Marah Gotcsik

Definition/Classification

Otitis media is inflammation of the middle ear. Dependent upon presentation, associated symptoms, and duration of symptoms, the diagnosis can be further classified as follows:

Acute otitis media (AOM) is infection of the middle ear with acute onset, middle-ear effusion (MEE) on exam, and presence of signs and symptoms of middle-ear inflammation. Treatment failure AOM is defined by lack of improvement despite 48–72 h of appropriate antibiotic therapy. Diagnosis of Recurrent AOM requires three or more episodes in 6 months or four or more episodes in 12 months.

Otitis media with effusion (OME) is middle-ear effusion without signs or symptoms of active infection. Chronic OME (COME) is when this effusion persists for more than 3 months.

Chronic suppurative otitis media (CSOM) is chronic inflammation of the middle ear and mastoid cavity presenting with otorrhea through a perforated tympanic membrane. The World Health Organization definition requires 2 weeks of otorrhea for diagnosis, whereas others have suggested 6 weeks of symptoms as a diagnostic requirement.

Epidemiology

Otitis media is one of the most common pediatric diagnoses worldwide, and the prevalence may be increasing: United States surveillance data by the Centers for Disease Control showed a 150% increase to 24.5 million office visits per year with a principal diagnosis of otitis media from 1975 to 1990. At least 70% of children have at least one episode of AOM before 2 years of age, and AOM is the most common reason for antibiotic prescription for children in the United States.

Approximately 90% of children are diagnosed with OME before school age. Most episodes resolve spontaneously, but 30–40% of children have recurrent OME and 5–10% of episodes last 1 year or longer.

Numerous host and environmental risk factors have been studied in association with AOM and OME (see

● *Table 70.1*). The significance of genetic predisposition to the development of otitis media has been demonstrated with twin studies showing heritability as high as 70%. Studies have examined race and likelihood of developing otitis media without clear consensus.

CSOM, though rare in the developed world, remains a major cause of morbidity and mortality in the developing world and within certain racial groups. Unfortunately, the lack of consensus definition and variability in methods makes meta-analysis studies difficult. The highest prevalence of CSOM is in Alaska Native, Native Greenland, Native American, and Australian Aborigine children (prevalence 7–46%). Children in the South Pacific Islands, Africa, Korea, India, and Saudi Arabia have a relatively high prevalence of 1–6% with some subregions being more affected than others. Highly developed countries, such as the US and UK, have the lowest prevalence at <1%.

Recurrent AOM is associated with development of CSOM, suggesting similar risk factors for both disease processes. In the antibiotic era, rates of CSOM have decreased, further supporting a link between nontreatment and progression to chronic disease. Additional identified risk factors include inadequate antibiotic treatment, frequent upper respiratory tract infections, nasal disease, and poor living conditions with poor access to medical care. It is encouraging that with intervention in these identified areas there can be a direct impact on health; one study targeting improvements in housing, hygiene, and nutrition resulted in a 50% decrease in CSOM in Maori children.

Pathogenesis

Development of otitis media is strongly associated with impairment of the structure and function of the eustachian tube. At baseline, the middle-ear space maintains a negative air pressure relative to the environment. This pressure is periodically relieved by opening the eustachian tube, for example, in the setting of yawning and chewing. If this pressure equalization cannot be achieved, inflammation can occur resulting in otitis media.

■ Table 70.1 Risk factors for development of otitis media

Host factors	Environmental factors	
Age <2 year	Bottle propping	
Atopy	Child care attendance	
Chronic sinusitis	Crowding in housing	
Ciliary dysfunction	Low socioeconomic status	
Cleft palate/Craniofacial anomalies	Non-breast feeding in infancy	
Immunocompromise	Passive smoke exposure	
Male sex	Winter season (respiratory virus exposure)	
Trisomy 21		

Craniofacial anomalies with distortion of ear and eustachian tube anatomy are an obvious cause of eustachian tube dysfunction. However, normal pediatric anatomy is also associated with impairment of pressure equalization. In infants and children, the eustachian tube can be as short as half the length of the adult eustachian tube and is comprised of highly compliant cartilage positioned at a near horizontal angle (as opposed to the adult eustachian tube which is more firm and lies in a 45° angle in relation to the axial plane). The characteristics of the pediatric eustachian tube compromise the ability to effectively ventilate the middle ear. Inflammation due to upper respiratory tract infections (URI), allergic rhinitis, and gastro-esophageal reflux further impairs eustachian tube function. Additionally, nasopharyngeal secretions can reflux or be insufflated into the middle-ear space, serving as nidus for infection.

Both bacteria and viruses have been implicated in otitis media. AOM and OME have similar associated pathogens, whereas CSOM is has a distinct microbial profile (*Table 70.2*).

Upper respiratory tract infections are important in the pathogenesis of AOM. AOM has been identified as a complication of 37% of URIs in children, as high as 50% with coronavirus, respiratory syncytial virus (RSV), and adenovirus infection, and as high as 60% in human metapneumovirus infection.

In studies examining middle-ear fluid in patients with AOM, viruses are isolated 20–50% of the time, alone or together with bacterial otopathogens. The most frequently isolated virus types from middle-ear fluid are RSV, parainfluenza, and influenza (A and B).

Bacteria are isolated from middle-ear fluid in AOM 50–90% of the time. Historically, the most common bacteria associated with otitis media is *Streptococcus*

pneumoniae. Following introduction of PCV7, vaccine serotypes of *S. pneumoniae* have been nearly completely eliminated, with *Haemophilus influenzae* becoming more prevalent. As with other *S. pneumoniae* infections, there is some evidence that non-vaccine serotypes are increasing to fill this niche – including identification of a multidrug resistant serotype 19A. The introduction of the 13-valent pneumococcal vaccine (PCV13) in 2010 will undoubtedly be associated with further changes in the bacterial otopathogens of AOM.

Development of OME is also associated with URI. URI can lead to OME nearly one quarter of the time, with a slightly higher incidence in the setting of influenza. Middle-ear fluid in COME was previously thought to be sterile based on culture results. However, the advent of PCR technology has allowed identification of bacterial DNA and mRNA in culture-negative middle-ear fluid. One hypothesis for this finding is the presence of biofilms – aggregated bacteria growing on a surface, surrounded by an extracellular matrix. Supporting this hypothesis are recent studies with visualization of biofilm on microscopy during placement of tympanostomy tubes in patients with COME.

Although CSOM is associated with recurrent AOM, the associated bacterial pathogens are different. Bacteria can reach the middle ear via the eustachian tube or via the external ear through the perforated tympanic membrane. There is wide variability in the prevalence of the pathogens; however, *Pseudomonas aeruginosa* is the most common, isolated from 18% to 67% of ear cultures. *P. aeruginosa* is implicated in the progressive destruction of middle-ear and mastoid structures. Unlike AOM and OME, fungi are a common pathogen, isolated from 50% of ear cultures from populations in hot, humid regions. Biofilms are also gaining attention as a possible cause for chronic infection in CSOM.

Pathology

The bony structures of the ear, the inner ear mucosa, and the tympanic membrane can readily heal from acute infection – including acute infection with perforation – without long term disability. In children with history of otitis media, tympanic membrane abnormalities present at 8 years of age nearly completely resolve by 18 years of age.

CSOM can result in damage of the typmanic membrane, the ossicles, and the mastoid space leading to osteoneogenesis, bony erosions, and osteitis of the temporal bones and ossicles.

■ Table 70.2

Pathogens identified by culture or PCR in middle-ear fluid of patients with AOM, OME, and CSOM

	AOM	OME	CSOM
Bacteria	Streptococcus pneumoniae	Streptococcus pneumoniae	Aerobic
	Haemophilus influenzae	Haemophilus influenzae	Pseudomonas aeruginosa
	Moraxella catarrhalis	Moraxella catarrhalis	Staphylococcus aureus
	Streptococcus pyogenes		Escherichia coli
	Staphylococcus aureus		Proteus mirabilis
	Streptococcus agalactiae (neonates)		Klebsiella species
	Gram negative bacilli (neonates)		Haemophilus influenzae
			Anaerobic
			Bacteroides
			Fusobacterium
Viruses	Respiratory syncytial virus	Rhinovirus	
	Influenza	Respiratory syncytial virus	
	Adenovirus	Coronavirus	
	Parainfluenza		
	Rhinovirus		
	Coronavirus		
Fungi			Aspergillus species
			Candida species

Clinical Manifestations

Acute Otitis Media

Specific signs and symptoms: Otalgia is the most common complaint in children with AOM. Although not a sensitive marker of disease, it does have a high positive predictive value for presence of infection. Other specific signs are otorrhea following acute tympanic membrane perforation, hearing loss, vertigo, nystagmus, and tinnitus. Ear swelling and facial paralysis are signs that may suggest a more invasive process within the mastoid space or the temporal bone, respectively.

Nonspecific signs and symptoms: Fever is present in one third to two thirds of patients with AOM, although fever $>40^{\circ}$ may suggest bacteremia or alternate focal infection. Additional nonspecific symptoms are irritability, headache, anorexia, vomiting, and diarrhea.

Otitis Media with Effusion

OME, by definition, has minimal symptoms, with mild hearing loss being the most common complaint. Symptoms of an underlying process resulting in oropharyngeal inflammation (URI, allergic rhinitis, gastro-esophageal reflux) may be present.

Chronic Suppurative Otitis Media

Patients with CSOM present with chronic otorrhea and may also have significant hearing loss. Conductive hearing loss of 20–60 dB may be present due to tympanic membrane perforation and damage to the ossicles. Sensorineural hearing loss may also occur in CSOM in association with loss of cochlear hair cells due to inflammation.

Diagnosis

Proper diagnosis of otitis media is imperative, especially in the era of increasing antibiotic resistance. Unlike other disease processes, otitis media remains a clinical diagnosis based on history and physical exam with pneumatic otoscopy.

Assessment of the tympanic membrane (TM) should include color, translucency, position, and mobility. A normal TM has a ground glass appearance, is translucent with

clear visualization of the bony landmarks, and has good movement with pneumatic otoscopy.

Proper equipment is paramount to adequate visualization of the TM. The best light source is a halogen bulb with at least 100 ft-candles and a well-charged battery. Speculum choice is also important. When available, reusable specula are preferable because of their length, size options, and glossy finish which facilitates light transmission into the ear canal. In choosing a speculum, the largest lumen possible that can comfortably fit into the cartilaginous portion of the canal should be selected. Small speculums not only limit the field of view, but may pass into the bony portion of the canal resulting in pain.

Visualization alone can provide clues to the presence of effusion but is not adequate for diagnosis. Pneumatic otoscopy is highly sensitive and specific for the presence of middle ear effusion. Pneumatic otoscopy is a multistep procedure designed to assess the TM response to both positive and negative pressure. The speculum is inserted into the ear with the no pressure on the bulb. Slight pressure is applied to the bulb to create positive pressure. Next, the seal is momentarily broken to allow for neutralization of pressure. It is important to realize that a normal eardrum will move with 10–15 mm H₂O, whereas a pneumatic otoscope can deliver as much as 1,000 mm H₂O when the bulb is fully depressed.

Tympanometry may be used as an adjunct to pneumatic otoscopy if available. With a tympanometer, the compliance of the TM is assessed by the reflection of sound waves off the TM (\bigcirc *Fig. 70.1*).

Acute Otitis Media

Although AOM is a common pediatric problem throughout the world, development of international diagnostic criteria has proved challenging. Previous definitions have been too broad, without clear distinction between AOM and OME. This lack of consensus has limited the ability to study diagnosis and treatment practices.

In 2004, the American Academy of Pediatrics (AAP) and American Academy of Family Practice (AAFP) published clinical practice guidelines on diagnosis of acute otitis media with more specific criteria:

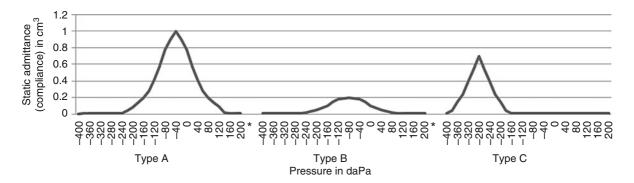
- 1. History of acute onset of signs and symptoms
- 2. Presence of MEE (bulging TM, limited or absent mobility of the TM, an air-fluid level behind the TM, or otorrhea)
- 3. Signs and symptoms of middle-ear inflammation (direct erythema of the TM, bullous myringitis, and distinct otalgia)

Otitis Media with Effusion

The commonly accepted diagnostic criteria for OME are:

- 1. Presence of MEE
- 2. No signs or symptoms of AOM

The 2004 AAP, AAFP, and American Academy of Otolaryngology – Head and Neck Surgery clinical practice guidelines strongly recommended that pneumatic



☐ Figure 70.1

Examples of tympanometry curves Type A: Normal TM compliance. Type B: Poor compliance of TM with limited to no movement, in extreme cases can be flat. Type B curves are strongly associated with MEE. Type B curves can also be found with TM perforation in the setting of high measured volume. Type C: Decreased amplitude of compliance and shifted to the left, suggesting increased negative pressure in the middle ear. Type C curves are not diagnostic of middle-ear effusion

otoscopy should be used as the primary diagnostic method for OME to assess for MEE, based on grade A evidence.

Chronic Suppurative Otitis Media

The World Health Organization (WHO) diagnostic criteria of CSOM are:

- 1. Tympanic membrane perforation
- Purulent otorrhea present continuously for at least two weeks

Treatment

Acute Otitis Media

In the developed world, there is large variation in practice of antibiotic prescription for AOM. In the setting of increasing antibiotic resistance, management practices for AOM have been reevaluated with emphasis on limiting antibiotic usage when possible. If left untreated, 61% of patients with AOM will have resolution of symptoms within 24 h, improving to 80% within 2-3 days. There is no significant difference in the incidence of suppurative complications, including mastoiditis, if antibiotics are initially withheld. This has led to a trend of expectant management with emphasis on pain control at the initial diagnosis of AOM. In certain groups with a high burden of disease, such as Australian Aborigines, national guidelines continue to support initiation of treatment at presentation. Rural populations may also not be appropriate for initial observation as close follow-up must be possible if a patient is not receiving antibiotics.

Numerous countries have adopted guidelines supporting a watch-and-wait approach to AOM, including the Netherlands, where there is the lowest rate of antibiotic prescription for AOM. The 2004 AAP guidelines stratify treatment by age and certainty of diagnosis. In otherwise healthy children with certain diagnosis, children under two years of age receive treatment, whereas those children greater that two years of age without significant ear pain or fever can be observed with follow-up. In cases of uncertain diagnosis, treatment is only recommended in children less than six months of age or if the child is six months to two years of age with significant ear pain or fever.

General Care

Supportive care is an important part of AOM management, including pain and fever control. There is no evidence for the use of antihistamines or decongestants in treatments of AOM.

Specific Treatment

Antibiotic choice in AOM is focused on medications with efficacy against *S. pneumoniae*, *H. influenzae*, and *Moraxella catarrhalis*.

First Line Therapy

Amoxicillin is widely accepted as first line therapy for non-penicillin allergic patients. However, the dosage and duration of treatment is highly variable. The Dutch College of General Practitioner Guidelines support lower dose therapy 30 mg/kg/day amoxicillin as compared to the AAP recommendations of 80–90 mg/kg/day. The rationale for higher dosing includes increased drug concentration in the middle ear and for the treatment of resistant strains of *S. pneumoniae*. In cases with fever >39 C or severe otalgia, the AAP recommends amoxicillin-clavulanic acid as first line therapy because of the potassium clavulanate's ability to inhibit B-lactamase-produced by many *H. influenzae* and *M. catarrhalis*.

In penicillin allergic patients there is less consensus, and recommendations include cephalosporins (cefdinir, cefuroxime, cefpodoxime, ceftriaxone), macrolides (azithromycin, clarithromycin, erythromycin), and sulfonamides (co-trimoxazole).

Treatment Failure Therapy

In cases of treatment failure, the AAP recommends amoxicillin-clavulanic acid, ceftriaxone, or clindamycin dependent on severity and if allergy to penicillin is present. The India-WHO Collaborative Programme also recommends amoxicillin-clavulanic acid as well as cefaclor.

Although tympanocentesis can be used in severe cases of treatment failure AOM, placement of tympanostomy tubes is reserved for children with recurrent otitis media. For children with tympanostomy tubes, AOM manifesting with otorrhea can be treated with ofloxacin otic solution or ciprofloxacin-dexamethasone otic solution.

Duration of Treatment

The recommended duration is also variable. The previous accepted duration of therapy for AOM was 10 days. Although this recommendation remains for younger children, children older than five years have demonstrated successful therapy with five to seven days of antibiotics. Older children may also be successfully treated with 1–5 days of azithromycin or 1–3 days of intramuscular ceftriaxone.

Future Development

As more national guidelines are endorsing the watch-andwait approach to AOM, antibiotic prescribing patterns and development of complications must be monitored closely.

Otitis Media with Effusion

General Care

Just as with AOM, the rate of spontaneous resolution in OME is high. Of children with OME developing following AOM, 75% have spontaneous resolution of MEE in 3 months. Of children with incidentally identified OME, 42% have resolution within 6 months with 72% showing improvement in tympanometry curves at that time. Therefore, the watch-and-wait approach is also prudent in OME.

During this period of observation, hearing assessment every 3–6 months in addition to otoscopic evaluation with pneumatic otoscopy is important for ongoing surveillance. Asymptomatic effusions can be managed conservatively without intervention.

Specific Treatment

There is no clear role for medications in treatment of OME. Antihistamines and decongestants are not indicated for treatment. Antibiotics and corticosteroids have not been shown to have lasting effect. In children with persistent effusion lasting more than 4 months complicated by hearing loss, developmental delay (or risk for developmental delay), damage to the tympanic membrane, or otalgia or balance problems, tympanostomy tube placement may be indicated to remove the middle-ear effusion.

Future Development

Development of widely accepted and followed consensus guidelines may allow for further understanding of observation and management of OME.

Chronic Suppurative Otitis Media

The hallmarks of management of CSOM are treatment of the infection and closure of the tympanic membrane.

Mastoidectomy with tympanoplasty is curative, but there is limited access to tertiary centers offering these services for many of the children who are most affected. If the infection has spread deep into the middle ear, there is diffuse mucosal disease, or cholesteatoma has developed, the patient will likely be refractory to medical therapy alone. For patients without evidence of suppurative complication, the WHO has created a conservative medical management algorithm focused on aural toilet in conjunction with antibiotic treatment. Topical treatment has been shown to be superior to systemic therapy as well as being more cost-effective. Neomycin-polymyxin is recommended as first line therapy, with transition to quinolones, such as ofloxacin or ciprofloxacin, or gentamicin if drainage persists 2 weeks and ear culture is positive for Pseudomonas. If the drainage resolves on this regimen, the patient must then be followed for closure of the tympanic membrane. If the perforation persists, tympanoplasty can be performed with goal to restore hearing. If the perforation persists and otorrhea recurs, mastoidectomy is indicated.

Prognosis

In general, prognosis following an episode of otitis media is good. However, suspicion should be maintained for the many complications that can develop, most associated with local spread of infection.

Intratemporal Complications

Hearing loss is the most prevalent complication of otitis media. Conductive hearing loss is most commonly due to MEE and, less frequently, atelectasis of the middle ear due to high negative pressures. Average hearing loss is 20–30 dB, with a range of 0–60 dB. Sensorineural hearing loss can also be present due to increased tension on the round window membrane and inflammation of the cochlear hair cells. Hearing loss in CSOM is more severe than in AOM

and OME. Hearing loss may be associated with impaired development of speech and language with resultant poor school performance.

Tympanic membrane perforation can occur in AOM and OME. The TM usually heals within 2–3 months. For chronic perforations, lasting longer than 3 months, tympanoplasty is indicated.

Mastoiditis, although decreasing incidence in the antibiotic era, is the most common suppurative complication of otitis media. As the mastoid gas cell system is connected to the middle ear, all episodes of otitis media likely result in some degree of mastoiditis. If the infection spreads to the periosteum, mastoiditis with periosteitis Presenting signs are fever, otalgia, postauricular erythema and mild tenderness, and possible anterior/inferior displacement of the pinna. The most severe form of mastoiditis occurs when infection spreads from the periosteum into the bone resulting in osteitis with potential for development of subperiosteal abscess. In mastoiditis with osteitis, postauricular erythema and tenderness are more pronounced, and the pinna is clearly displaced anteriorly and inferiorly. CT scan can be used to assess the extent of disease. Mastoiditis with periosteitis can be treated with parenteral antibiotics, tympanocentesis, and myringotomy. Mastoiditis with osteitis additionally requires mastoidectomy.

Cholesteatoma is a cystic mass of keratinized squamous cell epithelium and cholesterol in the middle ear. Cholesteatoma can be congenital or acquired, with acquired disease associated with chronic otitis media. Implantation cholesteatoma can develop following traumatic perforation of the TM or as a complication of ear surgery. In children, the most common location of cholesteatoma is the posterosuperior quadrant of the pars tensa. On otoscopy, a defect in the TM may be visualized with white greasy flakes of debris and sometimes foul smelling drainage. Findings on otoscopy can be subtle or difficult to visualize, making CT scan a useful diagnostic tool when there is high suspicion of disease. The squamous epithelium can extend inward, creating an expanding cystic cavity that causes progressive erosion of the ossicle, the mastoid space, and the temporal bone. Therefore, prompt surgical management is indicated.

Tympanosclerosis can occur with chronic middle-ear inflammation or as the result of trauma to the TM, including tympanostomy tube placement. On otoscopy, whitish plaques are visualized on the TM. Nodular deposits in the submucosal layers of the middle ear are also present. Conductive hearing loss can result if the ossicles become embedded in these deposits.

Suppurative labyrinthitis is a rare complication of AOM and OME, but can be seen in CSOM. The affected child

presents with sudden onset of vertigo, nausea/vomiting, disequilibrium, and severe sensorineural hearing loss.

Facial paralysis can occur as a complication of AOM due to the facial nerve's course through the middle ear. Treatment consists of parenteral antibiotics, tympanocentesis, and myringotomy. Immediate surgical mastoidectomy is indicated in children with CSOM or cholesteatoma.

Intracranial Complications

Intracranial complications of meningitis, epidural abscess, subdural empyema, focal encephalitis, brain abscess, lateral sinus thrombosis, and otitic hydrocephalus have decreased in the antibiotic era, but remain a significant problem in underdeveloped countries, especially in the setting of cholesteatoma and CSOM. Due to spreading infection, 24–44% of children presenting with an intracranial complication will be found to have more than one intracranial complication at the time of diagnosis.

Meningitis can occur due to direct spread of infection through the dura, inflammation due to an alternate intracranial complication (such as abscess), or can be a concurrent infection with hematogenous spread from the upper respiratory tract. Hematogenous spread is the most common etiology. Treatment is parenteral antibiotics.

Epidural abscess is a collection of granulation tissue and pus between the dura and the temporal bone and can result when the temporal bone is compromised by cholesteatoma or infection. Earache, low-grade fever, and temporal headache may be present, as well as profuse, creamy, pulsatile otorrhea. Many cases of epidural abscess are asymptomatic. Treatment is surgical drainage and directed therapy with parenteral antibiotics.

Subdural empyema, although more common in sinusitis, is a rare but serious complication of otitis media. A pus collection develops in the subdural space, usually due to direct extension of infection. Presentation can be severe with toxic appearance, fever, and focal neurologic signs. Treatment is parenteral antibiotics and neurosurgical drainage if indicated.

Focal otitic encephalitis is non-suppurative brain inflammation associated with chronic otitis media or one of the associated suppurative complications. Presentation may be similar to brain abscess, necessitating MRI to distinguish the two entities. Treatment consists of antibiotics for the inciting infection.

Brain abscess can occur with AOM, CSOM, and cholesteatoma. Infection progresses to the brain from a localized subdural abscess or leptomeningitis. Otogenic

brain abscess are located in the temporal lobe or the cerebellum, depending on site of infection invasion. Multiple abscesses are common. The bacteria found in abscesses are the most common bacteria associated with AOM and CSOM. Signs of general and focal neurologic signs generally appear 1 month following acute infection. Systemic signs maybe absent. Treatment is surgical debridement and parenteral antibiotics.

Lateral sinus thrombosis can occur as a result of mastoid inflammation. The infection spreads though the mastoid space into the sinus, and finally into the venous system leading to thrombus formation. Signs of infection are signs of systemic infection, increased intracranial pressure, and sequelae of septic thromboembolism. Lateral sinus thrombosis has a high co-diagnosis with other intracranial complications. Treatment is parenteral antibiotics. There is no consensus on use of anticoagulation. Otitic hydrocephalus, markedly increased intracranial pressure, is highly associated with lateral sinus thrombosis.

Prevention

Otitis media is associated with environmental factors and genetic predisposition to disease. Some of the major environmental associations such as crowding and child care attendance may be difficult to impact in large scale prevention efforts. Due to these challenges, vaccines targeting the most frequent pathogens may be the most effective means to prevent otitis media. The introduction of PCV7 showed decrease in AOM diagnosis, but increase in presence of non-vaccine serotypes. Introduction of PCV13 as well as development of a vaccine against non-typable *H. influenzae* has the potential to substantially decrease rates of AOM.

For children with recurrent otitis media, daily antibiotic prophylaxis reduces the probability of developing AOM, OME, and CSOM. There is no continuing benefit after antibiotics are stopped, but this management strategy may be beneficial during high risk periods. Usual dosing regimens are half the daily treatment dose of amoxicillin or sulfonamides.

References

- Acuin J (2004) Chronic suppurative otitis media: burden of illness and management options. World Health Organization, Geneva
- American Academy of Pediatrics (2004) Clinical practice guideline: otitis media with effusion. Pediatrics 113(5):1412–1429

- American Academy of Pediatrics Subcommittee on Management of Acute Otitis Media (2004) Diagnosis and management of acute otitis media. Pediatrics 113(5):1451–1465
- Bluestone CD, Klein JO (2007) Otitis media in infants and children, 4th edn. BC Decker, Hamilton
- Berkun Y, Nir-Paz R, Ben Ami A (2008) Acute otitis media in the first two months of life: characteristics and diagnostic difficulties. Arch Dis Child 93:690–694
- Bulut Y, Göven M, Otlu B et al (2007) Acute otitis media and respiratory viruses. Eur J Pediatr 166:223–228
- Carlson LH, Carlson RD (2003) Diagnosis. In: Rosenfeld RM, Bluestone CD (eds) Evidence based otitis media, 2nd edn. BC Decker, Hamilton
- Casey JR, Adlowitz DG, Pichichero ME (2010) New patterns in otopathogens causing acute otitis media six to eight years after introduction of the pneumococcal conjugate vaccine. Pediatr Infect Dis I 29:304–309
- Chonmaitree T, Revai K, Grady JJ et al (2008) Viral upper respiratory tract infection and otitis media complication in young children. Clin Infect Dis 46(6):815–823
- Coco A, Vernacchio L, Horst M, Anderson A (2010) Management of acute otitis media after publication of the 2004 AAP and AAFP clinical practice guideline. Pediatrics 125:214–220
- Coker TR, Chan LS, Newberry SJ (2010) Diagnosis, microbial epidemiology, and antibiotic treatment of acute otitis media in children: a systematic review. JAMA 304(19):2161–2169
- DeBeer BA, Schilder AGM, Zielhuis GA, Graamans K (2005) Natural course of tympanic membrane pathology related to otitis media and ventilation tubes between ages 8 and 18 years. Otol Neurotol 26:1016–1021
- Eskola J, Kilpi T, Palmu A et al (2001) Efficacy of a pneumococcal conjugate vaccine against acute otitis media. N Engl J Med 344(6): 403–409
- Gould JM, Matz PS (2010) Otitis media. Pediatr Rev 31(3):102-115
- Haggard M (2008) Otitis media: prospects for prevention. Vaccine 26S: G20–G24
- Hall-Stoodley L, Hu FZ, Gieseke A et al (2006) Direct detection of bacterial biofilms on the middle-ear mucosa of children with chronic otitis media. JAMA 296(2):202–211
- Hamamoto Y, Gotoh Y, Nakajo Y et al (2005) Impact of antibiotics on pathogens associated with otitis media with effusion. J Laryngol Otol 119:862–865
- Jensen PM, Lous J (1999) Criteria, performance and diagnostic problems in diagnosing acute otitis media. Fam Pract 16(3):262–268
- Leach AJ, Morris PS (2006) Antibiotics for the prevention of acute and chronic suppurative otitis media in children. Cochrane Database Syst Rev 4:CD004401. doi: 10.1002/14651858.CD004401.pub2
- Mackenzie GA, Carapetis JR, Leach AJ, Morris PS (2009) Pneumococcal vaccination and otitis media in Australian Aboriginal infants: comparison of two birth cohorts before and after introduction of the vaccine. BMC Pediatr 9:14
- McCaig LF, Besser RE, Hughes JM (2002) Trends in antimicrobial prescribing rates for children and adolescents. JAMA 287(23): 3096–3102
- Nederlands Huisartsen Genootschap (2006) Otitis media acuta bij kindirin. Available at http://nhg.artsennet.nl
- Onusko E (2004) Tympanometry. Am Fam Physician 70(9):1713–1720
- Pitkaranta A, Jero J, Arruda E, Virolainen A, Hayden FG (1998) Polymerase chain reaction—based detection of rhinovirus, respiratory syncytial virus, and coronavirus in otitis media with effusion. J Pediatr 133(3):390–394

- Pichichero ME, Casey JR (2007) Emergence of multiresistant serotype 19A pneumococcal strain not included in the 7-valent conjugate vaccine as an otopathogen in children. JAMA 298(15):1772–1778
- Post JC, Preston RA, Aul JJ et al (1995) Molecular analysis of bacterial pathogens in otitis media with effusion. JAMA 273:1598–1604
- Rosenfeld RM, Kay D (2003) Natural history of untreated otitis media. In:
 Rosenfeld RM, Bluestone CD (eds) Evidence based otitis media,
 2nd edn. BC Decker, Hamilton
- Rovers M, Haggard M, Gannon M et al (2002) Heritability of symptom domains in otitis media: a longitudinal study of 1, 373 twin pairs. Am J Epidemiol 155(10):958–964
- Schappert SM (1992) Office visits for Otitis Media: United States, 1975–90, Advance data from vital and health statistics of the centers for disease control. U.S. Department of Health and Human Services, Washington, DC, pub no. 214
- Schilder AGM, Lok W, Rovers MM (2004) International perspectives on management of acute otitis media: a qualitative review. Int J Pediatr Otorhinolaryngol 68:29–36

- Singh PP, Gupta N (2007) Diagnostic algorithm and standard treatment guidelines for management of common ear conditions. Developed under the Government of India – WHO Collaborative Programme (2006–2007)
- Smith AW, Hatcher J, Mackenzie IJ et al (1996) Randomized controlled trial of treatment of chronic suppurative otitis media in Kenyan schoolchildren. Lancet 348:1128–1133
- Verhoeff M, van der Veen EL, Rovers MM et al (2006) Chronic suppurative otitis media: a review. Int J Pediatr Otorhinolaryngol 70:1–12
- Vesa S, Kleemola M, Blomqvist S et al (2001) Epidemiology of documented viral respiratory infections and acute otitis media in a cohort of children followed from two to twenty-four months of age. Pediatr Infect Dis J 20:574–581
- World Health Organization (1998) Prevention of hearing impairment from chronic otitis media. Report of a WHO/CIBA foundation workshop

