Histopathological Analysis of Post-Laser-Assisted In Situ Keratomileusis Corneal Ectasia With Intrastromal Corneal Ring Segments

Keratectasia, an acquired, noninflammatory, outward bulging of the cornea, is associated with a progressive myopic shift in refraction, irregular astigmatism, corneal thinning, and scarring. It generally occurs in the thinnest portions of the cornea, with a central or inferonasal paracentral predilection; however, it may occur elsewhere. Ocular surgical procedures, particularly lamellar refractive surgery (eg, laser-assisted in situ keratomileusis [LASIK]), have been known to induce corneal ectasia. Because the flap produced by LASIK never completely heals, the residual bed likely provides most of the support to maintain corneal shape and function, and keratectasia occurs when the residual bed is unable to provide this support. The 2 main risk factors for post-LASIK keratectasia are a residual corneal bed of 250 μm or less and keratoconus.

Although post-LASIK corneal ectasia may be managed conservatively with rigid gas-permeable lenses, this nonsurgical treatment option may fail or may represent an unacceptable alternative for patients who underwent refractive surgery. In such cases, penetrating keratoplasty has been the principal surgical treatment. Recently, however, intrastromal corneal ring (ICR) segments have shown promise as an alternative surgical option. We describe a unique case of visually significant post-LASIK corneal ectasia initially managed with ICR segments that subsequently progressed, thus requiring penetrating keratoplasty.

Report of a Case. A 40-year-old man underwent bilateral LASIK for a preoperative refractive error of $-1.50 + 2.25 \times 090^\circ$ OD and $-1.75 + 2.50 \times 090^\circ$ OS, with topographies that showed symmetrical astigmatism in both eyes (Figure 1A). After myopic regression, an enhancement was performed to the patient’s right eye by relifting the flap 8 months after the

![Figure 1](https://www.archophthalmol.com/figures/)

**Figure 1.** Corneal topographies of the right eye preoperatively (A), 1 year after enhancement (B), 2 years after enhancement (C), and 9 months after intrastromal corneal ring implantation (D).
first procedure. One month after enhancement, uncorrected visual acuity (UCVA) was 20/25 OD and 20/20 OS, and best-corrected visual acuity (BCVA) was plano = 20/25 OD and plano +0.50 × 180° = 20/20 OS. The refractive correction was stable 1 year postoperatively, although some asymmetrical astigmatism was shown on topographies (Figure 1B). Fifteen months after enhancement, the patient began to complain of blurred, uncorrected vision in the right eye. This was found to be owing to high irregular astigmatism that progressively worsened over the next 8 months. Twenty-three months after enhancement, UCVA was 20/80 OD and 20/60 OS, and BCVA was −1.75 + 4.75 × 143° = 20/30 OD and −1.75 + 2.00 × 085° = 20/20 OS. Ultrasound pachymetry measured a central thickness of 477 µm OD and 492 µm OS. Topography was suggestive of corneal ectasia in the right eye (Figure 1C) and was unremarkable in the left eye. After discussing the options of rigid gas-permeable contact lenses, insertion of ICR segments, or corneal transplantation, contact lenses were tried without success. The patient then elected to have 2 0.35-mm ICR segments placed in the right cornea in divided sessions (Figure 2). The superior ring was placed using a conventional technique (ie, mechanical dissection), and 8 months later, the inferior ring was inserted using a femtosecond laser-created channel. Although UCVA initially improved to 20/60 and BCVA improved to +1.00 + 1.50 × 045° = 20/25, ectasia progressed throughout the next year (Figure 1D). Ultimately, the patient’s UCVA deteriorated to 20/100 with a BCVA of −3.25 + 4.50 × 125° = 20/50. When the patient again proved intolerant to rigid gas-permeable contact lenses, a penetrating keratoplasty was performed.

Gross examination of the 8.0-mm-diameter corneal button showed 2 ICR segments, one placed superiorly and the other placed inferiorly, with a 7.0-mm optical zone. The cornea was bisected for conventional histological analysis and transmission electron microscopy. Light microscopy showed a centrally thinned cornea with focal disrup-

tions in the Bowman layer, and peripheral focal thickened areas where the ICR segments were placed. A periodic acid–Schiff–positive lamellar LASIK scar was present along the interface below the LASIK flap, which left a 218-µm residual stromal corneal bed (Figure 3A-C).

![Figure 2](image-url) Clinical photograph of the right eye 1 day after the inferior intrastromal corneal ring segment was implanted. Arrow indicates crystalline deposits around the intrastromal corneal ring segment; arrowheads, incision. Inset, Higher magnification of the crystalline deposits.

![Figure 3](image-url) Photomicrographs from light microscopy of the central portion of the button, showing the inconspicuous laser-assisted in situ keratomileusis flap. Arrows indicate the laser-assisted in situ keratomileusis wound and the ectatic residual corneal bed. A few focal breaks were found in the Bowman layer. The specimens were stained with hematoxylin-eosin (original magnification ×25) (A), periodic acid–Schiff (original magnification ×25) (B), and toluidine blue (original magnification ×25) (C).
Hexagonal silhouettes were present where the ICR segments were placed, with anterior and posterior displacement of the adjacent collagen lamellae (Figure 4A and B). The stroma around the ICR segments stained more intensely with periodic acid–Schiff than normal corneal stroma do, and on transmission electron microscopy, the stroma contained deposits of electron-dense granular material with interspersed empty spaces (Figure 4C). There was no histological or ultrastructural difference between the mechanically created vs the femtosecond laser-created intrastromal channels. Overlying the ICR segments, a small area of epithelial hypoplasia was present. Inside this area was a zone of epithelial hyperplasia that tapered centrally to normal morphology and thickness (Figure 4A).

Comment. Approved for the treatment of 1.0 to 3.0 diopters (D) of myopia with up to 1 D of astigmatism, ICR segments correct refractive errors by acting as a spacer between collagen lamellae. This induces a shortening of the corneal arc length with peripheral corneal steepening over the region of ICR segments and central corneal flattening between segments. Because ICR segments induce a refractive correction without removing corneal tissue and may potentially reinforce the corneal stroma owing to the addition of material, they have been suggested as a reversible surgical option in patients with corneal ectasia, especially in those with keratoconus or post-LASIK corneal ectasia. Prospective results from 1-year follow-up in post-LASIK corneal ectasia cases suggest that ICR segments can sometimes acutely reduce corneal steepening and astigmatism, thereby improving UCVA, BCVA, and topographic regularity.

Our case is unique because it is one of only a few human histopathological specimens obtained from a cornea managed with ICR segments, and because the ectasia progressed over the first year despite ICR segment implantation. It is also unique in that one ring was implanted using conventional techniques and the other using a femtosecond laser. Previous animal studies that looked at the intrastromal insertion of polymethyl methacrylate prosthetic materials show keratocyte activation, new collagen formation, and lipid deposits in the corneal stroma around the implant. Our human histopathological findings of electron-
dense deposits containing interspersed collagen fibrils, extracellular empty spaces (likely clefts formed from lipid removal during processing), and persistently activated keratocytes are similar to these animal findings. The extracellular lipid collections, which likely correspond to the crystalline deposits seen clinically, probably arise from chronic mechanical irritation to keratocytes that continually strive to heal the stromal wound.

The breaks in the Bowman layer could correspond to predisposition to keratoconus and ectasia. Although the reinforced stromal bed was not strong enough to prevent further ectasia from developing in this case, longitudinal studies have not yet addressed the overall long-term success or stability of ICR segments for corneal ectasia beyond 1 year postoperatively. These studies are necessary to address whether the natural history of the ectasia changes after ICR segment implantation.

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Schisis in Sickle Cell Retinopathy

Retinal schisis is a rare but potentially serious complication of sickle cell retinopathy. It is related to chronic low-grade ischemia of the inner nuclear layer, which houses the Mullerian glia, the structural backbone of the retina. Schisis as part of proliferative sickle cell retinopathy is characterized clinically by a concave tractional retinal elevation, retinal nonperfusion, inner-layer breaks, absorption of laser by the outer layer, and a split pattern on optical coherence tomography. Two cases of retinal schisis are described herein, both featuring the conjunctival sickle sign and both eventually complicated by outer-layer breaks and retinal detachment that possibly might have been prevented by timely laser treatment.

**Figure 1.** Funduscopy, angiography, and optical coherence tomography results in patient 1. A, Drawing of the fundus showing the extent of schisis (hatched lines). B, Ocular coherence tomogram of the left macula. The Mullerian pillars indicate schisis. Photographs of the fundus of the left eye showing neovascularization of the disc (C) and temporal retinal elevation (D). Fluorescein angiogram of the arteriovenous phase focused on the fovea (E) and vessels of retinal elevation (F).