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Sir,

Giant retinal pigment epithelial rip in polypoidal choroidal vasculopathy with vitreous haemorrhage after pars plana vitrectomy

Polypoidal choroidal vasculopathy (PCV) is a variant type of age-related macular degeneration. We present an unusual case with PCV and vitreous haemorrhage, developing a giant retinal pigment epithelial rip after pars plana vitrectomy.

Case report

A 63-year-old man complained of a sudden painless visual field defect in the right eye since 2 weeks, along with floaters and progressive blurry vision that developed during the second week. At presentation, his visual acuity was counting fingers in the right eye. Funduscopic photograph of the right eye showed moderate vitreous haemorrhage and bullous subretinal haemorrhage in the inferotemporal area. Tissue plasminogen activator was injected intravitreally 24 h preoperatively, followed by phacoemulsification, intraocular lens implantation, and pars plana vitrectomy with endotamponade of 10% perfluoropropane. Three weeks later, funduscopic photograph of the eye showed a giant retinal pigment epithelial (RPE) tear outside the inferotemporal vascular arcade (Figure 1a). Fundus fluorescein angiography revealed an occult choroidal neovascularization and a giant RPE tear with pooling of the dye to the subretinal space (Figure 2a and b). Indocyanine green angiogram (IGA) showed a hyperfluorescent spot of polypoidal choroidal vasculopathy located beside the inferotemporal fovea (Figure 2c and d). IGA-guided laser photocoagulation was applied on the polypoidal lesion. At 12-month follow-up, best-correct visual acuity was 20/30 in the right eye. Fundoscopic examination showed a persistent RPE rip, residual subretinal haemorrhage, and resolution of submacular haemorrhage (Figure 1b).

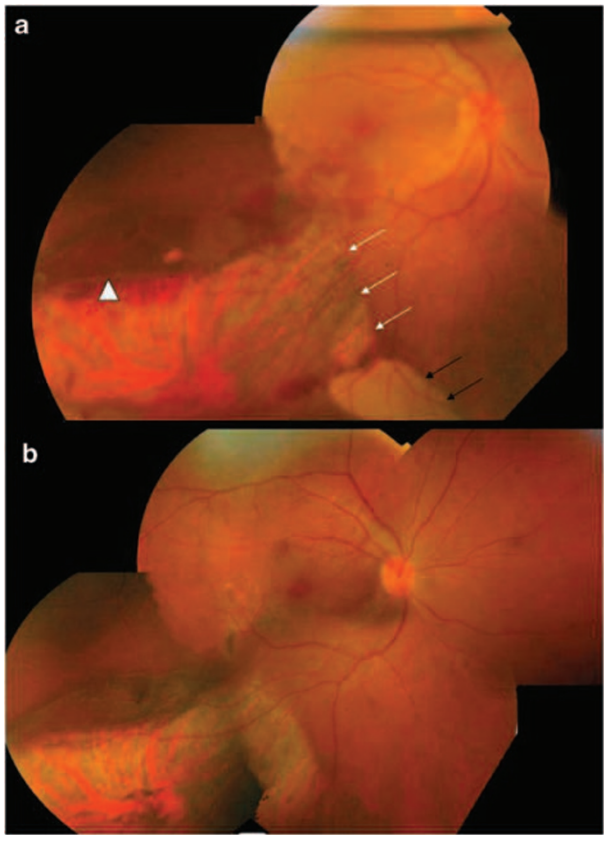

Figure 1 Fundus photographs of a patient with polypoidal choroidal vasculopathy in the right eye. (a) On day 14 after vitrectomy, fundus photograph of the right eye showed a giant retinal pigment epithelial (RPE) rip (white arrows) with rolling edge (arrowhead) and subretinal haemorrhage (black arrows). (b) Six months later, fundus photograph showed a persistent RPE rip, resolution of submacular haemorrhage, and residual subretinal haemorrhage adjacent to the RPE rip.

Comment

Large, thick subretinal haemorrhage and/or sub-RPE haemorrhage caused by PCV often result in a poor visual outcome, especially when the haemorrhage is massive and extends to the periphery.[1,2] In eyes with PCV, RPE tears can occur at the margin of serosanguineous pigment epithelial detachments—either spontaneously or after photodynamic therapy.[2] However, a giant RPE tear is uncommon and may cause serous and/or haemorrhagic subretinal detachment in such patients.

In our case, the visual field was initially affected by sub-RPE and/or subretinal haemorrhages; subsequently the central visual acuity was seriously impaired due to vitreous haemorrhage 1–2 weeks later. The occurrence of a giant RPE rip was unusual, given the huge pigment epithelial detachment, the subpigment epithelial haemorrhage, and choroidal neovascularization (PCV), exerting tangential as well as oblique subpigment epithelial haemorrhage. The surgical intervention certainly could add to the anterior-posterior and mechanical forces associated with the giant rip.

Spontaneous healing of RPE rip is proposed to occur by several mechanisms, including a layer of hypopigmented

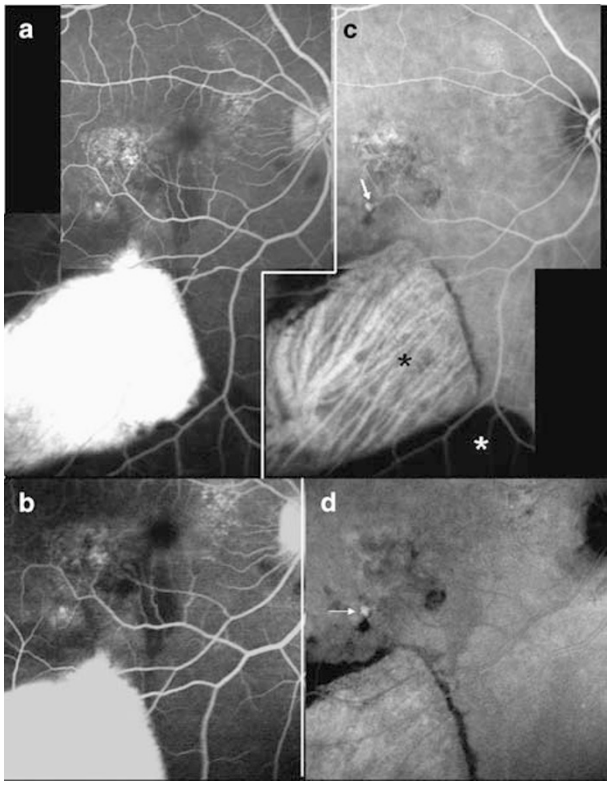

Figure 2 Fluorescein and indocyanine green angiograms of the eye with polypoidal choroidal vasculopathy. (a and b) Early and late phases of fundus fluorescein angiogram reveal an occult choroidal neovascularization and a giant retinal pigment epithelial (RPE) rip. Pooling of dye is seen in the area of RPE rip. (c and d) Fundus indocyanine green angiogram shows a hyperflourescent polypoidal lesion (arrowhead) inferotemporal to fovea, a hyperfluorescent area (black asterisk) over the giant RPE rip, and a hypofluorescent area (white asterisk) of subretinal haemorrhage.

RPE cells, atrophy of choriocapillaris, or deposition of fibrous tissue;[3] however, it is not presented in this case. Although massive subretinal haemorrhage with a giant RPE tear could cause a poor visual outcome in patients with PCV, the patient preserved a good final visual outcome.

Conflict of interest
The authors declare no conflict of interest.

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Sir,

Spectral-domain optical coherence tomography visualisation of retinal oxalosis in primary hyperoxaluria

Type 1 primary hyperoxaluria[1] is an autosomal recessive disorder caused by a deficiency of a liver enzyme (alanine-glyoxylate aminotransferase).[2] Eye involvement in primary hyperoxaluria consists of calcium oxalate deposits at the level of the retinal pigment epithelium (RPE) with surrounding areas of hypertrophic and hyperplastic RPE.[1,3] Some authors reported on unusual intraretinal distribution of oxalate deposits, apparently sparing the RPE (studies carried out on ocular tissues obtained at autopsy).[4,5]

Here, we describe the *in vivo* spectral-domain optical coherence tomography (Spectralis SD-OCT, Heidelberg Engineering, Heidelberg, Germany) findings in a patient with retinal oxalate deposits (retinal oxalosis) due to primary hyperoxaluria.

Case report
A 19-year-old man diagnosed with type 1 primary hyperoxaluria was referred to our Department. He underwent combined cadaveric liver and kidney transplantation 15 years before. The patient signed a comprehensive consent form according to good clinical practice guidelines, before proceeding with any examinations. His best-corrected visual acuity was 6/24 in the right eye and 20/38 in the left eye. On fundus biomicroscopy, both the eyes showed a fibro-atrophic lesion within the macular area, as well as retinal crystalline deposits, most of which appeared centred by patches of pigment (Figure 1); these deposits were widely distributed in the fundus, and appeared interposed between the RPE and the neurosensory retina. Fundus autofluorescence (AF) showed hyper-autofluorescent dots and ring-shaped areas of hyper-autofluorescence with central hypo-autofluorescence associated with crystal deposition (Figure 1). Interestingly, both fluorescein angiography (FA) (Figure 1) and indocyanine green angiography (Figure 1) revealed almost the same fluorescence features associated with crystal deposition, as the one seen on AF frames (hyper-fluorescent dots and ring-shaped areas of hyper-fluorescence with central hypo-fluorescence). Spectralis SD-OCT clearly showed the oxalate deposits as tiny hyper-reflective lesions localised within areas of dome-shaped elevated RPE (Figure 2).