

Post-traumatic Bilateral Facial Paralysis - A Case Report -

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Bilateral facial paralysis is a rare clinical entity. It is also diagnostic challenge which, unlike unilateral facial paralysis, it is can be difficult to recognize because of a lack of facial asymmetry. A 22 year-old man referred for neurological evaluation because of bilateral facial paralysis (House-Brackmann grade V). He has initially presented unconsciousness after he sustained closed head injury after a motor vehicle accident. Initial computed tomography (CT) scans revealed a small epidural hematoma, right temporal bone fracture and air densities around the basal cistern. On the 10th hospital day, he was noted to have incomplete closure of both eyes and a feeding difficulty with drooling. Electrodiagnostic testing confirmed the diagnosis of bilateral facial paralysis. The high-resolution CT scans showed bilateral temporal bone fractures with no facial canal involvement. There was no surgical intervention based on delayed onset of facial paralysis and the findings of the high-resolution CT scans. He recovered incompletely on post-injury 7 month without treatment. we report a rare case of post-traumatic bilateral facial paralysis with literature review.

Key Words: Facial paralysis · Head injuries · Temporal bone



INTRODUCTION

Facial paralysis can be an extremely debilitating disorder. Bilateral facial paralysis is a rare condition that is unusual in the neurosurgical literature. Its incidence is one case per 5 million population per year⁶⁾, when compared to unilateral facial paralysis with 1,000 cases per 5 million population per year¹⁴⁾. The causes of bilateral facial paralysis range from infections, tumors, head injuries, degenerative diseases, vascular diseases etc. Head injuries is responsible for about 5 per cent of all cases facial paralysis^{13,15)}. Temporal bone fracture is a well-known cause of facial paralysis, and is responsible for approximately 3 per cent of bilateral facial paralysis^{7,13)}. It is also diagnostic challenge which, unlike unilateral facial paralysis, it is can be difficult to recognize because of a lack of facial asymmetry.

In this report, the authors present a rare case of bilateral facial

paralysis due to bilateral temporal bone fractures and conducted a review of relevant literatures.



CASE REPORT

A 22 year-old man referred for a neurological evaluation on the post-injury 21 day because of bilateral facial paralysis (House and Brackmann (HB) grade¹⁰⁾ V). He was admitted to intensive care unit by closed head injury with multiple injuries including rib fractures, pelvic bone fracture after a motor vehicle collision. Initial neurological examinations disclosed stuporous mentality. Skull X-rays showed right temporal bone fracture (Fig. 1A). Initial head computed tomography (CT) scans revealed a small epidural hematoma, right temporal bone fracture and air densities around the basal cistern and severe brain edema (Fig. 1B, 1C). On the 10th hospital day, he was lethargic, oriented to time, place, person. He was noted to have incomplete closure of both eyes and drooling during a feeding trial. He had difficulty moving his lips when attempting to talk or chew. His facial expression was symmetric, but there is no emotional response. The diagnosis of bilateral facial paralysis was made based on the history, neurological examinations. Repeated neurological examinations revealed

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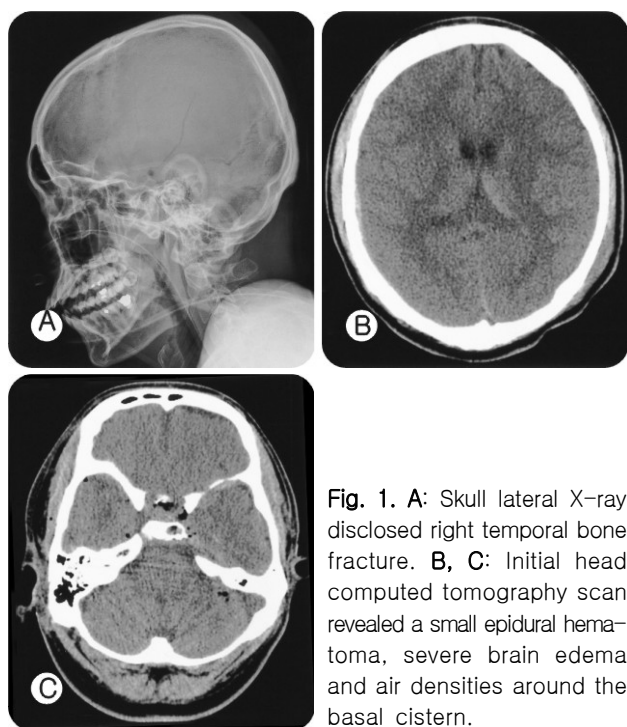


Fig. 1. **A:** Skull lateral X-ray disclosed right temporal bone fracture. **B, C:** Initial head computed tomography scan revealed a small epidural hematoma, severe brain edema and air densities around the basal cistern.

bilateral facial paralysis (HB grade V), impaired lacrimation in both eyes, hyperacusis, taste disturbance in anterior 2/3 of tongue. But, hearing and facial sensation was normal. Schirmer's test was impaired on only left eye. The audiometry was normal on the both ears. The laboratory examinations including complete blood cell counts, liver function tests, ESR, CRP, Lyme titer, VDRL and FTA-ABS, HSV titers, varicella-zoster virus titers, HIV titers, chest x-rays etc. was within normal limit. Electrodiagnostic testing confirmed the diagnosis of bilateral facial paralysis. Blink reflex tests showed bilateral peripheral conduction defects and electromyography (EMG) revealed increased insertional activity, fibrillations and positive sharp waves, severe denervation potentials in both sides.

A high-resolution CT scans showed bilateral temporal bone fractures with microfractures, hematoma fluid densities in both mastoid cavities (Fig. 2). Furthermore, the transverse fracture of right petrous bone was extended from squamous portion, the medial aspect of epitympanum to the vicinity of the geniculate ganglion, but, there was no involvement of the Fallopian canal. The longitudinal fracture of left petrous bone fracture was not distinct, but, the Fallopian canal was intact. Air density was observed in left internal acoustic meatus. Contrast-enhanced magnetic

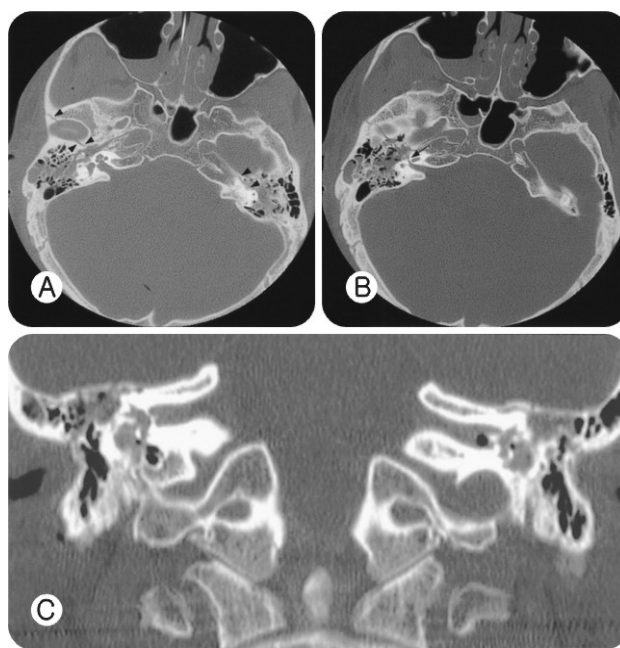


Fig. 2. **A, B, C:** High-resolution computed tomography scan showed bilateral temporal bone fractures (arrow head) with microfractures, hematoma fluid densities in both mastoid cavities. the transverse fracture of right petrous bone was distinct, but, there was no involvement of the Fallopian canal (black arrow).

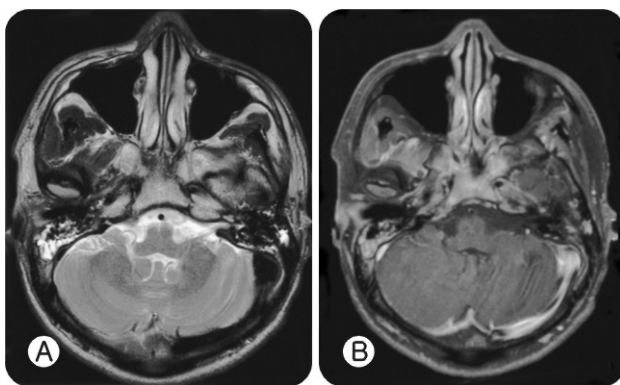


Fig. 3. **A:** T2-weighted magnetic resonance image revealed hemorrhagic densities in both mastoid cavities, but, no hemorrhage and edematous change around the facial nerve on both sides. **B:** Contrast-enhanced magnetic resonance image demonstrated no enhancement of the distal intrameatal and labyrinthine segments.

resonance (MR) imaging showed no enhancement of the distal intrameatal and labyrinthine segments (Fig. 3) The patient was placed on corticosteroids. Surgery was not carried out because of

delayed onset of facial paralysis and no Fallopian canal involvements in the high-resolution CT scans. There was gradual improvement of the paralysis on 8 weeks after diagnosis. He was a HB grade II/III 7 months after diagnosis.



DISCUSSION

Bilateral simultaneous facial paralysis is described as facial paralysis involving both sides of the face occurring within four weeks of each other, and is found in 0.3-2 per cent of facial paralysis¹⁶. The etiologies of bilateral facial paralysis range from infections, tumors, head injuries, degenerative diseases, vascular diseases, idiopathic. The most striking additions to the differential diagnosis of bilateral facial paralysis are Lyme disease (36%), most common etiology, Guillain-Barre syndrome (5%) and AIDS (0.9%)⁵. The common cause of bilateral facial paralysis is Lyme disease (36%), caused by *borrelia burgdorferi*, spirochete³. Facial paralysis has been shown to occur in 11 per cent of patients with Lyme disease and is bilateral in 30 per cent of these patients³. A diagnosis of a bilateral Bell's palsy (9%) should only be made after an exhaustive search for possible causes of the disorder have been excluded, which, is caused by viral infections, vasospasm, an autoimmune phenomenon⁵. Guillain-Barre syndrome is thought to be a post-infectious viral inflammatory polyradiculoneuritis. Bilateral facial palsy has been reported in 50% of patients with facial paralysis and may be the only clinical manifestation of Guillain-Barre syndrome⁵.

About 5 per cent of all cases of facial paralysis results from head injuries¹⁵. The temporal bone is affected in more than one third of basilar fractures¹². Whereas unilateral injury is commonly observed, bilateral temporal bone fractures are unusual¹⁴. Temporal bone fracture is a well-known cause of facial paralysis, and is responsible for approximately 3 per cent of bilateral facial paralysis^{7,8,13,14,15,16}. Unfortunately, the early diagnosis of bilateral facial paralysis in the traumatic brain injury can be particularly challenging due to the severe nature of the injury, associated cognitive and affective deficits and other secondary complications^{8,13,16}.

In the diagnosis of bilateral facial paralysis, the most important aspect of the evaluation is a thorough history and a complete physical examination, including complete head and neck examination, complete neurological examination, Shirmer's test, electrodiag-

nostic tests. All patients with bilateral facial paralysis should perform through evaluations. Unilateral facial paralysis was usually idiopathic (i.e. Bell's palsy), whereas bilateral paralysis usually has an underlying pathology⁶. In this case presented, The laboratory examinations including complete blood cell counts, liver function tests, ESR, CRP, blood glucose, Lyme titer, VDRL and FTA-ABS, HSV titers, varicella-zoster virus titers, HIV titers, chest x-rays were within normal limit.

EMG confirm the presence of a demyelinating neuropathy affecting both facial nerves and is helpful to determine prognosis. High-resolution CT scans make it possible to visualize the fracture line and its relationship to the Fallopian canal. Darrouzet et al.⁴ suggested that the findings of high-resolution CT scans played a important role in decision-making for surgery. Immediate-onset facial paralysis with temporal bone fracture through the Fallopian canal is an indication for early surgical intervention^{2,12}. Consequently, High-resolution CT scans, with the contribution of electrodagnostic tests and clinical judgment, have the greatest impact in decision making for treatment. Recently, contrastenhanced MR imaging can reveal inflammatory facial nerve lesions and traumatic nerve injury, including clinically silent damage in trauma; Enhancement of the distal intrameatal and labyrinthine segments is specific for facial nerve palsy¹¹.

The efficacy of steroids remains controversial in patients with facial paralysis. But, steroids is usually administrated to patients with either traumatic or infectious facial paralysis. Steroids have been used to reduce edema, swelling and scar formation. Marginal benefit of steroid treatment in idiopathic facial paralysis is demonstrated in a randomized double blind controlled study¹. Also, An important management aspect of patient care is the prevention of exposure keratitis with the use of artificial tears and lubricants.

The prognosis for bilateral facial paralysis is dependent upon the underlying etiology. If the etiology can be identified and successfully managed, the prognosis is excellent.

In this case with bilateral temporal bone fracture, transverse and longitudinal, respectively, both fractures did not extend into Fallopian canals in high-resolution CT scans. Accordingly, delayed-onset facial paralysis developed. This fact suggests that facial paralysis was caused by delayed arterial spasm, edema, external compression by hematoma fluid. Therefore, surgery is not indicated

in this case. Ultimately, on the post-injury 7 month, the patient recovered incompletely with conservative treatment.



CONCLUSION

Only early clinical detection and therapeutic interventions are important for functional recovery after facial nerve injury. In the traumatic brain injury with the temporal bone fracture, particularly, high suspicion of bilateral facial nerve injuries is always warranted because the lack of facial asymmetry can make the clinical detection of bilateral facial palsy more difficult.



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