Pharynx and Throat Emergencies

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PATHOPHYSIOLOGY

The structure of the oropharynx also provides multiple types of complications from pharyngeal emergencies. The most obvious potential complication from diseases of the airway is airway compromise. Restrictive spaces such as the hypopharynx and larynx (with the narrow vocal cords and superior epiglottis) provide ample opportunity for even mild inflammation or edema to significantly alter normal airflow through this region. In addition, the proximate location of the great vessels of the carotid artery and internal jugular veins provide fertile ground for infections or complications from airway procedures such as drainage of peritonsillar abscess. The fascial planes in the oropharynx reach toward vital structures, which when infected may have disastrous results. The deep fascial planes track posteriorly toward the vertebral column, inferiorly into the mediastinum, and superiorly near the cavernous sinus, thus providing opportunities for cervical vertebral osteomyelitis, mediastinitis, or cranial nerve problems.

ANATOMY

The pharyngeal anatomy resembles an inverted cone, with the narrowest part at the point where it attaches to the esophagus at the level of the cricoid cartilage and the widest part attaching to the base of the skull. It is a muscular and fibrous tube located immediately behind the nasal cavity and mouth and has many functions. The pharynx connects anteriorly and superiorly with the oral cavity for passage of nutrition and hydration into the esophagus. Inferiorly, it connects with the esophagus posteriorly and the larynx inferiorly and allows the complex selection of food and air to travel to different organ systems. The pharynx has abundant immunologic tissue to help fight infection—the palatine tonsils, adenoids, and lingual tonsils—which may predispose the host to dangerous circumstances (e.g., airway compromise) if the inflammatory response is too dramatic.

Oropharyngeal Complaints

Epidemiology

Upper respiratory complaints are the most common reason that patients seek medical care. Pharyngitis is diagnosed in more than 10 million patients per year. Millions of dollars are spent not only on medications and medical visits but also to arrange child care and sick leave for patients unable to go to work or school. Even though most causes of throat pain are benign and self-limited, a number of causes of sore throat may be severe and life-threatening.

The oropharynx plays a vital role in the health of every patient. Not only is it the needed entry point for food and nutrition, but oxygen and carbon dioxide must also move through the oropharynx every minute to maintain metabolic homeostasis. Any disease process that interferes with the function of the oropharynx has the potential to cause significant morbidity and perhaps mortality.
proximity to the tissues of the airway and also systemically. The local inflammation may give rise to straightforward complications such as otitis media, but more dramatic complications such as dehydration, tissue edema, and airway compromise may also occur.

Pharyngeal irritation and inflammation produce throat pain that is worsened by swallowing. Occasionally, this pain may radiate to the ears or feel pressure-like because the eustachian tubes may also be blocked or swollen. The tonsils and pharynx may be erythematos with or without tonsillar enlargement, exudates, petechiae, or lymphadenopathy. Subtle variations or systemic symptoms may be present to aid in the diagnosis, but exact determination of the specific clinical cause of the pharyngitis from clinical criteria alone is notoriously difficult.

**PATHOPHYSIOLOGY**

Viruses cause most cases of pharyngitis. Even the most common cause of bacterial pharyngitis in children, group A β-hemolytic Streptococcus (GAβHS), is responsible for only 30% of cases of pharyngitis. In adults, Streptococcus species account for 23% of cases, with Mycoplasma (9%) and Chlamydia (8%) species also being significant. Recently, Fusobacterium necrophorum, known to be a factor in Lemierre syndrome, has been shown to be a causal agent in 10% of cases of pharyngitis in young adults.

**PRESENTING SIGNS AND SYMPTOMS**

**VIRAL**

In addition to the characteristic pharyngeal pain and dysphagia, viral causes of pharyngitis may also produce low-grade fevers, cough, rhinorrhea, myalgias, or headaches. Viral causes may produce exudates as well, although cervical adenopathy is less common. Common viral causes include rhinoviruses, adenoviruses, Epstein-Barr virus (EBV), herpes simplex virus (HSV), and influenza and parainfluenza viruses. Less common viruses that may cause pharyngitis include respiratory syncytial virus, cytomegalovirus, and primary human immunodeficiency virus (HIV).

Pharyngitis in young adults may be due to infectious mononucleosis, an infection caused by EBV. It is often characterized by thick tonsillar exudates or membranes, as well as other systemic symptoms and signs. Splenomegaly (50%) is frequently present and generalized lymphadenopathy is usually present. Palatal petechiae and peri-orbital edema may likewise be seen.

Also a disease of young adults, HSV infection may produce a painful and characteristic pharyngitis. HSV pharyngitis is typically accompanied by painful vesicles on an erythematous base. These vesicles occur in the pharynx, lips, gums, or buccal mucosa. Fever, lymphadenopathy, and tonsillar exudates may also be present and last for 1 to 2 weeks. HSV pharyngitis may be either a primary infection or reactivation of a previous infection. In addition, bacterial superinfection of affected tissues may occur.

**BACTERIAL**

The most common cause of bacterial pharyngitis in children is GAβHS. It is less frequently implicated in patients older than 15 years. During epidemics, the incidence may double. Characteristic symptoms include tonsillar exudates, high fevers (temperature > 38.3°C), tender cervical adenopathy, and pharyngeal erythema. Headache, nausea, and abdominal pain may also be found. GAβHS pharyngitis usually lacks the traditional symptoms of viral infections (cough, rhinorrhea, myalgias). It occasionally produces a fine sandpaper-like rash that is termed scarlet fever.

Pharyngitis caused by *Mycoplasma pneumoniae* occurs in crowded conditions, may be associated with epidemics, and typically produces a mild pharyngitis. Symptoms include exudates and a hoarse voice, and it may also be associated with lower respiratory symptoms such as cough and occasionally dyspnea.

*Chlamydia pneumoniae* pharyngitis resembles *Mycoplasma* pharyngitis in its occurrence in epidemic and crowded conditions. This pharyngitis is classically described as severe and persistent with tenderness in the deep cervical lymph nodes and occasional associated sinusitis.

Gonococcal and *Chlamydia trachomatis* pharyngitis have varying manifestations from exudative to nonexudative, mildly symptomatic to severely symptomatic, and transient or persistent. These infections result from orogenital sexual transmission, and asymptomatic carriers exist and may unknowingly spread the disease.

*F. necrophorum*, known to be a factor in Lemierre syndrome, frequently causes pharyngitis in young adults and is a common causative agent of recurrent pharyngitis as well.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

**VIRAL**

Diagnostic testing for viral pharyngitis is limited to only a few different specific causes. EBV infection may be diagnosed with a few different tests. The peripheral blood smears of up to 75% of patients with EBV demonstrate atypical mononuclear cells. An EBV monospot test may be positive in up to 95% of adults and 90% of children, but the test may not be positive during the first few days of illness. EBV IgM antibodies develop in 100% of patients but may also initially be negative.

Herpes pharyngitis is diagnosed by serologic tests, herpes culture, or cytopathologic tests from lesion scrapings. Primary HIV pharyngitis may be diagnosed with serologic tests such as Western blot and enzyme-linked immunosorbent assay. New rapid HIV tests using buccal swabs have also been shown to be effective in an ED setting.

**BACTERIAL**

Diagnostic testing for GAβHS is a subject of some controversy. Although the diagnosis of GAβHS infection is important in

**DOCUMENTATION**

Visualization of the airway, including patients with suspected epiglottitis or supraglottitis

Work of breathing and use of accessory muscles

Patient’s ability to take fluids by mouth

Discussion (and understanding) with the patient and family of the need for close observation and what symptoms to return for arranged follow-up
result are treated. The guidelines have suggested different approaches to adults with pharyngitis, however. The IDSA guidelines propose treating all adults in a fashion similar to their pediatric recommendations. The ASIM guidelines, though, allow two additional approaches for adults, including performing a rapid test on all adults with a Centor score of 2 or 3 and treating those with a positive test result, as well as empirically treating all patients with a score of 4 or higher. The final approach endorsed by the ASIM suggests testing no adults but treating all adults with a Centor score of 3 or 4 empirically. A recent analysis comparing the different recommendations found that an approach using throat cultures had a sensitivity of 100%; approaches using rapid streptococcal tests involving clinical criteria alone had sensitivities of approximately 75%. Furthermore, the specificity of clinical criteria alone was below 50%, thus suggesting that many adults were prescribed antibiotics for pharyngitis unnecessarily.

Diagnostic testing for other causes of bacterial pharyngitis requires either culture on special media (Thayer-Martin agar for gonococcal infection) or specialized antigen or serologic testing. *Mycoplasma* infection may be detected by culture, serologic testing, or rapid antigen testing. Antigen detection may also be used for chlamydial infection, as well as culture or serologic testing.

**DIFFERENTIAL DIAGNOSIS**

In addition to the infectious causes of pharyngitis mentioned in this chapter, a number of other infectious and noninfectious causes must be considered in the differential diagnosis of patients with pharyngitis. Among dangerous infections, deep space infections such as retropharyngeal abscesses, peritonsillar abscesses, and epiglottitis must be considered. Noninfectious but potentially serious diseases in the differential diagnosis include drug or allergic reactions, foreign body or caustic ingestion, angioedema, and chemical or thermal burns.

**TREATMENT**

Fortunately, most cases of pharyngitis are acute and self-limited. Supportive care with analgesics and antipyretics and perhaps topical anesthetics (lozenges or sprays) helps in alleviating the symptoms. However, even most infectious causes of pharyngitis rarely cause serious complications.

Among viral causes, few allow specific treatment. HSV infection may be treated with acyclovir, foscarnet, or valacyclovir, all of which produce similar earlier resolution of symptoms but do not eradicate the etiologic agent. Infectious mononucleosis has no effective antiviral agent, but some important aspects of disposition remain important. Patients suspected of having infectious mononucleosis should be advised against contact sports for 6 to 8 weeks because of concern for potential serious splenic injury. Patients with infectious mononucleosis in whom tonsillar edema and hypertrophy severely limit adequate oral hydration secondary to dysphagia may receive corticosteroids (dexamethasone, 6 to 10 mg intramuscularly) to aid in relief of symptoms.

Of all bacterial causes, GABHS is the most common and most often studied. Regardless of the diagnostic strategy used, antimicrobial therapy directed at streptococcal species will prevent many serious complications of streptococcal pharyngitis, including rheumatic fever, accurate diagnosis of GABHS pharyngitis is notoriously difficult. The only valid method of diagnosing acute GABHS infection involves acute and chronic antistreptolysin O titers. However, this method is far from practical in the emergency setting. Throat cultures have a sensitivity of nearly 90% for detecting *Streptococcus pyogenes* in the pharynx, but their accuracy may vary, depending on recent antibiotic use and culture and collection techniques.

Rapid diagnostic testing for GABHS detects antigens via varying techniques, including latex agglutination, enzyme-linked or optical immunoassay, and DNA luminescent probes. Specificities are reported to be greater than 90% with sensitivities between 60% and 95%. A positive test appears to be a reliable indicator of the presence of GABHS in the pharynx, but a number of factors must be considered. Some patients with a positive test may be asymptomatic carriers, and rapid tests may be negative in patients with low bacterial counts.

In addition to cultures and rapid streptococcal tests, a number of authors have proposed clinical criteria to aid in the diagnosis of GABHS pharyngitis. The most well known are the Centor criteria and the McIssac modifications of these criteria. The modified Centor score gives one point each to temperature higher than 38°C, swollen tender anterior cervical nodes, tonsillar swelling or exudates, absence of upper respiratory tract symptoms (e.g., cough, coryza), and age between 3 and 15 years. If the patient is older than 45 years, a point is subtracted. If the score is 1 or less, no further testing or treatment is warranted. For scores of 2 to 3, further testing may be indicated, such as cultures or rapid streptococcal tests. For scores of 4 or higher, no further testing is required and all patients may receive antibiotics for GABHS pharyngitis (Box 29.1).

In two recent guidelines (from the Infectious Diseases Society of America [IDSA] and the American Society of Internal Medicine [ASIM]), slightly different approaches to patients with pharyngitis have been proposed. In children the guidelines are similar and call for the use of a rapid test in all children; those with a positive test are treated, those with a negative test undergo a throat culture, and those with a positive culture

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**BOX 29.1 Clinical Diagnosis of Streptococcal Pharyngitis**

The modified Centor criteria constitute a scoring system that can increase detection of group A β-hemolytic streptococcal pharyngitis. One point each is assigned to the following features:
- Temperature > 38°C
- Swollen, tender anterior cervical nodes
- Tonsillar swelling or exudates
- Absence of symptoms of upper respiratory tract infection (e.g., cough, coryza)
- Age between 3 and 15 years (if the patient is older than 45 years old, a point is taken off)

If the score is 1 or less—no treatment.
For scores of 2 to 3, further testing may be indicated, such as cultures or rapid streptococcal tests.
For scores of 4 or greater, no further testing is required and all patients may receive antibiotics for group A β-hemolytic streptococcal pharyngitis.
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probably be very effective. There are two main reasons to treat GABHS pharyngitis—to improve symptoms and decrease complications. Patients who receive antibiotics during the first 2 to 3 days of symptoms are likely to improve 1 to 2 days faster than those not taking antibiotics. In terms of complications, the most feared complication of GABHS infection is acute rheumatic fever. Antibiotic treatment within the first 9 days of infection decreases the rate of rheumatic fever after GABHS infection. However, the incidence of acute rheumatic fever has fallen drastically in the last few decades such that the number needed to treat to prevent one case of rheumatic fever has risen from 63 to now more than 4000. This is due to the amount of antibiotics prescribed for GABHS infection over the years, as well as the presence of less virulent strains of bacteria and improved living conditions. Antibiotic use also limits transmission to others and the amount of suppressive complications, including peritonsillar abscesses, retropharyngeal and other deep space infections, otitis, and mastoiditis. Use of antibiotics has no effect on the incidence of poststreptococcal glomerulonephritis.

Penicillin remains an effective choice for GABHS pharyngitis. Benzathine penicillin, 1.2 million units, or a 10-day course of penicillin VK, 500 mg orally twice per day, both effectively treat pharyngitis and reduce symptoms. The intramuscular route may be more effective in treating streptococcal pharyngitis secondary to compliance issues but is associated with more severe allergic reactions. Alternative regimens include macrolide antibiotics (e.g., erythromycin, azithromycin) and clindamycin. Oral cephalosporins have been shown to result in slightly improved cure rates when compared with penicillins and may be taken over a shorter course (5 days).

Mycoplasma pharyngitis may be treated with a course of macrolide antibiotics or tetracycline or doxycycline. *C. pneumoniae* pharyngitis is treated for 10 days with doxycycline, trimethoprim-sulfamethoxazole, or a macrolide. *C. trachomatis* pharyngitis may require repeated courses or prolonged use of antibiotics. *F. necrophorum* is susceptible to penicillin or clindamycin.

Corticosteroids, in conjunction with antibiotics, decrease the duration of symptoms in patients with pharyngitis without increasing complications. Steroids are particularly useful in patients with profound tonsillar hypertrophy and edema or in patients with severe dysphagia and mild dehydration. By reducing the inflammatory response, steroids allow patients to swallow without significantly reducing the pain sooner.

**PATIENT TEACHING TIPS**

<table>
<thead>
<tr>
<th>When to take antibiotics</th>
<th>When to take pain medicine</th>
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</thead>
<tbody>
<tr>
<td>Return for</td>
<td></td>
</tr>
<tr>
<td>Increased difficulty breathing</td>
<td>Increased difficulty swallowing</td>
</tr>
<tr>
<td>Worsening pain</td>
<td>Confusion or changes in thinking</td>
</tr>
<tr>
<td>Worsening fever</td>
<td></td>
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</table>

**DISPOSITION**

Patients with pharyngitis usually have full resolution of their symptoms without difficulty. However, life-threatening complications do occur. Infectious mononucleosis may lead to airway obstruction, epiglottitis, deep space infections, and distant or systemic disorders such as splenic injury, thrombocytopenia, or hemolytic anemia. Complications of GABHS pharyngitis include deep space infections, toxic shock syndrome, peritonsillar abscesses and autoimmune-related glomerulonephritis, rheumatic fever, and erythema nodosum. Patients in whom airway compromise is at all a concern should be admitted to the hospital for observation and personnel with advanced airway skills alerted for the potential need for a surgical airway.

**PERITONSILLITIS, PERITONSILLAR CELLULITIS, AND ABSCESS**

Peritonsillar cellulitis and peritonsillar abscess exemplify the continuum of peritonsillar infections best characterized as peritonsillitis. Peritonsillar infections represent the most common deep infection of the head and neck region. These infections occur more frequently in teenagers and young adults, but they can develop at any age. The infection begins most commonly when tonsillitis spreads beyond the boundary of the tonsillar capsule and invades the potential space between the lateral aspect of the tonsillar capsule and the superior constrictor muscle of the pharynx. The local spread of infection begins as cellulitis but progresses to abscess formation if left untreated. Predisposing factors include chronic tonsillitis, dental infections, smoking, and infectious mononucleosis. Peritonsillitis can occur in patients who have previously undergone complete tonsillectomy.

**EPIDEMIOLOGY AND PATHOPHYSIOLOGY**

Symptoms of peritonsillitis begin with the symptoms of tonsillitis and progress over a few days to increasing pain and unilateral symptoms. Patients with peritonsillitis often complain of dysphagia, odynophagia, drooling, trismus, and referred otalgia on the affected side. In addition, patients will describe changes in voice (hoarseness or a “hot potato” voice) as a result of decreased movement of the palate. Patients may report recurrent bouts of pharyngitis or trials of antibiotics without resolution of symptoms.

**SYMPTOMS**

On physical examination the patient may have trismus, which may prevent clear visualization of the pharynx but may respond to benzodiazepines. Erythematous mucosa and purulent exudates may be present. The distinguishing features of a peritonsillar abscess include unilateral swelling and displacement of the infected tonsil and frequently the soft palate toward the midline with resultant deviation of the uvula to the contralateral side. Peritonsillar abscesses are generally unilateral and can occur in the superior pole of the tonsil.
DIFFERENTIAL DIAGNOSIS

The differential diagnosis of peritonsillitis includes infectious mononucleosis, cervical adenitis, tubercular granuloma, internal carotid artery aneurysms, and neoplasms. Obviously, infectious causes will be manifested in a more rapid manner than chronic granulomas, masses, or aneurysms. Pulsatile masses or findings that do not suggest an acute infectious cause must be evaluated with other imaging techniques to elucidate the cause of the symptoms.

DIAGNOSTIC TESTING

Plain radiographs do not aid in the diagnosis or treatment of uncomplicated cases of peritonsillitis. Although it is often difficult to distinguish peritonsillar cellulitis from peritonsillar abscesses, a number of modalities can aid in diagnosis. For patients who cannot lie down or who are unable to cooperate with needle aspiration, intraoral ultrasound is a useful test. Computed tomography (CT) is helpful in delineating the extent and scope of the abscess, but it may be difficult for the patient to lie supine during the study.

TREATMENT

Pending airway obstruction requires emergency aspiration of a peritonsillar abscess. For less urgent conditions the classic recommendation was incision and drainage. Needle aspiration, with or without ultrasound guidance, provides a diagnosis, induces less pain, and is easier to perform than traditional incision and drainage. Approximately 10% of needle-aspirated abscesses require repeated aspiration.

Patients should receive antibiotics (penicillin or clindamycin) and steroids for relief of the pain and swelling, regardless of whether the abscess is drained. One strategy if the peritonsillitis is thought to be cellulitis or if the aspiration reveals no purulence is to start antibiotic and steroid treatment and assess the patient in 24 hours to see whether the symptoms are improving or another aspiration is required.

DISPOSITION

Most patients with peritonsillitis can be treated safely and discharged home with antibiotics, steroids, and close follow-up in 24 to 48 hours. For patients with severe dehydration or severe trismus requiring intravenous hydration, a brief inpatient stay may be required. Any patient with tissue edema and potential airway compromise must be admitted to the hospital for observation and potential aggressive airway management.

EPITROCHLEUM AND SUPRAGLOTTITIS

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Inflammation and edema of the epiglottis may result in rapid and life-threatening airway obstruction if not identified and treated effectively. In addition, the tissues immediately adjacent to the epiglottis (arytenoids, false vocal cords, and pharyngeal wall) may also become edematous and result in a similar infection known as supraglottitis. The signs and symptoms of the two diseases may be remarkably similar, although their populations may be somewhat different. Until introduction of the Haemophilus influenzae type B (Hib) vaccine in the mid-1980s, epiglottitis was a disease of children. The annual incidence decreased dramatically to a very low 0.3 to 0.6 per 100,000 children. Adult epiglottitis appears to be on the rise. One study showed a 31% rise in the incidence of adult epiglottitis over an 18-year period. Incidence rates for adults cluster near 3 per 100,000, with a case fatality rate reported to be as high as 7%. The lower overall bacteremia rates in adults than in children suggest that adults may have more viral causes, as well as other noninfectious causes. Noninfectious causes include trauma, ingestion of caustic substances, or thermal injuries from illicit drug inhalation. Of organisms recovered in blood cultures, Hib still predominates, but Streptococcus and Staphylococcus species may be isolated as well.

PRESENTING SIGNS AND SYMPTOMS

CHILDREN

Epiglottitis in children produces a dramatic progression from a relatively well child to a toxic-appearing one. Eighty-five percent of patients are sick for less than 24 hours. Affected children appear anxious, may be sitting forward in a tripod position, and may have difficult clearing secretions (Table 29.1).

ADULTS

Adults with epiglottitis have a mean age of between 42 and 50 years. Unlike children, adults are usually seen after 2 to 3 days of symptoms. Adults exhibit odynophagia and throat pain rather than the drooling and stridor of pediatric patients. This difference is thought to be due to the larger and more rigid trachea and lack of generous lymph tissue in adults. The pharyngeal pain may be disproportionate to the clinical findings. Fever is frequently absent in adults until the later stages. Additionally, epiglottitis or supraglottitis is frequently misdiagnosed in adults as streptococcal pharyngitis, only to have the true diagnosis made later (Table 29.2).

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<thead>
<tr>
<th>Table 29.1</th>
<th>Symptoms of Epiglottitis or Supraglottitis in Children</th>
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</thead>
<tbody>
<tr>
<td>SYMPTOM</td>
<td>FREQUENCY (%)</td>
</tr>
<tr>
<td>Fever</td>
<td>100</td>
</tr>
<tr>
<td>Difficulty breathing</td>
<td>97</td>
</tr>
<tr>
<td>Irritability</td>
<td>89</td>
</tr>
<tr>
<td>Change in voice or cry</td>
<td>82</td>
</tr>
<tr>
<td>Stridor</td>
<td>92</td>
</tr>
<tr>
<td>Retractions</td>
<td>88</td>
</tr>
</tbody>
</table>
complete airway obstruction without warning. Endotracheal intubation must be performed under direct visualization either with a laryngoscope or with a fiberoptic bronchoscope/laryngoscope. At all times, equipment and personnel for emergency cricothyrotomy must be present.

Patients should receive broad-spectrum antibiotics that include coverage against *H. influenzae*, as well as other common bacterial pathogens. Ampicillin-sulbactam and ceftriaxone are both acceptable first-line agents. Steroids and racemic epinephrine have not been conclusively proved to be of benefit in these patients.

**DISPOSITION**

All patients suspected of having epiglottitis or supraglottitis should be admitted to the hospital. Adults without respiratory distress (drooling, stridor, or dyspnea) may be admitted without intubation and observed, but in a carefully monitored setting. The vast majority of children with suspected epiglottitis should be intubated in the operating room and then admitted to the intensive care unit (ICU) for intravenous antibiotics. Rarely, a child with epiglottis may not require intubation. However, this is done only in consultation with the pediatric anesthesiologist and intensivist after they have evaluated the patient. Children with epiglottis who are not intubated should always be admitted to an ICU setting because of their very high potential for rapid deterioration.

**LUDWIG ANGINA**

**EPIDEMIOLOGY AND PATHOPHYSIOLOGY**

First described in 1836, Ludwig angina is a fast-spreading, potentially lethal infection of the submandibular, sublingual, and submental spaces. These spaces effectively function as one unit, and infection can easily spread between them. True Ludwig angina involves all of the submandibular spaces; however, serious and life-threatening infections can occur...
with infection of only some of the spaces. Before the advent of antibiotics and surgical decompression, mortality with this disease approached 50%, but it is now commonly reported to be less than 10%.13

Approximately 80% of cases involve patients with odontogenic disease. Patients with infection or recent extraction of the second or third molars are particularly at risk because the roots of these teeth extend below the mylohyoid ridge. Abscesses located in this region can easily perforate the lingual plate of the mandible, thereby allowing direct spread of infection to the submandibular spaces. Other predisposing conditions include deep lacerations or trauma to the floor of the mouth, mandibular fractures, and salivary calculi.

**PRESENTING SIGNS AND SYMPTOMS**

Patients with Ludwig angina appear ill and anxious. Males predominate over females by a ratio of 3:2. The classic symptoms include tooth pain (79%), swelling of the neck or chin (71%), dysphagia (52%), neck pain (33%), respiratory symptoms (dyspnea, tachypnea, stridor) (27%), and dysarthria (18%).

Physical findings in almost all patients include bilateral submandibular swelling, fever, and an elevated or protruding tongue (Figs. 29.2 through 29.4). Trismus is found in approximately half of all patients. Complications of Ludwig angina can be disastrous, with infection spreading to the mediastinum, pericardium, or pleural cavity, as well as the lungs and great vessels.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The differential diagnosis in a patient with suspected Ludwig angina should also include Lemierre syndrome (septic thrombophlebitis of the superior internal jugular vein), which is distinguished from Ludwig angina by the unilateral nature of the infection. Tumors in the floor of the mouth can be distinguished from Ludwig angina by their tendency to exhibit much slower growth. Mandibular abscesses are generally unilateral and produce less swelling of the floor of the mouth.

**DIAGNOSTIC TESTING**

Because Ludwig angina is predominantly a clinical diagnosis, diagnostic testing commonly delineates the extent of infection, not whether it is present. Soft tissue lateral neck radiographs may be helpful in showing edema of the soft tissues and air in the soft tissue spaces. If the patient is able to lie supine for a short while, helical CT may be helpful in delineating progression of the abscess and allow more accurate surgical débridement (Fig. 29.5, A and B). If the safety of the patient lying supine is at all in question, the airway must be secured first. At all times the utmost caution is required in these patients, and securing the airway will probably involve the joint capabilities of anesthesia, otorhinolaryngologic, and
general surgical colleagues. No airway maneuvers may be attempted until the equipment and personnel capable of obtaining a surgical airway are present at the bedside.

**TREATMENT**

Treatment of patients with Ludwig angina predominantly involves three issues: protection of the airway though nasal (fiberoptic), oropharyngeal, or surgical techniques; administration of antibiotics; and surgical débridement. The antibiotic selected should have broad-spectrum coverage because up to 50% of patients have polymicrobial involvement. Although *Streptococcus*, *Staphylococcus*, and *Bacteroides* are the organisms commonly cultured, other species have included *Klebsiella*, *H. influenzae*, *Proteus*, and *Pseudomonas* species. Once the airway is protected, 50% will resolve with only antibiotics.

**DISPOSITION**

All patients with Ludwig angina should be admitted to an ICU for further monitoring and fluid resuscitation should the clinical picture deteriorate into one of bacteremia and sepsis. ICU observation is required for those with the rare milder cases who are not immediately intubated, because progression to airway compromise and obstruction may be rapid.

**LEMIERRE SYNDROME**

**PATHOPHYSIOLOGY**

A particularly dangerous and intractable complication of nearby infections, Lemierre syndrome is a suppurative thrombophlebitis located within the internal jugular vein. This dangerous but fortunately rare complication of nearby infections in the pharynx or mastoid may produce septic emboli.

Lemierre syndrome is often caused by the organism *F. necrophorum*, a gram-negative anaerobe that produces endotoxin and is also responsible for many cases of acute and recurring pharyngitis, as well as peritonsillar abscesses. *F. necrophorum* can colonize the upper respiratory, gastrointestinal, and genitourinary tracts. Other organisms that may also cause this particular variant of septic thrombophlebitis include *Bacteroides melaninogenicus*, *Eikenella corroden*, and non–group A streptococci.

**PRESENTING SIGNS AND SYMPTOMS**

The signs and symptoms of Lemierre syndrome may mimic those of other localized infections and include fever, swelling, tenderness, and pain with range of motion of the neck. It does, however, include the additional feature of obstruction of normal blood flow in the affected internal jugular vein, which may cause unilateral swelling, edema, and induration in the tissues on the affected side, regardless of whether they actually infected.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

When considering Lemierre syndrome, one must also consider Ludwig angina or other deep space infections in the neck and parapharyngeal tissues. Whether from odontogenic, lymphatic, or other sources, deep space infections in the neck and pharynx must be taken very seriously.

**DIAGNOSTIC TESTING**

Lemierre syndrome is a diagnosis that when suspected requires confirmatory testing to document involvement of the

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Fig. 29.5  A, Computed tomography (CT) scan of Ludwig angina showing infection in the submandibular spaces (arrow). B, CT scan of Ludwig angina showing infection in the submandibular spaces.
internal jugular vein. Soft tissue lateral neck radiographs may be helpful in showing edema of the soft tissues and air in the soft tissue spaces, but they lack the specific information on vein involvement needed to make the diagnosis of Lemierre syndrome. The diagnosis is made most commonly via a contrast-enhanced CT scan of the neck. In addition, magnetic resonance imaging (MRI) may also delineate the extent of involvement of the jugular vein. Both these modalities will also provide information about the surrounding anatomy, including distortion of nearby tissues and localized pockets of infection that must be drained. Other tests that may aid in the diagnosis of Lemierre syndrome include duplex ultrasonography, retrograde venography, and gallium scanning. If there is any question about the safety of the patient lying supine, the airway must be secured first.

**TREATMENT**

Treatment of Lemierre syndrome must include both heparin and antibiotics. The thrombus provides a nidus of ongoing infection and, subsequently, septic emboli. The anticoagulation effectivly limits progression of the thrombosis and, in turn, limits the development of further septic emboli. The initial choice of antibiotics should be broad spectrum. Prolonged high-dose broad-spectrum antimicrobial therapy is recommended because of the high risk for septic emboli and endocarditis. Close consultation with an infectious disease specialist would be helpful in such cases.

**DISPOSITION**

Patients with Lemierre syndrome clearly need to be admitted to the hospital, and consultation with a vascular surgeon may be helpful if thrombectomy is being considered.

**RETROPHARYNGEAL ABSCESSESS**

**PATHOPHYSIOLOGY**

Even though retropharyngeal abscesses predominantly occur in the pediatric population, adults may also be affected. Pediatric patients possess prominent lymph nodes in the retropharyngeal area, which can become infected and progress to cellulitis and abscess formation. These nodes atrophy by 6 years of age, and the incidence of retropharyngeal abscess drops precipitously.

The cause of retropharyngeal abscesses in the nonpediatric population differs greatly from the local lymph node infections in the age group younger than 6 years. The cause of adult retropharyngeal abscesses ranges from extension of other pharyngeal infections (e.g., parotitis, peritonsillar abscess, tonsillitis, Ludwig angina, dental infections) to instrumentation and other trauma (fishbones or dental instruments) to hematologic spread of systemic infections. Microbiologic testing of retropharyngeal abscesses reveals a diverse array of causative bacteria. Although the preponderance of abscesses are polymicrobial (up to 86%), some species predominately. The majority of aerobic pathogens include *Streptococcus* and *Staphylococcus* species, and *Bacteroides*, *Fusiform*, and *Peptostreptococcus* species dominate the anaerobic isolates. Tuberculous and fungal causes must also be considered.

**PRESENTING SIGNS AND SYMPTOMS**

Patients will have a sore throat, as well as any number of other symptoms, including dysphagia, drooling, odynophagia, neck pain, and a muffled voice. The dysphonia frequently sounds like a duck quack (cri du canard). Patients may complain of a feeling of a lump in their throat and may prefer to lie supine or with their neck extended to keep the swelling in their posterior pharynx from compressing their upper airway. In the pediatric population, many case series have reported the mean age as being 3 years. It is uncommon for children in this age group to describe these symptoms, and the more common complaints are related to feeding, fever, and stridor.

On physical examination only a minority of adult patients (37%) will have visible swelling of the posterior pharynx. Other physical findings might include cervical lymphadenopathy, torticollis, and trismus. Palpation of the neck or moving the larynx and trachea side to side (tracheal “rock” sign) may induce pain. Pain and other symptoms out of proportion to the findings on physical examination require further diagnostic testing and evaluation.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Other possible diagnoses to consider in these patients include other pharyngeal infections such as pharyngitis, peritonsillar abscess, and infectious mononucleosis. Abrasions or caustic burns involving the posterior pharynx might produce similar symptoms of dysphagia and odynophagia. Other causes of retropharyngeal swelling and pain may include cervical fracture, hematoma, or other causes of tissue edema such as tendonitis. Finally, distinguishing between a true abscess and pharyngeal cellulitis may be difficult and might be accomplished only with imaging techniques.

**DIAGNOSTIC TESTING**

Imaging of retropharyngeal abscesses involves two main types of radiologic tests, plain lateral neck radiographs and CT scans. Early studies of normal lateral radiographs suggest that the upper limit of normal for the retropharyngeal spaces is 7 mm at C2 and 22 mm at C6. Anything beyond 7 mm is considered pathologic in both children and adults. In addition, retropharyngeal abscesses will displace the larynx and esophagus anteriorly. If there is still a question about the extent of involvement or the distinction between cellulitis and abscess, CT of the neck (Fig. 29.6) or MRI (Fig. 29.7) will easily elucidate the exact anatomic extension of the abscess. Of course, patients must be stable enough to lie flat in the supine position for the time needed to complete the study. In addition, intraoral ultrasound is another modality with great potential for distinguishing between abscess and cellulitis.
SECTION III  HEAD AND NECK DISORDERS

Fig. 29.6  Computed tomography scan of a retropharyngeal abscess (arrow).

Fig. 29.7  Magnetic resonance imaging of a retropharyngeal abscess (white arrow). Note extension into the vertebral bodies (osteomyelitis) (black arrow).

Fig. 29.8  Computed tomography scan of a retropharyngeal abscess (arrow) extending laterally and inferiorly.

Fig. 29.9  Computed tomography scan showing inferior extension of a retropharyngeal abscess into the mediastinum.

TREATMENT AND DISPOSITION

Retropharyngeal infections require broad-spectrum intravenous antibiotics. The antibiotics must cover anaerobes, as well as gram-positive and gram-negative species. Although most retropharyngeal abscesses require surgical drainage, selected cases may occasionally be managed with intravenous antibiotics alone.19 Even if surgical drainage is deferred, admission to a monitored bed after thorough airway evaluation is required.

Complications from retropharyngeal abscesses include spread of infection inferiorly or laterally (Fig. 29.8) into the mediastinum (Fig. 29.9), pericardium, and nearby vascular structures. Osteomyelitis (see Fig. 29.7), transverse myelitis, and epidural abscesses have also been reported.

SUGGESTED READINGS


REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES