DMEK for the treatment of interface fluid syndrome secondary to failed DSAEK graft: A case report and review of the literature

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A 52-year-old patient presented with LASIK interface fluid accumulation and a non-functioning primary DSAEK graft. Past ophthalmic history was relevant for: (1) phakic intraocular lens (PIOL) implantation with later refinement by LASIK; (2) combined PIOL explantation and refractive lens exchange due to corneal decompensation; and (3) primary DSAEK due to corneal endothelial failure. Aggressive postoperative IOP control is warranted to decrease the risk of interface fluid recurrence and damage to the optic nerve. Studies with larger patient numbers are encouraged to ascertain the role of EK for this indication.

1. Introduction

Interface fluid syndrome (IFS) is a rare but potentially vision-threatening complication of LASER in situ keratomileusis (LASIK), characterized by the accumulation of fluid in the flap interface. It was traditionally known as pressure-induced stromal keratitis (PISK), but IFS is now more widely used because: 1) although a rise in intraocular pressure (IOP) may be the main mechanism, interface fluid accumulation may occur in eyes with corneal endothelial cell failure without high intraocular pressure (IOP), and 2) the term keratitis is a misnomer, as there is no actual inflammation of keratocytes.3

In cases of IFS related to increased IOP, management generally consists of ocular hypotensive drugs and cessation of topical corticosteroids (CS). However, corneal transplantation may be needed when corneal endothelial failure is the main mechanism. To date, reports on endothelial keratoplasty (EK) for this indication are scarce.

We present a complex case of LASIK interface fluid syndrome (IFS) managed with Descemet membrane endothelial keratoplasty (DMEK), and provide a literature review on EK for the management of IFS.

ABSTRACT

Purpose: To report a case of Descemet membrane endothelial keratoplasty (DMEK) for the management of post-laser in situ keratomileusis (LASIK) interface fluid syndrome (IFS) secondary to failed Descemet stripping automated endothelial keratoplasty (DSAEK) graft, and to provide a literature review on endothelial keratoplasty (EK) for this indication.

Observations: A 52-year-old patient presented with LASIK interface fluid accumulation and a non-functioning primary DSAEK graft. Past ophthalmic history was relevant for: (1) phakic intraocular lens (PIOL) implantation with later refinement by LASIK; (2) combined PIOL explantation and refractive lens exchange due to accelerated endothelial cell loss (ECL); (3) primary DSAEK due to corneal decompensation.

A secondary EK graft (DMEK) was performed, and the patient was prospectively followed for 6 months (M6). DMEK surgery was uneventful, without postoperative graft detachment. Corneal clearing and resolution of interface fluid accumulation occurred during the first postoperative month. Best-corrected visual acuity (BCVA) improved from 20/800 Snellen to 20/25 Snellen at 3-month follow-up, remaining stable at M6. Due to a persistent rise in intraocular pressure (IOP), the patient underwent uneventful non-penetrating deep sclerectomy 2 months after DMEK, with controlled IOP and without accelerated ECL.

Conclusions and Importance: DMEK is feasible, effective, and safe in the management of IFS in cases where corneal endothelial failure plays a major role, even in complex eyes with previous EK grafts. Aggressive postoperative IOP control is warranted to decrease the risk of interface fluid recurrence and damage to the optic nerve. Studies with larger patient numbers are encouraged to ascertain the role of EK for this indication.

Abbreviations: DMEK, Descemet membrane endothelial keratoplasty; LASIK, LASER in situ keratomileusis; IOP, Intraocular pressure.

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2. Case report/findings

A patient who underwent DMEK to manage IFS and was prospectively followed for 6 months after DMEK. A 52-year-old woman presented to our Cornea and Refractive Surgery Unit in May 2021 for decreased visual acuity in her left eye (OS). The patient’s ophthalmic medical history showed a combination of bilateral implantation of an angle-supported anterior chamber phakic intraocular lens (PIOL) plus laser in situ keratomileusis (LASIK) in 2006, and PIOL explantation plus laser in situ keratomileusis (LASIK) in 2008 due to a failed DSAEK graft that was refractory to multiple medical and surgical therapies, including flap elevation and suturing.

Upon first presentation at our institution, best-corrected visual acuity (BCVA) was 0.80 logMAR (20/20 Snellen) in OD and 1.6 logMAR (20/800 Snellen) in OS. Diffuse corneal stromal edema with epithelial bullae due to a failed DSAEK graft was observed, with limited visualization of the anterior chamber (AC) details. Although IOP was 12 mmHg with Goldmann applanation tonometry (GAT), glaucomatous optic neuropathy was observed, with significant peripapillary retinal nerve fiber layer (pRNFL) thinning on optical coherence tomography (OCT) and corresponding visual field defect on static automated perimetry testing.

Anterior segment OCT (AS-OCT) scans (provided by the patient) revealed a thickened corneal stromal bed and presence of fluid in the LASIK flap interface that was refractory to multiple medical and surgical therapies, including flap elevation and suturing.

Upon presentation to our institution, best-corrected visual acuity (BCVA) was 0.80 logMAR (20/20 Snellen) in OD and 1.6 logMAR (20/800 Snellen) in OS. Diffuse corneal stromal edema with epithelial bullae due to a failed DSAEK graft was observed, with limited visualization of the anterior chamber (AC) details. Although IOP was 12 mmHg with Goldmann applanation tonometry (GAT), glaucomatous optic neuropathy was observed, with significant peripapillary retinal nerve fiber layer (pRNFL) thinning on optical coherence tomography (OCT) and corresponding visual field defect on static automated perimetry testing.

The decision was to first perform a DMEK. Under local anesthesia, removal of the failed DSAEK donor graft was performed under air. Descemetorhexis was not required, but the recipients’ posterior stroma was explored for irregularities and remnants or tags using gentle scraping with our spatula. An 8.0-mm DMEK graft (donor graft endothelial cell density = 3000 cells/mm²) was inserted using the technique we have previously described, and attached to the host posterior stroma with sulphur hexafluoride 20% (SF6) endotamponade. The postoperative treatment was as previously described for secondary EK.

The DMEK graft was completely attached in the early postoperative period, without early perioperative complications including need for re-bubbling and IOP spikes. However, the patient developed a significant rise in IOP of above 30 mmHg 2 months after the surgery, which lasted more than three weeks despite ocular hypotensive drugs (topical and systemic).

Table 1
Published reports on endothelial keratoplasty for the treatment of LASIK interface fluid syndrome.

| Authors [ref] | Journal (year) | Eyes (n) | EK technique | F-U | Outcome |
|--------------|----------------|---------|--------------|-----|---------|
| Hoffmann et al. 4 | J Cataract Refract Surg (2008) | 1 | DSEK | 3 months | DSEK insufficient to solve fluid graft detachment; CME treated operatively |
| Luceri et al. 5 | Cornea (2016) | 1 | DMEK | 6 months | Uneventful DMEK; IFS resolved 1 month post-operatively |
| Shajari et al. 6 | J Refract Surg (2017) | 1 | DMEK | 6 months | Partial GD requiring rebubbling + CME treated topically. |
| Srirampuretal. 7 | Ind J Ophthalmol (2019) | 1 | UT-DSEK | 6 months | Uneventful UT-DSEK; IFS resolved 6 months post-operatively |
| Galvís et al. 8 | Saudí J Ophthalmol (2019) | 1 | DMEK | 2 months | Uneventful DMEK; IFS resolved 5 days post-operatively |
| Wolf et al. 9 | J Refract Surg Case Rep (2021) | 1 | Secondary DSAEK (after failed DMEK which induced IFS) | 6 weeks after secondary EK | Uneventful DSAEK; IFS resolved 1 week post-operatively |
| Our study | – | 1 | Secondary DMEK (after failed DSAEK which induced IFS) | 6 months after secondary EK | Uneventful DMEK; IFS resolved in the first postoperative month |

Legend: LASIK – laser in situ keratomileusis; DSEK – Descemet stripping endothelial keratoplasty; DMEK – Descemet membrane endothelial keratoplasty; IFS – interface fluid syndrome; GD – graft detachment; CME – cystoid macular edema; DSAEK – Descemet stripping automated endothelial keratoplasty; UT-DSEK – ultra-thin DSAEK.
systemic), leading to the decision to perform a non-penetrating deep sclerectomy (NPDS). No major perioperative or postoperative complications occurred after NPDS, and IOP measured by GAT was 6 mmHg 6 months after DMEK (under maintenance of topical CS treatment and without need for hypotensive drops), without progression of pRNFL thinning on OCT.

BCVA improved to 0.15 logMAR (20/32 Snellen) at the one-month postoperative visit, and uncorrected distance visual acuity was 0.1 logMAR (20/25 Snellen) at the 3-month follow-up, remaining stable at the last follow-up, 6 months after DMEK. On slit-lamp examination a clear cornea was observed, with an obvious thinning of the corneal stromal bed (Fig. 1B); central corneal thickness after DMEK decreased from 567 μm to 481 μm, and AS-OCT confirmed resolved interface fluid accumulation (Fig. 1C). Final postoperative CEC density was 2457 cells/mm² (18% CEC loss).

3. Discussion

Our case highlights the two main proposed mechanisms of IFS.1,4 We postulate the patient developed IFS following DSAEK due to a combination of an individual CS-response and a thick, non-functioning DSAEK graft (corneal endothelial failure). The IOP measurements at the time of diagnosis may have been falsely low, as may occur in patients with IFS after myopic LASIK. The fact that the patient developed a significant IOP-CS response after DMEK requiring glaucoma surgery is indirect evidence of the putative role of IOP in our case.

DMEK was successful in our case, in line with the previously published reports on EK for the treatment of IFS (including DMEK, DSAEK or ultra-thin DSAEK) suggesting that DMEK effective for LASIK IFS with an associated endothelial pump failure.4,9 Although the published evidence is limited and consists only of a few number of cases, taken altogether they suggest that resolution of interface fluid is usually fast after EK, with a success rate of 85.7% and with very few complications (Table 1). Only one of the reported cases showed a DSAEK and flap elevation failure ending with a full-thickness keratoplasty.4

We also highlight the surgical complexity of this procedure because of the poor visualization of the AC details, which makes the manipulation and attachment of the DMEK graft surgically challenging. However, DMEK can and should be considered in cases of endothelial decompensation or previous EK graft failure, including complex eyes, as we have previously reported.10 Finally, we emphasize that careful management of IOP in the early postoperative period in any case but especially after lamellar surgery is important, to avoid the appearance or recurrence of flap interface fluid accumulation and to prevent the possible secondary damage of the optic nerve.

4. Conclusions

In conclusion, DMEK is a surgically feasible and effective procedure for the management of the post-LASIK IFS, even in complex eyes. Further studies with larger sample sizes are needed to ascertain the efficacy and safety of this technique.

Patient consent
Informed written consent was obtained from the patient. This report does not contain any personal information that could lead to the identification of the patient.

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Conflicts of interest
The authors have no financial disclosures (NMC, EA, RP, JPC, JLG).

Authorship
All authors attest that they meet the current ICMJE criteria for Authorship.

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References
1. Dawson DG, Schmack I, Holley GP, Waring 3rd GO, Grossniklaus HE, Edelhauser HF. Interface fluid syndrome in human eye bank corneas after LASIK: causes and pathogenesis. Ophthalmology. 2007;114(10):1848–1859. https://doi.org/10.1016/j.ophtha.2007.01.026.
2. Guehl JL, Morral M, Gris O, Elies D, Manero F. Bimanual technique for insertion and positioning of endothelium- Descemet membrane graft in Descemet membrane endothelial keratoplasty. Cornea. 2013;32(12):1521–1526. https://doi.org/10.1097/ICO.0b013e3182923aee.
3. Moura-Coelho N, Cunha JP, Morral M, Gris O, Manero F, Guehl JL. Secondary endothelial keratoplasty-A narrative review of the outcomes of secondary corneal endothelial allografts. Transplantation. 2021;105(12):e347–e345. https://doi.org/10.1097/TP.0000000000002795.
4. Hoffman RS, Fine IH, Packer M. Persistent interface fluid syndrome. J Cataract Refract Surg. 2008;34(8):1405–1408. https://doi.org/10.1016/j.jcrs.2008.03.042.
5. Luceri S, Baksoselah Z, Iyus A, Baydoun L, Mellges GR. Interface fluid syndrome after laser in situ keratomileusis (LASIK) because of fuchs endothelial dystrophy reversed by Descemet membrane endothelial keratoplasty (DMEK). Cornea. 2016;35(12):1658–1661. https://doi.org/10.1097/ICO.0000000000000971.
6. Shajari M, Rafiezadeh F, Pavlovic I, Kubiak KB, Kohnen T, Schmack I. Management of interface fluid syndrome after LASIK by Descemet membrane endothelial keratoplasty in a patient with fuchs corneal endothelial dystrophy. J Refract Surg. 2017;33(5):347–350. https://doi.org/10.3928/1081597X-20170210-01.
7. Sirrampur A, Kalwad A, Mansoori T, Agraharam S. Reversal of laser in situ keratomileusis interface fluid after Descemet stripping automated endothelial keratoplasty for pseudophakic bullous keratopathy. Indian J Ophthalmol. 2019;67(10):1740–1742. https://doi.org/10.4103/ijo.IJO_227_19.
8. Galvis V, Berrospi BD, Tello A, Santealla G. Interface Fluid Syndrome (IFS) following Toxic Anterior Segment Syndrome (TASS): not related to high intraocular pressure but to endothelial failure. Saudi J Ophthalmol. 2019;33(1):88–93. https://doi.org/10.1016/j.sjopt.2018.06.003.
9. Wolf BJ, Ma L, Batta P. Interface fluid syndrome after Descemet membrane endothelial keratoplasty. J Refract Surg Case Rep. 2021;11):e15–e18.
10. Moura-Coelho N, Cunha JP, Amich N, Gris O, Manero F, Guehl JL. Secondary DMEK after failed DSEK graft in complex eyes – retrospective case series [serial online]. J EuCornea. September 2021:2022. Accessed January 10, 2022.

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