Clinical Review

Bacterial Sinusitis

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Sinusitis may occur secondary to infectious agents, allergens, or pollutants. Bacteriologic studies carried out from sinus punctures revealed that Streptococcus pneumoniae and Hemophilus influenzae are the most common bacterial pathogens isolated. Staphylococcus aureus and Streptococcus pyogenes are not uncommon pathogens. Complications of sinusitis, including orbital cellulitis, usually are due to infection with Staphylococcus aureus and H influenzae. The recent increase in certain areas of the country of β-lactamase-producing strains of H influenzae is noted. When the etiology remains to be determined in the patient with acute bacterial sinusitis, initial therapy with an oral cephalosporin seems warranted.

Galen believed that the paranasal sinuses protected the lungs from cold air. Others have suggested that they protect the brain from the nasal stream.[1] The sinuses are bilaterally placed, epithelial-lined cavities that internally surround the face and nose (Figure 1). They are lined with columnar ciliated epithelial cells interspersed with mucus-secreting cells. The sinuses form embryologically. All of the sinuses drain into the nasal cavity.

The maxillary sinus does not become important clinically until 18 to 24 months of age.[2] The frontal sinuses may not be present radiologically until the eighth year of life. The ethmoidal sinuses are present at birth and are always clinically significant. The venous drainage of the sinus is into the cavernous sinus.[3] The cilia beat away from the central structures and with mucus production send particulate material to the meatal orifices.

Immunoglobulins are present in sinus mucus in higher quantities than in the bloodstream; presumably they play a role in host defense mechanisms. Immunoglobulin A (IgA) may bind to bacteria, thereby preventing colonization and subsequent infection. IgA and immunoglobulin G (IgG) are believed to be produced locally; in some cases IgA levels fall in the presence of purulent secretions.[4] Following drainage procedures, these levels rise, as do complement factor C3 and C4 levels.[5]

Infectious sinusitis accounts for 0.5 percent of all respiratory tract infections,[6] usually occurring in the fall, winter, and spring. Sinusitis may follow a variety of insults, including those from infectious agents or allergens or secondary to pollutants. Predisposing causes include developmental anatomical abnormalities, foreign bodies, dental infections, tumors, trauma, and nasal polyps. Certain drugs, including rauwolfia, may cause mucosal swelling, making the cells more prone to infection.

The most common initial events are infections with viruses: rhinoviruses, adenoviruses, influenza, and parainfluenza viruses. Viral infections may cause cilial hypotonia and lead to bacterial superinfection. In the presence of purulent secretions, the mean oxygen pressure (PO_2) and pH of

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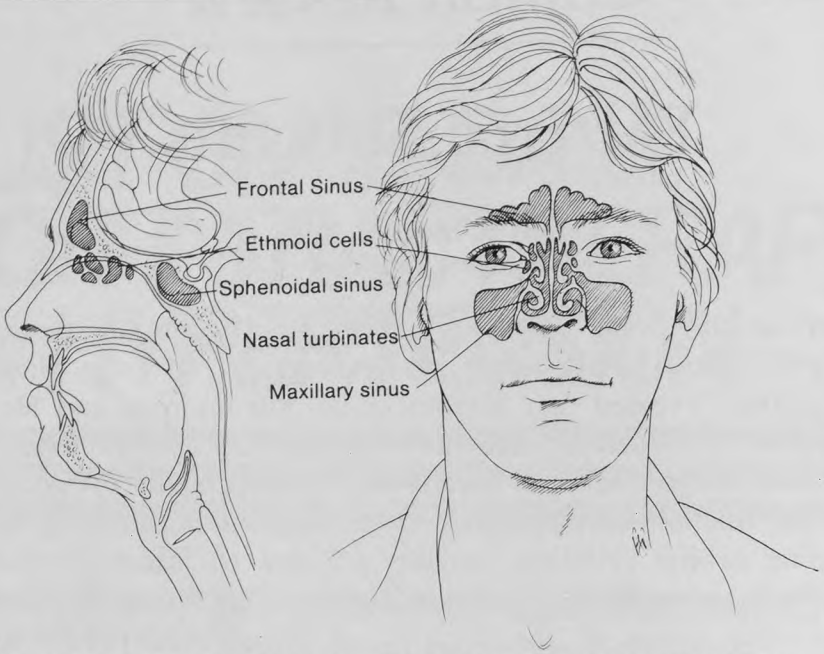

Figure 1. Lateral schematic view (left) and anterior view (right) of paranasal sinuses

secretions both decrease. The change in gas composition and slight increase in acidity not only may affect bacterial multiplication but also may interfere with local protective function and granulocytic bactericidal activity.[7]

Normally the paranasal sinuses are sterile. In the presence of acute inflammation, bacteria may accumulate in the sinuses and in some cases invade mucosal cells, leading to infection.

Chronic sinusitis usually follows repeated infections or insults to the tissues; in some cases normal drainage pathways are blocked and the process is self-continuing.

Table 1. Micro-organisms Usually Encountered in Bacterial Sinusitis

Micro-organism	Percent Occurrence
Streptococcus pneumoniae	30-40
Hemophilus influenzae*	30-40
Streptococcus pyogenes	<5
Staphylococcus aureus*	<5
Miscellaneous gram-negative and aerobes and anaerobes	<5

*Includes β-lactamase-producing strains

Etiology

Since bacteria isolated from the meatal openings or from nasal secretions may not reflect the agents present in the sinuses themselves,[8] antral punctures are necessary to define the true cause of infection. The predominating bacteria cultured in the majority of studies of antral punctures are listed in Table 1. In adults with acute maxillary sinusitis, 64 percent of the strains isolated were equally divided between Streptococcus pneumoniae and Hemophilus influenzae. In some studies, however, Staphylococcus aureus was the second most common isolate.[9,10] In Bridger's study[11] this organism was the sole isolate in 43 patients of 200 examined. Microaerophilic streptococci are often isolated. Of specimens taken from 52 sinuses during a Caldwell-Luc operation, 37 revealed mixed flora and 15 were single isolates. H influenzae were isolated in most cases.[12] Others have confirmed this observation.[13,14] Most of the older literature surveyed revealed the same proportion of bacterial species. Many investigators believe that micro-organisms isolated should be plated and counted. Organisms found in concentrations greater than 10^4 to 10^5 colony-forming units per milliliter probably represent infection, and lower

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counts represent colonization.[15] In one study viruses were cultured from 11 of 15 sinuses, with rhinovirus the most common isolate. The problem then is that the exact etiology cannot be predicted in the individual patient.

The bacterial causes of chronic sinusitis are difficult to evaluate in that anaerobic species are usually isolated; in most cases Peptostreptococcus, Fusobacterium, and Bacteroides sp are found. Concentrations greater than 10^4/mL are considered pathogenic. It has been suggested that low oxygen tension, poor drainage, use of vasoconstrictors, and local inflammation create the milieu for anaerobic bacterial growth.

Noninvasive Aspergillus infection in the immunocompetent host may present clinically as chronic sinusitis, in contrast to the fulminant infection seen in the immunocompromised patient.[16] Fulminant fungal sinus infection secondary to invasion with either Rhizopus (mucor) or Aspergillus has been described in the immunocompromised granulocytopenic host.[17]

The Clinical Syndrome

Acute sinusitis usually presents following an upper respiratory tract infection. Complaints of facial pain, purulent nasal discharge, and headache may predominate. The location of the pain, which often is referred, may suggest which sinus is involved—forehead pain, the frontal sinus; cheek pain and odontalgia, the maxillary sinus; pain at the bridge of the nose and eye, ethmoidal involvement. Sphenoidal involvement usually is referred to the top of the head, and the headache may be explosive or insidious. Approximately 50 percent of patients, usually the pediatric group, have fever. In some cases nasal secretion is absent because of a swollen meatus; in these cases infection may invade deeper structures leading to osteitis and osteomyelitis.

Patients with chronic sinusitis may report nasal stuffiness, fatigue, and other complaints. In some patients with urticaria the only substantial finding was sinus inflammation.[18] Pain in the teeth may represent referred pain from maxillary sinus infection.

In a review of children and adolescents aged 6 to 17 years, maxillary sinusitis (56.6 percent) and combination maxillary and ethmoidal sinusitis (24.5 percent) were the usual findings. Pansinusitis occurred in 10 percent of the children. The most frequently heard complaints were headache, rhinitis, cough, and fever. Physical findings of note were postnasal drainage, pharyngitis, tenderness to pressure over the sinus, and otitis media.[19] Some authors believe that symptoms may not help in differentiating rhinitis from sinusitis; however, a purulent nasal discharge is more suggestive of sinusitis.[20]

Sinus infection may spread to contiguous structures, and signs and symptoms referrable to this spreading may predominate. Orbital cellulitis and, more commonly, retro-orbital and infraorbital cellulitis may be secondary to ethmoid involvement. In one series covering a 25-year period in a children's hospital, 159 of 6,770 patients with sinusitis had orbitofrontal complications.[21] Involvement of the middle ear occurs by way of infection in the oropharynx or invasion of the eustachian tube. In some cases purulent bronchitis and bronchiectasis are associated with sinus involvement, and symptoms from the lower respiratory tract can predominate. Spread of the infection through vascular channels can lead to cavernous sinus thrombosis and osteomyelitis of the skull. In the antibiotic era it is not possible to estimate the incidence of these complications.

If the infection has spread to the intracranial cavity, signs and symptoms of infection at that site may predominate. Infection may spread through the bone of infected areas, a congenital opening, a fracture, or following surgery. Before antimicrobial drugs became available, thrombophlebitis of cortical, meningeal, and intracranial sinuses was seen. Subdural and epidural empyema also occurred, and meningitis was less common. Brain abscess has been reported following partially treated sinusitis or chronic infection. In these cases facial signs and behavior disturbances may reflect this occurrence.

Diagnosis

It may be difficult to diagnose sinusitis from the history, because a severe viral upper respiratory infection mimics the syndrome. Physical examination may reveal swollen, inflamed turbinates, and pus on the posterior pharynx or in the nasal passages. Examination of the teeth by tapping, especially the upper molars, should be performed since the roots are adjacent to the maxillary antrum. Sinusitis may occur after odontogenic infection secondary to extraction with perforation of the floor of the sinus.[22,23] Transillumination of the maxillary and

frontal sinuses with an ordinary flashlight may be of some help in elucidating the physical findings, and both sides should be examined. Decreased transmission of light has been associated with active infection in 25 percent of one series.[12] Normal transmission of light (or normal transillumination) argues against infection.

Radiologic examination of acutely infected sinuses will show thickening of the mucosae and air-fluid levels, or opacifications. Interpretation of these films is more difficult in the presence of chronic infection. Sonography and computed tomographic (CAT) scans are additional diagnostic modalities.

The presence of granulocytes in a Gram stain of the nasal exudate suggests an infectious etiology; a predominance of gram-positive cocci further suggests staphylococcal infection. Cultures of nasal discharges, however, are highly inaccurate in predicting the bacterial etiology from the involved sinus. If an exact etiologic diagnosis is necessary or desired (eg, in a patient failing to respond to empiric therapy or sinus infection associated with cranial complications), direct sinus puncture should be performed. This procedure, which should be performed only by qualified personnel, yields culturable material that elucidates the most common bacteria causing infection.[10,16,24-28] In most cases of sinusitis, however, unless the clinical condition warrants, it is not necessary to puncture the antrum. Material obtained by antral sinus puncture should be cultured aerobically and anaerobically.* Gram stain of this material should be performed seeking evidence of bacterial involvement and inflammatory cells (ie, polymorphonuclear leukocytes).

In patients who appear to have systemic involvement, blood cultures should be obtained. When evidence of spread (eg, to the cranial structures) is suggested, further diagnostic tests including CAT scans and a spinal puncture may be necessary.

Therapy

The decision to begin antibiotics should be made on the basis of history, suggestive physical findings such as fever, sinus tenderness, failure of transillumination or purulent nasal discharge, and corroborating radiologic examination. The choice of agent depends on its penetration into the sinus cavities and most probable etiology. As stated earlier, the most common bacterial isolates in acute infection are Streptococcus pneumoniae and H influenzae strains, followed by Staphylococcus aureus, Streptococcus pyogenes, and other gram-negative species. While amoxicillin and ampicillin might seem drugs of choice with H influenzae, potential problems can arise. β-Lactamase-producing strains from respiratory cultures have been reported nationwide with an average incidence of 22 percent reported in 1981, but some areas have reported up to 38 percent incidence. Penicillin, ampicillin, and amoxicillin are inadequate, since they are susceptible to enzymatic destruction. Cefaclor or trimethoprim-sulfamethoxazole (TMP-SMX) would be adequate in this situation. However, Streptococcus pyogenes, another cause of sinusitis, would not be covered by TMP-SMX. Staphylococcus aureus strains usually are penicillinase producers and, therefore, resistant to penicillin and ampicillin. A penicillinase-resistant semisynthetic oral penicillin such as dicloxacillin could be an alternative choice in this clinical situation. If H influenzae are involved, however, dicloxacillin would not be effective.

Unfortunately, there is no way to differentiate infection with Staphylococcus aureus from other frequently encountered organisms. It is of note that when complications occur, such as subdural empyema, epidural abscess, and orbital cellulitis, S aureus often is the cause.[29] H influenzae infection also may cause rapidly progressive complication such as orbital or facial cellulitis.

Therefore, when the causative bacterial organism is not known in a patient with acute bacterial sinusitis, empiric therapy with the oral cephalosporins, especially cefaclor, represents a logical choice. In fact, cephalosporins would be the only agents available to cover most likely pathogens, especially β-lactamase-producing H influenzae and S aureus, two pathogens most often responsible for serious complications. If a definite bacterial etiology is confirmed by antral puncture, then selective agents may be given.

Tetracycline, including minocycline and doxycycline, has been used to treat sinusitis. However, a small percentage of Streptococcus pneumoniae and Streptococcus pyogenes strains could be re-

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*Recommended media are blood agar, hemin, and NAD-supplemented chocolate blood agar, eosin-methylene blue agar or MacConkey agar, or thioglycollate supplemented with 5 to 10 percent rabbit serum or ascitic fluid (Manual of Clinical Microbiology, ed 3. Washington, DC, American Society for Microbiology, 1980, pp 70-71).

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sistant to tetracycline. More important, approximately 50 percent of S aureus strains may be resistant to tetracycline but more susceptible to minocycline and doxycycline. Side effects with these agents, including sun sensitivity, tooth staining in children, and oral and vaginal candidiasis, appear to be more frequent than with other agents.

In patients who fail to respond to antibiotics within 48 to 72 hours or whose condition deteriorates, surgical drainage may be necessary. Treatment of complications (eg, periorbital abscess, osteomyelitis, and subdural empyema) usually requires surgical intervention.

The use of oral decongestants is controversial; however, most otolaryngologists prefer their use. Antihistamines should be considered in patients with an allergic background or when nonbacterial sinusitis is considered.

The diagnosis and treatment of sinusitis in the immunocompromised host should be carried out rapidly. Following radiography, either maxillary puncture or a Caldwell-Luc procedure* is necessary for provisional diagnosis and therapy when this sinus is involved.

The treatment of fungal sinusitis is an emergency. The usual pathogens are Rhizopus and Aspergillus sp, which invade blood vessels, leading to gangrene and rapid tissue destruction. Treatment consists of surgical debridement and systemic amphotericin B, which in some cases should be instilled directly into the sinus.[17] When diabetes complicates the condition, correction of acidosis and fluid imbalance is also necessary.

Patients with chronic sinusitis should be evaluated in the same manner described for acute bacterial infection. Since bacterial isolates are usually anaerobes, the antibiotic agents described above should be efficacious. The majority of these strains are susceptible to benzylpenicillin.[30] In many of these patients drainage procedures are necessary along with adjunct antimicrobial therapy.

*A surgical procedure opening the maxillary antrum by way of an incision into the supradental fossa opposite the second molar tooth.

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