

Sir,
Bilateral foveal neurosensory detachment in hypertensive retinopathy demonstrated by optical coherence tomography

The ophthalmologic manifestations of hypertensive retinopathy are well described in the literature, and include retinal arteriolar narrowing, retinal hemorrhages, nerve-fiber layer infarcts, retinal vein or artery occlusion, and optic nerve head swelling.¹ Focal retinal pigment epithelium detachments as well as exudative neurosensory detachments may develop as well, although reported in the literature rarely.² Herein, we report a case of malignant hypertension associated with bilateral foveolar neurosensory detachment demonstrated by optical coherence tomography.

Case report

A 45-year-old Asian female presented with complaints of bilateral lower extremity swelling, headache and blurring of vision of 3 weeks duration. Her past medical history was significant for noninsulin dependent diabetes, which had been controlled with diet, hypertension, and ischaemic heart disease. Her blood pressure on arrival to the emergency department was 182/92 with blood glucose of 330. She was subsequently admitted to the inpatient service for further work-up and treatment of hypertension and hyperglycemia. Systemic examination and laboratory testing was significant for an abnormal urinalysis with 3+ proteinuria and 2+ hematuria, which was felt to be consistent with a diagnosis of nephrotic syndrome. However, after 1 week on the inpatient service, she refused further interventions, and was discharged from the hospital against medical advice before a renal biopsy could be performed.

The following week, she was seen in the ophthalmology clinic, on a regimen of lisinopril, glucophage, lasix, and metoprolol. Subjectively she noted decreased vision bilaterally for over 1 month, and denied any significant past ocular history other than refractive error. Visual acuity with correction was 20/70 right eye and 20/200 left eye. Anterior segment biomicroscopy, pupillary examination, and tonometry were unremarkable bilaterally. Ophthalmoscopy revealed narrowing of retinal arteries, bilateral retinal exudation with macular star formation and multiple retinal haemorrhages and cotton wool spots (Figure 1). There was no optic nerve head swelling evident, and she was diagnosed with grade 3 hypertensive retinopathy. Ocular coherence tomography examination of the macular area revealed bilateral foveolar serous

neurosensory detachment (Figure 2). Also of note are the numerous areas of high intensity signal intraretinally, corresponding to areas of lipid exudation. Clinically, there was a small amount of fibrinous material subretinally, which is manifest in the OCT scan as the signals scattered throughout the subretinal space.

The patient's blood pressure remained under good control medically, and seven months following her initial presentation her vision had returned to 20/30 right eye and 20/100 left eye, with resolution of the foveal detachments (Figure 3) but persistence of intraretinal exudates.

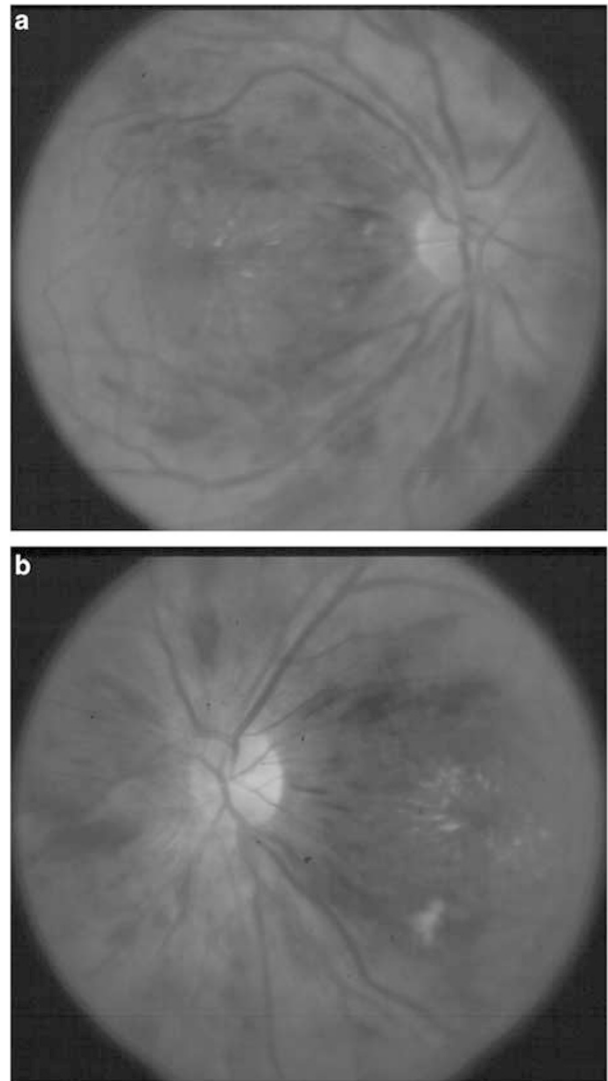


Figure 1 Fundus photographs of right eye (a) and left eye (b) demonstrate diffuse retinal arteriolar narrowing, intraretinal hemorrhages, cotton wool spots, and macular oedema with star formation.

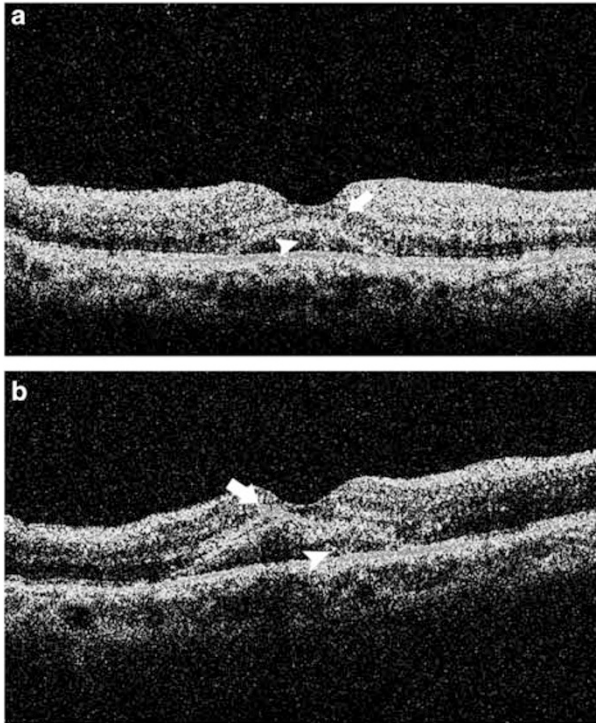


Figure 2 Optical coherence tomography of right eye (a) and left eye (b) shows bilateral, serous, foveal neurosensory detachment. Note the high signal areas intraretinally and subretinally, corresponding to intraretinal exudates (arrows) and subretinal fibrinous material noted clinically (arrowheads).

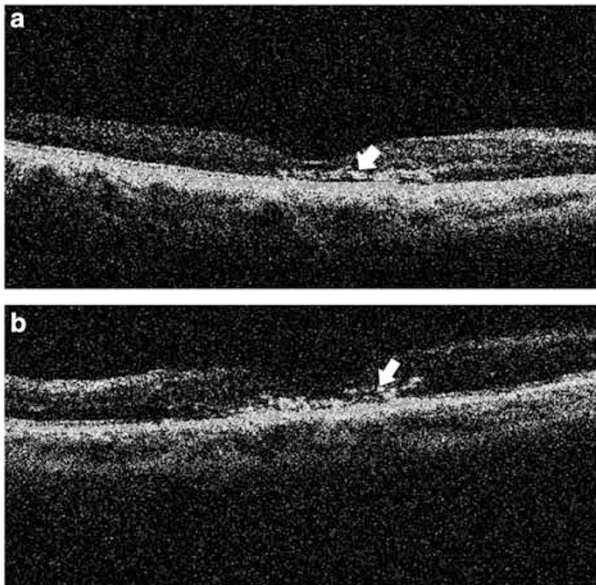


Figure 3 Optical coherence tomography of right eye (a) and left eye (b) demonstrates resolution of foveal neurosensory detachments seven months after initial presentation. Areas of high signal intensity indicate persistent intraretinal exudates (arrows).

Comment

Malignant or accelerated hypertension can be defined as rapidly progressive renal failure in the presence of moderate or severe hypertension, and is often associated with grade 3 or 4 hypertensive retinopathy.³ Neurosensory detachment associated with accelerated hypertension is thought to result from an acute ischaemic insult to the choroid, and has been reportedly infrequently in the literature. Visual loss secondary to hypertensive retinopathy can be secondary to vitreous haemorrhage, macular oedema, choroidal infarct, or optic nerve head swelling.⁴ The degree of visual recovery is variable and dependent upon successful treatment of the underlying hypertension. Based on our observations, there may be a subset of patients with moderate to severe visual loss secondary to foveolar neurosensory detachments. To our knowledge, this is the first report of a case of hypertensive retinopathy associated with bilateral foveolar neurosensory detachment as demonstrated by optical coherence tomography.

References

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