

Question 3	Who checks the calibration?
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- Servicing Engineer
- Orthoptist
- Nurse
- Senior House Officer
- Registrar/Staff Grade
- Consultant
- Other:.....

Question 4	What are your opinions regarding how often the Goldmann calibration should be checked?
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- Only after an abnormal result
- Before every clinic
- Daily
- At least once a week
- At least once every 3 months
- At least once every 6 months
- At least annually.
- Other:.....

Question 5	Who should check the Goldmann calibration?
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- Servicing Engineer
- Health Care Assistant
- Nurse
- Orthoptist
- The attending doctor
- Other:.....

Thank you for your time and patience!!

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Sir,
Circumferential epithelial defect at flap margins in patient with adenoviral conjunctivitis and previous LASIK

Laser *in situ* keratomileusis (LASIK) is an increasingly popular procedure for the treatment of myopia, astigmatism, and hyperopia. With time, the corneal flap created will adhere to the posterior stromal bed but the interface and flap remain at risk of complications, including diffuse lamellar keratitis (DLK) and traumatic dislocation, for many years following the procedure.^{1–3} We describe a patient who presented with viral conjunctivitis and a large epithelial defect at the flap margins 2 years following uncomplicated LASIK.

Case report

A 34-year-old man presented with a 1 week history of redness and watering of both eyes, associated with discomfort and blurred vision. The symptoms had initially started in the right eye and then spread to the left within days. He had undergone uncomplicated LASIK

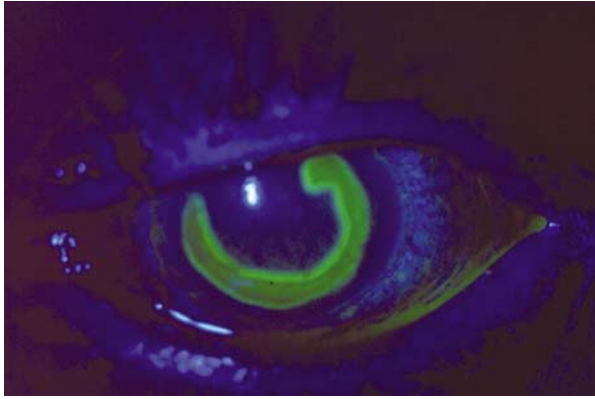


Figure 1 Photograph of patient's right eye on presentation, following application of proxymetacaine and fluorescein, showing circumferential epithelial defect at margins of LASIK flap.

procedures for myopia to both eyes 2 years previously in another centre, and experienced no eye problems in the meantime.

On examination, pinhole visual acuities were 6/24 in the right eye and 6/6 in the left. There was a watery discharge from both eyes with oedema of all eyelids. Both eyes had conjunctival injection with widespread follicles but no pseudomembrane formation. Both corneas had diffuse fine white epithelial dots, typical of the keratitis seen with adenoviral infection. The right cornea also had a 270° epithelial defect (see Figure 1); the location of the epithelial defect in the right eye corresponded to the position of the LASIK flap edge visible on the left cornea. Bacterial and viral swabs were taken, and adenovirus was subsequently confirmed on culture. On further questioning, the patient admitted to rubbing his eyes, although denied that it was vigorous.

The patient was treated with chloramphenicol ointment qds in both eyes, and on review the following week, the epithelial defect to the right cornea had closed completely, the flap was secure, and there was no evidence of epithelial ingrowth or opacification of the interface. There was also no evidence of a corneal dystrophy that might predispose to epithelial defects.

Comment

LASIK involves the creation of a corneal flap and subsequent ablation of the exposed stromal bed with an excimer laser. The flap edges progressively fibrose to make the flap more secure.⁴ However, in the early postoperative period (days to months), the interface remains at risk of complications including epithelial ingrowth, infection, DLK, fluid collections, and spontaneous shift or dislocation of the flap.^{3,5} It is now also apparent that the some of these risks persist for many years following uncomplicated procedures. DLK,

a non-infectious infiltrate occurring at the interface, can follow a variety of corneal or inflammatory insults, and has been reported in association with adenoviral conjunctivitis 2 years following uncomplicated LASIK.³ Late dislocation of the flap has been described up to 7 years after the initial procedure.^{1,2} Many cases occur following significant blunt trauma (such as being struck on the eyes with a finger or airbag), although more minor trauma may sometimes be responsible, and apparently spontaneous cases have also been described.⁶

Disturbances to the flap margin, whether epithelial alone or full flap thickness, will be associated with increased risks of infection, DLK or epithelial ingrowth and careful follow-up is advisable.^{3,5} A displaced flap may look similar to an epithelial defect, but care should be taken to distinguish between the two, as displaced flaps are best managed with early surgical repositioning to reduce the risk of developing fixed folds.⁵

The case of adenoviral conjunctivitis in a patient with previous LASIK described by Gris *et al*³ had diffuse opacification of the interface evident on presentation leading to the diagnosis of DLK. This was in addition to the epithelial white dots, typical of adenoviral conjunctivitis, seen also in our patient. Gris *et al*³ therefore treated their patient with a tapered dose of topical steroids and the interface infiltrate was seen to diminish within 10 weeks of presentation. Their case demonstrates the risk of late DLK, in response to an inflammatory insult, and this is a complication for which early diagnosis, and treatment with topical corticosteroids, is important for visual outcome.

The patient we described experienced a common conjunctival infection 2 years following his LASIK procedure. There was no evidence of the flap itself becoming shrivelled or dislocated but the stability of the overlying epithelium at the flap margin was compromised, leading to frank epithelial loss. This has not previously been described as such a late complication and may relate to the patient's history of conjunctivitis and eye rubbing.

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Sir,
An unusual presentation of Terson's syndrome

Terson's syndrome is an oculo-cerebral condition of retinal and vitreous haemorrhage most commonly associated with subarachnoid haemorrhage (SAH). It was first described by Albert Terson in 1900.¹ Intraocular haemorrhages have been documented in 10–40% of patients with SAH.² This figure may be substantially higher considering the mortality associated with this condition. Ocular haemorrhages tend to be bilateral but can be unocular. Early studies suggested that the size, number, and distribution of haemorrhages may be of relevance to the degree of intracranial haemorrhage.^{3,4} It has been found that any type of intraocular haemorrhage in patients with SAH may be associated with increased mortality, although retrospective studies confirm that vitreous haemorrhage is an indicator of poor prognosis in SAH.^{5,6} It has been postulated that the source of vitreous haemorrhage is due to damage to peripapillary tissue induced by intracranial hypertension transmitted via the intervaginal space of the optic nerve sheath.⁷ Typically visual loss is noted only after the patient regains consciousness.

We describe a case of visual loss secondary to Terson's syndrome from aneurysmal rupture that presented to the

ophthalmic emergency service without any classical symptoms or signs of SAH.

Case report

A 50-year-old gentleman presented to the Eye Casualty with gradual reduction in vision in his right eye over several days. He had had a 'flu like' illness approximately 3 weeks earlier with associated myalgia, sweats, pyrexia, vomiting, diarrhoea, and significant weight loss. This had resolved spontaneously. He took no medications and was a non-smoker. There was no significant past history.

On examination visual acuity was HM RE and 6/12 LE. Vitreous haemorrhage precluded the fundal view in the right eye, and the left eye showed multiple intra- and pre-retinal haemorrhages and disc swelling (Figure 1). An ultrasound scan of the right eye revealed no obvious fundal pathology. The rest of the ocular examination was unremarkable. Systemic examination was normal and it was noted that he had a cachectic appearance. It was felt that several of the retinal haemorrhages in his left eye



Figure 1 Fundus photographs of right and left eyes.