common. About 45% of the patients have associated systemic disease. Severe pain is also a prominent feature and is not typically seen in episcleritis or marginal keratitis, which need to be differentiated from scleritis.[12] There were several findings in our patient that supported the diagnosis of scleritis, including characteristic pain, lack of blanching with vasoconstrictor, circumferential spread and residual scleral changes. The response to intensive topical steroid and the corneal signs were somewhat atypical, but these features support the likely association between APS and scleritis, as they resemble some of the anterior segment features previously described in APS.[3]

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Sir,

Recurrent cerebral lupus heralded by an unusual combination of ocular manifestations

A 41-year-old Chinese woman presented with spontaneous progressive painless blurring of vision and facial swelling for a week. She had a history of systemic lupus erythematosus (SLE) for 3 years, initially presenting with oral ulcer, leucopenia, serositis, positive antinuclear antibody and cerebral lupus.[1] The disease was stabilised clinically and serologically with hydroxychloroquine 200 mg daily and prednisolone 2.5 mg on alternate days. On examination, visual acuity was 20/50 in both eyes. There was bilateral non-tender periorbital oedema (Fig. 1A, B) and conjunctival chemosis (Fig. 1C, D). There was no sign of scleritis. The anterior chamber and vitreous were clear. Contact lens biomicroscopy revealed a bilateral symmetrical macular oedema, with multiple foveal fine yellowish dots at the level of the retinal pigment epithelium (RPE) (Fig. 2). The discs and vessels appeared unremarkable. Blood pressure and serum albumin were normal.

A few hours later she became drowsy, which lasted for 4 days. She was further managed in the intensive care unit. Computed tomography of the brain was normal. Lumbar puncture showed an opening pressure of 10.4 cm H_2O, raised protein of 2.5 g/l (reference range 0.15–0.45 g/l) and a negative smear. She was treated as having recurrent cerebral lupus with intravenous methylprednisolone 500 mg daily for 3 days and a single pulse of cyclophosphamide 500 mg.

Two weeks later, the macular oedema, periorbital swelling and conjunctival chemosis were partially resolved. A fluorescein angiogram showed faint hyperfluorescence in the fovea in the early phase with no leakage in the late phase, which was compatible with RPE dysfunction secondary to choroidopathy. There was no evidence of retinal vasculitis or scleritis. A month later the macular oedema, periorbital swelling and conjunctival chemosis all subsided. Visual acuity was 20/30 in the right eye, 20/20 in the left. Twenty months later, visual acuity was 20/20 in both eyes. There were fine macular RPE stippling changes in both eyes (Fig. 3).

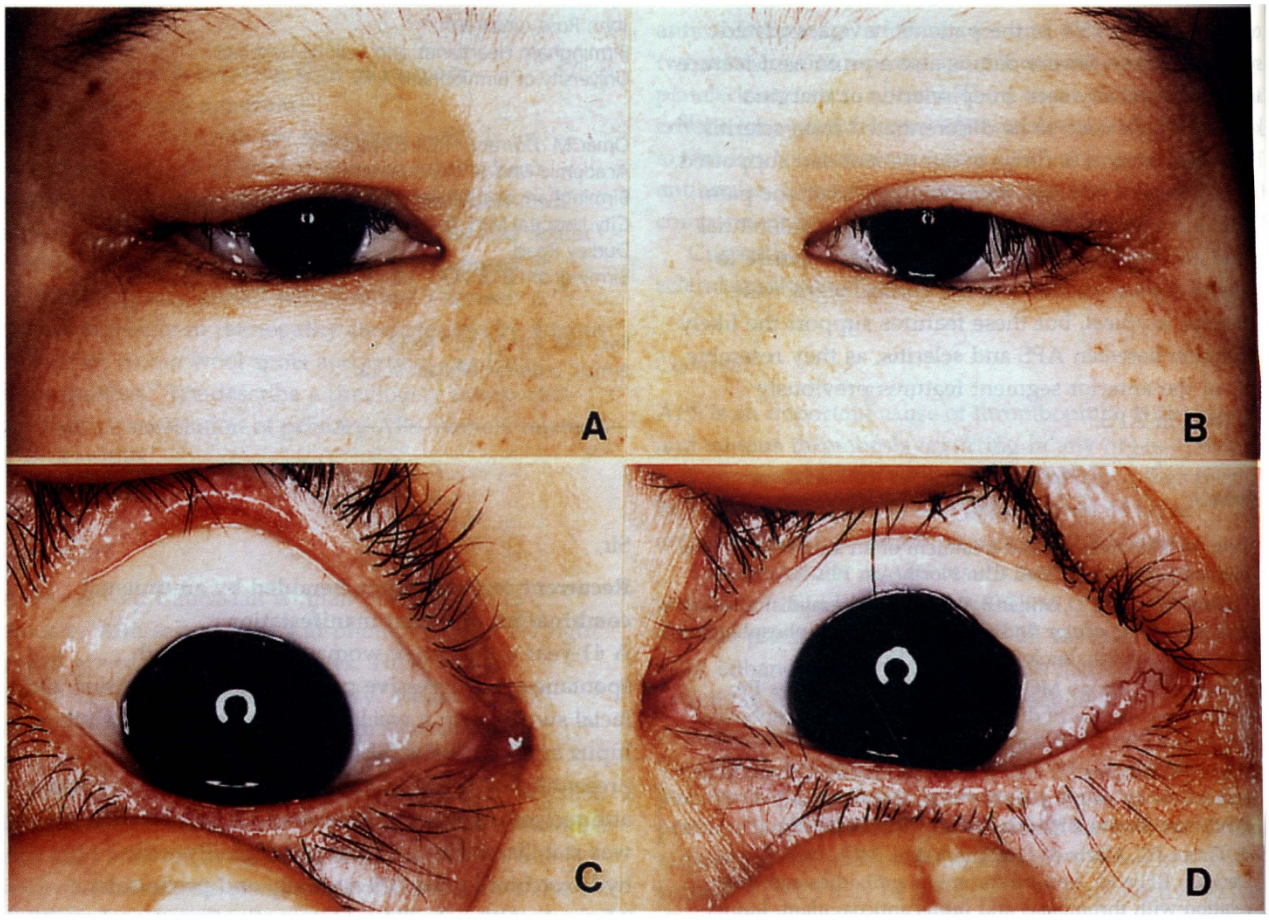

Fig. 1. *(A), (B) External photographs showing bilateral periorbital oedema. (C), (D) External photographs showing bilateral conjunctival chemosis. There is no sign of scleritis.*

Comment

Aronson *et al.*[2] reported a SLE patient with subretinal oedema. Immunoglobulin deposition over the choroidal capillaries and basement membranes of bulbar conjunctivas was disclosed subsequently at autopsy.

Leahey *et al.*[3] reported a SLE patient presenting with chemosis. The combination of bilateral macular oedema, periorbital swelling and conjunctival chemosis in SLE has not been reported before. This manifestation could result from ocular immune-complex vasculitis[2,4]

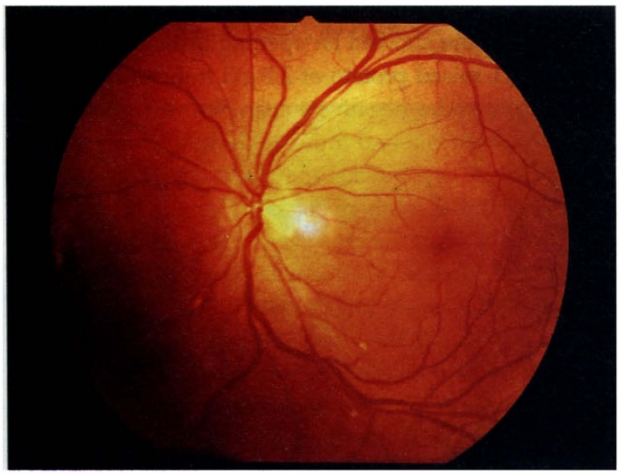

Fig. 2. *Fundus photograph of the left eye showing macular oedema of about 2 disc diameters, with foveal fine yellowish dots at the level of the retinal pigment epithelium. Three retinal pigment epithelial hypopigmented spots are present along inferior vascular arcades.*

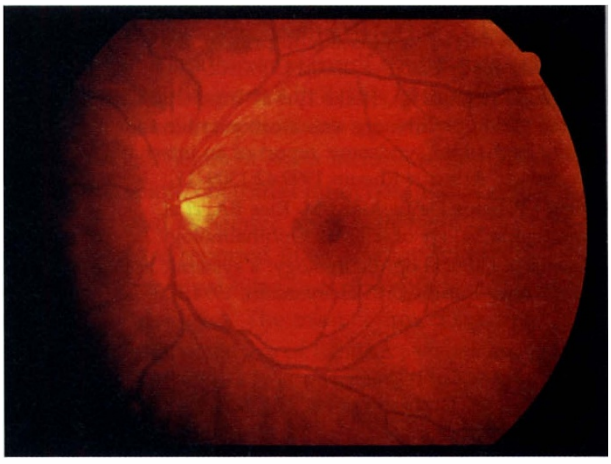

Fig. 3. *Fundus photograph of the left eye 20 months later showing fine macular retinal pigment epithelial stippling changes. The macular oedema has subsided. One retinal pigment epithelial hypopigmented spot is present superior to the disc.*

resulting in extravascular exudation of proteins. It heralded the exacerbation of disease in our patient. Hence, ocular involvement may serve as an indicator of disease reactivation in SLE.

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Sir,

Bilateral reactive subretinal abscesses following *S. pyogenes* septicaemia

Metastatic endophthalmitis can be seen in the context of generalised infection and its course depends on a number of factors including the virulence of the organism. Group A streptococcal endophthalmitis can present as a very aggressive infection with poor prognosis for vision.[1] We present a case of low-grade endophthalmitis associated with group A streptococcal meningitis, which presented after the treatment of the systemic illness and when the patient had been discharged home.

A previously healthy 68-year-old Caucasian woman was admitted with a 3 week history of general malaise and recent-onset lower back pain and rigors. On examination she was pyrexial (38 °C) with a reduced consciousness level, tenderness of the lumbar region and lower limb hyperreflexia. Group A *Streptococcus* was isolated from blood cultures and she was started on intravenous ceftriazone and amoxicillin. Lumbar puncture revealed markedly elevated protein (14.8 g/l, normal range 0–0.4 g/l) numerous neutrophils and occasional lymphocytes. *Streptococcus* A meningitis was diagnosed and the antibiotics were changed to high-dose intravenous benzylpenicillin and oral chloramphenicol according to antimicrobial sensitivities.

She continued to have a spiking pyrexia and MRI scanning of the lumbar region revealed L3,4 discitis with epidural and paraspinal inflammatory changes – presumably the source of the meningitis. With evidence of continuing inflammation despite her current antibiotic regimen, microbiological advice was sought. Broad antimicrobial coverage with oral clindamycin and ciprofloxacin together with intravenous cephradine was initiated and the patient's clinical condition began to improve. She was discharged home on oral clindamycin and ciprofloxacin.

Two months later, the patient presented to eye casualty complaining of blurred vision and floaters in both eyes since shortly after her original admission to hospital. She was still taking her oral antibiotics. On ophthalmic examination visual acuity was 6/12 in the right eye and 6/18 in the left. Apart from a few keratic precipitates, her anterior chambers were quiet with intraocular pressures of 11 mmHg in the right eye and 19 mmHg in the left. There was a mild vitritis with prominent pale subretinal lesions nasally in both eyes (Fig. 1), with no evidence of any immediate threat to vision.

Serology for both *Toxocara* and *Toxoplasma* was negative and it was assumed that the lesions were likely to be subretinal streptococcal abscesses. The patient continued to take oral ciprofloxacin and clindamycin for a further 9 months. Over the next 4 months lesions remained static and the vision improved to 6/9 in both

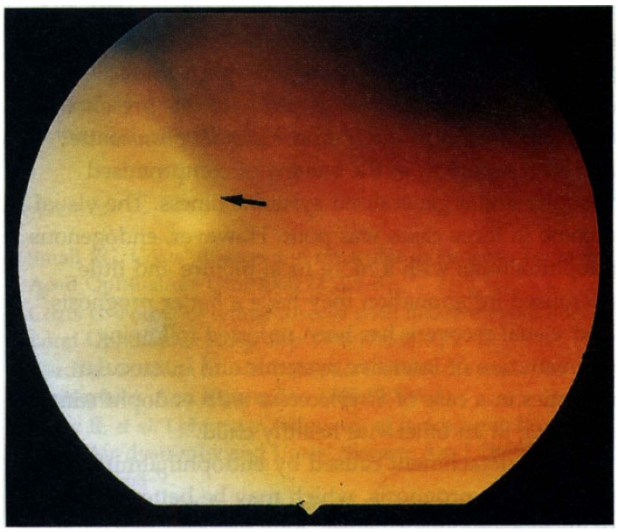

Fig. 1. *Photograph of the nasal aspect of the left fundus showing the large pale subretinal abscess (arrow).*