

A PROSPECTIVE STUDY ON OUTCOME OF ENDOSCOPIC OPTIC NERVE DECOMPRESSION FOR VISUAL LOSS AFTER CRANIO - ORBITAL TRAUMA

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HOW TO CITE THIS ARTICLE:

A. Shahul Hameed, P. Muraleedharan Nampoothiri, Bincy Joseph. "A Prospective Study on Outcome of Endoscopic Optic Nerve Decompression for Visual Loss after Cranio - Orbital Trauma". Journal of Evidence Based Medicine and Healthcare; Volume 1, Issue 7, September 2014; Page: 696-702.

ABSTRACT: Optic nerve damage after cranio-orbital trauma is Traumatic Optic Neuropathy (TON). The optic nerve may be damaged directly or indirectly after cranio-orbital trauma as a result of transection of nerve fibers, interruption of blood supply, or secondary hemorrhage and edema. The injury to the optic nerve fibers by transection or infarction at the time of injury results in permanent damage whereas neural dysfunction secondary to compression within the optic canal, as a result of edema and hemorrhage, may respond to medical or surgical intervention as optic nerve decompression. Present study was conducted to assess the outcome of endoscopic optic nerve decompression for visual loss after cranio-orbital trauma. Ten cases of traumatic optic neuropathy were included in this prospective study. All these were road traffic accident cases and were referred from ophthalmology department. RAPD was in grade 2-3 in all patients. Trans nasal endoscopic optic nerve decompression was done 2-8 days after trauma. Out of ten, 7 patients with residual vision ranging from perception of light to 2m CF and 1 patient with no residual vision (total 8 out of 10, 80%), had improvement and recovered to normal vision after trans nasal endoscopic optic nerve decompression. Two patients with no residual vision showed no improvement even after optic decompression. The majority of the patients who recovered to normal vision (5 out of 8, 63%) were operated 2-4 days after trauma. Trans nasal endoscopic optic nerve decompression is an effective and safe treatment for traumatic optic neuropathy and the factors which predict good prognosis for visual recovery include a short time interval between trauma and intervention, and residual vision at presentation.

KEYWORDS: 1. Visual loss 2. Traumatic optic neuropathy 3. Endoscopic optic nerve decompression.

INTRODUCTION: Optic Nerve is the "Nerve of vision" and extends from the brain through the skull into the eye. It can be divided into 3 segments: intra orbital, intra canalicular and intracranial. A portion of the optic nerve is enclosed in rigid, bony tunnel as it exits the skull. The optic canal which carries both the optic nerve and ophthalmic artery is formed by the lesser wing of sphenoid bone. Because of this configuration, any condition which causes swelling or compression of the optic nerve at this location may lead to a loss of vision or blindness because there is no room or space for the nerve to expand. The goal of optic nerve decompression is to remove a portion of the bony optic canal, thereby relieving some of the pressure on the optic nerve.¹

Common indications for optic nerve decompression are trauma, thyroid eye disease,

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neoplastic compression e.g. meningioma, chronic inflammatory conditions like Wegner's granulomatosis and fungal sinusitis.^{2,3}

Traumatic Optic Neuropathy (TON) is the optic nerve damage after cranio-orbital trauma. It may be damaged directly or indirectly after cranio-orbital trauma. Both direct and indirect injury may damage the optic nerve as a result of transection of nerve fibers, interruption of blood supply, or secondary hemorrhage and edema. Primary injury to the optic nerve fibers by transection or infarction at the time of injury results in permanent damage. Treatment modalities are effective only for secondary mechanisms of injury. These may be at the cellular level or from intracanalicular optic nerve swelling that further compromises an injured optic nerve. Treatment consists of attempts to limit secondary injury and salvage axons that survive the initial trauma.

Optic nerve decompression has been practiced since 1916 as a treatment for several disorders which cause loss of vision.^{4,5} Frontotemporal craniotomy was the common early method for decompressing the optic nerve.⁶ In 1926 Sewall used external ethmoidectomy to approach the medial optic canal and began extracranial optic nerve decompression.⁵ Since then other forms of extracranial decompression have been suggested including the transantral-ethmoidal approach, a combined medial and lateral orbitotomy, sublabial transnasal approach, intranasal microscopic technique and lateral facial approach.^{6,7}

Recently the endoscopic endonasal technique has been widely applied in the treatment of many disorders. In 1991 Aurbach widely applied the use of this technique for decompression of the optic nerve, in the German literature⁸. Later Luxenburger et al also reported their experience with endoscopic optic nerve decompression⁹. They concluded endoscopic decompression offers many advantages over the traditional approaches including decreased morbidity, rapid recovery time and no external scars.

A study of 10 patients who underwent endoscopic optic nerve decompression for traumatic optic neuropathy and its outcome is discussed in this article.

MATERIALS AND METHODS: The present study was conducted in Department of ENT, Government Medical College, Calicut between 12/2011 to 9/2013 on 10 cases of traumatic optic neuropathy. Institutional ethical clearance was obtained for the conduct of the study.

All patients were referred from ophthalmology department.

A detailed history of onset, progress of loss of vision, laterality, history of trauma and type of trauma, loss of consciousness or seizures following trauma was elicited. Treatment history was elicited with regards to the use of steroids following trauma.

A thorough general examination was carried out to assess the general condition. Face and orbit was examined for any bony deformity or facial asymmetry. Ophthalmologic evaluation was done to assess the extra ocular movements, pupillary reflexes, visual acuity and fundus examination.

In addition to routine blood investigations, radiologic assessment in terms of CT head, HRCT orbit and Para nasal sinuses were performed. Site of fracture and compression of optic nerve was assessed with the help of HRCT.

Transnasal endoscopic optic nerve decompression under local anesthesia was done for all patients. All the cases were operated by a single surgeon. This was done 2-8 days after trauma -

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5 patients after 1 week, 3 patients after 4 days, 2 patients after 2 days. Delay in surgery was due to co-existing problems like loss of consciousness, disorientation etc. Inj. Fortwin/Pethidine + Phenergan + Atropine/ Glycopyrrolate were given for premedication. Careful mucosal preparation is essential for good avascular field and anesthesia. Nose is packed under vision with cotton pledgets soaked in 4% lignocaine and adrenaline (1: 1000), with excess solution removed. Care is taken not to injure the mucosa. The nasal pack is retained for 10-15 minutes.

Patient is positioned supine with the head end elevated about 15 degree. The surgery was performed using 0 degree 4mm nasal endoscope.

Local infiltration with 2% xylocaine was given at the upper attachment of uncinate process, anterior part of middle turbinate and at the sphenopalatine area.

Uncinectomy, middle meatal antrostomy, anterior and posterior ethmoidectomy and sphenoidotomy was done. Blood clots from ethmoid and sphenoid cavities were cleared. Lamina papyracea delineated completely till the orbital apex and the anterior face of the sphenoid is opened widely. Optic nerve is identified within the optic canal in the sphenoid sinus. The lamina papyracea is fractured 1.5 cm anterior to the optic canal. The lamina is elevated off the orbital periosteum from anterior to posterior direction to expose the annulus of Zinn. When the optic canal is reached the thin lamina is replaced by thick bone of the lesser wing of sphenoid. This bone is removed. If lamina papyracea is already fractured, the fracture fragments slowly removed without damaging the orbital apex. Following this, decompression of the medial wall of the optic canal was continued backward with punch forceps. With this technique, the optic canal was decompressed 180 degrees medially, from the optic tubercle to near the optic chiasm. The sheath of the optic nerve was incised in three cases, when there is evidence of intra sheath hematoma as bulge and discoloration. Hemostasis ensured.

Ophthalmologic evaluation done on 2nd postoperative day and patient is discharged if no complications are encountered. Patient is called for follow up after one week, 3 weeks and 6 weeks.

RESULTS: The study included 10 patients. The age group ranged from 17-35 years and all were male. Road traffic accident especially bike accident was the cause for optic neuropathy in all patients. Loss of consciousness following trauma for 1-2 days was there for 5 patients and was due to intracranial pathologies as subarachnoid hemorrhage and extradural hematoma. All had unilateral traumatic optic neuropathy, of which 7(70%) patients had right sided and 3(30%) patients had left sided visual loss. RAPD was in grade 2-3 in all patients.

Based on visual acuity, patients were divided into 3 groups: Group 1- no perception of light, Group 2 - perception of light to 2m counting fingers and Group 3 - more than 2mCF. 30% of the patients (3 patients) were in group 1, 60% (6 patients) in group 2 and 10% (1 patient) in group 3.

HRCT of orbit and Para nasal sinuses was done in all patients prior to endoscopic optic nerve decompression. Site of fracture and compression of optic nerve was assessed with the help of HRCT. 4 patients had multiple fractures involving the lesser wing and greater wing of sphenoid and lateral wall of orbit, 4 had comminuted fracture of lateral wall of sphenoid sinus and optic canal, and 2 had medial blow out fracture orbit extending to orbital apex and hematoma.

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Transnasal endoscopic optic nerve decompression was done 2-8 days after trauma.

Based on the duration between date of trauma and surgery, patients were divided into 2 groups: Group 1 - those who underwent surgery within 4 days and Group 2 - those who underwent surgery between 5-8 days. Fifty percentage (5 patients) were in group 1 and fifty percentage (5 patients) in group 2.

Regarding per operative findings, in 8 cases, bony spicules/fragments was found pressing the optic nerve and was removed. No fracture fragments were identified over the nerve in 2 patients but there was hematoma compressing the nerve which was removed.

Post-operative visual acuity was examined on day 2, 1wk, 3wks and 6wks. Patient's vision was considered to have improved if there was an increase of 3 lines or more on Snellen's visual chart or the vision had increased from non-perception of light to perception of light, perception of light to hand movements, or hand movements to counting fingers.

Out of ten, 7 patients with residual vision ranging from perception of light to 2m CF and 1 patient with no residual vision (total 8 out of 10, 80%), had improvement and recovered to normal vision after transnasal endoscopic optic nerve decompression. Two patients with no residual vision showed no improvement even after optic decompression. The majority of the patients who recovered to normal vision (5 out of 8, 63%) were operated 2-4 days after trauma. Table 1 shows the outcome of endoscopic optic nerve decompression in our study.

Age	Day of OND	Preoperative vision	Post-operative vision – second day	Post-operative vision – second week
26	7	R – No PL	No PL	No PL
29	4	R – 6/60	6/12	6/9
19	7	L - < 1m CF	1m CF	6/36
24	8	R – PL (+)	6/60	6/12
35	8	R – No PL	No PL	No PL
23	8	R – No PL	PL (+)	6/60
30	2	L – 2m CF	6/24	6/9
17	2	R - 1m CF	6/60	6/36
24	4	L - 1m CF	6/24	6/9
26	4	R - 2m CF	6/60	6/12

Table 1: Outcome of endoscopic optic nerve decompression

DISCUSSION: In this study of endoscopic optic nerve decompression we got 10 cases of traumatic optic neuropathy, all following road traffic accidents leading to indirect TON, and young males were the usual victims. Decreased visual acuity was the only morbidity associated with traumatic optic neuropathy. The only ocular abnormality that was diagnostic of optic nerve injury was the presence of Relative Afferent Pupillary Defect (RAPD), which is elicited by swinging flashlight test. In the present study, the ocular manifestations that was most commonly associated with optic nerve injury was periorbital oedema, ecchymosis and subconjunctival

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hemorrhage with no immediate changes in the optic disc. The ocular manifestations observed in our study are similar to those reported in other studies (Steinsapir KD (1997)).¹⁰

The incidence of optic canal fracture in traumatic blindness has been variously reported from 6% to 92% (Osguthorpe JD, Sofferman RA (1988)).⁴ In this study, computed tomography revealed optic canal fracture in 8 patients (80%), 4 of them had associated orbital fracture. Thus from this study the common anatomical site of optic nerve compression is optic canal. This has been described in literature by Matsuzaki et al 1982.¹¹

The rationale of optic nerve decompression involves partially removing the optic canal to decompress the nerve within the canal in order to limit the damaging effect of compression and to reestablish nerve function. (Luxenberger W, Stammberger H, 1998,⁹ Kountakis SE, 2000.¹² Fujiani et al¹³ reported a 48% improvement in a large series of patients with optic nerve decompression. Kountakis SE¹² et al reported an improvement of 82% after surgery in their series of 17 patients.

Of the 10 patients in this study, 8(80%) showed improvement after surgery.

Regarding initial visual acuity, in this study 3 patients had no perception of light at diagnosis and 7 patients had residual vision ranging from perception of light to counting fingers. Only 1 of 3 patients without perception of light showed improvement postoperatively whereas 7 of 7 (100%) patients with initial residual vision showed improvement. This means that the visual acuity at presentation is one of the factors determining the outcome of endoscopic optic nerve decompression. Patients with residual vision at presentation has got better prognosis compared to those with no light perception preoperatively. This relationship is comparable to studies conducted by Chou et al in 1996¹⁴, Mine et al (1999).¹⁵ Wang et al (2001)¹⁶ and Zuo KJ et al (2009).¹⁷

In our study, out of ten, 7 patients with residual vision ranging from perception of light to 2m CF and 1 patient with no residual vision (total 8 out of 10, 80%), had improvement and recovered to normal vision after trans nasal endoscopic optic nerve decompression. Two patients with no residual vision showed no improvement even after optic decompression. The majority of the patients who recovered to normal vision (5 out of 8, 63%) were operated 2-4 days after trauma. This highlights the importance of early intervention for better prognosis. The results will be better if optic nerve decompression is done within one week of trauma. This is comparable to the study by Li et al in 1999.¹⁸ In their study with 45 patients 71% showed improvement. In a prospective non randomized study conducted by M.G. Rajiniganth et al in 2003¹⁹ which included 44 patients with TON, the visual improvement was achieved in 31(70%) when treatment was initiated within 7 days of injury, whereas only 10 patients (24%) showed improvement when the treatment was started after more than 7 days.

But there are controversies in the treatment of TON. There is not any specific guidance how to treat and whether to treat at all. The International Optic Nerve Trauma Study was organized to help clarify the value of different treatments of TON. Within 7 days of injury, one group of patients were untreated, second group was treated with corticosteroids, and the third was treated with optic canal decompression surgery. There were no significant differences between any of the treatment groups. The conclusion of the study was that neither

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corticosteroids nor optic canal surgery should be considered the standard of care for patients with TON.²⁰

CONCLUSION: 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”, or “we can do something and also hope that the vision will improve”. Most of the people are inclined toward the second option. Trans nasal endoscopic optic nerve decompression is one of the effective and safe treatments for compressive optic neuropathy. Traumatic injury is the most common cause for compressive optic neuropathy. Decreased visual acuity is the only ocular morbidity associated with traumatic optic neuropathy. The only ocular abnormality that is diagnostic of optic nerve injury is the presence of Relative Afferent Pupillary Defect (RAPD). Fracture involving the optic canal is the common anatomic site of compression. Factors which seem to indicate good prognosis for visual recovery include a short time interval between trauma and intervention, and residual vision at presentation.

REFERENCES:

1. Pletcher SD, Metson R. Endoscopic optic nerve decompression for nontraumatic optic neuropathy Arch Otolaryngol Head Neck Surg. 2007; 133(8): 780-783.
2. Valerie J Lund, Geoffrey. Orbital and optic nerve decompression. In: Scott-Brown's Otorhinolaryngology, Head and Neck Surgery. 7th ed. Vol-2, Hodder Arnold 2008. p.1677-1687.
3. Abdullah Al-Mujaini, UpenderWali, Mazin Alkhabori. FESS indications and complications in the ophthalmic field. OMJ. (2009); 24: 70-80.
4. Osguthorpe JD, Sofferman RA. Optic nerve decompression. Otolaryngol Clin North Am 1988; 21: 155-168.
5. Sofferman RA. The recovery potential of the optic nerve. Laryngoscope 1995; 105: 1-38.
6. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy. Surv Ophthalmol 1994; 6: 487-518.
7. Knox BE, Gates GA, Berry SM. Optic nerve decompression via the lateral facial approach. Laryngoscope 1990; 100: 458-462.
8. Silberman SJ, Chow JM. Endoscopic optic nerve decompression for the treatment of traumatic optic neuropathy. In: StankiewiczJA (Ed) Advanced Endoscopic Sinus Surgery. Mosby, St. Louis, USA, 1995 pp. 115-120.
9. Luxenberger W, Stammberger H, Jebeles JA, Walch C. Endoscopic optic nerve decompression: The Graz experience. Laryngoscope1998; 108: 873-882.
10. Steinsapir KD, Goldberg RA. Traumatic optic neuropathies. In: Walsh and Hoyt's Clinical Neuro-Ophthalmology. 5th ed. Baltimore, Md: William & Wilkins; 1997: 715-739.
11. Matsuzaki H, Kunita M, Kawai K. Optic nerve damage in head trauma: clinical and experimental studies. Jpn J Ophthalmol 1982; 26: 447-461.
12. Kountakis SE, Maillard AA, El-Harazi SM, et al. Endoscopic optic nerve decompression for traumatic blindness. Otolaryngol Head Neck Surg 2000; 123: 34-37.

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13. Fujitani T, Inoue K, Takahashi T, et al. Indirect traumatic optic nerve neuropathy-visual outcome of operative and non-operative cases. *Jpn J Ophthalmol* 1986; 30: 125–134.
14. Chou PI, Sadun AA, Chen YC, Su WY, Lin SZ, Lee CC. Clinical experiences in the management of traumatic optic neuropathy. *Neuro-ophthalmology* 1996; 18: 325–336.
15. Mine S, Yamakami I, Yamaura A, et al. Outcome of traumatic optic neuropathy. Comparison between surgical and nonsurgical treatment. *Acta Neurochir* 1999; 141: 27–30.
16. Wang BH, Robertson BC, Giroto JA, et al. Traumatic optic neuropathy: a review of 61 patients. *Plast Reconstr Surg* 2001; 107: 1655–1664.
17. Zuo KJ, Shi JB, Wen WP, Chen HX, Zhang XM, Xu G. Transnasal endoscopic optic nerve decompression for traumatic optic neuropathy: analysis of 155 cases. *Zhonghua Yi Xue Za Zhi*. 2009 Feb 17; 89(6): 389-92.
18. Li KK, Teknos TN, Lai A, et al. Traumatic optic neuropathy: results in 45 consecutive surgically treated patients. *Otolaryngol Head Neck Surg* 1999b; 120: 5–11.
19. M.G.Rajiniganth, MS; Ashok K.Gupta, MS, MNAMS; Amod Gupta, MS; Jayapally Rajiv Bapuraj, MS, PDCC. TON Visual outcome following combined therapy protocol, *Arch Otolaryngol Head Neck Surg*.2003; 129: 1203-1206.
20. Levin LA, Beck RW, Joseph MP, et al. The treatment of traumatic optic neuropathy: the International Optic Nerve Trauma Study. *Ophthalmology*. 1999; 106: 1268–1277.

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Date of Submission: 06/08/2014.
Date of Peer Review: 07/08/2014.
Date of Acceptance: 26/08/2014.
Date of Publishing: 08/09/2014.