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ORIGINAL RESEARCH—ENDOCRINE SURGERY

Revision endoscopic orbital decompression in the management of Graves' orbitopathy

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ABSTRACT

OBJECTIVE: Endoscopic orbital decompression has proved to be an effective treatment for Graves' orbitopathy. In select patients, however, persistent or recurrent orbital symptoms necessitate additional therapy. The objective of this study is to determine the safety and effectiveness of revision endoscopic orbital decompression in patients with refractory Graves' orbitopathy.

STUDY DESIGN: Case-control series.

SETTING: Academic medical center.

METHODS: The study population consisted of 10 patients with Graves' orbitopathy who underwent 13 revision endoscopic orbital decompressions (three bilateral cases) between 1991 and 2008. Outcome measures, including reduction in proptosis, improvement in visual acuity, and complication rates, were compared with a control cohort of 10 consecutive patients (16 orbits) who underwent primary endoscopic decompression during the same time period.

RESULTS: Indications for revision decompression included exposure keratopathy (n = 8, 62%), optic neuropathy (n = 3, 23%), and gaze restriction (n = 2, 15%). There were no intraoperative complications. Mean reduction in proptosis was 1.4 mm less for patients who underwent revision decompression compared with primary cases (mean decompression 3.6 ± 1.0 mm vs 5.0 ± 2.1 mm, respectively), although this difference was not statistically significant ($P = 0.13$). Visual acuity improved in 62 percent of revision cases, compared with 20 percent of primary cases ($P = 0.09$). Rates for postoperative complications, which included sinusitis and frontal mucocele formation, were also similar between revision and primary decompression groups (38% vs 13% respectively, $P = 0.17$).

CONCLUSIONS: This report is the first to describe the endoscopic technique for revision orbital decompression. It appears to be a safe and effective procedure for the treatment of refractory orbitopathy in patients with Graves' disease.

Graves' disease is a multisystem autoimmune disorder that affects the thyroid gland, skin, and orbit. Although hyperthyroidism is the most common presenting manifestation, up to 80 percent of patients with Graves' disease develop ocular findings.[1] Graves' orbitopathy results from the accumulation of lymphocytes and deposition of glycosaminoglycans in orbital soft tissues, leading to enlargement of extraocular muscles and orbital fat. In the restrictive confines of the bony orbit, the discrepancy between orbital volume and its expanded contents causes anterior displacement of the globe and posterior pressure in the orbital apex, which can result in disfiguring exophthalmos, exposure keratopathy, and optic nerve compression. Severe orbital disease poses a threat to vision in 3 to 5 percent of patients with Graves' orbitopathy.[2]

Since the early 1990s, endoscopic orbital decompression has become the surgical treatment of choice for patients with Graves' orbitopathy.[3-6] Compared with external approaches for orbital decompression, the endoscopic technique provides enhanced visualization of critical anatomical regions, including the skull base and orbital apex, and avoids facial or intraoral incisions. Endoscopic orbital decompression allows for removal of the entire medial orbital wall, as well as the medial portion of the orbital floor, along with the underlying periorbital fascia. This procedure enables the enlarged orbital muscles and fat to prolapse into the ethmoid and maxillary sinuses, allowing for the proptotic globe to recess into the orbit. With ocular recession ranging from 2 to 12 mm, this procedure improves cosmesis and reduces symptoms associated with exposure keratopathy and optic neuropathy.[7,8]

For the vast majority of Graves' patients, a single decompression procedure is successful at relieving orbital symptoms; however, symptoms may persist or recur despite surgical decompression in those with severe or progressive eye disease. Persistent orbitopathy can result from inadequate removal of the bony orbital walls or periorbital fascia, whereas recurrent orbitopathy can occur from progression or reactivation of Graves' disease. The purpose of this study is to determine the safety and effectiveness of endoscopic revision orbital decompression in patients with refractory Graves' orbitopathy.

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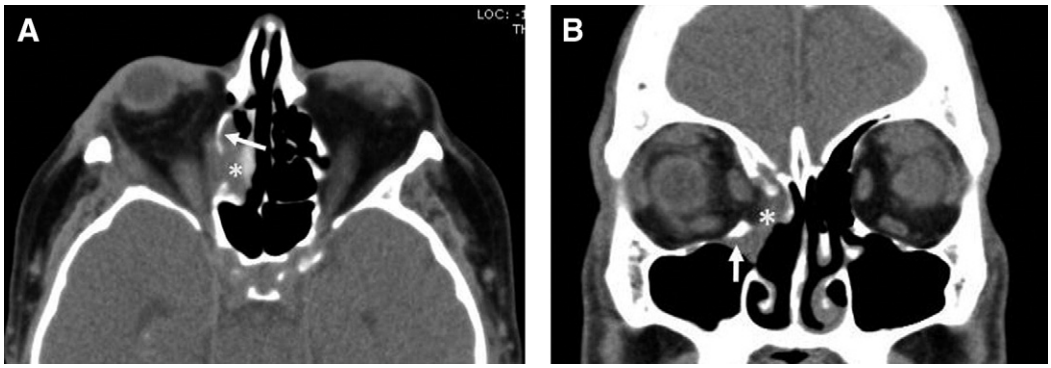

Figure 1 (**A**) Axial CT image of patient #3 showing retained portion of lamina papyracea (*arrow*) after prior right orbital decompression. (**B**) Coronal CT image of patient #1 showing retained portion of medial orbital floor (*arrow*) after prior right orbital decompression. Orbital contents have herniated into the sinuses from previous surgery (*asterisk*). Removal of remaining bone allowed for additional orbital decompression.

METHODS

Medical records of all 185 cases of endoscopic orbital decompression performed between December 24, 1991, and June 31, 2008, at the Massachusetts Eye and Ear Infirmary by the senior author (R.M.) were reviewed. Patients with Graves' orbitopathy who had undergone one or more prior orbital decompressions (endoscopic or nonendoscopic) on the same side were included in the study group. This group consisted of 10 patients who underwent 13 cases of revision decompression (three bilateral cases). Complete postoperative data were available in seven patients who underwent eight revision orbital decompressions. To match the sample size of the study group, the control cohort consisted of 10 consecutive patients with Graves' orbitopathy who underwent 16 endoscopic primary orbital decompressions (six bilateral cases) during the same time period. All bilateral decompressions were performed as staged procedures. Outcome measures included intraoperative and postoperative complications to assess safety, as well as reduction in proptosis, changes in visual acuity, and improvement in ocular motility to determine efficacy.

Although a larger study population would have been preferable for the purpose of statistical analysis, because of the relatively low failure rate of primary orbital decompression, only 10 patients were identified who required revision surgery over an 18-year period. Wilcoxon rank sum and χ^2 tests were used to compare patient characteristics between the study and control groups. Repeated measures analysis with mixed effects models and generalized estimating equations techniques were used to compare outcomes between the two cohorts. Statistical significance was defined as $P < 0.05$. Institutional review board approval for the study was obtained from the Human Subjects Committee of the Massachusetts Eye and Ear Infirmary.

Surgical Technique

Preoperative CT of the orbits and sinuses were reviewed to identify bony remnants of the medial orbital wall or floor whose removal would allow for further decompression (Fig 1). The initial steps for endoscopic revision orbital decompression are similar to those previously described for primary decompression.[7] Intraoperative submucosal injections of 1 percent lidocaine with epinephrine 1:100,000 are placed along the lateral nasal wall and medial orbital wall. Care is taken to avoid intraorbital injection of a large volume of anesthetic, which can result in transient pupillary dilation. If not previously completed, a generous maxillary antrostomy and total sphenoethmoidectomy are performed. The maxillary ostium needs to be large enough to access the medial orbital floor. Care must be taken to avoid injuring orbital contents that have herniated into the sinonasal cavities from prior surgery. Repeated palpation of the globe alerts the surgeon to previously decompressed regions.

Areas of retained lamina papyracea and orbital floor are identified by gentle palpation with a blunt instrument, such as an ostium seeker or spoon curette. Image-guidance technology can also be helpful to localize residual orbital bone to be removed. Remnant lamina papyracea is exposed by elevating and resecting overlying mucosa and scar tissue. The bone is then removed with a Blakesley forceps in a superior direction to the level of the ethmoid roof, in a posterior direction to the sphenoid face, in an anterior direction to the maxillary line (frontal process of maxilla), and in an inferior direction to the orbital floor. The medial orbital floor is removed in a lateral direction as far as the infraorbital canal in an attempt to achieve maximal inferior decompression.

In areas where periorbita and bone have been previously removed, dense scar tissue often reconstitutes the orbital walls. This tissue, along with any newly exposed periorbita, is carefully incised with a sickle knife, layer by layer, to allow for further decompression of orbital contents. These releasing incisions are made in a posterior to anterior direction so that herniating orbital fat will not obstruct visualization. Care should be taken not to bury the tip of the sickle knife, so as to prevent injury to the underlying orbital contents, including the enlarged medial and inferior rectus muscles. Nasal packing is not placed postoperatively to avoid placing pressure in the region of the orbital apex and optic nerve.

Table 1
Demographics for 10 patients who underwent 13 endoscopic revision orbital decompressions

Pt	Orbit	Age (y)/sex	Side	Indication	Months between surgeries	Initial surgery		Revision surgery	
						Approach	Walls removed	Approach	Walls removed
1	1	45M	R	ON	10	Endo	Med, Inf	ED, LD	Med, Lat
2	2	43M	L	GR	29	Comb	Med, Lat	ED	Med
3	3	60F	R	EK	5	Comb	Med, Lat	ED	Med
4	4	83M	R	EK	48	Ext	Med, Inf	ED, LD	Med, Lat
5	5	61F	L	EK	12	Ext	Med, Inf	ED	Med
6	6	39F	L	EK	0.25	Endo	Med	ED, FL	Med, Inf
7	7	55F	R	EK	24	Ext	Med, Inf	ED	Med
	8		L	EK	24	Ext	Med, Inf	ED	Med
8	9	59F	R	ON	60	Ext	Med, Lat, Inf	ED	Med
	10		L	ON	60	Ext	Med, Lat, Inf	ED	Med
9	11	50F	L	EK	36	Unk	Unknown	ED, LD	Med, Lat
10	12	50F	R	GR	55	Ext	Med, Lat, Inf	ED	Med
	13		L	EK	7	Ext	Med, Lat, Inf	ED	Med

EK, Exposure keratopathy; *Endo*, endoscopic; *Comb*, combined; *ED*, endoscopic decompression; *Ext*, external; *GR*, gaze restriction; *Inf*, inferior; *FL*, external lateral floor decompression; *Lat*, lateral; *LD*, external lateral decompression; *Med*, medial; *ON*, optic neuropathy; *Pt*, patient.

RESULTS

Demographics for the 10 patients who underwent revision decompression on 13 orbits are listed in Table 1. Indications for revision decompression included exposure keratopathy (n = 8, 62%), optic neuropathy (n = 3, 23%), and gaze restriction (n = 2, 15%) secondary to entrapment of enlarged orbital muscles within the bony opening that was created at the initial decompression. Previous primary decompressions were performed through external (n = 8, 62%), endoscopic (n = 2, 15%), combined (n = 2, 15%), or unknown (n = 1, 8%) approaches. Revision surgeries included endoscopic medial decompression alone (n = 9, 69%) and endoscopic medial decompression with concurrent external lateral wall or floor decompression (n = 4, 31%). The mean time interval between primary and revision surgeries was 28.5 months (range 0.25-60 months).

Patient demographics were found to be statistically comparable between the revision and primary decompression (control) cohorts (Table 2). There were no intraoperative complications in either group. All patients were discharged within 24 hours of surgery. Mean follow-up was 29.7 months (range 0.25-116 months) for the revision surgery group and 4.2 months (range 0.25-19 months) for the control group ($P = 0.16$).

Mean ocular recession was 1.4 mm less in revision decompression cases compared with controls (3.6 ± 1.0 mm vs 5.0 ± 2.1 mm, respectively), as shown in Table 3, although this difference was not found to be statistically significant ($P = 0.13$). Reduction in proptosis achieved by revision surgery was not dependent on whether the previous decompression had been performed by an endoscopic or external technique (3.3 ± 1.0 mm vs 4.0 ± 1.1 mm, respectively; $P = 0.34$), as shown in Table 4. Concurrent external lateral wall or floor decompression was performed at the time of revision surgery in four patients without a history of prior lateral decompression. There was also no significant difference in mean ocular recession for these patients who underwent endoscopic revision decompression with concurrent external lateral wall or floor decompression compared with patients who underwent endoscopic revision de-

Table 2
Patient demographics: Revision vs primary (control) decompression groups

	Revision	Primary	*P* value
No. of patients	10	10	
Age (y)			
Mean	54.5	43.1	0.11
Range	39-83	21-74	
Gender			
Male	3	3	1.0
Female	7	7	
Orbit			
Right	6	10	0.25
Left	7	6	
Surgical indication			
EK	8	12	0.40
ON	3	4	
GR	2	0	
Procedure			
ED	9	5	0.37
ED+LD or FL	4	11	

ED, Endoscopic decompression; *EK*, exposure keratopathy; *FL*, external lateral floor decompression; *GR*, gaze restriction; *LD*, external lateral decompression; *ON*, optic neuropathy.

Table 3
Mean ocular recession following endoscopic revision vs primary (control) decompression

Mean exophthalmometry reading (mm, range)	Revision (n = 8 orbits)	Primary (n = 16 orbits)	*P* value
Preop	27.6 ± 4.2 (23.0-36.0)	27.7 ± 2.3 (23.0-32.0)	0.91
Postop	24.0 ± 3.5 (21.0-31.0)	22.7 ± 2.4 (18.0-26.5)	0.32
Ocular recession (mm, 95% confidence interval)	3.6 ± 1.0 (2.8-4.5)	5.0 ± 2.1 (3.9-6.1)	0.13

Postop, Postoperative; *Preop,* preoperative.

compression alone (3.3 ± 1.2 mm vs 3.8 ± 1.0 mm, respectively; $P = 0.57$).

Exposure keratopathy improved in all cases in both the revision and primary groups. Preoperative diplopia improved, but did not resolve, in the two patients who had gaze restriction prior to revision surgery. Visual acuity improved one or more lines in 62 percent of patients in the revision decompression group, compared with 20 percent of patients in the primary decompression group ($P = 0.09$). No patient in the study had deterioration of vision. Postoperative visual acuity results were available in one of three patients who underwent revision orbital decompression for optic neuropathy. This patient's vision improved from finger count preoperatively to 20/100 postoperatively.

Complication rates were similar between revision decompression (n = 3) and control (n = 2) groups (38% vs 13%, respectively; $P = 0.17$), as shown in Table 5. One patient developed an ipsilateral frontal mucocele 8 years after revision decompression, which required endoscopic drainage. Another patient who underwent bilateral revision orbital decompressions developed chronic frontal headaches 17 months postoperatively. CT scan demonstrated bilateral frontal sinus opacification, which proved refractory to medical therapy, and she underwent frontal sinus obliteration 25 months after her revision decompression surgeries.

One patient in the control group developed acute sinusitis 1 week after orbital decompression, characterized as facial discomfort with mucopurulent drainage from the maxillary and ethmoid sinuses found on postoperative endoscopy. Symptoms resolved with a 10-day course of oral antibiotics. A second patient in the control group developed chronic sinusitis, characterized by facial pain for 5 months, and radiographic evidence of right frontal and sphenoid sinus opacification, which also responded to oral antibiotic therapy.

DISCUSSION

The clinical course of Graves' orbitopathy begins with an acute phase characterized by active inflammation lasting 6 to 18 months and progresses to a chronic phase associated with muscle fibrosis and a stabilization of the proptosis. Although it is preferable to perform orbital decompression during the chronic phase, in some patients, sight-threatening optic neuropathy necessitates surgery while the disease is still active. In such patients, orbital inflammation may progress after decompression resulting in a recurrence of ocular symptoms and the need for revision surgery.[9,10] In addition to such cases of recurrent orbitopathy, persistent disease is also an indication for revision decompression, particularly when insufficient bone has been removed at prior surgery to adequately relieve pressure on the orbital contents. In both cases, areas of residual bone or periorbita are sought and removed to further decompress the orbit.

Table 4
Effect of surgical approach on ocular recession attained from revision decompression

Exophthalmometry (mm; mean, range)	ED ± LD (n = 4 orbits)	External only (n = 4 orbits)	*P* value
Initial decompression			
Preop	25.8 ± 3.6 (23.0-31.0)	29.5 ± 4.4 (26.5-36.0)	0.24
Postop	22.5 ± 3.0 (21.0-27.0)	25.5 ± 3.7 (23.0-31.0)	0.25
Ocular recession (mm; 95% confidence interval)	3.3 ± 1.0 (1.7-4.8)	4.0 ± 1.1 (2.3-5.7)	0.34
Exophthalmometry (mm; mean, range)	**ED alone (n = 5 orbits)**	**ED + LD or FL (n = 3 orbits)**	***P* value**
Revision decompression			
Preop	28.0 ± 4.8 (24.0-36.0)	27.0 ± 4.0 (23.0-31.0)	0.77
Postop	24.2 ± 4.1 (21.0-31.0)	23.7 ± 3.1 (21.0-27.0)	0.85
Ocular recession (mm; 95% confidence interval)	3.8 ± 1.0 (2.5-5.1)	3.3 ± 1.2 (0.5-6.2)	0.57

ED, Endoscopic decompression; *LD,* external lateral decompression; *FL,* external floor decompression; *Postop,* postoperative; *Preop,* preoperative.

Table 5
Incidence of complications following revision vs primary (control) decompression

Complication	Revision (n = 8 orbits)	Primary (n = 16 orbits)	
Acute sinusitis	0% (n = 0)	6.3% (n = 1)	
Chronic sinusitis	25% (n = 2)	6.3% (n = 1)	
Frontal mucocele	12.5% (n = 1)	0% (n = 0)	
Total	37.5% (n = 3)	12.5% (n = 2)	$P = 0.17$

The present study is the first report to describe and evaluate the endoscopic technique for revision orbital decompression. Results suggest that it is both safe and effective for the treatment of patients with recurrent or persistent Graves' orbitopathy. There were no intraoperative complications, and all patients had brief hospital stays. Postoperative complications were limited to sinusitis, including frontal sinus mucocele. The authors believe that postoperative obstruction of the frontal sinus can be avoided by limiting bone removal and decompression in the region of the frontal recess. Although no orbital complications occurred in this study, revision decompression carries an inherently higher risk of injury to intraorbital structures compared with primary surgery, and increased care needs to be taken.

For patients with exposure keratopathy and proptosis, ocular recession is the primary goal of decompression surgery. In this study, the mean ocular recession of 3.6 mm in those who underwent endoscopic revision orbital decompression is consistent with results reported in the literature for endoscopic primary orbital decompression, ranging from 2.5 to 5.5 mm.[5-8,11] Although not statistically significant, there was a trend toward a lower mean reduction in proptosis in endoscopic revision decompression cases compared with controls. This finding may reflect the decreased quantity of residual bone and periorbita that can be removed in revision procedures. Increased orbital fibrosis and the inherent difficulty in operating in a previously manipulated surgical field may also be contributing factors. These factors may also explain why concurrent external procedures did not improve ocular recession for endoscopic revision cases in this study, whereas concurrent lateral wall removal has been shown to provide an additional 2 mm of reduction in proptosis in endoscopic primary decompression surgery.[7,8]

Endoscopic revision orbital decompression appears to be effective in improving compressive optic neuropathy and gaze restriction. More than half of patients who underwent revision decompression had improvement in visual acuity, and no patients in the study had deterioration of vision. Moreover, enlarging orbital muscles can become entrapped within the previous decompression site, causing impaired ocular motility and gaze restriction. By removing additional bone to enlarge the area of decompression and release the entrapped orbital muscles, revision decompression improved ocular motility in those patients who underwent revision surgery for gaze restriction.

One potential disadvantage of the endoscopic technique for revision orbital decompression is its limited access, allowing removal of only the medial orbital wall and floor. Because these walls are commonly addressed at the time of primary surgery, the amount of further decompression that can be obtained through a revision medial approach may be limited. Concurrent external procedures that address additional orbital walls can be performed if minimal decompression is attained intraoperatively through a medial approach; however, these procedures were not found to improve outcomes in this study. For patients who do not respond to revision decompression surgery, upper eyelid lengthening and eye muscle procedures can be considered. Another potential disadvantage of the endoscopic approach is visual obstruction caused by herniating orbital contents from prior medial decompression. This problem can usually be overcome by bluntly retracting the intranasal orbital contents laterally when positioning the endoscope to visualize the posterior nasal cavity.

Although statistically significant differences in mean ocular recession and incidence of postoperative sinusitis were not found between revision and primary groups, the small sample size limits the statistical power of this study. As with other procedures that are less commonly performed, the recruitment of a large number of patients for prospective study is neither practical nor feasible. Revision orbital surgery carries unique challenges that increase the difficulty and risks of this procedure. Nevertheless, the results of this preliminary study suggest that revision decompression can be performed without serious complications and can provide meaningful relief of symptoms of refractory Graves' orbitopathy. Additional multicenter trials with larger cohorts will be required to demonstrate with certainty if equivalency in clinical outcomes exists between endoscopic primary and revision orbital decompression.

CONCLUSION

Vision-threatening orbitopathy can recur or persist in patients with Graves' disease despite prior orbital decompression. The endoscopic technique for revision decompression can be performed safely and effectively for the treatment of refractory Graves' orbitopathy. Although no significant differences in clinical outcomes were identified between endoscopic revision and primary surgeries, additional studies with greater statistical power are necessary to demonstrate the equivalency of these procedures.

AUTHOR INFORMATION

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AUTHOR CONTRIBUTIONS

Man-Kit Leung, conception and design, data acquisition and analysis, drafting of article, final approval; **Michael Platt,** conception and design, revision of article, final approval; **Ralph Metson,** conception and design, data analysis, revision of article, final approval.

DISCLOSURES

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REFERENCES

1. Brent GA. Graves' disease. New Engl J Med 2008;358:2594–605.
2. Bartalena L, Baldeschi L, Dickinson AJ, et al. Consensus statement of the European group on Graves' orbitopathy (EUGOGO) on management of Graves' orbitopathy. Thyroid 2008;18:333–46.
3. Michel O, Bresgen K, Russmann W, et al. Endoscopically-controlled endonasal orbital decompression in malignant exophthalmos. Laryngorhinootologie 1991;70:656–62.
4. Kennedy DW, Goodstein ML, Miller NR, et al. Endoscopic transnasal orbital decompression. Arch Otolaryngol Head Neck Surg 1990;116:275–82.
5. Metson R, Dallow R, Shore J. Endoscopic orbital decompression. Laryngoscope 1994;103:950–7.
6. Metson R, Shore JW, Gliklich RE, et al. Endoscopic orbital decompression under local anesthesia. Otolaryngol Head Neck Surg 1995;113:661–7.
7. Metson R, Pletcher SD. Endoscopic orbital and optic nerve decompression. Otolaryngol Clin N Am 2006;39:551–61.
8. Pletcher SD, Sindwani R, Metson R. Endoscopic orbital and optic nerve decompression. Otolaryngol Clin N Am 2006;39:943–58.
9. Selva D, Chen C, King G. Late reactivation of thyroid orbitopathy. Clin Experiment Ophthalmol 2004;32:46–50.
10. Baldeschi L, Lupetti A, Phung V, et al. Reactivation of Graves' orbitopathy after rehabilitative orbital decompression. Ophthalmology 2007;114:1395–402.
11. Kasperbauer JL, Hinkley L. Endoscopic orbital decompression for Graves' orbitopathy. Am J Rhinol 2005;19:603–6.