(significantly red) whose incidence remained fairly similar. This group comprised mainly patients with conjunctival haemorrhage but it would be difficult to exclude those with persistent conjunctival injection. It is also anticipated that the surgical procedure might have contributed to increase the incidence of red eye but we were unable to explore this because of the design of the study. The main objective of our study was to assess conjunctival appearance following sub-Tenon's block by an objective method and no attempt was made to differentiate between conjunctival injection or haemorrhage or indeed the influence of the surgical procedure. A remediable weakness of our methodology therefore, was in not specifying whether redness was due to haemorrhage or injection. Future methodology would therefore have a grading for redness followed by a suffix indicating injection, haemorrhage or both. Although crude, we feel this method of assessment if universally adopted, would provide a more reproducible method of assessing conjunctival appearance after sub-Tenon's block. As sub-Tenon's technique is still imperfect and continuously evolving[9] a more objective and reproducible method of assessment is essential.

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Sir,

**Spontaneous cystoid macula oedema in chronic granulomatous disease: a new posterior segment sign**

Chronic granulomatous disease (CGD) is a rare inherited disorder in which phagocytes lack a functional NADPH oxidase and so cannot generate superoxide anions. This impaired function leads to the failure to kill catalase positive microbes such as *Staphylococcus aureus*.[1] The most common form is X-linked caused by mutations in CYBB encoding $gp91^{phox}$, the heavy chain of flavocytochrome b(558).[2] Systemically, patients are characterised by hypergammaglobulinemia, hepatosplenomegaly, and lymphadenopathy. Typical ocular lesions associated with this condition are chorioretinal scars. We present a case of a young male who developed spontaneous macula oedema associated with this condition, which has not previously been described as an ocular complication.

***Case report***

A 10-year-old male with a genetically proven diagnosis of CGD had been referred to the Southampton Eye Unit because of a vitreous haemorrhage in his left eye. He had been under follow-up at Great Ormond Street and Yeovil Hospitals. Previously, he had had a bone marrow transplant in July 2002 and consequently developed graft versus host disease following this, which responded to steroids. He had also been noted to have chorioretinal scars bilaterally (Figure 1) that had no effect clinically on his vision and appeared to be stable.

He initially presented as an emergency to Yeovil Hospital with sudden loss of vision in his left eye (VA = HM) on 21 July 2003 due to a vitreous haemorrhage. A B-scan showed flat retina and the haemorrhage gradually resolved with no evidence of

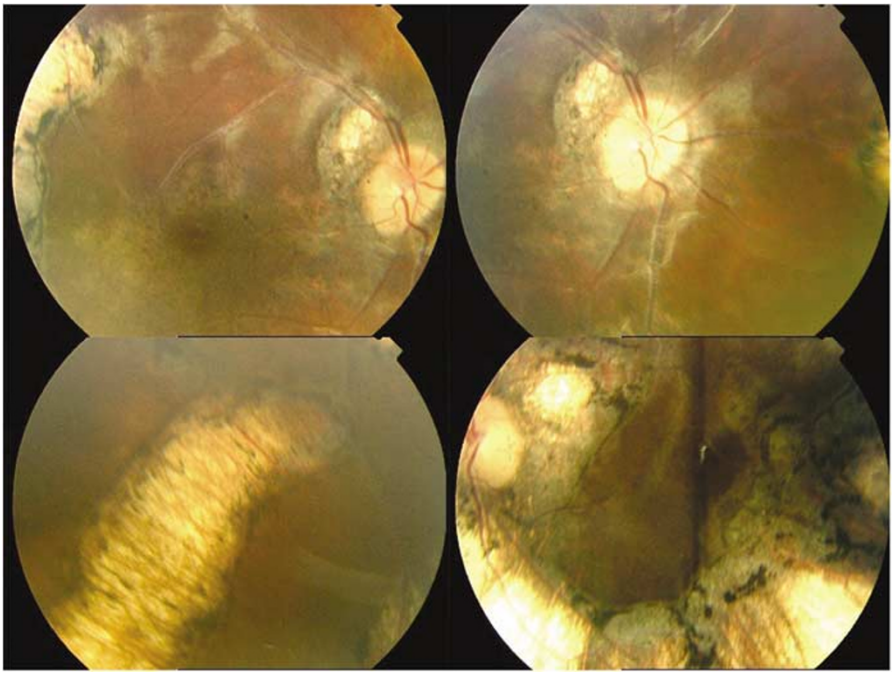

**Figure 1** Chorioretinal lesions in a child with CGD.

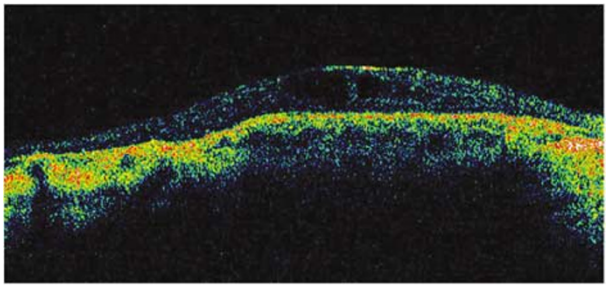

**Figure 2** OCT scans showing CMO.

neovascularisation seen. A referral to Southampton for a vitreoretinal opinion confirmed that the vitreous haemorrhage had resolved although the vision had only improved to 6/18 due to cystoid macula oedema (CMO) which had not been noted previously.

A 4-month follow-up showed no symptomatic change in vision at 6/18 in the left eye. An OCT was performed (Figure 2) and management was to observe. At 8 months, there was a worsening of the CMO. A subtenons injection of 30 mg of triamcinolone was performed in September 2004, which resulted in resolution of the CMO and an increase in vision to 6/6 in the left eye. Further 3-month follow-up revealed recurrence of the oedema with decrease in vision to 6/12 demonstrating only a transient effect of the triamcinolone for CMO. Clinically, the patient was happy with his vision and at this stage further observation only is required.

## Comment

Chronic granulomatous disease is a rare inherited disorder (X-linked or autosomal recessive) in which phagocytes lack the ability to undergo the respiratory burst necessary to kill certain types of bacteria and fungi leading to recurrent life-threatening infections. The exact incidence is unknown although estimated to be between 1 : 220 000–500 000.[3]

Initially described in the 1950s the patient invariably died in the first decade of life although significant advances have taken place since improving survival to a median of 25 years.[3]

Clinically, patients present with catalase positive infections in which granuloma formation and inflammatory processes predominate. The 'typical' retinal abnormality consists of 'punched out' chorioretinal lesions associated with pigment clumping lying along major retinal vessels.[4] Other documented ophthalmic complications in CGD include blepharoconjunctivitis and marginal keratitis.[5]

Spontaneous cystoid macula oedema has never been reported in association with this disease to our knowledge. The pathogenesis of this is unclear. It may be that the chronic CMO is a long-term complication of the bone marrow transplant (BMT) or graft versus host disease. BMT has been associated with a microvascular maculopathy consisting of cotton wool spots, exudates, haemorrhages, and CMO although there are no previous reports of isolated CMO as a late complication following

these events. In this case, the oedema initially underwent complete resolution following a subtenons injection of triamcinolone. The pathophysiology of this ocular manifestation in this condition is unknown.

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Sir,

**Indocyanine green angiography in diffuse unilateral subacute neuroretinitis**

Diffuse unilateral subacute neuroretinitis (DUSN) is a clinical syndrome caused by a subretinal nematode, initially described by Gass *et al*. [1] DUSN affects one or, occasionally, both eyes and usually results in severe loss of vision if left untreated. This disorder has two distinct stages. The early stage manifests inflammatory signs such as vitritis, papillitis, retinal vasculitis, and recurrent crops of evanescent yellow-white outer retinal lesions.[1] The late stage is characterized by progressive visual loss, optic atrophy, retinal vessel narrowing, and focal or diffuse retinal pigment epithelium (RPE) degeneration.[1]

Fluorescein angiography (FA) characteristics have already been reported in some cases of early and late-stage DUSN.[1] To our knowledge, this report describes for the first time the indocyanine green angiography (ICG-A) characteristics of a patient with early and late-stage DUSN.

### *Case report*

A 34-year-old man was referred to our Retina Clinic complaining of acute loss of vision of 7 days duration. His best-corrected visual acuity was 20/40 in the right eye (RE) and 20/20 in the left eye (LE). Slit-lamp examination of the anterior segments was unremarkable. Intraocular tension was within normal limits in both eyes. Fundus examination of the RE showed a pattern of multiple deep, 100–600 $\mu$m, yellow-whitish lesions situated near the infero-temporal vascular arcade. Some discrete yellow-white dots were also observed in the superior half of the fundus (Figure 1a). The posterior vitreous showed a mild inflammatory response. The optic disk and the retinal vessels were apparently healthy. The fundus of the LE was normal.

FA of the RE showed that the lesions masked fluorescence early during the study, and there was a significant hyperfluorescence in the late stages. Some late hyperfluorescence was also seen on the optic disc.

ICG-A of RE revealed that the lesions initially masked fluorescence. Most of these hypofluorescent dark spots were already visible in the early phase of the angiogram (Figure 1b), became more sharply delineated in the intermediate angiographic frames, but just a few spots remained hypofluorescent in the late frames (Figure 1d). The lesions seen in the initial phases of the ICG-A corresponded to those seen ophthalmoscopically. A fuzzy hyperfluorescence at the macular level, which started at the initial phases of ICG-A, persisted in the late frames of the angiogram.

We performed the following examinations: complete blood cell count, FTA-ABS, PPD, and chest X-ray. All results were normal. The diagnosis of DUSN was proposed, but after many visits we were not able to find the worm. Within 1 month, the corioretinal lesions became more evident in the superior half of the fundus and the patient's visual acuity dropped to 20/100 (Figure 1d). After 6 months, the worm was eventually located (Figure 2-b) and destroyed by laser photocoagulation. However, the patient's visual acuity was 20/400. Ophthalmoscopy performed at this time revealed round hypopigmented lesions throughout the posterior pole as well as diffuse RPE degeneration, mild optic disk atrophy and discrete