

Endoscopic orbital decompression for Graves' orbitopathy

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ABSTRACT

Aim: To study the efficacy of endonasal endoscopic orbital decompression in cases of Graves' orbitopathy. **Material and Methods:** A total of 24 orbits in 12 patients underwent endoscopic orbital decompression for graves orbitopathy in the period between October 2002 and December 2010. Indications for surgery included proptosis, corneal exposure, keratitis, and compressive optic neuropathy. Decompression was accomplished by the removal of the medial and part of inferior wall of the orbit and slitting of the orbital periosteum. Pre and postoperative exophthalmometry measurements and visual acuity were recorded and compared. **Results:** A mean orbital regression of 3.70 mm was noted following endoscopic decompression. The visual acuity improved significantly in one of two eyes decompressed for failing visual acuity secondary to optic nerve compression. Transient diplopia was invariable following surgery but resolved over the next 8 weeks. One case manifested unilateral frontal sinus obstruction symptoms 4 months postoperatively and responded to medical therapy. **Conclusion:** Endonasal endoscopic orbital decompression provides for an effective, safe, and minimally invasive treatment for proptosis and visual loss of Graves Orbitopathy. Long-term problems with diplopia were not noted in the endonasal endoscopic approach for orbital decompression.

Key words: Endoscopic, Graves disease, orbital decompression, proptosis, visual loss

INTRODUCTION

Graves disease is an autoimmune multisystem disorder. The orbital manifestations of Graves disease (Graves orbitopathy aka dysthyroidorbitopathy, Graves' ophthalmopathy) is consequent to immune complex deposition and inflammatory cell infiltration of the orbital fat and muscles and subsequent fibrosis. This leads to an increase in the intraorbital contents and the consequent proptosis and other symptomatology.

The clinical manifestations range from tearing, photophobia, proptosis, exposure keratitis, and diplopia to visual loss.

30 to 50% patients with Graves disease may manifest exophthalmos,^[1] but fortunately only 2 to 7% develop optic nerve involvement consequent to vascular compromise or direct nerve compression leading to visual loss.^[2]

In the early stages of the disease, the first line of management is by high doses of corticosteroids. However, the symptoms tend to recur after discontinuation of therapy, and the continuing use of corticosteroids over prolonged periods is limited by their side effects. Other treatment options which have been advocated are external beam radiation therapy and immunosuppressive therapy, but none of these treatments is consistently efficacious.^[3,4]

Surgical decompression is indicated for long-standing cases recalcitrant to steroid treatment and for acute cases with visual loss. Various external approaches have been adopted to decompress the orbit, of which the Walsh and Ogura approach has been the most favored.^[5] This approach provides an effective and cosmetically acceptable result by the removal of the inferior and medial walls of

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the orbit via the transantral route. The advent of nasal endoscopy over the last two decades has allowed for a similar decompression to be undertaken by the transnasal endoscopic route.

To the best of our knowledge, there have been no results of endoscopic orbital decompression reported from the Indian subcontinent where many patients report for treatment in advanced stages. A literature search over Pubmed, J-gate, and Google has yielded no published reports from the subcontinent on this aspect. This report details our experience with a series of 12 consecutive cases wherein the endoscopic approach was utilized to decompress 24 orbits in patients with graves orbitopathy.

MATERIALS AND METHODS

All cases of graves orbitopathy, referred from the Departments of Endocrinology and Ophthalmology, who underwent endoscopic orbital decompression during the period October 2002 to December 2010 at our center, were included in this study.

The relative exclusion criteria included the following:

- Active disease as judged by the clinical score without any acute visual loss
- Hyperthyroid or hypothyroid status (the patients were made euthyroid before subjecting them to surgery)
- Acute sinonasal infections
- ASA grade III or worse

All cases had a complete otolaryngological and ophthalmological examination including testing of visual acuity and assessment of proptosis using Hertel exophthalmometry. The Hertels exophthalmometer consists of a horizontal calibrated bar with movable carriers at each side. Each carrier consists of mirrors inclined at 45 degrees to reflect both the scale reading and the apex of the cornea. Notches on the side carriers are placed on the bony lateral orbital margins of the patient. The patient is then asked to fixate on a point on the examiner's forehead. The apex of the cornea of each eye is superimposed on the millimeter scale reading by the inclined mirrors.

The severity of orbitopathy was graded as per the American Thyroid Association (ATA) classification (*aka* NO SPECS Classification) [Table 1].^[6]

The disease activity was assessed using the clinical activity score (CAS) as described by Mourits *et al.*^[7] This scoring is based on four of the five classical signs of inflammation (i.e., pain, redness, swelling, and impaired function) and consists of 10 items [Table 2]. For each item

Table 1: Class/Grade of orbitopathy as per the American Thyroid Association

Class 0	No signs or symptoms
Class 1	Only signs, no symptoms (signs limited to upper lid retraction and stare, lid lag, mild proptosis)
Class 2	Soft tissue involvement with symptoms and signs <ol style="list-style-type: none"> Minimal Moderate Marked
Class 3	Proptosis (>3 mm) with or without symptoms <ol style="list-style-type: none"> 3- to 4-mm increase over upper normal 5- to 7-mm increase 8 or more mm increase
Class 4	Extraocular muscle involvement (usually with diplopia, other symptoms, signs) <ol style="list-style-type: none"> Limitation of motion at extremes of gaze Evident restriction of motion Fixation of a globe or globes
Class 5	Corneal involvement (primarily a result of lagophthalmos) <ol style="list-style-type: none"> Stippling of cornea Ulceration Clouding, necrosis, perforation
Class 6	Sight loss (caused by optic nerve involvement) <ol style="list-style-type: none"> Disc pallor or choking, or visual field defect: Visio 20/20 to 20/60 Same, but vision 20/70 to 20/200 Blindness (i.e., failure to perceive light, vision less than 20/200)

Table 2: The 10 items of clinical activity score for graves orbitopathy

Pain	1	painful oppressive feeling on or behind the globe, during the last 4 weeks
	2	pain on attempted up, side or down gaze during the last 4 weeks
Redness	3	redness of the eyelid(s)
	4	diffuse redness of the conjunctiva, covering at least one quadrant
Swelling	5	swelling of the eyelid(s)
	6	chemosis
	7	swollen caruncle
	8	Increase of proptosis of ≥ 2 mm during a period of 1-3 months
Impaired function	9	Decrease of eye movements in any direction $\geq 50^\circ$ during a period of 1-3 months
	10	Decrease of visual activity of ≥ 1 line(s) on the snellen chart (using a pinhole) during a period of 1-3 months

For each item present, 1 point is given. The sum of these points is the clinical activity score

present, one point is given and the sum of all the points is the score of the patient.

The thyroid status was assessed in all the cases.

Presurgical contrast-enhanced computed tomography was undertaken in all. CT scanning was undertaken to delineate the ethmo-sphenoid anatomical variations prior to surgical exploration, and also for the characteristic and confirmatory CT findings of graves orbitopathy ("tendon sparing" enlargement of extra-ocular muscles, and the "Coca Cola Bottle sign"-[Figure 1]).

The indications for surgery included the following:

- Proptosis with cosmetic disfigurement which had failed conservative treatment with steroids.
- Corneal exposure with keratitis secondary to proptosis
- Compressive optic neuropathy

The ophthalmological findings, grade of orbitopathy, and the indication for surgery for individual cases is listed in Table 3.

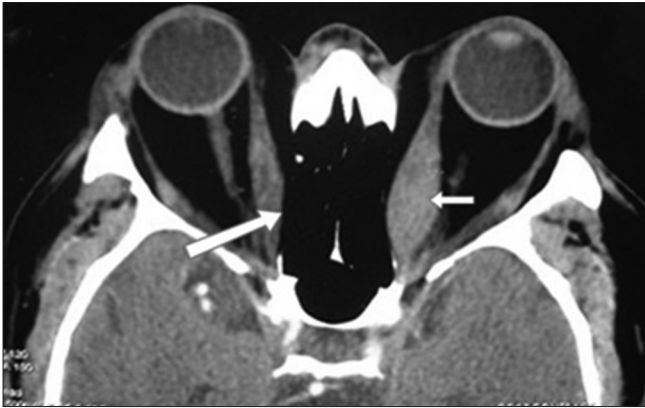


Figure 1: Axial sections of contrast-enhanced CT scans showing the characteristic tendon sparing enlargement of both medial recti (as indicated by the short arrow), and also a medialward expansion of both orbits leading to bilateral lateral compression of the ethmoids and the ethmosphenoid demonstrating a central waist akin to a "Coca Cola bottle" (long arrow)

Surgical technique

All cases had bilateral orbital decompression by the nasal endoscopic approach under general anesthesia. The initial operation involved bilateral wide endoscopic exposure of the maxillary sinuses and the ethmoid sinuses. Following an anterior ethmoidectomy, the maxillary sinus ostium was exposed and widely enlarged in all directions, particularly anteriorly (till the nasolacrimal duct) so as to create a large middle meatal antrostomy. The large antrostomy was necessary to provide good access to the orbital floor for the subsequent decompression and also to ensure that following the orbital decompression and prolapse of the orbital contents into the maxillary sinus, the surgically widened ostium would continue to remain patent for ventilation of the remaining maxillary sinus cavity.

A complete endoscopic ethmoidectomy (anterior and posterior ethmoidectomy) was then completed so as to expose the entire medial orbital wall (lamina papyracea). The sphenoid sinus and its drainage tract medial to the middle turbinate was left unviolated. The frontal sinus drainage tract with the overlying mucosa, in the roof of the anterior ethmoids, was also specifically preserved. Dissection was then continued inferiorly along the medial orbital wall to further expose the floor of the orbit till the inferior orbital nerve and its canal. This exposed the entire bony limits of the subsequent orbital decompression.

Table 3: Ophthalmological profile of the cases under study

Age/Sex	Preoperative status			Indication for surgery	Postoperative status		
	Proptosis (mm)	Visual acuity	Orbitopathy class/grade		Proptosis (mm)	Visual acuity	Orbitopathy class/grade
42/M	R-30 L-30	R-6/9 L-6/60	III C	Proptosis, Corneal exposure	R-26 L-25	R-6/9 L-6/60	III C
50/M	R-24 L-24	R-6/6 L-6/6	III B	Proptosis, Corneal exposure	R-21 L-21	R-6/6 L-6/6	III A
37/M	R-24 L-22	R-PL-ve L-6/18	VI C	Painful keratitis Bilateral	R-20 L-19	R-PL-veL-6/9	VI C
50/M	R-32 L-32	R-3/60 L- PL-ve	VI C	Rapidly decreasing vision/ compressive optic neuropathy	R-26 L-26	R-6/18 L-PL -ve	VI C
25/M	R-24 L-24	R-6/6 L-6/6	III B	Proptosis	R-21 L-21	R-6/6 L-6/6	III A
25/M	R-23 L-24	R-6/9 L-6/9	III B	Proptosis, corneal exposure	R-21 L-21	R-6/9 L-6/9	III A
47/M	R-30 L-30	R-6/12 L-6/18	IV B	Proptosis, corneal exposure	R-26 L-25	R-6/12 L-6/12	IV A
35/M	R-24 L-24	R-6/12 L-6/9	III B	Proptosis, corneal exposure	R-21 L-21	R-6/12 L-6/9	III A
60/M	R-24 L-22	R- 6/18 L- 6/9	V B	Proptosis, R exposure keratitis	R-20 L-20	R- 6/9 L-6/6	V B
24/F	R-28 L-30	R-6/6 L-6/9	IV C	Proptosis, Ophthalmoplegia	R-24 L-25	R- 6/6 L-6/9	IV A
20/F	R-22 L-22	R-6/6 L-6/6	III A	Proptosis, corneal exposure	R-18 L-18	R- 6/6 L-6/6	II A
18/M	R-27 L-27	R-6/9 L-6/6	III C	Proptosis, corneal exposure	R-24 L-24	R-6/9 L-6/6	III B

R: Right, L: left, PL -ve: No perception of light

The medial orbital wall (lamina papyracea) and the medial section of the orbital floor were then removed by initially fracturing the thin lamina papyracea and then insinuating a periosteal elevator and bone biting antegrade and retrograde forceps into the sub-periosteal space. Care was taken at this stage to not violate the orbital periosteal layer (periorbita) encasing the orbital contents. The bone removal extended from the level of the roof of the ethmoids above and continued along the medial wall to the orbital floor laterally till the infraorbital nerve/infraorbital canal [Figure 2a]. The posterior limit of removal was till the orbital apex lateral to the posterior ethmoid sinuses and the anterior limit was till the maxillary line and about 1-1.5 cm short of the orbital rim. The lamina papyracea along the frontal sinus drainage tract was specifically preserved.

The periorbita was then incised along its entire horizontal length from posterior to anterior by three parallel incisions placed approximately 1 cm apart [Figure 2b]. This resulted in prolapse of the orbital fat into the ethmoid and maxillary sinuses and a consequent decrease in proptosis.

Perioperatively, the patients received oral antibiotics and oral prednisone (0.5 mg/kg tapered over 1 week). All patients were followed up for a minimum period of 6 months.

Outcomes were determined by comparing the preoperative and 6-month postoperative proptosis measurements and visual acuity.

Any complication encountered was recorded.

RESULTS

A total of 24 orbits in 12 patients were decompressed during the said study period.

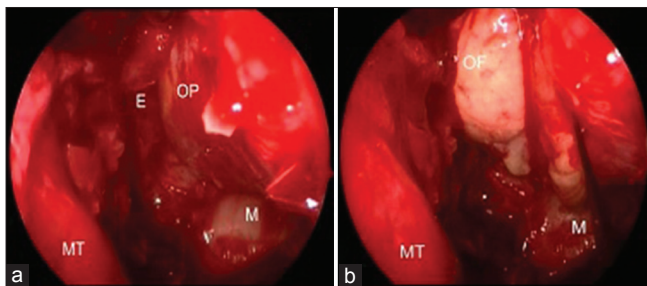


Figure 2: Surgical stills of left endoscopic orbital decompression. (a)-completed ethmoidectomy with extirpation of the ethmoid (E) and maxillary sinuses (M) and removal of medial and part of inferior orbital walls to expose the orbital periosteum (OP). The middle turbinate (MT) is seen in the medial part of the surgical field. (b)-Slitting of the orbital periosteum with resultant prolapse of the orbital fat (OF)

The case group included ten males and two females with their ages ranging from 20 to 50 years (mean age, 36.5 years).

Details of ophthalmological findings and the indications for surgery are listed in Table 3. The degree of eye protrusion as measured from the lateral canthus (normal ≤ 18 mm) ranged from 22 to 32 mm (mean, 25.9 mm). The duration of proptosis ranged from 1 to 8 years (mean, 3.5 years).

All patients (except case 4) had a low CAS of less than 4, indicating that the disease was inactive in all these cases. Case 4 had evidence of active disease but was taken up for emergency surgery as the patient had rapidly decreasing vision.

All the patients included in the study had been administered a course of oral wysolone in the dose of 1-1.5 mg/kg for 4 to 6 weeks in the past. Three cases had had more than one course of oral wysolone in the past.

No patient had any preoperative evidence of sinusitis.

All the patients were euthyroid at the time of surgery.

The commonest indication of surgery was proptosis with associated corneal exposure [Table 3]. Two cases sought surgery for cosmetically disfiguring proptosis alone (case 5 and case 10). In three cases (5 eyes), emergent surgery was necessitated because of further complications of painful exposure keratitis (3 eyes), or recent rapidly progressive bilateral visual loss suggesting optic nerve compression (2 eyes).

Following surgery, the proptosis improved by various degrees in all the cases (range, 2-6 mm; mean reduction, 3.70 mm). The painful keratitis manifested in three eyes healed in all, but all developed corneal opacities as a consequence of the keratitis. In the case with surgery undertaken for bilateral rapidly progressive visual loss with presumed optic nerve compression (case 4), significant improvement of vision was noted in the eye with partial visual loss, but no improvement was noted in the eye with complete visual loss.

Post surgery, transient new onset diplopia was invariable in all cases with binocular vision. This however resolved over the next 2 to 8 weeks, either spontaneously or with the aid of convergence exercises.

One case had symptoms of persistent frontal sinusitis manifesting 4 months after surgery. This resolved with conservative therapy of decongestants and a 3-week course

of antibiotics. A postoperative CT scan undertaken in this patient demonstrated prolapse of orbital contents into the ethmoid and maxillary sinuses [Figure 3].

DISCUSSION

Endoscopic decompression of the orbit produces an effective reduction in proptosis. Our experience of a mean regression of proptosis of about 3.70 mm is similar to what has been reported in previous studies adopting a similar approach^[8-15] [Table 4] and comparable to the reduction of 3 to 5.5 mm reported from the non-endoscopic Walsh Ogura technique.^[16,17]

Two eyes (case 4) manifested with optic nerve compression and progressive deterioration of visual acuity. Emergent surgery was effective in dramatically reversing visual loss in the one eye with partial visual loss, but was ineffective in the eye with complete visual loss. This is analogous to other situations with compressive optic neuropathy wherein complete visual loss, unless immediately treated within hours, leads to irreversible ischemic injury to the optic nerve.^[18]

The endoscopic approach offers the advantage of enabling a direct and complete access to the medial

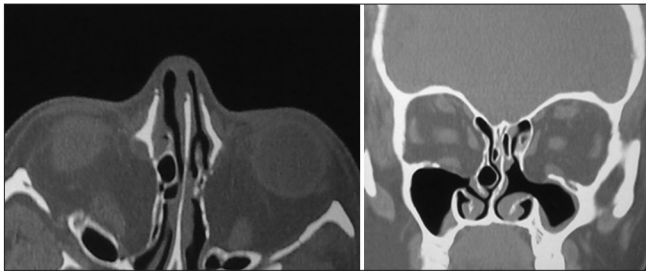


Figure 3: Postoperative scans demonstrating removal of the medial wall and part of the floor of the orbit and prolapse of the orbital contents into the adjacent sinus cavities. The axial and coronal scans indicate removal of the entire medial wall with the orbital tissues occupying the ethmoid sinuses and in contact with the middle turbinate. The coronal scan also demonstrates the removal of the medial half of the orbital floor and orbital tissues are seen prolapsing into the maxillary sinus

Table 4: Previous reports on endonasal endoscopic orbital decompression and thereported mean regression in proptosis

Study	Mean reduction (mm)
Malik, <i>et al.</i> ^[8]	3.7
Kasperbauer, <i>et al.</i> ^[9]	2.5
Mann W, <i>et al.</i> ^[10]	4.4
Koay, <i>et al.</i> ^[11]	3.9
Lund, <i>et al.</i> ^[12]	4.4
Wee, <i>et al.</i> ^[13]	4.4
Wright, <i>et al.</i> ^[14]	3.6
Neugebauer, <i>et al.</i> ^[15]	3.0

orbital wall (lamina papyracea) essential for safe and effective decompression of the orbit and the optic nerve. The transtrantral route on the other hand approaches the lamina papyracea obliquely, thus making the skull base more vulnerable to injury, and also restricting access to the posterior lamina papyracea.

Though the orbital floor removal is more restricted endoscopically as compared to other open approaches, the disadvantage of a limited removal of the floor for correction of proptosis is overcome by an unparalleled removal of the medial wall. Besides, a more aggressive removal of the floor may increase the incidence of hypoglobus and diplopia^[19,20] and infraorbital nerve injury.

In our series, new onset diplopia was seen in all cases (excepting the cases with no binocular vision) in the immediate postoperative period. However, this was transient and did not require any corrective surgery/corrective lenses. Reports mention that upto 69% of patients undergoing orbital decompression develop postoperative/worsening of pre-existing diplopia,^[19] and thus all patients should be counseled accordingly prior to surgery.

The surgical methods advocated to avoid diplopia are (a) preservation of a horizontal bony strut at the junction of the medial wall and floor of the orbit^[14] and (b) “balanced decompression” involving removal of both medial and lateral orbital walls^[21,22] so as to avoid the displacement of the orbit in any one direction. The endoscopic approach does not allow access to the lateral orbital wall, thus making it impossible to undertake the “balanced decompression” technique by a pure endoscopic approach. The alternate technique of preservation of the “inferomedial strut” too has proved unsuitable in our experience, as such preservation can lead to significant compromise of access to the subperiosteal plane along the orbital floor and may thus limit the surgeon in achieving a satisfactory decompression.

Nevertheless, our patients have had no long-term problems with regard to diplopia. This may be mainly related to the endoscopic approach providing for very clearly demarcated limits to the decompression and thus achieving very similar and symmetrical decompression on both sides. Furthermore, the excellent visualization afforded by the endoscopic approach enables for controlled incisions on the periosteum so as to symmetrically decompress the orbit, and also to preserve longitudinal strips of the periosteum which act as hammocks supporting the globe and the orbital contents. The minimally invasive nature of the endoscopic approach may also minimize postoperative

inflammation and the resultant variable fibrosis consequent to such inflammation.

CONCLUSION

Endoscopic nasal decompression of the orbit is a safe and effective approach for reducing the proptosis and decompression of the optic nerve in cases of Graves' orbitopathy.

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