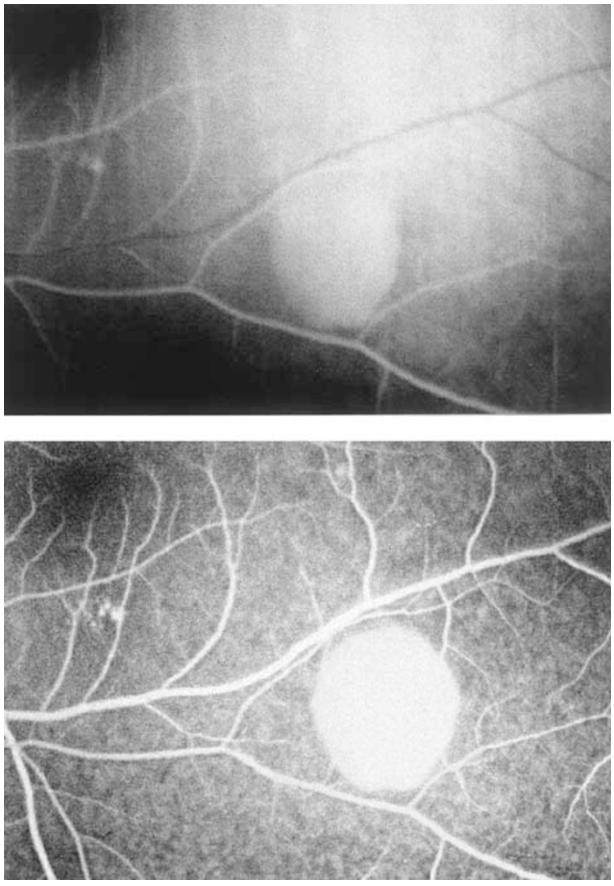

Figure 2 Fluorescein angiogram, left eye, showing (top) early homogeneous filling of pigment epithelial detachment and (bottom) increase in intensity but not size of hyperfluorescence in the late phase. Also seen are areas of RPE window defects inferior to the fovea.

choroid may either result in localized RPE changes such as pigment mottling and clumping or cause capillary and venous congestion with increased fluid transudation and RPE detachment.[4] Both types of lesions were present in our patient.

The histological hallmark of syphilis is obliterative endarteritis affecting the small arteries and arterioles.[5] At the level of choroidal vasculature, this would lead to choroidal ischaemia and might explain the ocular findings seen in our patient. However, ocular manifestations of syphilis are known to resolve rapidly with systemic treatment. Pigment epithelial detachments on the other hand, by their nature, would take time to settle once formed. This would explain the slow resolution of ocular findings in this case. The fact that no new detachments appeared over a 2-year follow-up supports the presence of an initial triggering event.

Unfortunately, there is no non-invasive method of establishing aetiology and the coincidental co-occurrence of these two events cannot be ruled out. Much more clinical evidence would be needed if a causal relationship was to be established.

In conclusion, we report here an interesting finding of multifocal asymptomatic RPE detachments in a patient of neurosyphilis.

Acknowledgement

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References

1 Margo CE, Hamed CM. Ocular syphilis. *Surv Ophthalmol* 1992; **37**: 203–204.
2 Giovanni A, Scassellati-Sforzolini B, D'Altobrando E *et al*. Choroidal findings in the course of idiopathic serous pigment epithelium detachment detected by indocyanine green videoangiography. *Retina* 1997; **17**: 286–293.
3 Uyama M, Matsunaga H, Matsubara T *et al*. Indocyanine green angiography and pathophysiology of multifocal posterior pigment epitheliopathy. *Retina* 1999; **19**: 12–21.
4 Prunte C, Flammer J. Choroidal capillary and venous congestion in central serous chorioretinopathy. *Am J Ophthalmol* 1996; **12**: 26–34.
5 von Lichtenberg F. Infectious disease. In: Cotran R, Kumar M, Robbins SL (eds). *Robbins Pathologic Basis of Disease*. WB Saunders Company: Philadelphia, 1989, pp. 368–371.

S Anand and AS Mushin

Department of Ophthalmology
Royal London Hospital
Whitechapel
London E1 1BB, UK

Correspondence: Dr S Anand
53, Croft Gardens
Birkby
Huddersfield
HD2 2FL
Tel: +44 797 6907 456
E-mail: seema180@hotmail.com

Sir,

Spontaneous suprachoroidal haemorrhage associated with high myopia and aspirin

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Spontaneous suprachoroidal haemorrhage occurs in age-related macular degeneration (ARMD) and anticoagulants are a recognised risk factor.[1] In highly

myopic patients, suprachoroidal haemorrhage occurs in situations when the fragile vasculature is put under additional stress such as in cataract surgery.[2] There have been no reports of large spontaneous suprachoroidal haemorrhage in myopic patients. The role of anticoagulants as a contributing factor in these patients is unknown.

Case report

A 78-year-old lady attended eye casualty complaining of a 2-day history of mild discomfort in the left eye, followed by an episode of complete loss of vision in the eye, which came back to normal after 1 h. The eye now felt tender and she complained of a visual disturbance 'like bubbles' in the temporal periphery of the left eye. She had had two similar episodes over the past 2 years, but had not sought medical help on those occasions.

She was hypertensive, on bendrofluazide, enalapril and 75 mg of aspirin, and a nonsmoker.

She had been seen in eye clinic since 1954 with high myopia ($-11.00/-2.00 \times 63$ right; $-9.00/-1.25 \times 104$ left). Her axial lengths were 31.18 mm right and 29.14 mm left. Her visual acuity since 1983 in the right eye was hand movements secondary to myopic macular degeneration, choroidal neovascular membrane and a dense brunescent cataract. The left eye's visual acuity was 6/12. The intraocular pressures were 17 mmHg in the right and 20 mmHg in the left. There was a nuclear sclerotic cataract in the left eye and no signs of haemorrhage in the vitreous. On fundoscopy she had extensive peripapillary atrophy and a myopic looking disc. There were multiple areas of chorioretinal atrophy. Anteriorly, encompassing 360° was a raised choroidal mass and there was extensive subretinal haemorrhage (Figure 1). B-Scan ultrasound confirmed the characteristics of a choroidal haemorrhage (Figure 2).

Routine blood tests including a clotting screen were entirely normal. She subsequently underwent fluorescein angiogram once the haemorrhage had resolved, which excluded a subretinal neovascular membrane or telangiectasia.

After 1 month, her visual symptoms and haemorrhages had resolved spontaneously, visual acuity remained 6/12.

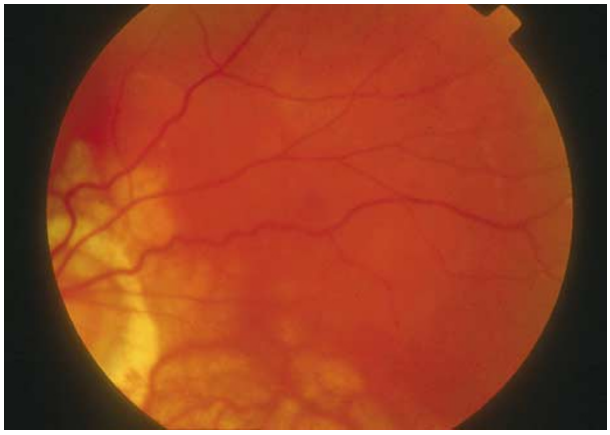

Figure 1 Fundus colour photograph of the left eye showing extensive subretinal haemorrhage, which encompassed 360°.

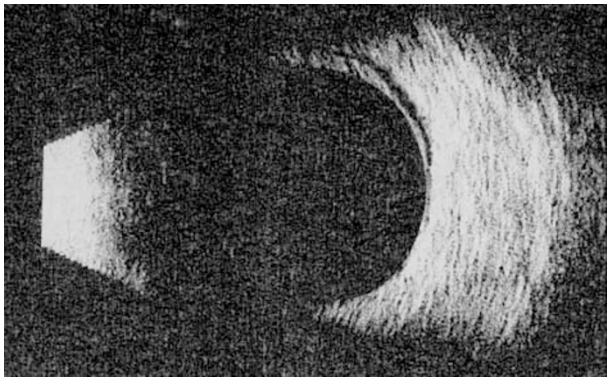

Figure 2 B-Scan ultrasound of the left eye confirming choroidal haemorrhage.

Comment

Following an exhaustive search of Medline/Pubmed and Ovid databases, large spontaneous suprachoroidal haemorrhage associated with high myopia has not, to our knowledge, been published previously in the literature. Myopia is a risk factor for suprachoroidal haemorrhage in cataract surgery because the longer axial length causes increased choroidal vascular fragility.[2] This patient was also hypertensive and it has been widely reported that hypertension, arteriosclerosis and advanced age are systemic risk factors for suprachoroidal haemorrhage in a surgical setting,[3] but the association with these risk factors in spontaneous haemorrhage is unknown. This patient may also have had ARMD, which may not have been apparent on the fluorescein angiogram. A review of ARMD complicated by massive haemorrhage reported on subretinal and vitreous haemorrhage only,[4] as suprachoroidal haemorrhage in ARMD appears to be relatively rare. On balance, therefore, we believe that although these other factors may have contributed to the haemorrhage, myopia was the primary cause.

This patient was also on low-dose aspirin and this may have contributed to the extent of her haemorrhage.[5] The risk of spontaneous suprachoroidal haemorrhage in association with myopia and the additional risk of anticlotting agents have not been studied. Subconjunctival haemorrhage, spontaneous hyphema, subretinal or vitreous haemorrhage and suprachoroidal

haemorrhage have been described with anticoagulation.[6] One case controlled retrospective study of 50 patients with ARMD showed an odds ratio of 11.6 that a patient with a massive intraorbital haemorrhage would also be on anticoagulants (warfarin or aspirin). In another study, the antiplatelet odds ratio (aspirin) was smaller at 2.1.[7]

Whether these risks can be extrapolated to myopic eyes is unknown. This rare case illustrates how high myopia associated with choroidal vasculature fragility exacerbated by hypertension could cause spontaneous haemorrhage. This was further aggravated by the presence of aspirin in this patient.

References

1 Lewis H, Sloan SH, Roos RY. Massive intraocular haemorrhage associated with anticoagulation and age related macular degeneration. *Graefes Arch Clin Exp Ophthalmol* 1988; **226**: 59–64.
2 Beatty S, Lotery A, Kent D, O'Driscoll A, Kilmartin DJ, Wallace D, Baglivo E. Acute suprachoroidal haemorrhage in ocular surgery. *Eye* 1998; **12**: 815–820.
3 Chu TG, Green RL. Suprachoroidal haemorrhage. *Surv Ophthalmol* 1999; **43**: 471–486.
4 el Baba F, Jarrett WH 2nd, Harbin TS Jr, Fine SL, Michels RG, Schachat AP. Massive hemorrhage complicating age-related macular degeneration. *Ophthalmology* 1986; **93**: 1581–1592.
5 Salomon O, Huna-Baron R, Steinberg DM, Kurtz S, Seligsohn U. Role of aspirin in reducing the frequency of second eye involvement in patients with non-arteritic anterior ischaemic optic neuropathy. *Eye* 1999; **13**: 357–359.
6 Alexandrakis G, Chaudhry NA, Liggett PE, Weitzman M. Spontaneous suprachoroidal haemorrhage in age related macular degeneration presenting as angle-closure glaucoma. *Retina* 1998; **18**: 485–486.
7 Tilanus MAD, Vaandrager W, Cuypers MHM. Relationship between anticoagulant medication and massive intraocular haemorrhage in age-related macular degeneration. *Graefes Arch Clin Exp Ophthalmol* 2000; **238**: 482–485.

M Chak[1] and TH Williamson[2]

[1]Department of Paediatric Epidemiology and Department of Ophthalmology
Institute of Child Health
30 Guilford Street
London WC1N 1EH, UK

[2]Ophthalmology Department
St Thomas' Hospital
Lambeth Palace Road
London, UK

Correspondence: M Chak
Tel: +44 207 905 2335
Fax: +44 207 242 2723
E-mail: m.chak@ich.ucl.ac.uk

Sir,

Closure of cyclodialysis cleft using diode laser

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Cyclodialysis clefts are a result of disinsertion of the ciliary body from the scleral spur. These can be caused traumatically, commonly following blunt injury, or iatrogenically following anterior chamber surgery. The consequence of this is a communication between the anterior chamber and the suprachoroidal space with a newly created path for aqueous drainage. As a result, the eye becomes hypotonus with choroidal effusions, macular folds and decreased visual acuity.

Identification of the cleft can be difficult using the slit-lamp gonioscope because of a shallow anterior chamber and a collapsed drainage angle. If the anterior chamber is deepened using viscoelastic, then the cleft can often be seen clearly.

Treatment of this condition aims to restore normal intraocular pressure, resolve choroidal effusions and as a consequence, restore visual function.

Medical management is based on the apposition of the ciliary body against the scleral spur and promotion of adherence by scar formation. This is enhanced by strong mydriasis for up to 6 weeks, anterior chamber inflammation and minimal use of steroid medications.

Several surgical methods have been employed in management of cyclodialysis clefts. These include invasive methods such as vitrectomy, cryotherapy and gas tamponade,[1] scleral buckle,[2] diathermy,[3] suturing[4] and endolaser.[5] Noninvasive methods have also been employed including trans-scleral YAG or diode laser.[6,7]

We describe two cases of successful cyclodialysis cleft closure using trans-scleral diode laser.

Case reports

Patient A is an 81-year-old lady who was diagnosed with primary open-angle glaucoma in 1997 and started on topical antihypertensive therapy. In view of inadequate intraocular pressure (IOP) control and progressing disease, right trabeculectomy was performed shortly followed by left trabeculectomy. IOPs were stabilised at 12 mmHg in both eyes. After 2 years, the patient underwent left cataract extraction (phacoemulsification and posterior chamber IOL). At 2 months following this, the patient presented with decreased visual acuity (VA) (6/24) and IOP of 0 mmHg with large choroidal effusions. A cyclodialysis cleft was identified in the inferotemporal aspect and closure was achieved using transcleral diode laser (21, 1.5 s burns at a power of 2000 mW) to the cleft under direct endoscopic visualisation through a corneal paracentesis. IOP