

Complications of sinusitis

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Abstract:

Anatomically paranasal sinuses are in close proximity to vital structures like brain and orbit. Lesions affecting nasal sinuses can affect these areas as well. Infections involving mucosal lining of paranasal sinuses can also spread to these adjacent vital areas. This article attempts to study the orbital and intra cranial complications of sinusitis.

Introduction:

Anatomically paranasal sinuses are closely related to orbit and skull base. They share common bony boundaries, and blood supply¹. Complications attributed to sinusitis are caused by spread of infections to adjacent areas².

Routes of spread:

1. Bacterial infections from the sinuses can spread through natural dehiscences and weakness of the bony barriers. In chronic infections the surrounding bone undergoes sclerosis, while in acute sinusitis massive osteolysis is commonly seen.
2. Lamina papyracea is a paper thin bone separating the orbit from the ethmoidal sinuses. Congenital dehiscences of this bone is commonly seen through which spread of infection can occur from the ethmoids into the orbit. In childhood the frontal sinuses are underdeveloped and orbital complications are caused commonly by acute ethmoiditis.
3. Floor of the frontal sinuses form the roof of the orbit. In older children and in adults frontal sinus infections can spread into the orbit causing orbital complications.
4. Infraorbital canal in the floor of the orbit is a weak area through which infections from orbit may enter into the maxillary sinus.
5. Spread of infection can also occur via diploic veins present in the frontal bone. These veins are known as the veins of Breschet. This is preceded by thrombophlebitis.
6. Venous connections between the sinuses and the orbit do not have any valves facilitating spread of infection from the sinuses to the orbit³.
7. The roots of the second premolar and the first upper molar are intimately related to the floor of the maxillary sinus. This facilitates a two way spread of infection. In cases of isolated maxillary sinusitis dental causes must be suspected.

Predisposing factors for development of complications following sinusitis:

1. Immunocompromised patient (e.g. HIV)
2. Diabetes mellitus

3. Irregular treatment for sinus infections
4. Inappropriate / Inadequate antibiotic therapy

Complications of sinusitis include:

1. Orbital complications (Commonest)
2. Intracranial complications⁴ (including meningitis, subdural empyema, intracerebral abscess, epidural abscess, cavernous sinus thrombosis)
3. Mucocele
4. Pyocele
5. Osteomyelitis
6. Pyocele
7. Facial cellulitis
8. Subperiosteal abscess

Orbital complications of sinusitis:

This is more common in younger individuals. Orbital complications are frequently caused by ethmoiditis because ethmoidal sinus shares its border with orbit. Lamina papyracea (paper thin bone) is the lateral barrier separating ethmoidal sinus from orbit. This bone can easily be breached during active infections. Ethmoiditis as a cause for orbital complication is rather common in young children, where as in adults frontal sinusitis happens to be the most common cause for orbital complications. In adults sphenoid sinusitis can involve optic nerve leading on to blindness. Left orbit is commonly involved than right one⁷. The incidence of orbital complications following sinusitis is highly variable i.e. More than 20%.⁸

Hubert's classification of orbital complications of sinusitis⁵:

Hubert classified orbital complications arising from sinusitis into five groups:

Group I: Inflammatory oedema of eyelids with or without oedema of orbital contents.

Group II: Subperiosteal abscess with oedema of lids or spread of pus to the lids.

Group III: Abscess of orbital tissues

Group IV: Mild to severe orbital cellulitis with phlebitis of ophthalmic veins

Group V: Cavernous sinus thrombosis

Smith & Spencer classification of orbital complications:

Group I: Preseptal cellulitis - Characterised by oedema of eyelids without tenderness, visual loss or limitation of ocular mobility.

Group II: Orbital cellulitis without abscess formation - characterised by diffuse oedema of adipose tissues of orbit.

Group III: Orbital cellulitis with subperiosteal abscess formation with displacement of the globe. May or may not be associated with visual loss. Ocular mobility is restricted.

Group IV: Orbital cellulitis with intraperiosteal abscess. Here the displacement of globe is severe with restriction of ocular mobility.

Group V: Cavernous sinus thrombosis.

Route of spread to orbit:

1. Through bony dehiscence / defect (may be congenital or acquired)
2. Through neurovascular foramen
3. Through venous channels

Chandler classified orbital infection into 5 stages⁶:

Stage I:

Periorbital cellulitis. Also known as preseptal cellulitis. It should not be confused with orbital cellulitis which occurs behind the orbital septum. This condition is actually inflammation and infection of the eyelid and portions of skin around the eye. These patients do not have proptosis, limitation of eye movement, painful eye movement and loss of vision. These features actually help in differentiating preseptal cellulitis from orbital cellulitis. Eyelids in these patients appear swollen but not tender. There is no chemosis.

Stage II:

Orbital cellulitis. Also known as post septal cellulitis. This condition is characterized by pronounced oedema and inflammation of orbital contents without abscess formation. These patients have varying degrees of proptosis, restriction of ocular movements, painful eye movements and chemosis. Since loss of vision could occur in these patients it is mandatory to monitor their vision on a regular basis.

Stage III:

Subperiosteal abscess. Abscess develops in these patients between bone and periosteum. Orbital contents are invariably displaced in an infero lateral direction due to mass effect of accumulating pus. Chemosis and proptosis are invariably present. Decreased ocular mobility and loss of vision may also occur in these patients.

Stage IV:

Orbital abscess. This involves collection of pus within the orbital contents. This is caused due to relentless progression of orbital cellulitis or rupture of orbital abscess. These patients have severe proptosis, complete ophthalmoplegia and commonly loss of vision also.

Stage V:

Cavernous sinus thrombosis. Development of bilateral ocular signs is the classic feature of this stage. This stage carries worse prognosis. Features of this stage include⁹:

1. Fever
2. Head ache
3. Photophobia

4. Proptosis
5. Ophthalmoplegia
6. Loss of vision
7. Cranial nerve palsies (3, 4, V1, V2, and 6th nerves)

Schramm's modification of Chandler's classification:

Schramm after studying his patients classified those patients with preseptal cellulitis with chemosis as a separate entity. Prognostically he placed these patients between Chandler's group I and group III patients. Schramm considered these patients as a separate entity as they did not consistently improve with antibiotics and surgery needs to be advocated.

Moloney's modification of Chandler's classification:

Moloney modified Chandler's classification by according lower priority to preseptal orbital infections. In a nutshell he divided orbital complications into preseptal and postseptal complications.

Signs indication post septal complications include:

1. Proptosis
2. Restriction of gaze
3. Decreased visual acuity
4. Color vision defects
5. Afferent pupillary defect

Intracranial complications:

Can arise from both acute and chronic sinusitis. It is associated with high incidence of morbidity and mortality¹¹. Intracranial complications are rather difficult to diagnose, and is highly dangerous. High degree of awareness of this condition is a must for positive diagnosis.

These include:

1. Meningitis
2. Subdural empyema
3. Intracerebral abscess
4. Epidural abscess
5. Cavernous sinus thrombosis
6. Sphenoid osteomyelitis

CT scan helps in identifying these patients with intracranial complications. Sphenoid sinusitis happens to be the important cause for intracranial complications. Ironically it causes very few or no rhinological signs and symptoms. Only presenting symptom being headache. It is always worthwhile to take CT scans in patients who present with atypical headache.

Routes of spread of infections from paranasal sinuses to intracranial structures:

1. Venous spread: Septic thrombophlebitis developed in the submucosal venous plexus which converge through Breschet veins in the frontal bone diploe. These veins are valveless,¹³ crossing anterior and posterior canal walls of the frontal sinus and converges on the meningeal veins. Transmission of infection is possible through this venous channel. Hence frontal sinusitis can cause osteomyelitis, extradural or subdural abscess.
2. Arterial spread: Spread of infection can also occur via arterial embolisation.
3. Direct erosion of the bony barrier separating intracranial contents from paranasal sinuses.



Clinical photograph of a patient with orbital cellulitis

Predisposing factors for intracranial complications following sinusitis¹⁴:

1. Diabetes mellitus
2. Chronic renal failure

Clinical features:

1. Head ache
2. Irritability
3. Neck stiffness
4. Febrile and toxic

These are pointers towards intracranial complications.

Brain abscesses involving frontal and parietal lobes are seen with frontal and ethmoidal sinus infections. Studies reveal that 40% of patients with brain abscess have permanent physical and mental disability.¹⁵ Cerebritis is the first stage in the development of brain abscess. It is characterised by accumulation of inflammatory cells inside the brain parenchyma. Aggressive treatment during this phase will avoid formation of brain abscess.

Subdural empyema is collection of pus between dura and arachnoid. Invariably it coexists with epidural abscess. Frontal sinus infections invariably causes this problem.

Meningitis is inflammation of meninges. Sphenoid and ethmoid air cell infections commonly cause meningitis. Patients appear toxic and febrile with neck stiffness.

Lumbar puncture should be done only after careful fundal examination ruling out elevated intracranial tension as it could lead to coning and impending death.

Imaging CT / MRI really clinches the diagnosis in these patients. Patients with abnormal headaches following sinus infections should always be submitted to Imaging to rule out intracranial complications as they are invariably fatal.

Management:

Medical management with broad spectrum antibiotics and drainage of the involved sinuses either endoscopically or external approach is the accepted management modality. Brain abscess should invariably be drained after a craniotomy. If brain abscess is present management of the same should take precedence over management of sinus infection because it can lead to permanent sequelae.

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