A 58-year-old contact-lens-intolerant man had myopic laser in situ keratomileusis (LASIK) 13 years ago followed by collagen crosslinking (CXL) in the right eye and intrastromal corneal ring segment (ICRS) implantation in the left eye 14 months ago because of late secondary ectasia. He came for consultation because of low quality of vision, theoretically because of high anisometropia.

Before LASIK, the refraction was $-5.50 -0.75 	imes 15$ in the right eye and $-5.50 -1.50 	imes 170$ in the left eye with a corrected distance visual acuity (CDVA) of 1.0 in both eyes; corneal topography (Figure 1); the other ophthalmologic findings were normal. Seven years later, the refraction was $+1.00 -3.50 	imes 60$ in the right eye and $+0.75 -5.50 	imes 115$ in the left eye; the CDVA was 0.8 and 0.5, respectively. Secondary ectasia was diagnosed because of a progressive increase in myopic astigmatism in both eyes and based on corneal topography (Figure 2). Thus, CXL was performed in the right eye and ICRS were implanted in the left eye. The refraction 4 months after both procedures was $-2.00 -2.00 	imes 70$ in the right eye and $+1.50 -2.50 	imes 135$ in the left eye; the CDVA was 0.6 and 0.9, respectively. Figure 3 shows the corneal topography. Now, 14 months after both procedures, the refraction is $+7.50 -7.00 	imes 60$ in the right eye and $-4.50 	imes 110$ in the left eye with a CDVA of 1.0 in both eyes. Figure 4 shows the corneal topography.

Taking into account that the patient is unable to use the full correction at the spectacle plane and considering the topographic and refractive data during the past 18 months, would you perform other evaluations? What is the best rehabilitation approach, nonsurgical or surgical, for both eyes?

**Figure 1.** Corneal topography before LASIK. Top: Right eye. Bottom: Left eye.

**Figure 2.** Corneal topography when secondary ectasia was confirmed. Top: Right eye. Bottom: Left eye.
This is an interesting case of bilateral ectasia after LASIK in a patient with apparently normal preoperative corneal topographies. The most likely causes for the ectasia are undiagnosed keratoconus and excessive keratectomy depth. It is possible that this patient had very early keratoconus in which the cone was small enough for epithelial thickness changes to compensate fully such that the front-surface topography appeared normal, as previously described. The corneal topography back surface shows a slightly elevated and eccentric apex, which may indicate the presence of a subsurface cone masked by epithelium. The keratometric map shows skew astigmatism in both eyes, which might also indicate keratoconus.

It would be interesting to obtain a flap-thickness measurement, ideally using very-high-frequency digital ultrasound given the length of time since LASIK to evaluate whether an unexpected excessively thick flap caused the ectasia. Microkeratomes at that time are reported to have flap thickness reproducibility from 15 μm to less than 30 μm. Some were labeled according to the mean, not the maximum, thickness, thus leading to a large underestimation of the predicted residual stromal thickness (RST). It would be interesting to know which microkeratome was used because a previously published model could predict the probability of leaving an RST of less than 200 μm, thus producing ectasia.

With respect to managing this patient, the conservative approach is to try rigid gas-permeable or hybrid contact lenses in both eyes, using brimonidine tartrate as required to reduce aberrations at night. Although this patient was contact lens intolerant preoperatively, this might have changed given the current situation.

The topography maps of the right eye show that the ectasia progressed despite the CXL procedure and the pachymetry thinned from 490 μm to 381 μm (even considering the potential inaccuracy of Orbscan pachymetry after LASIK). A surgical option would be topography-guided photorefractive keratectomy combined with a second CXL procedure to somewhat reduce the cone height and try to get the refraction back into full spectacle correction range. However, given the low corneal pachymetry and apparent failure of CXL to stop the ectatic process, removing more tissue from the thinnest part of the cornea may only yield diminishing returns. Another CXL procedure may require overhydrating the cornea using hypotonic solution to minimize the theoretical risk for endothelial damage. In reality, this option...
would probably only buy time before the inevitable next step; that is, deep lamellar keratoplasty transplantation.  

The topography scans of the left eye appear to show that the ectasia has not progressed; nevertheless, given the relative safety of CXL, I would suggest performing this prophylactically while the visual acuity is still reasonable. Understanding the anatomic relationship between the flap plane and the ICRS plane of insertion may reveal useful information to improve the astigmatic correction; rotating the ICRS might help given the variability in the biomechanical response of ectatic corneas and hence the shape change induced depending on the location of the implants. It is worth questioning the long-term validity of ICRS insertion in ectasia given that the mechanism of action is to “pull” on the central cornea, thus increasing the central load on a sector of corneal tissue that is already showing plastic deformation.

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REFERENCES


Post-LASIK ectasia is a challenging problem that we must solve because the involved patients are very disappointed that a procedure performed to allow spectacle independence has led to a refractive disaster. Of the proposed solutions, ICRS implantation and CXL are probably the most interesting, although their efficacy remains controversial. In this patient, CXL gave a worse outcome than ICRS implantation. The cylinder in the right eye after CXL increased from –2.00 × 70 at 4 months to the current –7.00 × 60. During the same period, the cylinder in the left eye, which had ICRS implantation, increased from –2.50 × 135 to –4.50 × 110. We are told the patient does not tolerate full spectacle correction and is asking for improvement. Before any surgical decision is made, we must carefully evaluate the corneal anatomy and shape. Thus, Scheimpflug or optical coherence tomography corneal analysis is mandatory to precisely assess the corneal thickness in the ectatic zone and in the surrounding zones.

The first nonsurgical option is rigid contact lenses. Several companies worldwide can provide custom contact lenses based on corneal shape and topography. Recently, it was found that patients tolerate custom soft contact lenses better than standard rigid lenses. Although this approach could work in the short term, it will not stop the ectasia from progressing. In addition, the patient may not tolerate or accept contact lens fitting.

If the patient is reasonably satisfied with vision in the left eye, my first surgical proposal would be to implant ICRS in the right cornea if the cornea is thick enough. Small-diameter segments should be used, which may control and reduce the ectasia and decrease the astigmatism. Should the cylinder correction decrease to 4.00 to 5.00 diopters, the required spectacle correction could be tolerated with minor vision impairment. The advantage of this solution is to avoid or defer corneal transplantation while maintaining some of the results of the refractive procedure.

The definitive solution to corneal ectasia is deep anterior lamellar keratoplasty (DALK), which should be performed in both eyes. However, even under the best circumstances, the recovery of vision could take more than 1 year and the operated eye will regain its original myopia almost completely. In my experience, these patients ask again for refractive surgery and require further counseling.

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REFERENCES

within the usual limits. However, the pre-LASIK posterior float in Figure 1 seems to be higher than 50 μm. This might indicate an unrecognized keratectasia, probably forme fruste keratoconus, given the age of the patient at the time of the initial surgery.

Two surgical approaches were chosen once the diagnosis of iatrogenic keratectasia after LASIK was made. The right eye received CXL treatment, whereas ICRS were implanted in the left eye. In the right eye, the postoperative figures show an arrest of keratectasia progression and an increase in CDVA due to corneal remodeling. In the left eye, the keratectasia seems to progress between 4 months (Figure 3) and 14 months (Figure 4) after ICRS implantation, although the CDVA increased as a result of the more regularized cornea.

The good CDVA with spectacle use in the right eye indicates that the symmetric part of the optical errors in this otherwise multifocal cornea allows a correction up to an acuity of 1.0. In view of the arrest of keratectasia progression, I would suggest contact lens adaptation or a toric phakic intraocular lens.

The topographies of the left eye seem to indicate a progression of the keratectasia. I would closely monitor the patient with a follow-up every 3 months and perform CXL if there is documented progression. At a later stage, and once the keratectasia has stopped, one might consider a limited topography-guided PRK (Athens protocol).

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