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Emergency Medicine in Review

ADULT AND PEDIATRIC PHARYNGITIS: A REVIEW

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□ **Abstract**—Acute pharyngitis is frequently encountered in the ambulatory care setting. Although usually of viral etiology, streptococcal disease is the focus of diagnostic efforts, in light of significant suppurative and nonsuppurative sequelae. The traditional symptoms of fever, adenopathy, and pharyngeal exudate are suggestive, but not diagnostic of streptococcal pharyngitis. Thus, the importance of diagnostic testing, including Group A beta hemolytic strep antigen screen and culture, is emphasized. Recent innovations in therapy include modification of antibiotic dosing regimens and use of cephalosporins to improve patient compliance.

□ **Keywords**—pharyngitis; neck abscess; streptococcus

INTRODUCTION

Pharyngitis is an inflammatory syndrome of the oropharynx attributed predominantly to infectious causes or, less commonly, to secondary involvement in systemic or noninfectious illness. This complaint is frequently encountered and is responsible for an estimated 40 million outpatient visits (1). The incidence of pharyngitis is significant when analysis of emergency department visits reveals this to be the third to seventh most common complaint (2,3). The importance of accurate, initial diagnosis and treatment is illustrated in a recent study where 80% of patients discharged with pharyngitis were noncompliant with their referral recommendations for follow-up evaluation (3).

The oropharynx consists of mucous membrane

layers composed of stratified squamous epithelium, and is the initiation point of the inflammatory process (4). Second, the muscle group consists of an external layer formed by the superior, middle, and inferior constrictors; and an internal layer composed of the stylo-, salpingo-, and palatopharyngeus (4). Third, the cervical fascia encases the fascial muscles and platysma, sternocleidomastoid, parotid and submandibular glands, esophagus, thyroid and strap muscles, and the prevertebral space (5). The lymphatic system, designated Waldeyer's ring, consists of the palatine tonsils located between the palatoglossal and palatopharyngeal pillars, nasopharyngeal and lingual tonsils (4,5). These structures drain via the jugulodigastric node and the anterior cervical chain, which is contiguous with the mediastinum (4,5). Thus, pharyngitis begins as inflammation of the mucous membranes with secondary involvement of the lymph node drainage system, rarely progressing to deep neck and mediastinal involvement.

PATHOGENESIS

The causes of pharyngitis are numerous, with a viral etiology most frequently described (Table 1). However, the prototype syndrome due to Group A Beta Hemolytic Streptococcus (GABHS) infection occurs in the range of 5% to 20% of cases (6,7). This morphologic classification of streptococcal species incorporates the hemolytic reaction where beta designates central clearing on a blood agar plate (7). Further

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Table 1. Etiology of Pharyngitis

I. Infectious Syndromes	
A. Bacterial	
1.	<i>Streptococcus pyogenes</i>
2.	<i>Chlamydia trachomatis</i>
3.	<i>Mycoplasma pneumoniae</i>
4.	<i>Haemophilus influenzae</i>
5.	<i>Treponema pallidum</i> (syphilis)
6.	<i>Neisseriae gonorrhoeae</i>
7.	<i>Mycobacterium tuberculosis</i>
8.	<i>Borrelia vincenti</i> (Vincent's angina)
9.	<i>Corynebacterium diphtheria</i>
10.	<i>Corynebacterium hemolyticum</i>
B. Fungal	
1.	<i>Candida albicans</i> (moniliasis)
C. Viral	
1.	Rhinovirus
2.	Coronavirus
3.	Adenovirus (pharyngoconjunctival fever)
4.	Coxsackie A (herpangina)
5.	Herpes simplex virus
6.	Aphthous stomatitis
7.	Parainfluenza
8.	Influenza
II. Systemic (Noninfectious) Syndromes	
A. Pemphigus vulgaris	
B. Erythema multiforme	
C. Neutropenia	
D. Allergic	

classification proceeds by examination of the cell wall containing the carbohydrate antigen, M protein, and mucopeptide components responsible for pathogenicity. Therefore, Group A or *Streptococcus pyogenes* species are responsible for most human disease (8,9). The pathogenesis of streptococcal pharyngitis involves airborne, direct contact, and, rarely, cases of food borne transmission (10,11). Virulence is dependent on the cell wall antigens, locally invasive cellular enzymes, such as streptokinase or hyaluronidase, and systemic effects of circulating proteins such as erythrogenic toxin found in scarlet fever (7,9).

EPIDEMIOLOGY

The epidemiology of streptococcal pharyngitis finds this to be a disease of youth, with 50% of the patients in the 5- to 15-year age group, and GABHS is the most common respiratory pathogen encountered in patients over 3 years of age (11-13). However, recent evaluation of infants found a 25% incidence of GABHS, previously felt to be almost nonexistent in the 0- to 3-year age group (10,12). Demographics also reveal a bimodal seasonal variation with a winter-spring (January to May) peak and a noncontiguous increase in cases identified in September, coincident with school exposure (12). However, this seasonal variation has not been described in all case series

(14). A prospective study of symptomatic adult patients found 9.7% with culture-proven GABHS pharyngitis, with a range of 2% to 5.3% for specific symptom complexes (15). Recently, the importance of mycoplasma, chlamydia, and corynebacterium as etiologic agents implicated in the development of adult pharyngitis has been noted, as opposed to streptococcal species (16).

DIAGNOSIS

The streptococcal pharyngitis syndrome complex includes malaise, anorexia,odynophagia, abdominal pain, headache, and moderate fever with a range of 39° to 40°C for adults and 40° to 40.5°C for pediatric cases (4,17). Fever is suggested to be the most commonly occurring symptom in a study of 448 children with GABHS in whom 90% had a temperature elevation of 37.3°C and 74% to 38.5°C, albeit a somewhat lower temperature threshold (13). Associated physical findings include first a nonadherent pharyngeal exudate in 45% of cases, a sensitive but not specific criterion as it can be found in a multitude of bacterial and viral conditions (11). Secondly, other associated oral findings include erythema, petechiae, and "doughnut lesions," a raised hemorrhagic area with a yellow exudative center in 10% (11). Thirdly, lymphoid hypertrophy in the form of tender (49%) and painless (36%) anterior cervical adenopathy is associated with streptococcal disease (18). Finally, the presentation of upper respiratory infection with cough and rhinorrhea has been suggested to indicate a nonstreptococcal etiology in adult patients (19). However, in pediatric patients with pharyngitis, 36% of those with cough and 45% with coryza symptoms had throat cultures positive for GABHS (11).

The diagnosis of streptococcal pharyngitis begins with recognition of characteristic symptoms and signs followed by corroborative laboratory evidence of infection. These clinical criteria of infection have been formalized into qualitative scoring systems predicting the likelihood of disease based on culture result. Breese utilized the season of occurrence (spring), age (5 to 10 years), and leukocyte count (20,400) as a predictive index for empiric therapy in pediatric patients (20). Walsh and colleagues correlated culture results with risk stratification into high and moderate profile groups based on findings of fever, cervical adenopathy, streptococcal exposure, pharyngeal exudate, and absence of cough (19). Centor and colleagues suggested empiric penicillin therapy based on the presence of two or more of these risk factors (21). These empiric therapy scales were

corroborated by Komaroff and colleagues, who found that if 3 of 3 clinical markers, including fever, tender adenopathy, and exudate, were present, therapy was suggested, as subsequent cultures revealed a 30% to 45% incidence of GABHS (15). However, an analysis of physicians' clinical estimates of disease likelihood found that only 10% of patients treated empirically were subsequently diagnosed with culture-proven streptococcus (22). Thus, even if 100% of clinical markers associated with pharyngitis are present, streptococcal disease occurs in fewer than 50% of patients encountered (23). These diagnostic scales based on clinical signs and symptoms must be interpreted in light of pretest disease probability. This is best approximated by combining disease prevalence with clinical markers. Thus, high disease prevalence or epidemic conditions would warrant empiric therapy, while lower prevalence endemic conditions rely on culture results for decision making (21).

Laboratory evaluation correlates leukocytosis or systemic white blood cell count greater than 8,500 with streptococcal infection (20). A Gram's stain analysis of pharyngeal exudate has demonstrated a 73% sensitivity and 96% specificity in the diagnosis of GABHS (22). A relatively new diagnostic modality is the rapid strep screen in which streptococcal antigen extracted from a throat swab is exposed to latex agglutination or an ELISA (enzyme-linked immunosorbent assay) reaction (21). Pilot clinical trials demonstrated an 83% sensitivity with some variability (80% to 95%) and a reproducible 99% specificity for the slide test (18,24). Subsequent studies revealed a 73% sensitivity with a minimal false positive rate, but an unacceptable false negative rate (7). Thus, Strandjord concluded that if the rapid strep screen was utilized, the ELISA test with a positive predictive value (PPV) of 88% was preferable to the latex agglutination test with a PPV of 58% (25). This significance is illustrated by the negative predictive value or absence of disease of 93% where culture positive GABHS is not detected by the strep screen, a significant error of omission. Thus, recommendations concerning the rapid strep screen include a double-swab specimen with a positive result warranting therapy and a negative screen requiring subsequent culture.

The diagnostic standard for streptococcal infection is the routine throat culture—specimens plated on a blood agar medium with bacitracin discs to which Group A beta hemolytic strep are susceptible (8). Proper technique includes sampling of the tonsils and peritonsillar pillars, as cultures of saliva and buccal mucosa often yield a negative result (26). Throat culture is 90% to 95% accurate, that is, a 5% to 10% false negative rate, when compared in serial speci-

mens; so there is a minimal but defined need to reculture a negative result assuming proper technique for untreated patients with repeat emergency department visits (22).

However, examination of outcome contingencies suggests that a negative culture does not exclude infection (2). Kaplan and colleagues, in a study of double-swab throat cultures, found a 10% discordance in simultaneous specimens (27). An early study of tonsillectomy patients found a difference of 16.2% between preoperative culture and histopathologic analysis postoperatively (28). It has been suggested that partially treated cases are also contributory to the false negative rate (29). Therefore, patients with persistent pharyngitis may warrant a second culture in untreated cases acknowledging a low yield. Those refractory to antibiotic therapy suggest a resistant bacteriologic or viral etiology, which may be demonstrated by serologic testing, that is, Monospot, ASO titer.

Similarly, a positive culture is not always correlated with clinical infection (2). A study of patients for symptomatic pharyngitis demonstrated that only 43% exhibit a specific antibody response indicative of infection as opposed to colonization (30). This carrier state can exist in 5% to 25% of patients with no established risk of transmission, symptomatic pharyngitis, or sequelae (30). However, it must be emphasized that this distinction may not be readily apparent in a limited patient visit. Therefore, a therapy for culture-proven GABHS avoids costly errors of omission, acknowledging a minor increase of overtreatment.

Another diagnostic dilemma involves non-Group-A beta hemolytic strep including Groups B, C, and G, streptococci previously felt to be normal flora (7,9). Serologic analyses for streptococcal antibodies and cellular products, including antistreptolysin O, streptonase B, and streptozyme, are sensitive and specific for retrospective diagnosis of disease or a carrier state (27). Recent evidence suggests that non-Group-A beta hemolytic strep can, in fact, cause symptomatic pharyngitis indicated by an increased ASO titer (26,31). However, the suppurative sequelae are minimal, and there are no nonsuppurative sequelae described (2). Thus, treatment may be warranted, as a favorable therapeutic response has been demonstrated.

Lastly, the identification of subclinical infection estimated to be found in 50% of all pharyngitis patients must be addressed (18). These patients may be minimally symptomatic yet be at risk for nonsuppurative complications. This patient group should be identified and treated in high prevalence states or in

family contacts of those affected, where a 25% rate of secondary infection has been described (18).

DIFFERENTIAL DIAGNOSIS

Chlamydia is another common cause of bacterial pharyngitis. *Chlamydia trachomatis* affects 2% of symptomatic adolescents, as well as adults who are predisposed by orogenital sexual activity (17). Komaroff reports an increasing incidence of both chlamydia and mycoplasma pharyngitis as determined by serological analysis (32). A recent report has suggested that arcanobacterium (corynebacterium) hemolyticum has superseded these two pathogens to become the most common cause of adult pharyngitis (16). Chlamydia has been identified in up to 20.5% of patients with pharyngitis accompanied by respiratory symptoms (32). Symptomatic patients may be treated with erythromycin or tetracycline (33). Mycoplasma pneumonia is another emerging new pathogen, with an incidence of 2% to 3% in adolescents and 10.6% in adults (32). Mycoplasma is also associated with lower respiratory tract infections in 11% of adults and 32% of children (34). Headache is a prominent symptom, and diagnosis is suggested by elevation of serum cold agglutinin titers, although nonspecific in pediatric patients less than 3 years of age (35). Erythromycin and tetracycline are similarly used for therapy (33). *Hemophilus influenza* affects pediatric patients predominantly with a syndrome that includes pharyngitis, otitis media, laryngotracheitis, or epiglottitis (17). Diagnosis is facilitated by culture analysis; therapy includes amoxicillin, noting a resistance rate of 10% to 30% for hemophilus type B strains (10).

Sexually transmitted disease may manifest as pharyngitis in selected patients. Syphilis due to *Treponema pallidum* can present as locally invasive pharyngitis in primary, secondary, or tertiary stages of disease (36). Symptoms begin with a classic chancre followed by adenopathy or mucosal erosions and finally a painless, gummatous lesion (17). Diagnosis is confirmed by dark field microscopy of the lesion or systemic serological analysis for RPR, VDRL, or FTA antibody. Therapy includes benzathine penicillin G (4,800,000 units) and, in penicillin allergic patients, tetracycline or erythromycin (500 mg qid) may be used for 15 days (33). Gonorrhea can present as locally invasive pharyngitis due to orogenital contact or secondary to disseminated disease (37). A study of adults with a history of orogenital contact found the incidence of gonococcal pharyngitis to be 5% to 25% in male homosexuals, 10% to 20% in heterosex-

ual women, and 3% in heterosexual males (37). Most patients infected with neisseriae gonorrhoea are asymptomatic (50%), although odynophagia, low-grade fever, and erythema without exudate occur (37). Therapy includes amoxicillin (3.0 g) along with probenecid (0.5 to 1.0 mg) and spectinomycin (2 mg), ceftriaxone (250 mg), or ciprofloxacin (500 mg) for resistant strains (33).

Tuberculosis may present as secondary pharyngitis after hematogenous dissemination of mycobacteria from the primary focus of infection (4). Patients usually have an advanced disease course, including active pulmonary cavitary or miliary tuberculosis (17). Symptoms include hoarseness and dysphagia secondary to pharyngeal ulceration. Diagnosis is suggested by culture, and therapy includes isoniazid, ethambutol, rifampin, or streptomycin (33).

Membranous pharyngitis with a gangrenous exudative appearance may be associated with Vincent's angina or diphtheria. Vincent's angina is an oropharyngeal ulcerative condition caused by fusobacterium necrophorum, borrelia vincenti (Vincent's fusiform bacillus), spirocheta denticolonica or peptococcus (17). This synergistic infection involves proliferation of indigenous flora, oral spirochetes, and anaerobes in the elderly or those with poor oral hygiene, which was first noted in World War II troops (trench mouth). Symptoms include odynophagia and fetid breath associated with gingival-buccal ulceration, membranous exudate, and submandibular lymphadenopathy (38). Diagnosis is made by clinical appearance or Gram's stain of the necrotic lesion (38). Therapy involves the use of penicillin, tetracycline, and oral oxidizing agents such as peroxide (12,33).

Another rare cause of necrotizing pharyngitis is corynebacterium diphtheriae. Diphtheria causes pharyngitis due to local invasion, and hematogenous spread of exotoxin can result in myocarditis (66%), cranial nerve paralysis (10%), hepatitis, or peripheral neuropathy (2,11). Physical signs include the presence of a grey adherent membrane, as opposed to the friable membrane associated with streptococcal disease, and "bull neck" anterior cervical adenopathy that may progress to complete airway obstruction (4). Diagnosis is facilitated by the Gram's stain presence of "chinese letter" bacterial forms, the Schick antibody test, or Loeffler medium culture (10). Therapy includes penicillin (25 to 50 mg/kg/day) or erythromycin (50 mg/kg/day) although antibiotics are not effective for circulating toxin (17,33). Antitoxin is most effective when administered within 3 days of onset of symptoms in a dose range of 20,000 to 100,000 units per day (12,17).

Fungal infections of the oropharynx are caused

predominantly by *Candida albicans*. This saprophyte, part of normal human flora in 25% of patients, may become an invasive pathogen in the immunocompromised host (17). Candidal pharyngitis is manifested as thrush in the infant due to bottle irritation (36). Adult patients are predisposed by immunosuppression, antibiotics, debilitation, diabetes mellitus, neck irradiation, HIV infection, and denture irritation (2). Symptoms include dysphagia and odynophagia along with an adherent white plaque with focal bleeding points (4). Diagnosis is aided by the observation of yeast forms on KOH preparation or Sabouraud's agar culture (38). Therapy includes oral preparation such as nystatin suspension (400,000 U qid) (17).

Viral infection is the most common cause of pharyngitis in adults and pediatric patients (39). A prospective study of exudative pharyngitis in children found 42% due to adenovirus, while 31% were caused by GABHS (39). Isolates of 200 viruses from 6 families have been implicated, including the ortho-, paramyxo-, picorna-, adeno-, and hepatoviridae (10). Viral pharyngitis is most often due to adenovirus (19%), Epstein-Barr (9%), parainfluenza (7%), influenza A (3%), Herpes simplex (2%), and respiratory syncytial virus (2%) (13,39). The disease process is caused by direct invasion of the oropharyngeal mucosa or secondary irritation due to nasopharyngeal colonization.

There is an age-related predisposition of disease, with pediatric patients affected by parainfluenza or respiratory syncytial virus, while influenza is more commonly found in adults (40). Viral pharyngitis also demonstrates a seasonal preference, occurring most often in the fall and winter (40). Symptoms include cough, rhinorrhea, myalgia, and headache accompanying the odynophagia (13). These associated upper respiratory symptoms are suggested to differentiate viral from streptococcal pharyngitis, but this contention has been disproven (19). Signs include a white exudate, as opposed to the purulent, yellow exudate of streptococcal disease, along with the absence of adenopathy (13). The diagnosis is based on clinical suspicion, and a laboratory analysis often reveals a normal leukocyte count. Therapy is supportive, with topical agents including viscous lidocaine (2%), dyclonine (0.5%), diphenhydramine, or phenol providing pain control (17). Careful consideration of the toxic side effects of these agents must be considered. However, agent-specific therapy is limited, amantadine for influenza A and acyclovir for herpes simplex infections.

There are several specific viral inflammatory disease entities that may be described by location of the

oropharynx affected. The posterior oropharynx is involved in herpangina, due to Coxsackie A types (2,4,5,6,8,10) presenting with severe odynophagia, fever, and salivation without URI symptoms (4). Pharyngeal involvement is manifested as 1- to 2-mm vesicles that subsequently ulcerate, resolving within 5 days. Variants include hand-foot-and-mouth disease caused by Coxsackie type A-16 with pharyngitis accompanied by vesicles on palm and sole surfaces, and lymphonodular pharyngitis due to Coxsackie A-10 features prominent lymphatic involvement (4,12). The middle oropharyngeal region is involved in aphthous stomatitis. Its etiology is idiopathic, but non-specific viral infection in patients predisposed to stress and anxiety is postulated (4). Symptoms include round, painful lesions that resolve within 2 weeks. Therapy includes corticosteroids, specifically Kenalog, an oral suspension, and topical tetracycline (250 mg/50 cc water) as an oral rinse (12,40). The anterior pharynx is affected by herpes simplex virus found in pediatric patients in the 2- to 5-year age range (39,40). Symptoms include fever, oral fetor, submaxillary adenopathy, and a gingivostomatitis that resolves in one to two weeks. Therapy is supportive but may include acyclovir in immunocompromised patients.

Infectious mononucleosis, attributed to the Epstein-Barr virus, is a common occult cause of pharyngitis affecting children and young adults (41,42). Patients with serologically proven mononucleosis have pharyngitis in almost all cases in children (99%) and in slightly fewer of the adults (85%) (41). The most prominent symptom is odynophagia in 80% of cases (41). Symptoms include anterior and posterior cervical adenopathy in 100%, gray pseudomembrane in 50%, and palatine petechiae in 33% of patients (41). The differential diagnosis of pseudomembranous pharyngitis includes candida and pemphigus vulgaris (17). Laboratory evaluation reveals a leukocytosis with a lymphocyte predominance, with 60% total or 10% atypical forms (43). Qualitative serologic diagnosis proceeds via assay for heterophile antibody by the monospot slide agglutination test or the Bunnell-Davidson hemolysis test (42). The monospot test is accurate in adults, but the true positive rate is only 90% in those 5 years of age, 75% for 2- to 4-year-olds, and 30% in the 0- to 20-year age range (42). Quantitative serologic analysis finds IgG or IgM antibody to the Epstein-Barr viral capsid antigen (43). Therapy is supportive, but may include penicillin for simultaneous streptococcus infection and steroids for respiratory obstruction.

Finally, pharyngitis can occur as a manifestation of systemic disease or noninfectious causes. Pemphi-

gus is an idiopathic condition in which 50% of patients present with oral involvement (17). These painless, bullous lesions soon progress to a fulminant, fibrinous exudate. The condition is diagnosed by acantholysis on skin biopsy or immunofluorescent antibody staining (4). Therapy includes steroids and immunosuppressant agents. Erythema multiforme or Steven-Johnson syndrome due to pharmacologic or idiopathic causes may also present with oropharyngeal involvement (17). Symptoms include oral vesicles and bullae associated with a white pseudomembrane along with cutaneous palm and sole lesions (4). Fulminant laryngeal inflammation is a rare presentation of neutropenia secondary to pharmacologic agents such as phenytoin, phenylbutazone, chloramphenicol, chlorpromazine, and mebromate, or the neoplastic involvement of leukemia (38). The patient has a toxic appearance with gingival bleeding and ulcerative gangrenous mucositis. Diagnosis and therapy are directed towards the underlying etiology or sepsis.

Allergic involvement of the oropharynx can be immediately life threatening, if airway compromise ensues. Quincke's disease is characterized by uvula and soft palate edema secondary to a presumed viral etiology (44). Symptoms include voice alterations and subjective throat fullness accompanied by watery, edematous appearance of the uvula. Therapy includes epinephrine, steroids, diphenhydramine, and surgical decompression (17). Evans reported the efficacy of nasal lidocaine (1%) and epinephrine (1 : 100,000) administered in 0.5-mg increments in reversing life-threatening laryngeal spasm (44). Angioneurotic edema is transient localized submucosal edema of the oropharynx and extremities. The hereditary variant is due to an autosomal dominant deficiency of C₁ esterase inhibitor, and is associated with a 5% to 50% mortality due to a subglottic edema (17,44). The atopic, nonhereditary variant is less commonly associated with pharyngeal involvement (44). Symptoms include urticaria and nonpitting swelling (4). Laboratory diagnosis finds decreased C₁ esterase inhibitor and complement (C₂, C₃) levels (44). Therapy includes the administration of epinephrine.

Perhaps the most crucial diagnostic challenge is distinguishing acute epiglottitis from pharyngitis. Although a rare occurrence, with an incidence of 10 : 100,000 in pediatric patients (<15 years) and 1-8 : 100,000 in adult patients, the morbidity and mortality due to airway obstruction are significant (45). Symptoms and signs include an inflamed epiglottis, stridor or secretion difficulty, and fever of 38°C, determined in a retrospective study of 841 patients (45). Clinical presentation is variable with the very

young (<2 years) manifesting a prominent fever (37.8°C), while only 45% have odynophagia (46). The classic presentation of epiglottitis with drooling is characteristic of older pediatric patients (>2 years), while the adult is found to have more benign symptomatic involvement (47). The etiology is most often *H influenza*, found in 92% of children and 53% of adults (45).

Diagnosis involves direct visualization of an inflamed epiglottis in a controlled setting. Radiographic criteria include an epiglottis to third cervical vertebrae width of 0.35 and height of 0.6, found to be 100% sensitive (48). Complications include acute airway obstruction and sepsis found more often in pediatric than adult disease (47). Therapy stresses airway control and treatment with ampicillin and chloramphenicol or a second generation cephalosporin (cefamandol, cefuroxime).

THERAPY

High risk groups for streptococcal pharyngitis have been suggested to include those with a history of prior rheumatic fever, scarlatiniform rash, diabetes mellitus, alcoholism, prolonged illness (6 days), and splenectomy (2). Therapy is advocated independent of culture evaluation in light of the hazards of sequelae. The inherent risks of empiric therapy include allergic reaction. Penicillin is the most common cause of fatal anaphylaxis. A pseudoallergic reaction can occur when drug use is complicated by coincident viral exanthem. Finally, induction of bacterial resistance has been cited with overtreatment, although there are currently no penicillin-resistant streptococcus strains in the United States (9,49). However, widespread empiric use of erythromycin for pharyngitis in Japan in 1975 resulted in the emergence of erythromycin-resistant streptococci in 50% of the cases (49).

The goals of therapy include hastening clinical recovery, decreasing the carrier state, and prevention of suppurative and nonsuppurative sequelae. Therapy is associated with symptomatic improvement, demonstrated by remission of fever by 24 hours, and decreasing symptoms and suppurative sequelae by 72 hours (50). Although the relapse rate in patients treated with penicillin versus placebo is identical (51), the incidence of suppurative sequelae is reduced slightly in treated (1.1%) compared to untreated (8.6%) patients (52).

The most significant nonsuppurative sequelae of streptococcal pharyngitis is rheumatic fever, with a 0.3% endemic and 3.0% epidemic incidence (53). It

was first reported by Wannamaker, in 1951, that penicillin therapy within the first 9 days of illness could prevent rheumatic fever (54). Recently, a resurgence in acute rheumatic fever has been attributed to increased streptococcal virulence, decreased reporting, or undue reliance on the rapid strep screen (55). Poststreptococcal glomerulonephritis due to serotype 12-M occurring 10 to 19 days after illness has also been described (56). Its course is not altered by antibiotic therapy (17,30,56). Finally, a reduction in the prevalence of the nasal carrier state can limit household spread.

The standard of therapy for streptococcal pharyngitis is penicillin. Routine dosing is oral penicillin VK in a 25- to 50-mg/kg/day pediatric and 1- to 2-g/day adult dose for 10 days of therapy (33). Intramuscular therapy includes benzathine penicillin G administered in a 300,000–600,000-unit dose for young children (< 30 kg) and a 900,000-unit dose for older pediatric patients (> 30 kg). Adults may be treated with Bicillin CR containing 900,000 units of benzathine penicillin G and 300,000 units of procaine penicillin (10,38). This route of administration is suggested to negate the issue of patient compliance with an oral regimen. However, the injection is painful, lessened somewhat in the Bicillin CR preparation, and is more expensive but offers a higher bacteriologic cure rate (90%) (57).

The superiority of the intramuscular compared with the oral penicillin formulation has been demonstrated by Kaplan where treatment failure was 8% and 38%, respectively (27). Analysis of treatment recommendations reveals that length of therapy is crucial, with a higher failure rate in patients treated for 7 rather than 10 days (58). This is significant, since the rate of compliance with medication recommendations is poor. Bergman reported that 71% of patients discontinued penicillin therapy by day 6 and 82% by day 9 of therapy (59). The dosing interval is also significant. Similar cure rates have been demonstrated in both adult and pediatric patients comparing bid to tid or qid administration regimens of the same total dose of penicillin or erythromycin (60,61). The reduction to bid dosing is suggested to improve patient compliance. The preferred route of administration has routinely been intramuscular with efficacy in 93% to 96% of cases, presumably due to compliance (57). However, the failure rate of intramuscular penicillin has increased from 10% to 21% since 1970 (57,62). Smith has suggested that treatment failure of streptococcal pharyngitis can occur in as many as 21% of cases, with 10% demonstrating *in vitro* tolerance to penicillin (62). However, this finding constitutes failure of GABHS eradication,

which may persist as a colonist, not necessarily producing symptoms. They suggest therapy with β -lactamase agents, specifically dicloxacillin, which may be efficacious, but not necessarily since suppurative complications are not increased (62).

Alternative therapy in penicillin-allergic patients includes erythromycin or cephalosporins. Tetracycline and sulfonamides cannot be recommended in light of failure rates of 15% and 80%, respectively (63). The second most frequently utilized agent for therapy of streptococcal pharyngitis is erythromycin. The erythromycin ethyl succinate preparation in a 40 mg/kg/24-hour pediatric and 1- to 2-g daily adult dose are utilized, avoiding the cholestatic jaundice risk of the estolate formulation (33). Again, it has been reported that bid versus qid administration of the same total dose provides similar cure rates, 93% and 90% respectively, due to increased patient compliance (64). Erythromycin is also advocated as primary empiric therapy, because of efficacy in streptococcal, nonstreptococcal, mycoplasma, and chlamydial pharyngitis (65). However, the incidence of increased gastrointestinal side effects is often prohibitive.

Cephalosporins are suggested as third line agents for streptococcal infection. The risk of penicillin allergy cross sensitivity is approximately 2%, with most allergic reactions classified as mild (66). Prospective clinical trials of cefadroxil, administered in 1-gram bid doses in adults and 30 mg/kg/24 hours in pediatric cases, demonstrated a decrease in disease recurrence, 0% to 3% compared to 9% to 13% in standard penicillin therapy (67,68). Advantages include better compliance with convenient bid dosing and shorter duration of therapy (5 days), but benefits may be offset by cost considerations.

The utility of newer antimicrobial agents such as the fluoroquinolones (ciprofloxacin, ofloxacin) and macrolides (clarithromycin, azithromycin) has yet to be explored.

Finally, surgical therapy for refractory tonsillitis is indicated with peritonsillar abscess, respiratory obstruction, or repetitive tonsillitis episodes. Recommendations originating from evaluation of pediatric patients suggests that tonsillectomy is warranted for 7 episodes per year, 5 episodes over 2 years, and 3 episodes of symptomatic tonsillitis over a 3 year period (69).

Streptococcal infection most frequently involves the palatine tonsils, but a variant involving the lingual tonsil is also described (70). Lingual tonsillitis incorporates similar symptomatology with the addition of altered phonation, pain localized to deep structures, discomfort with tongue depression and re-

sistance to protrusion, along with hyoid tenderness (17,70). The diagnosis is suggested by indirect laryngoscopy or a lateral neck radiography; standard pharyngitis therapy is instituted.

COMPLICATIONS

Pharyngitis therapy is also directed at suppurative complications, included superficial and deep neck abscesses. The most frequent complication is peritonsillar abscess (45%) localized to the superior pole of the tonsillar capsule and fascia of the superior constrictor muscle (71). The bacteriologic spectrum features aerobic and anaerobic streptococcus, along with staphylococcus species (72). However, Sprinkle found that only 8.3% of these cases were caused by GABHS (72). This complication affects teenagers and young adults, with children less than 12 years of age rarely affected (73). The disease course is suggested by remission of pharyngitis followed by clinical decompensation. Symptoms include unilateral pharyngeal pain associated with trismus due to lateral pyterigoid irritation, peritonsillar erythema or edema, and uvular deviation to the opposite side (17).

Diagnosis of peritonsillar abscess proceeds via transoral aspiration lateral to the superior tonsillar pole with a 95% success rate (74). Traditional therapy is based on hospital admission and open surgical drainage. Ophir suggests that patients with peritonsillar cellulitis without abscess or with abscess without associated trismus and airway compromise may be managed with needle aspiration and outpatient antibiotics with an 85% success rate (75). However, the expertise required and the difficulty with complications and patient follow-up suggest this to be a procedure best referred to the otolaryngologist in a controlled setting.

The superficial fascial spaces, including the canine, buccal, masticator, vestibular, and maxillary compartments and are prone to infection in 20.5% of cases by anaerobic (50%) or mixed (45%) bacteria (71,76). However, most are due to odontogenic involvement from poor oral hygiene (77). Therapy is surgical accompanied by antibiotic therapy, usually penicillin or clindamycin (77). The sublingual space composed of the sublingual and submaxillary areas are involved in Ludwig's angina, occurring in 21.5% of patients (76,77). This condition is predominantly secondary to dental involvement (80%), but may be a complication of lingual tonsillitis (70,71). Symptoms include odynophagia with tongue protrusion due to sublingual swelling (71).

The most likely complication of pharyngitis is the

parapharyngeal abscess, which is due to direct extension of the suppurative process and is the fifth in incidence of deep neck abscesses (3.4%) (71). Anatomically, the pterygomaxillary space consists of an anterior portion, which contains the tonsillar fossa and internal pterygoid muscle, and the posterior portion, which contains cranial nerves IX through XII, internal carotid artery, internal jugular vein, and sympathetic trunk (77). This condition is predominantly due to Group A and non-Group-A BHS. Symptoms include odynophagia, ipsilateral otalgia, and neck asymmetry and rigidity (17,73). The spread of infection proceeds from the anterior peritonsillar region, resulting in lateral pterygoid irritation and trismus, to the posterior area, resulting in midline tonsil deviation (76).

The retropharyngeal space, between the buccopharyngeal and alar fascia of the cervical spine, is the fourth most common deep neck abscess caused by suppuration of the retropharyngeal lymph node (9.6%) (71,78). This complication is usually due to anaerobic streptococci secondary to otitis media (17). Symptoms include odynophagia without trismus and a "Cri du Canard" (duck quack) voice (76). The patient maintains a hyperextended posture with torticollis to the uninvolved side (17,76). Diagnosis is assisted by lateral neck radiograph with demonstrates a soft tissue shadow greater than 3.5 to 7 mm at the C2 and 14 to 20 mm at the C6 level (79).

Lastly, the prevertebral space is a potential area between the retropharyngeal space and the alar prevertebral fascia. Involvement is secondary to anterior progression of bone disease such as osteomyelitis due to staphylococcus or mycobacteria (78). Neck swelling and inflammation are absent, but cervical tenderness is noted. Diagnosis is suggested by radiographic presentation of cervical spine involvement (79). Therapy for these suppurative complications is based on airway protection, usually followed by surgical intervention.

CONCLUSION

One of the most common presenting complaints in the emergency department is a symptom complex consistent with the diagnosis of pharyngitis. Streptococcal pharyngitis is suggested by fever, oropharyngeal exudate, anterior cervical adenopathy, and the absence of common cold symptoms. Diagnosis is suggested by a positive rapid strep latex agglutination or ELISA screen. However, a negative result should be followed by routine strep culture to avoid a missed diagnosis.

The goals of therapy are to hasten the resolution of symptoms and to decrease the rate of suppurative and nonsuppurative complications. Empiric treatment may be instituted in epidemic conditions with high disease prevalence. Selective therapy is utilized in endemic, low prevalence conditions, based on the presence of 3 of 4 clinical markers, a positive rapid strep screen, or positive GABHS culture if the rapid screen is negative.

Therapy may be instituted with penicillin or eryth-

romycin, effective for emerging new pathogens such as *H influenza*, *C trachomatis*, and *A hemolyticum*. Currently, the most effective regimen, independent of cost considerations, includes oral first generation cephalosporins, raising the issue of synergistic pathogens, such as *S aureus*.

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