Sir,

Multiple retinal haemorrhages (decompression retinopathy) following paracentesis for macular branch artery occlusion

A 21-year-old man sought treatment for sudden onset of a right inferior central scotoma. Examination revealed visual acuities of hand movements OD and 6/6 OS. Intraocular pressures (IOP) were 26 mmHg OD and 23 mmHg OS. Gonioscopy showed bilateral goniodysgensis with prominent anterior iris processes. Ophthalmoscopy disclosed a pale oedematous retina in the superior part of the right macula consistent with a superior macular branch artery occlusion. Both discs were cupped with cup disc ratios of 0.7 OD and 0.8 OS. Despite immediate treatment with intravenous Acetazolamide 500 mg and topical Timoptol 0.25%, visual acuity failed to improve. A 0.2 ml anterior chamber paracentesis was performed, during which the anterior chamber was noted to shallow abruptly with the formation of corneal folds but without corneal endothelial touch. No deformation of the globe occurred during the procedure. Immediate fundoscopy revealed a preretinal foveal haemorrhage. No change in acuity was noted. One day later, further superficial haemorrhages had developed over the optic disc and multiple white centred retinal haemorrhages (Roth spots) were noted in the temporal retina (Figures 1 and 2). Fundus fluorescein angiography confirmed a right superior macular branch artery occlusion (Figures 3 and 4).

General examination showed no other signs of capillary fragility, and findings from a systemic examination, including transoesophageal echocardiography, cranial, orbital, and carotid magnetic resonance imaging, lumbar puncture, abdominal ultrasound, and serial blood pressure measurements, were unremarkable. Blood tests for and including complete blood count, erythrocyte sedimentation rate, fasting blood glucose, C reactive protein, autoimmune screen, clotting screen, angiotensin converting enzyme, glucose, treponema, human immunodeficiency virus, blood, and urine culture were normal. After 4 months, visual acuity remained 2/60 OD and the fundal haemorrhages had resolved.

While the underlying cause of the patient's branch artery occlusion remains unexplained, the temporal relationship and the exclusion of diabetes mellitus, severe hypertension, leukaemia, infective endocarditis, and systemic vasculitis suggest that the development of the Roth spots and fundal haemorrhages were due to a paracentesis-associated decompression retinopathy.

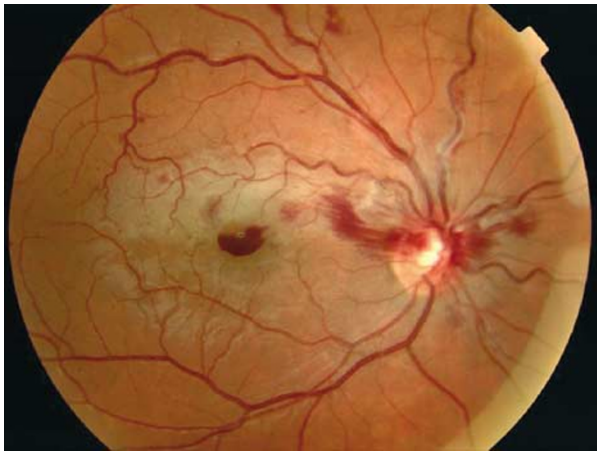

Figure 1 Colour fundus photograph taken 48 h after presentation of the right eye demonstrates marked retinal whitening in the superior aspect of the posterior pole associated with scattered preretinal haemorrhages.

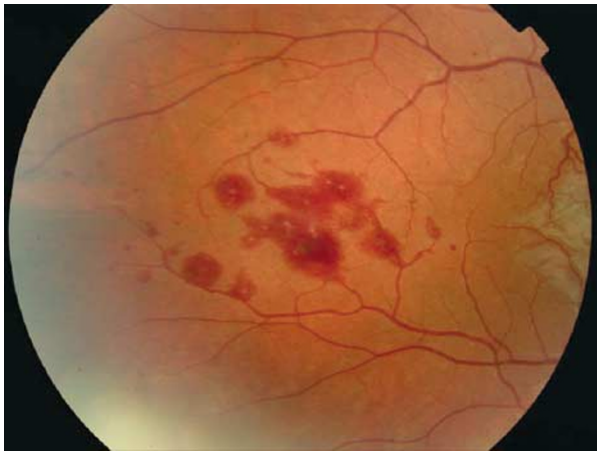

Figure 2 Fundus photograph of the right temporal retinal demonstrates multiple white centred haemorrhages (Roth spots).

Comment

A Roth spot is known to be composed of a fibrin platelet thrombus that arises at the site of capillary damage.[1,2] These white centred haemorrhages are observed in a variety of conditions, including ocular decompression.[3–5] Conditions that lead to an altered clotting state, capillary trauma, capillary endothelium ischaemia, or an increase in capillary fragility may lead to retinal haemorrhages.[3] Following paracentesis for acute angle closure glaucoma, no observed cases of decompression retinopathy were noted and it was suggested that a vasculopathy may be required to predispose a patient to decompression retinopathy.[6] In our case, the possibility of capillary fragility from an inherited or acquired abnormality is possible, but despite extensive investigation no systemic cause for the artery occlusion was found. A variety of hypotheses have been suggested for the cause of retinal

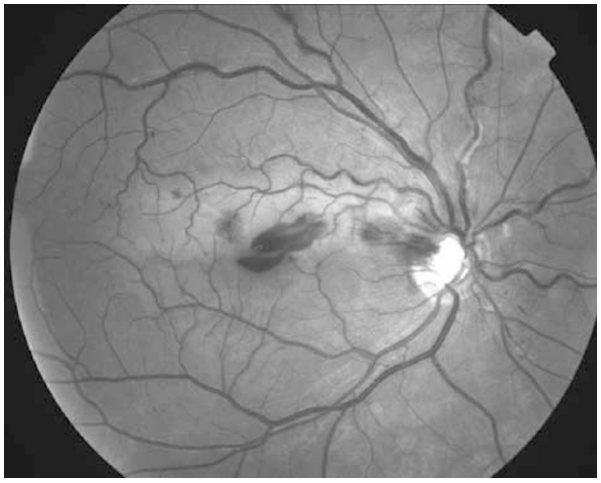

Figure 3 Red free fundus photograph of the right eye corresponding to Figure 1.

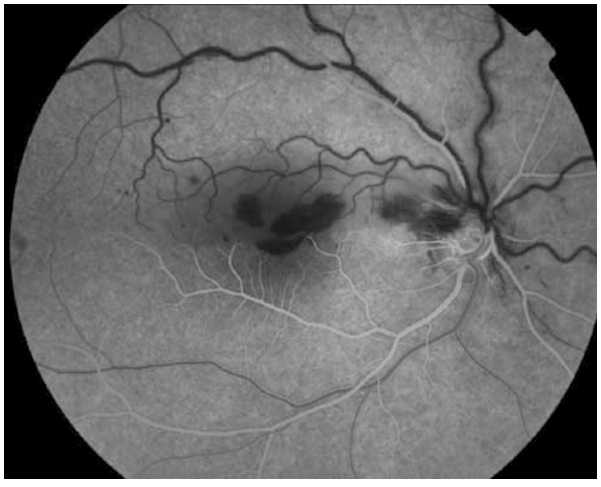

Figure 4 Arteriovenous phase of a fluorescein angiogram taken 48 h after presentation demonstrates delayed filling of a macular arteriole and masking defects due to the retinal oedema and overlying retinal haemorrhages.

haemorrhages following IOP reduction.[4] Normal IOP preserves the normal corneal–scleral curvature. Acute hypotony may result in structural changes to the eye tissues, and the effects vary depending on the hypotony duration. During hypotony, scleral collapse and deformity can occur. The inferior oblique inserts into the submacular area and its action causes horizontal folding of the choroid.[7] Perhaps the mechanical structural stresses associated with sudden IOP lowering in an already ischaemic macular lead to capillary fragility. The finding of a macular haemorrhage immediately following the paracentesis would be consistent with this. Alternatively, the increased pressure gradient across the walls of the retinal vasculature caused by the sudden drop in IOP may also have led to a transient increase in retinal blood flow, which either overwhelmed the integrity of the affected retinal arterioles or led to an uncontrolled increase in flow through ischaemic retinal venules, causing them to leak. The transient nature of the increase in retinal blood flow, does however remain speculative as flow through the affected retinal artery was not demonstrated on angiography performed 48 h after presentation.

We believe this case demonstrates the first case of multiple scattered white centred retinal haemorrhages in association with the macular artery occlusion ischaemia following rapid IOP reduction by paracentesis.

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