

**PHARYNGITIS (TONSILLOPHARYNGITIS)**

**Perspective and Principles of Disease**

Tonsillopharyngitis (from this point on referred to as pharyngitis) is an inflammatory syndrome of the oropharynx. Transmission is mainly through contact with respiratory secretions, but transmission through food and fomite contact is also possible. Although most cases of pharyngitis are uncomplicated and self-limited, the swelling may threaten airway patency or preclude ingestion of adequate liquids, thereby resulting in dehydration.

**Etiology**

Viruses are responsible for most cases of pharyngitis. Group A beta-hemolytic streptococcus (GABHS) is the most common bacterial cause of pharyngitis in children, with a peak incidence of 30%.1,2 Common causes of acute pharyngitis in adults are beta-hemolytic *Streptococcus*, *Mycoplasma pneumoniae*, and *Chlamydia pneumoniae*.1,6 Pharyngitis can also be caused by sexually transmitted diseases. Diphtheria is a potentially serious cause of pharyngitis.

Cultures obtained in cases of chronic or recurrent pharyngitis often grow mixed aerobic and anaerobic bacteria. Commonly isolated aerobic organisms include *Streptococcus aureus*, *Haemophilus influenzae*, and *Moraxella (Branhamella) catarrhalis*. The anaerobic bacteria most commonly isolated include *Bacteroides*, anaerobic gram-positive cocci, and *Fusobacterium*. β-Lactamase production is extremely common in bacteria responsible for chronic pharyngitis. Epstein-Barr virus (EBV) and *Actinomyces* are also implicated in chronic or recurrent pharyngitis. Rare causes of bacterial pharyngitis include *Francisella tularensis*, *Yersinia pestis*, and *Yersinia enterocolitica*.1,5

**Clinical Features**

The most common symptom is pharyngeal pain that is aggravated by swallowing and may radiate to the ears. Examination usually reveals fever, pharyngeal erythema, pharyngeal or tonsillar exudate, and tonsillar enlargement (Fig. 73-1). The infection tends to localize to lymphatic tissue and produces suppuration and swelling of the tonsils, along with tender cervical adenopathy. Occlusion of the eustachian tubes may result in secondary otitis media. Clinical differentiation of the etiologic organisms is virtually impossible.1,5

Viral pharyngitis usually occurs in conjunction with cough, rhinorrhea, myalgia, headache, stomatitis, conjunctivitis, exanthem, and odynophagia. Low-grade fever and pharyngeal exudates may be present. Cervical lymphadenopathy is generally absent.1,2 Mild pharyngeal edema and erythema associated with a “scratchy” throat are present in 50% of patients with the common cold. Systemic viral infections, including measles, cytomegalovirus (CMV), rubella, and human immunodeficiency virus (HIV), may initially manifest as mild pharyngitis.1,3 HIV and CMV pharyngitis may be clinically indistinguishable from infectious mononucleosis.1,2

Influenza occurs in epidemics and is associated with high fever, myalgia, and headache. Although 50 to 80% of patients with influenza experience pharyngeal discomfort, pharyngeal exudate and cervical lymphadenopathy are rare. Adenovirus may cause severe exudative pharyngitis with cervical adenitis similar to that in streptococcal pharyngitis. Thirty percent to 50% of cases of adenoviral pharyngitis are associated with a follicular, usually unilateral, conjunctivitis.1 Coxsackieviruses are the most frequent cause of hand-foot-and-mouth disease and herpangina.1,2

Pharyngitis is a common manifestation of infectious mononucleosis (caused by EBV) in young adults.1,3 Symptoms develop after an incubation period of 4 to 7 weeks. Fever and a tonsillar exudate or membrane (that is cheesy or creamy white) are often present. Cervical as well as generalized lymphadenopathy (90–100%) and splenomegaly (50%) are usually noted, and palatal petechiae may be present. Hepatomegaly is present in 10 to 15% of cases. Periorbital edema and rash are rare findings. In up to 90% of patients with mononucleosis who are inadvertently given ampicillin or amoxicillin, a diffuse macular rash develops that may be misdiagnosed as an allergic reaction.1,2

Patients with early (days to weeks) HIV infection can develop acute retroviral syndrome. This is manifested by fever, sore throat, generalized nontender lymphadenopathy, a diffuse maculopapular rash, arthralgias, mucocutaneous ulcerations, and, commonly, diarrhea. A nonexudative pharyngitis is present in 50 to 70% of patients. Oral thrush and ulcers may be present. Acute HIV infection can be differentiated from infectious mononucleosis by its more acute presentation, the absence of tonsillar hypertrophy or exudates, the frequent occurrence of rash, and the presence of oral ulcerations.1

Herd simplex also causes pharyngitis. These infections typically affect young adults. The presence of painful vesicles with erythematous bases is characteristic of herpes
pharyngitis. Ulcers may be present on the pharynx, lips, tongue, gums, and buccal mucosa. Pharyngeal erythema and exudate, fever, and tender lymphadenopathy are common for 1 to 2 weeks. In an immunocompromised host, large painful ulcers may be present. Herpes pharyngitis can be due to primary infection or reactivation. Concomitant bacterial superinfection may occur.1,2

GABHS pharyngitis is primarily a disease of children 5 to 15 years old and, in temperate climates, occurs in winter and early spring.1,2 It is responsible for less than 15% of cases of pharyngitis in patients older than 15 years and is rare in patients younger than 3 years. In epidemics, the incidence may double.1,2 GABHS pharyngitis is associated with sudden-onset sore throat, fever over 38.3°C (101°F), tonsillar erythema and exudates, palatal and uvular petechiae, uvular edema and erythema, and tender anterior cervical lymphadenopathy. Headache, nausea, vomiting, and abdominal pain may be present, especially in children. Cough, rhinorrhea, coryza, or other viral symptoms are usually absent. GABHS pharyngitis associated with a fine sandpaper erythematous rash that subsequently desquamates is termed scarlet fever. These findings, however, cannot be used to reliably diagnose or exclude streptococcal pharyngitis. Patients with recent exposure to others at risk for GABHS pharyngitis or in whom it has been diagnosed are more likely to become infected.1,2 Non–group A beta-hemolytic streptococcal species can cause pharyngitis indistinguishable from GABHS.1

Diphtheria is a potentially lethal cause of pharyngitis that is uncommon where adequate vaccinations are administered. U.S. serologic surveys indicate that a large percentage of adults and adolescents lack immunity to diphtheria toxin.5 Following a 2- to 4-day incubation period, patients develop malaise, sore throat, fever, and dysphagia. Examination early in the disease process may reveal pharyngeal erythema and isolated spots of gray or white exudate that later coalesce to form a pseudomembrane. This gray-green pseudomembrane is usually well demarcated and covers the nares, tonsils, soft palate, pharyngeal mucosa, and, occasionally, the uvula. The membrane may extend to involve the larynx and tracheobronchial tree, leading to hoarseness, cough, stridor, and airway obstruction. Tender and at times painful cervical lymphadenopathy may be found. Severe inflammation and edema can produce dysphonia and a characteristic “bull neck” appearance. Some strains of Corynebacterium diphtheriae produce a systemic toxin that may cause myocarditis, polyneuritis (at first autonomic and then peripheral), vascular collapse, diffuse focal organ necrosis, and death. Asymptomatic carriers may transmit the disease.1,2 Corynebacterium ulcerans is an animal pathogen passed on by consumption of raw milk that can produce infection indistinguishable from C. diphtheriae.

Aranobacterium haemolyticum (previously called Corynebacterium haemolyticum) typically affects the 10- to 30-year-old age group and can be indistinguishable from streptococcal pharyngitis. Most patients have an associated rash that may be scarlatiniform, urticarial, or erythema multiforme (occasionally skin manifestations may be the only complaint). Patients complain of a moderately severe sore throat and are usually non-toxic and afebrile. A. haemolyticum may cause a membranous pharyngitis that strongly mimics diphtheria; it is also associated with chronic tonsillitis.1,3

Anaerobic pharyngitis, or Vincent’s angina, is characterized by superficial ulceration and necrosis that often results in the formation of a pseudomembrane. Foul-smelling breath, odynophagia, submandibular lymphadenopathy, and exudate are often present. Patients typically have poor oral hygiene.3

Gonococcal pharyngitis is a sexually transmitted disease that may occur independently of genital infection. Those at highest risk are persons who practice receptive oral sex, especially men who have sex with men (in whom the incidence has been reported to be as high as 15%). Its severity is variable and it may result in an exudative or nonexudative pharyngitis. These differing manifestations can be explained in part by the lack of symptoms during the latent period of infection. Asymptomatic carriers are described, as is chronic and recurrent pharyngitis. Gonococcal pharyngitis is an important source of gonococcal meningitis.1,2 Syphilitic pharyngitis is a manifestation of primary or late (tertiary) syphilis and presents with painless mucosal ulcers. Chlamydia trachomatis pharyngitis is a sexually transmitted disease. Similar to gonococcal infection, C. trachomatis pharyngitis is associated with orogenital sex. Urogenital culturing is necessary along with treatment of sexual contacts. Patients are usually asymptomatic or may have only mild symptoms.1,2

Tuberculous pharyngitis usually occurs in patients with advanced disease. Symptoms and signs include hoarseness and dysphagia with pharyngeal ulcerations. Candidal pharyngitis is usually found in immunocompromised adults. Patients have dysphagia, odynophagia, and adherent white plaques with focal bleeding points.

Mycoplasma pneumoniae infection usually causes a mild pharyngitis. Mycoplasma infection occurs in epidemics and in crowded conditions and can be responsible for approximately 10% of cases of adult pharyngitis. Pharyngeal and tonsillar exudates, cervical lymphadenopathy, and hoarseness are common. Lower respiratory tract infection may also be present.1,3

Chlamydia pneumoniae pharyngitis resembles M. pneumoniae pharyngitis. It also occurs in epidemics or crowded conditions. Severe pharyngitis with laryngitis is suggestive of C. pneumoniae infection. Swelling and pain in the deep cervical lymph nodes may be prominent. Lower respiratory tract and concomitant sinusitis occur. The hallmarks of chlamydial pharyngitis are recurrence and persistence.1,2

Diagnostic Strategies
Monospot tests may be negative in up to 10% of patients with infectious mononucleosis, especially in the early stages of the illness. Immunoglobulin M (IgM) antibodies to EBV capsid antigen develop in 100% of cases. EBV nuclear antigens develop within 3 to 6 weeks and are useful if an initial negative test becomes positive at a later date. Peripheral blood smears demonstrate atypical mononuclear cells in 75% of patients, with the peak incidence occurring in the second to third week of illness.1,2 Herpes pharyngitis may be diagnosed by culture.
cytopathologic tests on scrapings of lesions, and serologic tests. Enzyme-linked immunosorbent assay testing for HIV can be falsely negative during the first 3 to 4 weeks of illness. During this period of time, quantitative assays for plasma RNA should be performed. 

Diagnosis of GABHS infection is important to prevent complications, particularly rheumatic fever. Even the most experienced practitioner has difficulty clinically diagnosing streptococcal pharyngitis. Several authors propose scoring systems based on clinical findings, but the only valid method of determining acute GABHS infection is by acute and convalescent antistreptolysin O titers, which is not practical in the emergency department. A single throat culture has a sensitivity of 90 to 95% in detecting Streptococcus pyogenes in the pharynx. Variables that affect the accuracy of throat cultures include collection and culturing technique, as well as the recent use of antibiotics.

Rapid diagnostic tests for GABHS detect streptococcal antigens. Rapid streptococcal tests (RSTs) have a reported specificity of 70 to 100% (with most being >95%) and a sensitivity of 31 to 100% (with most being 60–95%). Sensitivity and specificity in actual practice are lower than in controlled trials. Patients with positive cultures or RSTs may actually be carriers who may not need treatment and are at low risk for transmission and complications. The use of RSTs in patients without clinical findings consistent with GABHS may increase false-positive results. A positive RST seems to reliably indicate the presence of S. pyogenes in the pharynx. In contrast, RSTs are often negative in the setting of pharyngitis with a low bacterial count (these patients are still at risk for complications, including rheumatic fever). It is recommended that a negative RST in a child be followed by a confirmatory culture.

Diagnosis and treatment of GABHS in adults is controversial and the subject of two expert panel recommendations. It is agreed that antibiotics are overused in the treatment of pharyngitis, the use of clinical criteria in conjunction with RSTs improves the accuracy of RSTs, adults with negative RSTs do not require confirmatory cultures (because of the lower incidence of GABHS infection and the extremely low risk for complications), and neither testing nor antibiotic treatment should be used in patients who are clinically at low risk for GABHS infection. Both panels agree that the most useful clinical criteria for determining GABHS pharyngitis are the Centor criteria (Box 73-1).

The position of the Infectious Disease Society of America is that a positive throat culture or RST in addition to clinical symptoms and signs is needed to confirm the diagnosis of GABHS pharyngitis. The society stresses that clinical criteria alone are appropriate to determine which patients do not need testing but are insufficient, without bacterial confirmation, to diagnose GABHS pharyngitis.

Though controversial, we recommend that: Patients with one or none of the Centor criteria be not treated or treated. Treat patients with all four criteria without testing. Perform RSTs in patients with two or three criteria, and treat only those who have positive results (Box 73-2).

These recommendations apply only to immunocompetent patients with no underlying comorbid conditions or a history of rheumatic fever. They do not apply in settings of outbreaks of GABHS infection or rheumatic fever, nor are they appropriate in situations in which the endemic rate of rheumatic fever is higher than that in the United States. It is important to consider local epidemics and be prepared to revise the approach to treatment if evidence of GABHS infection or complications exists. Non–group A streptococcal pharyngitis should also be treated, since the same suppurative complications occur as with group A. Pharyngitis caused by other treatable organisms must also be considered and is associated with serious complications. Confirmation of diphtheria requires culturing on the proper media and immunologic testing (polymerase chain reaction). Toxicogenicity testing must also be performed. The diagnosis of A. haemolyticum infection should be considered if rash, including erythema multiforme, accompanies pharyngitis. The diagnosis of Vincent’s angina is based on clinical findings and Gram’s stain. In cases of possible gonococcal infection, a sample should be plated on Thayer-Martin agar. Tuberculous pharyngitis is diagnosed by acid-fast staining. Syphilitic pharyngitis is diagnosed with darkfield microscopy, direct immunofluorescence, and serologic testing. Candidal pharyngitis is diagnosed by noting yeast on potassium hydroxide preparations of throat swabs or Sabouraud’s agar. The diagnosis of mycoplasmal pharyngitis can be confirmed serologically or by culture. Rapid antigen tests for Mycoplasma are available. Chlamydial pharyngitis can be diagnosed by serologic testing, by culture, or by antigen detection tests. Studies of patients with chronic pharyngitis find that surface cultures do not correlate well with the causal pathogens, which are often concealed within the tonsillar crypts.

### Differential Considerations

The differential diagnosis of adult pharyngitis includes deep space infections, tumors, foreign bodies, pemphigus, Stevens-Johnson syndrome, drug reactions, allergic reactions, uvulitis, angioneuropathic edema, chemical and thermal burns, esophagitis, gastroesophageal reflux disease, crioarytenoid arthritis, thyroiditis, and epiglottitis.

### Management

Patients with pharyngitis should be treated symptomatically with topical anesthetic rinses or lozenges and with acetaminophen or ibuprofen. Oral hydration and saltwater gargles are helpful. Most cases of pharyngitis are self-limited and follow a benign course.

Treatment of infectious mononucleosis is supportive (see Chapter 128). Patients should avoid contact sports for 6 to 8 weeks to minimize the small risk of splenic rupture. Corticosteroids are indicated for patients with tonsillar hypertrophy that threatens airway patency, severe thrombocytopenia, or hemolytic anemia. Acyclovir, valacyclovir, or famciclovir is indicated in immunocompromised patients with herpetic pharyngitis and may be beneficial in the treatment of acute

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### BOX 73-1

**CENTOR CRITERIA FOR DETERMINING GROUP A BETA-HEMOLYTIC STREPTOCOCCAL PHARYNGITIS**

- Tonsillar exudates
- Tender anterior lymphadenopathy or lymphadenitis
- Absence of cough
- History of fever

### BOX 73-2

**CENTOR CRITERIA SCORING FOR DETERMINING TESTING AND TREATMENT FOR GROUP A BETA-HEMOLYTIC STREPTOCOCCAL PHARYNGITIS**

<table>
<thead>
<tr>
<th>Centor Score</th>
<th>Testing/Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–1</td>
<td>None</td>
</tr>
<tr>
<td>2–3</td>
<td>Treatment based on results of RST</td>
</tr>
<tr>
<td>4</td>
<td>Treat without testing</td>
</tr>
</tbody>
</table>
PART III

■ Pulmonary System

Treatment of streptococcal pharyngitis leads to a 13% earlier duration of disease. When diphtheria is strongly suspected on the basis of clinical findings, treatment must begin immediately. Airway collapse may occur suddenly and without warning. The mainstay of therapy is antitoxin (a horse serum product), that should be administered immediately on clinical suggestion of diphtheria. The dose of antitoxin varies widely and depends on the site of infection and the duration of symptoms. Antibiotics have little effect on the resolution of systemic toxicity, but they are useful in eradicating C. diphtheriae infection and preventing transmission. Infected patients should remain in strict isolation to prevent transmission. The antibiotic of choice is penicillin G for 5 days, followed by penicillin VK for 5 days, or erythromycin, 500 mg four times a day for 10 days. A small percentage of patients require an additional 10-day course of erythromycin for persistent infection. Rifampin, 600 mg/day for 10 days, is also effective in eradicating the carrier state of C. diphtheriae and treating erythromycin-resistant diphtheria. Diphtheria toxoid should be administered during convalescence and to unvaccinated close contacts.1,2,8

A. haemolyticum may be resistant to penicillin. Erythromycin, 250 mg orally four times a day for 10 days, is the treatment of choice.1,2 Vincent’s angina is treated with penicillin or clindamycin and rinses with an oral oxidizing agent (hydrogen peroxide).8 Gonococcal pharyngitis is often more difficult to eradicate than genital infections. Treatment is similar to that for gonococcal urethritis and consists of ceftriaxone (125 mg intramuscularly) or single-dose oral treatment with either ciprofloxacin (500 mg), ofloxacin (400 mg), or gatifloxacin (400 mg). Concomitant treatment of chlamydial infection with a single oral dose of 1 g of azithromycin or doxycycline, 100 mg orally twice a day for 7 days, is also recommended.2,8 Tuberculous pharyngitis is seen with disseminated disease. Patients should be isolated and treated with a multidrug regimen. Pharyngitis caused by primary syphilis is treated with 2.4 million U of benzathine penicillin (long-acting), with 14 days of tetracycline or doxycycline used as an alternative. Candidal pharyngitis is treated with systemic fluconazole or itraconazole. Alternative therapy includes nystatin (suspension or tablets) or oral clotrimazole for 14 days. Chronic suppression therapy with ketoconazole, clotrimazole, or fluconazole is usually required for HIV pharyngitis.8

M. pneumoniae is treated with erythromycin, tetracycline, or doxycycline for 7 to 14 days.2,8 Chlamydial pharyngitis is treated with doxycycline, trimethoprim-sulfamethoxazole, or a macrolide antibiotic. C. pneumoniae pharyngitis should be treated for 7 to 10 days to prevent treatment failure and recurrence. C. trachomatis pharyngitis may require prolonged or repeated courses of antibiotics.2,8

Treatment of recurrent or chronic tonsillitis should include β-lactamase-resistant antibiotics active against aerobic and anaerobic organisms. Choices include oral cephalosporins, amoxicillin-clavulanic acid, penicillin with rifampin or metronidazole, or clindamycin.2

Steroids given in conjunction with oral antibiotics in adults with acute pharyngitis may significantly shorten the duration of symptoms and provide a greater degree of pain relief without increasing complications. Oral (40-60 mg of prednisone per day for 1-5 days) or intramuscular (a single dose of 10 mg of dexamethasone) have been found to be equally effective.3,10

Disposition

Although most cases of pharyngitis follow a benign course, life-threatening complications can occur. Airway compromise from tonsillar enlargement, local and distant spread of infection, deep neck abscesses, necrotizing fasciitis, sleep apnea, bacteremia, sepsis, and death are reported.1,3

The use of antiretrovirals is indicated in acute HIV infection.1 Although many studies focus on GABHS pharyngitis, proper treatment of nonstreptococcal pharyngitis can also avoid serious complications. Because clinical judgment is insufficient and rapid diagnostic tests are not always accurate and diagnose only GABHS, this disease process is often treated empirically. The choice of antibiotic for the empirical treatment of adult pharyngitis is not fully elucidated. It is unclear how effective antibiotics are in uncomplicated cases of non-GABHS pharyngitis in adults. Antibiotics may modestly shorten the course of the disease process, but they are also associated with increased recurrence, increased bacterial drug resistance, decreased immune response, and patient expectations for antibiotics with subsequent episodes of pharyngitis.1,3-6

GABHS pharyngitis in children and adolescents must be treated adequately (within 9 days) to prevent rheumatic fever. The incidence of rheumatic fever parallels that of GABHS and has markedly diminished with the use of antibiotics. Patients with mild cases of GABHS pharyngitis may develop rheumatic fever. Current estimates show that rheumatic fever complicates 0.5% of cases of GABHS pharyngitis, but in epidemics the incidence increases to 3%. More troubling is an increase in sporadic outbreaks of rheumatic fever.5,3 The incidence and course of poststreptococcal glomerulonephritis caused by nephritogenic strains are unaffected by antibiotic therapy.1,3-5

Antibiotic therapy is extremely effective in eradicating GABHS and its other complications. Untreated, GABHS pharyngitis is a self-limited illness that lasts 3 to 4 days. Early antibiotic treatment of streptococcal pharyngitis leads to a 13% earlier resolution of symptoms and shortens the course of illness by about 1 day. Antibiotic therapy also decreases transmission, and patients are no longer infectious after 24 hours of antibiotic treatment.1,3-6

The antibiotic regimen of choice in adults for GABHS pharyngitis is either a single intramuscular injection of 1.2 million U of benzathine penicillin or a 10-day course of penicillin V, 500 mg orally twice a day. Less frequent dosing is less effective in preventing rheumatic fever.1,3-6 Intramuscular penicillin may be more effective than oral penicillin and ensures compliance, but allergic reactions are more severe as a result of pro-caine allergy, and treatment is more expensive. Penicillin failure usually reflects noncompliance, reinfection, or the presence of β-lactamase-producing organisms. Erythromycin is recommended for patients who are allergic to penicillin. A 1-g total daily dose must be given for 10 days, but dosing intervals of two, three, and four times a day are equally effective in preventing rheumatic fever.1,3-5 Cephalosporins or clindamycin are also acceptable for penicillin-allergic patients. Once-daily amoxicillin therapy may be effective in children.1,7 Oral cephalosporins or clindamycin may be more effective than penicillin in eradicating GABHS pharyngitis and some authors argue for their use as first-line agents.7 These alternative regimens should be used for patients not responding to penicillin or unable to tolerate either penicillin or erythromycin.1,3-6,7

Patients whose symptoms return within a few weeks of treatment may have been noncompliant with oral therapy or may have acquired a new infection (at times from asymptomatic close contacts). Evaluation and treatment should be similar to that of the first episode, with consideration given to treatment with intramuscular penicillin. Further recurrences mandate more extensive evaluation. Pharyngeal cultures should be obtained and consideration given to evaluating and treating close contacts for GABHS infection.5

Successful treatment of diphtheria is inversely related to the duration of disease. When diphtheria is strongly suspected on the basis of clinical findings, treatment must begin immedi-
Infectious mononucleosis may lead to hepatic dysfunction, splenic injury, neurologic disorders, pneumonitis, pericarditis, and hematologic disorders, including thrombocytopenia and hemolytic anemia. Complications of GABHS pharyngitis are both suppurative and non-suppurative. Suppurative complications include peritonsillar abscess, deep space abscesses, suppurative cervical lymphadenitis, otitis media, sinusitis, mastoiditis, bacteremia, sepsis, osteomyelitis, empyema, meningitis, and soft tissue infections. Non-suppurative complications include scarlet fever, rheumatic fever, post-streptococcal glomerulonephritis, non-rheumatic perimyocarditis, erythema nodosum, and streptococcal toxic shock syndrome. In contrast to rheumatic fever, other complications of GABHS pharyngitis are increasing in incidence and severity. A chronic carrier state of streptococcal infection exists and can persist for several months despite treatment. These patients are asymptomatic, at low risk for rheumatic fever, and not considered highly contagious. Non-group A streptococcal pharyngitis may be complicated by the same suppurative complications as group A infections. Scarlet fever and acute glomerulonephritis, but not rheumatic fever, are linked to group C and G pharyngitis.

**LINGUAL TONSILLITIS**

Lingual tonsillitis is a rarely diagnosed cause of pharyngitis that predominantly occurs in patients who have had palatine tonsils removed. The lingual tonsils are a collection of noncapsulated lymphoid tissue most commonly (size and location are highly variable) located symmetrically on either side of the midline just below the inferior pole of the palatine tonsils and anterior to the vallecula at the base of the tongue. This lymphoid tissue may enlarge after puberty, repeated infection, and tonsillectomy. Patients with lingual tonsillitis have a sore throat that worsens with movement of the tongue (including tongue depression) and phonation. The patient may have a classic “hot potato” voice (the muffled voice one has when eating very hot food) and complain of feeling a swelling in the throat. Dysphagia, fever, respiratory distress, and stridor may be present. Chronic or recurrent lingual tonsillitis may also cause a chronic cough or sleep apnea. Physical findings often include a normal-appearing pharynx with mild hyperemia. Direct or indirect laryngoscopy reveals an edematous lingual tonsil covered with a purulent exudate. Lateral soft tissue neck films aid in the diagnosis. These films demonstrate a tonsil covered with a purulent exudate. Management includes maintenance of airway patency, antibiotics, and supportive therapy. Rarely, acute lingual tonsillitis may be a life-threatening condition. Airway management includes warmed humidified oxygen, hydration, and corticosteroids. Nebulized epinephrine can relieve the acute respiratory distress and stridor. Antibiotics of choice are similar to those used for the treatment of pharyngitis.

**LARYNGITIS**

Laryngitis is manifested as hoarseness and aphony. It is usually caused by viral upper respiratory tract infections. In up to 10% of cases, bacteria (including streptococci and diphtheria) may be responsible. Other infectious causes include tuberculosis, syphilis, leprosy, actinomycosis, and fungal infections. These patients should be evaluated for epiglottitis. Noninfectious causes include tumors, caustic or thermal injuries, trauma, and esophageal reflux disease. Antibiotics are not indicated unless signs of bacterial infection are present. Steroids may hasten resolution of symptoms.

**ADULT EPIGLOTTITIS**

**Perspective**

Adult epiglottitis can lead to rapid, unpredictable airway obstruction. Before the introduction of *H. influenzae* vaccine, epiglottitis was primarily a pediatric disease. Although the incidence of pediatric epiglottitis has diminished, there is an increase in adult epiglottitis. Whether the increase is due to increased recognition or prevalence is unknown.

**Principles of Disease**

Adult epiglottitis is a localized cellulitis involving the supraglottic structures, including the base of the tongue, vallecula, aryepiglottic folds, arytenoid soft tissues, lingual tonsils, and the epiglottis. Inflammation does not extend to the infraglottic regions. Some adults have a normal epiglottis in the setting of severe supraglottic involvement. The term *supraglottitis* is a more accurate description of this disease process. Adults with epiglottic involvement are prone to epiglottic abscesses.

The most commonly isolated bacterial pathogen causing adult epiglottitis is *H. influenzae*, but it is only isolated from a minority of affected patients. *H. influenzae* infection is associated with a more aggressive disease course. In many cases, no organisms can be cultured from either blood or the supraglottic structures, which suggests that respiratory viruses may play an important etiologic role. The predominant organisms isolated from epiglottic abscesses are *Streptococcus* and *Staphylococcus* species. Adult epiglottitis may also result from thermal injury.

**Clinical Features**

Adult epiglottitis has no age or seasonal prevalence. Males and smokers are more commonly affected. Adults with epiglottitis typically experience a prodrome resembling that of a benign upper respiratory tract infection. The duration of the prodrome is usually 1 to 2 days but may be as long as 7 days or as short as several hours. Patients who have a rapid onset of the disease as well as those with comorbid conditions (especially diabetics) are more likely to require airway intervention.

Patients typically have dysphagia, odynophagia, and a sore throat. Pharyngeal pain may be severe and is often disproportionate to the clinical findings. Dysphonia and a muffled voice...
are common, while hoarseness is unusual. Fever is absent in up to 50% of cases and may develop only in the later stages of the disease. Tachycardia disproportionate to fever correlates with severe disease. Tenderness to palpation of the anterior aspect of the neck in the region of the hyoid and when moving the larynx side to side is a reliable finding in epiglottitis. Ear pain may be a manifestation of adult epiglottitis.

Concomitant uvulitis, pharyngitis, tonsillitis, Ludwig's angina, peritonsillar abscess, and parotitis can occur; therefore, these findings on pharyngeal examination do not exclude the diagnosis of epiglottitis. The classic symptoms and signs of imminent airway obstruction may not appear until immediately before complete obstruction occurs, thus the earlier signs of drooling and dysphonia are more compelling indicators of impending airway compromise. Patients who assume a classic sniffing position are at imminent risk for rapid airway obstruction. These patients should not be laid flat, and immediate preparations must be made to rapidly secure the airway (see Chapter 1).1,16,17

Diagnostic Strategies

Although severe cases of adult epiglottitis are easily recognized, a large number of less severe cases are initially misdiagnosed. In up to a third of adult patients, epiglottitis is present but not diagnosed within 48 hours of admission.4

Adult patients without respiratory distress should undergo fiberoptic or rigid direct laryngoscopy, or indirect laryngoscopy, but preparations should include the ability to provide immediate bag-mask ventilation, intubation, or cricothyrotomy. Laryngospasm and complete obstruction can occur during instrumentation of the inflamed airway. Flexible fiberoptic laryngoscopy is the preferred approach as it provides direct, minimally invasive examination of the upper airway and can be used to determine the need for, and imminence of, airway management. Laryngoscopy reveals a swollen epiglottis and surrounding structures (Fig. 73-3). The epiglottis may appear “cherry red” but is often pale and edematous. In patients with respiratory distress, drooling, aphonias, or stridor, indirect laryngoscopy is contraindicated and direct laryngoscopy should be undertaken only as part of a “double setup” with the ability to proceed immediately to cricothyrotomy.1,17

Lateral cervical soft tissue radiographic films have a sensitivity of up to 90% compared with direct laryngoscopy; however, normal soft tissue plain films do not exclude mild or moderate adult epiglottitis. Adults with possible epiglottitis and normal soft tissue radiographic films should undergo laryngoscopy as described earlier. Radiologic findings include obliteration of the vallecula, swelling of the arytenoids and aryepiglottic folds, edema of the prevertebral and retropharyngeal soft tissues, and “ballooning” of the hypopharynx and mesopharynx. The edematous epiglottis appears enlarged and thumb-shaped (Fig. 73-4). An epiglottic width greater than 8 mm or an aryepiglottic fold width greater than 7 mm is suggestive of epiglottitis.

Differential Considerations

Adult epiglottitis is often misdiagnosed as streptococcal pharyngitis. Other entities that must be considered include mononucleosis, deep space abscesses, lingual tonsillitis, diphtheria, pertussis, and croup. Noninfectious considerations include angioedema, allergic reactions, foreign body aspiration, laryngospasm, tumors, toxic inhalation or aspiration, and laryngeal trauma.

Management

Most adults with epiglottitis do not require intubation, but all patients with epiglottitis should be treated with extreme care because of the possibility of unpredictable sudden airway obstruction. Endotracheal intubation should always be performed under direct visualization. Awake fiberoptic intubation is the optimal method, but awake orotracheal intubation by direct laryngoscopy is also safe and effective.1,17 Blind nasotracheal intubation can lead to airway obstruction and is contraindicated in the setting of epiglottitis.
Antibiotics should be initiated against *H. influenzae* and other likely bacterial pathogens. First-line agents pending culture and sensitivity results are cefotaxime and ceftriaxone. Alternative antibiotics include ampicillin-sulbactam and trimethoprim-sulfamethoxazole.1,8,9 The role of steroids and racemic epinephrine is unresolved.

**Disposition**

Stable patients, particularly those who present more than 24 hours after onset of symptoms, who are without respiratory distress, and who are handling their secretions can be safely observed without intubation in the emergency department observation unit or a higher level inpatient unit (intermediate or intensive care unit). Such patients include those with mild swelling on laryngoscopy and without drooling, stridor, or dyspnea. Patients who have a rapidly progressive course, are immunocompromised or diabetic, or have an epiglottic abscess or significant epiglottic enlargement on plain film study or laryngoscopy are at high risk.1,16,17

Extraepiglottic infections are less likely to occur in adults than children. Meningitis, retropharyngeal abscesses, pneumonia, sepsis, acute respiratory distress syndrome, necrotizing fasciitis, mediastinitis, and pulmonary edema occur in conjunction with epiglottitis.

**DEEP SPACE INFECTIONS OF THE LOWER PART OF THE FACE AND NECK**

Patients with deep space infections of the head and neck (Fig. 73-5) can decompensate rapidly. The incidence and complications of deep space infections have decreased dramatically because of improved dental hygiene and the advent of antibiotics.18

The submandibular space comprises two spaces: the sublingual and submaxillary spaces. The submandibular space is involved in Ludwig’s angina.19 Five potential communicating spaces in the neck are clinically relevant: the peritonsillar, parapharyngeal, retropharyngeal, “danger,” and prevertebral spaces. The parapharyngeal space contains the carotid artery, the jugular vein, the cervical sympathetic chain, and cranial nerves IX through XII. The retropharyngeal space lies in the midline (medial to the parapharyngeal space) and extends from the base of the skull to the superior mediastinum (at about the level of T2). Retropharyngeal abscesses tend to occur lateral to the midline. Posterior to the retropharyngeal space lies the “danger space,” which extends from the base of the skull to the diaphragm. The prevertebral space extends from the base of the skull to the coccyx. Danger space and prevertebral abscesses are located in the midline. Infections in the retropharyngeal, danger, and prevertebral spaces easily access the mediastinum.18,20

The primary pathologic process of deep space infection is regional cellulitis. The fasciae may confine infections within their boundaries, thereby leading to abscess formation. Infections are most commonly a polymicrobial disease of mixed aerobic-anaerobic bacteria of oral origin. The most frequently isolated organisms are streptococci, staphylococci, and *Beta
trooides* species. β-Lactamase-producing organisms are isolated in up to two thirds of cases. Other organisms include *H. influenzae*, *Pseudomonas aeruginosa*, *Klebsiella* species, and *Candida albicans*.18,19,21

Computed tomography (CT) and magnetic resonance imaging (MRI) can help distinguish cellulitis from abscess formation and guide therapy. Patients with cellulitis usually respond well to high-dose antibiotic therapy. Patients with small abscesses may be successfully treated with high-dose intravenous antibiotics or needle aspiration. The presence of an abscess, however, usually requires surgical incision and drainage. With the exception of patients with uncomplicated peritonsillar abscesses, patients with deep space abscess usually require admission, intravenous (IV) antibiotics, and consultation with otolaryngologists for possible surgical intervention.22

There is little anatomic resistance to spread of infection within the fascial planes and spaces, which allows rapid spread of infection. Life-threatening complications can occur rapidly.

Airway distortion and trismus may complicate intubation attempts. Neuromuscular blockade is generally ill-advised, unless as part of a “double setup” with the ability to proceed directly to cricothyrotomy, because both intubation and bag-mask ventilation may be impossible. Awake techniques are preferable. Fiberoptic-guided intubation can be useful in this setting.18,19,22 Blind nasotracheal intubation can cause abscss rupture and further compromise and is thus contraindicated.19,23 Should it be necessary to secure an airway surgically, cricothyroidotomy is generally the procedure of choice, except in some cases of Ludwig’s angina, in which anatomic distortion may necessitate tracheostomy.

**PERITONSILLITIS (PERITONSILLAR CELLULITIS AND PERITONSILLAR ABSCESS)**

**Perspective**

Peritonsillar cellulitis and abscess should be regarded as the clinical continuum of peritonsillitis. Peritonsillar abscess, also termed *quinsy*, is the most common deep infection of the head and neck in adults.

**Principles of Disease**

Peritonsillitis may occur as a result of acute tonsillitis. Infection in either Weber’s glands or the tonsillar crypts invades the peritonsillar tissues and thereby leads to cellulitis and abscess formation. Fibrous fascial septae divide the peritonsillar space into compartments and direct the infection anteriorly and superiorly.1,18
Dental infections, chronic tonsillitis, infectious mononucleosis, smoking, chronic lymphocytic leukemia, and tonsilloliths are predisposing factors. Peritonsillar abscess occurs in patients who have undergone complete tonsillectomy and is seen in all age groups. Peritonsillitis recurs in up to 50% of patients, with the incidence of recurrent peritonsillar abscess of approximately 10%. The highest incidence of recurrence is seen in patients younger than 40 years and in those with a history of chronic tonsillitis.

Most peritonsillar abscesses are polymicrobial. In patients who have received antibiotics in whom peritonsillar abscesses develop, fewer aerobes and more β-lactamase-producing organisms are isolated.1,18

Clinical Features

There is often a delay of 2 to 5 days between abscess formation and local and systemic symptoms. Symptoms and signs include odynophagia, dysphagia, drooling, trismus, and referred otalgia. Patients may have a characteristic muffled, hot potato voice and rancid breath. Systemic manifestations include fever, malaise, and dehydration. Patients often relate a history of recurrent tonsillitis with multiple trials of antibiotics but without resolution.

The examination of the pharynx may be limited by trismus. Physical findings of peritonsillitis include inflamed and erythematous oral mucosa, purulent tonsillar exudates that obscure the tonsil, and tender cervical lymphadenopathy. Peritonsillar cellulitis mimics peritonsillar abscess. Peritonsillar abscess is characterized by a greater frequency of drooling, trismus, and dysphagia, whereas peritonsillar cellulitis is more commonly bilateral. The distinguishing feature of peritonsillar abscess is inferior medial displacement of the infected tonsil (at times involving the soft palate), with contralateral deviation of the uvula (Fig. 73-6). The abscess is generally unilateral and located in the superior pole of the tonsil. Bilateral peritonsillar abscesses occur occasionally.1,18

Diagnostic Strategies

Aspiration of pus establishes the diagnosis of peritonsillar abscess. Because patients with peritonsillar abscess have a 20% incidence of mononucleosis, laboratory testing for mononucleosis should be considered.

Roentgenographic examination in uncomplicated cases contributes little to the diagnosis. Contrast-enhanced CT and ultrasonography (both intraoral and transcutaneous) aid in differentiating peritonsillar abscess from cellulitis, especially when patients are unable to cooperate with needle aspiration. These modalities are also useful in diagnosing posteriorly and inferiorly located abscesses and in guiding needle aspiration.18,22,24

Differential Considerations

The differential diagnosis of peritonsillitis includes hypertrophic tonsillitis, infectious mononucleosis, tubercular granuloma, diphtheria, other deep space infections of the neck, cervical adenitis, congenital or traumatic internal carotid artery aneurysms, foreign bodies, and neoplasms.

Management

Emergency abscess aspiration is necessary in cases of complete or impending airway obstruction. Antibiotics alone may control peritonsillar cellulitis. Regimens include high-dose penicillin plus metronidazole, cefoxitin, ampicillin-sulbactam, and clindamycin. Alternative antimicrobial agents include a carbapenem, high-dose penicillin and rifampin, ticarcillin-clavulanate, or piperacillin-tazobactam.1,8,18 β-Lactamase-producing bacteria and poor penetration of antibiotics into the abscess limit the effectiveness of antibiotics. The use of steroids may be beneficial.25

Drainage of the abscess is curative. Needle aspiration of abscesses by both emergency physicians and otolaryngologists is diagnostic (although false-negative aspirations occur in approximately 10% of cases, and another 10% may require repeated aspirations) and therapeutic. This immediately relieves symptoms and is more cost-effective, less painful, and easier to perform than incision and drainage.1,18,24,25 Intraoral ultrasound-guided needle aspiration is a useful adjunct in the presence of trismus.24,25 Immediate tonsillectomy under general anesthesia may be needed in extremely young or uncooperative patients.

Disposition

Hospital admission is indicated for patients who have underlying disease, are dehydrated, appear toxic, are unable to tolerate oral fluids, are in severe pain, or have other significant complications. The most dangerous immediate complication of peritonsillitis is pharyngeal obstruction with upper airway compromise. Other complications include sepsis, abscess rupture and pulmonary aspiration leading to pneumonia, empyema, and pulmonary abscess formation. Infection can spread contiguously to the parapharyngeal and retropharyngeal spaces. Ludwig’s angina, mediastinal involvement (including mediastinitis, pneumonia, empyema, and pericarditis), myocarditis, carotid artery erosion, jugular vein thrombophlebitis, septic embolization, abscess formation, Lemierre’s syndrome (postanginal sepsis), and cervicopharyngeal necrotizing fasciitis can complicate peritonsillitis. Intracranial extension of peritonsillitis may result in meningitis, cavernous sinus thrombosis, and cerebral abscess.18

**Figure 73-6.** Peritonsillar abscess with uvular displacement to the right.

**LUDWIG’S ANGINA**

Perspective and Principles of Disease

Ludwig’s angina is a potentially fulminant disease process that can lead to death within hours.19,21,26 This is a progressive cellulitis of the connective tissues of the floor of the mouth and neck that begins in the submandibular space. Dental disease is the most common cause of Ludwig’s angina. An infected or recently extracted lower molar is noted in most affected
patients. Dentoalveolar abscesses easily break through the relatively thin cortex of the mandible below the mylohyoid ridge and infect the submandibular space. Other causes of Ludwig’s angina include a fractured mandible, foreign body or laceration in the floor of the mouth, tongue piercing, traumatic intubation and bronchoscopy, secondary infections of an oral malignancy, osteomyelitis, otitis media, submandibular sialadenitis, peritonsillar abscess, a furuncle, infected thyroglossal cyst, and sepsis.

Clinical Features

Infection of the sublingual and submaxillary spaces leads to edema and soft tissue displacement, which may result in airway obstruction. The most common presentation in patients with Ludwig’s angina includes dysphagia, odynophagia, neck swelling, and neck pain. Other symptoms and signs include dysphonia, a hot potato voice, dysarthria, drooling, tongue swelling, pain in the floor of the mouth, restricted neck movement, and sore throat. Patients should be questioned regarding recent dental extraction and disease. A foul taste in the patient’s mouth, feeling air release at the time of extraction, rapid development of crepitus, and unilateral pharyngitis in patients with recent dental extractions should suggest the diagnosis of Ludwig’s angina.

The most common physical findings in Ludwig’s angina are bilateral submandibular swelling and elevation or protrusion of the tongue. Other findings include elevation of the floor of the mouth, posterior displacement of the tongue, and a “woody” consistency of the floor of the mouth. The combination of tense edema and brawny induration of the neck above the hyoid may be present and is described as a “bull neck.” Marked tenderness to palpation of the neck and subcutaneous emphysema may be noted. Trismus and fever are usually present, but generally no palpable fluctuance or cervical lymphadenopathy. Tenderness to percussion may be elicited over the involved teeth.

Diagnostic Strategies

The diagnosis is made clinically by examination. Soft tissue plain films of the neck may confirm the diagnosis by showing swelling of the affected area and airway narrowing and by identifying gas collections. CT and MRI aid in the diagnosis of Ludwig’s angina and its complications. Ultrasonography is also useful in diagnosing abscesses and edema in the setting of Ludwig’s angina.

Differential Considerations

The differential diagnosis includes deep cervical node suppuration, peritonsillar and other deep neck space abscess, parotid and submandibular gland abscess, oral carcinoma, angioedema, submandibular hematoma, and laryngeal diphtheria.

Management

Sudden asphyxiation is the most common cause of death in patients with Ludwig’s angina. Stridor, tachypnea, dyspnea, inability to handle secretions, and agitation are all indications of impending airway compromise. Fiberoptic-guided oral or nasotracheal intubation under sedation with topical anesthesia is the preferred method of airway control. Endotracheal intubation may be difficult because of distortion of the upper airway, trismus, pooled secretions, the cephalad and posterior displacement of the tongue, inability to displace the tongue into the submandibular space, and a tendency for the development of laryngospasm. Cricothyrotomy may be difficult and opens tissue planes that increase the risk of spreading infection into the mediastinum, but is the procedure of choice if fiberoptic intubation is not available.

Emergent antibiotic regimens are similar to those for peritonsillar abscess. The value of corticosteroids in the setting of Ludwig’s angina is unclear. With the exception of dental extractions, surgery is reserved for patients who do not respond to medical therapy and those with crepitus and purulent collections.

Disposition

The mortality rate resulting from Ludwig’s angina is less than 10% with early aggressive antibiotic therapy and adequate protection of the airway. Infection can easily spread into other deep spaces of the neck and into the thoracic cavity and cause empyema, mediastinitis, mediastinal abscess, and pericarditis. Aspiration may lead to pneumonia and the formation of lung abscesses. Other complications include internal jugular vein thrombosis, carotid artery infection and erosion, bacteremia and sepsis, pneumoperitoneum, subphrenic abscess, cervicothoracic necrotizing fascitis, and spontaneous pneumothorax.

Retropharyngeal Abscess

Perspective and Principles of Disease

Retropharyngeal swelling reflects expansion of the retropharyngeal, danger, or prevertebral spaces. This discussion refers to infections in these spaces collectively as retropharyngeal abscesses.

Retropharyngeal abscess was previously a disease of childhood, with 96% of cases occurring in patients younger than 6 years. Adults are now increasingly affected. Children younger than 4 years have prominent retropharyngeal lymph nodes that may become infected and lead to retropharyngeal cellulitis and abscess formation. The increased use of antibiotics to treat pharyngitis in children has led to a declining incidence of retropharyngeal abscesses in this age group. These retropharyngeal nodes atrophy after 4 to 6 years of age, and thus the incidence and pathophysiology of this entity differ in adults.

In adult patients, cellulitis develops in the retropharyngeal area. Once the retropharyngeal space is involved, the infection spreads rapidly and an abscess may form. Nasopharyngitis, otitis media, parotitis, tonsillitis, peritonsillar abscess, dental infections and procedures, upper airway instrumentation, endoscopy, lateral pharyngeal space infection, and Ludwig’s angina are all implicated in the development of retropharyngeal abscesses. Other causes include blunt and penetrating trauma (usually from foreign bodies, commonly fish bones), ingestion of caustic substances, vertebral fractures, and hematologic spread from distant infection. Vertebral osteomyelitis and diskitis may lead to infection of the prevertebral space. Danger space infections are caused by extension of infection from either the retropharyngeal or prevertebral spaces. Underlying systemic disorders (e.g., diabetes and depressed immune system) may predispose individuals to retropharyngeal infections.

Retropharyngeal abscesses are most commonly polymicrobial with a mixture of aerobes and anaerobes. β-Lactamase-producing organisms are present in two thirds of the cases. Tuberculosis is rarely reported in the United States as a cause of retropharyngeal abscess. Staphylococcus is currently the most common cause of pyogenic vertebral osteomyelitis.
leading to the formation of retropharyngeal abscess. Dissemi-
nated coccidioidomycosis may also cause retropharyngeal
abscess.\textsuperscript{18,22,27,28}

\section*{Clinical Features}

Patients typically have a sore throat, dysphagia, odynophagia, drooling, a muffled voice, neck stiffness, neck pain, and fever. Dysphonia is usually present and is described as a duck “quack” (\textit{cri du canard}). Patients may complain of feeling a lump in their throat. Patients with a retropharyngeal abscess may appear quite ill and generally prefer to hold their necks extended and remain in the supine position. This position keeps the swollen posterior pharynx from compressing the upper airway. Forcing the patient to sit may lead to increased dyspnea.\textsuperscript{18,26}

Physical examination may reveal tender cervical lymphadenopathy, tender cervical musculature, neck swelling, torticollis, and often a high fever. Trismus may be present and make visualization of the pharynx difficult. In cases of retropharyngeal cellulitis, diffuse edema and erythema of the posterior pharynx are present.\textsuperscript{18,26} Once an abscess develops, palpation of the pharynx may demonstrate a unilateral mass if the retropharyngeal space is affected and a midline mass if the abscess is in the prevertebral or danger space. Palpation of a fluctuant mass is unreliable and carries a risk of inadvertent rupture of the abscess. Tenderness on moving the larynx and trachea side to side (tracheal “rock” sign) is commonly present. A retropharyngeal abscess may also cause pain in the back of the neck or shoulder that is precipitated by swallowing. Cold abscesses (caused by tuberculosis) are characterized by insidious onset, chronicity, constitutional symptoms, and less of a febrile response. Symptoms disproportionate to the findings should prompt further evaluation.\textsuperscript{28}

\section*{Diagnostic Strategies}

Diagnosis rests on the clinical findings and lateral cervical radiographs, CT, and MRI (Fig. 73-7). The soft tissue along the anterior bodies of C1-C4 should be less than 40\% of the diameter of the vertebral body just behind it; an increase in this tissue thickness suggests infection or abscess. Inspiratory lateral neck films often demonstrate thickening of the retropharyngeal soft tissues with forward displacement of the larynx and esophagus.\textsuperscript{20} The soft tissue swelling may be diffuse in the case of cellulitis or more focal if an abscess cavity is present. A pathologic process is suggested if the retropharyngeal space on lateral neck films (measured from the anteroinferior aspect of the second vertebral body to the posterior pharyngeal wall) is wider than 7 mm in both children and adults or the retrotra-
cheal space (measured from the anteroinferior aspect of the sixth vertebral body to the posterior pharyngeal wall) is more than 14 mm in children and 22 mm in adults. True lateral films with the neck fully extended during deep inspiration are the most reliable. Other radiographic findings include reversal of the normal lordosis of the cervical spine, air-fluid levels in the abscess cavity, foreign bodies, and vertebral body destruction.

Plain films may not be sufficiently sensitive to diagnose retropharyngeal abscess. CT or MRI should be performed in these instances. These studies not only aid in the diagnosis and differentiation between cellulitis and abscess but also determine the extent of the disease process and the presence of complications (Fig. 73-8).\textsuperscript{20,22,28} Ultrasonography is useful for differentiating retropharyngeal cellulitis from retropharyngeal abscess.

\section*{Differential Considerations}

The differential diagnosis includes retropharyngeal tumors, foreign bodies, inflammation, hematoma, aneurysms, hemorrhage, lymphadenopathy, and edema. Other considerations
include tendinitis of the longus colli muscle and retropharyngeal thyroid tissue.\textsuperscript{20}

**Management**

Patients with retropharyngeal cellulitis are best treated with high-dose IV antibiotics. Appropriate regimens are similar to those used for peritonsillar abscess. Tuberculosis and fungal infections must also be considered. Resolution of retropharyngeal cellulitis is possible without surgical intervention.\textsuperscript{5,18,22,26,28}

Generally, retropharyngeal abscesses are treated with antibiotics, in conjunction with operative incision and drainage. In selected cases, retropharyngeal abscesses can be treated successfully with antibiotics alone or in combination with needle aspiration. Cold abscesses should only be drained extraorally, unless the patient is in acute respiratory distress.\textsuperscript{22,26,28}

Neck immobilization may be necessary in patients with vertebral body destruction caused by osteomyelitis or atlantoaxial separation. These patients need neurosurgical or orthopedic evaluation and may require internal or external fixation.

**Disposition**

Airway compromise can be caused by anterior displacement of the pharyngeal tissues. Pulmonary complications include abscess rupture with aspiration and subsequent pneumonia, empyema, and asphyxiation. Extension of the infection along tissue planes and through other deep spaces may lead to mediastinitis and mediastinal abscess formation, pericarditis, pleuritis, and empyema. In addition, abscesses may track into the back of the neck and into the axilla. Vascular complications occur from the extension of the retropharyngeal abscess into the lateral pharyngeal space. Atraumatic atlantoaxial separation is due to damage to the transverse ligament of the atlas by the abscess. These patients may have neurologic symptoms and a widened predental space on plain films, CT scan, or MRI. Acute transverse myelitis and epidural abscesses also occur, and both can result in quadriplegia. Other complications include internal carotid pseudoaneurysm, erosion into the esophagus and auditory canal, necrotizing fasciitis of the neck, acute respiratory distress syndrome, sepsis, and death.\textsuperscript{18,28}

**PARAPHARYNGEAL ABSCESS**

**Perspective**

The parapharyngeal space, also known as the lateral pharyngeal and pharyngomaxillary space, is divided into two compartments by the styloid process. The anterior compartment contains connective tissue, muscle, and lymph nodes. The carotid sheath (which contains the carotid artery, internal jugular vein, vagus nerve, cranial nerves IX through XII, and the cervical sympathetic chain) runs in the posterior compartment.\textsuperscript{18}

**Principles of Disease**

Parapharyngeal abscesses are most often polymicrobial infections. Odontogenic and pharyngotonsillar infections are the most common causes of parapharyngeal space abscesses. Parapharyngeal space infections can also arise by contiguous spread from other surrounding deep neck space infections. Other causes include parotitis, sinusitis, spread from infected neck tumors, infected branchial cleft cysts, suppuration of local lymphadenitis, iatrogenic introduction of organisms during a mandibular nerve block or anesthesia for tonsillectomy, nasal intubation, dental extraction, chronic otitis with cholesteatoma, and mastoiditis.\textsuperscript{18}

**Clinical Findings**

Pain and swelling of the neck are the most common complaints. Odynophagia is present in most patients. A history of an antecedent sore throat may be elicited in some patients. Torticollis caused by irritation of the sternocleidomastoid muscle is also reported.\textsuperscript{18}

The classic physical findings of infection involving the anterior compartment of the parapharyngeal space are medial tonsillar displacement and posterolateral pharyngeal wall bulging. Other findings include fever, trismus (caused by irritation of the muscles of mastication), edema, and swelling at the angle of the jaw. An erythematous, tender, nonfluctuant swelling at the angle of the mandible is a consistent finding in patients with an anterior parapharyngeal abscess.\textsuperscript{18}

Involvement of the posterior space is associated with many of these same signs. If the anterior compartment is spared, however, little or no trismus occurs. Instead, posterior displacement of the tonsillar pillar and retropharyngeal swelling may be present.\textsuperscript{18}

**Diagnostic Strategies and Differential Considerations**

The diagnosis of parapharyngeal abscess is suggested by the presence of a severe sore throat with the characteristic physical findings. Blood cultures are usually sterile unless jugular vein thrombophlebitis is complicating the parapharyngeal space infection. Ultrasonography, CT, and MRI are more useful than lateral radiographs in diagnosing parapharyngeal abscesses and its complications. Angiography, Doppler flow studies, and magnetic resonance angiography may also be helpful in evaluating vascular complications.\textsuperscript{18,22,29}

The differential diagnosis includes infections in other deep spaces of the neck, tumors and metastatic lymph nodes, thyroiditis, branchial cleft cyst, and carotid artery aneurysms.

**Management**

Treatment includes high-dose IV antibiotics and consultation with an otolaryngologist for surgical drainage of the abscess cavity. Appropriate antibiotic regimens are discussed in the section on the treatment of peritonsillar abscess. Intravenous antibiotics alone will cure parapharyngeal space infections in selected patients and should be started on an emergency basis.\textsuperscript{5,18,22,29,30} Successful resolution of parapharyngeal abscesses with high-dose IV antibiotics and needle aspiration is reported.\textsuperscript{22,29}

**Disposition**

Complications of a parapharyngeal abscess include airway obstruction and abscess rupture with subsequent aspiration, pneumonia, and empyema. Infection can spread to surrounding spaces and into the mediastinum and pericardium. Such spread may lead to mediastinitis, mediastinal abscess, pericarditis, myocardial abscess, and empyema. Other complications include osteomyelitis of the mandible, cervicothoracic necrotizing fasciitis, parotid abscess, cavernous sinus thrombosis, and meningitis.\textsuperscript{16}

Posterior parapharyngeal space infections are particularly dangerous. These infections may affect the cervical sympathetic chain, carotid artery, or internal jugular vein. Ipsilateral
Horner’s syndrome and neuropathies of cranial nerves IX through XII may occur. Carotid artery erosion may lead to hemorrhage and the formation of aneurysms. Oral, nasal, and aural warning bleeding is common with carotid artery erosion, with aural bleeding being particularly ominous. Any unexplained bleeding associated with paraparonyeal or other deep neck space infection should be investigated thoroughly. Persistent peritonsillar swelling despite resolution of the paraparonyeal abscess or a tender unilateral pulsatile mass may indicate an arterial aneurysm. Aspiration or incision of a carotid artery aneurysm thought to be a paraparonyeal abscess may have disastrous complications.18

Involvement of the internal jugular vein may lead to septic thrombosis and Lemierre’s syndrome.13,14 This entity, also called postganginal septicemia, affects primarily young healthy patients and is easily confused with right-sided endocarditis or aspiration pneumonia. The manifestation is one of a pharyngitis that initially improves but is then followed by severe sepsis. It is thought that the pharyngeal infection spreads to the paraparonyeal space and causes septic thrombophlebitis of the jugular vein. Patients usually appear ill and are febrile. Metastatic infections involve primarily the lung and are manifested by bilateral nodular infiltrates, pleural effusion, and pneumothorax. Septic arthritis, osteomyelitis, soft tissue cellulitis and abscesses, meningitis, and a vesiculopustular rash are also reported as a result of septic embolization. Leukocytosis and elevated bilirubin and liver function test values, with and without hepatomegaly and jaundice, are often present. Albuminuria, hematuria, and elevations in serum creatinine and blood urea nitrogen are reported. Septic shock rarely develops, although acute respiratory distress syndrome, transient coagulopathies, and hypotension commonly occur. The most frequent cause of this entity is Fusobacterium (primarily Fusobacterium necrophorum), although S. aureus is the most common pathogen in IV drug users. Treatment consists of parenteral antibiotics and incision and drainage of abscesses. Jugular vein ligation and resection are necessary in patients with uncontrolled sepsis and respiratory failure caused by repeated septic pulmonary emboli. The value of anticoagulation is unknown.13,14

## RHINOSINUSITIS

### Perspective and Principles of Disease

It is estimated that 0.5 to 2% of viral upper respiratory tract infections are complicated by rhinosinusitis. Since sinusitis usually involves the nasal cavity, the term rhinosinusitis is preferred. These terms will be used interchangeably in this section.32,33

The paranasal sinuses (frontal, maxillary, ethmoid, and sphenoid) are named for the facial bones with which they are associated. Pneumatization may involve other bones but represents extension from the main sinus. The maxillary, anterior ethmoid, and frontal sinuses drain into the medial meatus, located between the inferior and middle nasal turbinates. This area is named the ostiomeatal complex and is the focal point of sinus disease. The posterior ethmoid sinus drains into the superior meatus and the sphenoid sinus just above the superior turbinate.32,33

A healthy sinus depends on a patent ostium with free air exchange and mucus drainage. A healthy sinus is sterile and does not accumulate mucus. Viral upper respiratory tract infections and allergic rhinitis are the most common causes of ostial obstruction with resultant sinusitis. Ciliary abnormality or immobility inhibits drainage and is another important cause of sinusitis. Ciliary dysfunction can be temporary (e.g., upper respiratory infection) or permanent (e.g., syndromes associated with ciliary structural abnormalities). Infection leads to increased mucus viscosity, thus further impeding drainage. Bacteria are introduced into the sinus by coughing and nose blowing. These processes lead to increased inflammation and bacterial overgrowth. Other factors predisposing to rhinosinusitis include immunocompromised status, nasal septal deviation and other structural abnormalities, nasal polyps, nasal tumors, trauma and fractures, rhinitis medicamentosa, rhinitis secondary to toxic mucosal exposure, barotrauma, foreign bodies, nasal cocaine abuse, and instrumentation (including nasogastric and nasotracheal intubation).32,33

Sinusitis can be classified into acute viral, acute bacterial, chronic, and recurrent acute variations. Approximately 90% of patients with colds have an element of the acute viral form. Acute viral sinusitis may lead to the development of the acute bacterial variety, S. pneumoniae, nontypable H. influenzae, and M. catarrhalis are the primary pathogens responsible for acute bacterial and recurrent acute sinusitis. P. aeruginosa is associated with sinusitis in the setting of HIV infection and cystic fibrosis. Anaerobic bacteria, streptococcal species, and S. aureus are more prominent causes of chronic sinusitis. Fungi also have a role in CS. Rhizopus, Aspergillus, Candida, Histoplasma, Blastomyces, Coccidioides, and Cryptococcus species, as well as other fungi, may cause sinusitis, primarily in immunocompromised hosts. It is important to distinguish infectious from allergic sinusitis. Allergic sinusitis is associated with sneezing, itchy eyes, allergen exposure, and previous episodes.32-35

### Clinical Features

Frontal sinusitis can cause severe headache localized to the forehead and orbit. Sphenoid sinusitis, may cause vague headaches and focal pain almost anywhere in the head. Maxillary sinusitis may be seen with pain over the zygoma, in the canine or bicuspid teeth, or periorbitally. Ethmoid sinusitis can cause medial canthal pain and periorbital or temporal headaches.32,33

The cardinal symptoms of acute rhinosinusitis are mucopurulent nasal discharge, nasal obstruction or congestion, and facial pain, fullness, or pressure lasting less than 4 weeks. Other symptoms and signs include postnasal drip (that may lead to coughing), pressure over the involved sinus, malaise, hypoxemia, anosmia, fever, maxillary dental pain, and ear fullness or pressure. Acute sinusitis typically progresses over a period of 7 to 10 days and resolves spontaneously. During the first 3 to 5 days of illness, it may be difficult to differentiate acute viral from acute bacterial sinusitis. The bacterial origin is suggested by worsening symptoms within 10 days, persistent symptoms after 10 days, or “double sickening,” which refers to patients who improve initially, only to have worsening sinus congestion and discomfort. Bacterial infection is also associated with more severe presentations and extranasal manifestations of infection.32,33

Chronic sinusitis is slow in onset, prolonged in duration (greater than 12 weeks), and recurrent in frequency. Symptoms can be nonspecific but are generally similar to those of acute disease. Symptoms of chronic disease may also include chronic cough, fetid breath, laryngitis, bronchitis, and worsening asthma. Recurrent acute sinusitis is diagnosed when four or more episodes of acute bacterial infection, without its signs or symptoms between episodes, occur per year. The presentation and treatment of recurrent acute disease is similar to that for acute bacterial sinusitis.32,33

Invasive fungal sinusitis (mucormycosis) is an aggressive opportunistic rhinocerebral infection that affects immunocompromised hosts. Mucormycosis (Rhizopus) is generally associ-
ated with fever, localized nasal pain, and cloudy rhinorrhea. On examination, the affected tissue (usually the turbinates) appears gray, friable, anesthetic, and nonbleeding because of infarction caused by mucormycotic angioinvasion. In advanced cases the affected tissues are necrotic and black, and the infection spreads beyond the sinus.

**Diagnostic Strategies**

Physical examination is best performed after the application of a topical decongestant. Mucosal erythema and edema are usually present. Purulent discharge from the nasal meatus may be observed if the sinus ostia are not completely obstructed. In the setting of acute sinusitis, nasal and nasopharyngeal cultures correlate poorly with cultures of sinus aspirates and cultures obtained at the time of open antrostomy and do not differentiate between acute viral and acute bacterial infections. Culture and biopsy are indicated in suggested chronic, recurrent acute, and fungal sinusitis. Radiographic examination should be limited to diagnosis of chronic or recurrent acute sinusitis, cases of questionable diagnoses, those with unresponsive disease, or investigation of complications. Axial and coronal CT is the imaging modality of choice. CT findings suggestive of sinusitis include air-fluid levels, sinus opacification, sinus wall displacement, and mucosal thickening (Fig. 73-9). CT is sensitive, though not specific. Incidental sinus mucosal thickening is seen in 40% of asymptomatic patients, and abnormal CT findings can also be noted in just half of patients with seasonal allergies. CT with IV contrast or MRI may be required to evaluate complications of rhinosinusitis; plain films have limited utility in the diagnosis of rhinosinusitis; positive findings are similar to those of CT. Sinus endoscopy is another option.

**Differential Considerations**

Rhinitis can be differentiated from sinusitis by the increased response of nasal obstruction to treatment, clear nasal discharge, and absence of pain. Rhinitis does not lead to ostial obstruction, and thus patients do not complain of facial pain. Malignancy, tension headache, vascular headache, foreign body, dental disease, brain abscess, epidural abscess, meningitis, and subdural empyema may also present in a manner similar to sinusitis.

**Management**

Analgesics, antipyretics, and decongestants play important roles in the symptomatic management of sinusitis. A large proportion of cases of viral and bacterial sinusitis resolve spontaneously, and antibiotic therapy offers only modest incremental benefit. Most patients do not require antibiotics, and antibiotics should be reserved for those with symptoms and signs of sinusitis who have not improved after 7 days, who worsen despite adequate symptomatic treatment, or who have moderate to severe symptoms, including fever and purulent discharge, strongly suggestive of bacterial sinusitis, regardless of the duration of symptoms. Patients who are at high risk for severe infection or complications should also be considered for antibiotic therapy. The choice of antibiotics must consider β-lactamase production and multidrug-resistant pneumococci. Amoxicillin administered for 7 to 10 days is still the first-line agent, but benefits are limited and treatment failures occur in areas with a high percentage of β-lactamase-producing bacteria. High-dose amoxicillin should be considered for patients who have a child in daycare in the household. Penicillin-allergic patients may be treated with trimethoprim-sulfamethoxazole or a macrolide antibiotic. A 3-day course of trimethoprim-sulfamethoxazole or azithromycin and decongestants may be as effective as the standard 10-day antibiotic course. Failure of symptoms to resolve after 7 days of therapy, or antibiotic usage in the past 4 to 6 weeks, necessitates a change to a broader spectrum antibiotic and reassessment of the patient to confirm the diagnosis of acute bacterial sinusitis. Appropriate management includes a 10- to 14-day course of high-dose amoxicillin-clavulanate, cefuroxime axetil, other second- or third-generation cephalosporins, clindamycin alone or in combination with ciprofloxacin, sulfamethoxazole, azithromycin, clarithromycin, or one of the respiratory fluoroquinolones (levofloxacin, moxifloxacin, gemifloxacin). Metronidazole may be added to any of these regimens to increase activity against anaerobic organisms. Antibiotics for chronic sinusitis should be effective against anaerobic and β-lactamase-producing bacteria. Treatment of life-threatening complications requires consultation and high-dose IV antibiotics, including cefuroxime, ceftriaxone, or ampicillin-sulbactam. Antifungals may be beneficial in the treatment of chronic sinusitis.

The goal of decongestant therapy is to reduce tissue edema, facilitate drainage, and maintain patency of the sinus ostia. Decongestants are available in topical and systemic preparations, both of which should be used simultaneously in conjunction with appropriate antibiotics. Topical agents provide more relief than systemic decongestants. Topical agents include 0.5% phenylephrine hydrochloride and 0.05% oxymetazoline hydrochloride. Topical agents should be used for only 3 to 5 days. Extended use results in rebound vasodilation and nasal obstruction, a condition termed rhinitis medicamentosa. Systemic oral adrenergic agonists (e.g., phenylpropanolamine or pseudoephedrine) reduce nasal blood flow and congestion. These medications should be used cautiously in patients taking tricyclic antidepressants, monoamine oxidase inhibitors, and nonselective beta-adrenergic blockers. Antihistamines should be reserved for the treatment of allergic sinusitis because these agents may impede sinus drainage. Second-generation H1-antagonists are preferred due to their better side effect profile. Topical, but not systemic, steroids

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**Figure 73-9.** Computed tomographic scan showing bilateral maxillary sinus opacification.
are indicated for chronic and allergic sinusitis. Systemic steroids may be indicated in allergic and chronic sinusitis with nasal polyps.\textsuperscript{32,34,38} Saline nasal irrigation is beneficial for treating acute bacterial, recurrent acute, and chronic sinusitis. Saline irrigation may be efficacious for the prevention of sinusitis. Hypertonic saline preparations have superior anti-inflammatory properties and may be more effective than normal saline.\textsuperscript{32,33}

**Complications**

Most cases of uncomplicated acute bacterial sinusitis can be treated on an outpatient basis with systemic decongestants, topical decongestants, and oral antibiotics. Failure of definitive antibiotic therapy suggests that the patient’s sinusitis has extended to the chronic stage and necessitates referral to an otolaryngologist. Treatment of chronic sinusitis requires a prolonged (3–6 week) course of antibiotics.

Frontal or sphenoid sinusitis with air-fluid levels may require hospitalization. A previously healthy, nontoxic patient with good home support can be treated as an outpatient but should return immediately for any symptoms or signs of complications, including severe headache, neurologic changes, or visual changes. Patients who appear toxic, who are immunoincompetent, or who have poor home resources require hospital admission and IV antibiotics.

Sinusitis is associated with an increased incidence of bronchitis and asthma. Infectious processes of the sinuses can spread to the orbit or central nervous system and can be fulminant. Sinusitis may extend to involve the bones and soft tissues of the face and orbit. Facial and periorbital cellulitis, peri orbital abscess, optic neuritis, blindness, and orbital abscess may develop. Patients with orbital complications may have marked swelling, proptosis, decreased ocular motility, and decreased visual acuity. Sinusitis may also lead to intracranial complications. Meningitis, cavernous sinus thrombosis, epidural or subdural empyema, and brain abscess occur. Intracranial involvement may result in headache, decreased sensorium, or focal neurologic deficits and has a rapidly progressive course. Acute fulminant fungal sinusitis requires IV antifungal therapy and aggressive surgical débridement.\textsuperscript{32,33,35} Complications of mucormycosis are directly related to delay in diagnosis and treatment. This opportunistic fungal infection rapidly progresses to involve the central nervous system and is associated with high morbidity and mortality rates.

### KEY CONCEPTS

- A severe sore throat with surprisingly minimal findings on examination of the oropharynx suggests serious soft tissue infection such as epiglottitis, retropharyngeal abscess, or peritonsillar abscess.
- Deep space cellulitis is difficult to differentiate from deep space abscess and may require needle aspiration after CT or MRI.
- Keep patients with upper airway infections in a position of comfort.
- Posterior parapharyngeal abscess may involve the cervical sympathetic chain, carotid artery, or internal jugular vein.
- Aspiration and incision of a carotid artery aneurysm thought to be a parapharyngeal abscess may be disastrous.
- Resolving pharyngitis followed by severe sepsis, right-sided endocarditis, or aspiration pneumonia should suggest septic thrombosis of the internal jugular vein.

*The references for this chapter can be found online by accessing the accompanying Expert Consult website.*