Sir,
Recurrence of giant pseudocyst in advanced bullous keratopathy

An 80-year-old man presented to eye casualty in 1995 with a 4-day history of discomfort in his right eye. His family doctor had noticed a polypoid lesion protruding from his right cornea. The patient could not recall the duration of this lesion.

In February 1988, he had undergone a right extracapsular cataract extraction (ECCE) with anterior chamber lens implant (AC-IOL) at a different hospital. No further details were available. An uneventful left ECCE with posterior chamber lens implant (PC-IOL) was carried out in 1989 in our institution. His right eye had remained chronically inflamed following his cataract surgery for which he received steroids and mydriatics.

Despite occasional episodes of moderate elevation in intraocular pressure (IOP) (21–25 mmHg), no regular hypotensive medications were required. In April 1989, the right cornea showed signs of early endothelial decompensation and visual acuity was counting fingers. He was discharged from the clinic 1 year later.

When he presented in 1995, visual acuity was light perception in the right eye and 6/9 with correction in the left eye. His right eye was injected and the cornea was grossly oedematous due to endothelial failure. He had a large vascularised, pedunculated opaque lesion extending from the infero-nasal aspect of his right cornea into the conjunctival sac (Figure 1a). Because of the unknown nature of the lesion, an excision biopsy was carried out. The biopsy showed hyperplastic and metaplastic conjunctival epithelium with a fibrinous exudate in the underlying stroma. The patient remained comfortable and was discharged from the clinic.

The patient was rereferred by his optician in May 2001 because of a change in appearance of his right cornea. On this occasion, a large bulla had developed in the infero-nasal cornea. This was surgically deroofed, and histological examination showed a cystic space with overlying vascularised tissue and hyperplastic surface epithelium. In March 2002, the patient was referred to the corneal specialist in our department because of recurrence of this bulla with associated discomfort.

A right penetrating keratoplasty and anterior vitrectomy with removal of the AC-IOL was performed in March 2002.

The histological appearances of the cornea were in keeping with very advanced bullous keratopathy. The central corneal epithelium was denuded and there was

Figure 1 (a) Clinical appearance of the corneal pseudocyst. (b) Histologic section of the corneal pseudocyst; it consisted of a fluid-filled cystic space (asterisk) covered by keratinising corneal epithelium (arrow) with underlying granulation tissue and areas of fibrin exudation.

marked pannus covering the surface, forming the floor of the bulla. The underlying stroma showed patchy scarring and marked vascularisation. The roof of the bulla consisted of a lining of granulation tissue with areas of fibrin exudation, covered by keratinising corneal epithelium (Figure 1b).

His recovery has been uneventful, and 8 months after the graft there are no signs of recurrence of his previous lesion. Aphakic visual acuity is now counting fingers due to 'dry' age-related macular degeneration. The optic disc remains healthy.

Comment

Pseudocysts are acquired fluid-filled cavities lined by fibro-connective tissue that differ from true cysts because

they lack an epithelial lining.¹ In our case, keratinising corneal epithelium covered the lesion rather than lining it, thus confirming it as a pseudocyst.

Corneal epithelial pseudocysts are a rare condition. They are the late consequence of inadequate healing and repair within the cornea following a variety of pathologic insults.²

A more common cause for pseudocyst formation is surgically induced trauma after cataract surgery. It has been well established that corneal endothelial decompensation leading to bullous keratopathy is a common cause of corneal epithelial bulla — a form of pseudocyst.³

A large pseudocyst following acute hydrops was described by Margo and Mosteller.¹

Pseudocyst formation is the structural manifestation of a failed reparative response to a specific injury. There seems to be a requirement for the initiation of bulla formation (eg corneal decompensation, raised IOP); injury to corneal endothelium leads to loss of normal fluid homeostasis within the corneal stroma. If the injury to the endothelium is severe enough, the accumulation of extracellular fluid continues until pockets of fluid form within the corneal stroma, with hydrostatic forces pushing aqueous fluid into the subepithelial space. The coexistence of a fibrovascular response (pannus) also seems to play an important role, reinforcing the superficial wall, therefore allowing enlargement to such a degree as is seen in our case.

The response of these lesions to treatment is limited. Simple drainage procedures or aspirations have no permanent benefit. Excision of at least a portion of the anterior wall with chemical cautery seems to be successful in some cases,⁴ but such treatments carry potential adverse visual consequences. An attempt to minimise these negative outcomes includes placement of lamellar grafts after excision of the anterior wall.⁵ Penetrating keratoplasty may be required when the pseudocyst involves the visual axis.⁶

To our knowledge, this is the first report of recurrent corneal pseudocyst following chronic corneal oedema secondary to pseudophakic bullous keratopathy. In these persistent lesions, if there is underlying corneal disease that would benefit from penetrating keratoplasty, combining this with excision of the lesion may result in satisfactory clearance.

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Eye (2004) **18**, 859–860. doi:10.1038/sj.eye.6701333

Published online 20 February 2004

Sir,

Endogenous endophthalmitis secondary to Lemierre's syndrome

Endogenous bacterial endophthalmitis is a rare condition with a prevalence of approximately 2–8% of all cases of endophthalmitis. The organisms commonly implicated are Streptococci, Staphylococci, *Escherichia coli*, *klebsiella*, *Bacillus cereus*, and *Listeria*. *Fusobacterium necrophorum* is a gram-negative anaerobic bacillus, which is a normal inhabitant of the human alimentary tract. The organism is an opportunistic pathogen, which causes a rare disease known as Lemierre's syndrome,¹ characterized by septic thrombosis of the internal jugular vein after oropharyngeal infection. *Fusobacterium necrophorum* has never been implicated as a cause of endogenous endophthalmitis. We report a first case of endogenous endophthalmitis in a patient with Lemierre's syndrome due to *Fusobacterium necrophorum*, which resolved without the need of intravitreal antibiotics.

Case report

A 53-year-old man was admitted in the chest medicine ward with a 5-day history of malaise, sore throat, and left-sided chest pain. On presentation, he was confused, febrile, hypoxic (oxygen saturation was 77% on air), and