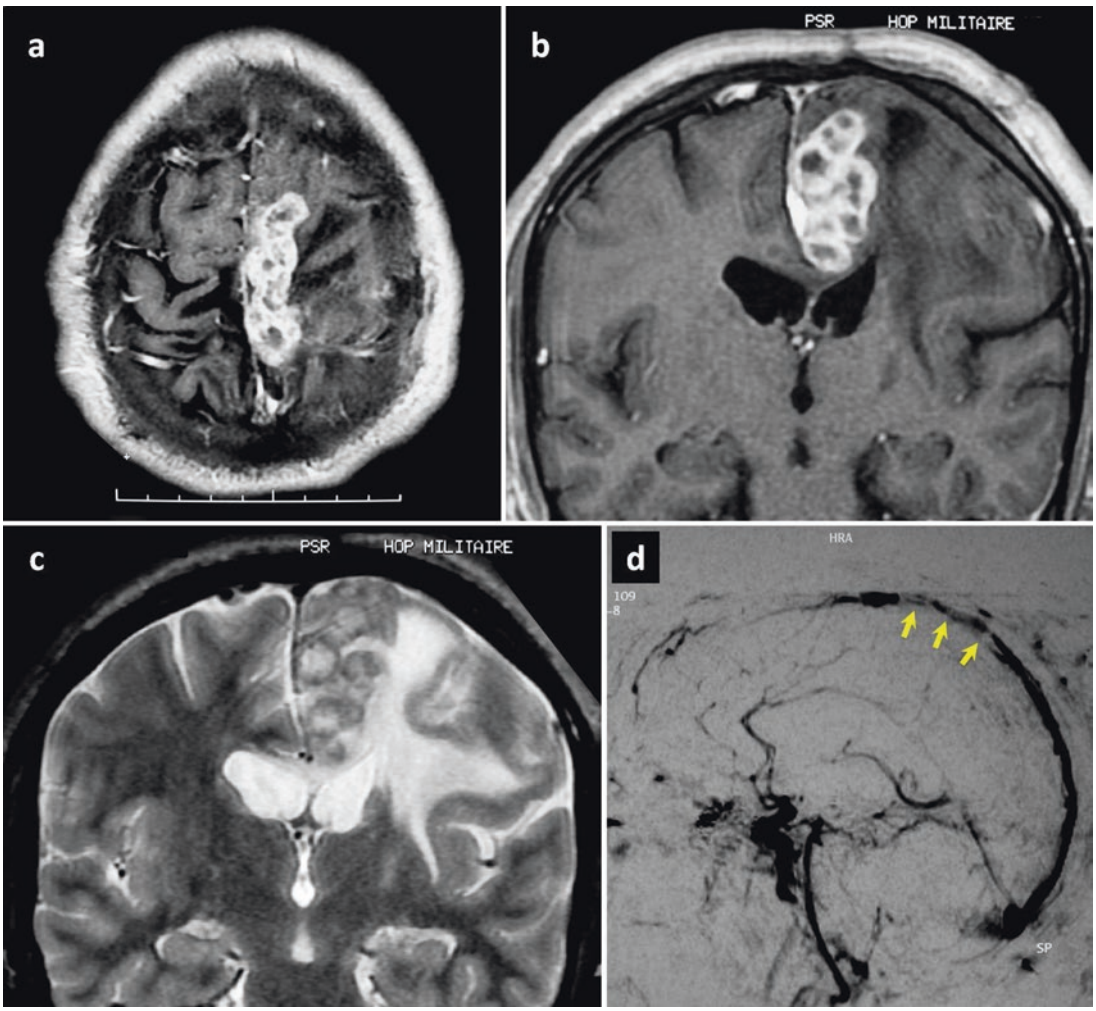


**Fig. 29.7** Case 29.3. Axial (**a**) and coronal (**b**) enhanced T1-weighted MR images, coronal T2-weighted image (**c**), and contrast-enhanced 3D MR venography (lateral view) (**d**) showing the interhemispheric fungal granulomatous lesions with perifocal edema. Note thrombosis of the adjacent superior sagittal sinus (*arrows*) (suspicion of neurocryptococcosis)

## Mucormycosis (Zygomycosis)

Mucormycosis or zygomycosis is an opportunistic, fulminant fungal infection that principally infects immunocompromised subjects. This rapidly fatal disease is increasingly emerging and is considered the third most common opportunistic fungal infection in immunocompromised and diabetic patients. *Rhizopus* species are the most common causative pathogens, followed by *Mucor* and *Absidia* from the order of *Mucorales*. The infection usually involves the lungs, gastrointestinal tract, and skin. Among all the types of mucormycosis, infection in the rhinocerebral area is the rarest but also the most fatal opportunistic infection. Brain involvement typically occurs from direct extension of a local infection of the face (especially paranasal sinuses and orbits) or nasopharynx. The fungus invades the arteries, leading to thrombosis that subsequently causes necrosis of soft tissues and bones of the skull base.

The usual symptoms of CNS mucormycosis are headache, nausea, fever, facial or periorbital swelling, proptosis, altered mental status, meningitis, visual impairment, multiple cranial nerve palsies, focal neurologic deficits, and seizures. In the advanced stage, with important skull base invasion, patients may develop visual loss, diabetes insipidus, bifrontal abscesses, cavernous sinus thrombosis, carotid artery occlusion, and (more rarely) mycotic aneurysm.

Neuroimaging findings are nonspecific and correlate with the invasion of the infection. CT scan and MRI can show paranasal sinusitis with mucosal thickening and facial and skull base erosion. Severe infection may involve the brain and orbits. Mucormycosis should always be considered in a patient with diabetic ketoacidosis and signs of facial, orbital, or nasopharyngeal infection. Blood and CSF cultures are negative. Serologic tests and molecular studies are not diagnostically useful. Diagnosis is usually confirmed by histopathologic examination or by culture of tissue invasion. The pathognomonic feature is the presence of invasive, nonseptate mycelia that are larger than other filamentous fungi, with the hyphae exhibiting right-angle and disorganized branching (see Fig. 2.30).

Early and aggressive surgical removal of all infected and devitalized tissue is considered the best approach, to avoid the extension of infection. Amphotericin B is the antifungal drug of choice. Alternatively, posaconazole can be considered. Hyperbaric oxygen therapy can also be given as an adjuvant.

CNS mucormycosis has a poor prognosis due to the underlying disease, the extent of local infection, delay in diagnosis and treatment, and incomplete surgical debridement. The mortality rate is about 60–80% for patients with rhinocerebral involvement. Mortality among diabetic patients seems to be lower than among those with other causes of immunosuppression. Survivors can have important postoperative facial deformities and permanent blindness.

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# Central Nervous System Infections in HIV Patients

# 30

Acquired immune deficiency syndrome (AIDS) occurs from human immunodeficiency virus (HIV) infection. Despite the accessibility of highly active antiretroviral therapy (HAART) since 1995 and the use of prophylactic antimicrobial regimens, opportunistic infections continue to have a devastating effect on HIV-infected patients, especially those diagnosed late or inadequately treated. About 1–10 per 1,000 patient–years has CNS opportunistic infections, and less than 5% of patients with AIDS have a normal brain on autopsy. Opportunistic microorganisms are less virulent under normal conditions (immunocompetent hosts), but individuals with immune dysfunction are more susceptible to mycobacterial, viral, fungal, and some parasitic infections. Neuroinfections can occur at any stage and may even precede the diagnosis of HIV infection. Clinically, CNS infections reflect intraparenchymal and leptomeningeal involvement, but clinical signs and symptoms may be blurred owing to alterations of host responses to pathogens, so it is important to preserve a broad perspective in the HIV population. Treatment of these neuroinfections is mainly medical, but neurosurgical consultation is often requested for diagnostic purposes (surgical biopsy), CSF shunting, and surgical resection of some lesions. Difficult-to-treat microorganisms and polymicrobial infections seen in HIV patients are associated with an increased relapse rate, as are a suboptimal choice of antibiotherapy or insufficient duration of treatment. As with other CNS and spinal infectious diseases, early diagnosis and effective management will reduce morbidity and mortality and will improve the patient's overall quality of life.

This chapter focuses on the CNS infections most frequently associated with HIV disease:

- Progressive multifocal leukoencephalopathy
- Toxoplasmosis
- Cryptococcosis
- Cytomegalovirus infection
- Tuberculosis

## Progressive Multifocal Leukoencephalopathy

Progressive multifocal leukoencephalopathy (PML) is a demyelinating disease of the CNS caused by the reactivation of the John Cunningham virus (JCV). Primary JCV infection is normally asymptomatic, and the virus remains latent in the kidneys, bone marrow, and lymphoid tissue in children and young adults. Presently the incidence of PML is estimated to be 1.3 cases per 1,000 person–years in the HIV population. In the brain, JCV infects mainly oligodendrocytes and astrocytes and sometimes cerebellar granular cells and cortical pyramidal neurons. Lesions are multifocal in the white matter. Any part of the CNS can be affected, particularly the frontal, parietal, and occipital lobes and the cerebellar peduncles. The spinal cord is rarely concerned. Most HIV patients have a CD4 (cluster of differentiation 4) cell count higher than 200 cells/mm$^3$.

The clinical course is usually insidious and progressive over weeks to months. Although the lesions are multifocal, often one lesion predominates clinically. Common signs and symptoms include headache, visual abnormalities, motor weakness, hemiparesis, hemisensory deficits, ataxia, and (less frequently) dementia and seizures. Meningitis syndrome is rare.

PML lesions are manifested on CT scans) as symmetric or asymmetric, multifocal areas of white matter demyelination without edema, enhancement, or mass effect. On MRI, the lesions are hypointense on T1-weighted images and hyperintense on both T2-weighted images and fluid-attenuated inversion recovery (FLAIR) sequences (Figs. 30.1 and 30.2). MRI findings are highly suggestive in the appropriate clinical setting. Routine CSF studies are unspecific. The neuropathology of PML includes a combination of demyelination, giant astrocytes with pleomorphic hyperchromatic nuclei, and altered oligodendrocytes with eosinophilic intranuclear inclusion bodies. The definitive diagnosis



A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4_30

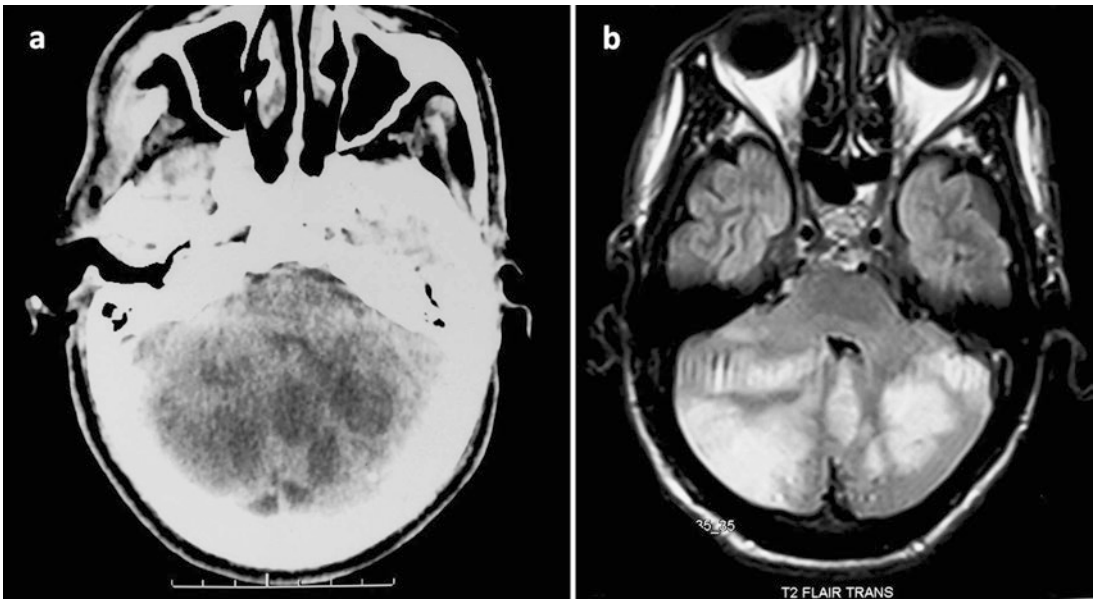


**Fig. 30.1** Case 30.1. Cerebellar form of progressive multifocal leukoencephalopathy. Axial cranial CT scan with contrast injection (**a**) and fluid-attenuated inversion recovery (FLAIR) sequence (**b**) revealing asymmetric focal zones of low density (**a**) and hyperintensity (**b**) involving the white matter of both cerebellar hemispheres, with fourth ventricle mass effect

is made by detection of JCV in CSF or brain biopsy by polymerase chain reaction (PCR).

There is no effective drug therapy against JCV infections. The basic treatment for PML is immune reconstitution with combination antiretroviral therapy. This approach has significantly decreased the incidence and improved the prognosis of PML. Corticosteroids may have some positive effects on brain inflammation. The survival rate is estimated to be 50–75%. Restoration of immune function as evaluated by an increase in the CD4 count plays an important role in the clearance of JCV from the CSF.

## Toxoplasmosis

Toxoplasmosis is discussed in Chap. 28. In this chapter, Figs. 30.3, 30.4, 30.5, 30.6, 30.7, 30.8, 30.9, 30.10, 30.11, 30.12, and 30.13 illustrate cases of toxoplasmosis-related granulomatous lesions and encephalitis in HIV patients.

## Cryptococcosis

Cryptococcosis is discussed in Chap. 29.

## Cytomegalovirus Infection

Cytomegalovirus (CMV) is a DNA virus belonging to the herpesvirus family. CMV is endemic worldwide and generally causes asymptomatic or clinically benign infections. Most individuals have been infected prior to reaching adulthood. CMV infection is one of the most often reported opportunistic infections in patients with HIV infection, with about five cases per 100 person–years. Generally, CMV infection of the nervous system presents in individuals with CD4 counts below 50/mm$^3$, CMV viremia, and other systemic sites. In the CNS, the disease often manifests as meningoencephalitis, polyradiculomyelitis, or both.

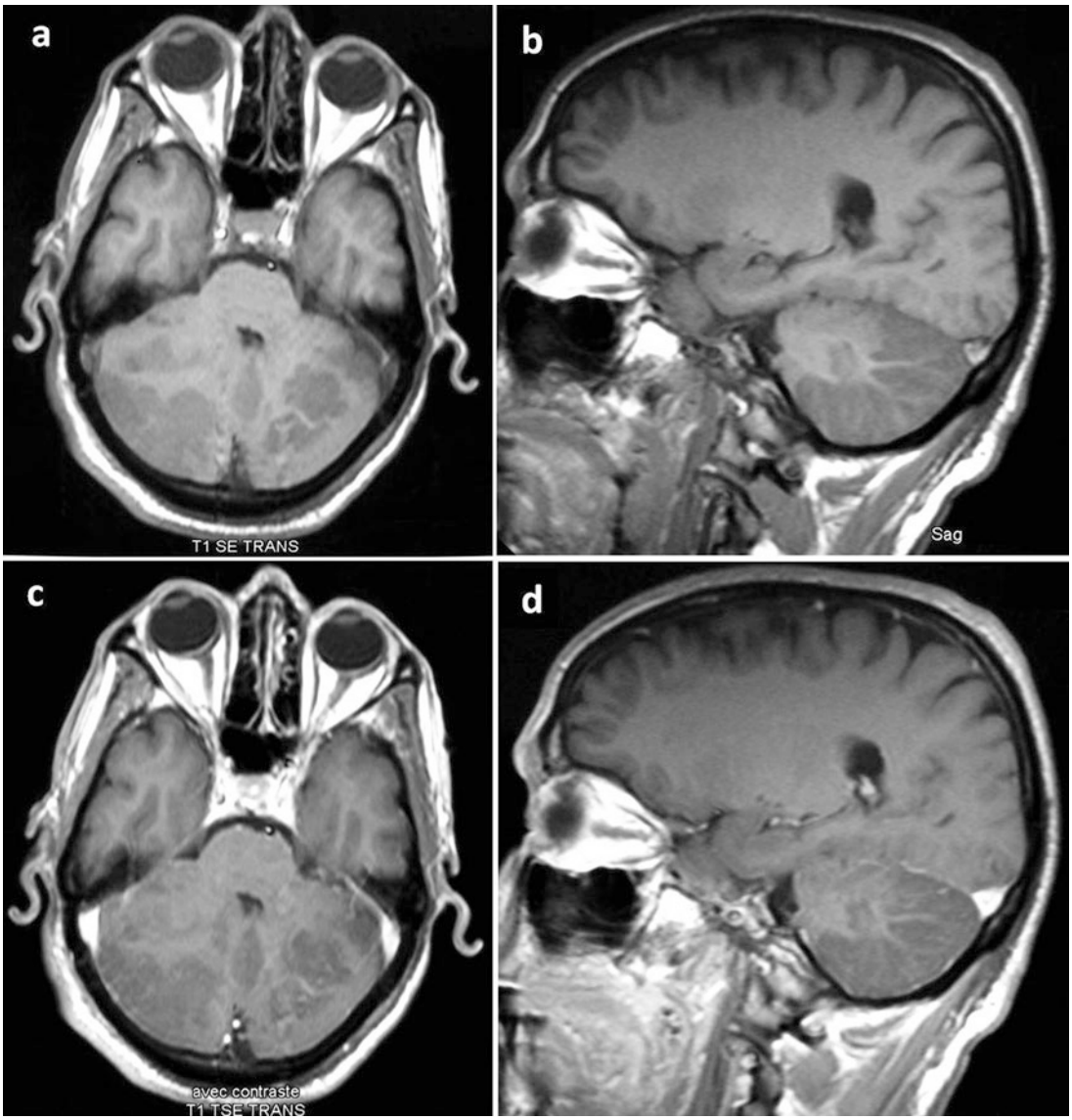


**Fig. 30.2** Case 30.1. Axial (**a**) and sagittal (**b**) nonenhanced T1-weighted images and images following gadolinium administration (**c**, **d**). The lesions are hypointense on T1-weighted images, without enhancement after gadolinium injection

Clinically, patients with meningoencephalitis may present with fever, lethargy, and confusion and, more seriously, with cranial nerve deficits, brain stem syndromes, or even coma. Neuroimaging typically shows periventricular and meningeal enhancement (Fig. 30.14). CSF analysis reveals pleocytosis with lymphocytic predominance, low to normal glucose level, and normal to increased protein. Some patients with CMV encephalitis may have concurrent involvement of the adrenal glands (hyponatremia) or retina (floaters, loss of peripheral vision, or central scotoma). The neuropathology of CMV encephalitis is variable, ranging from isolated CMV inclusions without inflammation or necrosis to severe necrotizing encephalitis, but the most common pathological finding is a microglial nodule encephalitis.

On the other hand, the clinical picture of polyradiculomyelitis is similar to that of Guillain–Barré syndrome, with

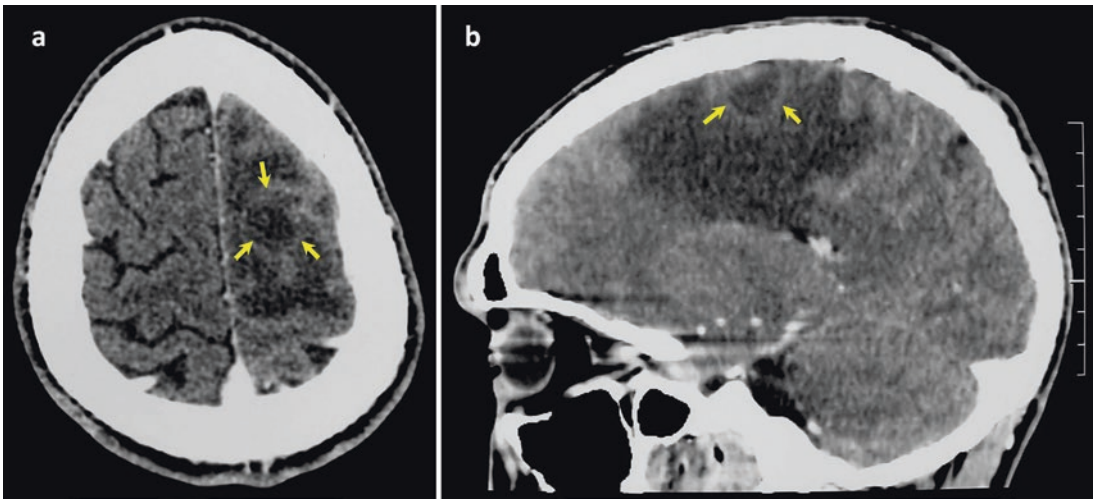


**Fig. 30.3** Case 30.2. *Toxoplasma gondii* brain granuloma in an HIV patient. Axial (**a**) and sagittal (**b**) post-contrast CT scan showing a ring-enhancing lesion (*arrows*) located in the left frontal area

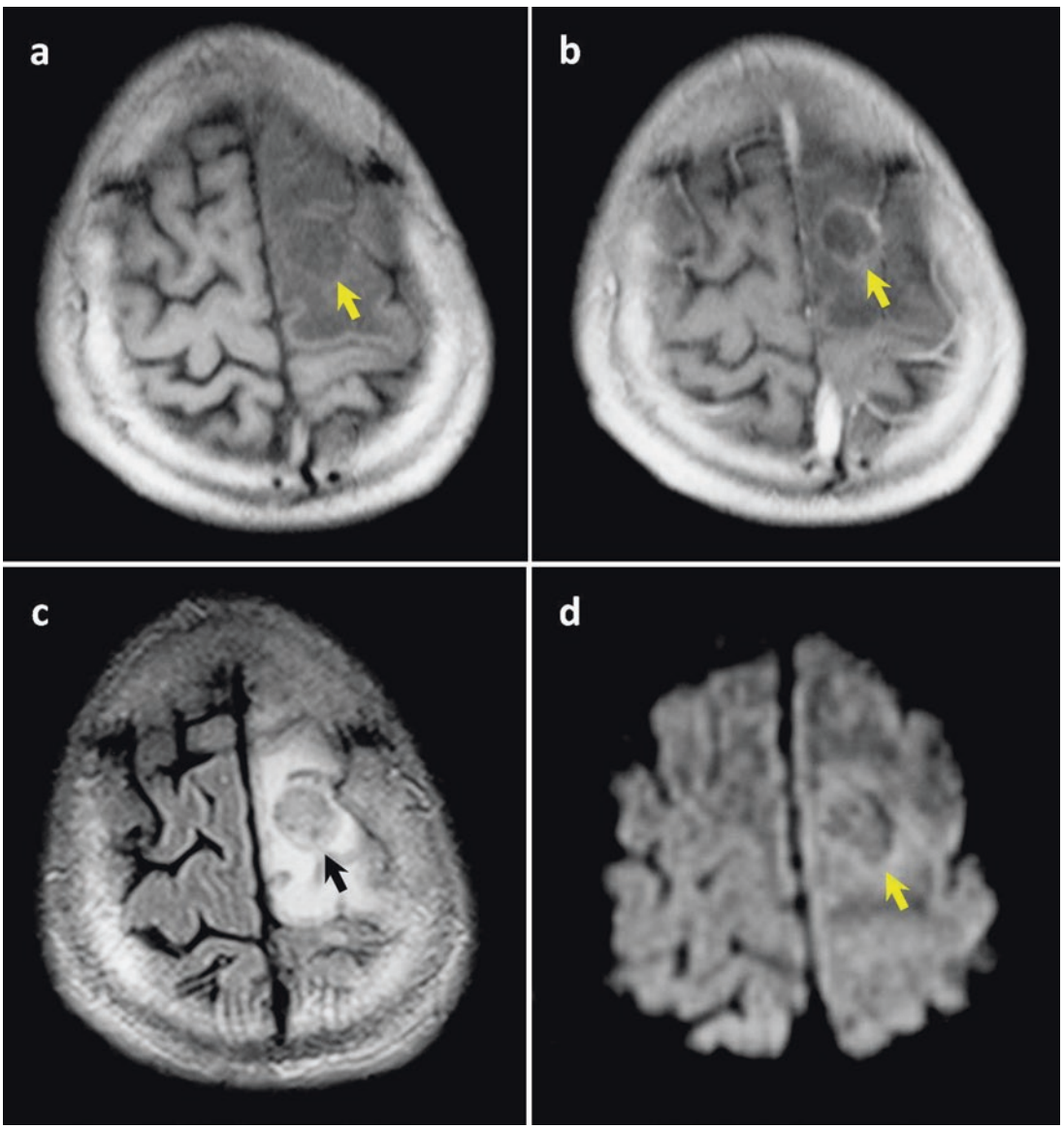


**Fig. 30.4** Case 30.2. Axial nonenhanced (**a**) and enhanced (**b**) T1-weighted images, FLAIR sequence (**c**), and diffusion-weighted image (**d**). This solitary, well-limited granuloma (*arrow*) is surrounded by extensive perifocal edema

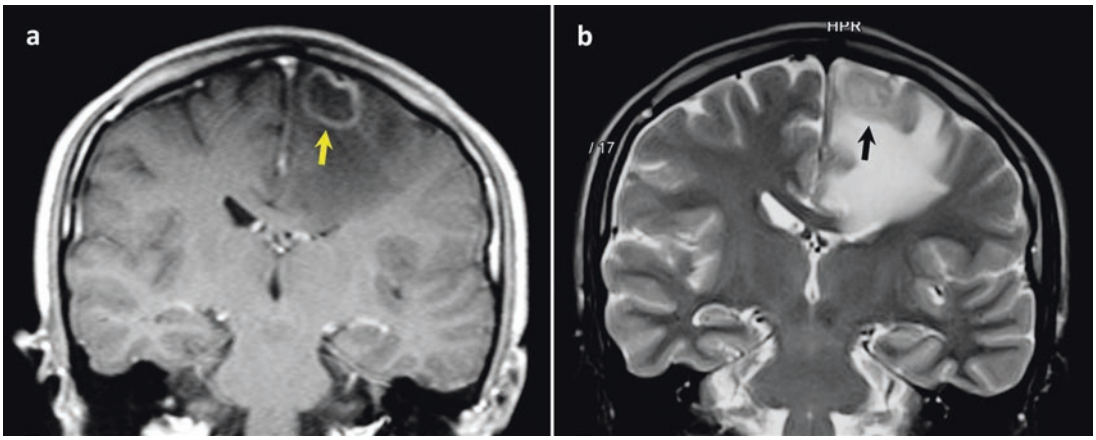


**Fig. 30.5** Case 30.2. Coronal enhanced T1-weighted (**a**) and T2-weighted (**b**) images revealing the left frontal parasagittal granuloma (*arrow*) with associated edema and ventricle mass effect

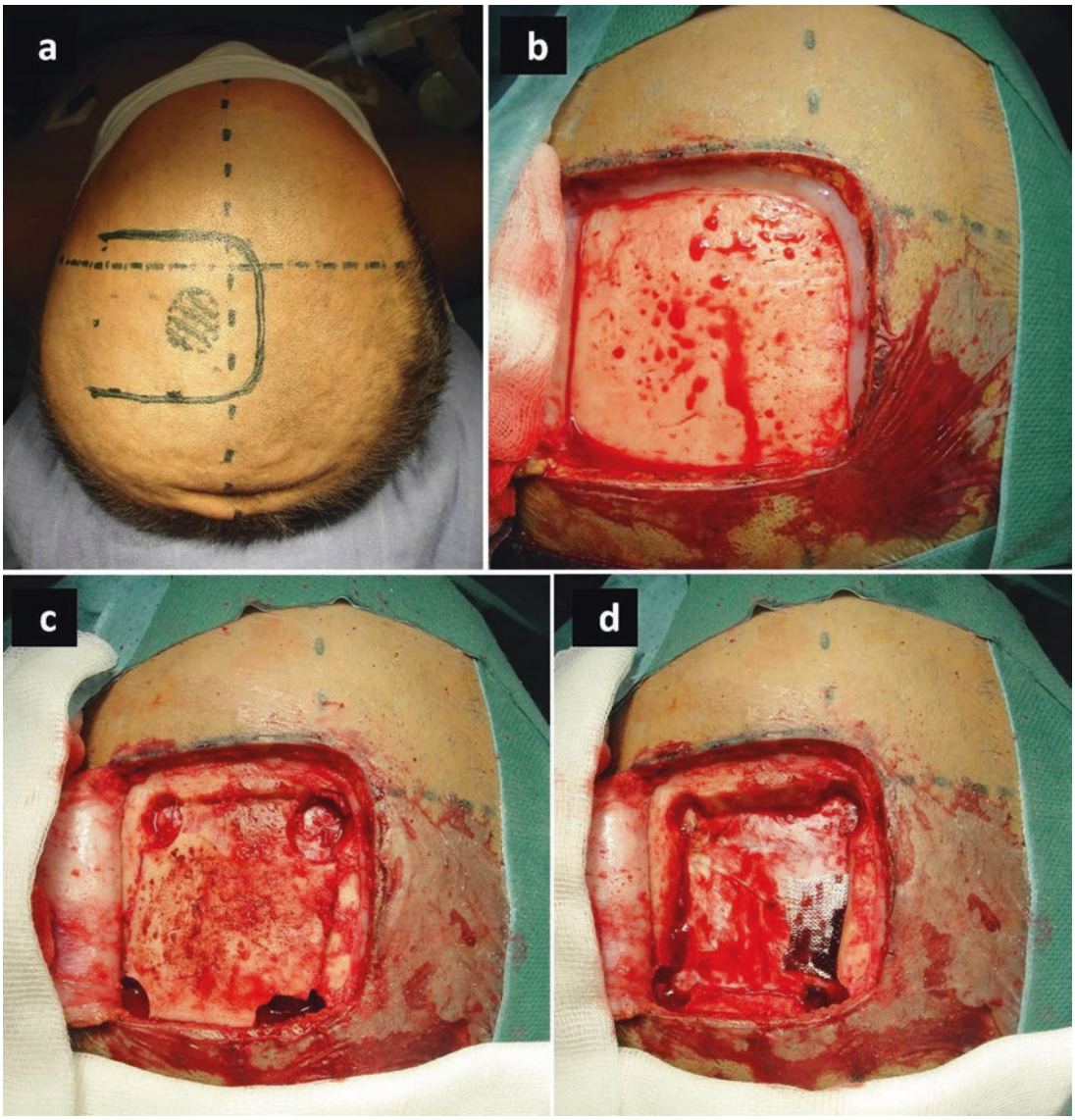


**Fig. 30.6** Case 30.2. The patient was operated on via a retrocoronal, parasagittal approach on the left side (**a**). Intraoperative views step-by-step: the skin flap is reflected (**b**), burr hole craniotomy is performed (**c**), and the free bone flap is removed (**d**)

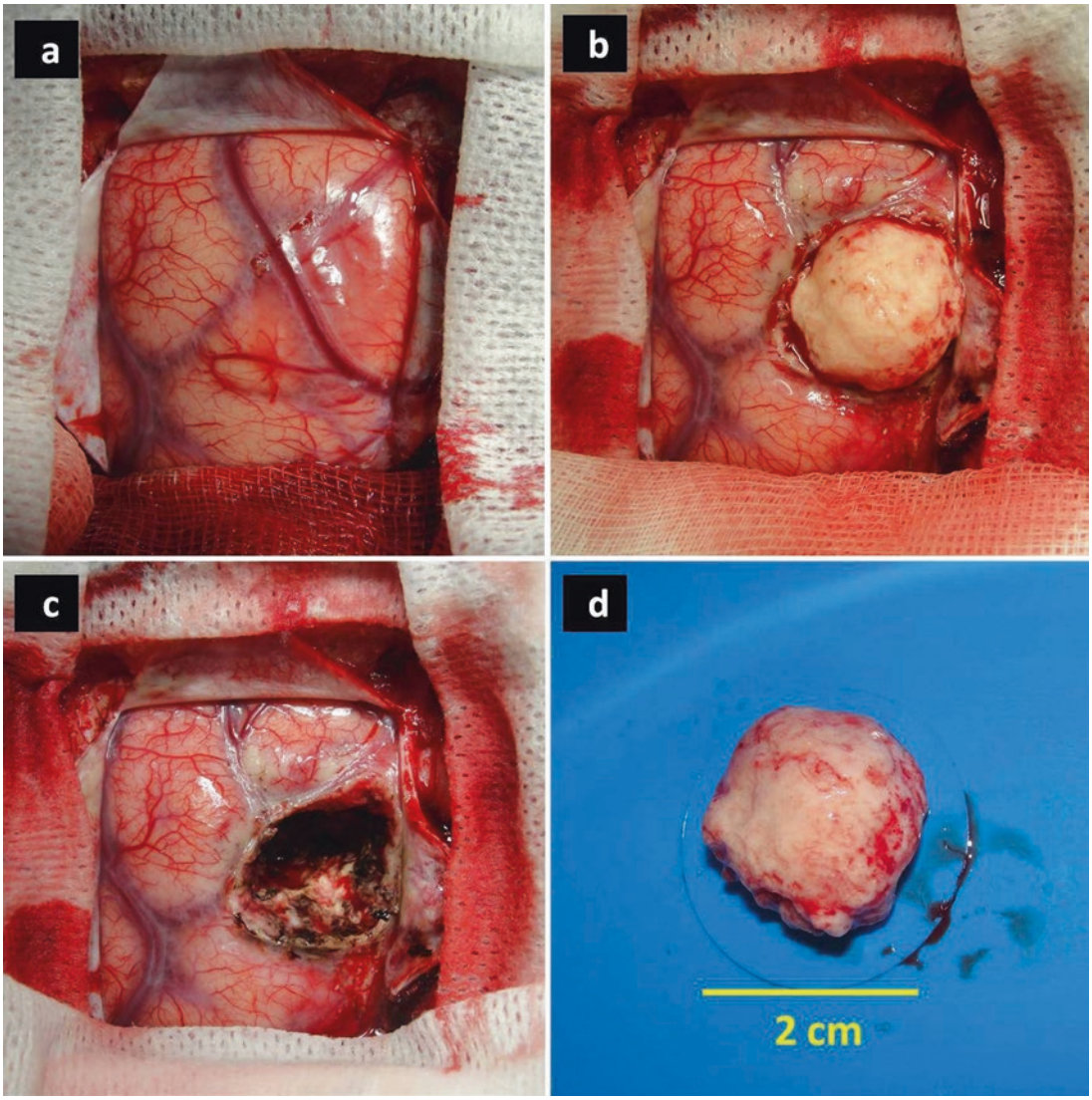


**Fig. 30.7** Case 30.2. Intraoperative views: the dura mater is opened (**a**), and the well-limited granuloma is completely removed from the surrounding brain parenchyma (**b**). Final appearance of the residual cerebral cavity after gentle hemostasis (**c**). Macroscopic appearance of the granuloma (**d**)

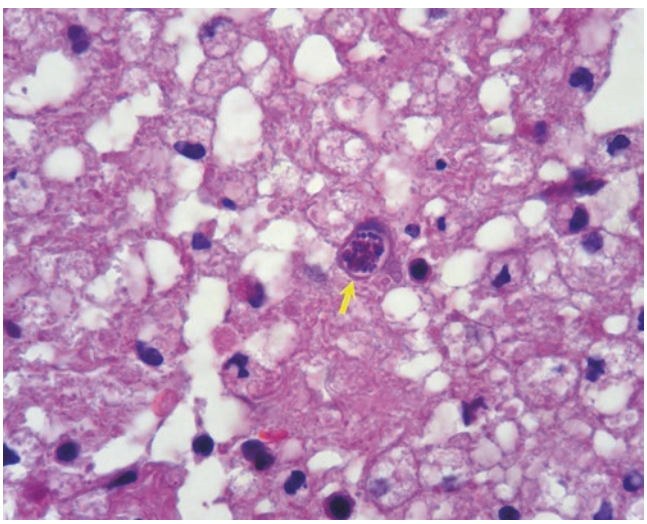

**Fig. 30.8** Case 30.2. Histopathologic features of toxoplasma encephalitis. A toxoplasma cyst (*arrow*) is noted in this necrotic brain granulomatous lesion, with medium-power magnification (hematoxylin–eosin staining)

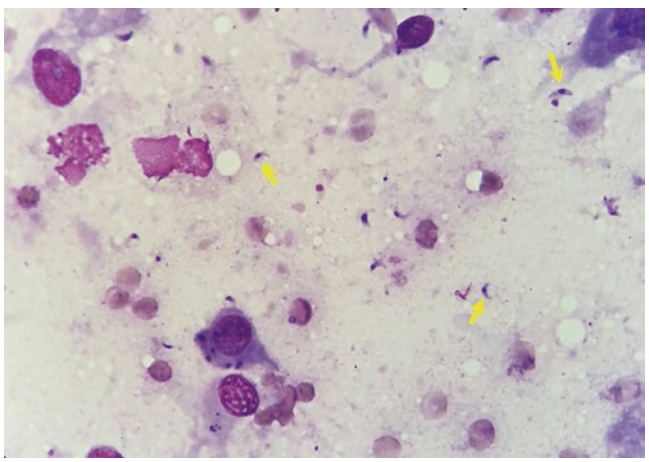

**Fig. 30.9** Case 30.2. *Toxoplasma gondii* tachyzoites (*arrows*), stained with May–Grünwald Giemsa (MGG), and obtained from the brain granuloma. Medium-power magnification (Courtesy of Pr. R. Moutaj, PharmD; Marrakech, Morocco)

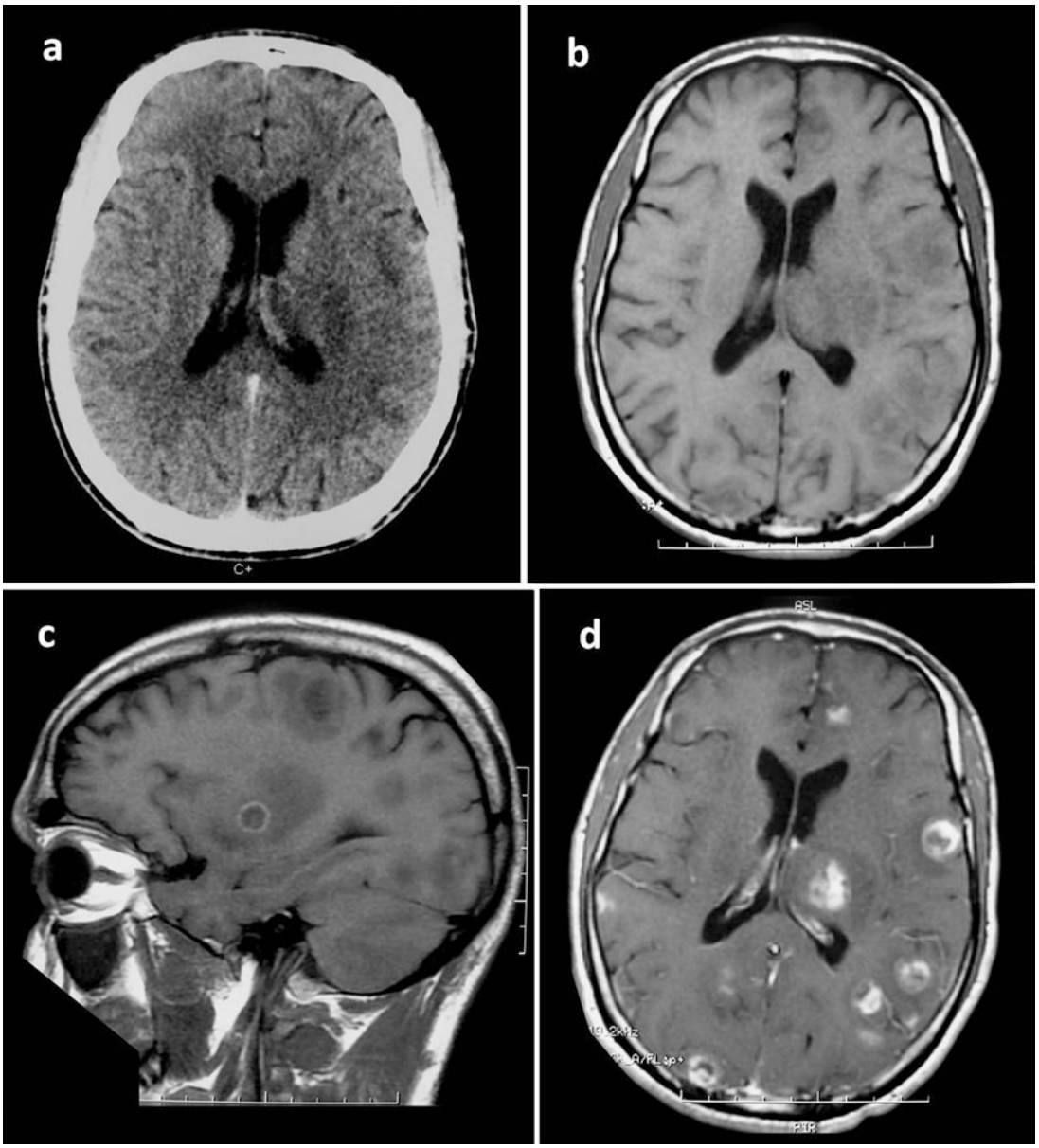


**Fig. 30.10** Case 30.3. Multiple brain granulomas caused by toxoplasmosis in an HIV-infected person. Axial post-contrast CT scan (**a**), axial (**b**) and sagittal (**c**) T1-weighted images without gadolinium injection, axial enhanced T1-weighted image (**d**). The granulomas (nodular and ring-enhancing lesions) are best seen on T1-weighted MRI following gadolinium injection (**d**)

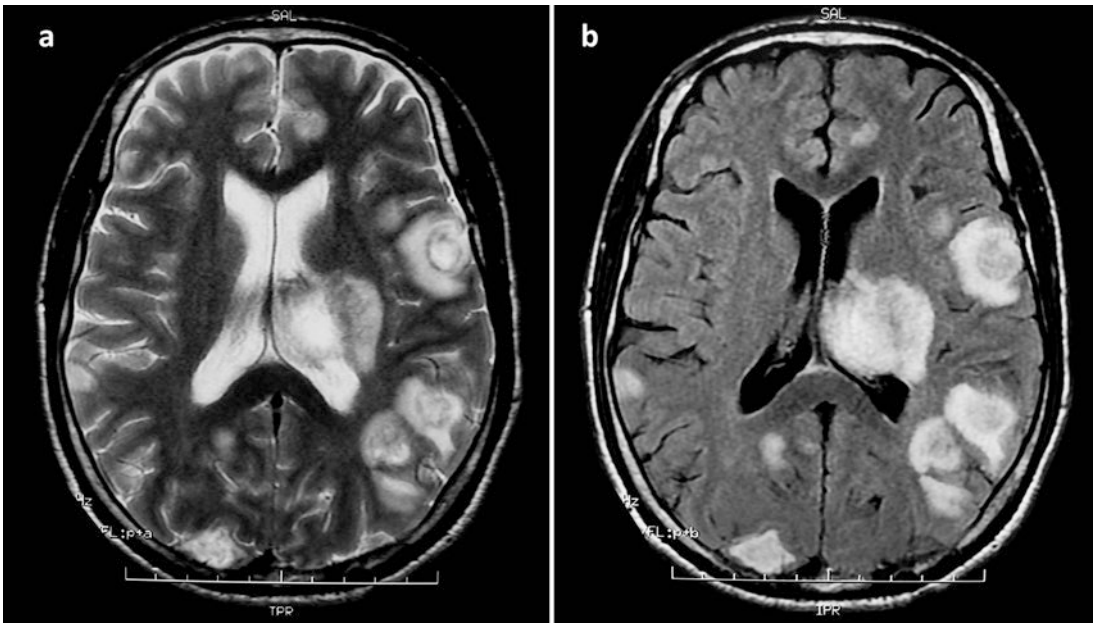


**Fig. 30.11** Case 30.3. The same granulomatous lesions in an axial T2-weighted image (**a**) and a FLAIR sequence (**b**), showing extensive perifocal edema

ascending bilateral extremity weakness associated with hyporeflexia or areflexia, as well as bladder and/or bowel incontinence. MRI may show gadolinium-enhancing and greatly thickened nerve roots.

CSF examination reveals a polymorphonuclear pleocytosis, low glucose, and elevated protein levels. The diagnosis of CNS CMV infection is based on PCR of CSF or brain tissue. Quantitative PCR results also can help in evaluating disease severity and screening for response to antiviral therapy.

The treatment of CMV infection in HIV patients is not well established. A combination antiretroviral therapy in addition to one anti-cytomegalovirus treatment (ganciclovir, valganciclovir, or cidofovir) is widely used. This treatment should be sustained until neurologic symptoms disappear.

## Tuberculosis

Tuberculosis is discussed in Chap. 23. In this chapter, Fig. 30.15 illustrates cerebellar tuberculomas in an HIV-infected patient.

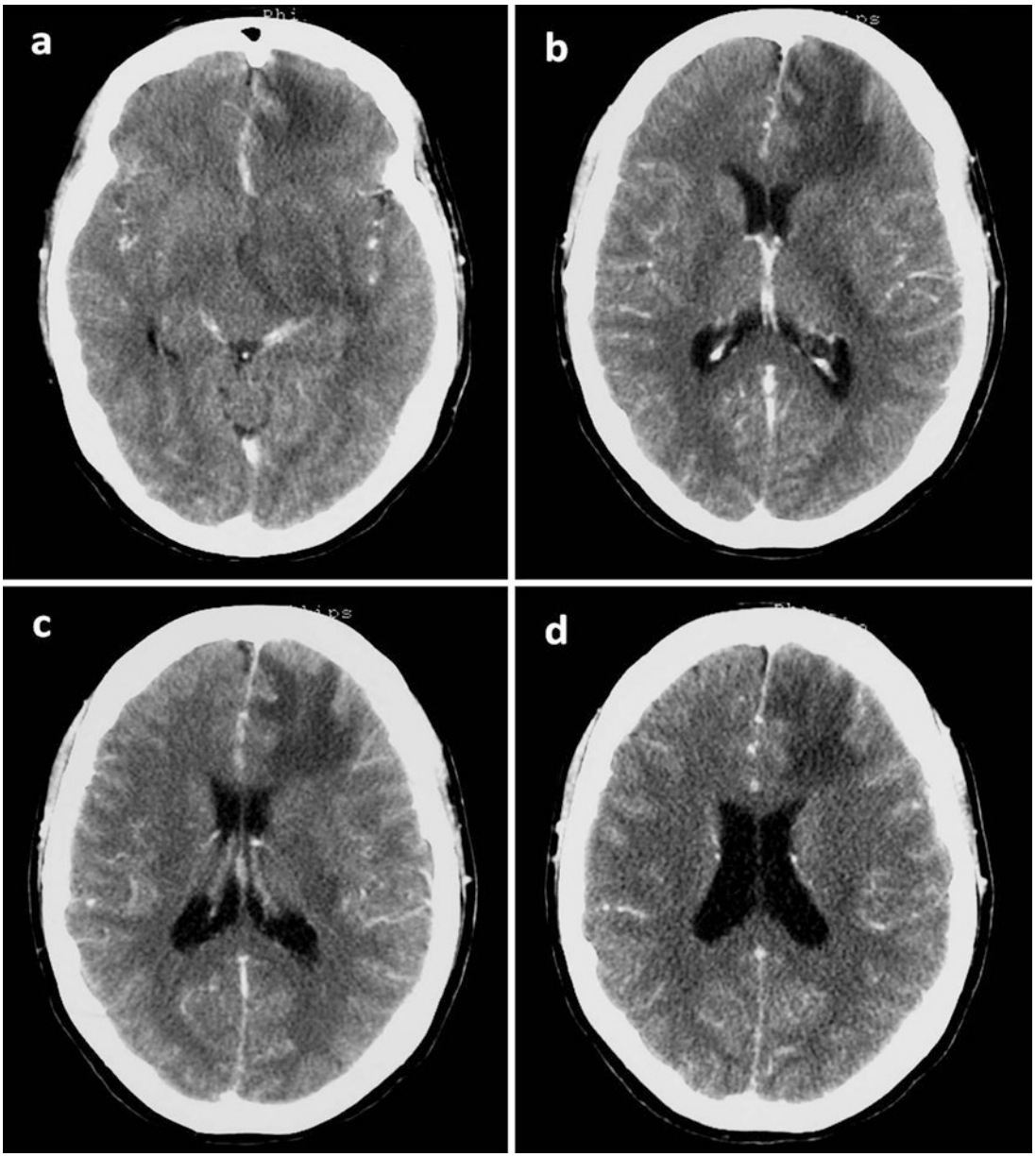


**Fig. 30.12** Case 30.4. HIV-associated toxoplasma encephalitis. (**a**–**d**) Axial cranial CT scanning following contrast administration reveals an extensive encephalitis that is predominant in the left frontal area and lacks contrast enhancement

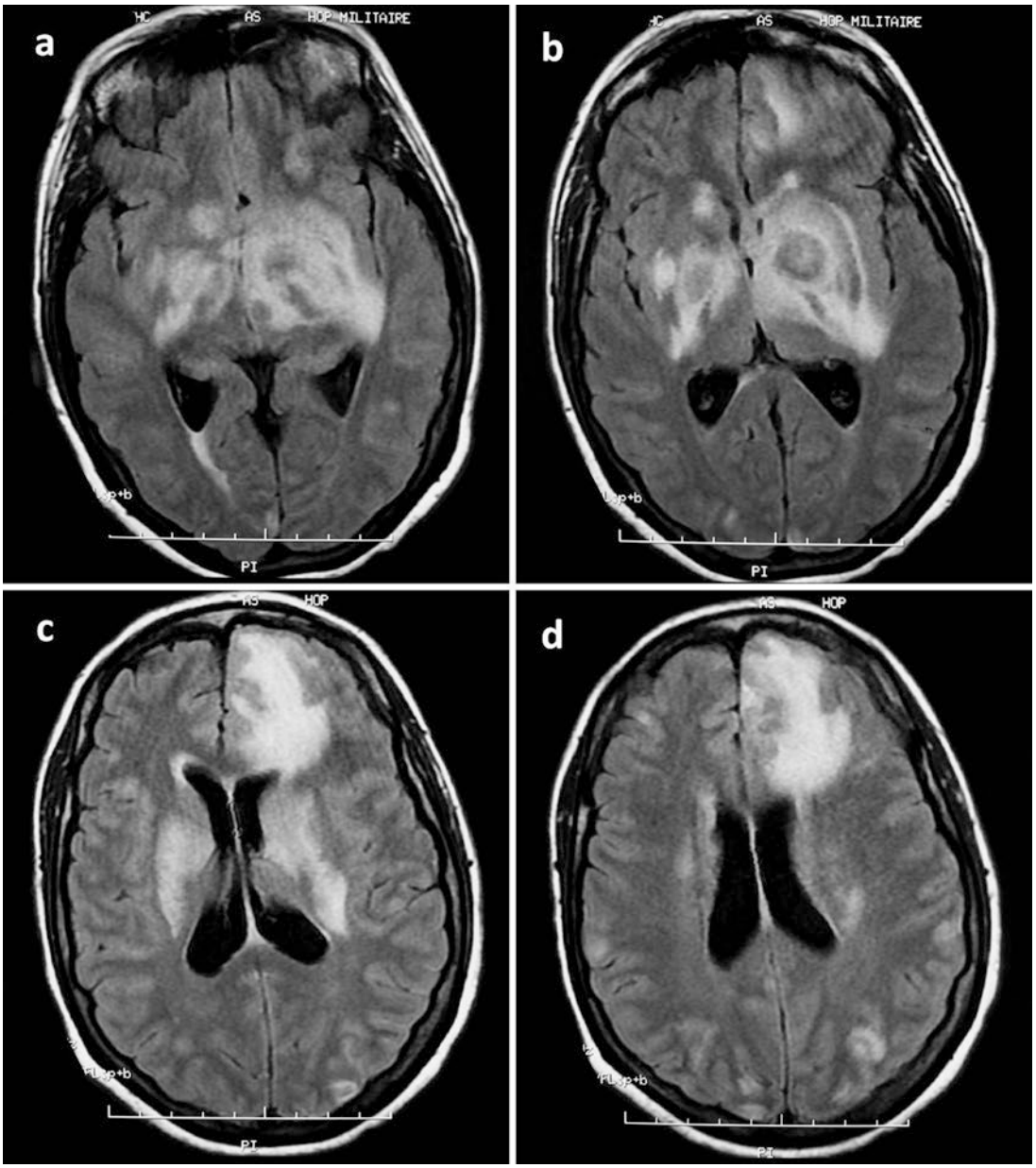


**Fig. 30.13** Case 30.4. HIV-associated toxoplasma encephalitis. (**a**–**d**) On axial MRI FLAIR sequence, the same lesions show extensive, nonspecific white matter abnormalities (deep and subcortical), also appearing in the basal ganglia and the periventricular area

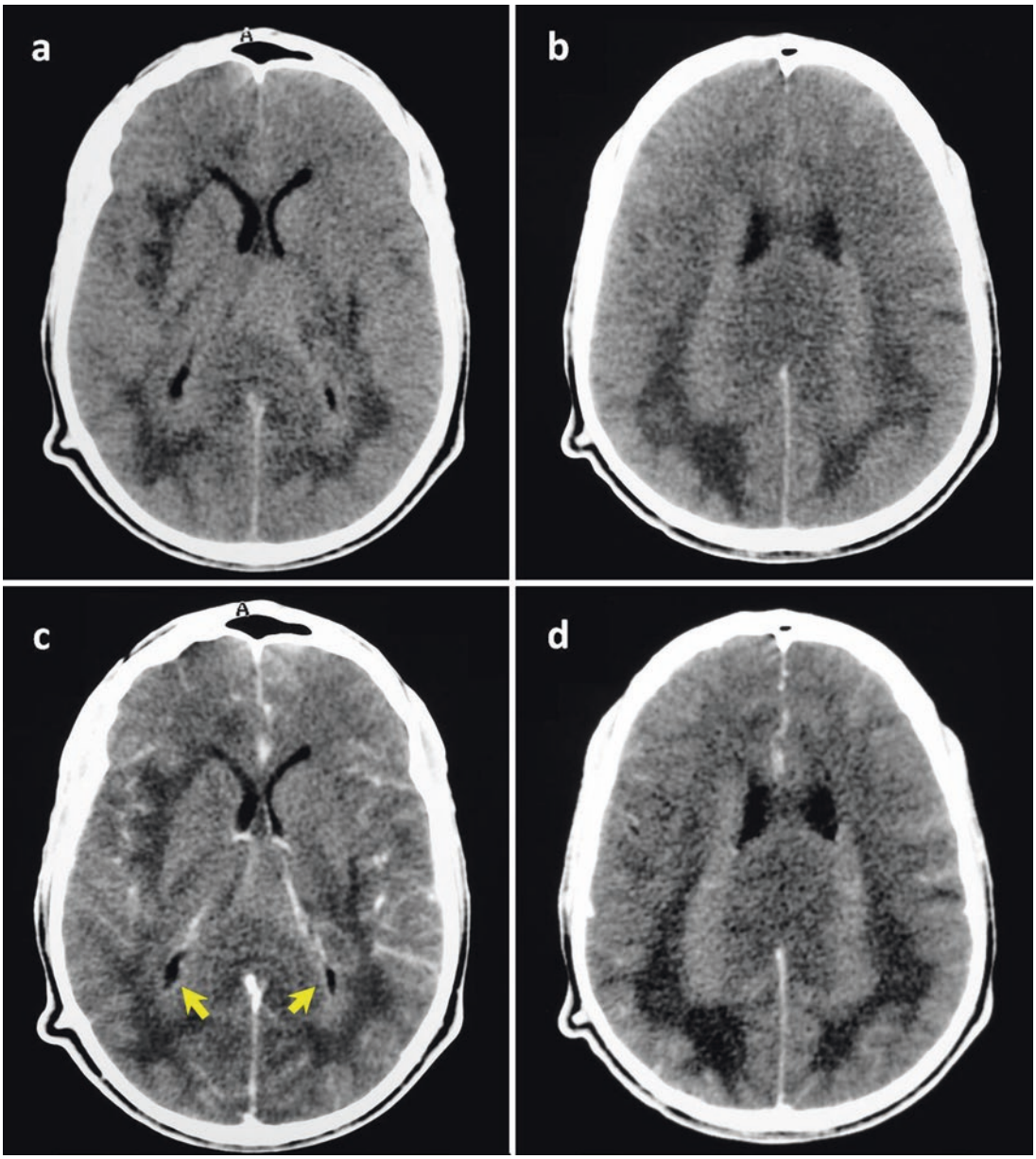


**Fig. 30.14** Extensive acute cytomegalovirus meningoencephalitis in an HIV-infected person. Axial brain CT scans before (**a**, **b**) and after (**c**, **d**) contrast injection reveals diffuse, nonspecific white matter abnormalities, which appear as areas of low attenuation with periventricular distribution and meningeal and mild ependymal enhancement (*arrows*). This patient's clinical condition did not support MRI

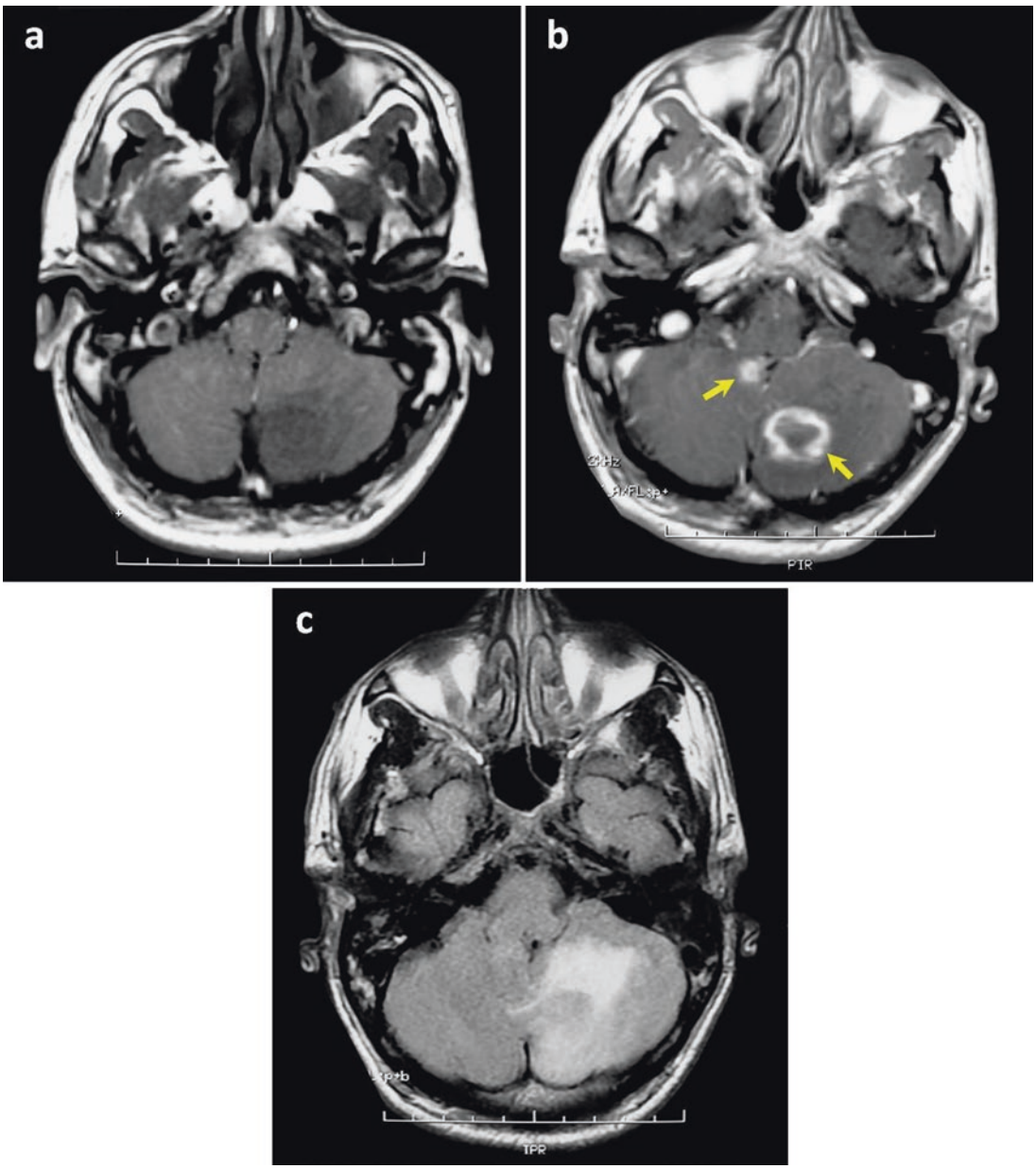


**Fig. 30.15** HIV-associated cerebellar tuberculomas. Axial nonenhanced (**a**) and enhanced (**b**) T1-weighted images and a FLAIR sequence (**c**) showing two granulomatous lesions of the posterior fossa (nodular and ring enhancement) (*arrows*) with surrounding edema

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A. Akhaddar, *Atlas of Infections in Neurosurgery and Spinal Surgery*, DOI 10.1007/978-3-319-60086-4