CASE REPORT

Pressure-induced stromal keratopathy after laser in situ keratomileusis: Acute and late-onset presentations

Jesus Cabral-Macias, MD, MSc, Guillermo García-De la Rosa, MD, Diana F. Rodríguez-Matilde, MD, Ivan Daryl Vela-Barrera, MD, Jasbeth Ledesma-Gil, MD, Arturo Ramirez-Miranda, MD, Enrique O. Graue-Hernandez, MD, FACS, MSc, Alejandro Navas, MD, FACS, PhD

We present a series of 4 cases of pressure-induced stromal keratopathy after laser in situ keratomileusis (LASIK). Four patients (5 eyes) with previous LASIK presented for poor visual acuity and ocular pain because of ocular hypertension. At examination, all cases revealed corneal haze and a space filled with fluid between the surgical flap and the residual stroma. All cases were managed with topical hypotensive treatment and one of them was also treated with a valve drainage device. Topical steroids restriction was indicated in all cases. Intraocular pressure (IOP) was normalized in all cases with subsequent interface fluid resolution and significant improvement of vision in most cases. Early recognition and appropriate treatment for pressure-induced stromal keratopathy is essential to avoid complications associated with prolonged elevated IOP. It is extremely important to measure the IOP in the peripheral cornea because IOP in the central cornea can be incorrectly measured with the characteristic interface fluid developed in this entity.

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Since its introduction, laser in situ keratomileusis (LASIK) has become the most commonly performed corneal refractive procedure.[1] Postoperative LASIK management is usually based on topical corticosteroids and antibiotics to reduce inflammation and prevent infectious keratitis, respectively. It has been reported that up to 7% of the population presents with elevated intraocular pressure (IOP) because of corticosteroids application.[2]

Among interface complications, pressure-induced stromal keratopathy is a rare but severe complication after the LASIK procedure.[3–5] Pressure-induced stromal keratopathy is defined as a fluid collection in the interface associated with elevated IOP attributable to steroid use and/or corneal endothelial dysfunction.[6] It can be misdiagnosed as diffuse lamellar keratitis (DLK), central toxic keratopathy, infectious keratitis, and epithelial ingrowth, among others.[7]

We present 5 eyes (4 patients) of pressure-induced stromal keratopathy after LASIK, with acute onset in 3 eyes and chronic onset in the 2 others.

CASE REPORTS

Case 1

A 19-year-old man with history of severe inflammatory juvenile acne had uneventful LASIK in both eyes in another center. He presented to our institution 34 days after the refractive procedure for ocular pain and diminished uncorrected distance visual acuity (UDVA). He reported chronic intake of retinoic acid for his acne condition.

The patient's preoperative manifest refraction was −8.00 −1.50 × 97 in the right eye and −7.00 −2.00 × 92 in the left eye. Postoperative management was based on gatifloxacin 0.3% eyedrops 3 times a day, fluorometholone acetate 4 times a day, and sodium carboxymethylcellulose 4 times a day. Three days after LASIK, the patient reported blurred vision and stated that he had corneal flap lifting and interface irrigation in both eyes at another ophthalmological center. He was treated with topical prednisolone acetate 1.0% every 2 hours, lubricants, and topical antibiotics. Nevertheless, 5 days after lifting the flap, he was prescribed hypotensive treatment with dorzolamide hydrochloride 2.0%, brimonidine tartrate 0.25%, and timolol maleate 0.5% twice a day because of ocular hypertension, as well as loteprednol, gatifloxacin, and sodium chloride.

At examination, his UDVA was hand motion in both eyes and his central IOP determined by applanation tonometry was 10 mm Hg in both eyes; however, his peripheral IOP was 72 mm Hg in

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From the Department of Cornea and Refractive Surgery (Cabral-Macias, García-De la Rosa, Rodríguez-Matilde, Vela-Barrera, Ramirez-Miranda, Graue-Hernandez, Graue-Hernandez) and the Department of Glaucoma (Ledesma-Gil), Instituto de Oftalmología Conde de Valenciana, Mexico City, Mexico.

Corresponding author: Alejandro Navas, MD, FACS, PhD, Department of Cornea and Refractive Surgery, Instituto de Oftalmología Conde de Valenciana, Chimalpopoca 14, Cuauhtémoc 06800, Mexico City, Mexico. Email: dr.alejandro.navas@gmail.com.

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both eyes using a handheld applanation tonometer (Tono-Pen XL, Automated Ophthalmics, Inc.). Slitlamp evaluation showed a central pocket of fluid in the LASIK interface in both eyes and moderate diffuse corneal opacity (Figure 1, *A* to *D*). The central corneal thickness (CCT) measured by optical coherence tomography (OCT) (Visante, Carl Zeiss Meditec AG) was 746 μm in the right eye and 654 μm in the left eye. The central LASIK flap thickness was 117 μm in the right eye and 108 μm in the left eye and the central interface fluid thickness was 208 μm and 168 μm, respectively (Figure 1, *E* and *F*). An afferent pupillary defect was found in both eyes. Topical steroids were discontinued and the patient was treated with a load of intravenous mannitol and 250 mg of oral acetazolamide 3 times a day, topical dorzolamide hydrochloride 2.0%, brimonidine tartrate 0.25%, and timolol maleate 0.5%.

After 30 days, the patient's UDVA was 20/200 Snellen in both eyes; peripheral corneal IOP was 10 mm Hg in the right eye and 15 mm Hg in the left eye. Interface fluid in the central pocket had disappeared and corneal opacity improved (Figure 2, *A* to *D*); oral acetazolamide was discontinued.

Three months later, the patient's UDVA was 20/60 and 20/100 in both eyes, respectively. Intraocular pressure was 12 mm Hg by applanation tonometry and 14 mm Hg by handheld applanation tonometry peripherally in both eyes. He developed epithelial ingrowth and subcapsular cataracts in both eyes. A fundoscopic evaluation showed a pale optic nerve and high cup–disc ratio of 0.90 bilaterally. Automated visual fields (Humphrey Field Analyzer, Carl Zeiss Meditec AG) obtained 3 months after LASIK demonstrated dense superior and inferior arcuate defects in both eyes consistent with glaucomatous optic neuropathy (Figure 3, *A* and *B*).

Despite that the patient was treated with antiglaucomatous maximum-tolerated medical therapy, after 6 months, his IOP measured with the handheld applanation tonometer was 42 mm Hg in the right eye and 34 mm Hg in the left eye, with decreased visual acuity of 20/200 and 20/100, respectively. He had Ahmed glaucoma valve (New World Medical, Inc.) implantation in both eyes (Figure 3, *C* to *F*). We avoided the use of postoperative steroids, using subconjunctival bevacizumab and a topical nonsteroidal antiinflammatory instead. No cataract removal was performed until his last examination. At present, the patient's corrected distance visual acuity (CDVA) is 20/80 in the right eye and 20/50 in the left eye with an IOP of 18 mm Hg and 12 mm Hg, respectively. The patient was referred to a psychiatrist because he had a suicide attempt attributable to severe depression.

Case 2

A 24-year-old woman was referred to our institution for an acute-onset nongranulomatous anterior uveitis in the right eye. She reported redness, pain, and blurred vision in the right eye 6 months after uneventful bilateral LASIK. The uveitis was treated with prednisolone acetate 1.0% for 4 months. The patient's UDVA was 20/400 in the right eye and 20/25 in the left eye. The CDVA was 20/100 with a refraction of −2.00 −2.00 ×15 in the right eye and 20/20 with −0.50 −0.50 × 5 in the left eye.

Biomicroscopy revealed mild lid edema, conjunctival hyperemia, and a pocket of fluid in the interface of the corneal flap in the affected right eye (Figure 4, *A* and *B*). The left eye examination was unremarkable. Although the IOP was 17 mm Hg in the right eye and 18 mm Hg in the left eye by applanation Goldmann tonometry, the central and peripheral values were 37 mm Hg and 42 mm Hg, respectively, in the right eye when measured with the handheld applanation tonometer. The fundoscopic evaluation was not assessable in the right eye and it was normal in left eye.

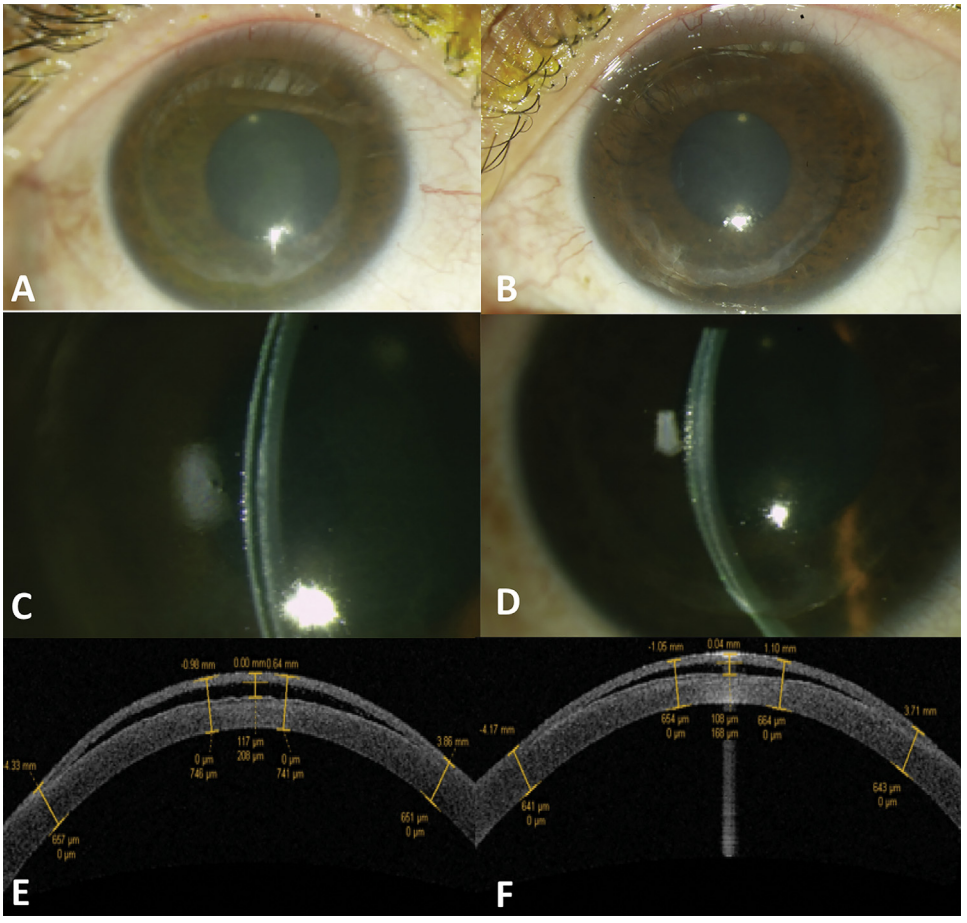

Figure 1. *A* and *B*: Right eye and left eye, respectively, in Case 1 with corneal edema involving the flap area. *C* and *D*: Clinical photographs showing an accumulation of fluid in the interface in the right eye and left eye, respectively. The pocket of fluid is more evident in the right eye. *E* and *F*: Optical coherence tomography shows a central corneal thickness of 746 μm in the right eye and 654 μm in the left eye, respectively. Central laser in situ keratomileusis flap thickness was 117 μm and 108 μm, respectively, and the central interface gap was 208 μm and 168 μm, respectively.