Case Series

Our Experience in Endoscopic Transnasal Optic Nerve Decompression

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ABSTRACT

Introduction

Traumatic optic neuropathy (TON), a severe complication following head injury, affects about 0.5% to 5% of patients with closed head injuries. Visual acuity (VA) loss associated with TON can be partial or complete and temporary or permanent. Increased intracanalicular pressure after an injury is considered to be one of the main pathophysiological manifestations. Mucoceles involving the Onodi cell and the sphenoidal sinus are rare, and optic nerve compression with decreased visual acuity may be the first presenting symptom. Endonasal endoscopic optic nerve decompression is a safe and highly effective treatment to reduce the hydrostatic pressure on the optic nerve in cases of optic neuropathies of various etiologies. This is to share our experience on transnasal endoscopic optic nerve decompression done for optic neuropathy due to traumatic optic neuropathy and non-traumatic optic neuropathy.

Case Series

A case series of three patients with traumatic optic neuropathy and one patient with non-traumatic optic neuropathy over a period of two years were studied. All four patients underwent endoscopic transnasal optic nerve decompression and surgical outcome in terms of visual recovery was observed.

Discussion

Endoscopic transnasal optic nerve decompression is a minimally invasive , safe and efficient method of treatment for traumatic and non-traumatic optic neuropathy. Our experience also suggest that surgical intervention need to be considered as early as possible especially for traumatic cases to enhance visual recovery.

Keywords

Optic Nerve Decompression; Traumatic Optic Neuropathy; Optic Nerve

The optic nerve can be divided into four segments: intraocular, intraorbital, intracanalicular, and intracranial.[4] The main purpose of optic nerve decompression is to relieve the compression forces within the intracanalicular portion of the optic nerve.[1,4] This area has been postulated to be the most susceptible site for pathologic compression of the optic nerve. Traumatic optic neuropathy (TON), a severe complication following head injury, affects about 0.5% to 5% of patients with closed head injuries.[1] Visual acuity (VA) loss associated with TON can be partial or complete and temporary or permanent. Increased intracanalicular pressure after an injury is considered to be one of the main pathophysiological manifestations.[2] Mucoceles involving the Onodi cell and the sphenoidal sinus are rare, and optic nerve compression with decreased visual acuity may be the first presenting symptom.[3]

For decades, treatment of traumatic and non traumatic optic nerve neuropathy was conservative and involved the intravenous application of high dose of corticosteroids.[4] The commonly used approaches for surgical decompression of optic nerve are transcranial and transnasal approaches.[5] Transcranial approach provides

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better surgical view and exposure but more complications in the form of brain retraction injuries.[5] Endonasal endoscopic optic nerve decompression is a safe and highly effective treatment to reduce the hydrostatic pressure on the optic nerve in cases of optic neuropathies of various etiologies.[6]

Case Series

Case details of three patients with traumatic optic neuropathy and one patient with non-traumatic optic neuropathy over a period of two years were studied. The four patients underwent endoscopic transnasal optic nerve decompression and surgical outcome in terms of visual recovery were observed.

Out of the four cases (see Table I), the first two cases presented with reduced vision in right eye for one week following head injury.

The 3rd case also had a similar presentation and visual and radiological finding presented within 24 hours.

The 4th case presented with progressive loss of vision in right eye for 1 month. It is a case of right compressive optic neuropathy.

Case 1:

A 38 year old male patient, presented with complaints of diminished vision in the right eye since one week, following head injury in RTA. Patient had a history of vomiting and loss of consciousness. Opthalmological examination revealed no perception of light in the right eye with Relative afferent pupillary defect (RAPD) with normal fundus. No ear/nose/throat abnormalities detected. CT scan showed fracture along the course of the right optic canal over the roof of the sphenoid sinus with fracture fragment impinging over the optic nerve (Fig.1). Trans endoscopic trasnsphenoethmoidal approach for optic nerve decompression was done under general anesthesia. Patient had no visual improvement after surgery.

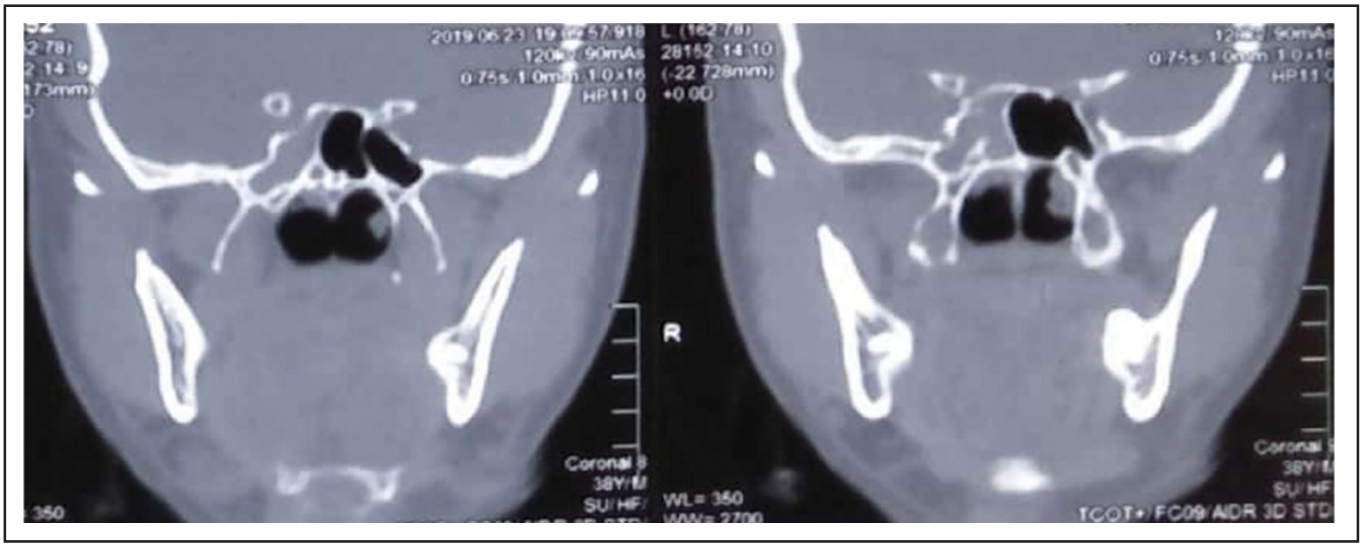

Fig. 1. CT scan Coronal View shows fracture over the roof of the sphenoid sinus with fracture fragment impinging over the optic nerve.

Case 2:

A 19 year old male patient, presented with complaints of diminished vision in the right eye following RTA one week back. He had history of loss of consciousness. On examination, the patient had no perception of light in the right eye with RAPD with normal fundus. No ear/nose/throat abnormalities detected. CT scan showed fracture along the course of the right optic canal with fracture

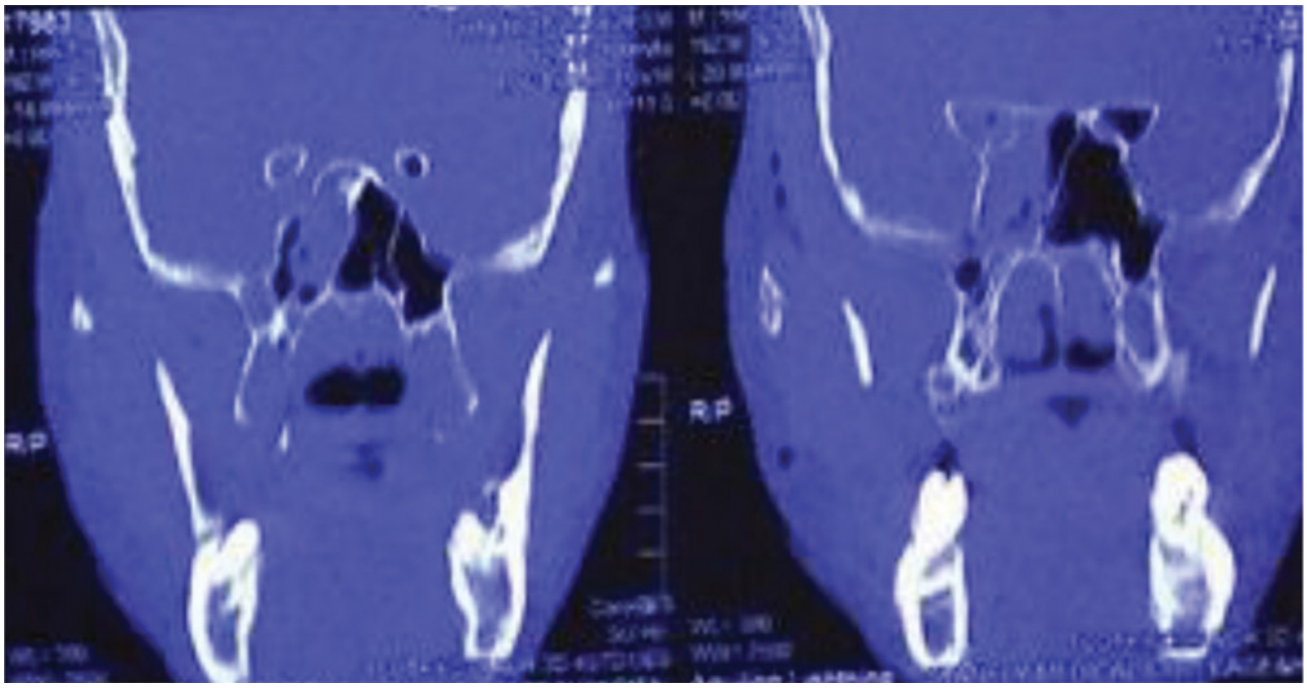

Fig. 2. CT scan coronal view shows fracture along the course of the right optic canal with fracture fragment impinging over the optic nerve

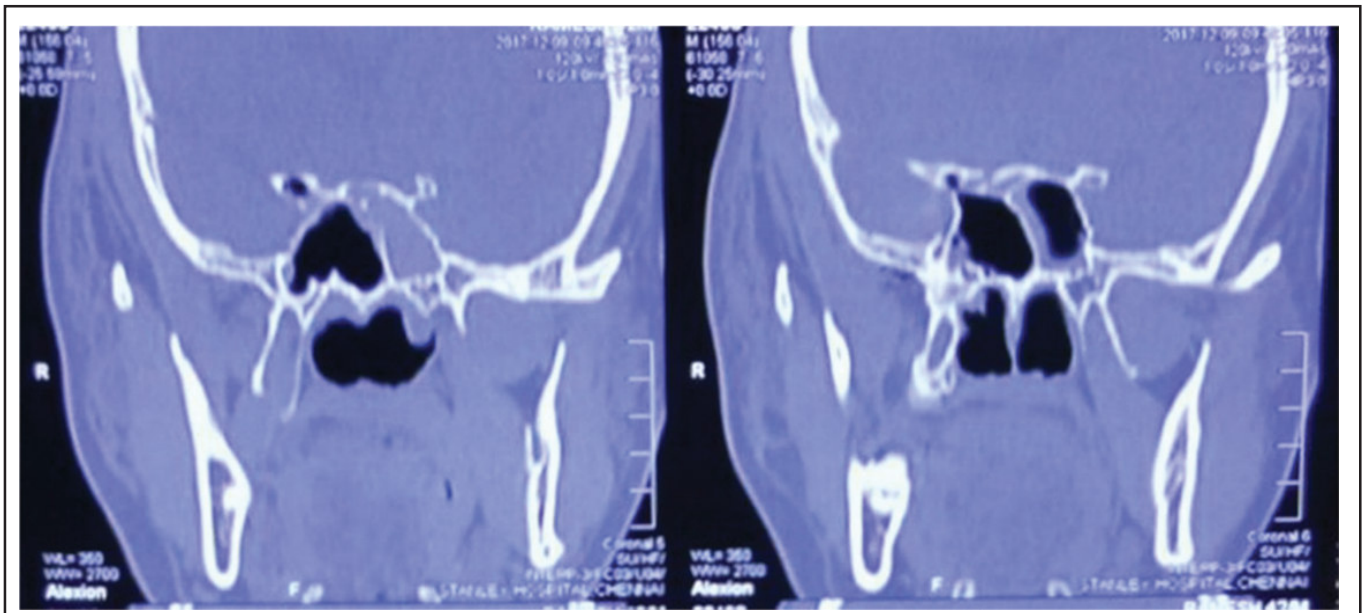

Fig. 3. CT scan coronal view shows fracture in the roof of right sphenoid sinus.

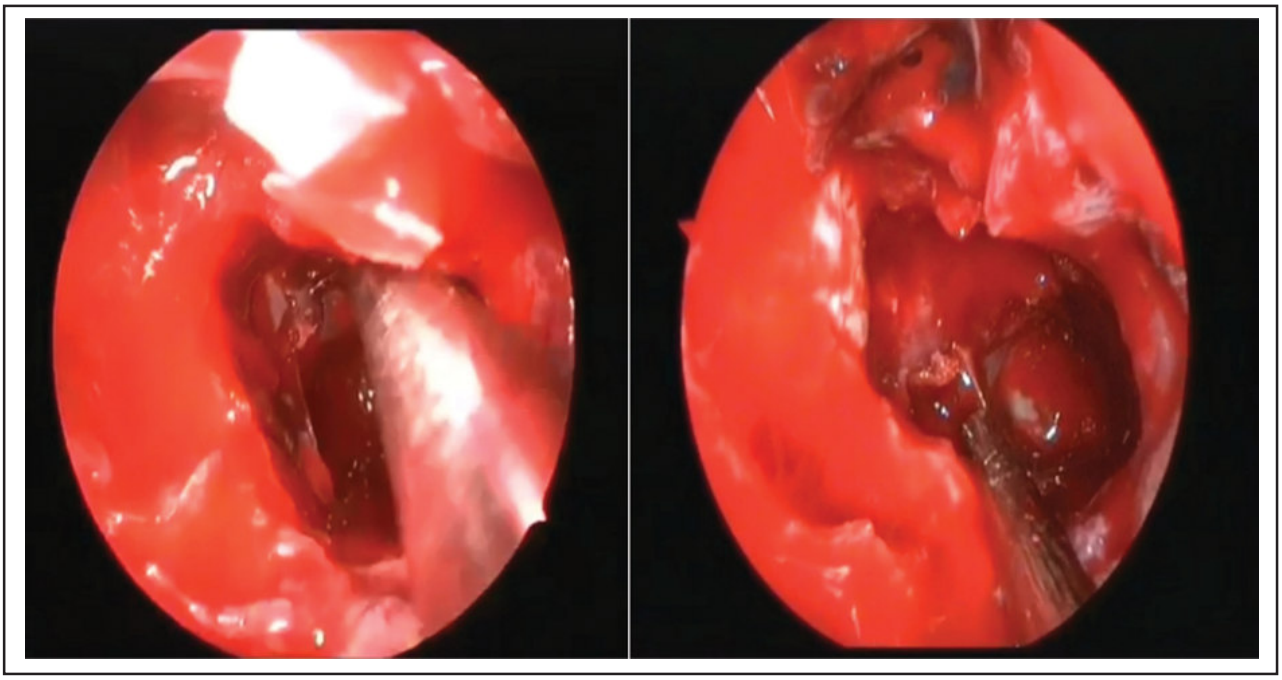

Fig. 4. Intra operative image shows fractured bone over optic canal and decompression of optic nerve.

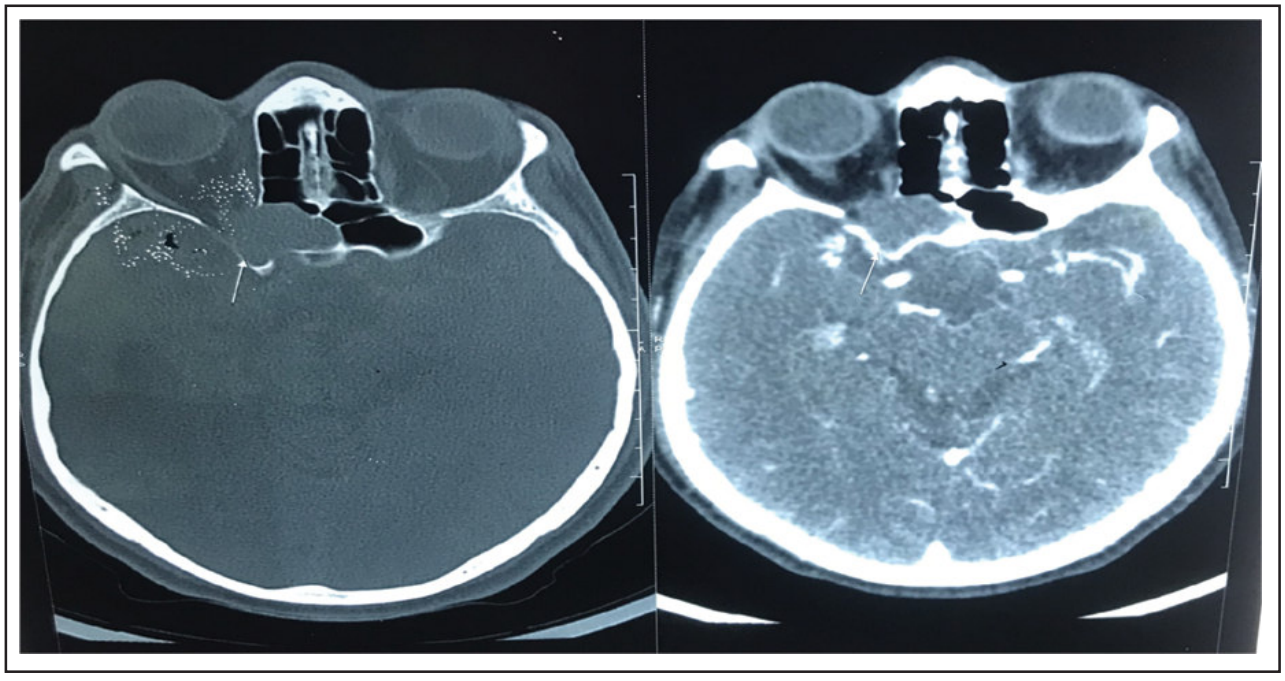

Fig. 5. CT scan axial view shows completely opacified expanded right sided onodi cell with rarefaction of the sinus which extends into pneumatized right anterior clinoid process and encasing the right optic canal which is suggestive of mucocele compressing the right optic canal

fragment impinging over the optic nerve (Fig. 2). Transnasal endoscopic sphenoethmoidal approach for optic nerve decompression was done. After surgery also patient had no visual improvement.

Case 3:

42 year old male patient, presented with diminished vision in left eye following RTA with head injury. Patient had a history of left nasal bleeding 1 episode, History of vomiting and Loss of consciousness. No ear/throat abnormalities detected. Examination of face revealed bilateral periorbital ecchymosis with chemosis and subconjunctival haemorrhage. There was no perception of light in the right eye with RAPD and normal fundus. CT scan coronal view showed fracture in the roof of right sphenoid sinus (Fig. 3). Intraoperative image shows fractured bone over the optic canal and decompression of optic nerve has been done (Fig. 4).

Case 4:

57 year old male patient presented with progressive loss of vision in right eye for the last 1 month, leading to complete loss of vision since 15 days. No history of trauma noted. Visual examination revealed only perception of light in right eye and RAPD with full range of extraocular movements. CT scan shows completely opacified expanded right sided onodi cell with rarefaction of the sinus wall which extends into pneumatized right anterior clinoid process and encasing the right optic canal which is suggestive of mucocele compressing the right optic canal (Fig. 5). After transendoscopic transs-phenoethmoido optic nerve decompression patient had a good visual recovery.

Trans-nasal endoscopic sphenoethmoidal approach for optic nerve decompression was done for all the cases. (Fig. 5) .

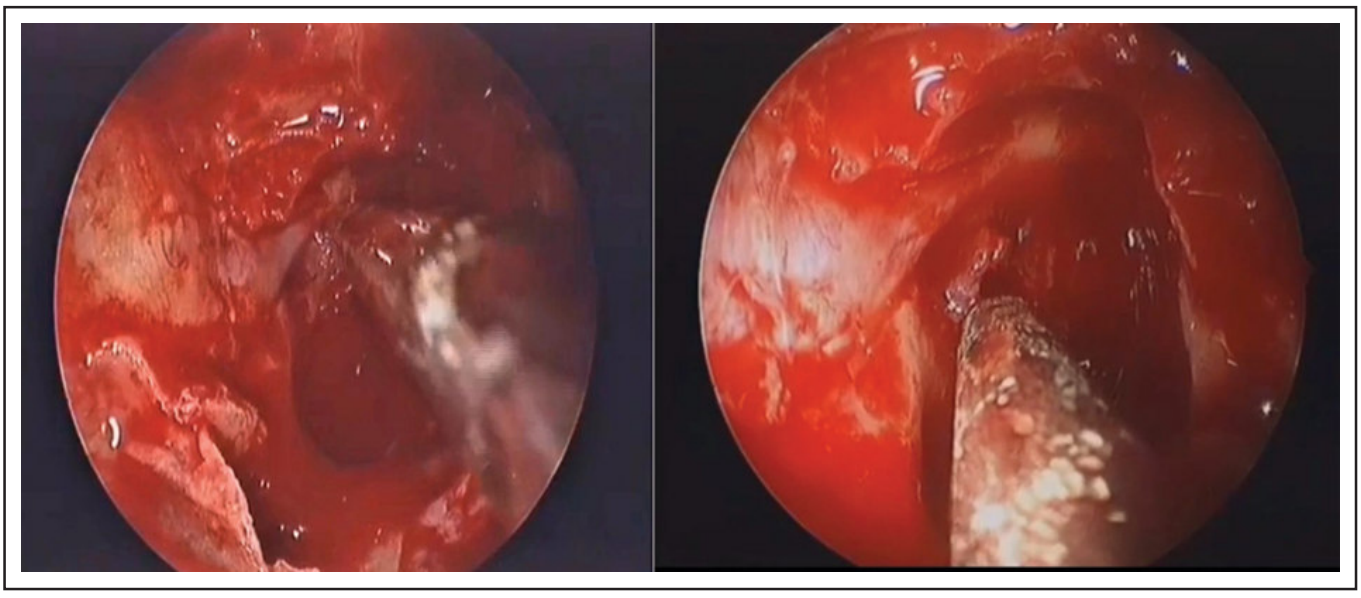

Fig. 5. Intra operative images showing optic canal before and after optic nerve decompression.

Results

Results were evaluated on the basis of data collected regarding mode of trauma, time of surgery, radiological evidence and visual acuity improvement post surgery. Out of 4 patients, 3 patients had traumatic optic neuropathy and one patient had compressive mucocele associated optic neuropathy (see Table I.)

Table I : Pre-operative and Post-operative comparison of visual acquity

Case	Diagnosis	Duration of symptom onset	Onset of vision loss	Presence of RAPD	Vision at presentation	Fundus	Time of intervention	Outcome
1	Right traumatic ON	1 week	Sudden	+	R eye- No PL	Normal	1 week	R eye- No PL
2	Right traumatic ON	1 week	Sudden	+	R eye- No PL	Normal	1 week	R eye- No PL
3	Right traumatic ON	24 hours	Sudden	+	R eye- No PL	Normal	24 hours	R eye- complete visual recovery
4	Right compressive ON	1 month	Gradual, progressive	+	R eye-PL+	Pale	1 month	R eye- complete visual recovery.

Discussion

Traumatic optic neuropathy (TON), a severe complication following head injury, affects about 0.5% to 5% of patients with closed head injuries.[1] TON can be caused by direct or indirect injury.[7] In our cases of TON it has been caused by indirect injury due to head trauma.

In our case of CON it has been caused by an onodi cell-related mucocele. It is an extremely rare complication with only a few reports in the literature.The onodi cell is the most posterior ethmoid air cell that is closely associated to optic nerve.[8] The pathogenesis is thought to be that of direct mechanical compression from the enlarging mucocele causing ischaemia resulting in an optic neuropathy. Surgery should be considered in all patients presenting with optic neuropathy secondary to an Onodi cell mucocele.

Trans nasal endoscopic sphenoethmoidal approach for optic nerve decompression was done for all the patients. After routine opening of ethmoid and sphenoid sinus, optic canal was visualised by bulge on lateral wall of sphenoid sinus and then followed anteriorly. Optic canal was then decompressed by removing the bony canal of optic nerve medially and exposing the nerve along its intracanalicular route. In all the cases optic nerve sheath was not incised and kept intact. Posterior limit of bone removal is where the nerve turns in transverse direction near optic chiasma.

Post operatively patients were managed by intravenous steroids with tapering dose up to 3 weeks, higher antibiotics, nasal douching and Saline spray were advised to prevent nasal crusting. Regular follow up every week till 1 month and then monthly till 6 months. In spite of the successful decompression of optic nerve in all 3 cases of TON, improvement in visual acuity was obtained only in the patients who presented earlier to the hospital. In the case of CON patient got improvement in visual acuity with successful optic nerve decompression even though the patient presented late to the hospital.

Conclusion

Endoscopic optic nerve decompression is a minimally invasive, safe and efficient method of treatment for traumatic and non-traumatic optic neuropathy. Our experience also suggests that surgical intervention needs to be considered as early as possible especially for traumatic cases to enhance visual recovery.

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