
Tuberculous Otitis Media: A Review of Literature

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Citation

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Abstract

Tuberculous otitis media is one of the most common infectious diseases of developing countries including Nepal. It rarely affects the middle ear. The objective is to review the literature on tuberculous otitis media and know the facts, incidence, etiology, clinical presentation, investigations and treatment of tuberculous otitis media. It is characterized by painless otorrhoea which fails to respond to the usual antimicrobial treatment, in a patient with evidence of tubercle infection elsewhere followed by multiple tympanic membrane perforations, abundant granulation tissue, and bone necrosis, preauricular lymphadenopathy. Deafness is out of proportion with the apparent degree of development of disease seen in the otoscopy. Combination chemotherapy is prescribed. Surgery may be required in some cases to remove sequestra and improve drainage. A high level of clinical suspicion is needed for early diagnosis and antitubercular therapy should be started as soon as possible to prevent the possible complication.

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INTRODUCTION

Tuberculosis remains the leading cause of death secondary to infectious diseases worldwide in persons older than 5 years [1]. Tuberculosis of middle ear is a comparatively rare entity usually seen in association with or secondary to pulmonary tuberculosis. Tuberculosis is one the major infectious disease with predominant involvement of lung and lymph nodes but tuberculosis of the middle ear is uncommon [2]. It is one of the most common infectious diseases of developing countries including Nepal [3]. TB can also be transmitted congenitally and is associated with a high incidence of ear involvement. However, congenital TB is extremely rare and hardly ever presents with isolated ear involvement.

It is difficult to assess its true incidence as the large reported series have been selected from hospitalized subgroups with established tuberculosis [4,5,6]. Early diagnosis and prompt treatment may prevent ear damage and the central nervous system complication. The objective of this study is to review the literature on tuberculous otitis media and know the facts, incidence, etiology, clinical presentation, investigations and treatment of tuberculous otitis media.

METHODS OF SELECTION

The literature of tuberculous otitis media was searched on MEDLINE, science direct and HINARI. Articles from 1907 AD-2006 AD were included in this review. It took around 3 months to get the full text of all these articles. The articles where the authors could not get the full text article were excluded from the study. Articles were searched in MEDLINE, science direct and HINARI using following keywords: etiology, clinical presentation, investigations and treatment.

HISTORY

The involvement of the temporal bone by tuberculosis was first described by Jean Louis Petit in 18th century [7]. The clinical signs of the disease were first outlined by Wilde in 1853 [8]. In 1882, Koch demonstrated Tuberculous bacillus and Esche isolated bacillus in the secretion of middle ear in 1883 [9,10].

INCIDENCE

It is difficult to assess its true incidence as the large reported series have been selected from hospitalized sub-groups with established tuberculosis [4,5,6]. Primary tuberculosis of the ear has rarely been reported, and the disease is usually secondary to infection in lungs, larynx, pharynx and nose [4,11]. In the west, the annual incidence of tuberculous otitis media has decreased during the past 60 years from 5.5 cases per 100,000 population before 1953 to 2.3 cases after 1953

[9,13,14]. This decrease has been attributed to the declining incidence of tuberculosis itself. However, in areas where tuberculosis is endemic, data have shown that there has been a steady increase in its incidence [15]. In preantibiotic era, 2-8% of all the cases of chronic suppurative otitis media were tuberculosis in nature and infants less than 1 year of age comprised 50% of these [16]. There are only very few cases of tuberculous otitis media reported in the literature. Mills study mentioned that the incidence of tuberculous otitis media has fallen dramatically since the beginning of this century [17]. At that time 3-5% of cases of otitis media were due to tubercle bacillus, whereas today the condition is rare [17]. Turner and Eraser study reported in 1915 that 2.8% of all cases of suppurative otitis media were due to tuberculosis.

Kirsch et al study revealed 9.5% of children with tuberculous otitis media were less than 5 years of age [16]. The incidence of tuberculosis of middle ear is very low, tuberculosis accounts for only 0.04% of all cases of chronic suppurative otitis media [12]. When it does occur, it is associated with substantial morbidity, and a delay in initiating therapy can lead to serious complications. Till the preparation of this manuscript, to the best of author's knowledge, there are no cases of tuberculous otitis media reported in the literature from Nepal. There were two cases of tuberculous otitis media in T.U. Teaching Hospital, Kathmandu, Nepal which is histologically proved, but they have not been reported till now. In view of the extremely low incidence (<1%) of ear disease, it often precludes the diagnosis, especially in the absence of concomitant tuberculous focus elsewhere [19].

ETIOPATHOGENESIS

Tuberculous otitis media (TOM) is caused by *Mycobacterium tuberculosis*, of which *bovis* and *hominis* are generally affecting the ears. Sometimes rare species of mycobacterial infections can cause atypical in special situations especially in immunodeficiencies. *Mycobacterium bovis* is less frequently seen than *Mycobacterium hominis*. TOM is usually due to ingestion of infected cow's milk.

The route of spread of tuberculosis to middle ear has been argued for many years; the most logical route of entry of organisms being via pharyngo- tympanic tube [20]. ADAMS in a study of tuberculosis patients undergoing thoracoplasty showed abnormal pharyngo- tympanic tube patency in all patients who developed otitis media [5]. Tuberculosis

involving tympanic membrane is usually secondary to pulmonary tuberculosis, spreading through the Eustachian tube, most often by the forceful expulsion of haemoptysis and infected blood into the tympanum. The condition usually begins as an apparent serous otitis media. Infection can also reach the middle ear via external auditory canal or by haematogenous spread. Proctor and windsay study found the strong evidence of tubercle bacilli reached the ear by haematogenous route [21]. The latter results in the direct involvement of the mastoid bone producing necrosis and it progress to involve middle ear.

CONGENITAL FORM

Rarely there can be a congenital tuberculous otitis media. The fetus or the newly born are susceptible to various forms of contamination; directly through the placental circulation; by aspiration of infected amniotic fluid or in the act of birth, by contact with infected genital mucosa. It can also occur with congenital form of transmission of infection from mother to fetus.

CLINICAL PRESENTATION

The clinical signs and symptoms of tuberculous otitis media were first documented in 1853 [22]. Since then, many so called characteristic clinical feature have been described in the literature [23]. Generally tuberculosis of middle ear is unilateral. Tuberculosis of middle ear is characterized by painless otorrhoea which fails to respond to the usual antimicrobial treatment, in a patient with evidence of tubercle infection elsewhere followed by multiple tympanic membrane perforations, abundant granulation tissue, and bone necrosis, preauricular lymphadenopathy [23,24]. There may be multiple perforations in the early stages, but they coalesce into a total tympanic membrane perforation accompanied by a pale granulation tissue [4,14,22].

MYERSON'S experience demonstrated that a discharge from the middle ear appearing without pain in a tuberculous individual should be considered Tuberculous [25]. In early stages of tuberculous otitis media, the drum looks dull and some dilated vessels can be observed [26]. The tympanic membrane then becomes thickened and landmarks are obliterated [26]. The exudate in the middle ear may be thick and is sometimes confused with the infected keratin debris of a cholesteatoma. Periauricular fistulas, lymphadenopathy, and facial palsy are infrequent findings. Late complications include facial paralysis, labyrinthitis, postauricular fistulae, subperiosteal abscess, petrous apicitis, and intracranial extension of infection. Facial nerve palsy has been reported in cases of tuberculosis otitis media even if the anti

tuberculosis therapy has been started. Associated facial nerve paralysis is seen in approximately 16% of adult cases and 35% of pediatric cases [15,27]. It should also be considered in patients when chronic otorrhoea occurs in recent immigrants from areas with high rate of infection [28]. Tuberculous otitis media is more likely to cause infection of the labyrinth than the usual purulent forms of otitis [29]. However, due to gradual spread of the disease, symptoms caused by involvement of the labyrinth are uncommon, even though the function is destroyed [26].

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of tuberculous otitis media includes fungal infections, Wegener's granulomatosis, midline granuloma, sarcoidosis, syphilis, necrotizing otitis externa, atypical mycobacterial infections, lymphoma, histiocytosis X and cholesteatoma [30]. These diagnoses can be ruled out clinically by the presence of pain and the type and consistency of the discharge. In diagnosing tuberculous otitis media, it is important to consider it as a differential diagnosis of chronic suppurative otitis media. The diagnosis of tuberculous otitis media is often missed in the early stages or is made only after surgical treatment for otitis media [12,13,31,32].

INVESTIGATION

PURE TONE AUDIOGRAM

The main audiological feature of TOM is the deafness out of proportion with the apparent degree of development of disease seen in the otoscopy. Generally it is moderate to severe hearing loss. It can be conductive, sensorineural or mixed hearing loss. However, McAdam and Rubio reported a case of slow development of hearing loss, suggesting therefore that the hearing can be variable [33].

RADIOLOGY

Radiological studies such as simple x-ray mastoid or computerized tomography (CT) scan revealed no specific characteristics, but together with clinical and other complementary tests, can strengthen the suspected diagnosis. It also helps to find out the degree of involvement of structures and enable for better planning when surgery is needed. Several authors argue that the detection of an x-ray of the mastoid shows well pneumatization and sometimes filled by soft tissue, in patients with clinical of chronic otitis media, suggests the possibility of etiology of tuberculosis [7,10,34]. It is essential to remember that a normal chest x-ray does not rule out the possibility of tubercular infection of

ear. Radiologic findings are often nonspecific. Bony erosion is uncommon [35], but demineralization of the bone has been reported. A well-pneumatized mastoid with chronic otitis media is suggestive of tuberculous otitis media but not diagnostic, as these cases can also have sclerotic and destructive mastoid lesions. Recent studies have shown that CT is the best modality available for the diagnosis of tuberculous mastoiditis; CT provides more information than do standard plain films and it is more accurate and useful than polycycloidal tomography and magnetic resonance imaging [36].

SKIN TEST

This is a routine screening test for tuberculosis. In this test purified protein derivative is used. It is positive in tuberculosis. But a negative test does not exclude the possibility of the presence of tuberculosis [35].

BACTERIOLOGICAL AND HISTOLOGICAL STUDIES

The diagnosis of tuberculous otitis media is based on demonstration of acid fast bacilli within granuloma in biopsy materials, with or without the culture of mycobacterium tuberculosis from the biopsy, aural discharge or aspirate of the middle ear. Demonstration of acid fast bacilli in the ear discharge is difficult due to superadded infection [2]. Unfortunately, culture of the discharge has a low yield. Therefore, the clinician must maintain a high index of suspicion, perform multiple cultures and look diligently for evidence of tuberculous infection of other organs [30]. The positivity of Acid Fast Bacilli in ear discharge varies from 5 to 35% and on repeated examinations it improves to 50% [37]. However, confirming the diagnosis can be difficult because the high rate of secondary bacterial infection of the tuberculous middle ear (79%) can prevent the identification of Mycobacterium tuberculosis on either staining or culture [9,38]. Antibiotic sensitivity to various anti tubercular drugs is gaining importance in recent years because of increase bacterial resistance. Diagnosis is made from direct smear examination and culture of discharge, histopathological examination from middle ear. Histology of tissues reveals granulations with epitheloid cells and multinucleated giant cells (Langhans giant cells), areas of central necrosis, lymphocytic infiltration, ulceration and signs of bone resorption. Histopathological examination of the involved middle ear and mastoid mucosa will show three types of changes: military, granulomatous and caseous [4]. The military type is associated with superficial infection, the granulomatous type with superficial bony involvement, and

the caseous type with massive necrosis and sequestration [4].

OTHER TESTS

If facilities are available, polymerase chain reaction (PCR) of the ear discharge can be done. Other investigations such as erythrocyte sedimentation rate, along with serological status to know the immune status of the patient are done.

TREATMENT

MEDICAL TREATMENT

Antituberculous therapy is the treatment of choice for tuberculous otitis media. The first cures for TOM through antibiotics were reported by Grief and Gould in 1948. The first therapy of success for TOM used only streptomycin, but the current standard chemotherapy using combination of drugs. It should be managed with antitubercular therapy (category-1). It includes 4 drug regimen in first two months (Isoniazid, Rifampicin, Pyrizinamide and Ethambutol) followed by 2 drug regimen in later 4 months (Isoniazid and Rifampicin). These regimens are given as per criteria of Nepal. Currently, the resistance to antitubercular drugs is a major problem and one of the main factors of difficulty in combating the disease.

SURGICAL TREATMENT

Myerson advised a radical mastoidectomy if any of the following complications develop: facial paralysis, subperiosteal abscess, labyrinthitis, mastoid tenderness and headache [25]. Surgery may be required in some cases to remove sequestra and improve drainage [3]. When surgery is combined with adequate chemotherapy, there is a good chance of healing with a dry ear with a good prognosis [17]. Recently, the role of surgery has been revised. In the past, it was done to provide drainage, to control spread to central nervous system and to relieve facial paralysis. The advent of specific chemotherapy has challenged all this, and today surgery should be reserved for decompression of the facial nerve and for removal of necrotic material which might provide a nidus for the organism to remain out of reach of anti tuberculous therapy. Sometimes, demonstration of sequestra in temporal bone during surgery will give a clue to diagnosis.

PROGNOSIS

In the past, many people died of tuberculous otitis media, before the advent of streptomycin. Now with combined anti-tuberculous therapy, the results improved. However, generally there is no hearing improvement [26]. The repair of hearing loss can be achieved after cessation of otorrhoea by

tympanoplasty. The recoveries of sensorineural hearing loss not usually occur with the healing process. Facial paralysis will improve partially or completely. The speed and extent of recovery were directly related to the time interval between the installation of facial paralysis and the start of treatment.

CONCLUSION

Tuberculous otitis media is a rare disease, if left untreated, can damage middle ear and other surrounding structures. It should be considered in differential diagnosis of chronic middle ear discharge that does not respond to usual therapy. Delay in diagnosis can lead to complication. A high level of clinical suspicion is needed for early diagnosis and antitubercular therapy should be started as soon as possible to prevent the possible complication.

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