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Tuberculous Otitis Media With Frontal Lobe Tuberculoma, A Rare Case Report

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I. Introduction

Tuberculosis (TB) caused by the Mycobacterium species bacteria, involves primarily the pulmonary system. It can spread to or involve other parts of the body with or without lung involvement. The term Extra pulmonary TB (EPTB) is used when the disease manifests in parts of the body other than the lung parenchyma. When there is a simultaneous infection of the lung and other body regions, it is categorized as pulmonary TB. The common sites of EPTB include lymph nodes, central nervous system (CNS), genitourinary system, gastrointestinal system, bones, and joints. In rare cases, TB of the skin, and eye are also reported.

This disease is one of the major healthcare issues in India, with an incidence of 210 in 1 lakh population.[1] EPTB makes up 20 percent of all TB cases. Among EPTB the common sites are lymph nodes followed by pleura, accounting for 50 and 24 percent of EPTB respectively. The incidence of TB otitis media is very low with an incidence of 1.5 to 4 percent of EPTB. CNS TB is another rare form of EPTB with an incidence of 1 percent of all TB cases, which roughly equates to 17000 cases all over India. [2]

TB otitis media is usually misdiagnosed or diagnosed late. It represents 0.04 percent of all the cases of Chronic otitis media.[3] The pathogenesis of TB OM has been hypothesized that the infection of the middle ear cleft can occur via three routes which include, entry of the organism via pharyngotympanic tube, hematogenous spread from neighboring or distant sites or direct implantation through an already existing tympanic membrane defect. In the initial acute stage, the manifestation is nonspecific, with clinical findings resembling an acute otitis media. In the later stages, typical clinical signs of TB OM may be noted such as multiple tubercles, multiple central perforations, pale granulations, and painless ear discharge which may not respond to usual antibiotics. The hearing loss will be out of proportion to the clinical picture. The hearing loss can be conductive because of ossicular chain discontinuity, sensory neural because of labyrinth involvement, or mixed hearing loss. Examination under a microscope may reveal pale granulations in the external canal or middle ear with bony sequestrum in the late stages.

CNS tuberculosis is an extremely rare form of EPTB. Symptoms and signs of raised intracranial pressure which include headache, blurry vision vomiting, fever, altered mental status, neural deficits, and fever are the usual presenting features. The disease manifestation varies according to the area of brain involvement. CNS TB has been classified as TB meningitis, spinal or cerebral tuberculoma, myelitis, and arachnoiditis. The key to the management of TB of CNS includes access to rapid CSF analysis, rapid neuroimaging facilities, and early initiation of Anti-tubercular therapy (ATT) and supportive care.

Here authors are reporting a case of EPTB with involvement of middle ear cleft and disseminated CNS involvement.

II. Case Presentation

A 15-year-old male from the town of Muthalamada, Kerala presented to the OPD with complaints of severe continuous headache which was not associated with vomiting, visual disturbances, altered sensorium or gait abnormality or neck stiffness, with no aggravating or relieving factors, since the last 1 month. The patient

also has a history of intermittent episodes of fever without chills more towards the end of the day. 1 month ago he underwent a left-sided modified radical mastoidectomy with type 3 tympanoplasty for complaints of left-sided scanty foul smelling ear discharge for 4 months. On examination, left-sided attic retraction was seen. Intraoperatively extensive pale granulation tissue involving epitympanum, meso tympanum, hypotympanum, and pro tympanum was noted. The malleus and incus were eroded, and the stapes suprastructure was engulfed by the granulation tissue. The patient also has a history of right-sided modified radical mastoidectomy with type 3 tympanoplasty 1 year back (data not available). Contrast enhanced computed tomography imaging of the (CECT) brain showed a partially defined lesion with an irregular margin in the left frontal lobe measuring 3.3X 3.3 X 3.4 cm. No calcifications were noted. On post-contrast, the lesion is heterogeneous with peripheral enhancement. Marked peri lesional edema was seen. Mass effect was seen in the form of effacement of Sylvian fissure, adjacent sulcal spaces, left lateral ventricle, and a midline shift of 10mm at the level of the lesion. Multiple subcentimeter ring-enhancing lesions are seen in the bilateral cerebral hemisphere and right cerebellum with a few showing mild perilesional edema. The intraoperative specimen on histopathological examination showed necrotizing granulomatous inflammation with Langerhans cells and multinucleated giant cells. Lumbar puncture was negative for spinal TB and sputum examination showed no evidence of pulmonary TB on Ziehl- Neelsen staining and Nucleic acid amplification tests. MRI with contrast confirmed multiple tuberculomas and a left temporal craniotomy was performed. the caseous material on biopsy showed TB bacilli, favoring a diagnosis of EPTB presenting with CNS and temporal bone involvement. After consultation with the district TB officer, ATT was initiated according to the National Tuberculosis Control Program category.

III. Discussion

Tuberculosis accounts for only 0.05–0.9% of all chronic otitis media cases [4]. Often, it is secondary to infection in the lungs, larynx, pharynx, or nose [5]. The tubercular bacilli enter the temporal bone via aspiration through the Eustachian tube, hematogenous spread from distant sites, or direct implantation through the external auditory canal and tympanic membrane perforation [6]

CNS-TB accounts for about 5–10% of all patients, with a mortality of up to 20% depending on the clinical stage [7,8]. Even more so, CNS tuberculoma is a rare entity in the pediatric population, and very few cases have been reported [9-11].

Hailing from a rural area in a country where TB is endemic, is highly likely to have contributed to acquiring a TB infection.

Once the organism is deposited in the lung alveoli, it results in the activation of the innate immune system via its interaction with macrophages and forms granulomas. The bacilli then disseminate to distant organs through the draining lymph nodes. In the CNS, the bacilli targets the microglial cells. Central nervous system tuberculomas start with the formation of tuberculous foci in the brain parenchyma, meninges, or the spinal cord.

Intracranial tuberculomas make up 5–30% of all intracranial lesions in developing countries [12]. The bacilli disrupt the blood-brain barrier (BBB). Vascular endothelial growth factor (VEGF) has been shown to increase BBB permeability and activate angiogenesis in the acute phase[13]. This activates the inflammatory cascade. K^{Trans} is a measure of BBB permeability that is obtained using dynamic contrast-enhanced MR perfusion. K^{Trans} is elevated in patients with intracranial tuberculoma [7]. The matrix metalloproteinases (MMPs) are responsible for the surrounding tissue destruction in intracranial tuberculoma [14].

Patients often present with subacute to chronic symptoms of elevated intracranial pressure, along with focal neurological deficits. The delay in early diagnosis and treatment is frequently due to the absence of a prior history of tuberculosis in more than half of the cases, the nonspecific nature of the initial clinical presentation, and the common radiological findings. A brain CT scan has relatively low specificity for diagnosing tuberculoma, with false positives reported in up to 80% of cases when used alone. [15] in an MRI tuberculomas appear as noncaseating, caseating with a solid center, and caseating with a liquefied center, based on the stage of maturation. Noncaseating tuberculomas typically appear hypointense on T1-weighted images (T1WI) with homogeneous nodular enhancement when contrast is applied and hyperintense on T2-weighted (T2WI) and FLAIR images. Caseating tuberculomas with a solid center show isointense or hypointense enhancement on both T1WI and T2WI, with an isointense or hyperintense rim on T2WI, and exhibit a ring-enhancing appearance with contrast. Caseating tuberculomas with liquefied centers are hypointense on T1WI, hyperintense with a hypointense rim on T2WI, and can present with a ring-enhancing lesion with contrast.[16,17]

The culture of mycobacteria from CSF is the gold standard, but the positive rate (5–58%) varies and remains considerably low, and the positivity in pediatric patients is even lower (10–20%). To increase positivity in smears or cultures, a minimum of 10% of CSF volume should be obtained for analysis [18,19].

The definitive diagnosis of the lesion is established through a brain biopsy. Open brain biopsy is a more invasive method compared to a stereotactic biopsy; however, it has a higher chance of obtaining diagnostic tissue [20]. Many surgeons consider stereotactic biopsy as a selected diagnostic method because it has lower chances of

obtaining a diagnostic sample rendering the need for an open biopsy [21, 22]. Microscopically, typical epithelioid and giant cell granuloma with central caseous necrosis are characteristic [23].

The management of intracranial tuberculoma and tuberculous otitis media includes symptomatic treatment, medical treatment with anti-tuberculosis medication, and possibly the surgical resection of the lesion. Glucocorticoids, such as dexamethasone, are clinically recommended to reduce the risk of inflammation, alleviate cerebral edema, and decrease intracranial pressure. The initial regimen of anti-tuberculosis medication includes isoniazid, rifampicin, pyrazinamide, and ethambutol (Streptomycin or a fluoroquinolone antibiotic can be used as an alternative) for 2–3 months. Isoniazid and rifampicin are then prescribed as consolidation treatment for 12 months. The regimen can be extended to 18 months if necessary. Surgical resection is considered in cases of a mass effect, intracranial hypertension, posterior fossa tuberculomas with hydrocephalus, visual disturbance, and progression of the tuberculoma while on anti-tuberculosis medication [24]

Among the five cases reported in a case series of 5 cases by Priti S Hajare et al., three presented with discharging sinuses, one with mastoid abscess, and one with tuberculous otitis media with pulmonary origin. All five cases of tubercular otitis media were confirmed with histopathological examination.[25]

In this patient type 1 tympanoplasty was performed and histopathological examination of the granulomatous tissue was obtained intraoperatively. This was followed by ATT as per the advice of the district TB officer.[26]

IV. Conclusion

In a country where TB is endemic, like India, it is important to have a high degree of suspicion in cases with remitting or recalcitrant otorrhoea. Treatment would be as per the latest guidelines.

Conflict of interest
The authors declare that they have no conflict of interest regarding the publication of this case report.

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Patient Consent
Obtained.