Sinusitis and its Complications in the Pediatric Patient

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Criteria for the diagnosis and specific therapy of acute sinusitis have not been developed for the pediatric age group. How then, does one distinguish a viral upper respiratory infection (requiring at most symptomatic treatment) from secondary bacterial infection of the sinuses (probably requiring specific antimicrobial therapy)? The continuity of the nasal and sinus membranes would seem to favor a simultaneous inflammatory response when a viral infection or offending allergen is present. Factors that prevent or obstruct the drainage of respiratory secretions are probably important in determining the likelihood of bacterial superinfection.

Knowledge of the embryology, anatomy, and physiology of the sinuses is essential to understanding the onset and progression of clinical sinus infection.

Embryology and Anatomy

All of the paranasal sinuses develop as outpouching of the nasal chamber with varying extensions into their respective bony vaults. The openings or ostia of each sinus differ in size and configuration. These differences contribute to the relative susceptibility of each sinus to inflammatory and infectious process. The general architecture of the nasal chamber and nasopharynx and the dynamic aspects of facial growth also contribute to the relative risk for involvement of each sinus.

The nasal chamber is formed by cartilage anteriorly and bone posteriorly. The alar cartilages and the caudal end of the septum form the entry way into the nose. They then give way laterally to the portion formed by the medial wall of the maxillary and ethmoid bone. The medial wall of the nasal chamber is the septum; the floor of the nose is formed by the palatal bone, and the superior border of the nasal cavity is the perpendicular plate of the ethmoid bone. Posteriorly this chamber opens into the nasopharynx. Along the lateral wall of the nasal chamber are three shelf-like structures - the inferior, middle and superior turbinates. Beneath each turbinate is the corresponding meatus. The frontal, maxillary, and anterior ethmoid sinuses open in the middle meatus; the sphenoid and posterior ethmoid cells open high in the nasal vault into the superior meatus.

The maxillary sinus develops early in the second trimester of fetal life as a lateral outpouching in the posterior aspect of the middle meatus. At birth it is a slit-like structure with its long axis parallel to the attachment of the inferior turbinate and its floor barely below that. The sinus cavity grows in width and height. Laterally, it has reached the infraorbital foramen by age one and passes beneath it by age two. Ultimately at full size the lateral border of the maxillary sinus will reach the lateral orbital rim. The position of the floor of the sinus is determined by the eruption of the dentition. The sinus floor remains above the nasal floor until age eight or nine. In adults, the maxillary sinus floor can be as much as 5 to 7 mm below the nasal floor depending on the dental anatomy. The connecting route between the maxillary sinus and the nose is the primary maxillary ostium, a duct, often almost a

centimeter long. Accessory maxillary ostia have also been described resulting from dehiscences of the nasal wall; these are generally not observed before age four. Infrequently one can also find septae in the maxillary sinus, resulting in separate compartments rather than a single large cavity. The volume of the fully developed maxillary sinus is approximately 12 to 15 mL.

The ethmoid sinus develops in the fourth month of gestation. It is not a single large cavity but a grouping of individual cells, 3 to 15 in number, each with their own opening, or ostium. Aeration of the ethmoid cells is variable leading to a honeycomb radiographic appearance. The cells are small anteriorly and large in the posterior group.

The boundaries of the ethmoid labyrinth include the anterior cranial fossa superiorly, the sphenoid sinus posteriorly, the orbit laterally, the nasal chamber and the middle turbinate inferomedially, and the lacrimal bone anteriorly. The walls of the ethmoid labyrinth are thin, especially in the lateral aspect bordering on the orbit. The lateral wall of the ethmoid sinus is referred to as the *lamina papyracea* ("paper wall"). Purulent infection may spread by direct extension from the ethmoid sinus through natural dehiscences in the bone to involve the orbit.

The variability of frontal sinus development is well known. In adults, 80 per cent will have bilateral but often asymmetric frontal sinuses, 1 to 4 per cent will have agenesis of the frontal sinuses, and the remainder will have unilateral hypoplasia. The frontal sinuses develop either from an anterior ethmoid cell or directly from an anterosuperior evagination of the nasal chamber. The position of the frontal sinus is supraorbital after age four but is not distinguishable from the ethmoid sinus until 6 to 8 years of age. After that, it progresses for another 8 to 10 years before reaching full adult size. Depending on the particular cell in the frontal recess or anterior ethmoid sinus which develops into the frontal sinus, the conduit between sinus and nasal cavity will be either a short and wide ostium or a long and narrow nasofrontal duct.

Although the sphenoid sinus occupies a strategic position in the base of the skull, its slow growth and relative isolation preserve it from frequent infection. Isolated involvement of the sphenoid sinuses is uncommon; however, they may be involved as part of a pansinusitis.

Physiology

Airflow through the nose allows for warming, humidification, and filtering of inspired gases. This flow is affected by septal deflections, turbinate enlargement, and obstructing masses such as polyps or adenoids.

The mucous membrane that lines the sinus cavities is pseudostratified ciliated columnar epithelium with goblet cells and submucosal glands. It is continuous with the respiratory epithelium that lines the nasal cavity. The mucus blanket secreted by the goblet and submucosal cells entraps particulate antigens. As the sinus cilia beat toward the ostia, the mucus and entrapped material are expelled into the nose. Interference with ciliary activity or obstruction of the sinus ostia will result in retained secretions.

Ostial size and configuration are important determinants of drainage. Although the maxillary sinus ostium is relatively large, there may be a 1 cm isthmus connecting the nasal cavity and maxillary sinus. In addition, the position of the maxillary sinus ostium high on the medial border of the sinus cavity prevents gravitational drainage of secretions. The drainage of each ethmoid cell independently into the either the middle or superior meatus is aided by gravity. However, the minute openings of the individual ostia are easily obstructed. The frontal sinus opening can be either large with a short course or narrow with a tortuous nasofrontal duct through the anterior ethmoid complex.

Obstruction of the sinus ostia occurs in acute sinusitis because of generalized hyperemia, edema, and copious mucus. If secondary bacterial infection supervenes, there is an increase in acute inflammatory cells and superficial erosion of the lining epithelium. Measurements of oxygen tension in the maxillary sinus secretions have been uniformly low during acute infection. In addition, the pH is also reduced and carbon dioxide content increased. This may contribute to ciliary dysfunction and impaired phagocytic activity.

Signs and Symptoms

Common symptoms of sinusitis in adults are facial pain, headache, and fever. In children over five years of age, sinusitis is signaled in a similar fashion to that in adults. These older children may complain of headache (retro- or supra-orbital pain) and facial pain or swelling, usually with a history of a recent upper respiratory infection. In the younger child, there are often less specific complaints. Nasal symptoms (purulent discharge and congestion) and cough, when they are protracted (> 7 days) or unusually severe, may indicate sinus infection. The characteristics of the cough are variable - dry or wet, night or day. A history of malodorous breath in the absence of pharyngitis or dental decay may also be a clue to the diagnosis of acute sinusitis in the preschooler.

Physical examination may contribute to the diagnosis of acute sinusitis in the older child but is rarely an aid in the younger child. The swelling and erythema of the nasal mucosa and turbinates are not specific. Cervical adenopathy is usually not impressive, fever is variable, and transillumination is difficult both to perform and interpret. If mucopurulent discharge is seen draining from the middle meatus or if facial swelling or tenderness is appreciated overlying the ethmoid, maxillary, or frontal sinuses, then sinusitis is a probable diagnosis.

Diagnostic Methods

When the history or physical examination suggest the diagnosis of acute sinusitis, several techniques are available to confirm or support the clinical impression.

Transillumination

Transillumination may be helpful in diagnosing inflammation of the maxillary or frontal sinuses. Two methods exist for assessing transillumination of the maxillary sinus. In adults, transillumination has received mixed reviews as a diagnostic aid in sinusitis. The technique is helpful if light transmission is normal or absent. "Reduced" transmission or "dull" transillumination are assessments that correlate poorly with clinical disease. The accuracy of

transillumination has not been evaluated in children. The increased thickness of both the soft tissue and bony vault in youngsters may limit the clinical usefulness of transillumination.

Radiography

In adults with acute maxillary sinusitis, the reliability of radiographic evaluation as a diagnostic tool has been repeatedly demonstrated. The standard radiographic views are an anteroposterior, occipitomental, and lateral projection of the skull. A reduction in the air content of the sinus cavity (semi or complete opacification), accumulation of fluid, or thickening of the mucous membranes are the radiographic changes that are appreciated in acute inflammation.

As a rule, and with only rare exceptions, radiographically normal sinuses can be expected to be fluid free. Completely opaque sinuses will have free fluid or pus by aspiration in 80 to 88 per cent of patients. In sinuses with thickened mucous membranes but central aeration, fluid is present approximately 50 per cent of the time. In general, the greater the mucous membrane thickening, the more likely that fluid is present. Diagnostic accuracy in predicting fluid can be increased in patients with mucous membrane thickening by doing an occipitomental projection in lateral position with the affected side downward.

In chronic maxillary sinusitis, the most frequent radiographic finding is thickening of the mucous membrane; retention cysts and polyps are occasionally seen.

In children, the accuracy of sinus radiographs as a measure of sinus infection has not been validated. Caution concerning radiographic interpretation has been advised by Caffey. He comments that the redundant sinus mucosa and even tears in infancy may produce radiographically opaque sinuses, which are not infected. Similarly, he cautions that asymmetry in facial bone or sinus development and/or overlying soft tissue swelling can produce differences in the apparent aeration of the sinuses and lead to misinterpretation of the radiographs. Other investigators have cast doubt on the diagnostic specificity of radiographs in acute sinus disease in children by demonstrating a similar prevalence of radiographic abnormalities in the sinuses of healthy children, as compared with the sinuses of children with suspected sinus infection. Those who defend the diagnostic value of radiographs in children with acute maxillary sinusitis have failed to document the infection by sinus aspiration. A study correlating clinical signs and symptoms of acute sinusitis with radiography and sinus aspirates is essential to establish the credibility of radiographic findings in the pediatric age group.

Ultrasonography

Two recent reports have evaluated ultrasonography as a diagnostic aid in maxillary sinusitis. The advantages of ultrasonography versus radiography are the use of non-ionizing radiation and better ability to discriminate between mucosal thickening and retained secretions. In a study of adult patients, Mann compared radiography, sinoscopy, and ultrasonography. Ultrasonography was found to be particularly useful in predicting the presence of fluid in patients whose radiographs showed partial or complete opacification. Conformity between the findings at ultrasound and irrigation was observed in 90 per cent of patients. Similarly, a study of 61 children ages 3 to 12 compared ultrasonography to

radiography and sinus aspiration. The agreement between irrigation findings and A-mode ultrasound was 96 per cent. Use of sinus ultrasonography at the Children's Hospital of Pittsburgh has revealed some technical and diagnostic difficulties. More experience will be required to accurately assess the value of ultrasonography in the diagnosis of sinusitis, particularly in children less than three years of age.

Sinus Aspiration

A positive culture of an aspirate of sinus secretions is the sine qua non for diagnosing bacterial sinusitis. Although by no means a routine procedure, aspiration of the maxillary sinus - the most accessible of the sinuses - can be easily accomplished in an outpatient setting with minimal discomfort to the patient. Puncture is best performed by the transantral route with the needle directed beneath the inferior turbinate through the lateral nasal wall. This route for aspiration is preferred in order to avoid injury and permanent damage to the natural ostium. If the patient is unusually apprehensive or too young to cooperate, a short-acting narcotic agent can be used for sedation.

Careful sterilization of the puncture site is essential to prevent contamination by nasal flora. Ten per cent cocaine applied intranasally will achieve mucosal anesthesia and antisepsis. Lidocaine should be injected into the submucosa at the site of the acutal puncture. Secretions obtained by aspiration should be submitted for cell count, gram stain, culture and sensitivity. A bacterial colony count will assure that the culture results reflect actual sinus infection rather than contamination; counts of 10^4 or greater colony forming units per mL give a high degree of assurance of in situ infection. Alternatively a gram stain preparation of sinus secretions may be performed. Bacteria in low colony count (likely contaminants) will not be seen on smear.

Indications for sinus aspiration in patients with suspected sinusitis include: clinical unresponsiveness to conventional therapy; sinus disease in an immunosuppressed patient; severe symptoms such as headache or facial pain; and life-threatening disease at the time of clinical presentation.

Microbiology

The use of various techniques to obtain, transport, and culture maxillary sinus secretions has resulted in differing and often contradictory reports regarding the microbiology of sinusitis. Failure to describe the patient population studied or eliminate patients with partial antimicrobial therapy further complicates comparison of various investigations. However, a knowledge of the bacteriology of secretions obtained directly from the maxillary sinus by needle aspiration (with careful avoidance of contamination from mucosal surfaces) is a necessary guide to specific antimicrobial therapy.

The role of anaerobic bacteria as pathogens in sinusitis has only recently been examined with adequate attention to anaerobic transport and culture techniques. Poor drainage of the inflamed sinus results in a lower pH and oxygen pressure, thereby providing an excellent environment for the growth of anaerobic bacteria. However the growth of anaerobic bacteria may be impaired in sinus secretions obtained by irrigation since irrigation raises the oxygen pressure and dilutes bacterial titers. Finally, few studies have looked for viral agents

as a cause of sinus infection despite evidence that viruses alone may produce acute sinus disease.

Acute Sinusitis

Two studies, performed 15 and 30 years ago, using specimens obtained by aspiration of free fluid from the maxillary sinus of adults, showed a predominance of *Streptococcus pneumoniae* and *Haemophilus influenzae* in patients with acute or chronic sinus infections. The routine comparison and excellent correlation of gram stains of the fluid with culture results strengthens the validity of these investigations, although quantitation of the bacteria was not done. More recent reports echoed these results and also noted a significant minority of infections to be caused by anaerobes, again in both acute and chronic cases. The most convincing data reported, in two elegant studies with careful attention to bacteriologic technique, show nontypable *H. influenzae* and *S. pneumoniae* to be the most commonly found pathogens, accounting for approximately 65 per cent of all significant bacterial strains recovered. Other bacteria implicated include Neisseria species, *Streptococcus pyogenes* (group A), and alpha hemolytic streptococcus. Mixed infection with heavy growth of two bacteria was occasionally found, although most cultures grew only a single organism. Viruses were recovered from 11 of the 70 positive specimens; there were six isolates of rhinovirus, three of influenza A, and two of parainfluenza virus. Five of these 11 specimens also had significant growth of bacteria.

A recent study performed in children with acute maxillary sinusitis has shown the bacteriology of sinus secretions to be similar to that found in adults. The predominant organisms include *S. pneumoniae, H. influenzae* (nontypable), and *N. catarrhalis.* Several viral isolates including adenovirus and parainfluenzae were recovered.

Chronic Sinusitis

Three studies performed with adequate microbiologic methods have examined the bacteriology of chronic sinusitis in adults. Anaerobic bacteria were the predominant pathogens in two of the studies, but *H. influenzae* and *Streptococcus viridans* predominated in the third. One of these studies also found a light growth of *Staphylococcus aureus* in a significant number of specimens. These discrepancies may be explained by differences in the subjects studied: some had chronic sinus disease for many years; others, more likely, had acute exacerbations of chronic conditions.

The single report of probable chronic sinus infection in children reviewed the bacteriology of sinus irrigations in patients undergoing myringotomy and tube placement. Unfortunately the patient population was poorly described and neither quantitation of bacteria nor gram stains of secretions were performed. The report suggests that *H. influenzae* may be the most frequent bacteria recovered in chronic maxillary sinus infection in youngsters.

Surface Cultures

It would be desirable to culture the nose, throat, or nasopharynx in patients with acute sinusitis if the predominant flora isolated from these surface cultures was predictive of the

bacteria recovered from the sinus secretions. A particularly rigorous study in adults demonstrates that there is no correlation between cultures obtained from the anterior nares or nasal vestibule and cultures of the sinus aspirate in acute maxillary sinusitis. Similarly, in a study of pediatric patients with acute maxillary sinus infection, a comparison of the predominant flora in nasopharyngeal and throat cultures (processed semiquantitatively) with the bacteria isolated from maxillary sinus secretions showed no correlation. Comparison of results of cultures obtained from the middlemeatus (with a nasal speculum) and those obtained from the sinus secretions shows a better correlation than the studies previously cited; however, the authors state that "in an individual patient with sinusitis it is more reliable to base therapy on the results of previous bacteriologic investigations than on the individual bacterial findings in the nose". We agree with this statement and cannot recommend the use of surface cultures as a guide to the bacteriology and therapy of acute sinusitis.

Treatment

Therapy for acute maxillary sinusitis in the preantibiotic era consisted of sinus aspiration and irrigations. The current availability of numerous antimicrobial agents, to which the bacteria recovered from sinus secretions are susceptible, prompts consideration of antimicrobials in lieu of multiple irrigation procedures in the treatment of sinus infection. The objectives of antimicrobial therapy of acute sinus infection are sterilization of the sinus secretions, prevention of suppurative orbital and intracranial complications, achievement of a rapid clinical cure, and prevention of chronic sinus disease.

Conflicting reports appear in the literature regarding efficacy of antimicrobials in the treatment of acute sinus infection in adults as judged by radiographic resolution and findings at subsequent irrigation procedures. An array of antimicrobial agents and varying dosage schedules make comparisons of different studies difficult and discrepancies hard to explain. However, several points emerge: (1) Appropriate antimicrobials eradicate susceptible microorganisms in sinus secretions whereas inappropriate agents fail to do so. (2) In order to accomplish sterilization of the sinus secretions, a level of antimicrobial agent exceeding the minimum inhibitory concentration of the infecting microorganism must be present in the sinus secretions. (3) In some instances in which adequate antimicrobial levels within sinus secretions are documented, sterilization of secretions is still not accomplished. This observation points to the importance of local defense mechanisms (such as ciliary activity and phagocytosis) which may be impaired inthe altered environment within purulent sinus secretions (decreased partial pressure of oxygen, increased carbon dioxide pressure, and decreased pH). Therefore, irrigation and drainage of sinus secretions may be required in some patient. (4) There does appear to be a decrease in the serious suppurative orbital and intracranial complications of paranasal sinus disease consequent to the use of systemic antimicrobials. (5) No controlled prospective studies comparing treatment regimens have been performed in the pediatric age group.

Antimicrobials

Until the results of controlled, prospective studies comparing treatment regimens are available, medical therapy with an antimicrobial agent is recommended in children diagnosed to have acute maxillary sinusitis. The relative frequency of the various bacterial agents suggests that ampicillin (100 mg/kg/day in four divided doses), or amoxicillin (40 mg/kg/day

in three divided doses) are appropriate agents. In areas in which ampicillin-resistant organisms are prevalent or when the patient is allergic to penicillin, or when there has been an apparent antibiotic failure, several alternative regimens are available. The combination agent sulfamethoxazole-trimethoprim (prescribed on the basis of 40 and 8 mg/kg/day respectively divided into two doses) has been shown to be efficacious in acute maxillary sinusitis in adults. It is important to remember, however, that this agent may be ineffective in patients with group A streptococcal infections. The new oral cephalosporin cefaclor (prescribed at 40 mg/kg/day in three divided doses) may be another substitute agent. Likewise, the combination of erythromycin-sulfasoxazole (prescribed on the basis of 50 and 150 mg/kg/day in four divided doses) is also suitable.

Decongestants and Antihistamines

The effectiveness of antihistamines or decongestants or combination antihistamine-decongestants applied topically (by inhalation) or administered by mouth in patients with acute or chornic sinus infection has not been adequately studied. Limited investigation of specific agents in clinical rhinitis have shown that some produce a decrease in nasal resistance. However, in the one study performed on patients with sinorhinitis, oral phenylpropanolamine did not significantly increase the size of the maxillary ostium. The effectiveness of any of these agents on the ultimate course of the respiratory illness or the incidence of complications has not been examined.

Complications

Orbital Cellulitis

Orbital cellulitis is the most frequent serious complication of acute sinusitis and despite antimicrobial therapy is a potentially life-threatening infection. The orbit is susceptible to contiguous infection from the paranasal sinuses because it is bordered on three sides by the sinuses. The orbital cavity is separated from the ethmoid air cells only by the thin *lamina papyracea,* and natural bony dehiscences are commonly found. In addition, the ophthalmic venous system has no valves; consequently, the extensive venous and lymphoid communications between the face, nasal cavity, pterygoid region, and the sinuses allow flow in either direction. These channels also connect directly with the cavernous sinus and intracranial venous system.

There has been much confusion in the literature concerning infections of or about the eye because of the frequent interchange of the terms inflammatory edema, periorbital, preseptal, and orbital cellulitis. The terms periorbital and preseptal refer to infection or inflammation outside or anterior to the orbital contents. The septum is a connective tissue reflection of periosteum which inserts into the eyelids and provides an anatomic barrier protecting the orbit. Orbital cellulitis implies infection within or involving the bony confines of the orbit. Inflammatory edema is tissue congestion caused by impaired venous drainage.

The child who appears with a "swollen eye" presents a difficult problem in differential diagnosis for the clinician. Sinus infection is a major predisposing cause of a swollen eye but there are other entities to be considered (Table 1). Infected periorbital lacerations, conjunctivities, dacryocystitis, systemic or contact allergy, seborrheic or eczematoid

dermatitis, and nasal vestibular infections may cause swelling about the eye. A last important category of infections are those cases of *H. influenza* type B (HIB) periorbital or so-called preseptal cellulitis. These HIB infections usually occur in children less than two or three years of age. They are characterized by an abrupt onset, rapid progression, and systemic toxicity. The children have high fever and often the periorbital tissue has a violaceous, almost hemorrhagic discoloration. The area is markedly swollen and tender. The texture of the skin is altered and there is induration of the subcutaneous tissue. Examination of the globe will show no proptosis and intact extraocular movements. HIB is frequently recovered from the culture of the blood or tissue aspirate. Because of the young age of most of these patients, interpretation of sinus radiographs is difficult. Since most *H. influenzae* recovered from sinus aspirates are nontypable, the relationship of this acute bacteremic HIB infection to sinusitis is unclear and we prefer to categorize it separately. These children should receive antimicrobial therapy appropriate for potentially invasive HIB disease.

Table 1. Differential Diagnosis of "Swollen Eye" (Excluding Paranasal Sinus-Related Problems)

Periorbital laceration or abrasion	Trauma should be obvious. Cellulitis is most often caused by group A streptococci or coagulase-positive staphylococci
Insect bite	The site of inoculation is usually evident. The surrounding area is nontender but often pruritic.
Allergy	Systemic allergy is usually not strictly unilateral. Contact allergy does not produce local or systemic signs of infection.
Conjunctivitis	The primary site of involvement is the conjunctiva, not the surrounding soft tissue. Slower progression; no systemic toxicity.
Dacrocystitis	Originates in medial lower lid. May progress to moderately severe cellulitis. Radiographic evidence of sinusitis is usually absent.
Dermatitis	Seborrheic or eczematoid skin changes apparent.
Nasal vestibular	Cellulitis begins about nasal vestibular area and produces cellulitis edema of the lower eye lid prior to development of cellulitis in the upper lid or orbit.
Preseptal or periorbital cellulitis	Intense infection in preseptal area usually caused by *H. influenzae* type b. There is often a violaceous or hemorrhagic appearance to the tense, indurated tissue about the eye.

Classification and Treatment

Once the diagnosis of an orbital complication of sinusitis has been clarified, the severity of involvement must be judged. The classification most frequently used in establishing the severity of the orbital cellulitis is shown in Table 2 (modified from Chandler). It is essential to establish the severity of the cellulitis clinically so that appropriate decisions can be made regarding specific therapy and the need for surgical drainage. The first stage of the Chandler classification, and the complication most often seen clinically, is inflammatory edema. This is not an actual infection of the orbit but rather swelling caused by impedance of the local venous drainage. As such, it is a warning of a potentially serious infection within the sinus. This inflammatory edema has also been referred to as "periorbital cellulitis". However, inflammatory edema in which the swelling is soft, nontender, with no induration

and only minimal cutaneous changes can be distinguished from true periorbital cellulitis caused by HIB.

Table 2. Classification of Orbital Cellulitis

I Inflammatory Edema	Inflammatory edema beginning in medial or lateral upper eyelid; usually nontender with only minimal skin changes. No induration, visual impairment, or limitation of extraocular movements.
II Orbital Cellulitis	Edema of orbital contents with varying degrees of proptosis, chemosis, limitation of extraocular movement and/or visual loss.
III Subperiosteal Abscess	Proptosis down and out with signs of orbital cellulitis (usually severe). Abscess beneath the periosteum of the ethmoid, frontal, or maxillary bone (in that order of frequency).
IV Orbital Abscess	Abscess within the fat or muscle cone in the posterior orbit. Severe chemosis and proptosis; complete ophthalmoplegia and moderate to severe visual loss present (globe displaced forward or down and out).
V Cavernous Sinus Thrombosis	Proptosis, globe fixation, severe loss of visual acuity, prostration, signs of meningitis, progresses to proptosis, chemosis, and visual loss in contralateral eye.

Infection from the paranasal sinuses may actually spread into the orbit. This is signaled by intense pain, swelling, and fever accompanied by proptosis, limitation of extraocular movement, and impaired vision. If the infection infiltrates the orbit directly there will be a diffuse cellulitis. Alternatively, there may be a more local accumulation of pus - a subperiosteal or orbital abscess.

If the infection has progressed beyond stage I, then intravenous antimicrobials and hospitalization are mandatory. Cultures of the blood and sinus aspirate should be performed aerobically and anaerobically. A lumbar puncture may be indicated in addition to studies to identify intracranial suppurative complications. Decompression will be required if there is a subperiosteal or orbital abscess: however, orbital cellulitis may respond to antimicrobials without surgical intervention. The choice of antimicrobials should be guided by knowledge of the usual bacteriology of acute sinusitis. If the gram stained smear of the sinus aspirate or abscess drainage shows unsuspected organisms, appropriate antimicrobials should be added until the results of the cultures are known.

Diagnostic Aids

In most instances the cause of the swollen eye can be established by history and physical examination. When sinusitis is the underlying problem, sinus radiographs will be abnormal. The common radiographic patterns are partial or complete opacification, mucous membrane thickening, or an air-fluid level. Usually the ethmoid and maxillary sinuses are involved simultaneously; however, there may be a pansinusitis involving the ethmoid, maxillary, frontal, and sphenoid sinuses. Occasionally overlying soft tissue swelling may

confuse the interpretation of the radiographs. If there is diffuse swelling about the eye including the bridge of the nose, the underlying sinus may appear hazy, even without in situ inflammation. However, there will usually be either ipsilateral or contralateral clouding of the sinuses in areas without overlying soft tissue swelling; this should help to clarify the diagnosis.

Ultrasound scanning on the B mode is occasionally helpful in detecting abscesses in the posterior orbit. However, computerized tomography (CT) is especially useful in defining and localizing the extent of abscesses in the posterior orbit. Simultaneously, a detailed examination of the sinuses can also be obtained by doing additional views on CT. CT should be reserved for patients in whom abscesses are suspected or when orbital cellulitis has not responded, as expected, to medical therapy.

Outcome

Frank orbital complications of sinusitis are fortunately rare. If the diagnosis and appropriate therapy are not carried out promptly, vision may be lost. Severe neurologic sequelae or death may follow cavernous sinus thrombosis.

Intracranial Complications of Acute Sinusitis in Children

Intracranial extension of infection is the second most common complication of acute sinus disease. Although the incidence of suppurative intracranial complications in patients with sinusitis is unknown, paranasal sinusitis is the source of 35 to 65 per cent of subdural empyemas.

Pathology and Pathogenesis

Infection may enter the intracranial compartment by two routes. Direct extension may occur through necrotic areas of osteomyelitis in the posterior wall of the frontal sinus. The underlying dura becomes thickened with inflammatory exudate, and a heavy layer of granulation tissue develops on its outer surface, forming an extradural empyema. Although the dura is generally resistant to infection, bacterial penetration may take place along the course of the small vessels that traverse its thickness. This results in a subdural empyema which then excites an intense inflammatory reaction in the subjacent arachnoid. This direct route of intracranial extension is more commonly associated with chronic otitis infection than with sinusitis.

An alternative route of intracranial bacterial entry is provided by the valveless venous network which interconnects the intracranial venous system and the vasculature of the sinus mucosa. Thrombophlebitis originaing in the mucosal veins progressively involves the emissary veins of the skull, the dural venous sinuses, the subdural veins, and finally the cerebral veins. By this mode of spread, the subdural space may be selectively infected without contamination of the intermediary structure; that is, a subdural empyema can exist without evidence of extradural pus or osteomyelitis. Intracranial extension of the infection by the venous rute is common in paranasal sinus disease, especially in its acute phase or during an acute exacerbation of chronic inflammation.

Further intracranial spread of infection depends on the competence of the arachnoid as a barrier to bacterial invasion. Despite its thinness, the abult arachnoid is relatively impermeable to infecting agents, and bacterial meningitis only rarely complicates subdural empyema. In infants, however, infection may be transmitted freely across a presumably immature arachnoid, and the incidence of bacterial meningitis was 75 per cent in one series of subdural empyema.

Although subarachnoid infection secondary to subdural empyema is uncommon in young children and adults, extensive cortical thrombophlebitis is a frequent complication. The involved gyri are edematous and hyperemic, often showing small foci of infarction. Sometimes septic thrombosis of a major dural sinus occurs, resulting in massive bilateral cerebral edema and hemorrhagic infarction. This explains the appearance of seizures, focal neurologic deficits, and increased intracranial pressure in patients with only a seemgingly insignificant amount of subdural pus. Inasmuch as cortical thrombophlebitis is less common in extradural empyema, focal signs are much rarer in this entity.

The mechanism of brain infection is more obscure. In the majority of abscesses secondary to sinus disease or subdural suppuration, the brain is adherent to the patch of inflamed dura. It is likely that an initial focus of ischemia or necrosis caused by cortical venous occlusion provides an ideal environment for the growth of anaerobic and microaerophilic organisms. Infection is then carried deeper into the white matter by the penetrating cerebral vessels. The resulting area of cerebritis then undergoes central liquefaction while the perimeter is gradually surrounded by a capsule made up of an inner layer of granulation tissue, a middle layer of collagen, and an outermost shell of glial cells. A "mature" abscess capsule takes two to three weeks to toughen. The fibroblasts responsible for capsule formation come from vessel walls. Since the deeper white matter is not as vascular as the cortex, the abscess wall near the ventricle is thinnest, and ventricular discharge of the purulent contents is not uncommon.

Four distinct sites of subdural empyema can be distinguished. The pus (1) may spread diffusely over the frontal-parietal convexity; (2) may be loculated into focal pockets anywhere over the hemisphere, but predominantly over the frontal pole and the occipital cortex (probably due to gravity in the supine patient); (3) may be interhemispheric (parafalcine), or (4) may be under the tentorium in the posterior fossa. Parafalcine suppuration often involves the opposite side by extending underneath the falx; the parafalcine space is also the favored site of recurrent infection.

Clinical Features

Many authors have noted a preponderance of males over females when reviewing suppurative intracranial complications of sinusitis. The peak age is between 10 and 20 years, although younger children are not immune. Four groups of symptoms and signs may be recognized:

Signs of pansinusitis. About 50 to 60 percent of patients with subdural empyema secondary to sinusitis present with signs and symptoms of acute frontal sinusitis or an acute exacerbation of a chronic pansinusitis. There is usually low grade fever, malaise, and frontal headache, often accompanied by marked forehead tenderness. Occasionally, subperiosteal pus

overlying the anterior wall of the frontal sinus may result in dramatic epicranial edema and a painful fluctuance (Pott's puffy tumour).

Signs of increased intracranial pressure. The initial headache worsens despite prolonged treatment with analgesics and oral antibiotics. Vomiting becomes intractable and the level of consciousness deteriorates gradually. This is usually a result of early cerebral edema in the area adjacent to the extradural or subdural pus. With an isolated extradural collection, cortical involvement is less severe and the patient usually remains alert. With subdural empyema, however, stupor and coma may supervene rapidly.

Signs of meningeal irritation. During the stage of depressed sensorium, there is usually evidence of meningeal irritation including nuchal rigidity and photophobia. This reflects an intense inflammatory response in the leptomeninges in contact with a subdural abscess rather than true septic leptomeningitis. Since signs of leptomeningeal inflammation are uncommon in pure extradural suppuration, a subdural accumulation should be suspected if protracted symptoms of fever and headache are accompanied by prominent signs of meningeal irritation.

Focal neurologic deficits. Focal neurologic deficits are usually caused by a combination of local brain compression and cortical venous thrombosis and infarction. A frontoparietal convexity subdural empyema causes contralateral brachiofacial weakness, contralateral conjugate gaze palsy, and expressive dysphasia. Lower limb involvement is usually late. Focal seizures involving the arm and face occur in over 60 per cent of patients with dorsolateral subdural empyema. With a parafalcine empyema, jacksonian seizures often begin in the foot and march upward to include the trunk, the arm, and finally the face. Weakness also primarily affects the lower extremity with sparing of speech and facial motor functions. Bilateral parafalcine collections may present with paraplegia simulating thoracic spinal cord compression.

In the terminal stage, the patient is comatose, hemiplegic, has evidence of generalized and meningeal sepsis, and shows signs of uncal or tonsillar herniation.

Diagnostic Methods

If the diagnosis of intracranial suppuration is suspected from clinical examination, a lumbar puncture should be deferred to avoid brain herniation. The usual cerebrospinal fluid findings associated with subdural or parenchymal abscesses consist of an elevated protein, pleocytosis with a variable neutrophil count, a normal glucose, and a sterile culture ("parameningeal" focus of infection).

Plain radiographs of the skull show evidence of pansinusitis in over 60 per cent of patients. Frontoethmoidal osteomyelitis is seen only when the sinusitis is chronic. In young children, suture diastasis may be the first clue to a raised intracranial pressure.

Both electroencephalography (EEG) and radionuclide brain scar are too nonspecific to have any diagnostic value. Although the EEG may demonstrate focal slowing and decreased amplitude, and radionuclide scan shows increased uptake over an area of empyema, 50 per cent of patients in several recent seris of subdural empyema have had normal EEGs

and radionuclide scans. Moreover, it is impossible, on radionuclide scans alone, to distinguish subdural empyema from subdural hematoma, infarction, cerebritis, and brain abscess.

The typical angiographic findings of a subdural empyema are displacement of the cortical vessels from the inner table of the skull and a midline shift of the anterior cerebral vessels. Inward displacement of the superior sagittal sinus indicates the presence of an extradural collection. With a parafalcine lesion, the proximal portion of the anterior cerebral artery is shifted to the opposite side, whereas the distal portion is displaced to the ipsilateral side away from the falx (the "S" sign). A hypertrophied falcine artery may also be seen. However, small interhemispheric and subfrontal empyemas unable to produce significant vascular displacement have been missed with angiography, and it may be difficult to identify a coexisting parenchymal abscess when extracerebral vascular distortion is prominent.

CT is now recognized to be the most definitive test for the diagnosis of intracranial infection. It is relatively noninvasive, is able to define even small purulent collections with eact localization, and minimizes the risk of missing a concomitant brain abscess or bilateral empyemas. A parenchymal abscess shows up as a lucent center with an intensely enhancing capsule and surrounding edema. With extracerebral pus, the remarkable degree of underlying cerebral edema and midline shift distinguishes it from a subdural or an extradural hematoma.

Treatment

Treatment of the intracranial complications of sinus infection requires antimicrobials, drainage, and excellent supportive care. There is evidence that preoperative meningitic doses of antibiotics may improve survival. Since the predominant organisms isolated from subdural empyema secondary to sinusitis include anaerobic and microaerophilic streptococci, nongroup A streptococci, *Staphylococcus aureus*, and a mixture of Proteus and other gram-negative rods, the initial antibiotic regimen prior to culture and sensitivity results should be a combination of penicillin G, a penicillinase-resistant penicillin, and chloramphenicol. The recovered pus may be sterile owing to partial treatment given beofre the diagnosis made.

Hyperosmolar agents should be given if intracranial hypertension threatens brain herniation. Systemic steroid is prescribed with caution because of its theoretical ability to suppress granulocytic and immune functions. Anticonvulsants should be given prophylactically to protect against a 79 per cent incidence of associated seizures.

Extradural and subdural empyemas should be dealth with by a generous craniotomy. The entire collection of pus can be evacuated and the area profusely irrigated with bacitracin solution under direct vision, and, with a judiciously fashioned flap, the opposite parafalcine space can be explored. Following drainage of an extradural abscess, the dura should be opened to rule out an underlying subdural empyema, since even a well-enhanced CT scan cannot distinguish between a purely extradural empyema and one with an associated subdural empyema. Extradural and subdural drains are left in place for 3 to 5 days for continuous drainage and intermittent antibiotic irrigation. In most cases of subdural empyema, the underlying brain is so swollen that the bone flap must be left out for external decompression. All osteomyelitic bone must be debrided. The frontal sinus is opened widely, its contents exenterated, and its cavity drained.

An underlying brain abscess is best handled by initial intracapsular evacuation and catheter drainage. This minimized iatrogenically induced deficits associated with radical excision of deep-seated lesions located in eloquent areas of the brain, and reduces the problem of multiloculation commonly encountered in repeated percutaneous aspiration.

Postoperatively, the patient should be maintained on intravenous antibiotics for a minimum of 2 to 3 weeks. Intermittent antibiotic irrigation of the abscess or empyema cavities can be done through the catheters until their removal in 3 to 5 days. The shrinking of the abscess can be followed accurately by serial CT scans.

Despite modern diagnostic and surgical capabilities, the mortality associated with subdural empyema and brain abscess remains over 25 per cent. Causes of death and permanent morbidity are related to delayed diagnosis, recurrent suppuration, missed concomitant parenchymal abscesses, extensive cortical and dural sinus thrombophlebitis, and fulminant bacterial meningitis in infants. Early diagnosis remains the most effective way for improving survival.