Endoscopic Orbital Decompression

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Introduction:

Orbital decompression surgery has been indicated in patients with compressive optic neuropathy, severe corneal exposure, cosmetic deformity due to proptosis. Traditional orbital decompression approaches were fraught with complications. With the advent of nasal endoscopes decompression is being carried out transnasally under endoscopic guidance. The entire medial wall of orbit can be taken down transnasally using nasal endoscope, and the inferior wall of orbit can be removed using the same approach. Currently endoscopic orbital decompression is being performed commonly with very minimal complications. The aim of this paper is to review the current literature on the subject.

Thyroid associated orbitopathy can cause severe facial disfigurement. In severe cases it could lead even to blindness. Surgical decompression of orbit could very well alter facial appearance. Patients with thyroid associated orbitopathy should be warned that only a marginal improvement to facial appearance is possible. Frequent surgical procedures may be needed to produce optimal results. This procedure should never be considered as a beautification exercise. Majority of Thyroid associated orbitopathy patients don't require surgical treatment. The need for surgery increases significantly with age. Need for surgery triples after the age of 50. During active phase of this disease Medical management with immunosuppressive measures should be the first line of management.

Clinical features: Before attempting to manage a patient with Thyroid associated orbitopathy accurate assessment should be made regarding the disease activity, temporal progression and its severity. Basic aim is to differentiate active stage of the disease from the burnt-out stage. Treatment of these two conditions are rather different. Active moderate to severe congestive orbitopathy may need active intervention whereas mild congestive orbitopathy needs observation 2. Vision threatening dysthyroid optic neuropathy occurs in less than 5% of patients with Graves' disease.

Clinical features of optic neuropathy / impending optic neuropathy include:

1. loss of visual acuity

2. Disturbances in color vision

3. Visual field defects

4. Afferent pupillary defect

5. Swelling involving optic disc

Diagnosis can be confirmed by measuring Visual Evoked Potentials. If it shows increase in latency or reduction in amplitude the diagnosis is confirmed. If these patients are not picked up early and aggressively treated 30% of them may suffer irreversible vision loss. Risk factors for optic neuropathy include older age, smoking and male sex.

Pathophysiological mechanism implicated in optic nerve involvement:

Compression of optic nerve / its blood supply by the orbital contents especially by the hypertrophied intraocular muscles have been implicated. Studies have correlated intraocular muscle size and restriction of ocular mobility with incidence of optic neuropathy. Proptosis does not correlate well with the risk of optic neuropathy. Because of the potential risk of blindness due to dysthyroid optic neuropathy, this condition should be managed under war footing. Immediate decompression surgeries in these patients do not result in better results when compared to high dose intravenous methyl prednisolone therapy. Therefore, high dose of intravenous methyl prednisolone has been advocated as the first line of treatment. If intravenous steroid does not improve the situation in a couple of days then orbital decompression surgery must be resorted to in order to save vision. Patients who do not respond adequately to intravenous steroid therapy should be suspected to have orbital apex compression syndrome. This can be addressed only by decompression of orbital apex area via medial orbitotomy. In some patients dysthyroid orbital neuropathy can occur without any compression in the orbital apex area. Increased orbital pressure may cause this condition. MRI scan helps in differentiating these two conditions. Orbital decompression surgeries to manage exophthalmos reduction should be performed only after ophthalmological symptoms have stabilized at least for a period of 3- 6 months. One should not be in a hurry to perform orbital decompression procedures, because studies have revealed that early surgeries is not of help, on the contrary if performed early during the course of the disease it could lead to orbital motility problems. If orbital decompression is indicated it should be performed before extraocular muscle / eye lid surgery, because it could affect both extraocular muscle balance and eyelid position.

Pathophysiology of orbital compartment syndrome:

Anatomically orbit is an enclosed cone shaped compartment. It is bounded by bone circumferentially posteriorly and by the orbital septum anteriorly. Orbital septum is a rather stiff and tight structure allowing limited forward displacement of the eye in response to increased orbital volume. Intact orbital septum can withstand pressures up to 50 mm Hg, rarely even up to 120 mm Hg in some patients. The term “Compartment syndrome” was first used in orthopedics to indicate increased tissue pressure

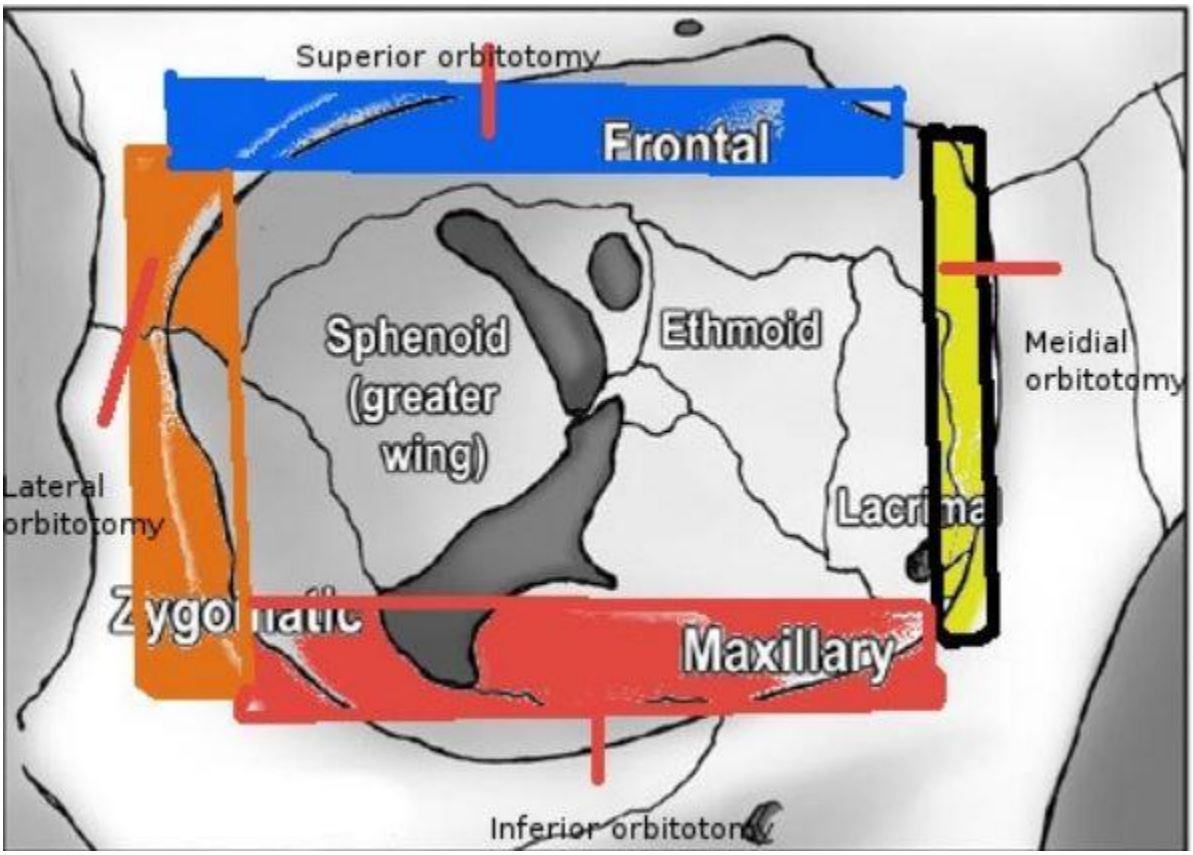
within enclosed space. The term “Orbital Compartment syndrome” was first used by Kratky et al in 1990. Significant increase in intraorbital pressure may compromise vascularity of optic nerve causing irreversible blindness. Inadequate blood flow in the posterior ciliary arteries, central retinal artery or vein or vasonervorum of optic nerve causes ischemic optic neuropathy or slow optic nerve degeneration. Thyroid associated orbitopathy should be differentiated from Dysthyroid optic neuropathy by performing ultrasound imaging of orbit. If B scan shows enlarged muscle bellies with normal tendons then the diagnosis of Thyroid associated orbitopathy is confirmed.

It helps in differentiating between active and inactive (burnt out diseases). In thyroid associated orbitopathy the extraocular muscles are isointense to normal muscle on T1-weighted images and hyperintense in T2-weighted images depending on tissue edema. Presence of tissue edema is an indication of active disease. The correlation between water content and inflammatory activity can be detected with MRI short term inversion recovery sequencing (STIR Sequencing). Only drawback of MRI is its inability to accurately image bony orbital structures. If decompression is being planned then CT imaging of orbit is a must.

Orbital decompression techniques:

History:

Earliest report of orbital decompression was published by Dollinger in 1911. In 1931 Naffziger popularized transfrontal orbital roof decompression. The advantage of this approach was that it allowed access to orbital apices of both sides and hence was very useful in managing bilateral disorders. The flip side was that proptosis reduction was not impressive. This procedure was also time-consuming needing assistance from neurosurgeon on the table. Communication of orbit with cranial contents lead to the development of pulsating proptosis. Sewall (1936) used medial approach to decompress orbit. In this approach the entire medial wall of orbit was taken down after performing a complete ethmoidectomy. If needed it can be extended up to the sphenoid sinus also allowing orbital contents to prolapse medially into the nasal cavity. Hirsch in 1950 used the technique described by Lewkowitz to perform inferior orbitotomy by removing the floor of the orbit through Caldwell – Luc approach. Walsh and Ogura in 1957 13 used Caldwell – Luc trans antral approach to decompress both inferior and medial orbital walls. Orbital contents were allowed to prolapse into maxillary antrum and nasal cavity. This approach had the advantage of doing away with skin incisions in the face. This approach too had its own flip side i.e. post-operative diplopia and infraorbital nerve hypesthesia. With the popular use of nasal endoscope, the entire nasal cavity could be accessed easily under endoscopic vision. Kennedy et al. 15 In 1990 performed endoscopic decompression of orbit by removing the medial wall of the orbit under endoscopic vision. Michel et al. In 2001 popularized this procedure.



Lateral orbitotomy – Dollinger (1911)

Superior orbitotomy – Naffziger (1931)

Medial orbitotomy – Sewall (1936)

Inferior orbitotomy – Hirsch (1950)

Bony orbital decompression:

Orbital decompression can be performed by removal of one or more walls of the orbit. Graded orbital decompression is always preferred depending on the degree of proptosis. This concept was first suggested by Kikkawa et al. Three wall decompression provides the best proptosis reduction with acceptable esthetic appeal. During 1980's two wall decompression involving medial and inferomedial walls of orbit was practiced. This procedure had a high incidence of post-operative diplopia due to inferior displacement of globe. This can easily be avoided by preserving the inferomedial strut between ethmoid and maxillary sinuses. Goldberg 16 et al. Demonstrated that deep lateral wall decompression alone caused 4.5 mm reduction in proptosis. He used the term extended lateral orbital decompression

to include three key areas: Lacrimal keyhole – area around lacrimal gland fossa Basin of the infraorbital fissure – the portion of zygomatic bone and lateral maxilla around infraorbital fissure.

Sphenoid door jam – Thick trigone of greater wing of sphenoid which borders infratemporal fossa laterally and middle cranial fossa posteriorly. This area makes the largest volume of bone contribution to orbit. Removal of bone from this area reduces proptosis by 6 mm.

Endoscopic Medial wall decompression:

This procedure is still under evaluation. Since the approach is trans nasal, facial incision is avoided. The medial wall of orbit is rather thin in this area. After exenteration of ethmoidal air cells this wall can easily be taken down allowing the orbital contents to prolapse into the nasal cavity. This procedure can be performed either under LA or GA. The nasal cavity is decongested. Complete uncinectomy and ethmoidectomy is performed. A wide middle meatal antrostomy is performed. The floor of the orbit and the posterior wall of maxilla should be clearly visible through the antrostomy. A wide antrostomy won't get blocked even after the prolapsing orbital content fills the nasal cavity and maxillary sinus. Infraorbital nerve should be visualized using a 45° endoscope because this represents the lateral limit of bone resection. Frontal recess area should be cleared adequately. Trans ethmoidal sphenoidotomy should also be performed. Anterior limit of resection corresponds to nasolacrimal duct, while superior limit corresponds to the floor of anterior cranial fossa marked by the presence of ethmoidal arteries. Inferiorly resection should stop at the level of insertion of inferior turbinate. Author invariably removes middle turbinate to create more space for the prolapsing orbital contents. Lamina papyracea should be completely skeletonized and removed using periosteal elevator. Lamina is removed carefully without traumatizing periorbita. It should completely be removed till the posterior ethmoid, close to the optic nerve where the bone is thicker. Only after fully exposing the periorbita should it be incised to allow fat to prolapse into the nasal cavity and maxillary sinus cavity. Endoscopic decompression could achieve proptosis reduction between 3 – 5 mm. Greater reduction can be achieved if combined with lateral orbitotomy.

It is very important to retain lamina papyracea in the region of frontal recess to prevent obstruction due to prolapsing orbital fat.

Complications of this procedure include:

1. Diplopia

2. Sinusitis

3. Frontal & maxillary sinus mucocele

4. CSF leak

Walsh – Ogura decompression: Traditionally this procedure has been performed to manage Graves ophthalmopathy. This surgery is performed via trans antral Caldwell Luc approach. Two walls of orbit are removed i.e. medial and inferior walls. Medial wall removal is difficult in this procedure as it is difficult to visualize lamina papyracea transantrally, hence it is virtually impossible to completely decompress medial wall of orbit 18. This procedure is entirely not risk free. If too much inferior wall is taken down it could cause hypoglobus (inferior displacement of orbit).

Fat removal orbital decompression:

This procedure was first reported by Olivari in 1988. This procedure was considered to be relatively safe when compared to bony decompression according to him. Removal of 6ml of fat on an average contributed to satisfactory results. It has been estimated that normal average orbital fat volume is about 8ml. This could increase to 10 ml in patients with thyroid associated orbitopathy. This procedure is suited for patients who have a volumetric increase in orbital fat deposition causing proptosis. Patient selection should be carefully made after performing MRI imaging of orbit. Infero medial removal of orbital fat could be a worthwhile option of treating proptosis as this area is devoid of crucial anatomical structures.

Lateral orbitotomy (lateral wall decompression):

This approach is credited with the maximum reduction of exopthalmos. Indications for this procedure include:

1. Esthetic rehabilitation for exphthalmos

2. Retrobulbar pressure

3. Exposure keratopathy / Lagopthalmos

4. Dysthyroid optic neuropathy

Procedure:

This surgery is ideally performed under general anesthesia. Skin incision begins at the lateral third of upper eyelid crease. It follows a sigmoid course over the zygomatic bone. Orbital rim is exposed by blunt dissection. Temporalis muscle in this area should also be removed till the periosteum becomes visible. This exposed periosteum is cut along the orbital rim and stripped away from the bone. Globe and orbital contents are transferred nasally using malleable retractors. Two osteotomies need to be performed to remove the lateral orbital wall. The first osteotomy is just above the frontozygomatic suture line and the next one is at the beginning of frontal process of zygoma. After complete removal of lateral orbital wall, the average increase in orbital volume works out to 1.6 ml. Periorbita is opened now. Prolapsing fat can be removed. A small suction drain is placed behind the globe and the wound is closed in layers. Compression bandage is a must during first 24 hours. Amount of blood in the drain and pupillary reflex should be constantly checked during the first 24 hours after surgery. It should be borne in mind that intraocular bleeding can cause precipitous increase in ocular pressure compromising vision.

Complications of this procedure include:

1. Diplopia

2. Loss of vision due to bleeding and increase in intraocular tension

3. Temporary numbness over zygomatico temporal supply area of trigeminal nerve

4. Mild oscillopsia during chewing

5. Temporalis muscle wasting

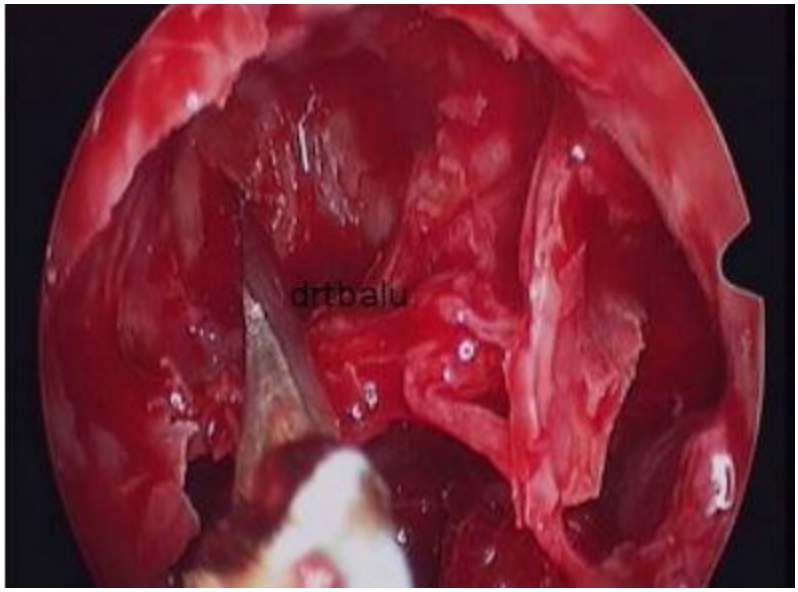


Image showing gradual removal of lamina papyracea

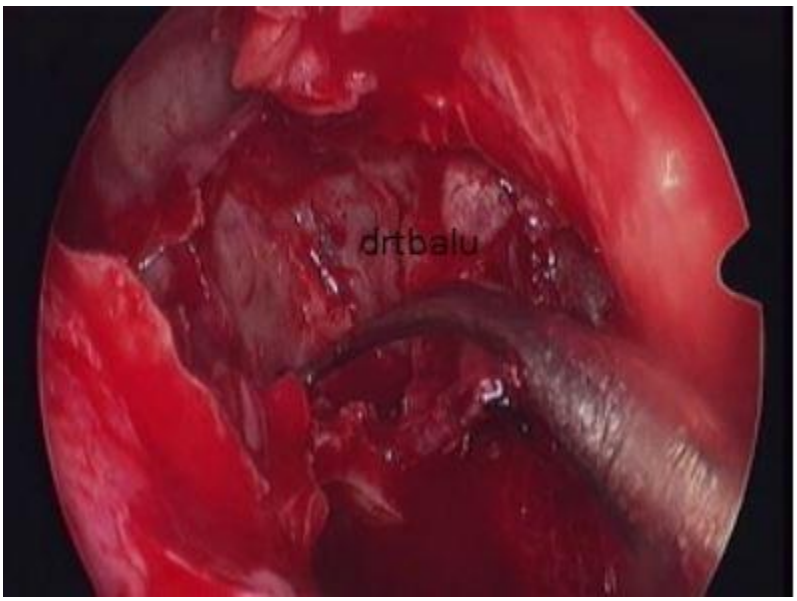


Figure showing removal of lower portion of medial wall of orbit

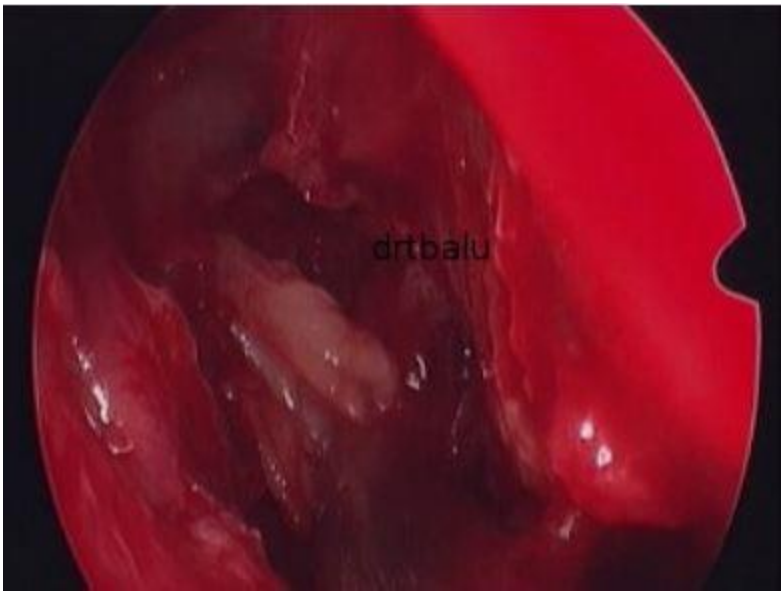


Image showing prolapse of orbital fat into the nasal cavity after removal of medial wall and incising the periorbita

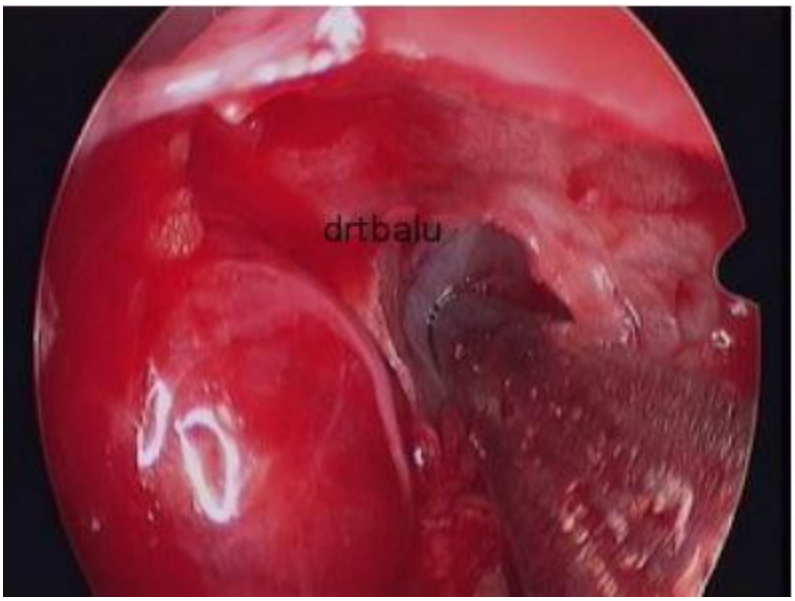


Image showing prolapse of orbital contents into the nasal cavity after complete removal of medial wall