TRANSNASAL ENDOSCOPIC ORBITAL DECOMPRESSION IN GRAVES' OPHTHALMOPATHY

Mohammad-Hossein Baradaranfar MD*, Payman Dabirmoghaddam MD

Graves' ophthalmopathy can cause proptosis, cosmetic problems, and visual impairment. The aim of this study was to assess the validity and limitation of transnasal endoscopic orbital decompression in Graves' ophthalmopathy. Between 1997 and 2003, 20 female and one male patients with Graves' ophthalmopathy underwent simultaneous bilateral transnasal endoscopic orbital decompression. All patients were nonresponders to medical management (including steroid therapy) but none of them received orbital irradiation. Preoperative and postoperative ophthalmologic examinations were recorded for all patients. In addition, photographs and CT scans of orbit and sinuses were done. After operation, visual acuity improved following orbital decompression with this technique. Diplopia in lateral gaze developed in 15 patients but only one of them required corrective surgery. The mean retrodisplacement was 4.1 mm that compares favorably with other series. After an average follow-up of 49 months, none of the patients developed sinusitis or mucocele. Endoscopic decompression provides excellent visualization without external incisions, and facilitates maximal decompression without the increased risk of hemorrhage, visual impairment, or infections.

Keywords • endoscopic surgery • Graves' ophthalmopathy • orbital decompression

Introduction

Graves' disease is a multisystem disorder and Graves' ophthalmopathy is one of the major complications. Depending on the diagnostic criteria used, the prevalence of Graves' orbitopathy is between 10% to 45%. However, the most severe form of this disease, with optic nerve involvement and visual impairment, occurs in only 2% to 5% of patients.¹

Although the muscles are enlarged on computed tomography (CT) scan, the myocytes themselves appear fairly normal histopathologically.² Formerly, decreased circulating suppressor T-cells and increased circulating autoantibodies against extraocular muscles were proposed as the mechanism of disease but this autoantibodies proved to be neither tissue nor disease specific.³

It seems that the retrobulbar fibroblasts are playing a key role in the pathogenesis of Graves ophthalmopathy. They secrete glycosaminoglycans which is the hallmark of Graves' ophthalmopathy. Furthermore, these cells can produce major histocompatibility complex class II, heat shock protein, and lymphocyte adhesion molecules.⁴ In addition, in a majority of patients with Graves' ophthalmopathy, autoantibodies against fibroblast antigen have been found. It has been suggested that these cells act as target and effector cells in the Graves' ophthalmopathy.⁵

The inferior rectus muscle is the most commonly involved followed by the medial rectus, superior rectus-levator complex, and lateral rectus.⁶ The physical examination reveals lid edema, retraction, chemosis, restricted extraocular motility, and proptosis.³

Although proptosis can be measured with various instruments (e.g., Hertle exophthalmometer), medical imaging is another way to provide objective measurements of proptosis. CT scan, furthermore, can demonstrate enlargement of
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extraocular muscles, crowding of the orbital apex, optic nerve swelling, and anatomical variation of the bony framework of the ethmoid sinuses.\textsuperscript{7}

For soft tissue changes and to demonstrate the direct compression of the optic nerve at the orbital apex, magnetic resonance imaging (MRI) with its \textit{T\textsubscript{2}} weighted sequence is more appropriate.\textsuperscript{8}

Orbital decompression is indicated for patients who are resistant to medical treatment. Surgical decompression is still the primary therapy for sight threatening Graves’ disease and is the most effective procedure for compressive optic neuropathy at the level of the orbital apex. By expanding the orbital confines it reduces intraorbital pressure, corneal exposure, proptosis, and optic nerve compression.\textsuperscript{3}

Historically, in 1911 Dollinger performed the first lateral orbitotomy for exophthalmos and subsequently other approaches were described by Naffziger, Sewall, and Hirsch, but the most popular surgical procedure was described by Walsh and Ogura in 1957.\textsuperscript{3} They proposed a technique for decompressing the orbital contents transantrally into the maxillary sinus and the ethmoid space.\textsuperscript{3} In 1990, Kennedy et al, first proposed to perform the Ogura technique transnasally under endoscopic guidance.\textsuperscript{9} The endoscopic approach allows for complete medial orbital wall decompression with excellent visualization of the key landmarks.

Here we describe the technique and results of endoscopic orbital decompression.

\textbf{Patients and Methods}

Between 1997 and 2003, twenty-one patients underwent endoscopic transnasal orbital decompression for Graves’ ophthalmopathy (Figure 1). Surgical decompression was performed by the senior author in all cases. Twenty patients were females and one patient was male with a mean age of 45 years. Only the patients who were resistant to medical therapy were selected and none of them received radiotherapy. Exposure keratitis was found in all of the patients who were and optic neuropathy in one of them.

Ophthalmologic examinations (visual field, visual acuity, fundoscopy, and exophthalmometry with Hurtle exophthalmometer) were done before and after surgery by the ophthalmologist. CT scan in axial and coronal planes were performed before surgery to determine the exact anatomy of sinuses and position of the lamina papyracea (Figure 2).

All patients underwent bilateral simultaneous endoscopic decompression under general anesthesia. After the induction of general anesthesia, cotton pledges soaked with 1:5,000 epinephrine were placed in nasal cavity for vasoconstriction. The nasal mucosa was injected with 1:200,000 epinephrine anterior to the middle turbinate and the mucosa on the lacrimal bone. The surgical instrumentation included a 4 mm endoscope with 0\textdegree and 30\textdegree angles, dissectors, suction elevators, and back biting forceps.

First, anterior and posterior bilateral ethmoidectomies were preformed, and then the frontal recess was completely opened and the mucosa of ethmoid cells were completely removed.

A wide middle meatal antrostomy was done and the mucosa of maxillary sinus was removed. The anterior wall of sphenoid sinus was removed to prevent possibility of its closure after extrusion of orbital contents. Lamina papyracea was carefully outlined and infraorbital canal—the lateral margin of decompression—was exactly determined. Then lamina papyracea was completely removed with the medial half of orbital floor. Using a sickle knife, the orbital periosteum was widely opened posteroanteriorly to allow orbital contents to
prolapse into the ethmoid and maxillary sinuses (Figure 3). The patients were observed overnight and were then discharged the next morning on oral antibiotic for 1 week (Figure 4A and 4B).

Results

Decompressions were performed for severe proptosis and exposure keratitis in 20 patients and for severe proptosis, exposure keratitis, and optic neuropathy in another patient. CT scans showed enlargement of the extraocular muscles in all patients. The average follow-up time was 49 months (range, 5 – 64 months). In all cases the visual acuity was improved following orbital decompression with this technique. Prior to surgery, the mean visual acuity was 20/40 in 20 patients, and in the remaining one with left eye optic neuropathy, the visual acuity was hand motion in the left eye and 20/80 in the right eye. The mean visual acuity was raised to 20/30 in 20 patients and in the remaining patient, with optic neuropathy, to 20/160 in the left eye and to 20/60 in the right eye after surgery (Table 1).

The mean preoperative exophthalmometric measurement was 24.2 mm which was reduced to 20.1 mm after surgery. Hurtle exophthalmometry recorded a mean retrodisplacement of 4.1 mm (Figure 4). Prior to surgery no patient had diplopia although, four cases had extraocular muscle dysfunction. After surgery, 14 patients acquired mild diplopia in lateral gaze that was improved several months later. Another patient acquired moderate diplopia that required corrective surgery.

Fundoscopy was normal in 20 patients and in another patient with left eye optic neuropathy, there were 2° disc pallor and 2° afferent pupillary defect in the left eye and normal fundoscopy in the right eye before surgery.

After surgery 20 patients had normal fundoscopy. In another patient with left eye optic neuropathy, there was 2° disc pallor without afferent pupillary defect; the disc pallor remained after operation but the right eye fundoscopy was normal (Table 1).

Knowledge of pathophysiology of the disease is essential for treating Graves’ ophthalmopathy. The disease is characterized by accumulation of hydrophilic mucopolysaccharides and immune complexes in extraocular muscles and retrobulbar fat. This in turn is due to limited orbital space, increase in intraocular pressure, limitation of extraocular muscle movements, diplopia, and finally exophthalmos and complications such as exposure keratitis, neuropathy, and blindness.

Indications of surgery are: 1) Optic neuropathy not responding to medical therapy, patient not tolerating steroids, or the disease recurs after stopping medications. 2) Progressive keratopathy. 3) Cosmetic reasons.

Discussion

Although cosmetic disfigurement has been cited as a surgical indication alone, we performed decompression primarily for exposure keratitis or optic neuropathy. Endoscopic decompression achieved an average retrodisplacement of 4.1 mm. This compares favorably with findings of other

Figure 3. Postoperative coronal CT scan.

Figure 4 (A and B). Postoperative pictures of a patient.
series including those of Rizk et al who reported an average of 4.8 mm of recession with combined (endoscopic and Caldwell-Luc) approach or those of Kennedy et al who achieved 4.7 mm of retrodisplacement with the intranasal approach alone. Endoscopic approach provided excellent visualization of the lamina papyracea posteriorly to the apex. The only limitation of this method is inability to remove the lateral wall and the anterior half of floor of orbit. So in patients who need extensive decompression, combined transnasal and Caldwell-Luc approaches are used.

In our series none of the patients required further surgery for asymmetric results. Such a good result was also achieved by Kennedy et al. In their study, gaze-induced diplopia developed in 15 patients after surgery but only one of them required corrective surgery. Other series also have reported an increase in diplopia requiring muscle surgery.

It has been theorized that diplopia may increase because of the release of orbital pressure that unmasks latent muscle dysfunction and intraoperative muscular trauma. Those patients with persistent diplopia after surgery will undergo muscle surgery, three or more months after the decompression surgery. Normal variations in anatomy of sphenoid sinus and its relations to carotid artery and optic nerve are very important and it is best to do a CT scan before surgery to prevent major complications. Graves’ ophthalmopathy is an autoimmune reaction causing concentration of glycosaminoglycans in orbital content.

Decompression of orbit could be done in patients who are resistant to medical therapy, through transnasal route, by endoscopic guidance, and it can relieve all symptoms except extraocular muscle dysfunction. Our experience in 21 patients in whom bilateral simultaneous decompressions were done, showed a decrease in proposis and an improvement in cosmetic appearance comparable to other studies.

Concerning functions, visual acuity was improved in all patients, and in the patient with optic neuropathy, due to compression, it was considerable. All patients with Graves’ ophthalmopathy should receive routine eye examination and if any functional impairment is noted they should receive aggressive medical therapy. If no improvement occurs, decompression of orbit should be planned. If there is a contraindication to surgery, radiotherapy is recommended. All the patients who are operated by orbital decompression should be informed on the possibility of postoperative diplopia.

Endoscopic decompression provides excellent visualization without external incisions, and facilitates maximal decompression without the increased risk of hemorrhage, visual impairment, or infections.

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