

Clinicopathological Report

Acute corneal hydrops with perforation after LASIK-associated keratectasia

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ABSTRACT

Herein a case of acute corneal hydrops with perforation in keratectasia 9 years after bilateral laser *in situ* keratomileusis is presented. A 27-year-old man presented with right eye sudden loss of vision. Right eye best-corrected visual acuity was counting fingers. Left eye best-corrected visual acuity was 6/9. Right eye slit-lamp examination revealed diffuse corneal oedema with central epithelial cystic change. The interface between the flap and residual stroma was open and filled with fluid. This separation extended nearly to the flap margin. Shallow anterior chamber and aqueous leakage were noted the following day. An emergency penetrating keratoplasty was performed to the right eye. Histopathology of corneal button revealed reactive keratocytes with irregular, wrinkled collagen formation over ablated stroma. Marked stromal oedema was observed. Twelve months after corneal transplantation, vision improved to 6/6 with correction in the right eye.

Key words: acute corneal hydrops, astigmatism, keratectasia, LASIK.

INTRODUCTION

Keratectasia is an insidious complication of laser *in situ* keratomileusis (LASIK), occurring months after an apparently uncomplicated procedure.¹ The development of acute corneal hydrops and perforation has not been reported previously. Chung *et al.* reported the first case of a patient with acute hydrops after LASIK, in whom forme fruste keratoconus was suspected before LASIK.² Here, we present a preoperatively normal case in which acute corneal hydrops and perforation in keratectasia evolved 9 years after an uneventful bilateral LASIK procedure.

CASE REPORT

A 27-year-old man was referred to our hospital in February 2005 with sudden onset of loss of vision in the right eye, which persisted for 2 days. The patient underwent an uneventful bilateral LASIK procedure for moderate myopia correction 9 years before presentation at another hospital. Preoperatively, the manifest refraction was -4.50 dioptres (D), yielding a best-corrected visual acuity (BCVA) of 6/6 in both eyes. Ultrasound pachymetry data were $561\ \mu\text{m}$ in the right and $566\ \mu\text{m}$ in the left eye. Corneal topography showed an asymmetric bow-tie pattern and no significant irregular astigmatism in either eye (Fig. 1a). Retrospective evaluation of the corneal topography showed that the inferior minus superior value was $0.408\ \text{D}$ in the right eye and $0.65\ \text{D}$ in the left eye.

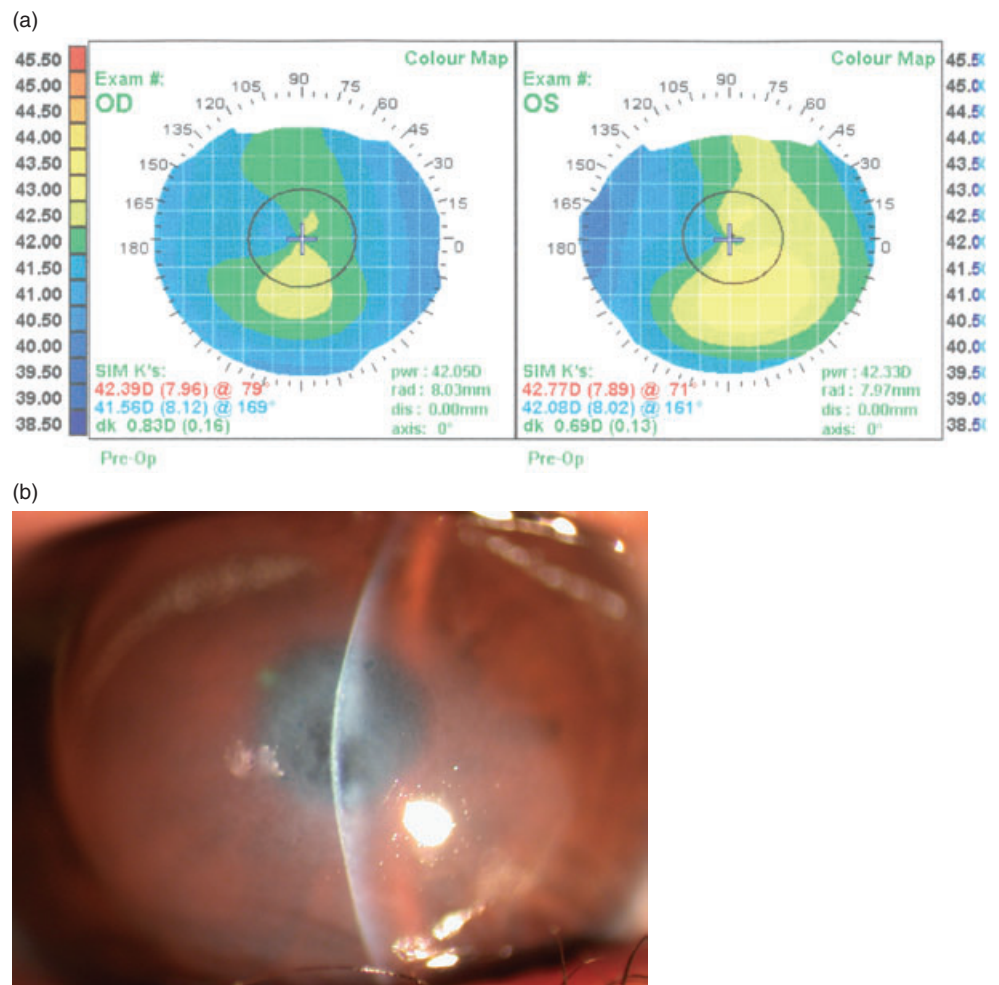
LASIK was performed on both eyes in January 1996. A Moria One microkeratome (Moria, Doylestown, PA, USA) was used to cut a flap of $130\ \mu\text{m}$. The predicted ablation depth was $58.5\ \mu\text{m}$ in the right eye and $58.5\ \mu\text{m}$ in the left eye. The predicted residual bed thickness was $372.5\ \mu\text{m}$ in the right eye and $377.5\ \mu\text{m}$ in the left eye. The ablation was performed using a VISX Star excimer laser (VISX, Santa Clara, CA, USA). The optical zone and transitional zone were, respectively, $5.5\ \text{mm}$ and $7.0\ \text{mm}$. The surgery was uneventful. Four weeks after the LASIK procedure, the uncorrected visual acuity (UCVA) was 6/6 in both eyes. Retrospectively, the postoperative intraocular pressure measurements during follow up were 6, 8 and 7 mmHg in the right eye and 5, 7 and 6 mmHg in the left eye measured with a Goldmann applanation tonometer. The patient then missed further follow up.

Two years after LASIK the visual acuity was found to have decreased in both eyes; high, irregular astigmatism was found to be present. Rigid gas-permeable contact lenses were fitted by optometrist's order. The BCVA improved to 6/7.5 in the right eye and 6/9 in the left eye. Contact lenses were

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Figure 1. (a) Preoperative corneal topography of the right eye (OD) and left eye (OS) shows an asymmetric bow-tie pattern with inferior minus superior value of 0.408 dioptres (right eye) and 0.65 dioptres (left eye). (b) Slit-lamp photography of the right eye demonstrates diffuse corneal oedema and central epithelial cystic change with a fluid-filled interface between the flap and residual stroma, which extends nearly to the flap margin.



issued on a daily wear planned replacement basis. According to the patient's statement, there were no recent changes in contact lens wear.

Two days before the patient was referred to our hospital, he experienced a sudden loss of vision in the right eye. Examination revealed right eye UCVA of counting fingers, which did not improve with refraction. Left eye UCVA was 6/24, improving to 6/9 with rigid gas-permeable lens. The intraocular pressures were 8 mmHg in the right eye and 9 mmHg in the left eye measured with a Goldmann applanation tonometer. Slit-lamp examination of the right eye disclosed diffuse corneal oedema with central epithelial cystic change. The interface between the flap and residual stroma was open and filled with fluid. The separation extended nearly to the flap margin (Fig. 1b).

The cornea topographic image (Orbscan II, Bausch & Lomb) of the right eye could not be taken because of extensive corneal oedema. The patient had no history of eye rubbing or atopy, no signs of an allergic or vernal keratoconjunctivitis on lid examination and was otherwise well. After admission, the patient was given topical antibiotics only. Unfortunately, a shallow anterior chamber and aqueous leakage were noted the next day. Emergency penetrating kerato-

plasty was performed to cover the greatest possible area of hydrops, with a recipient size of 7.5 mm and donor size of 7.75 mm. Histopathological examination of the corneal button revealed reactive keratocytes with irregular, wrinkled collagen formation over an ablated stroma. Marked stromal oedema, focal disruption of Descemet's membrane and loss of endothelial cells were also observed (Fig. 2). After 12-month follow up, vision improved to 6/6 with correction in the right eye.

DISCUSSION

First reported in 1998, keratectasia is a rare, but widely discussed, complication of LASIK.³ In more than half of the reported cases, the onset of recognized keratectasia developed more than 1 year after LASIK.⁴ Post-LASIK keratectasia has been reported in patients with keratoconus, forme fruste keratoconus, high myopia (>8 D) and thin residual stromal bed (<250 μm), as well as in those with no other recognized risk factors.⁵ Our patient underwent uneventful bilateral LASIK 9 years prior to presentation. The amount of myopic ablation in both eyes was 4.5 D and the residual bed thickness was 372.5 μm in the right eye and 377.5 μm in the

Table 1. Histopathology of secondary keratectasia (data taken from Argento *et al.*,⁶ with permission)

Structure	Secondary keratectasia	Our patient
Epithelium	Normal	Normal
Basement membrane	Normal	Normal
Bowman's layer complex	Normal	Normal
Stroma	Irregular lamellae with fibroblastic keratocytes	Reactive keratocytes with irregular, wrinkled collagen over ablated stroma, marked oedema
Descemet's membrane	Normal	Focal disruption
Endothelium	Minimal change	Loss of endothelial cell

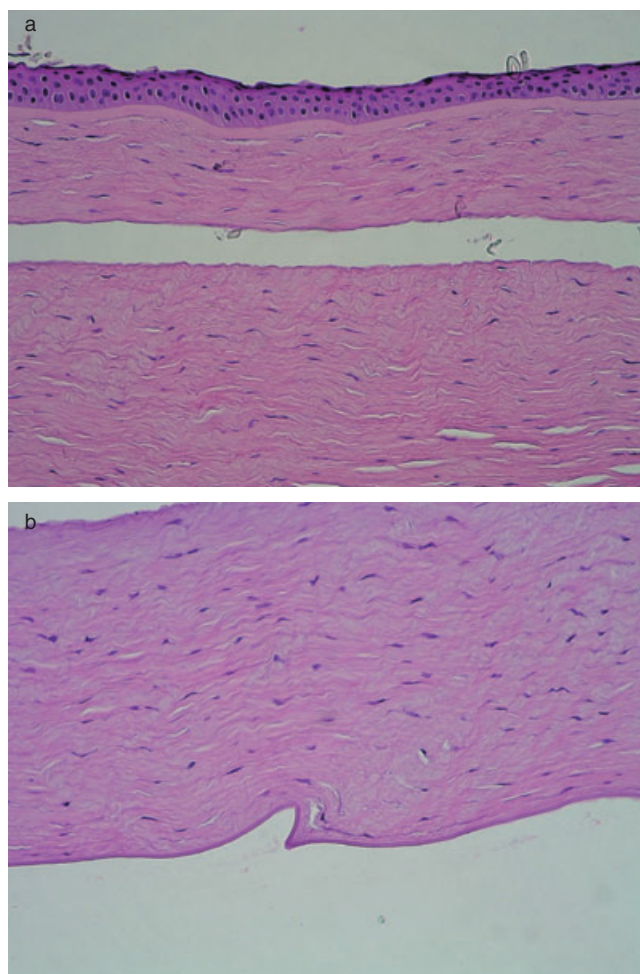


Figure 2. (a) Histopathological examination of the corneal button reveals reactive normal keratocytes with irregular, wrinkled collagen formation over an ablated stroma (haematoxylin and eosin, $\times 40$). (b) Focal disruption of Descemet's membrane and loss of endothelial cells (haematoxylin and eosin, $\times 100$).

left eye. The value of the inferior minus superior was 0.408 D in the right eye and 0.65 in the left eye. Nevertheless, keratectasia with acute corneal hydrops and perforation of the right eye then developed in this patient with no recognized risk factors.

The treatment of keratectasia is rarely an emergency, except in corneal hydrops.⁴ In more advanced keratoconus cases, acute corneal hydrops results from stromal imbibition

of aqueous through ruptures in Descemet's membrane. Spontaneous perforation in advanced keratoconus is extremely rare. In our case, the pathology showed separation of the flap and stromal bed; this was compatible with the clinical findings on slit-lamp examination. The area of perforation was not located in the pathology specimen. It is possible that poor endothelial function and disruption of Descemet's membrane allowed fluid to fill the cleft between the stroma and flap (as in the case described by Chung *et al.*²). This fluid then leaked around the flap edge or through an area of thinning in the flap rather than a full-thickness perforation.

In 2001, Argento *et al.* described the histopathological comparison between primary and secondary keratectasia.⁶ In our case, the stroma showed reactive keratocytes with irregular, wrinkled collagen formation and oedematous change. Descemet's membrane showed focal disruption, and there was a loss of endothelial cells. This finding in Descemet's membrane was not compatible with secondary keratectasia (Table 1). Although the course of the disease in our patient is similar to primary keratectasia, it is actually secondary keratectasia.

Kallinikos and Efron demonstrated that the mechanical stimulation of the corneal surface, due to the physical presence of a contact lens, induces the release of inflammatory mediators that cause keratocyte dysgenesis or apoptosis.⁷ Based on Wilson *et al.*'s hypothesis, keratoconus is an abnormality of the interactive epithelial-stromal system that causes a shift in the delicate balance between keratocyte proliferation and apoptosis.⁸ However, in our patient who wore rigid contact lens on a regular basis for about 7 years, the pathology did not show a decrease in keratocytes.

In conclusion, we cannot be certain of the mechanism of this patient's keratectasia after LASIK surgery. We can only rely on the account of preoperative measurements reporting an apparently normal preoperative corneal topography prior to LASIK. Regardless, the preoperative evaluation of a patient should accurately document family or patient history of keratoconus, history of severe eye rubbing, topographic evidence of keratoconus, history of orthokeratology or contact lens-associated corneal warpage.

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