Drugs 1998 Jul; 56 (1): 59-71
0012-6667/98/0007-0059/$13.00/0

Recognition and Management of Sinusitis

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Abstract

Acute sinusitis frequently follows upper respiratory tract infections. Patients complain of headache, facial pain, fever and purulent rhinorrhoea. Diagnosis is based upon the symptoms, and treatment comprises symptomatic relief with analgesics, topical or systemic decongestants and steam inhalation. If indicated, antibiotics should be given for an adequate period of time.

Patients with chronic sinusitis complain of a combination of nasal obstruction, rhinorrhoea and postnasal drip associated with intermittent facial pain, with symptoms persisting for 3 months or more. Predisposition to the condition may be caused by rhinitis (allergic or nonallergic) and anatomical variants. Failure of mucociliary transport and sinus ostial obstruction leads to mucosal oedema, mucous hypersecretion and chronic infection.

Current treatment aims are to control rhinitis and improve ventilation and function of the sinuses. Rhinitis may be controlled with the long term use of topical corticosteroids, mast cell stabilisers or antihistamines, either alone or in

combination. Secretions may be cleared with steam inhalation and/or saline nasal douching. Failure to control chronic sinusitis with medical treatment may indicate surgery. The aim of surgery is to improve ventilation and facilitate drainage of the sinuses, allowing the restoration of normal function. Removal of nasal polyps, reduction of inferior turbinates or septal straightening may be all that is required. Some patients will need endoscopic ethmoidectomy and middle meatal antrostomy. Improved ventilation in the ethmoid infundibulum may help to resolve disease in the maxillary and frontal sinuses.

Medical treatment of underlying rhinitis will need to be continued postoperatively, often in the long term, while special consideration needs to be paid to sinusitis in children, in relation to dental disease and in the immunosuppressed.

Complications of acute and chronic sinusitis include intraorbital and intracranial sepsis. These potentially lethal complications need urgent evaluation with high resolution computerised tomography (CT) scanning, intravenous administration of broad spectrum antibiotics (including anaerobic and microaerophilic cover) and urgent surgical drainage as appropriate.

Sinusitis is a significant source of morbidity, may exacerbate chest disease and causes significant economic loss.[1] Uncomplicated acute sinusitis is usually managed by general medical practitioners, whereas a proportion of chronic sinusitis is referred on to a specialist rhinologist. Acute sinusitis is characterised by episodes of inflammation in response to infection or allergy and may be influenced by anatomical variants. These may impair both sinus ventilation and mucociliary clearance.[2] The condition becomes chronic if symptoms persist for 3 months or more.

The anatomy of the paranasal sinuses is variable and has been extensively reviewed.[3] The paranasal sinus cavities are lined with ciliated pseudostratified columnar epithelium. The ostia of the frontal, maxillary and anterior ethmoid sinuses are located in the ostiomeatal complex lying lateral to the middle turbinate within the middle meatus. The posterior ethmoid and sphenoid sinuses open into the superior meatus and sphenoethmoid recess, respectively.

The respiratory cilia within the sinuses are covered by a protective mucous blanket which envelops bacteria and other irritants. It constantly moves along predetermined pathways towards the sinus ostia. The individual mucociliary pathways of the frontal and maxillary sinus are shown in figure 1.

Viruses, bacteria or allergens may predispose to sinusitis by disrupting mucociliary transport. Other causes are highlighted in figure 2.[4] Mucociliary transport is impaired by mucous hypersecretion and by inflammatory mediators released in response to infection or allergy. This leads to mucosal oedema, further loss of ciliary function, obstruction of the ostia, congestion, pain and sensation of pressure.[5] Ostial obstruction leads to absorption of oxygen by the vascular mucosa, and hypoxia within the sinus and retention of secretions. The resulting decrease in pH leads to further mucociliary dysfunction, metaplastic change to mucus-secreting goblet cells and structural damage to the epithelium. This has been correlated with a decrease in ciliary beat frequency from the normal 700 beats/min to less than 300 beats/min.[6] Progression of the disease leads to further oedema of the infected mucous membrane with irreversible damage and polyp formation.

1. Microbiology

1.1 Acute Sinusitis

70% of acute sinusitis is caused by *Streptococcus pneumoniae* and *Haemophilus influenzae*, of which a proportion produce β-lactamase. *Branhamella catarrhalis* is a common cause of acute

sinusitis in children. Despite the finding of *Staphylococcus aureus* in 30% of the asymptomatic population, this bacterium rarely causes acute sinusitis and the value of nasal swabs is limited. Over 200 viruses associated with the common cold have been implicated.[7] They act by disorganising the cilial movement, and their rapid replication within the cell leads to cell death, allowing secondary bacterial infection.

1.2 Chronic Sinusitis

Up to 60% of chronic sinusitis is caused by *H. influenzae*, but the isolation rate is variable. It is also caused by *S. aureus* and opportunistic pathogens including anaerobes such as α-haemolytic streptococcus, bacteroides, *Veillonella* and *Corynebacterium*.[8] *Pseudomonas aeruginosa* is prevalent in patients with nasal polyps or cystic fibrosis. Although fungi are normal commensals of the upper airway, they may lead to chronic sinusitis, especially in the diabetic or immunocompromised patient.

2. Symptoms and Signs

2.1 Acute Sinusitis

Acute sinusitis occurs when the mucociliary transport mechanism breaks down and infection overwhelms. It is often preceded by upper respiratory tract infection and is associated with mucopurulent rhinorrhoea, postnasal drip, cough, fever, nausea and congestion. Facial pain is often described above or below the eyes and may increase on bending forwards. The site of pain or headache may implicate infection in a specific sinus (fig. 3).

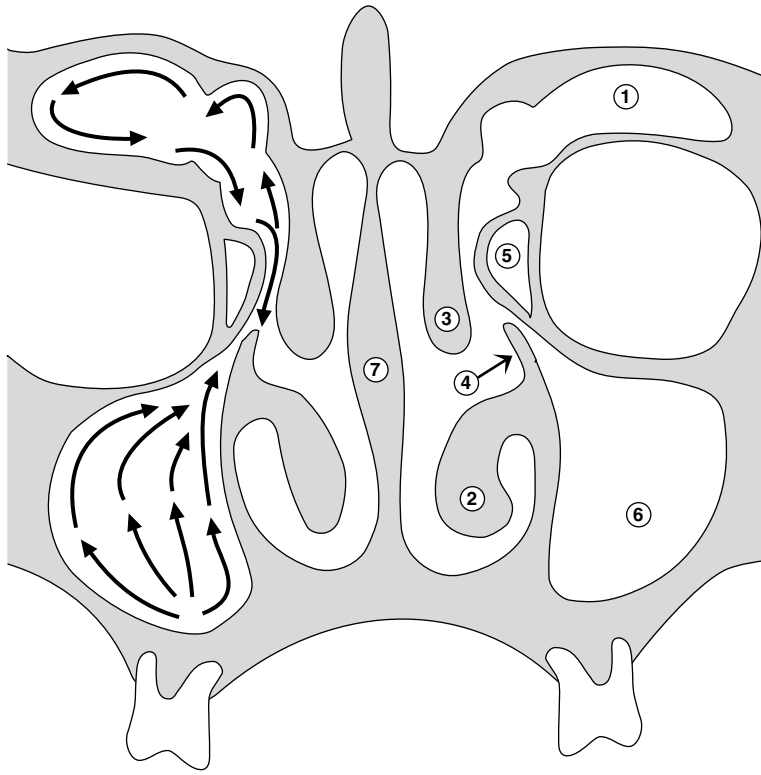

Fig. 1. Mucociliary pathways in the maxillary and frontal sinuses, shown by arrows. 1: frontal sinus; 2: inferior turbinate; 3: middle turbinate; 4: uncinate process; 5: ethmoid bulla; 6: maxillary sinus; 7: nasal septum.

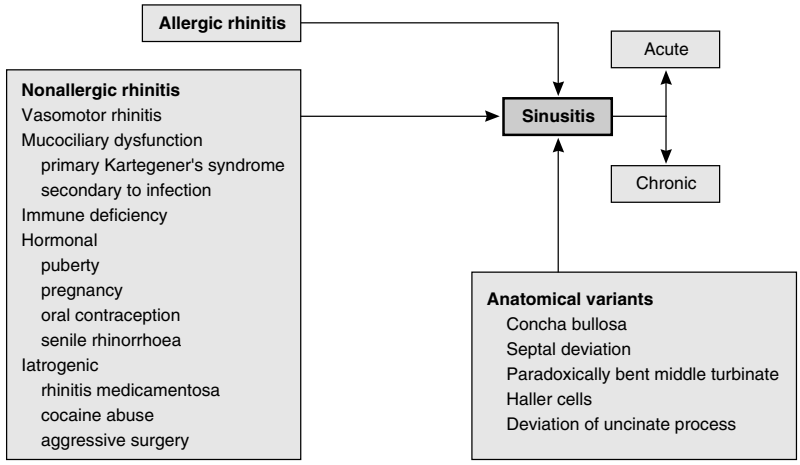

Fig. 2. Factors predisposing to sinusitis.

The signs in uncomplicated acute sinusitis are nonspecific. The nasal airway is congested and the mucosa hyperaemic. Examination of the nasal cavity with Thudicum's speculum has been superseded by rhinoscopy with either flexible or rigid endoscopy, allowing examination in greater detail. The latter has the advantage of better optical definition and the ability to be passed into the middle meatus, allowing identification of both pathology and some anatomical abnormalities. Such an examination may demonstrate pus in the middle meatus, confirming the diagnosis.

2.2 Chronic Sinusitis

Chronic sinusitis is difficult to define. It occurs when acute sinusitis fails to resolve in the presence of mucosal disease, such as allergic rhinitis, and when there is obstruction to the normal ventilation of the sinus involved. The symptoms of chronic sinusitis are similar to those of acute sinusitis, but are often less specific. A singular complaint of either a chronic unproductive cough, persistent laryngitis or a foul taste may be present. Mucopurulent rhinorrhoea occurs with acute exacerbations. Severe headache occurs in half of these patients. This may be maximal 3 to 4 hours after rising and then slowly decreases. Indications for more detailed evaluation include symptoms recurring 3 to 4 times a year, lack of control by steam and decongestants, symptoms that affect more than one anatomic site or where the sinusitis is associated with exacerbation of asthma. The elderly are an ever-increasing proportion of the general population in whom neglected sinusitis can exacerbate chest disease. This is more threatening to this age group and early aggressive medical intervention is important to reduce morbidity.[9]

Factors predisposing to chronic sinusitis include deviation of the nasal septum, septal spurs, hypertrophic turbinates and nasal polyps. Endoscopic examination may reveal other middle meatal abnormalities such as a medially bent uncinate process, an overhanging ethmoidal bulla or a paradoxically bent middle turbinate.The reduced nasal airway, hyperaemic mucosa and purulent secretions can lead to crust formation. Allergic rhinitis may be indicated by pale oedematous mucosa and nasal polyps.[2] Prolonged inflammation leads to polypoid change, notably within the middle meatus and along the middle turbinate. Again, the presence of pus in the middle meatus may be indicative of frontal, anterior ethmoid or maxillary sinusitis.

3. Investigations

3.1 Allergy

Specific allergens may be identified by skin prick testing. This is an inexpensive test during which a variety of allergens can be tested, but does carry a small but significant risk of anaphylaxis. The radioallergosorbent test (RAST) is more expensive and more time-consuming, but provides quantitative information about sensitivity to specific allergens.

3.2 Transillumination

Light is shone through the maxillary or frontal sinus in a completely darkened room. The amount of light transmitted through the palate is observed. Disadvantages include poor correlation with the presence of fluid as proven by antral washout,[10] poor interobserver consistency and the inability to evaluate the ethmoid and sphenoid sinuses. Its use is limited to those who resist ionising radiation or who are pregnant. A positive result can only be assumed when there are gross differences in sinus aeration on each side.

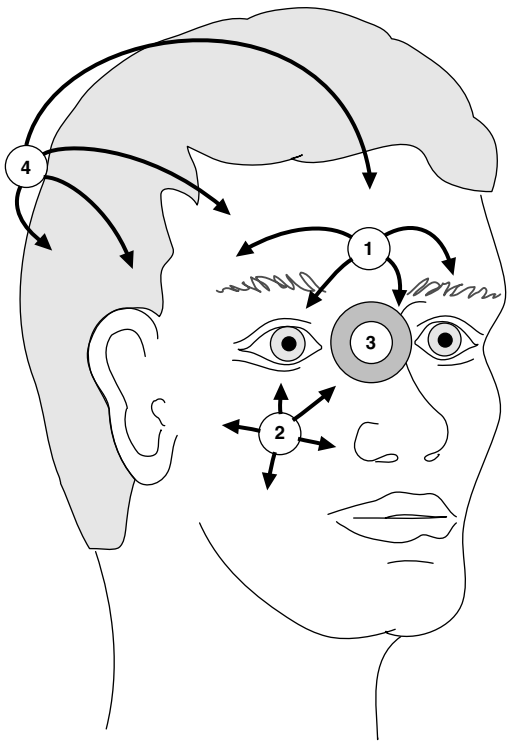

Fig. 3. Sites of referred pain from individual sinuses. 1: frontal sinusitis; 2. maxillary sinusitis; 3: anterior ethmoid sinusitis; 4: posterior ethmoid and sphenoid sinusitis.

3.3 Radiology

3.3.1 Plain x-Ray

Three views including an occipitomental (Waters), an occipitofrontal (Caldwell) and a lateral view to demonstrate the sphenoid sinus are described. Some departments offer only the occipitomental view, as little additional information can be gleaned from the other views and the place of such films in uncomplicated, acute sinusitis is debatable. In acute sinusitis the findings of air fluid levels or opacity rarely lead to a change in management and this has led to further debate regarding the usefulness of routine sinus x-rays in the primary care setting.[11]

The findings in chronic sinusitis are less clear-cut. Mucosal thickening or sinus opacity are commonly seen. Air fluid levels are unusual. Interpretation of plain films is subject to interobserver variation and there is poor correlation between the extent of sinus abnormality and the severity of the symptoms.[12] A correlation between the degree of opacification and antral puncture has been shown,[13] but there may also be abnormal radiological findings in asymptomatic patients as well as in up to 50% of children.

3.3.2 Computed Tomography

Coronal computerised tomography (CT) is currently the 'gold standard' of radiographic evaluation of chronic inflammation of the paranasal sinuses.[14] It is not economically possible or medically necessary to scan each patient presenting with symptoms suggestive of chronic sinusitis. CT scans should only be requested after failure of maximal medical treatment (and when the patient wishes to consider surgery), if a complication arises or if there is suspicion of malignancy.[2]

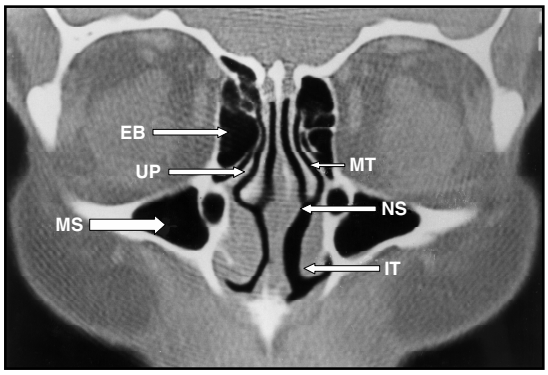

Fig. 4. Coronal computerised tomography (CT) scan demonstrating normal sinus anatomy and mucosa. **EB** = ethmoid bulla; **IT** = inferior turbinate; **MS** = maxillary sinus; **MT** = middle turbinate; **NS** = nasal septum; **UP** = uncinate process.

CT identifies soft tissue abnormalities and provides a surgical map of the paranasal sinuses. For the assessment of chronic inflammatory disease the ideal window settings lie between 2000 and 3000 Houndsfield units (HU), centred around minus 250HU. This permits preoperative assessment of the level of the cribriform plate, bony dehiscence of the lamina papyracea or optic canal and the relationship of the optic nerve to the posterior ethmoid sinus (Onodi cell) [fig. 4]. Anatomical variants are identified which are difficult to see on endoscopic examination of the nose. These may compromise the ostiomeatal complex, e.g. pneumatisation of the middle turbinate (concha bullosa) or Haller cells in the inferomedial borders of the orbit (fig. 5). Anatomical abnormalities occur with equal frequency in the population with and without chronic sinusitis. The combination of the abnormality with infection or allergy predisposes to the infection.[15]

CT is useful for demonstrating to patients the location of disease and the surgical approach, but the decision to operate should be based on the history and findings of endoscopic examination. 24 to 39% of the asymptomatic population may demonstrate mucosal disease and reliance on CT alone may be misleading.[16]

3.3.3 Magnetic Resonance Imaging (MRI)

Magnetic resonance imaging (MRI) provides high definition sections of soft tissue without demonstrating bone. Its role in sinus disease has been reviewed.[14] The mucosa of the ethmoid sinuses and nasal cavity have a natural cycle of vasodilatation and mucosal oedema followed by vasoconstriction and mucosal shrinkage. MRI is of limited value in investigations of chronic sinusitis which are secondary to disease of the anterior ethmoids, because the signal intensity of normal mucosa in this region in the oedematous phase of the nasal cycle is indistinguishable from that of extensive inflammatory disease. The frontal, maxillary and sphenoid sinuses do not have such a physiological cycle and MRI can be helpful.

Certain disease processes may be differentiated by MRI. Inflammatory conditions exhibit high signal intensity on T2 weighted images, whereas neoplastic processes (of which 90% are squamous cell carcinoma) assume an intermediate bright signal.[17] Fungal concretions have very low signal intensities similar to air on T2 weighted images.[18]

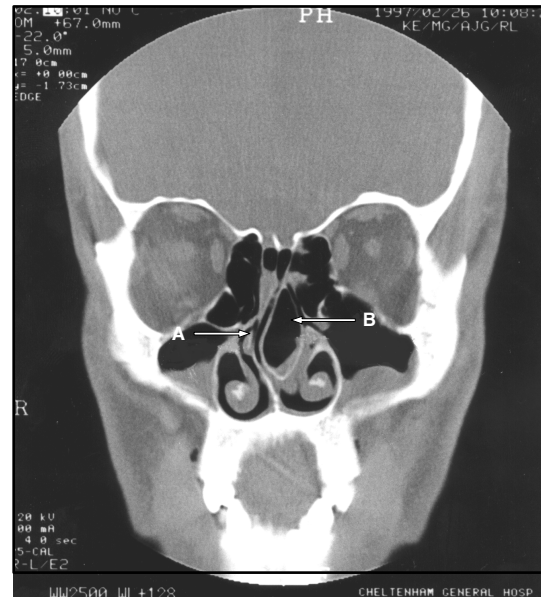

Fig. 5. Coronal computerised tomography (CT) scan showing deviated nasal septum and massive concha bullosa of left middle turbinate. A: deviated nasal septum; B: concha bullosa.

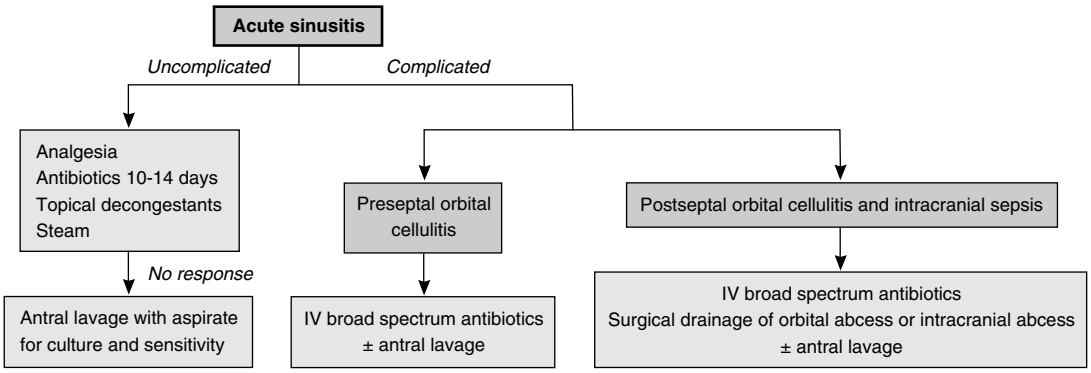

Fig. 6. Treatment plan for acute sinusitis. **IV** = intravenous.

4. Treatment

The aims of treatment of sinusitis are eradication of infection, reduction of inflammation and the restoration of ventilation of the sinuses allowing the return of normal mucociliary function.[4]

4.1 Medical Treatment

4.1.1 Acute Sinusitis

If treatment with antibiotics is indicated they should be prescribed for an adequate period of time (3 to 10 days) [figure 6]. The duration of the course depends on the type of antibiotic. A recent paper has suggested that the duration and relapses associated with acute sinusitis presenting to primary care are not significantly influenced by the prescription of antibiotics and has queried their indication.[19] Others have shown that there is a 90% clinical response to 3 days' treatment with oral β-lactam antibiotics, as well as 80% or more bacterial eradication.[20]

The efficacy of a variety of antibiotics against the commonly encountered pathogens has been compared by repeated antral puncture and culture of sinus aspirate without placebo controls. These include amoxicillin with and without clavulanic acid, cotrimoxazole (trimethoprim with sulfamethoxazole), cefuroxime, erythromycin and azithromycin. Differences were found in efficacy against β-lactamase–producing bacteria and anaerobes depending on the individual qualities of the antibiotics. Nearly one-third of *H. influenzae* produce β-lactamase and therefore amoxicillin will fail in 20 to 30% of cases. Amoxicillin with clavulanic acid, cotrimoxazole and cefuroxime axetil, among others, overcome this problem. Anaerobes isolated in sinusitis are usually sensitive to penicillin. Complications involving the orbit or intracranial cavity require parenteral broad spectrum coverage.

Topical corticosteroids have a limited role in the treatment of acute sinusitis although they may facilitate a more rapid reduction of facial pain.[21]

Topical decongestants can reduce local oedema, promoting drainage and ventilation. They vary in duration of action and there is little evidence to suggest that they influence the disease process. However, careful endoscopic placement of a pledget soaked in a vasoconstrictor can facilitate drainage of the frontal sinus without resorting to frontal trephine. Xylometazoline is a longer-acting decongestant which has the disadvantage of causing rebound vasodilatation after more than 5 days' use. Overuse results in rhinitis medicamentosa. It is not recommended in the long term treatment of sinusitis. Topical nasal decongestants are more effective if administered in the 'Mecca' position, which should be maintained for 2 to 3 minutes (fig. 7). Systemic decongestants provide symptomatic relief but have adverse effects secondary to α-adrenergic stimulation. These drugs have not been demonstrated to speed recovery.

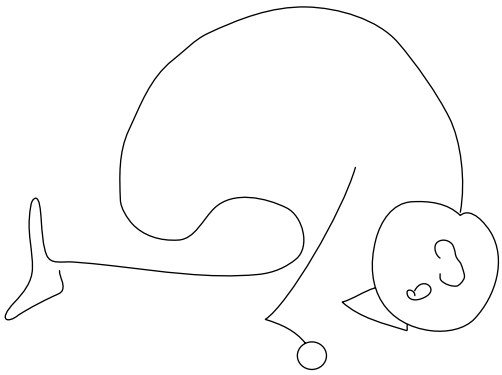

Fig. 7. Mecca position.

Mucolytics such as guaiphenesin may enhance nasal mucociliary clearance by improving the viscoelastic properties of the secretions. Penetration by antibiotics and topical medicines may also be increased.[22] Nasal toilet with steam inhalation and alkaline/saline douching provide symptomatic relief by softening crusts and moisturising dry mucosa.

4.1.2 Chronic Sinusitis

The anterior ethmoid complex has a key role in the development of the majority of recurrent or chronic sinusitis (fig. 8). Identification of predisposing factors is the key to successful treatment. Medical treatment including antibiotics, topical corticosteroids, antihistamines and mast cell stabilisers is required if infection arises secondary to pathophysiological abnormalities. If an anatomical abnormality is obviously predisposing to the condition, patients can benefit from precise surgical intervention. The majority of cases have a combination of pathophysiological and anatomical factors which predispose to chronic infection. For this reason, it is important that medical treatment both precede surgery and continue after surgery.

Initial treatment comprises a combination of antibiotic, topical corticosteroid and antihistamines. Appropriate antibiotics, similar to those discussed regarding acute sinusitis, are used for prolonged courses. Decongestants may have a role in symptom control and should not be used in the long term. Simple inhalation of steam helps with nasal toilet and promotes clearance of secretions, but there is little evidence to suggest that the disease process is influenced by such measures.

Topical corticosteroids suppress the inflammatory response and reduction of mucosal oedema around the ostiomeatal complex promotes drainage and ventilation of the sinuses. To be effective, these agents must be able to reach the affected mucosa and obstruction of the airway by hypertrophy of the turbinates, septal deviation or polyps must be corrected. The maximum effect of treatment does not occur for about 2 weeks and treatment thus should be continued for many months. Mild nasal irritation, crusting and bleeding are common adverse effects. Systemic effects are not noted with overuse until between 4 and 16 times the recommended dose is administered, depending on the preparation.[23]

The second generation antihistamines such as cetirizine and astemizole compete for H_1 binding sites on target cells in respiratory mucosa and do not cross the blood-brain barrier. These are less sedative than the first generation drugs and excess dryness in the nose, crusting and tachyphylaxis have not been observed.

Mast cell stabilisers such as topical sodium cromoglycate (cromolyn sodium) prevent and treat an allergic reaction that has already commenced. This treatment is useful in children and in those who resist the notion of taking corticosteroids. The disadvantage is the need for frequent application each day.

4.2 Surgical Treatment

4.2.1 Acute Sinusitis

Uncomplicated acute sinusitis usually responds to medical treatment. Persistent symptoms may require antral washout through the inferior meatus, either for a diagnostic aspirate or to provide therapeutic lavage. Culture of mucopus is important in disease resistant to medical treatment and is regarded as essential in patients with cystic fibrosis or nosocomial infection. Frontal sinusitis has needed frontal trephine in the past. Accurate endo-

scopic placement of pledgets soaked in decongestant into the frontal recess promotes the free flow of pus without having to resort to surgery in the majority of patients.

4.2.2 Chronic Sinusitis

Surgery is considered if chronic sinusitis fails to respond to adequate medical treatment, if complications are threatened or if an anatomical abnormality is identified which predisposes to recurrent acute sinusitis.

The ethmoid sinus was identified as the origin of chronic sinusitis as early as the nineteenth century, but surgery was focused on the maxillary sinus until the mid-1980s. Inferior meatal antrostomy (IMA) and the radical sublabial antrostomy described by Caldwell[24] and Luc[25] have both been popular in the management of chronic sinusitis and still have an occasional role. The IMA placed lateral to the inferior turbinate at the same level as the floor of the nose improves maxillary sinus drainage when excess secretions accumulate. In less overwhelming circumstances, the cilia continue to transport mucus upwards to the natural ostium.[26] If the ostium remains obstructed the secretions continue to accumulate. Spontaneous closure occurs in 45% of IMA and this is more likely if the diameter is less than 1cm and the child is less than 16 years old.[27]

The surgical approach to chronic sinusitis has been revolutionised by the work of Messerklinger[28] and Stammberger,[26,29] along with the advent of Hopkins rods and CT scanning. Functional endoscopic sinus surgery (FESS) is so named because the surgery is specifically targeted at restoring the physiological function of the sinuses. FESS has been quoted as having success rates of 80 to 95%.[23] Recurrence of disease following endoscopic sinus surgery is usually due to persistent disease that has become symptomatic. The rigid endoscope facilitates better diagnostic assessment of the nasal cavity and close monitoring of postoperative progress. Endoscopic intranasal surgery now includes dacrocystorhinostomy, drainage of mucocoeles, management of CSF rhinorrhoea, orbital decompression and decompression of the optic nerve following traumatic entrapment.

The resection of anatomical or soft tissue abnormality identified preoperatively within the ostiomeatal complex facilitates ventilation of the sinuses and promotes clearance of mucus from the

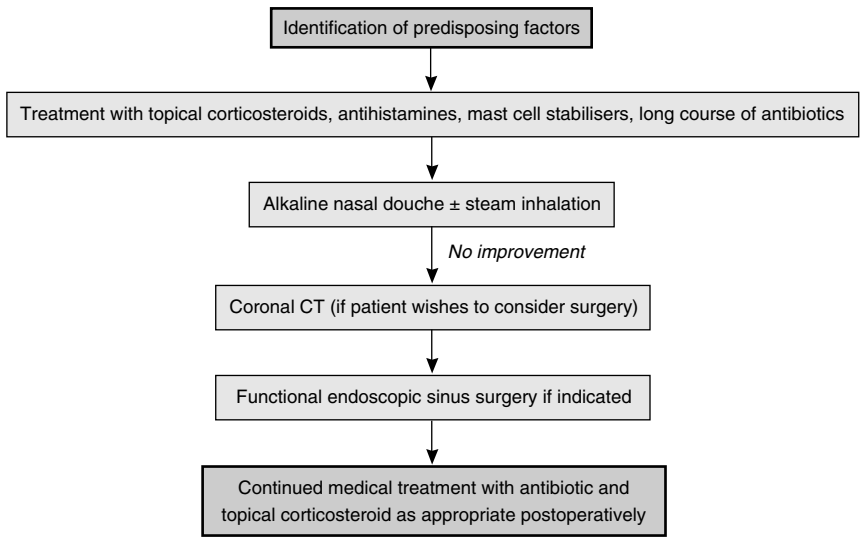

Fig. 8. Treatment plan for chronic sinusitis. **CT** = computerised tomography.

frontal and maxillary sinuses to the middle meatus.[30] Caution is advised in the vicinity of the frontal recess as scarring may lead to persistent frontal sinus disease or mucocoele formation. The posterior ethmoid may be entered through the ground lamella and dissection continued back to the sphenoid.

The management of chronic sinusitis does not end with a surgical procedure. Reversal of mucosal changes may take many months to occur. Alkaline/saline nasal douching with or without steaming are useful to loosen crusts and to cleanse the mucosa, and ideally should be conducted twice daily. Antibiotics, topical corticosteroids and antihistamines may need to be continued for several weeks until the postnasal drip ceases.

Endoscopic sinus surgery is associated with risks to both the orbit and the intracranial cavity. These include breach of the lamina papyracea with diplopia, intraorbital haemorrhage or infection, blindness following transection of the optic nerve, cerebrospinal fluid leak, usually through the lateral lamella of the cribriform plate, and injury to the internal carotid artery. The reported complication rates vary between 0.3 and 8%.[31] It has been noted that there may be a greater incidence of complications while the surgeon is on the learning curve. For this reason, practice cadaver dissections by surgeons should be mandatory.[31]

5. Special Considerations

5.1 Complications

The most worrying complications of both acute and chronic sinusitis involve the orbit and intracranial cavity. In the preantibiotic era, cellulitis of the orbit led to blindness in 20% of patients and death from intracranial sepsis in 17%.[32] Today, visual loss still occurs in up to 10%.[33] Orbital cellulitis is divided into 2 groups by its relationship to the orbital septum which runs from the orbital rim to the tarsal plate. Disease anterior to the orbital septum is more common and rarely threatens vision. Disease posterior to the orbital septum is either related to a subperiosteal collection of pus or to an intraorbital cellulitis/abscess. This causes proptosis and the risk of visual loss is high. Such cases require urgent assessment with an axial CT, administration of intravenous broad spectrum antibiotics and, if indicated, urgent surgical drainage. Other complications of chronic sinusitis include the development of mucocoeles or osteomyelitis. Intracranial abscess formation is often related to acute frontal sinusitis and microaerophilic streptococci are frequent offenders.

5.2 Fungal Sinusitis

Fungal sinusitis appears to be occurring more commonly in recent years. This may be secondary to better diagnostic facilities, a higher index of suspicion and an increase in predisposing factors such as radiotherapy, chemotherapy, immunosuppressive therapy and immunodeficiency diseases such as AIDS.

The clinical features are similar to those of chronic sinusitis and may include a mouldy smell or marked nasal crusting. The colour of the infected matter varies from white/yellow to dirty brown or grey/black, and it has the consistency of butter. Local bony destruction occurs with increasing pressure and inflammation. Radio-opaque masses may be seen within the sinus on CT or MRI. These are related to the heavy metals associated with the fungal nutrients. Sometimes a mass of hyphae sit directly on the mucous membrane. These can be removed endoscopically but if they are very dense a formal Caldwell-Luc procedure is indicated.[34]

Aggressive infection may occur in immunocompromised patients. Severe fungal infections were found at autopsy in 22 to 28% of leukaemic patients. This is characterised by soft tissue necrosis, fibrosis and fungal invasion of vessels. Aggressive medical treatment and wide debridement are essential to prevent orbital complications, meningitis and involvement of the central nervous system. Prolonged granulocytopenia predisposes to invasive fungal infection, predominantly in the lung, but severe disease of the sinuses has been reported. The fungal species include *Aspergillus*, *Candida*, *Rhizopus* and *Mucor*.

5.3 Dental Disease

Up to 10% of sinusitis may be related to periapical or periodontal disease, as well as oroantral fistula. The latter usually occurs following dental extraction, particularly of the molar teeth. The risk of oroantral fistula is greater in adults than in children because of the continued expansion of the sinus into the alveolar bone after the eruption of the secondary dentition.[35]

The infection presents with facial pain, swelling, tenderness and discharge from the nose or oral fistula.[35] Anaerobic organisms predominate and antibiotics should be chosen appropriately. It is essential that the dental disease be eradicated or the sinusitis will persist.

5.4 Paediatric Sinusitis

There is an increasing awareness of chronic sinus disease in children. Symptoms include rhinorrhoea, cough, fetor, headache and low-grade fever.[36]

Such infection may be related to hypertrophy or recurrent infections of the tonsils and adenoids, allergy and cleft palate. These factors should be addressed before considering sinus surgery. Enlarged adenoids may cause stasis of secretions and secondary inflammation of the sinus ostia. Removal of nonobstructive adenoids has been shown to be of little benefit.

CT again confirms the most common site of disease as being the ethmoid infundibulum and the anterior ethmoid complex. The most common bacteria are *S. pneumoniae*, *H. influenzae* and *B. catarrhalis*.[36] Culture of ethmoid biopsies found *B. catharralis* to be prevalent in children under 4 years of age and *S. aureus* to be prevalent in those over 4.[36] *S. aureus* and anaerobes were more prevalent in those whose symptoms had persisted for more than 1 year. Amoxicillin plus clavulanic acid, or cefuroxime are therefore appropriate in the management of chronic sinusitis in children.[36]

Sinus surgery may be indicated if intensive medical treatment fails. Antral washout requires extra care if the secondary dentition has not erupted. As with adults, endoscopic intranasal procedures have become more popular. However, before undertaking paediatric sinus surgery, it is important to be aware that interfering with the middle third of the growing face may lead to asymmetrical development, as has been demonstrated in piglets.[37]

Outpatient nasal endoscopy is not well tolerated by children and endoscopic surgery is considered after evaluation by CT scan. Paediatric endoscopic sinus surgery should always be conducted under general anaesthesia following the application of a topical decongestant. Occasionally, access to the middle meatus is restricted and a limited septoplasty or reduction of the middle turbinate is required. The surgical procedure is similar to that in adults except that the surgical risks are increased by operating in a confined space. Crusts and granulation tissue are removed under a second general anaesthetic 2 to 3 weeks after the initial procedure. 80% of the children with both asthma and sinusitis improved after their sinusitis was controlled.[38] Failure of surgery is associated with immunodeficiency, cystic fibrosis, immotile cilia syndrome and chronicity of disease.

5.5 Immunocompromised Patients

Patients who are HIV positive have deficits in both cell-mediated and humoral immunity and are more susceptible to bacterial infection. Sinusitis occurs in 75% of those with AIDS and is often extensive and difficult to treat, especially if the CD4 count is less than 200/mm^3. As in HIV-negative patients, the ethmoid and maxillary sinuses are again predominantly involved. The possibility of acute or chronic sinusitis must be entertained in an immunosuppressed patient presenting with fever, facial pain, swelling and nasal crusting or ulceration (fig. 9). Plain x-rays may only exhibit slight mucosal thickening, but if this is abnormal, antibacterial and antifungal agents should be started immediately. Failure to respond to medical treatment is an indication for antral aspiration to allow bacteriological identification and prescription of appropriate antibiotic treatment.[39]

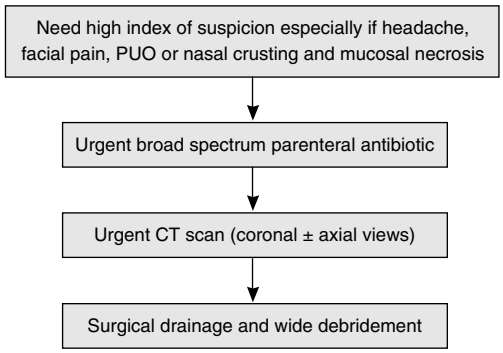

Fig. 9. Sinusitis in the immunocompromised patient. **CT** = computerised tomography; **PUO** = pyrexia of unknown origin.

Pathogens are usually bacterial, such as: staphylococcus, *S. pneumoniae* and *P. aeruginosa*. *Cryptococcus neoformans*, cytomegalovirus and a variety of fungi have also been identified.

6. Conclusions

Sinusitis can have both acute and chronic presentations. Acute sinusitis usually follows viral upper respiratory tract infection and is managed by nasal decongestion and broad spectrum antibiotics. Chronic sinusitis is more common in persons with rhinitis (allergic or non-allergic), although anatomical variants may also contribute to the problem.

The diagnosis is based on the history and examination. CT scanning is helping in confirming the extent of disease and in planning endoscopic sinus surgery. Emphasis must be placed on the medical management of chronic sinusitis with antibiotics, topical nasal corticosteroids and antihistamines. Surgery may reduce the severity of the symptoms and the frequency of infection, but medical treatment often needs to be continued post-operatively on a long term basis.

Finally, special considerations must be given to the complications of sinusitis, including fungal infection, the role of dental disease, paediatric infections and immunocompromised patients.

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