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A RARE CASE OF CONCOMITANT ABDUCENS AND CONTRALATERAL LOWER MOTOR NEURON FACIAL NERVE PALSIES FOLLOWING BLUNT HEAD TRAUMA ASSOCIATED WITH TEMPORAL BONE FRACTURE

Ophthalmology

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ABSTRACT

This case report describes a case of concomitant abducens and contralateral lower motor neuron facial nerve palsies following blunt head trauma associated with left temporal bone fracture. A 11year old boy was brought to the emergency department with alleged history of fall from swing set. Detailed ocular examination and assessment of function of all the twelve cranial nerves of the patient revealed left oculomotor nerve paresis, left abducens nerve palsy and right sided lower motor neuron (LMN) facial nerve palsy. The patient was run through various neuroimaging investigations which showed longitudinal fracture of the left temporal bone. The patient was treated aggressively with systemic and oral steroids. On discharge, the left oculomotor nerve paresis had healed completely with no improvement in the other two affected cranial nerves. During one month follow up, the abducens and facial nerve palsies still persisted.

KEYWORDS

Contralateral, Facial nerve palsy, Temporal bone fracture, Longitudinal fracture

INTRODUCTION

Temporal bones are the thickest skull base bones and constitute a large portion of the lateral wall and base of the skull. Various cranial nerves pass through the temporal bones and its fracture can have serious consequences. Fracture of these bones can result in damage to middle or inner ear structures, facial nerve palsy and cerebrospinal fluid leak.[1] Although facial nerve palsy is common in case of an ipsilateral temporal bone fracture, contralateral LMN facial palsy in concomitance with abducens nerve palsy has rarely been reported.

CASE REPORT

A 11year old boy was brought to the emergency department of Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai with alleged history of fall from swing set.

His chief complaints were diplopia on distance fixation in primary gaze which worsened on left gaze, left ear bleed and nasal bleed, contusion on the left temporal region of the head and a minor bruise on the right side of the forehead and right periorbital region. He gave no history of loss of consciousness, altered sensorium, convulsions and vomiting at the time of incident. Interestingly, he had no complaints of hearing loss.

He was conscious, cooperative, well oriented with time, place and person and hemodynamically stable. Head posture was normal except for slight head tilt to the left on attempted left gaze with no facial asymmetry [Figure 1].

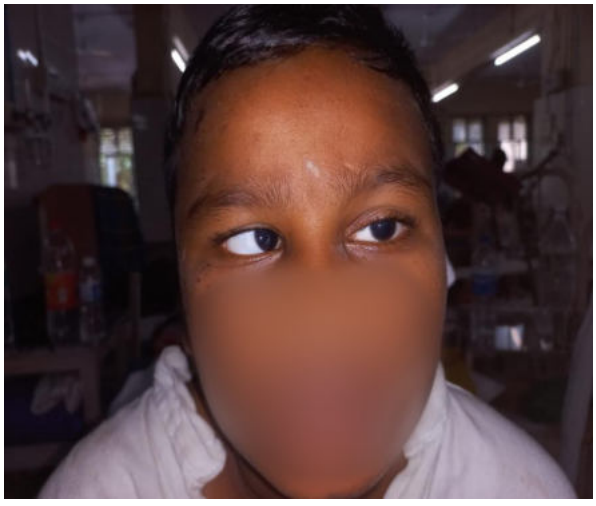

Figure 1: Head Tilt On Attempted Left Gaze

Extraocular movements of the right eye were full and free with overacting medial rectus muscle and there was complete absence of abduction movement and moderately limited elevation and depression movement of the left eye [Figure 2].

Figure 2: Extraocular Movements Showing Complete Absence Of Abduction Movement And Moderately Limited Elevation And Depression Movement Of The Left Eye

Visual acuity was 6/6 for distance and N6 for near in both eyes using Snellen vision chart. Color perception was normal in both eyes and was tested using pseudoisochromatic Ishihara test with thirty eight plates. Diplopia charting showed horizontal uncrossed diplopia with maximum separation of images on levoversion.

Slit lamp biomicroscopy was used to examine the anterior segment of both eyes. The right eye had lagophthalmos while the left eye had moderate ptosis and mydriatic pupil of four millimeter size which showed sluggish response to direct and consensual light [Figure3,4,5].

Posterior segment was evaluated using + 78 diopter/ + 90 diopter lens and indirect Ophthalmoscopy and it was within normal limits in both eyes. Hence, the ocular examination of the patient helped us to

clinically diagnose left oculomotor nerve paresis and left abducens nerve palsy.

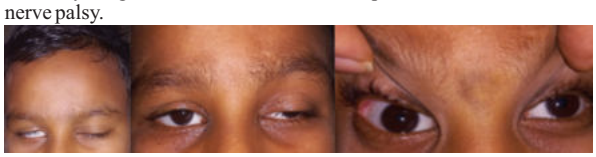

Figure 3: Right eye lagophthalmos

Figure 4: Left eye ptosis

Figure 5: Left eye mydriasis

Apart from the left oculomotor nerve and the left abducens nerve, the other cranial nerve which was impaired was the right facial nerve. On assessment of function of all the twelve cranial nerves individually, we found that the patient had right sided LMN facial nerve palsy. The positive findings which helped us to clinically diagnose it was as follows:

1) Loss of wrinkles on the right side of forehead
2) Right eye lagophthalmos
3) Loss of nasolabial folds on the right side of face and drooping of the angle of mouth on the right side on attempt to puff up cheeks [Figure 6]

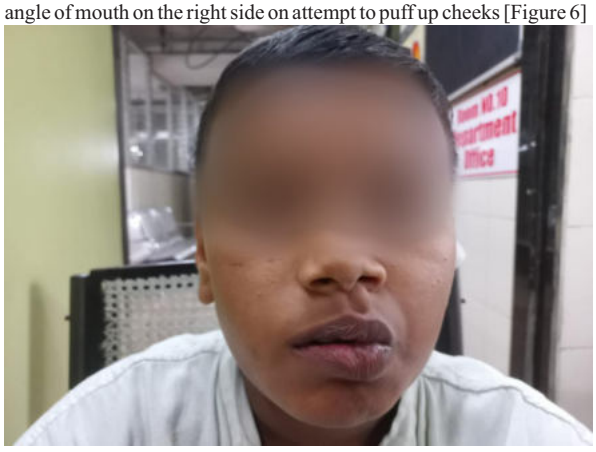

Figure 6: Loss Of Nasolabial Folds And Drooping Of Angle Of Mouth

It is important to note that the above mentioned clinical signs of the right LMN facial nerve palsy were seen 3 days after the incident of trauma. Following the clinical examination, routine blood investigations were carried out and he was run through various neuroimaging investigations. Plain axial Computed Tomography (CT) scan of brain on 160 slice MD CT scanner with thin sections, High Resolution Computed Tomography (HRCT) of temporal bones on 64 slice MD CT scanner with thin sections and Magnetic Resonance Imaging (MRI) scan of brain and temporal regions on 3.0 Tesla machine using T1 weighted gradient echo, T2 weighted fast spin echo and fast Fluid-Attenuated Inversion Recovery (FLAIR)sequences were performed.

The plain axial CT scan showed following findings [Figure 7,8]:

1) Linear undisplaced fracture of the right greater wing of sphenoid
2) Comminuted displaced fracture of left temporal bone involving the petrous part
3) Linear undisplaced fracture of the left parietal bone
4) Mild pneumocephalus

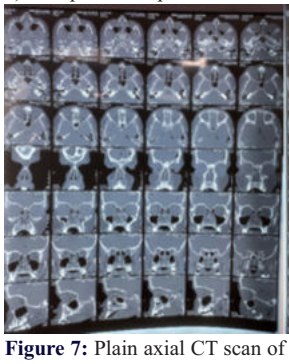

Figure 7: Plain axial CT scan of brain and orbit

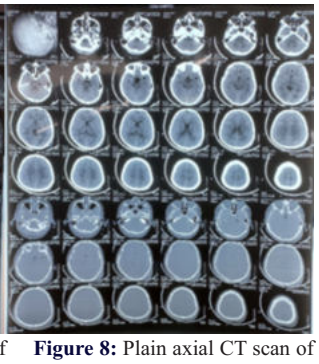

Figure 8: Plain axial CT scan of brain and orbit

HRCT of the left temporal bone showed following findings [Figure 9]:

1) Mixed (Otic capsule sparing) fracture involving the petrous temporal bone with fracture line extending to involve the tympanic part, tegmen tympani and tegmen mastoideum
2) Fracture line involving the anterior wall of the left carotid canal and bony part of the eustachian canal
3) Extensive soft tissue opacification noted in the middle ear, aditus to ad antrum and mastoid air cells suggestive of hemotympanum and hemomastoideum
4) Fracture line involving the lateral wall of the genu and tympanic segment of the left facial nerve canal while rest of the canal was normal
5) Middle ear ossicles were normal
6) The inner ear structures, the internal auditory canal and the external auditory canal were normal

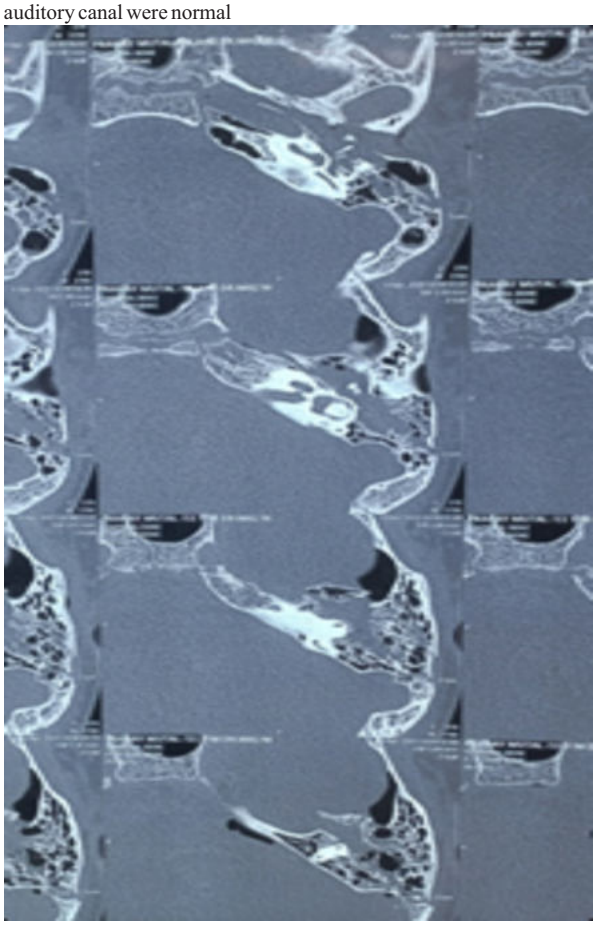

Figure 9: HRCT Of The Left Temporal Bone

HRCT of the right temporal bone showed no abnormalities and was within normal limits [Figure 10].

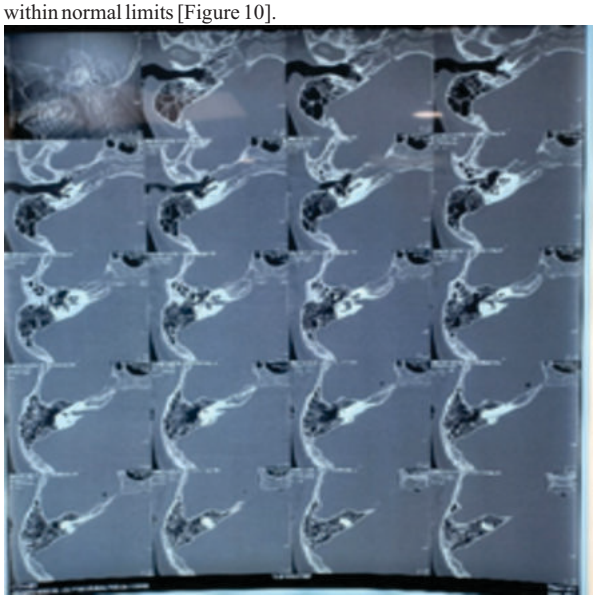

Figure 10: HRCT Of The Right Temporal Bone

MRI scan of brain showed following findings [Figure 11]:
1) Ill-defined non-enhancing T2/FLAIR hyperintense, T1 hypointense area in the cortical and subcortical region of the left temporal region along the tegmen mastoideum suggestive of non-hemorrhagic contusions
2) Rest of the supratentorial and infratentorial brain parenchyma was normal
3) Type 1 Anterior Inferior Cerebellar Artery vascular loop (AICA) was seen on the left side
4) Cochlea, vestibule and semicircular canals were normal

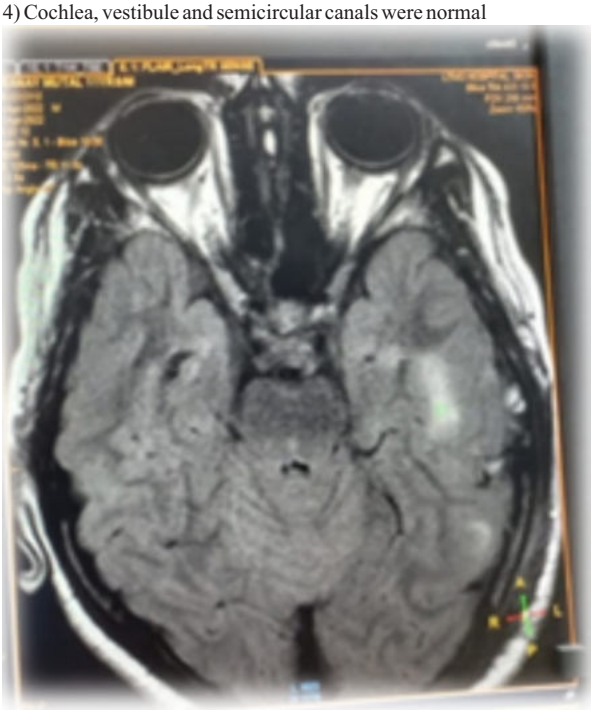

Figure 11: MRI Of The Brain

MRI of the left temporal region showed T1/T2 hyperintense non-enhancing soft tissue opacification involving the left mastoid air cells and left middle ear cavity suggestive of hemotympanum and hemomastoideum [Figure 12].

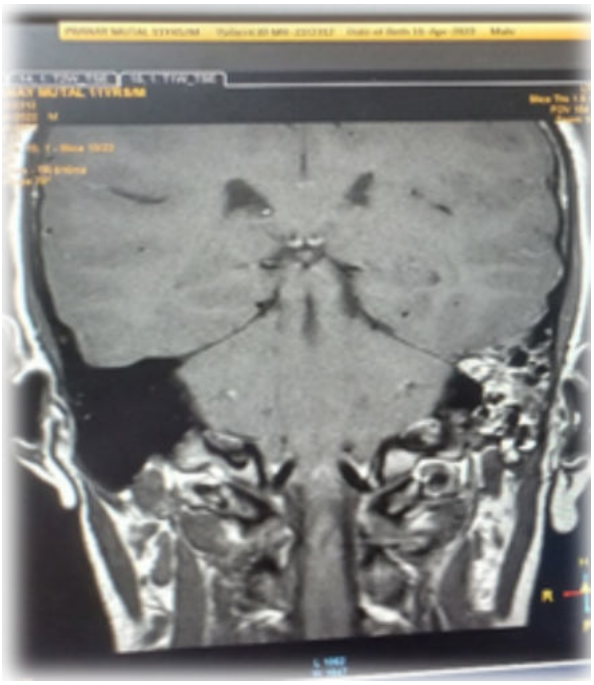

Figure 12: MRI Of The Left Temporal Region

MRI of the right temporal region showed no abnormalities and was within normal limits.

After hospitalization, the patient was administered intravenous infusion of one gram of methylprednisolone per day for three days followed by oral prednisolone of 1 milligram/ kilogram body weight/day which was gradually tapered over four to six weeks. He was also started on intravenous antibiotics and oral non-steroidal anti-inflammatory drugs. After two weeks of hospitalization, the patient was discharged. On discharge, the patient was relieved of pain and inflammation over the left temporal region of the head but had no relief from diplopia. The left oculomotor nerve paresis had healed completely with no improvement in the other two affected cranial nerves. During one month follow up, the abducens and facial nerve palsies still persisted.

DISCUSSION

The facial nerve is the seventh cranial nerve which comprises of motor, sensory and parasympathetic components. It is responsible for voluntary facial expressions, taste sensations in the anterior two-third of the tongue and control of salivary and lacrimal gland secretions.[2] It has three types of nuclei. They are:
1) Main motor nucleus
2) The parasympathetic nuclei
3) The sensory nucleus

Facial palsy is broadly classified into two types.[3] They are as follows:
1) Upper motor neuron facial palsy (UMN)
2) Lower motor neuron facial palsy (LMN)

The part of the main motor nucleus that supplies the muscles of the upper part of the face receives corticonuclear fibers from both cerebral hemispheres and the part of the nucleus that supplies the muscles of the lower part of the face receives corticonuclear fibers from the opposite cerebral hemisphere only.

Hence, in UMN facial palsy, there is unilateral facial palsy with sparing of the frontalis and orbicularis oculi muscles and the muscles of the lower part of the face are paralyzed in both UMN and LMN palsies. [3]

In UMN facial palsy, the voluntary facial movements might be impaired but the face may still move with emotional responses.

In LMN facial palsy, there is impairment of the muscles of facial expression for both voluntary and emotional responses.

Another important difference between the UMN and LMN facial palsy is that the clinical signs in UMN facial palsy are contralateral to the lesion while in LMN facial palsy, they are ipsilateral to the lesion. [3]

In this case report, the patient has loss of wrinkles on the right side of forehead, right eye lagophthalmos, loss of nasolabial folds on the right side and drooping of the angle of mouth on the right side, proving the involvement of the muscles of the both upper and lower part of the face and hence, justifying the clinical diagnosis of LMN facial palsy.

The involvement of the right part of the face and the lesion being the fracture of the left temporal bone indicates that the clinical signs are contralateral to the lesion which makes this case report a rare one. Possible mechanisms for the contralateral facial palsy in this case might be as follows:[4]
1) Severe emotional stress post trauma can trigger a dormant viral infection resulting in bell's palsy
2) Due to trauma, inflammatory reaction in and around facial nerve or mild swelling of the nerve in the facial canal can result in ischemia which in turn can decrease blood flow and oxygen to the nerve cells resulting in paralysis of the facial muscles.
3) Ischemia of the vasa nervorum supplying the facial nerve within the facial canal and a secondary ischemia induced by the bony canal might also cause bell's palsy
4) Demyelination of the facial nerve induced by an inflammatory process post trauma might cause bell's palsy

These pathological mechanisms causing facial nerve dysfunction would be better understood using neuroimaging investigation such as Steady state free precision (SSFP) MRI or three-dimensional (3D) constructive interference in a steady state (CISS) MRI sequence. [5]

Abducens nerve is the sixth cranial nerve which has the second longest intracranial course making it prone to injury following head trauma. It

exits the brainstem at the junction of the pons and pyramid of medulla, medial to the facial nerve. It then runs upwards and forward into the subarachnoid space between the pons and clivus.

During its course along the clivus, it is enclosed within a fibrous sheath called Dorello's canal. It runs upwards on the back of the petrous temporal bone near its apex. At the sharp upper border of the petrous bone, it bends forward at right angle under the petrosphenoidal ligament and enters the cavernous sinus by piercing its posterior wall at a point superior to the apex of petrous temporal bone. Hence, the abducens nerve usually gets injured at the petrous apex just before it enters the Dorello's canal following temporal bone fracture or head trauma. Since the facial nerve loops around the site of origin of the abducens nerve, it is usually impaired along with the latter following a head trauma. [6]

Oculomotor nerve is the third cranial nerve which has been affected along with the abducens nerve and the facial nerve in our patient. Injury to this nerve post head trauma mainly occurs due to the differential movements between the brainstem and supratentorial structures, which causes stretching of the nerve in turn resulting in distal fascicular damage.

The oculomotor nerve enters the cavernous sinus by piercing the posterior part of its roof on the lateral side of the posterior clinoid process. It pierces the dura of the cavernous sinus through the oculomotor triangle which consists of the anterior and posterior clinoid process and the petrous apex. The interclinoid ligament extending from the anterior to posterior clinoid process forms the medial margin of the triangle, the lateral margin by the anterior petroclinoid ligament extending from the anterior clinoid process to the petrous apex while the posterior margin is formed by the tough posterior petroclinoid ligament which extends from the posterior clinoid process to the petrous apex.

A direct traction injury due to the nerve stretching against the tough posterior petroclinoid ligament secondary to the downward displacement of the brainstem following head trauma results in swelling of the nerve and ischemia might occur due to the dural constriction at the point where the nerve pierces the dura of the cavernous sinus. [7]

Temporal bone fracture is usually a sequela of significant blunt head trauma resulting in serious complications such as facial nerve paralysis, cerebrospinal fluid leak, hearing loss and vascular injury.

Traditionally, it is classified in relation to the long axis of the petrous temporal bone, which runs obliquely from the petrous apex posterolaterally through the mastoid air cells. Using this plane, it is classified as follows: [1]
1) Longitudinal fractures
2) Transverse fractures
3) Mixed fractures

Longitudinal fractures constitute 70%-90% of the temporal bone fractures. They occur parallel to the long axis of the petrous temporal bone as a result of lateral impact to the skull. They have delayed onset and typically originate from the squamous temporal bone and extends to the petrous apex involving the walls of the external auditory canal (EAC), tympanic membrane, the glenoid fossa and the tympanic cavity. Complications include hemotympanum, conductive hearing loss (CHL), facial nerve paralysis and cerebrospinal fluid (CSF) otorrhea. Although less common, facial nerve paralysis occurs in 10%-20% of longitudinal fractures and the facial canal is usually affected at the geniculate ganglion or proximal tympanic segment. [1]

Transverse fractures constitute 10%-30% of the temporal bone fractures. They occur perpendicular to the long axis of the petrous temporal bone as a result of occipital impact to the skull and have an acute onset resulting in complete facial nerve paralysis. Complications include sensorineural hearing loss (SHL), pneumolabyrinth, facial nerve paralysis, CSF otorrhea and perilymphatic fistula.

Mixed fractures are the complex fractures which comprises both longitudinal and transverse elements. They are the most common type and complications include hemotympanum, perilymphatic fistula, CHL and SHL. [1]

Based on the involvement of the otic capsule, temporal bone fractures are classified as follows:
1) Otic capsule-sparing fractures
2) Otic capsule-violating fractures

Otic capsule-sparing fractures usually involve the squamous temporal bone and posterosuperior wall of the EAC and pass through mastoid air cells, middle ear, tegmen mastoideum and tegmen tympani. They can cause CHL or mixed hearing loss and epidural hematoma. The longitudinal fractures usually spare the otic capsule while the transverse fractures involve the otic capsule.

Otic capsule-violating fractures constitute 5%-20% of temporal bone fractures and is commonly involved in transverse fractures. Patients with otic capsule-violating fractures are twice likely to develop facial nerve paralysis, four times likely to develop CSF leakage and seven times likely to have profound hearing loss when compared with patients with otic capsule-sparing fractures. [1]

The important findings in the case history, clinical presentation and neuroimaging investigations in this case report which point towards otic capsule-sparing longitudinal fracture of the temporal bone are as follows:
1) Lateral or temporal impact to the skull
2) Delayed onset of the facial nerve paralysis
3) HRCT of the left temporal bone shows the fracture line running parallel to the long axis of the petrous temporal bone, sparing the otic capsule and extending to involve the tympanic part, tegmen tympani and tegmen mastoideum
4) HRCT and MRI of the left temporal bone shows soft tissue opacification involving the left middle ear and mastoid air cells suggestive of hemotympanum and hemomastoideum which are commonly seen in longitudinal temporal bone fractures.

CONCLUSION

Contralateral LMN facial palsy post head trauma associated with temporal bone fracture is a rare occurrence. Hence, detailed history with proper clinical evaluation of the patient with the help of advanced neuroimaging investigations such as SSFP MRI or 3D CISS MRI are very much essential to understand the possible pathological mechanism involved which in turn can help in accurate diagnosis, efficient treatment and management of the condition.

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