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Clinical challenges

Of grave concern

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1. Case report

A 73-year-old woman presented with 6 weeks of bilateral visual loss on a background of Graves orbitopathy, considered to be biochemically “burnt-out” 10 years before. The Graves disease had been treated with radioactive iodine, and she was on thyroxine replacement.

Her medical history is significant for brittle diabetes mellitus with episodes of asymptomatic hypoglycemia. In 2011 she had an episode of diabetic ketoacidosis. At presentation she was taking insulin, and her HbA1c was 9.3%. Other medical history was significant for a lumbar laminectomy in 2005 and a melanoma excised from her foot in 2011 with clear surgical margins.

Bilateral fat-only orbital decompression had been performed 4 months earlier for proptosis reduction and cosmesis. Preoperative visual acuity was documented as 20/20 OU before her decompression. She made an uneventful postoperative recovery.

Three months after the decompression she noted visual difficulty and recurrent proptosis. Her acuities were 20/80 OD, 20/60 OS, and she was unable to read beyond the Ishihara control plate. There was a right relative afferent pupillary defect. She had bilateral visual field loss on automated perimetry (Fig. 1). Clinically, she had severe right and moderate left proptosis with lid retraction. The conjunctiva was mildly chemotic (Fig. 2). The clinical activity score for thyroid orbitopathy was low. Her intraocular pressures were borderline-elevated, but increased with upgaze. There was no evidence of diabetic retinopathy or disk abnormalities.

Her erythrocyte sedimentation rate and C-reactive protein were normal for her age. Computed tomography imaging of the brain and orbits showed modest enlargement of extraocular muscles. Though the computed tomography did not suggest apical crowding, it was thought that the right optic

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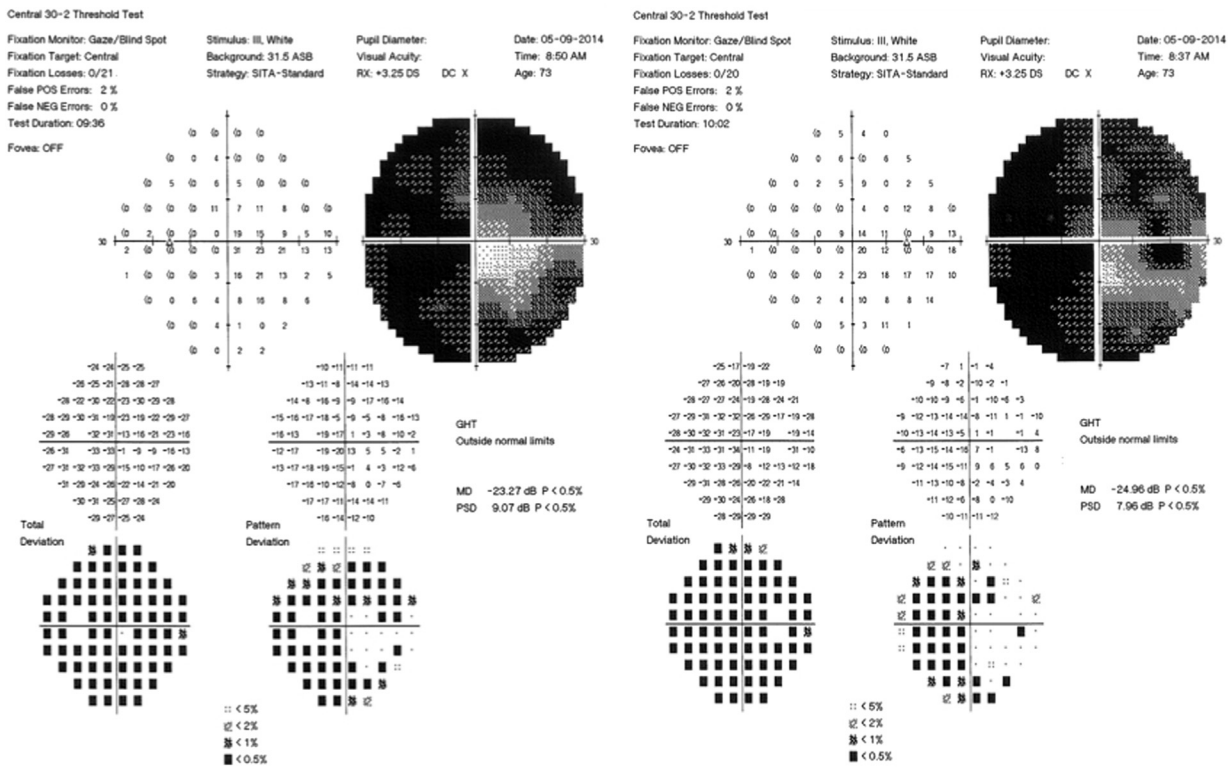


Fig. 1 – The 30-2 automated Humphrey visual fields, 4 months after fat-only orbital decompression, with severe bilateral field loss worse for the right eye. The right nasal field and the left temporal field show the greatest loss of sensitivity, therefore, there is a suggestion of a left homonymous hemianopia.

nerve appeared “straightened,” with bilateral fat streaking at the orbital apices. A magnetic resonance image (MRI) of the brain showed similar orbital changes (Fig. 3) and was interpreted as showing only cerebral microvascular disease.

How would you proceed?

2. Comments

2.1. Comments by Mark Moster, MD

The “straightforward” answer is to treat for thyroid ophthalmopathy with bilateral compressive optic neuropathy.

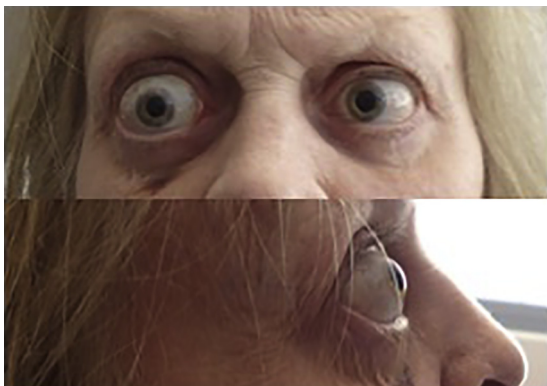


Fig. 2 – Bilateral proptosis, lid retraction, and mild conjunctival chemosis.

The diagnosis is based on decreased visual acuity, dyschromatopsia, right relative afferent pupillary defect, and visual field defects. If I were to treat for thyroid ophthalmopathy with optic nerve compression, I would likely start with systemic corticosteroids, with careful control of the diabetes. Radiation therapy would likely be avoided because the brittle diabetes may predispose to more severe complications of radiation. If the steroid response was suboptimal, then surgical orbital decompression would be the next step.

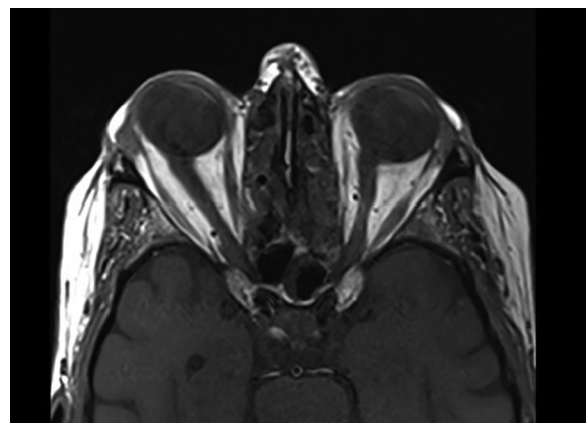


Fig. 3 – T1 axial orbital views show straightening of the right optic nerve but not the left. There is no enlargement of the extraocular muscles.

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