CHRONIC SUPPURATIVE OTITIS MEDIA

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Disclaimer

This presentation is for educational purposes only not for commercial activity.

- Persistant disease, Severe destruction

- It is characterized by:
  - Deafness
  - Ear discharge
  - T.m. perforation

Epidemiology

- Incidence-higher in developing countries because of poor socio-economic standards, poor nutrition and lack of health education.
- both sexes
- All age groups.
- In India, the overall prevalence is 46 and 16 persons per thousand in rural and urban population respectively.
- It is also the single most important cause of hearing impairment in rural population.

TYPES OF C.S.O.M.

- Tubo tympanic type — Safe
- Attico antral type — Unsafe

Differences between Tubotympanic and Attico-antral types of CSOM

	Tubotympanic or safe type	Atticoantral or unsafe type
Discharge	Profuse, mucoid, odourless	Scanty, purulent, foul-smelling
Perforation	Central	Attic or marginal
Granulations	Uncommon	Common
Polyp	Pale	Red and fleshy
Cholesteatoma	Absent	Present
Complications	Rare	Common
Audiogram	Mild to moderate conductive deafness	Conductive or mixed deafness

Aetiology

- The disease starts in childhood and is therefore common in that age group.

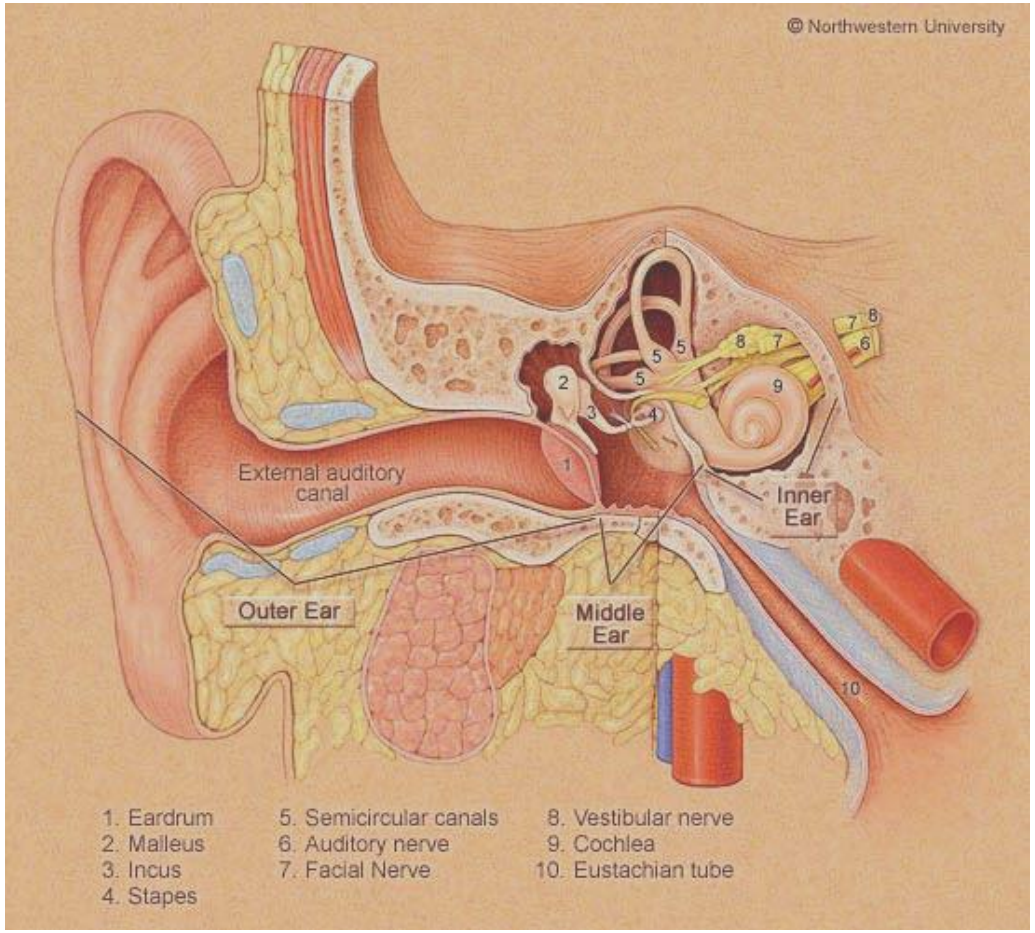
1. It is the seque la of *acute* otitis *media* usually following exanthematous fever and leaving behind a large central perforation. The perforation becomes permanent and permits repeated infection from the external ear. Also the middle ear mucosa gets exposed to the environment and sensitized to dust, pollen and other aeroallergens causing persistent otorrhoea.

2. Ascending infections *via* the eustachian tube. Infection from tonsils, adenoids and infected sinuses may be responsible for persistent or recurring otorrhoea.

3. Persistent mucoid otorrhoea is sometimes the result of allergy to ingestants such as milk, eggs, fish, etc.

TUBO TYMPANIC C.S.O.M

- Patency of Eustachian tube
- Nidus of infection in U.R.T.I.
- Immune status of patient
- Aerobic and Anaerobic



Pathology

- The tubotympanic disease remain localized to the mucosa and, that too, mostly to anteroinferior part of the middle ear cleft. Like any other chronic infection, the processes of healing and destruction go hand in hand and either of them may take advantage *over* the other, depending on the virulence of organism and resistance of the patient. Thus, acute exacerbations are not uncommon. The pathological changes seen in this type of CSOM are:

1. Perforation of pars tensa- It is a central perforation and its size and position varies.

2. Middle ear mucosa- It may be normal when disease is quiescent or inactive. It is edematous and velvety when disease is active.

3 . Polyp- A polyp is a smooth mass of edematous and inflamed mucosa which has protruded through a perforation and presents in the external canal. It is usually pale in contrast to pink, fleshy polyp seen in atticoantral disease.

Pathology

4. Ossicular chain- It is usually intact and mobile but may show some degree of necrosis, particularly of the long process of incus.

5. Tympanosclerosis- It is hyalinization and subsequent calcification of subepithelial connective tissue. It is seen in remnants of tympanic membrane or under the mucosa of middle ear. It is seen as white chalky deposit on the promontory, ossicles, joints, tendons and oval and round windows. Tympanosclerotic masses may interfere with the mobility of these structures and cause conductive deafness.

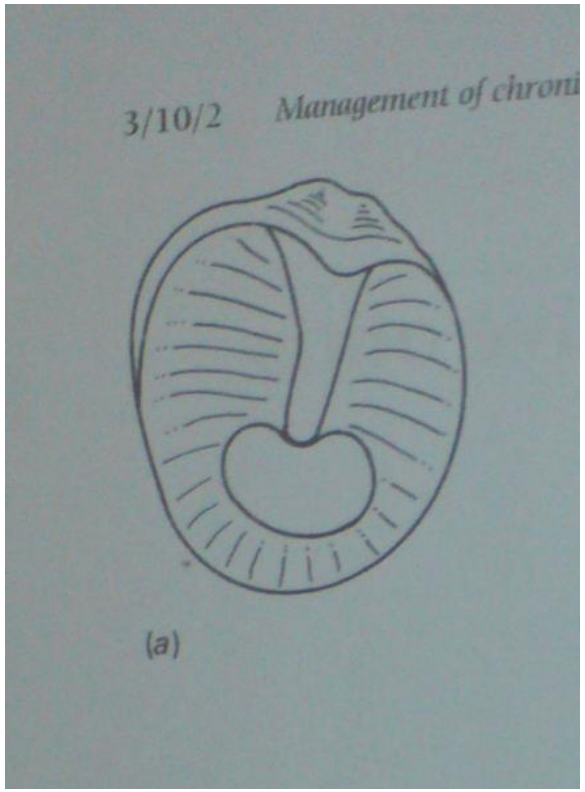
6. Fibrosis and adhesions- They are the result of healing process and may further impair mobility of ossicular chain or block the eustachian tube.

Bacteriology

- Pus culture shows both types of aerobic and anaerobic bacteria.
- Common aerobic organisms are- P. *aeruginosa, Proteus,* E. *coli* and *Staph. aureus.*
- Anaerobes include *Bacteroides fragilis* and anerobic *Streptococci.*

TUBO TYMPANIC C.S.O.M

- Deafness
- Discharge
- Central perforation



Central perforation
(anterior)

Central perforation
(medium sized)

Subtotal perforation

Total perforation with
destruction of even
the fibrous annulus

Attic perforation

Posterosuperior marginal
perforation

TYPES OF TUBO TYMPANIC C.S.O.M.

- Active Tubo Tympanic C.S.O.M.
- Inactive Tubo Tympanic C.S.O.M.

DIAGNOSIS
TUBO TYMPANIC C.S.O.M.

- History

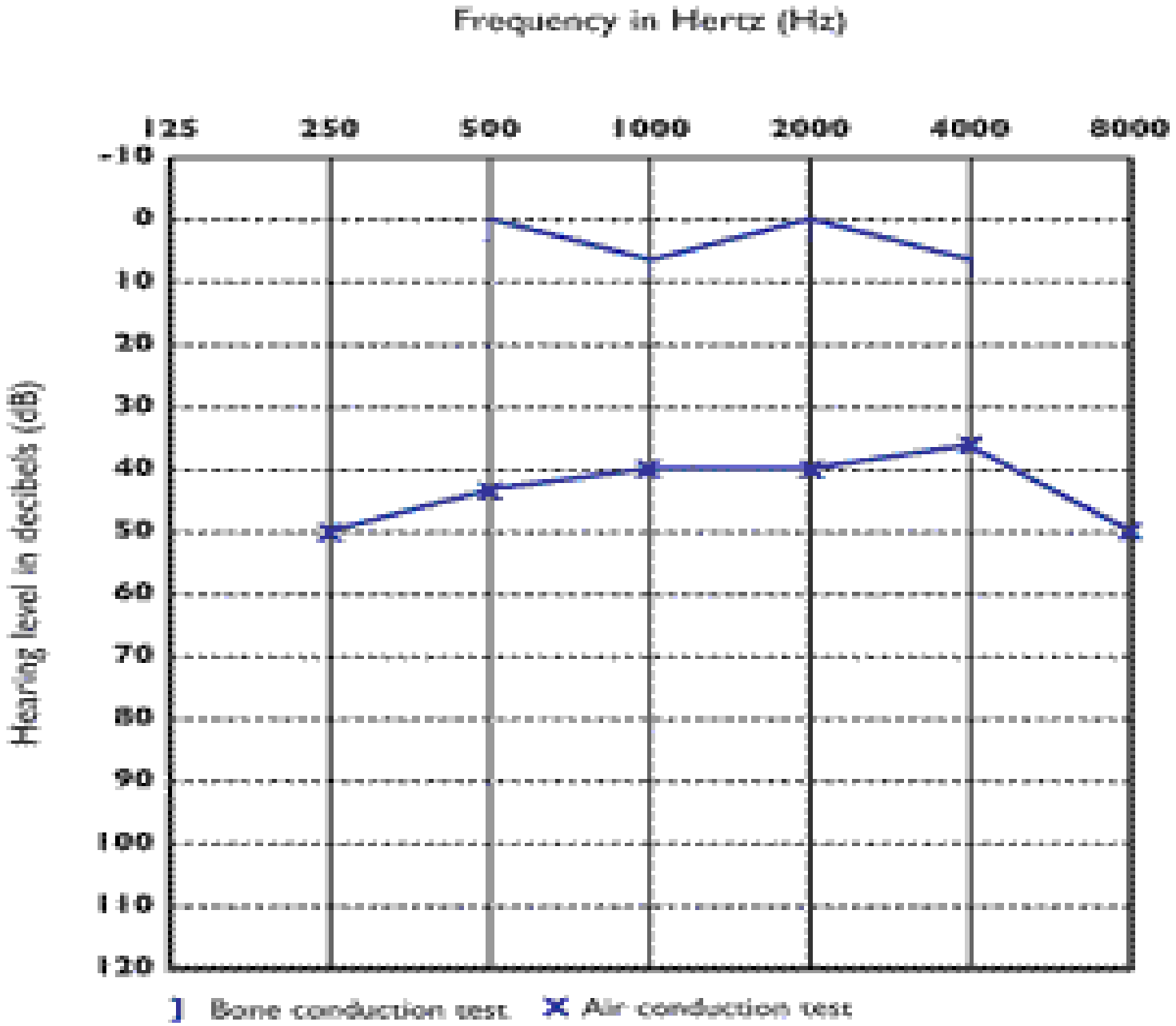
  Long standing, unilateral, bilateral, painless otorrhoea,
- Discharge intermittent, mucoid, mucopurulent,
- Non odorous
- Follow U.R.T.I. and entry of water.
- Deafness

Examination

- Main basis to assess activity, type, extent
- Inspection, otoscopy, E.U.M.
- Mucoid
- Perforation , central
- Pale mucosa
- Rarely polyp
- Pus for Culture & Sensitivity

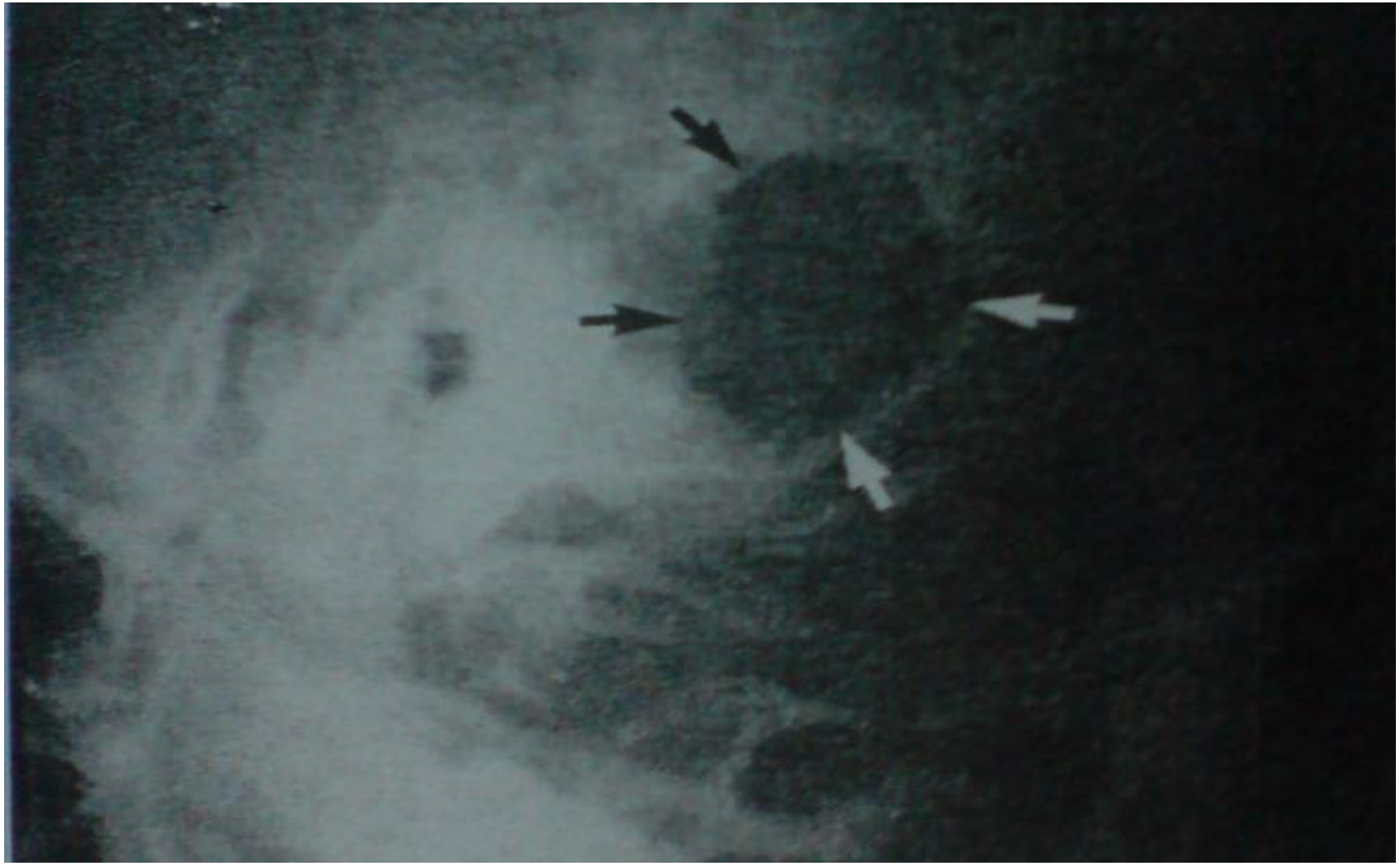
Audiological assessment

- Voice test
- Tuning fork test Rinne, Webers, A.B.C.
- Pure tone audiogram


Frequency in Hertz (Hz)
125
250
500
1000
2000
4000
8000
-10
0
10
20
30
40
50
60
70
80
90
100
110
120
Hearing level in decibels (dB)
] Bone conduction test
X Air conduction test

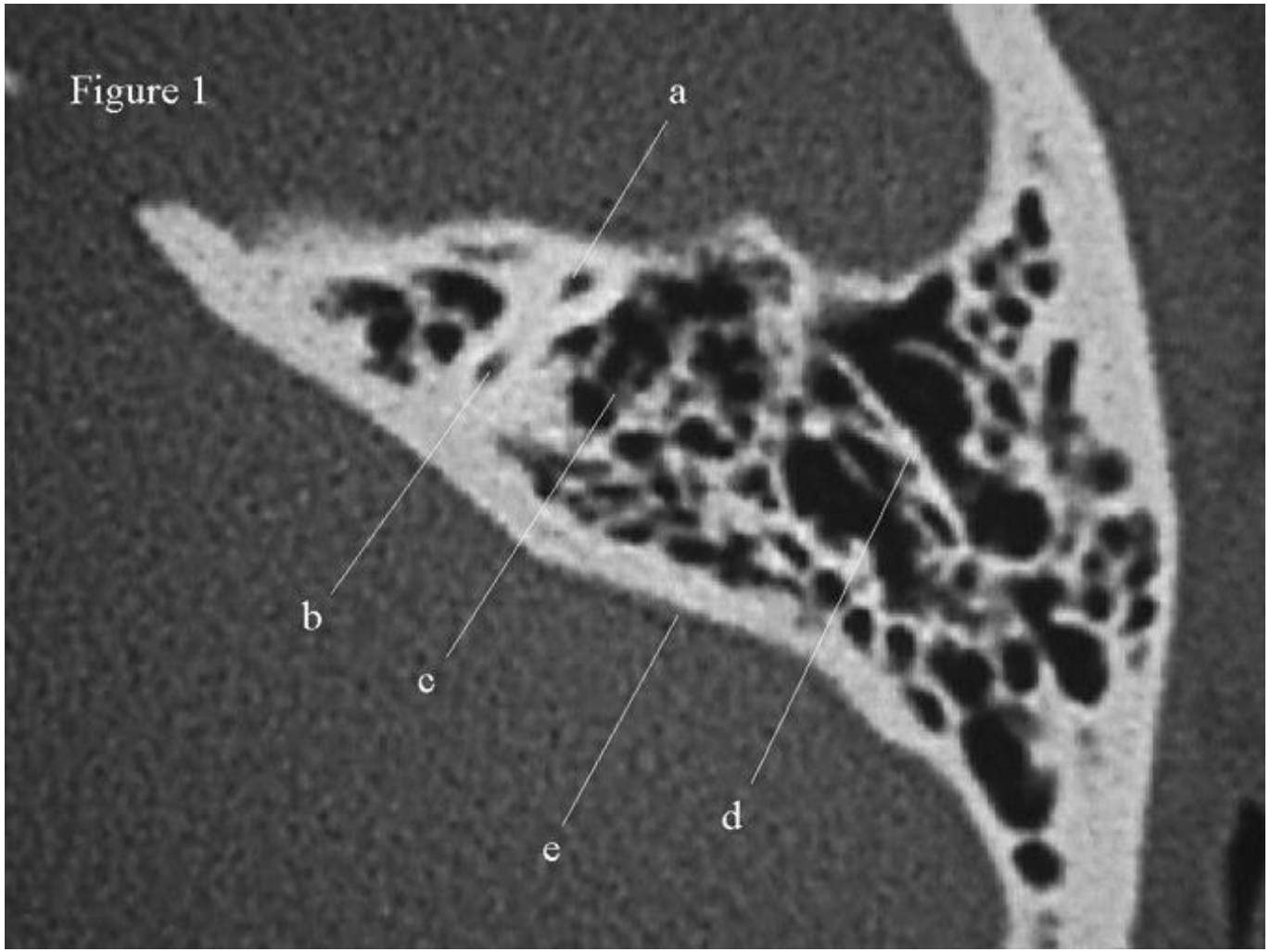
RADIOLOGICAL ASSESSMENT

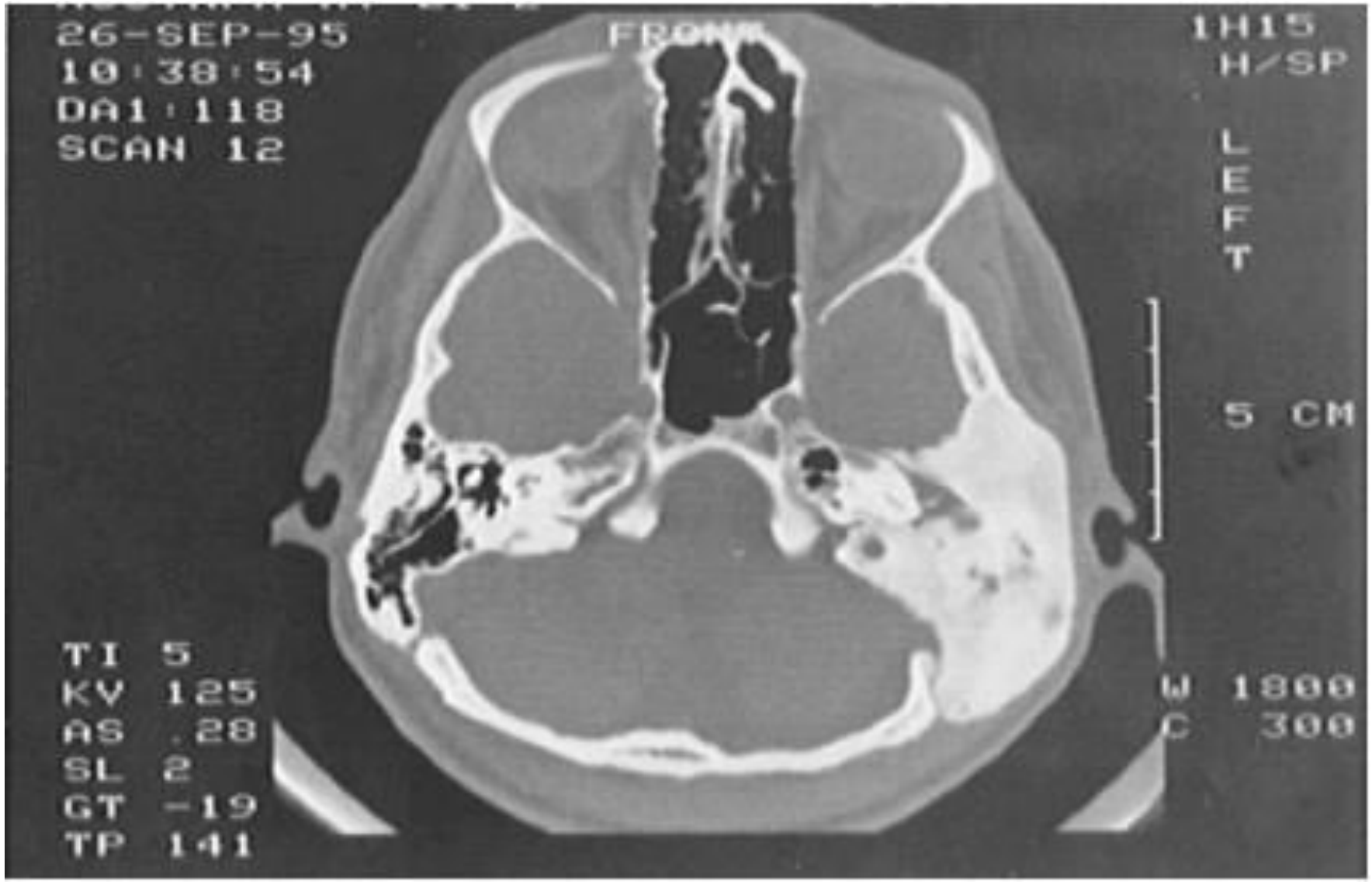
- X-rays mastoid



RADIOLOGICAL ASSESSMENT

- CT-scan temporal bone




26-SEP-95
10:38:54
DA1:118
SCAN 12
FRONT
1H15
H/SP
LEFT
5 CM
TI 5
KV 125
AS .28
SL 2
GT -19
TP 141
W 1800
C 300

treatment

- Aural toilet
  a. Cotton buds
  b. Suction and cleaning
- Antibiotics
  a. Topical antibiotics
  b. Systemic antibiotics

Surgical treatment

Precipitating disease

- Adenoid
- DNS
- Nasal polyps
- Aural polypectomy

Surgical Treatment

- Tympanoplasty- For functional reconstruction

ATTICO ANTRAL C.S.O.M.

- Cholesteatoma

Keratinizing Squamous Epithelium.

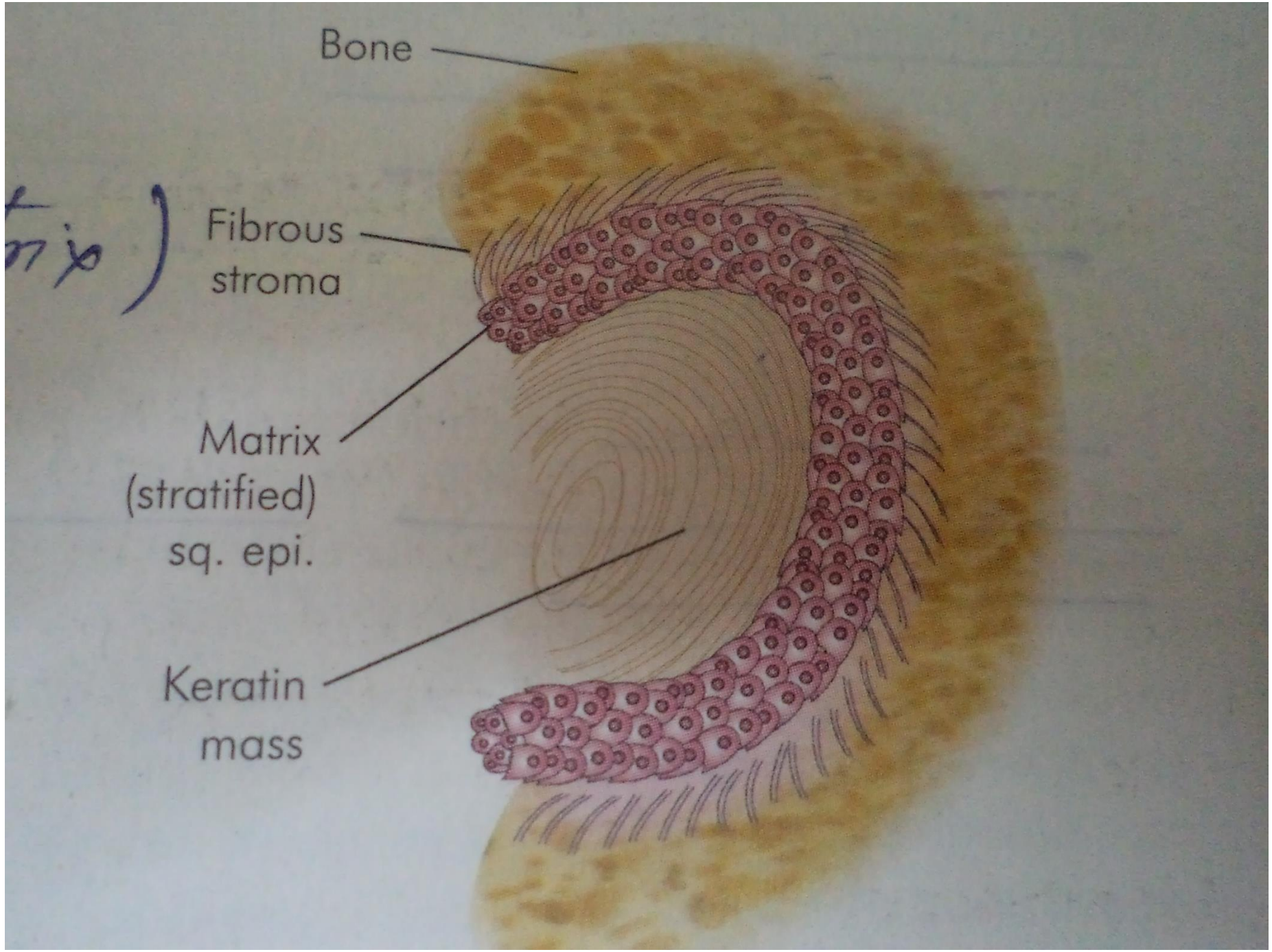
A small sac

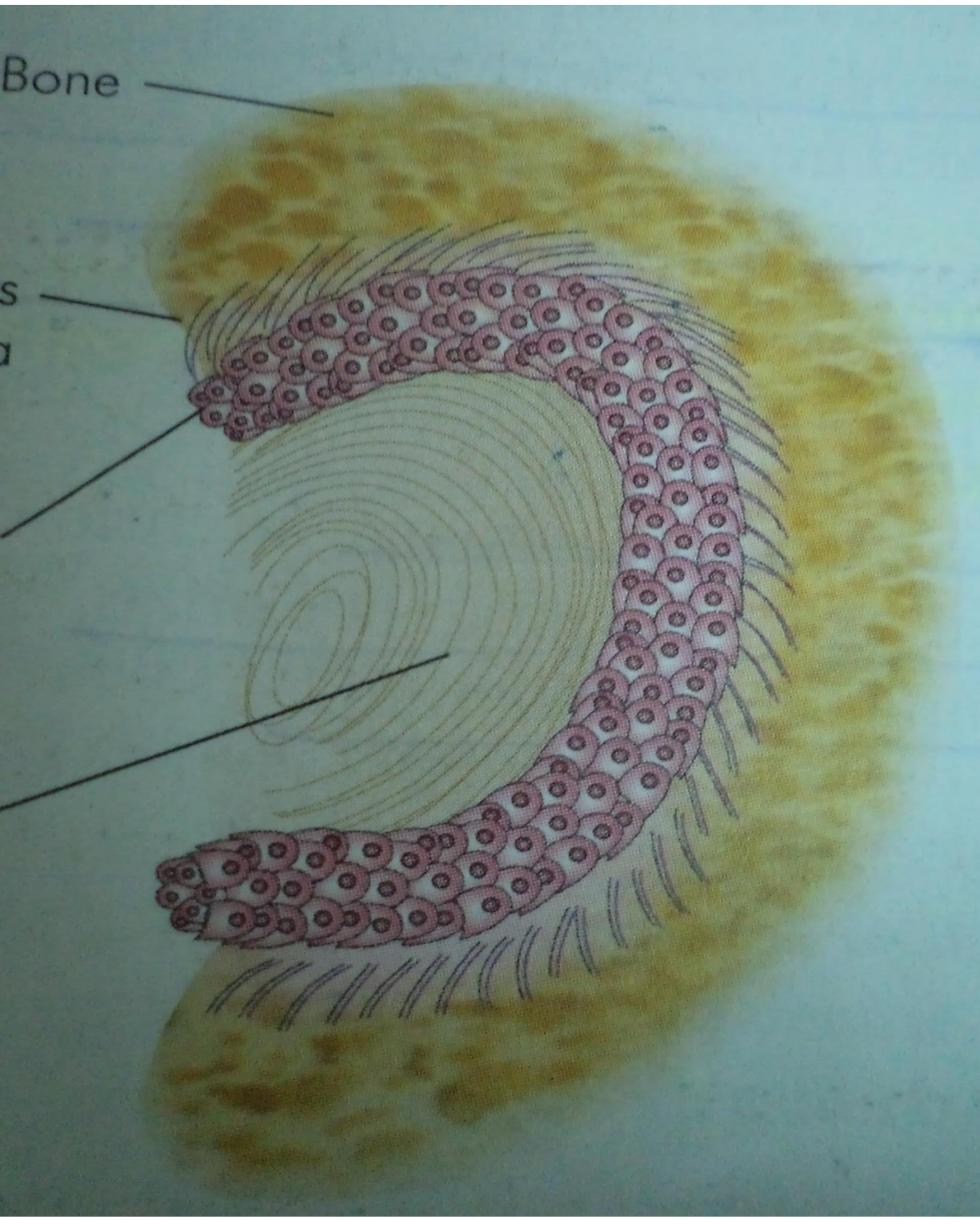
May involve whole middle ear cleft

cholesteatoma

- Keratinising squamous epithelium in the middle ear

- Ciliated columnar in ant & inferior part,
- Cuboidal in middle part & pavement like in the attic


Bone
Fibrous stroma
Matrix (stratified) sq. epi.
Keratin mass


Bone

HYPOTHESIS OF ORIGIN

- Presence of congenital cell rest
- Invagination theory (Wittmaack's theory)
- Basal cell hyperplasia (Ruedi's theory)
- Epithelial invasion (Habermann's theory)
- Metaplasia (Sade's Theory)

Congenital cholesteatoma

- It arises from the embryonic epidermal cell and rests in the middle ear cleft in the temporal bone. Congenital cholesteatoma occurs at three important sites: middle ear, petrous apex and the cerebellopontine angle, and produces symptomatology depending on its location.
- A middle ear congenital cholesteatoma presents as a white mass behind an intact tympanic membrane and causes conductive hearing loss. It may sometimes be discovered on routine examination of children or at the **time** of myringotomy.
- It may also spontaneously rupture through the tympanic membrane and present with a discharging ear indistinguishable from a case of chronic suppurative otitis media.

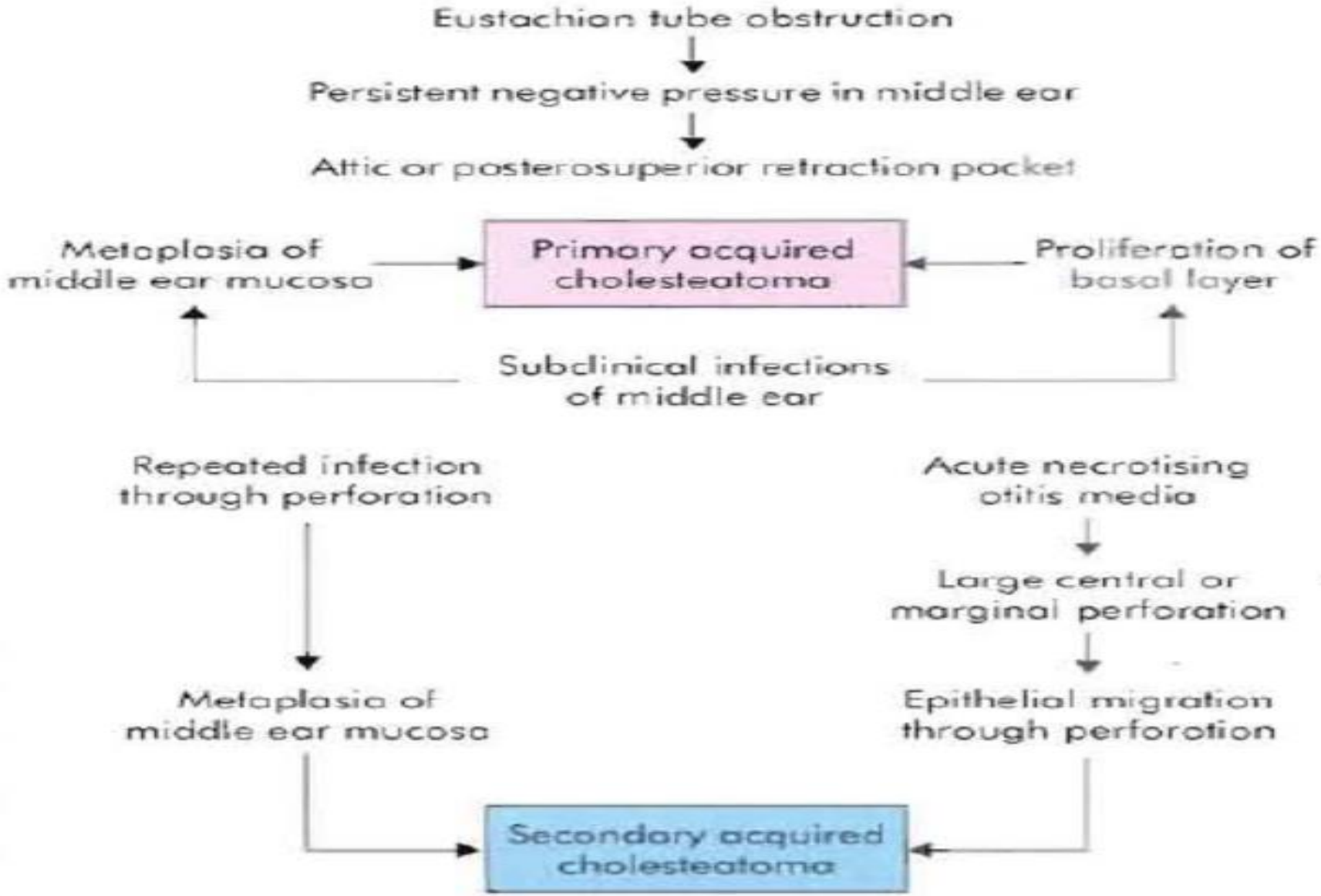
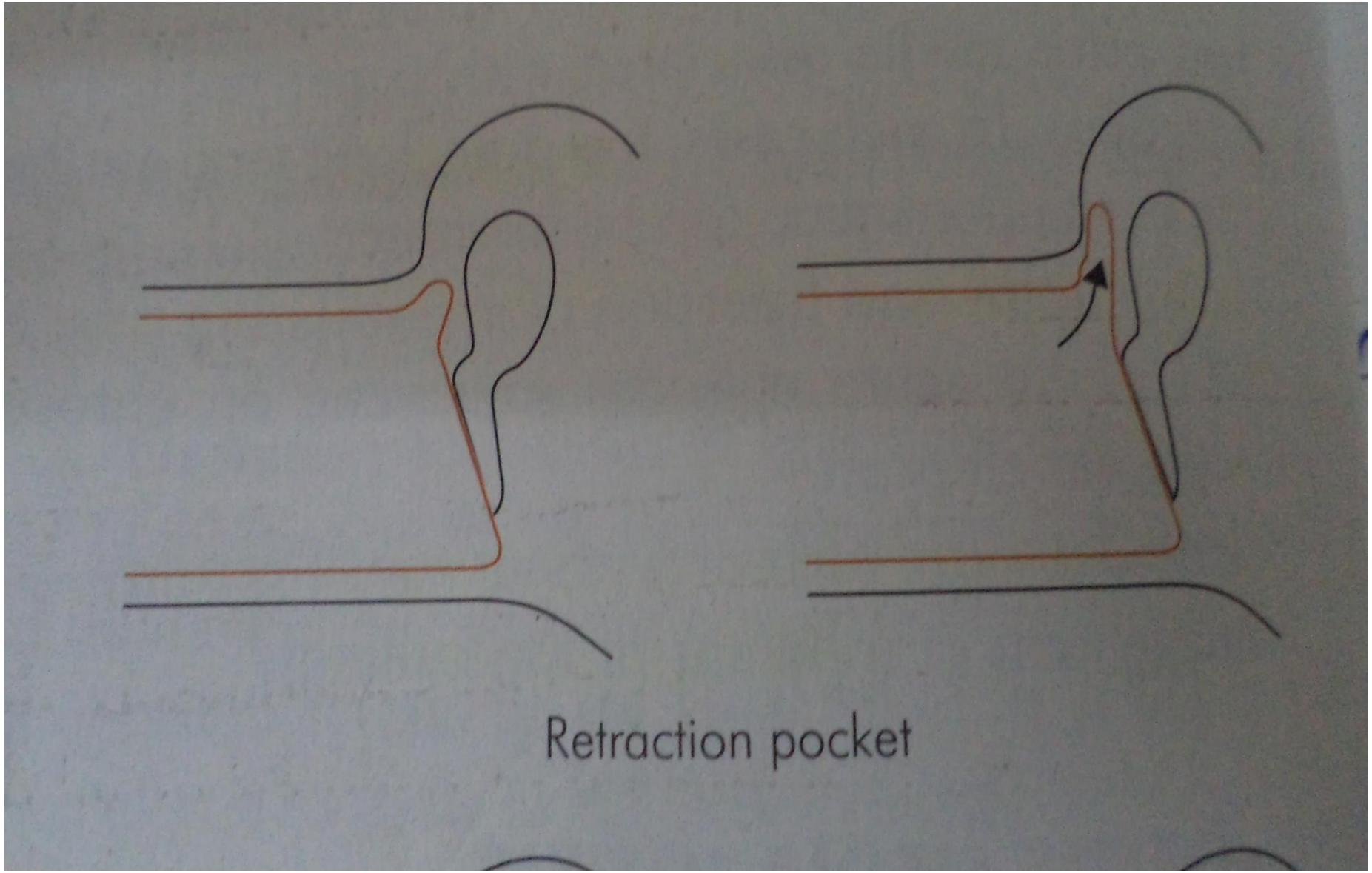
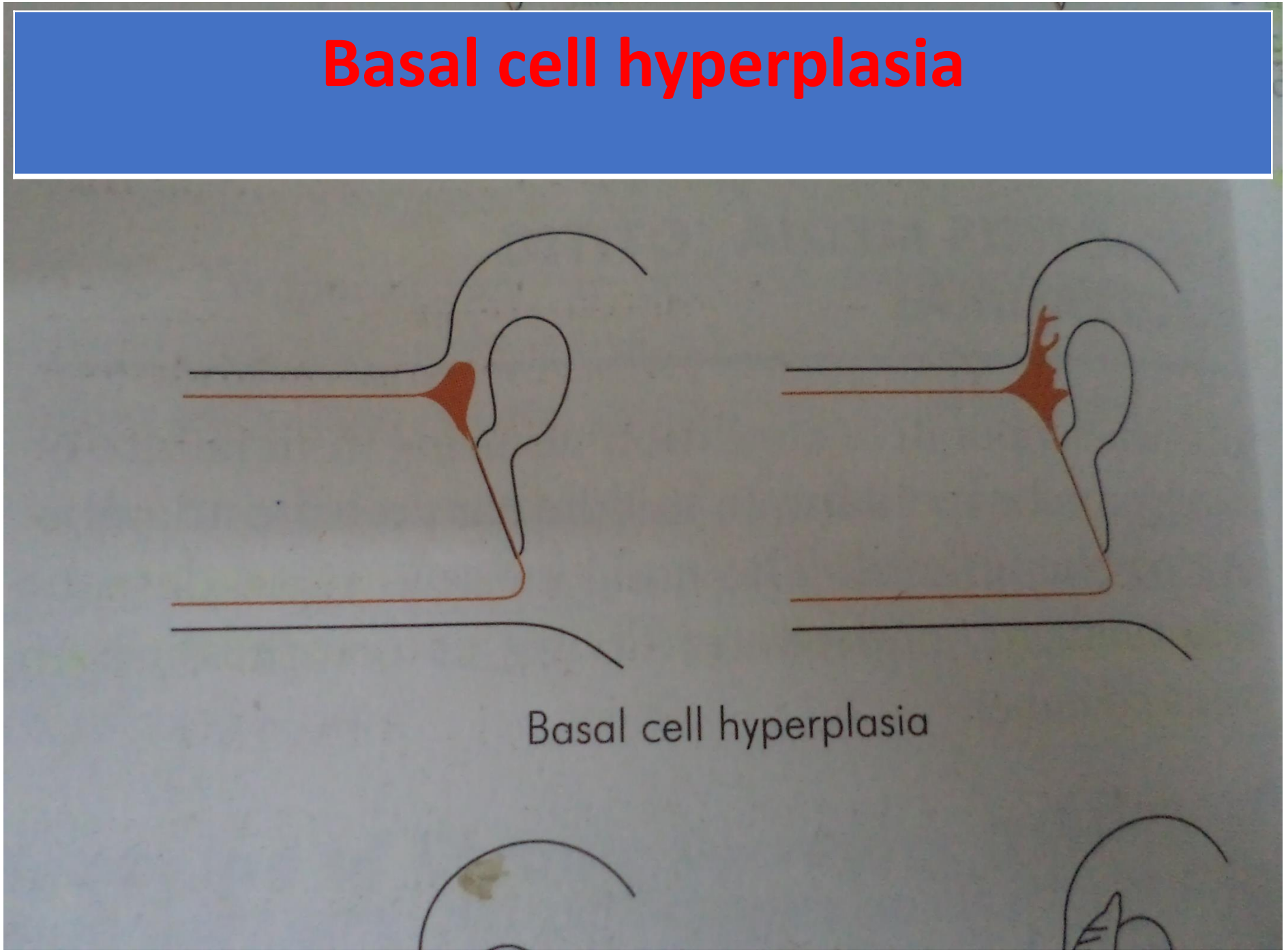


Fig. 11.3 Genesis of primary and secondary cholesteatoma.

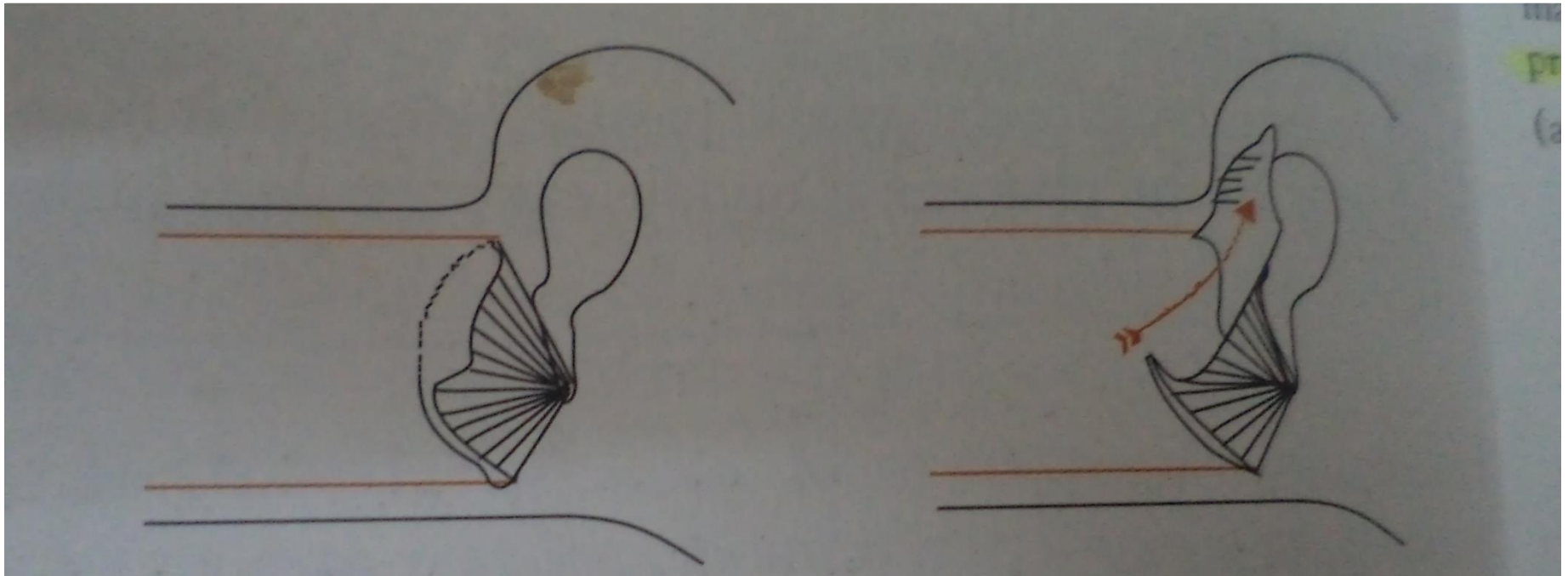


Retraction pocket

Basal cell hyperplasia



Epithelial invasion



Epithelial invasion through posterosuperior perforation

Fig. 11.2 Genesis of cholesteatoma.

Expansion of Cholesteatoma and destruction of Bone

- Once cholesteatoma enters the middle ear cleft, it invades the surrounding structures, first having followed the path of least resistance, and then by enzymatic bone destruction. An attic cholesteatoma may extend backwards into the aditus, antrum and mastoid; downwards into the mesotympanum; medially, it may surround the incus and/or head of malleus.
- Cholesteatoma has the property to destroy bone.
- It may cause destruction of ear ossicles, erosion of bony labyrinth, canal of facial nerve, sinus plate or tegmen tympani and thus cause several complications. Bone destruction by cholesteatoma has been attributed to various enzymes such as collagenase, acid phosphatase and proteolytic enzymes, liberated by ostoclasts and mononuclear inflammatory cells, seen in association with cholesteatoma. The earlier theory that cholesteatoma causes destruction of bone by pressure necrosis is not accepted these days .

Atticoantral Type of CSOM

- It involves posterosuperior part of middle ear cleft (attic, antrum and posterior tympanum and mastoid) and is associated with cholesteatoma, which, because of its bone eroding properties, causes risk of serious complications.

- For this reason, the disease is also called *unsafe* or *dangerous* type.

Pathology

- Atticoantral diseases is associated with the following pathological processes:

1. Cholesteatoma

2. Osteitis and granulation tissue. Osteitis involves outer attic wall and posterosuperior margin of the tympanic ring. A mass of granulation tissue surrounds the area of osteitis and may even fill the attic, antrum, posterior tympanum and mastoid. A fleshy red polypus may be seen filling the meatus.

3. Ossicular necrosis. It is common in atticoantral disease. Destruction may be limited to the long process of Incus or may also involve stapes superstructure, handle of malleus or the entire ossicular chain. Therefore, hearing loss is always greater than that of tubotympanic disease. Occasionally, the cholesteatoma bridges the gap caused by the destroyed ossicles, and hearing loss is not apparent.

4. Cholesterol granuloma. It is a mass of granulation tissue with foreign body giant cells surrounding the cholesterol crystals. It is a reaction to long-standing retention of secretions or haemorrhage, and mayor may not co-exist with cholesteatoma. When present in the mesotympanum, behind an intact drum, the latter appears blue.

Symptoms

- 1. Ear discharge. Usually scanty, but always foul smelling due to bone destruction. Discharge may be so scanty that the patient may not even be aware of it. Total duration of discharge from an ear which has been active recently should be viewed seriously, as perforation in these cases might be sealed by crusted discharge, inflamed and dry mucosa or a polyp, obstructing the free flow of discharge. Pus, in these cases, may find its way internally and cause complications.
- 2. Hearing loss. Hearing is normal when ossicular chain is intact or when cholesteatoma, having destroyed the ossicles, bridges the gap caused by destroyed ossicles(c*holesteatoma hearer).* Hearing loss is mostly conductive but sensorineural element may be added.
- 3. Bleeding. It may occur from granulations or the polyp when cleaning the ear.

Signs

1. Perforation. It is either attic or posterosuperior marginal type. A small attic perforation may be missed due to presence of a small amount of crusted discharge, Sometimes, the area of perforation is masked by a small granuloma.

2. Retraction pocket. An invagination of tympanic membrane is seen in the attic or posterosuperior area of pars tensa. Degree of retraction and invagination varies. In early stages, pocket is shallow and self-cleansing but later when pocket is deep, it accumulates keratin mass and gets infected.

3. Cholesteatoma. Pearly-white flakes of cholesteatoma can be sucked from the retraction pockets. Suction clearance and examination under operating microscope forms an important part of the clinical examination and assessment of any type of CSOM.

Treatment

- 1. Surgical. It is the mainstay of treatment. **Primary aim** is to *remove* the disease and render the ear safe, and **second in priority** is to preserve or reconstruct the hearing but never at the cost of the primary aim. Two types of surgical procedures are done to deal with cholesteatoma:

(a) *Canal wall down procedures*- They *leave* the mastoid cavity open into the external auditory canal so that the diseased area is fully exteriorized. The commonly performed operations for atticoantral disease are atticotomy, modified radical mastoidectomy and rarely, the radical mastoidectomy.

(b) *Canal wall up procedures*- Here disease is removed by combined approach through the meatus and mastoid but retaining the posterior bony meatal wall intact, thereby avoiding an open mastoid cavity. It *gives* dry ear and permits easy reconstruction of hearing mechanism. However, there is danger of leaving some cholesteatoma behind. Incidence of residual or recurrent cholesteatoma in these cases is *very* high and therefore long-term follow-up is essential. Some *even* advise routine re-exploration in all cases after 6 months or so. Canal wall up procedures are advised only in selected cases. In combined-approach or intact canal wall mastoidectomy, disease is removed both per-meatally and through cortical mastoidectomy and posterior tympanotomy, in which a window is created between the mastoid and middle ear, through the facial recess, to reach sinus tympani.

- 2. Reconstructive surgery. Hearing can be restored by myringoplasty or tympanoplasty. It can be done at the time of primary surgery or as a second stage procedure.
- Conservative treatment. It has a limited role in the management of cholesteatoma but can be tried in selected cases, when cholesteatoma is small and easily accessible to suction clearance under operating microscope. Repeated suction clearance and periodic check ups are essential.

It can also be tried out in elderly patients above 65 and those who are unfit for general anesthesia or those refusing surgery.

Other measures like aural toilet and dry ear precautions are also essential.

TUBERCULAR OTITIS MEDIA

- **Aetiology**

In most of the cases, infection is secondary to pulmonary tuberculosis; infection reaches the middle ear through eustachian tube. Sometimes, it is blood-borne from tubercular focus in the lungs, tonsils, cervical or mesenteric lymph nodes. Disease is mostly seen in *children* and *young adults.*

- **Pathology**

The process is slow and insidious. Tubercles appear in the submucosal layers of middle ear cleft and caseate. There is painless necrosis of tympanic membrane. Multiple perforations may form which coalesce to form a single large perforation. Middle ear and mastoid get filled with pale granulations. Caries of bone and ossicles may occur leading to complications. Mastoiditis, facial paralysis, postauricular fistula, osteomyelitis with formation of bony sequestrum and profound hearing loss are often seen in these cases.

- **Clinical Features**

1. Painless ear discharge. Earache is characteristically absent in cases of tubercular otitis media. Discharge is often foul-smelling because of the underlying bone destruction.

2. Perforation. Multiple perforations, 2 or 3 in number, are seen in pars tensa and form a classical sign of disease. These may coalesce into a single large perforation.

3. Hearing loss. There is severe hearing loss, out of proportion to symptoms. Mostly conductive, it may have sensorineural component due to involvement of labyrinth.

4. Facial paralysis. It is a common complication and may come unexpectedly. This may be the presenting feature in a child.

- **Diagnosis**

In the presence of secondary pyogenic infection, tubercular otitis media may be indistinguishable from chronic suppurative otitis media. Culture of ear discharge for tubercle bacilli, histopathological examination of granulations and X-ray chest, and other evidence of tuberculosis in the body help to confirm the diagnosis.

- **Treatment**

1. Systemic antitubercular therapy as being carried for primary disease.

2. Local treatment in the form of aural toilet, and control of secondary pyogenic infection.

3. Mastoid surgery indicated for complications. Healing is delayed in tuberculous cases. Wound break down and fistula formation are common. Reconstructive surgery of middle ear is delayed till antitubercular therapy has been completed.

SYPHILITIC OTITIS MEDIA

- Rare condition.
- Spirochetes reach middle ear through eustachian tube when syphilitic lesions are present in the nose or nasopharynx. Infection may also be blood-borne. Sensory end organs of the inner ear and their nerve is soon invaded by spirochetes leading to cholesteatoma and chronic suppurative otitis media, profound sensorineural hearing loss, tinnitus and vertigo.
- Bone necrosis and sequestrum formation are common, leading to foetid ear discharge. Secondary pyogenic infection may occur, giving a clinical picture very much like chronic suppurative otitis media.
- Definite diagnosis of syphilitic otitis media can only be made by specific treponemal antigen tests such as treponemal pallidum immobilization (TPI) test and fluorescent treponemal antibody absorption test (ITA-ABS). VDRL and Wasserman tests are nonspecific and may give false positive results.
- Treatment consists of antisyphilitic therapy with attention to aural toilet and control of secondary infection. Surgery may be required for removal of sequestra.