this condition, and whether it may be possible to predict those at a greater risk.

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This study has been approved by The Central and South Bristol Research Ethics Committee, United Bristol Healthcare NHS Trust, and informed consent has been attained, in accordance with the Declaration of Helsinki. As the patient has since passed away, informed consent for this work was obtained from the patient's next-of-kin. We believe that this, coupled with the complete anonymity of patient within the case report, is sufficient for publication of this work.

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Symptomatic interferon retinopathy successfully treated by hypertension management

Retinopathy is a common side effect of interferon treatment. We report a case of symptomatic retinopathy caused by interferon α in a hypertensive patient successfully treated by management of hypertension alone and without stopping interferon α. As far as we know, this is the first reported case of interferon retinopathy successfully treated by hypertension management.

Pathogenesis of interferon retinopathy is most likely related to microcirculation damage and the impairment of angiogenesis. If interferon α therapy offers significant clinical benefit but is complicated by retinopathy, by treating other sources of microcirculation injury such as hypertension or diabetes mellitus, symptomatic retinopathy may be successfully treated without ceasing interferon α treatment.

Case report

The patient is a 55-year-old gentleman with multiple lung metastases from renal cell carcinoma. He had normal vision and no previous history of hypertension. In a clinical trial, he was treated with 9 MIU interferon α-2a administered subcutaneously three times a week and 1000 mg bevacizumab intravenously every two weeks. He had excellent treatment response, with complete radiological remission of metastatic disease.

He developed blurred vision three months after starting interferon. As a side effect of bevacizumab he also became hypertensive, his blood pressure consistently 160/120 mmHg. On examination, visual acuity of his left eye was 6/6 and his right eye 6/5. Fundoscopic examination demonstrated multiple cotton wool spots in both retinas with no other signs of retinopathy (fig 1). Fluorescein angiography was unremarkable.

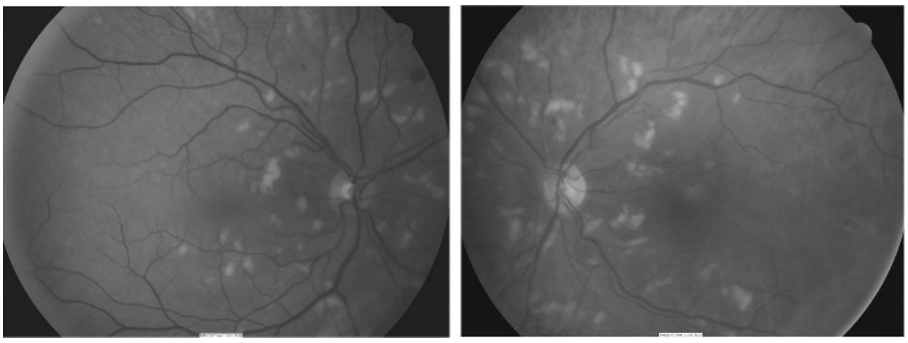

Figure 1 Retinal photography showing multiple cotton wool spots in both retinas before hypertension treatment.

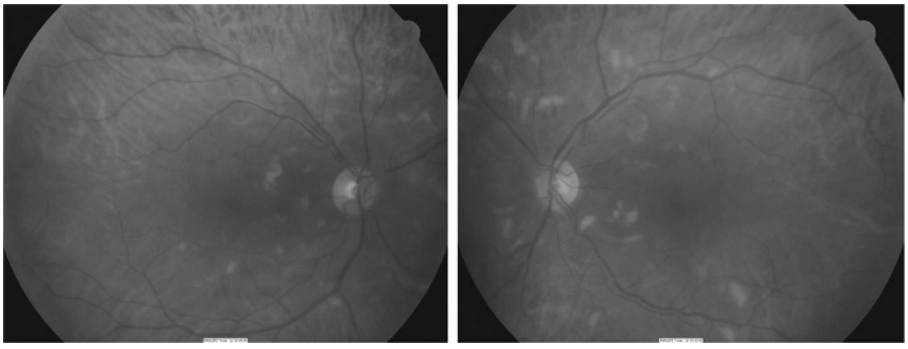

Figure 2 Retinal photography showing reduced cotton wool spots in both retinas after hypertension treatment.

Considering the excellent cancer response, benefits of continuing interferon treatment outweighed the risk of further visual impairment. The same interferon regimen was continued with close monitoring of retinopathy. Oral anti-hypertensive medication was started with reduction of blood pressure to 140/80. Three months later, blurred vision completely resolved, visual acuity normalized to 6/5 for both eyes and cotton-wool spots were significantly reduced (fig 2). He continued interferon treatment with no further complications.

Comment

Retinopathy, presenting with cotton wool spots, retinal haemorrhage or micro-aneurysms on fundoscopic examination, is the most common ophthalmic side effect of interferon occurring in 24%[1] of patients treated. Retinopathy rarely becomes symptomatic at viral hepatitis dosages, but at higher cancer treatment dosages there are more frequent case reports of symptomatic retinopathy.[2]

Pathogenesis of interferon retinopathy is still inconclusive. Ischaemia from microcirculation injury seems the most plausible explanation for retinopathy. Immune complexes deposition and inhibition of endothelial cell function, proliferation and migration have been suggested as mechanisms of microcirculation injury.[3 4] Indeed interferon-α has been shown to cause endothelial cell apoptosis in vitro.[5]

Reported risk factors for interferon retinopathy include hypertension,[1 6] diabetes mellitus,[6] high interferon dosages[1] and pegylated interferon.[1] Kawano found 80% of hypertensive patients treated with interferon developed

retinopathy[6] and d'Alteroche found hypertension carried a relative risk of 4.60 for developing retinopathy.[1] Of course hypertension and diabetes also disrupt microcirculation and can each independently cause retinopathy. We postulate for that for our patient, both interferon and hypertension contributed initially to retinopathy. By reducing blood pressure, the total amount of microcirculation injury was reduced even though effects of interferon remain unchanged, thus improving retinopathy.

In patients who develop symptomatic interferon retinopathy, if benefits of interferon treatment outweigh the impact of visual impairment, interferon may be continued. Hypertension and diabetes mellitus should be optimised to reduce retinal microcirculation injury load. Retinopathy may improve once these risk factors have been optimally controlled. Retinopathy should be closely monitored for progression.

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Keloid of the conjunctiva simulating a conjunctival malignancy

Keloids are usually formed in individuals after skin trauma and are thought to be the result of altered wound healing with excessive scar tissue formation.[1] We describe a bulbar conjunctival keloid confirmed by immunohistochemical analysis and scanning electron microscopy.

Case report

A 48-year-old Hispanic man presented with a medial conjunctival lesion in his left eye. The patient had underwent pterygium surgery in the same location 2 years previously. At 6 months after surgery, he noted progressive growth of a conjunctival mass that persisted over the next year. On examination, his vision was 20/25 in the right eye and 20/30 in the left eye. Slit-lamp examination revealed a 5 mm×10 mm firm, peduncular conjunctival mass in the left medial bulbar conjunctival region (fig 1A). There were several prominent blood vessels on its surface. Clinical impression was a conjunctival malignant neoplasm.

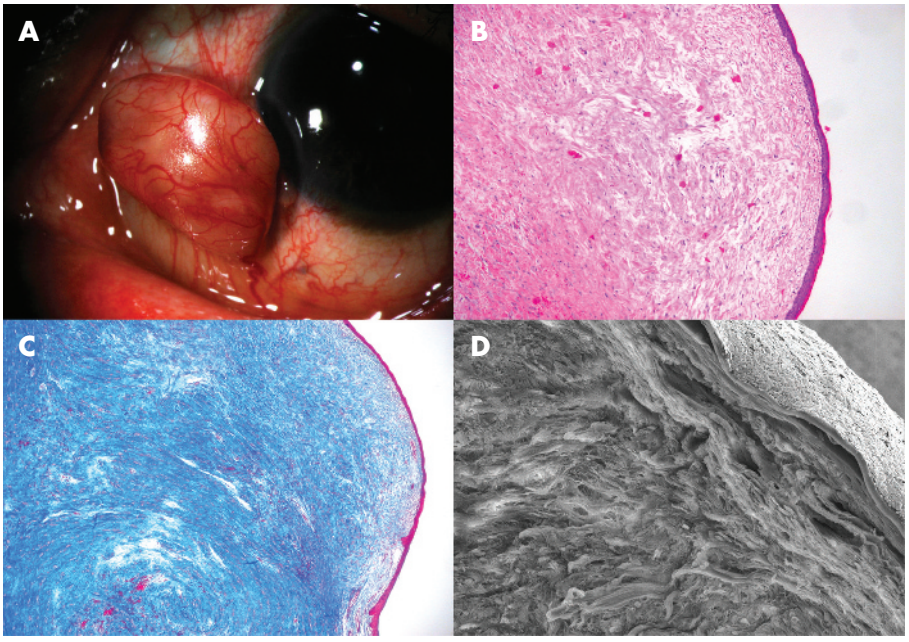

Figure 1 (A) Clinical photographs of the patient with conjunctival keloid. A peduncular conjunctival mass in the left medial bulbar conjunctival region shows prominent vascularity on its surface. (B) Histologically, conjunctival mass reveals thickened and closely packed collagen fibres in conjunctival stroma. (C) Collagen fibres stain positive for Masson trichrome. (D) Scanning electron microscopy shows random orientation of haphazardly connected collagen bundles towards the epithelial surface.

The patient underwent en bloc surgical resection, without violation of the capsule, and the lesion was sent for histopathological examination. Microscopically, the conjunctival mass revealed exuberant deposits of collagen fibres in the stroma, thickened and closely packed with hyalinisation and a paucity of cellular deposits (fig 1B). Collagen fibres stained positive for Masson's trichrome (fig 1C), and negative for CD 34 antigens and were minimally positive for α-smooth muscle actin antigen. Scanning electron microscopy revealed random orientation of haphazardly connected collagen bundles to epithelial surface (fig 1D), which are considered typical for keloid.[2] At 1 month of operation, the vision was unchanged and the conjunctiva was healed at the surgical site.

Comment

We are unaware of previous reports of conjunctival keloid and could find no reference in a computerised search using PubMed. Even though conjunctival keloids have not been reported, keloids involving cornea have been observed.[1 3 4] Keloids must be differentiated from hypertrophic scars. They both involve benign fibrous growth that occurs after trauma and show no morphological differences with light microscopy. However, they require different therapeutic approaches as keloids extend beyond the original wound, rarely regress and have a high rate of recurrence after surgical excision; whereas hypertrophic scars remain within the confines of the original wound, spontaneously regress and rarely recur after excision.[1 2 5]

Immunohistochemical analysis and scanning electron microscopy are essential to confirm the diagnosis of keloid and differentiate it from hypertrophic scar. The presence of α-smooth muscle actin-expressing myofibroblasts is a feature of hypertrophic scars, whereas keloids have only few α-smooth muscle actin-expressing myofibroblasts.[1 5] In this case, α-smooth muscle actin antigen expression is minimal, whereas scanning electron microscopy shows typical features of keloid collagen (fig 1D).

The common lesions at the site of pterygium excision include pyogenic granuloma, recurrence of pterygium and squamous neoplasias. The present case indicates the occurrence of keloid as well. Clinicians should consider keloid in the differential of conjunctival mass that occurs at the site of pterygium excision.

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