

# Persistent interface fluid syndrome

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We present an unusual case of persistent interface fluid that would not resolve despite normal intraocular pressure and corneal endothelial replacement with Descemet-stripping endothelial keratoplasty. Dissection, elevation, and repositioning of the laser in situ keratomileusis flap were required to resolve the interface fluid. Circumferential corneal graft–host margin scar formation acting as a mechanical strut may have been the cause of the intractable interface fluid.

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Interface fluid syndrome (IFS) is a rare but serious condition that occurs in eyes that have had lamellar corneal refractive surgery. Accumulation of fluid within a laser in situ keratomileusis (LASIK) interface has most often been attributed to increased intraocular pressure (IOP) from steroid-induced glaucoma,<sup>1–6</sup> but it has also been described in eyes with damaged or compromised endothelial cell function.<sup>7–11</sup> The IFS usually resolves with normalization of the IOP or return of normal endothelial cell function through pharmacological or surgical means.<sup>1,2,4–6,8,9</sup>

We present a case of persistent interface fluid in a patient who had had penetrating keratoplasty (PKP) followed by LASIK and LASIK enhancement procedures. The interface fluid persisted despite a normal IOP and a subsequent endothelial keratoplasty. It was ultimately resolved by flap elevation and repositioning.

## CASE REPORT

A 65-year-old woman had multiple surgical procedures in her right eye over a 13-year period: myopic automated lamellar keratoplasty in both eyes in 1993, LASIK in the right eye in 1997, phacoemulsification and intraocular lens implantation in the right eye in 1998, PKP for progressive corneal ectasia in 2002, LASIK (−12.25 +6.00 × 55) in 2004, and LASIK enhancement (−2.25 +4.25 × 105) in 2005. The LASIK keratotomy diameter was larger than the diameter of the corneal graft. In 2006, a graft rejection was diagnosed and the patient was placed on frequent doses of

topical prednisolone acetate 1%. After several months with no improvement in the clinical status, she was referred to a university corneal service and interface fluid secondary to steroid-induced glaucoma was diagnosed. A review of the patient's records revealed a peripheral corneal IOP measurement of 23 mm Hg. Topical steroids were discontinued, and she was started on brimonidine 0.1% 3 times a day and timolol 0.5% twice a day in the right eye and referred back to her primary ophthalmologist. After several weeks without improvement, she was referred to our clinic.

At the time of our examination, the patient was using the previously prescribed glaucoma medications. Visual acuity was counting fingers at 3 feet in the right eye and 20/25 in the left eye. Slitlamp examination showed a central pocket of fluid in the LASIK interface in the right eye that was isolated inside the corneal graft–host margin (Figure 1). Central corneal thickness measured by Visante OCT (optical coherence tomography) (Carl Zeiss Meditec) was 800 μm. Central LASIK flap thickness was 126 μm, central posterior stromal thickness was 430 μm, and central interface fluid thickness was calculated at 244 μm. There was no evidence of keratic precipitates. The temporal corneal periphery measured by Tono-Pen XL (Reichert, Inc.) was 21 mm Hg. The central corneal endothelial cell count (Topcon SP-2000P noncontact specular microscope, Topcon America Corp.) was not recordable in the right eye; it was 1895 cells/mm<sup>2</sup> in the left eye. Confocal microscopy was not available.

Interface fluid syndrome secondary to endothelial failure was diagnosed, and Descemet-stripping endothelial keratoplasty (DSEK) was performed in the right eye several weeks later. At the time of the DSEK procedure, full-thickness fenestrations were made in the cornea to ensure removal of any fluid from the interface between the posterior lamellar graft and the recipient cornea, in addition to possibly facilitating drainage of the LASIK interface fluid.<sup>12</sup> Two of 4 fenestrations were through regions of the cornea with underlying LASIK interface fluid. On the first postoperative day, 2 interfaces of fluid were present secondary to incomplete adherence of the endothelial graft (Figure 2). The posterior graft fully adhered to the recipient cornea after 1 week, and the posterior interface fluid was absorbed. Three weeks after the DSEK procedure, the anterior fluid pocket appeared unchanged; this was confirmed by OCT.

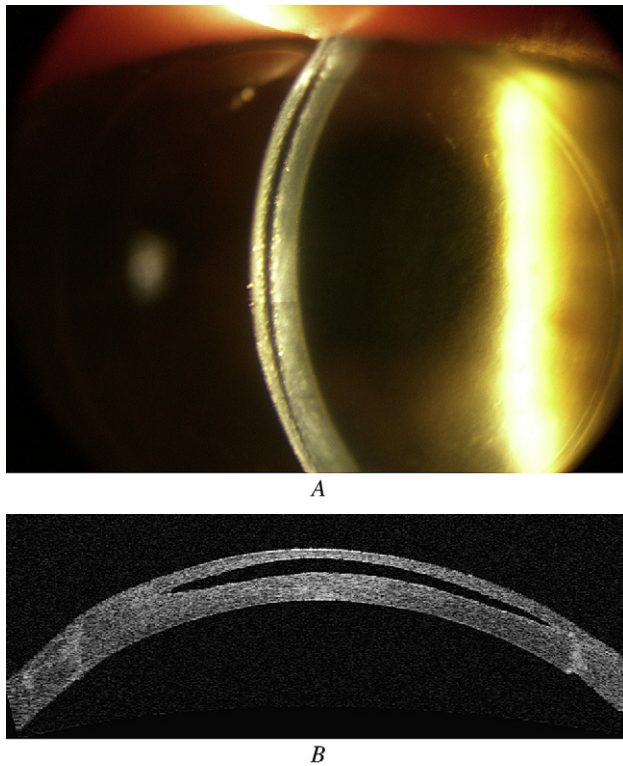
At a biannual DSEK forum, the case was discussed and flap elevation and repositioning were recommended to facilitate resolution of the interface fluid. Elevation of the LASIK flap was complicated by a significant circumferential

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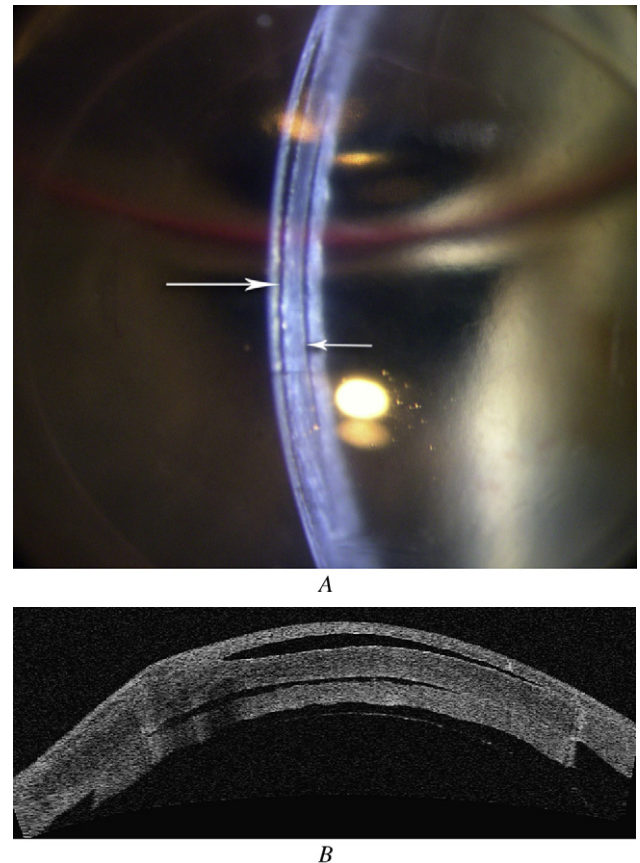
**Figure 1.** A: Fluid-filled pocket in the anterior corneal stroma. B: Horizontal OCT of the same area shows LASIK interface fluid isolating within the previous corneal graft-host margin.

adhesion at the junction of the penetrating graft-host margin and the LASIK keratectomy. After aggressive lysis of the scar with a Kritzinger-Updegraff flap elevator (AE-2835, ASICO), the flap was elevated and repositioned. No epithelium was present in the LASIK interface.

On the first postoperative day, interface fluid was not present (Figure 3) and remained resolved. Significant ground glass interface haze prevented improvement in vision to better than 20/100. Two months after the LASIK flap repositioning, PKP was performed. Eight months after the repeat PKP, the patient was correctable to 20/20<sup>+</sup> with a rigid gas-permeable contact lens.

## DISCUSSION

The importance of recognizing and treating IFS secondary to steroid-induced glaucoma cannot be over-emphasized. Falsely measured low IOP, from readings taken overlying the interface fluid, may cause a delay in diagnosis and treatment, resulting in significant visual loss.<sup>3,13</sup> In addition, this clinical setting may be exacerbated by an inaccurate diagnosis of diffuse lamellar keratitis and an increase in the dose of topical steroids.<sup>5,6</sup> Measuring the IOP in the corneal periphery, outside the area of interface fluid, will reveal a more accurate assessment of the true elevated IOP and aid in the diagnosis. When IFS develops despite relatively normal peripheral IOP measurements,

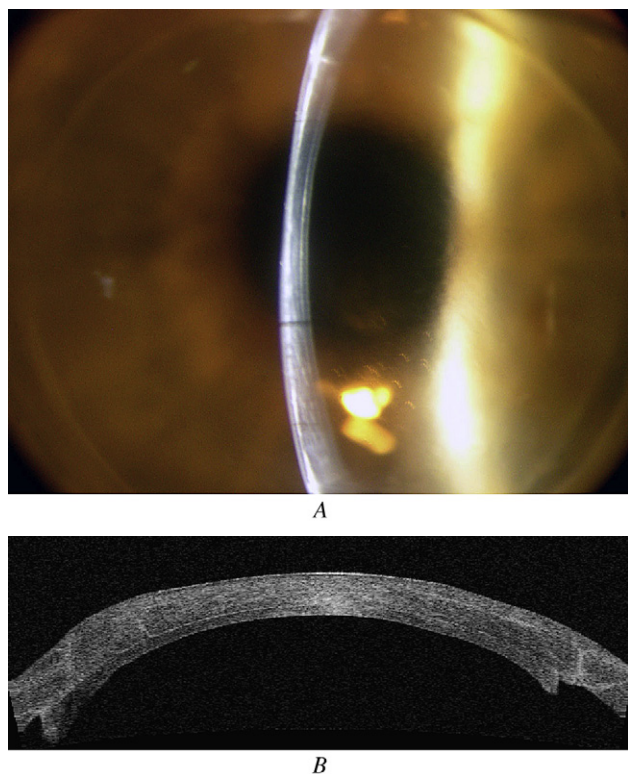


**Figure 2.** A: One day after DSEK in the right eye, 2 fluid-filled spaces corresponding to the original LASIK interface fluid (*large arrow*) and the interface between the posterior corneal surface and the endothelial graft (*small arrow*) are seen. B: Horizontal OCT shows incomplete adherence of the thickened endothelial graft.

the corneal endothelium becomes the suspect for the mechanism of interface fluid accumulation.

In this case, the lack of resolution of interface fluid despite cessation of topical steroids and the introduction of glaucoma medications suggested corneal endothelial failure as the cause of the interface fluid. This diagnosis was supported by an inability to measure the density of corneal endothelial cells and the likelihood that an eye that had had PKP would have a low cell count that might be at risk for endothelial failure. Theoretically, replacing the corneal endothelium with a DSEK procedure should restore adequate endothelial function and resolve the interface fluid.

The inability to clear the interface fluid despite a normal IOP and adequate endothelial function (demonstrated by adhesion of the endothelial keratoplasty to the posterior corneal surface over 1 week) suggested an additional mechanism for the persistent interface fluid. During elevation of the LASIK flap, an adherent scar was present at the intersection of the previous PKP graft-host margin and the LASIK keratectomy



**Figure 3.** A: One day after LASIK flap dissection, elevation, and repositioning, the LASIK interface fluid has resolved. B: Horizontal OCT confirms complete resolution of the interface fluid.

interface. This circumferential scar may have acted as a strut or “flying buttress” that secured the peripheral flap in place and prevented resolution of the interface fluid. A scenario of interface fluid accumulation by steroid glaucoma or corneal endothelial failure and subsequent circumferential scar formation (prior to resolution of the causative agent for the interface fluid) could explain a mechanism for IFS that would not resolve despite normal IOP and a functioning endothelium. Once the fluid accumulated and the LASIK flap changed its contour to accommodate the additional fluid, adherence of the peripheral flap with a circumferential scar at the graft-host junction would create a potential space that would not resolve without lysis of the scar and repositioning of the LASIK flap. This mechanism was perhaps validated by the complete and sustained resolution of the interface fluid immediately after lysis and repositioning.

Resolution of IFS will ordinarily occur with normalization of IOP or return of endothelial function without the need for flap lifting and repositioning. Although one of the earliest reports of IFS from steroid glaucoma resolved with flap elevation, it was performed simultaneously with initiation of glaucoma therapy and occurred in an eye that had LASIK alone without previous PKP.<sup>14</sup> This suggests that resolution

of the fluid was secondary to treatment of the elevated IOP rather than to drainage of the fluid that resulted from elevating the flap.

To our knowledge, persistent IFS requiring flap dissection, elevation, and repositioning has not been described and may be limited to corneas that have had PKP followed by LASIK and subsequently develop IFS. Persistent IFS may also result from significant circumferential scarring at the edge of a LASIK flap, without the contribution of the scar from a graft-host margin.

Our patient ultimately required a repeat PKP because of the delay in treatment of the IFS and the poor visual acuity that resulted from visually significant interface haze presumed secondary to keratocyte hydropic degeneration.<sup>15</sup> More than 5 months transpired between the time the IFS likely arose and the point at which it resolved by means of flap elevation. In hindsight, a prompt diagnosis of persistent IFS and an attempt at resolution with flap repositioning, prior to endothelial replacement, might have resulted in a better visual outcome without the need for PKP. We hope awareness of this variant of IFS will improve the management of patients who present with this condition.

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