uveitis is most likely because of an immune complex hypersensitivity reaction, and not because of any specific toxicity to the eye.[3] It could possibly be related to the individual's previous exposure to streptococcal antigens. Streptokinase may also be associated with other immunological reactions such as serum sickness[4] and Guillain–Barre syndrome.[5]

Apart from streptokinase, this gentleman also had diamorphine and cyclizine as part of his immediate medical treatment. Ocular side effects of these drugs include miosis for diamorphine and nonspecific blurred vision for cyclizine. However, anterior uveitis is not a recognised or reported side effect of either of these drugs. Hence, streptokinase was thought to be the most likely culprit in this case. Other causes of acute bilateral hypopyon include Behcet's disease, HLA B-27 positive status and endogenous endophthalmitis.

The widespread use of streptokinase as a thrombolytic agent could lead to an increased incidence of this immunological phenomenon. Therefore, it is important for ophthalmologists to recognise this unusual ocular hypersensitivity reaction, so that it will be managed appropriately and any unnecessarily invasive management such as the use of intravitreal or intracameral antibiotics will be avoided.

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Sir,

Isolated conjunctival neurofibromas at the puncta, an unusual cause of epiphora

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We report an unusual case of epiphora caused by mechanical obstruction of the puncta by isolated neurofibromas.

Case report

A 45-year-old lady, originally from Surinam, presented with a 3 year history of epiphora and irritation of both eyes. There was no other relevant past medical or family history. Visual acuity was normal. External examination revealed 'hypertrophy' of the upper and lower puncta (Figure 1) of both eyes. Peripheral vascularization of the cornea and hyperaemia of the conjunctiva were also noted. Excision biopsy of the flesh-coloured punctal lesions was performed. Histology revealed that the lesions were neurofibromas. There were no other ocular features of neurofibromatosis.

A thorough systemic examination including a neurology review with MRI revealed no other stigmata of neurofibromatosis and there was no family history of neurofibromatosis. An outpatient review 2 months later revealed resolution of her symptoms of epiphora.

Comment

Neurofibromas are benign peripheral nerve sheath tumours characterized by a combined proliferation of Schwann cells, endoneural fibroblasts, and axons. They are usually associated with systemic neurofibromatosis, but can occur as isolated lesions.[1]

Three types of neurofibroma occur in the orbit: solitary, diffuse, or plexiform (the latter is considered pathognomonic of neurofibromatosis). Ocular involvement may include the eyebrow, eyelids, conjunctiva, iris, choroid, optic nerve, and orbit.

Three features of this case are unusual. Firstly, isolated conjunctival neurofibromas are rare and have not been previously described at the puncta causing epiphora. Secondly, neurofibromas are usually associated with

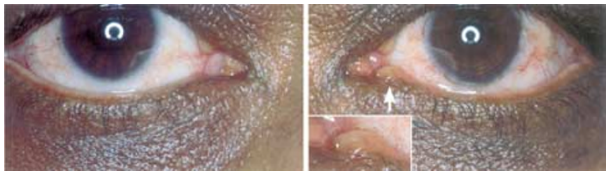

Figure 1 Hypertrophy of the upper and lower puncta of both eyes.

systemic neurofibromatosis. A thorough systemic examination in this patient showed no signs of neurofibromatosis. Thirdly, patients with these lesions often present at an earlier age than our patient in the second or third decade of life.

Kalina *et al*[2] reviewed the literature for isolated conjunctival neurofibromas and documented a detailed description of 13 cases in the literature including four from their own institution. Of the 13 cases, 10 had systemic neurofibromatosis. The lesions were located at the limbus in over half of the cases, and the remainder were located on the upper tarsal conjunctiva and on the temporal bulbar conjunctiva. At this location, they can be mistaken for a dermoid. None were documented at the puncta. Simple excision was curative in these cases, similar to ours, as the growth of these lesions is characteristically uniformly slow.

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Sir,

Central serous retinopathy masquerading as sympathetic ophthalmia

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Idiopathic central serous choroido-retinopathy (CSR) is a well-established clinical entity known to occur in type A personality individuals. Systemic corticosteroid therapy has also been recognised as a triggering factor.[1,2] We report a rare case of CSR developing in the fellow eye following enucleation and systemic corticosteroid therapy in a patient who had sustained a posterior globe rupture following a road traffic accident.

Case report

A 34-year-old male artist presented with a post-traumatic posterior globe rupture and optic nerve sheath haematoma of the left eye with multiple ipsilateral orbital fractures that were confirmed on ultrasonography and computerised tomography. The right eye was normal except for pre-existing myopia and no evidence of any macular pathology. He was treated with tetanus toxoid injection, intravenous antibiotics, analgesics, and oral prednisolone (1 mg/kg/day). His left eye was enucleated as a primary procedure after 6 days. The postoperative period was uneventful and the patient was discharged on oral prednisolone (1 mg/kg). Right eye vision remained normal with glasses. On the 15th postoperative day, the patient reported a marked decrease in vision of his right eye. The best-corrected visual acuity (BCVA) was 3/60 OD.

Biomicroscopic examination revealed mild retrolental flare with 1–2 cells with serous retinal detachment involving the macula. Humphrey perimetric examination (30-2) of the visual field showed a central scotoma. Fluorescein angiography (FA) showed a smoke stack leak typical of CSR (Figure 1). A provisional diagnosis of steroid- and stress-induced CSR was made. Gradual tapering of oral steroids was undertaken. Topical ketorolac (0.3%) and cycloplegic (atropine 1%) eye drops were prescribed under careful supervision. The retrolental flare and cells resolved within 24 h of topical treatment. A rapid improvement in visual acuity was noted over the next 4–5 days, and the patient was discharged with BCVA 6/18 OD. Visual fields repeated after 2 weeks demonstrated a decrease in the size of the scotoma. At 3 months follow-up, BCVA was 6/6 OD, and visual field defect had resolved. Serous retinal detachment had settled completely. FA showed only a small window defect (Figure 2).

Comment

This case represents the occurrence of CSR following high-dose systemic steroid therapy following a severe eye trauma. Central serous choroidoretinopathy is attributed to the disruption of the ionic pump of the RPE cells or owing to hyperpermeability of the choroidal vasculature. Glucocorticoids cause CSR probably because of increasing cAMP of RPE cells, and hence changing the