Transnasal Endoscopic Optic Nerve Decompression in Post Traumatic Optic Neuropathy

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Abstract To quantify the successful outcome in patients following optic nerve decompression in post traumatic unilateral optic neuropathy in form of improvement in visual acuity. A prospective study was carried out over a period of 5 years (January 2011 to June 2016) at civil hospital Ahmedabad. Total 20 patients were selected with optic neuropathy including patients with direct and indirect trauma to unilateral optic nerve, not responding to conservative management, leading to optic neuropathy and subsequent impairment in vision and blindness. Decompression was done via Transnasal-Ethmo-sphenoidal route and outcome was assessed in form of post-operative visual acuity improvement at 1 month, 6 months and 1 year follow up. After surgical decompression complete recovery of visual acuity was achieved in 16 (80%) patients and partial recovery in 4 (20%). Endoscopic transnasal approach is beneficial in traumatic optic neuropathy not responding to steroid therapy and can prevent permanent disability if earlier intervention is done prior to irreversible damage to the nerve. Endoscopic optic nerve surgery can decompress the traumatic and oedematous optic nerve with proper exposure of orbital apex and optic canal without any major intracranial, intraorbital and transnasal complications.

Keywords Optic nerve decompression · Transnasal · Post traumatic · Optic neuropathy · Impairment in vision

Introduction

Traumatic optic neuropathy is a severe damaging and disabling complication of closed head trauma. The hallmark of traumatic optic neuropathy is loss of visual function, which can manifest as subnormal visual acuity, visual field loss, or colour vision dysfunction. Visual acuity loss associated with traumatic optic neuropathy can be partial or complete and temporary or permanent. Traumatic optic neuropathy is seen in 0.5–5% of patients presenting with closed head trauma and 25% of patients presenting with midfacial fractures.

The optic nerve per se can be divided into four segments: intraocular, intraorbital, intracanalicular, and intracranial. The main purpose of optic nerve decompression is to relieve the compression forces within the intracanalicular portion of the optic nerve. The optic nerve canal is formed by two struts of the lesser wing of sphenoid. This canal carries the optic nerve and ophthalmic artery. Within the optic canal the nerve is ensheathed by three meningeal layers. At the orbital apex is the fibrous annulus of Zinn. This thick fibrous layer is the least expandable portion of the fibrous tissue around the optic nerve. This area has been postulated to be the most susceptible site for pathologic compression of the optic nerve [1].

The commonly used approaches for surgical decompression of optic nerve are transcranial and transnasal approaches. Transcranial approach provides better surgical view and exposure but more complications in the form of brain retraction injuries. Transnasal approach provides minimal invasion and complications but less exposure of the trauma site.

The aim of this paper is to assess the successful outcome following optic nerve decompression in post traumatic

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unilateral optic neuropathy in the form of improvement in visual acuity, to correlate between clinical and radiological findings in selecting patients who will be benefitted by surgery, to access and quantify improvement status in post surgical optic nerve decompression and to access the role of proper time selection of surgery on outcome.

Methods

A prospective case study was done on a total of 20 patients treated surgically for post traumatic optic nerve decompression. Data was analyzed on the basis of type of injury, cause of optic neuropathy and prognostic factors. This study includes patients presenting with unilateral optic neuropathy with direct or indirect trauma to optic nerve following trauma, patients not responding to conventional therapy of steroids and Patients presenting with progressive course. Patients who presented with optic nerve avulsion, injury to optic nerve in its intracranial course and old age patients who suffered from significant visual loss prior to trauma were not included in this study. All the patients selected for the study underwent trail of 3 days intravenous steroid (methylprednisolone 1 mg 8 hourly) followed by surgery.

Diagnosis was made on clinical basis as evidenced by optic neuropathy following a history of a blunt or a penetrating trauma. Various clinical tests are done such as Visual acuity testing, pupillary evaluation, EOM motility, Colour vision, Visual fields, and fundus examination. Radiologically patients underwent CT scan (Figs. 1, 2, 3).

We have used mainly transsphenoidal route for the decompression. Transnasal endoscopic transsphenoidal approach for optic nerve decompression was done under general anaesthesia. After routine opening of ethmoid and sphenoid sinus, optic canal is recognised and visualised by bulge on lateral wall of sphenoid sinus and

Fig. 1 Intraoperative picture showing decompressed optic nerve

Fig. 2 Impact of type of trauma on patient outcome

Fig. 3 Time interval between trauma and surgical intervention

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. Posterior limit of bone removal is when nerve turns in transverse direction near optic chiasma.

Management protocol included intravenous corticosteroids (methylprednisolone 1 g OD for 3 days) if visual acuity improves then shift to oral steroids and no surgical intervention is required, but if visual acuity does not improves then patient is taken for surgical decompression of optic nerve. Criteria for patient selection for surgery was mainly based on Radiologically evident bony fractured fragment impinging on optic nerve in the intracanalicular portion, or optic nerve sheath and/or failure to improve or deterioration of vision after 48 h of mega dose steroid therapy.

Post operatively patients were managed by Oral steroids in tapering dose up to 3 weeks, higher antibiotics, nasal douching and Saline spray to prevent nasal crusting and regular follow up every week till 1 month and then monthly till 6 months.

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 20 patients, 15 suffered from indirect trauma and 5 patients suffered from direct trauma. Out of 15 patients suffering from indirect trauma, 13 were benefitted and out of 5 patients suffering from direct trauma 3 were benefitted.

The major cause of optic neuropathy was diagnosed to be fracture of optic canal followed by haematoma, contusion and ischemia. The patients who presented with fractures were 11/20 and all of them were benefitted in terms of improvement in visual acuity.

Discussion

TON is an important cause of severe visual disability following blunt or penetrating head trauma. Classification of optic neuropathy can be on the basis of type of trauma. It can either be direct or indirect. Direct traumatic optic nerve injury occurs when the optic nerve is impinged, crushed, or transected. These injuries usually occur after open craniofacial trauma, such as penetrating wounds (from knives, pellets etc.) or extensive crush injury with displaced cranio-orbital fractures. Indirect trauma occurs most commonly after blunt trauma to superior orbital rim, lateral orbital wall or frontal area of the bony skull. Compression force from blunt trauma is transmitted to orbital apex and optic canal through orbital bone which results in oedema and ischemia of the optic nerve within the canal. Based on site of injury trauma can occur intra- orbital, intracanalicular or intracranial.

Classification of optic neuropathy [2]

- GRADE 1: No fracture, vision better than 6/60 on Snellen's chart.
- GRADE 2: No fracture, vision better than PL +ve but less than 6/60.
- GRADE 3: Fracture present or PL –ve.
- GRADE 4: Fracture present and PL –ve.

Patients with craniofacial trauma, cranio-orbital fractures or with direct trauma to orbit should elicit a high index of suspicion for traumatic optic neuropathy. Patients with traumatic optic neuropathy have serious and obvious craniofacial, neurosurgical, and other co morbidities but few cases were also there where there were no visible signs of injury. In addition, although 40–60% of patients with traumatic optic neuropathy present with a visual acuity of light perception or no light perception, nearly 20% of patients have a visual acuity of 6/60 or better. CT is better than MRI at delineating bony defects. It also acts as Intraoperative road map for the surgery. Fractures within the optic canal can be best located with thin-section CT scanning (1 mm cuts with 1-mm interval).

There are mainly two surgical endoscopic approach transsphenoidal and transsphenoid.

Anatomically, the optic canal is just superolateral to the sphenoid sinus as a part of the lesser sphenoid wing. The canal is wide with thin walls proximally toward the optic chiasm, and narrow with thick walls distally at the optic tubercle. The optic tubercle is the thick bulge of the medial aspect of the bone surrounding the optic foramen. Depending on the degree of pneumatization or the presence of supersphenoidal (Onodi) cellulae, the optic tubercle can be visualized within the sphenoid sinus or at the junction of the sphenoid and posterior ethmoid sinuses. In the case of high sphenoidal pneumatization, the use of an angled endoscope allows clear visualisation of the lateral sphenoidal wall and the orbit apex. This raises the possibility that optic nerve decompression can be performed through a single trans-sphenoidal approach rather than using complete ethmoidectomy.

Treatment options include steroids, surgical decompression, or both, according to patient selection. The use of high-dose steroids after optic nerve injury became more common during the 1980s, following the rationale that steroids may reduce post-traumatic oedema, contusion necrosis, and vasospasm, and thus aid functional recovery [3]. Previous studies have reported improvement in between 44 and 82% of patients following steroid therapy. However, in other studies, 57% of untreated patients showed improvement in visual acuity, indicating spontaneous remission [4]. The indications for endoscopic optic nerve decompression are also variable and differ according to doctor's preference. It has been observed that surgery should be performed after steroid trial as maximum patient tends to recover after medical therapy. Some authors suggested that endoscopic optic nerve decompression be performed if vision fails to improve after 72 h of methylprednisolone therapy; or if progressive visual loss during steroid therapy is observed [4]. However if there is radiologically evident compression of optic nerve then surgery may be the first line of management as it prevents any permanent damage to the nerve due to trauma. So despite of many studies we still fail to conclude the best management for post traumatic optic neuropathy.

The study that we conducted suggested:

- The patients who were operated within 72 h were most benefitted.
- Fracture of optic canal and haematoma resulting in optic neuropathy respond best to decompression.
- There was a 80% success rate in patients treated with optic nerve decompression
- The patients suffering from direct trauma to optic nerve have 27% less chances for recovery.

- The patients presenting with fracture of optic canal or orbit have more 40% more chances to be benefitted by surgery.
- The golden time for surgery is between 48 and 72 h as it prevents permanent damage to the nerve due to ischemia and oedema.
- Patients presenting with blunt trauma and mid facial injury tend to respond better with surgery.

Compliance with Ethical Standards

Conflict of interest The authors declares that they have no conflicts of interest.

Informed Consent Informed consent was obtained from all individual participants included in the study.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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