

Sir,

Alkali injury complicated by choroidal neovascularisation

Choroidal neovascularisation occurs in association with age-related macular degeneration, myopia, angioid streaks, ocular histoplasmosis and conditions associated with disruption of Bruch's membrane. It can occur without any associated ocular abnormality. Severe central visual loss occurs due to foveal involvement and macular scarring. The pathogenesis of choroidal neovascularisation is poorly understood. We report a case of an alkali injury which was complicated by the development of choroidal neovascularisation.

Case report

A 47-year-old Caucasian man presented to the Accident and Emergency department of the Birmingham and Midland Eye Centre. He had accidentally sprayed bleach to both eyes 3 hours previously. He had performed ocular irrigation immediately following the injury. He had previously had excellent visual acuity and was a low myope; his refraction was -2.50 DS in the right eye and -1.75 DS in the left eye. There was no history of previous ocular trauma. He was otherwise healthy. He had no history of foreign travel. There was no family history of ocular disease.

Immediate ocular irrigation was performed on arrival. The pH measured 8.00 in both eyes. Ocular examination revealed best corrected visual acuity was hand motions (HM) in the right eye and 6/18 in the left eye. Slit-lamp examination of the right eye revealed there was extensive epithelial loss affecting the cornea and conjunctiva, localised corneal oedema and limbal ischaemia extending for 240°, manifest by vascular occlusion. In the left eye there was mild corneal and conjunctival epithelial loss but no evidence of limbal ischaemia. Few fundal details could be discerned due to the anterior segment injuries. He was admitted for intensive treatment with topical corticosteroids, antibiotics, mydriatics, topical and systemic ascorbic acid.

Over the following 4 weeks, his visual acuity in the right eye failed to improve beyond HM and improved to 6/6 in the left eye. The anterior segment injuries healed with the exception of symblepharon formation in the upper and lower fornices of the right eye (Fig. 1). Fundoscopy revealed a neurosensory detachment overlying the fovea, with retinal pigment epithelial changes and a grey subretinal lesion (Fig. 2a). There was no evidence of peripheral chorioretinal scarring, no

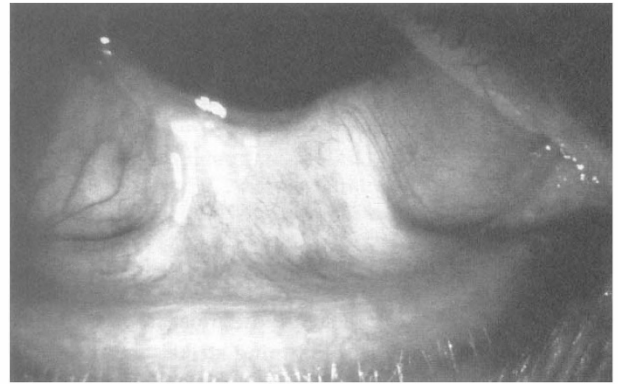


Fig. 1. Slit-lamp photograph of the right eye demonstrating conjunctival scarring and symblepharon formation inferiorly.

histoplasmosis spots and no peripapillary chorioretinal atrophy. Fluorescein angiography confirmed the presence of subfoveal classic choroidal neovascularisation (Fig. 2b, c). Due to the subfoveal location, it was judged that laser photocoagulation would not be of any benefit.

His acuity in the right eye has remained unchanged and he uses topical ocular lubricants to the right eye. His left eye is comfortable and normal.

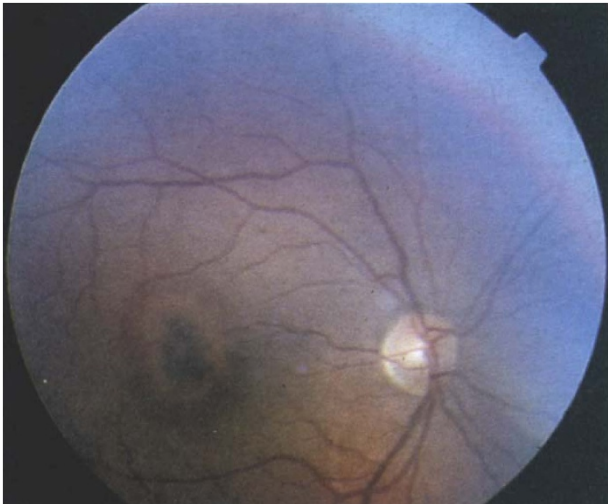
Comment

Choroidal neovascularisation is a devastating cause of central visual loss. Ninety per cent of all patients with severe visual loss due to age-related macular degeneration have choroidal neovascularisation. The pathogenesis of choroidal neovascularisation is, however, poorly understood. Alteration to Bruch's membrane plays a key role in the development of choroidal neovascularisation.¹

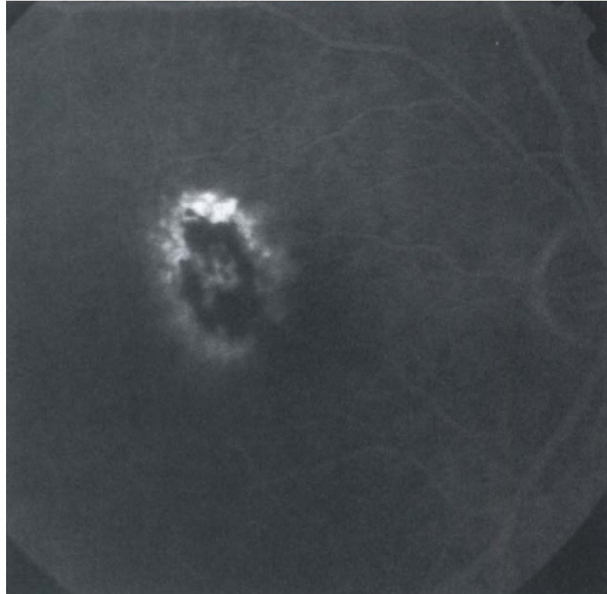
Multiple factors are required to induce choroidal neovascularisation. Factors are needed to cause degradation of the extracellular matrix and to stimulate growth of endothelial cells into blood vessels. Angiogenic factors necessary for the development of neovascularisation include vascular endothelial growth factor (VEGF) and fibroblast growth factor (FGF).²

The retinal pigment epithelium has been shown *in vivo* to produce angiogenic factors such as FGF and VEGF.³ Retinal pigment epithelium can be activated by a number of stimuli including hypoxia and laser photocoagulation. It has been shown that laser photocoagulation (associated with the development of choroidal neovascularisation) causes release of VEGF and FGF.⁴ It is not known why some eyes which undergo laser photocoagulation develop choroidal neovascularisation and others do not. Ischaemia may play a role, altering the balance between angiogenesis factors and angiogenesis inhibitors.

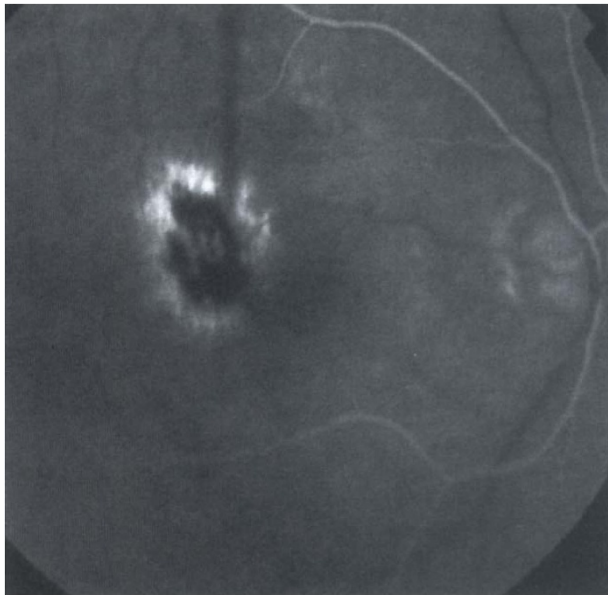
We report a patient who developed choroidal neovascularisation in association with an alkali injury. Alkali injuries cause ocular ischaemia. We believe that choroidal neovascularisation developed as a result of the



(a)



(c)



(b)

Fig. 2. (a) Fundus photograph of the right eye demonstrates a grey subretinal lesion beneath the fovea with surrounding retinal pigment epithelial atrophy. (b) Fluorescein angiogram of the right eye in the early phase (20.8 s) shows early lacy hyperfluorescence, representing an area of classic subfoveal choroidal neovascularisation. (c) The late-phase angiogram (3.06 min) reveals progressive leakage and a surrounding area of retinal pigment epithelial atrophy.

ocular injury and was possibly precipitated by the associated ocular ischaemia. Ischaemia may play a role in the development of choroidal neovascularisation.

References

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Sir,

Permanent corneal limbal stem cell dysfunction following radiotherapy for orbital lymphoma

We present a patient with unilateral stem cell deficiency following the administration of 4600 cGy in 21 fractions for a high-grade malignant B cell lymphoma of the orbit. The stem cell deficiency occurred without acute manifestations of radiation-induced toxicity to the cornea