Case Report

Late steroid intervention in traumatic optic neuropathy

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Abstract

Traumatic optic neuropathy after craniofacial injury was first described by Hippocrates. The optic nerve is vulnerable to indirect and direct trauma causing functional impairment of vision. Optic nerve injuries occur in the setting of head injury which is often a consequence of road traffic accidents or falls. The diagnosis of optic nerve injury may be delayed by the presence of other life-threatening injuries .We report a case of 60 yrs old male patient who met with a road side accident and reported to us with sudden painless loss of vision left eye after 15 days. On MRI brain and optic nerve he had small Extradural haemorrhage (EDH) along left anterior frontal lobe and there was enlargement of left optic nerve sheath. Currently, there is no validated approach to the management of traumatic optic neuropathy. Thus, with numerous conflicting reports on the management of traumatic optic neuropathy, there is little world consensus on the optimal management of this condition. Keeping this in view we devised high dose steroids to the patient to which he regained his vision in left eye 6/60. The main aim of this article is to review the treatment modalities in a case traumatic optic neuropathy.

Keywords: Extradural haemorrhage, high dose steroids, Traumatic optic neuropathy.

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Received Date: 16/02/2015 Revised Date: 23/02/2015 Accepted Date: 27/02/2015

Access this article online

Quick Response Code:

Website: www.statperson.com

DOI: 01 March 2015

INTRODUCTION

The commonest cause of optic nerve trauma is road-traffic accidents, when the patient has poly-trauma with head injury and the visual loss is noticed only after the general condition of the patient improves. Several varieties of direct optic nerve injury may be recognised ophthalmoscopically or with imaging techniques: optic nerve avulsion, transection, optic nerve sheath haemorrhage, orbital haemorrhage, and orbital emphysema. Indirect optic nerve injury is more common. The force of impact in a head injury may be transmitted to the optic nerve. This complication of head injury was known to Hippocrates who noted that blows to the eyebrow may cause blindness. The frequency of optic nerve injury occurring in closed head injury varies from 0.5 to 5%[2].

CASE REPORT

We report a case of 60 yrs old male patient who presented to Eye Out Patient Department after a road side accident with chief complaint of sudden painless loss of vision left eye (OS) noticed on 15th day post trauma after his periorbital swelling subsided. On vision assessment patient had 6/6 vision in right eye (OD) and denied perception of light in left eye. The patient had relative afferent pathway defect in left eye. Fundus examination was within normal limits. On MRI brain and optic nerve he had small EDH along left anterior frontal lobe (Figure 1) and there was enlargement of left optic nerve sheath (Figure 2). The intraorbital part measured 5 mm OD and

How to site this article: Vinod Sharma, Kalpana Sharma, Himanshu Goyal, Suresh Kumar, Kulbhushan Prakash Chaudhary. Late steroid intervention in traumatic optic neuropathy. *International Journal of Recent Trends in Science and Technology* March 2015; 14(2): 256-259 http://www.statperson.com (accessed 02 March 2015).

7.5 mm OS, the intra-canalicular part measured 2.9 mm OD and 5.5 mm OS suggesting fluid along left optic nerve and left orbital apex enhancing tissue. There was also haemorrhagic contusion in the cerebral hemisphere in the left fronto-parietal and temporal region (Figure 3). As there is no specific indication that the dose or timing of corticosteroid treatment or the timing of surgery was associated with an increased probability of visual improvement so we started the patient on intravenous (i.v.) 1 gm/day methylprednisolone for three days followed by oral steroids prednisolone 1mg/kg body weight for 10 days followed by gradual tapering to 5 mg/day for 5days. After 15 days of initiation of the treatment, the patient's vision recovered to 3/60 which subsequently improved to 6/60 on 30th day.

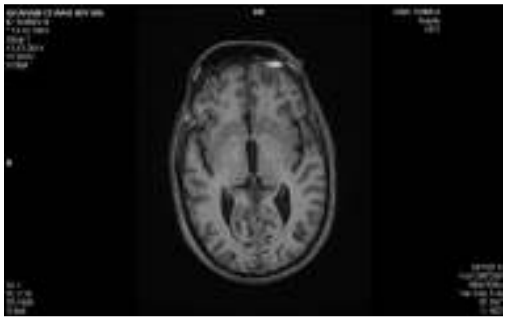

Figure 1: Axial T1 weighted image showing small EDH along left anterior frontal lobe

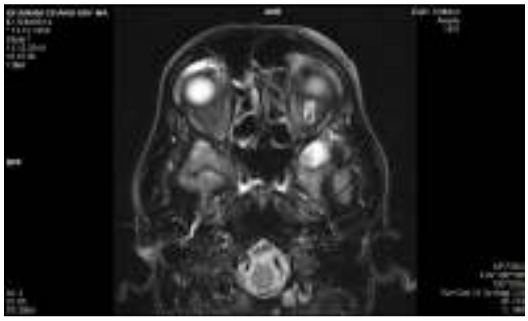

Figure 2: Axial T2 weighted image showing enlargement of left optic nerve sheath

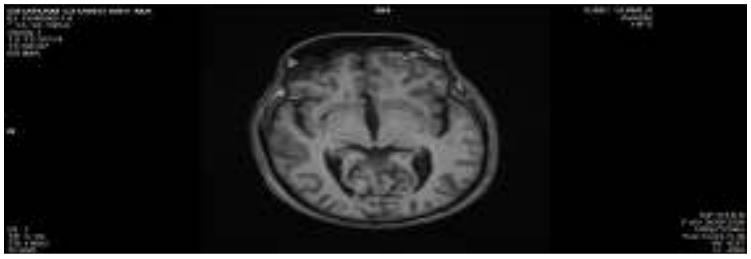

Figure 3: Axial T1 weighted image showing haemorrhagic contusion in the cerebral hemisphere in the left frontal region

DISCUSSION

The mechanism of damage to the optic nerve by closed head injury has been extensively studied. After blunt trauma to the superior orbital rim or fronto-temporal region of the cranium, compression forces are transmitted to the orbital apex and optic canal[3]. Within the canal, the optic nerve dura fuses with the periosteum of the bone. Since the vasculature of the optic nerve in the canal is pial, compression and contusion of the nerve produce a compartment syndrome whereby swelling exacerbates the ischaemia[4]. Panje *et al* suggested that frontal head trauma might cause stretching of the optic nerve[5]. Lessell proposed that ischemia is the pathogenetic mechanism in some cases[6]. Most cases of traumatic optic neuropathy occurred following road traffic accidents and the patients usually suffer from polytrauma with a poor general condition on admission. If the patient is unconscious, optic nerve injury is usually missed out until the patient regains consciousness and gives a history of loss of vision. Typically, the retina and optic disc appear normal at first, and the only objective finding at presentation is an afferent pupillary defect. Optic atrophy does not become apparent for 3-4 weeks[7]. Thus, precious time may be lost. In a series of 36 patients who sustained indirect injury to the optic nerve, Seiff reported that visual acuity improved in 62% and 33% of those given high dose i.v. dexamethasone and those given no treatment, respectively, with the difference being insignificant; however, the steroid-treated patients' vision improved at a significantly faster rate[8]. Spoor and colleagues compared results in patients treated with mega-dose i.v. methylprednisolone to outcomes in patients treated with high-dose dexamethasone. There was no significant difference between the two groups in terms of improved visual acuity. In addition, they found no statistical associations between the visual improvement that was achieved and total blindness at presentation, mechanism of injury, or time elapsed from injury to treatment[9]. However, Steinsapir and Goldberg suggested that mega-dose corticosteroid therapy must be started within 8 hours, which makes traumatic optic neuropathy a true ophthalmic emergency[2]. Wollin and Lavin described four patients with traumatic optic neuropathy who recovered vision after having presented with total blindness. Three

of these individuals were treated with corticosteroids, and one improved spontaneously[10]. The management of indirect optic nerve injury is controversial. There may be delay in diagnosis, and occasionally the loss of visual function appears as a delayed complication of head injury. The arguments for treatment of indirect optic nerve injuries are based upon the hypothesis that secondary injury to the axons occurs as a result of vasospasm and swelling within the optic canal. Experimental studies of optic nerve injury have employed crush, stretch, or severing injuries. The cellular mechanisms involved in CNS injury are incompletely understood. To summarize, several cellular messengers are activated by the trauma response. The release of oxygen-free radicals results in peroxidation of lipid cell membranes. Bradykinin and kallidin are activated following injury: these agents influence free radical production, intracellular calcium production, and arachidonic acid release from neurons. Subsequently cell mediated inflammation certainly plays a prominent part in experimental models of optic nerve injury. Currently, there is no validated approach to the management of traumatic optic neuropathy. With regard to treatment, there are presently three options: (i) careful observation; (ii) systemic corticosteroid therapy or; (iii) optic nerve decompression surgery. The rationale for i.v. corticosteroids in the treatment of traumatic optic neuropathy was derived from the results of the National Acute Spinal Cord Injury Study 2 (NASCIS 2). The NASCIS 2 was a multicenter clinical trial that evaluated patients with acute spinal cord injury treated with placebo, methylprednisolone or naloxone. Pharmacologically, corticosteroids are considered to reduce microvascular spasm and soft tissue edema via stabilization of the microvascular circulation and calcium homeostasis, thereby enhancing bloodflow and reducing cell death. The study showed that methylprednisolone 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. Although widely accepted, the question whether corticosteroids are of similar effect in the treatment of traumatic optic neuropathy is unproven. The majority of case reports and series with corticosteroids in traumatic optic neuropathy are retrospective, non-consecutive, non-randomized and uncontrolled. Meanwhile, several nonclinical studies have questioned the therapeutic benefit associated with corticosteroids in acute traumatic optic neuropathy[2]. The results from the CRASH trial indicated an even higher risk of mortality in patients with head injury treated with high-dose corticosteroids, thus, making the modality of management of this condition all the more precarious[11-18]. Recent studies have concluded that medical and/or surgical intervention might be of questionable value in many cases. It has been suggested that patients without negative prognostic indicators may be effectively managed with careful monitoring of their resolution. Furthermore, research has shown that there is no significant difference in final visual acuity relating to dose (low vs. high vs. mega) or timing of corticosteroid therapy, nor is there a significantly different outcome between patients treated with steroids or surgical decompression of the optic canal. Patients with traumatic optic neuropathy should, therefore, be addressed and managed via one or more of the above outlined options on an individual basis, following proper assessment and consultation[7,19].

CONCLUSION

Traumatic optic neuropathy associated with blunt forehead trauma is a potentially vision-threatening process that requires comprehensive clinical assessment and appropriate neuroimaging. A trial of i.v. methylprednisolone is worth giving at any duration of presentation within 4-6 weeks rather than waiting for the patient to go blind.

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Source of Support: None Declared
Conflict of Interest: None Declared