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Sir,

A Case of Unique Altitudinal Distribution of a Retinal Contusion (Berlin's Oedema) in a Healthy Teenager

A 16-year-old caucasian male presented complaining of diffuse headaches, lightheadedness and blurred vision in his left eye, 48 hours after suffering a blunt trauma by a fist to his left lateral orbit and temple. In the previous 12 hours before ophthalmological examination the patient had experienced loss of the entire inferior half of the left field of vision, accompanied by scintillations and nausea. Past history was unremarkable except that the patient had been smoking 20 cigarettes per day for the previous 2 years. Best corrected visual acuity was 20/20 in the right eye and 20/50 improved with pinhole to 20/40 in the left eye. There was +2 relative afferent pupillary defect in the left eye. Slit lamp examination of both eyes was within normal limits. Intraocular pressure was 18 mmHg in both eyes. Fundoscopic examination through a dilated pupil was normal in the right eye; in the left eye substantial retinal whitening was noted in the entire superior half of the posterior pole with minimal extension beyond the superior temporal vessels. The retinal pathological area transected the fovea in a perfect horizontal line, creating a 'hemicherry red spot' (Fig. 1). The left optic nerve head was normal in appearance with no signs of oedema, haemorrhages or vasculopathy. No evidence of occlusion or thromboembolism was found. Goldmann's visual field testing confirmed the clinical picture, demonstrating an inferior altitudinal defect in the left eye (Fig. 2). Ishihara tests for colour-blindness were normal in both eyes. Fluorescein angiography failed to show any abnormality. There was no evidence of vascular constriction, occlusion or perivascular leakage. The fluorescein transit time to the superior retina was not slowed in any way compared with the distribution to other areas of the retina. Heart and lung auscultation was normal. No bruits were heard along either carotid artery, and the rest of the physical examination was within normal limits. Computed tomography of the head and orbits did not reveal any pathological findings.

On follow-up examination, 1 month after the trauma, the visual acuity had improved to 20/20–1 in the left eye, but the retinal findings remained unchanged. On the patient's last examination, 3 months after the trauma, no further improvement was noticed in the visual acuity. The retinal area seemed less opaque, but the left visual field demonstrated the same inferior altitudinal defect.

Discussion

Retinal contusion is a countercoup injury which may occur centrally (Berlin's oedema) or peripherally. A few hours after such trauma the affected area of the outer retina becomes white and opaque, due to tissue disorganisation. The swollen layer blocks the background choroidal fluorescence in angiography but with no leakage. Prognosis is usually excellent, except for cases with severe pigment epithelial damage, subfoveal haemorrhage or choroidal rupture. When photoreceptors are destroyed, localised visual field defects are expected, but arcuate field defects are not found due to an intact overlying nerve fibre layer.[1]

Altitudinal hemianopsia has been reported with ischaemic optic neuropathy, optic neuritis, meningiomas, congenital nerve head anomalies, advanced glaucoma, enlargement of the internal carotid arteries, and paranasal sinus disease. Traumatic injuries have also been implicated, causing damage to the blood vessels supplying the optic nerve head via the relatively narrow subarachnoid space. Torsion and oedema compromise tissue oxygenation, resulting in a unilateral altitudinal defect having a horizontal border, great density and steep edges.[2] In a recent case report altitudinal hemianopsia developed after severe facial injuries during a motor vehicle accident.[3] But in contrast to our case, bone fragments were found to apply pressure to the optic nerve, and no retinal contusion was noticed. A history of ocular trauma was present in 5 of 27 patients under the age of 30 years reviewed at Wills Eye

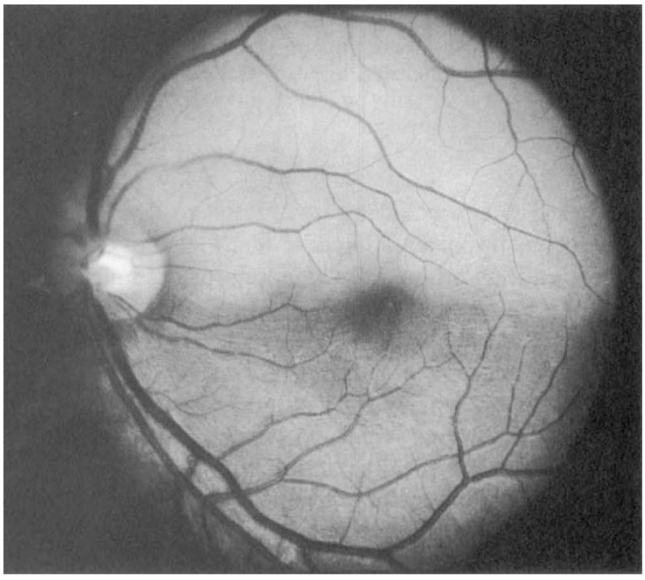

Fig. 1. *The left posterior pole. Note the opaque superior retina and the horizontal line, traversing through the fovea, separating healthy and diseased retina.*

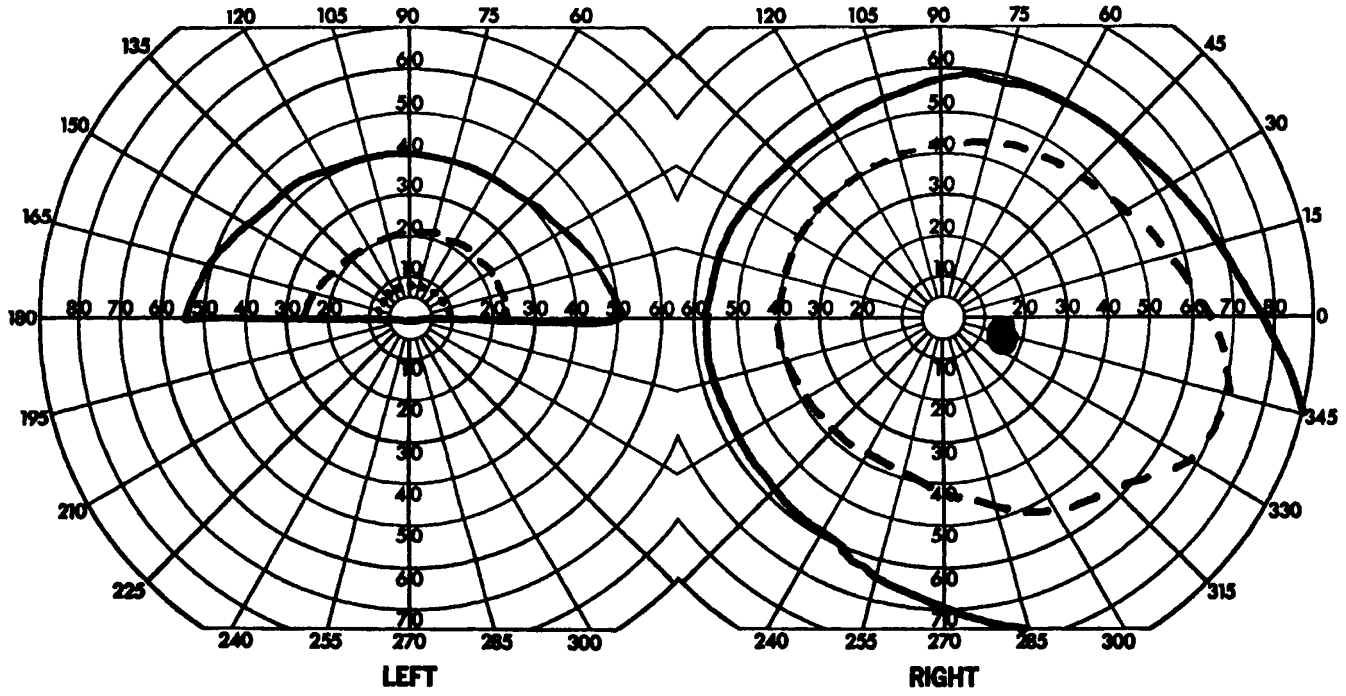

Fig. 2. *Goldmann's visual field chart. Note the inferior altitudinal defect in the left eye.*

Hospital for retinal arterial obstruction (RAO). Four of the 5 had an obvious direct insult: a retrobulbar haemorrhage for a surgical repair of a blow-out fracture, a knife wound, secondary glaucoma and hyphaema following a blunt trauma (in a patient with a sickle cell trait), and a metallic foreign body severing the optic nerve. In the fifth patient the pathogenesis was unclear. Goldmann's visual fields disclosed only scotomas with sloping borders and no specific mention of a horizontal lining. Visual field defects were also associated with traumatic choroidal rupture,[5] but there was no correlation between the ophthalmoscopic picture and the visual field defects as regards size, shape or location of the lesions. In none of the reported cases were the retinal contusion and field of vision delineated along the horizontal line without evidence of retinal vascular occlusion or optic nerve compression. One possible explanation for the retinal lesion in this case is a transient superior papillary arterial spasm or embolisation that, even when resolved, left permanent damage to the hypoxaemic retina.

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Sir,

Dangerous Reflections During Argon Laser Photocoagulation

For the past two years I have been using an HGM argon laser mounted in a Zeiss slit lamp, and have encountered a worrying problem. This takes the form of a dazzling, yellow-white reflex from the treatment beam, which appears whenever the laser is fired. It occurs with all types of contact lenses that I have used, and with different types of contact substance between the cornea and the contact lens. It is present, but in a very mild form, with other argon lasers I have used.

The reflexes are sufficiently dazzling in certain positions of gaze as to make prolonged laser photocoagulation acutely uncomfortable for the ophthalmologist. I showed a video of the reflexes, entitled 'Dangerous Reflections', at the recent annual meeting of the College, and I wonder whether any other ophthalmologists have experienced this type of problem with this laser or with others.

The reflex would seem to originate from the protective yellow filter with the laser and might be the 'filter fluorescence' that Sliney and Mainster have described.[1] After some delay, the suppliers of our laser suggested modifying the filter with a mask, thus reducing the filter