Pharyngitis

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Nelson Essentials of Pediatrics, Chapter 103, 409-411

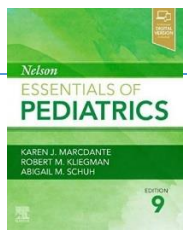
Etiology

Many infectious agents can cause pharyngitis (Table 103.1), viruses being the most common. **Group A streptococcus** *(Streptococcus pyogenes)* is the most common important bacterial cause of pharyngitis. Other bacterial organisms less often associated with pharyngitis include groups C and G streptococci (also β-hemolytic) and rarely *Arcanobacterium haemolyticum* (β-hemolytic, gram-positive rod) and *Francisella tularensis* (gram-negative coccobacillus and cause of tularemia). *Chlamydophila pneumonia* is associated with lower respiratory disease but can also cause sore throat. *Mycoplasma pneumoniae* is associated with atypical pneumonia and may cause mild pharyngitis without distinguishing clinical manifestations. *Neisseria gonorrhoeae* should be considered in a sexually active adolescent or adult with pharyngitis. Other bacteria, including *Staphylococcus aureus, Haemophilus influenzae* , and *S. pneumoniae* , are cultured frequently from the throats of children, but their role in causing pharyngitis is unclear.

Table 103.1 Major Microbial Causes of Acute Pharyngitis
Modified from Hayden GF, Hendley JO, Gwaltney JM Jr. Management of the ambulatory patient with a sore throat. Curr Clin Top Infect Dis. 1988;9:62–75.

Agent	Syndrome or disease	Estimated proportion of all pharyngitis (%)
BACTERIAL		
Group A streptococcus *(Streptococcus pyogenes)*	Pharyngitis, tonsillitis, scarlet fever	15–30
Group C and G streptococcus	Pharyngitis, tonsillitis	1–5
Arcanobacterium haemolyticum	Pharyngitis (scarlet fever–like syndrome)	0.5–3
Fusobacterium necrophorum	Lemierre syndrome	Unknown
Mycoplasma pneumoniae	Pharyngitis, pneumonia	Unknown
Other (e.g., *Neisseria gonorrhoeae, Corynebacterium diphtheriae, Francisella tularensis)*	Pharyngitis, laryngitis, tularemia	<5
VIRAL		
Rhinoviruses (>100 types)	URI	20
Coronaviruses (>4 types, including COVID-19)	URI, acute respiratory distress	>5
Adenoviruses (types 3, 4, 7, 14, 21)	Pharyngoconjunctival fever, acute respiratory disease	5
Herpes simplex viruses (types 1 and 2)	Gingivitis, stomatitis, pharyngitis	4
Parainfluenza viruses (types 1–4)	URI, croup	2
Influenza viruses (types A and B)	Influenza	2
Epstein-Barr virus	Mononucleosis	Unknown
Cytomegalovirus	Mononucleosis	Unknown
Coxsackie virus	Herpangina, hand, foot and mouth disease	Unknown
Unknown		40

URI , Upper respiratory tract infection.



Many viruses cause acute pharyngitis. Some viruses, such as adenoviruses, are more likely than others to cause pharyngitis as a prominent symptom, whereas other viruses, such as rhinoviruses, are more likely to cause pharyngitis as a minor part of an illness that primarily features other symptoms, such as rhinorrhea or cough. Epstein-Barr virus (EBV), enteroviruses (herpangina), herpes simplex virus (HSV), and primary HIV infection can also produce pharyngitis.

Epidemiology

Sore throat is the primary symptom in approximately one third of upper respiratory tract illnesses. Streptococcal pharyngitis is relatively uncommon before 2–3 years of age, but the incidence increases in young school-age children and then declines in late adolescence and adulthood. Streptococcal pharyngitis occurs throughout the year in temperate climates. The illness often spreads to siblings and classmates. Viral infections generally spread via close contact with an infected person and peak during winter and spring. EBV or cytomegalovirus-related **mononucleosis** (see Chapter 99) can feature pharyngitis as a prominent symptom and is most common in adolescents and young adults.

Clinical Manifestations

Pharyngeal inflammation causes cough, sore throat, dysphagia, and fever. If involvement of the tonsils is prominent, the term **tonsillitis** or **tonsillopharyngitis** is often used.

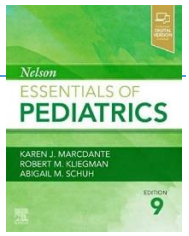
The onset of streptococcal pharyngitis is often rapid and associated with prominent sore throat and moderate to high fever. Headache, nausea, vomiting, and abdominal pain are frequent. In a typical, florid case, the pharynx is distinctly red. The tonsils are enlarged and may be covered with a yellow, blood-tinged exudate. There may be petechiae on the soft palate and posterior pharynx, and the uvula may be red, stippled, and swollen. Anterior cervical lymph nodes are enlarged and tender to touch. However, some children present with only mild pharyngeal erythema without tonsillar exudate or cervical lymphadenitis. Compared with classic streptococcal pharyngitis, the onset of viral pharyngitis is typically more gradual, and symptoms more often include rhinorrhea, cough, and diarrhea. Conjunctivitis, coryza, or oral ulcerations also suggest a viral etiology. The diagnosis of streptococcal pharyngitis cannot be made on clinical features alone.

In addition to sore throat and fever, some patients with streptococcal pharyngitis exhibit the stigmata of **scarlet fever** : circumoral pallor, strawberry tongue, and a fine diffuse erythematous macular-papular rash. The tongue initially has a white coating, but red and edematous lingual papillae later project through this coating, producing a **white strawberry tongue** . When the white coating peels off, the resulting **red strawberry tongue** is beefy red with prominent papillae. Patients infected with *A. haemolyticum* may present with similar findings.

Gingivostomatitis is characteristic of HSV type 1. **Herpangina** is an enteroviral infection with sudden onset of high fever, vomiting, headache, malaise, myalgia, poor intake, drooling, sore throat, and dysphagia. The oral lesions of herpangina may be nonspecific, but classically there are one or more small, tender, papular, or pinpoint vesicular lesions on an erythematous base scattered over the soft palate, uvula, and tongue. These vesicles enlarge from 1–4 mm over 3–4 days, rupture, and produce small, punched-out ulcers that persist for several days. Similar lesions may be seen in hand-foot-and-mouth disease caused by coxsackieviruses.

Laboratory Evaluation

The principal challenge is to distinguish pharyngitis caused by group A streptococcus from pharyngitis caused by viral organisms. A rapid streptococcal antigen test, a throat culture, or both should be performed to improve diagnostic precision and to identify children most likely to benefit from antibiotic therapy. Antigen-based rapid diagnostic tests for streptococcal pharyngitis have excellent specificity of 95–99%. However, the sensitivity of these rapid tests varies, and negative rapid tests should be confirmed by a throat culture or polymerase chain reaction (PCR) as these are the definitive



diagnostic tests to establish the presence of streptococcal pharyngitis. However, as many as 20% of positive cultures in children represent chronic **streptococcal carriage** and not acute pharyngitis.

The predictive values of white blood cell count, erythrocyte sedimentation rate, and C-reactive protein are not sufficient to distinguish streptococcal from nonstreptococcal pharyngitis, and these tests are not routinely recommended. The white blood cell count in patients with infectious mononucleosis usually shows a predominance of atypical lymphocytes.

Differential Diagnosis

The differential diagnosis of infectious pharyngitis includes other local infections of the oral cavity, retropharyngeal abscesses (*S. aureus* , streptococci, anaerobes), diphtheria (if unimmunized), peritonsillar abscesses (streptococci, anaerobes, or, rarely, *S. aureus*), and epiglottitis. In addition, neutropenic mucositis (leukemia, aplastic anemia), thrush (candidiasis secondary to T-cell immune deficiency), autoimmune ulceration (systemic lupus erythematosus, Behçet disease), and Kawasaki disease may cause pharyngitis. Pharyngitis can be a feature of the acute retrovirus syndrome associated with primary HIV infection (see Chapter 125).

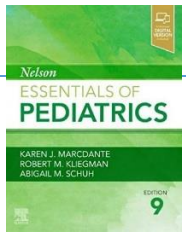
Vincent angina or **trench mouth** refers to a virulent form of anaerobic gingivitis and pharyngitis; gray pseudomembranes are found on the tonsils. **Lemierre syndrome** is acute pharyngitis complicated by suppurative thrombophlebitis of the internal jugular vein and septic pulmonary emboli. It primarily occurs in adolescents and is caused by *Fusobacter-ium necrophorum* (obligate anaerobic gram-negative bacilli). **Ludwig angina** is a mixed anaerobic bacterial cellulitis of the submandibular and sublingual regions. It is typically due to spreading from an abscess of the second or third mandibular molar. It also has been associated with tongue piercing. A propensity for rapid spread, glottic and lingual swelling, and consequent airway obstruction makes prompt intervention imperative.

A syndrome of **periodic fever** , **aphthous stomatitis** , **pharyngitis** , **and cervical adenitis (PFAPA)** is a rare cause of recurrent fever in children. The fevers begin at a young age (usually <5 years). Episodes last approximately 5 days, with a mean of 28 days between episodes. Episodes are shorter with oral prednisone and do not respond to nonsteroidal antiinflammatory drugs or antibiotics. Long-term sequelae do not develop.

Treatment

Even if untreated, most episodes of streptococcal pharyngitis resolve uneventfully over a few days. Early antimicrobial therapy accelerates clinical recovery by 24–48 hours. The major benefit of antimicrobial therapy is the prevention of acute rheumatic fever (see Chapter 146). Because the latent (incubation) period of **acute rheumatic fever** is relatively long (1–3 weeks), treatment instituted within 9 days of illness onset is virtually 100% successful in preventing rheumatic fever. Treatment begun after this period, although less than 100% successful, has some preventive value. Therefore antibiotic therapy should be started promptly in children with a positive rapid antigen test, throat culture, or PCR for group A streptococcus or a diagnosis of scarlet fever. In addition, a child who has symptomatic pharyngitis and a history of rheumatic fever, or a recent family history of rheumatic fever, or whose sibling has documented streptococcal infection should be treated.

A variety of antimicrobial agents can be used to treat streptococcal pharyngitis (Table 103.2). Penicillin or amoxicillin remain first-line therapy. Ten days of treatment is recommended to prevent rheumatic fever. Intramuscular penicillin G can be used for children who cannot tolerate oral therapy. Cephalosporins have superior pharyngeal bacterial eradication rates compared to penicillin; however, they are only recommended as an alternative option. Five days of azithromycin is also an alternate treatment option for group A streptococcal pharyngitis, although rates of resistance to azithromycin are higher than those to penicillins. Test of cure for patients who respond clinically to treatment is not required. Children with recurrent episodes of group A streptococcal pharyngitis pose a problem, but true treatment failure is rare. Recurrent infection may represent nonadherence, chronic streptococcal carriage in the setting of a viral pharyngitis, or reinfection.



A second course of treatment can be administered. Amoxicillin-clavulanate or clindamycin can be effective for eliminating streptococcal carriage.

Table 103.2 Antimicrobial Treatment of Group A Streptococcal Pharyngitis

Oral penicillin V (2–3 times daily for 10 days) 250–500 mg/dose × 10 days
Intramuscular benzathine penicillin G–for children ≤27 kg: 600,000 U; for larger children and adults: 1.2 million U, given as a single dose
Oral amoxicillin as a single daily dose (50 mg/kg; maximum, 1000–1200 mg) × 10 days
For persons allergic to penicillin:
Cephalexin 25 mg/kg/dose BID, maximum dose 500 mg/dose × 10 days
Clindamycin 6–7 mg/kg/dose TID, maximum dose 300 mg/dose × 10 days
For persons allergic to β-lactams:
Azithromycin, children: 12 mg/kg (maximum: 500 mg/dose) on day 1 followed by 6 mg/kg/dose (maximum: 250 mg/dose) OD on days 2 through 5

BID , Twice daily; *OD* , once daily; *TID* , three times daily.

Specific antiviral therapy is unavailable for most cases of viral pharyngitis. Patients with primary herpetic gingivostomatitis benefit from early treatment with oral acyclovir.

Complications and Prognosis

Pharyngitis caused by streptococci or respiratory viruses usually resolves completely. The complications of group A streptococcal pharyngitis include local suppurative complications, such as parapharyngeal abscess and other infections of the deep fascial spaces of the neck, and nonsuppurative complications, such as acute rheumatic fever and acute postinfectious glomerulonephritis. Viral upper respiratory tract infections, including infections caused by influenza A, adenoviruses, parainfluenza type 3, and rhinoviruses, may predispose the patient to bacterial middle ear infections.

Prevention

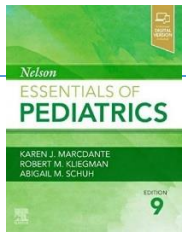
Antimicrobial prophylaxis with daily oral penicillin V prevents recurrent streptococcal infections and is recommended following an episode of acute rheumatic fever to prevent recurrences.

Pearls for Practitioners

• Viruses are the most common cause of pharyngitis. However, if group A streptococcus is isolated by rapid antigen test or culture, it should be treated in order to prevent acute rheumatic fever. Penicillin or amoxicillin are first-line therapies.

• Approximately 20% of school-age children are colonized with group A streptococcus without symptoms.

• A major reason to treat streptococcal pharyngitis is to prevent rheumatic fever. Streptococcal pharyngitis is relatively uncommon in young children under 3 years of age.



Sinusitis

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Nelson Essentials of Pediatrics, Chapter 104, 411-412

Etiology

Sinusitis is a suppurative infection of the paranasal sinuses and most commonly occurs as a complication of an upper respiratory tract infection (URI). Only the maxillary and ethmoid sinuses are present at birth, whereas the sphenoid sinuses are present by 5 years of age. Frontal sinuses begin to develop at 7 years of age and are not completely developed until adolescence. The ostia draining the sinuses are narrow (1–3 mm) and drain into the middle meatus in the **ostiomeatal complex** . The mucociliary system maintains the sinuses as normally sterile. Indwelling nasogastric and nasotracheal tubes predispose to nosocomial sinusitis.

Obstruction to mucociliary flow, such as mucosal edema resulting from a URI, impedes sinus drainage and predisposes to bacterial proliferation. The bacterial causes of most cases of acute sinusitis are *Streptococcus pneumoniae* , non-typable *Haemophilus influenzae* , and *Moraxella catarrhalis. Staphylococcus aureus* and anaerobes emerge as important pathogens in subacute and chronic sinusitis. Fungal sinusitis can occur in immunocompromised patients, especially those with prolonged and/or profound neutropenia.

Epidemiology

The true incidence of sinusitis is unknown. In addition to predisposing URI, other risk factors include allergic rhinitis, cystic fibrosis, immunodeficiency, HIV infection, nasogastric or nasotracheal intubation, primary ciliary dyskinesia, nasal polyps, and nasal foreign body.

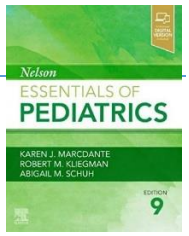
Clinical Manifestations

The most common presentation of acute bacterial sinusitis is persistent rhinorrhea, nasal congestion, and cough, especially at night. Symptoms should be persistent and not improving for more than 10 days to distinguish sinusitis from a URI. Less common symptoms include halitosis, headache, and facial swelling and tenderness. Alternate presentations of acute bacterial sinusitis include high fever and purulent nasal discharge for at least 3 days or a biphasic illness where a patient has typical URI symptoms for up to a week but then worsens with increasing respiratory symptoms and new or recurrent fever.

Chronic sinusitis includes upper respiratory symptoms for more than 30 days, often accompanied by sore throat and/or headache. Fever is rare.

Laboratory and Imaging Studies

Culture of the nasal mucosa is not useful. Sinus aspirate culture is the most accurate method of determining etiology but is not practical or necessary in immunocompetent patients.



Routine imaging is not recommended in uncomplicated infections. Plain x-ray, computed tomography (CT), and magnetic resonance imaging (MRI) may reveal sinus clouding, mucosal thickening, or air-fluid levels in the sinuses. However, abnormal radiographic findings do not differentiate infection from allergic disease and often show abnormalities in the sinuses of children with a simple viral URI. Conversely, normal imaging can have a high negative predictive value for bacterial sinusitis. Bony erosion may be seen on imaging studies in immunocompromised hosts.

Treatment

Amoxicillin for 10–14 days can be used as first-line therapy of uncomplicated sinusitis in children. Broadening therapy to amoxicillin-clavulanate is appropriate if there is no clinical response to amoxicillin within 2–3 days, if risk factors for resistant organisms are present (antibiotic treatment in the preceding 1–3 months, daycare attendance), if there is chronic sinusitis, or if any amount of eye swelling is present.

Complications and Prognosis

Complications of bacterial sinusitis include orbital cellulitis, epidural or subdural empyema, brain abscess, dural sinus thrombosis, osteomyelitis of the frontal sinus **(Pott puffy tumor)** , and meningitis. These all should be managed with sinus drainage and broad-spectrum parenteral antibiotics. Sinusitis also may exacerbate bronchoconstriction in patients with asthma.

Orbital (postseptal) cellulitis is a serious complication of sinusitis that follows bacterial spread into the orbit through the wall of the infected sinus. It typically begins as ethmoid sinusitis and spreads through the **lamina papyracea** , a thin, bony plate that separates the medial orbit and the ethmoid sinus. Orbital involvement can lead to subperiosteal abscess, cavernous sinus thrombosis, and vision loss. Clinical manifestations of orbital cellulitis include a swollen edematous eye, eye pain, proptosis, chemosis, and limitations and pain with extraocular muscle motion, diplopia, and reduced visual acuity. Infection of the orbit must be differentiated from that of the preseptal (anterior to the palpebral fascia) or periorbital space. **Preseptal (periorbital) cellulitis** is common in young children; these patients do not have proptosis or ophthalmoplegia. Preseptal cellulitis may be associated with a skin lesion or trauma and usually is caused by *S. aureus* or group A streptococcus.

The diagnosis of orbital cellulitis is confirmed by a CT scan of the orbit, which helps determine the extent of orbital infection and the need for surgical drainage. Therapy for orbital cellulitis involves broad-spectrum parenteral antibiotics, such as vancomycin or clindamycin with ceftriaxone. Preseptal cellulitis may be treated with oral antibiotics in children with mild disease.

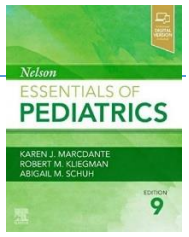
Prevention

The best means of prevention is good handwashing to minimize acquisition of URIs and management of allergic rhinitis.

Pearls for Practitioners

• The bacterial causes of acute sinusitis mirror those of otitis media and are Streptococcus pneumoniae , nontypable Haemophilus influenzae , and Moraxella catarrhalis .

• Complications of sinusitis include orbital cellulitis, Potts puffy tumor, and brain abscess.



Otitis Media

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Nelson Essentials of Pediatrics, Chapter 105, 412-415

Etiology

Otitis media (OM) is a suppurative infection of the middle ear cavity. Bacteria gain access to the middle ear when the normal patency of the eustachian tube is blocked by upper airway infection or hypertrophied adenoids. Air trapped in the middle ear is resorbed, creating negative pressure in this cavity and facilitating reflux of nasopharyngeal bacteria.

Bacteria are the most common pathogens in OM, most frequently as a co-infection with viruses. Viruses can be the sole pathogen in OM, but this is less common (<20%). The common bacterial pathogens are *Streptococcus pneumoniae* , nontypable *Haemophilus influenzae* , *Moraxella catarrhalis* , and, less frequently, group A streptococcus. *S. pneumoniae* that is **relatively resistant** to penicillin (minimal inhibitory concentration 2–8 μg/mL) or **highly resistant** to penicillin (minimal inhibitory concentration ≥8 μg/mL) is isolated with increasing frequency from young children, particularly those who attend day care or have recently received antibiotics.

Epidemiology

Diseases of the middle ear account for approximately one third of office visits to pediatricians. The peak incidence of acute otitis media (AOM) is between 6 and 15 months of life. By the third birthday, 80% of children will have experienced at least one episode of symptomatic or asymptomatic OM. OM is more common in younger children, children exposed to young children, and those with a positive family history. In most of the United States, OM is a seasonal disease with a distinct peak in late winter/early spring, which corresponds to the rhinovirus, respiratory syncytial virus, and influenza seasons.

Approximately 10% of children in the general population have **recurrent OM** , as defined by the presence of three or more AOM episodes in the preceding 6 months or four or more episodes in the preceding 12 months. Craniofacial anomalies (cleft palate) and immunodeficiencies often are associated with recurrent OM, although most children with recurrent AOM are otherwise healthy.

Clinical Manifestations

In infants, the most frequent symptoms of AOM are nonspecific and include fever, irritability, and poor feeding. In older children and adolescents, AOM usually is associated with fever and **otalgia** (acute ear pain). AOM also may present with **otorrhea** (ear drainage) after spontaneous rupture of the tympanic membrane. Signs of a common cold, which predisposes to AOM, are often present (see Chapter 102). A bulging tympanic membrane, air fluid level, or visualization of purulent material by otoscopy are reliable signs of infection (Table 105.1).

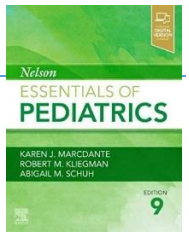


Table 105.1 Definition of Acute Otitis Media

A diagnosis of acute otitis media (AOM) requires:
History of acute onset of signs and symptoms
Presence of middle ear effusion
Signs and symptoms of middle ear inflammation
The definition of AOM includes all of the following:
Recent, usually abrupt, onset of signs and symptoms of middle ear inflammation and middle ear effusion
The presence of middle ear effusion that is indicated by any of the following:
Bulging of the tympanic membrane (TM)
Limited or absent mobility of the TM
Air-fluid level behind the TM
Abnormal TM color (white, yellow, amber)
Otorrhea
Signs or symptoms of middle ear inflammation as indicated by either:
Distinct erythema of the TM
Distinct otalgia (discomfort clearly referable to the ear that results in interference with or precludes normal activity or sleep)

Examination of the ears is essential for diagnosis and should be part of the physical examination of any child with fever. The hallmark of OM is the presence of effusion in the middle ear cavity (see Table 105.1). The presence of an effusion does not confirm the presence of a bacterial OM, but it does define the need for appropriate diagnosis and therapy.

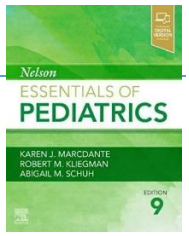
Pneumatic otoscopy , using an attachment to an otoscope, allows evaluation of ventilation of the middle ear and is a standard for clinical diagnosis. The tympanic membrane of the normal, air-filled middle ear has much greater compliance than if the middle ear is fluid filled. With AOM, the tympanic membrane is often characterized by **hyperemia** , or red color rather than the normal pearly gray color, but may appear pink, white, or yellow depending on the degree of bulging (Fig. 105.1). The light reflex is lost, and the middle ear structures are obscured and difficult to distinguish. There should be poor or absent mobility to negative and positive pressure; this is a necessary finding for the diagnosis of OM. A hole in the tympanic membrane or purulent drainage confirms perforation. Occasionally, bullae are present on the lateral aspect of the tympanic membrane, which characteristically are associated with severe ear pain.

Laboratory and Imaging Studies

Routine laboratory studies are not useful in the evaluation of OM. **Tympanometry** provides objective acoustic measurements of the tympanic membrane–middle ear system by reflection or absorption of sound energy from the external ear duct as pressure in the duct is varied. Measurements of the resulting **tympanogram** correlate well with the presence or absence of middle ear effusion.

Instruments using **acoustic reflectometry** are available for office use. Use of reflectometry as a screening test for AOM should be followed by examination with pneumatic otoscopy when abnormal reflectometry is identified.

Bacteria recovered from the nasopharynx do not correlate with bacteria isolated by tympanocentesis. Tympanocentesis and middle ear exudate culture are not always necessary, but they are required for accurate identification of bacterial pathogens and may be useful in neonates, immunocompromised patients, and patients not responding to therapy.



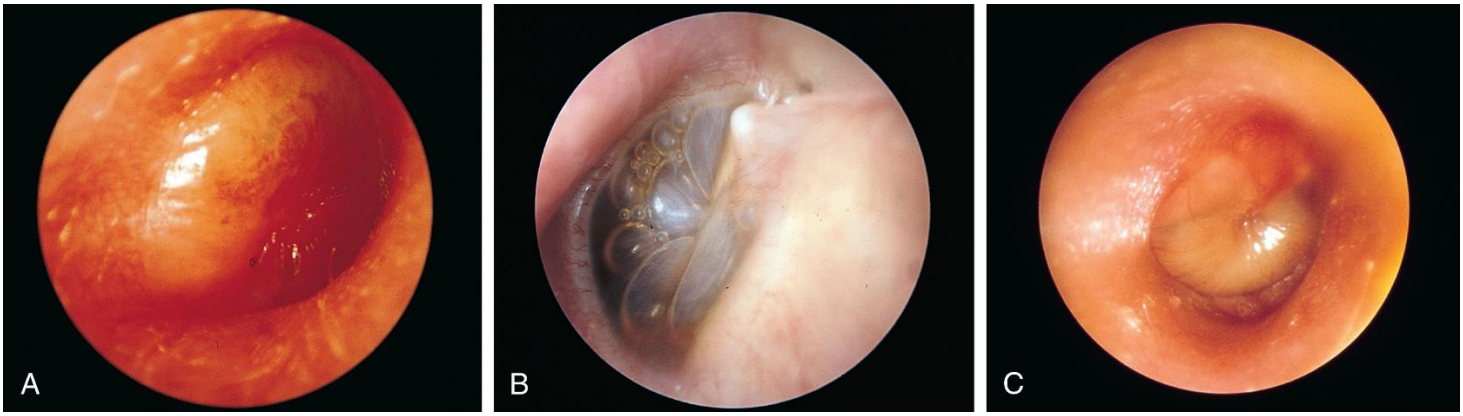


Figure 105.1

Acute otitis media. (A) This is the classic picture—an erythematous, opaque, bulging tympanic membrane. The light reflex is reduced, and the landmarks are partially obscured. Mobility is markedly reduced. (B) In this acutely febrile child who complained of otalgia, the presence of both air and fluid formed bubbles separated by grayish-yellow menisci. Even though the drum was not injected, this finding, combined with fever and otalgia, is consistent with acute infection. (C) In this child, the tympanic membrane was injected at the periphery, and a yellow purulent effusion caused the inferior portion to bulge outward. Mobility was markedly reduced.

(From Yellon RF, Chi DH. Otolaryngology. In: Zitelli BJ, McIntire SC, Nowalk AJ, eds. *Atlas of Pediatric Physical Diagnosis* . 7th ed. Philadelphia: Saunders; 2018:868-915 [fig. 24.24].)

Differential Diagnosis

The major difficulty is differentiation of AOM from **OM with effusion (OME)** , which also is referred to as **chronic OM** . AOM is accompanied by signs of acute illness, such as fever, pain, and upper respiratory tract inflammation. OME is the presence of effusion without any of the other signs and symptoms. OME may occur either as the sequela of AOM or eustachian tube dysfunction secondary to an upper respiratory tract infection. It may also predispose to the development of AOM. Because OME is not an acute infectious process, it is important to be able to differentiate between OME and AOM accurately to prevent overprescribing of antibiotics.

Treatment

Recommendations for treatment are based on age, certainty of diagnosis, and severity of illness (Table 105.2). The recommended first-line therapy for most children meeting criteria for antibiotic therapy is amoxicillin (80–90 mg/kg/day in two divided doses). Some children with mild illness or uncertain diagnosis may be observed if appropriate follow-up within 48–72 hours can be arranged with initiation of antibiotic therapy if symptoms do not self-resolve (see Table 105.2). The failure of initial therapy with amoxicillin at 3 days suggests infection with β-lactamase-producing *H. influenza* , *M. catarrhalis* , or resistant *S. pneumoniae* . Recommended next-step treatments include high-dose amoxicillin-clavulanate (amoxicillin 80–90 mg/kg/day), cefdinir, or ceftriaxone (50 mg/kg intramuscularly in daily doses for 3 days). Intramuscular ceftriaxone is especially appropriate for children with vomiting that precludes oral treatment. Tympanocentesis may be required for patients who are difficult to treat or who do not respond to therapy. Acetaminophen and ibuprofen are recommended for fever or pain. Decongestants or antihistamines are not effective.

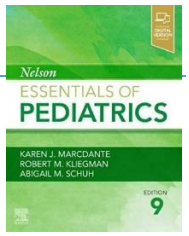


Table 105.2 Recommendations for Initial Management of Confirmed Acute Otitis Media *

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Age	AOM with otorrhea	AOM with severe symptoms †	Bilateral AOM without otorrhea	Unilateral AOM without otorrhea
6 mo to 2 yr	Antibiotics	Antibiotics	Antibiotics	Antibiotics or additional observation ‡
≥2 yr	Antibiotics	Antibiotics	Antibiotics or additional observation	Antibiotics or additional observation

AOM , Acute otitis media.

* Applies only to children with well-documented AOM with high certainty of diagnosis.

† A toxic-appearing child, persistent otalgia for >48 hr, temperature ≥39°C (102.2 °F) in the past 48 hr, or if there is uncertain follow-up.

‡ Requires shared decision making with parents and close follow-up within 48–72 hr.

Complications and Prognosis

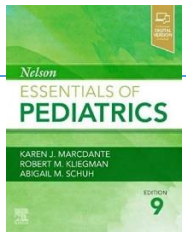
The complications of OM are tympanic membrane perforation, chronic effusion, chronic otorrhea, hearing loss, cholesteatoma (masslike keratinized epithelial growth), facial palsy, petrositis, intracranial extension (brain abscess, subdural empyema, or venous thrombosis), and mastoiditis. **Acute mastoiditis** is a suppurative complication of OM with inflammation and potential destruction of the mastoid air spaces. The disease progresses from a periostitis to an osteitis with mastoid abscess formation. Posterior auricular tenderness, swelling, and erythema, in addition to the signs of OM, are present. The pinna is displaced downward and outward. Computed tomography scan of the mastoid reveals clouding of the air cells, demineralization, or bone destruction. Treatment includes systemic antibiotics and drainage if the disease has progressed to abscess formation.

OME is the most frequent sequela of AOM and occurs most frequently in the first 2 years of life (up to 30–50% of children with AOM). **Persistent middle ear effusion** may last for many weeks or months in some children but usually resolves by 3 months following infection. Evaluating young children for this condition is an important part of all well-child examinations because, although not an infectious process, it is associated with hearing loss.

Conductive hearing loss should be assumed to be present with persistent middle ear effusion; the loss is mild to moderate and often is transient or fluctuating. In children at developmental risk or with frequent episodes of recurrent AOM, 3 months of persistent effusion with significant bilateral hearing loss is a reasonable indicator of need for intervention with insertion of pressure equalization tubes (tympanostomy tubes).

Prevention

Parents should be encouraged to continue exclusive breast-feeding as long as possible and should be cautioned about the risks of *bottle-propping* and of children taking a bottle to bed. The home should be a smoke-free environment, and pacifier use should be limited.



The conjugate *S. pneumoniae* vaccine reduces pneumococcal OM caused by vaccine serotypes by 50%, all pneumococcal OM by 33%, and all OM by 6–10%. Annual immunization against influenza virus may be helpful in high-risk children.

Pearls for Practitioners

• Poor or absent tympanic membrane mobility to negative and positive pressure using pneumatic otoscopy is a necessary finding for the diagnosis of acute otitis media (AOM).

• Complications of AOM include persistent middle ear effusion, hearing loss, mastoiditis, brain abscess, perforated tympanic membrane, and cholesteatoma.

• OM with effusion is the most frequent sequela of AOM and may result in persistent middle ear effusion, which may be associated with hearing loss.

• Not all episodes of AOM require antibiotic treatment.