Editorial

Current Management of Optic Nerve Injury

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Traumatic optic neuropathy (TON) denotes an acute injury to the optic nerve (ON) secondary to direct or indirect trauma. Direct trauma occurs usually due to penetrating injury causing shearing or hematoma of the ON. Indirect injury results from transmission of forces to optic apparatus after a blunt trauma.[1,2] The nerve in such injuries is usually injured at the transitions between mobile and fixed (intraorbital and intracanalicular) segments. The most common site of injury is intracanalicular (71.4%) followed by orbital apex (16.7%).[3] ON is tightly adherent to the dural sheath and periosteum in the intracanalicular portion. This makes it highly susceptible to deformative stresses of the skull bones, during injury. In the intracranial compartment, the most common site of injury is in the anterior cranial fossa, where ON is in proximity to falciform dural ligament.[4] Around two-third of TONs are associated with significant head injury, whereas only 2.3% patients with head injury have TON. Exact incidence of TON in head injury cases is difficult to ascertain as many patients with severe injury die or remain in comatose conditions. However, literature review reveals, reported incidence of 0.4%. Injury is more commonly reported in young males. Approximately 79% males with a median age of 31 years were reported to have suffered with TON in a series of 121 cases of British Study.[5] A pediatric study also reported similar findings of 60% males with mean age of 11.6 years.

Secondary injury to the ON can result from sequelae of acute injury, leading to edema of ON, which can cause retinal ganglion cell degeneration and further decrease in vascularity. Surgical optic canal decompression and use of steroids in such cases may be helpful. Classification for TON is summarized in ►Table 1.

TON patients usually present with uniocular or binocular decrease in vision following an injury. Initial diagnosis may be delayed in the setting of severe head injury and comatose patients. Therefore, a careful ophthalmologic examination is necessary in all cases of head injury. Presence of hematoma, laceration, raccoon eyes, swelling, and a bony step should be documented. Wherever feasible, visual acuity should be evaluated. In case of comatose patients, presence of relative afferent pupillary defect is a pathognomic finding. This finding may be absent in symmetrical bilateral TON. Impaired color vision and variable visual field defects may be documented in conscious patients. Fundus examination is necessary. It may reveal ON head swelling and adjacent retinal hemorrhages. It may also help in ruling out preexisting optic neuropathy in acute settings. Presence of normal ON head does not necessarily rule out TON, as optic atrophy findings may appear after 4 to 6 weeks. Approximately 89% patients of TON were reported to be having normal fundus in a study of 27 patients.[6]

At center, neuroimaging is performed for all cases of suspected TON. Primary investigation of choice in acute settings is computed tomography (CT) scans. It reveals the bony injury and is also helpful in quickly assessing for any intracranial and maxillofacial injuries.[7] CT is adequate for most penetrating foreign-body injuries, except glass and occasionally wood. Ultrasound may not be useful in an open globe injury.[8] Doppler study of the central retinal artery has been shown to have altered hemodynamics in TON.[9] Magnetic resonance imaging (MRI) is contraindicated in cases of metallic (ferromagnetic) penetrating injury. Diffusion tensor imaging can help in delineating TON.[10]

Visual evoked potential (VEP) helps in objectively assessing the ON and visual pathway, particularly in comatose patients or patients with bilateral TON. It is also a useful tool in predicting the prognosis. Absence of VEP portends a poor prognosis. Amplitude within 50% of normal is considered as favorable. Practical use of VEP in comatose patients is limited

Table 1 Classification of pathology of optic nerve injury

A. Primary pathology	• Concussion • Avulsion or tear • Contusion • Hemorrhage (intraneural/extraneural)
B. Secondary injury	• Edema • Ischemia • Microvascular thrombosis • Infarction of the nerve

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as shifting of equipment or patients for this test is not always feasible. Moreover, intracranial injuries along the visual pathways may lead to alteration of findings.[8,11]

Optical coherence tomography can show thinning of retinal nerve fiber layer. Its usefulness is limited to documenting the progression, over long-term follow-up.[12]

Treatment of TON is controversial to say the least. Even after multiple approaches being used, prognosis remains guarded for severely injured. Management options range from conservative follow-up to use of steroids or surgical decompression.

We prefer to conservatively follow up all patients with good baseline visual acuity, having no history or surrogate markers of severe head injury. Literature search reveals, that results of conservative follow-up are comparable to use of steroids or surgery or both.[8]

Steroid use in TON was based on its documented usefulness in acute spinal cord injury (SCI). However, many studies, including the NASCIS (National Acute Spinal Cord Injury Study) trials, have failed to prove its efficacy for SCI. A randomized, placebo-controlled, double-blind trial has similarly failed to prove the usefulness of high-dose corticosteroid treatment of recent TON when compared with a placebo.[13] Similar findings have been reported in two other studies as well.[6,14] An animal study in rat has proved the futility of steroid use (high, very high, and megadose of methylprednisolone) by showing that rats with similar injuries, when treated with saline instead of steroids, had higher number of axons and there was a dose-dependent decline with use of steroid.[15] Side effects of steroids in patients with head injury are detrimental, their usefulness is questionable, and trials have demonstrated increased risk of death. Therefore, we are no more favor high-dose steroid use in TON. At best, level II evidence can only support use of steroids in TON patients who do not have traumatic brain injury (TBI) or any contraindication for steroids (e.g., peptic ulcer, diabetes mellitus, glaucoma, etc.). Maximum permissible dose of 1 g of methylprednisolone or equivalent should be used.[8]

We recommend surgical decompression to a very select group of patients, where there is definitive evidence to impingement of ON due to a bony fragment or a foreign body. Patients with intraneural and ON sheath hematoma have also been described in literature, who may benefit with surgery.[8] Apart from these conditions, usefulness of surgery is questionable, as risks far outweigh the benefits. Our observations, over the years, have been corroborated by International Optic Nerve Trauma Study (IONTS). This nonrandomized multicenter trial of 133 patients, treated by conservative/methylprednisolone/surgical basis, has shown that neither of the option is more beneficial when compared with conservative follow-up. Moreover, 57% patients, who were not treated, showed three or more lines of improvement in visual acuity.[16]

There is no level I evidence to support the usefulness of ON fenestration and opening of annulus of Zinn. Multiple surgical techniques described in literature for surgical decompression are intracranial, endonasal, sublabial, or transethmoidal approaches. These can be performed using microscope or endoscope. Timing of surgery is also controversial. Surgical decompression, when deemed necessary, should be done within 1 week. Endoscopic approach has lesser morbidity, in terms of absence of a visible scar. Optic canal can be accessed above the optic carotid recess by this transnasal-transsphenoidal approach.[17–19] Prognosis even after this approach remains guarded. A study of 96 patients, in which endoscopic ON decompression was performed, reported an improvement in only 40% patients, who could read one more line on Snellen's chart. The most important factor was better preoperative visual acuity.[20] Similar findings along with timing of surgical intervention have been cited as important prognostic factors by other study as well.[21] Thakar et al have also shown that supplemental incision over ON sheath may be better than osseous decompression alone.[2]

Most important prognostic factor is presence of better visual acuity after injury. Kumaran et. al[8] have reported that "patients with TON due to indirect injury, having lower grade RAPD and showing signs of visual recovery within 48 hours have a better prognosis."

The reason for poor prognosis, even after use of all available treatment modalities, is inability of central nervous system neuron to regenerate. Moreover, compensatory circuit formation for visual pathway fibers is also questionable.[8] Thus new research is focusing on drugs that can help in improving neuroprotection and neuroregeneration. There is no level I or II evidence to support use of these newer treatment options. However, usefulness of eythropoeitin,[22] crystalline,[23] glutamate inhibitors (lomerizine and minocycline),[24] brain-derived neurotrophic factor,[25] tacrolimus,[26] and tissue necrosis factor-α and nitric oxide synthase inhibitors[27] is being investigated. Therapeutic hypothermia in rats has been shown to be protective,[28] but its usefulness in humans in view of existing brain trauma foundation guidelines for head injury in itself is questionable. Ocular transplantation in cold-blooded vertebrates has led to some recovery, but in mammals it invariably fails due to inability of ganglion cell axons to regenerate.[8]

Based on current literature review and Mahapatra's criteria,[11,29] our recommendations are summarized as follows:

- All TON patients, who are not having severe TBI or contraindication for steroid use, are given a short-duration course of steroid.
- VEP and clinical examination are repeated every 2 to 3 days for the first 3 weeks.
- Patients showing good recovery are treated conservatively.
- Surgery is indicated in patients in whom visual improvement is marginal and then remains static and cause of compression is discernable. It is preferable to perform surgery within 1 to 2 weeks.
- Emergency surgery is rarely indicated in patients, in whom visual deterioration is delayed in onset and occurs rapidly, even after steroid use.
- Use of newer drugs is not supported by any level I or II evidence.

Despite all treatment options, prognosis in TON remains guarded. However, protocol-based management and timely intervention in indicated cases may help in salvaging vision of many patients.

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