Overview article

DOI: 10.59715/pntjmp.4.2.1

Orbital decompression surgery in the treatment of thyroid eye disease

Le Tuyet Hoa
General Internal Medicine, Faculty of Medicine, Pham Ngoc Thach University of Medicine

Received: 25/10/2024
Revised: 20/3/2025
Accepted: 20/4/2025
Author contact:
Le Tuyet Hoa
Email:
letuyethoa@gmail.com
Phone: 0913156131

Abstract

Graves' orbitopathy is a complex autoimmune disorder of the orbit that causes the eye to appear disfigured or results in loss of vision. The treatment of critical cases can be very challenging. In 2022, the American Thyroid Association (ATA) and European Thyroid Association (ETA) joined forces to produce the Consensus Statement for TED, clearly mentioning the indications for orbital decompression surgery. Orbital decompression is widely presumed to improve exophthalmos and compressive or stretch optic neuropathy. The relative efficacy and complications of such surgery for the specific indications remain not yet definite. The paper showed the indications of orbit decompression surgery and reviewed current published evidence regarding the effectiveness and safety of these procedures for TED. Customized treatment plans incorporate individual anatomy, orbital pathology, and surgery objectives to maximize therapeutic benefit while minimizing morbidity.

Keywords: thyroid eye disease, orbital decompression surgery

1. INTRODUCTION

Thyroid eye disease (TED) is an eye abnormality associated with thyroid disease. Clinically, TED is most common in patients with Graves' disease, occurring at all ages. Most patients present with asymptomatic to mild eye disease, very few patients have severe exophthalmos or active eye disease, and optic nerve damage is even rarer. Over the past ten years, the Department of Endocrinology at Nguyen Tri Phuong Hospital has received only 0-3 cases of moderate to severe TED each year requiring inpatient treatment, and of these, about 4 patients have undergone orbital decompression surgery. This means that not many TED patients are eligible for orbital decompression surgery and few experienced surgeons perform the procedure, so we do not know much about the effectiveness and complications of this surgical method. This article presents the understanding of the indications, surgical approach, effectiveness, and related complications of orbital decompression surgery in the treatment of Graves' eye disease from the perspective of an Endocrinologist.

Patients with moderate to severe TED have a significant impact on their quality of life. They seek out ophthalmologists or endocrinologists. A study in China investigating current clinical practice on the management of TED found that patients with moderate-severe inactive TED sought out ophthalmologists (70.2%), who had a significantly higher preference for orbital surgery compared to endocrinologists (28.5%, $p < 0.001$). Conversely, endocrinologists (43.6%) had a higher inclination toward indicating intravenous corticosteroid compared to ophthalmologists (17.7%, $p < 0.001$) [1]. At Nguyen Tri Phuong Hospital, the Endocrinology Department prescribes glucocorticoid therapy in the active phase of the disease or in the case of optic nerve damage. If glucocorticoid therapy fails, orbital decompression intervention is considered, in which case consultation with the ENT department is preferred over coordination with the Ophthalmology department.

The treatment of orbitopathy in Vietnam is still difficult: (1) there are no biomarkers to predict the disease and no appropriate preventive solutions, (2) it is difficult to restore the eye condition to its pre-TED state even though there are imaging techniques to objectively assess the activity and severity of TED, (3) a multidisciplinary team has not yet been organized to care for and support people with severe TED, (4) there is still a lack of solid clinical data on the effectiveness and side effects of orbital decompression surgery. Although the FDA approved the monoclonal antibody teprotumumab in 2020 to treat moderate to severe TED in the United States as a second-line treatment after glucocorticoids, the drug has not been approved in Europe or the rest of the world, and the cost is very high.

2. PATHOLOGY OF THYROID EYE DISEASE, RELATED TO CLINICAL FEATURES AND IMAGING DIAGNOSIS

Thyroid eye disease affects eye function and causes loss of aesthetics. The disease typically accompanies Graves' disease, which can occur in people with hypothyroidism but is less common. Most Graves' eye disease has no clinical manifestations or is only mild and spontaneously regresses after taking antithyroid drugs. Severe forms affect 3%-5% of patients. Two age peaks of incidence are observed in the fifth and seventh decades of life, with slight differences between women and men [2]. This is an autoimmune disease due to the complex interaction between cellular immunity and humoral immunity [3], triggering the formation of specific antibodies in the eye against the TSH receptor expressed in the thyroid gland and orbital tissue (or orbit). Infiltration of B and T lymphocytes and other immune cells (monocytes, polymorphonuclear leukocytes, fibrocytes, and mast cells), T helper cytokines, TNF, and autoantibodies in the orbit play a major role in activating and maintaining severe inflammation in the orbit and periocular tissues, edema of the extraocular muscles and adipose tissue hyperplasia [4].

Graves' ophthalmopathy can be divided into two stages: active and inactive. Active ophthalmopathy is the inflammatory stage that progresses from 6 to 24 months, affecting the extraocular muscles and orbital adipose tissue [5]. Edema, inflammation, and accumulation of glycosaminoglycans increase orbital volume leading to proptosis, eyelid redness, conjunctival hyperemia, strabismus, corneal ulcers, or optic nerve damage. Widely used classifications for severity and activity include the NOSPEC classification, clinical activity score (CAS), European Group on Graves Orbitopathy (EUGOGO), and VISA (Vision, Inflammation, Strabismus, and Appearance) scores. In many cases, the progression of eye disease does not coincide with the thyrotoxicosis syndrome. Hyperthyroidism may remain stable, but active eye disease or exophthalmos can persists for many years after stopping antithyroid drugs.

Pathological biopsy provides the most accurate method for early diagnosis and staging of GO but has a high risk of complication. Imaging methods such as CT and MRI of the orbit provide fairly accurate information about retrobulbar lesions. MRI is superior due to its lack of radiation, and high soft tissue resolution, and recorded multiple parameters that allow better differentiation of soft tissue conditions than CT, including inflammation, steatosis, and fibrosis [6]. MRI of the orbit allows for clear visualization of pathologic changes including hypertrophy of the extraocular muscles, steatosis in the retrobulbar area, or fibrosis. MRI also detects early damage to the optic nerve (compressed optic nerves, optic neuritis, or stretched optic nerves) for early intervention to avoid permanent blindness. Therefore, an MRI of the orbit is very useful for doctors to choose the appropriate solution. It should be noted that there is always a 1-2 mm difference between the degree of exophthalmos measured by the Hertel exophthalmometer and the exophthalmos parameters on MRI [6].

3. INDICATIONS FOR ORBITAL DECOMPRESSION SURGERY IN TED

In 2022, the ATA and ETA issued a consensus on managing TED, supported by the American Academy of Ophthalmology (AAO) and the American Society of Ophthalmic Plastic and Reconstructive Surgery (ASOPRS) [7]. The guidelines specify indications for orbital decompression surgery, which is tailored to the disease phase. The active phase is most commonly treated medically with corticosteroids, orbital radiotherapy, and immunomodulatory agents to suppress inflammation. Surgical decompression in the acute phase is reserved for cases of medically refractory optic neuropathy. In the stable phase, rehabilitative surgery is sequenced to include orbital fat and/or bone decompression, strabismus surgery, and lastly eyelid surgery as required.

Thus, decompression surgery is indicated for (1) severe exophthalmos that causes eye deformity but is not active, correction and prevention of corneal disease due to an open lid, and (2) restoration of vision and restoration of optic nerve function if compressed. The goal of surgery in the treatment of TED is to reduce orbital pressure by removing fat; or opening one, two, three, or four bony walls with or without fat removal. Decompression surgery requires many specialties, including neurosurgery, otolaryngology, maxillofacial surgery, and ophthalmology. Surgeons need to determine the appropriate time for decompression surgery, individualize the decompression technique, monitor postoperative complications, and develop standards or tools to evaluate the effectiveness of the intervention.

4. ORBITAL DECOMPRESSION SURGERY

There have been many studies evaluating orbital decompression surgery but no consensus has been reached on the optimal surgical approach. There are no randomized controlled trials (RCTs) so it is not known which technique is better than the other. Synthesizing the analysis is difficult because the surgeon’s level of expertise, TED status, surgical indications, and assessment methods are all different. This surgery has evolved over 150 years, and through many improvements, the surgical technique is more sophisticated today. There are many different methods and techniques to remove the bone wall, remove the fat in the orbit, or both. Surgeons have more precise and effective bone removal tools. Furthermore, thanks to a full understanding of the pathogenesis of TED along with advances in computed tomography/MRI, doctors have decided to better individualize treatment. The success rate has increased significantly, and postoperative complications have decreased significantly. Before 2000, decompression techniques included [8]:

- Two and three-wall decompression: access from maxillary sinus - ethmoid sinus or three-wall including maxillary sinus - ethmoid sinus - outer wall (outer wall is also called lateral wall)
- Four-wall decompression: combining orbital-extradural decompression procedure removes part of all four orbital walls, incising around the orbit to let fat slide into the newly created cavity. This technique is only for patients requiring > 10 mm proptosis reduction.
- Remove fat from the inside and outside of the apex of the orbit. This technique is most effective for people with more adipose tissue proliferation than extraocular muscle hypertrophy. Removal of fat combined with the removal of the bone wall also helps reduce the exophthalmos further.
- Endoscopic transnasal decompression: the advantages of endoscopic surgery and an extra orbital approach is beneficial for patients with tight orbits or a globe at risk (eyes with prior corneal transplantation or glaucoma filtering procedure).
- Deep lateral wall decompression: remove bone along the lateral wall. If the thicker and deeper area of the lateral walls, removing the deep lateral wall will create a significant space outside the orbit. Only a small opening of the lateral wall can reduce the proptosis to the maximum.
- Transcaruncle orbitotomy [8],[9]: access to the orbit through the medial and inferior approaches to correct the fractured orbital wall and enlarge the orbit. However, the surgeon operates within a tight orbit making apical visualization difficult to see the apex of the orbit.
- Endoscopic medial wall, floor, and lateral walls decompression: this is an image-guided endoscopic balanced orbital decompression. This technique reduces double vision and increases the decompression efficiency [10]; for patients with optic nerve damage and who want to reduce proptosis by more than 6 mm.

The anatomy of the orbital bones is more sophisticated than just a simple bone box. Therefore, decompression surgery must be individualized based on the clinical symptoms and orbital bone anatomy. As currently conceptualized, the specialists view the following principles as key to customized orbital decompression: [8]:

1. Treatment of active phase TED is primarily medical, whereas stable phase TED is treated with elective surgery
2. All cases of proptosis are different. The relative contribution of volume expansion of the orbital fat hyperplasia and the extraocular muscles dictated the most appropriate operation.
3. Hertel measurements are not the best guide to surgical planning, and normative Hertel average are of no value.
4. Preoperative photos help to define the amount of proptosis reduction needed.

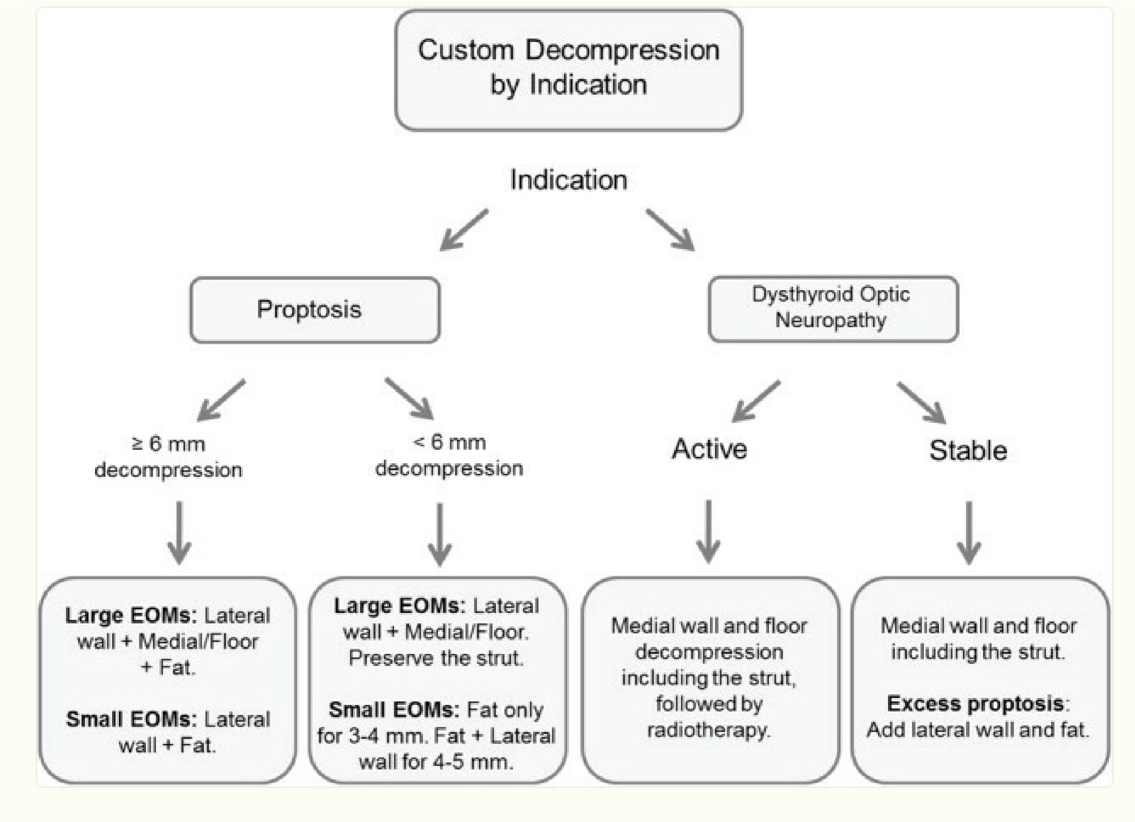

Fig.1 Custom orbital decompression by indications [8]. For example, if the indication is proptosis and a 6mm or more decompression is warranted for a patient with large extra-ocular muscles, a 3-wall decompression of the lateral wall, medial wall, floor, and the fat decompression is recommended.

Preoperative assessment is very important. First of all, surgeons should determine whether the operative indication is proptosis or dysthyroid optic neuropathy, and then determine the desired proptosis reduction. MRI of the orbit allows the surgeon to assess the ratio of the extraocular muscle to fat expansion and note the size and health of the sinuses. Finally, both surgeon and patients should understand the risks of post-operative strabismus, and the type of anticipated esotropia from the medial wall, hypotropia from the floor, and torsional diplopia from the extensive floor and medial wall decompression.

When the operation is defined, the choice of technique must be considered for each specific patient (Figure 1). Toodley further adds the following surgical tips [8]. For example, when decompressing the orbital floor, leave the floor lateral to the infraorbital nerve intact. Removal of the bone produces little additional volume expansion and increases the risk of infraorbital anesthesia. Leave the anterior floor medially to avoid hypoglobus and torsional diplopia. When deciding whether to remove the strut, consider the surgical goal. Remove the strut to maximize the decompression for the treatment of proptosis or dysthyroid optic neuropathy. In a 2017 study, of endoscopic two or three-wall decompression with and without strut removal, the mean reduction in proptosis was 3.6 mm for those with three-wall decompression with strut preservation vs. 7.65 mm for those with three-wall decompression with strut removal [11]. Similarly, in most cases, removing the periosteum maximizes surgical effect, but some cases develop postoperative diplopia, so the author recommends leaving the periosteum strip on the medial rectus muscle to reduce this risk. Failure of orbital decompression may occur due to preserved orbital bone, progressive orbitopathy, recurrent dysthyroid optic neuropathy, reformation of preoperative periosteal dimensions, or other optic neuropathy [8].

5. EVIDENCE OF THE RELATIVE EFFICACY AND COMPLICATIONS OF DECOMPRESSION SURGERY

Most of the published literature on orbital decompression consists of retrospective, cohort and case series studies. Because of the different stages of the disease, the treatment indications and methods used to evaluate the effectiveness are still inconsistent. There are no reports on the long-term effectiveness of decompression surgery. In other words, there is no consensus on which surgical techniques about the indications is the safest and most effective [12]. Currently, the main outcome measures include the postoperative decompression effect and the 6-month postoperative success rate; the secondary outcomes include the degree of eye mobility, eyelid abnormalities, improvement in NOSPEC grade, overall eye index, and CAS score. Postoperative adverse outcomes are recorded, including strabismus or postoperative vision loss or failure to continue treatment.

A large review of 3000 cases showed that orbital fat removal is safer than bony decompression, and the effectiveness is equivalent [13]. Removing 1 cc of orbital fat can reduce proptosis by 1 mm [8]. Fat removal alone can reduce proptosis by 4.2 mm in the short term after surgery and by 5.9 mm in the long term [5]. This technique reduces the rate of diplopia by 11% and reduces optic nerve compression in some cases [14]. In his review, Boboridis found that fat removal combined with osteotomy increases the effectiveness of surgery, but the statistically significant difference is only seen when the three osteotomies are removed [15]. Fat removal is less likely to cause diplopia and does not cause cerebrospinal fluid leakage, in contrast to decompression of the outer wall [5].

In cases of optic nerve compression, the first choice is high-dose corticosteroid infusion followed by emergency surgical decompression of the medial wall, if indicated, is the most effective solution [8]. The incision is made through the third eyelid to enter the medial wall, safely accessing the periosteum of the medial wall, removing the ethmoid bone, and minimizing nerve compression. This technique is equivalent to endoscopic access to the medial wall, which is considered superior to other decompression methods [9]. In patients with optic nerve compression and mild-moderate proptosis, a transconjunctival approach is recommended [16]. Several studies have shown that medial wall decompression reduces proptosis by 4.36 mm according to the Hertel scale [5]. Comparisons of the maxillary sinus, transnasal, transconjunctival, intranasal, or transcaruncular approaches [8] all reduce proptosis equally. However, Pleigo-Maldonado did not report whether transmaxillary sinus surgery is more effective in terms of CAS clinical activity or overall eye scale scores and secondary outcomes [17], and the transmaxillary sinus route has more complications such as diplopia (22/26 eyes vs. 13/18 eyes), infection (3/26 eyes vs. 0/18), and orbital nerve injury (13/26 eyes vs. 0/18).

In 2021, the Society of Ophthalmic Plastic and Reconstructive Surgery survey reported that lateral orbital wall decompression is a popular technique for the treatment of TED. 22.6% of surgeons preferred the single-wall procedure, and 36.8% of them chose only lateral wall decompression [18]. The lateral orbital wall decompression technique has many different incisions, minimal access from inside the eye or from outside the eye. The advantage of lateral wall decompression is that it is easy for the surgeon to access and see the orbital cavity, but there are not many RCTs to determine whether this technique is superior to others. The effectiveness of lateral wall decompression in reducing the apex pressure of the orbit (in cases with optic nerve compression) is not as high as that of opening the medial wall, but cutting the lateral wall reduces proptosis by 2.7-4.8 mm [5],[19]. Recently, deep lateral decompression has become more popular due to the response to decompression with minimal complications (combined with medial wall opening and fat removal) [5]. Postoperative diplopia after deep lateral wall surgery is only 0-8.6%, and a few other complications such as dry eyes, tremors, temporal hole sensation, rectus muscle injury, cerebrospinal fluid leakage [20].

The balanced decompression technique of removing the lateral and medial walls (with or without fat removal) is currently considered the most effective surgical method for severe proptosis without diplopia, reducing proptosis from 3.1-5.6 mm, with fewer complications [5].

For very severe proptosis, three-wall decompression is preferred (removing the lateral wall, medial wall, and floor) but has a higher risk of complications, while two-wall opening is for mild-moderate proptosis. A multicenter study of 139 euthyroid patients (n = 248 orbits) with inactive GO who underwent decompression surgery for disfiguring proptosis. Three-wall decompression resulted in higher reduction of proptosis compared to the tow-wall procedure (5.8 mm vs 4.6 mm) [21] or up to 7.5 mm, returning Hertel measurements to normal [22]. Anteroposterior three-wall decompression was more effective in reducing proptosis than transconjunctival lower eyelid three-wall technique. A linear regression analysis showed that the difference in surgical outcome was not due to preoperative exophthalmos. The three-wall were associated with more complications than two-wall decompression specifically in the coronal approach, mainly diplopia, and hypoglobus. Transconjunctival three-wall technique may cause maxillary sinus obstruction, hypoglobus, and persistent eyelid dissatisfaction in the swinging eyelid three-wall procedure [15]. Three-wall surgery via the anterior-posterior approach can cause temporal bossing, frontal muscle paralysis, and cerebrospinal fluid leakage. If three-wall surgery does not reduce optic nerve compression, consider orbital ceiling decompression, but it is not recommended because it is easy for brain herniation to further narrow the orbital cavity [6].

Removing the bone wall via coronal approach to decompress the optic nerve improved the CAS index and the overall eye score after 52 weeks of surgery by 17%, while the steroid group had a rate of 56% [21]. The trial included only 15 patients, randomly divided into 6 patients who underwent decompression surgery and 9 patients who used steroids. The surgical group had a temporary loss of sensation in the orbital area and 1 patient developed strabismus; immunosuppressants were still required during the 64-month follow-up (n= 5 patients), low strabismus correction surgery (n= 3), and then eyelid surgery (n= 5 patients). The steroid group had 5 patients who did not need decompression surgery but had many side effects of corticosteroids, 4 patients needed decompression surgery within 78 months of follow-up, low strabismus surgery (5 patients), and needed additional eyelid surgery (4 patients). Therefore, the long-term outcome of the surgical group to decompress the optic nerve seemed to be better and had fewer side effects. Another retrospective analysis of 81 patients who underwent decompression of the optic nerve through the coronal approach or through the eyelid showed that 19% had diplopia. The group that operated through the coronal approach had 20% of patients acquired abnormal eye movements (vs. 14% of the group that operated through the eyelid). The difference was not statistically significant. 12% of patients in the group operated through the anteroposterior approach had diplopia in many directions after surgery. 25% of patients needed strabismus surgery out of a total of 81 cases [23].

In Vietnam, a report at Hospital 103 in 2022 on 65 eyes with optic nerve compression underwent endoscopic orbital decompression surgery by opening the maxillary sinus, removing the entire anterior and posterior ethmoid, removing the trut bone, incising the lower wall of the orbit, pressing the eyeball from the outside to push the fat tissue passing through the bone opening. After 6 months, proptosis was reduced by 2 ± 1.06 mm, improved visual acuity by 3.1 Snellen lines, improved visual field, color vision, and papilledema [24]. Visual acuity improved early after surgery in the first week and was stable for up to 6 months. Complications were rare, such as bleeding in 9.6% of eyes, sinusitis in 3.1%, orbital cellulitis in 1.5%, and diplopia worse than 2.14%. Preoperative exophthalmos was correlated with postoperative proptosis reduction ($r= 0.51$, $p= 0.01$).

Also at Hospital 103 (2014), when evaluating orbital decompression surgery by incision of the entire lower eyelid into the medial wall and floor combined with fat removal in 65 eyes (43 eyes with optic nerve compression), the visual acuity increased by an average of two lines (on the Snellen chart) in the group indicated for optic nerve compression and increased by an average of 1 row in the group indicated for surgery due to proptosis. Proptosis reduced by 2.32 mm and 3.27 mm, in respective groups. Optic disc edema decreased or returned to normal 7 days after surgery in 91.4% of patients [25]. Lower eyelid retraction and lower eyelid deviation were significantly reduced by 47.7% (perhaps due to the technique of cutting the lower eyelid fascia). Postoperative diplopia occurred in 28.6% and 12.5%, respectively, in the two groups mentioned.

Other complications of orbital decompression surgery

Even with advanced surgical techniques, experienced surgeons still cannot completely avoid complications from mild to severe.

• Diplopia: due to the extraocular muscles not working together. After surgery, 18-29% of patients had diplopia, if only fat was removed, diplopia occurred in only 3.3% [5]. Diplopia is less common when decompressing the outer wall than other walls. Balanced decompression surgery can cause diplopia in 10-20%, with 3-wall surgery being the most common, 14-57% [5],[26]. Wide orbital floor excision can cause torsion diplopia

• Strabismus: medial strabismus may occur because the medial wall is removed, inferior strabismus when opening the orbital ethmoidal plate. Lateral decompression causes less postoperative strabismus than other walls and improves preoperative strabismus compared to balanced decompression [5]. Strabismus may spontaneously disappear after 3-4 weeks but recur if the disease continues to progress.

• Facial numbness: if the lateral wall is decompressed, facial numbness occurs in about 24%, usually mild and lasting < 3 months [26]

• Temporary/permanent paresthesia or loss of sensation in the area innervated by the infraorbital nerve: 24% [5]

• Voice changes

• Bleeding, hematoma, edema, sinusitis, corneal abrasions, cerebrospinal fluid leakage. The most common complications of endoscopic procedures are sinusitis, cerebrospinal fluid leakage, nasolacrimal duct injury, blepharoptosis, and failure to close the eyelids

• Blindness: the risk of blindness is the lowest [5],[15], only 0.09%-0.52% [5],[27].

Most patients only need decompression surgery once, the second decompression surgery is only 1.7-13.8%. The reason for re-surgery is usually proptosis or persistent optic neuropathy. Author Nguyen Chien Thang reported complications after open surgery of the inferior wall and floor such as bleeding 9.09%, lower eyelid entropion 4.54%, inflammation 2.2% and numbness in the sensory area of the maxillary nerve 36.36% [25]. Complications are all mild and easy to handle.

6. CONCLUSION

Orbital decompression surgery is performed for severe thyroid eye diseases with optic nerve damage that threatens vision and does not respond to corticosteroids. It can also be a cosmetic procedure for stable exophthalmos. Surgical techniques need to consider individual indications, preoperative orbital damages, and surgeon expertise to ensure good outcomes, minimize side effects, and improve the patient's quality of life. Future work will focus on the predictability of the decompression effect, mitigating the late recurrence of proptosis, reducing postoperative diplopia, and standardizing more predictable simultaneous decompression.

7. REFERENCES

1. Chen J, Li C, Teng W (2024). A Chinese survey of clinical practice on the management of thyroid eye disease. *European Thyroid Journal* 13 e230269
2. Wiersinga WM, Bartalena L (2002). Epidemiology and prevention of Graves' ophthalmopathy. *Thyroid* 12:855–860
3. Fang S, Lu Y, Huang Y (2021). Mechanisms that underly T cell immunity in Graves' orbitopathy. *Front Endocrinol* 12:648-732
4. Xavier NF, Lucena DR, Cruz AAV (2023). Monoclonal antibodies for the treatment of Graves orbitopathy: Precision medicine? *Ophthalmic Plast Constr Surg* 39 (4):307-315
5. Baeg J, Choi HS, Kim C (2023). Update on the surgical management of Graves'orbitopathy. *Frontiers in Endocrinology* (open access) doi. 10.3389/fendo.2022. 1080204
6. Song C, Luo Y, Yu G (2022). Current insights of applying MRI in Graves'Opthalmopathy. *Frontiers in Endocrinology* (open access) doi. 10.3389/fendo.2022.9915788
7. Burch HB, Perros P, Bednarczuk T (2022) Management of thyroid eye disease: A Consensus Statement by the American Thyroid Association and the European Thyroid Association. *European Thyroid Jour* 11(6):e220189
8. Tooley AA, Godfrey KJ, Kazim M (2019). Evolution of thyroid eye disease decompression – dysthyroid optic neuropathy. *Eye* 33:206-211
9. Shorr N et al (2000). Transcaruncular approach to the medial orbit and orbital apex. *Ophthalmology* 107:1459-1463
10. Kacker A et al (2003). Balanced orbit decompression for severe Graves' orbitopathy: A technique with a treatment algorithm. *Otolaryngol Head Neck Surg* 128:228-235
11. Finn AP et al (2017). A retrospective review of orbital decompression for thyroid orbitopathy with endoscopic preservation of the interomedial orbital bone strut. *Ophthalmic Plast Reconstr Surg* 33:334-339
12. Verity DH, Rose GE (2013). Acute thyroid eye disease (TED): principles of medical and surgical management. *Eye* (Lond) 27:308–3195
13. Richter DF, Stoff A, Olivari N (2007) Transpalpebral decompression

of endocrine ophthalmopathy by intraorbital fat removal (Olivari technique): Experience and progression after more than 3000 operations over 20 years. *Plast Reconstr Surg* 120:109-123
14. Prat MC et al (2015). Orbital fat decompression for thyroid eye disease: retrospective case review and criteria for optimal case selection. *Ophthalmic Plast Reconstr Surg* 31:215-218
15. Boboridis KG, Uddin J, Mikropoulus DG (2015) Critical appraisal on orbital decompression for thyroid disease: A systematic review and literature research. *Adv Ther* 32:595-611
16. Braun TL, Bhadkamkar MA, Jubbal KT (2017) Orbital decompression for thyroid eye disease. *Semi Plast Surg* 31:40-45
17. Plieo-Maldonado A, Miranda-Ruiz R, Vargas-Aguayo A (2000). Orbit decompression surgery in patients with exophthalmos caused by Graves-Basedow disease. *Gac Med Mex* 136:11–15
18. William SS, Sahu PD (2021). Surgical management of the orbit in thyroid eye disease: lateral orbital decompression. *Cur Opin Otolaryngl Head Neck Surg* 29 (4):289-293
19. Chang EL, Piva AP (2008) Temporal fossa orbital decompression for treatment of disfiguring thyroid-related orbitopathy. *Ophthalmology* 115:1613-1619
20. Cruz AAV, Equiterio BSN, Cunha BSA (2021) Deep lateral orbital decompression for Graves' orbitopathy: A systematic review. *Int Ophthalmol* 41:1929-1947
21. European Group on Graves Orbitopathy, Mourits MP, Bijl H (2009). Outcome of orbital decompression for disfiguring proptosis in patients with Graves' Orbitopathy using various surgical procedures. *Br J Ophthalmol* 93:1518–1523
22. Gioacchini FM, Kaleci S, Cassandro E (2021). Orbital wall decompression in the management of Graves'orbitopathy: A systematic review and meta-analysis. *Eur Arch Ortorhinolaryngol* 278:4135-4145
23. Paridaens A (1998) The incidence of diplopia following coronal and translid orbital decompression in Graves'orbitopathy. *Eye* 12:800-805
24. Phạm Thị Mỹ Hạnh, Nguyễn Chiến Thắng, Phạm Trọng Văn (2022). Nghiên cứu ứng dụng phẫu thuật nội soi giảm áp hốc mắt điều trị bệnh hốc mắt liên quan tuyến giáp. *Tạp chí Nghiên cứu Y Học 160* (12V2):236-245
25. Nguyễn Chiến Thắng (2014). Nghiên cứu ứng dụng phẫu thuật giảm áp hốc mắt điều trị bệnh mắt Basedow mức độ nặng. *Luận văn Tiến sĩ Y học chuyên ngành Nhãn Khoa năm 2014*, Trường Đại học Y Hà Nội.
26. Jefferis JM, Jones RK, Curri ZI (2018). Orbital decompression for thyroid eye disease: Methods, outcomes, and complication. *Eye* 32:626-636
27. Kansakar P, Sundar G (2020). Vision loss associated with orbital surgery – a major review. *Orbit* 39:197-208