

# Traumatic optic neuropathy: a review of 24 patients

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## Abstract

• **AIM:** To evaluate the clinical presentations of traumatic optic neuropathy and to assess the visual outcome of three groups of patients managed differently (conservative, intravenous corticosteroids only and combination of intravenous and oral corticosteroids) at an academic tertiary care referral centre.

• **METHODS:** A retrospective study was conducted involving 24 consecutive patients (27 eyes) with traumatic optic neuropathy attending Hospital Universiti Sains Malaysia from January 2007 till December 2009.

• **RESULTS:** Twenty-four patients (27 eyes) were included. All cases involved were male. Mean age was 33 years old. Motor vehicle accident was the major cause (83%). Both eyes were equally involved. Most of the eyes had poor vision on presentation (HM-NPL, 82%) with associated periorbital haematoma (22 eyes) and subconjunctival haemorrhage (20 eyes). Majority of patients (19 patients, 79%) presented with more than one bony fracture of skull or orbit and 5 patients (21%) had no fractures. None of the patients had evidence of optic nerve compression on CT scans or MRI done. Eleven patients (46%) had been treated with intravenous and oral corticosteroids. The other 7 patients (29%) were treated conservatively and the third group (6 patients, 25%) was on intravenous corticosteroids only. Eleven of 12 eyes (92%) treated with intravenous and oral corticosteroids had showed 1 line improvement of visual acuity. Those eyes treated conservatively (78%) had showed 1 line improvement of visual acuity. As for patients treated with intravenous corticosteroids only, four patients remained NPL, one patient had mild visual improvement and the other one's vision remained the same. The visual improvement in patients treated with conservative management was not significant ( $P = 0.386$ ). Patients treated with intravenous corticosteroids alone have shown no statistical visual improvement ( $P < 0.05$ ). Patients treated with intravenous followed by oral corticosteroids had significant visual improvement ( $P < 0.05$ ). There was no statistically significant difference in visual outcome between patients treated with corticosteroids and patients treated conservatively ( $P = 0.368$ ). No

patient underwent surgical decompression of the optic nerve. In this series, the follow-up ranged from 6 months to 3 years.

• **CONCLUSION:** Most of the traumatic optic neuropathy patients are presented with periorbital haematoma, subconjunctival haemorrhage and orbital wall fractures. Patients treated with intravenous followed by oral corticosteroids have better visual outcome compared to conservative management.

• **KEYWORDS:** traumatic optic neuropathy; orbital fractures; corticosteroids

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## INTRODUCTION

Optic neuropathy is a potential blinding complication of head or orbital trauma. The most common form of traumatic optic neuropathy is indirect damage to the optic nerve and has been reported following 0.5% to 5% of all closed head trauma<sup>[1]</sup>. It is defined as traumatic visual loss which occurs without external or initial ophthalmoscopic evidence of injury to the eye or its nerve<sup>[2]</sup>. Indirect injuries are caused by concussive forces that are transmitted to the optic nerve as a result of orbitofacial or cranial trauma<sup>[3,4]</sup>. This impact may generate a shock wave which can lead to optic nerve avulsion or posterior indirect traumatic optic neuropathy<sup>[5,6]</sup>. On the other hand, direct traumatic optic neuropathy results from direct trauma to the optic nerve from sharp objects, missiles and bony fragments<sup>[7]</sup>. The clinical presentations vary. The degree of the visual loss does not always correlate with the severity of trauma. The presence or severity of fractures of the orbit neither directly predicts the severity of visual loss nor determines prognosis<sup>[8]</sup>. The optimal treatment of traumatic optic neuropathy remains controversial. There has been no conclusive evidence for standardized treatment protocol due to lacking of large randomised control trials of management as a result of low incidence of this condition<sup>[2,9]</sup>. Observation, corticosteroids treatment and decompression of the optic nerve have been advocated and significant recovery of vision has been found in those treated with corticosteroids, optic nerve decompression, or both as compared to observation alone<sup>[10]</sup>.

## MATERIALS AND METHODS

**Subjects** A retrospective study was conducted involving 24 consecutive patients (27 eyes) with traumatic optic neuropathy attending Hospital Universiti Sains Malaysia (academic tertiary care referral centre) between January 2007

and December 2009. The patients' particulars were obtained using optic neuropathy database and the medical records were reviewed. The data of 24 patients (27 eyes) were analyzed. All the cases were male. The age ranged from 5 to 77 years old with mean age of 33 years old. The age group involved was predominantly 11-30 years old group (58%). The major causes of trauma were motor vehicle accident (83%), followed by blunt trauma (13%) and fall (4%). Most of the patients were healthy (71%) and a few of them had diabetes mellitus, hypertension and ischemic heart disease.

**Methods** All cases had complete eye examination including associated ocular injuries. The CT scans of brain and/or orbit were reviewed to evaluate the extent of injuries. The visual acuity was the main outcome measure of the study, which was measured by Snellen chart. The visual acuity was assessed at presentation, upon completing corticosteroids and on last follow-up. The associated ocular injuries were carefully evaluated. The treatment regimens were divided into 3 groups. This includes those who were treated conservatively, intravenous methylprednisolone 250mg *qid* for 3 days without commencement of oral prednisolone and a group which was started on intravenous methylprednisolone 250mg *qid* for 3 days followed by oral prednisolone 1 mg/kg for 11 days.

**RESULTS**

Most of the eyes had poor vision on presentation (HM-NPL, 63%). Periorbital haematoma was present in all cases with skull and/or orbital fractures (Table 1, 2). The CT scans or MRI of brain and/or orbit revealed seven patients with intracranial bleeding. Majority of patients (19 patients, 79%) presented with more than one bony fracture of skull and/or orbit and 5 patients (21%) had no fractures. None of the patients had evidence of optic nerve compression on CT scan or MRI. Majority of cases had generalized visual field defect on confrontation test, one patient with poor Glasgow Coma Scale and one paediatric case. All unilateral cases have reverse afferent pupillary defect positive except the three cases with bilateral involvement.

There were 3 groups of patients which had been managed differently. Eleven patients (46%) had been treated with intravenous methylprednisolone 250mg *qid* for 3 days followed by oral prednisolone 1 mg/kg for 11 days. The second group (7 patients, 29%) was treated conservatively due to several reasons including severe head injury (4 patients), uncontrolled diabetes mellitus (2 patients) and young age (1 patient). The third group (6 patients, 25%) was on intravenous methylprednisolone 250mg *qid* for 3 days only. Oral prednisolone was not commenced due to no improvement in the visual acuity after intravenous corticosteroid. There was 78% of patients had visual acuity between 5/60 to NPL and 22% had 6/12 or better in patients managed conservatively. In the third group, 92% of patients had visual acuity between 5/60 to NPL and no patient had 6/12 or better vision.

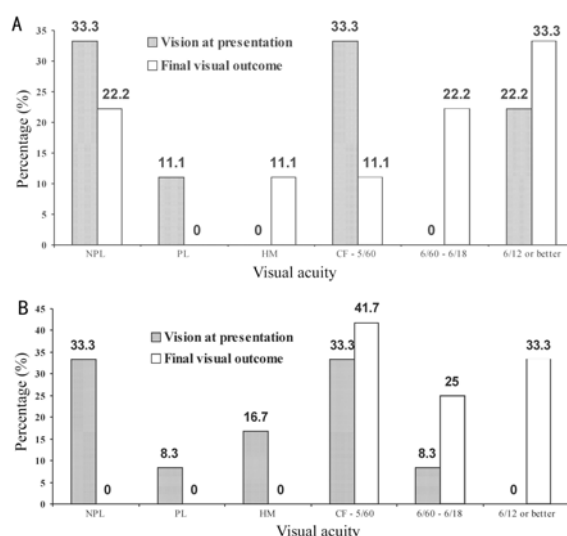
The follow-up ranged from 6 months to 3 years. Eleven of 12 eyes (92%) treated with intravenous methylprednisolone 250mg *qid* for 3 days followed by oral prednisolone 1mg/kg for 11 days had showed at least 1 line improvement of visual acuity

**Table 1 Associated eye injuries in 24 patients**

Types of injury	n
Periorbital hematoma	22
Subconjunctival hemorrhage	20
Scleral laceration	1
Exposure keratopathy	1
Corneal abrasion	1
Microhyphaema	2
Uveitis	1
Anterior lens subluxation	1
Comotio retinae (Berlin's edema)	2
Retroorbital hematoma	1
Carotid carvenous fistula	1
Other cranial nerve involvement	
3 <sup>rd</sup> cranial nerve	1
4 <sup>th</sup> cranial nerve	2
Total ophthalmoplegia	1

**Table 2 Orbital and skull fractures based on CT scan or MRI findings**

Findings	n
Orbital wall fracture	19
Lateral orbital wall	3
Medial orbital wall	5
Superior orbital wall	3
Orbital floor	2
Zygomatic complex	6
Skull fracture	10
Basal skull	2
Frontal bone	6
Parietal bone	1
Occipital bone	1
No fracture	5



**Figure 1 Comparison of visual acuity based on management**  
 A: Conservative management; B: Intravenous methylprednisolone and oral prednisolone. CF: Counting finger; HM: hand movement; PL: Perception of light; NPL: Non-perception of light.

(Figure 1). Those eyes treated conservatively (78%) had showed at least 1 line improvement of visual acuity (Figure 1).

Four patients treated with intravenous corticosteroids only remained NPL, one patient had mild visual improvement from HM to CF only (16.7%) and the other one's vision remained the same.

Patients treated with intravenous followed by oral corticosteroids had significant visual improvement ( $P < 0.05$ ). The visual improvement in patients treated with conservative management was not significant ( $P = 0.386$ ). Therefore, patients treated with intravenous followed by oral corticosteroids have better visual outcome compared to patients with conservative management. No patients underwent any surgical intervention.

## DISCUSSION

Traumatic optic neuropathy can cause optic nerve morbidity such as loss of vision, deficits in visual field, colour perception and an afferent pupillary defect. The diagnostic features of traumatic optic neuropathy are visual loss that occurs in the presence of a relative afferent pupillary defect without evidence of injury to the optic nerve or eye. The commonest site of indirect optic nerve injury is the optic canal<sup>[11]</sup>. Chou *et al*<sup>[12]</sup> had proposed that the damage of optic nerve at microscopic level, including contusion necrosis, nerve fibre tears and nerve infarction secondary to closed space edema, hemorrhage, thrombosis, vasospasm, impingement by bone spicules, and shearing of dural vessels in the optic canal. In an experimental study of primates, significant descending degeneration of the retinal ganglion did not occur until about 3 weeks after optic nerve transection, with maximum loss at 6-8 weeks following the injury<sup>[13-16]</sup>.

All our patients with traumatic optic neuropathy were young male, most aged 11 to 30 (58.3%) years old which is consistent with other studies<sup>[17-19]</sup>. Motor vehicle accidents (83%) were the main cause of traumatic optic neuropathy in our study, while blunt trauma had the second highest incidence rate (12.5%) and followed by fall (4.2%). Similar findings were showed in Sadeghi-Tari study<sup>[3,8,9]</sup>. The involvement of right and left eyes were almost similar. We followed up the patients between 6 months to 3 years and all the patients reached the final vision at 6 months' duration of follow-up as similar in other studies<sup>[20,21]</sup>.

In the present study, the morbidity associated with traumatic optic neuropathy was decreased visual acuity and relative afferent pupillary defect, which was elicited by swinging flashlight test. The ocular manifestations most commonly associated with optic nerve injury were periorbital hematoma and subconjunctival hemorrhage, with no immediate changes in the optic nerve. The ocular manifestations that were observed in our study are consistent with other studies<sup>[17,18]</sup>. Most of the eyes with traumatic optic neuropathy were associated with periorbital hematoma and orbital walls and/or skull fractures. All patients with NPL had periorbital hematoma with subconjunctival hemorrhage and 85% of NPL eyes were associated with orbital walls and skull fractures. These signs may indicate the increased morbidity to the optic nerve and strong relation to traumatic optic neuropathy in cases without evidence of optic nerve impingement or

compression.

The treatment of traumatic optic neuropathy includes keeping patients under observation, administering corticosteroid therapy, or performing optic nerve decompression with or without steroid therapy<sup>[22]</sup>. In our study, no surgical optic canal decompression was performed because none of our patients reported having any optic canal fracture or optic nerve impingement in radiological investigation. There is no available optimal management protocol, as most of the published data are either retrospective or presented in case reports<sup>[18]</sup>. The International Optic Nerve Trauma Study, which was undertaken to formulate an optimal management protocol, failed because of the limited number of eligible patients. The majority of published data do not clearly define the criteria of visual improvement; some studies defined improvement as an increase in 1 to 3 lines in visual acuity<sup>[11,20]</sup>. Methylprednisolone therapy was advocated as the initial treatment of choice because of its neuroprotective mechanism<sup>[23]</sup>. The exact mechanism of corticosteroids in reducing optic nerve injury is still unclear. Steroids may have the neuroprotective effects of decreasing the intraneural or extraneural edema and relieving compression of the nerve fibres. By reducing vasospasm steroids may also limit contusion necrosis of the nerve and block neuronal death in the setting of trauma is through inhibition of free radicals<sup>[9]</sup>.

The International Optic Nerve Trauma Study reported visual acuity improvement in 52% of patients treated with high dose steroids and 37% of patients treated with mega dose steroids in Sadeghi-Tari study<sup>[9]</sup> after three months' follow-up. Eleven of 12 eyes (92%) treated with intravenous methylprednisolone and oral prednisolone had improved at least 1 line of visual acuity, compared to the eyes (78%) that were managed conservatively. The visual improvement in patients treated with conservative management was not significant ( $P = 0.386$ ). Those patients treated with intravenous corticosteroids alone showed no visual improvement statistically ( $P < 0.05$ ). However, all eyes only treated with intravenous methylprednisolone did not show vision improvement after 3 days of steroid; therefore the oral steroids were discontinued. In this group, 67% of eyes remained NPL after 6 months of follow-up. It indicates that continuation of oral steroids was only beneficial to those eyes with immediate visual improvement after intravenous methylprednisolone.

There are a few limitations in our study. Firstly, this is a retrospective study that leads to the inability to randomly assign patients to each treatment option to ensure a meaningful comparison. Secondly, there is a lack of eligible patients in each group and the number of patients in each group is not similar. Furthermore, no surgical intervention or optic decompression treatment was included because none of them have optic nerve impingement or compression.

In 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 under conservative management.

The continuation of oral corticosteroid is only beneficial to those eyes with immediate visual improvement after intravenous corticosteroid.

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## 外伤性视神经病变 24 例回顾研究

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### 摘要

**目的:**探讨的外伤性视神经病变的临床表现,评估在眼科第三病区住院的经过三组不同处理(保守估计,单独静脉注射皮质类固醇,静脉注射和口服皮质类固醇联合治疗)的外伤性视神经病变患者的治疗效果。

**方法:**对 2007-01/2009-12 在马来西亚医科大学眼科连续住院的 24 例 27 眼外伤性视神经患者进行了回顾性研究。

**结果:**本次研究对象为 24 例 27 眼男性外伤性视神经患者(平均年龄为 33 岁)。车祸是导致发病的主要原因(83%)。大部分患者的视力低下(手动/眼前~无光感约占 82%),其中有 22 眼并发眶周水肿,20 眼并发有结膜下出血。并发多于一骨(颅骨或眶骨)骨折有 19 例(79%),5 例(21%)没有出现骨折。CT 扫描或核磁共振检查显示患者均没有视神经压迫的证据。第一组:11 例(46%)患者进行静脉注射联合口服类固醇的治疗;第二组:7 例患者(29%)进行保守治疗;第三组:6 例(25%)患者给予静脉注射皮质类固醇治疗。92%(11/12 眼)给予静脉注射和口服类固醇治疗的患者和 78% 保守治疗患者视力能提高 1 行。单独静脉注射皮质类固醇治疗的患者 4 例呈现无光感,1 例有轻度视力改善,而其他患者视力保持不变。保守治疗患者和单独静脉注射皮质类固醇患者视力改善不显著,且两组间相比在统计学上无显著性差异( $P=0.368$ )。静脉注射联合口服类固醇治疗的患者有明显的视觉改善( $P<0.05$ )。没有患者接受视神经减压手术。在本研究中,随访时间为 6mo~3a。

**结论:**大部分外伤性视神经病变患者都存在有眶周水肿,结膜下出血,眶壁骨折的症状。对比保守治疗,经过静脉注射和口服皮质类固醇联合治疗的患者有更好的治疗效果。

**关键词:**外伤性视神经病变;眼眶骨折;皮质类固醇