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CASE REPORT

# Bilateral Facial Paralysis Caused By Tuberculous Otitis Media: A Case Report

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A case of bilateral facial paralysis caused by tuberculosis is presented.

A 28-year-old woman was referred to our clinic for bilateral facial paralysis, who presented with hearing loss and vestibular symptoms. An audiogram revealed a mixed type hearing loss of 97dB in the left ear and of 65dB in the right ear. On temporal bone CT imaging, the patient was found to have bilateral granulation tissues occluding the tympanic cavity, and mastoid cells. Neurological examination showed bilateral House-Brackmann stage V peripheral facial paralysis. After the operation and antituberculosis treatment, the facial paralysis regressed to stage II on the right side, and stage III on the left side on the following fifth month.

Bilateral facial paralysis due to the tuberculosis otitis media is very rare; however, if the laboratory testing does not reveal any other cause, or if the response to treatment is unsatisfactory, tuberculous otitis should be suspected.

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## Introduction

Unilateral facial nerve palsy is a relatively common neurologic disorder, and its incidence is estimated at 20 to 25 per 100,000 population [1,2]. Bilateral involvement of the facial nerve, defined as appearance of paresis or paralysis of the contralateral facial nerve within 30 days of the onset of the first side [3], is a very rare entity, occurring in only 0.3% to 2% of patients with facial paralysis [4]. Adour found only 3 bilateral cases in a consecutive series of 1,000 patients with Bell's palsy [5].

Bilateral facial paralysis may appear often as one of the symptoms of a systemic disease requiring urgent treatment, including Lyme disease, Guillian-Barre syndrome, leukemia, sarcoidosis, bacterial meningitis, infectious mononucleosis, and cranial fracture [6, 7, 8, 9].

Early diagnosis of tuberculous otitis media is dependent on a high index of suspicion. It classically presents with an intractable, antibiotic-resistant, profuse, painless discharge from the ear. A large central perforation of the tympanic membrane is usual, but a total or multiple perforations may also be present. Middle ear mucosa is pale and the middle ear cavity is filled with pale, soft granulation tissue [10].

In the literature, a few cases describing nuclear Bell's palsy with pulmonary microbial infection and bilateral facial paralysis caused by tuberculosis mastoiditis have been reported. However, bilateral facial paralysis due to the tuberculosis otitis media is exceedingly rare. This case serves to demonstrate both the difficulty in establishing the diagnosis of otologic tuberculosis and its potentially devastating consequences.

## Case Report

A 28-year-old woman was referred to our clinic because of bilateral facial paralysis. Left facial paralysis occurred 15 days ago. While the patient received prednisolone and acyclovir treatment, right peripheric facial paralysis occurred. The patient complained of bilateral ear pain, vertigo, and tinnitus in the left ear. There was no history of ear discharge for at least one year. The patient had history of left facial paralysis one year ago and underwent surgery due to chronic otitis media. After the operation, the facial paralysis had recovered completely. Otoscopic examination revealed yellowish granulation tissues obliterating both ear canals. the tympanic membranes were perforated bilaterally. Neurological examination showed bilateral House-Brackmann stage V peripheral facial paralysis (Figure 1A). Other cranial nerves were normal upon neurologic examination. Romberg testing was negative. Cerebellar testing did not reveal any abnormality. Spontaneous or gaze nystagmus were not

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present. On her audiogram, a mixed type hearing loss of 97dB in the left ear and of 65dB in the right ear was present. Complete blood count, comprehensive metabolic panel, chest radiography, and brain computed tomography scan showed no abnormality. Temporal bone CT x-rays showed occlusion of the tympanic cavities bilaterally including epitympanic recesses, mastoid antrum and mastoid cells. Temporal MRI examination showed diffuse inflammation in both middle ears and mastoid cavities appearing hyperintense on T2 and showing slight enhancement with contrast (Figure 2). A right modified radical mastoidectomy was performed with total excision of the external ear canal skin since the skin of external ear canal was edematous and granulated. The mastoid antrum, mastoid cells, and the middle ear cavity were found to be obliterated with yellowish granulation tissues (Figure 3). A left radical mastoidectomy was done at the same because of the polypoid granulation tissues filling the mastoid and middle ear cavities. Ossicular chain was eroded and a defect was present at the oval window that was repaired with a cartilage graft. Bilateral fallopian canal were not dehiscent.

Pathological examination of the specimens revealed granulomatous inflammation (Figure 4A, 4B), which recalled the tuberculosis infection. The patient underwent bronchoscopy and bronchoalveolar lavage was performed. There was no mass in the lungs and no acid resistant bacteria growth was observed in the bronchoalveolar lavage fluid culture. The PPD test was measured as 11mm.

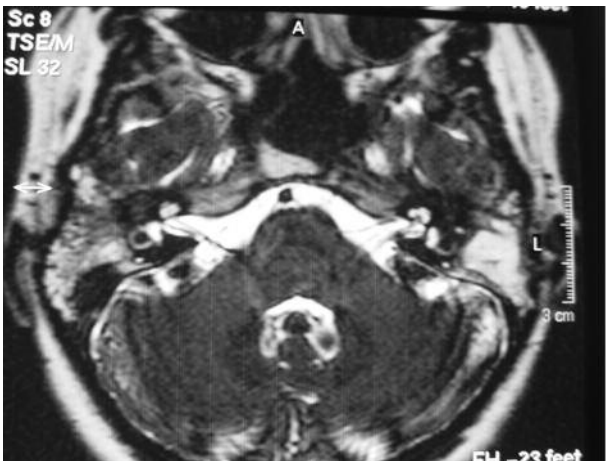


**Figure 2.** T2 MRI examination showing bilateral inflammatory signal enhancement in mastoid cells and 2x1 cm loculated cystic mass in the left ear

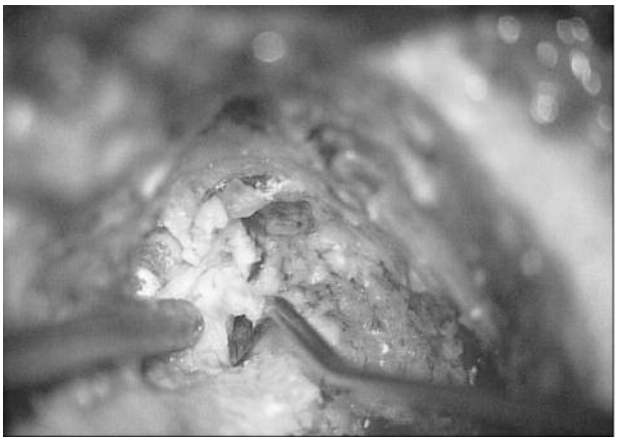

**Figure 3.** In the left ear mastoid antrum, mastoid cells, and the middle ear cavity were obliterated with yellowish granulation tissues

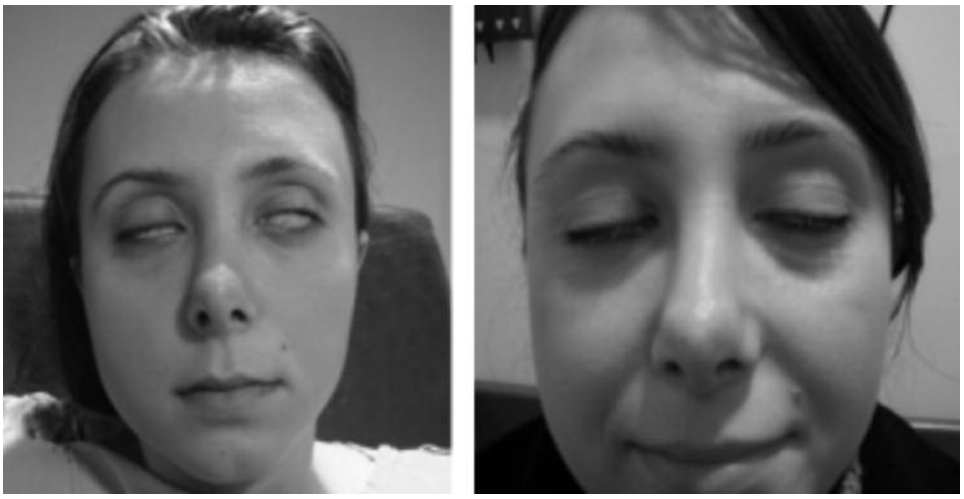

**Figure 1A-1B.** Patient had bilateral House-Brackmann stage V peripheral facial paralysis. The facial paralysis regressed to stage II on the right side, and stage III on the left side on the fifth month following the operation

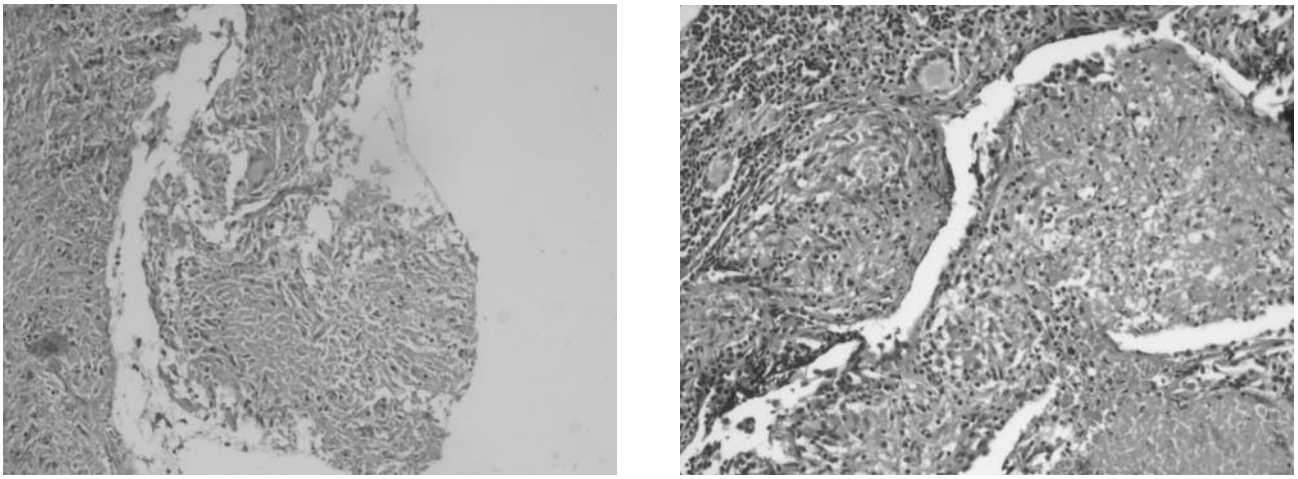

**Figure 4A-4B.** Pathological examination revealed granulomatous inflammation which consisted of large macrophages and lymphocytes

Antituberculosis treatment composed of isoniazide, rifampicin, ethambutol, and pyrazinamid was initiated. The facial paralysis regressed to stage II on the right side, and to stage III on the left side in the fifth month following the operation (Figure 1B). Audiological examination revealed a mixed type of 82 dB in the left ear and a 42 dB in the right ear.

## Discussion

The etiology of facial paralysis includes many conditions such as congenital, traumatic, infectious, neurological, metabolic, neoplastic, toxic, vascular, and idiopathic [3].

The priority in the work-up of bilateral facial paralysis is to rule out a life-threatening disease such as leukemia, Guillain-Barre syndrome, Lyme disease, bacterial meningitis, Moebius syndrome, and cranial trauma [6, 7, 8, 9]. Careful history taking, physical examination, appropriate laboratory studies, and appropriate radiologic examinations are essential for identifying the cause of bilateral facial nerve palsy. Magnetic resonance imaging is needed to demonstrate seventh cranial nerve lesions, tumor cell infiltration, and widening of the internal acoustic canal. Also, areas that are most important to visualize are the central nervous system, skull base, meninges, and cerebellopontine angle, which are best imaged by enhanced MRI.

Bilateral facial nerve paralysis from acute otitis media is extremely rare but has been reported by several authors [11, 12]. All reported cases resolved completely with antibiotics and tympanostomy tube placement. Epstein-Barr virus, HIV, and Mycoplasma pneumoniae ear infections have recently been reported as causes of bilateral facial nerve palsy [13, 14, 15].

The most common infectious cause of facial diplegia is Lyme disease, caused by Borrelia burgdorferi. It is commonly seen in the summer with its typical skin lesion; the erythema migrans. The diagnosis is made by an immunologic assay using antibody titers against the spirochete [8].

Guillain-Barre syndrome or ascending inflammatory demyelinating polyneuropathy presents as a progressive development of palsy of the voluntary muscles of the legs, arms, trunk, and face. The most commonly affected cranial nerves are IX, X, and VII [16]. Miller Fisher syndrome consists of a triad of ataxia, ophthalmoplegia, and areflexia. These two syndromes may include bilateral facial paralysis in 27% to 50% of the cases and can follow a Campylobacter jejuni infection [16].

Metabolic causes of adult facial diplegia include polyneuropathies caused by diabetes mellitus [8] and acute porphyria crisis [17].

Neurological complications such as cranial nerve palsies, mononeuritis multiplex, meningoencephalitis, and Guillain-Barre syndrome are present in 1-5% of all patients with acute EBV infection. Approximately 40% of the facial nerve palsies associated with EBV are bilateral [13].

In the literature, 20 cases of bilateral facial paralysis during the course of HIV infection have been described. HIV has been isolated from peripheral nerve tissue, supporting the hypothesis of a direct lesion of the facial nerve by the neurotropic virus [18].

Facial nerve palsy and otologic manifestations have been reported during the course of Wegener's granulomatosis (WG), but it is extremely rare as the presenting features. In the literature only two cases of bilateral facial palsy as the presenting sign of WG are reported. The testing of anticytoplasmic antibodies versus neutrophil polymorphonucleate granules (c-ANCA) are highly specific for the diagnosis of WG, being positive in 97% of the cases [19]. Involvement of the facial nerve is a relatively common neurological finding in sarcoidosis. However, bilateral involvement is very uncommon and is even more unusual as the presenting complaint [20].

The identification of a tuberculous bacillus at the site of the lesion obviously is confirmatory. However, this is not always possible and clinical (including skin testing), radiological and histopathological findings in association with good results achieved by anti-TB treatment are important for the diagnosis [21, 22].

Since the patient had bilateral facial paralysis, findings of labyrinthitis, otorrhea, and a history of previous ear surgery on presentation; it was assumed that the current condition was due to a complicated chronic otitis media and emergency surgery was undertaken. During the operation, the fallopian canals were observed to be intact which suggested that the facial paralysis was caused by an inflammatory process rather than mechanical. In the present case, we did not identify the tuberculous bacillus, but we had a pathological diagnosis suggesting a possible tuberculosis infection.

Antituberculous chemotherapy is the treatment of choice for this disorder. The role of surgery is limited to biopsy for diagnostic purposes, the management of intracranial complications and for removal of bony sequestrae[21, 22]. We performed bilateral mastoidectomies to remove the inflammatory tissues. This type of approach is a highly controversial for tuberculous mastoiditis, but the patient had no preoperative signs suggesting a tuberculosis infection. However, severe intraoperative findings such as an oval window defect with the potential of intracranial spread or total hearing loss were observed and we think that surgical removal of inflammatory tissues provided a faster response compared to medical treatment only.

## Conclusion

The prognosis for bilateral facial palsy is dependent on the underlying etiology. Detailed work-up is required to find the underlying cause. If the laboratory testing does not reveal any other cause, or if the response to treatment is unsatisfactory, tuberculous otitis should be suspected.

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