A Rare Case of Tubercular Cholesteatoma with TB Meningitis

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Abstract

Tubercular otitis media is an uncommon condition. Tuberculosis can affect any part of ear ranging from tympanic membrane to labyrinth. The symptoms of tubercular otitis media like painless otorrhea, multiple perforations, pale granulations, facial paralysis and severe SNHL though well described in literature, are not always present hence diagnosis is often missed. Diagnosis is usually made by clinical and histopathology examination of specimen obtained intra operatively. Here, we discuss clinical presentation, diagnosis and management of a case with cholesteatoma and TB meningitis.

Keywords

Tuberculosis, Cholesteatoma, Meningitis

1. Introduction

The incidence of tubercular otitis media ranges from 0.05% to 0.9% of all cases of chronic otitis media [1]. This is one of rarest forms of extrapulmonary tuberculosis and is often a missed diagnosis. There are no definite or pathognomic clinical signs and symptoms, making the diagnosis difficult in absence of laboratory findings [2].

Cholesteatoma in middle ear is not uncommon but presence of tubercular bacilli makes it a rare presentation. Tuberculosis should be included in the differential diagnosis of chronic middle ear infections not responding to routine management. We present an interesting case of tubercular otitis media with intracranial complication.

2. Case report

43 years old male presented to us with complaints of fever and left ear discharge for 20 days. He had history of similar discharge from left ear for which he un-
derwent surgery 10 years back. He also gave history of right ear surgery for discharge 5 years back. This Patient was receiving treatment by physician for last 20 days with no significant improvement. Patient also had hypertension for last 4 years and Diabetes Mellitus for 5 years for which he was on regular medication. On our examination, patient was febrile with PR 98/min and BP 150/100 mm Hg. There was associated neck pain and rigidity. Kernig’s sign was positive. Ophthalmologic examination showed right lateral rectus palsy. Detailed ENT examination was done which showed bilateral inadequate conchomeatoplasty. Right ear had healthy dry mastoid cavity with mobile neotympanum while left ear showed wet cavity with purulent discharge and cholesteatoma debris. Os sicles were not visualised. Facial nerve function was normal bilaterally. Pure tone audiometry showed 63.3 dB conductive deafness on right side and 65 dB mixed deafness on left side.

Considering the history that meningitis did not respond to high dose antibiotics (I/V Ceftriaxone 2 gm 8 hourly, IV Vancomycin 1 gm 12 hourly, IV Metrogyl 100 ml TDS) we suspected the disease to be of non pyogenic cause. MRI brain with venogram was done, which showed evidence of 1.3 × 1.6 cm thick walled peripherally enhancing abscess in left cerebellum with mild surrounding oedema (Figure 1). Left sigmoid sinus was compressed, however patent. CSF examination was done which was suggestive of chronic meningitis. Hence patient was planned for urgent left ear mastoid exploration. Revision mastoidectomy done. Erosion of horizontal and posterior semicircular canal noted. Facial nerve canal was dehiscent near second genu. There was large dehiscence of Posterior fossa dural plate. We were able to drain the posterior fossa extradural abscess through Trautman’s triangle. Pus, pale granulation tissue and cholesteatoma sac obtained intra operatively was sent for culture sensitivity, AFB and histopathology examination.

Pus culture and cholesteatoma matrix showed Staph aureus and AFB in many fields. Histopathology examination also showed presence of caseating granulomas with AFB in matrix (Figure 2). Fluorescent staining of cholesteatoma matrix was done, which also demonstrated AFB (Figure 3). Hence diagnosis of cholesteatoma with TB and TB Meningitis was made. AKT was started. Patient was followed up after 15 days. Symptoms dramatically improved with marked reduction in neck rigidity and diplopia. Lateral rectus palsy recovered fully within 3 weeks post operatively.

3. Discussion

Tubercular otitis media is a very unusual cause of chronic otitis media, and is rarely considered in the differential diagnosis. Although the pathogenesis of TOM is still controversial, three mechanisms explaining middle ear tuberculosis infection have been postulated: aspiration of mucus through the auditory tube, hematogenous transmission from other tuberculosis foci and direct implantation through the external auditory canal with tympanic membrane perforation [3] [4] [5].
Figure 1. MRI brain showing 1.3 × 1.6 cm thick walled peripherally enhancing abscess in left cerebellum with mild surrounding oedema.

Figure 2. H & E staining of cholesteatoma matrix showing caseating granulomas with AFB.

Figure 3. Fluorescent staining of cholesteatoma matrix showing AFB.
Like any other disease, complications of tubercular otitis media occur when there is delay in diagnosis. Complications can be intracranial or extracranial. Intracranial complications include meningitis, abscess (subdural and extradural) and otitic hydrocephalous. Intracranial extension of the disease can be direct through erosion of bony anatomical barrier that is tegmen tympani or hematogenous along sigmoid sinus.

Here, our patient had minimal vestibular symptoms despite erosion of lateral and posterior SCC. There was no facial palsy despite significant dehiscence of Facial canal. Also intracranial symptoms were minimal in spite of a large dural defect.

Complications can be prevented only if the diagnosis is made at early stage of the disease. Tuberculosis should be suspected when there are persistent symptoms not responding to routine antibiotics. Clinicians should have strong suspicion especially in a country with high incidence of this systemic disease.

4. Conclusion

The main objective behind this article is to make ENT surgeon vigilant about this rare manifestation of very common disease in the Indian sub continent. Tuberculosis should be included in the differential diagnosis of chronic otitis media not responding to routine therapy or when presenting with intra cranial complication. Early diagnosis and timely initiation of anti tubercular therapy can prevent life threatening complications.

References


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