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Conservative Management Of Post Traumatic Delayed Onset Facial Nerve Palsy – A Case Series

Dr. Sangeetha M[1], Dr. Ruta Shanmugam[2], Dr. Shanmugam V.U[3], Dr. Balaji Swaminathan[4], Dr. Choudhary C.S[5], Dr. Prakash P.G[6]

[1]Post Graduate, [2]Professor and Head, [3,4]Professor, [5]Assistant Professor, [6]Senior Resident, Department of ENT, Rajah Muthiah Medical College, Annamalai University, Chidambaram

***Corresponding Author:**
Dr. Shanmugam V.U.
Professor, Department of ENT, Rajah Muthiah Medical College, Annamalai University, Chidambaram

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Abstract

Background: Facial nerve palsy is an infrequent complication after head injury with or without temporal bone fractures and it can be either immediate or delayed in onset. The incidence of delayed onset facial nerve palsy is 2.2% after head injury. The Delayed onset facial palsy is the term used for facial palsy occurring 48 hours after head injury. The probable causes could be intraneural contusion, edema, hemorrhage, and consequent ischemia or presence of granulation tissue within the fallopian canal that eventually leads to facial nerve palsy.

Methodology: Data collected by History taking, , Ear, Nose, Throat examination, Pure tone audiometry, computed tomography, Electroneuronography evaluation for Delayed onset Facial nerve palsy after head injury were analyzed.

Results: In our study, there were 2 patients with delayed onset facial palsy after head injury. 1 patient with transverse fracture of temporal bone developed facial palsy in the 1st week after trauma. Another patient with longitudinal fracture of temporal bone developed facial palsy in the 2nd week after injury. Facial palsy recovery started after 7 days of steroid administration in both the patients.

Conclusion: Delayed onset facial palsy mainly occurred within 2 weeks after head injury, and they achieved good facial nerve recovery after conservative treatment.

Keywords: Facial nerve palsy, Delayed onset, Electroneuronography

Introduction

Facial nerve palsy is a rare complication after head injury with or without temporal bone fractures and it can be either immediate or delayed in onset. The incidence of delayed onset facial nerve palsy was 2.2% after head injury(1). The Delayed onset facial palsy is the term used for facial palsy occurring 48 hours after head injury. Facial nerve after leaving the pons, travels through the internal auditory canal and begins its tortuous journey within the fallopian canal. The narrowest part of the canal lies in the labyrinthine segment, especially in the perigeniculate area, which makes the nerve more vulnerable to swelling and compression. Head injury creates a sudden acceleration/deceleration effect which produces a shearing force that injures the neural structure. This results in intraneural contusion, edema, hemorrhage, and consequent ischemia that eventually leads to facial nerve palsy. Facial nerve palsy is clinically evaluated by House Brackmann (HB) scale(2). We report the clinical features and outcomes of delayed onset facial palsy after head injury, treated conservatively.

Case -1:

We present a case of 17 year old male with history of head injury and bilateral ear injury following road traffic accident. ENT examination revealed blood

clots in the external auditory canal and partially visualized tympanic membrane in both the ears. Pure tone audiometry showed Bilateral Mild High Frequency Sensorineural Hearing loss. CT Brain with temporal bone showed Transverse fracture involving the temporal bone and involving the mastoid air cells.

1 week later after head injury, the patient developed House Brackmann Grade 4 Facial palsy on the left side. Oral Prednisolone was started immediately after onset of delayed facial palsy for 2 weeks and clinically resolution was evident 1 week after onset of treatment. At the end of 2 weeks after onset of facial palsy, with conservative management patient's facial weakness resolved to House Brackmann Grade 2. Electroneuronography test was performed at 1 week after onset of facial palsy which revealed facial nerve degeneration of 52% and it improved to <35% at the end of 2 months.

Fig 1: CT Temporal bone showing Transverse fracture of Temporal bone & Pure Tone Audiometry showing Mild High Frequency Sensorineural Hearing loss

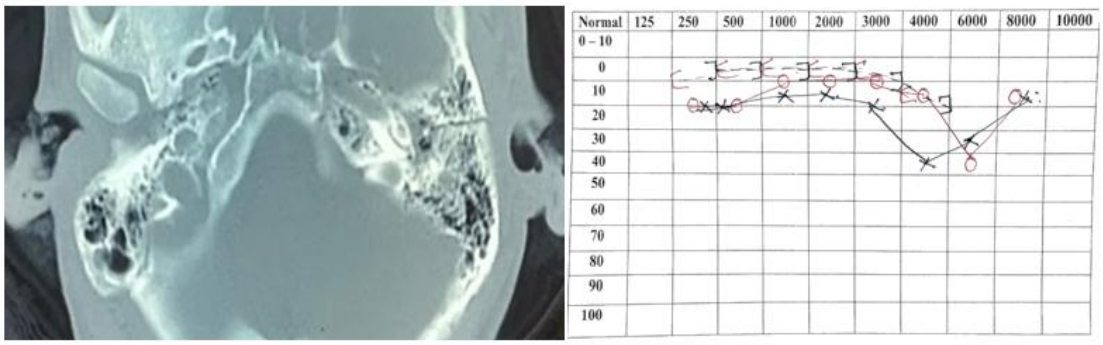


Fig 2: Electroneuronography test showing reduced compound muscle action potential with facial nerve degeneration of 52%

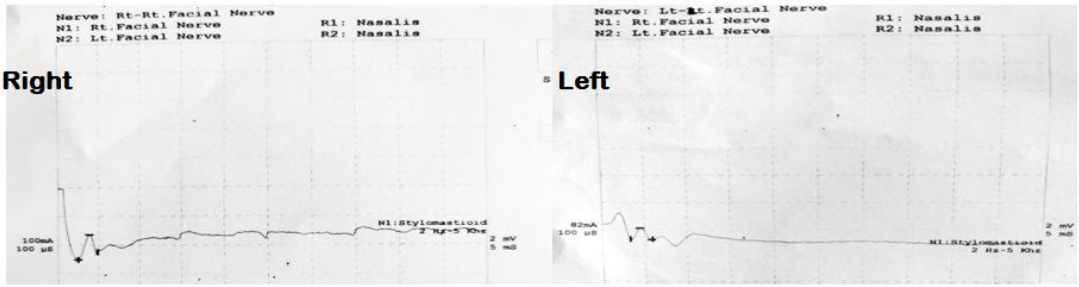
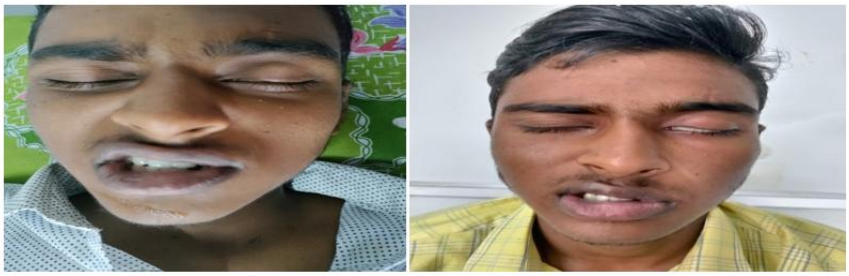


Fig 3: Clinical picture showing pre treatment and post treatment of facial palsy



ONSET OF FACIAL PALSY 1 WEEK AFTER HEAD INJURY

FACIAL PALSY RECOVERY AFTER 1 WEEK OF STEROID THERAPY

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Case -2:

A 23 year old male presented with history of head injury following road traffic accident. ENT examination revealed blood clots in the external auditory canal and Tympanic membrane was not visualized. Pure Tone Audiometry showed normal hearing status. CT Brain with temporal bone showed longitudinal fracture involving the Temporal bone and involving the mastoid air cells.

2 weeks later after head injury, he presented to ENT outpatient department with House Brackmann Grade 3 Facial palsy on the left side. Oral Prednisolone was started immediately after delayed onset facial palsy and continued for 2 weeks. Clinical resolution of facial weakness was evident after 1 week of treatment. At the end of 2 weeks after conservative management, facial palsy resolved to House Brackmann Grade 2 facial palsy. Electroneuronography test was performed at 1 week after onset of facial palsy which revealed facial nerve degeneration of 54% and it improved to <35% at the end of 2 months.

Fig 4: CT Temporal bone showing longitudinal fracture of Temporal bone

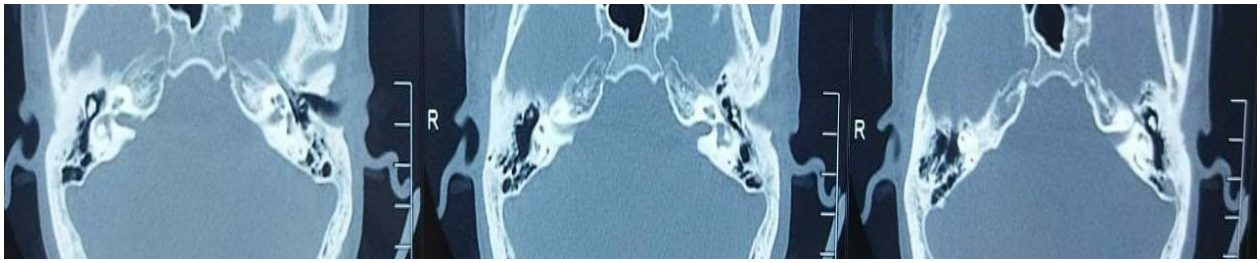
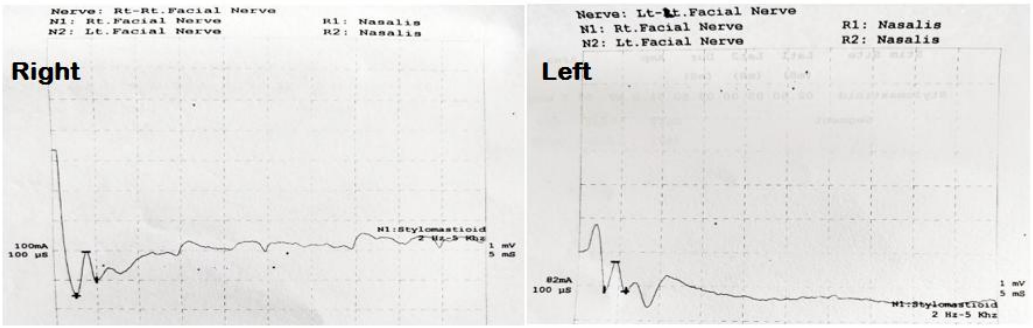


Fig 5: Electroneuronography test showing reduced compound muscle action potential with facial nerve degeneration of 54%



Result:

Delayed onset facial palsy affected the left side in both the patients and they were managed conservatively with oral Prednisolone for 2 weeks. After steroid therapy, both the patients showed clinical improvement from House Brackmann scale – Grade 4 to Grade 2 at the end of 2 months in case 1 and from House Brackmann scale – Grade 3 to Grade 2 at the end of 2 months in case 2. Electroneuronography test report showed reduced CMAP (Compound Muscle Action Potential) amplitude with facial nerve degeneration of less than 60% in both patients. When Electroneuronography study was repeated at the end of 2 months, the facial nerve degeneration was less than 35%. The report of these 2 patients demonstrates return of near normal facial nerve function upon conservative management during the follow up.

Discussion:

Immediate onset facial nerve palsy following head injury results from transection of the nerve, while delayed onset facial palsy is a result of haemorrhage into the facial canal - a space in which 30-50% is usually occupied by the facial nerve with

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encompassing blood vessels and connective tissue [5]. An expanding hematoma can compress the nerve and cause ischemic damage. The extent of the damage depends on the degree of pressure, mild pressure may cause neuropraxia or conduction block due to segmental demyelination while higher pressures could cause permanent axonal damage with subsequent denervation [2]. Other theories of pathogenesis are nerve oedema, delayed arterial spasm or compression of the facial nerve by bony fragments or granulation tissue in the fallopian canal that compresses the facial nerve [3]. The majority of temporal bone fractures are longitudinal while only 10% are transverse, 30-50% of these transverse fractures cause facial palsy due to direct nerve trauma and are more likely to be immediate onset; 10-25% of longitudinal fractures, will present with facial nerve palsy, out of which 88% will be of delayed onset facial nerve palsy. [4]

There is an association between Temporal bone fracture and facial nerve palsy. In our series, Delayed onset facial nerve palsy occurred at least 48 hours or longer after trauma which was consistent with the present literature. Ear bleeding and Temporal bone fracture are common clinical features in delayed onset facial palsy after head injury, therefore we should not ignore the possibility of delayed onset facial palsy if patients have both ear bleeding and temporal bone fracture after head injury. Recovery of Facial nerve was evaluated using House Brackmann (HB) scale and Electroneuronography study. In our series, House Brackmann scale Grade 3 and 4 patients recovered with conservative management. Clinical improvement in facial nerve function correlated with Electroneuronography study. Poor scores (i.e. > 90%) in Electroneuronography study in the 1st week is an indicator for early surgical intervention of the facial nerve.

Conclusion:

The Onset of facial nerve palsy, Degree of facial nerve palsy and Electroneuronography test forms the major predictors in the outcomes & management of Post-traumatic delayed onset Facial nerve palsy.

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