

Temporal Bone Fracture Leading to Facial Palsy - A Case Report

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INTRODUCTION

Facial nerve paralysis is a disorder causing noticeable disfigurement and emotional distress.¹⁻⁴ Facial palsy may affect normal daily functions such as eating and drinking. It may disrupt the protective function of the eye lids. Facial nerve palsy can occur from a variety of causes. It can be idiopathic or caused by trauma (motor vehicle accidents), iatrogenic (surgery), infections (Bell's palsy), radiation therapy and tumours.^{2,3,5-7} Temporal bone fractures are not rare injuries in patients having head trauma. 7 to 10% of temporal bone fractures are caused by road traffic accidents which lead to facial nerve palsy.^{4,7} A detailed history, clinical, otological and neurological examination, audiological evaluation and thin sections high resolution CT examination are important for a proper diagnosis and early intervention. Management of facial nerve paralysis with facial nerve decompression is a surgical challenge.^{8,9} Here, we report a case of lower motor neuron type of facial palsy in temporal bone trauma in an 18-yr. old male who improved with early surgical intervention.

PRESENTATION OF CASE

An 18-yr.-old male patient from a rural district came to the Department of Otorhinolaryngology OPD with the complaint of inability to close left eye, deviation of angle of mouth to right since a week. The patient gave history of alleged self-fall while riding a bike and head injury 1 week back. On examination, left lower motor neuron type of facial paralysis- House Brackmann grade 5-6 was present. Patient did not give history of loss of consciousness, nausea and vomiting, bleeding from the ear, diminished hearing following head injury. Otoscopic examination of left ear showed hemotympanum. Right ear was normal. Schirmer's test showed reduced lacrimation on left side. Patient gave history of hospitalisation for 5 days in a nearby hospital following trauma and was treated with oral corticosteroids and antibiotics. He had been prescribed oral prednisolone (1 mg/Kg).

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Figure 1.
Pre-Operative Picture Showing
Incomplete Closure of Left Eye

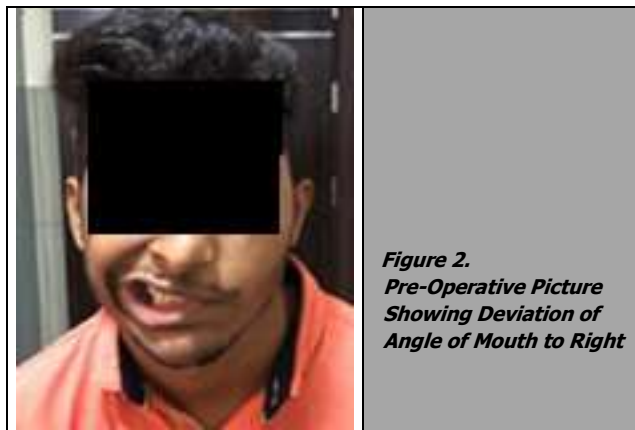


Figure 2.
Pre-Operative Picture Showing Deviation of Angle of Mouth to Right

CLINICAL DIAGNOSIS

Traumatic lower motor neuron type of facial palsy.

DISCUSSION OF MANAGEMENT

Pure tone audiometry showed left mild conductive hearing loss. HRCT scan of temporal bones was performed employing 0.625 mm sections. HRCT scan showed longitudinal fracture of left petro-mastoid temporal bone extending into proximal tympanic portion of facial nerve canal, anterior wall of external auditory canal, tegmen tympani, carotid canal and petrous apex with mild subluxation of incudo-malleal joint. Moderate left organised hemotympanum and haemomastoid was present. Nerve conduction study showed left facial nerve axonal neuropathy with Electromyography showing neurogenic changes.

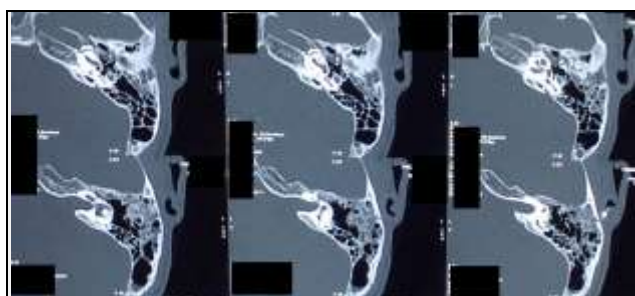


Figure 3. HRCT Scan of Temporal Bone Showing Longitudinal Fracture Extending into Proximal Tympanic Portion of Facial Nerve Canal, Anterior Wall of External Auditory Canal, Tegmen Tympani, Carotid Canal and Petrous Apex with Mild Subluxation of Incudo-Malleal Joint

Patient underwent left facial nerve decompression under General Anaesthesia. Through post aural approach-temporalis fascia graft was harvested and through transcanal route, tympanotomy was done. Following atticotomy, incudostapedia joint was visualised, incus disarticulated and removed to expose the tympanic segment of facial nerve, which was found to be dehiscent, vascular and oedematous. Facial nerve was exposed from first genu to second genu. Facial nerve sheath was incised with a tenotome, small piece of bony spicule impinging on first

genu was removed. Incudostapediopexy using sculpted incus and attic reconstruction using conchal cartilage was done. Post operatively, grade 6 facial palsy recovered to grade 2. Patient was treated with post-operative antibiotics and oral corticosteroids.



Figure 4.
Post-Operative Recovery of Eye Closure

DISCUSSION

Facial nerve paralysis (FNP) can be due to multiple aetiology such as iatrogenic, traumatic, infections and tumours involving the nerve. Seven to 10% of cases of FNP are caused by temporal bone fractures (TBF). TBF are classified into longitudinal, transverse and mixed depending on the site of fracture in relation to the axis of petrous pyramid. They have also been classified as otic capsule sparing or otic capsule violating fractures, labyrinthine and extra labyrinthine fractures, respectively.^{4,5} FNP associated with TBF can be immediate or delayed in onset based on the duration.

According to Ulrich et al., the geniculate ganglion is the most common site for FNP in longitudinal TBF.¹⁰ The fracture line passes superior to the external auditory canal entering the middle ear and along the facial nerve in the perigeniculate area.^{10,11,12,13} The mechanism of FNP may be due to traction by displaced bony segments or impingement by bony spicules in the perigeniculate area.^{4,13} Transverse fractures frequently involve the labyrinth compared to longitudinal fractures. A detailed clinical examination, audiological evaluation, radiological imaging and electrodiagnostic testing methods play an important role in assessing the prognosis of palsy. It is important to assess the site of lesion, status of tympanic membrane and middle ear, hearing level to help in the selection of surgical approach to improve prognosis.^{4-6,9,14}

High resolution computed tomography (HRCT) of the temporal bone is the investigation of choice for patients affected by TBF. It helps in preoperative surgical planning and better patient counselling.^{4-6,9,11} The entire course of facial nerve must be studied in detail on HRCT temporal bone. Fracture line, fracture fragments, bony spicules causing compression, evidence of localized expansion suggestive of intraneural haematoma or oedema must be looked for on HRCT temporal bone. A careful evaluation of ossicular chain status for various types of ossicular

dislocation should be done in association with TBF, because this may lead to conductive hearing loss.¹⁵

Electro-diagnostic tests are based on the principles of electric stimulation of nerve to evoke electromyography response. They help in evaluating the condition of the nerve and establishing the degree of dysfunction. Electromyography and electroneuronography are more commonly used for FNP. Electroneuronography can estimate the degenerative status of the nerve. It is useful between 4 and 21 days after onset of paralysis. Since it takes 3 days for Wallerian degeneration to occur, ENoG is not performed until the fourth day. Fisch advised that ENoG in traumatic FNP causing degeneration >90% within six days of onset of complete FNP needs immediate decompression.^{6,9,11,12}

Facial nerve decompression is a matter of controversy in regard to the duration of onset of palsy and time of surgery. Many studies have suggested that early decompression is better providing early expansion of the nerve by relieving edema. It helps to remove bony spicules impinging on the nerve and drain the blood collected from the fallopian canal. Delayed decompression leads to formation of fibrotic bands following pathological repair. Impinging bony fragments can affect nerve conduction. Delay in surgical decompression can compromise blood supply causing degeneration and shrinkage of nerve. It can lead to formation of scar, neuroma and atrophy of peripheral structures.^{1,7,9,12,14}

Many surgical approaches are available for facial nerve decompression. The route of surgical approach is determined by the portion of the facial nerve affected and the amount of residual hearing. Various surgical approaches include the translabyrinthine, transcanal, transmastoid or middle fossa approaches. In otic capsule violating fractures with no serviceable hearing, the translabyrinthine approach is preferred. In otic capsule sparing fractures, the transmastoid - supralabyrinthine and middle cranial fossa approaches are appropriate based on the site of lesion and mastoid pneumatization. In well pneumatized mastoids with nerve involvement limited to the perigeniculate area, transmastoid - supralabyrinthine access provides enough exposure, whereas a fracture involving the labyrinthine segment of facial nerve can be better accessed via middle fossa approach.^{1,5-7,9,11}

It can be concluded that early surgical decompression provides better results in terms of facial nerve function improvement, possibly because it enables early expansion of the nerve by removing impinged bony particles, avoiding compression from dislodged ossicles and by reducing traction injury from displaced bony segments. In short, timely surgical treatment with imaging guidance and facial nerve electrophysiology in cases of post traumatic facial palsy will result in good outcomes.

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