Chapter 52: Infections

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Humidification, filtering, and temperature regulation are important functions of the nose and paranasal sinuses. The nose and paranasal sinuses are connected through the various sinus ostia, and are lined with ciliated stratified columnar epithelium containing goblet cells.

Inflammation causes increased secretions and edema in the sinonasal mucosa. With progression of the inflammatory components, secretions may be retained within the paranasal sinuses because of altered ciliary function and/or obstruction of the relatively small sinus ostia. The antigravitational placement of the ostia, particularly in the maxillary sinus, also contributes to poor drainage. Sinus obstruction leads to a reduction in the partial pressure of oxygen within the sinuses and a relatively anaerobic environment. These factors create a milieu ideally suited for the growth of bacterial pathogens.

An inflammatory insult, such as an acute exacerbation of allergic rhinitis or a viral upper respiratory tract infection, frequently precedes purulent sinusitis. These inflammatory changes and retained secretions serve as an excellent medium for a bacterial superinfection. A sudden onset of purulent sinusitis in normal sinuses without a predisposing inflammatory episode is unusual except when a massive bacterial inoculation of the sinuses occurs during swimming or when there is direct spread of infection into the maxillary sinus as a consequence of a dental infection or dental manipulation. The proximity of the maxillary tooth roots to the antrum may allow the direct extension of a periapical abscess into the sinus, or the maxillary sinus may be entered during root canal therapy.

Because the lining of the nose and paranasal sinuses is in continuity, inflammation of the nasal cavity is usually associated with inflammation of the sinus lining. Consequently, viral rhinosinusitis may occur without the development of a bacterial infection. Distinguishing between generalized inflammation of the mucous membranes and bacterial sinusitis is important, since the treatment of these two conditions is quite different.

Pathogenesis

Studies have clearly demonstrated that no correlation exists between the growth on cultures obtained from the nose or nasopharynx and the bacteriology of material obtained with sinus aspiration or open antrostomy (Axelsson and Brorson, 1973; Gwaltney et al, 1981; Wald et al, 1981). *Streptococcus pneumoniae* and *Haemophilus influenzae* have been implicated as the primary pathogens in acute bacterial sinusitis (Evans et al, 1975; Gwaltney et al, 1981; Hamory et al, 1979; Wald et al, 1981). Other important pathogens include *Moraxella catarrhalis* and viruses (Gwaltney et al, 1981; Hamory et al, 1979; Wald et al, 1981).

An increasing effort has been made to identify anaerobic bacteria in acute sinusitis, since the reduced oxygen pressure and lowered pH present in an obstructed sinus would be expected to facilitate the growth of anaerobic bacteria. However, anaerobic organisms have been identified in fewer than 10% of patients with acute purulent sinusitis (Axelsson and Brorson, 1973; Carenfelt et al, 1978; Gwaltney et al, 1981; Jannert et al, 1982). In contrast, anaerobic bacteria appear to play an important role in chronic paranasal sinusitis (Frederick

and Braude, 1974; Karma et al, 1979; Su et al, 1983). The most commonly isolated anaerobic bacteria in chronic sinusitis are *Veillonella sp, Peptococcus sp,* and *Corynebacterium acnes.* Brook (1981) demonstrated anaerobic bacteria in the sinus aspirate of 100% of 40 children studied with chronic sinusitis, whereas 14 patients (38%) had mixed aerobic organisms.

Bacterial Infection

Acute sinusitis

Acute sinusitis may be thought of as an abscess or empyema. Treatment should be aimed at providing adequate drainage and eradication of local and systemic infection. In most cases drainage of the involved sinuses can be accomplished medically. Topical vasoconstrictors and systemic antihistamine decongestants frequently provide adequate drainage so that antibiotics can effectively treat the infection. The antibiotic employed should be directed toward the more commonly encountered organisms such as *S. pneumoniae, H. influenzae,* and *M. catarrhalis.* The increasing frequency of lactamase-producing organisms identified in sinusitis should influence the choice of antibiotic, especially in refractory or recurrent cases.

When a patient fails to improve in 3 to 5 days or continues to have symptoms beyond 10 to 14 days, one usually finds that drainage has been inadequate or a resistant organism has emerged. Under these circumstances specific antibiotic therapy is critical; sinus secretions should be obtained for Gram's stain, culture, and sensitivity studies. Where the infection continues to worsen despite appropriate antibiotic therapy or a complication develops, surgical drainage of the affected sinuses is required.

A number of techniques may be effectively used to establish drainage in acute maxillary sinusitis. The mucosa of the middle turbinate and middle meatus should first be vasoconstricted and the intended puncture site anesthetized. Either the canine fossa or the inferior meatus are ideal puncture sites. The contents of the maxillary sinus can then be displaced by passing a trocar into the maxillary sinus and irrigating with normal saline. The contents of the sinuses will flow out through the natural ostium. In recalcitrant cases functional endoscopic intranasal techniques can be employed using sinus endoscopy equipment to enlarge and then to irrigate through the natural ostium.

The traditional approach to empyema of the frontal sinus that fails to respond to conservative therapy is to trephine the sinus. This is accomplished through an incision made in the medial aspect of the upper eyelid, exposing the floor of the frontal sinus. The bone is removed with a cutting drill, specimens are obtained for culture and sensitivity, and the sinus is irrigated. In situations in which the frontal sinus is involved bilaterally, the intersinus septum can be taken down. A catheter is placed through the wound into the frontal sinus to allow irrigation with a decongestant-antibiotic solution (eg, Neosynephrine 0.25% with bacitracin), which is undertaken twice daily until free flow of the irrigating solution through the nasal frontal duct into the nose is achieved. Unfortunately it often takes 7 to 10 days to restore function of the nasal frontal duct.

Recently surgeons have reported successful management of acute frontal sinusitis with restoration of integrity of the nasal frontal duct by employing functional endoscopic sinus

surgical techniques (Schaeffer and Close, 1990).

Empyema of the ethmoid or sphenoid sinuses that fails to respond to medical therapy may require surgical drainage. This can be undertaken by using "traditional" surgical techniques such as intranasal ethmoidectomy or external sphenoethmoidectomy; however, an increasing proportions of surgeons are now comfortable with accomplishing ethmoidectomy and sphenoidotomy by using endoscopic techniques.

Chronic sinusitis

Chronic sinusitis is characterized by persistent suppuration lasting beyond the acute stages of infection. An arbitrary temporal definition has little real value. However, sinus symptoms lasting greater than 6 to 12 weeks can be considered chronic. Critical to the management of the patient with chronic sinusitis is a thorough search for underlying etiologic factors that are treatable. If, for example, unrecognized or uncontrolled nasal allergy causes the chronic sinusitis, the benefits of surgical management alone are usually limited in extent and brief in duration. Allergic patients should be identified and every effort made to treat the underlying allergies with appropriate desensitization. Topical steroids applied via nasal inhalers usually provide some degree of temporary control, particularly during peak periods of exposure to allergen. Systemic steroids are usually very effective in reducing nasal polyposis, but well-known side effects preclude their use as long-term therapy. When nasal polyposis is severe and refractory to medical therapy, surgical removal of the polyps and the mucosa from which they arise (that is, ethmoid or maxillary sinus mucosa) prevents recurrence for months or years. However, if the underlying allergies remain, the polyps will usually recur.

The initial treatment of a patient with chronic sinusitis should include antibiotics and decongestants. With chronic maxillary sinusitis, irrigation must usually be added to achieve adequate evacuation of exudate. If the patient fails to respond to medical management, surgical intervention aimed at debriding the diseased mucous membrane and establishing improved drainage from the sinuses is required.

Sinusitis in Children

At birth the paranasal sinuses are incompletely developed. The maxillary sinus is but a small air cell attached to the lateral wall of the nose, and the ethmoidal labyrinth is just beginning to pneumatize. The frontal sinus does not develop until 6 to 8 years of age; the sinuses do not complete their development until well into adolescence. The variability that occurs in the rate of pneumatization frequently creates confusion in the evaluation of children with purulent nasal discharge.

Wald et al (1981) outlined the pathophysiology of sinusitis in children. Symptoms in young children usually include halitosis, nasal discharge, and a cough that is characteristically protracted. In older children a history of antecedent upper respiratory tract infection usually exists. Subsequently, purulent rhinorrhea, nasal obstruction, and periorbital pain may develop. Exudate draining from the middle meatus in association with an upper respiratory tract infection and pain is indicative of acute paranasal sinusitis (Jannert et al, 1982).

The diagnosis may be confirmed radiographically. The presence of fluid or clouding of the sinuses correlates well with purulent sinusitis. Completely opaque sinuses will contain pus on aspiration in 80% to 88% of patients (Axelsson and Brorson, 1973; McNeill, 1963; Vusrinen et al, 1962). Sinuses that demonstrate thickened mucous membranes with central aeration contain pus on apsiration 50% of the time (Axelsson and Brorson, 1973). The interpretation of sinus radiographs in children must be undertaken with some caution, since asymmetry of sinus development may lead to misinterpretation (Caffey, 1977).

Children rarely develop allergic nasal polyps before age 10 (Schramm and Effron, 1980); therefore the presence of nasal polyps in a young child requires evaluation for cystic fibrosis.

Aspiration of an infected maxillary sinus in children usually requires a general anesthetic. Indications for sinus aspiration include a failure to respond to antibiotics and decongestants, evidence of sinusitis in an immunosuppressed patient, or the development of a complication of sinusitis.

Nonbacterial Infection

Fungal infections of the nose and paranasal sinuses are uncommon. Aspergillosis, mucormycosis, candidiasis, histoplasmosis, and coccidioidomycosis may occur. *Aspergillus fumigatus* is the most common fungal pathogen found in the paranasal sinuses, and the maxillary antrum is the sinus most often involved.

Recognition of a fungal infection of the paranasal sinuses requires an adequate index of suspicion. Immunosuppressed patients are clearly at highest risk. Radiographic changes may not be pathognomonic, but certain findings are highly suggestive of a fungal origin. Diagnosis is best established through antrostomy and biopsy.

Aspergillosis

Aspergillus is a common saprophyte of soil, dust, decaying matter, fruits, and grains. It is pathogenic in humans, animals, and birds. In the Sudan, aspergillosis is endemic; *Aspergillus* has been cultured from bedding, straw roofs, timber, and the earth floors of Sudanese dwellings. Elsewhere in the world aspergillosis is uncommon. *A. fumigatus* is clearly the most commonly encountered species of *Aspergillus* in the USA. By contrast, *A. flavus* is the sole causative agent identified in the Sudan. Other species, such as *A. niger, A. oryzae,* and *A. nidulans,* are rarely cultured from the nose and paranasal sinuses and may not be pathogenic.

The most common portal of entry for *Aspergillus* is the respiratory tract. Katzenstein et al (1983) have suggested that *Aspergillus* may be commonly present in the sinus mucin of allergic patients. Their study involved histologic review of sinus mucosal specimens from seven allergic patients. Septate hyphae resembling those of *Aspergillus* were found in the mucin in small clusters in all patients, but no evidence of tissue erosion was found. The relevance of these findings in uncomplicated allergic sinusitis is unclear.

An *Aspergillus* infection should be suspected if dark, thick, greasy material is found in the maxillary sinus. Culture of the nose rarely demonstrates the fungus. Even aspiration and irrigation of the maxillary sinus frequently do not yield a positive culture, an antrostomy with removal of infected tissue for histologic examination is usually required. Occasionally the diagnosis can be made on routine hematoxylin-and-eosin staining, but fungal stains such as Gridley's, periodic-acid Schiff (PAS), and methenamine-silver are usually necessary. The hyphae of *Aspergillus* are septate with a uniform diameter of 7 to 10 microm, and the septa branch at a 45-degree angle (Fig. 52-1). By contrast, *Mucor* has broad (10 to 15 microm), irregular, nonseptate hyphae that branch at variable angles. The pseudohyphae of *Candida* are thinner than the true hyphae of *Aspergillus* and are constricted at the level of each septum. Budding yeast forms are also present.

Aspergillus infections may be noninvasive, invasive, or fulminant (Jahrsdoerfer et al, 1979; Romett and Newman, 1982). The noninvasive type of infection results in a fungus ball or mycetoma within the sinus. A single sinus is characteristically involved. Symptoms of chronic rhinosinusitis with nasal congestion and rhinorrhea are frequently associated, although evidence of asthma or atopy is uncommon.

Noninvasive aspergillosis may become invasive given proper circumstances. Invasive aspergillosis may behave much like a malignant neoplasm, with destruction of bone, orbital structures, and occasionally intracranial structures.

Fulminant aspergillosis most commonly occurs in immunocompromised patients. The most susceptible persons are those undergoing irradiation and chemotherapy for myeloproliferative diseases. The fulminant form is characterized by the unrestricted spread of *Aspergillus* organisms well beyond the confines of the paranasal sinuses into orbital and intracranial structures. The hyphae invade dura, vessels, and bone. Treatment requires debridement and high doses of amphotericin B. Antifungal susceptibility testing may be important in determining alternative therapy, especially in intractable cases or when the patient does not tolerate amphotericin B.

Mucormycosis

Mucormycosis is caused by fungi in the class Phycomycetes. These fungi commonly are encountered in the environment within soil and dust. The organism enters the head and neck after inhalation into the nasal cavity. Given a susceptible host, an adequate inoculum, and a virulent strain, the fungus may directly invade vascular channels and cause hemorrhagic ischemia and thrombosis.

This relatively uncommon disease is frequently fatal. Survival for patients with no underlying disease or with diabetes may be as high as 60%. However, a survival rate of only 10% in patients with other, more significant underlying diseases has been reported (Blitzer, 1980). A number of clinical syndromes involving the Phycomycetes have been described. Rhinocerebral or central nervous system mucormycosis is clearly of the greatest importance to the otolaryngologist - head and neck surgeon (Baker, 1971). Pulmonary, gastrointestinal, and cardiac systems may be involved, as may the skin of patients with extensive thermal injuries.

Seventy percent of patients who develop rhinocerebral mucormycosis are in ketoacidosis (McNulty, 1982). This association is believed to result from the fact that the fungus thrives in an acid, glucose-rich medium. Also, patients in ketoacidosis have reduced resistance to the infection because of abnormal leukocytic chemotaxis and phagocytic activity. Other patients at risk include those with severe neutropenia and those receiving high-dose corticosteroids, chemotherapeutic agents, or broad-spectrum antibiotics. Anemia, uremia, and malnutrition have also been associated with mucormycosis.

The initial intranasal signs of infection are usually nonspecific and include engorgement of the turbinates and nasal obstruction. Making the diagnosis at this time requires a very high index of suspicion, usually based on the presence of persistent rhinitis in a severely impaired host. As the disease progresses, ischemia and thrombosis lead to necrosis of the turbinates and the development of a bloody nasal discharge. At this time the nasal turbinates characteristically appear black. The progression to invasive disease is signaled by ptosis, proptosis, ophthalmoplegia, trigeminal anesthesia, and facial palsy.

The diagnosis is best made from biopsy with microscopic evaluation of infected tissue. Special fungus stains (PAS, methenamine-silver) should be requested, although the characteristic hyphae frequently can be recognized on frozen section.

Management of rhinocerebral mucormycosis requires radical surgical debridement in concert with aggressive management of metabolic abnormalities. All tissue grossly involved must be removed, since antifungal therapy alone will not suffice. This frequently requires maxillectomy, sometimes with orbital exenteration. High-dose amphotericin B therapy is then instituted. An alternate-day regimen of 1 mg/kg to a total dose of 2 g is required. Because of the potential for nephrotoxicity, renal function must be closely followed. The addition of low doses of heparin to the intravenous infusion may help diminish amphotericin-related phlebitis, and antiinflammatory agents may help minimize other side effects.

Finn and Farmer (1982) reported on two patients with chronic rhinocerebral mucormycosis who developed chronic osteomyelitis and bony sequestration 8 months and 2 years, respectively, after control of the initial infection. These authors suggest that chronic or relapsing mucormycosis will be recognized more often as a clinical problem when more patients are salvaged from the acute stage of the disease.

Complications

Mucocele

Cystlike lesions of the paranasal sinuses have been recognized for more than 100 years. Rollet introduced the term *mucocele* in 1896 (Evans, 1981). The mucocele is a chronic, cystic lesion of the paranasal sinuses, which is lined with pseudostratified or low-columnar epithelium containing occasional goblet cells. These lesions expand slowly, frequently requiring 10 years or more to become symptomatic. Mucoceles expand concentrically and tend to be round or oval. With increasing size, bony erosion occurs, and the mucocele extends outside the sinus. The findings and symptoms associated with a mucocele depend on its location and the extent of bony erosion.

Some debate centers on the etiology of mucoceles (Close and O'Connor, 1983; Evans, 1981; Ohnishi et al, 1982). Some have suggested that mucoceles develop from obstruction of the sinus ostium, while others believe that mucocele formation occurs when there is obstruction of the duct of a minor salivary gland located within the lining of the paranasal sinus. In fact, either mechanism may give rise to a mucocele.

Symptoms may last from a few days to many years. In a review of 46 patients with frontoethmoidal mucoceles, Evans (1981) has reported that 22 patients (48%) had a history of nasal obstruction caused by polyps preceding the detection of the mucocele. Four other patients (9%) had nasal obstruction of unspecified cause. Two patients had a history of trauma, and two had a history of longstanding allergic rhinitis. Fourteen patients (30%) gave no previous history of nasal abnormality before the onset of symptoms.

Maxillary mucocele

The maxillary mucus-retention cyst is frequently observed as an incidental finding on radiographs of the sinuses or skull. These lesions rarely achieve sufficient size to cause bony erosion. Symptoms are unusual unless the location of the mucocele is such that the natural ostrium of the maxillary sinus is obstructed. Mucus-retention cysts of the maxillary antrum, when asymptomatic, rarely require specific therapy. Prospective radiographic evaluation of these patients frequently shows spontaneous regression over a period of months or years without therapy. When the nature of the lesion is in doubt, the maxillary retention cyst can frequently be aspirated through puncture of either the inferior meatus or the canine fossa.

Frontoethmoidal mucocele

The most common clinically significant mucocele arises in the frontal sinus. Frontal headache and proptosis are the most common presenting complaints, with displacement of the globe in a downward and outward direction. The diplopia caused by displacement of the globe is very bothersome to the patient (Figs. 52-2 and 52-3). Headache and deep nasal or periorbital pain usually occur. In contrast to acute or chronic sinusitis, nasal obstruction and rhinorrhea are unusual findings.

A frontoethmoidal mucocele on radiographic examination consists of clouding of the sinus, loss of the typical scalloped outline of the frontal sinus, and sclerosis of the surrounding skull (Fig. 52-4).

Sphenoethmoidal mucocele

Sphenoethmoidal mucoceles have been well described (Close and O'Connor, 1983; Lundgren and Olin, 1961; Nugent et al, 1970; Sellars and DeVilliers, 1981). These lesions may cause subtle symptoms initially. Headache with occipital, vertex, or deep nasal pain may accompany various ophthalmologic complaints, such as diplopia, visual field disturbance, and globe displacement. Pituitary disturbances are unusual but have been described. Diagnosis of a sphenoethmoidal mucocele is made based on an adequate index of suspicion and the characteristic radiographic findings.

Frontoethmoidal and sphenoethmoidal mucoceles require surgical removal. A

sphenoethmoidal mucocele should be opened widely into the nasal cavity. Frontal sinus mucoceles must be completely removed and the frontal sinus obliterated.

Orbital complications

The spread of infection to involve the orbital structures is the most common complication of sinusitis. Because the orbital contents are separated from the ethmoidal labyrinth only by the thin lamina papyracea, direct extension of infection into the orbit is common. In addition, the ethmoidal veins may become thrombophlebitic, resulting in the spread of infection into the orbit. The first indication of orbital involvement is usually inflammatory edema of the eyelids (Fig. 52-5). With progression of the cellulitis, erythema, progressive proptosis, and fever (to 101°F) occur. Early in the process extraocular muscle function and fundoscopic examination are usually normal, but as the cellulitis progresses, chemosis increases, ophthalmoplegia may develop, and funduscopic examination may demonstrate mild vascular congestion. Although the fever may rise to 102°F to 104°F, the patient usually is not particularly toxic. As the disease progresses, an abscess may form along the lamina papyracea or within the periorbita.

The following classification of orbital involvement caused by ethmoid sinusitis was developed by Chandler (1970) to aid in the correlation of physical findings with orbital disease:

1. Inflammatory edema - lid edema; no limitation of extraocular movement with normal acuity.

2. Orbital cellulitis - diffuse edema of orbital contents; no discrete abscess formation.

3. Subperiosteal abscess - purulent collection beneath periosteum of lamina papyracea; displacement of globe downward and laterally.

4. Orbital abscess - purulent collection within orbit; proptosis and chemosis with ophthalmoplegia and decreased vision.

5. Cavernous sinus thrombosis - bilateral eye findings; prostration; meningismus.

This represents a clinical approach to orbital inflammation aimed at predicting pathology. Today, modern imaging allows the practitioner to improve his diagnostic accuracy.

Purulent frontal sinusitis may also result in orbital complications. The floor of the frontal sinus is frequently the path of least resistance for the infection, since it is usually the thinnest wall (Fig. 52-6). An abscess along the orbital roof causes inferolateral displacement of the globe. When the infection spreads to become osteomyelitis of the frontal bone, a subperiosteal abscess develops over the anterior surface. This overlying soft tissue swelling is called *Pott's puffy tumor* (Fig. 52-7). As the osteomyelitis of the frontal bone advances, there is usually sequestration of necrotic bone through a cutaneous fistula in the upper lid.

Orbital inflammation and cellulitis may be adequately treated with sinus drainage and intravenous antibiotics. The patient should be hospitalized and carefully observed for evidence

of a developing orbital abscess or cavernous sinus thrombosis. When an abscess develops within the confines of the orbit, drainage is mandatory (Fig. 52-8). Failure to recognize and drain an orbital abscess may lead to permanent orbital sequelae and intracranial complications.

Surgical intervention is usually required if:

1. Orbital cellulitis continues to progress despite adequate levels of an appropriate intravenous antibiotic.

2. Physical signs (fever, erythema, edema, proptosis) regress slightly for 2 or 3 days and then stabilize or exacerbate.

3. Definite evidence of an abscess on ultrasound or CT scan exists.

4. Loss of visual acuity occurs.

Surgical treatment should include adequate drainage of the infected sinus (frontal sinus trephination or ethmoidectomy) and drainage of the orbital abscess. The latter should be approached directly through an eyelid or Sewell incision overlying the anticipated abscess site. Most abscesses are found along the lamina papyracea or frontal sinus floor, but if infection has extended through the periorbita, it should be widely opened to ensure adequate drainage. A drain should then be left in place until no further drainage occurs.

Schramm et al (1978) described the clinical characteristics of 134 patients with orbital complications of acute sinusitis whose cases were reviewed retrospectively. The patients ranged in age from 5 weeks to 66 years, but 75% were under 16 years of age. Over 80% of the patients had symptoms of an upper respiratory tract infection in the 2 weeks preceding hospitalization. Cultures were obtained from the conjunctiva, nose and nasopharynx, blood, tissue aspirate, and sinus if surgery was undertaken. In children, *H. influenzae* and *H. pneumoniae* were most commonly cultured from blood or sinus drainage (Fig. 52-9). Conjunctival cultures were frequently misleading. Similarly, nose and nasopharyngeal cultures rarely correlated with the results of percutaneous aspiration or culture of the sinus at surgery. Organisms most commonly cultured in adults included *S. pneumoniae* and microaerophilic *Streptococcus.*

The evaluation of these patients included careful physical examination, sinus radiographs, an ultrasonic B scan, and CT scanning. The B scan proved to be 90% effective in detecting abscesses in the anterior orbit or along the medial wall. CT scanning was more accurate and more valuable in identifying the posterior extension of the abscess.

All patients were hospitalized and treated with intravenous antibiotics. Ninty-seven patients (72%) responded to antibiotic therapy alone, and another 9 patients responded following irrigation of the sinus. Twenty-eight patients (21%) required surgical drainage of an orbital abscess. Incision and drainage were required much more commonly in adults than in children. Thirteen of 33 patients 16 years of age or older required surgery, whereas only 3 of 46 patients younger than 4 years old were treated surgically.

Cavernous sinus thrombosis

Differentiating orbital cellulitis and/or abscess from a developing cavernous sinus thrombosis may be difficult but is of vital importance because of the life-threatening nature of the latter. Infections of the paranasal sinuses or orbits may spread readily to the cavernous sinus, since the absence of valves in the orbital veins allows blood to flow either toward or away from the cavernous sinus.

The most important clinical signs of a developing cavernous sinus thrombosis include bilateral orbital involvement, rapidly progressive severe chemosis and ophthalmoplegia, severe retinal engorgement, fever to 105°F, and prostration. Even with rapid recognition and treatment, the condition may progress to loss of vision, meningitis, and even death. The thrombosis can usually be detected on CT scanning. Treatment includes intravenous antibiotics, drainage of any abscess, and orbital decompression if visual acuity declines. Many physicians advocate heparinization to minimize the progression of thrombosis (Fig. 52-10).

Intracranial complications

The precise incidence of intracranial complications of paranasal sinusitis is not known. However, sinusitis is reported to be the source of 35% to 65% of subdural abscesses (Fig. 52-11) (Hitchcock and Andreadis, 1964; Jenkins et al, 1968; Kaufman et al, 1975). Infection may gain access to the intracranial space by direct extension through a defect in the posterior wall of the frontal sinus caused by trauma or the infection itself. Retrograde thrombophlebitis of the valveless ophthalmic vessels may also offer a route of transmission for infected material into the intracranial cavity (Fig. 52-12). The subdural space may be involved even when no infection of the intervening tissues exists (Kaufman et al, 1975). The arachnoid is a good barrier to bacterial invasion, but thrombosis of dural vessels may lead to focal cerebral abscess, seizures, and neurologic deficits. Meningitis rarely develops in adults, but in infants bacterial meningitis frequently occurs.

Septic thrombosis of major dural sinuses usually results in massive cerebral edema and infarction. This rapidly progresses to decreased mentation, coma, and death.

Evidence of nuchal rigidity in a patient with sinusitis should alert the physician to the possibility of an intracranial complication. Patients should be hospitalized and evaluated carefully for evidence of progression of the disease. Intravenous antibiotics should be instituted. Signs of increased intracranial pressure manifested by headache, intractable vomiting, and deteriorating levels of consciousness must be viewed with gravity in a patient with sinusitis.

The management of intracranial sepsis that develops as a complication of sinusitis requires close collaboration between the neurosurgeon and otolaryngologist - head and neck surgeon. High-dose antibiotic therapy, management of increased intracranial pressure, and prevention of seizures must be instituted. Surgical drainage of an intracranial abscess when present should be planned in conjunction with the establishment of proper drainage of the involved sinus. Failure to recognize and treat the underlying sinus disease may result in recurrent or persistent intracranial disease.