ORIGINAL RESEARCH PAPER

Volume-8 | Issue-1 | January-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

ASSESMENT OF CRANIAL NERVE DEFICITS IN ACUTE HEAD INJURY PATIENTS:A PROSPECTIVE STUDY IN A TERTIARY CARE HOSPITAL

Neurosurgery

Dr Prabhat Kumar Senior resident, Department of Neurosurgery ,Vardhman Mahavir Medical College &Safdarjung Hospital,New Delhi

Dr Amit Dagar* Assistant Professor ,Department of Neurosurgery, Vardhman Mahavir Medical College &Safdarjung Hospital,New Delhi *Corresponding Author

Dr Karam Chand Sharma Professor, Department of Neurosurgery, Vardhman Mahavir Medical College &Safdarjung Hospital,New Delhi

Dr K B Shankar Professor and Head of Department of Neurosurgery, Vardhman Mahavir Medical College &Safdarjung Hospital,New Delhi

Dr Vivek Yadav Assistant Professor ,Department of Neurosurgery, Vardhman Mahavir Medical College &Safdarjung Hospital,New Delhi

ABSTRACT

BACKGROUND: Cranial nerve injury is common with TBI . It may result in significant consequences to the individual , which may be further compounded by the deficits caused by cortical injury.

METHODS: 300 patients with head injury were studied. Neurological and radiological examinations were performed . Patients were followed up for 6 months. Four check-ups were conducted during this period; on day 7, 1st,3rd and 6th month.

RESULTS: Out of 300 patients evaluated with head injury , 51 had cranial nerve palsies .Most common age group affected was third decade .RTA was the most common injury mode .Facial nerve was the most common and lower cranial nerves were the least commonly affected.

CONCLUSION: Cranial nerve injury is common with traumatic brain injury .All nerves achieve some amount of functional recovery with time. In patients with TBI, it is essential to identify cranial nerve injuries using physical examination and ancillary tests.

KEYWORDS

TBI:Traumatic Brain Injury GCS:Glasgow Coma Scale RTA:Road Traffic Accident

INTRODUCTION

Head injury is recognised as a major health problem and is a frequent cause of death and disability in young .[1] Urbanisation has contributed to rise in incidence of head injury in India.[2,3]

Injury to the cranial nerves is not uncommon with traumatic brain injury (TBI) and has even been seen to occur in TBI with a Glasgow coma scale[4] GCS of 14-15.

Perseverance is required for the diagnosis of cranial nerve injury in the presence of severe head injury.[5] Cranial nerves III, VI, and VII damage can be diagnosed even in the presence of coma.[6] Diagnosis of trochlear nerve injury requires a high level of patient's cooperation.[7]. Skull base fracture can be overlooked while treating other life-threatening injuries[8]. The incidence of cranial nerve injuries varies between 5 and 23 % in cranio-cerebral trauma .[9]

Trauma to the optic nerve(II) and ocular motor nerves(III,IV,VI) is intermediate in frequency, whereas the trigeminal nerve (V) and the lower cranial nerves(IX,X,XI,XII) are rarely injured.[10]

REVIEW OF LITERATURE

The olfactory cranial nerve[11] is reported to be the most often injured, followed next in frequency by the facial and vestibulo-cochlear nerves, less commonly the optic and oculomotor nerves, and rarely the trigeminal and lower cranial nerves.

Pupillary, oculo-vestibular, and oculo-caloric reflexes have been shown to have strong prognostic indications of mortality[12-16] and morbidity.[17]

Surgical decompression significantly increases the chance of recovery in these patients[18].

OLFACTORY NERVE

The olfactory system is the only sensory pathway that does not project to the thalamus before making its connections in the cortex.[19]

Costanzo and Becker[20] reported the incidence of 19.4% with moderate TBI and 24.5% with severe TBI.To examine its function, stimulants such as organic solvents or ammonia should be avoided as they may also stimulate cranial nerve V [21]. Partial recovery is thought to occur due to the neurofibrillary regrowth and central adaptation[22]. Costanzo and Becker [20] observed 33% recovery. When recovery occurs, it is usually noticed within the first 6 months and complete by 1 year after injury[21].

OPTIC NERVE

Its injury occurs in around 5% of patients with TBI [23]. Optic nerve palsy may occur with the globe injury[24]. It is subject to shearing forces as the brain and orbital contents are free to move, while the nerve is not [25]. It is injured most frequently in intra-canalicular portion [26].

Steroids and /or optic canal decompression can be of benefit [27-29].Other studies have shown that neither treatment improves visual outcome [30,31].

Injury to the cornea or anterior chamber may cause monocular diplopia [32]. The potential presence of occipital seizures should also be considered as a cause of blindness [33].

OCULOMOTOR NERVE

A palsy of this nerve can be caused by various brain disorders . Isolated oculomotor nerve palsy due to TBI alone are not unusual, occurring in 17% of individuals [34]. Patients with complete oculomotor palsy have higher incidence of traumatic subarachnoid haemorrhage (71%) or skull fractures (57%) [35].

The chances of complete recovery are poor [35], with 40% reported to have complete recovery [32]. Pleoptics have not proven to be very effective [36]. Strabismus surgery should be delayed 6-9 months post-injury, as some of patients tends to recover spontaneously [37]. The procedure has limited success. However, one study showed successful alignment in 81% of adults[38].

TROCHLEAR NERVE

Injury of the trochlear nerve may result from various causes. TBI is the most common cause. Trochlear nerve injury is reported to occur in 0.2%-1.4% of patients with TBI[32].

TRIGEMINAL NERVE

The incidence of trigeminal nerve injury after TBI is 1.4%-2%[9].
The roots and /or the gasserian ganglion may be damaged in transverse skull fractures [39] .

Stretch injuries damages the nerves at fixed attachments at points of sharp angulation [9]. Patient's dysesthesia responds to various anticonvulsants [40].

ABDUCENS NERVE

Abducens palsy is one of the most frequently reported ocular motor palsies [41]. The abducens palsy can serve as an early indicator of increased intracranial pressure due to various causes [42]. Injury to the abducens nerve in association with traumatic brain injury is reported to occur in 0.4%-4.1%[32].

FACIAL NERVE

Injury to the facial nerve is the second most common[11]. Temporal bone fractures are mostly longitudinal [22]. Transverse fractures result in complete cranial nerve VII paralysis in around 30%-50% of cases due to tearing of the nerve[21,22].

In longitudinal fracture ,corticosteroids should be initiated and facial nerve decompression considered [11]. The nerve is usually intact and mostly recovers in 6-8 weeks [21,22].

In case of complete facial nerve disruption, surgical techniques may be helpful. Nerve –muscle pedicle grafts from the contralateral side offers rapid return of nerve function without compromising neck function [43].

VESTIBULOCOCHLEAR NERVE

Sensorineural hearing loss occurs with TBI at an incidence of 85% and 71% in the absence of temporal bone fracture[44]. Transverse fractures have been reported to result in sensorineural hearing loss in 100% of cases[45]. Longitudinal fractures result in conductive hearing loss or mixed hearing loss[45]. Spontaneous recovery is reported in 80% of patient with conductive hearing loss occurring with temporal bone fractures[46]. Surgical repair is associated with excellent results in conductive hearing loss[45].

Medications commonly used for vestibular problems are generally not helpful and discouraged due to cognitive side effects and prevention of central adaptation[21].

Unilateral hearing loss may be improved using CROS (contralateral routing of signal) type hearing aid [21].

GLOSSOPHARYNGEAL NERVE

The incidence of glossopharyngeal dysfunction after trauma is 0.5-1%.Injury to the glossopharyngeal nerve usually involves the vagus nerve as well.

VAGUS NERVE

The incidence of vagal involvement in TBI 0.05-0.16%[9].
Pharyngeal exercises may improve dysarthria in individuals with mild dysfunction [47]. Surgical procedures such as thyroplasty and arytenoids adduction have been seen to benefit those with high vagal lesions[48].

SPINAL ACCESSORY NERVE

Trauma is the most common cause of spinal accessory nerve dysfunction. Aggressive physical therapy should be initiated as soon as possible.

HYPOGLOSSAL NERVE

Injury to the hypoglossal nerve occurs with penetrating wounds to the neck and submental region.

Although much is known about the mechanism of cranial nerve injuries in TBI, knowledge that seems to be lacking in this area is that of appropriate treatment. An important direction for future research would be to develop efficacious surgical techniques .

AIMS AND OBJECTIVES.

1. To study the incidence of cranial nerve deficits in mild to moderate acute head injury.
2. To observe the relationship between the circumstances and severity of closed head injuries and cranial nerve deficits.
3. To compare the clinical course and outcome of cranial nerve injuries in head injury patients on admission, at day 7 , at 1, 3 and 6 months.

MATERIALS AND METHODS

1. Study area: Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi.
2. Study population: admitted and seen patients of head injury at Safdarjung hospital.
3. Study period: two years.

Inclusion criteria.

All head injury patients admitted and seen in Safdarjung hospital with GCS Score of 9- 15

Exclusion criteria.

1 .Patients of poly trauma with life threatening systemic injuries.
2. Severe head injury patients with GCS </= 8
3. Non traumatic cranial nerve palsies.
4. Patients less than 18 years of age.
5. Patients not consenting for the study.

Methods.

In our study we studied 300 patients affected by mild and moderate head injury between July 2015 to December 2016. Detailed neurological examinations were performed in all patients. NCCT using brain and bone windows was performed when at least one of the following risk factors was present- posttraumatic amnesia, loss of consciousness, post traumatic seizure, headache, vomiting, focal neurological deficit, skull fracture, coagulopathy, anticoagulant therapy and in patients older than 60 years of age. Every patient with a neurological deficit, CT scan abnormalities, or persistent cerebral commotion was admitted to our neurological unit for clinical monitoring and to receive specific therapy. Anterior fossa, petrous bone, and posterior fossa CT scans; audiograms; visual evoked potentials monitoring; and other investigation were conducted in specific circumstances. Visual evoked potential monitoring was undertaken when a visual pathway disorder was suspected and pure tone audiogram allowed us to investigate the peripheral auditory pathway. From the CT scans parenchymal injury was classified into 9 types i.e frontal, parietal, temporal, occipital, cerebellar, multiple(involving more than one lobe or brain stem), brain stem, diffuse subarachnoid haemorrhage and no evidence of parenchymal injury. Bony injuries were classified into anterior, middle and posterior cranial fossa fractures; frontal, parietal, temporal and occipital fractures; temporal fractures sub classified into longitudinal, transverse and mixed fractures; orbital fractures into roof, floor, medial and lateral wall fractures. Patients were followed up for 6 months. Four check-ups were conducted during this period; on day 7, 1st,3rd and 6th month.

Statistical Analysis Method

The analysis included profiling of patients on different demographic, clinical and laboratory parameters. Quantitative data were presented in terms of means and standard deviation. Qualitative/categorical data were presented as absolute numbers and proportions. Cross tables were generated and chi square test was used for testing of significance. All statistical tests were two-sided, and P values less than 0.05 was considered as statistically significant. All statistical analysis were performed using SPSS, version 24.0.

RESULTS AND OBSERVATION

Out of 300 patients evaluated with head injury ,51 had cranial nerve injuries.There gender distribution is shown in the table .

Table 1

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Gender				0.363
Male	33 (64.7)	144 (57.8)	177 (59)	
Female	18 (35.3)	105 (42.2)	123 (41)	

Age distribution of patients with head injury and cranial nerve injury.

Table 2

	Overall (n=300)		Cranial Nerve Injury (n=51)	
	Number	Percentage	Number	Percentage
< = 20	15	5.0	6	11.8
21 - 30	61	20.3	19	37.3
31 - 40	97	32.3	17	33.3
41 - 50	74	24.7	7	13.7
51 - 60	38	12.7	2	3.9
> 60	15	5.0	0	0.0
Total	300	100.0	51	100.0

Mode of injury to the patients with head injury and cranial nerve injury

Table 3

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Mode of injury				0.096
RTA	42 (82.4)	167 (67.1)	209 (69.7)	
Fall from height	8 (15.7)	72 (28.9)	80 (26.7)	
Assault	1 (2)	10 (4)	11 (3.7)	

192 patients had suffered minor head injury whereas 108 patients suffered moderate head injury.

Loss of consciousness in patients with head injury and cranial nerve injury.

Table 4

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
LOC				0.226
1 present	51 (100)	242 (97.2)	293 (97.7)	
2 absent	0 (0)	7 (2.8)	7 (2.3)	

Seizure episode in patients with head injury and traumatic cranial nerve injury.

Table 5

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Seizure				0.271
1 present	14 (27.5)	51 (20.5)	65 (21.7)	
2 absent	37 (72.5)	198 (79.5)	235 8.3)	

Nasal bleed in patients with head injury and cranial nerve injury was statistically significant .

Table 6

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Nasal bleed				<0.0001
1 yes	30 (58.8)	67 (26.9)	97 (32.3)	
2 no	21 (41.2)	182 (73.1)	203 (67.7)	

.Ear bleed in patients with head injury and cranial nerve injury .41.2% patients out of 51 had ear bleed which was statistically significant .

Table 7

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Ear bleed				0.041
1 yes	21 (41.2)	67 (26.9)	88 (29.3)	
2 no	30 (58.8)	182 (73.1)	21 0.7)	

CSF rhinorrhea in patients with head injury and cranial nerve injury .

Table 8

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
CSF rhinorrhea				
1 yes	5 (9.8)	24 (9.6)	29 (9.7)	0.971
2 no	46 (90.2)	225 (90.4)	271 0.3)	

CSF otorrhoea in patients with head injury and cranial nerve injury was statistically relevant in cranial nerve injuries.

Table 9

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
CSF otorrhoea				0.005
1 yes	13 (25.5)	27 (10.8)	40 (13.3)	
2 no	38 (74.5)	222 (89.2)	260 6.7)	

Vertigo in patients with head injury and cranial nerve injury was statistically significant .

Table 10

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Vertigo				0.012
1 yes	8 (15.7)	14 (5.6)	22 (7.3)	
2 no	43 (84.3)	235 (94.4)	278 2.7)	

Ataxia in patients with head injury and cranial nerve injury.

Table 11

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Ataxia				0.602
1 present	5 (9.8)	19 (7.6)	24 (8)	
2 absent	46 (90.2)	230 (92.4)	2762)	

Parenchymal injuries and their location in patients with head injury and cranial nerve injury patients were statistically significant .

Table 12

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Parenchymal injury				<0.0001
1 frontal	16 (31.4)	13 (5.2)	29 (9.7)	
2 parietal	6 (11.8)	49 (19.7)	55 (18.3)	
3 temporal	9 (17.6)	53 (21.3)	62 (20.7)	
4 occipital	2 (3.9)	38 (15.3)	40 (13.3)	
5 cerebellar	0 (0)	32 (12.9)	32 (10.7)	
6 diffuse	13 (25.5)	27 (10.8)	40 (13.3)	
7 brainstem	0 (0)	19 (7.6)	19 (6.3)	
8 no parenchymal injury	3 (5.9)	11 (4.4)	14 (4.7)	
9 Sub arachnoid haemorrhage	2 (3.9)	7 (2.8)	7 (3)	

Skull fractures in patients with head injury and cranial nerve injury were statistically significant.

Table 13

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Skull fractures				
1 frontal	21 (41.2)	36 (14.5)	57 (19)	
2 temporal	17 (33.3)	60 (24.1)	77 (25.7)	
3 parietal	3 (5.9)	43 (17.3)	46 (15.3)	
5 4 occipital	1 (2)	29 (11.6)	30 (10)	
5 no fractures	9 (17.6)	81 (32.5)	90 30)	<0.0001

Temporal bone fracture in patients with head injury and cranial nerve injury .

Table 14

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Temporal bone fracture				0.371
1 longitudinal	10 (19.6)	35 (14.1)	45 (15)	
2 transverse	4 (7.8)	15 (6)	19 (6.3)	
3 mixed	4 (7.8)	10 (4)	14 (4.7)	
4 none	33 (64.7)	189 (75.9)	222 4)	

Skull base fracture in patients with head injury and cranial nerve palsy were significant statistically.

Table 15

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Skull base fracture				<0.0001
1 anterior	16 (31.4)	55 (22.1)	71 (23.7)	
2 middle	1 (2)	68 (27.3)	69 (23)	
3 posterior	1 (2)	23 (9.2)	24 (8)	
4 none	33 (64.7)	103 (41.4)	136 (45.3)	

.Facial bones fracture in patients with head injury and traumatic cranial nerve injury were significant statistically.

Table 16

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Facial fracture				• 0.002
1 le fort 1	0 (0)	13 (5.2)	13 (4.3)	
2 le fort 2	3 (5.9)	15 (6)	18 (6)	
3 le fort 3	7 (13.7)	8 (3.2)	15 (5)	
4 none	40 (78.4)	213 (85.5)	253 (84.3)	
5 not classified	1 (2)	0 (0)		

Orbital fracture in patients with head injury and cranial nerve injury was significant statistically.

Table 17

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Orbital fracture				<0.0001
1 roof	7 (13.7)	9 (3.6)	16 (5.3)	
2 floor	3 (5.9)	9 (3.6)	12 (4)	
3 medial wall	8 (15.7)	14 (5.6)	22 (7.3)	
4 lateral wall	2 (3.9)	0 (0)	2 (0.7)	
5 none	31 (60.8)	217 (87.1)	248 (82.7)	

The incidence of cranial nerve injuries was 17% in our study involving 300 patients.

Distribution of cranial nerve injury. Total number of patients with cranial nerve injury was 51. The incidence of cranial nerve palsies was 79.

Table 18

Cranial Nerve Type	Number of injury
CN I	11
CN II	12
CN III	12
CN IV	0
CN V	6
CN VI	8
CN VII	17
CN VIII	11
CN IX	1
CN X	1
CN XI	0
CN XII	0

Anosmia in patients with head injury and cranial nerve injury .

Table 19

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Anosmia				<0.0001
1 present	11 (21.56)	0 (0)	11 (3.6)	
2 absent	40 (78.43)	249 (100)	2896.4)	

Visual acuity in patients with head injury and cranial nerve injury .

Table 20

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Visual acuity				<0.0001
1 normal	39 (76.5)	249 (100)	288 (96)	
2 reduced	4 (7.8)	0 (0)	4 (1.3)	
3 PL negative	8 (15.7)	0 (0)	7 (2.7)	

VEP response in patients with head injury and optic nerve injury . VEP abnormalities were significantly correlated.

Table 21

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
VEP				<0.0001
2 delayed	6 (11.8)	0 (0)	6 (2)	
3 not assessed	40 (78.4)	249 (100)	289 6.3)	

Resting gaze abnormality in patients with head injury and cranial nerve injury was significantly correlated.

Table 22

	Yes (n=51)	No (n=249)	Total (n=300)	
Resting gaze				<0.0001
1 normal	46 (90.2)	249 (100)	295 (98.3)	
2 abnormal	5 (9.8)	0 (0)		

Presence of diplopia in patients with head injury and cranial nerve injury was significant statistically.

Table 23

	Yes (n=51)	No (n=249)	Total (n=300)	p-value
Diplopia				<0.0001
1 absent	47 (92.2)	249 (100)	296 (98.7)	
2 horizontal	3 (5.9)	0 (0)	3 (1)	
3 vertical	1 (2)	0 (0)	1 (0.3)	

3%patients presenting with head injury had sensory deficits in V1 segment of 5th nerve .In the nerve injury group this was 17.6%.

19.6% patients with cranial nerve injuries has House and Brackmann grade 5 injury;11.8% had grade4; 3.9% had grade 6 and 2% had grade 3 injury. There were no grade 1 and grade 2 injuries reported in our study. Pure tone audiogram abnormality in patients with impaired hearing was significant statistically .

Table 24

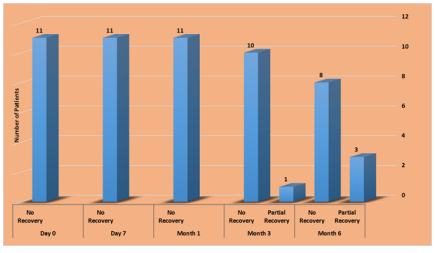
	Yes (n=51)	No (n=249)	Total (n=300)	p-value
PTA				<0.0001
1 conductive	4 (7.8)	0 (0)	4 (1.3)	
2 sensorineural	3 (5.9)	0 (0)	3 (1)	
3 mixed	1 (2)	0 (0)	1 (0.3)	
5 not assesed	43 (84.3)	249 (100)	292 7.3)	

0.3% patients i.e. one patient presented with complaints of nasal regurgitation, palatal dysarthria and was found to have palatal paralysis on the affected side and deviation of uvula. The p value of each is 0.027 and significant statistically.

In our study 5.7% patients had suffered from cranial nerve VII involvement followed by 4% for both cranial nerve II and cranial nerve III, 3.7% for both cranial nerve I and cranial nerve VIII, 2% for cranial nerve V, 0.3% for cranial nerve IX and cranial nerve X. No patients had traumatic cranial nerve injuries of cranial nerve IV,XI and XII.

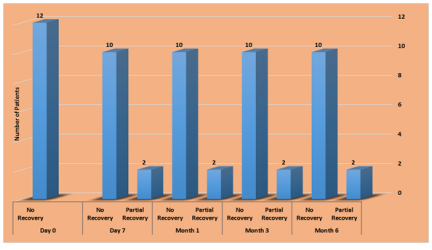
Out of 11 patients with CN I traumatic injury, 8 patients did not show any recovery and 3 patients showed partial recovery at the end of 6 months, one patient showed partial recovery at the end of 3 months which was static at the end of 6 months.

Figure 1



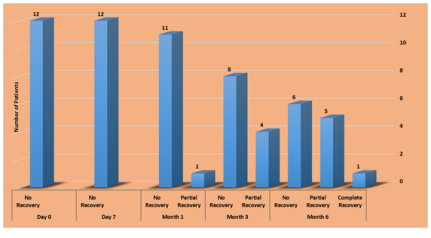
Out of 12 patients with unilateral CN II injury,2 showed partial improvement by the end of 7 days which was static over a 6 month follow up. Remaining 10 patients did not show any sign of recovery at the end of 6 months.

Figure 2



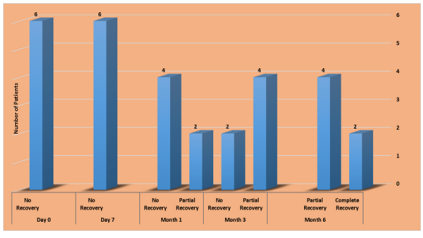
Out of 12 patients with CN III nerve injury, 1 patient recovered completely at the end of 6 months, 5 patients showed partial recovery whereas 6 patients did not show any recovery.

Figure 3



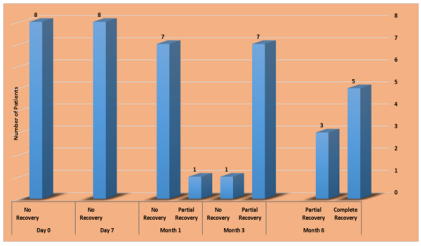
Out of 6 patients with CN V nerve injury, 2 patients had complete recovery at the end of 6 months whereas rest 4 showed partial recovery.

Figure 4



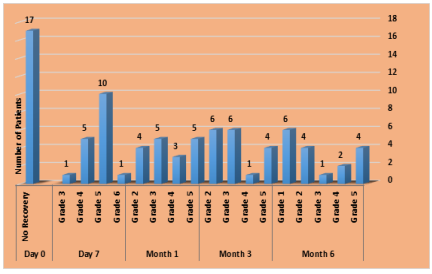
Out of 8 patients with CN VI injury, 5 patients showed complete recovery at the end of 6 months and in the rest 3 patients recovery was partial.

Figure 5



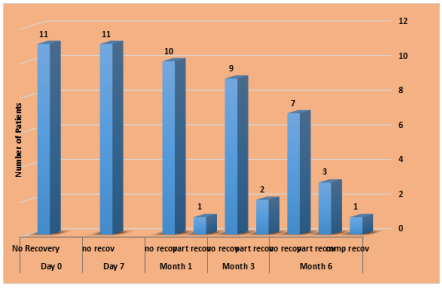
Out of 17 patient with CN VII injury, 10 had grade 5(House and Brackmann), 5 had grade 4, 1 had grade 6 and 1 had grade 3 palsy. At 6 month follow up 4 patients were having grade 5, 2 patients grade 2, 1 patient grade 1, 4 patients grade 4 and 6 patients had grade 1 palsy.

Figure 6



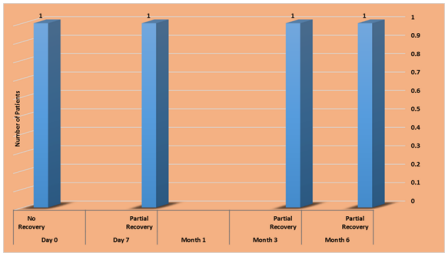
7 out of 11 patients with CNVIII nerve injury had sensorineural type, 3 conductive type and 1 mixed type of hearing loss. 5 out of 7 patients with sensorineural hearing loss did not show any improvement at the end of 6 month whereas 2 showed partial improvement. Out of 3 patients with conductive type of hearing loss 1 showed complete recovery, one showed partial recovery and 1 did not show any recovery at the end of 6 months. 1 patient with mixed hearing loss did not show any improvement at the end of 6 months.

Figure7



One patient diagnosed with CN IX and CN X nerve injury showed partial recovery at the end of 7 days which was static and did not show any further improvement at the end of 6 months.

Figure 8



DISCUSSION

TBI is a significant public health problem worldwide and is predicted to surpass many diseases as a major cause of death and disability by the year 2020.[49].

The incidence of cranial nerve injury in our study was 17%.
There were 51 patients with cranial nerve injuries and 79 cranial nerves involved in the study.

There were 35 patients with single cranial nerve involvement, 15 patients with involvement of 2 cranial nerves and 5 patients with involvement of 3 cranial nerves.

We found facial nerve injury in 17 patients, 12 patients had optic and oculomotor involvement, 11 patients had olfactory and vestibulocochlear involvement, 8 patients had involvement of abducens, 6 patients had involvement of trigeminal nerve, one patient with glossopharangeal and vagal nerve involvement. We had no patients with involvement of trochlear, spinal accessory or hypoglossal nerves.

The commonest cause of head injury was RTA(69.7%). The cranial nerve injury incidence was also maximum in RTA group(82.4%).

Gender

57.8% patients presenting with head injury were males, at par with the world data . The incidence of cranial nerve injury was also more in males (64.7%).

Age group

Bulk of the patients affected with cranial nerve injury in our study were in their productive years of life (21-30 years) 37.3% , (31-40) 33.3%, (41-50) 13.7%.

The incidence of cranial nerve injury in craniocerebral trauma varies from 5 to 23 %.[9]

Purab Patel et al[50] had studied 794 cases of consecutive head injury and found the incidence of cranial nerve injury to be 12.6%. The study had incidence of facial nerve injury to be the highest followed by oculomotor and optic nerve injuries.

Alejandro Fernandez Cohello et al.[51] showed incidence of olfactory nerve was highest followed by facial, oculomotor ,trochlear, abducens and optic nerve injuries in decreasing order. In contrast to the mentioned studies, we found facial nerve to be the commonest with other nerves palsy incidences in corroboration with the above study.

Chung et al[52] suggested that CN I, CN VII, CN VIII are injured the most often and that the lower cranial nerves are affected rarely.

Table 25

	CNI	CNII	CNIII	CNIV	CNV	CNVI	CNV II	CNVI II	CN IX-XII
P Patel et al.50	0.88 %	2.78 %	2.9%	2.14 %	-	3.02%	4.53 %	1.51 %	-
A F Coello et al.51	21%	8.1%	11.3 %	11.3 %	1.4%	12.9%	17.7 %	11.3 %	1.4%
Current study	13.9 %	15.8 %	15.8 %	-	7.59 %	10.12 %	21.51 %	13.9 %	1.2%

Olfactory nerve

The incidence of olfactory nerve injury was 13.9% in our study.
Olfactory nerve injury is reported to occur in 7% of all patients with TBI. Costanzo and Becker[20] reported 19.4% incidence with moderate TBI and 24.5% with severe TBI.
Costanzo and Becker[20] also observed 33% recovery over time.

Optic nerve

The incidence of optic nerve injury was 15.8% in our study.
Optic nerve injury is described as a rare injury[53] with few reported series in literature[54,55,56]. A large series from India[50] has reported incidence of 2.78%.

Mahapatra and Bhatia were first to carry out protocol based prospective study and established the role of VEP in the management[57].

Oculomotor nerve

Incidence of oculomotor nerve injury was 15.8% in our study.
Isolated oculomotor nerve palsy is reported to be 17%[34].

The chances of complete recovery are poor[35] with 40% reported to have complete recovery[32].

Trochlear nerve

We had no patient with trochlear nerve injury.
In one study, incidence of trochlear nerve injury is 0.2 -1.4% with TBI[32]

Trigeminal nerve

Incidence of trigeminal nerve injury was 7.59% in our study.
In some series, the incidence of trigeminal nerve injury is 1.4 to2%[9]

Abducens nerve

Incidence of abducens nerve injury was 10.12% in our study.
Its incidence in one series is 0.4 to4.1%[32].

Facial nerve

17 patients were found to have facial nerve involvement in our study(21.51%). 5 patients had isolated unilateral facial nerve involvement, 9 patients had associated involvement of vestibulococlear nerve, 4 patients had associated abducens nerve injury and one patient had oculomotor injury along with facial nerve. 10 patients had facial palsy House and Brackmann grade 5; five patients with grade 4; one each with grade 3 and 6. Out of 17 patients, 4 patients had residual grade 5 , 2 had grade 2 , 1 had grade 3 , 4 had grade 2 and 6 had grade 1 palsy. All patients with traumatic facial nerve injuries had shown some degree of improvement. 4 patients from the group had undergone decompression of the facial nerve and all had shown some degree of improvement.

Vestibulocochlear nerve

Eleven vestibulocochlear nerves were involved in our study of cranial nerve injury. All 11 nerves had associated facial nerve involvement and 2 had involvement of abducens along with facial. 7 patients had sensorineural hearing loss , 3 had conductive hearing loss and one had mixed hearing loss.

Conductive deafness usually shows signs of recovery; sensorineural deafness, however has worse prognosis, especially if the initial hearing loss was complete[58].This finding is consistent with our study findings.
The incidence of vestibulococlear nerve injury was 13.9% in our study.

Glossopharyngeal and Vagus nerves

There was one male patient with palsy of glossopharyngeal and vagal nerve in our study .The mode of injury was RTA . He had squamous temporal bone fracture and had difficulty in swallowing. On 6 months follow up patient did not show any improvement on clinical examination but complains of dysphagia and difficulty in swallowing had improved .

The incidence of glossopharyngeal and vagus nerve injury was 1.2% each in our study.

Spinal accessory and Hypoglossal nerves

There were no patients with involvement of spinal accessory and hypoglossal nerve involvement in our study.

Isolated unilateral hypoglossal nerve palsy after fracture of the occipital condyle is rare and usually occurs after a major trauma[59].

CONCLUSION

Cranial nerve injury is common with traumatic brain injury and can be seen with even moderate and mild head injury. It significantly affects the productive age group of the society and causes considerable morbidity .Facial nerve is one of the most common nerves affected by trauma; olfactory, vestibulo cochlear, optic and oculomotor nerves are also the common nerves affected by traumatic nerve injury. Lower cranial nerve injuries are rarely seen with mild and moderate head injury patients.

In patients with TBI, it is essential to identify cranial nerve injuries so that these deficits may be addressed, thereby optimizing the clinical outcome and quality of life.

Decompressive surgery for optic and facial nerves have limited use in lessening the morbidity and improving the quality of life.

Olfactory and optic nerve recovery is poorest .All nerves achieve some amount of functional recovery with time.

REFERENCES

1. Mahapatra AK, Jaiswal A.Epidemiology of head injury. Neuroscience Today.2003;7:29-32.
2. Mahapatra AK. Current management of head injury. Neurosciences Today.1997;1:197-204.
3. .Ministry of Transport, Govt of India. Report of Road Safety Cell; January 1993.
4. Jennett B, Teasdale G, eds. Management of Head Injuries. Philadelphial PA: F.A. Davis Company; 1981:272-278.
5. Keane JR: Eye signs of head trauma. In: Rose FC(ed): The Eye in General Medicine. London. Chapman and Hall, 1983.pp276-290.
6. Keane JR: Eye movements in comma. In: Albert DM, Jakobiee W(eds): Principles and Practice of Opthalmology. Philadelphia. WB Saunders. 1994. pp2499-2507
7. Keane JR: Traumatic internuclear ophtalmoplegia. J Clin Neuro-ophthalmol.1987;7:165-166
8. Katzen JT, Jarrahy R, Mathiasen RA, et al: Craniofacial and skull base trauma. J Trauma.2003: 54;1026-1034
9. Keane JR, Baloh RW: Postraumatic cranial neuropathies. Neurol Clin 10:849-867,1992
10. James R Keane and Robert W. Baloh, Post Traumatic Cranial Neuropathies, Neurology and Trauma.2nd edition.1992: 6;129
11. Weiner WJ, Goetz CG,eds. Neurology for the Non-Neurologist. 2nd ed. Philadelphia, PA:JB Lippencott Company;1989.
12. Zafonte RD ,Hammond FM, Peterson J. Predicting outcome in the slow to respond traumatically brain inured: acute and subacute parameter. Neurorehab. 1996;6(1):19-32.
13. Braakman R, Jennett WB, Minderhoud JM. Prognosis of posttraumatic vegetative state. Acta Neurochir (Wein). 1988:95(1-2):49-52.
14. Levati A, Farina ML, Vecchi G, Rossnada M, Marrubini MB. Prognosis of severe head injuries. J Neurosurg; 1982;57(6) 779-783.
15. Narayan RK, Greenberg RP, Miller JD, et al. Improved confidence of outcome prediction in severe head injury. A comaparative analysis of the clinical examination, multimodality evoked potentials, CT scanning, and intracranial pressure. J Neurosurg. 1981;54(6):751-62.
16. Braakman R, Gelpke GJ, Habbema JD, Maas AI, Minderhoud JM. Systematic selection of prognostic features in patients with severe head injury. Neurosurgery. 1980;6(4):362-370.
17. Alexandre A, Colombo F, Nertempi P, Benedetti A. Cognitive outcome and early indices of severity of head injury. J Neurosurg. 1983;59(5):751-761.
18. Jin H, Wand S, Hou L, et al .Clinical treatment of traumatic brain injury complicated by cranial nerve injury. Injury. 2010;41(9): 918-923.
19. Souayah N, speciality board review: Neurology. 2nd ed McGraw hill;2010:22.
20. Costanzo RM, Becker DFP. Sense of smell and taste disorders in head injury and neurosurgery patients. In: Meiselman HL, Rivlin RS, eds. Clinical Management of Taste and Smell. New York, NY: Macmillan Publishing Co; 1986:565-578.
21. Berrol S. Cranial nerve dysfunction: physical medicine and rehabilitation: state of the art review. Vol. 3. Philadelphia, PA: Hanley& Belfus, Inc;1989:85-93.
22. Horn LJ, Zasler ND, eds. Sensory perceptual disorders after traumatic brain injury. In: Thomas MD, Zasler ND, eds. Medical Rehabilitation of Traumatic Brain injury. Philadelphia, PA: Hanley & Belfus, Inc; 1996:499-514.
23. Rovit RL, Murali R. Injuries to the cranial nerves. In: Cooper PR, ed. Head injury. 3rd ed. Baltimore, MD: Lippincott Williams & Wilkins; 1993.
24. Padula WV. Neuro-optometric rehabilitation for persons with a TBI or CVA. Optom Vis Devel. 1992;23:4-8.
25. Glaser RA. Neuro_opthalmology. Baltimore, MD: Harper and Row; 1978:126.
26. Canavero S. Dynamic reverberation. A unified mechanism for central and phantom pain. Med Hypotheses. 1994;42(3): 203-207.
27. Awebuch GI, Sandyk R. Mexiletine for thalamic pain syndrome. Int J Neuroscj. 1990;55(2-4): 129-133.
28. Spoor TC, Mc Henry JG. Management of traumatic optic neuropathy. J Craniomaxillofac Trauma. 1996;2(1): 14-26.
29. Li KK, Teknos TN, Lai A, Lauretano A, Terrell J, Joseph MP. Extra- cranial optic nerve decompression: a 10- year review of 92 patients. J Craniofac Surg. 1999;10(5):454-459.
30. 20 Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: the international Optic Nerve Trauma Study. Opthalmology. 1999;106(7)1273-1277.
31. Steinsapir KD. Traumatic optic neuropathy. Curr Opin Opthalmol. 1999;10(5)340-342.
32. Keane JR. Neuro-ophthalmic signs and symptoms of hysteria. Neurology. 1982;32(7):757-762.
33. Obyan A, Zafonte R, Hammond F. Occipital status epileptics: and unusual cause of post traumatic blindness. Arch Phys Med Rehabil. 1995;76(11):1085.
34. Sabates NR, Gonce MA, Farris BK. Neuro-opthalmological findings in closed head trauma. J CLIN Neuropthalmol. 1998;9(6):33-39.
35. Tokuno T, Nakazawa k, Yoshida S,et al. Primary oculomotory nerve palsy due to head injury: analysis of ten cases(in japanese). No Shinkei Geka. 1995;23(6):497-501.
36. Vaughn D, Asbury T. General Ophthalmology. Los Altos, CA; Lange Medical Publication:1974:182.
37. Lagreze W A. Neuro-opthalmology of trauma. Curr Opin Opthalmol. 1998;9 (6):33-39.
38. Beauchamp GR, Black BC, Coats DK, et al. The management of strabismus in adults- 1. Clinical characteristics and treatment. J AAPOS. 2003;7(4):233-240.
39. Dacey RG, Jane JA. Craniocerebral trauma. In: Baker AB, Baker IH, eds. Clinical Neurology. Rev ed. Philadelphia, PA: Harper and Row; 1987:1-61.
40. Backonja MM. Use of anticonvulsants for treatment of neuropathic pain. Neurology. 2002; 59(5)(Suppl2):S14-S17.
41. Rosen's Emergency Medicine: concepts and clinical practice. 4thed. Mosby-Year Book, Inc;1998:1278.
42. Blumnfield H. Neuroanatomy through clinical cases. Sinauer Associates, inc 2002:540.
43. Kay PP, Kinney SE, Levine H, Tucker HM. Rehabilitation of facial paralysis in children. Arch Otolaryngol. 1983;109(10):642-647.
44. Podoshin L, Fradis M. Hearing loss after head injury. Arch Otolaryngol. 1975;10(1):15-18.
45. Sismanis A. Post concussive neuro ontological disorders. Phys Med Rehab: State of the Art Reviews. 1992;6 (1): 79-88
46. Tos M. Prognosis of hearing loss in temporal bone fractures. J Laryngol Otol. 1971; 85(11):1147-1159.
47. Tangeman PT, Wheeler J. Inner ear concussion syndrome: vestibular implications and physical therapy treatment .Top Acute Care Rehabil. 1986:1:72-73.
48. Yorkston KM, Beukelman DR, Strand EA, Bell KR. Management of Motor speech Disorder. 2nd ed. Austin, TX: Pro_ed, Inc; 1999.
49. World Health Organization. Projections of Mortality and Burden of Disease to 2030: Deaths by Income Group. Geneva; 2002 12/01/06.
50. Patel P, KalyanaramanS, Reginald J, Natarajan P, Ganapathy K et al; Post traumatic cranial nerve injury. Ind J Neurotrauma 2:27-32,2005
51. Alejandro Fernandez Coello, Andreu Gabarros Canals,Juan Martino Gonzalez et al; Cranial nerve injury after minor head trauma. J Neurosurg 113:547-555,2010.
52. Chung SM, Fenton GA, Schmidt JG, Selhorst JB: Trauma to the cranial nerves and brainstem, Narayan RK, Wilberger JE, Povlishock JT (eds): Neurotrauma. New York: McGraw-Hill, 1996, pp 621–638.
53. Crompton MR. Visual lesions in head injury. Brain 1970;93:785-92.
54. Fukado Y. Microsurgical transethmoidal optic nerve decompression. Experience with 750 cases. In, Janetta P, Samii M (eds). Cranial nerves. Springer-verlag 1981:pp125-8.
55. Mahapatra AK, Tandon DA, Bhatia R, Banerji AK. Optic nerve injury: A prospective study of 100 patients. Neurol India 1992; 40:17-21.
56. Mahapatra AK. Rationale of optic nerve decompression in optic nerve injury. Ind J Neurotrauma 2004;1:33-6.
57. Mahapatra AK, Tandon DA. A prospective study of 250 patients with optic nerve injury. In, Madjid Samii (Ed). Skull base: Anatomy, Radiology and Management. S Karger Basel 1994: pp305-9.
58. Bhatoe HS: Trauma to cranial nerves. Ind J Neurotrauma 4:89-100,2007
59. Kausik V, Kelly G, Richards SD, Saeed SR: Isolated unilateral hypoglossal nerve palsy after minor head trauma. Clin Neurol Neurosurg 105:42-47, 2002.