Management of Patients With Graves Orbitopathy Using Endonasal Endoscopic Techniques

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Objective: The aim of the authors' study is to show their surgical results in orbital decompression using different endonasal endoscopic techniques. These approaches are according to the degree of proptosis and the presence or not of sight threatening.

Methods: The authors performed 31 orbital decompressions on 20 Graves orbitopathy patients. Average age at surgery was 52 years. There were 5 males and 15 females. Five patients were diagnosed as having severe or for sight-threatening Graves orbitopathy. These included 3 men and 2 women having an average age of 54 years old. Minimum postsurgical follow-up was 12 months in all patients.

Results: Orbital decompression was performed in 15 patients for proptosis and in 5 patients for urgent sight threat. Thirteen orbits showed mild proptosis and 18 orbits presented moderate proptosis. In patients without sight threatening reduction of proptosis had a mean value of 2.8 mm as determined by exophtalmometry, being 3.3 mm when measured on magnetic resonance imaging. The mean millimeter in mild proptosis was between 1.5 and 1.7 and between 3.4 and 4.2 in moderate proptosis. In patients having sight threat mean visual acuity after surgery improved from 0.6 to 0.9.

Only 1 patient without diplopia preoperative developed diplopia after surgery (17%). In 55% of patients strabismus and/or eyelid surgery were required.

In postoperative follow-up, 2 patients developed a mucocele and 1 patient developed corneal erosion.

Conclusion: The authors recommend the preservation of the periorbital sling and the anterior ethmoido-maxillary angle in patients with mild-moderate exophthalmos and without threatened vision. In case of sight threatening the authors resected the most periorbita as much as possible.

Evidence-based medicine Level V.

Key Words: Decompression, endoscopic, Graves, orbitopathy, periorbita, sling

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Graves orbitopathy (GO) is an autoimmune inflammatory orbital disorder that represents the most frequent extrathyroidal manifestation of Graves disease. Approximately 95% of patients with GO have an associated disease in the thyroid, this being a diffuse goiter with hyperthyroidism in 85% of the cases. Graves orbitopathy can occur less frequently in euthyroid or hypothyroidism patients and in patients with Hashimoto thyroiditis.[1,2] GO's prevalence in Europe is about 10/10,000 people[3] and the annual incidence is more frequent in women than in men, having a ratio of 5.3 (16 per 100,000 population in women and 2.9 in men).[4] In most cases, it has a bilateral involvement and it is more frequent in Caucasian than in Asian populations.[4–6] Patients with GO are usually older than those having Graves disease without orbitopathy and frequently it is more severe in men older than 60.[2]

Around half of Graves disease patients present GO, but it is only clinically relevant in 20% to 30% of cases. Between 3% and 7% of patients with GO developed a very severe disease with corneal exposure and/or compressive optic neuropathy.[7,8]

OG's pathogenesis implies a complex network of factors. It seems demonstrated that the immune system recognizes an autoantigen, the thyrotropin receptor, which is located within the orbit and in the follicular endothelial thyroid cells.

The B cells produce autoantibodies against these antigens (TRAb) which stimulates the thyroid gland and the orbital fibroblasts secreting inflammatory cytokines (tumor necrosis factor alfa, interleukin (IL)-18, IL-6, IL-10, IL-17, interferon gamma) and glycosaminoglycan. In the orbit, this process causes an accumulation of fluid between the muscle fibers and adipose tissue hyperplasia. As a result, patients can present eyelid retraction and edema, proptosis, ocular motility imbalance, and optic neuropathy. Avoiding exogenous factors as cigarette smoking and stress it is essential for GO treatment, as well as restoring normal thyroid function since both, hyper and hypothyroidism, have a negative impact on the orbitopathy.

Specific treatment depends on the activity and severity of the orbitopathy.[1] First line of treatment in moderate-severe GO is high dose of intravenous corticoids. Partial responses or recurrences can be treated with orbital radiotherapy, immunosuppressive therapy, or biological treatments.[1,8,9]

Surgical management of the GO has 2 main indications.

1. Elective rehabilitative surgery to patients after the disease has been inactive for at least 6 months
2. Functional surgery for optic neuropathy and/or severe corneal exposure.

Different decompression techniques and cosmetic surgery have been applied so far. The aim of our study is to show our surgical results in orbital decompression and to compare the efficiency of endonasal endoscopic approach with or without partial preservation of the periorbita and/or the ethmoido- maxillary junction according to the various indications.

METHODS

Patients and Pathologies Characteristics

A multidisciplinary team belonging to the Orbitopathy Committee at our Institution had previously studied all patients. From January 2008 to January 2018, we performed 31 orbital decompressions on 20 GO patients (Supplementary Digital Content, Table 1, http://links.lww.com/SCS/B908).

All patients were evaluated preoperatively with clinical activity score, thyroid function, endonasal endoscopy, Hertel exophtalmometry, strabismus cover test, visual acuity without optical correction, visual upper field, and intraocular pressure by pneumotonometer.

Neuroradiological assessment (computed tomography scan and orbital MRI [magnetic resonance imaging]) included the orbital proptosis in millimeters, the number of extrinsic orbital muscles involved in volume, signal, and contrast enhancement and compression and or /elongation of the optic nerves.

Average age at surgery was 52 years ranging from 35 and 82. There were 5 males and 15 females. Most of the patients were euthyroid before surgery (63%), the 26% were hypothyroid and only the 10% were operated while having hyperthyroidism. Fifty per cent of patients (10/20) were smokers at the time of surgery.

Five patients were diagnosed as having severe or sight threatening GO because of optic neuropathy, ulcer, and/or corneal perforation. These included 3 men and 2 women having an average age of 54 years, with normal thyroid function at the time of surgery and 60% with active tobacco consumptions at the time. Minimum postsurgical follow-up was 12 months in all patients.

Surgical Technique

We performed a priority surgery in sight threatening GO or in cases of active orbitopathy without response or tolerance to corticotherapy or monoclonal therapy. In these cases, we always remove the lamina papiracea, resect the posterior ethmoido-maxillary junction, and also resect the most periorbita as much as possible.

We performed a programmed surgery for rehabilitation of sequels in patients with a thyroid functional stability more than 6 months. In these cases, the type of surgery was according to GO severity.

Mild proptosis (<3 mm value above the reference by sex and ethnicity): no surgical indication except for exceptional situations with significant functional and psychological repercussions and always under a committee's agreement decision. We performed a medial decompression where we respect entirely the ethmoido-maxillary angle and a band of periorbita (Fig. 1).

Moderate proptosis (3–7 mm value higher than the reference by sex and ethnicity): Decompression of 2 walls (medial and lateral) frequently associated with lipectomy. In this medial decompression we resected the posterior ethmoido-maxillary angle and respected 2 bands of periorbita (Fig. 2). In the lateral wall decompression we performed an incision at fold from the center to the temporal periphery. We did a dissection by layers (orbicularis muscle, septum) and performed a dissection of the zygomatic arch in subperiostical plane. We made superior and inferior lateral wall osteotomies and then this bone was released. Then we milled the greater sphenoid wing and opened the periorbita with or without lipectomy. Finally, we did the zygomatic arch repositioning with mini plates and a layered suture (Fig. 3).

Severe proptosis (> 7 mm value higher than the reference by sex and ethnicity): Decompression of 3 walls (lateral–medial–inferior) frequently associated with lipectomy. We performed the same medial decompression than in moderate proptosis cases but without orbital sling preservation.

RESULTS

Orbital decompression was performed in 15 patients (75%) for proptosis and in 5 patients (25%) for urgent sight threat. Thirteen orbits showed mild proptosis, this being moderate in 18. Mean preoperatively clinical activity score was 1.8 (SD: 2.2) being 1.2 in

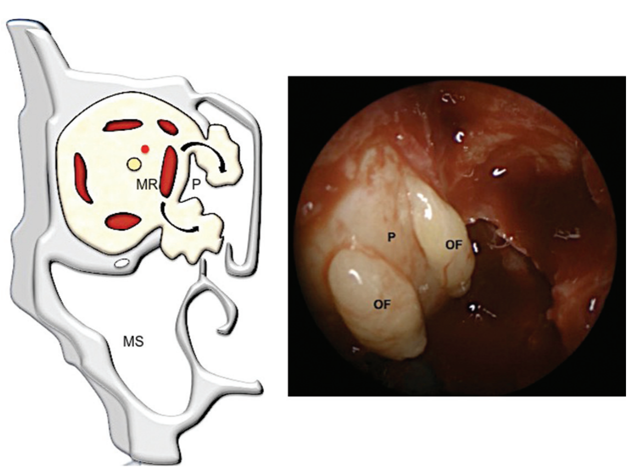

FIGURE 1. Mild proptosis. Medial decompression with a band of periorbita overlying the medial rectus muscle. MR, medial rectus; MS, maxillary sinus; OF, orbital fat; P, periorbita.

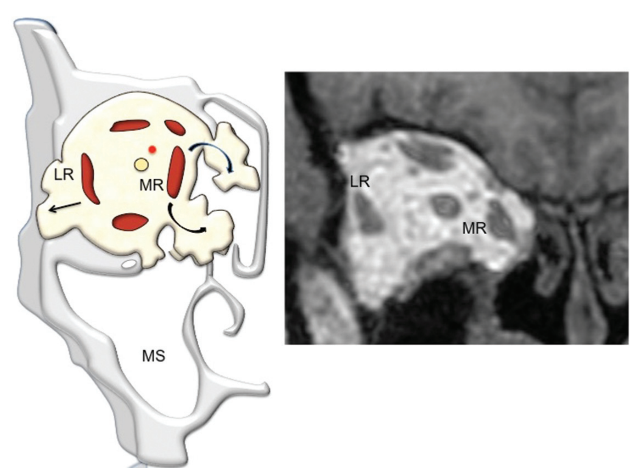

FIGURE 2. Moderate proptosis. Medial and lateral decompression with 2 bands of periorbita and with resection of the posterior ethmoido-maxillary angle. LR, lateral rectus; MR, medial rectus; MS, maxillary sinus.

FIGURE 3. Patient before surgery and following 2 walls decompression 1 year later.

patients without sight threatening and 3.7 in the rest. Mean degrees of eye protusion were 23 mm and 23.6 mm measured by Hertel exophtalmometry and by MRI respectively. The mean degree of eye protusion according to GO severity (presence or not of sight threatening) is shown in the Supplementary Digital Content, Table 2, http://links.lww.com/SCS/B908. Mean intraocular pressure was 18.9 mm Hg having a range of 12 to 35 mm Hg. In patients having sight threat mean intraocular pressure was superior to in patients without severe GO (21.6 mm Hg versus 17.7 mm Hg, see Supplementary Digital Content, Table 2, http://links.lww.com/SCS/B908).

Average visual acuity was 0.8 with a ranging from 0.2 to 1. In patients with very severe GO it was 0.6 and 0.9 in patients without sight threatening. In patients having sight threat mean visual acuity after surgery improved to 0.9.

In patients without sight threatening reduction of proptosis had a mean value of 2.8 mm as determined by Hertel exophtalmometry, being 3.3 mm when measured on MRI. The mean millimeter in mild proptosis was between 1.5 and 1.7 and between 3.4 and 4.2 in moderate proptosis.

Preoperative diplopia was observed in 70% of patients (14/20). Most of them did not improve their diplopia after surgery (11/14). Six patients did not present preoperatory diplopia but only 1 from these developed diplopia after surgery (17%).

After orbital decompression, in 55% of patients (11/20) strabismus and/or eyelid surgery were required.

In postoperative follow-up, 2 patients developed a mucocele, 1 located in the ethmoidal sinuses, and 1 in the maxillary sinus. One patient developed corneal erosion. None of the patients suffered from epistaxis or epiphora.

DISCUSSION

Several approaches and different degrees of extension of the orbital walls removed have been published in the management of the GO. For many years, the standards approaches have been the external and the transantral. In 1889, Dollinger J. was the first to suggest the orbital enlargement by lateral incision for exophthalmos. Later, Walsh and Ogura in 1949 first employed the transantral approach of the orbital contents.[10,11]

With the development of the endonasal endoscopic techniques, orbital decompression is nowadays carried out only endonasally or combined with external approaches. Endoscopic endonasal technique allows an excellent approach to the inferomedial orbital wall. According to different series combined external and endonasal endoscopic approaches are at present the most employed, obtaining excellent results in decreasing proptosis and improvement visual acuity. Leon et al[12] showed in a review of more than 4000 orbital surgeries that decompression of the combined transpalpebral and endoscopic approach resulted in the highest average reduction in proptosis.

Whichever approach was used, proptosis decrease is correlated with the number and location of the walls removed.

It has been reported a mean of 8 mm after 3 walls decompression, between 4 and 6 mm with inferior and medial or lateral decompression and only between 2 and 3 mm with lateral wall removing.[13]

In a large series of patients reviewed, Kingdom et al[14] found a reduction in proptosis of 3.9 to 4.7 mm in 2 walls (inferior and medial), between 3.2 and 5.9 mm if the walls resected were medial and lateral and a maximum reduction when 3 walls were removed (5.6–6.9 mm).

In our series, patients treated with 2 walls decompression (moderate exophthalmos) showed similar results than the other series with mean reduction between 3.4 and 4.2 mm. However, we achieved lower results in mild proptosis (1.2–2.4 mm). Probably, these results are due to our conservative procedures performed when we carry out medial wall decompression only. In these patients we prefer to be less aggressive with the decompression to avoid a possible diplopia.

In fact, the main disadvantage of incorporating the medial wall in an orbital decompression is a high percentage of postsurgical diplopia, which it can represent more than 50% in some series. Kingdom et al[14] reviewed the literature and showed a rate of new diplopia between 14% and 64% when the walls removed were the inferior and medial. Mainville and Jordan et al[15] published a 30% of new-onset diplopia when it was removed the same 2 walls, the medial and inferior. Fabian et al[16] published similar rates of new-onset diplopia when the orbital decompression included the inferior and medial wall (33%).

We totally agreed with these authors who suggest different measures to avoid high rate of diplopia (balanced decompression, periorbita, and ethmoido-maxillary angle preservation).

The endoscopic medial approach produces an asymmetric decompression that could be compensated including the lateral wall (balanced decompression). Different series demonstrated a reduction of the postsurgical diplopia, between 0% and 15% when the balanced decompression was used.[13,17–21]

When the periorbita (orbital periostium) is incised, major expansion of orbital components is reached but it is clearly demonstrated that the incidence of diplopia is lower if the periorbita remains intact.[15] Metson and Samaha[22] described in 2002 the preservation of medial periorbital strip along the medial rectus muscle revealing any case of diplopia in 13 patients studied. Jimenez Chobillon and Lopez-Oliver[23] performing a similar technique showed a satisfactory reduction in proptosis with only 7% cases of diplopia.

Several series confirmed that when the ethmoido-maxillary junction was preserved with or without a periorbital sling did not adversely affect the reduction in proptosis, and the incidence of new-onset postoperative diplopia was between 0% and 17%.[24–27]

Yao et al[24] described a modified technique, in which preserves the anterior ethmoido-maxillary junction which represents the main support to the orbit.

We currently decide the size of decompression according to the degree of proptosis by giving priority to the lack of postsurgical double vision.

For that reason, in the mild proptosis we only remove the medial wall with a band of periorbita and preserve entirely the ethmoido-maxillary angle. In moderate—severe cases we perform balanced decompression and resect the posterior area of the ethmoido-maxillary angle. In addition, in severe cases we remove all periorbita as possible. When we began to make this surgical orbitopathy in the 90s, we presented more than 50% of new diplopia (cases series no published). Making use of these conservative procedures according to the degree of proptosis we have achieved a new diplopia rate lower than 20%.

The compressive optic neuropathy occurs between 5% and 8.6% of patients with GO and represent approximately the 40% of the indications of the thyroid ophthalmic surgery.[28,29] In our center this surgery represents the 25% of the total surgeries. This percentage probably will diminish in the future due to improvements in medical treatment.[8]. Patients with neuropathy are frequently men older than 60 years old with tobacco consumptions and with thyroid dysfunction. These epidemiological information are in accordance with ours. Our patients with sight threatening GO are predominately older men with tobacco consumption.

Compression of the optic nerve or the vascular supply (ischemic neuropathy) is the main mechanism to explain the sight threatening. In few cases it can be due to prolonged optic nerve stretching. Most of patients do not present higher degrees of proptosis. Probably rigidity, inflexibility of the orbital septum provides the nerve compression without proptosis. Several publications demonstrated that the degree of proptosis is not correlated with the severity of neuropathy. European Group on Graves Orbitopathy observed a mean proptosis of 22.1 mm in 47 patients with optic neuropathy.[30] In our study the mean degree of proptosis was lower in sight threatening GO than in others (21.7–22.1 versus 23.5–24.2).

Orbital decompression in the presence of optic neuropathy provides a rapid improvement of the visual function in the 70% to 95% of cases using different techniques[13] All our patients with sight threatening improved the visual acuity (0.6–0.9) after surgery. Several reports showed in computed tomography studies that the apical muscle crowding is related with the optic neuropathy.[31] Weis et al[32] evaluates 198 orbits with optic neuropathy founding that the medial rectus muscle volume was the only independent significant predictor of compressive neuropathy. For that reason, we perform medial decompression but without orbital sling preservation and including the posterior ethmoido-maxillary angle. In its intraorbital route, the optic nerve courses lateral to medial towards to the chiasm. Therefore, the optic nerve is located more medially on the vicinity of the most posterior ethmoidal cells. That is the reason to remove the posterior angle (palatine bone) and not the anterior that remains intact (maxillary bone).

Finally, we have never performed an optic canal decompression in these patients because the aetiology of the loss of vision is the optic nerve compression, which is located in the apex orbit and not inside the canal, where no fat neither muscles are found.

CONCLUSION

We think that severe corneal exposure and optic neuropathy must be treated with more aggressive surgeries as soon as possible to avoid vision loss. Nevertheless if the main objective is proptosis reduction we should be more conservatives to diminish a new diplopia. Orbital decompression should be according to the degree of proptosis not neglecting a main objective, the lack of postsurgical double vision.

We recommend the preservation of the periorbital sling and the anterior ethmoido-maxillary angle in patients with mild–moderate exophthalmos and without threatened vision. For sight threatening we resect the most medial periorbita as much as possible.

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