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Prim Care Clin Office Pract
35 (2008) 11–24

PRIMARY CARE:
CLINICS IN
OFFICE PRACTICE

The Diagnosis and Management of Acute and Chronic Sinusitis

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The objective of this article is to review the diagnosis and management of both acute and chronic sinusitis. Areas discussed include the prevalence of disease, our current understanding of disease pathogenesis, diagnosis, and contemporary treatment.

Prevalence and disease burden

Sinusitis affects an estimated 16% of the adult population in the United States, which translated into an astonishing 5.8 billion dollars of direct health care costs in 1996 [1]. The great majority of patients present to their primary care physician, resulting in approximately 18 million office visits a year. From 1990 through 1992, total restricted activity days numbered 73 million [2]. Degree of impairment from sinusitis is substantial, and is comparable to other chronic diseases, such as chronic obstructive lung disease, angina, and back pain [3].

Anatomy

The sinuses are air-filled cavities, which are lined with classical, pseudostradified and ciliated columnar epithelium. The host defense system works to keep this pathogen free in a number of ways. In an immunocompetent host, secretory IgA and proper mucocilliary clearance through a patent ostium prevent local mucosal damage.

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0095-4543/08/$ - see front matter
doi:10.1016/j.pop.2007.09.002 *primarycare.theclinics.com*

Proper function of the sinuses involves several key points [4], including: (1) mucus that is of appropriate viscosity, composition, and volume, (2) normal mucociliary flow, and (3) open ostia to allow adequate drainage and aeration.The cilia help to clear secretions by sweeping them toward a patent ostial opening and into the nasal cavity. In the maxillary sinuses, proper ciliary function is especially important because the direction of drainage is against the pull of gravity. The ostiomeatal complex (OMC) is a narrow drainage pathway located in the middle meatus, which allows ventilation of the anterior ethmoid, frontal, and maxillary sinus.

Definitions

Sinusitis can be broadly defined as inflammation of one or more of the paranasal sinuses. Classically, sinusitis is characterized as the following:

Acute—symptoms last less than 4 weeks
Subacute—symptoms last 4 to 8 weeks
Chronic—symptoms last longer than 8 weeks
Recurrent—three or more acute episodes a year

Acute sinusitis can be further defined as an infection of the paranasal sinuses, with accompanying symptoms present for more than 10 days and less than 4 weeks.

To fully define chronic sinusitis has been difficult. Because of the variation in clinical expression of the disease, and the discordance between patient symptoms and objective findings, no one set of diagnostic criteria has been agreed on by all clinicians. Furthermore, before much of the microbiologic or pathologic data regarding this disease had been shown, chronic sinusitis was thought to be a chronologic extension of acute sinusitis. However, it is now thought that chronic sinusitis is a much different disease. In contrast to acute sinusitis, most chronic sinusitis is not an infectious disease and is better thought of as an inflammatory disease, much akin to asthma.

Pathogenesis and contributing factors

Acute sinusitis

Several factors promote the development of acute sinusitis. In most cases, bacterial sinusitis is preceded by a viral upper respiratory infection, which in turn leads to sinus inflammation and obstruction of the OMC. As a result, drainage and ventilation of the maxillary, anterior ethmoid, and frontal sinuses are compromised. Once this occurs, both the pH and oxygen content decrease, the cilia are less functional, mucosa are damaged, and the microenvironment becomes more susceptible to infection. Approximately 0.5% to 2% of viral sinusitis progress into bacterial infections [5]. To distinguish

between bacterial and viral sinusitis can be difficult. Typically viral sinusitis resolves in 7 to 10 days [6], whereas bacterial sinusitis remains persistent [7].

Rhinovirus is the most common viral pathogen and is easily transmissible. In a study of healthy volunteers, 95% of individuals challenged with intranasal rhinovirus drops became infected, and three quarters of them became symptomatic. Within 10 hours, newly replicating virus was found in the nasal secretions [8]. As confirmed by sinus puncture, *Streptococcus pneumoniae, Haemophilus influenza*, and *Moraxella catarrhalis* make up the majority of the community acquired bacterial pathogens [9]. One possible mechanism for introduction of pathogens from the nasal passages into the sinuses may actually be through nose blowing. This processes creates a negative intranasal pressure with such force that nasal fluid is propelled from the middle meatus into the sinus cavity [10].

Chronic sinusitis

The pathogenesis of chronic sinusitis is poorly understood. The mechanisms that contribute to the chronicity of the disease include mucociliary dysfunction, mucostasis, hypoxia, and release of microbial products. However, the initial stimulus and subsequent perpetuation of these processes is unclear. Some theories have implicated anatomic, infectious, allergic, and inflammatory disease, but none have been proven.

Unlike acute sinusitis, the role of ostiomeatal complex blockage is uncertain. In a comparison of CT scans between patients with chronic sinusitis and healthy controls, there was no difference in the patency of the ostiomeatal complex [11]. Also in contrast to acute sinusitis, the role of infection as the driving force behind most chronic sinusitis has been brought into question. While the most common pathogens in acute sinusitis include *Streptococcus pneumoniae, Haemophilus influenzae,* and *Moraxella catarrhalis,* pathogens found in chronic sinusitis are usually a mixture of aerobic and anaerobic bacteria, including *Staphylococcus aureus* and coagulase-negative *Staphylococci.* Whether these organisms are pathologic, or are merely colonizing agents, is difficult to determine. Reports of the prevalence of anaerobic species differ widely, and range from as high as 80% to 100% in children [12], or to as low as 0% to 25% in adults [13,14]. Furthermore, treatment with antibiotics tends to provide only transient benefit. Granted, a small subset of patients with chronic sinusitis may be infectious in nature, but it is usually in association with an underlying immunodeficiency, such as immunoglobulin deficiency, HIV, cystic fibrosis, or Kartagener syndrome [15].

Several other mechanisms of disease have been previously proposed. In these cases, the inflammatory response is against the microbe as an antigen, and not as an invasive pathogen per se. One theory proposes that immune hyperresponsiveness to colonizing bacteria, such as *Staphylococcus aureus,* may play a role in chronic sinusitis with polyps [16]. Yet another theory

proposes that colonizing fungi serve as the antigen, which will be discussed at the end of this article [17].

Regardless of the initial stimulus, the inflammatory process ensues, with a predominance of eosinophils. Furthermore, chronic sinusitis with and without polyps differ in their specific histopathologic presentation. In nasal samples of patients with polyps, there were significantly more eosinophils, plasma cells, and stromal edema compared with those without polyps. The investigators argued that because a substantial difference was found between these groups, they should be treated as separate entities, and not a continuum of one [18].

Perhaps an understanding of the pathophysiology of chronic sinusitis can be gleaned by its close association with other allergic diseases, such as allergic rhinitis, asthma, and aspirin sensitivity. Based on CT studies, anywhere from 74% to 90% of asthmatics have sinus mucosal abnormalities, albeit asymptomatic [19]. In addition, chronic sinusitis was associated with allergic rhinitis in 40% to 84% of adult patients [20]. Even so, a direct causal role between these diseases has never been shown.

Lastly, gastroesophageal reflux (GERD) has been implicated as a cause of sinusitis. Gastric acid can reflux directly into the nasopharynx and, in theory, can cause inflammation of the sinus ostium, and pH probe studies have shown a much higher incidence of GERD in patients with chronic sinusitis. In an uncontrolled study of 19 adults with chronic sinusitis, 68% had symptoms of GERD, and 78% had abnormal esophageal pH probe results. After a subset of these subjects was treated with proton pump inhibitors, 67% had an improvement in sinus symptoms [21].

Diagnosis

Physical examination

The nasal mucosa is best visualized after application of a topical vasoconstrictive agent, such as oxymetazoine, and use of a nasal speculum. One approach to the exam should include notice of the color, edema, character of nasal secretions, presence of polyps, and structure of the nasal septum [22]. Purulent discharge from the middle meatus is highly predictive of bacterial sinusitis [23,24]. Palpation for tenderness of both the maxillary and frontal sinuses are helpful. Because a small proportion of cases of maxillary sinusitis may be caused by tooth infection, one should also check for maxillary teeth tenderness by tapping with a tongue blade [25]. Transillumination of the sinuses is an additional diagnostic test, and is limited to the frontal and maxillary sinuses, as other sinuses are too distal to examine. To examine the maxillary sinus, a light source is placed over the infraorbital rim, and light transmission is observed through the hard palate. The utility of this test is debatable [22].

Imaging

Imaging of the sinuses is usually reserved to confirm the diagnosis, if history and physical are equivocal, or if conventional treatment has failed. Modalities include plain radiograph, CT, ultrasound, and MRI.

Plain X-rays come in several views. The Caldwell (anterior-posterior), Waters (occipito-mental), and lateral films provide views of the frontal sinus, maxillary, and sphenoid sinuses, respectively. Unlike the CT scan, the ethmoid sinus is not well visualized. Significant opacification or mucosal thickening and air-fluid level are all signs of disease; however, there is no ability to predict the response to antibiotics based on the radiographic extent of disease. MRI is best used to evaluate soft tissue structures, and can distinguish between inflammatory and malignant disease. MRI is also useful to determine the extent of the complications of sinusitis, such as intracranial or orbital involvement. Ultrasound, although limited, is an alternative technique to evaluate the maxillary and frontal sinuses without exposure to ionizing radiation. This is an especially viable option for pregnant women.

CT is the modality of choice, and is better able to evaluate the ethmoid sinuses compared with plain X-ray. CT is also much better than MRI for evaluation of boney structures. The ability to visualize detailed anatomy is helpful in preoperative planning. However, CT is unable to distinguish between viral or bacterial sinusitis. In one study, 31 healthy adult volunteers with "a fresh common cold," 71% of whom described nasal or head congestion, underwent CT sinus imaging early on in their illness. Of the patients with congestion, 100% had an abnormality in one or more of their sinuses, compared with 56% of those who did not have congestion. Fourteen subjects returned for repeat imaging, and without interim antibiotics, 79% of the subjects showed either resolution or marked improvement [26]. In addition, a significant number of patients have incidental mucosal changes on CT, in the absence of symptoms [27]. Moreover the extent of mucosal changes on CT does not correlate with severity of symptoms [28,29].

Culture

Identification of the pathologic organism is best done through maxillary sinus aspiration. After sterilization of the puncture site, usually through the lateral wall of the inferior meatus, contents of the maxillary sinus are aspirated. The invasive nature of this procedure often limits its use. As a less invasive approach, endoscopically obtained cultures of the middle meatus, may be a possible surrogate. However, the same organisms have been found to colonize the middle meatus in healthy children, as those with sinusitis, so the mere presence of the organism does not prove infection [30]. In adults, good correlation has been shown between endoscopically obtained cultures of the middle meatus, and those of direct antral culture [31].

Acute sinusitis

The diagnosis of sinusitis is usually made on clinical grounds, which include both the history and physical examination and, if appropriate, diagnostic procedures. Symptoms of acute sinusitis often overlap with those of other diagnosis, such as allergic rhinitis and the common cold. Several studies have attempted to determine the relationship between the signs and symptoms of sinusitis, and benchmarks such as sinus puncture, CT, plain X-ray, and ultrasound.

In a primary care clinic in Norway, 201 patients with a clinical diagnosis of acute sinusitis underwent CT scan. Of these patients, 63% met the clinic's definition of acute sinusitis by having either an air fluid level or total opacification. The presence of two phases of illness, purulent rhinorrhea, erythrocyte sedimentation rate greater than 10 mm, and purulent secretion noted in the nasal cavity, were all independently associated with acute sinusitis, and a combination of three out of four of these criteria gave a specificity of 81% and a sensitivity of 66% [32].

Williams and colleagues [33] conducted a study of adult men who presented to a primary care clinic with either rhinorrhea, facial pain, or a self-suspected diagnosis of sinusitis, and compared their symptoms to findings of sinusitis on X-ray. The overall prevalence of sinusitis was 38%. They found the following symptoms were most sensitive: presence of colored discharge, cough, and sneezing with a sensitivity of 72%, 70%, and 70%, respectively. However, not surprisingly, the specificity of these symptoms was much less (52%, 44%, and 34% respectively). The most specific symptom (93%) was maxillary toothache; however, this was found in only a small subset of patients.

Van Duijn and colleagues [32] reported a study of European patients who presented to their primary care providers. They compared an algorithm of five symptoms, which included preceding common cold, purulent rhinorrhea, pain on bending, unilateral maxillary pain, and pain in teeth, to findings on ultrasound, a technique primarily used in Europe. Even with this set of criteria, the proportion of correct diagnosis was a little over one half. In this study, the most sensitive indicator was history of preceding cold (85%), and most specific indicator was pain in teeth (83%).

Perhaps the gold standard for the diagnosis of sinusitis is the finding of purulent material through maxillary sinus aspiration. In marked contrast to the studies discussed previously, Hansen and colleagues [34] found no independent association between purulent aspirate and the following symptoms: preceding upper respiratory tract infection, maxillary pain, tenderness of maxillary sinus, maxillary toothache, purulent nasal discharge, and visualization of purulent material on the posterior wall of the pharynx. In summary, there are no signs and symptoms of sinusitis that are both highly sensitive and specific. Most will agree that if symptoms persist beyond 7 to 10 days, a diagnosis of bacterial sinusitis should be entertained [35].

Although rare, complications of acute sinusitis can occur through direct, local extension. With antibiotic treatment, complications occur with an estimated frequency of 1 per 10,000 cases [36]. Clinical presentation may include facial edema, cellulitis, orbital, visual, and meningeal involvement. In these cases, aggressive treatment, which may include surgical intervention, is warranted.

Chronic sinusitis

Unfortunately, clinical criteria to diagnose chronic sinusitis, as well as the predictive value of these criteria, are sorely lacking. Historically, the diagnosis of chronic sinusitis was based on several clinical symptoms, similar to the presentation of acute sinusitis, although often less dramatic; however, none of these symptoms are specific to sinusitis. In particular, headache, as the sole presenting symptom, is not likely chronic sinusitis.

On the other hand, nasal endoscopy is useful. Evidence of nasal secretions, nasal polyps, and deformation of the middle meatus have been shown to distinguish patients with extensive sinus disease, as defined by CT image criteria, compared with either the control group or to those with limited disease [37]. Plain X-rays are often insufficiently sensitive to diagnose chronic sinusitis and do not provide the anatomic detail required for preoperative evaluation. Although CT is recommended, this alone is still not evidence enough to make the diagnosis. CT should be performed at least 2 weeks after an upper respiratory infection, and more than 4 weeks after treatment of acute bacterial sinusitis, to evaluate underlying chronic disease. Therefore it is recommended that a combination of clinical signs and symptoms, nasal endoscopy, and CT be used to make the diagnosis of chronic sinusitis.

Treatment

Acute sinusitis

The diagnosis of acute sinusitis prompts countless number of antibiotic prescriptions per year. Although the vast majority of cases of acute sinusitis resolve without treatment, antibiotics are prescribed for an estimated 85% to 98% of cases presented to a primary care clinic [9]. Antibiotics, compared with placebo, do reduce treatment failures in bacterial sinusitis by almost one half (from 31% to 16%) [38]. If culture results are unavailable, the antibiotic should target the most common bacterial pathogens. These include *S. pneumoniae, H. influenzae, and M. catarrhalis*. Antibiotic resistance is on the rise and almost half of *S. pneumoniae* is now resistant to penicillin, and the majority of both *H. influenzae* and *M. catarrhalis* are B-lactamase positive [39]. The choice of antibiotic should take into account a number of factors, such as geographic prevalence of resistance patterns, predicted efficacy, cost, side effects, and ease of ''use.''

The American College of Physicians published practice guidelines for the treatment of acute sinusitis [40]. This position publication was endorsed by a number of groups, including the Centers for Disease Control and Prevention, the American Academy of Family Physicians, the American College of Physicians, American Society of Internal Medicine, and the Infectious Disease Society of America. In this publication they give the following practice guidelines:

1. Sinus radiography is not recommended for the diagnosis of uncomplicated sinusitis.
2. Acute bacterial sinusitis does not require antibiotic treatment, especially if symptoms are mild or moderate.
3. Patients with severe or persistent moderate symptoms and specific findings of bacterial sinusitis should be treated with antibiotics. Narrow-spectrum antibiotics (including amoxicillin, doxycycline and trimethoprim-sulfamethoxazole) are reasonable first-line agents.

Amoxicillin is a reasonable first line antibiotic choice for both adults and children, unless there is a high prevalence of B-lactamase producing strains. The higher dose (90 mg/kg/day) is recommended for children at higher risk of amoxicillin resistance, such as those who attend day care, were recently treated with antibiotics, or are under the age of 2 years. The addition of potassium clavulanate can also counter this antibiotic resistance. The most common side effects include abdominal cramping and diarrhea, which are quickly reversed upon discontinuation of the drug. Trimethoprim-sulfamethoxazole is an alternative antibiotic in penicillin-allergic individuals; however, up to 20% of *S. pneumoniae* may be resistant to this alternative. In a meta-analysis of several randomized trials, folate inhibitors were found to be as effective as the newer, more costly antibiotics [38]; however, even the investigators cede the limitations of their data, so this should be interpreted with caution. In contrast to amoxicillin, doxycycline provides broader antibiotic coverage, including activity against B-lactamase producing strains of *H. influenzae* and *M. catarrhalis*.

First generation cephalosporins, such as cephalexin and cefadroxil, do not provide adequate coverage against *H. influenzae* and should not be used. Second generation cephalosporins, such as cefuroxime axetil and cefprozil, as well as third generation cephalosporins, such as cefpodoxime axetil, and cefdinir, are appropriate choices.

The first ketolide, telithromycin, was initially indicated for acute sinusitis, but this was revoked after reports of severe hepatotoxicity. The fluoroquinolones, including ciprofloxacin, levofloxacin, and moxifloxacin, offer broad-spectrum antimicrobial coverage, and are all indicated for acute sinusitis. Because of the concern for adverse effect on the development of joints, these should be avoided in children. These medications can also prolong the QT interval, so should be used with caution in patients at risk for arrhythmia. No controlled studies have examined the length of treatment. Generally,

antibiotics should be prescribed for 10 to 14 days, or 7 days after the patient is symptom free. If symptoms fail to improve in 48 to 72 hours, it is reasonable to switch to a second line antibiotic. The most commonly prescribed antibiotics are found in Table 1.

In general, antihistamines are not recommended in the treatment of acute sinusitis unless the patient has underlying allergic rhinitis. However, antihistamines have been shown to decrease sneezing and rhinorrhea in the common cold [41,42]. Although topical and oral decongestants are often used in the treatment of the symptoms of sinusitis, no prospective trials have been performed. These agents do have a modest effect in decreasing nasal airway resistance, and in theory may widen the ostia and improve nasal ventilation. Chronic use of topical decongestants beyond 3 to 5 days should be discouraged, as they may result in significant rebound hyperemia and rhinitis medicamentosa.

Nasal corticosteroids have been shown to decrease the inflammatory process of the nasal mucosa after nasal antigen challenge, and can modify both the early and late allergic response. As an extension, it is reasonable to consider that nasal corticosteroids may decrease the inflammatory response in sinusitis. Nasal corticosteroids have been studied as adjunctive therapy to antibiotic therapy and found significant reduction in several symptom scores; in addition, they show no increase in adverse events [43].

Table 1
Oral antibiotics for sinusitis

Antibiotic	Pediatric dosage	Adult dosage
First line therapy		
Amoxicillin	45 mg/kg/day or 90 mg/kg/day divided	500 mg bid
Second line therapy		
Amoxicillin/potassium clavulanate	22.5 mg/kg/day–45 mg/kg/day divided (Dose based on amoxicillin component)	500 mg–875 mg bid
Azithromycin	10 mg/kg/day on day 1, then 5 mg/kg/day on days 2–5	500 mg qd on day 1, then 250 mg qd on days 2–5
Cefdinir	14 mg/kg/day	300 mg bid
Cefpodoxime	10 mg/kg qd	200 mg bid
Cefprozil	15 mg/kg bid	250 mg–500 mg bid
Cefuroxime	15 mg/kg/day bid	250 mg bid
Ciprofloxacin		500 mg bid
Clarithromycin	7.5 mg/kg bid	500 mg bid
Clindamycin	8 mg/kg/day–20 mg/kg/day divided qid	150 mg–450 mg qid
Doxycycline		100 mg–200 mg qd
Gatifloxacin		400 mg qd
Levofloxacin		500 mg qd
Sulfamethoxazole/ trimethoprim	6 mg/kg/day–12 mg/kg/day divided (based on trimethoprim)	800/160 mg bid

However, it should be noted that nasal corticosteroids do not have a Food and Drug Administration-approved indication for treatment of acute sinusitis.

Surgical intervention of acute sinusitis is rare, but may be needed in the case of complications of sinusitis, or in those patients who continue to have severe symptoms and are unresponsive to medical therapy.

Chronic sinusitis

Corticosteroids (CCSs) are potent anti-inflammatory agents, and as such, would seem to be a logical choice to treat chronic sinusitis. Although intranasal CCSs are unlikely to reach the paranasal sinuses, they do improve nasal congestion, which is often a significant symptomatic component in chronic sinusitis. Intranasal CCSs have also been shown to shrink nasal polyps. These benefits, combined with their relatively safe profile, make topical intranasal steroids a reasonable adjunctive therapy. Systemic corticosteroids are also widely used in clinical practice. Recently, a double-blind placebo-controlled trial of prednisolone, 50 mg daily for 14 days versus placebo, demonstrated improvement of sinonasal polyposis as measured by symptom scores, nasal endoscopy, and MRI [44].

The use of antibiotic treatment in chronic sinusitis is quite controversial. Patients with chronic sinusitis may also present with acute bacterial sinusitis, and in these patients antibiotics are indicated. Immunocompromised patients are at higher risk of a chronic infectious process, and may need to be treated with antimicrobial therapy. However, often acute exacerbations may be caused by reasons noninfectious in nature, such as allergic or nonallergic rhinitis. In these cases, treating the underlying disease is more appropriate.

Aspirin sensitivity is often present in patients with nasal polyps. In patients with aspirin-exacerbated respiratory disease (AERD), aspirin desensitization, followed by long term treatment (650 mg twice a day), have demonstrated improvement of clinical outcomes and decrease in the requirement for systemic corticosteroids [45].

Cysteinyl leukotrienes are proinflammatory mediators, and are especially elevated in patients with chronic sinusitis and AERD. Several pharmacologic agents target disruption of this pathway, and are collectively known as leukotriene modifiers. In a placebo controlled study of aspirin intolerant asthmatics, zileuton, one such leukotriene modifier, reduced polyp size and restored the sense of smell [46].

Surgical management may be indicated in cases refractory to medical management. In a randomized controlled study comparing medical versus combined medical and surgical treatment of nasal polyposis, medical treatment alone was often sufficient to treat most symptoms. However, if the primary complaint is nasal obstruction, despite corticosteroid treatment, surgical intervention is indicated [47].

The role of fungus in sinusitis

Two specific cases of fungal sinusitis are worth mention. The first, allergic fungal sinusitis (AFS) is a well known, distinct entity of sinusitis, and is best characterized as the upper airway equivalent to allergic bronchopulmonary aspergillosis. AFS is a noninvasive form of sinusitis, which is characterized by thick mucus, often described as peanut butter-like in consistency. Histologic findings include fungal hyphae and degranulating eosinophils embedded within mucinous material. Most patients also present with peripheral blood eosinophilia, nasal polyposis, and evidence of allergy to fungus (by skin testing or fungal antigen specific IgE). Treatment requires surgical debridement and corticosteroid therapy.

An active controversy in the literature revolves around the role of fungi as a major contributor to the pathogenesis of most chronic sinusitis. Fungi are ubiquitous organisms, and one group has been able to collect and culture fungi in virtually all patients with chronic sinusitis. Surprisingly, a similarly high rate of colonization was found in healthy controls [48]. Therefore, the mere presence of fungi is not sufficient to cause disease. The investigators argue that in a susceptible host, an immunologic response is mounted, including the proliferation or recruitment of eosinophils, which results in the clinical expression of chronic sinusitis. If this were true, then eradication of the fungi should result in improvement in disease course. This has been investigated in several trials with mixed results. In a randomized placebo-controlled double-blind trial, 24 subjects completed 6 months of treatment with intranasal amphotericin B solution versus placebo. The treatment group exhibited both improved CT scores and endoscopy, but no change in symptoms over placebo [49]. In contrast, two European trials have shown no clinical benefit [50,51]. Overall, there is not enough data to routinely justify nasal antifungal therapy, and the authors do not prescribe this in our clinical practice.

Summary

In summary, acute and chronic sinusitis are common diseases and account for a significant number of visits to the primary care office. Both are associated with significant morbidity and consumption of health care dollars. Acute sinusitis is caused by an infectious process and can often be difficult to distinguish from a viral upper respiratory infection, as signs, symptoms, and even the results of most diagnostic tests overlap. Treatment of choice is antibiotic therapy, and adjunctive therapy may or may not add benefit. In contrast, chronic sinusitis is an inflammatory disease. Contrary to common practice, long term antibiotics are likely not useful. Instead, corticosteroids, both in intranasal form and, if necessary, oral systemic form, are more efficacious. In select patients with nasal polyposis and AERD, both leukotriene modifiers and aspirin desensitization may be useful.

References

[1] Ray NF, Baraniuk JN, Thamer M, et al. Health care expenditures for sinusitis in 1996: contributions of asthma, rhinitis, and other airway disorders. J Allergy Clin Immunol 1999;103: 408–14.

[2] Anand VK. Epidemiology and economic impact of rhinosinusitis. Ann Otol Rhinol Laryngol 2004;193(Suppl):S3–5.

[3] Glikilich RE, Metson R. The health impact of chronic sinusitis in patients seeking otolaryngologic care. Otolaryngol Head Neck Surg 1995;113:104–9.

[4] Senior BA, Kennedy DW. Management of sinusitis in the asthmatic patient. Ann Allergy Asthma Immunol 1996;77:6–19.

[5] Gwaltney JM. Acute community-acquired sinusitis. Clin Infect Dis 1996;23:1209–23.

[6] Gwaltney JM Jr, Hendley JO, Simon G, et al. Rhinovirus infections in an industrial population. II. Characteristics of illness and antibody response. JAMA 1967;202:494–500.

[7] Piccirillo JF. Acute bacterial sinusitis. N Engl J Med 2004;351:902–10.

[8] Gwaltney JM Jr, Hayden FG. Psychological stress and the common cold. N Engl J Med 1992;326:644–6.

[9] Anon JB, Jacobs MR, Poole MD, et al. Sinus and Allergy Health Partnership. Antimicrobial treatment guidelines for acute bacterial rhinosinusitis. Otolaryngol Head Neck Surg 2004;130(Suppl 1):S1–45.

[10] Gwaltney JM Jr, Hendley JO, Philips CD, et al. Nose blowing propels nasal fluid into the paranasal sinuses. Clin Infect Dis 2000;30:387–91.

[11] Jones NS, Strobl A, Holland I. A study of the CT findings in 100 patients with rhinosiusitis and 100 controls. Clin Otolaryngol 1997;22:47–51.

[12] Brook I, Yocum P, Shah K. Aerobic and anaerobic bacteriology of concurrent chronic otitis media with effusion and chronic sinusitis in children. Arch Otolaryngol Head Neck Surg 2000;126(2):174–6.

[13] Rontal M, Bernstein JM, Rontal E, et al. Bacteriologic findings from the nose, ethmoid, and bloodstream during endoscopic surgery for chronic rhinosinusitis: implications for antibiotic therapy. Am J Rhinol 1999;13(2):91–6.

[14] Klossek JM, Dubreuil L, Richet H, et al. Bacteriology of chronic purulent secretions in chronic rhinosinuisitis. J Laryngol Otol 1998;112(12):1162–6.

[15] Steinke JW, Borish L. The role of allergy in chronic rhinosinusitis. Immunol Allergy Clin N Am 2004;24:45–57.

[16] Van Zele T, Gevaert P, Watelet JB, et al. *Staphylococcus aureus* colonization and IgE antibody formation to enterotoxins is increased in nasal polyposis. J Allergy Clin Immunol 2004; 114:981–3.

[17] Shin S-H, Ponikau JU, Sherris DA, et al. Rhinosinusitis: an enhanced immune response to ubiquitous airborne fungi. J Allergy Clin Immunol 2004;114:1369–75.

[18] Pozehl D, Moeller P, Riechelmann H, et al. Distinct features of chronic rhinosinusitis with and without nasal polyps. Allergy 2006;61:1275–9.

[19] Borish L. Sinusitis and asthma: entering the realm of evidence-based medicine. J Allergy Clin Immunol 2002;109(4):606–8.

[20] Smart BA. Pediatric Rhinosinusitis and Its Relationship to Asthma and Allergic Rhinitis. Pediatric Asthma, Allergy and Immunology 2005;18:88–98.

[21] Dibaise JK, Huerter JV, Quigley EM. Sinusitis and gastroesophageal reflux disease. Ann Intern Med 1998;129:1078–83.

[22] Williams JW, Simel DL. Does this patient have sinusitis? Diagnosing acute sinusitis by history and physical examination. JAMA 1993;270(10):1242–6.

[23] Lindbaek M, Hjortdahl P. The clinical diagnosis of acute purulent sinusitis in general patience—a review. Br J Gen Pract 2002;52:491–5.

[24] Lacroix JS, Ricchetti A, Lew D, et al. Symptoms and clinical and radiological signs predicting the presence of pathogenic bacteria in acute rhinosinusitis. Acta Otolaryngol 2002;122:192–6.

[25] Loyal V, Jones J, Noyek A. Management of odotogenic maxillary sinus disease. Otolaryngol Clin North Am 1976;9:213–22.
[26] Gwaltney JM Jr, Phillips CD, Miller RD, et al. Computed tomographic study of the common cold. N Engl J Med 1994;330:25–30.
[27] Jones NS. CT of the paranasal sinuses: a review of the correlation with clinical, surgical and histopathological findings. Clin Otolaryngol 2002;27:11–7.
[28] Bhattacharyya T, Piccirillo J, Wippold FJ. Relationship between patient-based descriptions of sinusitis and paranasal sinus computed tomographic findings. Arch Otolaryngol Head Neck Surg 1997;123(11):1189–91.
[29] Mudgil SP, Wise SW, Hopper KD, et al. Correlation between presumed sinusitis-induced pain and paranasal sinus computed tomographic findings. Ann Allergy Asthma Immunol 2002;88:223–6.
[30] Gordts F, Nasser IA, Clement PAR, et al. Bacteriology of the middle meatus in children. Int J Pediatr Otorhinolaryngol 1999;48:163–7.
[31] Gold SM, Tami TA. Role of middle meatus aspiration culture in the diagnosis of chronic sinusitis. Laryngoscope 1997;107:1586–9.
[32] Van Duijn NP, Brouwer HJ, Lamberts H. Use of symptoms and signs to diagnose maxillary sinusitis in general practice: comparison with ultrasonography. BMJ 1992;305: 684–7.
[33] Williams JW Jr, Simel DL, Roerts L, et al. Clinical evaluation for sinusitis. Making the diagnosis by history and physical examination. Ann Intern Med 1992;117(9):705–10.
[34] Hansen JG, Schmidt H, Rosborg J, et al. Predicting acute maxillary sinusitis in a general practice population. BMJ 1995;311(6999):233–6.
[35] Sande MA, Gwaltney JM. Acute community-acquired bacterial sinusitis: continuing challenges and current management. Clin Infect Dis 2004;39:S151–8.
[36] Balk EM, Zucker D, Engels EA, et al. Strategies for diagnosing and treating suspected acute bacterial sinusitis: a cost-effectiveness analysis. J Gen Intern Med 2001;16:701–11.
[37] ten Brinke A, Grootendorst D, Schmidt JT, et al. Chronic sinusitis in severe asthma is related to sputum eosinophilia. J Allergy Clin Immunol 2002;109(4):621–6.
[38] de Ferranti SD, Ioannidis JP, Lau J, et al. Are amoxycillin and folate inhibitors as effective as other antibiotics in acute sinusitis? A meta-analysis. BMJ 1998;317:362–7.
[39] Jacobs MR, Bajaksouzian S, Zilles A, et al. Suceptibilities of *Streptococcus pneumoniae* and *Haemophilus influenzae* to 10 oral antimicrobial agents based o pharmacodynamic parameters: 1997 US surveillance study. Antimicrob Agents Chemother 1999;43:1901–8.
[40] Snow V, Mottur-Pilson C, Hickner JM. Principles of appropriate antibiotic use for acute sinusitis in adults. Ann Intern Med 2001;134:495–7.
[41] Gwaltney JM Jr, Druce HM. Efficacy of brompheniramine maleate for the treatment of rhinovirus colds. Clin Infect Dis 1997;25:1188–94.
[42] Turner RB, Sperber SJ, Sorrentino JV, et al. Effectiveness of clemastine fumarate for treatment of rhinorrhea and sneezing associated with the common cold. Clin Infect Dis 1997;25: 824–30.
[43] Meltzer EO, Charous BL, Busse WW, et al. Added relief in the treatment of acute recurrent sinusitis with adjunctive mometxone furate nasal spray. J Allergy Clin Immunol 2000;106: 630–7.
[44] Hissaria P, Smith W, Wormald P, et al. Short course of systemic corticosteroids in sinuonasal polyposis: a double-blind, randomized, placebo-controlled trial with evaluation of outcome measures. J Allergy Clin Immunol 2006;118(1):128–33.
[45] Stevenson DD, Hankammer MA, Mathison DA, et al. Aspirin desensitization treatment of aspirin sensitive rhinosinusitis—asthmatic patients: long term outcomes. J Allergy Clin Immunol 1996;98:751–8.
[46] Dahlen B, Nizankowska E, Szczeklik A, et al. Benefits from adding the 5-lipoxygenase inhibitor zileuton to conventional therapy in aspirin-tolerant asthmatics. Am J Respir Crit Care Med 1998;157:1187–94.

[47] Blomqvist EH, Lundbald L, Anggard A, et al. A randomized controlled study evaluating medical treatment versus surgical treatment in addition to medical treatment of nasal polyposis. J Allergy Clin Immunol 2001;107(2):224–8.
[48] Potikau JU, Sherris DA, Kern EB, et al. The diagnosis and incidence of allergic fungal sinusitis. Mayo Clin Proc 1999;74:877–84.
[49] Ponikau JU, Sherris D, Weaver A, et al. Treatment of chronic rhinosinusitis with intranasal amphotericin B: a randomized, placebo-controlled, double-blind pilot trial. J Allergy Clin Immunol 2005;115(1):125–31.
[50] Weschta M, Rimek D, Formanek M, et al. Topical antifungal treatment of chronic rhinosinusitis with nasal polyps: a randomized, double-blind clinical trial. J Allergy Clin Immunol 2004;113(6):1122–8.
[51] Ebbens FA, Scadding GK, Badia L, et al. Amphotericin B nasal lavages: not a solution for patients with chronic rhinosinusitis. J Allergy Clin Immunol 2006;118(5):1149–56.