# Tonsillitis and its complications

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Peritonsillar cellulitis and abscess are the most common deep infections of the head and neck in children. Inadequate treatment of these infections may have devastating consequences.

# What is the palatine tonsil?

The palatine tonsil represents the largest accumulation of lymphoid tissue in the head and neck region. Each tonsil has a compact body with a definite thin capsule on its deep surface. A stratified squamous epithelium lines the outer surface of the tonsil and invaginates deeply into the lymphoid tissue to form multiple crypts. Figure 1 demonstrates normal tonsils.

The tonsillar fossa is composed of three muscles: the palatoglossus muscle, the palatopharyngeal muscle, and the superior constrictor muscle. The palatoglossus muscle forms the anterior pillar and the palatopharyngeal muscle forms the posterior pillar. The tonsillar bed is formed by the superior constrictor muscle of the

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Figure 1. Photograph of oropharynx. The tonsils are normal and the uvula is in the midline.

pharynx. The arterial blood supply of the tonsil enters primarily at the lower pole and is derived from the tonsillar branch of the dorsal lingual artery, the ascending palatine artery and the tonsillar branch of the facial artery. The ascending pharyngeal artery and



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the lesser palatine artery also contribute to the vascular supply at the upper pole. Venous blood drains through the peritonsillar plexus around the capsule. The plexus then drains into the lingual and pharyngeal veins, which in turn drain into the internal jugular vein.

The nerve supply of the tonsillar region is through the tonsillar branches of the glossopharyngeal nerve and the descending branches of the lesser palatine nerves. The cause of referred otalgia with tonsillitis is through the tympanic branch of the glossopharyngeal nerve. The lymphatic drainage courses through the upper deep cervical lymph nodes.

## What function do tonsils serve?

The tonsils are predominantly β-cell organs with β-lymphocytes comprising 50% to 65% of all tonsillar lymphocytes. T-cell lymphocytes comprise approximately 40% of tonsillar lymphocytes and 3% are mature plasma cells. Tonsils are involved in inducing secretory immunity and regulating immunoglobulin production. The tonsils are favourably located to mediate immunologic protection of the upper aerodigestive tract as they are exposed to airborne antigens. Moreover, there

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are 10 to 30 crypts in each tonsil that are ideally suited to trapping foreign material and transporting it to the lymphoid follicles. The proliferation of  $\beta$  cells in the germinal centres of the tonsils in response to antigenic signals is one of the most important tonsillar functions.

The human tonsils are immunologically most active between the ages of four and 10. Involution of the tonsils begins after puberty, resulting in a decrease in the  $\beta$ -cell population and a relative increase in the ratio of T- to  $\beta$ -cells.

Although the overall immunoglobulin production is reduced, there is still considerable β-cell activity if seen in clinically healthy tonsils. The immunologic consequences of tonsillectomy are unclear. It is evident, however, that tonsillectomy does not result in a major immunologic deficiency.

Pathogens		Clinical Appearance of Tonsils
Viruses	Rhinovirus, adenovirus, influenza virus, parainfluenza virus, etc.	Enlarged, erythematous.
	Coxsackie virus (herpangina).	Aphthous-like ulcers on tonsillar pillars.
	Epstein-Barr virus (mononucleosis syndrome).	Very large, swollen, and dirty-grey appearance.
Bacteria Aerobic	Streptococcus pyogenes and other streptococcal species.	Enlarged, erythematous, with yellowish-white spots. May have membrane or purulent exudate.
	Neisseria gonorrhoeae.	Acute purulent exudates.
	Corynebacterium diphtheriae.	Exudative pharyngotonsillitis with thick pharyngeal membrane.
Anaerobic	Bacteroides species.	Enlarged, erythematous.
Yeast	Candida species.	White plaques with raw undersurface.
Spirochetes	Treponema pallidum (syphilis).	Oral chancres of the lip, tongue, tonsil and palate. Superficial greyish patches of mucus membrane with reddish border.
	Spirochaete denticolata and treponema vincentii (Vincent's angina).	Membrane on tonsil with underlying ulcer.

# What is the microbiology of tonsillitis?

Many organisms can induce inflammation of the tonsils. These include bacteria, viruses, yeasts, and parasites. Several pathogens and their clinical hallmarks are summarised in Table 1. Some of the infectious organisms are part of the normal oropharyngeal flora whereas others are external pathogens. Because the oropharynx is colonised by many organisms, most infections are polymicrobial. These organisms work synergistically and can be demonstrated in mixed aerobic and anaerobic infections.

Group A Streptococcus is the most common bacterial cause of acute pharyngitis. This pathogen has importance from a public health standpoint in that it is a potential precursor to two serious sequelae: acute rheumatic fever and post-streptococcal glomerulonephritis. For this reason, most authorities recommend that



Figure 2. Left peritonsillar cellulitis.



Figure 3. Left peritonsillar abscess. Notice the shift of the uvula to the right side and the redness of the left tonsillar pillars.

the diagnosis of Group A Hemolytic Streptococcal Pharyngitis be verified by microbiology tests (swabbing) in patients who appear, on the basis of clinical and epidemiological evidence, to have this illness. A full course of antibiotic therapy is recommended in these patients.

# What are the complications of tonsillitis?

The complications of tonsillitis may be classified into suppurative and nonsuppurative complications. The nonsuppurative complications include scarlet fever, acute rheumatic fever, and post-strepto-coccal glomerulonephritis. Suppurative complications include peritonsillar, parapharyngeal and retropharyngeal abscess formation.

Scarlet fever is secondary to acute streptococcal tonsillitis or pharyngitis with production of endotoxins by the bacteria. Clinical signs include an erythematous rash, severe lymphadenopathy, fever,

tachycardia, and a yellow exudate overlying erythematous tonsils. Acute rheumatic fever is a syndrome that follows Group A Streptococcal Pharyngitis for one

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to four weeks. Certain proteins found in heart muscle appear to be antigenetically similar to protein found on the streptococcus. This is believed to be the method of infection of cardiac tissue. Post-streptococcal glomerulonephritis may be seen after both pharyngeal and skin infections. The typical patient develops an acute nephritic syndrome one to two weeks after a strepto-

coccal infection. The infection is secondary to the presence of a common antigen in the glomerulus and in the streptococcus. Antibiotic therapy may not necessarily alter the natural history of glomerulonephritis. A tonsillectomy may be necessary to eliminate the source of infection.

Peritonsillar abscess most commonly occurs in patients with recurrent tonsillitis or in those with chronic tonsillitis who have been inadequately treated. The spread of infection is from the superior pole of the tonsil with pus formation between the tonsil bed and the tonsillar capsule. Figures 2 and 3 demonstrate left

peritonsillar cellulitis and abscess respectively. This infection usually occurs unilaterally and the pain is quite severe. Drooling is caused by odynophagia and dysphagia. Trismus is frequently present as a result of irritation of the pterygoid musculature by the pus and inflammation. There is gross unilateral swelling of the palate and anterior pillar with displacement of the tonsil downward and medially with deviation of the uvula toward the opposite side. Cultures of peritonsillar abscess usually show a polymicrobial infection, both aerobic and anaerobic.

An abscess in the parapharyngeal space can develop if infection or pus drains from either the tonsils or from a peri-

Suspicion of abscess include a history of recurrent tonsillitis, inadequate antibiotic treatment and a long duration of illness.

tonsillar abscess through the superior constrictor muscle. The abscess is located between the superior constrictor muscle and the deep cervical fascia and causes displacement of the tonsil on the lateral pharyngeal wall toward the midline. Involvement of the adjacent pterygoid and paraspinal muscles with the

inflammatory process results in trismus and a stiff neck. Progression of the infection of the abscess may spread down the carotid sheath into the mediastinum. As with most soft-tissue infections of the neck, lateral pharyngeal space infections are polymicrobial and reflect the oropharyngeal flora.

A retropharyngeal abscess may also result from a peritonsillar abscess. The source of the abscess is a chain of lymph nodes on either side of the midline in the retropharyngeal space. These lymph nodes receive drainage from the nose, paranasal sinuses, pharynx and Eustachian tube. Children usually present with irritability, fever, dysphagia,

muffled speech, noisy breathing, stiff neck, and cervical lymphadenopathy.

#### Table 2

# Current indications for tonsillectomy (1) with or without adenoidectomy (2)

#### Infection

- Recurrent tonsillitis (more than seven per year or five per year for two years or three per year for three years). (1)
- 2. Persistent, chronic tonsillitis. (1)
- Recurrent otitis media unresponsive to medical or previous placement of PETubes. (2)
- 4. Chronic/recurrent nasopharyngitis. (2)
- 5. Chronic/recurrent sinusitis. (2)

#### **Obstruction**

- Hypertrophy with obstruction unresponsive to antibiotics with or without obstructive apnea, severe dysphagia, and failure to thrive.
  - -If adenoids enlarged only. (2)
  - -If tonsils enlarged only. (1)
  - -If tonsils and adenoides are enlarged. (1,2)
- Nasal obstruction with speech abnormalities, orodental abnormalities.
  (1)

#### Miscellaneous

- Recurrent peritonsillar abscess or peritonsillar abscess with previous history of recurrent or persistent tonsillitis. (1)
- 2. Unilateral tonsillar hypertrophy. (1)
- 3. Hemorrhagic tonsillitis. (1)
- 4. Chronic tonsillolithiasis. (1)



Figure 4. CT showing right peritonsillar abscess.

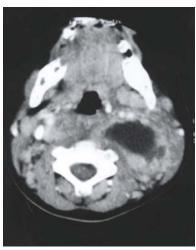


Figure 5. Left parapharyngeal abscess.

# How do I manage peritonsillar infections?

Cellulitis should be differentiated from abscess in the management of peritonsillar infections. Some abscesses may be clinically obvious whereas others are less obvious. Clues in the history that increase the suspicion of abscess include a history of recurrent tonsillitis, inadequate antibiotic treatment and a long duration of illness. In a cooperative child, needle aspiration may be used to obtain a test aspirate and identify the site of the abscess. A computed tomography scan is required to confirm a diagnosis of parapharyngeal abscess (Figures 4 and 5).

Peritonsillar cellulitis is treated with oral or intravenous antibiotics depending on the severity of the infection. Clindamycin is especially useful against polymicrobial-mixed pathogen infections prevalent in tonsillitis and deep neck space abscesses of oral origin. Clindamycin is effective against all streptococci, most pneumococci, and most penicillin-resistant (but not methicillin-resistant) staphylococci. Clindamycin is superior to penicillin for eradication of streptococci in tonsillo-pharyngitis, probably because the polymicrobial flora (producing beta lactamases) renders penicillin ineffective.

The use of needle aspiration and incision and drainage are the mainstay of treatment of peritonsillar abscess in the cooperative patient. A tonsillectomy is then performed four to twelve weeks later in the patient with a history of recurrent tonsillitis. In the uncooperative child with a prior history of

recurrent peritonsillar abscess or recurrent tonsillitis severe enough to warrant tonsillectomy, the operation is indicated (Table 2). Quinsy tonsillectomy is particularly favoured in children because they are likely to experience further episodes of tonsillitis. Needle aspiration or incision and drainage with a child under local anaesthesia is often difficult or impossible. An algorithm of management of peritonsillar infections is illustrated in Figure 6.

Initial management of parapharyngeal and retropharyngeal abscesses should include aggressive antibiotic therapy, fluid replacement, and close observation. This is promptly followed by surgical intervention that is required to bring about

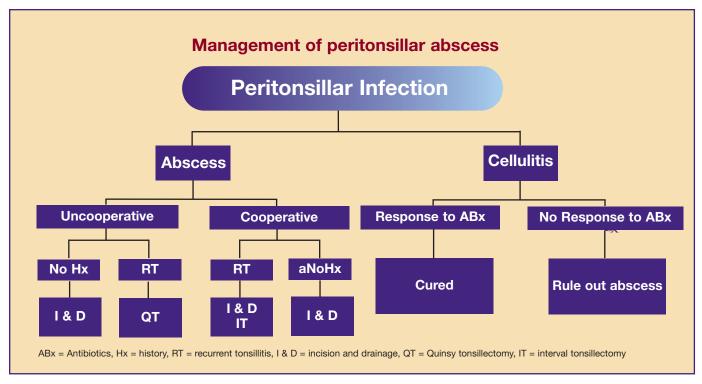


Figure 6. Management algorithm for peritonsillar abscess.

resolution of these infections. Transoral and external approaches may be used to drain these collections. Gram stain, culture, and antimicrobial sensitivities should be obtained on the purulent material.  $\mathbb{D}_{k}$ 

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