

**Chronic Otitis Media
with and without
Cholesteatoma**

Outlines

● Definition

- Epidemiology
- Classification
- Etiology
- Microbiology
- Pathogenesis
- Clinical presentation
- Work up
- Treatment

Definition

- Chronic otitis media (COM) is a long standing infection of part or whole of the middle ear cleft characterized by ear discharge and a permanent perforation.
- A perforation becomes permanent when its edges are covered by squamous epithelium and it does not heal spontaneously.



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- **Epidemiology**

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Epidemiology

- Not well defined.
- In UK:0.9% of children
0.5% of adults
- No gender difference
- Native Americans, Eskimos, Australian aborigines.
- Increase incidence in poor socioeconomic standards , poor nutrition , lack of health education and increase smoking.

Outlines

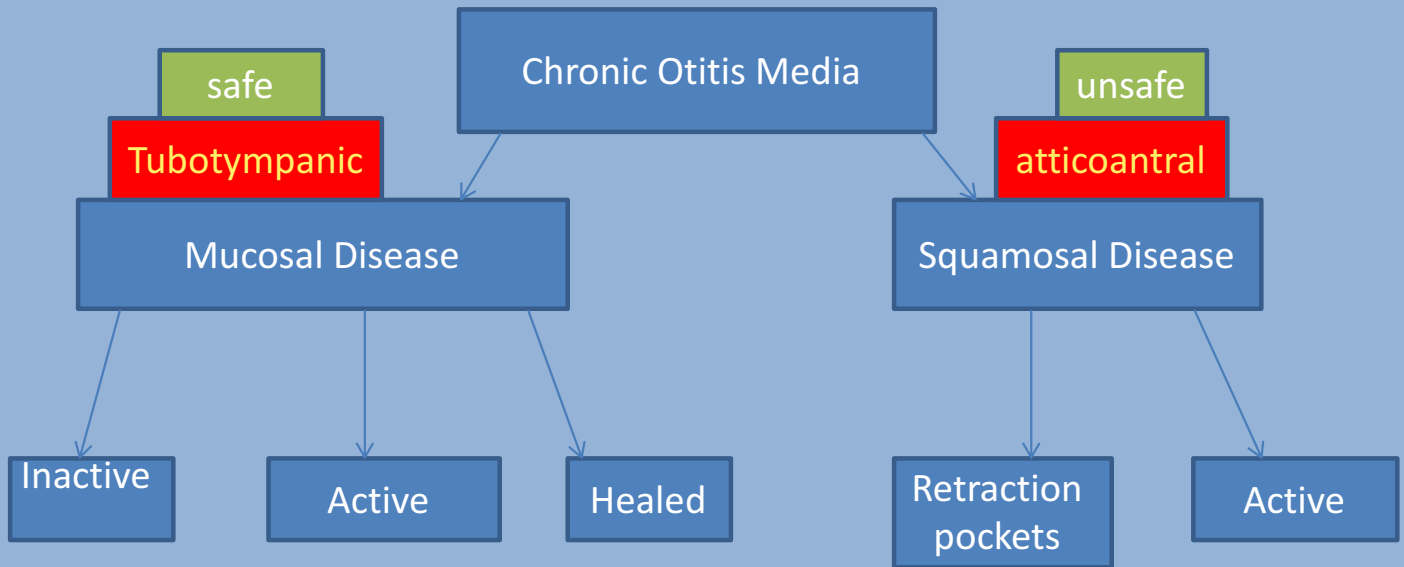
- Definition
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Classification

- Active Vs Inactive
- Mucosal Vs squamosal
- Tubotympanic Vs Atticoantral
- Safe Vs unsafe
- With or without cholesteatoma.

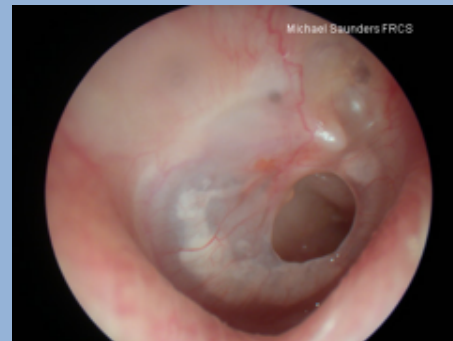
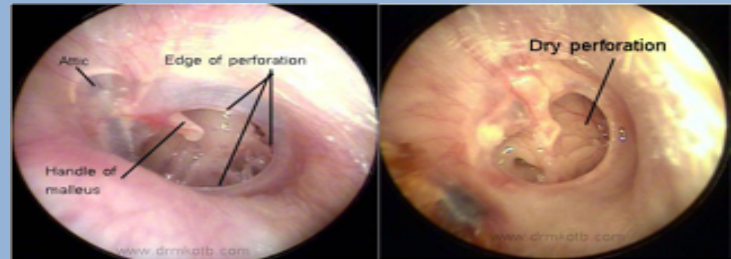


Classification



Inactive Mucosal COM

- Permanent Perforation of the Pars tensa.
- Middle ear and mastoid mucosa is not inflamed.
- Lamina propria around the perforation may be thickened.
- Mucocutaneous junction is at the margin of perforation.



Active mucosal COM

- Chronic inflammation of the mucosa
- Mucopurulent discharge
- Aural polyps
- Resorption of ossicular chain
- Tympanosclerosis



Healed COM

- Healed perforation
(Dimeric membrane)

secondary membo.



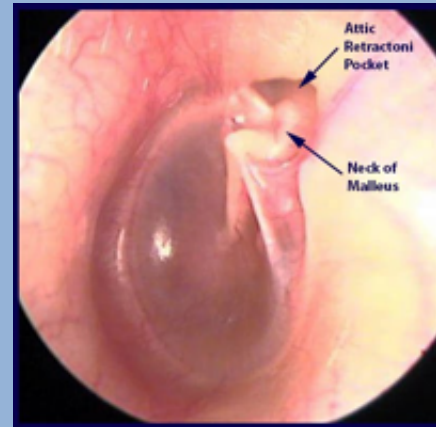
- Tympanosclerosis.

- Fibrocystic and fibro
osseous sclerosis



Inactive squamous epithelial COM

- Retraction pocket (atelectasis)
- Epidermization: Replacement of middle ear mucosa by keratinizing squamous epithelium without retention of keratin debris.
- Often remains quiescent and doesn't progress to cholesteatoma or active suppuration
- Not indication for surgical intervention



Active squamous COM(Cholesteatoma)

- Presence of cholesteatoma
 - Can be
 - dry(filled with keratin debris) or
 - Wet
- (active bacterial superinfection)
(malodorous otorrhea)



Active squamous COM(Cholesteatoma)

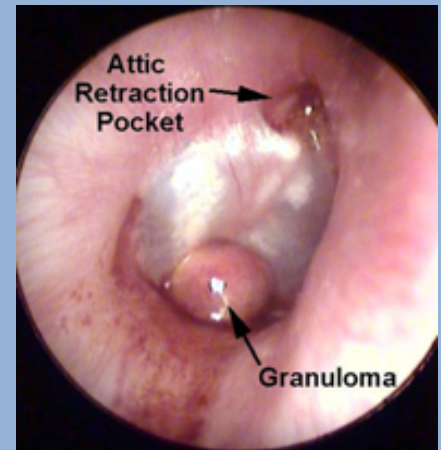
- Osteitis ,granulation tissue,
aural polyp

- Ossicular necrosis

- Cholesterol granuloma:

Mass of granulation tissue with foreign body giant cells surrounding a cholesterol crystal.

It is a reaction to long-standing retention of secretions or haemorrhage.



CSOM

	Tubotympanic (safe)	atticoantral (unsafe)
Discharge	profuse mucoid	scanty purulent Foul smell
Perforation	central	Marginal
Granulation	uncommon	Common
Polyp	pale	Red, fleshy
Cholesteatom	absent	Present
Complication	rare	Common
Audiogram	mild, moderate, conductive, deafness	Conductive or mixed deafness

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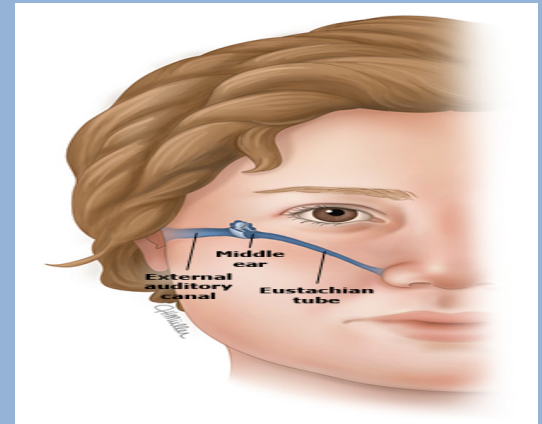
Etiology

- Mechanism of infection:

- ❑ Translocation from EAC through perforation.
- ❑ Reflux of ET

- Risk factors

- Hx of multiple episodes of AOM
- Living in crowded conditions.
- Day care facility attendance
- Being a member of large family
- Craniofacial abnormalities(eg cleft palate ,Down syndrome,....)



Outlines

- Definition
- Epidemiology
- Classification
- Etiology

● **Microbiology**

- Pathogenesis
- Clinical presentation
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Common pathogens

- Pseudomonas aeruginosa (48-98%)
- Staph. Aureus (15-30%)
- Klebsiella (15-30%)
- Proteus (10-15%)
- Polymicrobial (5-10%)
- Anearobes (20-50%)
- Fungi



Outlines

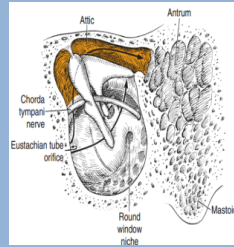
- Definition
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- Microbiology

● Pathogenesis

- Clinical presentation
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Pathogenesis of COM

- Factors allow active infection to develop into chronic are unclear.
- Episode of active infection → irritation and inflammation of mucosa → mucosal edema and ulceration → breaking of epithelial lining → attempts to resolve infection → granulation tissue ,polyps → viscous circle → destroy bony margins and complications

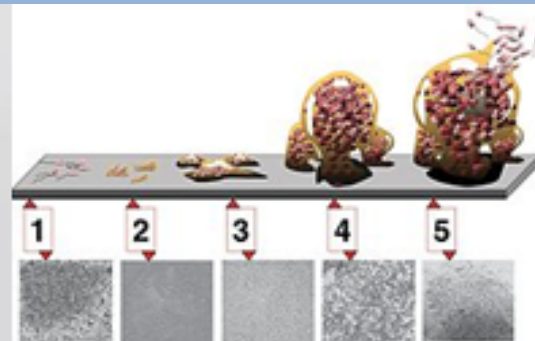


Biofilms

- Relatively new theory on etiology of COM.

- Significantly different characteristics from free-floating (planktonic) bacteria:

- Decreased metabolic rate
- Different gene expression
- **Encased** within matrix of extracellular polymeric substance
- Inhibits innate host immune response as leukocytes are unable to penetrate the matrix
- **Antibiotic resistance**
- Production of **efflux pumps** not seen in planktonic bacteria.



Five stages of biofilm development: (1) Initial attachment, (2) Irreversible attachment, (3) Maturation I, (4) Maturation II, and (5) Dispersion.

Pathogenesis of COM with Cholesteatoma

- Simple definition of cholesteatoma is skin in the wrong place!!
- Misnomer
- Microscopically

- **Expansile lesion of temporal bone composed of:**

- **Cystic content:** Desquamated keratin center
- **Matrix:** Keratinizing stratified squamous epithelium
- **Perimatrix:** granulation tissue that secretes multiple proteolytic enzymes capable of bone destruction.

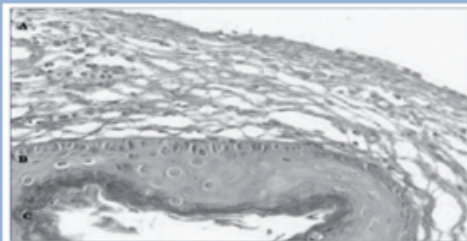


Figure 1. Digital images of the slide, with cross-section of the cholesteatoma, stained with Hematoxylin-Eosin. We can see three forming parts: A - A perimatrix - subepithelial connective tissue, containing collagen, elastic fibers, fibroblasts and inflammatory cells. B - A matrix -epithelium similar to normal skin epidermis. C - Cystic content - formed by keratin.

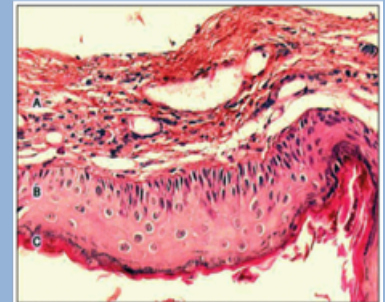
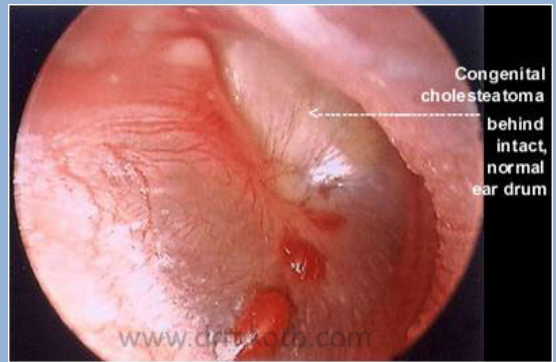


Figure 1. Digitalized image of the lamina, cross section of a cholesteatoma, stained with Hematoxylin-Eosin. We can see three forming parts: A - perimatrix, B - matrix, C - cystic content.

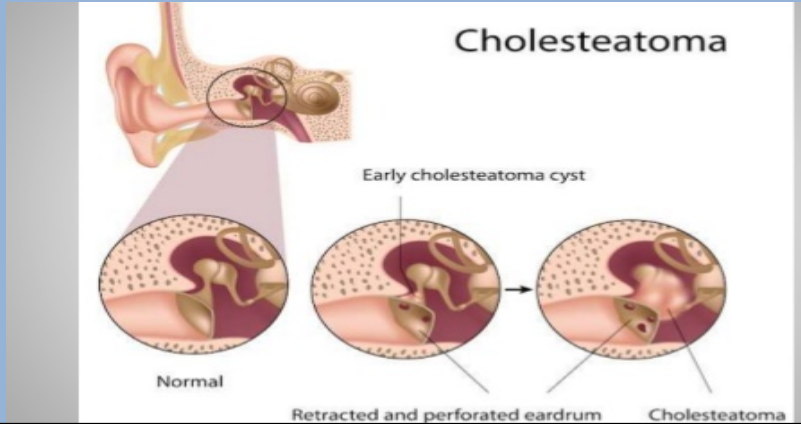
- ***Congenital***
cholesteatomas

- originate from areas of keratinizing epithelium within the middle ear cleft.



- ***Acquired***
cholesteatomas

- Four basic theories



Congenital Cholesteatoma

- From areas of keratinizing epithelium in the middle ear cleft in the developing fetus (anterior tympanum).
- Pearl-like mass behind usually intact TM
- M:F 3:1, 4.5 y/o
- Stages:
 - 1: Limited to one quadrant
 - 2: Multiple quadrants without ossicular involvement.
 - 3: ossicular involvement without mastoid
 - 4: Mastoid



Acquired Cholesteatoma

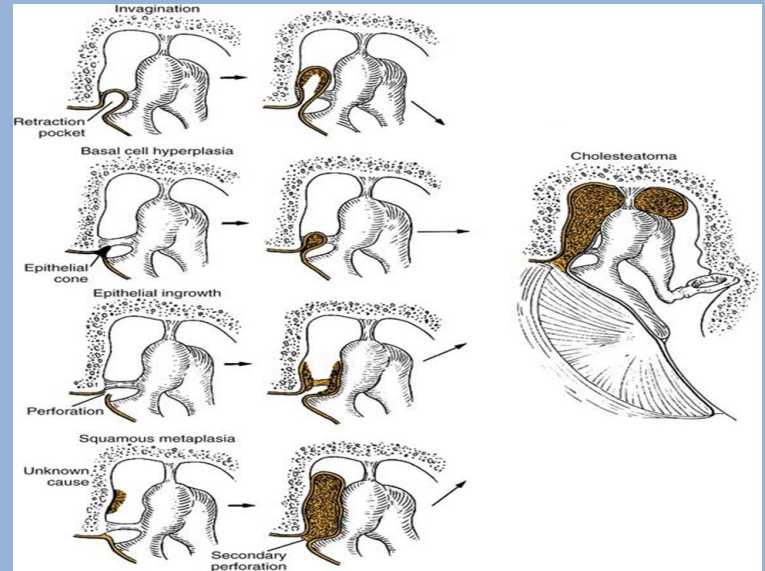
- Four Theories:

- ❖ Invagination theory

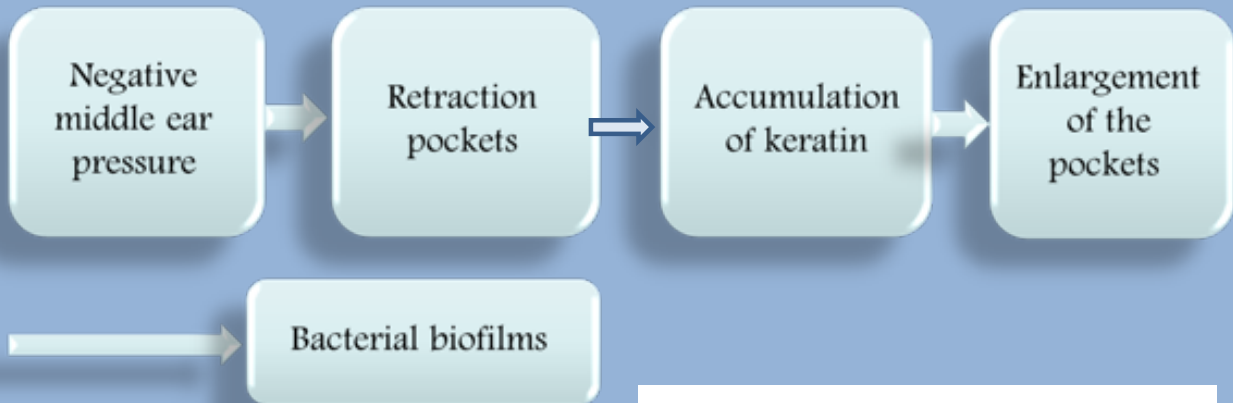
- ❖ Basal cell hyperplasia

- ❖ Epithelial invasion

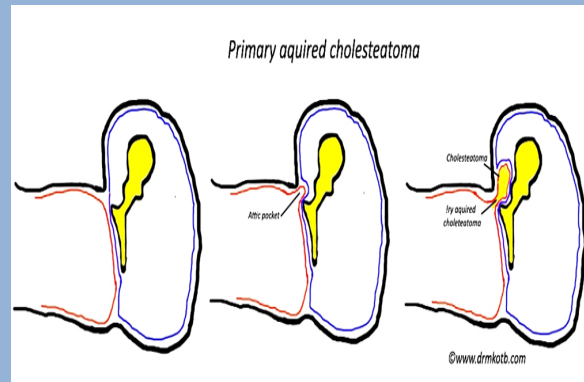
- ❖ Squamous metaplasia



Invagination Theory

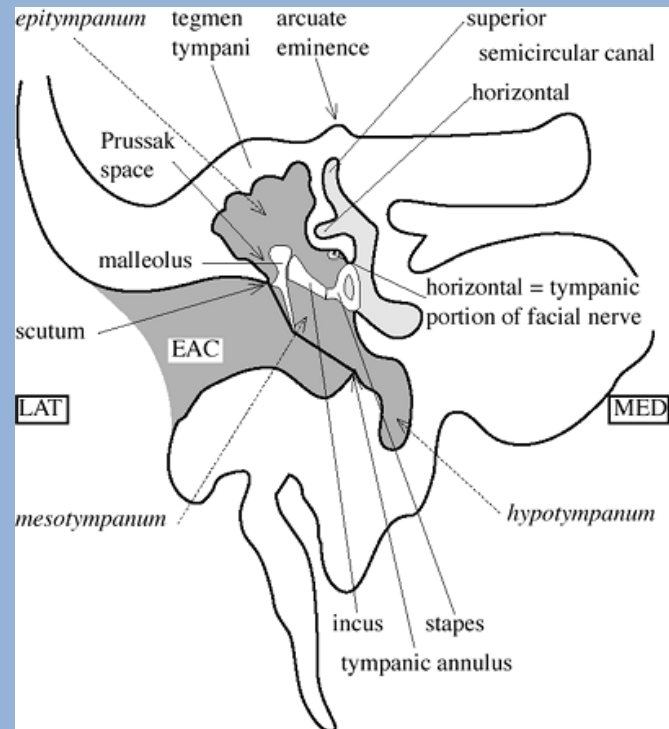


- **Attic cholesteatoma.**
- Primary acquired cholesteatoma
- Posterior superior part of the TM & pars flaccida



Toss's grades of retraction pockets

- Grade I : The **pars flaccida is retracted**, but is not in contact with the neck of the malleus.
- Grade II : The retracted pars flaccida is in **contact with the neck of the malleus** clothing it
- Grade III : **Minimal erosion of the outer attic wall**
- Grade IV : The **outer attic wall is drastically eroded**



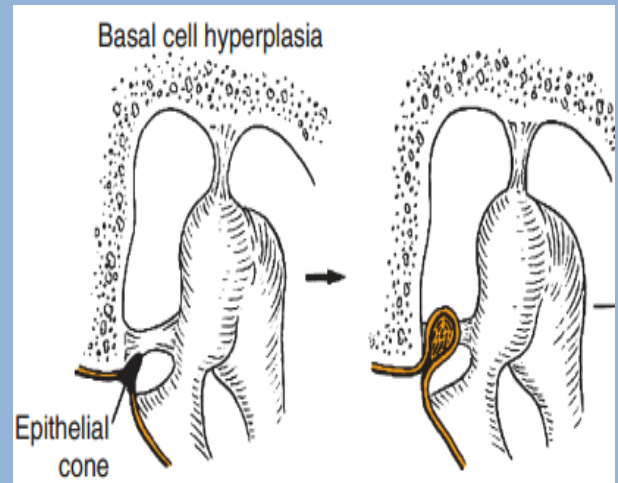
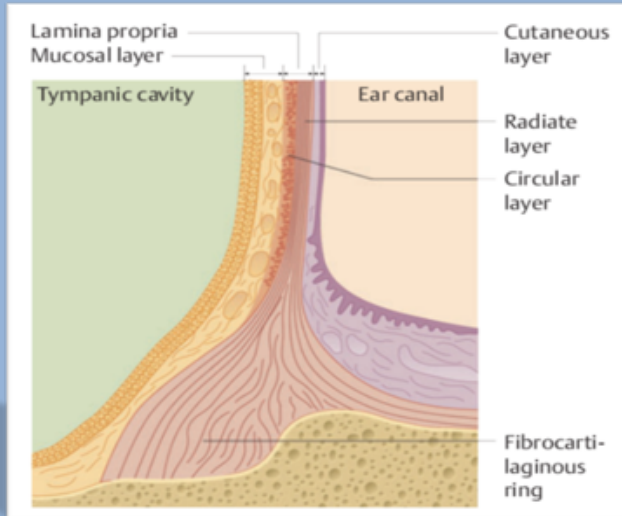
Basal Cell Hyperplasia

Basal lamina
disruptions

invasion of
epithelial
cones

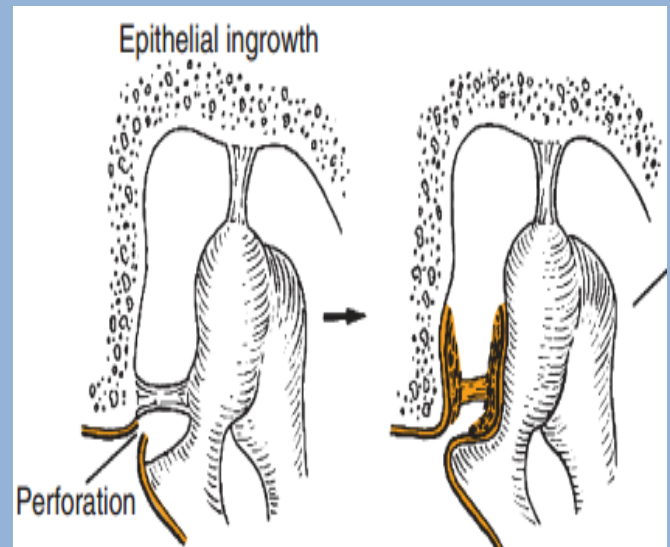
microcholesteatomas

Perforate TM
secondarily



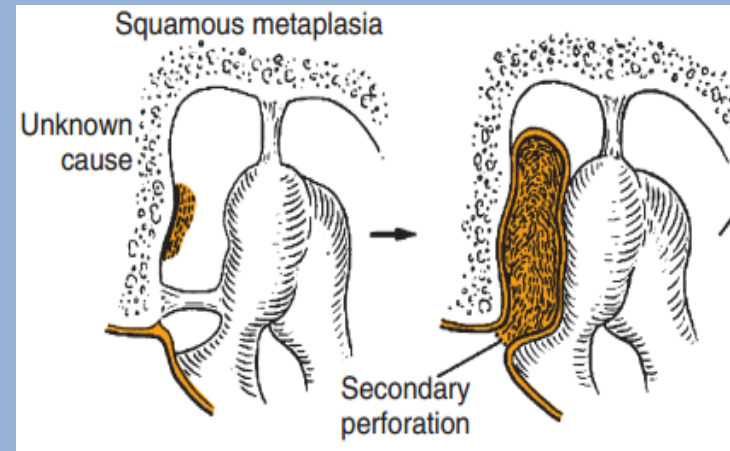
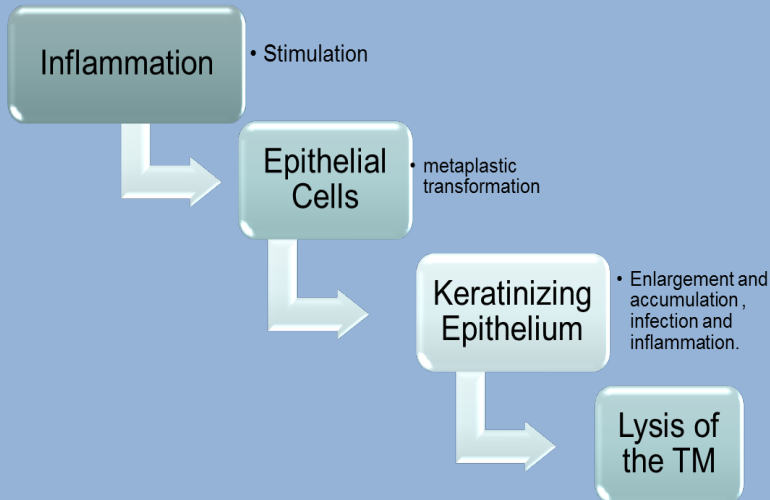
Epithelial Invasion Theory

- Secondary acquired cholesteatoma
- Keratinizing squamous epithelium from the surface of the TM migrates through perforation.
- Contact guidance & contact inhibition.

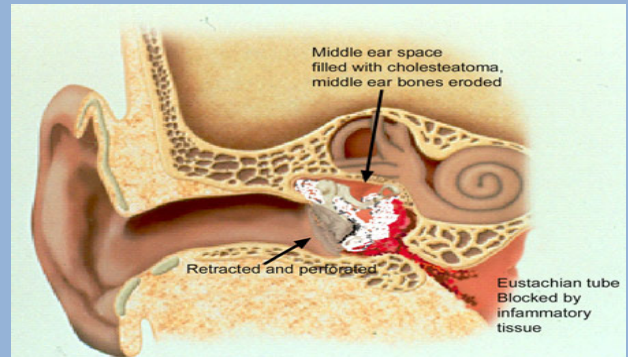


Squamous Metaplasia Theory

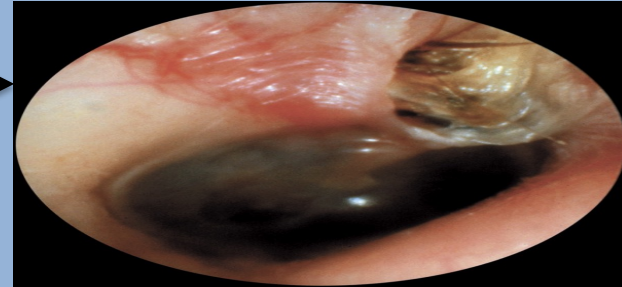
- Metaplasia of simple squamous or cuboidal epithelium in the middle ear cleft into keratinizing epithelium.



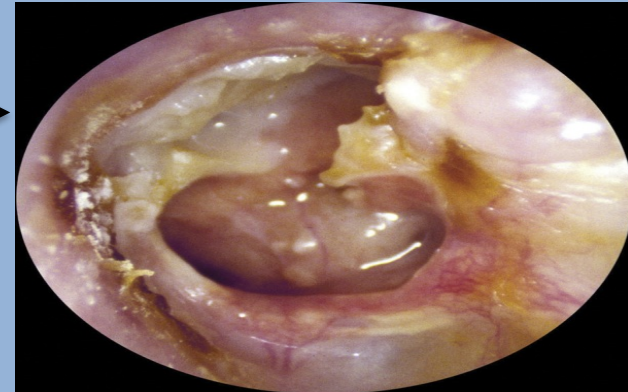
- Each of these theories accounts for a proportion of acquired cholesteatoma.
- Regardless of pathogenesis, cholesteatoma is prone to recurrent infections and they characteristically erodes ossicles and otic capsule.



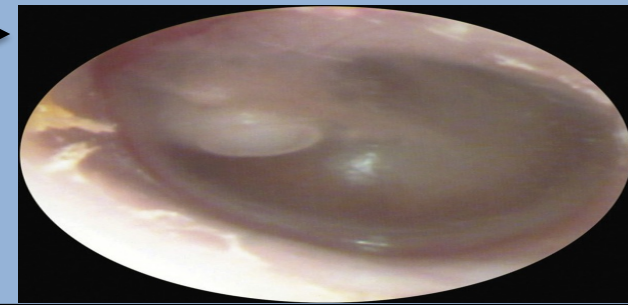
- A typical attic retraction cholesteatoma (**primary acquired cholesteatoma**).



- keratinizing epithelium has migrated through a perforation into the middle ear (**secondary acquired cholesteatoma**)



- Behind or within an intact tympanic membrane (**congenital cholesteatoma**)



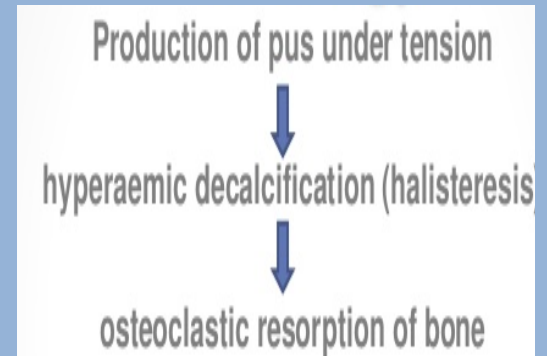
Bone erosion in Cholesteatoma & COM

1- Pressure necrosis (First theory proposed by Walsh in 1951)

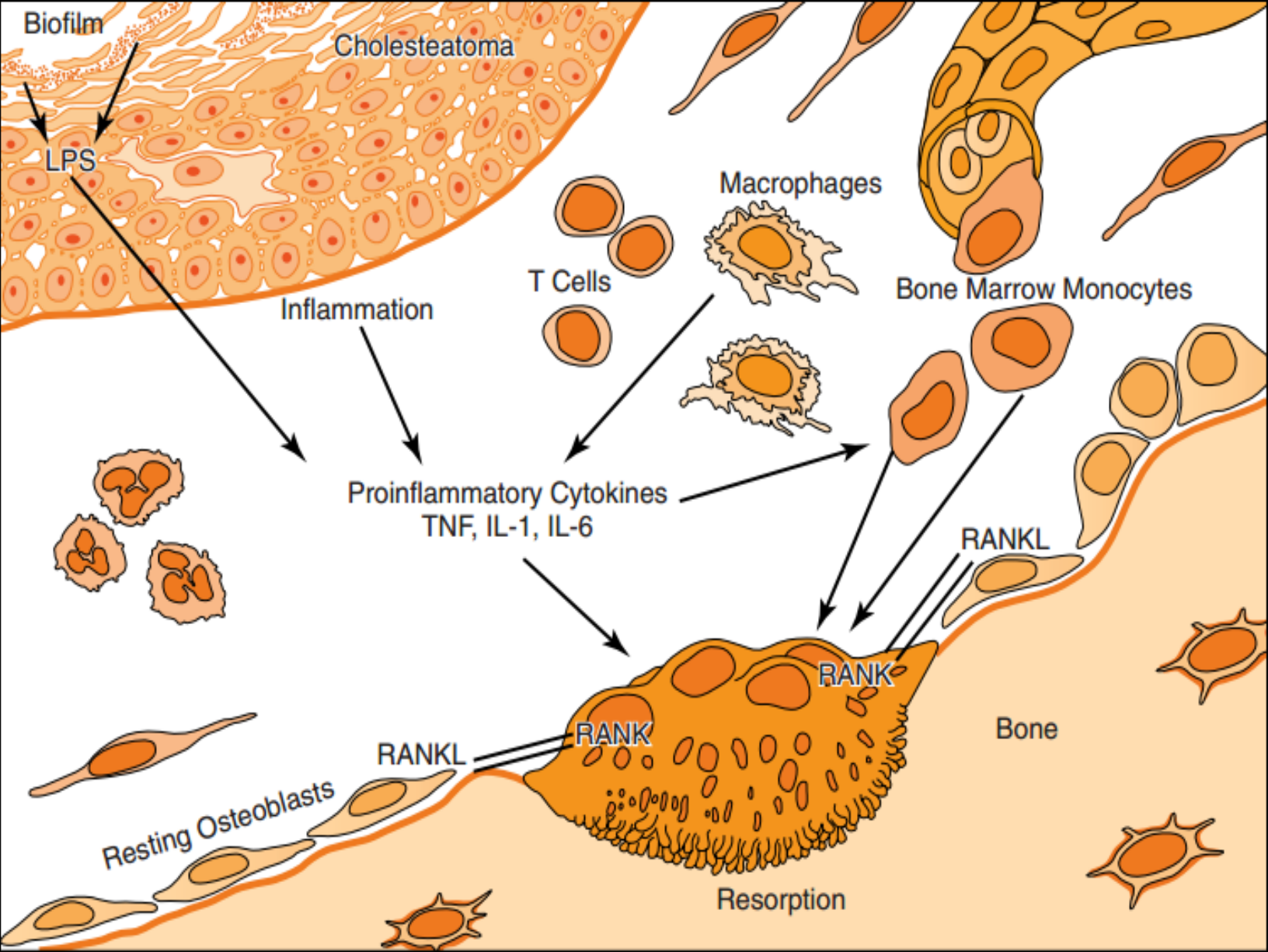
2- Hyperemic Decalcification: Vascularization of perimatrix is 5 folds of middle ear mucosa (Halisterisis)

3-Enzymatic induced dissolution of bone

(Acid phosphatase, collagenase, acid protease)



Inflammatory process within temporal bone simulate osteoclasts by many factors such as PG, LT, macrophages and lymphocytes



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Clinical Presentation

- Hearing impairment (80%)(usually CHL)
- Mucopurulent otorrhea (Active)
- Otalgia is uncommon
- Complications



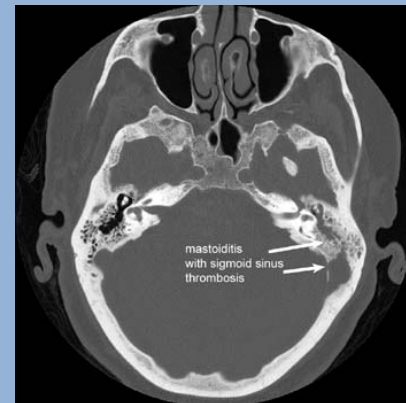
Complications

- Intratemporal complications:

- Petrositis(Gradenigo syndrome)
- Facial paralysis
- Labyrinthitis.

- Intracranial complications:

- lateral sinus thrombosis
- Meningitis
- intracranial abscess.



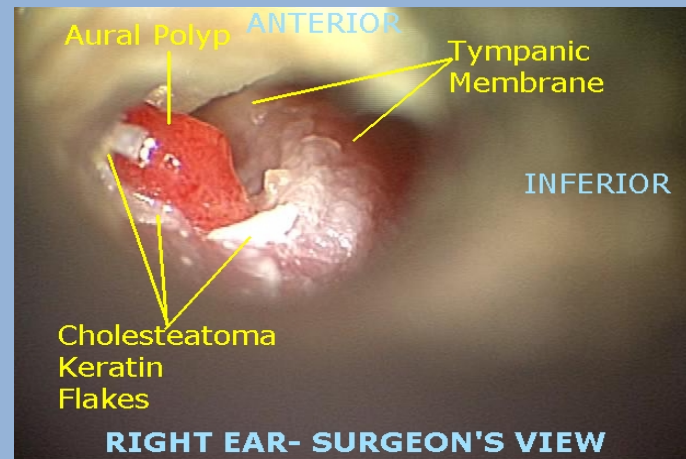
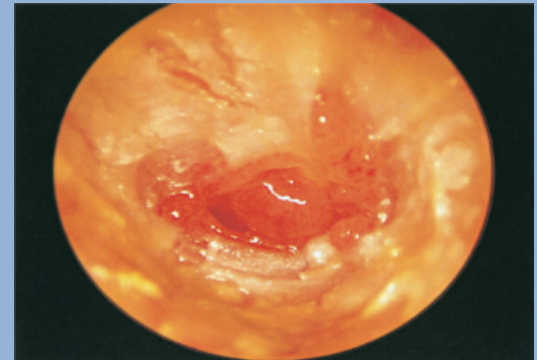
Physical Examination

Otoscopic exam is the **GOLD** standard for diagnosis



What to look for ?

- ❑ TM perforation(Central,marginal,attic)
- ❑ Active or inactive
- ❑ Granulation tissue
- ❑ Polyps
- ❑ Cholesteatoma
- ❑ Necrosis of long process of incus
- ❑ Complications
- ❑ Operation scars.



- Fistula test

Positive suggests erosion of inner ear, most commonly LSCC.



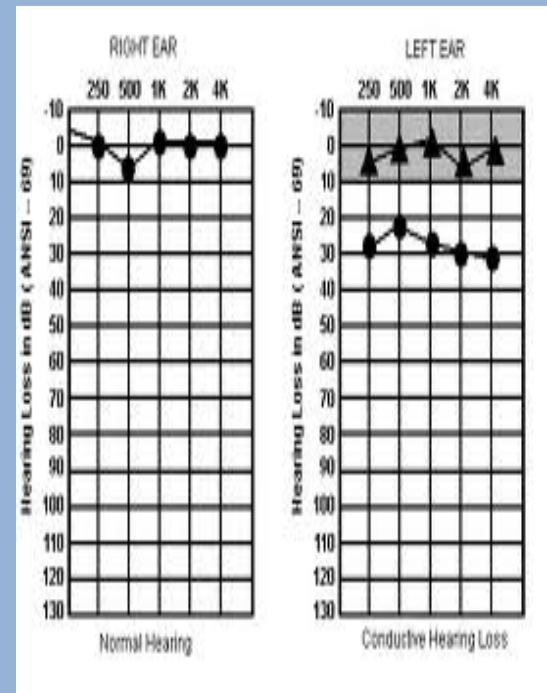
The basis of this test is to induce nystagmus by producing pressure changes in external canal which are then transmitted to labyrinth, stimulation of labyrinth produces nystagmus

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Diagnosis

- Culture and sensitivity.
- Audiology:
 - Pure tone audiometry:
 - Esp. if ear dry, prior to any surgical intervention.
 - Usually **CHL** (But may be SNHL)
 - Air bone gap depends on:
 - **Size of perforation**
 - **Erosion of ossicles**
 - **Significant granulation tissue around ossicles**

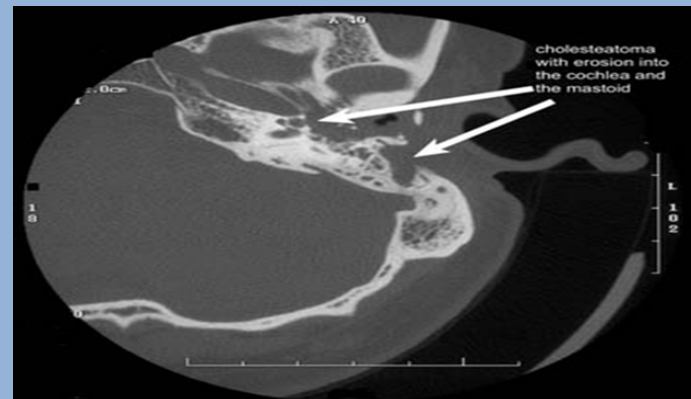
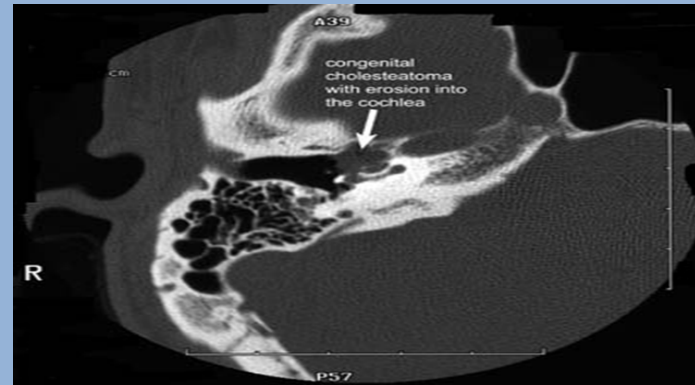


Radiology

- CT scan:

Fine cuts axial-coronal

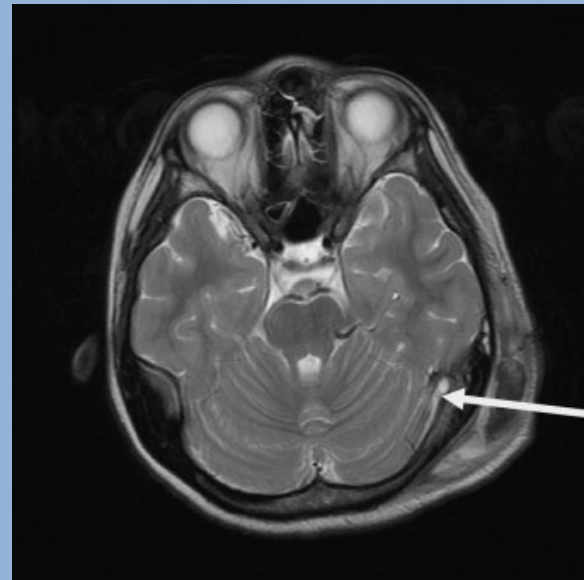
- Indications:
 - Unresponsive to treatment.
 - Cholesteatoma
 - Suspected complications
 - Prior to surgery



Radiology

- MRI:
- Intratemporal or intracranial complications.

- Useful:
 - Dural inflammation
 - Sigmoid sinus thrombosis
 - Labyrinthitis
 - Abscesses



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- **Treatment:** Medical
Surgical

Treatment

- Goals:

- Stop otorrhea
- Heal TM
- Eradicate current infection
- Prevent complications
- Prevent recurrence



Medical Treatment

- Aural toilet
- Topical antibiotics
- Granulation tissue control
- Systemic antibiotics

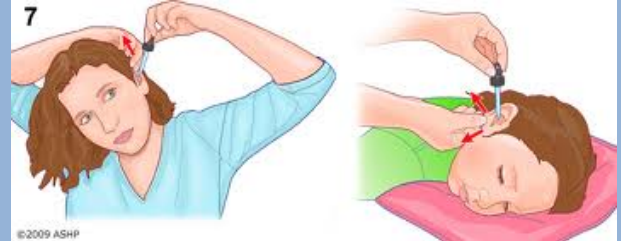
Aural toilet

- Critical process in management of COM
- Penetration of topical agents
- Using microscope
- Aural irrigation with 1.5% acetic acid to eliminate pseudomonas infection.



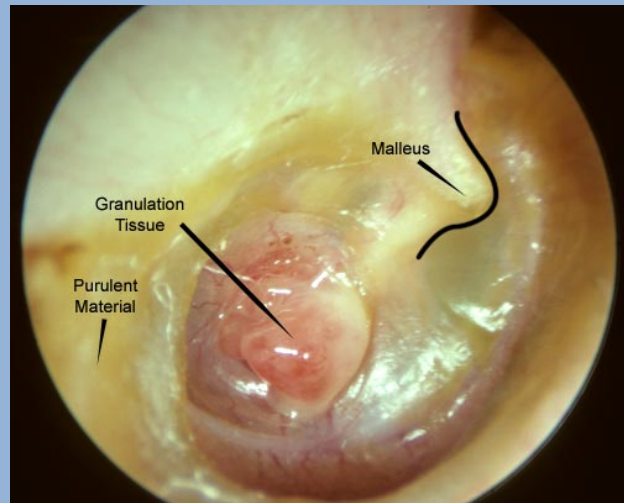
Topical Antibiotics

- First line of treatment of uncomplicated otorrhea.
- More effective than systemic Antibiotic:
 - Difficulty of systemic Antibiotic to penetrate
 - High concentration of topical antibiotics
 - Good safety profile
- Topical flouoroquinolones are preferred.
- Aminoglycosides are used with caution (vestibular dysfunction ,SNHL),but in most cases they don't penetrate inner ear (round window) .
- Topical steroids is considered if granulation tissue is present.



Granulation tissue control

- Prevents topical antimicrobial agents from penetrating the site of infection.
- Controlled by:
 - Antimicrobial drops
 - Topical steroids.
 - Cautery(microbipolar ,chemical)
 - Excision



Systemic Antibiotic

- Failure of topical treatment (due to failure of delivery more than resistance).
- Patient with high risk for complications
- Culture and sensitivity
- Aminoglycosides, piperacillin, ceftazidime, quinolones.
- Continued for at least 3-4 days after cessation of otorrhea.



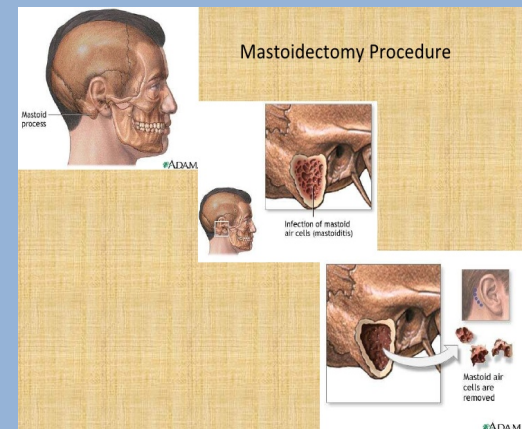
Surgical Treatment



- General indications:
 - Perforation that persists beyond 6 weeks
 - Otorrhea that persists for longer than 6 weeks despite antibiotic use
 - Cholesteatoma formation
 - Radiographic evidence of chronic mastoiditis
 - Conductive hearing loss.
- The principle aim of surgery is first to clear out the disease and only then if possible to reconstruct the patient's hearing.

Mastoidectomy

- Cortical mastoidectomy:
 - Canal wall up (Closed-cavity)
 - Canal wall down (Open cavity procedure)
- Radical mastoidectomy
- Modified radical mastoidectomy.



Thank You

