Traumatic optic neuropathy after head injury

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Abstract

Purpose: To study the clinical profile, prognostic factors and the effect of systemic steroids in the management of traumatic optic neuropathy.

Materials and Method: A minimum of forty four patients with closed head injury with complaints of any of the following: decreased visual acquity following trauma, RAPD, defective colour vision, visual field defects or CT scan showing fracture of any orbital wall were selected randomly and detailed ocular examination was done. Patients were treated with IVMP.

Results: Patient age ranged from 11-69 years. The causes of head injury were road traffic accidents (86.4%), accidental falls (9.1%), and assault (4.5%). Majority of patients were male (93.18%). Unilateral involvement was seen in 95.45% cases. 61.36% cases were drowsy, 36.36% cases conscious and 2.27% cases were unconscious after the injury. In this study 34.78% of patients had profound loss of vision PL+ or HM (hand movements) as the initial vision. 13.04% cases had a visual acquity 6/18 or better. In 43.47% cases extraocular movements were restricted. RAPD was present in 95.5% of eyes. 82.60% of patients presented with sub conjunctival hemorrhage. CT scan coronal view showed optic nerve sheath thickening in 17.39% patients. The most common fractures noted were fracture floor of orbit in 23.19% cases and medial wall of orbit in 15.21% cases. 38 patients were treated with intravenous Methyl prednisolone within 24 -72 hrs. of suspicion of traumatic optic neuropathy. Initial ophthalmoscopic examination was conducted on 46 eyes and optic disc was normal in all cases except one who showed mild disc edema. In 6 to 12 weeks more than 50% optic discs became pale. Optic disc pallor developed in about 81.5 % cases in 6 months. In the present study out of six; 5 patients showed spontaneous improvement without treatment, but treated patients appeared to have a better visual acuity and visual fields.

Conclusion: Road traffic accidents were the most common cause associated with TON and males being more commonly involved. Treated patients had a better visual prognosis. On follow up, it was noted that visual acuity improved in patients who had initial vision more than light perception and in patients with no optic canal fractures.

Keywords: Head injury, Optic canal fracture, Optic nerve, Steroids, Traumatic optic neuropathy

Introduction

Traumatic optic neuropathy (TON) is a form of optic neuropathy due to an acute injury of the optic nerve secondary to trauma. Traumatic nerve damage after craniofacial injury was first described by Hippocrates.(1)

The optic nerve is vulnerable to both direct and indirect trauma causing functional impairment of vision. Direct injury arises from penetrating trauma, especially orbital fractures associated with mid-facial fractures. Indirect optic nerve injury is more common. The force of impact in a head injury may be transmitted to the optic nerve.

The hallmark of all optic neuropathy is loss of visual function. Traumatic optic neuropathy occurs in about 0.5-5% of patients presenting with closed head trauma and in 2.5% of patients presenting with mid-facial fracture.(2)

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.

The pathogenesis of traumatic optic neuropathy is unclear. Direct and indirect injuries both cause mechanical and ischemic damage to the optic nerve. It is now realized that neural damage occurs at cellular and sub cellular levels following ischemia. The roles of oxygen free radicals, enzymes, cytokines, intracellular calcium and other form of reperfusion damage are being slowly uncovered through basic science research.(3,4)

The diagnosis of traumatic optic neuropathy is clinical. Following mid-facial and cranial trauma, a high index of suspicion for a traumatic optic neuropathy is to be entertained.(3,4,5)

The optimal treatment for this condition is controversial and the management tends to be on the basis of personal preference. The management of a patient with traumatic optic neuropathy is essentially by a multi-disciplinary approach involving the ophthalmologist, physician, neuro-surgeon, and an otorhinolaryngologist. Most widely accepted contemporary treatments for traumatic optic neuropathy include: Careful observation, systemic corticosteroid therapy, and optic nerve decompression by surgery.(4,5,6)

An attempt has been made in this study, to analyse the etiology, clinical profile, management and visual outcome in patients with traumatic optic neuropathy following closed head injury.

Material and Method

A minimum of 44 patients with closed head injury with complaints of any of the following: decreased visual acquity following trauma, RAPD, defective

colour vision, visual field defects or CT scan showing fracture of any orbital wall were selected randomly irrespective of age, gender, place and mode of injury. Study was done in ophthalmology department of Santosh Medical College and Hospital, Ghaziabad during a period from January 2015 to June 2016.

A detailed history was obtained from all patients or from relative if patient was unconscious. Patient's name, age, sex, nature of injury, laterality of eye, use of helmet and status at the time of injury were noted.

A thorough clinical examination was undertaken with a torch light to see any external wound near eye, periorbital emphysema, proptosis, and subconjunctival hemorrhage. Extra ocular movements were checked. Best corrected visual acuity was quantitated at bedside with a near card/reduced Snellen eye chart. Pupillary reaction was checked. An evaluation for a relative afferent pupillary defect (RAPD) was noted. A relative afferent pupillary deficit is a necessary condition for the diagnosis of TON in unilateral cases. This is the most important clinical sign in an unresponsive patient.

The fundoscopic examination was done with the use of a direct ophthalmoscope, or the slit lamp biomicroscope after full dilatation of pupil with a short acting mydriatic agent. Thorough fundus examination was done to see the macula, disc and rest of the retina. Because the location of injury in most cases is within the posterior orbit or the optic canal, the optic disc in most cases, typically appeared normal, on fundoscopic examination on initial diagnosis.[7] Optic nerve atrophy usually appears 3-4 weeks after the traumatic event,[8] and the disc acquires a diffuse pallor.

Color vision defect was detected by using the red capped eye drop bottle, especially in one eye compared to the other. Visual field charting was done to see the visual field defects.

In the post-trauma setting, CT scanning is the preferred modality for demonstrating the presence of an optic canal fracture, a displaced bony fragment impinging upon the optic nerve, a metallic foreign body in the orbit, orbital emphysema, or an optic nerve sheath hematoma.

In the present study, patients were treated with intravenous Methyl prednisolone within 24 -72 hours of detection of traumatic optic neuropathy, 500 mg twice daily for three days followed by oral prednisolone 1 mg/kg/day for two weeks in tapering doses. All cases were given H2 antagonists. Patients were regularly followed up at 1 week, 1 month, 3 months and then at 6 months.

Results and Discussion

The following observations were made during the study:

Out of 44 cases, 5(11.36%) cases were in 10-20 years of age group, 16(36.36%) in 21-30 years age group, 13(29.54%) were in 31-40 years age group, 6(13.63%) in 41-50 years, 3(6.82%) were in 51-60 years and 1(2.27% 69years). Overall, 41(93.18%) cases were males and 3(6.81%) were females.

Out of 44 cases, 38(86.4%) cases sustained head injury due to road traffic accidents, 4(9.1%) by accidental falls and 2(4.5%) because of assaults. 42(95.45%) cases had unilateral involvement while bilateral involvement was seen in 2(4.55%) cases. Among 42 cases of unilateral involvement, right eye was involved in 14(33.33%) and left eye in 28(66.7%) cases. Out of 28 cases involved in two wheeler accidents, only 2(7.14%) cases were wearing helmet at the time of accident. A total of 16(36.36%) cases were conscious, 27(61.36%) were drowsy and 1(2.27%) case was unconscious. No perception of light was seen in 3(6.52%) cases while 16(34.78%) cases had hand movement to PL+ vision. 12(26.08%) cases had visual acquity of <5/60 and 9(19.56%) cases were having a visual acquity ranging from 6/24 to 6/60. 6(13.04%) cases had a visual acquity of 6/18 or better. Out of 46 involved eyes 20(43.47%) had restriction of extraocular movements.

RAPD was detected in 42(91.30%) eyes. 4(8.69%) cases has traumatic mydriasis. Cases with less than 6/60 visual acquity had marked RAPD. Visual field charting was difficult in initial stages due to drowsiness of the patients but later on visual field charting showed central and paracentral visual field defects in patients who retained adequate vision. Red color desaturation was present in all cases of traumatic optic neuropathy. On initial ophthalmoscopic examination optic disc was normal in 44(95.65%) eyes and 1(2.2%) eye showed optic disc edema and in 1(2.2%) eye retinal hemorrhage was seen. In 6 to 12 weeks more than 50% discs became pale. Optic disc pallor developed in about 81.5% cases in 6 months.

Injury near eyebrow and forehead was present in 30(65.21%) cases. Ecchymosis in 40(86.95%) cases, periorbital emphysema in 27(58.69%) cases, subconjunctival hemorrhage in 38(82.60%) cases and proptosis was seen in 9(19.56%) cases. CT scan head and orbit was done in all cases as a part of neurosurgical examination. CT scan coronal view showed optic nerve sheath thickening in 8(17.39%) cases. The most common fracture was # floor of orbit 11(23.91%) cases followed by # medial wall of orbit 7(15.21%) cases. # body of sphenoid was present in 4(8.69%) cases and # lateral wall of orbit in 1(2.17%) case. Optic canal fracture was seen in 1(2.17%). 40(86.95%) cases were treated with 500 mg intravenous methyl prednisolone over 30 minutes time, twice daily for 3 days, followed by oral prednisolone 1 mg/kg/day for two weeks in tapering doses. All cases were given H2 antagonists and antacids. A total of six cases were managed conservatively, as they presented after 72 hours of the trauma. Five cases showed spontaneous improvement without treatment.

Table 1: Age distribution

Age	No. of cases	Percentage (%)
10-20	5	11.36%
21-30	16	36.36%
31-40	13	29.54%
41-50	6	13.63%
51-60	3	6.82
61-70	1	2.27
Total	44	

Table 2: Initial visual acquity of affected eye

Visual acuity	Total no of eyes	Percentage (%)
>6/18	6	13.04%
6/24 – 6/60	9	19.56%
<5/60	12	26.08%
HM and PL +	16	34.78%
NO PL	3	6.52%

Table 3: Associated periocular injuries

Clinical findings	No of eyes (46)	Percentage (%)
Injury near eye brow and forehead	30	65.21%
Ecchymosis	40	86.95%
Peri orbital emphysema	27	58.69%
Subconjunctival hemorrhage	38	82.60%
Proptosis	09	19.56%
Lid avulsion from medial canthus	1	02.2%

Discussion

Age incidence: In this study age ranged from 11 to 69 years. It was noted that patients between 20-50 years age group were most commonly (79.53%) affected with a peak incidence of TON in the age group 21 to 30 years(36.36%) followed by patients between 31-40 years(29.54%) age group. Similar results were observed in a study done by M. G. Rajiganth, Ashok K. Gupta, et al.[9] in 2003 where the average age of the patients with traumatic optic neuropathy was 28 years.

Sex incidence: In the present study, out of 44 patients, 41 were males and only 3 were females which are consistent with other studies.[9,10,11] In the study done by M. G. Rajinigath,[9] Ashok K. Gupta et al. in 2003, predominantly male (93%) patients were involved. In yet another study done by Harsha Bhattacharjee et al.[12] (2008) it was noted that male patients were more often involved (91%) as compared to females (9%).

Nature of injury: In this study road traffic accidents were commonest cause of trauma (86.40%) than assault or other modes of head injury. Similar findings were shown in Sadeghi-Tari study.[13] Car crashes were the most common cause of trauma (63%), followed by falls and assaults in the study done by M. G. Rajiniganth,[9] Ashok K. Gupta, et al. in 2003.

Similar findings were observed in the International Optic Nerve Trauma Study. Lessell[14] found that the commonest mode of such an injury was fall from bicycle, closely followed by automobile collision. In the study of traumatic optic neuropathy by James W. Gigantelli[15] (2003) following causes of injury were reported: trauma due to motor vehicles (motorcycle and bicycle accidents), falls, assaults, penetrating orbital trauma, stab wounds, gunshot wounds, foreign bodies, and recreational sports. In children trauma was often due to motor vehicle accidents or fall.

Laterality: In the present study out of 44 cases unilateral involvement was seen in 91.3% cases. Bilateral involvement was less common. RE was involved in 33.33% cases. In the study done by Harsha Bhattacharjee et al. (2008),[12] right and left laterality of the injury was 11 (31%) and 23 (69%) respectively.

Patient's status: In the present study, majority of the patients were drowsy 27(61.36%) cases and non-co-operative in causality consultation. In the study done by Harsha Bhattacharjee et al (2008)[12] twenty eight (80%) cases out of 35 cases were unconscious. Thirty-three patients (75%) presented with loss of consciousness in the study done by M. G. Rajiniganth,[9] Ashok K. Gupta, et al. in 2003.

Initial visual acquity: In this study 16 cases (34.78%) had profound loss of vision PL+/- or HM (hand movements) as the initial vision. Visual acuity ranged from no perception of light in 12 (34%) cases to 20/30 in one (3%) case in study done by Harsha Bhattacharjee[12] et al. (2008). 81.81% cases presented with complete loss of vision (no PL), in the study by M.G. Rajiniganth,[9] Ashok K. Gupta, et al. in 2003.

Extraocular movement: In current study extra ocular movements were affected in 20(43.47%) out of 46 eyes. Extra ocular movements were full in 26 eyes (53.35%). Ptosis with restricted extraocular movement in 3 patients (7%) out of 44 patients was observed by M.G. Rajiniganth,[9] Ashok K. Gupta, et al. in 2003.

Visual field defect: In the present study, visual field charting was difficult in the initial stages due to drowsiness of the patients and later on visual field charting showed central and paracentral visual field defects in patients who retained adequate vision. The commonest Visual field defects detected were central, paracentral scotomas, sector like defects and altitudinal (usually inferior), hemianopsia. (Traquair 1929).

Color vision defect: Red color desaturation was present in all cases with traumatic optic neuropathy. Patients with optic neuropathy often have red color desaturation and a positive response would be that the red color looks faded, pink or washed out.[14,16]

RAPD: In this study Relative afferent pupillary defect was detected in 42eyes (95.5%). Similar results were observed in the study done by Harsha Bhattacharjee et al. (2008)[12]. In the study done by M. G. Rajiniganth,[9]

Ashok K. Gupta, et al. in 2003 a relative afferent pupillary defect was detected in 40 patients (91%).

Ophthalmoscopic examination: In the present study ocular media was clear in all cases. Ophthalmic examination showed normal fundus in 44 eyes, mild disc edema in one eye and retinal hemorrhage in another eye. Similar observation was an initial fundus evaluation revealing a normal optic disc in 40 patients (91%) in the study done by, M. G. Rajiniganth,[9] Ashok K. Gupta, et al. in 2003. Whereas according to the study done by Harsha Bhattacharjee et al. (2008)[12] variable amounts of optic disc changes were found. It revealed generalized pallor in 23 (66%) cases, normal disc appearance in five (14%), temporal pallor in four (11%) cases, primary looking optic atrophy in two (6%) cases, doubtful pallor in one (3%) case, hyperaemic disc in one (3%) case. In the present study on follow up examination, two patients developed disc pallor after two weeks. In 6 to 12 weeks more than 50% optic discs became pale.

CT scan findings: In this study CT scan coronal view showed optic nerve sheath thickening in 8 patients. The most common fractures noted were fracture floor of orbit in 11 cases (23.91%) and medial wall of orbit in 7 cases (15.21%). The HRCT and MRI of the cases did not reveal any abnormal findings, except in two cases one had hairline fracture in the orbital plate of the frontal bone and the other has diffused swelling of the optic nerve in the study done by Harsha Bhattacharjee,[12] Kasturi Bhattacharjee et al in 2008, on 35 cases. CT scan revealed a canalicular fracture in the optic canal in 15 patients (34%0 in the study done by M. G. Raginiganth,[9] Ashok K. Gupta et al in 2003.

Treatment: In the present study, 39 patients were treated with intravenous Methyl prednisolone within 24 -72 hrs of detection of traumatic optic neuropathy, 500mg twice daily for three days followed by oral prednisolone 1mg /kg/day for two weeks in tapering doses. All cases were given H2 antagonists and antacids. In our study, no surgical optic canal decompression was performed because none of our patients reported having optic nerve impingement in radiological investigations. A total of six cases with unilateral involvement were managed conservatively, as they presented after 72 hours of the trauma. Spontaneous improvement of vision was noted in 5 out of 6 cases managed conservatively. Improvement in vision occurs in 20-40% of patients who are left untreated (Walsh).[17] In the study done by Harsha Bhattacharjee et al[12] (2008), 18% of the cases had variable and spontaneous visual improvement in reference to the presenting vision during final checkups. In the study done by M.G. Raginiganth,[9] Ashok K. Gupta et all in 2003, out of the 44 patients, 10 showed improvement after steroid therapy and 11 showed improvement after endoscopic optic nerve decompression, with an overall improvement of 48%. Harsha Bhattacharjee et al.(2008)[12] could not find any obvious benefit of IVMP in management of traumatic optic neuropathy cases as observed in the International Optic Nerve Trauma Study.[4]

In the present study patients treated with corticosteroids had a better visual prognosis. Treatment with mega dose corticosteroids seems to improve vision more quickly than treatment conventional intravenous corticosteroids. However there was no significant difference in the final visual outcome. Initial visual acuity and subsequent improvement was much poorer in patients with hemosinus sphenoid and ethmoid. Hence presence of poor initial visual acuity (No PL), presence of optic canal fractures, presence of multiple fractures of orbital walls, no improvement in vision within 48 hours of starting intravenous methyl prednisolone were all indicators of a poor visual prognosis in this study.

The treatment of TON is a somewhat controversial. There is not any specific guidance how to treat and weather to treat at all. But the treated patients statistically did better than the untreated patients.

Since there are no definite recommendations other than to treat on the individual basis, we can tell the patient "we can do nothing and hope that the vision will improve". The families of such patients should be taken in to confidence and explained that there is no proven therapy for such patients.

Conclusion

From this study we have concluded that most of the traumatic optic neuropathy patients were presented with ecchymosis and subconjunctival haemorrhage. Patients treated with corticosteroids had a better visual outcome. Road traffic accidents were the most common cause associated with TON and males being more commonly involved. On follow up, it was noted that visual acuity improved in patients who had initial vision more than light perception and in patients with no optic canal fractures.

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