Optometry: Open Access
ISSN: 2476-2075
Eleni, Optom Open Access 2023, 8:2
Research Article
Open Access

Eye Disease: Orbital Inflammatory Manifestation of Autoimmune Thyroid disease

Maria Eleni*

Department of Ophthalmology, Attikon Hospital, University of Athens, Greece

Abstract

Thyroid eye disease is an orbital inflammatory manifestation of autoimmune thyroid disease that results in orbital congestion and can lead to significant cosmetic disfigurement, diplopia, and vision loss. Typically, there is an active, inflammatory phase that transitions into a quiescent, fibrotic state. Management of this condition consists of regulation of the underlying thyroid disease, modulation of risk factors, supportive care for symptoms, and both medical and surgical treatment of ocular sequelae. Orbital decompression is generally indicated in 2 main subsets of cases: in active disease that includes ulcerative keratitis from severe corneal exposure or compressive optic neuropathy that does not resolve with high-dose corticosteroids, and in quiescent cases with persistent congestive or exposure symptoms and/or cosmetic deformity. Decompression may involve the medial wall, the lateral wall, the orbital floor, or any combination thereof, and this decision is dependent on surgeon preference and the overall goal of decompression. The medial wall is commonly selected due to the ease of approach, the potential for orbital volume expansion, and the opportunity for direct decompression of the optic nerve in cases of compressive optic neuropathy. Various surgical approaches to the medial wall have been proposed. The transcaruncular approach offers immediate access to the orbit with direct exposure, excellent visualization of the medial wall and the medial portion of the orbital floor, and the absence of cutaneous scars.

Keywords: Orbital decompression transcaruncular; Orbital decompression thyroid eye disease; Graves' disease; Graves's ophthalmopathy thyroid-associated; Orbito path surgical technique

Introduction

Thyroid-associated orbitopathy (TAO) can be functionally disabling and, in severe cases, may result in permanent visual loss. It may also cause significant facial disfigurement.

Although many authors use the term "cosmetic decompression," it must be kept in mind that this in fact represents reconstructive surgery because it addresses an abnormality caused by a disease. While decompression surgery has the potential to improve facial appearance, patients should be carefully informed that it is often impossible to restore their look to what it has been before the disease began to modify the tissues involved. Frequently multiple surgical procedures are required, ranging from orbital decompression surgery via strabismus surgery to lid surgery, again emphasizing that these procedures are not performed for the purpose of beautification [1].

It is important to note that most TAO patients will not require surgical treatment. In 1996 Bartley et al. demonstrated that only 20% of their patients had one or more surgical procedures. The cumulative probability of having surgery was initially 5% by 1 year after first diagnosis of the disease, rising to 9.3% by 2 years, to 15.9% by 5 years, and to 21.8% by 10 years. The need for surgery was significantly related to age, with a 2.6 times greater overall risk in patients older than 50 years.

Medical measures are the first-line treatment in the active stage of TAO. In the in-active stage of the disease or if medical therapy fails in sight threatening situations in active eye disease, the surgeon will be called upon to improve the patient's condition.

Clinical findings and indications for orbital decompression

Treatment of TAO requires an accurate assessment of disease activity, temporal progression, and severity. The aim of diagnosis is to differentiate the active stage-which represents a potential threatening of visual functions—from the inactive "burnt-out" stage of the disease [2].

Active moderate or severe congestive orbitopathy usually asks for immediate intervention, whereas active mild orbitopathy may only require supportive measures and a period of observation to discover whether disease is improving or worsening.

Sight-threatening dysthyroid optic neuropathy (DON) occurs in about 5% of patients with Graves' disease. Clinical findings can be loss of visual acuity or colour vision deficiency, visual field defects, relative afferent pupillary defect, or optic disc swelling. DON can be confirmed by visual evoked potentials with a significant increase in latency and/or reduction of amplitude. Without treatment, irreversible visual loss occurs in 30% of these cases. Older age, male gender, and smoking are important factors associated with an increased risk for DON.

The most widely accepted pathophysiologic mechanism for optic nerve involvement is compression of the nerve or its blood supply by the orbital contents in the orbital apex, mainly the extraocular muscles (EOMS). Many studies have shown a relationship between muscle size, restriction of motility, and DON, while proptosis itself did not correlate well with the risk for DON [3].

Because of the potential risk for blinding DON requires immediate intervention. Wakelkamp et al. demonstrated in a randomized clinical trial that in the event of DON immediate decompressive surgery does

***Corresponding author:** Maria Eleni, Department of Ophthalmology, Attikon Hospital, University of Athens, Greece, E-mail: MariaEleni@edu.com

Received: 03-Mar-2023, Manuscript No: omoa-23-91614, **Editor assigned:** 06-Mar-2023, PreQC No: omoa-23-91614 (PQ), **Reviewed:** 20-Mar-2023, QC No: omoa-23-91614, **Revised:** 25-Mar-2023, Manuscript No: omoa-23-91614 (R) **Published:** 31-Mar-2023, DOI: 10.4172/2476-2075.1000191

Citation: Eleni M (2023) Eye Disease: Orbital Inflammatory Manifestation of Autoimmune Thyroid disease. Optom Open Access 8: 191.

not result in a better outcome compared to medical immunosuppressive treatment. Therefore high-dose intravenous methylprednisolone therapy is recommended as the first-line treatment. However, if medical treatment does not improve visual functions within a few days or if there is a further deterioration, surgery appears to be the only way to avoid persistent visual loss due to optic nerve atrophy. The apical syndrome with congestion of the optic nerve in the orbital apex is best treated by a mechanical decompression that addresses the location of the compressive component, that is, by resection of the medial wall in the deep orbit [4]. In those rare instances where DON occurs in the absence of apical compression, increased orbital pressure may be a causative factor in the sense of an orbital compartment syndrome. Appropriate imaging techniques, for example, MRI, are mandatory for differentiating DON caused by apical compression or by compartment syndrome.

In the absence of DON elective reconstructive surgery for exophthalmos reduction or to relieve diffuse retrobulbar pressure sensation is usually considered after ophthalmological findings have been stable for at least 3–6 months. Early rehabilitative orbital decompression does not improve surgical outcome and is associated with a higher risk of induced motility problems. In general, if orbital decompression is needed, it has to be performed before EOM or eyelid surgery because it can affect both extraocular muscle balance and eyelid position.

The orbit is an enclosed cone-shaped compartment bounded by bone poster circumferentially and by the orbital septum anteriorly. The latter tight structure allows only limited forward displacement of the eye in response to increased orbital volume, such as what occurs in TAO. The intact orbital septum can withstand experimental pressures of 50 mm Hg and up to 120 mm Hg in some cases. By analogy with the pathophysiological processes described in a surgical and orthopaedic setting that terms an increased tissue pressure in an enclosed space as an "compartment syndrome, Kratky et al. first transferred this term to certain orbital conditions and described it as the "orbital compartment syndrome" in 1990: "Because of their confined anatomy, the orbital contents display the pressure-volume dynamics of a closed compartment. In some cases a significant rise in intraorbital pressure may compromise the perfusion of susceptible tissues and result in visual loss." Orbital pressure is measured to be 3–6 mm Hg in healthy individuals and at 7–15 mm Hg in TAO patients [5]. The final common pathway to visual loss in orbital compartment syndrome appears to be damage to the optic nerve fibres. Inadequate blood flow in the posterior ciliary arteries, the central retinal artery or vein, or the vasa nervorum of the optic nerve results in a variety of clinical presentations, including ischaemic optic neuropathy, central retinal artery or vein occlusion, or slow cavernous optic nerve degeneration.

Riemann et al. determined orbital tissue pressure of 4.0 ± 1.5 mm Hg in a larger series of 18 healthy orbits. Orbital compliance was 0.74 ± 0.31 mL/mm Hg after retrobulbar injection of local anaesthetic. In a later study the same authors showed that resting orbital tissue pressure was 9.7 ± 4.8 mm Hg in TAO patients and, of greater practical importance as a reaction to a further increase in volume, orbital compliance was significantly lower with 0.27 ± 0.21 mL/mm Hg. Resting orbital tissue pressure was even higher in TAO patients with DON, thus demonstrating the validity of this concept [6].

DON may result partially or in certain selected cases totally from an orbital compartment syndrome, and it should be kept in mind that orbital imaging without signs of apical EOM crowding and consecutive optic nerve congestion does not exclude DON. DON is a clinical diagnosis related to a disturbance of visual functions as reduction in visual acuity and/or colour vision, visual field defects, relative afferent pupil defects, and disc congestion. Patients with apical muscle crowding might benefit from medial wall decompression in the orbital apex, but this procedure is accompanied by a high risk of postoperative diplopia. In patients with no apical muscle crowding but with a presumptive orbital compartment syndrome the lateral technique should be considered with regard to the obviously missing influence on EOM motility.

Subjective pressure behind the globe as a sign of congestion is associated with a tight septum and a higher likelihood of DON. But it also becomes increasingly a more frequent indication for lateral wall decompression [7]. Postoperatively there is dramatic resolution of the congestive component of this condition due to the release of orbital pressure.

Experimentally, the amount of measured orbital pressure release depends significantly on two factors: first, on removal of the orbital wall and, second, on incision of the periorbita. Surprisingly, almost no increase in effect was achieved by adding further orbital walls.

The role of imaging techniques

In conjunction with the typical clinical signs of TAO, ultrasonography is sufficient to diagnose the condition. If B-scans show enlarged muscle bellies with normal tendon size, the clinical diagnosis of TAO is confirmed. Internal muscle reflectivity in A- and B-scans may be inversely proportional to disease activity.

Further information especially concerning the anatomical details and morphologic changes of the orbital soft tissues in the orbital apex can be assessed by computed tomography (CT) or magnetic resonance imaging (MRI). Magnetic resonance imaging (MRI) can be used to differentiate radiographically between active and inactive diseases [8]. In TAO the extraocular muscles are isointense to normal muscle on T1-weighted MRI and hyperintense on T2 depending on the degree of oedema. The absence of oedema may demonstrate a fibrotic phase. The correlation of water content (oedema) and inflammatory activity can also be detected with MRI short-term inversion recovery (STIR) sequencing. Latest results on the predictive value of the signal intensity ratio (SIR) in MRI-TIRM suggest a correlation between SIR and the clinical activity score (CAS). To differentiate patients with active from inactive eyes disease a cut-off value of >2.5 at 1.5 Tesla was determined.

The disadvantage of MRI is the poor visualisation of bony structures, making it less suitable as a preparatory assessment for decompression surgery. In comparison CT displays an excellent view of the bony orbit and paranasal sinuses; an information that is mandatory if orbital decompression surgery is being considered.

Bony Orbital Decompression (BOD)

Bony decompression may involve single or multiple walls of the orbit. Kikkawa et al. have proposed a "graded orbital decompression based on the severity of proptosis." Using the categories defined by Kalmann, these authors performed lateral orbital wall decompression with orbital fat removal if exophthalmos was less than 22 mm, additional medial wall decompression if exophthalmos was between 22 and 25 mm, and 3-wall decompression with removal of the orbital floor if exophthalmos was greater than 25 mm [9].

The use of a coronal decompression has been detailed in various publications. In most cases 3-wall decompression is attempted, which results in very effective exophthalmos reduction and improved aesthetic

outcome. The main advantage is that the incision can be hidden in patients with an adequate hairline. Hidden incisions are certainly preferable, but they can also be camouflaged by using an upper eyelid crease incision or swinging-eyelid approach for the lateral wall, an inferior fornix transconjunctival incision for the orbital floor, and a transcaruncular incision or endonasal approach for the medial wall. There has been a trend in recent years to abandon the coronal approach in favour of the alternatives mentioned.

As mentioned before two-wall decompression involving the medial wall and the medial aspect of the floor was still the most popular approach until the 1980s. The high incidence of postoperative diplopia because of an inferior globe displacement was avoided by preserving the inferomedial strut located at the junction of the maxillary and ethmoid sinuses.

"Balanced" decompression of the medial and lateral orbital walls has gained recent popularity because it may also lessen the occurrence of induced strabismus [10]. It is postulated that this approach may limit inferomedial displacement of the globe and produce an equivalent prolapse of the medial and lateral rectus muscles into the newly created space.

In a retrospective study Goldberg et al. demonstrated that balanced decompression is not more effective compared to deep lateral wall decompression alone in terms of average proptosis reduction. Interestingly, preoperative strabismus resolved spontaneously in 25% of cases in the balanced decompression group and in 60% of cases in the lateral decompression group. New-onset strabismus was found in 33% in the balanced decompression group compared to just 7% in the lateral wall decompression group. Goldberg et al. used CT to calculate the volume of bone available for removal in the deep lateral bony orbit. The "extended lateral orbit" was subdivided into three areas: the "lacrimal keyhole the "basin of the inferior orbital fissure", and the "sphenoid door jamb". The "sphenoid door jamb" makes the largest contribution to the total bone volume of the three areas potentially available for decompression. Proptosis reduction was as much as 6 mm. The authors estimated that 0.8 mm proptosis reduction might be achieved for every mL of bone removed.

An additional alternative for improving the effect on aesthetic rehabilitation is the insertion of subperiosteal orbital rim on lay implants, which are mostly used to camouflage remaining proptosis after decompression surgery. Possible risks include lower eyelid restriction, implant infection, and visible implant edges [11].

The usefulness of endoscopic techniques for medial orbital decompression is still under evaluation. In an early, small series Kennedy et al. reported improvement in visual acuity and globe protrusion in 9 out of 16 orbits. Lund et al. showed mean improvements in axial proptosis of 4.4 mm with an endonasal approach compared to 3.8 mm with an external procedure. Whether stereotactic navigation in decompression surgery as described by Miller and Maloof offers significant advantages remains to be proven.

Postoperative vision loss

The most worrying complication of orbital decompression surgery is postoperative worsening or even loss of vision, which may occur intraoperatively, related to vascular or pressure damage to the optic nerve or globe, and postoperatively, related to orbital haemorrhage or vasospastic ischaemia.

Irrespectively, gentle surgical technique and good haemostasis during orbital decompression surgery are mandatory. We always use a suction drain to remove fluid from the operation site because we feel that this can reduce the risk of visual loss. Visual loss never occurred in our own patient series after orbital decompression surgery though one patient did experience vision loss due to intraorbital bleeding the day after removal of an orbital apex haemangioma. However, if loss of visual impairment is recognized postoperatively, rapid evaluation for evacuable haematoma or ischemic event is compelling [12]. Patients should therefore remain in hospital until the drain has stopped evacuating blood or wound fluid and the drain has been removed. Although vision loss in orbital surgery is fortunately rare, it cannot be prevented entirely and must be kept in mind especially the first days after surgery.

Conclusion

A number of relatively safe surgical procedures for orbital decompression surgery currently exist, and the approach chosen will be governed by the experience available in the particular centre but should furthermore be tailored to the patient's needs. It is necessary to emphasize that proper decompression requires bracing or even removing the periorbit. The amount of proptosis reduction is influenced by preoperative Hertel values and is greater where exophthalmos is more severe.

Current trends in orbital decompression surgery account for the patients preoperative characteristics and intend to limit major complications. These include new-onset diplopia or worsening of preexisting motility deficits related to muscular fibrosis due to TAO and visible and disturbing scars which can be reduced or even avoided by camouflaging incisions. In the absence of DON we prefer the lateral wall decompression technique described above because of the following.

For the future a better understanding of the immunological and pathophysiological context of TAO should help to avoid severe and sight-threatening courses of the disease asking for aggressive surgical interventions in the active stage of the disease. But independently, the currently available surgical techniques overall represent save techniques to prevent blinding of the patient and furthermore to improve facial appearance and therefore to improve quality of life.

References

1. Jimenez JR, Ortiz C, erez-Ocon FP, Jim enez R (2009) Optical image quality and visual performance for patients with keratitis. Cornea 28: 783–788.
2. Ortiz C, Jimenez JR, erez-Ocn FP, Castro JJ, Gonzlez Anera R (2010) Retinal-image quality and contrast-sensitivity function in age-related macular degeneration. Curr Eye Res 35: 757–761.
3. Anera RG, Castro JJ, Jimenez JR, Villa C (2011) Optical quality and visual discrimination capacity after myopic LASIK with a standard and aspheric ablation profile. J Refract Surg. 27: 597–601.
4. Oshika T, Tokunaga T, Samejima T, Miyata K, Kawana K, et al. (2009) Influence of pupil diameter on the relation between ocular higher-order aberration and contrast sensitivity after laser in situ keratomileusis. Invest Ophthalmol Vis Sci 47: 1334–1338.
5. Fan R, He T, Qiu Y, Xu SY, Li YY, et al. (2012) Comparison of wave front aberrations under cycloplegic, scotopic and photopic conditions using Wave scan. Arquivos Brasileiros de Oftalmologia 75: 116–121.
6. Anera RG, Castro JJ, Jimenez JR, Villa C (2011) New testing software for quantifying discrimination capacity in subjects with ocular pathologies. J Biomed Opt 16: 123-127.
7. Jimenez JR, Ortiz C, erez-Ocon FP, Jim enez R (2000) Effects of alcohol and marijuana on dynamic visual acuity: I. Threshold measurements. Percept psychophys 18: 441–446.
8. Oshika T, Tokunaga T, Samejima T, Miyata K, Kawana K, et al. (2012) Visual functions and acute ingestion of alcohol. Ophthalmic Physiol Opt 16: 460–466.

9. Anera RG, Castro JJ, Jimenez JR, Villa C (2001) Effects of alcohol on performance on a distraction task during simulated driving. Clin Exp Med 33: 617–625.

10. Jimenez JR, Ortiz C, erez-Ocon FP, Jim enez R (2012) The effects of ingested alcohol on accommodative, fusional, and dark vergence. Percept psychophys 39: 25–31.

11. Fan R, He T, Qiu Y, Xu SY, Li YY, et al. (2013) Oral alcohol administration disturbs tear film and ocular surface. Ophthalmology 119: 965–971.

12. Oshika T, Tokunaga T, Samejima T, Miyata K, Kawana K, et al. (2012) Accuracy and usefulness of a breath alcohol analyzer. Ann Emerg Med 13: 516–520.