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Visual outcome in patients with traumatic optic neuropathy

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ABSTRACT

Purpose: To assess the visual outcome in patients with decreased vision due to traumatic optic neuropathy who were managed differently either by conservative method, iv corticosteroids, or decompression surgery
Materials and Methods: A retrospective study was conducted involving 25 consecutive patients (25 eyes) with unilateral blindness as a result of head injury with regard to their visual status, CT scan, and MRI scan.
Results: Patients were divided into 3 groups. In first group, 12 of 14 eyes treated with intravenous followed by oral corticosteroids had shown 2 line improvement of visual acuity, where as in the second group, 8 of 9 patients treated conservatively had shown 1 line of improvement and in third group of patients who had decompression surgery, 2 of 2 had shown 1 line of improvement. The vision of 3 patients remained the same when they presented 1 month after injury.
Conclusion: Patients treated with intravenous followed by oral corticosteroids have better visual outcome compared to those who underwent conservative management and decompression surgery. Patients who had vision better than counting fingers and initiation of treatment with in 8 hours of injury showed significant improvement.

1. Introduction

Traumatic loss of vision, along with deficits in visual field, colour perception and an afferent pupillary defect is called traumatic optic neuropathy (TON).[1] It occurs most commonly after blunt trauma to the eyeball, mostly a consequence of road traffic accidents or falls.

Injuries are traditionally classified as direct and indirect. A direct injury is where the damage is the result of contact between the optic nerve and an external object e.g., surgical knife, bullet, piece of glass. In contrast, an indirect injury occurs when the force of collision is transferred through the bones of the skull to the optic nerve e.g., a blow to the forehead. These are often associated with high velocity deceleration injuries.

Optic nerve injury following orbital hemorrhage, which does not fit into either category, results in an orbital compartment syndrome[2] with elevated orbital pressure compromising the circulation of the optic nerve. Although it is most common following an insult to the optic nerve such as retrobulbar injections, blepharoplasty, repair of orbital fractures. It has been documented to occur spontaneously in association with sickle-cell disease coagulopathies, thrombolytic therapy and childbirth.

Orbital emphysema occurs when air becomes trapped in the orbit due to a ball-valve mechanism typically following orbital fracture. Vomiting and nose blowing in the presence of an orbital fracture may force air into the orbit and result in optic nerve compromise.[2]

2. Materials and Methods

A retrospective study was conducted involving 25 consecutive patients (25 eyes) with traumatic optic neuropathy attending Ophthalmology department at a tertiary care center from October 2019 to March 2020.

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Table 1: Demographic and clnical characterstics were noted.

Characterstics	Total (n= 25)
Mean Age	34 Years
Sex	
Male	25
Female	00
Eye	
Right	12
Left	13
Injury type	
RTA	22
Fall	01
Assault	02
Baseline visual acuity	
NPL	00
PL	01
HM	03
6/60 to CFCF	08
6/24 to 6/36	11
6/6 to 6/18	02

Table 2: Comparision of visual acuities during follow up

Type of Management & Vision at the time of presentation	Vision	
	Post treatment	After 1 month
Corticosteroids		
-6/24	6/18	6/12
-6/36	6/18	6/18
-6/60	6/24	6/24
-2/60	3/60	4/60
-1/60	1/60	1/60
-HM	CFCF	1/60
-PL	PL	PL
Conservative management		
-6/12	/9	6/9
-6/18	6/12	6/12
-6/60	6/36	6/36
-3/60	4/60	4/60
-HM	HM	HM
Surgical Decompression		
-HM	CFCF	CFCF
-CFCF	1/60	1/60
Patients 8% with compressed optic		

Cases with pre-existing ocular conditions that might affect assessment of visual function were excluded. All cases had a complete ocular examination including best corrected visual acuity (Table 1) colour vision, IOP measurement, pupils assessed for relative afferent pupillary defect, ocular motility and fundus examination on admission, immediately post treatment and 1 month later and had CT scans (axial and coronal) of orbit and brain and MRI accordingly. Visual acuity was the main outcome measure of the study, which was measured by Snellen chart. A written informed consent was taken prior to starting of treatment.

Traumatic optic neuropathy is mainly a clinical diagnosis, based on history, reduced visual acuity and presence of RAPD. Diagnosis is supported by initial assessment of CT scan, MRI scan and VEP at the time of presentation. Out of all, 17 patients had more than one bony fracture of skull or orbit on CT scan, 2 patients had evidence of optic nerve compression on MRI., Pattern reversal VEP showed normal in 11 patients and abnormal in 14 patients. Fundus is normal in all patients at the time of presentations

All patients were divided into 3 groups, who had been managed differently. First group (14 patients, 56%) treated with intravenous methylprednisolone given as an initial dose of 30 mg/kg followed by a continuous infusion of 5.4 mg/kg/hr for 3 days followed by oral prednisolone 1mg/kg for 11days. The second group (9 patients, 36%) was treated conservatively due to several reasons. including severe head injury, uncontrolled diabetes mellitus and young age. Third group (2 patients, 8%) with compressed optic nerve was treated by optic nerve decompression.

3. Results

Twenty five patients (25 eyes) were included. All cases involved were males. Mean age was 33 years old. (Table 1) Motor vehicle accident was the major cause (88%). Both eyes were equally involved. Most of the eyes had poor vision on presentation (6/60-NPL, 68%) (Table 1) with associated periorbital haematoma (24 eyes) and subconjunctival haemorrhage (21 eyes).

Majority of patients (17 patients, 68%) presented with more than one bony fracture of skull or orbit and 8 patients (32 %) had no fractures and out off all 2 patients had evidence of optic nerve compression on MRI scan. Out of 3 groups, first group (14 patients, 56%) was treated with intravenous methyl prednisolone initial dose of 30 mg/kg followed by a continuous infusion of 5.4 mg/kg/hr, according to national acute spinal cord injury study-2(NASCIS-2),[3] second group was treated conservatively (9 patients, 36%). Third group (2 patients, 8%) with compressed optic nerve was treated by optic nerve decompression. 12 of 14 eyes (91.7%) treated with intravenous followed by oral corticosteroids had shown 2 line improvement of visual acuity in first group where as in second group 8 of 9 patients treated conservatively also had shown 1 line of improvement. Third group of patients 2 of 2 had shown 1 line of improvement. 3 patients vision was remained the same as they presented 1 month after injury, 1 month post treatment (Table 2). RAPD persisted in 3 patients, on fundus examination, optic atrophy is seen at the time of 1 month followup. VEP also remained abnormal in these 3 patients

The main mechanism by which corticosteroids are thought to block neuronal death in the setting of trauma is inhibition of free radicals, decrease intra-neuronal or extraneuronal oedema, reduce vasospasm limiting contusion.

4. Discussion

Optic nerve injury is a rare condition, nevertheless it is important as it could cause significant visual loss and even blindness. In India, it is estimated that over 500,000 people suffer from some form of head injury every year. Given a 2% incidence of optic nerve injury in association with head injury, i.e over 10,000 would develop optic nerve injury leading to traumatic optic neuropathy.[3] When the optic nerve enters the optic foramen its dural sheath becomes continuous with the lining of the orbit and the optic foramen, rendering it immobile. This portion of the nerve, is the most common site of optic nerve injury.[4]

Causes of traumatic optic neuropathy can be divided as RTA injuries, falls, intra ocular foreign bodies, assaults, trivial causes such as weightlifting as well as following endoscopic sinus surgery.[5] It may occur directly or indirectly after cranio orbital trauma. Causes of damage include optic nerve transection, avulsion, ischemia, orbital hemorrhage and edema. Direct optic nerve injuries arise from penetrating trauma, especially orbital fractures associated with mid-facial fractures; whereas indirect optic nerve injuries occur when the force of impact is imparted into the skull and transmitted to the optic nerve. Orbital hemorrhage compromises the circulation to the optic nerve, resulting in injury secondary to orbital compartment syndrome. Primary injury to the optic nerve fibers by transection or infarction at the time of injury results in permanent damage. However, neural dysfunction secondary to compression within the optic canal, as a result of edema and hemorrhage, other factors causing secondary neuronal damage are generation of free radicals and initition of apoptosis, may respond to medical or surgical intervention.

Ocular manifestations are, moderate to severely reduced visual acuity even NPL in some cases with RAPD. Visual fields may help in localizing the site of optic nerve damage. High-resolution computed tomography (CT) is the diagnostic procedure of choice as it can delineate bony fractures better than magnetic resonance imaging (MRI). VEP is done to assess the electrophysiological improvement at the time of presentation and followup. The primary optic nerve injuries are not treatable. The secondary effects of the primary injury — edema hemorrhage, free radical injury may be treatable, by mega doses of iv methyl prednisolone or immediate decompression by lateral canthotomy and cantholysis, or conservative management.

According to literature, there has been, no large prospective placebo controlled trials for evaluating the role of steroids for treatment of TON done till date. Steroids as a therapy for traumatic optic neuropathy has been accepted after the results of National Acute Spinal Cord Injury Study (NASCIS-2) which showed positive results when systemic corticosteroids were used in patients of acute spinal cord trauma.NASCIS-2 was a multicenter, randomized, double-blind, placebo-controlled study involving patients with acute spinal cord injury. When compared with the placebo, treatment with methylprednisolone within 8 hours of injury resulted in a significant improvement in motor and sensory function.[6]

The international optic nerve trauma study, in which visual outcomes were compared with patients following conservative management, high dose of systemic corticosteroids given within 7 days of the injury, and optic canal decompression with or without corticosteroids and performed within 7 days of the injury, had shown no significant benefit for either corticosteroids or optic canal decompression in patients of traumatic optic neuropathy.[7,8] Surgical decompression of optic nerve can be done in selected patients. The goal of optic nerve decompression is to provide surgical relief of pressure on the intracanalicular segment of the optic nerve.[9,10]

Some studies had shown good visual outcomes with steroids[11,12] and some studies revealed the high rate of spontaneous recovery.[13]

5. Conclusion

Most of the traumatic optic neuropathy patients were presented with periorbital hematoma, subconjunctival hemorrhage and orbital wall fractures. Patients treated with intravenous followed by oral corticosteroids have better visual outcome compared to those who underwent conservative management and decompression surgery. Patients who had vision better than counting fingers and initiation of treatment within 8 hours of injury showed significant improvement.

6. Source of Funding

None.

7. Conflict of Interest

The authors declare that there is no conflict of interest.

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