Neuroradiology (2009) 51:385–396
DOI 10.1007/s00234-009-0505-z

DIAGNOSTIC NEURORADIOLOGY

Orbital and anterior visual pathway infection and inflammation

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Received: 3 December 2008 / Accepted: 28 January 2009 / Published online: 17 February 2009

Abstract
Introduction Orbital and anterior visual pathway infection and inflammation represent approximately 10–15% of all orbital pathology. Both conditions can occur separately but occasionally they can be observed simultaneously.
Methods While the diagnosis of infection is usually straightforward, it is important to depict the lesions and to know the potential devastating complications. CT plays an important role in confirming the clinical suspicion of orbital infection.
Results The diagnosis and differential diagnosis of inflammation is more challenging. Differentiating inflammation from lymphoproliferative diseases and tumours can be difficult.
Conclusion MR imaging plays an important role but a dedicated orbital imaging protocol is mandatory.

Keywords Orbit · Infection · Inflammation · CT · MR imaging

Introduction

Orbital infection and inflammation can be considered as separate conditions, but infection may also lead to secondary inflammation of surrounding tissue and vice versa.

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Orbital infections develop via direct inoculation, extension from adjacent structures or haematogenous spread. The spread of infection from the orbit to the cavernous sinus occurs by the extended valveless venous network and may cause potentially life-threatening complications.

Orbital and anterior visual pathway inflammation may affect all age groups but is more common in adults. Orbital inflammation can be related to systemic inflammatory diseases but can also be idiopathic and can present both acutely or chronically.

In a survey of 1,264 patients with orbital tumours, a total of 133 (10.5%) inflammatory/infectious lesions were found. Idiopathic nongranulomatous inflammation was the most common abnormality (74%), followed by infections (10%) and granulomatous inflammation (9%) [1].

The role of imaging in the diagnosis of infection and inflammation will be reviewed.

Anatomy

Because of the complexity of orbital anatomy and its role in orbital infection, a brief review is given below.

The orbit is a cavity that contains the eye, orbital musculature, blood vessels and nerves. An important structure that marks the anterior border of the orbit is a fibrous band that separates the pre- and post-septal space. It is called the orbital septum (or palpebral ligament) and connects the two ends of the tarsus with the orbital wall. This structure forms a barrier to the spread of infection from superficial tissue into the orbit.

Only a thin plate of bone, the lamina papyracea, separates the medial orbital wall from the ethmoid sinuses. The superior and inferior orbital walls are also bounded by sinuses.

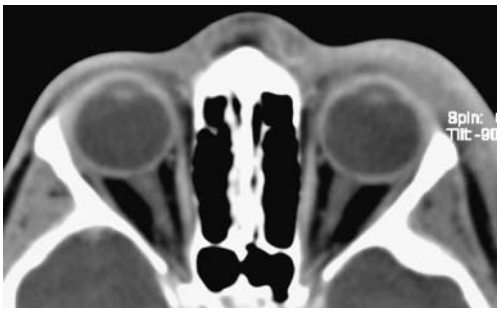

Fig. 1 Pre-septal cellulitis. A 10-year-old boy with swelling of the soft tissue of the face on the left side following an insect bite. Axial contrast-enhanced CT shows thickening of the left pre-septal soft tissues. There is no extension into the orbit

The optic nerve and the ophthalmic artery pass through the optic canal, which lies within the sphenoid bone at the orbital apex.

The superior orbital fissure is formed by the greater and lesser wings of the sphenoid bone. The oculomotor, trochlear, ophthalmic and abducens nerve enter the orbit through this fissure together with the superior ophthalmic vein.

The inferior orbital fissure is bordered by the sphenoid, maxillary and palatine bones and transmits the maxillary nerve, the infraorbital vessels and the ascending branch from the sphenopalatine ganglion.The spread of infection from the orbit to the cavernous sinus occurs by the valveless orbital veins. This valveless venous network is called the cerebrospinal venous system and extends from the head to the pelvis and consists of a group of veins and venous plexuses that freely communicate. The first of the two main divisions of this system are the intracranial veins (the cortical veins, the dural sinuses, the cavernous sinuses and the ophthalmic veins). The second main division, the vertebral venous system, includes the vertebral venous plexuses [2].The flow in these valveless veins is bidirectional and has a large capacity. This system plays an important role in the regulation of intracranial pressure, the spread of tumours, infection or emboli. Bacterial sinusitis may spread to the cerebrospinal venous system

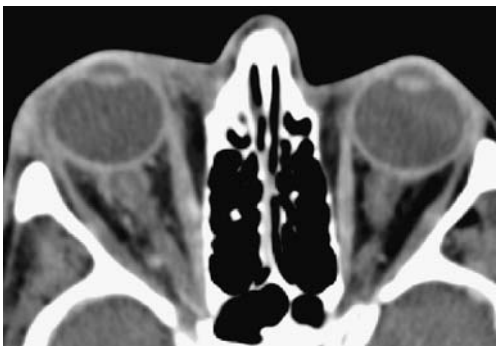

Fig. 2 Orbital cellulitis. An 8-year-old boy with fever and swollen eyelid on the right side. Axial contrast-enhanced CT shows infiltration of the orbital fat surrounding the right optic nerve and focal thickening of tissues at Tenon's capsule

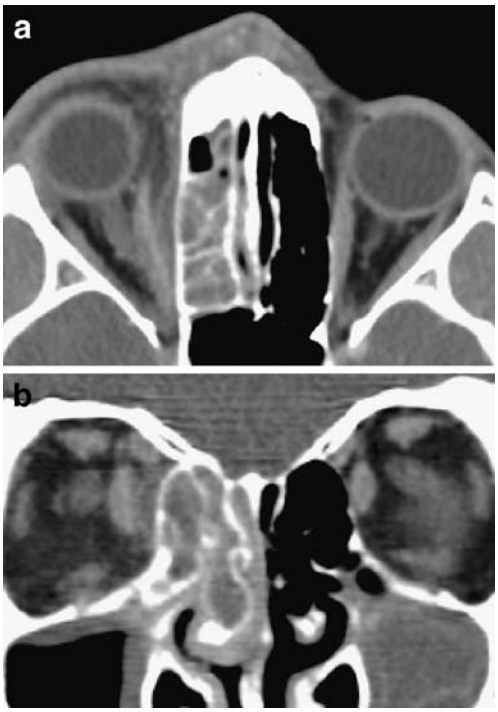

Fig. 3 Subperiosteal abscess. A 17-year-old boy with pain and diplopia. Axial (**a**) and coronal (**b**) contrast-enhanced CT shows paranasal sinus inflammation on the right side with adjacent subperiosteal collection in the orbit

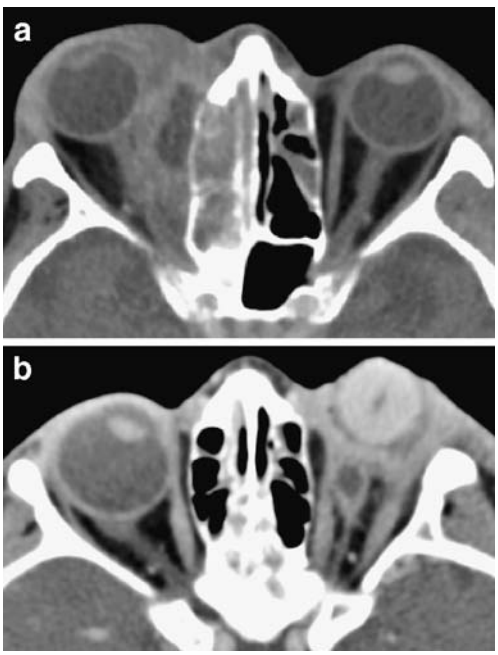

Fig. 4 Orbital abscess. A 16-year-old boy with pain, swelling and limited eye movements. Axial contrast-enhanced CT shows ethmoiditis and orbital cellulitis with pus collection in the right orbit (**a**). A 9-year-old girl with a prosthesis for congenital microphthalmos presenting with proptosis, pain and pus formation on the left side. On CT, there is evidence of orbital infection surrounding the prosthesis and a small orbital abscess (**b**)

through the cavernous sinus and then spread intracranially, causing meningitis, subdural empyema, intracerebral abscess, epidural abscess or superior sagittal sinus thrombosis. Septic thrombosis of the dural venous sinuses most frequently involves the cavernous sinuses. Cutaneous infections around the teeth, in the oral cavity or around the orbit are known to spread to the cavernous sinus [2].

Imaging strategy

An imaging examination is often requested because of the frequent inability to assess vision due to pain, edema, proptosis and/or ophthalmoplegia. The absence of clinical improvement within 24 h after the administration of intravenous antibiotics or the clinical suspicion of central nervous system involvement is an indication for urgent computed tomography (CT).

Although CT still remains the modality of choice for the diagnostic workup of orbital infection in the acute stage, magnetic resonance imaging (MRI) should be considered particularly in the paediatric population because of the radiation to the eyes.

Nowadays, multidetector row CT is used in most institutions. We are currently using a slice thickness of 0.75 mm and an incremental factor of 0.4 for CT of the

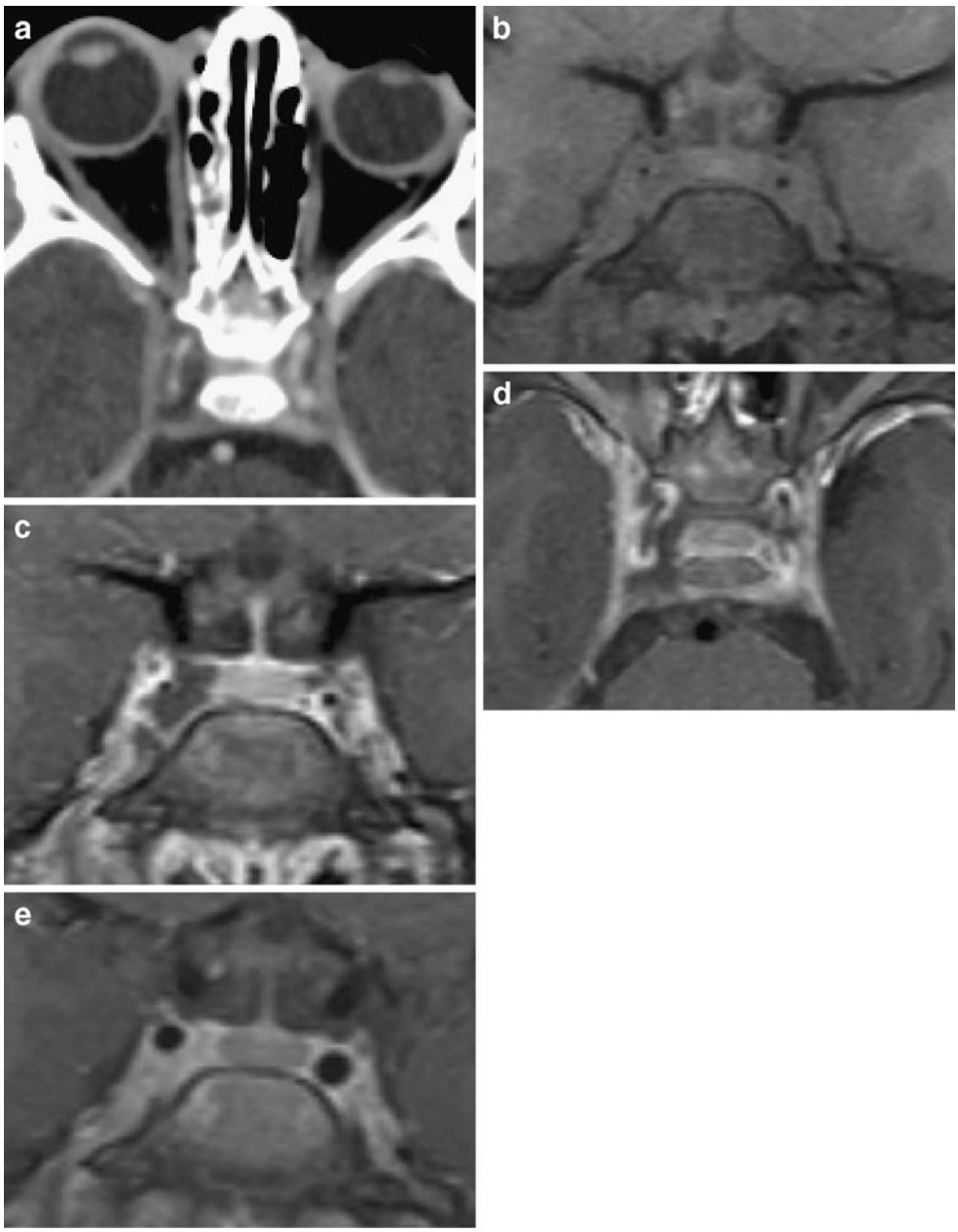

Fig. 5 Cavernous sinus thrombophlebitis. A 5-year-old boy with right otitis media and meningitis presenting with right-sided ophthalmoplegia. Axial contrast-enhanced CT (**a**), coronal T1-weighted pre-contrast (**b**) and coronal/axial post-contrast MRI results (**c**, **d**). Nearly complete thrombosis is seen in the right cavernous sinus and partial thrombosis in the left cavernous sinus. Note the narrowing of the cavernous portion of the carotid arteries. Follow-up coronal T1-weighted post-contrast MRI result shows nearly complete normalization of the cavernous sinus (**e**)

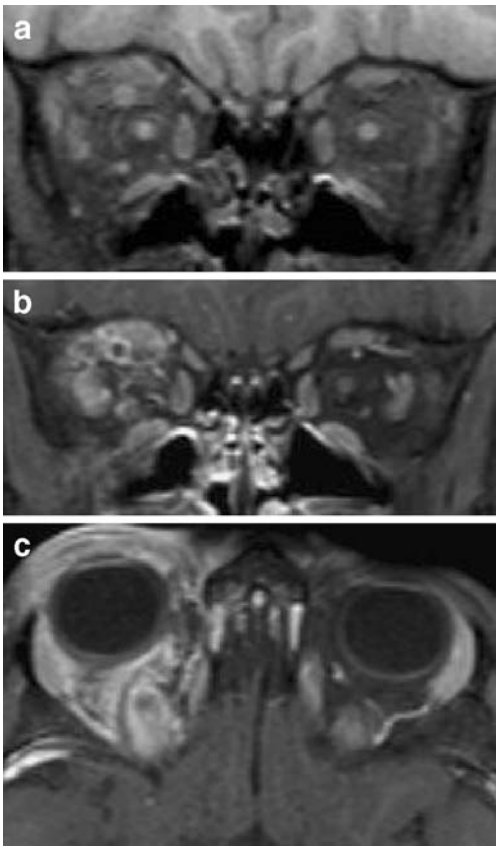

Fig. 6 Superior ophthalmic vein thrombosis. A 52-year-old woman with painful proptosis. Coronal T1-weighted pre-contrast (**a**) and coronal/axial (**b**, **c**) post-contrast MRI scans showing infiltration surrounding the right superior ophthalmic vein and infiltration of the orbital fat

orbits. Thin sections are required in order for us to be able to detect subtle changes, e.g. discrete wall thickening of the globe or beginning infiltration in the orbital apex.

CT and CT angiography certainly can be used for assessing the cavernous sinus.

MRI is the preferred imaging modality for a more detailed assessment of the orbits, anterior visual pathway and cavernous sinuses when an inflammatory disease is suspected. Following a routine brain MRI, we use axial and coronal 3-mm fat-saturated T1-weighted images before and after gadolinium enhancement and 2-mm axial T2-weighted images of the orbit.

For the investigation of the optic neuritis and chiasmitis, we recommend the use of a coronal double-echo (DE) short-tau inversion recovery (STIR) sequence, perpendicular to the course of the optic nerve.

Whenever a systemic pathology is suspected, brain MRI should be performed.

Discussion

Orbital infection

Sinusitis plays an important role in the development of orbital infections. Sixty percent of the infections develop from the direct spread of sinusitis and they are potentially devastating infections that can quickly result in blindness, meningitis or death. The major pathogens include *Staphylococcus aureus*, B-haemolytic *Streptococci* and *Haemophilus influenzae* type B [3–5].

Orbital infections develop via direct inoculation, extension from adjacent structures or haematogenous spread [6–8]. Complications of acute bacterial sinusitis are related to local extension into the central nervous system (meningitis), orbit of the eye (orbital cellulitis) and periorbital tissues (osteitis of the sinus bones) [9]. It can affect all age groups but is more common in children [10].

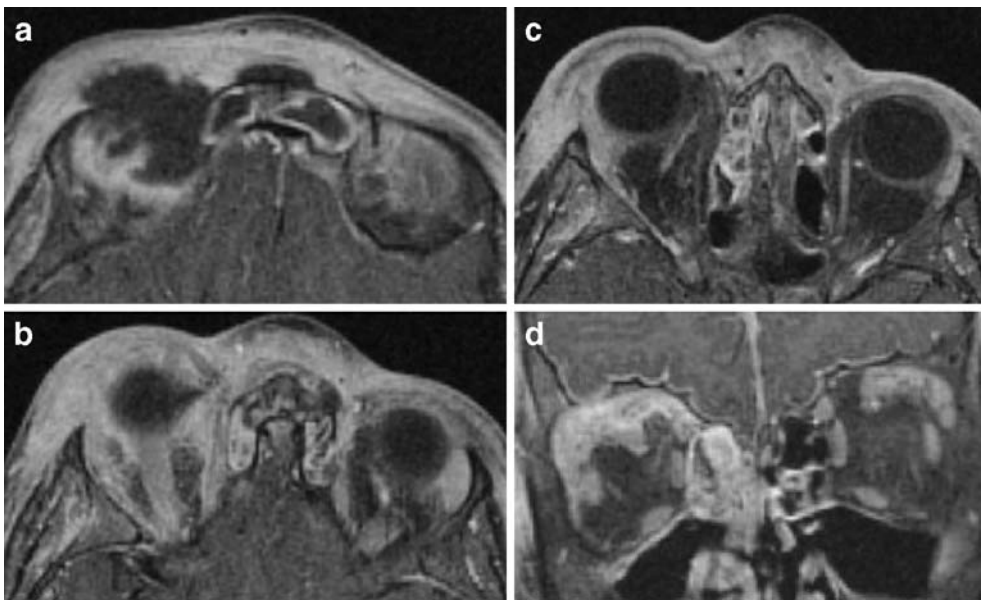

Fig. 7 Pott's puffy tumour. A 13-year-old boy with fever, headache and swelling of the eyes. Axial (**a**–**c**) and coronal (**d**) post-contrast T1-weighted images show the thickening of the pre-septal soft tissues on the right side with orbital extension. There is a breakthrough of the frontal sinusitis into the orbit

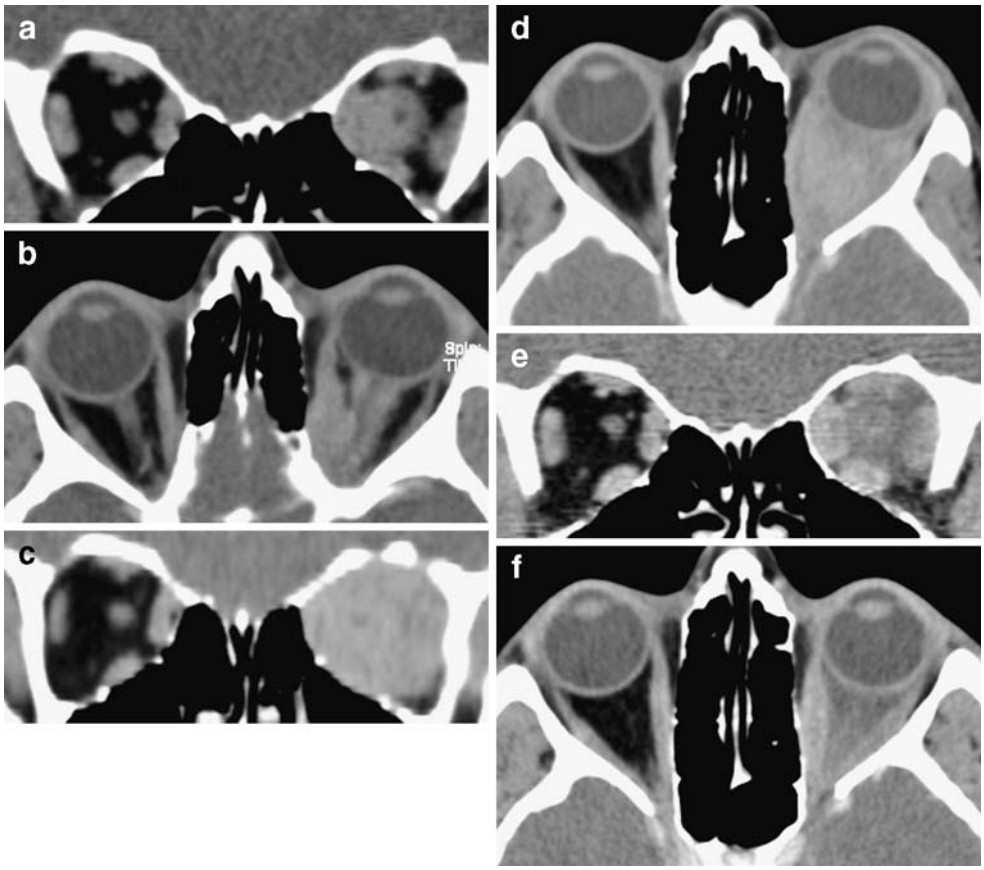

Fig. 8 Idiopathic orbital inflammatory syndrome. A 29-year-old woman with painful proptosis on the left side. Pathology revealed lymphoid nodules but no evidence of lymphoma. Coronal and axial contrast-enhanced CT on initial presentation (**a**, **b**), during clinical progressive symptomatology and prior to biopsy and treatment (**c**, **d**) and following treatment with radiotherapy (**e**, **f**). Note the diffuse scleral thickening and the hyperdensity of the retrobulbar fat

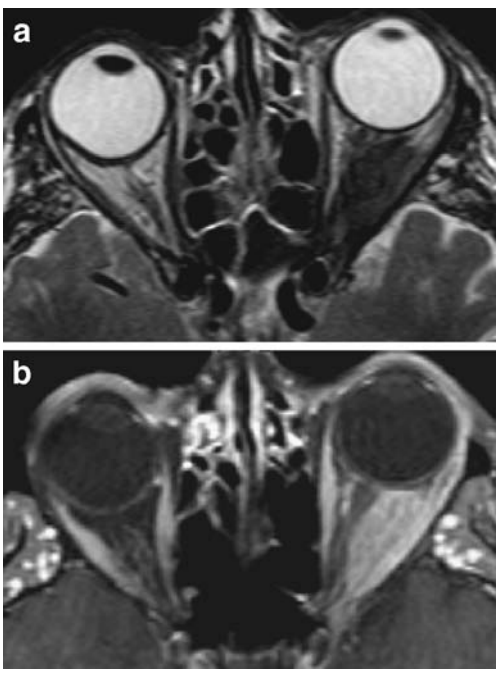

Fig. 9 Mucosa-associated lymphoid tissue lymphoma. A 74-year-old woman with swollen lower eye lid and ptosis of the upper eye lid. Axial T2-weighted (**a**) and contrast-enhanced T1-weighted (**b**) images show an intraconal lesion in the left orbit, displacing the optic nerve

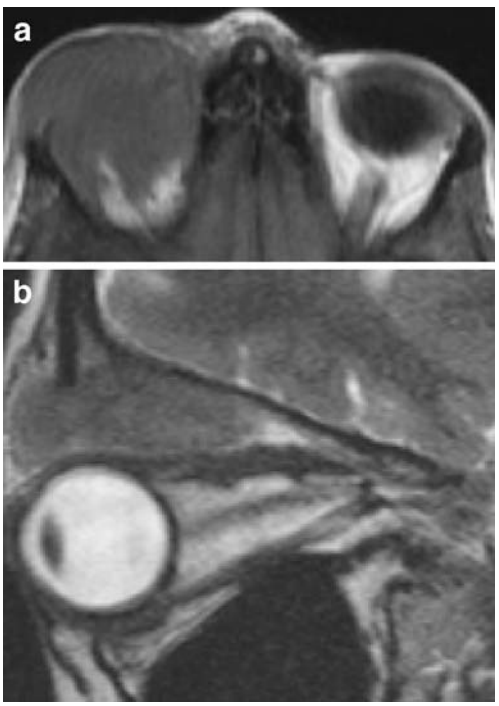

Fig. 10 Chronic myeloid leukaemia. A 32-year-old pregnant woman with proptosis. Axial T1-weighted (**a**) and sagittal T2-weighted (**b**) MRI scans show a soft tissue mass in the right orbital roof

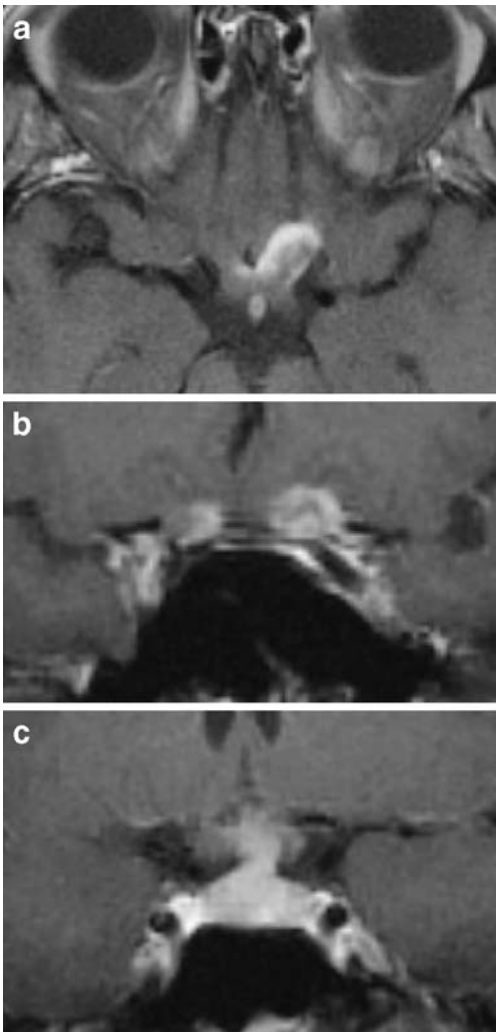

Fig. 11 Optic nerve neurosarcoidosis. A 28-year old woman with loss of vision and hypothalamic–pituitary axis dysfunction. Axial (**a**) and coronal (**b**, **c**) T1-weighted post-contrast (**a**) images show involvement of the pituitary stalk, optic chiasm and intracranial portion of the optic nerves

Orbital infections are classified as pre-septal cellulitis, orbital cellulitis, subperiosteal abscess, orbital abscess and finally cavernous sinus thrombosis/thrombophlebitis, known as the Chandler classification [8, 11, 12]. The entities are not necessarily seen in this ranking.

Pre-septal or periorbital cellulitis is an inflammatory edema of the eyelids and periorbital skin without extension into the orbit (Fig. 1). It is characterized by acute eyelid edema, tenderness and chemosis and is most commonly seen following a dacryocystitis, sinusitis/upper respiratory tract infection or trauma (including insect bites) [5].

Because the facial veins are valveless, pre-septal cellulitis may spread posteriorly to produce an orbital infection [7, 8]. It is important to distinguish pre-septal cellulitis from orbital cellulitis because the latter form must be carefully monitored and early intervention is necessary to prevent serious complications [11]. The treatment of pre-septal cellulitis consists of broad-spectrum oral antibiotics. Children under 3 years of age are often hospitalized for intravenous administration. CT should be performed whenever there is no improvement within the first 36–48 h.

Post-septal/orbital cellulitis is an infection of the soft tissue of the orbit without abscess formation and is far more frequent in children (Fig. 2). Patients with this infection may develop orbital signs and symptoms as vision loss, decreased eye motility, globe displacement and proptosis. Orbital cellulitis is best diagnosed by CT and is almost always associated with sinusitis. Typically, a diffuse infiltration of the orbital fat will be seen with increased density. Orbital cellulitis, in the presence of orbital signs, is treated by intravenous administration of antibiotics.

Subperiosteal abscesses are collections of purulent material between the orbital bony wall and periosteum (Fig. 3). This infection may develop from orbital cellulitis or directly from spread of an adjacent infection [5]. This diagnosis is confirmed by CT showing a swelling, often along the medial wall of the orbit, with displacement of the extra-ocular eye muscle and occasionally traction on the optic nerve. Limitations of ocular motility and directional proptosis may be present from the intra-orbital mass effect and entrapment of the extra-ocular muscles [7, 8]. If the

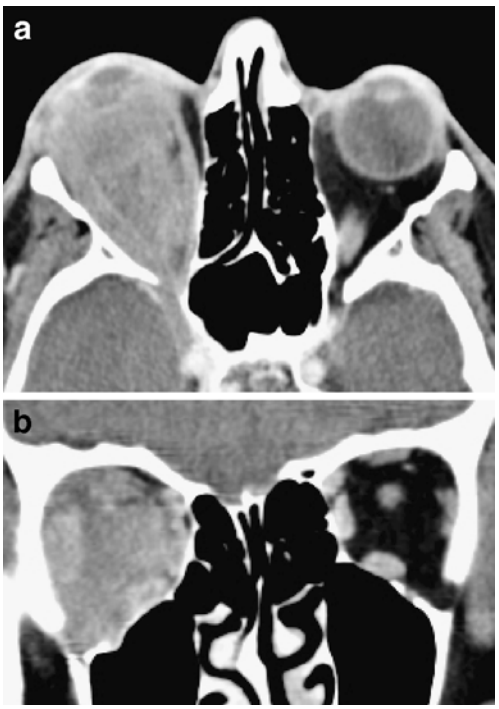

Fig. 12 Wegener granulomatosis. A 42-year-old man with a history of recurrent retinal detachments presenting with painful swelling of the periocular soft tissues. Axial and coronal contrast-enhanced CT (**a**, **b**) in a patient with extensive orbital involvement and invasion of the eye on the right side

patient does not show improvement within 24 h, a repeat CT scan is recommended [6, 11].

Orbital abscesses are collections of pus within the orbital soft tissue (Fig. 4). The physical signs include severe exophthalmos and chemosis, with complete ophthalmoplegia, visual impairment, as well as venous engorgement or papilledema on funduscopic examination [6–8]. CT is necessary to delineate the abscess. Intracranial involvement should always be suspected and ruled out [10].

Surgical treatment is necessary whenever there is a well-defined abscess, when vision is compromised or when there is complete ophthalmoplegia.

Extra-orbital extension is the last stage of orbital infections. Possible intracranial complications consist of an intracranial abscess, meningitis or cavernous sinus thrombosis [11]. Cavernous sinus thrombosis/thrombophlebitis is an infectious process of the cavernous sinus. Infectious thrombosis is most commonly spread from the orbit via the valveless orbital veins into the cavernous sinus [7, 8] (Fig. 5). Since the cavernous sinus is connected through the midline, thrombosis is frequently bilateral. Complete ophthalmoplegia often occurs when thrombosis of the sinus causes palsy of cranial nerves III, IV, V1 and VI and the sympathetic fibres. Cavernous sinus thrombosis can occur from any stage of orbital infections [7, 8]. Both

Fig. 14 Myositis. A 27-year-old woman with painful periorbital swelling and painful eye movements. Coronal contrast-enhanced CT shows thickening of the extra-ocular superior rectus muscle on the left side

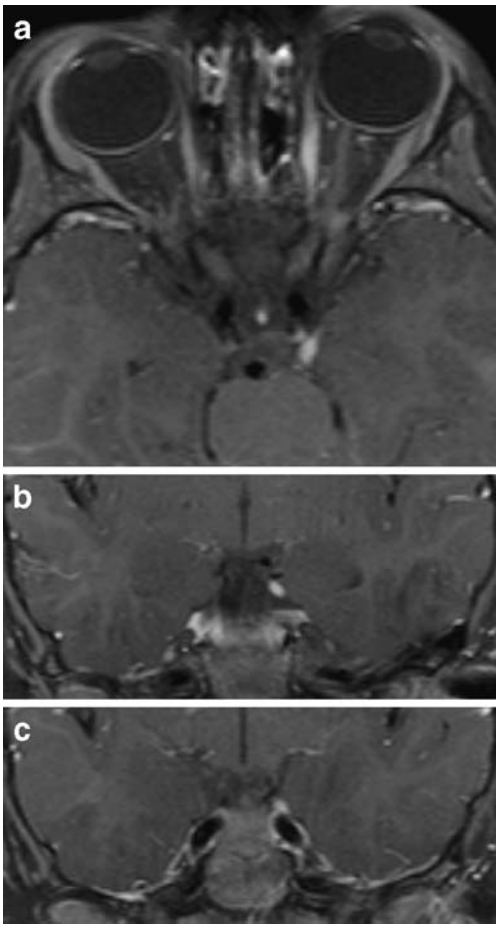

Fig. 13 Third nerve neuritis. A 29-year-old woman with left-sided third nerve paresis. Axial and coronal contrast-enhanced T1-weighted images show tickening and enhancement of the left oculomotor nerve (**a**–**c**). Note that the normal right oculomotor nerve is barely visible

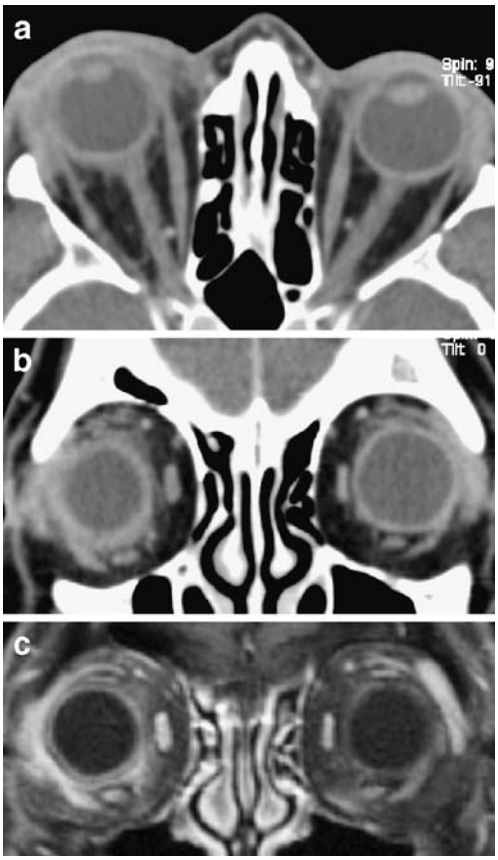

Fig. 15 Posterior scleritis. A 70-year-old woman with a systemic disease (recurrent polyarthritis and scleritis) presenting with a painful red eye. Axial and coronal contrast-enhanced CT (**a**, **b**) and coronal T1-weighted contrast-enhanced MRI results (**c**) show uveoscleral thickening on the right side with thickened Tenon's capsule. There is infiltration in the adjacent fat and in the lacrimal gland

CT and MRI can confirm the clinical suspicion. It is necessary to obtain detailed imaging of the cavernous sinuses before and after the injection of a contrast agent.

Infectious signs can also remain limited to the superior ophthalmic vein (Fig. 6) and can be considered a possible precursor of cavernous sinus thrombosis. The differential diagnosis with orbital cellulitis can be difficult. Neuroimaging certainly plays a role in this difficult clinical setting.

Pott's puffy tumour (or osteomyelitis of the frontal bone) is an extremely rare and potentially life-threatening complication of frontal sinusitis with possible extension into the orbit and formation of subperiosteal abscess (Fig. 7). First described by Percival Pott in 1760, Pott's puffy tumour manifests as a progressive headache, soft tissue swelling, fever and nasal symptoms [13, 14]. Potential complications include meningitis, superior sagittal sinus thrombosis and epidural, subdural or cerebral abscesses [14–16].

The mortality rate from these complications is significant despite antibiotic therapy and has been estimated as between 5% and 10% [13].

The treatment involves a combined surgical and antibiotic approach to prevent further suppurative complications [9].

Orbital inflammation

Idiopathic orbital inflammatory syndrome (orbital pseudotumour) can present either acutely or chronically and may involve the retrobulbar region and various orbital structures (Fig. 8). Localized orbital inflammation may affect the eye, lacrimal gland, extra-ocular muscle or optic nerve sheath. Usually, there is no identifiable causative agent. This type of inflammation is most common in adulthood and may account for 5% to 15% of all orbital lesions. Possible associated systemic inflammatory diseases include autoimmune thyroid disease, sarcoidosis, Wegener's granulomatosis, Crohn's disease, systemic lupus erythematosus, Erdheim–Chester and Langerhans cell histiocytosis [17]. Finally, orbital inflammation can also be due to an associated pathology such as neoplasm or trauma.

The acute form is painful and is characterized by swelling and proptosis while the chronic form evolves over several weeks and lacks inflammatory signs. There is usually no fever and no leukocytosis. On imaging, a retrobulbar infiltration is observed affecting one or more orbital structures and ranging from subtle changes to an almost complete invasion of the orbit. On pathology, a non-specific infiltrate of lymphocytes, plasma cells and macrophages is seen [18]. The differential diagnosis with orbital cellulitis, thyroid eye disease and lymphoma can be difficult.

Chronic orbital inflammatory disease is difficult to diagnose and may require a biopsy.

Idiopathic sclerosing orbital inflammation is a rare, chronic and progressive syndrome, characterized as fibrosclerosis on histopathology [19].

In a recent review on lymphoproliferative diseases of the orbit, the authors found a majority of mucosa-associated lymphoid tissue lymphoma (75%) [20]. The lesions tend to be limited to a local infiltration that responds well to local treatment and carries a better prognosis. Lymphoid hyperplasia was found in approximately 9%, diffuse large B-cell lymphoma in approximately 5% and mantle cell lymphoma in 3%. Most lesions were found in the conjunctiva (41%), followed by the orbit (33%) and the eyelid (26%). On imaging, a lymphoid infiltration is difficult to differentiate from lymphoma and leukaemia (Fig. 9).

Orbital leukaemic tumours are often a manifestation of acute myeloid leukaemia and usually present in the first decade of life [21]. They are thought to originate in the bone marrow and spread via Haversian canals to penetrate the periosteum. They are also called granulocytic sarcoma or chloroma, because of the green aspect due to the pigmented enzyme myeloperoxidase. On imaging, a homogeneous mass is seen, moulding the bone and often involving the lateral wall of the orbit (Fig. 10). On T1-weighted images they tend to be isointense to muscle, while on T2-weighted imaging they return a signal similar to that of white matter.

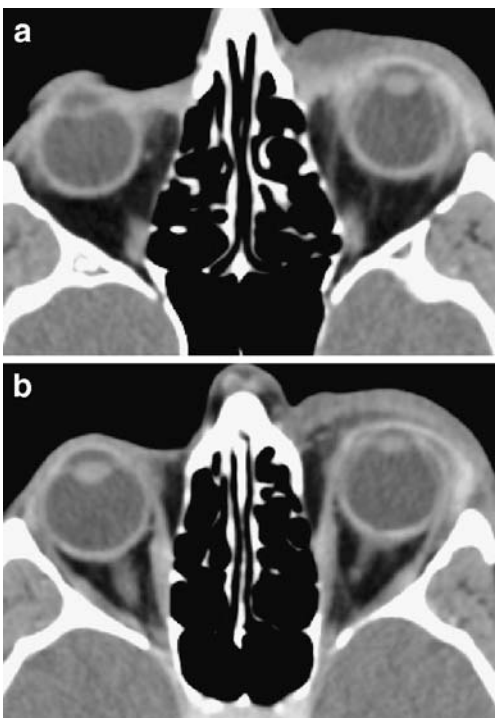

Fig. 16 Choroidal detachment. A 60-year-old woman with painful proptosis, chemosis and limited eye motility. Axial contrast-enhanced CT (**a**, **b**) demonstrates the choroidal detachment in the left eye in a patient with posterior scleritis. Note the thickening of the pre-septal tissues on the left side

In sarcoidosis uveitis, optic neuropathy, involvement of the lacrimal gland, extra-ocular muscle or orbital fat can be observed in up to 50% of the patients (Fig. 11) [22]. The role of imaging consists of defining pathological involvement and identifying associated systemic disease. The treatment consists of steroids but some chronic forms may not be responsive and may require radiation therapy or immune modulation, especially in the chronic forms of orbital inflammation. On imaging, an infiltration of the orbital fat is seen, occasionally limited to the orbital apex but often involving the orbit and occasionally the extra-ocular muscles and lacrimal glands. It can be associated with uveoscleral thickening and enhancement of Tenon's space. Optic nerve involvement by sarcoidosis can appear similar to optic nerve glioma [23].

Wegener's granulomatosis is characterized by vasculitis and necrotizing granulomas. Scleritis, uveitis and neuritis can be seen and are likely due to contiguous spread from the paranasal sinuses. The disease can fill the entire orbit and osseous destruction can occasionally be observed (Fig. 12).

Ophthalmoplegic migraine can present as painful ophthalmoplegia and, on MRI, enhancement of the oculomotor nerve can be observed (Fig. 13) [24]. Isolated enhancement of the oculomotor nerve has been reported in many different entities, e.g. diabetic neuropathy, neurosarcoidosis, Lyme disease and infectious mononucleosis.

If one or more extra-ocular muscles are involved, the term myositis is preferred (Fig. 14). The involvement of the muscle tendon and the irregular borders of the thickened muscle(s) is helpful in differentiating infectious myositis from Graves' ophthalmopathy.

Inflammatory changes of the eye and anterior visual pathway can be observed simultaneously or in association with pre-septal/orbital cellulitis.

Scleritis can be visualized on imaging and can be seen as an isolated inflammation or as part of a systemic disease. The sclera is one of the preferential sites of involvement in inflammatory orbital disease. In posterior scleritis, a thickening of the sclera will be visible with strong contrast uptake (Fig. 15). Choroidal detachment can occur when fluid accumulates between the sclera and Tenon's capsule (Fig. 16).

Uveitis can be observed in a large variety of diseases and can mimick posterior scleritis and even lymphomatous/leukaemic infiltration [25].

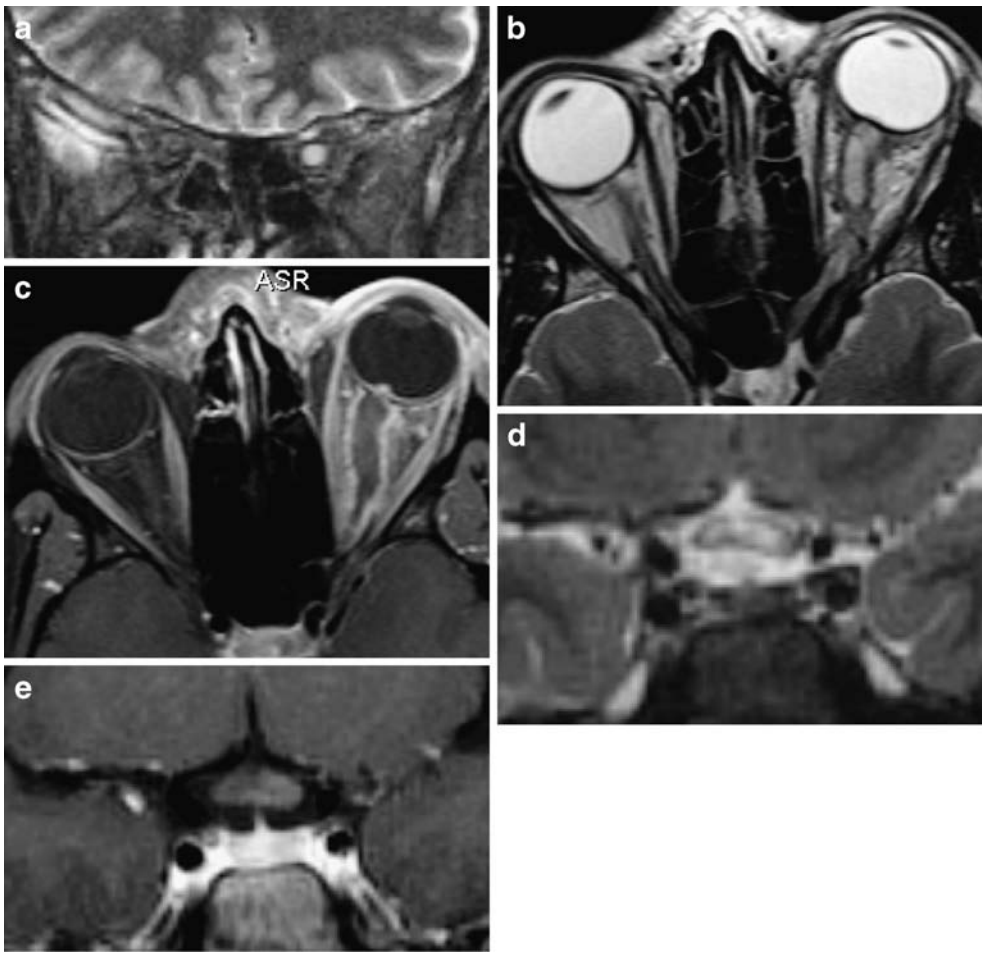

Fig. 17 Optic neuritis. Coronal T2-weighted DESTIR image (**a**) in a 21-year-old woman patient with MS and optic neuritis. A 23-year-old woman with pain and progressive loss of vision. Axial T2- and T1-weighted post-contrast (**b**, **c**) images of idiopathic optic perineuritis on the left side with diffuse infiltration of the orbital fat. A 12-year-old girl with psoriasis, recurrent optic neuritis and arthritis presenting with new episode of optic neuritis. Coronal T2- (**d**) and T1-weighted post-contrast (**e**) image in a child with recurrent optic neuritis on a probable auto-immune basis. The chiasm appears swollen and returns a high signal centrally. Peripheral enhancement is seen following administration of gadolinium

Idiopathic optic neuritis is characterized by unilateral subacute, often painful, visual loss without systemic or neurological symptoms. A relative afferent papillary defect is a sensitive and specific sign of optic neuritis. A reduced amplitude of the P100 component of the visual evoked potential is observed. There should be no evidence of vascular, infectious, toxic, metabolic or hereditary optic neuropathy. It is more common in women between 14 and 45 years of age [26]. In order to be classified as idiopathic, the clinical episode should peak within 4 weeks and there should be a recovery within 3 months after the onset of symptoms.

There is often an excellent visual recovery but the risk of developing multiple sclerosis (MS) exists. The presence of brain abnormalities at the time of the optic neuritis episode is a strong predictor of the 15-year risk [26]. In a series of 389 patients who were followed prospectively for 15 years, 25% of the patients without brain lesions developed MS while 72% of the patients with brain abnormalities developed MS [27].

It is also of interest to note the possible association between MS and Leber's hereditary optic neuropathy. Therefore, screening for Leber's mutations in MS patients with visual loss is recommended because of the prognostic and genetic implications. It has been suggested that these patients may have a characteristic appearance of lesions on brain MRI [28]. The lesions had indistinct margins, had a tubular appearance and were either larger or smaller than MS plaques.

In typical acute optic neuritis, imaging the orbit/optic nerve is not always necessary and contrast-enhanced images are certainly not indicated. Brain MRI should always be obtained in these patients to evaluate the risk of MS. MRI is abnormal in about 50% of patients with initial presentation of isolated optic neuritis. A normal brain MRI is thought to be associated with a better outcome. An effective treatment for optic neuritis has been shown to reduce the development of clinically definite MS [29].

On coronal STIR images, the affected optic nerve appears bright while the normal optic nerve appears black with the surrounding bright halo representing the CSF within the dura (Fig. 17a)

The differential diagnosis with anterior ischemic optic neuropathy is very difficult both clinically and radiologically. A retrospective study on 64 patients with either optic neuritis or anterior ischemic optic neuropathy revealed abnormalities in the optic nerve on MRI in respectively 97% and 16% of the patients [30].

The simultaneous occurrence of optic neuritis and myelitis, neuromyelitis optica, can be distinguished from MS by determination of the biomarker, an auto-antibody that recognizes aquaporin 4 as its target [31].

Optic perineuritis is observed in older patients and tends to have sparing of central vision. On imaging,

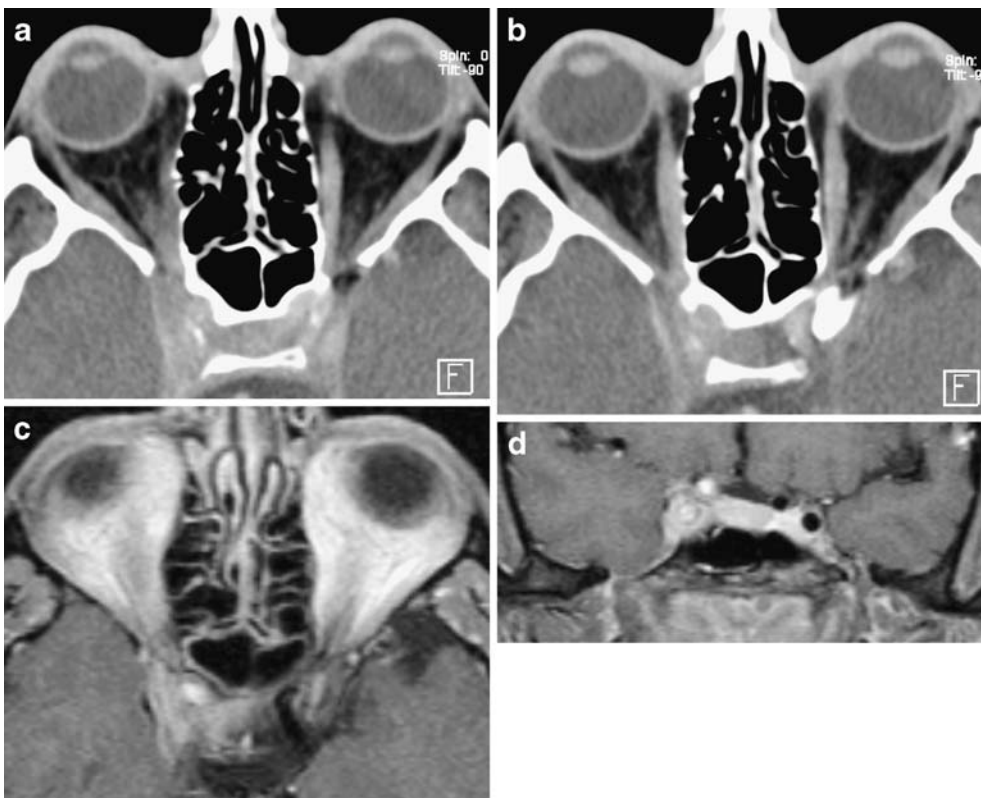

Fig. 18 Tolosa–Hunt syndrome. A 45-year-old man with headache and painful proptosis. Axial contrast-enhanced CT (**a**, **b**) show obliteration of the right orbital apex and thickening of the right cavernous sinus. Axial (**c**) and coronal (**d**) T1-weighted post-contrast MRI scans show the involvement of the cavernous sinus with thrombophlebitis and narrowing of the carotid artery

there is enhancement around the optic nerve (Fig. 17b, c) [32]. It is thought to be caused by an inflammatory condition of unknown aetiology. On pathology, lymphocytes, plasma cells and macrophages can be found. Corticosteroids are indicated and may result in complete healing but progression to fibrous encasement has also been described [33]. Occasionally, the differential diagnosis with optic nerve sheath meningioma can remain difficult [34].

Chiasmal optic neuritis can be the initial manifestation of MS but has also been reported in auto-immune demyelinating disorders and viral diseases (Fig. 17d, e) [35, 36].

Tolosa–Hunt syndrome is a non-specific inflammatory/granulomatous process involving the cavernous sinus and superior orbital fissure/orbital apex (Fig. 18) [37]. The International Headache Society established the following diagnostic criteria: one or more episodes of unilateral orbital pain persisting for weeks if untreated; paresis of one or more of cranial nerves III, IV and/or VI and/or demonstration of a granuloma by MRI or biopsy; and paresis coinciding with the onset of pain and paresis resolving within 72 h when treated with corticosteroids [38]. Sensory loss in the distribution of cranial nerve V, often the ophthalmic division, and in the optic nerve have been reported.

Neuroimaging plays an important role because of the danger of taking a biopsy. CT and MRI both can depict granulomatous tissue in the cavernous sinus with thickening of the wall of the intracavernous portion of the carotid artery and occasionally narrowing of the artery (Fig. 18). Extension into the superior orbital fissure is commonly seen.

Conclusion

A high degree of clinical suspicion together with the imaging findings is important in reaching a diagnosis of orbital inflammation and/or infection. The clinical presentation is often impressive and urgent imaging is usually requested. CT certainly continues to play an essential role in assessing the acute infectious conditions.

MRI is more appropriate in the diagnostic workup of a suspected inflammatory disease of the anterior visual pathway and/or orbit. The detection and precise location of the abnormalities is not always straightforward. The differential diagnosis between inflammatory diseases and lymphoproliferative disorders and tumours can remain difficult or even impossible. An appropriate MRI strategy is therefore essential to assess the complex anatomy of the orbit/cavernous sinus and to detect the sometimes subtle abnormalities.

Conflict of interest statement We declare that we have no conflict of interest.

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