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Concepts and Clinical Practice

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61

Upper Respiratory Tract Infections

Matthew A. Roginski and Patricia Ruth Atchinson

KEY CONCEPTS

- Viral infections cause most cases of pharyngitis. Patients should not be treated with antibiotics based on symptoms and exam alone. Patients with a Centor criteria score of 0 or 1 do not require further testing or treatment. Those with a score of 2 or greater should undergo rapid antigen testing with treatment decisions based on results. Throat cultures are recommended in children but are not necessary in adults.
- Antibiotics for group A *Streptococcus* pharyngitis are aimed at symptom reduction, decreasing transmission, and decreasing suppurative complications. Rheumatic fever is very rare and likely from a shift in streptococcal M proteins rather than antibiotic use.
- Infectious pharyngitis presents with an acute onset and resolves within days. In subacute and chronic cases of pharyngitis, consider abscess formation, neoplastic causes, HIV, and autoimmune disease.
- In streptococcal pharyngitis, a single high dose of corticosteroids, such as 10 mg dexamethasone, is safe and reduces symptom severity.
- Consider deep space infection and epiglottitis in patients who present with neck pain, hoarseness, and have a benign oropharyngeal exam.
- Airway edema from palatine or lingual tonsillitis, epiglottitis, or deep space infection is rare, but life-threatening.
- Deep space infections such as retropharyngeal and parapharyngeal abscesses are difficult to diagnose clinically, and contrast-enhanced CT is recommended.
- Transcervical or intraoral ultrasound is useful in the diagnosis and treatment of peritonsillar abscess.
- Acute rhinosinusitis is likely viral and will resolve with supportive care, including nasal irrigation with hypertonic saline.
- Although rarely needed, first-line antibiotic treatment for acute rhinosinusitis is amoxicillin or amoxicillin-clavulanate for 5 days.

PHARYNGITIS

Foundations

Tonsillopharyngitis (pharyngitis) is generally a benign, self-limited inflammatory syndrome of the oropharynx. Although most cases are mild, severe cases may lead to airway swelling, dehydration from decreased oral intake, and suppurative complications, including peritonsillar abscess, deep space infection, and hematogenous spread. A majority of cases are viral and caused by common cold viruses. Bacterial infection is responsible for approximately 5% to 10% of adult and 20% to 30% of pediatric cases. Common bacterial causes of pharyngitis include group A beta-hemolytic *Streptococcus* (GAS), non–group A *Streptococcus*, *Fusobacterium*, and mixed aerobes and anaerobes (Table 61.1). Transmission occurs through direct person-to-person contact or via aerosolized respiratory secretions. Although fomite transmission is rare, crowded conditions such as in schools, daycare, and military training facilities increase transmission rates.

Tonsils are lymphoid tissue covered with respiratory epithelial tissue. Waldeyer ring refers to the lymphoid tissue in the pharynx consisting of the palatine tonsils (commonly referred to as tonsils), pharyngeal tonsils (adenoids), tubal tonsils (surrounding eustachian tubes), and lingual tonsils at the base of the tongue. Tonsillar infection, inflammation, and hypertrophy may occur in any of these locations (Fig. 61.1). Although rare, lingual tonsillitis predominantly occurs in patients who had their palatine tonsils removed.

Clinical Features

Symptom chronicity, associated complaints, patient comorbidities, and patient risk factors are important considerations. Acute presentations are more likely to be infectious, whereas more chronic presentations raise concern for noninfectious or neoplastic etiologies. Sore throat, odynophagia, fever, malaise, and tender anterior cervical adenopathy are the most common symptoms. Although presentation differs by the disease process, erythema, edema, and petechiae of the oropharynx (Fig. 61.2), as well as palatine tonsillar plaques (Fig. 61.3), are common findings.

Viruses that cause the common cold are responsible for 30% to 60% of pharyngitis cases (see Table 61.1). Symptoms may overlap with GAS but are unlikely to be as severe and are associated with rhinorrhea, cough, conjunctivitis, congestion, and headache. Viral symptoms usually precede symptoms of a sore throat. Inflammation and hypertrophy of tissue in the Waldeyer ring without exudate is common.

Infectious mononucleosis, caused by the Epstein-Barr virus (EBV), classically presents with the triad of fever, tonsillar pharyngitis, and posterior cervical lymphadenopathy. The incubation period is 3 to 7 weeks, and patients experience a prodrome of fever, chills, and malaise. Most patients have exudative pharyngitis with tonsillar hypertrophy (see Fig. 61.3). Petechiae are intermittently present at the junction of the hard and soft palate (see Fig. 61.2). In severe cases, upper airway swelling may lead to difficulty managing secretions, stridor, and dyspnea. Splenomegaly is present in approximately half the cases. A pruritic morbilliform rash may occur regardless of contact with beta-lactam antibiotics. Jaundice is rare.

Influenza may present with nonexudative pharyngitis and sore throat along with generalized fever, chills, myalgia, and headaches. Human immunodeficiency virus (HIV) and cytomegalovirus can present as a mononucleosis-like illness. Pharyngitis and hypertrophy of tissue in the Waldeyer ring may also occur. The acute retroviral syndrome of primary HIV infection includes fever, sore throat, nontender lymphadenopathy, diffuse maculopapular rash, arthralgias, mucocutaneous ulcerations, and diarrhea. Spread by sexual contact, herpes simplex pharyngitis presents with painful vesicles or ulcerations on an erythematous base on the lips, tongue, palate, or mucosa (Fig. 61.4). Patients with oropharyngeal herpes have palatal hyperemia with sore throat, odynophagia, stomatitis, and tender cervical adenopathy. Immunosuppressed patients with oropharyngeal herpes may present with large ulcerations, and bacterial superinfection is possible. Coxsackie virus may also cause herpangina

TABLE 61.1 Infectious and Noninfectious Causes of Pharyngitis

INFECTIOUS ETIOLOGIES				NONINFECTIOUS ETIOLOGIES
Bacterial	**Viral**	**Fungal**	**Adjacent Infections**	
Group A β-hemolytic strep	Rhinovirus	Candida	Retropharyngeal abscess	Tumor
Groups C and B β-hemolytic strep	Coronavirus		Parapharyngeal abscess	Autoimmune disease
Fusobacterium	Parainfluenza		Epiglottitis	Neurogenic pain
Neisseria gonorrhoeae	Adenovirus		Ludwig angina	Foreign body
Corynebacterium diphtheriae	Influenza			Trauma
Chlamydia	Human immunodeficiency virus			Medication induced
Mycoplasma pneumoniae	Epstein-Barr virus			Stevens-Johnson syndrome
Arcanobacterium haemolyticum	Herpes virus			Allergic reaction
	Cytomegalovirus			Esophageal reflux
				Environmental exposure

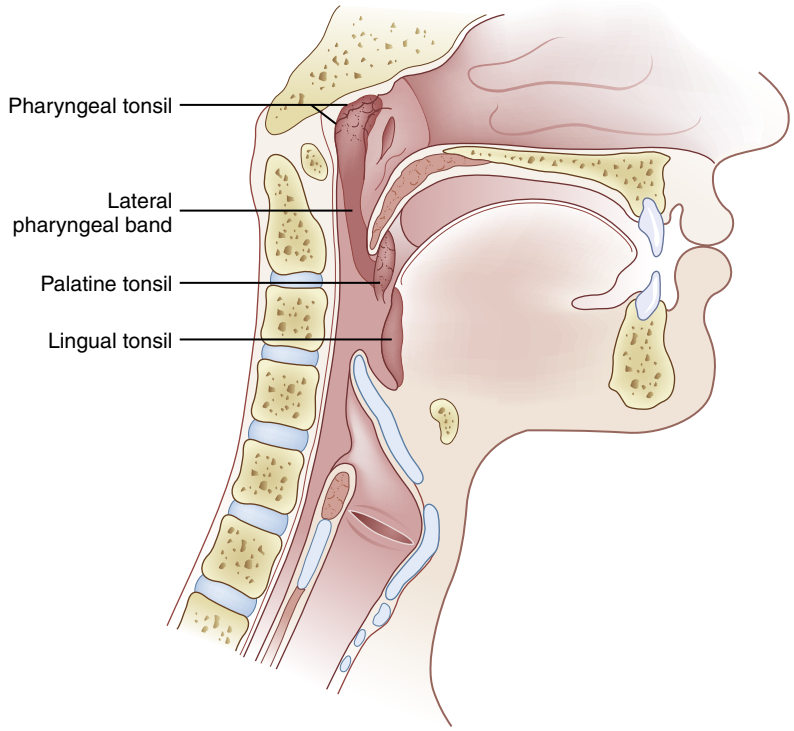

Fig. 61.1 Sagittal anatomic illustration of locations of tonsils in Waldeyer ring. Note the lingual tonsils on the posterior tongue. (From Embryology, anatomy, and histology of the pharynx. In: Wenig, BM. *Atlas of Head and Neck Pathology*. Philadelphia: Elsevier; 2016: 399-406. Fig. 8-2.)

Fig. 61.2 Palatal petechiae. (Courtesy Centers for Disease Control and Prevention and Dr. Heinz F. Eichenwald.)

with oral and pharyngeal erythematous papulo-vascular ulcerations. Coxsackie virus also causes small tender, nonpruritic, cutaneous lesions on the palms, soles, and buttocks.

Group A beta-hemolytic *Streptococcus* is a gram-positive coccus that grows in chains and is the most frequent cause of bacterial pharyngitis, most commonly in children ages 5 to 15 years. Humans are the only carrier of GAS, and asymptomatic carrier status is uncommon. The incubation period is typically 2 to 5 days. Virulence factors of GAS include host inflammatory mediators, bacterial cell wall, and secreted enzymes and exotoxins. Infection is most common in the fall and winter. Untreated, symptoms last 3 to 7 days, and patients are contagious up to one week after symptom resolution. Treated, symptoms resolve approximately 16 hours sooner than in untreated patients, and the contagious period decreases to 24 hours after the start of antibiotics. Rapid onset of sore throat, odynophagia, cervical adenopathy, fevers, chills, and neck stiffness are characteristic. Headache, abdominal pain,

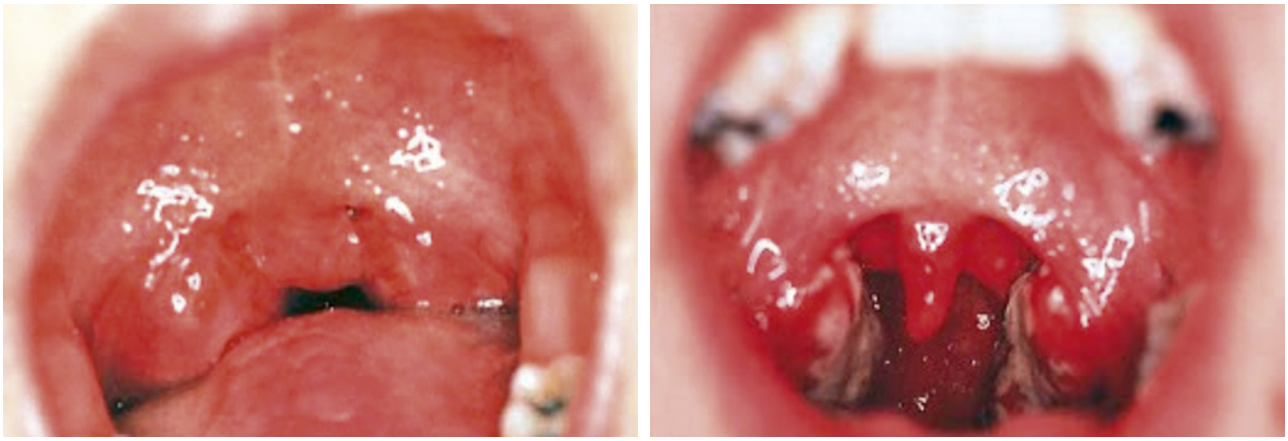

Fig. 61.3 Photograph of enlarged palatine tonsils. (A) shows nonexudative pharyngitis. The diffuse tonsillar and pharyngeal erythema seen here is a nonspecific finding that can be produced by a variety of pathogens. (B) shows exudative pharyngitis with white plaques on the tonsils and hyperemia, most commonly seen in either group A streptococcal or Epstein-Barr virus infection. (From Wetmore RF. Tonsils and adenoids. In: *Nelson Textbook of Pediatrics*. 2019. Pages 2198-2202.e.1. 2020.)

Fig. 61.4 Photograph of oral ulcerations from recurrent oropharyngeal herpes infection. (From Hellstein JW, Fielding CG, Castle JT. *Vesiculobullous Diseases. Oral and Maxillofacial Surgery*. Elsevier. Published January 1, 2018. Pages 550-574. © 2018. Figure 24-4 Recurrent herpes of palate.)

nausea, and vomiting may be present. GAS does not present with trismus, cough, conjunctivitis, diarrhea, rhinorrhea, or oral ulcerations.[1]

Exam findings include symmetric erythema and edema of the oropharynx, gray/white tonsillar exudates (see Fig. 61.3), palatal petechiae (see Fig. 61.2), and tender cervical adenopathy. GAS pharyngitis associated with a desquamating, fine, sandpaper-like rash is called *scarlet fever* and is related to an exotoxin-producing strain of GAS. Suppurative complications of GAS include acute otitis media, mastoiditis, meningitis, peritonsillar and retropharyngeal abscess, and rarely, necrotizing fasciitis or hematogenous spread to distant sites.[2] Acute rheumatic fever and post-streptococcal glomerulonephritis are rare in the general population. The decrease in the frequency of acute rheumatic fever is likely due to a shift in the streptococcal M protein types leading to decreased rheumatogenicity rather than due to the increased use of antibiotics.[2]

Non–group A *Streptococcus* also causes acute pharyngitis with a similar presentation to GAS, but acute infection is difficult to distinguish from normal upper respiratory flora on respiratory culture. *Fusobacterium necrophorum* is an anaerobic gram-negative rod that is part of the normal oral flora and causes pharyngitis in patients 15 to 45 years old with a similar presentation to GAS. *Fusobacterium* is the primary causative agent in septic jugular vein thrombophlebitis (Lemierre syndrome). The early identification and treatment (Table 61.2) of *Fusobacterium* in pharyngitis remains unclear; *Fusobacterium* should be considered in young adults with ongoing severe symptoms.[3-5]

Arcanobacterium haemolyticum is a nonmotile beta-hemolytic gram-positive bacillus that is not part of the upper respiratory flora and is associated with a minority of pharyngitis cases with peak prevalence in the late teens. It is associated with deep space infections such as retropharyngeal abscesses. Along with pharyngitis, patients may also experience an urticarial, maculopapular rash that spares the face, palms, and soles. *Francisella tularensis* is a zoonotic gram-negative bacillus that may cause a false-positive monospot test and has atypical lymphocytes on peripheral smear. It can present as pharyngitis and a flu-like illness in patients with a contaminated food or water source.[3,4] *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* pharyngitis may occur as part of an outbreak in crowded conditions. Lower respiratory tract infection and rhinosinusitis may occur.

Neisseria gonorrhoeae is an intracellular gram-negative diplococcus that is transmitted through sexual contact. Patients are usually symptomatic with tonsillitis and found to have enlarged tonsils with a white-yellow exudate in the tonsillar crypts. *Chlamydia trachomatis* pharyngitis has a similar presentation to gonococcal pharyngitis and is transmitted through sexual contact. *Treponema pallidum* is the spirochete that causes syphilis. Primary infection can present with painless oral ulceration. Secondary syphilis may have associated pharyngitis with localized adenopathy that can be mistaken for carcinoma. Patients with atypical presentations, risk factors for sexually transmitted infections, or no other apparent cause should have a focused sexual history and appropriate testing based on their response.

Diphtheria is caused by the gram-positive bacillus *Corynebacterium diphtheriae*. Although the incidence has decreased because of vaccination, diphtheria should be considered in patients who traveled to endemic areas or who are not vaccinated. The incubation period is 2 to 5 days. Early symptoms are typical of generalized pharyngitis with sore throat, cervical adenopathy, and fever. Toxin-producing strains may create a pseudomembrane that is grayish-green to black (if bleeding occurs) that tightly adheres to mucosal tissue and is friable. Severe forms may obstruct the airway and may be associated with extensive swelling of the tonsils, uvula, and anterior neck. Toxins produced can affect distant sites causing myocarditis, neuritis, and acute tubular necrosis. Non–toxin producing strains cause moderate to severe pharyngitis without pseudomembrane formation.

TABLE 61.2 Treatment Recommendations for Pharyngitis[a]

Organism	Treatment
Group A *Streptococcus*	Penicillin V, Oral • Children 250 mg two or three times daily for 10 days • Adolescents and adults 500 mg twice daily for 10 days Penicillin VK • Children <27 kg: 250 mg two or three times daily • Children ≥27 kg or adults: 500 mg two or three times daily Penicillin G, IM • <27 kg: 600,000 units; >27 kg 1.2 million U once Amoxicillin • 50 mg/kg once daily (max 1000 mg) or 25 mg/kg (max 500 mg) twice daily for 10 days Single-dose corticosteroids • Dexamethasone 0.6 mg/kg up to 10 mg PO Penicillin Allergic Cephalosporins • Cephalexin 20 mg/kg/dose (max 500 mg/dose) twice daily for 10 days Clindamycin • 7 mg/kg/dose (max 300 mg/dose) three times daily for 10 days Azithromycin • 12 mg/kg (max 500 mg/dose) for 5 days
Fusobacterium and anaerobic infections	Metronidazole • Adults 500 mg IV every 8 hours; children 10 to 15 mg/kg (max 500 mg/dose) IV every 8 hours AND Ceftriaxone • Adults 2 g IV every 24 hours; children 50 mg/kg (max 2 g/dose) IV every 24 hours OR Piperacillin-tazobactam • Adult 3.375 g IV every 6 hours, children 100 mg/kg of piperacillin (max 4.5 g/dose) IV every 6 to 8 hours
Diphtheria	Antitoxin (request from CDC) AND penicillin • Adults: Penicillin 2 to 3 million units/day IV in divided doses every 4 to 6 hours Children: Penicillin 150,000 to 250,000 units/kg/day IV in divided doses every 6 hours. The AAP recommends 14 days of treatment
Gonococcal	Ceftriaxone 500 mg IM ×1 (1g IM x1 if weight >150 kg) and doxycycline 100 mg twice daily for seven days (doxycycline is needed until chlamydia is ruled out)
Herpes	Acyclovir 200 mg 5 times/day for 7 days OR Valacyclovir 1 gm twice daily for 7 days OR Famciclovir 250 mg three times/day
Candida	Mild: Clotrimazole troches or nystatin swish/swallow Moderate to severe: adults, fluconazole 200 mg on day one and 100 to 200 mg once daily for 7 days; children, fluconazole 6 to 12 mg/kg (max 200 mg/dose) daily

[a]Renally dose antibiotics, antivirals, and antifungals.

Oral candidiasis presents with a white pseudomembrane over the tongue, buccal mucosa, palate, and oropharynx that can be scraped off with a tongue depressor. It occurs most commonly in immunocompromised patients, the elderly, patients with recent antibiotic exposure, and those on chronic steroids. Chronic hyperplastic candidiasis may present as elevated white plaques that cannot be scraped off of the buccal mucosa.

BOX 61.1 Centor Criteria for Determining Group A Beta-Hemolytic Streptococcal Pharyngitis

Swollen tonsils and exudates
Tender anterior cervical adenopathy
Absent cough
Fever

Differential Diagnosis for Emergency Presentation

A focused differential for pharyngitis is largely guided by the history and physical exam with attention to systemic signs and symptoms. Table 61.1 lists the infectious and noninfectious causes of pharyngitis.

Diagnostic Testing

Diagnostic testing is not required if features strongly suggest a viral etiology: associated with rhinorrhea, cough, conjunctivitis, congestion, and headache which usually precede symptoms of a sore throat. Testing for Epstein-Barr virus (EBV) can be considered in patients with splenomegaly, posterior cervical adenopathy, palatal petechiae, and those patients with persistent symptoms despite adequate treatment for GAS pharyngitis. The initial testing for infectious mononucleosis is a heterophile antibody test (Monospot). The Monospot has high specificity but variable sensitivity, with false-negative results in children and early in the infection. If the antibody test is negative and the patient remains symptomatic, we recommend follow-up with their primary care physician and retesting in 7 to 10 days or checking an EBV viral capsid antigen IgM. Viral load is not validated for infectious mononucleosis. Mononucleosis causes an absolute lymphocytosis with greater than 10% atypical lymphocyte count due to EBV's effect on B lymphocytes and the cytotoxic T-cell response. Influenza is tested with a PCR nasal or oral pharyngeal swab. Influenza testing should occur in patients with fevers, myalgia, headache, and sore throat occurring when influenza viruses are circulating in the community, when the patient is being admitted to the hospital, or when testing will influence management. HIV testing is performed with a fourth-generation combination assay. It should be performed in patients with risk factors and persistent pharyngitis symptoms without any other etiology. Herpes is diagnosed with PCR testing of a viral swab from an ulcer or unroofed vesicle or HSV IgG and IgM serology.

Rapid antigen testing is recommended as a first-line diagnostic test for GAS pharyngitis. These patients present with sore throat, fever, exudative pharyngitis, and cervical lymphadenopathy without associated cough or rhinorrhea. Scoring systems such as the Centor criteria (Box 61.1) should be used to identify adults who do not require further testing or treatment. The score should not be used in patients who are immunocompromised, have complicated comorbid conditions, or have symptoms for greater than 5 days. Patients with zero or one Centor criterion should not be tested or treated. Empiric treatment is not recommended for any patient.[1,5] Rapid antigen tests are highly sensitive and specific when performed correctly (swabbing the bilateral tonsils and the posterior pharynx, avoiding the buccal mucosa and tongue). A positive test indicates the presence of GAS and does not require follow-up testing. If the antigen testing is negative, a confirmatory culture on sheep blood agar is recommended in children, can be considered in adolescents, and is not necessary in adults. There is no role for antistreptolysin O titers.

Patients with persistent symptoms and unknown etiology should undergo throat culture. *Arcanobacterium haemolyticum* is performed on human blood agar. Diphtheria requires culture on Loeffler medium. The laboratory should be notified if there is concern for *Arcanobacterium* or diphtheria, which would be indicated in patients with severe pharyngitis or the appearance of gray pseudomembrane in the posterior oropharynx. Candida is diagnosed when budding yeast with or without pseudohyphae is seen on the Gram stain or potassium hydroxide stain. A throat swab with a nucleic acid amplification test is recommended for *Chlamydia trachomatis* and *Neisseria gonorrhoeae.*

Management

For a majority of cases, supportive care alone with nonsteroidal antiinflammatory medications or acetaminophen will be sufficient. There is no direct evidence of benefit from warm salt gargles, lozenges, soft or cold foods, or humidified topical analgesics. Viscous lidocaine should be avoided due to the potential for suppression of cough and gag and risk for aspiration. Treatment for infectious mononucleosis is supportive. Corticosteroids are not recommended for infectious mononucleosis,[6] except in the cases of significant oropharyngeal edema with stridor or change in phonation. Treatment of influenza should be started as soon as possible on documented or suspected influenza cases in hospitalized patients, children less than 2 years old, adults 65 years and older, pregnant women and within 2 weeks postpartum, patients with immunosuppression, and patients with chronic cardiac, pulmonary, hepatic, renal or hematologic disorders. Clinicians can consider antiviral treatment in patients not at high risk for complications and outpatients with symptom onset less than 2 days before presentation, and in those symptomatic with household contact with those at high risk for compilations of influenza, or health care workers with contact with those at high risk for complications.[7] Treatment consists of antiviral medication (oral oseltamivir, inhaled zanamivir, or intravenous peramivir.) HIV requires referral to appropriate providers for the initiation of antiretroviral medications.

The rationale for the treatment of GAS is to decrease the length of symptoms, suppurative complications, and the infectious period. Prophylactic antibiotics to close contacts of infected patients is not recommended. Retesting after treatment is not required unless there are recurrent symptoms. Treatment failures may be related to carrier status, lack of compliance, recurrent exposure, resistant bacteria, or eradication of protective flora. Treatments of pharyngitis are listed on Table 61.2. We recommend steroid administration for GAS. A single dose of corticosteroids such as dexamethasone appears to be safe and leads to a decrease in symptom duration.[8-10] Patients with recurrent episodes of GAS should be referred to an otolaryngologist for consideration of tonsillectomy.[11] Diphtheria is treated with diphtheria antitoxin (DAT) and penicillin or erythromycin. Mild candidal pharyngitis treatment is topical with clotrimazole troches or nystatin swish and swallow, whereas moderate to severe forms require systemic therapy with fluconazole.

Disposition

Patients with uncomplicated pharyngitis may be discharged and treated as outpatients. Patients with evidence of upper airway obstruction including stridor, difficulty managing secretions, and changes in phonation or severe systemic symptoms such as hypotension or altered mental status require consultation with an otolaryngologist and admission to the hospital.

LARYNGITIS

Foundations

Acute laryngitis is an inflammation of the larynx which is predominantly caused by viral infections. Chronic laryngitis is diagnosed after three weeks of continuous symptoms and may be due to gastroesophageal reflux, overuse of the voice, trauma, thermal and chemical burns, irritants, and allergic reactions.

Diffuse inflammation of the larynx caused predominantly by viral infection results in mucosal edema and laryngeal obstruction. It occurs most commonly in children ages 1 to 5 years and is more frequently seen in the winter and spring.[12] Immunocompromised patients can develop laryngitis caused by opportunistic fungal and viral infections. Patients with frequent use of inhaled corticosteroids are also at increased risk for fungal laryngitis.

Clinical Features

Laryngeal inflammation and edema cause sore throat, hoarse voice, inspiratory stridor, fever, and barking cough. Chronic laryngitis may additionally present with globus sensation and excessive throat clearing. Laryngitis may exist on its own or be part of a constellation of symptoms of upper airway infection including epiglottitis. Epiglottitis generally includes inflammation of the arytenoids and aryepiglottic folds and is sometimes referred to as supraglottitis. Inflammation of the glottis, arytenoids, and aryepiglottic folds increases the potential for acute upper airway obstruction, though the majority of cases of laryngitis are self-limited.

Differential Diagnosis for Emergency Presentation

The etiologies of laryngitis are similar to pharyngitis displayed in Table 61.1, with the majority being caused by the viruses that cause the common cold. The differential diagnosis includes infectious etiologies such as retropharyngeal abscess and noninfectious etiologies such as anaphylaxis, angioedema, tumor, thyroiditis, chemical or thermal injury, and foreign body.

Diagnostic Testing

There is no specific diagnostic testing for laryngitis, and the majority of cases will resolve within two weeks. Routine viral testing or bacterial cultures are not recommended.

Management

The majority of patients with laryngitis will improve without intervention. Systemic steroids have been shown to decrease symptom severity in pediatric patients; we recommend dexamethasone 0.6 mg/kg given via intramuscular, intravenous, or oral route depending on patient ability to tolerate oral intake and intravenous access availability with a maximum dose of 10 mg. Immunocompromised patients and patients with symptoms persisting for greater than two weeks should be referred to otolaryngology.

Disposition

Laryngitis can be treated on an outpatient basis. Exceptions occur when symptoms of laryngitis are occurring concomitantly with symptoms of supraglottitis/epiglottitis.

EPIGLOTTITIS

Foundations

Acute epiglottitis is an inflammation of the epiglottis and commonly the supraglottic region, including the arytenoids, base of the tongue, and vallecula. It is sometimes referred to as supraglottitis, but we will use the term epiglottitis. It is a rare but potentially life-threatening disease because of rapidly occurring airway obstruction and asphyxiation. Since the widespread adoption of the *Haemophilus influenzae* type B conjugate vaccine, epiglottitis is more common in adults than children.[13] Patients with diabetes, immunosuppression, and substance abuse issues, including tobacco and alcohol, are at increased risk for its

Fig. 61.5 Lateral neck x-ray with an edematous epiglottis: "thumb print sign."

development.[14,15] The majority of cases are caused by bacterial infection, including *Haemophilus influenzae* (type B and non-typeable), *Streptococcus pneumoniae,* and *Staphylococcus* subspecies. Noninfectious causes of epiglottitis include burns, trauma, and inhalational injury.[16]

Clinical Features

Symptoms are on a spectrum from sore throat, dysphagia, odynophagia, fever, hoarseness, and foreign body sensation in early stages to stridor, inability to manage secretions, and breathlessness with signs of airway compromise. Patients may sit forward in a sniffing position to maintain airway patency if there is sufficient swelling.

Differential Diagnosis

The differential diagnosis for epiglottitis includes infectious etiologies such as retropharyngeal abscess and noninfectious etiologies such as anaphylaxis, angioedema, tumor, thyroiditis, chemical or thermal injury, and foreign body.

Diagnostic Testing

Flexible laryngoscopy is the gold standard for diagnosis of epiglottitis and should be performed in patients presenting with symptoms of epiglottitis including sore throat, voice changes, and stridor. Lateral neck radiographs with an epiglottis width greater than 5.5 mm ("thumb sign" Fig. 61.5) have moderate sensitivity[17] but cannot be used to rule out epiglottitis.[18] We recommend against an initial diagnostic CT scan of the neck because of the need for supine positioning and potential for airway obstruction. Do not swab the posterior throat or use a tongue depressor because this may exacerbate the edema.

Management

All patients with epiglottitis should receive early airway evaluation assessing for voice changes, ability to manage secretions, position of comfort, and intravenous antibiotics. Antibiotic recommendations are outlined in Table 61.3. Corticosteroids can decrease supraglottic edema and may decrease the need for airway intervention we recommend dexamethasone 0.6 mg/kg with a maximum dose of 10 mg, though there is a paucity of randomized trials to guide steroid dosing.[10] Nebulized 2.25% racemic epinephrine diluted in normal saline and administered via jet nebulizer every 3 to 4 hours may decrease edema. Although oral or nasotracheal intubation is frequently unnecessary, the swelling typical of the epiglottitis (Fig. 61.6) can cause significant difficulty with intubation attempts and require an emergent surgical airway.[14] Given the high risk for failure to obtain an airway, we recommend early consultation with otolaryngology and anesthesia for intubation in the operating room. The need for emergency intubation increases in patients exhibiting dyspnea, stridor, drooling, tachypnea, and with the mouth held open or sitting forward in the sniffing position. If emergency intubation is required, we recommend awake fiberoptic intubation without the use of paralytics.

Disposition

Patients with epiglottitis require admission due to the risk for rapid airway obstruction. Patients who require emergency airway intervention or who are at high risk for deterioration should be admitted to the intensive care unit.

PERITONSILLITIS: PERITONSILLAR CELLULITIS AND ABSCESS

Foundations

Peritonsillar cellulitis is an infection of the peritonsillar tissue not associated with a collection of purulent material. Peritonsillar abscess is a collection of pus between the palatine tonsillar capsule and the superior constrictor muscle of the palatopharyngeus muscle. Fibrous septae in the peritonsillar space are responsible for directing the infection anteriorly and superiorly. Intratonsillar abscess refers to a collection of pus within the tonsillar parenchyma.

Peritonsillar abscess is most frequently seen in adults younger than 40 years old. Risk factors for developing a peritonsillar abscess include recent streptococcal tonsillitis, mononucleosis, obstruction or infection of the Weber glands, smoking, and dental or periodontal disease. There is an association with antiinflammatory medication use.[19,20] Infections are usually polymicrobial, but *Streptococcus pyogenes* is most frequently isolated. Recurrent peritonsillar abscesses have a high incidence of *Fusobacterium.*[21] Complications of peritonsillar abscess include abscess rupture into the airway and spread into the adjacent peritonsillar space.

Clinical Features

Symptoms include unilateral sore throat, odynophagia, dysphagia, fever, malaise, drooling, muffled voice, trismus, and ipsilateral otalgia. There is usually a delay between the time of symptom onset to abscess formation. Once an abscess has formed, the most common physical exam finding is a tense, erythematous, and edematous anterior tonsillar pillar with displacement of the infected tonsil and uvula towards the contralateral tonsil (Fig. 61.7). The tonsils and oral mucosa are usually erythematous with an exudate. Peritonsillar cellulitis may have a similar appearance without displacement of the tonsil and uvula. Associated findings are tonsillar edema, trismus, drooling, and tender cervical adenopathy. Peritonsillar abscess is usually unilateral, but bilateral abscesses occasionally occur.

TABLE 61.3 Microbiology[46] and Recommended Antimicrobials for Epiglottitis, Peritonsillar Abscess, Parapharyngeal Abscess, and Retropharyngeal Abscess

Condition	Organisms	Antimicrobial[a]
Epiglottitis/Supraglottitis	Normal Host: • Haemophilus influenzae • Streptococcus pneumoniae • Beta-hemolytic streptococci • Staphylococcus aureus • Neisseria meningitidis	• Ampicillin-sulbactam OR ceftriaxone AND • Vancomycin OR clindamycin (if low MRSA suspicion)
	Immunocompromised: • Above infection and • Pasteurella mul*tocida* • *Aspergillus* spp.	• Cefepime or piperacillin tazobactam AND Vancomycin
Parapharyngeal abscess Retropharyngeal abscess Peritonsillar abscess Ludwig angina	Community: • Streptococcus pyogenes • Staphylococcus aureus • Streptococcus milleri group • Arcanobacterium hemolyticum • Mixed oral flora	Ampicillin-sulbactam OR ceftriaxone and metronidazole OR Clindamycin and levofloxacin
	Immunocompromised	Cefepime and metronidazole OR Piperacillin tazobactam AND Vancomycin
	MRSA risk (eg, IVDA, diabetes)	Add vancomycin or linezolid

MRSA, methicillin-resistant *staphylococcus aureus*; *IVDA,* intravenous drug abuse.
[a]Renally dose antibiotics.

Differential Diagnosis for Emergency Presentation

The differential diagnosis for peritonsillitis is similar to retropharyngeal abscess displayed in Box 61.2. This includes infectious and noninfectious inflammation or infiltration of any of the surrounding structures including peritonsillar cellulitis, benign hypertrophy, lymphoma or tumor, pharyngitis, deep neck infection, carotid aneurysm, epiglottitis, and mononucleosis.

Diagnostic Testing

Although the diagnosis may be made clinically, ultrasound and contrast-enhanced CT aid in differentiation from cellulitis, characterization of the abscess, and identification of deep space infections. Transcervical ultrasound is useful in children.[22-24] While operator dependent,[25] a linear probe under the mandible can identify peritonsillar fluid collections. In pediatric patients, an ultrasound first approach can decrease the length of stay without increasing the frequency of return visits.[11,26-28] In older patients without trismus, intraoral ultrasound is a useful tool in the identification of an abscess (Fig. 61.8). In both transcervical and intraoral modalities the limitations of ultrasound include misinterpretation of a parapharyngeal abscess as a peritonsillar abscesses. Contrast-enhanced CT is recommended in patients in whom ultrasound is not technically possible or limited, swelling is significantly greater than anticipated in a patient with an otherwise mild presentation, or when there is a concern for a deep space infection (Fig. 61.9A and B).

Management

Peritonsillar cellulitis and intratonsillar abscess require medical therapy alone. Antibiotic options are listed in Table 61.3. Adjunctive treatment with a single dose of corticosteroids, such as dexamethasone 0.6 mg/kg with a maximum dose of 10 mg appears to be safe and effective, decreasing pain, edema, trismus, and leads to a faster recovery without deleterious effects.[11,26-28] In cases of a small peritonsillar abscess (<1 cm) antibiotics and steroids alone may be sufficient.

Larger abscesses benefit from drainage.[29] Needle aspiration or surgical incision and drainage can usually be accomplished in an awake patient with the use of topical lidocaine or benzocaine for anesthesia. Ultrasound guidance can increase diagnostic yield and identify the carotid artery during the procedure. Although needle aspiration is less painful and can be easier to perform, recurrence rates in needle aspiration may be higher compared with surgical incision and drainage.[30] Routine culture of purulent material is not indicated unless the patient is diabetic or immunocompromised, or if there are recurrent infections.

Disposition

Most patients are successfully managed as outpatients with a course of antibiotics after treatment in the emergency department. Hospital admission should be considered in patients who are toxic appearing, require intravenous hydration and analgesia, show signs of parapharyngeal extension on CT scan, or who are immunocompromised with multiple comorbidities.

LUDWIG ANGINA

Foundations

Ludwig angina is a rapidly progressive, bilateral, gangrenous cellulitis of all submandibular spaces that can rapidly lead to death within hours. Ludwig angina is a bilateral process because of the communication among the open posterior aspect of the submandibular spaces. Dental

origin from the mandibular molars is the most common cause of Ludwig angina. Mandibular molar roots insert below the mylohyoid muscle on the mandible, and the lingual aspect of the mandible is a thin osseous structure. Additional causes of Ludwig angina are mandibular fracture, oral trauma (tongue piercing, lingual laceration, iatrogenic lacerations from intubation), secondary infection of oral malignancy, suppurative parotitis, and adjacent head and neck infections. Infection may spread posteriorly between the submandibular and parapharyngeal spaces via the styloglossus muscle leading to deep neck infections. Ludwig angina occurs more frequently in immunocompromised and diabetic patients.

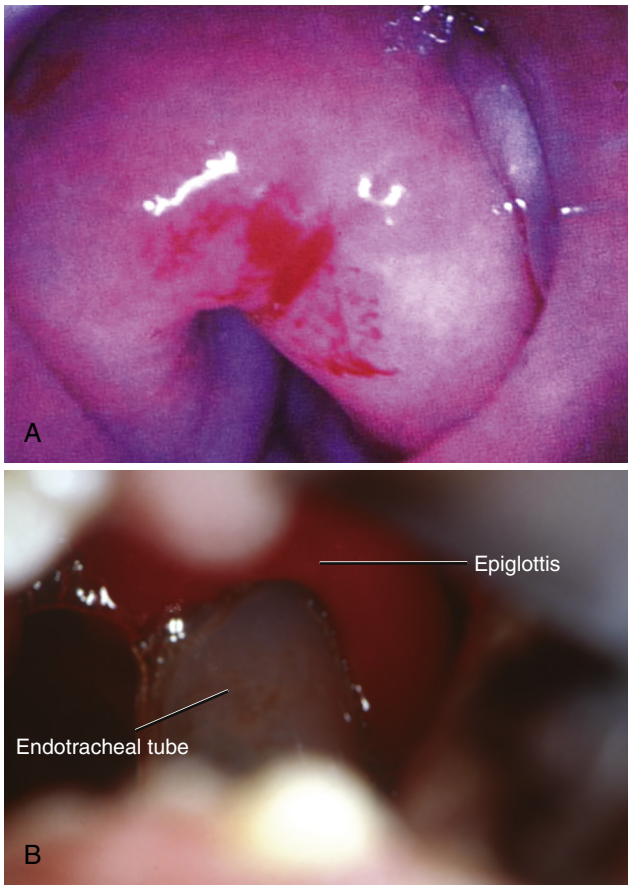

Fig. 61.6 Epiglottitis. Image A shows an enlarged and swollen epiglottis without visible vocal chords. Image B shows an enlarged epiglottis over an endotracheal tube. (From Hannallah RS, Brown KA, Verghese ST. Otorhinolaryngologic procedures. In: Coté CJ, Lerman J, Anderson BJ, eds. *A Practice of Anesthesia for Infants and Children*, 6th edition. Philadelphia: Elsevier; 2019: Figure 33.23)

Clinical Features

Patients may report recent dental infection, dental procedure, or dental caries with progressive pain and swelling. Patients present with dysphagia, odynophagia, drooling, swelling of the floor of the mouth, tongue displacement, muffled voice, fever, and neck stiffness. The most common physical exam findings are submental and submandibular swelling with protrusion of the tongue. There may be elevation of the floor of the mouth with a woody consistency on palpation. The patient's neck may have tense brawny edema from the submandibular region to the hyoid, commonly described as a bull neck. As the cellulitis progresses, the patient may have trismus, inability to manage secretions, dyspnea, a hoarse voice, and progressive anxiety secondary to airway impingement. Cervical adenopathy and palpable fluctuance are usually absent.

Differential Diagnosis for Emergency Presentation

The differential diagnosis includes viral and bacterial infections of the oropharyngeal region including deep space infections such as pharyngeal or retropharyngeal abscess, parotid or submandibular gland abscess, oropharyngeal tumors, sublingual hematomas, glossal and posterior oropharyngeal angioedema, and laryngeal diphtheria.

Diagnostic Testing

The diagnosis of Ludwig angina is made clinically. Contrast-enhanced CT scan may aid in the identification of deep neck structures involved and the extent of infection.

Management

Asphyxia from progressive edema is the leading cause of death in Ludwig's angina. Airway management is essential when there are any signs of airway compromise, including dyspnea, tachypnea, inability to manage secretions, agitation, stridor, and progressive edema. An awake fiberoptic nasal or oral intubation with a flexible bronchoscope after adequate topicalization is recommended. Oral laryngoscopy may be difficult because of edema and tongue displacement. Paralysis prior to securing the airway is not advised.

A surgical airway may be required if endotracheal intubation cannot be accomplished. Surgical airways can be more challenging

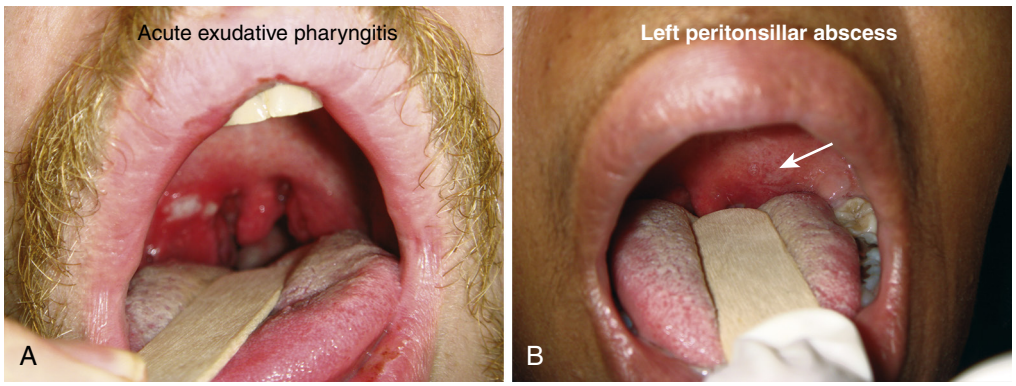

Fig. 61.7 Exudative pharyngitis versus peritonsillar abscess. Photograph A depicts acute exudative pharyngitis with tonsillar enlargement, erythema, and exudate. Photograph B shows left tonsillar bulging displacing the uvula to the right. (From Riviello RJ. Otolaryngologic procedures. In: Roberts JR, Custalow CB, Thomsen TW, eds. *Roberts and Hedges' Clinical Procedures in Emergency Medicine and Acute Care*, 7th edition. Philadelphia: Elsevier; 2019: Figure 63.7)

BOX 61.2 Differential Diagnosis of Suppurative Pharyngeal Infections

Retropharyngeal cellulitis and retropharyngeal abscess
Parapharyngeal abscess
Peritonsillar abscess
Retropharyngeal tumors
Tendinitis of the longus colli muscle
Meningitis
Hematoma secondary to trauma
Carotid artery aneurysm

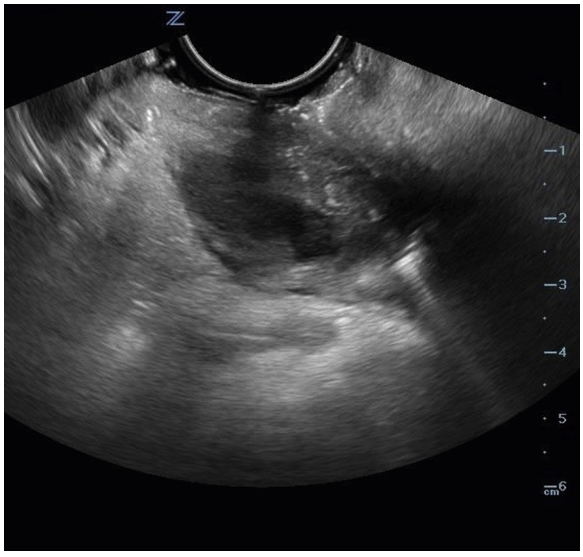

Fig. 61.8 Intraoral ultrasound with an endocavitary probe demonstrating a hypoechoic collection consistent with a peritonsillar abscess. (Image courtesy of Dr. Lindsay Reardon.)

in Ludwig angina due to anterior neck distortion from edema and the risk of spreading infection from dissecting tissue planes (Fig. 61.10). Intravenous antibiotics are of paramount importance once the patency of the airway is ensured (see Table 61.3.) There usually is not a role for surgical intervention with the exception of an infected tooth extraction, débridement of necrotizing infection, or drainage of purulent collections.

Disposition

Patients with Ludwig angina should be admitted to the intensive care unit.

RETROPHARYNGEAL ABSCESS

Foundations

Retropharyngeal abscess is an infection of the deep neck spaces that can lead to life-threatening complications, including airway compromise and mediastinal spread of infection. The retropharyngeal space is posterior to the hypopharynx and esophagus in the midline neck, immediately anterior to the danger and prevertebral spaces that extend to the diaphragm and coccyx, respectively (Fig. 61.11). It is bordered laterally by the carotid sheath/parapharyngeal space. Infections may spread from one deep space to another and involve the carotid sheath, mediastinum, and chest.

Retropharyngeal abscesses most commonly occur in children younger than 5 years old with a male predominance.[31] Children have lymph nodes present in the retropharyngeal space that are the lymphatic drainage of the nasal cavity, paranasal sinuses, oropharynx, hypopharyngeal space, middle ear, and eustachian tubes but atrophy prior to puberty. Upper respiratory tract infections, otitis media, and sinusitis can lead to suppurative lymphadenitis and abscess formation.[31] A minority of children with Kawasaki disease also have a deep neck infection.[32,33] In adults, retropharyngeal abscess formation can be caused by penetrating trauma, foreign body (fish or chicken bones) or iatrogenic instrumentation (feeding tube, laryngoscope, suctioning),

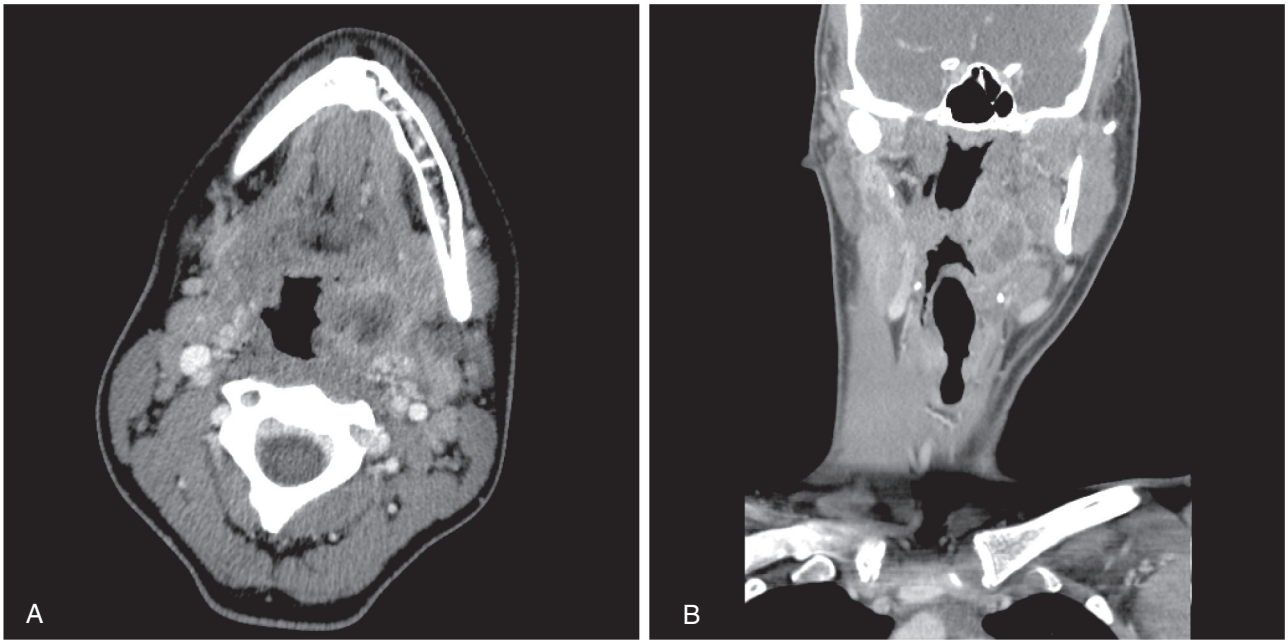

Fig. 61.9 Contrast-enhanced axial (A) and coronal (B) CT image of a left-sided peripherally enhancing and centrally hypoattenuating peritonsillar abscess. There is mild mass effect on the oropharyngeal airway and mild inflammation of the fat in the left parapharyngeal space.

Fig. 61.10 Anterior neck swelling and erythema in Ludwig angina. (From Bernardoni B, Grosso R, Powell E, et al. Case study in critical care transport: a 51-year-old male with Ludwig angina. *Air Med J* 2017;36(2):45-48.)

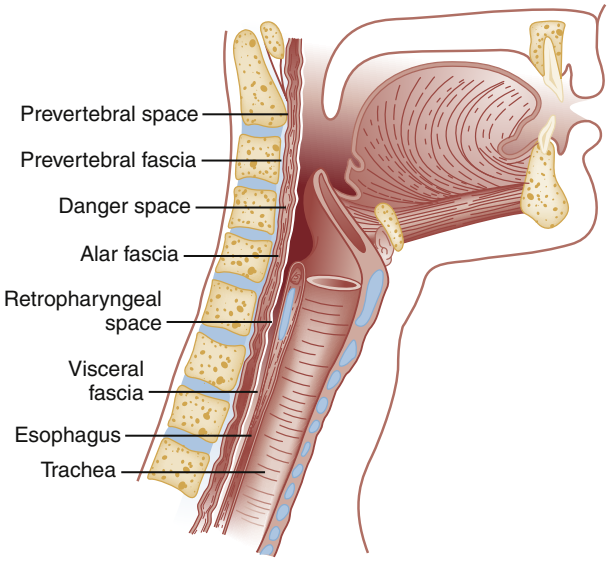

Fig. 61.11 Anatomic illustration of the lateral view of the neck showing of the prevertebral space, danger space, and retropharyngeal space.

spread from adjacent infections (dental or peritonsillar), hematologic spread of infections, and infections of the spine communicating with the prevertebral space. Risk factors include immunosuppression, chronic steroid use, diabetes, and human immunodeficiency virus. Diabetic patients have a higher prevalence of multispace spread of infection.[34] Adolescents and adults with a prior tonsillectomy also have an increased risk of deep neck infections.[35]

Microbiology of retropharyngeal abscesses is polymicrobial with a mixture of aerobes and anaerobes as described in Table 61.3. Intravenous drug abuse increases the frequency of methicillin-resistant *Staphylococcus aureus* infections.[36] A history of immunocompromise or prior nosocomial infection increases the risk of drug-resistant organisms as well as the risk for tuberculous or fungal infection. Complications from

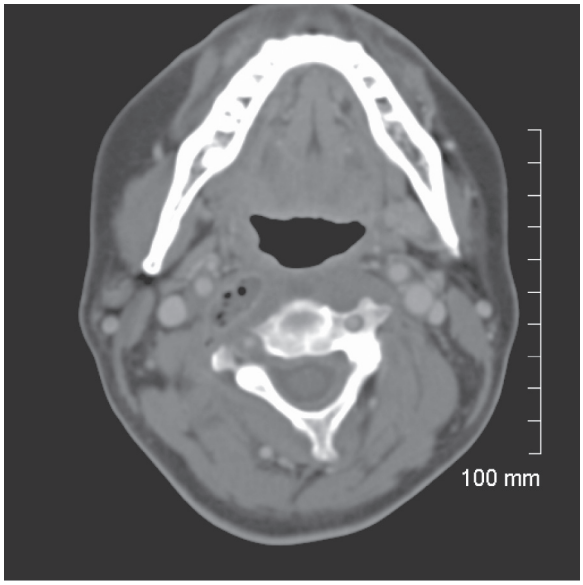

Fig. 61.12 Axial CT scan image demonstrating right sided retropharyngeal abscess.

retropharyngeal abscess include airway compromise, abscess rupture leading to aspiration of pus, and involvement of the carotid sheath. The carotid sheath is surrounded by deep fascia and includes the carotid artery, internal jugular vein, and vagus nerve, and thus infection can result in compromise of any of these structures including arterial erosion, aneurysm, Lemierre syndrome, palsy of cranial nerves IX–XII, and mediastinitis.

Clinical Features

Patients with a retropharyngeal abscess may present with fever, sore throat, dysphagia, odynophagia, drooling, pain, hoarseness, trismus, and neck stiffness. As the abscess and inflammation become more severe, patients may appear toxic and hold their heads in a sniffing position while sitting upright. Advanced cases can cause ligamentous and osseous destruction, resulting in torticollis and severe pain. The patient may experience referred pain to the posterior neck and shoulder when swallowing.

Physical examination findings of tender cervical lymphadenopathy, neck swelling, torticollis, and fever are common. Retropharyngeal cellulitis or early abscess present with diffuse edema and erythema of the posterior pharynx. Depending on the structures involved, patients may present with trismus, making intraoral physical examination difficult and not advised. Tenderness with external laryngeal and tracheal manipulation is commonly present.

Differential Diagnosis for Emergency Presentation

The differential diagnosis for retropharyngeal abscess is similar to peritonsillar abscess and parapharyngeal abscess displayed in Box 61.2.

Diagnostic Testing

Contrast-enhanced CT scan is the preferred diagnostic test for the identification of retropharyngeal abscess. CT findings are characterized by a fluid collection with central hypodensity and complete ring enhancement with scalloping (Figs. 61.12 and 61.13). Fat stranding and edema characterized by low-density thickening without peripheral enhancement may represent an early phase of infection before an abscess develops. If contrast-enhanced CT imaging is not possible, additional studies may include non-contrast-enhanced CT scan and ultrasound imaging. Lateral neck radiographs taken during inspiration

while the neck is extended will suggest a pathologic process if there is swelling but are unlikely to differentiate abscess from edema and the structures involved. On lateral neck radiographs, a retropharyngeal space measured from the anteroinferior aspect of the second vertebral body to the posterior pharyngeal wall that is wider than 7 mm (children and adults) or a retrotracheal space measured at the sixth vertebral body more than 14 mm in children and 22 mm in adults is abnormal and suggests an inflammatory process.

Management

Initial management should focus on airway stabilization and broad-spectrum intravenous antibiotics. Intravenous antibiotic selection is displayed in Table 61.3. Short-term, high-dose, adjunctive corticosteroids such as dexamethasone 0.6 mg/kg with maximum dose of 10 mg are often used with antibiotics and may be associated with a decreased need for surgical drainage.[11,34,37] In cases of a retropharyngeal abscess less than 2 cm, medical management alone may be sufficient. In immunosuppressed patients and those living in areas with high rates of tuberculosis with insidious onset of symptoms and a high suspicion for tuberculous or fungal infection, consultation with an infectious disease specialist along with an otolaryngologist is recommended. In cases with evidence of ligamentous and osseous destruction, neck immobilization and further dedicated cervical spine imaging may be required.

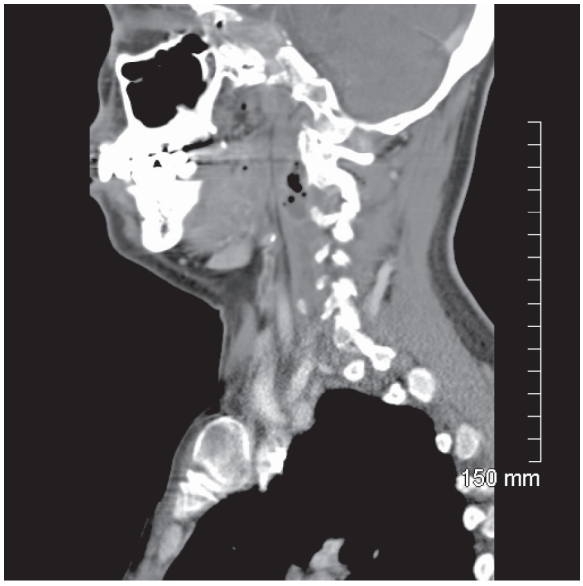

Fig. 61.13 Sagittal CT scan demonstrating right sided retropharyngeal abscess.

Disposition

Patients with a retropharyngeal abscess should be admitted to the hospital after otolaryngology consultation in the emergency department. In general, these patients are at high risk for airway compromise and should be admitted to an intensive care unit. If the patient has early, mild disease with a stable airway exam they may be suitable for admission to a floor bed.

PARAPHARYNGEAL ABSCESS

Foundations

Parapharyngeal abscess is a serious infection with close proximity to the airway, carotid sheath, and mediastinum. Seen in Figure 61.14, the parapharyngeal space is found on either side of the neck and extends from the skull base to the styloglossus muscle at the angle of the mandible. The posterior aspect of the parapharyngeal space is separated from the midline retropharyngeal space and danger space by a fascial plane. The carotid sheath is found in the retrostyloid compartment of the parapharyngeal space and contains the common carotid artery, internal jugular vein, the sympathetic plexus, cranial nerves IX through XII, and lymph nodes. Parapharyngeal space abscesses usually emanate from dental or peritonsillar infections with similar organisms. Spread from adjacent infections, suppurative lymphadenitis, iatrogenic via

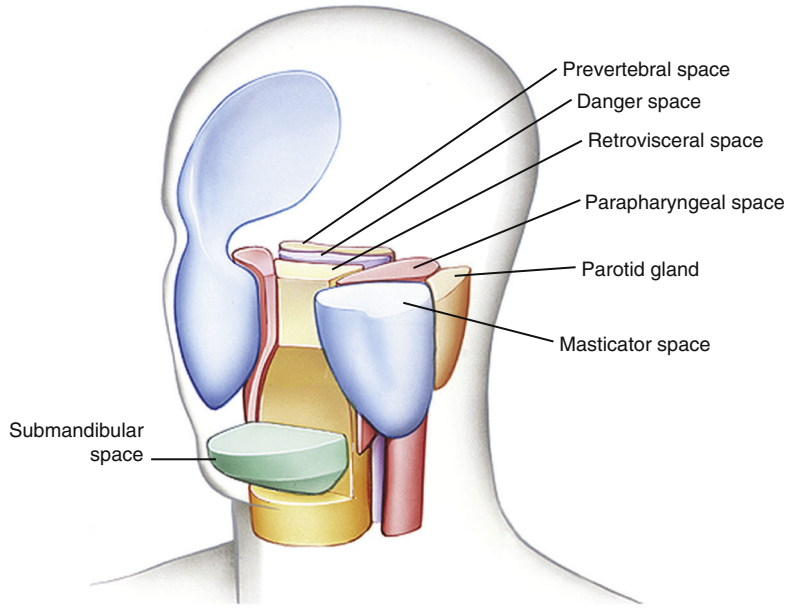

Fig. 61.14 Block diagram of the deep spaces in the neck showing the relationship of the parapharyngeal space, danger space, and prevertebral space. (From Som PM, Curtin HD. Parapharyngeal and masticator space lesions. In: Som PM, Curtin HD, eds. *Head and Neck Imaging*, 5th edition. St. Louis: Mosby, Inc.; 2011.)

local infiltration for surgical procedures or nerve blocks, infected neck tumors or cysts, parotitis, and sinusitis are rare causes.

Complications of a parapharyngeal abscess range from airway obstruction secondary to edema, neck or mediastinal spread, vascular involvement, and nerve compromise. Abscess rupture with pus into the airway can result in pneumonia, lung abscess or empyema. Mediastinal spread is possible through connection with the retropharyngeal space and danger space. Involvement of the carotid sheath has the potential to affect the sympathetic chain ganglia causing an ipsilateral Horner syndrome and neuropathies of cranial nerves IX–XII. Communication with the carotid artery may cause erosion, aneurysm, and rupture. Any signs of bleeding should be investigated with CT or MR angiography. Lemierre syndrome should be considered when sore throat or tonsillitis is followed by sepsis and multisystem involvement. Proptosis, impaired extraocular movement, or pupillary changes should prompt consideration of cavernous sinus thrombosis, a life-threatening complication with spread of infection through the ophthalmic venous system. Parapharyngeal abscess may have local spread causing mandibular osteonecrosis and parotid abscess.

Clinical Features

Symptoms include pain and swelling of the neck, odynophagia, pain with mastication, and torticollis if the sternocleidomastoid muscle is involved. Physical exam findings vary depending on the extent of the infection. The sternocleidomastoid muscle may obscure exam findings of an abscess. Fever, trismus, edema, and pain with movement of the sternocleidomastoid muscle may be observed. Patients with infection involving the anterior aspect of the parapharyngeal space may have medial tonsillar displacement and pharyngeal edema.

Differential Diagnosis for Emergency Presentation

The differential diagnosis for parapharyngeal abscess is similar to peritonsillar abscess and retropharyngeal abscess displayed in Box 61.2.

Diagnostic Testing

Contrast-enhanced CT is the preferred diagnostic study (Fig. 61.15). External ultrasound can provide useful information but may be limited in parapharyngeal abscess. Dedicated CT angiography or Doppler assessment are recommended if vascular complications are suspected.

Management

Initial management is focused on airway management, early treatment with broad-spectrum antibiotics listed in Table 61.3, dexamethasone 0.6 mg/kg intravenous or intramuscular with maximum dose 10 mg, and consultation with an otolaryngologist. Airway management is essential when there are any signs of airway compromise, including dyspnea, tachypnea, and inability to manage secretions, agitation, or stridor. An awake fiberoptic nasal or oral intubation with a flexible bronchoscope after adequate topicalization is recommended. Paralysis prior to securing the airway is not advised.

Disposition

Consultation with an otolaryngologist and admission to the hospital are recommended. Admission to the ICU is recommended should there be signs of airway or vascular compromise.

RHINOSINUSITIS

Foundations

Rhinosinusitis is inflammation of the upper airways and paranasal sinuses associated with nasal discharge, facial pain or pressure, nasal blockade, and a sense of fullness mostly associated with an infectious source (viral, bacterial, fungal.) Acute rhinosinusitis is classified as having symptoms for fewer than 4 weeks. Viral etiologies tend to have symptoms that peak and resolve in a few days, whereas bacterial etiologies tend to last longer than 10 days with persistent symptoms or will worsen after a period of improving symptoms. Rhinovirus, adenovirus, influenza or parainfluenza virus are the most common precipitants.[38] *Streptococcus pneumoniae*, non-typable *Haemophilus influenzae*, and *Moraxella catarrhalis* are the most common bacterial causes of maxillary sinusitis.[32,39] *Staphylococcus aureus*, gram-negative bacilli, and *Streptococcus* spp. are associated with subacute, chronic, and health care–associated sinusitis.

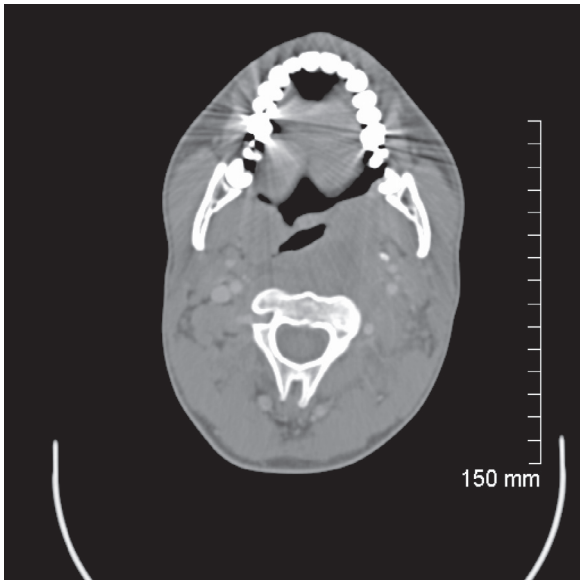

Fig. 61.15 CT scan showing a left-sided parapharyngeal abscess.

Structural abnormalities that predispose patients to acute rhinosinusitis include nasal septal deviation, infraorbital ethmoid air cells larger than 3 mm, accessory ostia in the common drainage pathway, conchae bullosa, oroantral fistula, and maxillary dental disease. Viral infection may be antecedent to bacterial infection because mucosal swelling and inflammation impair drainage and promote ciliary dyskinesia providing an environment for bacterial growth.[32] This may be the etiology of the double sickening phenomenon when patients report improvement of symptoms followed by worsening. It is impossible to differentiate between the viral and bacterial etiologies based on symptoms alone, and thus it is difficult to characterize the preponderance of antecedent infection. Rarely, acute rhinosinusitis may be attributed to periapical infection of the maxillary molars with direct extension into the sinus space.[38] Chronic rhinosinusitis causes symptoms for greater than twelve weeks. Chronic rhinosinusitis is not associated with structural abnormalities and is more commonly related to inflammatory disorders. Noninfectious etiologies of rhinosinusitis include allergic etiologies and vascular engorgement associated with pregnancy mimicking rhinosinusitis.

Complications from rhinosinusitis are extremely rare, but include cellulitis, meningitis, and orbital or intracranial abscess. Patients with poorly controlled diabetes and immunosuppression are at increased risk of invasive fungal species including *Aspergillus* spp., mucormycosis, and *Fusarium* spp.

Clinical Features

Symptoms of rhinosinusitis include purulent nasal discharge, facial pain or pressure, posterior nasal drip, and decreased sense of smell. These symptoms may be associated with sore throat, hoarseness,

cough, headache, fatigue, malaise, and fever. Differentiation between viral and bacterial rhinosinusitis is not possible based on any one symptom and relies mostly upon the time course of symptoms. Bacterial causes are more likely to be associated with prolonged symptoms lasting longer than 10 days, frequently with a worsening of symptoms after initial improvement, severe symptoms for greater than 3 to 4 days, and unilateral maxillary tooth or cheek pain. Allergic rhinosinusitis is more likely to be associated with watery rhinorrhea, ocular itching with watery discharge, and sneezing.[38,40] On exam, patients have purulent nasal discharge and may have pain with percussion of sinuses or maxillary teeth. High fever, nasal crusting or severe facial pain in immunocompromised patients is concerning for invasive infection.

Differential Diagnosis for Emergency Presentation

Differential diagnosis includes allergic rhinitis, dental infection, headache syndromes (including cluster headache, tension headache, and vascular headache), tumor, and intracranial abscess.

Diagnostic Testing

The diagnosis of acute rhinosinusitis is clinical. The diagnosis of bacterial rhinosinusitis requires one of the three criteria[5,41]: at least 10 days of persistent symptoms without improvement, three to four days of severe symptoms including a fever greater than 39 degrees Celsius with nasal discharge or facial pain without improvement, or onset of progressive symptoms with worsening symptoms after initial improvement. Visual examination of the nares directly or via nasal endoscopy may reveal swelling of the turbinates and purulent appearing discharge. Routine CT imaging is not indicated. However, if performed for other reasons, CT may reveal thickening of the walls of both nasal passages, engorgement of inferior turbinates, obstruction of the ethmoid infundibulum, and abnormalities of maxillary, sphenoid, and frontal sinuses.

The gold standard for diagnosis of bacterial rhinosinusitis is culture of secretions obtained via sinus puncture. This is not recommended in the emergency department. Culture taken via sinus puncture in patients with radiographic features of rhinosinusitis or with purulence of discharge has been found to correlate with positive cultures 50% of the time and the sinus puncture is painful and carries potential risks to the patient.[32]

Management

The vast majority of acute rhinosinusitis is self-resolving and management should focus on symptom management and patient education. We recommend symptom management with acetaminophen 650 mg (15 mg/kg for children) every 6 to 8 hours and ibuprofen 800 mg (10 mg/kg for children) every 8 hours, nasal irrigation with saline 1 to 2 sprays each nostril every 4 hours, and intranasal corticosteroids such as fluticasone propionate or mometasone furoate 2 sprays in each nostril once daily.[38,42,43] Data on the efficacy of decongestants and mucolytics are lacking and they are not recommended. Systemic corticosteroids for acute rhinosinusitis are not recommended. Routine use of antihistamines is not recommended unless there are additional allergic symptoms.

Given the risks and lack of benefit of routine antibiotic use in rhinosinusitis, antibiotics should only be used for patients who meet the definition of bacterial rhinosinusitis.[44] Overall, the use of antibiotics is contested, and the benefits are small, with cure rates at 7 to 15 days slightly higher in patients treated with antibiotics than in those treated with placebo.[38] In bacterial rhinosinusitis there is controversy as to when antibiotics should be initiated. Some guidelines propose initiation at the time of diagnosis[41] while others advocate for a watchful waiting approach for well appearing patients and initiation of antibiotics if symptoms do not improve within 7 days of diagnosis.[45] For ED patients we recommend starting the antibiotics at the time of ED presentation. Antibiotics should be given to patients with multiple comorbidities, immunosuppression, prior sinus surgery, or symptoms not isolated to the maxillary sinus. Amoxicillin 500 mg three times daily or amoxicillin-clavulanate 875 mg/125 mg twice daily for 5 days are first-line antibiotics. Amoxicillin-clavulanate should be used if the patient smokes, has diabetes, has had recent antibiotics, is older than 65 years, or is a health care worker. Doxycycline 100 mg twice daily may be given for penicillin allergic patients. Levofloxacin and moxifloxacin are also second-line agents but should be reserved for patients without other options because of the side effect profile. Avoid macrolides (azithromycin), trimethoprim-sulfamethoxazole, and second- and third-generation cephalosporins because of increasing *S. pneumoniae* resistance.

The length of antibiotic treatment is also controversial with ranges of 5 to 14 days. We recommend the initial treatment course of 5 days because there are similar treatment effects with reduced exposure to antibiotics. Children should be treated for 10 days. For adults with recurrent episodes of rhinosinusitis or prior antibiotic exposure the treatment course should be 10 days. Chronic sinusitis is not likely to be from an infectious etiology and should be managed with saline rinses and intranasal corticosteroids. If there is concern for an invasive fungal infection typified by severe rapidly progressive symptoms in patients with immunosuppression, patients should have emergent consultation with an otolaryngologist and begin empiric therapy with intravenous antifungal.

Disposition

Uncomplicated rhinosinusitis can be discharged home. Admission should be considered in patients presenting with systemic evidence of illness, immunosuppression, or complications. Referral to an otolaryngologist is indicated in patients presenting with recurrent acute rhinosinusitis (greater than 4 distinct episodes of rhinosinusitis within a calendar year).[46]

The references for this chapter can be found online at ExpertConsult.com.

REFERENCES

1. Shulman ST, Bisno AL, Clegg HW, et al. Clinical practice guideline for the diagnosis and management of group a streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. *Clin Infect Dis Off Publ Infect Dis Soc Am*. 2012;55(10):1279–1282. https://doi.org/10.1093/cid/cis847.
2. Berkley J. Management of pharyngitis. *Circulation*. 2018;138(18):1920–1922. https://doi.org/10.1161/CIRCULATIONAHA.118.035900.
3. Burckhardt F, Hoffmann D, Jahn K, et al. Oropharyngeal tularemia from freshly pressed grape must. *N Engl J Med*. 2018;379(2):197–199. https://doi.org/10.1056/NEJMc1800353.
4. Maurin M, Gyuranecz M. Tularaemia: clinical aspects in Europe. *Lancet Infect Dis*. 2016;16(1):113–124. https://doi.org/10.1016/S1473-3099(15)00355-2.
5. Harris AM, Hicks LA, Qaseem A. High Value Care Task Force of the American College of Physicians and for the Centers for Disease Control and Prevention. Appropriate antibiotic use for acute respiratory tract infection in adults: advice for high-value care from the American College of Physicians and the Centers for Disease Control and Prevention. *Ann Intern Med*. 2016;164(6):425–434. https://doi.org/10.7326/M15-1840.
6. Rezk E, Nofal YH, Hamzeh A, Aboujaib MF, AlKheder MA, Hammad MFA. Steroids for symptom control in infectious mononucleosis. *Cochrane Database Syst Rev*. 2015;11. https://doi.org/10.1002/14651858.CD004402.pub3.
7. Uyeki TM, Bernstein HH, Bradley JS, et al. Clinical practice guidelines by the infectious diseases society of America: 2018 update on diagnosis, treatment, chemoprophylaxis, and institutional outbreak management of seasonal influenzaa. *Clin Infect Dis Off Publ Infect Dis Soc Am*. 2019;68(6):895–902. https://doi.org/10.1093/cid/ciy874.
8. Hayward GN, Hay AD, Moore MV, et al. Effect of oral dexamethasone without immediate antibiotics vs placebo on acute sore throat in adults. *J Am Med Assoc*. 2017;317(15):1535–1543. https://doi.org/10.1001/jama.2017.3417.
9. Sadeghirad B, Siemieniuk RAC, Brignardello-Petersen R, et al. Corticosteroids for treatment of sore throat: systematic review and meta-analysis of randomised trials. *BMJ*. 2017;358:j3887. https://doi.org/10.1136/bmj.j3887.
10. Kent S, Hennedige A, McDonald C, et al. Systematic review of the role of corticosteroids in cervicofacial infections. *Br J Oral Maxillofac Surg*. 2019;57(3):196–206. https://doi.org/10.1016/j.bjoms.2019.01.010.
11. Burton MJ, Glasziou PP, Chong LY, Venekamp RP. Tonsillectomy or adenotonsillectomy versus non-surgical treatment for chronic/recurrent acute tonsillitis. *Cochrane Database Syst Rev*. 2014;11:CD001802. https://doi.org/10.1002/14651858.CD001802.pub3.
12. Reveiz L, Cardona AF. Antibiotics for acute laryngitis in adults. *Cochrane Database Syst Rev*. 2015;2015(5). https://doi.org/10.1002/14651858.CD004783.pub5.
13. Hanna J, Brauer PR, Berson E, Mehra S. Adult epiglottitis: trends and predictors of mortality in over 30 thousand cases from 2007 to 2014. *Laryngoscope*. 2019;129(5):1107–1112. https://doi.org/10.1002/lary.27741.
14. Galitz YS, Shoffel-Havakuk H, Cohen O, Halperin D, Lahav Y. Adult acute supraglottitis: analysis of 358 patients for predictors of airway intervention. *Laryngoscope*. 2017;127(9):2106–2112. https://doi.org/10.1002/lary.26609.
15. Ovnat Tamir S, Marom T, Barbalat I, Spevak S, Goldfarb A, Roth Y. Adult supraglottitis: changing trends. *Eur Arch Oto-Rhino-Laryngol*. 2015;272(4):929–935. https://doi.org/10.1007/s00405-014-3464-x.
16. Lichtor JL, Roche Rodriguez M, Aaronson NL, Spock T, Goodman TR, Baum ED. Epiglottitis: it hasn't gone away. *Anesthesiology*. 2016;124(6):1404–1407. https://doi.org/10.1097/ALN.0000000000001125.
17. Fujiwara T, Miyata T, Tokumasu H, Gemba H, Fukuoka T. Diagnostic accuracy of radiographs for detecting supraglottitis: a systematic review and meta-analysis. *Acute Med Surg*. 2017;4(2):190–197. https://doi.org/10.1002/ams2.256.
18. Lee SH, Yun SJ, Kim DH, Jo HH, Ryu S. Do we need a change in ED diagnostic strategy for adult acute epiglottitis? *Am J Emerg Med*. 2017;35(10):1519–1524. https://doi.org/10.1016/j.ajem.2017.04.039.
19. Lepelletier D, Pinaud V, Le Conte P, et al. Is there an association between prior anti-inflammatory drug exposure and occurrence of peritonsillar abscess (PTA)? A national multicenter prospective observational case-control study. *Eur J Clin Microbiol Infect Dis Off Publ Eur Soc Clin Microbiol*. 2017;36(1):57–63. https://doi.org/10.1007/s10096-016-2770-1.
20. Piroulas C, Devillers L, Souty C, Sicsic J, Boisnault P, François M. Non-steroids anti-inflammatory drugs and risk of peritonsillar abscess in pharyngitis: a French longitudinal study in primary care. *Fam Pract*. 2019;36(4):425–430. https://doi.org/10.1093/fampra/cmy111.
21. Ali SA, Kovatch KJ, Smith J, et al. Predictors of intratonsillar versus peritonsillar abscess: a case-control series. *Laryngoscope*. 2019;129(6):1354–1359. https://doi.org/10.1002/lary.27615.
22. Bandarkar AN, Adeyiga AO, Fordham MT, Preciado D, Reilly BK. Tonsil ultrasound: technical approach and spectrum of pediatric peritonsillar infections. *Pediatr Radiol*. 2016;46(7):1059–1067. https://doi.org/10.1007/s00247-015-3505-7.
23. Halm BM, Ng C, Larrabee YC. Diagnosis of a peritonsillar abscess by transcutaneous point-of-care ultrasound in the pediatric emergency department. *Pediatr Emerg Care*. 2016;32(7):489–492. https://doi.org/10.1097/PEC.0000000000000843.
24. Fordham MT, Rock AN, Bandarkar A, et al. Transcervical ultrasonography in the diagnosis of pediatric peritonsillar abscess. *Laryngoscope*. 2015;125(12):2799–2804. https://doi.org/10.1002/lary.25354.
25. Coquia SF, Hamper UM, Holman ME, et al. Visualization of the oropharynx with transcervical ultrasound. *Am J Roentgenol*. 2015;205(6):1288–1294. https://doi.org/10.2214/AJR.15.14299.
26. Hur K, Zhou S, Kysh L. Adjunct steroids in the treatment of peritonsillar abscess: a systematic review. *Laryngoscope*. 2018;128(1):72–77. https://doi.org/10.1002/lary.26672.
27. Koçak HE, Acıpayam H, Elbistanlı MS, et al. Is corticosteroid a treatment choice for the management of peritonsillar abscess? *Auris Nasus Larynx*. 2018;45(2):291–294. https://doi.org/10.1016/j.anl.2017.04.008.
28. Lee YJ, Jeong YM, Lee HS, Hwang SH. The efficacy of corticosteroids in the treatment of peritonsillar abscess: a meta-analysis. *Clin Exp Otorhinolaryngol*. 2016;9(2):89–97. https://doi.org/10.21053/ceo.2014.01851.
29. Souza DLS, Cabrera D, Gilani WI, et al. Comparison of medical versus surgical management of peritonsillar abscess: a retrospective observational study. *Laryngoscope*. 2016;126(7):1529–1534. https://doi.org/10.1002/lary.25960.
30. Chang BA, Thamboo A, Burton MJ, Diamond C, Nunez DA. Needle aspiration versus incision and drainage for the treatment of peritonsillar abscess. *Cochrane Database Syst Rev*. 2016;12:CD006287. https://doi.org/10.1002/14651858.CD006287.pub4.
31. Woods CR, Cash ED, Smith AM, et al. Retropharyngeal and parapharyngeal abscesses among children and adolescents in the United States: epidemiology and management trends, 2003-2012. *J Pediatric Infect Dis Soc*. 2016;5(3):259–268. https://doi.org/10.1093/jpids/piv010.
32. Smith SS, Ference EH, Evans CT, Tan BK, Kern RC, Chandra RK. The prevalence of bacterial infection in acute rhinosinusitis: a Systematic review and meta-analysis. *Laryngoscope*. 2015;125(1):57–69. https://doi.org/10.1002/lary.24709.
33. Inagaki K, Blackshear C, Hobbs CV. Deep neck space involvement of Kawasaki disease in the US: a population-based study. *J Pediatr*. 2019;215:118–122. https://doi.org/10.1016/j.jpeds.2019.07.054.
34. Hidaka H, Yamaguchi T, Hasegawa J, et al. Clinical and bacteriological influence of diabetes mellitus on deep neck infection: systematic review and meta-analysis. *Head Neck*. 2015;37(10):1536–1546. https://doi.org/10.1002/hed.23776.
35. Kim SY, Min C, Lee WH, Choi HG. Tonsillectomy increases the risk of retropharyngeal and parapharyngeal abscesses in adults, but not in children: a national cohort study. *PLoS One*. 2018;13(3):e0193913. https://doi.org/10.1371/journal.pone.0193913.
36. Jackson KA, Bohm MK, Brooks JT, et al. Invasive methicillin-resistant Staphylococcus aureus infections among persons who inject drugs - six sites, 2005-2016. *MMWR Morb Mortal Wkly Rep*. 2018;67(22):625–628. https://doi.org/10.15585/mmwr.mm6722a2.

37. Tansey JB, Hamblin J, Mamidala M, et al. Dexamethasone use in the treatment of pediatric deep neck space infections. *Ann Otol Rhinol Laryngol*. 2019. 3489419890349. https://doi.org/10.1177/0003489419890349.
38. Orlandi RR, Kingdom TT, Hwang PH, et al. International consensus statement on allergy and rhinology: rhinosinusitis. *Int Forum Allergy Rhinol*. 2016;6(S1):S22–S209. https://doi.org/10.1002/alr.21695.
39. Miller JM, Binnicker MJ, Campbell S, et al. A guide to utilization of the Microbiology laboratory for diagnosis of infectious diseases: 2018 update by the infectious diseases society of America and the American Society for Microbiology. *Clin Infect Dis Off Publ Infect Dis Soc Am*. 2018;67(6):813–816. https://doi.org/10.1093/cid/ciy584.
40. Tomassen P, Vandeplas G, Van Zele T, et al. Inflammatory endotypes of chronic rhinosinusitis based on cluster analysis of biomarkers. *J Allergy Clin Immunol*. 2016;137(5):1449–1456.e4. https://doi.org/10.1016/j.jaci.2015.12.1324.
41. Chow AW, Benninger MS, Brook I, et al. IDSA clinical practice guideline for acute bacterial rhinosinusitis in children and adults. *Clin Infect Dis Off Publ Infect Dis Soc Am*. 2012;54(8):e72–e112. https://doi.org/10.1093/cid/cir1043.
42. Sabino HAC, Valera FCP, Aragon DC, et al. Amoxicillin-clavulanate for patients with acute exacerbation of chronic rhinosinusitis: a prospective, double-blinded, placebo-controlled trial. *Int Forum Allergy Rhinol*. 2017;7(2):135–142. https://doi.org/10.1002/alr.21846.
43. Ragab A, Farahat T, Al-Hendawy G, Samaka R, Ragab S, El-Ghobashy A. Nasal saline irrigation with or without systemic antibiotics in treatment of children with acute rhinosinusitis. *Int J Pediatr Otorhinolaryngol*. 2015;79(12):2178–2186. https://doi.org/10.1016/j.ijporl.2015.09.045.
44. Lemiengre MB, Driel ML van, Merenstein D, Liira H, Mäkelä M, Sutter AID. Antibiotics for acute rhinosinusitis in adults. *Cochrane Database Syst Rev*. 2018;9. https://doi.org/10.1002/14651858.CD006089.pub5.
45. Rosenfeld RM, Piccirillo JF, Chandrasekhar SS, et al. Clinical practice guideline (update): adult sinusitis. *Otolaryngol-Head Neck Surg Off J Am Acad Otolaryngol-Head Neck Surg*. 2015;152(suppl 2):S1–S39. https://doi.org/10.1177/0194599815572097.
46. Costa ML, Psaltis AJ, Nayak JV, Hwang PH. Medical therapy vs surgery for recurrent acute rhinosinusitis. *Int Forum Allergy Rhinol*. 2015;5(8):667–673. https://doi.org/10.1002/alr.21533.

CHAPTER 61: QUESTIONS AND ANSWERS

1. A previously well 18-year-old male presents with throat pain and fever to 39 °C for the past 2 days. He denies vomiting, diarrhea, cough, or rhinorrhea. He is not sexually active. Physical exam is notable for tender anterior cervical lymphadenopathy and symmetrically swollen and erythematous palatine tonsils with a gray-white tonsillar exudate. The remainder of his neck and physical exam is benign. What is the most appropriate next step?

a. Empirically prescribe amoxicillin for 10 days
b. Obtain a complete blood count with differential
c. Obtain an intraoral tonsillar ultrasound
d. Perform a rapid strep antigen test and prescribe antibiotics only if there is a positive result

Answer: D. This patient is presenting with pharyngitis, with sore throat, fever, exudative pharyngitis, and cervical lymphadenopathy without associated cough or rhinorrhea. A majority of cases are viral and caused by common cold viruses. Bacterial infection is only responsible for approximately 5% to 10% of adult and 20% to 30% of pediatric cases. Rapid antigen testing is recommended as a first-line diagnostic test for GAS pharyngitis, because it is highly sensitive and specific when performed correctly. A positive test indicates the presence of GAS and does not require follow up testing.

2. A 19-year-old female patient presents to the emergency department with vision changes and headaches. She reports a sore throat 10 days ago started to improve, but never completely resolved. For the past 3 days she noted increasing left-sided neck pain and fevers. On exam she appears uncomfortable with a fever and mild tachycardia. She is warm and diaphoretic with a dry forehead on the left. Her left eyelid is slightly lower than the right and her left pupil is 3 mm smaller than the right. Her extraocular movements are intact, and her intraoral exam shows symmetric tonsils with mild erythema and a midline uvula. She has mild trismus. What is most likely causing her symptoms?

a. Parapharyngeal abscess with invasion of the carotid sheath
b. Peritonsillar abscess with compression of the carotid sheath
c. Spread of infection into the cavernous sinus causing thrombosis and intracranial abscess
d. Submandibular space infection and spread into retropharynx

Answer: A. Complications from retropharyngeal or parapharyngeal abscess include airway compromise, abscess rupture leading to aspiration of pus, and involvement of the carotid sheath. The carotid sheath is surrounded by deep fascia and includes the carotid artery, internal jugular vein, and vagus nerve, and thus infection can result in compromise of any of these structures including arterial erosion, aneurysm, Lemierre syndrome, palsy of cranial nerves IX–XII, and mediastinitis.

3. A 34-year-old male presents with complaints of high fever, left-sided face pain, and purulent nasal discharge for 10 days. He has been attempting symptom management with nasal saline irrigation and intranasal corticosteroids, but after initial improvement reports worsening symptoms. What is the next step in treatment?

a. CT scan of his face to evaluate for complications of rhinosinusitis
b. Obtain bacterial culture via sinus puncture
c. Prescribe a 5-day course of amoxicillin
d. Supportive care with antiinflammatory medications, rest, and continued nasal saline irrigation

Answer: C. The diagnosis of acute rhinosinusitis is clinical. The diagnosis of bacterial rhinosinusitis requires one of the three criteria: at least 10 days of persistent symptoms without improvement, three to four days of severe symptoms including a fever greater than 39 degrees Celsius with nasal discharge or facial pain without improvement, or onset of progressive symptoms with worsening symptoms after initial improvement. Given the risks and lack of benefit of routine antibiotic use in rhinosinusitis, antibiotics should only be used for patients who meet the definition of bacterial rhinosinusitis with these criteria.

4. Patients presenting with symptoms concerning for a retropharyngeal abscess are best evaluated using what modality?

a. Contrast-enhanced CT
b. Intraoral ultrasound
c. Lateral neck x-ray
d. Visualization with a fiberoptic scope

Answer: A. Contrast-enhanced CT scan is the preferred diagnostic test for the identification of retropharyngeal abscess. CT findings are characterized by a fluid collection with central hypodensity and complete ring enhancement with scalloping. Fat stranding and edema characterized by low-density thickening without peripheral enhancement may represent an early phase of infection before an abscess develops.

CHAPTER 61: QUESTIONS AND ANSWERS—cont'd

5. This bacterium is frequently implicated in non-GAS pharyngitis and is also most commonly implicated in suppurative jugular thrombophlebitis:

a. *Arcanobacterium haemolyticum*
b. *Fusobacterium necrophorum*
c. *Haemophilus influenzae* type B
d. Methicillin-resistant *Staphylococcus aureus*

Answer: B. *Fusobacterium necrophorum* is an anaerobic gram-negative rod that is part of the normal oral flora and causes pharyngitis in patients 15 to 45 years old with a similar presentation to GAS. *Fusobacterium* is the primary causative agent in septic jugular vein thrombophlebitis (Lemierre syndrome). *Fusobacterium* should be considered in young adults with ongoing severe symptoms.