Traumatic Optic Neuropathy [TON] - A Review

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Traumatic Optic Neuropathy [TON] is a form of optic neuropathy typically caused by indirect optic nerve injury resulting in dramatic impairment of visual function accompanied by an ipsilateral relative afferent pupillary defect (Marcus-Gunn pupil).

Incidence

Traumatic optic neuropathy in the United States occurs in 0.5-5 % of patients presenting with closed head trauma and in 2.5 % of patients presenting with midfacial fracture. International rates of traumatic optic neuropathy vary from country to country. Rates depend on the occurrence of causative events, such as nonfatal motor vehicle accidents and assault.

It is generally presumed that penetrating injuries of the orbit are associated with direct optic nerve injury. The incidence of TON after craniofacial trauma has been reported to be 0.5-1.5 % in older series. However, recent surveys report a higher figure; 2-5 %. This increased incidence may represent the diagnosis of subtle forms of TON closer to the time of injury. These types of cases appear to represent a smaller percentage of cases in the earlier studies on TON and may have been missed at that time. Males represent the majority afflicted with TON, comprising 60-95 % of cases. In one series focused on children, 40 % of the cases were female.

Although the degree of visual loss after indirect traumatic optic neuropathy may be quite variable, approximately 50 % of patients are left with “light perception” vision, making traumatic optic neuropathy a significant cause of permanent visual loss.

There may be associated fractures in the form of anterior orbital fracture, posterior orbital fracture, orbital blow-in or blow-out fracture.

Etiology

Traumatic optic neuropathy is associated with high momentum deceleration injuries and midfacial trauma. Loss of consciousness is associated with TON in 40-72 % of cases. Motor vehicle and bicycle accidents are the most frequent causes, accounting for 17-63 % of cases depending on the series. Other causes include impact over the frontal region by falling debris, assault, stab wounds, gun shot wounds, seemingly trivial injuries, and endoscopic sinus surgery.

Relevant Anatomy

The human orbit is pyramidal, with its base oriented anteriorly and its apex oriented posteriorly. The orbital walls converge posteriorly near the superior orbital fissure and optic foramen. The optic canal is separated from the superior orbital fissure by the optic strut of the sphenoid. The sphenoid body and lesser wing comprise the bony walls of the canal. In the adult human, the optic canal is approximately 6.5 mm in diameter and 8-10 mm in length. Although the canal is elliptical in true cross section, its walls can be described by redefining it as a base-up triangle. The canal’s roof separates the optic nerve from the sub frontal space of the anterior cranial fossa.
The optic strut bridges the sphenoid lateral wing and body and comprises the optic canal’s lateral wall. This wall separates the canal from the superior orbital fissure and its contained neurovascular structures. The canal’s lateral and medial walls separate it from the sphenoid sinus and, in approximately 12% of patients, the Onodi cells of the posterior ethmoidal sinus. Structures that pass through the optic canal include the optic nerve axons, their supportive glia, the ophthalmic artery, and branches of the carotid sympathetic plexus of the autonomic nervous system.

The optic nerve is 3-4 mm in diameter and measures 35-50 mm from the retina to the optic chiasm. The nerve is composed of intraocular (~1 mm), intraorbital (20-30 mm), intracanalicular (5-11 mm), and intracranial (3-16 mm) segments. The axons comprising the nerve have their origin in the nerve fiber layer of the retina and extend beyond the chiasm and optic tracts before synapsing within the lateral geniculate body. The topographic organization of the axons, as arranged by the retina, is preserved within the optic nerve. Except for its intraocular segment, the axons of the optic nerve are myelinated. Because the optic nerve is a white matter tract of the CNS, oligodendrocytes comprise two thirds of glia and are responsible for the production of myelin. Astrocytes are also present within the glial septa and provide nutritional support to the axons.

Throughout its intraorbital and intracanalicular course, the optic nerve remains surrounded by pia, arachnoid, and dura mater (optic nerve sheath). Within the optic canal, the dura remains fused to the sphenoid periosteum and, thus, is the only segment of the nerve or its sheath that is tightly fixed in space. At the posterior foramen of the optic canal, the optic nerve sheath reflects away from the nerve to fuse with the dura lining the calvaria. Therefore, the intracranial optic nerve lies within the subarachnoidal space.

Pial branches of the internal carotid, anterior cerebral, and anterior communicating arteries perfuse the intracranial optic nerve. Small pial branches from the ophthalmic artery supply the intracanalicular optic nerve. The intracranial optic nerve is supplied by perforating branches derived from the ophthalmic artery. The arterial circle of Zinn-Haller supplies the intraocular optic nerve with contributions from the posterior ciliary arteries, the pial arterial network, and the peripapillary choroidal vasculature.

**Classification**

Optic nerve injuries can be divided into direct or indirect injuries based on type of injury.

**Indirect Optic nerve injury**

Closed head trauma leads to indirect optic nerve injury which can be classified anatomically as

1. **Anterior**: the central retinal artery enters and the central retinal vein exits the optic nerve 8-12 mm posterior to the insertion of the nerve into the globe. Injuries anterior to this site are termed anterior.
2. **Posterior**: the injury is posterior to site of entry of the central retinal artery and exit of central retinal vein.

Anterior injuries disturb the retinal circulation while posterior injuries are associated with normal retinal circulation. Typically in the latter, no immediate change is seen on fundus examination. The disc appears normal for 3-5 wks after which it becomes pale as descending optic atrophy sets in.

**Direct optic nerve injury**

Direct optic nerve injuries result from objects that penetrate the orbit and impinge on the optic nerve causing optic neuropathy by partial or complete transection of the optic nerve sheath. Hemorrhages within and around the nerve may also occur.

Gunshot wounds, sharpnel, stab wounds etc are commonly reported etiological agents.

Unlike indirect optic nerve injuries, direct injuries lead to immediate changes in the fundus which can simulate central retinal artery occlusion, central retinal vein occlusion or anterior ischemic optic neuropathy. These can be detected on ophthalmoscopic examination.

**Pathogenesis**

Direct and indirect injuries both cause mechanical and ischemic damage to the optic nerve. Sometimes the
ocular injuries may be so subtle that there may be no external evidence. Generally, direct injuries have a prognosis that is worse than indirect TON. Two mechanisms, primary and secondary, operate resulting in such damage to the optic nerve.

**Primary: Shearing Injury**

Traumatic optic neuropathy, in its most common form, is an indirect event that occurs during or shortly after blunt trauma to the superior orbital rim, lateral orbital rim, frontal area, or cranium. The most widely held belief maintains that compression forces from the trauma are transmitted via the orbital bones to the orbital apex and optic canal. Laser interferometry studies demonstrate the same. Elastic deformation of the sphenoid then allows transfer of the shearing force of deceleration to the intracanalicular segment of the optic nerve. Contusion of the intracanalicular optic nerve axons and pial microvasculature produces localized optic nerve ischemia and edema. Edema of ischemic axons results in further neural compression within the fixed-diameter bony optic canal. A positive feedback loop is precipitated which triggers development of an intracanalicular compartment syndrome.

Focal axonal abnormality is induced, characterized by impaired axonal transport. The nerve is functionally separated into a proximal and distal fragment, within 6-24 hours after injury. The distal portion undergoes Wallerian degeneration while the proximal portion i.e. cell body undergoes apoptosis.

Tearing injuries of the microvasculature accompany the shearing injury to the axons leading to hemorrhage in the optic nerve and its sheaths.

Thus, primary mechanisms cause permanent damage to optic nerve axons at the moment of impact.

**Secondary**

Owing to disturbances of cellular homeostasis adjacent to areas of irreversible optic nerve damage, diverse and interrelated mechanisms operate which lead to loss of axons which had survived the original insult. These mechanisms are:

1. Ischemia and reperfusion injury – partial ischemia develops due to cessation of blood flow. However reperfusion of these transiently ischemic regions leads to peroxidation of cell membrane lipids leading to generation of oxygen free radicals which cause tissue damage.

2. Bradykinin: This is activated following trauma, and it leads to release of arachidonic acid from neurons. The prostaglandins derived from arachidonic acid metabolism, free radicals and lipid peroxides lead to edema in the optic canal, which further aggravates the ischemia.

3. Calcium ions: Following optic nerve ischemia, calcium ions enter the intracellular compartment through voltage and receptor gated channels. Increased intracellular concentration of calcium ions acts as a metabolic toxin and leads to cell death.

4. Cell mediated mechanisms: Polymorphonuclear [PMN] cells predominate in the first two days following trauma, which are then replaced by macrophages in 5-7 days. While PMNs lead to immediate damage, macrophages cause delayed tissue damage, demyelination and gliosis.

**Clinical Assessment**

The diagnosis of traumatic optic neuropathy is clinical. Following midfacial and cranial trauma, a high index of suspicion for a traumatic optic neuropathy is to be entertained. Patients with traumatic optic neuropathy typically experience sudden, severe, unilateral vision loss following blunt injury to the head or face. The condition may manifest immediately or within hours or days following the trauma. Occasionally, the vision loss may be insidious, and in some cases the patient may be unaware of any visual deficit until it is detected by routine examination.

Unfortunately there are a number of limiting factors to the assessment of patients with suspected optic nerve damage, such as presence of injuries to other major organ systems, patient’s level of consciousness, ability and willingness to cooperate with the examination. Attempt should be made to obtain as much information as is possible.

Most commonly, patients are males and in their teens or twenties. The history is varied, and often includes a
blow to the head severe enough to induce loss of consciousness or high-speed penetration of the globe by foreign material. Or the trauma may seem trivial, and the patient neurologically intact. In some cases, there is obvious evidence of injury to the orbit such as periorbital or ocular hemorrhage, ecchymosis or lacerations, in other cases there may be no evidence at all.

**History**

A comprehensive history should be obtained from the patient if stable, or from relatives, friends or eye witnesses to the injury. It is also essential to determine if the patient had any visual deficits before the trauma due to concomitant ocular disease. A medical, drug and drug allergy history should also be obtained if possible.

**Examination**

A comprehensive ophthalmic examination should be performed on all patients in whom traumatic optic neuropathy is suspected and should include the following assessments:

1. **Visual Acuity**: should be determined ideally using a Snellen’s chart or hand held near vision cards. The incidence of no light perception vision following TON varies significantly with most studies of 15 or more cases reporting an incidence that ranges from 22 % to 78 %\(^1\). About 20 % may have acuity better than 20/200. However a high index of suspicion is to be maintained to avoid missing more subtle cases of visual loss. It should be kept in mind that in less than 10 % of cases a delayed visual loss develops due to secondary optic nerve injury. Hence visual acuity should be assessed again after 24 hrs \(^5\).

2. **Relative afferent pupillary defect [RAPD]**: RAPD is elicited with the swinging flashlight test. Light that shines into a normal eye stimulates the pupil of that eye to constrict and also stimulates the pupil of the other eye to constrict consensually. There is less pupillomotor stimulation reaching the brainstem when the light shines into the eye with optic nerve injury compared to the uninjured side, so the pupillary response is diminished. This relative afferent pupillary deficit is the basis for the swinging flashlight test. \(^1, 2, 3, 4\) This is the most important clinical sign in an unresponsive patient. Test can be very useful for detecting unilateral optic nerve injury in an unresponsive patient and in the absence of fundus findings. Deficits greater than 2.1 log units when measured with neutral density filters are predictive of poor visual prognosis \(^1\). A relative afferent pupillary defect may not be present if TON is present bilaterally. However its presence doesn’t necessarily imply little or no vision in that eye. Only when the direct reflex is absent, but consensual reflex is retained, it can be deduced that there is no light perception in that eye \(^2\).

3. **Colour vision**: The patient is asked to look at a red object, one eye at a time. The object may be perceived as black, brown, or a faded red by the affected eye \(^2, 3\).

4. **Visual Fields**: Though there is no pathognomonic field defect that is diagnostic of optic nerve trauma \(^1\), fields should be assessed in an awake and cooperative patient as it provides rough information about possible location of optic nerve damage. Visual field loss following partial avulsion of the optic nerve from the globe tends to correspond with the lesion. Altitudinal defects with macular and upper field sparing, nerve fiber bundles defects, generalized constriction and depression, as well as central and paracentral scotomas are also reported \(^1\). In the absence of formal visual fields, bedside confrontational visual fields are useful in patient assessment. The patient is asked to detect hand movements or light in various regions of the visual field.

5. **Ophthalmoscopy**: Ophthalmoscopy is performed with the aid of a short-acting mydriatic agent on all stable patients. The retinal and choroidal circulation, optic nerve head morphology are evaluated. The presence of ring-shaped hemorrhage adjacent to the optic nerve head indicates a partial or complete avulsion of the optic nerve head. Anterior optic neuropathies produce disturbances in circulation leading to arterial and venous obstruction and disc swelling. Hemorrhages in the optic nerve sheath posterior to origin of central retinal vessels may spare the circulation but produce disc swelling. Bilateral disc swelling
suggests papilledema. Optic atrophy in the setting of acute head trauma with evidence of optic neuropathy indicates that some disturbance of the optic nerve was present before the trauma, and was not caused by it. Finally, damage to distal optic nerve in the orbit, optic canal, or intracranial cavity does not cause any change in appearance for about 3-5 weeks.

6. Ocular adnexa: Examination may reveal orbital rim and wall fractures, orbital edema, proptosis or enophthalmos, or extra ocular muscle dysfunction. Signs of penetrating injuries, such as protruding foreign bodies, extruding orbital contents, or conjunctival laceration, may range from obvious to subtle.

7. Intraocular pressure: Tonometry should be performed in intact globes. Increased intraocular pressure may accompany an orbital hematoma, diffuse orbital hemorrhage, orbital emphysema, or soft tissue edema.

**Visual Evoked Potential (VEP)**

Due to difficulty in neuro-ophthalmological testing on visual pathway functioning in severely injured patients or even during craniomaxillofacial reconstruction, VEP and electroretinogram (ERG) are believed to be reliable electrophysiological methods to collect distinct information whether the visual pathway function is intact, pathological but still present or absent. VEP is also a diagnostic consideration in patients who have suspected bilateral optic nerve injury. Logistically, a neurophysiological evaluation may be complicated by the inability to transport the patient to the neurophysiology laboratory or to perform a bedside VEP. Visual recovery is unlikely when VEP results are not recordable. In unilateral cases of traumatic optic neuropathy, flash VEP amplitude ratio (affected side/normal side) greater than 0.5 appears predictive of a favourable, long-term visual outcome.

The electrophysiological evaluation along with the multiplanar CT is important for the immediate identification of optic nerve trauma. The results of this evaluation will provide diagnostic information on whether surgical intervention and/or conservative therapy are required to prevent secondary optic nerve damage.

**Imaging**

In polytraumatized patients with loss of consciousness, CT scan with clinical exploration is the most important method for the assessment of traumatic optic neuropathy in the acute emergency setting. It may reveal specific pathology compromising the optic nerve, including optic nerve sheath hematoma, fractures involving the greater or lesser wing of the sphenoid, subperiosteal hematoma, hemorrhage affecting the orbital apex, ethmoid or sphenoid sinus, and pneumoencephalus. Fractures through the optic canal can be best depicted with thin-section CT scanning (e.g., 1.5-mm cuts with 1-mm intervals).

While CT scanning is clearly superior to magnetic resonance imaging (MRI) in delineating fractures of bone, MRI is superior to CT scanning for soft tissue. Often both CT and MRI are required to evaluate a given clinical situation. Magnetic resonance imaging should be deferred until a metallic orbital or intraocular foreign body has been ruled out by CT scan or conventional x-ray. Finally, CT scanning is critical for surgical planning if optic canal decompression is contemplated.

Although OCT has for the most part been used to evaluate RNFL thickness, recent software improvements have made it possible to measure macular thickness as well. The efficacy of macular thickness measurements in documenting progressive neural damage is being evaluated in a longitudinal study using OCT. No study has yet evaluated the role of macular thickness measurements in conditions associated with acute ganglion cell loss, such as optic nerve trauma. Previous studies have documented peripapillary RNFL thickness reduction after indirect traumatic optic neuropathy.

**Management of TON: The Controversies**

The management of a patient with TON is essentially by a multi-disciplinary approach involving the ophthalmologist, physician, neuro-surgeon, and an otorhinolaryngologist.

Several controversies exist concerning the management of TON. The optimum management protocol is yet to be elucidated as there is paucity of prospective large-scale clinical trials. Most widely accepted contemporary treatments for traumatic optic neuropathy include observation, steroids, and surgical decompression.
Since 1990 there have been at least 16 studies of TON with at least 10 patients per series for a total of 715 patients. It is difficult to compare one study with another in any meaningful way because of marked variation in how patients were collected and examined, time lag between injury and presentation, follow-up, dose of corticosteroid employed, concomitant optic canal decompression, and how patient details have been summarized and presented. The timing and type of decompression procedure and selected use and optimal dosing of perioperative corticosteroids have also been widely reported but have not been validated by controlled outcome trials.

The International Optic Nerve Trauma Study was organized to help clarify the value of treatment of TON. Initially, a pilot study was organized to assess recruitment feasibility for a randomized, double-blind study.

76 investigators from 16 countries were involved in the study. They were asked to complete and submit standardized data forms to the data coordination center, on all patients who were examined during the study period. There was neither a predetermined sample size, nor protocol for examination, steroid treatment, surgical management and follow-up schedule. The form was to contain history of injury, examination findings, results of CT scan if performed, description of the management [which was to be based on investigator’s customary practice], and surgical complications if any. Six months from the date of injury, a second data form was to be completed providing details of all follow-up vision examinations.

Patients with indirect optic neuropathy only, who had a vision assessment within 3 days of injury, were to be included in the study. Among the 206 patients for whom data forms were submitted, only 133 patients qualified for inclusion in the study. Since recruitment was insufficient, the study was converted to a comparative, nonrandomized interventional study. Patients received steroid treatment alone, surgery with or without steroids, or no treatment. There was no uniformity to the administered corticosteroid treatments. The study failed to find benefit for either corticosteroid therapy or optic canal decompression. The study has limitations, but it represents the largest, most unbiased study of TON to date.

The Study was organized with a strong bias that treatment for TON is beneficial. All but nine out of 133 patients enrolled in the International Optic Nerve Trauma Study received surgery or corticosteroids within 7 days of treatment. The authors stated that their results and the literature on TON provide “sufficient evidence to conclude that neither corticosteroids nor optic canal surgery should be considered the standard of care for patients with traumatic optic neuropathy.” They further suggest that it is appropriate to make individualized treatment decisions for a particular patient.

1. Medical

The mainstay of medical management of TON is the use of mega dose steroids, the use of which was extrapolated from the National Acute Spinal Cord Injury Study 2 [NASCIS II], a multicenter clinical trial that evaluated patients with acute spinal cord injury. In this study, patients were treated with placebo, methylprednisolone [MP], or naloxone. Pharmacologically, high-dose or mega-dose methylprednisolone therapy is associated with stabilization of the microvascular circulation and calcium homeostasis. The study showed that methylprednisolone (30 mg/kg loading dose, followed by 5.4 mg/kg/h for 24 h) started within 8 hours of injury was associated with a significant improvement in both motor and sensory function compared to patients treated with a placebo. Whether methylprednisolone therapy is similarly effective in the treatment of traumatic optic neuropathy is unproven. Moreover, analysis of both the NASCIS II and the more recent NASCIS III has raised questions regarding the statistical assumptions made by these studies. The benefit seen in NASCIS II may be a statistical artifact.

2. Surgical

Surgical decompression of the optic canal and the intracanalicular optic nerve sheath has been advocated for the management of indirect traumatic optic neuropathy. However there is no consensus on the role as well as the optimum timing of the surgical intervention. As it is thought that increased intracanalicular pressure causes vascular compromise with ischemia and interruption of neuro-feedback mechanisms leading to blindness, optic nerve decompression theoretically relieves annular
strangulation and reestablishes nerve function. Based on this theory, approaches to the optic nerve have been devised since traumatic optic neuropathy was first diagnosed. These procedures are complimentary to steroids, which reduce inflammation and edema and are widely used to treat traumatic visual loss.\textsuperscript{1, 2, 3, 4, 5}

Criteria for adequate surgical decompression:\textsuperscript{2, 3, 4}

1. Removal of at least 50\% of the circumference of the osseous canal.
2. Removal of bone along the entire length of the canal.
3. Total longitudinal incision of the dural sheath including the annulus of Zinn.

Various surgical approaches for decompression of the optic canal include trans-frontal craniotomy, extra-nasal trans-ethmoidal, trans-nasal trans-ethmoidal, lateral facial, sublabial and endoscopic approaches.\textsuperscript{1} The following is a brief description of the most commonly used approaches:

**Extra-nasal trans-ethmoidal**

This extra-cranial approach is the most popular approach and avoids the necessity of lifting the frontal lobe of the brain. However, since the optic nerve and the carotid artery are closely associated in the sphenoid sinus, damage to the carotid artery can occur during surgery.\textsuperscript{1}

**Endoscopic**\textsuperscript{9, 10}

The endoscopic approach is based on the fact that the optic canals almost always protrude into the sphenoid sinus wall. The procedure is performed using 0° and 30° nasal telescopes with the patients under general anesthesia. After ethmoidectomy and sphenoidotomy, the bulge caused by the internal carotid artery and optic nerve is identified in the lateral wall of the sphenoid sinus. The medial wall of the optic canal is thinned out with a micro-drill and removed with a microcurette. The annulus of Zinn and the optic nerve sheath are not incised.

The endoscopic technique should not be used in rare cases of conchal pneumatization. Optic nerve decompression with minimal morbidity and marked recovery of vision following the procedure has been claimed.

### Tran nasal trans-sphenoidal\textsuperscript{11}

All cases are operated under general anesthesia with hypotension and with 15° head end elevation. The nasal cavity is decongested using xylocaine with adrenaline in a concentration of 1:1, 00,000. The middle turbinate is medialized and bulla opened, ground lamella is then entered, posterior ethmoidal and sphenoid sinus are entered sequentially. The sinus ostium is widened in the inferomedial direction and lateral wall bone is removed in the region of the optic canal in whole of the length of canal. The nerve is decompressed in the whole segment of the canal and then medicated pack is kept in nasal cavity.

### TON in children

The vision loss caused by TON can be partial or complete, temporary or permanent. The management protocol is better defined in the adult population but its safety and efficacy is not yet established in the pediatric population.\textsuperscript{11} Early intervention has been recommended based on the difference in anatomy of the optic canal in adults and children. In adults, the optic canal is approximately 6.5 mm in diameter and 8–10 mm in length with the optic nerve having a diameter of 3–4 mm with a volume of around 1 ml. Structures that pass through the optic canal include the optic nerve axons, their supportive glia, the ophthalmic artery, and branches of the carotid sympathetic plexus of the autonomic nervous system. Gupta et al opined that, in children, as the optic nerve canal is smaller, a lesser volume is available for the nerve to expand, thus early visual impairment could occur following trauma. The neuronal degeneration hence will occur if intervention not carried out early during the course of injury. Thus, they recommended early intervention in cases of traumatic optic neuropathy in children rather than waiting for spontaneous recovery and also opined that combined use of surgery and steroids might help around 80\% children to regain vision.

The surgical technique used by Gupta et al was trans-nasal trans-sphenoidal optic nerve decompression, which was claimed to be minimally invasive and provided better surgical visualization, reduced hospitalization and avoided the morbidity associated with intracranial approaches.\textsuperscript{11}
Surgical treatment of optic nerve sheath hematoma and orbital hemorrhage

An optic nerve sheath hematoma can be evacuated by a medial or lateral orbitotomy depending on the location of the hematoma 1, 4.

In cases of orbital hemorrhage, a lateral canthotomy and cantholysis is done to permit expansion of orbital contents. This is followed by orbital imaging to look for subperiosteal hemorrhage or any other causes of visual loss. In case there is no visual improvement, an orbital decompression is to be considered. Systemic steroids are not used in this setting.

Some untreated cases of TON have been observed to improve even when the initial vision is no light perception. This rate of spontaneous visual improvement means that patients with TON may show improvement irrespective of whether treatment is instituted or not. The challenge comes in demonstrating that a particular intervention causes a rate of visual improvement different from the spontaneous rate of visual improvement. Due to the study variations mentioned earlier, older studies that were more observational and less interventional are unsuitable as controls for the more recent interventional studies 1.

Thus, a practical approach to management of TON can be put forth as follows 1-11:

1. In absence of any contraindications, the patient should be treated with systemic steroids: Methylprednisolone 30mg/kg as loading dose, 5.4mg/kg/hr maintenance thereafter for 48 hrs.
2. Failure to improve dictates a rapid taper and discontinuation.
3. Patients who improve can be switched over to a gradual tapering dose.
4. If the patient relapses when corticosteroids are discontinued, surgical decompression is to be considered.
5. In general, patients with visual acuity of 20/40 or worse can be taken up for surgical decompression.
6. Unconscious patients should not undergo decompressive surgery unless it is incidental to another operative procedure.
7. Two pronged approach with steroids and early surgical intervention may be considered in children.

Follow-Up

Recovery of visual function following traumatic optic neuropathy can be objectively defined by using serial assessment of multiple visual function parameters (e.g., visual acuity, visual field, quantification of afferent pupillary defect, assessment of abnormal color vision). Daily follow-up evaluations must be performed during the acute phase following trauma, immediately after surgical therapy, and during the period of mega-dose corticosteroid therapy. Less frequent examinations (i.e., every 4-7 days) are warranted during the intermediate period following trauma, surgery, or discontinuation of steroid therapy. Long-term observation is appropriate at a point 3 months or more from the date of injury to document the final level of visual function.

Prognosis

On the basis of several studies, the following four variables were considered to be poor prognostic factors for recovery of visual function 1, 4, 10, 12:

1. Presence of blood within the posterior ethmoidal cells
2. Age over 40 years
3. Loss of consciousness associated with traumatic optic neuropathy, and

Apart from these, posterior orbital fractures were found to be associated with a worse visual outcome than anterior fractures 6.

Recovery documented at the first follow up visit after treatment was significantly associated with recovery at the last follow up visit. These four negative prognostic signs in patients affected by traumatic optic neuropathy may be useful in predicting the visual outcome in patients developing visual loss after head trauma and in deciding on the need for surgical treatment. However recovery of vision can occur with or without treatment.
Conclusion

Traumatic Optic Neuropathy is a rare though important cause of visual morbidity in patients with closed head injuries and maxillofacial trauma. A high index of clinical suspicion, thorough clinical examination and radiological investigations performed at the earliest, aid in arriving at the diagnosis. Management is by a multidisciplinary approach, the options being observation, mega-dose corticosteroids [Methylprednisolone] or surgical decompression of the optic nerve. A consensus is yet to be established on the optimum management protocol for this condition, owing to the lack of large scale clinical trials. The prognosis is variable, though generally poor.

References