CASE REPORT

Femtosecond laser–assisted anterior lamellar keratoplasty in recurrent epithelial ingrowth after laser in situ keratomileusis

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A 25-year-old woman presented with extensive epithelial ingrowth and corneal scarring in the left eye after a laser in situ keratomileusis (LASIK) retreatment 1 year after primary LASIK. The patient had earlier enhancement for the treatment of overcorrection and subsequently had a flap lift and epithelial ingrowth scraping with alcohol application but had a recurrence in 2 months. Slitlamp biomicroscopy and anterior segment optical coherence tomography showed extensive epithelial ingrowth with thinning and partial resorption of the LASIK flap.

Epithelial ingrowth is a known complication of laser in situ keratomileusis (LASIK). Its incidence varies between 1% to 20%, with a higher rate reported after repeat surgery or trauma.1,2 Its management depends on the size and location of the ingrowth, whether it is progressive or stationary, whether it affects the corrected distance visual acuity (CDVA), and the associated flap changes.3,4 We report a case of grade 3 post-LASIK recurrent epithelial ingrowth along with corneal scarring that was treated with femtosecond laser–assisted anterior lamellar keratoplasty with no evidence of recurrence through 20 months of postoperative follow-up. To our knowledge, this is the first reported case of extensive recurrent epithelial ingrowth associated with flap melt and scarring that was successfully treated with femtosecond laser–assisted anterior lamellar keratoplasty.

CASE REPORT

A 25-year-old woman had microkeratome LASIK in both eyes for simple myopia elsewhere 3 years ago. The preoperative refraction was −3.5 diopters (D) and −3.0 D in right eye and left eye, respectively, with a CDVA of 20/20 in both eyes. She had an overcorrection of 1.5 D in the left eye and 6 months after the initial LASIK, the flap was lifted and a hyperopic enhancement performed. Two months after the enhancement, epithelial ingrowth was noted in the left eye and it was progressing. The flap was manually lifted and the epithelium removed with a combination of mechanical debridement and alcohol wash to both stroma and cap. After this, the eye was uncomfortable and photophobic and considerable inflammation was noted at the cap interface.

When the patient was referred to us 11 months after the initial LASIK procedure, the uncorrected distance visual acuity (UDVA) in the left eye was 20/200 and the CDVA was 20/80, and the UDVA in the right eye was 20/20. The cornea in the left eye showed extensive areas of epithelial ingrowth starting inferiorly and extending to the pupillary area. Two zones of flap–edge scarring were evident inferotemporally and inferonasally, most probably subsequent to flap necrosis (Figure 1). Anterior segment optical coherence tomography (AS-OCT) imaging showed areas of variable flap thickness and scarring with epithelial ingrowth under the flap to a total depth of 170 μm (Figure 2). Corneal topography showed central irregular astigmatism.

In view of the progressive nature of the ingrowth, which was more than 2.0 mm from the flap edge, as well as the involvement of the pupillary area and a reduction in CDVA attributable to the ingrowth, it was decided to intervene surgically. The patient had femtosecond laser–assisted anterior lamellar keratoplasty in the left eye. A recipient corneal dissection 250 μm deep with a residual stromal bed of approximately 280 μm and a diameter of 8.0 mm was planned. The size of the graft corneal button and host corneal bed did not match, even after 2 attempts. The first donor lenticule (8.0 mm) was undersized for the host bed. The second lenticule (8.2 mm) was a bit larger. Therefore, the initial smaller lenticule was used and the gap at the graft–host junction was filled with an annular outer ring of the second larger lenticule trephined and hand fashioned to fit snugly in the gap. Histology of the host corneal button confirmed epithelial ingrowth under the flap along with epithelial cyst formation (Figure 3).
Postoperatively, the lenticule was well attached and the peripheral rim of corneal tissue was well apposed (Figure 4). The attachment of the lenticule and rim was confirmed on AS-OCT (Figure 2). Topical steroid eyedrops were used in tapering doses for 6 months and then discontinued. The patient’s CDVA improved to 20/40 at the 6-month follow-up, with a refraction of $C_{1.00} / C_{0.40} / C_{2.165}$ along with improvement in the astigmatism. There was no evidence of recurrence. A topography-guided photorefractive keratectomy was performed in the left eye 1 year later. The UDVA in the left eye improved to 20/25.

**DISCUSSION**
The management of epithelial ingrowth has to be tailored according to each case. Small isolated nonprogressive areas of epithelial ingrowth not affecting visual acuity can be observed. In progressive and recurrent cases, surgical intervention is warranted. Many treatment modalities for the surgical treatment of post-LASIK epithelial ingrowth have been described in the literature.3,5

Our patient had severe grade 3 recurrent epithelial ingrowth along with corneal scarring subsequent to flap necrosis. Typically, flap lifting and epithelium ingrowth scraping are adequate for epithelial ingrowth removal if the flap integrity is maintained. However, in our case, this was probably not a good approach because the chances of recurrence were high because of the irregular and scarred flap edge. Potentially significant anterior stromal haze and the unpredictable nature of the refractive outcome

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**Figure 1.** Slitlamp photograph of the cornea of the right eye shows islands of epithelial ingrowth extending from the inferior flap edge to the pupillary area. Areas of scarring (black arrows) are seen inferotemporally and inferonasally toward the edge of the flap.

**Figure 2.** a: Anterior segment OCT imaging showing areas of epithelial ingrowth in the center under the flap (white solid arrow) flanked on either side by scarring with overlying stromal thinning (white lined arrow). The intervening flap edge (black solid arrow) can be identified. b, c, and d: Postoperative AS-OCT images show the well-attached lenticule and the peripheral annular rim.

**Figure 3.** a: Scanning photomicrograph of the excised host corneal lenticule shows sheets of epithelial cells growing under the flap. b: Epithelial cyst formation under the flap. c: The site of entry of epithelial cells through the edge of the flap (arrow) (H&E = hematoxylin–eosin; mag = magnification).
with less residual stroma to ablate after a flap amputation make it an unfavorable option.

Femtosecond laser–assisted anterior lamellar keratoplasty is sutureless and has been reported to provide accurate restoration of corneal thickness and contour. Shetty et al. concluded that femtosecond laser–assisted anterior lamellar keratoplasty is a safe and effective alternative to deep anterior lamellar keratoplasty or penetrating keratoplasty in the treatment of anterior stromal opacities. Femtosecond laser–assisted anterior lamellar keratoplasty also replaces the entire complex of epithelial ingrowth, corneal scarring, and irregular thinned flap with a healthy, optically clear donor cornea.

The size of the host bed and the donor button did not match twice. This could be because of the unavailability of nomograms for various corneal curvatures. This disparity can be attributed to the steeper host cornea because of the thick epithelial ingrowth under the flap as compared with that in normal corneas. There were no postoperative adverse events during the follow-up period, and overall improvement in the patient’s subjective symptoms, visual acuity, and topographic findings were observed.

In conclusion, femtosecond laser–assisted anterior lamellar keratoplasty seems to be an efficient and promising treatment modality for the management of severe and recurrent post-LASIK epithelial ingrowth with flap melt and subsequent scarring. To our knowledge, this is the first case report describing the use of femtosecond laser–assisted anterior lamellar keratoplasty for the treatment of post-LASIK epithelial ingrowth. Additional experience with this technique is required to evaluate its efficacy and validate it fully. Modifications in the routine nomograms have to be considered to ensure the size of the host bed is as close as possible to that of the donor button.

REFERENCES

Disclosures: Neither author has a financial or proprietary interest in any material or method mentioned.