# Chapter 10 Tonsillitis and Peritonsillar Abscess

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### **Abbreviations**

AAO-HNS American Academy of Otolaryngology—Head and Neck Surgery

ARF Acute rheumatic fever
ASO Anti-streptolysin-O
CMV Cytomegalovirus
CN IX Cranial nerve IX
CT Computed tomography
EBV Epstein-Barr virus

GABHS Group A β-hemolytic streptococcus

HSV Herpes simplex virus

IDSA Infectious Disease Society of America NSAID Non-steroidal anti-inflammatory PCR Polymerase chain reaction

PFAPA Periodic fevers, aphthous stomatitis, pharyngitis, and adenitis

PSGN Post-streptococcal glomerulonephritis

RADT Rapid antigen detection test SDB Sleep disordered breathing

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### **Anatomy**

Tonsils are lymphoid organs found in the nasopharynx and oropharynx. There are three main sets of tonsils: pharyngeal tonsils, lingual tonsils and palatine tonsils. The pharyngeal tonsils are more commonly referred to as the adenoids and reside midline in the nasopharynx. The lingual tonsils are found on the posterior one-third of the tongue. The palatine tonsils are nestled in the tonsillar fossa, defined by the tonsillar pillars of the oropharynx. The anterior pillar is comprised of the palatoglossus muscle, whereas the palatopharyngeus muscle makes up the posterior pillar. These tonsils are encapsulated by a specialized portion of the pharyngobasilar fascia. They are the most commonly infected set of tonsils and will be henceforth referred to as the "tonsils."

# Neurovascular Supply

Numerous blood vessels supply the tonsils, all branches of the external carotid artery. These arteries include branches of ascending pharyngeal, dorsal lingual, facial, and maxillary arteries, with the tonsillar branch of the facial artery being the greatest contributor.

Venous drainage of the tonsils is via the lingual and pharyngeal veins. Lymphatic drainage of the tonsils is primarily via the jugulodigastric system [1, 2].

Sensory innervation to the region is via the glossopharyngeal nerve (CN IX).

## Relationship to Surrounding Structures

#### Carotid Artery

The external branch of the carotid artery is located just lateral to the tonsillar fossa. The internal carotid artery is about 2 cm posterolateral to the deep portion of the tonsillar fossa. These structures can be compromised by acute processes or as a result of interventions taken in the tonsillar region (Fig. 10.1).

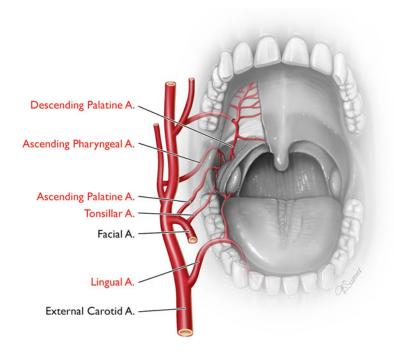
#### **Pterygoid Muscles**

The capsule of the tonsil is separated from the superior constrictor muscle by loose connective tissue. Lateral to the superior constrictor muscle lies the parapharyngeal space. The lateral border of the parapharyngeal space consists of the medial pterygoid muscle which can get irritated and inflamed in the event of parapharyngeal irritation or infection, resulting in trismus.

# Crypts

The tonsils are not smooth; instead they have numerous crypts or pits where food can get caught. Food accumulated within these crypts forms small stone-like structures known as tonsilliths which can then lead to inflammation and chronic throat pain.

Fig. 10.1 Vascular anatomy of the tonsil (Printed with permission from Texas Children's Hospital)



## **Immunologic Function of Tonsils**

The tonsils are lymphoepithelial organs that function as secondary lymphoid organs. They contain specialized epithelial M cells that capture and transport antigens entering through the mouth and nose to extrafollicular regions or lymphoid follicles. The lymphoid follicles then release antibody-expressing memory B cells or plasma cells that migrate to the tonsils and produce antibodies. These antibodies are subsequently released into the tonsillar crypt lumen. All five isotypes of immunoglobulins are produced in the tonsil. The most important of these isotypes is IgA which functions as an important component of the mucosal immune system of the upper airway [3].

The tonsils are at their largest size during the most active immunologic activity, which is estimated to be between the ages of 3 and 10 years. After this period they display spontaneous age-depended involution [3]. Chronic or recurrent tonsillitis alters the tonsillar immune system by causing shedding of the M cells and the tonsillar immunologic response to antigens weakens. The clinical significance of this dysfunction is controversial. There are no data demonstrating significant change in the systemic immune system after tonsillectomy [3].

#### **Tonsillitis**

Tonsillitis is inflammation of the tonsils, specifically the palatine tonsils.

# **Epidemiology**

Acute pharyngitis is one of the most common illnesses seen in the primary care setting accounting for up to 1.2 % of all emergency department visits and up to 6 % of office visits for children and

adolescents [4, 5]. Most cases in children are observed during winter and early spring when respiratory viruses are more common. During the summer months enteroviruses are responsible for the majority of cases [6]. Tonsillitis caused by Group A β-hemolytic streptococcus (GABHS) most commonly occurs in children 5–15 years old, affecting less than 15 % of children younger than 3 years old, 24 % of children less than 5 years old, and 37 % of school-aged children [7]. The financial burden of GABHS tonsillitis is estimated to be between \$224 and \$539 million per year with more than half being associated to non-medical costs [8]. *Neisseria gonorrhoeae* is an important pathogen in sexually active individuals or in victims of sexual abuse [6]. Repeated episodes of all-cause tonsillitis is reported in 0.9 % of children less than 1 year old and 5.3 % of children between the ages of 1 and 4 years old [9].

## **Microbiology**

Tonsillitis may be caused by a viral or bacterial infection of the tonsils, most commonly the palatine tonsils. Viral etiologies are the most common cause of tonsillitis in the pediatric population. Common viral pathogens include enteroviruses, particularly coxsackie virus, respiratory viruses (e.g. adenovirus, rhinovirus, influenza virus, coronavirus, parainfluenza virus and respiratory syncytial virus), and viruses of the herpesviridae family like Epstein-Barr Virus (EBV), cytomegalovirus (CMV) and herpes simplex virus (HSV) [7]. The most common bacterial pathogen implicated in acute tonsillitis is GABHS, accounting for up to 30 % of all episodes of acute pharyngotonsillitis in children. Less frequent bacterial causes include *Staphylococcus aureus*, *Streptococcus pneumoniae*, Group C streptococcus, *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*, *Corynebacterium diphtheriae*, *Arcanobacterium haemolyticum*, *Neisseria gonorrhea*, *Francisella tularensis*, *Yersinia enterocolitica*, and mixed anaerobic flora from the oral cavity [7]. *Fusobacterium necrophorum*, a gram-negative aerobic bacilli, and the most common cause of Lemierre's syndrome, has been cultured from adolescents and young adults with uncomplicated tonsillitis [10].

# **Symptoms**

Patients with tonsillitis present with a variety of symptoms that include sore throat, fever, chills, odynophagia, cervical adenopathy, trismus, halitosis, erythematous and exudative tonsils and tonsillar pillars (Fig. 10.2). The presence of conjunctivitis, coryza, cough, stomatitis, diarrhea and hoarseness strongly suggest a viral etiology. Children younger than 3 years of age may have an atypical presentation of GABHS infection called streptococcosis, which is characterized by fever, mucopurulent or serous rhinitis, and adenopathy, followed by irritability, loss of appetite and lethargy. Exudative tonsillitis is rare in this age group. On physical exam, it is often difficult to distinguish between viral and bacterial tonsillitis, but some clinical findings may provide important clues of the etiologic agent. For example, HSV typically presents with stomatitis, EBV may include lymphadenitis and coxsackie virus infections may present with throat ulcers (herpangina) or as part of hand-foot-mouth disease.

# **Complications**

Complications of tonsillitis can be suppurative or non-suppurative in nature. Suppurative complications include peritonsillar abscess, parapharyngeal or retropharyngeal space abscess, and suppurative cervical lymphadenitis. Acute airway compromise, rheumatic fever, glomerulonephritis, and scarlet

**Fig. 10.2** Tonsillitis with suppuration (Photo courtesy of Dr. Daniel P. Fox)



fever are non-suppurative complications of tonsillitis caused by GABHS. Streptococcal toxic shock syndrome, an uncommon but rapidly progressive disease, can complicate cases of pharyngitis caused by a toxic-producing strain of GABHS [11].

## Diagnosis

Tonsillitis is primarily a clinical diagnosis. Supportive tests include throat cultures, GABHS rapid antigen test, and anti-streptolysin-O (ASO), anti-deoxyribonuclease B (anti-Dnase B), anti-hyaluronidase and anti-streptokinase antibody titers [12]. Other tests may be helpful based on clinical suspicion, for example, EBV specific serology or Monospot (heterophile antibody) test, EBV polymerase chain reaction (PCR) or HSV PCR as needed. The monospot test is particularly insensitive in young children, with only 25–50 % of children under the age of 12 years infected with EBV having a positive Monospot test [13]. Specific EBV serology to detect antibodies against viral capsid antigens (VCA) that includes VCA-IgG and VCA-IgM in conjunction with antibodies against Epstein-Barr nuclear antigen or EBNA are the preferred diagnostic method in this age group. A real-time EBV PCR assay is helpful in patients with immunocompromising conditions and to confirm the diagnosis in patients with negative serology but strong clinical suspicion of infection [14].

The most important step in diagnosis is distinguishing between viral and GABHS tonsillitis as anti-bacterial agents are not effective in the treatment viral tonsillitis. Furthermore, with a few rare exceptions (e.g. Arcanobacterium haemolyticum, Neisseria gonorrhoeae and Fusobacterium spp.) anti-microbial treatment is not beneficial for bacterial causes of tonsillitis except GABHS given that there is not a significant reduction in the rate of complications or in duration of clinical symptoms [7]. Seventy percent of patients presenting with sore throat are treated with antibiotics while only 20–30 % have documented GABHS tonsillitis. Antibiotic treatment may be associated with adverse drug events that range from mild diarrhea to severe allergic reactions. Thus, the utility of these drugs must be determined in order to avoid potential selection of resistant organisms, exposure to adverse events associated with anti-microbial use, and extra cost. Treatment of GABHS is instrumental in preventing the potentially long-term and life-threatening complications associated with this pathogen,

specifically and most importantly, ARF. Treatment also aids in the control of acute signs and symptoms, prevention of suppurative complications, and decreased transmission of GABHS to close contacts [7]. Throat pain and fever self-resolve by 1 week and 3–5 days, respectively, after onset if left untreated; if treated, both symptoms resolve within 3 days [15]. The organisms are eradicated from the pharynx after 10 days of treatment. ARF can be prevented even if therapy is initiated after 9 days of onset [11]. Of note, treatment does not prevent the development of PSGN [7].

The Infectious Disease Society of America (IDSA) recommends testing for GABHS unless a patient presents with symptoms strongly suggestive of a viral etiology; examples of such symptoms include cough, coryza, rhinorrhea, stomatitis or hoarseness. Testing for GABHS is also not indicated in children less than 3 years old. Children in this age group do not present with classic symptoms of GABHS tonsillitis and the incidence of ARF is rare, affecting approximately 0.2 % of children [7, 9]. Testing for GABHS in these children should only be pursued in the presence of other risk factors such as school-aged sibling with documented infection by GABHS, close household contact with diagnosis of symptomatic disease, or with personal or family history of a GABHS complication (ARF) [7].

One of the most commonly used in-office diagnostic tests for GABHS is the Rapid Antigen Detection Test (RADT). This test is done via throat swab of the surface of either tonsil or tonsillar fossa and posterior pharyngeal wall. Swabs of other areas of the oropharynx or oral cavity may lead to false negatives. An enzyme immunoassay test with turn-around times as little as 5 min is then done. It is 95 % specific and 70–90 % sensitive based on the type or manufacturer of RADT used. In the case of a positive RADT, children should be treated with antibiotics. In the case of a negative RADT, the IDSA recommends a throat culture be done during the same office visit. Due to the variability in sensitivity of RDTA based on manufacturer, the high rate of GABHS in children and implications of complications, a throat culture is recommended in order to capture any false negatives. The rapid turnaround time for RADT makes it useful for rapid identification and treatment of GABHS. Rapid treatment decreases the risk of spread of GABHS among close contacts, the amount of time missed from school or work for caregivers, and the duration and severity of acute signs and symptoms of GABHS tonsillitis [7].

Throat cultures are recommended in children in the case of negative RADT prior to the administration of antibiotics in order to avoid false negative results. A single throat swab has a 90–95 % sensitivity rate when done correctly. A throat swab similar to the RADT test is done and is then either processed in an in-office laboratory or sent to a microbiology laboratory. If the cultures are grown in-office, specific instructions must be followed. The swab is processed on a sheep's blood agar plate and incubated at 35–37 °C for 18–24 h. While treatment decisions can be made based on growth patterns at 24 h, a plate with no growth should be re-examined at 48 h to ensure a correct diagnosis. Two major disadvantages of using throat cultures for diagnostic purposes are the training and cost associated with accurate testing as well as delayed diagnosis due to processing time. However, even a delayed diagnosis can be beneficial. Studies show that treatment of GABHS tonsillitis can be delayed up to 9 days from the onset of symptoms and still effectively prevent complications such as ARF [7, 16, 17]. Therefore, regardless of the delay in treatment, throat cultures should be done in children with negative RADT [7].

Other testing options include anti-streptococcal antibody titers; however, these titers are not helpful in the diagnosis of acute GABHS tonsillitis. Rather, they are indicative of previous infection. Antibody titers become positive 3–8 weeks after an acute infection and may persist for up to a year after the resolution of the infection. Thus, they may be useful in determining the etiology of complications [7, 17, 18].

Children with recurrent tonsillitis are sometimes chronic carriers of GABHS with superimposed viral infections. Up to 20 % of asymptomatic school aged children can be carriers of GABHS in the winter and spring months [7, 19]. The IDSA does not recommend identification or treatment of these chronic carriers for several reasons. Distinguishing chronic carriers from recurrent acutely infected children is not possible with the current diagnostic modalities, chronic carriers of GABHS are unlikely

to spread bacteria to close contacts and they are at minimal to no risk of developing complications of GABHS [7]. Moreover, eradication of GABHS from colonized tonsils and adenoids is much more difficult than treatment of acute GABHS tonsillitis. However, certain specific circumstances do call for treatment of chronic carriers of GABHS [7]. These indications, along with treatment options, are discussed below in the section entitled "Treatment of Tonsillitis."

Routine post-treatment RADT or throat cultures to confirm eradication of GABHS are not recommended. Post-treatment testing can be pursued in the case of a patient at high risk for developing ARF (personal or family history of ARF) or recurrent classic symptoms of GABHS tonsillitis shortly after the completion of treatment. Testing or treatment of asymptomatic household contacts is not recommended as it has not been shown to decrease the incidence of subsequent GABHS tonsillitis [7].

#### **Treatment**

Treatment of viral tonsillitis primarily consists of supportive measures including bed rest, hydration, analgesics, and oral hygiene. Most cases of viral tonsillitis self-resolve in 3–4 days. Recommended analgesics include acetaminophen and non-steroidal anti-inflammatory drugs (NSAIDs). Aspirin should be avoided due to the risk of Reye's syndrome, a rare severe illness characterized by rapidly progressive encephalopathy with liver dysfunction and a mortality rate of up to 40 % in children and adolescents suffering from a viral infection, especially varicella-zoster or influenza, in association with the use of salicylates [20]. Other NSAIDs such as ibuprofen or diclofenac can be used. NSAIDs and acetaminophen not only provide pain control but also act to reduce fever. Corticosteroids have proven beneficial in the reduction of the duration and severity of other signs and symptoms, but they do not affect pain levels. Thus, they are not recommended for symptomatic control in acute tonsillitis [7, 21].

Acute bacterial tonsillitis is treated with anti-microbial therapy in addition to the supportive measures listed above. Penicillins target the most commonly implicated pathogen, GABHS. They are narrow spectrum drugs with the greatest safety profile and provide the highest efficacy at a lower cost than other alternatives. Furthermore, there have been no documented cases of penicillin resistant GABHS. A ten-day course of oral penicillin or amoxicillin or a one-time dose of intramuscular benzathine penicillin G is the treatment of choice. An amoxicillin suspension is preferred for younger children due to once a day dosing and better taste that facilitates improved compliance. While a clinical response should be achieved within 24–48 h of beginning antibiotic therapy, a 10 day course of antibiotics has been shown to achieve the maximum rates of pharyngeal eradication of bacteria [7].

Patients with previous non-anaphylactic allergic reactions to penicillin can be treated with first generation cephalosporins for 10 days. Narrow spectrum first generation cephalosporins such as cefadroxil and cephalexin are preferred over broad spectrum cephalosporins such as cefaclor, cefuroxime, cefixime, cefdinir, and cefpodoxime. Approximately 10 % of patients allergic to penicillins will also be allergic to cephalosporins. These patients can be treated with a 10 day course of clindamycin, clarithromycin or a 5 day course of azithromycin. Erythromycin should be reserved for treatment resistant infections due to its high rate of gastrointestinal side effects. Rate of GABHS antibiotic resistance in the United States are approximately 1 % to clindamycin and 5–8 % to macrolides [7, 22].

Ampicillin and oral penicillin-based antibiotics can cause a generalized papular rash in the setting of infectious mononucleosis. Thus, if infectious mononucleosis is suspected, treatment with antibiotics is not recommended.

The IDSA discourages the use of several antibiotics for the treatment of GABHS tonsillitis. Given the high prevalence of resistant strains of GABHS, tetracyclines are not recommended and trimethoprim-sulfamethoxazole does not effectively eradicate GABHS in acute tonsillitis. Newer fluoroquinolones such as levofloxacin and moxifloxacin have proven active against GABHS in vitro but no in vivo efficacy has been documented. Fluoroquinolones are also expensive, broad-spectrum antibiotics

with emerging resistance to *Streptococcus pneumoniae* worldwide and are not recommended in children 18 years of age or younger due to their potential for joint and cartilage toxicity [7, 23, 24].

Recurrent tonsillitis can be treated with penicillin, cephalosporins, macrolides, or clindamycin. If tonsillitis recurs shortly after the completion of a course of antibiotics, intramuscular penicillin should be considered. Alternatively, a 3–6 week course of a penicillin coupled with a beta lactamase inhibitor such as amoxicillin plus clavulanate has been shown to be effective in treatment of recurrent tonsillitis [7].

As discussed previously, routine treatment of chronic carriers of GABHS is not recommended. However, there are a few specific indications for treatment. According to the IDSA and the American Academy of Pediatrics, chronic carriers should be treated in the following circumstances: (1) during a local outbreak of ARF, PSGN, or invasive GABHS infection, (2) outbreaks of GABHS pharyngitis in a closed community, (3) personal or family history of ARF, (4) excessive family or caregiver anxiety about a GABHS infection, or (5) if tonsillectomy is being considered only on the basis of chronic carriage of GABHS. Patients meeting any of the above criteria should be treated with oral clindamycin, oral penicillin plus rifampin, oral amoxicillin plus clavulanate, or intramuscular penicillin plus oral rifampin [7, 11].

Tonsillectomy should be considered for patients suffering from chronic or recurrent tonsillitis whose frequency of infection does not decrease despite appropriate antibiotic treatment and with no other explanation for tonsillitis. Specific indications for tonsillectomy are further discussed in the section entitled "Tonsillectomy."

### Peritonsillar Abscess

Peritonsillar abscess is one of the most common deep space head and neck infections in children. This collection of pus is thought to be formed most commonly as a result of spread of infection from the tonsils or the minor salivary glands of Weber, found on the superior tonsillar pole. The abscess forms deep to the tonsillar capsule between the tonsil, the superior constrictor muscle, and the palatopharyngeus muscle. The most common location is superior and medial to the tonsil; however it can occur lateral to the tonsil or even inferior [3].

# **Epidemiology**

Peritonsillar abscess comprises 30 % of all soft tissue head and neck infections. In patients younger than 20 years old, the incidence of peritonsillar abscess is 0.82–0.94 cases per 10,000 patients. It is most commonly diagnosed in adolescents and young adults, but can occur in any age group with an average age at diagnosis of 13.6 years old [25].

# Microbiology

Peritonsillar abscesses are generally polymicrobial, representing the normal flora of the oral cavity and tonsillar area. Aerobes such as GABHS, *Streptococcus viridans*, *Staphylococcus aureus* and *Haemophilus influenza*, and anaerobes such as *Bacteroides* spp., *Fusobacterium necrophorum* and *Peptostreptococcus* spp. that make up normal oral flora are frequently reported [26]. The most commonly isolated pathogen is GABHS.

### Clinical Presentation

Findings at presentation commonly include fever, odynophagia, trismus, erythema, bulging of the soft palate with deviation of the uvula, unilateral otalgia, drooling, and "hot potato" voice (Fig. 10.3). Trismus is a key finding in patients with peritonsillar abscess and is likely related to peritonsillar inflammation of the pterygoid muscles. Inability to swallow or significant odynophagia usually results in dehydration in younger patients.

## Diagnosis and Imaging

The diagnosis of peritonsillar abscess is typically a clinical one; however, computed tomography (CT) can be utilized in atypical presentation such as when trismus limits the utility of a physical exam, or in uncooperative young children (Fig. 10.4). While the use of intra-oral ultrasound for diagnosis of peritonsillar abscess has been suggested, it is not yet widely used [27]. An elevated white blood cell count and C-reactive protein are commonly found. Throat culture and testing for infectious mononucleosis may be helpful to evaluate for other disease processes.

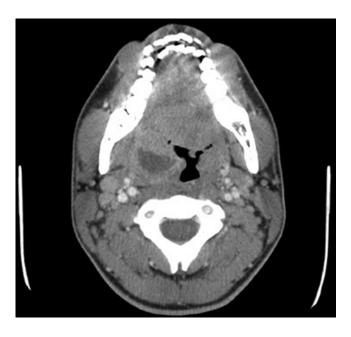
# **Complications**

Complications of a peritonsillar abscess include airway distress, parapharyngeal or retropharyngeal abscess, aspiration pneumonia, and erosion into the carotid sheath. Lemierre's syndrome, a severe disease characterized by thrombophlebitis of the internal jugular vein with metastatic septic emboli as a result of an acute oropharyngeal infection, is another potential complication [28].

**Fig. 10.3** Left peritonsillar abscess. Note the bulging soft palate (Photo courtesy of Dr. Amy L. Richter)



**Fig. 10.4** CT scan of a right peritonsillar abscess



### Management

Definitive treatment consists of incision and drainage or needle aspiration of abscess contents, antibiotics, and elective tonsillectomy after resolution of infection. In rare cases, Quinsy tonsillectomy at the time of infection can be considered. Indications for Quinsy tonsillectomy are discussed below in the section entitled "Quinsy tonsillectomy."

Drainage of the abscess leads to immediate improvement in pain and hastens recovery. Drainage can be done with local anesthesia in the cooperative awake patient or in the operating room. Children are more likely to undergo drainage in the operating room. When performing awake, transoral drainage, a pre-procedure dose of an opioid can be helpful with patient tolerance and the degree of trismus. Needle aspiration or incision and drainage appear to have equal efficacy [29]. Purulent material should be sent for aerobic and anaerobic culture.

Complications of drainage include bleeding, airway obstruction, and possible puncture of the carotid artery. Ten to twenty percent of children undergoing incision and drainage or needle aspiration of a recurrent peritonsillar abscess will require a subsequent tonsillectomy for persistent symptoms or residual abscess contents [3, 30, 31]. Peritonsillar abscesses recur 9–22 % of the time depending on the definition of recurrence which varies by practitioner and system [29, 32].

The use of tonsillectomy as a treatment for peritonsillar abscess remains controversial. It is favored by some practitioners in the setting of recurrent peritonsillar abscesses. A tonsillectomy at the time of infection (Quinsy tonsillectomy) can be considered in rare cases (see section entitled "Quinsy tonsillectomy"). An interval tonsillectomy 4–6 weeks after the resolution of infection may be performed in patients with recurrent tonsillitis.

#### Antimicrobials

Antimicrobials are used as adjunctive therapy for peritonsillar abscess. Combination therapy with penicillin and metronidazole is 98–99 % effective [32]. First generation cephalosporins can be used in patients with a non-anaphylactic penicillin allergy. Patients with previous anaphylactic reactions to

penicillin can be treated with clindamycin, clarithromycin or azithromycin. Supportive therapy with hydration, pain control, and corticosteroids should also be administered [29, 30, 32, 33].

## **Tonsillectomy**

## **Epidemiology**

Tonsillectomy is one of the most common ambulatory surgeries performed in the pediatric population. Recent studies show that 530,000 tonsillectomies are performed per year in children less than 15 years old in the United States [3]. A bimodal distribution of tonsillectomies is observed with the two most frequent age groups being 5–8 years old and 17–21 years old [31]. A tonsillectomy entails the removal of the palatine tonsils with their capsule from the tonsillar fossa.

#### **Indications**

The indications for tonsillectomy include recurrent infection and sleep disordered breathing (SDB). The American Academy of Otolaryngology—Head and Neck Surgery (AAO-HNS) recommends that children that suffered from greater than seven infections in the last year or greater than five infections per year in the last 2 years or greater than three infections per year in the last 3 years and fulfilled one or more of the following criteria should undergo a tonsillectomy with or without an adenoidectomy: temperature greater than 38.3 °C, cervical adenopathy, tonsillar exudate, or positive test for GABHS. Children that do not meet these criteria but have multiple antibiotic allergies or intolerances or suffer from periodic fevers, aphthous stomatitis, pharyngitis and adenitis (PFAPA syndrome) or with a history of peritonsillar abscesses may also be considered candidates for tonsillectomy. A significant amount of missed school or work for patients and/or caregivers due to SDB or recurrent infections should also be considered when creating a treatment plan [3]. The AAO-HNS emphasizes that children that do not meet this criteria may not significantly benefit from undergoing a tonsillectomy. Guidelines suggest close observation and recording of frequency of episodes and symptoms instead of invasive intervention.

The use of tonsillectomy as treatment of PFAPA is still controversial. The AAO-HNS recommends consideration of tonsillectomy in these cases depending on the frequency of symptomatic illness, severity of infection and the patient's response to medical management, commonly steroid therapy [3]. Two randomized control trials showed statistically significant benefit of tonsillectomy to treat PFAPA [34, 35].

Tonsillectomy is recommended in the case of SDB if caused by hypertrophic tonsils and there is significant possibility of improvement of other co-morbidities caused by SDB. Examples of such co-morbidities include growth retardation, poor school performance, and behavioral problems. The decision to undergo surgery must be made in close communication with the child's caregiver(s) [3].

# **Complications**

Complications of a tonsillectomy include throat pain, post-operative nausea and vomiting, dehydration due to delayed oral intake, post-obstructive pulmonary edema, velopharyngeal insufficiency and nasopharyngeal stenosis in the case of concurrent adenoidectomy, hemorrhage and death.

The most common morbidity of tonsillectomy is throat pain. Treatment includes over the counter analgesics and hydration. Commonly used analgesics include acetaminophen and acetaminophen plus hydrocodone. The use of non-steroidal anti-inflammatory drugs (NSAIDs) is generally not recommended due to a potential risk of post-operative bleeding. However, several studies show that NSAIDs do not significantly increase the number of post-tonsillectomy bleeds requiring surgical or non-surgical intervention and that they decrease the incidence of post-operative vomiting [36]. Other studies show that while aspirin is associated with increased risk of post-tonsillectomy bleeding, non-aspirin NSAIDs do not significantly increase this risk with one exception [37]. Intravenous ketorolac has been associated with post-tonsillectomy hemorrhage rates as high as 17 % [3, 38].

Studies show that post-tonsillectomy pain in children is undertreated by caregivers, primarily due to caregiver attempt at balancing pain control with overtreatment [39]. The AAO-HNS guidelines state that no specific medication or dosing interval (as needed versus scheduled) has been proven superior. It is most important that caregivers assess and re-assess a child's pain level even when the child does not spontaneously complain of pain [3].

Post-tonsillectomy hemorrhage is a much less common but the most concerning complication of tonsillectomy. It is the most common complication brought to the attention of medical personnel. Post-tonsillectomy hemorrhage is stratified based on time after surgery in order to help delineate the cause of bleeding. Primary hemorrhage is bleeding occurring within the first 24 h after tonsillectomy and occurs in 0.2–2.2 % of patients. The most common cause is surgical technique or reopening of blood vessels. Secondary hemorrhage is bleeding that occurs more than 24 h after surgery, most commonly on post-operative days 5–10. Secondary hemorrhage is most commonly due to sloughing of the eschar as the tonsillar bed heals and occurs in 0.1–3 % of patients [40]. The incidence of post-tonsillectomy hemorrhage has been noted to range significantly due to the definition of clinically significant bleeding and the consideration of primary or secondary hemorrhage only. The use of specific surgical techniques to reduce the incidence of post-tonsillectomy hemorrhage is still under investigation [40]. Bleeding following a tonsillectomy requires clinical evaluation and profuse bleeding may be treated with cauterization, inpatient observation, transfusion, or surgery.

The rate of mortality associated with tonsillectomy has been cited as less than 1 in 20,000 [41]. The most common causes of tonsillectomy-associated death include bleeding and opioid related respiratory depression [31].

# Quinsy Tonsillectomy

A Quinsy tonsillectomy is done at the time of tonsillar infection. While tonsillectomy is generally recommended after the resolution of infection, it can be considered at the time of infection in a select few cases. Indications include peritonsillar abscess in younger children; recurrent or unresponsive cases of peritonsillar abscess or in the setting of previous history of deep neck abscess, and peritonsillar abscess presenting with severe airway compromise [42]. Due to the inflammation in an infected field, the risk of intraoperative, and potentially post-operative, bleeding is increased. Thus, candidates for Quinsy tonsillectomy must be carefully and selectively chosen.

### Conclusion

Tonsillitis and peritonsillar abscess are frequently seen in the pediatric population. Antimicrobial management should be directed by RADT or positive throat cultures. In the case of peritonsillar abscess, acute drainage of the pus is the definitive treatment in addition to the use of adjunctive antimicrobial therapy. Quinsy tonsillectomy can lead to bleeding complications and is typically reserved for rare cases.

### References

- DelGaudio J, Chen A. Oral cavity and oropharynx. In: Wood W, Staley C, Skandalakis J, editors. Anatomic basis
  of tumor surgery. 2nd ed. Heidelberg: Springer; 2010.
- Drake R, Vogl W, Mitchell A. Gray's anatomy for students. 2nd ed. Philadelphia, PA: Churchill Livingstone/ Elsevier; 2010.
- 3. 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.
- National Hospital Ambulatory Medical Care Survey: 2011 Emergency Department Summary Tables. 2011. http:// www.cdc.gov/nchs/ahcd.htm. Accessed 12 Oct 2014.
- Nash DR, Harman J, Wald ER, Kelleher KJ. Antibiotic prescribing by primary care physicians for children with upper respiratory tract infections. Arch Pediatr Adolesc Med. 2002;156(11):1114–9.
- Arnold J, Nizet V. Pharyngitis. In: Long S, Pickering L, Prober C, editors. Principles and practice of pediatric infectious diseases. Philadelphia, PA: Elsevier Saunders; 2012. p. 200.
- Shulman ST, Bisno AL, Clegg HW, Gerber MA, Kaplan EL, Lee G, et al. Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. Clin Infect Dis. 2012;55(10):1279–82.
- 8. Pfoh E, Wessels MR, Goldmann D, Lee GM. Burden and economic cost of group A streptococcal pharyngitis. Pediatrics. 2008;121(2):229–34.
- Hardy AM. Incidence and impact of selected infectious diseases in childhood. Vital Health Stat. 1991; 10(180):1–22.
- Jensen A, Hagelskjaer Kristensen L, Prag J. Detection of Fusobacterium necrophorum subsp. funduliforme in tonsillitis in young adults by real-time PCR. Clin Microbiol Infect. 2007;13(7):695–701.
- 11. American Academy of Pediatrics. In: Pickering L, Baker C, Kimberlin D, Long S, editors. Red Book: 2012 Report of the Committee on Infectious Diseases. Elk Grove Village, IL: American Academy of Pediatrics; 2012. p. 668.
- 12. Breda L, Nozzi M, De Sanctis S, Chiarelli F. Laboratory tests in the diagnosis and follow-up of pediatric rheumatic diseases: an update. Semin Arthritis Rheum. 2010;40(1):53–72.
- 13. Luzuriaga K, Sullivan JL. Infectious mononucleosis. N Engl J Med. 2010;362(21):1993–2000.
- 14. Pitetti RD, Laus S, Wadowsky RM. Clinical evaluation of a quantitative real time polymerase chain reaction assay for diagnosis of primary Epstein-Barr virus infection in children. Pediatr Infect Dis J. 2003;22(8):736–9.
- Spinks A, Glasziou PP, Del Mar CB. Antibiotics for sore throat. Cochrane Database Syst Rev. 2013;11, CD000023.
- 16. Catanzaro FJ, Stetson CA, Morris AJ, Chamovitz R, Rammelkamp Jr CH, Stolzer BL, et al. The role of the streptococcus in the pathogenesis of rheumatic fever. Am J Med. 1954;17(6):749–56.
- 17. Gerber MA, Baltimore RS, Eaton CB, Gewitz M, Rowley AH, Shulman ST, 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.
- 18. Johnson DR, Kurlan R, Leckman J, Kaplan EL. The human immune response to streptococcal extracellular antigens: clinical, diagnostic, and potential pathogenetic implications. Clin Infect Dis. 2010;50(4):481–90.
- 19. Martin JM, Green M, Barbadora KA, Wald ER. Group A streptococci among school-aged children: clinical characteristics and the carrier state. Pediatrics. 2004;114(5):1212–9.
- Auriel E, Regev K, Korczyn AD. Nonsteroidal anti-inflammatory drugs exposure and the central nervous system. Handb Clin Neurol. 2014;119:577–84.
- 21. Bulloch B, Kabani A, Tenenbein M. Oral dexamethasone for the treatment of pain in children with acute pharyngitis: a randomized, double-blind, placebo-controlled trial. Ann Emerg Med. 2003;41(5):601–8.
- Tanz RR, Shulman ST, Shortridge VD, Kabat W, Kabat K, Cederlund E, et al. Community-based surveillance in the united states of macrolide-resistant pediatric pharyngeal group A streptococci during 3 respiratory disease seasons. Clin Infect Dis. 2004;39(12):1794

  –801.
- 23. Bradley JS, Jackson MA. The use of systemic and topical fluoroquinolones. Pediatrics. 2011;128(4):e1034-45.
- 24. Leibovitz E. The use of fluoroquinolones in children. Curr Opin Pediatr. 2006;18(1):64-70.
- Novis SJ, Pritchett CV, Thorne MC, Sun GH. Pediatric deep space neck infections in U.S. children, 2000–2009. Int J Pediatr Otorhinolaryngol. 2014;78(5):832–6.
- Brook I. Microbiology and management of peritonsillar, retropharyngeal, and parapharyngeal abscesses. J Oral Maxillofac Surg. 2004;62(12):1545–50.
- 27. Schraff S, McGinn JD, Derkay CS. Peritonsillar abscess in children: a 10-year review of diagnosis and management. Int J Pediatr Otorhinolaryngol. 2001;57(3):213–8.
- 28. Syed MI, Baring D, Addidle M, Murray C, Adams C. Lemierre syndrome: two cases and a review. Laryngoscope. 2007;117(9):1605–10.

29. Johnson RF, Stewart MG, Wright CC. An evidence-based review of the treatment of peritonsillar abscess. Otolaryngol Head Neck Surg. 2003;128(3):332–43.

- 30. Herzon FS, Martin AD. Medical and surgical treatment of peritonsillar, retropharyngeal, and parapharyngeal abscesses. Curr Infect Dis Rep. 2006;8(3):196–202.
- 31. Herzon FS, Harris P. Mosher Award thesis. Peritonsillar abscess: incidence, current management practices, and a proposal for treatment guidelines. Laryngoscope. 1995;105(8 Pt 3 Suppl 74):1–17.
- 32. Powell J, Wilson JA. An evidence-based review of peritonsillar abscess. Clin Otolaryngol. 2012;37(2):136-45.
- 33. Baldassari C, Shah RK. Pediatric peritonsillar abscess: an overview. Infect Disord Drug Targets. 2012;12(4): 277–80.
- 34. Garavello W, Romagnoli M, Gaini RM. Effectiveness of adenotonsillectomy in PFAPA syndrome: a randomized study. J Pediatr. 2009;155(2):250–3.
- 35. Renko M, Salo E, Putto-Laurila A, Saxen H, Mattila PS, Luotonen J, et al. A randomized, controlled trial of tonsillectomy in periodic fever, aphthous stomatitis, pharyngitis, and adenitis syndrome. J Pediatr. 2007;151(3):289–92.
- 36. Lewis SR, Nicholson A, Cardwell ME, Siviter G, Smith AF. Nonsteroidal anti-inflammatory drugs and perioperative bleeding in paediatric tonsillectomy. Cochrane Database Syst Rev. 2013;7, CD003591.
- 37. Krishna S, Hughes LF, Lin SY. Postoperative hemorrhage with nonsteroidal anti-inflammatory drug use after tonsillectomy: a meta-analysis. Arch Otolaryngol Head Neck Surg. 2003;129(10):1086–9.
- 38. Judkins JH, Dray TG, Hubbell RN. Intraoperative ketorolac and posttonsillectomy bleeding. Arch Otolaryngol Head Neck Surg. 1996;122(9):937–40.
- 39. Barraclough J, Anari S. Tonsillectomy for recurrent sore throats in children: indications, outcomes, and efficacy. Otolaryngol Head Neck Surg. 2014;150(5):722–9.
- 40. Windfuhr JP, Chen YS, Remmert S. Hemorrhage following tonsillectomy and adenoidectomy in 15,218 patients. Otolaryngol Head Neck Surg. 2005;132(2):281–6.
- 41. Shay S, Shapiro NL, Bhattacharyya N. Revisit rates and diagnoses following pediatric tonsillectomy in a large multistate population. Laryngoscope. 2015;125(2):457–61.
- 42. Page C, Chassery G, Boute P, Obongo R, Strunski V. Immediate tonsillectomy: indications for use as first-line surgical management of peritonsillar abscess (quinsy) and parapharyngeal abscess. J Laryngol Otol. 2010;124(10):1085–90.