CHAPTER 62

Otolaryngology*

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OTITIS MEDIA

Principles

Otitis media is broadly defined as inflammation of the inner ear and is a continuum of disease. Acute otitis media is defined as the signs and symptoms of an acute infection, with evidence of effusion; this has also been called acute suppurative or purulent otitis media. Otitis media with effusion (OME) includes effusion without signs or symptoms of an acute infection; additional descriptive terms include serous, mucoid, nonsuppurative, and secretory otitis media. Chronic otitis media or chronic suppurative otitis media refers to chronic discharge from the ear through perforation of an intact membrane. Recurrent otitis media is defined by three or more episodes over 6 months or four episodes in 1 year.

Acute otitis media (AOM) is one of the most common diseases affecting preschool children in the United States and represents the most common indication for antibiotic usage and pediatric outpatient visits.[1] More than 80% of children will have at least one episode of AOM during their lifetime and, by 3 years of age, up to 40% will have had at least three episodes. In 2011, there were 6.21 million patient visits with a diagnosis of otitis media.[2] The financial repercussions are enormous, with one estimate that it adds $2.88 billion to annual health care expenses.[1]

Male gender, daycare attendance, parental smoking, pacifier use, family history of middle ear disease, premature birth, and lower socioeconomic status have been implicated as risk factors. Children with anatomic abnormalities, such as cleft palate and Down syndrome, have a higher rate of OM, probably because of eustachian tube abnormalities. Some immunocompromised patients, including patients with human immunodeficiency virus (HIV) infection, may have recurrent OM as an initial symptom of their underlying disease. OM and upper respiratory infections occur primarily in the winter. Breast-feeding seems to be protective. Immunizations for pneumococcus and influenza provide some protection but the decrease in overall episodes of otitis media is multifactorial. These factors include improved diagnosis, public education campaigns, and decreasing exposure to secondhand smoke. AOM is much less common in adults and is treated with the same antibiotics as for younger populations. OME is also less common in adults and is frequently associated with sinus disease, smoking-induced nasopharyngeal lymphoid hyperplasia, adult-onset adenoidal hypertrophy, and head and neck tumors such as nasopharyngeal carcinomas.[3]

Anatomy and Pathophysiology

Eustachian tube dysfunction is the central theme of most theories of AOM pathogenesis. The eustachian tube, between the middle ear cavity and nasopharynx, ventilates the middle ear to equilibrate pressure, allows for middle ear drainage, and provides protection from nasopharyngeal secretions. In young children, the eustachian tube is short and horizontal. As individuals age, the eustachian tube widens, doubles in length, becomes more vertically oriented, and stiffens, which may explain the decreased incidence of AOM in adults. Normally, the tube is collapsed, but it opens during yawning, chewing, and swallowing.

The eustachian tube may become mechanically or functionally obstructed, decreasing middle ear ventilation. Examples of mechanical obstruction include inflammation from an upper respiratory infection, hypertrophied adenoids, and a cleft palate. Functional obstruction from persistent tubal collapse occurs primarily in young children, who have less fibrocartilage support of the medial eustachian tube than older children or adults. There is general consensus that AOM occurs as a consequence of an upper respiratory infection resulting in eustachian tube dysfunction and subsequent negative middle ear cavity pressure, causing a transudate of fluid that combines with the reflux of nasopharyngeal secretions and bacteria. As such, there is a proliferation of bacteria and viruses.

The advent of reverse transcriptase polymerase chain reaction technology and other techniques for viral identification has led to improvements in diagnosis, and thus the number of viral agents identified in the middle ear has increased. In pediatric patients, middle ear cultures have been positive for viruses 48% to 70% of the time, with viral and bacterial coinfection occurring between 45% and 66% of the time. Respiratory syncytial virus is the most common virus, but parainfluenza virus, influenza virus, rhinovirus, and adenovirus have also been found in the middle ear aspirates of children. Viruses contribute to a poor treatment outcome by increasing middle ear inflammation, decreasing neutrophil function, and decreasing antibiotic penetration into the middle ear. The most common causes of bacterial infection in children are *Streptococcus pneumoniae, Haemophilus influenzae* (primarily nontypeable), and *Moraxella (Branhamella) catarrhalis. Streptococcus pyogenes, Staphylococcus aureus,* and gram-negative bacteria are much less common. The widespread use of the pneumococcal seven-valent conjugate vaccine (PCV-7) and subsequent pneumococcal 13-valent conjugate vaccine (PCV-13) have changed the frequency of these common organisms, with *H. influenzae* increasing in frequency, particularly in persistent AOM and treatment failures.

In young children, it was previously believed that gram-negative organisms and *S. aureus* were the causative organisms. Although these bacteria may be the causes in intubated patients or patients in the neonatal intensive care unit, healthy newborns tend to be infected by the same pathogens as healthy older children. Bullous myringitis produces bullae on the tympanic membrane (TM) in up to 5% of cases of OM in children younger than 2 years. Although it was previously thought to be caused by *Mycoplasma pneumoniae, M. pneumoniae* is uncommon; a culture of middle ear aspirates in this condition generally grow the usual organisms that cause AOM in all age groups. Bullous myringitis is therefore treated with the same antibiotics.

More than 70% of children with purulent conjunctivitis may have OM, a symptom complex described as the otitis-conjunctivitis syndrome, which is predominantly caused by *H. influenza.* Other

*The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

less likely organisms that can cause AOM include *Mycobacterium tuberculosis* (primarily in children) and *Chlamydia trachomatis* (most commonly seen in children <6 months).

Up to 10% of the general pediatric population may be at risk for developing four or more episodes of OM in the first year of life; these children are generally said to be otitis-prone. They may have subtle immunologic abnormalities or a greater baseline colonization of viruses and bacteria than the general population.

Clinical Features

Ear pain, unilateral or bilateral, is the most important symptom for making the diagnosis of AOM. Patients with AOM may present with a multitude of symptoms, such as cough, upper respiratory tract symptoms, poor appetite, diarrhea, vomiting, fever, and pulling at the ears, all of which are nonspecific. In fact, there is no constellation of symptoms or scoring systems that predict AOM, and examination of the tympanic membrane is essential to make the diagnosis.[4]

During the physical examination, the auricle and external canal are inspected for signs of erythema, discharge, and/or tenderness. If the canal is occluded with cerumen, curettage may clear the canal to improve visibility. In the cooperative child, the placement of 3% hydrogen peroxide or emulsifying drops, followed by gentle irrigation, may cleanse the canal if curettage is unsuccessful. The normal TM may be red, pink, yellow, or a normal pearly gray or translucent. The presence of erythema in itself does not indicate infection because crying or fever may cause hyperemia; however, a TM that is distinctly red (defined as hemorrhagic, strongly or moderately red) suggests AOM. Landmarks on the TM that should be visible include the pars flaccida, malleolus, and light reflex below the umbo. The presence of opacification, bubbles, air fluid levels, or retraction of the TM are suggestive of middle ear effusion. AOM is a visual diagnosis. A bulging tympanic membrane in a patient with signs and symptoms of acute infection is diagnostic of acute infection.[5] Demonstration of tympanic membrane immobility by pneumatic otoscopy is useful in distinguishing the presence of effusion in cases in which the provider is uncertain, but this is difficult, even with experienced providers.[6] A comparison examination of the other ear may help in confirming suspected infection.

In neonates, the TM is in a highly oblique position and normally appears thickened and opaque in the first few weeks of life. With tympanostomy tubes, even in the absence of infection, the TM may have decreased mobility, altered landmarks, opacity, or dullness. If the tube is patent, erythema and discharge indicate infection. If the tube is not patent, typical erythema, bulging of the TM, and immobility indicate AOM.

Before the use of antibiotics, there was a 20% incidence of complications from AOM, with mastoiditis and otic meningitis being relatively common. Complications are intratemporal or intracranial, occurring in adults and children. The development of either complication of OM occurs by one of three mechanisms: (1) direct extension of infection through bone weakened by osteomyelitis or cholesteatoma; (2) retrograde spread of infection by thrombophlebitis; or (3) extension of infection along preformed pathways, such as the round or oval windows or through dehiscences that are the result of congenital malformations. The use of antibiotics has led to a reduction of all complications to less than 1%.[7]

Usually, TM perforation occurs at the pars tensa and resolves spontaneously. It may persist for a longer period, resulting in a chronic perforation, chronic OM, or both. Chronic otitis media refers to inflammation of the middle ear that persists for 6 weeks or longer, accompanied by discharge through perforation of an intact membrane. Cholesteatoma is an accumulation of keratin-producing squamous epithelium in the middle ear and may result in erosion of bone within the middle ear cavity. It is seen most often in OME, in which retraction of the TM is a common problem, and its presence may alter the course of some treatment modalities.

The facial nerve courses through the middle ear, and facial paralysis is a known complication in OM. The exact mechanism is unknown, but the paralysis may be a result of infection, surrounding osteitis, facial nerve swelling, demyelination of the facial nerve from bacterial toxins, or facial nerve ischemia. Bony destruction as a result of mastoiditis can result in a defect over the semicircular canals, resulting in a labyrinth fistula. The patient may present with vertigo and some degree of hearing loss.

Meningitis is the most common intracranial complication of AOM,[8] resulting from hematogenous spread and direct invasion. Brain abscesses are usually caused by chronic otitis and are the second most common intracranial complication. Extradural abscesses, subdural empyema, and lateral venous sinus thrombosis have all been identified as complications of OM.

Differential Diagnoses

AOM is not common in children younger than 6 months as a result of the protection of maternal antibodies acquired transplacentally. Other sources of infection should be investigated in a febrile, ill-appearing infant (see Chapter 166). In addition to OM, other causes of otalgia include OME, trauma, foreign bodies, and complications of OM, such as mastoiditis and referred pain from the teeth, sinuses, throat, or temporomandibular joint.

Diagnostic Testing

Pneumatic otoscopy to confirm bulging and immobility of the TM is the primary diagnostic modality for AOM.

Management

Physicians in the Netherlands in the early 1990s had suggested that OM is a self-limited disease and recommended observation as an initial treatment option, followed by the use of antibiotics if the patient's condition did not improve within 72 hours. The American Academy of Pediatrics (AAP) and the American Academy of Family Physicians developed guidelines in 2004 for the diagnosis and management of AOM and updated them in 2013.[9] The guidelines cover diagnosis, pain management, observation, and antibiotic recommendations and apply to healthy children and not those with anatomic conditions that put them at risk for infections. Given the multidisciplinary approach, we recommend adherence to these guidelines.

The 2013 guidelines recommend these diagnostic criteria[10]:

1. Moderate to severe bulging of the TM or new onset of otorrhea not due to otitis externa *or*
2. Children who present with mild bulging of the TM and recent (<48 hours) ear pain (holding, tugging, rubbing of the ear in a nonverbal child) or intense erythema of the TM.

AOM can cause substantial pain, which should be appropriately addressed. Acetaminophen and ibuprofen are safe, over-the-counter, first-line analgesics. The use of opioid analgesia has not been well studied. Benzocaine-antipyrine, a local anesthetic, may be helpful in some patients with an intact TM. It has been shown to be more effective than placebo and is an additional option that we recommend for pain relief.

Children younger than 2 years, those with bilateral OM, or those with otorrhea gain the greatest benefit from antibiotic treatment but, because more than 80% of cases of AOM resolve spontaneously, the use of observation versus antibiotics has been advocated. This approach of watchful waiting for 48 hours has resulted in lower rates of antibiotic-resistant bacteria. The delay

TABLE 62.1

Recommendations for Initial Management for Uncomplicated Acute Otitis Media (AOM)

AGE	OTORRHEA WITH AOM[a]	UNILATERAL OR BILATERAL AOM[a] WITH SEVERE SYMPTOMS[b]	BILATERAL AOM[a] WITHOUT OTORRHEA	UNILATERAL AOM[a] WITHOUT OTORRHEA
6 mo–2 yr	Antibiotic therapy	Antibiotic therapy	Antibiotic therapy	Antibiotic therapy or additional observation
≥2 yr	Antibiotic therapy	Antibiotic therapy	Antibiotic therapy or additional observation	Antibiotic therapy or additional observation[c]

[a]Applies only to children with well-documented AOM with high certainty of diagnosis.
[b]A toxic-appearing child, persistent otalgia >48 hr, temperature >39°C (102.2°F) in the past 48 hr, or if there is uncertain access to follow-up after the visit.
[c]This plan is initial management and provides an opportunity for shared decision making with the child's family for those categories appropriate for additional observation, if offered; a mechanism must be in place to ensure follow-up and begin antibiotics if the child worsens or fails to improve within 48–72 hr of AOM onset.
Adapted from Lieberthal AS, Carroll AE, Chonmaitree T, et al: Clinical Practice Guideline: the diagnosis and management of acute otitis media. Pediatrics 131:e964–e999, 2013.

does not worsen recovery but may be associated with transient worsening of a child's condition.[10] The observation option has been restricted to healthy children older than 6 months. In children between 6 months to 2 years of age, treatment recommendations are based on the certainty of the diagnosis and severity of illness. In patients with unilateral AOM without otorrhea, observation is an option if the diagnosis is uncertain. In children older than 2 years, treatment is necessary only for patients with severe illness, defined as severe otalgia or temperature higher than 39°C (102°F) or patients with otorrhea. Children older than 2 years can be treated or clinically observed. Table 62.1 summarizes the AAP recommendations.

Observation recommendations are also based on the reliability of the caregivers and ability for close follow-up. Providers should involve the parents in the discussion, with shared decision making. If there is concern about the ability to get follow-up, give parents a safety net prescription to be filled if the patient's condition does not improve within 48 hours. Several studies in the emergency department (ED) have shown success with use of this approach. An analysis from the National Ambulatory Medical Care Survey has revealed that management without antibiotics has not increased since the guidelines were published, although children who did not receive antibiotics were more likely to have mild infections.[11] There are no data on the use of observation in adult patients, so they should be treated with amoxicillin, 500 mg tid for 10 days.

The decision to treat is balanced against the medication's adverse effects, which may include allergic reactions, gastric upset, accelerated bacterial resistance, and unfavorable changes in the bacterial flora. Several large systematic reviews have revealed that antibiotics are modestly more effective than no treatment, but 4% to 10% of children experience adverse effects from the treatment itself.[12] Two randomized controlled trials comparing amoxicillin-clavulanate versus placebo in a total of 610 patients have reported modestly improved time to resolution of symptoms and otoscopic findings but with more side effects, with diarrhea being the most common.[13,14] Although some authorities believe that these studies settled the treatment controversy, the studies were far from conclusive. Observation in children from 6 months to 2 year of age with unilateral AOM without otorrhea, or children older than 2 years with a nondraining ear or lacking severe symptoms, remains an acceptable and recommended treatment.

Amoxicillin's cost, efficacy, safety profile, and palatability justify its recommendation as the first-line agent in the non–penicillin-allergic patient. It can be given at 80–90 mg/kg/day in two divided doses. This higher dose is preferred because it is effective against susceptible and intermediately resistant strains of *S. pneumonia*, and because 15% to 20% of children have poor gastrointestinal absorption of amoxicillin.

In patients with reported allergies, a distinction should be made between types I and II hypersensitivity. There is only minimal cross-reactivity to cephalosporins for patients with penicillin allergy, and the use of a second- or third-generation cephalosporin is generally considered safe, unless the child has a previous adverse reaction to cephalosporins. In patients with type II hypersensitivity, alternate treatment options include cefdinir (14 mg/kg per day in one or two doses), cefuroxime (30 mg/kg per day in two divided doses), cefpodoxime (10 mg/kg once daily), and intramuscular ceftriaxone (50 mg/kg per day) IV or IM for 1 to 3 days. Patients with type I sensitivity are problematic in that macrolides have poor sensitivity against *S. pneumoniae* and *H. influenzae*, and clindamycin has poor sensitivity against *H. influenzae*. In patients with severe allergy, we recommend azithromycin, 10 mg/kg, as a first dose, followed by 5 mg/kg for days 2 through 5 or clindamycin, 30 to 40 mg/kg per day tid.

Children who have taken amoxicillin in the previous 30 days, those with concurrent conjunctivitis, or those for whom coverage with β-lactamase–positive *H. influenzae* and *M. catarrhalis* is desired should be initially treated with high-dose amoxicillin-clavulanic acid (90 mg/kg per day amoxicillin and 6.4 mg/kg/day clavulanate) tid.[12]

Patients should be reevaluated in 3 days if there is no improvement. Treatment failure is defined by lack of clinical improvement in signs and symptoms, such as ear pain, fever, and TM findings of redness, bulging, or otorrhea. The reasons for treatment failure may include the wrong initial diagnosis or antibiotic resistance.[15] In these cases, treatment includes agents effective against the β-lactamase–producing organisms *H. influenzae* and *M. catarrhalis*. Recommended agents include amoxicillin-clavulanate (80–90 mg of the amoxicillin component/kg per day) and intramuscular ceftriaxone (50 mg/kg for 1–3 days). Table 62.2 summarizes the AAP guidelines for antibiotic treatment.

Patients with AOM for whom treatment with a conventional β-lactam antibiotic has failed and β-lactam–allergic patients for whom macrolide therapy has failed should be referred to a pediatric infectious disease specialist or otolaryngologist. These patients may need a myringotomy and treatment with a fluoroquinolone, which is not US Food and Drug Administration (FDA)–approved for children. Response to antibiotics is only one of a number of factors that affect clinical outcome. Other factors include eustachian tube function, coinfection with nonbacterial pathogens, and host immune response. Local practice patterns and antimicrobial sensitivities may also play a role in the type of treatment given. Treatment historically involved a 10-day course. Numerous studies have compared traditional treatment courses with shorter therapy, which is most appropriate for uncomplicated AOM. Patients younger than 2 years, those with TM perforations, or those with chronic or recurrent infections should be treated with a 10-day course. Children older than 2 years with a first-time infection and an intact TM can be treated with a 5- to 7-day course. The antibiotic treatment of AOM in adults is the same as for children. There is no indication for the use of

TABLE 62.2

Recommended Antibiotics for Initial or Delayed Treatment and for Patients Who Have Failed Initial Antibiotic Treatment

INITIAL IMMEDIATE OR DELAYED ANTIBIOTIC TREATMENT		ANTIBIOTIC TREATMENT AFTER 48–72 H OF FAILURE OF INITIAL ANTIBIOTIC TREATMENT	
Recommended First-Line Treatment	**Alternative Treatment (if Penicillin-Allergic)**	**Recommended First-Line Treatment**	**Alternative Treatment**
Amoxicillin (80–90 mg/kg/day in two divided doses)	Cefdinir (14 mg/kg/day in one or two doses)	Amoxicillin-clavulanate[a] (90 mg/kg/day of amoxicillin, with 64 mg/kg/day of clavulanate in two divided doses)	Ceftriaxone, 3 days Clindamycin (30–40 mg/kg/day in three divided doses), with or without third-generation cephalosporin
or	Cefuroxime (30 mg/kg/day in two divided doses)	or	Failure of second antibiotic
Amoxicillin-clavulanate[a] (90 mg/kg/day of amoxicillin, with 64 mg/kg/day of clavulanate [amoxicillin to clavulanate ratio, 14:1] in two divided doses)	Cefpodoxime (10 mg/kg/day in two divided doses)	Ceftriaxone (50 mg IM or IV for 3 days)	Clindamycin (30–40 mg/kg/day in three divided doses) plus third-generation cephalosporin
	Ceftriaxone (50 mg IM or IV/day for 1 or 3 days)		Tympanocentesis[b] Consult specialist[b]

[a]May be considered for patients who have received amoxicillin in the previous 30 days or who have otitis-conjunctivitis syndrome.
[b]Perform tympanocentesis and drainage if skilled in the procedure or seek a consultation from an otolaryngologist for tympanocentesis and drainage. If the tympanocentesis reveals multidrug-resistant bacteria, seek and infectious disease specialist consultation.
[c]Cefdinir, cefuroxime, and ceftriaxone are highly unlikely to be associated with cross-reactivity with penicillin allergy on the basis of their distinct chemical structures.
Adapted from Lieberthal AS, Carroll AE, Chonmaitree T, et al: Clinical Practice Guideline: the diagnosis and management of acute otitis media. Pediatrics 131:e964–e999, 2013.

antihistamines, decongestants, steroids, or tympanostomy tubes for an acute episode of AOM.

After a 10-day treatment with antibiotics, 50% of children may exhibit OME, but 90% of OME cases resolve within 3 months. However, about 30% to 40% of children have recurrent OME, and 5% to 10% of cases last 12 months or longer. The treatment of OME is controversial, with one large systematic review suggesting that tympanostomy tubes decrease effusion and improve hearing over a short period without affecting speech, language, or other functional outcomes.[16] There is little benefit from antibiotics, and they should not be used. Antihistamines, decongestants, steroids, or surgical procedures are not beneficial for patients with OME. Myringotomy and tympanostomy tubes may be beneficial in children who have had OME for more than 4 months with persistent hearing loss, those with hearing loss greater than 40 dB, children with structural damage to the TM or middle ear, and children with persistent OM who are at risk for speech, language, or hearing problems. Tonsillectomy is not beneficial, but adenoidectomy may be helpful for older children who have a specific indication, such as nasal obstruction or chronic adenoiditis.

Emergency clinicians may encounter three types of otitis media associated with a perforation of the TM:

1. Acute otitis media complicated by perforation of the tympanic membrane, presenting as otorrhea
2. Otitis media in patients with tympanostomy tubes
3. Chronic suppurative otitis media defined as tympanic membrane perforation with chronic inflammation of the middle ear and persistent otorrhea for 2 weeks to 3 months.

As noted earlier, tympanic membrane perforation is a known complication of AOM and, in most cases, will heal spontaneously. Patients presenting with AOM and otorrhea should be treated with oral high-dose amoxicillin, as if the TM were not ruptured. There is no advantage to adding topical therapy.

Tympanostomy tubes have also been used in recurrent AOM unresponsive to prophylactic antibiotics, for complications of AOM, and for complications of eustachian tube dysfunction, including TM retraction with hearing loss, ossicular erosions, and retraction pocket formation. Thus, tympanostomy tube insertion is one of the most common operative procedures for children in the United States, and emergency clinicians will frequently encounter patients with drainage from these tubes. In general, increased drainage from these tubes is as a result of an acute infection. The organisms involved are the same ones that cause AOM, particularly in children younger than 2 years, but *Pseudomonas aeruginosa, S. aureus, and Staphylococcus epidermidis* are also implicated. Fluoroquinolone drops are the only medications FDA-approved for use in patients with a nonintact tympanic membrane. In the acute setting, topical antibiotic administration with 5 ofloxacin drops to the affected ear bid or 4 drops of ciprofloxacin-dexamethasone bid for 7 days is an effective treatment. Systemic treatment (usually with amoxicillin-clavulanate, 45 mg/kg bid) should be reserved for patients showing signs of complicated or invasive infections or signs of systemic disease.

Chronic suppurative otitis media (CSOM) is one of the most common childhood infectious disease worldwide and is the most common cause of hearing impairment in the developing world, although it is infrequently seen in the developed world.[1] Again, *P. aeruginosa and S. aureus* are the most common organisms. Because of the tympanic membrane perforation, we recommend topical treatment with quinolone antibiotics.

Disposition

Patients should be seen in 48 to 72 hours if there is no improvement. Children who improve can be followed up in 8 to 12 weeks to ensure resolution of any residual effusion. Patients with complications need ear, nose, and throat (ENT) referral. Adults who have persistent OME warrant ENT referral to rule out nasopharyngeal carcinoma.

OTITIS EXTERNA

Principles

External otitis is an inflammation of the external auditory canal. The external auditory canal is lined with squamous epithelial cells

and cerumen glands that provide a protective lipid layer. This protective layer may be disrupted by high humidity, increased temperature, maceration of the skin after prolonged exposure to moisture, and local trauma (eg, cotton swabs or the use of hearing aids), resulting in the introduction of bacteria. Otitis externa (OE) is usually caused by *P. aeruginosa* and *S. aureus* but can also be polymicrobial. Occurring most often in the summer and in tropical climates, it is also known as swimmer's ear or tropical ear.

Clinical Features

The diagnosis is made clinically. The external auditory canal may be initially pruritic and may become erythematous and increasingly swollen. Symptoms include otalgia and ear fullness, as well as possible hearing loss or jaw pain. Physical findings include erythema or edema of the canal; pulling on the auricle or tragus classically reproduces the discomfort. There may be associated lymphadenitis, TM erythema, or local cellulitis. The disease may progress to a chronic form, with itching, eczema, and flaking of the epithelium, which may be from a bacterial, fungal, or dermatologic condition. In children, it is usually secondary to chronic OM.

Differential Diagnoses

It may be difficult to distinguish OE from OM with drainage from a ruptured TM, particularly in children. The TM may be erythematous in both conditions, and the edema may preclude diagnosis. The discharge may be from OE or a perforated TM and, in equivocal cases, it is prudent to treat for both conditions.

Otomycosis or fungal infection can occur as a primary or secondary infection and accounts for 10% of cases of OE. Itching is the prominent symptom, often with minimal pain or otorrhea. Aspergillosis is the cause in most cases. Otomycosis usually appears in individuals in tropical climates, diabetics, and immunocompromised patients. Treatment involves cleansing and the use of acidifying and antifungal ear drops, such as acetic acid, or a topical antifungal such as clotrimazole.

Furunculosis is a small, erythematous, and well-circumscribed infection of the cartilaginous portions of the external canal, usually caused by *S. aureus.* There is usually no drainage; treatment involves incision, drainage, and oral antibiotics effective for cellulitis based on local sensitivity. Cellulitis of the auricle and canal may cause erythema, induration, and other systemic signs. Clindamycin, 450 mg qid, will cover *S. aureus* and methicillin-resistant *Staphylococcus aureus* (MRSA). Skin conditions such as eczema, seborrhea, and contact dermatitis can all mimic otitis externa. A careful history about possible skin diseases, as well as medication and exposure history, should be elicited. Exposure to reactive metals such as nickel from devices such as hearing aids and chemicals from cosmetics and shampoos are also possible culprits.

Herpes zoster oticus, also known as the Ramsay Hunt syndrome, is a viral manifestation of disease affecting the auricle, with resulting facial paralysis that may involve multiple cranial nerves. It initially causes pain, erythema, and swelling, with vesicles developing approximately 3 to 7 days later. Treatment consists of analgesia and antivirals (acyclovir, 800 mg five times/day, famciclovir, 500 mg, or valacyclovir, 1000 mg tid), but there is little evidence supporting its efficacy.

Diagnostic Testing

OE is a clinical diagnosis. No additional testing is indicated.

Management

Treating OE involves cleaning the canal and treating the infection. The external canal may be cleaned with a small cotton swab or combination of gentle suctioning and irrigation, depending on the amount of obstructing exudates and whether there is an intact TM. Cleansing solutions include tap water, sterile saline, 2% acetic acid, and Burow's solution.

Topical antibiotics are highly effective for OE treatment, with clinical cure rates of 65% to 80% within 10 days. A combination of polymyxin B, neomycin, and hydrocortisone (Cortisporin) can be given at a dose of 3 or 4 drops to the affected ear qid, although occasionally patients develop cutaneous sensitivity to the neomycin. Ofloxacin (5 drops) or ciprofloxacin with hydrocortisone (3 drops) bid may result in improved patient compliance. The addition of steroid drops may decrease inflammation and the formation of granulation tissue in the canal, but this has not been proven.

Care should be taken if there is a concern for TM perforation. As noted, quinolone drops have a better safety profile than neomycin-containing drops, which are ototoxic, especially after prolonged or repeated use.[17,18] Having the patient lie down for 5 minutes after the solution has been placed may obviate the need for packing. Commercially available wicks made of compressed cotton or hydroxycellulose facilitate medication delivery. The wick is placed 10 to 12 mm into the canal, moistened with antibiotic drops, and left in place for 2 to 3 days. The wick generally falls out or, if left in place, may become a foreign body in the ear. Therefore, a patient should follow up with her or his primary care physician. There is no evidence that systemic antibiotics alone or in combination with topical preparations improve treatment outcome compared with topical antibiotics alone, but systemic medication, such as ciprofloxacin (500 mg bid), are indicated for immunocompromised patients with diabetes or HIV infection or for those with infections involving the skin and periauricular areas.[17] OE can be extremely painful, and severe symptoms may require opiate analgesia. Topical anesthesia, such as benzocaine with or without antipyrine, may also be used for pain relief.

Disposition

Patients with otitis externa rarely require admission. If it does not respond to therapy in 2 to 3 days, other conditions such as necrotizing external otitis should be considered. Patients who have a wick placed should be evaluated in 2 to 3 days to ensure improvement of the condition and that the wick is removed.

NECROTIZING (MALIGNANT) EXTERNAL OTITIS

Principles

Previously known as malignant otitis externa because of its associated high mortality rate, necrotizing external otitis (NEO) is an extremely form of OE. Patients affected include older diabetics, those with acquired immunodeficiency syndrome (AIDS) and, rarely, immunocompromised children. *Pseudomonas* is the predominant pathogen, but *S. aureus, S. epidermidis, Proteus mirabilis, Klebsiella, Aspergillus,* and *Salmonella* have all been described as causative organisms. The infection begins in the external canal and progresses through the periauricular tissue and cartilaginous bony junction of the external auditory meatus. It then spreads into the adjacent tissues along clefts in the floor of the meatus known as the fissures of Santorini. It may spread to the base of the skull at the temporal bone, with a resultant *skull-base osteomyelitis,* another term often used to describe this entity. The facial nerve is the first cranial nerve affected, but other nerves may also be involved. The pathogenesis is uncertain but may be related to vascular insufficiency or immune dysfunction.

Clinical Features

Patients may have persistent otorrhea unresponsive to topical medications, severe otalgia, headache, and periauricular pain and swelling. The diagnosis should be considered in patients at risk who have a prolonged course of OE. The characteristic clinical finding is granulation tissue on the floor of the ear canal at the bony cartilaginous junction. Cranial nerve VII is most commonly involved; involvement manifests with facial paralysis, which occurs when the stylomastoid foramen is involved. Further extension can result in involvement of the glossopharyngeal, vagal, spinal accessory, hypoglossal, trigeminal, and abducens nerves. Cranial nerve involvement is not associated with increased mortality rates. Additional complications include meningitis, brain abscess, and thrombosis of the sigmoid sinus.

Differential Diagnoses

Patients with necrotizing otitis will present with severe ear pain. Other differential considerations include severe otitis externa, otitis media, otitis media complications, trauma, and referred pain from the teeth, sinuses, throat or temporal mandibular joint.

Diagnostic Testing

There is no single diagnostic criterion for necrotizing external otitis. The diagnosis is made from a range of clinical, laboratory, and radiographic findings. The C-reactive protein (CRP) level and erythrocyte sedimentation rate (ESR) may be elevated, but they are nonspecific markers. In the ED, computed tomography (CT) is the initial study of choice and, in most cases, will identify bony erosion and soft tissue abnormalities. Magnetic resonance imaging (MRI) is better at delineating responses to therapy. This disease should be considered in all patients with risk factors who have failed to respond to antimicrobial therapy for temporal bone inflammation and otalgia.[19]

Management

If NOE is suspected, consultation should be made with an otolaryngologist. The patient presentation will determine disposition. Patients who appear ill require admission for IV fluoroquinolones, such as ciprofloxacin, 400 mg IV q8h, to ensure that there is an adequate clinical response. The patient can then be switched to oral ciprofloxacin, given its bioavailability and penetration to bone. Treatment may be required for 6 to 8 weeks. Although extensive surgical treatment was previously required, its use is now limited to diagnostic confirmation or débridement of granulation tissue. Although some have recommended hyperbaric treatment for advanced disease with significant skull base or intracranial involvement, there is little evidence of its effectiveness.

Disposition

The decision for admission versus outpatient management should be made in consultation with an otolaryngologist.

MASTOIDITIS

Principles

Mastoiditis is the most frequent suppurative complication of OM, although the incidence of acute and chronic mastoiditis has decreased significantly since the advent of antibiotics. Although it is still associated primarily with AOM, some patients have not had a preceding episode of OM. Mastoiditis also has been described as a complication of leukemia, mononucleosis, sarcoma of the temporal bone, and Kawasaki disease.

Acute mastoiditis is a natural extension of middle ear infections because the mastoid air cells are generally inflamed during an episode of AOM. The aditus ad antrum is a narrow connection between the middle ear and mastoid air cells. If this connection becomes blocked, a closed space is formed, with the potential for abscess development and bone destruction. The infection may spread from the mastoid air cells by venous channels, resulting in inflammation of the overlying periosteum. Progression results in the destruction of the mastoid bone trabeculae and coalescence of the cells, resulting in acute mastoid osteitis or coalescent mastoiditis. The resulting pus may track through many routes: (1) through the aditus ad antrum, with resultant spontaneous resolution; (2) laterally to the surface of the mastoid process, resulting in a subperiosteal abscess; (3) anteriorly, forming an abscess below the pinna or behind the sternocleidomastoid muscle of the neck (often called a Bezold abscess); (4) medially to the petrous air cells of the temporal bone, resulting in a rare condition known *as petrositis*; and (5) posterior to the occipital bone, resulting in osteomyelitis of the calvaria or a Citelli abscess.

Chronic mastoiditis is generally a complication of chronic OM. There may be extensive invasion of granulation tissue from the middle ear into the mastoid air cells. Another entity, latent or masked mastoiditis, also has been described. It is indolent in nature, with minimal signs and symptoms, little or no fever, and a history of otalgia. The TM may be intact or perforated. Suspicion should be raised by the presence of intracranial complications without an apparent source. Patients at risk include newborns and immunosuppressed patients (eg, those who have undergone recent chemotherapy or steroid administration, diabetic or geriatric patients).

S. pneumoniae continues to be the leading cause of acute mastoiditis in the post–heptavalent pneumococcal vaccine era.[20] The introduction of the pneumococcal conjugate vaccine has resulted in an increase of a particularly virulent strain, serotype 19A, although the PCV-13 included this serotype. Other organisms include group A streptococci, *S. aureus, H. influenzae, and P. aeruginosa.*[21] Chronic mastoiditis also often has mixed cultures, with *P. aeruginosa* being the predominant organism.

Clinical Features

Clinical findings in acute mastoiditis include fever, headache, otalgia, and erythema. Pain is universally present. There are no specific diagnostic criteria, but the most common physical findings are postauricular erythema and tenderness, protrusion of the auricle, and an abnormal TM. The TM is similar to that in AOM—erythema, bulging, and decreased mobility—but may be normal in 10% of cases. Suspicion should be heightened if symptoms of AOM have lasted longer than 2 weeks. In chronic mastoiditis, symptoms include persistent drainage through the perforated TM, redness, edema, and retroauricular sensitivity.

Differential Diagnoses

The differential diagnosis includes severe otitis media, external otitis, skull fracture, lymphadenopathy or lymphadenitis, and deep space neck infections.

Diagnostic Testing

Although the diagnosis of mastoiditis can be made clinically in patients with typical findings, a CT scan is indicated in patients with neurologic symptoms, when an intracranial complication is suspected, or there is failure to improve with conservative therapy.[22] Fig. 62.1 is a CT scan of acute mastoiditis.

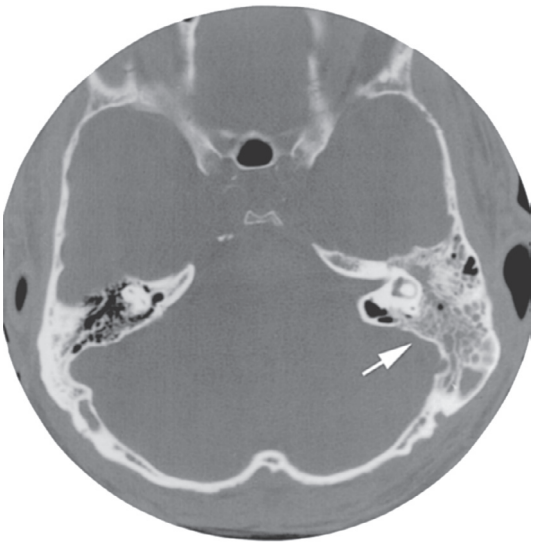


Fig. 62.1. CT scan of mastoiditis. (From McWhorter AJ, Limb CJ, Niparko JK: Otologic and skull base emergencies. In Eisele DW, McQuone SJ, editors: Emergencies of the head and neck, St. Louis, 2000, Mosby, p 384.)

Management

The initial treatment of choice in the emergency department is the administration of antibiotics, such as vancomycin, 15 to 20 mg IV bid, and a third-generation cephalosporin such as ceftriaxone (50 mg/kg per day). Surgical procedures may range from myringotomy and tympanostomy tube placement (for drainage and identification of the offending organism) to mastoidectomy and drainage for more extensive disease progression.

Disposition

Hospitalization is usually necessary for the administration of IV antibiotics. Early otolaryngologic referral is also recommended for possible aspiration and drainage of the middle ear, as well as the management of any potential complications.

SUDDEN HEARING LOSS

Principles

Sudden sensorineural hearing loss (SSNHL), defined as the idiopathic loss of hearing of 30 dB over at least three test frequencies occurring over a period of less than 3 days, is considered an otolaryngologic emergency. Any age group can be affected, but the peak incidence occurs in the fifth or sixth decade of life, with an equal gender distribution. The overall incidence ranges from 5 to 20/100,000 people/year. Its severity ranges from difficulty with conversation to complete hearing loss.

SSNHL is idiopathic in 70% of cases, infectious in 13%, and related to otologic disease, trauma, vascular disease, hematologic disorders, or neoplasm in the vast majority of other cases.[23] A delay in diagnosis is common because the patient may report ear fullness that is often attributed to cerumen impaction or congestion from upper respiratory infections. Tinnitus is a common finding. The likelihood of recovery is related to the severity of the hearing loss, age of the patient, and associated vestibular symptoms. A history should include the time of onset, history of trauma or recent illnesses, medications, and presence of otologic and neurologic symptoms.

Clinical Features

The physical examination includes a thorough inspection of the external canal and TM integrity.

Differential Diagnoses

The differential for hearing loss is broad and can be differentiated into causes that involve the outer, middle, or inner ear. Outer ear causes include cerumen impaction and OE. Middle ear causes include otitis media and tympanic membrane perforation. Inner ear causes include medications, barotrauma, and autoimmune disease.

Diagnostic Testing

Weber's test for hearing and Rinne's test may help in distinguishing conductive versus sensorineural deficits. A comprehensive neurologic examination, including cranial nerve and cerebellar testing, may localize brainstem involvement. Laboratory testing and CT scanning are not indicated in the ED evaluation unless the physical examination points to a space-occupying lesion (ie, focal neurologic deficits not referable to the ear). MRI of the brain with gadolinium is the study of choice to identify retrocochlear pathology but should be performed in consultation with an otolaryngologist.

Management

A tapered dose of oral steroids is the most common treatment, although their efficacy is unproven. The dose is 1 mg/kg, up to 60 mg, tapered over 10 to 14 days.[24] Additional treatments have included intratympanic steroids, hyperbaric oxygen, antiviral therapy, zinc,[25] vasoactive and hemodilution therapies, dextran, and magnesium, all with mixed results. Given the lack of treatment options for this condition, we recommend that a steroid taper be offered.

Disposition

Patients should get expeditious ENT referral on discharge from the ED.

EPISTAXIS

Principles

Epistaxis is a common otolaryngologic problem, with 60% of people experiencing it in their lifetime, although only 6% require medical treatment.[26] It accounts for about 1 in 200 emergency room visits,[27] with less than 0.2% ultimately requiring hospitalization.[28] There is a bimodal distribution of children younger than 10 years and adults older than 50 years. Epistaxis is more common in colder seasons and in northern climates because of decreased humidity and subsequent drying of the nasal mucosa.[29] Nasal bleeding is a frightening condition for patients but is seldom life-threatening. A solid understanding of physiology and treatment allows for prompt and efficient management of the disorder.

Anterior epistaxis accounts for 90% of all nosebleeds and usually involves Kiesselbach's plexus on the anteroinferior nasal septum. Epistaxis is unilateral and can be controlled with anterior packing. Accounting for 10% of nosebleeds, and usually arising from a posterior branch of the sphenopalatine artery, posterior epistaxis differs from anterior bleeding in that it is more severe and occurs mostly in older adults with multiple comorbidities.[30]

Three arteries with anastomoses between them supply the nasal area. The sphenopalatine artery supplies the turbinates and

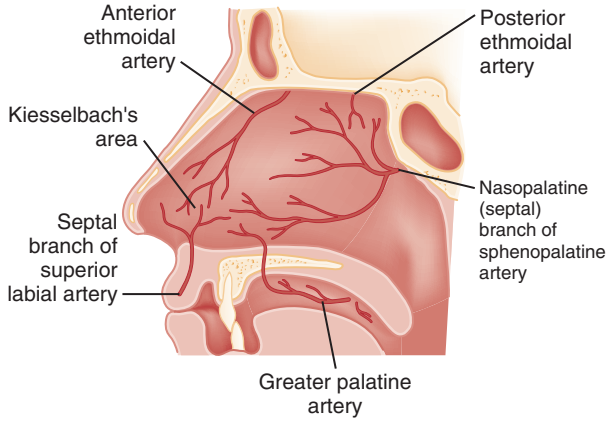


Fig. 62.2. Arterial supply to the medial wall of the nose.

meatus laterally and the posterior and inferior septum medially. The anterior and posterior ethmoidal arteries from the ophthalmic branch of the internal carotid artery supply the superior mucosa medially and laterally. The superior labial branch of the facial artery provides circulation to the anterior mucosal septum and anterior lateral mucosa (Fig. 62.2).

There are many reasons for epistaxis, but the most common are an upper respiratory infection with concomitant mucosal congestion and vasodilation and trauma, either accidental or iatrogenic (ie, nose picking; Box 62.1).

Clinical Features

A past medical history with particular emphasis on trauma, medical conditions, and medications that could cause epistaxis should be elicited. Patients often are anxious and hypertensive. An elevated blood pressure is usually from stress and anxiety and resolves with treatment. Hypertension has never been shown to cause epistaxis, although it can worsen the bleeding when present.[31] Sedation with a benzodiazepine or narcotic may help these patients.

Differential Diagnosis

The differential diagnosis includes nasal trauma, infections, nasal foreign bodies, and bleeding disorders.

Diagnostic Testing

Identifying the source of the bleeding is often difficult. If the nose is actively bleeding, the patient should clear clots by blowing the nose and then applying bilateral pressure on the nasal septum by compressing the cartilaginous part of the nose for 10 to 15 minutes. Spraying oxymetazoline into each nare twice before applying pressure will optimize hemostasis and facilitate inspection after the pressure is released. This simple maneuver also educates the patient on how to self-manage further episodes. It is important to optimize the examination. The floor of the nose should be parallel to the room floor. If the head is tilted, only the anterior and upper aspect of the nares can be visualized. The nasal speculum should be opened in a vertical direction rather than side to side in the nares, so as not to obscure the septum, which is the area of greatest interest. During this time, materials for illumination, suction, visualization, and treatment should be assembled. Discharge without identification and treatment of the bleeding site often results in recurrences. Anterior clots may give the appearance of posterior epistaxis if the blood runs posteriorly. Persistent bleeding should be controlled with pledgets soaked in cocaine, lidocaine-epinephrine, or oxymetazoline to promote vasoconstriction and anesthesia. Routine laboratory testing is usually unnecessary unless the patient is anticoagulated or has an underlying condition.

BOX 62.1

Causes of Epistaxis

LOCAL CAUSES
Nasal or facial trauma
Upper respiratory tract infections
Nose picking
Allergies
Low home humidity
Nasal polyps
Foreign body in the nose
Environmental irritants
Nasopharyngeal mucormycosis
Traumatic internal carotid artery aneurysm
Chlamydial rhinitis neonatorum
Neoplasms
Septal deviation
Surgery (postoperative epistaxis)

IDIOPATHIC EPISTAXIS
Habitual
Familial

SYSTEMIC CAUSES
Atherosclerosis of nasal blood vessels
Anticoagulant therapy
Pregnancy
Barotrauma
Hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber disease)
Blood dyscrasias (eg, hemophilia, leukemia, lymphoma, polycythemia vera, anemias, idiopathic thrombocytopenic purpura, granulocytosis, inherited platelet disorders, acquired platelet disorders [ie, aspirin use])
Hepatic disease
Rupture of internal carotid artery aneurysm
Diabetes mellitus
Alcoholism
Vitamin K deficiency
Folic acid deficiency
Chronic nephritis
Chemotherapy
Blood transfusion reactions
Migraine headache
Chronic use of nasal vasoconstrictors
Cocaine use
Drug induced thrombocytopenia

Management

Identify and treat the source of bleeding, because the most significant risk factor for recurrent bleeding is not identifying the bleeding point.[32] Application of silver nitrate chemically cauterizes the area but is often unsuccessful during active bleeding, so hemostasis should be secured first. With 4 to 5 seconds of application, nitric acid is formed and coagulates tissue. Coagulation should never be maintained longer than 15 seconds because septal damage may occur. The area should be cauterized from the periphery to the center and superiorly to inferiorly to avoid blood, which renders the silver nitrate sticks ineffectual. Bilateral application of silver nitrate to the septum is not advised because it may deprive the septum of a blood supply and theoretically could lead to necrosis.

If cautery is unsuccessful, topical thrombogenic agents, such as absorbable gelatin sponge (Gelfoam) and absorbable knitted fabric (Surgicel), can be tried. Tranexamic acid may be an option if bleeding continues. Tranexamic acid works by irreversibly binding and blocking the lysine binding sites on plasminogen molecules, resulting in inhibition of plasminogen activator and fibrinolysis. It has also been successfully used in 109 patients and resulted in much quicker resolution of bleeding and faster ED discharge when compared to nasal packing.[33] The injectable solution (500 mg in 5 mL) is applied to a 15-cm nasal pledget and applied to the anterior nares.[33]

If bleeding persists, the next step is the use of a nasal tampon. Nasal tampons work by three mechanisms: direct pressure, decreased bleeding from mucosal irritation from the foreign body, and indirect pressure from further surrounding clot formation. Cutting them to fit the contour of the nares and lubricating them with an antibiotic ointment makes the application easier. For large noses, a second tampon may be required. Occasionally, for uncontrolled bleeding despite the presence of a tampon, a second tampon should be inserted into the opposite nare. If bleeding still continues, a nasal balloon catheter with fibrin colloid material, such as Rapid Rhino (Smith & Nephew, Austin, TX), may be used. These devices are moistened with saline, so lubricants are unnecessary. They are placed in the floor of the nose and inflated with air. The fibrin colloid forms a hemostatic dressing. A second balloon in the opposite nose may be required if one side is unsuccessful.

Toxic shock syndrome (TSS) due to *S. aureus* has been reported in patients with nasal packing. Although many providers prophylactically give antibiotics after nasal packing, no study has shown that antibiotics are preventive for TSS or sinusitis, and the incidence of TSS is rare (16/100,000 population). We do not recommend routine antibiotic prophylaxis after nasal packing. Packing is uncomfortable and the patient may require opioids in the ED and on discharge. The packs are left in for 48 hours to minimize rebleeding and removed at 48 hours to avoid tissue necrosis associated with prolonged placement.

Posterior epistaxis is suggested when bleeding occurs with a properly placed anterior nasal pack. In this case, a posterior pack is necessary with a Foley catheter or commercially available balloon. A standard Foley catheter may be inserted into the nasopharynx, partially inflated with 5 to 7 mL of water, and then pulled anteriorly, creating pressure posteriorly with an additional 5 to 7 mL of water added to the balloon, but caution should be exercised to avoid pressure necrosis. Water, rather than saline, should be used because saline can crystallize and cause problems with balloon deflation. Vaseline gauze should be packed firmly around the catheter anteriorly. Fig. 62.3 shows how the Foley catheter is placed.

The commercially available devices have anterior and posterior balloons. Similar to Foley placement, the device is placed into the nose, inflated, and pulled anteriorly. Once seated, the anterior balloon should be slowly inflated to the point that the patient can tolerate.

If these techniques do not provide successful control, otolaryngologic consultation is necessary. Surgical ligation has been the treatment of choice for intractable bleeding but endovascular embolization has emerged as a treatment alternative. The decision to choose surgery over embolization is influenced by factors such as patient comorbidity, presence of anticoagulation, institutional experience, patient preference, and health care costs.[27,34,35] Transnasal endoscopic surgery has advantages in that it visualizes bleeding location, improves the diagnosis of other causes, and is associated with lower health care costs and complications such as blindness. The advantages of embolization include avoiding general anesthesia, improving the diagnosis of vascular pathology, and causing less trauma to the nasal mucosa.[30] In one national survey, patients who underwent endovascular embolization had higher rates of head and neck cancer, hereditary hemorrhagic telangiectasia, and arteriovenous malformation compared with patients who underwent surgical ligation.[34]

Disposition

There has been concern that patients with posterior nasal packs may develop hypoxia as a result of a nasopulmonary reflex. However, there is little evidence to support this theory.[36] Adverse respiratory events are due to a combination of factors such as sedation, underlying cardiovascular or pulmonary disease, and severe obstructive sleep apnea.[37] Most patients with posterior nasal packing can be admitted to a setting with continuous pulse oximetry, but patients with serious comorbidities such as heart disease or obstructive sleep apnea may require a higher level of care.[38]

SIALOLITHIASIS

Stones of the salivary glands occur in 1% of the population. They are usually found in those between 30 and 50 years of age. The most common gland affected is the submandibular (submaxillary) gland, accounting for 80% to 95% of cases. Stones are found less commonly in the sublingual and parotid glands. Sialolithiasis is uncommon in children, occurring in only 3% to 5% of the population.[37]

The exact causative mechanism is unclear, but sialolithiasis is thought to be due to increased viscosity of the saliva and the long upward curvature of the submandibular (Wharton's) duct. Stasis and inflammation result in precipitation of calcified stones after a nidus of a complex glycoprotein combines with calcium and phosphate. Risk factors include dehydration, diuretic or anticholinergic medications, trauma, gout, and a history of smoking.[39]

Clinical Features

Leading to swelling and pain, obstruction by a sialolith is usually associated with mealtime, when salivary secretion is enhanced.[40] Patients generally present with pain, swelling, and tenderness of the gland. If the gland is infected, the patient may have systemic symptoms, such as fever or chills. The area may be erythematous, with purulence coming from the duct, a condition termed *sialadenitis*.[41] *S. aureus, Streptococcus viridans, S. pneumoniae,* and *H. influenzae* predominate in bacterial infections. Children differ in that they have a shorter duration of symptoms, and their stones present more distally in ducts than those found in adults.[37]

Differential Diagnosis

The differential diagnosis includes salivary gland pathology, lymph node disease, granulomatous process, soft tissue mass, and neoplastic lesion.

Diagnostic Testing

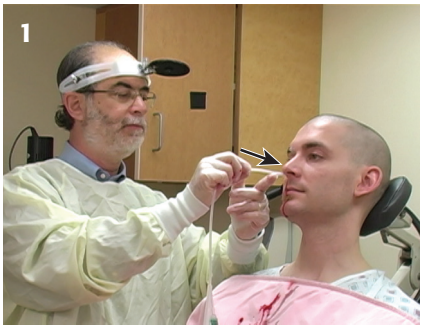
CT without contrast is very sensitive for calculi of all sizes and remains the gold standard, although there is the associated risk of ionizing radiation. Although there have been reports of ultrasonography recognizing up to 90% of stones larger than 2 mm, it does not allow reliable exclusion of small salivary calculi.[42] Both modalities may help identify other causes of inflammation, such as an abscess or cellulitis.

Management

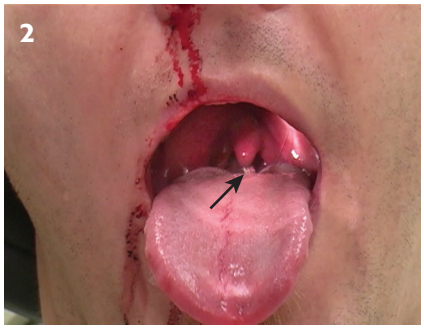
If the stone is palpable, gently massage the gland in an attempt to extract the stone. Additional measures include sialogogues (tart

EPISTAXIS MANAGEMENT: POSTERIOR PACKING WITH INFLATABLE DEVICES

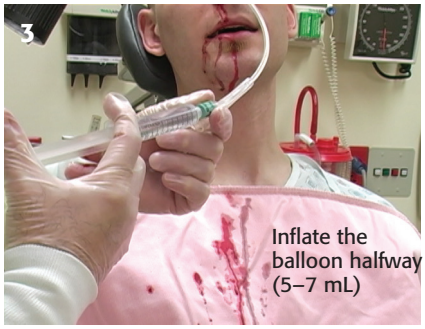
A



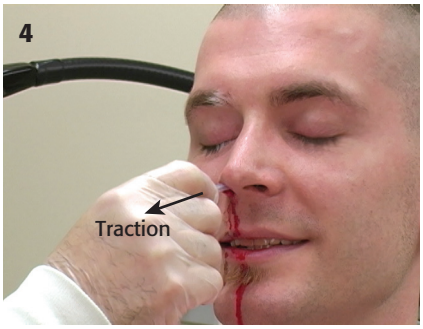
Insert a 12-Fr Foley catheter through the naris and into the posterior pharynx.



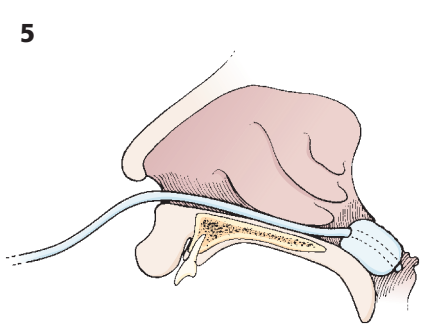
Look into the mouth to confirm that the catheter is properly positioned.



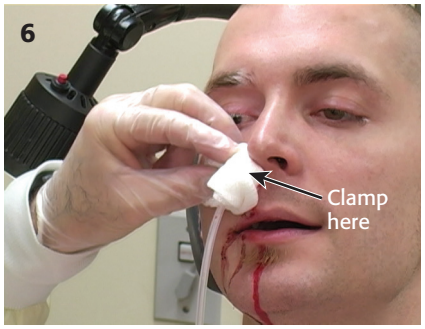
Inflate the balloon halfway with about 5–7 mL of water.



Slowly pull the catheter into the posterior nasopharynx up against the posterior aspect of the middle turbinate.

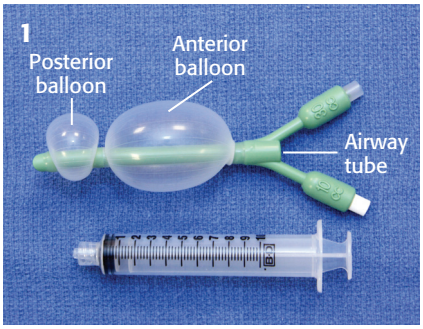


Foley catheter in proper position in the posterior nasopharynx. Inflate the balloon with another 5–7 mL of water.

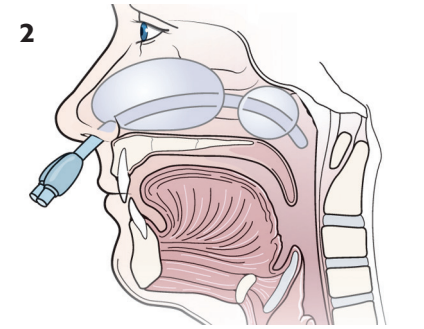


While maintaining traction, place anterior packing with layered gauze. Packing of the opposite side may be required to prevent septal deviation. Place a piece of gauze on the exposed catheter and secure with an umbilical clamp.

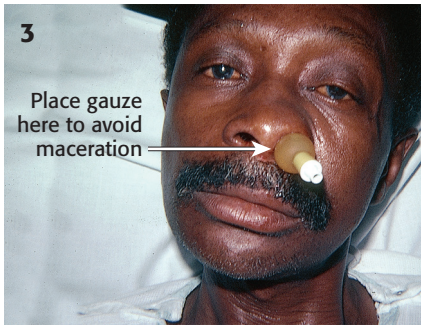
B



Double-balloon epistaxis catheters have both an anterior and posterior balloon, and some have an integral airway tube. These devices serve as an anterior and posterior pack. They are easily inserted and are often successful in the temporary control of posterior epistaxis in the ED.



Insert the lubricated device along the nasal floor as far back as possible. Inflate the posterior balloon halfway with air, apply traction to pull the balloon up against the middle turbinate, and then complete the inflation. Maintain the position of the balloon and then inflate the anterior balloon with 30 mL of air.



This patient with posterior epistaxis was successfully treated in the ED and discharged. Historically, most patients with posterior packs were admitted to the hospital; however, the ease and safety of balloon devices allow selected patients to be treated as outpatients. Consider admission for older adults and those with pulmonary or cardiovascular disease.

Fig. 62.3. Management of epistaxis—posterior packing with inflatable devices. **A,** Foley catheter technique. **B,** Dual-balloon tamponade catheter. (Adapted from Riviello RJ: Otolaryngologic procedures. In Roberts JR: Roberts and Hedges' clinical procedures in emergency medicine, Philadelphia, 2013, Elsevier/Saunders, p 1330.)

hard candies to promote glandular secretions), analgesia with antiinflammatory medications, or opioids. When infection is present, antibiotics covering the affected organisms, such as cephalexin, 500 mg qid, or clindamycin, 450 mg tid (in the penicillin-allergic patient), are appropriate.

Disposition

Stones larger than 5 mm or stones located within the gland or in the proximal duct are often resistant to conservative measures. These may require surgical or minimal invasive treatment by an otolaryngologist or oral surgeon.[43]

NECK MASSES

Principles

Neck masses are a relatively common clinical finding, with a multitude of causes. The differential diagnosis can generally be broken down into three categories—inflammatory, congenital, or neoplastic. Children and young adults are more likely to have benign disorders, such as inflammatory or congenital abnormalities, including thyroglossal or branchial cleft cysts. Adult neck masses are more likely to be neoplastic. In general, 80% of nonthyroid neck masses in adults are neoplastic, of which 80% are malignant. In children, however, more than 80% of neck masses are benign. This is often referred to as the rule of 80, or the 80% rule. Risk factors that may predispose patients to ENT malignancies include alcohol and tobacco use, viruses such as herpes, genetics, diet, and excessive exposures to ultraviolet sunlight, dust, or chemicals.

Identifying the parotid and submandibular glands, thyroid cartilage, thyroid gland, and lymph nodes can help distinguish normal structures from other masses (Fig. 62.4). The neck is divided into cervical triangles, with the sternocleidomastoid muscle as the common boundary. The anterior portion is bordered by the midline of the neck, inferior aspect of the mandible superiorly, and anterior border of the neck posteriorly. Lesions of the skin, scalp, oral cavity, oropharynx, hypopharynx, larynx, and tongue may manifest here. The posterior triangle is bordered by the sternocleidomastoid anteriorly, posteriorly by the trapezius muscle, and inferiorly by the clavicle. Lesions in this area may include those from the nasopharynx and metastatic lesions from the lung and gastrointestinal and genitourinary tracts.

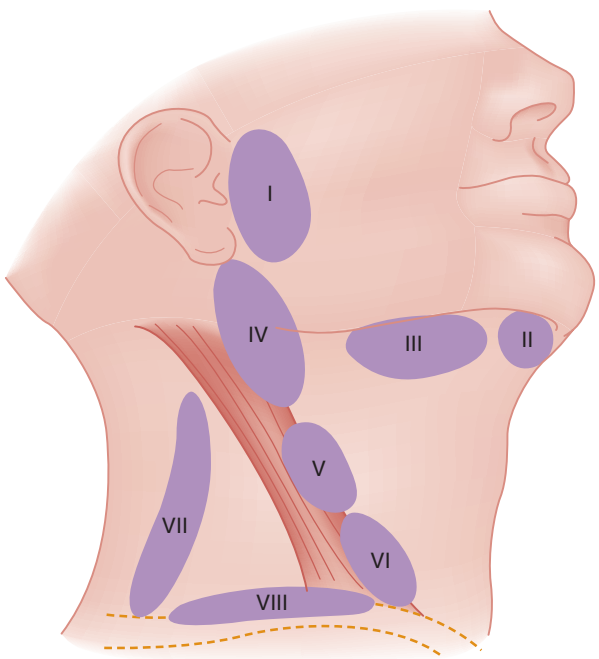


Fig. 62.4. Major lymph node groups in the head and neck. *I,* Parotid nodes; *II,* submental nodes; *III,* submandibular nodes; *IV,* jugulodigastric nodes (superior jugular nodes); *V,* midjugular nodes; *VI,* lower jugular nodes; *VII,* spinal accessory nodes; *VIII,* subclavian nodes. Groups VI and VII are often termed *scalene nodes*. (Adapted from Moloy PJ: How to [and how not to] manage the patient with lump in the neck. In American Academy of Otolaryngology–Head and Neck Surgery Foundation: Common problems of the head and neck region, Philadelphia, 1995, WB Saunders, p 134.)

Clinical Features

Important associated symptoms include dysphagia, odynophagia, otalgia, stridor, speech disorders, and globus phenomena. Dysphagia, or difficulty swallowing, may be caused by physical obstruction or neurologic disorders. Odynophagia is pain on swallowing and can have a number of causes, such as tonsillitis or carcinoma of the pharynx. In an adult, a sore throat that lasts for several weeks should raise the suspicion of a neoplastic process. Otalgia is pain felt in the ear that may be referred from the larynx, pharynx, or cranial nerves V, IX, or X. Referred ear pain is an ominous sign in adults and should be presumed to be cancer until proved otherwise. Similarly, unilateral OME in adults should be considered nasopharyngeal carcinoma until proven otherwise.

Stridor, specifically inspiratory stridor, is diagnostic of upper airway obstruction. It localizes a lesion to above or at the level of the larynx. In adults, the presence of stridor with a neck mass increases the possibility of carcinoma. Speech disorders, particularly so-called hot potato speech, are suggestive of space-occupying lesions above the oropharynx, such as a peritonsillar abscess. Globus is the symptom of having a lump in the throat. It has occurred in almost everyone at one time or another, is localized to the pharynx, and is often a functional complaint. Hoarseness is a fairly common complaint, with a myriad of causes ranging from viral pharyngitis to laryngeal cancer. Also, similar to the term *dizziness,* the term *hoarseness* has many descriptions, including breathiness, muffling, harshness, scratchiness, and unnatural deepening of the voice. Hoarseness lasting longer than 2 weeks should be investigated further. Additional history about the location of the mass, rate of growth, presence of pain, and constitutional symptoms, such as fever, night sweats, and weight loss, are also helpful.

The head and neck examination may identify masses, lesions, mucosal ulcerations or discolorations, and cranial nerve abnormalities. The mass itself should be palpated for location, size, and consistency. Benign lymph nodes are generally mobile, soft, fleshy, and smaller than 1 to 1.5 cm, so any hard nodes larger than 1.5 cm with decreased mobility should be considered abnormal and as warning signs of malignancy.

Differential Diagnoses

Box 62.2 lists common possibilities for the differential diagnosis of neck masses.

Diagnostic Testing

The diagnostic strategy is tailored to results of the history and physical examination. Patients with hoarseness lasting longer than 2 weeks should be referred to an otolaryngologist for a flexible endoscopic examination unless they have developed acute stridor, dyspnea, or sense of acute deterioration. These patients should have otolaryngologic consultation in the ED, and most will need flexible endoscopic examination of the upper airway. In the ED, chest radiography is an initial test to identify possible lung pathology as a source. CT of the neck with contrast is the initial study of choice to delineate significant neck masses better.

BOX 62.2

Differential Diagnosis of Neck Masses

INFLAMMATORY
Adenitis
Bacterial (*Streptococcus, Staphylococcus*)
Viral (HIV, EBV, HSV)
Fungal (coccidioidomycosis)
Parasitic (toxoplasmosis)
Cat scratch disease
Tularemia
Local cutaneous infections
Sialoadenitis (parotid and submaxillary glands)
Thyroiditis
Mycobacterium avium-intracellulare
Mycobacterium tuberculosis

CONGENITAL OR DEVELOPMENTAL
Brachial cleft cyst
Thyroglossal duct cyst
Dermoid cyst
Cystic hydromas
Torticollis
Thymic masses
Teratomas
Ranula
Lymphangioma
Laryngocele

NEOPLASTIC
Benign
Mesenchymal tumors (eg, lipoma, fibroma, neural tumor)
Salivary gland masses
Vascular abnormalities (eg, hemangioma, AVM, lymphangioma, aneurysm)

Malignant
Primary tumors
Sarcoma
Salivary gland tumor
Thyroid or parathyroid tumors
Lymphoma

Metastasis
From primary head and neck tumors
From infraclavicular primary tumors (eg, lung or esophageal cancer)

AVM, Arteriovenous malformation; *EBV,* Epstein-Barr virus; *HIV,* human immunodeficiency virus; *HSV,* herpes simplex virus.

Management and Disposition

Most masses in children are inflammatory. Thus, it is a reasonable strategy to start the patient on antibiotics, with a 2-week follow-up. If inflammation is thought to be the cause of the neck mass in an adult, a similar strategy can be used. However, adults need ENT referral if the mass does not resolve in 2 weeks, is enlarging or fixed, or is associated with matted cervical lymph nodes, or if the masses are noted in the parotid or thyroid gland.

KEY CONCEPTS

- Most cases of AOM resolve spontaneously. Nontoxic children from 6 months to 2 years of age with unilateral AOM and those older than 2 years with unilateral or bilateral AOM may be observed for 3 days to determine whether antibiotics are required. When indicated, amoxicillin is the initial choice for treatment of AOM, 80 to 90 mg/kg per day.
- Otitis externa is treated with topical antibiotic drops. Only fluoroquinolone drops are FDA-approved for use when a tympanic perforation may be present. Necrotizing OE should be considered in immunocompromised patients who have persistent otitis externa.
- Patients with epistaxis with posterior nasal packing should be admitted to the hospital.
- Bullous myringitis is caused by the usual organisms that cause otitis media.
- Adult patients with AOM should be treated with amoxicillin, 500 mg tid.
- The diagnosis of AOM is made by a bulging TM and signs and symptoms of acute infection.
- Acute hearing loss is most often idiopathic. A 10- to 14-day steroid taper is usually prescribed but is not known to provide benefit.
- Hoarseness or an unexplained neck mass that persists for longer than 2 weeks requires ENT referral.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

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CHAPTER 62: QUESTIONS & ANSWERS

62.1. Which of the following clinical symptoms is most useful in diagnosing acute otitis media (OM)?
- **A.** Cough
- **B.** Decreased appetite
- **C.** Ear pain
- **D.** Fever
- **E.** Vomiting

Answer: C. Although all the symptoms of acute OM are nonspecific, ear pain appears to be the most useful.

62.2. A 56-year-old man presents with sudden onset of hearing loss in his left ear. He also complains of tinnitus. His neurologic examination is otherwise unremarkable. What should be the next step in the patient's management?
- **A.** Consult a neurologist.
- **B.** Consult an otolaryngologist.
- **C.** Obtain a head computed tomography (CT) scan.
- **D.** Obtain a magnetic resonance imaging (MRI) scan with gadolinium.
- **E.** Start a steroid taper.

Answer: B. Sudden sensorineural hearing within 72 hours is considered an otolaryngologic emergency. The evaluation and potential treatment options, including steroids, hyperbaric oxygen, and antiviral agents, are best performed in consultation with an otolaryngologist.

62.3. A 30-year-old woman presents with onset of a severe right posterior occipital headache and low-grade fever. Her physical examination reveals an area of erythema and swelling posterior to the right ear and a nonmobile tympanic membrane in that ear. What is the most appropriate next diagnostic step?
- **A.** ENT referral STAT to the operating room (OR)
- **B.** CT scan
- **C.** Lumbar puncture
- **D.** MRI with gadolinium
- **E.** No further diagnostic evaluation necessary

Answer: B. Clinical findings in acute mastoiditis may include fever, headache, otalgia, and posterior auricular erythema and tenderness. Although there are no specific diagnostic criteria, an initial step would be a CT scan to identify mastoid inflammation and possible bony erosion. MRI would be indicated if there is concern for intracranial extension.

62.4. All the following are implicated as risk factors in OM except:
- **A.** Children with cleft palate
- **B.** Daycare attendance
- **C.** Female gender
- **D.** Immunocompromised patient
- **E.** Parental smoking

Answer: C. Male gender appears to be a risk factor for middle ear disease, as well as daycare attendance, parental smoking, immunocompromised patients, and children with anatomic abnormalities such as cleft palate or Down syndrome. Breast-feeding appears to be protective.

62.5. An 18-month-old boy returns to the emergency department (ED) 4 days after being diagnosed with left OM. He was prescribed amoxicillin, 90 mg/kg/day, and the parents reported compliance. He has continued ear tugging, fever, and irritability. He is tolerating PO nutrition with no vomiting or diarrhea. Physical examination reveals an alert crying male with oral temperature 101.5°F, heart rate 136 beats/min, and respiratory rate 24 breaths/min. His physical examination is otherwise negative except for severe erythema of the left tympanic membrane, with obscure landmarks and loss of mobility. What is the most appropriate next step in this patient's management?

A. Admit for intravenous antibiotics.
B. Change therapy to an oral cephalosporin.
C. Draw blood cultures and continue current amoxicillin regimen.
D. Intramuscular ceftriaxone is given.
E. Lumbar puncture is performed.

Answer: D. Otitis media treatment failures at 3 days should receive intramuscular ceftriaxone. Continued use of a failing regimen would not be indicated. The child exhibits no signs or symptoms warranting a lumbar puncture and no immediate criteria for hospital admission.

62.6. A 13-year-old diabetic girl presents with left otalgia, left facial palsy, and fever. Physical examination reveals a left peripheral seventh nerve palsy, intense left otitis externa, diffuse tenderness of the pinna, and mild weakness of the left trapezius muscle. What is the most likely diagnosis?

A. Acute mastoiditis
B. Left temporal brain abscess resulting from left-sided otitis
C. Malignant otitis externa
D. Meningitis
E. Sigmoid sinus thrombosis

Answer: C. Necrotizing (malignant) otitis externa is a result of chronic otitis externa often seen in immunocompromised patients. The facial nerve is the cranial nerve usually affected, but the glossopharyngeal, vagal, accessory, abducens, and trigeminal nerves may also be involved. When otoscopic view permits, granulation tissue in the floor of the external canal at the bone–cartilage junction is characteristic. CT is the imaging technique of choice and is able to indicate bony erosions and abscess formation. Ciprofloxacin is the antibiotic of choice. All the other choices are recognized complications.

62.7. The management of anterior and posterior epistaxis is similar regarding which of the following?

A. Antibiotic requirements after packing
B. Duration of packing
C. Indications for hospitalization
D. Surveillance for secondary complications
E. Value of topical cauterization

Answer: C. Strong evidence for postpacking of antibiotics are lacking in both situations. Anterior packs are left in place for approximately 48 hours, whereas posterior packs may require 3 to 5 days. Patients requiring posterior nasal packs for epistaxis typically need hospitalization for supplemental oxygen and surveillance for pack expulsion with rebleeding, dysrhythmias, bradycardia, aspiration, and stroke.

62.8. Which of the following statements is true regarding inspiratory stridor?

A. It implies a palatal or uveal obstruction.
B. It is diagnostic of tracheal pathology.
C. It is typically accompanied by hoarseness.
D. It localizes a lesion at or above the vocal cord.
E. It may be seen with extremely severe asthma exacerbations.

Answer: D. Inspiratory respiratory distress (stridor) implies an extrathoracic flow obstruction. This may be laryngeal, epiglottis, or pharyngeal. Asthma, emphysema, and aspirated foreign bodies all have expiratory airflow limitations. Inspiratory stridor may or may not directly involve the larynx and may not be accompanied by hoarseness.