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A Case Series on Traumatic Optic Neuropathy

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Abstract: *BACKGROUD: Traumatic optic neuropathy (TON) refers to an acute injury of the optic nerve secondary to trauma. The optic nerve axons may be damaged either directly or indirectly and the visual loss may be partial or complete. Optic nerve decompression with steroids or surgical interventions or both has therefore been advocated as a means of improving visual prognosis in TON.OBJECTIVES:The aim of the study is to retrospectively analyse the most common cause ,age group ,gender,fracturerate,presenting visual acuity and postmedicaltreatment visual acuity.METHODS: Inclusion criteria:isolated traumatic optic neuropathy with atleast one follow up visit.A retrospective study of 50 patients with isolated traumatic optic neuropathy with atleast one followup was done.They were given iv methylprednisolone,iv steroids and oral steroids and tab.methycobalamine based on severity at the time of presentation.RESULTS:Most common gender affected were males(98%) than females.Most common age group was 33yrs(range 4yrs to 66yrs) and cause was roadtraffic accident around 81.6%,fracture rate about 53%.Left eye was most commonly affeceted (around 60.4%).22% presented with vision with No PL,24% from PL+ to (½)/60,20% with vision 1/60 to 5/60,27% presented with vision ranging 6/60 to 6/9,6% with 6/6.Post treatment vision ranged:10%-NO PL,24%-PL+ to (½)/60,24%-1/60 to5/60,18%-6/60 to 6/9,22%-6/6*

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I. Introduction

1. Classification

Traumatic optic neuropathy (TON) refers to any insult to the optic nerve secondary to trauma. It can be classified depending on the site of injury (optic nerve head, intraorbital, intracanalicular, or intracranial) or according to the mode of injury (direct or indirect).[1,2] In direct TON, there is significant anatomical disruption to the optic nerve, for example, from a projectile penetrating the orbit at high velocity (Fig. 1), or as a result of optic nerve avulsion (Fig. 2). Indirect TON is caused by the 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 particularly susceptible to this form of injury, because the dural sheath is tightly adherent to the periosteum at this specific location.[3,4]The intracranial portion of the optic nerve in close proximity to the falciformdural fold is the next most common site at risk of injury.[5] In one report using computerized tomography (CT) imaging, about half of all TON cases were found to have an associated sphenoidal bone fracture, an indirect measure of the significant compressive forces involved at impact.[6] However, both direct and indirect mechanisms can contribute to optic nerve damage, and a clear distinction is not always possible.

II. Materials And Methods

In this retrospective study, all patients diagnosed as isolated TON during a period of 1 years (from January 2018 to December 2018) were reviewed. A total of 50 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.

Detailed history with special attention to mode of trauma, presence of intracranial or other systemic injures, 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 6months duration. For the purpose of analysis, we divided the subjects into three groups. Group 1: Patients who did not receive any treatment but ony tablet methylcobolamine as placebo; 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.

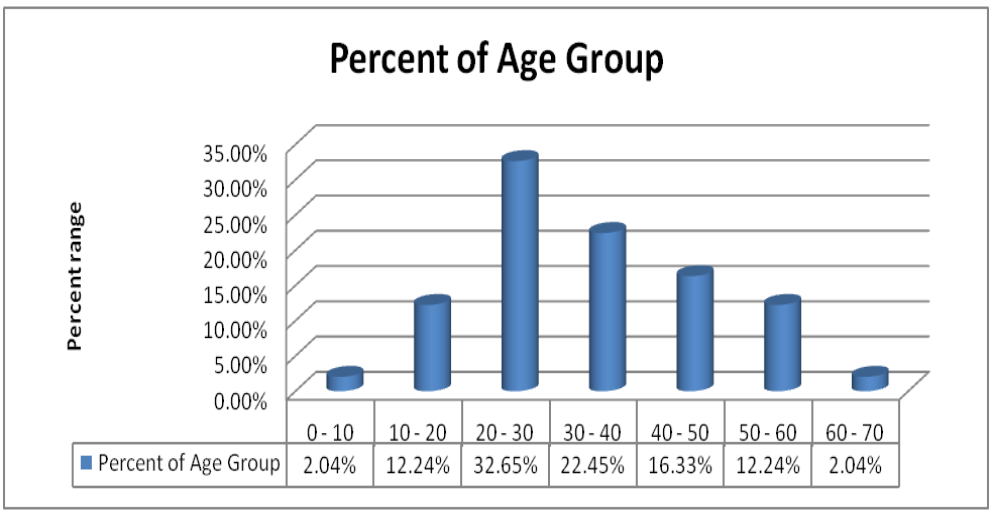
III. Results

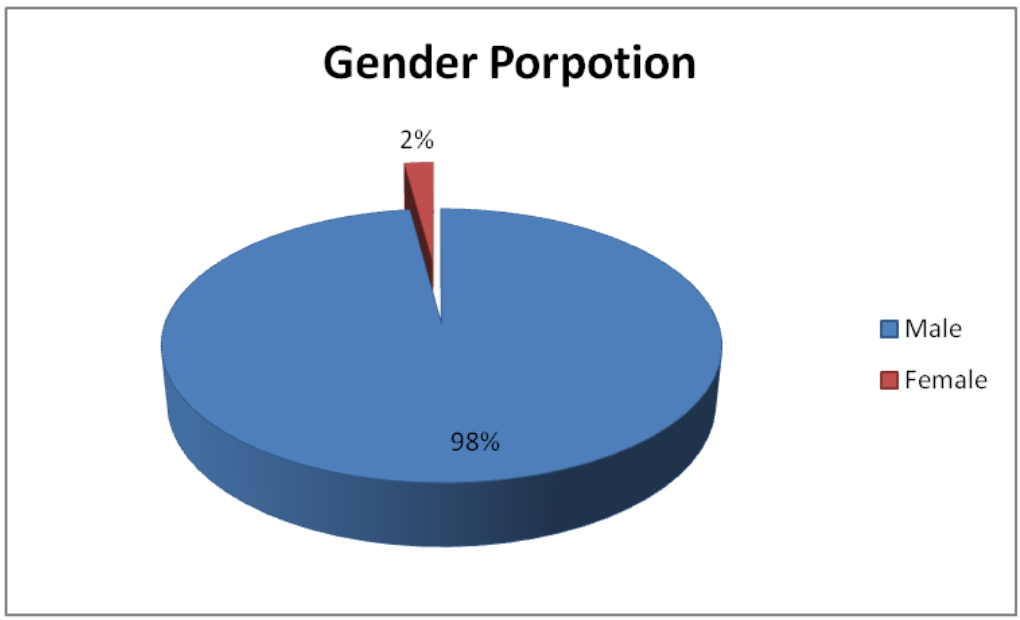
There were 50 cases identified with the diagnosis of isolated TON. 2patients were excluded because of lack of followup.

The mean age of the patients present in the study was 33.37years, with the age range from 4 to 66years. Out of total 50 subjects, 97.96% were males and 2.04%were females.

R.% (*n* = 19) of the subjects. There were no cases of bilateral TON in our study.Fracture rate was 58(52.25%)

All the patients had complaints of diminution of vision. Other common symptoms included loss of consciousness, headache, redness, and deviation of the eyes.Presenting visual acuity in affected eye was 1/2/60 to no PL in 24(48.98%),5/60 to 1/60 in 7(14.29%),6/60 to 6/9 in 16(32.65%) and 6/6 in 2(4.08%).





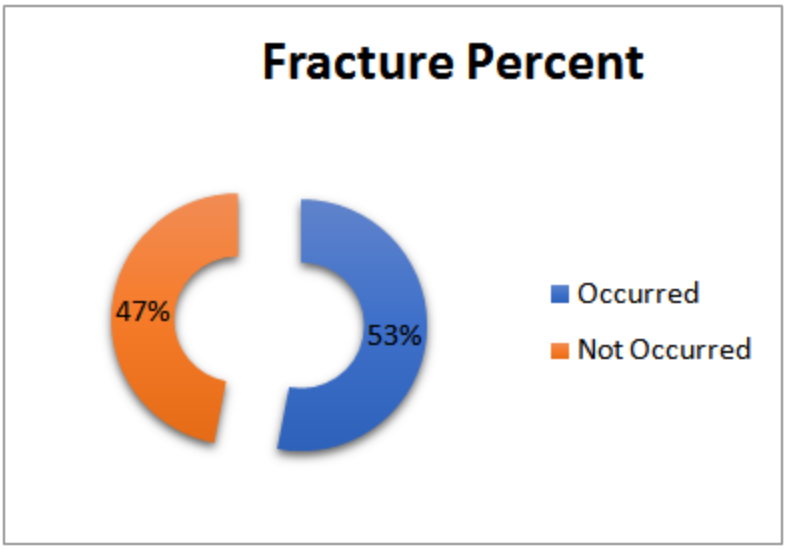


Figure 1 shows the distribution of visual acuity in all the subjects. All the subjects had RAPD on swinging flash light test.
Fig1

Eye(1 missing)	
Right Eye	19(39.58%)
Left Eye	29(60.42%)
Pre Visual Acuity	
6/6	2(4.08%)
6/9 – 6/60	16(32.65%)
5/60 – 1/60	7(14.29%)
½,60 -NOPL	24(48.98%)
Post Visual Acuity	
6/6	11(22.45%)
6/9 – 6/60	10 (20.41%)
5/60 – 1/60	12(24.49%)
½,60 –NOPL	16(32.65%)
NA	

IV. 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 peripapillaryhaemorrhage 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 intraorbitalhaemorrhage 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 mm^2. 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]

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.

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.

Positive radio-imaging findings were present in 60% of patients. 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 reports[21] also have noted an underestimation of fractures by a CT scan, which were later discovered on surgical exploration.

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],

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.

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V. 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.

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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.

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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