Otitis Externa and Malignant Otitis Externa—for the Hospitalist/Internist

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KEYWORDS

• Otitis externa • Otomycosis • External auditory canal • Osteomyelitis
• Malignant otitis externa

KEY POINTS

- Acute otitis externa (AOE) is an infection involving inflammation of the auricle or external auditory canal (EAC) lasting less than 6 weeks.
- AOE is most often treated with antibiotic drops with Pseudomonas aeruginosa coverage (ie, ofloxacin).
- Chronic otitis externa involves auricle or EAC inflammation lasting more than 3 months and requires routine debridement and topical antibiotic coverage. Culture-driven management is important.
- Malignant otitis externa (MOE) is an osteomyelitis of the temporal bone and a potentially deadly complication of otitis externa.
- Patients with uncontrolled diabetes or immunocompromising illness are most vulnerable for progressing to osteomyelitis. Management with extended intravenous antibiotics or antifungals is required.
- Otomycosis is a noninvasive fungal infection with white patches or spotted black spores visible in the EAC, often associated with recent topical antibiotic use.

INTRODUCTION

Otitis externa (OE) is a common ailment encountered and managed in the outpatient setting. However, these cases can become severe and result in complications that lead to neurologic deficits and require inpatient management. It is important to be able to diagnose and treat OE as the lifetime incidence is approximately 10% in the United States.[1] Ear pathology can be challenging to differentiate, but familiarizing oneself with the various disease processes, paired with a thorough history and physical examination, will make clinicians well equipped to tackle the outer ear.

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Abbreviations	
AOE	acute otitis externa
AOM	acute otitis media
CN	cranial nerve
COE	chronic otitis externa
CT	computed tomography
EAC	external auditory canal
IV	intravenous
MOE	malignant otitis externa
OE	otitis externa
TM	tympanic membrane

BACKGROUND

Anatomy

Auricle

The auricle, also known as the pinna, is a complex structure that consists of skin, cartilage, and intrinsic muscles. Fig. 1 depicts the major auricular landmarks. The helix is the cartilaginous outer rim of the ear that extends from the insertion onto the scalp to the ear lobe. The crus of helix is a continuation of the helix posterior-inferiorly. The antihelix is a Y-shaped cartilaginous ridge that bifurcates into the anterior and posterior crus of the antihelix.[2] The space between these cruses is termed the triangular fossa. The concha is a fossa bordered superiorly by the antihelix and anteriorly by the external auditory canal (EAC), also known as the external auditory meatus. The tragus is a protrusion of skin-covered cartilage that is just anterior to the EAC.[2]

The auricle receives sensory innervation from both cranial and spinal nerves. The mandibular division of the trigeminal nerve (V3), the facial nerve (VII), and the vagus nerve (X) are the cranial nerve (CN) components; the lesser occipital and greater auricular nerves, both from C2 and C3, are the spinal nerve components.[3] The facial nerve also supplies motor innervation to the auricle.[3]

The auricle receives blood supply via the superficial temporal, posterior auricular, and occipital artery, all of which are branches of the external carotid.[3,4]

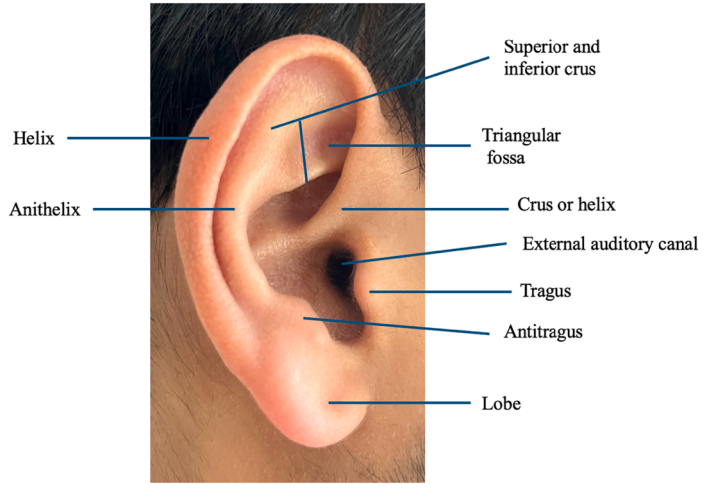

Fig. 1. Anatomic auricular landmarks are labeled in the image.

External auditory canal

The EAC is typically 2.5 cm in length and 8 mm in diameter in adults. It has a sigmoid or "S" shape, which is the reason pulling the auricle posteriorly and superiorly offers a better view of the entire EAC and tympanic membrane (TM) during otoscopic examination. The lateral one-third of the EAC is cartilaginous and the medial two-thirds is bone.[5]

Sensory innervation to the EAC is supplied by the nervus intermedius (a branch of CN VII), the auriculotemporal nerve (CN V3), and the auricular branch of the vagus nerve (CN X). The EAC lacks motor innervation and the blood supply originates from the external carotid and maxillary artery, both branches of the external carotid artery.[6]

Physiology

The auricle funnels sound waves into the EAC, which improves the delivery of sound to the TM. The pinna and EAC are particularly helpful in amplifying mid-to-high-frequency sound. They are estimated to contribute a gain of approximately 10 dB in sound. The skin of the EAC is rich in ceruminous and sebaceous glands. These glands secrete a waxy substance that has a slightly acidic pH that inhibits bacterial growth.[7]

Otitis externa (OE) is defined as inflammation of the external ear canal that can be infectious or noninfectious in origin. OE is classified as acute (lasting <6 weeks) or chronic (lasting >3 months).

ACUTE OTITIS EXTERNA

Pathogenesis/Diagnosis

Acute otitis externa (AOE) is cellulitis of the ear canal skin and dermis with diffuse inflammation of the canal. Edema can extend to the TM or to the auricle. The diagnosis for AOE is clinical. Patients must meet these 3 criteria for diagnosis:

1. Signs of ear canal inflammation, which includes tenderness of the external ear or edema and erythema of the EAC with or without otorrhea
2. Symptoms of canal inflammation, including otalgia, itching, or fullness
3. Edema occurs rapidly over the course of typically 48 hours and lasts up to 6 weeks.[1,8,9]

Microbiology

Ninety-eight percent of cases of AOE are bacterial in origin, and the remaining 2% are secondary to fungi. The most common pathogens are *Pseudomonas aeruginosa* (38%), *Staphylococcus epidermis* (9.7%), and *Staphylococcus aureus* (7.8%).[4,8,10] Fungal involvement is more common with chronic otitis externa (COE), which is discussed later in this article.

Risk Factors

AOE is more common in warmer climates or areas of increased humidity. Increased water exposure from swimming is a risk factor and OE is often referred to as "swimmer's ear." Water quality is associated with the risk of acute otitis media (AOM), but even pools or hot tubs that are compliant with water quality standards can pose a risk of infection. Skin flora itself may be the source of infection in some patients. Some patients are more susceptible to AOE based on genetics and having stenotic EACs. Having lesions obstructing the EAC such as exostosis (broad bony growths along the EAC) increases a patient's risk for developing recurrent AOE.[11] Patients in an immunocompromised state secondary to ailments such as human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome, chemotherapy, or diabetes are at an increased risk for developing more severe infections.[4]

History

As with all head and neck disease processes, a thorough history and physical examination are essential. Clinicians should understand the duration of disease, frequency, triggers, and prior treatments attempted. Presenting symptoms typically include otalgia or ear pain (70%), itching (60%), aural fullness (22%), or hearing loss (32%). Patients may have referred pain, resulting in jaw pain with chewing. Some patients can also develop thick otorrhea or ear drainage.[1,4]

Physical Examination

Patients may have developed regional cellulitis with edema of the pinna, erythema, and tenderness; this is termed complicated OE. For AOE, pulling the pinna or tragus can reproduce the patient's ear pain. Patients can have regional lymphadenitis as well. On otoscopy, patients will have diffuse ear canal edema and/or erythema. The TM can also become inflamed, thereby appearing edematous and erythematous. Otorrhea may be present. Irrigations or debridement of the ear canal may be required to evacuate the canal of cerumen or debris, allowing for a proper evaluation of the TM (methods to do so are discussed in the "Management" section of AOE later).

AOE is often mistaken with AOM, particularly when the inflammation from AOE travels to the TM. Thus, it is important to evaluate for ear canal edema, which supports a diagnosis of AOE. Fluid behind the TM supports a diagnosis of AOM.

Workup

Laboratory work and imaging are not recommended for uncomplicated AOM. If patients develop pinna fluctuance or pain out of proportion to examination findings, then laboratories or imaging may be indicated. Starting with a complete blood count and computed tomography (CT) temporal bone with contrast is reasonable in those settings. Culture of the ear canal is recommended to ensure appropriate microbial coverage, particularly in recurrent disease. Audiograms are not initially indicated for this disease process.[1,8]

Management

Antimicrobial therapy

Topical antimicrobials are the treatment of choice for AOE that is limited to the EAC. Topical therapy typically elicits symptom improvement in 24 to 48 hours and resolves AOE within 7 to 10 days in 65% to 90% of cases.[1,12] Topical quinolones, such as ofloxacin or ciprofloxacin, are the antibiotic of choice because of their *P aeruginosa* coverage.[1] Other topical antibiotic families to consider include aminoglycoside and polymyxin B.[1] A low-pH antiseptic such as acetic acid can also be considered. In patients with a TM perforation, aminoglycosides should be avoided due to ototoxic and vestibulotoxic effects.[1]

Drops should be applied with the patient lying down and the affected ear upward. The pinna may be gently pulled posteriorly to release trapped air, or patients can perform tragal pumping to aid in getting the drops into the ear canal. The patient should remain with the affected ear pointing up for about 3 to 5 minutes. The canal should be left open to dry to avoid trapping moisture and infected debris.

Adverse reactions to antibiotic drops are relatively uncommon (2% of patients) and include rash, discomfort, otalgia, dizziness, vertigo, fungal overgrowth, and reduced hearing.[1] Oral antibiotics are given in 20% to 40% of patients with AOE.[1] However, oral antibiotics are usually ineffective against *P aeruginosa* and *S aureus* in the ear canal, may have adverse effects, and may breed resistance.[1] If there is extension from the EAC to the auricle or periauricular region, then systemic antibiotic therapy should

be offered. If the patient does not have clinical improvement in 48 to 72 hours, then the clinician should reassess the patient to exclude other ear pathology such as underlying skin conditions or growths (discussed further in "Otitis Externa (noninfectious)" section).[13] See **Table 1** for summary of key points regarding OE and other pathology reviewed in this article.

Aural toilet

Clinicians should offer ear debridement at the time of patient evaluation. One method of debridement involves tilting the patient's head over a sink or basin and irrigating with normal saline, also referred to as an aural toilet. Another option is for clinicians to use a working otoscope head, which allows for suction/debridement and visualization. If clinicians do not have adequate equipment to safely clean the ear canal, then patient should be referred to an otolaryngology clinic for further management.

Wicks are important when edema of the ear canal prevents topical medication from entering the cavity. While no trials have proven the efficacy of wicks, they are consistently used given the theoretic benefit in medication delivery and eliminating a stenotic EAC.[1] Ideally, the wick should be made of cellulose because it expands when exposed to moisture. Cotton should not be used as a substitute because of the risk of fragmentation causing further occlusion of the EAC. A follow-up appointment should be made in approximately 5 to 7 days to remove the wick in the clinic.[14] Wicks will occasionally fall out on their own once the EAC edema begins to resolve.

Prevention

Strategies for preventing AOE target limiting water exposure and moisture retention to the EAC. Recommendations include ceasing Q-tip use to prevent obstructing the EAC with cerumen, avoiding frequent irrigations (frequent cleaning will increase the local pH and promote bacterial growth), using earplugs with swimming, applying acidifying ear drops before/after exposure to water, and drying the ear with a hair dryer after water exposure.[1]

Analgesia

Oral agents such as acetaminophen or nonsteroidal anti-inflammatory drugs typically provide relief. In select patients, a low-dose narcotic medication may be required.[15]

Admission Versus Outpatient

Patients with AOE typically can be managed on an outpatient basis. However, AOE can progress to malignant OE (discussed later in this article), which is an indication for admission for intravenous (IV) antibiotics.

CHRONIC OTITIS EXTERNA

Pathogenesis/Diagnosis

COE is defined as signs and symptoms of OE lasting more than 3 months.[4]

Microbiology

COE is caused by similar bacterial culprits as AOE[16]—*P aeruginosa*, *S epidermis*, *S aureus*.[4,8,10] Fungal involvement is more common with COE (*Aspergillus* and *Candida*) and is discussed in the next section (Otomycosis).[17,18]

Risk Factors

These patients often have predisposing factors including prolonged moisture exposure; this can be from living in humid environments or narrow ear canals that prevent

Table 1
Summary of key points regarding otitis externa and other pathology reviewed in this article

	Common Pathogens (Prevalence)	Duration	Key History	Examination Findings	Treatment
Acute bacterial OE	• *P aeruginosa* (20%–60%) • *S aureus* (10%–70%)	• 48 h to 6 wk	• Increased water exposure	• Tender, erythematous, edematous pinna • Diffuse EAC edema and erythema • Possible TM erythema or edema (without fluid behind TM)	• Debridement • Topical antibiotics (eg, ofloxacin) • Cellulose-based wicks if stenotic EAC • Oral antibiotics only if symptoms extend to auricle or periauricular region
Chronic bacterial OE	• Same as above	• >3 mo	• Stenotic EAC • Eczema • EAC trauma from cleaning or foreign bodies • Hearing aids	• Pinna and/or EAC edema and erythema • Skin changes • Underlying mass in EAC	• Debridement • Topical antibiotics plus steroids (ie, ofloxacin + 0.1% dexamethasone drops) • 1:1: acetic acid to sterile water irrigations
Otomycosis	• *Aspergillus* (60%–90%) • *Candida* (10%–40%)	• 1–3 wk	• Recent otologic topical antibiotics • Diabetes • Immunosuppression	• Thick otorrhea • Pruritic • White or black debris with white sprouting hyphae	• Debridement • Topical antifungals (clotrimazole, miconazole, bifonazole, ciclopiroxolamine, and tolnaftate) • Systemic antifungals reserved for refractory disease or complications
MOE	• *P aeruginosa* (90%)	• <1 wk	• Diabetes • Immunosuppression	• Granulation tissue at bony cartilaginous junction • Exquisitely tender, out of proportion to examination • Possible CN VII or VIII deficits	• Typically, IV antibiotics for 6–7 wk • Recommend infectious disease consult • Possible myringotomy tube placement

adequate ventilation. Other inciting factors include dermatologic conditions such as eczema, trauma to the ear canal from aggressive ear cleaning, insertion of foreign bodies, or use of hearing aids.[13,19]

History

The evaluation of COE is similar to that of AOE. Give attention to prior treatments trialed including antibiotic courses, ear cleaning regimens, and surgeries.

Physical Examination

Evaluate the auricle and EAC for dermatologic abnormalities such as skin thickening, scaling, and discoloration that may point to a dermatologic etiology. If able to safely clean the EAC with irrigation or a working head otoscope, this would offer a better examination of the EAC skin. Particularly in COE, there may be an underlying mass, anatomic anomalies, or canal cholesteatoma.

Workup

Culturing the substrate is highly recommended to appropriately treat the disease. Imaging is not indicated. Audiograms are generally not indicated for this disease process.[1]

Management

The underlying cause of COE should be addressed. Dermatologic or autoimmune conditions can compromise the overlying skin and alter the bacterial flora of the EAC, thus increasing the risk for infection.[20] The same techniques for keeping the ear dry as discussed with AOE should be employed. Patients should also avoid inducing additional microtrauma to the EAC by cleaning with cotton-tip applicators or similar objects at home.

These patients can benefit from adding a steroidal component to their topical antibiotics. This will help reduce edema in the EAC, allowing for more space for drainage and ventilation. Dexamethasone 0.1% ophthalmic drops are a frequently used solution.[13,21] A combined antibiotic and steroid drop, which contains both ciprofloxacin and dexamethasone, is available. However, this combination drop is often more expensive for patients, in which case the steroid and antibiotic drops can be ordered separately. Patients can also apply 0.1% betamethasone valerate lotion or 0.1% triamcinolone cream with their pinky finger to the opening of the EAC; advise patients to tilt their head so the affected ear points to the roof, and the cream will migrate medially.[1] These patients will need frequent clinic visits to perform routine evaluation and cleaning. Patients may initially be seen weekly, and then visits are spaced out as clinical improvement is noted. If patients are being seen weekly and have reaccumulation of debris by the time of the next clinic visit, then clinicians should consider advising these patients to irrigate their ear with 1:1 acetic acid to sterile water to acidify the EAC.[22]

COE is challenging to manage, and referral to an otolaryngologist is warranted if patients continue to have symptoms.

OTOMYCOSIS

Definition/Pathogenesis

Otomycosis is a fungal infection of the EAC.[23] Fungi can grow atop purulence or mucus in the ear canal without inducing symptoms. However, if the fungi blooms, this can result in a local inflammatory response, resulting in aural fullness and otalgia.[4] In most cases, otomycosis is not invasive and can be treated with topical

antifungal medications and ear canal hygiene. However, in rare circumstances, particularly in individuals with weakened immune systems, otomycosis can become invasive, spreading to deeper structures like the eardrum or the temporal bone.

Microbiology

Aspergillus (60%–90%) and *Candida* (10%–40%) are the most common pathogens.[17,18]

Risk Factors

Otomycosis is more common in humid regions, patients who underwent a prolonged course of topical antibiotics, immunocompromised patients, or those with diabetes.[17,24]

History

A similar history as that for AOE should be obtained. A history of recent otologic topical antibiotic use is common. These patients will often have thick otorrhea that can be black, gray, green, yellow, or white in color.[17] They also often experience persistent pruritus and pain.

Physical Examination

Candidal OE typically has white debris with white, sprouting hyphae. Aspergillus often has a "wet newspaper" appearance with a white base and overlying black debris.[1] If patients have persistent symptoms following appropriate topical therapy for bacterial OE, then a fungal infection should be considered.[24]

Management

First-line therapy is debridement and topical antifungal drops. Clotrimazole, miconazole, bifonazole, ciclopiroxolamine, and tolnaftate are safe choices for the treatment of otomycosis.[1,17] One percent clotrimazole cream once daily for 7 to 10 days is a commonly prescribed regimen.[25] Systemic antifungal therapies, which are typically the triazole antifungal family, are rarely required for otomycosis; however, oral or IV therapy can be considered for patients with refractory disease to topical therapy, osteomyelitis, or intracranial complications.[1]

MALIGNANT OTITIS EXTERNA

Definition/Pathogenesis

Malignant otitis externa (MOE) is osteomyelitis of the temporal bone.[26] It is a misnomer given that this disease process is not cancerous. Another common name is necrotizing OE. It is a rare disease process with an incidence of 0.2 per 100,000 people.[27] It is an aggressive AOE infection that has extended into the surrounding skull base. Thus, MOE most frequently occurs in patients who are in an immunocompromised state.

Microbiology

P aeruginosa is the most common culprit, isolated in 90% of cases.[1]

Risk Factors

Common comorbidities include uncontrolled diabetes, autoimmune disease, or ongoing chemo/immunotherapy.[28,29]

History

Similar to AOE, patients will experience otalgia, otorrhea, aural fullness, and hearing loss. This disease process can extend into the middle ear, inner ear, intracranially, temporomandibular joint, masticator space, and parotid gland.[26] Thus, patients can develop hearing loss, facial nerve paralysis, vertiginous episodes, and jaw pain.[30]

Physical Examination

The characteristic examination finding is granulation tissue at the bony cartilaginous junction located at the lateral third of the EAC, however not all patients will have this finding.[31] Patients will have exquisite tenderness on examination. Patients may have auricular changes as well. During a patient's inpatient stay, it is important to evaluate the ear canal daily to monitor for improvement.[26] Perform a thorough CN examination, with special emphasis on evaluating CN VII and VIII daily.

Workup

Patients will have elevated ESR and CRP levels. These laboratories can be used to trend response to treatment.[32] Blood glucose levels should be checked and hemoglobin A1c (A1C) should be ordered if blood glucose levels are elevated.[30]

Imaging is essential for the diagnosis and should include a CT temporal bone without contrast to evaluate for bony erosion and MRI brain with and without contrast with fine cuts through the temporal bone to assess for enhancement as well as surrounding soft tissue.[33] An audiogram does not need to be ordered in acute cases but should be ordered on first clinic follow-up.

Management

Antimicrobial

First-line therapy is systemic IV antibiotics that cover pseudomonal and staphylococcal infection, including *methicillin-resistant Staphylococcus*. Common courses include 6 to 7 weeks of ceftazidime or meropenem plus vancomycin.[1,30] Due to their high oral bioavailability and bone penetration, oral quinolones can be considered for cases of MOE that have pseudomonal isolate.[34] Infectious disease should be consulted to assist with dosing and managing antibiotics outpatient.[26]

The role of topical therapy is unclear. A short course of topical antibiotics may be helpful for patients with a myringotomy and neurologic findings.[1,26] To improve administration of topical therapies, a wick can be placed.

Surgery

The otolaryngology team should be consulted to evaluate the ear and review imaging. If there is middle ear disease, the otolaryngology team may elect to perform a myringotomy and tube placement, a procedure that can be performed in the clinic or in the operating room. This will aid in source control if the infection has spread to the middle ear. For more aggressive disease that is refractory to medical therapy, defined as antibiotic treatment for 6 weeks, debridement of necrotic bone may be required.[32] In these rare cases, a mastoidectomy would be required. This is a procedure performed by the otolaryngologist in the operating room, where the mastoid bone is opened in order to debride diseased bone and ventilate the ear. The purpose is to remove necrotic and infected bone, thereby reducing infected tissue load and improving delivery of IV antibiotic therapy to vascularized temporal bone. It is also a good source of tissue for cultures.

If patients do not experience clinical improvement in 1 to 2 weeks, then other diagnoses should be considered, including cancer of the EAC.

OTITIS EXTERNA (NONINFECTIOUS)

There are several noninfectious illnesses of the external ear that can mimic AOE. A thorough history with specific attention to duration of symptoms and systemic signs can help differentiate these pathologies.

Atopic Dermatitis (Eczema)

Atopic dermatitis is a common manifestation of the "atopic triad," which includes seasonal allergies and asthma. Patients with atopic dermatitis of the ear often endorse chronic pruritus in other areas of the body, which may have been ongoing since childhood. The inflamed tissue may weep a serous solution, which patients may notice on their pillows in the morning. Patients can present with symptoms of auricular dermatitis at any age. The skin of the auricle and EAC will vary depending on the severity of the disease. In milder cases, the soft tissue will be erythematous, edematous, and have xerotic scaling. In more advanced cases, the skin will appear hyperpigmented and lichenified. Patients can develop painful fissures at the insertion site of the tragus.

Treatment includes emollients and topical corticosteroids. 0.1% triamcinolone bid or 0.01% fluocinolone acetonide oil to the affected area for 2 weeks are common treatments. Afterward, patients can continue using the cream as needed.[35,36] To avoid a superimposed infection, patients should practice AOE preventative technique such as drying the ear after swimming and avoiding inner ear headphones. Mupirocin ointment can be used for superimposed impetigo, and physicians can add systemic therapy as indicated.[35]

Seborrheic Dermatitis

Seborrheic dermatitis is the inflammation of sebaceous areas of the body, including the ears, scalp, face, chest, and back. It presents as a greasy, yellow scaling with surrounding erythema. Seborrheic dermatitis is due to an overabundance of *Malassezia* yeast. It occurs more frequently during infancy or adulthood but can happen at any age. Patients more at risk are those with HIV, psoriasis, Parkinson's disease, and mental health conditions. Triggers include stress and hormonal changes.[37]

Treatment includes topical antifungals, such as ketoconazole cream and topical or systemic anti-inflammatory medication.[38,39] Over-the-counter dandruff shampoos containing selenium sulfide or zinc pyrithione are useful adjuncts. Typically, it is recommended to allow these shampoos to remain on the scalp for several minutes before washing off to maximize utility.[35]

Contact Dermatitis

Irritant and allergic contact dermatitis are 2 forms of contact dermatitis of the EAC. Irritant contact dermatitis is direct damage to the soft tissue of the EAC secondary to chemical exposure, which is less common than allergic contact dermatitis. Allergic contact dermatitis occurs more frequently in patients with known allergic reactions to substrates. Common allergens include metals (commonly nickel), detergents, soap, rubbers, and chemicals used to prepare plastic. Common culprits include earrings and hearing aids.[37] Contact dermatitis will present with inflammatory signs at the site of insult. Patients may experience otalgia and pruritus. It typically does not have associated otorrhea. Once the irritant is removed, symptoms tend to gradually resolve.[37,40]

Management entails removing the irritant and applying a topical steroid or topical anti-inflammatory. Triamcinolone cream and calcineurin inhibitors (0.1% tacrolimus

ointment or 1% pimecrolimus cream) are frequently prescribed.[37,41,42] Oral antihistamines can alleviate itching in cases of allergic dermatitis. Oral steroid therapy may be indicated in more severe cases.

Other Pathology

There are several other disease processes that can mimic AOE. Furunculosis is the presence of infected hair follicles, often secondary to infection with skin flora, typically *S aureus*. On examination, patients typically have oily skin and will have small pustules at hair follicles. Treatment is systemic antibiotics.

Cholesteatoma, which is a cyst filled with keratinized debris, is often located in the middle ear. However, it can also develop in the EAC (a canal cholesteatoma). Patients typically have otalgia without other otologic symptoms. The EAC will have exposed bone as well as soft and irregular white debris. The TM may become involved, and the cholesteatoma will erode through the margin of the eardrum, resulting in a perforation.

Finally, herpes zoster oticus (Ramsay Hunt syndrome) is a reactivation of the varicella-zoster virus, which results in inflammation of the geniculate ganglion of the facial nerve. Patients will experience symptoms including ipsilateral otalgia, facial paresis, taste changes, and decreased lacrimation. On examination, there may be vesicles in the EAC or on the auricle. Treatment includes systemic antiviral therapy and systemic steroids.[43]

SUMMARY

OE has varying degrees of chronicity and severity. OE can be infectious or noninfectious in etiology. AOE is characterized by less than 6 weeks of otalgia and otorrhea; the vast majority of AOE is bacterial (infectious) in origin. COE lasts more than 3 months. Treatment of both is similar, which includes topical antibiotics and appropriate aural hygiene. COE often requires routine debridement by an otolaryngologist. MOE is osteomyelitis of the temporal bone. Patients will have excruciating pain on examination and classic granulation tissue at the cartilaginous–bony junction. These patients are often diabetic or otherwise immunocompromised. Radiographic findings include temporal bone erosion along with T1 osseous enhancement on MRI. Treatment is prolonged IV antibiotics and source control.

CLINICS CARE POINTS

- AOE is an infection of the external ear, which presents as inflammation and pain of the auricle or EAC, lasting less than 6 weeks. Treatment is typically antibiotic drops with pseudomonal coverage (ie, ofloxacin).
- COE is a prolonged episode of OE lasting more than 3 months. Management includes routine debridement, a combination of antibiotic and steroid drops, possible wick placement in the EAC, and treating the underlying pathology (eg, eczema and trauma).
- MOE or osteomyelitis of the temporal bone typically affects patients with uncontrolled diabetes or immunocompromising illness. Treatment is managing the patients' underlying immunologic conditions, IV antibiotics, and can include surgical debridement.
- Otomycosis is generally a noninvasive fungal infection of the EAC. Prior topical antibiotic therapy is a risk factor. The EAC has debris that ranges from patches of white to spotted black spores. Otomycosis can very rarely be invasive with immunocompromise being the greatest risk factor for this.

DISCLOSURE

None.

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