Case Report

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# Tuberculous Otitis Media With Incompletely Recovered Facial Paralysis – A Case Report

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## 1. Bullet Point For Clinical Significances

- Tuberculous otitis media (TOM) accounts for only 0.04~0.9% of all cases of chronic suppurative otitis media and is difficult to diagnose because of its low incidence and low suspicion.
- Classic symptoms and signs of TOM, such as painlessness, profuse otorrhea, whitish granulation tissue and multiple perforations are not exactly matched to TOM, recently.
- Although there is no definite consensus of surgical role on TOM, first choice of treatment of TOM would be chemotherapy and surgery may take a secondary role to that of chemotherapy.

## 2. Abstract

### 2.1. Objectives:
Tuberculous otitis media (TOM) is rare and difficult to diagnose because of its low incidence and low suspicion. We experienced the patient with otorrhea and facial palsy and performed unnecessary operation because of delayed diagnosis with low suspicion. We report this case for the purpose of being helpful to other clinicians.

### 2.2. Method:
Clinical findings and courses of case was descripted in detail and literatures about diagnosis and management of TOM were reviewed.

### 2.3. Case report:
A 76 year-old woman suffered from otorrhea and facial palsy visited for treatment and eradicative operation was performed with impression of a chronic suppurative otitis media with facial palsy. Histopathologic findings confirmed the diagnosis as TOM and anti-tuberculous medications were started with some delay. Although findings of inflammation were subsided, incomplete recovery of facial palsy was still remained.

### 2.4. Conclusion:
Facial nerve paralysis could be accompanied with higher frequency in TOM when it is compared to chronic otitis media. Although early diagnosis and treatment is imperative, it is not easy to diagnose early because its signs and symptoms are variable and are not always compatible to classic symptoms. The mainstay of treatment of treatment for TOM is medical therapy with the antituberculous drugs and surgery is known to have limited role.

## 3. Keyword:
Tuberculosis; Otitis media; Facial paralysis; Decompression, surgical

## 4. Introduction

Tuberculous otitis media (TOM) is a rare cause of otorrhea, accounting for between 0.05 – 0.9% of chronic infections of the middle ear. [1,2] Complications include fistulae, facial nerve paralysis, labyrinthitis, osteomyelitis, mastoiditis, hearing loss and spread of infection to the central nervous system. [3] It is therefore imperative that this condition is diagnosed and treated early. Early diagnosis of this disorder is not easy because its scant incidence produces a low index of suspicion in the otologists, [2,4]and its signs and symptoms are variable and have non-specific manifestations with respect to other types of chronic otitis media. [5] Also specific tests for tuberculosis are not routinely performed to patients of otorrhea. The mainstay of treatment of treatment for TOM is medical therapy with the antituberculous. [6] In case of facial paralysis, the prognosis of facial paralysis is not dependent on decompression, but rather on early diagnosis and treatment with antituberculous drugs. [6]
Authors experienced the patient who suffered chronic otorrhea with facial paralysis and estimated the patient as chronic suppurative otitis media. Eradicative surgery with facial nerve decompression was performed. TOM was diagnosed after surgery and started the antituberculous medication with a little delay. Although otorrhea and granulation tissue was controlled after surgery, facial paralysis was persistent after surgery. Authors actually missed the TOM because of its variable and non-specific signs and symptoms. We reported our experience along with literature

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review and it would be helpful to other otologists to diagnose the TOM.

**5. Case report**

A 76 year-old woman visited our department for treatment of right otorrhea for 14 days. She suffered sudden onset of right facial paralysis and hearing loss and visited local tertiary referral hospital 2 months ago. She was diagnosed as idiopathic facial paralysis with 8th nerve involvement and treated with high dose systemic steroid till 14 days. After treatment, facial paralysis improved moderately. Profuse discharge from right ear developed 14 days before visiting our clinic. Ear discharge was persistent despite of treatment. At the outpatient clinic, she complained incomplete recovered right facial paralysis, right hearing loss, and otorrhea. She has no medical history of diabetes mellitus, hypertension, or pulmonary tuberculosis. On physical examination, total perforation of tympanic membrane (TM) and profuse purulent otorrhea was observed (Fig. 1) and facial paralysis was identified as grade II using the House-Brackmann grading system (Forehead wrinkling, eye closure, mouth movement were visible but minimal asymmetry was also visible during face movement.). Pure tone audiometry showed profound sensorineural hearing loss on right side (98 dB HL of air conduction threshold, 98 dB HL of bone conduction threshold) and mixed hearing loss on left side (60 dB HL of air conduction threshold, 31 dB HL of bone conduction threshold). Initial culture study was performed but it did not identified pathogens. She was initially treated with oral antibiotics and otic drop of quinolone at the outpatient clinic for four weeks. The amount of otorrhea was slightly decreased but still persistent. Because of incomplete recovered facial paralysis and persistent otorrhea, surgical procedure was scheduled to improved facial paralysis and control otorrhea.

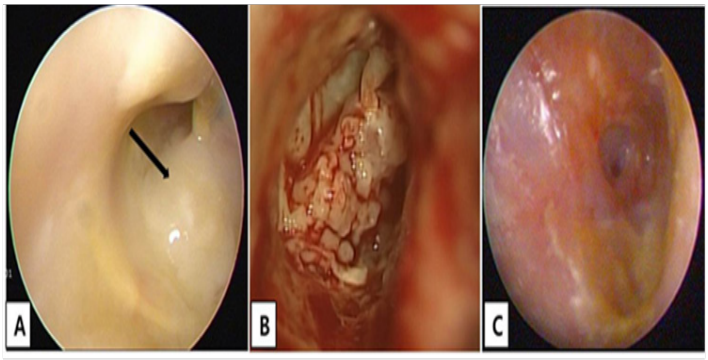


**Figure 1:** Microscopic findings of tympanic membrane and middle ear cavity. (A) Preoperative finding of tympanic membrane. Profuse otorrhea is visible and whitish granulation tissue (black arrow) is also visible in the middle ear cavity (black arrow). (B) Whitish, abundant granulation tissue is filled in whole middle ear mucosa. (C) Postoperative finding. Dry state is maintained with retraction of tympanic membrane at Eustachian tube area.

A preoperative temporal bone computed tomography (TBCT) revealed that a soft tissue density lesion filled the middle ear cavity (MEC), mastoid cells and antrum. There was no finding of attic destruction, bone destruction, cochlear fistula and perilymphatic fistula. Ossicular continuity and dehiscence of bony fallopian canal were not evaluated by TBCT because of fully filled soft tissue attenuation lesion (Fig. 2).

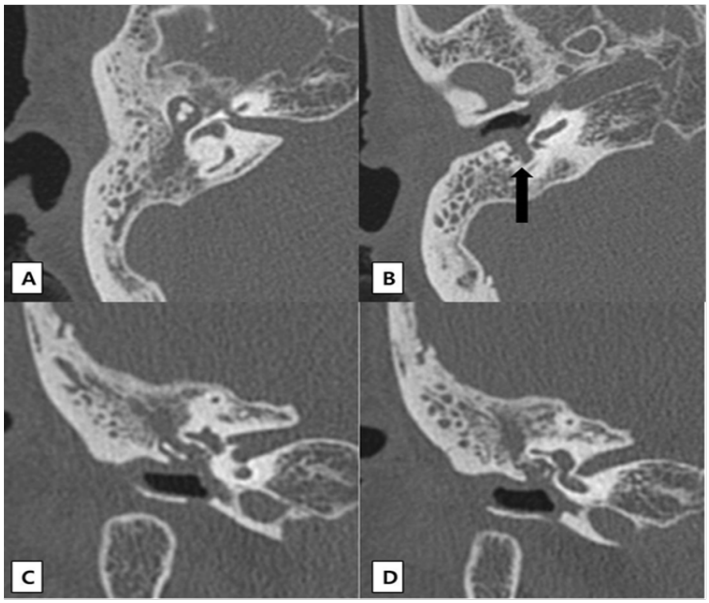


**Figure 2:** Temporal bone computed tomography showed soft tissue attenuation lesion filled in whole mastoid air cells, antrum, and middle ear cavity. (A, B) Axial view. Soft tissue attenuation lesion extend into facial recess and tympanic sinus (black arrow). (C, D) Coronal view. Mastoid bone is well developed with many air cells and attic is intact. Ossicular continuity and dehiscence of fallopian canal are not observed because of soft tissue attenuation lesion.

Total perforation of TM and whitish, pale granulation tissue in the MEC were observed during surgery (Fig. 1) Granulation tissue was filled in all MEC (hypotympanum, mesotympanum, protympanum, and epitympanum) and surrounded the ossicles. The shape of ossicles was relatively intact, but lenticular process was eroded and incudostapedial joint was separated. Mastoid was well pneumatized state but whitish granulation tissue was filled in mastoid air cells, antrum and aditus(Fig. 3). Biopsy was performed at middle ear mucosa and granulation tissue. With consideration of age, hearing status, and mucosal status, radical mastoidectomy with removal of middle ear mucosa was performed. Dehiscence of fallopian canal at proximal portion of second genu was observed and nerve sheath was protruded and torn through dehiscence. Facial nerve decompression was performed from distal geniculate ganglion to distal second genu and nerve sheath was not opened (Fig. 3). Eustachian tube orifice was obstructed with cortical bone chip and denuded MEC was covered with fascia. Histopathologic finding was reported that chronic granulomatous inflammation with caseous necrosis and many bacilli were identified on acid fast bacilli (AFB) stain. The patient was started antituberculous medication with oral rifampin, isoniazid, ethambutol, and pyrazinamide 10 days after operation. After six

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months, the right ear maintained dry state with retraction of protympanic portion (Fig. 1) and there was no change in hearing. Facial paralysis was not changed with House-Brackmann grade II. The patient was still treated with antituberculous medication.

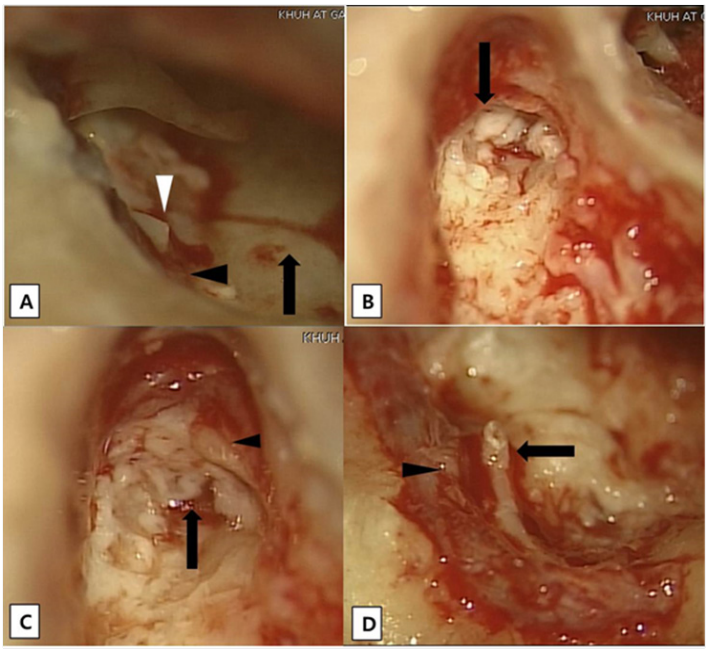


**Figure 3:** Intraoperative findings. (A) Ossicles are relatively normal looking but incudostapedial joint is separated (white arrow head: long process of incus, black arrow head: stapes head, black arrow: denuded promontory). (B) Whitish granulation tissue is existed at the aditus (black arrow: granulation tissue filled in aditus). (C) Granulation tissue surrounds the ossicles (black arrow: granulation tissue, black arrow head: incus body). (D) Damaged portion of facial nerve is visible after facial nerve decompression (black arrow: stapes, black arrow head: protruded and damaged portion of facial nerve).

**6. Discussion**

TOM is a very infrequent cause of otorrhea, [7,8] and the diagnosis of this disease is difficult because of several factors: 1) low incidence (less than 0.9%) of chronic suppurative otitis media makes it difficult to suspect; 2) the clinical signs are variable and often different from the classic description; 3) there is an infrequent association with other systemic involvement of tuberculosis; 4) high rate of false-negative microbiologic examination because of slow growing nature of Mycobacterium species. [9] Peripheral facial palsy has been described in 15% to 40% of TOM cases, mostly in children. [5,6] Facial palsy is a rare but recognized complication of cholesteatoma, of which it can unusually be a presenting feature. Its prevalence has decreased from 1~2.3% of cholesteatoma presentations in the 1950s to 0.04~0.16% of those today. [10,11] Rate of facial palsy in TOM is usually higher than other chronic otitis media. The presence of facial palsy should raise the suspicion of TOM, especially in the absence of cholesteatoma. [12,13] CT findings of TOM in other previous reports included total occupation of the middle ear cavity without evidence of bone erosion and cochlear fistula formation and widespread bone destruction in case of advanced disease. [7,14,15] Sclerotic changes of the mastoid bone in patients with TOM appeared to be less severe than that in other chronic otitis media. Because bony destruction could be a sign of cholesteatoma, acquired cholesteatoma occurs mostly frequently in the Prussak's space, the bone erosion that involves the scutum can be easily be differentiated from the cortical erosion caused by TOM. TOM was not considered at the early stage of treatment in this case and this would be a mistake of authors. But some points, such as profuse otorrhea regardless of antibiotics treatment, large perforation without definite sign of cholesteatoma (attic destruction), and relatively pneumatized temporal bone with total occupation of the middle ear cavity without evidence of bone erosion could be the factors to suspect the TOM in this case. The patient was treated with high dose steroid because of sudden onset of facial paralysis and hearing loss. Profuse otorrhea with incomplete recovered facial palsy was developed after steroid treatment. Idiopathic facial palsy with 8th nerve involvement could be possible.

However, rare incidence of idiopathic facial paralysis with 8th nerve involvement and dehiscence of fallopian canal observed in surgical field can suggest the early TOM at the initial stage of facial paralysis and hearing loss. High dose steroid used initial state could aggravate the tuberculous infection and profuse otorrhea and TM perforation could be developed. The treatment of choice for TOM is the standard pharmacological treatment used for other forms of tuberculosis. [6] Previous studies have reported rapid resolution of otorrhea, closure of tympanic perforations, and disappearance of facial palsy. [6,16] The effects of treatment on hearing loss have been classed as unpredictable. Early treatment onset is the best guarantee of optimal recovery without complications. The overall role of surgery in TOM remains controversial. Exploration or decompressive surgery to facial palsy is regarded as totally unnecessary as recovery is dependent on early diagnosis and treatment with antituberculous drugs. Another case report about TOM with facial palsy suggested the surgery to TOM because resolution of granulation tissue may be slow in TOM, and that therefore the inflammatory process continues to be a threat to nerve integrity. [17] Although there is no definite consensus of surgical role on TOM, first choice of treatment of TOM would be chemotherapy and surgery may take a secondary role to that of chemotherapy. In this case, authors did not suspect the TOM during surgery and decompression was performed. Chemotherapy started 10 days after surgery. It could be possible that incomplete final recovery of facial palsy could be caused by injury during surgery. Because many cases of TOM are hard to diagnose, careful and minimal surgery may provide a more prompt and accurate diagnosis for suspicious cases. This will avoid a delayed diagnosis and possible complications, and may provide more rapid, complete healing as well. If tuberculosis has not been suspected clinically, the diagnosis will be missed. Clinical signs, characteristic CT findings, and laboratory findings will be considered comprehensively. And clinicians, suspicion and knowledge will be the most important factor to diagnose the TOM.

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