### **TUBERCULOUS OTITIS MEDIA – REVISITED WITH 3 INTERESTING CASE REPORTS**

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**ABSTRACT:** Tuberculous otitis media is a rare disease that is clinically variable and nonspecific. Tuberculous otitis media can be difficult to diagnose because it can easily be confused with other acute or chronic middle ear conditions. The signs and symptoms are variable and nonspecific and often differ from classic descriptions. Furthermore, no two cases may not necessarily present itself clinically in the same manner. Cases of chronic otitis media that are unresponsive to the usual therapy or show unexpected postoperative evolution should be investigated for tuberculosis. Tuberculous otitis media should be suspected after failure of current antibiotics or persistent effusion after tympanoplasty or mastoidectomy. Because of these factors, the diagnosis is often made during surgery or postoperatively. Late diagnosis delays the start of treatment, thereby increasing the risk of complications.

KEY-WORDS: Tuberculous otitis media, chronic otitis media, mastoidectomy

**Key Messages:** Tuberculous otitis media is a rare disease, which still persists in clinical practice despite effective antitubercular treatment. It may have varied modes of presentation making early diagnosis difficult. If left undiagnosed can cause significant damage to middle ear and other surrounding structures

**INTRODUCTION:** Tuberculosis remains the leading cause of death secondary to infectious diseases worldwide in persons older than 5 years. Tuberculosis of middle ear is a comparatively rare entity usually seen in association with or secondary to pulmonary tuberculosis. Tuberculosis is one the major infectious disease with predominant involvement of lung and lymph nodes but tuberculosis of the middle ear is uncommon. Tuberculous otitis media is generally considered a disease of children and young adults, as patients <15 years of age account for 84% of all cases.

**Case report 1:** A 19 year old female came to our institution with a history of right ear discharge since 4 yrs which was yellowish, scanty, continuous, foul smelling and non blood stained. She was treated with local and systemic antibiotics but her discharge did not reduce with medications. She also complained of decreased hearing on the right side. She had a history of abdominal Kochs 3yrs back for which she took AKT (anti Koch treatment) for 6 months. One year back she had a tubercular gluteal abscess which was drained under spinal anaesthesia. She took medications (AKT) for 9 months for the latter.

Examination revealed a central perforation with granulations in middle ear. Ear swab for culture and sensitivity showed no bacterial growth. Her pure tone audiogram showed right sided severe mixed hearing loss. Schuler's view X-ray mastoid showed loss of pneumatization of right mastoid region. CBC, blood urea and electrolytes were insignificant. Chest x-ray was unremarkable.

Patient was operated and intra-operatively granulations were seen in the middle ear which was sent for histopathology. Intra-operatively another ear swab was collected for acid fast bacilli (AFB), culture and sensitivity and Zeil Nelson staining which returned as negative. Conchal cartilage was used as a middle ear spacer and type I tympanoplasty was done. Histopathology report of the middle ear granulations revealed chronic granulomatous inflammation with epitheloid giant cells consistent with tuberculosis (figure 1). Postoperatively, the patient had an uneventful recovery from surgery, was discharged home and monitored closely on regular follow-up visits.

**Case report 2:** A 23 year old male patient presented with the chief complaints of severe throbbing headache associated with severe nausea and giddiness. Patient gave a history of long standing scanty, purulent and occasionally blood stained discharge from the right ear. Clinically the patient looked toxic with high grade fever. Otoscopy revealed granulation tissue in the attic region and pure tone audiometry reported a profound hearing loss. HRCT Temporal bone with contrast showed signs suggestive of chronic otitis media with mastoiditis and the MR venography reported thrombosis of the Right Transverse and the sigmoid sinus (figure 2). A radical mastoidectomy was performed which revealed a dehiscent sinus plate with perisinus abscess (figure 3). Intra-operatively, granulations from the middle ear were sent for biopsy which revealed langerhans giant cells with chronic granulomatous inflammation suggestive of tuberculous otitis media. Appropriate antitubercular treatment was started, patient was discharged and called for regular ENT follow ups.

**Case report 3:** A 14 year old adolescent male came to our institution with complaints of headache and giddiness along with a history of right ear purulent, scanty and continuous discharge since 6-8 months. On examination the patient showed signs of lateral rectus palsy (figure 4). Otoscopy findings revealed granulations over the tympanic membrane which were then examined under a microscope and sent for biopsy. Biopsy reports revealed a chronic granulomatous lesion with multinucleated langerhans giant cells indicating tuberculous infection. HRCT temporal bone showed signs of mastoiditis with petrositis (figure 5). Other routine investigations including CBC, BUN, x-ray chest were insignificant. The patient was started on appropriate antitubercular therapy and a close follow-up was kept for signs of improvement.

**DISCUSSION:** Tuberculous otitis media (TOM) is caused by Mycobacterium tuberculosis, of which bovis and hominis generally affect the ears. The pathogenesis of tuberculous otitis media involves one of three major mechanism viz. primarily, aspiration of mucus through the eustachian tube, blood-borne dissemination from other tuberculous foci, or in rare cases, direct implantation through the external auditory canal and a tympanic membrane perforation.<sup>1</sup>

Generally tuberculosis of middle ear is unilateral, characterized by painless otorrhoea which fails to respond to the usual antimicrobial treatment, in a patient with evidence of tubercle infection elsewhere. The classic symptoms include multiple tympanic membrane perforations, abundant granulation tissue, bone necrosis, and preauricular lymphadenopathy. Most patients also experience an associated acute-onset hearing loss that is disproportionate to the extent of their disease<sup>1,2</sup>. The hearing loss in tubercular otitis media can be either conductive (90% of patients), sensorineural ( $\sim$ 8%), or mixed ( $\sim$ 2%)<sup>2,3</sup>. Characteristically, there is little or no otalgia. Infiltration of tubercules into the nerve endings leads to neuropathic manifestations like anaesthesia, paraesthesia, etc, thus,

leading to painless otorrhoea. The otorrhoea, unlike seen in suppurative otitis media, is not due to a built up of tension but rather due to the granulations formed over areas of tympanic membrane as a result of the tubercular process. Examination of the affected ear will usually reveal pale-yellow granulation tissue on a thickened and hyperemic tympanic membrane<sup>1,4</sup>. Multiple foci of tubercle bacilli lead to formation of multiple submucosal granulomas over the tympanic membrane. Perforations usually occur in the areas of tubercules early in the disease as a result of caseous necrosis giving rise to the classical picture of multiple perforations<sup>4</sup>. There may be multiple perforations in the early stages, but they coalesce into a total tympanic membrane perforation accompanied by a pale granulation tissue<sup>1</sup>. In early stages of tuberculous otitis media, the drum looks dull and some dilated vessels can be observed. The tympanic membrane then becomes thickened and landmarks are obliterated.<sup>3</sup> The exudate in the middle ear may be thick and is sometimes confused with the infected keratin debris of a cholesteatoma. The consistency of the discharge ranges from thick and mucoid to thin and watery.<sup>1</sup> The granulomatous process often produces a visible destruction of the ossicles, which can be seen through the tympanic membrane perforation<sup>1,5,6</sup>. As the disease progresses, the amount of granulation tissue can become profuse and lead to an attic-antral blockage. A direct extension of the mucosal disease can lead to mastoiditis or tuberculous osteomyelitis of the temporal bone. Periauricular fistulas, lymphadenopathy, and facial palsy are infrequent findings. Late complications include facial paralysis, labyrinthitis, postauricular fistulae, subperiosteal abscess, petrous apicitis, and intracranial extension of infection. Facial nerve palsy has been reported in cases of tuberculosis otitis media even if the anti tuberculosis therapy has been started.<sup>3,7,8</sup> Tuberculous otitis media is more likely to cause infection of the labyrinth than the usual purulent forms of otitis<sup>3</sup>.

However, it might be prudent to remember that tuberculous otitis media more often than not may have an unusual presentation. In most cases, only a high degree of clinical suspicion may lead towards an accurate diagnosis. As demonstrated above by our three case reports, tuberculous otitis media could simply present as otorrhoea not responding to long-term antibiotics, or could also present with complications like lateral sinus thrombophlebitis or even petrositis. The latter two cases might have gone undiagnosed as TBOM if the intraoperative granulations found in the middle ear had not been subjected to appropriate tubercular testing.

Relevant investigations include pure tone audiometry to assess degree of hearing loss, x-ray or preferably computerized tomography (CT) of mastoid bones, and more importantly bacteriology and histopathology of the biopsy, aural discharge or aspirate of the middle ear.<sup>9,10,11</sup>

Antitubercular therapy is the mainstay treatment for tuberculous otitis media with any mode of presentation. The current standard therapy (DOTS-category-1) includes a 4 drug regimen in first two months (Isoniazid, Rifampicin, Pyrazinamide and Ethambutol) followed by a 2 drug regimen in later 4 months (Isoniazid and Rifampicin). Surgical intervention is added to drug therapy and is the type of surgery depends on the clinical presentation. More radical surgical procedure may be required in cases of complications such as facial paralysis, subperiosteal abscess, labyrinthitis, mastoid tenderness and headache.<sup>1</sup>

**CONCLUSION:** Tuberculous otitis media is a rare disease, which still persists in clinical practice despite the advent of effective antitubercular treatment. It may have varied modes of presentation making early diagnosis difficult If left undiagnosed and therefore, untreated, it can damage middle

ear and other surrounding structures. It should be considered in differential diagnosis of chronic middle ear discharge that does not respond to usual therapy. Delay in diagnosis can lead to complication. A high level of clinical suspicion is needed for early diagnosis and antitubercular therapy should be started as soon as possible to prevent the possible complications.

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Fig. 1: High power microscopy of biopsy from middle ear granulations illustrates multinucleated langerhans giant cells indicating a granulomatous



Fig. 2: Illustrates MR Venography showing right sigmoid sinus thrombosis.



Fig. 3: Intra-operative picture of pus pouring out from the perisinus (sigmoid) abscess during modified radical mastoidectomy for complicated tuberculous otitis media.



Fig. 4: Illustrates right lateral rectus palsy complicating tuberculous otitis media.



#### Fig. 5: Illustrates HRCT temporal bone showing right petrositis.

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