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Case Report

An unreadable face: our experience

Albin B. Payyappilly*, Juthika S. Rai, B. V. Chandre Gowda, Nagaraj B. Thimmasettaiah, Marie C. Sharafine Stephenyar

Department of Otorhinolaryngology, M. V. J. Medical College and Research Hospital, Bangalore, Karnataka, India

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***Correspondence:**
Dr. Albin B. Payyappilly,
E-mail: albin2rhyme@gmail.com

ABSTRACT

Bilateral facial nerve palsy (FNP) is a rare condition, representing less than 2% of all cases of FNP. Majority of these patients have underlying medical conditions, ranging from neurologic, infectious, neoplastic, traumatic, or metabolic disorders. The differential diagnosis of its causes is extensive and hence can present as a diagnostic challenge. We report a case of a 27-year-old male who presented to the ENT OPD with sequential bilateral facial nerve paralysis which was delayed onset and post traumatic. This article is an attempt by the authors to stress the importance of combing clinical examination and radiological findings to decision making and to evaluate the various treatment options in this rare entity.

Keywords: Facial paralysis, Temporal bone fracture, House brackman grade

INTRODUCTION

Bilateral facial nerve palsy (FNP) is a rare clinical entity and unlike its unilateral counterpart, it is seldomly seen as a consequence of Bell's palsy.[1]

There have been several causes attributed to this condition of which traumatic fracture of the temporal bone is a significant contributor.[2] Post traumatic bilateral FNP apart from being credited as rare also has unique otoneurologic considerations and is a socially debilitating condition.[3] In addition to the above, the lack of facial asymmetry which is typically present in unilateral facial paralysis makes this entity a diagnostic challenge.[3] We are presenting a case of delayed onset bilateral lower motor neuron FNP which developed after 14 days of head injury along with a brief discussion of its diagnostic challenges and appropriate management, thus reiterating the need for further study on this perplexing yet intriguing entity.

CASE REPORT

A 27-year-old male came to our outpatient department with complaints of intermittent right ear bleed for one month duration. Patient presented with history of road traffic accident one month back following which he sustained injury to the head with history of loss of consciousness for a duration of 30 minutes along with right ear and nose-bleed. At the time of initial presentation following the head injury, patient was conscious, oriented without any neurological deficit. His vitals were normal.

Routine CT head revealed acute SDH in left frontal convexity, minimal SAH in left frontal region and small acute haemorrhagic contusions in bilateral cerebral hemispheres. Longitudinal fractures were seen on bilateral petrous bone extending into squamous part and parietal bones on either side associated with right hemotympanum. There was no evidence suggestive of CSF otorrhea or any neurological deficit. The Patient

was managed conservatively and his stay in the hospital was uneventful. The Patient was then discharged on ninth day of admission.

Figure 1: (A) Inability to close both eyelids. (B) Absent frowning of forehead. (C) Bilateral air leakage when blown against resistance.

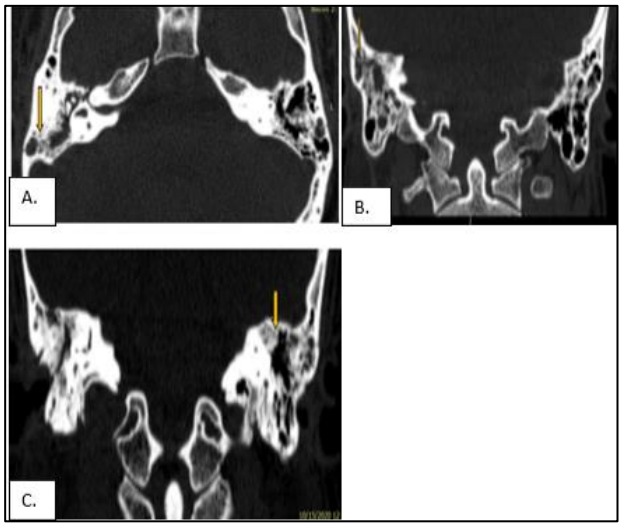

Figure 2: (A) Axial HRCT temporal bone showing Fracture of right petrous bone + heamotympanum. (B) Coronal view showing fracture of right petrous bone. (C) Coronal view showing fracture of left petrous bone.

Five days following his discharge from hospital, the patient developed inability to close both eyes and difficulty in moving his lips. On presentation to the outpatient department, patient was afebrile without any features suggestive of meningitis. His facial expression was symmetric without any emotional response. His hearing on both sides were decreased and facial sensation was normal. A detailed neurological examination revealed bilateral lower motor neuron facial palsy House Brackman grade 6. Pure tone audiometry was then done which showed moderately severe mixed hearing loss in both ears. Further evaluation in the form of topo diagnostic tests revealed the following. Schirmer's test was positive on right side and negative on left side and there was taste disturbance on anterior 2/3 of the tongue.

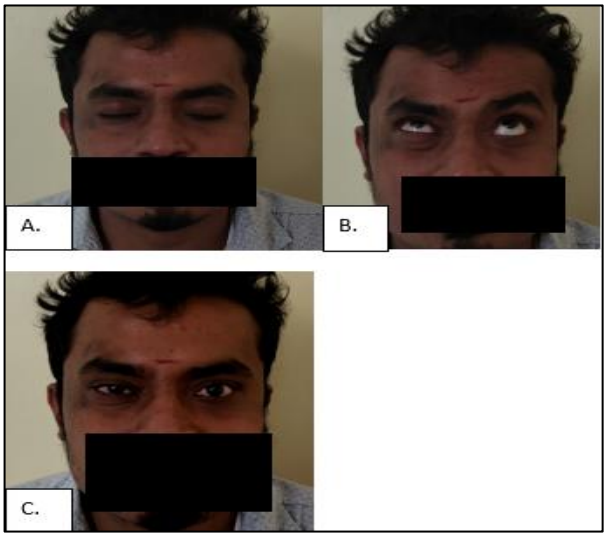

Figure 3: (A) Patient able to close both the eyelids after 6 months of follow up. (B) Able to frown forehead. (C) Able to blow against resistance.

Based on the history and a thorough neurological examination, a diagnosis of bilateral facial nerve paralysis was made (Figure 1A-C). Electro diagnostic testing confirmed the diagnosis of bilateral facial paralysis.

CT head with HRCT temporal bone was done. HRCT of right temporal bone revealed longitudinal fracture across mastoid and mastoid antrum extending into tegmen plate. HRCT left temporal bone showed undisplaced fracture through lateral cortex of mastoid extending into the squamous part (Figure 2A-C). Patient was managed conservatively with systemic antibiotics, systemic steroid (prednisolone) which was tapered after 3 days and multivitamins. This was then followed by oral steroid in a dose of 20 mg thrice a day for the initial 5 days which was tapered gradually over a period of 15 days. Ophthalmic care was also given in consultation with Ophthalmology and he was prescribed Moxifloxacin eye drops with artificial tear drops and an eye patch.

The aural bleed subsided by the third day and left side facial weakness improved. The patient was discharged after seven days on oral medication and was counselled about eye care and physiotherapy to the facial muscles.

On follow up of two months, weakness on left side of the face had improved to House Brackman grade V and on right side the weakness of grade VI was persisting.

At follow up of 3 months his facial weakness was still persisting with only minimal improvement. Patient was

sent for electroneuronography study which showed more than 90% axonal loss on right side. A further option of surgical intervention was given and patient was referred to a higher centre.

The patient opted to wait for a period of another 3 months at the end of which he presented to the OPD for follow up. A thorough neurological examination revealed significant improvement of his facial weakness.

After 6 months of follow up from initial insult there was a significant improvement in both motor and sensory components of facial nerve which improved his quality of life physically, mentally and socially.

He was able to close both the eyelids completely without any ophthalmic complications, and after a short period of time, he also regained the ability to frown his forehead, blow against resistance and maintained nasolabial fold with mild residual disease on left side.

Patient regained the initial loss of taste sensation on the anterior 2/3rd of tongue. His repeat Pure Tone Audiometry done after 6 months improved from bilateral moderately severe mixed hearing loss to bilateral mild conductive hearing loss. There was an overall improvement from House Brackman grade VI to grade I on right side and grade VI to grade III on left side (Figure 3A-C).

DISCUSSION

Bilateral FNP is a rare entity with an incidence of 1 in 5 million population per year.[1] These numbers represent less than 2% of all FNP.[2] The facial nerve is the second most common cranial nerve, after Olfactory nerve to be involved in head injuries, although some authors report it to be the most common.[4] Trauma is the second most common identifiable cause of facial nerve paralysis with Temporal bone fracture being a well-known cause of facial paralysis, and is responsible for approximately 3 per cent of bilateral facial paralysis.[2] The causes attributed to this rare entity has a varied spectrum ranging from degenerative diseases, tumours, infections, vascular diseases and head injuries.[3]

Throughout its course from the pons to the extracranial segment the facial nerve is subject to trauma either by inadvertent iatrogenic injury or accidental trauma. Injury to the intratemporal part of the facial nerve is caused most commonly by temporal bone fractures resulting from trauma sustained in motor vehicle accidents or other blunt or penetrating injuries.[4,5] Unilateral FNP is more common with transverse fractures (40-50%), while longitudinal fractures make up about 20% of the cases.[6]

De Villiers proposed that the longitudinal fracture of petrous part of temporal bone can lead to backward displacement of the petrous apex and coronal splitting of the body of sphenoid leading to mirror image fracture in the opposite temporal bone producing bilateral FNP, while a transverse fracture of the petrous bone will not involve bilateral facial nerves.[6] A possible explanation of delayed FNP is either the bleeding into the facial canal with increasing size of hematoma or delayed swelling of the nerve leading to compression within its fibrous sheath or epineurium.[7]

The principles of management of bilateral facial nerve paralysis varies with the time of onset of the condition. Immediate onset of paralysis following injury warrants immediate surgical repair if the patient is stable clinically or within the next 72 hours. However, delayed onset of bilateral facial paralysis following temporal bone fractures warrants a more cautious approach with a decision between conservative and surgical treatments.[7]

The early diagnosis of bilateral facial paralysis in traumatic brain injury can be particularly challenging clinically due to lack of facial asymmetry, hence the need for all patients with bilateral facial paralysis to be thoroughly evaluated. A detailed history, complete physical and neurological examination, topognostic tests and electro diagnostic tests are paramount in the evaluation and diagnosis of bilateral facial paralysis. Electromyography (EMG) plays a vital role in predicting prognosis. It confirms the presence of voluntary action potentials which indicate the possibility of nerve reinnervation even in the absence of facial movement.[8]

Radiological evaluation in the form of High-resolution computed tomography (HRCT) of the temporal bone with 1 mm thin cuts in axial and coronal planes with “bone window” enhancement of temporal bone is an invaluable diagnostic tool for traumatic FNP, as it can visualize the fracture line and its relationship to the fallopian canal9. Conservative treatment in the form of steroids and vasodilators are recommended for patients with delayed onset facial weakness along with other supportive therapy and frequent follow up. In cases of non-recovery or within six months after trauma late surgery may be recommended.[9]

MR imaging with contrast can reveal inflammatory facial nerve lesions and traumatic nerve injury. Enhancement of the distal intrameatal and labyrinthine segments is specific for FNP.[10]

The surgical approach depends on the site of the injury to the nerve and hearing status. If the hearing is preserved then a trans mastoid or middle fossa approach is used to decompress the nerve, where as in patients where hearing has not been preserved, a trans labyrinthine approach can be used. Nerve must be fully exposed during exploration to identify all injured segments and removal of any fracture fragments coursing compression. In all cases a direct end-to-end anastomosis should be performed and is preferable to grafting if a tension-free suture line can be obtained in presence of complete transection of the nerve.[7]

In our case, the patient although was offered an option of surgical intervention at the end of 3 months, he preferred to delay it till a period of 6 months at the end of which there was significant improvement in his condition which facilitated the return of his quality of life to near normal. Hence, he did not opt for any further intervention.

CONCLUSION

Traumatic delayed bilateral FNP is a rare clinical entity. Due to the lack of facial asymmetry which is typically seen in unilateral facial paralysis, bilateral FNP is also a diagnostic challenge. A thorough history with complete physical and neurological examination along with electro diagnostic tests is mandatory to diagnose bilateral facial paralysis. HRCT 1 mm thin cuts of temporal bone and contrast MRI are the important diagnostic tools with prognostic values. The prognosis for bilateral facial paralysis depends upon the underlying aetiology, having excellent prognosis in the presence of an identifiable cause. We acknowledge that a more detailed understanding of the prognostic indicators and treatment protocols are warranted in the form of further research on this very intriguing condition.

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