CHAPTER 8.
MIDDLE EAR INFECTIONS

Infected middle ear conditions

a) Acute Otitis Media  
b) Chronic Otitis media  
c) Cholesteaoma

1. ACUTE OTITIS MEDIA (AOM)

AOM is one of the common childhood infections. The sufferer is typically a child of tender years, often in the early day care situation. The problem may be accompanied by other ill health and is prevalent in lower socio-economic and indigenous populations.

PATHOGENESIS

AOM arises from the relationship of the ear and nose. As the ear is in direct continuity with the postnasal space via the Eustachian tube, nasal sepsis has a ready portal of entry into the ear. The ear, being lined with simple squamous epithelium with relatively few ciliated cells, is susceptible to bacterial infection.

Infection in the nose presents as a mouth-breathing child with a purulent rhinorrhea, and possibly attendant lower respiratory problems.

Figure 1: AOM. An inflamed and distended drum, due to the pressure of purulent exudate evident in the middle ear.

The problems are frequent in winter, in line with the onset of viral URTIs. The latter result in damaged ciliary action in the nose, inflammation and exudate. The pooled fluids provide ideal breeding sites for bacterial infections acquired from contact with the peer group, the nose becoming rapidly soiled with infected mucus. Strep. Pneumonia, Haemophilus influenza and Moraxella catarrhalis are the main pathogens plus some Strep. pyogenes and Staph. aureus.

Sneezing or other actions insufflate the infected debris into the ear, where acute inflammation rapidly advances. Outpouring of exudate accumulates under pressure within the middle ear, epithelial damage and oedema of the Eustachian tube obstructing drainage.
Tension on the drum results in distension of the highly inflamed membrane. Blebs of serous or purulent debris form, then the drum ruptures, draining profuse seropurulent fluid via the EAC. This releases bacteria and toxins from this site and relieves pressure on the vascular supply. Further expulsion of the infected debris, plus improved immunological response reduces inflammation and oedema. As the tubal function recovers and the ciliary action within improves, aeration recommences, improving over 2-3 weeks. Some cases, however, may be troubled by impeded clearance and persisting OME.

Figure 2: Resolving AOM. A purulent effusion is seen behind the drum, but the inflammation has settled and a fluid level is evident behind the anterior drum.

1.2 PRESENTATION

Commonly, there is an antecedent URTI with nasal obstruction and rhinorrhea. This is followed by a blocked sensation then mounting deafness, discomfort and pain in the affected ear. Adults describe difficulty or pain when attempting auto-inflation to improve the blockage. The pain may be agonising.

General malaise and fever may be concurrent, especially in children, in whom otorrhoea is not uncommonly this first indication of ear involvement.

Figure 3: Mucopurulent otorrhoea from a perforated AOM. Discharge may be so profuse that the fluid visibly drains down the neck.

Examination of the ear reveals the distended and reddened drum as the purulent exudate progresses. Serous blebs may resemble bullous myringitis. Conductive deafness is present, and the seropurulent or bloodstained otorrhoea may have commenced prior to presentation. The perforation is usually small, and not readily seen without suction toilet and good magnification. Normally, spontaneous closure of the drum is followed by rapid resolution of discomfort, although deafness and a blocked sensation trouble adults for 2-3 weeks afterwards.

In other cases, particularly children, resolution is more prolonged, the drum taking on a subacute inflamed character, with less inflammation, but with an opaque grey appearance marked out by vascularity radiating from the umbo.

Related pathology may be observed on the URT. Mouth breathing, mucoid rhinorrhea, and large tonsils suggest enlarged and chronically infected adenoids as the basis of the infection. Accompanying obstructive sleep apnoea may be present, producing a sallow fatigued appearance.
1.3. INVESTIGATION

Suction toilet and drum inspection under the operating microscope may demonstrate the typical acute infection rather than a flare-up of more chronic disease (COM or cholesteatoma).

Bacteriology is not commonly employed due to the rapidity of both the disease pattern and its resolution, but ear or nasal cultures may be useful in persisting or repetitive cases to demonstrate atypical pathogens.

Audiology is normally left until resolution appears complete, as the hearing is normally depressed until this time. Follow up testing and tympanometry are useful to exclude lingering OME.

Radiology is used to ascertain the status of the adenoids or sinuses. Less commonly, it is used to check for covert mastoid disease.

1.4. MANAGEMENT

The AOM patient may be unwell and in pain; prompt and efficient management is desirable. Initial pain relief is indicated. Treatment of the infection is directed by the likely response to therapy. The great majority of pathogens are sensitive to amoxicillin-clavulanic acid or cefaclor, which are therefore the agents of choice. If allergy to both is present, use erythromycin or clarithromycin. Topical and systemic decongestants (antihistamine-sympathomimetic combinations) relieve nasal obstruction and promote drainage of infection, for comfort and to reduce recurrent infection.

If otorrhoea is present, clean the external canal, then use an absorbent antibiotic-soaked wick to minimise residual debris. Follow up with aqueous antibiotic drops for a week. Avoid oily drops; these tend to accumulate in the deep canal, prolonging deafness.

Figure 4: Acute myringotomy. Drainage of the middle ear is undertaken via the anteroinferior quadrant. This avoids the vital structures in the other areas of the middle ear. m: malleus; i: incus; s: stapes; ct: chorda tympani; rw: round window; et Eustachian tube; M: myringotomy site.

Pain may be severe. In these cases an acute myringotomy and vent tube insertion is beneficial to reduce the discomfort of pressure, and promote recovery.

This can be done in adults under topical phenol anaesthesia, similar to the OME procedure, or under general anaesthesia in children. Further management is as per the above for perforated AOM.

1.5. RECURRENT AOM

Repeated attacks generally indicate earlier surgery, but if unavailable or avoided, a trial of a six week course of amoxicillin on a normal dose b.d. basis, preferably accompanied by systemic nasal decongestants to minimise nasal origin recurrence. Refractory cases will require vent tube
insertions and adenoidectomy. The tubes abolish repeated pain and nocturnal distress, which partly explains their efficacy in this situation.

Over many years, a persistent reluctance to employ antibiotics in AOM has been expressed. Significantly this has been partly based on studies that have lacked expert otolaryngological input. AOM in children is commonly difficult to diagnose if the child is fractious, if there is wax in the canal, if the examiner lacks either cleaning expertise or the necessary instrumentation, if the clinician is not expert in discerning the stages of AOM from OME, or if the lighting used is sub-optimal (operating microscopes are often needed). Data derived from situations where these necessities are not met is suspect at best.

AOM is a potentially severe infection with a range of major and unpredictable complications, outlined below. In those communities where primary antibiotic care is often unavailable (e.g. indigenous centres) advanced pathology is prevalent. In developed communities where such treatment is available, the "mastoid wards" of yesteryear, full of chronic cases, were closed with the advent of penicillin.

1.6. COMPLICATIONS OF AOM

a) Chronic Otitis Media

Whilst most cases settle with prompt antibiotic management, some cases of more virulent or neglected infection result in chronic drum perforation, with secondary deafness or ongoing infection, requiring surgical correction. Deafness may result from ossicular damage or scarring, or persistent tubal dysfunction.

b) Acute Mastoiditis

If not treated promptly, fulminating infections may cause diffuse infection throughout the mastoid air cell system, often in infants. Mounting pressure of purulent effusions in the mastoid may produce a rupture through the mastoid cortex over the antrum, causing a fluctuant subperiosteal abscess posterosuperior to the auricle, causing the ear to protrude markedly. In a sick child with this unilateral ear appearance, acute mastoiditis should be immediately suspected.

In the past, AOM was a not uncommon cause of major complications. This is no longer the case, as these infections succumb readily to appropriate treatment but some persist.
Figure 6. Acute mastoiditis. An abscess above and behind the ear, with protrusion of the auricle should alert the clinician to the possibility of mastoiditis.

Immediate parenteral AOM antibiotic management is appropriate, together with a grommet insertion to expedite drainage. Postaural incision and drainage, or a cortical mastoidectomy is needed for more advanced cases.

c) Facial palsy
Paralysis of the facial muscles due to AOM requires immediate management to salvage neural function. Grommet insertion and steroid treatment are optimal, in addition to parenteral antibiotics. For cases such as this, or cholesteatoma, AOM should be viewed as an otological emergency, as these have a good prognosis if diagnosed and managed promptly but not if treatment is delayed.

d) Petrositis
Extension of air cell disease into the petrous bone, causing apical cell infection may inflame the fifth (trigeminal) and sixth (abducens) cranial nerves as they cross the apex, causing Gradenigo syndrome (ipsilateral lateral rectus palsy, facial pain and otorrhea).

e) & f) Sensorineural Deafness, Meningitis
Sensorineural deafness may succeed AOM either via direct penetration of the otic capsule, or due to meningitis. Both incur a risk of labyrinthine ossification that may prevent cochlear implantation.
Figure 7: Sequelae and complications of AOM
2. CHRONIC OTITIS MEDIA

(COM, Chronic Suppurative Otitis Media (CSOM)).

Figure 8: COM: perforation of the drum may be associated with major ossicular damage. m: malleus, i: incus, p: perforation, 1: incus, 2 satpes superstructure, 3: malleus handle.

COM refers to those ears in which the drum has a chronic, non-healing drum perforation. The drum defect may or may not be associated with ossicular fixation or necrosis, middle ear infection or mastoiditis, tympanosclerosis, or middle ear fibrosis.

2.1. AETIOLOGY

a) The drum perforation may result from a virulent infection (bacterial or fungal).
b) Malnutrition or immune-deficient states may render the ear susceptible.
c) Trauma may result in a non-healing defect.
d) Previous tubal insufficiency and drum retraction and atrophy may render the drum prone to perforation.

Figure 9: COM: an inferior perforation of the pars tensa is present, resulting from a long term grommet insertion.

2.2. PATHOGENESIS

Initial perforation of the drum by one of the above mechanisms exposes the middle ear to infection from the EAC, particularly water-borne soiling.

Chronic infection by gram-negative organisms is common, resulting in otorrhoea and caked debris accumulation in the EAC. Repeated middle ear infection leads to a gradual metaplasia into a more ciliated respiratory type epithelium that adds to the mucoid discharge. Chronic mastoid infection frequently follows. Further infection may cause scarring and tympanosclerotic deposits, and conductive losses.

Figure 10: COM: total loss of the pars tensa, as a result of virulent streptococcal infection. The middle ear is free of infection.
2.3. PRESENTATION

Pain is unusual, being associated normally with acute infection flare-ups.

Otorrhoea may be absent, or intermittent and mucoid with further URTI, or with transcanal soiling. The discharge may become persistent and copious if chronic mastoiditis occurs.

Conductive deafness is present, becoming more severe with progressive involvement of the middle ear contents. Gradual sensorineural losses may develop in chronic cases, possibly due to ototoxic effects from the infected site.

Tinnitus tends to be popping or gurgling, perhaps with electronic overtones if SND is present.

Examination may show adherent dry crusted debris lining the EAC, or mucoid purulent fluid obscuring the drum site. Cleaning the external canal shows the drum defect and associated middle ear changes. The mucosa may be polypoid and the EAC skin may demonstrate glistening or reddened chronic myringitic changes, especially around the drum site. If chronic mastoiditis is present, pulsatile mucoid otorrhoea may be present.

Figure 11: Longstanding COM. There is a moderately large central perforation and extensive myringitic changes causing a thickened glistening appearance.

Audiology shows conductive losses up to 35-40 db in larger perforations, more if ossicular necrosis/fixation has occurred. Higher frequency SND may be noted in more chronic cases.

Figure 12: Audiology of the above case. Conductive deafness with a bilateral high frequency SND indicative of an older patient. Greater SND in the affected Rt ear suggests longevity and possible mild ototoxicity.
Complications of COM

Apart from the chain and chronic mastoiditis changes as mentioned above, the complications are as per AOM and usually due to acute flare-ups of infection.

2.4. MANAGEMENT OF COM

a) Conservative

Local and systemic measures are undertaken to control infection and as a prelude to repair.

Meticulous cleaning by suction toilet under microscopy is optimal to clear debris, followed by topical antibiotics (gentamicin or ciprofloxacin) delivered by wick or drops. Broad spectrum antibiotics are used systemically if active infection is present.

b) Surgical

Surgical management is required for definitive repair:

- a) Drum repair: Myringoplasty
- b) Chain Reconstruction: Ossiculoplasty
- c) Elimination of mastoiditis: Intact canal wall mastoidectomy

“Tympanoplasty” is a looser term, covering combinations of the above.

3. CHOLESTEATOMA

3.1. PATHOLOGY

Cholesteatoma is a cystic or sac-like lesion of skin, identical to that of the eardrum, found in the middle ear. Congenital and acquired patterns are found.

Congenital patterns are cystic and derived from rests of epithelium derived from the hollowing out of the original ectodermal ingrowth that forms the EAC. These occur behind an intact drum.

Acquired patterns are derived from drum invagination. Three patterns are noted:

- a) Attic, arising from the pars flaccida.
- b) Pars tensa, from collapse of the larger drum face.
- c) Combined attic-pars tensa disease.

Figure 13: Patterns of Cholesteatoma:

1. Congenital, 2. Attic, 3. Pars tensa,
2+3: Combined attic, pars tensa.
Chronic tubal insufficiency is the likely culprit in the majority of cases. Resultant negative middle ear pressure produces drum collapse, in pocketing, weakening, then failure of the self-cleaning epithelial migration mechanisms. This leads to accumulation of keratinaceous debris, infection, then gradual osteolytic erosion of the surrounding structures. The disease may extend to fill the middle ear cleft, from the Eustachian tube to the mastoid tip.

A past history of grommet insertions may be given.

Attic disease frequently has a shorter history, suggestive of a recent invagination. They tend to occur in more well-developed and aerated mastoids, without effusions. In contrast, the pars tensa and combined patterns tend towards stonier mastoids and more overt evidence of tubal failure: drum collapse and effusions.

3.2 PRESENTATION:

Congenital patterns present as “pearly” cysts, either as a small lesion in the antero-superior middle ear, or as more extensive types that fill the middle ear, the attic and perhaps beyond.

Attic lesions are seen as a defect above the handle of the malleus, with keratin within, either a dry plug, or infected matter. The latter not uncommonly displays an attic polyp of granulations that may fill the EAC.

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Figure 14: Congenital cholesteatoma. A classic “pearl” is seen just anterior to the handle oc of the malleus.

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Figure 15: Attic cholesteatoma. A pars flaccida invagination is filled with necrotic keratin. Concurrent middle ear effusion.

Pars tensa patterns occur from gradual collapse of the pars tensa, usually the posterior half, filling the middle ear and penetrating into the attic. These are Stage IV adhesive otitis cases.

Figure 16: Pars tensa pattern cholesteatoma. Sever collapse of the posterior pars tensa has lead to drum invagination, keratin accumulation and granulations.

Combined pattern cases display both attic and pars tensa pockets and disease.
3.3 MANAGEMENT

a) Conservative

Conservative management is similar to that for COM, but is generally a prelude to surgery. Given the nature of this disease, prolonged cleaning and medical treatment is appropriate only in those unfit for surgery or when this is not available.

b) Surgery

Three modalities are available:

i. Transcanal – for lesser disease, confined to the middle ear.

ii. Open cavity surgery
   a) Atticotomy
   b) Open cavity mastoidectomy (canal wall down, CWD, radical or modified radical mastoidectomy)

iii. Intact canal wall mastoidectomy (ICW, Canal wall up CWU, Combined approach tympanoplasty, CAT)

(See Middle Ear Surgery)

Pain is uncommon, unless severe inflammation of the surrounding tissues has occurred.

The otorrhoea is often foul and/or bloodstained from a polyp formation.

Debris similar to that of COM is found in the EAC.

Conductive deafness is usual, but the cholesteatoma itself transmits sound in some cases that display only minimal losses. A blocked sensation is common.

Tinnitus resembles that of COM.

Vertigo due to the formation of a fistula into the inner ear is not uncommon, and a warning for the surgeon to take care. A fistula test may be positive (pressure on an occluded EAC, causing vertigo).

It should be noted that, in general, granulations in the deep EAC frequently accompany more substantial pathology that requires specialist ontological management. These cases should be referred for such at an early stage.

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**Figure 17:** Combined pattern cholesteatoma showing both pars tensa and attic collapse, with progressive erosion of the EAC wall.
3.3. COMPLICATIONS OF CHOLESTEATOMA

The complications of cholesteatoma reflect the direction of spread, whether medially, superiorly, or posteriorly.

a) Medial extension

Destruction of the drum and ossicular chain are very common, causing conductive deafness

Facial paralysis occurs when infection compromises the VII (facial) nerve, usually in the horizontal section of its course through the middle ear, just above the stapes.

Figure 18: Complications of medial spread of cholesteatoma: 1. Ossicular necrosis, 2. Facial palsy, 3. Otic capsule fistula, bacterial labyrinthitis

Further medial extension may erode the otic capsule, resulting in a fistula. Bacterial invasion of the inner ear results in bacterial labyrinthitis and a dead ear with the triple symptoms of loud tinnitus, profound sensorineural deafness and severe rotatory vertigo followed by disabling disequilibrium, and later labyrinthine ossification.

Superior extension

Initial breaching of the tympanic plate may produce an extradural abscess. Meningitis may follow perforation of the dura, or a subdural abscess formation.

Figure 19: Lateral semicircular fistula secondary to advanced cholesteatomatous otic capsule erosion.


Further penetration of the temporal lobe produces cerebritis or a temporal lobe abscess with a range of symptoms:

a) General neurosurgical space-occupying lesion signs (headache, disorientation, malaise)

b) Dysarthria

c) Hallucinations of taste and smell, deja vu
Posterior extension

Acute mastoiditis and postaural abscess formation produces a characteristic pinna protrusion and postaural inflamed mass.

Deep cervical (Bezold's) abscesses may form upon penetration of the adjacent cervical soft tissues.

Lateral sinus thrombosis may extend to compromise the arachnoid villae, producing otitic hydrocephalus, or cavernous sinus thrombosis.

Invasion of the posterior fossa may produce a cerebellar abscess, with muscular incoordination, dizziness or meningitis.

Retro-labyrinthine extension may render removal of the disease difficult, perhaps necessitating labyrinthectomy.

Less commonly, anterior extension of infection may produce Gradenigo syndrome.

Iatrogenic complications related to removal of the disease include facial palsy, dead ear, CNS complications, or cavity symptomatology from open cavity surgery.