Orbital Complications of Sinusitis

Sinusitis can lead to local and systemic complications. Anatomically, most local complications are linked directly to the paranasal sinuses and other structures of the head, neck, and chest. The structures involved most often are those of the orbit, cranium, chest, and nose. Sinusitis can lead to the development of local complications, such as orbital cellulitis, subperiosteal abscess, and orbital abscess, intracranial disorders (brain abscess [BA], subdural empyema [SE] and meningitis, facial osteomyelitis, and thrombosis of the cavernous sinus and cortical vein (1,2). The precise rates of these complications are not known, but they occur in about 5% of patients hospitalized for sinusitis.

This page describes the microbiology, and antimicrobial management of orbital and intracranial complications of sinusitis. It is imperative to consider also the use of surgical treatment in these conditions. However, this review is not describing these issues.

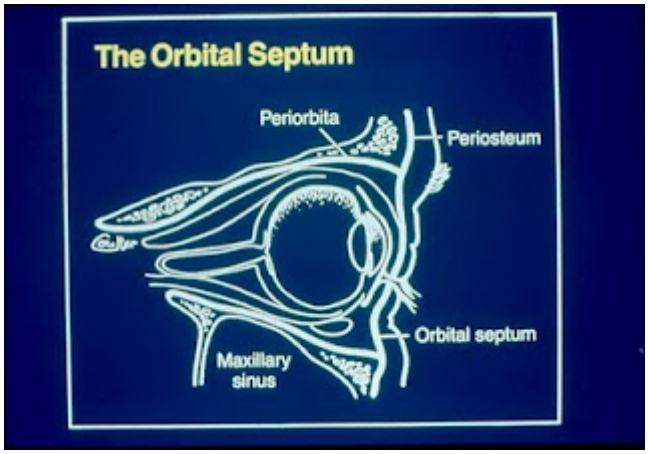
Orbital complications

Pathogenesis

The orbit is susceptible to contiguous spread of infection from the sinuses as it is surrounded by sinuses on three sides. This is more accentuated in children, because of their thinner bony septa and sinus wall, greater porosity of bones, open suture lines, and larger vascular foramina. Differential diagnosis of orbital involvement should include bacteremia (caused by *Haemophilus influenzae* or *Streptococcus pneumoniae*), facial infections, trauma, iatrogenic causes, tumors, and dacryocystitis. However, sinusitis is responsible for a least 75% of cases (3), and orbital complication may be the first and only presenting sign of sinusitis (1).

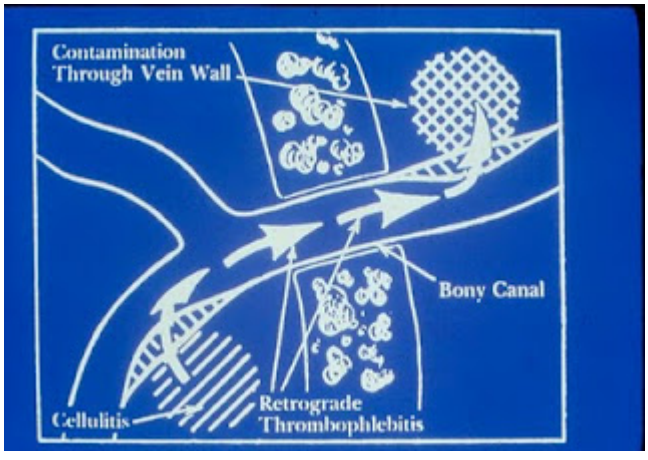
The orbit is separated from the ethmoid cells and maxillary sinus by thin bony plates (called the lamina papyracea) only, which have naturally congenital bony dehiscences. Infections can spread directly by penetration of the thin bones or through the small bony dehiscence.

Orbital anatomy



Infection can also extend directly by traversing through the anterior and posterior ethmoid foraminas. Since the ophthalmic venous system has no valves, the extensive venous and lymphatic communication between the sinuses and the surrounding structures allows flow in either direction, which enables retrograde thrombophlebitis and spread of the infection.

Intra-cranial spread of infection from the periorbital area through retrograde thrombophlebitis.



Orbital complications have been categorized by Chandler et al. (4) into five separate stages according to severity.

Table: Orbital complications of sinusitis (4).

Class 1: Inflamatory edema and preseptal cellulites.

Class 2: Orbital cellulitis.

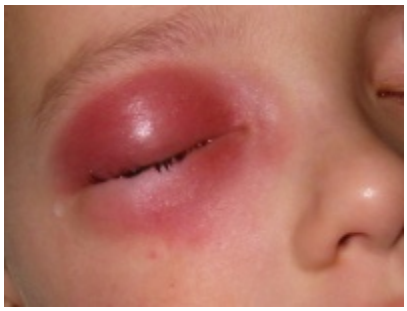
Class 3: Subperiostal abscess.

Class 4: Orbital abscess.

Class 5: Cavernous sinus thrombosis.

Establishing the severity of the infection helps to make the appropriate decision with regard to medical and surgical therapy. The first class is an inflammatory eyelid edema or preseptal cellulitis, which results from venous obstruction caused by infection-induced inflammatory pressure on the ethmoid vessels. The patients present generally with signs and symptoms of sinusitis associated with edema and erythema of the eyelids, which can progress causing the eye to become swollen shut. However, no associated proptosis, visual impairment, or limitation of extraocular muscle mobility occurs. Barriers that limit the progression and spread of the infection to the orbit are the orbital septum and the tarsal plate.

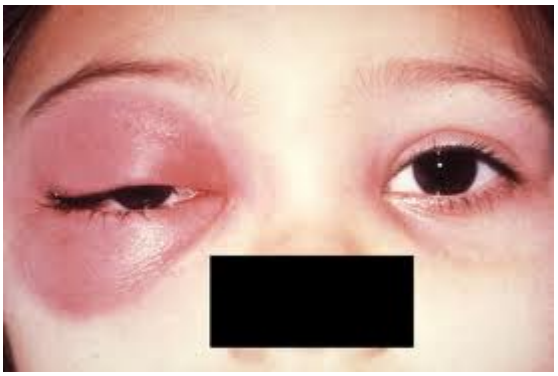
Orbital cellulitis



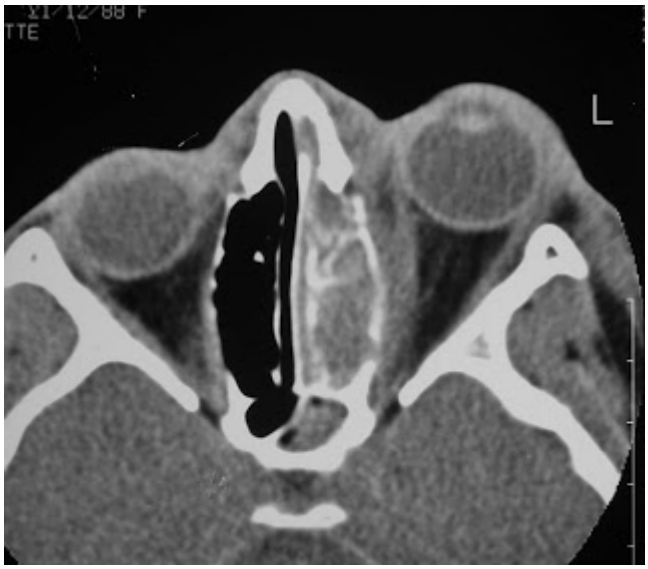
The second class of orbital complications is orbital cellulitis, which represents an inflammation and cellulitis of the orbital contents with varying degrees of proptosis, chemosis, limitation of extraocular movement, and/or visual loss that depend on the severity of the process. Orbital involvement causes diffuse edema and bacterial infiltration of the adipose tissue, but no abscess.

The third class is subperiosteal abscess, which is often a progression of orbital cellulitis, and forms beneath the periosteum of the ethmoid, frontal, and maxillary bone. The abscess can cause limited or extended lateral (if it originated from the ethmoids) and downward (if it originated from the frontal sinus) displacements of the globe. As long as the infection is confined to the subperiosteal plane, no impairment of vision, ophthalmoplegia, or conjunctival signs occur. Vision is generally normal in the early stages, but can become impaired.

Subperiostal abscess



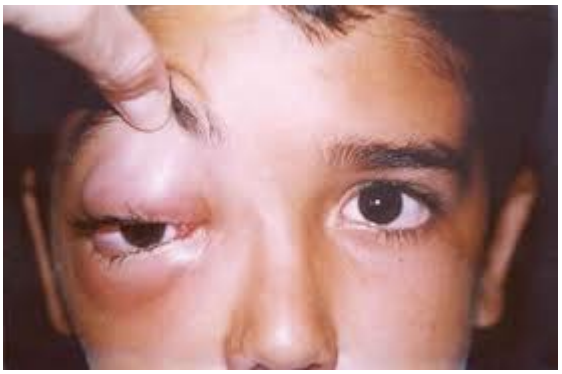
Axial enhanced CT- left medial orbital subperiosteal abscess secondary to left ethmoid sinusitis



The fourth class is orbital abscess, which represents pus accumulation in the orbital soft tissues behind the globe. The abscess develops because of an extended infection into the orbital fat and is associated with inflammatory edema, purulence, and fat necrosis. Severe chemosis, ptosis and complete ophthalmoplegia (cranial nerves II, III, IV, V, and VI are involved) and moderate-to-severe visual loss are present. The visual impairment is attributed to an increase in orbital pressure

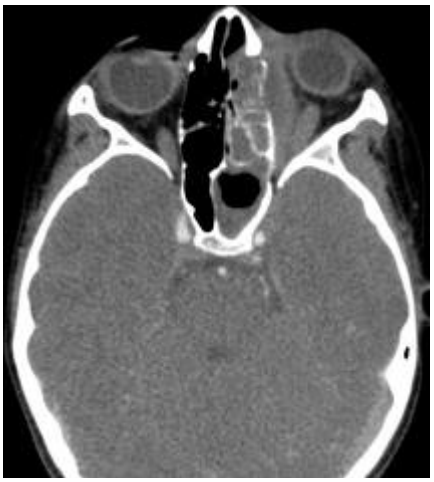
that causes retinal artery occlusion or optic neuritis. If prompt surgical and medical therapy is not provided, permanent blindness can result40. Generally, a displacement of the globe forward, or downward and outward, occurs.

Orbital abscess



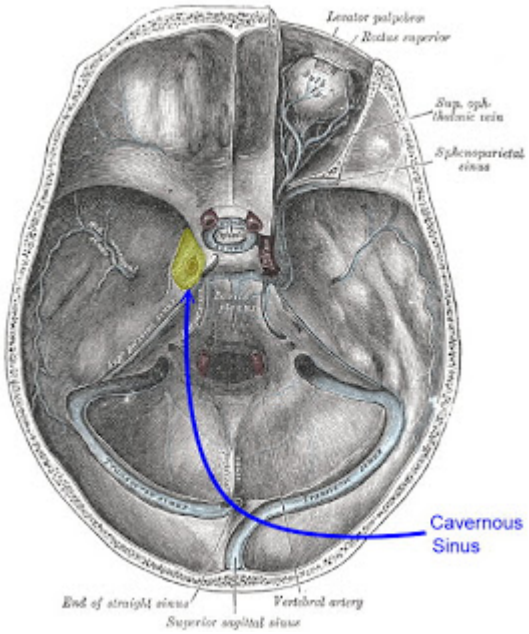
Supper-perosteal orbital abscess

CT of Orbital cellulitis with subperiosteal abscess



The fifth class is cavernous sinus thrombosis (CST), which is retro-orbital and occurs as an extension of the orbital infection.

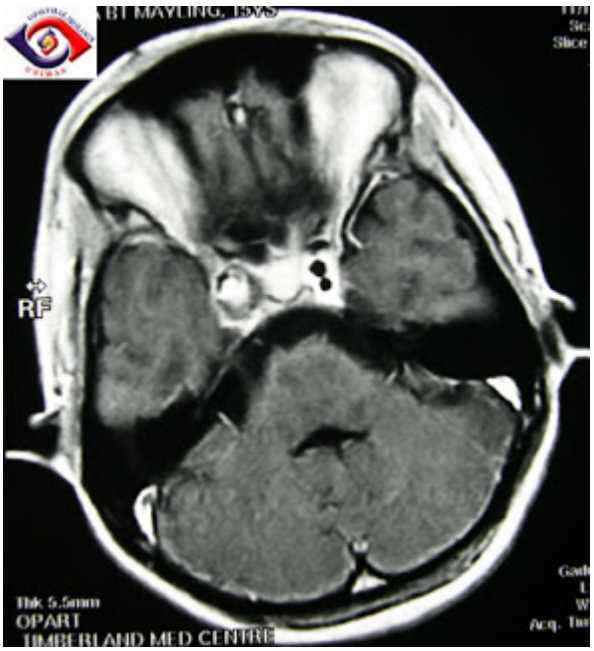
Location of the cavernous sinus



This mode of spread is possible because of the absence of valves in the orbital veins that

communicate with the cavernous sinus. This is a life-threatening complication that is diagnosed by ptosis, orbital pain, severe loss of visual acuity, prostration, hypoesthesia, dysesthesia, and paresthesia along cranial nerves VI or VII, rapid progression chemosis and limitation of extraocular muscle motility, severe retinal venous engorgement, spread of orbital cellulitis and visual loss to the contralateral eye, and clinical deterioration with the development of meningitis, toxicity, and sepsis. Temperature is high with septic emboli, which cause fever spikes. The rate of blindness and death is up to 20% (5-7).

Enhancement of the right cavernous sinus following gadolinium injection



Microbiology

The most common pathogens in cellulitis and abscesses are those seen in acute and chronic sinusitis, depending on the length and etiology of the primary sinusitis. These include *Streptococcus pneumoniae, Haemophilus influenzae, S. aureus*, anaerobic bacteria (*Prevotella, Porphyromonas, Fusobacterium* and *Peptostreptococcus* spp.) (1, 8).

We evaluated aspirates of 18 acutely infected maxillary sinuses that were associated with odontogenic infection in children who presented with periorbital cellulitis (9). A total of 54 isolates were recovered (3.0/ The predominant aerobic and facultatives were α-hemolytic streptococci (4 isolates), microaerophilic streptococci (3), and *Streptococcus pyogenes* and *Staphylococcus aureus* (2 each). The predominant anaerobic bacteria were anaerobic Gram-negative bacilli (17), *Peptostreptococcus* spp.), *Fusobacterium* spp. (8), and *Propionibacterium acnes* (2).

The organisms isolated in CST are *S. aureus* (50-70% of instances), *Streptococcus* spp. (20%) and Gram-negative anaerobic bacilli (pigmented *Prevotella* and *Porphyromonas* spp., and *Fusobacterium* spp.) (10,11). Similar organisms can be recovered from orbital abscesses and their corresponding maxillary sinusitis (12).

Treatment

Medical treatment should be vigorous and aggressive from the early stages of periorbital cellulitis. If this is not done, the infection can progress to orbital cellulitis and abscess. The outcome of medical management depends to a large extent on the duration and stage of the orbital involvement. If orbital cellulitis or abscess is suspected, an ophthalmologist should be consulted. If rapidly advancing infection is suspected, time is crucial and imaging studies and therapeutic measures should be instituted without delay.

Patients with mild inflammatory eyelid edema or preseptal cellulitis (class 1) can be treated with oral antibiotics and decongestants, especially if they have not been treated with antimicrobial

agents before. The most effective available oral ones are cefuroxime axetil amoxicillin-clavulanate. However, close supervision and follow-up is mandatory, and the initiation of parenteral antimicrobial agents in the hospital should be undertaken if postseptal involvement (classes 2 to 5) is suspected or has developed.

The parenteral agents include ceftriaxone or cefotaxime plus coverage for anaerobic bacteria (addition of metronidazole or clindamycin). Drugs that have good brain-blood barrier penetration are preferred.

Antimicrobial agents that generally provide coverage for methicillin-sensitive S. aureus as well as aerobic and anaerobic bacteria include cefoxitin, carbapenems, and the combination of a penicillin (e.g. ticarcillin) and a beta-lactamase inhibitor (e.g. clavulanic acid). Metronidazole is administered in combination with an agent effective against aerobic or facultative streptococci and *S. aureus.* A glycopeptide (e.g. vancomycin) should be administered in cases where methicillin-resistant *S. aureus* (MRSA) is present or suspected.

Treatment of CST, includes high doses of parenteral wide-spectrum antimicrobial agents. The use of anticoagulants and corticosteroids is controversial (13). Anticoagulants are used to prevent further thrombosis, and the fibrinolytic activity of urokinase helps dissolve the clot.

Early diagnosis and vigorous treatment can yield a survival rate of 70-75%. However, permanent sequelae such as blindness and other cranial nerve palsies are common in survivors (14).

Material used from:

http://sinusitisunderstood.blogspot.com/p/complications-and-referal.html