Progressive Keratectasia After Laser in situ Keratomileusis

Ahmad Salamat Rad, MD; Mahmoud Jabbarvand, MD; Nader Saifi, MD

ABSTRACT

PURPOSE: We describe ten patients who developed progressive keratectasia following laser in situ keratomileusis (LASIK) and identify possible factors that may lead to ectasia.

METHODS: In this retrospective study, we reviewed the files of 3,634 patients (6941 eyes) who had LASIK between March 2000 and April 2003. Ten patients (14 eyes, 0.2%) developed progressive keratectasia. We also evaluated consequent therapeutic measures and final visual status of these patients.

RESULTS: Patients were examined at a mean 24.9 ± 8.1 months after LASIK. Ectasia developed within a mean 14 ± 0.3 months after surgery. At baseline, mean keratometric power was 44.7 ± 2.30 D, mean corneal thickness was 516 ± 18.9 µm, and mean attempted correction was -10.85 ± 3.20 D. We found a statistically significant correlation between residual stromal thickness, attempted correction, and occurrence of progressive keratectasia. We also found that preexisting abnormal corneal topography was a risk factor for progressive keratectasia. Ultimately, most patients had reasonable visual acuity after penetrating keratoplasty.

CONCLUSION: Progressive keratectasia is a vision threatening complication of LASIK that may occur in previously healthy or diseased eyes. The most important risk factors are residual stromal thickness and preexisting abnormal corneal topography. Penetrating keratoplasty may be a reasonable therapeutic measure for severe cases of progressive keratectasia. [*J Refract Surg* 2004; 20(suppl):S718-S722]

From the Cornea and Refractive Section, Novin Didegan Eye Center, Tehran, Iran.

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Correspondence: Ahmad Salamat Rad, MD, P.O. Box 13185-773, Tehran, Iran. E-mail: a_s_rad@iranmedical.com

Given the fast visual recovery and low rate of complications, laser in situ keratomileusis (LASIK) has gained worldwide acceptance as the procedure of choice for correction of refractive errors.[1,2] However, it can have serious complications—corneal ectasia is one of the vision threatening complications after LASIK. Various studies have been published regarding its diagnosis, treatment, and prevention; and several diagnostic criteria and preventive measures have been described.[1-12] In most reports, eyes developed progressive thinning of the cornea associated with increasing myopia and astigmatism, and progressive steepening of the central or inferior cornea.[1-5,11,12]

This disorder has several names, including but not limited to LASIK-induced corneal ectasia, post-LASIK keratectasia, iatrogenic keratectasia, iatrogenic keratoconus, and progressive post-LASIK keratectasia (PPLK).[1,3,5,7,8,11] We prefer progressive keratectasia after LASIK, as the phrase defines the etiology, natural history, and pathology of the disorder.

In this retrospective study we examined 14 eyes of 10 patients who developed progressive keratectasia after LASIK for correction of myopia, in order to identify factors that may have contributed to complications. We also evaluated the results of penetrating keratoplasty in these eyes.

PATIENTS AND METHODS

In this retrospective study, we reviewed the medical charts of 3634 patients (6941 eyes) who had LASIK between March 2000 and September 2003. Ten patients (14 eyes, 0.2%) developed progressive keratectasia. All patients underwent a thorough eye examination before LASIK, which included uncorrected visual acuity (UCVA), best spectacle-corrected visual acuity (BSCVA), autorefraction (AR600, Nidek, Gamagori, Japan), topography (Topo-Map, CSO, and Orbscan, Orbtek, Salt Lake City, UT), pachymetry, (UP-1000, Nidek and Orbscan) and tonometry.

Table 1
Preoperative Clinical Data for Eyes With Progressive Keratectasia After LASIK

Patient/Eye	BSCVA*	Spherical Equivalent Refraction (D)	Mean Keratometric Power (D)	Corneal Thickness (µm)	Corneal Topography	IOP (mmHg)
1/R	0.9	-14.50	43.20	545	Round	13
2/R	0.8	-8.00	46.40	505	ABT	14
2/L	0.7	-10.50	47.50	500	ABT	14
3/L	1.0	-6.75	44.40	515	SBT	12
4/L	1.0	-7.25	43.50	512	ABT	15
5/R	0.9	-13.50	42.40	522	Oval	12
5/L	0.9	-13.00	42.80	514	Oval	13
6/R	0.8	-10.50	48.20	492	ABT	12
6/L	0.9	-10.00	48.30	493	ABT	12
7/R	0.7	-14.50	42.50	537	SBT	12
8/R	1.0	-5.75	44.40	520	SBT	14
9/R	0.7	-8.75	47.80	492	ABT	13
10/R	0.5	-14.00	42.40	538	SBT	13
10/L	0.5	-15.00	43.10	542	SBT	14

**Best spectacle-corrected visual acuity*
ABT=Asymmetric bowtie; SBT=symmetric bowtie

Table 2
Postoperative Clinical Data at Last Follow-up for Eyes With Progressive Keratectasia After LASIK

Patient/Eye	BSCVA*	Spherical Equivalent Refraction (D)	Site of Steepening	Corneal Thickness (µm)	Residual Stromal Thickness (µm)	IOP (mmHg)
1/R	0.2	-10.50	central	360	200	13
2/R	0.3	-8.00	inferior	390	230	13
2/L	0.1	-11.00	inferior	375	215	13
3/L	0.5	-6.50	central	396	236	11
4/L	0.6	-4.75	central	406	246	14
5/R	0.2	-7.50	central	376	216	13
5/L	0.3	-8.00	central	368	208	13
6/R	0.2	-11.00	inferior	365	205	12
6/L	0.5	-8.50	inferior	373	213	12
7/R	0.3	-9.50	inferior	397	237	13
8/R	0.6	-4.25	central	409	249	14
9/R	0.2	-9.50	inferior	382	222	12
10/R	0.4	-7.50	central	388	228	13
10/L	0.2	-8.50	central	375	215	13

**Best spectacle-corrected visual acuity*

After creation of a superior hinge flap with a Moria CB mickrokeratome (Moria SA, Antony, France), a Nidek EC-5000 excimer laser was used to ablate the cornea. The following information was recorded from our review of patient charts: age, gender, affected eye, BSCVA, refraction, topography, pachymetry, and tonometry.

Progressive keratectasia was diagnosed by decreasing visual acuity, unstable refraction, progressive topographic steepening, (≥1.00 diopter [D] for each 6-month period of follow-up), and corneal thinning (≥20 µm for each 6-month period of follow-up).

RESULTS

Mean patient age was 36.5 ± 11.2 years (range 22 to 53 yr). According to our center's data bank, the mean patient age for LASIK was 25.3 ± 8.2 years.

Six patients (60%) were females and four (40%) were males. Mean follow-up was 24.9 ± 8.1 months (range 14 to 38 mo).

Preoperatively, mean BSCVA was 0.8 (range 0.5 to 1.0), mean spherical equivalent refraction was -10.85 ± 3.20 D (range -5.75 to -15.00 D), mean keratometric power was 44.7 ± 2.30 D (range 42.40 to 48.30 D), and mean corneal thickness (pachymetry) was 516 ± 18.9 µm (range 492 to 547 µm). Corneal topographic patterns included asymmetric bow-tie in six eyes (42.8%); symmetric bow-tie in five eyes (35.7%), and round or oval in three eyes (21.4%). The mean intraocular pressure (IOP) was 13.07 ± 0.9 mmHg (range 12 to 15 mmHg) (Table 1).

At the last examination after LASIK, mean BSCVA was 0.3 (range 0.1 to 0.6) and the mean spherical equivalent refraction was -8.00 ± 2.00 D (range -4.25 to -11.00 D)—both reveal the worsening visual status of patients (Table 2). The mean postoperative corneal thickness (pachymetry) was 382.8 ± 15.1 µm (range 360 to 409 µm) and the mean calculated residual stromal thickness was 222.8 ± 15.1 µm (range 200 to 249 µm). We calculated residual corneal thickness by subtracting estimated flap thickness from preoperative corneal thickness, although this was not always precise (Table 2). Mean IOP was 12.7 ± 0.8 mmHg (range 11 to 14 mmHg).

Progressive keratectasia developed within a mean of 14 ± 0.3 months (range 11 to 17.5 mo). It occurred in eight right eyes (57.2%) and six left eyes (42.8%); four patients (40%) developed bilateral disease .We found no statistically significant difference between unilateral and bilateral cases in terms of age (P=.2), gender (P=.3), magnitude of refractive error (P=.1), mean keratometric power (P=.3), or mean corneal thickness (P=.2).

Corneal steepening occurred centrally in eight eyes (57.2%) and inferiorly in six eyes (42.8%) (Table 2). In all but one of the inferior steepening eyes, there were previous corneal risk factors including decreased corneal thickness, high keratometric power, and abnormal topographic patterns. The mean preoperative corneal thickness of the central steepening group was 526.2 ± 13.9 µm and in the inferior steepening group, 503.1 ± 17.3 µm. Of eight central steepening cases, only one (12.5%) had abnormal topography, but five of six (83.3%) inferior steepening cases had abnormal topography. There was a statistically significant difference between the central and inferior steepening groups in pre-existing topographic abnormalities (P=.02) and corneal thickness (P=.01).

Table 3
Clinical Data for Eyes With Progressive Keratectasia After LASIK Followed by Penetrating Keratoplasty

Patient/Eye	BSCVA*	Spherical Equivalent Refraction (D)
1/R	0.9	-2.50
2/R	0.7	-3.75
5/R	0.7	+0.75†
5/L	0.8	-1.50
6/R	0.7	-0.75
7/R	0.6	-4.50
9/R	0.7	-3.75
10/R	0.5	+1.50

*Best spectacle-corrected visual acuity
†After LASIK for correction of myopic astigmatism following penetrating keratoplasty

Seven patients eventually underwent penetrating keratoplasty and all of them had reasonable BSCVA and refractive errors after surgery. At last follow-up, mean BSCVA was 0.7 (range 0.5 to 0.9) and mean spherical equivalent refraction was -2.15 ± 2.35 D (range +1.50 to -6.00 D) (Table 3).

The results of histopathological examinations of the corneal buttons after keratoplasty were surprisingly insignificant and included normal epithelium, normal Bowman's layer, irregular lamellae with decreased density, and normal Descemet's membrane.

DISCUSSION

The occurrence of keratectasia following LASIK is a great concern in modern refractive surgery and has been reported since 1998.[3,4] Several studies have been published about its etiology, natural history, treatment, and prevention.[1-12] We think that the term progressive post-LASIK keratectasia (PPLK), first introduced by Comaish and Lawless, may be the best name, as it describes etiology, natural history, and pathology of the disorder.[12]

Although the incidence of progressive keratectasia after LASIK is unknown, in one large study the incidence was 0.66%.[2] The disorder may occur within a few months and up to 4 years after LASIK.[1-5,11,12,14]

Some factors thought to be responsible for development of progressive keratectasia after LASIK are:

1) Residual stromal thickness. Although it is difficult to determine what constitutes a safe residual stromal thickness, 250 µm and more is widely accepted as a reasonable limit to prevent

progressive keratectasia after LASIK.[1,4,13-16] Regarding preoperative corneal thickness, this limit was not achieved in any of our cases. Although Flanagan and Binder reported several patients with a residual stromal thickness less than 250 µm who did not develop progressive keratectasia after LASIK during follow-up of more than 3 years[17], and Argento and colleagues reported a few cases of progressive keratectasia after LASIK, even with residual stromal thickness of more than 250 µm[10], we think that maintaining this limit can prevent most, and perhaps all, instances of progressive keratectasia after LASIK. The anterior 100 to 120 µm of cornea is more compact and more resistant to mechanical deformation than the rest of cornea.[18] To create a flap in this area we cut the lamellae, and since the flap forms a relatively weak attachment to the rest of stroma after LASIK, it may not contribute to the biomechanics of cornea as it did before.[3,4] Hence, we think keeping as much residual stroma as possible is critical.

2) Pre-existing corneal pathology. We found corneal risk factors including low pachymetry measurements, abnormal corneal topography, and high keratometric power in 6 of 14 eyes (43%). These risk factors were found in only one of eight central steepening cases and in all but one in the inferior steepening group. This is similar to the findings reported by Faraj and colleagues, who suggested that progressive keratectasia after LASIK in normal eyes presents as central steepening, and as inferior steepening in eyes that had preoperative pathology such as keratoconus or forme fruste keratoconus.[19] Regarding these data, we suggest dividing cases of progressive keratectasia after LASIK into two subgroups: with central steepening and no previous corneal risk factors, and with inferior steepening and previous corneal risk factors. Additional study is required to confirm this hypothesis.

3) Attempted correction. In 13 of 14 eyes (94.7%), attempted correction was >-6.00 D. In the ophthalmic literature, we found that the attempted correction in the majority of eyes with progressive keratectasia after LASIK was >-6.00 D[1-7,14,15], but reports also exist with lower corrections, making the etiopathology of the disorder complex.[1,14,20,21]

4) Age. The mean age of our patients was 36.5 ± 11.2 years (range 22 to 53 yr). According to our center's data bank, mean age of LASIK patients is 25.3 ± 8.2 years. Although cases of progressive keratectasia after LASIK are few, the age range of the patients is clearly higher. Whether age itself can change the corneal response to the laser, its tolerance to ablation energy, or wound healing after LASIK is a matter of controversy, and we found different hypotheses in the literature.[2,22-25]

There may be multiple risk factors for developing progressive keratectasia after LASIK, such as low residual stromal thickness, previous corneal risk factors, attempted correction, age, and other factors not yet identified. We should carefully monitor the residual stromal thickness and corneal topography of LASIK candidates and avoid performing LASIK in doubtful cases, as there are alternative procedures for correction of refractive errors: photorefractive keratectomy, laser subepithelial keratomileusis, phakic intraocular lens, and clear lens extraction. We suggest that a multi-center study be conducted to elucidate factors that might contribute to development of progressive keratectasia after LASIK and define a standard protocol for reporting cases.

REFERENCES

1. Argento C, Cosentino MJ, Tytium A, Rapetti G, Zarate J. Corneal ectasia after laser in situ keratomileusis. J Cataract Refract Surg 2001;27:1440-1448.
2. Pallikaris IG, Kymionis GD, Astyrakakis NI. Corneal ectasia induced by laser in situ keratomileusis. J Cataract Refract Surg 2001;27:1796-1802.
3. Seiler TK, Konfala K, Richter G. Iatrogenic keratectasia after laser in situ keratomileusis. J Refract Surg 1998; 14:312-317.
4. Geggel HS, Talley AR. Delayed onset keratectasia following laser in situ keratomileusis. J Cataract Refract Surg 1999; 25:582-586.
5. McLeod SD, Kisla TA, Caro NC, McMahon TT. Iatrogenic keratoconus: corneal ectasia following laser in situ keratomileusis for myopia. Arch Ophthalmol 2000; 118:282-284.
6. Özdamar A, Aras C, Ustundag C, Bahcecioglu H, Ozkan S. Corneal iron ring associated with iatrogenic keratectasia after myopic laser in situ keratomileusis. J Cataract Refract Surg 2000;26:1684-1686.
7. Jabbur NS, Stark WJ, Green WR. Corneal ectasia after laser in situ keratomileusis. Arch Ophthalmol 2001; 119: 1714-1716.
8. Spadea L, Palmieri G, Mosca L, Fasciani R, Balestrazzi E. Iatrogenic keratectasia following laser in situ keratomileusis. J Refract Surg 2002;18:475-480.
9. Alio JL, Salem TF, Artola A, Osman A. Intracorneal rings to correct corneal ectasia after laser in situ keratomileusis. J Cataract Refract Surg 2002;28:1568-1574.
10. Siganos CS, Kymionis GD, Astyrakakis NI, Pallikaris IG. Management of corneal ectasia after laser in situ keratomileusis with Intacs. J Refract Surg 2002;18:43-46.
11. Binder PS. Ectasia after laser in situ keratomileusis. J Cataract Refract Surg 2003;29:2419-2429.
12. Comaish IF, Lawless M. Progressive post LASIK keratectasia. J Cataract Refract Surg 2002;28:2206-2213.
13. Kohnen T. Iatrogenic keratectasia: current knowledge, current measurements [Editorial]. J Cataract Refract Surg 2002;28:2065-2066.
14. Randleman JB, Russell B, Ward MA, Thompson KP, Stulting RD. Risk factors and prognosis for corneal ectasia

after LASIK. Ophthalmology 2003;110:267-275.
15. Joo C-K, Kim T-G. Corneal ectasia detected after laser in situ keratomileusis for correction of less than -12 diopters of myopia. J Cataract Refract Surg 2000:26:292-295.
16. Vinciguerra P, Camesasca FI. Prevention of corneal ectasia in laser in situ keratomileusis. J Refract Surg 2001; 17(suppl):S187-S189.
17. Flanagan GF, Binder PS. Estimating residual stromal thickness before and after laser in situ keratomileusis. J Cataract Refract Surg 2003;29:1674-1683.
18. Bron AJ. The architecture of the corneal stroma [Editorial]. Br J Ophthalmol 2001;85:379-381.
19. Faraj HG, Gatinel D, Chastang P, Huang-Xuan T. Corneal ectasia after LASIK [Letter]. J Cataract Refract Surg 2003;29:220.
20. Amoils SP, Deist MB, Gous P, Amoils PM. Iatrogenic keratectasia after laser in situ keratomileusis for less than -4.00 to -7.00 diopters of myopia. J Cataract Refract Surg 2000; 26:967-977.
21. Ou RJ, Shaw EL, Glasgow BJ. Keratectasia after laser in situ keratomileusis (LASIK): evaluation of residual stromal bed thickness. Am J Opthalmol 2002;134:771-773.
22. Detorakis ET, Siganos DS, Kozobolis VP, Pallikaris IG. Corneal epithelial wound healing after excimer laser photorefractive and photoastigmatic keratectomy (PRK and PARK). Cornea 1999;18:25-28.
23. Hefetz L, Domnitz Y, Haviv D, Krakowsky D, Kibarsky Y, Abrahami S, Nemet P. Influence of patient age on refraction and corneal haze after photorefractive keratectomy. Br J Ophthalmol 1997; 81:637-638.
24. Liu JC, McDonald MB, Varnell R, Andrade HA. Myopic excimer laser photorefractive keratectomy: an analysis of clinical correlations. Refract Corneal Surg 1990:6:321-328.
25. Bashour M. Risk factors for epithelial erosions in laser in situ keratomileusis. J Cataract Refract Surg 2002;28: 1780-1788.