



External Otitis

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External otitis or otitis externa (OE) is a very common problem. This chapter will cover the definition of OE, the pathophysiology of OE and then go through how otitis externa presents clinically and finally address several treatment options.

Definition

Otitis externa is an infection with inflammation of the external auditory canal. The diagnosis and treatment of this painful condition is relatively straightforward. In North America it felt that 98% of OE is bacterial. The incidence of OE is 1:100-1:250 people/year so it can be expected that 10% of the general population will get OE at least once in their lifetime. There is regional and seasonal variation with humid climates having a higher incidence as well as during summer when there is much use of swimming pools.

External otitis can be either a primary infection of the ear canal or can be a secondary infection that originates in the middle ear and then through a perforation or tube extends to the outer ear. This chapter will address only primary external otitis.

Pathophysiology

The outer ear or external ear is the area between the ear drum and the pinna so it is composed primarily of the ear canal. The purpose of the ear canal is to conduct sound from the environment around us to the middle ear which eventually transmits amplified sound to the brain. The outer ear must get the sound close to the brain for good transmission without contaminating the brain from the environment. It is very precisely designed. The ear canal is layered with skin which is the armor of the ear canal that prevents invasion of normal skin bacteria. The skin, however, is prone to maceration if exposed to high humidity which combined with the fact that humans are 90% salt water would be very poor defense if the body had not developed cerumen glands which make a waxy, water-repellent substance that coats the ear canal and thereby is protective and assists in humidity control of the ear canal. In addition, cerumen is acidic which is anti-bacterial for most waterborne bacteria, particularly *Pseudomonas aeruginosa*.

An infection of the ear canal results when the specifically designed homeostasis of the ear canal becomes unbalanced. This can be from water exposure, changes in humidity or elimination of cerumen. It can also be from direct injury to the skin armor such as with traumatic self instrumentation of the ear canal. There are also

specific disease states that render a person more prone to the development of OE such as psoriasis, diabetes or other causes of immunosuppression.

Clinical presentation

When a patient presents with a possible infection of the ear, a history is elicited with respect to prior history of OE, prior placement of pressure equalizing tubes, or known eardrum perforation. Symptoms of the present episode are then reviewed including the time of onset, the duration of the symptoms, the severity of the symptoms and whether the patient's symptoms are specific to the ear only or are becoming more generalized. It is also important to know if the patient has had any fever or if there is a perceived hearing loss.

On physical examination, the patient is first assessed for pain when moving the pinna, then ear canal patency is determined and the ear drum is visualized if possible. In the ear canal the examiner should look for edema of the ear canal, debris within the canal, purulence, or erythema. The erythema may extend outward onto the pinna as an early sign of cellulitis. The tympanic membrane is then assessed if possible. Note that all four quadrants of the eardrum must be seen to rule out a perforation or retained foreign body. The quality of the drum whether stretched or desquamating is noted as well as the character of the canal debris. Primary external otitis is typically chunky and desquamating in a very swollen ear canal while secondary OE through a perforation or tube is typically creamy purulence without associated canal edema. Mucopurulent or even serosanguinous drainage can be noted.

There are known predisposing risk factors for OE, such as getting water stuck in your ear canal after swimming, humid weather that increases maceration, and known trauma, as from self-cleaning of the ear or having the ear be a little itchy and the resultant use of a bob pen or some other instrument in order to scratch that itch. Having dermatitis within the ear canal is another type of diagnosis that would make a person prone to developing OE. Anyone who wears hearing aids keeps the ear canal extra humid, thus increasing the risk of maceration. And of course anyone with immunosuppression is at increased risk for OE. If you have a child with OE and they do not respond to the typical treatment one should suspect diabetes mellitus as it is quite possible that this maybe their presenting symptoms and the diabetes can be diagnosed just from an episode of OE.

Diagnosis

OE actually develops quite quickly over a 48 hour period although the person may not present for care immediately. The definition of OE is of a sudden onset of symptoms within 3 weeks of presentation. In the early phase of the development of OE, the patient complains of pain, pruritus or itchiness. On physical exam there is swelling of the ear canal and erythema of the ear drum upon physical examination. Later on severe pain develops and much more significant erythema and edema are seen on examination. Because of the canal swelling, the patient will also complain of aural fullness with hearing loss and on physical examination there is more squamous cell desquamation discharge occluding the canal. Finally, the patient may progress to infection spreading through the fissures of Santorini

in the ear canal and develop cellulites of the pinna and adjacent skin and eventually regional lymphadenopathy.

When the ear canal edema progresses to a point of occlusion treatment as well as culture and debridement becomes difficult to perform as well as being significantly painful for the patient to undergo.

Treatment

Treatment of OE can be divided into **three patient types**. The **first** is a patient with **mild OE**. The patient has ear pain and the canal itches but it hurts to move ear and the eardrum is visualized with no perforation seen. With no perforation present, several drops of hydrogen peroxide may be useful to remove the debris from the ear canal in the practitioner's office. Treatment is then **analgesics** either **oral or topical for pain relief**, as well as the use of either **acetic acid or vinegar and/or alcohol drops or ototopical antibiotics**. In the **second type of patient**, a **moderate OE**, the symptoms are perhaps more severe and on physical examination the eardrum is only partially seen but the canal can be appreciated to still be patent, so that drops can still be transverse the ear canal. In the moderate OE infection however, the drops used for topical pain relief are too viscous to move down the ear canal from where the pain is coming so **oral analgesics** will need to be prescribed. Also because of the swelling, a thinner treatment drop is required to traverse the canal so you will need an **aqueous drop in your ototopical antibiotics** and because you can not see the entire eardrum to be sure there is no perforation only a non-ototoxic form of eardrops should be considered. **Systemic antibiotics can be useful only** if the infection extends to the surrounding skin as **cellulitis** or in a patient who is **immunocompromised**. This is because the ear canal has very poor blood supply for the delivery of systemic antibiotics during its very acute inflammatory stage and only topical antibiotics will be effective.

The **third** type of OE is the **severe type** where the ear canal is found on physical examination to be swollen shut or very nearly so such that no eardrum is visualized. Typically in order to effectively debride and clean these severe ears a referral to the otolaryngologist is required – plus you don't want that child to be tortured in your office and then to have to take care of them ongoing basis in a primary care office, therefore it is often better to let the specialist deal with that severe of an infection. In this situation it may be necessary **to place an expandable sponge wick into the ear canal**. This allows the ototopical drop to penetrate the entire canal for treatment of the infection. Severe otitis is so very painful that a **stronger analgesic** such as hydrocodone may be necessary to obtain some pain relief. It should be emphasized that the **antibiotic chosen needs to be both, thin or aqueous, as well as be non-ototoxic**. **Systemic antibiotics are reserved for extension of the infection** into the pinna or onto the face as cellulitis. Remember that severe or difficult infections could be the initial presentation of a child who is immunocompromised.

As further discussion of the treatment of OE, the microbiology of external otitis should be considered. When cultured, the prevailing organism is *Pseudomonas aeruginosa* with *Staphylococcus aureus* coming in as the second most common causative organism. There are no oral antibiotics approved for use in children that

cover *Pseudomonas aeruginosa*. And as noted above, there is very poor penetration of a systemic antibiotic into the ear canal skin because of the poor vascularity of the area. Otological treatment of the bacterial infection is considered a much better choice for both the microbiological coverage as well as the fact that an otological drop has a very high concentration. The high concentration of the antibiotic drop decreases greatly the development of any antibiotic resistance to the medication. Once the microbiology is known another consideration when choosing a drop is its potential ototoxicity. Anytime the tympanic membrane cannot be determined to be intact by visualizing the entire surface a perforation is potentially present and no drop containing an ototoxic substance such as the antibiotic class of aminoglycosides or the preservative of ethylene glycol would be considered safe. Final consideration for the treatment of OE must include compliance of the patient and the effectiveness of the drop penetration. When the canal is swollen we need a thin aqueous solution, as any suspension may be too thick to penetrate the ear canal. We also want to be sure that the drops will be well tolerated. If the solution is very acidic, it will sting and burn and the patient will not allow further use of the drops. The drop chosen therefore needs to have a balanced pH. Also any time we consider a treatment we want to look at the cost effectiveness of the treatment. It has been shown that dexamethasone within the drops may reduce the pain by a few hours over drops that do not contain steroids. However drops that contain less potent steroids don't show any difference in the amount of pain relief when compared to drops without steroid. Currently the cost for a quinolone drop with dexamethasone is much more than without steroid in the US. Recently the otological drop ofloxacin has become generic so the cost will typically rival the more viscous aminoglycoside combination drop so may be the best first line choice.

Conclusion

In conclusion, external otitis such as from swimming or from a primarily humid environment can be treated based on severity. If the infection is mild, debridement of the ear canal and the use of an acetic acid drop to finish cleaning may be all that is required for resolution of infection. If the infection is more moderately severe with a swollen canal, an aqueous antibiotic drop and appropriate pain control is indicated. Finally, if the OE is severe, an appropriate drop for Pseudomonas and staphylococcal infection coverage in an aqueous non-ototoxic drop is chosen. Wick placement may be needed as well as more significant narcotic pain control.

Recommended readings

1. Manolidis S et al: Comparative efficacy of aminoglycoside versus fluoroquinolone topical antibiotic drops. *Otolaryngol Head Neck Surg* 2004;130:S83 [PMID: 15054366].
2. Roland PS et al: Consensus panel on role of potentially ototoxic antibiotics for topical middle ear use: introduction, methodology and recommendations. *Otolaryngol Head Neck Surg* 2004;130:S51 [PMID: 15543836].