Original Research Article

Study on involvement of cranial nerves in head injuries and to evaluate clinicoradiological correlation

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Abstract

Introduction: The incidence of cranial nerve injuries in craniocerebral trauma varies between 5-23 percent in various literature and can occur by sudden acceleration/deceleration, brain stem injury or injury to the skull base. **Aims**: To evaluate the involvement of cranial nerves in mild and moderate head injuries and to find out clinicoradiological correlation**. Materials and methods**: The present prospective observational and descriptive study was done at Kamineni hospitals, L.B. Nagar, Hyderabad on 950 patients to evaluate the involvement of cranial nerves in mild and moderate head injuries and to assess the clinicoradiological correlation. **Results**: Cranial nerve injuries were seen in 134 cases, but cumulative number of cranial nerve palsies was 248. However, out of 134 cases radiological correlation was seen in only 114 cases. Significant association was found between involvement of cranial nerve and clinical and radiological findings ($p<0.05$). Cranial nerve VI was involved in most of the cases (42), followed by cranial nerve III (41), cranial nerve VII (41). Cranial nerve I was involved in 23 cases, II and IV in 28 cases each, V in 20 cases, cranial nerve VIII in 16 cases, IX and X in 4 cases each. **Conclusion**: Meticulous clinical and radiological examination, documentation and follow-up are essential to detect cranial nerve injuries mild and moderate head injuries

Keywords: Cranial nerve, Head injuries, Glasgow Coma Scale (GCS)

Introduction

Cranial nerves are the nerves that emerge directly from the brain (including the brainstem), of which there are conventionally considered twelve pairs. Cranial nerves relay information between the brain and parts of the body, primarily to and from regions of the head and neck, including the special senses of vision, taste, smell, and hearing[1]. The cranial nerves emerge from the central nervous system above the level of the first vertebrae of the vertebral column. Each cranial nerve is paired and is present on both sides. There are conventionally twelve pairs of cranial nerves[2].

The Olfactory nerves (I) and optic nerves (II) emerge from the cerebrum, and the remaining ten pairs arise from the brainstem, which is the lower part of the brain[3]. In order of most cranial to caudal, these include the Oculomotor nerve (III), the Trochlear nerve (IV), the Trigeminal nerve (V), the Abducens nerve (VI), the Facial nerve (VII), the Vestibulocochlear nerve (VIII), the Glossopharyngeal nerve (IX), the Vagus nerve (X), the Accessory nerve (XI) and the Hypoglossal nerve (XII). The cranial nerves are considered components of the peripheral nervous system (PNS)[3], although on a structural level the olfactory (I), optic (II), and trigeminal (V) nerves are more accurately considered part of the central nervous system (CNS)[4]. Many of the cranial nerves with nuclei within the brain stem contain sensory and motor components. The sensory fibre components have their cell bodies located in ganglia outside the central nervous system and the motor fibre element have their cell bodies within the central nervous system.

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The head and neck accounts for only 12% of body surface area but is densely innervated with cranial nerves that perform diverse biological functions. Afferent cranial nerves play important roles in assimilating sensory information, while efferent nerves execute motor functions. Cranial nerve injuries are often overlooked in cases of neurotrauma, which are diagnosed later during recovery. Most of these injuries do not require active intervention in the acute stage. Cranial nerve injuries are important cause of morbidity, which requires long-term management, repeated surgical procedures or reconstructive measures. Management of optic nerve injury remains controversial, and injury to lower cranial nerves may influence the ultimate outcome due to paralysis of aerodigestive passage. Cranial nerve injury in the setting of head injury should be diagnosed early, so that appropriate treatment can be planned early.Cranial nerve injury is an important component of neurotrauma, which may not be readily apparent in the emergency room where the patient is brought in after having sustained head injury. The incidence of cranial nerve injury in craniocerebral trauma varies between 5 and 23 percent[5]. Initial evaluation by history and 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. Complete evaluation of individual cranial nerves, a time- consuming and elaborate exercise even in a conscious patient, may not be feasible in a comatose patient or in one with altered sensorium.

Cranial nerves, along with major arteries and bridging veins, act as anchors to the brain in a sea of cerebrospinal fluid. Injury to the cranial nerves can occur by shearing forces, rapid acceleration/deceleration, injury to the skull base, penetrating craniocerebral injuries, especially those through the skull base and as a sequel to various surgical procedures. Computed tomography (CT) scan is used to quickly assess the extent of injury and to triage the patients. CT scan allows, usually, an indirect view of the nerve and is

useful to demonstrate the intra osseous segments of cranial nerves, the foramina through which they exit skull base and their pathologic changes. Hence the present Prospective Observational and Descriptive study was done at Kamineni hospitals, L.B. nagar , hyderabad to evaluate the involvement of cranial nerves in mild and moderate head injuries and assess the clinico radiological correlation.

Material and methods

The Prospective Observational and Descriptive study was done at our tertiary care centre in the department of Neurosurgery, Kamineni Hospitals, L.B. nagar, Hyderabad on patients admitted (inpatient). All 950 inpatients of minor head injury (GCS 13-15) and moderate head injuries (GCS 9-12) which improve to GCS 14/15 within 4 weeks of trauma were included. .

Considering a confidence level of 95% and confidence interval of 3.1 the number of patients in our study to achieve statistical significance is 999. This was calculated by Survey System. The Survey System ignores the population size when it is "large" or unknown. Population size is only likely to be a factor when you work with a relatively small and known group of people (e.g., the members of an association). Hence a sample size of 950 patients was considered adequate for our study.

Inclusion criteria

All cases of minor head injuries (GCS 13-15) and moderate head injuries(GCS 9-12) which improved to GCS 14/15 within 4 weeks of trauma

Exclusion criteria

Severe head injury cases (GCS <8) Patients having cranial nerve involvement due to causes other than trauma, previous history of cranial nerve paresis

After approval from the Institutional Ethics Committee a valid informed consent was taken. Once the patients were enrolled for the study, a thorough history and physical examination was done as per proforma. An informed consent was taken in written from patients or patient's attendant. The patients were grouped into mild and moderate head injury based on the GCS at the time of admission mild (13-15), moderate (9-12). All patients were investigated with CT scan of brain at the time of admission. Clinical examination of cranial nerves was done meticulously on a daily basis. 3D CT scan, audiogram, Visual Evoked Potential, Fundus examination and other investigations was done when clinically indicated and patients were managed further accordingly. Patients were followed up at one-month, 3 months and 6 months interval.

Statistical Analysis

Quantitative data is presented with the help of Mean and Standard deviation. Comparison among the study groups is done with the help of unpaired t test as per results of normality test. Qualitative data is presented with the help of frequency and percentage table. Association among the study groups is assessed with the help of Fisher test, student 't' test and Chi-Square test. 'p' value less than 0.05 is taken as significant**.**

Pearson's chi-squared test

Where X2 = Pearson's cumulative test statistic.

Oi = an observed frequency;

Ei = an expected frequency, asserted by the null hypothesis;

n = the number of cells in the table.

Results were graphically represented where deemed necessary.

Appropriate statistical software, including but not restricted to MS Excel, SPSS ver. 20 were used for statistical analysis. Graphical representation was done in MS Excel 2010.

$$X^2 = \sum_{i=1}^{n} \frac{(O_i - E_i)^2}{E_i}$$

Results

Table 1:Distribution according to age group of study population

Age group (years)	Number	Percent
0 to 15 years	93	9.8
16 to 30 years	301	31.6
31 to 45 years	280	29.6
46 to 60 years	157	16.5
More than 60 years	119	12.5
Total	950	100

Most of the participants were from age group 16 to 30 years (31.6%) followed by 31 to 45 years (29.6%). 16.5% were 46 to 60 years of age.

Table 2:Distribution according to gender

Gender	Mild GCS	Moderate GCS
Male	321	217
Female	189	223
Total	510	440

Males with moderate GCS were 217 while those with mild GCS were 321 in number. Females with moderate GCS were 223 and mild GCS were 189. No significant association was found between gender and GCS (p>0.05).

Table 3: Mode of injury

Mode of injury	Mild GCS	Moderate GCS
RTA	318	213
Fall	169	180
Assault	11	17
Others	12	30

RTA was most common mode of injury in 318 cases with mild GCS while 213 cases with moderate GCS, fall was seen in 169 cases with mild GCS and 180 cases with moderate GCS. No significant association was found between mode of injury and GCS.

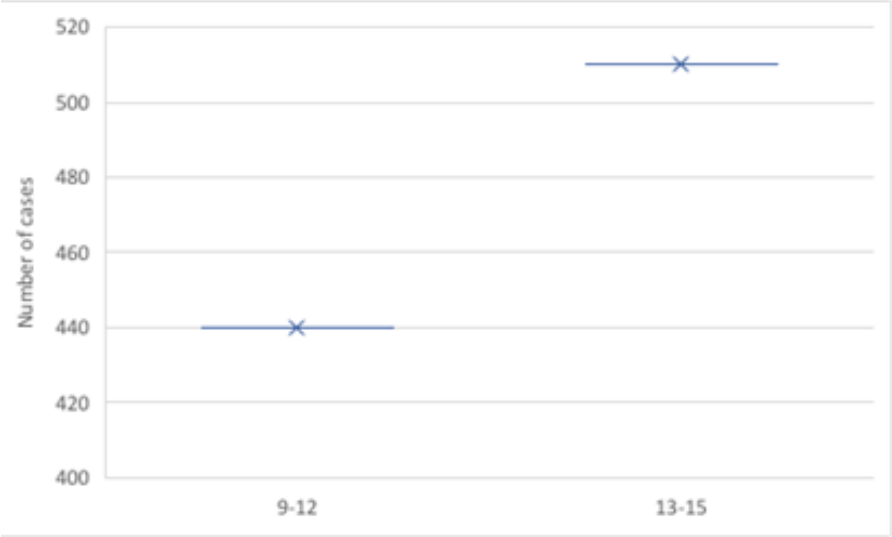

Fig 1: Distribution according to GCS score

Most of the cases had mild GCS score (53.6%) while 46.4% had moderate GCS score.

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Table 4: Involvement of cranial nerve Clinically and Radiologically

CN involved	Clinically	Radiologically
First cranial nerve	12	8
Second cranial nerve	6	4
Third cranial nerve	13	11
Fourth cranial nerve	2	2
Fifth cranial nerve	7	7
Sixth cranial nerve	9	8
Seventh cranial nerve	12	10
Eighth cranial nerve	8	8
Second, third, fourth & sixth	16	14
Sixth & seventh	8	6
Seventh & eighth	8	8
Third & fourth	2	2
First, third, fourth & sixth	5	5
First & second	8	7
Third, fourth, fifth & sixth	5	5
Fifth & seventh	9	5
Seventh, ninth & tenth	4	4
Total	134	114

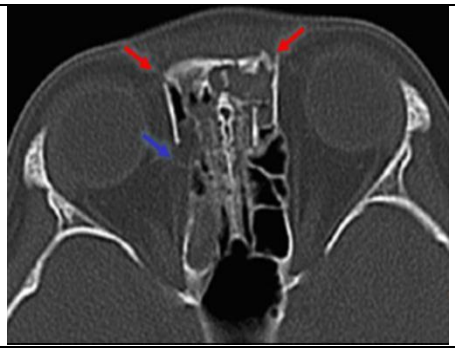
On clinical examination, 134 cases were found to have either isolated or multiple cranial nerve injuries. However, radiological correlation was seen in only 114 cases. Significant association was found between involvement of cranial nerve and clinical and radiological findings ($p<0.05$).

Table-5: Distribution according to cranial nerve involvement in total cases

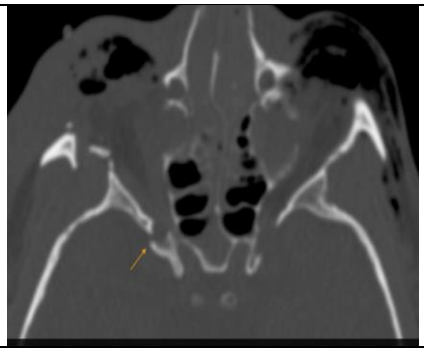
Cranial nerves	Involvement (No. of cases out of total)	Fractures associated		
I	23 (2.4%)	Fracture cribriform plate 12	Anterior skull base fracture 19	
II	28 (2.9%)	Optic foramen fracture 14	Orbital wall fracture-Med/Lat/Sup/Inf 22	Fracture in region of sella and clinoid 2
III	41 (4.3%)	Fracture at superior orbital fissure 22	Orbital wall fracture 37	Maxillary fracture
IV	28 (2.9%)	Fracture at superior orbital fissure 23	Orbital wall fracture	Maxillary fracture 16
V	20 (2.1%)	Fracture skull base involving middle fossa (ovale & rotundum) 2	Maxillary fracture 13	Mandibular fracture 5
VI	42 (4.4%)	Fracture at superior orbital fissure 25	Orbital wall fracture 3	Transverse

				sphenoidal fracture 3
VII	41 (4.3%)	Transverse/ Longitudinal temporal bone fracture 10	Internal acoustic meatus fracture 5	
VIII	16 (1.6%)	Longitudinal fracture 16	Ossicular discontinuity 4	
IX	4 (0.4%)		Jugular foramen fracture 4	
X	4 (0.4%)		Jugular foramen fracture 4	
XI	0 (0%)	Occipital condyle fracture	Jugular foramen fracture	
XII	0 (0%)	Occipital condyle fracture		

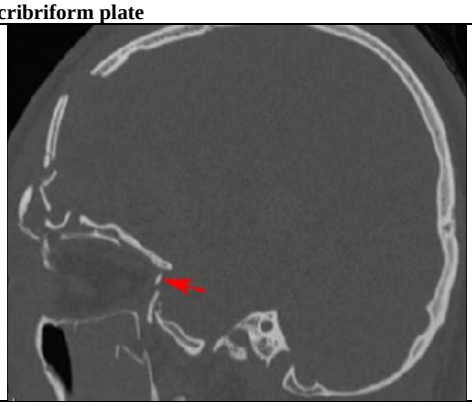
Cranial nerve VI was involved in most of the cases (42), followed by cranial nerve III (41), cranial nerve VII (41). Cranial nerve I was involved in 23 cases, II and IV in 28 cases each, V in 20 cases, cranial nerve VIII in 16 cases, IX and X in 4 cases each.



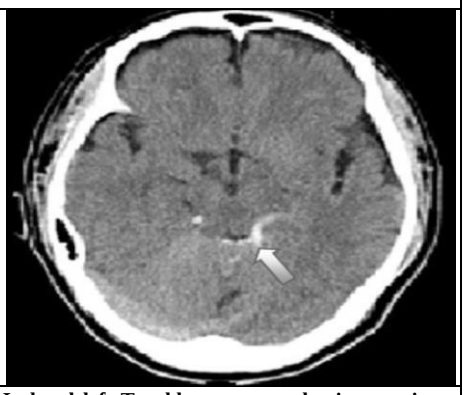
Scan of a patient with Anosmia- CT showing anterior skull base fracture causing disruption of cribriform plate



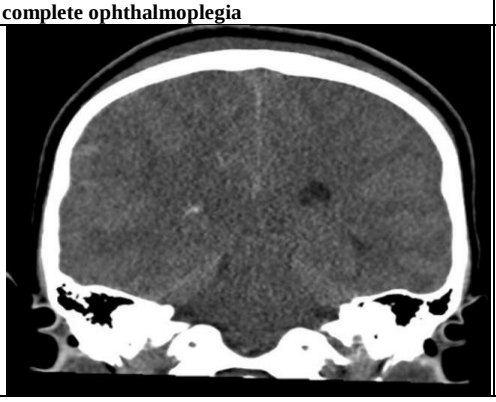
Isolated right optic nerve injury- fracture extending into optic canal

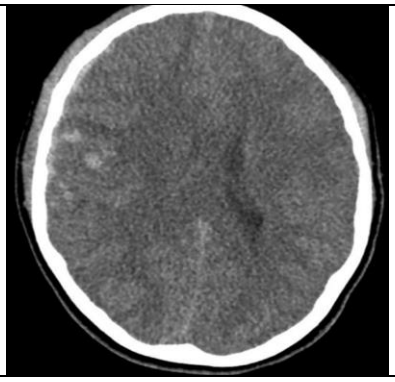


Fracture involving superior orbital fissure causing complete ophthalmoplegia

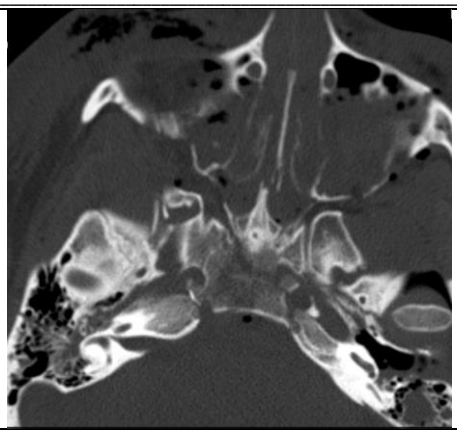


Isolated left Trochlear nerve palsy in a patient where scan shows left ambient cistern SAH

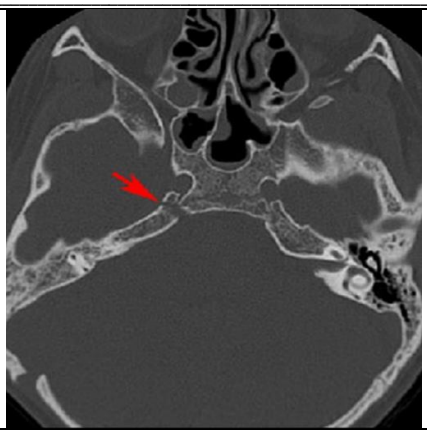




Isolated 6th nerve palsy due to raised ICP and entrapment between herniating brain and tentorium



Fracture of basisphenoid extending to involve foramen rotundum and ovale



Petrous apex fracture causing ipsilateral fifth and seventh nerve injury

Fig 2: Cases studied in present study

Discussion

The Present prospective Observational and Descriptive study was done at our tertiary care centre on 950 patients to evaluate the involvement of cranial nerves in mild and moderate head injuries and assess the clinicoradiological correlation. In the present study, most of the participants were from age group 16 to 30 years (31.6%) followed by 31 to 45 years (29.5%). 16.5% were 46 to 60 years of age. Total number of Males suffering traumatic head injury were 510 in comparison to 440 females. No significant association was found between gender and GCS ($p>0.05$). This is similar to the studies of Patel P et al[6], Kumar P et al[7]and Shekhar C et al[8]. It was observed in the present study that RTA was most common mode of injury seen among 531 cases of mild and moderate head injury. Fall was seen in 349 cases. No significant association was found between mode of injury and GCS. This finding was consistent with the studies of Kumar P et al[7], Shekhar C et al[8], Keki ET et al[9], Singh I et al[10]and Odebode TO et al[11].

In the present study, most of the cases (53.7%) had mild GCS score while 46.3% had moderate GCS score. This is in concordance to the studies of Shekhar C et al[8], Patel P et al[6], Kumar P et al[7]and Mugala DD et al[12]. Shekhar C et al[8]retrospective, prospective management study observed as per Glasgow coma scale mild, moderate & severe grade of TBI was seen in 62%, 22% & 16% cases respectively. Kumar P et al[7] prospective study assessing cranial nerve deficits in acute head injury observed commonest cause was RTA (69.7%). The cranial nerve injury incidence was also maximum in RTA group (82.4%). On the other hand, Shekhar C et al[8] found fall from height was the main cause of TBI (56%) followed by road traffic injury (RTI) (36%). On clinical examination, 134 cases were found to have either isolated or multiple cranial nerve injuries. However, radiological correlation was seen in only 114 cases. Significant association was found between involvement of cranial nerve and clinical and radiological findings. The remaining cranial nerve injuries where radiological correlation could not be found were attributed to either neuropraxia, shear injury or nerve contusion. Significant association was found between involvement of cranial nerve and clinical and radiological findings ($p<0.05$). Similar observations were noted in the studies of Patel P et al[6], Kumar P et al[7], Coello AF et al[13]and Col Bhatoe HS et al[5].

Olfactory Nerve Injury

23 patients (2.4%) had post-traumatic olfactory dysfunction. 12 patients presented with anosmia alone and in 12 other patients olfactory nerve injury was present along with multiple cranial nerve injuries. 19 cases had fractures involving anterior skull base while fracture extending to involve the cribriform plate was seen in 12 cases. Almost all patients with only olfactory nerve dysfunction had sustained mild head injury. CSF rhinorrhea was commonly associated. There was partial improvement in perception of smell with 6 months of follow up. Partial recovery is thought to occur due to neurofibrillary regrowth and central adaptation.

Optic nerve injury

28 patients (2.9%) had post-traumatic optic nerve injury. All cases presented with unilateral blindness and no case of bilateral optic nerve involvement was noted in our series. Isolated optic nerve injury was seen in 6 cases while associated multiple cranial nerve injury were seen in 22 cases. Orbital fractures were present in almost all (22 cases). Multiple orbital fractures extending up to optic foramen was seen among 14 cases. Anterior skull base fracture extending to involve the sella or clinoid was seen in only 2 cases. Intravenous steroid (Methylprednisolone) followed by oral steroid in tapering doses were given to all patients as per protocol suggested by Mahapatra and Bhatia. Improvement of visual acuity was varied. None of our patients underwent optic nerve decompression surgery.

Oculomotor nerve injury

Total 41 (4.3%) patients developed post- traumatic oculomotor nerve injury. Isolated oculomotor nerve injury was seen in 15 cases and remaining 26 cases were associated with ophthalmoplegia. Almost all cases had associated orbital wall fractures (37 cases). In most of the cases of ophthalmoplegia, fracture line was seen involving the superior orbital fissure (22 cases). Cases with isolated nerve injury showed good recovery which was attributed to some component of neuropraxia or concussion type of injury. However, recovery period for complete third nerve palsy along with ophthalmoplegia was long. The chances of complete recovery are poor.

Trochlear nerve injury

28 (2.9%) patients were found to have post-traumatic trochlear nerve injury. Isolated trochlear nerve injury was seen in only 2 cases and both presented with Diffuse axonal injury with a shear injury .Dorsal midbrain. Trochlear nerve injury along with multiple nerve injuries was seen in 28 cases. Fracture involving superior orbital fissure was seen in 23 cases and associated maxillary fractures were seen in 16 cases. Spontaneous recovery was seen in few. However, observation was trochlear nerve was more prone to partial and delayed recovery.

Trigeminal nerve injury

20 patients (2.1%) were found to have decreased facial sensation and chewing difficulties. Almost all cases presented with fractures involving the maxilla and mandible. In 2 cases middle fossa skull base fracture was noted extending upto foramen ovale and rotundum. Good recovery was seen in follow-up period. Many a times the decrease in facial sensation is because of traumatic facial edema which later recovers. Chewing difficulty/restriction is also due to pain after fractures and not due to damage to the nerve proper.

Abducens nerve injury

There were 42 (4.4%) patients who presented with sixth cranial nerve palsy. Among these, isolated nerve palsy was seen in 8 cases and

remaining 34 patients had multiple cranial nerve injury. Most often isolated 6th nerve palsy was seen among patients with a large EDH/SDH requiring evacuation and resolved immediately after decrease in ICP. In almost all cases of ophthalmoplegia fracture was seen involving the superior orbital fissure (25 cases). Transverse sphenoid fracture was seen in 3 cases compressing the nerve between free tentorial edge and fracture. Abducens nerve injury along with multiple ocular nerve injuries showed delayed improvement.

Facial nerve injury

Post traumatic facial nerve injury was seen in 41 cases (4.1 %). Isolated facial nerve injury was seen in 12 cases which were invariably associated with transverse/longitudinal petrous fractures. Fractures causing disruption of internal acoustic meatus was seen in 5 cases. UMN facial nerve injury along with unilateral 6th nerve palsy was seen due to large EDH/SDH/contusions compressing the motor strip and causing mass effect. Treatment given was short course of oral steroids in a tapering regimen. In contrast to patients with early facial palsy, patients with delayed onset show better improvement. Also, one observation was that UMN facial palsy showed better recovery than LMN facial palsy.

Vestibulo-cochlear nerve injury

Eighth nerve involvement was seen in 16 cases (1.6%). 8 patients developed sensory neural hearing loss and other 8 patients had mixed hearing loss. Sensorineural hearing loss was always associated with Petrous temporal fractures. Ossicular chain disruption with hemotympanum was seen in 4 cases. Overall, only few patients showed hearing improvement. Also, patients with conductive hearing loss showed minimal improvement as compared to sensorineural hearing loss.

Multiple cranial nerve injury

Most common group of multiple cranial nerve injuries was seen among the ocular nerves causing complete ophthalmoplegia and total visual loss accounting for 16 cases. They were mostly seen in high velocity trauma causing marked disruption of orbital walls. UMN facial and unilateral 6th nerve palsy was seen again in lesions causing mass effect and requiring surgical decompression procedures. Ipsilateral 5th and 7th nerve involvement was seen secondary to gross maxillary and mandibular fractures causing damage to the nerves along their exit foraminas. Involvement of seventh, ninth and tenth nerve was seen in only 4 cases with posterior fossa skull base fractures extending up to the jugular foramen. These patients showed no clinical improvement at the end of follow-up but the complains of swallowing difficulty had completely subsided. Recovery is long in multiple cranial nerve injuries probably because of severe form of head injury.

Patel P et al[6] found sixty-seven patients (67%) had single cranial nerve injury. Multiple cranial nerve injury was seen in 32 patients (32%).

Seven patients (0.88%) had post-traumatic olfactory dysfunction. CSF rhinorrhea (3 patients) & total loss of smell (5 patients) were common findings as were in my study.

Twenty-two patients (2.78%) had post-traumatic optic nerve injury. 11 patients had skull base fractures (7 patients with severe head injury). 3 patients with bony impingement of optic nerve underwent surgery under steroid cover but showed minimal to no improvement. Improvement is visual acuity was varied with follow up period ranging from 8 months to 2 years period.

23 patients developed post-traumatic oculomotor nerve injury with an incidence of 2.9% including 11 patients with multiple cranial nerve injury. Bilateral oculomotor nerve palsy and complete oculomotor nerve palsy were more common with severe head injury. The recovery period for complete third nerve palsy was long (6 weeks to several months). Seventeen patients had post-traumatic trochlear nerve injury with an incidence of 2.14%. Midfacial fractures and superomedial orbital wall fractures were commonly associated.

24 patients had sixth cranial nerve palsy with an incidence of 3.02%. Skull base fractures (16 patients) involving clivus (4 patients) was the common findings. All patients with only 6th nerve injury showed better improvement within 3 to 8 weeks. Facial nerve injury was seen in 36 patients (4.53%). Longitudinal fractures were most common followed by transverse and mixed fractures. Incidence of multiple cranial nerve injuries was 4.03%. Sixth and seventh cranial nerves were more commonly involved followed by third, second and fourth cranial nerve.

Kumar P et al[7] did a very similar study as of mine and provided following incidence of each cranial nerve in mild and moderate head injury.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. He found 35 patients with single cranial nerve involvement, 15 patients with involvement of 2 cranial nerves and 5 patients with involvement of 3 cranial nerves.

Coello AF et al[13]also did an amazing study and found 49 patients with CN injury, an incidence of 0.3%. Posttraumatic single nerve palsy was observed in 38 (77.6%) patients and multiple nerve injury in 11 (22.4%). 13 cases of olfactory nerve injury (21 %), 5 cases of optic nerve injury (8.1 %), oculomotor and trochlear injury in 7 cases (11.3 %) each respectively, 1 case of trigeminal (1.4 %), 8 cases of abducens (12.9 %), 11 patients of facial injury (17.7 %), 7 patients of eighth (11.3 %) and 1 case of lower cranial nerves injury (1.4 %) He also observed that when more than 1 CN was involved, the most frequent association occurred between CNs VII and VIII.

Conclusion

Cranial nerves are injured before, during or after their passage through the skull. Meticulous clinical and radiological examination, documentation and follow up are essential to detect these injuries. In the future, the already significant role of imaging in guiding therapy may grow. Technological improvements continue to reduce scanning time and improve resolution. New methods are being developed to quantify damage on images and perhaps improve predictive power. A growing number of minimally invasive, image-guided techniques are replacing open surgical techniques. Imaging is increasingly vital to the development of new therapies and may be used to measure patient response to these therapies. Imaging has and will continue to influence therapy and may improve outcomes for what is clearly a significant health care problem. Delayed recovery can occur with other associated injuries. Early recognition and treatment may provide beneficial effects.

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