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Traumatic bilateral facial nerve palsy

Mridula V Rao[1]*, Harish Chander Goel[2], Sitaram Bharath[3]

1. Senior consultant in ENT, Primary Health centre, Directorate of health services, Sanquelim, 403505, Goa, India
2. Professor and HOD, Department of ENT and Head and Neck Surgery, Goa Medical College, Goa university, Goa 403201, India
3. Senior resident , Department of Radiology, Goa Medical College, Goa university, Goa 403201, India

***Corresponding author:** Primary Health center, Directorate of health services, Sanquelim 403505, Goa, India; E-mail: drmridular@gmail.com

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ABSTRACT

Facial nerve palsy complicates 7% of the temporal bone fractures. However in majority of the cases post traumatic facial nerve paralysis is unilateral. It is rare to develop bilateral facial nerve palsy after trauma. A 30 year old female sustained head injury following a motor vehicular collision. Glasgow coma score was 15/15. Computed tomography of the brain showed subdural hematoma in left frontotemporoparietal region with subarachnoid hemorrhage. Patient was admitted under neurosurgery. On the 4th day of injury, the patient developed inability to close both eyes and lack of facial expression. The high resolution computed tomography scan of the temporal bone showed bilateral fracture of the temporal bone not involving the fallopian canal. Based on high resolution computed tomography scan findings and delayed onset of facial paralysis patient was treated with short course of prednisolone and facial physiotherapy without surgical intervention reported on both sides. Patient showed significant recovery in facial nerve activity.

Keywords: Facial palsy; Trauma; Bilateral

1. INTRODUCTION

Facial nerve palsy may represent an important complication of head trauma. Facial palsy is most commonly unilateral and idiopathic. Bilateral facial palsy is an extremely rare event reported, with an incidence of one case per 5 million population, as compared to unilateral facial palsy with incidence of 1000-1250 cases per 5 million population per year

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Figure 1

Showing flattening of bilateral nasolabial folds, loss of wrinkling of forehead and inability to close both eyes without facial asymmetry

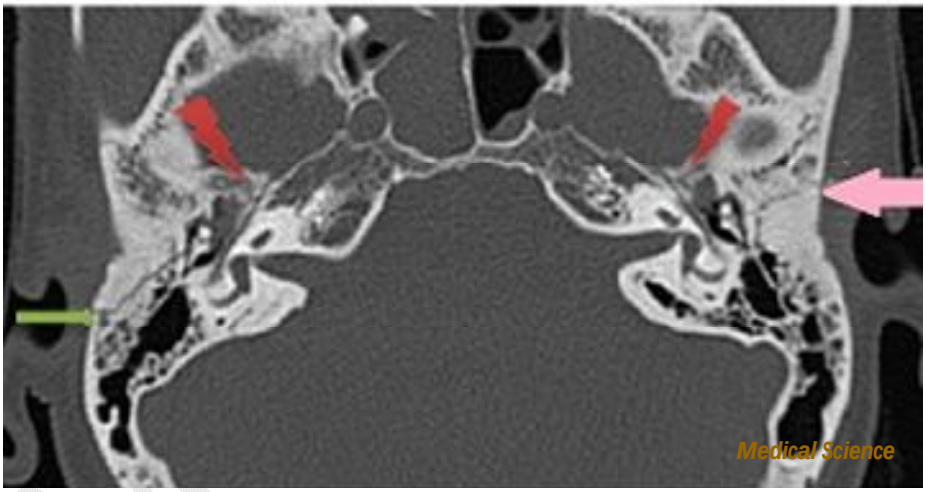

Figure 2

High resolution computed tomography scan showing longitudinal fracture of temporal bone on the right side and transverse fracture of the temporal bone on the left side shown with thin and thick arrows respectively. Red pointers indicating fallopian canal on both sides

(George et al., 1991; Sherwin et al., 1987; Robert Mills et al., 2008). Motor vehicular accidents are the most common cause of temporal bone fractures. Falls, bicycle accidents, firearms and physical assaults are other reported causes. Facial palsy may lead to lack of facial expression which has an immense psychological impact. Due to associated head injury, requiring more attention for treatment patient and surgeon may not be conscious of injury to facial nerve and diagnosis is further difficult in case of bilateral facial involvement due to lack of asymmetry. However suspicion and early detection plays a vital role in management of patient. We report rare case of bilateral facial palsy in a young female due to head trauma.

2. CASE REPORT

A 30 year old female had motor vehicular accident resulting from fall from a two wheeler. Patient was conscious and oriented with injury to both ears and nose. Computed tomography scan of brain showed subdural hematoma 3.8mm in size in the left frontotemporoprietal region with subarachnoid hemorrhage with fracture of right parietal and mastoid bone. Patient was admitted in neurosurgery ward. On 4 day of admission patient noticed dribbling of saliva from both sides of mouth and was unable to close her both eyes, without facial asymmetry (Figure 1). On examination patient was found to be having bilateral facial palsy. Pure tone audiometry showed bilateral moderate conductive hearing loss.

High resolution computed tomography was ordered which showed longitudinal fracture of temporal bone on right side and transverse fracture of temporal bone on left side without involving the fallopian canal as shown in (Figure 2). There wasossicular chain discontinuity on either side.

Electroneuronography study showed bilateral low amplitude CMAP suggestive of bilateral axon-loss facial neuropathy. Patient was treated conservatively with a of course of prednisolone for 10 days along with multivitamins and facial physiotherapy. Review of patient after 4 months showed marked improvement in facial function on both sides.

3. DISCUSSION

Trauma is responsible for less than <5% cases of facial paralysis (Steenerson, 1987). The most common causes of bilateral facial palsy are GullianBarre syndrome, sarcoidosis, bacterial meningitis, leukaemia, syphilis, leprosy, Lyme's

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disease, Moebius syndrome, infectious mononucleosis, skull fracture. Bilateral facial palsy is rarely idiopathic. Bilateral facial palsy is a rare clinical entity (Price et al., 2002). Head trauma is a deceleration injury. The facial nerve is tethered in the perigeniculate ganglion region by the greater superficial petrosal nerve. This tethering and sudden deceleration creates a shearing force on the facial nerve resulting in intraneural contusion, edema and hemmorhage. If shearing force is great enough nerve will transect. Distal to pyramidal eminence is the second most common site for facial nerve injury. The more common type of injury is immediate onset, which usually results from direct laceration or contusion of the nerve at the site of temporal bone fracture. Delayed onset palsy is most commonly caused by edema, external compression by hematoma fluid, or arterial or venous thrombosis and is not usually noticed until 4-5 days after head injury. This results in partial or complete degeneration of the nerve (Li et al., 2004).

Longitudinal fractures comprise of 70-90% of the temporal bone fractures. It follows the long axis of the petrous bone. The mechanism of fracture starts with a blow to the lateral (temporoparietal) skull. Between 14-30% of longitudinal fractures are bilateral. The classic triad of physical findings in longitudinal temporal bone fractures is bloody otorrhea, external auditory canal bony step-off and tympanic membrane disruption that is often associated with conductive hearing loss (Cannon et al., 1983; Tos, 1973). Transverse fractures cross the perpendicular axis of the petrous pyramid and comprise 10-30% of the temporal bone fractures. These fractures are associated with a higher rate of facial nerve palsy, vertigo, sensorineural hearing loss (Cannon et al., 1983; Tos, 1973).

A diagnosis of facial palsy should be suspected in all patients with head injury to enable early diagnosis and intervention. Further bilateral delayed onset post traumatic facial palsy is rare compared to bilateral immediate onset facial palsy hence making the diagnosis even more difficult. A detailed ENT examination along with high resolution computed tomography scan with 1-1.5mm cuts should be carried out. Facial nerve injury is usually distal to geniculate ganglion in longitudinal facture and proximal to geniculate ganglion in transverse fractures (Thanos Bibas et al., 2008). High resolution computed tomography can accurately determine the fracture extent, ossicular continuity and integrity of fallopian canal. In our case the patient has conductive hearing loss hence it was necessary to rule out ossicular disruption. Electrophysiological tests evaluate the degree of dysfunction of facial nerve and provide the information on the potential for recovery. Electrophysiological tests may be carried out in patients with facial palsy, but they are of limited use in case of bilateral facial palsy due to lack of comparison between the two sides.

Temporal bone fractures involving the fallopian canal will usually present with immediate onset facial palsy. However injuries without involving fallopian canal may present later on as in our case. Therefore a continuous observation for delayed facial palsy should be made for atleast 10-14 days. The early diagnosis of facial palsy is vital to start early treatment for complete recovery of facial palsy.

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