Traumatic Late Flap Dehiscence and *Enterobacter* Keratitis Following LASIK

Leanne M. Cheung, MBBS(Hons), MPH;
Daya Papalkar, MBBS; Patrick Versace, FRANZCO

ABSTRACT

PURPOSE: To report a case of traumatic flap dehiscence and *Enterobacter* keratitis 34 months after LASIK.

METHODS: A 36-year-old man sustained a flap dehiscence following traumatic right eye gouging by a seagull claw. He presented the following day with uncorrected visual acuity (UCVA) in the affected eye of 3/200 and organic foreign body deposits underneath the flap. Systemic and topical antibiotics were administered and urgent surgical debridement and replacement of the LASIK flap was performed. An *Enterobacter* species was cultured from an intraoperative swab.

RESULTS: After a prolonged postoperative course, including administration of topical ofloxacin, tobramycin, chloramphenicol, and dexamethasone, UCVA returned to 20/20.

CONCLUSIONS: Good visual outcome after early debridement and appropriate antibiotics was achieved. Patients should be advised to seek prompt ophthalmic consultation for injury after LASIK. [*J Refract Surg.* 2006;22:402-404.]

Laser in situ keratomileusis (LASIK) is the most commonly performed refractive surgical procedure. Late traumatic dehiscence of the flap is a rare, but recognized complication, and has been reported up to 4 years after surgery.[1] This demonstrates that the flap-bed stromal interface is a potential zone of weakness for years following the procedure. Microbial keratitis is a vision-threatening, but rare, complication of LASIK.[2,3] This article presents a case of late traumatic flap dehiscence with associated *Enterobacter* keratitis, which was successfully managed with early debridement and specific antibiotics.

From the Department of Ophthalmology, Prince of Wales Hospital, Randwick and The University of New South Wales (Cheung, Papalkar, Versace); and The Sydney Eye and Laservision Centre, Bondi Junction, New South Wales (Versace), Sydney, Australia.

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Correspondence: Patrick Versace, FRANZCO, Level 2, 75 Grafton St, Bondi Junction, NSW 2022 Australia. Tel: 61 2 9386 3666; Fax: 61 2 9386 3699; E-mail: patrick.versace@bigpond.com

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CASE REPORT

A 36-year-old man had bilateral LASIK with a superior hinge in June 2000. The preoperative refraction was −3.25 in the right eye and −3.0 −0.5 × 84 in the left eye, with a best spectacle-corrected visual acuity (BSCVA) of 20/15 bilaterally. Bilateral 180 µm, 9.5-mm diameter LASIK flaps were created with a Hansatome microkeratome (Bausch & Lomb, Rochester, NY) and LASIK was performed with the Technolas 217 (Bausch & Lomb). Ablation depth was 61 µm in the right eye and 68 µm in the left eye. One month after LASIK, uncorrected visual acuity (UCVA) was 20/16 bilaterally.

At 34 months postoperatively, he was attacked by a seagull. The claw of the seagull gouged the right cornea despite the fact that the patient was wearing sunglasses. The patient did not rub the eye or disturb the flap. He presented the following day with decreased visual acuity, pain, and photophobia.

Uncorrected visual acuity was 3/200 in the right eye and 20/16 in the left eye. The cornea was edematous and had central deposits of sand and other unidentified organic foreign bodies under the lamellar flap. The lamellar flap had repositioned smoothly to its origin, and no flap wrinkling or epithelial defect was noted. Seidel test was negative. No anterior chamber reaction was observed. There was an associated subconjunctival hemorrhage.

The organic debris under the flap was considered to be a source of infection and hence microbiologic consultation was sought regarding broad-spectrum antibacterial treatment. Intravenous meropenem 1 g three times daily and hourly topical ofloxacin 0.3% and topical 0.5% chloramphenicol were administered as well as tetanus toxoid and immunoglobulin. Urgent surgery (>24 hours after injury) was performed under topical anesthesia. The lamellar flap was reflected, and the stromal bed and lamellar flap were explored. Multiple organic foreign bodies including sand granules and dirt were debrided with a Weck-Cel (Medtronic Ophthalmics, Jacksonville, Fla) from the dry stromal bed and undersurface of the lamellar flap. Bacterial and fungal swabs were sent for microscopy, culture, and sensitivity. The lamellar flap was irrigated and repositioned using the Weck-Cel. Postoperatively, topical dexamethasone 0.1% was prescribed twice daily. The meropenem caused an allergic skin reaction and was changed to oral ciprofloxacin 750 mg twice daily and metronidazole 400 mg three times daily. On the second postoperative day, UCVA in the right eye was count-fingers. On postoperative day 3, the organism was isolated to be an *Enterobacter* species, sensitive to ciprofloxacin and gentamicin. Oral antibiotics were ceased and topical therapy was unchanged. Uncorrect-

ed visual acuity was 20/80 in the right eye and pinhole (PH) 20/40. The allergic skin reaction did not resolve, thus topical ofloxacin was changed to tobramycin. One month postoperatively, topical antibiotics were ceased, preservative-free lubricants were started, and dexamethasone 0.1% four times daily was continued for persisting subepithelial haze with UCVA 20/60, PH 20/30. The patient was weaned from dexamethasone 0.1% over the following weeks. Five months postoperatively, the flap was well healed, with an inactive scar and UCVA 20/30, PH 20/16+.

A gradual healing response resulted in improved vision over 12 months postoperatively. No abscess formation or inflammation developed. The patient's final UCVA in the right eye was 20/20, and BSCVA was 20/16 with correction of $+0.75 -0.25 \times 90$.

DISCUSSION

This article presents a case of *Enterobactor* keratitis following late traumatic flap dehiscence. In a systematic review conducted by Chang et al[3] of infectious keratitis after LASIK, Gram-negative organisms were found to be a rare cause of infectious keratitis, with only one case (due to *Pseudomonas aeruginosa*) in the literature. In an ASCRS survey of 338,550 LASIK procedures, only 2 of 116 cases of infectious keratitis due to Gram-negative organisms were reported.[2]

Despite various mechanisms of trauma producing flap dehiscence,[4] there is only one previous report of subsequent infectious keratitis. Kim et al[5] reported a case of *Nocardia* keratitis following late traumatic flap dehiscence attributed to the patient rubbing his eye after being hit by a gun spring. Despite flap irrigation and repositioning and topical ofloxacin, microbial keratitis subsequently developed, requiring debridement >2 weeks after the injury. The infection resolved with topical antibiotics (trimethoprim-sulfamethoxazole).

Culture media for infectious keratitis after LASIK should include blood agar, chocolate agar, Sabouraud's agar, thioglycollate broth and Lowenstein-Jensen medium; scrapings should be stained with Gram, Gomori's methenamine silver, and Ziehl-Neelsen.[2] This approach will identify atypical mycobacteria, fungi, and *Nocardia, Staphylococcus,* and *Streptococcus* species.

A MEDLINE search revealed that our case is the fourth longest time period reported in the literature for flap dehiscence following LASIK.[1,6,7] The trauma involved in this case was a high impact, sharp object eye gouging. However, this case provides further evidence that even years after LASIK, there may still be a preexisting zone of weakness due to insufficient healing between the disrupted lamellae and also peripherally between the flap and peripheral cornea. The most common vision-threatening complications after traumatic late flap dehiscence are epithelial ingrowth and flap folds.[6]

Many mechanisms of late flap dehiscence have previously been reported,[4] ranging from unrecognized or mild trauma, such as from a fingernail while dressing,[8] to more severe trauma, such as from a thrown football[9] or deployed airbag.[7] Traumatic flap dehiscence may also occur during subsequent surgery on the eye, hence it is important for ophthalmologists to be aware of the potential for this injury to occur.[10] Interestingly, rabbit LASIK flaps are stable following simulated aircraft ejection at a vertical force of 9*g*, suggesting that directly applied tangential force with a high coefficient of friction is required to produce a traumatic flap dehiscence.[11] Previous studies have recommended the use of protective eyewear following LASIK for contact sports.[4,9,12] The mechanism of injury in our patient was severe trauma (eye gouging) and occurred despite wearing sunglasses. It also involved deposition of organic debris under the LASIK flap, resulting in infectious keratitis.

Following late traumatic flap dehiscence most patients have good visual outcomes with appropriate treatment,[4] often requiring exploration and repositioning of the lamellar flap and prophylactic antibiotics. Patients should be advised to seek prompt ophthalmic consultation for injury after LASIK. Despite a flap dehiscence and associated infectious keratitis with a virulent organism, our patient had a good visual outcome with UCVA of 20/20.

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Prophylactic Perioperative Antiviral Therapy for LASIK in Patients With Inactive Herpetic Keratitis

Mª Victoria de Rojas Silva, MD, PhD;
Elío Díez-Feijóo, MD, PhD; Jaime Javaloy, MD, PhD;
Manuel Sánchez-Salorio, MD, PhD

ABSTRACT

PURPOSE: To report the outcome of LASIK in patients with inactive herpetic keratitis in which perioperative antiviral prophylaxis was used to prevent the recurrence of ocular herpes.

METHODS: We report an uncontrolled series of five patients with inactive herpetic keratitis for at least 1 year before surgery in whom LASIK was successfully performed. All patients showed normal topography, pachymetry, and corneal sensitivity with no central corneal scarring. Perioperative prophylaxis was used in each case with oral valacyclovir and topical acyclovir ointment.

RESULTS: None of the eyes developed reactivation of herpetic keratitis during follow-up.

CONCLUSIONS: This study suggests that perioperative antiviral prophylaxis may protect the cornea from herpes simplex virus reactivation after LASIK. [*J Refract Surg*. 2006;22:404-406.]

From Fundación Instituto Galego de Oftalmoloxía. Universidad de Santiago de Compostela, Spain (de Rojas Silva, Díez-Feijóo, Sánchez-Salorio); and Vissum, Miguel Hernández University, Alicante, Spain (Javaloy).

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Correspondence: Mª Victoria de Rojas Silva, MD, PhD, Fundación Instituto Galego de Oftalmoloxía, Rúa Ramón Baltar, s/n (Hospital Provincial de Conxo), 15706 Santiago de Compostela, Spain. Tel: 34 981 534105; Fax: 34 981 531627; E-mail: vderojas@terra.es

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An area of concern for ophthalmologists and patients undergoing excimer laser photoablative procedures is whether latent herpes simplex virus (HSV) will reactivate and cause keratitis in eyes postoperatively.

Recurrences of HSV-1 keratitis have been reported after photorefractive keratectomy (PRK), phototherapeutic keratectomy, and LASIK.[1-4] In addition, both surface excimer laser ablation and LASIK can reactivate latent HSV type-1 in the rabbit model.[5,6]

Dhaliwal et al[7,8] demonstrated that antiviral prophylaxis with systemic valacyclovir significantly reduced HSV-1 ocular sheeding after LASIK or PRK in the New Zealand White rabbit latency model. Recently, Jarade and Tabbara[9] performed LASIK in three eyes of three patients with inactive unilateral herpetic keratitis and superficial stromal scar. Oral and topical acyclovir was prescribed perioperatively and no topical steroids were prescribed.

This article presents five cases of myopic LASIK in eyes with inactive herpetic keratitis for at least 1 year, in which perioperative antiviral prophylaxis was used to prevent the recurrence of ocular herpes.

PATIENTS AND METHODS

Five patients underwent bilateral myopic LASIK. Informed consent was obtained from all patients after they received a detailed description of the surgical procedures and known risks. All patients had a history of unilateral herpes simplex keratitis with inactive disease for a minimum of 1 year before the LASIK procedure. Four eyes had subepithelial scarring that did not interfere with the visual axis (peripheral in three cases and paracentral in one case).

Patient data and preoperative cycloplegic refraction and best spectacle-corrected visual acuity (BSCVA) are summarized in the Table. All patients showed normal topography, pachymetry map (Orbscan; Bausch & Lomb, Rochester, NY), and corneal sensitivity (Cochet-Bonnet esthesiometer; Luneau, Paris, France). Three patients were contact lens intolerant.

LASIK procedures were performed with a standardized technique using the Hansatome microkeratome (Bausch & Lomb) and the Technolas 217-C excimer laser (Bausch & Lomb).

Patients were treated with valacyclovir 500 mg twice daily for 1 week preoperatively and 2 weeks postoperatively. After surgery, all patients received 0.1% fluorometholone and 0.3% ofloxacin four times daily for 5 days and topical acyclovir ointment at bedtime for 2 weeks. Patients were examined 24 hours, 4 days, 1 week, 2 weeks, and 1, 3, 6 (three cases), and 18 (two cases) months postoperatively.

Postoperatively, uncorrected visual acuities were

TABLE

Summary of Data for Five Patients With Inactive Herpectic Keratitis Who Underwent LASIK

Patient No./ Sex/Age (y)	No. Episodes of Herpetic Keratitis Before LASIK	Preoperative			Postoperative UCVA
		Refraction	Pachymetry (μm)	BSCVA	
1/M/25	2	−6 −0.5 × 80°	580	20/25	20/25
2/F/38	3	−2.5 −0.25 × 180°	590	20/20	20/20
3/F/26	2	−2 −0.5 × 165°	560	20/20	20/20
4/F/35	1	−1.75 −0.5 × 10°	524	20/25	20/20
5/M/42	1	−4 sph	530	20/20	20/20

BSCVA = best spectacle-corrected visual acuity, UCVA = uncorrected visual acuity

equal or better than preoperative BSCVA (Table). None of the eyes developed reactivation of the herpetic keratitis during follow-up. One woman suffered herpetic eyelid lesions triggered by a fever episode without corneal involvement 4 months after surgery.

DISCUSSION

Little information exists in the literature regarding the proper approach when considering LASIK in a patient with inactive herpetic keratitis. Although the excimer laser has not been conclusively proven to act as a stimulus for the reactivation of latent HSV-1 in humans, clinical reports and experimental studies strongly suggest that LASIK could trigger reactivation of herpetic keratitis in patients with a history of recurrent ocular herpes.[1-6]

Evidence suggests that prophylaxis with oral acyclovir can prevent recurrent ocular herpetic episodes.[10] In humans, antiviral prophylaxis with oral acyclovir has been successfully used to reduce the frequency of recurrent genital and orolabial HSV infections. The Herpetic Eye Disease Study demonstrated further that oral acyclovir decreases the recurrence rate of herpetic stromal disease.[10] This evidence suggests that the risk of reactivation after LASIK could be decreased with perioperative antiviral prophylaxis. Systemic administration of valacyclovir significantly reduced HSV-1 ocular sheeding following LASIK in the New Zealand White rabbit latency model.[7] Previous reports on the topic preliminarily showed that LASIK was safe and effective in the treatment of refractive errors and ablation of midstromal scars in three patients with inactive herpetic keratitis who had prophylactic perioperative antiviral therapy.[9]

We present further evidence with a series of five successful cases of LASIK in inactive herpetic keratitis. This is the largest series reported to date. Different from the previous reports, the refractive error of our patients was not affected by the herpetic keratitis and they did not show any corneal opacities affecting the visual axis. The success of antiviral prophylaxis with acyclovir requires that adequate oral doses be taken to achieve the necessary therapeutic serum levels. In the New Zealand White rabbit latency model, the acyclovir serum levels that significantly reduced HSV-1 ocular sheeding were similar to those demonstrated in patients after a single 500- or 1000-mg dose.[7] Valacyclovir was used in our patients because it offers an advantage over acyclovir itself by producing greater serum concentrations of acyclovir with a smaller oral dose and provides the added benefit of more convenient dosing.[10] Although oral acyclovir has been shown to reach therapeutic levels in tears, virtually eliminating the need for a topical antiviral agent, we added acyclovir ointment at bedtime to follow previously reported guidelines for prophylaxis.[9] In the absence of oral antiviral administration, a single application of acyclovir ointment would not be a generally accepted prophylactic dosage. Under this treatment and even with the use of a low dose of topical corticosteroids for a short period of time, no recurrences of herpetic keratitis or corneal complications occurred in the present series.

One significant concern with performing LASIK on patients with history of herpes keratitis, apart from recurrences triggered by surgery itself, is the behavior of future recurrences in a cornea with a lamellar structure. Could recurrences after LASIK potentially have more serious visual consequences than in a virgin cornea? Could patients develop an associated diffuse lamellar keratitis? Could diagnosis and management be more troublesome?

Due to the limited number of cases and the lack of a control group, we can not determine whether prophylactic antiviral therapy is necessary before LASIK in all

patients with inactive herpetic keratitis, and the most effective treatment regimen (dose and time before and after surgery) remains to be defined.

We recommend caution in the selection of candidates with history of herpetic keratitis for LASIK surgery. In the preoperative evaluation for refractive surgery, it is important to obtain a clinical history consistent with herpetic keratitis to identify patients at risk of reactivation—subepithelial corneal scarring is not always present. Once a patient with a known history of herpetic keratitis is identified, and in the absence of any other contraindication for LASIK, we would base our selection of the case on the following criteria: inactive disease for a minimum of 1 year, normal corneal sensitivity, normal pachymetry map excluding focal thinning, regular topography, and normal BSCVA (no opacities involving the visual axis). The patient must be informed of the risk of reactivation. Should the surgeon decide to perform LASIK, the most reasonable effective clinical strategy would be to use perioperative antiviral prophylaxis.

In accordance with previous data, the study presented offers further evidence that perioperative antiviral prophylaxis may protect the cornea from reactivation of HSV after LASIK. Nevertheless, given the limited number of cases and the lack of a control group, further studies are necessary to establish the need for perioperative systemic antiviral prophylaxis in all patients and the optimum treatment regimen as well as the safety and efficacy of LASIK in this patient population.

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NeuroVision Treatment for Low Myopia Following LASIK Regression

Kooi Ling Lim, BOptom(Hons); Han Bor Fam, MD

ABSTRACT

PURPOSE: To evaluate a novel non-surgical method for improving vision in a refractive surgery patient.

METHODS: A 45-year-old man who had undergone LASIK 5 years previously presented with blurred distance vision. Unaided vision in the right eye was $20/32^{-2}$ and 20/20 in the left eye. He enrolled for NeuroVision treatment (NeuroVision Pte Ltd, Singapore), a computer-based interface in which a repetitive set of visual exercises is performed for 10 to 12 weeks.

RESULTS: After 35 sessions, unaided visual acuity in the right eye was $20/16^{-3}$ and $20/20^{-1}$ in the left eye, representing 2.8 lines of improvement in the right eye and 1.6 lines in the left eye.

CONCLUSIONS: NeuroVision, a noninvasive treatment based on the concept of perceptual learning, is a benefit in cases in which surgical enhancement is not recommended. [*J Refract Surg*. 2006;22:406-408.]

A recent study of common complaints of dissatisfied refractive surgery patients showed that 59% were unhappy because of blurred distance vision and 43.5% because of poor night vision.[1] A novel method for improving vision in these post-refractive surgery patients without further surgery is being tested using NeuroVision (NeuroVision Pte Ltd, Singapore). NeuroVision is a computer-based treatment interface where a repetitive set of visual exercises is performed

From iLaser Centre, Island Hospital, Penang, Malaysia (Lim); and The Eye Institute, Tan Tock Seng Hospital, Singapore (Fam).

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

Correspondence: Kooi Ling Lim, BOptom(Hons), iLaser Centre, 3rd Fl Island Hospital, 308 Macalister Rd, 10450 Penang, Malaysia. E-mail: kooiling@islandhospital.com

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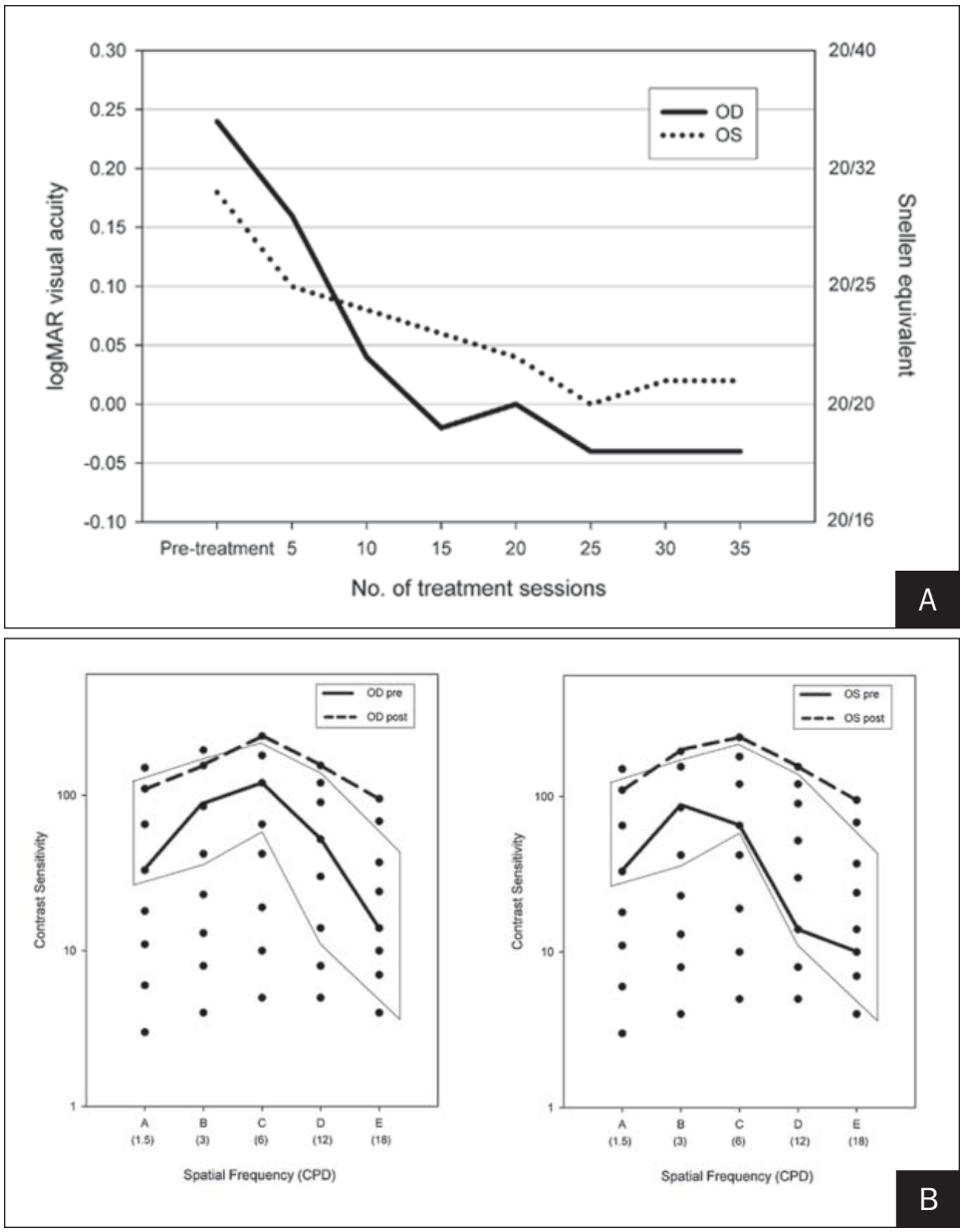

Figure. A) Change in unaided visual acuity and **B)** contrast sensitivity during the NeuroVision treatment.

for 10 to 12 weeks. It is founded on the concept of perceptual learning previously described for the treatment of amblyopia.[2,3]

CASE REPORT

A healthy 45-year old man underwent LASIK in November 2000 to correct $-9.50\ -1.00 \times 180$ (20/20) in the right eye and $-9.00\ -1.25 \times 175$ (20/20) in the left eye with targeted emmetropia in both eyes. In January 2005, the patient returned complaining of blurred distance vision. Unaided vision was $20/32^{-2}$ (0.24 logMAR) in the right eye and 20/40 (0.30 logMAR) in the left eye with a refraction of $-0.25\ -0.75 \times 155$ (20/20) in the right eye and $-1.00\ -0.50 \times 50$ (20/20) in the left eye. Although presbyopic, he did not have any problems with unaided near vision but had begun using glasses when driving at night 1 year after the initial LASIK. Cycloplegic refraction did not reveal any accommodative element in the residual myopia. The patient underwent a full ophthalmic assessment to ensure that his eyes were free from pathology. A LASIK enhancement was discussed and the patient was discouraged from correcting his unintended monovision, as it would lead to loss of near vision.

The patient enrolled for the NeuroVision treatment and upon completing treatment (after 35 sessions) in early June 2005, his unaided visual acuity was $20/16^{-3}$ (-0.04 logMAR) in the right eye and $20/20^{-1}$ (0.02 log-

MAR) in the left eye with no change in the manifest or cycloplegic refraction (Fig). This represents 2.8 lines of visual improvement in the right eye and 1.6 lines in the left eye. The repeatability (95% limits of agreement) of this set of logMAR charts in our clinic setting was previously tested and found to be 0.09 logMAR units.[4] This shows the improved visual acuity reported by the patient is actual improvement and not an artifact of noise in the visual acuity measurements. The contrast sensitivity function measured with the Sine Wave Contrast Test (Stereo Optical Co Ltd, Chicago, Ill) also showed improvement at every measured spatial frequency (see Fig). The patient reported marked improvement in photopic and mesopic vision and no longer relied on glasses.

DISCUSSION

As described in the literature on perceptual learning,[5] in neurons in the primary visual cortex, stimuli present within the receptive field can be facilitated or suppressed by other stimuli present outside the receptive field. Modulation of neuronal responses by stimuli falling outside the receptive field represents a neural mechanism for enhancing visual perception. By repetitively stimulating this neural mechanism, the enhanced visual perception may be transferred to other higher-level visual tasks such as visual acuity and contrast sensitivity.

The availability of a noninvasive treatment to improve unaided visual acuity and contrast sensitivity in patients following refractive surgery is a benefit to any refractive surgery practice especially in cases where enhancement is contraindicated.

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Photorefractive Keratectomy in Megalophthalmos Anterior

Éva Gallyas, MD; Vanda Hidasi, MD, PhD; István Ferincz; Lajos Kolozsvári, MD, PhD

ABSTRACT

PURPOSE: To evaluate the results of photorefractive keratectomy (PRK) for the correction of myopia and myopic astigmatism in megalophthalmos anterior.

METHODS: Four eyes of two brothers with megalophthalmos anterior were treated with PRK. In patient 1, best spectacle-corrected visual acuity (BSCVA) was 20/20 in both eyes with a refraction of $-4.50\ -4.50 \times 180°$ in the right eye and $-3.75\ -3.00 \times 175°$ in the left eye. In patient 2, BSCVA was 20/25 in both eyes with a refraction of $-4.25 \times 166°$ in the right eye and $+0.50\ -4.00 \times 175°$ in the left eye.

RESULTS: Topographic map, slit-lamp, ultrasound biomicroscopy, and postoperative course (no progression), supported with vectorial analysis, demonstrated megalophthalmos anterior. During 24-month follow-up, mild haze was observed and BSCVA was maintained.

CONCLUSIONS: Myopia and astigmatism are often observed in this type of nonprogressive corneal dysgenesis. Based on this fact and our results, we recommend PRK in cases of megalophthalmos anterior. [*J Refract Surg*. 2006;22:408-411.]

Megalophthalmos anterior is a distinctive type of corneal dysgenesis. This group of disorders can best be described as a disturbance in the growth and development of neural crest cells. A large myopic astigmatic refractive error often results from the abnormal optical architecture.[1] Corneal thickness and endothelial cell density are generally normal. The iris root may exhibit transillumination defects as

From KLIN Ophthalmologic Ltd (Gallyas, Hidasi); the Department of Ophthalmology, University of Szeged, Faculty of Medicine, Szeged (Gallyas, Kolozsvári); and Focus Medical (Ferincz), Budapest, Hungary.

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Correspondence: Éva Gallyas, MD, KLIN Ophthalmologic Ltd, Korányi fasor 10-11, 6701 - Szeged, Hungary. Tel: 3662 545092; Fax: 3662 544573; E-mail: gallyas@opht.szote.u-szeged.hu

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TABLE

Data on Two Brothers With Megalophthalmos Anterior Who Underwent PRK

	Patient 1		Patient 2	
	Right Eye	Left Eye	Right Eye	Left Eye
UCVA	20/80	20/80	20/40	20/80
Manifest refraction (D)	−4.50 −4.50 × 180°	−3.75 −3.00 × 175°	−4.25 × 166°	+0.50 −4.00 × 175°
Cycloplegic refraction (D)	−4.50 −4.50 × 180°	−3.75 −3.00 × 175°	−3.75 × 166°	+0.50 −4.00 × 175°
BSCVA	20/20	20/20	20/25	20/25
Keratometry (D)	47.25 180° / 51.25 90°	48.00 0° / 51.25 90°	47.50 170° / 51.00 80°	47.00 5° / 50.75 95°
Corneal horizontal diameter (mm)	12	11	11	11
Pachymetry (optical, μm)	460	460	480	470
Depth of anterior chamber (mm)	5.2	5.2	4.8	4.8
Axial length (mm)*	24.37	23.97	23.25	22.78

UCVA = uncorrected visual acuity, BSCVA = best spectacle-corrected visual acuity
**Ultrasonography with A-mode scan (Ultrascan® Dig. BTM 4000)*

a result of attenuation of the dilatator muscle. Because of the abnormal spatial relationship of structures in the anterior segment and stretching of zonules, miosis, iridodonesis, phacodonesis, and lens subluxation or dislocation may occur and it may later result in secondary lens-induced glaucoma.[2-4] The lens, furthermore, may become prematurely cataractous. Marfan's syndrome, Apert's syndrome, and mucolipidosis type II have been found in association with this disorder.[5] It is important to emphasize that corneal dysgeneses are nonprogressive abnormalities with no progressive thinning of the cornea and no decrease of endothelial cells.

PATIENTS AND METHODS

Two brothers aged 26 and 27 years, respectively, presented with myopia and astigmatism. Both patients had megalophthalmos anterior. Their father had good visual acuity and only required presbyopic correction. Their mother needed correction formerly, but the refraction was unknown. No history of ocular or any other disease was found among family members. Preoperative refractive and descriptive data are shown in the Table.

Corneal topography (Topographic Modeling System TMS-1; Computed Anatomy Inc, New York, NY) demonstrated a "bow-tie" appearance in both cases, typical of regular astigmatism (Fig). Examination by slit-lamp microscope revealed clear cornea with normal thickness[6] and open iridocorneal angles in both patients.

Pseudopolicoria, transillumination defects, full-thickness iris holes in the periphery, iridodonesis, moderate mydriasis, and phacodonesis were present in both patients. Lenses were clear in patient 1, mild peripheral opacities were seen in patient 2. Intraocular pressures (applanation tonometry) were <17 mmHg. Normal fundus was found by direct ophthalmoscopy. Ultrasound biomicroscopy (Ultrasound Biomicroscope System Model 840, 50 MHz; Zeiss Humphrey, San Leandro, Calif) revealed sparsely (probably reduced by 20% to 25%) elongated and more slender ciliary processes.[3,7] There were no associated systemic disorders. Phenotypically, the patients were different: patient 1 had brown hair and brown iris and patient 2 had blond hair and blue iris.

No contraindications for photorefractive keratectomy were present after complete ophthalmological examination. Laser in situ keratomileusis was not performed due to accentuated anterior corneal curvature and increased corneal asphericity.

Procedures were performed with topical anesthesia (tetracain hydrochloride) and with a 193-nm argon-fluoride Schwind Keratom 2 excimer laser (Schwind, Kleinostheim, Germany) (fluency 160 mJ/cm^2, repetition rate 13 Hz, ablation diameter 7.5×5.7 mm or 8.1×5.7 mm).

Postoperative treatment was neomycin drops five times daily during the first 5 days, and fluorometholon drops between weeks 3 and 12, continuously decreasing the dosage.

Fellow eyes were operated 3 months later. Examinations were conducted 1, 5, and 21 days and 3, 5, 6, 12, and 24 months postoperatively.

RESULTS

The postoperative period was characterized by satisfactory refractive results. Uncorrected visual acuity

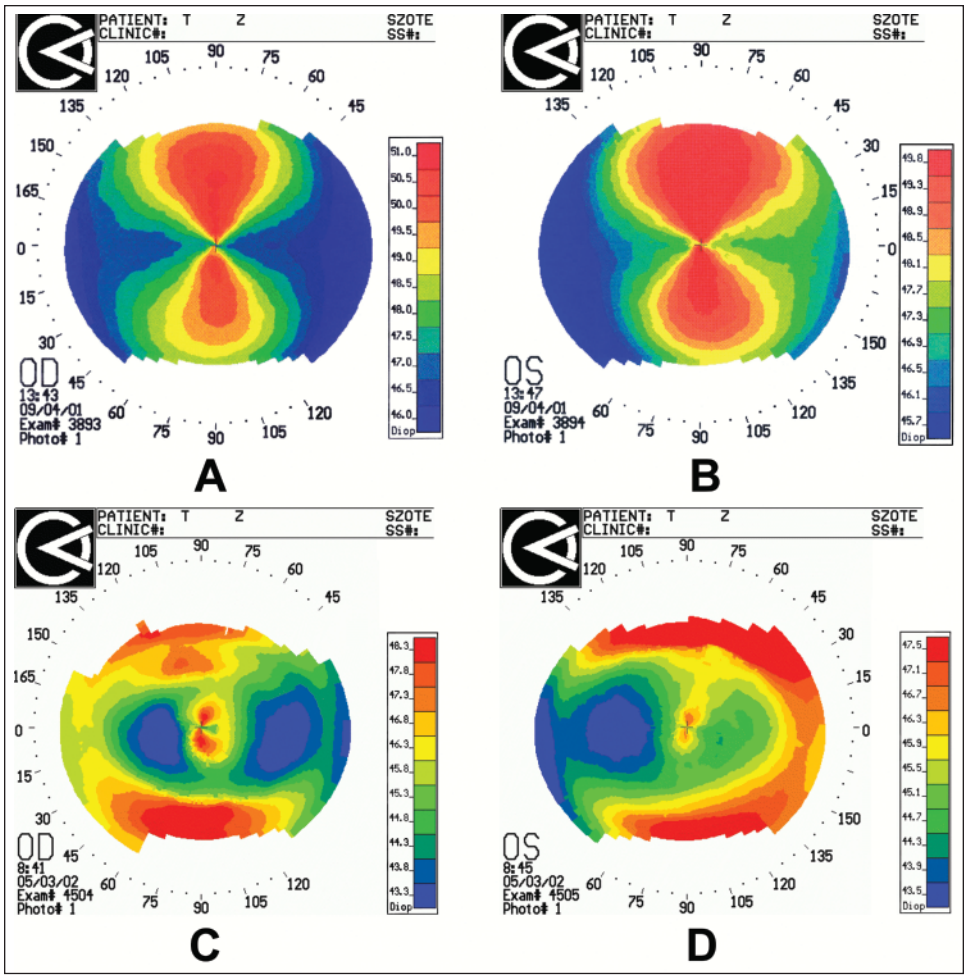

Figure. Topographic maps of patient 1—**A)** right eye and **B)** left eye before PRK and **C)** right eye and **D)** left eye after PRK.

(UCVA) and best spectacle-corrected visual acuity (BSCVA) improved in all eyes, and these refractive results were maintained in both eyes in patient 2 and in only the left eye in patient 1 during 24-month follow-up. In patient 1, a large corneal erosion occurred in the right eye from a high-pressure air-blow machine 4 months after PRK. Best spectacle-corrected visual acuity was 20/20 with a refraction of -1.25 $-1.00 \times 8°$ 24 months postoperatively. Mild haze was observed in all eyes (maximal grade according to Hanna 1.0) and disappeared at 3 months.

Vector analysis of surgically induced spherocylinder did not show any regression in patient 2, whereas slight regression—no greater than after "normal PRK"—was observed in the left eye of patient 1. The regression that occurred in the right eye of patient 1 was probably a consequence of the blunt trauma at 4 months postoperatively. The calculation was based on the principle that assumes the surgically induced spherocylinder ($S_{SURG}/C_{SURG} \times \beta$) is "crossed" with the preoperative refraction to produce the postoperative refraction:

$$S_I/C_I \times \alpha + S_{SURG}/C_{SURG} \times \beta = S_R/C_R \times \gamma,$$

and S_I and C_I are the initial or preoperative spherical and astigmatism vector at α axis (in "plus" cylinder notation), S_R and C_R are the resultant or postoperative spherical and astigmatism vector at γ axis (in "plus" cylinder notation), and C_{SURG} is the surgically induced astigmatism vector at β axis (in "minus" cylinder notation).

DISCUSSION

During the preoperative period we excluded the following diagnoses: keratoconus, keratoglobus, simple megalocornea, and megalophthalmos. Keratoconus is a progressive disease, with characteristic signs on the topographic map, ie, asymmetric bow-tie appearance, etc. In keratoglobus, the corneal thickness is about one-third of normal thickness (typically <200 µm), and no lens and iris abnormalities were found. In simple megalocornea, only the cornea is larger and no other ocular abnormalities are present. Furthermore, megalocornea is usually an inherited autosomal dominant trait. The patients' parents did not have any disorders similar to anterior megalophthalmos. The two patients, who were brothers, were phenotypically different. One had brown hair and

brown eyes, and the other had blond hair and blue eyes. In contrast, anterior megalophthalmos is generally inherited in X-linked recessive manner. This explains the parents' ophthalmic "health." Patients with megalophthalmos have much longer eye length and the whole eye is "bigger."[2]

Our findings, especially the topographic map (see Fig), slit-lamp and ultrasound biomicroscope features, and the postoperative course (no pathological progression), supported by vectorial analysis, confirmed anterior megalophthalmos.

Based on our results, we recommend PRK to treat myopia and astigmatism in megalophthalmos anterior. The clinical response after PRK in simple megalocornea and anterior megalophthalmos is similar to that in normal cases. Postoperative results support our diagnosis (no sign of progression) and our expectations.

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