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Tuberculous Otomastoiditis: A Case Report

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Abstract

Tuberculosis is on the rise in the recent years. Commonest presentation is tuberculosis of the lungs. Tuberculosis of the middle ear cleft is relatively uncommon and often missed by clinicians. A case of tuberculous otomastoiditis with intracranial complication is presented. This case report is to emphasize the fact that high index of clinical suspicion is necessary for the early diagnosis and treatment of this entity which can cause fatal consequences.

Keywords: *Tuberculosis, Otomastoiditis, Facial palsy*

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Introduction

The incidence of tuberculosis has declined in most developed countries as a result of improvement in health care, effective chemotherapy, pasteurization and BCG vaccination[1,2]. Tuberculosis of the middle ear cleft is probably under-diagnosed due to the variation in the classical clinical features as well as lack of histological examination and mycobacterial cultures at all times[2,3]. Thus the delay in diagnosis can lead to irreversible complications.

This article highlights a case of tuberculous otomastoiditis in which the patient developed intracranial complication i.e. hydrocephalus secondary to tuberculous meningitis after the start of anti-tuberculous chemotherapy.

Case Report

A 16 year old girl presented to casualty with a one month history of intermittent fever, right sided ear discharge and facial asymmetry. Two months prior to that, she had been followed up at the Outpatient clinic in Hospital Kuala Lumpur, a tertiary referral center in Malaysia, for right sided otalgia, hearing loss and ear discharge. There were no other neurological symptoms. She denied any contact with a patient with tuberculosis.

On examination she was febrile with right sided lower motor neuron facial palsy. Otoscopy of the right ear showed polyp occupying external auditory canal and purulent discharge with no view of the tympanic membrane. There was no other neurological deficit. A computerized tomographic (CT) scan of the temporal bone demonstrated soft tissue density in the right external auditory canal extending to the middle ear and right mastoid air cells. The scan also showed destruction of right middle ear structures, mastoid air cells and right facial canal, with thinning of right tegmen tympani and meningeal involvement. Ear swab sent for microscopic analysis as well as culture and sensitivity study did not show the involvement of any organisms. Pure tone audiometry showed profound mixed hearing loss in right ear and mild sensorineural hearing loss on left ear. The patient was admitted to ENT (ear, nose and throat) ward and started on broad spectrum intra venous antibiotics (ceftriaxone 2 g stat followed by 1 g daily and metronidazole 500 mg three times a day). Exploration of the right mastoid was performed. Intra-operative polypoidal tissue was found occupying the external auditory canal with total tympanic membrane perforation. The middle ear was full of granulation tissue with erosion of long process of incus and supra structure of stapes. Facial nerve was dehiscent at the second genu.

A modified radical mastoidectomy was performed. Biopsies were sent for histopathological examination. Postoperative period was uneventful. Histopathological examination report revealed inflamed fibrocollagenous tissue with no granuloma or malignancy. The patient was followed up for four weeks post operatively during which the facial palsy remained the same and the ear was dry.

Eight weeks postoperatively, she presented with history of cough and bilateral neck swelling. Subsequently chest x-ray was done and it showed pulmonary involvement. Sputum Ziehl Neelson staining was negative. Cultures of sputum yielded tuberculous bacilli. She was started on anti-tuberculous treatment (isoniazid 250 mg, ethambutol 800 mg, pyrazinamide 1 g, rifampicin 450 mg and pyridoxine 10 mg daily) and discharged.

Despite being on chemotherapy for two months, the patient was readmitted into neurosurgical ward with high fever, right ear discharge for five days and altered sensorium (drowsiness) for one day duration. On examination she was febrile with neck stiffness and positive Kernig's sign. Her Glasgow Coma Scale was 9/11 with sluggishly reactive pupils (bilaterally). Provisional diagnosis of meningitis secondary to tuberculous mastoiditis was made. CT scan of the brain showed communicating hydrocephalus with no intra

cerebral lesion. She underwent extra ventricular drain and ear examination under general anesthesia. Intraoperatively, the patient was found to be having clear cerebrospinal fluid under high pressure. The right ear with minimal pus, granulation tissue and the mastoid cavity were clear. She was treated with broad spectrum intravenous antibiotics (ceftriaxone 2 g stat followed by 1 g daily and metronidazole 500 mg three times a day) and supportive care. Her general condition deteriorated and died two days later.

Discussion

Recently, there has been a resurgence of cases of tuberculosis, some of them occurring in association with HIV/AIDS[4]. Tuberculosis still remains one of the most common lethal infections in the world. The occurrence of the disease in the middle ear was first demonstrated more than a century ago[4]. Primary infection of the middle ear cleft is thought to be rare, and infection is usually due to hematogenous or lymphatic spread or is spread via the Eustachian tube or a preexisting tympanic membrane perforation[2]. The clinical features of this disease have been changing over the years. Classical features such as painless profuse otorrhoea, multiple tympanic membrane perforation, exuberant granulations (described in the early literature) are less frequently seen[1,2,3]. Facial palsy remains a cardinal feature of tuberculous otitis media although facial paralysis is not exclusive to tuberculous infection of middle ear cleft[3,4]. Bone destruction is often a rapid and early feature of tuberculous infection with destruction of ossicles and even cortical bone over the mastoid tip. It is not therefore surprising that the thin bony capsule of the facial nerve is often involved leading to facial palsy as a presenting feature[3]. Tuberculosis must be a prime suspect in case of facial paralysis in chronic middle ear disease with out cholesteatoma[4]. Facial nerve palsy without choleasteatoma was well demonstrated in the patient being reported. Even on histopathological examination, the lesion was not typical of tuberculous granuloma.

Due to variable presentation and changes in the classical features, the diagnosis is often delayed for months or even years and this in turn delays the commencement of anti-tuberculous chemotherapy. Therefore, a high index of clinical suspicion is required for an early diagnosis and initiation of treatment. Timely commencement of therapy can lead to a full recovery and prevent any complication.

Although concomitant pulmonary lesions are found in about 50 % patients[4], there may or may not be a history of contact with a tuberculous patient, as in our patient who had no contact with pulmonary tuberculosis patient.

Bacteriological examination of the aural discharge is not very reliable. Although ear swab from our patient was examined microbiologically, tuberculous culture is not routinely performed for ear swabs. Evaluation should be started with smears, cultures, PCR of otic secretions, a PPD test, chest x-ray and biopsy of granulation tissue. The most reliable diagnostic method remains the histopathological examination of granulation tissue[1,2,4]. High resolution computerized tomograms of the temporal bone are more useful than plain films in providing information about the extent of disease, complications and demonstrate the anatomy[2].

The treatment of choice is anti-tuberculous chemotherapy[1,2,3,4,5]. Traditionally, surgical decompression has been advocated to treat the complication of a facial palsy. However surgery may be required for exploratory and biopsy purposes. Abundant granulation tissue in the middle ear and mastoid air cells in the absence of cholesteatoma, should raise the possibility of tuberculous involvement, especially in the presence of facial paralysis and hearing loss[4].

Conclusion

Otomastoiditis is an uncommon manifesttation of tuberculosis but should be considered in the differential diagnosis of persistent otitis media. In an era of globalization caused by extensive population migration from third world countries to first world countries, a high index of suspicion is important to diagnose tuberculous infection of middle ear cleft. This case highlights the difficulty in establishing the diagnosis of tuberculous otomastoiditis and the irreversible complications.

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