Objective: To evaluate the efficacy and safety of phototherapeutic keratectomy (PTK) in the treatment of symptomatic anterior basement membrane dystrophy following laser in situ keratomileusis (LASIK).

Methods: In a retrospective study, 10 eyes of 10 patients that developed symptomatic anterior basement membrane dystrophy following LASIK for myopia were treated with PTK using the VISX S2 (VISX Inc, Santa Clara, Calif) excimer laser. Primary outcome measurements including corneal clarity, resolution of symptoms, uncorrected visual acuity (UCVA), manifest refraction, best spectacle-corrected visual acuity (BSCVA), and complications were evaluated preoperatively, 1 day postoperatively, and at the last postoperative follow-up visit.

Results: At the last follow-up visit (mean [SD], 8.8 [5.5] months; range, 4-22 months), 100% of the eyes had clear corneas with no evidence of anterior basement membrane dystrophy, and all eyes were asymptomatic. Mean spherical equivalent changed from −0.75 (0.99) diopters (D) (range, −2.75 to +0.25 D) preoperatively to −0.51 (0.80) D (range, −1.63 to +1.00 D) at the last follow-up visit (P = .64). Uncorrected visual acuity improved from 20/20 or better in 1 eye (10%) and 20/40 or better in 5 eyes (50%) preoperatively to 20/20 or better in 5 eyes (50%) and 20/40 or better in 7 eyes (70%) postoperatively. No eyes lost lines of BSCVA, 2 eyes gained 1 line, 2 eyes gained 2 lines, and 1 eye gained 4 lines. There was a statistically significant improvement in mean logMAR BSCVA postoperatively, improving from 0.06 (0.16) (range, −0.1 to +0.3) to −0.08 (0.07) (range, −0.1 to +0.1) (P = .04). Postoperative complications included diffuse lamellar keratitis that resolved after treatment without sequelae (20%) and induced myopia exceeding −1.50 D (10%).

Conclusion: Phototherapeutic keratectomy for the treatment of symptomatic anterior basement membrane dystrophy following LASIK treatment is safe and effective.

Arch Ophthalmol. 2002;120:722-727

RESULTS

Ten eyes of 10 patients were treated for symptomatic ABMD after LASIK with PTK (Table). Four patients were female (4 eyes) and 6 were male (6 eyes). The mean (SD) age was 43.8 (7.7) years (age range, 32-57 years). No eyes had known ABMD preoperatively. All eyes underwent LASIK for myopia with a goal of emmetropia. One eye underwent primary LASIK, then LASIK retreatment for residual myopia. At the time of primary LASIK, 7 eyes had poorly adherent epithelium immediately postoperatively. On postoperative day 1, five eyes had loose epithelium, 2 eyes were diag-
PATIENTS AND METHODS

A retrospective review was conducted of 10 eyes of 10 patients treated with PTK using the VISX S2 Excimer Laser System (VISX Inc, Santa Clara, Calif) for symptomatic ABMD following myopic LASIK treatment. The 10 eyes had ABMD undetected at the preoperative LASIK examination because of a lack of signs or symptoms. All eyes were diagnosed as having symptomatic ABMD post-LASIK treatment, with symptoms including recurrent erosions, decreased vision, or visual distortion. The eyes underwent treatment for ABMD with PTK after failing conservative therapy including topical lubricants, hyperosmotics, or bandage contact lenses. The pre-LASIK examination results, LASIK surgical report, postoperative course, PTK treatment report, and post-PTK examination results were reviewed. All eyes had undergone myopic LASIK using the VISX S2 excimer laser and a 9.5-mm flap created with the Hansatome microkeratome (Bausch & Lomb Surgical Inc, Rochester, NY).

Phototherapeutic keratectomy was performed by a single surgeon (E.E.M.). After providing informed consent, patients were given a combination therapy of 0.5% topical proparacaine hydrochloride (Ophthotic; Alkerman Inc, Irvine, Calif), 0.1% diclofenac sodium (Voltaren; CIBA Vision Ophthalmics, Duluth, Ga), and 0.3% ciprofloxacin hydrochloride (Ciloxan; Alcon Laboratories Inc, Fort Worth, Tex) in the operative eye immediately before the surgical procedure. Patients were taken to the laser and reclined to a supine position. A sterile drape and a wire eyelid speculum were placed in the operative eye. The epithelium was removed by placing a corneal light protector soaked in 20% alcohol on the cornea for 30 seconds, followed by manual debridement with a photorefractive keratectomy spatula. The laser was then properly centered and focused. Six pulses at a pulse rate of 2 pulses per second were applied using a 6-mm optical zone. The beam was then reduced to a 3-mm optical zone, and the entire peripheral cornea was treated with 6 pulses using a combination of manual head movement and joystick control. No masking agent was used.

The eye was then irrigated with a balanced salt solution followed by instillation of 0.3% ciprofloxacin hydrochloride. Combination therapy of 0.1% diclofenac sodium and 1% prednisolone acetate (Pred Forte; Allergan Inc) were administered, and a bandage contact lens was placed. Patients were treated postoperatively with 0.3% ciprofloxacin hydrochloride, 4 times daily, and 1% prednisolone acetate, 4 times daily, until reepithelialization. In eyes developing diffuse lamellar keratitis (DLK), the 1% prednisolone acetate was increased to every 2 hours with close observation until the DLK resolved. After reepithelialization, the bandage contact lens was removed and 0.1% fluorometholone acetate (Flarex; Alcon Laboratories Inc) was given 2 times daily for 2 weeks.

Outcome measures included corneal clarity on slit-lamp biomicroscopy, resolution of symptoms, uncorrected visual acuity (UCVA), manifest refraction, best spectacle-corrected visual acuity (BSCVA), and complications. Postoperative examinations were performed at 1 day. The frequency of subsequent follow-up examinations varied according to the healing response, and was determined by the surgeon. The mean (SD) follow-up period was 8.8 (5.5) months (range, 4-22 months). Statistical analysis was performed with the paired t test using Microsoft Excel 2000 (Microsoft Corp, Seattle, Wash). P ≤ .05 was considered statistically significant. For statistical analysis of UCVA and BSCVA, Snellen visual acuity was converted to logMAR notation.

UCVA FINDINGS

Prior to PTK, UCVA was 20/20 or better in 1 eye and 20/40 or better in 5 eyes. At the last follow-up visit post-PTK, the UCVA for 5 eyes was 20/20 or better and for 7 eyes was 20/40 or better (Figure 3). The mean (SD) logMAR UCVA before PTK was 0.45 (0.50) (range, 0-1.7); the mean logMAR UCVA after PTK was 0.17 (0.27) (range, –0.1 to +0.6) (P = .16). No patients described persistent visual distortion or monocular diplopia following PTK.

BSCVA FINDINGS

No eyes lost lines of BSCVA at the last follow-up visit. The BSCVA remained the same in 3 eyes, gained 1 line in 2 eyes, gained 2 lines in 2 eyes, and gained 4 lines in 1 eye (Figure 4) (excludes 2 eyes that had variable refractions preoperatively and had no recorded BSCVA). The mean preoperative logMAR BSCVA was 0.06 (0.16) (range, –0.1 to 0.3). The mean postoperative BSCVA was –0.08 (0.07) (range, –0.1 to 0.1) (P = .04).
Mean (SD) spherical equivalent before PTK was −0.75 (0.99) D (range, −2.75 to +0.25 D) (excludes 2 eyes with variable preoperative refraction). Mean spherical equivalent after PTK was −0.51 (0.80) D (range, −1.63 to +1.00 D) (P = .64). Preoperatively, 3 eyes were within ±0.5 D of emmetropia, and 6 eyes were within ±1.0 D of emmetropia. Postoperatively, 4 eyes were within ±0.5 D of emmetropia, and 8 eyes were within ±1.0 D of emmetropia (includes all 10 eyes). The mean (SD) refractive shift was +0.23 (1.35) D (range, −1.25 to +2.25 D). Four eyes had hyperopic shifts (range, +0.13 to +2.25 D), and 4 eyes had myopic shifts (range, −1.00 to −0.25). There were no eyes with induced astigmatism or worsening of preoperative astigmatism.

COMPLICATIONS

There were no intraoperative complications. Postoperative complications included DLK (stages 1 and 2) in 2 eyes that resolved after treatment without sequelae and induced myopia more than −1.50 D in 1 eye. There were no eyes with induced hyperopia by more than +1.00 D. One eye had trace anterior haze that was visually insignificant at 3 months post-PTK and completely resolved by 12 months post-PTK. Uncorrected visual acuity in this eye was 20/25 and BSCVA was 20/20 at the last follow-up visit.

Anterior basement membrane dystrophy is the most common corneal dystrophy. Histopathologically, a multilaminar basement membrane, intraepithelial microcysts, and incomplete basement membrane complexes with no anchoring fibrils and few hemidesmosomes characterize map-dot-fingerprint dystrophy. These findings result in inadequate adherence of the corneal epithelium to the Bowman layer, predisposing these eyes to recurrent erosions. Minor trauma can lead to areas of poorly adherent epithelium as well as the appearance of microcysts, map lines, and fingerprint lines. Reports of patients with ABMD who have undergone LASIK describe epithelial sloughing during the microkeratome pass, leading to complications such as flap distortion, interface epithelial growth, flap keratolysis, and corneal scarring.1

While known ABMD is a contraindication to LASIK, ABMD may be undetected during the preoperative evaluation. These eyes with undetected with ABMD may undergo LASIK and may develop complications such as epithelial sloughing during the microkeratome pass, recurrent erosions postoperatively, and map-dot-fingerprint dystrophy on post-LASIK slitlamp biomicroscopic examination. With the increasing number of LASIK procedures being performed, and the prevalence of ABMD estimated at 2% to 42% of the population, refractive surgeons must carefully screen preoperatively for eyes with ABMD. When ABMD occurs in a patient after LASIK, decreased vision, visual distortion, and painful recurrent erosions compromise an otherwise potentially successful surgery. Epithelial defects after LASIK have also been associated with late-onset DLK, placing patients with recurrent erosions after LASIK at further risk for vision loss. Having safe and effective treatment options for symptomatic ABMD after LASIK is necessary to prevent loss of BSCVA and pain associated with recurrent erosions.

In eyes that do not respond to conservative management with topical hyperosmotics, topical lubricants,
bandage contact lenses, or patching, PTK is an option for the treatment of symptomatic ABMD. Phototherapeutic keratectomy is approved for the treatment of decreased BSCVA and pain that results from pathologic conditions involving the anterior one third of the cornea. Patients with corneal dystrophies, irregular corneal surfaces, or corneal scars and opacities can be treated for symptom improvement or improvement in visual acuity.8 Phototherapeutic keratectomy has been shown to be an effective treatment for recurrent erosions occurring as a result of ABMD, other corneal dystrophies, trauma, or idiopathic erosions.9-19 The procedure removes a few microns of tissue from the anterior cornea, allowing for the formation of a new basement membrane. Histological examination of monkey corneas after excimer laser ablation revealed increased amounts of type VI collagen, a major component of the anchoring fibrils, with reestablishment of a nearly continuous anchoring fibril zone.20 These changes are proposed to improve epithelial adhesion, thus preventing recurrent erosions.

Phototherapeutic keratectomy after LASIK has had limited reports previously. One study used transepithelial PTK 3 months post-LASIK to achieve epithelial smoothing in 3 eyes that had large epithelial defects (4-5
These epithelial defects resulted in irregular astigmatism or against-the-rule astigmatism, and patients complained of blurred and double vision. These eyes were not diagnosed as having ABMD. Good visual results were reported in 2 eyes, improving from decimal BSCVA of 0.75 and 0.85 pre-PTK to 0.95 and 1.0 at 1 month post-PTK. There were no problems with corneal healing in these eyes after PTK.

Our study shows that PTK can also be effective in treating eyes with symptomatic ABMD after LASIK. The results of our study suggest that PTK can improve vision, improve corneal clarity, and resolve recurrent erosions. A statistically significant improvement in BSCVA occurred in the study eyes. Eyes with monocular diplopia before PTK had no recurrence of visual distortions after PTK. All eyes had clear corneas without recurrence of dystrophic changes during the follow-up period. No eye had a recurrence of epithelial erosions. Previously reported complications such as visually significant corneal haze, induced hyperopia, and recurrent symptoms were not seen in our study. Refractive changes were observed in our series of eyes, with a mean hyperopic shift.

All types of refractive changes after PTK have been reported, with the greatest risk for hyperopia. Phototherapeutic keratectomy performed on the central cornea induces flattening, causing a hyperopic shift, whereas peripheral treatments may cause central flattening or steepening. Steepening may occur when more tissue is ablated peripherally than centrally, inducing a myopic shift. Furthermore, clearer optical zones post-PTK allowing more reliable refraction may also be a reason for refractive changes seen.

Our study showed a mean (SD) hyperopic shift of +0.27 (1.32) D, but the refractive change ranged from −1.00 to +2.25 D. One eye had a post-PTK refractive error of more than −1.50 D of myopia, and no eyes had more than +1.00 D of hyperopia. Previous studies of PTK have reported mean overall hyperopic shifts ranging from +0.34 to +3.42 D with hyperopic shifts occurring in 22.1% to 50% of eyes. Studies have also reported myopic shifts ranging from +0.5 to −4.76 D in 6.7% to 41.2% of eyes. Many studies report no statistically significant change in refraction, with stable refractions in 28.8% to 67.5% of eyes. In 1 study, astigmatic error was reported to increase in 15.6% of eyes with preoperative astigmatism, but no irregular astigmatism occurred and no astigmatism was induced in eyes without preoperative astigmatism. Others have reported a decrease of irregular astigmatism due to a reduction in the epithelial irregularity and the creation of a more uniform corneal contour, and no cases of induced irregular astigmatism.

Refractive shifts can be expected after PTK, an important consideration in eyes that have undergone LASIK with a goal of emmetropia. However, the minimal number of pulses necessary to treat ABMD may cause less refractive change than PTK treatment of other corneal disorders. Refractive shifts may also be minimized by using large treatment zones, blending the treatment zone, using a transition zone, limiting the amount of tissue ablated, and using a masking agent. Moving the eye with a rotary motion during ablation can also smooth the transition at the margins of the ablation and minimize the refractive change. Irregular astigmatism can be avoided by on-axis ablations, ignoring visually insignificant opacities.

Besides the refractive shifts seen in our study, few other complications were observed. One eye had trace anterior haze that was visually insignificant at 3 months postoperatively and completely resolved by 12 months postoperatively. Two eyes developed DLK after PTK. The episodes of DLK (stage 1 and 2) resolved without sequelae after treatment and may have been associated with the disruption of the epithelium during PTK. Previous reports of late-onset DLK associated with epithelial defects propose that disruption of the epithelium may attract a leukocytic cellular infiltration in the flap interface via a wound-healing cascade. Patients undergoing PTK after LASIK need to be closely monitored for the development of DLK postoperatively.

Other complications previously reported in eyes with ABMD after LASIK and in eyes treated with PTK were not seen in our study. In a report of epithelial sloughing secondary to ABMD during LASIK, complications included epithelial ingrowth in 62% of eyes, 67% of which progressed to flap keratolysis. No occurrence of epithelial ingrowth or flap melt was seen in our study. The major complications of PTK include delayed reepithelialization, stromal melting, infectious keratitis, reactivation of latent herpes virus, corneal scarring, visually significant subepithelial haze, and recurrence of symptoms. No eyes in our study experienced delayed reepithelialization, stromal melting, infections, or scarring. Recurrence rates after PTK for recurrent erosions have been reported from 0% to 42% at follow-up intervals ranging from 2 weeks to 70 months postoperatively. There were no recurrences of epithelial erosions at last follow-up in our study; all corneas were clear.

In the preoperative evaluation for refractive surgery, obtaining a clinical history of symptoms related to ABMD and close examination of the cornea is necessary. It is important to realize, however, that clinical signs in asymptomatic eyes may be absent, and despite meticulous examination, ABMD may be undetected. Eyes with ABMD may be initially seen with epithelial sloughing during the microkeratome pass, or may present postoperatively with recurrent erosions, decreased vision, or visual distortions. These eyes are at risk for interface epithelial ingrowth, flap melting, and loss of BSCVA. Such complications limit the results of refractive surgery. Our study suggests that in patients unresponsive to conservative treatment, PTK is a safe and effective method of treatment for symptomatic AMBD occurring after LASIK. Phototherapeutic keratectomy may improve vision and resolve symptoms without adverse outcomes such as visually significant corneal scarring or haze. Further study is needed to investigate the results of PTK after LASIK in a larger sample size for a longer postoperative course to further characterize refractive changes, stability of postoperative refraction, and recurrence rates of symptoms and corneal dystrophic changes.
Submitted for publication December 11, 2001; final revision received, not applicable; accepted February 28, 2002.

This study was presented in part at the Symposium on Cataract, Intraocular Lens, and Refractive Surgery, American Society of Cataract and Refractive Surgery, San Diego, Calif, May 1, 2001.

Corresponding author and reprints: Edward E. Manche, MD, Department of Ophthalmology, Stanford University School of Medicine, 900 Blake Wilbur Dr, Stanford, CA 94305 (e-mail: edward.manche@stanford.edu).

REFERENCES