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Sir,

Retinal neovascularisation and posterior penetrating ocular injury

Penetrating ocular injuries may induce a variety of haemorrhagic, inflammatory and structural sequelae, including proliferative vitreoretinopathy, sympathetic ophthalmitis, retinal detachment and intraocular haemorrhage. Despite an environment favouring the development of reparative fibrovascularisation, retinal neovascularisation following retinal detachment or trauma is uncommon. We present a case of unilateral peripheral retinal neovascularisation developing after posterior penetrating ocular trauma in the absence of other identifiable vasculogenic stimuli.

Case report

A 21-year-old white man sustained a penetrating injury of the right eye from a hand-held 7 cm spike whilst repairing a car. He underwent primary repair of a 4 mm circumferential nasal limbal wound with repositioning of prolapsed iris. There was no retained intraocular foreign body. Post-operative examination revealed a nasal retinal impact site with attached gel and a localised area of vitreous incarceration to the nasal limbal wound, although the retina was otherwise normal. The eye made an uneventful recovery, achieving 6/5 unaided vision.

Three months following the injury, peripheral retinal neovascularisation was identified in the right eye at the posterior border of the vitreous base. Fluorescein angiography revealed peripheral non-perfusion throughout 360° of the right eye with scattered peripheral sea-fan neovascularisation in all quadrants, but no vascular sheathing or features of vasculitis (Fig. 1). The left eye was normal to clinical and angiographic examination. Blood pressure, digital ophthalmodynamometry, full blood count, ESR, serum electrolytes, glucose, ACE, anticardiolipin antibody, haemoglobin electrophoresis, immunological tests, and investigations of the anticoagulant and fibrinolytic system were normal, although protein S was raised to 160% (normal range, 70–140%). The clinical appearance is unchanged at 3 years' follow-up.

Comment

Retinal neovascularisation develops in response to multiple extracellular modulating and cellular factors acting on endothelial cells at discontinuities in the basement membrane.[1] Such factors promote peripheral retinal neovascularisation in a variety of ischaemic and

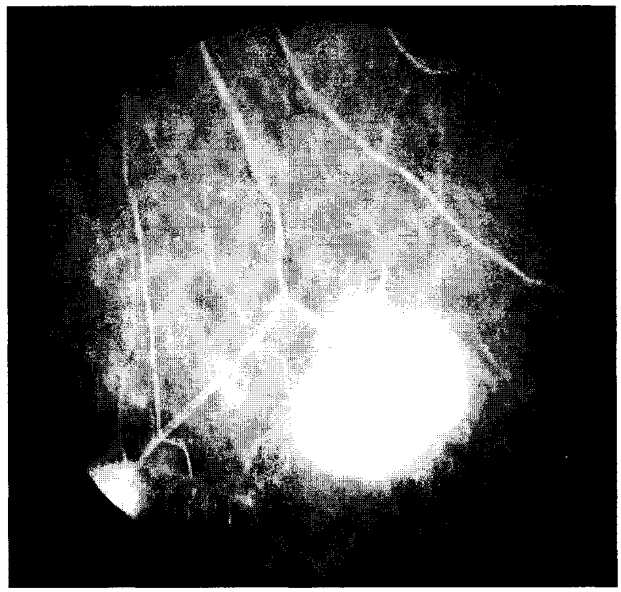

Fig. 1. *Fluorescein angiogram demonstrating peripheral non-perfusion and neovascularisation 'sea-fans'.*

inflammatory disorders including sickle cell retinopathy, Eales' disease, Behçet's disease, diabetes, talc retinopathy, retinopathy of prematurity, dominant exudative vitreoretinopathy and, occasionally, retinal detachment. However, the above diagnoses were felt to be unlikely. Caucasian race and normal haemoglobin electrophoresis excluded sickle cell retinopathy; unilateral findings and absence of evidence for drug abuse argued against talc retinopathy; unilateral findings, an absent family history and a normal birth weight made retinopathy of prematurity and familial exudative vitreoretinopathy unlikely; and normal blood sugar, blood pressure and digital ophthalmodynamometry did not support other possible causes of peripheral neovascularisation.

There was no evidence to suggest that systemic vascular or coagulation abnormalities played a role in the development of retinal neovascularisation, and the only abnormal finding was a slightly raised protein S level. This is unlikely to be significant, because, although low protein S levels can be associated with retinal vascular thrombosis,[2] there is no reported association between thrombosis and high protein S.

We may be witnessing the development of an inflammatory disorder such as Eales' disease. However, the combination of lack of progression during 3 years of observation, absence of bilateral findings and lack of inflammatory signs such as vitreous cells or retinal vascular sheathing, is very atypical.

The unilateral development of retinal neovascularisation within 3 months of a penetrating ocular injury raises the possibility that trauma played an aetiological role. To the best of our knowledge this development has not previously been reported, although it is not unprecedented for retinal neovascularisation to develop in extensive retinal detachment and proliferative vitreoretinopathy.[3,4] Indeed, it is surprising that neovascularisation is not more commonly encountered in proliferative vitreoretinopathy (PVR) associated with penetrating ocular trauma, given the presence of similar growth factors in ocular neovascularisation and PVR.[5] It is possible that the inflammatory and wound healing response, in combination with vitreous traction at the impact site in this case, influenced the balance of angiogenic factors in favour of neovascularisation in this eye. In addition, 360° retinal traction from the posterior vitreous insertion may have contributed to retinal ischaemia, although, conversely, vitreous traction may have resulted from retinal ischaemia inducing a fibrovascular response.

The unexpected development of unilateral peripheral retinal neovascularisation following posterior segment penetration emphasises the need for peripheral retinal examination in the follow-up of such patients. The exact pathogenesis of unilateral peripheral retinal neovascularisation in this case remains debatable. However, the chronological sequence and the absence of an identifiable cause despite exhaustive investigations and prolonged follow-up implicate a stimulatory role for posterior penetrating ocular trauma.

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Sir,

Allergic contact dermatitis reactions to corticosteroids in periorbital inflammation and conjunctivitis

Delayed hypersensitivity to corticosteroids in topical preparations is increasingly recognised by dermatologists as a cause of contact dermatitis.

Allergic contact dermatitis is diagnosed by patch testing, an investigative tool first described in 1896[1] and today performed according to a standard protocol.[2] In our department more than 1000 patients are patch tested annually. We have found that nearly 6% of patients have a relevant, allergic reaction to topical corticosteroids,[3] most commonly tixocortol-21-pivalate (a marker of hydrocortisone allergy[4]), hydrocortisone butyrate or budesonide.

Over a period of 18 months we have investigated 40 patients with persistent periorbital inflammation. In 3 of these patch tests to corticosteroid preparations were positive and subsequent avoidance has been associated with resolution.

Case reports

Case 1 A 52-year-old man with no previous history of skin disease presented a 1-month history of painful red eye secondary to trauma. A diagnosis of conjunctivitis was made and when this failed to respond to either chloramphenicol or Fucidin (Leo) drops from his general practitioner, he was referred to an ophthalmologist.