systemic neurofibromatosis. A thorough systemic examination in this patient showed no signs of neurofibromatosis. Thirdly, patients with these lesions often present at an earlier age than our patient in the second or third decade of life.

Kalina *et al*[2] reviewed the literature for isolated conjunctival neurofibromas and documented a detailed description of 13 cases in the literature including four from their own institution. Of the 13 cases, 10 had systemic neurofibromatosis. The lesions were located at the limbus in over half of the cases, and the remainder were located on the upper tarsal conjunctiva and on the temporal bulbar conjunctiva. At this location, they can be mistaken for a dermoid. None were documented at the puncta. Simple excision was curative in these cases, similar to ours, as the growth of these lesions is characteristically uniformly slow.

References

1 Knight III WA, Murphy WK, Gottlieb JA. Neurofibromatosis associated with malignant neurofibromas. *Arch Dermatol* 1973; **107**: 747–750.
2 Kalina PH, Bartley GB, Campbell RJ, Buettner H. Isolated neurofibroma of the conjunctiva. *Am J Ophthalmol* 1992; **113**(1): 112–113.

S Fenton[1] and MPh Mourits[2]

[1]Orbital Unit
Department of Ophthalmology
Cork University Hospital, Cork
Ireland

[2]Orbital Unit
Department of Ophthalmology
University Medical Centre
PO Box 85500 3508 GA Utrecht
The Netherlands

Correspondence: S Fenton
Tel: +35 321 4922 652
Fax: +35 321 4922 2656
E-mail: fentons@shb.ie

Sir,

Central serous retinopathy masquerading as sympathetic ophthalmia

Eye (2003) **17,** 666–667. doi:10.1038/sj.eye.6700423

Idiopathic central serous choroido-retinopathy (CSR) is a well-established clinical entity known to occur in type A personality individuals. Systemic corticosteroid therapy has also been recognised as a triggering factor.[1,2] We report a rare case of CSR developing in the fellow eye following enucleation and systemic corticosteroid therapy in a patient who had sustained a posterior globe rupture following a road traffic accident.

Case report

A 34-year-old male artist presented with a post-traumatic posterior globe rupture and optic nerve sheath haematoma of the left eye with multiple ipsilateral orbital fractures that were confirmed on ultrasonography and computerised tomography. The right eye was normal except for pre-existing myopia and no evidence of any macular pathology. He was treated with tetanus toxoid injection, intravenous antibiotics, analgesics, and oral prednisolone (1 mg/kg/day). His left eye was enucleated as a primary procedure after 6 days. The postoperative period was uneventful and the patient was discharged on oral prednisolone (1 mg/kg). Right eye vision remained normal with glasses. On the 15th postoperative day, the patient reported a marked decrease in vision of his right eye. The best-corrected visual acuity (BCVA) was 3/60 OD.

Biomicroscopic examination revealed mild retrolental flare with 1–2 cells with serous retinal detachment involving the macula. Humphrey perimetric examination (30-2) of the visual field showed a central scotoma. Fluorescein angiography (FA) showed a smoke stack leak typical of CSR (Figure 1). A provisional diagnosis of steroid- and stress-induced CSR was made. Gradual tapering of oral steroids was undertaken. Topical ketorolac (0.3%) and cycloplegic (atropine 1%) eye drops were prescribed under careful supervision. The retrolental flare and cells resolved within 24 h of topical treatment. A rapid improvement in visual acuity was noted over the next 4–5 days, and the patient was discharged with BCVA 6/18 OD. Visual fields repeated after 2 weeks demonstrated a decrease in the size of the scotoma. At 3 months follow-up, BCVA was 6/6 OD, and visual field defect had resolved. Serous retinal detachment had settled completely. FA showed only a small window defect (Figure 2).

Comment

This case represents the occurrence of CSR following high-dose systemic steroid therapy following a severe eye trauma. Central serous choroidoretinopathy is attributed to the disruption of the ionic pump of the RPE cells or owing to hyperpermeability of the choroidal vasculature. Glucocorticoids cause CSR probably because of increasing cAMP of RPE cells, and hence changing the

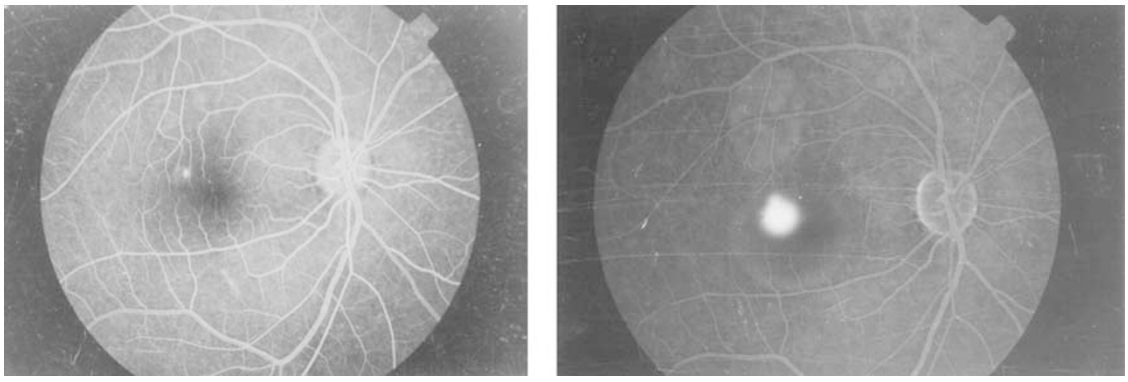

Figure 1 Early (left) and late (right) phases of fundus fluorescein angiogram showing a leak typical of CSR in the right eye at presentation.

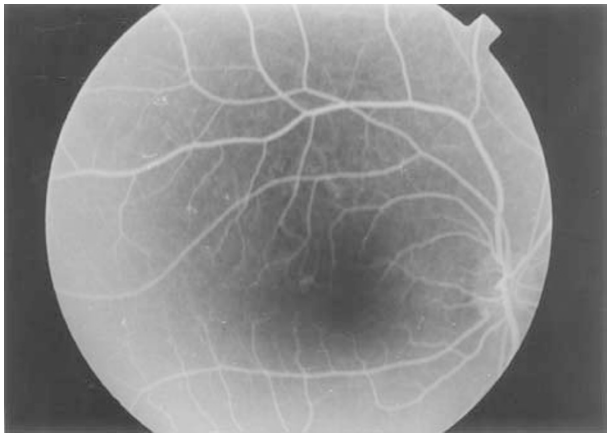

Figure 2 Fluorescein angiogram of right eye 12 weeks after stopping oral steroids showing a small window defect.

ionic pump function or by altering the permeability of the blood aqueous barrier and disrupting the outer blood retinal barrier.[3] Retrolental flare with few cells observed in this case could have been owing to breakdown of the blood aqueous barrier.

This case raises the question of whether prophylactic steroid therapy for sympathetic ophthalmia is justified particularly when it has been reported not to be foolproof.[4] It highlights the importance of considering CSR masquerading as sympathetic ophthalmia in such situations. Clinical improvement following tapering of steroids supports the diagnosis.

We wish to emphasise the importance of fundus FA in investigating patients at risk for sympathetic ophthalmia who present with diminished vision in the fellow eye. Ophthalmologists must be aware of the possibility of CSR precipitated by stress and steroids as in this case, as the management of CSR is completely different from that of sympathetic ophthalmia and the use of corticosteroids in such a case can be detrimental.

References

1 Bouzas EA, Moret P, Pournaras CJ. Central serous chorioretinopathy complicating solar retinopathy treated with glucocorticoids. *Graefes Arch Clin Exp Ophthalmol* 1999; **237**: 166–168.
2 Sharma OP, Rao N, Roy M. Sarcoidosis and central serous retinopathy: a dangerous combination. Sarcoidosis. *Vasc Diffuse Lung Dis* 1998; **15**: 189–191.
3 Zamir E. Central serous retinopathy associated with adrenocorticotrophic hormone therapy. A case report and hypothesis. *Graefes Arch Clin Exp Ophthalmol* 1997; **235**: 339–344.
4 Michael L Kay, Myron Manoff, James A Katowitz. Development of sympathetic uveitis inspite of corticosteroid therapy. *Am J Ophthalmol* 1974; **78**: 90–94.

R Tandon, M Vanathi, L Verma and A Bharadwaj

Dr Rajendra Prasad Centre for Ophthalmic Sciences AIIMS, New Delhi 110029, India

Correspondence: R Tandon
Fax: +91 11 685 2919
E-mail: radhika_tan@yahoo.com

Sir,

Traumatic optic nerve avulsion: role of ultrasonography
Eye (2003) **17,** 667–670. doi:10.1038/sj.eye.6700411

Optic nerve avulsion is a rare presentation of ocular trauma. The diagnosis of this entity is often obscured by the presence of concomitant vitreous haemorrhage, which precludes the visualization of optic head excavation. Various electrodiagnostic and expensive neuroimaging studies have not proved helpful in substantiating the diagnosis of this entity in the early stages and may not be readily available in most of the