Aug 2: Disorders of External Ear

**Aug 2: Disorders of the External Ear (Cornell)**

**Vacation: Vicki ; Preceptor: none**

1. (Amy) Discuss the embryology of the external ear. At what time during embryogenesis does the auricle develop?

Pharyngeal grooves are invaginations of surface ectoderm. The first pharyngeal groove thickens during the 8th week of gestation, giving rise to the epithelial lining of the external auditory meatus. The concha is formed by ectoderm from the first pharyngeal groove. Pharyngeal groove 1 is surrounded by six auricular hillocks called the Hillocks of His, which arise from condensation of the mesoderm of the first and second arches occurs during the 6th week of gestation. The first three hillocks are derived from the first arch, and the last three are derived from the second arch.

The cartilage forms during the 7th week. On the 12th week, the auricle is formed by fusion of the Hillocks. By week 20, the auricle has reached its adult shape, although it does not reach adult size until age 9.

**Hillocks of His:** 1-tragus 2-helical crus 3-helix 4-antihelix 5-antitragus 6-lobule and lower helix

2. (Dara) Name the normal landmarks of the auricle? What are the normal dimensions of the auricle?

The dimensions and proportions of the auricle are critical for reconstruction. The vertical height of the ear is roughly equal to the distance from the lateral orbital rim to the helical root at the level of the brow. The width of the ear is approximately 55% of its height. This corresponds to an average size of 63.5 mm x 35.3 mm in males and 59.0 mm x 32.5 mm in females. The helical rim protrudes between 20 and 30 degrees from the skull, which corresponds to 1 to 2.5 cm. The cephaloauricular angle is generally less than 45 degrees. The vertical axis of the ear is tilted posteriorly (when relating the apex of the helix to the lobule) 15 to 20 degrees. The superior level of the ear is at the same height as the lateral brow. The inferior aspect of the ear is at the same height as the nasal base.

3. (Deya) Discuss the etiology, management and potential complications of auricular hematoma.

**Auricular hematoma** – accumulation of blood in the subperichondrial space usually secondary to blunt trauma.

- **Etiology** – Cartilage lacks its own blood supply and relies on the vascularity of the surrounding perichondrium via diffusion. Shearing forces secondary to blunt trauma to the pinna disrupt the perichondrium, leading to an accumulation of blood in the subperichondrium. This in turn creates a barrier for diffusion b/w the cartilage and the perichondrial vascularity, leading to necrosis of the cartilage and predisposing it to infection and further injury.
- **Complications** – cartilaginous necrosis and permanent disfigurement such as the “cauliflower ear.” To prevent this, treatment must never be delayed beyond 7 days and the sooner you treat it, the better.
- **Treatment** – evacuation of the hematoma. Done using a skin incision parallel to the natural auricular skin folds. The irrigation of the evacuated space with topical antibiotics is recommended to reduce likelihood of infection. Splinting after drainage will prevent reaccumulation (options include cotton bolster, plaster molds, and silicon putty). Note that aspiration of the hematoma is not sufficient due to likelihood of reaccumulation.
4. (Amy again) From an embryologic standpoint, explain a first branchial cleft anomaly (type I and II) and why the facial nerve needs to be considered in surgical excision of this anomaly.

First branchial anomalies comprise approximately 1% of all branchiogenic anomalies. They result from developmental aberrations of the cleft between the first and second branchial arches, resulting in cysts, fistulae, or sinuses. The type I first branchial cleft anomaly is ectodermal in origin and is considered to be a duplication of the external auditory canal, while the type II anomalies contain both ectodermal elements (skin) and mesodermal elements (cartilage).

The type I first branchial anomaly is very rare and typically appears in the periauricular region as a cyst, sinus, or a fistula connecting to the skin and the external auditory canal. These lesions are lined with squamous epithelium and appear posterior, inferior or medial to the concha and pinna. They are typically lateral to CN VII and may located in the angle of mandible or be embedded in the parotid tissue, making them difficult to distinguish from a solid parotid mass on clinical exam. If present, the sinus tract parallels the external auditory canal. The treatment is surgical excision in the noninfected state.

The type II first branchial cleft anomalies are much more numerous and are typically located just inferior or posterior to the angle of the mandible. They are associated with the submandibular gland or found in the anterior triangle of the neck, and they frequently contain a fistula to the concha, EAC, or neck. Type II cysts contain skin as well as cartilage and course over the angle of the mandible, through the parotid, ending at the bony-cartilaginous junction of the EAC. These lesions are intimately associated with the parotid gland, and they can lie lateral, medial, or in between the branches of the facial nerve. Patients may present with a parotid mass, parotitis, otalgia, or ototrahea if there is a tract with the EAC. The definitive treatment is surgical, usually with a superficial parotidectomy with identification of the facial nerve. Portions of the cartilaginous external auditory canal may need to be excised along with involved skin.

(ABOVE) First branchial cleft cyst, Type II: non-enhancing mass posterior to R submandibular gland

5. (Dara again) What is microtia? What are the different classifications and the overall incidence?

Microtia by definition means “small ear.” Occurring once in about every 7,000 to 8,000 live births, microtia represents the most common major congenital anomaly of the external ear. Microtia may be either uni- or bilateral and occurs more often in right ears and in males, and more often in Hispanics and Asians than blacks and whites. Fewer than 15% of the cases have a positive family history. Among associated malformations, facial cleft and cardiac defects are the most common followed by anophthalmia or microphthalmia, limb reduction defects, severe renal malformation, and holoprosencephaly. Some feel that microtia represents the mild end of the spectrum of hemifacial microsomia, with Goldenhar syndrome being the severe end of the spectrum. Microtia is classified as follows from less severe (grade I) to total anotia:

- **Grade I**: The pinna is malformed and smaller than normal. Most of the characteristics of the pinna, such as the helix, triangular fossa, and scaphae, are present with relatively good definition.
- **Grade II**: The pinna is smaller and less developed than in grade I. The helix may not be fully developed. The triangular fossa, scaphae, and antihelix have much less definition.
- **Grade III**: The pinna is essentially absent, except for a vertical skin remnant. The superior aspect of this remnant consists of underlying unorganized cartilage, and the inferior aspect consists of a relatively well-formed lobule.
- **Anotia**: Total absence of the pinna is observed.
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6. (Josh) Discuss the appropriate evaluation for the patient with unilateral microtia?
   • The external ear, EAC, and TM are inspected and compared to the contralateral side
   • Rule out additional congenital abnormalities (esp. mandible, mouth, maxilla, eyes and neck, e.g. epibulbar dermoids, coloboma, maxillary/mandibular asymmetry, vertebral abnormalities)
   • Audiogram (OAE/ABR if <7mo) to confirm normal hearing in unaffected ear
   • Axial and coronal fine-cut CT of temporal bones (deferred until age 5-6), skull base, maxilla, mandible and neck (if vertebral abnormalities are suspected) to evaluate middle ear, mastoid pneumatization, and symmetry of facial skeletal growth
   • Craniofacial multidisciplinary referral if multiple anomalies found

7. (Josh) What additional considerations need to be made in the case of bilateral microtia with complete aural atresia? What is the incidence of bilateral occurrence?
   Patients with bilateral microtia with canal atresia will require bone conduction hearing aids, ideally applied at 3-4 weeks of age. A decision about the possibility of an implantable hearing device (BAHA) is not recommended until auricular reconstruction is completed. Hearing amplification is not necessary in patients with unilateral atresia if hearing is normal in the contralateral ear.

Unilateral cases outnumber bilateral cases by a 4:1 ratio. Bilateral microtia occurs in 0.0075% of live births

8. (Caroline) Discuss the course of the facial nerve in cases of aural atresia.
   - CN VII aberrant in 25-30% of patients
   - 2 main differences in facial nerve course: (1) more acute turn at the second genu, thus causing (2) the mastoid segment of the facial nerve to course more anterior and lateral than usual
   - crosses the middle ear at the level of the round window and leaves the temporal bone at the glenoid fossa instead of the stylomastoid foramen. In surgery, it possesses an inferior and posterior relationship with the atretic plate.

9. (Caroline) What is the ideal age for reconstruction of aural atresia? What are the important considerations for timing?
   - 2 absolute requirements for surgical consideration:
     1) normal inner ear morphology demonstrated on CT scan
     2) normal cochlear function demonstrated by audiologic testing
   - Unilateral atresia patients usually do not require any intervention as long as normal hearing is present in the contralateral ear.
   - Surgical repair deferred until about 6 years of age
   IMPORTANT CONSIDERATIONS:
   - Until repair, need to continue mental development and speech skills which require bone conducting appliances (for patients with cochlear function)
   - Undergo microtia repair first (if needed) to avoid scar tissue formation
     o around 5-6 years old when costal cartilage is sufficiently developed for harvesting and there is optimal development of the mastoid process
     o Aural atresia repair usually performed after the cartilaginous auricular framework is placed and the ear lobe is created and before the creation of the tragus and auricle elevation.

10. (Tali) Discuss the rehabilitation options for unilateral microtia. Discuss the different surgical stages and their timing.
    Grade I - normal ear, Grade II- some of the auricular framework is present, but there are obvious deformities, Grade III- peanut ear deformity, which encompasses anotia
    Reconstruction options:
    1. Prosthesis: good cosmetic result, dislodgement embarrassing, requires care

Pre-op Planning
   • Microtia repair before atresia to preserve virgin blood supply, limited cosmetic result if auricle reconstructed around drilled bony canal.
   • CT scan prior to surgery to assess middle ear development: need fine cuts axial and coronal
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Stage I: Goal: Framework fabrication and insertion: Age 5-8 (age 6: ~85% of adult ear height)
- Template creation: A film pattern is traced from the normal ear and reversed to plan the new framework. Carving template 1-2 mm smaller than contralateral ear
- Harvest contralateral rib + perichondrium. First free-floating rub used for helix and synchondrosis of ribs 6&7 for framework. Triangular fossa and scapha carved out
- Implant cartilage in subcutaneous pocket.
- In unilateral microtia, the position of the vestige from the lateral canthus is approximately the same as the normal ear’s helical root from the lateral canthus. If the patient has a low hairline, then it can either be adjusted after the reconstruction is completed or a smaller framework can be made and the opposite normal ear reduced.
- May use post-op mastoid dressing (24-48h) then glasscok x 2 weeks. Drains in place 2-4 days
- 2-3 months wait until next stage

Stage II: Goal: Lobule Transposition:
- Lobule remnant raised as simple transposition flap (inferiorly based) flap by incising around it; an incision is also made at the proposed superior inset margin.
- To avoid excessive protrusion of the lobule, the superior posterior incision on the back of the ear should be made fairly high.
- Remove redundant skin from site of native cartilage excision
- Glasscok x 1 week, keep wound moist with antibiotic ointment
- 3 months until stage III

Stage III: Goal: Creation of posterior sulcus/projection of ear away from mastoid
- Harvest STSG from groin/abdomen
- Transplant into incision creating posterior sulcus
- Advance posterior auricular skin into sulcus to project ear laterally. May insert cartilage graft for more projection
- Beware anomalous course of facial nerve: use nerve monitoring
- Some consider atresia repair as 3rd stage if sensorineural hearing intact

Stage IV: Goal: Tragal Reconstruction
- Composite chondrocutaneous graft harvested from conchal bowl of normal ear and applied beneath a tragal flap developed by J-shaped incision
- May need to graft donor site if primary closure caused distortion of the normal ear
- To accentuate conchal depth excess soft tissue in the bowl is excised.

Stage V: Auricular Elevation
- This step not always necessary but may need to further elevate ear off of mastoid. Involves raising skin flaps in posterior sulcus and possible skin grafts.

11. What are the stated success rates for surgical repair? What are the most common complications?

The outcomes of microtia repair are reflected in the psychological benefit and satisfaction of the patient and/or parent. Brent surveyed his patients in 1992 and noted that those grading themselves as severely affected prior to surgery were 100% pleased with the result. Among patients who considered themselves moderately disturbed by microtia and underwent the procedure when younger than 14 years, 95.5% were satisfied with the decision to pursue surgery.

Complications:

<table>
<thead>
<tr>
<th>Immediate</th>
<th>Delayed</th>
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</thead>
<tbody>
<tr>
<td>hematoma</td>
<td>Atelectasis most common complication</td>
</tr>
<tr>
<td>skin flap necrosis</td>
<td>Graft exposure</td>
</tr>
<tr>
<td>pneumothorax, pneumomediastinum</td>
<td>Malpositioning</td>
</tr>
<tr>
<td>infection → chondritis → graft loss</td>
<td>Scar contraction/hypertrophic scar/keloid</td>
</tr>
</tbody>
</table>

12. Review the anatomy of the external auditory canal. Describe the “ideal” external canal environment.

Morphology:
- EAC is typically 24 mm in length with a volume capacity of 1-2mL.
- The lateral one-third of canal is made of fibrocartilage, while medial two-thirds is osseus.
- External canal is straight in early childhood, and takes an “S” shape by age 9.

Skin:
- EAC is lined by stratified squamous epithelium that is continuous with skin of pinna and epithelial covering of TM.
- Subcutaneous layer of cartilaginous portion (~1mm thick) contains hair follicles, sebaceous glands, and ceruminous glands.
- Ceruminous glands are modified apocrine sweat glands surrounded by myoepithelial cells and are organized in apopilosebaceous units.
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- Epithelium of EAC migrates laterally at rate of ~0.07mm/day, thought to occur at the basal layer, and allows the canal to remain unobstructed by debris that could impair transmission of sound and form a nidus of infection.

Innervation:
- Auricular branch of vagus n. (Arnold’s nerve) innervates inferior bony canal, posterosuperior cartilaginous canal and TM.
- Auricular branch of facial n. innervates posterosuperior bony EAC.
- Auriculotemporal branch of mandibular division of trigeminal (V3), and glossopharyngeal contributions are not well delineated.

Vascular Supply:
- Posterior auricular artery and auriculotemporal branch of superficial temporal artery arise from external carotid artery, and supply the auricle and lateral EAC.
- Deep auricular branch of maxillary artery supply medial EAC and external surface of TM.

Environment:
- “Ideal” environment is dry, preventing maceration, with an acidic pH that provides an inhospitable environment for pathogens.
- Cerumen provides a hydrophobic coating for the canal, preventing maceration.
- Antibacterial properties of cerumen shown in vitro, yet its effects in vivo are unclear.

13. (Deya again) Differentiate exostoses vs. osteomas. Exostoses and osteomas are both benign EAC overgrowths. They can be very difficult to clinically differentiate the two but here are some key differences:

<table>
<thead>
<tr>
<th></th>
<th>Exostoses</th>
<th>Osteomas</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Location</strong></td>
<td>Benign periosteal outgrowths that occur in the bony canal</td>
<td>Benign bony growth, often at bony cartilaginous junction of tympanomastoid suture line</td>
</tr>
<tr>
<td><strong>Number/Side</strong></td>
<td>Multiple and bilateral</td>
<td>Single and unilateral</td>
</tr>
<tr>
<td><strong>Base</strong></td>
<td>Broad base</td>
<td>Pedunculated</td>
</tr>
<tr>
<td><strong>Etiology</strong></td>
<td>Reactive bone formation to exposures to cold water “surfer’s ear”</td>
<td>Benign osseous neoplasm</td>
</tr>
</tbody>
</table>

In general, treatment for either condition is not usually required. However, surgical removal of either may be necessary for persistent accumulation of debris medially to the growths, recurrent otitis externa from fluid retention, conductive hearing loss, or interference with insertion of a hearing aid.

14. (Scott) Acute otitis externa...bacteriology and treatment (combined below)

15. (Scott again) Why does fungal otitis externa occur? How can you tell it is fungus? What is the treatment?

Acute OE is a superficial infection of the skin of the EAC with two common initiating events: moisture trapping and trauma to the EAC. This introduces bacteria, leading to occlusion of the apopilosebaceous units ➔ inflammatory response with skin edema ➔ exudate +/- pus.

If severe it may cause cellulitis of face/neck surrounding the EAC and/or osteomyelitis of the temporal bone. **Bacterial pathogens:** Pseudomonas aeruginosa (38%), Staphylococcus aureus, and gram negative rods (Proteus species, Diphteroids, E. Coli), Staphylococcus epidermis **Fungal pathogens:** Aspergillus (80%), Candida

Fungal OE may result from overtreatment of EAC with topical antibiotics, or it occasionally may present de novo from moisture trapped in the EAC.

Fungal infections: 1) tend to have severe itch but less pain than bacterial OE; 2) thick white/gray discharge; 3) visible fungal elements / fuzzy appearance.
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Necrotizing OE: immunocomprised (DM, post-transplant, HIV) patients, true osteomyelitis of temporal bone; may present as deep-seated ear pain.

**Treatment:**
1. Most respond well to topical treatment (antibiotic ear drops +/- steroid). Includes Cortisporin (Neomycin, polymyxin B, hydrocortisone), Floxin, Ciprofloxacine, Tobradex, Gentamycin, Ciprodex.
2. Topical acidifying/drying agents (Vosol, Acetic Acid, EtOH+vinegar+H20): good for fungal OE
3. Antiseptic treatments: acetic acid, boric acid, ichthanimol, phenol, aluminium acetate, gentian violet, thymol, thimerosal (Merthidate), cresylate, EtOH
4. May need wick placement for severe edema to allow topical medication to penetrate EAC.
5. Serial cleanings for severe OE, fungal infections.
6. Oral antibiotics for patients with cellulitis or EAC edema preventing topical penetration (Cipro).
7. IV antibiotics for patients with NOE or failed oral antibiotics.
8. Surgical debridement for those with NOE.
9. Incision and drainage if abscess forms (typically S. aureus).

16. (Kathy again) Discuss the various ways to manage cerumen impaction. Which do you prefer and why. How will you instruct patients on proper aural hygiene?

Cerumen impaction can be managed conservatively by administration of cerumen dissolving otic soln such as “Debrox” daily for several weeks. It may also removed in the office with cerumen loops (curette) and/or suction if the cerumen is soft, or alligator forceps or right-angle hook if cerumen is hard.

Proper aural hygiene involves keeping the EAC dry with earplugs or cotton ball coated with Vaseline is patient is to be submerged in water. Avoidance of overzealous cerumen removal, especially with Q-tips is also recommended.


The following system was developed based on a retrospective review of 32 patients at Pittsburgh with primary SCCA of the EAC. Extent of disease was based on CT findings and confirmed/upgraded as needed based on intraoperative and histologic findings.

**TABLE 1. Pittsburgh tumor staging system (modified)**

<table>
<thead>
<tr>
<th>Stage</th>
<th>CT or pathologic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>Tumor limited to the EAC without bony erosion or evidence of soft tissue involvement</td>
</tr>
<tr>
<td>T2</td>
<td>Tumor with limited EAC bone erosion (not full thickness) or limited (&lt;0.5 cm) soft tissue involvement</td>
</tr>
<tr>
<td>T3</td>
<td>Tumor eroding the osseous EAC (full thickness) with limited (&lt;0.5 cm) soft tissue involvement, or tumor involving the middle ear and/or mastoid</td>
</tr>
<tr>
<td>T4</td>
<td>Tumor eroding the cochlea, petrous apex, medial wall of the middle ear, carotid canal, jugular foramen, or dura, or with extensive soft tissue involvement (&gt;0.5 cm), such as involvement of TMJ or styloid process, or evidence of facial paresis</td>
</tr>
</tbody>
</table>

CT, computed tomography; EAC, external auditory canal; TMJ, temporomandibular joint.

**TABLE 3. Two-year survival by stage**

<table>
<thead>
<tr>
<th>Stage</th>
<th>n</th>
<th>Survived</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>7</td>
<td>7</td>
<td>100</td>
</tr>
<tr>
<td>T2</td>
<td>5</td>
<td>4</td>
<td>80</td>
</tr>
<tr>
<td>T3</td>
<td>6</td>
<td>3</td>
<td>50</td>
</tr>
<tr>
<td>T4</td>
<td>14</td>
<td>1</td>
<td>7</td>
</tr>
</tbody>
</table>

Other staging systems:
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<table>
<thead>
<tr>
<th>Author, date</th>
<th>Subjects</th>
<th>Staging system</th>
<th>Outcome (in months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crabtree et al., (15), 1976</td>
<td>SCCA, BCCA, ACC, EAC</td>
<td>Localized: confined to EAC and mastoid</td>
<td>15/17</td>
</tr>
<tr>
<td>Stell and McCormick (11), 1985</td>
<td>SCCA, BCCA, ACC, AC</td>
<td>T2 limited to site of origin</td>
<td>Survival for stages T2 and T3 were significantly poorer than T1</td>
</tr>
<tr>
<td>Kinney (2), 1989</td>
<td>SCCA of EAC</td>
<td>T2 extends beyond site of origin, but not beyond organ of origin</td>
<td>85%</td>
</tr>
<tr>
<td>Shih and Crabtree (4), 1990</td>
<td>SCCA, BCCA, ACC, AC, MM of EAC</td>
<td>Stage 1 localized to EAC</td>
<td>6/10</td>
</tr>
<tr>
<td>Spector (3), 1991</td>
<td>SCCA of EAC</td>
<td>Stage 2 extends into temporal bone</td>
<td>(NED at 11 years)</td>
</tr>
<tr>
<td></td>
<td>I, 17 (1960–1980)</td>
<td>Stage 3 extends to parotid, neck, skull base, clivus</td>
<td>3/10 (60%)</td>
</tr>
<tr>
<td></td>
<td>II, 34 (1960-1980)</td>
<td>Deep invasion</td>
<td>9/14 (64%) for group II</td>
</tr>
<tr>
<td>Pensak et al., (9), 1996</td>
<td>SCCA, BCCA, ACC, chondrosarcoma, AC, osteogenic sarcoma, carcinoma of temporal bone</td>
<td>Stage 1 tumor in a single site, &lt;1 cm</td>
<td>3/3 (100%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage 2 tumor in a single site, &gt;1 cm</td>
<td>11/14 (78%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage 3—transnasal extension</td>
<td>0/3 (0%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage IV—maxillary or ethmoid extension</td>
<td>0/3 (0%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage V—paranasal or contiguous extension</td>
<td>0/3 (0%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage VI—intracranial extension</td>
<td>0/3 (0%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage VI—intracranial extension</td>
<td>0/3 (0%)</td>
</tr>
<tr>
<td>Masood and M. et al. (10), 1998</td>
<td>SCCA, BCCA of EAC</td>
<td>Stage 1—tumor in EAC</td>
<td>3/10 (60%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage 2—tumor in temporal bone</td>
<td>0/3 (0%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage 3—tumor in mastoid, facial nerve</td>
<td>100%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stage 4—spread to nerves, muscles, bone, sinuses, or skull</td>
<td>(overall failure rate)</td>
</tr>
</tbody>
</table>

ACC, adenoid cystic carcinoma; AC, adenocarcinoma; MM, malignant melanoma; TMJ, temporomandibular joint; NED, no evidence of disease; SCCA, squamous cell carcinoma; BCCA, basal cell carcinoma; EAC, external auditory canal; ME, middle ear.


Chronic stenosing external otitis (deep meatal stenosis) is an uncommon condition that is difficult to treat. Its natural history is that of an active, relentlessly progressive, infected stage characterized by chronic drainage, followed by a quiescent, mature, fibrotic phase in which conductive hearing loss is the main symptom. Physical exam shows inflamed, infected, thickened canal wall skin, often associated with granulation tissue. In the mature phase, there is stenosis or atresia of the canal by a fibrous plug. The cause is unknown. Surgery should not be considered until the active phase has passed and the fibrous plug has developed in the medial external auditory meatus.

Surgical treatment described in the article involves excision of all fibrous tissue and if necessary the drum, a wide canalplasty, a meatoplasty. It is imperative that all fibrous tissue be removed, or the risk of re-stenosis is high. If necessary, reconstruction is done with a fascial graft followed by split thickness skin grafts. Emphasis is placed on maintaining the anterior tympanoexternal angle to 90 degrees or more.


Report of 30 patients seen over 4 years that had MOE without DM. Otolgia with sx for at least 2 weeks (avg 32 days) was common presenting sx. Granulation, otorhea, edema, erythema all other presenting signs. 2/3 had abscess or necrotic tissue. Te99m was used to confirm osteomyelitis of the mastoid in many cases. Pseudomonas was isolated from all patients. Tx was ofloxin drops and IV abx (piperacillin or tobra) for avg of 19 days, as well as debridement. None of the pts were diabetic on further testing.

20. **(Jeff)** Keratitis obturans-describe the typical presentation and management.

I think they meant Keratosis obturans. Typical presentation is the presence of a thick dense plug of keratin in the eac causing CHL. Often they have severe pain, secondary otitis and granulation. TM thickening and mucosalization can occur, as well as canal skin inflammation. There is NO bone absorption. Etiology thought to be faulty eac migration patterns, also seen in post-XRT patients. Local cleaning/debridement is general management. Not to be confused with eac cholesteatoma (EACC) which present similarly but differs because there is the presence of bony erosion.