Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 3: Middle Ear and Mastoid

Chapter 28: Acute Otitis Media and Mastoiditis

George E. Shambaugh, Tawfik F. Girgis

Acute otitis media and mastoiditis occupied the major and almost exclusive attention and concern of physicians in the otologic sphere until the advent of specific antimicrobial therapy in the mid-1930s. The strategic location of the middle ear cleft and mastoid air cells separated from the sigmoid sinus and meninges by a mere thin shell of bone makes every infection of the middle ear capable of intracranial extension. As Hippocrates said: "Acute pain of the ear with continued strong fever is to be dreaded, for there is danger that the man may become delirious and die".

With the advent of antibiotics, the majority of patients with acute otitis media and mastoiditis are controlled by medical treatment. Only a few cases do not respond to medical treatment and require surgery to prevent intracranial complications. Although the incidence of draining ears has been reduced remarkably since antibiotics have been used, secretory otitis media has become a very common sequela following good response of otitis media to the antibiotics.

Classification of Otitis Media

Acute

There are three types of acute otitis media.

Bacterial Otitis Media. This is the usual and most common type of middle ear infection.

Viral Myringitis. This may be present with or without otitis media.

Necrotic Otitis Media. This is rarely seen at present. It occurs chiefly in young children who are severely ill or suffer from a systemic disease. It is usually followed by sequelae, including chronic otitis media.

Secretory (Serous) Otitis Media. This is the type that is seen most frequently today, predominantly in children.

Chronic

There are four types of chronic otitis media: (1) suppurative, mucosal type, (2) allergic, (3) tubercular, and (4) with cholesteatoma.

This chapter is concerned with the three types of acute otitis media and mastoiditis. Chronic otitis media is discussed in Chapter 29.

Epidemiology

Extrinsic and intrinsic factors contribute to the incidence of otitis media.

Extrinsic Factors

Seasonal Variations. There is definitely an increased incidence of otitis media during the colder months. Most episodes are preceded by upper respiratory infection.

Intrinsic Factors

Age. The incidence of otitis media diminishes with increasing age. Several reasons may account for this:

1. With increased age, the incidence of upper respiratory infections diminishes.

2. Short, straight eustachian tubes in infants and young children allow more ready access of bacteria to the middle ear. Infants who are few in a supine position are prone to regurgitation through the eustachian tube to the middle ear.

3. Abundance of lymphoid tissue in children as compared with adults produces obstruction of the eustachian tube.

Allergies. Swelling of mucosa around and in the eustachian tube can predispose to recurrent middle ear infections.

Cleft Palate. At least 50 per cent of patients with cleft palate develop recurrent otitis media, frequently with subsequent complications (eg, cholesteatoma, adhesive otitis media).

Pathology and Clinical Course of Acute Bacterial Otitis Media

Acute bacterial otitis media is an extremely common condition, especially in the young. It is so frequent in sick infants that routine examination of the middle ear at autopsy by Bezold (1908) nearly a century ago revealed infection of the middle ear in 80 per cent, although not all showed clinical evidence of infection during life.

Fortunately the great ability of the respiratory mucosa to overcome an acute infection is shared by the mucosa of the tympanic cavity and its extensions into the pneumatic cells of the mastoid and petrous pyramid. As a result, *acute otitis media with mastoiditis is essentially a self-limiting infection that even without treatment tends toward healing, with restitution of the tissues to normal.* Since the majority of cases are due to the beta-hemolytic streptococcus or the

pneumococcus, both of them highly susceptible to penicillin, the usual clinical course is shortened and terminated in most cases by early and adequate antibiotic therapy. However, the five pathologic stages through which an acute otitis media may progress, each with its characteristic clinical manifestations, and after any of which the infection might terminate spontaneously without any treatment, are described here.

Stage 1: Hyperemia

The earliest pathologic change and clinical manifestation of acute bacterial otitis media is hyperemia of the mucosa of the eustachian tube, tympanic cavity, and mastoid cells. The eustachian tube lumen becomes occluded, with alteration of air pressure in the middle ear. When the hyperemia develops slowly, the middle ear air is absorbed with a negative pressure; when the mucosal hyperemia comes on rapidly, the air is under increased pressure. In either event there is a sense of fullness in the ear and mild conductive hearing impairment as a result of unequal air pressure on the two sides of the tympanic membrane. The latter, on inspection, is hyperemic, especially along the handle of the malleus, in the pars flaccida and around the periphery, but the membrane at this early stage is not thickened enough to obscure the landmarks of the short process and malleus handle. Fever and earache may be present but neither is severe at this stage.

Stage 2: Exudation

Hyperemia of the mucoperiosteum of the tympanic cavity, epitympanum, antrum, and mastoid air cells is soon followed by escape from the dilated permeable capillaries of serum, fibrin, red cells, and polymorphonuclear leukocytes. At the same time many of the cuboidal epithelial cells of the tympanic cavity begin to become mucus-producing goblet cells. Very soon the middle ear cavity and mastoid air cells become filled with *exudate under pressure.* The tympanic membrane becomes thickened so that its landmarks are obscured, it bulges markedly, there is a marked conductive hearing loss, and earache and pain are severe. Rapid systemic resorption of toxic products of inflammation by the tympanic and mastoid mucosa results in toxicity and fever, which is often high, especially in infants. In infants and small children, in whom the mastoid cortex is thin and porous, there will be tenderness and edema over the mastoid antrum. By x-ray the tympanic cavity and mastoid air-cell are seen to be diffusely clouded, but without alteration of the cell partitions.

Stage 3: Suppuration

The tympanic membrane perforates spontaneously or by myringotomy, with copious drainage of hemorrhagic or serosanguineous fluid that soon assumes a mucopurulent character. The severe pain toxicity and fever of stage 2 caused by pressure of exudate on inflamed mucosa begin to recede, and mastoid edema and tenderness subside. The tympanic membrane perforation is always in the pars tensa, is always small, and remains just large enough to permit the escape of the exudate. These characteristics are noteworthy and in sharp contrast to the disease known as necrotic otitis media, described later. Radiography of the mastoid continues to show clouding of the tympanum and air-cells, but as yet without bony changes in the cell partitions. A marked

conductive hearing loss persists.

Stage 4: Coalescence

Keeping in mind the self-limiting tendency for acute bacterial otitis media to resolve spontaneously after one of the earlier stages, a small proportion of untreated patients, between 1 and 5 per cent in different epidemics, will continue to have suppuration until progressive hyperemic thickening of the mucoperiosteum begins to obstruct free drainage of mucopurulent secretions. This obstruction is greatest in the mucosal folds of the epitympanum and the smaller air-cells around the antrum. Pus under pressure in the larger peripheral air-cells begins to cause decalcification and osteoclastic resorption of the bony cell partitions, so that the separate air-cells begin to coalesce into larger irregular cavities filled with hypertrophied mucosa, granulations, and pus under pressure. The osteoclastic erosion of bone proceeds in all directions, including the thin inner table and the thicker outer table of the mastoid process, forming an extradural or perisinus abscess within, or a subperiosteal abscess on the outside.

The symptoms and signs of the stage of coalescence are continued mucopurulent discharge from the middle ear for more than 2 weeks, with the purulent component predominating. The discharge fluctuates in amount and is generally greater at night because of increased hyperemic edema in the recumbent posture. Pain and mastoid tenderness, which had improved when the ear began to drain, now recur and also tend to be worse at night, although usually less severe than in stage 2. Low-grade fever and leukocytosis, again less than in stage 2 but now of graver import, accompany the retention of pus under pressure in the mastoid air-cells. Now for the first time radiography begins to show decalcification and destruction of mastoid air cell partitions. The marked conductive hearing loss continues.

Additional signs of coalescent mastoiditis are sagging of the posterosuperior bony meatal wall as a result of periosteal thickening adjacent to the mastoid antrum, best detected by comparing the osseous meatus of the normal and diseased side; periosteal thickening and deep bone tenderness over the mastoid tip, best detected by standing behind the patient and palpating both sides simultaneously; and nipple formation of the tympanic membrane perforation as a result of protrusion of edematous membrane.

Complications

Extension of bacterial infection beyond the confines of the tympanic cavity and mastoid air-cells produces a complication. Most frequent is a subperiosteal mastoid abscess, a perisinus abscess, or an extradural abscess. Less frequent but more serious is leptomeningitis, sigmoid sinus thrombophlebitis, brain abscess, suppurative labyrinthitis, petrositis, or facial nerve palsy.

The pathologic process by which a complication develops depends on the stage of the acute otitis media. Early, in stage 1, 2 or 3, there may be a progressive thrombophlebitis of venules in the tympanic and mastoid mucoperiosteum that can extend through intact bone to the sigmoid sinus, meninges, brain, labyrinth, or facial nerve. The beta-hemolytic streptococcus and

type III pneumococcus are particularly capable of producing a complication by thrombophlebitic extension.

Later, in stage 4, complications are the result of the progressive bone erosion of coalescent mastoiditis. To understand exactly how the bone erosion proceeds, one cannot improve upon the description of Scheibe (1904) written more than 90 years ago.

The lining of the cells contains very many blood vessels, is 40 to 80 times as thick as in the normal and changed into a lobulated granulation tissue rich in lymphocytes. The bony inner surface of the cells is eroded all over by small dimples, and in each one there is a multinucleated giant cell (osteoclast). These erosions extend over the adjoining bony canals and cavities containing marrow. They become enlarged and contain more cells than normal. Little by little in all directions an excentrical enlargement of the cavities filled with pus is thus effected, until it reaches the dura inwardly, or most frequently the protruding wall of the sinus, and outwardly the periosteum at some place of the mastoid process. Thus a fistulous formation occurs on the lateral surface if the process is not interfered with. The formation of osteoclasts and their bone-wrecking occupation comes to a standstill as soon as the pus is evacuated, and a layer of cells containing one nucleus takes their place on the walls of the diseased spaces. They are the osteoblasts which at once form osteoid substances and this later on becomes ossified all around the focus. At a greater distance from the focus of suppuration the occupation of the osteoblasts and the formation of osteoid tissue begins long before the evacuation of pus.

It is most important for the surgeon to recognize that the altered bone in acute coalescent mastoiditis is not necrotic as so often erroneously described in the operative notes. Rather, the bone is softened by decalcification and osteoclasis, but it is living bone with the ability to form new bone as soon as the pus under pressure is relieved.

Acute Necrotic Otitis Media

This special form of acute bacterial otitis media occurs chiefly in children severely ill with exanthem or other systemic infection. Although rarely seen today because of more frequent antibiotic treatment of otitis media, this peculiar disease has an importance out of proportion to its frequency because of its sequelae, which are still encountered in adults who acquired this disease in childhood. Because very few today's otologists have had the opportunity to observe the early stages of otitis media in children acutely ill with measles and scarlet fever, the great majority are completely unfamiliar with the clinical picture and pathology of this type of acute otitis media. The senior author of this chapter served as otologic consultant for some years in the preantibiotic era at the Chicago Municipal Contagious Disease Hospital, where he had a unique opportunity to observe cases of acute necrotic otitis media, to operate on some of the patients for meningitis or other complications, and to see the late sequelae.

In cases of acute necrotic otitis media, from the very onset of the middle ear infection, impaired host resistance combines with the necrotizing toxins of the virulent organism, most often the beta-hemolytic streptococcus, to cause actual necrosis of localized gangrene of tissue. The

necrotizing toxins first cause death of tissue with the poorest blood supply, starting with the part tensa of the tympanic membrane between its periphery and the malleus handle. The devitalized tissue soon sloughs, leaving a large, typically kidney-shaped perforation within a day or two of the onset of ear symptoms. In more severe cases the entire pars tensa sloughs, the malleus handle becomes necrotic, and the mucosa on the promontory sloughs, leaving bare white bone. Rarely the necrotic process may include the bony sulcus tympanicus, the entire ossicular chain, and bone in the walls of the attic and antrum. In one case observed by the author in the preantibiotic days in a small child desperately ill with scarlet fever, the entire mastoid process at operation was found to consist of dead-white, bloodless, necrotic bone, while the adjacent dura was leathery and bloodless. In another patient with scarlet fever otitis media, the labyrinthine capsule around the semicircular canals of both sides became necrotic and finally formed sequestra that were subsequently removed. Miraculously the cochlea was not involved on one side and some hearing remained in this child, but there was total loss of vestibular response from both ears.

Bezold (1908), with characteristic clarity of expression, compared the usual self-limiting acute bacterial otitis media with acute necrotic otitis. Of the usual acute otitis media, he said:

The suppuration may have run its course in a few days, but it may also last several weeks or months with or without interruptions. I have seen it last even longer than a year in some cases, and yet it healed, the perforation closing and the normal function returning. I have *never* in an otherwise healthy organism, seen a *genuine* suppuration of the middle ear become chronic or develop a persisting or enlarging perforation.

In sharp contrast to the usual acute otitis media, Bezold then noted the peculiar type sometimes encountered during the exanthema:

The clinical picture in the ear differs totally from the very beginning from that in genuine suppuration of the middle ear. I saw, for example, discolored fetid secretions in the meatus of an ear in which an extensive perforation had taken place during the previous night ... I saw another case where within six days the whole tympanic membrane was destroyed and the handle of the malleus became necrotic, at the height of the scarlet fever.

The death of tissue in acute necrotic otitis media occurs early in the infection during the most toxic stage of the exanthema. Owing to the loss of tissue, acute necrotic otitis media is always followed by residual irreversible pathology, which takes one of the following forms:

1. When the necrotizing process is confined to the pars tensa, it results in a large, usually kidney-shaped perforation. Sometimes this heals by a thin atrophic scar in the center of a permanently thickened tympanic membrane. Sometimes a large centrally located perforation persists, with mucosa of the middle ear left exposed to dust, debris, and bacteria from the external meatus, and with increased susceptibility to ascending infections through the eustachian tube.

2. When the necrotizing process includes the annulus and sulcus tympanicus and portions of the mucosa of the middle ear, healing finally occurs by ingrowth of stratified squamous epithelium from the external meatus to cover the denuded promontory, usually extending into the attic and antrum. Desquamation of the cornified layer accumulates in the attic and antrum as a cholesteatoma. Because it is secondary to an acute necrotic otitis media, such a cholesteatoma may be termed secondary to distinguish it from primary congenital cholesteatoma and attic retraction cholesteatoma with an intact part tensa.

The diagnosis and treatment of secondary cholesteatoma is considered in subsequent chapters.

3. In exceptionally severe and extensive necrotic otitis media, areas of bone in the wall of the attic or antrum become devitalized. When large they eventually separate from the adjacent living bone and form sequestra. Chronic suppuration from this foreign body will continue until it is removed. When the area of bone necrosis is smaller, it remains ass a focus of chronic osteitis or osteomyelitis with chronic suppuration until the focus of bone caries is removed.

Prophylaxis of Recurring Acute Otitis Media

As mentioned earlier, the most common predisposing factor to acute bacterial otitis media is impaired ventilation of the eustachian tube. Deficient host resistance to the specific organism, also noted above, accounts for the high frequency of *Haemophilus influenzae* otitis media in children under the age of 5. Impaired resistance in severe general infections, such as influenza, exanthemata and agammaglobulinemia, predisposes to bacterial invasion of the middle ear. In children who have recurrent attacks of otitis media and do not have cleft palate, the usual etiology is either hypertrophied adenoids or allergy. If it is adenoids, adenoidectomy is indicated, possibly with tonsillectomy if the child has had recurrent attacks of tonsillitis. Perennial nasal allergy can be due to inhalant or food allergy. Most commonly this will be a house dust or mold allergy, best controlled by the optimum dosage method of Hansel as determined by Rinkel's (1951) skin titration technique. An underlying food allergy should be suspected when the child gives a history of infantile eczema or colic, in which case the usual offender is likely to be cow's milk. Other possible factors are foods taken daily or frequently, such as wheat, eggs, and orange or apple juice. Methods of diagnosing the offending food include trial by elimination, Rinkel's provocative feeding test after four days' omission, the intradermal or sublingual provocative-neutralization tests of Lee (1969) and (1972) Miller, and the Drs. Bryans' (1967) cytotoxic food test. The details of allergic diagnosis and management are outside the province of this chapter and can be found in the writings of Rinkel et al (1951), Lee et al (1969), Miller (1972), Derlacki (1951), Rapp (1981), and the Drs. Bryan (1967).

In rare instances, agammaglobulinemia is the etiology and has to be corrected to prevent recurrent middle ear infection. Insertion of ventilation tubes is indicated in recurrent secretory otitis media, especially when secretory otitis media persists in between attacks of acute otitis media and when allergic therapy fails to control the problem.

Diagnosis of Acute Otitis Media

Sometimes diagnosis in the child is difficult because of the small ear canal, difficulty of cleaning cerumen, and congestion of the tympanic membrane when the child is screaming, thus resembling acute otitis media. Good lighting, magnification, and dexterity in cleaning cerumen are paramount for the diagnosis. Using the pneumatic otoscope helps in evaluating the mobility of the tympanic membrane. The presence of effusion in the middle ear produces diminished mobility of the tympanic membrane. This can be confirmed by tympanometry, in which a type II tympanogram with a flat curve is obtained. An audiogram usually shows a 20 to 30 decibel hearing loss for the speech frequencies.

As pressure increases in the tympanic cavity, the tympanic membrane ruptures. Drainage is usually sanguineous at first, followed by purulent discharge. If this is cultured within the first few hours, reliable results can be obtained. However, later on, there are too many contaminants from the external canal, thus making a culture unreliable. Throat cultures are also unreliable, since they do not usually indicate the nature of the organism in the tympanic cavity.

Treatment and Prophylaxis of Acute Bacterial Otitis Media and Mastoiditis

As we have seen, the mucoperiosteum of the mastoid antrum and pneumatic cells is involved in all but the earliest cases of acute bacterial otitis media. The self-limiting tendency toward spontaneous resolution is great even without antibiotic therapy, with return of the structures to normal, including normal hearing, in nearly all cases. The value of antibiotics is to arrest the process in its early stages so as to abort a possibly severe febrile illness that might otherwise last days to weeks with the possibility of complications.

Pediatricians in increasing numbers are prescribing prophylactic antibiotics during winter months for children with frequently recurring otitis media. The most widely used antibiotics are Gantanol, Gantrisin, and Septra, prescribed as a maintenance dose of half the therapeutic dose. The authors question this practice as well as the practice of prescribing a broad-spectrum antibiotics such as amoxicillin for every mild earache because this alters the normal flora of the intestinal tract, leading to overgrowth of *Candida albicans.* Candidiasis is an increasing problem in children as a result of overuse of broad-spectrum antibiotics. The symptoms of this condition are well described in Crook's "The Yeast Connection". With appropriate anti-*Candida* dietary measures combined with Nystatin or Candistat the symptoms of chronic ill health are soon alleviated.

The prophylaxis of frequently recurring otitis media in children requires recognition of common food allergies, so frequent during the first 5 years of life. With complete elimination of the offending foods or foods, the adoption of Rinkel's rotary, diversified diet, and sublingual drops of the neutralizing dose for foods that react, the cycle of one attack after another of acute middle ear infections is broken without overuse of broad-spectrum antibiotics. The publication by Rapp (1980) on otitis media in children is recommended.

The therapeutic treatment of acute otitis media occurring at any age requires first that the diagnosis has been made by otoscopic examination. Pain in the ear is not sufficient evidence of otitis media, since referred pain from an erupting tooth in a child or a diseased tooth in an adult can produce a severe earache. Other common conditions that cause pain in the ear without a middle ear infection are temporomandibular joint problems, parotitis, and cervical adenitis.

Viral infections of the ear, diagnosed by the presence of bullae on the tympanic membrane and adjacent meatal wall, can be extremely painful; however, unless the hearing is impaired and there is fever, antibiotics are not indicated. A typical episode of acute otitis media will be characterized by redness and thickening of the tympanic membrane with loss of its landmarks and with impaired hearing and fever.

Ideally one should identify the causative organism in order to prescribe the appropriate antibiotic. When the thickened tympanic membrane is bulging, myringotomy relieves the severe pain and affords the opportunity to obtain a culture from the tip of the myringotomy knife. Once the tympanic membrane has ruptured and the ear is draining, culture made from the suppurative material in the meatus will be contaminated by meatal organisms and will indicate less accurately the pathogenic organism. Practically, one usually prescribe an antibiotic without culture before the ear begins to drain, thus aborting a severe infection.

In children under the age of 5, *Haemophilus influenzae* is a frequent pathogen. Some strains of this organism produce beta-lactamase and are therefore resistant to ampicillin and amoxicillin, the antibiotics usually prescribed for this organism. Cefaclor, a second-generation cephalosporin, is effective against nearly all pathogens causing otitis media in children, the exception being some strains of beta-lactamase-producing *Branhamella catarrhalis.* Septra, a combination of trimethoprim and sulfamethoxazole, is also effective against nearly all pathogenic organisms causing otitis media. A newer antibiotic, a combination of amoxicillin and potassium clavulanate (Augmentin), is recommended by Bluestone (1984) "as successfully eliminating almost all the bacterial pathogens that commonly cause acute otitis media within three to six days. While equal or possibly superior to cefaclor, Augmentin may cause more adverse effects, usually gastrointestinal".

Acute otitis media in older children and adults is most often of streptococcal or pneumococcal origin. Erythromycin orally is effective in most cases, with less alteration of intestinal flora and fewer side effects than broad-spectrum antibiotics such as amoxicillin and ampicillin. The rule that an antibiotic should be prescribed for at least 7 to 10 days ensures that the infection will not flare due to undertreatment. A recent article by Klein and Daum (1985) lists the useful antibiotics currently available for acute otitis media (Table 10.

Table 1. Antibiotics Useful in Otitis Media

Drug	Total Daily Dose (mg/kg)	No of Divided Doses
Amoxicillin	25-50	3
Ampicillin	50-100	4
Amoxicillin/clavulanate potassium	40 / 10	3
Trimethoprim/sulfamethoxazole	8 / 40	2
Erythromycin/sulfamethoxazole	50 / 150	4
Cefaclor	40	3
Bacampicillin	80	2
Cyclacillin	50	4.

Incision and drainage of a fluctuant subperiosteal abscess is occasionally indicated in a neglected acute otitis media. Mastoidectomy for coalescent mastoiditis is almost never needed today, so effective are antibiotics for acute otitis media. Nasal decongestants locally and systemically are often prescribed in children and adults with an acute otitis media, with a possibly beneficial effect on the upper respiratory infection that accompanies acute otitis media.

Conclusion

In conclusion, it is well to recapitulate the salient features of viral myringitis, acute bacterial otitis media, and acute necrotic otitis media.

Acute viral myringitis is easily distinguished from an acute otitis media by the normal hearing and absence of significant fever. It is a self-limited process. Puncture of the hemorrhagic blebs in the tympanic membrane and meatal wall can be done to relieve severe pain. Perforation of the tympanic membrane is not indicated.

Acute bacterial otitis media, most often due to the beta-hemolytic streptococcus or pneumococcus and, in children under 5, to *Haemophilus influenzae*, runs a characteristic clinical and pathologic course with the possibility of a complication that may occur early but is more likely to occur in the stage of coalescent mastoiditis. Early and adequate antibiotic therapy nearly always arrests the inflammatory process in an early stage, minimizing the risk of a complication. Adequate antibiotic treatment includes selecting an antibiotic to which the causative bacterium is sensitive and giving it until all evidence of otitis media has resolved, usually for at least 10 days. Myringotomy, with culture of the tip of the myringotomy knife, is the best method of ascertaining the organism causing the middle ear infection. If the patient is first seen in the stage of exudation with bulging of the thickened tympanic membrane and severe pain, myringotomy also helps to relieve the pain. Many cases of acute bacterial otitis media will resolve with antibiotic treatment begun early in stage 1, obviating the need for myringotomy. Mastoid surgery is hardly ever required today for acute bacterial otitis media and mastoiditis, the overwhelming majority of cases resolving spontaneously or with the help of antibiotic treatment when the appropriate antibiotic is utilized in full therapeutic doses. The rare case of coalescent mastoiditis

with or without a complication should be treated by the simple or complete mastoid operation combined with appropriate antibiotic therapy. The details of the indications and technique of this procedure will be found in another chapter of this volume.

Acute bacterial otitis media throughout its course, even to the stage of coalescent mastoiditis and without antibiotic treatment, remains a self-limiting type of infection which, if the patient does not succumb to intracranial extension, eventually heals with an intact tympanic membrane and normal hearing. We can agree completely with Bezold (1908) that never, in an otherwise healthy patient, have we seen an acute bacterial otitis media become chronic or develop a persisting or enlarging perforation.

Quite the contrary is acute necrotic otitis media, rarely seen today, but which accounts for nearly all large persisting perforations of the tympanic membrane, for cholesteatoma of the attic and antrum associated with large pars tensa perforations, and for occasional cases of chronic osteitis or osteomyelitis without cholesteatoma.