CASE REPORT

Post-traumatic Immediate onset Facial Palsy-delayed Exploration with Complete Recovery

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ABSTRACT

Post-traumatic immediate onset facial palsy is well-documented clinical entity. However, it is usually described in transverse fracture of temporal bone requiring early surgical intervention. Here we report a case of immediate onset facial palsy following longitudinal fracture of temporal bone in a 50-year-old male patient who presented to us 6 months after the closed head trauma. Electroneurography and electromyography showed complete nerve degeneration. Facial palsy was completely recovered after delayed reconstruction almost 7 months after the onset. Here longitudinal fracture of temporal bone caused pressure over the incus which in turn had pressed over the dehiscent facial nerve in its tympanic segment up to the second genu thereby resulted in immediate onset facial palsy.

Keywords: Post-traumatic immediate onset facial palsy, Temporal bone fracture, Delayed exploration, Facial nerve decompression.


Source of support: Nil
Conflict of interest: None declared

CASE REPORT

A 50-year-old male patient came with complaints of 6 months history of immediate onset right-sided facial palsy following head injury due to road traffic accident 6 months back. Patient had developed facial deviation on the left side and had inability to close his right eye (Fig. 1A). Patient received IV corticosteroids for 15 days followed by oral steroids for 6 weeks for present illness but only resulted in partial relief (Fig. 1B).

Following specific investigations were done:

- Pure tone audiogram
- HRCT temporal bone and MRI brain with contrast
- Electromyography (EMG) nerve conduction study for facial nerve.

Pure tone audiogram revealed moderately severe sensorineural loss at high frequency both the ears. However, hearing was normal in speech frequencies. High resolution CT scan of temporal bone revealed longitudinal fracture involving the mastoid segment of right temporal bone with medial extension of the fracture line involving the petrous temporal bone passing just above the internal auditory canal and anterior wall of external auditory canal (Figs 2A and B). MRI brain revealed multifocal parenchymal contusions with gliotic scars in the anterior frontal cortex, the gyrus recti. Electromyography and nerve conduction study of facial nerve at the time of presentation revealed absent facial conduction on right side suggestive of complete right sided lower motor neuron facial palsy.

Subsequently, patient received intravenous corticosteroids for 15 days followed by oral steroids for 6 weeks along with eye care, facial exercises (active as well as passive).

EMG then again repeated after course of steroids revealed evidence of right facial neuropathy, severe and partial axon degenerative type however, there was evidence of ongoing reinnervation in the muscles sampled. However, the recovery

Figs 1A and B: (A) Clinical photograph of patient showing right facial palsy grade V (House and Brackmann classification) patient had deviation of angle of mouth toward left side, (B) clinical photograph of patient showing right facial palsy depicting loss of wrinkling of forehead on right side
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Fig. 2A: High resolution CT scan temporal bone showing longitudinal fracture of temporal bone, depicted by an arrow pointing toward fracture line (plain scan, transverse section, slice thickness 0.6 mm)

Fig. 2B: High resolution CT scan temporal bone showing longitudinal fracture of temporal bone, depicted by an arrow pointing toward fracture line (plain scan, coronal section, slice thickness 0.6 mm)

was only partial (Figs 3A and B). Hence, the patient was worked up for facial nerve decompression.

On posterior tympanotomy on right side, it was found that the facial nerve was dehiscent in its tympanic segment genu, just below the incus extending up to second genu (Fig. 4).

Fracture line was seen passing through bony canal longitudinally, extending medially causing pressure over the incus and malleus.

Dehiscent facial nerve was edematous. Decompression of the facial nerve is performed in all its segments (labyrinthine, tympanic and mastoid) and then it was lifted out of its canal followed by widening of fallopian canal and replacement of nerve back in the widened canal was carried out.

Immediately following the surgery, facial palsy was completely recovered and recovery was uneventful (Figs 5A to C).

DISCUSSION

Among the various etiological factors, trauma is the second most common cause of facial palsy. Temporal bone fractures are extremely common with head injuries. Fractures involving the temporal bone are classified depending on the relationship of the fracture line to the long axis of the petrous part of the temporal bone as longitudinal, transverse and mixed. Most (80%) temporal bone fractures are longitudinal and usually result from blows to the temporal or parietal areas. The fracture line usually runs anterior to the otic capsule and involves the external and middle ears resulting in bleeding from the ear with conductive hearing loss due to ossicular disruption (Fig. 6). Since, the fracture does not involve the otic capsule, sensorineural hearing loss is not seen. The facial nerve canal is usually spared and facial palsy occurring in longitudinal fractures is usually delayed in onset and is due to nerve edema in most of the cases. Such cases can be conserved with medical line of management which includes steroids will result in improvement of the function of the facial nerve. Transverse fractures usually result from frontal or occipital blows and accounts for approximately 20% of temporal bone fractures. The fracture line passes through the otic capsule thus damaging the inner ear. A pure transverse fracture can result in a hemotympanum and it is characterized by sensorineural hearing loss, tinnitus, nausea, vomiting, vertigo and facial palsy on the affected side. Fifty percent of these patients develop facial palsy which is immediate in onset. In severe head injuries there may be combination of
the longitudinal and transverse fracture with a loose fragment of bone, which can result in facial palsy. The facial palsy seen in these mixed (comminuted) fractures of the temporal bone is usually immediate in onset. Thus immediate onset facial palsy is more commonly seen in transverse fracture and mixed fractures of temporal bone. For immediate onset facial palsy early surgical exploration is recommended. It usually indicates tearing of the facial nerve, impaling of the nerve by bone, or entrapment in a fracture line. Facial nerve decompression is indicated if 90 to 95% loss of function is seen at the very early period on ENoG or if there is axonal degeneration on EMG lately with no sign of recovery.\textsuperscript{3,4} As per literature survey, there is no consensus regarding the timing of surgical intervention and the role of electrophysiological testing.\textsuperscript{3-5} The exploration of the facial nerve is recommended preferably within 72 hours as after 72 hours the process of Wallerian degeneration sets in. According to McCabe (1972), if 72 hours have passed, the optimum time for repair of the facial nerve is on the 21st day as the nerve cell body is maximally capable of passing the axoplasmic filaments across the neuronal gap.\textsuperscript{6}

Immediate onset facial palsy in the longitudinal fracture of temporal bone is a rare clinical entity. In the present case, the fracture line of longitudinal fracture of temporal bone caused pressure over ossicles thereby lead pressure over dehiscent facial nerve in its tympanic segment thereby caused immediate onset facial palsy. In the present case, although the patient presented almost 6 months after the trauma and underwent decompression almost 7 months after the trauma the recovery was complete following delayed decompression.

CONCLUSION
Although, immediate onset facial palsy is more commonly seen in transverse fractures of temporal bone, it can occur in patients with purely longitudinal fracture of temporal bone. So far very few cases of immediate onset facial palsy due to longitudinal fracture of temporal bone have been described in the literature; the present case was probably the first case of its kind of immediate onset facial palsy due to compression of dehiscent nerve in purely longitudinal fractures of temporal bone. Although, it is thought that late surgical intervention is unlikely to yield further improvement in the facial nerve function experience with this case suggests that surgical exploration of the facial nerve is indicated at any time, as it may be beneficial even in very old injuries. Although medical line of management is recommended for facial palsy due to longitudinal fracture of temporal bone, the decision for surgical exploration should not be postponed further if clinical and electrophysiological recovery is incomplete. Since, no consensus exists today on the management of immediate-onset post-traumatic facial nerve paralysis and controversy
surrounds the timing of surgical intervention as well as the role of electrophysiological testing, each case needs to be assessed individually for appropriate surgical intervention.

ACKNOWLEDGMENT

Authors would like to solicit Dr BR Dhareshwar, Medical Director, Jagjivan Ram Hospital for allowing us to publish this case report.

REFERENCES


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