International Journal of Otorhinolaryngology and Head and Neck Surgery
Joseph B et al. Int J Otorhinolaryngol Head Neck Surg. 2022 Jan;8(1):14-20
http://www.ijorl.com
pISSN 2454-5929 | eISSN 2454-5937

DOI: https://dx.doi.org/10.18203/issn.2454-5929.ijohns20214841

Original Research Article

Endoscopic optic nerve decompression in traumatic optic neuropathy: our experience in a tertiary care centre

Bincy Joseph[1], Sapna S. Nambiar[2], K. Ramachandran[3], Suma Radhakrishnan[4]*

[1]Kerala Government Health Service, Kannur, Kerala, India
[2]Department of ENT, Government Medical College Kozhikode, Kerala, India
[3]Department of ENT, KMCT Medical College, Kozhikode, Kerala, India
[4]Department of ENT, Government Medical College Manjeri, Kerala, India

Received: 16 November 2021
Revised: 03 December 2021
Accepted: 04 December 2021

***Correspondence:**
Dr. Suma Radhakrishnan,
E-mail: drsumavijay@gmail.com

ABSTRACT

Background: Traumatic optic neuropathy (TON) a vision threatening disorder requires early diagnosis and prompt treatment. High dose steroid injections, optic nerve decompression or combined therapy are the available current treatment options. This study aims to determine the visual outcome with transnasal endoscopic optic nerve decompression in patients with TON having no improvement in vision despite high dose steroids.
Methods: A prospective study was conducted at the department of ENT, government medical college Kozhikode; on patients who presented with loss of vision following history of trauma. All patients suspected of compressive optic neuropathy received injection methyl prednisolone (30 mg/kg/day) with assessment of vision and HRCT scan. Patients with deterioration or no improvement in vision despite high steroid therapy were taken up for trans-nasal endoscopic optic nerve decompression.
Results: In our study 19 patients with TON underwent trans-nasal endoscopic optic nerve decompression. 11(57.9%) patients had improvement of vision, 7 (36.8%) patients had no improvement of vision and 1 (5.3%) patient had worsening of vision. The visual improvement was seen in 8 (80%) patients when treatment was initiated within 7 days and in only 3(33.3%) patients when treatment was initiated after 7 days. The visual acuity at presentation and time interval between trauma and intervention are factors that determine better visual outcomes.
Conclusions: The decreased visual acuity in TON requires prompt treatment. High dose steroid must be started at once when it is suspected or diagnosed. The timely surgical intervention with trans-nasal endoscopic optic nerve decompression is a relatively safe and effective technique enabling better visual prognosis.

Keywords: Trans-nasal, Endoscopic, Optic nerve decompression, TON

INTRODUCTION

Traumatic optic neuropathy (TON) management has always been a topic of debate and remains controversial even today. There is no available optimal treatment protocol as most of the published data are either retrospective or presented in case reports. Our study aims to bring out the visual outcome following trans-nasal endoscopic optic nerve decompression in an attempt to identify the optimum management strategy help preserve vision following indirect TON. 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, intracanalicular 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. Due to 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] The common indications of optic nerve decompression include TON, compressive optic neuropathy due to thyroid eye diseases, neoplasms like meningioma, chronic inflammatory diseases like Wegener's granulomatosis and invasive fungal sinusitis.[2] Optic nerve decompression has been practiced since 1916 as a treatment for several disorders which cause loss of vision.[3] Frontotemporal craniotomy was the common early method for decompressing the optic nerve.[4] In 1926 Sewall used external ethmoidectomy to approach the medial optic canal and began extracranial optic nerve decompression. Since then, other forms of extracranial decompression have been suggested including the trans-antral ethmoidal approach, a combined medial and lateral orbitotomy, sublabial trans-nasal approach, intranasal microscopic technique and lateral facial approach.[5]Recently the endoscopic endonasal technique has been widely applied in the treatment of many disorders. In 1991 Aurbach widely applied the use of the technique for decompression of the optic nerve, in the German literature. Later Luxenburger et al also reported their experience with endoscopic optic nerve decompression.[6]They concluded endoscopic decompression offers many advantages over the traditional approaches including decreased morbidity, rapid recovery time and no external scars.

Aim

The aim of the study was to determine the visual outcome of trans-nasal endoscopic optic nerve decompression in TON.

METHODS

This study is a prospective study conducted at the department of ENT, government medical college Kozhikode; a tertiary care hospital in Northern Kerala, for a period of 21 months from 01st December 2011 to 30th September 2013. The study was approved by the institutional research and ethics committee. All patients who presented or were referred to the department of ENT with loss of vision following history of trauma were included in the study. However, patients with diagnosed malignancy or other infective causes of compressive optic neuropathy were excluded from the study. All patients presented to the ENT department with TON during the study period satisfying the inclusion and exclusion criteria were included in the study. Patients with severe head injury, seriously ill or in coma were also excluded from study as they were all unfit for anaesthesia and therefore could not be taken up for the procedure.

A complete history of the onset, progress of loss of vision, laterality, history of trauma, type of trauma, loss of consciousness or seizures following trauma were elicited. A thorough examination was carried out to assess the general condition, extent of trauma with assessment of face and orbit for bony deformities and facial asymmetry. All patients underwent complete ophthalmic evaluation to assess the extraocular movements, pupillary reflexes, visual acuity and fundus examination. The relevant investigations including complete blood investigations and radiological evaluation with HRCT head, orbit and paranasal sinuses were carried out. The site of the fracture and compression of optic nerve were assessed with the help of HRCT. The patients were all treated with high dose steroids for the diagnosed TON.

All the patients received injection methyl prednisolone 1 gm IV per day for 3 days followed by oral steroids in tapering doses till the date of surgery. They were evaluated daily and an improvement in vision was assessed. In case of no improvement in vision patients were taken up for endoscopic optic nerve decompression.

The endoscopic optic nerve decompression was done under general anesthesia. Adequate mucosal preparation was carried out with nasal packing using cotton pledgets soaked in 4% lignocaine with adrenaline (1:1000). The surgery began with uncinectomy, middle meatal antrostomy, anterior and posterior ethmoidectomy and sphenoidotomy. Lamina papyracea was completely delineated till the orbital apex and anterior face of the sphenoid was widely opened. The optic nerve is then identified in the optic canal in the sphenoid sinus. The lamina papyracea is fractured 1.5cm anterior to the optic canal. The lamina is elevated off the orbital periosteum from anterior to posterior direction to expose the annulus of Zinn. At the orbital apex the thin lamina is replaced by thick bone of the lesser wing of sphenoid and this bone is removed. In cases where the lamina papyracea is already fractured, the fractured segments are slowly removed without damaging the orbital apex. Subsequently decompression of the medial wall of the optic canal is carried out. The optic canal was decompressed 180 degrees medially from the optic tubercle to near the optic chiasm. The orbital periosteum is incised in all cases from posterior to anterior direction allowing orbital fat to prolapse with an attempt made to ensure decompression. The optic nerve sheath is incised in selected cases with evidence of intra sheath hematoma. Post procedure hemostasis was ensured.

Postoperatively patients were treated with inj. dexamethasone for 2 days to reduce any post operative edema. The visual outcome is assessed in terms of visual acuity, colour vision and field of vision in the immediate post operative period, 1 week, 3 weeks and 6 weeks respectively. The ophthalmologic evaluation was done on the second postoperative day and patient discharged if no complications encountered with follow up advised at 1 week, 3 weeks and 6 weeks following the endoscopic optic nerve decompression. All analyses were done using SPSS 16. A $p<0.05$ was considered to indicate statistical

significance. All categorical data were presented as frequency and percentage. The association between the exposure variable and outcome was analyzed using the Pearson chi-square test.

RESULTS

A total of 19 patients were included in the study. The study had patients in the age group 11-58 years with a mean age of 27.26 years. All patients were males and all had unilateral TON, of which 13 (68.4%) patients had right sided and 6 (31.6%) had left sided visual loss. The cases included 17 (89.5%) road traffic accidents (RTA), one case of assault and one case of blunt trauma each. Of the road traffic accidents all were bike accidents except one wherein the patient was a pedestrian who was hit by a motorcycle. The history of loss of consciousness for a short duration was obtained in 11 (57.9%) patients.

All the patients in the study except one had sudden onset loss of vision. 11 (57.9%) patients gave history of loss of consciousness for a short period following trauma. On the basis of visual acuity patients were divided into 3 groups: Group 1: No perception of light (No PL), group 2: Perception of light to 2 meter counting fingers and group 3: more than 2 meter counting fingers.

In our study 9 (47.4%) patients were in group 1 and 10 (52.6%) patients were in group 2 respectively as shown in Table 1.

Table 1: Groups based on visual acuity.

Groups	Visual acuity	Frequency	Percentage (%)
1	No perception of light (PL)	9	47.4
2	PL to 2 m FC (finger counting)	10	52.6
3	>2 m FC	0	0

The assessment of pupillary reflexes revealed 9(47.4%) patients had grade 1 or grade 2 RAPD; 6 (31.6%) patients had grade 3 RAPD and 4 (20.1%) patients had grade 4 RAPD. The initial fundus examination revealed normal optic disc in 17 (89.5%) patients with the fundus showing temporal pallor in two patients. The 14 (73.7%) patients had periorbital edema, ecchymosis and subconjunctival hemorrhage. The extra-ocular eye movements were restricted in 10 (52.6%) patients; it was mechanical in 8 with 2 patients having multiple cranial nerve involvement.

All patients in our study received high dose steroid therapy immediately after diagnosis. The 15 patients received Inj. Methyl prednisolone 1 gm IV per day for 3 days followed by oral steroids in tapering doses till the date of surgery. 3 patients received injection methyl prednisolone 1 gm IV per day for 2 days and one patient received it only for one day as they were taken up for surgery after the period mentioned since there was worsening of the visual acuity noted in them. However, none of the patients in our study showed any improvement in visual acuity following steroid therapy. On the basis of duration between date of trauma and surgery the patients were divided into 3 groups: Group 1: Those who underwent surgery within 3 days, group 2: Those who underwent surgery between 4-7 days and group 3: Those who underwent surgery after 7 days.

In our study 5 (26.3%) patients were in group 1 and group 2 respectively and 9 (47.4%) patients in group 3 as shown in Table 2.

Table 2: Groups based on time interval between date of trauma and surgery.

Groups	Duration (Days)	Frequency	Percentage (%)
1	<3	5	26.3
2	4-7	5	26.3
3	>7	9	47.4

All our patients underwent HRCT Nose and paranasal sinuses with orbit with 17 (89.5%) patients having fractures involving the optic canal and 2 (10.5%) patients having orbital fractures without optic canal involvement (Figure 1).

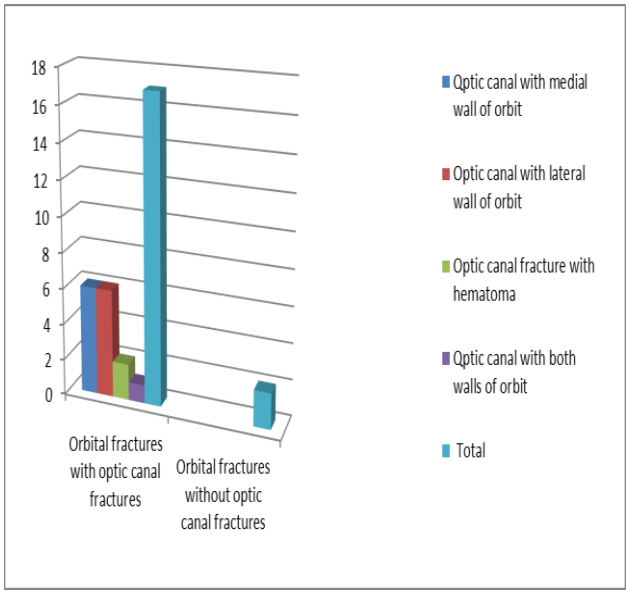

Figure 1: HRCT findings in patients.

The per operative findings confirmed 8 (42.1%) patients to have optic canal fractures with fracture fragment compressing the optic nerve, 7 (36.8%) patients had optic canal fractures associated with orbital apex hematoma compressing the optic nerve and 2 (10.5%) patients had no fracture fragments identified over the nerve but there was hematoma compressing the nerve. There were 2

(10.5%) patients in whom no source of compression was identified per operatively (Figure 2).

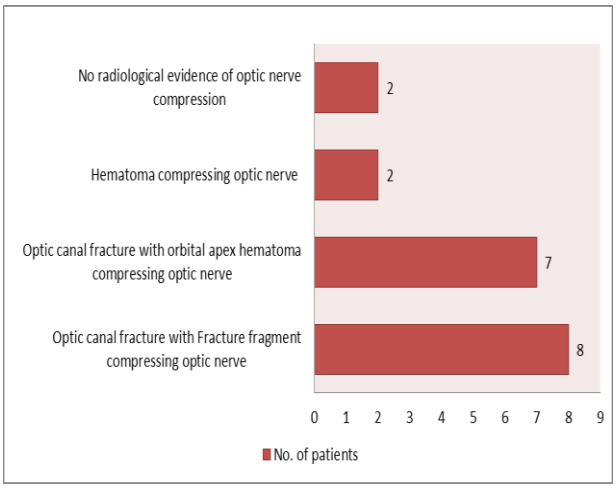

Figure 2: Per operative findings.

The post operative assessment included evaluation of visual acuity on day 2, 1 week, 3 weeks and 6 weeks respectively. The vision was considered to have improved if there was an increase of 3 lines or more on Snellan visual chart or the vision had improved from non-perception of light to perception of light, to perception of hand movements from perception of light, to counting of fingers from perception of hand movements. Of the total 19 patients who underwent endoscopic optic nerve decompression 11 (57.9%) patients had improvement of vision, 7 (36.8%) patients had no improvement of vision and 1 (5.3%) patient had worsening of vision. In all the 11 patients with improvement in vision, the vision improved within one week. The world health organization (WHO) definition of blindness considers treatment to be successful when vision is improved to at least 3/60.[7] Of the 11 patients with improvement of vision post operatively, 9 patients regained their vision to >3/60. The colour vision was regained in all 11 patients with improvement of vision. The field of vision was normal in 4 patients, 3 patients were observed to have peripheral field defect and 4 patients had defective central defect. The post operative complications identified included CSF leak in 2 patients which was identified and repaired during surgery. Both the patients had associated skull base fractures as seen in the HRCT scan taken pre-operatively. The worsening of vision despite surgery was observed in one patient, who presented with partial loss of vision, non-responsive to high dose steroids and underwent surgery on the 10th day following trauma.

The improvement in vision post endoscopic optic nerve decompression was found in 2 (22.2%) patients in group 1 with no residual vision and 9 (90%) patients in group 2 with perception of light to 2 m counting fingers (Table 3). Chi square test was done (p=0.003) which showed there is statistical significance between the initial residual vision and the outcome of surgery.

Table 3: Pre-operative visual acuity and improvement in vision.

Groups (Table 1)	Improvement in vision, (%)		Total, (%)
	Yes	No	
Group 1- No PL	2 (22.2)	7 (77.8)	9 (100)
Group 2- PL to 2 m FC	9 (90)	1 (10)	10 (100)
	11 (57.9)	8 (42.1)	19 (100)

In the case of duration between trauma and surgery 4 (80%) patients in both group 1 and 2 and 3 (33%) patients in Group 3 had improvement of vision with surgery (Table 4).

Table 4: Improvement in vision and time interval between trauma and date of surgery.

Groups (Table 2)	No. of patients	Improvement in vision (%)		Success of surgery (%)	
		Yes	No	Yes	No
Group 1	5	4 (80)	1 (20)	4	1
Group 2	5	4 (80)	1 (20)	4	1
Group 3	9	3 (33.3)	6 (66)	2 (22)	7 (78)

In our study there is no difference in outcome between group 1 and group 2 which means the vision outcome is better if optic nerve decompression is done within 1 week of trauma. Chi-square test was done comparing the duration before surgery with improvement of vision (p=0.04) which is statistically significant. The per-operative reason for visual loss was compression of the optic nerve by fracture fragment from the optic canal or hematoma. We could not identify any definite evidence of optic nerve compression in 2 cases and these patients did not show any improvement with surgery.

DISCUSSION

TON is an uncommon vision-threatening disorder that can be caused by ocular or head trauma and is categorized into direct and indirect TON. The overall incidence of TON is 0.7-2.5%, and indirect TON has a higher prevalence than direct TON.[8] The pathogenesis of indirect TON has not been fully elucidated, and the management of TON remains controversial. Currently, the main treatment options for TON are 1) systemic steroid therapy; 2) optic nerve decompression; or 3) combined treatment of steroid therapy and optic nerve decompression.[9,10] In our study endoscopic optic nerve decompression was done for 19 patients of TON mainly following road traffic accidents causing indirect TON. All the patients were young males who are the usual victims in RTA. Decreased visual acuity is the only morbidity associated with TON. Visual acuity (VA) may

range from normal to no light perception, and 40-60% of cases have light perception or worse at the time of first ophthalmic visit. In our study the ocular manifestations that were most commonly associated with optic nerve injury include periorbital edema, ecchymosis and subconjunctival hemorrhage with no immediate changes in the optic disc as reported in other studies. Clinical findings that help diagnose TON include (1) ocular injury, (2) a relative afferent pupillary defect (RAPD), (3) variable degrees of vision loss, (4) color vision disorder, and (5) different degrees of visual field defects. RAPD is a valuable finding, and in cases with mild TON, it may be the only clinical finding before overt optic nerve atrophy. The presence of RAPD is elicited by swinging flashlight test. The fact that RAPD is negative in bilaterally symmetric cases should be considered.

Patients with head or oculofacial trauma and simultaneous symptoms of optic nerve damage (unilateral or bilateral decreased VA, visual field defect, and an afferent pupillary defect on examination) should undergo urgent radiological investigations CT is the best and most accessible imaging method for detecting optic canal fractures, orbital wall fractures, and the presence of blood in the orbit.[11] It is very helpful in diagnosing direct or indirect TON and can also be used as a guide map for surgical interventions. In our study all patients had HRCT scan of the nose, paranasal sinuses and orbit taken, with optic canal fracture seen in 17(89.5%) patients; 12 of who had associated orbital fractures. The optic canal is the common anatomical site of optic nerve compression in our study as has been described in literature by Matsuzaki et al.[12]

The international optic nerve trauma study (IONTS) is the largest comparative study analyzing 133 cases of indirect TON treated with corticosteroids, surgical decompression of the optic nerve, and observation. Following adjustment for baseline VA, there were no significant differences between the three groups. Neither the dose nor the timing was associated with a higher probability of visual recovery. The improvement rates reported by other case series for steroid therapy in TON are comparable to IONTS, being around 50%.[13] Methylprednisolone therapy is advocated as the initial treatment of choice because of its neuroprotective mechanism.[14] All patients in our study received Inj. Methylprednisolone and were assessed with improvement in visual status. 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 re-establish nerve function.[6,15] Releasing the compression exerted by edema, hematoma, or fractured bone segments on the optic nerve is the rationale for surgery in TON. It can be indicated in the following situations: presence of bone segments or hematoma compressing the nerve in initial post trauma images, poor response to initial medical treatment, evidence of optic nerve damage in preoperative VEP scan, or lack of evident damage to ocular tissues and intracranial optic nerve.[16] The documented improvement after optic nerve decompression with or without steroid therapy varies from 27-82%.[13,15] Fujani et al reported a 48% improvement in a large series of patients with optic nerve decompression.[17] Kountakis et al reported an improvement of 82% after surgery in their series of 17 patients.[15] In our study 11 (58%) patients showed improvement after surgery. Statistical analysis revealed total blindness and late intervention as poor prognostic factors. In our study with regard to the initial visual acuity 9 patients had no perception of light at diagnosis and 10 patients had residual vision ranging from perception of light to counting fingers. Only 2 of the 9 (22.2%) patients without perception of light showed improvement postoperatively whereas 9 of 10 (90%) patients with initial residual vision showed improvement. This reveals 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 have got better prognosis compared to those with no light perception preoperatively. Baseline visual acuity is the main predictor of the final outcome; therefore, initially poor visual acuity is associated with limited or no visual recovery.[18] In our study visual improvement was noted in 11 of 19 (57.9%) patients, among them 8 of 10 (80%) patients in whom the surgery was done within 7 days of injury showed improvement whereas only 3 of 9 (33%) patients showed improvement when surgery was done after 7 days. 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 wherein 71% of the 45 patients showed visual improvement.[19] In the study by Rajiniganth et al 44 patients with TON were included; the visual improvement was seen in 31 (70%) when treatment was initiated within 7 days of injury, whereas only 10(24%) patients showed improvement when treatment was started after more than 7 days.[20]

The severity of trauma could be a prognostic factor as evidenced by poor prognosis in patients who presented with loss of consciousness (Carta et al) or multiple orbital fractures in CT scan (Cook et al).[21,22] In our study 75% patients without history of loss of consciousness showed improvement, but only 45.5% patients with initial loss of consciousness showed improvement.

The endoscopic approach of optic nerve decompression offers many advantages over the traditional approaches e.g., decreased morbidity, preservation of olfaction, rapid recovery time, lack of adverse cosmetic effects and less operative stress.[20] As with any surgical procedure, this approach also has some disadvantages and limitations. An iatrogenic injury to the nerve fascicle can occur during any surgical procedure that involves the optic nerve and canalicular endoscopic optic nerve decompression carries additional risks and requires the skills of an experienced endoscopic surgeon. In our study

2 patients had a CSF leak identified per operatively and treated by simultaneous skull base repair. Both these patients had associated skull base fractures as evidenced in the HRCT scan. The worsening of vision was observed in one patient who presented with partial loss of vision, non-responsive to high dose steroids and underwent surgery on the 10th day after trauma. Endoscopic optic nerve decompression is a minimally invasive procedure and has proved to be safe and cosmetically acceptable when performed by an experienced surgeon.[20]

Limitations

Main limitation of the study is the small sample size within the time frame of 21 months.

CONCLUSION

The trans-nasal endoscopic optic nerve decompression is an effective and safe surgical treatment option for TON. Traumatic injury is the most common cause of compressive optic neuropathy. Decreased visual acuity and loss of colour vision are the only ocular morbidities associated with TON. The ocular abnormality diagnostic of optic nerve injury is the presence of Relative afferent papillary defect (RAPD). High dose steroid injections (IV methylprednisolone) must be started immediately when TON is suspected or diagnosed. The common anatomic site of compression of the optic nerve is the optic canal. Timely surgical intervention could be beneficial in patients with failure of improvement of vision after 72 hours of methylprednisolone therapy; progressive visual loss despite steroid therapy; or total blindness with computed tomographic evidence of optic nerve compression. The factors which predict good prognosis for visual recovery include visual acuity at presentation and time interval between trauma and intervention. The factors predictive of worse prognosis include complete blindness at presentation, severity of injury and increase in interval between trauma and intervention if more than one week.

Recommendations

The need for an optimal treatment strategy in TON calls for a randomized, prospective, double blinded clinical trial.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Pletcher SD, Metson R. Endoscopic optic nerve decompression for nontraumatic optic neuropathy. Arch Otolaryngol Head Neck Surg. 2007;133(8):780-3.
2. Lund VJ, Geoffrey. Orbital and optic nerve decompression. In Scott-Brown's Otorhinolaryngology, Head and Neck Surgery. 7Th edi, Hodder Arnold. 2008;2:1677-8.
3. Osguthorpe JD, Sofferman RA. Optic nerve decompression. Otolaryngol Clin North Am.1988;21:155-69.
4. Steinsapir KD, Goldberg RA. Traumatic optic neuropathies. In: Walsh and Hoyt's Clinical Neuro-Ophthalmology, 5th ed. Baltimore, Md: William and Wilkins. 1997;715-39.
5. Knox BE, Gates GA, Berry SM. Optic nerve decompression via the lateral facial approach. Laryngoscope. 1990;100:458-62.
6. Luxenberger W, Stammberger H, Jebeles JA. Endoscopic optic decompression: the Graz experience. Laryngoscope. 1998;108:873-82.
7. Data on blindness throughout the world. WHO Chron. 1979;33:275-83. Available at: https://www.who.int/news-room/fact-sheets/detail/blindness-and-visual-impairment. Accessed on 3 January 2021.
8. Karimi S, Arabi A, Ansari I, Shahraki T, Safi S. A Systematic Literature Review on Traumatic Optic Neuropathy. J Ophthalmol. 2021;5553885:10.
9. Yu-Wai-Man P, Griffiths PG. Steroids for traumatic optic neuropathy. Cochrane Database Syst Rev. 2013;(6):CD006032.
10. Yu-Wai-Man P, Griffiths PG. Surgery for traumatic optic neuropathy. Cochrane Database Syst Rev. 2013;(6):CD005024.
11. Hathiram BT, Khattar VS, Sonawane HP, Watve PJ. Traumatic optic neuropathy-our experience. Indian J Otolaryngol Head Neck Surg. 2010;62(3):229-35.
12. Matsuzaki H, Kunita M, Kawai K. Optic nerve damage in head trauma: clinical and experimental studies. Jpn J Ophthalmol. 1982;26:447-61.
13. Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: the International Optic Nerve Trauma Study. Ophthalmology. 1999;106:1268-77.
14. Hall ED. The neuroprotective pharmacology of methylprednisolone. J Neurosurg. 1992;76:13-22.
15. Kountakis SE, Maillard AA, El-Harizi SM, Longhini L, Urso RG. Endoscopic optic nerve decompression for traumatic blindness. Otolaryngol Head Neck Surg. 2000;123:34-7.
16. Oh HJ, Yeo DJ, Hwang SC. Surgical treatment for traumatic optic neuropathy. Kor J Neurotrauma. 2018;14(2):55-60.
17. Fujitani T, Inoue K, Takahashi T. Indirect traumatic optic nerve neuropathy- visual outcome of operative and non-operative cases. Jpn J Ophthalmol. 1986;30:125-34.
18. Yan W, Chen Y, Qian Z. Incidence of optic canal fracture in the traumatic optic neuropathy and its effect on the visual outcome. Bri J Ophthalmol. 2017;101(3):261-7.
19. Li KK, Teknos TN, Lai A. Traumatic optic neuropathy: results in 45 consecutive surgically

treated patients. Otolaryngol Head Neck Surg. 1999;120:5-11.
20. 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):12036.
21. Carta A, Ferrigno L, Salvo M, Bianchi-Marzoli S, Boschi A, Carta F. Visual prognosis after indirect traumatic optic neuropathy. J Neurol Neurosurg Psychiatry. 2003;74:246-8.
22. Cook MW, Levin LA, Joseph MP, Pinczower EF. Traumatic optic neuropathy. A meta-analysis. Arch Otolaryngol Head Neck Surg. 1996;122:389-92.

Cite this article as: Joseph B, Nambiar SS, Ramachandran K, Radhakrishnan S. Endoscopic optic nerve decompression in traumatic optic neuropathy: our experience in a tertiary care centre. Int J Otorhinolaryngol Head Neck Surg 2022;8:14-20.