points of the study and thank him and his colleagues for their remarks.

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Proprietary interest: none

*Eye* (2007) **21,** 1131–1132; doi:10.1038/sj.eye.6702907; published online 29 June 2007

Sir,

## Delayed transient macular ischaemia due to ocular siderosis

We present a case of unilateral, transient macular ischaemia, presenting 1 year after vitrectomy, following classical ocular siderosis.

### *Case report*

A 35-year-old man reported blurred vision in his left eye for 4 months; he recalled hammering concrete 6 months previously. Left visual acuity was 6/24, 6/9 pinhole, right 6/4. The left eye displayed classical ocular siderosis, with mydriasis, iris rust staining, ferrous lenticular deposits, and posterior subcapsular cataract. Additionally, there was a full-thickness corneal scar, traumatic iridotomy and an inferior pars plana foreign body (FB). The right eye was normal throughout.

Left pattern electroretinogram and rod-specific responses were virtually undetectable. Left maximal responses were profoundly electronegative with a normal a-wave.

He underwent three-port pars plana vitrectomy, forcep FB removal, phacoemulsification, and lens implantation, without intravitreal antibiotics. Visual acuity was 6/6 2 weeks post-operatively, and 1 year later remained 6/6; however, examination revealed new macular cotton wool spots (Figure 1). Fundus fluorescein angiography (FFA) showed patchy macular capillary non-perfusion and cystoid macular oedema, which was not observable clinically (Figure 2).

Despite proven macular ischaemia, visual acuity and electrodiagnostics remained unchanged. The right eye remained normal. Blood pressure, full blood count, glucose, electrolytes, autoantibodies, and clotting were normal. Over 6 months, the cotton wool spots changed slightly in pattern, and then disappeared.

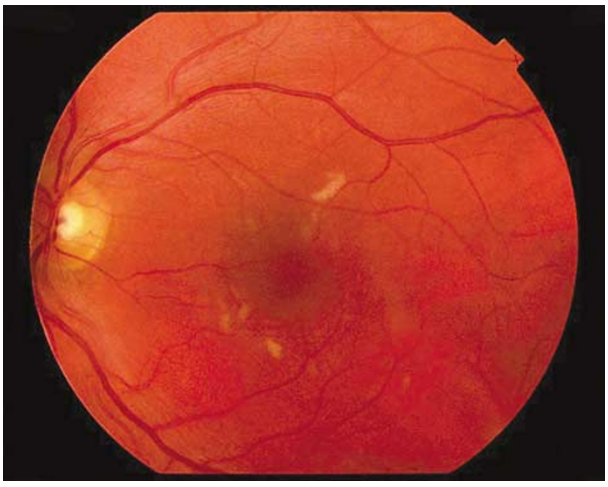

**Figure 1** Fundus photograph of the left eye reveals macular cotton wool spots.

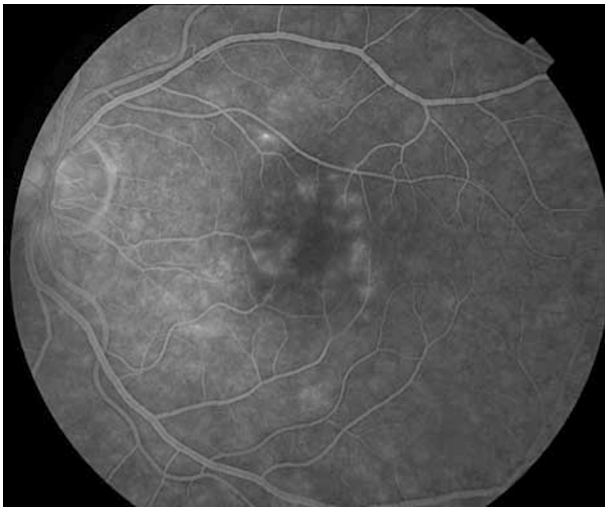

**Figure 2** Fundus fluorescein angiogram of the left eye reveals patchy macular capillary non-perfusion and cystoid macular oedema.

### Comment

In this classical case of ocular siderosis, transient ischaemic microangiopathy developed 1 year after vitrectomy and FB removal. Visual acuity and electrodiagnostics were unaffected. Alternative local and systemic causes of retinal ischaemia were excluded.

Cystoid macular oedema, arteriolar attenuation, and retinal pigment epithelium changes owing to siderosis have been reported, mimicking retinitis pigmentosa.[1–3] A gradient of microvascular ischaemia away from an iron FB has been reported;[1] in our case, FB and ischaemia are distant. In a similar report of delayed toxic macular microvasculopathy, visual acuity decreased 1 year after vitrectomy and FB removal, with perifoveal arcade staining on FFA and presumed perivascular iron deposits.[2]

Pathologically, iron dispersion throughout the globe results in intraretinal accumulation within intracellular siderosomes and oxidative injury.[4,5] We propose this to

be the likely cause of the transient macular ischaemia, a little recognised sequela of ocular siderosis.

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None of the authors listed have proprietary interests to declare. No research funding was received. This material has not been presented previously.

*Eye* (2007) **21,** 1132–1133; doi:10.1038/sj.eye.6702910; published online 15 June 2007