Endoscopic Transnasal Optic nerve Decompression

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Introduction:

The optic nerve is the second cranial nerve and is the nerve of vision. It is about 5 cm long and is divided into three segments. About 3 cm of the nerve is in the orbit and is protected by orbital fat around it. 1 cm of the nerve is enclosed in a bony canal on the lateral wall of the sphenoid sinus. Another 1 cm of the nerve lie intracranial or within the brain cavity. Vision loss may occur from compression of the nerve from injury (due to hematoma), mucocele of sphenoid sinus or the posterior ethmoids.
Endoscopic optic nerve decompression can be performed with very little or nil morbidity. This procedure is mainly done for traumatic optic neuropathy. This procedure can be performed under General anesthesia. There is virtually no scar and the duration of hospital stay is not more than a couple of days.

History:

Hippocrates was the first to note the association of trauma just above the eyebrow and gradual vision loss. During the 18th century the association between frontal trauma and loss of vision without evidence of ocular injury was very well appreciated. It was Battle in 1890 who distinguished the difference between penetrating direct injury from non penetrating indirect optic nerve injuries. 20th century saw significant progress in classification, pathophysiology and management of optic nerve injuries.

Historically three treatment modalities have been advocated for traumatic optic neuropathy. They include observation, medical corticosteroid therapy and optic nerve decompression. During early 1900's transcanal deroofing of the optic canal was widely practised for traumatic optic neuropathy treatment. It was in 1920 Sewell performed a transethmoidal optic canal decompressikon by removing lamina papyracea and medial wall of optic canal. Recent advances in endoscopic instrumentation and intranasal sinus surgical techniques have refined the entire process of optic nerve decompression. Currently intranasal transethmoidal transphenoidal endoscopic approach is gaining popularity.

Indications:

1. Traumatic optic neuropathy

2. Skull base tumors involving optic nerve

3. Firbo-osseus lesions of skull base encroaching on to the optic nerve canal

4. Graves ophthalmopathy associated with optic neuropathy

5. Idiopathic intracranial hypertension

Contraindications:

1. Complete disruption of optic nerve or chiasma

2. Complete atrophy of the nerve

3. Carotid cavrnous fistula

Surgical anatomy:

Optic canal:

The optic nerve enters the optic canal at the superomedial corner of the orbital apex. This canal is about 10 mm long. It contains the optic nerve, ophthalmic artery and sympathetic plexus.

Lateral – Optic canal is seperated from the superior orbital fissure by a bony ridge known as the optic strut.

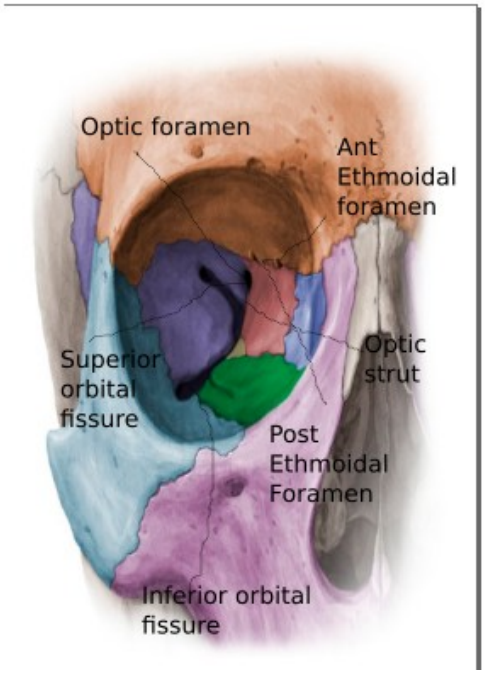
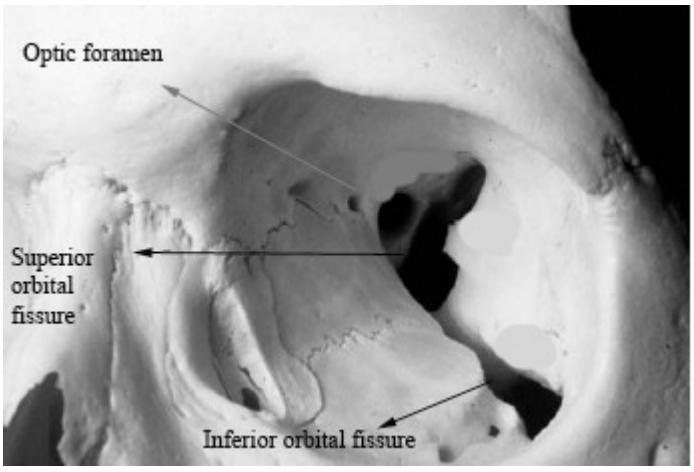


Image showing orbital anatomy

The tendon to which the extraocular muscles are attached (Annulus of Zinn) is attached to the upper, medial and lower margins of the optic canal from 12 – 6 O clock. The extraocular muscles that insert into the Annulus of Zinn include superior, medial, inferior and lateral rectus muscles.

The optic canal courses in a posteromedial direction. The walls of the optic canal is formed by the body of sphenoid, and the lesser wing of sphenoid. At the intracranial end it is shielded laterally by the anterior clinoid process. The lateral wall of the optic canal is formed by the optic strut that blends superolaterally into the anterior clinoid process. The superior wall is formed by the anterior root of the lesser wing of sphenoid bone; the medial wall is formed by the sphenoid bone.



Close up view of orbit showing the optic foramen

Ophthalmic artery:

This artery arises from the medial aspect of the anterior loop of the internal carotid artery just above the cavernous sinus. Intracranially it is located medially and below the optic nerve. In the optic canal the artery passes within the optic nerve

sheath and below the optic nerve. There are variations of the position of the ophthalmic artery within the canal. It can be found anywhere between 3 and 6 O' clock so caution must be exercised when incising the optic nerve sheath.

Optic nerve:

The optic nerve exits the orbit via the optic canal and courses posteromedially to the optic chiasma where there is a partial decussation of its fibers from the temporal visual fields of both eyes. Optic nerve unlike peripheral nerves is ensheathed in all three meningeal layers. The reason behind this unique feature is that t he optic nerves are part of central nervous system as they are outpouchings of the diencephalon during embryonic development. This nerve hence is not capable of regeneration. Damage to optic nerve produces irreversible blindness.

Traumatic optic neuropathy:

This is a condition in which acute injury to the optic nerve from direct / indirect trauma results in vision loss. The most common cause of traumatic optic neuropathy is indirect injury to the optic nerve. This is thought to be the result of transmitted shock from the orbital impact to the intracanalicular portion of the optic nerve. Direct traumatic optic neuropathy results from penetrating injury or from bony fragments in the optic canal piercing the optic nerve. Sometimes orbital hemorrhage and optic nerve sheath hematoma can also cause optic neuropathy by direct compression.

Classification of traumatic optic neuropathy:

Traumatic optic neuropathy can be classified according to the location of injury.

1. Head of the optic nerve

2. Intraorbital segment

3. Intracanalicular segment
4. Intracranial segment

The most common sites of indirect traumatic optic neuropathy are the intracanalicular segment (since the nerve is adherent to the periosteum) and the intracranial segment.

The nerve can also be compressed at the level of intracranial segment and the optic chiasma. This is usually cauesd by tumors like meningioma and pituitary adenomas. Decompression of these segments require transplanum and transsellar approaches.

Clinical features of traumatic optic neuropathy:

1. Vision loss after blunt / penetrating trauma

2. Slit lamp examination and fundus examination are normal

3. Defects in color vision

4. Defects in visual fields

Pupillary reaction:

An afferent pupillary defect is a necessary finding in these patients. Normally light shone in one eye causes equal pupillary constriction on both sides. In patients with afferent pupillary defect, light in the affected eye causes only mild construction of pupils, while light in the unaffected eye cause normal constriction on both sides.

Symptoms include:

1. Blurry vision

2. Scotomas

3. Visual field defects

4. Decreased color vision

Diagnosis of traumatic optic neuropathy is purely clinical. CT can be performed to visualize the optic nerve as well as the optic canal. Optic canal should be clearly evaluated for evidence of fracture.

Automatic visual field testing (Humphrey visual field testing) can be used to document visual field defects.

Visual evoked potential can be used to document the electrical activity of the optic nerve.

Pathophysiology of optic neuropathy:

It is rather poorly understood. Some of the accepted facts include:

1. Optic nerve avulsion

2. Optic nerve sheath hematoma

3. Penetrating FB or bony fracture

Traumatic optic neuropathy is an indirect event that occurs shortly after or during blunt trauma to the superior orbital rim, lateral orbital rim, frontal area or the cranium. This is postulated to occur due to transmitted forces via the orbital bones to the orbital apex and optic canal. Elastic deformation forces of the sphenoid bone allows transfer of the force to the intracanalicular segment of the optic nerve.

Contusion of intracanalicular portion of optic nerve produces localized optic nerve ischemia and edema. The edematous ischemic axons result in further neural compression within the fixed diameter optic canal predisposing to the development of intracanalicular compartment syndrome. The basis of optic nerve decompression is enlarging this bottle neck area of optic foramen in order to prevent ischema caused due to nerve swelling.

Endoscopic optic nerve decompression controversies:

Nerve decompression should be performed only for indirect traumatic optic neuritis. Direct traumatic optic neuritis is an irreversible injury. Studies reveal taht there is a close association between initial visual acuity and final results after the procedure. Patients who are blind and have extremely poor light perception when examined first are poor candidates for the procedure. Fractures involving the optic canal as well as a fragment impinging on the nerve carry worse prognosis.

Traumatic optic neuropathy is the most common indication for optic nerve decompression. Decompression is ideally considered only in cases where there is a displaced fracture of the optic canal, with no evidence of anatomical disruption of the nerve. In patients with preserved light perception surgical decompression is considered with / without administration of steroids.

Timing of intervention is also controversial, but ideally speaking decompression should be done as soon as possible after optic neuropathy is diagnosed, and especially so if it is of sudden onset. In patients with traumatic optic neuropathy along with fractures of sphenoid wing and anterior clinoid process with displacement, lateral decompression via pterional approach should be considered.

Procedure:

The instruments used in endoscopic sinus surgery are used in this surgery also. In addition through cut dissecting instruments and powered instruments are also used. A 4 mm fine diamond burr is commonly used. When using powered drill adequate irrigation should be ensured in order to avoid thermal damage to the nerve during drilling process. The entire procedure is performed ideally under general anesthesia with the patient supine and head elevated.

The nasal cavity is packed with 4 % xylocaine with 1 in 100,000 units adrenaline. This decongests the nose and shrinks the turbinate thereby increasing the working space for the surgeon. It also reduces mucosal bleed during the entire process.

Technique:

1. Anterior and posterior ethmoidectomy is performed first. In addition natural ostium of the maxillary sinus is also widened. A complete ethmoidectomy will ensure that the lamina papyracea is exposed in its entirity.

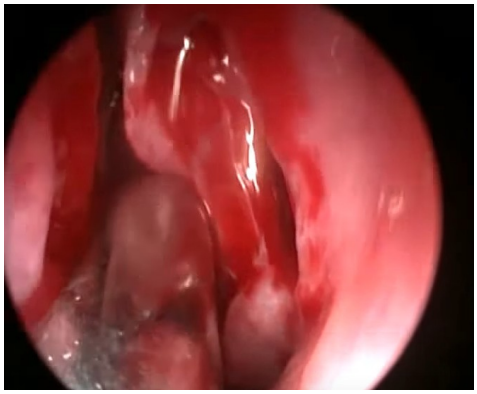
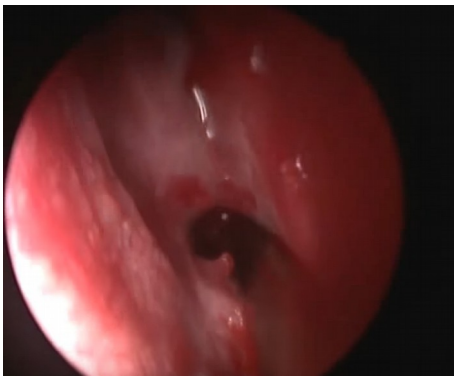


Image showing uncinate being medialized and removed



Screenshot showing sphenoid ostium exposed

A wide sphenoidotomy is performed. The anterior wall of sphenoid is resected to the level of skull base and up to the level of lamina papyracea. This procedure helps in the identification of orbital axis and the orbital apex.

Identification of the orbital apex and the optic canal:

The safest way to identify these structures is to resect the lamina papyracea posteriorly, starting about 10 – 15 mm anterior to the face of the sphenoid sinus. Since lamina papyacea can be separated and removed with a Freer's elevator. If it is thick then it needs to be drilled using a 4 mm diamond burr and reduce it to an egg shell thickness. Care should be taken not to injure the periorbita and the underlying extraocular muscles. If periorbita are injured then fat could be seen protruding into the operating field.

After removal of posterior portion of lamina papyracea, the periorbita is followed posteriorly where it could be seen converging at the orbital apex. The thick bone between the posterior ethmoid and the sphenoid is known as the optic tubercle.

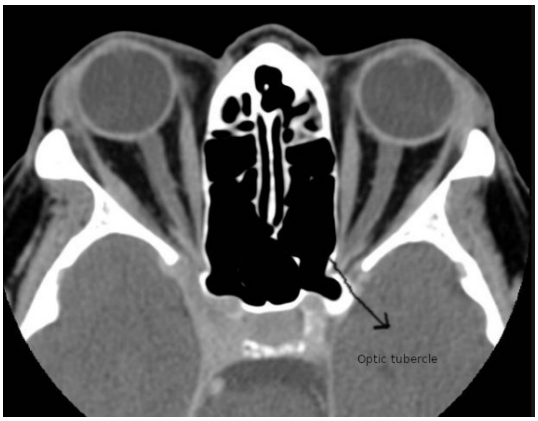
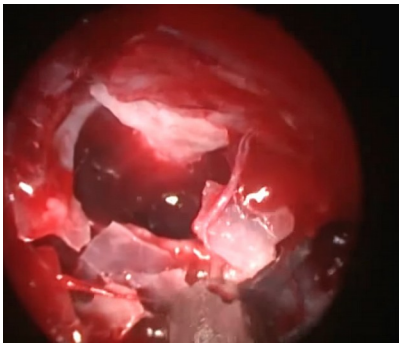


Image showing Optic tubercle



Sphenoid ostium seen widened

The annulus of Zinn is attached to the superior, inferior and medial margins of the orbital junction The bony protrusion of the optic canal into the sphenoid sinus is identified. It is the continuation of the optic tubercle. Diamond burr is used to thin this area of bone to egg shell thickness. This thinned out bone is removed using a Freer elevator. This exposes the optic nerve sheath. The optic nerve sheath is incised along the optic nerve and through the annulus of zinn. The incision is placed at the superomedial quadrant, as the ophthalmic artery is located in the inferomedial quadrant of the optic canal.

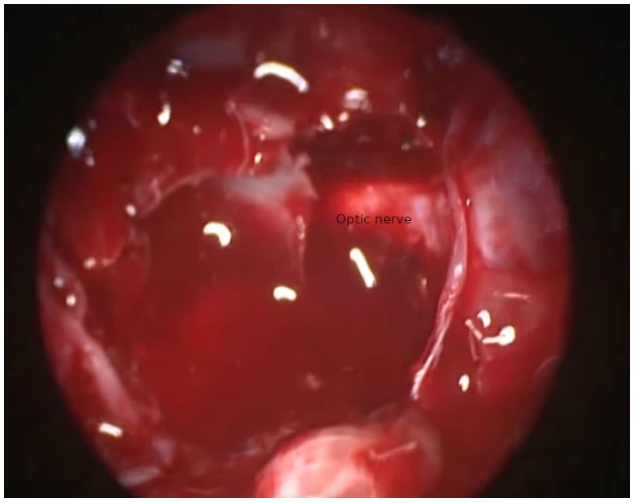


Image showing optic nerve exposed