Skull Base Lesions

34

Ying Zou, Shuang Xia, Zujiang Xiong, Hui Dai, and Heng Liu

34.1 Skull Base Osteomyelitis

34.1.1 Overview

Skull osteomyelitis is a disease of skull infection and destruction caused by aerobic or anaerobic bacteria, mycobacteria or fungi, which is most common in frontal bone and parietal bone. Skull base osteomyelitis (SBO) is rare, and the etiology of SBO is not completely clear. SBO usually occurs in elderly diabetic patients or immunocompromised patients and often involves temporal bone, skull base, cranial nerves, and brain tissue. Its clinical manifestations are atypical, and its aggravation can threaten life. It generally develops rapidly and its prognosis is poor. SBO is often secondary to malignant external otitis (MEO) with invasive bone destruction. The lesions of MEO begin in the external auditory canal, and granulation tissue and cartilage necrosisare found at the bottom of the external auditory canal. The necrosis usually occurs at the junction of cartilage and soft tissue in the external auditory canal, and then spreads to the peripheral area. When the disease develops further, acute or chronic inflammation invades the stylomastoid foramen and jugular foramen through cartilage fissure of external auditory canal and milk fissure of middle eardrum, which leads to osteomyelitis of temporal bone or skull base osteomyelitis, leading to paralysis of cranial nerves function, meningitis or brain abscess, etc. Chronic mastoiditis and rhinosinusitis are also inducing factors of SBO. In addition, iatrogenic factors can also cause SBO, such as maxillectomy and mastoidectomy. Radiation osteomyelitis usually causes inflammation and necrosis of bone due to malignant tumors such as nasopharynx after high-dose radiotherapy.

Y. Zou
The First Affiliated Hospital of Tianjin University of TCM, Tianjin, China

S. Xia
Tianjin First Central Hospital, Tianjin, China

Z. Xiong
Chongqing Fifth People's Hospital, Chongqing, China

H. Dai · H. Liu (✉)
Affiliated Hospital of Zunyi Medical University, Zunyi, China

34.1.2 Pathology Findings

The main pathogen of SBO is Pseudomonas aeruginosa, and its internal and external toxins can destroy the peripheral tissues. Because of its strong virulence, infesction is difficult to control, which can cause severe fatal infection. Other pathogens include Staphylococcus epidermidis, Streptococcus pneumoniae, Aspergillus, etc. Lee et al. [1] reported SBO caused by Enterobacter aerogenes infection in cases of sigmoid sinus thrombosis and necrotic pulmonary embolism.

1. Acute Suppurative Osteomyelitis It can be divided into the following three stages based on pathological changes.

1. Subperiosteal abscess stage: inflammatory cell infiltration in bone marrow cavity, mainly lymphocytes and plasma cells, which may contain a small amount of purulent blood.
2. Subperiosteal abscess stage: After 3–4 days of onset, pus in bone marrow cavity increased to subperiosteal, forming subperiosteal abscess.
3. Periosteum destruction stage: 7–8 days after onset, subperiosteal empyema penetrated into soft tissue, resulting in periosteum necrosis, destruction of bone blood supply, bone destruction in severe cases, blood vessel embolism, and formation of sequestrum.

2. Chronic Suppurative Osteomyelitis It is usually evolved from acute suppurative osteomyelitis. Plenty of lymphocytes, proliferating fibroblasts, and mesenchymal cells infiltrated into the bone marrow cavity, and bone reconstruction began with bone destruction, showing periosteum repair and hyperostosis.

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34.1.3 Imaging Findings

The main manifestations of the disease are bone destruction, sequestrum formation, subperiosteal abscess, hyperostosis and osteosclerosis, periosteal reaction, and involvement of adjacent structures. Meanwhile, it is often complicated with the primary lesions of paranasal sinus, middle ear mastoid and orbit, such as rhinosinusitis, otomastoiditis, cholesteatoma, intraorbital cellulitis, and so on. Osteomyelitis located in the anterior skull base is mostly caused by ethmoidal sinusitis, frontal sinusitis and orbital inflammatory diseases.

1. CT Examination The manifestation of osteomyelitis in early stage is the decrease of local density of skull plate diploe. With the progression of pathological changes, bone destruction began to appear. Bone destruction manifests as skull plate diploe, irregular low-density area in internal and external plates, and clear or blurred border. Sequestrums are punctate or irregular high-density bone masses disconnected with the surrounding bone. Subperiosteal abscess is not common in osteomyelitis at the skull base. When inflammation involves the orbit, abscess formation inferior to the orbital periosteum, showing fusiform and stripy slow-density shadows inferior to the periosteum. By enhanced scan, the thickened periosteum can be enhanced, but the abscess is not enhanced. Hyperostosis and osteosclerosis show thickening of the internal and external plates of skull, increased density of diploe and unclear margin. Periosteal reaction is uncommon, showing thin-line, stripy, or layered high-density shadow. Extracranial soft tissue invaded shows swelling of soft tissue, thickening of muscles, unclear margin, and increased density of intermuscular fat space. Enhanced scan shows that the involved muscles and intermuscular space is heterogeneous enhancement, and the lesion scope is more clearly shown. If an abscess is formed in soft tissue, it shows a low-density area, and the abscess wall can be enhanced by enhanced scan (Figs. 34.1 and 34.2).

2. MRI Examination It shows bone marrow edema in early stage of osteomyelitis, with bone marrow manifesting as hypointense on T_1WI and slightly hyperintense on T_2WI, with irregular shape and unclear margin. Bone destruction shows hypointense on T_1WI and hyperintense on T_2WI, the signal is heterogeneous and enhancement of involved area can be found by enhanced scan. Sequestrum shows irregular hypointense area which is disconnected with surrounding bone. The subperiosteal abscess shows hypointense, isointense, or hyperintense on T_1WI and mostly hyperintense on T_2WI according to pus components. By enhanced scan, the thickened periosteum could be enhanced, but the abscess shows no enhancement. Hyperostosis and osteosclerosis show bone thickening, and bone marrow cavity shows hypointense on T_1WI and hypointense on T_2WI. Periosteal reaction shows hypointense on T_1WI and hypointense on T_2WI. Extracranial soft tissue invasion manifests as muscle swelling, with hypointense on T_1WI and hyperintense on T_2WI, and heterogeneous linear and stripy hypointense shadows in hyperintense area of intermuscular fat. By enhanced scan, the lesions are heterogeneous enhancement. If abscess is formed, the abscess wall is enhanced in a ring shape.

34.1.4 Key Points of Diagnosis

1. Often have a history of rhinosinusitis, otomastoiditis, orbital inflammatory diseases, etc.
2. With symptoms of bacterial poisoning, blood routine often shows abnormal results.
3. Acute osteomyelitis manifests as bone destruction, subperiosteal abscess, periosteal reaction, and inflammatory changes of adjacent soft tissues.
4. Chronic osteomyelitis manifests as hyperostosis, osteosclerosis, and deformity.

34.1.5 Differential Diagnosis

1. Eosinophilic Granuloma It is mostly found in children and adolescents, with long course of disease, mild clinical symptoms, eosinophilia in blood routine, clear margin of bone destruction, mostly solitary, complicated with soft tissue mass.

2. Metastatic Tumor It is mostly found in middle-aged and elderly people, usually with a history of primary tumors. Osteolytic bone destruction is more common, which can be solitary or multiple, with irregular shape and blurred margin, confluent foci, and can be complicated with soft tissue mass. Osteosclerosis and periosteal reaction are rare.

34.1.6 Status Quo and Progress of Research

CT is the preferred imaging method for skull base osteomyelitis, especially when the lesion is secondary to malignant external otitis, it can show moth-eaten bone destruction of bone of skull base [2]. The lateral soft tissue along petrosal bone and occipital bone can show "oval sign" on the bone window due to the remodeling and erosion of lateral bone edge caused by soft tissue inflammation. There is significant hole erosion in jugular foramen, carotid canal and stylomastoid foramen. Enhanced examination can be used to evaluate the patency of carotid artery and jugular vein to determine whether there are other malignant tumors.

MRI can be used as a supplement to CT examination, and MRI is more helpful to detect whether inflammation involves intracranial. Whether it is direct, peripheral, or vascular, MRI can better evaluate the involvement of occipital foramen [3–5].

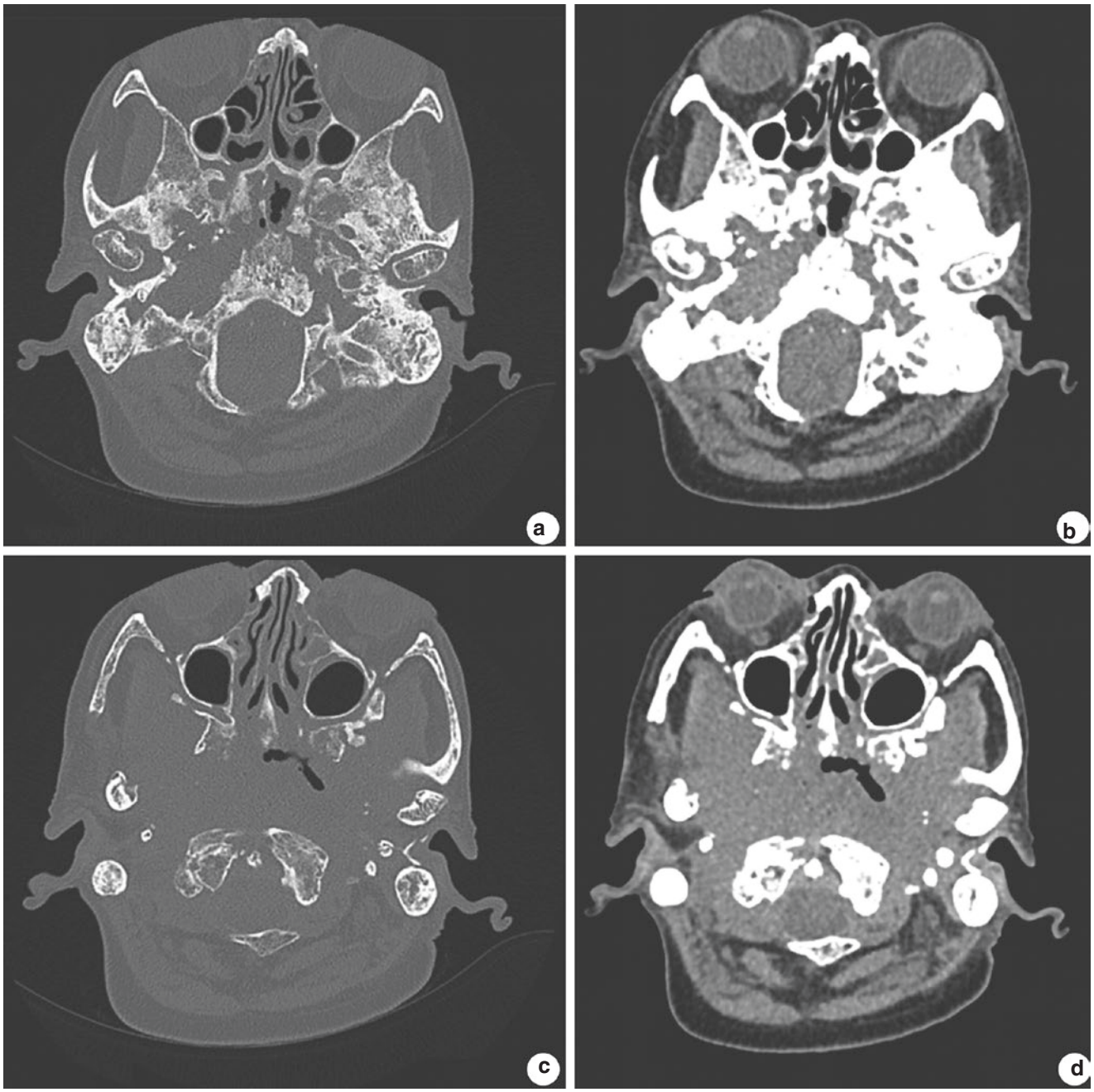

Fig. 34.1 Radiation osteomyelitis of bone of skull base. A 65-year-old female patient Postoperative radiotherapy for nasopharyngeal carcinoma. (**a**–**d**) Non-enhanced CT scan shows diffuse bone cortex discontinuity in the sphenoid bone, occipital bone, mastoid portion of bilateral temporal bones, and bilateral mandible, and bone trabecula in disorderly arrangement and with cellular change and local osteosclerosis. The nasopharyngeal roof is thickened with unclear margin, and irregular soft tissue density shadows in bilateral parapharyngeal space

34.2 Skull Base Bone Tuberculosis

34.2.1 Overview

Bone tuberculosis accounts for about 1% of systemic mycobacterium tuberculosis infection, skull tuberculosis accounts for only 0.20–1.37%, and bone of skull base tuberculosis is even rarer. Although the incidence of tuberculosis has greatly increased in the world, there are only a few reports of skull tuberculosis, especially skull base tuberculosis, only sporadic reports [6]. Bone tuberculosis of skull base is usually caused by the direct spread of tuberculosis in adjacent parts (such as orbit, paranasal sinus, nasopharynx, pituitary gland, meninges of skull base, etc.), or by the spread of blood and lymph circulation, and a few are caused by the hematogenous dissemination of tuberculosis of lung and kidney.

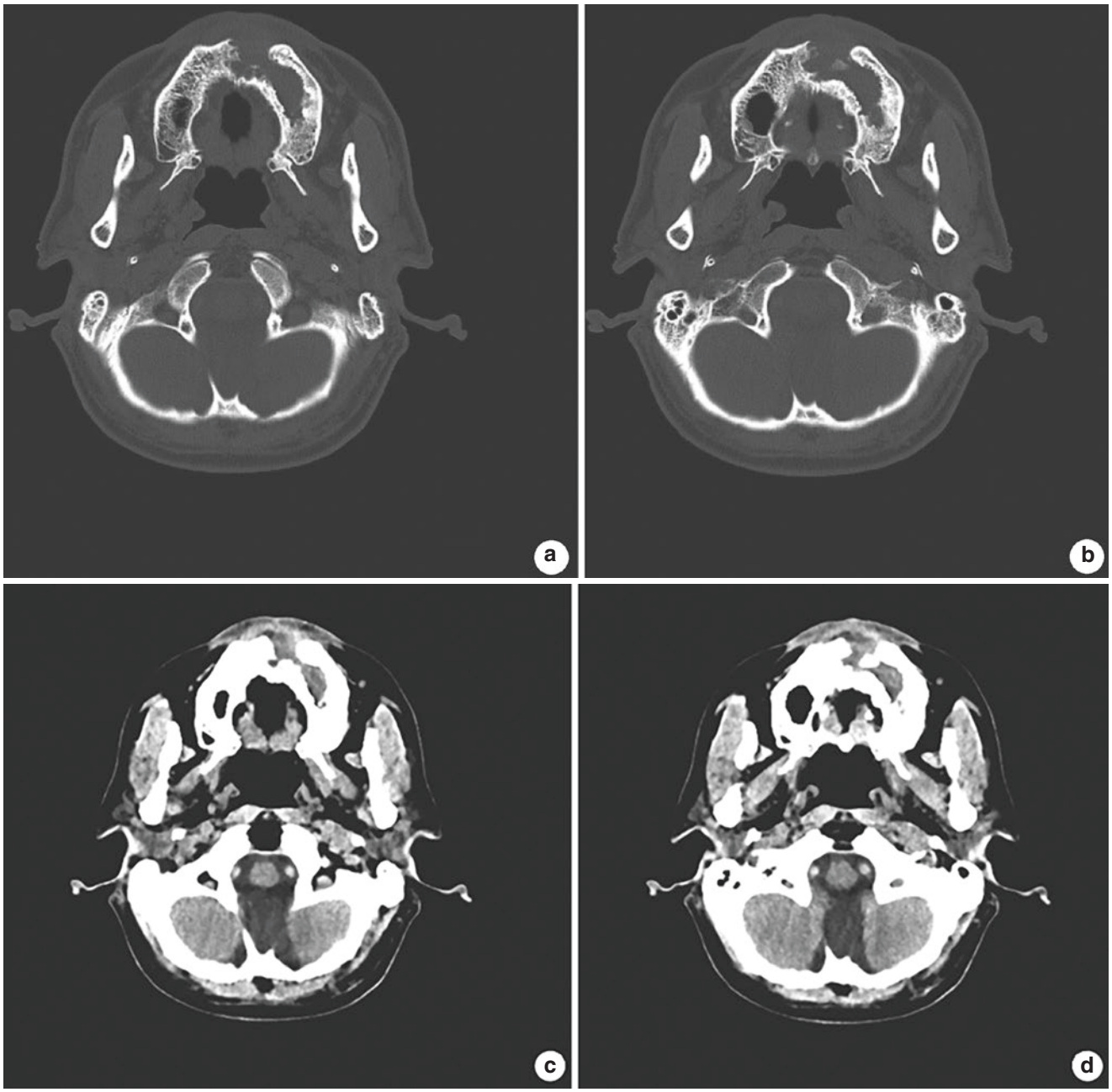

Fig. 34.2 Odontogenic maxillary osteomyelitis. A 60-year-old male patient. Pain in the left maxilla region with fever for more than half a month. (**a**, **b**) Non-enhanced CT scan on bone window shows bone destruction in the middle and left alveolar process of maxilla and edge sclerosis; (**c**, **d**) Non-enhanced soft tissue window CT scan shows axial soft tissue swelling

34.2.2 Pathology Findings

The pathological changes of skull tuberculosis are the same as those of bone tuberculosis in other parts, mainly showing three manifestations: exudation, degeneration, and proliferation. Macrophages and neutrophils are the main exudative lesions, complicated with cellulose exudation. Metamorphosis turned into caseous necrosis with calcification and sequestrum formation. Hyperplastic diseases become epitheloid cell proliferation, including Langerhans cells.

34.2.3 Imaging Findings

The main manifestations of the disease are bone destruction, sequestrum formation with hyperostosis and osteosclerosis, the adjacent structures are usually involved, and the periosteal reaction is mild or with unremarkable periosteal reaction. Meanwhile, there are multiple tuberculosis focus in adjacent structures such as paranasal sinus and orbit. Most of the lesions located in the anterior skull base were caused by the spread of tuberculosis in ethmoidal sinus and frontal sinus.

1. CT Examination The bone destruction usually shows that it penetrates the bony defect area of the inner and outer plates of skull, which is replaced by soft tissue density shadows, with irregular shape, clear or blurred margin, and unremarkable periosteal reaction. A few sequestrums and punctate calcifications can be found in the bony defect area. Sequestrums are small, showing irregular high-density bone masses which are disconnected with the surrounding bone. Hyperostosis and osteosclerosis is limited, which is manifested by thickening of skull around the destruction area and increasing density of diploe. Invasion to peripheral soft tissue shows soft tissue swelling, which could form irregular mass with unclear margin. In enhanced scan, the involved soft tissues show heterogeneous enhancement, and the lesion area is more clearly shown (Fig. 34.3).

2. MRI Examination As the pathological components of tuberculosis focus are complex, and exudative lesions, proliferative lesions, caseous necrosis, and calcification are mixed, so the MRI signals are very complex. Bone destruction shows hypointense on T_1WI and hyperintense on T_2WI. Sequestrums and calcification foci show punctate and small stripy hypointense on T_1WI and hypointense on T_2WI. The invasion to peripheral soft tissues often shows as mass shadow, isointense is dominant. Caseous necrosis shows hypointense on T_1WI and hyperintense on T_2WI. By enhanced scan, the components of proliferative granuloma in the lesion show marked enhancement, while the other components were not enhanced, but the adjacent meninges were enhanced.

34.2.4 Key Points of Diagnosis

1. Often have tuberculosis or other parts of the extrapulmonary tuberculosis history.
2. Tuberculosis infection often occurs in paranasal sinus area.
3. Bone destruction, formation of sequestrum with hyperostosis and osteosclerosis, involvement of adjacent structures, light periosteal reaction, or unremarkable periosteal reaction.

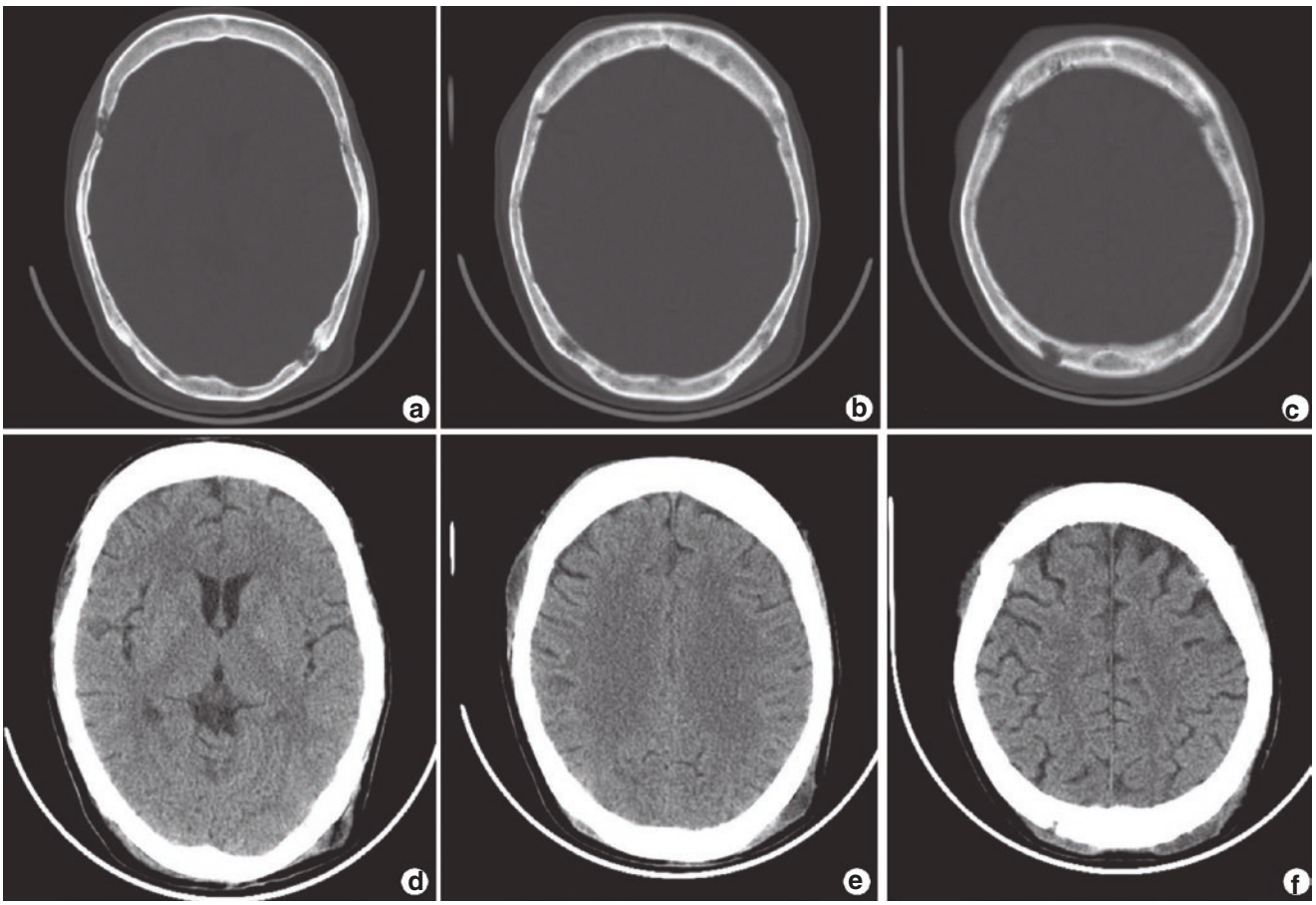

Fig. 34.3 Multiple bone tuberculosis in occipital bone. A 55-year-old female patient. Head lump was found with anorexia for more than half a month, and cough and expectoration increased for 1 week. According to diagnosis, the patient suffered from secondary pulmonary tuberculosis, bronchial tuberculosis, left ninth and tenth posterior rib tuberculosis and thoracic 12 cone tuberculosis. (**a**–**c**) Non-enhanced brain CT scan on bone window shows multiple bone destruction of skull; (**d**–**f**) Non-enhanced brain CT scan on soft tissue window shows that the bone destruction area is replaced by soft tissue density shadows, forming irregular mass with unclear margin

34.2.5 Differential Diagnosis

1. Myeloma It is mostly found in the elderly. Laboratory test shows that blood calcium is increased, serum-specific immunoglobulin is increased, with positive Bence Jones protein in urine. Bones of the whole body with abundant red bone marrow are usually involved. Skull is one of the common parts, which manifests as multiple, penetrating bone destruction, with quasi-circular shape, clear margin, and no sclerotic margin. May be complicated with soft tissue mass.

2. Eosinophilic Granuloma It is mostly found in children and adolescents, with long course of disease, mild clinical symptoms, eosinophilia in blood routine, clear margin of bone destruction, mostly solitary, complicated with soft tissue mass.

3. Metastatic Tumor It is mostly found in middle-aged and elderly people, usually with a history of primary tumors. Osteolytic bone destruction is more common, which can be solitary or multiple, with irregular shape and blurred margin, confluent foci, and can be complicated with soft tissue mass. Osteosclerosis and periosteal reaction are rare.

34.2.6 Status Quo and Progress of Research

For skull base bone tuberculosis, the diagnostic value of plain film is limited, and skull can show localized or diffuse solubility or localized periosteal lesions.

CT and MRI can clearly show osteolytic skull lesions, which destroy bone adjacent to epidural soft tissue mass, and show peripheral enhancement, low density, epidural mass, and peripheral enhancement performance is not clear, all of which indicate tuberculosis, and most patients have good clinical and laboratory performance.

34.3 Fungal Infection of Skull Base

34.3.1 Overview

Fungal Infection of skull base is not rare, and it usually occurs in patients with low immunity and long-term use of glucocorticoid or antibiotics. Fungal Infection of skull base is mostly rhinogenic, and often secondary to acute and chronic invasive fungal rhinosinusitis and allergic fungal rhinosinusitis.

34.3.2 Pathology Findings

The pathological basis of Fungal Infection of skull base caused by acute and chronic invasive fungal rhinosinusitis is that plenty of fungi in paranasal sinus invade adjacent skull base tissues or organs, mainly through blood vessels. The gross specimens of acute invasive fungal rhinosinusitis are black or dark brown, and most of them are shapeless broken tissues. Histologically, there is a large coagulation necrosis in the tissue, in which hyphae can be found. Visual observations of chronic invasive fungal rhinosinusitis are severe congestion and polypoid lesion of nasal mucosa, or surface covered with yellow or black massive soft tissue-like tumor. It can be seen through an electron microscope that submucosal issues have been invaded, including bones and blood vessels. Chronic suppurative granulomatous inflammation is the main form, which is usually accompanied by chronic nonspecific inflammation. In addition, coagulation necrosis and fungal vasculitis may also occur, with fungal hyphae observable in the necrotic tissues.

The pathological manifestations of allergic fungal rhinosinusitis are jam or putty-like yellow or yellow-brown secretions in the cavity of diseased paranasal sinus, with fungal hyphae or fungal spores observable on the secretion smear. Histopathologically, massive eosinophil infiltration can be observed.

34.3.3 Imaging Findings

1. CT Examination The CT findings of early lesions of acute and chronic invasive fungal rhinosinusitis are not specific, and only minor inflammatory changes such as thickening of the nasal cavity and/or paranasal sinus mucosa can be observed. The characteristic CT manifestations of acute invasive fungal rhinosinusitis at the progressive stage are progressive bone destruction, extensive lesions, easily spread to the orbit and skull, diffuse enhancement of the optic nerve and meninges, and intracranial abscess or infarction. The typical CT manifestations of chronic invasive fungal rhinosinusitis are bone expansion and destruction of involved sinus wall. In severe cases, defects may be formed, and adjacent bones may be subject to different degrees of proliferation and sclerosis. The sinus cavity is filled with irregular soft tissue shadow with uniform density and rare calcification. The characteristic CT manifestations of allergic fungal rhinosinusitis are unilateral or bilateral multi-sinus cavity enlargement and consolidation. Strip-like and cloud-like high-density shadows are visible in the consolidation tissue, with unilateral or bilateral nasal polyps. The lesions sometimes may destroy the bone at the skull base and involve the intracranial part, which is manifested as the soft tissue shadows in the paranasal sinuses protruding into the skull through the bony defect area at the skull base (Figs. 34.4 and 34.5).

2. MRI Examination The characteristic manifestations of acute invasive fungal rhinosinusitis include a wide range of involvement, mild disease of paranasal sinus or severely involved tissues and organs such as adjacent skull base,

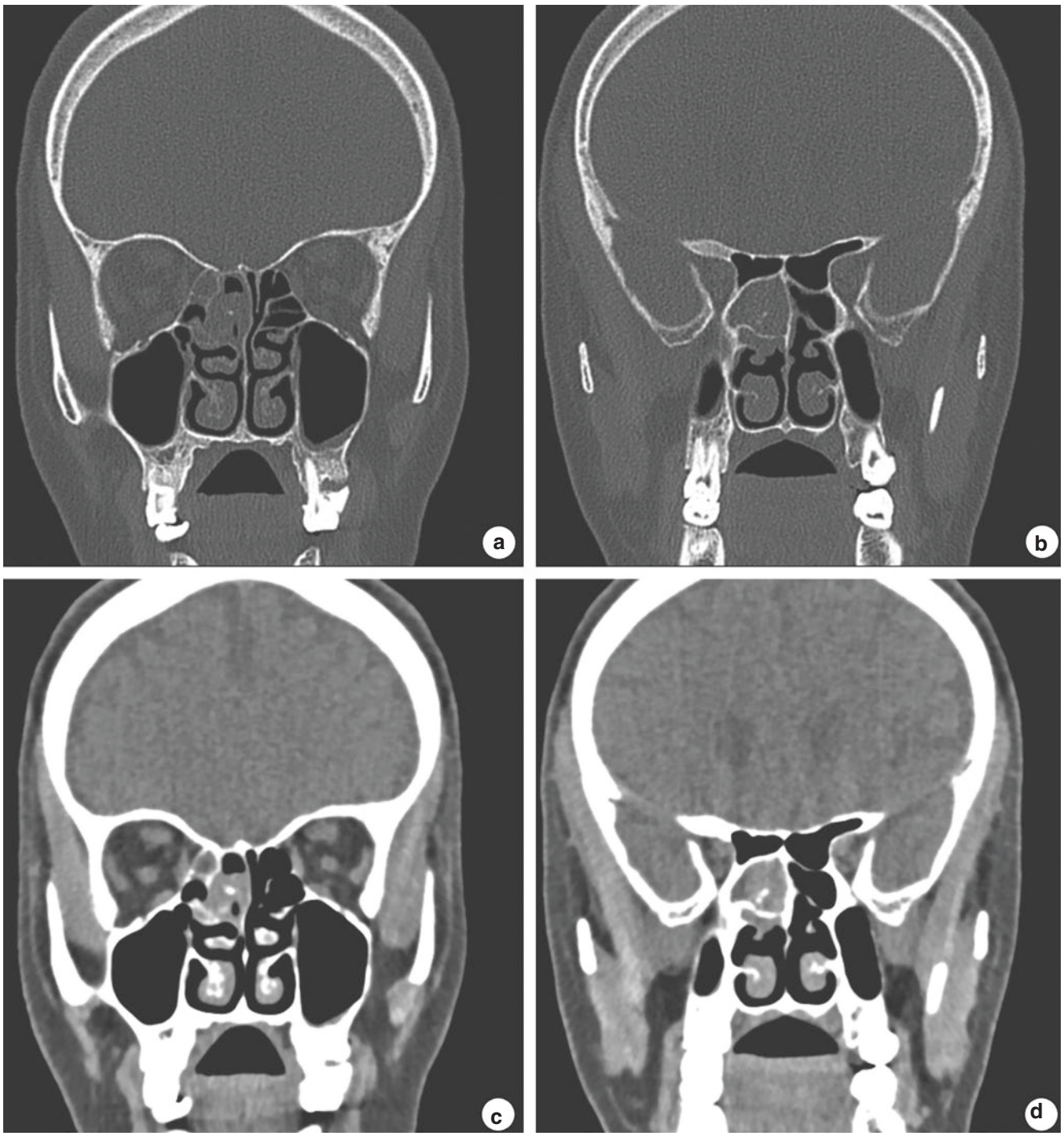

Fig. 34.4 Fungal sphenoiditis (1). A 60-year-old female patient. (**a**–**d**) The non-enhanced CT scan of paranasal sinus (coronal) shows soft tissue density shadows in the sphenoid sinus and right ethmoidal sinus, calcified shadow in them, unobstructed ostiomeatal complex, and unobstructed meatus nasi communis

orbits, and maxillofacial region. The fungal granuloma shows heterogeneous signal on T_1WI and significantly changing signals on T_2WI. The slight hypointense area in the lesion is the characteristic manifestation of fungal infection. The basicranial fungal meningitis shows isointense or hypointense on T_1WI, and mostly isointense or slightly hypointense on T2WI. Enhanced scan shows irregular enhancement of the meninges at the skull base or of the entire cerebral hemisphere, with possible local extradural abscess and cerebral dura mater or arachnoid enhancement. The chronic invasive fungal rhinosinusitis shows mostly isointense on T_1WI but uncertain signal on T_2WI, with mainly hyperintense at the early stage and hypointense at the advanced stage. The signal is often heterogeneous. The enhanced scan shows uneven enhancement of lesions. The allergic fungal rhinosinusitis shows

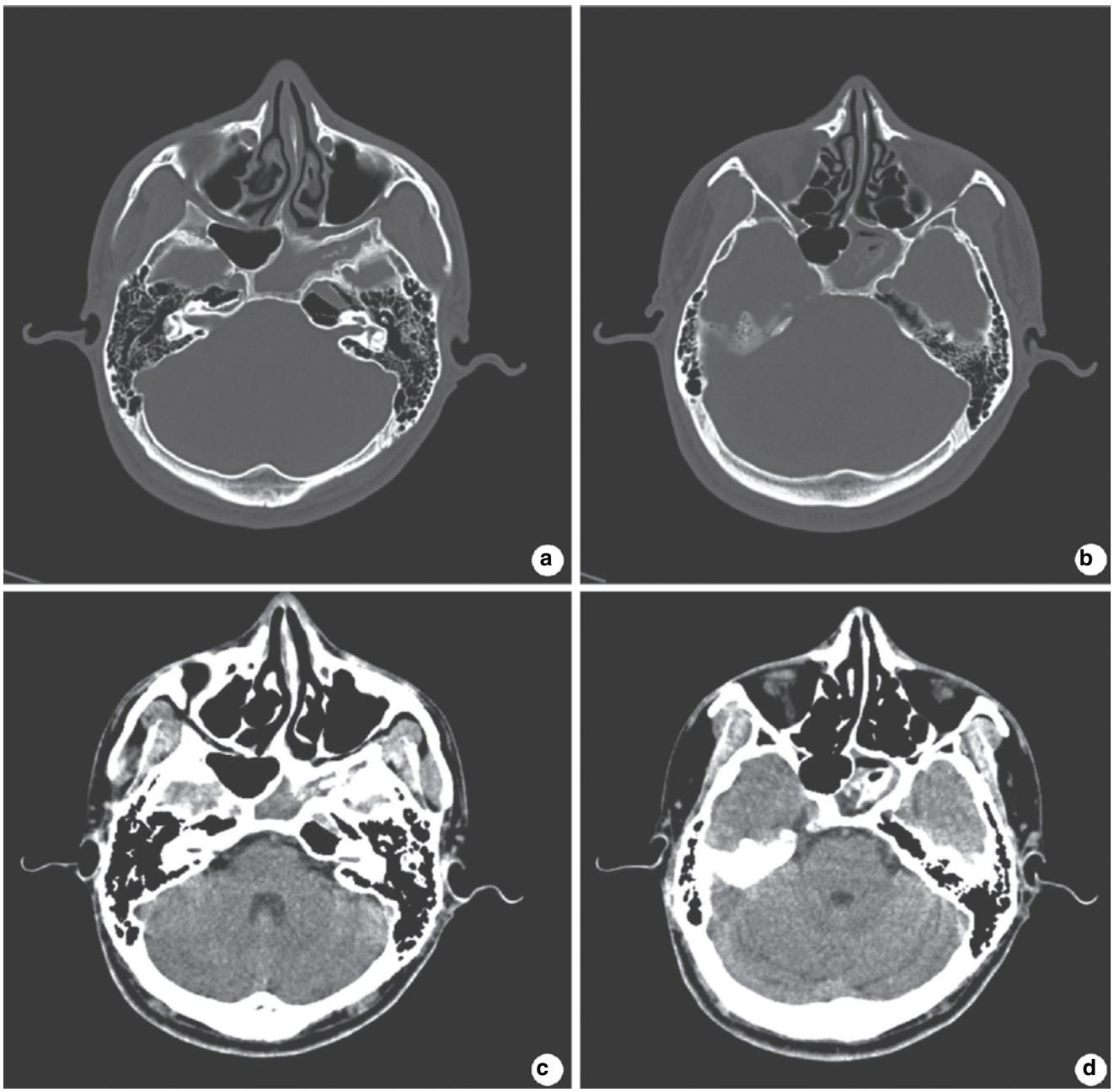

Fig. 34.5 Fungal sphenoiditis (2). A 28-year-old male patient. (**a**–**d**) The non-enhanced CT scan of the skull shows soft tissue density shadows in the sphenoid sinus, with visible calcified shadows and local sphenoid bone osteosclerosis

isointense or slightly hyperintense on T_1WI but extremely hypointense on T_2WI, and the enhanced scan shows its linear enhancement.

34.3.4 Key Points of Diagnosis

1. Acute Invasive Fungal Rhinosinusitis

1. The patient has a medical history of diabetes or is administered with a lot of hormones or antibiotics recently.
2. The patient has a persistent severe headache with or without symptoms of basicranial neurologic impairment.
3. The severity of rhinosinusitis is disproportionate to that of the invaded peripheral tissues.
4. The MRI examination shows a wide range of peripheral tissues and organs invaded by the lesion as well as thickening and enhancement of adjacent basicranial meninges.
5. The antifungal therapy is effective.

2. Chronic Invasive Fungal Rhinosinusitis

1. The disease is common in adults and sometimes in patients with immune deficiencies such as diabetes and leukemia.

2. The disease progresses slowly, and its early symptoms are similar to those of non-invasive fungal rhinosinusitis at early stage. Months or years later, symptoms such as headache, proptosis, impaired vision, and cranial nerve injury may occur.
3. Rhinosinusitis mostly involves single sinus involvement, with the maxillary sinus the most common. The sinus cavity expands, the bone of sinus wall is destroyed, and the bone adjacent to the bony defect area shows varying degrees of proliferation and sclerosis.
4. MRI examination can show the extent of involvement by lesions.

3. Allergic Fungal Rhinosinusitis

1. The disease is common in young patients with allergies but without immune deficiency.
2. The disease involves multiple paranasal sinuses on one or both sides, and the CT soft tissue window shows enlargement and consolidation of the sinus cavity involved by the lesions as well as multiple strip-like or cloud-like high-density shadows.
3. The CT bone window shows that the lesions erode the bone at the adjacent skull base and protrude the skull.
4. The disease is commonly accompanied by unilateral or bilateral nasal polyps.

34.3.5 Differential Diagnosis

1. Acute Invasive Fungal Rhinosinusitis When the lesion has no obvious bone changes, it is mainly distinguished from the non-fungal inflammation of skull base, including tuberculous meningitis and non-specific inflammation. Some cases are accompanied by bone erosion and destruction in the lesion area, requiring to distinguish it from the malignant tumor of skull base. Tuberculous meningitis usually occurs in young people, most of whom have a history of tuberculosis. It can be definitively diagnosed by biochemical analysis of the cerebrospinal fluid obtained by lumbar puncture as well as bacterioscopy. The lesion of basicranial non-specific inflammation usually shows more hyperintense on T_2WI, while that of the fungal inflammation shows a slightly hypointense on T_2WI, which is helpful for distinguishment. Malignant tumors of the skull base are mostly manifested as irregular-shaped soft tissue masses of skull base, which is accompanied by osteolytic destruction but without any history of diabetes or extensive use of hormones or antibiotics.

2. Chronic Invasive Fungal Rhinosinusitis The disease is mainly distinguished from paranasal sinus cancer invading the skull base. Sinus cancer has a short course and progresses quickly. Paranasal sinus cancer has a short course of disease and progress quickly in most cases. Common in the maxillary sinus, it is manifested as extensive bone destruction in the sinus wall, mostly without hyperostosis and osteosclerosis, as well as heterogeneous soft tissue density.

3. Allergic Fungal Rhinosinusitis It is necessary to distinguish it from the skull base abnormalities caused by other factors when the skull base is invaded. It is easy to distinguish it in combination with the characteristic CT manifestations of paranasal sinus and nasal cavity.

34.3.6 Status Quo and Progress of Research

CT examination is a primary examination approach when the fungal rhinosinusitis invades the skull base. It can clearly show the destruction and invasion of skull base bone by the lesions. Composed of a cone X-ray beam and a flat panel detector, cone-beam CT (CBCT) can move around the patient's head. It was originally used for dental imaging and has been widely used in ENT examinations. Compared with the traditional multidetector CT (MDCT), CBCT has higher resolution and less radiation exposure. Intrasinus calcification is a common manifestation of fungal rhinosinusitis, especially aspergillosis. CBCT can improve the detection rate of calcification to a certain extent [7].

Among all fungal rhinosinusitis, acute invasive fungal rhinosinusitis (AIFR) is dangerous and may endanger the patient's life. Some scholars [8] have established a simple and reliable CT-based diagnostic model through research, which can be used as a routine screening tool for high-risk patients. Ideally, this model will make the diagnosis or exclusion of AIFR more reliable than any previous one. Choi et al. [9] studied the relationship between the MRI imaging characteristics and AIFR and the prognosis and found that AIFR showed frequent invasion outside the paranasal sinus as well as variable MRI enhancement patterns. About half of the cases show the lack of contrast enhancement (LoCE) enhancement mode. Among various clinical radiological factors, LoCE enhancement mode is a unique prognostic factor.

34.4 Tolosa-Hunt Syndrome

34.4.1 Overview

Tolosa–Hunt syndrome (THS) is an idiopathic inflammation that often involves cavernous sinus and orbital apex. Its exact cause is not yet clear, and it may be an allergic disease Its typical clinical manifestation is the painful ophthalmoplegia caused by the inflammation of surrounding cavernous sinus. Tolosa-Hunt syndrome is essentially an exclusive clinical diagnosis. Because the diagnosis is mainly based on clinical features, the diagnosis of this

disease lacks objective evidence [10]. The disease usually occurs in 35- to 75-year-old persons, especially the about 50-year-old ones. No significant gender difference is observed, with slightly more male patients. It is generally unilateral, with no significant between left and rights sides. The first symptom is usually unilateral postorbital intractable pain, which usually occurs before ophthalmoplegia. The cause of such pain is mostly caused by the stimulated first branch of the trigeminal nerve. Most patients show the symptoms of cranial nerve paralysis after suffering ophthalmodynia for a period of time, which range from hours to 6 months. In this case, the cranial nerves III-VI are mainly involved. The clinical manifestations include ptosis of the affected side; paralysis of the extraocular muscles, which may be accompanied by strabismus, diplopia, corediastasis, and the pupil not reacting to the light; and eyeball fixation in a tiny minority of cases. Occasionally, it may affect optic nerves, facial nerves, and sympathetic nerves around the arteries, and the signs of obstructed orbital venous return may also occur. It is significantly effective to treat the disease with corticoid, and the pain disappears within days. It is relatively slow to recover from the symptoms of cranial nerve injury and they are easy to relapse, but the prognosis is good, and there are fewer patients remaining cranial nerve dysfunction.

34.4.2 Pathology Findings

The main pathological features of THS are the infiltration of lymphocytes and plasma cells, and the thickening of cerebral dura mater in the cavernous sinus. SIPHA periarteritis, or localized duritis pachymeningitis of the cavernous sinus.

34.4.3 Imaging Findings

1. CT Examination The extent of lesion mostly involves both the orbital apex and the adjacent cerebral dura mater. Enhanced scan shows marked enhancement of the lesions of cavernous sinus and orbital apex on the affected side as well as thickening and strip-like marked enhancement of the involved cerebral dura mater. The cavernous sinus area on the lesion side shows asymmetric enlargement, with or without enhancement. The internal carotid artery becomes narrower, and the superior orbital fissure and the orbital apex expand.

2. MRI Examination The disease is manifested as widening of the cavernous sinus on the affected side. Due to the large difference in size of the cavernous sinus between individuals, there is no recognized standard value. Therefore, whether the cavernous sinus is widened is mainly determined by comparing the cavernous sinuses on affected and unaffected sides. Re-examination after treatment can help confirm the diagnosis if the lesions are significantly reduced or disappeared. Thin-slice MRI enhanced examination is a main imaging method for diagnosing the disease. When the Tolosa-Hunt syndrome of cavernous sinus is suspected clinically, the acquisition slice is generally 2–3 mm thick, and there is no increment enhanced examination. Thin-slice MRI enhanced examination can show the inflammatory changes of frontal area of cavernous sinus, superior orbital fissure, and orbital apex. The signal characteristics are nonspecific and may be manifested as follows: Compared with muscle, the affected area shows isointense to hyperintense on T_1WI and hyperintense on T_2WI. The T1WI enhanced examination may show enhancement of the lesion at the active stage, and the resolution has improved after treatment (Figs. 34.6 and 34.7).

34.4.4 Key Points of Diagnosis

1. Tolosa–Hunt syndrome is mainly manifested as widening of the cavernous sinus. The thin-slice MRI enhanced examination can clearly show the marked enhancement of widened cavernous sinus, which may be accompanied by enhancement of the adjacent cerebral dura mater.
2. Symptoms are relieved after hormone treatment, and reduction or disappearance of the original lesions can be used as the diagnostic criteria for THS.

34.4.5 Differential Diagnosis

1. Painful Ophthalmoplegia Caused by Nasopharyngeal Carcinoma The onset is insidious and gradually worsens. Generally, it is first manifested as unilateral ophthalmoplegia, and then involves the opposite side and other cranial nerves. CT examination carried out at the advanced stage can show bone destruction, and the diagnosis can be confirmed by a biopsy of the nasopharyngeal cavity.

2. Sphenoid Sinus Cyst The onset is most subacute. The disease is manifested as obvious local tenderness and protruding eyeballs. In addition to cranial nerves III-VI, it may also involve the optic nerve. At the early stage of onset, the symptoms can be alleviated by treatment with dehydrators and steroid hormones. Its diagnosis can be confirmed by CT and MRI examinations.

3. Intracerebral Aneurysm (Posterior Communicating Artery Aneurysms) The disease only involves the second

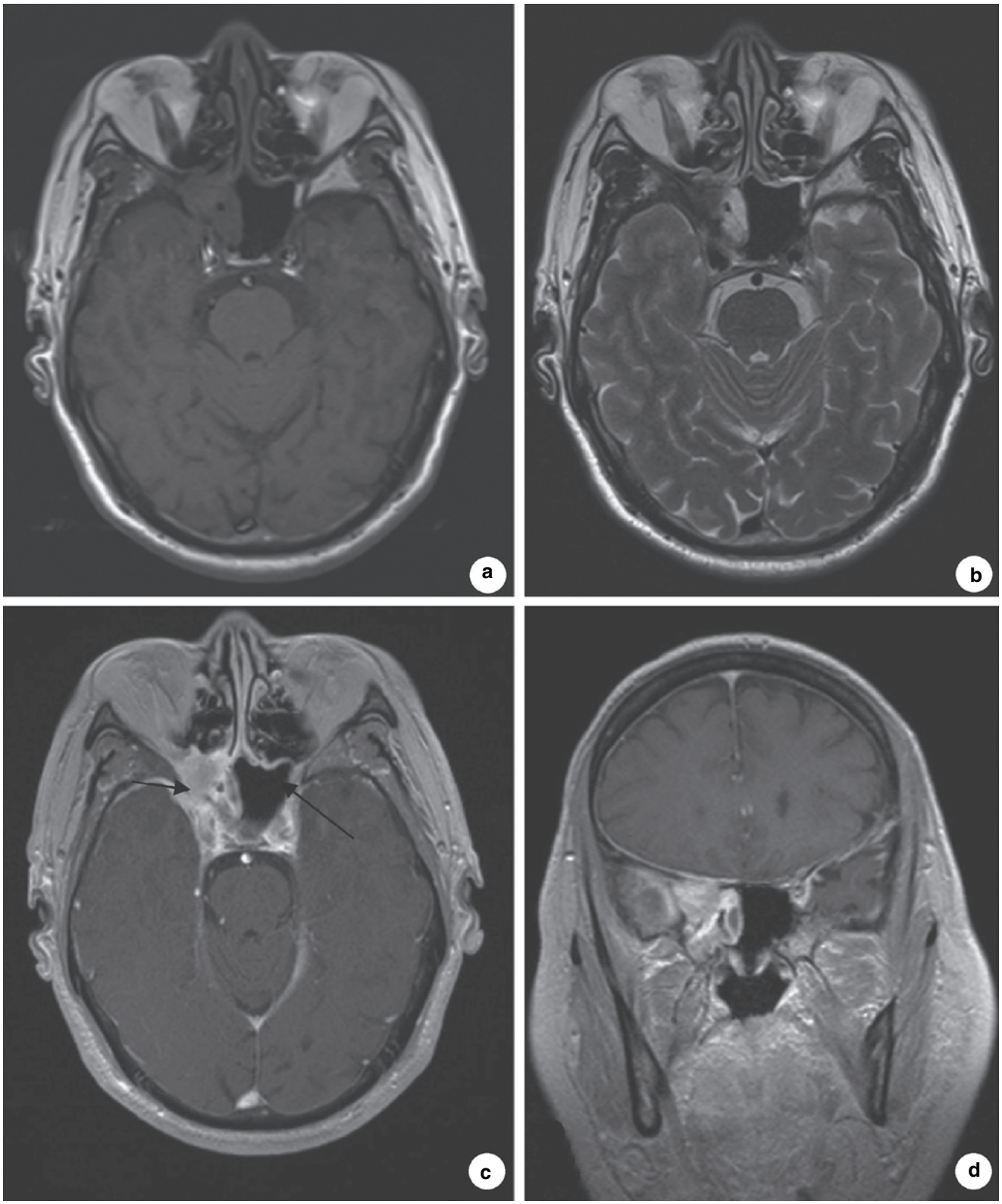

Fig. 34.6 Tolosa-Hunt syndrome (1). A 60-year-old female patient. Pain in both eyes for more than 1 month, and strabismus in the right eye for 1 week. (**a**, **b**) Non-enhanced MRI scan by T_1WI and T_2WI shows widening of the right cavernous sinus and heterogeneous signals. (**c**, **d**) Enhanced scan of T_1WI-CE transverse plane and coronal shows marked enhancement of the meninges adjacent to the right cavernous sinus, local thickening, and heterogeneous signals

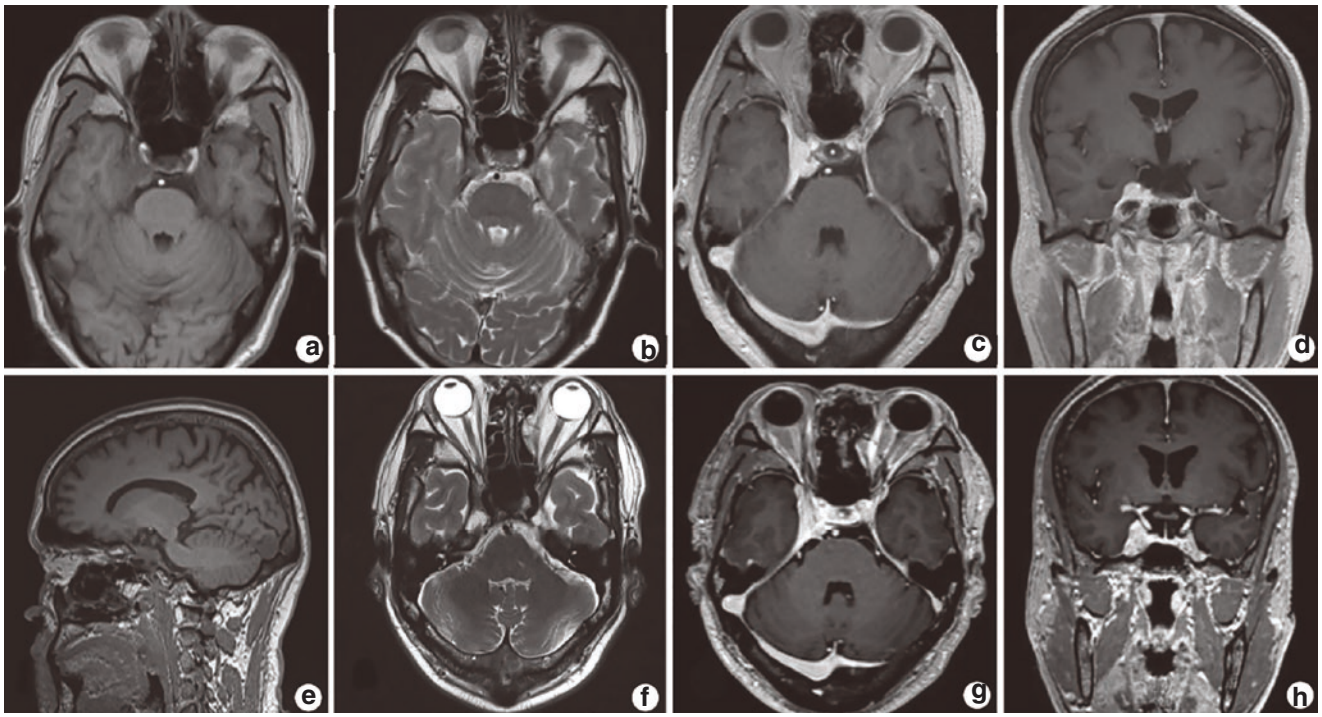

Fig. 34.7 Tolosa-Hunt syndrome (2). A 67-year-old female patient. Both eyes seeing an article as if it is two ones suddenly for 1 week. (**a**, **b**) Non-enhanced MRI scan by T_1WI and T_2WI shows local widening of the right cavernous sinus and visible abnormal shadow. (**c**, **d**) MRI enhanced examination shows abnormal reinforced nodule in the rear part of the right cavernous sinus. (**e**, **f**) After treatment with methylprednisolone, MRI re-examination shows that the abnormal enhanced nodule in the rear part of the right cavernous sinus has become smaller

cranial nerves, generally with light pain. It can be definitively diagnosed by angiography. The onset is slow, and it is accompanied by impaired vision and visual field defect. Its diagnosis can be confirmed by X-ray sella turcica plain film, CT examination, and MRI examination.

34.4.6 Status Quo and Progress of Research

THS is a rare neurological and ophthalmological disease, and MRI is the preferred imageological examination method. Compared with traditional brain MRI scan, thin-slice MRI scan and enhanced scan of cavernous sinus can better show the location, signal performance and scope of the lesion, which is helpful for diagnosis, differential diagnosis and follow-up observation of THS.

The advantages of 128-slice CT scan for TSH: After the CT scan is completed, any slice thickness and inter-slice increment can be reconstructed to avoid the shortcoming of conventional head MRI scan, i.e., some lesion details are missing due to big slice thickness and inter-slice increment. Even the thin-slice scan of cavernous sinus area has the disadvantages of small SFOV and inter-slice increment. After the 128-slice CT scan is completed, multi-plane recombination at any angle and slice thickness can be carried out, which can make the bilateral structure symmetrical and facilitate bilateral comparative observation. Once the MRI examination is completed, neither the angle nor the slice thickness can be adjusted. This is particularly important for the diagnosis and differential diagnosis of THS because the current THS imaging diagnosis mainly depends on bilateral contrast observation. After the 128-slice CT enhanced scan is completed, direct head angiography and blood vessel analysis can be performed, while MRI requires a separate blood vessel scan sequence. CT can easily show the calcification of blood vessel wall, skull base, and adjacent cerebral dura mater, helpful for the differential diagnosis of skull base tuberculosis, fungal infection of areas adjacent to the cavernous sinus, and so on. In addition, THS is often accompanied by severe pain, even nausea, vomiting, and other symptoms, making it difficult to check for immobilization for a long time. The quick speed of 128-slice CT examination is one of its advantages. Combined with CT post-processing VR imaging, it can show the tortuosity, dilation, and other changes of the drainage veins adjacent to the cavernous sinus. With quick scan speed and abundant image post-processing software, the 128-slice CT gradually shows its advantages in diagnosis and differential diagnosis of THS.

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