Supplement to
THE JOURNAL OF
Allergy AND Clinical Immunology

VOLUME 102 NUMBER 6, PART 2

Preface

Sinusitis develops in approximately 31 million Americans each year. An average of 4 days are lost from work each year because of acute sinusitis. Acute sinusitis typically follows a viral upper respiratory infection or an allergic reaction. Swelling of the nasal mucous membranes may obstruct drainage from the sinus outflow tracts, causing mucus to collect in the paranasal sinuses. This pool of retained secretions may become infected, leading to congestion and inflammation of the sinus mucosa and the classic symptoms associated with sinusitis. Treatment is aimed at killing the overgrown bacteria and cleansing the sinuses.

Four documents comprise this practice parameter on sinusitis: (1) an executive summary that reviews, in narrative format, the key clinical issues considered in the parameter documents; (2) a management algorithm with narrative annotations designed to assist clinical decision making; (3) a document listing only numbered summary statements that is intended to promote rapid review and identification of material comprehensively discussed in the final document; and (4) the complete guidelines document, which is organized so that the numbered, key summary statements precede relevant supporting text and citations of evidence-based publications. This format provides a ready reference for any physician who evaluates and treats a patient with suspected sinusitis. In particular, the algorithm and its accompanying annotations are designed to present a global and useful approach to both diagnosis and management. Clinical decision points are clearly shown, and each of these proceeds step-wise to logical implementation strategies. If further justification is required at any step in the algorithm, the evidentiary-based guidelines text can and should be consulted. In addition, guidance about appropriate referral of refractory cases, either because of treatment failure or for further investigation of possible associated conditions, is provided.

The greatest majority of patients with sinusitis seek care from their primary care physician; in fact, there are more than 18 million office visits to primary care physicians for this diagnosis each year. Various subspecialists (allergists and otolaryngologists) also see cases of sinusitis, especially in those patients who are more difficult to treat. It is incumbent on all physicians treating sinusitis to be knowledgeable concerning the latest information on pathophysiology, diagnosis, and management, especially in light of the rapidity with which infective organisms are able to change their character.

This practice parameter includes anatomic, allergic, immunologic, and physiologic considerations, as well as clinical diagnosis, differential diagnosis, diagnostic testing, and treatment. Predisposing factors, such as allergy, upper respiratory infections, anatomic abnormalities, immotile cilia syndrome, cystic fibrosis, immune deficiencies, and environmental factors (eg, smoking and pollution), will be addressed. Medical and surgical therapies will be discussed.

An initial draft of parameters was prepared by a work group of experts in the field who carefully reviewed the current medical literature. This material then underwent extensive peer review, revision, and annotation by external reviewers and by the Joint Task Force on Practice Parameters for Allergy and Immunology, a national panel of allergist-immunologists appointed by its cosponsoring organizations: the American Academy of Allergy, Asthma and Immunology; the American College of Allergy, Asthma and Immunology; and the Joint Council of Allergy, Asthma and Immunology. The parameters were reviewed and approved by the cosponsoring organizations and thereby represent an evidence-based, broadly accepted consensus opinion.

The Joint Task Force is grateful for the cosponsoring organizations' financial support and encouragement. The Joint Task Force would especially like to thank the many individuals who have donated substantial time and effort in producing this document that is intended to improve the quality of care of many millions of sinusitis patients.

Reprint requests: Joint Council of Allergy, Asthma and Immunology, 50 N. Brockway St., #3-3, Palatine, IL 60067.
J Allergy Clin Immunol 1998;102:S107-44.

0091-6749/98 $5.00 + 0 **1/0/94390**

I. Executive summary of sinusitis practice parameters

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Sinusitis, defined as inflammation of one or more paranasal sinuses, has been characterized as "acute" when lasting 3 to 8 weeks and "chronic" when lasting longer. Viral upper respiratory infections frequently precede subsequent bacterial invasion of the sinuses by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*. These bacteria may also be found in patients with chronic sinusitis, as well as organisms such as *Pseudomonas aeruginosa*, group A *Streptococcus*, *Staphylococcus aureus*, and certain anaerobes. Various fungi can also be found, especially in immunocompromised individuals.

Predisposing factors for sinusitis include allergic rhinitis, occupational rhinitis, vasomotor rhinitis, nasal polyps, rhinitis medicamentosa, and immunodeficiency. In addition, sinusitis is found more commonly in patients with cystic fibrosis, Wegener's granulomatosis, HIV infection, Kartagener's syndrome, immotile cilia syndrome, and tumors. Certain anatomic variations can also predispose to sinusitis.

Prominent symptoms of sinusitis include nasal congestion, purulent rhinorrhea, postnasal drip, facial or dental pain, headache, hyposmia, and cough. Typical signs include tenderness over the sinus cavities, mucosal edema, purulent nasal secretions, increased posterior pharyngeal secretions, and periorbital edema.

Although transillumination and endoscopy may be helpful in the diagnosis of sinusitis in selected individuals, standard radiographs, or better yet computerized tomography, confirm the diagnosis. Magnetic resonance imaging is preferred when fungal sinusitis and various tumors are suspected.

Laboratory evaluation of chronic or recurrent sinusitis may include the following: nasal cytology, sweat chloride tests, ciliary function studies, and tests for immunodeficiency. Nasal cytology is useful in the clinical evaluation of underlying allergic rhinitis, nonallergic rhinitis with eosinophilia syndrome, nasal polyposis, and aspirin-sensitive patients. Quantitative sweat chloride tests for diagnosis of cystic fibrosis should be considered in children with nasal polyps and/or colonization of the nose and sinuses with *Pseudomonas* sp. Tests for immunodeficiency (eg, quantitative immunoglobulins, functional antibody tests, and serum IgE) complement components may be useful if either congenital or acquired immunodeficiency is suspected in cases of recurrent sinusitis.

Paranasal sinus biopsy specimens may be required to determine whether a lesion is neoplastic, confirm the presence of suspected fungal disease, or assess the possibility of granulomatous disease. Nasal mucosa from the posterior portion of the inferior turbinates is the preferred biopsy site for primary ciliary dysfunction.

Medications used as primary or secondary therapy for sinusitis include the following. (1) Antibiotics specific for the spectrum of sinusitis organisms. Although a 14-day course is usually adequate for patients with acute disease, chronic sinusitis should be treated for 8 weeks or longer. The choice of antibiotic is based on the predicted effectiveness in specific locations. However, other factors, such as increased β–lactamase resistance, cost, and potential side effects have to be considered. (2) Antihistamines. Although they are not indicated for use in acute bacterial sinusitis, they may have a secondary role in ameliorating chronic sinusitis in patients with concomitant allergic rhinitis. (3) α-Adrenergic decongestants. These act to reduce turbinate swelling and improve ostial patency by their vasoconstrictive effects. (4) Nasal glucocorticoids. These have been shown to be efficacious adjuncts to antibiotic therapy. (5) Adjunctive therapies such as saline irrigations, mucolytics, and expectorants. These may provide symptomatic benefit in selected cases. (6) Intravenous gamma globulin. The use of this treatment modality for sinusitis is indicated only in individuals with proven functional impairment of humoral immunity.

Although children with cystic fibrosis and concomitant sinusitis generally respond to prolonged treatment with conventional oral antibiotics, older children and adults who are colonized with *Pseudomonas aeruginosa* frequently require oral quinolones or intravenous tobramycin or ceftazidime to control an acute exacerbation.

There is an association between sinusitis and asthma. The incidence of sinusitis in asthmatic subjects ranges from 40% to 75%. Effective medical or surgical management of sinusitis results in both objective and subjective improvement of asthma. In patients for whom medical therapy has failed, functional endoscopic surgery in particular and occasionally other forms of traditional sinus surgery, may result in significant improvement of not only the underlying sinus disease but also of asthma.

Consultation with a specialist should be sought when (1) there is a need to clarify the allergic or immunologic basis for sinusitis; (2) sinusitis is refractory to the usual antibiotic treatment; (3) sinusitis is recurrent; (4) sinusitis is associated with unusual opportunistic infections; and/or (5) sinusitis significantly affects performance and quality of life. Consultation is also appropriate when concomitant conditions are present that complicate assessment or treatment, including chronic otitis media, bronchial asthma, nasal polyps, recurrent pneumonia, immunodeficiencies, allergic fungal disease, granulomas, and multiple antibiotic sensitivities.

II. Algorithm and annotations of sinusitis practice parameters

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A. ALGORITHM

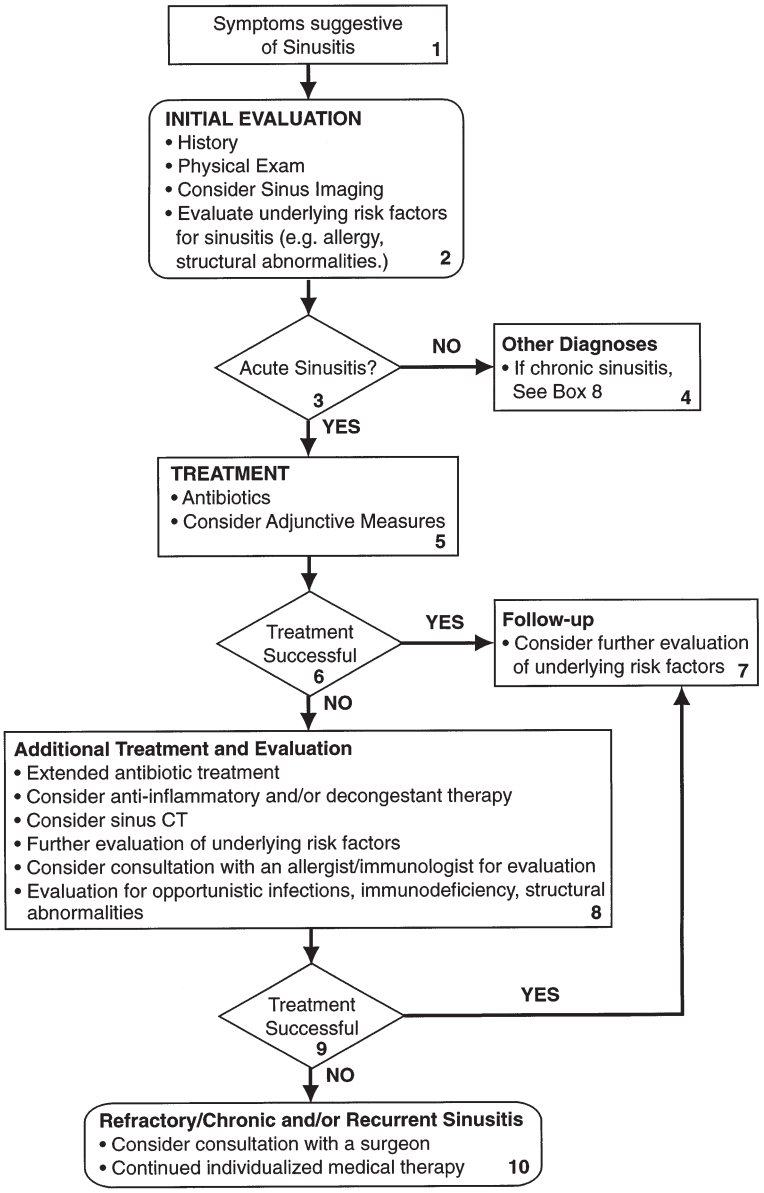

FIG 1 Algorithm for Sinusitus

B. ANNOTATIONS

1. Symptoms suggestive of acute sinusitis

- Acute sinusitis typically presents as a persistent upper respiratory infection.
- In adults, prominent symptoms include nasal congestion, purulent rhinorrhea, postnasal drainage, facial or dental pain, headache, and cough frequently with a more severe nocturnal component.
- Any patient with orbital pain, swelling of forehead, and/or diplopia should be urgently scheduled for evaluation.
- Children with acute sinusitis may also exhibit increased irritability and vomiting occurring in association with gagging on mucus and/or prolonged cough.
- In all age groups, less frequent symptoms associated with acute sinusitis include fever, nausea, malaise, fatigue, halitosis, hyposmia, and sore throat.

2. Office visit

- Review medical history for diagnosis of sinusitis and underlying risk factors.
- General examination includes an evaluation for signs of upper airway and sinus inflammation associated with nasal mucosa edema, purulent secretions, and increased localized blood flow. Typical clinical signs include tenderness overlying the sinuses, dark circles beneath the eyes, and/or periorbital edema. Pharyngeal erythema, lymphoid hyperplasia, and posterior pharyngeal purulent material is also frequently observed.
- Nasal examination in patients with acute sinusitis may reveal mucosal erythema and purulent secretions. Nasal polyps may contribute to nasal congestion and can be a source of recurrent sinusitis by obstructing the sinus ostia. In adults, nasal polyps may be associated with nonsteroidal antiinflammatory drug sensitivity and asthma. Nasal polyps are relatively uncommon in children, and their presence should prompt evaluation for possible cystic fibrosis. Ear examination in patients with suspected acute sinusitis frequently will reveal middle ear abnormalities and associated eustachian tube dysfunction.
- Acute or chronic sinusitis may initiate or worsen asthma and bronchial hyperresponsiveness. Accordingly, chest auscultation and other objective measurements of airflow obstruction, such as spirometry, should be considered in any patient with possible sinusitis and cough.
- Patients with obvious acute sinusitis should be carefully reviewed for any possible evidence of complicating factors, including the presence of external facial swelling/erythema over an involved sinus, visual changes, abnormal extraocular movements, proptosis, periorbital inflammation/edema/erythema/cellulitis, any suggestion of intracranial involvement, or central nervous system manifested as abnormal neurologic signs.
- In general, radiographs are not necessary in making the diagnosis of acute sinusitis. Occasionally imaging studies may be useful to support the diagnosis or provide evidence of the degree of mucosal involvement, thereby guiding more aggressive therapy. Radiographic signs compatible with sinusitis include greater than 6 mm mucosal thickening in the maxillary sinuses, greater than 33% loss of air space volume within the maxillary sinuses, or opacification/air fluid levels in any of the paranasal sinuses. Waters view radiographs are a useful screen in adults and children over 1 year of age because nearly all patients will have a maxillary component to their sinusitis. A limited coronal sinus computed tomographic scan should be consistent if isolated ethmoid sinusitis is suspected. Axial sinus computed tomography is indicated in suspected orbital involvement, and sinus magnetic resonance imaging can provide useful information with related soft tissue involvement.
- Nasal cultures are not reliable for establishing the diagnosis or for determining a specific causative microorganism. Maxillary antrum aspiration for culture is definitive but is indicated only when precise microbial identification is essential.

3. Acute sinusitis

- Acute sinusitis is defined as symptoms and signs for 3 to 4 weeks, although others have modified the definition to 8 weeks. The diagnosis of acute sinusitis is based primarily on the clinical history, the physical examination, and possibly other ancillary evaluations, including nasal cytology or radiographic imaging. In most instances, the diagnosis is made presumptively, and treatment is initiated. Clinical resolution usually occurs within 3 to 4 weeks.

4. Other diagnoses

Differential diagnoses include:

- allergic and nonallergic rhinitis
- upper respiratory infection
- nasal septum deviation
- nasal polyps
- nasopharyngeal tumor, granulomata

5. Treatment

Antibiotics

- Amoxicillin or trimethoprim-sulfamethoxazole (TMP/ SMX) are generally effective, inexpensive, and well tolerated. For patients allergic to both amoxicillin and TMP/SMX, alternatives would include quinolones, cephalosporins, or macrolides. These alternatives should also be considered in situations where microorganisms resistant to amoxicillin or TMP/SMX are prevalent.
- Acute sinusitis generally responds to treatment for 10 to 14 days. Some physicians continue treatment for 7 days after the patient is well. It is important to instruct the patient to complete the course of antibiotics.
- One approach is to start the patient on amoxicillin or TMP/SMX for 5 to 7 days and determine whether the signs and symptoms are improving (clearing of secretions and generally improved well-being). If the

patient is improving, continue this treatment until the patient is well for 7 days (generally a 10- to 14-day course). If after 5 to 7 days the patient has not shown improvement, switch to cefuroxime axetil, amoxicillin/clavulanate, or clarithromycin (or other appropriate, broad spectrum, potent antibiotics) until the patient is well for 7 days.

Corticosteroids

- The use of nasal corticosteroids is rational particularly in patients with underlying rhinitis or associated bronchial hyperresponsiveness.
- Although efficacy has not yet been proven, the short-term use of oral corticosteroids as an adjunct in treating patients with acute sinusitis is reasonable when the patient has had significant anatomic obstruction, invasive nasal polyposis, or has demonstrated marked mucosal edema radiographically.

Saline/mucolytics

- Saline nasal sprays or lavage may be a useful adjunct by liquefying secretions and decreasing the risk of crusting near the sinus ostia.
- There is no conclusive evidence that mucolytics, such as guaifenesin, are useful adjuncts in treating acute sinusitis.

α-Adrenergic decongestants

- Topical decongestants (eg, oxymetazoline and phenylephrine) and oral decongestants (eg, pseudoephedrine) reduce mucosal blood flow, decrease tissue edema and nasal resistance, and may enhance drainage of secretions from the sinus ostia.
- The use of topical decongestants beyond 3 to 5 days may induce rhinitis medicamentosa with associated increased congestion and refractoriness to subsequent decongestant therapy.

Education

- The following comfort measures may be helpful: adequate rest, adequate hydration, analgesics as needed, warm facial packs, steamy showers, and sleeping with the head of bed elevated.
- Prevention measures may include appropriate treatment of allergies and viral upper respiratory tract infections and avoidance of adverse environmental factors, such as cigarette smoke, pollution, and barotrauma.
- Patients should be instructed to phone if symptoms worsen (eg, especially with headache or high fever) or if symptoms have not resolved within 5 to 7 days of treatment (see annotation #10).

6. Treatment successful?

Complete response

- Patient is improved symptomatically to near normal.

Partial response

- Patient is symptomatically improved but not back to normal at the end of the first course of antibiotics.

Poor response

- Patient has little or no symptomatic improvement after the first course of antibiotic therapy.

7. Follow-up

- No further evaluation for resolved, uncomplicated sinusitis.
- Consider further evaluation of underlying risk factors, such as allergic and nonallergic rhinitis and structural abnormalities.

8. Additional treatment and evaluation

- For partial response, continue antibiotic treatment for another 10 to 14 days or consider antibiotic choices listed under "poor responses."
- For poor response to treatment with amoxicillin or TMP/SMX, an antibiotic should be prescribed that covers resistant bacteria. Appropriate choices include amoxicillin/potassium clavulanate, cefuroxime, cefpodoxime, cefprozil, cefixime, ceftibuten, loracarbef, azithromycin, and clarithromycin. In adults, expanded spectrum quinolones, such as ciprofloxacin, levofloxacin, grepafloxacin, or trovafloxacin, may also be a consideration.
- Persistent sinusitis, defined as failure after 21 to 28 days of initial antibiotic treatment may be caused by pathogens not adequately covered by prior antibiotics, the presence of nasal polyps, or noncompliance. The use of broader spectrum single agents such as amoxicillin/potassium clavulanate, cefuroxime, or cefpodoxime should be considered with or without the addition of anaerobic coverage with clindamycin or metronidazole.
- Reinforce the comfort and prevention measures outlined in annotation #5.
- Consider sinus computed tomographic scan if not already done.
- Underlying risk factors should be evaluated in a more detailed manner.
- Consider consultation with allergist/immunologist for treatment of underlying allergic factors and evaluation of opportunistic infection, immunodeficiency, and structural abnormality.

Recurrent sinusitis

- Repeated episodes of acute sinusitis typically 3 or more times per year.
- Patients with chronic or recurrent sinusitis should be evaluated for underlying rhinitis, immunodeficiency, and anatomic abnormalities.

Rhinitis

- Patients with suspected allergic rhinitis in conjunction with sinusitis should be evaluated by an allergist/immunologist competent in the evaluation of IgE sensitization to inhalant allergens.
- Emphasis of therapy for allergic rhinitis includes environmental control, antihistamines and nasal corticosteroids, and, in selected patients, allergen immunotherapy.

- Other rhinitic conditions (vasomotor, NARES, rhinitis medicamentosa) may also lead to sinusitis, and the consultant must be capable of differentiating these conditions and initiating appropriate course of therapy.

Immunodeficiency

- Referral to an allergist/immunologist is particularly indicated in patients with chronic or recurrent sinusitis associated with otitis media, bronchitis, bronchiectasis, or pneumonia and in patients who have undergone prior surgical procedures and continue to experience sinusitis. This evaluation may include measurement of quantitative serum IgG, IgA, and IgM and assessment of specific antibody responses to protein and polysaccharide antigens, such as tetanus toxoid or Pneumovax.

9. Treatment successful?

- See annotation #6.

10. Follow-up

- See annotation #7.

11. Chronic sinusitis

Chronic sinusitis

- Signs and symptoms compatible with sinusitis persisting 3 to 8 weeks or longer.

Chronic sinusitis with ostiomeatal obstruction

- If the patient has a significant nasal septal defect that compresses the middle turbinate into the ostiomeatal complex or obstruction of the sinus outflow tracts caused by middle turbinate deformity or the presence of accessory structures that block sinus drainage, consider consultation with a surgeon. The presence of obstructing nasal polyps, after an appropriate course of treatment that may include a trial of oral corticosteroids, is also an indication for referral. Finally, a patient with recurrent or chronic symptoms despite aggressive medical management of 4 to 6 months may also benefit from widening of the ostiomeatal outflow tract.
- Evaluation should include coronal sinus computed tomography with extra cuts through the ostiomeatal complex to clarify the extent of disease and specific location or locations. Significant anatomic abnormalities might prompt evaluation by an otolaryngologist at the same time that underlying rhinitis and immunodeficiency is explored.
- Evaluation may also include nasal/sinus biopsy in suspected cases of neoplasia, fungal disease, granulomatous disease, or abnormal ciliary structure and/or function.
- In patients with "borderline" anatomic abnormalities, every effort should be made to maximize treatment for underlying rhinitis or immunodeficiency before proceeding with surgical intervention.
- Contemporary surgical therapy involves functional endoscopic sinus surgery.
- Most patients benefit from continued individualized medical therapy, including allergy management, after surgery.

III. Summary statements of sinusitus practice parameters

INDICATIONS FOR REFERRAL; INTERACTIONS BETWEEN CONSULTANTS AND REFERRING PHYSICIAN

- Referral should be considered when serious complications (eg, otitis media, asthma, bronchiectasis, fungal sinusitis, multiple antibiotic allergies, etc.) occur and/or when quality of life is compromised.
- Referral is indicated if an underlying or immunologic basis is suspected.
- The consultant should delineate imunologic/allergic factors, assist in a treatment plan including avoidance measures, pharmacotherapy, education, and immunotherapy.
- An otolaryngologic surgeon should be consulted when anatomical factors impair optimal medical management, biopsy and/or cultures are required, or severe complications ensue.
- The otolaryngologic surgeon should inform the consulting physician about the needs, risks, or possible alternatives of surgical interventions.

DEFINITIONS, ANATOMIC CONSIDERATIONS, SINUS PHYSIOLOGY, MICROBIOLOGY

A. Definitions

- Sinusitis is defined as inflammation of 1 or more of the paranasal sinuses. The most common cause of sinusitis is infection. Classification of sinusitis is frequently based on duration of symptoms and/or imaging characteristics.
- There is no universally accepted classification of sinusitis, but commonly used terminology is as follows:
  - A. Acute sinusitis. Symptoms for 3 to 4 weeks (with some clinicians modifying this definition to 8 weeks) consisting of some or all of the following: persistent symptoms of an upper respiratory infection, purulent rhinorrhea, postnasal drainage, anosmia, nasal congestion, facial pain, headache, fever, cough and purulent discharge.
  - B. Chronic sinusitis. Symptoms for 3 to 8 weeks or longer of varying severity consisting of the same symptoms as seen in acute sinusitis. In chronic sinusitis there should be abnormal findings on computed tomography or magnetic resonance imaging. Some patients with chronic sinusitis may present with vague or insidious symptoms.
  - C. Recurrent sinusitis. Three or more episodes of acute sinusitis per year. Patients with recurrent sinusitis may be infected by different organisms at different times.

B. Anatomic considerations

- The sinuses develop at different ages during childhood.
- The optic nerve, cavernous sinus, carotid artery, and sella turcica are adjacent to the sphenoid sinus. Tumors and infection in the sphenoid sinuses can progress to involve these structures.
- The ethmoid bulla cells can occasionally enlarge into the anterior attachment of the middle turbinate causing variable degrees of pneumatization of the turbinate (concha bullosa). The enlarged turbinate can obstruct ventilation of the middle meatus and may cause sinusitis. Frontal recess cells can be variable in number and size. When present, they can impinge in the nasofrontal duct, leading to frontal sinusitis.
- Septal deviation can predispose to sinusitis if the deviation narrows the middle meatus. With long-standing deviation, atrophy of the middle turbinate and narrowing of the ostiomeatal complex is commonly seen.

C. Sinus physiology

- The sinuses are air-filled cavities with classical pseudostratified ciliated columnar epithelium interspersed with goblet cells. The cilia sweep mucus towards the ostial opening.
- Obstruction of sinus ostia may lead to mucous impaction and decreased oxygenation in the sinus cavities.
- During obstruction of the ostia, the pressure in the sinus cavity can decrease, which in turn causes the symptom of pain, particularly in the frontal region.

D. Microbiology

Bacterial

- In acute sinus disease viral upper respiratory infections frequently precede bacterial superinfection by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*.
- Both *M catarrhalis* and *H influenzae* may be β-lactamase producing and thereby amoxicillin resistant.
- *Streptococcus pneumoniae* are becoming more penicillin resistant. It is not uncommon for 40% of sinus isolates to be penicillin resistant.
- In addition to the organisms mentioned above, the most common organisms in chronic sinusitis, are *Pseudomonas aeruginosa*, Group A streptococcus and *Staphylococcus aureus*, as well as anaerobes such as *Bacteroides* spp, Fusobacteria and *Propionibacterium acnes*.
- In contrast to community-acquired sinusitis, the usual pathogens in nosocomial sinusitis are gram-negative enterics (such as *P aeruginosa*, *Klebsiella pneumoniae*,

Enterobacter spp, *Proteus mirabilis*, *Serratia marcescens*) and grampositive cocci (occasionally streptococci and staphylococci).

Fungal

- *Aspergillus fumigatus* is said to be the most common cause of fungal sinusitis in immunocompetent individuals.
- Allergic fungal sinusitis may be caused by *Aspergillus* spp, *Myriodontium keratinophilum*, *Bipolaris* spp, *Dreschlera* spp, *Curvularia* spp, and *Alternaria* spp.

CLINICAL DIAGNOSIS

A. Clinical history

- Acute sinusitis is typically first seen as an upper respiratory infection that has persisted beyond 5 to 7 days.
- The diagnosis of sinusitis is based on a combination of clinical history with physical examination, nasal cytology, and/or imaging studies.
- Factors that may predispose to sinusitis include allergic or occupational rhinitis, vasomotor rhinitis, nasal polyps, rhinitis medicamentosa, and immunodeficiency. For many patients, the clinical history should address these factors.
- The differential diagnosis of sinusitis includes cystic fibrosis, Wegener's granulomatosis, HIV infection, Kartagener's syndrome, immotile cilia syndrome, and tumors.

B. Clinical examination

- The occurrence of a common cold or persistent allergic or nonallergic rhinitis are common precursors of acute and chronic sinusitis.
- Prominent symptoms include nasal congestion, purulent rhinorrhea, postnasal drainage, facial or dental pain, headache, hyposmia, and cough.
- Typical clinical signs include tenderness overlying the sinuses, mucosal erythema, nasal purulent secretions, increased posterior pharyngeal secretions, and periorbital edema.
- If performed properly in adults, transillumination may be a useful diagnostic technique when combined with abnormal signs and symptoms.
- More detailed examination for underlying risk factors may be required if sinusitis becomes chronic.

- Endoscopy is a quick and safe way to visualize the posterior nasal structures and may aid in the diagnosis.

IMAGING STUDIES IN THE EVALUATION OF SINUSITUS

- Imaging studies may be required when the symptoms are vague, physical findings are equivocal, or there is poor response to the initial management.
- Standard radiographs may be used for detection of acute sinus disease, but they are insensitive, especially in ethmoid disease.
- Computed tomography is the preferred imaging technique for preoperative evaluation of the nose and paranasal sinuses secondary to obstruction of the ostiomeatal complex.
- Although magnetic resonance imaging has limitations in the definition of the bony anatomy, it is particularly sensitive for evaluation of the frontal, maxillary, and sphenoid sinuses for fungal sinusitis and tumors and the differential diagnosis between inflammatory diseases and malignant tumors.
- Ultrasonography has limited utility but may be applicable in pregnant women and for determining the amount of retained secretions.

LABORATORY TESTS

- Laboratory evaluation of chronic or recurrent sinusitis may include nasal cytology, a sweat chloride test, ciliary function studies, and tests for immunodeficiency.
- Nasal cytology is useful in the clinical evaluation of underlying allergic rhinitis, the nonallergic rhinitis with eosinophilia syndrome, nasal polyposis, and aspirin sensitivity.
- Quantitative sweat chloride tests for diagnosis of cystic fibrosis should be considered in children with nasal polyps and/or colonization of the nose and sinuses with *Pseudomonas* spp.
- Tests for immunodeficiency (eg, quantitative immunoglobulins, antibody tests, serum IgE, and complement components) may be useful if either congenital or acquired immunodeficiency is suspected in cases of recurrent sinusitis.

RHINOLARYNGOSCOPY

- Fiberoptic rhinoscopy permits more detailed examination of the anterior and posterior nasal and pharyngeal structures.
- Fiberoptic rhinoscopy may be considered for some patients with chronic or recurrent sinusitis to assess structural abnormalities, fungal disease, or granulomatous lesions.

BIOPSY OF THE NOSE AND PARANASAL SINUSES

- Paranasal sinus biopsies may be required to determine whether a lesion is neoplastic or to confirm the presence of suspected fungal disease and the possibility of granulomatous disease.
- Nasal mucosa harvested from the posterior portion of the inferior turbinate is the preferred biopsy site for primary cilia dysfunction.

CONGENITAL OR ACQUIRED IMMUNODEFICIENCY

- The majority of patients with congenital immune deficiency and associated sinusitis will have defects in

humoral immunity. However, sinusitis is common in acquired immune deficiency (AIDS), in which both humoral and cellular impairments are present. Appropriate laboratory studies of both humoral and cellular immunity include quantitative immunoglobulin assays; specific antibody responses to tetanus toxoid, pneumococcal, or *Hemophilus influenzae* vaccines; measurement of complement components; and key cellular responses.
- The most common congenital immunodeficiency syndromes in this group are common-variable immunodeficiency, IgA deficiency, Wiskott-Aldrich syndrome, ataxia telangiectasia, X-linked immunodeficiency with normal, or elevated IgM and X-linked agammaglobulinemia.

ASTHMA

- The association between sinusitis and asthma has long been appreciated and is generally stated to range from 40% to 75%.
- Although a number of theories have been proposed to explain this relationship, no direct causal factor has yet been found.
- Studies in both adults and children have clearly shown that medical and surgical management of sinusitis results in objective and subjective improvement of asthma.

ALLERGIC RHINITIS

- Allergic rhinitis commonly precedes the development of recurrent or chronic sinusitis because the associated nasal obstruction and inflammation interrupts normal mucociliary clearance and leads to retention of mucopurulent secretions within the sinus cavities.
- Patients with suspected allergic rhinitis in conjunction with sinusitis may benefit from evaluation by an allergist/immunologist who, in most instances, will perform prick/puncture tests to clarify the role of allergies.
- Treatment of allergic rhinitis should include environmental control; medications such as systemic and topical antihistamines, decongestants, nasal cromolyn, anticholinergics, and glucocorticosteroids; and, in appropriate patients, allergen immunotherapy.
- Other forms of rhinitis (eg, vasomotor rhinitis and NARES) also commonly precede the development of recurrent or chronic sinusitis.

CYSTIC FIBROSIS

- Chronic sinusitis is an important source of morbidity in nearly all patients with cystic fibrosis, creating nasal obstruction, post nasal drainage, headache, and potential exacerbation of pulmonary obstruction.
- Pathogens in patients with cystic fibrosis and sinusitis include *Pseudomonas aeruginosa*, *Escherichia coli*, *Staphylococcus aureus*, and *Aspergillus fumigatus*, in addition to the more common polysaccharide-encapsulated organisms.
- Although children with cystic fibrosis and sinusitis generally respond to prolonged treatment with conventional oral antibiotics, older children and adults who are colonized with *P aeruginosa* frequently require oral quinolones, intravenous tobramycin, or ceftazadime to control acute exacerbations.

ANTIBIOTICS

- Antibiotics are the primary therapy for bacterial sinusitis.
- The most common bacteria observed are polysaccharide encapsulated organisms of which 30% to 40% produce β-lactamase.
- Appropriate duration of antibiotic therapy for acute sinusitis is not well defined, although a 14-day course is probably adequate for most patients with acute disease.
- Chronic sinusitis should be treated until the patient is well for 7 days before stopping therapy.
- Choice of antibiotic should be based on predicted effectiveness, cost and side effects.

ANTIHISTAMINES

- There are no data presently to recommend the use of H_1 antihistamines in acute bacterial sinusitis.
- There may be a role for antihistamines in chronic sinusitis, especially in patients with allergic rhinitis.

α-ADRENERGIC DECONGESTANTS

- Both topical and oral decongestants are often used in the therapy of acute or chronic sinusitis because it is thought that they may widen ostial patency and reduce turbinate swelling.
- Prospective studies are lacking and are needed to assess the value of α-adrenergic agents in the treatment of sinusitis.

GLUCOCORTICOSTEROIDS

- The use of systemic steroid therapy for sinus disease has not been studied systematically in a well-controlled or blinded manner.
- A few recent studies suggest that the addition of intranasal steroids as an adjunct to antibiotic therapy is beneficial in the treatment of sinusitis.
- The relative safety of directed therapy with inhaled steroids makes their use a likely mode of treatment for sinusitis, at least for the treatment of any underlying rhinitis.

ADJUNCTIVE THERAPIES INCLUDING SALINE, MUCOLYTICS, AND EXPECTORANTS

- There are inadequate data to recommend use of wetting agents as individual therapy for sinusitis.

- Clinical practice, as well as folklore, supports the use of wetting agents for symptomatic treatment as part of a pharmacologic regimen.
- There are several scientific studies that imply, but do not directly confirm, a role for these agents in sinusitis.
- The safety profile of each agent should be carefully considered for individual patients.
- Use of all these agents as prophylaxis for exacerbations of chronic sinusitis is empiric and not supported by clinical data.

INTRAVENOUS IMMUNE GLOBULIN

- Immunodeficiency is one of the underlying factors for the development of chronic and recurrent sinusitis.
- Intravenous immunoglobulin is indicated for use in patients with impaired humoral immunity.

SURGICAL CONSIDERATIONS

- Antral puncture and irrigation is an office procedure that has a place in the management of acute ethmomaxillary sinusitis refractory to medical therapy or in an immunosuppressed patient in whom early identification of pathogenic organisms is paramount.
- The term *functional endoscopic sinus surgery* is based on the clinical and experimental findings that ostial obstruction is the final common pathway in the development of sinusitis.

IV. Complete guidelines and references

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A. INDICATIONS FOR REFERRAL TO A CONSULTANT AND CONSULTATION WITH A SPECIALIST

i. Indications for referral

a. When the condition or its treatment is interfering with a patient's performance or causing significant loss of school or work on a chronic or recurrent basis or when the patient's quality of life is significantly affected.
b. When there are complications of sinusitis, such as otitis, asthma, bronchiectasis nasal polyps, or bronchitis
c. When there is consideration for an allergic or immunologic basis for the sinusitis.
d. When the condition becomes chronic, persists for several months, or recurs 2 to 3 times per year despite treatment by the primary care physician.
e. When there is the need for complex pharmacology, such as a patient with multiple antibiotic allergies, allergic fungal sinusitis, or resistant pathogens.

ii. What the consultant should provide to the referring physician

a. Clarification of allergic, immunologic, or nonallergic etiologic basis for the patient's condition
b. Assessment of nasal and sinus outflow track anatomy and any contribution these anatomic factors have in the causation of the sinus problems.
c. Identification of specific allergens or other triggers for the patient's condition and education in ways to avoid exposure to these triggers.
d. Assistance in developing an effective treatment plan, including patient education, allergy avoidance, pharmacotherapy, antiinfectious therapy, and immunotherapy if appropriate
e. Provision of specialized services, such as preparation of extracts and provision of immunotherapy.

iii. Indications for referral to an otolaryngologic surgeon

a. When anatomical defects exist that obstruct the sinus outflow track, including the ostiomeatal complex and adenoidal tissues and are thought to be contributing to recurrent or chronic sinusitis.
b. When nasal polyps obstruct the sinus drainage and persist despite appropriate medical treatment.
c. When there is recurrent or persistent sinusitis, despite adequate trials of medical management. Adequate medical management minimally involves multiple courses of antibiotics chosen to cover the spectrum of pathogens anticipated to be causing the disease.
d. For biopsy of the nasal mucosa to rule out granulomatous disease, neoplasms, ciliary dyskinesia, or fungal infections.
e. When maxillary antral puncture for culture or relief of pain is required.
f. For sinusitis complicated by brain abscess, meningitis, cavernous sinus thrombosis, or Pott's tumor (infectious erosion of the ethmoid or frontal sinus).

iv. What the otolaryngologic surgeon should provide the referring physician

a. Assessment of the need for surgical revision of abnormal anatomy and the likelihood that such revision will reduce the recurrent or chronic sinus disease.
b. Determination of whether an adequate trial of medical, allergic, or immunologic treatment has been provided before recommending surgery.
c. Recommendation of specific procedure or procedures for the specific patient and discussion of the merits of alternative approaches.
d. Conservative prediction of the discomfort, inconvenience. and dangers from the procedures proposed.

B. DEFINITIONS, ANATOMIC CONSIDERATIONS, SINUS PHYSIOLOGY, MICROBIOLOGY

Bi. Definitions

SUMMARY STATEMENTS

1. Sinusitis is defined as inflammation of 1 or more of the paranasal sinuses. The most common cause of sinusitis is infection. Classification of sinusitis is frequently based on duration of symptoms and/or the specific sinus involved.
2. There is no universally accepted classification of sinusitis, but commonly used terminology is as follows:
   a. Acute sinusitis. Symptoms for 3 to 4 weeks (with some clinicians modifying this definition to 8 weeks) consisting of some or all of the following: persistent symptoms of an upper respiratory infection, purulent rhinorrhea, postnasal drainage, anosmia, nasal congestion, facial pain, headache, fever, and cough.
   b. Chronic sinusitis. Symptoms for 3 to 8 weeks or longer of varying severity consisting of the same

symptoms as seen in acute sinusitis. In chronic sinusitis, there should be abnormal findings on computed tomography or magnetic resonance imaging. Some patients with chronic sinusitis may be first seen with vague or insidious symptoms.

c. Recurrent sinusitis. Three or more episodes of acute sinusitis per year. Patients with recurrent sinusitis may be infected by different organisms at different times.

Sinusitis is defined as inflammation of 1 or more of the paranasal sinuses. The most common cause of sinusitis is infection. Classification of sinusitis is frequently based on duration of symptoms, the specific sinus involved, and/or imaging characteristics.[1-7] There is no universally accepted classification of sinusitis, but commonly used terminology is as follows:

1. Acute sinusitis. Symptoms of 3 to 4 weeks (some clinicians modify this definition to 8 weeks) consisting of some or all of the following: persistent symptoms of an upper respiratory infection, purulent rhinorrhea, postnasal drainage, anosmia, nasal congestion, facial pain, headache, fever, and cough.
2. Chronic sinusitis. symptoms for more than 4 weeks of varying severity, consisting of the same symptoms as seen in acute sinusitis (others have defined chronic sinusitis as symptoms for up to 8 weeks or longer and have used the term subacute sinusitis to define symptoms lasting 3 weeks). In chronic sinusitis, there should be abnormal findings on computed tomography or magnetic resonance imaging. Some patients with chronic sinusitis may be first seen with vague or insidious symptoms.
3. Recurrent sinusitis. Three or more episodes of acute sinusitis per year. Patients with recurrent sinusitis may be infected by different organisms at different times.

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Bii. Anatomic considerations

SUMMARY STATEMENTS

1. The sinuses develop at different ages during childhood.
2. The optic nerve, cavernous sinus, carotid artery, and sella turcica are adjacent to the sphenoid sinus. Tumors and infection in the sphenoid sinuses can progress to involve these structures.
3. The ethmoid bulla cells can occasionally enlarge into the anterior attachment of the middle turbinate causing variable degrees of pneumatization of the turbinate (concha bullosa). The enlarged turbinate can obstruct ventilation of the middle meatus and may cause sinusitis. Frontal recess cells can be variable in number and size. When present, they can impinge in the nasofrontal duct leading to frontal sinusitis.
4. Septal deviation can predispose to sinusitis if the deviation narrows the middle meatus. With long standing deviation, atrophy of the middle turbinate and narrowing of the ostiomeatal complex is commonly seen.

DEVELOPMENT

The maxillary sinus is the first to begin significant pneumatization between birth and 12 months. It begins to enlarge laterally along the floor of the orbit at about age 3 years. The floor of the maxillary sinus reaches the level of the nose by 12 years of age. Adult size is achieved in midadolescence. Rudimentary ethmoid sinuses are present at birth but do not begin to enlarge until the child is between 3 and 7 years of age. Adult size of the ethmoid sinuses is reached at 12 to 14 years of age. The sphenoid sinuses, which originate from the nasal cupola, do not reach significant size until 4 to 5 years of age. Sphenoid development, which is complete by midadolescence, can be highly variable in the degree of pneumatization of the greater and lesser wings of the sphenoid and the pterygoid processes. The frontal sinuses are the last to develop. Typically not present at birth, the frontal sinuses begin as outgrowths of nasal mucosa into the frontal recess from the middle meatus. They do not usually reach significant size until midadolescence. Like the sphenoid sinuses, the frontal sinuses can be quite variable in their degree of pneumatization. Difference in development between the right and left frontal sinuses are often found within individuals.

ANATOMY

The ethmoid sinuses consist of a complex "honeycomb" of cells varying between 4 and 17 in number. The average individual has 9 cells. The ethmoid sinuses are commonly divided into 2 groups: the anterior cells and the posterior cells. Some authors further separate the ethmoid bulla from the anterior cells**,** calling them the middle ethmoid cells.

A common occurrence is the complete absence of a frontal sinus. The degree of pneumatization of the frontal sinus is variable.

The paired sphenoid sinuses are separated by an intrasinus septum. The optic nerve courses over the lateral superior surface of the sphenoid sinus. The carotid artery within the cavernous sinus is just lateral, and the

maxillary nerve (part of the fifth cranial nerve) is inferolateral anteriorly. The pituitary, located in the sella turcica, can be approached through the posterior superior wall of the sphenoid sinus. Tumors and infection in the sphenoid sinuses can progress to involve these structures.

It is important to realize that ethmoid cells or sinuses are not completely contained in the ethmoid bone. For example, the ethmoid sinus in the course of its development may invade the middle turbinate forming concha bullosa. The ethmoid bulla cells can occasionally enlarge into the anterior attachment of the middle turbinate, causing variable degrees of pneumatization of the turbinate (concha bullosa). The enlarged turbinate obstructs ventilation of the middle meatus and commonly causes lateralization of the uncinate process and narrowing of the ethmoid infundibulum. Similarly, a cell invading the medial floor of the orbit is known as a Haller cell. Those cells outside the ethmoid bone proper are called extramural. These cells are located in the medial inferior orbit and usually form the medial wall of the ethmoid infundibulum. Because of this relationship, they can markedly narrow the infundibulum, causing the obstruction of the maxillary and anterior ethmoid sinuses. Thus Haller cells are frequently present in association with sinus disease.

Frontal recess cells can be variable in number and size. When present, they can impinge in the nasofrontal duct, leading to frontal sinusitis.

Septal deviation can predispose to sinusitis if the deviation narrows the middle meatus. With long-standing deviation, atrophy of the middle turbinate and narrowing of the ostiomeatal complex is commonly seen.

Biii. Sinus physiology

SUMMARY STATEMENTS

1. The sinuses are air-filled cavities with classical pseudostratified ciliated columnar epithelium interspersed with goblet cells. The cilia sweep mucus towards the ostial opening.
2. Obstruction of sinus ostia may lead to mucous impaction and decrease oxygenation in the sinus cavities.
3. During obstruction of the ostia, the pressure in the sinus cavity can decrease, which in turn causes the symptom of pain, particularly in the frontal region.

The sinus cavities are air filled with classical pseudostratified ciliated columnar epithelium interspersed with goblet cells. The cilia sweep mucus towards the ostial opening. Blood flow in the maxillary sinus is roughly estimated to be 100 mL/100 g tissue per minute, which is similar to that found in the nose but higher than that found in the brain. Obstruction of the ostia can lead to mucous impaction and decrease oxygenation in the sinus cavities. This in turn may lead to further complications (discussed in further sections). There is very little gas exchange in the sinuses except during ostial obstruction, when the oxygen concentrations can fall to close to 0% with purulent secretions but not with nonpurulent secretions. The growth of bacteria is facilitated in this anaerobic environment.

During obstruction of the ostia, the pressure in the sinus cavity can decrease, which in turn causes the symptom of pain, particularly in the frontal region.[1] This pressure decrease may range from 20 to 30 mm H_2O, with the lowest pressure being –66 mm H_2O. Transudation may start when the pressure is lower than 20 to 30 mm H_2O below 0. This decrease in pressure is preceded by a transient pressure increase caused by the rise in CO_2, whereas the decrease in pressure is principally caused by O_2 absorption.[2] However, in acute purulent sinusitis, the pressure may sometimes be as high as a 100 mm H_2O.[3] Purulent secretions have a low oxygen content and the pain may be due to a combination of inflammation originating from the mucosa and pressure from the secretions on the inside walls of the sinus.

During deep sea diving, the change in sinus pressure may be very high, causing transudation, bleeding, and edema, especially when pressures exceed 350 to 500 mm H_2O. During flying, there is usually less change in pressure than diving. When there is obstruction of the ostia, changes in sinus pressure similar to that of diving may occur.

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Biv. Microbiology

SUMMARY STATEMENTS

Bacterial

- In acute sinus disease viral upper respiratory infections frequently precede bacterial superinfection by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*. Both *M catarrhalis* and *H influenzae* may produce β-lactamase and thereby be amoxicillin resistant.
- *Streptococcus pneumoniae* are becoming more penicillin resistant. It is not uncommon for 40% of sinus isolates to be penicillin resistant.
- In addition to the organisms mentioned above, the most common organisms in chronic sinusitis, are *Pseudomonas aeruginosa*, Group A streptococcus, and *Staphylococcus aureus*, as well as anaerobes, such as *Bacteroides* spp, Fusobacteria, and *P acnes*.
- In contrast to community-acquired sinusitis, the usual pathogens in nosocomial sinusitis are gram-negative enterics (such as *Pseudomonas aeruginosa, Klebsiella pneumoniae*, *Enterobacter* spp, *Proteus mirabilis, Serratia marcescens*) and gram-positive cocci (occasionally streptococci and staphylococci).

Fungal

- *Aspergillus fumigatus* is said to be the most common cause of fungal sinusitis in immunocompetent individuals.
- Allergic fungal sinusitis may be caused by *Aspergillus*, *Myriodontium Keratinophilum, Bipolaris* spp, *Dreschlera* spp, *Curvularia* spp, and *Alternaria* spp.

1. Bacterial The microbiology of paranasal sinus infections can be anticipated according to the age of the patient, clinical presentation, and immunocompetency of the host.[1-13] In acute sinus disease viral upper respiratory infections frequently precede bacterial superinfection by *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis*.[1-13] Both *Morexella catarrhalis* and *Haemophilus influenzae* may produce β-lactamase and thereby be amoxicillin resistant.

The most common organisms in chronic sinusitis, in addition to the organisms mentioned above, are *Pseudomonas aeruginosa*, Group A streptococcus, and *Staphylococcus aureus*, as well as anaerobes such as *Bacteroides* spp, Fusobacteria, and *P acnes*.[14,15]

In contrast to community-acquired sinusitis, the usual pathogens in nosocomial sinusitis are gram-negative enterics (such as *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, *Enterobacter* spp, *Proteus mirabilis*, and *Serratia marcescens*) and gram-positive cocci (occasionally streptococci and staphylococci).[16-20]

2. Fungal Fungal sinusitis is being increasingly recognized as a cause of chronic sinusitis in immunocompetent hosts.[21-24]

Aspergillus fumigatus is said to be the most common cause of fungal sinusitis in immunocompetent individuals.[23] The organism can colonize the sinuses, external auditory canal, or the tracheobronchial tree. It is not transmitted between patients; sources of infection are endogenous. The disease may take 1 of 3 forms: noninvasive, invasive, and disseminated. The noninvasive type is first seen as chronic rhinitis and nasal obstruction and may be allergic or nonallergic. If undiagnosed, it may go on to cause invasion. This presentation is similar to an intracranial mass. Fulminant disseminated disease occurs when the organism becomes locally aggressive in immunosuppressed hosts and invades the bloodstream, seeding lungs, liver, spleen, and bone and central nervous systems.

Other fungal species reported to cause disease in normal hosts include *Aspergillus flavus, Aspergillus niger*,[25] *Sporothrix schenkii*,[26] *Schizophyllum commune*,[27] *Emericella nidulans*,[28] *Pseudoallescheria boydii*,[29-33] *Paecilomyces* spp,[34] *Candida* spp,[35] *Mucor* spp,[36] *Basidobolus haptosporus*,[37] *Stemphyllium mucorsporidium*,[38] *Penicillium melinii*,[38] and *Bipolaris* spp.[39] Sinusitis has also been caused by dematiaceous fungi other than *Bipolaris*, including *Drechslera hawaiiensis*,[40] *Dreschslera spicifera*,[41] *Alternaria* spp,[42,43] *Exserohilum* spp, and *Curvularia lunata*.[44] These fungi are common saprophytes, and infection is acquired by inhalation of fungal spores. Dematiaceous fungi are similar to *Aspergillus* spp, which are characterized by histologically septate hyphal organisms. Some reports of *Aspergillus* sinusitis without culture confirmation may actually be cases caused by other dematiaceous fungi. Allergic fungal sinusitis may be caused by *Aspergillus* spp, *Myriodontium Keratinophilum*, or *Bipolaris*, *Dreschlera*, *Curvularia*, and *Alternaria* spp.[45-49]

Patients particularly prone to fungal infections of the paranasal sinuses include diabetics, patients with leukemia and solid malignancies who are febrile and neutropenic (most of whom will have received broad-spectrum antimicrobial therapy), patients receiving high-dose steroid therapy (eg, patients with connective tissue disease or transplant recipients), and patients with severe impairment of cell-mediated immunity (transplant recipients or persons with congenital T-cell immunodeficiencies).[50,51]

The most common cause of fungal sinusitis in immunosuppressed patients are *Aspergillus* spp.[16] Much less commonly, acute or chronic sinusitis may be caused by *Candida* spp or *Mucor* spp; the latter agent most frequently affects diabetic patients. In addition, *Pseudoallescheria boydii*, *Alternaria* spp, *Exserohilum* spp, and *Bipolaris* spp have been observed to cause sinusitis in immunosuppressed patients.[24,25,50]

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C. CLINICAL DIAGNOSIS

Ci. Clinical history

SUMMARY STATEMENTS

- Acute sinusitis is typically first seen as an upper respiratory infection that has persisted beyond 5 to 7 days.
- The diagnosis of sinusitis is based on a combination of clinical history with physical examination, nasal cytology, and/or imaging studies.
- Factors that may predispose to sinusitis include allergic or occupational rhinitis, vasomotor rhinitis, nasal polyps, rhinitis medicamentosa, and immunodeficiency. For many patients, the clinical history should address these factors.
- The differential diagnosis of sinusitis includes cystic fibrosis, Wegener's granulomatosis, HIV infection, Kartagener's syndrome, immotile cilia syndrome, and tumors.

The diagnosis of sinusitis is based on a combination of clinical history with physical examination; laboratory studies, including nasal cytology; and/or imaging studies (also see sections on Rhinolaryngoscopy and Biopsy). Acute sinusitis is typically first seen as an upper respiratory infection that has persisted beyond 5 to 7 days. Prominent symptoms in adults include nasal congestion, purulent rhinorrhea, postnasal drainage, facial or dental pain, headache, and cough (frequently with a more severe nocturnal component).[1,2] Children, in addition to the symptoms seen in adults, frequently exhibit increased irritability, vomiting that occurs in association with gagging on mucus, and/or prolonged cough.[3,4] Less predictable signs/symptoms include fever, nausea, malaise, fatigue, halitosis, or sore throat.

Symptoms of chronic sinusitis are similar to those observed with acute sinusitis but may be less obvious.[5] Patients with rhinitis may not realize that they have chronic sinusitis, but rather complain that their usual medications are no longer efficacious.

TABLE I. Differential diagnosis of rhinosinusitis

Infectious rhinitis
Viral upper respiratory tract infections
Sinusitis
Allergic rhinitis
Seasonal
Perennial
Nonallergic rhinitis
Vasomotor rhinitis
Aspirin intolerance
Eosinophilic nonallergic rhinitis (NARES)
Rhinitis medicamentosa
Decongestants
β-Blockers
Birth control pills
Antihypertensives
Rhinitis secondary to
Pregnancy
Hypothyroidism
Horner's syndrome
Wegener's granulomatosis
Anatomical abnormalities causing rhinitis
Foreign body
Nasal polyps
Nasal septal deviation
Enlarged tonsils and adenoids
Concha bullosa and other abnormalities of the middle turbinates
Tumors
Cerebral spinal fluid rhinorrhea

Adapted from Kaliner MA. Medical management of sinusitis. Am J Med Sci 1998;16:21-8.

TABLE II. Conditions that predispose to chronic sinusitis

Allergic and nonallergic rhinitis
Anatomic abnormality of the ostiomeatal complex
Nasal anatomical variations
Septal deviation
Concha bullosa
Paradoxical curvature of the middle turbinate
Haller cells
Cystic fibrosis
Common variable immunoglobulin deficiency
Specific antibody deficiency
IgG subclass deficiency
IgA deficiency
Ciliary dyskinesia, Kartagener's syndrome, Young's syndrome
Aspirin sensitivity
Acquired immunodeficiency syndrome
Bronchiectasis
Rhinitis medicamentosa
Cocaine abuse
Wegener's granulomatosis

Adapted from Kaliner MA. Medical management of sinusitis. Am J Med Sci 1998;316:21-8.

In taking the medical history, consideration should be given to the differential diagnosis of rhinosinusitis and other conditions that are associated with chronic sinusitis, such as cystic fibrosis, Wegener's granulomatosis, HIV infection, Kartagener's syndrome, immotile cilia syndrome, and tumors (Table I).

Factors that may predispose to sinusitis include allergic or occupational rhinitis, vasomotor rhinitis, nasal polyps, rhinitis medicamentosa, and immunodeficiency (Table II). For many patients, the clinical history should include this information. Sinusitis is often associated with and may worsen asthma.

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Cii. Clinical examination

SUMMARY STATEMENTS

1. The occurrence of a common cold or persistent allergic or nonallergic rhinitis are common precursors of acute and chronic sinusitis.
2. Prominent symptoms include nasal congestion, purulent rhinorrhea, postnasal drainage, facial or dental pain, headache, hyposmia, and cough.
3. Typical clinical signs include tenderness overlying the sinuses, mucosal erythema, nasal purulent secretions, increased posterior pharyngeal secretions, and periorbital edema.
4. If performed properly in adults, transillumination may be a useful diagnostic technique when combined with abnormal signs and symptoms.
5. More detailed examination for underlying risk factors may be required if sinusitis becomes chronic.
6. Endoscopy is a quick and safe way to visualize the posterior nasal structures and may aid in the diagnosis.

Acute sinusitis is a very common complication of the common cold.[1-3] The clinician whose medical history provides reason to suspect sinusitis must perform a clinical examination to differentiate sinusitis from a simple upper respiratory infection or allergic inflammation, which are both well-recognized risk factors for the development of acute sinusitis by means of an obstruction of the sinus ostia.[3,4] Most patients with sinusitis are not acutely ill. Fever is more common in children than adults. In the majority of cases, however, the body temperature is normal.

Inspection of the facial features may reveal signs of underlying allergy and or sinusitis. The allergic facies include the presence of dark circles under the eyes, a long thin face, a high arched palate, and/or periorbital edema (swelling of the upper and lower eyelids with discoloration of overlying skin), which is most noticeable in the morning on arising. Noteworthy signs in children include a transverse nasal crease and Morgan-Dennie's

lines (accentuated skin folds radiating from the inner canthus and traversing half to two-thirds the length of the lower lid margin).[5] Sinusitis may be heralded by swelling over the affected sinus, diplopia, proptosis, and rarely Pott's puffy tumor.[6] Palpation of the maxillary and frontal sinuses may reveal tenderness and soft tissue swelling. The sphenoid and ethmoid sinuses cannot be assessed adequately by physical examination.

The examiner should assess the condition of the nasal mucosa and secretions. A short wide nasal speculum permits viewing of the anterior part of the nasal passage. The nasal mucosa may be red and swollen in both infectious rhinitis and sinusitis, whereas the allergic mucosa is often pale and swollen with a watery secretion. Secretions are clear and watery at the onset of upper respiratory tract infections but become thicker, colored, and opaque after a few days. Usually, the discharge will remain purulent for several days and then clear again to a mucoid or watery consistency before resolution shortly thereafter. Colds generally last 5 to 7 days and rarely as long as 10 days. Persistent purulent nasal secretions usually seen coming from inside the middle meatus area are characteristic of sinusitis. The secretions can be green, yellow-green, or gray in sinusitis.

Purulent exudates in the middle meatus are thought to be highly predictive of sinusitis but may be difficult to visualize unless the nasal mucosa is decongested with a vasoconstrictor (eg, ½% phenylephrine).[7] The absence of visible purulent secretions does not rule out active sinus infections because drainage may be impeded by sinus ostia obstruction or may be directed posteriorly. Prolonged inflammatory changes of the nasal mucosa may lead to the development of nasal polyps or polypoid degeneration of the nasal mucosa, most frequently in the area of the middle meatus. As is the case for middle meatal secretions, polyps are best detected after the inferior and middle turbinates have been reduced by a topical decongestant.

The oropharynx should be examined for the presence of posterior pharyngeal mucopurulent secretions. Occasionally, tenderness of the maxillary teeth may be present because the roots of these teeth project into the floor of the maxillary sinus. Some cases of maxillary sinusitis are secondary to dental root infection or may occur after dental procedures, which introduce oral bacteria into the sinuses.[8] The presence of bad breath is often observed.[2] Examination of the ears in children may reveal otitis media, which is more commonly associated with sinusitis in children.[9] Auscultation of the chest for wheezing will help the clinician identify those patients who have the well-recognized syndrome of nasal polyposis, hyperplastic rhinosinusitis, asthma, and aspirin intolerance.[10]

Transillumination of the maxillary sinuses may be a useful adjunct if performed under proper conditions of a completely darkened room, allowance for adequate time for dark adaptation and an acquisition of skill in interpreting the findings.[11] The transillumination results are reported as opaque (no transmission), dull (reduced transmission) or normal (as expected in a normal person). Although the discriminate value of this examination technique alone is still controversial,[11-14] abnormal transillumination combined with purulent nasal secretions and 3 symptoms (maxillary toothache, poor response to nasal decongestants, and a history of colored nasal discharge) was the best predictor of sinusitis in one prospective study.[12] A change in transillumination from prior examinations, when combined with signs or symptoms suggestive of sinusitis, reinforces the diagnosis.

Despite the relative poor sensitivity and specificity of each of the examination methods described above, physicians appear to integrate physical signs and symptoms into an overall evaluation that accurately diagnoses sinusitis.[12] This applies equally well to primary care and specialty physicians.

Although the preceding examining methods are well suited to the patient with 1 or 2 episodes of sinusitis, a modified approach is warranted if the patient has experienced chronic or recurrent sinusitis episodes in 1 year. The clinical examination should evaluate the patient for septal deviation, nasal polyps, foreign bodies, and tumors. The physical examination should also include signs of immune deficiency (other infected sites, such as otitis media[15] or pneumonia), complications of primary infections (eg, mastoiditis), poor growth in children, unexplained dermatitis, absence of lymphoid tissue, cystic fibrosis (poor growth, nasal polyps, barrel chest, digital clubbing, and diffuse chest abnormalities on auscultation), immotile cilia or Kartagener's syndrome (nasal polyps, digital clubbing, and situs inversus), nasal septal deviation, nasal foreign bodies, and tumors (Table II).

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D. IMAGING STUDIES IN THE EVALUATION OF SINUSITIS

SUMMARY STATEMENTS

1. Imaging studies may be required when the symptoms are vague, physical findings are equivocal, or there is poor response to the initial management.
2. Standard radiographs may be used for detection of acute sinus disease, but they are insensitive, especially in ethmoid disease.
3. Computed tomography is the preferred imaging technique for preoperative evaluation of the nose and paranasal sinuses secondary to obstruction of the ostiomeatal complex.
4. Although magnetic resonance imaging has limitations in the definition of the bony anatomy, it is particularly sensitive for evaluation of the frontal, maxillary, and sphenoid sinuses for fungal sinusitis and tumors and the differential diagnosis between inflammatory diseases and malignant tumors.
5. Ultrasonography has limited utility but may be applicable in pregnant women and for determining the amount of retained secretions.

Although sinusitis is a common medical problem, definitive diagnosis by history and physical examination alone is often difficult. The greatest clinical challenge is to document the presence and extent of disease within the paranasal sinuses and to distinguish between sinusitis and rhinitis. Percussion, transillumination, and anterior rhinoscopy often are of limited diagnostic value.[1] Although sinus ultrasonography has occasionally been used, roentgenographic examination has been the primary diagnostic procedure used to confirm a clinical impression of sinusitis or document disease in the major sinus cavities.[2,3] However, computed tomography (CT) and magnetic resonance imaging (MRI) are becoming accepted as the standard imaging techniques for definitive diagnostic evaluation of patients with both acute and chronic sinusitis. The clinical utility, relative safety, ease of performance, availability, and cost of these imaging techniques vary considerably, and all have inherent limitations.

The indications for obtaining imaging confirmation are in most cases self-evident. Most often, these techniques are required when the symptoms are vague, physical findings are equivocal, or there is poor response to the initial courses of management. In a situation in which multiple antibiotic hypersensitivity exists and the risk of reaction to a new antibiotic is greater than normal, it is very important to document that sinusitis exists before treatment. Persistent refractory conjunctivitis, especially in children, may suggest the possibility of underlying chronic sinus disease, which needs to be confirmed by imaging. Acute, severe eye pain or occipital pain may occur as presenting symptoms, and imaging under these conditions is essential to rule out frontal and/or sphenoid sinusitis. A patient with severe, chronic underlying asthma may be suspected of having chronic sinus disease, which further aggravates the lower inflammatory disease. Finally, in cases of chronic and unremitting sinus disease, unusual diseases, such as allergic sinus mycosis and Wegener's granulomatosis, may be suggested by the newer, more sophisticated imaging techniques.

Imaging techniques

Ultrasonography

A-mode ultrasound is a safe, quick, noninvasive technique that has been proposed as a diagnostic screening tool for sinus pathology.[4,5]

The rationale for use of A-mode ultrasound in the evaluation of sinus disease is based on the principle that ultrasound waves are reflected at the boundary of 2 media with differing acoustic characteristics. A spike or echo is observed when the sound is reflected. Sound waves are readily transmitted in fluid but are reflected by air. If the sinus contains fluid, an echo is expected from the bony back wall of the sinus, but if the sinus is normally aerated, the sound will be reflected rather than transmitted, and only the bony echo from the anterior wall will be observed.[4,5]

In general, the usefulness of this method compared with routine radiography is very limited because of insensitivity.[6-8]

It was hoped that ultrasound would provide reliable diagnostic results that could facilitate early diagnosis in the clinic or office setting at reduced cost and radiation exposure to the patient.[8-12] It may have some utility as a diagnostic screen in pregnant women to avoid ionizing radiation.[8] Additionally, because of the benign nature and ease of performance of the examination, it was hoped that ultrasound would have potential as a screening modality for easier detection of sinus disease. However, ultrasonography can be used to evaluate only the maxillary and frontal sinuses. In 1 comparative study between radiography and ultrasonography, it was concluded that ultrasonography better measured retained secretions (excluding very small amounts), whereas radiography more accurately detected mucosal swelling and thickening.[13,14]

In another study comparing ultrasound with radiography in 30 children and adults, the false-positive rate was, respectively, 39% to 45% and the false-negative rate was 42% to 56%. The authors concluded that ultrasound is not sufficiently accurate to be used as a substitute for radiography in diagnosing sinusitis.[6]

Radiologic evaluation

The primary goals of radiologic imaging of the paranasal sinuses are to provide an accurate image of the regional anatomy and to establish the presence and extent of sinus disease. This information may assist in planning and monitoring medical therapy and provide an anatomic guide to facilitate surgical treatment.[15]

Radiography (X-ray)

Standard radiographs are still the most frequently used radiologic imaging modality for evaluating sinus disease. The Caldwell (anterior-posterior) and Waters views best demonstrate the frontal and maxillary sinuses. The lateral view is the best choice for visualization of the sphenoid sinus and adenoidal tissue in children. Substituting a single Waters view for a 4-view sinus series is an acceptable screening strategy for diagnosing maxillary sinusitis.[16] The fine bony anatomy of the ethmoid sinuses is not adequately displayed on any of the views because of the problem of structural superimposition.

Interpretation of standard radiographs may be controversial. Opacification, moderate-to-severe mucosal thickening, polyps, or air fluid levels in patients with persistent symptoms are generally considered indicative of sinusitis. Some authors claim that any opacification is abnormal and that the only normal sinus is a clear sinus, but others stress that sinus opacification is commonly observed in patients with acute upper respiratory infections. Additionally, overlay of anatomic structures may mimic mucosal thickening or air-fluid levels, and a hypoplastic sinus may be misinterpreted as pathologic opacification. Other abnormalities may be related to residual postoperative changes.

Although many consider standard sinus radiographs useful in the diagnosis and monitoring of acute sinusitis, they are of limited value in the evaluation of chronic unremittant sinusitis. Standard radiographs are inadequate for determination of the need for, or guidance of, endoscopic sinus surgery in both children and adults.[17] Lateral views are of limited value in children less than 3 years of age and tend to overestimate sphenoidal clouding in children 3 to 5 years of age.[17]

Computed tomography

CT is currently the modality of choice in the preoperative evaluation of the nose and paranasal sinuses and is the gold standard for precise delineation of inflammatory sinus disease secondary to obstruction of the ostiomeatal complex.[18] High resolution CT can demonstrate disease that is not shown on routine x-ray films.[17,19,20] It can also delineate pathologic variations and demonstrate anatomic structures inaccessible by physical examination or endoscopy. Because of its axial plane of view and superb contrast resolution, CT is the method of choice for examining the complex anatomy of the sinuses. If intravenous contrast enhancement is used, CT can reliably distinguish mucosal thickening from fluid in the maxillary antrum. The coronal plane most closely correlates with the surgical approach and best shows both the ostiomeatal channels and the relationship between brain, fovea ethmoidalis, and ethmoid sinuses. Claustrophobia and limitation of neck movements, particularly in patients over the age of 60, are the most common factors leading to suboptimal scans.[21]

In routine CT studies, the radiation dose to the patient is not negligible.[8,18] Although low-dose CT increases image graininess, it does not induce many errors of interpretation, except that the thickness and integrity of the ethmoid septa are sometimes more difficult to evaluate in cases of extensive sinus disease.[18] Thus low-dose CT may be useful in the evaluation of inflammatory disease of the sinuses, complemented by 1 or 2 sections obtained with standard dose settings, focused on questionably abnormal bone septa.[18]

Standard radiographs are frequently ordered in preference to CT because of reduced cost.[22] However, an average 4-slice, limited-cut, coronal-plane CT of the sinuses takes 20 minutes to perform at a cost only slightly greater than that of a standard set of radiographs.[22] It provides high resolution bone and soft tissue imaging of the key surgical areas.

Many otolaryngologists consider high-resolution CT to be a mandatory part of the chronic rhinosinusitis preoperative assessment. The planning and safety of functional endoscopic sinus surgery is greatly improved by CT imaging.[22,23] CT can provide excellent definition of the paranasal sinuses, particularly the ethmoids, which is a prerequisite for endoscopic surgery.[23]

CT may be especially helpful in the diagnosis of fungal sinusitis because it delineates unilateral lesions of 1 or more sinuses, nodular mucoperiosteal thickening, focal areas of bone destruction, and very dense intrasinus concretions.[15,24] In most cases a combination of these findings suggests fungal disease.[24]

MRI

MRI provides better imaging of soft tissue than CT, but it is less suited to imaging the bony anatomy of this region. Because bone and air yield similar signal intensities on MRI, precise definition of the ostiomeatal air passages and their bony perimeters is more difficult by this technique. Furthermore, in the patient with extensive inflammatory disease, the signal intensity of the pathologic process is indistinguishable from the appearance of the normal mucosa in the edematous phase of the nasal cycle. These factors limit the MRI evaluation of underlying anatomy in a patient with chronic inflammatory disease, especially because the mucosa in the ethmoid sinus "cycles" with the mucosa in the nasal cavity. Although MRI has significant limitations in the definition of anatomy, it is extremely sensitive in evaluation of paranasal sinus mucosal disease in the frontal, maxillary, and sphenoid sinuses because the mucosa in these sinuses does not undergo the cyclic edema.[25]

The etiology of certain disease processes may be better differentiated by MRI. MRI often aids in the differential diagnosis between inflammatory diseases and malignant tumors.[26,27] MRI is useful for cases complicated by

orbital or intracranial extension. Bacterial and viral inflammation have a high signal intensity on T2-weighted images, whereas neoplastic processes, primarily squamous cell carcinoma, demonstrate an intermediate bright signal on T2-weighted images. Fungal concretions have a very low signal intensity on T2-weighted images similar to that of air.[15] MRI or CT is significantly more sensitive than plain radiography ($P < .001$) in defining the extent of the disease, particularly with posterior sinus involvement.[28] Hypointense signals may be present on MRI sequences despite CT evidence of the presence of high-attenuation material filling the sinus.

CT compared with MRI

Both CT and MRI are generally considered to be superior to x-ray tests in the diagnosis of inflammatory conditions of the nose and paranasal sinuses. When an inflammatory condition is known to be present, MRI is more accurate than CT, but the latter (thanks to its optimal delineation of both bony walls and soft tissues) is superior to MRI in the differential diagnosis of a generic disease of the nose and paranasal sinuses (ie, when the inflammatory nature is questionable). CT is especially useful in emphasizing the ostiomeatal channels and is preferred for studying the ethmoid and sphenoid sinuses.[21,26,29,30] On balance, CT emerges as the diagnostic gold standard of sinus disease because of its diagnostic accuracy, easy access, and lower cost.

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E. LABORATORY TESTS

SUMMARY STATEMENTS

1. Laboratory evaluation of chronic or recurrent sinusitis may include any of the following: nasal cytology, sweat chloride test, ciliary function studies, and tests for immunodeficiency.
2. Nasal cytology is useful in the clinical evaluation of underlying allergic rhinitis, the nonallergic rhinitis with eosinophilia syndrome, nasal polyposis, and aspirin sensitivity.
3. Quantitative sweat chloride tests for diagnosis of cystic fibrosis should be considered in children with nasal polyps and/or colonization of the nose and sinuses with *Pseudomonas* spp.
4. Tests for immunodeficiency (eg, quantitative immunoglobulins, antibody tests, serum IgE, and complement components) may be useful if either congenital or acquired immunodeficiency is suspected in cases of recurrent sinusitis.

Other diagnostic methods may be indicated for investigating the patients with chronic or recurrent sinusitis,

especially if bronchitis or bronchiectasis is present. This section will review the laboratory evaluation of patients with chronic sinusitis for which no obvious causal link has been found (eg, allergic or infectious rhinitis). Laboratory studies that are of major importance include the following: nasal cytology, the sweat chloride test, ciliary dysfunction studies, and other miscellaneous tests that aid in the confirmation of several diseases either causing or associated with chronic sinusitis.

Nasal cytology

Nasal cytology is a tool used in the evaluation of rhinitis. It is a useful adjunct in determining whether chronic rhinitis conditions are associated with sinusitis. However, many centers do not include cytology as a diagnostic step in evaluating possible sinusitis, and the clinician must decide if this procedure is significantly helpful to warrant its performance. Several methods of collecting nasal secretions are described. The patient can blow secretions into wax paper or Saran wrap, which is the most simple and effective way of collecting a sample. The sample can also be collected by nasal lavage with a cytology brush, cotton swab, or a Rhinoprobe (Synbiotics Corporation, San Diego, Calif). By collecting secretions from the blowing technique, less cellular material may be collected.[1] This technique is useful for detecting nasal eosinophils in allergic rhinitis, especially during seasonal exacerbations when the patient is symptomatic. Because nasal lavage is a dilution process, the sample needs to be concentrated; it is minimally invasive but reflects an anatomically wider sample of nasal material.[2]

Once the sample is collected, it must be stained. After cytofixation or heat fixation, Hansel stain, a commonly used stain to identify eosinophils, is often used. Other stains, such as the Papanicolaou, hematoxylin-eosin, acidified toluidine blue, or May-Grunwald-Giemsa stains, can be used to identify other elements, including mast cells, basophils, neutrophils, nonciliated epithelial cells, squamous epithelial cells, pollen grains, and debris. Nasal cytology may be affected by topical steroids.

Several distinct syndromes that may be associated with sinusitis have been defined by nasal cytology. These include allergic rhinitis, vasomotor rhinitis, nonallergic rhinitis with eosinophils, bacterial sinusitis, and metaplasia secondary to environmental exposures.[3] Both subjective and semiquantitative methods exist for assessing nasal cytology, leading to limitations in the interpretation and quantitation of results.[4-6] Studies defining the sensitivity and specificity of nasal cytology in these diseases are limited. Nasal smear eosinophils may be present in allergic rhinitis, the nonallergic rhinitis with eosinophils syndrome, nasal polyposis, and aspirin sensitivity.[7,8]

Comparative studies of nasal cytology with global and radiographic assessments of sinusitis are limited and conflicting. In these studies the specificity of nasal neutrophils in sinusitis ranged from 40% to 90% and sensitivity varied between 67% and 80%.[9,10] These disparate results are further complicated by the fact that acute viral infections can lead to neutrophilic nasal secretions and transient abnormal imaging studies.[10] Furthermore, it is possible for patients to have concurrent allergy and bacterial sinusitis (ie, a mixture of eosinophils and neutrophils). Therefore the clinical usefulness of nasal cytology in evaluating sinusitis is limited.

Sweat test

The Gibson-Cooke sweat test (quantitative pilocarpine iontophoresis) is the gold standard for identifying patients with cystic fibrosis.[11] Chloride values greater than 60 mEq/L in children are considered diagnostic, provided at least 75 mg of sweat is collected. Adults can have values 10 to 15 mEq/L higher than those of children.[12] Although sodium is measured in some laboratories, it is more variable than chloride, which should always be measured for a definitive diagnosis.[13] Indications for a sweat test in patients with sinusitis include the observation of nasal polyps and/or chronic colonization of the nose and sinuses with *Pseudomonas* spp.[14-19]

Ciliary tests

Congenital abnormalities of ciliary structures are occasionally considered as possible explanations for recurrent sinusitis. These rare disorders are autosomal recessive and occur in 1 in 20,000 live births but may be found in as many as 5% of patients with chronic respiratory infections arising in the first weeks of life.[20,21] Most patients with these disorders have a triad of recurrent otitis, recurrent sinusitis, and frequent pneumonia associated with bronchiectasis.[22-25] In half the cases situs inversus is present (Kartagener's syndrome) and provides the best clue to the presence of a ciliary disorder. It is believed that the mechanism of increased susceptibility to bacterial infection is loss of normal mucociliary transport, although some defects are also associated with defective neutrophil chemotaxis.[23]

Functional tests of cilia include visual and videoscopic measurements of tissue and mucociliary transport, such as the rate of saccharin or disc movement tests.[26,27] Visual assessments of ciliary function have been done by collecting ciliated cells on a cytology brush and suspending the material on a glass slide.[28,29] The ciliary motion is then observed by simple light or phase microscopy, with a ciliary beat frequency less than 10 beats/second considered below the normal range.[21]

The saccharin test is used as a screening test.[30] It is performed by placing a small volume of saccharin sodium at the bottom of the inferior nasal meatus. After 3 minutes, the subjects swallow every 30 seconds until a sweet taste is detected.[26] Values greater than 15 to 30 minutes are considered abnormal.[31] Definitive confirmation of ciliary abnormalities requires electron microscopy.[32-34]

Among the criteria for ultrastructural analysis are the type of defect (dynein defects being the most specific), the percent of cilia having the defect, the distribution of the defect (bronchial versus nasal), and the number of cilia present.[32-35]

Miscellaneous tests in the differential diagnosis of chronic sinusitis

Patients with immunodeficient states, both congenital and acquired, are often first seen with severe, recurrent sinusitis. In such situations it is appropriate to evaluate quantitative immunoglobulin isotypes, including IgE (see section on congenital or acquired immunodeficiency). In rare instances total complement and complement components may be obtained. The diagnosis of acquired immunodeficiency is established by HIV antibody and/or antigen tests.

In suspected cases of Wegener's granulomatosis, an antineutrophil cytoplasmic antibody test may be a diagnostic adjunct to tissue biopsy.[36] Similarly, elevated angiotensin-converting enzyme and soluble IL-2 receptor levels may aid in the diagnosis of active sarcoid lesions in nasal or sinus locations.[37]

The suspicion of sinus allergic mycoses may be enhanced by high serum IgE levels and the presence of precipitating IgG and/or specific IgE antibodies to a variety of saprophytic fungi, which have been cultured in this disease.

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F. RHINOLARYNGOSCOPY

SUMMARY STATEMENTS

1. Fiberoptic rhinoscopy permits more detailed examination of the anterior and posterior nasal and pharyngeal structures.
2. Fiberoptic rhinoscopy may be considered for some patients with chronic or recurrent sinusitis to assess structural abnormalities, fungal disease, or granulomatous lesions.

In some situations in which sinus disease is suspected, and the routine anterior nasal speculum examination has had limited diagnostic value, endoscopy of the upper airway should be considered to permit more detailed exam-

ination of the anterior and posterior nasopharyngeal recessed structures, such as sinus ostia, sphenoethmoidal recess, and eustachian tube ostia. Direct vision of the epiglottis, glottis, and vocal cords is an additional advantage of this technique.[1,2]

Instruments

Two types of endoscopes are available. The optically excellent Hopkins rigid nasal endoscope permits extensive examination of the nasal cavities and portions of the nasopharynx, as well as high-quality photography of the structures examined.[3] Fiberoptic rhinoscopy is a convenient, cost-effective, and safe alternative. The fiberoptic rhinoscope permits quick and thorough examination of most areas of the upper airway without discomfort to the patient. Abnormalities of the septum, turbinates, mucosa, nasopharynx, adenoids, eustachian tube orifice, tonsils, posterior tongue, epiglottis, glottis, and vocal cords can easily be seen.[4] The origin and course of nasal polyps can be identified, and the effect of treatment can be followed.

Indications for endoscopic evaluation for sinus disease

Fiberoptic rhinoscopy may be considered for some patients with acute, chronic, or recurrent sinusitis. This technique may reveal the presence or extent of nasal polyps, septal deviation, and/or mucopurulent secretions.

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G. BIOPSY OF THE NOSE AND PARANASAL SINUSES

SUMMARY STATEMENTS

1. Paranasal sinus biopsies may be required to determine whether a lesion is neoplastic or to confirm the presence of suspected fungal disease and the possibility of granulomatous disease.
2. Nasal mucosa harvested from the posterior portion of the inferior turbinate is the preferred biopsy site for primary cilia dysfunction.

Tissue biopsy may be considered for the differential diagnosis of nasal or paranasal lesions that obstruct sinus cavities and give rise to chronic, recurrent sinus disease. Thus cardinal reasons for obtaining paranasal sinus biopsies are (1) to determine whether the lesion is neoplastic and if so, what kind of neoplasm it is; (2) to confirm the presence of suspected fungal disease; and (3) to establish a diagnosis when granulomatous disease cannot be differentiated clinically or radiographically from other forms of chronic inflammatory rhinosinusitis. Tissue biopsy specimens are also required for evaluating ciliary function and structure.

Nasal and paranasal sinus neoplasms

Because the sinuses are not accessible for direct physical examination, and because early symptoms of sinus tumors, such as nasal obstruction, anosmia, rhinorrhea, and pain are also common symptoms of inflammatory sinusitis, most sinus neoplasms are not diagnosed at an early stage. This is probably a major factor in the relatively poor prognosis of malignant sinus neoplasms. Often, clinical suspicion of tumor is not raised until epistaxis, proptosis, trismus, facial swelling or cranial nerve (I/VI) dysfunction develop in a patient, most of which are indications of extensive tumor involvement. Early diagnosis of nasal and sinus neoplasm is enhanced by use of computed tomography and/or magnetic resonance imaging.

Tumors originating in the nose or sinuses cover a wide range of histologic types, both malignant and benign. Usually it is not possible to reliably predict tumor histologic type on the basis of clinical or radiographic findings, and thus biopsy is performed before definitive therapy, which will then be based on tumor histology.[1] An exception to this is juvenile angiofibroma. Diagnosis of this lesion is made by clinical and radiologic findings of a vascular posterior nasal or nasopharyngeal mass in an adolescent or preadolescent male, and biopsy should not be performed because of the risk of significant hemorrhage.[2]

Invasive fungal infections

Rhinocerebral mucormycosis is a potentially fatal invasive fungal infection produced by Phycomycetes, which includes the genera *Absidia*, *Mucor*, and *Rhizopus*. A similar but somewhat less fulminating infection can be caused by *Aspergillus* spp**.** Although invasive fungal disease has been reported in otherwise normal patients, it remains predominantly a disease of immunocompromised patients. Mucormycosis occurs most commonly in patients with diabetes mellitus, hematologic malignancies, adrenal suppression, chronic renal failure, hematologic dyscrasia, and other immunologically compromised conditions. Patients undergoing chemotherapy are also at increased risk as are intravenous drug users and HIV-positive patients.

Because invasive fungal infections may progress very rapidly, it is necessary to maintain a high degree of suspicion when dealing with this group of patients. The infection spreads rapidly along blood vessels, producing necrosis. Patients are often first seen with facial pain, proptosis, ophthalmoplegia, or facial necrosis**.** Late radiographic findings may include sinus opacification, mucoperiosteal thickening, bone erosion, and cavernous sinus thrombosis, but these findings may be absent or minimal early in the course of the disease. On endoscopic visualization, early lesions may appear as brick red areas on the nasal mucosa, but more typically they appear as black, necrotic areas. Tissue necrosis often renders the involved area anesthetic. Abnormal-appearing lesions should undergo biopsy promptly, and the specimen should be sent for fungal

stains and fungal culture. Early diagnosis is mandatory if these patients are to be salvaged.[3-5]

Granulaomatous inflammatory disease

A number of chronic granulomatous inflammatory diseases, both idiopathic and infectious, involve the nose and paranasal sinuses. These conditions may cause ulceration, necrosis, or hyperplastic mucosa within the nasal cavity or paranasal sinuses. Involvement may be patchy or diffuse and is often difficult to differentiate from chronic nongranulomatous inflammatory conditions. The granulomatosis diseases include idiopathic processes, such as Wegener's granulomatosis and sarcoidosis, as well as infectious processes, such as rhinoscleroma, tuberculosis, leprosy, syphilis, and fungal infections. Biopsy in combination with clinical, serologic, and radiographic findings is usually necessary for diagnosis. Many of the organisms that cause chronic granulomatous inflammation are difficult to detect without special staining. Tissue should be submitted for histologic examination to rule out neoplasia and for special staining and culture of mycobacteria and fungi.[5,6]

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H. CONGENITAL OR ACQUIRED IMMUNODEFICIENCY

SUMMARY STATEMENTS

1. The majority of patients with congenital immune deficiency and associated sinusitis will have defects in humoral immunity. However, sinusitis is common in acquired immune deficiency (AIDS), in which both humoral and cellular impairments are present. Appropriate laboratory studies of both humoral and cellular immunity include quantitative immunoglobulin assays; specific antibody responses to tetanus toxoid, pneumococcal, or *Hemophilus influenzae* vaccines; and measurement of complement components and key cellular responses.
2. The most common congenital immunodeficiency syndromes in this group are common-variable immunodeficiency, IgA deficiency Wiskott-Aldrich, ataxia telangiectasia, X-linked immunodeficiency with normal or elevated IgM, and X-linked agammaglobulinemia.

The possibility of immunodeficiency should be considered in patients with chronic or recurrent sinusitis, especially when associated with bronchitis or bronchiectasis. Evaluation of immunodeficiency begins with a careful history and physical examination. The physical examination of patients with suspected congenital immunodeficiency should focus on findings associated with specific diseases, such as absence of tonsillar tissue, ocular telangiectasia, skin and mucous membrane infections, eczema, clubbing, rales rhondii, and purpura.[1-3] The majority of patients with congenital immunodeficiency with sinusitis will have defects in humoral immunity[4-6]; however, sinusitis is common in acquired immune deficiency syndrome (AIDS), in which both humoral and cellular impairments are present.

The indications for pursuing an immunodeficiency evaluation depend on the age, medical history, physical exam, and lifestyle of the patient. For example, in an infant of less than 2 years of age with recurrent and life-threatening infections of the sinuses and other organs, one should pursue this evaluation in a expeditious manner. In addition, infections with organisms of low pathogenicity should alert the physician to the probability of a congenital immune deficiency. Similar indications for evaluation of immune deficiency exist for AIDS in both children and adults.

Appropriate laboratory studies of both humoral and cellular immunity include quantitative immunoglobulin assays, specific antibody responses, complement components, and measurements of T-cell responses. In principle the response to any protein antigen can be measured, but in practice measurement of the response to tetanus immunization offers several advantages. Most patients have been immunized with tetanus antigen, and 90% to 100% of children will have protective antibody titers on completion of primary immunization.[7] Tetanus toxoid is a potent antigen, and a poor response to immunization usually indicates a significant defect in humoral immunity.[8] Pneumovax or *Haemophilus influenzae* B may also be used to measure antibody production.

Patients have also been described with recurrent sinopulmonary infections and normal total IgE levels but nondetectable IgG_2 and IgG_4 in their serum.[9,10-12] In general, the bulk of antibody response to carbohydrate antigens is of the IgG_2 subclass.[13,14] This is especially true of the antibody directed against *Haemophilus influenzae*, a common respiratory tract pathogen.[15] In general, the antibody response to carbohydrate antigens correlates with IgG_2 levels.[16] In older patients symptomatic IgG subclass deficiency may correlate with defects in specific antibody response, especially to polysaccharide antigens.[17] Antibody responses to pneumococcal antigens may be useful, but the response of children less than 8 years old may not be complete.[10] Poor antibody responses to pneumococcal serotypes 3 and 7 appear to correlate most closely with defects in humoral immunity.[8,18-21]

In addition to the immune defects discussed above, chronic or recurrent sinus disease is a common feature of several other immunodeficiency diseases, which may require additional diagnostic studies. The most common congenital immunodeficiency

Syndromes in this group are common-variable immunodeficiency, Wiskott-Aldrich,[22,23] ataxia telangiectasia,[2,3,24-26] x-linked immunodeficiency with normal or elevated IgM,[1,27-31] and X-linked agammaglobulinemia.[1,32-35]

The incidence of sinusitis in HIV infection varies from 30% to 68%.[36-39] There may be a direct relationship between CD_4 deficiency and sinusitis.[36,40] Although hypergammaglobulinemia is a frequent occurrence in HIV infection, specific antibody responses are abnormal.[41-43] The microbiology of sinusitis in patients with HIV is similar to that seen in other patients with recurrent and chronic sinusitis. However, pathogens, such as *Pseudomonas* spp, *Staphylococcus aureus*, *Klebsiella* spp, *Escherichia coli*, *Candida* spp, *Cryptococcus* spp, and *Cytomegalovirus* spp have been isolated.[40-43] The treatment of sinusitis in these patients is similar to that seen in nonimmunized patients but with a longer duration of therapy.

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I. ASTHMA

SUMMARY STATEMENTS

1. The association between sinusitis and asthma has long been appreciated and is generally stated to range from 40% to 75%.
2. Although a number of theories have been proposed to explain this relationship, no direct causal factor has yet been found.
3. Studies in both adults and children have clearly shown that medical and surgical management of sinusitis results in objective and subjective improvement of asthma.

The association between sinusitis and asthma has long been appreciated. The incidence of sinusitis in asthmatic subjects is generally stated to range from 40% to 75%.[1-7] Although these studies strongly suggest that sinusitis triggers or worsens asthma, it could be argued that they merely coexist and represent different end products of the same process (inflammation) occurring in different organ systems.

Mechanism relating sinusitis and asthma

Various mechanisms have been proposed to explain the relationship between sinusitis and asthma. The 5 most common are sinonasobronchial reflex[8-19]; inhalation of cold, dry air[20-25]; aspiration of nasal secretions[26-32]; cellular and soluble mediators[33-36]; and diminished β-agonist responsiveness.[37-39]

Implementations of treatment

Perhaps the most direct evidence of a cause-and-effect relationship has been provided by studies that show that appropriate treatment of sinusitis by medical intervention can result in significant improvement of asthma symptoms.[40-42] Additionally, sinus surgery in patients with asthma has been shown to bring about improvement in lower airway disease, although adequate controls have not been incorporated in most studies. In one study Weille[43] examined 500 patients with asthma, 72% of whom had concomitant chronic sinus disease. Of 100 patients who underwent sinus surgery, 56 subsequently experienced improvements in chest symptoms; complete resolution of asthma occurred in 10. Twenty-three of 24 patients with simultaneous chronic sinusitis and asthma experienced a 75% or greater improvement in asthma symptoms after surgical drainage in another study.[44] This association is supported by other researchers, including Slavin,[45] who reported that lower airway symptoms were significantly reduced after nasal surgery in patients with severe asthma that often required daily oral corticosteroid therapy. In a follow-up of similar patients, 60% were found to have experienced improved asthma symptoms that persisted for 5 years.

In another study of sinus surgery in patients with asthma, 17 patients were treated with nasal surgery because of severe sinus disease. Fifteen of these patients experienced improved sinus symptoms, and 13 experienced significantly improved asthma symptoms, postoperatively.[46] Most of these patients underwent the Montgomery procedure, in which the mucosal lining of the sinuses is obliterated, and adipose tissue from the abdomen is implanted. This procedure promotes the formation of fibrous tissue, which helps to reduce the recurrence of infection. In one patient severe asthma symptoms that could not be controlled with high-dose corticosteroids developed after the procedure. When the implant was removed, however, control of the asthma was regained. This phenomenon supports the idea that sinus disease and asthma exacerbations are related.

Conclusion

The association of sinusitis and asthma seems to be more than an epiphenomenon. Studies in both adults and children have clearly shown that medical and surgical management of sinusitis results in both objective and subjective improvement of the asthmatic state. This is suggestive of a cause and effect relationship (ie, sinusitis in some way directly worsens asthma). Though several theoretical mechanisms exist, none have been conclusively proven.

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J. ALLERGIC RHINITIS

SUMMARY STATEMENTS

1. Allergic rhinitis commonly precedes the development of recurrent or chronic sinusitis because the associated nasal obstruction and inflammation interrupts normal mucociliary clearance and leads to retention of mucopurulent secretions within the sinus cavities.
2. Patients with suspected allergic rhinitis in conjunction with sinusitis may benefit from evaluation by an allergist/immunologist who, in most instances, will perform prick/puncture tests to clarify the role of allergies.
3. Treatment of allergic rhinitis should include environmental control; medications, such as systemic and topical antihistamines, decongestants, nasal cromolyn, anticholinergics, and glucocorticosteroids; and, in appropriate patients, allergen immunotherapy.
4. Other forms of rhinitis (eg, vasomotor rhinitis or NARES) also commonly precede the development of recurrent or chronic sinusitis.

Allergic rhinitis is one of the most common chronic diseases, accounting for approximately 2.5% of all physician visits. The incidence is estimated at 10% of the population at any time, with a cumulative prevalence varying up to 20%. Allergic rhinitis usually develops during childhood at the average age of 10 years, although 30% of patients have their onset of symptoms after the age of 30 years.[1-3] Allergic rhinitis is a major predisposing factor for the development of sinusitis in both the adult and pediatric population. In children, evidence of allergic rhinitis has been found in 36% to 60% of patients with chronic sinusitis.[4-6] Young adults with acute maxillary sinusitis had a 25% to 31% incidence of allergic rhinitis[7]; whereas chronic sinusitis was associated with allergic rhinitis in 40% to 80% of adult patients.[8-10]

Newman et al[11] found an association between extensive sinus disease, quantitated by computerized tomography, and allergy in 78% of patients and an association with asthma in 71% of patients.

One of the most suggestive experimental links is derived from a study reported by Pelikan et al[12] in which rhinomanometry was performed and maxillary sinus x-ray films were taken before and after nasal provocation tests. Of 73 separate provocation tests, 41 led to early-phase responses only, 18 led to late-phase responses only, and 10 reactions yielded both early and late responses. Thirty-two of these patients experienced increased sinus mucosal edema and opacification of the paranasal sinuses as revealed by sinus x-ray films. Concomitantly, increased pressure in the maxillary sinus, acute headaches, and associated otalgia were reported by patients. In addition, 3 of the patients without an obvious clinical response had thickened mucosa and subjective symptoms. In view of the difficulty in performing direct paranasal sinus challenges, these nasal challenges provide a link between rhinitis and sinusitis.

Because of the close relationship between allergic rhinitis and sinusitis, patients with sinusitis, especially of a chronic or recurrent nature, should have an allergy evaluation.

Allergy evaluation usually includes a careful history and skin tests. Skin testing measures the presence of specific IgE reacting to the antigen, with release of mediators causing a local reaction. The measurement of the

resultant wheal and flare is a marker of sensitivity to that antigen. Good testing requires skill at performing the test, knowledge of local allergens, the patients' probability of exposure, and use of positive (histamine) and negative (saline) controls. The patient should not use antihistamines for 2 to 7 days (several weeks may be needed for astemizole) or other drugs that may blunt or interfere with testing. A positive test response may signify sensitivity to the antigen if the symptoms correlate to the positive response, or it may be a sign of sensitization without symptoms or a marker of latent allergy. Skin tests may be positive in patients for up to 3 years before clinical symptoms develop. The most rapid and safe method is prick-puncture testing. If the prick-puncture test responses are negative or equivocal, intracutaneous testing may be done. This procedure involves injecting small amounts of antigen under the skin and measuring the resultant wheal and flare. Intracutaneous tests should not be done unless prick testing is done first because of a higher risk of anaphylaxis. Intracutaneous testing with food antigens is not recommended because of poor specificity.

In vitro measurements include measurement of total IgE and antigen-specific IgE by RAST or ELISA testing. Indications for in vitro testing include (1) extensive dermatitis or urticaria; 2) patients who are unable to stop their antihistamines for skin testing; 3) patients with poor skin reactivity; and 4) patients with a history of severe anaphylaxis to a particular antigen in whom skin testing would be dangerous.[13-17]

Management of allergic rhinitis is focused on environmental controls, pharmacologic management of symptoms, and immunotherapy with an immunomodulating agent. The effective treatment of allergic rhinitis may lead to a decreased frequency of sinusitis by reducing the inflammation and swelling that compromises the sinus ostia.

Although this section did not specifically cover other types of rhinitis (eg, vasomotor rhinitis and NARES), their overall effects in leading to sinusitis are similar.

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K. CYSTIC FIBROSIS

SUMMARY STATEMENTS

1. Chronic sinusitis is an important source of morbidity in nearly all patients with cystic fibrosis, creating nasal obstruction, postnasal drainage, headache, and potential exacerbation of pulmonary obstruction.
2. Pathogens in patients with cystic fibrosis with sinusitis include *Pseudomonas aeruginosa*, *Escherichia coli*, *Staphylococcus aureus*, and *Aspergillus fumigatus*, in addition to the more common polysaccharide-encapsulated organisms.
3. Although children with cystic fibrosis and sinusitis generally respond to prolonged treatment with conventional oral antibiotics, older children and adults who are colonized with *Pseudomonas aeruginosa* frequently require oral quinolones, intravenous tobramycin, or ceftazadime to control acute exacerbations.

Cystic fibrosis (CF), the most common lethal autosomal-recessive disease in whites, is caused by mutations in the CF transmembrane conductance regulator (CFTR) gene on the long arm of chromosome 7.[1] Clinical disease results from those mutations in CFTR that lead to defective transmembrane conductances of chloride ions and thereby to impaired transport of water. As a consequence, the viscosity of all exocrine fluids increases substantially, principally in the lung and gastrointestinal tract, but also in the upper respiratory tract, including the paranasal sinuses.

Radiographic evidence of sinusitis has been known for decades to be present in patients with CF. An increasing number of individuals who have minimal respiratory symptoms and no gastrointestinal symptoms, but who have chronic sinusitis and/or infertility, are being diagnosed by molecular analysis for CFTR mutations.[2,3]

Upper respiratory tract disease in patients with CF

In the upper respiratory tract dehydration of mucosal fluids and increased sulfation of mucous glycoproteins

results in retention of viscous, tenacious sinus secretions and predisposes to bacterial infection, which is manifested in virtually all CF patients as chronic pansinusitis. Such infection further stimulates mucus production, perpetuating chronic sinusitis. In most instances the inspissated secretions are so thick that perfusion of antibiotics into the secretions is limited and removal of the secretions can occur only by surgical curettage.[4]

Patients with CF are predisposed to chronic sinusitis not only as a consequence of inspissated respiratory secretions but also because of the presence of nasal polyps, which occur with a high frequency in patients with CF. On the other hand, the ostiomeatal complex is often patent in individuals with CF in contrast to the situation in patients without CF, in whom obstruction occurs in a majority of cases of sinusitis.[5] Thus in CF both the retention of viscous, tenacious sinus secretions, as well as nasal obstruction caused by the presence of polyps, predispose to the development of mucosal infection.

Radiographic evidence of sinus disease is invariably present in patients with CF, with a prevalence of 92% to 100% in patients over 2 years of age.[6-12]

The recognition that sinusitis occurs in the majority of patients with CF has been accompanied by a greater appreciation of the role of sinusitis in the exacerbation of lower respiratory tract disease in CF.[12,13]

Nasal polyps and mucoceles

Although sinusitis in patients with CF is thought to be caused mainly by the presence of inspissated mucosal fluids, obstruction of the nose caused by nasal polyps or mucoceles (cyst-like, mucus-containing structures that often erode into surrounding bone) can greatly exacerbate sinus disease. Numerous studies have shown that the incidence of nasal polyposis in patients with CF ranges from 10% to 50%, with the frequency being higher in populations of older patients.[14-21]

Symptoms and signs of sinusitis in patients with CF

Symptoms and signs of sinusitis in patients with CF are similar to those found in patients without CF. However, patients often grow accustomed to these sinusitis-related symptoms and tend not to complain of them.[4,22]

The findings on physical examination are similar to those found with sinusitis in general, except that nasal polyps are often seen arising from maxillary and/or ethmoid sinuses and appearing as gelatinous, gray tissue with very fine blood vessels.

Bacteriology/microbiology

The pathogens that cause sinusitis in patients with CF are similar to those that cause endobronchial infection in these patients, and include *Pseudomonas aeruginosa* most commonly, as well as *Haemophilus influenzae*, streptococci, *Escherichia coli*, *Staphylococcus aureus*, diphtheroids, and anaerobes.[6] The bacteria isolated from the sinuses and from the lungs of a given individual with CF have been shown to be similar, although not identical, in terms of species, antibiotic resistance patterns, and genotypes.[7] Fungi also have been cultured with increasing frequency from the sinuses of patients with CF. Isolation of *Aspergillus fumigatus* from the sinuses is associated with an allergic fungal disease similar immunopathologically to allergic bronchopulmonary aspergillosis.[23] *Aspergillus fumigatus* accounts for the majority of cases, although another fungus, *Pseudallescheria boydii*, has been isolated from patients with CF and may cause a similar syndrome.[24]

Sinus X-Rays

Roentgenographic examination of the sinuses of patients with CF shows panopacification of the sinuses in 92% to 100% of patients beyond 8 months of age.[15]

Therapy for sinusitis in patients with CF

In young children who are not yet colonized by *Pseudomonas* spp, oral antibiotics appear to be efficacious in treating sinusitis, particularly when antibiotics effective against both *Staphylococcus aureus* and *Hemophilus influenzae* are used. However, large dosages and long courses (3 to 6 weeks) of antibiotics (eg, cefaclor, amoxicillin-clavulanate, cefuroxime axetil, azithromycin, and clarithromycin) appear to be required, presumably because penetration of the antibiotics and drainage of secretions is poor in patients with CF. In older children who are colonized with *Pseudomonas aeruginosa*, antipseudomonal antibiotics, such as oral quinolones (ciprofloxacin, ofloxacin), can be used. Treatment failures are frequent, however, and intravenous antibiotics, such as tobramycin and/or ceftazidime, are often required to control acute exacerbations of chronic sinusitis.

Because thickened secretions continue to form in the sinuses, particularly during upper respiratory infection, and because oral or intravenous antibiotics rarely sterilize the sinuses of patients with CF, long-term treatment may require intermittent irrigation of the sinuses with antipseudomonal antibiotics every 3 to 4 weeks.[25]

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L. ANTIBIOTICS

SUMMARY STATEMENTS

1. Antibiotics are the primary therapy for bacterial sinusitis.
2. The most common bacteria observed are polysaccharide-encapsulated organisms, of which 30% to 40% are produce β-lactamase.
3. The appropriate duration of antibiotic therapy for acute sinusitis is not well defined, although a 14-day course is probably adequate for most patients with acute disease.
4. Chronic sinusitis should be treated until the patient is well for 7 days before stopping therapy.
5. Choice of antibiotic should be based on predicted effectiveness, cost, and side effects.

Although adjunctive measures, such as saline nasal lavage, decongestants, and corticosteroids, may be important in enhancing mucociliary clearance, antibiotics remain the primary form of medical treatment for sinusitis (Table I). Because direct antral puncture is rarely performed in clinical practice, the appropriate choice of antimicrobial agent should be based on likely bacterial pathogens consistent with the clinical history. Regardless of whether the sinus disease has been acute or chronic, the most common bacteria observed are polysaccharide-encapsulated organisms, including *Streptococcus pneumoniae*, *Morexella catarrhalis*, and *Haemophilus influenzae*. Resistance to *Streptococcus pneumoniae* mediated by alteration in penicillin-binding proteins has been increasing over the last several years, and at present, 20% to 30% of strains are relatively resistant.[1] In most geographic areas, 30% to 40% of *Haemophilus influenzae* and 75% to 95% of *Morexella catarrhalis* are β-lactamase positive.[2] Although the relative prevalence of these bacteria will vary regionally, if one assumes that these bacteria are equally likely to cause disease, then 20% to 30% of the polysaccharide pathogens will be resistant to amoxicillin. Studies of patients with more protracted or refractory disease suggest that anaerobic bacteria and staphylococci are increasingly being identified as pathogens.

In many geographic areas amoxicillin remains a reasonable initial antibiotic choice in patients with uncomplicated disease. It is generally effective, it is relatively inexpensive, and side effects are rare. The only significant drawback of amoxicillin is lack of effectiveness against β-lactamase–producing strains. This negative aspect can be overcome by the addition of a β-lactam salt, potassium clavulanate, which can inhibit the β-lactamase enzymes. Such a combination of amoxicillin/potassium clavulanate is typically effective against most β-lactamase–producing *Haemophilus influenzae*, *Morexella catarrhalis*, *Staphylococcus aureus*, and many other anaerobic bacteria.[3] Unfortunately, intolerable gastrointestinal symptoms, including cramping and diarrhea, may occur. These side effects usually reverse quickly when the agent is discontinued.

Sulfamethoxazole-trimethoprim (SMX-TMP) is another excellent consideration as "first-line" therapy for sinusitis. Although allergic reactions can occur with sulfamethoxazole, SMX-TMP has an adequate antimicrobial spectrum, particularly for gram-negative organisms, it is inexpensive, and it only requires twice daily administration. Resistance by group A streptococci and *Streptococcus pneumoniae* has been observed.

Erythromycin-sulfisoxazole is a combination more typically used in pediatric patients, which has an enhanced spectrum. Although erythromycin is largely effective against gram-positive cocci, sulfisoxazole adds a complementary gram-negative spectrum. The disadvantages of erythromycin/sulfisoxazole include frequent gastrointestinal side effects and generally the need for 4 times daily administration. Children can exhibit sensitivity to the sulfa component in the form of urticaria or severe erythema multiform.

Cephalosporins are commonly prescribed for both acute and chronic sinusitis.[4] First-generation agents, such as cephalexin, have the disadvantage of poor *Haemophilus influenzae* coverage. Cefaclor, a second-generation drug, provides better coverage, but resistance to some *Haemophilus influenzae* and *Morexella*

catarrhalis has been reported. Cefaclor appears to be optimally effective only with 3 times daily administration (now twice daily) and has a relatively high prevalence of serum sickness–like reactions.[5] Both cefadroxil and cefaclor have rather poor activity against certain gram-negative bacteria.

Third-generation cephalosporins include cefuroxime axetil, cefpodoxime proxetil, and cefprozil. These drugs all have the advantage of twice daily administration and significantly enhanced activity against β-lactamase–producing *Haemophilus influenzae*, *Morexella catarrhalis*, and *Staphylococcus aureus*.[6,7] All of these agents are available as suspensions, and therefore they can be easily used in young children.

Cefixime and ceftibuten are third-generation cephalosporins, which can be given orally once daily. When cefixime was first introduced, reduced activity against *Streprococcus pneumoniae* was rarely of clinical significance.[8] However, with the emergence of increasingly resistant strains, clinical effectiveness has been compromised.[9]

Other appropriate broad-spectrum agents include 2 erythromycin analogs, azithromycin and clarithromycin, and loracarbef (a carbacefem). Similar to several of the newer cephalosporins, clarithromycin and loracarbef enhance compliance because of twice daily administration. Azithromycin, given once daily, lacks a specific indication for sinusitis but has been used for its treatment. In adults, ciprofloxacin, levofloxacin, grepafloxacin, and trovafloxacin presently have specific indications for the treatment of sinusitis. Sparfloxacin may show enhanced gram-positive coverage but lacks the specific indication for sinusitis and has a significant risk for phototoxicity. There has been concern about adverse effects on developmental joint formation with all quinolones.[10]

In protracted or severe cases of sinusitis, the possibility of anaerobic pathogens should be considered. Generally, these organisms are sensitive to penicillin, and many of the others, including *Bacteroides* spp, respond to amoxicillin/clavulanate. If the clinical course suggests that an anaerobe is a likely pathogen, the use of clindamycin or metronidazole is another alternative. In particularly refractive cases combination therapy with a broad-spectrum antibiotic and metronidazole or clindamycin should be contemplated. That such a combination is often successful should not be surprising considering that 25% of cultures from patients with chronic (>3 weeks) sinusitis yield multiple isolates.[11] Although clindamycin is generally well tolerated, patients should be alerted to the possibility of pseudomembranous enterocolitis and told to contact their physician with any sign of diarrhea.

The appropriate duration of antibiotic therapy for sinusitis is not well defined. A 14-day course of antibiotic may be adequate for most patients with acute disease. If there is no clinical improvement within 5 days of initiating antimicrobial therapy, an alternative antibiotic should be considered.

Chronic sinusitis generally requires prolonged treatment, presumably because the underlying mucosa is more significantly diseased, and routes of drainage are less optimal. Patients may require 3, 4, or even 6 weeks of treatment before resolution is observed. On the basis of clinical experience, it would seem appropriate that the course of antimicrobial treatment be continued for at least 1 week after the patient appears symptom free.

The prophylactic use of antibiotics in patients with recurrent sinusitis can be considered, particularly in the setting of "borderline" immune or anatomic abnormalities. Often, these patients lack clear-cut indications for intravenous gamma globulin or surgical intervention and yet continue to have a poor clinical course with their sinusitis. An initial approach with antibiotic prophylaxis might include initiating antibiotic therapy at the first sign of experiencing nasal symptoms. If this fails, the next step would include once daily administration of an antibiotic during the fall and winter, when viral upper respiratory infections are more likely to compromise ostiomeatal clearance. Regrettably, there are no published studies on antibiotic prophylaxis in patients with recurrent sinusitis. However, the potential value of this approach is suggested from studies of children with recurrent otitis media.[12,13]

TABLE I. Antibiotics for sinusitis

Antibiotic	Pediatric dosage	Adult dosage
Amoxicillin	13 mg/kg TID	250-500 mg TID
Amoxicillin/ potassium clavulanate	13 mg/kg TID*	250-500 mg TID*
	22.5 mg/kg BID*	500-875 mg/kg BID*
Erythromycin/ sulfisoxazole	12.5/37.5 mg/kg QID	–
Sulfamethoxazole/ trimethoprim	200/40 mg/kg BID	800/160 BID
Cefaclor	13 mg/kg TID	250-500 mg TID
Cefadroxil	15 mg/kg BID	500-1000 mg BID
Cefuroxime	7.5 mg/kg BID	250-500 mg BID
Cefpodoxime	5 mg/kg BID	200-400 mg BID
Cefprozil	15 mg/kg BID	250-500 mg BID
Cefixime	8 mg/kg QD	400 mg QD
Ceftibuten	9 mg/kg QD	400 mg QD
Loracarbef	7.5 mg/kg BID	200-400 mg BID
Azithromycin	5 mg/kg QD†	250 mg QD†
Clarithromycin	7.5 mg/kg BID	500 mg BID
Ciprofloxacin	—	500-700 mg BID
Levofloxicin	—	500 mg QD
Grepafloxacin	—	400 mg QD
Trovafloxacin	—	200 mg QD
Sparfloxacin	—	100 mg QD‡
Clindamycin	5 mg/kg TID	150-450mg TID, QID
Metronidazole	7.5 mg/kg TID	250-500mg TID, QID

Modified from Virant FS, Shapiro GG. Sinusitis. In: Tierney DF, editor. Current pulmonology. St Louis: Mosby. In press.
TID, Three times daily; *BID*, twice daily; *QID*, four times daily; *QD*, every day.
*Based on amoxicillin component.
†Typically 5-day course after 10 mg/kg (pediatric) or 500 mg (adult) load equals 10 days total therapy.
‡After 200 mg loading dose.

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M. ANTIHISTAMINES

SUMMARY STATEMENTS

1. There are no data presently to recommend the use of H_1 antihistamines in acute bacterial sinusitis.
2. There may be a role for antihistamines in chronic sinusitis, especially in patients with allergic rhinitis.

Both first- and second-generation H_1 antihistamines have a major role in the treatment of allergic rhinitis, allergic conjunctivitis, cutaneous allergic disorders, and anaphylaxis. A concise role for the use of these agents in the treatment of sinusitis has not been elucidated. There are no recent studies evaluating the role of H_1 antihistamines in the treatment of sinusitis. Several review articles and book chapters on treatment of acute and chronic sinusitis have been written recently, but rarely is the use of antihistamines addressed.[1-7] Despite these articles, antihistamines are widely sold for this condition.[8,9]

At the present time, there are no data to recommend the use of H_1 antihistamines in acute bacterial sinusitis. There may be a role for these agents in chronic sinusitis in conjunction with rhinitis. In patients with allergic rhinitis, H_1 antihistamines may help as adjuvant therapy for chronic sinusitis and help prevent its occurrence by reducing seasonal edema. In general, second-generation antihistamines that are devoid of anticholinergic drying effects should be selected.

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N. α-ADRENERGIC DECONGESTANTS

SUMMARY STATEMENTS

1. Both topical and oral decongestants are often used in the therapy of acute or chronic sinusitis because it is thought that they may widen ostial patency and reduce turbinate swelling.
2. Prospective studies are lacking and are needed to assess the value of α-adrenergic agents in the treatment of sinusitis.

The basis for ancillary therapy of sinusitis with α-adrenergic decongestants derives from anecdotal accounts and personal beliefs rather than definitive data. The recent appreciation that noninfectious inflammation in the nose and sinus predispose to infectious sinusitis has stimulated renewed interest in the development and documentation of efficacious ancillary therapies that could supplement or abrogate antibiotic use.[1] It has been thought that both topical and oral decongestants used in the therapy of acute or chronic sinusitis widen ostia potency and reduce turbinate swelling, thus promoting sinus and nasal ventilation.

Pharmacology

Decongestants can produce their effects by 2 broad mechanisms. First, direct-acting sympathomimetic drugs activate α-adrenergic receptors resulting in vasoconstriction.[2] Second, indirect-acting sympathomimetic agents function by being taken up into the prejunctional nerve terminal where they displace norepinephrine from storage vesicles. The dispensed norepinephrine is then released from the sympathetic nerve terminal to

postjunctional α-adrenergic receptors producing vasoconstriction.

Side effects of decongestant

Topical agents used as nasal sprays act rapidly, usually within minutes, and therapeutic doses have no systemic side effects. Rebound hyperemia or chemical rhinitis, however, is a frequent side effect in patients who use the drugs over an extended period of time.

Oral decongestants cause generalized constriction of blood vessels, and increased arterial pressure is always of concern. Most available oral agents, however, cause blood pressure elevations in normal persons only at doses that significantly exceed the recommended dose. Other possible adverse effects are reflex bradycardia, urinary retention, mydriasis (with effects on glaucoma), and effects on endocrine and other regulators of metabolic function. Only 3 drugs are commonly used as oral decongestants: phenylpropanolamine, pseudoephedrine, and phenylephrine. Direct-acting sympathomimetics, such as phenylephrine and oxymetazoline, activate α_1- and α_2-adrenergic receptors, respectively, and both cause vasoconstriction and a decrease in nasal congestion. These drugs can be used topically during acute upper respiratory infections to reduce edema, and it is possible that they may help keep sinus ostia patent.[3] Oxymetazoline can be used to shrink the nasal mucosa followed by antiinflammatory steroid nasal preparations to help treat chronic sinusitis.

Prospective studies are lacking and are needed to access the value of α-adrenergic agents in the treatment of chronic sinusitis.[4,5]

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O. GLUCOCORTICOSTEROIDS

SUMMARY STATEMENTS

1. The use of systemic steroid therapy for sinus disease has not been studied systematically in a well-controlled or blinded manner.
2. A few recent studies suggest that the addition of intranasal steroids as an adjunct to antibiotic therapy is beneficial in the treatment of sinusitis.
3. The relative safety of directed therapy with inhaled steroids makes their use a likely mode of treatment for sinusitis or at least for treatment of any underlying rhinitis.

Researchers and clinicians agree on the potential usefulness of corticosteroids as potent antiinflammatory agents and the fact that sinusitis is an inflammatory disease. The leap from this logical association to clinical proof of the effectiveness of corticosteroids in managing sinus disease has been difficult.

As in asthma, the eosinophil plays a major role in sinusitis. Harlin et al[1] examined paranasal sinus tissue from 26 patients with chronic sinusitis with or without allergic rhinitis or asthma. Eosinophilic infiltration of the mucosa was a characteristic finding as was a marked association between the presence of extracellular deposition of eosinophilic major basic protein and damage to the mucosa. There was a notable similarity between the histopathology of the paranasal respiratory epithelium and that seen in asthma, in which the use of corticosteroids to decrease eosinophil-related inflammatory damage is well accepted. The authors conclude that the eosinophil acts as an effector cell in chronic inflammatory disease of the paranasal respiratory epithelium just as in the inflammation associated with asthma. Hamilos et al[2] also confirmed the importance of eosinophilic inflammation in chronic hyperplastic sinusitis and observed a strong correlation between GM-CSF and IL-3 cytokines with eosinophilic accumulation.

In a clinical study[3] aimed at developing a technique for assessing the severity of chronic sinusitis, 104 patients undergoing surgery for chronic sinusitis were evaluated with computed tomography, total serum IgE measurement, measurement of specific IgE concentration for common allergens, and peripheral eosinophil count. Extensive disease was present in 39% of patients and was correlated with asthma, specific IgE antibodies, and eosinophilia. The presence of peripheral eosinophilia indicated a high likelihood of extensive sinus disease.

Increased numbers of activated eosinophils have been found in nasal polyposis, a risk factor for sinusitis. Although examination of the nasal mucosa of unaffected subjects revealed no activated eosinophils, examination of nasal polyps and the nasal mucosa of patients with polyps revealed that a significant portion of eosinophils that were present were activated. This portion declined after topical corticosteroid treatment, suggesting that the inflammatory reaction was reduced.[4-6]

The antiinflammatory activities of corticosteroids include decreased vascular permeability, inhibition of release, and/or formation of mucous secretogogues, including histamine, leukotrienes, platelet-activating factor, and prostanoids, as well as inhibition of inflammatory cell infiltration.

Topical corticosteroids have a clear effect on inflammatory cell influx into the nasal mucosa after nasal antigen challenge.[7] Pretreatment of nasal mucosa with inhaled steroid has been shown to modify both the immediate- and late-phase response after antigen challenge.[8] Glucocorticosteroids also inhibit antigen-induced nasal hyperresponsiveness to histamine.[9]

Numerous clinical trials attest to the efficacy of topical corticosteroids in controlling symptoms of allergic rhinitis.[10,11]

The similarity of the respiratory epithelium in the nose and paranasal sinuses, as well as the contiguity of these areas, would lead one to expect that sinusitis might also be treatable with inhaled corticosteroids. Yet anatomic barriers, including the narrow ostiomeatal complex and the wall-like ethmoid bulla, make access to the interior sinus mucosa unlikely, unpredictable, and unmeasurable. Whether nasal steroid therapy can sufficiently decrease nasal inflammation and improve mucociliary transport to the point where the ostiomeatal complex becomes competent is unknown.

The use of systemic steroid therapy for sinus disease has also not been studied. Nevertheless, it is not uncommon for physicians to use a course of prednisone in conjunction with antibiotics for persistent sinusitis. This therapy is based on the theory that inflammation, and not just infection, participate in difficult-to-manage sinus disease.

The relative safety of nasal steroids makes their use an attractive mode of treatment for sinusitis. French investigators[12] compared sinus irrigation with the steroid tixocortol pivalate along with neomycin versus neomycin alone in a double-blind, randomized trial of 60 patients with chronic sinusitis who performed intranasal lavages with these medications daily for 11 days. Symptoms and manometric readings were used to assess ostial obstruction. The addition of tixocortol decreased obstruction from 36% to 69%, apparently due to diminishing the inflammatory process.

Qvarnberg et al[13] evaluated budesonide nasal aerosol versus placebo added to a regimen of sinus washings and erythromycin for chronic or recurrent sinus disease in 40 patients. This was a double-blind, randomized evaluation. The budesonide group had less nasal symptoms and facial pain. There were no other significant differences in outcome between the groups, with similar numbers in each group having positive sinus cultures and inflammatory cells in lavage fluid and requiring sinus surgery.

The utility of nasal flunisolide in conjunction with amoxicillin/clavulanate for treating acute sinusitis and for preventing recurrences has been reported.[14] In a multicenter, double-blind, randomized, parallel trial, patients aged 14 years or older with radiograph-documented sinusitis received amoxicillin/clavulanate (500 mg 3 times daily) for 3 weeks. One half of the group received flunisolide nasal spray (100 μg), and the other half received placebo, 3 times daily for 3 weeks. After finishing the antibiotic, the flunisolide or placebo spray therapy was continued for another 4 weeks. Both groups improved significantly. Global assessment of effectiveness and turbinate swelling were significantly better with flunisolide than placebo during the first 3 weeks of therapy. During the prophylactic phase of therapy, there was no difference in recurrences of sinusitis between the 2 groups. The authors conclude that the addition of flunisolide nasal spray as an adjunct to antibiotic for the treatment of sinusitis was judged the most effective treatment in the global evaluation and tended to improve symptoms, to decrease inflammatory cells in nasal secretions, to normalize ultrasound, and to aid in regression of radiographic abnormalities compared with placebo spray. However, despite symptomatic improvement in many patients, many had continuing symptoms, radiographs remained abnormal in 80% of patients, and there were frequent recurrences. In a more recent study 89 children received 3 weeks of amoxicillin/clavulanate (40 mg/kg 3 times daily) plus nasal budesonide 50 mg per nostril twice daily (n = 43) or placebo (n = 46). Cough and nasal discharge significantly improved only in the flunisolide group at the end of the second week, confirming the ancillary role of topical steroids.[15]

Currently, nasal steroid therapy has become an accepted adjunct in treating both acute and chronic sinusitis. Several intranasal steroids are now available in the US: flunisolide, beclomethasone, triamcinolone, fluticasone, budesonide, and mometasone. Each of these has been proven to be effective in treatment of allergic rhinitis and may be a useful addition in sinus disease.

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P. ADJUNCTIVE THERAPIES INCLUDING SALINE, MUCOLYTICS, AND EXPECTORANTS

SUMMARY STATEMENTS

1. There are inadequate data to recommend use of wetting agents as individual therapy for sinusitis.
2. Clinical practice, as well as folklore, supports the use of wetting agents for symptomatic treatment as part of a pharmacologic regimen.
3. There are several scientific studies that imply, but do not directly confirm, a role for these agents in sinusitis.
4. The safety profile of each agent should be carefully considered for individual patients.
5. Use of all these agents as prophylaxis for exacerbations of chronic sinusitis is empiric and not supported by clinical data.

Medical management of sinusitis is based on empiric goals. Adjunctive agents are frequently prescribed in addition to antibiotics to aid drainage of retained secretions through the sinus ostia into the nasal cavity. Use is based on clinical experience.[1-3]

Rationale for adjunctive medication to antibiotics

Management of acute sinusitis usually includes an oral antibiotic. However, it has been estimated that about 45% of cases will resolve without the use of antibiotics. In one study of 50 patients with chronic anterior or posterior purulent nasal discharge, 20 patients were treated with topical dexamethasone and decongestant sprays, 20 patients with the same medications plus topical neomycin, and 10 with matched placebo sprays or propellants alone. Significantly more patients receiving the active treatments demonstrated improvement in symptoms compared with those receiving placebo. The authors concluded that improving sinus drainage permitted host mechanisms to recover and that topical antibiotics provided no additional benefit. However, systemic antibiotics were not used.[4] In another study of 80 allergic children with asthma 4 to 14 years of age, sinus abnormalities were detected on x-ray film in 55 patients. The findings of mucosal thickening greater than 2 mm, opacification, or air-fluid level were defined as abnormal radiographs. Those children with purulent postnasal drip (n = 13) were treated with ampicillin, phenylephrine, and tripolidine. In 42 children without purulent drainage, the ampicillin was replaced by intranasal beclomethasone spray for 1 month, together with phenylephrine and tripolidine. In both treatment groups sinus radiographs improved with a decrease in severity of the asthma. However, the presence of bacterial sinusitis was not adequately documented, and all children received topical decongestants, and therefore that the role of the intranasal beclomethasone could not be adequately assessed.[5]

Both these studies suggest that systemic antibiotics are not required. However, the serious complications of sinusitis, such as intracranial extension of infection, have decreased in frequency since the advent of antibiotics. Consequently, antibiotics are now generally prescribed as primary therapy.

Although treatment of sinusitis is associated with resolution of radiographic abnormalities and improvement in the symptoms of asthma, scarce data exist on the efficacy of treatment for chronic sinusitis. On the basis of our understanding of the pathophysiology of chronic sinusitis, medical management should be designed to effectively treat infection in the sinuses, reduce tissue swelling in the region of the sinus ostia to facilitate drainage of retained secretions, promote ciliary function, and maintain ostial patency both during and after therapy. Combination treatment protocols are commonly used on the basis of clinical experience rather than formal trials.[6]

Pharmacologic agents

Guaifenesin

Guaifenesin (glyceryl guaicolate; 3-(2-methoxyphonoxy)-1, 2-propanediol) is a water- and alcohol-soluble substance that has been used as an expectorant to loosen phlegm and bronchial secretions in the symptomatic management of coughs associated with the common cold, bronchitis, laryngitis, pharyngitis, influenza, and measles, as well as sinusitis when these conditions are complicated by tenacious mucus and/or mucus plugs and congestion. By reducing viscosity of secretions, guaifenesin increases the efficiency of the cough reflex and of ciliary action in removing accumulated secretions from the trachea and bronchi. There is clinical evidence that guaifenesin is an effective expectorant in that it increases expectorated sputum volume over the first 4 to 6 days of a productive cough, decreases sputum viscosity and difficulty in expectoration, and improves associated symptoms. There is currently insufficient evidence to support efficacy of the drug as an adjunct in sinusitis.[7] High-dose guaifenesin (1200 mg twice daily) has been used empirically for its ability to thin tenacious respiratory secretions on the basis of clinical efficacy in chronic bronchitis.[8] No clinical trials have been reported in sinusitis to demonstrate its efficacy. Guaifenesin is considered a pregnancy category C drug because animal reproduction studies have not been conducted.[9]

Iodine

Iodine-containing compounds, such as potassium iodide or iodinated glycerol, might be expected to have similar effects as guaifenesin. However, they are of limited clinical use, perhaps because of the potential adverse

effects of iodine hypersensitivity on chronic ingestion. Potassium iodide syrup is considered an expectorant in the symptomatic treatment of chronic pulmonary diseases in which tenacious mucus complicates the problem, including bronchial asthma, bronchitis, and pulmonary emphysema. Side effects may include gastrointestinal upset, metallic taste, minor skin eruptions, nausea, vomiting, and epigastric pain.[10] No studies in sinusitis have been reported.

Antibiotics as mucolytics

The mucolytic properties of antibiotics have been investigated. Norfloxacin reduced the elastic modulus (G') of mucus in one study of patients with chronic sinusitis,[11] and erythromycin reduces respiratory glycoconjugate secretion in an in vitro preparation of human airways.[12] On the basis of the scarcity of current data, antibiotics should be selected primarily on their antimicrobial profile.

Nonpharmacologic adjuncts

Many nonpharmacologic measures are advocated for symptomatic relief of acute sinusitis. Because scientific data on efficacy are lacking, physicians may dismiss some of these measures as folk medicine. For many patients, however, 1 or more of these treatments may provide effective relief of distressing symptoms while the infection is resolving. Unfortunately, most of these measures are short-lived in effectiveness, and they must be repeated as symptoms recur.

Saline

Steam and saline prevent crusting of secretions in the nasal cavity and especially in the region of the ostiomeatal complex. By liquefying secretions, they also help mucociliary clearance and reduce the feeling of facial pressure. Repetitive saline applications also act as a mild vasoconstrictor of nasal blood flow.[13,14] Saline nebulizations were shown to be beneficial in one study in children.[15]

Nasal instillation of saline spray 2 or 3 times a day, between steam treatments, provides a mild decongestant action. Saline spray also helps to liquefy nasal secretions and moisturize the nasal and sinus mucosa. Irrigation with saline, or antral lavage, is a familiar procedure, but one which is best left to ear, nose, and throat surgeons after aspiration of purulent matter from the sinuses of severely congested patients.[11]

Steam

The traditional method of steam inhalation is to instruct the patient to do the following:

1. Pour boiling water in a pan or basin on a low table.
2. Sit at the table with a towel draped over the head to make a tent over the pan of water.
3. Hold the face a few inches above the water and breathe through the nose for approximately 10 minutes.

This procedure liquefies and softens crusts while moisturizing the dry, inflamed mucosa.

Many patients find that 2 such treatments a day provide some effective symptomatic relief. If a patient is for some reason unable to perform this simple procedure, using a vaporizer or a facial sauna or taking long, hot showers may be beneficial, but none of these alternatives is a good substitute for the hot water and tent method. The ritual of boiling the kettle, preparing the tent, and relaxing over the steamy brew probably has a good psychologic effect, enhancing the therapeutic benefit. Patients, however, should be reminded not to breathe steam directly from a boiling kettle.

Propylene glycol

Although propylene/polyethylene glycol and saline have been used in clinical studies as placebos, Spector et al[16] investigated their possible therapeutic role as wetting agents. Eighteen patients with perennial rhinitis were studied for 4 weeks of active treatment. Both agents produced symptomatic improvement and objective improvements in airway obstruction at 2 and 4 weeks.

Hot air

Some patients report benefit from breathing hot, dry air. This procedure dries secretions and also generates or enhances a feeling of well being. Air at 41˚C has been reported to be virucidal in vitro,[19] and some commercially available devices provide heated air. However, maxillary sinusitis is usually bacterial, not viral, and only anecdotal evidence supports the premise that hot, dry air is virucidal in vivo. Several studies with different devices have yielded differing results.[17-20]

Astringents

Adding pine oil, mentholated preparations such as Vicks VapoRub, oil of eucalyptus, or similar aromatics may add to the beneficial effect of the steam treatment. Such additions may help relieve stuffiness or at least give a subjective sensation of increased air flow. Again, there are no scientific data to support this view, but patients think they work.

Spicy foods

Garlic has an active ingredient (n-allythiosulphinate) that provides short-lived decongestant effects. Eating foods highly seasoned with garlic has been considered therapeutic. Ziment[21] included a recipe for his wife's garlic-and-chicken soup in his textbook on treatment of respiratory ailments. Encapsulated garlic powder is sometimes recommended by patients who do not like the flavor of the garlic in their food. Chewing horseradish root, which is available from many food markets, is another home remedy reported effective in "clearing the sinuses" by some patients. Again, no scientific data support its reported benefits.

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Q. INTRAVENOUS IMMUNE GLOBULIN

SUMMARY STATEMENTS

1. Immunodeficiency is one of the underlying factors for the development of chronic and recurrent sinusitis.
2. Intravenous immune globulin (IVIG) is indicated for use in patients with impaired humoral immunity.

Immunodeficiency is one of the underlying factors for the development of chronic and recurrent sinusitis. At the present time, intravenous immune globulin (IVIG) is indicated in the treatment of antibody deficiency syndromes, including X-linked agammaglobulinemia, common variable immunodeficiency, Wiskott-Aldrich syndrome, and hyper-IgM syndrome.[1-7] The indication for IVIG in other conditions, such as selective antibody deficiency syndromes or IgG subclass deficiencies, is more controversial.[8-12] Both qualitative and quantitative defects in the humoral immune system may result in recurrent or chronic (>3 months) sinopulmonary bacterial infections.[13] In patients with immunoglobulin deficiency, complications of chronic sinusitis include subperiosteal abscess and intracranial abscesses. Infected sinus cavities may also act as a focus for systemic infections because the response to antibiotic therapy is limited without an effective humoral immune system. In such situations replacement of deficient or dysfunctional gamma globulin with IVIG is a potentially life-saving therapy.

The use of IVIG is indicated for use in patients with qualitative or quantitative humoral immune deficiency in which chronic sinusitis may be a significant associated disease. In those with specific antibody deficiency with normal total immunoglobulins, chronic sinusitis is often a major complication and at times may be the only significant infectious process. Many immunodeficient patients undergo multiple surgical procedures and receive chronic antibiotic therapy without significant resolution of their sinusitis. In these patients who fail medical and surgical therapies and have documented failure to respond to polysaccharide antigens, a trial of IVIG therapy is warranted.[1,7] Treatment is maintained throughout the fall and winter months and may be discontinued during the summer if the patient is disease free.

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R. SURGICAL CONSIDERATIONS

SUMMARY STATEMENTS

1. Antral puncture and irrigation is an office procedure that has a place in the management of acute ethmomaxillary sinusitis refractory to medical therapy or in acute sinusitis in an immunosuppressed patient in whom early identification of pathogenic organisms is paramount.
2. The term "functional endoscopic sinus surgery" is based on the clinical and experimental findings that ostial obstruction is the final common pathway in the development of sinusitis.

The surgical approach to sinus disease has undergone a dramatic transition, resulting from renewed insights into sinus physiology and the widespread use of rigid nasal telescopes. Earlier in this century, several authors were the first to note the importance of the ethmoid sinuses in the etiology of frontal and maxillary sinusitis disease.[1,2] Our current knowledge indicates that limited and localized inflammation in the anterior ethmoid sinus, and particularly the ostiomeatal complex, is sufficient to cause frontal and/or maxillary sinusitis. Whereas past traditional sinus surgery often attempted to completely strip inflamed mucosa from the larger sinuses, believing it to be irreversibly diseased, current surgical therapy aims to remove only the causative ethmoid disease and reestablish ventilation and drainage of the larger dependent sinuses. Modern endoscopic sinus surgery is thus functional and restorative because its goal is to restore normal sinus physiology with minimal removal of mucosa.

Nasal endoscopy aids in the diagnosis of ethmoid disease. It is more sensitive than plain roentgenography and more cost-effective than sinus computed tomography for diagnosing ethmoid sinusitis, and repeat endoscopic evaluations make it possible to more objectively follow a patient's response to medical and surgical therapy.[3]

Surgical approaches

The surgical approaches to sinusitis can be broadly categorized into traditional and endoscopic methods. Traditional surgical approaches can be further classified by the particular sinus on which they were performed and include traditional ethmoid, maxillary, and frontal sinus surgery.[4,5]

The endoscopic approach in sinus surgery

The term "functional endoscopic sinus surgery" is predicated on the clinical and experimental finding that ostial obstruction is the final common pathway in the development of sinusitis.[6] When 2 mucosal surfaces come into firm contact, disruption of normal mucociliary flow occurs at that point.[3] The potential for obstruction of secretions and persistent local inflammation then exists, and sinusitis may ensue. The key to the functional endoscopic approach is therefore the accurate identification of areas in which ventilation or mucociliary clearance may be obstructed. Recent advances in endoscopic surgery involve computer-assisted navigation within the naris and powered instrumentation.[7-9]

Operative intervention

Endoscopic sinus surgery can be performed under either local or general anesthesia on an outpatient basis. Patients with asthma or other underlying medical conditions are usually kept overnight for observation. If the procedure is performed under local anesthesia, intravenous sedation is administered by the anesthesiologist, and the patient is monitored. The entire operation is carried out under endoscopic visualization through the nasal cavities and involves no external incisions. The extent of surgical dissection is dictated by the amount and location of disease identified on preoperative computed tomography, as well as that encountered during surgery.[10] Surgical intervention for the treatment of recurrent sinusitis carries a severe complication rate of 0.5% in 200,000 cases per year.[11]

Conclusions

In summary, great advances in the diagnosis and surgical treatment of sinusitis have been made in the last decade, principally through the use of nasal endoscopy and coronal computed tomographic scanning. The rigid nasal telescope has dramatically improved our ability to visualize the ostiomeatal complex, which is the critical area in the pathogenesis of chronic sinusitis. Functional endoscopic surgery of the sinuses results in significant subjective improvement in the great majority of cases.[12]

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