Complications of Suppurative Otitis Media

Complications of CSOM are associated with a high morbidity & may be life threatening. Cholesteatoma, atticoantral mucosal disease & ASOM cause complications by spread of infection:

1. Directly via the oval window to reach the labyrinth, through osteomyelitic bone to reach the dura & lateral sinus or to affect a congenitally dehiscent facial nerve.
2. By retrograde propagation of small foci of thrombophlebitis which may extend through the temporal bone & dura to the major venous sinuses to cause a lateral sinus thrombosis & by further extension a cerebellar or temporal lobe abscess.
3. Along the periarteriolar spaces to cause a temporal or cerebellar abscess. Browning in a retrospective study has calculated that the risk of a patient with CSOM developing an intracerebral abscess is 1 in 3500.

Classification

- **Extracranial**
  1. **Chronic otitis externa & meatal stenosis.**
  2. **ossicular discontinuity from ossicular erosion.**
  3. **middle ear adhesions.**
  4. **Tympanosclerosis which may spread from the tympanic membrane over the ossicular chain fixation.**
  5. **Squamous cell carcinoma of the middle ear.**
  6. **Lower motor neuron facial nerve palsy.**
  7. **Serous or purulent Labyrinthitis.**
  8. **Petrositis & Gradingo's syndrome(signs of ASOM, an ipsilateral abducent nerve palsy & pain in the distribution of the ipsilateral trigeminal nerve).**
  9. **Labyrinthine fistula.**
- **Intracranial**
  1. **Lateral (transverse & sigmoid) sinus thrombosis. This may extend to involve the superior & inferior petrosal sinus, the cavernous sinus, the sinus confluence, the superior sagittal sinus & the internal jugular vein. There is often a concomitant or subdural abscess which may have precipitated the formation of the thrombus.**
  2. **Meningitis.**
  3. **Extradural, subdural, intracerebral(cerebellar & temporal lobe) abscess.**
  4. **Otitic hydrocephalus.**

Clinical Features

Patients with acute intracranial complications usually present to the neurosurgeons & are most likely to be seen by an ENT surgeon after recovery from the acute episode. Patients with CSOM who present with unilateral or occipital headaches, visual disturbance, vomiting, clumsiness, forgetfulness or drowsiness should have a full neurological examination looking in particular for signs of raised intracranialpressure, meningitis & localizing cerebellar & temporo-parietal lobe signs. A deep throbbing otalgia & serosanguinous discharge may herald malignant change.

Investigations

A high definition CTscan of the petrous temporal bone will show the extent of mastoid disease although it may not distinguish cholesteatoma from mucosal disease. A gadolinium enhanced magnetic resonance scan is now the investigation of choice for the diagnosis of an intracranial venous thrombosis (simple thrombus shows an intermediate

signal, vascularized thrombus, granulation tissue & slow flowing blood a high signal, & fast flowing blood no signal) & intracranial abscess (shows a center of low attenuation with an outer rim of high signal).

Treatment

- High dose intravenous antibiotics to commence after taking a culture- swab of the aural discharge.
- Neurosurgeons to manage intracerebral abscess.
- Treatment of initiating otological disorder.

1. Subdural and Extradural abscesses: require a cortical mastoidectomy to provide adequate exposure before drainage. A lateral sinus thrombosis if not responding to high dose intravenous antibiotics should have a cortical mastoidectomy & the lateral sinus exposed. The diagnosis should be confirmed by needling the sinus. After confirmation some practitioners propose tying the internal jugular vein high up in the neck to prevent infective Embolization during evacuation of infected clot. The lateral sinus is opened, the clot evacuated & the sinus obliterated with temporalis muscle flap reinforced by a bismuth iodoform paraffin paste(BIPP) mastoid pack.

2. A facial palsy: secondary to ASOM is invariably a neuropraxia. The nerve does not require decompressing & should recover rapidly with aggressive treatment of the infection. The facial palsy in CSOM is usually secondary to compression from cholesteatoma or granulation tissue. Most otologists advocate an urgent mastoidectomy & decompression of the vertical segment of the facial nerve though this has recently been challenged. If there is actively discharging ear others would observe for at least 48hours with the patient on intravenous antibiotics. In this instance the palsy may be a neuropraxia of a dehiscent horizontal segment of the nerve, found in 6% of ears.

3. Labyrinthine fistulae: may be caused by erosion of bone by cholesteatoma & by osteitis with the formation of granulation tissue. In cholesteatoma the matrix usually becomes apposed to the endosteum within the fistula & a protective walling off does not arise. If a fistula is suspected from clinical signs & operative findings then either:!) the matrix can be left over the affected portion of the labyrinth & a canal wall down procedure performed leaving an open cavity, or • alternatively,2) the matrix can be peeled off under constant irrigation & the fistula immediately sealed with fascia or muscle as the final manoeuvre in surgery.