Sir,

Central serous chorioretinopathy as a complication of postcardiac transplant corticosteroid therapy

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Idiopathic central serous chorioretinopathy is a well-recognized clinical syndrome characterized by a well-defined shallow sensory retinal detachment in the macular region.¹ Rare variants of central serous chorioretinopathy have been reported in patients receiving systemic corticosteroid therapy.² We report a patient with atypical chorioretinopathy following postcardiac transplantation steroid therapy.

Case report

A 61-year-old man with a history of progressive visual loss in both eyes was referred with the diagnosis of atypical macular degeneration. The visual loss was slow over the preceding 4 years and started simultaneously about 6 months after the cardiac transplant. He was in remission following treatment for Hodgkin's lymphoma with chemotherapy and thoracic external beam radiotherapy 10 years earlier. In 1995, cardiac transplantation was performed for cardiomyopathy. For 3 months after the cardiac transplant he received high-dose prednisone (up to 80 mg/day), and at the time of our evaluation was on low-dose prednisone (5 mg/day), antihypertensives, and other immunosuppressants.

Figure 1 (a) Right eye fundus photograph showing multifocal retinal pigment epithelial alterations in the macular region. (b) Left eye fundus photograph showing multifocal retinal pigment epithelial alterations in the macular region and a vertical tract of retinal pigment epithelial migration and atrophy, suggestive of antecedent retinal detachment. (c) Optical coherent tomography showing a shallow neurosensory retinal detachment in the right eye. (d) The detached retina in the left eye is thinner than the adjacent attached retina indicating retinal atrophy.

The best-corrected visual acuity was 20/30 right eye and 20/400 left eye. The anterior segment of each eye was normal. Ophthalmoscopic examination showed multifocal alterations in the retinal pigment epithelium and a pocket of subretinal fluid in the macular region of both eyes (Figure 1a). A vertical tract of bone spicule pattern extended inferiorly in the left eye (Figure 1b). Optical coherent tomography confirmed the presence of localized shallow neurosensory retinal detachment in the macular region of both eyes (Figures 1c and d).³ With fluorescein angiography, mottled early and late hyperfluorescence was observed in the macular region of both eyes with a linear tract of hyperfluorescence in the left eye (Figure 2). The diagnosis of atypical central serous chorioretinopathy secondary to chronic use of corticosteroids was considered.

Comment

Central serous chorioretinopathy typically occurs in young healthy adults and is usually idiopathic. Central

serous chorioretinopathy is more prevalent in endogenous hypercortisolism and occurs in patients receiving corticosteroid therapy.^{2,4} Rapid onset of chorioretinopathy within a month of starting therapy with a total minimum dosage of 200 mg of prednisolone has been reported.⁵ Rare variants of central serous chorioretinopathy with chronic, bilateral, extrafoveal, multifocal, and bullous retinal detachments have been observed in patients undergoing cardiac transplantation.⁶ Ill-defined posterior chorioretinopathy in organ transplant recipients has also been reported. As these patients undergo various haemodynamic alterations and receive numerous immunosuppressive agents including steroids, it is difficult to implicate a single agent or mechanism in the pathogenesis of chorioretinopathy.⁷ Although the fundus findings in our patient are most consistent with the chronic usage of systemic corticosteroid therapy, the exact role of corticosteroids in inducing chorioretinopathy remains to be established.⁸

Patients with corticosteroid-induced chorioretinopathy should be considered for discontinuation or reduction in dosage of corticosteroids in consultations with their internists. The visual prognosis with corticosteroid-induced chorioretinopathy remains guarded especially because the patient's systemic condition may require them to have long-term corticosteroid therapy.

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References

- 1 Gass JDM. Pathogenesis of disciform detachment of the neuroepithelium. Part II. Idiopathic central serous choroidopathy. *Am J Ophthalmol* 1967; **63**: 587–615.
- 2 Wakakura M, Ishikawa S. Central serous chorioretinopathy complicating systemic corticosteroid treatment. *Br J Ophthalmol* 1984; **68**: 329–331.
- 3 Iida T, Hagimura N, Sato T, Kishi S. Evaluation of central serous chorioretinopathy with optical coherence tomography. *Am J Ophthalmol* 2000; **129**: 16–20.
- 4 Bouzas EA, Scott MH, Mastorakos G, Chrousos GO, Kaiser-Kupfer MI. Central serous chorioretinopathy in endogenous hypercortisolism. *Arch Ophthalmol* 1993; **111**: 1229–1233.
- 5 Harada T, Harada K. Six cases of central serous choroidopathy induced by systemic corticosteroid therapy. *Doc Ophthalmol* 1985; **60**: 37–44.
- 6 Friberg TR, Eller AW. Serosus retinal detachment resembling central serous chorioretinopathy following organ transplantation. *Gracfes Arch Clin Exp Ophthalmol* 1990; **228**: 305–309.

Figure 2 (a) Fluorescein angiogram (venous phase) showing multifocal macular hyperfluorescence in the right eye and (b) showing a vertical tract of hyperfluorescence corresponding to the area of RPE atrophy in the left eye.

- 7 Gass JDM, Slamovits TL, Fuller DG, Gieser RG, Lean JS. Posterior chorioretinopathy and retinal detachment after organ transplantation. *Arch Ophthalmol* 1992; **110**: 1717–1722.
- 8 Gass JDM, Little H. Bilateral bullous exudative retinal detachments complicating idiopathic central serous chorioretinopathy during systemic corticosteroid therapy. *Ophthalmology* 1995; **102**: 737–747.

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Sir,

Multifocal asymptomatic retinal pigment epithelial detachments in neurosyphilis

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Syphilis is a great imitator with protean ocular and systemic manifestations. It has been associated with a variety of posterior segment lesions. Reported manifestations include chorioretinitis, vasculitis, choroidal neovascular membrane, pigmentary retinopathy, retinal necrosis, retinal detachment, uveal effusion, central retinal vein occlusion, and big blind spot syndrome.¹ We report here a patient of neurosyphilis who presented with multifocal asymptomatic retinal pigment epithelium (RPE) detachments. To the best of our knowledge, there is no previous report of RPE detachment or the related finding² of central serous chorioretinopathy (CSC) in syphilis.

Case report

A 41-year-old male presented to the accident and emergency unit with generalized seizures. A diagnosis of neurosyphilis was made on the basis of positive serum and CSF VDRL along with positive TPHA and FTA-ABS. HIV testing was negative. He responded to a 17-day course of procaine penicillin with oral probenecid. Systemic steroids were not used in his treatment at any time. He was routinely referred to ophthalmology to rule out ocular involvement.

He had no ocular symptoms. Unaided visual acuities were 6/5. There was no evidence of present or past

intraocular inflammation. Fundus examination revealed bilateral, multifocal, well-circumscribed retinal pigment epithelium detachments. There were multiple lesions in both eyes. The right eye had one large detachment, about six disc diameters in size along the inferotemporal arcade (Figure 1). There were two smaller detachments, about a half disc diameter in size nasal to the fovea (Figure 1), and another about one disc diameter in size nasal to the disc. The left eye also had a large lesion, about four disc diameters in size, along the inferotemporal arcade (Figure 2) and another smaller one along the superotemporal arcade. There were bilateral scattered areas of pigment mottling and hypopigmentation. Fluorescein angiography showed early homogeneous filling of the detachments (Figure 2, top), with increase in intensity but not size of hyperfluorescence in the late phase (Figure 2, bottom), confirming the diagnosis of RPE detachment. Some scattered areas of localized hyperfluorescence were also present in both eyes, consistent with RPE window defects (Figure 2).

The patient was followed up at regular intervals. The detachments gradually subsided. At the last follow-up, 2 years after presentation, all the lesions had resolved. The patient maintained his vision at 6/5 bilaterally, with no evidence of inflammation in either eye.

Comment

Idiopathic serous detachments of the RPE in patients below 50 years of age are thought to be variants of CSC.² Although the pathophysiology remains controversial, most agree that increased choroidal vascular permeability is a prerequisite.^{2,3} It has further been suggested that choroidal ischaemia may be the primary event responsible for the altered permeability.⁴ This model of CSC suggests that ischaemia at the level of the

Figure 1 RPE detachments along the inferotemporal arcade and nasal to the macula, right eye.