Introduction

Acute sinusitis is one of the leading diagnoses made in ambulatory medicine. The National Ambulatory Medical Care Survey (NAMCS) estimates that 20 million cases of acute bacterial rhinosinusitis (ABRS) occur each year [1]. The incidence of acute frontal sinusitis (AFS) specifically is considerably lower, less common than maxillary sinusitis in adults and ethmoid sinusitis in children. Medical therapies for acute sinusitis result in expenditures of $3.5 billion per year in the United States. Of all antibiotics prescribed in 2002, 9% of pediatric prescriptions and 18% of adult prescriptions were written for a diagnosis of acute sinusitis [1].

AFS occurs most commonly in adolescent males and young men. While the reasons for the male pred-
lection are unknown, the age predilection appears likely due to the peak vascularity and peak development of the frontal sinuses between the ages of 7 and 20. Although AFS is largely a self-limited disease, complications of acute sinusitis can have catastrophic clinical consequences if not detected promptly.

Etiology and Pathophysiology of Acute Frontal Sinusitis

Acute frontal sinusitis is most commonly preceded by a viral upper respiratory tract infection. Human rhinovirus is implicated in 50% of cases, but other viruses may include coronavirus, influenza, parainfluenza, respiratory syncytial virus, adenovirus, and enterovirus. The peak prevalence of these viruses occurs in early fall and spring, which parallels the peak incidence of ABRS. Viruses upregulate pro-inflammatory cytokines such as interleukin-1, interleukin-6, interleukin-8, tumor necrosis factor-α, as well as other inflammatory mediators such as histamine and bradykinin. Viruses also suppress neutrophil, macrophage, and lymphocyte function and can thereby inhibit the immune response [2]. The viral induction of the inflammatory cascade results in acute mucosal edema, occlusion of sinus ostia, and impaired mucociliary clearance. Mucus stasis can then favor the proliferation of pathogenic micro-organisms, resulting in acute bacterial sinusitis. Other risk factors for acute sinusitis include a variety of host factors: septal deviation, nasal polyposis, and immunodeficiency/immunosuppression, among others.

While acute sinusitis typically affects the ethmoid and maxillary sinuses, progression of disease to involve the frontal sinus may be influenced by anatomical variations of the frontal sinus. The frontal sinus begins developing at age 3. Four frontal pits along the upper lateral wall of the embryological middle meatus differentiate into the anterior ethmoid cells. The second of these furrows evaginates from the anterior ethmoid region into the frontal bone, creating the frontal sinus [3]. Because the frontal sinus is embryologically derived from pneumatization of the ethmoid, frontal sinus outflow is thus influenced and defined by the degree of pneumatization of the ethmoid labyrinth. A variety of ethmoid-derived structures that comprise the frontal recess can thus narrow the outflow tract and predispose to acute frontal sinusitis. These structures may include agger nasi cells anteriorly, the bulla lamella posteriorly, supraorbital ethmoid cells laterally, and type I–IV frontal cells [3].

Uncomplicated Acute Frontal Sinusitis

Diagnosis

AFS is principally a clinical diagnosis based on type and duration of symptoms. CT scans, when ordered to diagnose acute bacterial sinusitis, may yield false positives. Gwaltney et al. showed that 87% of adults with acute onset of upper respiratory tract infection (URI) symptoms demonstrate CT evidence of nasal cavity mucosal thickening and sinus opacification [4]. They also showed that after 2 weeks without antibiotic therapy, repeat CT scans showed improvement in 79% of 14 patients with these findings. Sinus aspiration studies have shown that significant bacterial growth occurs in approximately 60% of patients with URI symptoms lasting for 10 days or more [5]. Therefore persistent or worsening symptoms after 10 days may indicate a bacterial infection [1].

In 1997 the American Academy of Otolaryngology-Head and Neck Surgery Foundation assembled the Rhinosinusitis Task Force (RSTF) to develop clinical definitions of rhinosinusitis. Rhinosinusitis as defined by the RSTF is “inflammation of the nasal cavity and paranasal sinus” [6]. The RSTF subclassified rhinosinusitis into three major clinical categories based on duration of symptoms: acute, with symptoms lasting less than 4 weeks; subacute, between 4 and 12 weeks; and chronic, greater than 12 weeks.

By RSTF guidelines, patients with rhinosinusitis must meet a variety of symptomatic major and minor criteria.

The major criteria defined by the RSTF include:

- Facial pain or pressure
- Nasal congestion
- Nasal obstruction
- Purulent rhinorrhea
- Hyposmia or anosmia
- Fever (for acute rhinosinusitis only)
- Purulence on nasal exam