

Traumatic Optic Neuropath: Is this the Ideal Management Protocol?

Ehtesham Ahmad Raushan¹, Alok Kumar²

^{1,2}Senior Resident, NMCH, Patna.

Abstract

Background: The aim of this randomized prospective study was to evaluate the management protocol decided in the cases of Traumatic optic neuropathy. **Subjects and Methods:** All the cases with a diagnosis of Traumatic optic neuropathy were managed using medical management with the steroids and cases not responding were operated upon by transnasal trans sphenoidal optic nerve decompression and the results were evaluated. **Results:** Patients presenting early and those who were operated early in the course of their illness showed a better recovery. **Conclusion:** Early intervention and following a defined management protocol can help preventing the blindness in as many as 80% of the cases following the injury.

Keywords: Traumatic optic neuropathy. Management, ideal.

Corresponding Author: Dr. Ehtesham Ahmad Raushan, Senior resident, N M C H, Patna.

Received: December 2019

Accepted: December 2019

Introduction

Optic nerve injury, an uncommon entity, is reported in 0.5 – 5% of the closed head injuries but the incidence is on the rise due to increasing incidence of high speed motor vehicle accidents. The acute onset of the vision loss the often complicated circumstances of the injury. The role of multiple disciplines and ill defined management protocol has led to interest in the condition¹. The management protocol is undergoing a rapid transition. There are only a few studies in the English Literature which have dealt with traumatic optic neuropathy.

We here report our experience of combined therapy protocol on cases of traumatic optic neuropathy.

Subjects and Methods

In this prospective study one hundred and three cases between the age group of 4 years to 53 years who presented to the emergency services department of Otolaryngology with clinical features of traumatic optic neuropathy over around 2 years period were included. The mode of injury and the interval between the injury and the arrival to our hospital were recorded. The patients visual status was evaluated at initial presentation. Computed tomography (CT) scan of the head and the orbit was performed in all the patients. All the cases were subjected to intravenous (IV) methyl prednisolone 20 mg /kg body weight per day in a single dose for a period of 3 days. Patients who improved minimally or remained static or worsened during the course of steroids were subjected to endonasal endoscopic optic nerve canal decompression. All the cases were subjected to endonasal endoscopic optic never canal decompression. All

the cases were subjected to oral steroids in the dose of 1 mg/kg/day in the tapering dose over two weeks period after the injectable steroids. The radiological and surgical finding were correlated. The results were analyzed using unpaired t-test.

Results

Out of the 103 case, there were 63 males and 40 females. The age ranged from 4 years to 53 years with a mean age of 33.63 years. The etiology was head injury as a result of road traffic accidents in 84 cases (81.55%) and blunt trauma to the head in 13 cases (12.62%) during recreational activities and as a result of intranasal injury by a blunt object in six of the cases (5.82%) Right side was affected in 65 cases (63.10%) and left in 38 (36.9%) Cases. Cases were divided into three groups on the basis of the interval between the injury and presentation to the Hospital with group 1 presenting within 24 hours of trauma. Group 2 presenting between 24 hours to 7 days and group 3 comprised of cases that presented after 7 days of trauma. There were 20 cases (19.41%) in group 1, 73 cases (70.87%) in group 2 and ten cases (9.70%) in group 3 with largest interval being 12 days. Forty three cases (41.74%) had presented with total loss of vision. CT scan revealed hematoma in the ipsilateral sphenoid sinus in 85 cases (82.53%) with fracture in the region of the optic canal (Figure 1) in 28 cases (27.18%) and fracture in the region of lamina papyracea in a8 (7.77%) cases. All of the cases were subjected to injectable methyl prednisolone and out of these 69 cases (66.99%) that were positive for perception of light at presentation. 41 cases (39.81%) improved and did not require any surgical intervention. The 34 cases that had no vision at presentation. 20 cases (19.41%) improved following the

medical therapy. A total of 42 cases (40.77%) were subjected to the surgical intervention and 24 (23.30%) of these cases showed visual improvement following surgery. Surgical findings in the cases were fracture of the optic canal in the cases with the edema of the optic nerve. Optic nerve sheath was not incised in any of the cases. There was a poor correlation of the radiological findings and the surgical findings with a correlation index of 0.4. None of the cases had any complications related to the surgical procedure.

Surgical Procedure:

All the cases were operated under general anesthesia with hypotension and with 15 degree head end elevation. The nasal cavity was decongested using xylocaine with adrenaline in a concentration of 1: 1,00,000. The middle turbinate was medialized and bulla was opened, ground lamella was then entered, posterior ethmoids and then the sphenoid sinus was entered and sinus was entered and sinus was widened in the inferomedial direction and lateral wall bone was removed in the region of the optic canal in whole of the length of canal. The nerve was decompressed in the whole segment of the canal then medicated pack was kept in nasal cavity which was removed on the first postoperative day.

Discussion

The relationship between frontal trauma and vision loss in the absence of ocular injury was appreciated by the 18th century. In the late 1800s. Battle first distinguished penetrating from non-penetrating indirect optic nerve injuries⁴. Trauma-induced injury to the optic nerve can occur anywhere along the nerve's intraorbital to intracranial course. The hallmark of the optic neuropathy. Traumatic or otherwise, is loss of visual function. This loss can manifest as subnormal visual acuity, visual field loss, or color vision dysfunction. The management protocol is undergoing a period of rapid transition from only medical to combined therapy management. A few studies done so far have not been conclusive in reaching a definitive protocol for the management.

In this study, We found males to be affected more often than females which is in accordance with the literature. The most common mode of injury was road traffic accidents in 82% cases whereas literature mentions slightly lower figure ranging from 50 to 62%.^[2,3] All of our cases had unilateral traumatic optic neuropathy but one of the studies mentions 14% cases with bilateral injury.^[2]

There was a poor correlation between the operative and the radiological findings where the cases had fracture of the optic canal on exploration did not have the radiological evidence of same.

We found that in group 1, nineteen cases out of twenty recovered following the steroids only and did not require any surgical intervention that improved following the decompression ($p < 0.001$). The cases in group 2, 54.54% recovered only with the steroid therapy. The remaining

cases which did not intervention. 60% of these improved making a total of 82.19% for the group 2 ($p < 0.05$). The cases in group 3, 20% improved following the steroid therapy and another 30% improved after the surgical intervention with a total of 50%. There was significant improvement in the cases and they continued to improve further on follow up. We did not incise the sheath in any of the cases as per the authors experience in dealing with the condition whereas literature mentions fenestration of the optic nerve sheath with no significant improvement in the vision.^[3,5] In the studies done so far, the surgical intervention was delay for a long period for up to 3 weeks with a final result of improvement in the vision in around 50% whereas we feel that early intervention helped in recovery of the vision in as much as 83% of the cases in our study.^[3,4,6] One of the study conducted showed the efficacy of delayed decompression.^[6]

The role of methylprednisolone also has been controversial.^[5,8,12,16,19] The reason for the early intervention as we recommend is to do with the anatomy of the optic canal. In the adult human, the optic canal is approximately 6.5 mm in diameter and 8-10 mm in length with the optic nerve having a diameter of 3-4 mm with a volume of around 1 ml. Structures that pass through the optic canal include the optic nerve axons, their supportive glia, the ophthalmic artery, and branches of the caotid sympathetic plexus of the autonomic nervous system. In the pediatric population, the optic nerve canal is bound to be small therefore having a lesser volume for the nerve to expand. Thus early visual impairment following the trauma. The neuronal degeneration hence will occur if intervention not carried out early during the course of injury.^[13-15] So we here recommend early intervention in the cases of traumatic optic neuropathy rather than waiting for spontaneous recovery. The role of surgery also has been ill defined,^[9,11,17,18] and the combined use of surgery and steroids might help around 80% cases to regain the vision. Prognostically. Several clinical signs are grave indicators for poor visual recovery. Presence of blood within the posterior ethmoidal air cells, Patient age older than 40 years, loss of consciousness associated with traumatic optic neuropathy, and failure or recovery after 48 hours of corticosteroid therapy. The presence of "no light perception" visual acuity following trauma does not currently carry any predictive value. Data on this finding have been variable in multiple clinical series.^[7-10]

Conclusion

The above said management has led to the recovery of vision in around 82% of the cases. The medical management can help around 60% of the cases and rest can be dealt with the surgical decompression of the nerve. Moreover the surgical procedure carried out is minimally invasive and is associated with a better surgical visualization, reduced hospitalization and avoids the morbidity associated with the intracranial approaches.

References

1. Coe MW, Levin LA, Joseph MP, Pinczower EF: Traumatic optic neuropathy. A meta-analysis. Arch Otolaryngol Head Neck Surg 1996 ; 122 (4) : 389 – 92.
2. Mahapatra AK, Tandon DA. Traumatic optic neuropathy in children: A prospective study. Pediatr Neurosurg 1993; 19(1): 34-9.
3. Goldenberg-Cohen N, Miller NR, Repka MX. Traumatic optic neuropathy in children and adolescents. JAAPOS 2004; 8(1):20-7.
4. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy. Surv Ophthalmol 1994; 38 (6): 487-518.
5. Rajiniganth MG, Gupta AK, Gupta A, Bapuraj JR. Traumatic optic neuropathy: visual outcome following combined therapy protocol. Arch Otolaryngol Head Neck Surg 2003; 129(11): 1203-6
6. Thakar A, Mahapatra AK, Tandon DA. Delayed optic nerve decompression for indirect optic nerve injury. Laryngoscope 2003; 113(1): 112-9.
7. Carta A, Ferrigno L, Salvo M, et al: visual prognosis after indirect traumatic optic neuropathy. J Neurol Neurosurg Psychiatry 2003 Feb; 74(2): 246-8.
8. Chuenkongkaew W, Chirapapaisan N: A prospective randomized trial of megadose methylprednisolone and high dose dexamethasone for traumatic optic neuropathy. J Med Assoc Thai 2002 May ; 85 (5): 597-603.
9. Girard BC, Bouzas EA, Lamas G, Soudant J: Visual improvement after transethmoid-sphenoid Decompression in optic nerve injuries. J Clin Neuro-ophthalmol 1992 Sep;12(3) 142-8.
10. Holmes MD, Sires BS: Flash visual evoked potentials predict visual outcome in traumatic optic neuropathy. Ophthalmol Plast Reconstr Surg 2004 Sep;20(5) : 342-6.
11. Joseph MP, Lessell S, Rizzo J, Momose KJ : Extracranial optic nerve decompression for traumatic optic neuropathy. Arch Ophthalmol 1990 Aug; 108(80): 109-3.
12. Kitthaweesin K, Yospaiboon Y: Dexamethasone and methylprednisolone in treatment of indirect traumatic optic neuropathy. J Med Assoc Thai 2001 May; 84(5) : 628-34.
13. Levin LA, Beck RW, Joseph MP, et al: The treatment of traumatic optic neuropathy: the international Optic Nerve Trauma Study. Ophthalmology 1999 Jul; 106(7): 1268-77.
14. Medeiros FA, Moura FC, Vessani RM, Susanna R Jr.; Axonal loss after traumatic optic neuropathy documented by optical coherence tomography. Am J Ophthalmol 2003 Mar; 135(3) : 406-8.
15. Medeiros FA, Susanna R: Retinal nerve fiber layer loss after traumatic optic neuropathy detected scanning laser polarimetry. Arch Ophthalmol 2001 Jun; 119(6): 920-1.
16. Ohlsson M, Westerlund U, Langmoss IA, Svensson M: Methylprednisolone treatment does not influence axonal regeneration or degeneration following optic nerve injury in the adult rat. J Neuroophthalmol 2004 Mar; 24(1) : 11-8.
17. Wang BH, Robertson BC, Giroto JA, et al: Traumatic optic neuropathy: a review of 61 patients. Plast Reconstr Surg 2001 Jun; 107(7): 1655-64.
18. Wohlrab TM, Maas S, de Carpentier JP: Surgical decompression in traumatic optic neuropathy. Acta Ophthalmol scand 2002 Jun; 80(3) : 287-93.
19. Yip CC, Chng NW, Au Eong KG, et al: Low-dose intravenous methylprednisolone or conservative treatment in the management of traumatic optic neuropathy. Eur J Ophthalmol 2002 Jul-Aug; Eur J ophthalmol 2002 Jul-Aug; 12 (4): 309-14.

Copyright: © the author(s), 2019. It is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY 4.0), which permits authors to retain ownership of the copyright for their content, and allow anyone to download, reuse, reprint, modify, distribute and/or copy the content as long as the original authors and source are cited.

How to cite this article: Raushan EA, Kumar A. Traumatic Optic Neuropath: Is this the Ideal Management Protocol? Asian J. Med. Res. 2019;8(4):EN01-EN03.

DOI: [dx.doi.org/10.21276/ajmr.2019.8.4.EN1](https://doi.org/10.21276/ajmr.2019.8.4.EN1)

Source of Support: Nil, **Conflict of Interest:** None declared.