



Incidence of traumatic optic neuropathy in head injury patients in a tertiary health care center

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Abstract

Purpose: To study the incidence of traumatic optic neuropathy (TON) in closed head injury patients.

Materials and Methods: A prospective study comprising 85 closed head injury patients for a period from February 2020 to March 2020. Head injury patients were examined and assessment of external injury, anterior segment examination, pupillary reaction, visual acuity, assessment of extra-ocular movements, visual field assessment by finger confrontation, fundus examination by direct and indirect ophthalmoscopy was done.

Results: In this study the incidence of TON was found to be 1.17% and the mean age of 39.28 years.

Conclusion: Traumatic optic neuropathy is one of the important neuro-ophthalmic manifestations of closed head injury. It has a positive correlation with severe head trauma. Patients with TON can present with a variable degree of vision loss, with most cases present with severe vision loss. In the acute phase, the optic nerve usually appears normal on fundoscopic examination. Hence high incidence of suspicion is required to diagnosis this rare complication as this study showed TON in closed head injury patients.

Keywords: traumatic optic neuropathy, head injury, visual acuity

Introduction

Traumatic optic neuropathy (TON) is the injury to the optic nerve usually follows ocular, orbital or head trauma as sudden visual loss that cannot be explained by other ocular pathology. Optic nerve distress is frequently connected with severe head trauma and multisystem injury. TON occurs in most of the cases of motor vehicle accidents, which produce high energy deceleration type head trauma. Patients with unilateral traumatic neuropathy demonstrate a relative afferent pupillary defect and decreased vision.

Optic neuropathy is a potential blinding complication of head or orbital trauma. The most common form of traumatic optic neuropathy is indirect damage to the optic nerve and has been reported following 0.5% to 5% of all head trauma patients [1, 2]. It is defined as traumatic visual loss which occurs without initial ophthalmoscopic evidence of injury to the eye ball or optic nerve.

Indirect injuries are caused by concussive forces that are transmitted to the optic nerve as a result of orbito-facial or cranial trauma [3, 4]. This impact generate a shock wave which can lead to optic nerve avulsion or posterior indirect TON [5, 6]. Direct TON results from direct trauma to the nerve from sharp objects, missiles and bony fragments [7]. The clinical presentation may vary. The degree of the visual loss does not always correlate with the severity of trauma. The optimal treatment of TON remains controversial. There has been no conclusive evidence for standardized treatment protocol. Observation, corticosteroids treatment and decompression of the nerve.

Materials and Methods

Total of 85 patients of closed head trauma, reporting to the emergency department at R.L.J. Hospital and Research Centre, Tamaka, Kolar attached to Sri Devaraj Urs Medical College between February 2020 and March 2020, were

included in the study.

Study design: Retrospective observational study.

Inclusion criteria

1. All patients reporting to the emergency department with closed head injury.

Exclusion criteria

1. Previous history of any neurological disease.
2. Previous history of ocular trauma

The study comprised the analysis of 85 patients who were diagnosed of closed head injury. Detailed history regarding injury was documented. Patients were examined at the time of presentation, and follow-up was done at 1 week, 4 weeks and 6 weeks. Ophthalmic examination included assessment of external injury, anterior segment examination, pupillary reaction, visual acuity, assessment of extra-ocular movements, visual field assessment by finger confrontation, fundus examination by direct and indirect ophthalmoscopy.

Results

Total of 85 cases were included in the study out of which 60 were males and 25 were females. Mean age of distribution in the study was 39.28 years.

Table 1: Distribution of patients according to age and sex

Age (Years)	Male	Female	Total
<20	3	-	3
21-30	23	11	34
31-40	13	4	17
41-50	10	8	18
51-60	4	2	6
61-70	5	-	5
>70	2	-	2

We came across many different types of ocular manifestations in closed head injury patients out of which one case of TON was reported in our study.

Discussion

In our study young male patients were the most affected group. There were 60 males and 25 females. For men, the age ranged from 4 months to 70 years with the mean age of 34.14 years. The age range for female patients was from 21 to 75 years, mean being the 45.43 years. The majority of males who were affected had age ranging from 21-30 years (23 cases) and the majority of affected females were also in the age group 21-30 years (11 cases).

Our study showed pupillary abnormalities in 3.52 % and 1.17% patients had traumatic optic neuropathy. In the study by Masila *et al*^[8] 39 eyes had atypical pupil reaction and 2 patients had third cranial nerve palsy. According to Kulkarni *et al*^[9], involvement of pupil was present in 5% cases and it was the most consistent neuro-ophthalmic sign. Sixth cranial nerve palsy was recorded in 2% of head injury cases. Traumatic optic neuropathy is seen in 0.5% of cases and third nerve palsy is seen in 1.5% of the cases.

Severe head trauma and posterior segment findings had constant relation, without any exception. Severe head trauma was also positively correlated with the neuro-ophthalmic findings. According to Odebo *et al*^[10], severe head injury patients had only soft tissue injury to the eye, adnexa and periorbital region, 50% of the times. 43.75% patients with severe head injury had neuro-ophthalmic manifestations in association with damage to ocular soft tissue, adnexa and periorbital region as well. Patients with extreme severity of head injury had rupture of the globe and fracture orbit as well as neuro-ophthalmic manifestations and soft tissue damage to eye, adnexa and periorbital region in 6.25 % cases. According to Kulkarni *et al*^[7], 82.7% patients with less severe head injury had eye involvement of no neurological significance. 82.8% of patients of moderately injured head had ocular involvement. 9(31.03%) patients had pupillary signs. Papilloedema was observed in 10.34% cases, lateral rectus palsy in 13.79% cases, ecchymosis and orbital margin fractures in 6.9% cases each, ptosis, and traumatic optic neuropathy in 3.45% cases each. The findings of these studies largely correlate with the findings of our study.

In our study out of 85 cases only one case had TON. This case presented with perception of light and lacerated wound in the left upper eyelid of 3 x 1 cm with periorbital edema with ecchymosis. Pupillary examination showed grade 3 RAPD in left eye. Dilated fundoscopic examination showed normal fundus study at presentation. CT brain plain showed Sub-dural haemorrhage in parietal lobe with mild cerebral edema. No associated orbital fracture was noted. Patient was suspected to have left eye TON and started him on intravenous methyl prednisolone 1 gm once daily for 3 days after physician and neurosurgeon opinion and examined for any visual improvement but patient did not show any improvement. Dilated fundoscopic examination was repeated on second day and showed normal fundus study. On follow up, due to complications of head injury patient died on the 4th day of presentation.

With this incidence of TON in our study is 1.17%. Larger sample size would have given better understanding of the incidence of TON in closed head injury patients.

Conclusions

Traumatic optic neuropathy is one of the important neuro-ophthalmic manifestations of closed head injury. It has a positive correlation with severe head trauma. Patients with TON can present with a variable degree of vision loss with most cases present with severe vision loss. In the acute phase, the optic nerve usually appears normal on fundoscopic examination.

References

1. Carta A, Ferrigno L, Salva M, Marzoli S, Boschi A, Carta F. Visual prognosis after indirect traumatic optic neuropathy. *J Neurol Neurosurg Psychiatry*. 2003; 74:246-48.
2. Kovacic M, Gracner T, Gracner B. Indirect Traumatic Optic Neuropathy – two case reports. *Coll Antropol*. 2001; 25:57-61.
3. Glaser JS. Traumatic Optic Neuropathy. In: Glaser L, Glaser JS, editors. *Neuro-ophthalmology*. 3rd ed. Lippincott Williams and Wilkins, 1999, p186-88.
4. Beretska JS, Rizzo JF. Controversy in the management of traumatic optic neuropathy. *Int Ophthalmol Clin*. 1994; 34:87-96.
5. Liu GT, Volpe NJ, Galetta SL. Philadelphia: WB Saunders; *Neuro-ophthalmology: Diagnosis and Management*, 2001, p170-72.
6. Kline LB, Morawetz RB, Swaid NS. Indirect injury of the optic nerve. *Neurosurgery*. 1984; 14:756-64.
7. Nazir SA, Westfall CT, Chacko JG, Philips PH, Stack BC. Visual recovery after direct traumatic optic neuropathy. *Am J Otolaryngol*. 2010; 31:193-94.
8. Faith M, Githinji KJ, Sheila M, Margaret N. Ocular findings in patients with head injury. *International Journal of Medical and Clinical Sciences*. 2014; 1(2):009-017.
9. Kulkarni AR, Aggarwal SP, Kulkarni RR, Deshpande MD, Walimbe PB, Labhsetwar AS. Ocular manifestations of head injury: A clinical study. *Eye (Lond)*. 2005; 19:1257-63.
10. Odebo TO, Ademola-Popoola DS, Ojo TA, Ayanniyi AA. Ocular and visual complications of head injury. *Eye*. 2005; 19:561-66.