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Headaches due to nasal and paranasal sinus disease

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Sinusitis

Sinus infections are much less common today than they were in the preantibiotic era, but they still are overdiagnosed. Acute sinusitis, a relatively uncommon cause of headache, is the result of infection of one or more of the cranial sinuses (Fig. 1). Acute sinusitis usually is characterized by purulent discharge in the nasal passages and a pain profile determined by the site of infection. Sinusitis is overdiagnosed as a cause of headache because of the belief that pain over the sinuses must be related to the sinuses. In fact, frontal head pain more often is caused by migraine and tension-type headache. It should not follow that if a patient fails to respond to treatment for migraine and tension-type headache one should reconsider the diagnosis of sinus disease. Whether or not nasal obstruction can lead to chronic headache is controversial [1]. Paradoxically, sinus disease also tends to be underdiagnosed, as sphenoid sinus infection frequently is missed [2].

Because sphenoid sinusitis differs from other forms of sinusitis in clinical features and treatment, it is considered separately in this article. Although it represents only 3% of sinusitis cases, its importance is out of proportion to its prevalence because it is potentially life threatening.

Sinusitis, which affects more than 31 million people in the United States, resulted in 16 million physician visits in 1985 [3]. By 1994, the National Health Interview Survey estimated that 35 million people were affected [4]. A National Health Interview Survey conducted in the United States between 1990 and 1992 found that chronic sinusitis was the second most frequent disease after orthopedic deformities, with an annual average of 33.1 million cases [5]. The prevalence of acute sinusitis is increasing, according to data from the National Ambulatory Medical Care Survey, up from 0.2% of diagnoses at office visits in 1990 to 0.4% of diagnoses at office visits in 1995

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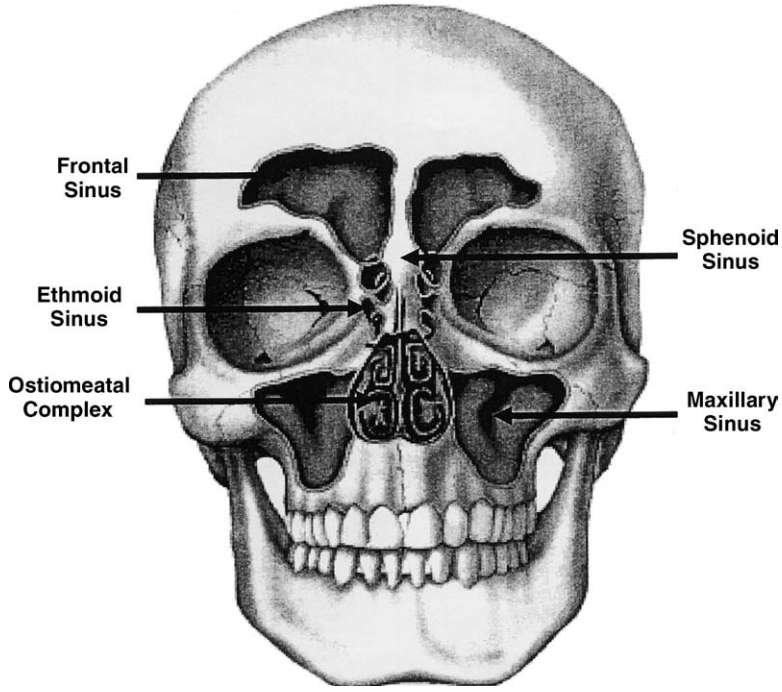

Fig. 1. Diagram illustrating location of cranial sinuses.

[4]. Approximately 0.5% of upper respiratory infections in adults are complicated by sinusitis [6]. As many as 38% of patients who have symptoms of sinusitis in adult general medicine clinics may have acute bacterial rhinosinusitis. In otolaryngology practices, the prevalence was higher (50% to 80%). Although sinusitis generally is more common in children than adults, frontal and sphenoid sinusitis are rare in children. In the primary care setting, between 6% and 18% of children presenting with upper respiratory infections may have acute bacterial sinusitis [4]. In the preantibiotic era, the sphenoid sinus was involved in as many as 33% of cases of sinusitis. Its incidence today is approximately 3% [2].

The maxillary and ethmoid sinuses, both present at birth, are the most common sites of clinical infection in children. The sphenoid sinus develops after the age of 2 years and starts to pneumatize at the age of 8 years. The frontal sinuses develop from the anterior ethmoid sinus at approximately 6 years of age. The frontal and sphenoid sinuses become clinically important in the teenage years, and they frequently become infected in pansinusitis. Isolated sphenoid sinusitis is rare [7,8]. The clinical diagnosis of sinusitis usually is based on symptoms that suggest maxillary or frontal sinus involvement. Ethmoid sinusitis frequently is a cause of frontal and maxillary sinusitis [9,10]. Obstruction of the ostiomeatal complex, the common

drainage pathway for the ethmoid, frontal, and maxillary sinuses, is the usual precursor to sinus disease [11].

Anatomy and physiology

The ethmoid bone, a T-shaped structure that supports the bilateral ethmoid labyrinth, forms the lateral nasal wall. The horizontal limb of the T is formed by the cribriform plate, from which the ethmoid labyrinth is suspended. This is a complex structure that has multiple bony septa and the medial projections of the superior and middle turbinates. Lateral to the uncinate process, which is a secondary projection of the ethmoid bone, is the infundibulum, a recess into which the maxillary sinus drains. The infundibulum drains into the hiatus semilunaris, which in turn drains into the middle meatus, which is located between the uncinate process and the middle turbinate. The frontal sinus drains into the frontal recess, which may drain into either the middle meatus or the ethmoidal infundibulum. This region is known as the ostiomeatal complex [11] (maxillary sinus ostium, infundibulum, hiatus semilunaris, middle turbinate, ethmoidal bulla, and frontal ostium). The sphenoidal sinus and posterior ethmoidal cells drain into the sphenoethmoidal recess.

The primary functions of the nasal passages are to warm, humidify, and filter inspired air. The paranasal sinuses are air-filled cavities that connect with the nasal airway. They are lined with pseudostratified ciliated epithelial tissue, which is covered by a thin layer of mucus. Large inhaled particulate matter passes over this constantly moving ciliated epithelial layer and is deposited there. The cilia and the mucous layer are in constant motion in a predetermined direction. Mucus and debris are transported toward the ostia by the beating of the cilia and are expelled into the nasal airway [7,11,12]. Any bacterial contamination of the sinuses is effectively cleared by this mechanism. If the sinus ostia are obstructed, mucociliary flow is interrupted. Obstruction causes the oxygen tension within the sinus to decrease and the carbon dioxide tension to increase. This anaerobic, high-carbon dioxide, stagnant environment can facilitate bacterial growth [11].

For many years, surgical drainage of the sinuses, avoiding the region of the natural ostia, was the treatment of choice for sinus infections. This procedure alleviated the acute sinus infection but did not prevent re-accumulation of mucus within the sinus. Because the normal beat of the cilia transports mucus toward the natural ostium, surgically creating a new ostium at a site distant from the natural ostium, thus fails to direct the flow of mucus to the new opening [11].

All sinuses normally contain anaerobic bacteria, and more than one third harbor a mixed environment of aerobic and anaerobic organisms. Ciliary dysfunction and retention of secretions that are the result of ostial obstruction can result in bacterial proliferation and sinus infection. Aerobes that are present in normal and disease states include the Gram-positive

streptococci (α, β, and *Streptococcus pneumoniae*) and *Staphylococcus aureus* and Gram-negative *Moraxella catarrhalis*, *Haemophilus influenzae*, and *Escherichia coli*. Anaerobic organisms include the Gram-positive peptococci and propionibacterium species. The bacteroides and fusobacterium species also play a role in chronic sinusitis [7,13].

Systemic diseases that predispose to sinusitis include cystic fibrosis, immune deficiency, bronchiectasis, and the immobile cilia syndrome. Local factors include upper respiratory infections (usually viral), allergic rhinitis, overuse of topical decongestants, hypertrophied adenoids, deviated nasal septum, nasal polyps, tumors, and cigarette smoke [7]. The most common predisposing factor is mucosal inflammation from viral upper respiratory infection or allergic rhinitis [6]. The sinuses are involved in nearly 90% of viral upper respiratory infections. Eighty-seven percent of patients who have a common cold and no previous history of rhinosinusitis have maxillary sinus abnormalities, 65% ethmoid sinus abnormalities, and 30% to 40% frontal or sphenoid sinus abnormalities on CT. The abnormalities are most likely the result of highly viscid secretions in the sinuses. In 77% of patients, the infundibulum is occluded. In most patients these abnormalities resolve spontaneously, but some develop secondary bacterial infections [14]. Foreign bodies are a common cause of obstruction in children, and 10% of sinus infections have a dental origin [6]. Loss of immunocompetence related to HIV infection, chemotherapy, posttransplant immunosuppression, insulin-dependent diabetes mellitus, or some connective tissue disorders predisposes patients to rhinosinusitis and increases the likelihood of its persistence. Rhinosinusitis is common in the ICU, because prolonged supine positioning compromises mucociliary clearance and adds to the problems created by mucosal drying from transnasal supplemental oxygenation and sinus ostial obstruction from nasotracheal or nasogastric tubes. Rhinosinusitis occurs in 95.5% of bedridden ICU patients who have a nasogastric or nasotracheal tube in place for at least a week [15]. Unobstructed flow through the sinus ostia and its narrow communicating passage within the ostiomeatal complex is integral to mucociliary clearance and ventilation. Persistent low-grade inflammation in the ethmoid sinus may cause few localizing symptoms but can predispose to recurrent maxillary and frontal sinus infections [7,11].

Diagnostic testing

The physical examination may not be helpful, particularly in sphenoid sinusitis. Not all patients are febrile, and sinus tenderness is not always present. Pus is not always seen in sphenoid sinusitis. Kibblewhite and associates found purulent exudate in only 3 of 14 patients [16]. Transillumination of the sinuses has low sensitivity and specificity [17], and routine anterior rhinoscopy performed with a headlight and nasal speculum allows only limited inspection of the anterior nasal cavity.

Standard radiography

Standard radiography is inadequate for the clinical evaluation of sinusitis because it does not evaluate the anterior ethmoid air cells, the upper two thirds of the nasal cavity, or the infundibular, middle meatus, or frontal recess air passages [12].

Neuroimaging

CT is the optimal radiographic study to assess the paranasal sinuses for evidence of disease. The mucosa of the normal, noninfected sinus approximates the bone so closely that it cannot be visualized on CT. Therefore, any soft tissue seen within a sinus is abnormal [18]. CT may demonstrate mucosal thickening, sclerosis, clouding, or air-fluid levels. Imaging must be performed in the coronal plane to adequately demonstrate the ethmoid complex. It can reveal the extent of mucosal disease in the ostiomeatal complex. The test-retest reliability of CT in the assessment of chronic rhinosinusitis was high and stable in a prospective series of patients scheduled for endoscopic sinus surgery [19]. The prevalence of reversible sinus abnormalities on CT in patients who have the common cold is high [14]. This suggests that CT may not be specific for bacterial infections [6]. Middle meatus involvement was present in 72 of 100 CT examinations of patients who had chronic sinusitis. Anterior ethmoid sinus infection was found in every patient who had frontal or maxillary sinusitis. Middle meatal disease was found in the rest of these patients; it extended to, and occluded, the frontal recess in the patients who had frontal sinusitis and extended to, and occluded, the infundibulum in all cases of maxillary mucoperiosteal disease [3].

Incidental anatomic abnormalities within the paranasal sinuses are common. Incidental anatomic abnormalities on CT scans occur in 27% to 45% of asymptomatic individuals [20]. Patients undergoing endoscopic sinus surgery for chronic rhinosinusitis were evaluated with CT and staged according to the Lund system. (Each paranasal sinus [anterior ethmoid, posterior ethmoid, maxillary, frontal, and sphenoid sinus for each side] was given a score of 0 for no opacification, 1 for partial opacification, or 2 for total opacification.) The ostiomeatal complex was assigned a score of 0 for patent or 2 for obstructed. The Lund score ranged from 0 to 24. Controls were patients undergoing sinus CT for other reasons. In the disease-positive group of patients who had chronic rhinosinusitis, the mean Lund score was 9.8 (9.0–10.6). The mean Lund score of the control group (without disease) was 4.3 (3.5–5.0). The AUC for the receiver-operator characteristic was 0.802 ($P < .001$). Using a Lund score cut-off value of greater than 2 as abnormal, the sinus CT exhibited sensitivity and specificity of 94% and 41%, respectively. Increasing the cut-off value to 4 changed the sensitivity and specificity to 85% and 59%, respectively [20].

Sinus CT scan discriminates with good sensitivity and moderate specificity between patients who do and do not have chronic rhinosinusitis.

The positive and negative predictive values depend on the a priori prevalence of chronic rhinosinusitis. Lund scores of 0 or 1 are unlikely to represent true chronic rhinosinusitis, whereas Lund scores of 4 or greater are highly likely to represent true chronic rhinosinusitis. Lund scores of 2 to 3 are ambiguous, and further clinical evaluation or follow-up is warranted [20].

During the edematous phase of the nasal cycle, normal nasal mucosa on T2-weighted image can resemble pathologic change. Despite these specificity problems, MRI is more sensitive than CT in detecting fungal infection [12]. Maxillary mucosal thickening >6 mm, complete sinus opacification, and air-fluid levels on neuroimaging correlate to positive sinus cultures [21]. Thirty percent to 40% of the normal population, however, has mucosal thickening on CT evaluation [22]. The 1999 Agency for Healthcare Policy and Research (AHCPR) meta-analysis of six studies showed that sinus radiography has moderate sensitivity (76%) and specificity (79%) compared with sinus puncture in the diagnosis of acute bacterial rhinosinusitis. CT or MRI is necessary to definitively diagnose sphenoid sinusitis, because plain radiographs are nondiagnostic in approximately 26% of cases [20]. CT scanning is the gold standard for the diagnosis of sphenoid sinus disease; MRI is an adjunct.

Transillumination, ultrasonography, and anterior rhinoscopy

Transillumination of the sinuses has low sensitivity and specificity [17]. Ultrasonography has lower sensitivity and specificity than sinus radiography [17]. Routine anterior rhinoscopy performed with a headlight and nasal speculum allows only limited inspection of the anterior nasal cavity.

Diagnostic fiberoptic endoscopy

The flexible fiberoptic rhinoscope allows direct visualization of the nasal passages and sinus drainage areas (ostiomeatal complex) and is complementary to CT or MRI. A trained operator can perform this procedure easily, and the patient tolerates it well. Infection is easily diagnosed if purulent material is seen emanating from the sinus drainage region. Mucosal sinus thickening frequently is present in normal, nonsymptomatic patients. In these cases, endoscopy should be positive before a diagnosis of sinusitis is made [11,13]. Sphenoid sinusitis is an exception to this generalization.

Endoscopy should be considered when a patient who is suspected of having a sinus-related problem fails conservative medical treatment and has an inconclusive CT or MRI. Some physicians use endoscopy before neuroimaging. Negative neuroimaging and endoscopy usually, but not always, rules out sinus disease [12].

Castellanos and Axelrod [23] evaluated 246 patients who had undiagnosed headache and a negative neurologic evaluation. Ninety-eight had only

rhinoscopic evidence of sinusitis, 84 had both rhinoscopic and standard radiographic evidence of sinusitis, and 64 had neither. Patients were treated with antibiotics for 4 weeks, at which time only those patients who had rhinoscopic or radiologic evidence of sinusitis reported headache improvement. This was an open, uncontrolled study, but repeat rhinoscopic evaluation showed clearing of infection coincident with headache improvement.

Clinical findings

In 1996, the American Academy of Otolaryngology-Head and Neck Surgery standardized the terminology for paranasal infections [24]. The term rhinosinusitis was believed more appropriate than sinusitis because rhinitis typically precedes sinusitis, purulent sinusitis without rhinitis is rare, the mucosa of the nose and sinuses are contiguous, and symptoms of nasal obstruction and discharge are prominent in sinusitis [25]. The diagnosis of rhinosinusitis usually is based on symptoms indicating maxillary or frontal sinus involvement. This may occur secondary to, and is frequently a result of, ethmoid disease. Obstruction of the sinus ostia is the usual precursor of sinusitis [12,26].

Rhinosinusitis is divided into four categories based on the temporal course and the signs and symptoms of the disease (Table 1): (1) acute rhinosinusitis is sudden in onset; it lasts from 1 day to 4 weeks and there is complete resolution of the symptoms; (2) recurrent acute rhinosinusitis requires four or more episodes of acute rhinosinusitis, lasting at least 7 days each, in any 1-year period; (3) subacute rhinosinusitis is continuous with acute rhinosinusitis and lasts from 4 to 12 weeks [27]; and (4) chronic rhinosinusitis requires that signs or symptoms persist for 12 weeks or longer and may be punctuated by acute infectious episodes.

Most cases of infectious rhinosinusitis that last less than 7 days are viral. Acute bacterial sinusitis in adults most often presents with $\geq$7 days of purulent anterior rhinorrhea, nasal congestion, postnasal drip, facial or dental pain/pressure, and cough, frequently with a nighttime component (Box 1).

Facial tenderness and pain, nasal congestion, and purulent nasal discharge are common manifestations of acute sinus infection. Other ''classic'' signs and symptoms include anosmia, pain upon mastication, and halitosis. An upper respiratory infection or a history of one may be present [17]. Although fever is present in approximately 50% of adults and 60% of children and headache is common, the symptoms of headache, facial pain, and fever often are of minimal value in the diagnosis of sinusitis. Williams et al [28] looked at the sensitivity and specificity of individual symptoms in making the diagnosis of sinusitis. No single item was both sensitive and specific. Maxillary toothache was highly specific (93%), but only 11% of the patients had this symptom. Logistic regression analysis showed five independent predictions of sinusitis: maxillary toothache (odds ratio [OR] 2.9), abnormal transillumination (OR 2.7, sensitivity 73%, specificity 54%),

Table 1
Classification of adult rhinosinusitis

Classification	Duration	Strong history	Include in differential	Special Notes
Acute	≤4 weeks	≥2 major factors, 1 major factor and 2 minor factors, or nasal purulence on examination	1 major factor or ≥2 minor factors	Fever or facial pain does not constitute a suggestive history in the absence of other nasal signs or symptoms; consider acute bacterial rhinosinusitis if symptoms worsen after 5 days, persist for > 10 days, or are out of proportion to those typically associated with viral infection
Subacute	4–12 weeks	Same as chronic	Same as chronic	Complete resolution after effective medical therapy
Recurrent acute	≥4 episodes per year, with each episode lasting ≥7–10 days and no intervening signs and symptoms of chronic rhinosinusitis	Same as acute		
Chronic	≥12 weeks	≥2 major factors, 1 major factor and 2 minor factors, or nasal purulence on examination	1 major factor or ≥2 minor factors	Facial pain does not constitute a suggestive history in the absence of other nasal signs or symptoms
Acute exacerbations of chronic	Sudden worsening of chronic rhinosinusitis, with return to baseline after treatment			

Adapted from Lanza DC, Kennedy DW. Adult rhinosinusitis defined. Otolaryngol Head Neck Surg 1997;117:S1–7; with permission.

poor response to decongestants (OR 2.4), purulent discharge (OR 2.9), and colored nasal discharge (OR 2.2). The data did not support the other textbook findings for sinusitis (an antecedent upper respiratory infection or history of facial pain). ''Headache'' had an odds ratio of 1.0, with 68% sensitivity and 30% specificity. The low specificity is because of the lack of descriptive features of the headache. Facial pain and itchy eyes had an OR of 1.0. Fever, sweats, or chills were found in 48% of patients, with an OR of 0.9 (sensitivity 45%, specificity 51%). It has been suggested that highly specific symptoms, such as facial erythema or maxillary toothache, or

Box 1. Factors associated with the diagnosis of chronic rhinosinusitis

Major factors
- Facial pain/pressure*
- Facial congestion/fullness
- Nasal obstruction/blockage
- Nasal discharge/purulence/discolored postnasal discharge drainage
- Hyposmia/anosmia
- Purulence in nasal cavity on examination
- Fever (acute rhinosinusitis only)**

Minor factors
- Headache
- Fever (all nonacute)
- Halitosis
- Fatigue
- Dental pain
- Cough
- Ear pain/pressure/fullness

* Facial pain/pressure alone does not constitute a suggestive history for rhinosinusitis in the absence of another major nasal symptom or sign.

** Fever in acute sinusitis alone does not constitute a strongly suggestive history for acute sinusitis in the absence of another major nasal symptom or sign.

Adapted from Lanza DC, Kennedy DW. Adult rhinosinusitis defined. Otolaryngol Head Neck Surg 1997;117:S1–7.

symptoms that persist for more than 10 days warrant a diagnosis and treatment [29]. The AHCPR report [4], based on limited evidence, suggested that diagnostic accuracy may be similar to that of sinus radiography when three or four of the following symptoms are present: purulent rhinorrhea with unilateral predominance, local pain with unilateral predominance, bilateral purulent rhinorrhea, and pus in the nasal cavity.

Naranch et al [30] compared sinus and systemic tenderness in rhinosinusitis with other disorders. Cutaneous pressures (kg/cm(2)) causing pain at 5 sinus and 18 systemic sites were measured. Lower sinus thresholds were found in the rhinosinusitis groups. Sinus and systemic thresholds were 44% lower in chronic fatigue syndrome subjects than in nonchronic fatigue syndrome subjects, suggesting that systemic hyperalgesia contributes to chronic fatigue syndrome, sinus tenderness, and rhinosinusitis complaints.

Children with acute and chronic sinusitis almost always present with purulent nasal discharge and cough, which are not characteristic in adults. Fever is infrequent, even with acute sinusitis, and usually is associated with complicated acute sinusitis [31].

Sinus infection can result in acute suppurative meningitis, subdural or epidural abscess, and brain abscess. In addition, osteomyelitis and subperiosteal abscess can occur. Infection of the ethmoid, and, to a lesser extent, the sphenoid sinuses, is responsible for orbital complications, including edema, orbital cellulitis, and subperiosteal and orbital abscess [7]. A mucocele is a mucus-containing cyst located in the sinuses. These are most common (and benign) in the maxillary sinus (mucus retention cyst). Those located in the frontal, sphenoid, or ethmoid sinus can enlarge and erode into the surrounding structures. A pyocele is an infected mucocele [8,32].

Wolff [33] showed that the sinuses themselves are relatively insensitive to pain. The pain associated with sinusitis comes from engorged and inflamed nasal structures: nasofrontal ducts, turbinates, ostia, and superior nasal spaces. Headache associated with paranasal sinus disease usually has a deeper, dull, aching quality combined with a heaviness and fullness. It seldom is associated with nausea and vomiting.

The IHS has established new criteria for acute sinus headache (Box 2) [34]. To qualify as rhinosinusitis headache, all must be present. These criteria may not be valid for sphenoid sinusitis, however, as purulent discharge often is lacking, and headache may precede sinus drainage. Once drainage begins, obstruction is relieved and the headache may begin to abate.

All sinusitis pain is not the same. Maxillary sinusitis pain most typically is located in the cheek, the gums, and the teeth of the upper jaw. Ethmoid sinusitis pain is felt between the eyes. The eyeball may be tender and pain may be aggravated by eye movement. Frontal sinusitis pain is felt mainly in the forehead. Sphenoid sinusitis pain is felt in the vertex, but has a more general localization. Ethmoid and maxillary sinusitis usually is associated with rhinitis.

Hypertrophic turbinates, atrophic sinus membranes, and nasal passage abnormalities resulting from septal deflection are other conditions that may cause headache; however, they are not sufficiently validated as a cause of headache. Migraine and tension-type headache often are confused with true

Box 2. Rhinosinusitis headache

- Diagnostic criteria: pain in one or more regions of the head, face, ears, or teeth
- Clinical, laboratory, or imaging evidence of an acute rhinosinusitis (eg, purulence in the nasal cavity, nasal obstruction, fever, hypo-osmia/anosmia, CT, MRI, or fiberoptioc nasal endoscopy findings)
- Simultaneous onset of headache and rhinosinusitis
- Headache lasts <7 days after remission or successful treatment of acute rhinosinusitis

sinus headache because of similarity in location. To diagnose IHS sinus headache, the previously defined criteria must be fulfilled strictly.

Differential diagnosis

Although hypertrophic turbinates, atrophic sinus membranes, and nasal passage abnormalities caused by septal deflection may cause headache, these causes have not been validated by the IHS. Whether or not nasal obstruction can lead to chronic headache is controversial [1]. Migraine and tension-type headache often are confused with true sinus headache because of their similar locations. Some patients, in addition to having all the features of migraine without aura, have head pain in the facial areas, associated congestion of the nose, and headache triggered by weather changes. None of these patients has purulent nasal discharge or the other abnormalities seen in acute rhinosinusitis. It is necessary, therefore, to differentiate headaches caused by rhinosinusitis from so-called ''sinus headaches,'' which are headache attacks fulfilling the criteria of migraine without aura with prominent autonomic symptoms in the nose or of migraine without aura triggered by nasal changes.

In a population-based headache study [35] of 23,564 subjects, 4967 individuals called their headache migraine and 3074 individuals had headache that met IHS migraine criteria. Among those with IHS migraine, only 53.4% recognized their headaches as migraine; stress headaches ($n = 345$) and sinus headaches ($n = 365$) were the most common erroneous labels reported. People are confused by their headache location. Because the sinuses are close to the eyes, individuals may attribute headaches located in the frontal, supraorbital, or infraorbital region to the sinuses.

In a clinic-based study, headache symptoms, headache-associated disability, and response to therapy among patients who had self-described sinus headache were assessed [36]. Patients had to have self-described sinus headaches and at least one migraine symptom: moderate to severe pain, nausea or vomiting, photophobia or phonophobia, unilateral pain, pain worsening with activity, or pulsating pain. They were excluded if they had a previous migraine diagnosis or exposure to triptans, headaches associated with fever or purulent nasal discharge, or radiographic evidence of a sinus infection.

A selected group of patients who had self-described sinus headache had IHS migraine (70%) or migrainous (28%) headache. Most had nasal symptoms, including stuffiness (74%) and runny nose, and 45% said their headaches were precipitated by changes in the weather. Thus, patients who believe they have sinus headache, have no signs or symptoms of rhinosinusitis, and have one symptom of migraine, have migraine.

The relationship between headache and subacute and chronic sinus disease is highly controversial. Radiographic evidence of sinus disease is common and does not establish the headache's etiology [22]. Headache associated with sinus disease usually is continuous, not intermittent.

Chronic sinusitis frequently is associated with engorged and swollen nasal mucosa and a purulent or sanguinopurulent nasal discharge. The IHS has not validated chronic sinusitis as a cause of headache or facial pain unless it relapses into an acute stage [37].

Faleck et al [38] reported that 10% of 150 children and adolescents who presented with chronic, nonprogressive headache, clinically indistinguishable from "muscle contraction" headache, had radiographic evidence of sinus pathology. None had prominent respiratory symptoms. All improved with treatment directed toward the sinus pathology. Although some had complete sinus opacification, none had endoscopy performed to show active disease in the ostia.

Treatment

The following are management goals for the treatment of sinusitis:

- Treat the bacterial infection
- Reduce ostial swelling
- Drain the sinuses
- Maintain sinus ostia patency.

Rhinitis and sinusitis may be difficult to distinguish from each other on the basis of history alone. Most acute upper respiratory infections are viral and do not require antibiotic treatment. Symptoms that persist for $\geq$7 days make acute bacterial sinusitis more likely and the use of antibiotics appropriate. Chronic sinusitis may have an infectious or noninfectious basis. Underlying disorders that predispose to chronic sinusitis should be identified and treated as part of the treatment of chronic sinusitis [39].

Uncomplicated sinusitis, other than sphenoid sinusitis, should be treated with a broad-spectrum oral antibiotic for 10 to 14 days. Because nasal culture does not correlate to sinus pathogens, initial treatment is empiric [17]. Steam and saline prevent crusting of secretions in the nasal cavity and facilitate mucociliary clearance. Locally active vasoconstrictor agents provide symptomatic relief by shrinking inflamed and swollen nasal mucosa. Their use should be limited to 3 to 4 days to prevent rebound vasodilation. Oral decongestants should be used if prolonged treatment (>3 days) is necessary. These agents are α-adrenergic agonists that reduce nasal blood flow without the risk of rebound vasodilation [17].

Antihistamines are not effective in the management of acute rhinitis. Anti-inflammatory topical corticosteroids may help maintain ostial patency. Adding fluticasone to xylometazoline and antimicrobial therapy with cefuroxime improves clinical success rates and accelerates the recovery of patients who have a history of chronic rhinitis or recurrent sinusitis who present for treatment of acute rhinosinusitis [40]. Treatment failure and recurrent infections are indications for neuroimaging and endoscopy to search for a source of obstruction. Sinus sampling for culture should be

considered. Endoscopic nasal surgery may be necessary to reopen and maintain the patency of the sinus ostia and ostiomeatal complex [17].

Complications should be treated with high doses of intravenous antibiotics and surgical drainage, if appropriate, of any enclosed space.

Sphenoid sinusitis

Sphenoid sinusitis, because of its rarity, unique location, and complications, is discussed separately. It is an uncommon infection that accounts for approximately 3% of all cases of acute sinusitis. It usually is accompanied by pansinusitis; it occurs alone less commonly. It frequently is misdiagnosed [41], because the sphenoid sinus is not visualized adequately with routine sinus radiographs and is not accessible to direct clinical examination, even with the flexible endoscope. Although sphenoid sinusitis is an uncommon cause of headache, it is potentially associated with significant morbidity and mortality and requires early identification and aggressive management [2,16,41].

The sphenoid sinus is contained within the body of the sphenoid bone deep in the nasal cavity and is divided in half by the intersphenoid septum. Each sinus communicates with the sphenoethmoidal recess, located at the posterior superior aspect of the superior concha. The sphenoidal sinuses are present as minute cavities at birth and their main development does not occur until puberty [42].

The roof of the sphenoid sinus is related to the middle cranial fossa and the pituitary gland in the sella turcica: lateral is the cavernous sinus; posterior is the clivus and pons; anterior are the posterior nasal cavity, posterior ethmoid cells, and cribriform plate; and inferior is the nasopharynx. The cavernous sinus contains the internal carotid arteries and the third, fourth, fifth, and seventh cranial nerves. The maxillary division of the fifth nerve may indent the wall of the sphenoid sinus. The sphenoid walls can be extremely thin, and sometimes the sinus cavity is separated from the adjacent structure by just a thin mucosal barrier. Because of the close proximity to the cortical venous system, cranial nerves, and meninges, infection may spread to these structures and present as a central nervous system infection or neurologic catastrophe [2,43].

Symptoms

Headache is the most common symptom of acute sphenoid sinusitis: it is present in all patients who are able to complain about it. The headache is aggravated by standing, walking, bending, or coughing. It often interferes with sleep and is poorly relieved by opioids. Its location is variable: vertex headache is rare; frontal, occipital, or temporal headache, or a combination of these locations is most common.

Periorbital pain is common. This is in contrast to the common teaching that retro-orbital or vertex headache is the most common presenting

symptom of sphenoid sinusitis [2,16,41,44–46]. Nausea and vomiting occur frequently, but nasal discharge, stuffiness, and postnasal drip are unusual. Fever occurs in more than half of patients who have acute sphenoid sinusitis.

Diagnosis

The diagnosis of sphenoid sinusitis frequently is delayed. Sphenoid sinusitis should be included in the differential diagnosis of acute or subacute headache. It may be mistaken for frontal or ethmoid sinusitis, aseptic meningitis, brain abscess, or septic thrombophlebitis. It can mimic trigeminal neuralgia, migraine, carotid artery aneurysm, or brain tumor [2,16,41].

A severe, intractable, new-onset headache that interferes with sleep and is not relieved by simple analgesics should alert one to the diagnosis of sphenoid sinusitis. The headache increases in severity and has no specific location. Pain or paraesthesias in the facial distribution of the fifth nerve and photophobia or eye tearing are suggestive of sphenoid sinusitis [2,16,41,44,45,47].

The physical examination may not be helpful. Not all patients are febrile, sinus tenderness rarely is present, and pus is not always seen, although Lew et al [2] state that a careful examination of the nose and throat often demonstrates pus. Whether or not this reflects advanced disease or the presence of pansinusitis is uncertain. In a more recent series of 14 patients who had acute sphenoid sinusitis, Kibblewhite et al [16] found purulent exudate in only 3 patients.

Neuroimaging is necessary to diagnose sphenoid sinusitis definitively. All of Kibblewhite et al's cases were diagnosed by radiograph. [16] Some cases can be diagnosed by plain sinus radiographs, but, because of the superimposition of soft tissues, plain radiographs are nondiagnostic in approximately 25% of cases. [41] If sphenoid sinusitis is suspected and plain radiographs are nondiagnostic, CT or MRI is indicated (Fig. 14.3).

In a high-risk group of 300 patients referred with a clinical diagnosis of sinusitis, 68% had abnormal plain radiographs, but none had sphenoid sinus abnormalities, suggesting that the specificity of plain radiographs is very high [48]. The mucosa of the sinus approximates the bone so closely that it cannot be visualized on CT. Therefore, any soft tissue bulge seen in the sinus is abnormal. [18] Digre et al [49] reviewed 300 CT or MRI radiographic studies. The sphenoid sinus was visualized in all cases. Abnormalities were detected in 7% of routine CT scans, 8% of posterior fossa scans, and 6% of MRI scans. Of the 21 patients who had sphenoid abnormalities, 24% in their highly selected sample had important clinical related disease.

Complications

Major complications of sphenoid sinusitis include bacterial meningitis, [2] cavernous sinus thrombosis [2,16,41,43], subdural abscess [2,16,41], cortical

vein thrombosis [2,16,41], ophthalmoplegia, and pituitary insufficiency [2,16,41]. Sphenoid sinusitis can present as aseptic meningitis because of the presence of a parameningeal focus [50]. Patients can present with the complications of sphenoid sinusitis, including visual loss mimicking optic neuritis, multiple cranial nerve palsies, or papilledema. Sudden onset, as a result of cavernous sinus thrombosis, can mimic a subarachnoid hemorrhage [51].

Øktedalen and Lilleås [52] reported four patients admitted to an infectious disease department with meningitis, sepsis, and orbital cellulitis. Diagnosis was difficult in all cases. All four patients had fever and headache. Three of the four had normal plain sinus radiographs. CT scan diagnosed all cases. Six of Lew et al's [2] 15 acute cases had meningitis, 5 had cavernous sinus thrombosis, 1 had cortical vein thrombosis, 1 had unilateral ophthalmoplegia, and 1 had orbital cellulitis. Eight of Kibblewhite et al's [16] 14 patients had complications on admission. None of Goldman's [41] patients had complications. The difference in the complication rate is a result of selection bias: Goldman et al's [41] patients were retrieved from emergency room records, Lew et al's [2], Øktedalen and Lilleås' [52], and Kibblewhite et al's [16] from inpatient records.

Treatment

Sphenoid sinusitis without complications may be managed with high-dose intravenous antibiotics and topical and systemic decongestants for 10 to 14 days [16,41]. If the fever (if present) and the headache do not start to improve in 24 to 48 hours, or if any complications are present or develop, sphenoid sinus drainage is indicated [41].

Nasal headache

Many rhinologists hold the controversial belief that septal deformation, especially of traumatic origin, may exert pressure on the sensitive structure of the lateral nasal wall, causing referred pain and chronic headache. McAuliffe et al [53] studied the sensitivity of the nasal cavities and paranasal sinuses using touch, pressure, and faradic stimulation. The nasal turbinates and sinus ostia were much more sensitive than the mucosal lining of the septum and the paranasal sinuses. Most of the pain elicited was referred pain. It was of increased intensity, longer duration, and referred to larger areas in subjects who had swelling and engorgement of the nasal turbinates and the sinus ostia.

Schønsted-Madsen et al [1] followed up 444 patients who had nasal obstruction, 157 of whom had headache. Treatment consisted of septoplastic surgery, reconstruction of the nasal pyramids, or submucosal conchotomy. The headache usually was localized to the forehead, glabella, or above and around the eyes. Thirty-six patients had constant, 48 daily, 56

weekly, and 17 monthly headache. Fifty-seven patients had mild headache, 66 had moderate headache, and 34 had severe headache. Many of these patients misused analgesics. Eighty percent of the patients who underwent surgery were relieved of nasal obstruction (the primary reason for surgery), and 60% of the patients who underwent surgery were relieved of chronic headache. If the surgery relieved the nasal obstruction, 80% had headache relief; however, if the surgery failed, only 30% had headache relief.

Clerico reported 10 patients who had intractable migraine, tension-type, or cluster headache without significant nasal or sinus symptoms. Various intranasal and sinus abnormalities, such as anatomic variation or subclinical inflammation, were found on CT or nasal endoscopy. The patients were treated medically or surgically and all improved [54]. Low and Willatt reported 106 patients who had a submucous resection for a deviated nasal septum. Almost half (47.4%) had recurring headaches preoperatively. Postoperatively, 63.6% had complete or partial relief at follow-up for as long as 18 months. Although 79.3% of patients had headache relief when evaluated before 1 year, only 46.2% had relief after 1 year [55].

These studies do not account for the historical relationship between headache onset and the development of nasal obstruction or for the analgesic or decongestant overuse that may produce daily headache. In addition, any surgical procedure has a powerful placebo effect. The studies do suggest that some patients who have nasal obstruction have headache that is relieved by successful medical or surgical treatment. Because migraine prevalence in the population is approximately 12%, episodic tension-type headache prevalence approximately 90%, and chronic tension-type headache prevalence approximately 3%, these data are difficult to interpret. In addition, these studies had no control group and only responders were reported in Clerico's study. In controlled trials of medication, the placebo effect can be quite large.

In a retrospective review of operative notes of 170 patients who underwent functional endoscopic sinus surgery, 50 patients (29%) who had a history of chronic headaches were identified. Thirty-seven met the predetermined inclusion criteria for this study, which were (1) a history of chronic headaches, (2) rhinologic cause for these headaches suggested by the presence of contact points (documented by nasal endoscopy or CT scans), (3) no other origin or cause of headaches after a thorough evaluation, and (4) surgical intervention that included relief of contact points by inferior, middle, or superior turbinoplasty. After surgery, 29 of the 34 patients (85%) in the study group reported a decrease in headache frequency. There were many patients who had severe contact points on CT scan, however, and did not complain of headaches. In fact, most patients who had headaches and contact points also had concurrent chronic sinusitis, which served as the primary indication for surgery in this patient population [56].

Other open studies [57,58] and a review [59] suggest that headache can be the only clinical presentation of sinus or nasal pathology. These studies do not use IHS criteria for headache or the new diagnostic criteria for sinusitis.

With the common involvement of the sinuses on CT with no symptoms of sinusitis it is difficult to comment favorably on these open trials.

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