CHAPTER 1
Pain in the Ear

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Acute otitis externa

Acute otitis externa may be either diffuse – involving all the skin of the external meatus – or localized as a furuncle (Fig. 1.4).

Pain is one of six symptoms that may indicate ear disease (Box 1.1). Inflammatory causes of pain are recognized by inspection of the external ear and tympanic membrane. An otoscope is usually used in general practice, but otologists always use a headlight or head mirror to provide vision coincident with the direction of illumination, allowing manipulation with freed hands and instruments for the removal of wax or debris, and for the assessment of drum mobility with a pneumatic speculum (Fig. 1.1).

A binocular microscope is invariably used for fine manipulation with micro instruments and suction apparatus for accurate assessment under magnification of six times or more (Fig. 1.2).

If the external ear canal and the tympanic membrane are definitely normal, then pain cannot arise from ear disease. The reliability of this judgement depends on the skill and experience of the examiner. A tympanic membrane may show subtle changes, which are not easily recognized, while some abnormalities are irrelevant. If in any doubt, an otological opinion should be sought (Fig. 1.3).

OVERVIEW

- Pain in the ear (otalgia) arises from:
  - acute inflammatory disease of the external ear or middle ear cleft;
  - diseases not primarily in the ear;
  - referral from other sites;
  - neurological disease;
  - psychogenic.

Box 1.1 Symptoms of ear disease

- pain
- discharge
- hearing loss
- tinnitus
- vertigo
- facial palsy

Acute otitis externa

Acute otitis externa may be either diffuse – involving all the skin of the external meatus – or localized as a furuncle (Fig. 1.4).
A furuncle is a very tender swelling (a boil). It is always in the outer ear canal, as there are no hair follicles in the inner bony meatus. Hearing is impaired only if the meatus becomes blocked by swelling or discharge, and fever occurs only if infection spreads in front of the ear, as cellulitis or erysipelas. Superficially tender enlarged nodes may be palpable in front of or behind the ear. The pinna is tender to movement in acute otitis externa, but this is not the case in acute otitis media. Discharge, if any, is usually thick and scanty, unlike the copious mucoid discharge through tympanic membrane perforation from acute middle ear infections. Fungal skin infections cause severe pain with wet keratin desquamation and black or coloured granules of the fruiting heads of conidiophores.

Treatment of acute otitis externa
Systemic antibiotics are advised in acute otitis externa only if there is fever or lymphadenitis. Sometimes, meatal swelling must be reduced by inserting a ribbon gauze wick painted with a deliquescent substance such as magnesium sulphate paste, or glycerine and 10% ichthammol (Fig. 1.5). Proprietary 'Pope' wicks (Xomed) are thin and stiff to enable careful insertion, and they then soften and swell gently when moistened with liquid medication. A wick should be replaced daily until skin swelling subsides. Ear drops may then be used – either aluminium acetate to ‘toughen’ the skin or topical antibiotics, such as gentamicin, framycetin or neomycin, combined with steroids. Topical clotrimazole is a useful antifungal agent. Systemic analgesics, together with warmth, applied through a hot pad or heat lamp, relieve pain. Recurrent furunculosis should raise a suspicion of diabetes.
Acute suppurative otitis media

Acute suppurative otitis media causes deep-seated pain, impaired hearing and systemic illness with fever. A blocked feeling in the ear then pain and fever, are followed by discharge if the tympanic membrane perforates – with relief of pain. The whole middle ear cleft is affected. This is the entire air-containing space comprising the Eustachian tube, the middle ear cavity, the mastoid antrum and its adjacent mastoid air cells (Fig. 1.6). For this reason, deep pressure over the mastoid antrum elicits tenderness in acute otitis media; this does not imply the development of mastoiditis. Bacterial infection is usually by Streptococcus pneumoniae, or Haemophilus influenzae in very young children. Diagnosis is made by inspecting the tympanic membrane, but this may be prevented by wax, or by swelling from a secondary otitis externa. Only if the whole drum can be certified as normal and there is no conductive hearing loss (demonstrated by tuning fork tests) can otitis media confidently be excluded. Adjacent lymph nodes are never enlarged in simple otitis media.

Treatment of acute suppurative otitis media

Systemic antibiotics are recommended. The commonest infecting organisms are Streptococcus pneumoniae, Haemophilus influenzae and Moraxella (Branhamella) catarrhalis. The antibiotic of choice, effective against all these, is amoxycillin. If β-lactamase-producing organisms are likely, amoxycillin combined with clavulanic acid (Augmentin) or trimethoprin and sulphamethoxazole may be preferred. Oral administration is advised, even for the first dose, and medication must be continued for at least 5 days. Supplementary treatment includes pain relief by analgesics and warmth. Warm olive oil drops are soothing. If the tympanic membrane perforates, the ensuing discharge should be cultured, but an antibiotic should be changed on clinical and not bacteriological grounds. Rarely, the drum may bulge under pressure without rupture, requiring urgent incision to release pus (myringotomy).

Recurrent acute otitis media may be provoked by predisposing causes, such as persisting middle ear effusions, when a potentially infected accumulation of mucus persists in the middle ear cleft. Myringotomy with insertion of a ventilation tube or ‘grommet’ may then be advisable. Adenoid enlargement with repeated infection is probably also a causative factor, but the role of adenoidectomy remains controversial. In the absence of predisposing factors, each attack should be treated as it arises. After any episode, return to normal is expected and should be confirmed within 3 weeks.

Acute (coalescent) mastoiditis

Acute mastoiditis is caused by the breakdown of the thin bony partitions (trabeculae) between the mastoid air cells, which then become coalescent (Fig. 1.7). This process takes 2–3 weeks to occur fully. Throughout that time, there is, in most cases, continuing and increasingly copious discharge through a perforation in the drum, with general malaise and fever, unless this has been suppressed by antibiotics. If a patient has pain a few days after the tympanic membrane has been reliably judged to be normal, then that patient cannot have developed mastoiditis. Difficulties arise when a patient is thought to have recovered from acute otitis media, but in reality the condition has ‘grumbled on’, perhaps by suppression of systemic effects with antibiotics. Mastoiditis should be suspected in any patient with continuous discharge from the middle ear for over 10 days, particularly if he or she is continually unwell. Radiographs or, better, CT scans of the mastoid air cells may help to diagnose the condition, but not always. Only if they show a clearly aerated normal cell system (Fig. 1.8) can mastoiditis be excluded. The classical appearance of breakdown of intracellular trabeculae is not always apparent. Otitis externa may cause apparent haziness of the air cell system because of oedema of the soft tissues over the mastoid process. The often-described traditional classical sign of a swelling behind the ear with downward displacement of the pinna implies a subperiosteal abscess. This is a complication rather than a feature of mastoiditis. A subperiosteal abscess can also, by erosion of the bony outer attic wall, cause swelling in the roof of the deep part of the external ear canal, in contrast with a furuncle, which arises only in its outer part. If any doubt persists after mastoid imaging, surgical exploration is advisable.
Other complications of acute suppurrative otitis media

These are all also possible complications of the bone erosive forms of chronic suppurrative otitis media (see Chapter 2 and Fig. 1.9). They arise if infection spreads beyond the middle ear cleft itself. Complications occurring within the petrous temporal bone include facial palsy, suppurrative labyrinthitis and lateral sinus thrombophlebitis; those occurring within the cranial cavity are meningitis, extradural abscess, subdural abscess and brain abscess (in the temporal lobe or cerebellum).

Chronic secretory otitis media (otitis media with effusion)

Niggly, short-lived pain is a common feature of ‘glue ear’. The drum looks abnormal because of the effusion (Fig. 1.10). Classically, there is injection with visible radial vessels, which may prompt a misdiagnosis of otitis media. The colour may be yellowish or sometimes blue. The child is well and afebrile, however, and the associated hearing loss has usually been recognized for some time.

An essential diagnostic feature, which can be elicited by an otologist using a headlight and a pneumatic speculum, is altered mobility of the tympanic membrane. It may be totally immobile when external ear canal air pressure is raised and reduced, or there may be sluggish outward movement followed by a rapid ‘snap’ back when the partial vacuum is released. This altered mobility can also be demonstrated by tympanometry using an impedance measuring meter during continuously changing ear canal pressure – from above to below normal atmospheric level. Simple, automatic tympanometers print out a quickly available chart indicating middle ear air pressure and its changes (if any) as the external ear air pressure is raised and then lowered. However, there can be technical problems in using these devices reliably.

‘Malignant’ otitis externa

This is a rare but serious form of infection (not neoplastic, despite the name), caused by Pseudomonas aeruginosa, arising usually in elderly diabetics. It should be suspected if patients in this group...
suffer severe pain, excessive for the signs of otitis externa. Infection invades the bony base of the skull and adjacent soft tissues. Facial paralysis and other cranial nerve palsies may develop, and mortality used to be high. A suspicious finding is granulation tissue in the ear canal, which must be investigated by CT scanning. Treatment with intravenous gentamicin, or with oral ciprofloxacin, is administered continuously for several weeks, and must not cease before recovery from pain.

Other causes of pain

Bullous myringitis is another cause of severe pain. Viral (probably influenzal) infection causes haemorrhagic blistering of the ear drum and external ear canal. There is often an associated haemorrhagic effusion in the middle ear and it may be difficult to distinguish this condition from otitis media. For that reason alone antibiotics may be administered, but the only necessary treatment is potent analgesia.

Referred pain

If the external ear canal and drum are normal, with normal movement of the drum on examination with a pneumatic speculum, pain cannot be due to disease of the ear. It may well be referred from territory sharing its ultimate sensory innervation with the outer or middle ear (Fig. 1.11). Pain therefore may arise from:

(a) The oropharynx (IXth nerve) in tonsillitis or carcinoma of the posterior third of the tongue.
(b) The laryngopharynx (Xth nerve) in carcinoma of the pyriform fossa.
(c) Upper molar teeth, temporomandibular joint or parotid gland (Vth nerve mandibular division). Parotid causes are usually obvious: impacted wisdom teeth may not be. Temporomandibular joint troubles often follow changes in bite caused by new dentures, extraction or grinding down.
(d) The cervical spine (C2 and C3). Pain is often worse at night when the head lies awkwardly. Neck support often provides relief, as does a neck pillow under the side of the neck during sleep. If there is no inflammatory ear disease and no disease in sites from which pain might be referred to the ear, remaining possibilities include glossopharyngeal neuralgia, migrainous neuralgia or psychogenic pain. Often no cause can be found; it may sometimes be attributed to depression and a trial of antidepressive medication can be advised.

Further reading