

Published in final edited form as:

J Cataract Refract Surg. 2012 June ; 38(6): 1014–1019. doi:10.1016/j.jcrs.2011.12.030.

Diffuse lamellar keratitis after laser in situ keratomileusis with femtosecond laser flap creation

Fernando H. de Paula, MD, Christian G. Khairallah, MD, Leslie M. Niziol, MS, David C. Musch, PhD, MPH, and Roni M. Shtein, MD, MS

Department of Ophthalmology and Visual Sciences (de Paula, Khairallah, Niziol, Musch, Shtein), W.K. Kellogg Eye Center, and the Department of Epidemiology (Musch), School of Public Health, University of Michigan, Ann Arbor, Michigan, USA; the Department of Ophthalmology (de Paula), Division of Cornea and External Disease, Santa Casa de São Paulo, São Paulo, Brazil

Abstract

PURPOSE—To identify possible associations with the development of diffuse lamellar keratitis (DLK) after laser in situ keratomileusis (LASIK) with femtosecond laser flap creation.

SETTING—University-based academic practice, Ann Arbor, Michigan, USA.

DESIGN—Case-control study.

METHODS—Myopic LASIK was performed between October 2006 and December 2010 using an Intralase 60 kHz femtosecond laser for flap creation. Preoperative clinical characteristics, treatment parameters, and intraoperative and postoperative complications were recorded. Statistical comparisons were made using *t*, chi-square, and Fisher exact tests and repeated-measures logistic regression to adjust for inter-eye dependency.

RESULTS—The study enrolled 801 eyes (419 patients). Ninety-nine eyes (12.4%) of 70 patients developed DLK; most cases comprised mild flap interface inflammation and were treated with a routine postoperative antiinflammatory regimen. Twenty-two eyes (2.7%) required more than 1 week of antiinflammatory treatment. There was a statistically significant increase in the incidence of DLK with larger flap diameter ($P = .0171$), higher side-cut energy ($P = .0037$), and higher raster energy ($P = .0033$). Patients with DLK were less likely to achieve corrected distance visual acuity of 20/20 or better 1 day postoperatively ($P = .0453$). The difference in acuity was no longer present at 1 week. There were no significant associations between the incidence of DLK and preoperative refractive error, flap thickness, ablation depth, or other treatment parameters.

CONCLUSIONS—Diffuse lamellar keratitis after LASIK with femtosecond laser flap creation tended to be mild with little effect on visual acuity. Higher energy level for flap creation and larger flap diameter were associated with an increased risk for DLK.

© 2012 American Society of Cataract and Refractive Surgery and European Society of Cataract and Refractive Surgeons. Published by Elsevier Inc. All rights reserved.

Corresponding author: Roni M. Shtein, MD, MS, W.K. Kellogg Eye Center, 1000 Wall Street, Ann Arbor, Michigan 48105, USA. ronim@med.umich.edu.

Presented at the Association for Research in Vision and Ophthalmology, Fort Lauderdale, Florida, USA, May 2011.

Financial Disclosure: No author has a financial or proprietary interest in any material or method mentioned.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

With the number of laser in situ keratomileusis (LASIK) cases performed in the United States exceeding 630 000 in 2009,¹ most eyecare providers are becoming increasingly aware of the complications related to lamellar refractive surgery. Diffuse lamellar keratitis (DLK) is one of the most common postoperative complications of LASIK surgery. It was first described by Smith and Maloney² in 1998 as a granular white cellular infiltrate within the LASIK flap interface. It represents a sterile inflammatory reaction, usually in the immediate postoperative period. Although most cases resolve, some progress to severe inflammation and permanent scarring.³ Given the potential for serious vision-threatening consequences, vigilance and prompt treatment are required.

The etiology of DLK is not well understood. It has been associated with factors such as bacterial endotoxin,⁴ chemicals or debris,⁵ surgical gloves,⁶ and surgical marking pens.^{7,8} Patient factors shown to affect the risk for DLK include meibomian gland secretions and peripheral immune infiltrates,^{9,10} atopy,¹¹ and iatrogenic epithelial defects.^{12,13} Ultimately, DLK is likely the result of how a patient's endogenous factors respond to exogenous exposures.¹⁴

Diffuse lamellar keratitis after LASIK has been reported to occur at higher frequency with femtosecond laser flap creation than with microkeratome flap creation. The incidence of DLK is estimated to range from 0.2% to 19.4% after femtosecond laser flap creation¹⁵⁻¹⁹ and from 0.1% to 7.7% after microkeratome flap creation.^{18,20-25}

With the increased use of the femtosecond laser for flap creation, there is interest in understanding this phenomenon. Studies¹⁷⁻¹⁹ have shown that high laser energy levels and the ensuing photodisruption-induced tissue injury and accumulation of gas bubbles can lead to an increased inflammatory response and DLK.

Recently, it was reported that reducing femtosecond energy settings, especially side-cut energy, decreases the incidence for DLK.¹⁷⁻¹⁹ Our previous study evaluating the incidence of DLK after LASIK using 3 femtosecond laser frequencies¹⁵ found no statistically significant difference between the lasers. However, that study included surgeries with minimal variability in side-cut or bed energy. The purpose of this study was to identify possible associations with the development of DLK after LASIK performed using the same femtosecond laser for flap creation.

PATIENTS AND METHODS

This study, which was approved by the University of Michigan Institutional Review Board, retrospectively reviewed the records of consecutive patients who had myopic LASIK using the Intralase 60 kHz femtosecond laser (Abbott Medical Optics, Inc.) for flap creation between October 2006 and December 2010. The Technolas 217 excimer laser (Bausch & Lomb) was used for stromal ablation in all cases. In bilateral surgeries, the right eye was always treated first with the femtosecond laser and excimer laser.

Preoperative clinical characteristics and treatment parameters as well as intraoperative and postoperative complications were collected and analyzed. The following clinical data were collected: date of LASIK, patient age and sex, presence of meibomian gland dysfunction, history of ocular allergies, corneal thickness, preoperative Schirmer values after topical anesthesia, preoperative intraocular pressure (IOP), and preoperative use of ocular medications. The following treatment data were recorded: optical zone, raster energy, raster spot separation, side-cut energy, intended flap thickness, flap diameter, ablation depth, and treated refractive error. Residual bed thickness was calculated by subtracting the ablation depth and flap thickness from the preoperative corneal thickness. The uncorrected (UDVA)

and corrected (CDVA) distance visual acuities were recorded 1 day and 1 week after LASIK.

For patients who developed DLK, the day of onset, DLK grade, treatment, and day of resolution were recorded. The DLK was graded according to the staging described by Linebarger et al.¹⁴ as follows: stage 1 = faint white granular infiltrate confined to flap periphery; stage 2 = white granular infiltrate diffusely distributed in flap interface; stage 3 = diffuse white granular infiltrate centrally more confluent and densely clumped; stage 4 = central striae, bullae formation, stromal melting, and permanent scarring.

The standard postoperative treatment protocol involved the use of corticosteroid and antibiotic eyedrops, separately or in combination, starting the day of or the day after surgery, depending on the surgeon's preference. After the first postoperative week, the eyedrops were stopped. If DLK was detected, more frequent corticosteroids were started and tapered according to the surgeon's preference and as indicated by the clinical course.

Statistical comparisons between the group that developed DLK and the group with no DLK were performed using *t* tests for continuous variables and the chi-square or Fisher exact test for categorical variables. Univariate repeated-measures logistic regression models using generalized estimating equations to adjust for inter-eye dependency were used to assess the association between rates of DLK and clinical and treatment variables. A *P* value less than 0.05 was considered statistically significant. All analyses were performed using SAS software (version 9.2, SAS Institute, Inc.).

RESULTS

Eight hundred one eyes of 419 patients were included in this analysis. Binocular LASIK was performed in 382 patients and monocular LASIK in 37 patients. The mean age of the 232 (55.4) women and 187 men (44.6) was 38 years \pm 10 (SD) (range 21 to 72 years). Table 1 shows the demographic data of patients who developed DLK and those who did not develop DLK.

Diffuse lamellar keratitis developed in 99 eyes of 70 patients, 29 bilateral and 41 unilateral (Figure 1). Of the 41 cases of unilateral DLK, 37 occurred after bilateral LASIK; of those cases, 18 (48.7%) involved the right eye and 19 (51.4%) involved the left eye. The overall incidence of DLK was 12.4%. No significant differences in age or sex were found. Preoperative characteristics, including corneal thickness, IOP, presence of allergy or meibomian gland dysfunction, and Schirmer test results, were not significantly different between those who developed DLK and those who did not.

Most cases of DLK represented mild inflammation that required little, if any, additional antiinflammatory treatment for resolution. Of the 99 eyes with DLK, 22 (2.7% of total) required more than 1 week of treatment. In 77 eyes (77.8%), DLK resolved within the first week after LASIK, 9 (9.1%) resolved within 2 weeks, and 13 (13.1%) required up to 1 month of treatment. Table 2 shows the number and percentage of eyes that developed DLK stages 1 to 3. No eye developed stage 4 DLK.

An increased incidence of DLK was associated with several femtosecond treatment parameters (Table 3), including larger flap diameter ($P = .0171$), increased levels of raster energy ($P = .0033$), and side-cut energy ($P = .0037$) used for flap creation. There was no difference in the incidence of DLK with variations in flap thickness. However, the flap thickness ranged from 100 to 140 μm only. There were no significant associations between the incidence of DLK and preoperative refractive error, treated refraction, ablation depth, or other treatment parameters.

The postoperative eyedrop regimen was surgeon-dependent and consisted of the use of topical steroids starting on the day of surgery in 371 eyes and starting on the first postoperative day in 282 eyes. When comparing these 2 groups, there was no difference in the incidence of DLK ($P = .5193$). Patients with DLK were less likely to achieve a CDVA of 20/20 or better on the first postoperative day ($P = .0453$) (Table 4). Forty-five eyes had minor complications (microstriae, epithelial defect, or small epithelial ingrowth) in the perioperative period; however, they were not associated with the incidence of DLK ($P = .1751$). There were no major LASIK complications.

DISCUSSION

Femtosecond laser-assisted LASIK flap creation has been associated with a higher risk for DLK than microkeratome flap creation.^{18,19} The precise reasons for the increased incidence of inflammation after femtosecond flap creation are not completely understood. Confocal and histological studies^{26,27} found greater stromal cell apoptosis, monocyte influx, and a higher wound-healing index with femtosecond laser flap creation than with microkeratome flap creation.

In this study, the overall rate of DLK was 12.4%, which is higher than in some other recent reports of post-LASIK DLK.¹⁶⁻¹⁸ This is most likely because of the meticulous notation of the presence of even minimal inflammation in the flap interface in our study population. Only 22 (2.7%) of all eyes required treatment for DLK beyond 1 week postoperatively, which is an incidence more consistent with that reported in the recent literature. Of the cases of DLK in our study, 30 eyes (30.3%) were maintained on the standard topical corticosteroid treatment regimen of 4 times a day and 69 eyes (69.7%) were treated with more frequent corticosteroid eyedrops. Only 3 eyes (3.0%) required oral corticosteroids, and none required surgical flap manipulations for treatment.

Flap diameter was associated with development of DLK in this study. Patients who developed DLK had a larger flap (mean 8.8 ± 0.2 mm; range 8.0 to 9.4 mm) than patients who did not develop DLK (mean 8.7 ± 0.3 mm; range 8.0 to 9.5 mm). This 0.1 mm difference was statistically significant ($P = .0171$). One possible explanation for this finding is the closer proximity of the larger diameter flaps to the limbus and limbal blood vessels, which could potentially expose the flap to more inflammatory mediators and cells from the limbus. However, we do not necessarily recommend reduction of flap size based on these findings, especially considering the benign appearance of most of our DLK cases and the lack of long-term visual consequences.

We also found a significantly decreased incidence of DLK with decreased femtosecond raster and side-cut energy settings, confirming previous reports of a lower incidence of DLK with reduced energy settings.¹⁷⁻¹⁹ Based on the results in previous laboratory studies,^{26,27} we suspect that lower laser energy settings induce less inflammatory stimulus, leading to fewer inflammatory mediators and less cellular immune response. The exact relationship between laser energy and DLK development, whether a possible linear relationship or a threshold energy level above which DLK would occur, remains to be determined.

Gil-Cazorla et al.¹⁸ posit that their low incidence of DLK (0.5%) may be the result of their standard postoperative regimen of corticosteroid eyedrops given 8 times daily for the first postoperative week. In our patient cohort, there was no evidence that earlier postoperative exposure to corticosteroid eyedrops (ie, on the day of surgery) had an effect on the incidence of DLK ($P = .5193$).

We found no effect of flap thickness on the incidence of DLK ($P = .4574$). In our previous analysis of factors affecting incidence of DLK after femtosecond LASIK with varied laser

frequency,¹⁵ we found a higher incidence of DLK with thinner flaps in patients treated with the 30 kHz femtosecond laser. This was not the case in the 60 kHz group in the previous analysis, and the lack of an association between DLK and flap thickness was maintained in the current study.

We found no other preoperative or treatment variables associated with increased risk for DLK. This includes no significant association with preoperative meibomian gland dysfunction or patient atopy, both of which have been found to be associated with an increased risk for DLK in previous studies.^{9–11}

Most cases of DLK in our study were stage 1 or 2 and resolved within 2 weeks with minimal sequelae. Although patients who developed DLK were less likely than those who did not to achieve a 20/20 CDVA 1 day after LASIK ($P = .0453$), this difference did not persist after 1 day and most patients with DLK (96.7%) achieved a UDVA better than 20/40 by 1 week postoperatively.

Although our study is limited by its retrospective observational design, to our knowledge it is the only one to have found an association between flap diameter and the incidence of DLK. Our study findings suggest that a larger flap diameter is associated with an increased risk for DLK. Our results also confirm the higher incidence of DLK with higher femtosecond energy settings, especially raster and side-cut energy. Finally, we found that DLK after LASIK with femtosecond laser flap creation tends to be mild, with little effect on visual acuity.

Acknowledgments

Supported by National Institutes of Health/National Eye Institute grant EY017885 (Dr. Shtein) and a departmental grant from Research to Prevent Blindness, New York, NY, USA.

References

1. Cannady, K. Comprehensive Report on the Global Refractive Surgery Market. 16. Market Scope, LLC; 2011. 2011 Available at: http://dev.market-scope.com/market_reports/2012/01/2011-comprehensive-report-on-t-7.html
2. Smith RJ, Maloney RK. Diffuse lamellar keratitis; a new syndrome in lamellar refractive surgery. *Ophthalmology*. 1998; 105:1721–1726. [PubMed: 9754183]
3. Parolini B, Marcon G, Panozzo GA. Central necrotic lamellar inflammation after laser in situ keratomileusis. *J Refract Surg*. 2001; 17:110–112. [PubMed: 11310759]
4. Holland SP, Mathias RG, Morck DW, Chiu J, Slade SG. Diffuse lamellar keratitis related to endotoxins released from sterilizer reservoir biofilms. *Ophthalmology*. 2000; 107:1227–1233. discussion by EJ Holland, 1233–1234. [PubMed: 10889090]
5. Yuhan KR, Nguyen L, Boxer Wachler BS. Role of instrument cleaning and maintenance in the development of diffuse lamellar keratitis. *Ophthalmology*. 2002; 109:400–403. discussion by SN Rao, RJ Epstein, 403–404. [PubMed: 11825830]
6. Hoffman RS, Fine IH, Packer M, Reynolds TP, Van Bebber C. Surgical glove-associated diffuse lamellar keratitis. *Cornea*. 2005; 24:699–704. [PubMed: 16015089]
7. Hadden OB, McGhee CNJ, Morris AT, Gray TB, Ring CP, Watson ASJ. Outbreak of diffuse lamellar keratitis caused by marking-pen toxicity. *J Cataract Refract Surg*. 2008; 34:1121–1124. [PubMed: 18571079]
8. Rosman M, Chua W-H, Tseng PSF, Wee T-L, Chan W-K. Diffuse lamellar keratitis after laser in situ keratomileusis associated with surgical marker pens. *J Cataract Refract Surg*. 2008; 34:974–979. [PubMed: 18499004]
9. Fogla R, Rao SK, Padmanabhan P. Diffuse lamellar keratitis: are meibomian secretions responsible? [letter]. *J Cataract Refract Surg*. 2001; 27:493–495. [PubMed: 11393150]

10. Ambrósio R Jr, Periman LM, Netto MV, Wilson SE. Bilateral marginal sterile infiltrates and diffuse lamellar keratitis after laser in situ keratomileusis. *J Refract Surg.* 2003; 19:154–158. [PubMed: 12701721]
11. Boorstein SM, Henk HJ, Elnor VM. Atopy: a patient-specific risk factor for diffuse lamellar keratitis. *Ophthalmology.* 2003; 110:131–137. [PubMed: 12511358]
12. Shah MN, Misra M, Wilhelmus KR, Koch DD. Diffuse lamellar keratitis associated with epithelial defects after laser in situ keratomileusis. *J Cataract Refract Surg.* 2000; 26:1312–1318. [PubMed: 11020615]
13. Noda-Tsuruya T, Toda I, Asano-Kato N, Hori-Komai Y, Fukumoto T, Tsubota K. Risk factors for development of diffuse lamellar keratitis after laser in situ keratomileusis. *J Refract Surg.* 2004; 20:72–75. [PubMed: 14763474]
14. Linebarger EJ, Hardten DR, Lindstrom RL. Diffuse lamellar keratitis: diagnosis and management. *J Cataract Refract Surg.* 2000; 26:1072–1077. [PubMed: 10946202]
15. Choe CH, Guss C, Musch DC, Niziol LM, Shtein RM. Incidence of diffuse lamellar keratitis after LASIK with 15 KHz, 30 KHz, and 60 KHz femtosecond laser flap creation. *J Cataract Refract Surg.* 2010; 36:1912–1918. [PubMed: 21029900]
16. Haft P, Yoo SH, Kymionis GD, Ide T, O'Brien TP, Culbertson WW. Complications of LASIK flaps made by the IntraLase 15- and 30-kHz femtosecond lasers. *J Refract Surg.* 2009; 25:979–984. [PubMed: 19921765]
17. Binder PS. One thousand consecutive IntraLase laser in situ keratomileusis flaps. *J Cataract Refract Surg.* 2006; 32:962–969. [PubMed: 16814054]
18. Gil-Cazorla R, Teus MA, de Benito-Llopis L, Fuentes I. Incidence of diffuse lamellar keratitis after laser in situ keratomileusis associated with the IntraLase 15 kHz femtosecond laser and Moria M2 microkeratome. *J Cataract Refract Surg.* 2008; 34:28–31. [PubMed: 18165077]
19. Moshirfar M, Gardiner JP, Schliesser JA, Espandar L, Feiz V, Mifflin MD, Chang JC. Laser in situ keratomileusis flap complications using mechanical microkeratome versus femtosecond laser: retrospective comparison. *J Cataract Refract Surg.* 2010; 36:1925–1933. [PubMed: 21029902]
20. Thammano P, Rana AN, Talamo JH. Diffuse lamellar keratitis after laser in situ keratomileusis with the Moria LSK-One and Carriazo-Barraquer microkeratomes. *J Cataract Refract Surg.* 2003; 29:1962–1968. [PubMed: 14604718]
21. McLeod SD, Tham VM-B, Phan ST, Hwang DG, Rizen M, Abbott RL. Bilateral diffuse lamellar keratitis following bilateral simultaneous versus sequential laser in situ keratomileusis. *Br J Ophthalmol.* 2003; 87:1086–1087. Available at: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1771840/pdf/bjo08701086.pdf>. [PubMed: 12928271]
22. Hoffman RS, Fine IH, Packer M. Incidence and outcomes of LASIK with diffuse lamellar keratitis treated with topical and oral corticosteroids. *J Cataract Refract Surg.* 2003; 29:451–456. [PubMed: 12663005]
23. Johnson JD, Harissi-Dagher M, Pineda R, Yoo S, Azar DT. Diffuse lamellar keratitis: incidence, associations, outcomes, and a new classification system. *J Cataract Refract Surg.* 2001; 27:1560–1566. [PubMed: 11687352]
24. Lin RT, Maloney RK. Flap complications associated with lamellar refractive surgery. *Am J Ophthalmol.* 1999; 127:129–136. [PubMed: 10030552]
25. Wilson SE, Ambrósio R Jr. Sporadic diffuse lamellar keratitis (DLK) after LASIK. *Cornea.* 2002; 21:560–563. [PubMed: 12131030]
26. Netto MV, Mohan RR, Medeiros FW, Dupps WJ Jr, Sinha S, Krueger RR, Stapleton WM, Rayborn M, Suto C, Wilson SE. Femtosecond laser and microkeratome corneal flaps: comparison of stromal wound healing and inflammation. *J Refract Surg.* 2007; 23:667–676. Available at: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2698458/pdf/nihms118773.pdf>. [PubMed: 17912936]
27. Kim JY, Kim MJ, Kim T-I, Choi H-J, Pak JH, Tchah H. A femtosecond laser creates a stronger flap than a mechanical microkeratome. *Invest Ophthalmol Vis Sci.* 2006; 47:599–604. Available at: <http://www.iovs.org/cgi/reprint/47/2/599>. [PubMed: 16431956]

WHAT WAS KNOWN

- Diffuse lamellar keratitis has been reported to occur at higher frequency with femtosecond LASIK; however, the specific parameters associated with development of DLK are not well understood.

WHAT THIS PAPER ADDS

- Larger flap diameter, higher femtosecond raster, and side-cut energy are associated with an increased risk for DLK.
- Diffuse lamellar keratitis after LASIK with femtosecond laser flap creation tends to be mild, with little effect on visual acuity.

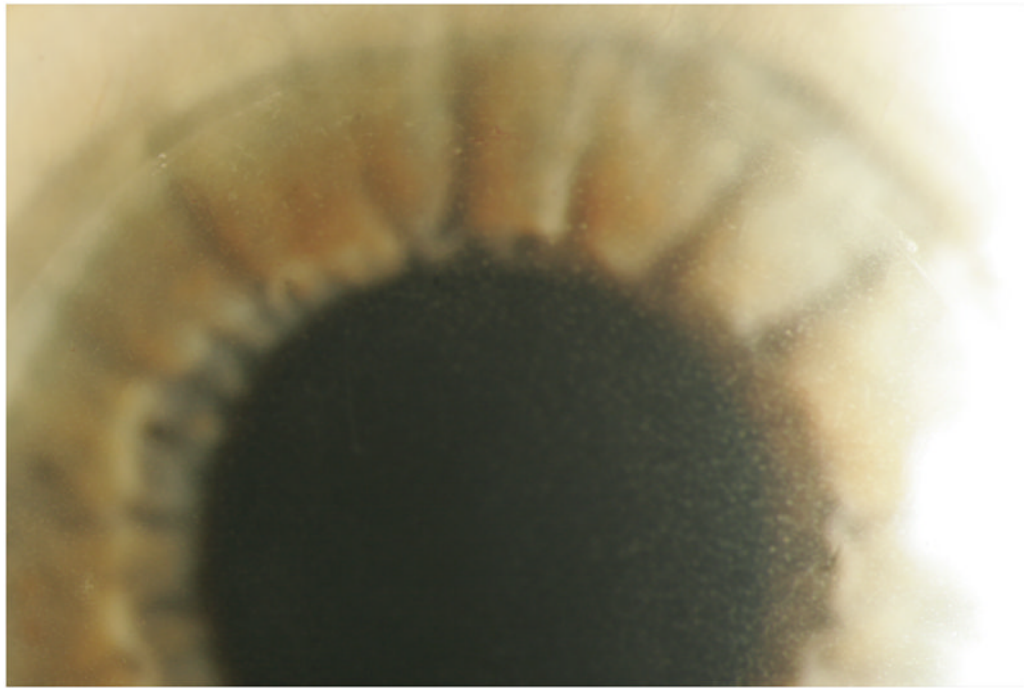


Figure 1.
An eye with grade 1 DLK 1 day after LASIK with flap creation using a 60 kHz femtosecond laser (clinical slitlamp photograph).
An increased risk for developing DLK after femtosecond LASIK was associated with a higher energy level for flap creation and a larger flap diameter.

Table 1

Demographics of the groups with DLK and without DLK.

Parameter	No DLK	DLK	<i>P</i> Value*
Patients (n)	349	70	—
Eyes, n (%)	702 (87.6)	99 (12.4)	—
Mean age (y) ± SD	38.5 ± 10.5	38.1 ± 9.4	.7674
Sex, n (%)			.9494
Female	193 (55.3)	39 (55.7)	
Male	156 (44.7)	31 (44.3)	

DLK = diffuse lamellar keratitis

* *t* test for continuous variables; chi-square test for categorical variables

Table 2

Incidence and rate of DLK by stage.

DLK Stage	Frequency (n)	Rate (%)
1	90	11.2
2	8	1.0
3	1	0.1

DLK = diffuse lamellar keratitis

Table 3

Treatment parameters in the groups with DLK and without DLK.

Parameter	Mean \pm SD		P Value*
	No DLK (n = 702)	DLK (n = 99)	
Flap thickness (μm)	117.0 \pm 6.5	116.8 \pm 7.3	.4574
Flap diameter (mm)	8.7 \pm 0.3	8.8 \pm 0.2	.0171
Side-cut energy (μJ)	1.89 \pm 0.47	2.04 \pm 0.20	.0037
Raster energy (μJ)	1.55 \pm 0.20	1.62 \pm 0.11	.0033
Optical zone (mm)	6.6 \pm 0.4	6.5 \pm 0.4	.6659
Ablation depth (μm)	86.6 \pm 36.8	83.9 \pm 38.1	.7311
Residual bed (μm)	356.1 \pm 42.1	357.4 \pm 37.8	.8952
Treated SE (D)	-3.7 \pm 1.8	-3.8 \pm 1.9	.7806

DLK = diffuse lamellar keratitis; SE = spherical equivalent

*From generalized estimating equation model adjusting for inter-eye correlation

Table 4

Visual acuity outcomes 1 day and 1 week after LASIK.

Parameter	No DLK, n (%)	DLK, n (%)	OR*	(95% CI)	P Value [†]
UDVA 1 d postop					
20/20	495 (70.6)	62 (62.6)	0.75	0.26, 2.11	.5792
20/25–20/40	182 (26.0)	31 (31.3)	0.81	0.30, 2.17	.6745
<20/40	24 (3.4)	6 (6.1)	—	—	—
CDVA 1 d postop					
20/20	564 (80.7)	72 (72.7)	0.37	0.14, 0.98	.0453
20/25–20/40	129 (18.5)	24 (24.2)	0.48	0.18, 1.24	.1304
<20/40	6 (0.9)	3 (3.0)	—	—	—
UDVA 1 wk postop					
20/20	483 (72.0)	60 (65.9)	1.39	0.27, 7.27	.6960
20/25–20/40	172 (25.6)	28 (30.8)	1.52	0.28, 8.40	.6303
<20/40	16 (2.4)	3 (3.3)	—	—	—
CDVA 1 wk postop [‡]					
20/20	579 (86.3)	76 (83.5)	0.75	0.47, 1.19	.2158
<20/20	92 (13.4)	15 (16.5)	—	—	—

CDVA = corrected distance visual acuity; CI = confidence interval; DLK = diffuse lamellar keratitis; OR = odds ratio; UDVA = uncorrected distance visual acuity

* Calculated versus <20/40 or versus <20/20 for last row

[†] From generalized estimating equation model adjusting for inter-eye correlation

[‡] Sample size in the <20/40 range was only 2; thus, the 20/25–20/40 and <20/40 categories were combined.