Orbital Decompression for Thyroid Eye Disease

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Abstract

Although most cases of thyroid eye disease (TED) can be managed medically, some refractory or severe cases are treated surgically with orbital decompression. Due to a lack of randomized controlled trials comparing surgical techniques for orbital decompression, none have been deemed superior. Thus, each case of TED is managed based on patient characteristics and surgeon experience. Surgical considerations include the extent of bony wall removal, the surgical approach, the choice of incision, and the use of fat decompression. Outcomes vary based on surgical indications and techniques; hence, vision can improve or worsen after the surgery.

Thyroid Eye Disease

Epidemiology

Thyroid eye disease (TED) is the most common extrathyroid presentation of Graves’ disease. It affects 25 to 50% of patients, with severe cases occurring in 3 to 5% of patients. This autoimmune disorder has an annual incidence of 16 per 100,000 in women and 3 per 100,000 in men, and most commonly affects adults in their fourth and fifth decades of life. Although most commonly associated with Graves’ disease, TED has also been linked to other thyroid diseases such as Hashimoto’s thyroiditis.1–3

Pathogenesis

The pathogenesis of TED is not fully understood, so there is no definitive medical treatment for the disease.4 Several mechanisms have been proposed, involving both cellular and humoral immunity. Some studies suggest that antibodies may have a direct effect on orbital connective tissue, leading to the clinical presentation of the disease.5–7 The disease has an initial inflammatory stage of lymphocytic infiltration, fibroblast activation, and glycosaminoglycan deposition in orbital and peri-orbital tissues. The abnormal accumulation of hyaluronic acid within muscle and adipose tissue causes edema and volume expansion. Medical treatment during this stage consists of immunosuppression and medical management of the dysthyroid.8 The inflammatory phase is followed by a fibrotic stage during which there is fibrosis of the extraocular muscles and fat hypertrophy in the orbit. The fibrotic stage can produce tissue scarring that may require surgical correction.9,10 The disease then enters a quiescent phase of static disease burden, although some patients may experience a reactivation of inflammation. Smoking is associated with more severe TED symptoms, recalcitrance to medical treatment, and reactivation of the disease. Radioiodine therapy for hyperthyroidism has been shown to cause the progression of thyroid ophthalmopathy in 15% of treated patients, so concurrent steroid therapy is suggested with radioiodine therapy for severe orbitopathy.3

Clinical Presentation

Expansion of the orbital contents and fibrosis leads to exophthalmos and eyelid retraction,11 which can cause exposure keratopathy and corneal ulceration.12 Other clinical manifestations include double vision, conjunctival congestion, and extraocular muscle motility disturbances.4 However, the most feared complication of TED is optic neuropathy. Approximately 3 to 5% of patients with severe TED experience reduced vision due to optic nerve compression. Without treatment, 30% of these patients develop permanent vision loss.1
Treatment Options
Medical therapy is generally preferred over immediate orbital decompression surgery in the absence of optic neuropathy or other immediate surgical indications such as severe ocular surface compromise and subluxation of the globe.\(^1\)\(^3\) Initial medical therapy for severe and active TED involves pulses of methylprednisolone. Although oral and intravenous (IV) steroids are both effective, IV steroids are more efficacious and better tolerated.\(^1\)\(^4\) Orbital radiation therapy has proven effective for mild-to-moderate active TED, especially for improving motility.\(^1\)\(^5\)\(^6\)\(^7\) Combining radiation and steroids has proven more efficacious than either treatment modality alone.\(^1\)\(^7\) When medical management fails, orbital decompression can be an effective treatment for optic nerve compression, exposure keratopathy, and cosmetic irregularities produced by the disease.\(^1\)\(^8\)\(^9\)\(^1\)\(^0\)

Surgical Indications and Timing
Most patients with TED respond to conservative measures and do not require surgical treatment. Approximately 5% of patients with TED undergo surgery in the first year after diagnosis, and up to 20% have surgery in the first 10 years.\(^1\) Indications for acute decompression include optic neuropathy, globe subluxation, corneal ulceration that does not respond to medical management, and prevention of exposure.\(^1\)\(^1\) Some cases of optic neuropathy may be amenable to corticosteroids prior to decompression, but this only applies to minor degrees of optic neuropathy.\(^4\) Indications for delayed decompression include diplopia, proptosis, orbital pain, and ocular hypertension. Additionally, orbital decompression can be offered to the patient to manage the cosmetic effects of TED. Usually, delayed decompression occurs 6 months after the acute episode of TED to allow the active inflammatory phase to pass.\(^1\)\(^8\) It is important to perform orbital decompression before muscle surgery, and muscle surgery before eyelid adjustments.\(^2\)\(^0\)

Preoperative Management
Prior to surgery, the patient should have a complete clinical exam along with high-resolution computed tomography (CT) or magnetic resonance imaging (MRI) of the orbit and paranasal sinuses. The clinical examination should pay special attention to upper and lower lid retraction, lagophthalmos, ocular motility, and corneal exposure. Old photographs of the patient should be obtained to determine the amount of proptosis reduction needed to return the patient to the predisease state. Proptosis can be measured clinically via Hertel exophthalmometry and radiologically via CT; these methods have proven to be equally effective.\(^2\)\(^1\) Imaging can be used to predict how much proptosis can be relieved by orbital decompression.\(^2\)\(^2\) Short tau inversion recovery sequences on MRI can also help determine whether there is any active inflammation. Computed tomography is also needed if stereotactic guidance will be used during the procedure.

Orbital Decompression Overview
The goals of surgery are to reduce or reverse vision loss, prevent ocular surface damage, and relieve orbital congestion. Additional goals are to reduce proptosis, diplopia, lid retraction, chemosis, lid edema, and fat prolapse.\(^1\) Orbital decompression expands the volume of the orbit by partial removal of the bony orbital walls and/or orbital fat.\(^1\)\(^0\) There are several surgical techniques for decompression. Given the lack of randomized controlled trials comparing these methods, none have been deemed superior. The best method is therefore one individualized to the patient and depends on surgeon preference and experience.\(^1\)\(^1\)\(^1\)

Orbital decompression was first described by Dollinger in 1911, with the introduction of lateral wall decompression. In this technique, the lateral wall is removed and orbital contents are decompressed into the temporalis fossa. In 1921, Naffziger introduced the transcoronal approach, which allows superior decompression.\(^8\) The Walsh-Ogura technique was subsequently described: In this procedure, the orbital floor followed by the medial wall is removed.\(^2\)\(^3\) Further modifications combined these approaches, removing multiple walls concurrently. There are several approaches to access the orbit. With the introduction of the endoscopic transnasal approach, surgeons are now able to completely remove bone along the orbital apex and optic canal medially.\(^1\)

Surgical Considerations
Considerations prior to surgery include the extent of bony wall removal, the surgical approach, the choice of incision, and whether to use fat decompression. Removal of each wall reduces proptosis in varying amounts, and studies vary on the amount of proptosis reduction achieved with removal of each wall. In general, fat decompression adds an additional 2 to 4 mm of proptosis reduction, particularly in the setting of increased orbital fat.\(^8\) For high degrees of proptosis, three-wall decompression is the preferred method in our practice. This usually consists of a combination of external and endonasal approaches.\(^1\)\(^1\)\(^1\) If three-wall decompression fails to relieve compressive neuropathy, orbital roof decompression may be considered.\(^1\)\(^9\) When less proptosis is present, one might consider a balanced two-wall decompression of the medial and lateral walls combined with orbital fat decompression. This technique reduces proptosis and has a lower incidence of postoperative diplopia when compared with a three-wall decompression.\(^2\)\(^4\)

The surgical approach depends on which walls the surgeon wishes to access. The medial wall borders the ethmoid air cells, and a posterior medial decompression is especially effective for treating compressive optic neuropathy (Fig. 1). However, motility disturbances or diplopia may be worsened with medial wall decompression, and the patient should be counseled on the possible need for strabismus surgery after decompression.\(^2\)\(^2\) The medial wall can be accessed via the endoscopic nasal approach, which provides better visualization and provides access for more posterior decompression in a safer fashion. The medial wall is also accessible via a transcaruncular incision, which is more effective at achieving proptosis reduction than at achieving optic nerve decompression.

The orbital floor overlies the maxillary sinus and is divided into medial and lateral portions by the infraorbital groove and
nerve (Fig. 2). The orbital floor can be accessed by a transconjunctival incision. The lateral wall is divided into anterior and deep sections. This wall is accessible via an upper eyelid crease incision, extended lateral canthal incision, coronal incision, or lateral orbital rim incision (Fig. 3). The lateral and inferior walls can be accessed simultaneously via a lateral canthal “swinging eyelid” flap with the release of the canthal tendon, which is our preferred method.8,11

Surgical Techniques

Orbital decompression is performed in the operating room under general anesthesia. Preoperative IV antibiotics and corticosteroids are given. After induction of anesthesia, 1 or 2% lidocaine with 1:100,000 epinephrine is injected at the planned incision site.

Medial Wall Decompression

After the injection of lidocaine into the medial canthal region, a transcaruncular incision is created using Westcott scissors. The medial wall is then identified and the overlying periosteum is opened with monopolar cautery and elevated using a Freer elevator (Fig. 4). The bone posterior to the posterior lacrimal crest is then removed with Kerrison rongeurs, leaving the anterior portion of the medial wall intact. After hemostasis is achieved, the caruncle is approximated using a single 6-0 plain gut suture. When a more posterior decompression is necessary, this procedure is combined with an endoscopic transnasal approach performed by one of our skull-base otolaryngology colleagues.

Floor Decompression

The inferior fornix of the lower eyelid is injected with lidocaine. Then, a transconjunctival incision extending from the caruncle to the lateral canthus is made 4 mm inferior to the inferior border of the tarsal plate (Fig. 5). With the conjunctiva and lower eyelid retractors on superior tension, blunt dissection is performed down to the arcus marginalis at the inferior orbital rim. The periosteum is opened with monopolar cautery and elevated off the orbital floor with a Freer elevator. Bone medial to the infraorbital nerve is removed using Kerrison rongeurs, leaving the strut intact. The conjunctiva is closed with two absorbable sutures.

Lateral Wall Decompression

The lateral canthal area is injected with lidocaine. The lateral wall is approached via a lateral canthotomy incision combined with a short upper eyelid crease incision. The lateral orbital rim is exposed and periosteum incised with monopolar cautery, then reflected nasally with a Freer elevator (Fig. 6). The zygomaticotemporal and zygomaticofacial neurovascular bundles are cauterized and ligated to

Fig. 1 Medial wall of the orbit highlighted (courtesy of Roger A. Dailey, MD, FACS).

Fig. 3 Lateral orbital wall highlighted (courtesy of Roger A. Dailey, MD, FACS).

Fig. 2 Orbital floor highlighted (courtesy of Roger A. Dailey, MD, FACS).

Fig. 4 Medial wall exposure using the monopolar cautery to incise the periosteum overlying the bone (courtesy of Roger A. Dailey, MD, FACS).
prevent hemorrhage. A surgical drill is used to sculpt the internal orbital bone, expanding the orbit laterally. Bone resection is then performed superiorly to the lacrimal fossa, inferiorly to just lateral to the infraorbital canal, and laterally until the temporalis muscle is exposed. Posteriorly, the trigonal bone of the greater wing of the sphenoid is resected along with its marrow space. The marrow is highly vascularized and the underlying dura is exposed during this stage. Extreme care must be taken to control any bleeding and avoid damage to the dura. The periorbita is then incised above and below the lateral rectus muscle, allowing herniation of the orbital contents. At this time, the inferolateral fat pocket can be removed. If orbital floor decompression is also planned, the floor can be accessed via cantholysis of the lateral canthal tendon and the creation of a swinging lower eyelid flap with a transconjunctival incision. A 4–0 Vicryl suture is used to close the periosteum. The lateral canthal tendon, if divided, is attached to the periosteum with absorbable suture. The commissure is then reapproximated in layers.\(^{22,24}\)

**Endoscopic Techniques**

A transnasal endoscopic approach can be used to decompress the medial wall. Topical oxymetazoline or cocaine on cotton pledgets is placed in the nose to vasoconstrict the nasal mucosa; lidocaine is injected into the lateral nasal wall. An uncinectomy can be performed if there is insufficient space in the middle meatus to accommodate the orbital contents. If performed, an uncinectomy and a maxillary antrostomy are completed prior to proceeding with the procedure. The infraorbital nerve can be visualized on the floor of the orbit and should be avoided. An endoscopic sphenoethmoidectomy is then performed. A spoon curette or periorbital elevator is used to penetrate the bone and open the medial orbital wall. Bone fragments are removed with Blakesley forceps. The medial orbital floor is then fractured with a spoon curette, and the orbital floor medial to the infraorbital nerve is removed. The periorbita is now fully exposed and is incised, allowing the prolapse of orbital contents into the ethmoid and maxillary cavities. Often, an endoscopic medial wall decompression is combined with a lateral or inferior wall decompression. The endoscopic approach can also be used as an adjunct to transfacial approaches to facilitate debridement. The endoscopic approach has the added benefit of decompressing the optic nerve if the need arises. It can also be used with stereotactic guidance to enhance surgical navigation.\(^{22,25}\)

**Orbital Fat Decompression**

The transconjunctival incision is routinely used for orbital fat decompression. However, when the globe is too tightly opposed to the lower eyelid, a transcutaneous approach may be necessary such as a lateral canthotomy. The orbital septum is exposed and opened, allowing the fat pads to herniate through the septum. The orbital fat can then be removed, with malleable retractors protecting the globe and eyelid. Fat removal can be performed with scissors or by monopolar cauterity. After hemostasis is achieved, the conjunctiva is closed with absorbable sutures or the canthal structures are reapproximated, depending on the approach. If deemed appropriate, the superonasal fat pad can also be accessed via an eyelid crease incision. Adding a superior fat decompression can increase reduction of proptosis by another 0.5 mm.\(^{26}\)

**Postoperative Care**

Patients are observed for several hours after surgery. Ophthalmology "vital signs" should be checked including visual acuity, pupils, motility, and intraocular pressure.\(^{27}\) After this period of observation, the patient is sent home with oral corticosteroids and oral antibiotics. The patient is also instructed to return to the clinic the next day for follow-up. Alternatively, the surgeon may opt to place a pressure patch over the surgical eye and evaluate the patient the next day in the clinic.

**Outcomes and Complications**

A goal of orbital decompression is to decrease proptosis. Decompression of the medial and inferior walls has been shown to reduce proptosis by 4.4 to 4.7 mm. Medial wall decompression has been shown to produce greater orbital expansion than lateral wall surgery.\(^{20}\) The transantral approach achieves a greater amount of proptosis reduction than the endoscopic approach.\(^{12}\)
Thyroid eye disease can adversely affect a patient’s quality of life (QOL), and changes in patient QOL after therapeutic interventions are increasingly recognized as an important surgical outcomes measure. Health-related QOL is assessed after eye surgeries with the Graves’ ophthalmopathy quality of life (GO-QOL) questionnaire.\(^2\) After an orbital decompression, the improvements in QOL are most related to visual function and appearance.\(^{29,30}\)

The overall complication rate after orbital decompression is 9.3%.\(^3\) Immediate complications include periorbital ecchymosis and edema, postoperative hemorrhage, and infection. Vision loss can occur, but is uncommon. Additional complications include sinusitis, cerebrospinal fluid leak, hematoma, paresthesia, and hypesthesia.\(^1,28\)

Vision-related complications include residual field defects, diplopia, worsening of motility, and strabismus.\(^12,19\) Visual acuity after decompression was shown to improve in 44 to 55% of patients, to remain stable in 27 to 36%, and to worsen in 18 to 20% of patients. Visual acuity was most improved in patients undergoing decompression for optic neuropathy, with 82 to 88% of these patients demonstrating increased postoperative vision.\(^1\) Orbital decompression was shown to improve diplopia in 28.1% of patients; however, the surgery can induce diplopia in 29.7% of patients. Opening the periorbita is associated with increased new-onset diplopia.\(^32\)

Another important vision outcome is sensory disturbances, which are common, but tolerable in most cases.\(^12\)

**Conclusion**

Thyroid eye disease can be associated with significant morbidity and mortality. Understanding orbital and eyelid anatomy is essential in providing the appropriate surgical approach in these patients. The specific surgical approach should be tailored to each patient to provide optimal results.

**References**