COMPLICATIONS OF COM

Complications of COM arise when infections spreads from middle ear cleft to structures normally separated by bone

FACTORS AFFECTING DEVELOPMENT OF COMPLICATIONS

1. Age – low immunity means children and elderly
2. Low socio economic status
3. Poor nutrition
4. Environment
5. Immunocompromised
6. Virulence of organism
7. Preformed pathways
8. Inadequate drainage i.e. eustachian tube block
9. Cholesteatoma

CLASSIFICATION

Intratemporal

- Mastoiditis
- Facial palsy
- Labynthitis
- Labrynthine fistula
- Petrositis
- Postauricular fistula
- Subperiosteal abscess

Extratemporal (Extracranial)

- Bezold's abscess
- Citelli's abscess
- Luc's abscess
- Zygomatic abscess
- Subcutaneous abscess
- Subperiosteal abscess

Intracranial

- Meningitis
- Brain abscess
- Subdural
- Extradural abscess
- Venous sinus thrombophlebitis
- Otitic hydrocephalus

Systemic Complications
- Septicemia
- Otogenic tetanus

**SEQULE OF OTITIS MEDIA**
- Perforation of TM
- Ossicular erosion
- Atelectasis and adhesive otitis media
- Tympanosclerosis
- Cholesteatoma
- CHL & SNHL
- Speech impairment
- Learning disabilities

**ROUTES OF ENTRY**
1. Bony erosion (cholesteatoma destruction, osteitis)
2. Retrograde Thrombophlebitis
3. **Anatomical pathway**: oval window, round window, internal auditory canal, suture line, cochlear & vestibular aqueduct
4. **Congenital bony defects**: facial canal, tegmen plate
5. **Acquired bony defects**: fracture, neoplasm, stapedectomy
6. **Peri-arteriolar space of Virchow-Robin**: spread into brain

**INTRATEMPORAL COMPLICATIONS:**

**ACUTE COALESCENT MASTOIDITIS**:
Acute infectious process of the mastoid bone with characteristic loss of trabecular bone (i.e. infection spreads from mucosa to bone)

**AETIOLOGY**:
- AOM
- Children
- High virulence of organism (beta haemolytic streptococci)
- Low immunity of host (diabetes, exanthematous fever, measles)
- 25% seen in sclerotic temporal bone with COM and cholesteatoma.

**PATHOLOGY**:
- Hyperemia and edema of mucoperiosteal lining of mastoid air cells \(\Rightarrow\) blocks narrow aditus \(\Rightarrow\) disrupts aeration \(\Rightarrow\) Mucus memb thickens and impaired ciliary function leads to impaired drainage through ET \(\Rightarrow\) Serous discharge becomes purulent as inflamm cells accumulate \(\Rightarrow\) Continuous inflammation, purulent discharge, hyperemia, edema leads to **venous stasis, localized acidosis and decalcification of bony septa** (hyperemic decalcification aka halisteresis) \(\Rightarrow\) destruction and coalescence of mastoid air cells \(\Rightarrow\) single cavity mastoid (empyema of mastoid)
- Pus may break through mastoid cortex leading to subperiosteal abscess.
- May burst open to form discharging fistula

**CLINICAL FEATURES**:
Symptoms:
- Pain behind ear
- Fever
- Persistent ear discharge

Signs:
- Persistent ear discharge (usually pulsatile ➔ Light house effect) reservoir sign
- Mastoid tenderness - "Ironed out feel" of mastoid due to oedema of periosteum, mastoid tip, post auricular groove, and root of zygoma
- Pinna pushed forwards and inferiorly
- Otoscopy ➔ TM perf with sagging of posterosuperior meatal wall (due to underlying osteitis)
- CHL

INVESTIGATIONs:
- Blood, ESR, Pus culture, Xray mastoid, CT scan

D/D:
- Meatal furunculosis
- Suppuration of mastoid lymph nodes
- Infected sebaceous cyst

TREATMENT:
- Antibiotics - iv for 2 to 3 weeks broad spectrum (vancomycin 10mg/kg/dose, cefotaxime, ceftazidime)
- If no TM perforation then myringotomy with grommet insertion
- Cortical mastoidectomy

COMPLICATIONS:
- Subperiosteal abscess
- Labyrinthitis
- Facial paralysis
- Petrositis
- Extradural
- Subdural
- Meningitis
- LS thrombophlebitis
- Otitic hydrocephalous
- Abscesses
  (Postauricular, bezold, luc, cetelli, zygomatic, subcutaneous, subperiosteal)
- Postauricular ➔ over Mc Evans triangle, pus travels along vascular channels of lamina cribrosa
- Zygomatic abscess ➔ posterior root of zygoma, infront and above pinna, upper eyelid also involved, pus deep to temporalis muscle
- Bezold abscess ➔ pus breaks through mastoid tip and presents as swelling in upper neck, May lie deep to SCM, follow Post belly of digastric and present between tip of mastoid and angle of jaw, reach parapharyngeal space or track down carotid vessels
**CLINICAL FEATURES:**

- Sudden onset, torticollis
- Purulent otorrhoea history

**D/D**

- Upper jugular lymphadenitis
- Parotid abscess
- Infected brachial cyst
- Parapharyngeal abscess
- Jugular vein thrombosis

**TREATMENT**

- Treat mastoiditis, antibiotics and open drainage of abscess

**LUCs ABSCESS:**

- Meatal abscess i.e. deep part of osseous meatus
- Citelli’s abscess : Behind mastoid more towards occipital bone. Some consider diagastric abscess as citelli abscess
- Parapharyngeal or retropharyngeal abscess : peritubal cell involvement leading to pus through eustachian tube.

**MASKED MASTOIDITIS:**

- Slow destruction of mastoid air cells without acute signs and symptoms of acute mastoiditis.
- No Pain, no fever, no discharge, no mastoid swelling but mastoidectomy may show extensive destruction of air cells with granulation tissue
- It is not surprising to note erosion of tegmen tympani and sinus plate with extradural and perisinus abscess.

**AETIOLOGY**

- Inadequate antibiotic therapy

**PATHOLOGY**

- Focal area of persistent infection somewhere in mastoid does not respond to antibiotics

**CLINICAL FEATURES**

- Often patient is child, persistent hearing loss and mild pain behind ear
- TM has loss of translucency and appears thick
- Mastoid tenderness present

**INVESIGATIONS:**

- PTA shows CHL
- X ray reveals clouding of air cells

**TREATMENT:**
Cortical mastoidectomy with antibiotics

**PETROUS APICITIS :**

**DEFINITION :**
- Spread of infection to petrous part of temporal bone.
- Petrous apex is a truncated pyramid that is portion of temporal bone medial to inner ear labyrinth
- **Petrous apex bisected by Internal auditory canal in coronal plane Anteriorly into peritubal area and posteriorly into perilabyrinthine area.**

**PATHOPHYSIOLOGY :**
- Can be **pneumatised, diploic or sclerotic.**
- 2 cell tracts recognized for pneumatisation :
  - One ➔ from mastoid ➔ behind and above bony labyrinth ➔ petrous apex
  - Second ➔ Anteroinferior tract starting at hypotympanum near Eustachian tube ➔ around cochlea ➔ reach petrous apex.
- Can be totally asymptomatic to coalescence, **abscess formation.**
- There can be formation of granulation tissue with chronic bone erosion.
- Rarely there can be extension of cholesteatoma into the apex.
- Can involve **trigeminal ganglion and CN VI**

**ORGANISMS INVOLVED :**
- Pneumococcus, H.Influenza and staphylococcus aureus are main organisms causing Petrositis.

**CLINICAL FEATURES :**
- Gradinegos Syndrome (Otorrhoea, eye pain and 6th palsy in **Dorellos canal**)
- Retro orbital pain is due to irritation of trigeminal ganglion in **(Meckel's cave)**
- This triad can also indicate the development petrous apex extra dural abscess.
- Persistent ear discharge even after cortical or radical mastoidectomy.
- Fever, headache vomiting and neck rigidity.
- 7th and 8th nerve involvement can also occur.
- Facial paralysis.
- Recurrent vertigo.

**DIAGNOSIS**
- CT and MRI (to differentiate between diploic marrow containing apex from fluid or pus and also cholesteatoma), Tc99 scan for diagnosis but not follow-up.

**TREATMENT :**
- Because of anatomical complexity and need to work around carotid artery, petrous apex cell disease cannot be excised.
- **IV antibiotics** (broad spectrum)
- **Surgery** : in case of presence of abscess, necrotic bone, persistent infection despite medical therapy
- Cortical, modified radical or radical mastoidectomies required if not done already and discharging tract followed and diseased bone curetted.
Even partial apicetomy can lead antibiotics and local immunity to take control.

**APPROACHES:**
- Sub cochlear (farrier's), Infraabrynthine (Dearmin & Farrier), Retrolabrynthine (Thornwalds), Subarcuate (freckners's), Middle cranial fossa (eagleton's), Peritubal (Ramadier's & Almoor's)
- Best route is infracochelear.
- Hearing destroying transcochlear and translabyrinthine approach
- **EAGLETON’S MIDDLE CRANIAL FOSSA APPROACH**
- **FRENCKNER'S SUBARCUATE APPROACH.** (through arch of superior semicircular canal)
- **THORNWALDT’S RETRO-LABYRINTHINE APPROACH**
- **DEARMIN & FARRIOR’S INFRA-LABYRINTHINE APPROACH**
- **FARRIER’S HYPOXYMPANIC SUB-COCHLEAR APPROACH**
- **LEMPERT RAMADIER’S PERI-TUBAL APPROACH**
- **KOPETSKY ALMOOR’S PERI-TUBAL APPROACH**

- **** GLASSCOCK’s triangle ➔ **medially** GSPN, **base** mandibular division of trigeminal and **laterally** by line from the foramen spinosum to arcuate eminence.
- **Kawase's triangle:** **Laterally** GSPN, **Medially** petrous ridge(superior petrosal sinus) and **base** arcuate eminence

**LABYRINTHITIS:**
- Definition
- Types:
  - Three types ➔ Diffuse serous, diffuse suppurative and circumscribed or localized.

**SUPPURATIVE**
- Diffuse pyogenic infection of the labyrinth with diffuse loss of vestibular and cochlear function.
- Direct Spread of infection from middle ear to labyrinth from pathological or surgically formed fistula.

**AETIOLOGY:**
- Usually follows serous labyrinthitis

**ORGANISMS:**
- Pseudomonas, H Influenza and Staph aureus

**PATHOLOGY:** 4 stages

**Serous (irritative stage)**
- **Sero fibrinous exudates rich in specific Ig’s** is the earliest reaction of inner ear to the pathogens

**Purulent stage (manifest stage)**
Intra and extra cellular organisms fill the perilymphatic space with Serofibrinous Exudate with increased protein content.
Also vasodilatation, thrombosis, exudation from modiolar vessels, necrotic changes of end organs follow.

**Fibrous stage**

- Fibroblastic proliferation within perilymphatic spaces. Fibroblasts + small capillaries = Granulation tissue.
- Usually seen 2 weeks following infection
- Pus cells can be seen

**Osseous stage**

- First seen in anterior labyrinth in the perilymphatic spaces in basal cochlear turn.
- Disease can be aborted in serous stage but once purulent stage is reached disease progresses.

**CLINICAL FEATURES:**

- Serous Stage ➔ TVS, nausea vomiting, spontaneous nystagmus towards affected ear until generalised suppuration and destruction of labyrinth takes place at which the nystagmus changes to normal ear. Usually change occurs in a day
- Purulent stage ➔ Violently ill, profound hearing loss, incapacitating vertigo, nausea vomiting, spontaneous nystagmus towards normal ear, any movement aggravates vertigo so patient lies still with affected ear uppermost. Fever usually present
- Fibrous Stage ➔ few weeks to few months, complete deafness, vertigo disappears but positional vertigo may persist.
- Healed (Osseous) stage ➔ 2 to 3 months later. Many years required for complete ossification of membranous labyrinth. Complete loss of auditory and vestibular functions, Occasionally a positional dizziness.

**INVESTIGSTIONS:**

- MRI = T1 enhancement of contrast
- CT for possible site of labyrinthine erosion.

**TREATMENT:**

- IV antibiotics
- Labyrinthine sedatives = Prochlorperazine and Antiemetics
- Proper rest and hydration
- Middle ear disease control with mastoid exploration (Should be delayed until initial vertigo has subsided)
- Cawthorne Cooksey vestibular exercises (eye and head exercises)

**SEROUS LABRYNTHITIS:**

- Sterile inflammation of inner ear secondary to chemical and toxic irritation of membranous labyrinth which are released by organisms and not by direct invasion.
**PATHOLOGY**

- Perilymphatic and to some extent endolymphatic space is filled with eosinophilic seroproteinoid, fibrinous, or fine granular material hindering sound transmission. Endolymphatic hydrops results due to impaired absorption of proteinoid material and clogging of endolymphatic duct and sac.
  - 2 forms
    - 1. Insidious form ➔
      - Minimal dose of bacterial toxins across oval and round windows. Remains silent, subtle cochlear changes in form Serofibrinous exudates in basal turn. High tone SNHL.
    - 2. Toxic Form ➔
      - Large amount of bacterial toxins. Vertigo and nystagmus to affected ear with SNHL.

**TREATMENT**

- Intensive antibiotic therapy
- Steroid therapy
- Surgery for middle ear and mastoid pathology

**LABYRINTHINE FISTULA (Circumscribed labyrinthitis):**

- MC complication of COM with cholesteatoma
- Loss of enchondral bone over SCC without loss of perilymph.
- MC - Arch of Lateral SCC eroded
- Occurs in both Mucosal and Squamosal varieties
- Other areas of fistula include superior and posterior SCC and cochlea
- Surgically induced – 10 – 15%

**PATHOPHYSIOLOGY:**

- Inflammatory mediators (in absence of cholesteatoma)
- Pressure from cholesteatoma itself or active mediators from matrix
- Provokes osteitis in labyrinth with osteoclastic predominance

**DORNOFFER AND MILEWSKI classification:**

- Type 1 = fistula with bone erosion but intact endosteum
- Type 2a = endosteum breached but perilymphatic space preserved
- Type 2b = perilymph voided
- Type 3 = Membranous labyrinth and endolymph also disrupted.
- First Demineralisation ➔ bone keeps becoming thinner ➔ BLUE LINE parallel to SCC lumen

**DIANOSIS ➔**

**Clinical features:**

- Short lived episodic vertigo
- Positive fistula test (nystagmus to affected side)
- LH SR PV (Lateral SCC causes Horizontal, Superior SCC causes rotational deviation, Posterior SCC causes Vertical nystagmus)
- CT scan is helpful to diagnose fistula
Definitive diagnosis is made only intraoperatively

**TREATMENT:**

- Antivertigo and antiemetic
- Surgery is treatment of choice
- Tympanomastoidectomy for treatment of cholesteatoma.
- 2 schools of thoughts →
  - Canal wall down → remove chols → leave fistula covered with matrix exteriorising it into the cavity (as complete removal of matrix may lead to SNHL).
  - Second is to completely remove chole and repair bony defect.
- 2b and 3 have worse outcomes. Intraoperative steroids help.
- Small fistulas can be removed safely
- Large fistulas need to be covered with bone plate or fascia.
- Iatrogenic SNHL very common with cochlear fistulas

**Facial Palsy:**

- Within 1st wk: due to nerve sheath edema
- After 2 wks: due to bone erosion
- Lower motor neuron palsy
- Common in tubercular otitis media

Via Tegmen Plate → Temporal abscess

Via Trautmann’s triangle → Cerebellar abscess

**Temporal Lobe**

- **Nominal aphasia**
- **Quadratic homonymous**
  - hemianopia (C/L)
- **Epileptic seizures**
- **Pupillary dilatation**
- **Hallucination (smell & taste)**
- **C/L hemiplegia**

**Cerebellum**

- I/L nystagmus
- I/L weakness
- I/L hypotonia
- I/L ataxia
- Intention tremor
- Dysdiadochokinesia
BRAIN ABSCESS:

**DEFINITION:**

- Bacteria or fungi infect brain and infected brain cells, WBCs, live and dead bacteria fungi, collect in area of brain and tissue forms around this area forming a mass
- Mass puts pressure on delicate brain tissue.
- Infected material can block blood vessels of brain also

**ROUTES OF SPREAD:**

- Direct via tegmen tympani
- Retrograde thrombophlebitis (usually extradural abscess)
- Cerebellar abscess can develop through trautmans triangle
- Associated with ED abscess, sigmoid sinus thrombosis, perisinus abscess or labyrinthitis.

**AETIOLOGY:**

- 50% in adults and 25% in children are otogenic in origin
- Median age 30 to 45 yrs
- Children - AOM and adults follows COM and Cholesteatoma
- Cerebral abscess twice as common as cerebellar abscess
- ORGANISMS: Proteus, Pseudomonas, H Influenza, S. Haemolyticus, peptostreptococcus and bacteroides

**PATHOLOGY:**

- Brain abscess has 4 stages (Invasion ➔ Localization ➔ Enlargement ➔ termination)

**Stage of Invasion:**

- Headache, fever, malaise and drowsiness.

**Stage of Localization:**

- Capsule formation with no symptoms
- May last for several weeks

**Stage of enlargement:**

- Abscess begins to enlarge and a zone of oedema develops around abscess and is responsible for aggravation of symptoms.
- Raised ICT with focal signs and symptoms

**Stage of termination:**

- Can result in fatal meningitis due to rupture of abscess in white matter

**CLINICAL FEATURES:**
Asso with labrynthitis, perisinus abscess, extradural abscess, sinus thrombosis, meningitis

MC presentation is after 2 weeks

Can be devided into:

Those due to raised ICT

Those due to area of brain affected (localising features)

In children head may enlarge due to non fusion of sutures

ICT raise leads to Headache, nausea, vomiting projectile more so in cerebellar lesions, altered consciousness, Papilloedema usually after 2 to 3 weeks, slow pulse and subnormal temperature.

Meningitis if present may lead to neck stiffness

TEMPORAL LOBE ABSCESS ➔ MC

Nominal aphasia (if dominant lobe affected), focal fits, C/L upper quadrantic homonymous hemianopia, C/L motor paralysis, transtentorial herniation leads to pupillary changes and oculomotor palsy.

CEREBELLAR ABSCESS ➔

Occipital Headache, spontaneous nystagmus, Ipsilateral hypotonia, weakness, Ipsi ataxia, past pointing, intention tremor, titubition, dysdiadokokinesis.

Suddenly increasing headache shows rupture of abscess

Investigations:

Skull X rays (midline shift, if pineal gland calcified, reveals gas in abscess cavity).

Xray mastoid or CT of temporal bone for ear disease

Lumbar puncture (Increase proteins, normal glucose), WBC, polymorphs and lymphocytes

TREATMENT:

Head up, mannitol, lumbar drain, hyperventilation

IV antibiotics

3rd generation Cephalosporins (Ceftriaxone 2gm BD for 2 weeks)DO gm DO baar for DO hafta

Ceftazidime (1gm iv TID) (Pseudomonas)

4th generation Cefipime 2gm iv BD

Inj Ampicillin 500mg 4th hrly ie 6 times a day

Inj Vancomycin 2gm iv BD (MRSA)

Bacteroides fragilis - metronidazole QID(7.5mg/kg iv every 6 hours)

Aminoglycosides may be required for proteus or pseudomonas (2mg/kg TID)

Raised ICT reduced by Dexamethasone 4mg iv QID or Dexona 8mg iv BD

Mannitol 20% in doses of 0.5 gm/kg (inj mannitol 100ml TID)

Neurosurgical approach ➔ Trautmans triangle (cerebellar abscess) or tegmen antri approach (temporal lobe abscess). Options include stereotactic aspiration or repeated aspiration through burr hole. If size doesn’t decrease on CT then excision is only left modality.

Open incision of the abscess and evacualtion of pus

LATERAL SINUS THROMBOPHLEBITIS (SIGMOID SINUS + TRANSVERSE SINUS THROMBOSIS)

Definition:
Inflammation of inner wall of lateral venous sinus (sigmoid sinus) with formation of a thrombus usually as a complication of mastoiditis.

**AETIOLOGY:**

- Acute coalescent mastoiditis, Masked mastoiditis or COM with cholesteatoma

**PATHOLOGY**

- Stages include:
  1. **Formation of perisinus abscess:** Abscess forms in relation to outer dural wall of the sinus. If direct spread bone may be eroded over it or can be intact in case of spread via venous route.
  2. **Endophlebitis and mural thrombus formation:** Inflammation spreads to inner wall of venous sinus with deposition of fibrin, platelets, blood cells leading to thrombus formation within lumen of sinus.
  3. **Obliteration of sinus lumen and intrasinus abscess** Org may invade causing intrasinus abscess May release septic emboli in systemic circulation and cause septecemia.
  4. **Extension of thrombus** Thrombotic process continues proximally and distally though septic emboli keep dislodging time to time. Proximally it may spread to confluence of sinuses i.e., superior sagittal sinus and cavernous sinus and distally into mastoid emissary vein, to jugular bulb or jugular vein.

**Bacteriology:**

- B. Proteus, Pseudomonas, E coli, staph

**Clinical Features:**

- **Picket fence fever** - (hectic type of fever with chills and rigors) coincides with septic emboli dislodgement
- 1 or more peaks a day
- Irregular episodes of chills and rigors
- Profuse sweating follows fall in temperature
- **Headache**
- **Progressive anaemia and emaciation**
- **Griesingers sign** – due to thrombosis of mastoid emissary vein, oedema over posterior part of mastoid
- **Papilloedema** (if extension of clot in superior sagittal sinus can lead to blurring of disc margins, retinal hemorrhages)
- **Quickenstedts test = Tobey Ayers test** ➔ jugular compression (n side comp leads to raised ICT but not on diseased side)
- **Crowe beck test** (Engorgement of retinal veins on pressing healthy side jugular vein)
- Tenderness along jugular vein

**INVESTIGATIONS:**

- Blood culture
- CSF - normal
✓ X ray mastoids - clouding of air cells
✓ CT ➔ Delta Sign (triangular area with rim of enhancement with central low density area is seen in posterior cranial fossa on axial cuts) As walls of sinus enhance but lumen doesn’t due to thrombus

**COMPLICATIONS**:

✓ Meningitis
✓ Subdural abscess
✓ Cerebellar abscess
✓ Septecemia and pyemia in lungs, bone, joints, SC tissue
✓ Cavernous sinus thrombosis
✓ Jugular vein thrombosis
✓ Jugular bulb thr
✓ Mastoid emissary vein
✓ Otitic hydrocephalous

**MANAGEMENT**:

✓ IV antibiotics as discussed
✓ Mastoidectomy
✓ Only in Lateral sinus thr it is prudent to perform an early MRM to clear infective foci
✓ Sinus bony plate is removed to expose the dura and drain perisinus abscess.
✓ An infected clot or intrasinus abscess must be drained after packing sinus from both sides
✓ Pack is removed 5 to 6 days post operatively and wound secondarily closed.
✓ Ligation of Internal Jugular vein is rarely required (only if medical and surgical tech fail)
✓ Anticoagulant therapy ➔ only if clot till Cavernous sinus
✓ Repeated Blood transfusions for anaemia

**OTITIC HYDROCEPHALOUS aka Symond's Syndrome**:

**DEFINITION AND MECHANISM**:

✓ Benign Intracranial HTN post Lateral sinus thrombophlebitis extension to superior sagittal sinus and failure of arachnoid villi to absorb CSF

**CLINICAL FEATURES**:

✓ Severe intermittent headache with nausea and vomiting
✓ 6th nerve palsy leading to diplopia
✓ Papilloedema and optic atrophy

**SIGNS**:

✓ Papilloedema
✓ Nystagmus
✓ CSF normal except raised pressure

**TREATMENT**:

✓ Aim is to reduce CSF pressure to prevent optic atrophy and blindness
Acetazolamide, Corticosteroids, repeated lumbar punctures or placement of lumbar drain, sometimes lumboperitoneal shunt
Treat cause

**EXTRADURAL / EPIDURAL ABCESS:**

✓ Collection of pus under bone and dura of skull.
✓ Normally there is no separation between the two but if infection occurs in adjacent structures like skull bones and mastoids, pus can spread.

**PATHOLOGY:**

✓ AOM ➔ bone over dura destroyed by hyperemic decalcification
✓ COM ➔ Cholesteatoma destroys
✓ Spread of infection can occur by venous thrombophlebitis where dura remains intact.
✓ Affected dura may appear unhealthy and discoloured and covered with granulations.
✓ Can lie in relation to middle or posterior cranial fossa or perisinus abscess.

**CLINICAL FEATURES:**

✓ Mostly asymptomatic and silent and discovered accidentally
✓ Focal neurological signs and raised ICP is uncommon
✓ Local pain, tenderness and oedema will be found on examination of skull.
✓ Persistent headache on side
✓ Low grade fever with malaise
✓ Severe ear pain
✓ Pulsatile purulent ear discharge
✓ Disappearance of headache once pus flows out of ear

**INVESTIGATIONS:**

✓ Contrast enhanced CT or MRI

**TREATMENT:**

✓ Antibiotics
✓ Mastoidectomy
✓ Remove overlying bone till healthy dura reached. In cases where tegmen tympani and sinus plate are intact and there is suspicion of abscess, they are removed deliberately to access and drain abscess.

**SUBDURAL ABCESS:**

✓ Pus between dura and arachnoid mater
✓ Routes of spread ➔ same
✓ Causes pressure symptoms
✓ More common to accumulate over cerebral convexities and over parafalcine region and above tentorium cerebella.

**CLINICAL FEATURES:**

✓ Raised ICT features
✓ Meningeal irritation
✓ Pressure symptoms
✓ Thrombophlebitis of cortical vein of cerebellum

**INVESTIGATIONS:**

✓ CT and MRI
✓ TREATMENT
✓ Lumbar puncture should be done as it is a cause of cerebellar herniation
✓ Burr hole with wide exploration of brain
✓ Craniotomy
✓ Antibiotics
✓ Repeat scans
✓ Treat middle ear disease
✓ Raised ICT management
✓ Anticonvulsants specially in subdural empyema

MENINGITIS:
✓ Inflammation of leptomeninges i.e. pia and arachnoid usually with bacterial invasion of CSF in subarachnoid space.
✓ MC intracranial complication of Otitis media

Routes of Spread: are same

Pathophysiology:
✓ May be asso with all other complications

CLINICAL FEATURES:
✓ Raised ICT
✓ Meningeal irritation (kernigs, brudzinski’s sign, exagerrated tendon reflexes.)

Diagnosis:
✓ CT and MRI
✓ CSF is turbid, cell count raised , raised proteins, sugar and chlorides reduced

TREATMENT
✓ Antibiotics
✓ Corticosteroids
✓ Surgical - Mastoidectomy