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Thyroid Orbitopathy: What the General Ophthalmologist Should Know

By Nancy Tucker, MD

Thyroid orbitopathy can be a devastating illness for the thousands of patients who develop this condition each year. The frustration for both patients and physicians relates to battling a disease that presently has few good treatment options and about which much remains unknown. However, there have been some advances over the past decade in both understanding its pathophysiology and in the treatment options for this illness. This issue of *Ophthalmology Rounds* provides an overview of thyroid orbitopathy and describes the recent therapeutic advances that may help the general ophthalmologist in dealing with this very challenging group of patients.

A variety of terms have been used to describe the constellation of eye findings that can occur in patients with thyroid disease and there is no universal agreement as to which descriptive term is the most appropriate. Graves' ophthalmopathy, infiltrative ophthalmopathy, thyroid eye disease, thyroid ophthalmopathy, thyroid orbitopathy, dysthyroid orbitopathy, endocrine ophthalmopathy, endocrine exophthalmos, and malignant exophthalmos are among the terms that have been used. This author prefers the term "thyroid orbitopathy," since the disease is known to occur in conditions other than Graves' disease, these patients may be euthyroid, and the orbit is the primary site of involvement.

Most patients with thyroid orbitopathy have Graves' disease. However, other thyroid conditions can be associated with the same constellation of orbital findings, including Hashimoto's thyroiditis, thyroid carcinoma, and primary hyperthyroidism. Approximately 40% of patients with Graves' disease have, or will develop, thyroid orbitopathy; but subclinical orbital alterations can be detected on ultrasonography or computed tomography (CT) in almost all cases. Of the patients with thyroid orbitopathy, approximately 80% are clinically hyperthyroid and 20% are clinically euthyroid.[1] Most patients with euthyroid Graves' orbitopathy will have some detectable laboratory evidence of subclinical hyperthyroidism.[2] In general, patients with euthyroid Graves' disease tend to have less severe orbitopathy.[3]

Orbitopathy generally presents within 18 months of the diagnosis of thyroid disease; however, it often occurs simultaneously. Its onset is unpredictable and can precede the diagnosis or occur many years after the onset of Graves' disease. Thyroid orbitopathy is usually a slowly progressive condition that may have a fluctuating course before stabilizing and eventually resolving. It may last from 6 months to several years.

Etiology

Thyroid orbitopathy is more common in women than in men (ratio 8:1) and usually presents between the ages of 30 and 50 years. There is an increased prevalence among smokers. Recently, it has been found that smokers are not only more likely to develop thyroid orbitopathy, but their orbitopathy tends to be worse and less likely to respond to steroid or radiation treatment. The mechanism that links smoking to thyroid orbitopathy is speculative. Possibly the decreased immunosuppression in smokers allows greater expression of autoimmune processes, or the resultant orbital hypoxia, thiocyanate, and nicotine-associated adrenergic stimulation of the thyroid gland may be contributing factors.

There is also a genetic predisposition and human leukocyte antigen (HLA) association in thyroid orbitopathy. There is an increased prevalence of HLA-B8 and HLA-DR3 in patients with Graves' disease and of HLA-DR5 in patients with Hashimoto's thyroiditis. The association with HLA is of no predictive value for the development of orbitopathy in patients with Graves' disease. In keeping with its underlying autoimmune nature, patients with thyroid orbitopathy may have other organ-specific or generalized autoimmune disorders. Finally, prior neck irradiation, myasthenia gravis, and superior limbic keratitis are risk factors for Graves' hyperthyroidism.[4]

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Thyroid orbitopathy is an autoimmune disease, but the precise nature of this autoimmune process, the source of the offending antibodies, and specific targeting of the thyroid gland, orbit, and pretibial skin in individuals with Graves' disease is still poorly understood and largely speculative. Several autoantibodies have been isolated and 3 of these can be measured routinely: the anti-microsomal antibody, the anti-thyroglobulin antibody, and the anti-TSH receptor antibody. It is not certain whether these antibodies play a major role in the pathogenesis of the eye disease or whether they are simply markers of the orbital autoimmune inflammatory process. Also, although antibodies can be isolated from most patients with thyroid orbitopathy, they are not present in all patients and often there is no correlation between the antibody level and the clinical progression or severity of the eye disease.

Most patients do not require antibody testing. In general, patients suspected of having thyroid orbitopathy should undergo standard thyroid function tests (TFTs) to detect hyperthyroidism. The thyroid-stimulating hormone (TSH) immunoradiometric assay reliably separates "normal" from" hyper" and "hypothyroid" states. It is the most useful first-line TFT in patients with thyroid disease. One strategy for laboratory testing is to obtain the TSH first. If the result is low, then hyperthyroidism is diagnosed. If the result is high, hypothyroidism is diagnosed. For either of these scenarios, overt disease can be distinguished from subclinical disease by measuring the free T_4. Although this stepwise approach is scientifically sound, from a practical point of view – to avoid the delay and inconvenience of the two-stage approach – most clinicians routinely order the free T_4 and the TSH together.

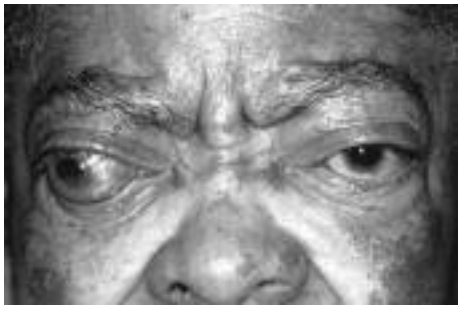
If the TSH is normal, the patient is said to be euthyroid. A euthyroid patient with obvious clinical findings consistent with thyroid orbitopathy need not undergo further laboratory testing; the diagnosis of euthyroid orbitopathy can be made on clinical grounds. However, if signs of possible thyroid orbitopathy are subtle or atypical, further investigations are indicated to substantiate the diagnosis. An orbital B-scan to assess muscle size and thyroid antibodies is helpful. TSH-receptor antibodies are present in 85% to 95% of patients with untreated Graves' hyperthyroidism. However, in the usual population of patients being tested (ie, euthyroid orbitopathy),TSH antibodies are found in only 50%, antimicrosomal antibodies in 60%, and antithyroglobulin antibodies in 30% of patients.[5] Thus, positive test results help support a diagnosis of autoimmune thyroid disease, but a negative test result certainly does not rule it out.

These and possibly other antibodies may be responsible for the clinical manifestations of thyroid orbitopathy. They most likely activate T- and B-lymphocytes in the orbital tissues, as well as in the thyroid gland. The earliest change appears to be inflammation of the endomysial connective tissues in the extraocular muscles. T- and B-lymphocytes then cause activation and proliferation of fibroblasts via a mechanism that is not completely understood, but is likely mediated by cytokines. Stimulated endomysial fibroblasts produce glycosaminoglycans and collagen, which cause edema and fibrosis.

Clinical manifestations

The clinical manifestations of thyroid orbitopathy are related to a triad of inflammation, edema, and fibrosis in a variety of orbital tissues, including the extraocular muscles, Müller's muscle, the capsulopalpebral fascia, the levator muscle and, to a lesser extent, the orbital fat and lacrimal gland. Involvement of the extraocular muscles is the most

Figure 1: Thyroid orbitopathy with proptosis and inferior corneal exposure keratitis



significant. The following structures are notably unaffected by direct inflammatory infiltration: the tendinous insertions of the extraocular muscles, the optic nerve meninges, and the globe itself.

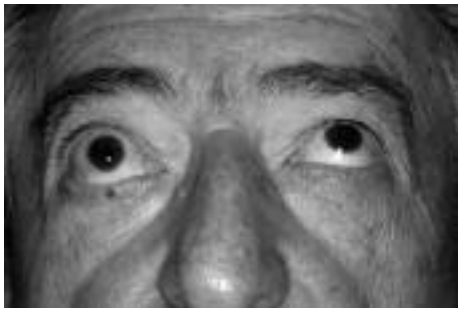
Eyelid changes: Thyroid orbitopathy is the most common cause of upper eyelid retraction, which may be due to adrenergic overaction of Müller's muscle or to fibrosis and functional shortening of the levator muscle. With minimal eyelid retraction, a misdiagnosis of ptosis of the opposite eyelid can easily be made. The upper eyelid retraction observed in Graves' disease has a characteristic temporal flare. Lower eyelid retraction may result from similar changes in the capsulopalpebral fascia. Proptosis accentuates the cosmetic appearance of both upper and lower eyelid retraction.

Soft tissue involvement: Careful examination in these patients may reveal eyelid edema, conjunctival chemosis, and injection of the conjunctival and episcleral vessels overlying the insertions of the extraocular muscles. The inflammation, enlargement, and fibrotic changes of the extraocular muscles can result in motility disturbances and diplopia. Other inflammatory signs such as eyelid and periorbital edema, conjunctival chemosis and erythema, and enlargement of the lacrimal gland are frequently seen.

Exophthalmos: Exophthalmos contributes to exposure keratitis and eyelid retraction (Figure 1). Thyroid orbitopathy is the most common cause of both unilateral and bilateral proptosis in adults. Typically, the proptosis is bilateral, but may be asymmetric. Thyroid orbitopathy results in axial proptosis; displacement of the globe in any other direction is suggestive of another diagnosis. Forward displacement of the globe occurs as the muscle and connective tissue volume behind the eye increases, elevating the pressure within the confines of the bony orbit. Proptosis is partially limited by the orbital septum and the posterior attachment of the extraocular muscles at the apex. When pressure within the retrobulbar tissues exceeds the forces counteracting proptosis, the rare complication of subluxation of the globe anterior to the eyelids may occur.

Extraocular muscle involvement: The most common motility abnormality is limitation of elevation owing to fibrosis of the inferior rectus muscle, which results in diplopia on upgaze (Figure 2). The second most common muscle to be clinically involved is the medial rectus, followed by the superior rectus/levator complex, and finally the lateral rectus. However, any single muscle or combination of muscles is possible. Diplopia is caused by fibrosis of the ocular muscles that prevents full extension when their antagonist muscles contract. If there is a history of variable diplopia, particularly if this is associated with a variable fatigue-related ptosis, the possibility of myasthenia gravis must be considered. Myasthenia gravis, which is

Figure 2: Restriction of extraocular movement in upgaze



also an autoimmune disease, occurs more commonly in patients with thyroid orbitopathy as compared to the general population.

The increased intraocular pressure measured during upgaze in patients with thyroid orbitopathy has been a controversial finding. When restriction of the inferior rectus muscle occurs, the intraocular pressure may increase by ≥6 mm Hg in upgaze as compared with primary gaze. Increased intraocular pressure in upgaze is a normal phenomenon exaggerated by thyroid orbitopathy. In patients with severe infiltrative disease, there is increased pressure on upgaze as compared with normal controls and patients with mild disease. Often, this is not an indicator of early disease because it occurs infrequently in patients with minimal eye findings.[6]

Corneal involvement: Corneal involvement due to exposure keratitis may result from proptosis, upper eyelid retraction, lower eyelid retraction, lagophthalmos, or a combination of these. Primary lacrimal gland dysfunction may also be present in Graves' orbitopathy. Although still speculative, there is some evidence of altered protein composition in the tears of patients with thyroid orbitopathy. This change might be caused by decreased tear production or by a general change in tear composition.[7] Exposure keratitis may range from minimal inferior punctate staining to severe keratitis and even corneal ulceration.

Optic neuropathy: The prevalence of optic neuropathy with visual loss in patients with thyroid orbitopathy is <5%.[8] Optic neuropathy is, however, the most common cause of blindness secondary to thyroid orbitopathy. Its onset is often insidious and may be masked by other symptoms. It is more common in older patients (age 50 to 70 years), males, and patients with diabetes. Optic neuropathy is usually bilateral, but up to one-third of cases may be unilateral.[9]

Most cases of optic neuropathy are due to compression of the optic nerve by enlarged extraocular muscles at the orbital apex. Patients with compressive optic neuropathy have more symmetric involvement of the extraocular muscles as compared with most patients with thyroid disease.[10] Although patients with optic neuropathy usually have proptosis, optic neuropathy can occur without significant proptosis in patients whose orbital septum efficiently limits anterior globe displacement despite increased retrobulbar pressure. Very rarely, optic neuropathy can occur without significant muscle enlargement. In these cases, it is postulated that a short optic nerve is being stretched or the optic nerve is being compressed by surrounding orbital fat.[11] These cases are so rare that optic neuropathy, in the absence of muscle enlargement or proptosis, should be investigated thoroughly for being unrelated to thyroid causes.

Although a history of decreased vision should be carefully sought, it is important to realize that optic neuropathy can occur in a significant number (18%) of patients with visual acuities in the range of 20/20 to 20/25. An afferent pupillary defect is present in 35%; an abnormal disc (either swollen or pale) is seen in only 52%; and visual field defects are present in 66%.[9] The Farnsworth-Munsell 100-hue colour vision test is a sensitive indicator of optic nerve dysfunction, but pseudoisochromatic screening procedures (eg, Ishihara plates) rarely identify an acquired colour defect unless optic neuropathy is severe.[9]

Management

The management of the patient with thyroid orbitopathy falls into 4 general categories.

- First, there is the patient with signs suggestive of thyroid orbitopathy, in whom the diagnosis has not been previously suspected.
- The second group includes patients with known Graves' disease who are referred by the endocrinologist prior to initiating I^{131} treatment.
- Next is the group with known thyroid orbitopathy being followed during their active/unstable phase.
- Finally, there is the group of patients with thyroid orbitopathy whose condition has finally stabilized.

Undiagnosed: Clinical presentations in which the possibility of thyroid orbitopathy is often overlooked include ocular irritation, lacrimation, and minimal eyelid retraction in early thyroid orbitopathy. Findings on examination include subtle upper or lower eyelid retraction, which may be unilateral or bilateral, unilateral ptosis, minimal proptosis, adult-onset diplopia, inferior exposure keratopathy, or mild unilateral ptosis. Unilateral ptosis is not a manifestation of thyroid orbitopathy, but patients with subtle unilateral lid retraction most frequently present complaining of a drooping eyelid on the opposite side. It is important to maintain a high index of suspicion in the middle-aged patient (30-50 years old), who does not wear contact lenses, and presents with new-onset unilateral "ptosis." In this clinical setting, upper lid retraction would be a much more common cause for the asymmetry than a unilateral ptosis.

In the group of patients with "possible" thyroid orbitopathy, other subtle signs on examination may help to substantiate the diagnosis. These include Von Graefe's lid lag (lag of the downward movement of the upper eyelid on slow downgaze), Griffith's lower lid lag on upgaze, Stellwag's incomplete and infrequent blinking, Kocher's spasmodic retraction of the upper lid during fixation, Rosenbach's tremor of gently closed lids, Gifford's difficult eversion of the upper lid, and Grove's resistance to downward pulling of the upper eyelid. To look for Grove's sign, the patient is asked to look down; the upper eyelashes are then grasped and the resistance to downward pulling is assessed. Grove's sign is positive when a significant amount of resistance is felt and indicates that the levator muscle is probably involved.

If TFTs are normal in patients with suspected possible thyroid orbitopathy, thyroid antibodies may be useful. If these fail to demonstrate an association, an orbital ultrasound performed by an experienced ultrasonographer is very helpful. Most patients with Graves' disease, even those without overt eye findings, have some ultrasonographic evidence of extraocular muscle involvement.[12] In this setting, ultrasound is more sensitive than CT or magnetic resonance imaging (MRI) to pick up minimal muscle enlargement.

It may also be helpful to distinguish between active and inactive disease.

If suspicion of thyroid orbitopathy is related to unilateral proptosis with increased resistance to retropulsion, a CT or MRI is indicated to rule-out an orbital tumour. A CT scan may also be helpful to distinguish thyroid orbitopathy from orbital inflammatory pseudotumour. The most characteristic CT finding in thyroid orbitopathy is fusiform enlargement of the extraocular muscles, which is usually bilateral, symmetric, with sharply defined borders, and sparing of the tendinous insertions. The pattern of muscle enlargement on CT parallels that seen clinically. The inferior rectus is most commonly involved, followed by the medial rectus, superior rectus, and lateral rectus. In contrast, orbital myositis/pseudotumour most commonly involves the medial rectus (57%), followed by the lateral rectus (36%).[13] It is more likely to be unilateral, involving a single muscle, with tendon involvement visible on ultrasonography or CT. Differentiating between myositis and thyroid muscle enlargement on CT, however, may be difficult because tendon swelling in myositis is not a consistent finding and multiple muscles may be affected.[14]

Diagnosed: The second group of patients commonly seen by ophthalmologists includes those sent by an endocrinologist for eye examination prior to starting I^{131}. The issue with these patients is whether or not they have pre-existing eye disease and whether steroids should be recommended. This became an important consideration following a publication in the *New England Journal of Medicine* in 1998.[15] It had previously been shown in 1992 that, compared to other forms of anti-thyroid therapy, I^{131} was more likely to be followed by the onset or exacerbation of thyroid orbitopathy.[16] The likelihood was approximately 15% as compared to 3% with propylthiouracil. Within the overall group of 15%, individuals who were most affected were those with pre-existing thyroid orbitopathy (25% as compared to 8%). Then in 1998, Bartelena et al reported that the increased risk of thyroid orbitopathy associated with I^{131} could be completely prevented with prednisone. It was recommended that patients with pre-existing thyroid orbitopathy receive steroid prophylaxis.[16] Nevertheless, oculoplastic surgeons appear divided with regards to recommending steroid prophylaxis. Some feel that the advantage of preventing progression of thyroid orbitopathy is not offset by the possible side effects associated with prednisone. This author's bias is toward steroid coverage for 2 weeks before, and a tapering course for 3 weeks after I^{131} treatment in patients with pre-existing thyroid orbitopathy.

Active thyroid orbitopathy: The third, and largest group of thyroid patients seen by the general ophthalmologist are patients with known active thyroid orbitopathy. The role of the ophthalmologist is to monitor these patients for serious complications of thyroid orbitopathy (eg, optic neuropathy and exposure keratitis) and to provide counseling and appropriate temporizing therapy until the disease stabilizes. Most patients will remain active for 12-18 months (although this is quite variable), then stabilize, and eventually even improve. During the active phase, patients are often frustrated by the apparent lack of concern their ophthalmologist seems to have in terms of initiating effective therapy as the severity of their condition continues to progress. The ophthalmologist should educate the patient regarding thyroid orbitopathy and the overall management strategy. The importance of smoking must be explained to the patient. In short, the single most important factor that will determine if a patient with Graves' disease develops orbitopathy is smoking. In fact, when compared to nonsmokers, smokers are 4 times more likely to develop orbitopathy; 4 times more likely to experience worsening of orbitopathy with I^{131}; and less likely to respond to treatment (with either prednisone or radiation).[17] Besides encouraging the patient to stop smoking, the general management strategy during the active phase of thyroid orbitopathy is basically to keep the patient as comfortable as possible until the condition becomes stable and definitive reconstructive surgery can be planned. The patient, nevertheless, needs to be monitored closely and treated if sight-threatening complications develop.

BoTox for Upper Eyelid Retraction: Symptoms of exposure keratitis-related to proptosis, eyelid retraction, and lagophthalmos can usually be controlled using ocular lubricants. If upper eyelid retraction is contributing to exposure symptoms and disturbing the patient, BoTox should be considered. BoTox can be injected via an anterior, or posterior approach. This author prefers the posterior approach since I feel that BoTox action in the orbicularis should be minimized. With decreased orbicularis tone, exposure keratitis, and lagophthalmos may worsen. This could be a problem if the upper eyelid retraction does not respond well, but partial 7th cranial nerve paralysis is induced. I inject posteriorly between the middle and outer third of the eyelid, just beneath the conjunctiva. I usually start with 5-10 units, and titrate upwards if necessary to achieve the desired lid height. The patient must understand the risk of overcorrection and resultant ptosis. In addition, not all patients with upper eyelid retraction respond to BoTox. Patients least likely to respond are those with significant levator and Müller muscle fibrosis. However, the results are usually excellent and patients feel encouraged that something is being done for them as they wait for the active phase to subside.

Steroids for diplopia: Diplopia during the active phase can also be disabling. If diplopia is associated with significant congestive thyroid orbitopathy, a trial of steroids is a good option. A short course of high-dose steroid (eg, 60 mg prednisone daily for 5 days) usually allows the ophthalmologist to determine if prednisone is likely to be helpful. If there is no response during this time, high-dose prednisone can be stopped safely without tapering. If some benefit is noted, the dose can be continued for an additional 1-2 weeks until maximum benefit is achieved. Prednisone is then tapered slowly over several weeks to months. Patients must be made aware of prednisone-related side effects and the possibility of adverse reactions, including diabetes, hypertension, osteoporosis, and avascular necrosis of the hip. Over the short-term, patients are most likely to notice increased energy, difficulty sleeping, increased appetite, and weight gain. Steroid psychosis rarely ensues.

Patients responding well to prednisone should be slowly tapered off medication. This can take several weeks, but more commonly several months. If continued tapering is impeded by recurrence of symptoms on lower doses or prednisone-related complications, radiation treatment should be considered. Patients are often alarmed at the mention of radiation, believing that they will become sick and lose their hair. The ophthalmologist can be instrumental in reassuring the patient that the medical risks of remaining on long-term prednisone far outweigh the risk of low-dose radiation. The standard regimen involves 2,000 rads (given as 200 rads in 10 treatment sessions over a 2-week period). When patients understand that this dose is generally associated with only temporary ocular dryness and minimal risk of cataract, they are more receptive to this alternative. In most cases of thyroid orbitopathy, radiation is a one-time-deal. However, in severe cases, radiation may be used for severe congestive thyroid orbitopathy initially, and then later for compressive optic neuropathy. In this case, complications of radiation associated with an additional 2,000 rads are cumulative upon the initial 2,000 rad treatment. At 4,000 rads, the patient is likely to develop a cataract and is at a low risk of developing radiation retinopathy and/or optic neuropathy. These more serious complications become more likely at 6,000 rads.

Radiation: Orbital radiation treatment does not produce immediate results. In fact, there is generally no noticeable effect for the first 2 weeks, frequent worsening by the third to fourth week, but gradual improvement by the fifth to sixth weeks. Further improvement continues for up to 3 months. Given this pattern, it is wise to prescribe prednisone for the first month, and then slowly taper it over the following weeks. Improvement in diplopia, and proptosis is variable, occurring in approximately 50% of patients. The efficacy of radiation treatment for moderate thyroid orbitopathy has recently been questioned.[18] However, many oculoplastic surgeons believe that it can be effective if used judiciously in select patients. In general, patients with acute inflammatory disease who have responded well to prednisone comprise the same acute inflammatory group that will respond to radiation.

Optic neuropathy: The most important aspect of managing the patient with active thyroid orbitopathy is maintaining a high index of suspicion for optic neuropathy. Certain patients are more prone to develop compressive optic neuropathy. As one would suspect, patients with significant congestive thyroid orbitopathy with restriction of extra-ocular movement and proptosis, are at risk. However, there is a significant number of patients without severe orbitopathy who are also at risk. Risk factors include older age, male gender, and patients with diabetes. If thyroid orbitopathy is suspected, an abnormality of the visual field should be sought. A CT scan with particular attention to the orbital apex on coronal views is also important for detecting apical crowding. This is almost always present and strongly supports a diagnosis of thyroid compressive optic neuropathy. Close monitoring of visual acuity and visual fields is useful to determine if the optic neuropathy is improving or progressing. This is a reversible cause of vision loss if detected and treated in a timely fashion. In general, the more dense the visual loss, the more quickly the patient will progress to irreversible vision loss. A mild visual deficit may be observed conservatively for weeks, or even months, without adverse effects. However, optic neuropathy can be complicated by rapidly progressive severe visual loss and, therefore, once diagnosed, treatment should be instituted.

Although there are 3 treatment options for compressive optic neuropathy (steroids, radiation, and orbital decompression), there is no general consensus regarding its preferred management. Some centres recommend radiation or even surgery as first-line therapy; however, most recommend a trial of steroids as the initial approach. My own treatment strategy is as follows. If the patient has a good response to prednisone, but the benefit is not maintained with tapering or adverse effects of prednisone ensue, radiation is recommended. However, it is important to keep in mind that, following radiation, the onset of a beneficial effect will only be apparent 6 weeks later. Therefore, if the visual loss is significant, a more prompt visual rehabilitation is necessary to avoid permanent visual loss and radiation may not be appropriate. If no visual improvement occurs with prednisone, or visual loss is still significant despite some improvement, one should quickly proceed to surgical orbital decompression. Orbital decompression for optic neuropathy is generally a 3-wall decompression with particular attention being paid to decompression of the most posterior ethmoidal air cells. Visual improvement may take several days, and continued improvement can occur over several weeks following successful orbital decompression. If visual loss prior to decompression is not longstanding, most patients will enjoy complete visual recovery. Less commonly, incomplete visual recovery occurs, despite adequate surgical decompression. In these cases, adjunctive radiation is an option.

Inactive thyroid orbitopathy: Once thyroid orbitopathy has stabilized, reconstructive surgery can be planned for patients with persistent proptosis, diplopia, or eyelid retraction. This is the most common group of thyroid patients seen by oculoplastic surgeons. At this point, patients are much easier to deal with since they have gained an understanding of their disease and their expectations are more realistic. They are now happy to have finally arrived at the stage where definitive plans for surgical rehabilitation can be offered.

Surgical rehabilitation is a stepwise process. In patients with significant proptosis, diplopia, and eyelid retraction, the first surgical intervention should be orbital decompression, followed by strabismus surgery, correction of upper and/or lower eyelid retraction, and finally by blepharoplasty if indicated. Of course, not all of these surgeries are necessary in every patient. Surgeries are generally separated by approximately 6 weeks and most oculoplastic surgeons perform orbital decompressions one at a time. The advantage of this stepwise approach is that it minimizes the total number of surgeries and revisions necessary to achieve the desired end result.

Orbital decompression may involve 1, 2, or 3 orbital walls, depending on the severity of the proptosis. As well, orbital fat decompression can be done alone or in conjunction with bony decompression. Fat decompression will generally achieve an additional

1-3 mm of reduction in proptosis (average 2 mm). Strabismus surgery generally involves recession rather than resection of the extraocular muscles. Many surgeons prefer adjustable sutures in thyroid patients. Correction of lower eyelid retraction requires the use of a spacer material. Autogenous grafts such as upper eyelid tarsus, ear cartilage, hard palate, and nasal septum are good choices. Banked tissue such as sclera must be oversized, as contraction is more pronounced with autologous material. Correction of lower eyelid retraction is more difficult if proptosis has not been properly addressed. In this setting, it is important not to horizontally tighten without adequate vertical support, as the lower eyelid may be pulled under the ocular globe, (analogous to tightening the belt around a beer-belly). Correction of upper eyelid retraction does not require spacer material. Müller's muscle can be excised posteriorly, and/or the levator aponeurosis can be detached anteriorly. Blepharoplasty surgery is the final step. General principles of surgery apply; however, it is important to realize that the fat is frequently more vascular and fibrotic. Care must be taken not to be overly aggressive with skin removal in lower eyelid blepharoplasty since these patients may be at an increased risk of lower eyelid retraction, particularly in the setting of pre-existing residual proptosis.

Conclusion

The above-described nonsurgical and surgical approaches to thyroid orbitopathy should be considered only as guidelines, since the condition is highly variable and each case must be considered individually. There is no general consensus as to the best nonsurgical or surgical approach. As the understanding of this autoimmune disease grows, more specific treatments will likely become available in the future. Until then, this article provides a framework to help manage patients with thyroid orbitopathy.

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Upcoming events

Oct. 19, 2006	VPP Dr. Richard Abbott "Medical Error and How It Has Affected Our Professors"
Oct. 26, 2006	VPP/Combined TOS Rounds Dr. Eric Donnenfeld, Rockville Centre, NY "Multifocal IOL's"
Nov. 2, 2006	VPP Dr. Alex Levin "Trying That New Procedure: Is It Ethical?"
Nov. 11-14, 2006	American Academy of Ophthalmology, Las Vegas, Nevada
Nov. 16, 2006	VPP Dr. Wai Man Chan, Kowloon, Hong Kong "Central Serous Choroidopathy Myth and Reality"
Nov. 23, 2006	VPP Dr. Alan Bird, London, England "AMD"
Nov. 30, 2006	VPP Dr, Jose-Luis Tovilla, Mexico City, Mexico "Facial Palsy"
Dec. 8-9, 2006	**Update in Medicine and Ophthalmology** The Old Mill Inn, Toronto, Ontario **Advance Notice/Brochure Link:** http://www.cme.utoronto.ca/PDF/OPT0602.pdf Phone: 416.978-2719 Fax: 416.946-7028 Toll Free: 1.888.512.8173 Email: ce.med@utoronto.ca Website: www.cme.utoronto.ca

Note: VPP rounds (September 2006 to May 2007) will be held at Sunnybrook Health Sciences Centre, 2075 Bayview Avenue, Toronto, 'E' Wing, Ground Floor, Room EG61 at 5:30PM – 7:30PM.

Disclosure Statement: Dr. Tucker has stated that she has no disclosures to announce in association with this publication.

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