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A Study on Incidence Presentation and Outcome of Traumatic Cranial Nerve Injuries in Mild to Moderate Head Injuries in a Tertiary Care Hospital Salem

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ABSTRACT

The incidence of cranial nerve injury in craniocerebral trauma varies between 5 and 23% in various studies. To assess the incidence of CNI in TBI patients and identify the involvement of CN in mild to moderate head injury. A prospective hospital-based observational study was conducted on the incidence, clinical presentation and outcome of cranial nerve injuries (CNI) in 30 consecutive cases of traumatic brain injuries (TBI). After post-operatively patients were followed at 1st month, 3rd month, 6th month within the one-year duration. The incidence of CNI in TBI patients was 4.347% (30 patients). The facial nerve was the most common cranial nerve to be involved (50%), followed by the olfactory nerve (20%), optic nerve (10%), oculomotor nerve (10%) and one each in the trigeminal nerve (3.33%), abducens nerve (3.33%) and vestibulocochlear nerve (3.33%). The study showed that Post-op follow-up within 12 months duration shows 9 (30%) patients with complete recovery, 15 (50%) patients with partial recovery, and 6 (20%) patients with no recovery. Cranial nerve injury was more common in patients with severe head injury ($p<0.005$), younger age group, the associated base of skull fractures and facial fractures ($p<0.005$). 70% of patients had a delayed presentation of CNI. Younger age group and delayed onset of deficit are associated with a significantly better outcome. CNI is a major cause of morbidity in TBI patients. A significant percentage of patients present with delayed presentation of CNI. All patients admitted with TBI should be examined meticulously for CNI on follow-up.

INTRODUCTION

From a global perspective, traumatic brain injury (TBI) constitutes a major cause of death and disability making this condition a pressing issue for public health services across the world[1]. Posttraumatic cranial nerve injuries are not uncommon and can occur by shearing forces, rapid acceleration/deceleration, or injury to the skull base. The incidence of cranial nerve injury in craniocerebral trauma varies between 5 and 23 percent in various literature[2]. Initial evaluation by history, clinical and neurological evaluation effectively assesses the sensorium, cortical function and hemodynamic status. Added to this, a rapid evaluation of ocular movements, pupils, facial symmetry and laryngeal function establishes the functional integrity of the cranial nerves. Cranial nerves along with major arteries and bridging veins act as anchors to the brain in a sea of cerebro spinal fluid. Injury to cranial nerves can occur by shearing forces, rapid acceleration/deceleration, injury to skull base, penetrating cranio-cerebral injuries, especially those through the skull base and as a sequel to various surgical procedures. The consequences of cranial nerve injuries are mild to severe morbidity. Most of these injuries do not require active intervention in acute stage, but requires long term follow up, repetitive reconstructive procedures. Numerous studies analyzed cranial nerve palsy after severe head injury, however there are nonspecific studies about cranial nerve injury following mild to moderate head trauma (initial GCS score 9-15)[3,4]. The epidemiology and implications of associated cranial nerve injuries (CNI) in mild to moderate TBI are largely unknown. Hence this study would like to prospectively evaluate the incidence and outcome of traumatic cranial nerve deficits. The primary outcome was to identify the incidence and outcome of cranial nerve injury in mild to moderate head injury patients and their recovery in a tertiary care hospital in Salem. The secondary outcome was the recovery of the function over a period of time.

MATERIALS AND METHODS

This is a Prospective Hospital-based Observational cross-sectional study done within a period of 12 months duration at the Department of Neurosurgery, GMKMCH, SALEM. The study protocol was approved by the Institutional Ethics committee. An individual consent form from the patient/attendee was obtained during the study enrolment. All the males and females of age between 18-75 yr presented with cranial nerve injuries after head trauma with GCS = 9. Severe head injury patients (GCS = 8) were excluded from the study. History of drooping of the shoulder, deviation of the tongue, weakness of upper limbs and lower limbs. Built, nourishment, Pallor/cyanosis/clubbing/ jaundice/ lymphadenopathy/pedal edema, Neurocutaneous markers were recorded. Computed tomography scanning using brain and bone windows were performed when at least one of the following risk factor was present-post-traumatic amnesia, loss of consciousness, post-traumatic seizures, headache, vomiting, focal neurological deficit, skull fracture, coagulopathy, anticoagulation therapy.

A special investigation like Orbital, Anterior fossa, petrous bone, posterior fossa CT scans, CT cisternogram, audio grams, visual evoked potential, MRI scans were conducted in specific circumstances like visual defects, CSF rhinorrhea, hearing defects etc. CT scans, 3 types of traumatic injury were differentiated: no lesion, skull fractures, brain parenchymal injury like a contusion, SDH, EDH, SAH. After post-operatively patients were followed at 1st month, 3rd month, 6th month within the one-year duration. Recovery was assessed in the form of complete recovery, partial recovery, no recovery.

Analysis: Metric variables are presented as mean values with standard deviation (SD) and counts as percentages. For mean values and percentages, a 95% confidence interval (CI) was reported in the CNI. A Chi-squared test was performed in the sense of targeted testing ($p<0.01$). Statistical procedures were conducted with IBM SPSS Statistics (version 24, International Business Machines Corporation, Armonk, NY, USA).

RESULTS AND DISCUSSIONS

Demographics and Etiology: A total of 1050 cases were admitted to our hospital with a head injury during the study period, among them, 690 cases had mild to moderate head injury. We identified a total of 30 patients with cranial nerve injury, with an incidence of 4.34%. Our study revealed the maximum head injuries with a mean age of 45.3±20.5 years (range from 20-60 years). Males accounted for 70% of our study population (21 males as compared with 9 females). Road traffic accident (RTA) was the most common mode of injury, which accounted for 80% of the cases (n = 24), while only 20% of the cases were due to low-velocity injuries (each 2 in fall from height+ Assault+Others) (n = 6). Initial presenting injury was mild (GCS: 13-15) in 17 (56.67%) patients and moderate (GCS-9-12) in 13 (43.33%) patients.

Distribution of Cranial Nerves Involved (CNI): In our study population, the Facial nerve was the most common CN to be involved (50%), followed by the olfactory nerve (20%), optic nerve (10%), oculomotor nerve (10%) and one each in trigeminal nerve (3.33%), abducens nerve (3.33%) and vestibulocochlear nerve (3.33%). The most affected cranial nerve was facial nerve (cranial nerve VII), followed by optic nerve (cranial nerve II). When more than one cranial nerve

Table 1: Distribution of posttraumatic cranial nerves involved

Cranial nerve Injured	No. of patients	Percentage
Olfactory	6	20
Optic	3	10
Oculomotor	3	10
Trigeminal	1	3.33
Abducens	1	3.33
Facial	15	50
Vestibulocochlear	1	3.33
Multiple cranial nerves		
I, II and III	1	3.33
III, IV and V	2	6.66
VI and VII	1	3.33
II, III, IV and VI	2	6.66
VII and VIII	1	3.33

involved, the most frequent association occurred between CNs III, IV and VI. Post traumatic single cranial nerve palsy was observed in 23 (76.67%) patients and multiple cranial nerves were affected in 7 (23.33%) patients.

Olfactory Nerve Injury: Six patients (20%) had post-traumatic olfactory dysfunction. One of these patients had multiple cranial nerve injuries of optic and oculomotor nerves. Five patients had olfactory nerve injury only. In these patients only one patient had a moderate head injury, the remaining five patients sustained a mild head injury. A patient with multiple cranial nerve injuries had a moderate head injury. Among 6 olfactory dysfunction patients, 2 patients had unilateral olfactory dysfunction, remaining patients had bilateral dysfunction. Among these 6 olfactory dysfunction patients, 2 patients had cribriform plate fracture, 1 patient had bifrontal contusions after occipital blow (fracture), 1 patient had frontal depressed fracture with rt. Frontal EDH for which he underwent emergency craniotomy and evacuation of EDH. One patient had multiple facial bone fractures. Among 6 olfactory dysfunction patients, 2 patients had CSF rhinorrhea, 1 patient underwent surgical repair for CSF rhinorrhea which although could not gain their olfaction but their CSF leak has satisfactorily stopped. Out of 6 olfactory dysfunction patients, two patients showed partial improvement after a follow-up within 12 months, remaining five patients have shown no improvement after follow-up within 12 months.

Optic Nerve Injury: Three patients (10%) had post-traumatic optic nerve injury. Mild head injury was common (2) compared with moderate head injury (1). Frontal (2) and frontotemporal (1) injury was the common mode of injury. All patients had orbital fractures. Multiple orbital fractures extend up to the orbital apex (1 patient), lateral wall (1 patient) and medial wall (1 patient). In all patient's visual acuity was compared using a standard ophthalmological conversion from the values of no light perception, light perception, perception of hand movement and finger counting. Three patients were treated conservatively with methylprednisolone, two cases were improved within 12 months up, in the remaining one patient there is no improvement in vision.

Oculomotor Nerve Injury: Three patients (10%) developed post-traumatic oculomotor nerve injury. Mild head injury patients(2) had partial IIIrd nerve palsy with involvement of superior rectus and elevator palpebrales superiores resulting in ptosis, upward gaze paresis. Among three patients, 1 had extensive skull-base fracture with involvement of III, IV, VI, V1. All patients managed conservatively. Out of 3 patients, 2 patients recovered partially, 1 patient did not recover.

Trigeminal Nerve Injury: Only one patient had Vth nerve palsy. The patient presented with sensory loss in that area. Managed conservatively. The patient developed paraesthesias in that area which are managed with carbamazepine which is recovered after 3 months of follow-up.

Abducens Nerve Injury: Only one patient had an abducens nerve injury. This patient had multiple cranial nerve palsies. This patient was female. This patient presented with a mild head injury. This patient managed conservatively. This patient recovered after the follow-up within 12 months period.

Facial Nerve Injury: A total of 15 patients had facial nerve injuries. Among them, 10 patients had isolated facial nerve palsy and 5 patients had multiple cranial nerve palsies. Among them 13 patients were male and 2 patients were females. One patient had bilateral facial nerve palsy. 9 patients presented with mild head injury and 6 patients presented with moderate head injury. All were managed conservatively. There is partial improvement in 12 patients.

Vestibulo Cochlear Nerve Injury: Only one patient had VIIIth cranial nerve. This patient had isolated VIIIth nerve palsy. This patient was male. This patient presented with a mild grade head injury and managed conservatively. Partial improvement in the hearing was observed after the follow-up within 12 months period. None of them suffered from lower cranial nerve injuries.

Outcome: CNI was more common in patients with moderate head injury, younger age group and associated skull base and facial fractures. Recovery was recorded in 70% cases (n = 21): 13 (43.33%) patients had partial recovery, while 8 (26.67%) patients had complete recovery. Younger age group and delayed onset of deficit were associated with significantly better outcomes. This study aimed to determine the prevalence of CNI following mild to moderate head

trauma in our hospital. We studied 30 patients with cranial nerve injury following mild to moderate head trauma to document its incidence, correlate CN lesion with CT scan findings, and evaluate the clinical outcome of every cranial nerve injured. Among the total 30 cases, 17 (56.67%) patients had mild, 13 (43.33%) patients had a moderate head injury. Thirty patients were affected in our study, with an incidence of 4.347%. There is a male preponderance 70% male than female (30%). 23 (76.67%) patients had single cranial nerve injury; 7 (23.33%) patients had multiple cranial nerve injuries. The commonest associations were III, IV, VI, II, III, IV, VI, VII, VIII. The single nerve most affected is CN VII. These results are consistent with Patel *et al*. study. The olfactory nerve is the most frequently injured cranial nerve in the literature with a wide range from 4-60%[2]. In our study, 20% of patients presented with olfactory nerve dysfunction. This can be explained by the patient population presenting to the tertiary care center, self-assessment may under or overestimate the incidence of olfactory nerve dysfunction because of the lack of awareness of olfactory function. 40% of patients with olfactory dysfunction were aware of their dysfunction[5]. Out of 6 patients, two patients showed partial improvement within a follow-up of 12 months, and the remaining patients have shown no improvement. Kern *et al*.[6] found that recovery occurred in more than one-third of their cases. This is consistent with our study.

In our study, 3 patients had post traumatic optic nerve injury. Mild head injury was common in this. Three patients were treated conservatively with methylprednisolone, two were improved after 3 months follow up, in remaining one patient there is no improvement in vision. In the literature, traumatic optic nerve injuries occur in 0.5-5% of head injuries[7]. Three patients (3.33%) developed post-traumatic oculomotor nerve injury. In these patients, maximum patients had multiple cranial nerve palsies. Mild head injury patients (2) had partial IIIrd nerve palsy with involvement of superior rectus and elevator palpebrales superiores resulting in ptosis, upward gaze paresis. The incidence of oculomotor palsy was 2.9% in Patel *et al*.[8] study. The complex anatomy of the oculomotor nerve makes clinical findings highly variable according to the location of the injury. Oculomotor nerve injury is associated with lower GCS compared with other cranial neuropathies[9]. The prognosis of traumatic oculomotor palsy is poor and full recovery is uncommon. A prolonged period (up to years) of healing is usually anticipated. This is also correlating with a recovery rate <50%, which is also partial[10]. Damage to the trigeminal nerve most often occurs to its peripheral branches during severe maxillofacial and skull base injuries. The patient managed conservatively, later the patient developed cutaneous hyperesthesia, which was managed successfully with carbamazepine for 6 months. In the literature review, craniofacial trauma is the most common form of injury and 70% of patients suffer from paraesthesias[11].

In our study, only one patient presented with abducens palsy and had multiple cranial nerve palsies. The patient managed conservatively. This patient recovered after a follow-up within 12 months. These findings are consistent with reports from Bhatoe[12] and Chung *et al*.[13], who suggested an observation period of 3 months to allow sufficient time for maximal spontaneous recovery. In our study 15 (50%) patients suffered from facial nerve injury, among them, 10 patients had isolated facial nerve palsy and 5 patients had multiple cranial nerve palsies, making it the most common cranial nerve to be injured in head trauma. This is consistent with report from Patel *et al*. study. All patients in this population were managed conservatively. There is partial improvement in 15 patients. The commonest mode of injury is a road traffic accident. The significance of delay in onset of symptoms of facial palsy was compared with those of immediate onset in terms of prognosis with conservative management. 50% of our patients with facial palsy had intermediate HB grade (grade IV) at presentation. Functional improvement was considered as recovery. In our study, five of the six patients with delayed onset facial nerve paralysis showed complete recovery at 6 months with conservative treatment. Our results are in concurrence with the findings of Turel *et al*.[14].

It was suggested that delayed onset incomplete facial nerve injuries have a good prognosis when conservative treatment was advocated. Trauma to the CN VIII varies from concussion of cochlea and semicircular ducts to fracture of the petrous bone. Conductive deafness usually shows signs of recovery, sensorineural deafness, however, has a worse prognosis, especially if the initial hearing loss was complete. This finding is consistent with our results. In our study, only one (3.33%) patient was suffered from hearing loss, among them one had isolated cochlear nerve injury. This patient was evaluated with audiometry and managed conservatively. Our study showed that Post-op follow-up within 12 months duration shows 9 (30%) patients with complete recovery, 15 (50%) patients with partial recovery, and 6 (20%) patients with no recovery. The patients with partial recovery are expected to attain complete recovery in the coming weeks or months. Coello *et al*., reported a 69% recovery of CN deficit, while Patel *et al*. showed 60% recovery of CN deficit. The common diagnostic difficulties in this study were, Perseverance

is required to diagnose cranial nerve injury in the presence of moderate head injury. Unlike hearing loss and vision, the sense of smell was difficult to evaluate. Moderate head injury may obscure all but, third, sixth and seventh cranial nerve damage. Fourth nerve injury diagnosis in association with third nerve injury and in altered sensorium patients. In patients with vertical diplopia and orbital trauma, it is very difficult to differentiate whether nerve injury or superior oblique muscle injury caused it. With auditory canal blockage, it is difficult to distinguish between eighth nerve damage or ossicular disruption.

CONCLUSION

The incidence of cranial nerve injury following mild to moderate head injury is 4.25%. Young people are the most affected. Motor vehicle accidents are the most common cause of injury. The cranial nerves that presented with the highest incidence of palsy in our study were the facial, oculomotor, optic, olfactory nerves in that order. In contrast, the trigeminal were least affected and lower cranial nerves were not injured. Most of the cranial nerve injuries are managed conservatively with an acceptable outcome. Decompression of the optic nerve by surgery was the only procedure in 1 case in our study Surgery for CSF rhinorrhea with anterior cranial fossa corpetting was done 1 case. Most of the cranial nerve injuries were due to a fracture of the base of the skull which was a high-impact head injury. Meticulous neurological examination of all CNs should be conducted in all TBI patients on follow-up.

REFERENCES

1. Hyder, A.A, C.A. Wunderlich,P. Puvanachandra, G. Gururaj ,O.C Kobusingye, 2007. The impact of traumatic brain injuries: a global perspective. Neur. Rehabil., 22: 341-353.
2. Keane, J.R, R.W. Baloh, 1992. Post-traumatic cranial neuropathies. In, Evans RW (ed.). The Neurology of Trauma. Philadelphia. Saunders, 10: 849-868.
3. Teasdale, G. and B. Jennett, 1974. Assessment of coma and impaired consciousness. The Lancet, 304: 81-84.
4. Gjerris, F., 1976. Traumatic lesion of visual pathway. In: Handbook of Clinical Neurology., Vinken, P.J. and G.W. Bruyn, (Eds.)., North Holland Publishing Company,, Amsterdam, Netherlands, pp:
5. Haxel, B.R., L. Grant and A. Mackay-Sim, 2008. Olfactory dysfunction after head injury. J. Head Trauma Rehabilitation, 23: 407-413.
6. Kern, R.C., B. Quinn, G. Rosseau and A.I. Farbman, 2000. Post-traumatic olfactory dysfunction. The Laryngoscope, 110: 2106-2109.
7. Steinsapir, K.D. and R.A. Goldberg, 1994. Traumatic optic neuropathy. Survey Ophthalmol., 38: 487-518.
8. Patel, P., S. Kalyanaraman, J. Reginald, P. Natarajan and K. Ganapathy *et al.*, 2005. Post-traumatic cranial nerve injury. The Indian J. Neurotrauma, 2: 27-32.
9. Dhaliwal, A., A.L. West, J.D. Trobe and D.C. Musch, 2006. Third, fourth, and sixth cranial nerve palsies following closed head injury. J. Neuro-Ophthalmol., 26: 4-10.
10. Heinze, J., 1969. Cranial nerve avulsion and other neural injuries in road accidents. Med. J. Australia, 2: 1246-1249.
11. Sandstedt, P. and S. Sörensen, 1995. Neurosensory disturbances of the trigeminal nerve. J. Oral Maxillofacial Surg., 53: 498-505.
12. Bhatoe, H.S., 2007. Trauma to the cranial nerves. Indian J. Neurotrauma, 4: 89-100.
13. Chaung, S.M., G.A.Fenton, J.G.Schmidt, J.B. Selhorst, 1996. Trauma to the cranial nerves and brain stem. In: Neurotrauma,, Narayan,R.K., J.E. Wilberger,J.T. Povlishock, (Ed.)., McGraw-Hill,, New York.,, pp: 621-638.
14. Turel, K.E., N.K. Sharma, J. Verghese and S. Desai, 2005. Post traumatic facial paralysis treatment options and strategies. The Indian J. Neurotrauma, 2: 33-34.