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.

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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

ophthalmodynamometry) and they then reflect the differing patterns of branching of the central retinal and posterior ciliary arteries.[2,6]

This alternative diagnosis circumvents the requirement for extensive investigation in a young patient like this, not least the futile search for a source of arterial embolism. It also obviates any need to postulate an underlying retinal microvasculopathy of systemic origin to account for the 'decompression retinopathy'.[1] Thus, hemisphere retinal vein obstruction predicts and explains the haemorrhagic consequences of a procedure (ie, paracentesis) that is ineffectual anyway in its aim of improving cilioretinal perfusion. I say this having made the same mistake myself 30 years ago.[7] Ocular hypertension as noted in this patient may have been a factor in determining the extent of the retinal haemorrhage developing after the sudden lowering of intraocular pressure. Moreover, it was probably pathogenic in respect of the original hemisphere venous obstruction.

Unfortunately, the loculated haemorrhage that developed at the fovea as a result of the paracentesis may well have had an adverse effect on this patient's eventual visual acuity. Spontaneous improvement in vision is otherwise the rule when only a sector of the perifoveal capillary net has had its perfusion compromised as part of a combined retinal venous and cilioretinal occlusion.[5] This highlights the importance of recognising the true nature of such occlusions, which represent a high proportion of the vascular accidents affecting the retina of young adults, have no association with serious systemic disease, and tend to resolve without intervention.[5,8]

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Sir,

Reply to Professor McLeod

We would like to thank Professor McLeod's interesting and stimulating comments regarding our recent case report concerning the formation of Roth spots and retinal haemorrhages after paracentesis for a presumed segmental retinal artery occlusion.[1] We agree with Professor McLeod that it is of fundamental importance to determine whether the initial event in our patient's disorder was arterial or venous in nature, as this could affect future prognosis and would have altered the acute management. Our patient presented within a few hours of a sudden and profound onset of a central scotoma, which reduced his visual acuity to hand movements. When he was examined, he was found to have an area of retinal thickening and pallor in the superior macular area. There were no retinal haemorrhages or venous changes noted. Based on the clinical history and the clinical findings, a presumed segmental retinal arterial occlusion was diagnosed and treated accordingly. The retinal venous changes and haemorrhages developed immediately after the paracentesis, suggesting that they were secondary to this intervention, perhaps due to an increase in the transmural pressure gradient across the affected retinal veins.

When the fundus fluorescein angiogram was performed, immediately after the paracentesis, the combination of arterial occlusion and masking haemorrhage prevented one determining if the occluded vessel was a branch of the central retinal artery or a true cilio-retinal artery. Subsequent FFA after resolution of the fundal changes and possible recanalisation of the