ACUTE SUPPURATIVE OTITIS MEDIA

Definition

Otitis media is an inflammation of part or all of the mucosa of the middle-ear cleft, the collective term for the Eustachian tube, tympanic cavity, attic, aditus, antrum and mastoid air cells.

Classification

It may be classified as acute or chronic with the suffix suppurative or non-suppurative. A third category, 'specific otitis media' has been used to describe tuberculous and syphilitic otitis media as it may present acutely or chronically, with or without suppuration and a fourth category namely 'adhesive otitis media' has been used to describe tympanosclerosis (hyaline degeneration and calcification) and adhesion formation within the tympanic cavity but should more accurately be regarded as a sequelae of otitis media.

Acute suppurative otitis media (ASOM)

Aetiology

This is a bacterial disease caused by pus forming organisms, *Streptococcus pneumoniae* (40%), *Haemophilus influenzae* (30%) and *Branhamella (Moraxella) catarrhalis* (10%) being the most commonly implicated. It may occur as a primary or a secondary infection after a viral acute non-suppurative otitis media. Bacteria enter the middle-ear cleft via the Eustachian tube, a perforated tympanic membrane or more rarely be blood-borne. Infants have a short, wide more horizontally placed Eustachian tube, allowing contamination from the regurgitation of feed and when actively vomiting. Teething increases the incidence of infection. Poor sanitation and hygiene, overcrowding and malnutrition are predisposing factors. Children aged 3–7 years have the highest incidence, direct extension from a bacterial or secondary to a viral upper respiratory tract infection being the most common aetiological factor. Risk factors in all age groups are recurrent or chronic rhinosinusitis, adenoiditis, chest disease and Eustachian tube dysfunction. Causes of the latter are nasopharyngeal tumours including adenoidal hypertrophy, abnormal Eustachian patency, cleft palate and submucous cleft palate. Pathogenic bacteria have been isolated from the nasopharynx in up to 97% of children with ASOM.

Clinical features

The two main symptoms are:

• *Pain*, which may increase rapidly in intensity to become deep and throbbing.

• *Deafness,* initially described as a blocked ear and secondary to Eustachian tube dysfunction. Deafness progresses as suppuration occurs and both symptoms may rapidly improve if rupture of the tympanic membrane produces a mucopurulent otorrhoea. The initial event after infection is mucosal oedema causing Eustachian tube occlusion and a dull tympanic membrane on examination. Hyperaemia rapidly follows and leashes of vessels may be seen running along or parallel to the malleus handle. Soon radial vessels are visible on the drumhead and a middle ear effusion occurs. The drumhead takes on a full (i.e. opposite to retraction), red, angry appearance and pus may be seen bulging postero-inferiorly. Pressure necrosis of this region may cause the drumhead to rupture allowing mucopus to drain into the external ear canal. Children are usually fretful with a high pyrexia (> 39°C) and there may be signs of complications of ASOM.

Treatment

1. *Rest* in a warm, well humidified room.

2. *Antibiotics* in an adequate dose administered until resolution. Amoxicillin will cover the most common pathogens provided the patient is not allergic to penicillin and the organisms are not resistant, the latter occurring in about 14% of cases but resistance is increasing. In these circumstances, gleaned from bacterial sensitivity studies or lack of clinical response, a -lactamase resistant antibiotic should be chosen, for example coamoxiclav. Oral medication is adequate in the absence of complications.
3. *Systemic and topical decongestants* have a theoretical adjuvant role; they have not been proven to be of significant value.
4. Conditions predisposing to ASOM should be treated on their own merit after resolution.

Complications

• *Mucositis may progress to an osteomyelitis,* namely acute mastoiditis, if the mastoid air cells are affected and acute petrositis should the petrous apex become involved. Gradenigo's syndrome, comprising signs of ASOM, an ipsilateral abducent nerve palsy causing paralysis of the external rectus and pain in the distribution of the ipsilateral trigeminal nerve, is a classic feature of petrositis. The respective cranial nerves are only separated from the petrous apex by a layer of dura so that an extradural abscess or pachymeningitis (meningitis extending to the dural layer) of this region from a generalized meningitis may also cause the combined cranial nerve signs.
• *Meningitis.*
• *Citelli's abscess* (a subperiosteal abscess which has spread through the medial aspect of the mastoid, into the digastric fossa) or *Bezold's abscess* (an abscess which has tracked inferiorly within the sheath of sternomastoid to form a fluctuant mass along its anterior border).
• *Extradural and subdural abscess.*
• *Cerebellar, temporal lobe and perisinus abscess.*
• *Lateral sinus thrombosis,* rarely extending in an antegrade direction to thrombose the internal jugular vein and in a retrograde direction causing a cavernous sinus thrombosis.
• *Otitic hydrocephalus.*
• *Lower motor neurone facial nerve paralysis.* The population at-risk (6%) are that group of patients with a congenital dehiscence of the horizontal portion of the facial nerve.
• *Serous and suppurative labyrinthitis.*

Sequelae of ASOM

• *Non-suppurative middle-ear effusion.* These persist for over 30 days in 40% of children and for over 3 months in 10%.
• *High-tone sensorineural hearing* loss, perhaps secondary to bacterial toxins migrating across the round window.
• *Tympanic membrane perforation.*
• *Adhesions* between the tympanic membrane, ossicles and the medial wall of the middle ear.
• *Tympanosclerosis* which may spread from the tympanic membrane to the ossicular chain, fixing the latter.
• *Erosion of the ossicular chain,* in particular the long process of the incus, especially following recurrent episodes of ASOM.
• Sequelae of ASOM complications.