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.

Prednisolone plus neomycin drops were prescribed and after initial benefit these were associated with marked periorbital oedema and inflammation. When these signs persisted and betamethasone valerate drops (0.1%) were ineffective, contact allergy was suspected and the patient was referred for patch testing. This revealed delayed hypersensitivity to betamethasone valerate and neomycin (Table 1). Avoidance of these medicaments and the use of topical clobetasone butyrate 0.05% twice daily to eyelid skin was associated with rapid recovery.

Case 2 A 52-year-old woman who had suffered eczema as a child and had a contact allergy to perfume demonstrated 7 years previously, presented with a 2 year history of periorbital eczema and allergic conjunctivitis despite fragrance avoidance. This failed to respond to hydrocortisone 0.1% ointment. She had developed a mild eczematous eruption on the hands 3 years previously for which she had also used hydrocortisone 0.1% ointment. Patch testing confirmed fragrance allergy and demonstrated allergy to hydrocortisone (Table 1), subsequent avoidance of which has been associated with resolution of the eye problem.

Case 3 A 43-year-old woman with 13 years of persistent hand eczema treated variously with either hydrocortisone 0.1%, hydrocortisone butyrate 0.1%, clobetasol 0.05% or alclometasone 0.05%, gave a 6-month history of periorbital inflammation and oedema which had led her to discontinue the use of soft contact lenses. This eruption was exacerbated by topical hydrocortisone and patch testing demonstrated allergy to hydrocortisone and hydrocortisone butyrate (Table 1).

Relevant positive hypersensitivity reactions were noted to the constituents of eye medicaments in 18 of the remaining 37 patients. These were to anti-microbials in 10 patients (aminoglycosides, chloramphenicol, polymixin B and sodium fusidate), preservatives in 7 patients (phenylmercuric acetate and thiomersal) and antiseptics in 4 patients (chlorhexidine digluconate and benzalkonium chloride). No contact sensitivity to eye medicaments or cosmetics was demonstrable in 19 patients.

Table 1. *Results of patch testing in 3 patients*

	Case 1	Case 2	Case 3
Tixocortol-21-pivalate[a]		++	+
Hydrocortisone butyrate			++
Betamethasone valerate	++		
Neomycin	++		
Balsam of Peru[b]		++	
Predsol N	++		

[a]Tixocortol-21-pivalate is a marker of hydrocortisone allergy.
[b]Balsam of Peru is a marker of fragrance allergy.

Discussion

Allergic contact dermatitis must be considered in those patients with periorbital inflammation who fail to improve or deteriorate following treatment. Both the active ingredients and the excipients of topical medicaments can produce delayed hypersensitivity reactions. Patch testing should be undertaken to investigate all relevant, potential allergens including the components of any ophthalmological or dermatological corticosteroid preparations used by the patient.

In our first case there was no record of previous corticosteroid use and we believe the patient was sensitised through contact with eye drops containing betamethasone valerate and neomycin prescribed for conjunctivitis. In cases 2 and 3 the persisting inflammation was predominantly in the eyelid skin. It is probable that the corticosteroid sensitisation resulted from hand–eye transfer of the preparations used for hand dermatitis.[5]

Patch testing uncovered relevant contact sensitivities allowing us to give avoidance advice which resulted in complete resolution of the periorbital problems in all 3 patients. We feel that ophthalmologists should be aware of the potential for allergy to corticosteroids and the effectiveness of patch testing in differentiating sensitivities. Delayed hypersensitivity thus demonstrated may have important implications for subsequent systemic steroid therapy. Systemic hydrocortisone has been shown to induce cutaneous reactions at the site of allergic contact dermatitis[6] as well as more generalised erythema.[7]

We thank Mr B. Leatherbarrow, consultant ophthalmologist, and Dr N. Telfer, consultant dermatological surgeon.

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