pseudophakic cystoid macular edema. *Acta Ophthalmol Scand* 2003; **81**: 648–652.
4 Karacorlu M, Ozdemir H, Karacorlu S. Intravitreal triamcinolone acetonide for the treatment of central retinal vein occlusion in young patients. *Retina* 2004; **24**: 324–327.
5 Karacorlu M, Mudun B, Ozdemir H, Karacorlu S, Burumcek E. Intravitreal triamcinolone for the treatment of cystoid macular edema secondary to Behcet disease. *Am J Ophthalmol* 2004; **138**: 289–291.
6 Ozdemir H, Karacorlu M, Karacorlu S. Regression of serous macular detachment after intravitreal triamcinolone acetonide in patients with diabetic macular edema. *Am J Ophthalmol* 2005; **140**: 251–255.
7 Miyake K, Ota I, Ibaraki N, Akura J, Ichihashi S, Shibuyo Y *et al*. Enhanced disruption of the blood-aqueous barrier and the incidence of angiographic cystoid macular edema by topical timolol and its preservative in early postoperative pseudophakia. *Arch Ophthalmol* 2001; **119**: 387–394.

M Karacorlu

Istanbul Retina Institute Ophthalmology, Hakki Yeten Cad. No: 8/7, Fulya, Besiktas, Istanbul 34349, Turkey

Correspondence: M Karacorlu,
Tel: +90 212 2313121;
Fax: +90 212 2332425.
E-mail: mkaracorlu@superonline.com

Eye (2006) **20,** 862–863. doi:10.1038/sj.eye.6702041; published online 12 August 2005;

Sir,

A case of occult giant cell arteritis presenting with bilateral cotton wool spots

Early diagnosis and treatment of giant cell arteritis (GCA) is important to prevent blindness in one or both eyes.[1] Occult giant cell arteritis, now a well-established entity, is defined as ocular involvement of GCA without any systemic symptoms and signs of giant cell arteritis.[1] The ocular symptoms and ischaemic lesions can be seen in a variety of combinations. We describe a case of occult GCA who presented with history of transient visual loss and bilateral cotton wool spots (CWSs).

Case report

A 59-year-old male Caucasian, who was otherwise fit, sought consultation complaining of a history of transient bilateral visual blur lasting for a few hours followed by complete resolution. Questioning specifically for systemic signs and symptoms associated with GCA at or before the onset of visual symptoms proved negative. His past medical history was unremarkable.

Visual acuities in both eyes were 6/6. His colour vision, visual fields, pupillary exam and anterior segment examination were normal. Fundus examination showed numerous elevated, whitish CWSs in both eyes with normal looking optic discs and maculae (Figure 1). The patient's superficial temporal arteries were nontender and pulsatile on both sides. His cardiovascular exam including carotid auscultation was normal.

ESR was elevated at 112 mm/h and CRP was 85 units. Fluorescein angiography showed poor and reduced filling of the retinal circulation with normal choroidal circulation and no disc oedema. He was not started on any treatment but underwent an urgent temporal artery biopsy. Histopathology showed mononuclear cell infiltration of the vessel wall, fragmentation of the internal elastic lamina, and multinucleated giant cells thus confirming the diagnosis of GCA. While awaiting his biopsy results, he re-presented with vision in his right eye having reduced to counting fingers with no obvious disc swelling. He was commenced on high-dose oral steroids and his vision improved to 6/6 with some constriction of peripheral visual field.

Comment

Occult GCA, a potential cause of blindness, is defined as ocular involvement of GCA without any systemic

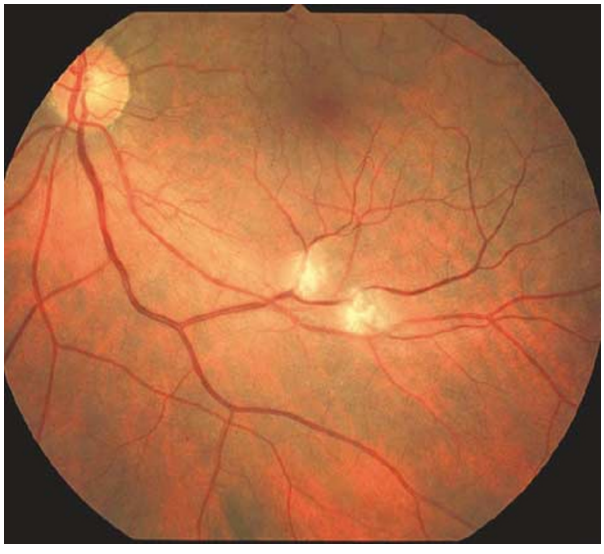

Figure 1 Fundus photograph showing CWSs, normal looking disc, and macula.

symptoms and signs of GCA.[1] The ocular symptoms include visual loss, amaurosis fugax, diplopia and eye pain.[1] Ocular ischaemic lesions described are anterior ischaemic optic neuropathy, posterior ischaemic optic neuropathy, central retinal artery occlusion, cilioretinal artery occlusion and CWSs.[1] The symptoms and signs can be seen in a variety of combinations.

CWS is an important clinical sign of associated systemic disease. It is widely held to reflect focal inner retinal ischaemic lesions; however recent perspective[2] suggest CWSs to comprise localised accumulation of axoplasmic debris within adjacent bundles of unmyelinated ganglion cell axons.

Hayreh *et al*[1] describes CWSs in up to one third of eyes with visual loss during the early stages of occult GCA. In a recent study, Asensio *et al*[3] describes two patients who were presented with single isolated CWS as the only clinical manifestation. They suggest CWSs can be an early ophthalmoscopic finding in GCA and can precede an important visual loss.

In our patient, transient visual blur was the only presenting symptom and CWSs were sole manifestation of temporal artery biopsy positive occult GCA. Although he developed reduction in vision related to poor central retinal artery perfusion, prompt systemic steroid treatment resulted in him regaining his central vision at its previous level.

We are not aware of any publication in which bilateral CWSs are the only presenting sign of occult GCA. In conclusion, the sole finding of CWSs and nonspecific visual symptoms in a patient aged >55 years needs to be investigated further to exclude the possibility of occult GCA.

References

1 Hayreh SS, Podhajsky PA, Zimmerman B. Occult giant cell arteritis: ocular manifestations. *Am J Ophthalmol* 1998; **125**: 521–526.
2 McLeod D. Why cotton wool spots should not be regarded as retinal nerve fibre layer infarcts. *Br J Ophthalmol* 2005; **89**: 229–237.
3 Asensio Sanchez VM, Pegalajar Maeso M, Rodriguez Bravo I, Carrasco E. Cotton wool spots as the first manifestation of giant cell arteritis: two cases. *Arch Soc Esp Oftalmol* 2004; **79**(9): 449–451.

P Velusami[1], M Doherty[1] and L Gnanaraj[2]

[1]Department of Ophthalmology, Royal Victoria Infirmary, Newcastle upon Tyne, UK

[2]Department of Ophthalmology, Sunderland Eye Infirmary, Sunderland, UK

Correspondence: L Gnanaraj,
Department of Ophthalmology, Sunderland Eye Infirmary, Eye Clinic, Queen Alexandra Road, Sunderland, Tyne and Wear, SR29HP, UK
Tel: +44 191 5699963;
Fax: +44 191 5699060.
E-mail: Lawrenceg@doctors.org.uk

The authors have no proprietary interest in any of the materials used in this study

Eye (2006) **20,** 863–864. doi:10.1038/sj.eye.6702038; published online 12 August 2005

Sir,

Inappropriate investigation and management of a retinal vascular occlusion

Gupta and co-workers[1] recently presented an interesting example of segmental macular retinal infarction whose aetiology was not discoverable despite multiple invasive tests and whose management by paracentesis was complicated by submembranous prefoveal haemorrhage and Roth's spots. From the angiographic evidence available, however, it appears highly likely that their 21-year-old patient was not the victim of an isolated macular branch arterial occlusion as your correspondents had supposed. Rather this was an example of (superior) hemisphere retinal vein obstruction with secondary inner retinal infarction in the territory of a cilioretinal arteriole. Although 'nonischaemic' obstruction of the *central* retinal vein is not infrequently complicated by cilioretinal infarction, cilioretinal ischaemia secondary to *hemisphere* venous obstruction has been reported only once before.[2]

Importantly, cilioretinal infarction from hemisphere retinal vein occlusion provides an opportunity to remove any lingering doubt that may exist as to the inter-relationship between the venous and arteriolar occlusions.[3,4] Failure of perfusion affects only that part of the cilioretinal circulation drained by the obstructed hemisphere vein so, of the two, the venous obstruction must be the instigating occlusion.[2] The basis of the association between these two vascular events is said to be the lower perfusion pressure in the inner retina supplied by cilioretinal arterioles in comparison with that in the territory of the central retinal artery,[5] but this assertion tends to hide the true picture. Significant differential effects on perfusion are only manifest when blood circulation through the inner retina is seriously challenged (eg, by marked elevation of the pressure in the central retinal vein or during