ORIGINAL ARTICLE

Clinical Evaluation and Treatment Outcome of Traumatic Optic Neuropathy in Nepal: A Retrospective Case Series

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ABSTRACT

This study aims to report the clinical features and role of different treatment modalities in final visual outcome in traumatic optic neuropathy (TON). The authors retrospectively reviewed the records of patients with TON over 4 years. There were 37 patients of unilateral TON. Mean age was 28.70 ± 15.20 years (range: 8–90) and 89% ($n = 33$) were males. Road traffic accident was the common cause (43.2%), followed by fall injury (35.1%). There was improvement of visual acuity in 51.4% ($n = 19$) cases. Out of different treatment modalities, high-dose intravenous methylprednisolone (1 g/day) led to significant improvement in final visual acuity ($p = 0.013$). There was no significant improvement in final visual outcome in patients with poor initial visual acuity and those with intracranial injuries.

ARTICLE HISTORY

Received 5 April 2017
Revised 12 May 2017
Accepted 13 May 2017

KEYWORDS

Intracranial injury;
methylprednisolone; oral
steroids; traumatic optic
neuropathy; visual outcome

Introduction

Traumatic optic neuropathy (TON) is an uncommon but often devastating cause of permanent visual loss after blunt or penetrating injury. The incidence of TON after craniofacial trauma has been reported to be 0.5–1.5% in older series.¹ More recent surveys of craniofacial trauma suggest a higher incidence of TON of 2–5%.²

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, and seemingly trivial injuries.^{3–5} TON has also been documented following simple blepharoplasty,⁶ retrobulbar anaesthesia,⁷ orbital and endoscopic sinus surgeries,⁸ and prolonged orthopaedic or neurosurgical procedures with the patient in a face-down position where malposition of the face on the headrest may inadvertently tamponade the globe, on which the entire weight of the head is supported for hours at a time.⁹

Males tend to represent the majority afflicted with TON, constituting 60–95% of cases.^{10,11} The majority of the cases of TON belong to the productive age group of 20–40 years.¹²

Most of the cases have associated closed head injuries, and various studies have shown that loss of consciousness is associated with TON in 40–70% of cases.^{13,14} The hallmark of TON is loss of visual function manifesting as decreased best-corrected visual acuity, loss of visual field or abnormal colour vision, and presence of relative afferent pupillary defect in unilateral TON cases. Electrophysiological tests such as visual evoked potential (VEP) may be useful for diagnosing TON in comatose patients with relative afferent pupillary defect (RAPD) or bilateral cases of TON. VEP is also useful for estimating the visual prognosis.^{3,15,16} Optical coherence tomography may help to monitor axonal loss following TON. Neuroimaging is helpful in localising the site of optic nerve injury. Optic nerve position, orbital haematoma, orbital oedema, intra-sheath haematoma, non-organic foreign bodies, and most importantly bony fractures can be readily recognised by computed tomography (CT) scan with 1.5-mm axial sections. Magnetic resonance imaging (MRI) is more sensitive to reveal associated brain injuries and focal oedema of the optic nerve or optic nerve sheath enhancement with gadolinium.

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The management of TON is controversial.^{3,11,17,18} Various treatment modalities have been tried, including conservative management, megadose steroids, high-dose steroids, low-dose steroids, and surgical management in the form of decompression of optic canal in canalicular fracture and optic nerve sheath fenestration in anterior TON.

Here we describe the clinical findings of TON in patients presenting with craniofacial injuries and the role of different modalities of treatment in final visual outcome.

Materials and methods

In this retrospective study, charts of all patients diagnosed as TON in the neuro-ophthalmology clinic of a tertiary eye centre at Kathmandu during a period of 4 years (from January 2012 to December 2015) were reviewed. A total of 37 patients were diagnosed as TON during this period. All patients who had a history of recent trauma and complained of decreased best-corrected visual acuity and abnormal visual functions such as visual field, abnormal colour vision, or contrast sensitivity in the presence of RAPD were included in our study. In cases with bilateral involvement and where there was doubt about the diagnosis, visual evoked potential (VEP) was done to confirm the diagnosis. Cases with open globe injuries, traumatic cataract, vitreous haemorrhage, retinal detachment, and cortical blindness were excluded.

Detailed history with special attention to mode of trauma, presence of intracranial or other systemic injuries, interval between time of trauma and intervention, history of treatment received elsewhere, and any pre-existing ocular or systemic conditions was noted. Detailed ocular examination was done, including presenting visual acuity, pupillary size and reactions, grading of RAPD, and dilated fundus examination in all cases. Additional tests to assess the function of the optic nerve such as colour vision, contrast sensitivity, and visual field were done whenever possible. Relevant investigations like radiological findings, treatment modalities used, and final visual outcome was recorded in a proforma.

The mode of treatment was decided upon considering both the presenting visual acuity and the patients' choice of treatment. Patients who had relatively poor initial visual acuity were advised for intravenous steroids. All the patients were counselled about the different modalities of treatment and the non-superiority of any modes over other. The final decision was taken considering the duration of trauma, presenting visual acuity, and presence of any contraindication to steroids. Subjects were followed up from at least 6 weeks' to 4 years' duration. For the purpose of analysis, we divided the subjects into three groups. Group 1: Patients who did not receive any treatment; Group 2: Patients who received low-dose steroids (1 mg/kg/day) for 7 days followed by gradual tapering up to a period of 6 weeks; Group 3: Patients who received intravenous high-dose steroids (1 g/day in two divided doses for 3 days) followed by oral dose of 1 mg/kg/day, which was gradually tapered and stopped after 6 weeks. For the purpose of analysis, the visual acuity was converted to logMAR.

Ethical approval was granted by the institutional review board of our institute. The research proposal adhered to the provision of the Declaration of Helsinki. The data were analysed using SPSS version 20 (IBM, Armonk, NY, USA).

Results

There were 40 cases identified with the diagnosis of TON. Out of them, 3 cases were excluded; one was a case of cortical blindness, 1 had associated open globe injuries, and 1 patient who did not have a 6-week follow-up. Thirty-seven cases were included in this study for data analysis.

The mean age of the patients present in the study was 28.70 ± 15.20 years, with the age range from 8 to 90 years. Out of total 37 subjects, 89.2% ($n = 33$) were males and 10.8% ($n = 4$) were females.

Road traffic accidents (RTAs) was the most common aetiology for trauma in 43.2% ($n = 16$) of the subjects. Among the 16 cases of RTA, 10 cases were accidents associated with two-wheelers and 6 cases were four-wheelers. Other aetiologies were fall injury in 35.1% ($n = 13$) where 4 cases

had suffered trivial trauma with minimal external injuries. Physical assaults led to TON in 8.1% ($n = 3$) of the subjects. All the subjects had indirect mode of trauma. Left eye was affected in 56.8% ($n = 21$) patients, whereas right eye was affected in 40.5% ($n = 15$) of the subjects. There were no cases of bilateral TON in our study.

All the patients had complaints of diminution of vision. Other common symptoms included loss of consciousness, headache, redness, and deviation of the eyes. Twelve patients (32.4%) had a history of loss of consciousness after the trauma, 29.7% ($n = 11$) had history of headache, and 13.5% ($n = 5$) of the subjects had deviation of the eyes after the trauma. Mean presenting visual acuity was 2.09 ± 1.60 logMAR in affected eye, ranging from no perception of light in 6 (16.2%) cases to 6/9 in 3 (8.1%) cases. Figure 1 shows the distribution of visual acuity in all the subjects. All the subjects had RAPD on swinging flash light test.

Figure 1. Distribution of presenting visual acuity in the affected eye.

Figure 2. Box plot showing distribution of initial visual acuities (logMAR) among cases with TON with different radiological findings.

Regarding associated ocular findings in subjects diagnosed with traumatic optic neuropathy, 21.6% ($n = 8$) had subconjunctival haemorrhage, and laceration of the eyelids and ecchymosis were present in equal 18.9% ($n = 7$) of the cases, respectively. However, more than one of these ocular findings were present in 24.3% ($n = 9$) of the cases. Regarding the appearance of optic disc at presentation, 20 had normal disc, 6 had optic disc oedema, and 11 cases had varying degree of temporal pallor. Retinal findings were present in 13.5% ($n = 5$) subjects, which included commotio retinae (3 cases), retinal nerve fibre layer oedema (1 case), and Purtscher-like retinopathy (1 case). Cases were divided into four groups on basis of duration from trauma to initiation of therapy, with group 1 duration within 48 hours, group 2 duration from 48 hours to 7 days, group 3 from 7 days to 28 days, and group 4 composed of subjects who underwent therapy greater than 1 months. There were 8 cases (21.6%) in group 1, 13 cases (35.1%) in group 2, 12 cases (32.4%) in group 3, and 4 cases (10.8%) in largest intervals, being group 4.

Radiological investigations were performed in all patients; 33 had computed tomography scans and the remaining 4 had magnetic resonance imaging. These radiological findings were categorised in five subgroups. Table 1 shows detailed imaging findings in all subjects. Radiological studies could not detect optic canal fracture, which is commonly reported in other literature. Figure 2 shows distribution of initial visual acuities among cases with TON with different radiological findings. Cases with orbital bone fractures, intracranial pathology and thickening of optic nerve sheath in radioimaging studies had a poorer initial visual acuity.

Treatment and visual outcome of all cases

Table 2 shows demographic and clinical findings in relation to different treatment methods. Thirty-two subjects were treated for their traumatic optic

Table 1. Radiological findings in patients with traumatic optic neuropathy.

Radiological finding	Frequency (n)	Percentage
Normal	15	40.5
Orbital bone fractures	7	18.9
Facial bone fractures not involving orbit	4	10.8
Intracranial pathologies	8	21.6
Thickening of optic nerve sheath	3	8.1

Table 2. Demographic and clinical findings in relation to different treatment methods.

Characteristic	No treatment	Oral steroids	Intravenous steroids (%)	Total (N = 37) (%)	3-Group p-value*
Age, mean (SD)	29.80 (10.73)	20.71 (7.86)	30.72 (16.99)	28.70 (15.20)	0.046
Sex, n (%)					
Male	5 (15.2%)	6 (18.2%)	22 (66.7%)	33 (100%)	
Female	0	1 (25%)	3 (75%)	4 (100%)	
Cause of injury, n (%)					
RTA	5 (31.2%)	5 (31.2%)	6 (37.5%)	16 (100%)	
Falls	0	2 (15.4%)	11 (84.6%)	13 (100%)	
Assaults	0	0	3 (100%)	3 (100%)	
Others	0	0	5 (100%)	5 (100%)	
Initial visual acuity, n (%)					0.266
NPL	0	0	6 (100%)	6 (100%)	
<1/60 to PL	0	4 (40%)	6 (60%)	10 (100%)	
<3/60 to 1/60	1 (20%)	1 (20%)	3 (60%)	5 (100%)	
<6/60 to 3/60	1 (25%)	0	3 (75%)	4 (100%)	
<6/18 to 6/60	1 (25%)	0	3 (75%)	4 (100%)	
6/18 to 6/6	2 (25%)	2 (25%)	4 (50%)	8 (100%)	

*Kruskal-Wallis test.

Table 3. Comparison of initial and final visual acuities with different modalities of treatment.

Mode of treatment	Initial visual acuity	Final visual acuity	p-value
No treatment	0.86 ± 0.67	0.86 ± 0.69	1.00
Low-dose steroid	2.08 ± 1.57	1.68 ± 1.47	0.300
High-dose steroid	2.34 ± 1.66	1.74 ± 1.68	0.013*

*Significantly different at $p = 0.05$ from normal by paired t test.

neuropathy. Optic nerve decompression surgery was not performed in any subjects. Seven subjects (18.9%) were subjected to low-dose steroid (1 mg/kg/day for 7 days followed by gradual tapering up to a period of 6 weeks), and 25 subjects (67.6%) were subjected to injectable methyl prednisolone (1 g/day in two divided doses for 3 days followed by oral dose of 1 mg/kg/day, which was gradually tapered and stopped after 6 weeks). The remaining 5 subjects (13.5%) were not treated because of the late presentation and onset of optic atrophy.

After undergoing the different modalities of treatment, there was improvement of visual acuity in 51.4% ($n = 19$) of the subjects and no improvement was seen in 48.6% ($n = 18$) of the subjects. Final visual acuity after suitable treatment was 1.61 ± 1.54 logMAR. The improvement in visual acuity

was statistically significant. ($p = 0.006$, paired t test). Table 3 shows comparison of initial and final visual acuities with different modalities of treatment. Out of six subjects with no perception of light, only 2 of the cases (10.5%) improved following medical therapy. Out of 30 patients, 12 subjects (32.4%) had vision better than 6/18 in the affected eye at the most recent follow-up. Table 4 shows final visual acuity for all patients. In relation to the duration from trauma to the initiation of treatment as shown in Table 5, out of 21 patients who presented within 7 days, 13 (35.14%) had improvement in vision and 8 (21.62%) patients did not have any improvement in vision.

Table 6 shows correlation of radiological findings with improvement in vision status. Seven (38.9%) patients having intracranial pathology

Table 4. Final visual acuity for all patients.

Final visual acuity	Observation (n = 5)	Oral corticosteroids (n = 7)	Intravenous steroids (n = 25)	Total (N = 37)
6/6 to 6/18	2	3	7	12
<6/18 to 6/60	1	0	6	7
<6/60 to 3/60	1	1	1	3
<3/60 to 1/60	0	1	2	3
<1/60 to PL	1	2	4	7
NPL	0	0	5	5

Table 5. Final status of vision in relation to the duration from trauma to the initiation of treatment.

Duration	Vision status	
	Improved (%)	Not improved (%)
Within 48 hours	5 (26.3)	3 (16.7)
48 hours to 7 days	8 (42.1)	5 (27.8)
7 days to 1 month	4 (21.1)	8 (44.4)
Greater than 1 month	2 (10.5)	2 (11.1)
Total	19 (100)	18 (100)

Table 6. Correlation of radiological findings with final visual status.

Radiological finding	Vision status	
	Improved (%)	Not improved (%)
Normal	11 (57.9)	4 (22.2)
Orbital bone fractures	4 (21.1)	3 (16.7)
Facial bone fractures not involving orbit	2 (10.5)	2 (11.1)
Intracranial pathology	1 (5.3)	7 (38.9)
Thickening of optic nerve sheath	1 (5.3)	2 (11.1)

have no improvement in vision even after undergoing medical therapy.

Discussion

Injuries leading to TON can be classified as direct or indirect depending on the mechanism of trauma. Direct injuries occur when the optic nerve is injured directly by a projectile, knife, or other object that penetrates the orbit. Indirect optic neuropathy is diagnosed when the injury to the nerve results from the non-penetrating effects of trauma. The mechanisms include trauma by bony fragment, optic nerve sheath haematoma, and concussion injury occurring when the force of trauma is imparted to the skull and transmitted into the optic nerve. All the cases in our study were due to indirect injuries. Generally, direct optic nerve injuries are less common and tend to have worse visual prognosis, which is why more clinical research is done in indirect injuries, where the opportunity for visual recovery is more.^{3,10}

Another way to classify TON is based on the site of injury. Intraocular optic nerve injury resulting from violent rotation of the globe leads to avulsion of the distal end of the optic nerve and usually assumes a typical fundus picture of peripapillary haemorrhage and disruption of the choroid. In the orbit, the nerve is redundant and is cushioned by orbital fat, hence the less chance of

indirect injury. Trauma in this region is mainly due to intraorbital haemorrhage or emphysema causing either ischaemia or elevated intraorbital pressure compromising the circulation of optic nerve known as orbital compartmental syndrome. Intracanalicular injury is the most common site for TON and is associated with high-momentum decelerating injuries, especially in frontotemporal region. Optic nerve is strongly tethered to bone at the orbital opening of the optic canal, in the canal itself, and at the intracranial entrance of the canal. Moreover, the optic canal has a mean subdural cross-sectional space of only 1.84 mm². Thus, even small amounts of bleeding or oedema may infarct the nerve and the fracture of canal may injure the nerve. At both ends of the canal, the nerve is also subjected to shearing forces, because the brain and orbital contents are free to move, but the intracanalicular portion of the nerve is not. The intracranial optic nerve is the next most common site of injury, followed by injuries that also involve the chiasm that produces characteristic visual field changes.¹⁹

Our study had 37 cases of traumatic optic neuropathy in 4 years, which is higher compared with most other studies.^{11,18,20} This may be due to referral bias, as being a multidisciplinary tertiary-level hospital, all patients of craniofacial trauma were sent for early ophthalmological evaluation. Another cause for higher number of TON was the large number of RTAs, especially among two-wheelers in our city. Our results were similar to other studies where males in the productive age group, that is, mainly late 20s to mid-30s, were affected.^{10,11,15,18} Similar to the findings in other studies,³⁻⁵ we found that RTA was the major cause of TON (43.2%), followed by fall injury (38.1%) and physical assaults (8.3%). Fall injury was reported as the most common cause in some reports from India.²¹ Every year, the number of road traffic accident in Nepal is rising, with the highest number reported in the most recent year. Thus, the importance of safety precautions while driving, especially among two-wheelers, has to be advocated. We have come across only unilateral optic nerve injuries, whereas the literature cites the rate of bilateral injuries to be as high as 14%.²¹ Bilateral optic nerve injuries are usually

encountered in high-speed motor vehicle collision or fall injury from height in face-down position where the impact damages both optic nerve.

Most of the studies have reported variable degrees of visual loss that may not always correlate with the severity of trauma. Many studies have found that even trivial trauma following fall injury or RTAs without vehicular collision may lead to severe loss of vision especially if there is impact over the lateral part of the forehead or over the orbital rim.^{15,18} Immediate vision loss was noted in 60% patients and may imply more severe and irreversible damage to the optic nerve such as optic nerve avulsion or transection, whereas delayed-onset visual loss may occur owing to optic nerve oedema and may have a better visual prognosis. However, in cases of children, patients with massive lid swelling or in comatose patients, there might be a delay in detection of visual loss. In our study, visual outcome was similar in both patients who had immediate loss of vision and delayed loss of vision. In all cases, the presence of RAPD observed by a swinging flash light test remains the most useful clinical test to diagnose optic nerve dysfunction. Hence, in any case of trauma to the head or face, a swinging flash light test must be done at the initial presentation for early detection of traumatic optic neuropathy, even in unconscious patients.

Loss of consciousness following trauma was noted in 32.4% of cases, which was similar to the study by Goldenberg-Cohen et al. (34%),¹¹ but slightly less than reported by Mahapatra and Tandon (50%)²¹ and Levin et al. (57%).¹⁸ The most common optic disc finding was a normal looking disc. Those presenting with pallor of the disc were those who had presented relatively later in our study. So although they had better visual acuity at presentation, in spite of the various treatment modalities used, there was no significant improvement in final visual acuity. Patients who had oedematous disc, although they had worse presenting visual acuity, had significant improvement in vision after treatment. Brodsky et al.²² and Goldenberg-Cohen et al.¹¹ have also reported favourable prognosis for visual recovery in cases with optic nerve swelling after blunt trauma. One patient had a finding of Purtscher-like retinopathy

after fall from a tree, and the vision was perception of light present with accurate projection of rays. CT scan showed a swollen intraorbital optic nerve sheath suggestive of optic nerve sheath haematoma. The vision after treatment with oral steroids deteriorated to no perception of light, and optic disc pallor developed.

Positive radio-imaging findings were present in 60% of patients. Four patients had an MRI scan and rest all had CT scans. All the patients who had an MRI scan demonstrated normal findings. MRI scans did not give any added benefit in patients with traumatic optic neuropathy. We tried to analyse the radio-imaging findings with the presenting visual acuity. We found that patients who had worse visual acuity at presentation often had positive findings in imaging studies, ranging from fractures in the orbital bone, intracranial injury, and thickening of the optic nerve sheath suggesting the severity of trauma. However, by radio-imaging study, none of the patients in our study demonstrated fracture in the optic canal, which is reported to be found in 14–44% of cases in other studies.^{11,21} This is probably due to the larger (3 mm) slices taken for the CT scan, which may have missed the fracture of the optic canal. Specifically requesting for acquisition of smaller slices in CT scan may help to visualise the minute details, absence of which may have led to misinterpretation of findings in our study. Various reports²¹ also have noted an underestimation of fractures by a CT scan, which were later discovered on surgical exploration.

Improvement in visual acuity was observed 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.¹⁸ Comparing the different treatment modalities, there was significant improvement in final visual acuity in patients receiving intravenous high-dose steroid compared with the other two groups. There was also improvement in the oral low-dose steroid group, but the data were not clinically significant. Two patients where we started treatment in the form of intravenous methylprednisolone at the fifth week had greater than 2-line improvement in final visual acuity. None of the patients had any major steroid-related side effects in our study. Another important finding in our

study was that out of 8 patients, 7 patients who had intracranial injuries did not have improvement in final visual outcome despite any mode of treatment. Out of the 8 cases of TON with associated intracranial injuries, 2 cases received no treatment, oral steroids was given in 4 cases, and intravenous methylprednisolone in 2 cases. Similarly, out of 3 patients who had optic nerve sheath thickening, only 1 case had 1-line improvement in visual acuity. 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.^{3,23} The Corticosteroid Randomization After Significant Head Injury (CRASH) trial, 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.²³ Individuals with traumatic optic neuropathy often have concomitant head trauma. High-dose corticosteroids for traumatic optic neuropathy may result in a measurable loss of life in patients who also have a brain injury.³

Similar to the findings in other studies, the patients with poor initial vision had worse visual outcome irrespective of any of the treatment modalities.^{10, 11} VEP was not done in all patients. We could retrieve VEP findings of only 9 patients. The findings in VEP were abnormal, showing prolonged latency in all 9 cases with reduction in amplitudes in 5 cases. Several studies have noted that presence of a normal or even abnormal but detectable VEP was associated with a high chance of visual recovery, whereas an absent VEP at presentation was almost never associated with substantial improvement.²¹

We tried to analyse the improvement in visual acuity in relation to the duration from trauma to the initiation of treatment. We did not find any significant difference in visual outcome in patients who received early treatment. Out of 4 cases, 2 cases had improvement in visual acuity after receiving steroid even after 4 weeks of trauma.

Any patient with traumatic optic neuropathy, especially when there is associated head injury, requires a comprehensive work-up with collaboration of the trauma physician, head and neck

surgeon, ophthalmologists, and neurosurgeons. The choice of treatment depends on the individual patient and the treating physician. However, a trial of high-dose steroid can be given in patients without significant head injury. Our sample size was small and being a retrospective study, there was inherent selection bias in the study. Based on our finding, we may not be able to set future recommendations; however, these observations are helpful and add to the literature about the clinical pattern and treatment outcome of traumatic optic neuropathy in Nepal.

Conclusion

Traumatic optic neuropathy can lead to severe and permanent visual loss in young male individuals sustaining head trauma. Poor initial visual acuity and intracranial injuries are predictors of poor final visual outcome. High-dose intravenous steroid improves final visual outcome in patients with traumatic optic neuropathy provided that intracranial injury is ruled out.

Limitations

This was a retrospective study with flaws in the study design, as it was not controlled or randomised and the selection bias for method of treatment could not be removed. There were small numbers of patients in the study groups.

Acknowledgements

The authors thank the medical record unit of B. P. Koirala Lions Centre for Ophthalmic Studies for providing the records of patients with traumatic optic neuropathy.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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