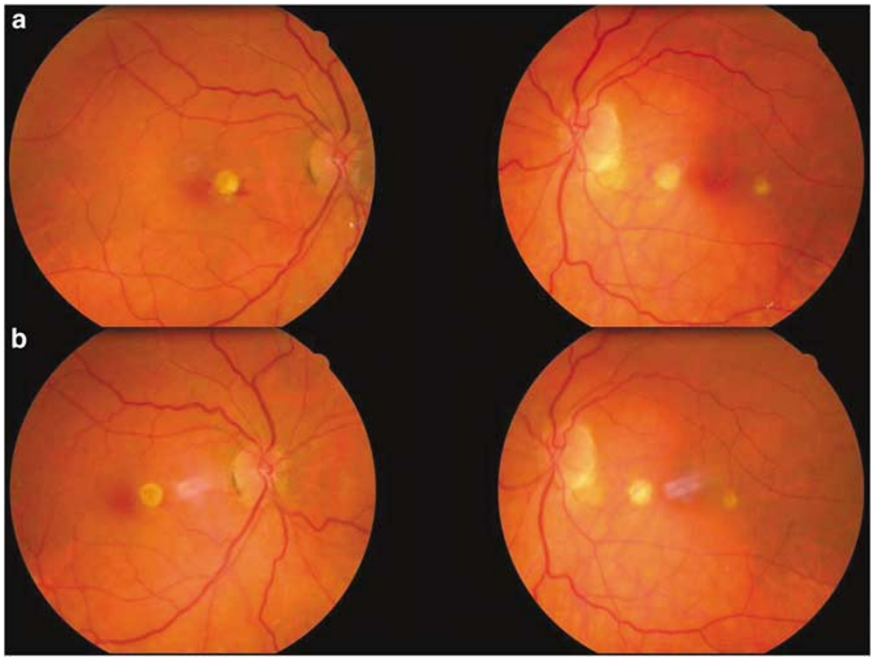

Figure 1 Both eye retinographies showing active chorioretinal lesions due to *Candida* on the baseline visit (a) and chorioretinal scars after treatment (b).

chorioretinal lesions after 3 weeks (Figure 1b). Final visual acuity was 20/30 in both eyes.

Comment

TNF-α is a proinflammatory cytokine, which plays an important role in the pathogenesis of inmune-mediated diseases and in the immune mechanisms against infection. The use of TNF-α inhibitors has been associated with an increased rate of intracellular infections.[4] Even so, there is little evidence about *Candida* infections among patients treated with etanercept. Wallis *et al*[4] cited a rate of 7.1 *Candida* infections (no reference to ocular affectation) per 100 000 patients who received etanercept. Its use, associated to chronic corticosteroid treatment and intravenous catheter, led to a fungal septicemia in our patient, which was followed by the bilateral chorioretinitis. In conclusion, we should consider *Candida* species as a possible etiology of chorioretinitis in patients taking etanercept.

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Sir,

Combined pharmacotherapy and thermotherapy for chronic central serous chorioretinopathy with anterior segment neovascularisation

Retinal vascular nonperfusion has long been recognised in chronic central serous chorioretinopathy (CCSC).[1] Occlusion of the peripheral vessels is often misdiagnosed as vasculitis, resulting in administration of corticosteroids.[1–3] We describe management of CCSC complicated by rubeosis in similar circumstances.

Case report

A 38-year-old healthy man presented with diminished vision in both eyes for many months. Best-corrected visual acuity was 6/24 in the right eye and 6/12 in the left eye. He was treated with oral corticosteroids elsewhere for presumed retinal vasculitis. He had no history of any previous ocular or systemic disease. Systemic examination and laboratory work-up by the

hospital internist were unremarkable. On ocular examination, anterior segment was unremarkable in the right eye; gonioscopy revealed neovascularisation of iris (NVI) and angle (NVA) in the left eye; the NVI is shown in Figure 1a. Ophthalmoscopy showed bilateral serous retinal detachments, subretinal precipitates/fibrin, and pigmentary alterations, suggestive of CCSC (Figure 1b). The left eye additionally had a neovascular frond inferiorly (NVE). Fluorescein angiography (FA) revealed parafoveal window defects and multiple leaks bilaterally. NVE in the left eye bordered extensive capillary nonperfusion (Figure 1c). Optical coherence tomography (OCT) showed shallow retinal detachments in both the eyes and cystoid macular oedema in the left (Figure 1d).

The patient was offered and explained about three treatment options: conventional photocoagulation, photodynamic therapy (PDT), and transpupillary thermotherapy (TTT). The patient declined PDT for financial reasons; TTT was considered the next best

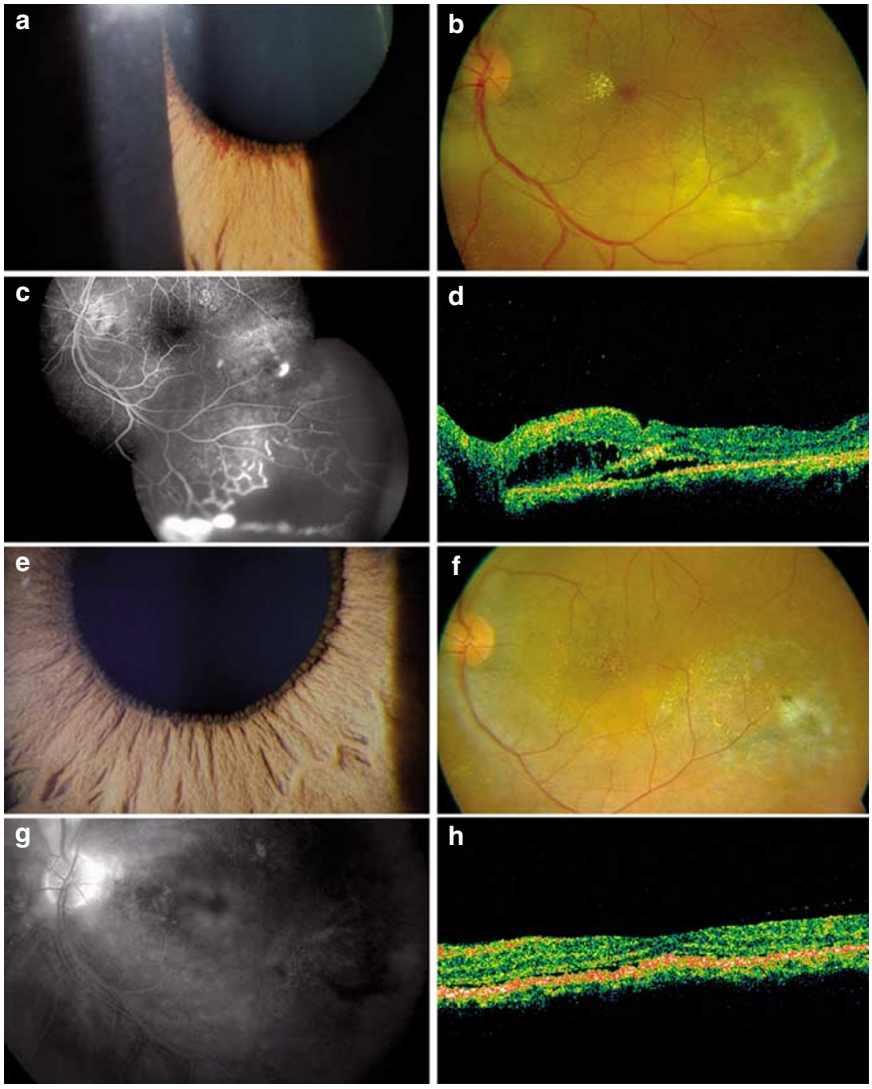

Figure 1 Pretreatment (a–d) and post-treatment (e–h) clinical, angiographic, and tomographic pictures of the patient's left eye. (a) Slit-lamp photograph showing neovascularisation of iris (NVI) along 6–7 o'clock meridians. (b) Fundus shows shallow serous detachment of the macula with retinal folds, subretinal precipitates nasal to the fovea, and large areas of subretinal fibrin accumulation around the optic nerve head and inferotemporal macula. (c) Midphase fluorescein angiogram montage reveals perifoveal areas of transmitted hyperfluorescence, consistent with diffuse retinal pigment epithelial degeneration. Two inkblot leaks are also visible in the inferotemporal area surrounded by soft 'ooze-like' leaks. Peripheral new vessels bordering the large inferior zone of capillary dropout leak profusely. (d) Horizontal 10 mm optical coherence tomography (OCT) scan through the macula demonstrates shallow foveal detachment and gross outer retinal cystoid changes in the nasal macula. (e) Slit-lamp view of the same sector of iris reveals complete resolution of the rubeosis. (f) Fundus examination shows resolution of the serous detachment of the macula and subretinal fibrin, replaced by pigmentary changes inferotemporal to the fovea, in the area treated with transpupillary thermotherapy. (g) Late-phase angiogram shows complete resolution of the temporal angiographic leaks. Fovea shows minimal hyperfluorescence. Peripheral neovascularisation had also regressed completely (not in picture). (h) OCT (horizontal 5 mm scan through the macula) showing trace subretinal fluid under fovea as well as nasal retina; the latter shows no cystoid changes now.

alternative due to its subthreshold nature. After obtaining the approval of the Institutional Review Board and informed consent of the patient, he was treated with TTT on a slit-lamp-mounted 810 nm diode laser. Two 3 mm spots (650 mW, 1 min) covered the angiographic leaks inferotemporal to the fovea in the left eye. The next day, TTT was delivered (five 2 mm spots; 220 mW, 1 min) to the right eye. No retinal blanching was observed in either eye. On the same day, intravitreal bevacizumab, 1.25 mg/0.05 ml (Avastin; Genentech, San Francisco, CA, USA), was injected in the left eye. One month later, there was a substantial reduction in the macular detachments in both the eyes; NVI, NVA, and NVE also resolved in the left eye. FA revealed regressing leaks bilaterally. At 4 months, no recurrence of NVI,NVA, or NVE was noted. FA showed complete cessation of angiographic leakage, and OCT showed near-flat maculae bilaterally (Figure 1e–h). Pretreatment visual acuity was maintained.

Comment

Although acute CSC has a favourable natural course, in about 5% cases, it is more severe and chronic, with poor visual outcomes. The more severe variants have been reported to be more common in Asians and Latinos.[2] CCSC with inferior longstanding RD may occasionally develop loss of retinal capillaries, NVE, and vitreous haemorrhage.[1–3] In spite of FA evidence of CCSC, many of these cases are misdiagnosed as retinoschisis, vasculitis or exudative uveal diseases, with misdirected medical or surgical treatment.[1–3] Treatment with corticosteroids tends to further increase the serofibrinous exudation and aggravate the retinal detachment,[2] as could have happened in our patient. Although retinal neovascularisation has been reported previously,[1–3] we are not aware of any literature report of anterior segment neovascularisation in CCSC. Although others have reported spontaneous regression of retinal neovascularisation after resolution of CCSC,[1,3] we chose to proactively treat the NVI/NVA because of the unacceptable risk of neovascular glaucoma. We preferred TTT to conventional photocoagulation to minimise retinal damage, in view of the diffuse treatment area. TTT has already been successfully used to treat CCSC.[4] Bevacizumab was chosen for its documented ability to reverse retinal/iris neovascularisation.[5] A potentially devastating complication in CCSC was averted, and a good vision was maintained by combining antiangiogenetic pharmacotherapy with subthreshold thermotherapy.

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