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Clinical Profile of Traumatic optic neuropathy patients in Tertiary hospital in Hilly region

Dr Ashish Kakkar, Dr Priyanka Gupta, Dr Monica Jain
Corresponding Author- Dr.Ashish Kakkar

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Traumatic optic neuropathy (TON) is caused by acute injury to optic nerve by trauma. TON may be due to direct or indirect trauma. Indirect trauma is more common than direct occurring in 0.5 % to 5 % of all closed head trauma cases.Primary damage occurs when there is an immediate disruption (direct trauma) or shearing (indirect trauma) of retina ganglion cells (RGC) axons. The inflammation and vascular dysfunction that follows gives rise to secondary damage. Patientsoften have elements of both the mechanism. The common causes include road traffic accidents, fall from height, assault.Patient usually presents with sudden loss of vision after trauma.[1 2 3 4] The diagnosis of TON is primarily clinical based on history of acute trauma to head or orbit, decreased visual acuity, presence of relative afferent pupillary defect(RAPD). Visual evoked potential (VEP) may further aid in diagnosis.

In this study we evaluatedthe clinical profile of patients of TON presenting in multi-speciality tertiary hospital in hilly region.

I. Methods

This study was conducted at multi-speciality tertiary referral hospital. Retrospective data was collected from March 2016 to March 2019 of patients which came to eye out patient department (OPD) or eye consultation was sent from neurosurgery department with recent history of eye or head trauma and diminution of vision. All patients underwent thorough history taking and clinical examination. Ocular examination includedvisual acuity, intraocular pressure,anterior segment examination including pupillary examination, dilatedposterior segment examination. CT head and orbit and Visual evoked potential was done in all cases. Patients with other causes of diminution of vision post trauma like vitreous haemorrhage, choroidal rupture, subretinal bleed,macular hole, retinal detachment, cortical blindness, optic nerve avulsion were excluded from the study. All patients which presented between 72 hrs of trauma were given pulse steroids (1g methylprednisolone for 3 days) followed by oral steroids in tapering doses for 11 days after clearance from neuro surgery department.

II. Results

A total of 21 patients were enrolled in the study of which 18 (85 %) were males. Majority of patients(48%) were in age group 21 to 30 years with mean of 27±8.24 years.Most common cause of trauma was road traffic accidents that too while driving two wheelers. Fall from height was the second most common cause of trauma in our study. Most of the patient's 53 %(11) visual acuity was more than 6/60. In three patients (14%) visual acuity was no light perception. RAPD was present in all patients. VEP was abnormal (decreased amplitudes and prolonged latency) in all cases. Most of the patients 17 (81%) presented within 7 days of history of trauma. Four patients(19%) presented for ocular examination after one week of trauma as they had associated head injury too. Associated findings in CT orbit and brain seen were orbital haematoma, fracture of lateral and superior wall of orbit, orbital emphysema,optic canal fracture, subdural haematoma, frontal bone fracture, sphenoid fracture.Eleven out of twenty-one patients received pulse steroids treatment whereas ten patients were not given steroids either due to late presentation or non-clearance from neurosurgeon. No surgical intervention for TON was done in any patient. Nine out of eleven treated and seven out of ten non treated patients showed more than two-line improvement on Snellen's visual acuity chart at 3 months of follow up. Fourteen out twenty-one patients regained more than 6/18 final visual acuity at 3 months.

III. Discussion

Traumatic optic neuropathy though uncommon but can have serious consequences on vision. It can be classified depending on the site of injury (optic nerve head, infraorbital, intracanalicular, or intracranial) or according to the mode of injury (direct or indirect).[4] In direct TON, trauma causes anatomical disruption to the optic nerve, for example, from a projectile penetrating the orbit at high velocity or as a result of optic nerve avulsion . Indirect TON as name suggest is by transmission of forces to the optic nerve from a distant site, without any overt damage to the surrounding tissue structures. The deformative stress transmitted to the skull from blunt trauma is concentrated in the region of the optic canal. The intracanalicular segment of the optic nerve is most commonly involved this form of injury, because the dural sheath is tightly adherent to the periosteum at this specific location.[5] [6] The intracranial portion of the optic nerve in close proximity to the falciform dural fold is the next most common site at risk of injury.[7]Direct and indirect mechanism may both affect the optic nerve in a case and clear distinction may not be possible always.

Reported incidence of TON in various published case series is between 0.7–2.5%.[8] [9] [10] Our hospital is a multi-speciality referral tertiary hospital with lots of patients of trauma being managed in neurosurgery department. Male preponderance in younger age group was seen in our study as seen in other reports also.[10] [12] [13] Road traffic accidents were most common cause followed by fall from heights. Males are usually involved in outdoor activities and driving especially two wheelers in Indian profile. The most common cause of TON is road traffic accidents, followed by fall injury. Other causes include frontal impact by falling debris, assault, stab wounds, gunshot, skateboarding, bottle-cork injuries.

Most of patients presented within 3 days of trauma as associated periocular findings like lid oedema, subconjunctival haemorrhage, racoon eyes made them recognise even the subtle visual changes.

Four patients presented after 7 days as they were admitted in Intensive care units for head trauma or unconsciousness after trauma. It is when they regained consciousness, they realised problems with the vision and further in case of acute trauma addressing the life-threatening complications gains importance over visual problems. All cases were unilateral in our study.

Visual impairment in TON can vary from mild blurring to no light perception. Most patient's 38 % (8 patients) visual acuity in ourstudy was between 6/60 -6/18 followed by 7(33%) patients who had visual acuity between hand movements to 6/60. Three patients were No light perception at presentation.Visual recovery rate of 40–60% has been reported for indirect TON cases managed conservatively, with baseline visual acuity being the most important predictor of final outcome.[13] [14] [15]

Baseline visual acuity has been reported as the one of the prognostic factors in determining final visual acuity. Those presenting with poor visual acuity tend to have poor visual recovery.[15]

RAPD was the most consistent finding and was present in all the cases.

VEP was abnormal in all cases. NO PL eyes showed flat VEP.VEP has been used as an adjunctive investigation apart from clinical examination. The presence and amplitude of VEP has role in predicting long term recovery. VEP testing should be performed bilaterally, using the normal side as a control. When the VEP amplitude is within 50% of the normal side, the patient might have a favourable outcome.[16] **[17] [18]**

On fundus examination optic disc examination showed normal optic disc in 90 % of patients.

Intravenous methylprednisolone was given in 11 patients. remaining 10 were not given either due late presentation or non-clearance from neurosurgeon. There was no statistical difference between treated and non-treated patients in our study. The International Optic Nerve Trauma Study (IONTS) is the largest, prospective, multicentre study of TON published to date.[19] It was intended to be a randomized controlled trial, but it had to be converted to an observational study after 2 years owing to recruitment failure. The analysis included a total of 133 people with indirect TON treated within 7 days of injury and categorized into three groups: untreated (n = 9), steroids (n = 85), or optic canal decompression surgery (n = 33). The majority of patients in the steroid group had either a megadose (40%) or very high-dose regimen (18%), and all the participants in the surgical group, except for one, also received steroids. Follow-up data were available for 104 cases at 1 month and for 40 cases at 6 months. After adjustment for baseline visual acuity, no significant differences were found between the three treatment groups. A three-line increase in visual acuity or more occurred in 57% of the untreated group, 52% of the steroid group, and 32% of the surgery group.[19] The results did not showany increasedlikelihood of visual recovery with higher doses of steroids or with earlier initiation of treatment.Supraphysiological doses of steroids can have negative effect on neuronal survival by suppressing key endogenous neuroprotective pathways.[20]Therefore, a maximum daily dose of 1 g intravenous methylprednisolone has been advocated in TON to minimize the risk of neurotoxicity.

Recent studies have also advocated not giving steroids routinely in all patients with acute traumatic brain injury, as there are reports of increase in mortality in steroid-treated group.[21] The Corticosteroid Randomization After Significant Head Injury (CRASH) trial, was a randomised placebo-controlled multi-centre trial of early steroids in 10,008 adults with head injury, showed that there was a higher risk of death from all causes 2 weeks after trauma in the corticosteroid-treated patients.[22] [23]

The management of TON remains controversial. Some clinicians favour observation aloneothers prefer to intervene with systemic steroids, surgical decompression of the optic canal, or both. There is weak evidenceavailable for various treatment modalities.Routine use of high-dose steroids or surgery in TON has associated risks. There is a relatively high rate of spontaneous visual recovery among patients managed conservatively, and the possible adverse effects of intervention therefore need to be even more carefully considered in the balance. High-dose to megadose steroids are relatively safe, but serious complications can occur and these need to be considered, especially if pre-existing susceptibility factors are present.[21 22 23] The CRASH (Corticosteroid Randomisation After Significant Head injury) showedimprovement in visual acuity in 51.4% cases of the entire study population, which was similar to previous studies that reported 50% probability of improvement in cases with TON. Patients with TON, who sustain indirect trauma, do not lose consciousness, have a good initial VA, have lower grade RAPD, and show signs of visual recovery within 48 hours have a better prognosis.[24] Absence of an optic canal fracture has been found to be a good prognostic factor in some studies but not in others.[25 26]

Shortcomings in our study include small sample size and proper randomisation. Larger trials with a greater number of patients may help us in guiding standard treatment protocols.

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Demographic data of 21 patients of TON

Characteristic	**TON patients**
Age, mean, (SD), Y	27(8)
Sex, No.M: F	18:3
No. of pts (%) with Trauma to presentation interval <1 days 1-3 days 4-7 days >7 days	 3(14%) 8(38%) 6(28%) 4(19%)
Type of trauma, No. (%) Road traffic accident Fall injury Cosco Ball injury Assault	 16(76%) 2(9%) 1(5%) 2(9%)

Visual acuity and IOP of patients with TON

Characteristic	At presentation	At 3 months
Visual acuity No PL <6/60 6/36-6/18 >6/12	 3(14%) 8(38%) 7(33%) 3(14%)	 2(9%) 2(9%) 13(62%) 4(19%)
Mean baseline IOP	16	14

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