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REVIEW ARTICLE

PRIMARY TUBERCULOSIS OTITIS MEDIA: CASE PRESENTATION AND REVIEW OF LITERATURE

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ABSTRACT: INTRODUCTION: Tuberculous otitis media is a rare cause of chronic suppurative infection of the middle ear and mastoid. The incidence of tuberculosis in the middle ear is very low and accounts for only 0.04% of all cases of chronic Suppurative otitis media. Its diagnosis is often delayed because it can easily be confused with other acute or chronic middle ear conditions, still the treatment of tuberculous otitis media is medical treatment with anti -tuberculous drugs and mastoid exploration if the temporal bone is involved and also to clear the disease from the middle ear cavity to avoid further complications. **CASE PRESENTATION:** A 21 year male patient presented with ear discharge, deafness and headache, diagnosed as primary tuberculous otitis media, treated with mastoidectomy and anti-tuberculous treatment. **DISCUSSION:** Primary tuberculous otitis media is very rare condition in adults, it is commonly seen in children which is also secondary to pulmonary tuberculosis / extra pulmonary tuberculosis the treatment of choice is anti tuberculous treatment for 06 months, surgery indicated to clear the disease from middle ear, temporal bone and to avoid further complications. **CONCLUSION:** Even though primary tuberculosis in middle ear and mastoid is very rare, the diagnosis is possible only with histological findings. In our case there was no signs of pulmonary / extra pulmonary tuberculosis, treated with mastoidectomy followed by anti - tuberculous treatment.

KEYWORDS: Acid-fast bacilli, Langerhans giant cells, Mycobacterium, Otorrhoea, Tuberculosis, Ziehl-Neelsen Stain.

INTRODUCTION: What is Tuberculosis?

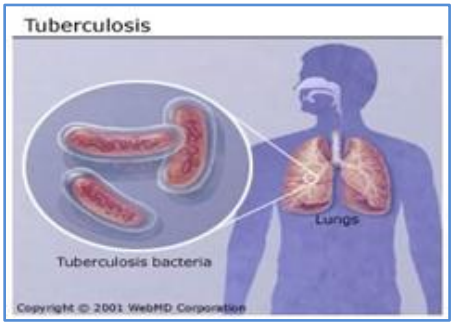

Figure 1

Tuberculosis is a bacterial infection the spread through the lymph nodes and bloodstream to any organ of the body. It is most often found in the lungs. Most people who exposed to TB never develop symptoms because the bacteria live in an inactive form in the body. But if the immune

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system weakens, such as in people with HIV or elderly adults, TB bacteria can become active. In their active state, TB bacteria cause damage to tissue in the organs they infect, active TB disease can be fatal if untreated, because the bacteria that cause tuberculosis are transmitted through the air, the disease can be contagious. It was once a widespread disease, virtually wiped out with help of antibiotics developed in the 1950s, but the disease has resurfaced in potent new forms – multidrug – resistant TB and extensively drug-resistant TB, today these new and dangerous forms of the disease-resistant to some of the commonly used medical treatments–have created a public health crisis in many large population worldwide.

CAUSES: The main cause of Tuberculosis is mycobacterium tuberculosis, a small aerobic non-motile bacillus. (Fig No.2)

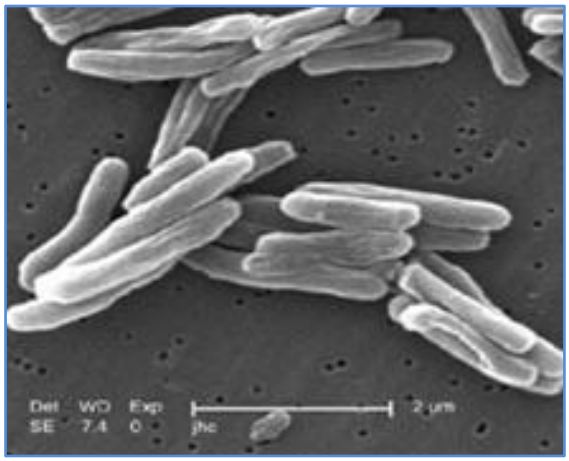

Figure 2: Tuberculosis Bacilli

The high lipid content of this pathogen accounts of many of its unique clinical characteristics. It divides every 16 to 20 hours, which is an extremely slow rate compared with other bacteria, which usually divides in less than an hour. Mycobacteria have an outer membrane lipid bilayer. If a gram stain is performed, MTB either stains very weakly "Gram-positive" or does not retain dye as a result of the high lipid and mycolic acid content of its cell wall. MTB can withstand weak disinfectants and survive in a dry state for weeks. The M. Tuberculosis complex (MTUBERCULOSIS.) includes four other TB-causing mycobacteria: M. bovis. M. africanum, M. Canetti, and M. microti, M. africanum is not widespread, but it is a significant cause of tuberculosis in parts of Africa. M. bovis was once a common cause of tuberculosis, but the introduction of pasteurized milk has largely eliminated this as a public health problem in developed countries. M. Canetti is rare and seems to be limited to the Horn of Africa, although a few cases have been seen in African emigrants. M. Microti is also rare and is mostly seen in immune deficient people, although the prevalence of this pathogen has possibly been significantly underestimated.

Tuberculous otitis media is a rare form of chronic otitis media and extra pulmonary tuberculosis, incidence of tuberculosis is 0.04–1% and 4% of Head and Neck tuberculosis[1] In 1960 Tuberculosis bacilli were isolated from the ear, much later than it was first isolated by Robert Koch on 24th March1882.[2]

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Figure 3: Robert Koch

Tuberculosis remains the leading cause of death secondary to infectious diseases worldwide in persons older than 5 years.[3] Tuberculosis of middle ear is a comparatively rare entity usually seen in patients with or secondary to pulmonary tuberculosis. It is one of the major infectious disease with predominant involvement of lung and lymph nodes but tuberculosis of middle ear is uncommon.[4] It is most common infectious diseases of developing countries including India and Nepal.[5] It is difficult to assess its true incidence as the large reported series have been selected from hospitalized subgroups with established tuberculosis,[6,7,8] early diagnosis and prompt treatment may prevent ear damage and central nervous system complications. The involvement of temporal bone by tuberculosis was first described by Jean lonis petit in 18th century.[9] The clinical signs of the disease were first outlined by wilde in 1853.[10]

Tuberculous otitis media is the result of haematogenous spread of the infection in patients with other forms of tuberculosis rarely, it is the result of infection imported through perforated tympanic membrane. In children, aspiration of infected milk through the Eustachian tube during drinking or nursing was a very common way of infection in the first half the 20th century.[11] The disease became considerably rare in children by making BCG vaccination obligatory and with pasteurization of milk. Today, tuberculous otitis media is uncommon and is rarely thought of. The classic description of tuberculous otitis media indicates multiple perforations of tympanic membrane, painful suppurative otorrhoea, and preauricular adenopathy, frequent complications like paralysis of the facial nerve, sensorineural hearing loss (SNHL) and association with pulmonary tuberculosis. A recent description of the disease includes large tympanic membrane perforation, conductive hearing loss that suddenly becomes sensorineural, with pale granulation tissue and dense secretion similar to infected cholesteatoma. Cervical lymphadenopathy and facial palsy are rare.[12]

Making the diagnosis is difficult: the process lasts from 14 to 70 days, because the culture of the tissue or secretion is usually negative. According to data, positive acidoalcohol fast bacilli (AFB) smears are uncommon (2-4%), while histopathological examination rarely indicates tuberculous granuloma, but more frequently necrotizing granuloma[13,14,15] chain reaction (PCR) testing represents the only hope, although there are opinions that this method is not reliable. The CT of the temporal bone does not necessarily indicate bone destruction.

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CASE PRESENTATION: A 21 age old male patient reported to OPD with complaints of:

1. Foul-Smelling, scanty purulent discharge from (L) ear since 2 months.
2. History of loss of hearing – 02 months, (L) Ear.
3. (L) Post aural swelling – 01 week, history of headache present.

Patient was admitted and all the necessary investigations were done along with CT scan temporal bone,

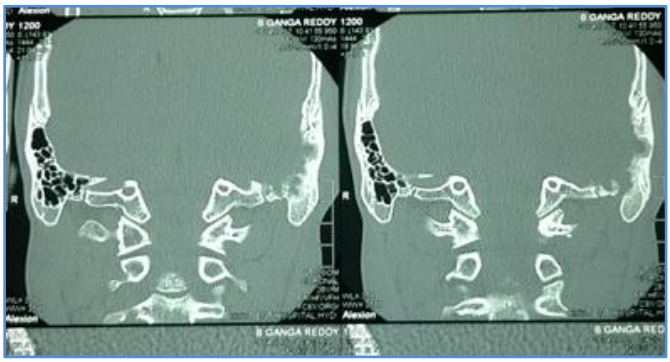

Figure 4: CT scan Temporal bone

All the investigations were within normal limits except Hbs Ag (+) and CT Temporal Bone shows (L) Mastoiditis, subsequently examination under microscope was done, pale granulations present in the external auditory canal, middle ear cannot be visualized and Incision and drainage of post aural swelling was done at the same sitting, the granulations sent for HPE, shows - granulation tissue with many granulomatous epithelial cells, giant cells adjacent skeletal muscle tissue epidermis and dermis–Tuberculous granulation tissue.

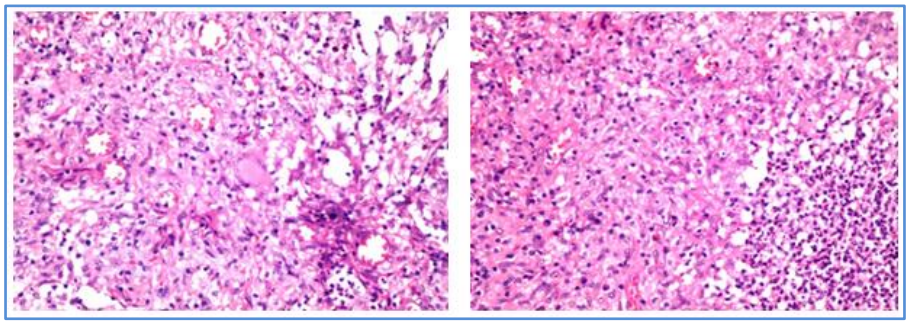

Figure 5 & 6: HPE Report

Patient subjected for further investigations to rule out pulmonary/ extra pulmonary tuberculosis.

- Sputum for AFB - Negative.
- Chest X- ray - Normal.
- CBC, ESR - Within normal limits.
- Montoux Test - Negative.

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Patient was diagnosed as primary tuberculous otitis media and planned for mastoid exploration under general anesthesia, because of extensive granulations were cleared from the Middle ear and mastoid, ossicles were necrosed, total disease was cleared, TROP is placed over the foot plate of stapes, as the patient came from a very remote village, thinking that patient may not be turn up for second surgery, reconstruction surgery was planned.[16]

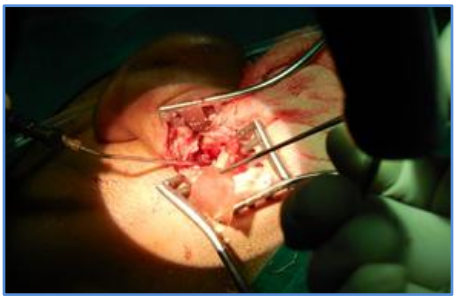

Figure 7: Intra operative Photo

Temporalis facia graft placed medial to the tympanic membrane, wound closed in layers, mastoid bandage applied – Post operative recovery was uneventful, sutures removed on 7th day, wound was healthy, patient kept on ATT (Anti -Tuberculous Treatment) for 06months. Patient doing well by the time of publishing this paper, surgery has a minimal role but may be useful to provide polyp or granulation tissue for histology and for treating complications, surgery in aural tuberculosis aims to correct sequelae following medical treatment to cure the disease[17,18,19] the time gap between biopsy and mastoidectomy was very less only 10days, hence ATT was started postoperatively.

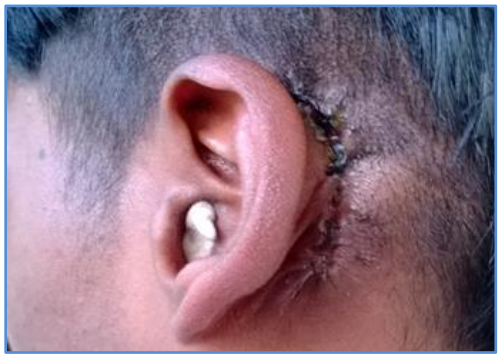

Figure 8: Post-Operative Photo

DISCUSSION: Tuberculous otitis media is an uncommon condition in adult, or at least is seldom diagnosed, modern authors agree that it occurs in about 10% of phthisical patients. The disease is classified as:

- Primary in the Ear and Mastoid process and
- Secondary to tuberculosis elsewhere in the body.

Primary tuberculosis is seen almost always in children, and present quite different features to the secondary tuberculosis of the middle ear met with in adults, the adults however are not all

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together immune to the primary lesion,[20,21] the pathway of infection in adult cases is usually by the Eustachian tube (J.S. Frazar).[22] Ormerod has described a tuberculous infiltration of the Eustachian cushion as occurring in some cases[23] a minority of cases appear to be due to a blood-borne infection.

In spite of therapy progress and prevention of tuberculosis, today it is still the most common infection worldwide. It is estimated that currently there are about eight million people in the world with active tuberculosis.[24] Luckly, tuberculosis of the middle ear and of the temporal bone are rare with the incidence of 0.04 to 0.9% of all suffering from TUBERCULOSIS., or 0.04% of all suffering from chronical otitis, or 4% of the patients with TUBERCULOSIS. of the head and neck.[25] Tuberculous otitis media remains a diagnostic challenge for many clinicians, because of its unspecific clinical features and inability to confirm the infection by microbiological and histo pathological examinations. Today clinical features are altered and cotemporary diagnostic criteria are defined by literature data, the existence and course of chronic otitis media are more important clinical characteristics of TOM than positive culture of mycobacterium.[26] The clinical features of the disease are either a total defect of the tympanic membrane or a completely intact tympanic membrane. If the membrane is intact, it is pale, tense and immobile, with a strong vascular pattern, multiple perforations are very rare and atypical. Ear suppuration does not react to antibiotic therapy applied either locally or parenterally.

The mucosa of the mastoid cavity is changed, with granulation tissue which is pale an similar to oedematous mucosa, temporal bone destruction can exist in a sense of sequenstration or fistulas, especially cochlear, and in radiography imaging, it is not different from other nonspecific osteomyelitic processes. Cases with a sudden onset of the disease, pain and hearing loss, pre-auricular swelling and granulation tissue in the external auditory canal prominating through tympanic perforation have been also described. CT imaging in these cases show the clouding of the mastoid without necrosis of the bone, while histological analysis shows granuloma with necrosis. Imbalance between clinical findings and functional disorders which can be drastic, like facial palsy (16% in adults, 35% in children) or a sudden or progressive SNHL is especially important.[27]

Histopathological features of tuberculous otitis media are not a typically formed granuloma, only langerhans gaint cells can be disclosed, which can suggest other diseases like syphilis, other granulomatous inflammations, mycoses etc. A very common finding is necrotic tissue in frozen sections. Histo-pathological findings indicated necrotic osteomyelitis in patients with associated miliary tuberculosis., and in other patients nonspecific lymphoplasmocytic infiltration with presence of macrophages. Ziehl-Neelsen staining of the granuloma could not reveal mycobacteria.[28,29]

Microbiological confirmation is often difficult or impossible to obtain, even with PSR method with Ziehl-Neelsen staining of the smears and tissue, rarely find red-coloured bacilli, and PCR testing is not accurate enough. In cases of TOM it is almost impossible to apply the diagnostic principles for extra pulmonary tuberculosis.. In these patients it is hard to prove the existence of tuberculosis. infection with microbiological, histological or cytological analysis of tissue and fluids. Often the limiting factor is a small amount of tissue or fluid. The pathogenesis of tuberculous otitis media involves three major mechanisms. The first mechanism is aspiration of mucus through the Eustachian tube, the second is blood-borne dissemination from other tuberculous foci and the third is direct implantation through the external auditory canal and a tympanic membrane perforation[30] TOM is used to be more common in children than in adults possibly because the Eustachian tube anatomy of children permits reflux of material into the middle ear cavity.[31] Generally tuberculosis of the middle ear is unilateral.[32] This is surprising as one would expect it to be bilateral since a possible route for

middle ear infection is through the Eustachian tube. Associated facial nerve paralysis is seen in approximately 16% of adult cases and 35% of pediatric cases.[33] Most patients with TOM have unnecessary surgery only to have the diagnosis made from histology or tissue culture, as about 50% of patients with TOM have radiographic pulmonary tuberculosis[34] appropriate evaluation for TOM includes a chest film.

LEARNING POINTS:

- Otorrhoea in a patient with known or suspected active pulmonary tuberculosis should be assumed to be tuberculous otitis media until proven otherwise.
- The clinical features in every case of chronic otitis media should be re-evaluated keeping the possibility of tuberculosis in mind.
- Early diagnosis and prompt treatment with anti-tuberculous drugs has made the condition less devastating.

TREATMENT OF TUBERCULOSIS OTITIS MEDIA: Anti-tuberculous therapy is the treatment of choice for TOM. The first successful treatment of TOM with antibiotics was reported in 1948 by Grief and Gould who administered streptomycin. Current standard chemotherapy uses a combination of drugs. TOM should be managed with anti-tubercular therapy (Category1). Various chemotherapy modalities are available. The first includes a four – drug regimen in the first 2months (isoniazid, rifampicin, pyrizinamide and ethambutol) followed by a two drug regimen in the following 04 months (isoniazid and rifampicin). Another modality involves the use of the same four drugs (isoniazid, rifampicin, pyrizinamide and ethambutol) for a period of 06 months, this has proved effective in many centres. Most patients will require at least 6 months of anti-tuberculous therapy. A notable example is a combination of pyrazinamide 500mg isoniazid 300mg, rifampicin 600mg, ethambutol 400mg and vitamin B6 for a planned duration of 6-9months.

Surgery is carried out for functional reconstruction, to remove bony sequestrate and to treat complications. Myerson and Gilbert advised radical mastoidectomy if any of the following complications develop: facial paralysis, subperiosteal abscess, labyrinthitis, mastoid tenderness or headache. When surgery is combined with adequate chemotherapy, there is a fair chance of healing with a dry ear and a good prognosis.

CONCLUSION: The basic principle in the diagnosis of tuberculous otitis media is suspected tuberculosis process in patients with active infection, a positive illness history, patients living in endemic environment or those exposed to tuberculosis. or in contact with other persons suffering from tuberculosis, we should suspect tuberculosis process if otorrhea is of shorter duration (A few months) and if there is a rapid development of otogenic complications. We are here presenting a case of primary tuberculous otitis media in a adult which is a rare form of presentation (usually tuberculous otitis media is seen in children) without secondary to pulmonary tuberculosis or extra pulmonary tuberculosis, treated with mastoid exploration followed by anti tuberculous treatment.

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