Original Research Article

Analytical study of 100 cases of traumatic optic neuropathy

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Abstract

Background: Traumatic optic neuropathy is a devastating potential complication of closed head injury. Approximately 1.5% to 5% of patients with closed head injuries have damage to visual pathways (4-6 / 100,000 general population / yr.). **Aim:** The purpose of this study is to analyse the clinical profile, response to mega dose steroid therapy and visual function outcome in patients with traumatic optic neuropathy presented to the neuroophthalmology clinic at a Tertiary Care Centre. **Methods:** The study was conducted over a period of two years. Patient's visual acuity, pupillary reaction, colour vision, visual field and fundus were assessed. Follow-up examination done at one month, three months, six months and one year. Results: In our study, Out of 100 patients studied, most of them being males (96 %), with road traffic accident being the common mode of trauma with only 25% of them showed visual recovery to some degree with or without treatment with corticosteroids. **Conclusion:** Traumatic optic neuropathy takes important place in permanent loss of vision in young males. Road traffic accidents especially two wheelers are at a particular risk.
Key Word: Traumatic optic neuropathy, mega dose steroid, visual outcome.

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INTRODUCTION

Traumatic optic neuropathy is a devastating potential complication of closed head injury. The hallmark of an optic neuropathy, traumatic loss of visual function, which can manifest by subnormal visual acuity, visual field loss, or colour vision dysfunction[1]. The presence of an afferent pupillary defect strongly suggests a prechiasmal location for the injury and is necessary to validate the diagnosis of traumatic optic neuropathy. Vision loss associated with traumatic optic neuropathy can be partial or complete and temporary or permanent[16]. Approximately 1.5% to 5% of patients with closed head injuries have damage to visual pathways (4-6/100,000 general population/yr) These injuries can be divided into anterior and posterior lesions. Anterior lesions shows ophthalmoscopic abnormalities and usually associated with a variety of easily recognized injuries to the globe. Anterior lesions may include optic nerve avulsion, traumatic anterior ischemic optic neuropathy, anterior optic nerve sheath hematoma[8]. Posterior lesions are free of ophthalmoscopic findings, but disc edema and disc pallor do occur[5,11]. Posterior traumatic optic neuropathy is characterised by visual loss that occur in the presence of an afferent pupillary defect but without evidence of injury to the eye or optic nerve.

METHODOLOGY

A prospective observational case study on the pattern of traumatic optic neuropathy and analysis of clinical profile, response to treatment and visual function outcome was conducted in the department of neuro ophthalmology at Tertiary Care Centre. The study was conducted over a period of two years. The inclusion criteria were patients with history of impact to head and orbit, reduced best corrected visual acuity in one eye,

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relative afferent pupillary defect, defective colour vision, field defects, fundus changes and associated extra ocular muscle palsy, but patients with major head injury, unconscious patients, eyes with penetrating trauma, candidates for decompression surgery, clinical features requiring neurological / neurosurgical interventions were excluded from the study. The patients were followed up at one month, three months, six months and one year. An informed consent was taken from all eligible patients for inclusion in the study. The patient particulars like name, age, sex, address, were documented. A detail history regarding the cause of blindness like time and nature of the trauma, level of consciousness after trauma, site of injury, onset and duration of symptoms and time of presentation, were noted. The patients were also enquired about H/O systemic illness or surgical interventions if any example; Sinus or cranial surgeries which could influence the diagnosis. A patient presented to the neuro ophthalmology clinic has to undergo routine examination of visual acuity, refraction, pupillary reaction, slit lamp examination, fundus, tonometry examination, colour vision, field of vision and extra ocular movements were checked whenever necessary and possible. If visual acuity is poor or patient is unco-operative projection field or at least confrontation field, have been assessed. X-ray skull lateral view, orbit PA view, CT orbit with optic canal axial and coronal view, CT brain plain and contrast were taken when found necessary and possible. Cases were referred to neurology / EN T /orbit department in view of surgical management if required. Patient's visual acuity, pupillary reaction, colour vision, visual field and fundus were assessed during follow-up examination at one month, three months, six months and one year.

RESULTS

From the table 1, it can be seen that traumatic optic neuropathy constituted about 5.2 % of the neuro ophthalmology cases within the specified period of study. Fig 1, shows that in our study 70% belonged to the age group 20 -40years. The minimum age was 12yrs and the maximum age was 52 years. The mean age was 30.22years with SD of 9.28. Out of 100 patients, 96 were male (96%), 4 were female (4%). Fig 2 shows Forehead was the common site of injury (52%) followed by eye brow (33%) and side of face (9%). Of the 100 patients 56% of them had involvement of right eye and 44% of them had involvement of left eye. In our study, mode of injury was Motor vehicle and bicycle accidents for the majority of cases (65%) followed by fall (19%) and others (16%) as shown in Fig 3. Majority of cases presented with immediate onset of visual symptoms (96%). Only 4% of cases presented with late onset of visual symptoms, this is because they would have developed the visual symptoms early but could only noticed it later. Table 2 shows most of the cases presented within 1 month from the onset of symptoms after trauma**.** About 27% of patients had no light perception at the time of presentation. However 8% of patients had better than 6/60 at the time of presentation as shown in table 3.In our study 57% of patients had normal disc. 28% of patients had full pallor of disc and about 15% had partial pallor at the time of presentation as shown in table 4. About 55% of cases presented without any radiological abnormality or fracture.45% had associated orbital, cranial or facial bone fractures evident radiologically. Table 5 shows that among 100 cases, 27 cases were observed, 22 cases were treated with oral steroids, 50 cases were given parenteral steroids followed by oral steroids. One case treated with endoscopic optic nerve decompression. Table 6 shows out of 100 cases only 25 cases showed improvement in visual function (three lines increase in VA in Snellen VA chart), 73 cases had static (patients with no improvement [69] and those with < three lines improvement [4]) and 2 cases had deteriorated visual function, both had poor VA at the time of presentation**.** Out of 72 patients treated with corticosteroid (oral-22, parentral-50) only 23 patients (31.94%) showed visual improvement (oral-3, parentral-20). One patient who presented 1 month after injury had # medial wall of optic canal treated with endo-nasal optic nerve decompression showed visual improvement from 4/60 to 6/36 as shown in table 7. Fig 4 and Table 8 shows Among 100 patients 8 patients had VA > 6/60 and all showed improved visual function with treatment.

Table 1: Incidence

	No of pts	Percentage
Total no of neuroophthalmic cases in the study period	1923	100%
No of traumatic optic neuropathy cases	100	5.2%

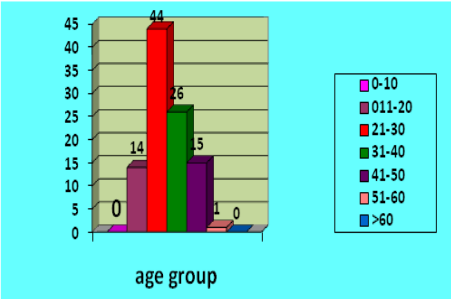

Figure 1: Age distribution

Table 2: Time of presentation

Time of presentation	No of cases	Percentage
0 – 1 week	49	49%
1 week to 1 month	23	23%
1 – 3 months	24	24%
>3 months	4	4%

Table 3: Presenting visual acuity

Visual acuity	Frequency	Percentage
no PL	27	27%
PL +	20	20%
HM + - CFCF	23	23%
1/60 – 3/60	14	14%
4/60 -6/60	8	8%
>6/60-6/18	8	8%

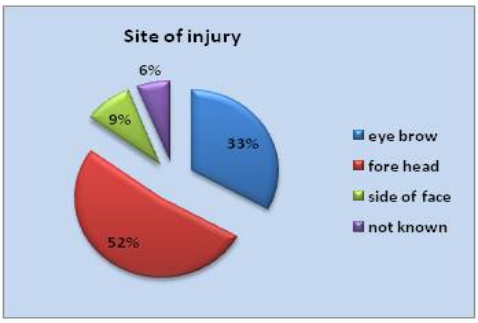

Figure 2: Site of injury

Table 4: Optic nerve head status

Fundus	Frequency	Percentage
Normal	57	57%
Pale disc	28	28%
Partially pale	15	15%

Table 5: Treatment

	Frequency	Percentage
Observed	27	27%
Oral steroids	22	22%
Parenteral steroids	50	50%
Surgery	1	1%

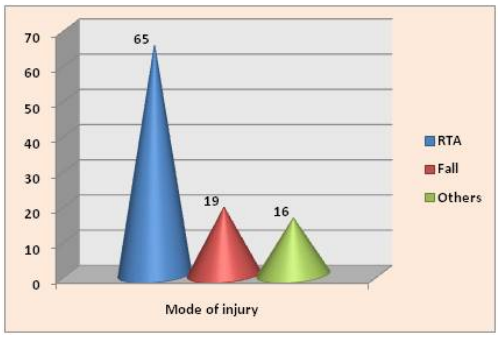

Figure 3: Mode of injury

Table 6: Visual outcome

Static	73	73%
Improved	25	25%
Deteriorated	2	2%

Table 7: Visual outcome based on the treatment

	No of pts treated	No of pts showed improved VA	Percentage
Steroid	72	23	31.94%
Surgery	1	1	100%
Observed	27	1	3.70%

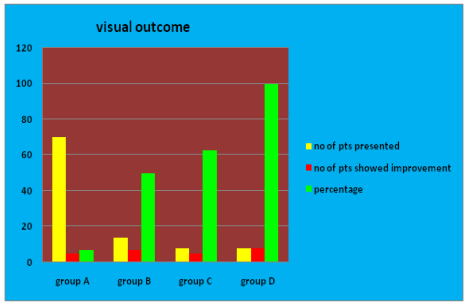

Figure 4: Visual outcome based on presenting visual acuity

Table 8: Visual improvement depending on presenting visual acuity:

	No of pts presented	No of pts showed improvement	%
No PL to HM (group A)	70	5	7.14%
1/60-3/60 (group B)	14	7	50%
4/60-6/60 (group C)	8	5	62.50%
6/36-6/18 (group D)	8	8	100%

DISCUSSION

In our study it is evident that traumatic optic neuropathy is devastating cause of permanent visual loss. Concussive force to head especially forehead transmits shock wave to optic canal. Visual loss is usually instantaneous. Blunt trauma, penetrating injuries and self mutilation are the most common causes of optic nerve injury. Blunt trauma classically occurs following rapid deceleration injuries to the anterofrontal region of the head[23]. Trauma to the outer third of the supraorbital rim is transmitted directly to the optic canal, where the optic nerve is tethered at both ends by dura. Conversely, the optic nerve is not taut in the orbit and protected by orbital fat and resistant to injury at this site. The severity of the trauma does not always correlate with the visual loss. Incidents such as minor fall after tripping or hitting the side of the head against a solid object resulting in a frontal blow are adequate to produce a posterior traumatic optic neuropathy. The most common mechanism of injury is motor cycle accidents[4,13]. Most of them are solo spills. Also the presence of or severity of the orbital fractures neither directly predicts the severity of visual loss nor determines the prognosis. Fracture of the medial orbital wall, floor, zygoma or optic canal may be present[15]. One patient of optic canal fracture may regain normal vision without intervention but another with no fracture may present with no light perception that persists despite all intervention. Traumatic optic neuropathy is most often seen in young males in their 2nd or 3rd decades of life[4]. In one study, 5 patients older than 40 years of age were found to have a worse visual outcome independent of the mechanism of injury, severity of the visual loss or the interventions utilized. Visual deficits range from mild decrease in visual acuity

with subtle field defect to complete loss of light perception. In most cases, the visual loss is severe and instantaneous. Even seemingly trivial trauma may result in dramatic optic nerve impairment. The severity of the visual loss does not necessarily correlate with the degree of overall trauma. Visual evoked potentials (Flash) have limited utility and may give false negative results prior to the onset of optic neuropathy. Although most patients with traumatic optic neuropathy have normal imaging studies, CT without contrast should be performed in all cases. Spiral CT allows rapid data acquisition in uncooperative adults and children. Imaging will allow identification of associated fractures, optic nerve avulsion, transection, optic nerve sheath hematoma and optic nerve compression due to an orbital hematoma. The optic nerve injury may not be isolated. Associated fractures and injuries can be identified when the scope of evaluation is expanded to include otolaryngology, oral, maxillofacial and neurosurgery colleagues. Magnetic resonance imaging is only indicated if intracranial injuries are present that are inadequately detailed with CT imaging. However concurrent orbito-facial fractures, extraocular nerve palsies and type of injury have no relevance to the final visual results. This current study was carried over a period of 2 year; consecutive 100 patients were selected to determine the pattern of disease in a tertiary care centre like our institute. According to Tang[14] (1986), he reported that out of 11patients who received mega dose steroid, 36% showed visual improvement[14]. In our study, this case series has shown that there are 31.94% of patients treated with steroids showed visual improvement. Analysing the demographic details in our study it is seen that most of our patients (70%) were 20-40 years of age. The male patients were predominant in the study. This correlates with the higher rate of motor cycle accidents as the etiology[4]. Traumatic optic neuropathy is a significant cause of post traumatic visual loss. The responsible blunt trauma to the frontal region may be minor or severe and accompanied by multiple adjacent fractures. Careful documentation of visual acuity, pupillary function and red desaturation is essential to guide management. CT imaging should be performed to document such abnormalities such as optic nerve avulsion, optic nerve sheath hematoma, orbital hematoma and optic canal fracture with fragments. If a structural abnormality is present or if the patient's visual acuity deteriorates on steroids optic nerve decompression should be offered[25] Management of this disorder remains very controversial[6]. In summary, a short course of high-dose steroid may be considered unless there is clear evidence of optic nerve transection or avulsion by clinical or radiographic criteria[7].

CONCLUSION

In our study the clinical profile of 100 eligible patients who presented with loss of vision following trauma were analyzed. Considering the overall picture, it is evident that TON takes important place in permanent loss of vision in young males. Current clinical evidence is not sufficient to suggest the treatment option. Future developments in this field require randomised controlled clinical trials with extensive follow up to decide the treatment option. However it is evident that RTA especially two wheelers are at a particular risk and the incidence can be reduced by promoting helmets.

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