60 U on densitometry scale. This 'hanging drop' or 'tear drop' sign on Scheimpflug photography is probably created by preexisting PCD with herniation of dense posterior plaque through it.

Comment
This simulated posterior lenticonus producing hanging drop/tear drop sign should be taken as diagnostic of PCD in posterior polar cataracts. To our knowledge, this is the first report of PCD with coexistent posterior polar cataract being characterized on Scheimpflug imaging.

Conflict of interest
The authors declare no conflict of interest.

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Eye (2010) **24,** 737–738; doi:10.1038/eye.2009.134; published online 12 June 2009

Sir,

Management of inadvertent peribulbar injection of acetazolamide: a case report

Drug errors can have severe consequences. Here we describe an inadvertent peribulbar injection of acetazolamide instead of local anaesthetic agent, prior to cataract surgery.

Case report
A 63-year-old male with glaucoma was to undergo right cataract surgery under peribulbar anaesthesia. Intravenous (IV) acetazolamide (500 mg in 10 ml) was planned for intra-operative use but had been drawn up pre-operatively. Eight millilitres of this solution were inadvertently given as a peribulbar injection by the anaesthetist (not one of the authors) instead of the anaesthetic agent. The patient complained of disproportionate pain during injection. The mistake was recognized and surgery deferred. On examination vision was maintained, but ocular motility was reduced by 50% in all directions of gaze. There was marked lid oedema with mild conjunctival chemosis. The patient was promptly given 200 ml of IV mannitol 20% to reduce the intraorbital pressure. An orbital opinion was sought and as there was no information in the literature or from the poisons unit regarding further management, the patient was given IV methyl prednisolone (500 mg) stat and prophylactic IV cefuroxime (750 mg) on an empirical basis and admitted for regular monitoring. Subsequently he was started on oral prednisolone (40 mg) for 5 days. His ocular motility recovered to normal and the lid oedema and chemosis settled in 48 h. A month later he underwent right cataract surgery. Eighteen months after the incidence his vision is 6/5 in the right eye with full ocular motility and no lid or orbital problems.

Comment
Some medications can cause severe soft tissue and skin necrosis when accidentally injected or extravasated into soft tissues. Extravasation of acetazolamide (a high-risk vesicant drug, pH 9.1) causing soft-tissue necrosis of the forearm has been reported once.[1] No specific antidote is available to counteract acetazolamide. In this patient, IV methyl prednisolone may have had a role in the prevention of complications. The diluted acetazolamide (500 mg in 10 ml water) could be another factor. As a general rule, prevention is the cornerstone and avoiding similar problems can be achieved by using a clear labelling system[2] and drawing up the required injection immediately before its administration.

Conflict of interest
The authors declare no conflict of interest.

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We report a unique case of inadvertant periorbital injection of acetazolamide and its management.

Eye (2010) **24,** 738; doi:10.1038/eye.2009.139; published online 26 June 2009

Sir,

Histopathology and treatment of corneal disease in keratitis, ichthyosis, and deafness (KID) syndrome

A 34-year-old male with keratitis–ichthyosis–deafness (KID) syndrome and documented mutation in the *GJB2*

gene was examined for progressive corneal neovascularization in the left eye. He had been treated for over 10 years with intermittent topical corticosteroids; systemic doxycycline; and a penetrating keratoplasty, which had failed because of recurrent surface disease.

Examination revealed a visual acuity of 20/30 in the right eye and HM in the left eye, partial loss of eyebrows, complete loss of eyelashes, and atrophy of meibomian gland openings (Figure 1a and b). Tear production was low according to Schirmer's test. The right cornea showed abnormal peripheral epithelium with fine superficial neovascularization (Figure 1c). The left cornea had extensive superficial and deep neovascularization and scarring (Figure 1d).

The patient underwent a keratolimbal allograft and a penetrating keratoplasty in the left eye with systemic immunosuppression consisting of prednisone, tacrolimus, and mycophenolate. Histopathology showed thickened epithelium with parakeratosis and dyskeratosis, but no goblet cells in the cornea (Figure 2). The conjunctiva had very few goblet cells with occasional keratinization.

Postoperatively, the patient developed significant inflammation and suture induced neovascularization resulting in surface failure and stromal scarring. Therefore, he required a repeat keratolimbal allograft and penetrating keratoplasty. He subsequently maintained a vision of 20/50–20/100 for 4 years before gradually developing recurrent surface disease (Figure 3). The patient is being considered for a Boston Keratoprosthesis.

The right eye has been managed effectively with cyclosporine 0.05% two to three times a day, and a large scleral gas permeable contact lens which significantly reduces the patient's photophobia. His visual acuity remains 20/40.

Previous treatments of the corneal disease in KID syndrome have included superficial keratectomy and penetrating keratoplasty, both of which lead to recurrence.[1,2] Topical corticosteroids and cyclosporin have been shown to improve the surface disease, whereas systemic treatment with vitamin A actually worsens the surface disease.[3,4] Previously, amniotic membrane and limbal transplantation were not successful in a patient.[5]

This report shows that the corneal epithelium in our patient with KID syndrome had abnormal differentiation. Although we could not confirm true limbal stem cell deficiency, the improvement with limbal transplantation lends further support to this possibility.

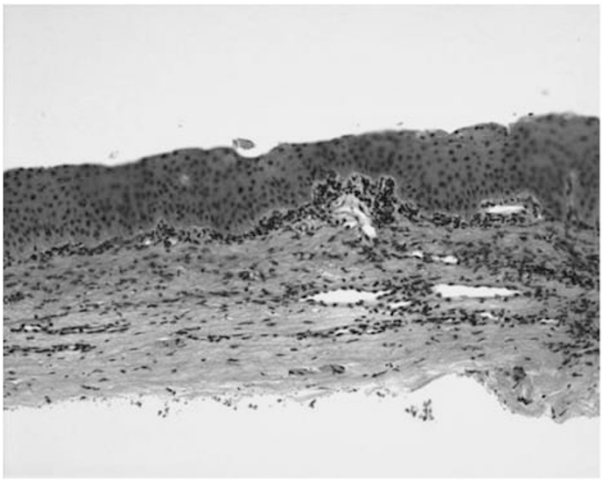

Figure 2 Photomicrograph of the corneal button. The corneal epithelium is thickened with poorly differentiated epithelial cells towards the surface. There is loss of Bowman's layer, chronic inflammation in the sub-epithelial region, and neovascularization in the stroma.

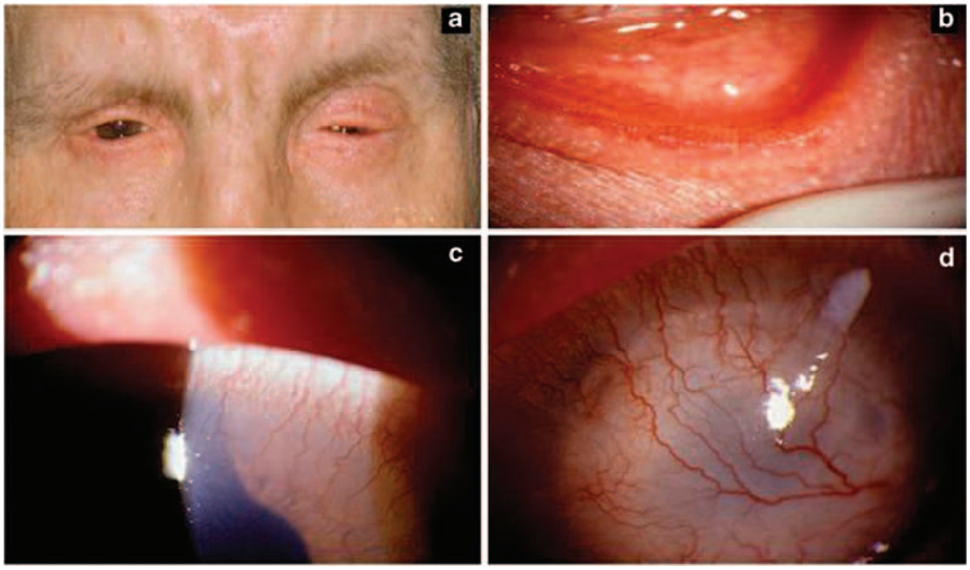

Figure 1 Clinical pictures of patient with keratitis–ichthyosis–deafness (KID) syndrome. (a) Forehead skin with deep furrows, complete loss of eyelashes and partial loss of eyebrows. (b) Loss of eyelashes with atrophy and plugging of the meibomian gland orifices. (c) The right cornea showing a rim of abnormal epithelium with superficial neovascularization peripherally. (d) The left cornea showing significant stromal scarring with extensive neovascularization.

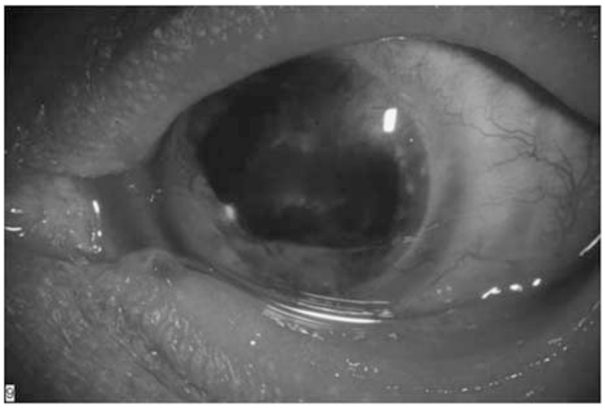

Figure 3 Postoperative appearance of the left eye at 3 years after keratolimbal allograft and penetrating keratoplasty. There is mild recurrence of the surface disease with superficial neovascularization in some areas.

Conflict of interest

The authors declare no conflict of interest.

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Eye (2010) **24,** 738–740; doi:10.1038/eye.2009.178; published online 10 July 2009

Sir,

Infectious scleritis and surgical induced necrotizing scleritis

We read with great interest the article 'Microbial scleritis—experience from a developing country' by Jain *et al*[1] from India. Infectious scleritis, although rarely discussed in western literature, is not so unusual in Asia. We are very glad to see this study, which reveals completely different pathogens of the infectious scleritis as we have known in Taiwan.

We thank the authors who have cited our article many times in their article, but in the discussion section they cited our early hypothesis that surgical induced necrotizing scleritis (SINS) may be a prodromal factor to induce the infectious scleritis, and they concluded that not a single collagen vascular disease can be identified in their own series and others. We would like to point out that to inspect our hypothesis, we have performed a prospective study and published the results in *Cornea*, 2006, titled 'Immunological and clinical manifestations of infectious scleritis after pterygium excision'.[2] In that study we have referred our cases of infectious scleritis to a rheumatologist, who performed a thorough examination of these cases and reached the conclusion that no underline autoimmune disease associated with these 18 eyes of 18 patients (16 bacteria, 2 fungi) can be identified. We would like to confirm that 'the infectious scleritis is different from the post-surgical necrotizing scleritis both in clinical and immunological aspects except for the similar long latent period.' With this article, we reject our earlier hypothesis and maintain that the mystery of long latent period on infectious scleritis after surgery is still unresolved.

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Eye (2010) **24,** 740; doi:10.1038/eye.2009.144; published online 12 June 2009

Sir,

Histopathological findings in an epimacular membrane after intraoperative use of perfluorocarbon liquid

We describe the histopathology of an epiretinal membrane (ERM) that developed after intraoperative use of perfluorocarbon liquids (PFCL: fully fluorinated compounds with high specific gravities[1]).