Figure 1 Left eye funduscopy showing localized retinal detachment with horseshoe retinal tear at 2 O'clock and an avulsed neovascular frond with ghosting of superotemporal blood vessels.

following BRVO are of two types—holes without traction and tears with vitreous traction with or without retinal neovascularisation.^{1–3} Tears and retinal detachment due to traction on neovascular tissue are infrequent.

A strong association between BRVO with vitreous haemorrhage and posterior tractional retinal breaks was reported by Joondeph *et al.*² They reviewed 358 cases with BRVO and reported 1.6% incidence of posterior tractional breaks leading to rhegmatogenous retinal detachment in 0.6% of patients.¹ Kir *et al.*⁴ reported 3% incidence of retinal breaks and 1.3% of rhegmatogenous retinal detachment.

Retinal breaks are almost exclusively found in the distribution of the occluded vessel and often located at or posterior to equator.⁴

The most widely accepted hypothesis for pathogenesis of tractional retinal break in BRVO is vitreoretinal traction caused by vitreous contraction following retinal neovascularisation.^{1–3} The influence of laser photocoagulation and/or vitreous haemorrhage resulting in retinal break formation remains controversial.⁵

Our case emphasises that vitreous haemorrhage in eyes with BRVO should alert the examiner to closely monitor the situation since traction on neovascular fronds may lead to retinal breaks and consequent retinal detachment.

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Sir, Bilateral acute retinal necrosis syndrome following herpes simplex type 1 encephalitis

Acute retinal necrosis syndrome (ARN) associated with herpes simplex viral encephalitis has been well described. Bilateral acute retinal necrosis (BARN) in this context is described but is extremely rare. We present a case of BARN following recent herpes simplex type 1 (HSV-1) encephalitis (HSE) in an immune competent patient.

Case report

A 59-year-old man presented to the ophthalmologist complaining of reduced visual acuity of 6/12 in his left eye. ARN was not recognised until he represented 2 weeks later with further decline in his left eye vision to hand movements and right eye vision reduced to 6/18. On recognition of BARN he was referred to our unit for further management. On arrival, he had bilateral florid, granulomatous uveitis and optic disc swelling. There was widespread bilateral retinal vasculitis and 360° confluent retinitis. In the left eye, the retinitis extended up to the disc nasally and there was widespread retinal detachment extending to the temporal arcades, Figure 1.

Figure 1 Colour fundus photograph showing retinal necrosis in the periphery of the left eye.

Figure 2 CT scan showing low attenuation in the left temporal lobe.

At 1 month previously, the patient had been admitted with malaise, fever, and speech disturbance. CT imaging showed a low attenuation area in the left temporal lobe, Figure 2 and HSV-1 was detected on polymerase chain reaction (PCR) testing of a cerebrospinal fluid sample. He was treated with intravenous aciclovir 10 mg/kg TDS for 2 weeks and oral corticosteroid.

On admission to our unit, he was restarted on intravenous aciclovir 10 mg/kg TDS. He underwent left eye vitrectomy, 360° laser retinopexy, and silicone oil tamponade. The right eye also received prophylactic 360° laser retinopexy. Both eyes received intravitreal foscarnet, 2.4 mg in 0.1 ml. The patient was maintained on intravenous aciclovir for 14 days. Oral prednisolone was introduced after 48 h of intravenous aciclovir for his severe intraocular inflammation. PCR studies of the left vitreous sample were positive for HSV-1 and negative for other herpetic viruses. Lymphocyte surface markers, immunoglobulins and HIV testing were all normal or negative.

The patient was discharged on oral valaciclovir 1 g TDS in addition to a reducing dose of oral steroid. After 2 weeks, the retinal detachment in the left eye recurred in spite of silicone oil tamponade and he also developed a temporal, peripheral retinal detachment in the right eye. At 4 months, the retinal detachments are limited to the anterior edge of the laser barricade and do not extend across the temporal arcades in either eye. His vision is unchanged from discharge at 6/5 right eye, CFs left eye.

We plan to continue valaciclovir for at least 6 months.

Comment

ARN is a rapidly progressive retinal infection caused by herpes viruses. It usually occurs in immunocompetent patients. Although most cases are unilateral, bilateral disease has been reported in up to 35% of patients.¹ This figure is probably determined by early recognition and treatment of first eye involvement.² Acute retinal necrosis is a clinical diagnosis and standard diagnostic criteria have been published by Holland *et al.*³ VZV and HSV-1 are associated with acute retinal necrosis in the older population while HSV-2 is the more common pathogen in under 25-year olds.⁴

We have found 20 cases of unilateral ARN associated with HSE in the absence of human immune deficiency virus in the literature in the last 20 years.^{1,4-16} Pepose *et al.*¹⁵ reports a case of HSE 3 weeks following ARN. All others report ARN following HSE with variable time courses (mean of 6/12 years, median 5 years, range 10 days–20 years). BARN following HSE could only be found in seven case reports.¹⁷⁻²² The majority of patients in the unilateral ARN group had no other factors which

might be associated with impaired host defence. In contrast, the reported cases of BARN following encephalitis all had some associated factors that may have reduced host defence. In two of the seven case reports, the initial encephalitis was either not recognised or there was a significant delay in treatment.¹⁷ Two report association with high-dose intrathecal or pulsed intravenous steroid.^{18,19} One case occurred during iatrogenic immunosuppression with chemotherapy agents²⁰ and a further case followed craniotomy and oral dexamethasone for a supra sellar lesion.²¹ Finally, there was one case of HSV-2 in a neonate.²² In our case, the only possible compromising factor was administration of dexamethasone over a 6-day period. However, this was given in addition to aciclovir and might be regarded as a standard part of his encephalitis therapy. In this respect, our patient is unique to others reported in the literature with BARN following HSE.

Prompt treatment of HSE is essential as there is a high mortality and morbidity. Early treatment may be important for limiting reactivation or neuronal translocation that could otherwise lead to further virus spread to other CNS sites including the eye. Standard antiviral treatment regimens do not usually extend beyond 14 days.²³ As ARN is potentially blinding with a retinal detachment rate of between 50 and 75%,²⁴ we question this short-term approach. In general, BARN tends to occur quite soon following encephalitis with most reported within 5 months and only one reported after 42 months.¹⁸ We suggest that oral antiviral agents should be introduced after IV therapy for at least 6 months following HSE to reduce the risk of acute retinal necrosis. Oral valaciclovir, a prodrug of aciclovir has excellent bioavailability and is very well tolerated.²⁵ Patients with HSV encephalitis should be examined carefully for ocular disease. Patients who have associated factors that may affect host defence should be viewed with a high index of suspicion. Physicians should request immediate specialist review of any patient with HSE developing signs or symptoms of ocular inflammation. Thorough peripheral retinal examination is particularly advised for any patient presenting with uveitis and a previous history of HSE.

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Sir,
**Massive haemorrhagic retinal detachment after
transpupillary thermotherapy for choroidal
neovascularisation**

Transpupillary thermotherapy (TTT) has been successfully used for treatment of subfoveal choroidal neovascularisation (CNV).¹ Safety however remains a concern, given its narrow therapeutic window.² We report a haemorrhagic retinal detachment (HRD) complicating TTT for CNV.

Case report

A 75-year-old diabetic, hypertensive, and cardiac man complained of poor vision OD for 2 months. Best-

corrected visual acuity was 6/18 OD, and 6/6 OS. Anterior segment OU was unremarkable except bilateral pseudophakia. Fundus examination OD showed a large CNV with serous macular detachment and subretinal haemorrhage (Figure 1a). There was no evidence of age-related macular degeneration (AMD), or polypoidal choroidal vasculopathy (PCV). The left fundus was normal. Fundus fluorescein angiogram OD showed a predominantly occult CNV (Figure 1b). Indocyanine-green angiography ruled out PCV.

The options of observation, PDT or TTT were offered. With patient's informed consent and approval of the institutional review board, TTT was performed with a slit-lamp mounted 810-nm diode laser. Threshold power was determined with an inferonasal test spot. The lesion was treated with five 500–600 mW spots, each 3 mm, lasting 1 min. Power was reduced by 10% over the haemorrhage; increased similarly over serous detachment. No retinal blanching occurred.

The patient's vision suddenly dropped to hand motions 10 days post-TTT. Fundus examination revealed a massive posterior HRD (Figure 1c). Haematological investigations, cardiac, and carotid status were normal; he was not on anticoagulants. B-scan ultrasonography confirmed clinical findings; there was no suprachoroidal haemorrhage. After 6 weeks, vision remained poor; fundus was not visible. Ultrasound revealed breakthrough vitreous haemorrhage, with reduced height of HRD (Figure 1d). The patient did not follow-up subsequently. When contacted a year later, he reported no change in visual status.

Comment

AMD-related CNV may rarely bleed spontaneously at the stage of disciform scar, predisposed by systemic anticoagulants, hypertension, and cardiovascular disease.³ Idiopathic CNV has a better prognosis; but older patients and larger lesions fare worse, irrespective of the baseline acuity.⁴ In our patient, HRD was probably caused by TTT-induced vasodilatation, and subsequent closure,² resulting in dehiscence of choroidal vessels, weakened by age, hypertension, atherosclerosis, and diabetes. The use of multiple burns might also have contributed to HRD, by excessive thermal damage at the overlapping edges. However, we did not observe any visible retinal whitening, popping sound, or patient discomfort during or after TTT. Most patients with occult CNV have lesions larger than two disc areas,⁵ and therefore require either multiple spots,⁶ or large-spot TTT.^{7,8} There is no evidence in the literature of the superiority of one technique over the other. Similar to