PROBLEM BASED LEARNING
KELAS REGULER
SISTEM INDRA KHUSUS

- Modul Gangguan Pendengaran

- Modul Gangguan Penciuman

Diberikan Pada Mahasiswa Semester V
Fakultas Kedokteran Unhas

Fakultas Kedokteran
Universitas Hasanuddin
2016

MODUL THT (Tutorial 1 & 2)

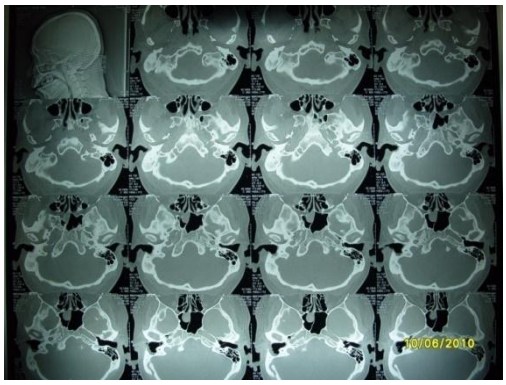
FACULTY OF MEDICINE HASANUDDIN UNIVERSITY
OBJECTIVE STRUCTURED ORAL CASE ANALYSIS
SPECIAL SENSE SYSTEM
OTORHINOLARYNGOLOGY
ACADEMIC YEAR 2013-2014

CASE 1

Women, A, 25 years came to the clinic with chief complaints of hearing loss in right ear since 1 year ago accompanied by a purulent ear charged, smells, sometimes pain and itching and headache. No complaints of vertigo. History of frequent ear probe, a history of the same disease is often recurrent, a family history of atopy denied.

Physical examinations:

General conditions: Good, not anemic. Height: 160 cm, Weight: 60 kg.
Vital signs are within normal limit.
Inspection : Auricel normal
Palpation : No edema and no tenderness of tragus and retroauricel
Otoskopi : Right external acoustic meatus and right tympanic membrane hyperemic, subtotal central perforation of right tympanic membrane, mucoid secretions in right tympanic cavity and the external acoustic meatus
Left acoustic meatus and left tympanic membrane normal
Faringoscopy : Within normal limits
Anterior Rhinoscopy : Nasal cavity, nasal septum and nasal turbinate normal
Laboratory examination: Hb 14 g / dl, leukocytes 12,600 g%, LED 45 / 1 hour
Pure Tone Audiometry Examination :
Ear artery: Mild-Severe Hearing Loss Conductive (60 dB)
Ear Sinistra: Normal Hearing (20 dB)
Examination of vestibular function: no canal paresis
Chest X-Ray : Normal
Mastoid CT-Scan :



Diagnosis : Right Chronic Supurative Otitis Media

BASED ON THE ABOVE CASE, EACH STUDENTS ARE ASSIGNED TO :

1. **MAKE A MIND MAP OF CHRONIC SUPURATIVE OTITIS MEDIS:**
2. **SYMPTOMS AND SIGNS OF HEARING DISTURBANCE**
3. **EXPLAIN THE ETIOLOGY AND PATOFISIOLOGY CHRONIC SUPURATIVE OTITIS MEDIS.**
4. **EXPLAIN THE CLINICAL MANIFESTATION CHRONIC SUPURATIVE OTITIS MEDIS, INCLUDE THE SIGN AND SIMPTOMS.**
5. **EXPLAIN THE TREATMENT, COMPLICATION AND PROGNOSIS.**
6. **PRESENT AND DISCUSS IN CLASS**

FACULTY OF MEDICINE HASANUDDIN UNIVERSITY
OBJECTIVE STRUCTURED ORAL CASE ANALYSIS
SPECIAL SENSE SYSTEM
OTORHINOLARYNGOLOGY
ACADEMIC YEAR 2011-2012

CASE 1

Women, A, 25 years came to the clinic with chief complaints of hearing loss in right ear since 1 year ago accompanied by a purulent ear charged, smells, sometimes pain and itching and headache. No complaints of vertigo. History of frequent ear probe, a history of the same disease is often recurrent, a family history of atopy denied.

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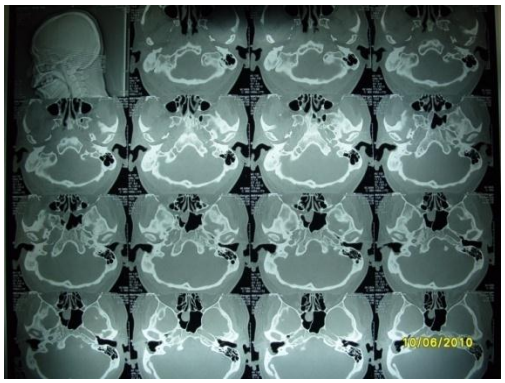

Diagnosis : Right Chronic Supurative Otitis Media

Therapy : Antibiotics, antiinflammation, nasal dekongestan dan mucolitic

EACH STUDENTS ARE ASSIGNED TO :

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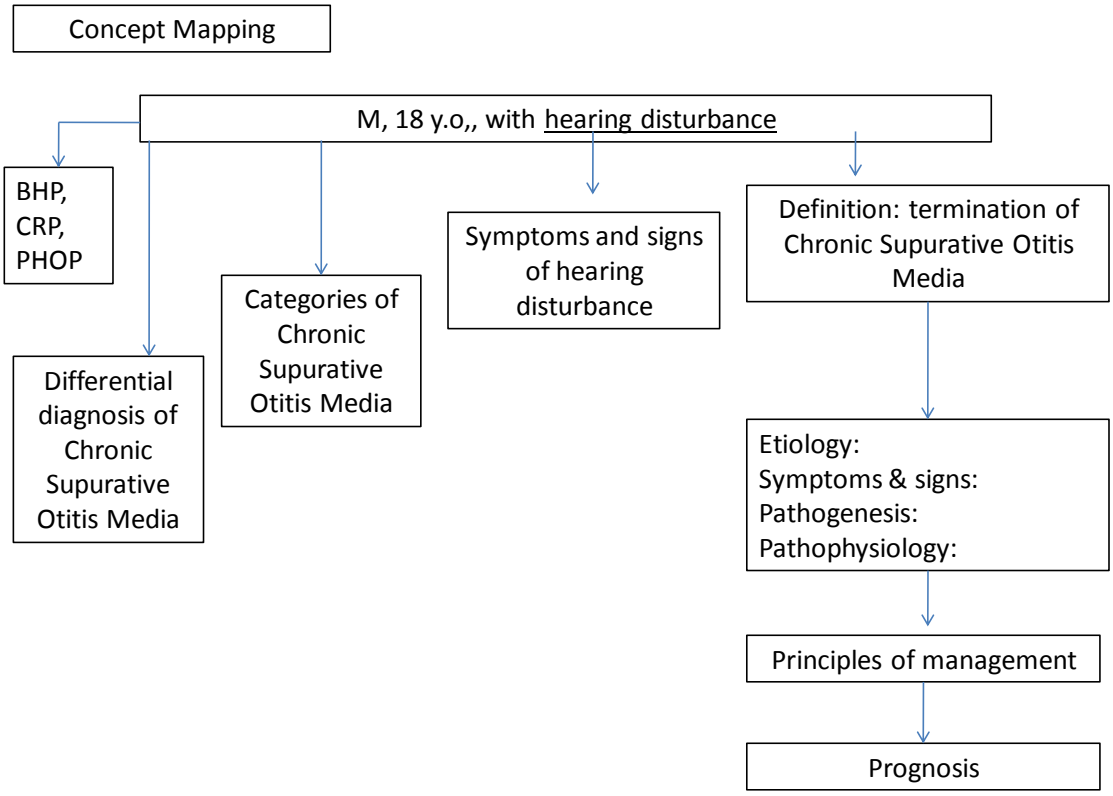
FACULTY OF MEDICINE HASANUDDIN UNIVERSITY
OBJECTIVE STRUCTURED ORAL CASE ANALYSIS
SPECIAL SENSE SYSTEM
OTORHYNOLARYNGOLOGY
Final Scoring Sheet Case (example)

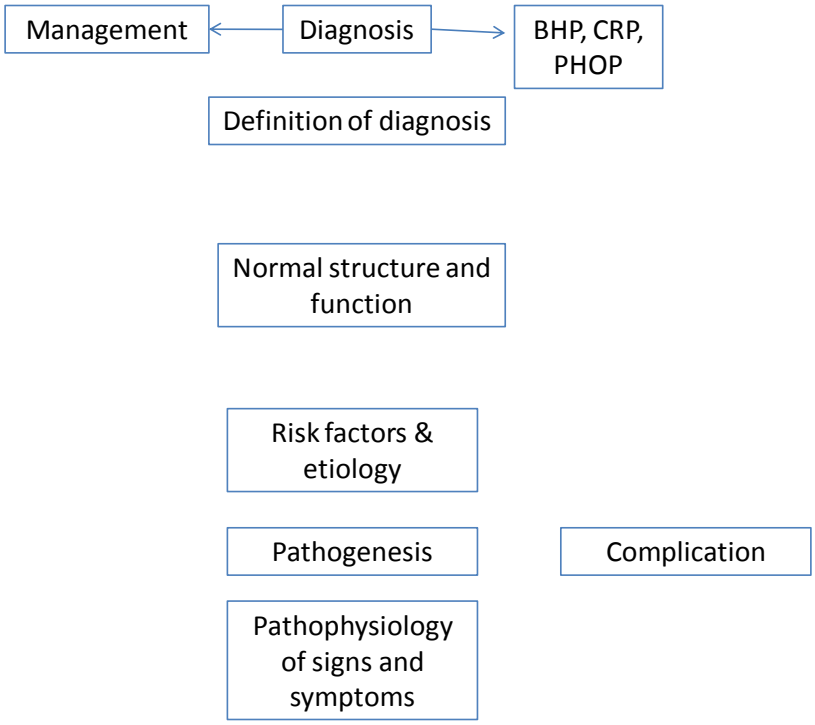
Student's Name :	**Student's Batch Number :**
Examiner 1 Name : Signature :	**Examiner 2** Name : Signature :

No	Issues to Analyze	Max Score	Student's Score
1	Case review	**5**	
2	Concept mapping	**15**	
3	Symptoms and signs of hearing disturbance	**5**	
4	Etiology of chronic supurative otitis media	**5**	
5	Symptoms and signs of chronic supurative otitis media	**5**	
6	Pathogenesis of chronic supurative otitis media	**10**	
7	Pathophysiology of symptoms and signs of chronic supurative otitis media	**20**	
8	Principles of management threatened chronic supurative otitis media	**5**	
9	Prognosis of threatened chronic supurative otitis media	**3**	
10	Categories of supurative otitis media	**3**	
11	Differential diagnosis of chronic supurative otitis media	**4**	
12	BHP (4), CRP (3), PHOP (3)	**10**	
13	Performance a. Attitude (4) b. systematic presentation (3) c. communication skill (3)	**10**	
TOTAL SCORE		100	

TUTOR'S GUIDE

1. Mind Map




Management
Diagnosis
BHP, CRP, PHOP
Definition of diagnosis
Normal structure and function
Risk factors & etiology
Pathogenesis
Complication
Pathophysiology of signs and symptoms

2. CHRONIC SUPURATIVE OTITIS MEDIA

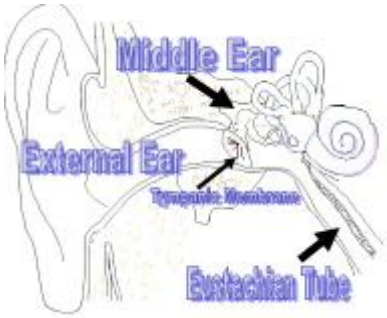
Background

Chronic suppurative otitis media (CSOM) is a perforated tympanic membrane with persistent drainage from the middle ear. It is a disease that is well known to otolaryngologists. CSOM is defined as chronic otorrhea (ie, lasting >6-12 wk) through a perforated tympanic membrane.[1, 2] Chronic suppuration can occur with or without cholesteatoma, and the clinical history of both conditions can be very similar. The treatment plan for cholesteatoma always includes tympanomastoid surgery with medical treatment as an adjunct.

CSOM differs from chronic serous otitis media in that chronic serous otitis media may be defined as a middle ear effusion without perforation that is reported to persist for more than 1-3 months. The chronically draining ear in CSOM can be difficult to treat.[3] McKenzie and Brothwell demonstrated evidence of chronic suppurative otitis in a skull found in Norfolk, United Kingdom, which is thought to be from the Anglo-Saxon period.[4] Radiologic changes in the mastoid caused by previous infection have been seen in a number of specimens, including 417 temporal bones from South Dakota Indian burials and 15 prehistoric Iranian temporal bones.[5, 6]

Anatomy

The middle ear cleft can be thought of as a 6-sided cube. Its lateral boundary, the tympanic membrane, separates it from the outer ear. Its medial boundary is formed by the promontory, which denotes the basal turn of the cochlea. Anteriorly, it is related to the tendon of tensor tympani superiorly and the opening of the eustachian tube inferiorly. Posteriorly, it is related superiorly to the aditus, which connects the middle ear cavity with the mastoid antrum, and inferiorly to the facial ridge. The roof of the middle ear cavity is formed by the tegmen tympani, and the floor of the middle ear cavity lies in close relation to the jugular foramen. (See the image below displaying anatomy of the ear).



Anatomy of the external and middle ear.

The anterior and posterior malleolar folds, which radiate from the short process of the malleus, form the boundary between the pars flaccida and the pars tensa, which lie above and below it, respectively. Atticoantral disease predominantly affects the pars flaccida, and tubotympanic disease affects the pars tensa.

The middle ear cavity also consists of the ossicular chain (malleus, incus, and stapes). The ossicular chain connects the tympanic membrane, in which the handle of the malleus is embedded, to the oval window, on which sits the footplate of the stapes. In atticoantral disease, the ossicular chain is frequently affected by cholesteatoma, thereby causing hearing loss. The malleus and or incus may be sacrificed if they are extensively involved by cholesteatoma. In these cases, a planned second-stage reconstruction is often appropriate.

Pathophysiology

CSOM is initiated by an episode of acute infection. The pathophysiology of CSOM begins with irritation and subsequent inflammation of the middle ear mucosa. The inflammatory response creates mucosal edema. Ongoing inflammation eventually leads to mucosal ulceration and consequent breakdown of the epithelial lining. The host's attempt at resolving the infection or inflammatory insult manifests as granulation tissue, which can develop into polyps within the middle ear space. The cycle of inflammation, ulceration, infection, and granulation tissue formation may continue, eventually destroying the surrounding bony margins and ultimately leading to the various complications of CSOM.[7, 8]

Common bacteria

Pseudomonas aeruginosa, Staphylococcus aureus, Proteusspecies, Klebsiella pneumoniae, and diphtheroids are the most common bacteria cultured from chronically draining ears. Anaerobes and fungi may grow concurrently with the aerobes in a symbiotic relationship. The clinical significance of this relationship, although unproven, is theorized to be an increased virulence of the infection. Understanding the microbiology of this disease enables the clinician to create a treatment plan with the greatest efficacy and least morbidity.

P aeruginosa is the most commonly recovered organism from the chronically draining ear. Various researchers over the past few decades have recovered pseudomonads from 48-98% of patients with CSOM.

S aureus is the second most common organism isolated from chronically diseased middle ears. Reported data estimate infection rates from 15-30% of culture-positive draining ears. The remainder of infections are caused by a large variety of gram-negative organisms. *Klebsiella* (10-21%) and *Proteus* (10-15%) species are slightly more common than other gram-negative organisms.

Polymicrobial infections are seen in 5-10% of cases, often demonstrating a combination of gram-negative organisms and *S aureus*. The anaerobes (*Bacteroides*, *Peptostreptococcus*, *Peptococcus*) and fungi (*Aspergillus*, *Candida*) complete the spectrum of colonizing organisms responsible for this disease. The anaerobes make up 20-50% of the isolates in CSOM and tend to be associated with cholesteatoma. Fungi have been reported in up to 25% of cases, but their pathogenic contribution to this disease is unclear.

Etiology

The diagnosis of CSOM requires a perforated tympanic membrane. These perforations may arise traumatically, iatrogenically with tube placement, or after an episode of acute otitis media, which decompresses through a tympanic perforation.[3, 9, 10, 11]

The mechanism of infection of the middle ear cleft is postulated to be translocation of bacteria from the external auditory canal through a perforation into the middle ear. Some authors suggest that the pathogenic organisms may enter through reflux of the eustachian tube. The data supporting this theory are inconclusive. Most of the pathogenic bacteria are those common to the external auditory canal.

The risk of developing otorrhea (but not necessarily CSOM) through a ventilation tube is reportedly 21-50%. Annually, more than a million tubes are placed in the United States for recurrent otitis media and otitis media with effusion. Studies have reported that 1-3% of patients with ventilation tubes develop this disease.

The risk of developing CSOM increases with the following circumstances[12]:

- A history of multiple episodes of acute otitis media
- Living in crowded conditions
- Day care facility attendance
- Being a member of a large family

Studies trying to correlate the frequency of the disease with parental education, passive smoke, breastfeeding, socioeconomic status, and the annual number of upper respiratory tract infections are inconclusive.

Patients with craniofacial anomalies are special populations at risk for CSOM. Cleft palate, Down syndrome, cri du chat syndrome, choanal atresia, cleft lip, and microcephaly are other diagnoses that increase the risk of CSOM, presumably from altered eustachian tube anatomy and function.

Epidemiology

The larger the tympanic membrane perforation, the more likely the patient is to develop CSOM. Some studies estimate the yearly incidence of CSOM to be 39 cases per 100,000 persons in children and adolescents aged 15 years and younger. In Britain, 0.9% of children and 0.5% of adults have CSOM. In Israel, only 0.039% of children are affected.[13]

Certain population subsets are at increased risk for developing CSOM. The Native American and Eskimo populations demonstrate an increased risk of infection. Eight percent of Native Americans and up to 12% of Eskimos are affected by CSOM. The anatomy and function of the eustachian tube play a significant role in this increased risk. The eustachian tube is wider and more open in these populations than in others, thus placing them at increased risk for nasal reflux

of bacteria common to acute otitis media and recurrent acute otitis media and leading to more frequent development of CSOM.

Other populations at increased risk include children from Guam, Hong Kong, South Africa, and the Solomon Islands. The prevalence of CSOM appears to be distributed equally between males and females.Exact prevalence in different age groups is unknown; however, some studies estimate the yearly incidence of CSOM to be 39 cases per 100,000 in children and adolescents aged 15 years and younger.[12]

Prognosis

Patients with CSOM have a good prognosis with respect to control of infection. The recovery of associated hearing loss varies depending on the cause. Conductive hearing loss can often be partially corrected with surgery. The goal of treatment is to provide the patient a safe ear.

Much of the morbidity of CSOM comes from the associated conductive hearing loss and the social stigma of an often fetid fluid draining from the affected ear. The mortality of CSOM arises from associated intracranial complications. CSOM itself is not a fatal disease. Although some studies report sensorineural hearing loss as a morbid complication of CSOM, other evidence conflicts with this claim

Complications of Disease

In the present era of antibiotics, complications from CSOM are rarely seen because of early antibiotic intervention. However, surgery does play an important role in managing CSOM with or without cholesteatoma.

CSOM without prompt, proper treatment can progress to a variety of mild to life-threatening complications that can be separated into 2 subgroups: intratemporal and intracranial.[14] Intratemporal complications include petrositis, facial paralysis, and labyrinthitis. Intracranial complications include lateral sinus thrombophlebitis, meningitis, and intracranial abscess. Sequelae include hearing loss, acquired cholesteatoma, and tympanosclerosis.

Petrositis

Petrositis occurs when the infection extends beyond the confines of the middle ear and mastoid into the petrous apex. Patients may present with Gradenigo syndrome (ie, retro-orbital pain, aural discharge, and abducens palsy). A CT scan of the head and temporal bone helps define the extent of the disease, diagnose any intracranial spread, and plan a surgical approach. Treatment includes systemic antibiotics with petrosectomy.

Facial paralysis

Facial paralysis can be observed in CSOM with or without cholesteatoma. Surgical exploration with removal of diseased mucosa, granulation tissue, and inspissated pus (usually by mastoidectomy) should be undertaken promptly.

Labyrinthitis

Labyrinthitis occurs when the infection spreads to the inner ear. This may happen emergently or over an extended period. The infection gains access to the inner ear through the round and oval windows or through one of the semicircular canals exposed by bony erosion. The 4 categories of labyrinthitis have been recognized as acute serous, acute suppurative, chronic, and labyrinthine sclerosis.

The symptoms of acute serous labyrinthitis are acute onset of vertigo and hearing loss. Early surgical exploration to remove the infection mitigates damage to the labyrinth.

Patients with acute suppurative labyrinthitis present with profound hearing loss, tinnitus, and vertigo with associated nausea and vomiting. Patients initially demonstrate nystagmus with the rapid component directed toward the affected ear; they later demonstrate nystagmus away from the affected ear after destruction of the membranous labyrinth. Treatment includes aggressive surgical debridement (including labyrinthectomy) to prevent the possibly lethal intracranial complications of meningitis or encephalitis. Administration of broad-spectrum antibiotics with cerebrospinal fluid penetration is also necessary. Culture and sensitivities should direct any changes in the antibiotic regimen.

Chronic labyrinthitis is characterized by the gradual onset of vertigo, tinnitus, and hearing loss. Most commonly, the infection reaches the labyrinth through the lateral canal. Treatment involves mastoidectomy, culture, and appropriate medical therapy.

Labyrinthine sclerosis occurs as the inflammation in the labyrinth causes the body to replace it with fibrous tissue and new bone.

Lateral sinus thrombophlebitis

Lateral sinus thrombophlebitis occurs as the infection extends through the mastoid bone into the sigmoid or lateral sinus. The infected thrombus may release septic embolic causing distal infarcts. Patients present with altered mental status, possible seizures, and fever. Mastoidectomy with surgical excision of the thrombus and culture-directed antimicrobial treatment are the first steps in the management of sinus thrombophlebitis.

Meningitis

Meningitis develops as a consequence of direct or hematogenous spread of the infection. If meningitis is suspected, a lumbar puncture should be performed to recover the causative organism for culture and sensitivity prior to the initiation of empiric broad-spectrum antibiotic

therapy. When stable, patients are taken to the operating room for surgical removal of the cholesteatoma or middle ear infection. Patients with CSOM may develop intracranial abscesses, but it is rare.

Intracranial abscesses

The various intracranial abscesses that may occur can be extradural, subdural, or parenchymal.

A patient with an extradural abscess may present with meningitic signs and symptoms or may be asymptomatic. Regardless of the presentation, imaging to define the abscess should be acquired, and the abscess should be drained with the assistance of neurosurgeons as needed.

Patients with subdural abscesses are very ill and exhibit meningeal signs, possible seizures, and hemiplegia. Prompt neurosurgical consultation, adequate imaging, drainage, and antibiotics are the appropriate treatment. Otologic surgery to remove the nidus of infection is necessary once the patient has stabilized.

Parenchymal abscesses occur as the infection spreads through the tegmen tympani or tegmen mastoideum to the temporal lobe or the cerebellum. Their presentation may be indolent, as this disease initially grows in "silent" areas of the brain. However, if the clinician suspects intracranial involvement, the previous plan of imaging, neurosurgical drainage, and antibiotic therapy is the standard of care.

Conductive hearing loss as a consequence of CSOM may result from the perforated tympanic membrane, a disruption in the ossicular chain, or both. Surgical removal of the infection and cholesteatoma with ossicular chain reconstruction mitigates morbidity associated with decreased hearing.

Diagnostic Considerations

Problems to be considered include the following:

- Langerhans cell histiocytosis
- Neoplasia
- Foreign body
- Cholesteatoma
- Sigmoid sinus thrombosis
- Brain abscess
- Otitic hydrocephalus
- Extradural abscess
- Meningitis
- Tuberculosis
- Petrositis
- Labyrinthitis
- Wegener Granulomatosis

Approach Considerations

Reasonable chronic suppurative otitis media (CSOM) treatment plans can be developed without lab studies. Prior to instituting systemic therapy, a culture should be obtained for sensitivity.

If CSOM is unresponsive to medical treatment, a fine-cut CT scan of the temporal bone may provide an explanation. A fine-cut CT scan may reveal bone erosion from a cholesteatoma, ossicular erosion, involvement of the petrous apex, coalescent mastoiditis, erosion of the fallopian canal, and a subperiosteal abscess. Possible reasons for failed treatment include an occult cholesteatoma or a foreign body.

CT scanning is a necessary adjunct to treatment if the clinician suspects a neoplasm or anticipates intratemporal or intracranial complications.

MRI scans of the temporal bone and brain should be obtained if intratemporal or intracranial complications are suspected. By clearly depicting soft tissues, MRI can reveal dural inflammation, sigmoid sinus thrombosis, labyrinthitis, and extradural and intracranial abscesses.

An audiogram should be performed. Conductive hearing loss is expected, but mixed hearing loss may indicate more extensive disease and should alert the treating physician of impending complications.

Approach Considerations

Patients with chronic suppurative otitis media (CSOM) respond more frequently to topical therapy than to systemic therapy. Successful topical therapy consists of 3 important components: selection of an appropriate antibiotic drop, regular aggressive aural toilet, and control of granulation tissue.

Inpatient care is rarely necessary for the patient with CSOM. In patients for whom the otolaryngologist chooses systemic parenteral antibiotics, inpatient hospitalization may be required. Otherwise, excluding complications, this disease can be treated effectively in the outpatient setting. Patients who present with suspected intracranial complications to hospitals that function without CT scanning capabilities or neurosurgical care should be transferred as soon as possible to an institution with such capabilities. Antibiotic therapy should be started prior to transfer.

Antibiotic Drops

The antibiotic should have an appropriate spectrum of activity that includes gram-negative organisms (especially pseudomonads) and gram-positive organisms (especially *S aureus*) . Aminoglycosides and the fluoroquinolones are antibiotics that meet this initial criterion. Topical antibiotic drops containing aminoglycosides have been marketed and used for more than 20 years.

Neomycin and polymyxin B

Most drops marketed specifically for otologic use contain neomycin combined with a cationic detergent (polymyxin B). Neomycin has remained fairly effective over the last 2 decades for gram-positive organisms, but it has lost almost all of its effectiveness for combating gram-negative organisms. Dohar's studies indicate that fewer than 20% of gram-negative organisms remain sensitive to neomycin; however, polymyxin B has remained effective for gram-negative bacteria. The combination consequently remains reasonably effective from an antimicrobial point of view.[15, 16, 17]

Gentamicin, dexamethasone, and tobramycin

Gentamicin- and tobramycin-containing ophthalmic drops have been widely used off-label for the treatment of otologic infections. A fixed ratio combination of tobramycin and dexamethasone (TobraDex) has been especially popular in the United States, while gentamicin-containing drops have been more popular in Canada and Europe.

Aminoglycosides

All aminoglycosides have significant potential toxicity. Some are more vestibular-toxic than cochlear-toxic and, therefore, are more likely to produce vestibular dysfunction than hearing loss. For other aminoglycosides, the opposite is true. Studies designed to detect hearing loss from the use of ototopical aminoglycosides demonstrate that the incidence of aminoglycoside-related hearing loss is, at worst, low. Recent information, however, suggests that the potential for vestibular toxicity may be much higher, especially if preparations containing gentamicin are used.

pH level of drops

Otic drops differ in pH. Drops designed for otic use are often buffered slightly to an acidic pH because the normal environment of the external auditory canal is acidic. These drops can be extremely painful if they penetrate into the middle ear, especially if the middle ear mucosa is normal. While low pH is an advantage when treating infections in the external auditory canal, the advantage is lost in the middle ear because the normal pH of the middle ear is neutral. Within the middle ear space, the potential for low pH solutions to cause pain or to irritate mucosa can render them disadvantageous.

Most ototopic antibiotic steroid combinations are at least somewhat acidic because it is almost impossible to keep either quinolones or aminoglycosides in solution at a neutral or basic pH. The acidity of polymyxin, neomycin, and hydrocortisone varies from as low as 3.5 to 4.5. Ciprofloxacin and hydrocortisone combinations have a pH of 4.5-5.0, as do tobramycin and dexamethasone combinations.

Viscosity of drops

Ototopical preparations vary in viscosity. Preparations containing an antibiotic are usually solutions and have relatively low viscosities approaching that of water (1.0 cP). Preparations containing a steroid are often of considerably higher viscosity, ranging from 2-8 cP. Polyviscous solutions may effectively coat and remain in contact with tissues for longer periods, although they are less likely to move through or around small spaces (eg, tympanostomy tubes, granulation tissue, polyps) than are preparations of lower viscosity.

Bacterial resistance

Some controversy surrounds the development of bacterial resistance due to ototopical treatment. Recent studies have not identified any increase in bacterial resistance through ototopical antibiotic administration. Specifically, the concentration in quinolone ototopical drops overwhelms the most resistant pseudomonal and staphylococcal strains. Failure of topical antibiotic delivery to the pathogenic organisms should be considered a cause of persistent infections.

Steroid-containing drops

Roland et al demonstrated that the anti-inflammatory effect of steroids is an important advantage when significant amounts of granulation tissue are present.[18] Ototopicals with steroids were superior to steroid-free ototopicals in reducing granulation tissue at days 11 and 18 of treatment. The steroid-containing drops should be considered in CSOM with granulation tissue.

Aural Toilet

Aural toilet is a critical process in the treatment of CSOM. The external auditory canal and tissues lateral to the infected middle ear are often covered with mucoid exudate or desquamated epithelium. Topically applied preparations cannot penetrate affected tissues until these interposing materials are removed.

Traditionally, in otolaryngology, aural toilet has been achieved using the microscope and microinstruments to mechanically remove mucoid exudates, desquamated epithelium, and other interposing materials. For best results, aural toilet should be performed 2-3 times per day just before the administration of topical antimicrobial agents.

Aural irrigation is an effective alternative that is often less burdensome for patients and physicians. A solution of 50% peroxide and 50% sterile water is generally painless and effective. Thirty to 40 mL of this solution can be irrigated through the external auditory canal, using a small syringe or bulb-type aspirator. The irrigant solution can be allowed to drain out for 5-10 minutes prior to instilling the ototopical antimicrobial.

Granulation Tissue Control

Granulation tissue often fills the middle ear and medial portions of the external auditory canal. Granulation tissue can prevent topically applied antimicrobial agents from penetrating the site of infection. The use of topical antimicrobial drops is the first step in controlling granulation. These drops help reduce granulation tissue by eliminating infection and by removing the inciting irritating inflammation. As previously discussed, most physicians believe that topical steroids are important and hasten the resolution of middle ear granulation, thus improving penetration of topically delivered antimicrobial agents.

Cautery is often used to reduce the amount of granulation tissue and to control its formation. Microbipolar cautery can be used in the office, but chemical cautery is used more commonly. Silver nitrate can conveniently be applied in the form of silver nitrate sticks. Caution must be exercised, as the depth of the chemical burn induced by the application of chemical agents, including silver nitrate, is uncontrolled. Excision of granulation tissue can be accomplished in the office with the use of a microscope and microinstruments. Silver nitrate is often used to control bleeding and to enhance the efficacy of granulation tissue removal.

An important part (perhaps the most important part) of tympanomastoidectomy for the treatment of CSOM consists of removing and controlling granulation tissue within the middle ear, mastoid, and mastoid antrum.

Failure of Topical Treatment

Failures of topical antimicrobial therapy are almost always failures of delivery. Specifically, failure of delivery describes the inability of an appropriate topical antibiotic to reach the specific site of infection within the middle ear. Various elements may obstruct the delivery of the medication, including infectious debris, granulation tissue, cholesteatoma, neoplasia, cerumen, and others. When topical therapy fails, the patient needs a thorough evaluation for anatomic obstruction, including microscopic examination and radiologic studies as needed. Additionally, a clear understanding of the very high concentration of the antibiotic within topical preparations must be kept in mind.

The minimal inhibitory concentrations (MICs) for *S aureus, S pneumoniae,* and the other organisms that commonly cause CSOM are generally 1-2 mcg/mL. Generally, intravenously administered aminoglycosides and any pseudomonal cephalosporins can slightly exceed these levels.

Orally administered fluoroquinolones also slightly exceed the MICs of most of the relevant organisms (oral administration achieves blood levels as high as those achieved with parenteral administration). Concentrations of medicines in the middle ear fluid rarely exceed 4-6 mcg/mL. In contrast, a 0.3% topical antibiotic solution contains 3000 mcg/mL, a concentration 100-1000 times that which can be achieved using systemic administration. Moreover, this concentration greatly exceeds the MIC for any relevant organism.

Because of the high concentrations of antimicrobial agents, topical therapy is more likely to be effective than systemic therapy. Studies comparing systemic administration to topical administration show that topical cure rates nearly double systemic rates. Topical therapy does not fail because the organism is resistant; even supposedly resistant organisms succumb to these very high concentrations. For instance, even an extraordinarily resistant strain of *S aureus* with an MIC of 256 mcg/mL cannot survive in an environment in which the concentration of antibiotic is 3000 mcg/mL. The emergence of resistance to topical therapy is extremely uncommon. The rapid kill rates and high concentrations of topically administrated drops do not permit even mutant strains with higher MICs to survive.

Consequently, when topical therapy for CSOM fails, it is almost never because of antimicrobial resistance; therefore, culture and sensitivity are of little benefit as long as therapy is topical. Sensitivity reports from the clinical laboratory are irrelevant. Sensitivity testing in the clinical laboratory is designed for the tissue concentrations achievable by systemic administration. Consequently, a pseudomonad with an MIC of 48 mcg/mL is likely to be reported as resistant by the clinical laboratory.

Systemic Therapy

Systemic therapy should be reserved for cases of CSOM that fail to respond to topical therapy. Topical therapy presumably fails because the antibiotics cannot reach infected tissues. Systemic therapy is expected to succeed in the penetration of the tissues.

If a focus of infection in the mastoid cannot be reached by topical drops, there is a reasonable chance that systemically administered antibiotics can penetrate these areas in sufficient concentrations to control or eliminate infection, although the concentrations are lower. Ototopical therapy is generally continued once systemic therapy is begun. Indeed, since systemic therapy frequently involves hospitalization for the intravenous administration of drugs, aural toilet can frequently be intensified. The ability to perform reliable aural toilet may be as important as the systemic antimicrobial therapy in eliminating the disease for some patients.

Prior to instituting systemic therapy, a culture should be obtained for sensitivity. Sensitivity testing is important when systemic therapy is being considered. The antibiotics should be selected on the basis of the resulting sensitivity profile. The narrowest spectrum antibiotic with the fewest adverse effects and complications should be used.

All of the aminoglycosides are potentially useful, although tobramycin has been shown to be more effective against pseudomonads than gentamicin. Dohar et al have shown that piperacillin is probably the most effective antibiotic. Ceftazidime remains a useful choice for many patients. Systemic therapy should be continued for 3-4 weeks. Most individuals experience the cessation of otorrhea in shorter periods. Antimicrobial therapy should probably be continued at least 3-4 days after the cessation of otorrhea.

Ciprofloxacin remains the most effective of the quinolones for pseudomonads. Some of the late-generation "respiratory quinolones" appear to be more efficacious for *S aureus.* Fluoroquinolones are not approved for use in children because animal studies using juvenile

subjects have shown that they elicit joint injury. Nevertheless, a large database of children with cystic fibrosis who have been treated with systemic fluoroquinolones at relatively high doses for prolonged periods demonstrates that the risk of joint injury appears to be absent or very low. No cases of permanent joint injury have been reported.

A few children of the many thousands treated have developed pain that remitted with the cessation of therapy. Given the real potential toxicity of intravenously administered antibiotics (especially the aminoglycosides), serious consideration should be given to the use of oral fluoroquinolones when treating children with CSOM unresponsive to topical therapy. Potentially, the fluoroquinolones are the most useful class of oral antibiotics for treating CSOM. Oral therapy achieves serum concentrations as high as parenteral therapy, obviating the need for intravenous delivery.

The risk of injury, adverse reaction, or significant adverse effects appears to be lower overall with systemic quinolones than with many of the other antibiotics normally used to treat gram-negative infections. Most parents are amenable to the off-label use of oral fluoroquinolones if they understand the relative risks and potential benefits offered by this class of drugs in comparison to the variable alternatives.

Surgery should be considered if CSOM fails to respond to a combination of topical and systemic therapy. A tympanomastoidectomy can eliminate infection and stop otorrhea in 80% of patients.

Surgery for Chronic Ear Disease

Patients with CSOM that is unresponsive to topical and/or systemic medical therapy with appropriate aural toilet and control of granulation tissue require surgery. The modern surgery for chronic otitis media was popularized in the 1950s. Prior to this, ear surgery was primarily successful at draining active infection, and there was less concern about long-term functional outcomes. Current goals for surgery for chronic ear disease include a dry, safe ear and the preservation of the normal structure and functioning to the greatest extent possible.

In patients with CSOM without cholesteatoma, surgery is considered if the perforation is persistent and long-standing and causes clinical symptoms, such as recurrent ear discharge and hearing loss. The age, general physical condition, fitness for general anesthesia, and coexisting diseases of the patient also play an important role in considering surgery.

General indications for surgery are as follows:

- Perforation that persists beyond 6 weeks
- Otorrhea that persists for longer than 6 weeks despite antibiotic use
- Cholesteatoma formation
- Radiographic evidence of chronic mastoiditis, such as coalescent mastoiditis
- Conductive hearing loss

For patients with early or mild CSOM cholesteatoma, aural toilet and repeated suction clearance of the ear with watchful expectancy may be performed; for patients with advanced disease, exploration of the mastoid and tympanoplasty is recommended.

The principal aim of surgery for CSOM is first to clear out the disease and only then, if possible, to reconstruct the patient's hearing. Hearing reconstruction is often completed in a planned second-stage operation in patients with cholesteatoma. Staging the ear allows for the development of a healthy, air-containing middle ear space. Further inspection of the middle ear and mastoid cavity can confirm that the cholesteatoma has been eradicated. Silastic or other material is often placed in the middle ear and mastoid cavity to prevent postoperative scarring. This material is then removed during the second-stage procedure.

Contraindications (relative and absolute) to surgery for tubotympanic disease are as follows:

- Surgery on the only hearing ear
- Poor general physical condition, old age, or debility that makes general anesthesia risky
- Patients unwilling to undergo surgery
- Surgery on patients with unilateral vestibular ablation

Contraindications to surgery for atticoantral disease are as follows:

- Early or mild cholesteatoma amenable to aural toilet
- Patients who are severely ill and those with complications secondary to cholesteatoma, such as a brain abscess (drainage of the brain abscess and intravenous administration of antibiotics should be considered first)

Myringoplasty and Tympanoplasty

Myringoplasty is the operation specifically designed to close tympanic membrane defects. The approach to the ear can be transcanal, endaural, or retroauricular. The transcanal approach requires less surgical exposure and leads to faster healing. The downside is the potential limitation of exposure. The endaural approach can improve exposure in ears with a lateral soft tissue or cartilage overgrowth, but again, it tends to limit the surgical view. The retroauricular approach allows for maximal exposure but requires an external skin incision.

Two main surgical techniques of tympanoplasty are commonly used: the underlay and the overlay. The underlay technique involves placing the graft material underneath (or medial to) the eardrum. The underlay technique requires dissection and elevation of a tympanomeatal flap. The margins of the perforation are freshened by removing the epithelium from the edges of the hole. The graft material is tucked underneath the eardrum and is sometimes supported with Gelfoam. Then, the reconstituted eardrum is flipped back to its normal resting position, and the ear canal is filled with packing material. The lateral graft technique requires removal of the ear canal and tympanic membrane epithelium, as well as a canaloplasty. This technique is particularly well suited to revision tympanoplasty or ears with narrow canals. It is somewhat more technically demanding but has a very high success rate, particularly in scarred tympanic membranes.

The overlay technique involves grafting lateral to the eardrum. Various graft materials may be used. The most common materials are temporalis fascia, tragal perichondrium, and vein graft. An additional technique is the "stuff through." This may be useful for small perforations in otherwise healthy ears. This procedure essentially freshens the edges of the perforation and then fills it with a plug of tissue, usually fat.

Chronic otitis media with cholesteatoma

A range of surgical procedures are available for the management of CSOM with cholesteatoma, and the choice of procedure depends on the extent and the severity of the disease and the hearing of the individual. The ultimate aim of the procedure is to provide the patient with a safe, dry ear. Hearing improvement is a secondary consideration and, if attempted, is usually performed during a second-stage surgery. Hearing reconstruction should not be performed at the cost of or by compromising the clearance of the disease in the patient.

Tympanoplasty is performed to eradicate disease from the middle ear and to reconstruct the hearing mechanism, with or without grafting of the tympanic membrane. The 5 different types of tympanoplasties have been defined. These are primarily of historical interest. The following definitions describing the 5 types of tympanoplasties are used for middle ear surgery and mastoid surgery:

- Type 1 is simple closure of the tympanic membrane perforation without reconstructing the ossicular chain
- Type 2 is any kind of ossicular reconstruction involving the malleus, the incus, or both; the stapes head is intact
- Type 3 involves putting the tympanic membrane graft over the head of the stapes
- Type 4 occurs when the stapes head is absent but the footplate is present; the stapes footplate is exteriorized to the mastoid cavity, and the graft is placed over the rest of the middle ear cavity, including the round window; hence, the phase difference is maintained.
- Type 5 is also called the fenestration operation; it involves making a fenestra in the lateral semicircular canal and then putting a graft over it; this is not often performed today

Tympanoplasty is broken down into 2 primary types: lateral grafting and medial grafting. In the lateral graft technique, the graft material is laid laterally to the annulus after the remnant of squamous tissue is denuded. In medial grafting, the annulus is raised and the graft slipped medially.

Mastoidectomy

Cortical mastoidectomy is also known as the Schwartze operation. It consists of the removal of the outer wall of the mastoid cortex and the exteriorization of all the mastoid air cells. This may be performed immediately in coalescent mastoiditis, in which case a drain may be left postoperatively.

Canal wall-up mastoidectomy refers to the removal of mastoid air cells while retaining the posterior canal wall. Using this approach with a facial recess (drilling the bone of the posterior mesotympanum or facial recess between the incus, the chorda tympani nerve, and the facial nerve), the middle ear structures can be accessed for careful dissection of the cholesteatoma. This approach leaves the normal ear canal anatomy intact, thereby preventing the potential problems seen with a mastoid cavity. This is also the common approach for cochlear implantation.

Modified radical mastoidectomy differs from radical mastoidectomy in that the ossicles and the tympanic membrane remnants are preserved for possible hearing reconstruction at a later stage. Radical mastoidectomy involves eradication of all disease from the middle ear and the mastoid and exteriorization of these structures into a single cavity. It also includes removing the entire tympanic membrane and the ossicles (except the stapes footplate) and closing the eustachian tube opening. Currently, this procedure is performed only in very unusual situations.

Postoperative Details

With mastoidectomy/tympanoplasty, ear packing can be removed after 3 weeks (earlier if infected). Often, ear drops are prescribed to be started 3 weeks after surgery. The packing is then removed at 5-6 weeks postoperatively, particularly in the lateral graft tympanoplasty, which requires additional healing time.

The patient receives follow-up care regularly until the canal or cavity is well epithelialized. At each follow-up visit, any signs of recurrent cholesteatoma are noted. If any hearing reconstruction/ossiculoplasty has been performed, an audiogram is indicated at 3 months. Once the canal is healed, water precautions can be stopped. If a canal wall-down mastoidectomy is performed, water entrance may still be discouraged. The mastoid cavity can be irrigated with a solution of alcohol and vinegar as needed. Routine cleaning of the mastoid cavity may also be indicated if canal wall-down procedures are performed.

Surgical Complications

Complications of tympanoplasty may include the following:

- Graft failure rates range from 10-20% depending on the technique used and the experience of the surgeon
- Infection is a potential complication with any surgical procedure but is rarely seen in tympanoplasty; some surgeons recommend perioperative antibiotics, but there are little data to support their use
- Hematomas can develop with postauricular approaches
- Taste disturbance occurs secondary to damage to the chorda tympani nerve; it tends to be self-limiting but can be disturbing to patients
- Ear numbness can result if the postauricular incision severs peripheral sensory nerves, leading to some numbness of the pinna and lobule; this is particularly disturbing to patients with pierced ears who depend on the feeling in the lobule to place earrings

- Conductive hearing loss can occur secondary to ossicular disruption or sclerosis; scarring of the neo-tympanic membrane that leads to lateralization can also cause a conductive loss
- Sensorineural hearing loss is rarely seen but is considered a serious complication
- Vertigos can occur during any middle ear procedure and is usually self-limiting; however, if it is severe or persistent, further workup may be in order
- Facial paralysis is rare but is sometimes seen secondary to local anesthetic infiltration, although anesthetic-related paralysis usually resolves soon after surgery

Complications of mastoidectomy or tympanomastoidectomy include those listed above and the following:

- CSF leak may occur if the dura is encountered and violated during the dissection; this can often be repaired if recognized during surgery
- Intracranial complications can include brain abscess, meningitis, or physical damage to the brain itself

Surgical Prognosis

Tympanoplasty provides most patients with a healed, dry ear. In patients with cholesteatoma, staged procedures are often necessary, and residual cholesteatoma is evaluated during ossicular reconstruction. Keep in mind that chronic ear disease is just that—chronic. These patients often suffer recurrence over time, and regular lifetime surveillance is normal.

The general and most desirable outcome for a patient who has undergone a tympanomastoidectomy is a dry, nondischarging, healthy ear. Long-term follow-up care of these patients is essential to detect the recurrence of cholesteatoma at its earliest onset. In such cases, a second procedure may be necessary. The likelihood of hearing preservation depends on the extent of the disease and the involvement of the ossicles, and it varies widely.

The following measures help prevent recurrence and allow for early intervention in patients with recurrent infections:

- Patients should be advised to keep their ears dry to prevent future complications, even after medical treatment results in a safe and dry ear; swimming is not contraindicated if patients dry their ears afterward
- Tympanoplasty, a surgery that seals the perforation, prevents the translocation of bacteria from the external ear canal into the middle ear; the uninflamed, protected middle ear mucosa deters future development of CSOM
- Early symptoms of aural fullness, otalgia with or without fever, and headache warrant evaluation by an otolaryngologist in patients with a recent history of CSOM

References

1. Matsuda Y, Kurita T, Ueda Y, Ito S, Nakashima T. Effect of tympanic membrane perforation on middle-ear sound transmission. *J Laryngol Otol*. May 2009;123 Suppl 31:81-9. [Medline].
2. Wright D, Safranek S. Treatment of otitis media with perforated tympanic membrane. *Am Fam Physician*. Apr 15 2009;79(8):650, 654. [Medline].
3. Vikram BK, Khaja N, Udayashankar SG, Venkatesha BK, Manjunath D. Clinico-epidemiological study of complicated and uncomplicated chronic suppurative otitis media. *J Laryngol Otol*. May 2008;122(5):442-6. [Medline].
4. McKenzie W, Brothwell D. Disease in the Ear. *Disease in Antiquity*. 1967;464-73.
5. Gregg JB, Steele JP, Holzhueter A. Roentgenographic evaluation of temporal bones from South Dakota Indian burials. *American Journal of Physical Anthropology*. 1965;23:51-62.
6. Rathbun TA, Mallin R. Middle ear disease in a prehistoric Iranian population. *Bull N Y Acad Med*. Dec 1977;53(10):901-5. [Medline]. [Full Text].
7. Meyerhoff WL, Kim CS, Paparella MM. Pathology of chronic otitis media. *Ann Otol Rhinol Laryngol*. Nov-Dec 1978;87(6 Pt 1):749-60. [Medline].
8. Kenna MA. Microbiology of Chronic Suppurative Otitis Media. *Ann Otol Rhinol Laryngol*. 1988;97(suppl 131):9-10.
9. Kenna MA. Etiology and Pathogenesis of Chronic Suppurative Otitis Media. *Ann Otol Rhinol Laryngol*. 1988;97(Suppl 131):16-17.
10. Matsuda Y, Kurita T, Ueda Y, Ito S, Nakashima T. Effect of tympanic membrane perforation on middle-ear sound transmission. *J Laryngol Otol*. May 2009;123 Suppl 31:81-9. [Medline].
11. Wright D, Safranek S. Treatment of otitis media with perforated tympanic membrane. *Am Fam Physician*. Apr 15 2009;79(8):650, 654. [Medline].
12. van der Veen EL, Schilder AG, van Heerbeek N, et al. Predictors of chronic suppurative otitis media in children. *Arch Otolaryngol Head Neck Surg*. Oct 2006;132(10):1115-8. [Medline].
13. Vikram BK, Khaja N, Udayashankar SG, Venkatesha BK, Manjunath D. Clinico-epidemiological study of complicated and uncomplicated chronic suppurative otitis media. *J Laryngol Otol*. May 2008;122(5):442-6. [Medline].
14. Smith JA, Danner CJ. Complications of chronic otitis media and cholesteatoma. *Otolaryngol Clin North Am*. Dec 2006;39(6):1237-55. [Medline].
15. Dohar JE. Old and New Ototopical Agents for the Acute and Chronic Draining Ear. Seminars in Otitis Media Management. *1*. 1998;1-14.
16. Dohar JE, Alper CM, Rose EA, Doyle WJ, Casselbrant ML, Kenna MA. Treatment of chronic suppurative otitis media with topical ciprofloxacin. *Ann Otol Rhinol Laryngol*. Oct 1998;107(10 Pt 1):865-71. [Medline].
17. Dohar JE, Kenna MA, Wadowsky RM. Therapeutic implications in the treatment of aural Pseudomonas infections based on in vitro susceptibility patterns. *Arch Otolaryngol Head Neck Surg*. Sep 1995;121(9):1022-5. [Medline].