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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, Oтомastoiditis, 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
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cerebral lesion. She underwent extra
terantrum drain and ear examination under
eral anesthesia. Intraoperatively, the
atient was found to be having clear
cerebrospinal fluid under high pressure. The
ight ear with minimal pus, granulation tissue
nd the mastoid cavity were clear. She was
treated with broad spectrum intravenous
itiotics (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
Tuberculosis still remains one of the most
ommon lethal infections in the world. The
occurrence of the disease in the middle ear
was first demonstrated more than a century
ago4. 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
reexisting tympanic membrane perforation2.
he clinical features of this disease have
been changing over the years. Classical
eatures such as painless profuse otorrhoea,
multiple tympanic membrane perforation,
exuberant granulations (described in the
early literature) are less frequently seen1,2,3.
acial palsy remains a cardinal feature of
tuberculous otitis media although facial
alysis is not exclusive to tuberculous
fection of middle ear cleft3,4. Bone
destruction is often a rapid and early feature
of tuberculosis infection with destruction of
ssicles and even cortical bone over the
astoid tip. It is not therefore surprising that
he thin bony capsule of the facial nerve is
en involved leading to facial palsy as a
resenting feature5. Tuberculosis must be a
me suspect in case of facial paralysis in
choric middle ear disease with out
olesteatoma6. Facial nerve palsy without
olesteatoma was well demonstrated in
he patient being reported. Even on

histopathological examination, the lesion was
ot 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
urn 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 % patients4, there may or
may not be a history of contact with a
tuberculous patient, as in our patient who
ad no contact with pulmonary tuberculosis
patient.

Bacteriological examination of the aural
discharge is not very reliable. Although ear
swab from our patient was examined
icrobiologically, tuberculous culture is not
outine performed for ear swabs.
valuation 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 gran-
ulation tissue1,2,4. High resolution computed
erized tomograms of the temporal bone are
ore useful than plain films in providing
formation about the extent of disease,
complications and demonstrate the
atomy2.

The treatment of choice is anti-tuberculous
hemotherapy1,2,3,4,5. Traditionally, surgical
decompression has been advocated to treat
the complication of a facial palsy. However
urgery may be required for exploratory and
opsy purposes. Abundant granulation
tissue in the middle ear and mastoid air cells
 absence of cholesteatoma, should
aise the possibility of tuberculous
volvement, especially in the presence of
acial paralysis and hearing loss4.
Conclusion

Otomastoiditis is an uncommon manifestation 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.

References