Early Onset Ectasia Following Laser in situ Keratomileusis: Case Report and Literature Review

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ABSTRACT

PURPOSE: Laser in situ keratomileusis (LASIK) has been associated with the development of postoperative corneal ectasia. We present a case of early onset ectasia after LASIK, review known risk factors in development, and discuss possible strategies for prevention.

METHODS: A 39-year-old man underwent bilateral LASIK for moderate myopia. Preoperative cycloplegic refractions were -9.00 +0.25 x 140° OD and -7.75 sphere OS. Corneal topography demonstrated mild inferior steepening bilaterally although definite evidence of keratoconus by either the Klyce/Maeda and Smolek/Klyce keratoconus screening tests was not present. Following the creation of flaps with 160-μm plates, ablations of 93 μm OD and 80 μm OS were performed, estimated to leave residual stromal beds of at least 314 μm OD and 330 μm OS.

RESULTS: On the first postoperative day, uncorrected visual acuities were 20/400 OD and 20/400 OS. On the fifth postoperative day, the patient's uncorrected visual acuity was 20/400 OD, and 20/300 OS. Corneal topography of the right eye showed profound inferior steepening with an apical corneal power in excess of 57 D; topography of the left eye showed mild inferior steepening. Eighteen months after surgery, best corrected visual acuity was 20/40 OD and 20/30 OS with rigid gas permeable contact lenses.

CONCLUSIONS: This case highlights the need for a high index of suspicion when one notes an asymmetric bow-tie pattern on preoperative LASIK corneal topography, despite seemingly safe estimates of residual stromal bed thickness. [J Refract Surg 2002;18:177-194]

LASIK in situ keratomileusis (LASIK) is currently the most common refractive procedure performed in the United States. LASIK is performed by creating a flap of corneal tissue consisting of epithelium, Bowman's layer, and anterior stromal tissue, followed by a refractive photoablation of the exposed stromal bed with an excimer laser. Successful corrections up to -26.00 diopters (D) of myopia have been reported, but most refractive surgeons in the United States do not routinely perform corrections above -12.00 D.1 However, since the keratectomy depth is directly proportional to the attempted correction at a given treatment diameter, the biomechanical stability of the residual stroma is of concern, especially in higher treatment ranges. A recent search revealed more than 25 cases of progressive and visually threatening ectasia in the peer-reviewed literature.2-6 Table 1 summarizes these cases, which presented clinically 1 week to 27 months after the initial surgery.

Ectasia following LASIK has been compared to idiopathic keratoconus, as these patients often develop a progressive central or inferior area of focal topographic corneal steepening. However, postoperative LASIK ectasia differs fundamentally from idiopathic keratoconus in that its etiology is biomechanical.7 Although there is no proven etiology for idiopathic keratoconus, there are biochemical changes associated with and a genetic predisposition for this ectasia.8,9 We report a case of early onset corneal ectasia following LASIK.

CASE REPORT

A 39-year-old Caucasian man sought refractive surgery to correct myopia. He had a 10-year history of soft contact lens wear. Manifest refraction prior to surgery was -9.50 +0.50 x 140° OD and -8.00 +0.25 x 25° OS, yielding a visual acuity of 20/20 OU. Cycloplegic refractions were -9.00 +0.25 x 140° OD and -7.75 sphere OS. No scissoring of the red reflex...
was noted on retinoscopy with a dilated pupil. He reported infrequent changes in his prescription and good visual acuity for a number of years prior to surgery. Preoperative slit-lamp examination performed by a fellowship-trained corneal specialist revealed no corneal findings typical of keratoconus or any other pathology in either eye. Central ultrasonic pachymetry measurements were 537 μm OD and 540 μm OS.

TMS-2 (Tommy, Waltham, MA) corneal topography of the right eye demonstrated mild inferior steepening with simulated keratometry readings of 43.37/44.12 D at 115°. The left eye also demonstrated inferior steepening with simulated keratometry readings of 43.25/44.25 D at 25° (Fig 1). Keratoconus screening by the Klyce/Maeda and Smolek/Klyce tests were negative in the right eye (Fig 2). In the left eye, the Klyce/Maeda test was “suspicious” at a level of 34%, but the Smolek/Klyce test was negative (Fig 3).

The patient underwent bilateral simultaneous LASIK; the Hansatome automated microkeratome (Bausch and Lomb Surgical, Claremont CA) with a 160-μm depth plate was used. In our experience, this microkeratome has produced flap thicknesses of 110 to 130 μm, with a mean of 120 μm. Utilizing the VISX Star-S2 laser (Santa Clara, CA), ablations in the stromal bed of 93 μm OD and 80 μm OS were performed. This was estimated to leave minimum residual stromal beds of at least 314 μm OD and 330 μm OS. No intraoperative pachymetry was performed. On the first postoperative day, the patient's uncorrected visual acuity was 20/400 OD and 20/40 OS. On the fifth postoperative day, the patient's uncorrected visual acuity was 20/400 OD and 20/300 OS. Corneal topography studies revealed simulated keratometry readings of 55.40 D at 101°/49.60 D at 11° OD, and 41.10 D at 20°/40.50 D at 118° (Fig 4). One week after surgery, the patient's uncorrected visual acuity was 20/400 OD, and 20/200 OS, and his best spectacle-corrected visual acuity was 20/400 OD and 20/30 OS with a refraction of -4.50 sphere OD and -2.25 sphere OS. Slit-lamp examination demonstrated deep stromal folds and posterior corneal steepening in the right eye. Slit-lamp examination of the left eye was unremarkable. No evidence of diffuse lamellar keratitis was noted in either eye. One month after surgery, the patient's uncorrected visual acuity was 20/400 OD and 20/200 OS. The patient was fitted with a rigid gas permeable lens in both eyes, which yielded visual acuity of 20/40 OD and 20/25 OS. Central ultrasound pachymetry measurements were 446 μm OD and 463 μm OS. Seven months after surgery best spectacle-corrected visual acuity was 20/30 OD and 20/50 OS. Slit-lamp examination showed rare deep pre-Desemet's membrane opacities in the right eye. No iron lines or other stigmata of keratoconus were noted in either eye. Ultrasonic pachymetry measurements were 440 μm OD and 435 μm OS. Corneal topography of the right eye showed profound inferior steepening in a keratoconus pattern with an apical corneal power in excess of 57 D; the left eye showed mild inferior steepening (Fig 5). He
Figure 1. Preoperative TMS-2 (Tomey) corneal topography of both eyes (normalized scale).

Figure 2. Preoperative keratoconus screening (TMS-2) of the right eye.
was examined most recently 18 months following surgery without significant change in his clinical findings. His best-corrected visual acuity with rigid gas permeable contact lenses was 20/40 OD and 20/30 OS.

DISCUSSION

The pathophysiology of corneal ectasia following LASIK has not yet been established. One proposed mechanism is that intraocular pressure causes forward bowing and thinning of a structurally compromised cornea. In idiopathic keratoconus not associated with LASIK, there is some support in the literature that thinned, ectatic corneas have reduced structural integrity. Andreassen and colleagues have shown in theory that keratoconic corneas have reduced biomechanical strength when compared with normal corneas. This observation has not been confirmed in eyes with ectasia following LASIK. Possible factors that may contribute to structural compromise of the cornea after LASIK include decreased thickness of the residual stromal bed due to an excessively deep keratectomy, depth and width of the ablation, the nature of postoperative wound healing, and intraocular pressure (Fig 5).

Much attention has been focused on the thickness of the residual stromal bed. Because the anterior corneal flap may not contribute to the biomechanical stability of the cornea, the stress supported by the residual stromal bed is a major concern. Several studies have attempted to calculate what the minimal residual stromal bed thickness needs to be in order to minimize the risk of ectasia. Based on his work with keratomileusis—one of the early procedures upon which LASIK is based—Barraquer recommended that "in order to avoid postoperative ectasias in myopia corrections, the cornea must not be left thinner than 0.30 mm in its center." This
often-quoted axiom does not indicate whether he was referring to the total central postoperative thickness or the thickness of the residual stromal bed alone.

In three cases of progressive corneal ectasia and keratoconus-like steepening in previously normal eyes following LASIK, Seiler and associates estimated that the residual stromal bed thickness was less than 200 µm in one patient and between 200 and 250 µm in the others.⁴ Andreoessen estimated that for the normal cornea, a residual stromal bed thickness of less than 250 µm might produce a cornea with tangential elastic modulus comparable to that of a keratoconic cornea.¹⁴ This calculation was based on the assumption that the biomechanical parameters are constant throughout the cornea. This assumption, however, may be questioned as Park and associates showed that the deeper corneal stroma has less biomechanical strength than the anterior layers.¹⁵ The CRS LASIK Study used a calculated residual depth of 250 µm as a lower limit, and reported no cases of ectasia in over 3000 eyes submitted to the FDA for LASIK approval (20,000 eyes in the larger database), with 6 months of follow-up (FDA panel meeting, July 23, 1999). Based on this work, most authors have advocated leaving a minimum residual stromal bed thickness of 250 µm.⁴,⁶
Other researchers have attempted to elucidate the acceptable minimum residual thickness as a percentage of total thickness. Lyle and Jin reported a high incidence of progressive corneal ectasia following automated lamellar keratoplasty (ALK) for hyperopia. The depth of the lamellar cut in this patient group ranged from 52% to 70%. However, their cases included corneas that had undergone prior radial keratotomy and were thus structurally weakened. ALK for hyperopia was thought to work through a mechanism of "controlled ectasia," which treated hyperopia through the induction of central corneal steepening. ALK for hyperopia has largely been abandoned, however, due to the fact that some of these patients went on to develop frank keratoconus following the procedure. It is possible that our patient had flaps that were thicker than anticipated, but there was nothing to suggest that this was the case intraoperatively.

The ablation depth may also play a role in the development of ectasia. This is based on the nomograms for refractive photorefractive keratectomy (PRK), which calculate the indices of ablation in reference to Bowman's membrane and the anterior stroma. The ablation depth can be approximated using the simplified version of Munnerlyn's formula: ablation depth = [optic zone] x [diopters divided by 3]. However, the ablation depth predicted by this equation tends to overestimate the depth removed by various lasers that have incorporated aspheric and multi-zone algorithms into treatments. Also, this formula does not take into account the variation in biomechanical characteristics at different points in the cornea. Some authors report that ablation per pulse is greater in the middle of the stroma as compared to the superficial layer, which would lead to an ablation depth that is deeper than the value predicted. In addition, the biomechanical strength of the posterior stroma is weaker than the anterior stroma, which may serve to further promote ectasia in deeper ablations.

The width of the ablation is another factor that may affect the overall strength of the cornea. Although the ablation zone diameter is generally 6.0 to 6.5 mm, some surgeons increase the ablation zone in patients with large pupils or significant astigmatism. Based on Munnerlyn's formula, as the width of the ablation increases, the thickness of the residual stromal bed decreases significantly. Thus, with wider ablations, the safety margin for the prevention of the development of ectasia is decreased.

A minimal thickness of 250 μm or 50% thickness in the residual stromal bed may not be enough in eyes with subclinical or forme fruste keratoconus. Forme fruste keratoconus is an abortive form of keratoconus with a stable refraction and corneal curvature. There is no consensus on what minimal topographic criteria are required to make the diagnosis of forme fruste keratoconus. Waring and Rabinowitz introduced the term "keratoconus suspect" to describe videokeratography that the physician believes may progress to keratoconus, based on a subjective impression.21,22 These eyes are probably predisposed to the development of ectasia after LASIK. Seiler and Quirke described progressive corneal ectasia that occurred in a patient with a preoperative asymmetric bow-tie pattern that they interpreted as forme fruste keratoconus. McLeod and colleagues described a case of corneal ectasia after LASIK in patient with an asymmetric bow-tie pattern, asymmetric topographic scan findings between the two eyes, and poor initial best spectacle-corrected visual acuity. The incidence of ectasia following LASIK in patients with forme fruste keratoconus is probably higher than formally reported, as many reported cases of postoperative LASIK ectasia do not include a description of preoperative topographic features that is sufficiently detailed to determine how many of those patients also had forme fruste keratoconus.

Possible Prevention Strategies

In the case reported herein, the estimated minimum residual stromal bed was 314 μm in the right eye and 330 μm in the left eye. Neither estimate violated the 250-μm limit or 50% minimum residual thickness suggested as safe in the literature. Preoperative corneal topographies of both eyes demonstrated mild inferior steepening with simulated keratometry readings of 43.37/44.12 D at 115° OD and 43.25/44.25 D at 25° OS. Keratoconus screening by the Klyce/Maeda and Smolek/Klyce tests were not definitively positive for keratoconus, but it is possible that other keratoconus screening tests might have revealed an abnormality. It is also possible that preoperative Orbscan (Bausch & Lomb Surgical, Claremont, CA) studies, not widely available at the time of this patient's surgery, may have revealed a posterior corneal abnormality that predisposed the patient to the development of ectasia. However, the role of Orbscan screening in these patients is still controversial. This case highlights the need for a high index of suspicion when one notes an asymmetric bow-tie pattern or other subtle
The thickness of the microkeratome flap is an important source of inaccuracy in calculating the residual stromal bed thickness. The translational velocity, oscillation frequency of the blade, blade characteristics, and the thickness of the microkeratome plate can all affect keratectomy depth. The Hansatome automated microkeratome, for example, typically yields a mean flap thickness of about 30 μm thinner than labeled, and a standard deviation of 20 to 30 μm. It may be necessary to utilize a thinner microkeratome plate with thinner corneas, although this may increase the risk of buttonholing the flap. If proper protocols are followed, in the present case, it is unlikely that an excessively thick flap would occur.

Preoperative corneal topography and optical coherence tomography may provide useful information to help identify those patients at higher risk for developing ectasia. Both technologies can identify keratoconus and forme fruste keratoconus. The Orbscan may also prove useful in identifying which patients are at a high risk for ectasia, although the efficacy of this test has not been established.

Flap thickness and residual stromal bed depth can be approximated intraoperatively using non-contact optical pachymetry. However, these measurements can be affected by corneal hydration, and tend to change even during observation. Nevertheless, intraoperative readings that suggest a thicker than expected flap has been created may lead to modification of the surgical plan to prevent ablation greater than 250 μm.

In the future, preoperative measurement of the biomechanical constants of the cornea may aid in identification of patients at high risk for developing ectasia. Promising approaches for in vivo evaluation of biomechanic constants include measurement of birefringence of the cornea, ultrasound velocity within the cornea, and mechanical spectroscopy.

REFERENCES

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