

Impact of sinusitis in cystic fibrosis

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The cause of cystic fibrosis has been determined to be faulty ionic transport of chloride across the apical membrane of epithelial cells lining exocrine glands. The subnormal ionic transport leads to dehydration of extra cellular fluids and the development of thickened inspissated mucous secretions. The vast majority of patients with cystic fibrosis develop sinus disease with panopacification of the sinuses present in 90% to 100% of patients older than 8 months of age. Indications for surgical management of sinusitis in children with cystic fibrosis include (1) chronic nasal obstruction with mouth breathing, (2) chronic purulent draining nasal secretions unresponsive to medical treatment, and (3) persistent headaches thought to be related to sinusitis. Operative therapy is based on computerized tomographic scan findings and can be performed endoscopically. Postoperative management is critical for ensuring successful surgical results. Antibiotics, topical steroids, and cleansing of the surgical fields must be performed on a regular basis to ensure adequate healing. The impact of sinusitis on the cystic fibrosis population is significant. Approximately 20% of patients will eventually require surgical treatment of their sinuses. Chronic sinusitis may cause deformities of the external nasal skeleton, a loss of the sense of smell, and headaches. (J ALLERGY CLIN IMMUNOL 1992;90:547-52.)

Key words: Sinusitis, cystic fibrosis

CF is a genetic disease with autosomal recessive inheritance that affects exocrine gland function. It is the most common lethal genetic disorder in the white population with a carrier rate of 5%.¹ The predominant clinical manifestations of the illness are chronic endobronchial infections associated with progressive obstructive pulmonary disease and intestinal malabsorption resulting from pancreatic insufficiency. Respiratory failure and cor pulmonale remain the primary causes of death in this disease. However, median survival has increased significantly during the past decade from 19 years in 1980 to 28 years in 1990 (30 years in males and 25 years in females).²

In addition to improved survival, the past decade has brought a series of research breakthroughs in understanding the genetic and molecular basis of this disease. The initial insights into the pathophysiology came between 1981 and 1983,^{3,4} when abnormal ion transport was observed in epithelial cells lining exocrine glands. It is now known that the ion transport defect is due to reduced chloride permeability across

Abbreviations used

CF: Cystic fibrosis

CT: Computerized tomography

the apical membrane of these cells⁵ and almost total loss of the chloride secretory response to β -adrenergic agonists.⁶ The net effect of the altered ion movement is a flow of sodium chloride into the epithelial cells with passive water absorption. These fluxes of salt and water lead to dehydration of extracellular fluids in the ducts of the exocrine glands (or airways) and altering viscoelastic properties of exported products such as mucins. Thickened inspissated mucus secretions blocking the excretory ducts of exocrine glands is the hallmark pathologic finding of this disease.⁷

In 1989 the cystic fibrosis gene was identified within region q31 of chromosome 7.^{8,9} The protein product of this gene has also been identified and is called the CF transmembrane regulator.¹⁰ It is a membrane-bound protein that has been shown to be capable of chloride ion transport. In two crucial complementation experiments, scientists showed that the CF ion defect could be corrected by transfecting a normal copy of the CF gene into epithelial cells from patients with CF.^{11,12} The most common defect (70% of alleles) in individuals with CF is a 3 nucleotide deletion (D) in the gene, which results in the absence of the amino acid, phenylalanine (F), at the 508th position of the

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CF transmembrane regulator protein. Thus this CF allele is called DF₅₀₈.¹³ The remaining 30% of alleles are made up of more than 100 distinct mutations, and many have yet to be identified.

With all the recent research advances, it is feasible that gene and/or protein replacement therapy will be available within the next decade.

SINUS DISEASE IN CF

The vast majority of patients with CF develop sinus disease at some time in their lives. Panopacification of the paranasal sinuses is present on x-ray films in 90% to 100% of patients older than 8 months of age.¹⁴ The frontal sinuses rarely develop in these patients, probably because of early onset of sinusitis, which prevents pneumatization.¹⁵ A common association with sinusitis is nasal polyposis, with a frequency ranging from 10% to 32%.^{16,17} Although radiographic changes may be evident at an earlier age, presentation for sinus symptoms and polyposis is most common at ages 5 to 14 years and is rare in adults.¹⁶ The polyps in patients with CF are similar to allergy-related polyps with mucous gland hyperplasia, mucous cysts, and focal transitional or squamous cell metaplasia of epithelium. However, the CF polyps are distinct in that there is a delicate, relatively normal basement membrane, few (<4%) eosinophils, and the mucous glands contain acid mucins rather than neutral mucins.¹⁸

PRINCIPLES OF MUCOCILIARY TRANSPORT

Normal drainage of the paranasal sinuses is a multifaceted process that involves interaction between both the secretory and transport mechanisms. This is dependent on the amount of mucus produced, its composition, the effectiveness of ciliary clearance, resorption of mucus, and the condition of the ostia that provide ventilation and the opportunity for clearance of mucus produced within the sinuses.¹⁹

Normal mucus produced in the healthy state has two layers: an inner serous layer called the sol phase, in which the cilia recover from their active beat, and an outer more viscous layer, the gel phase, which is transported by the ciliary beat. Proper balance between the inner sol and outer gel phase is of critical importance for normal mucociliary clearance to occur. Fine particulate matter becomes incorporated into the gel phase under normal conditions, and together with the gel phase is transported out of the sinuses. Nasal airflow may influence transport mechanisms through suction or negative pressure that promotes transportation of mucus up and out of the sinuses.

The cilia beat in a synchronized and metachronized fashion that is poorly understood. The ciliary beat

frequency is affected by various substances that can increase or decrease the frequency of ciliary beats. The cilia move primarily in the sol phase of the secretion, and the gel phase is actually transported over the sol phase by the cilia when their tips touch this overlying material during their short active beat. The mucosal glands lining the sinuses are regulated predominantly by parasympathetic nerve fibers. Sympathetic fibers may reach the glands through blood vessels supplied to the mucosa.¹⁹

If the composition of the secretion is changed so that the mucus produced becomes more viscous, transport toward the ostium slows considerably, and the gel layer becomes demonstrably thicker. This results in a collection of thick mucus that is retained in the sinus for a varying period of time. If there is lack of secretion or a loss of humidity at the surface that cannot be compensated for by mucous glands or goblet cells, mucus becomes increasingly thick and viscous, and the sol phase may become extremely thin, allowing the gel phase to come in intense contact with the cilia and thus impeding their action. With continuing dehydration, one can see a wormlike movement in the mucous layer with only a minimal movement of the cilia. After this dry mucus is dissolved or removed, the cilia can beat again with the normal speed and pattern. It is this pathologic situation that is exaggerated in cystic fibrosis.

General factors that also affect the ciliary function include long-standing dehydration, medications such as atropine or antihistamines, which slow the action of cilia, cigarette smoke, or chemical toxins.

A reduced oxygen supply also slows ciliary movement even when the blood supply is normal. Enriched oxygenation can raise the frequency of ciliary beat frequency by 30% to 50%. Drugs, such as caffeine, theophylline, digitalis, and phenylephrine (Neo-Synephrine), also increase ciliary beat frequency.

The cause of sinus disease and nasal polyposis in CF is the altered viscoelastic properties of mucous secretions that lead to mechanical obstruction of the sinus ostia,^{20, 21} and impaired mucociliary transport through ostia. Obstruction is followed by a decrease in oxygen partial pressure (PO₂) and an increase in carbon dioxide partial pressure (PCO₂), which leads to ciliary injury, mucosal edema, and further inflammation.²² Swelling and inflammation from respiratory, viral, and bacterial infections may further obstruct flow by the ostia. Patients with CF appear to be no more susceptible to common respiratory viral infections than unaffected siblings.^{23, 24} In contrast, their upper and lower respiratory tracts are chronically colonized with bacteria, principally *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and nontypeable *Haemophilus influenzae*.

mophilus influenzae.¹ A study of bacterial cultures from transantral maxillary sinus aspirates of 20 patients with CF found that 19 of 20 (95%) had at least one positive ($\geq 10^4$ colony-forming units/ml) bacterial culture.²⁵ The most common pathogens recovered were *P. aeruginosa*, *H. influenzae*, and streptococci. *S. aureus* was isolated from only one patient.

ROLE OF ATOPY IN CF SINUS DISEASE

Several reports have reviewed the potential association between atopy and both upper and lower respiratory tract symptoms in CF.^{16, 26-30} Stern et al.¹⁶ found that the incidence of respiratory allergy symptoms (rhinitis or asthma) among patients with nasal polyps was 39%. In patients with CF, a high prevalence of recurrent wheezing is noted,²⁷ which may be related to the underlying endobronchial³¹ infections. However, the frequency of other allergic symptoms, such as pollen-induced asthma, seasonal allergic rhinitis, and eczema, are similar to that in normal children.³¹ CF has a high prevalence of positive skin prick test reactions.²⁶ The most common positive response is to *Aspergillus fumigatus*^{30, 32} and has been reported to be as high as 35% to 60%.³⁰ It appears that patients who are colonized with *P. aeruginosa* are most likely to be skin prick test-positive to *A. fumigatus*.³² The prevalence of positive skin tests to nonmold allergens is similar to control subjects.³⁰

CLINICAL PRESENTATION OF SINUS DISEASE

The presentation of sinus disease in these patients is often subtle. The vast majority do not have symptoms³³ in spite of sinus opacification on radiographs. Headaches are noted in adolescent and adult patients but rarely in preteenage patients. Most younger patients (or their parents) report symptoms related to nasal obstruction and purulent rhinorrhea,^{16, 33} including mouth breathing, snoring, postnasal drainage with morning cough, halitosis, and constant throat clearing. Because the frontal sinuses rarely develop in patients with CF,¹⁵ most symptoms are related to ethmoid and maxillary disease.

The most common findings on physical examination are related to chronic nasal obstruction and purulent drainage. The younger child is frequently mouth breathing, and thick purulent discharge is present in both nares and in the posterior pharynx. The posterior pharynx will also often have a "cobblestone" appearance. Anterior rhinoscopy reveals swollen, erythematous nasal mucosa and/or polyps.¹⁴ Broadening of the nasal bridge is seen in some school-age and adolescent patients³⁴ because of thickening of the bony walls of the ethmoid sinuses and in severe cases may

lead to proptosis. Tenderness on palpation of sinuses is a rare finding.

DIAGNOSTIC EVALUATION

The diagnosis of symptomatic sinusitis is usually based on clinical presentation. Bacterial cultures are sometimes collected from the nasopharynx or posterior oropharynx. No studies have compared nasopharyngeal and/or oropharyngeal cultures with transmaxillary sinus aspirates. Studies^{35, 36} comparing nasopharyngeal and oropharyngeal cultures with bronchial specimens have shown oropharyngeal cultures to be more accurate; nasopharyngeal cultures are frequently contaminated with indigenous upper respiratory tract flora. Our experience has shown that respiratory viral fluorescent antibody studies are frequently uninterpretable because of the thick mucus and cellular debris.

As noted above,¹⁴ radiographic studies demonstrate opacification of maxillary and ethmoid sinuses as well as poorly developed frontal sinuses. It is unusual to detect acute changes at the time of increased sinus symptoms. In recent years, CT of the sinuses has been more useful.³⁷

TREATMENT

The initial treatment for sinusitis in the CF population is oral antibiotics.^{16, 20, 33} Because it is unusual to collect a transmaxillary aspiration culture, empiric use of antibiotics is common. The duration of therapy should be a minimum of 3 weeks and frequently 4 to 6 weeks.

Nasal steroids have gained increased acceptance for treatment of nasal polyps, although no controlled studies have documented efficacy in this population. Antihistamines are of limited value unless the patient has a clear history of seasonal allergies and may lead to thickening of mucous secretions.

A large number of patients with CF have no success with medical treatment and must be considered for surgical drainage procedures. Indications for surgery include (1) chronic nasal obstruction with mouth breathing, (2) chronic purulent draining unresponsive to medical treatment (minimum of 2 months), and (3) persistent headaches.

OPERATIVE TREATMENT

Operative treatment is directed toward providing drainage of sinuses, access for cleaning and irrigation, and elimination of polyps and inflammation with preservation of normal anatomic landmarks. Once the appropriate indications have been met by any individual patient, preparation for surgery can take place in conjunction with the pulmonary or CF specialist who

manages the patient on a continuous basis. Consultation with the anesthesia staff should also be undertaken to minimize any problems that might ensue as a result of the administration of general anesthesia. Preoperative evaluation should include CT scanning of the paranasal sinuses,³⁷ which can be used as an operative road map. Coagulation studies should also be obtained before surgery to avoid any excessive blood loss as a result of coagulopathies. Once these further criteria have been met, a surgical procedure is planned on the basis of the CT scan and can generally be performed intranasally or endoscopically. The use of Caldwell-Luc-type procedures in which the maxillary sinus is opened through a sublabial incision is generally avoided in pediatric patients because permanent damage to underlying teeth and the sinus mucosa may result from such procedures. Simple removal of any polyps that are present in the nasal cavity will generally result in a shorter interval between surgical procedures, and a thorough extirpation of the source of the polyps should be performed if possible. A complete ethmoidectomy is performed with a sphenoidotomy if indicated. A large middle meatal antral window is created at the site of the natural ostia, and large inferior meatal windows are also created to permit gravitational drainage and adequate moisturizing through irrigation of the nasal cavity. All anatomic landmarks and turbinates are preserved to reduce crusting and drying in the nose that would otherwise ensue if they were removed. Once an adequate surgical procedure has been performed allowing drainage of the ethmoid, maxillary, and sphenoid sinuses, packing may be needed to prevent excess bleeding caused by the degree of inflammation present at the time of surgery. The packing can generally be removed within 24 hours, and the patient may be discharged from the hospital. The patient is instructed to use saline solution irrigations on a regular basis to diminish crusting. The patient is rescheduled to be examined in approximately 1 week, at which time the cavities are thoroughly cleaned and debrided. If necessary, this may need to be performed under anesthesia. After 1 week, the patient is placed on topical corticosteroid nasal spray to diminish any continued inflammatory process occurring within the nasal mucosa.

POSTOPERATIVE MANAGEMENT

The most critical factor in ensuring a successful result subsequent to surgery is related to the postoperative care and management of patients who have undergone surgery. The postoperative care must include treatment with antibiotics indicated by cultures of mucoid debris and mucosa during the surgical pro-

cedure. In addition, frequent cleansing of the cavities and drainage sites created must be performed on a frequent (at least weekly) basis. Once the cavities have healed, topical corticosteroid nasal spray can be used to help prevent the redevelopment of nasal polyposis. Irrigations with normal saline solution are used to prevent the reaccumulation of infected mucus on a daily basis. Once the nasal mucosa and sinus mucosa become stabilized, the patient is followed every 3 to 4 months. Acute exacerbations of sinusitis may be treated with intermittent antibiotics as needed. It is not unusual for the younger patient (<10 years of age) to require multiple surgical procedures.

IMPACT OF SINUSITIS IN CF

Because of the nearly universal finding of pansinopneumonia in the CF population, it would be difficult to evaluate the impact of sinus disease on survival in the CF population. Nasal polyposis is a self-limited aspect of sinus disease, with frequent spontaneous resolution by late adolescence.¹⁶

Sinus disease and polyps, however, may have a significant impact on quality of life and morbidity. Approximately 10% to 20% of patients require surgery for sinus-related disease at some time in their lives. The child with chronic nasal obstruction and purulent rhinitis frequently has difficulty sleeping and night cough. Anosmia and loss of taste are common and may affect appetite. We have frequently demonstrated catch-up in weight gain within 3 months after sinus surgery, and patients report that their ability to smell returns. Chronic mouth breathing may also be associated with halitosis. Cosmetic problems, including broadening of the nasal bridge and protrusion of polyps from the nares, may be of particular concern in the young adolescent.^{16, 34}

Headaches are a common symptom in the older adolescent with CF. Although these patients will frequently associate headaches with exacerbation in their sinus symptoms, in our experience medical and surgical treatment of sinus disease has had little or no impact on the pain. We recommend that further evaluation and treatment of headaches be pursued before any surgical intervention.

The chronic bacterial infection remains localized in the sinus cavities with almost no risk of orbital cellulitis or osteomyelitis. There have been several case reports of brain abscesses,³⁹ but this complication is more likely a result of hematogenous spread rather than direct invasion from the sinuses. The low incidence of invasive disease may be from the frequent use of antibiotics in this population or thickening of bone resulting from chronic inflammatory osteitis.

The effect of sinus disease on bronchial hyperreactivity is not clear. In asthmatic patients the association between sinusitis and exacerbation in reactive airway disease has been reported.⁴⁰ Airway hyperreactivity among patients with CF ranges from 24%⁴¹ to 51%⁴² by inhalation bronchoprovocation with histamine or methacholine. The cause of this hyperresponsive state appears to be chronic inflammation and perhaps epithelial injury resulting from chronic endobronchial infection⁴³ rather than atopy.⁴⁴ It is reasonable to assume that chronic sinus infection and inflammation may contribute to airway hyperreactivity. However, the concomitant lower airway infections in these patients may have a greater impact.

In summary, sinus disease is common among patients with CF and may lead to significant morbidity. All treatment and medical and surgical regimens are transiently effective, and none is curative. Fortunately, the symptoms of sinus disease and polyposis frequently resolve as patients reach adulthood.

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DISCUSSION

Dr. Richardson. Patients with CF have this increased adherence of *Pseudomonas* to the mucosa, which

seems to be very difficult to dislodge for some unknown reason.

Dr. Kaliner. We were curious to see if CF had an inherent mucosal defect, which we actually anticipated. Thus we measured their specific and nonspecific immune molecules in the nasal washings. We were unable to determine any abnormality.

Complications of sinusitis

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Even though they seldomly occur, the complications of sinusitis may be life-threatening. Complications can be local, orbital, and intracranial problems or combinations thereof. Orbital complications are the most frequent, and children with acute ethmoiditis are especially prone to them. To prevent permanent loss of vision, immediate and intense therapy is most important. Intracranial complications can have few symptoms, and discordance between symptoms and severity is not uncommon, which involves the importance of early radiologic diagnosis with computed tomographic or magnetic resonance imaging scans. Orbital and intracranial complications of sinusitis are medical emergencies and must be treated by specialists. Whenever possible, the underlying sinus infection should be drained at the same time. All physicians treating acute and chronic sinusitis must keep the potentially life-threatening complications of sinusitis in mind and remain suspicious because early recognition and treatment are crucial in these cases. (J ALLERGY CLIN IMMUNOL 1992;90:552-4.)

Key words: Sinusitis, infection, intraorbital, intracranial

Although sinusitis is a common disorder, major complications seldom occur. This article presents a brief description of the most frequent, major complications of sinusitis.

Complications occur most frequently in children and patients with depressed immune functions. In children most complications follow acute infections; involvement of the particular sinus determines the type of complication. *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Staphylococcus* are the most frequently cultured bacteria.¹ Severe fungal infections, which mostly occur in immunosuppressed patients, cause diagnostic and therapeutic problems.²

Because the eyes and the brain neighbor the paranasal sinuses, complications of sinusitis are medical emergencies, and patients should be hospitalized and treated by specialists. Furthermore, complications can arise rapidly. For example, a patient may rapidly develop purulent nasal discharge, headaches and fever after swimming or diving. Within a day, the patient may show signs of periorbital swelling, proptosis, chemosis and decreased visual acuity, which indicate the onset of an orbital complication.

Complications of sinusitis can be divided into local, orbital, and intracranial problems, but combinations often occur. For example, a bony erosion of the lamina papyracea caused by acute ethmoiditis can lead to the formation of an orbital abscess, which, if untreated, can spread to the cavernous sinus.

LOCAL COMPLICATIONS

Mucocele or mucopyocele are chronic, cystic lesions of the paranasal sinuses. They grow slowly and expand beyond the limits of the sinus by pressure and

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