Epithelial downgrowth after Descemet-stripping automated endothelial keratoplasty

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A 66-year-old man presented with decreased vision and corneal edema after 2 failed Descemet-stripping automated endothelial keratoplasty (DSAEK) graft procedures in the left eye. An uneventful third DSAEK procedure combined with anterior vitrectomy through the previous limbal wound was performed. Postoperative recovery was uneventful. Histopathology of the excised failed graft revealed conjunctival epithelium on the posterior surface of the tissue. At 1 year, the endothelial cell count was 1997 cells/mm^2 and the uncorrected visual acuity was 20/20^{-2}. At 18 months, the graft remained clear with no signs of epithelial downgrowth. Clinicians should be aware that epithelial downgrowth can occur following DSAEK surgery. Fortunately, excision of the prior DSAEK graft with removal of the active epithelial membrane appears to have been a successful treament in this patient.

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Epithelial downgrowth is a rare but serious consequence following compromise of the ocular integrity by penetrating trauma or intraocular surgery. Historically, epithelial downgrowth was described most commonly after cataract extraction.[1] Epithelial downgrowth is also well documented following penetrating keratoplasty (PKP) as well as less invasive ocular surgeries such as anterior chamber aspiration and glaucoma procedures.[2]

The pathology leading to epithelial downgrowth is not known but is probably related to poor closure and healing of wounds. Wounds may be compromised by the presence of vitreous, iris, or other debris within incisions, which can lead to leaks or fistulas.[3–6] The development of epithelial downgrowth is likely multifactorial, however, and may include a history of multiple operations with or without the presence of significant inflammation, corneal vascularity, and endothelial cell damage. All these conditions may be contributing factors.

Endothelial keratoplasty has recently revolutionized the field of corneal surgery. Although Tillet[7] published the first successful case of posterior lamellar keratoplasty in 1956, endothelial keratoplasty underwent the most rapid advancement and mainstream acceptance following Melles et al.'s[8] description in 1998 of sutureless replacement of the endothelium using only the posterior portion of the donor tissue. Endothelial keratoplasty was described as deep lamellar endothelial keratoplasty by Terry and Ousley[9] and subsequently as Descemet-stripping endothelial keratoplasty by Price and Price.[10] Endothelial keratoplasty has proven to be less invasive than PKP, yielding a theoretically stronger globe[11] and faster visual rehabilitation. Although there have been 3 reports of epithelial ingrowth following endothelial keratoplasty surgery[12–14] and 1 report of epithelial downgrowth occurring as an extension of epithelial ingrowth into the graft–host interface,[15]

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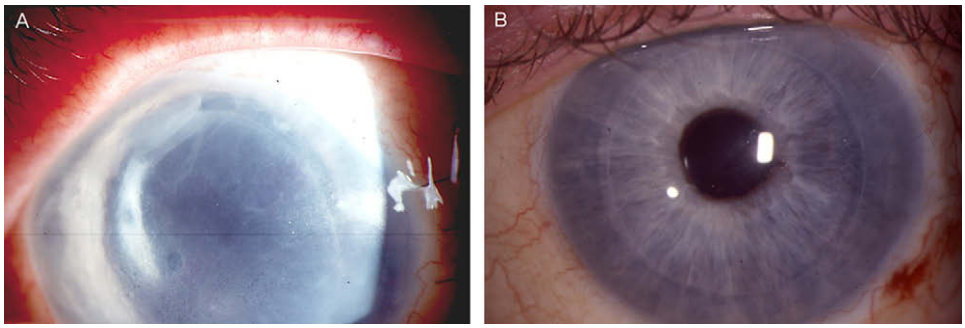

Figure 1. *A*: Preoperative photography showing failed DSAEK graft with stromal edema and poor view of anterior chamber. *B*: One year after replaced DSAEK graft with anterior vitrectomy, the pupil is no longer peaked.

we believe this is the first case of DSAEK primary graft failure with epithelial downgrowth occurring in a patient after 2 DSAEK procedures. The unique feature of our case is that the histopathology demonstrated conjunctival epithelium extending over the donor endothelium as the etiology of the graft failure.

CASE REPORT

A 64-year-old man with Fuchs dystrophy and a history of cataract surgery in the left eye 5 years earlier presented to an outside corneal surgeon (not one of the authors) with slowly progressive corneal edema. On November 17, 2005, uneventful DSAEK was performed in the left eye. On postoperative day 1, a partial dislocation was noted and air was reinjected. The graft reattached but did not clear. After what appeared to be a primary graft failure, repeat DSAEK was performed on February 14, 2006. Again, the surgery was uneventful and the graft was well positioned but remained edematous and did not clear. The patient was referred to our center for a second opinion and subsequent surgical rehabilitation.

On presentation, 6 months after the first DSAEK, the patient complained of pain, light sensitivity, and redness with decreased visual acuity. On examination, the best spectacle-corrected visual acuity was 20/400. Central corneal thickness was 806 µm and the intraocular pressure (IOP), 15 mm Hg. Slitlamp examination demonstrated complete stromal edema without keratic precipitates or anterior chamber inflammation. The pupil was noted to be peaked toward the temporal wound, and vitreous strands were noted attached to the posterior wound margin (Figure 1, *A*). There were no signs of epithelial downgrowth, with no membranes, cysts, or scalloped margins noted on any area of the posterior surface of the cornea.

The patient was offered the options PKP or a third DSAEK and after a thorough discussion of the risks and benefits of each option, he decided to proceed with another DSAEK surgery. Surgery was performed on June 27, 2006, and included a limited anterior vitrectomy, which cleared the anterior chamber of vitreous and rounded the pupil. The failed DSAEK graft was removed by opening the graft–host interface at the temporal edge using sharp and blunt dissection with a crescent blade and a spatula. Once the interface was opened, the blunt spatula easily separated the horizontal donor–recipient interface and then broke the sealed edges of the graft. The tissue was removed, fixed in formalin, and sent to pathology. The remainder of the surgery followed our standard DSAEK technique, including peripheral stromal scraping.

On the first postoperative day, the graft was adherent and thin. The uncorrected visual acuity (UCVA) was 20/100. At the 1-week examination, the UCVA was 20/80 and the IOP was 15 mm Hg.

The histopathology report of the removed failed graft described multilayered surface conjunctival epithelial cells with occasional goblet cells extending over the surgical margin of the specimen and onto the posterior surface. These findings were consistent with epithelial downgrowth (Figure 2) originating from the conjunctiva.

Despite the findings of epithelial downgrowth, the patient continued to improve postoperatively, with no signs of recurrence. Postoperatively, what was thought to be a small

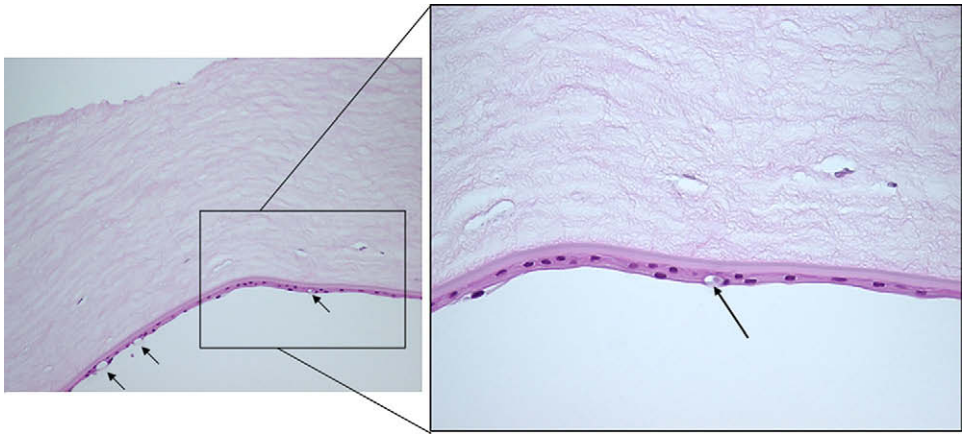

Figure 2. Histopathology of the removed DSAEK graft demonstrating multilayered surface conjunctival epithelial cells with occasional goblet cells (*arrows*) extending over the surgical margin of the specimen and onto the posterior surface, consistent with epithelial downgrowth.