Personal Practice

Acute Otitis Media

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Acute otitis media produces inflammation of the lining mucosa of middle ear. It can extend upto the mastoid antrum and its cell system(l). The disease occurs frequently in infants and children. Different population based studies(2-7) have indicated that children, by the age of 6 years, get at least one episode of otitis media in 76 to 95% of cases. If an infant gets a middle ear infection before the age of one year, about 50% of such children are susceptible to chronic otitis media later. According to Teele(3), by the age of three, 83% of children had experienced one or more episode of acute otitis media and 46% 3 or more episodes. The peak incidence occurs in the preschool children, often before the age of two years. It occurs more commonly in cold and temperate climates and in winter season and also in the poor population with lower hygienic and nutritional conditions(2,5).

Etiology

Infection spreads to the middle ear most commonly through the eustachian tube which connects the middle ear to the nasopharynx and nose. The eustachian tube is relatively short, wide and straight in the infant and young children. The infected material from the nose, nasopharynx and paranasal sinuses can pass easily along the eustachian tube, specially during coughing sneezing, vomiting and feeding in the supine position. This period of tubal incompetence also coincides with the period of increased susceptibility of a child to viral upper respiratory tract infection. The adenoids tend to block the eustachian tube and act as a focus of infection (l,2,6,7). Other nasopharyngeal masses like teratomas, angiofibromas and antrochoanal polyps may also contribute in the same way. Chronic rhinitis and sinusitis provide a ready source of infection which can travel up the eustachian tube. Edema of the eustachian tube in allergic patients may contribute to tubal obstruction and the consequent recurrent attacks of acute otitis media. Similarly, a pre-existing middle ear effusion may act as a culture medium for the invading pathogens (6,7). Children with cleft palate have a higher incidence of both serous and acute otitis media owing to tubal dysfunction. Repeated viral upper respiratory tract infections often result in bacterial infection causing otitis media. Infection may also occur through the tympanic membrance(l). This may be through a preexisting perforation during bathing and hair washing, in which case the patient has acute infection superimposed on chronic otitis media. The infection may also occur due to traumatic perforation by an unsterile object, or following operative interventions like myringotomy or through a grommet, (ventilating tube) used for middle ear effusion and foreign body removal(l,7).

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Bacteriology of Otitis Media

The most common organisms isolated in acute otitis media are *Streptococcus pneumoniae* and *Haemophilus influenzae,* followed by beta hemolytic *Streptococci, Staphylococcus aureas* and Neisseria *Catarrhalis.* Gram negative bacilli like, *Pseudomonas aeruginosa, Klebisiella pneumoniae* and *Proteus* species have also been isolated, more commonly in acute on chronic infection(l,3,5,7).

Pathological Aspects

The inflammatory process consists of middle ear mucosal edema with increased secretion, hyperemia, hypersecretion and pus formation. Mucosal edema contributes to tubal occlusion leading to impaired drainage. Ciliary destruction that occurs in the inflammatory processes(8,9), also impairs drainage. The mastoid air cells, being in continuity with the middle ear are almost always involved. If there is breakdown of the bony walls of the air cells, it can lead to coalescent mastoiditis(l). Accumulated pus in the tympanic cleft under pressure may lead to the rupture of tympanic membrane. Spread of infection can occur owing to retrograde thrombophlebitis, bone necrosis, congenital dehiscences, and existing fracture lines, leading to various complications of otitis media. These include subperiosteal abscesses in the post-aural region, neck abscess, suppurative labrynthitis, facial paralysis, petrositis and petrous apex infections. Intracranial complications such as meningitis, extradural, subdural and brain abscesses, lateral sinus thrombosis and otitis hydrocephalus can also occur in otitis media(l-7).

Clinical Features

The presentation may vary from a minor short lasting (few hours) episode of ear ache with hyperemic tympanic membrane during an upper respiratory tract infection to fulminating illness with life threatening complications(l,2). The usual *common* symptom is pain in the affected ear which may be localized deep inside the ear. It is usually described by the patient as a throbbing or pulsating pain. A younger child would be repeatedly tugging at his or her ear and becomes more irritable. This is usually preceded by a viral upper respiratory tract infection. Deafness in the affected ear may be noticed. Impaired hearing becomes more evident when it occurs bilaterally. These symptoms could gradually resolve or intense pain may occur which is usually followed by rupture of the eardrum leading to mucopurulant discharge with relief from pain.

The earliest sign on otoscopy is hyperemia. It occurs along the handle of malleus, the periphery of the drum, and the pars flaccida. This spreads to the whole of the tympanic membrane as the disease progresses. When pus starts to accumulate, the tympanic membrane becomes opaque or yellowish and begins to bulge, mainly posteriorly. If the eardrum has ruptured, mucoid to mucopurulant discharge is seen. A small central perforation may be visualized on cleaning out the pus, but it is often missed as the edematous mucosa of the middle ear tends to obscure the gap. The perforation, however, can be elicited by pneumatic inflation(l-9). Tuning fork test indicates a conductive deafness. Mastoid tenderness may be present fairly early, but can assume significance if it persists despite definite treatment. The nose and throat would show signs of an upper respiratory tract infection. Pyrexia and other systemic signs of infection may be present and seen in a pronounced manner in younger children and neonates.

Signs and symptoms of intracranial compilations' should be diligently excluded. Persisting tenderness and edema of the mastoid, sagging of the posterosuperior canal wall, granulations protruding through the tympanic membrane perforation and discharge,

continuing for over three weeks indicate mastoiditis(l,2,7).

During resolution, the hyperemia fades away and the perforation most often heals on its own, sometimes, leaving a thin scar or a white patch on the tympanic membrane.

Investigations

No investigation is really mandatory in acute otitis media(l). When otorrhea is present, an ear swab for culture and sensitivity should be taken prior to starting antibiotic treatment. If there is no ear discharge, a nose and throat swab would probably yield the same organisms. A blood count, including differential count is helpful, especially in patients with suspected complications. A rising blood count suggests that pus has accumulated somewhere in the body. Pure tone audiometry may be done once the severe pain and fever have subsided. It would usually indicate a conductive deafness, which would serve as a baseline for reference future(10-13). Tympanometry would not be very useful in the acute stage. Mastoid X-rays are useful if mastoiditis is diagnosed(l). Clouding of air cells is invariably present. Destruction of bony walls of the air cells suggests a coalescent mastoiditis which indicates the need for surgical intervention.

Diagnosis and Differential Diagnosis

The diagnosis of acute otitis media is a fairly straightforward clinical decision based on a history of otalgia and deafness coupled with the findings of an inflamed eardrum on examination. However, at times the condition needs to be differentiated from other causes of otalgia such as otitis externa, impacted wax, tympanic membrane hyperemia due to crying, myringitis, and referred otalgia caused by tonsillitis or dental infection. In otitis externa, discharge is usually non-mucoid and hearing is only slightly impaired while itching is a common feature. At times, if the child is not gently examined, the eardrum may appear red owing to the hyperemia caused by excessive crying resulting in confusion with otitis media. Impacted wax may also cause pain and deafness. The presence of upper respiratory tract infection is a clinical pointer towards otitis media in such a situation if the tympanic membrane cannot be evaluated. The condition needs to be treated as otitis media only apart from using wax softeners.

Treatment

General medical management as for any acute febrile condition needs to be done(l). This includes rest, adequate nutrition and hydration, analgesia and antipyretics. *Antibiotics* are indicated fairly early in the course of the disease(14-17), despite the fact that a proportion of cases may be of viral origin. Routine early use of antibiotics has led to a significant decline in complications associated with acute otitis media and the concomitant morbidity and mortality(7). In most cases, the oral route of administration is sufficient and the antibiotic should be continued for 5-10 days. The antibiotic should preferably be started after taking an ear swab(l,5). Amoxycillin is the preferred drug(15). Erythromycin, cotrimoxazole and cefaclor are also used. Severe and complicated cases may require hospitalization and intravenous use of antibiotics, such as ampicillin, flucloxacillin and metronidazole(7,ll). Recently a combination of amoxycillin with clauvunate is preferred; its penetration in middle ear periosteum is effective. *Systemic decongestants* like pseudoephedrine are often prescribed, but have not proved better than placebos. These drugs may cause excitement and psychotic symptoms in some children and

it is better to avoid them. *Topical nasal decongestants* like oxymetazoline and xylometazoline have also been used in the belief that they would help open up the blocked eustachian tube(7,18). These can be used for 5-7days in doses of 3 drops 2-3 times per day. They help significantly to control the associated upper respiratory tract infection. In case there is ear discharge, this should be gently mopped or sucked out from the ear canal to prevent excoriation of the ear canal skin and a cotton wool may be kept in the orifice. Different topical antibiotic drops like chloromycetin, gentamycin and neosporin are fairly commonly used in practice although they are not at all necessary.

Surgery

In case the patient does not respond to medical management and pus accumulates under pressure in the middle ear behind an intact tympanic membrane, myringotomy(15) may be required to release the pressure inside. Such an action would prevent a spontaneous rupture of the ear drum and thereby a permanent perforation would be avoided. In children, myringotomy is carried out under general anesthesia(l,7). Using full aseptic precautions, under the operating microscope, with an angled myringotome, a "J" shaped incision is made in the tympanic membrane usually in the bulged portion or in the posteroinferior quadrant. At this site, damage to the ossicular chain is avoided, and the drum heals fairly rapidly. The shape of the incision prevents the hole from closing up immediately, thus facilitating adequate drainage. The pus usually gushes out on myringotomy and is then gently sucked out because a forceful suction can cause sensorineural hearing loss. A culture and sensitivity of the pus should be done. Ventilating tubes should not be inserted in acute otitis media, but may be indicated later for persistent middle ear effusion, which is one of the sequelae of acute otitis media. Adequate antibiotics are recommended to continue even after myringotomy and evacuation of pus in the middle ear.

Prevention

Preventive measures need to be taken in children by early treatment of viral upper respiratory tract infection and adopting appropriate measures in feeding infants and children. Adequate treatment for nose, sinus and nasopharyngeal infection is also essential. Recurrence may be prevented by removal of adenoids, if they are considered to be the origin of infection. X-ray soft tissue neck including nasopharynx to evaluate adenoids is essential in such a situation before undertaking adenoidectomy(7).

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NOTES AND NEWS

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These events are being organized by the Ranchi Branch of IAP on the 16th, 17th and 18th February, 1997 at the Indian Institute of Coal Management, Ranchi. The Registration forms are being individually posted to all the IAP members from the East Zone. For further details please contact: Dr. Krishna Kumar, Organizing Secretary, East Zone IAP Conference, 50 Doctors Colony, RMCH, Ranchi-834 009 (Bihar). Tel. No. (0651) 207443, 310340; Fax (0651)205144.