Sinusitis and its complications

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Sinusitis can lead to life-threatening complications which can be avoided by a careful clinical approach.

Sinus function depends on the adequate clearance of normal mucus by cilia through a patent ostium into the nasal cavity.

Aetiology of sinusitis

The paranasal sinuses and nasal cavity are lined by ciliated pseudostratified columnar epithelium, often referred to as respiratory mucosa. Goblet cells and submucosal glands produce secretions that consist of a surface mucus layer and a deeper aqueous layer. The blanket of mucus is transported out of the sinus via the ostium of the sinus, by ciliary activity, the so-called mucociliary transport system.

Replenishment of the mucus layer is continuous, and its clearance removes bacteria and other micro-organisms and so acts as a barrier to infection. Alteration in sinus ostium patency, ciliary function, or in the quality of secretions can lead to an inflammation within the paranasal sinuses. This in turn leads to decreased ciliary function, increased mucus production and an oedematous obstruction of the sinus ostium, establishing a vicious cycle. Ostial obstruction causes mucus stagnation and a hypoxic environment within the sinus, both of which favour anaerobic bacterial growth. Normal sinuses are not sterile but contain the same bacteria that cause acute sinusitis, highlighting the role of ostial obstruction. The result is mucosal inflammation that causes the conversion of ciliated epithelium to secretory epithelium which may be irreversible. The quality of the mucus changes - it becomes thick, and is ineffectively cleared fro the sinus by damaged cilia. Leukocytes attracted to the area release enzymes that damage the mucosa and cilia. In addition, leukocyte function is impaired by low immunoglobulin levels within the mucus and by a low oxygen concentration.

The most important factor initiating sinusitis is blockage of the sinus ostium. This is commonly caused by mucosal oedema that is secondary to a viral or bacterial upper respiratory tract infection or to an allergic condition of the nasal mucosa. The ethmoid sinuses have the narrowest ostia and are the most susceptible to ostial obstruction and hence infection. Sinusitis is acute if total resolution occurs without any mucosal damage, and chronic if the disease resolves with mucosal damage. Alternatively acute sinusitis refers to symptoms lasting less than 4 weeks and chronic sinusitis to longer than 3 months. Allergic rhinitis, nasal polyps, anatomical abnormalities and immunological diseases including HIV infection predispose to chronic sinusitis.

Bacteriology

Acute sinusitis is most commonly caused by *Streptococcus pneumoniae* and *Haemophilus influenzae.* In addition *Moraxella catarrhalis* is responsible for 20% of sinusitis in children. These bacteria may all produce beta-lactamase. In nosocomial sinusitis Gram-negative bacteria predominate, *Pseudomonas aeruginosa, Klebsiella pneumoniae, Enterobacter* species, *Proteus mirabilis* and *Escherichia coli* being the commonest. In chronic sinusitis anaerobes play a major role (88%), especially anaerobic cocci and *Bacteroides.* In 30% of cases there is a mixture of anaerobes and aerobes. *Streptococcus* species and *Staphylococcus aureus* are the prominent aerobes. Fungal infections of the sinuses may occur, usually in patients whose immunity has been compromised by diabetes or other conditions. *Mucoraceae* and *Aspergillus* may both aggressively invade local and regional tissue.

Clinical presentation

Patients with sinusitis may complain of headache, facial pain, fever, nasal congestion and anterior or posterior mucopurulent nasal discharge (Table I).

Table I. **Presentation of acute sinusitis**

- ➤ Headache
- ➤ Facial pain
- ➤ Fever
- ➤ Nasal congestion
- ➤ Purulent nasal discharge
- ➤ Pus in the middle meatus
- ➤ Opacified sinus radiologically.

Diagnosis

Nasal endoscopy with visualisation of the middle meatus, the region of the ostiomeatal complex, and evaluation of nasal anatomy is easily performed under topical anaesthesia.

A clear or normal plain sinus X-ray makes significant pathology unlikely. An air-fluid level, usually of the maxillary or frontal sinus, is significant in all ages and indicates evaluation by nasal endoscopy or computed tomography (CT). Mucosal thickening is nonspecific, especially in the young. Unilateral complete opacification or a sinus is abnormal and correlates well with sinusitis.

A CT scan is the single best imaging technique for paranasal sinus disease and is indicated when there is a lack of clinical improvement on medical therapy, when there is sinusitis with complications or to evaluate chronic refractory sinusitis. Magnetic resonance imaging due

to its excellent soft-tissue resolution is well suited to distinguish fungal sinusitis, sinus neoplasia and intracranial extension of sinus disease.

Management

The primary aim of management is resolution of infection leading to patency of sinus ostia. Antibiotic therapy is the primary treatment in acute sinusitis. This will facilitate recovery, prevent complications of sinusitis, and prevent progressive mucosal damage. The antibiotics should cover beta-lactamase producing strains, be given in sufficient dose to reach the minimum inhibitory concentration within the sinus secretions, and the course should last for at least 10-14 days until symptoms have resolved. Antibiotic selection, such as amoxicillin with clavulanic acid or cefuroxime is empirical, as nasal cultures are unreliable and maxillary sinus aspiration is not performed in uncomplicated cases. There is a poor correlation between the bacteria from a nasal culture and a sinus aspiration and so a general nasal swab is not usually helpful. However, a swab of the middle meatus taken carefully may be useful.

Decongestants reduce tissue oedema and so play an important role. They can be given topically or systemically. Topical decongestants such as oxymetazoline and xylometazoline should not be given for more than 7 days otherwise rebound rhinitis occurs. For a longer period of decongestion systemic preparations such as pseudoephedrine are used. They are alpha-adrenergic drugs (sympathomimetics) which reduce nasal congestion, but they can stimulate the CNS and CVS.

Mucolytic agents, which include systemic preparations such as bromhexine and carbocisteine and topical preparations such as mesna, decrease the tenacity of the mucus and hence facilitate drainage.

Saline or alkaline nasal irrigation and/or steam inhalation provides great benefit by removing thickened secretions and should be done several times a day.

Topical corticosteroids such as beclomethasone, fluticasone and triamcinolone reduce mucosal oedema especially when an allergy is the precipitating factor. Antibiotic therapy should be started well before adding nasal steroids, as the steroids may inhibit the natural defense mechanism of the sinuses. Their maximum benefit may only occur after 1-2 weeks of treatment and they are mainly indicated in chronic sinusitis and allergic rhinosinusitis.

Antihistamines are not indicated for sinusitis as they cause thickening of mucosal secretions. Maxillary sinus washout (antral lavage) is indicated for patients with acute sinusitis who have not responded to 3-5 days of antibiotics or who are immunocompromised.

The pathogenesis of chronic sinusitis is ostial obstruction with secondary bacterial infection, and so there is an important role for surgery to re-establish sinus ventilation and drainage. Patients who fail comprehensive medical therapy of at least 4 weeks, or who have demonstrable abnormalities of the ostiomeatal complex on endoscopy and CT scan are candidates

for surgery.

The complications of sinusitis can be local, orbital and intracranial and may be life-threatening.

Orbital Complications of Sinusitis

The loose skin of the eyelid lies over the orbicularis oculi muscle, deep to which is the tarsal plate. This is connected to the orbital rim by a continuation of the orbital periosteum, called the orbital septum. The periorbital fat lies behind the orbital septum. Within the periorbita, the muscular cone of extraocular muscles further divide the orbit into an intraconal compartment and an extraconal space.

Acute and chronic sinusitis can cause orbital complications to arise because of the proximity of the eye to the paranasal sinuses. In the adult over half of the circumference of the orbit consists of bony sinus walls. The floor of the orbit is in common with the roof of the maxillary sinus and the medial wall of the orbit, the lamina papyracea, is in common with the medial wall of the ethmoid sinuses. The roof of the orbit anteriorly is in common with the medial wall of the ethmoid sinuses and the floor of the frontal sinus, while it may be related to the sphenoid sinus posteriorly. These common walls are exceptionally dehiscent. They contain fissures and have the foramina of the anterior and posterior ethmoid vessels. In these areas the barrier to the spread of infection is limited to the submucosa of the sinus and the periorbita of the orbit. Spread of infection to the orbit occurs either by direct penetration through bony dehiscence, osteitis of bone, or retrograde thrombophlebitis along the many valveless vessels of the ophthalmic venous system that communicate between the sinuses and the orbit. Approximately 75% of orbital infections are directly related to sinusitis, mostly ethmoid sinus disease. Other causes include cutaneous trauma or infection, dacryocystitis, conjunctivitis, periorbital surgery and orbital fractures. Orbital complications of sinusitis occur more commonly in children because children have increased frequency of upper respiratory tract infections, more diploic bone in their sinus walls, increased bone vascularity, relatively thin bones and relatively small sinus ostia. The commonest organisms are *H. influenzae, Staphylococcus* and *Streptococcus.*

Clinical presentation

The eyelids are always swollen, a key sign to the diagnosis. There is a progressive spectrum of orbital signs and symptoms as the contents of the orbit become infected, as described by Hubert and modified by Chandler. The first stage is that of periorbital cellulitis or preseptal cellulitis where the spread of oedema and erythema in a posterior direction is limited by the orbital septum (Figs 1 & 2). This affects the eyelids only and although the eyelids may be swollen shut, the contents and function of the orbit posterior to the orbital septum are normal.

When infection breaks through the orbital septum or spreads directly from the ethmoids through the lamina papyracea, an acute inflammation of the soft tissue that surrounds the globe occurs which is called orbital cellulitis. The signs and symptoms that orbital cellulitis produces

include orbital pain and proptosis which are due to oedema and inflammation of the orbital contents (Fig. 3). Obstruction to conjunctival lymphatics causes conjunctival oedema or chemosis (Fig. 4). As inflammation of the extraocular muscles occurs, reduction in eye movement presents as strabismus and ophthalmoplegia (decreased ocular motility). With intraconal involvement, inflammation of fat and connective tissue around the optic nerve leads to decreased visual acuity and possibly blindness.

A subperiosteal abscess is a collection of pus under the periosteum lining the orbit and may cause proptosis by displacing the globe. Orbital abscesses result from either a preseptal abscess, a subperiosteal abscess or from orbital cellulitis. Clinically severe proptosis, ophthalmoplegia, chemosis, optic neuritis, vascular ischaemia and blindness may occur.

Cavernous sinus thrombosis or thrombophlebitis, a rare but catastrophic complication, occurs secondary to sinus infection with orbital involvement. It eventually becomes bilateral and produces severe headaches, symptoms of meningitis, eye pain and decreasing vision, bilateral proptosis, ophthalmoplegia, chemosis and blindness (Table II).

Table II. **Presentation of orbital infection**

- ➤ Orbital pain
- ➤ Oedema of the eyelids
- ➤ Cellulitis of the eyelids
- ➤ Proptosis
- ➤ Conjunctival oedema
- ➤ Decreased eye movement
- ➤ Decreased vision.

Diagnosis

In addition to a thorough examination of the nasal cavity and sinuses an ophthalmological examination is mandatory in all orbital complications.

A CT scan is essential to evaluate the orbital involvement of disease, to assess sinusitis as the aetiology of the orbital infection, and to exclude other complications such as intracranial infection.

Thickening of the sinus mucosa or opacification of the sinus denotes sinus disease. In the orbit a subperiosteal abscess is well seen, as will be swelling of orbital muscles and an orbital abscess (Figs 5-7).

Management

In children with preseptal cellulitis secondary to acute sinusitis, antibiotics will usually result in resolution. The acute sinusitis should be treated in the usual way. More severe cases will need hospitalisation and intravenous antibiotics. Preseptal and post-septal abscesses require drainage. Subperiosteal and orbital abscesses are also absolute indications for surgery.

Orbital cellulitis can be treated expectantly with high-dose intravenous antibiotics, guided by Gram staining or microscopy, culture and sensitivity of intranasal pus. Conservative management can only be undertaken provided frequent observation of the clinical status can be reliably undertaken including visual acuity, ocular mobility, spread of periorbital erythema and temperature.

If rapid resolution does not occur, or if there is any evidence of progression of disease, immediate surgical intervention is necessary. In orbital cellulitis, external fronto-ethmoidectomy, sphenoidotomy and opening of the natural maxillary sinus ostia or antral lavage are done to provide surgical drainage and orbital decompression.

Intracranial Complications of Sinusitis (4-10% of Sinusitis)

Intracranial complications usually result from acute sinusitis, or an acute exacerbation of chronic sinusitis. The sinus most commonly involved in intracranial infection, which is usually a frontal lobe abscess or meningitis, is the frontal sinus followed by the ethmoid and sphenoid sinuses. Sinusitis is the commonest source of infection in patients who have a brain abscess.

The most significant route of spread of infection is via normal anatomic pathways, by thrombophlebitis originating in the mucosal veins and spreading along the emissary veins of the skull to the dural venous sinuses and subdural veins and finally the cerebral veins. Infection may also spread directly via congenital dehiscences, and avascular osteonecrosis or fracture sites. Subdural empyema or cerebral abscess may result while intermediary structures may be spared, so there may be no extradural abscess or osteomyelitis. The underlying dura becomes thickened as granulation tissue develops. Cortical thrombophlebitis with infarction or osteomyelitis of the cranial bones may also occur.

Clinical presentation

A high index of suspicion is needed in patients with acute and chronic sinusitis who present with frontal headaches, low-grade fever, lethargy, malaise, meningism, impaired consciousness or altered personality. Focal neurological deficits, seizures and signs of raised intracranial pressure indicate overt cerebral involvement (Table III).

Table III. **Presentation of intracranial infection**

- ➤ Headache
- ➤ Fever
- ➤ Lethargy
- ➤ Neck stiffness
- ➤ Nausea or vomiting
- ➤ Focal neurological deficits
- ➤ Change in consciousness
- ➤ Seizures.

Diagnosis

High-resolution CT scan with contrast is the investigation of choice. Axial and coronal images are used to delineate sinus involvement. A lumbar puncture to diagnose meningitis should only be done after a CT scan has excluded an intracranial abscess, and there are no other indications of raised intracranial pressure.

Management

The patient should be managed in conjunction with other specialists, including a neurosurgeon. Initially, intravenous antibiotics should be commended. The antibiotics should be modified as soon as culture results are obtained and should be continued for about 4 weeks. Prophylactic anticonvulsant therapy should also be commenced.

Surgical management consists of drainage of the intracranial sepsis and the sinuses. Osteomyelitis often involves the frontal bone that has an extensive diploic system. Infection leads to avascular necrosis, sequestra formation, and subperiosteal abscesses. Clinically the patients present with a localised subperiosteal abscess, a Pott's puffy tumour, or with a more diffuse spreading type of osteomyelitis. The area must be widely debrided and long-term antibiotics given.

Meningitis

Meningitis is the commonest intracranial complication of sinusitis, usually secondary to sphenoid and ethmoid infections. Classically the patient presents with headache, fever, neck stiffness, irritability, tiredness and delirium. If meningitis is suspected, a CT scan should be done prior to a lumbar puncture. Meningitis is treated with antibiotics but the sinuses should be drained as soon as the patient is medically fit.

Extradural abscess

This is a collection of pus between the dura and the calvarium. Signs and symptoms may be minimal and subtle. A persistent headache or spiking fever should raise the index of suspicion. Management is surgical with long-term antibiotics.

Subdural abscess

This is a collection of pus between the dura and pia mater. Extensive cortical thrombophlebitis is a frequent complication. The disease has a fulminant course and is a neurosurgical emergency. Early symptoms include intense headache, meningism and fever. Later, decreased level of consciousness, hemiparesis and seizures may occur.

Early diagnosis is essential and is confirmed by CT scan.

Management is surgical drainage, often via a craniotomy, and antibiotics. The sinuses are drained at the same time as the intracranial abscess.

Cerebral abscess

With meningitis this is the commonest intracranial complication of sinusitis and is secondary to infection in the frontal or ethmoid sinuses. It is usually a result of retrograde thrombophlebitis with cerebritis, necrosis, liquefaction and abscess formation within the cerebral parenchyma.

Encapsulation usually develops within about 2 weeks.

Initial signs are mild and include fever, lethargy, agitation and headache. This is followed by a quiescent period and then signs of increased intracranial pressure and mass effects occur.

CT scan confirms diagnosis.

Management is drainage or, if possible, excision of the abscess and long-term antibiotics.

Venous sinus thrombophlebitis

The classic orbital findings of cavernous sinus thrombophlebitis are chemosis, periorbital oedema, proptosis, decreased eye movement and decreased visual acuity. The process may be bilateral, in which case the contralateral eye will also be involved. The temperature may spike due to showers of septic emboli. As the infection spreads seizures and decreased level of consciousness may ensue. A high index of suspicion is needed in the presence of orbital infection. A CT scan with contrast or MRI scan will confirm the diagnosis and blood cultures indicate the responsible organism.

Antibiotics are given and the patient is closely monitored for other signs of intracranial sepsis. The affected sinus should be drained as soon as the patient is fit for an anaesthetic. Early recognition and treatment are crucial in these cases.

In a Nutshell

Healthy sinuses depend on normal mucus and mucosa, functioning cilia and patent ostia.

Sinusitis most commonly follows an upper respiratory tract infection.

Sinusitis may be acute or chronic.

The complications of sinusitis may be life-threatening.

The complications of sinusitis can be local, orbital or intracranial.

Always consider sinusitis in a patient who presents with a unilateral swollen eye.

Up to 75% of orbital infection is directly related to sinusitis.

Meningitis is the commonest intracranial complication of sinusitis.

Sinusitis is the commonest source of infection in patients with a brain abscess.

A CT scan is the single best imaging technique for paranasal sinus disease.