of the corneal reparative process, without direct involvement of iris tissue.[11]

In a recent report of 2 cases of bilateral keloid-like myofibroblastic proliferation of the cornea, without a history of trauma or chronic inflammation, it was proposed that previously unrecognised corneal infection could introduce microbial antigens, to invoke an immune-mediated reaction resulting in a hypertrophic scar.[9]

The common occurrence of bilateral corneal keloids in Lowe's syndrome[7] suggests that in these patients keloid formation may be related to the underlying systemic abnormality. Although cataract and glaucoma surgery is commonly undertaken in such children, routine surgical procedures have not been implicated in keloid formation.

It has been suggested that amino acids may leak into the cornea from abnormal new blood vessels, thus stimulating keloid formation. Substances from the anterior chamber may also leak through defective endothelium. Tripathi and co-authors[12] described bilateral corneal keloid in an 11-year-old boy with Lowe's syndrome who had previously undergone goniotomy to both eyes, and noted that keloid formation had developed in areas not traumatised by the procedure, with no histological evidence of surgical trauma in the area of keloid formation. Similarly, in our case, bilateral corneal keloids developed many years following surgery, with lesions located inferiorly and not related to the site of previous surgery.

The management of corneal keloid poses many problems. Surgical excision of keloids often results in recurrence and enlargement of the lesions.[1] Although essentially benign, keloids may severely affect vision, and large hypertrophic scars can interfere with lid closure.

The deep stromal involvement of the lesions precludes lamellar keratoplasty. Equally, penetrating keratoplasty in these young and mentally retarded individuals may not be considered a viable option.[8]

Treatment should be directed at removing the existing lesion and preventing recurrence by inhibiting fibroblastic proliferation and collagen synthesis. The use of radiotherapy in the management of keloids in general has been reported – in particular, the use of superficial X-rays or strontium-90 beta-rays. Other physical forms of treatment include ultrasound, cryotherapy, pressure therapy and laser. Dermal keloids have also responded to intralesional injection of steroid.[1] Cibis and co-authors[7] commented on the persistence of mast cells in their ultrastructural analysis of corneal keloid, and suggested that suppression of inflammation by topical steroid or the use of mast cell stabilisers may be useful in the management of this condition.

The management of corneal keloids should therefore be conservative in the first instance, with surgical intervention reserved for those lesions which severley affect vision or impede adequate lid closure.

A. M. McElvanney*
H. P. Adhikary

Royal Preston Hospital
Sharoe Green Lane
Preston PR2 4HT
UK.

*Correspondence address:
Manchester Royal Eye Hospital
Oxford Road
Manchester M13 9WH
UK.

References

1. Datubo-Brown DD. Keloids: a review of the literature. Br J Plastic Surg 1990;43:70–7.
2. Smith HC. Keloid of the cornea. Trans Am Ophthalmol Soc 1940;38:519–38.
3. Fenton RH, Tredici TJ. Hypertrophic corneal scars (keloids). Surv Ophthalmol 1964;9:561–6.
4. Farkas TG, Znajda JP. Keloid of the cornea. Am J Ophthalmol 1968;66:319–23.
5. O'Grady RB, Kirk HQ. Corneal keloids. Am J Ophthalmol 1972;73:206–13.
6. Frederique G, Howard RO, Boniuk V. Corneal ulcers in rubeola. Am J Ophthalmol 1969;68:996–1003.
7. Cibis GW, Tripathi RC, Tripathi BJ, Harris DJ. Corneal keloid in Lowe's syndrome. Arch Ophthalmol 1982;100:1795–9.
8. Weiner MJ, Albert DM. Congenital corneal keloid. Acta Ophthalmol (Copenh) 1989;67:188–96.
9. Holbach LM, Font RL, Shivitz IA, Jones DB. Bilateral keloid-like myofibroblastic proliferations of the cornea in children. Ophthalmology 1990;97:1188–93.
10. Lowe CU, Terrey M, MacLachlan EA. Organic-aciduria, decreased renal ammonia production, hydrophthalmos, and mental retardation: a clinical entity. Am J Dis Child 1952;83:164–84.
11. Shoukrey NM, Tabbara KF. Ultrastructural study of a corneal keloid. Eye 1993;7:379–87.
12. Tripathi RC, Cibis GW, Tripathi BJ. Symposium on ocular pathology: Lowe's syndrome. Trans Ophthalmol Soc UK 1980;100:132–9.

Sir,

Localised Giant Papillary Conjunctivitis Secondary to a Dermolipoma

Dermolipomas are solid choristomas formed from displaced embryonic material, destined to become skin, that has been sequestered to the conjunctiva.[1] They typically occur on the superolateral epibulbar surface with a posterior extension into the orbit.[2] The appearance is of a smooth pinkish-yellow mass in the conjunctiva. Histologically they consist of a keratinised squamous epithelium containing adnexal

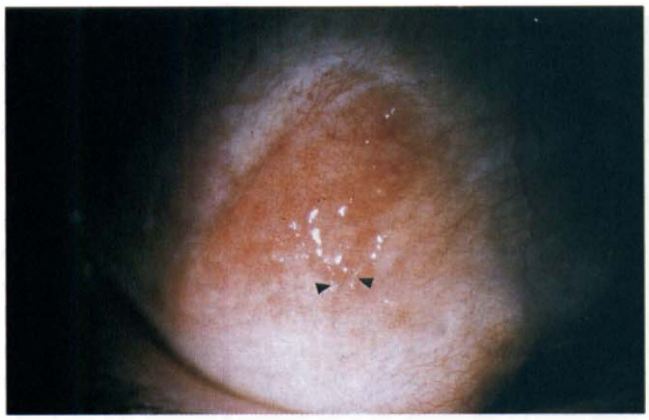

Fig. 1. *Dermolipoma of right superotemporal epibulbar surface showing fine hairs (arrows).*

structures and fat and connective tissue septae in the underlying dermis.[1]

The lesions are often cosmetically embarrassing but do not usually cause functional problems, although fine hairs on the surface may cause irritation. Surgical excision should be conservative and confined to the irritating dermal surface or to the visible portion of the lesion, since more extensive dissection may lead to severe post-operative complications;[3,4] these include damage to lacrimal gland ductules (resulting in keratitis sicca), tethering of lateral and superior rectus muscles with resultant strabismus, and scar tissue adhesions causing mechanical ptosis.[5]

Case Report

A 14-year-old girl presented with a dermolipoma of the right eye. Two years previously she had undergone attempted excision of the lesion but felt that it was unchanged post-operatively. She complained of the cosmetic appearance of the dermolipoma and also that the vision in the right eye blurred intermittently throughout the day, but could be cleared by blinking.

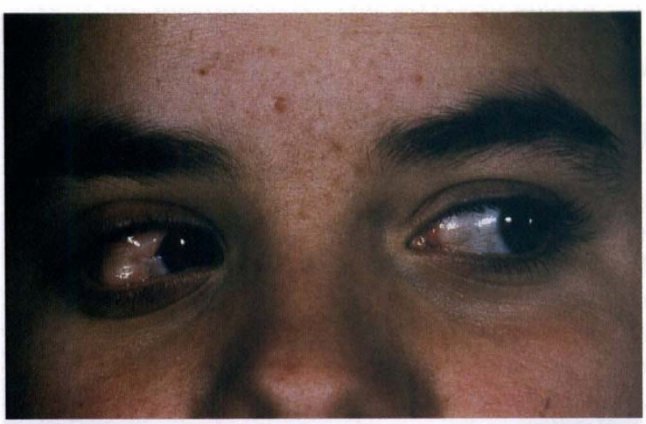

Fig. 2. *Previously operated dermolipoma of right eye with lateral ptosis and a lateral canthal conjunctival scar slightly restricting adduction.*

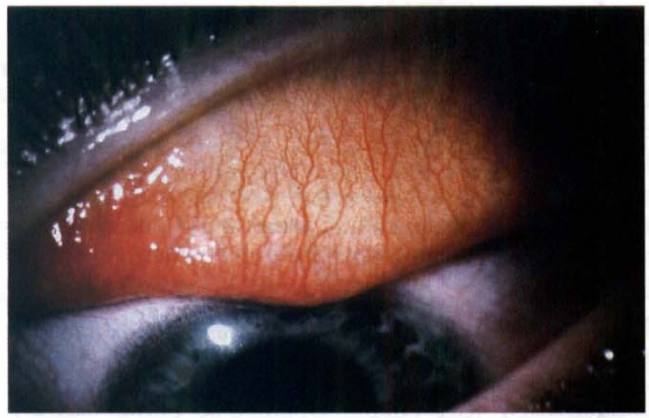

Fig. 3. *Giant papillary conjunctivitis of the lateral third of the right upper tarsus.*

On examination the visual acuity was 6/6 right, 6/5 left, unaided. There was a dermolipoma in the superolateral aspect of the right epibulbar surface which bore many fine hairs on its surface (Fig. 1). A horizontal conjunctival scar was adherent to the lateral canthus, minimally restricting adduction of the eye and the upper eyelid showed a slight lateral droop (Fig. 2). There was accumulation of mucus in the lower fornix spreading across the cornea. Eversion of the upper lid revealed a marked giant papillary conjunctivitis confined to the lateral third of the tarsal conjunctiva (Fig. 3).

The patient is awaiting excision of the hair-bearing epithelium and microscopic dissection of the interpalpebral portion of the dermolipoma with mucous membrane graft.

Comment

Irritation from a dermolipoma is due to the ectopic hair-bearing skin on the surface of the lesion and, in order to eradicate this symptom successfully, this area needs to be excised. Our patient showed signs of chronic irritation with giant papillary conjunctivitis confined to an area of tarsal conjunctiva adjacent to the hair-bearing part of the dermolipoma – a sign which we have observed in some other cases of hair-bearing dermolipomas. She was troubled by mucus secretion from the conjunctivitis which obscured her vision when it smeared across the visual axis. This report illustrates the association between hair-bearing dermolipoma and adjacent giant papillary conjunctivitis.

Ruth M. Manners
Steven J. Vardy
Geoffrey E. Rose

Moorfields Eye Hospital
City Road
London EC1V 2PD
UK

References

1. Rootman J, Lapointe JS. Structural lesions. In: Rootman J. Diseases of the orbit. Philadelphia: JB Lippincott, 1988:494–7.
2. Eijpe AA, Koornneef L, Bras J, Verbeeten B, Peeters FL, Zonneveld FW. Dermolipoma: characteristic CT appearance. Doc Ophthalmol 1990;74:321–8.
3. McNab AA, Wright JE, Caswell AG. Clinical features and surgical managment of dermolipomas. Aust NZ J Ophthalmol 1990;18:159–62.
4. Beard C. Dermolipoma surgery or 'an ounce of prevention is worth a pound of cure'. Ophthalmic Plast Reconstr Surg 1990;6:153–7.
5. Dortzbach RK, Kronish JW. Orbital disease. In: Dortzbach RK, editor. Ophthalmic plastic surgery: prevention and management of complications. New York: Raven Press, 1994:332–3.

Sir,

Ectopic Retinal Tacks

Retinal tacks provide a means of achieving intra-operative retinal fixation in complicated retinal detachments. On rare occasions they may become dislodged and can move within the globe. We describe a case in which this occurred with displacement of retinal tacks into the anterior chamber.

Case Report

A 59-year-old man was referred in June 1993 with a right retinal detachment. He had already undergone surgery at his local hospital for a superonasal detachment and received treatment with cryotherapy and a 5 mm radial explant. This proved unsuccessful and his retina subsequently redetached.

At presentation here, his visual acuity was perception of light with the right eye and 6/6 with the left. Fundal examination revealed a total right retinal detachment with an open break superiorly and grade D proliferative vitreoretinopathy (PVR). A pars plana vitreolensectomy was performed the following day under general anaesthesia. A large relieving retinotomy was created inferiorly to release traction from the PVR. Laser photocoagulation was applied to the edge of the retinotomy and the retina was flattened per-operatively with the aid of two tacks (Grieshaber 611.94) and silicone oil. The retina subsequently remained attached and the patient's visual acuity improved to counting fingers.

During a routine clinic visit, 9 months after the surgery, the patient mentioned that his right eye had been painful for the preceding fortnight. Slitlamp biomicroscopy revealed a retinal tack lying inferiorly in the anterior chamber with adjacent stromal and epithelial oedema (Fig. 1). Numerous folds in Descemet's membrane were present together with rubeosis iridis and a moderate degree of uveitis. After admission the tack was removed through a small limbal paracentesis under general anaesthesia.

Two months later, at a subsequent clinic visit, a second tack was found to be present in the inferior anterior chamber. Corneal changes similar to those described above were present (Fig. 2). This tack was removed using a similar technique and the patient has made a good recovery from this further surgery.

Discussion

Conventional retinal reattachment techniques have a lower success rate in complicated retinal detachments. Unrelieved retinal traction makes standard methods of retinopexy such as cryotherapy or photocoagulation, unsuccessful. Retinal tacks provide a means of apposing retina to retinal pigment epithelium (RPE), facilitating effective internal tamponade with gas or silicone oil. Tacks are also occasionally useful in the treatment of giant retinal tears and following relieving retinotomies to immobilise freely mobile retina. Their role in treating these conditions has, however, been largely supplanted by perfluorocarbon liquids.[1]

The first retinal tacks,[2] introduced in 1983, were made from polyacetal, an extremely hard plastic. Subsequently, inert metallic tacks[3] have been designed and those currently available are made from titanium[4] (Fig. 3). These tacks have minimal

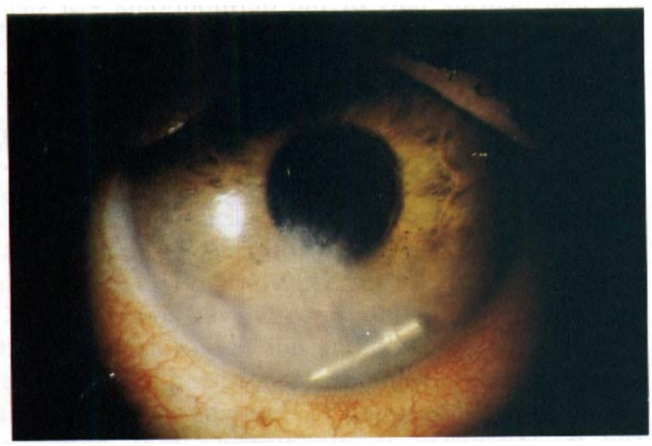

Fig. 1. *Slit lamp biomicroscopy of the right eye showing a retinal tack lying inferiorly in the anterior chamber with adjacent stromal and epithelial oedema.*

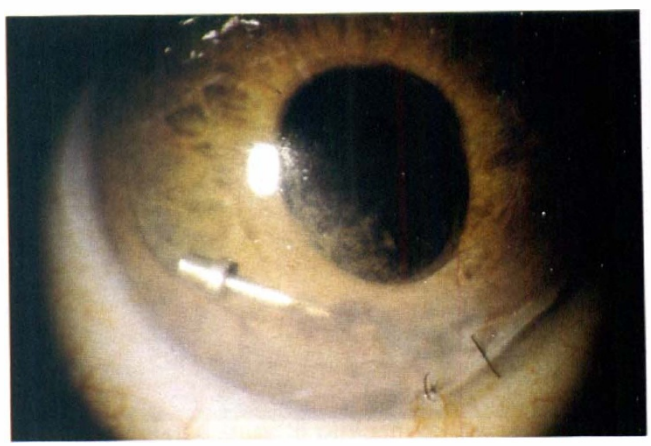

Fig. 2. *A second tack in the inferior chamber found at a clinic visit 2 months after the first tack.*