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Combined endoscopic and subciliary orbital decompression for thyroid-related compressive optic neuropathy*

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SUMMARY

Compressive optic neuropathy is a feared, although unusual, complication of thyroid-related orbitopathy. A variety of surgical approaches have been described to achieve orbital decompression and alleviate the hallmark apical orbital crowding of this condition. We describe a subciliary anterior orbitotomy approach to the floor combined with an endoscopic medial wall resection. The anterior orbitotomy allows removal of the bones of the orbital floor both medial and lateral to the canal of the infraorbital nerve. The anterior orbital floor is retained for globe support. This combined approach retains the low morbidity of the endoscopic operation while achieving increased apical medial orbital wall and orbital floor decompression. We describe two illustrative cases where this approach produced a dramatic improvement in visual function. The surgical refinements associated with this combined approach offer technical advantages over other operations in the treatment of thyroid-related compressive optic neuropathy.

Keywords: orbital decompression, thyroid-related optic neuropathy

INTRODUCTION

The association between orbital and thyroid disease has been noted for nearly 200 years (Graves, 1835). Not the least of the controversies surrounding this condition is what it should best be called. It represents an orbital process so that "orbitopathy" is more appropriate than "ophthalmopathy." Although the association with thyroid disease is not absolute, at least by current serological testing, it is sufficiently frequent as to warrant its retention in the title. Consequently, the condition is, perhaps, best referred to as "thyroid-related orbitopathy."

Thyroid-related orbitopathy affects a predominantly female population. It is four or five times as common in women (Rootman, 1988) and is associated with the HLA-DR3 genotype in a number of cases (Farid et al., 1979). The disease has a presumed autoimmune basis, perhaps because of shared aspects of antigenicity between the thyroid gland and orbital tissue in general and the extra-ocular muscles in particular (Salvi et al., 1988). Eighty per cent of cases occur during or after an episode of hyperthyroidism (Bartley et al., 1996). The pathological appearance will vary between patients and in the same patient at different stages of the disease process, but is generally characterized by inflammatory cell infiltration, mucopolysaccharide deposition, collagen deposition and fibrosis.

An understanding of the natural history of the disease is important in clinical decision-making. The course of activity of thyroid-related orbitopathy is usually between 6 and 30 months. Although there may be day-to-day fluctuations in the state of the disease, recurrence of active disease is rare. The clinical signs (proptosis and eyelid retraction) however persist, even when the disease is quiescent. Failure to understand the largely self-limited nature of the disease process may lead to rather too-dramatic treatments being employed when patients are acutely symptomatic. The clinical signs of thyroid-related orbitopathy range through eyelid retraction, periorbital oedema, chemosis, exophthalmos and extra-ocular muscle dysfunction to compressive optic neuropathy (Werner, 1969; Bartley et al., 1996). CT scans nicely demonstrate enlarged extra-ocular muscle volume with characteristic fusiform muscle enlargement and sparing of

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the tendons. These studies typically provide an objective evaluation of the degree of exophthalmos and optic nerve stretching and help differentiate the diagnosis from orbital myositis, where both the extra-ocular muscle and its tendon tend to be enlarged.
Most patients with thyroid-related orbitopathy do not need surgery. They can often be managed successfully by employing a number of measures, such as head-of-bed elevation at night, cool compresses, ocular lubricants and moisture chambers. Patients may also be managed by surgery, short of orbital decompression. These operations have the goal of providing ocular surface protection as well as resolving eyelid retraction. These procedures include upper eyelid-lengthening by way of levator muscle recession with muellerectomy or lid-resetting procedures, such as lower eyelid retractor extirpation with spacer grafts.
A small percentage of patients with thyroid-related orbitopathy go on to develop vision-threatening compressive optic neuropathy. Rootman, in a review of 675 patients with thyroid-related orbitopathy (1988), found an 8.6% incidence of compressive optic neuropathy. Compressive optic neuropathy often develops insidiously. The patients may not be aware of the earliest symptoms of this stage of the disease, such as desaturation of colour vision, which may only be evident on specific colour testing. Treatments for compressive optic neuropathy have included plasmaphoresis (Kelly et al., 1983) and cyclophosphamide, based on the presumed autoimmune nature of the condition. Corticosteroids and radiation, however, form the two mainstays of nonsurgical means to decompress the orbit. Corticosteroid therapy is initiated with doses of prednisone, ranging from 60 to 120 mg/day, with gradual titration downward when a response is achieved. A response can be achieved in most patients, the difficulty being maintenance of the response on lower doses. Orbital irradiation is employed for slightly different indications and with different timing in different institutions. We employ orbit radiation for "hot" congested orbits characterized by marked chemosis, injection and swelling. Radiation is directed at the posterior two-thirds of the orbit only, taking precautions to shield the anterior portion of the globe (Petersen et al., 1990). A course of 2,000 rads is administered in 10 fractions. Large series have shown a response rate approaching two-thirds. Our patients receiving radiation are also placed on steroids, as radiation may acutely cause an increase in orbital swelling. Beneficial effects of radiation are seen in 4-6 weeks.
While the often-cited article by Walsh and Ogura (1957) included corneal changes, ophthalmoplegia, chemosis and cosmesis as indications for orbital decompression, we have reserved surgical decompression in the majority of cases for patients with failing vision. Surgical decompression of all walls of the orbit has been described. Medial wall decompression was described by Sewall in 1936; lateral wall surgery was described in 1889 by Kronlein, and superior decompression was described in 1931 by Nafzinger, a neurosurgeon. Walsh and Ogura (1957) described a series of transantral removal of the medial orbital wall and medial aspect of the orbital floor. Most recent interest has focused on Kennedy's description of endoscopic decompression (Kennedy et al., 1991) and Trokel's description of orbital decompression by orbital fat removal only via an eyelid crease or subciliary incision (Trokel et al., 1993). Trokel's technique achieves minimal decompression and is not broadly applicable to all degrees of disease. Khan et al. (1995) described a combined external ethmoidectomy and transconjunctival approach for orbital decompression.

MATERIAL AND METHODS

The operation we use employs a combination of endoscopic medial wall decompression and an anterior orbitotomy for removal of the orbital floor. The anterior orbitotomy can be performed by either a subciliary or transconjunctival approach. Pre-operative ophthalmological evaluation included assessment of visual acuity, colour vision, ocular motility, eyelid position, visual fields and Hertel's measurements. The otolaryngologist performs a nasal endoscopy to assess septal position and surgical access as well as to exclude other pathology. The pre-operative sinus CT scans are reviewed to look at the size and configuration of the sinuses and other coincident disease. The patients are photographed and endocrinological evaluation is obtained in all patients.
The operation is performed under general anaesthesia. The nose is endoscopically packed with 0.05% oxymetazoline hydrochloride-soaked neuropledgets after haemostatic injections of 1:100,000 epinephrine. A subciliary incision is marked and similarly injected. A 4-0 silk suture is placed through the eyelid margin and secured to place the lower eyelid on stretch. An incision is made and a skin muscle flap is raised, developing a plane between the orbicularis muscle and orbital septum. Care is taken not to breach the orbital septum to prevent inconvenient periorbital fat herniation into the operative field. The dissection is extended to the orbital rim where the periosteum is sharply incised along its anterior face and elevated, exposing the bones of the orbital floor.
Attention then turns to the medial orbital wall. Under endoscopic control, an uncinectomy is performed and the natural ostium of the maxillary sinus positively identified. A total ethmoidectomy is performed. A so-called "mega-antrostomy" is fashioned. This involves enlarging the natural ostium at the expense of each of its dimensions, except anteriorly. The antrostomy is enlarged posteriorly to the back wall of the maxillary sinus, inferiorly to the inferior turbinate and superiorly to the orbital floor. The thin bone of the medial orbital wall is then endoscopically removed, except for two areas. Bone adjacent to the frontal recess is maintained. A small strut of bone is also maintained anteriorly at the junction between the medial wall and the orbital floor. This helps to reduce rotation of the decompressed globe and may decrease the likelihood of post-operative diplopia (Goldberg et al., 1992). This strut is remote from the apex and does not interfere with the surgical goal of apical decompression. The medial orbital wall is taken down back to the face of the sphenoid. The middle turbinate is retained.
From above, through the subciliary incision, the orbital floor is removed. A small hemostat is used to puncture the medial portion of the floor. Using a Freer elevator, thin segments of the bony floor are removed. Thicker segments are removed with

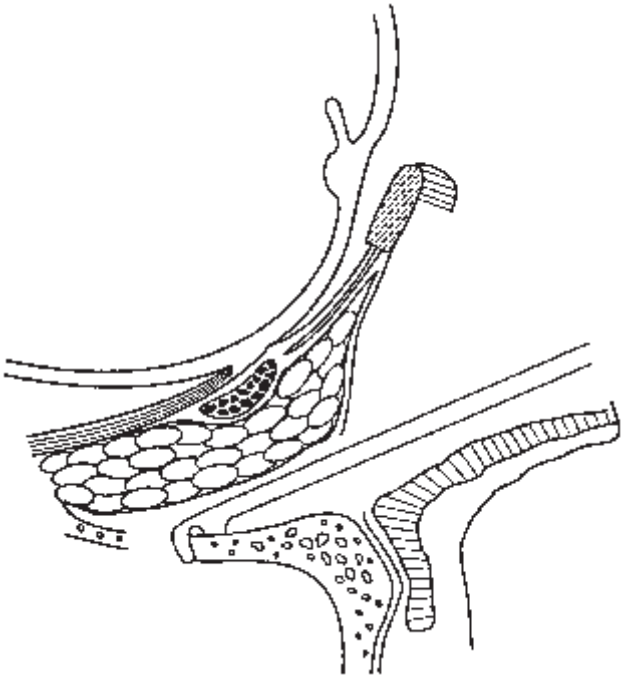

Figure 1. Removal of bones of orbital floor through an anterior orbitotomy approach via a subciliary eyelid incision.

rongeurs (Figure 1). Bone can be removed medial and lateral to the infraorbital nerve. The infraorbital nerve is left within the infraorbital canal anteriorly; however, all of the surrounding bone is removed posteriorly. The anterior 15% to 25% of the orbital floor is maintained to avoid globe ptosis. The posterior extent of the orbital floor removal is the posterior wall of the maxillary sinus. After removal of all required bone, the periorbita is incised. This is performed at the extremities of the operative field initially so that prolapse of orbital fat does not obscure the surgeon's subsequent view. A custom-bent vitrectomy knife is used to incise the periorbita along the medial wall and floor, beginning at the apex, to achieve a balanced opening of the periorbita. The incisions are made in the quadrants between the extra ocular muscles. Gentle orbital pressure enhances the prolapse of fat, as does division of any residual periorbital bands. The periosteum is closed at the orbital rim and the subciliary incision is closed. No nasal packing is used. The patient's visual function is carefully monitored as soon as the patient is able to cooperate.

RESULTS

Case 1

A 54-year-old woman with a 5-year history of hyperthyroidism treated with ^{131}I and levothyroxine replacement presented with a 1-year history of thyroid-related orbitopathy. Three months prior to this evaluation, the vision in her left eye began to decrease but was initially attributed to cataract formation. The vision in the left eye continued to steadily worsen. Presenting visual acuity was 20/20 in the right eye and 20/80 in the left eye with an afferent pupillary defect on the left. Visual field testing showed a left central and paracentral scotoma (Figure 2A). Computed tomography showed bilateral extra-ocular muscle enlargement with crowding of the orbital apex.

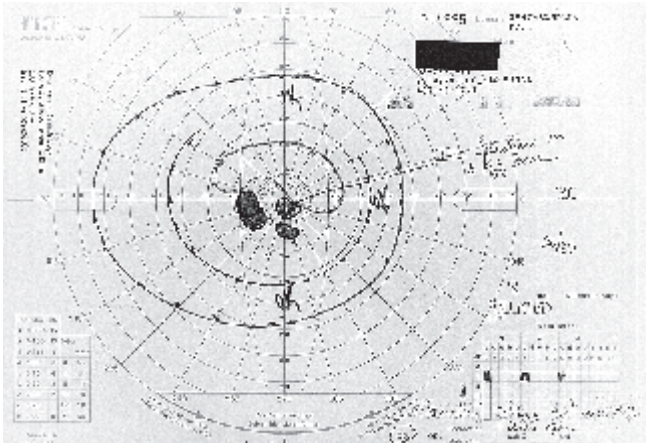

Figure 2A. Case 1, pre-operative visual fields.

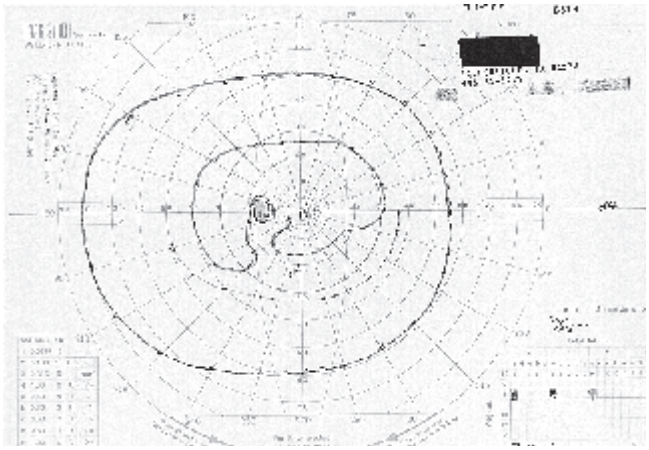

Figure 2B. Case 1, post-operative visual fields.

The patient was treated for compression optic neuropathy with corticosteroids, initially intravenously (125 mg, every 6 h) for 24 h, then orally (80 mg prednisone, once daily) for five days, with stabilization of symptoms. A combined approach left orbital decompression was performed 10 days later, as described. One week post-operatively, her visual acuity was improved to 20/30 in the left eye with resolution of the visual field defects (Figure 2B). At the 6-month follow up, the visual acuity was 20/25 with a normal visual field. Hertel's measurements had improved to 19 mm from 24 mm prior to surgery. She has maintained this level of visual function for 18 months. The only post-operative complication was a left esotropia and hypotropia that was corrected one year later with strabismus surgery.

Case 2

A 66-year-old man with a 10-month history of hyperthyroidism treated with ^{131}I and levothyroxine replacement presented with a 6-month history of thyroid-related orbitopathy and recent visual loss bilaterally. His visual acuity was 20/80 in the right eye. In his left eye, he was only able to count fingers at three feet. An afferent pupillary defect was present on the left. Visual field testing showed bilateral central and paracentral scotomas (Figures 3A and 3B). Computed tomography was consistent with thyroid-related orbitopathy. This patient was started on intravenous corticosteroids (125 mg, every 6 h) followed by oral prednisone (80 mg, daily) to medically decompress the orbits. One week later, his visual acuity had improved to 20/20 in the

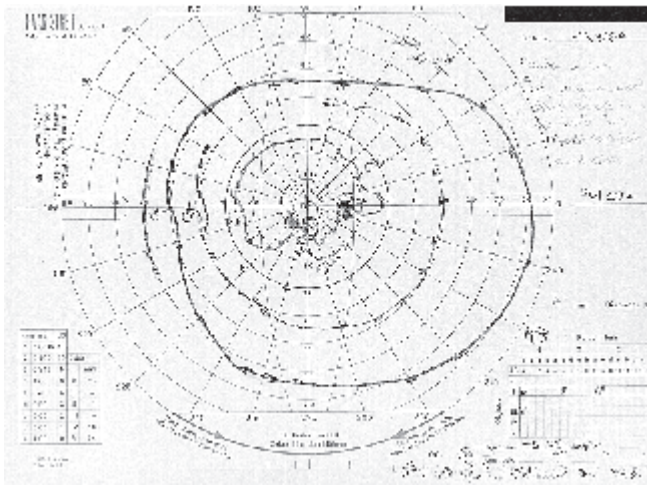

Figure 3A. Case 2, pre-operative visual fields, right eye.

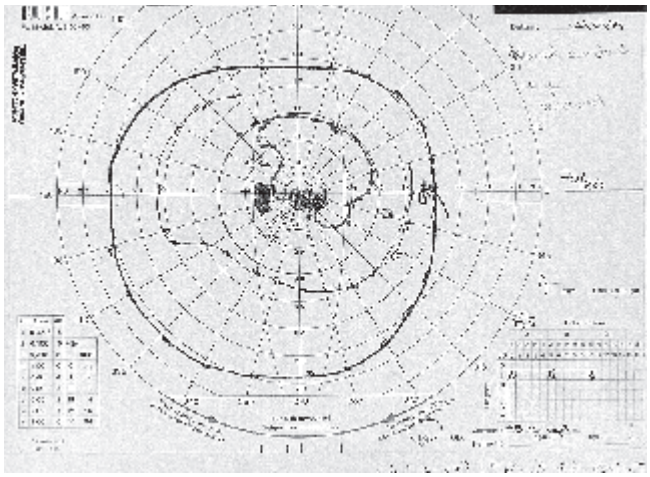

Figure 3B. Case 2, pre-operative visual fields, left eye.

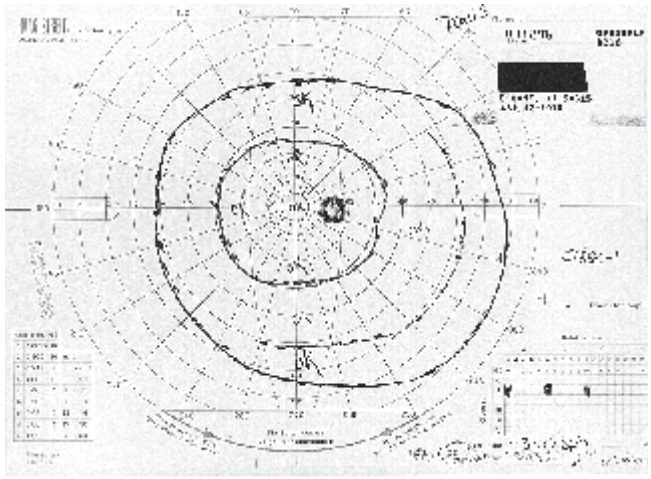

Figure 4A. Case 2, post-operative visual fields, right eye.

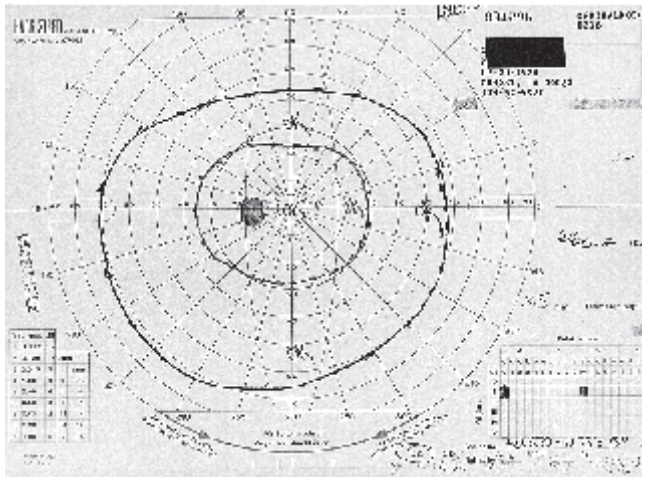

Figure 4B. Case 2, post-operative visual fields, left eye.

right eye and 20/50 in the left with minimal change in his visual fields. The patient elected to have bilateral radiation treatments and this was done with 2,000 rads in 10 fractions. The corticosteroids were continued but slowly tapered during the radiation treatments. The patient returned two weeks after being off steroids and two months after radiation treatments with a complaint that the vision in both eyes was decreasing again. On examination, his visual acuity was 20/30 in the right eye and 20/60 in the left, with worsening of the visual field defects (Figure 3A). Oral corticosteroids were initiated again and the patient underwent bilateral combined approach orbital decompressions. On the 1-week follow-up visit, the visual acuity had improved to 20/20 in the right eye and 20/25 in the left. The corticosteroids were tapered over four weeks after surgery. At the 1-month follow up, the visual field defects had resolved (Figures 4A and 4B) and the visual acuity was 20/15 on the right and 20/20 on the left. Post-operative Hertel's measurements were 19 mm/21 mm, compared with 23 mm/25 mm pre-operatively. The patient has maintained this level of visual function for 18 months. The only post-operative complication was diplopia due to an esotropia that was corrected with strabismus surgery seven months after the decompression surgery.

DISCUSSION

The goal of surgical orbital decompression for thyroid-related compressive optic neuropathy is to achieve decompression of the optic nerve at the orbital apex, overcoming the hallmark orbital apical crowding of this disease. The Walsh-Ogura transantral orbital decompression (Walsh and Ogura, 1957) was long held as the "gold standard" for orbital decompression. Anderson et al. (1981) and McCord (1981) described a transorbital approach to decompression, allowing direct removal of the bones of the medial and inferior orbital walls. This procedure could be accomplished through either a subciliary eyelid incision or a transconjunctival incision. Kennedy et al. (1991) described endoscopic decompression, obtaining comparable Hertel's measurements to the Walsh-Ogura series. They described a series of eight patients and 15 orbits treated with endoscopic decompression, either alone or in conjunction with lateral wall advancement. This technique produced good results with a marked decrease in perioperative morbidity over the transantral route. Kennedy et al. (1991) cited restrictions of the endoscopic technique as limited decompression of the orbital floor in two areas, anteriorly and lateral to the infraorbital nerve. Furthermore, this operation is difficult or impossible to perform with present instrumentation when the orbital floor is made up of hard, thick bone.

The operation we perform utilizes the endoscopic approach for the medial wall and an anterior orbitotomy approach to the orbital floor. This combined approach offers the advantages of endoscopic decompression of the medial wall as well as direct orbital floor decompression. The most anterior orbital floor is maintained. While Kennedy et al. (1991) describe maintenance of the anterior floor as a limitation of the endoscopic technique, we feel that keeping the anterior-most floor is an advantage when good apical decompression has been obtained. The anterior floor provides orbital support, without limiting apical decompression.

Our technique for medial wall decompression is similar to other descriptions of this procedure. It is important to retain bony support in the frontal recess. Keeping this bone in no way affects apical decompression and, more particularly, reduces the likelihood of post-operative frontal sinus obstruction from prolapsing fat. The retention of the middle turbinate, again, does not affect decompression. Its retention makes sphenoid obstruction less likely post-operatively and obviates the small risk of CSF leak associated with its sacrifice. In the creation of the "mega-antrostomy," preservation of the anterior lip of natural ostium prevents circumferential scarring and reduces the likelihood of trauma to the nasolacrimal duct.

The complications we have seen in both of our patients were hypaesthesia in the distribution of the infraorbital nerve and postoperative diplopia. The infraorbital nerve hypaesthesia was transient and both patients have been followed with complete return of sensation. In a series of 305 patients undergoing transantral decompression, Warren et al. (1985) noted 20% transient and 5% permanent post-operative infraorbital nerve hypaesthesia. Conceivably, the anterior orbitotomy approach to the floor, with its capacity for bone removal medial and lateral to the nerve, could make the likelihood of a neuropraxic infraorbital nerve injury higher than in an endoscopic approach alone. In any event, this complication was temporary. No globe ptosis has been noted in the follow-up period of more than 18 months. Of more consequence was the development of post-operative diplopia in both our patients. Both patients underwent subsequent successful adjustable extra-ocular muscle surgery. Rates of diplopia following transantral orbital decompression as high as 79% have been cited (DeSanto, 1980). We hope that further refinement of the technique of retaining an anterior bony buttress at the junction of the floor and medial wall will limit post-operative rotation and diplopia.

CONCLUSIONS

Combined endoscopic and subciliary orbital decompression is a safe and dramatically effective technique for the treatment of compressive optic neuropathy. This procedure combines the advantages of transorbital decompression of the orbital floor with the improved visualization of the medial apex offered endoscopically. Post-operative diplopia may be reduced by refinement of the retained anterior strut of bone at the junction of the floor and medial wall of the orbit.

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