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A prospective study on the incidence and outcome of cranial nerve injuries in patients with traumatic brain injuries

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ABSTRACT

Background: The worldwide mortality and morbidity rates are highest for traumatic brain injury (TBI), which frequently involves damage to the cranial nerves (CNs). The occurrence of CN injury (CNI) in craniocerebral trauma ranges from 5 to 23%. **Aims and Objectives:** The objective of this study is to evaluate the occurrence of CNI in TBI patients within our population and determine the association between CN involvement and the severity of head injuries. In addition, we aim to assess the outcomes of patients who experience CNI in cases of head trauma. **Materials and Methods:** In our institution, a prospective observational analytical study was conducted from July 2022 to June 2023. The study included 100 patients aged over 1 year who had sustained head injuries. These patients were followed up for a period of 6 months. **Results:** Our study included a total of 100 patients, revealing that the highest number of head injuries occurred within the age range of 20–60 years, with an average age of 46 years. Among the study population, males accounted for 74%. Of these patients, 66 had mild head injuries, 22 had moderate head injuries, and 12 were admitted with severe head injuries. We discovered that 15% of TBI patients experienced CNIs. Specifically, five patients had a single CN palsy, while three patients had multiple CN palsies. Road traffic accidents accounted for 78% of the cases, making them the most common cause of injury, while low-velocity injuries only made up 22% of the cases. The facial nerve was the CN most frequently affected, followed by the olfactory nerve, optic nerve, and vestibulocochlear nerve. Among the 15 patients with CNI, seven had delayed symptoms, whereas eight patients presented with immediate symptoms. **Conclusion:** A substantial proportion of patients experience a delayed onset of CNI symptoms. Therefore, it is essential to perform a comprehensive neurological examination of all CNs during follow-up for all TBI patients.

Key words: Head injury; Cranial nerve injury; Traumatic brain injury

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INTRODUCTION

Traumatic brain injury (TBI) is globally recognized as the primary cause of both mortality and morbidity.[1] Cranial nerve injuries (CNIs) frequently occur in association with TBI.[2] The incidence of CNI in craniocerebral trauma ranges from 5 to 23%.[3] An initial assessment, consisting of a thorough examination of history, clinical findings, and neurological evaluation, effectively evaluates the individual's level of consciousness, cortical functioning, and hemodynamic status. Furthermore, a prompt assessment of ocular movements, pupil reactions, facial symmetry, and laryngeal function provides crucial insights into the functional integrity of the cranial nerves (CNs).

Aims and objectives

The objective of this study is to evaluate the occurrence of CNI in TBI patients within our population and determine the association between CN involvement and the severity of head injuries. In addition, we aim to assess the outcomes of patients who experience CNI in cases of head trauma.

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MATERIALS AND METHODS

We conducted a prospective observational analytical study at our institution from July 2022 to June 2023 to investigate various aspects related to head injuries. The study included a total of 100 patients aged over 1 year who had sustained head injuries, and they were followed up for a period of 6 months. All patients underwent thorough clinical and neurological examinations. Computed tomography (CT) scans using brain and bone windows were performed when specific risk factors were present, such as posttraumatic amnesia, loss of consciousness, posttraumatic seizures, headache, vomiting, focal neurological deficit, skull fracture, coagulopathy, or anticoagulation therapy.

The assessment of CNs was conducted promptly using the following methods: examination for nasal bleed, cerebrospinal fluid (CSF) rhinorrhea, ptosis, proptosis, complaints of double vision, finger counting in each eye separately, poor eye closure, facial weakness, assessment of ocular movements, corneal reflex, asymmetry, pupillary light reflex for afferent pupillary defect, hearing defect, ear bleed, CSF otorrhea, hemotympanum, rupture of the tympanic membrane, hoarseness of voice, inability to cough, and indirect laryngoscopy for vocal cord movements. Difficulty in swallowing and articulating words was also evaluated.

Special investigations, such as orbital, anterior fossa, petrous bone, and posterior fossa CT scans, CT cisternograms, audiograms, visual evoked potentials (VEPs), and MRI scans, were performed under specific circumstances, such as visual defects, CSF rhinorrhea, or hearing defects.

The CT scans helped differentiate between three types of traumatic injuries: No lesion, skull fractures, and parenchymal brain injuries such as contusions, subdural hematomas, epidural hematomas, and subarachnoid hemorrhages.

The relative afferent pupillary defect was employed as a dependable diagnostic test for detecting unilateral optic nerve injury, even in patients with moderate-to-severe head injuries.[4] A visual acuity evaluation was carried out in responsive patients, whereas a flash VEP was utilized in unresponsive patients.

The House–Brackmann (HB) 6-point scale was utilized to grade facial nerve injuries,[5] categorizing the severity of facial palsy as low grade (HB 2-3), intermediate (HB 4), or high grade (HB 5-6).[6] Incomplete palsy was considered when the HB scale ranged from II to IV, and complete palsy was defined as HB scales V to VI.

Clinical recovery from CN palsy was classified into three grades: No recovery, partial recovery, or complete recovery.[7] Recovery was assessed throughout the follow-up period and correlated with associated radiological findings and specific treatments, including surgical decompression and the administration of steroids.

The overall outcomes of TBI patients with CNIs were studied and compared with those of patients without CN deficits.

RESULTS

Following the application of the inclusion and exclusion criteria, our study included a total of 100 patients. We observed a higher incidence of head injuries among individuals aged 20–60 years, with a mean age of 44 years (with a standard deviation of 12.6). Males accounted for 74% of the study population. Table 1 shows the gender distribution and Figure 1 shows the age distribution. Among the patients, 66 had mild head injuries, 22 had moderate head injuries, and 12 were admitted with severe head injuries. Figure 2 shows head injury type distribution.

The incidence of CNI in TBI patients was found to be 15%. Table 2 shows the distribution of CNIs in our study population. Five patients experienced a single CN palsy, while three patients had multiple CN palsies. Road traffic accidents (RTA) were the most common mode of injury, accounting for 78% of the cases, whereas low-velocity injuries were responsible for 22% of the cases. The facial nerve was the most commonly involved CN, followed by the olfactory nerve, optic nerve, and vestibulocochlear nerve. Out of the 15 patients with CNIs, 7 presented with delayed symptoms, while 8 patients had an immediate presentation. Table 2 and Figure 3 show for more details.

Table 1: Gender Distribution

Gender	No. of patients
Male	74
Female	26

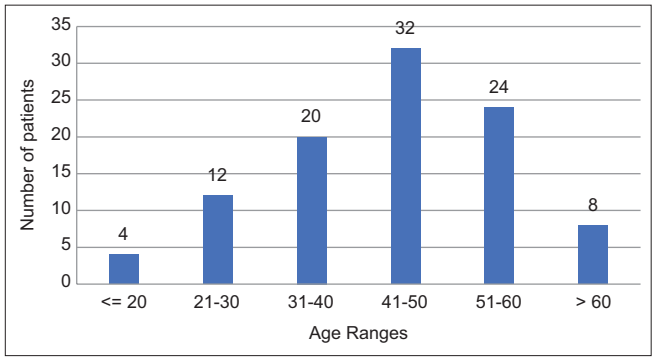

Figure 1: Age distribution

DISCUSSION

With the ongoing increase in high-velocity accidents and incidents of violence in recent decades, the issue of acute head trauma has become critically important. The immediate and long-term consequences of trauma can result in severe disabilities, with CNIs playing a significant role in these disabilities. In our study, out of the 100 patients included, 74 were male and 26 were female. This male predominance aligns with the findings of Equabal's study,[8] which reported an 83% male preponderance. Among the head injury cases requiring hospital admission, severe head trauma resulting from RTA was the primary cause. The Traumatic Coma Data Bank prospective study[9] also revealed that motor vehicle crashes accounted for 55% of the injuries, which is consistent with our study findings.

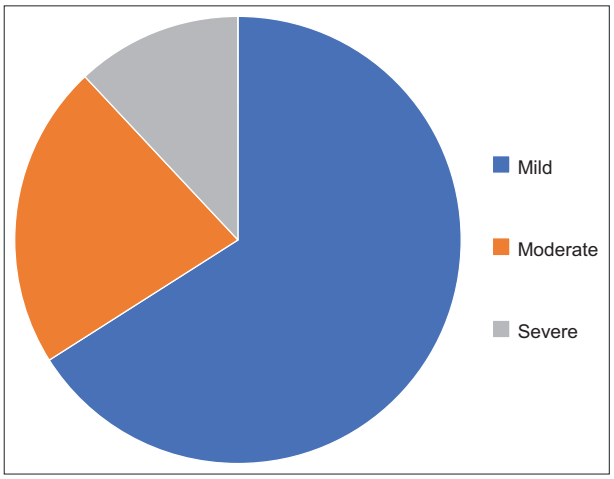

Figure 2: Type of head injuries

RTA remains the most common cause of TBI across all age groups. Our results corroborate other studies conducted by Borkar et al.,[10] Equabal et al.,[8] and Yattoo and Tabish.[11] After the initial resuscitation, all head injury patients underwent close monitoring for CNIs. A thorough neurological examination of all 12 pairs of CNs was conducted to detect any deficits. Patients were examined upon admission, at discharge, and every month during the 6-month follow-up period to minimize the possibility of missing CNIs at the time of presentation. This approach allowed us to identify both immediate and delayed presentations of CN involvement. However, it is worth noting that the involvement of the olfactory nerve and vestibulocochlear nerve can be easily missed. In our study population, of the two patients with olfactory nerve injuries, one had a mild head injury, while the other had a severe head injury.

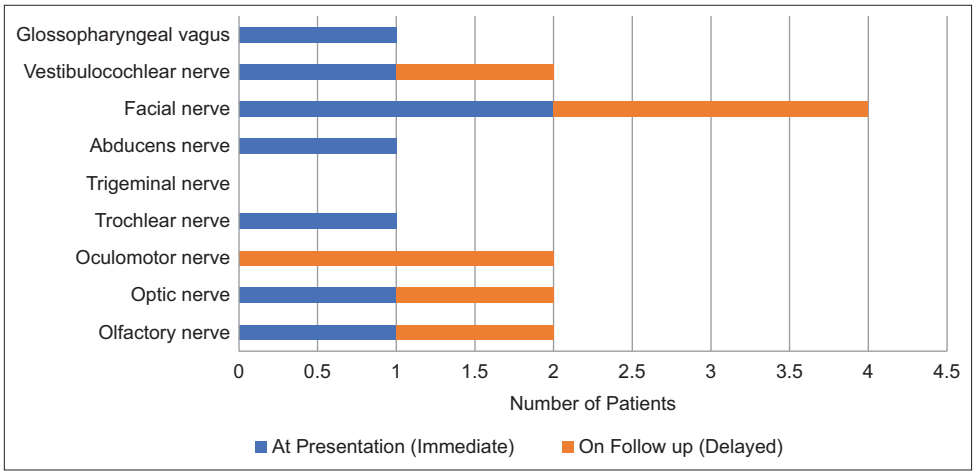

Figure 3: Onset of cranial nerve injuries

Table 2: Distribution of posttraumatic cranial nerves involved

Cranial nerve injured	Number of patients	At presentation (Immediate)	On follow-up (Delayed)
Olfactory nerve	2	1	1
Optic nerve	2	1	1
Oculomotor nerve	2	0	2
Trochlear nerve	1	1	0
Trigeminal nerve	0	0	0
Abducens nerve	1	1	0
Facial nerve	4	2	2
Vestibulocochlear nerve	2	1	1
Glossopharyngeal/vagus	1	1	0

Similarly, one out of two patients with vestibulocochlear nerve injuries had a mild head injury, while the other had a severe head injury. The prospective nature of our study further enhances the reliability of patient examinations, reducing the chances of missing CNIs.

CNIs were observed in 15 out of 100 TBI patients. Among these 15 patients, 12 experienced RTA-related injuries, and the facial nerve was the most commonly injured CN, affecting four patients. In two patients, injuries were identified in the olfactory nerve, optic nerve, and vestibulocochlear nerve. Our results differ from many published studies where anosmia was the most common manifestation of post-traumatic CNIs. However, the specific patient population presenting at our institution may explain this disparity in results. No patients in our study exhibited trigeminal nerve involvement. The incidence of CNIs was found to be higher in patients with severe head injuries, as indicated by the initial Glasgow Coma Scale score. Approximately 30% of patients with severe head injuries presented with CN deficits, compared to 10% of patients with mild-to-moderate head injuries.

VEP monitoring was performed in patients with suspected bilateral visual pathway disorders. Among the patients who underwent VEP, two exhibited severe optic neuropathy. Out of the 15 patients with CNIs, only one required surgical intervention for optic nerve injury, specifically optic nerve decompression. The remaining patients were managed conservatively. In cases of lower CN palsy, CT scans revealed occipital condylar fractures (OCF). Dysphagia was addressed by providing patients with nasogastric tube feeding. The timing of CN palsy onset was predominantly delayed, consistent with the findings of Puvanendran's study,[12] which reported an 82% delay in onset. In our study, the incidence of delayed presentations was 72%, a statistically significant difference with a $P<0.001$. The delay in onset varied from 3 days to 3 weeks, with facial palsy typically manifesting between 3 and 7 days. These results align closely with the findings of studies conducted by Patel et al., and Puvanendran et al. Delayed CN palsy may be attributed to edema causing compression of the nerves or increasing hematoma size exerting pressure on the nerves within the rigid bony cage, leading to ischemic nerve damage. When the pressure is mild, it may result in neuropraxia or conduction block due to segmental demyelination. More severe damage can lead to axonal damage and denervation.

Among all the CNs, the facial nerve is particularly vulnerable to injury due to its complex course through the temporal bone, in close proximity to structures such as the middle ear. The HB 6-point scale is the most commonly used standardized tool for assessing the degree of facial weakness in cases of facial nerve injury.[13] Other scales, such as the Terzis and Noah, Burres and Fisch, Nottingham, and Sunnybrook scales,[14-17] are also utilized. Table 3 provides the HB grading scale at presentation and at the 6-month follow-up. Incomplete facial palsy was more prevalent, with only one out of four patients exhibiting complete facial palsy. These findings align with Popović et al.'s study,[18] which reported incidence rates of 69.5% and 62.9% for incomplete facial palsy. The prognosis and management of delayed-onset facial nerve paralysis were compared to those of immediate-onset cases. At the 6-month follow-up, five out of six patients with delayed-onset facial nerve paralysis achieved complete recovery with conservative treatment. Our results are consistent with those of Turel et al.,[19] where no surgical intervention was required for patients with delayed facial paralysis.

Regarding the outcome of CNIs in TBI patients, our study showed that at 6 months, one patient exhibited partial recovery, two patients achieved complete recovery, and one patient showed no recovery. Patients with partial recovery are expected to attain complete recovery in the following weeks or months. When considering both partial and complete recovery, 75% of the cases exhibited signs of recovery. Steroid treatment was associated with recovery in 25% of the patients, while 75% of patients who did not receive steroids showed recovery. However, this difference was not statistically significant.

Limitations of the study

Exclusions were made for patients who passed away during the study duration. In addition, the diagnostic criteria for CNIs lack clarity and vary based on evaluations conducted at individual hospitals. Furthermore, in cases of impaired

Table 3: HB facial grading system at presentation and at 6-month follow-up

Case	Severity of head injury	Site of facial nerve injury	Onset of presentation	HB facial nerve grading system at presentation	HB facial nerve grading system at 6-month follow-up	Recovery at 6 months
1	Severe	Left	Immediate	Grade 4	Grade 2	Partial
2	Severe	Right	Delayed	Grade 4	Grade 1	Complete
3	Moderate	Left	Delayed	Grade 2	Grade 1	Complete
4	Mild	Left	Immediate	Grade 2	Grade 2	No recovery

HB: House–Brackmann

consciousness, such as severe head trauma, there is a possibility of overlooking CN damage.

CONCLUSION

A significant proportion of patients exhibits delayed onset of CNI symptoms. Therefore, it is crucial to perform thorough neurological examinations of all CNs in follow-up assessments for all TBI patients.

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ETHICAL COMMITTEE APPROVAL

Letter No. 1012, dated March 13, 2022. GRMC/IEC/2022/1012.

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NK- Definition of intellectual content, literature survey, prepared first draft of manuscript, implementation of study protocol, data collection, data analysis, manuscript preparation and submission of article; **VKK-** Concept, design, clinical protocol, manuscript preparation, editing and revision; **AS-** Review manuscript, preparation of tables and graphs, coordination and manuscript revision.

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