Endoscopic Orbital Decompression for Graves' Ophthalmopathy*

Jaime Levy MD[1], Moshe Puterman MD[2], Tova Lifshitz MD[1], Mira Marcus MD[1], Avichai Segal MD[1] and Tova Monos MD[1]

Departments of [1]Ophthalmology and [2]Otolaryngology and Head & Neck Surgery, Soroka Medical Center and Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer Sheva, Israel

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Abstract

Background: In patients with Graves' ophthalmopathy, orbital decompression surgery is indicated for compressive optic neuropathy, severe corneal exposure, or for cosmetic deformity due to proptosis. Traditionally this has been performed through a transantral approach, but the associated complication rate is high. More recently, endoscopic orbital decompression has been performed successfully with significantly fewer postoperative complications.

Objective: To report our experience of endoscopic orbital decompression in patients with severe Graves' ophthalmopathy.

Methods: Three patients (five eyes) underwent endoscopic orbital decompression for Graves' ophthalmopathy at Soroka Medical Center between the years 2000 and 2002. The indications for surgery were compressive optic neuropathy in three eyes, severe corneal exposure in one eye, and severe proptosis not cosmetically acceptable for the patient in one case. An intranasal endoscopic approach with the removal of the medial orbital wall and medial part of the floor was performed.

Results: In all five eyes an average reduction of 5 mm in proptosis was achieved. Soon after surgery, visual acuity improved in the three cases with compressive optic neuropathy, and exposure keratopathy and cosmetic appearance also improved. The diplopia remained unchanged. No complications were observed postoperatively.

Conclusions: Endoscopic orbital decompression with removal of the medial orbital wall and medial part of the floor in the five reported eyes was an effective and safe procedure for treatment of severe Graves' ophthalmopathy. A close collaboration between ophthalmologists and otorhinolaryngologists skilled in endoscopic sinus surgery is crucial for the correct management of these patients.

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Thyroid eye disease, or Graves' disease, is an autoimmune disorder in which there is lymphocytic infiltration of the extraocular muscles and an accumulation of glycosaminoglycans in the orbital fat, leading to an increase in the volume of orbital tissue [1]. Approximately half of all patients with Graves' disease develop ophthalmopathy [2]. Given an average orbital volume of 26 ml, an increase of only 4 ml (16%) will result in 6 mm proptosis. When severe, the proptosis can result in significant visual consequences. The inability to close the eyelids may cause exposure keratopathy [3], asymmetrically impaired extraocular muscle function may lead to progressive diplopia [4], and compression of the optic nerve or its vasculature by the enlarged extraocular muscles may result in optic neuropathy. The optic neuropathy produces progressive loss of visual acuity, decreased color vision, and defects in the visual field, usually central scotomas [3]. The reported incidence of patients who develop compressive optic neuropathy is 8.6%.

The clinical course of ophthalmopathy in Graves' disease appears to be independent of the clinical course of thyroid disease [5] and treatment [6]. The condition can be divided into an early, evolving (inflammatory) phase and a late, stable phase. Exacerbations and remissions of inflammation within the orbit characterize the early phase, which typically lasts 6 to 18 months [1]. Systemic corticosteroids are useful in this phase. However, treatment may need to be continued for a long time, causing side effects. External beam radiation may be employed to limit the amount of proptosis and optic nerve compression. The orbital manifestations during the stable phase are generally unresponsive to steroids and radiotherapy. Surgery is undertaken during this phase, and includes strabismus repair, lid retraction surgery and orbital decompression.

When other therapeutic modalities fail, orbital decompression surgery is indicated for compressive optic neuropathy or severe corneal exposure. It is also used to minimize cosmetic deformity due to proptosis. Surgical decompression involves removing one to four of the bony orbital walls and incising the orbital periostium, allowing the swollen orbital contents to prolapse into the adjacent spaces. This can be achieved either externally or using an endoscopic approach. Since 1990, endoscopic orbital decompression has been found both effective and associated with a low morbidity [7].

Patients and Methods

Three patients (five eyes) underwent endoscopic orbital decompression at Soroka Medical Center between 2000 and 2002. The patients, one man and two women, had an age range of 44 to 50 years. The indications for surgery were compressive optic neuropathy in three eyes, severe corneal exposure keratopathy in one eye, and significant proptosis in one eye causing a cosmetic problem.

Preoperative assessment comprised a complete ophthalmologic and orthoptic assessment, including visual acuity, visual fields, slit-lamp examination of anterior and posterior segments, intraocular pressure measurements, and Hertel exophthalmometry to assess the degree of exophthalmos and ocular motility and complaints of diplopia. Preoperative and postoperative photographs were obtained. Otolaryngologic assessment included nasal endoscopy to identify any sinus or septal pathology.

A diagnosis of compressive optic neuropathy was made based

* The authors have no proprietary interest in any of the materials or techniques used in this study.

on the following findings: decrease in visual acuity not explained by the refractive state or anterior segment findings, defective visual fields with no prior record of glaucoma or other medical history, presence of optic disc congestion, radiographic confirmation of compressive neuropathy, acquired dyschromatopsia, and afferent pupillary defect. Computed tomography scans of the orbits and sinuses were obtained in both axial and coronal planes to assess the anatomy, demonstrate any covert sinus pathology, and confirm the classical features of thyroid eye disease.

The surgical technique has been reported previously [7,8]. The aim is to remove the entire medial wall and part of the floor of the orbit so that the orbital contents can prolapse into the ethmoidal and maxillary cavities. All the procedures were performed under general anesthesia, with the patient in a supine position and the head slightly elevated. Surgery was done by experienced ear, nose and throat (M.P.) and oculoplastic surgeons (T.M.). The nasal cavity was instilled with a mixture of 2 ml of 10% cocaine and 1 ml mixture of 1:1,000 adrenaline solution followed by infiltration of the lateral nasal wall with a mixture of 1:80,000 adrenaline and 2% xylocaine. A standard endoscopic sphenoethmoidectomy was performed. The medial wall of the orbit was carefully skeletonized and fully exposed as far back as the orbital apex. A generous middle meatal antrostomy was performed for access to the orbital floor. The thin bone of the medial orbital wall (lamina papyracea) was removed after careful separation from the periorbita with a Freer elevator. Bony decompression of the orbital floor was extended up to the infraorbital nerve canal to avoid damaging the nerve. Because surgical access to the orbital floor through the middle meatal opening is restricted, only the medial 0.5–1 cm of the bone was removed. Excessive decompression of the orbital floor may lead to postoperative diplopia or aggravate a preexisting diplopia, and increases the risk of recurrent maxillary sinusitis [9]. Care was taken to avoid penetrating the periorbita prior to completing all the bone work, since premature prolapse of orbital fat may obscure the surgical field. Once the periorbita was fully exposed, multiple full-thickness parallel linear incisions were made with a sickle knife in a posterior-anterior direction. This resulted in herniation of the orbital fat into the ethmoidal and maxillary cavities. The immediate extent of decompression was assessed by gently palpating the orbit and endoscopically viewing the transmission of the palpations to the herniated orbital contents. In cases in which decompression of the optic nerve was required, the thick bone over the orbital apex was first thinned down with a drill and then removed with a Freer elevator. At the end of the operation, a Merocel sponge in a rubber finger-cot was coated with antibiotic ointment and placed lateral to the middle turbinate. The patient was maintained on oral amoxicillin-clavulanic acid 500 mg 3 times a day for 5 days postoperatively. The sponge was removed the following day and the

Table 1. Preoperative and postoperative clinical findings

	Patient 1 (male, 50 yrs)		Patient 2 (female, 47 yrs)		Patient 3 (female, 44 yrs)	
	Right eye	**Left eye**	**Right eye**	**Left eye**	**Right eye**	**Left eye**
Procedure	E	E	E	E	E	No
Indication for surgery	CON	EK	CON	CON	Cosmesis	
Hertel (mm)						
Preoperative	35	32	24	25	28	23
Postoperative	29	26	19	19	25	
Differential	6	6	5	5	3	
Visual acuity						
Preoperative	6/36	6/15	6/36	6/12	6/36*	
Postoperative	6/9	6/9	6/6	6/9	6/36	
Exposure keratopathy						
Preoperative	Severe	Severe	Mild	Mild	Mild	
Postoperative	No	No	No	No	No	
Diplopia						
Preoperative	Yes		Yes		Yes	
Postoperative	Yes		Yes		Yes	
IOP (mmHg)						
Preoperative	23	21	27	24	18	
Postoperative	12	12	18	17	15	
Ocular motility	Stable	Stable	Stable	Stable	Stable	
Optic disk	Normal appearance before and after the operation					
Visual field						
Preoperative	Inferior defect	Normal	Inferior defect	Normal	Normal	
Postoperative	Normal	Normal	Normal	Normal	Normal	
Complications	No	No	No	No	No	
Follow-up (mos)	22	24	24	24	36	

* Low vision due to high myopia.
CON = compressive optic neuropathy, EK = exposure keratopathy, IOP = intraocular pressure

maxillary sinus was suctioned free of blood under endoscopic visualization. The patients were advised not to blow their nose for a week.

Results

The results of preoperative and postoperative eye examinations are shown in Table 1. Endoscopic orbital decompression was performed in five eyes (three patients). The follow-up period was at least 22 months. In all five eyes the proptosis was reduced by 3 to 6 mm (average 5 mm). In the three eyes in which surgery was performed because of compressive optic neuropathy, visual acuity improved immediately after surgery. Exposure keratopathy improved in all five eyes during the first week after surgery. Preoperative diplopia was present in all eyes. Patient 1 underwent extraocular muscle surgery for correction of diplopia 3 months after the orbital decompression and had diplopia only on far lateral gaze and in upgaze. After decompression, the diplopia in the other two patients remained unchanged with no change in ocular motility. The patient whose surgery was performed for cosmetic reasons was pleased with her postoperative appearance. No complications were observed postoperatively.

Discussion

Surgical decompression for severe thyroid eye disease has been performed for almost a century. In 1911, Dollinger [10] performed a lateral wall decompression, however the extent of decompression provided was minimal. Naffziger [11] developed a superior decompression of the orbital contents into the anterior cranial fossa. This approach requires a craniotomy, with consequent risks of meningitis and leakage of cerebrospinal fluid. Consequently, Sewall [12] described a medial orbital wall decompression through an external ethmoidectomy approach. Hirsch [13] was the first to employ a Caldwell-Luc approach to obtain decompression by removal of the orbital floor. In 1957, Walsh and Ogura [14] extended this approach to include a medial wall decompression. With this procedure, an average of 4–6 mm reduction in proptosis can be obtained. But this approach requires a generous antrotomy and is associated with the morbidity of a Caldwell-Luc procedure. The postoperative morbidity includes considerable swelling and discomfort immediately following surgery, persistent numbness over the maxillary region after division of the anterior-superior alveolar nerves, acute sinusitis and oroantral fistula [15].

Endoscopic decompression for thyroid eye disease has been performed since 1990 [7]. With improved technology in fiber optic instrumentation, intranasal surgery on orbital structures is safe and effective. The endoscopic approach obviously avoids an external scar, does not disrupt the attachment of the inferior oblique muscle or medial canthal ligament, is associated with a lower incidence of postoperative complications, and avoids damage to the nasolacrimal duct and infraorbital nerve as identification of the nerve is possible and it is not traumatized.

In the five eyes reported here, the average reduction in proptosis was 5 mm (range 3–6 mm). Similar results were previously reported. Kennedy et al. [7] achieved a reduction in proptosis by an average of 4.7 mm (range 3–5 mm) in five orbits; however, two of their procedures involved an exploratory Caldwell-Luc antrotomy with additional decompression of the orbital floor. Lund and co-workers [16] achieved a similar reduction in proptosis, irrespective of whether an external ethmoidectomy or an endoscopic approach was utilized. The endoscopic approach was reported to be associated with fewer complications. Asaria et al. [8] reported a reduction in proptosis by an average of 3.79 mm (range 2–7 mm) when performing removal of the medial orbital wall and part of the floor.

Visual acuity improved in four eyes after surgery; the eye with no change in vision after the surgical procedure had macular changes. These results are similar to the previously reported rates [7,8]. Exposure keratopathy usually disappears after the surgical procedure [7]. In our series it resolved in all five eyes. In the three eyes in which surgery was performed because of compressive optic neuropathy, visual acuity improved immediately after surgery. This finding is similar to the reported improvement in visual acuity with different endoscopic techniques [17–20].

Controversy exists regarding the incidence of postoperative diplopia and worsening of preexisting diplopia. In the swollen orbit there is usually considerable restriction of eye movement in all directions. When the external restriction of proptosis is relieved by decompression, the less affected muscles are able to function, but the more swollen and fibrotic muscles (most commonly the medial rectus, inferior rectus, or both) are not [7]. The net result is an asymmetric limitation of eye movement instead of the preexisting symmetric limitation. In our series, none of the patients had worsening of the diplopia, which is similar to a previous report with the same technique [8]. The diplopia is almost always horizontal because of the predominant myopathy of the medial rectus. Strabismus has been successfully repaired by recession of both medial rectus muscles in the same surgical session at the end of the orbital decompression or several months after the procedure [21].

A variety of surgical techniques has been described for reducing the incidence of postoperative diplopia or for improving postoperative results, including balanced endoscopic decompression to the medial and lateral orbital walls [17,18], combined transconjunctival and transnasal endoscopic approaches [19,20], preservation of a portion of periorbita to prevent prolapse of the medial rectus muscle [22], fat removal [23,24], and balanced medial and lateral wall decompression with fat removal without removing the orbital floor [25]. The advantages and limitations of all these techniques are beyond the scope of this work. The choice of procedure depends principally on the experience of the surgical team, the indication for surgery (i.e., the orbital sling technique is not appropriate in cases of optic neuropathy in which maximal decompression in the region of the orbital apex is necessary), and anatomic variations. In the hands of each surgeon, the technique he or she is familiar with will be the most satisfactory. As in other areas of medicine, when there are so many therapeutic options none is much better than the other.

All the endoscopic techniques share a low rate of postoperative complications, allow a maximal posterior orbital decompression at the orbital apex (an area not fully accessible through the transantral routes), precisely and safely remove the structures, and are not

associated with postoperative dysesthesia of the infraorbital nerve or incision-related cosmetic problems. With the surgical technique that we used, including ablation of the lamina papyracea up to its posterior insertion and the medial part of the inferior orbital wall, a greater volume of periorbital tissue prolapse into the nasal cavities is achieved, thus relieving the intraorbital pressure in severe cases.

There are a few limitations to this study. The first is the small number of eyes operated on and the consequent difficulty in drawing conclusions, although our results are similar to the larger series previously reported. Second is the lack of comparison with other endoscopic techniques. As we commented before, a considerable number of techniques has been described. No results are consistently better than others and no large randomized studies comparing different procedures have yet been reported.

In conclusion, severe Graves' ophthalmopathy requiring orbital decompression can be safely managed by an endoscopic approach with removal of the medial orbital wall and medial part of the floor. A close collaboration between ophthalmologists and otorhinolaryngologists skilled in endoscopic sinus surgery is crucial for the correct management of these patients.

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Correspondence: Dr. J. Levy, Dept. of Ophthalmology, Soroka Medical Center, P.O. Box 151, Beer Sheva 84101, Israel.
Phone: (972-8) 640-0379
Fax: (972-8) 640-3927
email: ljaime@bgu.ac.il

Capsule

Autophagy and Parkinson's disease

The cause of Parkinson's disease, the second most common neurodegenerative disorder, remains unknown. It is widely suspected that Lewy bodies, the intraneuronal signature of the disease, and perhaps neuronal death, result from aberrant degradation of synuclein, a protein that is known to play a role in the pathogenesis of Parkinson's disease. Cuervo et al. show that wild-type synuclein is degraded in lysosomes by chaperone-mediated autophagy. In contrast, the pathogenic synuclein mutants are not degraded, and actually block chaperone-mediated autophagy. This finding may explain the basis by which mutant synucleins cause familial Parkinson's disease.

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E. Israeli