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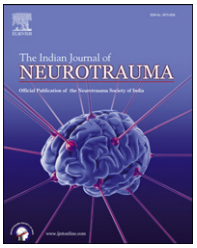

Case Report

Delayed onset post-traumatic bilateral facial nerve paralysis – A rare case report

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ARTICLE INFO

Article history:
Received 31 October 2012
Accepted 26 November 2012
Available online 10 December 2012

Keywords:
Post-traumatic
Bilateral
Facial palsy
Delayed

ABSTRACT

Bilateral facial nerve palsy [FNP] is a rare condition which in most instances is idiopathic. Traumatic brain injury causing bilateral FNP is even more rare and brings with it unique neurosurgical considerations. Delayed presentation of bilateral FNP after mild head injury is socially debilitating as well as neurosurgically puzzling. We present a case of delayed onset bilateral FNP who developed FNP two weeks after mild head injury along with a brief discussion of the peculiar anatomical characteristics and management options of this condition.

1. Introduction

Bilateral FNP with an incidence of 1 per 5,000,000 population[1] is quite rare and represents less than 2% of all FNP. Bell's palsy accounts for only 23% of bilateral facial paralysis while trauma is responsible for less than 5% cases of facial paralysis.[2]

2. Case report

A 17-year-old male presented to us with history of a brief episode of loss of consciousness after a road traffic accident. On examination patient was conscious oriented with a GCS of 15/15. He had no focal neurological deficits. On routine CT brain there was fracture of left temporal bone, there was no intra/extra parenchymal hematoma (Fig. 1). Patient was discharged after few days on oral analgesics. Two weeks later the patient presented with complaints of slurring of speech, on examination he had bilateral LMN facial palsy House/Brackmann grade 3 (Fig. 2). MRI brain revealed multiple small hemorrhagic contusions of right frontoparietal and temporal lobes (Fig. 3). An HRCT of both temporal bones revealed vertical fracture in left mastoid air cell extending up to petrous part of temporal bone and partially involving left facial canal along with another fracture line running through right

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0973-0508/$ – see front matter
http://dx.doi.org/10.1016/j.ijnt.2012.11.008

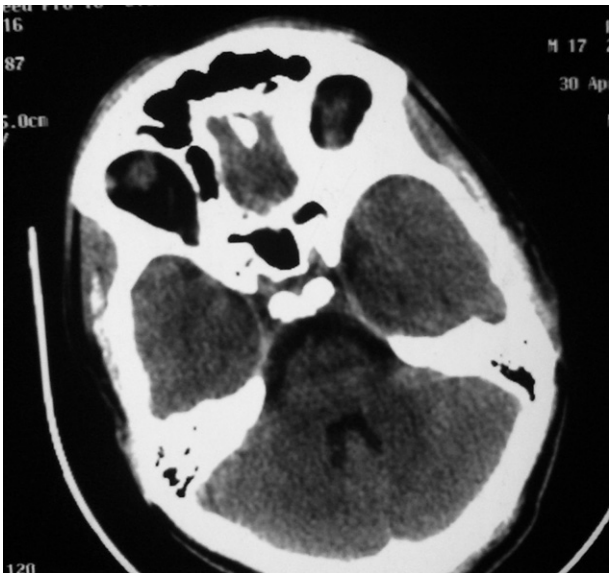

Fig. 1 – Initial plain CT brain.

temporal bone upto internal acoustic meatus (Fig. 4). EMG and NCV were done which showed a decline in cMAP of bilateral facial nerve suggestive of axonal affection. The patient was started on steroids along with physiotherapy to the facial muscles and was discharged after a few days. On last follow up of three weeks weakness had not improved.

3. Discussion

The facial nerve is the motor cranial nerve which is most commonly affected in closed head injuries. Trauma is the second most common identifiable cause of facial nerve paralysis. Immediate FNP after head injury is easily explained but the mechanism of delayed FNP is not clear.

Bilateral FNP warrants evaluation for underlying diseases such as Guillain–Barre syndrome, multiple idiopathic cranial neuropathies, Lyme disease, sarcoidosis, diabetes mellitus, HIV infection, syphilis and infectious mononucleosis.[3]

The delay in onset of facial palsy after head injury varies from 2 to 21 days.

Anatomically the facial nerve occupies only 30–50% of the cross-sectional area of the facial canal with the remainder being occupied by blood vessels with loosely arranged connective tissue. Labyrinthine segment is the most delicate and narrow part of the facial nerve. Degenerative and fibrotic changes after severe injury affect this region more than any part of the facial nerve.

A possible explanation of delayed FNP is bleeding into the facial.

Canal with increasing size of hematoma in the limited non-expanding bony tube compressing the facial nerve and ultimately causing ischemic damage.

Mild pressure would result in neuropraxia or conduction block due to segmental demyelination while higher pressures would cause axonal damage with denervation.

The trauma to the nerve may cause delayed swelling of the nerve leading to compression within its fibrous sheath or epineurium which could also be a cause of delayed FNP.

Severely head injured patients generally have a poor general condition and relative paucity of facial and eyelid movements which makes early detection of bilateral facial nerve injury difficult. Early electrophysiological workup is also often not possible. The severity of head injury may also lead to death in many cases before identification of facial weakness. Bilateral facial nerve palsies associated with head injury usually result from extensive skull base fractures.

Mild head injury causing bilateral facial palsy, as in our case, is uncommon.[4]

Longitudinal fractures of the petrous bone account for 90% of all fractures and causes facial nerve injury in 10–25% of cases[5] whereas transverse fractures of the petrous accounts for 10% of all temporal bone fractures and causes facial nerve

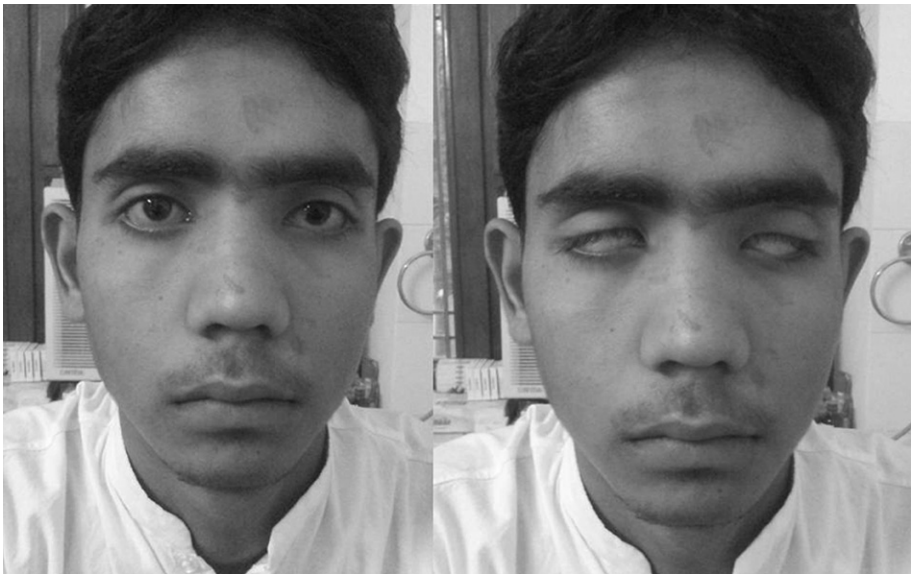

Fig. 2 – Clinical photograph showing bilateral facial palsy.

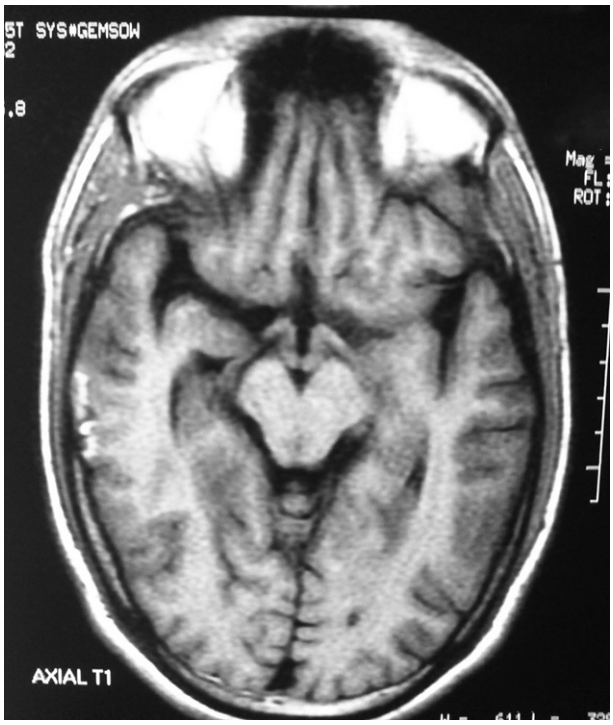

Fig. 3 – MRI brain showing multiple small contusions.

injury in 30–50% of cases.[6] In both cases injury to the facial nerve is usually in the perigeniculate region.

Bilateral traumatic facial nerve palsies usually result from longitudinal petrous fractures across the skull base. The mechanics of fracture dislocation of petrous temporal bone is complicated and involves backward displacement of the petrous apex, coronal splitting of the body of sphenoid leading to mirror image fracture in the opposite temporal bone. Transverse fracture of the petrous bone will not involve both facial nerves.[7] Injury to cranial nerves 5th, 6th, 7th and 8th can also occur as a consequence of the longitudinal fracture and backward displacement of the petrous bone.

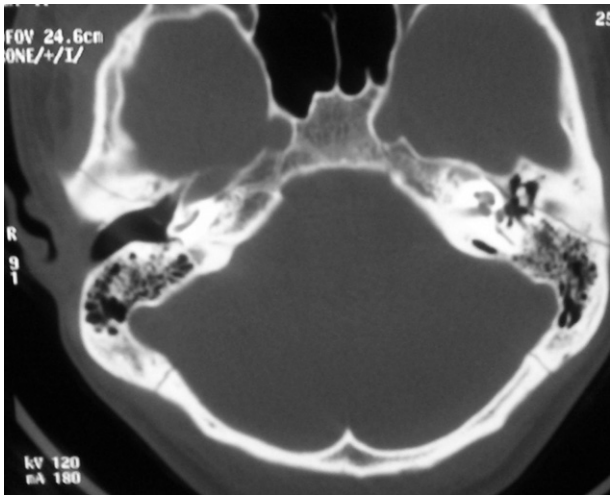

Fig. 4 – CT temporal bones showing bilateral fractures.

Management of traumatic facial nerve disorders is challenging. The type of injury, sudden or delayed-onset, complete or partial paralysis, localization of the injury, and severity of conduction block based on the electrophysiological tests are the main determinants of the prognosis.

High resolution computed tomography (HRCT) is a useful diagnostic tool traumatic FNP, as it can visualize the fracture line and its relationship to the fallopian canal. Immediate onset facial palsy with fracture line running through the fallopian canal is a definite indication for early surgical intervention[8] as is 90–95% loss of function is seen at the very early period on ENoG.

Traumatic injury involving the geniculate ganglion may present with profound retrograde degeneration through the labyrinthine and distal meatal segments of the facial nerve even though the fracture line involved only the horizontal segment only.[9]

Electroneurography performed a few days after trauma is valuable to differentiate which cases will eventually result with HBG-1/II or HBG-VI. But it fails to provide any information about the level of injury between HBG-II and VI this is where EMG is valuable the late follow up period.[10]

Patients with delayed onset facial weakness or incomplete facial weakness should be managed conservatively with steroids and vasodilators. Late surgery may be recommended in cases of non-recovery or within 6 months after trauma.[8]

For incomplete facial nerve paralysis or for delayed onset paralysis associated with a temporal bone fracture, facial nerve testing should be obtained on day 4 after onset. If advanced degeneration has occurred, the nerve should be surgically explored and decompressed.

The surgical approach depends on the site of the injury to the nerve and hearing status. As most of the lesions are perigeniculate middle fossa approach is used to decompress the nerve in the temporal bone in patients with intact hearing whereas if hearing is absent, translabyrinthine approach can also be used.

During exploration the nerve must be fully exposed in order to identify all injured segments, and remove any compression from fracture fragments. The nerve sheath should be incised and any hematomas within the sheath must be carefully evacuated. If complete transection of the nerve is found during exploration, a direct end-to-end anastomosis should be performed if possible. It is important to handle all neural tissue as atraumatically as possible, using microvascular instruments and techniques.

If a direct end-to-end anastomosis creates tension, or when segments of the nerve are missing or severely damaged, interpositional grafts from the greater auricular, medial antebrachial cutaneous, or sural nerve should be used.

4. Conclusion

FNP occurring after trauma is likely to be missed if associated with severe traumatic brain injury. In cases where it is diagnosed, whether unilateral or bilateral it warrants a thorough evaluation including HRCT of temporal bone and electrophysiological testing. Apart from definite indications of surgery such as immediate onset FNP with fracture line

running through fallopian canal or bony fragment impinging on nerve, gradual improvement can be expected in most cases with conservative management.

Conflicts of interest

All authors have none to declare.

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