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Tuberculous Otitis Media-A Case Report

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Abstract
Tuberculous otitis media (TOM) is a rare cause of chronic suppurative infection of the middle ear, which can easily be confused with other chronic middle ear conditions. The classical features of the disease have been changing in recent years. Unusual features such as otalgia, bilateral ear involvement, single perforation on tympanic membrane and absence of facial nerve palsy also should alert the physician for early diagnosis. Knowledge of this disease is important because early diagnosis and therapy may prevent or limit complications and needless surgery. To increase awareness of this condition, we describe one case of TOM and we review the literature.

Key words: Tuberculous, otitis media, otorrhea

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INTRODUCTION

The true incidence of tuberculous otitis media (TOM) is unknown but, it is thought to be underdiagnosed throughout the world, especially in the underdeveloped countries [1]. In developed countries, TOM is a rare cause of otorrhea, accounting for between 0.05% and 0.9% of chronic infections of the middle ear [2, 3]. Characteristic clinical features of TOM have been described in the literature, including a profuse purulent ear discharge, profound hearing loss, multiple perforations of the eardrum, and facial paralysis [1, 2, 4]. When it does occur, it is associated with substantial morbidity, and a delay in initiating therapy can lead to serious complications. So we want to report this case to alert physicians to some unusual clinical features of TOM that we have encountered and to discuss the diagnostic work-up and management based on literature review.

CASE REPORT

A 39-year-old man was referred to our hospital with a two-month history of progressive hearing loss, otalgia, fever, weakness, and cough. Physical examination of the both ears showed purulent yellow discharges at middle ear spaces and external canal lumens, inflamed and edematous external canal wall skins and central perforation of tympanic membranes. His other systemic examination findings were normal, except for anemic conjunctivae. Initial laboratory data revealed a peripheral white blood cell count of 11600/mm^3, hemoglobin of 10.4g/dl, and hematocrit of 30.2%. Chest radiography demonstrated bilateral extensive consolidations and cavitations at upper and middle zones (Figure 1). Examination revealed a 45 dB conductive hearing loss at both ears on pure tone audiometry. Serology for human immunodeficiency virus was negative. Because of the association of a lung infiltration with cavitation and perforated tympanic membranes with purulent drainage, active pulmonary and middle ear tuberculosis was suspected. Examination of the both auricular secretions and sputum using the Ziehl-Neelson staining method revealed acid-fast bacilli (AFB). Three sputum smears of the patient were 2+ for AFB and, both ear drainage smears were 1+ for AFB. The non-specific culture of ear drainage yielded methicillin sensitive Staphylococcus aureus. Cultures of all sputa and ear drainage grew Mycobacterium tuberculosis in Löwenstein-Jensen culture. Drug susceptibility testing showed that Mycobacterium tuberculosis was sensitive to isoniazid, rifampicin, ethambutol and streptomycine. The treatment was started with oral isoniazid, rifampicin, pyrazinamide and ethambutol immediately after the positive smear results were obtained. For secondary infection cefazolin was given. After the initiation of treatment sputum smears became negative, and ear drainage resolved by the end of three months of therapy. A total of 6 months of antituberculous therapy was planned. His family and other potential respiratory contacts were routinely evaluated and preventive therapy was given to his daughter.

DISCUSSION

The incidence of TOM is very low; tuberculosis accounts for only 0.04% of all cases of chronic suppurative otitis media in the UK [5,6]. The prevalence of active or inactive pulmonary tuberculosis in patients with TOM ranges from 14% to 93% [1,7-9]. On the other hand about 2% of patients with active pulmonary tuberculosis may have TOM [3].

The classic clinical features of TOM were described by Wallmer, in 1953, as painless otorrhea, multiple tympanic membrane perforations, pale granulation tissue, ipsilateral facial nerve paralysis, early severe hearing loss, and bone necrosis [4]. However, these classical features are rarely ob-

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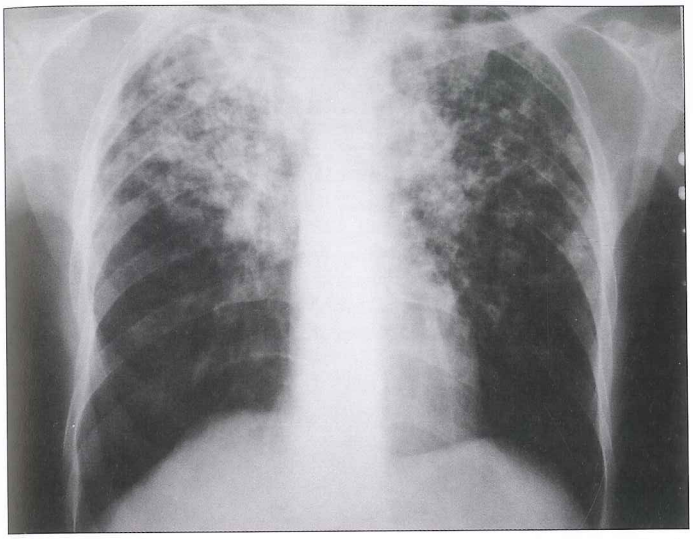

Figure 1. Chest film is showing diffuse infiltration and cavitation at upper and middle zones of the both lung

served today. Recently a review of all reports of TOM in the English literature by Skolnik et al refuted these findings. Their research showed that facial palsy is present in only 16% of cases and multiple tympanic perforations are equally rare [3]. Facial nerve palsy is more likely in children than adults [7,8,10]. Out of the 323 cases of tuberculous otomastoiditis reviewed by Mjoen et al., only 4.8% were reported to have multiple perforations of the tympanic membrane [11]. The otorrhea described as being painless, can be painful [1, 9]. Otalgia may due to granulation tissue presenting in the middle ear and passible bacterial superinfection. In series of Nishiike et al., none of the patients had multiple perforations, facial nerve palsy and bone erosion [12]. Generally tuberculosis of the middle ear is unilateral [2, 10, 13], bilateral disease seen in our patient is uncommon.

The pathogenesis of TOM involves three major mechanisms. The first mechanism is aspiration of mucus through the eustachian tube. The second is the blood-borne dissemination from other tuberculous foci. The third is that, direct implantation through the external auditory canal and a tympanic membrane perforation [9]. TOM was more common in children than adults possibly because of eustachian tube anatomy of children permits reflux of material into the middle ear cavity [3, 9].

AFB is rarely cultured since in extrapulmonary tuberculosis the mycobacterial counts are low [13, 14]. Smears of ear drainage with TOM are positive for AFB in 0%-to 20% of the cases and cultures are positive for Mycobacterium tuberculosis in 5% to 44% of the cases [1, 2, 7, 9]. Bacteriological examination of the ear drainage is not very reliable as the presence of other organisms such as Staphylococcus, Pseudomonas, Klebsiella, Proteus, and Streptococus can interfere with the growth of Mycobacterium tuberculosis [9]. The presence of secondary infection may cause the delay of the diagnosis. So if atypical clinical features are noted in a case of chronic otitis media, repeated cultures of the ear discharge should be obtained.

Most patients with TOM have needless surgery only to have the diagnosis made from histology or tissue culture [1, 7-9]. A high index of suspicion for TOM is required to make the correct preoperative diagnosis [9]. About 50% of patients with TOM have radiographic pulmonary tuberculosis [1, 15]. For this reason appropriate evaluation for TOM includes a chest film, purified protein derivative skin testing, and smears or cultures of aural discharges secretions, sputum or other appropriate body secretions for mycobacteria. Also gene amplification technigues such as polymerase chain reaction can be useful [16]. If the cause of suppurative infection of the middle ear is still undiagnosed than operative biopsy may be required for diagnosis [13].

The differential diagnosis of TOM includes bacterial otitis media, histoplasmosis, blastomycosis, nocardiosis, atypical mycobacterial infections, syphilis, midline granuloma, necrotizing external otitis, lymphoma, sarcoidosis, Wegener's granulomatosis, and histiocytosis X [17].

Otitis media due to nontuberculous mycobacterial infection is increasingly seen in patients with pre-existing ear disease and after surgical and aural interventions. Nontuberculous mycobacterial infections in surgical patients have been reported in wide variety of settings. The use of colonized aqueous solutions and inadequate sterilization or disinfection of surgical equipment are often factors in these infections [18]. Contaminated otolaryngostomy equipment led to Mycobacterium chelonae otitis media in 17 patients [19]. Mycobacterium fortuitum mastoiditis has also been reported after myringotomy and tympanostomy tube placement [20].

The effective drug therapy of TOM is equivalent to the regimen for pulmonary tuberculosis. Once appropriate medical therapy is instituted, aural discharge usually resolves within 1 to 5 months. Patients will require at least 6 months of antituberculous therapy for cure [1, 8,10]. Surgery is carried out for functional reconstruction, to remove bony sequestrate and to treat complications [2, 9,14].

Complications associated with TOM include postauricular fistula, facial nerve paralysis, labyrinthitis, tuberculous osteomyelitis of the petrous pyramid, acute mastoiditis, hearing loss, and spread of the infection to the central nervous system [3-5,9,21].

CONCLUSION

The clinical symptoms and signs should be reviewed in every case of chronic otitis media keeping the possibility of tuberculosis in mind. Otorrhea in a patient with known or suspected active pulmonary tuberculosis should be assumed to be TOM until proven otherwise. Due to clinical features of this disease have been changing over the years unusual features such as otalgia, bilateral ear involvement, single perforation on tympanic membrane and absence of facial nerve palsy also should serve as an alert.

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