A Prospective Study on the Incidence and Outcome of Cranial Nerve Injuries in Patients with Traumatic Brain Injuries

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Abstract

Background 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.

Methodology A prospective study was conducted on the incidence and outcome of cranial nerve injuries (CNI) in 256 consecutive cases of traumatic brain injuries (TBI). Patients over the age of 5 years with a follow-up of 6 months in the period from September 2017 to November 2018 in our institution were sampled.

Results A total of 256 patients were included in our study. The incidence of CNI in TBI patients was 14.8% (38 patients). Facial nerve was the most common cranial nerve to be involved, followed by olfactory nerve and vestibulocochlear nerve. Cranial nerve injury was more common in patients with severe head injury ($p < 0.005$), younger age group, associated base of skull fractures ($p < 0.001$), and facial fractures ($p < 0.005$). Twenty-eight patients (73.7%) had a delayed presentation of CNI ($p < 0.001$). Of the 73.6% patients who recovered, 16 (42%) patients had partial recovery, while 12 (31%) patients had complete recovery. Younger age group ($p < 0.05$) and delayed onset of deficit ($p < 0.001$) were associated with significant better outcome.

Conclusions CNI are a major cause of morbidity in TBI patients. All patients admitted with TBI should be examined meticulously for CNI on follow-up.

Keywords
- head injury
- cranial nerve
- traumatic brain injury

Introduction

Traumatic brain injury (TBI) is the leading cause of mortality and morbidity in adult population.[1,2] Injury to the cranial nerves (CN) is not uncommon.[3] The incidence of cranial nerve injury (CNI) in craniocerebral trauma varies between 5 and 23 percent.[4,5] Most of the studies in literature are retrospective studies, with very few prospective studies. The present study aims to assess the incidence and outcome of CNI in patients with TBI.

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Aims and Objectives

This study aims to assess the incidence of CNI in TBI patients in our population and identify the involvement of CN with the severity of head injury. We also aim to assess the outcome of patients with CNI in head trauma patients.

Materials and Methods

We conducted a hospital-based, prospective observational analytical study in our institution from September 2017 to November 2018. A total of 256 patients with head injury over the age of 5 years, with a follow-up of 6 months, were included in this study. SPSS version 22.0 software (IBM) was used for statistical analysis. The patients were examined immediately during the secondary survey for CNI. In addition, patients were examined every day until the day of discharge and then on monthly follow-up for 6 months. Severity of TBI was assessed using initial Glasgow coma scale (GCS) and Marshall CT grading of TBI. Incidence of cranial nerve palsies was studied in relation to the mode of injury, severity of head injury, and radiological findings.

In the acute setting, all patients were clinically examined for pupillary reflex. Relative afferent pupillary defect (RAPD) is a reasonably reliable test to diagnose unilateral optic nerve injury, even in patients with moderate-to-severe head injury.[6] In responsive patients, visual acuity was assessed, while flash visual-evoked potential (VEP) was done in unresponsive patients when indicated.

The House–Brackmann (HB) 6-point scale was utilized for grading facial nerve injury.[7,8] Severity of facial palsy was classified into low grade (HB 2–3), intermediate (HB 4), and high grade (HB 5–6).[9] HB grade II to IV was considered as incomplete palsy and complete palsy when the HB scale was V to VI. Clinical recovery from CN palsy was classified into three grades: no recovery (no evidence of clinical improvement in the nerve function), partial recovery (some objective clinical recovery), or complete recovery (at the end of the follow-up period, the patient was asymptomatic).[10] Recovery was also correlated with the demographic profile, associated radiological findings, specific treatment including surgical decompression, administration of steroid and specific rehabilitative measures done. Overall outcome of TBI patients with CN injuries were also studied and compared with patients without CN deficits.

Results

Demographics

After applying the inclusion and exclusion criteria, 256 patients were included in our study. Our study revealed maximum head injuries in the age group of 20 to 60 years (64.1%), with a mean age of 46.03 years and a standard deviation (SD) of 20.08 years. Males accounted for 74.6% of our study population (191 males as compared with 65 females). A total of 171 (66.8%) patients had mild head injury, 56 (21.9%) had moderate head injury, and 29 (11.3%) patients were admitted with severe head injury.

The incidence of cranial nerve injury in TBI patients was 14.8% (38 out of 256). ►**Table 1** shows the distribution of CNI in our study population. Thirty patients (78.9%) had single CN palsy, while multiple CN palsies were noted in eight patients (21.1%). Road traffic accident (RTA) was the most common mode of injury, which accounted for 78.1% of the cases, while only 21.9% of the cases were due to low-velocity injuries. As much as 17.24% patients with a Marshall grading of < 2 on CT had CN deficit as opposed to 14.14% patients with Marshall grading >2 on CT with CN palsy. Facial nerve was the most common CN to be involved, followed by olfactory nerve and vestibulocochlear nerve. Of the 38 patients with CNI, twenty-eight patients (73.7%) had a delayed presentation of CNI compared with 10 (26.3%) patients with immediate presentation, and the results were found to be statistically significant ($p < 0.001$) (►**Table 2**).

CNI was more common in patients with severe head injury, younger age group, and associated skull base and facial fractures. CN deficit was noted in 31.1% of patients with severe head injury as opposed to 11% and 10% in mild-to-moderate head injury, respectively, and the difference was statistically significant ($p < 0.005$). As much as 34% patients with

Table 1 Distribution of posttraumatic cranial nerves involved

Cranial nerve injured	Number of patients	Percentage (%)
Olfactory nerve	9	18.75
Optic nerve	7	14.6
Oculomotor nerve	7	14.6
Trochlear nerve	2	4.1
Trigeminal nerve	0	0
Abducens nerve	2	4.1
Facial nerve	**10**	**20.8**
Vestibulocochlear nerve	9	18.75
Glossopharyngeal, vagus	2	4.1

Note: Bold value signifies incidence in the present study.

Table 2 Onset of presentation of CNI

Cranial nerve	Onset of CNI	
	At presentation (immediate)	On follow-up (delayed)
Olfactory nerve	2	7
Optic nerve	1	6
Oculomotor nerve	0	7
Trochlear nerve	2	0
Trigeminal nerve	0	0
Abducens nerve	1	1
Facial nerve	4	6
Vestibulocochlear nerve	3	6
Glossopharyngeal, vagus	1	1

Abbreviation: CNI, cranial nerve injury.

skull base fractures had CN deficit as opposed to only 7.81% patients having CN palsy without skull base fractures, and this difference was found to be statistically significant ($p < 0.001$). CNI was noted in 24% with facial fractures as opposed to only 9.6% patients having CN palsy without facial fractures. This difference was found to be statistically significant with a p value of < 0.005.

Outcome of Cranial Nerve Injury (CNI) in TBI patients

Recovery was recorded in 73.6% cases: 16 (42%) patients had partial recovery, while 12 (31%) patients had complete recovery. Recovery was noted in 67.9% patients who had delayed onset of CN deficit as opposed to only 32% recovery in patients with immediate presentation. This difference was found to be statistically significant with a p value of < 0.001.

Recovery was seen in 81.25% patients under the age of 60 years, while only 33% recovery was seen in the older age group of > 60 years. This difference was found to be statistically significant ($p < 0.05$).

These imply that younger age group and delayed onset of deficit were associated with significant better outcome.

Discussion

With the constant increase in high-velocity accidents and violence over past decades, the matter of acute head trauma is one of prime importance. The acute effects and the chronic sequelae of trauma are often severely disabling, with CNI contributing to the disability. Maximum head injuries were seen in the age group of 20 to 60 years–64.1% (average age–46.03 years). There was a definitive male preponderance with 74.6% patients being males. This is similar to the study conducted by Equabal,[11] which showed male preponderance of 83%. However, there was no significant difference in the incidence of CNI, depending on sex. The head injuries which need admission to hospitals are mainly high-velocity injuries, that is, due to RTA. In the prospective study[12] of the Traumatic Coma Data Bank (TCDB), motor vehicle crashes were the cause of injury in 55% of patients aged 15 to 25 years, whereas only approximately 5% suffered falls. However, in the age range above 55 years, 45% suffered falls and only approximately 15% were in motor vehicle crashes. However, this was not the findings in our study. RTA is the most common cause of the TBI in all age groups. Our results agree with other studies such as Borkar,[13] Equabal (2005),[11] and Yattoo.[14]

All head injury patients, after the initial resuscitation, were closely monitored for CNI. Meticulous neurological examination of all the 12 pairs of CNs were done to find out any CN deficits. They were examined immediately during the secondary survey to look for CNI. In addition, patients were examined every day until the day of discharge and then on monthly follow-up for 6 months. This method of examination was chosen to negate the possibility of missing the CNI at the time of presentation. This technique helped to identify which CNs were immediately involved and those that had a delayed presentation.

There, however, was a possibility of missing the involvement of olfactory nerve and vestibulocochlear nerve in the acute setting, especially in patients with a low GCS score. However, in our study population, of the nine patients with olfactory nerve injury, eight patients (89%) had mild head injury, while only one patient had severe head injury. Seven of the nine patients (79%) with vestibulocochlear nerve injury had mild head injury, while moderate-to-severe head injury accounted for one patient each.

Our study was prospective in nature, which further strengthens the credibility of examination of patients and bringing down the chances of missing the CNI.

CNI were noted in 38 of 256 patients with TBI–an incidence of 14.8%. Our results agree with other studies such as Patel et al[5] and Jin et al.[4] However, Coello et al[10] in his study had an incidence of only 0.3%. This disparity in incidence is likely due to the inclusion criteria where they had studied only cases with mild head injury (►**Table 3**). Moderate-to-severe head injuries were not included in their study. Of the 38 patients, RTA were the cause in 33 patients (86.9%).

In our study, facial nerve was injured in 10 cases (26%) and was the most common CN to be injured. Olfactory nerve and vestibulocochlear nerve were injured in 23% patients. Our results vary from that of published literature, where anosmia was the most common manifestation of posttraumatic CNI. The patient population that presented to our institution could explain the reason for this disparity in results.[3] Trigeminal nerve involvement was not seen in any of the patients. Incidence of CNI was found to be higher in patients with severe

Table 3 Incidence and order of involvement of CNI among various studies

Study	Cranial Nerve Injury	Incidence (%)	MC CN involved	Cranial Nerve Injury	
				Single	Multiple
Apollo Hospital, Chennai[5]	100	12.6	Facial N (20%) Optic N (11%)	67%	33%
Coello et al[10]	49	0.3	Olfactory N(26%) Facial N (22%)	38 (77.6%)	11 (22.4%)
Jin H et al[4]	312	9.1	Optic N (25%) Olfactory N (21%) Facial N (15%)	218 (69.8%)	94 (30.1%)
Present	38	**14.8**	Facial N (26%) Olfactory N (23%) Vestibulocochlear N (23%)	30 (78.9%)	8 (21.1%)

Abbreviation: CNI, cranial nerve injury.
Note: Bold value signifies incidence in the present study.

head injury as assessed by the initial GCS score. As much as 31.1% of patients with severe head injury had CN deficit as opposed to 11% and 10% in mild-to-moderate head injury, respectively. This difference was statistically significant with a *p* value of <0.005. However, this finding is contradictory to the findings of Coello et al[10] who reported that trivial head trauma that causes a minor head injury (GCS score 14–15) can result in CN palsies with a similar distribution to moderate-to-severe head injuries.

Treatment Protocol for CNI

As and when CNI was clinically demonstrated, patients were subjected to CT brain and bone windows. In the case of facial nerve injury, high-resolution computerized tomography (HRCT) of temporal bone (1 mm slices) and CT orbit (1–2 mm slices) in patients with optic nerve injury was done to find out any transection of the nerve or any impingement of the nerve with broken bone fragments. Once a surgically reconstructive cause for the nerve injury, cerebrospinal fluid (CSF) rhinorrhea/otorrhea and pneumocephalus were excluded, the patient was started on high-dose steroids intravenously (IV). Of the 10 patients with facial nerve injury, four patients were started on steroids. Three of the seven patients with optic nerve injury were started on steroids. The patients were started on IV methylprednisolone (1 g IV once a day for 3 days and then switched over to oral prednisolone at 1 mg/kg/day for a maximum of 60 mg/day). In patients with low GCS score and lower CN palsy, these were administered through nasogastric tube feed. The dose was slowly tapered and stopped over a period of 20 days. In addition, conservative measures like eye padding at night, lubricant eye drops, and facial physiotherapy were initiated for all patients with facial nerve injury. Auditory assessment with pure tone audiogram (PTA) and tympanometry were done for patients with facial and vestibulocochlear nerve injury once their GCS scores improved.

Visual-evoked response (VER) may be useful to document nerve conduction and is helpful in the unresponsive patient suspected of having traumatic optic neuropathy.[5,15,16] This is especially true in possible bilateral cases where an afferent pupillary deficit may not be evident. An unresponsive patient with midline fractures and other than normal pupillary responses warrants a VEP monitoring.[6] Due to logistic reasons, VEP monitoring was done in patients with suspected bilateral visual pathway disorder.[6,10] Of the patients in whom VEP was undertaken, two of them showed severe optic neuropathy.

Of the 38 patients with CNI, only one patient (2.6%) required surgical intervention for optic nerve injury. CT of this patient noted fracture of the lesser wing of sphenoid, with posterior ethmoid sphenoid junction closely abutting the optic nerve at the region of orbital apex. Optic nerve decompression was done under cover of methylprednisolone. The rest of the patients were managed with conservative measures for CNI.

In case of lower CN palsy (noted in two patients), occipital condylar fractures (OCF) was noted in CT. Dysphagia was treated by feeding the patient with a nasogastric tube. Once swallowing was re-established at the end of 6 months in one patient, who had complete recovery, oral feeding was resumed.

Timing of CN Palsy

Puvanendran[17] in his study has reported that 82% had a delayed onset of presentation of CNI. This was in terms with our study where the incidence of delayed presentation was 73.7%. This difference was found to be statistically significant with a *p* value of < 0.001. The delay in onset varied from 3 days to 3 weeks in our study. Facial palsy came on between 3 to 7 days. This result was almost similar to the study by Patel et al and Puvanendran. Delayed CN palsy is possibly the result of edema (or bleeding into the facial canal), causing compression of the CN. Increasing size of the hematoma could press the CN over the rigid bony cage, ultimately cutting off the blood supply and causing ischemic damage to the nerve. When the pressure is mild, there would be only a neuropraxia or conduction block due to segmental demyelination. If the damage were more severe, there would be axonal damage with denervation.[17] An inflammatory reaction following the trauma, in and around the nerve, or a swelling of the nerve in the canal could also lead to ischemia, postulating the delayed onset of CN deficit.

CNI was noted in one-third of cases with skull base fractures as opposed to only 7.81% patients having CN palsy without skull base fractures. Coello et al has reported an incidence of CNI with skull base fractures to be 44%, which is significantly higher than that in our study.

Facial Nerve Injury in TBI Patients

Of all the CNs, the facial nerve is most susceptible to injury due to its complex course through the temporal bone, with proximity to structures such as the middle ear.[3] The HB 6-point scale of facial nerve function is the most commonly used standardized tool for assessing the degree of facial weakness.[7,8] To ensure interobserver reliability, the HB grading was performed by two authors in all patients. This scale is deficient, however, in terms of characterizing facial paralysis localized to one particular facial distribution.[18] Other scales include the Terzis–Noah, Burres–Fisch, Nottingham, and Sunnybrook.[19-22]

►**Table 4** shows the HB grading scale at presentation and at 6 months follow-up. HB grade II to IV was considered as incomplete palsy and complete palsy when the HB scale was V to VI.[7,8] Of the 10 patients in our study, traumatic facial palsy was predominantly incomplete (70%). Only three out of 10 patients (30%) had complete facial palsy. Our results corresponded to the findings of Patel et al[5] and Popovic et al[23] where the incidence of incomplete facial palsy was 69.5% and 62.9%, respectively.

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

Table 4 HB facial grading system at presentation and at 6 months follow-up

Case	Severity of head injury	Site of facial nerve injury	Onset of presentation	HB facial nerve grading system		Recovery at 6 months
				At presentation	At 6 months follow-up	
1	Severe	Right	Immediate	Grade IV	Grade II	Partial
2	Mild	Right	Delayed	Grade V	Grade I	Complete
3	Severe	Left	Delayed	Grade IV	Grade I	Complete
4	Mild	Right	Immediate	Grade III	Grade I	Complete
5	Severe	Right	Delayed	Grade IV	Grade I	Complete
6	Severe	Left	Immediate	Grade IV	Grade I	Complete
7	Moderate	Right	Immediate	Grade V	Grade III	Partial
8	Mild	Right	Delayed	Grade V	Grade V	No recovery
9	Moderate	Right	Delayed	Grade II	Grade I	Complete
10	Moderate	Left	Delayed	Grade IV	Grade I	Complete

Abbreviation: HB, House–Brackmann.

at 6 months with conservative treatment. Our results are in concurrence with the findings of Turel et al.[24] The authors suggested that delayed onset incomplete facial nerve injuries have good prognosis when conservative treatment was advocated. Similar findings were noted by Chang et al.[25] No surgical intervention was required for their patients with delayed facial paralysis.

Outcome of Cranial Nerve Injury (CNI) in TBI Patients

Our study showed that at 6 months, 16 (42%) patients had partial recovery, 12 (31%) patients had complete recovery, while 10 (26%) patients had no recovery at 6 months. The patients with partial recovery are expected to attain complete recovery in the coming weeks or months. Taking recovery as including both partial and complete recovery, 73.6% cases showed signs of recovery. Coello et al reported a 69% recovery of CN deficit, while Patel et al showed 60% recovery of CN deficit.

In our study, recovery was reported in 81.25% patients under the age of 60 years, while only 33% recovery was seen in the older age group of > 60 years. As much as 67.9% patients who had delayed onset of CN deficit showed recovery as opposed to only 32% recovery seen in patients with immediate presentation. Turner[26] reported a satisfactory recovery of CN function in 82% cases of delayed onset, while only 53% had recovery after immediate paralysis of CNs. Recovery was seen in 25% patients who were given steroids as opposed to only 75% recovery seen in patients who were not given steroids. This difference was not found to be statistically significant. There is, however, a bias as only the patients with optic nerve and facial nerve injury receive steroids; hence, this result may not hold significance.

Limitation

Since patients who expired in the study period were excluded from this study, exact assessment of the Glasgow outcome score (GOS) will have limitations, as death is also included in the outcome score.

Conclusion

Significant percentage of patients present with delayed presentation of CNI. Hence, meticulous neurological examination of all CNs should be conducted in all TBI patients on follow-up.

Authors' Contributions

J.C.V.–Principal investigator; conducting the study, preparing the manuscript, and collecting data.
N.B.–Preparing the manuscript and analyzing and collecting data.
R.K.–Coinvestigator; defining intellectual content and reviewing manuscript.
J.P.A.–Coinvestigator; defining intellectual content and reviewing manuscript.
J.M.–Coinvestigator; reviewing manuscript.

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Conflict of Interest

None declared.

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