Keratitis and Hyperopic Refractive Shift Induced by SLT

This case report presents a new complication of SLT.

BY ROBERT E. MARQUIS, MD, PHD

CASE HISTORY

A 55-year-old woman was referred to our practice for a glaucoma evaluation about 6 months ago. The referring doctor was concerned about her ocular hypertension, mild asymmetry in her cup-to-disc ratios, and a positive family history of glaucoma in her mother and uncle. The patient's medical history included restless leg syndrome and hypothyroidism, for which she took Mirapex (Boehringer Ingelheim Pharmaceuticals, Inc.) and Levothroid (Forest Laboratories, Inc.). The only other ocular history included mild epithelial basement membrane dystrophy and an uncomplicated blepharoplasty 3 years earlier.

EXAMINATION

On initial examination (Figure 1), the patient's BSCVA was 20/20- in each eye with a myopic refraction of -4.00 +1.25 X 5 OD and -3.75 +1.00 X 175 OS. The IOP measured 21 mm Hg OU by Goldman tonometry at 10:00 AM. The central corneal thickness measured 563 µm OD and 544 µm OS. The slit-lamp examination was normal in both eyes except for mild map-dot-fingerprint dystrophy noted superiorly and mild nuclear sclerosis. Gonioscopy revealed wide-open angles with mild trabecular pigmentation in both eyes. The cup-to-disc ratio was about 0.4 OD and 0.25 OS; mild relative thinning of the inferior rim was noted bilaterally. The remainder of the posterior pole was unremarkable.

TEST RESULTS, DIAGNOSIS, AND THERAPEUTIC PLAN

A 30-2 threshold frequency doubling technology perimetry (Humphrey Matrix; Carl Zeiss Meditec, Inc., Dublin, CA) showed superior nasal step changes consistent with the inferior retinal nerve fiber layer losses observed on optical coherence tomography in both eyes. I discussed these findings with the patient and made the diagnosis of open-angle glaucoma. The initial treatment options were discussed, including traditional topical medical therapies and selective laser trabeculoplasty (SLT). The patient elected to have SLT as the first-line therapy for her glaucoma.

SLT PROCEDURE

Three weeks after the initial evaluation, SLT was performed uneventfully on the patient's right eye. One hundred applications of 1.3 mJ were directed to the inferior 270° of the trabecular meshwork. The patient tolerated the procedure well, and she returned home after a 30-minute postoperative IOP check. The eye was treated for 1 week with Xibrom (bromfenac 0.09%; Ista Pharmaceuticals, Inc.) b.i.d. to limit postoperative inflammation.

On the follow-up visit 3 weeks later, the patient had no complaints, and her vision was normal in both eyes. Her visual acuity was unchanged, and the IOP went from 20 mm Hg OU preoperatively to 15 mm Hg OD and 18 mm Hg OS. The patient elected to have SLT in her left eye that day. The procedure was similar, although slightly less energy was applied: 98 pulses of 1.3 mJ were delivered to the inferior 270° of the trabecular meshwork of the left eye. Postoperative therapy was the same for the left eye as it had been for the right.

DEVELOPMENT OF POST-SLT KERATITIS

On postoperative day 5, the patient complained of pain in her left eye and was brought in for an examination that day. Her BSCVA had declined to 20/100 OS, and the slit-lamp examination showed anterior corneal stromal haze and rare cells in the anterior chamber. The IOP was 20 mm Hg, and there was no epithelial defect. The patient was treated with Lotemax (loteprednol

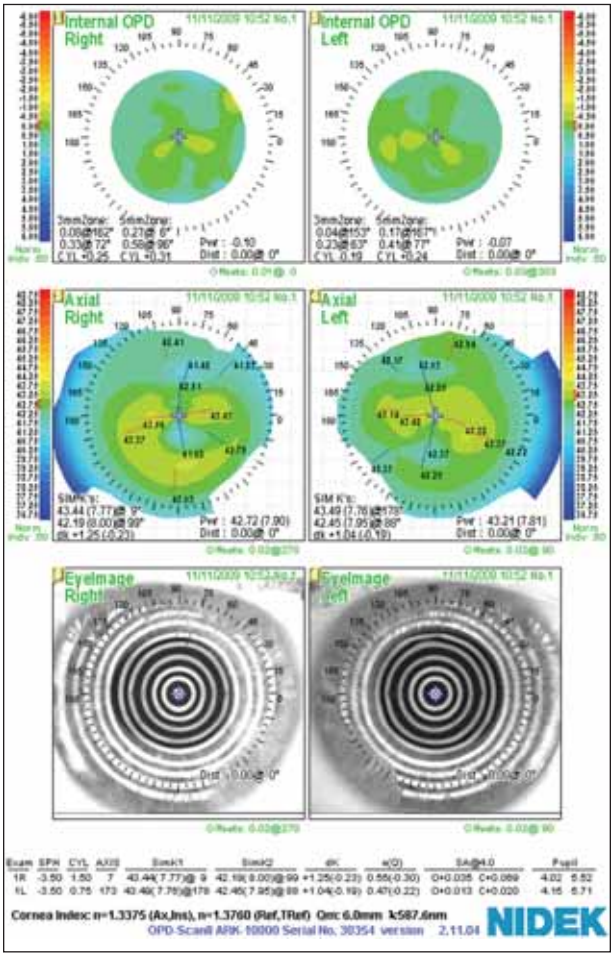

Figure 1. Preoperative topography showed similar corneal power in both eyes.

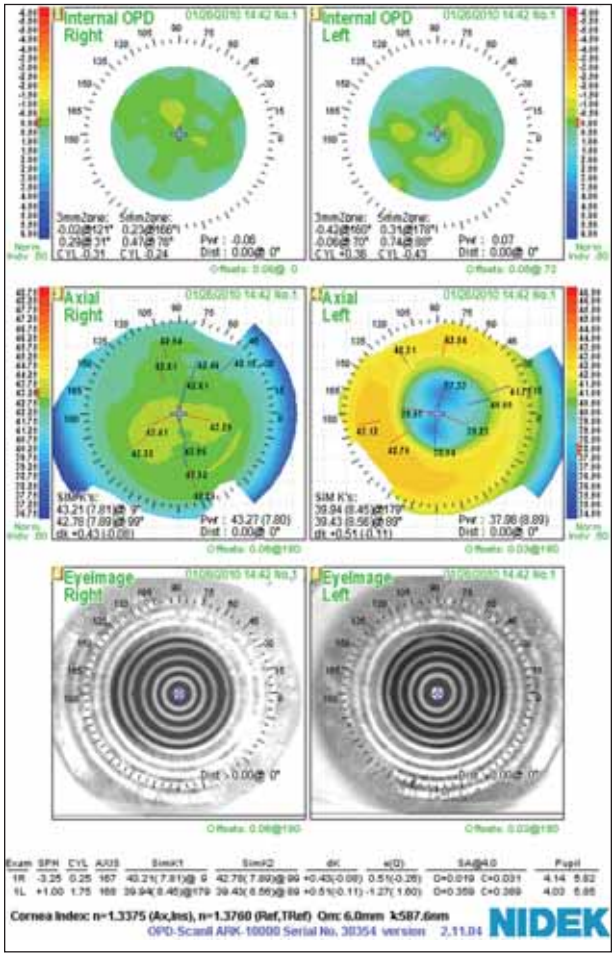

Figure 2. Corneal topography 5 days postoperatively revealed central corneal flattening.

etabonate ophthalmic suspension 0.5%; Bausch + Lomb) b.i.d. in her left eye. When she returned in 1 week, she reported a resolution of the pain but persistently blurry vision in her left eye. Her spectacle correction produced 20/200 acuity OS. Corneal topography revealed central flattening (Figure 2), and the patient's refraction showed a marked hyperopic shift to +1.00 +1.75 X 168 with 20/60 acuity. The examination was remarkable for persistent anterior corneal stromal haze but no anterior chamber cells or flare, indicating resolution of the patient's iritis.

RESOLUTION OF KERATITIS

Over the following few weeks, the keratitis in the patient's left eye continued to improve. There was a concomitant decrease in the central corneal flattening observed on topography and an attenuation of the hyperopic shift. Six weeks after the SLT procedure, her left eye had a manifest refraction of -0.75 +1.75 X 175 = 20/25. Four months after SLT, the refraction and acuity of her left eye remained stable, and pachymetry showed less central corneal thinning (562 µm OD and 498 µm OS). At the patient's 5-month follow-up visit, pachymetry revealed a central corneal thickness of 533 µm OS, close to the preoperative value of 544 µm, and the keratitis had resolved. However, 2.00 D of anisometropia persisted (Figure 3), which was not present preoperatively. The patient's IOP was well controlled, measuring 15 mm Hg OD and 13 mm Hg OS.

DISCUSSION

Laser trabeculoplasty (LTP) is a commonly performed and relatively safe and cost-effective therapy that reduces IOP in glaucoma patients. Argon laser trabeculoplasty (ALT) and SLT are the two most commonly performed LTP procedures. Complications of LTP include

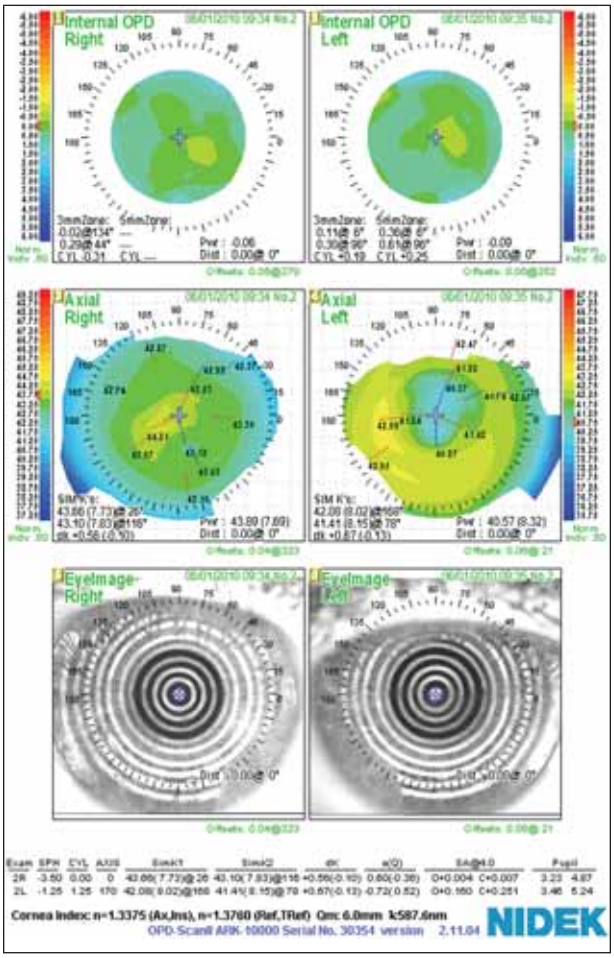

Figure 3. Corneal topography taken 5 months postoperatively. Two diopters of anisometropia persisted, which were not present preoperatively.

iritis and a brief elevation of the IOP in the immediate postoperative period.[1,2] During the past several years, SLT has become the most common type of LTP performed. Because of the number of procedures performed annually, any complication that may occur after SLT, even if relatively rare, is of interest to the surgeon and patient. The present case report represents one incident in more than 2,000 SLT procedures performed in our clinic during the past 6 years; SLT-induced keratitis and a concomitant refractive shift therefore likely represent a relatively rare event. As of this publication, we found only one other report of post-SLT keratitis. In this case report, the keratitis occurred in an eye that had undergone LASIK and manifested as diffuse lamellar keratitis, a common complication of LASIK.[3] The mechanism of these two types of keratitis seems dissimilar, since no prior corneal flap had been made in our case.

The fact that the post-SLT keratitis occurred in only one of two similarly treated eyes of the same patient presents additional challenges in determining an etiology. If there were a simple genetic predisposition, we would expect similar outcomes in both of the similarly treated eyes. Instead, we are left to speculate on what might have been different about the patient's left eye that led to the keratitis and hyperopic shift. Because a goniolens is used to apply the laser energy to the trabecular meshwork, the laser beam does not pass through the central cornea, the region that later became inflamed and flat. The keratitis and central corneal flattening must then be an indirect effect of the laser treatment, rather than a direct "burn" effect that might have occurred if laser energy had been focused through, or absorbed by, the central cornea.

We could speculate that the trabecular meshwork's absorption of energy might result in the production of a biochemical activating agent that could diffuse through the anterior chamber and enter the central corneal stroma via the endothelium. This unknown chemical trigger or messenger could activate collagenases or other enzymes that would then cause compression and flattening of the central cornea. A presumptive mechanism of SLT postulates that laser energy stimulates cytokine production from the trabecular meshwork. This cytokine then attracts macrophages that migrate into the trabecular meshwork and phagocytose debris from the extracellular matrix there. A cleaner trabecular meshwork would facilitate increased aqueous outflow and lower IOP. Might this same presumptive cytokine trigger the post-SLT keratitis observed in the present case?

CONCLUSION

Fortunately, our patient continues slowly to improve, and she has recovered a BSCVA of 20/25 OS. SLT remains a safe and effective therapy for glaucoma patients and retains a favorable side-effect profile compared with ALT. The incidence of iritis and elevated IOP is lower for SLT than ALT.[4] Moreover, the absence of peripheral anterior synechiae formation after SLT compares favorably with the 40% rate of peripheral anterior synechiae formation after ALT that was reported in the Glaucoma Laser Trial.[4]

The rare possibility of post-SLT keratitis should not demote SLT to a less favorable position in the spectrum of glaucoma therapies. However, informed consent prior to SLT will include mention of this possibility in our practice. The low incidence (one of 2,000 in the present report) of this post-SLT complication will likely hinder progress toward deciphering its etiology. As further cases are reported and compiled, however, predisposing factors may be determined. These could, in turn, be employed to

Are Post-SLT Keratitis and Central Toxic Keratopathy the Same Condition?

BY STEVEN J. DELL, MD

Central toxic keratopathy (CTK) is the name given by Somnez and Maloney to a rare condition seen after LASIK and PRK that results in central corneal opacification with tissue loss and a significant hyperopic shift.[1] Onset typically occurs on postoperative days 3 to 9, and the condition may last many months.[1] Others have described what appears to be the same condition but have given it different names.[2-4]

CTK is often preceded by diffuse lamellar keratitis (DLK),[5] and some surgeons argue that CTK may simply be a variant of severe, grade 4 DLK. There is no clear evidence regarding the exact etiology of CTK, but it has been suggested that the laser's activation of various substances in the LASIK interface such as meibomian gland secretions, povidone-iodine, or talc may be involved. Of importance, the condition has also been reported in PRK patients where there is no flap interface. To my knowledge, CTK has not been reported in cases without laser application to the cornea. The typical course is significant central corneal opacification extending deeply into the stroma, with a hyperopic shift due to tissue loss, followed by gradual resolution. Corneal thickness eventually increases toward the pre-CTK level due to epithelial hypertrophy. Some experts state that the condition is noninflammatory and does not respond to topical steroids.[1] There is controversy over how these eyes should be managed, as one might expect for a rare condition with an unknown etiology.

The case of post-SLT keratitis that Dr. Marquis presents bears many similarities with CTK—namely, laser application to the anterior segment with subsequent central corneal opacity, loss of corneal stromal tissue, and hyperopic shift, followed by gradual partial resolution. As Dr. Marquis points out, the laser application in SLT does not occur to the cornea itself. Any effect on the cornea from post-SLT keratitis seems to be an indirect one. Similarly, in the case of CTK, the stromal involvement is not confined to the level of the cornea receiving laser application; rather, it extends throughout much of the thickness of the corneal stroma. It makes sense that the loss of tissue from both of these entities could be due to something similar to the photoactivation of collagenase. In CTK, the degree of irregular astigmatism caused by the tissue loss is typically greater than that seen in this case. This difference may be due to the absence of a corneal flap, or perhaps it is simply a question of the vastly greater magnitude of laser energy delivered to the eye by the excimer laser.

Although both conditions seem to be quite rare, perhaps future study will help us determine whether they are related, the same, or distinct entities.

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improve patient-selection criteria for SLT and to prevent the complication. ❑

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