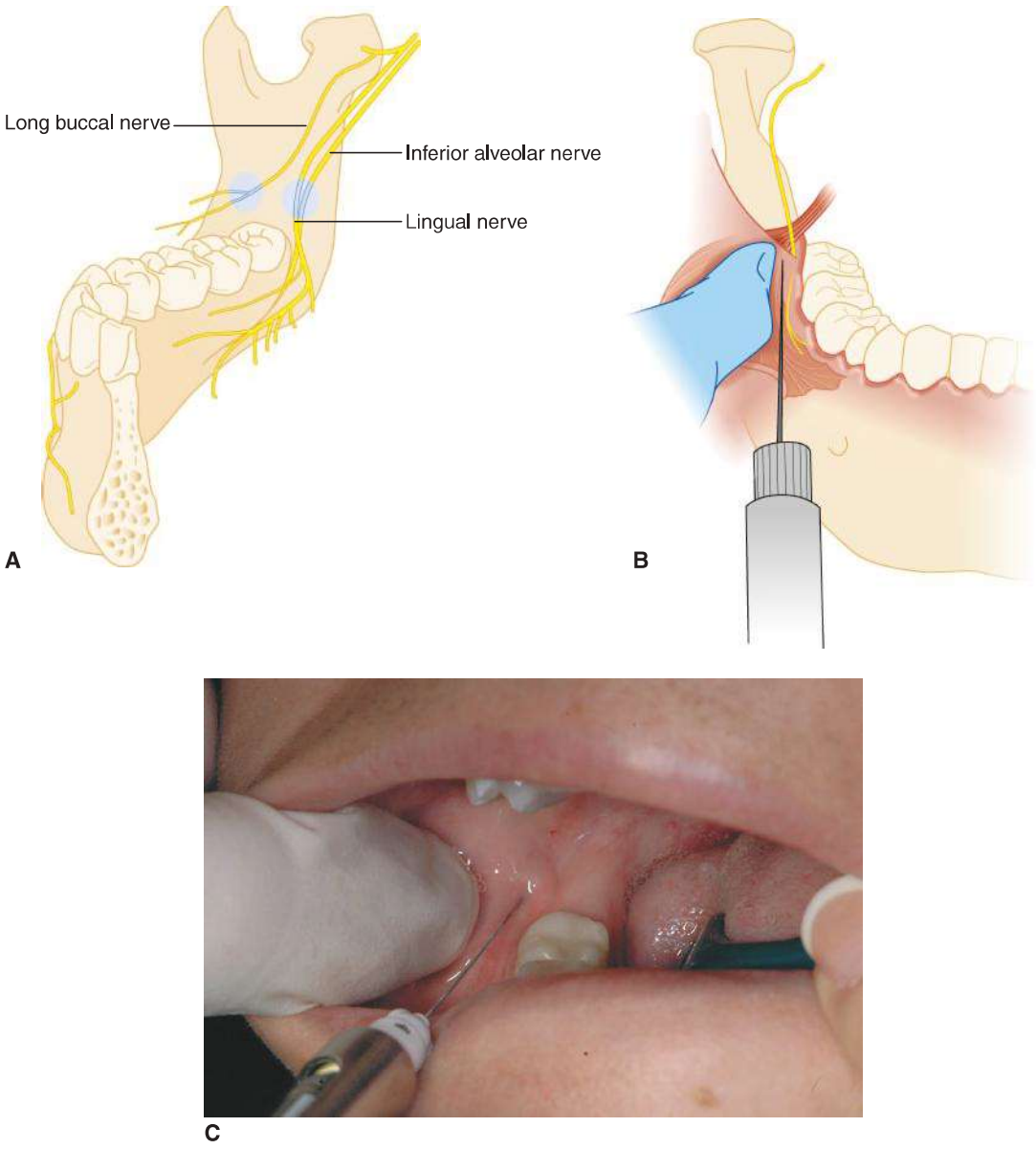

FIGURE 245-18. The lingual nerve block and the long buccal nerve block. **A.** The shaded areas represent the area in which anesthesia should be deposited for their respective blocks. The lingual nerve runs superficial and medial to the inferior alveolar nerve and can be easily anesthetized as part of the inferior alveolar nerve block. The lingual nerve block can be performed by depositing anesthesia about half the depth of the inferior alveolar nerve block. **B.** A diagrammatic representation of the long buccal nerve block. The long buccal nerve requires a separate injection of a small quantity of anesthetic just lateral to the molars in the buccal mucosa. **C.** Clinical photograph of the long buccal nerve block.

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 246

Neck and Upper Airway

Nicholas D. Hartman

This chapter reviews infectious and noninfectious conditions that can obstruct the upper airway. These disorders must be recognized quickly because early airway management may be lifesaving. Neck trauma is discussed in the chapter 260, "Trauma to the Neck," and angioedema is discussed in the chapter 14, "Anaphylaxis, Allergies, and Angioedema."

NECK AND UPPER AIRWAY INFECTIONS

PHARYNGITIS/TONSILLITIS

Viruses account for the majority of cases of pharyngitis or tonsillitis. Acute viral pharyngitis is most commonly caused by rhinovirus but can be caused by multiple other viral agents (**Table 246-1**).[1]

VIRAL PHARYNGITIS

Viral pharyngitis generally displays a vesicular or petechial pattern on the soft palate and tonsils and is associated with rhinorrhea. However, in

TABLE 246-1 Microbial Causes of Acute Pharyngitis

Pathogen	Syndrome/Disease	Estimated % of Cases*
Viral		
Rhinovirus (100 types, 1 subtype)	Common cold	15–20
Coronavirus (3+ types)	Common cold	>5
Adenovirus (types 3, 4, 7, 14, 21)	Pharyngoconjunctival fever, acute respiratory disease	6
Herpes simplex virus (type 1, 2)	Gingivitis, stomatitis, pharyngitis	4
Parainfluenza virus (types 1–4)	Common cold, croup	2
Influenza virus (types A, B)	Influenza	2
Respiratory syncytial virus	Bronchiolitis, pharyngitis	1–2
Coxsackievirus A (types 2, 4, 5, 6, 8, 10)	Herpangina	<1
Epstein-Barr virus	Infectious mononucleosis	<1
Cytomegalovirus	Infectious mononucleosis	<1
Human immunodeficiency virus type 1	Acute retroviral syndrome	<1
Bacterial		
Streptococcus pyogenes (GABHS)	Pharyngitis, tonsillitis, scarlet fever	10–22
Fusobacterium necrophorum	Pharyngitis, tonsillitis, Lemierre's syndrome	5–10
Streptococcus dysgalactiae subspecies *equisimilis* (formerly group C β-hemolytic streptococci)	Pharyngitis, tonsillitis	3–5
Neisseria gonorrhoeae	Pharyngitis	<1
Corynebacterium diphtheriae	Diphtheria	<1
Arcanobacterium haemolyticum	Pharyngitis	<1
Chlamydial		
Chlamydia pneumonia	Pneumonia, bronchitis, pharyngitis	<1
Mycoplasmal		
Mycoplasma pneumonia	Pneumonia, bronchitis, pharyngitis	<1

Abbreviation: GABHS = group A β-hemolytic *Streptococcus*.
*Estimates of percentage of all cases of pharyngitis due to the indicated organism.

patients with nonstreptococcal pharyngitis (mostly viral), 16% have tonsillar exudate, 55% have cervical adenopathy, and 64% lack cough.[2] Most cases of viral pharyngitis require no specific diagnostic testing. There are three notable exceptions where testing may be indicated: suspected influenza, infectious mononucleosis, and acute retroviral syndrome. See Centers for Disease Control and Prevention influenza Web site for testing and treatment recommendations (http://www.cdc.gov/flu/). Infectious mononucleosis, influenza herpesvirus, and cytomegalovirus infections are discussed in the chapter 153, "Serious Viral Infections." The acute retroviral syndrome of early human immunodeficiency virus infection can also mimic mononucleosis. Symptoms of pharyngitis develop 2 to 4 weeks after exposure and resolve within 2 weeks. See the chapter 154, "Human Immunodeficiency Virus Infection" for recommendations on testing and treatment. Non–human immunodeficiency virus, noninfluenza viral pharyngitis should be treated symptomatically with oral hydration, antipyretics, analgesics, and rest. Patients unable to tolerate oral fluids or who become dehydrated should be given IV fluids.

BACTERIAL PHARYNGITIS

Group A β-Hemolytic *Streptococcus* **Group A β-hemolytic *Streptococcus* (GABHS) pharyngitis,** Lancefield group A species of *Streptococcus pyogenes*, is responsible for 5% to 15% of pharyngitis in adults.[1,2] After an incubation period of 2 to 5 days, patients develop the sudden onset of sore throat, painful swallowing, chills, and fever. Headache, nausea, and vomiting are common. Signs and symptoms of GABHS pharyngitis include marked erythema of the tonsils and tonsillar pillars (found in 62% of cases); tonsillar exudate (32%); and enlarged, tender cervical lymph nodes (76%).[2] A 2012 epidemiologic study found that only 6% of GABHS cases had fever and 28% had cough.[2] Patients may have uvular edema, myalgias, and malaise but are less likely to have rhinorrhea or conjunctivitis compared to viral pharyngitis.

Uvula edema, sometimes referred to as *Quincke's edema*, can be associated with upper airway infections such as GABHS pharyngitis, peritonsillar abscess, or epiglottitis. It can also be idiopathic. If it is an isolated finding and symptoms are uncomfortable to the patient, dexamethasone, 4 milligrams IV or PO, can be given as a single dose in the ED.

The original Centor criteria listed four clinical indicators of GABHS pharyngitis: (1) tonsillar exudates, (2) tender anterior cervical adenopathy, (3) absence of cough, and (4) history of fever.[2] The Centers for Disease Control and Prevention reversed its prior recommendation for empiric treatment based on clinical findings in 2012 in concert with the Infectious Diseases Society of America.[3] The Centers for Disease Control and Prevention and Infectious Diseases Society of America recommend using two or more Centor criteria as a threshold for selecting patients for rapid strep testing and treating only those with positive tests.[3,4] Guidelines do not recommend throat cultures in adult patients with one or fewer Centor criteria or routine throat culture for those with negative rapid strep tests, unless considering other bacterial pathogens[3]

Untreated, GABHS infection lasts 7 to 10 days. Antibiotic therapy of GABHS hastens resolution by 1 to 2 days if initiated within 2 to 3 days of symptom onset and prevents suppurative complications and rheumatic fever, although not glomerulonephritis.[3] **GABHS has never been resistant to penicillin, so penicillin remains the recommended first-line drug for GABHS.**[5,6] Adults should receive a single IM dose of 1.2 million units of benzathine penicillin G, 500 milligrams of penicillin VK PO two times daily for 10 days, or amoxicillin 500 milligrams PO two times daily or 1000 milligrams one time daily. A first-generation cephalosporin antibiotic or clindamycin may be used for penicillin-allergic patients.[5] A single dose of PO or IM dexamethasone in immunocompetent adults with moderate to severe pharyngitis can achieve an earlier onset of pain relief and a shorter duration of pain.[7]

Other Causes of Bacterial Pharyngitis Several other bacteria can cause pharyngitis, although these infections are less common (Table 246-1). ***S. dysgalactiae* subspecies *equisimilis***, previously known as β-hemolytic groups C and G streptococci, are important pathogens causing pharyngitis, skin infections, and more serious infections such as meningitis or toxic shock syndrome in the elderly or immunocompromised.[8] *S. dysgalactiae* subspecies *equisimilis* frequently colonizes the upper respiratory tract (60% who are culture positive are asymptomatic),[8] so distinguishing acute infection from a carrier state may be difficult;[9] treatment is recommend for patients with acute symptoms.[8] *S. dysgalactiae* subspecies *equisimilis* pharyngitis is almost uniformly susceptible to penicillin.[8] Clindamycin and fluoroquinolones are alternatives.[8]

Fusobacterium necrophorum, a gram-negative anaerobe,[10] is the causative agent in **Lemierre's syndrome**, a complication of pharyngitis causing suppurative thrombophlebitis of the internal jugular vein, with or without bacteremia and septic emboli. Suspect *F. necrophorum* in adolescents or young adults with worsening symptoms and neck swelling.[11] Treatment is with penicillin, clindamycin, or third-generation cephalosporins; *F. necrophorum* resistance to macrolides is high.[11]

Gonococcal pharyngitis is usually associated with genital infection and is treated by the same antibiotics. **Diphtheria** is caused by *Corynebacterium diphtheriae* and is rare in well-immunized populations. It is characterized by a slow onset of mild to moderate pharyngeal discomfort and low-grade fever. On physical examination, a gray membrane is

seen adherent to the tonsillar or pharyngeal surface and may extend to the uvula, soft palate, pharynx, and larynx. Treatment is with diphtheria antitoxin and metronidazole to prevent transmission to others.

PERITONSILLAR ABSCESS

A peritonsillar abscess is a collection of purulent material between the tonsillar capsule, the superior constrictor, and palatopharyngeus muscles. Risk factors include periodontal disease, smoking, chronic tonsillitis, multiple trials of antibiotics, and previous peritonsillar abscess.[12] Peritonsillar abscess develops primarily in adolescents and young adults without seasonal variation as previously thought.[12,13] Although peritonsillar abscesses are typically polymicrobial infections, in patients 15 to 24 years of age, *Fusobacterium necrophorum* has been the most common organism in many communities.[13,14]

CLINICAL FEATURES AND DIAGNOSIS

Patients with peritonsillar abscess (adolescents and adults) appear ill and present with sore throat (99%), fever (54%), malaise, odynophagia, dysphagia, and/or otalgia.[15] Physical signs include inferior and medial displacement of the infected tonsil(s) (46%), contralateral deflection of the swollen uvula (43%), tender cervical lymphadenopathy (41%), trismus (32%), muffled voice ("hot potato voice"), palatal edema, and dehydration[15] (**Figure 246-1**). The differential diagnosis of a peritonsillar abscess includes peritonsillar cellulitis, mononucleosis, lymphoma, herpes simplex tonsillitis, retropharyngeal abscess, neoplasm, and internal carotid artery aneurysm. In peritonsillar cellulitis, erythema and edema of the tonsillar pillar and soft palate are evident, but pus has not yet formed. Diagnosis of a peritonsillar abscess is often made by history and physical examination alone. When the diagnosis is in question, intraoral US has a sensitivity of 89% to 95% with a specificity of 79% to 100% for peritonsillar abscess.[16] CT scan with contrast is indicated if there is concern for spread beyond the peritonsillar space or lateral neck space complications.[16]

TREATMENT

Treatment options include drainage of the abscess by needle aspiration, incision and drainage, or, rarely, immediate tonsillectomy. Choice of treatment depends on clinical symptoms, degree of patient cooperation, history of previous tonsil disease, and healthcare personnel experience. There is no difference in outcome when comparing needle aspiration with incision and drainage.[16] Abscess tonsillectomy ("quinsy tonsillectomy") should only be considered when patients have strong indication for tonsillectomy, such as sleep apnea, recurrent tonsillitis, or recurrent peritonsillar abscess.[16] Needle aspiration is minimally invasive, less painful than incision and drainage, and may be performed by general or specialized medical personnel. Approximately 90% of patients will be treated effectively after a single needle aspiration.[16]

Needle aspiration should be performed by an individual trained in the technique. First, apply lidocaine spray or gel or benzocaine-tetracaine spray to the overlying mucosa. Then inject 1 to 2 mL of lidocaine with epinephrine into the mucosa of the anterior tonsillar pillar using a 25-gauge needle **The drainage needle should penetrate no more than 1 cm because the internal carotid artery usually lies laterally and posterior to the posterior edge of the tonsil.** The plastic sheath of the needle can be cut 1 cm from its tip to serve as a guard. If the internal carotid artery lies more medial and anterior, it can usually be palpated in this area. Once adequate anesthesia is achieved, introduce an 18-gauge needle just lateral to the tonsil, approximately halfway between the base of the uvula and the maxillary alveolar ridge, until the abscess cavity is encountered and pus is aspirated. Often, multiple aspirations may be required to find the abscess. If not done previously, a contrast CT scan of the neck is recommended when the results of needle aspiration are negative and a parapharyngeal or retropharyngeal space process is suspected.

Initial therapy should include a 10-day course of antimicrobials effective against group A *Streptococcus* and oral anaerobes (including *F. necrophorum*). Proven agents are penicillin VK plus metronidazole[15] or clindamycin for penicillin-allergic patients. Toxic patients or patients unable to take medicine PO should receive piperacillin-tazobactam, 3.375 grams IV, or similar agent. Single IV use of high-dose steroid (methylprednisolone, 125 milligrams, or dexamethasone, 10 milligrams) in addition to antibiotics and drainage improves severity and duration of pain.[15,17] Provide follow-up within 24 to 36 hours of aspiration, with instructions to return to the ED if worse. If the patient is not improving, consider repeating the aspiration, otolaryngologic consultation for incision and drainage or tonsillectomy, or obtaining a CT scan to confirm or reject the diagnosis. Complications of a peritonsillar abscess include airway obstruction, rupture of the abscess with aspiration of the contents, hemorrhage secondary to erosion of carotid sheath, retropharyngeal abscess, mediastinitis, and poststreptococcal sequelae.

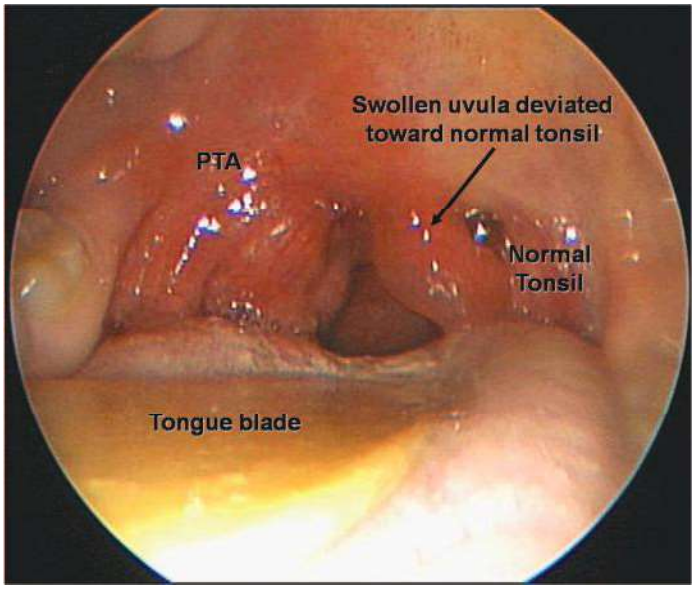

FIGURE 246-1. Right peritonsillar abscess (PTA) displacing right tonsil medially and the uvula toward the normal left tonsil. Abscess is between the right tonsil and the superior constrictor muscles.

ADULT EPIGLOTTITIS (SUPRAGLOTTITIS)

Epiglottitis is an inflammatory condition, usually infectious, primarily of the epiglottis but often including the entire supraglottic region (many prefer the term *supraglottitis*). It can lead to rapid airway obstruction. Prior to the introduction of a conjugate vaccine for *Haemophilus influenzae* type b in the 1980s, most cases of epiglottitis affected children age 1 to 5 years. In the postvaccine era, the dramatic decline in pediatric cases has confined the disease primarily to adults, with an estimated mean age of 45 years.[18,19] Most cases in adults are caused by *Streptococcus* species, *Staphylococcus* species, viruses, and fungi, although most frequently, no organism can be isolated.[19] Risk factors for mortality in patients with epiglottitis are advanced age and male sex.[20]

CLINICAL FEATURES AND DIAGNOSIS

Symptoms are typically a 1- to 2-day history of worsening dysphagia, odynophagia, and dyspnea, particularly in the supine position. The clinical triad of the "three Ds" (drooling, dysphagia, and distress) is a classic but infrequent presentation. Other symptoms are fever, tachycardia, cervical adenopathy, and anterior neck tenderness with pain on gentle palpation of the larynx and upper trachea. Stridor is primarily inspiratory. Patients often position themselves sitting up, leaning forward, mouth open, head extended, and panting.

Diagnosis is clinical and confirmed by radiographs or transnasal fiberoptic laryngoscopy. Lateral cervical soft tissue radiographs demonstrate obliteration of the vallecula, swelling of the aryepiglottic folds,

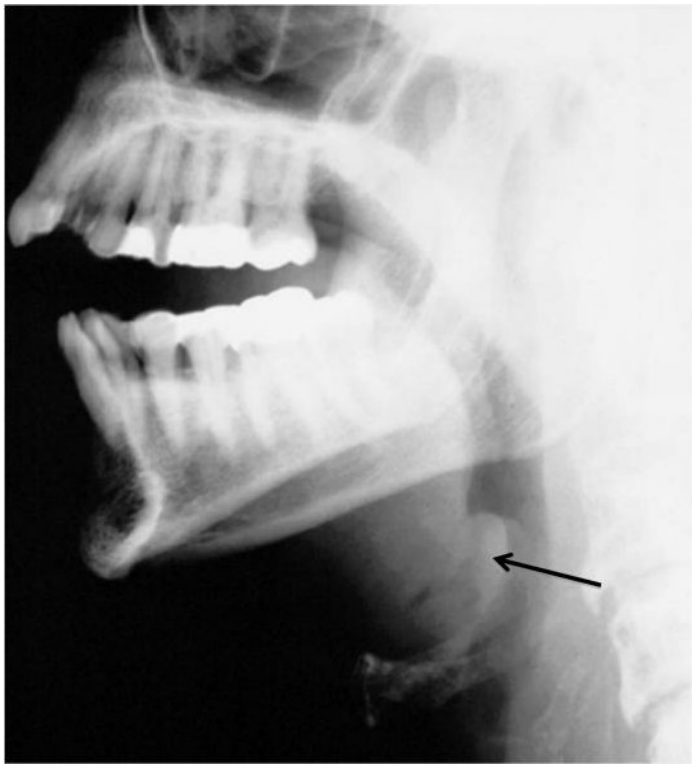

FIGURE 246-2. Acute epiglottitis. *Arrow* points to thickened epiglottis resembling a thumb print on a soft tissue lateral radiograph.

edema of the prevertebral and retropharyngeal soft tissues, and ballooning of the hypopharynx (**Figure 246-2**). The epiglottis appears enlarged and thumb-shaped. Direct laryngoscopy examination can confirm the diagnosis in adults if necessary but should be done carefully to avoid sudden, unpredictable airway obstruction. **Patients with worsening dyspnea in the supine position should *not* be sent to the CT scanner; CT of the neck is not needed to make the diagnosis.**

TREATMENT

Obtain immediate otolaryngologic consultation for suspected epiglottitis. Be prepared to establish a definitive airway. Patients should not be left unattended, and they should remain sitting up. Initial treatment consists of supplemental humidified oxygen, IV hydration, cardiac monitoring, pulse oximetry, and IV antibiotics. Humidification and hydration can help decrease the risk for sudden airway blockage. Steroids are often given to decrease airway inflammation and edema (methylprednisolone, 125 milligrams IV).

In adults, the need for intubation usually can be determined by transnasal fiberoptic examination of the supraglottis. Intubation is generally accomplished by "awake" fiberoptic intubation in the operating room, with preparations for immediate awake tracheostomy or cricothyrotomy. In cases of airway obstruction in the ED, be prepared for a very difficult intubation secondary to the swollen, distorted anatomy. In the case of intubation failure, the last resorts for preserving the airway are cricothyrotomy and needle cricothyrotomy.

Current antibiotic recommendations are cefotaxime 50 milligrams/kg IV every 8 hours plus vancomycin 15 milligrams/kg every 12 hours.[21] Alternative antibiotics include ampicillin-sulbactam, ceftriaxone, or piperacillin-tazobactam.[21] Respiratory fluoroquinolones are an option for patients with severe penicillin allergies.

RETROPHARYNGEAL ABSCESS

The retropharyngeal space is a potential space anterior to the prevertebral fascia that extends from the base of the skull to the tracheal bifurcation. In adults, a retropharyngeal abscess is usually due to intraoral procedures, trauma, foreign bodies such as a fishbone, or extension from odontogenic infection.[22] Cultures from retropharyngeal abscesses are usually polymicrobial: group A β-hemolytic streptococci, *Staphylococcus aureus* (including methicillin-resistant *S. aureus*), *H. influenzae*, and *Bacteroides*, *Peptostreptococcus*, and *Fusobacterium* species.

CLINICAL FEATURES AND DIAGNOSIS

The most common symptoms in adults are sore throat, dysphagia, neck pain, and less commonly, stridor. In addition, patients may also have complaints of cervical lymphadenopathy, poor oral intake, muffled voice, and respiratory distress. Visible neck swelling is not common.

A lateral soft tissue radiograph of the neck taken during inspiration with moderate cervical extension can demonstrate thickening and protrusion of the retropharyngeal wall, classically with 5 to 7 cm of prevertebral widening at the second cervical vertebra.[23] However, contrast-enhanced CT scan of the neck is the test of choice for diagnosis of a retropharyngeal abscess.[24] Early CT findings may reflect reactive, nonsuppurative edema, mild fat stranding with discernible tissue planes, linear fluid, minimal mass effect, and no associated enhancement. Necrotic nodes with central low attenuation and ring enhancement reflect an abscess (**Figure 246-3**). A patient with airway distress should not be sent unobserved for CT scanning.

TREATMENT

Obtain immediate otolaryngologic consultation. Provide IV hydration and antibiotic treatment with either clindamycin or cefoxitin IV; alternatively, piperacillin-tazobactam or ampicillin-sulbactam may be used.[21] Although a few patients with small abscess cavities may be managed with IV antibiotics alone, most patients will require surgical intervention. Catastrophic complications from retropharyngeal abscess include extension of

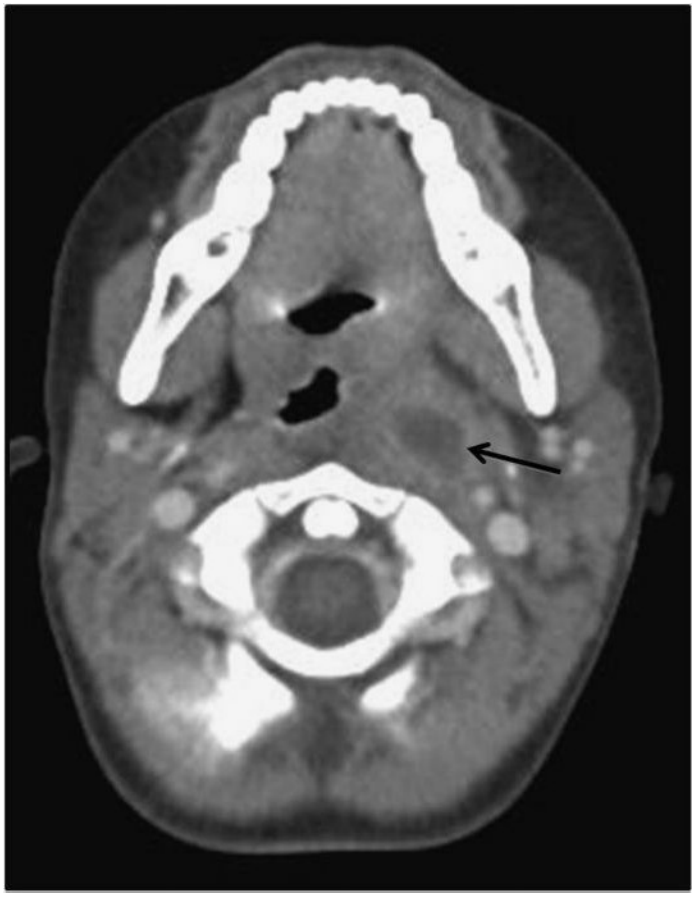

FIGURE 246-3. Contrasted CT of a left retropharyngeal abscess (*arrow*).

the infection into the mediastinum and upper airway asphyxia from direct pressure or aspiration after sudden rupture of the abscess.[25]

ODONTOGENIC ABSCESS

Odontogenic infections can arise from an infected tooth or after a tooth extraction. Development of the infection varies from <1 day to up to 1 to 3 weeks after the onset of tooth pain and may occur despite oral antibiotics. Odontogenic infections are polymicrobial; the most common bacteria are *Streptococci viridians*, *Peptostreptococcus*, *Prevotella*, and staphylococci.[26] **Most deep neck infections originate from an odontogenic source, usually the mandibular teeth.** Dental abscesses may spread into the parapharyngeal and retropharyngeal spaces. Presenting features include neck mass, trismus, fever, leukocytosis, dysphagia, and dyspnea. Potential complications include necrotizing fasciitis, descending necrotizing mediastinitis, orbital infections, and hematogenous dissemination to distant organs.

CLINICAL FEATURES

See chapter 245,"Oral and Dental Emergencies" for management of dental infections isolated to the mandible or maxilla. Soft tissue extension from odontogenic infection ranges from diffuse cellulitis to abscess formation in labial or buccal gingiva. In some cases, intraoral or dentocutaneous fistula formation may occur. Fascial layers of the head and neck produce planes or potential spaces for infectious spread. Infections associated with maxillary teeth tend to spread into potential spaces in the face. Infections of maxillary molars tend to involve the masticator space, which can extend into the parapharyngeal space and downward into the neck and mediastinum. Infections of anterior mandibular teeth tend to spread into the neck. Infections of anterior teeth, bicuspids, and first molars of the mandible tend to enter the sublingual space, with edema of the floor of the mouth with little extraoral swelling. Involvement of the submandibular space is typically the result of second and third mandibular molar infections.

DIAGNOSIS AND TREATMENT

Superficial odontogenic abscesses can be diagnosed with US at the bedside.[27] For diagnosis of suspected deep space infections, contrast-enhanced CT scan is recommended to identify the need for surgical management.[27] Treatment of odontogenic infections includes appropriate antibiotic therapy (aerobic and anaerobic coverage) and surgical drainage of abscesses. Penicillin VK and amoxicillin remain appropriate options for outpatient treatment; amoxicillin-clavulanate, clindamycin, cefuroxime, and levofloxacin are second-line choices.[28,29] Patients with deep-neck infections require IV antibiotics; ampicillin-sulbactam with clindamycin and ciprofloxacin is one recommended regimen. Other useful agents include piperacillin-tazobactam, imipenem-cilastatin, and ertapenem.

COMPLICATIONS

Ludwig's angina is infection of the submental, sublingual, and submandibular spaces. Patients usually present with poor dental hygiene, dysphagia, and odynophagia. Clinical examination reveals trismus and edema of the entire upper neck and floor of mouth. Infection progresses rapidly and can posteriorly displace the tongue, causing airway compromise. Definitive airway management should be considered early in the course, including awake fiberoptic intubation or awake tracheostomy.[30] Stridor, difficulty managing secretions, and cyanosis are late signs and require emergent airway management. Systemic antibiotics are not a substitute for definitive airway management because it may take >1 week for edema resolution with antibiotic therapy.

Patients with **necrotizing infections** are critically ill, with overlying skin discoloration, crepitus of the subcutaneous tissue, and systemic signs, including fever, tachycardia, hypotension, and confusion. CT reveals subcutaneous emphysema, deep tissue gas, and pockets of suppuration (**Figure 246-4**). Aerobic and anaerobic cultures are necessary for identification of causative organisms. Therapy of necrotizing fasciitis is immediate surgery with fasciotomy with wide local debridement and broad-spectrum IV antibiotics. Mediastinal extension places the patient at risk for great vessel erosion, retroperitoneal extension, pleural abscess, pericardial effusion, and sepsis; mortality ranges from 10% to 40%.[31] Tracheostomy should be performed if airway obstruction develops. Surgery can be lifesaving, and immediate surgical consultation is required for this rapidly progressing disease.[31]

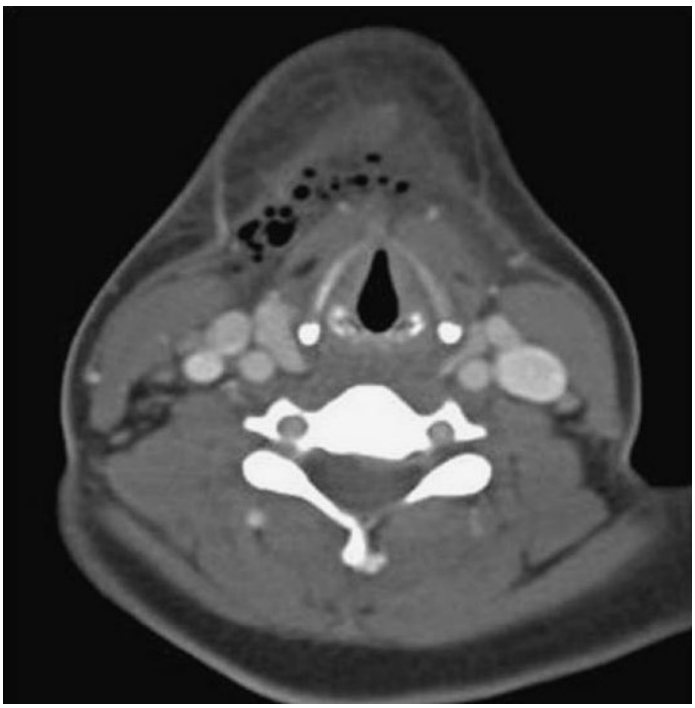

FIGURE 246-4. CT demonstrating necrotizing fasciitis with gas in the deep tissue of the anterior neck.

NECK AND UPPER AIRWAY MASSES

CLINICAL FEATURES

Neck masses (**Figures 246-5 and 246-6**) can result from congenital, infectious, glandular, or neoplastic disorders. Enlargement may lead to airway compromise, dehydration secondary to dysphagia and odynophagia, or secondary infected. Age of the patient and characteristics including location of the mass may aid in the diagnosis (**Tables 246-2 and 246-3**).[32] Neck masses in children are discussed in the chapter 122, "Neck Masses in Infants and Children," in the Pediatrics section. **In adults >40 years old, up to 80% of lateral neck masses persistent for >6 weeks are malignant.**[33]

DIAGNOSIS AND MANAGEMENT

The urgency for evaluation of a neck mass depends on patient acuity.[33] Patients with airway compromise or significant dysphagia and odynophagia should be evaluated by flexible nasopharyngolaryngoscopy **before** CT scan. CT scan will delineate the extent of the mass and likely will be required for surgical intervention. If no airway compromise or dehydration is present, the patient should follow up with primary care for outpatient imaging and further evaluation. The final diagnosis for a neck or upper airway mass will not be made in the ED. All neck masses should have follow-up for diagnosis and treatment.

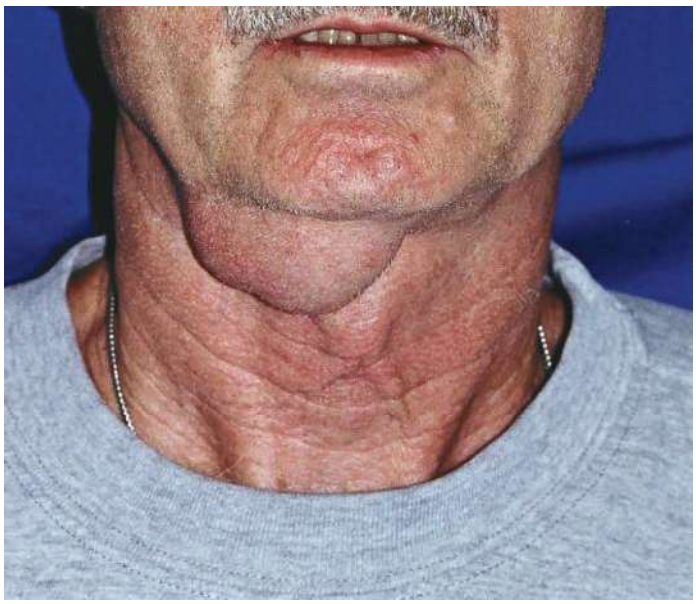

FIGURE 246-5. Right plunging ranula presenting as a painless ballotable submandibular mass.

Empiric antibiotic therapy should be initiated for inflammatory lymph nodes, usually with cephalexin, 250 to 500 milligrams PO three to four times daily; amoxicillin, 250 to 500 milligrams PO three times daily; or clindamycin, 300 milligrams three to four times daily. Resolution is expected in 2 weeks for those lesions that are due to infection alone. Empiric therapy of sialoadenitis should include staphylococcal coverage most commonly, clindamycin[34] (see chapter 118, "Neck Masses in Children").

TABLE 246-2 Neck Masses in Young and Older Adults

Young Adult	Adult
Reactive lymphadenopathy	Metastatic aerodigestive tract carcinoma
Mononucleosis	Salivary gland infection or neoplasm
Lymphoma	
Branchial cleft cyst	Lymphoma
Thyroglossal duct cyst	Thyroid disorder
	Tuberculosis

POSTTONSILLECTOMY BLEEDING

Tonsillectomy is the second most common reason for care in pediatric hospitals in the United States.[35] Postoperative bleeding is a well-known complication of tonsillectomy that can, rarely, lead to death from airway obstruction or hemorrhagic shock. Rate of secondary hemorrhage varies according to the method used for the procedure. The incidence of posttonsillectomy bleeding ranges from 1% to 8.8%, with approximately half requiring surgical intervention for control of bleeding.[36]

Although bleeding can be seen within 24 hours of surgery, most significant hemorrhage occurs between postoperative days 5 and 10. There is a significantly higher incidence of bleeding in patients between 21 and 30 years of age, as well as those over age 70.[36] Posttonsillectomy bleeding can be fatal and requires prompt intervention with control of the airway. An otolaryngologist should be consulted early.

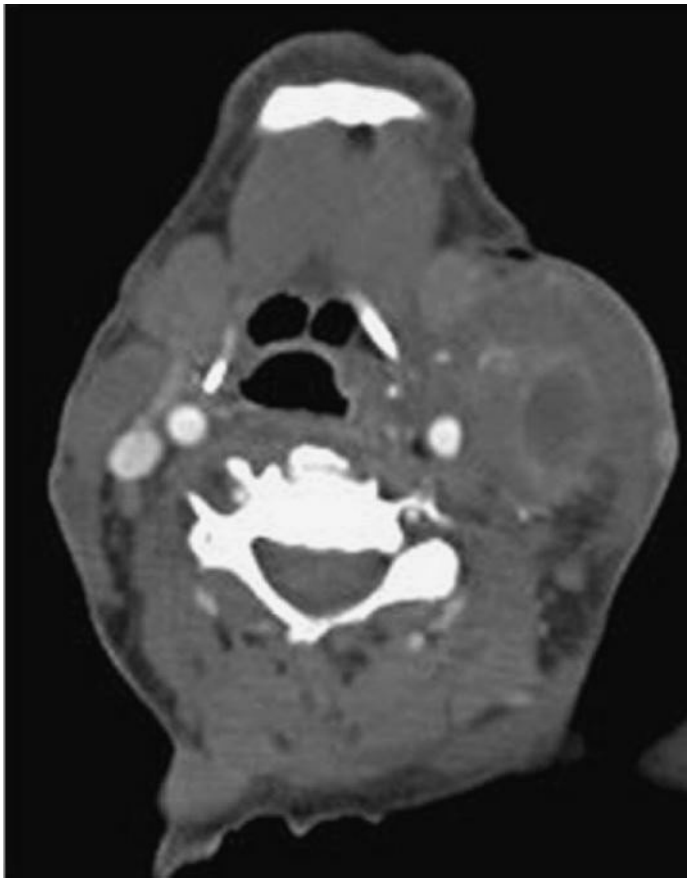

FIGURE 246-6. Hypopharyngeal squamous cell carcinoma metastatic to left cervical lymph nodes. Note the thrombosis of the left jugular vein with displacement of the airway to the right.