Idiopathic Recurrence of Diffuse Lamellar Keratitis After LASIK

George D. Kymionis, MD, PhD; Vassilios F. Diakonis, MD; Dimitrios I. Bouzoukis, MD; Ileana Lampropoulou, MD, PhD; Aristofanis I. Pallikaris, MSc, PhD

ABSTRACT

PURPOSE: To report a case of late recurrence of bilateral diffuse lamellar keratitis (DLK) after LASIK.

METHODS: A 39-year-old woman presented in the early postoperative period with bilateral DLK after hyperopic LASIK and was treated with topical steroids. One year after and with no obvious cause (idiopathic), recurrence of the same stage (stage III) of disease was observed.

RESULTS: Slit-lamp examination revealed diffuse, multifocal, and granular haze in the interface. The microbiology culture was negative. Confocal microscopy demonstrated multiple activated keratocytes, debris, and inflammatory cells adjacent to the flap interface. After intense treatment with topical corticosteroids, DLK resolved and corneal transparency was achieved with complete restoration of visual acuity.

CONCLUSIONS: Diffuse lamellar keratitis may recur in LASIK patients with previous episodes without an obvious cause (idiopathic). Early diagnosis and treatment with topical corticosteroids can achieve complete resolution without visual loss—even in advanced stages of DLK. [*J Refract Surg*. 2007;23:720-721.]

Diffuse lamellar keratitis (DLK) is a relatively infrequent complication. In the literature, the reported incidence of this complication varies depending on the etiology and the severity, from 0.2% in mild cases to 30% in so-called epidemic DLK.[1] The diagnosis is made during the first postoperative days (1-4) because inflammation tends to increase in the first 6 postoperative days.

Late-onset DLK associated with variable conditions after LASIK (ie, epithelial defects, trauma, iritis, viral conjunctivitis) has been reported.[2-5] This report describes a patient who presented 12 months postoperatively with bilateral recurrent DLK without any of the conditions (idiopathic) currently reported to occur after LASIK.

From the Department of Ophthalmology (Kymionis) and the Institute of Vision and Optics (Kymionis, Diakonis, Bouzoukis, Pallikaris), University of Crete; and the Department of Microbiology, University of Athens (Lampropoulou), Greece.

The authors have no financial or proprietary interest in the materials presented herein.

Correspondence: George D. Kymionis, MD, PhD, Institute of Vision and Optics, University of Crete, Medical School, Dept of Ophthalmology, 71110 Heraklion, Crete, Greece. Tel: 30 2810 371800; Fax: 30 2810 394653; E-mail: kymionis@med.uoc.gr

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CASE REPORT

Uneventful bilateral LASIK for hyperopia and astigmatism was performed in a 39-year-old woman 12 months prior to presentation. No history of atopy or other ocular or systemic disease was noted. No surgical complications occurred; and on the first postoperative day, no infiltrates or epithelial defects were found. On the third postoperative day, the patient presented with light sensitivity. Multiple infiltrates scattered diffusely throughout the interface were found in both eyes (DLK stage III). Hourly topical steroids (dexamethasone 1%) were added for 6 days. During the following 3 weeks, topical steroids were continued, tapering the dose as the inflammatory infiltrate at the interface diminished.

Three weeks after the last follow-up examination (12 months postoperatively), the patient presented with a 2-day history of bilateral decrease of vision without any concomitant symptoms. The corrected visual acuity was 20/63 in the right eye and 20/80 in the left eye. The patient had no history of infection, trauma, or any other symptoms (conjunctival hyperemia, tearing, etc) that correlated with the recurrence of DLK.

Slit-lamp examination showed multiple areas of interface infiltrates in both eyes (stage III) (Fig 1). Intraocular pressure was normal (measured at the corneal center and periphery using applanation tonometry [mean 12 mmHg and 14 mmHg, respectively]). The remainder of the anterior (fluorescein staining on the corneal surface or at the flap edge, reaction in the anterior chamber, or discharge) and posterior segment examination was within normal limits. Conjunctival microbiology culture showed a negative result.

During the first postoperative examination, confocal microscopy exhibited multiple active keratocytes with spindle-shaped nuclei and mononuclear cells sparsely arranged between the keratocyte nuclei. Clumped remnants of the infiltrate and interface debris were observed adjacent to the flap interface (Fig 2). A diagnosis of late recurrent idiopathic DLK was made and corticosteroids (dexamethasone 1%, hourly) were prescribed in combination with a tapering course of oral steroid, starting at 30 mg once a day. The inflammation responded rapidly to the treatment and by day 9, the infiltrates had resolved and both eyes were comfortable with an improvement in visual acuity. The corticosteroids were tapered over the next 5 weeks without any evidence of DLK recurrence. Two months later, the

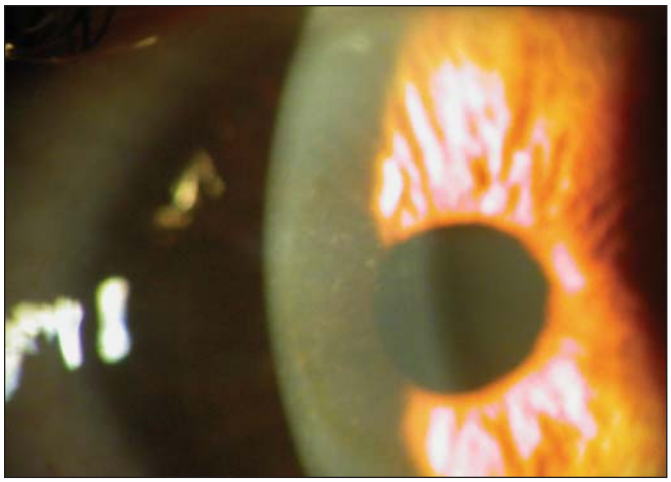

Figure 1. Slit-lamp photograph of the right eye demonstrates DLK with diffuse faint haze and clumped opacities in the flap interface. Additional corneal iron ring deposits outside the ablated area were observed.

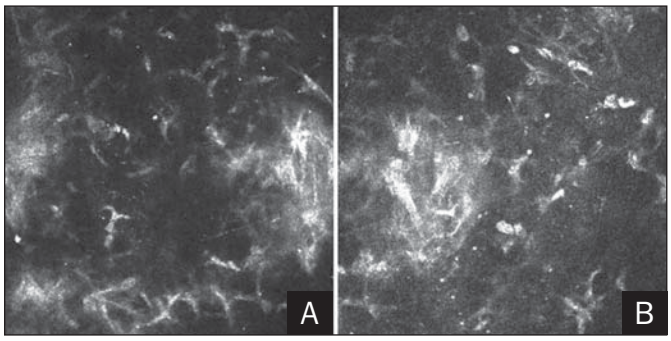

Figure 2. Confocal scanning laser (Heidelberg Retina Tomography II/ Rostock Cornea Module) microscopic image of the flap interface at the initial examination demonstrates **A)** increased keratocyte activation as stellate figures and **B)** sparsely arranged mononuclear cells. In addition, both images exhibit small debris and clumped remnants of the infiltrate with increased scattering.

uncorrected visual acuity and best spectacle-corrected visual acuity had returned to the pre-episode levels.

DISCUSSION

This report describes a patient who presented 12 months after LASIK with recurrent bilateral DLK and none of the conditions (idiopathic) currently reported to occur after LASIK. It is possible that during LASIK, the initial etiologic factor (previously inert agent) of the first episode of DLK was activated by an unknown (possible allergic) factor that led to the recurrence of DLK. Although both episodes of DLK (initial and late recurrence) were at the same advanced stage (stage III) of disease, they rapidly responded to topical and systemic steroids without surgical intervention. Therefore, early diagnosis and treatment with intensive steroids could resolve inflammation and restore vision.

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Small Spot Phototherapeutic Keratectomy for Recurrent Corneal Erosion

John A. van Westenbrugge, MD

ABSTRACT

PURPOSE: To describe small spot phototherapeutic keratectomy (PTK) using the NIDEK EC-5000 excimer laser for the treatment of recurrent corneal erosion.

METHODS: This retrospective study analyzed patients with recurrent corneal erosion who were treated using small spot PTK. An electronic medical records database was used to retrieve patient data.

RESULTS: Ten consecutive eyes in nine patients were evaluated after small spot PTK. Eight (80%) recovered without further erosion.

CONCLUSIONS: Small spot PTK is a safe and effective alternative to traditional PTK and to corneal micropuncture in the treatment of recurrent corneal erosion. [*J Refract Surg*. 2007;23:721-724.]

Recurrent corneal erosion is a relatively common condition that causes significant pain, decreased vision in some patients, and frequent down time. Conservative measures are rarely, if ever, effective.

From the Gimbel Eye Centre, Calgary, Alberta, Canada.

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Correspondence: John A. van Westenbrugge, MD, Gimbel Eye Centre, 450, 4935 – 40 Ave NW, Calgary, Alberta, Canada T3A 2N1. Tel: 403.286.3022; Fax: 403.286.2943; E-mail: miferens@gimbel.com

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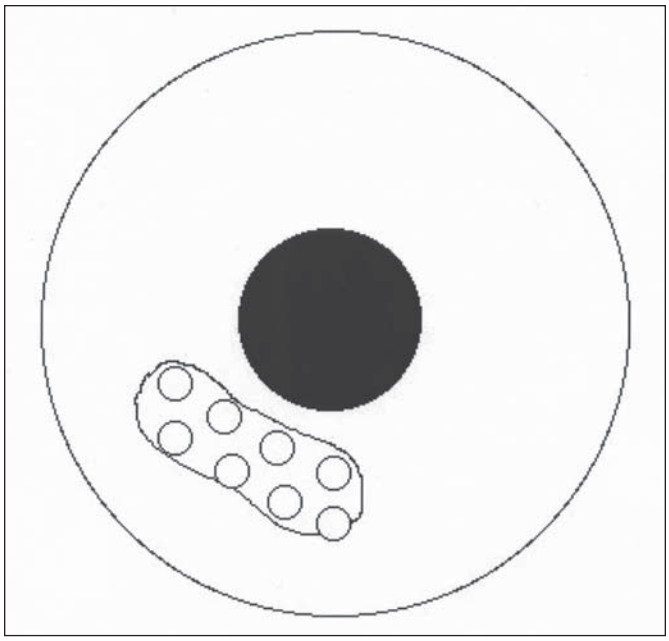

Figure. Schematic of small spot PTK with the NIDEK excimer laser shows treatment pattern for loose epithelium caused by recurrent corneal erosion.

The two methods currently favored for more definitive treatment have disadvantages.[1-5] In corneal micropuncture, small scars are created with a sterile hypodermic needle. The tip is bent to avoid the risk of corneal perforation. The punctures usually are distributed 0.5 to 1.0 mm apart over the affected area. The epithelium and stroma (only a small fraction of a millimeter) are penetrated under topical anesthesia. In my experience, the eye typically responds with inflammation and significant discomfort. Therefore, for corneal micropuncture, I usually treat aggressively with a bandage contact lens, antibiotic and mild steroids (fluorometholone 0.1%) four times daily, and diclofenac 0.1% every 6 hours as needed to improve comfort. The contact lens usually can be removed within 24 hours if desired, but can be kept in for a week or more to allow the fibrosis process of adherence to occur at the micropuncture sites. One disadvantage of this method is the small permanent white marks left on the cornea. The marks do not seem to be optically significant, and treatment has been performed across the visual axis.[6,7] Further, it does not address the issue of a poor quality surface that caused the recurrent corneal erosion to begin with.

The alternative to micropuncture is to resurface the central cornea with a wide area removal of epithelium (6- to 8-mm diameter) and a phototherapeutic keratectomy (PTK) removal of 4 to 10 µm of stroma uniformly across the whole area. Recommendations in the literature vary as to the depth that is effective.[8-10] Postoperative recovery is similar to photorefractive keratectomy (PRK), with blurred vision and discomfort for the first 4 to 7 days. In addition, this tissue removal may alter the refraction in the hyperopic direction.

TECHNIQUE

Having observed the limitations of the above two techniques, I devised an alternate treatment using the NIDEK EC-5000 (NIDEK Co Ltd, Gamagori, Japan) excimer laser.

As with micropuncture, the eye is observed after episodes of recurrent corneal erosion to determine the location of the loose epithelium. If conservative measures have failed and micropuncture or PTK would normally be considered as the next step, I then apply small spot PTK. The procedure is performed under topical anesthesia and a lid speculum is inserted. The area of loose epithelium is identified according to clinical diagrams. A cellulose sponge is applied gently to the area to clear excess surface fluid, avoiding tearing, wrinkling, or otherwise disrupting the epithelium. The epithelium frequently is very loose in these areas. The laser is in PTK mode, on manual setting to allow continuous ongoing laser output when the foot pedal is depressed, and set for a 1-mm treatment zone.

Room lights are very dim or turned off, and the laser illumination is turned as low as possible while still enabling visualization of the necessary landmarks to ensure that treatment is stable at one location. With the patient as still as possible while fixating on the flashing green diode of the laser, or any other recognizable stable spot straight ahead in the field of view, the laser treatment is then applied—keeping the treatment as steady on the selected spot as possible. If the lighting is appropriately low, a distinct light blue fluorescence is observed when the laser interacts with the epithelium. The number of microns of treatment is monitored. Generally at 50 µm, there is a breakthrough to the stroma in the center of the 1-mm spot. The treatment depth is noted on the NIDEK laser computer screen in microns and another 8- to 10-µm treatment is performed. Spots of treatment, approximately 1 mm apart, are repeated in a similar fashion until the entire loose epithelium area is covered (Fig). A bandage contact lens is then inserted and an antibiotic and fluorometholone 0.1% are taken four times daily. The bandage contact lens can be removed the next day and the medication discontinued based on the appearance of the eye and the epithelium.

ADVANTAGES AND DISADVANTAGES OF SMALL SPOT PTK FOR RECURRENT CORNEAL EROSION

Small spot PTK is significantly more comfortable than micropuncture and wide area PTK. Small spot PTK allows for faster recovery and achievement of stability. Unlike wide area PTK, I have not noticed a

TABLE

Treatment Parameters of Small Spot PTK for Recurrent Corneal Erosion Using the NIDEK EC-5000 Excimer Laser in 10 Eyes From 2001 to 2006

Patient Sex/Age (y)	Year	Eye	No. of Scans	Spot Size (mm)	No. of Spots	Symptoms to Date	Enhancement
M/53	2001	Right	374	0.5	4	RCE 2002	394 scans 6 spots 0.5 mm
F/43	2002	Right	562	0.5	4	—	—
F/59	2002	Left	962	0.5	9	—	—
M/28	2002	Right	380	0.5	4	—	—
F/31	2003	Left	739	1.0	8	—	—
M/58	2003	Left	567	1.0	7	RCE 2004	406 scans 8 spots 1.0 mm
M/44*	2004	Right	537	1.0	6	—	—
M/45*	2005	Left	867	1.0	11	—	—
M/49	2006	Right	553	1.0	8	—	—
F/58	2006	Right	773	1.0	10	—	—

RCE = recurrent corneal erosion
*Patient with bilateral small spot PTK.

change in refraction with small spot PTK. I have not used it in the central pupil zone across the visual axis because I have not had any recurrent corneal erosion cases involving the center. Unlike micropuncture, small spot PTK creates small areas of clean new surface for adherence, and the small depression of each spot should create additional lateral stability. Small spot PTK leaves no visible spots or scars.

As for disadvantages, small spot PTK requires the use of an excimer laser. As with micropuncture and wide area PTK, additional treatment may be required if the recurrent corneal erosion is not fully stabilized after the first small spot PTK. Moreover, small spot PTK has not yet been tested in the central cornea involving the pupil and visual axis, nor has it been tested using lasers other than the NIDEK excimer laser.

RESULTS

The Table summarizes the patient data since I began performing small spot PTK in 2001. To date, 10 eyes have been treated in 9 patients. Two eyes required retreatment for recurrent corneal erosion, whereas the others have remained asymptomatic.

DISCUSSION

Small spot PTK performed with a NIDEK excimer laser seems to offer a quick and comfortable alternative to conventional micropuncture and wide area PTK. It appears to be safe and as effective as the current techniques.

In order to formally establish its effectiveness and safety, a controlled study could be done. Corneal mapping could be used to determine its effect on the curvature of the epithelial surface after recovery. If no changes are observed in corneal curvature in the treatment area, then treatment of the central cornea should be safe as well.

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Effect of Mitomycin C on the Corneal Endothelium When Used for Corneal Subepithelial Haze Prophylaxis Following Photorefractive Keratectomy

Dennis H. Goldsberry, MD; Randy J. Epstein, MD; Parag A. Majmudar, MD; Rachel H. Epstein, BA; Richard F. Dennis, MD; Glenn Holley, MS; Henry F. Edelhauser, PhD

ABSTRACT

PURPOSE: To evaluate the potential effect of topical mitomycin C (MMC) on the corneal endothelium of myopic patients undergoing photorefractive keratectomy (PRK).

METHODS: Sixteen eyes with a planned ablation depth >75 μm underwent PRK followed by 0.02% MMC applied for 12 seconds using a methylcellulose sponge. Endothelial specular microscopy was performed with the Keeler-Konan specular photomicroscope in 16 eyes before and at least 1 year after surgery. Mean follow-up was 18 months (range: 12 to 24 months). Mean cell density, coefficient of variation of mean cell area, and percentage of hexagonal cells were measured and calculated using computerized morphometric analysis.

RESULTS: Mean endothelial cell densities before and after surgery were 2882±783 cells/mm^2 (range: 1511 to 4022 cells/mm^2) and 2867±588 cells/mm^2 (range: 1638 to 3881 cells/mm^2), respectively (P>.05). Mean coefficient of variation before and after surgery was 0.30±0.07 (range: 0.23 to 0.49) and 0.26±0.04 (range: 0.22 to 0.33), respectively (P=.06). Mean percentage of hexagonal cells before and after surgery was 61%±6.8% (range: 47% to 70%) and 66%±6.7% (range: 54% to 75%), respectively (P=.12).

CONCLUSIONS: Administration of MMC for haze prophylaxis following PRK did not have a significant effect on quantitative endothelial cell density or qualitative morphometric parameters in this study. [*J Refract Surg*. 2007;23:724-727.]

Mitomycin C (MMC) is a non-cell–cycle specific inhibitor of DNA synthesis that has been used over the past two decades for many ophthalmic applications, including corneal haze prevention following surface ablation with the excimer laser.[1-3] Mitomycin C has been shown to induce apoptosis in cultured keratocytes, and its mechanism of action in decreasing corneal haze may be related to its ability to inhibit myofibroblast transformation by stimulated keratocytes.[4]

Complications from MMC have been reported when it has been used for other ophthalmic applications, such as in glaucoma or pterygium surgery.[5-7] Because MMC preferentially targets actively proliferating cells, it would be expected to affect activated keratocytes rather than endothelial cells, which are not mitotically active.[8] However, endothelial cell toxicity has been noted with either prolonged application or when higher concentrations of MMC have been used.[9] Specular microscopy with morphometric analysis has been shown to be a sensitive indicator of endothelial cell function.[10,11] Thus, we evaluated the possible impact of low-dose, short-duration MMC on the corneal endothelium using pre- and postoperative specular microscopy, to assess endothelial cell density and morphology in myopic patients undergoing photorefractive keratectomy (PRK).

PATIENTS AND METHODS

Sixteen eyes of eight moderately myopic patients who were not candidates for LASIK underwent PRK with a Technolas 217z excimer laser (Bausch & Lomb, Rochester, NY). All patients had corneas that were too thin to safely undergo LASIK and planned ablation depths of >75 μm, placing them at higher risk for subepithelial haze formation after PRK.[12]

Photorefractive keratectomy was followed by a single 12-second application of 0.02% MMC to the central cornea using a methylcellulose sponge (Merocel Corneal Light Shield #22362; Medtronics Ophthalmic, Jacksonville, Fla). A Keeler-Konan specular photomicroscope (Konan Medical Inc, Hyogo, Japan) was used to image the corneal endothelium prior to surgery and at least 1 year postoperatively, with mean follow-up of 18 months (range: 12 to 24 months); computerized morphometric analysis was performed as previously

From the Cornea Service, Department of Ophthalmology, Rush University Medical Center (Goldsberry, R.J. Epstein, Majmudar, Dennis), and Loyola University (R.H. Epstein), Chicago, Ill; and the Department of Ophthalmology, Emory University (Holley, Edelhauser), Atlanta, Ga.

The authors have no proprietary interest in the materials presented herein.

Correspondence: Randy J. Epstein, MD, 1725 W Harrison St, Ste 928, Chicago, IL 60612. E-mail: repstein@chicagocornea.com

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TABLE

Mean Endothelial Cell Density and Morphometric Analysis Before and After Photorefractive Keratectomy With Mitomycin C in 16 Eyes

	Mean (Range)		
	Preoperative	**Postoperative**	***P* Value***
Endothelial cell density (cells/mm^2)	2882 (1511 to 4022)	2867 (1638 to 3881)	.68
Coefficient of variation	0.30 (0.23 to 0.49)	0.26 (0.22 to 0.33)	.06
Percentage of hexagonal cells	61 (47 to 70)	66 (54 to 75)	.12

**Mann-Whitney test.*

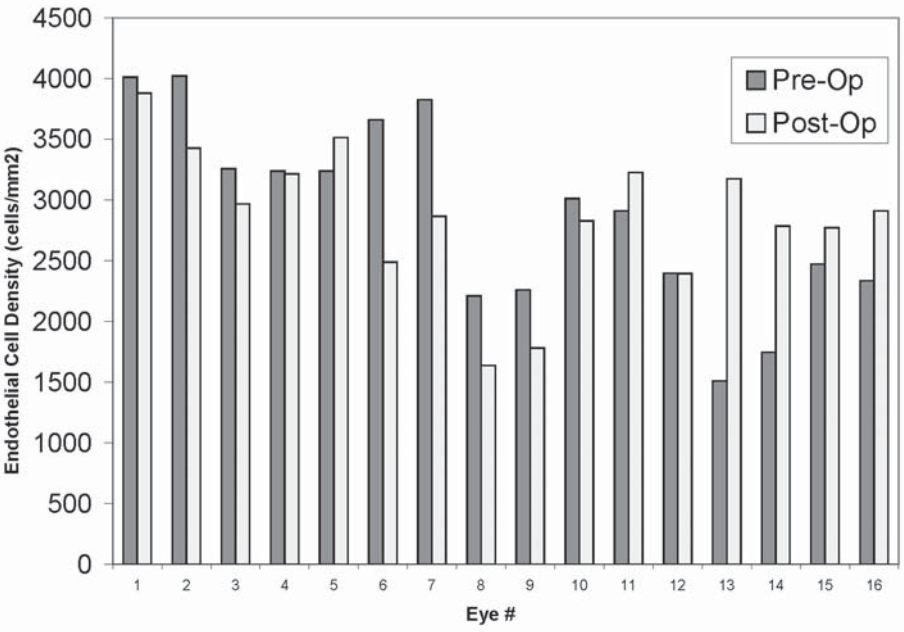

Figure 1. Endothelial cell densities from specular microscopy data for all 16 eyes demonstrating no significant difference between pre- and postoperative measurements (*P*=.68).

described.[10] Using a digitizing tablet, the endothelial cells were hand-traced and input into the computer using cell apices or corners method. Endothelial cell density, coefficient of variation, and percentage of hexagonal cells were compared using a non-parametric Mann-Whitney test for statistical significance (Minitab 14; Minitab Inc, State College, Pa).

RESULTS

Mean endothelial cell densities before and after surgery were 2882±783 cells/mm^2 (range: 1511 to 4022 cells/mm^2) and 2867±588 cells/mm^2 (range: 1638 to 3881 cells/mm^2), respectively—a difference that was not statistically significant (*P*=.68) (Table, Fig 1). Mean coefficient of variation before and after surgery was 0.30±0.07 (range: 0.23 to 0.49) and 0.26±0.04 (range: 0.22 to 0.33), respectively (Fig 2). This difference was also not significant (*P*=.06). Mean percentage of hexagonal cells before and after surgery was 61%±6.8% (range: 47% to 70%) and 66%±6.7% (range: 54% to 75%), respectively (Fig 3). The difference was not significant (*P*=.12). No patient developed postoperative haze.

DISCUSSION

Although the argument has been made that haze after PRK is now less common and possible to treat should it occur,[13] it is our strong belief that it is easier to prevent haze from forming in the first place.[14] Visual outcomes are generally superior,[15] and there is less morbidity to the cornea. In this regard, prophylactic MMC application may be a valuable adjunctive treatment in high-risk cases. However, concern exists regarding the potential toxicity of MMC, and therefore its risk/benefit ratio must be evaluated.

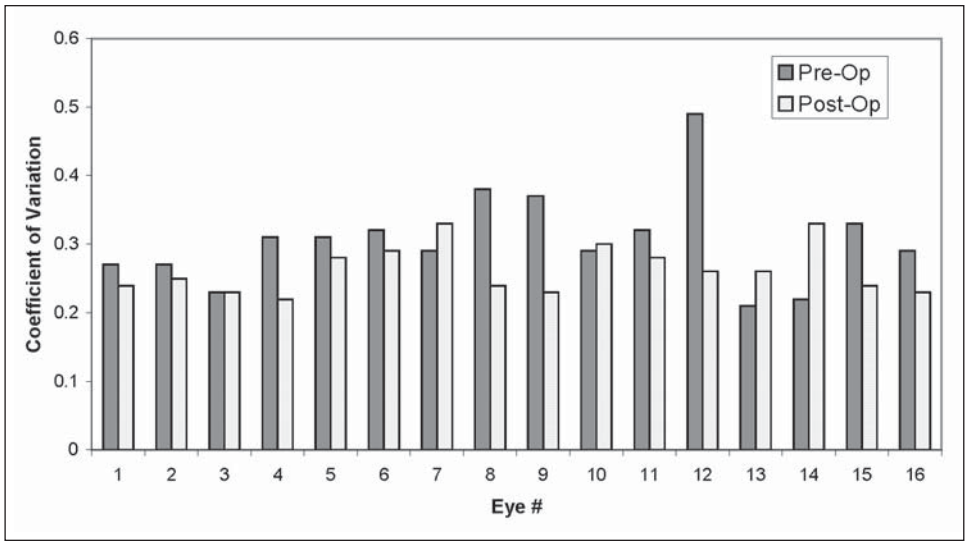

Figure 2. Coefficient of variation from morphometric analysis of specular microscopy data for all 16 eyes demostrating no significant difference between pre- and postoperative measurements (P=.06).

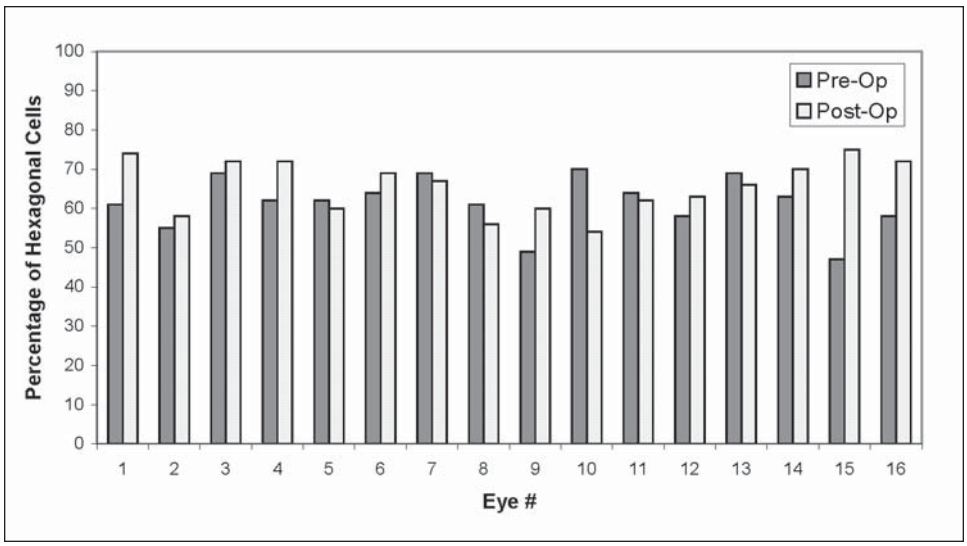

Figure 3. Percentage of hexagonal cells from morphometric analysis of specular microscopy data for all 16 eyes demonstrating no significant difference between pre- and postoperative measurements (P=.12).

Worldwide experience with topical MMC in PRK over the past 9 years has shown that the incidence of complications is low.[14-17] A number of researchers are in the process of using confocal microscopy to analyze the effects of MMC in vivo. Lee et al[16] demonstrated that endothelial cell density was not affected by MMC use following PRK in a large case series of >1000 eyes. Gambato et al[17] showed that activated keratocytes and extracellular matrix were present to a significant degree in untreated eyes versus eyes that had been treated with MMC intraoperatively, whereas the corneal endothelium remained unchanged in both groups. Furthermore, it may be possible that even lower doses of MMC are effective at preventing corneal haze, further minimizing potential complications.[18] Although the excimer laser itself has been shown to cause short-term transient endothelial changes, no long-term effect has been observed.[19,20]

In this study, the effect of topical 0.02% MMC (12 seconds) on corneal endothelial cell density, coefficient of variation of cell size, and percentage of hexagonal cells was found to be negligible when compared to the baseline values. No statistically significant differences were noted in any of the quantitative or qualitative endothelial morphometric parameters studied. Thus, the administration of MMC for haze prophylaxis following PRK did not have a significant effect on the corneal endothelium in this study.

Although this study is limited by a small population size and large range of preoperative endothelial cell densities, long-term follow-up data of >1 year, which is critical in a study of this nature, were obtained. Complications resulting from the use of MMC on the cornea have been reported, but only when used in higher doses and for longer durations than we have recommended in previous publications.[2,3] The lack of evidence indicating endothelial toxicity should provide surgeons with a higher comfort level when using MMC prophylactically in the manner reported herein.

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Aureobasidium pullulans Fungal Keratitis Following LASEK

Kenneth J. Maverick, MD; Michael S. Conners, MD

ABSTRACT

PURPOSE: To describe a patient who developed *Aureobasidium pullulans* keratitis following refractive laser epithelial keratomileusis (LASEK).

METHODS: A 52-year-old woman was referred to a tertiary care center 1 month after LASEK for treatment of a corneal ulcer that was unresponsive to conventional therapy. Mycology culture and fungal stain identified *Aureobasidium* as the infectious organism.

RESULTS: The infection responded well to treatment with topical natamycin and systemic itraconazole.

CONCLUSIONS: Treatment with topical natamycin and systemic itraconazole is effective against *Aureobasidium pullulans* keratitis. [*J Refract Surg*. 2007;23:727-729.]

Aureobasidium is a dematiaceous or "pigmented" fungus commonly isolated from plant debris, soil, wood, textiles, and indoor air environment. This yeast-like fungus frequently is found on caulk or damp window frames in bathrooms. *Aureobasidium* may be pink or black in color and is a type of mildew. Although it seldom causes infections, it is commonly allergenic.

Aureobasidium, commonly found on skin or beneath fingernails, is a rare cause of human infection, and a frequent contaminant in external cultures.[1] Rare cases of *Aureobasidium* infection have been confirmed, the most common being peritonitis in patients undergoing chronic peritoneal dialysis.[2] Rare cases of eye infections have been reported with one study identifying *Aureobasidium* as the causative agent in 1.6% of fungal ulcers.[3]

From Focal Point Vision Correction, San Antonio, Tex (Maverick); and Department of Ophthalmology, Washington University, St Louis, Mo (Maverick, Conners).

The authors have no proprietary interest in the materials presented herein.

Correspondence: Kenneth J. Maverick, MD, Focal Point Vision Correction, San Antonio, TX 78229. Tel: 314.454.9688; Fax: 314.362.3725; E-mail: maverick@focalpointvision.com

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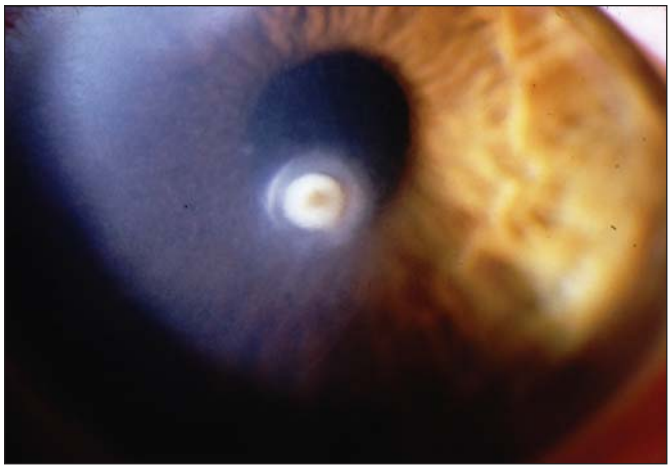

Figure 1. Slit-lamp photograph showing paracentral dense infiltrate with central black pigmentation and an overlying 0.75×0.75-mm epithelial defect with surrounding punctate keratopathy.

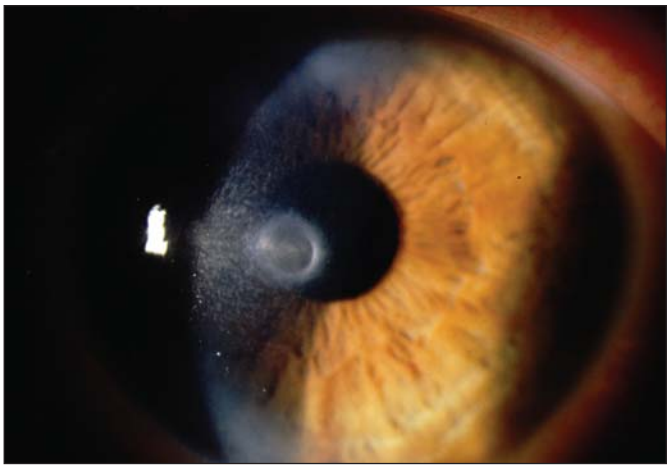

Figure 2. Slit-lamp photograph showing resolution of *Aureobasidium* keratitis after treatment with residual scar and stromal thinning.

CASE REPORT

A 52-year-old woman, who had undergone laser epithelial keratomileusis (LASEK), presented approximately 1 month after the initial procedure with redness, pain, and photophobia in the left eye. The right eye was unaffected. She developed an infiltrate and was treated with levofloxacin (Quixin; Santen Inc, Napa, Calif) ophthalmic drops every hour for 6 days. She was also treated with loteprednol etabonate (Lotemax; Bausch & Lomb, Rochester, NY) twice daily in the left eye. Because the patient's symptoms did not improve, topical gentamycin and cefazolin were prescribed every 30 minutes for 6 days. Again, no improvement occurred.

The patient was referred to a tertiary care center 42 days after LASEK for further evaluation. Uncorrected visual acuity was 20/25 in the right eye and 20/320 in the left eye, with a pin hole to 20/50. Tonometry pressures for right and left eyes were 13 and 10 mmHg, respectively. Examination of the right eye was normal. The left eye showed mild periorbital swelling with 3+ injection. A mixed papillary follicular reaction occurred. The left cornea showed mild superior subepithelial scarring. A dense paracentral infiltrate was seen with central black pigmentation and an overlying 0.75×0.75-mm epithelial defect with surrounding punctate keratopathy (Fig 1). The infiltrate was approximately 50% in depth. A mild anterior chamber reaction occurred. The lens was clear with fundus appearing normal. A culture taken 2 weeks prior showed pigmented yeast—reported preliminarily as *Exophiala*.

The corneal infiltrate was pan-cultured again with specimens sent on Lowenstein-Jensen agar and brain–heart infusion agar. The patient was admitted to the hospital and received hourly natamycin topical drops, polysporin ointment every 8 hours, and itraconozole 100 mg twice daily.

Over the next few days, the patient showed marked improvement with epithelial defect healing, infiltrate clearing, and eye quieting. Natamycin was slowly tapered, whereas itraconzole was continued systemically, and artificial tears and polysporin administered daily. After 1 month of continued improvement, treatment with loteprednol etabonate twice daily was started, with uncorrected vision subsequently reaching 20/32. The lesion healed with a 50% stromal defect that filled in with epithelium and a hazy scar (Fig 2).

DISCUSSION

This patient demonstrates infection by an ordinary, benign, ubiquitous, pigmented fungus following LASEK surgery. We believe the resulting keratitis was not caused by intraoperative LASEK instrument contamination as the fellow eye was uninvolved and no additional cases were reported. Furthermore, *Aureobasidium* is a relatively fast-growing fungus, reaching maturity after only 7 days of incubation. This patient presented 1 month after surgery, which suggests self-infection, possibly from a fingernail.

This case illustrates presentation and course of a pigmented fungal keratitis, which was unresponsive to topical antibiotic and steroid treatment. Although initially identified as a yeast, this yeast-like fungus matured into a uniquely pigmented appearance in the cornea and was confirmed as *Aureobasidium* by culture. *Aureobasidium* fungal keratitis responded rapidly to topical natamycin and systemic itraconazole.

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Traumatic Flap Dislocation 4 Years After LASIK Due to Air Bag Injury

Manuel Ramírez, MD; Hugo Quiroz-Mercado, MD;
Everardo Hernandez-Quintela, MD, MSc;
Ramón Naranjo-Tackman, MD

ABSTRACT

PURPOSE: To report a patient who developed corneal flap dislocation following air bag injury 48 months after LASIK.

METHODS: Evaluation by slit-lamp microscopy and fluorescein angiography.

RESULTS: A 29-year-old man was treated after air bag injury that occurred 48 months after LASIK. Examination revealed corneal flap dislocation, with severe folds and flap edema. Preoperative visual acuity was finger counting at 1 m. Visual acuity was 20/400 24 hours after repositioning the corneal flap. Retinal angiography revealed Berlin macular edema, which was injected with periocular steroids. Five days after injection, visual acuity remained 20/400, but improved to 20/40 1 month after injection.

CONCLUSIONS: Significant trauma can dislocate a corneal flap many months after surgery. [*J Refract Surg*. 2007;23:729-730.]

Laser in situ keratomileusis (LASIK) is one of the most frequently performed refractive surgery procedures. Complications of the corneal flap have been reported intraoperatively and after LASIK. These may include corneal flap folds, buttonholes,[1] and partial flap.[2] Corneal flap dislocation is a complication after LASIK that requires correction by repositioning.[3] We report traumatic corneal flap dislocation due to air bag injury 48 months after LASIK.

From the Departments of Cornea and Refractive Surgery (Ramírez, Hernandez-Quintela, Naranjo-Tackman) and Retina (Quiroz-Mercado), Asociación Para Evitar la Ceguera en México, Hospital "Luis Sánchez Bulnes," Universidad Nacional Autónoma de México, Mexico City, Mexico.

Supported by Asociación Para Evitar la Ceguera en México I.A.P., Mexico City, Mexico.

Correspondence: Manuel Ramírez, MD, Asociación Para Evitar la Ceguera en México, Hospital "Luis Sánchez Bulnes," Vicente García Torres #46, San Lucas Coyoacán 04030, Mexico City, Mexico. Tel: 525 659 2550; Fax: 525 659 2111; E-mail: mramirezf@medicasur.org.mx or mramirezfdz@igo.com.mx

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CASE REPORT

A 29-year-old man presented to the Cornea and Refractive Surgery Service with ocular pain in the right eye, severe photophobia, and decreased visual acuity secondary to air bag injury due to a car accident that occurred 2 days prior to presentation.

Visual acuity was finger counting at 1 m in the right eye and 20/20 in the left eye. Slit-lamp examination of the right eye revealed moderate upper eyelid edema, ciliary and conjunctival inflammation, and a nasal hinge corneal flap dislocated nasally with severe folds and flap edema. No loss of corneal flap tissue or corneal infiltrates compatible with diffuse lamellar keratitis was noted (Fig 1). A detailed retinal examination was not possible due to distortion from the flap folds and edema. The left eye was normal on slit-lamp examination.

The corneal flap was repositioned at the initial visit, which also involved epithelial debridement of the exposed stroma interface. Lifting and repositioning the flap to the normal position was difficult due to its abnormal position and fold memory. A bandage contact lens was placed at the end of the procedure. Postoperative treatment included topical tobramycin and dexamethasone (Tobradex; Alcon Laboratories Inc, Ft Worth, Tex) 4 times daily for 1 week, then tapered to 3 times daily for 2 weeks. The patient's visual acuity was 20/400 24 hours postoperatively. Slit-lamp examination revealed the corneal flap to be in a normal position with moderate edema and small folds outside of the visual axis (Fig 2).

Postoperative retinal examination of the posterior segment of the right eye revealed normal vitreous with no hemorrhage strings or retinal detachment, and Berlin's macular edema with grayish-white discoloration.

Retinal angiography showed early blocked fluorescence. Periocular betamethasone (Celestone Soluspan; Schering-Plough, SA de CV, Mexico DF, Mexico) injection was performed 1 day postoperatively. Visual acuity remained 20/400 5 days after injection. One month after periocular steroid injection, visual acuity improved to 20/40. Six months after injection, visual acuity was 20/30, and slit-lamp examination showed persistent small folds outside the visual axis.

DISCUSSION

The most common corneal complications following blunt trauma are flap wrinkling and flap dislocation. These complications are serious and require rapid sur-

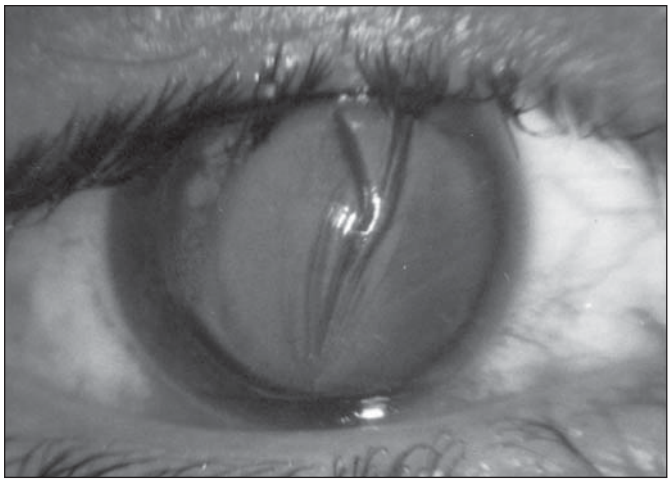

Figure 1. Corneal flap with nasal dislocation, severe folds, and flap edema 48 hours after air bag injury.

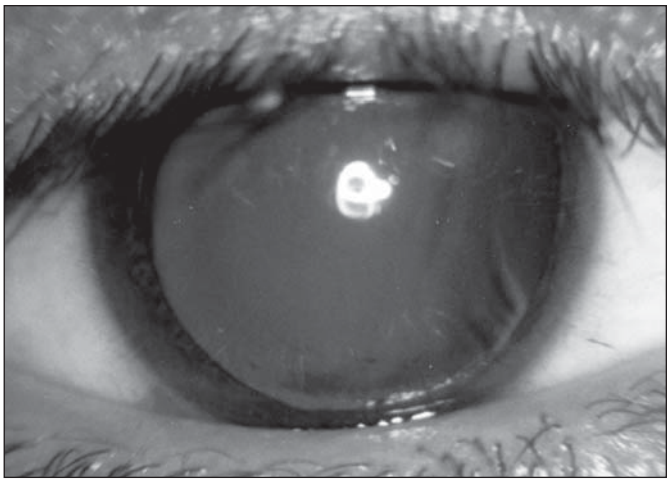

Figure 2. Corneal flap with normal flap position, moderate edema, and persistent small folds outside of the visual axis.

gical intervention.[3,4] The corneal flap can be easily displaced following trauma many months after LASIK.[3]

In review of the literature, two previous reports of air bag trauma after LASIK are described. Lemley et al[5] reported partial corneal flap dislocation and anterior chamber hyphema caused by air bag deployment 17 months after LASIK. Eight months after corneal flap repositioning visual acuity was 20/40. Norden et al[6] described air bag trauma 3 weeks after LASIK, resulting in flap folds, corneal edema, anterior chamber cellular reaction, and Berlin retinal edema. Repositioning of the corneal flap resulted in 20/20−2 visual acuity 1 month postoperatively. In our case, visual acuity was 20/40 at 6 weeks postoperatively (1 month after periocular steroid injection), which improved to 20/30 at 5-month follow-up. Berlin edema is a frequent complication that occurs after severe blunt ocular trauma, such as air bag injury,[7] although at least one previous report suggested this damage may be reversible.[8]

The healing process at the corneal flap wound interface persists for several months after LASIK, which consists of disorganized collagen fibers that can be seen along the interface of the corneal flap creating a hypocellular primitive stromal scar.[9,10] The risk of trauma, such as that associated with some occupations or participation in sports, should be discussed with the patient preoperatively and during follow-up to LASIK surgery.

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