Chapter 5

# Orbital Cellulitis

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Additional information is available at the end of the chapter

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**Abstract**

Infection in the soft tissues of the orbit, posterior to the orbital septum results in orbital cellulitis. This is a very serious condition, which may occur as a complication of sinusitis by contiguous spread or may result from haematogenous spread or from trauma. Orbital cellulitis presents with periorbital swelling, proptosis, conjunctival chemosis and injection, extraocular motility deficits and visual loss. It requires comanagement by the ophthalmologist and otorhinolaryngologist when secondary to sinusitis. It is important that this condition is recognized early, and immediate management is done to prevent impending visual loss and further complications of periosteal abscesses, meningitis, cavernous sinus thrombosis and death. This chapter reviews the epidemiology of orbital cellulitis, pathogenesis, causative organisms, investigations (including imaging of the sinuses) and treatment. Prognostic factors and conditions that complicate this such as diabetes will also be discussed.

**Keywords:** orbital, sinusitis, periorbital swelling, proptosis, brain abscess, meningitis

## 1. Introduction

Orbital cellulitis is the involvement of the orbital tissues behind the orbital septum with inflammation or infection. The orbital septum is an important dividing landmark, as infection and inflammation occurring anterior to it is called preseptal cellulitis, which is managed differently than that occurring posterior to it, orbital cellulitis.

Orbital cellulitis is an inflammatory process and is generally used to describe infectious inflammation [1]. The sinuses are closely associated with the orbit and are commonly the source of infection from direct contiguous spread. It is important that orbital cellulitis is diagnosed, investigated with imaging to determine if the source is from the sinuses and treatment (medical and/or surgical) commenced early to prevent serious complications,

including cerebral abscess and meningitis. Sinus surgery may be required for the treatment of orbital cellulitis secondary to sinusitis or pansinusitis. The management of this condition may, therefore, require a multidisciplinary team of the ophthalmologist, otolaryngologist, infection specialist and neurosurgeon.

## 2. Orbital anatomy

The bony orbit is a pear-shaped cavity which houses the eyeball with its adnexae (lacrimal gland) and orbital fat. The volume of the orbit is ~30 ml of which the eyeball takes up 6 ml (20%). The orbit is related superiorly to the frontal sinus, inferiorly to the maxillary sinus, medially the ethmoid sinus and anterior aspect of the sphenoid sinus.

The anterior border of the orbit is the **orbital septum**, which separates the lid from the orbit. The orbital septum, a fibrous tissue arises from the periosteum of the superior and inferior orbital rims, divides the plane of the inflammation or infection into preseptal or postseptal (orbital cellulitis) (**Figure 1**). Infection anterior to the orbital septum is called preseptal cellulitis and can be managed by oral antibiotics. However, when the infection is posterior to the orbital septum, it results in orbital cellulitis which is an ophthalmic emergency requiring in hospital treatment.

The orbit is bounded superiorly by the **roof** (the lesser wing of the sphenoid bone and orbital plate of the frontal bone), which is below the anterior cranial fossa and frontal sinus. The greater wing of the sphenoid and the zygomatic bone make up the **lateral wall** (**Figure 2**).

Three bones make the **floor of the orbit,** the zygomatic, maxillary and palatine. Blow out fractures commonly occur in the posterior medial aspect of the maxilla. The orbital floor is also the superior boundary of the maxillary sinus.

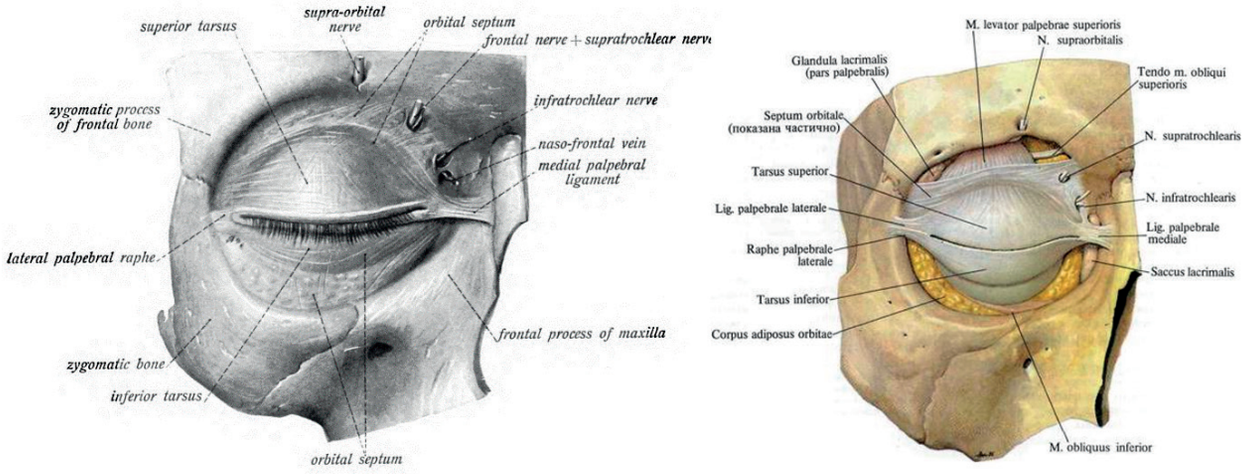


**Figure 1.** An anatomical illustration from the 1909 edition of Sobotta's Anatomy. https://commons.wikimedia.org/wiki/File:Sobo_1909_770.png#/media/File:Sobo_1909_770.png. Source: Riordan-Eva and Cunningham [2]. 

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Four bones make up the **medial wall**; maxillary (frontal process), lacrimal, ethmoid and sphenoid bone (**Figure 3**). The lamina papyracea, which forms part of the medial wall, is paper-thin and perforated by numerous foramina for nerves and blood vessels, which makes easy contiguous spread from the ethmoid sinuses to the orbit in the spread of orbital cellulitis.

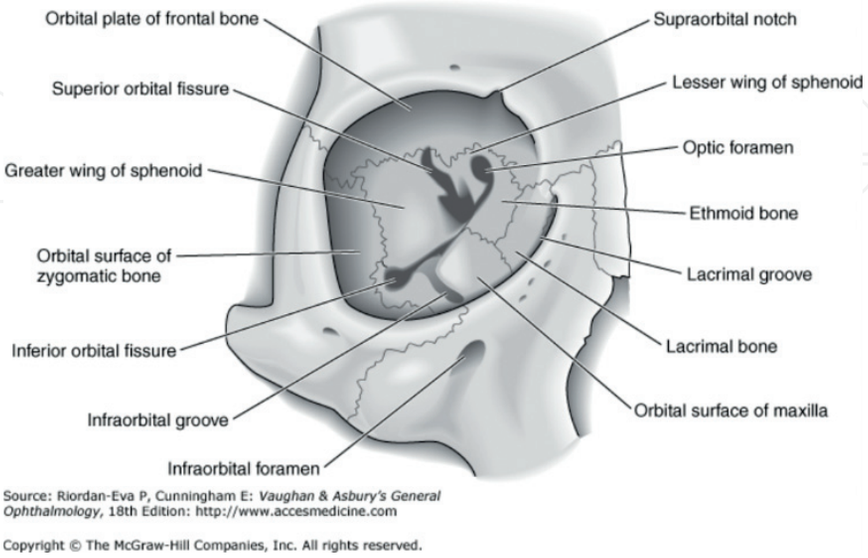


**Figure 2.** Anterior view of bones of right orbit. Source: Riordan-Eva and Cunningham [2]. Copyright © 2018 McGraw-Hill Education. All rights reserved.

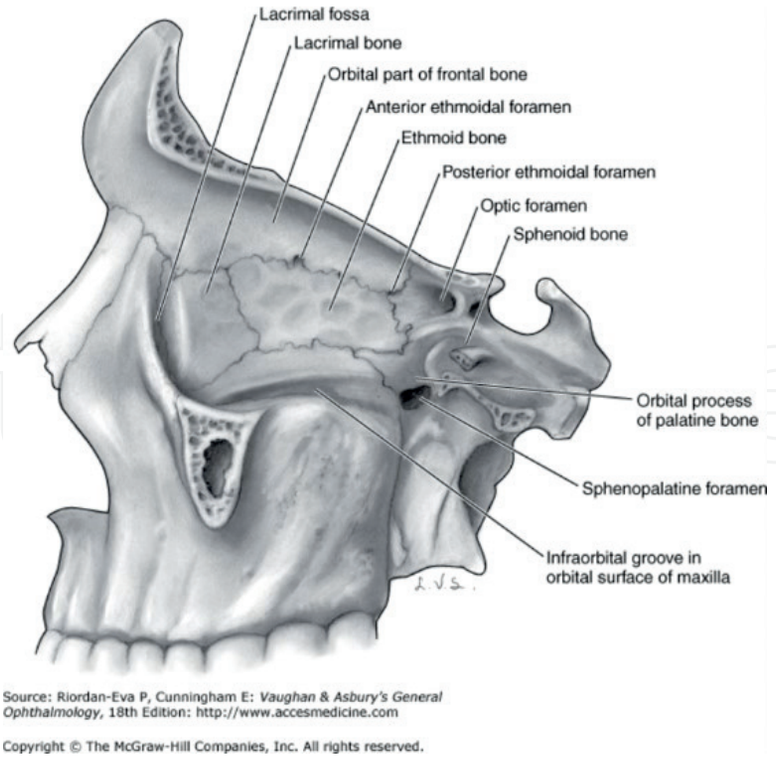


**Figure 3.** Medial wall of the orbit. Source: Riordan-Eva and Cunningham [2]. Copyright © 2018 McGraw-Hill Education. All rights reserved.

The **superior orbital fissure** is between the greater and lesser wings of the sphenoid and allows communication between the cranium and the orbit (**Figure 2**). This fissure is divided into the superior and inferior portion. The superior portion transmits the superior ophthalmic vein, lacrimal, frontal and trochlear nerves. The inferior portion transmits the abducens nerve, superior and inferior divisions of the oculomotor nerve and the sympathetic fibres from the cavernous plexus. Inflammation of the superior orbital fissure and orbital apex is called Tolosa-Hunt syndrome.

The **inferior orbital fissure** is located between the greater wing of the sphenoid and the maxillary bone, which divides the lateral orbital wall from the orbital floor (**Figure 2**). It connects the pterygopalatine and infratemporal fossae with the orbit and transmits the maxillary and zygomatic nerves in addition to the branches of the inferior ophthalmic vein.

The lesser wing of the sphenoid has the **optic foramen** through which the optic nerve and ophthalmic artery is transmitted from the middle cranial fossa to the orbit.

### 2.1. Epidemiology of orbital cellulitis

Orbital cellulitis may occur at all age groups but is more commonly seen in children. The incidence in children is 1.6 per 100,000 compared to adults 0.1 per 100,000 [3]. Gender distribution is usually equal; however, the males predominate in some countries because of work-related injuries as in India and Nigeria [1]. Orbital cellulitis has its peak incidence in winter and early spring [1, 4] and is least frequent (19.4%) in the summer months [5].

In the western countries, patients have an average duration of symptoms for 4.4 days and an average hospital stay of 5.8–6.2 days compared with developing countries, where the average symptom duration is 5.2–10.6 days, prior to presentation and have a longer average hospital stay of 9–13.7 days [1]. In developing countries, late presentation results in poor prognosis [6].

### 2.2. Pathogenesis of orbital cellulitis

Orbital cellulitis may result from direct contiguous spread (e.g. sinuses or dental), exogenous (e.g. trauma or surgery) and endogenous (haematogenous). Orbital cellulitis is unilateral in greater than 90% of cases [7]. Most cases of orbital cellulitis result from the extension of infection from the paranasal sinuses [1, 6]. Approximately, 1.3–5.6% of sinusitis results in orbital cellulitis and 80% of all complications of acute rhinosinusitis are orbital (**Table 1**) [4, 10–12].

The ethmoid sinuses are the most frequent source of infection in 43–100% of cases [1]. This may be due to the thin medial orbital wall. Other predisposing factors for the orbital spread include lack of lymphatics and valveless veins of the orbit and foramina of the orbital bones. Childhood orbital cellulitis may involve more than one sinus in 15.7–38% of cases, whereas in adults the multiple sinus involvement was <11%.

Upper respiratory tract infections are a major cause of orbital cellulitis and can reflect the seasonal distribution of the disease [1, 4]. Contiguous spread may also occur from endophthalmitis, pan ophthalmitis, dental abscesses and extension from preseptal cellulitis. Dental infections can result

| Group | Chandler et al. | Moloney et al. |
|---|---|---|
| I | Preseptal cellulitis | Preseptal cellulitis |
| II | Orbital cellulitis | Subperiosteal abscess |
| III | Subperiosteal abscess | Orbital cellulitis |
| IV | Orbital abscess | Orbital abscess |
| V | Cavernous sinus thrombosis | Cavernous sinus thrombosis |

**Table 1.** Classification of the complications of sinusitis by Chandler et al. [8] and modification by Moloney [9].

in odontogenic orbital cellulitis with spread through the maxillary sinus. Haematogenous spread from a bacteraemia may occur and a bilateral orbital cellulitis has been reported in a case of infective endocarditis [13].

Trauma is a predisposing factor, which may be a direct penetrating injury or orbital fractures. Orbital cellulitis may occur from direct spread from the sinuses as seen in trauma resulting in a blow-out fracture of the orbit. Orbital foreign bodies can be metallic or organic, with the latter (e.g. wood) containing significant bacteria [14]. Less commonly it has been reported after surgery usually with the use of an explant such as aqueous drainage device (glaucoma surgery) or silicone scleral sponges (retinal detachment repair) [15, 16].

### *2.2.1. Microorganisms*

*Staphylococcus aureus* and *Streptococcus* species are the most common causative organisms [1]. There are increasing cases of methicillin resistant *Staphylococcus aureus* (MRSA). *Streptococcus pneumoniae* is seen more commonly in younger children and *Group A Streptococcus* in older children. In one study from Scotland, *Streptococcus* (66%) and *Haemophilus* (46%) were the most common pathogens in children [3]. Less commonly coagulase-negative *Staphylococcus, Haemophilus influenza, Klebsiella pneumoniae, Moraxella catarrhalis* and other respiratory tract anaerobes were implicated [17]. *H. Influenzae* used to be a common pathogen; however, this has significantly reduced after the introduction of the *H. influenzae* vaccine [17–19].

Post traumatic cases are usually due to *S. aureus* and *S. pyogenes. Streptococcus* infections can lead to a necrotizing lid disease and necrotizing fasciitis [1]. Trauma with penetration of organic foreign body may have *Staphylococcus epidermidis, Staphylococcus aureus, Enterobacter agglomerans and Clostridium perfringens* as the offending organisms [14]. Odontogenic infections resulting in orbital cellulitis usually have mixed aerobic and anaerobic bacteria.

Fungal infections are usually seen in high risk patients such as diabetes mellitus, immunocompromised patients, patients on chronic steroids or antibiotic treatment. Mucormycosis and aspergillosis are the most common types. Fungi can invade blood vessel walls causing a thrombosing vasculitis. This can cause significant severe complications of ophthalmic vascular thrombosis, cavernous sinus thrombosis, meningoencephalitis, brain abscess and ultimately a high mortality rate [1].

### 2.3. Clinical features

Orbital cellulitis presents with periorbital oedema and cellulitis of the eyelids, ptosis, red eye with conjunctival chemosis and pain on eye movements. As it worsens proptosis, reduced vision and double vision occur due to limitation of extraocular movements with the orbital swelling (**Figure 4**). Most common symptoms are reduction in vision (66.6%), proptosis and ptosis (33.3%) [12]. Patients may also experience pain on eye movements due to inflammation of the extraocular muscles. Patients may give a history of sinusitis or an upper respiratory tract infection.

Constitutional signs such as fever, malaise, loss of appetite are usually present. Headache may occur in 10% of patients. Children are more likely to have a fever and a higher leukocytosis [7]. Children less than a year may present with fever, periorbital edema and erythema with reduced appetite and lethargy [1]. Patients between 1 and 7 years old are less likely to have proptosis and ophthalmoplegia compared to older children and adults.

Clinical signs include proptosis, limitation of extraocular movements, reduced vision, a relative afferent pupillary defect (RAPD) and impaired colour vision. Compression of the central retinal artery can compromise vision from optic nerve ischemia with resulting infarction of the sclera, choroid and retina [7]. Secondary inflammation can result in iridocyclitis, vitritis and septic pan ophthalmitis. Increase in the intraocular pressure, glaucoma, due to the increased congestion of the episcleral vessels can further reduce the vision.

This patient requires urgent ophthalmic admission, imaging, commencement of intravenous (IV) antibiotics and monitoring. Colour vision and pupillary reactions should be monitored every 4–6 h, in addition to the proptosis with the Hertel exophthalmometer.

Increased inflammation and congestion in the orbit can result in an orbital apex syndrome. The pupillary reactions should be monitored for a RAPD which suggests an optic neuropathy secondary to an orbital apex syndrome. Patients with a RAPD, elevated intraocular pressures and complete ophthalmoplegia can develop permanent loss of vision in that eye if orbital pressure is not relieved urgently.

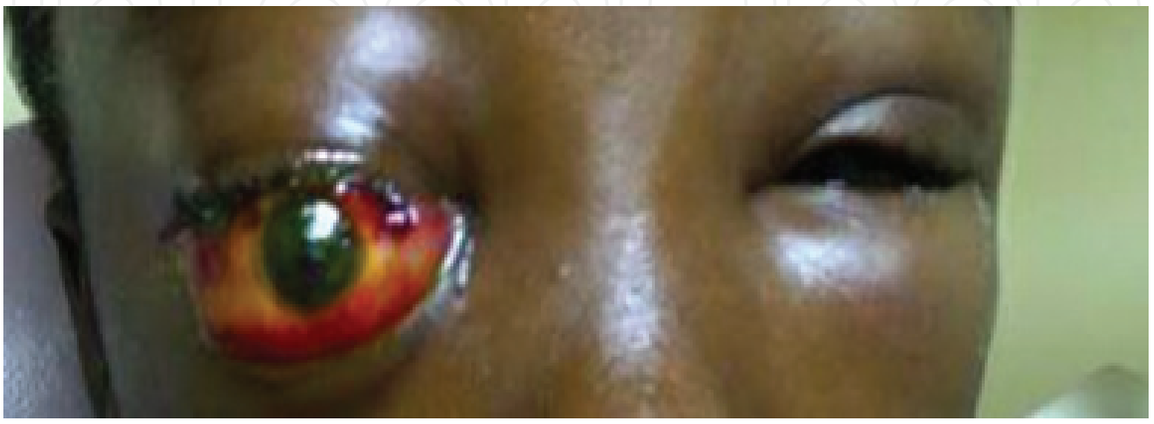

**Figure 4.** Right orbital cellulitis with periorbital swelling and proptosis.

Odontogenic orbital cellulitis can rapidly progress and cause blindness from a severe tense orbit, with resulting central retinal artery occlusion and ischemic optic neuropathy [20]. With increasing proptosis, corneal ulceration from exposure keratopathy may occur.

### *2.3.1. Special clinical scenarios*

#### *2.3.1.1. Fungal orbital cellulitis*

This condition can be caused by the fungus of the order *Mucorales*, usually found in the soil among decaying organic matter [21]. *Rhizopus, Rhizomucor and Mucor* species are most common and are related to pathogenesis of vascular invasion with resulting thrombosis and necrosis of tissues. Elevated levels of iron and glucose in the serum are a predisposing factor [21]. Aspergillosis, caused by the fungus *Eurotiales*, genus *Aspergillus* can be non-invasive or invasive and is associated with vascular involvement and bony erosion, initially affecting the sinuses then spreading to the orbit.

Diagnosis is usually done by biopsy or culture and requires aggressive treatment with intravenous antifungals and may require orbital exenteration. Cases of non-invasive aspergillosis do not require orbital exenteration. Invasive aspergillosis has been reported to masquerade as giant cell arteritis, with symptoms of jaw claudication, scalp tenderness and weight loss [21]. The initial onset of fungal orbital cellulitis can be insidious then rapidly progressive, so a high index of suspicion must be present.

#### *2.3.1.2. Rhino-orbital mucormycosis*

This is a rare opportunistic infection caused by the fungi "Mucoraceae." This usually occurs in patients with immunosuppression and diabetic ketoacidosis but has been seen in patients with myelodysplastic syndrome, chronic hepatitis C infection, polysubstance abuse, alcoholic liver cirrhosis and Crohn's disease with systemic immunosuppression [21]. Uncontrolled diabetic ketoacidosis is the most commonly associated condition in orbital mucormycosis.

Patients may present with gradual onset of facial and periorbital swelling, double vision and loss of vision. Septic necrosis can cause black eschar from ischemic infarction on the palate, turbinates, skin and eye lids. Complications can result in retinal vascular occlusion, cranial palsies and cerebrovascular occlusion. The onset may be insidious in immunosuppressed patients and a high index of suspicion must be present to prevent the delay in diagnosis and treatment.

Fungal infections can be acquired from inhalation of spores with resulting upper respiratory tract infection then sinus involvement, orbital cellulitis with contiguous spread to the brain. Mucormycosis initially involves the maxillary and ethmoid sinus, thereafter spreading to the orbit and brain. The severity of the hyphae invasion of blood vessels results in an occlusive vasculitis with ischemia and infarction of orbital tissues, which can eventually become fatal.

Fungal orbital cellulitis from aspergillosis has been reported in a patient with myelodysplastic syndrome and portal hypertension, with the initial presentation mimicking giant cell arteritis [21]. Gradual onset of periorbital and facial swelling, diplopia and visual loss may occur. Black

eschar results from ischemic infarction and septic necrosis of the palates, turbinates, nasal septum and eyelids and may present with ophthalmoplegia. The progression is slower than bacterial orbital cellulitis.

High risk of mortality is associated with bilateral orbital involvement, diabetes, renal transplantation with immunosuppression, leukaemia and hemiparesis [21]. A rare case of spread from a fungal nasal septal abscess *Scedosporium apiospermum* resulting in an orbital apex syndrome has been reported in a diabetic patient, which resulted in blindness [22]. Mucormycosis in immunocompetent patients is rare, and there has only been one reported case of zygomycetes infection in an immunocompetent child [23].

*2.3.1.3. Allergic aspergillosis sinusitis*

Allergic aspergillosis sinusitis occurs in immunocompetent patients who have nasal polyposis and chronic sinusitis. About 17% of allergic fungal sinusitis will present as orbital cellulitis. Patients will have an eosinophilia, with thick mucin in the sinuses on CT scans. Sinus biopsy reveals peanut butter like mucus with eosinophils and extra-mucosal fungal hyphae. Endoscopic debridement of the sinuses, treatment with corticosteroids is recommended [24].

*2.3.2. Investigations (bloods and swabs)*

Patients with orbital cellulitis require in hospital management. Blood investigations include full blood count. The leukocytosis is usually over 15,000 cells/microliter. Erythrocyte sedimentation rate (ESR) and blood cultures should also be done. Blood cultures are infrequently positive.

Conjunctival swabs and blood cultures usually have a low yield and may not be representative of organisms causing an orbital abscess. If meningeal or cerebral signs develop a lumbar puncture may be indicated to rule out intracranial complications in additional to imaging.

When fungal orbital cellulitis is suspected, intranasal biopsies sent for frozen section looking for hyphae elements can be helpful [21]. Diagnosis is confirmed by biopsy by the necrotic tissues in the nasopharynx or involved sinus. Zygomycosis has non-septate large branching hyphae that stain with hematoxylin-eosin stain. Aspergillus species stain with the Grocott-Gomori methenamine-silver nitrate showing septate branching hyphae of uniform width.

*2.3.3. Investigations (imaging)*

The most commonly affected sinus is the ethmoid (91.6%) (**Figure 5**) [12]. X-rays of the sinuses can show fluid-filled cavities in the sinus and may show thickened mucous membranes. However, CT scan imaging of the orbit and sinuses is the usual modality of choice for diagnosis and monitoring as it shows more definition. It is indicated in inflammation with proptosis, external ophthalmoplegia and reduced vision. Other indications include no improvement or deterioration of the patient's condition within 24 h or non-resolving pyrexia over 36 h.

The CT scan demonstrates the sinuses involved and size and location of possible orbital abscesses (**Figures 5** and **6a**, **b**). Sinus X-ray can show an air-fluid level, for orbital abscesses with gas [25]. Ultrasound can detect abscesses of the anterior orbit with 90% accuracy [6].

CT scans have additional benefits as it also determines the inflammatory changes in the orbit and identifies potential sources of infection including a foreign body. It defines size and location of an orbital abscess and subperiosteal abscesses (**Figure 7**). Early abscesses may appear as increased soft tissue density and when enlarged, a fluid collection with rim enhancement may be present. Identification of orbital abscesses can be challenging on CT and a third of abscesses may be missed if the coronal sections are not done [26]. Contrast media can enhance

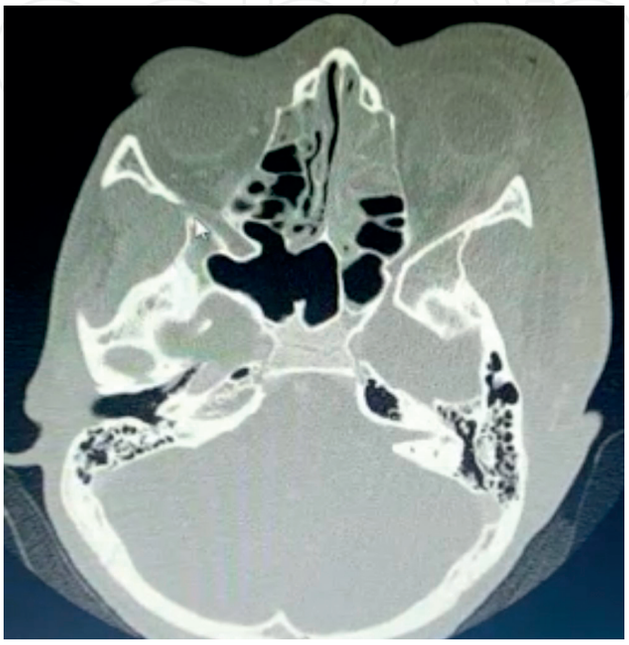

**Figure 5.** Left orbital cellulitis secondary to a left ethmoiditis with left periorbital oedema and associated mucosal thickening in the right ethmoid sinus (axial CT scan).

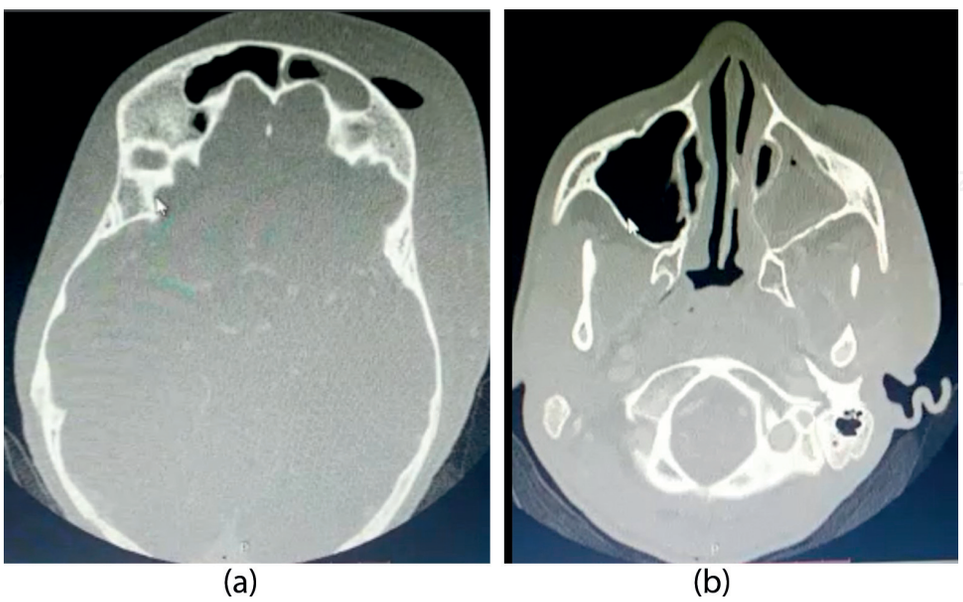


**Figure 6.** (a) Axial CT scan showing left frontal sinusitis associated with swelling of the left side of the face and air in the soft tissues. There is associated mild mucosal thickening in the right frontal sinus. (b) Axial CT scan showing left maxillary sinusitis.

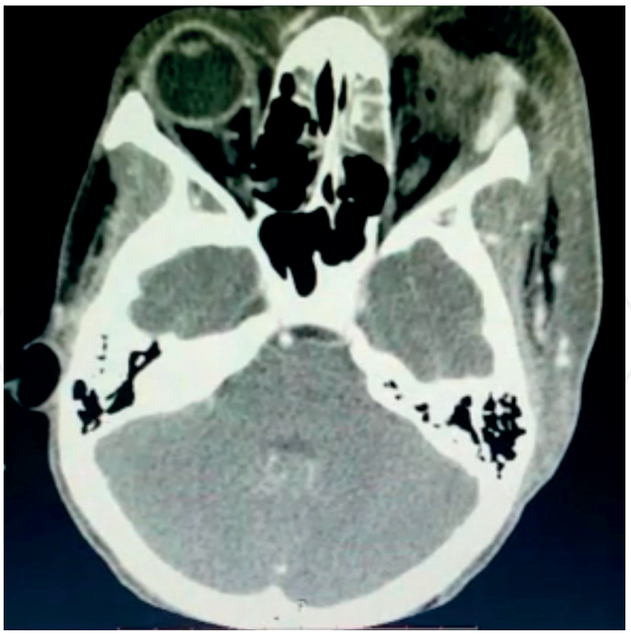

**Figure 7.** CT scan of the orbits showing air present in a localized loculated collection in the superior orbit.

the surrounding wall of the abscess on CT scanning and can differentiate between an abscess and orbital inflammation. The orbital abscess size may increase during the first few days of intravenous antibiotics [26].

The presence of neurological signs requires imaging for intracranial extension. MRI imaging can supplement CT scans with better resolution of orbital soft tissues. Fat saturated T2weighted MRI and diffusion weighted imaging are helpful. MRI is superior to CT for imaging orbital and subperiosteal abscesses and intracranial involvement and reduces the exposure to radiation [6]. In cases with possible complications, a MRI or CT venogram can help elucidate the presence of a cavernous sinus thrombosis.

### 2.4. Treatment

Orbital cellulitis requires in hospital management with intravenous broad-spectrum antibiotics. This should cover most Gram-positive and Gram-negative bacteria. Treatment of the predisposing factor, for example sinusitis, should be implemented early. Treatment is initially with 1–2 weeks of intravenous antibiotics followed by 4 weeks of oral antibiotics [4]. Management of these cases is multidisciplinary with involvement of the ophthalmologist, otolaryngologist, infectious disease and neurosurgical specialists.

Nasal decongestants help to promote spontaneous drainage of the infected sinus and early intervention to drain the involved sinus.

#### *2.4.1. Antibiotics*

Broad spectrum intravenous antibiotics are used empirically. In a Canadian study on orbital cellulitis in children, the commonest combination was IV cefuroxime (24%), IV clindamycin +

IV cephalosporin (21%) and IV cloxacillin + IV cefotaxime (18%) [17]. Subperiosteal abscess were noted in 31.5% of patients but only 21% of patients required surgical intervention [17]. In adults, high dose IV {Augmentin (amoxil and claviolonic), ceftriaxone and sulbactum} and metronidazole have been found to be effective [12].

Children have simpler infections than adults with one aerobic pathogen [7]. Children, 9 years and older and adults may have multiple aerobic and anaerobic organisms which may require medical and surgical treatment. There is a sliding scale of risk and older patients should undergo sinus surgery early before the development of orbital or intracranial abscesses.

CT scans are not predictive of clinical course for orbital abscesses [7]. Expansion of an abscess on CT scan in the first few days is not indicative of failure of antibiotics [26]. However, if visual function is compromised, drainage of the abscess is warranted. Drainage within 24 h is recommended if the orbital abscess is large (superior or inferior), dental involvement (children >9 years), evidence of intracranial extension, involvement of the frontal sinuses [7]. Children <9 years can be monitored with an expectant approach if they have a medial subperiosteal abscess (modest size), no visual loss, nor intracranial or frontal sinus involvement [7]. The patient must undergo continual monitoring of their optic nerve function (Snellen vision, RAPD, colour vision, pupillary reactions) and level of consciousness.

Fungal cellulitis requires aggressive antifungal treatment and may require orbital exenteration and yet still have a high mortality rate [21]. The treatment regime for fungal orbital cellulitis involves:

- Intravenous (IV) amphotericin and irrigation of amphotericin
- Aggressive surgical debridement -Wide excision of devitalized and necrotic tissues
- Adjunctive hyperbaric oxygen
- Correction of metabolic defect
- Exenteration in severe unresponsive cases.

Orbital fungal cellulitis is treated with intravenous anti-fungal. Intravenous amphotericin B can be used initially then posaconazole orally when discharged. Voriconazole or amphotericin B can be used for invasive aspergillosis. In mucormycosis, intravenous amphotericin B may be used or IV micafungin as an adjunctive treatment. In some cases, a suture tarsorrhaphy (closure of the eye lids) can be done and an irrigation cannula placed to deliver intraorbital amphotericin B [21].

Intra orbital catheter delivery of amphotericin B can be used as an adjunctive therapy with early aggressive surgical debridement when required. For invasive aspergillosis, voriconazole or amphotericin B may be used. The onset of fungal orbital cellulitis can initially be insidious then progress rapidly, so a high index of suspicion is important.

### *2.4.2. Surgical treatment*

Approximately, 12–15% of patients require surgical management [5, 27]. Children 10–19 years old were more likely to require surgical intervention and much older patients with leukocytosis

[5]. The presence of acute and chronic sinusitis, proptosis, diplopia, conjunctival chemosis increases the odds ratio of surgical intervention.

Surgical treatment is used for treatment of the source of infections (pan sinusitis) and complication of orbital cellulitis (intraorbital or intracranial) with good result (**Figure 8a** and **b**). Drainage of a subperiosteal abscess requires an incision to the periosteum. Insertion of a drain for several days may be used. Functional endoscopic sinus surgery (FESS) can be done for some periosteal abscesses, eliminating the need for an external ethmoidectomy and facial scar [4]. In fungal orbital cellulitis, early diagnosis and initiation of treatment may also require limited debridement. However, severe invasive fungal orbital cellulitis may require exenteration.

*2.4.3. Role of corticosteroids*

Oral steroids may be used with caution as an adjunct to intravenous antibiotic therapy, as it can hasten the resolution of the inflammation, reducing the duration of the intravenous antibiotics and length of the hospital stay. It also has a low risk of exacerbating infection [28]. Steroids are started after a positive response to intravenous antibiotics has occurred [28]. Children with orbital cellulitis treated concurrently with intravenous steroids (IV dexamethasone 0.3 mg/kg/d Q6H for 3 days) had significantly shorter hospital stays than those without (3.8 vs. 6.7 days, $p < 0.001$) [29]. A short course of systemic steroids concurrent with IV antibiotics appears to be safe and efficacious [29]. The hospital stay was shorter for the children who had IV steroids, whether they had surgical intervention.

## 2.5. Complications

Complications from orbital cellulitis can result from mechanical factors in the orbit or haematogenous and contiguous spread. There are valveless veins around the orbit which predispose to this spread.

**Ocular complications** result from proptosis and increased pressure in the orbit. It includes exposure keratopathy, glaucoma, central retinal artery or vein occlusion, optic neuropathy from an orbital apex syndrome.

The other complications of orbital cellulitis include subperiosteal abscess, intracranial complications (cavernous sinus thrombosis, meningitis and brain abscess) [12]. Approximately, 0.3–5.1% develop orbital or subperiosteal abscess [10, 11]. Development of orbital abscess does not correlate specifically with the patient's vision, proptosis or any other sign [25].

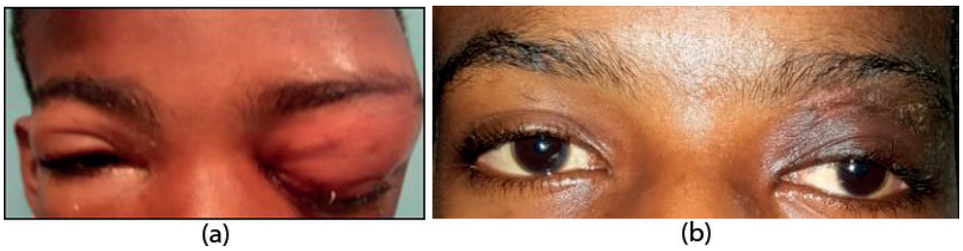


**Figure 8.** (a) Left orbital cellulitis. (b) Six weeks post-surgical intervention and intravenous antibiotics.

**Orbital or periosteal abscesses** should be suspected in patients with progressive proptosis with globe displacement, swinging pyrexia and failure to improve despite intravenous antibiotics. They are usually localized adjacent to the affected sinus in the subperiosteal space, usually the medial orbital wall. Serial imaging may be required.

In younger children <9 years, they may develop isolated medial or inferior subperiosteal orbital abscesses with good vision and mild to moderate proptosis, however, may settle with medical treatment.

Indications for surgical intervention for subperiosteal abscess [30] are:

- Patients ≥9 year old
- Non-medial location of subperiosteal abscess
- Involvement of frontal sinusitis
- Large subperiosteal abscess
- Suspicion of anaerobic infection
- Chronic sinusitis
- Acute optic nerve compromise
- Infection of dental origin

Orbital abscess is more likely in post-traumatic or post-operative cases. Before antibiotics, death from **meningitis** occurred in 17% of cases and blindness in 20% [8]. Present day, ~ 1.9% of patients will develop meningitis and 7–23% can result in blindness, from ocular complications such as corneal ulcer, central retinal artery occlusion or optic atrophy. Delay in required surgical intervention also results in a poor prognosis [8].

Orbitocranial complication of acute sinusitis in children, though uncommon can be life threatening causing high morbidity if diagnosed late. They may require additional procedures such as endoscopic sinus surgery, orbital decompression or subdural empyema drainage [31].

Intracranial complications are uncommon but can be very serious. Meningitis, brain abscess and cavernous sinus thrombosis can occur. Brain abscesses must be considered in patients who have the classic triad of headache, fever and neurological deficit, but may be present in <50% of cases, however, a headache may be present in 70% of cases [30, 32]. The neurosurgical team must be involved as neurosurgical drainage may be required. Cavernous sinus thrombosis must be considered in patients with rapid progression of proptosis, ipsilateral ophthalmoplegia. These patients may also have clinical signs of severe headache, nausea and vomiting. Orbital cellulitis is an inflammatory and infective disease of the orbit which can have visual threatening and life-threatening complications. It is important to diagnose, investigated and treat early to reduce complication and morbidity.

## Conflict of interest

The author has no conflict of interest.

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