right eye showed a lamellar macular hole with a longest basal diameter of 1280 μm, without presence of vitreoretinal abnormalities (Figure 1a and b). In the left eye a lamellar macular hole was detectable with a longest basal diameter of 810 μm. A preretinal membrane extending through the posterior pole and a hyaloidal adherence at one edge of the foveal pit were associated to the hole. Coronal C-scans showed the lateral extent of the hole and the preretinal membrane. The latter was recognizable as a hyper-reflective wrinkling of the retinal surface (Figure 1c–f).

Case 2 was a 69-year-old man with a history of bilateral high myopia and neovascularization in the right eye. He presented a spherical equivalent refractive error of −7 D OD and −19 D OS, a best-corrected visual acuity of 20/200 OD, 20/50 OS. Fundus examination of the right eye disclosed a macular disciform scar, whereas in the left eye a macular hole was present. En face OCT examination of the left eye showed a lamellar macular hole with a diameter of 770 μm, associated with posterior retinal detachment (Figure 1g–i).

Comment

Macular holes in highly myopic eyes may be associated with a rhegmatogenous retinal detachment surrounding the hole.[5] Although ocular elongation and retinal thinning may contribute to macular hole formation, retinal detachment may be promoted by the weak attachment between the neurosensory retina and the retinal pigment epithelium in the posterior staphyloma. Because in highly myopic eyes the retina, pigment epithelial cells, or both are atrophic, it is usually difficult to detect macular holes with biomicroscopy. In both evaluated cases en face OCT has provided good quality visualization of both the cross-sectional and lateral extension of the hole and the associated posterior retinal detachment and preretinal membrane. The en face OCT software has allowed the fine measurement of the hole basal width.

Overlay of coronal scans on red-free fundus images associated with the possibility to measure the diameter of the hole allow a prompt evaluation of the extent of these abnormalities, representing a noninvasive alternative for the follow-up of their changes.

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Sir,

Bilateral serous retinal detachment as the first manifestation of paroxysmal nocturnal haemoglobinuria

Paroxysmal nocturnal haemoglobinura (PNH) is an acquired clonal stem cell disorder resulting from a somatic mutation in the haematopoietic stem cell. It is characterized by intravascular haemolysis, cytopenia, frequent infections, bone marrow hypoplasia, and high incidence of life-threatening venous thrombosis.[1,2] There is a report on ocular manifestations of PNH where bilateral papilloedema was described.[3]

Case report

A 43-year-old man who had experienced blurred vision for 1 week visited our clinic. He had no known systemic disease but his general looking appearance was pale. Best-corrected visual acuity (BCVA) was 20/400 in the right eye, 20/200 in the left eye, and both anterior segments were normal on biomicroscopy. Funduscopy and optical coherence tomography (OCT) revealed

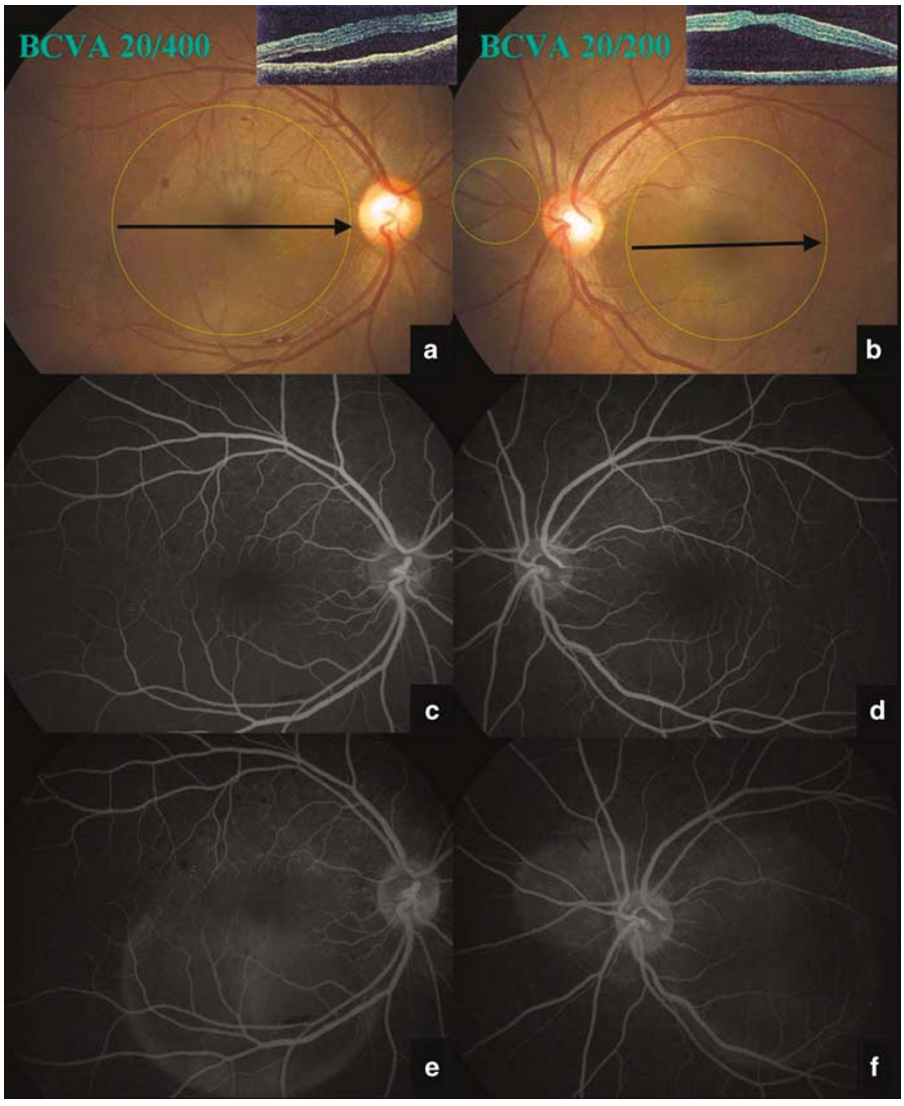

Figure 1 Fundus photographs and macular OCTs (a, b) and early (c, d), and late phase (e, f) of fluorescein angiography of the patient. In fundus photographs, there were bilateral subretinal serous fluid at the posterior poles with some haemorrhagic dots at parafoveal area and macular OCTs also revealed bilateral serous retinal detachments (a, b). Fluorescein angiography demonstrated bilateral multiple choroidal leaking point at early phase (c, d) and subretinal accumulation of fluorescein involving fovea at late phase (e, f).

bilateral serous retinal detachment with some haemorrhagic dots at posterior pole (Figure 1a, b). Fluorescein angiography showed bilateral multiple pin-pointed choroidal leaking with the pooling of dye at posterior pole (Figure 1c–f). Laboratory examination revealed leucocytosis, anaemia, thrombocytopenia, and reticulocytosis. He was referred to a haematologist. Coombs test was negative and bone marrow biopsy showed moderate granulocytic hypoplasia; PNH flow cytometry was positive. The final diagnosis was PNH and steroid pulse therapy (1 g/day) was started with blood transfusion.

After 3 days of steroid therapy, his BCVA was 20/25 in the both eyes. The funduscopy, OCT, and fluorescein angiography showed resolution of detachment (Figure 2a, b). He was discharged with tapered dosage of steroid. During the 2-month period of follow-up, there has been no relapse of the subretinal fluid.

Comment

We may assume that the cause of serous retinal detachment is from the venous thrombosis that can occur in the choroidal circulation. Prolonged excessive stress on the retinal pigment epithelium (RPE) cells causes impaired function of RPE cells and fluid leakage from choriocapillaries, ultimately pooling fluid into the sub-RPE and subretinal space.[4]

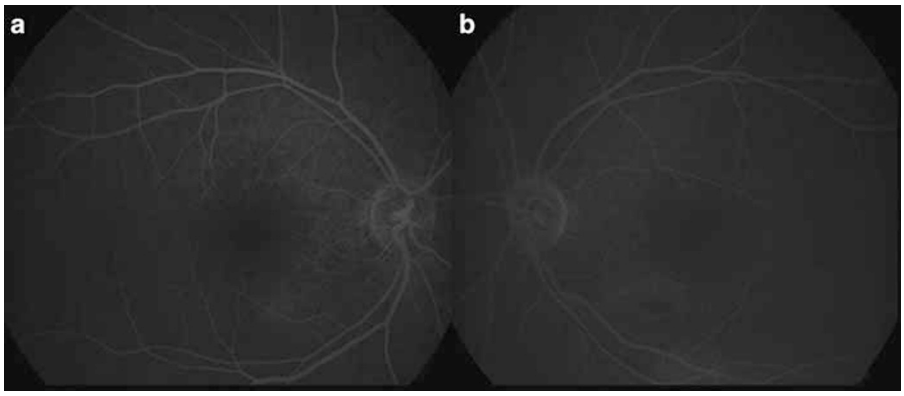

Figure 2 At 3 weeks after steroid pulse therapy, late-phase fluorescein angiography of the right eye (a) and the left eye (b) of the patient. Late-phase fluorescein angiography showed resolution of serous retinal detachment and fluorescein leakage (a, b).

This case is noteworthy for two reasons. First, the ophthalmologist can be the first medical contact, before systemic symptoms appears in PNH. Had the patient not been checked with laboratory examination in our case, other problems may have ensued. Second, this is the first report of PNH with bilateral serous retinal detachment.

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Sir,

Alcohol debridement for recurrent corneal erosions

We read with interest the paper by Ramamurthi *et al*[1] on recurrent corneal erosions. The authors should be congratulated for such a comprehensive review of this common condition. However, we feel that the use of alcohol debridement of the epithelium as an alternative treatment method should have been included.

Alcohol debridement has been mentioned in the literature since 2000.[2] Its use first gained popularity after it was used in LASEK. It was noted that alcohol debridement cleaved a smooth plane and was associated with faster visual rehabilitation and reduced postoperative haze.[3] Dua *et al*[4] started to use this technique for recurrent erosion after noticing that following alcohol debridement there was increased difficulty debriding the epithelium if repeat LASEK was required. In their study,[4] on the use of alcohol debridement for recurrent erosions, 75% had complete resolution of symptoms after 1 month of treatment.

We ourselves have used alcohol debridement as a treatment method in York since 2003 with comparable success rates. The technique we use is similar to that described by Dua *et al*.[4] We apply 20% alcohol in a corneal well for 60 s. The epithelium is then rinsed with balanced salt solution and the loose epithelium removed. We then insert a bandage contact lens for 1 week or until the epithelium is healed.

Mah, as quoted by Lipner,[5] felt that alcohol debridement does not present a valid alternative for recurrent erosions, feeling that it is more complicated and that the results are the same as those with mechanical epithelial debridement or microstromal puncture. However, we disagree. While we acknowledge that it is not a suitable treatment option for all patients, for recurrent erosion caused by localised trauma, in the absence of a dystrophy, it is a cheap, successful and