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Endonasal Endoscopic Orbital Decompression in Patients with Graves' Ophthalmopathy

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Aim. To present the results of endonasal endoscopic orbital decompression in patients with Graves' ophthalmopathy.

Methods. Endonasal endoscopic orbital decompression was performed in 32 orbits of 21 patients with Graves' ophthalmopathy. In 17 patients the surgery was performed because of active ophthalmopathy non-responsive to conservative treatment, and in 4 patients for esthetic reasons. Preoperative and postoperative examination included visual acuity, examination of the eyelids and cornea, ocular motility, cover testing, Hertel exophthalmometry, and applanation tonometry.

Results. Visual acuity improved from preoperative 0.81 ± 0.28 (mean $\pm$ standard deviation) to postoperative 0.92 ± 0.21 ($p = 0.0032$, Student t-test). Retraction of upper and lower eyelids, as well as exposure keratitis, was reduced after operation ($p < 0.001$). Mean proptosis reduction in all orbits was 4.6 ± 1.7 mm ($p < 0.001$). An average reduction of intraocular pressure was 3.4 ± 3.0 mmHg ($p < 0.001$). New-onset diplopia developed in 8 patients. Diplopia persisted in 9 out of 11 patients who had preoperative diplopia. Two patients experienced postoperative relief of diplopia. Ocular motility was subsequently corrected by eye muscle surgery in 13 eyes, whereas prisms were used in other 5 manifestly strabic eyes.

Conclusions. Endonasal endoscopic orbital decompression procedure improved visual acuity, decreased proptosis and intraocular pressure, and also had favorable cosmetic results in most patients. Post decompression diplopia and strabismus were successfully managed by either eye muscle surgery or application of prisms.

Key words: *decompression, surgical; diplopia; exophthalmos; Graves' disease; intraocular pressure; visual acuity*

Graves' ophthalmopathy or thyroid eye disease is an immune disorder resulting in the inflammation of extraocular muscles and inflammatory cellular infiltration of the interstitial tissues, orbital fat, and lachrymal glands, which increase the volume of the orbital contents. This brings about the increase in the intraorbital pressure, with further fluid retention within the orbit. These changes lead to the characteristic clinical features including proptosis, eyelid retraction, and restrictive myopathy (1,2). Severe soft tissue involvement, with exposure keratitis and compressive optic neuropathy, present the most serious signs of disease (1,2).

Whereas the majority of patients has mild ophthalmopathy and requires only local supportive measures to control slight eye manifestations, the minority of patients with severe disease (3-5%) needs aggressive treatments that usually require a multidisciplinary approach (3,4). The three main forms of treatment are corticosteroids, retro bulbar radiotherapy, and orbital decompression. New treatments, such as anti-inflammatory and immunosuppressive drugs (somatostatin analogs and antioxidants) are also under evaluation (5,6). The choice of treatment mainly depends on disease activity, according to the clinical criteria proposed by Mourits et al (7), which is thought to be an important determinant in proper selection of patients for treatment.

Orbital decompression as a treatment of Graves' ophthalmopathy has traditionally been indicated for patients with exposure keratitis, compressive optic neuropathy and/or severe orbital inflammation with pain (4). In recent years, an increasing number of patients with disfiguring proptosis are being surgically treated for esthetic reasons (8). Since 1911, when Dollinger (9) first described surgical orbital decompression, many different techniques and approaches have been described, including one-, two-, and three-wall decompressions with orbital fat removal (9-12). Each of them results in a more or less successful reduction of the proptosis and improvement of visual acuity, but the mayor disadvantage is the risk of postoperative motility imbalance and diplopia.

The aim of our study was to review the results of endonasal endoscopic orbital decompression in 21 patients with Graves' ophthalmopathy, treated surgically either because of active ophthalmopathy or esthetic reasons.

Patients and Methods

Patients

Between January 1998 and December 2003, endonasal endoscopic orbital decompression was performed on 32 orbits of 21 patients with Graves' ophthalmopathy at the ENT Department of the Zagreb University Hospital Center (Tables 1 and 2). Eleven patients were operated bilaterally and 10 unilaterally. All patients had been previously examined at the Department of Ophthalmology and had received conservative treatment with corticosteroids (60-100 mg prednisone/day orally as the initial dose with slow tapering over a long period of time; some patients were treated with retrobulbar injections), botulinum toxin injection, radiotherapy (20 Gy, administered in 10 low doses), or a combination of these treatments. The ophthalmologist recommended endonasal endoscopic orbital decompression in 17 patients because of active ophthalmopathy with rapidly decreasing vision caused by compressive optic neuropathy and/or exposure keratitis or with other severe symptoms not responding to conservative treatment, and in 4 patients for esthetic reasons when the ophthalmopathy was stable and inactive. A diagnosis of compressive optic neuropathy was made according to the following findings: decrease in visual acuity not explained by the refractive state or anterior segment findings, defective visual fields in Goldmann perimetry with no prior record of glaucoma, neurological disease or other medical history, or presence of optic disc congestion. Anterior segment signs included superficial punctate keratitis, superior limbic keratoconjunctivitis, conjunctival injection and/or conjunctival chemosis. In patients with severe proptosis and eyelid retraction, corneal infiltration and ulceration were also found.

Table 1. Characteristics of 21 patients (32 orbits) with Graves' ophthalmopathy undergoing endonasal endoscopic orbital decompression

Parameter	No. (%) of patients
Men/women	8/13
Age (years, median, range)	44 (30-73)
Preoperative treatment:	
corticosteroids systemically	17
corticosteroids locally (retrobulbar)*	20
Botulinum toxin*	23
radiotherapy*	11

*No. of eyes.

Table 2. Clinical characteristics of 21 patients (32 orbits) with Graves' ophthalmopathy before and after endonasal endoscopic orbital decompression

Parameter	Decompression before	Decompression after	p
Best-corrected Snellen visual acuity (mean ± SD)	0.81 ± 0.28	0.92 ± 0.21	0.0032*
Retraction of eyelids (No. of eyes)			
upper	30	17	< 0.001†
lower	24	14	< 0.001†
Exposure keratitis (No. of eyes)	20	9	< 0.001†
Hertel exophtalmometry (mm, mean ± SD)	23.7 ± 2.4	19.0 ± 2.6	< 0.001*
Intraocular pressure (mmHg, mean ± SD)	19.4 ± 3.4	16.2 ± 3.0	< 0.001*
Diplopia in primary position (No. of patients)	11	17	0.009†
Ocular motility disturbance (No. of eyes)	28	24	0.030†

*Student's t-test.
†Chi-square test.

Operative Technique

Endonasal endoscopic orbital decompression consists of the removal of ethmoid chambers, taking out the medial orbital wall, and performing several parallel and horizontal incisions of the periorbita allowing the orbital contents to prolapse into the sinus cavities. This enlarges the orbital cavity on account of ethmoidal sinuses. The operation is performed under general hypotensive anesthesia, with infiltration of the operating field with the 1% xylocaine solution containing epinephrine (1:200, 000) to produce additional vasoconstriction and dry operating field. When removing the medial orbital wall, one must pay attention to be as precise and radical as possible, particularly in the upwards direction (skull base), inferiorly down to the thick part of the bony frame of the maxillary sinus, ie, the lowest borderline of the orbital floor, anteriorly up to the insertion of the uncinate processus, and posteriorly to the common tendinous ring, which always designates the end of the orbital apex and the beginning of the optic nerve canal. In most cases, we routinely tried to approach the orbital floor, ie, perform large middle antrostomy up to the infraorbital nerve, avoiding its damage at any cost. In the vast majority of cases we did not remove a longitudinal piece of the thick maxillary bone between the medial orbital wall and the orbital floor, since we realized that six months or later after the procedure the globe can drop down because of lack of the bonny support, resulting in late vertical diplopia, which is rather difficult to restore. The removal of the bony parts begins usually from the middle parts of the described area, proceeds backwards towards the common tendinous ring, continues upwards to the anterior skull base and then downwards to the thick longitudinal bone, finishing with the most anterior parts around the insertion of the uncinate processus. Once the whole medial part of the periorbital area is widely exposed and free of all bony fragments, a series of horizontal incisions are performed, starting from the upper and deepest parts. While making incisions, the globe must be gently pressed from outside. After finishing all planned incisions, the globe must be pressed more radically, just to enable the "delivery" of the retrobulbar fat, ie to move retrobulbar contents from the orbital cavity to the new space of the ethmoidal sinus.

Outcome Measures

Preoperative and postoperative examinations, which included best-corrected Snellen visual acuity, examination of the eyelids and cornea, ocular motility, cover testing, Hertel exophthalmometry and applanation tonometry, were performed at the Department of Ophthalmology. Eyelid retraction was measured in mm, in the primary eye position and without accommodation. As the upper eyelid margin normally rests at or 2 mm below the superior corneal limbus, eyelid retraction was considered when the margin was above the limbus, allowing sclera to be visible ("scleral show"). Likewise, the lower eyelid normally rests at the inferior corneal limbus; retraction was considered when sclera showed below the limbus. Diplopia was not recorded in serious orbitopathies because the patients were not conscious of the disturbance of the eye muscles. Development of diplopia after orbital decompression in such cases (new-onset diplopia) was thus a sign of improvement.

Statistical Analysis

Statistical analysis was performed by using Microsoft Excel Program for Windows XP. Values were reported as mean ± standard deviation (SD) or median and range. Comparisons between the pre- and post-treatment values were made by the Student's t-test. Frequencies were tested with the chi-square test. Differences were considered significant at the $p < 0.01$ level.

Results

The median duration of follow-up in our study was 12.5 months (range, 3-38).

Treatment

Best-corrected Snellen visual acuity improved from preoperative 0.81 ± 0.28 to 0.92 ± 0.21 postoperatively in all patients ($p = 0.0032$, Table 2). A single eye experienced a decrease in vision after surgery, due to cataract, ie, the vision was not decreased as a result of the surgical procedure.

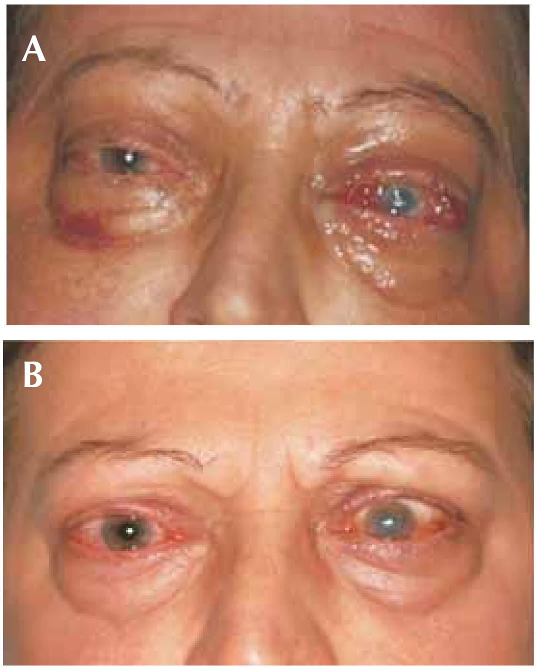

Figure 1. A patient with severe active Graves' ophthalmopathy with neuropathy who underwent urgent bilateral orbital decompression. **A.** Preoperative photograph of the patient. **B.** Postoperative photograph of the patient 6 weeks after bilateral orbital decompression with residual corneal opacities.

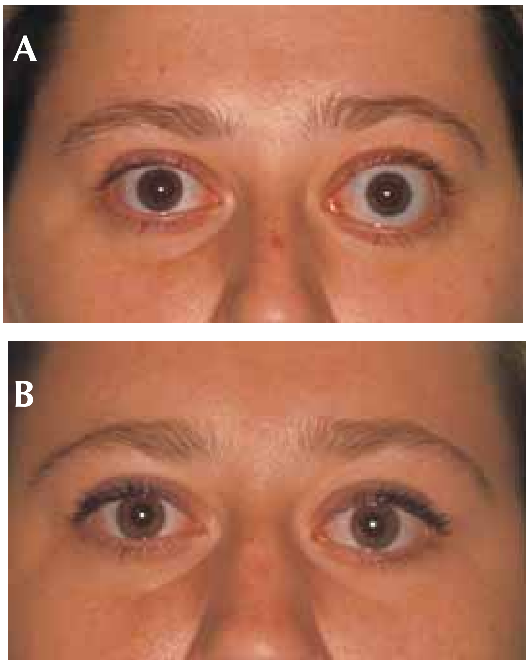

Figure 2. A patient with inactive Graves' ophthalmopathy who underwent left-side orbital decompression. **A.** Preoperative photograph of the patient. **B.** Postoperative photograph of the same patient one month after left-side orbital decompression.

Retraction of the upper and lower eyelids, as well as exposure keratitis, was reduced after operation ($p < 0.001$, Table 2). Proptosis decreased in all orbits after the decompression, with a mean decrease of 4.6 ± 1.7 mm, ranging from 2 up to 8 mm ($p < 0.001$). The preoperative mean Hertel exophthalmometry value was 23.7 ± 2.4 mm and the mean postoperative value was 19.0 ± 2.6 mm (Table 2). Urgent bilateral orbital decompression in a patient with severe active Graves' ophthalmopathy (Fig. 1) yielded good results, as well as left-side orbital decompression performed in a patient with inactive Graves' ophthalmopathy for esthetic reasons (Fig. 2).

Intraocular pressure decreased by a mean of 3.4 ± 3.0 mmHg, from preoperative 19.4 ± 3.4 to postoperative 16.2 ± 3.0 mmHg ($p < 0.001$, Table 2).

Complications and Side Effects

Of the 21 patients who underwent orbital decompression, 11 had diplopia in the primary position before surgery. In two latter patients, decompression resolved the diplopia, and in 9 the diplopia persisted. New-onset diplopia developed in 8 patients (Table 2).

Ocular motility disturbance was found in the majority of patients (28 of 32 eyes) before surgery. In 18 of these eyes, the motility worsened after the operation, in 6 it remained unchanged, and ocular motility improved in only 4 eyes after the surgery (Table 2). The most common type of motility disturbance was esotropia and/or hypotropia. They were subsequently corrected by eye muscle surgery in 13 out of 18 eyes, while in the remaining 5 manifestly strabic eyes prisms were used.

Three patients had slight intraoperative bleeding, mostly at the level of the posterior part of the anterior skull base, which was successfully controlled by gauze-flakes immersed previously in a vasoactive, decongestive solution. This bleeding did not disturb the usual progress of the surgery. A single patient had postoperative periorbital hematoma, which resolved spontaneously within the next 48 hours, requiring no additional treatment.

Discussion

Our study showed that surgical decompression of the orbit can resolve some of the symptoms of Graves' ophtalmophaty. Many different techniques and approaches have been reported for the treatment of Graves' ophtalmophaty (9,13-15). In 1957, Walsh and Ogura (16) described the transantral decompression, which has been the most common and favored technique until today. With this technique, both the medial and inferior orbital walls are removed, and this could also be performed through a translid or transconjunctival approach. In 1990, Kennedy described the first endoscopically assisted orbital decompression in patients with Graves' ophthalmopathy, after which the external approaches to the paranasal sinuses have been abandoned in favor of the endonasal endoscopically controlled techniques (17-19). The advantages of this procedure are avoiding bone removal and lessened morbidity compared with external ethmoidectomy or transantral surgery, also less dysesthesia of the infraorbital nerve, with fewer tooth problems and cosmetic disfiguring by uncontrolled scars (20,21). However, diplopia often develops and/or worsens postoperatively, as an undesired side effect of this technique.

In our study, best-corrected Snellen visual acuity improved after surgery. We also observed the reduction in intraocular pressure. These findings were expected, because orbital decompression increases the size of the orbital cavity, which reduces the infraorbital pressure, further fluid retention, and compression of the optic nerve. This and the reduction of proptosis and eyelid retraction significantly reduced the severity of exposure keratitis, which additionally improved the vision. Similar improvement in visual acuity, as well as reduction of intraocular pressure, was reported in other studies (22-24).

The variation in the average reduction in proptosis in our study was similar to that reported in other studies (22-24). This variation can be explained by several factors, such as the degree of preoperative exophthalmos, amount of bone removed, and different follow-up times. The greatest effect on the exophthalmos can be expected within six months of operation, but with extended follow-up the improvement can be sustained (10). In our study, the longest follow-up was 38 months.

We observed postoperative diplopia in 17 patients: in 9 patients preoperative diplopia remained unchanged and 8 patients developed new-onset diplopia. The most common type of motility disturbance in our patients was esotropia and/or hypotropia, because of the predominant myopathy of the medial and inferior rectus. The reported frequency of postoperative diplopia after orbital decompression varies considerably. In a large study of 305 patients with dysthyroid exophthalmopathy undergoing transnasal orbital decompression, Warren et al (25) observed immediate postoperative diplopia in 60.8% of patients. Out of those, a quarter required eye muscle surgery some months later, while diplopia in other patients was corrected by prisms. In another large study, comprising altogether 145 orbits of 78 patients, postoperative ocular motility imbalance was found in 58 patients (22), which required simultaneous eye muscle surgery in the same surgical session, immediately after the transnasal decompression. Recently, new modifications of the technique have been described in order to reduce the incidence of postoperative diplopia (26,27). A three-wall decompression also seems to yield less diplopia, which partly could be explained by the more symmetric relaxation of the orbital tissues. Roncevic & Roncevic (28) found no case of ocular motility imbalance after three-wall decompression, whereas Kalman et al (29) had only 3.2% new-onset diplopia in their study. The large differences in postoperative diplopia can be explained by a number of factors. As mentioned above, it is likely that the three-wall approach does not change the balance in the orbit as much as the two-wall technique, where the orbital content tends to be pushed medially. The severity of preoperative myopathy also seems to affect the results. The amount of bone removed is another possible factor. In our study, patients generally had marked ophthalmopathy, which can explain the relatively high incidence of postoperative motility imbalance, as well as the reduction in proptosis and intraocular pressure, and also improvement in visual acuity. Postoperative new-onset diplopia or worsened preoperative diplopia is an annoying complication, but on the other hand it can be corrected by eye muscle surgery or prisms. It is of course important to inform the patients of this potential complication.

In conclusion, we believe that orbital decompression should be performed in patients with active Graves' ophthalmopathy and severe compressive optic neuropathy and/or exposure keratitis not responding to conservative treatment, but also in patients with prominent exophthalmos for aesthetic reasons. We think that the endonasal endoscopic technique is safe and has satisfactory effects on proptosis and intraocular pressure reduction, as well as improvement of visual acuity. But, the risk of increasing postoperative diplopia requiring corrective eye muscle surgery or prisms is still considerable. Although it can successfully be managed by either corrective eye muscle surgery or application of prisms, it is likely that some new modification of the standard decompression technique should be considered for the treatment of these patients.

References

1 Eckstein A, Esser J. Endocrine orbit disorders. Pathogenesis, clinical presentation and examination, stage-dependent therapy [in German]. Ophthalmologe. 2003;100:857-80.

2 Bahn RS. Clinical review 157. Pathophysiology of Graves' ophthalmopathy: the cycle of disease. J Clin Endocrinol Metab. 2003;88:1939-46.

3 Tremolada C. Tremolada MA. The "triple technique" for treating stable Graves' ophthalmopathy. Plast Reconstr Surg. 1997;100:40-8.

4 Bartalena L, Marcocci C, Pinchera A. Treating severe Graves' ophthalmopathy. Baillieres Clinl Endocrinol Metab. 1997;11:521-36.

5 Weetman AP, Wiersinga WM. Current management of thyroid-associated ophthalmopathy in Europe. Results of an international survey. Clin Endocrinol. 1998;49:21-8.

6 Bartalena L, Pinchera A, Marcocci C. Management of Graves' ophthalmopathy: reality and perspectives. Endocr Rev. 2000;21:168-99.

7 Mourits MP, Koornneef L, Wiersinga WM, Prummel MF, Berghout A, van der Gaag R. Clinical criteria for the assessment of disease activity in Graves' ophthalmopathy: a novel approach. Br J Ophthalmol. 1989;73:639-44.

8 Lyons CJ, Rootman J. Orbital decompression for disfiguring exophthalmos in thyroid orbitopathy. Ophthalmology. 1994;101:223-30.

9 Dollinger J. Die Druckentlastung der Augenhöhle durch Entfernung der äußeren Orbitalwand bei hochgradigem Exophtalmus (Morbus Basedowii) und konsekutiver Hornhauterkrankung. Dtsch Med Wochenschr. 1911;37:1888-90.

10 Garrity JA. The surgical management of Graves' ophthalmopathy. Curr Opin Ophthalmol. 1994;5:39-44.

11 Leone CR Jr, Piest KL, Newman RJ. Medial and lateral wall decompression for thyroid ophthalmopathy. Am J Ophthalmol. 1989;108:160-6.

12 Goldberg RA, Kim AJ, Kerivan KM. The lacrimal keyhole, orbital door jamb, and basin of the inferior orbital fissure. Three areas of deep bone in the lateral orbit. Arch Ophthalmol. 1998;116:1618-24.

13 Naffziger HC. Progressive exophthalmos following thyroidectomy: its pathology and treatment. Ann Surg. 1931;94:582-6.

14 Sewall EC. Operative control of progressive exophthalmos. Arch Otolaryngol Head Neck Surg. 1936;24: 621-4.

15 Hirsch O. Surgical decompression of malignant exophthalmos. Arch Otolaryngol Head Neck Surg. 1950; 51:325-34.

16 Walsh TE, Ogura JH. Tranantral orbital decompression for malignant exophthalmos. Laryngoscope. 1957;67: 544-68.

17 Kennedy DW, Goodstein ML, Miller NR, Zinreich SJ. Endoscopic transnasal orbital decompression. Arch Otolaryngol Head Neck Surg. 1990;116:275-82.

18 Michel O, Bresgen K, Russmann W, Thumfart WF, Stennert E. Endoscopically-controlled endonasal orbital decompression in malignant exophthalmos [in German]. Laryngorhinootologie. 1991;70:656-62.

19 Mann W, Kahaly G, Lieb W, Rothoff T, Springborn S. Orbital decompression for endocrine ophthalmopathy: the endonasal approach. Dev Ophtalmol. 1993;25: 142-50.

20 Metson R, Dallow RL, Shore JW. Endoscopic orbital decompression. Laryngoscope. 1994;104:950-7.

21 Koay B, Bates G, Elston J. Endoscopic orbital decompression for dysthyroid eye disease. J Laryngol Otol. 1997;111:946-9.

22 Michel O, Oberlander N, Neugebauer P, Neugebauer A, Russmann W. Follow-up of transnasal orbital decompression in severe Graves' ophthalmopathy. Ophthalmology. 2001;108:400-4.

23 Eloy P, Trussart C, Jouzdani E, Collet S, Rombaux P, Bertrand B. Transnasal endoscopic orbital decompression and Graves' ophtalmopathy. Acta Otorhinolaryngol Belg. 2000;54:165-74.

24 Yuen AP, Kwan KY, Chan E, Kung AW, Lam KS. Endoscopic transnasal orbital decompression for thyrotoxic orbitopathy. Hong Kong Med J. 2002;8:406-10.

25 Warren JD, Spector JG, Burde R. Long-term follow-up and recent observations of 305 cases of orbital decompression for dysthyroid orbitopathy. Laryngoscope. 1989;99:35-40.

26 Wright ED, Davidson J, Codere F, Desrosiers M. Endoscopic orbital decompression with preservation of an inferomedial bony strut: minimization of postoperative diplopia. J Otolaryngol. 1999;28:252-6.

27 Metson R, Samaha M. Reduction of diplopia following endoscopic orbital decompression: the orbital sling technique. Laryngoscope. 2002;112:1753-7.

28 Roncevic R, Roncevic D. Surgical treatment of severe dysthyroid ophthalmopathy: long-term results. J Craniomaxillofac Surg. 1995;23:355-62.

29 Kalmann R, Mourits MP, van der Pol JP, Koornneef L. Coronal approach for rehabilitative orbital decompression in Graves' ophthalmopathy. Br J Ophthalmol. 1997;81:41-5.

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