

Corneal Ectasia After Laser In Situ Keratomileusis After Laser Thermal Keratoplasty

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Purpose: To report a case of corneal ectasia following hyperopic laser in situ keratomileusis in a patient who had previous laser thermal keratoplasty.

Methods: Case report.

Results: We report a case of a 66-year-old emmetropic man initially presented with complaint of difficulty reading without correction. Laser thermal keratoplasty (LTK) was performed on the non-dominant right eye, resulting in successful monovision. Three years later, the patient presented with decreased unaided near vision caused by hyperopic regression of the LTK. Conventional hyperopic laser in situ keratomileusis (LASIK) was performed, again resulting in successful monovision. More than three years later, the patient returned with worsening near vision. A focal corneal ectasia was noted in the same location as the 6 o'clock LTK leukoma.

Conclusions: To our knowledge, this is the first report of ectasia occurring after LASIK following LTK. Consideration should be given to performing photorefractive keratectomy (PRK) instead of LASIK following thermal keratoplasty.

Key Words: corneal ectasia, laser thermal keratoplasty, laser in situ keratomileusis

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Laser thermal keratoplasty (LTK) has been used for correction of hyperopia. Regression of initial refractive effect can occur.^{1,2} Several case series in the literature report that laser in situ keratomileusis (LASIK) is a safe alternative for treating hyperopic regression after LTK.^{3–5}

To our knowledge, this is the first report of corneal ectasia occurring after LASIK after LTK.

CASE REPORT

A 66-year-old man initially presented with the desire to reduce dependence on reading glasses. He had no prior ocular surgeries. Uncorrected visual acuities for both eyes were 20/20 for distance and J8 for near. With manifest refractions of $-0.25 + 0.50 \times 180$ OD and

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plano $+ 0.25 \times 180$ OS, add $+1.50$ in both eyes (OU), he achieved 20/20+ for distance and J1 for near in both eyes. He was left eye dominant. Slit-lamp examination revealed normal findings, including clear crystalline lenses in both eyes. Corneal topography of the right eye revealed no abnormalities, 1 diopter (D) of with-the-rule astigmatism, and mean keratometry reading of 41.3 D (Fig. 1). Preoperative central corneal thickness was 552 μm by ultrasound. LTK was performed in October 2000 on the right eye with a target of -1.50 D for monovision. Sixteen spots (two 8-spot rings) at 6 and 7 mm were performed. On postoperative day 1, the patient was happy with an uncorrected near vision of J2.

Three years later, the patient presented with decreased unaided near vision, worse in low light and in the mornings. Uncorrected visual acuity for the right eye was 20/40 for distance and J6 for near. Manifest refraction of the right eye was $-0.75 + 1.25 \times 175$, resulting in 20/20 vision. With a trial lens of $+1.50 + 1.25 \times 175$, the patient was able to see near vision of J2. Slit-lamp examination revealed clear crystalline lenses. Axial corneal topography showed uniform central steepening (a typical appearance after LTK) and mean keratometry of 42.5 D (Fig. 2). This minimal increase in keratometry reading compared with the pre-LTK topography (mean keratometry of 41.3 D) and clear status of the crystalline lens suggest that the decreased near vision was primarily caused by regression of effect from the LTK rather than significant presbyopic change. Hyperopic conventional LASIK was performed (LadarVISION 4000, Moria M2 microkeratome, superior hinge) in November 2003 with a target of -2.25 D, a 6.0-mm optical zone, a 1.5-mm blend zone, and a deepest ablation of 40 μm occurring at the 6-mm diameter. Postflap lift pachymetry was 346 μm , yielding a flap thickness of 179 μm measured by subtraction ultrasound pachymetry. No complications occurred. On postoperative day 1, the patient was happy with an uncorrected near visual acuity of J2.

Another 3 years later, the patient presented with worsening vision in his right eye for 3 months. Uncorrected visual acuity was count fingers at 2 feet for distance and J5 for near. Manifest refraction was $-2.75 + 1.50 \times 170$, resulting in 20/30 best-corrected visual acuity with a poor end point. Wavefront refraction was $-3.39 + 4.02 \times 180$. Manifest refraction using the wavefront measurements as a starting point did not yield improved best-corrected visual acuity. Slit-lamp examination revealed 1+ nuclear sclerosis and a remarkable area of focal ectasia posterior to the 6-o'clock LTK leukoma (Fig. 3A). Orbscan (Bausch & Lomb, Salt Lake City, UT) scanning slit-beam topography demonstrated marked irregular astigmatism in a pellucid-like pattern (3.9 D cylinder, irregularity index at 3 mm: 4.8). This marked irregular astigmatism is caused by posterior and anterior corneal steepening at the 6-mm optical zone in the same location as the 6-o'clock leukoma (Fig. 3B).

DISCUSSION

Previous studies have recognized reduced predictability⁴ and surface irregularities³ after LASIK after LTK. However, the general consensus in the current literature supports LASIK

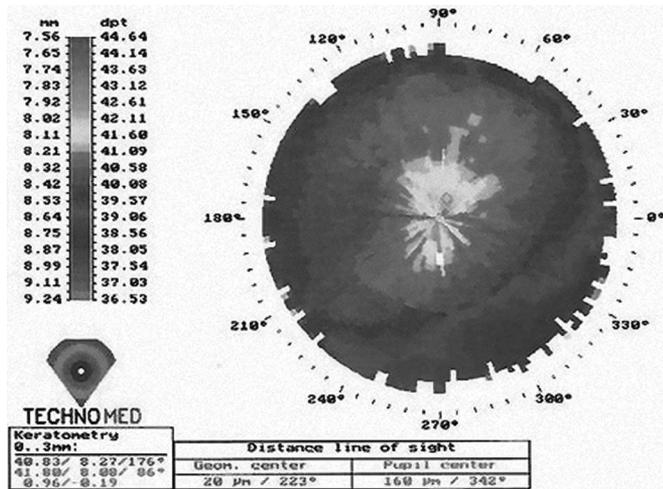


FIGURE 1. Corneal topography of the right eye before any surgeries.

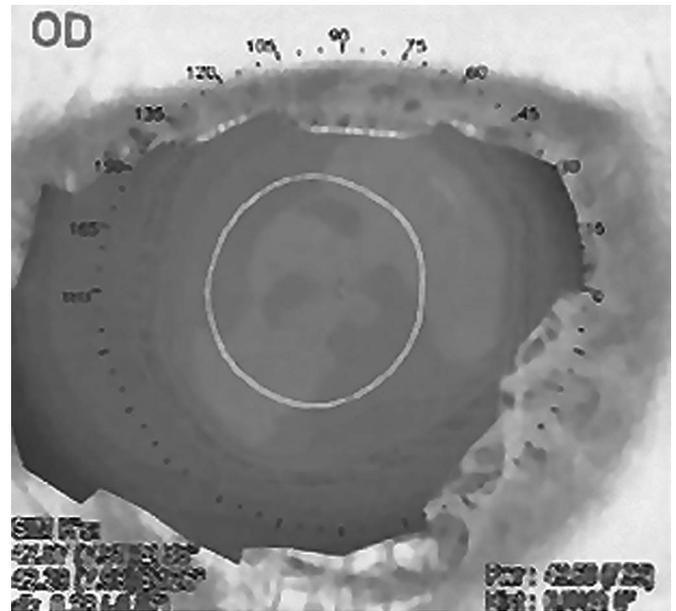


FIGURE 2. Corneal topography of the right eye after LTK showing uniform central steepening.

after LTK as a safe surgical option for hyperopic regression.³⁻⁵ These studies are limited by their follow-up time, with 24 months being the maximum duration reported. We report a case of corneal ectasia occurring 3 years after LASIK after LTK in a patient with none of the established risk factors for ectasia after primary LASIK.

Additive biomechanical weakening of this patient's cornea is likely the cause of the ectasia. Although it has not been reported, we have occasionally observed an area of lucency in the subepithelial stroma of an LTK spot. A similar lucency, cylindrical in shape, surrounding the area of the probe tip, and extending to the depth of the leukoma, has been observed on occasion in conductive keratoplasty (CK) leukomas. These areas of lucency may represent areas of focal necrosis because of thermal energy deposition.^{6,7} In this patient, the first biomechanical weakening event may have

been this focal anterior stromal necrosis. The second biomechanical insult was a relatively thick LASIK flap. The central flap thickness was 179 μm by subtraction ultrasound pachymetry. The flap was probably thicker at the 6-mm optical zone because of the meniscus flap profile typical of pivoting, superior hinge microkeratomes.⁸ The third weakening event was the hyperopic ablation. The deepest ablation occurred at the 6-mm optical zone, the exact location of the previously placed LTK leukoma.

LASIK after CK has also been reported as a safe treatment option for residual hyperopia with astigmatism, with no intraoperative or postoperative complications, a predictable

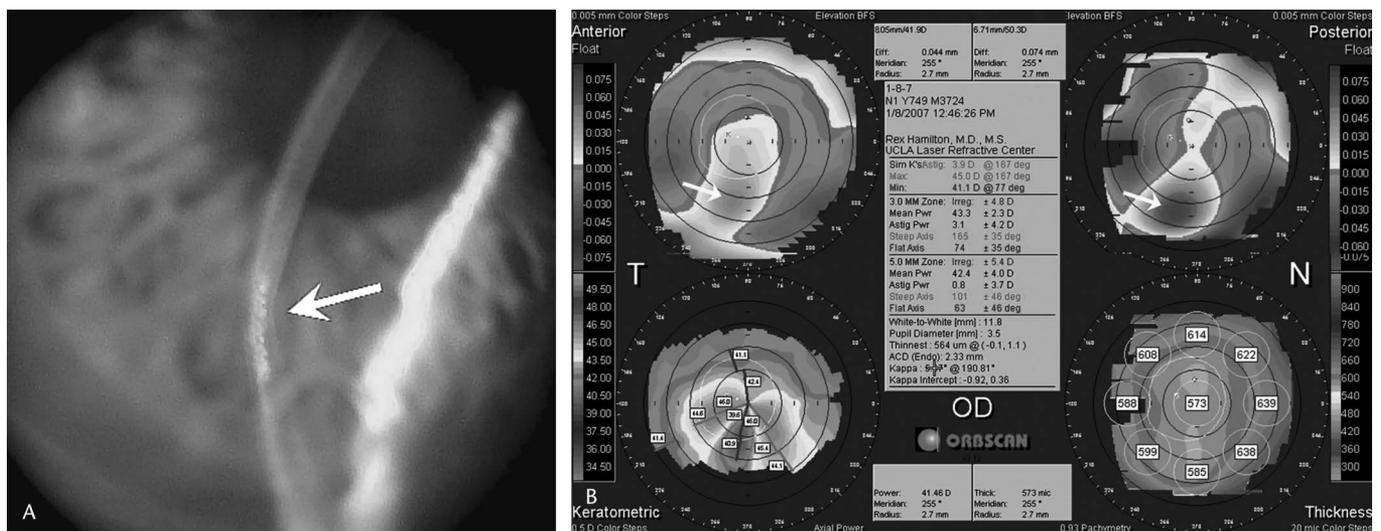


FIGURE 3. A, Slit-lamp photograph showing area of focal ectasia posterior to the 6-o'clock LTK leukoma (arrow). B, Orbscan scanning slit-beam topography showing abnormal elevation on both the anterior and the posterior elevation maps (arrows) at the 6-mm optical zone in the same location as the 6-o'clock LTK leukoma.

refractive outcome, and improved visual acuity.^{9,10} These reports, however, are limited by their 6-month follow-up. Although CK uses radiofrequency energy, unlike LTK that uses laser energy, hyperopic LASIK after CK may also have a risk of ectasia. Performing photorefractive keratectomy with mitomycin-C, instead of hyperopic laser in situ keratomileusis (H-LASIK) after regressed LTK or CK may reduce this risk.

REFERENCES

1. Alio JL, Ismail MM, Sanchez Pego JL. Correction of hyperopia with non-contact Ho:YAG laser thermal keratoplasty. *J Refract Surg.* 1997;13:17–22.
2. Eggink CA, Bardak Y, Cuypers MH, et al. Treatment of hyperopia with contact Ho:YAG laser thermal keratoplasty. *J Refract Surg.* 1999;15:16–22.
3. Attia W, Perez-Santonja JJ, Alio JL. Laser in situ keratomileusis for recurrent hyperopia following laser thermal keratoplasty. *J Refract Surg.* 2000;16:163–169.
4. Thompson MJ, Brightbill FS. Laser in situ keratomileusis for astigmatism following laser thermal keratoplasty. *J Cataract Refract Surg.* 2007;33:142–143.
5. Portellinha W, Nakano K, Oliveira M, et al. Laser in situ keratomileusis for hyperopia after thermal keratoplasty. *J Refract Surg.* 1999;15(Suppl 2):S218–S220.
6. Pearce J, Thomsen S. Rate process analysis of thermal damage. In: Welch AJ, vGM, eds. *Optical-Thermal Response of Laser-Irradiated Tissue.* New York: Plenum; 1995. p. 561–605.
7. Sporn E, Genth U, Schmalfuss K, et al. Thermomechanical behavior of the cornea. *Ger J Ophthalmol.* 1996;5:322–327.
8. Alio JL, Pinero DP. Very high-frequency digital ultrasound measurement of the LASIK flap thickness profile using the IntraLase femtosecond laser and M2 and Carriazo-Pendular microkeratomes. *J Refract Surg.* 2008;24:12–23.
9. Klein S, Fry K, Hersh PS. Laser in situ keratomileusis after conductive keratoplasty. *J Cataract Refract Surg.* 2004;30:702–705.
10. Kymionis GD, Aslanides IM, Khoury AN, et al. Laser in situ keratomileusis for residual hyperopic astigmatism after conductive keratoplasty. *J Refract Surg.* 2004;20:276–278.