Acute Pharyngitis

Pharyngitis refers to inflammation of the pharynx, including erythema, edema, exudates, or an enanthem (ulcers, vesicles). Pharyngeal inflammation can be related to environmental exposures, such as tobacco smoke, air pollutants, and allergens; from contact with caustic substances, hot food, and liquids; and from infectious agents. The pharynx and mouth can be involved in various inflammatory conditions such as the periodic fever, aphthous stomatitis, pharyngitis, adenitis (PFAPA) syndrome, Kawasaki disease, inflammatory bowel disease, Stevens-Johnson syndrome, and systemic lupus erythematous.

Noninfectious etiologies are typically evident from history and physical exam, but it can be more challenging to distinguish from among the numerous infectious causes of acute pharyngitis.

Acute infections of the upper respiratory tract account for a substantial number of visits to pediatricians and many feature sore throat as a symptom or evidence of pharyngitis on physical examination. The usual clinical task is to distinguish important, potentially serious, and treatable causes of acute pharyngitis from those that are self-limited and require no specific treatment or follow-up. Specifically, identifying patients who have group A streptococcus (GAS; Streptococcus pyogenes) pharyngitis and treating them with antibiotics forms the core of the management paradigm.

INFECTIOUS ETIOLOGIES

Table 381-1 Infectious Agents That Cause Pharyngitis

VIRUSES	BACTERIA
Adenovirus	*Streptococcus pyogenes* (Group A streptococcus)
Coronavirus	*Arcanobacterium haemolyticum*
Cytomegalovirus	*Fusobacterium necrophorum*
Epstein-Barr virus	*Corynebacterium diphtheriae*
Enteroviruses	*Neisseria gonorrhoeae*
Herpes simplex virus	Group C streptococci
Human immunodeficiency virus	Group G streptococci
Human metapneumovirus	*Francisella tularensis*
Influenza viruses	*Chlamydophila pneumoniae*
Measles virus	*Chlamydia trachomatis*
Parainfluenza viruses	*Mycoplasma pneumoniae*
Respiratory syncytial virus	
Rhinoviruses	

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Viruses

In North America and most industrialized countries GAS is the most important bacterial cause of acute pharyngitis, but viruses predominate as acute infectious causes of pharyngitis. Viral upper respiratory tract infections are typically spread by contact with oral or respiratory secretions and

occur most commonly in fall, winter, and spring, that is, the “respiratory season.” Important viruses that cause pharyngitis include influenza, parainfluenza, adenoviruses, coronaviruses, enteroviruses, rhinoviruses , respiratory syncytial virus, cytomegalovirus, Epstein-Barr virus, herpes simplex virus, and human metapneumovirus . Most viral pharyngitis, except mononucleosis, is mild. Common nonspecific symptoms such as rhinorrhea and cough develop gradually before they become prominent. However, specific findings are sometimes helpful in identifying the infectious viral agent.

Gingivostomatitis and ulcerating vesicles throughout the anterior pharynx and on the lips are seen in primary oral herpes simplex virus infection. High fever and difficulty taking oral fluids are common. This infection can last for 14 days.

Discrete papulovesicular lesions or ulcerations in the posterior oropharynx, severe throat pain, and fever are characteristic of herpangina, caused by various enteroviruses. In hand-foot-mouth disease there are vesicles or ulcers throughout the oropharynx, vesicles on the palms and soles, and sometimes on the trunk and extremities; Coxsackie A16 is the most common agent, but Enterovirus 71 and Coxsackie A6 can also cause this syndrome. Enteroviral infections are most common in the summer.

Various adenoviruses cause pharyngitis. When there is concurrent conjunctivitis the syndrome is called pharyngoconjunctival fever. The pharyngitis tends to resolve within 7 days but conjunctivitis may persist for up to 14 days. Pharyngoconjunctival fever can be epidemic or sporadic; outbreaks have been associated with exposure in swimming pools.

Intense, diffuse pharyngeal erythema and Koplik spots, the pathognomonic enanthem, occur in advance of the characteristic rash of measles. Splenomegaly or hepatomegaly may be the clue to EpsteinBarr virus infectious mononucleosis in an adolescent with exudative tonsillitis. Primary infection with HIV can manifest as the acute retroviral syndrome, with non-exudative pharyngitis, fever, maculopapular rash, arthralgia, myalgia, adenopathy, and often a maculopapular rash.

Bacteria Other Than Group A Streptococcus

In addition to GAS, bacteria that cause pharyngitis include group C and group G streptococcus, Arcanobacterium haemolyticum , Francisella tularensis , Neisseria gonorrhoeae, Mycoplasma pneumoniae, Chlamydophila (formerly Chlamydia) pneumoniae, Chlamydia trachomatis, Fusobacterium necrophorum, and Corynebacterium diphtheriae. Haemophilus influenzae and Streptococcus pneumoniae may be cultured from the throats of children with pharyngitis, but their role in causing pharyngitis has not been established.

Group C and Group G streptococcus and A. haemolyticum pharyngitis have been diagnosed most commonly in adolescents and adults.

They resemble group A β-hemolytic streptococcus (GABHS) pharyngitis and a scarlet fever–like rash may be present with A. haemolyticum infections.

F. necrophorum has been suggested to be a fairly common cause of pharyngitis in older adolescents and adults (15-30 yr old). Prevalence in studies has varied from 10-48% of patients with non-GABHS pharyngitis, but large surveillance studies have not been performed.

F.necrophorum pharyngitis is associated with development of Lemierre syndrome, internal jugular vein septic thrombophlebitis. Approximately 80% of cases of Lemierre syndrome are caused by this bacterium.

Patients present initially with fever, sore throat, exudative pharyngitis, and/or peritonsillar abscess. The symptoms may persist, neck pain and swelling develop, and the patient appears toxic. Septic shock may ensue along with metastatic complications from septic emboli that can involve the lungs, bones and joints, central nervous system, abdominal organs, and soft tissues. The case fatality rate is 4-9%.

Gonococcal pharyngeal infections are usually asymptomatic but can cause acute pharyngitis with fever and cervical lymphadenitis. Young children with proven gonococcal disease should be evaluated for sexual abuse.

Diphtheria is extremely rare in most developed countries thanks to extensive immunization with diphtheria toxoid. However, it remains endemic in many areas of the world, including the former Soviet bloc countries, Africa, Asia, the Middle East, and Latin America. It can be considered in patients with recent travel to or from these areas and in unimmunized patients. Key physical findings are bull neck (extreme neck swelling) and a gray pharyngeal pseudomembrane that can cause respiratory obstruction.

Ingestion of water, milk, or undercooked meat contaminated by F. tularensis can lead to oropharyngeal tularemia. Severe throat pain, tonsillitis, cervical adenitis, oral ulcerations, and a pseudomembrane (as in diphtheria) may be present. M. pneumoniae and C. pneumoniae cause pharyngitis, but other upper and lower respiratory infections are more important and more readily recognized. Development of a severe or persistent cough subsequent to pharyngitis may be the clue to infection with one of these organisms.

Group A Streptococcus

Streptococcal pharyngitis is relatively uncommon before 2-3 yr of age, is quite common among children 5-15 yr old, and declines in frequency in late adolescence and adulthood. Illness occurs throughout the year but is most prevalent in winter and spring. It is readily spread among siblings and schoolmates. GAS causes 15-30% of pharyngitis in school-age children.

Colonization of the pharynx by GAS can result in either asymptomatic carriage or acute infection. After an incubation period of 2-5 days, pharyngeal infection with GAS classically presents as rapid onset of significant sore throat and fever. The pharynx is red, the tonsils are enlarged and often covered with a white, grayish, or yellow exudate that may be blood-tinged. There may be petechiae or “doughnut” lesions on the soft palate and posterior pharynx and the uvula may be red and swollen. The surface of the tongue can resemble a strawberry when the papillae are inflamed and prominent (“strawberry tongue”).

Initially, the tongue is often coated white, and with the swollen papillae it is called a “white strawberry tongue.” When the white coating is gone after a few days, the tongue is often quite red, and is called a “red strawberry tongue.” Enlarged and tender anterior cervical lymph nodes are frequently present. Headache, abdominal pain, and vomiting are frequently associated with the infection, but in the absence of clinical pharyngitis, gastrointestinal signs and symptoms should not be attributed to GAS. Ear pain is a frequent complaint but the tympanic membranes are usually normal. Diarrhea, cough, coryza, ulcerations, croup/ laryngitis/hoarseness, and

conjunctivitis are not associated with GAS pharyngitis and increase the likelihood of a viral etiology.

Patients infected with GAS that produce streptococcal pyrogenic exotoxin A, B, or C may demonstrate the fine red, papular ("sandpaper") rash of scarlet fever. It begins on the face and then becomes generalized. The cheeks are red and the area around the mouth is more pale, giving the appearance of circumoral pallor. The rash blanches with pressure and it may be more intense in skin creases, especially in the antecubital fossae, axillae, and inguinal creases (Pastia's lines or Pastia's sign). Pastia's lines are sometimes petechial or slightly hemorrhagic.

Capillary fragility can cause petechiae distal to a tourniquet or constriction from clothing, a positive tourniquet test or Rumpel-Leeds phenomenon. Erythema fades in a few days and when the rash resolves it typically peels like a mild sunburn. Sometimes there is sheet-like desquamation around the free margins of the finger nails. Streptococcal pyrogenic exotoxin A, encoded by the gene spe A, is the exotoxin most commonly associated with scarlet fever.

The M protein is an important GAS virulence factor that facilitates resistance to phagocytosis. The M protein is encoded by the emm gene and determines the M type (or emm type). Molecular methods have identified more than 200 emm genes (emm types). The M protein is immunogenic; an individual can experience multiple episodes of GAS pharyngitis in a lifetime because natural immunity is M type-specific.

Numerous GAS M types can circulate in a community simultaneously and they enter and leave communities unpredictably and for unknown reasons.

DIAGNOSIS

The clinical presentations of streptococcal and viral pharyngitis often overlap. In particular, the pharyngitis of mononucleosis can be difficult to distinguish from GAS pharyngitis. Physicians relying solely on clinical judgment often overestimate the likelihood of a streptococcal etiology. Various clinical scoring systems have been described to assist in identifying patients who are likely to have GAS pharyngitis. Criteria developed for adults and modified for children by McIsaac give 1 point for each of the following criteria: history of temperature >38°C (100.4°F); absence of cough; tender anterior cervical adenopathy; tonsillar swelling or exudates; and age 3-14 yr. It subtracts a point for age ≥45 yr. At best, a McIsaac score ≥4 is associated with a positive laboratory test for GAS in less than 70% of children with pharyngitis (Table 381-2), so it, too, overestimates the likelihood of GAS. Consequently, laboratory testing is essential for accurate diagnosis. Clinical findings and/or scoring systems can best be used to assist the clinician in identifying patients in need of testing. Streptococcal antibody tests are not useful in assessing patients with acute pharyngitis.

Throat culture and rapid antigen-detection tests (RADTs) are the diagnostic tests for GAS available in routine clinical care. Throat culture remains the "gold standard" for diagnosing streptococcal pharyngitis. There are both false-negative cultures as a consequence of sampling errors or prior antibiotic treatment and false-positive cultures as a consequence of misidentification of other bacteria as GAS.

Some laboratories prefer nucleic acid testing that is specific for GAS and no longer use culture to confirm the diagnosis. A child who is chronically colonized with GAS (streptococcal carrier) can have a positive culture if it is obtained when the child is evaluated for pharyngitis that is actually caused by a viral infection.

Table 381-2 Positive Predictive Value of McIsaac Score in Children in Clinical Studies*

SCORE	McISAAC, 2004 (N = 454)	EDMONSON, 2005 (N = 1184)	TANZ, 2009 (N = 1848)	FINE, 2012 (N = 64,789)
0	—	—	7%	17%
1	—	0.5%	19%	23%
2	20.5%	8.9%	20%	34%
3	27.5%	42.4%	29%	50%
≥4	67.8%	48.2%	49%	68%
GAS prevalence	34%	38%	30%	37%

*One point is given for each of the following criteria: history of temperature >38°C (100.4°F); absence of cough; tender anterior cervical adenopathy; tonsillar swelling or exudates; and age 3-14 yr. Note that the Centor score lacks only the age criterion. Positive predictive value refers to the proportion of patients with documented GAS by rapid antigen-detection test and/or throat culture.

Streptococcal RADTs detect the group A carbohydrate of GAS. They are used by the vast majority of office-based pediatricians. All RADTs have very high specificity, generally ≥95%, so when a RADT is positive it is assumed to be accurate and throat culture is unnecessary. Because RADTs are generally less sensitive than culture, confirming a negative rapid test with a throat culture is recommended.

RADTs and throat culture exhibit spectrum bias: They are more sensitive when the pretest probability of GAS is high (signs and symptoms are typical of GAS infection) and less sensitive when the pretest probability is low. Avoidance of testing when patients have signs and symptoms more suggestive of a viral infection is recommended.

Testing for bacteria other than GAS is performed infrequently, and should be reserved for patients with persistent symptoms and symptoms suggestive of a specific non-GAS bacterial pharyngitis, for example, when there is concern for gonococcal infection or sexual abuse. Special culture media and a prolonged incubation are required to detect A. haemolyticum. Viral cultures are often unavailable and are generally too expensive and slow to be clinically useful. Polymerase chain reaction is more rapid and multiplex polymerase chain reaction testing for respiratory pathogens can identify a variety of viral and bacterial agents within a few hours. This may be useful in determining the isolation needs of hospitalized patients, assisting in patient prognosis, and epidemiology, but in the absence of specific treatment for most viral infections such testing is usually not necessary. A complete blood cell count showing many atypical lymphocytes and a positive mononucleosis slide agglutination test can help to confirm a clinical diagnosis of Epstein-Barr virus infectious mononucleosis.

TREATMENT

Specific therapy is unavailable for most viral pharyngitis. However, nonspecific, symptomatic therapy can be an important part of the overall treatment plan. An oral antipyretic/analgesic agent (acetaminophen or ibuprofen) can relieve fever and sore throat pain. Anesthetic sprays and lozenges (often containing benzocaine, phenol, or menthol) can provide local relief in children who are developmentally appropriate to use them. Systemic corticosteroids are sometimes used in children who have evidence of upper airway compromise due to mononucleosis. Although corticosteroids are used fairly commonly in adults with pharyngitis, large scale studies capable of providing safety and efficacy data are lacking in children. Corticosteroids cannot be recommended for treatment of most pediatric pharyngitis.

Antibiotic therapy of bacterial pharyngitis depends on the organism identified. On the basis of in vitro susceptibility data, oral penicillin is often suggested for patients with group C streptococcal isolates and oral erythromycin is recommended for patients with A. haemolyticum, but the clinical benefit of such treatment is uncertain.

Most untreated episodes of GAS pharyngitis resolve uneventfully within 5 days, but early antibiotic therapy hastens clinical recovery by 12-24 hr. The primary benefit and intent of antibiotic treatment is the prevention of acute rheumatic fever (ARF); it is highly effective when started within 9 days of onset of illness. Antibiotic therapy does not prevent acute poststreptococcal glomerulonephritis (APSGN). Antibiotic therapy should not be delayed for children with symptomatic pharyngitis and a positive GAS RADT or throat culture. Presumptive antibiotic treatment can be started when there is a clinical diagnosis of scarlet fever, asymptomatic child has a household contact with documented streptococcal pharyngitis, or a history of ARF in the patient or a family member, but a diagnostic test should be performed to confirm the presence of GAS.

A variety of antimicrobial agents are effective for GAS pharyngitis (Table 381-3). Group A streptococci are universally susceptible to penicillin and all other β-lactam antibiotics. Penicillin is inexpensive, has a narrow spectrum of activity, and few adverse effects. Amoxicillin is preferred for children because of taste, availability as chewable tablets and liquid, and convenience of once-daily dosing. The duration of oral penicillin and amoxicillin therapy is 10 days. A single intramuscular dose of benzathine penicillin or a benzathine-procaine penicillin G combination is effective and ensures compliance. Follow-up testing for GAS is unnecessary after completion of therapy and is not recommended unless symptoms recur.

Patients allergic to penicillin can be treated with a 10-day course of a narrow-spectrum (first-generation) cephalosporin (cephalexin or cefadroxil) if the previous reaction to penicillin was not an immediate, type I hypersensitivity reaction. Most often, penicillin-allergic patients are treated for 10 days with erythromycin, clarithromycin, or clindamycin, or for 5 days with azithromycin.

The increased use of macrolides and related antibiotics for a variety of infections, especially the azalide, azithromycin, is associated with increased rates of resistance to these drugs among GAS in many countries. Approximately 5% of GAS in the United States and more than 10% in Canada are macrolide-resistant (macrolide resistance includes azalide resistance), but there is considerable local variation in both countries. Some macrolide-resistant GAS isolates are also resistant to clindamycin. Although not a major hindrance for treatment of pharyngitis, clindamycin resistance may be important in management of invasive GAS infections. Use of these antibiotics should be restricted to patients who cannot safely receive a β–lactam drug for GAS pharyngitis. Tetracyclines, fluoroquinolones, or sulfonamides should not be used to treat GAS pharyngitis.

CHRONIC GROUP A STREPTOCOCCUS CARRIERS

Patients who continue to harbor GAS in the pharynx despite appropriate antibiotic therapy are streptococcal carriers. They have little or no evidence of an immune response to the organism. The pathogenesis of chronic carriage is not known. Carriage generally poses little risk to patients and their contacts, but it can confound testing in subsequent.

Table 381-3 Recommended Treatment for Acute Streptococcal Pharyngitis

MOST PATIENTS				
	WEIGHT <27 kg	**WEIGHT ≥27 kg**	**ROUTE**	**DURATION**
Amoxicillin	50 mg/kg once daily (maximum 1000 mg)		Oral	10 days
Penicillin V	250 mg bid	500 mg bid	Oral	10 days
Benzathine penicillin G	600,000 units	1.2 million units	IM	Once
Benzathine penicillin G + procaine penicillin G	900,000 units + 300,000 units	900,000 units + 300,000 units	IM	Once

PENICILLIN-ALLERGIC PATIENTS			
	ORAL DOSE	**FREQUENCY**	**DURATION**
Cephalosporins*	Varies with agent chosen		10 days
Erythromycin Ethylsuccinate	40 mg/kg/day up to 1000 mg/day	bid	10 days
Estolate	20-40 mg/kg/day up to 1000 mg/day	bid	10 days
Clarithromycin	15 mg/kg/day up to 500 mg/day	bid	10 days
Azithromycin†	12 mg/kg day 1; 6 mg/kg days 2-5	qd	5 days
Clindamycin	20 mg/kg/day up to 1.8 g/day	tid	10 days

*First-generation cephalosporins are preferred; dosage and frequency vary among agents. Do not use in patients with history of immediate (anaphylactic) hypersensitivity to penicillin or other β-lactam antibiotics.
†Maximum dose is 500 mg the 1st day, 250 mg subsequent days.

episodes of sore throat. Patients with repeated test-positive pharyngitis create anxiety among their families and physicians. It is usually unnecessary to attempt to eliminate chronic carriage. Instead, evaluation and treatment of pharyngitis should be undertaken without regard for chronic carriage, treating test-positive patients in routine fashion and avoiding antibiotics in patients who have negative tests. Expert opinion suggests that eradication might be attempted in select circumstances: a community outbreak of ARF or APSGN; personal or family history of ARF; an outbreak of GAS pharyngitis in a closed or semiclosed community, nursing home or healthcare facility; repeated episodes of symptomatic GAS pharyngitis in a family with "ping pong" spread among family members despite adequate therapy; when tonsillectomy is being considered because of chronic carriage or recurrent streptococcal pharyngitis; and extreme, unmanageable anxiety related to GAS carriage ("streptophobia") among family members. Clindamycin given by mouth for 10 days is effective therapy (20 mg/kg/day divided in 3 doses; adult dose 150-450 mg tid). Amoxicillin-clavulanate (40 mg amoxicillin/kg/day up to 2000 mg amoxicillin/day divided tid for 10 days) and 4 days of oral rifampin plus either intramuscular benzathine penicillin given once or oral penicillin given for 10 days have also been used.

RECURRENT PHARYNGITIS

True recurrent GAS pharyngitis can occur for several reasons: reinfection with the same M type if type-specific antibody has not developed; poor compliance with oral antibiotic therapy; macrolide resistance if a macrolide was used for treatment; and infection with a new M type.

Unfortunately, determining the GAS M type in an acute infection is not available to the clinician. Treatment with intramuscular benzathine penicillin eliminates nonadherence to therapy. Apparent recurrences can represent pharyngitis of another cause in the presence of streptococcal carriage. Chronic GAS carriage is particularly likely if the illnesses are mild and otherwise atypical for GAS pharyngitis.

Undocumented histories of recurrent pharyngitis are an inadequate basis for recommending tonsillectomy.

Tonsillectomy may lower the incidence of pharyngitis for 1-2 yr among children with frequent episodes of documented pharyngitis (≥7 episodes in the previous year or ≥5 in each of the preceding 2 yr, or ≥3 in each of the previous 3 yr). However, the frequency of pharyngitis (GAS and non-GAS) generally declines over time. By 2 yr posttonsillectomy the incidence of pharyngitis in severely affected children is similar among those who have tonsillectomy and those who do not.

Few children are so severely affected and the limited clinical benefit of tonsillectomy for most must be balanced against the risks of anesthesia and surgery.

Recurrent GAS pharyngitis is rarely, if ever, a sign of an immune disorder. However, recurrent pharyngitis can be part of a recurrent fever or autoinflammatory syndrome such as PFAPA syndrome. Prolonged pharyngitis (>1 wk) can occur in infectious mononucleosis and Lemierre syndrome, but it also suggests the possibility of another disorder such as neutropenia, a recurrent fever syndrome, or an autoimmune disease such as systemic lupus or inflammatory bowel disease.

In such instances, pharyngitis would be one of a number of clinical findings that together should suggest the underlying diagnosis.

COMPLICATIONS AND PROGNOSIS

Viral respiratory tract infections can predispose to bacterial middle ear infections and bacterial sinusitis. The complications of GAS pharyngitis include local suppurative complications, such as parapharyngeal abscess, and subsequent nonsuppurative illnesses, such as ARF, APSGN, poststreptococcal reactive arthritis, and possibly PANDAS (pediatric autoimmune neuropsychiatric disorders associated with S.pyogenes) or CANS (childhood acute neuropsychiatric symptoms).

PREVENTION

A variety of GAS vaccines are being developed. A recombinant multivalent M-type vaccine uses the terminal portions of various M proteins to take advantage of their immunogenicity. Other vaccines are based on more conserved GAS epitopes in order to avoid the necessity of matching the vaccine with the M types prevalent in a community or target population. None of the investigational GAS vaccines are near licensing for use. Antimicrobial prophylaxis with daily oral penicillin prevents recurrent GAS infections but is recommended only to prevent recurrences of ARF.

Retropharyngeal Abscess, Lateral Pharyngeal (Parapharyngeal) Abscess, and Peritonsillar Cellulitis/Abscess

The retropharyngeal and the lateral pharyngeal lymph nodes that drain the mucosal surfaces of the upper airway and digestive tracts are located in the neck within the retropharyngeal space (located between the pharynx and the cervical vertebrae and extending down into the superior mediastinum) and the lateral pharyngeal space (bounded by the pharynx medially, the carotid sheath posteriorly, and the muscles of the styloid process laterally). The lymph nodes in these

deep neck spaces communicate with each other, allowing bacteria from either cellulitis or node abscess to spread to other nodes. Infection of the nodes usually occurs as a result of extension from a localized infection of the oropharynx.

A retropharyngeal abscess can also result from penetrating trauma to the oropharynx, dental infection, and vertebral osteomyelitis. Once infected, the nodes may progress through 3 stages:

cellulitis, phlegmon, and abscess. Infection in the retropharyngeal and lateral pharyngeal spaces can result in airway compromise or posterior mediastinitis, making timely diagnosis important.

RETROPHARYNGEAL AND LATERAL PHARYNGEAL ABSCESS

Retropharyngeal abscess occurs most commonly in children younger than 3-4 yr of age; as the retropharyngeal nodes involute after 5 yr of age, infection in older children and adults is much less common.

Boys are affected more often than girls and approximately two-thirds of patients have a history of recent ear, nose, or throat infection.

Clinical manifestations of retropharyngeal abscess are nonspecific and include fever, irritability, decreased oral intake, and drooling. Neck stiffness, torticollis, and refusal to move the neck may also be present.

The verbal child might complain of sore throat and neck pain. Other signs can include muffled voice, stridor, respiratory distress, or even obstructive sleep apnea. Physical examination can reveal bulging of the posterior pharyngeal wall, although this is present in <50% of infants with retropharyngeal abscess. Cervical lymphadenopathy may also be present. Lateral pharyngeal abscess commonly presents as fever, dysphagia, and a prominent bulge of the lateral pharyngeal wall, sometimes with medial displacement of the tonsil.

The differential diagnosis includes acute epiglottitis and foreign body aspiration. In the young child with limited neck mobility, meningitis must also be considered. Other possibilities include lymphoma, hematoma, and vertebral osteomyelitis.

Incision and drainage and culture of an abscessed node provides the definitive diagnosis, but CT can be useful in identifying the presence of a retropharyngeal, lateral pharyngeal, or parapharyngeal abscess (Figs. 382-1 and 382-2). With CT scans, deep neck infections can be accurately identified and localized, but CT accurately identifies abscess formation in only 63% of patients. Soft-tissue neck films taken during inspiration with the neck extended might show increased width or an air–fluid level in the retropharyngeal space. CT with contrast medium enhancement can reveal central lucency, ring enhancement, or scalloping of the walls of a lymph node. Scalloping of the abscess wall is thought to be a late finding and predicts abscess formation.

Retropharyngeal and lateral pharyngeal infections are most often polymicrobial; the usual pathogens include group A streptococcus, oropharyngeal anaerobic bacteria, and Staphylococcus aureus. In children younger than age 2 yr, there has been an increase in the incidence of retropharyngeal abscess, particularly with S. aureus, including methicillin-resistant strains. Mediastinitis may be identified on CT in some of these patients. Other pathogens can include Haemophilus influenzae, Klebsiella, and Mycobacterium avium-intracellulare.

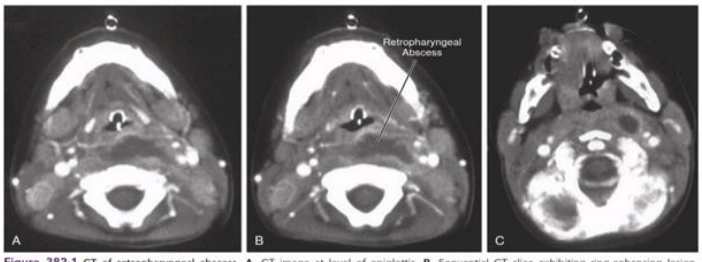

Figure 382-1 CT of retropharyngeal abscess. **A,** CT image at level of epiglottis. **B,** Sequential CT slice exhibiting ring-enhancing lesion. **C,** Further sequential CT slice demonstrating inferior extent of lesion. *(From Philpott CM, Selvadurai D, Banerjee AR: Paediatric retropharyngeal abscess, J Laryngol Otol 118:925, 2004.)*

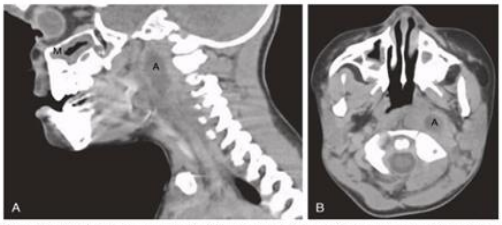

Figure 382-2 CT of parapharyngeal abscess in a 3 yr old child. **A,** Sagittal section demonstrating parapharyngeal abscess *(A)* and mucosal swelling *(M)* in the maxillary sinus. **B,** Coronal section of parapharyngeal abscess *(A)*.

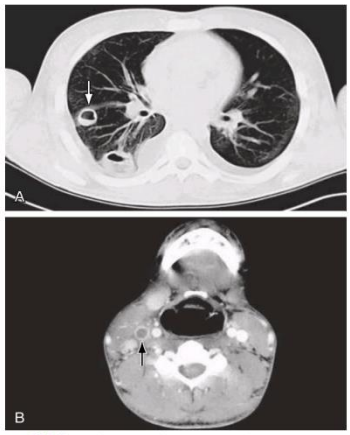

Figure 382-3 CT of Lemierre disease. **A,** CT demonstrating nodular appearance of pulmonary infiltrates *(arrow)*. **B,** CT of neck demonstrating thrombosis of right internal jugular vein *(arrow)*. *(From Plymyer MR, Zoccola DC, Tallarita G: An 18 year old man presenting with sepsis following a recent pharyngeal infection, Arch Pathol Lab Med 128:813, 2004. Reprinted with permission from Archives of Pathology & Laboratory Medicine. Copyright 2004. College of American Pathologists.)*

Treatment options include intravenous antibiotics with or without surgical drainage. A third-generation cephalosporin combined with ampicillin-sulbactam or clindamycin to provide anaerobic coverage is effective. The increasing prevalence of methicillin-resistant S. aureus can influence empiric antibiotic therapy. Studies show that >50% of children with retropharyngeal or lateral pharyngeal abscess as identified by CT can be successfully treated without surgical drainage. Drainage is necessary in the patient with respiratory distress or failure to improve with intravenous antibiotic treatment. The optimal duration of treatment is unknown, but therapy for several days with intravenous antibiotics until the patient has begun to improve followed by a course of oral antibiotic is typically used.

Complications of retropharyngeal or lateral pharyngeal abscess include significant upper airway obstruction, rupture leading to aspiration pneumonia, and extension to the mediastinum. Thrombophlebitis of the internal jugular vein and erosion of the carotid artery sheath can also occur.

An uncommon but characteristic infection of the parapharyngeal space is Lemierre disease, in which infection from the oropharynx extends to cause septic thrombophlebitis of the internal jugular vein and embolic abscesses in the lungs (Fig. 382-3). The causative pathogen is Fusobacterium necrophorum, an anaerobic bacterial constituent of the oropharyngeal flora.

The typical presentation is that of a previously healthy adolescent or young adult with a history of recent pharyngitis who becomes acutely ill with fever, hypoxia, tachypnea, and respiratory distress. Chest radiography demonstrates multiple cavitary nodules, often bilateral and often accompanied by pleural effusion. Blood culture may be positive. Treatment involves prolonged intravenous antibiotic therapy with penicillin or cefoxitin; surgical drainage of extrapulmonary metastatic abscesses may be necessary.

PERITONSILLAR CELLULITIS AND/OR ABSCESS

Peritonsillar cellulitis and/or abscess, which is relatively common compared to the deep neck infections, is caused by bacterial invasion through the capsule of the tonsil, leading to cellulitis and/or abscess formation in the surrounding tissues. The typical patient with a peritonsillar abscess is an adolescent with a recent history of acute pharyngotonsillitis. Clinical manifestations include sore throat, fever, trismus and dysphagia.

Physical examination reveals an asymmetric tonsillar bulge with displacement of the uvula. An asymmetric tonsillar bulge is diagnostic, but it may be poorly visualized because of trismus. CT is helpful for revealing the abscess. Group A streptococci and mixed oropharyngeal anaerobes are the most common pathogens, with more than 4 bacterial isolates per abscess typically recovered by needle aspiration.

Treatment includes surgical drainage and antibiotic therapy effective against group A streptococci and anaerobes. Surgical drainage may be accomplished through needle aspiration, incision and drainage, or tonsillectomy. Needle aspiration can involve aspiration of the superior, middle, and inferior aspects of the tonsil to locate the abscess. Intraoral ultrasound can be used to diagnose and guide needle aspiration of a peritonsillar abscess. General anesthesia may be required for the uncooperative patient. Approximately 95% of peritonsillar abscesses resolve after needle aspiration and antibiotic therapy. A small percentage of these patients require a repeat needle aspiration. The 5% with infections that fail to resolve after needle aspiration require incision and drainage. Tonsillectomy should be considered if there is failure to improve within 24 hr of antibiotic therapy and needle aspiration, history of recurrent peritonsillar abscess or recurrent tonsillitis, or complications from peritonsillar abscess. The feared, albeit rare, complication is rupture of the abscess, with resultant aspiration pneumonitis. There is a 10% recurrence risk for peritonsillar abscess.

Tonsils and Adenoids

ANATOMY

The Waldeyer ring (the lymphoid tissue surrounding the opening of the oral and nasal cavities into the pharynx) comprises the palatine tonsils, the pharyngeal tonsil or adenoid, lymphoid tissue surrounding the eustachian tube orifice in the lateral walls of the nasopharynx, the lingual tonsil at the base of the tongue, and scattered lymphoid tissue throughout the remainder of the pharynx, particularly behind the posterior pharyngeal pillars and along the posterior pharyngeal wall.

The palatine tonsil consists of lymphoid tissue located between the palatoglossal fold (anterior tonsillar pillar) and the palatopharyngeal fold (posterior tonsillar pillar) forms. This lymphoid tissue is separated from the surrounding pharyngeal musculature by a thick fibrous capsule. The adenoid is a single aggregation of lymphoid tissue that occupies the space between the nasal septum and the posterior pharyngeal wall. A thin fibrous capsule separates it from the underlying structures; the adenoid does not contain the complex crypts that are found in the palatine tonsils but rather more simple crypts. Lymphoid tissue at the base of the tongue forms the lingual tonsil that also contains simple tonsillar crypts.

NORMAL FUNCTION

Located at the opening of the pharynx to the external environment, the tonsils and adenoid are well situated to provide primary defense against foreign matter. The immunologic role of the tonsils and adenoids is to induce secretory immunity and to regulate the production of the secretory immunoglobulins. Deep crevices within tonsillar tissue form tonsillar crypts that are lined with squamous epithelium and host a concentration of lymphocytes at their bases. The lymphoid tissue of the Waldeyer ring is most immunologically active between 4 and 10 yr of age, with a decrease after puberty. Adenotonsillar hypertrophy is greatest between ages 3 and 6 yr; in most children tonsils begin to involute after age 8 yr. No major immunologic deficiency has been demonstrated after removal of either or both of the tonsils and adenoid.

PATHOLOGY

Most episodes of acute pharyngotonsillitis are caused by viruses. Group A β-hemolytic streptococcus (GABHS) is the most common cause of bacterial infection in the pharynx

Chronic Infection

The tonsils and adenoids can be chronically infected by multiple microbes, which can include a high incidence of β-lactamase– producing organisms. Both aerobic species, such as streptococci and Haemophilus influenzae, and anaerobic species, such as Peptostreptococcus, Prevotella, and Fusobacterium, contribute. The tonsillar crypts can accumulate desquamated epithelial cells, lymphocytes, bacteria, and other debris, causing cryptic tonsillitis. With time, these cryptic plugs can calcify into tonsillar concretions or tonsillolith. Biofilms appear to play a role in chronic inflammation of the tonsils.

Airway Obstruction

Both the tonsils and adenoids are a major cause of upper airway obstruction in children. Airway obstruction in children is typically manifested in sleep-disordered breathing, including obstructive sleep apnea, obstructive sleep hypopnea, and upper airway resistance syndrome. Sleep-disordered breathing secondary to adenotonsillar breathing is a cause of growth failure.

Tonsillar Neoplasm

Rapid enlargement of one tonsil is highly suggestive of a tonsillar malignancy, typically lymphoma in children.

CLINICAL MANIFESTATIONS

Symptoms of GABHS infection include odynophagia, dry throat, malaise, fever and chills, dysphagia, referred otalgia, headache, muscular aches, and enlarged cervical nodes. Signs include dry tongue, erythematous enlarged tonsils, tonsillar or pharyngeal exudate, palatine petechiae, and enlargement and tenderness of the jugulodigastric lymph nodes (Fig. 383-1).

Chronic Infection

Children with chronic or cryptic tonsillitis often present with halitosis, chronic sore throats, foreign-body sensation, or a history of expelling foul-tasting and foul-smelling cheesy lumps. Examination reveals tonsils of a range of sizes which often they contain copious debris within the crypts. The offending organism is not usually GABHS.

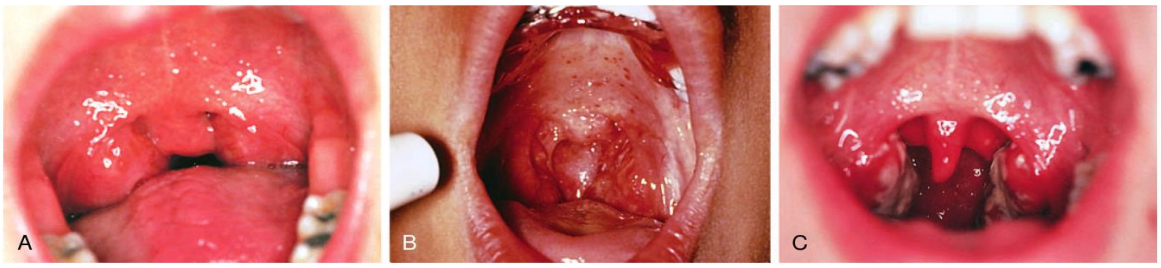

Figure 383-1 Pharyngotonsillitis. This common syndrome has a number of causative pathogens and a wide spectrum of severity. **A,** The diffuse tonsillar and pharyngeal erythema seen here is a nonspecific finding that can be produced by a variety of pathogens. **B,** This intense erythema, seen in association with acute tonsillar enlargement and palatal petechiae, is highly suggestive of group A β-streptococcal infection, though other pathogens can produce these findings. **C,** This picture of exudative tonsillitis is most commonly seen with either group A streptococcal or Epstein-Barr virus infection. (***B** courtesy of Michael Sherlock, MD, Lutherville, MD. From Yellon RF, McBride TP, Davis HW: Otolaryngology. In Zitelli BJ, Davis HW, editors:* Atlas of pediatric physical diagnosis, *ed 4, Philadelphia, 2002, Mosby, p. 852.)*

Airway Obstruction

The diagnosis of airway obstruction can frequently be made by history and physical examination. Daytime symptoms of airway obstruction, secondary to adenotonsillar hypertrophy, include chronic mouth breathing, nasal obstruction, hyponasal speech, hyposmia, decreased appetite, poor school performance, and, rarely, symptoms of right-sided heart failure. Nighttime symptoms consist of loud snoring, choking, gasping, frank apnea, restless sleep, abnormal sleep positions, somnambulism, night terrors, diaphoresis, enuresis, and sleep talking. Large tonsils are typically seen on examination, although the absolute size might not indicate the degree of obstruction. The size of the adenoid tissue can be demonstrated on a lateral neck radiograph or with flexible endoscopy. Other signs that can contribute to airway obstruction include the presence of a craniofacial syndrome or hypotonia.

Tonsillar Neoplasm

The rapid unilateral enlargement of a tonsil, especially if accompanied by systemic signs of night sweats, fever, weight loss, and lymphadenopathy, is highly suggestive of a tonsillar malignancy. The diagnosis of a tonsillar malignancy should also be entertained if the tonsil appears grossly abnormal. Among 54,901 patients undergoing tonsillectomy, 54 malignancies were identified (0.087% prevalence); all but 6 malignancies had been suspected based on suspicious anatomic features preoperatively.

TREATMENT Medical Management

The treatment of acute pharyngotonsillitis antibiotic treatment of GABHS as pharyngitis . Because copathogens such as staphylococci or anaerobes can produce β-lactamase that can inactivate penicillin, the use of cephalosporins or clindamycin may be more efficacious in the treatment of chronic throat infections. Tonsillolith or debris may be expressed manually with either a cottontipped applicator or a water jet. Chronically infected tonsillar crypts can be cauterized using silver nitrate.

Tonsillectomy

Tonsillectomy alone is most commonly performed for recurrent or chronic pharyngotonsillitis. Tonsillectomy has been shown to be effective in reducing the number of infections and the symptoms of chronic tonsillitis such as halitosis, persistent or recurrent sore throats, and recurrent cervical adenitis. In resistant cases of cryptic tonsillitis, tonsillectomy may be curative. Rarely in children, tonsillectomy is indicated for biopsy of a unilaterally enlarged tonsil to exclude a neoplasm. or to treat recurrent hemorrhage from superficial tonsillar blood vessels. Tonsillectomy has not been shown to offer clinical benefit over conservative treatment in children with mild symptoms.

There are large variations in surgical rates among children across countries: 144 in 10,000 in Italy; 115 in 10,000 in the Netherlands; 65 in 10,000 in England; and 50 in 10,000 in the United States. Rates are generally higher in boys. With the issuance of practice guidelines, these variations may decrease. The American Academy of Otolaryngology (AAO)–Head and Neck Surgery Taskforce on Clinical Practice Guidelines: Tonsillectomy in Children issued evidence-based guidelines in 2011 (Table 383-1).

Table 383-1 Paradise Criteria for Tonsillectomy

CRITERION	DEFINITION
Minimum frequency of sore throat episodes	At least 7 episodes in the previous year, at least 5 episodes in each of the previous 2 yr, or at least 3 episodes in each of the previous 3 yr
Clinical features	Sore throat plus at least 1 of the following features qualifies as a counting episode: Temperature of greater than 38.3°C (100.9°F) Cervical adenopathy (tender lymph nodes or lymph node size >2 cm) Tonsillar exudate Culture positive for group A β-hemolytic streptococcus
Treatment	Antibiotics administered in the conventional dosage for proved or suspected streptococcal episodes
Documentation	Each episode of throat infection and its qualifying features substantiated by contemporaneous notation in a medical record If the episodes are not fully documented, subsequent observance by the physician of 2 episodes of throat infection with patterns of frequency and clinical features consistent with the initial history*

*Allows for tonsillectomy in patients who meet all but the documentation criterion. A 12 mo observation period is usually recommended before consideration of tonsillectomy.

Adapted from Baugh RF, Archer SM, Mitchell RB, et al: American Academy of Otolaryngology–Head and Neck Surgery Foundation. Clinical practice guideline: tonsillectomy in children. Otolaryngol Head Neck Surg *144(1 Suppl):S8, 2011, Table 5.*

Adenoidectomy

Adenoidectomy alone may be indicated for the treatment of chronic nasal infection (chronic adenoiditis), chronic sinus infections that have failed medical management, and recurrent bouts of acute otitis media, including those in children with tympanostomy tubes who suffer from recurrent otorrhea. Adenoidectomy may be helpful in children with chronic or recurrent otitis media with effusion.

Adenoidectomy alone may be curative in the management of patients with nasal obstruction, chronic mouth breathing, and loud snoring suggesting sleep-disordered breathing.

Adenoidectomy may also be indicated for children in whom upper airway obstruction is suspected of causing craniofacial or occlusive developmental abnormalities.

Tonsillectomy and Adenoidectomy

The criteria for both tonsillectomy and adenoidectomy for recurrent infection are the same as those for tonsillectomy alone. The other major indication for performing both procedures together is upper airway obstruction secondary to adenotonsillar hypertrophy that results in sleep-disordered breathing, failure to thrive, craniofacial or occlusive developmental abnormalities, speech abnormalities, or, rarely, cor pulmonale. A high proportion of children with failure to thrive in the context of adenotonsillar hypertrophy resulting in sleep disorder experiences significant growth acceleration after adenotonsillectomy.

COMPLICATIONS

The 2 major complications of untreated GABHS infection are poststreptococcal glomerulonephritis and acute rheumatic fever.

Peritonsillar Infection

Peritonsillar infection can occur as either cellulitis or a frank abscess in the region superior and lateral to the tonsillar capsule. These infections usually occur in children with a history of recurrent tonsillar infection and are polymicrobial, including both aerobes and anaerobes. Unilateral throat pain, referred otalgia, drooling, and trismus are presenting symptoms. The affected tonsil is displaced down and medial by swelling of the anterior tonsillar pillar and palate. The diagnosis of an abscess can be confirmed by CT or by needle aspiration, the contents of which should be sent for culture.

Retropharyngeal Space Infection

Infections in the retropharyngeal space develop in the lymph nodes that drain the oropharynx, nose, and nasopharynx.

Parapharyngeal Space Infection

Tonsillar infection can extend into the parapharyngeal space, causing symptoms of fever, neck pain and stiffness, and signs of swelling of the lateral pharyngeal wall and neck on the affected side. The diagnosis is confirmed by contrast medium–enhanced CT, and treatment includes intravenous antibiotics and external incision and drainage if an abscess is demonstrated on CT. Septic thrombophlebitis of the jugular vein, Lemierre syndrome, manifests with fever, toxicity, neck pain and stiffness, and respiratory distress as a result of multiple septic pulmonary emboli and is a complication of a parapharyngeal space or odontogenic infection from Fusobacterium necrophorum.

Concurrent Epstein-Barr virus mononucleosis can be a predisposing event before the sudden onset of fever, chills, and respiratory distress in an adolescent patient. Treatment includes highdose intravenous antibiotics (ampicillin-sulbactam, clindamycin, penicillin, or ciprofloxacin) and heparinization.

CHRONIC AIRWAY OBSTRUCTION

Although rare, children with chronic airway obstruction from enlarged tonsils and adenoids can present with cor pulmonale.

The effects of chronic airway obstruction and mouth breathing on facial growth remain a subject of controversy. Studies of chronic mouth breathing, both in humans and animals, have shown changes in facial development, including prolongation of the total anterior facial height and a tendency toward a retrognathic mandible, the so-called adenoid facies. Adenotonsillectomy can reverse some of these abnormalities. Other studies have disputed these findings.

Tonsillectomy and Adenoidectomy

The risks and potential benefits of surgery must be considered (Table 383-3). Bleeding can occur in the immediate postoperative period or be delayed (consider von Willebrand disease) after separation of the eschar. The Clinical Guidelines for Tonsillectomy include a recommendation

for a single intravenous dose of intraoperative dexamethasone (0.5 mg/kg), which decreases postoperative nausea and vomiting and reduces swelling. There is no evidence that use of dexamethasone in postoperative tonsillectomy patients results in an increased risk of postoperative bleeding. Routine use of antibiotics in the postoperative period is ineffective and thus the American Academy of Otolaryngology Clinical Practice Guidelines advise against its use. Codeine is associated with excessive sedation and fatalities and is not recommended.

Swelling of the tongue and soft palate can lead to acute airway obstruction in the 1st few hr after surgery. Children with underlying hypotonia or craniofacial anomalies are at greater risk for suffering this complication. Dehydration from odynophagia is not uncommon in the 1st postoperative week. Rare complications include velopharyngeal insufficiency, nasopharyngeal or oropharyngeal stenosis, and psychologic problems.

Table 383-3 Risks and Potential Benefits of Tonsillectomy or Adenoidectomy or Both

RISKS
Cost*
Risk of anesthetic accidents
Malignant hyperthermia
Cardiac arrhythmia
Vocal cord trauma
Aspiration with resulting bronchopulmonary obstruction or infection
Risk of miscellaneous surgical or postoperative complications
Hemorrhage
Airway obstruction from edema of tongue, palate, or nasopharynx, or retropharyngeal hematoma
Central apnea
Prolonged muscular paralysis
Dehydration
Palatopharyngeal insufficiency
Otitis media
Nasopharyngeal stenosis
Refractory torticollis
Facial edema
Emotional upset
Unknown risks
POTENTIAL BENEFITS
Reduction in frequency of ear, nose, throat illness, and thus in
Discomfort
Inconvenience
School absence
Parental anxiety
Work missed by parents
Costs of physician visits and drugs
Reduction in nasal obstruction with improved
Respiratory function
Comfort
Sleep
Craniofacial growth and development
Appearance
Reduction in hearing impairment
Improved growth and overall well-being
Reduction in long-term parental anxiety

*Cost for tonsillectomy alone and adenoidectomy alone are somewhat lower.
Modified from Bluestone CD, editor: Pediatric otolaryngology, *ed 4, Philadelphia, 2003, WB Saunders, p. 1213.*