add to already burdened clinics would be prohibitive. At present no test is available that is able to predict either those patients who are at risk of developing amiodarone-induced optic neuropathy or those patients who develop the pre-symptomatic state on the drug. Therefore, until a reliable screening test is developed, patients should be advised to visit their doctors should any visual symptoms occur, and ophthalmologists seeing patients with swollen optic discs who are using amiodarone should consider the possibility of amiodarone-induced optic neuropathy.

References

1. British National Formulary, no. 33. London: British Medical Association and the Royal Pharmaceutical Society of Great Britain, 1997.
2. Gittinger JW, Asdourian GK. Papillopathy caused by amiodarone. Arch Ophthalmol 1987;105:349–51.
3. Chew E, Ghosh M, McCulloch C. Amiodarone-induced cornea verticillata. Can J Ophthalmol 1982;17:96–9.
4. Nazarian SM, Jay WM. Bilateral optic neuropathy associated with amiodarone therapy. Clin Neuro-ophthalmol 1988;8:25–8.
5. Mansour AM, Pulkin JE, O'Grady R. Optic nerve ultrastructure following amiodarone therapy. J Clin Neuro-ophthalmol 1988;8:231–7.
6. Duff GR, Fraser AG. Impairment of colour vision associated with amiodarone keratopathy. Acta Ophthalmol (Copenh) 1987;65:48–52.
7. Sedwick LA. Getting to the heart of visual loss: when cardiac medicine may be dangerous to the optic nerves. Surv Ophthalmol 1992;36:366–72.
8. Feiner LA, Younge BR, Kazmier FJ, Stricker BH, Fraunfelder FT. Optic neuropathy and amiodarone therapy. Mayo Clin Proc 1987;62:702–17.
9. Kreig P, Schipper I. Bilateral Optikusneuropathie nach Amiodarone-therapie. (Bilateral optic neuropathy following amiodarone therapy.) Klin Monatsbl Augenheilkd 1992;200:128–32.
10. Gittinger JW, Asdourian GK. Amiodarone-related optic neuropathy [letter]. Mayo Clin Proc 1988;62:210.
11. Younge BR. Amiodarone optic neuropathy. J Clin Neuro-ophthalmol 1988;8:29.

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Sir,

Transient retinal arterial compromise in Graves' orbitopathy

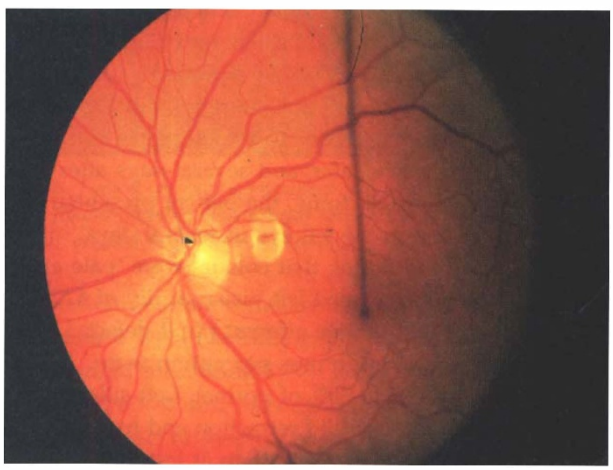
Graves' orbitopathy is a well-recognised autoimmune process, whose pathogenesis is still being elucidated.[1] Severe visual loss may occur because of compressive optic neuropathy or corneal exposure, and diplopia, choroidal folds and transient hypermetropia are also well recognised.[2]

We describe a single patient with Graves' orbitopathy, in whom elevation of intraocular pressure in upgaze occurred to such a marked level that the retinal arterial circulation was temporarily compromised.

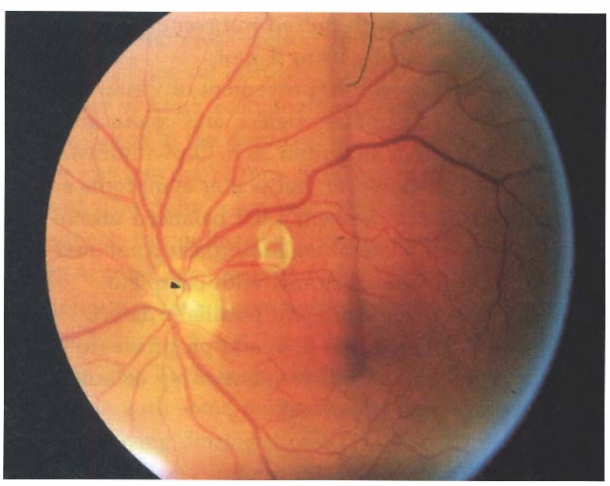
Case report

A 70-year-old Caucasian woman was referred from her endocrinologist for management of her left orbital discomfort associated with thyroid orbitopathy 5 months after she had been diagnosed with hyperthyroidism.

Initial examination revealed corrected visual acuity of 6/5 in each eye, with normal pupils. There was obvious chemosis and proptosis of the left eye, measured at 20 mm on the left side compared with 16 mm on the right side using the Luedde system.[3] A left hypotropia was present in the primary position, with defective elevation on adduction and abduction, and 2 mm of left upper eyelid retraction relieved on downgaze.[4] Intraocular pressures were 20 mmHg bilaterally in the primary position, but were measured at 36 mmHg in the right eye and 54 mmHg in the left eye on attempted upgaze, at which point the left retinal arterial system was noted to



(a)



(b)

Fig. 1. *(a) Left fundus showing normal retinal vessels in primary gaze. (b) Same left fundus showing collapsed arterial vessels (arrowhead) at the disc during systole when the patient was attempting upgaze.*

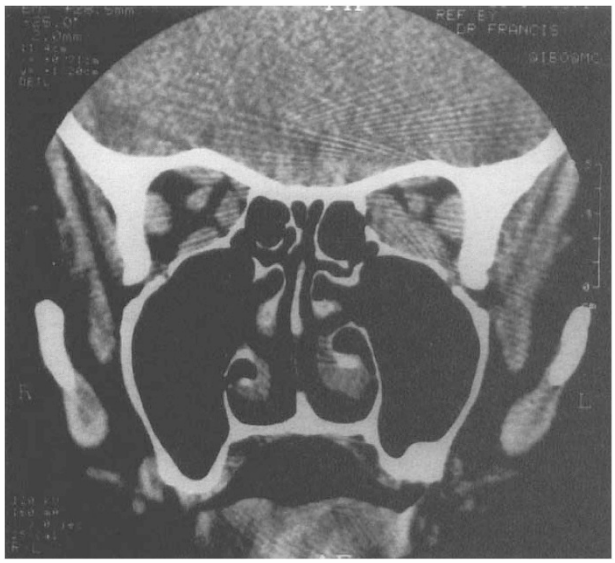


Fig. 2. *Coronal CT scan showing crowded muscles at the left orbital apex.*

pulsate at the optic nerve head (Fig. 1), and the patient experienced extreme discomfort and 'blurring' of vision in that eye. A coronal CT scan of the orbits revealed gross enlargement of the left inferior, lateral and medial rectus muscles with crowding at the orbital apex (Fig. 2).

The patient's subsequent course included symptomatic treatment with oral diuretics and topical lubricants, and she was advised to avoid upgaze as much as possible. Eight weeks later, and several days after an uneventful total thyroidectomy, the patient presented with reduced vision in the left eye of several days' duration, specifically noting that colours were 'pale and faded'. Examination revealed left visual acuity of 6/24 with a grade 2/4 left relative afferent pupil defect. Left colour saturation and brightness perception were subjectively decreased to 30% of normal. Left-sided chemosis, proptosis, upper lid retraction and hypotropia were noted to be slightly greater than at initial presentation.

The diagnosis of compressive optic neuropathy was made, and treatment with pulsed intravenous steroids was commenced, initially using 1 g of methylprednisolone, with improvement of visual acuity, the relative afferent pupillary defect, and colour and brightness perception, all within 36 h. On withdrawal of the corticosteroids, however, her ocular symptoms and signs recurred. She underwent a left balanced orbital decompression via a lateral wall approach combined with an external ethmoidectomy, with complete resolution of her relative afferent pupillary defect, recovery of visual acuity (to 6/4) and proptosis (to 15 mm), and subjective improvement of her colour and brightness perception to normal.

Discussion

Patients with thyroid orbitopathy may have elevated intraocular pressure on attempted upgaze related to the pressure of an inelastic inferior rectus muscle complex against the eye,[5] and the increased external pressure on the globe due to maximal contraction of the superior rectus. The finding of increased intraocular pressure on upgaze has been reported in 22–76% of patients with Graves' orbitopathy, but limited to a rise in intraocular pressure of only 8 mmHg from the primary position.[6,7]

It was noteworthy that of the four cases reported in 1953 by Braley,[5] and whose pressures were measured with the Schiötz tonometer, case 1 had a pressure of 80 mmHg in the primary position in one eye and 60 mmHg in the other eye, reducing by 20 mmHg in each eye on downgaze. However, the accuracy in using the Schiötz tonometer to measure intraocular pressure in upgaze is questionable in supine patients with tense orbits.

The finding of variable intraocular pressure with different positions of gaze may offer supporting evidence for the early diagnosis of the thyroid orbitopathy, and avoid the erroneous diagnosis of glaucoma in these patients. An increase in intraocular pressure on upgaze is said to correlate positively with the severity of proptosis[7] and the degree of muscle involvement,[8] thus paralleling the level of disease activity.

Other restrictive eye movement syndromes such as orbital pseudotumour, lymphoma and orbital floor fracture with inferior rectus entrapment have been shown to increase intraocular pressure on upgaze. However, in contrast to these patients with unilateral pathology, patients with Graves' orbitopathy tend to demonstrate the intraocular pressure differential bilaterally,[9] as did our case. Further, marked intraocular pressure rises can occur with strabismus surgery as the surgical assistant exerts traction on the tight inferior rectus muscle, or may occur in orbital surgery such as in optic nerve sheath decompression with strenuous abduction of the eye by the surgical assistant. This passive (external) pressure elevation can of course produce similar pressure rises to those seen in our thyroid patient purely by the patient simply elevating the eyes, but occur in anaesthetised patients and must not be maintained for extended periods.

We believe this is the first report of a case of increased intraocular pressure involving a pressure rise of greater than 30 mmHg, and to such a level that the retinal arterial circulation was transiently compromised. Although this is unlikely to represent a frequent mechanism for possible loss of vision in Graves' orbitopathy, it clearly documents the potential adverse effect of sustained upgaze in this group of patients. We would counsel our patients to avoid the discomfort and potential threat to their retinal arterial circulation by avoiding prolonged upgaze.

References

1. Bahn RS, Heufelder AE. Pathogenesis of Graves' ophthalmology. N Engl J Med 1993;329:1468–75.
2. Garrity JA, Fatourechi V, Bergstralh EJ, Bartley GB, Beatty CW, de Santo LW, Gorman CA. Results of transantral orbital decompression in 428 patients with severe Graves' ophthalmopathy. Am J Ophthalmol 1993;116:533–47.

3. Chang AA, Bank A, Francis IC, Kappagoda MB. Clinical exophthalmometry: a comparative study of the Luedde and Hertel exophthalmometers. Aust N Z J Ophthalmol 1995;23:315–8.
4. Burde RM, Savino PJ, Trobe JD. Clinical decisions in neuro-ophthalmology. St Louis: CV Mosby, 1985:277.
5. Braley AE. Malignant exophthalmos. Am J Ophthalmol 1953;36:1286–90.
6. Allen C, Stetz D, Roman SH, Podos S, Som P, Davies TF. Prevalence and clinical associations of intraocular pressure changes in Graves' disease. J Clin Endocrinol Metab 1985;61:183–7.
7. Gamblin GT, Harper DG, Galentine P, Buck DR, Chernow B, Eil C. Prevalence of intraocular pressure in Graves' disease: evidence of frequent subclinical ophthalmopathy. N Engl J Med 1983;308:240–4.
8. Fishman DR, Benes SC. Upgaze intraocular pressure changes and strabismus in Graves' ophthalmopathy. J Clin Neuroophthalmol 1991;11:162–5.
9. Spierer A, Eisenstein Z. The role of increased intraocular pressure on upgaze in the assessment of Graves' ophthalmopathy. Ophthalmology 1991;98:1491–4.

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Sir,

Histology of xanthelasma lesion treated by argon laser photocoagulation

Xanthelasma palpebrarum is characterised by yellow lipomatous plaques found on the eyelids, particularly in middle-aged women.[1] Although surgical excision is the most commonly used treatment, it is of limited use in larger lesions, where eyelid closure may then become compromised.[2] Both CO_2 lasers, and more recently argon lasers, have been successfully used to treat xanthelasma.[3–5] We present the histological changes in a case of xanthelasma palpebrarum following argon laser treatment, which to the best of our knowledge have not previously been described.

Case report

A 49-year-old woman presented with a 2-year history of yellow plaque-like lesions on the nasal aspect of her upper eyelids, which troubled her cosmetically. There was no other significant ophthalmic or medical history of note.

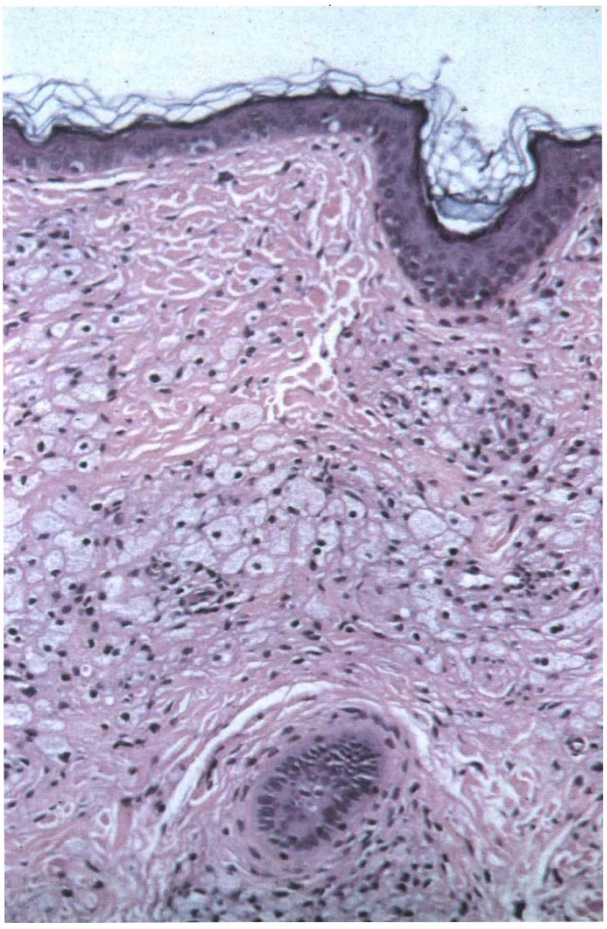

Fig. 1. *Typical pre-laser appearances of xanthelasma showing large, pale, lipid-containing macrophages scattered throughout the dermis. (H&E, original magnification ×200).*

On examination visual acuities were 6/9 in both eyes. The lesions were clinically evident as xanthelasma, that on the right measuring 8 × 5 mm while that on the left measured 6 × 4 mm. No other abnormalities were found on ophthalmic or medical examination. The results of serum haematological and biochemical investigations, including the fasting lipid profile, were within normal limits.

The left lesion was excised under local anaesthetic and showed histological features typical of xanthelasma, with a number of large, pale, foaming, lipid-laden macrophages in the dermis (Fig. 1). The depth of the lesion was less than 1 mm. No chronic inflammation was present. The right lesion was treated under local anaesthesia by confluent photocoagulation with an argon laser (Argon-Green, energy 600 mW, spot size 1 mm, continuous mode). Three weeks later the lesion was biopsied and a 4 × 4 × 2 mm sample sent for histological analysis (Fig. 2). This showed mildly thickened epidermis with some perinuclear cytoplasmic clearing. The underlying dermis showed mild scarring with plump fibroblasts and a mild diffuse chronic inflammatory infiltrate. No lipid-laden macrophages were noted. These striking changes presumably represent laser-induced damage. The 3 week period was chosen to assess the changes to the lipid-laden