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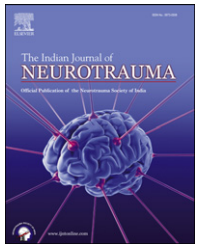

Case Report

Multiple cranial nerve palsies after head injury. A case report

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ABSTRACT

Injuries to the third fourth, sixth and seventh cranial nerves are common after head injury and may be missed during the initial assessment. Bilateral palsies of the third fourth and sixth nerves are also rare. We report a case of a 16-year-old male having bilateral third (partial), bilateral fourth, bilateral sixth and right lower motor neuron (LMN) facial nerve palsy after severe head injury. The only ocular movement patient was able to perform was adduction of the left eye. His CT scan revealed pneumocephalus (frontal and around the brain stem) and a midbrain contusion. He was managed conservatively. These palsies gradually improved over two years.

1. Introduction

Injuries to the third fourth sixth and seventh cranial (LMN)) nerves are common after head injury and may be missed during the initial assessment.[1,2] The incidence of cranial nerve injury in head injury varies between 5 and 23 percent.[2,3] Cranial nerve injuries associated with closed head injury has been found to be associated with a higher severity of injury.[4] Trauma forms an important etiological factor for multiple cranial nerve palsies.[5] Bilateral palsies of the third fourth and sixth nerves are also rare.[6] We report a case of a 16-year-old male having bilateral third (partial), bilateral fourth, bilateral sixth and right facial nerve palsy (LMN) after severe head injury. The only ocular movement patient was able to perform was adduction of the left eye. These palsies gradually improved over two years.

2. Case report

A 16-year-old male patient was admitted to the casualty with a history of driving a two wheeler without wearing a helmet and being hit by a speeding car. Patient immediately lost consciousness. Left ear bleed was present. There was no history of seizures, vomiting or CSF leak. Patient was immediately intubated and emergency management was performed. On examination his Glasgow Coma Score (GCS) was E1VTM4, left pupil was 5 mm fixed; right 3.5 mm reacting to light (both direct and consensual). There was no associated chest, abdomen and long bone injury. His blood parameters were within normal limits. A plain Computerised Tomography (CT) scan showed bifrontal and perimesencephalic pneumocephalus, cerebral oedema, small haemorrhage within the midbrain and a fracture of the temporal bone on

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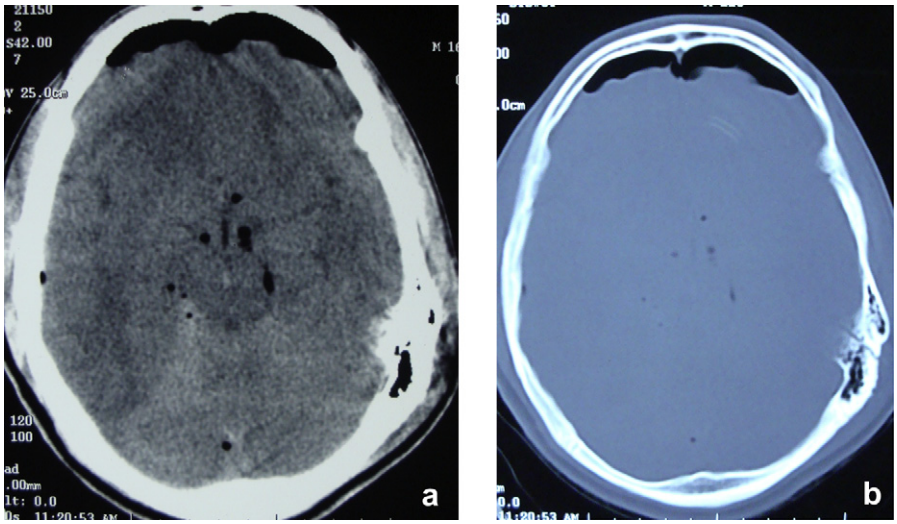

Fig. 1 – a: Plain CT head showing perimesencephalic and bifrontal pneumocephalus. b: Bone window showing fracture of left temporal bone.

the left (Fig. 1a, b). Patient was put on ventilatory support, and started on phenytoin sodium, 20% mannitol, frusemide and antibiotics. His vitals, haematological and biochemical parameters were stable all throughout. On day 3 he was tracheostomized. Patient regained consciousness on day 6. Mannitol and frusemide were tapered and withdrawn. Gradually the patient became fully conscious and alert. His tracheotomy was closed and oral feed started.

On a more detailed neurological examination we found right lower motor type facial palsy (Fig. 2) and bilateral ptosis (Fig. 3). Left pupil was 5 mm fixed; right 3.5 mm reacting to light, both direct and consensual palsy of bilateral 4th and 6th nerve was also there (Fig. 4). On looking to left (abduction) he developed nystagmus. Dolls eye and convergence were absent. The only movement he was able to perform was to adduct his left eye. Vision was normal in bilateral eyes. Other cranial nerves were normal. Motor and sensory examination was normal. Deep tendon reflexes were normal and planters were bilaterally extensor.

Patient was discharged on day 11 .On follow up at one month he was conscious alert oriented and his Glasgow outcome score was 5, however the extra ocular movements did not improve. At one year facial palsy completely recovered and the he was able to abduct the left eye. At two years patient had right 6th nerve palsy and left 4th nerve palsy, slight ptosis of left eye (Fig. 5). Rest all movements were present. Vision was normal. There was no nystagmus.

3. Discussion

Multiple cranial nerve palsies following closed head injury have been well documented.[3] Cranial nerve injuries are usually missed during the initial assessment of a head injury patient and hence the need to examine for, 4th 6th and 7th nerve for injuries.[1]

Head trauma accounts for 8–16% of all occulomotor palsies,[6,7] and occulomotor palsy is seen in 2.9–17.9% of all

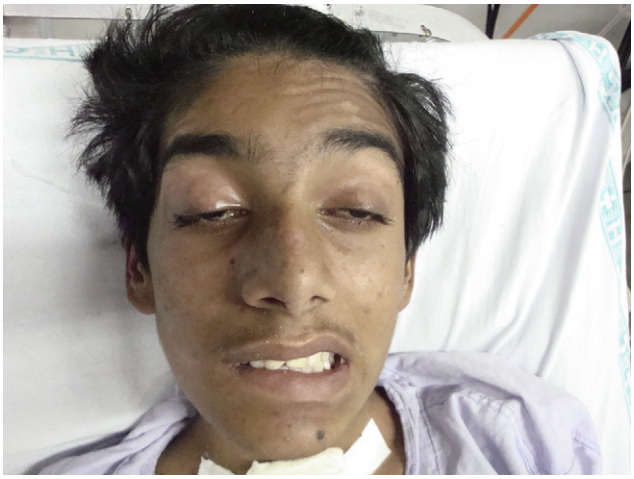

Fig. 2 – Clinical examination showing right facial palsy.

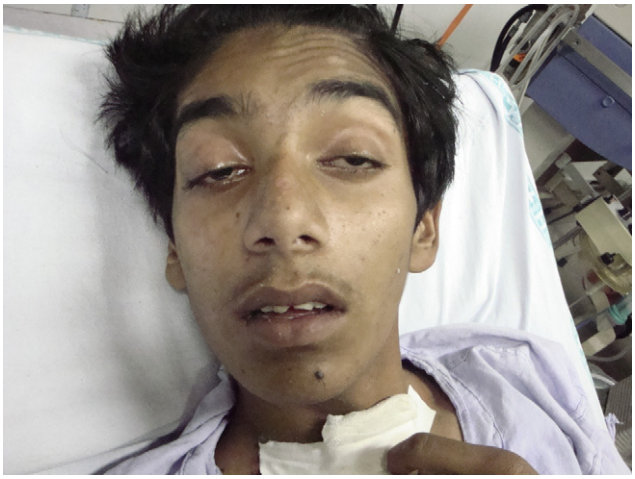

Fig. 3 – Clinical examination showing bilateral ptosis.

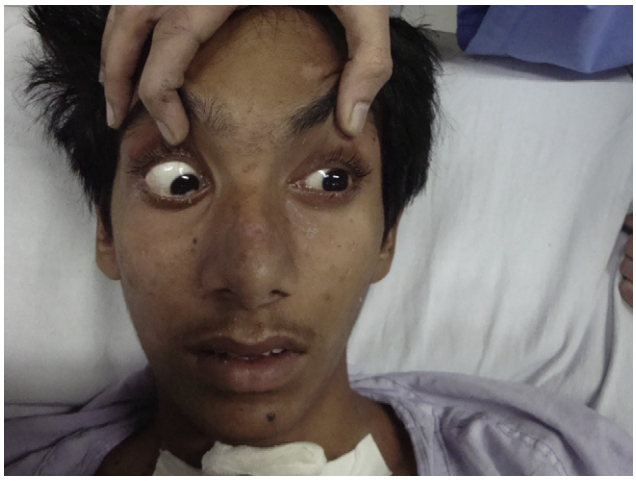

Fig. 4 – Ocular motor examination showing preserved extra ocular movement.

head injuries,[2,8] including those patients with multiple cranial nerve involvement. Third nerve involvement in CHI is more likely to be due to an expanding mass lesion.[9,10] Bilateral 3rd nerve palsy following CHI has also been well documented.[9] The mechanism of these injuries is complex.[10] Midbrain haematoma in the tectal region, compression of the nerve at the tentorial hiatus by uncus, avulsion or stretching of the nerve at mesencephalo-pontine junction are mechanisms of 3rd nerve palsy in head injury.[2,11] Occulomotor palsy may occur with other nerves in trauma along with skull base or clival fractures.

The incidence of trochlear nerve injury in head injuries is 2–2.14%,[2,12] and is associated with other cranial nerve injuries. Bilateral traumatic 4th nerve palsy is mostly due to trauma.[12] Trochlear nerve may be injured in isolation in its subarachnoid course. A sudden deceleration impact or blow to the head may cause the brain to move back, and the brain stem to impact against the tentorium, resulting in trochlear nerve injury. The injury can occur in dorsal midbrain, or in the free edge of the tentorium.[12]

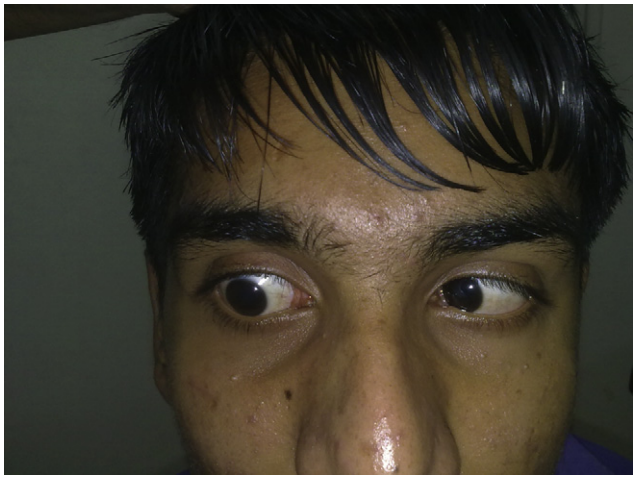

Fig. 5 – Clinical examination at two years.

Head injury accounts for about 3–15% of abducens palsies and may be associated with multiple cranial nerve injuries.[6,8] An incidence of 3% has been reported.[2] The long intradural course, its passage over the petrous ridge with its relative fixity under the petroclinoid ligament and to the cavernous sinus makes it vulnerable to stretch or tear.[13,14]

Trauma is an important cause of LMN type facial nerve palsy apart from Bells' palsy and Cerebellopontine angle tumours.[10,11] An incidence of 2–15.3% has been reported in head injury.[11] Facial nerve may be injured at its site of tethering at the geniculate ganglion, shearing can cause inintraneural contusion, oedema and haemorrhage or transaction.[2,10,11]

The multiple palsies in our patient may be explained by the small contusion in the midbrain and air around the brain stem causing direct compression.

4. Conclusions

Multiple cranial nerve palsies after head injury may not carry a bad prognosis as previously thought and may be reversible, hence the need to actively treat such patients. Moreover if multiple cranial nerve injuries are detected there is a need to further evaluate for brain stem injury by MRI.

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None.

Conflicts of interest

All authors have none to declare.

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