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CHAPTER 11

Pharyngitis, Stomatitis, Peritonsillar, and Retropharyngeal Abscess

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PHARYNGITIS

Definition

Pharyngitis is defined as inflammation of the pharynx most often involving the palatine tonsils, if present. Anatomically, the oral pharynx is bordered anteriorly by the dorsum of the tongue and the palatoglossal fold, which delineates the limit of the oral cavity. The oral pharynx is normally a heavily colonized site. The palatine tonsils are located in the lateral wall of the pharynx. They are highly vascularized lymphoid aggregates between the mucosal folds created by the palatoglossus and palatopharyngeus muscles. They are covered by stratified squamous epithelium, which continues down into deep crypts. These anatomic features make the tonsils susceptible to infection and the surrounding tissues at risk for extension of that infection. Tonsils vary widely in size and may be sessile or pedunculated.

Epidemiology

Upper respiratory tract infections, including pharyngitis, are responsible for more annual physician visits in the United States than any other infectious disease. Most of these visits occur during the winter when respiratory viruses are prevalent. The causes of pharyngitis are both bacterial and viral. Overall most cases of pharyngitis have a viral etiology. Of the bacterial causes, group A β-hemolytic streptococcal (GAβHS) pharyngitis is the most common.

GAβHS pharyngitis is endemic in the United States and accounts for 15% to 30% of all episodes of pharyngitis.

Cases generally peak in the late winter and early spring. Children 5 to 11 years of age are most commonly infected. Crowded living conditions facilitate the person-to-person spread of GAβHS. Throat culture surveys of asymptomatic children during school outbreaks of pharyngitis have yielded GAβHS prevalence rates as high as 50%. Untreated GAβHS pharyngitis is particularly contagious early in the acute illness and for the first 2 weeks after the organism has been acquired. The incubation period is 2 to 5 days. Carriage of GAβHS may persist for months, but the risk of transmission from chronic carriers to others is minimal.

Etiology

The most common viral causes of pharyngitis include rhinovirus, coronavirus, adenovirus (types 3, 4, 7, 14, and 21), herpes simplex types 1 and 2, parainfluenza virus (types 1 through 4), influenza types A and B, coxsackievirus A (types 2, 4, 5, 6, 8, and 10), Epstein-Barr virus (EBV), cytomegalovirus (CMV), and human immunodeficiency virus (HIV) type 1. Some viral causes of pharyngitis can mimic a bacterial etiology (Table 11–1). With the exception of pharyngitis caused by adenovirus and EBV, pharyngitis caused by respiratory viruses is generally mild and is accompanied by cough and coryza.

Adenoviral pharyngitis is typically more severe with fever, erythema of the pharynx, follicular hyperplasia of the palatine tonsils with purulent exudates, and cervical lymph node enlargement. When accompanied by conjunctivitis, this syndrome is called *pharyngoconjunctival fever.* This syndrome can occur sporadically and in epidemics. Adenoviral pharyngitis can persist as long as 7 days and conjunctivitis for 10 to 14 days.

Infectious mononucleosis caused by EBV can cause a severe pharyngitis with prominent tonsillar enlargement and erythema with purulent exudates; it is often confused with GAβHS disease. Fever and pharyngitis last 1 to 3 weeks. EBV infection is also associated with hepatosplenomegaly and generalized lymphadenopathy, which usually subside over 3 to 6 weeks. Infectious mononucleosis is generally a disease of adolescents and young adults.

Acute retroviral syndrome, a manifestation of acute HIV infection, typically has an incubation period that ranges from 3 to 5 weeks with symptoms that include fever, nonexudative pharyngitis, lymphadenopathy, arthralgia, myalgia, and lethargy. In 40% to 80% of patients, a maculopapular rash is present.

Common bacterial causes of pharyngitis are GAβHS, group C β-hemolytic streptococci, *Neisseria gonorrhoeae*, *Arcanobacterium haemolyticum,* and *Corynebacterium diphtheriae*. *Chlamydia pneumoniae* and *Mycoplasma pneumoniae* can also cause pharyngitis (Table 11–2). Of the bacterial causes of pharyngitis, GAβHS is by far the most common, causing 15% to 30% of cases of pharyngitis in children; affected children are primarily of school age.

Humans are the primary reservoir of *A. haemolyticum,* which is spread person to person via droplet respiratory secretions. However, isolation of this organism from the nasopharynx of asymptomatic patients is rare. This organism primarily infects adolescents and young adults and accounts for 0.5% to 3% of cases of acute pharyngitis. The incubation period is unknown.

Gonococcal pharyngitis, which is typically asymptomatic, should be suspected in patients who practice fellatio and who present with a mild pharyngitis. It can occur in the absence of genital infection. It can also present in prepubertal children as a result of sexual abuse.

Pathogenesis

The pathogenesis of pharyngitis typically involves inhalation of organisms in large droplets or by direct contact with respiratory secretions. Because the free surface of the tonsils is covered by stratified squamous epithelium, which extends inward into numerous branching crypts, the palatine tonsils are susceptible to infection. The lymph nodules lie beneath this epithelium and along the crypts. The lateral or deep surface of the tonsils is covered by a fibrous connective tissue capsule. Because the tonsils are highly vascularized, extension of infection is not uncommon.

Table 11-1 Viral Agents That Can Mimic Bacterial Pharyngitis

Adenovirus
Epstein-Barr virus (EBV)
Cytomegalovirus (CMV)
Herpes simplex (primary infection)
Coxsackie A virus (herpangina)
Human immunodeficiency virus (acute retroviral syndrome)

Table 11-2 Bacterial Causes of Pharyngitis

Streptococcus pyogenes (GAβHS)
Group C streptococci
Group G streptococci
Arcanobacterium haemolyticum
Neisseria gonorrhoeae
Mycoplasma pneumoniae
Chlamydia pneumoniae
Corynebacterium diphtheriae
Yersinia enterocolitica
Francisella tularensis
Coxiella burnetii

GAβHS, group A β-hemolytic streptococcus.

Clinical Presentation

History

The typical patient with GAβHS pharyngitis is a school-age child with the sudden onset of fever and sore throat in the late winter or spring. Many of the viral etiologies of pharyngitis also circulate in the winter; however, GAβHS is unusual in children younger than 3 years old, and therefore fever and pharyngitis in this age group is more likely to be of viral etiology.

Symptoms

The classic symptoms of GAβHS pharyngitis include a sudden onset of sore throat, dysphagia, fever, abdominal pain with or without nausea and vomiting, and headache. Symptoms of coryza, hoarseness, cough, and diarrhea suggest a viral cause. Cough, in particular, is considered a negative predictor of GAβHS pharyngitis. Therefore indicators of low risk for GAβHS include the absence of fever (without the use of antipyretics), the absence of pharyngeal erythema, and the presence of obvious symptoms of the common cold such as cough and coryza.

Physical Findings

The typical signs of GAβHS are tonsillopharyngeal erythema with purulent exudates, soft palate petechiae, erythematous and edematous uvula, and tender anterior cervical lymphadenitis (Figure 11–1). Physical findings that are not suggestive of GAβHS include conjunctivitis, anterior stomatitis, and ulcerative lesions in the pharynx. A scarlatiniform eruption may accompany the pharyngitis. This entity, called *scarlet fever*, is not usually seen in patients younger than 3 years of age or in adults. The rash of scarlet fever is a result of the presence of a bacteriophage that produces an erythrogenic toxin, exotoxin A, carried by some GAβHS strains.

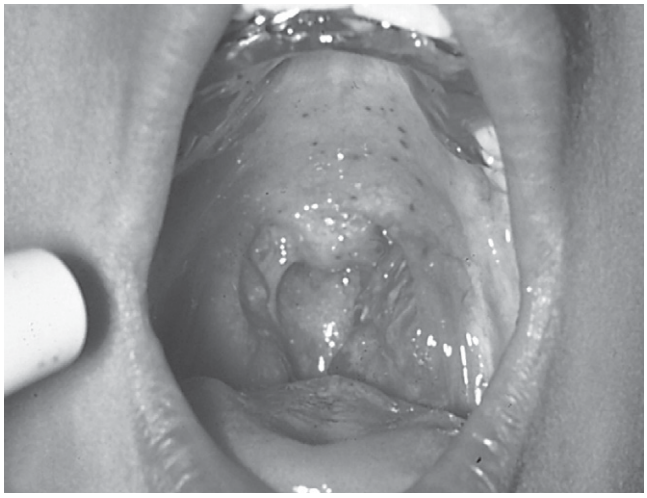


Figure 11-1 Group A β-hemolytic streptococcal pharyngitis demonstrating acute tonsillar enlargement, intense erythema, and palatal petechiae. (From Zitelli B, Davis H, eds: The Atlas of Pediatric Physical Diagnosis, 4th ed. Philadelphia: Elsevier, 2002.)

A. haemolyticum (formerly known as *Corynebacterium haemolyticum)* is often indistinguishable from GAβHS pharyngitis clinically; however, palatal petechiae and strawberry tongue are usually absent. Typical symptoms include fever, pharyngeal exudates, lymphadenopathy, and a maculopapular or scarlatiniform rash in approximately 50% of patients. The rash begins on the extensor surfaces of the distal extremities, spreading centripetally to the chest and back and sparing the face, palms, and soles. In addition, a membranous pharyngitis that mimics diphtheria has been attributed to *A. haemolyticum.* Suppurative complications of *A. haemolyticum* pharyngitis include peritonsillar abscess (PTA) similar to that of GAβHS infection (see later).

Laboratory Findings

Patients with pharyngitis, without common cold symptoms, should be tested for the presence of GAβHS in the throat by a rapid antigen detection test and a throat culture. A throat culture, when properly performed, remains the gold standard for the diagnosis of GAβHS. The sensitivity of a throat culture is 90% or higher, whereas the sensitivity of the rapid antigen test varies from 70% to 90% depending upon the test. Gonococcal pharyngitis can be diagnosed by Gram stain of a pharyngeal/tonsillar swab looking for intracellular gram-negative diplococci and by gonococcal culture. (Better yield is obtained when the specimen is inoculated in the patient-care area directly onto nutritive growth media such as modified Thayer-Martin medium and incubated immediately at 35° to 37° C in an atmosphere of 3% to 10% carbon dioxide). Interpretation of culture results as *Neisseria gonorrhoeae* from the pharynx of young children should be done cautiously because of the high carriage rate of nonpathogenic *Neisseria* species. Ligase chain reaction to amplify gonococcal-specific nucleic acid should not be used for the diagnosis of gonococcal pharyngitis because false-positive results can occur. All patients with presumed or proven gonococcal pharyngitis should also be evaluated for concurrent syphilis, hepatitis B, HIV, and *Chlamydia trachomatis* infections.

Although *A. haemolyticum* can be grown on standard blood agar plates, the colonies are small, with narrow bands of hemolysis that may not be visible for 48 to 72 hours. Growth can be enhanced by the use of rabbit or human blood agar and incubation in 5% carbon dioxide, which results in larger colony sizes and wider zones of hemolysis.

The respiratory viruses can be diagnosed by rapid antigen detection tests, polymerase chain reaction (PCR) tests, and viral culture of the pharynx. EBV typically

causes a relative and absolute lymphocytosis with more than 10% atypical lymphocytes and thrombocytopenia. Heterophil antibody is present in 90% of affected adolescents and adults within the first 2 to 3 weeks of illness. False-negative results can be seen in patients younger than 5 years of age and require testing of IgM and IgG antibody to EBV viral capsid antigen (VCA IgM and VCA IgG) in order to establish the diagnosis of acute EBV infection; both are typically elevated in acute EBV infection. The presence of antibodies to Epstein-Barr nuclear antigen (EBNA) suggests prior rather than acute infection.

Acute retroviral syndrome typically presents with negative HIV antibodies and require tests for HIV type 1 RNA or p24 antigen to definitively make the diagnosis.

Radiologic Findings

Radiologic studies are generally not indicated for the diagnosis of pharyngitis, but can assist in establishing the diagnosis of some of the suppurative complications of GAβHS pharyngitis, such as PTA and retropharyngeal abscess (RPA), which are discussed later in this chapter.

Differential Diagnosis

Noninfectious causes of inflammatory tonsillopharyngitis include tonsillar cancer, which is rare in the pediatric population, systemic lupus erythematosus (SLE), Behçet's disease, bullous pemphigoid, and Kawasaki's syndrome. Periodic fever associated with aphthous stomatitis, pharyngitis, and cervical adenitis (PFAPA) typically occurs in patients younger than 5 years of age.

Treatment and Expected Outcome

Pharyngitis caused by viruses is treated symptomatically with warm saline gargles, rest, analgesics such as ibuprofen or acetaminophen, and plenty of liquids. Antibiotics are not indicated and these infections resolve without directed therapy.

A. haemolyticum is susceptible *in vitro* to erythromycin, clindamycin, tetracycline, and chloramphenicol. Susceptibility to penicillin is variable and treatment failures have been reported. Resistance to trimethoprim-sulfamethoxazole is common. The drug of choice is erythromycin, although no prospective clinical trials have been performed.

Gonococcal pharyngitis, if uncomplicated, can be treated with a single dose of intramuscular (IM) ceftriaxone (125 mg) or if the patient is 18 years of age or older, a single dose of an oral fluoroquinolone such as ciprofloxacin (500 mg) or ofloxacin (400 mg) in nonpregnant females. In addition, the patient should also be treated for possible chlamydia co-infection at a genital site with a single dose of azithromycin 20 mg/kg (maximum of 1 g) or if the patient is 8 years of age or older, doxycycline (100 mg) in nonpregnant females twice daily for 7 days.

GAβHS requires antimicrobial therapy in order to prevent the development of acute rheumatic fever (ARF) and to reduce the risk of suppurative complications such as PTA, retropharyngeal abscess, cervical adenitis, sinusitis, mastoiditis, otitis media, and bacteremia leading to metastatic infection. Antimicrobial therapy also shortens the duration of symptoms of GAβHS pharyngitis and reduces the period of contagiousness. However, antibiotic therapy does not affect the risk of poststreptococcal acute glomerulonephritis. If therapy is started before laboratory results are available, it should be discontinued if GAβHS is not identified. The recommended antibiotic is penicillin V. Patients allergic to penicillin can receive clindamycin, macrolides, or second generation cephalosporins (Table 11–3). Penicillin is effective in preventing ARF when therapy is started within 9 days after the onset of the acute illness. Oral treatment should be given for the full 10 days to prevent ARF. Some studies suggest that shorter courses result in microbiologic cure, although the impact of shorter courses of therapy on the risk of ARF is not known. Amoxicillin can be used in place of penicillin V. Macrolides such as clarithromycin for 10 days or azithromycin for 5 days also are effective. Erythromycin resistance is still uncommon in most areas in the United States. First generation oral cephalosporins for a 10-day course are an acceptable alternative. GAβHS is frequently resistant to tetracyclines and sulfonamides,

Table 11-3 Recommended Antimicrobial Therapy for GAβHS Pharyngitis

Drug	Dose	Duration
Penicillin V (oral)	400,000 U (250 mg) 2 to 3 times/day (children < 27 kg) 500 mg 2 to 3 times/day (heavier children, adolescents, and adults)	10 days
Penicillin G benzathine (IM)	600,000 U (children < 27 kg) 1.2 million U (heavier children, adolescents, and adults)	Once
Erythromycin estolate (oral)	20 to 40 mg/kg/day in 2 to 4 divided doses	10 days
Erythromycin succinate (oral)	40 mg/kg/day in 2 to 4 divided doses. Max dose, 1 g/day	10 days

and these agents should not be used as empirical therapy. Whereas sulfonamides do not eradicate GAβHS, they remain effective for continuous prophylaxis against recurrent rheumatic fever.

STOMATITIS

Definition

Stomatitis refers to inflammation of the stoma or mouth, anatomically delineated anteriorly by the lips and posteriorly by the anterior tonsillar pillars. The roof of the mouth consists of the hard and soft palate. The floor consists of mucosa overlying the sublingual and submandibular glands with the orifices of both of these glands opening into the anterior floor of the mouth. The lateral walls of the mouth are covered in buccal mucosa where one can find the orifice of the parotid glands opposite the upper second molars (Stenson's duct). Depending on the etiology of the stomatitis, some or all of these anatomic sites may be involved. The gingiva, which surround the dentition, are most often involved in stomatitis. Stomatitis may involve diffuse erythema and edema or discrete lesions of the papulovesicular or ulcerative types.

Epidemiology

The most common infectious agent to cause stomatitis is herpes simplex virus (HSV). Infection with HSV-1 (and less commonly HSV-2) usually results from direct contact with infected oral secretions or lesions. In the United States, more than 70% of persons have been infected with HSV by 12 years of age. Children 2 to 4 years of age are the most susceptible to HSV infections because of the lack of passively acquired maternal antibody and the practice of hand to mouth exploration. It is more common in children who attend daycare because there is likelihood of contact with oral secretions. The incubation period for HSV infection ranges from 2 days to 2 weeks.

Another common viral cause of stomatitis is enterovirus (coxsackie A and B, and echovirus). Enteroviral infections are spread by fecal-oral and respiratory routes. Enteroviruses may survive on environmental surfaces to allow for fomite transmission. In temperate climates infection is most common during the summer and early fall, although in tropical regions enterovirus is prevalent all year. Enterovirus mainly causes disease in children, especially those from lower socioeconomic backgrounds. In the United States, healthy children from the southern states are more frequently colonized (7% to 14%) than those in the north (0% to 2%). Spread to nonimmune individuals is high in situations where there is crowding or close contact such as in households, closed institutions, and summer camps. Viral shedding can occur without signs of clinical illness. Usually respiratory shedding lasts for a week or less, but fecal shedding can occur for several weeks after the onset of infection. The incubation period is 3 to 6 days.

Etiology

The most common cause of ulcerating vesicular stomatitis is HSV, typically HSV-1, but HSV-2 can also be a cause. Other viruses can cause stomatitis (Table 11–4). *Treponema palladium*, the infectious agent of syphilis, can present with a painless ulcer in the oral cavity.

Pathogenesis

The pathogenesis of stomatitis begins with viral entry into epithelial cells lining the oral cavity structures, followed by viral replication and tissue destruction. Epithelial cells full of virus rupture, allowing spread of the virus to neighboring cells. Healing involves crusting of vesicular lesions and reepithelialization. The herpetic vesicle is located intraepidermally and is characterized by the presence of ballooning degeneration, inflammation, and multinucleated giant cells. Although herpetic stomatitis is a self-limiting illness, the virus is transported to the trigeminal ganglia, where a latent or dormant infection is established and remains for life.

Clinical Presentation

History

Primary oral infection with HSV occurs most commonly in young children and is not difficult to recognize. The typical child is between 2 and 4 years of age. Although most primary infections of the oral cavity with HSV are asymptomatic, acute gingivostomatitis develops in some children. Reactivation of dormant virus can occur, often precipitated by factors such as stress, trauma, illness, or immune suppression. Virus then travels along the neuron back to the original site of infection. Recurrent infections are usually asymptomatic, but viral shedding does occur. When clinically evident, recurrent infections are rarely of the same magnitude as the primary infection and typically involve single labial lesions.

Table 11-4 Viral Etiology of Stomatitis

HSV-1, HSV-2
Coxsackie A, B
Echovirus
Varicella zoster
Epstein-Barr virus

HSV, herpes simplex virus.

Herpangina, caused by coxsackie A and B virus and echoviruses, typically produces discrete lesions in lower numbers and with less erythema and pain than HSV infections.

Erythematous mucositis induced by chemotherapy is usually seen within 3 to 5 days after initiation of chemotherapy with ulcerations developing after 7 days. Chemotherapy-induced mucositis tends to persist for 10 to 14 days after the chemotherapy is given. The most frequently involved sites are the tongue and the buccal and labial mucosa.

Symptoms

HSV usually produces an acute gingivostomatitis with ulcerating vesicles throughout the anterior portions of the mouth, including the lips. There is usually sparing of the posterior pharynx unlike the involvement seen in herpangina. High temperature is common and pain is intense, which leads to refusal by the patient to eat or drink. Typically the symptoms of herpangina are not as intense as HSV stomatitis. Symptoms of HSV gingivostomatitis typically range in duration from 5 to 14 days and in severity from mild to severe. The virus can be shed for weeks following symptom resolution. Herpangina symptoms usually resolve within 7 days.

Coxsackie A16 causes the majority of cases of hand-foot-mouth disease, in which painful vesicles and ulcers can occur throughout the oropharynx as well as on the palms and soles, perianally, and occasionally on the trunk or extremities. This infection usually lasts for 7 days and resolves spontaneously.

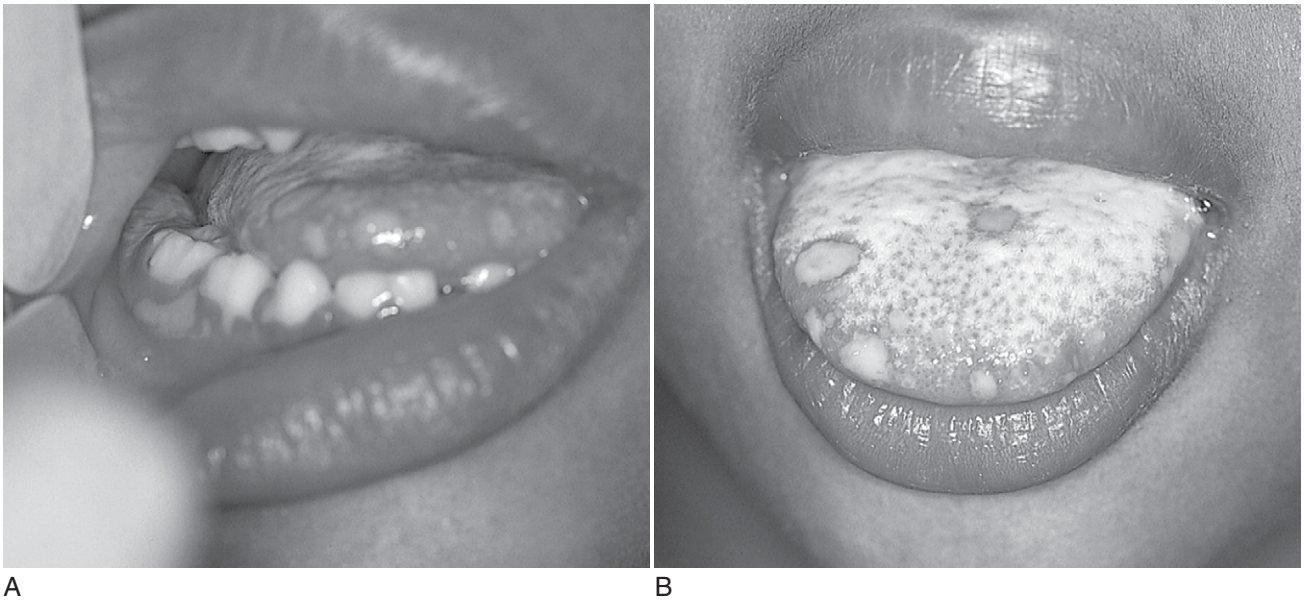
Physical Findings

Physical findings consistent with HSV gingivostomatitis include high temperature, irritability, and gingiva that are painful, erythematous, and inflamed and that tend to bleed easily. Ulcerations with erythematous halos can be seen on the buccal and labial mucosa, gingival, tongue, tonsillar pillars, and hard palate (Figure 11–2). Often halitosis is present secondary to ketosis and tissue destruction in the oral cavity. Patients usually have bilateral anterior cervical lymphadenopathy. Submental, submaxillary, and tonsillar adenopathy can also be present. Clinical signs of dehydration often are present.

Herpangina is characterized by fever and distinct, painful, gray-white papulovesicular lesions surrounded by a halo of erythema in the posterior oropharynx (Figure 11–3). These lesions ulcerate.

Laboratory Findings

A complete blood count may reveal leukocytosis with a predominance of lymphocytes. Diagnostic tests to identify the etiologic agent include viral culture, direct antigen (HSV-1 or HSV-2) detection and PCR. HSV viruses grow rapidly (usually less than 48 hours) in tissue culture. Isolating enterovirus from any specimen except feces usually can be considered causally related to a patient's illness. PCR is generally considered the gold standard for diagnosis of enterovirus and is approved for use on cerebrospinal fluid, nasopharyngeal or throat swabs, stool or rectal swab, and frozen tissue.



A B

Figure 11-2 Herpes simplex virus gingivostomatitis characterized by discrete mucosal ulcerations and diffuse gingival erythema and edema ***(A)*** and by numerous yellow ulcerations with thin-walled erythematous halos on patient's tongue ***(B)***. (From Zitelli B, Davis H, eds: The Atlas of Pediatric Physical Diagnosis, 4th ed. Philadelphia: Elsevier, 2002.)

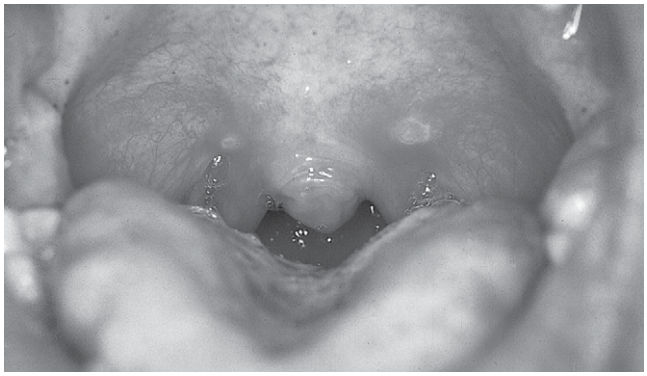


Figure 11-3 Herpangina characterized by painful, shallow, yellow ulcers surrounded by erythematous halos on uvula and anterior tonsillar pillars. (From Zitelli B, Davis H, eds: The Atlas of Pediatric Physical Diagnosis, 4th ed. Philadelphia: Elsevier, 2002.)

Radiologic Findings

Radiologic testing is not necessary to establish a diagnosis.

Differential Diagnosis

Noninfectious causes of stomatitis are listed in Table 11–5. The diagnostic criteria for PFAPA requires the presence of regularly recurring fevers with early age of onset (younger than 5 years of age), the absence of neutropenia, and at least one of the following clinical signs: aphthous stomatitis or cervical lymphadenitis and pharyngitis, followed by completely asymptomatic intervals between episodes and normal growth and development. Behçet's syndrome manifests with aphthous ulcers of various sizes (from 1 to 3 cm) in the oral cavity associated with genital ulcers, iridocyclitis, and synovitis. Patients can also have erythema nodosum, thrombophlebitis, and meningoencephalitis. The fever usually lasts more than 1 week, but Behçet's syndrome does not show the periodicity of PFAPA. Chemotherapeutic agents that are directly toxic to the mucosa can cause decreased proliferation of the basal epithelial cells, which results in thinning of the surface epithelium (erythematous mucositis). This can progress to focal or generalized mucosal degeneration (ulcerative mucositis).

Table 11-5 Noninfectious Causes of Stomatitis

PFAPA
Cyclic neutropenia
Agranulocytosis
Stevens Johnson syndrome
Radiation induced
Drug induced (chemotherapy, antibiotics)
Histiocytosis X
Inflammatory bowel disease
Behçet's syndrome

PFAPA, periodic fever associated with aphthous stomatitis, pharyngitis, and cervical adenitis.

Treatment and Expected Outcome

HSV stomatitis and herpangina are self-limited illnesses. Most of the therapy for stomatitis is supportive and involves pain management and fluid resuscitation. Occasionally severely ill patients will present with dehydration and require intravenous hydration. Nonacidic fluids (e.g., apple juice, liquid gelatin), lukewarm broth, and cold, soft solids such as yogurt, pudding, popsicles, and Jell-O are recommended. Certain products should be avoided such as mouthwashes that contain alcohol, phenol, aromatics, and other irritating chemicals. Preparations that contain petrolatum or glycerin should be avoided because they may result in desiccation of tissues. Spicy and acidic foods as well as foods with a hard consistency should also be avoided. Acetaminophen is useful for pain and fever reduction. Topical anesthetics such as viscous lidocaine are generally not recommended. Young patients with HSV stomatitis who do not have control of their secretions require exclusion from daycare. Hospitalized patients with severe HSV stomatitis require contact precautions. There are limited data concerning the use of acyclovir in mucocutaneous HSV infections in immunocompetent hosts. Small studies have shown some therapeutic benefit of oral acyclovir in primary gingivostomatitis. Likewise, studies in adults with recurrent HSV labialis have shown minimal therapeutic benefit from oral acyclovir. Topical acyclovir is not effective.

PERITONSILLAR ABSCESS

Definition

Peritonsillar abscess ("quinsy" meaning "dog strangling") is defined as a collection of pus located between the tonsillar capsule (the pharyngobasilar fascia), the superior constrictor muscle, and the palatopharyngeus muscle.

Epidemiology

PTA is the most common deep space head and neck infection. It usually occurs in older school-age children, adolescents, and young adults as a complication of recurrent bacterial tonsillitis or a secondary bacterial infection following viral pharyngitis.

Etiology

PTA is thought to arise from contiguous spread of infection from the tonsil or the mucous glands of Weber located in the superior tonsillar pole. Cultures of the

purulent material usually grow several bacteria (average number of isolates is 5); GAβHS (33% to 50% of patients), and *Staphylococcus aureus* (15% to 25% of patients) are the most common isolates (Table 11–6). The majority of organisms isolated from PTA are β-lactamase producers. There have been case reports of *A. haemolyticum* as a cause of PTA. *H. influenzae* type B (Hib) has virtually been eliminated as a cause of PTA as a result of universal Hib vaccination. In patients with EBV infection, the bacteria obtained from PTA are similar to those found in patients without EBV. Rarely, *Mycobacterium tuberculosis* or atypical mycobacteria as well as fungal species can cause PTA.

Pathogenesis

The exact pathogenesis of PTA is not known, but usually involves infection that begins as acute tonsillitis that progresses to peritonsillitis and ends with formation of an abscess. Purulent material collects between the fibrous capsule of the tonsil, usually at the upper pole and the superior constrictor muscle of the pharynx. Another possible mechanism involves the Weber glands, which are salivary glands located above the tonsillar area in the soft palate that clear the tonsillar area of debris. If these ducts become obstructed because of tissue necrosis and inflammation, an abscess develops in the peritonsillar area. Bacterial superinfection in EBV may occur as a consequence of the substantial edema and inflammation in the potential space between the superior constrictor muscle and the tonsillar capsule that facilitates secondary bacterial invasion.

Clinical Presentation

History

Most patients present for evaluation after less than 1 week of symptoms. A past history of pharyngitis or tonsillitis occurs in approximately half of patients. Often patients have already received antibiotics for pharyngitis before presenting with a tonsillar abscess. Occasionally PTA can present as a fever of unknown origin with only mild sore throat and no obvious signs of pharyngeal inflammation.

Table 11-6 Bacterial Causes of Peritonsillar Abscess

Streptococcus pyogenes (GAβHS)
Staphylococcus aureus
Streptococcus agalactiae (group B streptococcus)
Haemophilus influenzae
Fusobacterium necrophorum
Bacteroides species
Porphyromonas species
Peptostreptococcus species
Prevotella melaninogenica
Mycobacterium

GAβHS, group A β-hemolytic streptococcus.

Symptoms

The most common presenting symptoms include sore throat or neck pain, odynophagia or dysphagia, fever, and decreased oral intake.

Physical Findings

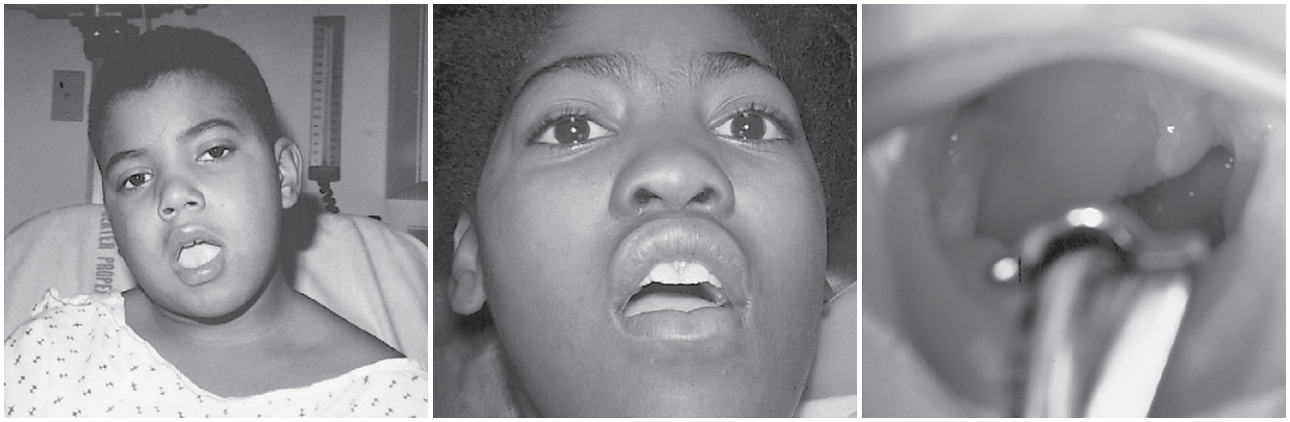
Physical examination typically reveals cervical adenopathy, uvular deviation (in half of patients), muffled voice ("hot potato" voice), and trismus. The "hot potato" voice results from palatal edema and spasm of the internal pterygoid muscle that elevates the palate. Trismus occurs in two thirds of children with significant peritonsillar infection and is associated with impairment of palatal movement as a result of edema (Figure 11–4). Patients often present with drooling secondary to odynophagia. A detailed examination of the throat may be difficult to perform, especially in young children. More commonly the abscess is unilateral; bilateral disease is an unusual variant and is more difficult to diagnose secondary to the lack of asymmetry. If the abscess is large, the soft palate and uvula usually are deviated from the affected side and show signs of inflammation (see Figure 11–4). Ipsilateral, tender anterior cervical lymphadenopathy is typically present. Clinical signs of dehydration may be present.

Laboratory Findings

Typically patients have an elevated white blood cell count with a left shift and elevated acute phase reactants. Aerobic and anaerobic cultures of tonsillar aspirates should be obtained. If a mycobacterial species is suspected, then acid-fast stains and mycobacterial cultures should be performed. In addition, if a fungal pathogen is suspected, fungal stains and fungal cultures should be obtained. Blood cultures are usually obtained but are often negative.

Radiologic Findings

Computed tomography (CT) scan of the neck with contrast is recommended due to the physical limitations of examining a small oropharynx and the presence of trismus complicating the ability to obtain a thorough oropharyngeal examination. Findings suggestive of abscess include an area of low attenuation, ring enhancement, and edema of the surrounding soft tissue. Findings suggestive of a phlegmon or cellulitis are tissue edema with the lack of ring enhancement.



A B C

Figure 11-4 Peritonsillar abscess demonstrating torticollis ***(A)***, trismus ***(B)***, and inflamed soft palatal mass that obscures the tonsil and bulges forward and toward midline, deviating the uvula ***(C)***. (From Zitelli B, Davis H, eds: The Atlas of Pediatric Physical Diagnosis, 4th ed. Philadelphia: Elsevier, 2002.)

Differential Diagnosis

Squamous cell carcinoma of the tonsil should be in the differential in adults, but this has not been reported in children. Rarely, a PTA can occur as a consequence of tonsillar carcinoma. Tonsillar lymphoma should be in the differential in a child with unilateral tonsillar enlargement.

Treatment and Expected Outcome

PTA requires prompt medical evaluation due to the risk of potential spread through the muscle into the parapharyngeal or deep neck spaces. Enlargement of the tonsils can lead to airway obstruction, and rupture of the abscess can cause aspiration of infected material and resultant pneumonia. Once an abscess is identified by CT scan, incision and drainage is required and is preferred over a needle aspiration to ensure adequate drainage and to obtain material for culture. Needle aspiration can be performed only if the abscess is located within the superior pole in a cooperative patient. Antibiotic therapy alone is insufficient. More frequently children require management in the operating room, and they undergo tonsillectomy more frequently than do adults. It is recommended that children with a history of recurrent tonsillitis undergo immediate tonsillectomy when they present with a tonsillar abscess. Routine performance of a tonsillectomy in patients without recurrent histories along with or after medical management is still controversial. Postoperatively, most patients require intravenous hydration, antibiotics, airway monitoring, and pain management. Initial antibiotic choice should provide coverage for GAβHS including exotoxin-producing strains, *S. aureus* including methicillin-resistant *S. aureus*, and β-lactamase–producing anaerobes. Recommended initial therapy may include clindamycin, second generation cephalosporins with good anaerobic coverage (e.g., cefoxitin), or ampicillin-sulbactam. Definitive antibiotic therapy should be guided by culture results. For infections with *A. haemolyticum,* antimicrobial susceptibility testing should be performed to guide the choice of antibiotics. When patients are able, antibiotics can be changed to oral preparations to complete the therapy, which is usually for at least 10 days.

In patients in whom imaging fails to demonstrate a true abscess (i.e., a phlegmon), the patient is usually hospitalized to receive intravenous antibiotics. Typically, either a true abscess develops or the case resolves within 1 to 2 days. A CT scan should be repeated in 2 to 3 days to document whether the inflammation has improved or an abscess has developed.

RETROPHARYNGEAL ABSCESS

Definition

Retropharyngeal abscess is a collection of purulent material located in the deep tissues of the neck. It is typically considered a medical emergency secondary to the possible complications of airway compromise, invasion of contiguous structures, and sepsis. Retropharyngeal cellulitis or phlegmon is a condition that precedes an organized abscess. The retropharyngeal space extends longitudinally downward from the base of the skull to the posterior mediastinum. Its posterior border is the prevertebral fascia and its anterior border is the pretracheal fascia. The carotid sheaths form the lateral border. Lemierre's syndrome, a complication of RPA, is associated

with septic thrombophlebitis of the tonsillar vein and internal jugular vein with resultant metastatic abscesses to distant sites such as lung, joints, and bones.

Epidemiology

Nontraumatic RPA is more common in young children, with the vast majority of cases occurring in patients younger than 6 years of age. Since the cervical lymphatic system in the space between the posterior pharyngeal wall and the prevertebral fascia atrophies with age, it is more likely that younger children will present with an RPA of medical origin. Adolescents and adults are more likely to have an RPA of traumatic origin (regional trauma, foreign body ingestion, trauma from procedures), and Lemierre's syndrome is typically seen in adolescents and young adults.

Etiology

RPA and cellulitis are typically caused by aerobic organisms alone (GAβHS and *S. aureus* predominate) or in combination with anaerobes. The majority of isolates are β-lactamase producers. The microorganisms that cause RPA are similar to those causing PTA (Table 11–6). *Fusobacterium necrophorum*, an anaerobic gram-negative rod, is associated with Lemierre's syndrome.

Pathogenesis

RPA occurs as a consequence of infections o f the nasopharynx, paranasal sinuses, or middle ear. The infection is thought to extend to lymph nodes between the posterior pharyngeal wall and prevertebral fascia. Lemierre's syndrome has been shown to follow some cases of acute EBV infection; therefore it has been hypothesized that a primary viral throat infection plays a role in the pathogenesis of RPA. In addition, nicotine from cigarette smoke has been shown to potentiate the toxins made by some oral anaerobes and perhaps to increase the risk of developing infection with these organisms.

Clinical Presentation

History

Children usually present with fever, irritability, and refusal to eat. Respiratory complaints may not be present. Often patients will have a history of a preceding viral upper respiratory infection. As with a PTA, patients have often received antibiotics recently for presumptive treatment of GAβHS pharyngitis.

Symptoms

The clinical presentation can be subtle and variable. Most typically, children present with fever, sore throat, and neck swelling. Pain with neck extension occurs in some patients. Drooling and respiratory distress are uncommon manifestations.

Physical Findings

Unilateral posterior pharyngeal bulging, limitation of neck extension and flexion, and torticollis are often present (Figure 11–5). Patients usually prefer to keep their

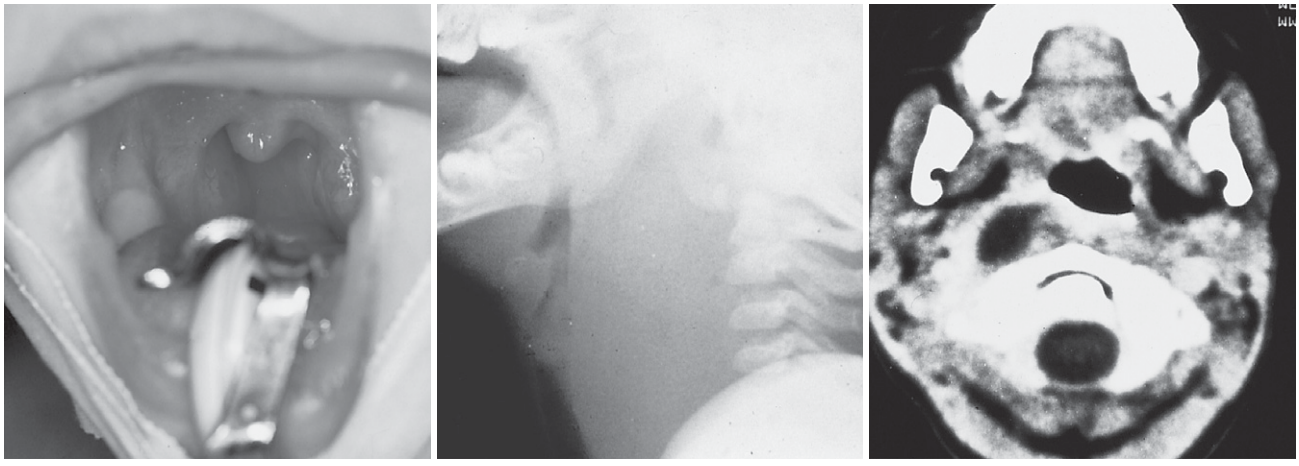


Figure 11-5 Retropharyngeal abscess. ***A,*** Intense erythema and swelling of the posterior pharyngeal wall. ***B,*** Lateral neck film showing prominent prevertebral soft tissue swelling that displaces the trachea forward. ***C,*** Computed tomography scan with contrast revealing a thick-walled abscess cavity in the retropharyngeal space. (From Zitelli B, Davis H, eds: The Atlas of Pediatric Physical Diagnosis, 4th ed. Philadelphia: Elsevier, 2002.)

neck neutral and complain of pain with neck extension more commonly than with flexion. These clinical signs are often mistaken for meningeal irritation; however, patients with RPA usually have less lethargy than patients with meningitis. Most patients present with neck swelling and fever. Patients can present with drooling and signs of respiratory distress, but these findings do not have to be present.

Laboratory Findings

Patients may have elevated white blood cell counts with a left shift as well as elevated acute phase reactants. Blood cultures are usually obtained but are often negative. Aerobic and anaerobic cultures with antimicrobial susceptibility should be obtained at the time of surgical drainage and transported promptly in the proper media to sustain their growth. Intraoperative cultures usually grow mixed flora including GAβHS, *S. aureus, H. influenzae*, gram-negative bacilli, and anaerobes. The predominant anaerobic species isolated are *Bacteroides*, *Porphyromonas*, *Prevotella*, *Peptostreptococcus,* and *Fusobacterium*. Patients with Lemierre's syndrome often have elevated liver transaminases and bilirubin levels.

Radiologic Findings

Plain radiographs show widened prevertebral soft tissues on lateral view of the neck. The presence of gas or air-fluid levels within the retropharyngeal space and loss of the normal cervical lordosis are also important clues that can be seen on plain films (see Figure 11–5). CT scan with contrast is the preferred imaging modality; findings are abnormal in the majority of patients. CT is indicated especially when an abscess is suspected to extend into deep neck tissues in order to best delineate the affected anatomy (see Figure 11–5). CT can differentiate a true abscess from a cellulitis or phlegmon, which is characterized radiographically as an edematous area without ring enhancement. If Lemierre's syndrome is suspected, Doppler ultrasound can demonstrate thrombosis of the internal jugular vein. Ultrasound can miss a fresh thrombus with low echogenicity and does not provide a good image beneath the clavicle and mandible. Magnetic resonance imaging has been used successfully to identify thrombus when ultrasound is not diagnostic. Septic emboli to the lung produce the characteristic radiographic appearance of multiple peripheral round and wedge-shaped opacities that rapidly progress to cavitation. Some patients with Lemierre's syndrome can have nonspecific, patchy consolidation suggestive of bronchopneumonia. CT scan of the chest with contrast can reveal septic infarcts and peripheral lesions, which enhance.

Differential Diagnosis

The differential diagnosis includes the causes of pharyngitis, acute EBV infection, a noninfectious mass in the retropharyngeal space, meningitis secondary to the common presenting sign of neck stiffness, and epiglottitis secondary to the signs of drooling and respiratory distress. The differential diagnosis of Lemierre's syndrome includes leptospirosis, acute bacterial pneumonia, especially staphylococcal secondary to cavitation, aspiration pneumonia, atypical pneumonia, endocarditis with septic embolization, and intra-abdominal infection.

Treatment and Expected Outcome

RPA can spontaneously rupture and result in aspiration. Therefore repeated throat examinations with forceful use of tongue depressors should be discouraged. In addition, contiguous spread to the posterior mediastinum and parapharyngeal space can occur. Spread to the prevertebral space with risk of development of brain abscess and meningitis can occur. Lemierre's syndrome can result in septic emboli to the lung and resultant pneumonia. Sepsis can also complicate an RPA.

The traditional and preferred management has been surgical drainage of the abscess with an intraoral incision. Surgery usually takes place on the first or second hospitalization day. Some cases in the literature have successfully been treated using antibiotics alone if treated during an early stage of infection. However, once a true abscess has formed, surgical drainage in conjunction with antibiotic therapy is recommended. Initial antibiotic therapy should include coverage for both aerobes and anaerobes as well as be stable against β-lactamases. Initial antibiotic choices for hospitalized patients include ampicillin-sulbactam, second generation cephalosporins with anaerobic activity (e.g., cefoxitin), or a third generation cephalosporin plus clindamycin. Definitive antibiotic therapy should be determined based on culture and susceptibility results.

Therapy of Lemierre's syndrome is usually done in consultation with critical care and infectious diseases personnel. Retrograde propagation of internal jugular vein thrombosis to involve the cranial sinuses including the cavernous or sigmoid sinuses has been documented. The role for anticoagulation is controversial because the outcome of most patients is good without it, and no controlled studies have been done to assess the value of heparin therapy in thrombophlebitis of the internal jugular vein.

MAJOR POINTS

- Viral pharyngitis is usually accompanied by cough and coryza.
- GAβHS pharyngitis requires antimicrobial therapy in order to prevent the development of acute rheumatic fever and to reduce the risk of the suppurative complications.
- Herpes simplex virus stomatitis and herpangina are self-limited illnesses.
- Herpangina involves the posterior pharynx, whereas HSV usually involves the anterior portions of the mouth.
- The microorganisms that cause peritonsillar abscess (PTA) and retropharyngeal abscess (RPA) are similar.
- PTA usually occurs in older children, adolescents, and young adults, whereas RPA typically occurs in younger children; both conditions require drainage and antibiotics.

SUGGESTED READINGS

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