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Incidence of Traumatic Optic Neuropathy In Closed head trauma-Review of literature.

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Abstract
Purpose: *Hospital based retrospective study of the incidence of Traumatic Optic Neuropathy in closed head trauma in patients, for a period of 6 months.*
Study design: *Retrospective study.*
Methodology: *This is a Hospital based retrospective study of 100 patients of various age groups with closed head injuries for a period of 6 months from january 2016 to june 2016. Patients in whom TON was suspected, were subjected to visual acuity, slit lamp examination, ophthalmoscopic examination, X Rays, CT Brain, VEP.*
Results and Observations: *Amongst 100 patients, 80 were male, 5 were female, and 15 were children. 20 patients showed Traumatic Optic Neuropathy, out of which, 16 patients were male with a mean age of 30 yrs, 3 were children and one was female.*
Conclusion: *The incidence of traumatic optic neuropathy (TON) in closed traumatic head injury in various studies ranges from 0.5-5%. In our study, TON was observed in closed head injuries involving frontal bone. Motor vehicle and bicycle accidents account for the majority of causes, followed by falls and assaults. Patients with traumatic optic neuropathy can present with a variable degree of vision loss (decreased visual acuity, visual field abnormalities, or loss of color vision). Most cases (up to 60%) present with severe vision loss of light perception (LP) or worse. In the acute phase, the optic nerve usually appears normal on funduscopic examination, but optic nerve atrophy is often seen 3-6 weeks after the injury.*
Keywords: *Traumatic Optic neuropathy, closed head injury, visual acuity.*

I. Introduction

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 closed head trauma[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 orbitofacial or cranial trauma[3],[4]. This impact may generate a shock wave which can lead to optic nerve avulsion or posterior indirect traumatic optic neuropathy[5],[6]. On the other hand, direct traumatic optic neuropathy results from direct trauma to the optic nerve from sharp objects, missiles and bony fragments[7]. The clinical presentations vary. The degree of the visual loss does not always correlate with the severity of trauma. The presence or severity of fractures of the orbit neither directly predicts the severity of visual loss nor determines prognosis[8]. The optimal treatment of traumatic optic neuropathy remains controversial. There has been no conclusive evidence for standardized treatment protocol due to lacking of large randomised control trials of management as a result of low incidence of this condition[9]. Observation, corticosteroids treatment and decompression of the optic nerve have been advocated and significant recovery of vision has been found in those treated with corticosteroids, optic nerve decompression, or both as compared to observation alone[10]. Most cases (up to 60%) present with severe vision loss of light perception (LP) or worse. In the acute phase, the optic nerve usually appears normal on funduscopic examination, but optic nerve atrophy is often seen 3-6 weeks after the injury.

II. Materials And Methods

A Hospital based retrospective study was conducted in100 patients of closed head injury, for a period of 6 months, from january 2016 to june 2016, out of which, 80 were male, 5 were female, and 15 were children. 20 patients showed Traumatic Optic Neuropathy. Out of 20 TON patients,16 were male with a mean age of 30 yrs, one was female and 3 were children. The major causes of trauma were motor vehicle accident (83.3%), followed by blunt trauma (12.5%) and fall (4.2%).

Inclusion Criteria:
Patients of all ages with closed head trauma are included in this study.

Exclusion Criteria:
[1]. Patients having head trauma with penetrating injuries of eye ball,
[2]. Patients who already have low vision due to other eye diseases and,
[3]. Debilitated patients are excluded in the study

III. Methods

All cases subjected to complete eye examination including associated ocular injuries. Ocular Adenexa Examination revealed orbital rim and wall fractures, orbital edema, and lacerations. Visual acuity is normal for all the patients at the time of presentation, and after 3-5 weeks, it gradually reduced from CF 1m to PL. RAPD not detected immediately after injury. It is observed after 3 weeks. Ophthalmoscopic examination is performed with the aid of short acting mydriatic in all stable patients. All patients showed normal fundus picture at the time of injury. Fundus changes started after 3 weeks. The CT scans of brain and/or orbit were reviewed to evaluate the extent of injuries. The treatment regimens given to all patients include intravenous methylprednisolone 250mg *qid* for 3 days followed by oral prednisolone 1mg/kg for 11 days. The visual acuity was the main outcome measure of the study, which was measured by Snellen chart. The visual acuity was assessed at presentation, and after completing corticosteroid treatment. The associated ocular injuries were carefully evaluated.

IV. Results

Amongst 100 patients, 80 were male, 5 were female, and 15 were children (table **1**). 20 patients showed Traumatic Optic Neuropathy, out of which,16 patients were male with a mean age of 30 yrs, one was female and 3 were children(table **2**). Most of the eyes had normal vision at the time of presentation and after 3-5 weeks, it gradually reduced from CF 1m to PL. Periorbital haematoma and lacerations were present in all cases with skull and/or orbital fractures. RAPD not detected immediately after injury. It is observed after 3-5 weeks in all patients with TON. All patients showed normal fundus picture at the time of injury. Fundus changes started after 3 weeks, which showed pale optic disc and absent foveal reflex. Colour vision done after five weeks revealed that there is loss of colour vision in all patients. Majority(15) patients presented with one bony fracture of skull and/or orbit and 5 patients had no fractures.(table **3**). None of the patients had evidence of optic nerve compression on CT scan.

Table[1]

Total number of patients with various age groups with closed head injuries, for a period of 6 months from january 2016 to june 2016.	100
Number of Male patients with closed head injuries, amongst 100 patients.	80
Number of Female patients with closed head injuries, amongst 100 patients.	5
Number of children with closed head injuries, amongst 100 patients.	15

Table [2]

Total number of patients with various age groups with closed head injuries, for a period of 6 months from january 2016 to june 2016.	100
Number of patients with Traumatic Optic Neuropathy(TON)	20
Number of Male patients with TON	16
Number of Female patients with TON	1
Number of Children with TON	3

Table [3]

Out of 20 patients with TON number with skull/orbital fractures	15
Out of 20 patients with TON number without skull fractures	5

Patients treated with intravenous followed by oral corticosteroids had significant visual improvement(P<0.05).

V. Discussion

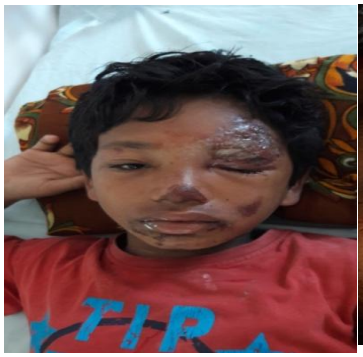
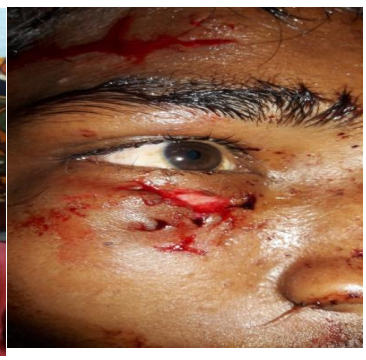
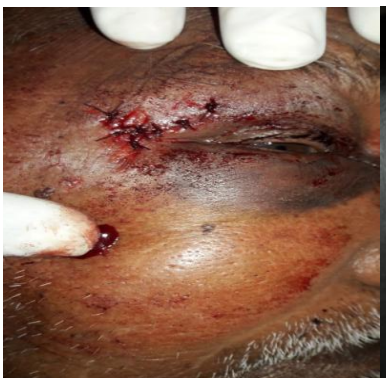
Traumatic optic neuropathy can cause optic nerve morbidity such as loss of vision, deficits in visual field, colour perception and an afferent pupillary defect. The diagnostic features of traumatic optic neuropathy are visual loss that occurs in the presence of a relative afferent pupillary defect without evidence of injury to the optic nerve or eye. The commonest site of indirect optic nerve injury is the optic canal[11]. Chou *et al*[12]had proposed that the damage of optic nerve at microscopic level, including contusion necrosis, nerve fibre tears and nerve infarction secondary to closed space edema, hemorrhage, thrombosis, vasospasm, impingement by bone spicules, and shearing of dural vessels in the optic canal. In an experimental study of primates, significant descending degeneration of the retinal ganglion did not occur until about 3 weeks after optic nerve transection, with maximum loss at 6-8 weeks following the injury.[13]-[16]

All our patients with traumatic optic neuropathy were young male, mostly at age 11 to 30 (58.3%) years old which is consistent with other studies[17]-[19]. Motor vehicle accidents (83.3%) were the main cause of traumatic optic neuropathy in our study, while blunt trauma had the second highest incidence rate (12.5%) and followed by fall (4.2%). Similar findings were showed in Sadeghi-Tari study[3],[8],[9]. The involvement of right and left eyes were almost similar. We followed up the patients for 3 months.

In the present study, the morbidity associated with traumatic optic neuropathy was decreased visual acuity and relative afferent pupillary defect, which was elicited by swinging flashlight test. The ocular manifestations most commonly associated with optic nerve injury were periorbital hematoma, lacerations, and subconjunctival hemorrhage, with no immediate changes in the optic nerve. The ocular manifestations that were observed in our study are consistent with other studies[17],[18]. Most of the eyes with traumatic optic neuropathy were associated with periorbital hematoma and orbital walls and/or skull fractures. All patients had periorbital hematoma with subconjunctival hemorrhage and 84.6% eyes were associated with orbital walls and skull fractures. These signs may indicate the increased morbidity to the optic nerve and strong relation to traumatic optic neuropathy in cases without evidence of optic nerve impingement or compression.

The treatment of traumatic optic neuropathy includes keeping patients under observation, administering corticosteroid therapy, or performing optic nerve decompression with or without steroid therapy[22]. In our study, no surgical optic canal decompression was performed because none of our patients reported having any optic canal fracture or optic nerve impingement in radiological investigation. There is no available optimal management protocol, as most of the published data are either retrospective or presented in case reports[18]. The majority of published data do not clearly define the criteria of visual improvement; some studies defined improvement as an increase in 1 to 3 lines in visual acuity[11],[20]. Methylprednisolone therapy was advocated as the initial treatment of choice because of its neuroprotective mechanism[23]. The exact mechanism of corticosteroids in reducing optic nerve injury is still unclear. Steroids may have the neuroprotective effects of decreasing the intraneural or extraneural edema and relieving compression of the nerve fibres. By reducing vasospasm steroids may also limit contusion necrosis of the nerve and block neuronal death in the setting of trauma is through inhibition of free radicals[9].

In conclusion, most of the traumatic optic neuropathy patients were presented with periorbital hematoma, subconjunctival hemorrhage, orbital wall fractures and lacerations. Patients treated with intravenous methyl prednisolone followed by oral corticosteroids have better visual outcome compared to those under conservative management. The continuation of oral corticosteroid is only beneficial to those eyes with immediate visual improvement after intravenous corticosteroid.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