Traumatic optic neuropathy in a tertiary eye care hospital of India

Ravindra Kumar Chowdhury[1], Anuradha Pradhan[2,*], Jayashree Dora[3]

[1]Assistant Professor, [2]Senior Resident, [3]Professor, VSS Institute of Medical Science & Research, Burla, Odisha, India

***Corresponding Author:**
Email: dr.anuradha.in@gmail.com

Abstract

Aim: To study the clinical profile and outcome of traumatic optic neuropathy in a tertiary care eye center of India.

Materials and Methods: This prospective study was conducted in 24 eyes of 22 consecutive patients with traumatic optic neuropathy who attended the outpatient ophthalmology department of a tertiary health care center of India from January 2014 to December 2016. The patients were equally divided into 2 groups. Those patients treated with intravenous methylprednisolone with a dose of 1gm for 3 days followed by oral prednisolone with a dose of 1 mg/kg body weight for 11 days were included in group A and those observed with placebo treatment only were included in group B.

Results: Twenty-one patients were males and only one patient was female. The most common age group was 21- 30 years with mean age of 29.29 years. The causes of traumatic optic neuropathy were found to be motor vehicle accident (68.18%) followed by blunt trauma (22.72%) and fall (9.09%). Most of the eyes had the vision of hand movement to no perception of light on presentation (54.16%). The common extraocular associations were the periorbital hematoma (91.66%) and lid laceration (83.33%). Most of the patients (86.36%) were associated with multiple fractures of the skull and orbital bone and 13.63% of patients were not associated with any fracture. Eight of eleven patients (72.72%) in group A had shown 1 line improvement of visual acuity following treatment whereas six of eleven patients (54.54%) of Group B had shown 1 line improvement.The follow-up period of each patient in our study was 6 months.

Conclusion: The common extraocular manifestations observed in our study were the periorbital hematoma, lid laceration, and bony fractures.Traumatic optic neuropathy in our study had a better visual outcome in steroid treatment group than those observed with placebo management.

Keywords: Traumatic optic neuropathy, Corticosteroid, Orbital fracture.

Introduction

Traumatic optic neuropathy can leads to significant vision loss with a relative afferent pupillary defect in an otherwise clear optical media and normal retina.[1-4]

Following orbital trauma, there is an immediate shearing of retinal ganglion cell axons, resulting in a neuronal loss. Within the tight compartment of the optic canal, optic nerve swelling occurs secondary to direct mechanical trauma and vascular ischemia. The ensuing compartment syndrome further impairs the compromised blood supply to surviving retinal ganglion cells leading to apoptotic cell death. Optic nerve decompression is done by medical or surgical methods to break this vicious cycle and to preserve the remaining retinal ganglion cells that survived the initial insult.

The injury to the optic nerve can be either due to direct or indirect mode. Direct injury to the optic nerve can be caused either by sharp objects or bony fragments.It mainly causes a tear or interruption of the nerve.[4] On the other hand, orbito-facial, and cranial injury cause indirect injury by the concussion.[5,6] This leads to a reactionary edema in the optic nerve sheath compromising the vascular and neurotrophic supply of the retinal ganglion cells ultimately leading to retrograde degeneration of the ganglion cells.

There is no standardized treatment protocol of traumatic optic neuropathy till now. Observation, corticosteroids treatment and decompression of the optic nerve are the available treatment modalities.[7] This study was conducted to know the clinical profile and outcome of traumatic neuropathy in this part of India.

Materials and Methods

It was a prospective study including 24 eyes of 22 consecutive patients attending the outpatient department of a tertiary care eye center between January 2014 to December 2016. Informed consent was taken from all the patients for inclusion in the study.

Inclusion Criteria: Traumatic optic neuropathy patients with extraocular injuries

Exclusion Criteria: Patients with intraocular injuries

Complete eye examination was done in all the patients. CT scans of brain and orbit were done to know the extent of injuries including bony fractures.The vision of the patient was assessed with Snellen chart immediately and at 3 months after treatment.

The patients were equally divided into 2 groups. Those treated with intravenous methylprednisolone followed by oral prednisolone were included in group A and those observed with placebo treatment in group B.The dose of intravenous methylprednisolone was 1 gm IV for 3 days and that of oral prednisolone was 1mg/kg body weight for 11 days.

Result

Twenty-one patients were males and 1 patient was female. The age of the patients ranged from 1.5 to 65 years with a mean age of 29.29 years. 21-30 years of age group were commonly involved accounting for

45.45% of study population. The predominant causes of trauma were motor vehicle accident (68.18%), blunt trauma (22.72%) and fall (9.09%).

54.16% eyes had a vision of hand movement to a perception of light on presentation. The periorbital hematoma was present in 91.66% of eyes. 86.3% of our patients presented with the fracture of the skull and orbital bone.No evidence of optic nerve compression on CT scan of brain and orbit was found in any of our patients. All the cases had RAPD (relative afferent pupillary defect) except the two patients with bilateral ocular involvement.

In the group A, the pretreatment visual acuity was between 5/60 to no perception of light in nine cases and 6/12 or better in two cases. In group B, ten cases had visual acuity between 5/60 to no perception of light and no case had a visual acuity better than 6/12.

The patients were followed-up for 6 months in both the group. In group A, 8 of 11 eyes (72.72%) showed one line improvement of visual acuity after treatment. Whereas in group B, 6 of 11 eyes (54.54%) showed one line improvement. Patients treated with combined intravenous and oral corticosteroids had better visual outcome compared to patients kept under observation in our result.

Table 1: Age Distribution of patients

Age group in years	No of patients	Percentage (n=22)
< 10	1	4.54
11- 20	4	18.18
21- 30	10	45.45
31- 40	3	13.63
41- 50	2	9.09
51- 60	1	4.54
>60	1	4.54

Table 2: Cause of Optic neuropathy

Cause	No of patients	Percentage (n=22)
RTA	15	68.18
Blunt trauma	5	22.72
fall	2	9.09

Table 3: Visual status before and after treatment

Visual acuity	Before Treatment		After Treatment	
	Group A	Group B	Group A	Group B
NPL	4	4	1	2
PL	3	1	1	2
HM	0	1	1	1
CF-5/60	2	4	3	5
6/60-6/18	0	1	4	1
>/=6/12	2	0	1	0

Table 4: Extraocular association with optic neuropathy

Type of injury	No of eyes	Percentage (n=24)
Lid laceration	20	83.33
Periorbital hematoma	22	91.66

Table 5: Association of orbital and skull fracture

	No of patients	Percentage (n=22)
Orbital and skull fracture	19	86.36
No bony fracture	3	13.63

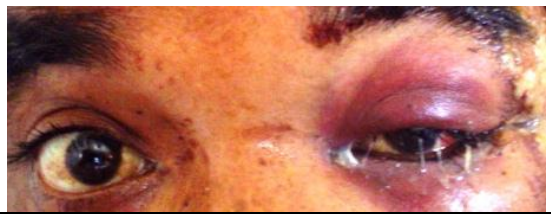

Fig. 1: Traumatic optic neuropathy with periorbital hematoma

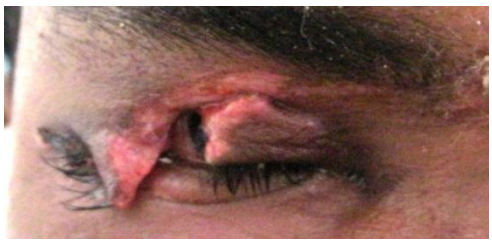

Fig. 2: Traumatic Optic Neuropathy with Upper Lid Laceration

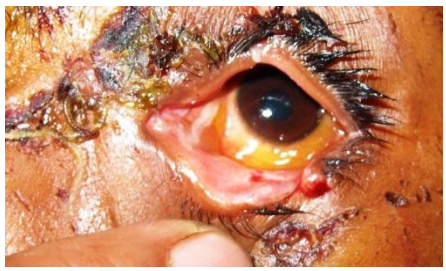

Fig. 3: Traumatic Optic Neuropathy with Lower lid Laceration

Fig. 4: Traumatic optic neuropathy with periorbital ecchymosis

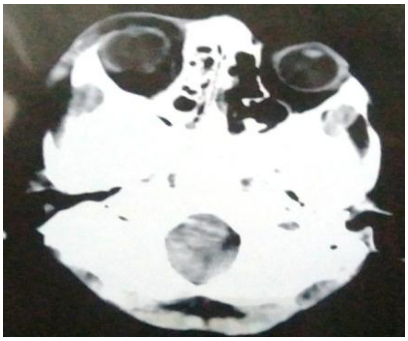

Fig. 5: CT scan of orbit showing fracture of medial wall of left orbit with Traumatic optic neuropathy

Discussion

Optic neuropathy is one of the common cause of blindness in head injury. The mode of injury to the optic nerve in head injury can be either can be direct or indirect. The optic canal is the commonest site of indirect optic nerve injury.[8] Chou et al propose that optic nerve damage can occur due to edema, hemorrhage in a closed space and tearing of dural sheath of the nerve in the tight optic canal.[9]

The age group commonly involved in our study was 21 to 30 years (45.45%) which is consistent with Rajinganth et al and Entezari et al.[10,11] The probable reason for this is reckless driving by this age group of patients. Motor vehicle accidents (68.18%) were the main cause followed by blunt trauma (22.72%) and fall (9.09%) in our study which is consistent with Sadeghi-Tari study.[3,8,9] The most common extra-ocular manifestations were periorbital hematoma and lid laceration which are consistent with other studies.[10,12]

The standard treatment for optic neuropathy includes observation, corticosteroid therapy and optic nerve decompression.[14] The criteria for visual improvement following treatment was not clearly defined in the majority of published data. However few studies showed improvement as an increase in 1 to 3 lines in visual acuity.[8,13] Methylprednisolone is preferred as the initial treatment of choice because of its neuroprotective mechanism.[15] But the exact mechanism of its neuroprotective action is not clear. However, it mainly relieves the compression of the optic nerve fiber by reducing the intraneural and extraneural edema. The other mechanism of its neuroprotective action includes inhibition of the free radicals formation, limitation of contusion necrosis of the nerve and blockage of neuronal death by decreasing the vasospasm.[1] The international optic nerve trauma study showed improvement of visual acuity in 52% of patients treated with high dose of steroids. And in Sadeghi-Tari study, the improvement in visual acuity was 37% in three month follow-up period following megadoses of steroids.[1]

The follow-up period in our study was 6 months. In group A, 8 of 11 eyes (72.72%) had shown at least 1 line improvement of visual acuity and in the group, B 6 of 11 eyes (54.54%) observed with placebo management had shown at least 1 line improvement of visual acuity. The percentage of visual acuity improvement is high in our study because of less number of sample size in our study.

In conclusion, most of the traumatic optic neuropathy patients of our study were presented with periorbital hematoma, lid laceration, and orbital wall fractures. Intravenous followed by oral corticosteroids had better visual outcome compared to those under conservative placebo management.

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