Pediatric ENT Infections

Cemal Cingi
Emin Sami Arısoy
Nuray Bayar Muluk
Editors

Springer

Acute Tonsillopharyngitis in Children

44

Necdet Demir, Nuray Bayar Muluk, and Dennis Chua

44.1 Introduction

Tonsillopharyngitis can be defined as a condition in which the pharynx and/or palatine tonsils become acutely infected and is accompanied by sore throat, difficulty swallowing, pyrexia and lymphadenopathy in the cervical region. It can be diagnosed clinically, diagnosis being confirmed microbiologically or via the rapid antigen test [1].

Inflammation solely affecting the tonsils of the pharynx is tonsillitis, but, as the adenoids and lingual tonsils are frequently also involved, pharyngitis may be an equally appropriate term to use. For our purposes we can equate pharyngotonsillitis with adeno-tonsillitis, noting that lingual tonsillitis has a meaning restricted to inflammation occurring in the lymphoid tissue situated at the lingual base [2].

The pharynx may be inflamed or irritated by various pathogens: viruses, such as the Adenoviridae, Enteroviridae and Epstein-Barr virus [EBV] occur in juveniles and are treated routinely on a conservative basis; pathogenic bacteria such as GABHS necessitate antibacterial chemotherapy. However, in all cases in which children are affected, regardless of aetiology, care must be taken to prevent dehydration or other common clinical sequelae [3].

N. Demir (✉)
Section of Otorhinolaryngology, VM Medical Park Pendik Hospital, Pendik, İstanbul, Turkey

N. Bayar Muluk
Department of Otorhinolaryngology, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkey

D. Chua
Section of Otorhinolaryngology, and Ear, Nose, and Throat Surgeons Medical Centre, Mount Elizabeth Hospital, Singapore, Singapore

C. Cingi et al. (eds.), *Pediatric ENT Infections*,
https://doi.org/10.1007/978-3-030-80691-0_44

44.2 Aetiology

Tonsillitis and its sequelae are caused by both host and parasite factors; thus overcrowding and malnutrition are implicated as are the following pathogens, which account for the majority of cases: Herpes simplex, EBV, Cytomegalovirus, other members of the Herpesviridae, the Adenoviridae and the Measles virus [2].

Tonsillopharyngitis is generally secondary to viral infection: commonly, a coryza-causing virus, e.g. rhinovirus, adenovirus, influenza virus, respiratory syncytial virus; more rarely, EBV (Epstein-Barr virus, the infectious agent in mononucleosis) or HIV (human immunodeficiency virus).

Bacteria cause less than one in three cases of tonsillopharyngitis; however, in these cases, the usual pathogens responsible are Group A Streptococci and typically this so-called "strep throat" afflicts children aged between 5 and 15 years old, being less often seen in children younger than 3, or older adults. If untreated, the illness may be complicated by tonsillar abscess and cellulitis, rheumatic fever or glomerulonephritis. On occasion, gonococcal infection or diphtheria may present as tonsillopharyngitis [4].

Of the between 15% and 30% of cases attributable to bacterial causes, anaerobes are a key cause, but the majority of infections are with the GABHS organism, Streptococcus pyogenes. This organism has the ability to bind to adhesin molecules found in the epithelium of the tonsil and, indeed, antibody coating of the pathogen may be a key step in the development of tonsillitis secondary to bacterial infection [2].

Acute pharyngitis on rare occasions may be due to Mycoplasma pneumoniae, Corynebacterium diphtheriae or Chlamydia pneumoniae and the sexually transmitted pathogen Neisseria gonorrhoea is also a possible cause. Whilst the UK and Nordic countries consider Arcanobacterium haemolyticum a significant cause of acute pharyngitis, the USA does not count the organism as a significant pharyngitic pathogen. A. haemolyticum is associated with a dermatological eruption resembling Scarlatina [2].

Tonsillopharyngitis in a pyrexial patient experiencing a sudden sore throat and whose tonsils are exuding pus, with neck glands painful to the touch, usually resolves on its own within a 2–5-day period and is most likely due to Streptococcus pyogenes. This organism is also referred to as Group A Streptococcus (GAS). Sore throats of longer duration are rarely due to GAS [5].

44.3 Epidemiology

The peak occurrence of the condition is in the paediatric age range, with the caveat that it is rare in those younger than 2 years of age. Aetiology is age-related, cases in those aged 5–15 typically being Streptococcal in origin, whilst in younger children a virus is suspected. Peritonsillar abscess (PTA) has a predilection for adolescents, but may occur at a younger age [2].

Pharyngitis is frequent in upper respiratory tract infection. The carrier rate in children is 2.5–10.9%. 15.9% of school age children on average were carriers of GAS according to one study [6, 7].

44.4 Symptoms

Acute tonsillitis in children presents with sore throat, pyrexia, halitosis, dysphagia, odynophagia and cervical lymphadenopathy. The obstructed airway may lead to mouth breathing, snoring or sleep-disordered breathing such as momentarily ceasing to breathe whilst asleep or sleep apnoea. Patients are commonly debilitated and lethargic. Whilst adequate symptomatic resolution is frequent within 3–4 days, even well-treated cases may last up to a fortnight.

A patient with seven episodes of tonsillitis in 1 year, 5 for 2 years running, or 3 in each year for a 3-year period, all of which have been proven microbiologically as streptococcal should be diagnosed with recurrent streptococcal tonsillitis. In such cases, chronic sore throat, foetid breath, longstanding cervical lymphadenopathy and tonsillitis may be seen. Carriers can easily infect children [2].

A child suffering from tonsillopharyngitis will exhibit pyrexia, sore throat, halitosis, dysphagia, odynophagia and cervical lymph glands which are painful to touch.

44.5 Diagnosis

44.5.1 Examination

Acute tonsillitis is apparent when a pyrexial patient has swollen and erythematous tonsils, possibly with exuded pus. If petechiae are visible on the palate, Streptococcus pyogenes (a Group A β-haemolytic organism) or EBV may be responsible, the former usually occurring in the age range 5–15 years.

Lymph nodes painful on palpation and a stiff neck are seen in acute tonsillitis. Mucous membranes and skin turgor should be assessed to determine fluid status. In adolescents and younger children, EBV-associated infectious mononucleosis needs to be excluded; especially if acute tonsillitis is present alongside lymphadenopathy in the neck, axilla and/or groin, an enlarged spleen, lethargy and malaise and slight elevation in temperature. EBV tonsillitis may produce a greyish membrane over inflamed tonsils, removal of which does not result in bleeding. The palate may have an eroded mucosa with bruising of the hard palate [2].

44.5.2 Investigations

The Gold Standard for diagnosing GABHS infection is by throat swab and culture. Antibiotics are the most effective (90–95%) in this group and can definitely be employed. Given the problems associated with bacterial resistance, swab and

culture of throat organisms in cases of tonsillitis may be considered essential. It is ineffective to rely solely on clinical observation and on whether exudates are seen, erythema and lymphadenopathy observed and pyrexia recorded to distinguish between tonsillitis secondary to a virus or secondary to GABHS [2]. Thus, microbiological culture, despite a potential delay of up to 2 days, remains the best way to diagnose accurately. Whilst sensitivity and specificity for GABHS are both generally high using the throat culture method, it is worth bearing in mind that the protocol followed in obtaining the swab and the culture medium employed does have some effect on the final result.

Where appropriate, Monospot, Full blood count and electrolyte should be performed [2].

There are also Rapid Antigen Detection Tests (RADT) to which the majority of clinical settings have access and which is helpful when treatment needs to begin without delay. In combination with throat culture, RADT achieves high reliability. RADT alone is 70–90% as sensitive and 95–100% as specific as throat culture [3].

Thus, a form of RADT and culture is standard in most settings, even more so in the Developed World. Where testing has needed to be performed serially, RADT then culture for the RADT test negatives has shown highest efficacy and has the potential to reduce unwarranted antimicrobial therapy through restricting antibiotic prescribing to cases confirmed by either initial screen or subsequent culture [8].

Serum immunoglobulin titres (antistreptolysin-O, antideoxyribonuclease-B (anti-DNAse-B)) may be used to demonstrate antecedent infection in cases of acute rheumatic fever, glomerulonephritis and the other sequelae of Group A streptococcal pharyngitis [2].

Virology. In cases of suspected EBV, a full blood count can support the diagnosis by detecting atypical leucocytes as can a Monospot or other heterophile antibody detection method. A subclinical hepatitic picture is also seen in EBV with mildly elevated transaminases [3].

Nonetheless, monospot testing may produce false negatives in patients aged under 6 years as well as in the initial symptomatic 7-day period. Monospot has a 90% true positive rate in adolescents whose diagnosis is eventually proven serologically [3].

In a viral epidemic, such as H1N1 influenza, where sore throat features as a presenting complaint, it is at the clinician's discretion whether to investigate a possible streptococcal aetiology at the initial time of presentation or subsequently after symptoms have continued for some time [3].

Imaging studies in the form of X-rays are of no diagnostic merit in acute tonsillitis except where there has been a progression to deeper seated infection behind the oropharyngeal fascia. In the latter case, lateral plain film or contrast-enhanced CT are indicated [2].

44.6 Treatment

44.6.1 Medical Treatment

Patients with acute tonsillitis require little beyond ensuring adequate hydration and absorbing sufficient calories alongside adequate analgesia and antipyretic. If the patient cannot eat and drink enough, antibiotic therapy, fluids and analgesia will need to be provided intravenously. Patients with good oral intake do not need hospitalization and indeed IV fluids may be provided outside hospital if a suitably qualified attendant is available. Steroidal therapy supplied via an IV line may be indicated to reduce severe throat oedema [2].

To prevent the possibility of too readily diagnosing bacterial cause and thus overprescribing antimicrobial therapy, a clinical scoring method should precede sending swabs for culture or using RADT. Treatment aims to alleviate symptoms, shorten the time for which a patient remains contagious, and obviate suppuration locally or systemic involvement. In an ideal world, prescribing antimicrobials would only happen following laboratory confirmation. Where RADT is unfeasible or results in a probable false negative, culture remains the diagnosis test par excellence and if the clinician has confidence in the eventual diagnosis, antimicrobial therapy can be undertaken. The first line is usually Penicillin with Amoxicillin an acceptable alternative. Co-amoxiclav should not be used empirically. Nor are the Macrolides to be used first line except where a Penicillin reaction may occur and to treat carriers of GABHS [9].

Antibiotic treatment versus GABHS causing acute pharyngitis aims to [5]:

Alleviate the harshness of symptoms and shorten their length. Likewise in cases complicated by suppuration, reduce the likelihood of other, non-suppuration-related conditions such as acute rheumatic fever.

The best evidence for a role of antibiotics in achieving this is found in acute rheumatic fever, whereas glomerulonephritis and PANDAS (paediatric autoimmune neuropsychiatric disorder associated with group A streptococci syndrome) have less conclusive evidence [10]. PANDAS is covered elsewhere but the evidence for antibiotics having a preventive effect is inconclusive [5].

Diminution of Transmissivity to Others in Proximity Via a Reduction in Infective Potential

A patient with symptoms of pharyngitis due to a documented GABHS infection, whether detected by RADT or culture, warrants treatment with antibiotics [11]. Infection with Group C or G Streptococci can also be treated with antibiotics to alleviate symptoms. Which antibiotic to employ in these situations will be discussed below, but, given that acute rheumatic fever is not a consequence of Group C or G streptococci, 5 rather than 10 days' prescription is adequate [12–14].

Streptococcal infection (or the less seldom encountered Corynebacterium diphtheriae and Neisseria gonorrhoeae) is a clear indication for antibiotic prescription. Other bacterial agents responsible for pharyngitis are not effectively treated by antimicrobials and entails unjustified financial burdens, side-effects and contributes to microbial resistance [5].

44.6.1.1 Timing of Therapy

Antibiotics may be started empirically where a case of pharyngitis presents with clinical and epidemiological features strongly suggestive of GABHS, but does not yet have positive laboratory testing. Where such confirmation from the laboratory is not forthcoming, stopping the antibiotic is best [5].

A latent period of 48–96 h is normal in GABHS pharyngitis. Systemic symptoms, including fever, abate normally within 3 or 4 days regardless of antibiotic use. [15] Research has shown that Penicillin produces symptomatic improvement up to 48 h earlier if administered in the first 2 days, compared with placebo. [16–20].

Some patients with negative RADT will have swabs that culture the pathogen. Even if these patients are improving clinically, they still need antimicrobial treatment to diminish the chance of passing on the infection [5].

On area of controversy concerns whether treating too early may in fact suppress the patient's immunoglobulin response and act to promote recurrent pharyngitis. A study of cases of GABHS pharyngitis comparing those receiving treatment at first consultation versus a second group in which treatment did not begin within the first 48 hours, concluded that the first group had eightfold more recurrent infection. [17].

44.6.1.2 Antibiotics for Group A Streptococcus

The treatment armamentarium in GABHS pharyngitis includes Penicillin and derivatives (such as Ampicillin and Amoxicillin), the Cephalosporins, Macrolides and Clindamycin. [21]. It does not include Sulphonamides, Fluoroquinolones or Tetracyclines since organisms are either resistant or are simply not eradicated in the pharynx by these drugs. Penicillin which has been administered intramuscularly is currently the sole agent proven in controlled trials to obviate the first signs of rheumatic fever [22, 23].

44.6.2 Surgery

Tonsillectomy should be offered where: the patient has had pharyngitis of streptococcal origin, for which microbiological documentation exists, more than six times in 1 year; more than five times per year for 2 years in succession: three episodes of either infectious tonsillitis or adenitis for three consecutive years even with adequate drug treatment; chronic (recurrent) tonsillitis in a carrier of Streptococcus, where antibiotics not deactivated by the β-lactamase enzyme have failed to eradicate carriage [2].

Children with the following may also be offered tonsillectomy: several allergies or sensitivities to antimicrobials; PFAPA (periodic fever, aphthous stomatitis, pharyngitis and adenitis); previous peritonsillar abscess [24].

Only seldomly is acute lingual tonsillitis a reason for surgery, but if this otherwise rare condition occurs many times or is severe, tonsillitis may be offered. If the tonsils have hypertrophied due to mononucleosis and continue to impinge symptomatically on the airway after the illness resolves, they may need to be removed surgically [2].

44.6.2.1 Tonsillectomy Indications

The following are sufficient in themselves to indicate a tonsillectomy [25]:

Tonsillomegaly leading to upper airway partial occlusion, great difficulty swallowing, disordered sleep or cardiopulmonary involvement.

Medical treatment followed by surgical drainage of the peritonsillar abscess (provided this was not when the disease was acute) which failed.

Febrile convulsions secondary to tonsillitis.

Histopathologically mandated.

Whilst in the following cases, tonsillectomy may be needed: [25].

Infectious tonsillitis occurring more than three times in 12 months even when otherwise adequately treated medically.

Ongoing dysgeusia or halitosis secondary to chronic tonsillar infection which fails to respond medically.

Streptococcal carriage not eradicable by antimicrobials unaffected by beta-lactamase.

Hypertrophic tonsil on one side which is likely to be cancerous.

The following methods may be employed for tonsillar dissection and excision [26–29]:

Cold steel methods (such as curette or scissors).

Monopolar cautery.

Bipolar cautery (microscopically assisted or not).

COBLATION (radiofrequency ablation, can reduce tonsillar bulk).

Titanium-bladed Scalpel of harmonic type.

Microdebrider-assisted intracapsular tonsillectomy or use of other powered instrumentation.

Haemostasis may be achieved intraoperationally by the following: [25].

Sponge pressure lasting minutes.

Administration of bismuth subgallate.

Tie use.

Cautery employing suction.

Bipolar cautery.

References

1. Sasaki CT. Tonsillopharyngitis. MSD Manual. https://www.msdmanuals.com/professional/ear,-nose,-and-throat-disorders/oral-and-pharyngeal-disorders/tonsillopharyngitis (Accessed online at August 11, 2020).
2. Shah UK. Tonsillitis and peritonsillar abscess. In: Meyers AD (Ed.). Medscape. Updated: Jan 19, 2017. http://emedicine.medscape.com/article/871977-overview (Accessed August 11, 2020).

3. Simon HK. Pediatric pharyngitis. In: Steele RW (Ed.). Medscape. Updated: Apr 26, 2016. http://emedicine.medscape.com/article/967384-overview#a4 (Accessed online at August 11, 2020).
4. Sasaki CT. Tonsillopharyngitis (Tonsillitis; Pharyngitis). Merck Manual Consumer version. http://www.merckmanuals.com/home/ear,-nose,-and-throat-disorders/mouth-and-throat-disorders/tonsillopharyngitis#v8369892 (Accessed online at August 11, 2020).
5. Pichichero ME. Treatment and prevention of streptococcal tonsillopharyngitis. In: Sexton DJ, Edwards MS, Bond S (Eds.). UpToDate. https://www.uptodate.com/contents/treatment-and-prevention-of-streptococcal-tonsillopharyngitis (Accessed online at August 11, 2020).
6. Pichichero ME, Casey JR. Defining and dealing with carriers of group a streptococci. Contemp Pediatr. 2003;1:46.
7. Wald ER. Commentary: antibiotic treatment of pharyngitis. Pediatr Rev. 2001;22(8):255–6.
8. Ayanruoh S, Waseem M, Quee F, Humphrey A, Reynolds T. Impact of rapid streptococcal test on antibiotic use in a pediatric emergency department. Pediatr Emerg Care. 2009;25(11):748–50.
9. Piñeiro Pérez R, Hijano Bandera F, Alvez González F, Fernández Landaluce A, Silva Rico JC, Pérez Cánovas C, Calvo Rey C, Cilleruelo Ortega MJ. Consensus document on the diagnosis and treatment of acute tonsillopharyngitis. An Pediatr. 2011;75(5):342. https://doi.org/10.1016/j.anpedi.2011.07.015.
10. Potter EV, Svartman M, Mohammed I, Cox R, Poon-King T, Earle DP. Tropical acute rheumatic fever and associated streptococcal infections compared with concurrent acute glomerulonephritis. J Pediatr. 1978;92(2):325.
11. Harris AM, Hicks LA, Qaseem A. High value care task force of the American College of Physicians and for the Centers for Disease Control and Prevention. Appropriate antibiotic use for acute respiratory tract infection in adults: advice for high-value care from the American College of Physicians and the Centers for Disease Control and Prevention. Ann Intern Med. 2016;164(6):425–34.
12. Gerber MA, Baltimore RS, Eaton CB, et al. Prevention of rheumatic fever and diagnosis and treatment of acute Streptococcal pharyngitis: a scientific statement from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee of the Council on Cardiovascular Disease in the Young, the Interdisciplinary Council on Functional Genomics and Translational Biology, and the Interdisciplinary Council on Quality of Care and Outcomes Research: endorsed by the American Academy of Pediatrics. Circulation. 2009;119(11):1541–51.
13. Meier FA, Centor RM, Graham L Jr, Dalton HP. Clinical and microbiological evidence for endemic pharyngitis among adults due to group C streptococci. Arch Intern Med. 1990;150(4):825–9.
14. Turner JC, Hayden FG, Lobo MC, et al. Epidemiologic evidence for Lancefield group C beta-hemolytic streptococci as a cause of exudative pharyngitis in college students. J Clin Microbiol. 1997;35(1):1–4.
15. Brink WR, Rammelkamp CH Jr, Denny FW, Wannamaker LW. Effect in penicillin and aureomycin on the natural course of streptococcal tonsillitis and pharyngitis. Am J Med. 1951;10(3):300–8.
16. Randolph MF, Gerber MA, DeMeo KK, Wright L. Effect of antibiotic therapy on the clinical course of streptococcal pharyngitis. J Pediatr. 1985;106(6):870–5.
17. Pichichero ME, Disney FA, Talpey WB, et al. Adverse and beneficial effects of immediate treatment of group a beta-hemolytic streptococcal pharyngitis with penicillin. Pediatr Infect Dis J. 1987;6(7):635–43.
18. Krober MS, Bass JW, Michels GN. Streptococcal pharyngitis. Placebo-controlled double-blind evaluation of clinical response to penicillin therapy. JAMA. 1985;253(9):1271–4.
19. Del Mar CB, Glasziou PP, Spinks AB. Antibiotics for sore throat. Cochrane Database Syst Rev. 2000;3:CD000023.
20. Gilbert GG, Pruitt BE. School health education in the United States. Hygie. 1984;3(4):10–5.
21. Betriu C, Sanchez A, Gomez M, et al. Antibiotic susceptibility of group a streptococci: a 6-year follow-up study. Antimicrob Agents Chemother. 1993;37:1717.

22. Denny FW, Wannamaker LW, Brink WR, et al. Prevention of rheumatic fever; treatment of the preceding streptococcic infection. J Am Med Assoc. 1950;143:151.
23. Wannamaker LW, Rammelkamp CH Jr, Denny FW, et al. Prophylaxis of acute rheumatic fever by treatment of the preceding streptococcal infection with various amounts of depot penicillin. Am J Med. 1951;10:673.
24. Baugh RF, Archer SM, Mitchell RB, Rosenfeld RM, Amin R, Burns JJ, et al. Clinical practice guideline: tonsillectomy in children. Otolaryngol Head Neck Surg. 2011;144(1 Suppl):S1–30.
25. Drake AF. Tonsillectomy. In: Meyers AD (Ed.). Medscape. Updated: Oct 23, 2015 http://reference.medscape.com/article/872119-overview#a10 (Accessed online at August 11, 2020).
26. Carr MM, Muecke CJ, Sohmer B, Nasser JG, Finley GA. Comparison of postoperative pain: tonsillectomy by blunt dissection or electrocautery dissection. J Otolaryngol. 2001;30(1):10–4.
27. Pizzuto MP, Brodsky L, Duffy L, Gendler J, Nauenberg E. A comparison of microbipolar cautery dissection to hot knife and cold knife cautery tonsillectomy. Int J Pediatr Otorhinolaryngol. 2000;52(3):239–46.
28. Lee KC, Bent JP 3rd, Dolitsky JN, Hinchcliffe AM, Mansfield EL, White AK. Surgical advances in tonsillectomy: report of a roundtable discussion. Ear Nose Throat J. 2004;83(8 Suppl 3):4–13.
29. Nelson LM. Radiofrequency treatment for obstructive tonsillar hypertrophy. Arch Otolaryngol Head Neck Surg. 2000;126(6):736–40.