Traumatic Flap Striae 6 Years After LASIK: Case Report and Literature Review

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ABSTRACT

PURPOSE: To report a case of traumatic flap striae without flap dislocation 6 years after LASIK and provide a literature review of surgical flap striae, late traumatic flap striae, and their management.

METHODS: A 28-year-old man presented with late traumatic flap striae without concurrent flap dislocation, which closely approximated the longest reported interval between LASIK and the development of flap striae.

RESULTS: In the absence of flap dislocation, the finding of striae alone was subtle and went undetected initially. The flap was successfully refloated, stretched, and smoothed with recovery of 20/20 vision.

CONCLUSIONS: Traumatic LASIK flap complications may occur many years after the original procedure. This report presents the first case of late traumatic flap striae without concurrent flap dislocation. Proper management can restore good visual function. [*J Refract Surg*. 2009;xx:xxx-xxx.]

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Flap striae are recognized complications of LASIK and present most frequently in the intraoperative and immediate postoperative periods.[1] The LASIK flap remains vulnerable to late striae formation in the context of trauma and flap dislocation.[2] Although reports of late traumatic striae are fortunately rare, they suggest that flaps remain at risk for months to years after surgery.[3-11] The full duration of risk is unknown but, to our knowledge, traumatic striae have presented up to 7 years after LASIK,[3] always in the setting of simultaneous flap dislocation (Table).[3-11]

We describe a case of isolated flap striae without concurrent flap dislocation due to trauma 6 years after LASIK. We also report the visualization of striae by anterior segment optical coherence tomography (AS-OCT) and our subsequent management technique, followed by a literature review of flap striae and their management.

CASE REPORT

A 28-year-old Caucasian man was referred to our office for the management of new LASIK flap striae with loss of corrected distance visual acuity (CDVA) in the right eye after blunt trauma. In July 2002, he had undergone uncomplicated bilateral LASIK at a private refractive surgery center for full correction of myopia with astigmatism of $-3.75\ +0.50 \times 62°$ in the right eye and $-3.75\ +0.50 \times 68°$ in the left eye. A Moria 130-µm microkeratome (Moria, Antony, France) was used to create superiorly hinged flaps with excimer laser ablation. One day postoperatively, uncorrected distance visual acuity (UDVA) was 20/30 in the right eye and 20/20 in the

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TABLE

Summary of Traumatic Flap Striae Cases ≥6 months After LASIK

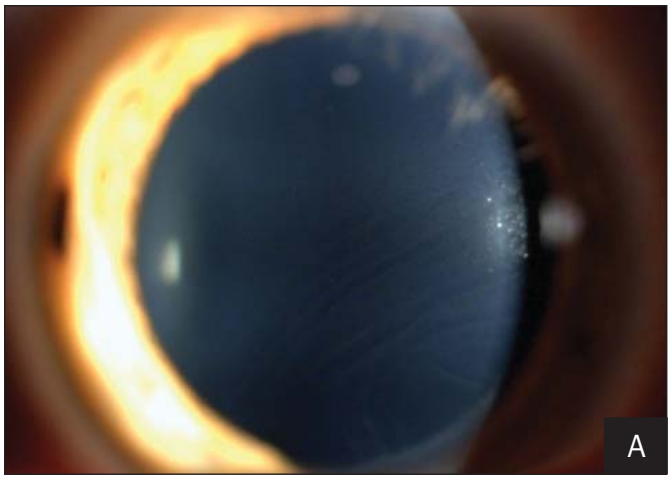
Authors (Year)	Age (y)/Sex	Eye	Time After LASIK (mo)	Traumatic Agent	Concurrent Dislocation	Technique	Final VA
Cheng et al[3] (2006)	23/M	Left	84	Fingernail	Yes	Lift, scrape, stretch-smooth	20/20
Jin & Merkley[4] (2006)	33/M	Left	51	Wood chip	Yes	Lift, scrape, stretch-smooth, perform enhancement	20/20
Ramirez et al[5] (2007)	29/M	Right	48	Airbag	Yes	Lift, scrape, stretch-smooth	20/30
Nilforoushan et al[6] (2005)	28/F	Left	47	Human fist	Yes	Lift, scrape, stretch-smooth	20/20
Iskander et al[7] (2001)	20/F	Right	38	Airbag	Yes	Lift, stretch-smooth	20/20
Tumbocon et al[8] (2003)	25/M	Right	23*	Canine paw	Yes	Lift, scrape, stretch-smooth	20/20
Heickell et al[9] (2004)	20/M	Right	17	Tree branch	Yes	Manage conservatively†	20/20
Pereira et al[10] (2006)	40/F	Left	12*	Electric sander	Yes	Lift, scrape, stretch-smooth, suture	20/20
Iskander et al[7] (2004)	32/M	Left	11	Cable	Yes	Lift, stretch-smooth	20/20
Tumbocon et al[8] (2003)	35/M	Left	7	Rock	Yes	Lift, scrape, stretch-smooth	20/30
Geggel and Coday [11] (2001)	37/M	Left	6	Tree branch	Yes	Lift, use hypotonic saline, create epithelial breaks	20/20

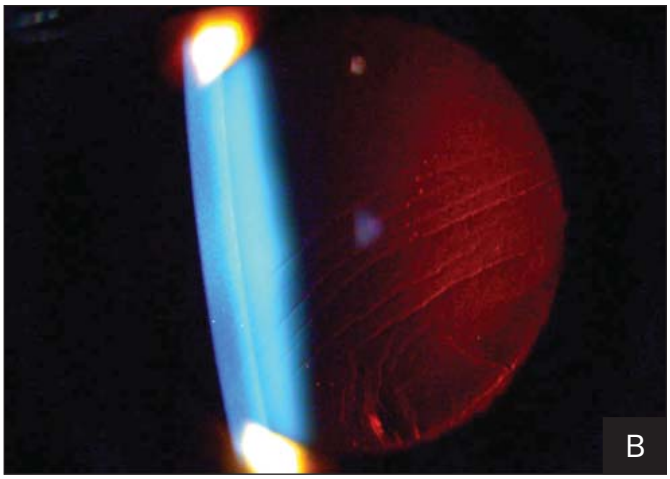
VA = visual acuity
**After repeat LASIK (enhancement).*
†Striae were not visually significant.

left eye. The patient was subsequently lost to follow-up. Six uneventful years after LASIK, he was punched in the right eye, which resulted in pain and immediate decreased vision. Prompt emergency room examination revealed right eye UDVA of 20/70 with upper lid lacerations and iris hyphema with normal intraocular pressures (IOP). The fellow eye was uninjured. He was treated for lacerations and pain.

Three days later, UDVA was 20/70^{-1} and CDVA was 20/40^{-1} in the right eye with an IOP of 5 mmHg. Slit-lamp examination of the right eye revealed periorbital ecchymoses, lid lacerations, flat scant subconjunctival hemorrhage, microhyphema, and deep angles without recession. Funduscopic examination was unremarkable. Prednisolone acetate 1% (Pred Forte; Allergan Inc, Irvine, Calif) drops were started in the right eye, four times daily. Blurry vision persisted 2 weeks after trauma and CDVA was 20/40^{-2} with a manifest refraction of $-0.25 +1.25 \times 37°$. Slit-lamp examination revealed previously unseen oblique wrinkles in the central flap, bisecting the flap from 2 o'clock to 7 o'clock, and in the periphery without exposed stromal bed. He was treated with aggressive artificial tear lubrication and referred to our office 25 days after initial trauma for management of the flap striae and astigmatism. Corrected distance visual acuity had improved to 20/30 but remained blurry and bothersome to the patient. Multiple grade three[2] central and peripheral flap striae were visualized on slit-lamp examination and best seen with retroillumination. The flap did not appear dislocated at any margin and was otherwise clear without haze or epithelial ingrowth. Flap striae, but not flap dislocation, were also noted on AS-OCT (Visante OCT; Carl Zeiss Meditec, Dublin, Calif) (Fig 1). The patient elected to proceed with punctal plug placement and continue conservative management.

Due to persistent blurry vision 2 months after trauma, the patient consented to a stretch and smooth procedure. The flap was lifted and refloated with a hypotonic balanced salt solution (BSS) -H^2O mix (50:50). A moist cellulose sponge (WECK-CEL; Medtronic Xomed Inc, Jacksonville, Fla) was used both to stretch the flap perpendicular to the striae and to smooth the stromal bed. Additional stretching was performed prior to repositioning and irrigation under the flap. Prednisolone acetate 1% and





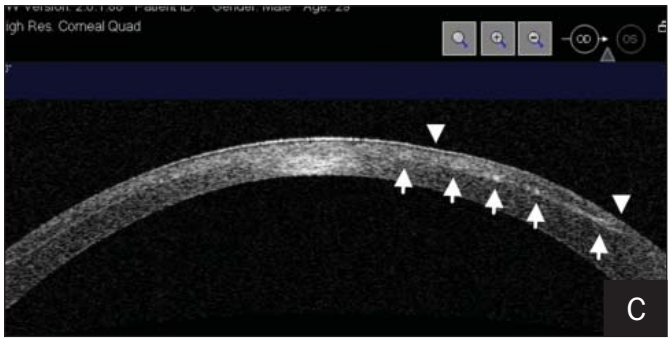


Figure 1. A) Slit-lamp photograph of the right eye at presentation demonstrates multiple oblique flap striae involving the central LASIK flap. Perpendicular striae are also seen overlying the inferonasal iris border. **B)** Striae appear more prominently with retroillumination. **C)** Anterior segment optical coherence tomography (Visante OCT, Carl Zeiss Meditec) shows focal interface hyperreflectivity where striae intersect the horizontal meridian (arrows). The associated epithelial surface is mildly raised (between arrowheads) compared to the unaffected temporal side. The flap margin appears intact (rightmost arrowhead).

gatifloxacin 0.3% (Zymar, Allergan Inc) drops were instilled and a bandage contact lens and plastic protective eye shield were placed. One week later, UDVA was $20/25^{+2}$ and no striae were seen on slit-lamp examination.

At 1-month postoperative follow-up, the patient reported 3 days' duration of redness with foreign body sensation on prednisolone acetate 1% and ketorolac 0.5% (Acular, Allergan Inc) drops and was found to have superficial punctate keratitis. Uncorrected distance visual acuity was 20/20 and the flap was otherwise clear and well-positioned without evidence of striae, stromal haze, or epithelial ingrowth. Symptoms improved on gatifloxacin 0.3% and artificial tears. Vision remained stable 7 months after trauma, and no striae were detected.

DISCUSSION

Flap striae are historically among the most common complications associated with the corneal flap.[12] Prevalence varied between 3.4% and 20% in some of the earliest LASIK studies[13-17] and between 0.8% and 8.5% at the turn of the millennium.[18] Rates from newer keratomileusis data approach zero.[19,20] Many predisposing factors have been identified in the effort to minimize risk, both patient- and surgeon-related. Most authors agree that at least one inherent patient factor, high myopia, favors flap striae. Because deeper stromal bed ablation is needed, curvature mismatch with the posterior flap is exaggerated,[21] the so-called tenting effect.[22] Other authors emphasize prevention via patient education to avoid postoperative factors such as eye rubbing, squeezing, and blinking as well as operator errors involving drop applicators (contact with the flap) and protective eye shields (malpositioning, noncompliance).[2]

Similarly, multiple surgical variables have been examined. One retrospective study of 120 myopic eyes that underwent LASIK with a microkeratome found peripheral striae in 7% of thin flaps (<110 µm) as compared to 2% of medium flaps (110 to 140 µm) and none in thick flaps (>140 µm).[23] However, flap thickness per se may not factor as heavily as variation in thickness within each flap. A retrospective series of 3009 eyes after thin-flap (sub-Bowman's) keratomileusis with femtosecond laser–generated thin flaps reported no striae.[20] A microkeratome reported to create predictably thin flaps was used in 42 eyes without striae.[24] Other intraoperative etiologies of striae include poor alignment after flap placement, movement of the flap, improper insertion of bandage contact lens, and removal of eyelid speculum.[25] High volume surgeons (>10 procedures/week) reported fewer striae than their low volume counterparts,[26] and beginning surgeons may have rates as high as 10% initially.[1]

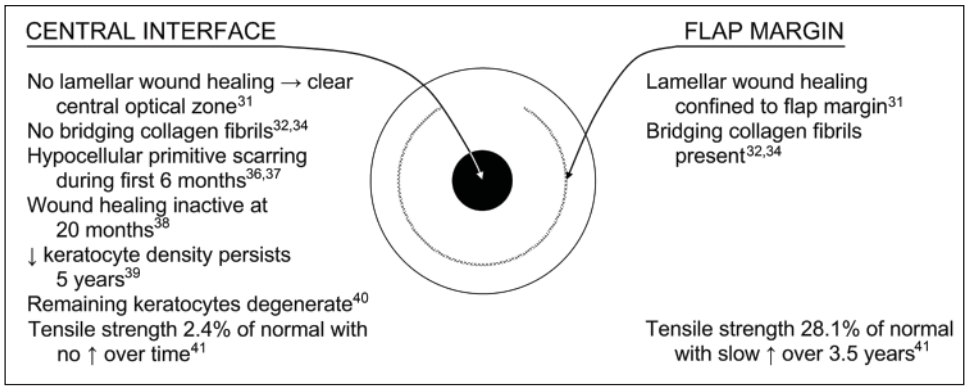


Figure 2. Wound healing of the LASIK flap.

Intraoperative striae may escape detection under the operating microscope.[27] The observation that they preferentially appear against a funduscopic red reflex[25] forms the basis for prompt postoperative dilated slit-lamp evaluation using both direct and retroillumination.[2] However, a minority of striae will still remain invisible. Fluorescein examination under cobalt filter reveals uneven pooling of tear film 1 to 2 seconds after blinking and is more sensitive than slit-lamp with retroillumination.[28] In general, slit-lamp screening is sufficient in the absence of unexplained decrease in CDVA. Using these methods, most striae can be identified within the first 24 to 48 hours.

Even so, flap striae formation is not confined to the intraoperative and immediate postoperative periods. Delayed trauma has been shown to cause flap defects,[2] demonstrating that LASIK flaps remain vulnerable to traumatic dehiscence and dislocation even 6 or 7 years after surgery.[3,29] Not all traumatic flap dislocations are associated with flap striae but, to date, all late traumatic flap striae have been associated with flap dislocations. We reviewed 11 cases of traumatic flap striae occurring ⩾6 months after LASIK or last retreatment (Table).[3-11] The longest of these was 7 years.[3] All 11 cases featured flap striae presenting jointly with flap dislocations. For the majority of these cases, collisions with everyday objects such as fingernails,[3] airbags,[5,7] fists,[6,8] and tree branches[9,11] generated sufficient force to cause flap trauma.

Diminished wound healing at the LASIK flap–bed interface prevents loss of corneal transparency but also weakens flap adhesion (Fig 2). Lamellar wound healing is confined to the flap margin, leaving the central optical zone clear in both rabbits[30] and humans.[31,32] Bridging collagen fibrils are absent at the flap–bed interface except at the flap edge in rabbits[33] and humans.[32,34] Animal studies showed that wound healing in the interface region is disorganized and delayed up to 9 months after LASIK in rabbits.[35] In human eyes, a hypocellular primitive scar forms during the first 6 months after LASIK[36,37] and wound healing appears inactive at 20 months.[38] Deficits in keratocyte density adjacent to the LASIK interface persist 5 years after LASIK[39] and remaining keratocytes at the interface display degenerative changes.[40] Not surprisingly, tensile strength of LASIK wounds is worst centrally (2.4% of normal) and modestly better at the flap edge (28.1% of normal),[41] although these ex vivo data do not account for adhesion due to endothelial transport.[42] Central wound healing did not increase over time while healing of the flap margin increased over more than 3 years after LASIK. These findings are consistent with the clinical observation that LASIK flaps are easily lifted once fibrous adhesions at peripheral flap edges are interrupted, even many years after LASIK.[32,41] Furthermore, if interface transparency is indicative of absent wound healing, one might expect that the interface remains a potential space[43] and flap adhesion is impaired for the lifetime of the flap.

We report an unusual case of late traumatic flap striae without concurrent flap dislocation. The 6-year delay between LASIK and acquisition of striae approaches the 7 years reported elsewhere.[3] In contrast to that case and similar reports of late traumatic striae, our patient did not present with associated flap dislocation, showing that traumatic shear forces can wrinkle the flap without first disrupting the strongest adhesions at the flap edge. The presentation was further delayed when striae were not identified by routine slit-lamp examination until 2 weeks after trauma. Initial screening by ophthalmologists less experienced in refractive surgery may miss post-traumatic striae, which often can be difficult to recognize. It is possible that due to the associated ocular pathology of anterior segment inflammation with microhyphema, flap striae were not considered as a cause of decreased visual acuity. Translating these observations into practice, flap striae should be considered and ruled out in any postoperative LASIK patient who experiences ocular trauma. Late trauma on the scale of years does not itself rule out flap striae, especially when there is unexplained loss of CDVA. The absence of flap dislocation upon presentation also does not itself rule out striae.

Unlike the 11 previous cases of late traumatic flap striae,[3-11] this report finds that dislocation is not a prerequisite for all flap striae. The flap shifts that occur during dislocation indubitably predispose to striae but are not required. Minimal flap shifts can cause striae and we speculate that these may occur with the recruitment of tissue laxity from elsewhere, perhaps due to persistence of the tenting effect, poor interface healing in the central optical zone, or both. Such striae may be subtle and require fluorescein or additional studies, sometimes even AS-OCT, for prompt detection. Although AS-OCT can detect flap dislocations with greater sensitivity than slit-lamp microscopy,[44] this is the first demonstration that AS-OCT is also effective in the visualization of subtler flap striae.

Once detected, striae can be classified using the Probst grading system,[2] but generally those involving the visual axis contribute most to impairment of CDVA[45] and require treatment. Numerous methods have been described including the stretch and smooth technique using blunt forceps,[22] sponge spears, Caro irons,[12] or hyperthermic spatulas[46]; flap hydration using hypotonic saline ranging from 50% to 80% BSS[8] to deionized water[47]; epithelial debridement[8,48,49]; interrupted[50] or running flap sutures[51-53]; reverse geometry contact lens[54]; flap applanation; conductive keratoplasty to tighten the central cornea[55]; transepithelial phototherapeutic keratectomy (PTK)[56]; and wavefront-guided photorefractive keratectomy with mitomycin C.[57] These treatments have been used in various permutations, even at the slit lamp.[51,58]

Flap lifting and refloating is accepted as initial management by most authors followed by their choice of the aforementioned maneuvers.[1,2,27] Striae usually persist immediately following treatment with only marginal empirical improvement. Instead, they often disappear by postoperative day 1,[2] suggesting that the hydration status of the flap and stromal proteoglycans[45] may play a role after repositioning. Some authors therefore advocate hypotonic irrigation and hydration, whereas others caution that the risk of keratocyte lysis may outweigh the benefits of a more hydrating osmotic gradient.[25,48] Likewise, some authors report success with gentle epithelial abrasion localized to areas of striae to release traction.[8] Because epithelial defect increases the risk of diffuse lamellar keratitis and stromal haze,[48] others reserve abrasion for mature or refractory striae or epithelial ingrowth. Use of PTK involves similar considerations. Fortunately, nearly all published cases of haze secondary to epithelial debridement or PTK of striae were steroid-responsive. Additional PTK risks include hyperopic shift and irregular astigmatism. Suturing incurs a theoretical risk of folds or astigmatism.

Most authors agree that striae should be diagnosed early and managed promptly because flap flexibility is progressively lost and irregularities become more permanent over time.[2,27] Stretching and smoothing, for example, is most appropriate in the first 2 weeks and becomes less likely to succeed as striae mature. Successful treatment may then require additional, more intensive maneuvers such as flap suturing, epithelial abrasion, or PTK of Bowman's layer. Gas-permeable contact lenses may be indicated for truly refractory striae with persistent astigmatism. Although the first 24 to 48 postoperative hours is a recommended target window to avoid those scenarios, good results are frequently reported within the first week and successful repositioning has been described at the slit-lamp up to 2 weeks after surgery.[51] Regardless, higher success rates with promptly managed striae should not discourage attempts to treat longer-standing striae. Anecdotal evidence suggests that patients perceive disproportionately greater improvement from otherwise modest objective gains in vision.[25]

Currently, no consensus treatment exists for visually significant flap striae. We are similarly unaware of indications to treat traumatic striae differently than postoperative striae. Instead, our opinion is that refractive surgeons choose from the myriad available options based on grade, maturity, and treatment history for any given case of striae. In the presented case, conservative therapy was continued for 2 months with modest improvement. Flap refloating with hypotonic saline and the described stretch and smooth technique achieved a successful result at that time, despite 8 weeks elapsing since the trauma. Our positive outcome was similar to that reported for late traumatic striae with flap dislocation, most of which underwent repositioning, scraping, and stretching.[3-11] Final UDVA was 20/30 or better in all cases and 20/20 in 82%.

Nevertheless, the best course of action remains prevention. It is imperative that late traumatic flap complications should be discussed with prospective LASIK patients. Military, law enforcement, and contact sport personnel should be counseled to consider surface ablation or wear eye protection. Regarding the latter, however, we recommend that safety counseling be broadened to all patients in light of the mundane mechanisms of injury seen and the random nature of trauma. Given the growing evidence for chronic flap vulnerability and the relative youth of many LASIK recipients, long-term risks are increasingly relevant. This case describes the first occurrence of late flap trauma to generate striae without dislocation. It approximates the longest reported interval after LASIK and is also the first to describe the successful use of AS-OCT for visualization of flap striae.

AUTHOR CONTRIBUTIONS

Study concept and design (R.U.); data collection (M.T.F.); interpretation and analysis of data (R.U., M.T.F.); drafting of the manuscript (R.U., M.T.F.); critical revision of the manuscript (R.U., M.T.F.); supervision (R.U.)

REFERENCES

1. Schallhorn SC, Amesbury EC, Tanzer DJ. Avoidance, recognition, and management of LASIK complications. *Am J Ophthalmol*. 2006;141:733-739.
2. Probst LE. Myopic and hyperopic LASIK complications. In: Probst LE, ed. *LASIK: Advances, Controversies, and Customs*. Thorofare, NJ: SLACK Incorporated; 2003:150-152.
3. Cheng AC, Rao SK, Leung GY, Young AL, Lam DS. Late traumatic flap dislocations after LASIK. *J Refract Surg*. 2006;22:500-504.
4. Jin GJ, Merkley KH. Laceration and partial dislocation of LASIK flaps 7 and 4 years postoperatively with 20/20 visual acuity after repair. *J Refract Surg*. 2006;22:904-905.
5. Ramirez M, Quiroz-Mercado H, Hernandez-Quintela E, Narango-Tackman R. Traumatic flap dislocation 4 years after LASIK due to air bag injury. *J Refract Surg*. 2007;23:729-730.
6. Nilforoushan MR, Speaker MG, Latkany R. Traumatic flap dislocation 4 years after laser in situ keratomileusis. *J Cataract Refract Surg*. 2005;31:1664-1665.
7. Iskander NG, Peters NT, Anderson Penno EE, Gimbel HV. Late traumatic flap dislocation after laser in situ keratomileusis. *J Cataract Refract Surg*. 2001;27:1111-1114.
8. Tumbocon JA, Paul R, Slomovic A, Rootman DS. Late traumatic displacement of laser in situ keratomileusis flaps. *Cornea*. 2003;22:66-69.
9. Heickell AG, Vesaluoma MH, Tervo TM, Vannas A, Krootila K. Late traumatic dislocation of laser in situ keratomileusis flaps. *J Cataract Refract Surg*. 2004;30:253-256.
10. Pereira Cda R, Narvaez J, King JA, Seery LS, Gimbel HV. Late-onset traumatic dislocation with central tissue loss of laser in situ keratomileusis flap. *Cornea*. 2006;25:1107-1110.
11. Geggel HS, Coday MP. Late-onset traumatic laser in situ keratomileusis (LASIK) flap dehiscence. *Am J Ophthalmol*. 2001;131:505-506.
12. Machat JJ. LASIK complications and their management. In: Machat JJ, ed. *Excimer Laser Refractive Surgery: Practice and Principles*. Thorofare, NJ: SLACK Incorporated; 1996:359-400.
13. Buratto L, Ferrari M, Rama P. Excimer laser intrastromal keratomileusis. *Am J Ophthalmol*. 1992;113:291-295.
14. Buratto L, Ferrari M, Genisi C. Myopic keratomileusis with the excimer laser: one-year follow up. *Refract Corneal Surg*. 1993;9:12-19.
15. Pérez-Santonja JJ, Bellot J, Claramonte P, Ismail MM, Alió JL. Laser in situ keratomileusis to correct high myopia. *J Cataract Refract Surg*. 1997;23:372-385.
16. Gimbel HV, Basti S, Kaye GB, Ferensowicz M. Experience during the learning curve of laser in situ keratomileusis. *J Cataract Refract Surg*. 1996;22:542-550.
17. Kremer I, Blumenthal M. Myopic keratomileusis in situ combined with VISX 20/20 photorefractive keratectomy. *J Cataract Refract Surg*. 1995;21:508-511.
18. Lin RT, Maloney RK. Flap complications associated with lamellar refractive surgery. *Am J Ophthalmol*. 1999;127:129-136.
19. Kato N, Toda I, Hori-Komai Y, Sakai C, Tsubota K. Five-year outcome of LASIK for myopia. *Ophthalmology*. 2008;115:839-844.e2.
20. Chang JS. Complications of sub-Bowman's keratomileusis with a femtosecond laser in 3009 eyes. *J Refract Surg*. 2008;24:S97-S101.
21. Charman WN. Mismatch between flap and stromal areas after laser in situ keratomileusis as source of flap striae. *J Cataract Refract Surg*. 2002;28:2146-2152.
22. Probst LE, Machat J. Removal of flap striae following laser in situ keratomileusis. *J Cataract Refract Surg*. 1998;24:153-155.
23. Esquenazi S, Bui V, Grunstein L, Esquenazi I. Safety and stability of laser in situ keratomileusis for myopic correction performed under thin flaps. *Can J Ophthalmol*. 2007;42:592-599.
24. Duffey RJ. Thin flap laser in situ keratomileusis: flap dimensions with the Moria LSK-One manual microkeratome using the 100-microm head. *J Cataract Refract Surg*. 2005;31:1159-1162.
25. Ambrosio R Jr, Wilson SE. Complications of laser in situ keratomileusis: etiology, prevention, and treatment. *J Refract Surg*. 2001;17:350-379.
26. Solomon KD, Holzer MP, Sandoval HP, Vargas LG, Werner L, Vroman DT, Kasper TJ, Apple DJ. Refractive Surgery Survey 2001. *J Cataract Refract Surg*. 2002;28:346-355.
27. von Kulajta P, Stark WJ, O'Brien TP. Management of flap striae. *Int Ophthalmol Clin*. 2000;40:87-92.
28. Rabinowitz YS, Rasheed K. Fluorescein test for the detection of striae in the corneal flap after laser in situ keratomileusis. *Am J Ophthalmol*. 1999;127:717-718.
29. Landau D, Levy J, Solomon A, Lifshitz T, Orucov F, Strassman E, Frucht-Pery J. Traumatic corneal flap dislocation one to six years after LASIK in nine eyes with a favorable outcome. *J Refract Surg*. 2006;22:884-889.
30. Pérez-Santonja JJ, Linna TU, Tervo KM, Sakla HF, Alió y Sanz JL, Tervo TM. Corneal wound healing after laser in situ keratomileusis in rabbits. *J Refract Surg*. 1998;14:602-609.
31. Philipp WE, Speicher L, Gottinger W. Histological and immunohistochemical findings after laser in situ keratomileusis in human corneas. *J Cataract Refract Surg*. 2003;29:808-820.
32. Priglinger SG, May CA, Alge CS, Wolf A, Neubauer AS, Haritoglou C, Kampik A, Welge-Lussen U. Immunohistochemical findings after LASIK confirm in vitro LASIK model. *Cornea*. 2006;25:331-335.
33. Park CK, Kim JH. Comparison of wound healing after photorefractive keratectomy and laser in situ keratomileusis in rabbits. *J Cataract Refract Surg*. 1999;25:842-850.
34. Rumelt S, Cohen I, Skandarani P, Delarea Y, Ben Shaul Y, Rehany U. Ultrastructure of the lamellar corneal wound after laser in situ keratomileusis in human eye. *J Cataract Refract Surg*. 2001;27:1323-1327.
35. Kato T, Nakayasu K, Hosoda Y, Watanabe Y, Kanai A. Corneal wound healing following laser in situ keratomileusis (LASIK): a histopathological study in rabbits. *Br J Ophthalmol*. 1999;83:1302-1305.
36. Dawson DG, Holley GP, Geroski DH, Waring GO III, Grossniklaus HE, Edelhauser HF. Ex vivo confocal microscopy of human LASIK corneas with histologic and ultrastructural correlation. *Ophthalmology*. 2005;112:634-644.
37. Dawson DG, Kramer TR, Grossniklaus HE, Waring GO III, Edelhauser HF. Histologic, ultrastructural, and immunofluorescent evaluation of human laser-assisted in situ keratomileusis corneal wounds. *Arch Ophthalmol*. 2005;123:741-756.
38. Anderson NJ, Edelhauser HF, Sharara N, Thompson KP,

Rubinfeld RS, Devaney DM, L'Hernault N, Grossniklaus HE. Histologic and ultrastructural findings in human corneas after successful laser in situ keratomileusis. *Arch Ophthalmol*. 2002;120:288-293.

39. Erie JC, Patel SV, McLaren JW, Hodge DO, Bourne WM. Corneal keratocyte deficits after photorefractive keratectomy and laser in situ keratomileusis. *Am J Ophthalmol*. 2006;141:799-809.
40. Mootha VV, Dawson D, Kumar A, Gleiser J, Qualls C, Albert DM. Slitlamp, specular, and light microscopic findings of human donor corneas after laser-assisted in situ keratomileusis. *Arch Ophthalmol*. 2004;122:686-692.
41. Schmack I, Dawson DG, McCarey BE, Waring GO III, Grossniklaus HE, Edelhauser HF. Cohesive tensile strength of human LASIK wounds with histologic, ultrastructural, and clinical correlations. *J Refract Surg*. 2005;21:433-445.
42. Bissen-Miyajima H, Nakamura K, Kaido M, Shimmura S, Tsubota K. Role of the endothelial pump in flap adhesion after laser in situ keratomileusis. *J Cataract Refract Surg*. 2004;30:1989-1992.
43. Dawson DG, Schmack I, Holley GP, Waring GO III, Grossniklaus HE, Edelhauser HF. Interface fluid syndrome in human eye bank corneas after LASIK: causes and pathogenesis. *Ophthalmololgy*. 2007;114:1848-1859.
44. Ustundag C, Bahcecioglu H, Ozdamar A, Aras C, Yildirim R, Ozkan S. Optical coherence tomography for evaluation of anatomical changes in the cornea after laser in situ keratomileusis. *J Cataract Refract Surg*. 2000;26:1458-1462.
45. Hernandez-Matamoros J, Iradier MT, Moreno E. Treating folds and striae after laser in situ keratomileusis. *J Cataract Refract Surg*. 2001;27:350-352.
46. Donnenfeld ED, Perry HD, Doshi SJ, Biser SA, Solomon R. Hyperthermic treatment of post-LASIK corneal striae. *J Cataract Refract Surg*. 2004;30:620-625.
47. Muñoz G, Alió JL, Pérez-Santonja JJ, Attia WH. Successful treatment of severe wrinkled corneal flap after laser in situ keratomileusis with deionized water. *Am J Ophthalmol*. 2000;129:91-92.
48. Kuo IC, Ou R, Hwang DG. Flap haze after epithelial debridement and flap hydration for treatment of post-laser in situ keratomileusis striae. *Cornea*. 2001;20:339-341.
49. Buhren J, Kohnen T. Corneal wound healing after laser in situ keratomileusis flap lift and epithelial abrasion. *J Cataract Refract Surg*. 2003;29:2007-2012.
50. Jackson DW, Hamill MB, Koch DD. Laser in situ keratomileusis flap suturing to treat recalcitrant flap striae. *J Cataract Refract Surg*. 2003;29:264-269.
51. Lichter H, Russell GE, Waring GO III. Repositioning the laser in situ keratomileusis flap at the slit lamp. *J Refract Surg*. 2004;20:166-169.
52. Mackool RJ, Monsanto VR. Sequential lift and suture technique for post-LASIK corneal striae. *J Cataract Refract Surg*. 2003;29:785-787.
53. Biser SA, Bloom AH, Donnenfeld ED, Perry HD, Solomon R, Doshi S. Flap folds after femtosecond LASIK. *Eye Contact Lens*. 2003;29:252-254.
54. Lin JC, Rapuano CJ, Cohen EJ. RK4 lens fitting for a flap striae in a LASIK patient. *Eye Contact Lens*. 2003;29:76-78.
55. Pallikaris IG, Naoumidi TL, Astyrakakis NI. Conductive keratoplasty to correct hyperopic astigmatism. *J Refract Surg*. 2003;19:425-432.
56. Ashrafzadeh A, Steinert RF. Results of phototherapeutic keratectomy in the management of flap striae after LASIK before and after developing a standardized protocol: long-term follow up of an expanded patient population. *Ophthalmology*. 2007;114:1118-1123.
57. Kuo IC, Jabbur NS, O'Brien TP. Photorefractive keratectomy for refractory laser in situ keratomileusis flap striae. *J Cataract Refract Surg*. 2008;34:330-333.
58. Solomon R, Donnenfeld ED, Perry HD, Doshi S, Biser S. Slitlamp stretching of the corneal flap after laser in situ keratomileusis to reduce corneal striae. *J Cataract Refract Surg*. 2003;29:1292-1296.