retinopathy⁶ and d'Altoche 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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doi: 10.1136/bjo.2006.112532

Accepted 11 December 2006

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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 undergone pterygium surgery in the same location 2 years previously. At 6 months after surgery, he noted progressive growth of a conjunctival mass that persisted

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.

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.

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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doi: 10.1136/bjo.2006.112227

Accepted 13 December 2006

Funding: This study was funded by the Research to Prevent Blindness, New York, USA, and NIH grant EY03040. RNK is a Heed Fellow and is supported by the Heed Ophthalmic Foundation.

Competing interests: None declared.

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Spontaneous resolution of an inflammation-associated epiretinal membrane with previously documented posterior vitreous detachment

Although resolution of idiopathic epiretinal membranes (ERM) with a subsequent posterior vitreous detachment (PVD) has been reported, we report a rare case of resolution of an inflammation-associated ERM in a young patient that was associated with a pre-existing PVD.^{1–3}

Case report

A 16-year-old white man with a history of intermediate uveitis and negative systemic work-up was seen at the Barnes Retina Institute for marked distortion and decreased visual acuity. Visual acuity was 20/20 in the unaffected right eye, 20/70 with marked distortion in the left eye. Fundus examination of the left eye revealed a previously noted Weiss ring, and a distinct cellular epiretinal membrane (ERM) with associated retinal thickening and distortion (fig 1A–E). Fluorescein angiography showed intraretinal leakage associated with the ERM (fig 1C). Treatment options including surgery were discussed but the patient elected to observe. Five months later, visual acuity was 20/25 in the left eye and Amsler grid testing showed no distortion. The ERM had spontaneously resolved with minimal retinal folds remaining (fig 2B); there was no leakage on fluorescein angiography (fig 2C); optical coherence tomography (OCT) showed significant resolution of neurosensory retinal thickening.

Comment

Without surgical intervention, ERM is typically a stable or slowly progressive condition.⁴ Resolution typically has been linked to the development of a posterior vitreous detachment (PVD). Spontaneous regression has been noted in the three instances in the English-speaking literature but with adequate colour depiction in only one instance.^{1–3} All previously published articles pertain to idiopathic macular pucker without a pre-existing PVD. Secondary ERM, unlike idiopathic ERM, are associated with pre-existing ocular disease such as diabetes, retinal vascular occlusion, vasculitis, trauma, vitreous haemorrhage or uveitis. Although the exact pathogenesis of ERM is not completely understood, they are composed of retinal pigment epithelial (RPE) cells, fibroblasts, vascular endothelial cells, monocytes and glial cells. Glial cells may be an important component of ERM as they produce several cytokines such as vascular endothelial growth factor (VEGF), growth factors such as fibroblast growth factor (FGF) and transcription factors as NK- κ B and AP-1 that might

Figure 1 Color fundus photograph (A) and high magnification of macula (D) demonstrates the ERM. Red-free photograph (B). Late-phase fluorescein angiogram shows significant ERM-associated leakage (C). OCT with arrow pointing to ERM (E).

stimulate inflammation and propagate the growth of secondary ERM.⁵ In our patient, a PVD with Weiss ring was noted at presentation in this uveitis-related ERM. To our knowledge, we present the first photographic documentation of spontaneous resolution of an inflammatory epiretinal membrane in the presence of a pre-existing PVD.

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doi: 10.1136/bjo.2006.113597

Accepted 2 January 2007

Competing interests: None.

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