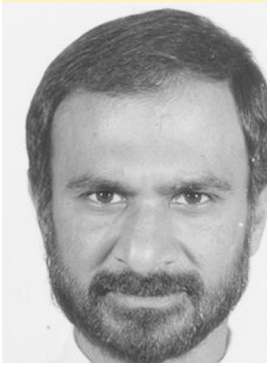
COMMON INFECTIONS OF THE EAR

Ear infections, both of the inner and outer ear, are common in general practice.



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OTITIS EXTERNA

Infection of the external auditory canal (EAC) is common and in certain circumstances can be life threatening. The EAC has highly effective mechanisms to prevent infection, which include migration of the skin covering the tympanic membrane and deep external canal, and the production of wax that has antibacterial and antifungal properties. Infection (otitis externa) will usually arise when these defences are overcome. Symptoms of otitis externa include otalgia, pruritus, discharge and hearing loss. The patient may experience pain with tragal pressure, or when the auricle is pulled upwards. The skin lining the ear canal is usually swollen and inflamed, and may be lined with debris. An approach to diagnosis and management of otitis externa is shown in Table I.

Patients with **non-infective otitis externa** present with itch of the ear canal and they are usually in the 40 - 50-year age group. On examination the ear canal is absolutely normal and there is absence of wax. The treatment of choice is application of combination steroid/antifungal/antibiotic ointment (e.g. Kenacomb) with a cotton bud daily for 3 days.

Patients with **fungal otitis externa** present with 'blockage' of the ear. On examination white debris similar to the cream of milk with black or white fungal spores is noted in the external ear canal. Treatment is to remove the debris by either syringing or suction and then packing the ear canal with 10 mm wide and 75 mm long ribbon gauze impregnated with combination steroid/antifungal/
antibiotic ointment (e.g Kenacomb) for 48 hours. The treatment is repeated until the patient is asymptomatic. Fungal and bacterial infections commonly occur together.

The patients with **bacterial otitis externa** are usually young adults who present with severe otalgia and fever after swimming in infected waters such as dams, rivers or the sea. On examination there is marked oedema of the ear canal with complete occlusion of the lumen. The treatment includes packing the ear canal with 10 mm wide and 75 mm long ribbon gauze impregnated with ichthammol glycerin, antibiotics and analgesics. The ear pack must be changed every 24 hours until the oedema subsides completely.

Necrotising otitis externa with skull base osteitis

This is a condition peculiar to elderly diabetic patients living in hot humid regions. The disease commences as osteitis of the

Table I. **Approach to otitis externa**

Type	Associated symptom	Characteristic features in external ear canal	Precipitating factors	Treatment
Non-infective	Itch	Normal	Nil	Acetic acid drops/steroid drops if severe
Fungal	Hearing loss	White debris with fungal spores	Nil	Acetic acid drops/combination steroid/anti-fungal/antibiotic (e.g. Kenacomb) ear pack if severe
Bacterial	Severe pain	Oedema with occlusion of the EAC	Swimming	Ichthammol glycerin, combination steroid/antifungal/antibiotic (e.g. Kenacomb), intravenous co-amoxiclav if severe
Necrotising	Deep-seated earache	Granulation tissue at junction of bony and cartilaginous EAC	Diabetes	Intravenous antibiotics (see text)

tympanic plate of the temporal bone and then spreads posteriorly to involve the tympanomastoid bone and medially along the base of the petrous bone towards the foramen magnum.

The exact aetiology is unknown but *Pseudomonas aeruginosa* has been persistently isolated from pus swab. Pseudomonas has the propensity to attach itself to diseased blood vessels leading to thrombosis, which explains the necrosis of soft tissue and bone in diabetic patients who already have microangiopathy.

Four stages of the disease are recognised:

- Stage 1 — Osteitis of the tympanic plate of temporal bone only. The patient presents with deep-seated, agonising and unrelenting earache which is worst at night. The sign that heralds osteitis of the tympanic plate is granulation tissue on the floor of the ear canal at the junction of the tympanic plate and the cartilaginous portion of the ear canal.
- Stage 2 — Osteitis of tympanic plate and stylomastoid bone. The patient presents with lower motor neuron VII cranial nerve palsy.
- Stage 3 — Osteitis of the base of the petrous bone with involvement of the jugular foramen and hypoglossal canal. The patient presents with IX, X, XI and XII cranial nerve palsies.
- Stage 4 — Intracranial extension.

Diagnosis

The diagnosis is clinical and the golden rule applies: Any diabetic patient who presents with deep-seated agonising earache has skull base osteitis until proven otherwise. The investigation of choice is technetium^{99} bone scan.

Treatment

The treatment of choice is intravenous antibiotics consisting of aminoglycoside, piperacillin and metronidazole for a minimum period of 6 weeks. In those patients with impaired renal function, the recommended treatment is intravenous ciprofloxacin and metronidazole.

Response to treatment is monitored by performing serial technetium bone scans at 3-weekly intervals. Antibiotics are only stopped when the bone scan is negative.

Prognosis

Prognosis is dependent on the stage of the disease. With stage 1 disease the prognosis is excellent; complete recovery occurs in all patients. The prognosis is poor in advanced disease.

ACUTE OTITIS MEDIA

Acute otitis media is inflammation of the mucoperiosteal lining of the middle ear cleft, i.e. the Eustachian tube, tympanic cavity, attic, mastoid antrum, and mastoid air cells. When inflammation affects the bony wall or spreads beyond the walls into the adjacent area, it is referred to as a complication of otitis media, e.g. otitis media with meningitis or otitis media with facial palsy.

Otitis media is common in children, with peak incidence in the 1 - 2-year age group, and usually follows upper respiratory tract infection. The infection spreads to the middle ear via the Eustachian tube.

Four stages of acute otitis media are recognised:

- Stage 1 — Stage of tubal occlusion is characterised by negative middle ear pressure with an effusion. Adult patients complain of a blocked ear and autophonia (echoing of one's own voice). The ear drum is retracted and the light reflex may be absent. There may be clinical evidence of middle ear effusion, for example air bubbles and fluid in the middle ear space.[1]
- Stage 2 — Stage of presuppuration. The middle ear effusion becomes infected and increases in

quantity and the patients have a fever and complain of throbbing earache. Children often vomit. The tympanic membrane is inflamed and bulges outwards.[1]

- Stage 3 — Stage of suppuration. The intratympanic pressure increases and occludes the blood supply resulting in necrosis and rupture of the tympanic membrane. This event is characterised by the patients reporting excruciating pain followed by a 'pop' and discharge, with immediate relief of earache. On examination a central perforation of the tympanic membrane is noted with pus oozing from the middle ear into the external ear canal.[1]
- Stage 4 — Stage of resolution. In 80% of patients complete resolution occurs: the otorrhoea subsides and the perforation heals spontaneously after a week. In 20% of patients, incomplete resolution occurs: the perforation and otorrhoea persist or the perforation heals with middle ear effusion.[1]

Acute otitis media is the most common reason for antibiotic prescription in children. Accurate identification of the causative pathogen can only be made by performing tympanocentesis — an invasive procedure that is almost never performed by clinicians. Therapy is therefore empiric, based on clinical studies identifying causative organisms, and on knowledge of antibiotic susceptibility patterns of the most common causative organisms.

Predominant pathogens are *Streptococcus pneumoniae, Haemophilus influenzae* and *Moraxella catarrhalis* (see Table II).

Otitis media caused by pneumococcus is least likely to resolve spontaneously, and it is important that first-line empiric antibiotics treat pneumococcal infection adequately. There has been a worldwide increase in drug-resistant *Streptococcus pneumoniae* (DRSP), and empiric therapy needs to take this into account. Of importance, resistance is due to a change in the affinity of penicillin-binding proteins in the bacterial cell wall and not to beta-lactamase production, as is the case with resistant *H. influenzae*. Thus DRSP has increased resistance to all beta-lactam antibiotics, including the cephalosporins.[2]

Table III gives the minimum inhibitory concentration required to treat DRSP for a selection of beta-lactam antibiotics that are commonly prescribed for the treatment of respiratory tract infections. The table includes the antibiotic level that is reached at the site of action — the middle ear fluid. Of importance, cefpodoxime, cefaclor and loracarbef are ineffective agents for treatment of DRSP. Amoxicillin remains the drug of choice when treating otitis media due to DRSP, and levels adequate for the treatment of DRSP may be attained by using higher doses. Ceftriaxone is an excellent agent, but must be given parenterally, and is therefore most useful in treating the younger child with otitis media who is very unwell and cannot tolerate oral medication.[2]

Benefits of antibiotic treatment for otitis media

A Cochrane review of antibiotics for acute otitis media in children showed no reduction in pain at 24 hours in children treated with antibiotics, compared with those treated with placebo. By 24 hours, two-thirds of children had recovered spontaneously, whether or not they were treated with antibiotics. Overall, antibiotics showed only modest benefit over placebo in pain reduction. Seven per cent fewer children had pain after 2 - 7 days, which means that 15 children needed to be treated with antibiotics to prevent 1 child from experiencing pain after 2 - 7 days.[2]

Because many cases of otitis media will resolve spontaneously, benefits of antibiotic therapy must be weighed up against the risk of adverse reactions to antibiotic therapy. In an older child who is otherwise well, a delay in initiating antibiotics may be considered. Antibiotics may, however, play an important role in reducing the risk of complications of otitis media, particularly mastoiditis.

Recommended first-line treatment for otitis media

An assessment of the risk of infection with DRSP must be made. Risk factors for infection due to resistant *Streptococcus pneumoniae* are as follows:

- age less than 2 years
- antibiotic treatment in the last 1 - 3 months
- attendance at a day care centre.

A child who is at high risk of DRSP infection should be treated empirically with high-dose amoxicillin (80 - 90 mg/kg/day of amoxicillin, in 3 divided doses). Those with a low risk of infection with DRSP may be treated with amoxicillin 40 - 45 mg/kg/day.

Management of clinically defined treatment failure

Treatment failure is a lack of clinical improvement after 3 days of therapy, with persistence of ear pain, fever, and redness and bulging of the tympanic membrane, or ottorrhoea. Treatment may fail due to lack of compliance, due to infection with DRSP which has been inadequately treated, or due to inappropriate choice of antibiotic (see Table III), or too low a dose of amoxicillin. Treatment with amoxicillin may also fail in cases where the causative organism is resist-

Table II. **Otitis media — causative organisms**

Organism	Percentage
S. pneumoniae	40%
H. influenzae	20%
M. catarrhalis	10%
Other streptococci	5 - 10%
Staphylococci	5%
No organism isolated	10 - 20%

Table III. **Activity of a selection of beta-lactam drugs, at standard doses, against DRSP[2]**

Beta-lactam antibiotic	MIC_{90} (µg/ml) Penicillin-susceptible strain	MIC_{90} (µg/ml) Penicillin-intermediate strain	MIC_{90} (µg/ml) Penicillin-resistant strain	Peak serum concentration (µg/ml)	Peak middle ear fluid concentration (µg/ml)
Amoxicillin	0.03	0.1 - 1	2 - 4	3.5 - 7	1 - 6
Ceftriaxone (e.g. Rocephin)	0.06	1	1 - 4	171	35
Cefuroxime (e.g. Zinnat)	0.125	1 - 4	4	2 - 7	1
Cefpodoxime (e.g. Orelox)	0.06	1 - 4	4	1 - 4	0.2
Cefaclor (e.g. Ceclor)	1	64	128	7 - 13	0.5 - 4
Loracarbef (e.g. Lorabid)	2	64	128	13 - 19	2

Table IV. **Complications of otitis media**

Extracranial complications	Presentation
Post-auricular abscess	Post-auricular swelling
Facial palsy	Drooling of saliva from angle of mouth and inability to close the ipsilateral eye on the same side
Labyrinthitis	Dizziness with nausea and vomiting
Bezold's abscess	Swelling of the mastoid tip
Petrous apicitis	Ipsilateral otorrhoea, VI cranial nerve palsy, and facial pain
Intracranial complications	
Extradural empyema	'Silent' or nuchal rigidity and pyrexia
Subdural empyema	Headache, pyrexia, with or without hemiparesis
Brain abscess:	
• Temporal lobe	Nuchal rigidity, aphasia, hemiparesis
• Cerebellar	Dizziness and unstable gait
• Lateral sinus thrombosis	Headache, pyrexia, and rigors, pain over the anterior border of sternocleidomastoid muscle
Meningitis	Severe headache, pyrexia, neck stiffness
Otitic hydrocephalus	Headache, nausea, and vomiting

ant due to beta-lactamase production (e.g. beta-lactamase-producing *H. influenzae*).[2]

Suitable antibiotics for management of treatment failure include high-dose amoxicillin-clavulanate (80 - 90 mg/kg/day of the amoxicillin component, with 6.4 mg/kg/day of clavulanate) or intramuscular ceftriaxone 50 mg/kg/day for 3 days.

CHRONIC OTITIS MEDIA

Chronic otitis media is osteitis of the mastoid bone and is characterised by persistent otorrhoea and tympanic membrane perforation. Two types are recognised — non-cholesteatomatous and cholesteatomatous chronic otitis media. The former is usually associated with central perforation and the latter with posterior superior marginal perforation. It is important to make the distinction at the initial visit, so that appropriate treatment can be instituted immediately.

Cholesteatoma

Cholesteatoma is accumulated desquamated squamous epithelium in the middle ear cleft. Under normal circumstances squamous epithelium is present only in the outer layer of the tympanic membrane and the skin of the external ear canal. No squamous epithelium is present in the middle ear cleft.
The underlying pathophysiology in

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cholesteatoma formation is Eustachian tube dysfunction.
With occlusion of the Eustachian tube a negative pressure is created within the middle ear cleft, resulting in medial retraction of the superior posterior part of the tympanic membrane (pars flaccida) into the epitympanum (attic). The squamous epithelium in the retraction pocket continuously desquamates and if successful outward migration does not occur, the squamous epithelium accumulates in the pocket and this is referred to as cholesteatoma. As more desquamated squamous epithelium accumulates, the retraction pocket gets larger and extends into the mastoid antrum. Cholesteatoma is considered a serious condition because it promotes the growth of bacteria and releases proteolytic enzymes, which cause bone resorption. Both these factors are responsible for the spread of the infection within and outside the temporal bone, resulting in complications associated with chronic otitis media.

The diagnosis of cholesteatoma is clinical, the presence of whitish, cheese-like material is diagnostic. The most common site is the attic and a polyp in the ear canal often heralds the presence of cholesteatoma.

The treatment of choice for cholesteatoma is complete surgical excision of the cholesteatomatous sac by a modified radical mastoidectomy. There is no place for conservative treatment.

Management of a non-cholesteatomatous discharging ear

The treatment aim is to dry the ear so that spontaneous healing of the tympanic membrane can take place. There are four sequential steps to achieve a dry ear:

- Step 1. Antibiotics, aural toilet, and application of eardrops. The 10-day antibiotic treatment consists of amoxicillin, co-amoxiclav or cefuroxime. Aural toilet must be performed twice daily with cotton wool and sticks (commercially manufactured cotton wool buds are not recommended). Immediately after dry mopping, ear drops containing 0.5% phenol must be applied. This treatment must be continued for 1 month. Patients in whom this treatment fails are moved to the next step.
- Step 2. Remove possible predisposing factors. Tonsillectomy, adenoidectomy and bilateral antral wash-out must be performed. Radiograph of the paranasal sinuses and lateral neck may be requested to confirm or exclude sinusitis and/or enlarged adenoid glands. Remember that radiological absence of adenoid gland hypertrophy does not exclude chronic adenoiditis. After removal of predisposing factors, continue with aural toilet and application of eardrops for 1 month. If otorrhoea fails to subside, then move to the next step.
- Step 3. Intravenous antibiotic therapy. A pus swab is taken and ampicillin (50 mg/kg/d) with metronidazole (20 mg/kg/d) are administered intravenously for 48 hours. The response is reviewed. If the otorrhoea is subsiding, then continue with the treatment another 8 days. If the otorrhoea remains unchanged check the culture and sensitivity results of the pus swab and prescribe appropriate antibiotics and continue treatment for 10 days. Patients in whom otorrhoea fails to subside are moved to the next step.
  The success rate of drying the ear with conservative treatment (steps 1, 2, and 3) is 90%.
- Step 4. Mastoidectomy is the surgical procedure for eradicating disease from the mastoid bone. It involves exenterating all the diseased mastoid bone with a micro-surgical drill and removing infected granulation tissue from the middle ear, attic and mastoid cavities. Simple or cortical mastoidectomy is recommended for the non-cholesteatomatous ear.

Mastoidectomy is a surgical procedure to eradicate disease from the middle ear cleft and basically two types are recognised — cortical and modified radical mastoidectomy. Cortical mastoidectomy is indicated for non-cholesteatomatous chronic otitis media and modified radical mastoidectomy for cholesteatomatous chronic otitis media.

The anatomical difference between the two is that in the modified radical mastoidectomy the bony partition between the external ear canal and the mastoid cavity is excised completely while in the cortical mastoidectomy it is retained. The functional difference between the two procedures is that the modified radical mastoidectomy is associated with approximately 60 decibel hearing loss.

Mastoidectomy can be performed at any age depending on the indication. **Tympanoplasty** is a surgical procedure to restore hearing and it ranges

from a simple myringoplasty (reconstruction of the tympanic membrane) to more complex ossiculoplasty (reconstruction of the ossicles).

Unlike mastoidectomy, tympanoplasty cannot be performed at any age. It must be performed at an age when the conditions are optimal to give the best results. The recommended age for tympanoplasty is 10 years and older because after this age not only does the incidence of upper respiratory tract infection decrease but, more importantly, the Eustachian tube function improves.

Complications are commonly associated with chronic rather than acute otitis media (see Table IV). They can be divided into extracranial and intracranial complications.

The most common extracranial complication is post-auricular subperiosteal abscess (mastoiditis). Facial palsy is a rare complication of chronic suppurative otitis media; if present then TB mastoiditis must be suspected. The treatment for all extracranial complications is urgent intravenous antibiotic therapy, consisting of ampicillin 60 mg/kg/d, metronidazole 20 mg/kg/d and mastoidectomy performed as soon as possible, preferably within 12 hours of presentation.

Intracranial complications must be suspected in all patients who present with severe headaches, nuchal rigidity or localising neurological signs with chronic discharging ears. The investigation of choice is computed tomography scan of the brain. The treatment for all patients with otogenic intracranial complications is intravenous antibiotics (ampicillin 60 mg/kg/d, metronidazole 20 mg/kg/d, and chloramphenicol 30 mg/kg/d), mastoidectomy and surgical drainage of the intracranial abscess. The neurosurgical procedure and mastoidectomy must be performed under the same anaesthesia with the neurosurgical procedure always preceding the mastoidectomy. Surgery must be undertaken as soon as possible, not later than 12 hours after presentation.[3]

Otogenic intracranial complication is a serious condition with a mortality of 10 - 30%.

Tuberculous otitis media is a disease of children — 80% are younger than 10 years of age. The typical clinical features include painless and profuse otorrhoea, multiple tympanic membrane perforations, pale granulations, lower motor neuron facial palsy (40% incidence), disproportionate hearing loss, and bone necrosis with sequestra formation. Evidence of TB in other sites reinforces the diagnosis, e.g. pulmonary TB (95%) and preauricular lymphadenopathy (23%). The diagnosis is confirmed on histology of granulation tissue biopsied either from the middle ear space or mastoid cavity.[4] The treatment is antituberculous therapy for a minimum period of 6 months.

References available on request.

IN A NUTSHELL

The tragus, the tortuous S-shape course of the ear canal and the obliquity of the tympanic membrane offer protection to the delicate middle ear structures.

The underlying pathophysiology of otitis externa is maceration of the skin of the external ear canal. The hair and lipid content of cerumen renders the ear canal impervious to water, thus preventing infection.

Wax is protective and should not be removed. It contains lysozyme and immunoglobulins that inhibit the growth of bacteria and fungi.

Four types of otitis externa are recognised, non-infective, fungal, bacterial and diabetic.

Diabetic otitis externa is a life-threatening condition and in order to prevent mortality early diagnosis and treatment with intravenous triple antibiotics is mandatory.

The causative organisms in acute otitis media are *Haemophilus influenzae* and streptococcus and the treatment of choice for children living in rural areas is procaine penicillin and amoxicillin and for those living in urban areas either Augmentin or cefuroxime.

Two types of chronic otitis media are recognised — cholesteatomatous and non-cholesteatomatous chronic otitis media.

Cholesteatomatous chronic otitis media is regarded a serious condition because cholesteatoma releases proteolytic enzymes that cause bone resorption with extension of infection into the bony labyrinth, facial canal and intracranially.

Chronic otitis is regarded as a life-threatening condition because of its association with intracranial complications, which carries a mortality of 10 - 30%.

Tuberculous otitis media is common in children. The golden rule is that any child who presents with ipisilateral otorrhoea and lower motor neuron VII cranial nerve palsy has tuberculous otitis media until proven otherwise.