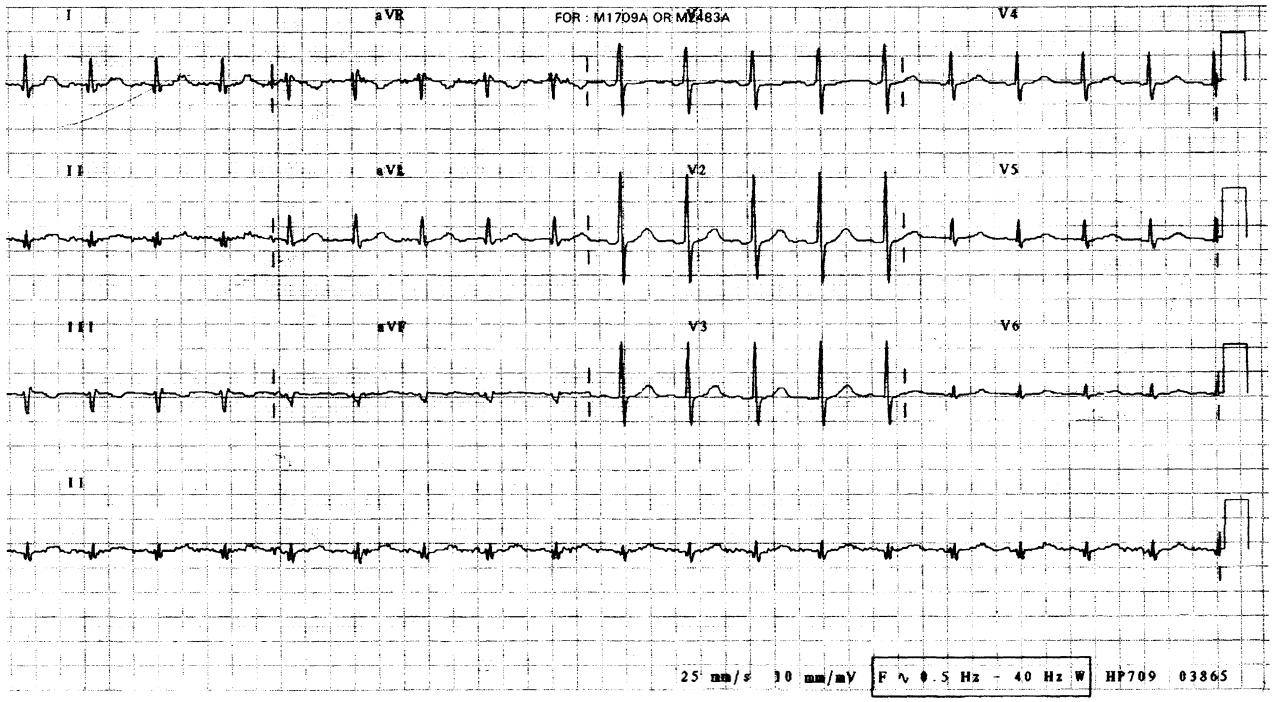

Fig. 3. *ECG showing sinus rhythm following resolution of the arrhythmia.*

Steel and Thorn[1] showed Mydricaine No. 1 to produce tachycardia and hypertension in 20% of patients. A case study of a patient with a known cardiac history developing myocardial ischaemia following subconjunctival Mydricaine No. 2 has been reported.[2] To our knowledge there are no reported cases of any patient developing supraventricular ectopics and supraventricular tachycardia following subconjunctival injection of Mydricaine No. 2.

Due to the fact that Mydricaine No. 2 is produced under special licence it is not listed in the BNF or MIMS and there is no data sheet provided with this drug, which makes it difficult to find information regarding possible side effects resulting from its usage. Steel and Thorn[1] suggested that observations be performed during and following administration of Mydricaine, though the form and timing of these observations has not been specified. We suggest that administration guidelines for this commonly used formulation should be drawn up. Being an unlicensed product the responsibility following administration lies with the prescriber,[4] who unfortunately at present does not have access to possible complications that may result from its usage.

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Sir,

Leber's hereditary optic neuropathy following trauma

Leber's hereditary optic neuropathy (LHON) usually presents as subacute, bilateral, sequential optic neuropathy and is associated with mutations in mitochondrial DNA.[1] Environmental factors including alcohol and tobacco may play a role in precipitating neuropathy.

We report a case of LHON following relatively mild trauma and discuss the implications for our understanding and diagnosis of this unusual condition.

Case report

An 18-year-old man was seen within a few hours of an assault during which he had been punched in the face several times. He had not lost consciousness and there was no radiological evidence of any fracture.

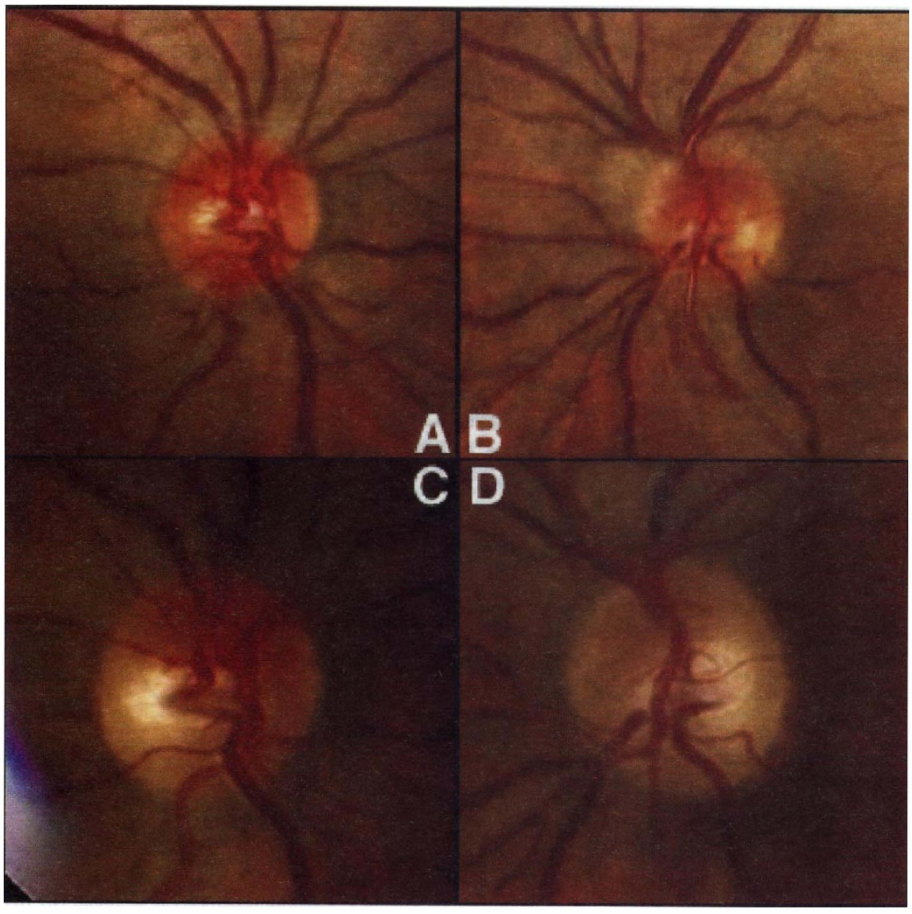

Fig. 1. *Optic discs. (A) Right eye, 1 week following injury. (B) Left eye, 1 week following injury, showing disc swelling and hyperaemia. (C) Right eye, 3 months following injury, showing temporal optic atrophy. (D) Left eye, 3 months following injury, showing marked optic atrophy.*

Unaided acuity was 6/9 right, 6/12 left. He had bilateral periorbital bruising, bilateral mild subconjunctival haemorrhages and mild commotio in the left supero-temporal retina. There was no afferent pupillary defect and no proptosis. Anterior segments, intraocular pressures and external ocular movements were all normal.

Six days later, left visual acuity had gradually deteriorated to 2/60 with no subjective red desaturation or loss of brightness. There was a left afferent pupillary defect and Ishihara colour vision testing was markedly reduced on the left. Both optic discs cups were small and on the left there was mild disc swelling, a small peripapillary nerve fibre layer haemorrhage (Fig. 1B) and a centrocaecal scotoma (Fig. 2A). Magnetic resonance imaging of the head and orbits was normal with no signs of orbital trauma and electrodiagnostic tests showed severe left optic nerve dysfunction. A diagnosis of traumatic optic neuropathy was made.

Two months later, he presented with gradual loss of vision in the right eye over 1 week. Acuity was 3/36 right, HM left. The right optic disc was now hyperaemic and the left was atrophic with temporal pallor. He reported moderate tobacco and alcohol intake. There was no history of eye disease among his extended family which included several matrilineal uncles and great-uncles. Folate and B_{12} levels were normal. Electrodiagnostic tests now showed bilateral optic neuropathy. Perimetry showed bilateral centrocaecal scotomas (Fig. 2C, D) and over the next 2 weeks temporal pallor ensued in the right optic disc (Fig. 1C).

Mitochondrial DNA testing showed a G to A substitution at position 11778 (with no abnormality at 14484 or 3460) confirming a diagnosis of LHON.

Comment

The 11778 G to A mutation is the commonest of the three 'primary' mutations which together have been found in 97% of European and Australasian families with LHON.[2,3] Sporadic cases are not uncommon and, even within established pedigrees, penetrance in males is around 50%. The effect of this mutation is to change a conserved arginine to histidine at amino acid position 340 of ND4. This protein (in common with those affected by the other two 'primary' mutations, 3460 and 14484) is a subunit of mitochondrial complex I, NADH CoQ1 reductase.

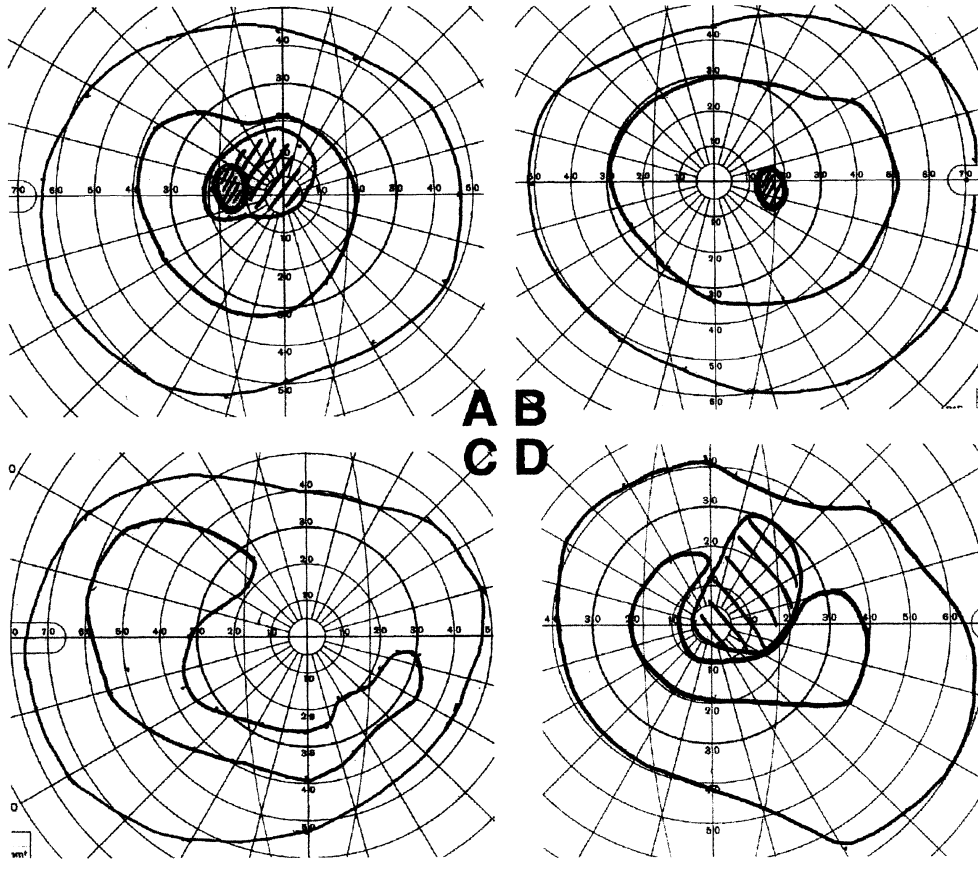

Fig. 2. *Goldmann visual fields. (A) Left eye, 1 week following injury, showing centrocaecal scotoma. (B) Right eye, 1 week following injury. (C) Left eye, 3 months following injury, showing severe centrocaecal scotoma. (D) Right eye, 3 months following injury, showing centrocaecal scotoma.*

It has been proposed that mitochondrial respiratory chain dysfunction may reduce ATP production, reducing axoplasmic transport, resulting in axon swelling.[4] This may rapidly progress as swelling compromises the local microcirculation and impaired transport of mitochondria further reduces ATP production. The lamina cribrosa may form an anatomical 'chokepoint' making ganglion cells particularly vulnerable in this region.[5] In addition to reducing ATP production, mitochondrial dysfunction may also induce ganglion cell apoptosis, possibly via formation of free radicals.[6]

Environmental factors may influence the course of LHON. A case–control study found a significant increase in tobacco and alcohol consumption at age of onset in patients with some types of LHON.[7] Anecdotal reports suggest several other possible precipitants including diabetes, vitamin B_{12} deficiency, carbon monoxide, ethambutol and other toxins.[1]

Optic neuropathy in LHON often markedly reduces colour vision whilst pupil reactions may be normal. In this case colour vision was subjectively normal and an afferent pupillary defect was apparent. Trauma may directly cause optic neuropathy by several mechanisms including compression, swelling and ischaemia. In this case, however, the level of injury would not have been expected to cause the acute neuropathy which was evident 1 week later. As in other cases where LHON appears to have been related to environmental factors, it appears that a mild acute insult may have been enough to precipitate neuropathy in optic nerves already compromised by mitochondrial dysfunction.

Mitochondrial DNA testing has shown that the clinical features of LHON are more varied than previously recognised. Whilst established LHON is not yet treatable, accurate diagnosis has significant practical implications. Asymptomatic carriers could be advised to avoid possible precipitating factors such as alcohol and tobacco. Genetic counselling can be useful for patients and their families. Mitochondrial DNA testing for LHON should be considered in patients with optic neuropathy following trauma.

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Sir,

Episcleral osseous choristoma

Episcleral osseous choristoma is a rare benign lesion of episcleral connective tissue that characteristically occurs in otherwise normal eyes. It was first described by Von Graefe in 1863.[1]

Case report

An 8-year-old Caucasian girl presented with a 6 month history of a slowly enlarging painless lump on the upper outer corner of her left eye. Examination revealed a pea-sized, whitish, firm subconjunctival lesion in the superior temporal quadrant of the left eye (Fig. 1). The remainder of the ocular examination showed no abnormalities. A diagnosis of epibulbar dermoid was made. The tissue was excised under general anaesthesia.

Intraoperatively, adequate exposure of the lesion was obtained by inserting a 7/0 corneal fixation Ethilon suture at the limbus, adjacent to the lesion, and clipping the ends of the suture, on to the drapes, in the direction of the opposite quadrant. An incision was made into the conjunctiva overlying the lesion. The conjunctiva was

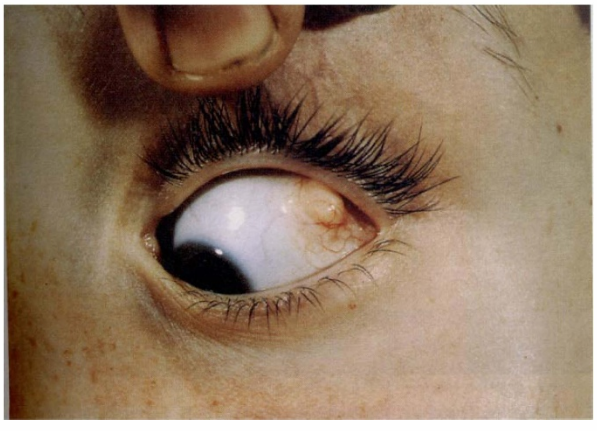

Fig. 1. *Slit-lamp photograph of the subconjunctival lesion in the supero-temporal quadrant of the left eye.*

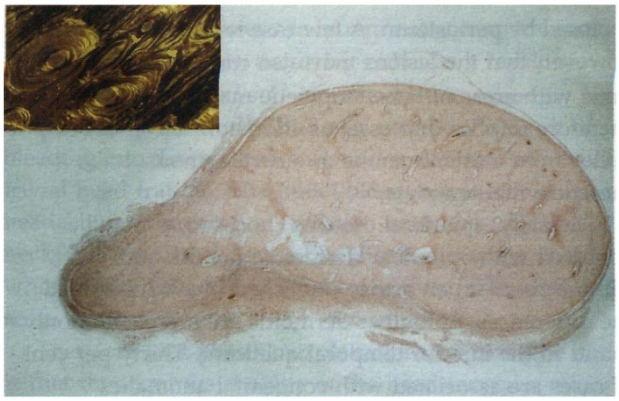

Fig. 2. *Histological appearance of the excised lesion, showing dense compact lamellar bone, arranged in Haversian systems and enclosed by periosteum. This picture is characteristic of most episcleral osseous choristomas (H&E; ×4.5 original magnification). Inset: Polarised filters (×25 original magnification).*

dissected off the lesion using a pair of Wescott's scissors. Once adequate exposure was obtained, the lesion was carefully excised using a No. 15 Bard-Parker blade. Haemostasis was achieved with the aid of bipolar diathermy. The conjunctiva was repaired with interrupted 7/0 vicryl sutures. The corneal fixation suture was removed and chloramphenicol ointment was applied topically. Thereafter, a protective Cartella shield was placed over the eye.

The specimen, a bony-hard ovoid nodule measuring 0.7 cm, was placed in formalin solution and sent to the pathology department for histological assessment. This showed an osteoma composed of compact lamellar bone, namely a choristoma (Fig. 2).

Comment

Choristomas are defined as congenital overgrowth of normal tissue elements at sites where they do not normally occur. Single tissue choristomas consist of dermis-like tissue or ectopic tissue of meso-ectodermal origin, e.g. lacrimal and other glands, fat, brain, cartilage, bone and teeth. Complex or composite choristomas contain a combination of tissue from various origins. Osseous choristomas are rare lesions of the conjunctiva, episclera and choroid.

Episcleral osseous choristomas are usually detected in early infancy or childhood. They vary in size from pea- to almond-sized. They tend to increase in size either in early childhood or around puberty.[2] Extraocular lesions commonly occur superotemporally, 5–10 mm from the limbus. There have, however, been a few case reports of occurrences at unusual locations. Oritz and Yannoff[3] reported a case of epipalpebral osseous choristomas found in the conjunctiva of the right lower lid. Melki *et al.*[4] reported on a case involving the superior rectus sheath while Ferry and Hein[5] reported on a lesion attached to the lateral rectus sheath. Macroscopically these lesions are composed of compact bone surrounded by fibrous tissue. The histological appearance is that of dense osseous tissue arranged in Haversian systems, and