

Traumatic Optic Neuropathy

Ayyaz Hussain Awan

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authors affiliations
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Correspondence to:
Ayyaz Hussain Awan
Consultant Ophthalmologist
PNS Shifa
Karachi

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Purpose: To present a case of unilateral indirect traumatic optic neuropathy (TON) and stress the importance of early megadose steroid treatment.

Material and Methods: A 15 years old boy presented with loss of vision left eye following a road traffic accident 4 weeks back. On examination of the left eye his visual acuity was no perception of light and relative afferent papillary defect (RAPD) was positive. Anterior and posterior segment examination was normal. There were no retinal hemorrhages. The patient was diagnosed as a case of left indirect TON. All investigations were within normal limits except VEP, which showed an abnormal response in the left eye.

Results: On examination at 6 weeks post-trauma there was 15-20° exotropia left eye with poor recovery. Visual acuity in the left eye remained NPL and RAPD was positive. On fundoscopy the optic disk was yellow white in color with normal appearing retinal vessels.

Conclusion: TON is a neuro-ophthalmic emergency and early intervention is crucial. Intravenous megadose methylprednisolone (MP) within 8 hours of TON can save useful vision in some cases.

Traumatic Optic neuropathy (TON) is an uncommon but a devastating cause of permanent visual loss following contusive injuries to the head, particularly to the forehead. The impact transmits a shockwave to the optic canal, damaging the optic nerve. Typically the optic nerve head and fundus are initially normal, the only objective finding being a relative afferent pupillary defect (RAPD)¹.

CASE REPORT

A 15 years old boy presented with loss of vision in the left eye following a road traffic accident which occurred four weeks back. There was history of loss of consciousness for 1-1½ hours and bleeding from the wound on the left forehead. On examination there was a scar mark on the left forehead. On ocular examination visual acuity in the left eye was no perception of light (NPL) and relative afferent pupillary defect (RAPD) was positive. Ocular

movements were full with no visible tropia. On Slit lamp Examination anterior segment was normal. Fundus examination showed a healthy disc with a cup disc ratio (CD ratio) of 0.3 and a normal macula. Intraocular pressure (IOP) was 16mm of Hg in the left eye. Visual acuity in the right eye was 6/6 with a normal anterior and posterior segment examination. A provisional diagnosis of TON (LE) was made. On investigation, blood complete, urine routine, blood sugar (R) and X-Ray skull were within normal limits. Ocufer (Flubriprofen) eye drops were started 6 hourly in the left eye and prednisolone 60 mg was given in 4 divided doses orally for one week. Patient was asked to report back after having a computed tomography (CT) and magnetic resonance imaging (MRI) scan of the brain and orbit and visually evoked potential (VEP). He reported back after 2 weeks (6 weeks post-trauma). On examination visual acuity was NPL in the left eye with a 15° - 20° exotropia showing a poor recovery. Optic disc was pale yellow. CT and MRI

scan of brain and orbit were within normal limits. VEP examination on checkerboard pattern reversal stimulation and flash technique showed an abnormal result. There was prolonged P-100 latency on left side is suggestive of left optic pathway dysfunction. However there was presence of waves on P-100 flash technique showing at least relative integrity of optic pathway but having less specificity and sensitivity. Right optic pathway was intact.

DISCUSSION

TON is divided into direct and indirect injuries. Direct injuries have a worse prognosis and occur when an object penetrates the orbit and damages the optic nerve^{2,3}. Indirect TON is a closed injury produced by force imparted to the skull and transmitted into the optic nerve. Injury mechanisms are classified as primary and secondary. Primary mechanisms result in permanent axonal injury at the moment of impact. In contrast, secondary^{4,5} mechanisms cause damage to the optic nerve axons subsequent to the force of impact. Intracanalicular optic nerve is the most common site for TON followed by intracranial optic nerve and injuries that involve the chiasma⁶.

Injuries anterior to where the central retinal artery enters the nerve disturb the retinal circulation⁷ shown by presence of a cherry red spot or central retinal edema. Partial or complete optic nerve avulsions from the globe produce a partial, or respectively, a complete ring of hemorrhage at the optic nerve head⁸ followed by massive proliferation of connective tissue around the disc. Posttraumatic rupture of the posterior ciliary arteries is seen as disturbance of the retinal pigment epithelium. Various visual field defects are an inferior altitudinal defect with macular and upper field sparing, central and paracentral scotomas, nerve fiber bundles defects and generalized constriction and depression⁹.

Computed tomography (CT) scanning with axial and coronal views may reveal specific pathology compromising the optic nerve, including optic nerve sheath hematoma, presumed arachnoid cyst, fractures involving the greater or lesser wing of the sphenoid, subperiosteal hematoma, hemorrhage affecting the orbital apex, ethmoid or sphenoid sinus and pneumocephalus.^{10,11} MRI can detect presence and longevity of hemorrhage within the optic nerve sheath, swelling of the optic nerve in the tight optic canal, and thickening of the optic nerve sheath. The VEP is commonly used to study or detect various disorders of the afferent visual pathway.

The idea of using very high doses of corticosteroids (megadose) to treat TON was incorporated from research and clinical practice in treating spinal cord and brain injuries. The Second National Acute Spinal Cord Injury Study (NASCIS II) was a multicenter, randomized, double-blind, placebo-controlled study of patients with acute spinal cord injury designed to test the usefulness of megadose steroids in acute spinal cord injury. Patients were randomized to one of three treatment arms within 12 hours of injury: placebo, naloxone, and Methylprednisolone (MP). MP was administered as an initial dose of 30 mg/kg followed by a continuous infusion of 5.4 mg/kg/hour. Treatment with MP within 8 hours resulted in a statistically significant improvement in motor and sensory function compared to placebo-treated patients¹².

The rationale for high-dose steroid use is based on the ability of steroids to reduce trauma-induced edema, microvascular spasm, and nerve cell necrosis.

In case of TON when there is no contraindication to steroid use, a loading dose of MP 30 mg/kg intravenously, followed by 15 mg/kg administered 2 hours later, and then 15 mg/kg every 6 hours is recommended. If visual function improves, the steroid doses are continued for an additional 5 days, then tapered rapidly. When no improvement occurs within 48 to 72 hours, steroid administration is discontinued without a tapering dose¹³.

Optic canal decompression is advocated when a trial of high-dose steroids does not produce a favorable response¹⁴ or there is presumed impingement of the optic nerve by bone fragments¹⁵ and in cases of delayed visual loss. The surgery is performed by a variety of approaches, including transthemoidal, transantral, transnasal, supraorbital-cranial and uncommonly via a lateral facial approach¹⁶.

CONCLUSION

Megadose corticosteroids in cases with TON presenting within 8 hrs of injury and optic nerve decompression for the relief of optic nerve swelling in cases with impingement of the optic nerve by bone fragments especially in cases with delayed visual loss has been favored. Even though questions have been raised regarding the value of these treatments they are the only option and hope available for the patient and doctor both.

Author's affiliation

Ayyaz Hussain Awan
Classified eye Specialist
PNS Hafeez (Naval Hospital)
Sector E-8, Islamabad

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Surgery is more effective in lowering IOP but the immediate and late complications particularly cataract changes have to be considered and of particular importance is the postoperative bleb management.

Prof. M Lateef Chaudhry