Published in final edited form as:

Ophthalmic Surg Lasers Imaging. 2008 ; 39(4 Suppl): S80–S82.

High-resolution Imaging of Complicated LASIK Flap Interface Fluid Syndrome

Jose Luiz Branco Ramos, MD, Sheng Zhou, MD, Christopher Yo, MD, PhD, Maolong Tang, PhD, and David Huang, MD, PhD

From the Doheny Eye Institute and Department of Ophthalmology, Keck School of Medicine of the University of Southern California, Los Angeles (JLBR, SZ, MT, DH); and the Southern California Permanente Medical Group, Baldwin Park Medical Center, Baldwin Park, California (CY).

Abstract

The authors report a case of post-LASIK interface fluid syndrome that led to epithelial ingrowth, a sequela that had not been reported to date. The interface fluid was caused by steroid-induced ocular hypertension. On post-LASIK day 49, the interface fluid, epithelial ingrowth, and noncellular reflective deposits were visualized by confocal microscopy and high-resolution Fourier-domain optical coherence tomography. No inflammatory cells or infectious organisms were seen. These high-resolution imaging technologies were useful in the noninvasive evaluation of the location and nature of flap interface pathologies at the microstructural level.

INTRODUCTION

Interface fluid syndrome is an uncommon complication of LASIK that is associated with ocular hypertension or endothelial cell dysfunction. The most commonly reported etiology is steroid-induced ocular hypertension.¹ With its characteristic appearance, including a diffuse haze and a fluid gap in the interface, interface fluid syndrome could be easily mistaken for diffuse lamellar keratitis,² leading to continued treatment with topical corticosteroids and an increase in intraocular pressure (IOP). We describe a case of interface fluid syndrome that led to epithelial ingrowth, which further complicated the clinical presentation. The complicated interface pathology that included fluid, epithelium, and haze was delineated with confocal microscopy (Confoscan4; Nidek, Japan) and a high-resolution Fourier-domain optical coherence tomography (FD-OCT) system (RTVue; Optovue, Inc., Fremont, Calif.).

CASE REPORT

A 42-year-old man underwent uncomplicated LASIK surgery of his right eye for anisometropia. The preoperative manifest refraction was $-5.00 +6.00 \times 109$ and the best spectacle-corrected visual acuity was 20/25. The IOP was 18 mm Hg. Fundus examination showed a cup/disk ratio of 0.6 with intact disk margins. The central corneal thickness was 563 μm .

One day after LASIK was performed, pinhole visual acuity was 20/25. Slit-lamp examination showed a well-aligned flap with clear interface. Topical prednisolone acetate 1% and levofloxacin 0.5% four times a day were prescribed.

One week after LASIK was performed, the patient reported eye pain and worsening vision in the right eye. The uncorrected visual acuity was 20/400, and IOP by Goldmann tonometer measured 24 mm Hg. Topical brimonidine twice a day and oral acetazolamide 500 mg daily were prescribed for 2 days. On the next day, the patient had no eye pain, and uncorrected visual acuity was 20/30 and IOP was 14 mm Hg.

Fifteen days after LASIK was performed, a mild interface haze was noted, and diffuse lamellar keratitis was suspected. Oral prednisone 50 mg daily and topical prednisolone acetate 1% hourly were prescribed. Twenty-one days after surgery, the interface haze had not improved, and the flap was lifted and irrigated. Hourly topical steroids and IOP medications were continued.

Thirty-seven days after LASIK was performed, visual acuity was count fingers in the right eye, and diffuse flap edema, interface haze, and an interface fluid layer were noted. These findings persisted until day 46.

On postoperative day 49, slit-lamp examination showed flap edema, interface fluid layer, and interface opacity (Fig. 1). IOP was 39 mm Hg as measured by a Tono-Pen (Reichert, Depew, NY) in the peripheral cornea and 3 mm Hg as measured with a Goldmann tonometer in the central cornea. Topical steroid was reduced to twice a day on suspicion of steroid-induced ocular hypertension and was discontinued 8 days later. The cornea was scanned with FD-OCT (Fig. 2) and confocal microscopy (Fig. 3).

At the last visit (5 months post-LASIK), the interface fluid has resolved, and best corrected visual acuity was 20/30. The interface haze was significantly reduced. An area of epithelial ingrowth 1×5 mm remained under the flap, and IOP was 21 mm Hg.

DISCUSSION

Interface fluid syndrome is a LASIK complication that was first reported in 1999 by Lyle and Jin.³ They described a diffuse, smudgy, nongranular haze confined to the central and paracentral areas of the LASIK interface. Commonly, cases of interface fluid syndrome are associated with steroid-induced ocular hypertension; they do not respond to topical corticosteroids, and they typically resolve with discontinuation of topical corticosteroids and application of IOP-lowering medications.^{1,4,5} Interface fluid syndrome is associated with falsely low central IOP measurements due to cushioning by the fluid-filled pocket. A more accurate IOP reading can be obtained by applying the Goldmann or Tono-Pen tonometer peripheral to the LASIK flap.⁶

Our case shares the common features described above, with the additional complication of epithelial ingrowth. The microstructure of these pathologies was imaged with both FD-OCT and confocal microscopy.

OCT is a noncontact, cross-sectional imaging technology⁷ that had been used previously to image fluid collection as a nonreflective space in interface fluid syndrome.⁸ The RTVue FD-OCT uses an 830-nm wavelength, performs 26,000 axial scans per second, and has an axial resolution of 5 μ m in tissue. The RTVue was originally developed for retinal imaging. A corneal adaptor module (CAM) was used to allow anterior segment imaging. The CAM includes adaptor lenses to produce a telecentric scan geometry for corneal imaging and special software to dewarp the effects of index transition. The higher resolution and definition of the RTVue-CAM allowed us to visualize several regions and layers in the interface (Fig. 2). The area of epithelial ingrowth had a zone of medium internal reflectivity and a zone of low internal reflectivity, which correspond to the opaque and clear areas on slit-lamp examination (Fig. 1). The fluid space had no reflectivity. There were thin layers of medium reflectivity that

sandwiched both the epithelial ingrowth and the interface fluid. These layers correspond to the granular haze observed on slit-lamp examination.

Confocal microscopy performed at the inferior paracentral cornea confirmed the presence of epithelial cells in the interface (Fig. 3A). By through-focusing, we determined that amorphous reflective material was present below the interface epithelium (Fig. 3B). No inflammatory cells were seen.

These two high-resolution imaging modalities were complementary. Confocal microscopy has the resolution to identify cells but has a restricted submillimeter scan width. OCT has the wider scan range to allow visualization of the distribution of different pathologies. Use of these noninvasive imaging techniques, however, does not resolve all questions. Histopathology is probably needed to further elucidate the composition of the amorphous reflective material in the haze and the clear and opaque areas of epithelial ingrowth.

Epithelial ingrowth is a unique feature of our case that had not been previously described as a sequelae of interface fluid syndrome. We hypothesize that the interface fluid tents up the flap and may have created a space at the flap edge and interface for epithelial ingrowth.

In summary, we described the appearance of epithelium, haze, and fluid in the flap interface as visualized by both high-resolution FD-OCT and confocal microscopy. This knowledge may help clinicians differentiate between inflammation, haze, epithelium, and fluid in the interface and aid in the management of post-LASIK complications.

Acknowledgements

Supported by NIH grants R01 EY018184 and P30 EY03040; a grant from Optovue, Inc.; and an unrestricted grant from Research to Prevent Blindness, Inc. Drs. Tang and Huang receive grant support and patent royalty from Optovue, Inc.; Dr. Huang also received stock options and travel support from Optovue.

References

1. Dawson DG, Schmack I, Holley GP, Waring GO III, Grossniklaus HE, Edelhauser. Interface fluid syndrome in human eye bank corneas after LASIK: causes and pathogenesis. *Ophthalmology* 2007;114:1848–1859. [PubMed: 17908592]
2. Fogla R, Rao SK, Padmanabhan P. Interface fluid after laser in situ keratomileusis. *J Cataract Refract Surg* 2001;27:1526–1528. [PubMed: 11566545]
3. Lyle WA, Jin GJ. Interface fluid associated with diffuse lamellar keratitis and epithelial ingrowth after laser in situ keratomileusis. *J Cataract Refract Surg* 1999;25:1009–1012. [PubMed: 10404381]
4. Rehany U, Bersudsky V, Rumelt S. Paradoxical hypotony after laser in situ keratomileusis. *J Cataract Refract Surg* 2000;26:1823–1826. [PubMed: 11134885]
5. Parek JG, Raviv T, Speaker MG. Grossly false applanation tonometry associated with interface fluid in susceptible LASIK patients. *J Cataract Refract Surg* 2001;27:1143–1144. [PubMed: 11530785]
6. Belin MW, Hannush SB, Yau CW, Schultze RL. Elevated intraocular pressure-induced interlamellar stromal keratitis. *Ophthalmology* 2002;109:1929–1933. [PubMed: 12359617]
7. Huang D, Swanson EA, Lin CP, et al. Optical coherence tomography. *Science* 1991;254:1178–1181. [PubMed: 1957169]
8. Wirbelauer C, Pham DT. Imaging of interface fluid after laser in situ keratomileusis with corneal optical coherence tomography. *J Cataract Refract Surg* 2005;31:853–856. [PubMed: 15899468]

Figure 1.

Slit-lamp photography on post-LASIK day 49 showed flap edema, interface fluid, and several zones of opacity. Inferiorly, there is a dense opacity interspersed with streaks and pearls of clear material. The central and superior areas of fluid accumulation are associated with scattered granular haze. The line and arrow indicate the OCT scan length and direction.

Figure 2.
OCT (6.0×1.9 mm) showing areas of epithelial ingrowth and fluid under the LASIK flap.

Figure 3. Confocal microscopic images of the flap interface showing (A) highly reflective extracellular material and (B) disorganized epithelial cells.