The Management of DIPLOPIA Secondary to THYROID OPHTHALMOPATHY With Vision Therapy and Prism Therapy: A Case Report

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Abstract

RN, a 38-year-old African American male, presented to the optometry clinic at Northport Veterans' Administration Medical Center with complaints of constant double vision. A diagnosis of thyroid ophthalmopathy was made and the patient underwent both medical and radiation therapy in the subsequent months. During the same time period of thyroid treatments, he was prescribed office-based optometric vision therapy to help eliminate the diplopia. In addition, he was prescribed compensating prisms, a regimen of home therapy consisting of techniques to help improve duction, version, vergence, saccadic abilities and binocular fusion. After 18 months of vision therapy the patient was able to sustain fusion comfortably in all positions of gaze with minimal prismatic assistance.

Key Words

vision therapy, thyroid eye disease, Grave's Ophthalmopathy, diplopia, prism

Thyroid eye disease (Grave's Ophthalmopathy) may present with a variety of ocular and systemic signs and symptoms. The ocular signs are often the reason the patient seeks care. Therefore, it is prudent that eyecare practitioners be familiar with the presenting signs of thyroid eye disease, and its appropriate diagnosis, treatment and management.

In many cases, the chief complaint may be prominent eyes, eyelid swelling, double vision, foreign body sensation, pain, photophobia, or decreased vision in one or both eyes. Critical ocular signs include retraction of the eyelids (Dalrymple's sign), eyelid lag on downward gaze (VonGraefe's sign), and often, unilateral or bilateral proptosis. When extraocular muscles are involved, elevation and abduction are commonly restricted. The forced duction test will demonstrate resistance when the eye is pushed or pulled into the affected positions of gaze. Orbital CT scan will often show thickening of the involved extraocular muscles with sparing of the tendon.[1]

Optic nerve compression secondary to thickened extraocular muscles at the orbital apex can produce an afferent pupillary defect, reduced color vision, and visual field and visual acuity loss. The optic disc may also be swollen. It is important to be aware that optic nerve compression can develop in the presence of even minimal exophthalmos, and that involvement of more than one muscle, with restriction of both elevation and horizontal eye movements, may be an indication that the patient is at risk for this complication.[1]

Other ocular signs include reduced frequency of blinking (stare), injection of the blood vessels over the insertion sites of involved extraocular muscles, resistance to retropulsion, eyelid edema, superficial punctate keratitis, or ulceration from exposure keratopathy.[1]

Systemic signs of hyperthyroidism may include rapid pulse, hot and dry skin, heat intolerance, diffusely enlarged thyroid gland (goiter), weight loss, muscle wasting with proximal muscle weakness, hand tremor, pretibial dermopathy or myxedema, and sometimes cardiac arrhythmias. Some patients with Grave's disease are euthyroid (i.e., thyroid function tests are normal.) Myasthenia Gravis with fluctuating double vision and ptosis is often present.[1]

Diagnosis

The recommended optometric workup of a patient with known or suspected thyroid eye disease should include a thorough case history and a comprehensive examination in order to establish a diagnosis, and especially to rule out exposure keratopathy and optic nerve compression. A visual field examination is necessary when signs or symptoms of optic nerve compression are present. Functional assessment should include an evaluation of the range of motion of the oculomotor system as well as the manifest deviation, which, if present, can be assessed in several ways: motor fields, monocular field of view with a perimeter, Parks Three Step, and Hess Lancaster Tests. The binocular deviation in different fields of gaze may be measured with a prism bar and cover

test neutralization. Another method to use is a hand-held Maddox Rod/Risley Prism. It allows free-space measurement of the angle in all nine positions of gaze and at any distance.[2] If proptosis is present the amount can be measured with an exophthalmometer. CT scan of the orbit (axial and coronal views), and thyroid function tests (T3,T4, TSH) are performed as needed to establish the diagnosis.[1]

Management

The patient must be referred to a medical internist or endocrinologist for management of the systemic thyroid disease. Exposure keratopathy is best managed with artificial tears and lubricating ointment, or by taping the eyelids closed at night. Orbital disease may need to be treated more aggressively when the exposure keratopathy is worsening despite treatment (or is already severe), or optic nerve compression is developing.[1]

Optic nerve compression is the most urgent ocular complication of thyroid eye disease, and requires immediate attention. Patients with advanced exposure keratopathy and severe proptosis also require prompt attention. Patients with minimal exposure problems and mild to moderate proptosis are reevaluated every 3 to 6 months. Patients who develop fluctuation of diplopia and/or ptosis should be considered for an edrophonium chloride (Tensilon) test to rule out Myasthenia Gravis.[1]

Any functional problems affecting the patients' performance or binocular functioning should be addressed with prisms, lenses, optometric vision therapy or appropriate combinations. The following case report demonstrates this treatment regimen.

Case Report: History

RN, a 38-year-old African American male, was diagnosed with Grave's Disease by the Northport Veterans' Affairs Medical Center (VAMC) Endocrinology Clinic in November, 1991. In September, 1992, he was examined in the Ophthalmology Clinic with a chief complaint of a slow progressive visual decline in the left eye over the past month, and a secondary complaint of double vision. His best corrected distance visual acuity at this visit was OD 20/25, OS 20/400. He had decreased color vision in both eyes. His left eye showed a positive afferent pupillary defect, a central scotoma and optic neuropathy. The left eye was restricted on upgaze and abduction. His exophthalmometry readings were OD 25 and OS 27 (see Figure 1). Goldman tonometry readings were OD 23mm Hg, and OS 22mm Hg at 9:00AM. He was diagnosed with compressive Grave's ophthalmopathy OS, and was started on oral Prednisone, 100mg QID, with a recommendation for radiation treatment.

Figure 1. Bilateral proptosis

One week later vision had improved to OD 20/20, OS 20/50. All pupillary reflexes were normal and there was no evidence of an afferent defect. Exophthalmometry readings were OD 25 and OS 24. Motilities were unchanged from the previous visit. Goldman tonometry readings were 26mm Hg in each eye at 10:00AM. Prednisone was continued, and Betagan 0.5% BID OU was added to control the intraocular pressures. (The Betagan was a temporary medication until RN was tapered off the Prednisone.) A few weeks later RN began radiation treatment at 2000 rads. By November, 1992, the radiation treatments had ended. Goldman tonometry results were OD 18mm Hg and OS 20mm Hg at 11:00AM (without Betagan) and exophthalmometry readings were OD 23 and OS 21. Vision had returned to 20/20 in each eye, but RN still had constant double vision and was referred to the Optometry Clinic for prism evaluation.

RN first presented to the Optometry Clinic during December, 1992, with complaints of constant double vision in primary and left gaze. He reported that his left eye was restricted in movement and that objects in space had a tilted appearance. Other functional visual problems included headaches and asthenopia associated with near visual tasks unless he held reading material at approximately 10 inches. He had ceased driving because of the double vision and was unemployed as a result of his inability to drive to work. His medical records document concern and depression about his eye condition. When wearing the patch prescribed by his previous eye doctor, he expressed lack of confidence in his mobility. He reported that he had fallen in the street and had trouble with balance.

RN's short term goals were to have single vision in primary and left gaze, and to eliminate any restrictions of gaze. He wanted to watch TV, read, drive and walk with confidence. Ultimately he wanted to go to college.

Our short term goals were to reduce the frequency of diplopia as well as improve stability of fusion and increase fusional ranges so that RN could function more efficiently and fulfill his aspirations.

Summary of Optometric Diagnostic Testing

RN presented with constant diplopia secondary to Grave's ophthalmopathy. A noncomitant, constant, left esotropia was documented, with diplopia increasing in superior right and superior left gaze. The angle of turn, assessed with cover testing and prism neutralization in primary gaze, was 25-30 prism diopters at distance and 20-25 prism diopters at near. His centration point was 6"-7".

Red lens testing in the 9 cardinal positions of gaze revealed a noncomitant constant left esotropia, neutralized with the following lenses: 12 prism diopters base out (BO) in superior right gaze; 8 prism diopters BO in superior left gaze; 6 prism diopters BO in inferior right and right gaze; orthophoria in inferior, inferior left, and left gaze; 5 prism diopters BO in superior gaze; 8 prism diopters BO in primary gaze. Motor field testing revealed

limited supra-abduction, supra-adduction and elevation of the left eye. Since there was no hyper deviation in primary gaze, a Park's Three Step test was not performed. It was concluded, based on the medical diagnosis and CT scan that several of the extraocular muscles of the left eye were fibrotic and swollen and were limiting the range of motion.

A good prognosis for reducing the frequency of diplopia utilizing prism and vision therapy was presented to the patient. Projected length of treatment was estimated at 50 to 60 visits.

Overview of Vision Therapy Management

A total of 25 diopters base-out Fresnel prism was prescribed, split between the two eyes, in conjunction with an out of office vision therapy program. In order to develop an awareness of the feeling of his eyes in various positions of gaze, and to prevent or decrease any secondary contractures, RN was prescribed monocular four corner saccades and thumb rotations as ocular calisthetics.

He was next seen in the Optometry Clinic during April, 1993. At this point, he was wearing the prisms for all distance and most near activities and he reported that diplopia was eliminated. His home therapy regimen was increased to include pointer-straw sequences with an emphasis on expanding divergence ability. Additionally, the Brock String[3] sequence was introduced emphasizing peripheral awareness and the feeling tone of his eyes when diverging. RN was also prescribed index card saccadics for out-of-office therapy. He was instructed to write letters or numbers on the index cards and tape them to the wall in a variety of patterns, including horizontal, vertical, diagonal or random patterns. Kinesthetic support and visual feedback was provided by having RN aim a flashlight at each target.[4]

As home vision therapy progressed, we began seeing RN more frequently for in-office vision therapy. At this time motorized rotator pursuits with a flashlight for proprioceptive and multisystem feedback was added. The following month, computerized therapy sequences were introduced to develop and expand both divergent and convergent flat fusional ranges. Large targets were used initially, with an emphasis on divergence and peripheral awareness. Split procedures using

Figure 2. RN performing multiple Brock String therapy

a vertical prism for dissociation were presented in order to develop alignment with sensory motor feedback. Accommodative facility was enhanced using standard accommodative rock techniques.

RN was also being followed in the Ophthalmology Clinic and at this point was offered strabismus surgery. He refused the surgery, since his double vision was eliminated, and expressed a desire to continue vision therapy in an attempt to reduce the prism to an amount that was more cosmetically acceptable.

In early June, 1993, due to RN's dramatic improvement in fusional ranges, the correction was reduced to 15 diopters base-out Fresnel prisms, split between the two eyes. (His objective angle measured 20 diopters at distance and 16 diopters at near.) This reduction in the prism correction was too great of a change for RN to maintain single vision consistently and 2 weeks later we found it necessary to increase the amount of prism back to 25 diopters base-out split between the two eyes.

Weekly vision therapy was continued over the next few months with the addition of fusion therapy with vectograms, both at near and projected at distance. Computer-generated therapy continued and additional home therapy techniques using two paper towel tubes with a vertical band in one, and a horizontal band in the other, were added. RN's goal was to fuse the two into a "+" while diverging the tubes.

In September, 1993, RN reported near vision blur. A refraction revealed early presbyopia. Consequently, a RX was ordered with a +0.50 diopter sphere OU add, and 6 prism diopters base-out in front of each eye for near. Stereopsis measured with Titmus Stereofly Tester[a] was forty seconds of arc. The following month we were able to decrease the prism in his distance glasses to 20 diopters. RN reported no diplopia with these glasses.

The final phase of vision therapy included the Aperture Rule,[b] Wayne Saccadic Fixator,[c] multiple Brock Strings (see Figure 2) and Electronic Brock String[c] with jump ductions to a distant target. An anaglyph trainer was utilized for out-of-office therapy to further increase divergence ranges and improve fusion stability. RN was encouraged to maintain awareness when his eyes were aligned and to be cognizant of kinesthetic-proprioceptive feedback when his eyes diverged during home and office therapy.

The prism correction was again decreased in June, 1994, to 18 diopters. RN was still able to maintain single vision and proved quite diligent with his home therapy. A mirror cheiroscope[b] and a Brewster stereoscope[b] were added to his out-of-office therapy. In-office therapy continued, mainly using a computer-based program to develop and expand vergence ranges, both BI and BO, starting with stereo targets and working toward flat fusion targets.

RN's in-office therapy was reduced to once a month and he presently continues to do the home therapy techniques. The most recent evaluation (January 1995) documented a further reduction of prism at distance to 12 diopters BO, split and ground into his prescription.

Conclusion

This case is another example which illustrates how the combination of medical management (the treatment of the disease entity) and optometric management (the treatment of functional problems) when appropriate, can help patients with binocular vision problems secondary to a systemic condition.[5] This case presentation also demonstrates the effectiveness of optometric vision therapy in combination with prism for the treatment of noncomitant strabismus. A combination of prism, out-of-office and in-office therapy was used to develop fusional ranges and increase stability of fusion without the need for surgery. The quality of RN's life has

dramatically improved. Currently, he is a full-time college student pursuing a degree in psychology. He reports that he no longer has difficulty walking or driving. It was recommended that RN continue to do the home therapy. He will be periodically evaluated to assure that fusional ranges are maintained and that he is still functioning well.

Optometrists can perform a unique role in the management of binocular vision problems secondary to systemic disease. Excellent results were obtained for RN through appropriate use of prism combined with vision therapy.

Sources

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b. Bernell Corporation, 750 Lincolnway East, PO 4637, South Bend, IN, 46634-4637
c. Wayne Engineering, 1825 Willow Rd., Northfield, IL, 60093

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