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# OTOGENIC BRAIN ABSCESS, LATERAL SINUS THROMBOPHLEBITIS & OTITIC HYDROCEPHALUS



#### OTOGENIC BRAIN ABSCESS

#### **ROUTE OF INFECTION**

- direct extension of middle ear infection through the tegmen or by retrograde thrombophlebitis.
- Cerebellar abscess also develops as a direct extension through the Trautmann's triangle or by retrograde thrombophlebitis



#### **BACTERIOLOGY**

#### **Aerobic**

- pyogenic staphylococci
- Streptococcus pneumoniae,
- Streptococcus haemolyticus,
- Proteus mirabilis,
- Escherichia coli and Pseudomonas aeruginosa

#### Anaerobic

- Peptostreptococcus
- Bacteroides fragilis.
- Haemophilus influenzae



#### **PATHOLOGY**

- Brain abscess develops through four stages.
- 1. Stage of invasion (initial encephalitis)
   Patient may have headache, low-grade fever, malaise and drowsiness.
- 2. Stage of localization (latent abscess)
   no symptoms during this stage. Nature tries to localize
   the pus by formation of a capsule.



#### 3. Stage of enlargement (manifest abscess)

Abscess begins to enlarge. A zone of oedema appears round the abscess and is responsible for aggravation of symptoms

Clinical features at this stage are due to:

- (a) Raised intracranial tension.
- (b) Disturbance of function in the cerebrum or cerebellum, causing focal symptoms and signs.



• Stage of termination (rupture of abscess).

An expanding abscess in the white matter of brain ruptures into the ventricle or subarachnoid space resulting in fatal meningitis



#### **CLINICAL FEATURES**

#### Symptoms and signs of raised intracranial tension

- Headache.
- Nausea and vomiting
- Level of consciousness
- Papilloedema
- Slow pulse and subnormal temperature



#### **Localizing features**

- Nominal aphasia.
- Homonymous hemianopia.
- Contralateral motor paralysis.
- Epileptic fits.
- Pupillary changes and oculomotor palsy.



- Headache
- Spontaneous nystagmus
- Ipsilateral hypotonia and weakness.
- Ipsilateral ataxia.
- Past-pointing and intention tremor
- Dysdiadochokinesia.



#### **INVESTIGATIONS**

- Skull X-rays
- CT scan
- X-ray mastoids or CT scan of the temporal bone for evaluation of associated ear disease.
- Lumbar puncture.

## CT scan showing left-sided cerebellar abscess



## CT scan of right-sided otogenic cerebral abscess.





#### **TREATMENT**

- MEDICAL
- High doses of antibiotics are given parenterally.
- Raised intracranial tension can be lowered by dexamethasone, 4 mg i.v. 6 hourly or mannitol 20% in doses of 0.5 g/kgbody weight
- Neurosurgical.
- Abscess is approached through a sterilefield.
- Options include: (i) repeated aspiration through a burr hole, (ii) excision of abscess and (iii) open incision ofthe abscess and evacuation of pus.

### LATERAL SINUS THROMBOPHLEBITIS (SYN. SIGMOID SINUS THROMBOSIS)

- It is an inflammation of inner wall of lateral venous sinus with formation of an intrasinus thrombus.
- AETIOLOGY
- complication of acute coalescent mastoiditis, masked mastoiditis or chronic suppuration of middle ear and cholesteatoma.



#### **PATHOLOGY**

- 1. Formation of perisinus abscess.
- 2. Endophlebitis and mural thrombus formation.
- 3. Obliteration of sinus lumen and intrasinus abscess.
- 4. Extension of thrombus.



- 1. Formation of perisinus abscess.
- Abscess forms in relation to outer dural wall of the sinus. Overlying bony dural plate may have been destroyed by coalescent bone erosionor cholesteatoma. Sometimes, it remains intact when route of infection was by thrombophlebitic process.



- 2. Endophlebitis and mural thrombus formation.
- Inflammation spreads to inner wall of the venous sinus with deposition of fibrin, platelets and blood cells leading to thrombus formation within the lumen of sinus.



- 3. Obliteration of sinus lumen and intrasinus abscess.
- Mural thrombus enlarges to occlude the sinus lumen completely. Organisms may invade the thrombus causing intrasinus abscess which may release infected emboli into the blood stream causing septicaemia.



- 4. Extension of thrombus.
- Though central part of thrombus breaks down due to intrasinus abscess, thrombotic process continues both proximally and distally. Proximally, it may spread to confluence of sinuses and to superior sagittal sinus or cavernous sinus, and distally, into mastoid emissary vein, to jugular bulb or jugular vein.



#### CLINICAL FEATURES

- This is due to septicaemia, often coinciding with release of septic emboli into blood stream. Fever is irregular having one or more peaks a day. It is usually accompanied by chills and rigors. Profuse sweating follows fall of temperature. Clinical picture resembles malaria but lacks regularity. In between the bouts of fever, patient is alert

### FirstRanker.com © 2. Headache.

- 3. Progressive anaemia and emaciation.
- 4. Griesinger's sign. This is due to thrombosis of mastoid emissary vein. Oedema appears over the posterior part of mastoid.



- This is to record CSF pressure by manometer and to see the effect of manual compression of one or both jugular veins. Compression of vein on the thrombosed side produces no effect while compression of vein on healthy side produces rapid rise in CSF pressure which will be equal to bilateral compression of jugular veins.
- 8. Tenderness along jugular vein.



- ▼ 7. Crowe–Beck test.
- Pressure on jugular vein of healthy side produces engorgement of retinal veins (seen by ophthalmoscopy) and supraorbital veins. Engorgement of veins subsides on release of pressure.



#### **INVESTIGATIONS**

- 1. Blood smear is done to rule out malaria
- 2. Blood culture
- **3. CSF examination**
- 4. X-ray mastoids may show clouding of air cells (acute mastoiditis) or destruction of bone (cholesteatoma)
- 6. Culture and sensitivity of ear swab.



- 5. Imaging studies.
- Contrast-enhanced CT scan can show sinus thrombosis by typical delta sign. It is a triangular area with rim enhancement and central low density area is seen in posterior cranial fossa on axial cuts. MR imaging better delineates thrombus. "Delta sign" may also be seen on contrast-enhanced MRI.



#### **TREATMENT**

- 1. Intravenous antibacterial therapy.
- 2. Mastoidectomy and exposure of sinus A complete cortical or modified radical mastoidectomy is performed, depending on whether sinus thrombosis has complicated acute or chronic middle ear disease. Sinus bony plate is removed toexpose the dura and drain the perisinus abscess



• 3. Ligation of internal jugular vein.

It is rarely required these days. It is indicated when antibiotic and surgical treatment have failed to control embolic phenomenon and rigors, or tenderness and swelling along jugular vein is spreading.

4. Anticoagulant therapy.



#### OTITIC HYDROCEPHALUS

- It is characterized by raised intracranial pressure with normal CSF findings.
- It is seen in children and adolescents with acute or chronic middle ear infections.



#### **MECHANISM**

- Lateral sinus thrombosis accompanying middle ear infection causes obstruction to venous return.
- If thrombosis extends to superior sagittal sinus, it will also impede the function of arachnoid villi to absorb CSF.
- Both these factors result in raised intracranial tension.



#### **Symptoms**

- 1. Severe headache, sometimes intermittent, is the presenting feature. It may be accompanied by nausea and vomiting.
- 2. Diplopia due to paralysis of VIth cranial nerve.
- 3. Blurring of vision due to papilloedema or optic atrophy.



#### **SIGNS**

- 2. Nystagmus due to raised intracranial tension.
- 3. Lumbar puncture. CSF pressure exceeds 300 mm H2O (normal 70–120 mm H2O). It is otherwise normal in cell,protein and sugar content and is bacteriologically sterile.



#### **TREATMENT**

- The aim is to reduce CSF pressure to prevent optic atrophy and blindness. This is achieved medically by acetazolamide and corticosteroids and repeated lumbar puncture or placement of a lumbar drain.
- Sometimes, draining CSF into the peritoneal cavity (lumboperitoneal shunt) is necessary.
- Middle ear infection may require antibiotic therapy and mastoid exploration to deal with sinus thrombosis.