Letters

OBSERVATION

Delayed-Onset Interface Fluid Syndrome After Laser-Assisted In Situ Keratomileusis Secondary to Descemet Stripping Automated Endothelial Keratoplasty

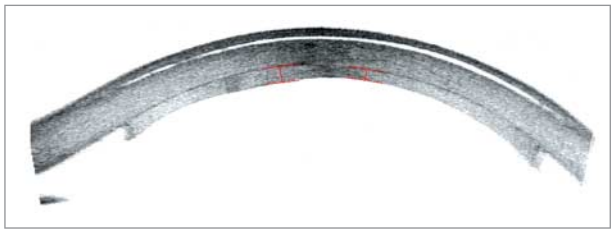
Descemet stripping automated endothelial keratoplasty (DSAEK) has become the preferred surgical treatment for patients with endothelial dysfunction.[1] Interface fluid syndrome (IFS) is an uncommon complication after laser-assisted in situ keratomileusis (LASIK) in which diffuse pockets of fluid collect within the interface. Typical cases of IFS are associated with steroid-induced intraoperative pressure (IOP) increase after LASIK.[2,3] We describe a patient who had undergone anterior chamber phakic intraocular lens (IOL) implantation and LASIK for high myopia. Five years later, she required DSAEK owing to endothelial dysfunction. She presented with IFS in the immediate postoperative period.

Report of a Case | A woman in her 30s with high myopia in her left eye underwent surgery elsewhere in 2006 with anterior chamber phakic IOL (I-Care; Corneal) implantation in her left eye, and 3 months later she underwent LASIK to refine mild residual myopia. Preoperative refractive data were not available when we examined the patient, but she stated she had corrected distance visual acuity of 20/40 OS. The patient explained that 4 years later, the original surgeon explanted the phakic IOL owing to damage to the endothelium. The patient presented to our clinic in 2011 with corneal edema, corrected distance visual acuity of hand motions, and IOP of 10 mm Hg.

We elected to perform a triple procedure (phacoemulsification, IOL implantation, and DSAEK) as previously described.[4] We implanted a donor disc of 8.5 mm.

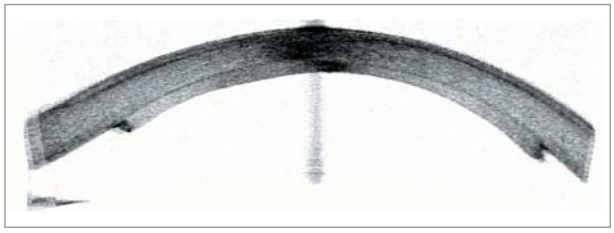
Ten days postoperatively, the patient had painless visual blurring in the left eye and IOP of 16 mm Hg as measured by Goldmann applanation tonometry. Slitlamp examination revealed a diffuse cloudy interface and flap edema, and digital IOP was clearly high. There was no anterior chamber reaction or conjunctival hyperemia. After LASIK, the LASIK flap interface always provides a potential space for fluid accumulation with high IOP and corneal edema. When IFS is present, all forms of applanation tonometry are inaccurate and give a lower measurement owing to the compressible fluid-filled interface; optical coherence tomography and/or a careful slitlamp examination is necessary to make an accurate diagnosis. Based on a working diagnosis of IFS due to steroid-responsive ocular hypertension, time-domain anterior segment optical coherence tomography (Visante; Zeiss) was performed. It confirmed the diagnosis of IFS (**Figure 1**). To control the IOP, topical dexamethasone sodium phosphate eyedrops were discontinued and systemic (oral) dorzolamide hydrochloride (250 mg 3 times daily) was prescribed, along with brimonidine tartrate (2 mg/mL) plus timolol maleate (5 mg/mL) twice daily. Two weeks later, fluid collection and flap edema had resolved on slitlamp examination. These findings were confirmed on optical coherence tomography (**Figure 2**). Oral dorzolamide was discontinued.

Figure 1. Interface Fluid Syndrome After Descemet Stripping Automated Endothelial Keratoplasty in a Patient Who Has Undergone Laser-Assisted In Situ Keratomileusis



Anterior segment optical coherence tomographic image (Visante; Zeiss) 10 days after Descemet stripping automated endothelial keratoplasty. A space without a signal between the laser-assisted in situ keratomileusis flap and the anterior stroma of the recipient cornea is clearly seen. The donor disc is well attached. Red bars indicate the thickness of the disc used in Descemet stripping automated endothelial keratoplasty.

Figure 2. Complete Resolution of Interface Fluid Syndrome After Hypotensive Treatment



Anterior segment optical coherence tomographic image (Visante; Zeiss) 24 days after Descemet stripping automated endothelial keratoplasty. Complete resolution of fluid accumulation in the laser-assisted in situ keratomileusis interface is shown (after the patient received hypotensive agents for 14 days).

Two and a half years after DSAEK, IOP was maintained at 12 mm Hg, the cornea was clear, and corrected distance visual acuity was 20/50 with a manifest refraction of −1.25 − 1.00 × 85°.

Discussion | Interface fluid syndrome is described as a flap-related complication of LASIK that is frequently caused by a steroid-induced IOP increase. An interface inflammatory reaction may also develop years after LASIK, which is considered late-onset diffuse lamellar keratitis.

To our knowledge, this is the first case report of IFS in a patient who underwent DSAEK 5 years after LASIK. Interestingly, in this case, fluid accumulation was seen in the interface of the LASIK flap and not in the potential space between

the donor disc and the recipient posterior stroma. This observation may suggest that the stromal interface of the LASIK wound has weaker cohesive tensile strength than that produced after DSAEK in the interface between the anterior surface of the donor disc and the posterior recipient stroma.

Ophthalmologists should consider the possibility of IFS after endothelial keratoplasty in a patient who has undergone LASIK previously.

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