Indirect Traumatic Optic Neuropathy- Retrospective Interventional Case Series from a Tertiary Care Center in Eastern Nepal

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ABSTRACT

To report the demography, nature of insult and ocular characteristics in patients presenting with indirect traumatic optic neuropathy and to evaluate the visual outcome with mega dose methylprednisolone therapy.

Retrospective hospital data analysis of all patients admitted with indirect traumatic optic neuropathy and treated with mega dose methylprednisolone therapy in the last three years was conducted.

Nine patients (M:F= 8:1) were identified with unilateral optic nerve injury. Road traffic injury was the most common cause of injury.

Lid ecchymosis and swelling on the same side was the most frequent ocular abnormality noted, followed by loss of consciousness.

Time of presentation varied from 3 hours to 11 days. All patients except two had visual acuity of no light perception at presentation. Following therapy there was improvement in visual acuity in two patients who presented within two days of injury.

Patients present late with traumatic optic neuropathy in this hospital. Most of the patients are with no light perception. High dose steroids was found to be beneficial only in patients presenting early (<two days).

Key Words: Traumatic Optic neuropathy, indirect, direct injuries, methylprednisolone, visual.
INTRODUCTION
Traumatic optic nerve injury is divided into direct and indirect types, depending on whether the optic nerve is damaged due to the contact with the agent of trauma or not.¹

Most of the studies related to traumatic optic nerve injury have been done on the indirect type of optic nerve injuries. But still the pathogenesis and thus the management options of this type of injury are unclear.²

Various modalities described in the management of indirect optic nerve trauma are observation, steroids, surgical decompression of optic nerve canal or various combinations of these.³

Though none of the treatment modalities till date have been shown to be superior to the other, much work has to be done to find out the best modality or other newer possible options for such injury.³

There has been no previous report on the profile and management outcome of indirect optic nerve injuries with megadoses of steroids from Nepal. This article is our experience in management of such injuries in the eastern region of Nepal.

MATERIALS AND METHODS
Retrospective analysis of hospital (B P Koirala Institute of Health Sciences, Dharan, Nepal) data of all patients admitted with the diagnosis of indirect type of traumatic optic neuropathy and treated with mega dose methylprednisolone was carried out for the last three years.

Diagnosis of traumatic optic neuropathy was based on acute decrease in vision following trauma associated with relative afferent pupillary defect (RAPD) and or loss of colour vision in Ishihara's pseudosietochromatic chart or visual field defect. In all cases computed tomography (CT) scan was done. Those patients not having a CT scan after injury or showing abnormality in the optic nerve were excluded.

According to the protocol of our institute, we included all patients presenting within seven days of injury for mega dose methylprednisolone therapy. One patient who presented after 11 days of injury was also included. In all cases steroid therapy was initiated within one hour of presentation to the hospital.

Patients admitted in other specialties and having multiple injuries were not included due to non-availability, initial evaluation and proper follow-up.

Also excluded were the patients who declined to receive therapy or did not complete the full dose of steroid. (One

<table>
<thead>
<tr>
<th>S.N.</th>
<th>Age/sex/ eye</th>
<th>Nature of trauma</th>
<th>Time of Presentation</th>
<th>Associated Ocular injury</th>
<th>Other Injuries</th>
<th>Visual acuity at presentation</th>
<th>Visual acuity after steroid therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40/M/OD</td>
<td>Fall from height</td>
<td>6day</td>
<td>Periorbital swelling, lid oedema, ecchymosis,</td>
<td>-</td>
<td>NPL</td>
<td>NPL</td>
</tr>
<tr>
<td>2</td>
<td>20/M/OS</td>
<td>Trauma with nail</td>
<td>10days</td>
<td>Periorbital swelling, lid oedema, ecchymosis</td>
<td>-</td>
<td>NPL</td>
<td>NPL</td>
</tr>
<tr>
<td>3</td>
<td>45/M/OS</td>
<td>RTA</td>
<td>5days</td>
<td>Lid laceration</td>
<td>-</td>
<td>NPL</td>
<td>NPL</td>
</tr>
<tr>
<td>4</td>
<td>7/M/OD</td>
<td>Fall from bike</td>
<td>2days</td>
<td>Mild Lid oedema, Berlin’s oedema</td>
<td>-</td>
<td>NPL</td>
<td>1/60</td>
</tr>
<tr>
<td>5</td>
<td>30/F/OD</td>
<td>RTA</td>
<td>6days</td>
<td>Subconjunctival haemorrhage, Berlin’s oedema.</td>
<td>-</td>
<td>NPL</td>
<td>NPL</td>
</tr>
<tr>
<td>6</td>
<td>45/M/OD</td>
<td>Assault</td>
<td>3 hrs</td>
<td>Lid laceration, Subconjunctival haemorrhage</td>
<td>#Medial wall orbit (Undisplaced), Haemoinus maxilla</td>
<td>6/60</td>
<td>6/18</td>
</tr>
<tr>
<td>7</td>
<td>40/M/OS</td>
<td>RTA</td>
<td>3 days</td>
<td>Laceration temporal side, lid oedema, ecchymosis,</td>
<td># temporal bone (Undisplaced )</td>
<td>NPL</td>
<td>NPL</td>
</tr>
<tr>
<td>8</td>
<td>35/M/OS</td>
<td>RTA</td>
<td>2 days</td>
<td>Lid oedema</td>
<td>-</td>
<td>NPL</td>
<td>1/60</td>
</tr>
<tr>
<td>9</td>
<td>42/M/OD</td>
<td>RTA</td>
<td>2 days</td>
<td>Lid oedema</td>
<td>Extrudal haemorrhage</td>
<td>NPL</td>
<td>NPL</td>
</tr>
</tbody>
</table>

• M - Male
• F - Female
• RTA - Road traffic accident
• NPL - No perception of light
• # - Fracture
patient was referred to another center on request, after 24 hours of therapy). Patients having direct trauma to optic nerve, open globe injuries or trauma to the posterior segment preventing evaluation of disc or where diagnosis of optic nerve trauma couldn’t be made with certain- 

In all the patients, after obtaining informed consent and complete initial ophthalmic and systemic evaluation, i.v. methyl prednisolone was started in a loading dose of 30 mg/kg over 30 minutes followed by 5.4 mg/kg/hr for 48 hrs. Following this, they were prescribed a tapering dose of oral steroids (tab prednisolone 1mg/kg/day) for two weeks. In all patients, visual acuity, colour vision, RAPD and optic disc evaluation were done on the first, second and third days. Patients were discharged on the third day, and were asked to follow up at two weeks, one month and three months.

RESULTS

We encountered nine patients in the last three years who fulfilled our inclusion criteria. The details of the patients are enumerated in the Table I.

The mean age of the patients was $33.78 \pm 12.84$ years (Median = 40 years) with the gender ratio of male: female being 8:1. The right eye was more frequently involved (R:L = 5:4). None of the patients had bilateral injuries.

Road traffic accident was the most common (five cases) cause of trauma. Seven out of the nine patients noted loss of consciousness for a brief period after the injury though none except one had any head injury as evident on CT scan. Two patients, one each, had undisplaced fracture of the medial wall and temporal wall of the orbit.

Except for one patient, all the patients had some form of lid injury. Six patients had lid ecchymosis and three had laceration involving the lids on the same side.

The time of presentation varied from three hours to 11 days following injury (mean = 4.01 \( \pm \) 3.02 days).

At the time of presentation only two patients had visual acuity more than light perception. The rest presented with no light perception.

Three days following the steroid therapy, two patients had improvement in visual acuity. Patient No. 4 and 6 had improvement in visual acuity to finger counting 1 meter and 6/18 respectively.

None of the patients receiving steroids reported any side effects due to steroids.

All the patients were followed-up till three months, except patient No. 6 who did not come for follow-up after one month. At the time of the last follow-up, his visual acuity was 6/18 and there was defective colour vision along with superior altitudinal visual field defect in goldmann perimetry. The result of the examination of the disc was within normal limits, except for some pallor on the temporal side.

Patient No. 4 developed disc pallor during follow up and vision remained to counting fingers till one meter.

The rest of all the patients at three months of follow-up were found to have different degrees of disc pallor and RAPD without any improvement in visual acuity.

DISCUSSION

This limited case series indicates that the patients in this region present late following injuries and most of the patients referred to this hospital have severe vision loss at presentation. Visual recovery was observed only in patients who presented early (within two days) or had a better vision at presentation.

Because the optic nerve at the optic canal is thought to be analogous to the spinal cord, management of indirect optic nerve injuries is thought to be similar to that of acute spinal injuries.\(^1\)

National acute spinal cord injury study (NASCIS II & III) described the beneficial effect of high doses of methylprednisolone in the treatment of acute spinal injuries. The beneficial effect of this therapy was evident when patients were treated within eight hours of injury and for 24-48 hrs.\(^4,5\)

Another drug tried is Tirilazad mesylate, which is a synthetic steroid, but it was not found to be as effective as methylprednisolone.\(^6\)

Experimental studies on the other hand have shown evidence that high-dose methylprednisolone can actually decrease the number of the axons in the optic nerve following crush injuries.

The international optic nerve study group, the only multicentric study, after gathering samples from multiple centers treating optic nerve injuries, failed to come to a conclusion as to whether optic canal decompression,
steroids or just observation is superior. They finally concluded that none can be taken as a standard line of therapy.\textsuperscript{3}

Here at our center, optic nerve decompression is not routinely performed and high dose steroid therapy is preferred for the consenting patients.

**PATIENT’S CHARACTERISTICS**

Our institute is situated at the eastern part of Nepal and is the only referral hospital for patients with such injuries. Various factors might be responsible for the delayed presentation at our center, mainly lack of proper transportation and poverty. It is also likely that the health personnel who evaluate these patients at the peripheral centers might not be well-trained enough to recognize this type of injuries and refer them. This might be one reason why we have a high number of patients with no perception of light in our series. Referral of patients with moderate or mild decrease in vision may be delayed because of the other co-existing injuries (periocular and intraocular). It is also likely that those patients experiencing some visual improvement as suggested by some studies during the initial period might wait for a longer period before presenting with visual loss.\textsuperscript{3}

**VISUAL ACUITY**

It is suggested that patients having indirect optic nerve trauma can either have an axonal transection injury or there is loss of axoplasmic transport. Only the latter type of injury is however thought to recover since transected axons are thought to regenerate only under unusual circumstances.\textsuperscript{3,8,9}

Various mechanisms have been proposed for the loss of axoplasmic transport and all the current therapies are directed towards its improvement.

It has been proposed that indirect injury to the optic nerve is due to vasospasm and swelling within the optic canal. Experimental studies suggested that several cellular messengers are liberated which leads to liberation of oxygen-free radicals. Oxygen free radicals are responsible for peroxidation of lipid cell membranes of the axons. It has also been shown that bradykinin and kallidin are activated following injury; these agents influence free-radical production, intracellular calcium production and arachidonic acid release from neurons. Cell-mediated inflammation, which occurs following injury, also has a prominent role in experimental models of optic nerve injuries.\textsuperscript{10}

Megadose corticosteroids are thought to act as a neuroprotective agent by preventing free-radical-induced damage to the axonal membrane.\textsuperscript{11-13}

It is also known that the loss of axoplasmic transport for a longer time may result in apoptosis of retinal ganglion cells after which the axoplasmic transport will not improve.\textsuperscript{3,14}

Considering these factors, it seems that there should be a fixed time frame after which any type of therapy will not be beneficial, though this was otherwise concluded by “The international optic nerve study group”.\textsuperscript{3}

High dose steroids as used in NACIS 2 regimen is reported to be tolerated well without significant side effects. Adverse effects of high dose pulse steroids mentioned in the literature include gastrointestinal side effects, steroids induced lethargy, psychosis, seizures and steroid induced cardiac conductive abnormality. Aseptic necrosis of joints, impaired wound healing, immunosuppression and glucose intolerance observed in patients on prolonged treatment with steroids are not reported in patients treated with megadose steroids.\textsuperscript{15}

In our short series, it seems that the patients presenting early (with in 48 hrs) benefited from the megadose steroids and thus early presentation could be a factor responsible for better outcome. As this is a non-randomized short series, any conclusion has to be drawn with caution.

One patient, who had improvement in vision, also had Berlin’s oedema. Though there was no change in the appearance of retinal whitening after the high dose steroids, it is likely that there will be some decrease in retinal oedema with steroids. We, however, have not come across any report stating improvement in vision following high dose steroids after Berlin’s oedema.

We are however confident that the other patient benefited from steroids since he did not have any other associated injuries preventing him to see.

**NASCIS-II & III regimen is recommended for all patients presenting within eight hours of spinal injury; this was however, not stated for patients with traumatic optic neuropathy.**\textsuperscript{4,5} However, it is reported in some series that visual improvement following these injuries is less likely after seven days.\textsuperscript{3}

It is also not known which patients will gain from this type of therapy, since it has been shown that patient’s
characteristic, mechanism of injury, duration of injury and severity of visual loss do not predict the beneficial effect of steroids.3

On the other hand, Carta, Ferrigno et al suggested in their series of 35 cases that blood in the posterior ethmoidal cells, age > 40 years, loss of consciousness and absence of improvement after 48 hours of steroid therapy related to poor visual outcome.16 In our series we found younger age, early presentation (< 48 hours) and therapy relaxes to better visual outcome.

CONCLUSION
Patients in Eastern Nepal having traumatic optic neuropathy often present with severe vision loss. A large randomized study is required to conclude whether any form of therapy is beneficial in these patients.

REFERENCES: