Complicated Otitis Externa

Eric Rosenberger, MS4
Faculty Advisor: Tomoko Makishima. MD, PhD
The University of Texas Medical Branch
Department of Otolaryngology
Grand Rounds Presentation
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Auricle:
1. Begins development during the 6th week of gestation
2. Derived from mesoderm of the 1st and 2nd branchial arches, forming 6 His hillocks
3. Reaches adult shape by the 20th week of gestation, but the adult size is not reached until age 9 years
Embryology cont.

EAC:
1. Begins to form during the 8th week of gestation, when the surface ectoderm of the 1st pharyngeal groove thickens and grows toward the middle ear. This core of tissue begins to resorb by the 21 weeks' gestation to form a channel that is complete by 28 weeks' gestation. The canal reaches adult size by age 9 years and ossifies completely by age 3 years.
Introduction to Otitis Externa

• Most ear canal infections are due to excessive moisture providing suitable conditions for bacterial overgrowth
• Acute otitis externa occurs in 4 of every 1000 people per year
• Otitis externa is defined as chronic when the duration of the infection exceeds 4 weeks or when more than 4 episodes occur in 1 year
In the patient history:

- 1 to 2 days of progressive ear pain
- Exposure to water
- Itching
- Purulent discharge
- Conductive hearing loss
- Feeling of fullness or pressure
On physical exam:

• **sine qua non of otitis externa** = *pain on gentle traction of the external ear*
• Periauricular adenitis
• Speculum examination reveals erythema, edema of the epithelium, and accumulation of moist debris in the canal
• Spores and hyphae may be seen in the external canal, if etiology is fungal
• Eczema of the pinna may be present
• (CN) involvement is **not** associated with simple otitis externa.
Eczema

- External clue to OE
Speculum findings:

- the canal may be so swollen that a view into the ear is impossible
- In swimmers, divers and surfers, chronic water exposure can lead to the growth of bony swellings in the canal known as *exostoses*. These can interfere with the drainage of wax and predispose to infection.
Differential diagnoses:

- Otitis media
- Ramsay Hunt syndrome
- Furuncle
- Skull base osteomyelitis
- Preauricular cyst and fistula
- Lacerations
- Atopic dermatitis
- Cerumen impaction
- Exostosis and osteoma
- Foreign body
- Acute (bullous) and chronic (granular) myringitis
Differential diagnoses:

- **Ramsay Hunt syndrome:**
  - This condition, more accurately known as herpes zoster oticus, is caused by varicella-zoster viral infection. Ramsay Hunt syndrome is characterized by facial nerve paralysis and sensorineural hearing loss, with bullous myringitis and a vesicular eruption of the concha of the pinna and the EAC. A painful otitis externa may be present as well. Treatment includes use of an antiviral agent (eg, valacyclovir) and systemic steroids. The role of facial nerve decompression remains controversial.

- **Furuncle:**
  - Staphylococcal infection of a hair follicle is the usual cause of a furuncle. This infection occurs in the lateral cartilaginous hair-bearing portion of the EAC. On otoscopic examination, a furuncle is a localized infection, which may develop into an abscess, rather than the diffuse inflammatory process characteristic of otitis externa.
Differential diagnoses:

- **Skull base osteomyelitis:**
  - This serious infection, also known as malignant otitis externa, occurs most often in patients who are diabetic or immunocompromised. The pathogenic bacteria are usually *Pseudomonas aeruginosa*. Other predisposing conditions include arteriosclerosis, immunosuppression, chemotherapy, steroid use, and other immunodeficient states. The diagnosis is strongly suggested by a history of diabetes mellitus, severe otalgia, cranial neuropathies, and characteristic EAC findings. The EAC may be filled with friable granulation tissue, which is primarily found inferiorly. Because this presentation may be identical to that of a soft tissue malignancy, prudence dictates a tissue biopsy, even if a history of diabetes mellitus is present. Bare bone of the EAC floor may be exposed; small bony sequestra may be observed as well. CT scanning demonstrates bone erosion, and gallium scanning can be performed at points throughout treatment to monitor resolution. Treatment consists of administration of an antipseudomonal IV antibiotic such as ceftazidime (in some cases) or oral ciprofloxacin (in less dramatic cases). Extended treatment for at least 6 weeks is most appropriate. Hyperbaric oxygen therapy may also be effective. Surgical debridement is reserved for granulation tissue and bony sequestra.
Differential diagnoses:

- **Preauricular cyst and fistula:**
  - Abnormal development of the first and second branchial arch may result in the formation of a preauricular cyst or fistula, which may manifest as persistent discharge or recurrent infection. A draining sinus may be present anterior to the tragus; when infected, the cyst distends with pus and the overlying skin is erythematous. These lesions are managed by complete surgical excision if they become repeatedly infected. The facial nerve is at risk of injury during the excision of these lesions because of the close relationship of the preauricular cyst or fistula to the superior branches of the facial nerve within the parotid gland. First branchial cleft anomalies have a more complex embryologic origin than preauricular cysts and fistulas. These lesions may not have an obvious sinus tract on the skin and may manifest as an abscess extending deeply into the EAC, parotid, and/or neck.
Differential diagnoses:

- **Lacerations:**
  - Full-thickness auricular lacerations may be observed after blunt or sharp trauma. These injuries are managed surgically by closing both the perichondrium and the skin. In contrast, external canal lacerations may occur after attempts at cleaning the ear canal using cotton-tipped applicators. These lacerations are usually managed by microscopically placing any skin flaps in their normal position, packing the ear canal, and administering topical antibiotic drops.

- **Atopic dermatitis:**
  - Drug sensitivity to topical antibiotic solutions is well known. Neomycin allergy occurs in up to 5% of patients treated with the medication. Suspect drug sensitivity if worsening of symptoms associated with skin excoriation and weeping occurs in the distribution of the topical medication exposure after application of drops. Metal sensitivity also manifests as excoriation, erythema, and edema around the exposure site (e.g., a piercing hole). A common allergen is nickel, an impurity that may be present in precious metals. Atopic dermatitis is managed by removal of the allergen, such as an earring, and beginning topical steroid and antibiotics if the wound is secondarily infected. The diagnosis of metal sensitivity is confirmed by performing a skin patch test.
Differential diagnoses:

- **Cerumen impaction:**
  - Cerumen impaction is the most common abnormality found on otoscopic examination, yet only a small proportion of the general population requires regular disimpaction because the EAC has the innate ability to produce and clear itself of cerumen. Cerumen may vary in color and consistency and may exist with other pathologies. Of note, debris in the EAC from cholesteatoma or tumors may be confused with cerumen, indicating that considerable care is required when attempting debridement of the EAC. Debridement may be accomplished with microinstruments or by aspirating the ear canal contents with a No 5 or No 7 Barton suction, while under direct vision through the otoscope or microscope. Irrigation of the ear canal is another option, but use of a pressurized irrigation system entails the risk of trauma.
Differential diagnoses:

- **Exostosis and osteoma:**
  - The 2 most common bony lesions of the EAC, exostoses and osteomas, differ histologically and clinically. Exostoses tend to arise from the anterior and/or posterior floor of the medial EAC. Exostoses have a sessile base and are covered with normal-appearing skin. Both anterior and posterior exostoses may be found simultaneously. Osteomas may arise from any region of the bony EAC and often are pedunculated. Osteomas may also be either single or multiple and are covered by normal skin. Exostosis and osteomas require surgical treatment only if they are so large that they lead to a conductive hearing loss or intractable otitis externa.
Differential diagnoses:

- **Foreign body:**

  Foreign bodies are not infrequently encountered in the EAC. In children, parts of toys or even food may be found in the EAC, and, thus, appearance varies. In adults, fragments of cotton swabs are the most common finding. Erythema and edema surrounding the foreign body are commonly present. Using microinstruments, the foreign body may be removed under a microscope, depending on the patient's ability to cooperate.
Differential diagnoses:

- **Acute (bullous) and chronic (granular) myringitis:**
  - Acute myringitis is usually caused by a mycoplasma or viral infection and is observed in adults and children. It is characterized by hemorrhagic bullae involving the tympanic membrane and a flulike syndrome. It is self-limiting and requires pain and fever management. Chronic myringitis is defined as deepithelization of the tympanic membrane, granulation tissue formation, and discharge. Treatment includes topical application of eardrops, a caustic solution in unresponsive cases, and mechanical removal of polypoidal granulations.
Organisms

1. *Pseudomonas* species
2. Staphylococci
3. Streptococci/Gram negative rods
4. Fungi (*Aspergillus*/*Candida* species)
Identify:

- These black dots (spores) are the appearance of fungal infection (*aspergillus niger*), with other fungi the spores may be white or yellow.

- *chronic otitis externa*: Although the canal wall is not swollen, the skin is excoriated and red. The drum is essentially normal.
Labs/workup

Usually after failed empiric therapy:

- bacterial and fungal culture
- Adults with otitis externa: screening blood glucose and/or a urine dipstick test to rule out occult diabetes.
- Additional tests (if available):
  - Gram stain of d/c
  - KOH prep smear (within 10 min)
Imaging

• Imaging studies are not required for simple otitis externa

• However, in patients with suspected malignant otitis media (diabetic or immunocompromised):
  – scan using CT with contrast
  Additional/Optional:
  – MRI of the temporal bone
  – triple-phase bone scanning using Technetium-99m
  – gallium scanning
Topical Treatment (assumes intact TM)

- Acetic acid with and without hydrocortisone (EarSol HC, VoSoL HC, Acetasol HC)
  - Apply in affected ear TID
- Neomycin, polymyxin B, and hydrocortisone (Cortisporin Otic)
  - Apply in affected ear TID
- Ciprofloxacin (Ciloxan)
  - Apply gtt in affected ear BID
- Ofloxicin (Floxin)
  - Apply in affected ear BID or 10 drops in affected ear QD
- Nystatin powder (Mycostatin, Nilstat)/Boric acid powder
  - 1-2 puffs from handheld nebulizer q1wk
Mr. A.T is a 53 y/o Hispanic male with PMHx sig. for well controlled DM (HbA1C 6.5) and severe fungal otitis externa 7 yrs ago requiring gross debridement and hospitalization. CC = clear, non-purulent, non-odorous d/c from his left ear for the past 10 days following an URI. Pt. denies dizziness, increasing pain, or fever.

On exam,
- Right Ear: right TM intact, non-erythematous, no fluid present
- Left Ear: EAC appears white and wet with friable cheesy material present. Non-bloody. Large central perforation present.
Next step in management?

- Labs?
- Imaging?
- Empiric therapy with f/u?
  - What will you prescribe?
Treatment plan

- Obtain fungal and bacterial cultures
- No imaging necessary
- Tolnaftate 1% topical in L ear BID x 7 days (he didn’t use)
- Ofloxacin 0.3% otic, 4 gtts in L ear BID x 7 days
- F/U in 2 wks
On arriving in the ER for simple EO consult you find pt has:

- Severe, unrelenting, deep-seated otalgia
- Temporal headaches
- Purulent otorrhea
- Dysphagia, hoarseness, and/or facial nerve dysfunction
Physical exam:

- Inflammatory changes are observed in the external auditory canal and the periauricular soft tissue
- The pain is out of proportion to the physical examination findings
- Marked tenderness is present in the soft tissue between the mandible ramus and mastoid tip
- Granulation tissue is present at the floor of the osseocartilaginous junction. This finding is virtually pathognomonic of malignant external otitis (MEO).
- Fever is uncommon, but if present, usu > 39C
Orders?

- Labs
  - Cultures (bacteria & fungi)
  - Glucose monitoring
- Imaging
  - **CT scanning** or MRI of the temporal bone
  - triple-phase bone scanning
  - gallium scanning
Next step

- Admit patient
- Place on empiric IV Antibiotics until organism is isolated through culture
- **painless relief**
- Once organism isolated, treat appropriately
- Consult Infectious Disease
- Use decreased **severe pain** as marker of improvement
- Surgery is necessary **only if necrosis is present**
Treatment

- meticulous glucose control if diabetic
- aural toilet
- systemic and ototopic antimicrobial therapy (fluoroquinolone)
- hyperbaric oxygen therapy
- debridement
Treatment options

• Ciprofloxacin 1500-2250 mg/d PO/IV divided bid/tid
  – Resistance seen in up to 33% of pts with MOE who fail OP treatment
• Ceftazidime 1-2 g IV q8h
• Ticarcillin/clavulanate (Timentin) 3.1 g IV q6h
Duration of Treatment and F/U

- Treatment response should be evaluated with a gallium citrate (Ga 67) scan every 4-6 weeks during treatment.
- Benecke recommended ending treatment 1 week after the gallium citrate Ga 67 scan findings return to normal and confirming this with a repeat scan 1 month after the treatment is stopped.
- Using this protocol, average duration of treatment was 8.8 weeks with a range of 4-17 weeks.
- Study followed 13 pts gathered over 4 yrs in the Los Angeles area
Duration of Treatment and F/U

- **Osteomyelitis of the skull base** is the most severe form of malignant otitis externa. As a result of having treated 13 patients with skull base osteomyelitis over a 4-year period, we have developed a method of staging and monitoring this malady using gallium and technetium scanning techniques. Stage I is localized to soft tissues, stage II is limited osteomyelitis, and stage III represents extensive skull base osteomyelitis. All stages are treated with appropriate anti-pseudomonal antibiotics. The duration of therapy depends upon the clearing of inflammation as shown on the gallium scan. Each case must be looked at independently and not subjected to an arbitrary treatment protocol.

- Gallium-67 citrate accumulates in areas of active inflammation by binding to leukocytes and forming a complex with lactoferrin [26]. Hence, nuclear scanning with gallium-67 will be positive for soft tissue and bone infections. Enhanced uptake will be present in areas of skull base osteomyelitis, but unlike the technetium-99 scan, it returns to normal sooner once the infection is resolved (Figs. 3 and 4) [27]. This scan should be repeated every 4 weeks to monitor antibiotic response until it is normal [8], although some patients may have persistence of uptake despite clinical resolution of infection [7]. A more accurate evaluation of response to treatment that seems to correlate with clinical response has been proposed in the form of the lesion to nonlesion ratio.
References: