

Tuberculous Otomastoiditis, an Uncommon Presentation of Extrapulmonary Tuberculosis in a Immunocompetant Child

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Abstract

Case Report

Tuberculosis is a major public health problem in our country with predominant involvement of lung and lymph nodes which. Tuberculous mastoiditis is quite rare and is often diagnosed late. Early diagnosis and effective treatment may prevent ear damage as well as central nervous system complication. In this report, we describe the case of a 15-month-old male with a history of fever and yellow discharge. CT scan of temporal bones showed fluid accumulation in mastoid cells and in the middle ear with destruction of the left mastoid cortex and partial ossicular erosion. It's associated with a collection of soft parts opposite with peripheral enhancement after contrast application with left sigmoid sinus thrombosis. Necrotizing granulomatous inflammation was present in the mastoid specimens in all cases. Confirmatory diagnosis was made via GeneXpert polymerase chain reaction (PCR). The clinical outcome was favorable after anti-tuberculosis treatment.

Keywords: Tuberculous mastoiditis- Immunocompetant child- CT scan.

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INTRODUCTION

In Morocco, tuberculosis is a major public health problem. It is a contagious disease transmission is usually due to droplet infection. According to WHO, about 10 million people are infected in the world mostly in developing countries [1]. It is caused by "mycobacterium tuberculosis" which can be very aggressive when there is local and/or general immune deficiency. The pulmonary system is mainly affected, followed by lymph nodes and visceral organs [2].

Extrapulmonary TB (EPTB) constitutes about 15-20% of new tuberculosis (TB) cases in immunocompetent patients and accounts for more than 50% of new TB cases in HIV-positive individuals [3]. Tuberculous otitis media (TOM) is a rare disease and accounts for 4% of head and neck TB and 0.05-0.09% of chronic infections of the middle ear [4,5]. Not suspecting TOM in children can delay diagnosis and have serious consequences, such as hearing loss and delayed speech and language acquisition, and ultimately undermining neurodevelopment, especially if the disease complicates to TB mastoiditis [6].

CASE REPORT

We report the case of a 15-month-old male with a history of fever and yellow left ear discharge with foul smelling, painless, and non-blood stained. There was no history of facial nerve paralysis, night sweats, upper respiratory symptoms, dizziness, weight loss, prior ear infections, or history of tuberculosis.

At that time, he was afebrile and had no lymphadenopathy. An otoscopic examination of the left ear revealed a yellow discharge. His other systemic examination findings were normal. Serology for human immunodeficiency virus was negative.

CT scan of temporal bone showed fluid accumulation in mastoid cells and in the middle ear with destruction of the left mastoid cortex extended to tegmen antri, with ossicular and scutum erosion, and left sigmoid sinus plate with sinus thrombosis. It's associated with a collection of soft parts opposite with peripheral enhancement after contrast application. Necrotizing granulomatous inflammation was present in the mastoid specimen. A confirmatory diagnosis was made via GeneXpert polymerase chain reaction (PCR).

The clinical outcome was favorable after anti-tuberculosis treatment.



Figure 1: CT scan of temporal bone in axial (A) and coronal (B) cross sections showed fluid accumulation in mastoid cells and in the middle ear with destruction of the left mastoid cortex (red arrow) extended to tegmen antri (blue arrow)

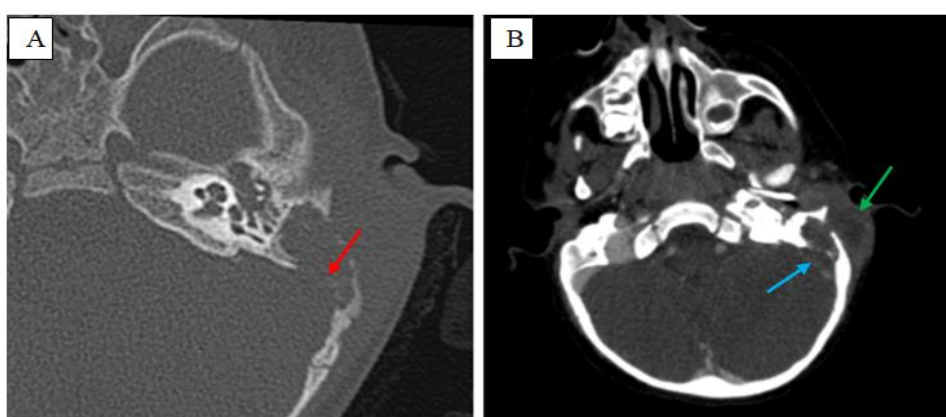


Figure 2: CT scan of temporal bone in axial section (A) and brain CT scan with contrast enhancement showed destruction of left sigmoid sinus plate (red arrow) with sinus thrombosis (blue arrow). It's associated with a collection of soft parts with peripheral enhancement after contrast application (green arrow)

DISCUSSION

Worldwide, tuberculosis is a widespread disease, with 10 million 8.7 million new cases occurring annually [1]. Its etiologic agent, *Mycobacterium tuberculosis*, essentially causes pneumonia. However, this organism affects the middle ear in rare cases, accounting for 0.04–0.9% of all chronic middle ear otitis cases [5].

Tuberculous mastoiditis was first described by Jean Louis Petit in the 18th century. In 1853 Wilde presented the classical picture of tuberculosis otitis media presented as painless ear discharge of insidious onset with multiple perforations in the tympanic membrane, and pale granulations in the middle ear cleft. Politzer discussed the destructive nature of this disease; in 1882 Koch discovered tubercle bacilli [1].

Three theories are quoted in the literature to describe the pathogenesis of TOM:

- Hematogenous transmission;
- Direct extension via the Eustachian tube,
- Direct implantation through a perforation of the tympanic membrane.6,7

Tuberculous otitis media remains a diagnostic challenge because of its non-specific clinical features and the difficulty in confirming the diagnosis by microbiological tests of ear discharge [7]. Classical features of TOM like painless otorrhoea and multiple tympanic membrane perforations are not constant features. Facial nerve palsy is rare [8]. Facial nerve palsy associated with tuberculous mastoiditis is seen in approximately 16% of adult cases and 35% of pediatric cases [9]. Complications of tuberculous otitis media can include mastoiditis, conductive hearing loss, periauricular fistulae, and central nervous system involvement [10].

The diagnosis of tuberculous otitis media requires a high index of suspicion. According to most authors, histological findings, such as granulomas, Langhans giant cells, and caseation necrosis, associated with biomolecular positivity (PCR), represent the cardinal diagnostic elements of TOM [11]. Stains and cultures of ear drainage are often negative. The diagnosis is often made by biopsy of the granulation tissue in the middle ear [10].

CT scan is reportedly the best imaging technique for TOM [12]. Soft tissue attenuation in the entire middle ear cavity, preservation of the mastoid air cells without sclerotic change, and soft tissue extension to, or mucosal thickening of the external auditory canal were more frequent in patients with TOM compared to those having pyogenic chronic otitis with or without cholesteatoma [13]. Erosion of the ossicles and scutum was more frequent in TOM [13]. Temporal bone CT scans of 23 South Korean patients with TOM showed bone destruction that involved the cortex of either the external auditory canal or the outer cortex of the mastoid bone in 26.1% of cases [14]. More marked bone destruction involving the base of the skull as well as the mastoid bone was found in only one patient (4.3%).

After proper diagnosis, the initial management of tuberculous otitis media should be medical. Antituberculosis drugs dramatically improve the prognosis in most patients [15]. Surgical intervention should be added to drug therapy in cases of complications [1]. When surgery is required, the techniques and guidelines are the same as those for the surgical treatment of chronic bacterial otitis media, with or without cholesteatoma [16].

CONCLUSION

Tuberculous otitis media and tuberculous mastoiditis are very rare. Their diagnosis requires a high index of suspicion. Untreated tuberculous can result in the permanent, severe sequelae, such as facial paralysis, hearing impairment, and intracranial dissemination of infection. Early suspicion and timely diagnosis are of great importance in the resolution of disease and prevention of such serious complications. Including this disease in the differential diagnosis and considering it will lead to prompt treatment and prevention of serious sequelae.

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