

# Rhinosinusitis and Tonsillopharyngitis

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## Rhinosinusitis

Sinusitis is characterized by mucosal inflammation of the sinuses which is almost always accompanied by inflammation of the nasal passages. Since nasal mucosa is contiguous with paranasal sinus mucosa, the term *sinusitis* is often used interchangeably with *rhinosinusitis* [1]; the latter term will be used in this chapter. Rhinosinusitis can be acute (less than 4 weeks' duration), subacute (4–12 weeks), or chronic (greater than 12 weeks) [2].

## Epidemiology

Rhinosinusitis is extremely common; in a 2008 national survey, 1 in 7 adults reported having been diagnosed with rhinosinusitis in the previous 12 months [3]. The estimated prevalence of chronic rhinosinusitis in the USA ranges from 2 % to 16 % [4]. Women are disproportionately affected compared to men, and both acute and chronic rhinosinusitis are most prevalent in middle-aged adults compared to any other age group [3]. Primary care physicians and specialists manage rhinosinusitis with equivalent technical efficiency, with primary care physicians providing less costly treatment [5]. Chronic rhinosinusitis has a high economic burden; in 2007, total expenditures in the United States were estimated to be \$8.6 billion [4].

## Risk Factors

Rhinosinusitis is more common in patients with comorbid asthma and allergic rhinitis [6]. Other predisposing factors are listed in Table 1 [2]. Studies investigating the relationship between smoking and rhinosinusitis are conflicting [4].

## Microbiology

The vast majority of cases of acute rhinosinusitis are viral in etiology. The incidence is high; the average adult is affected an estimated 2–5 times per year. Secondary bacterial infection is uncommon and complicates only 0.5–2 % of cases [7]. The two most common bacterial causes of rhinosinusitis are *Streptococcus pneumoniae* and *Haemophilus influenzae*. Less common pathogens include *Moraxella catarrhalis*, *Streptococcus pyogenes*, and *Staphylococcus aureus* [1].

## Clinical Presentation

The classic clinical presentation of rhinosinusitis includes nasal congestion, mucopurulent nasal discharge, facial pain or pressure, and fever. Associated symptoms include anosmia, hyposmia, aural fullness, cough, headache, and toothache [2, 8].

## Diagnosis

The diagnosis of bacterial rhinosinusitis is clinical. Previous studies have used criteria based on symptoms (Table 2) [1, 9]. The diagnosis of rhinosinusitis requires the presence of at least two major criteria or one

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**Table 1** Predisposing factors for rhinosinusitis

Systemic	Viral URI
	Allergy/asthma
	Immotile cilia (e.g., Kartagener syndrome)
	Cystic fibrosis
	Immune disorder
	Gastrointestinal reflux disease
Local	Trauma
	Rhinitis
Mechanical	Choanal atresia
	Deviated septum
	Polyps/foreign body
	Hypertrophy of turbinate or adenoids

Source: Ref. [2]

**Table 2** Conventional criteria for the diagnosis of sinusitis

Major symptoms	Minor symptoms
Purulent anterior nasal discharge	Headache
Purulent or discolored posterior nasal discharge	Ear pain, pressure, fullness
	Halitosis
Nasal congestion or obstruction	Dental pain
Facial congestion or fullness	Cough
Hyposmia or anosmia	Fever (for subacute or chronic sinusitis)
Fever (for acute sinusitis only)	Fatigue

Source: Ref. [1]

major plus two minor criteria. Diagnoses made by these criteria correlated with radiographic evidence of sinus involvement, but did not distinguish between a bacterial and viral etiology. For this reason, the Infectious Diseases Society of America (IDSA) has adopted guidelines based on characteristic patterns that take into account duration, severity, temporal progression, and “double sickening” to differentiate bacterial from viral rhinosinusitis [1]. The diagnosis of bacterial rhinosinusitis requires any of the three following clinical presentations: (a) *persistent* symptoms or signs compatible with acute rhinosinusitis lasting for at least 10 days without improvement, (b) *severe* symptoms or either fever of at least 39 °C or purulent nasal discharge or facial pain lasting for at least 3–4 consecutive days at the onset of illness, and (c) *worsening* symptoms or signs including new onset of fever, headache, or increased nasal discharge that were initially improving 5–6 days following an upper respiratory infection.

### Physical Exam

The IDSA guidelines mentioned previously are the cornerstone of diagnosis, but the following physical exam findings support a suspected diagnosis of bacterial rhinosinusitis: purulent nasal discharge, nasal obstruction, sinus tenderness, nasal mucosal erythema and edema, and/or infraorbital venous pooling [8, 10, 11]. No validated studies have examined the predictive value of specific signs more likely to be associated with a bacterial rather than viral etiology [1].

## Diagnostic Imaging and Laboratory Studies

Although rhinosinusitis is a clinical diagnosis, there are particular settings in which imaging may be useful. Plain radiography is universally recognized as neither useful nor cost effective [12]. Computed tomography is the preferred imaging modality. Imaging may be considered in the following situations: severe or recurrent disease, suspected complications, immunocompromised states, and prior to surgery [7–9]. It should be noted, however, that the severity of symptoms does not correlate with CT findings [13]. Nasal endoscopy, while it allows for better visualization of nasal purulence compared to anterior nasal exam, is often impractical for primary care physicians and is not essential for diagnosis [12]. Cultures obtained from endoscopic aspirates or sinus puncture are considered the gold standard for confirming a bacterial versus viral etiology [14] in order to identify causative organisms in patients with complicated rhinosinusitis, who are immunosuppressed, or who are refractory to treatment [7, 9]. However, these tests are invasive and lack feasibility in primary care settings [14].

## Treatment

### Antimicrobial Therapy

Antibiotic therapy should be initiated once the clinical diagnosis of bacterial rhinosinusitis has been established by the IDSA criteria previously described. Antibiotics initiated in this setting shorten the duration of illness, offer more prompt symptomatic relief, and prevent recurrence and suppurative complications [1]. Standard-dose amoxicillin-clavulanate (875 mg/125 mg twice daily), rather than amoxicillin alone, is recommended as first-line treatment due to the high prevalence of  $\beta$ -lactamase-producing *H. influenzae* [1]. However, standard-dose amoxicillin-clavulanate is inadequate for penicillin-non-susceptible (PNS) *S. pneumoniae*, which have a mutation in the penicillin-binding protein 3 that is unaffected by the addition of a  $\beta$ -lactamase inhibitor. Thus, in patients with certain risk factors, high-dose amoxicillin-clavulanate (i.e., 2 g/125 mg twice daily) is recommended as first-line treatment. Risk factors for PNS include residence in geographic regions with high ( $\geq 10\%$ ) endemic rates of PNS *S. pneumoniae*, severe infection (e.g., signs of systemic toxicity with fever of  $\geq 39^\circ\text{C}$  [ $102^\circ\text{F}$ ]), age  $>65$  years, recent hospitalization, patients who are immunocompromised, or antibiotic use within the past month [1]. Respiratory fluoroquinolones are also highly active against PNS *S. pneumoniae* and *H. influenzae*, but are not superior to  $\beta$ -lactam antibiotics [1]. Options for patients with  $\beta$ -lactam antibiotic allergy include doxycycline (100 mg twice daily or 200 mg daily), levofloxacin (500 mg daily), or moxifloxacin (400 mg daily).

### Adjunctive Therapies

Symptomatic management may include analgesics, antipyretics, intranasal glucocorticoids, hydration, and possibly nasal saline irrigation [1, 15].

Intranasal glucocorticoids are helpful in reducing or relieving symptoms compared to placebo when used as monotherapy or as an adjunct to antibiotics. Higher doses have a stronger effect on relieving symptoms without a significant increase in adverse effects [16].

The benefit of nasal saline irrigation is unclear, but is relatively safe and may reduce time off from work. Minor adverse effects, such as dry nose and irritation, are experienced by fewer than half of users. The optimal concentration, frequency, volume, and technique for irrigation have not been determined [17].

There is little evidence that topical or oral decongestants provide benefit as adjunctive treatment to antibiotics based on symptom scores, histologic changes, or radiographic findings [1]. However, some patients do report symptomatic improvement, so decongestants may be considered for patients with viral rhinosinusitis for whom antibiotic therapy is not indicated [1]. Topical decongestants should be used with

caution, however, since they can trigger rebound congestion and inflammation, especially when used for more than 3 days.

There is also scant evidence that antihistamines provide significant relief in patients with rhinosinusitis, but they may be beneficial in patients with concomitant allergic rhinitis [1]. First-generation antihistamines should be avoided in the elderly, who are more susceptible to anticholinergic effects [18].

Both decongestants and antihistamines should be avoided in children under 2 years of age. The use of these medications may increase morbidity, and a small number of deaths in this population have been reported [19].

Mucolytics thin mucus and improve nasal drainage, but there is no evidence supporting their effectiveness in rhinosinusitis [8].

Systemic steroids or leukotriene inhibitors may be considered in chronic rhinosinusitis, especially in patients with nasal polyps [2, 20, 21].

### **Chronic Rhinosinusitis**

There is a lack of consensus about treatment for chronic rhinosinusitis, likely due to its inherent heterogeneity [12, 15]; therefore referral to an otolaryngologist is warranted in cases of acute rhinosinusitis that do not improve after maximal medical therapy or recurrent infections (defined as 3–4 episodes per year) [1, 7]. In such cases, predisposing medical conditions, such as immunodeficiency, allergic disease, diabetes mellitus, or immotile cilia syndrome, should be considered.

### **Surgical Management**

Surgery is reserved for patients with chronic rhinosinusitis who have failed maximal medical therapy (typically defined as therapy for 4–6 weeks) or who have underlying anatomic abnormalities as well as clear demonstration of rhinosinusitis by CT imaging or endoscopic examination [2, 15]. 90 % of adult patients experience symptomatic improvement after surgery. Surgical intervention is rarely indicated in children [2].

### **Tonsillopharyngitis**

The subjective complaint of sore throat is often described clinically as pharyngitis, but multiple contiguous anatomic structures – including the tonsils, adenoids, nasopharynx, posterior pharynx, uvula, and soft palate – are stretched with swallowing and perceived asodynophagia when inflamed [22]. Thus, in this chapter, discomfort, pain, and scratchiness of the throat will be referred to as one entity: *tonsillopharyngitis*.

### **Epidemiology**

Tonsillopharyngitis is among the most common reasons for primary care visits [23], accounting for 6 % of visits by children to family physicians and pediatricians [24]. Throat-related symptoms were the 14th most common reasons for physician visits in 2010 [25] and the 9th most common reason for emergency room visits in 2011 [26]. The estimated economic burden of group A streptococcal tonsillopharyngitis specifically has been estimated to be between \$224 and \$539 million annually, with children missing an average of 4.5 days of school and parents missing 1.8 days of work in order to care for them [27].

## Etiologies

### Infectious

The most common cause of tonsillopharyngitis is viral infection. Common viruses include rhinovirus, coronavirus, adenovirus, parainfluenza, influenza, echovirus, reovirus, respiratory syncytial virus, herpes simplex virus, coxsackievirus, and Epstein-Barr virus [28, 29]. Bacterial causes include streptococci, *Corynebacterium diphtheriae*, *Neisseria gonorrhoeae*, *Chlamydia pneumoniae*, and *Mycoplasma pneumoniae*. Group A streptococcus (GAS) is the most common bacterial cause. Thrush, most commonly caused by *Candida* species, can also cause sore throat [28].

### Inflammatory

Inflammatory causes of tonsillopharyngitis include laryngopharyngeal reflux, allergic rhinitis with postnasal drip, foreign body, chronic mouth breathing, mucositis, muscle tension dysphonia, vocal cord granuloma, rheumatoid arthritis, gout, pemphigus, and Kawasaki's disease [28].

### Clinical Presentation of GAS Tonsillopharyngitis

The characteristic presentation of GAS tonsillopharyngitis includes abrupt onset of sore throat, odynophagia, fever, headache, abdominal pain, nausea, and vomiting [23]. Physical exam findings may include tonsillopharyngeal erythema and exudate, a beefy-red and swollen uvula, soft palate petechiae, anterior cervical lymphadenopathy, and a scarlatiniform rash.

## Laboratory Diagnosis

The Infectious Disease Society of America (IDSA) guidelines recommend testing for GAS tonsillopharyngitis by rapid antigen detection test (RADT) and/or culture to distinguish GAS from viral tonsillopharyngitis, except when features such as oral ulcers, rhinorrhea, cough, and/or hoarseness strongly suggest a viral etiology [30]. In children and adolescents, a throat culture should be sent after a negative RADT to rule out infection, since the sensitivity of throat culture is 90–95 % while that of the RADT is 70–90 %. A backup culture is not necessary after a positive RADT, as the test is highly specific (approximately 95 %) [30].

## Complications of GAS Tonsillopharyngitis

### Suppurative Complications

Peritonsillar abscesses are most commonly seen in patients between ages 20–40 with recurrent or chronic tonsillopharyngitis that has been inadequately treated [29]. The abscess develops in the space between the lateral aspect of the tonsil and the pharyngeal constrictor muscle [29]. Peritonsillar abscess can be difficult to distinguish from severe tonsillopharyngitis, as both can present with asymmetric tonsillar hypertrophy and drooling. The hallmark of peritonsillar abscess, however, is trismus, which results from inflammation and pus that has tracked above the pterygoid region [28]. Other characteristic symptoms include a muffled “hot potato” voice, severe unilateral sore throat, edema of the ipsilateral soft palate, and deviation of the uvula [28]. When the diagnosis is in question, ultrasonography (transcutaneous or intraoral) or CT imaging can aid in identifying an abscess and thus distinguish from peritonsillar cellulitis [31]. If there is concern for spread of infection into the lateral neck, contrast imaging with MRI or CT is indicated.

Retropharyngeal abscesses affect younger children, typically between 1 and 5 years of age. The characteristic presentation includes neck stiffness, dysphagia, odynophagia, and high fever following

an upper respiratory infection [28]. The airway can be affected and, depending on the degree of obstruction, can manifest as a muffled voice, drooling, trismus, stridor, tachypnea, or tripod positioning. There may be external neck swelling. Immediate consultation with an otolaryngologist and anesthesiologist is warranted given the potential for life-threatening airway compromise. Once the patient is stable, imaging should be obtained. CT of the neck with contrast is the modality of choice [28].

Other suppurative complications of GAS tonsillopharyngitis include cervical lymphadenitis, sinusitis, otitis media, and mastoiditis [32].

### **Nonsuppurative Complications**

Nonsuppurative complications of GAS tonsillopharyngitis are postinfectious and immunologically mediated and include acute rheumatic fever, acute poststreptococcal glomerulonephritis, and poststreptococcal reactive arthritis [32].

### **Treatment of GAS Tonsillopharyngitis**

All patients with uncomplicated GAS tonsillopharyngitis should be treated with antibiotic therapy. Treatment accomplishes three objectives: (1) prevention of both suppurative complications and acute rheumatic fever; (2) decreased communicability, which allows patients to return to work or school; and (3) shortened duration of illness [23]. First-line treatment options are penicillin (250 mg twice to three times daily in children, 250 mg four times daily or 500 mg twice daily in adolescents and adults) or amoxicillin (50 mg/kg daily with a maximum dose of 1,000 mg/day) for 10 days. Penicillin-allergic patients may be treated with a first-generation cephalosporin such as cephalexin (20 mg/kg/dose twice daily with a maximum of 500 mg/dose) or cefadroxil (30 mg/kg daily with a maximum of 1,000 mg/day) for 10 days, clindamycin (7 mg/kg/dose twice daily with a maximum of 300 mg/dose) for 10 days, or azithromycin (12 mg/kg once daily with a maximum of 500 mg/day) for 5 days [30]. There is no evidence that one antibiotic is superior to another [33].

Adjunctive treatment with acetaminophen or nonsteroidal anti-inflammatory agents can be useful in controlling fever and pain [23]. Complementary therapies, including acupuncture, herbal and dietary supplements, have not been shown to be beneficial [34].

Treatment of peritonsillar abscess includes hydration, incision and drainage under local anesthesia, and antibiotics aimed at both aerobic and anaerobic bacteria [28, 29]. Tonsillectomy is indicated if incision and drainage fails to completely drain the abscess [29].

Treatment of retropharyngeal abscess involves immediate intravenous antibiotic therapy aimed at gram-positive aerobes and anaerobes [28]. Small (<2 cm) retropharyngeal abscesses can often be treated with antibiotics alone, but if a patient fails to improve after 48 hours of therapy, incision and drainage is indicated [28].

### **Chronic Carriers, Recurrent Infection, and Asymptomatic Contacts**

Chronic carriers have GAS present in the pharynx but no immunologic response to the organism [30]. In temperate climates during the winter and spring months, as many as 20 % of school-aged children are asymptomatic carriers. Antimicrobial therapy is generally not indicated in these patients, as they are not likely to be contagious or develop suppurative or nonsuppurative complications [30]. However, there are certain indications where eradication of GAS carriage should be considered: during a community outbreak of acute rheumatic fever, in the context of poststreptococcal glomerulonephritis or invasive GAS infection, during an outbreak of GAS tonsillopharyngitis in a closed or partially closed community, the presence of a personal or family history of acute rheumatic fever, in a family with significant anxiety about GAS infections, or when tonsillectomy is under consideration solely because of carriage [30].

There are several explanations for patients with recurrent episodes of GAS tonsillopharyngitis: repeated viral infections in a chronic GAS carrier, noncompliance with antibiotic therapy, or a new infection acquired from a close contact [30]. Test of cure is not indicated, as antibiotic failure is rare if taken as prescribed. If “ping-ponging” of infection within a family is suspected, simultaneously obtaining RADT or cultures from all members and treating those that are positive is reasonable [30].

Asymptomatic contacts should not be treated. Antibiotic prophylaxis of household contacts with penicillin has not been shown to decrease the incidence of developing subsequent GAS tonsillopharyngitis [30].

## Tonsillectomy

Tonsillectomy is one of the most common procedures performed in the United States, with more than 530,000 carried out on children younger than 15 years of age each year [35]. The American Academy of Otolaryngology recommends tonsillectomy for children with 7 episodes or more of tonsillopharyngitis in the past year, 5 episodes or more per year in the past 2 years, or 3 episodes or more per year in the past 3 years. Tonsillectomy has been shown to improve quality of life in children with recurrent tonsillopharyngitis by reducing the number of throat infections, healthcare provider visits, and need for antibiotic therapy [35]. Children who have more severe or frequent episodes of tonsillopharyngitis may benefit more from tonsillectomy than less severely affected children [35]. Tonsillectomy may also be beneficial in patients with multiple antibiotic allergies, PFAPA syndrome (periodic fever, aphthous stomatitis, pharyngitis, and adenitis), and recurrent peritonsillar abscess [35]. There is limited evidence on the effectiveness of tonsillectomy in adults with recurrent tonsillopharyngitis [36], but there is some retrospective data that there is a positive and prolonged effect on quality of life, especially with regards to younger patients with more severe symptoms [37].

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