Eye in Polytrauma

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Introduction:
Motor vehicle accidents, fall from height, assault, industrial accidents especially blast injuries are the most common causes of poly-trauma associated with ocular and orbital injuries. Many of these patients also present with head-injuries (closed or open), therefore in addition to the ocular and adnexal injuries, neuroophthalmic involvement due to head injury often cause loss of vision. Injury to the anterior and posterior visual pathways, the pupillomotor pathways, the cranial nerves and the supranuclear and internuclear gaze pathways can all occur in a patient of polytrauma.[1]

Demographics
Most polytrauma patients are males with 4:1 preponderance over females. The most susceptible age group a young adult between 20 to 40 years. The most common causes of polytrauma in Indian population are motor vehicle accidents (especially two wheeler riders without helmets), industrial accidents, followed by assault. The incidence of sports associated injuries is less in Indian population than that quoted in western literature.[2]

Evaluation of Patients
Polytrauma patients with injury to the visual system often have concomitant neurologic, orthopaedic and internal organ injuries some of which may be life threatening. As with all trauma patients, initial assessment and management must emphasize airway management, control of haemorrhage, hemodynamic stabilization and neurologic assessment. Airway management may require anterior positioning of mandible or placement of nasopharyngeal, oropharyngeal device. Brisk bleeding may accompany injury to the craniofacial region due to the rich vascular supply. Intracranial injuries often accompany craniofacial trauma, hence patient’s level of consciousness should be assessed early to establish a baseline. Once airway management, hemodynamic stabilization is achieved and level of consciousness is determined, a detailed history and physical examination are conducted.

A multidisciplinary approach with cooperation of neurosurgery, general and trauma surgery, otolaryngology and facial trauma surgeons is imperative for optimizing prompt evaluation and treatment of patients. The appropriate time for ophthalmologic consultation should be as early as possible once the patient’s vital signs stabilize. The presence of ocular injuries may influence the timing of repair of craniofacial injuries, and also because early treatment of trauma related visual loss within 6-8 hours of injury has been shown to be beneficial to the patient.

Obtaining history and conducting thorough ophthalmologic examination of the polytraumatised patient with altered sensorium often presents a challenge to the examiner. Visual acuity and colour vision testing may be difficult if the patient has decreased level of consciousness or is heavily sedated. However, the assessment of gross visual acuity even light perception may be prognostic in the light of neuroophthalmic findings. If a open globe injury is evident or suspicion is high, immediate surgical repair of the globe under general anaesthesia is indicated and should take precedence over other nonlife saving interventions. In the absence of open globe injuries, adnexal injuries like lid and forehead lacerations, fracture of bones of the forehead and orbital rim fractures should be carefully looked for. Assessment of pupillary reactions both to direct and consensual to rule out afferent pupillary defect suggesting a traumatic optic neuropathy is urgent and very important especially in a patient in whom vision cannot be recorded due to altered sensorium. Presence of traumatic mydriasis, miosis, changes in size and shape of pupils due to coexisting cerebral edema, injury to the midbrain, pons may confuse the examiner but every attempt must be made to rule out TON. Failure of the dilated pupil to constrict with 1% pilocarpine suggests a structural injury to the iris. Traumatic miosis that increases in dim light and is associated with narrowing of palpebral fissure suggests a Horner’s syndrome. The above findings with neck pain should alert the examiner to rule out carotid – cavernous fistula caused by trauma. Ocular movements should be assessed whenever possible. Presence of ptosis, vertical and horizontal diplopia, movement restriction and gaze palsy suggests a cranial nerve palsy and can help locate the site of injury. Unilateral exophthalmos with orbital bruit, raised intra ocular pressure and extra ocular muscle palsy are findings characteristic of traumatic carotid – cavernous fistula. Oculomotor nerve palsy occurring after trauma is usually associated with decreased level of consciousness and severe head injury. Absent corneal sensation suggests involvement of trigeminal nerve while lagophthalmos indicates facial nerve injury.

Fundus examination must be done in a patient with visual dysfunction following trauma. However if the patient has altered level of consciousness the ophthalmologist must...
consult the attending house surgeon before dilating the pupil for fundus examination. The time and type of drug used to dilate the pupils must be documented. It is advisable to use short acting mydriatic in these patients.

Traumatic optic neuropathy is the most important cause for visual loss (often irreversible) in poly trauma. [1]

**Traumatic optic Neuropathy**

Traumatic optic neuropathy (TON) is loss of visual function which is associated with trauma. The vision loss can manifest as sub normal visual acuity, visual field loss or colour vision dysfunction accompanied by ipsilateral Relative afferent pupillary defect (RAPD). TON is uncommon but important sequel of closed head injury. It occurs in 1.6% of head trauma cases and in 2.5% of patients with mid facial fractures.[2,3]

**TON is classically separated into two types.**

1. Direct optic nerve injuries: result from orbital or cerebral trauma that transgress the normal tissue planes and disrupt the anatomic and functional integrity of optic nerve like optic nerve avulsion and transection of optic nerve.
2. Indirect optic nerve injuries: is caused by forces transmitted at a distance from the optic nerve. The anatomy and function of optic nerve is compromised by the energy absorbed by the nerve at the moment of impact.
3. A subset of injuries can cause TON due to diffuse orbital haemorrhage, retro bulbar haemorrhage/hematoma, optic nerve sheath hematoma or orbital emphysema.

Trauma induced damage to the optic nerve can occur anywhere along its course.

1. Optic disc trauma
2. Anterior optic neuropathy: within 10mm posterior to the globe has opthalmoscopic features of CRAO, CRVO or AION.
3. Posterior optic neuropathy: from 10mm posterior to the globe, the intracanalicular part and intra cranial part. The fundus picture is normal for 3 – 6 weeks following which a temporal pallor of disc is seen.

The major focus of this discussion will be on indirect posterior optic nerve injuries. The intracanalicular part is the commonest site accounting for 81% followed by the intra cranial part (54%).

**Mechanism of Injuries:**

The most common form occurs during or shortly after a blunt trauma to the superior orbital rim, frontal area or cranium. The compression forces from the trauma are transmitted via the orbital bones to the orbital apex or the optic canal. The firm attachment of the dural sheath of the optic nerve in the optic canal makes it particularly susceptible to acceleration – deceleration injuries. In addition the vascular supply to this portion is subject to disruption from shear injury or from compression when the nerve swells up within the confines of the optic canal.

**Type of Injury:**

The injury to the optic nerve is a combination of both mechanical and ischemic damage. Injury may be – primary injuries due to haemorrhage into the nerve or its sheath, lacerations and contusions of the axons. Secondary injuries occur due to vascular obstruction or an ischemic damage initiated by shear injury with actual vascular disruption.

When there has been a mid facial or cranial trauma a high index of suspicion of optic nerve dysfunction must be kept in mind. Visual loss is typically immediate and severe with 24 – 86% of patients having no perception of light in a reported case series. There may be associated loss of consciousness (40 – 72% of cases).[2,3]

In some cases, there may be prominent orbital edema and haemorrhage where as in there may be little external evidence of injury.

**Clinical Evaluation:**

Finding of decreased visual acuity with RAPD in the absence of intra ocular pathology, suggests a posterior optic nerve injury. Determining the time of visual loss relative to the injury is a strong prognostic indication (immediate visual loss – poor prognosis, than a gradual visual loss). Red desaturation test is a sensitive test for optic nerve dysfunction.

The presence of RAPD must be quantified using neutral density filters (0.3 – 3 log units). This helps in evaluating the severity of visual loss and also in monitoring the recovery.

**Investigations:**

Visual fields: There are no pathognomonic visual field defects. It may be altitudinal, central scotomas or hemianopias (intra cranial)

VEP is especially useful in unresponsive patients and in cases of unilateral optic nerve injury, but may not be possible for logistic reasons

In unilateral TON, a flash VEP amplitude ratio (affected/normal side) greater than 0.5 appears to be predictive of favorable long term visual outcome.

Imaging of the orbits and brain must be done for detecting fractures of optic canal, bony fragment impinging on optic nerve, orbital and intra sheath hematomas. The fracture of sphenoid bone shows the severity of the trauma. [4]

The treatment of TON is controversial due to lack of prospective, randomized, controlled studies. But it has been proven that visual improvement following treatment was significantly better than the recovery with no treatment (86% compared to 20 – 40%).

The corner stone of treatment is early use of high dose I.V. Methyl Prednisone. The rationale for this is based on lab
studies and on the National Acute Spinal Card injury study II, which showed that early use of steroids within 8 hours can reduce edema and tissue damage.[5]

Several mechanisms have been proposed to explain the neuroprotective effect of intravenous methyl prednisolone.

• Inhibition of oxygen free radical induced lipid peroxidation. This is dose dependent.

• Other effects which are less well understood include – support of energy metabolism, prevention of post traumatic ischemia, reversal of intra cellular calcium accumulation etc.

There are some factors which may indicate poor visual outcome as shown by some studies. These include, presence of blood in the posterior ethmoidal air cells, patients older than 40 years, loss of consciousness, failure of recovery after 48 hours, Flash VEP amplitude ratio les then 0.5 and APD more than 2.1 log units when measured with neutral density filters.

These predictive factors of visual outcome important for counselling patients regarding treatment and also for decision making regarding surgical decompression.

A meta-analysis of TON.[2] showed that recovery is related to the severity of initial injury and devised a grading system based on Visual acuity, the locations and type of fracture

Grade I – VA > 20/200 and without a posterior orbital fracture

Grade II – VA – 20/200 – light perception

Grade III – No light Perception or with a non displaced posterior orbital fracture

Grade IV – No light Perception and a displaced posterior orbital fracture.

This grading is useful for comparing studies and treatment protocols.

**Treatment Protocol:**[5,6]

Establish the diagnosis of TON based on reduction of visual acuity and presence of RAPD (quantitative RAPD using neutral density filters).

Rule out contraindications to high steroids such as pre existing infectious diseases, peptic ulcer disease, uncontrolled diabetes mellitus, pneumocephalus etc.

Institute high dose I.V. Methyl prednisolone (Optimal time to start is within 8 hours of injury)

Loading dose of 30mg/Kg body weight infused over 30 minutes.

Maintenance dose of 5.4mg/Kg / hour for 48 hours.

Monitor therapeutic response with serial visual acuity and RAPD measurements.

After 48 hours of I.V. Methyl Prednisolone. If visual acuity and RAPD improves, change to oral prednisolone and taper rapidly over 15 days.

If visual acuity and RAPD worsens during oral steroid therapy, reinstitute high dose I.V. M.P and consider surgical decompression.

Immediate surgical decompression of optic canal is considered in the following situations:

• Bony fragments impinging on the optic nerve

• Orbital and intra sheath hematomas

**Surgical Decompression:**[7, 8]

Procedure of choice is Transethmoidal – sphenoidal optic canal decompression. No correlation was found between outcome and timing of surgery, but preferably within one week. Surgery is not done in unconscious patients.

Decompression of optic canal consists of

• Removal of at least 50% of the circumference of osseous canal

• Removal of bone along the entire length

• Longitudinal incision of the dural sheath, including the annulus of Zinn.

As there are no prospective, randomized, controlled studies, the IONT study was proposed, (which initially started as a prospective study but later got converted into an observation study). The study concluded that there was insufficient evidence that either mega dose steroids or surgical decompression could be considered the standard of care[9].

**Newer drugs in the experimental stage:**[9]

The role of I.V. Mannitol should be considered, as brain edema causes loss of auto regulation of blood flow resulting in poor perfusion pressure to the optic nerve.

The goal in treatment TON must be early recognition and appropriate intervention.

Other possible neuro-ophthalmic findings in patients with poly trauma (specially head injury[11]

Bilateral pinpoint pupils – narcotic intoxication, pontine haemorrhage, parasympathomimetic drug ingestion

**Cranial nerve palsies**

• 3rd nerve injury – Ptosis with anisoria

• 3rd nerve with contralateral tremor – red nucleus injury

• 3rd nerve with contralateral hemiparesis – fasciculus injury

• 3rd nerve with contralateral ataxia – cerebellar injury

• 4th nerve injury – vertical diplopia with tilting of images

• 6th nerve injury – horizontal diplopia

• Multiple cranial nerve injury – cavernous sinus injury, Caroticocavernous fistula ,Cranial base fracture , Brain stem injury

• Horizontal gaze palsy – frontal lobe, pontine injury
References: