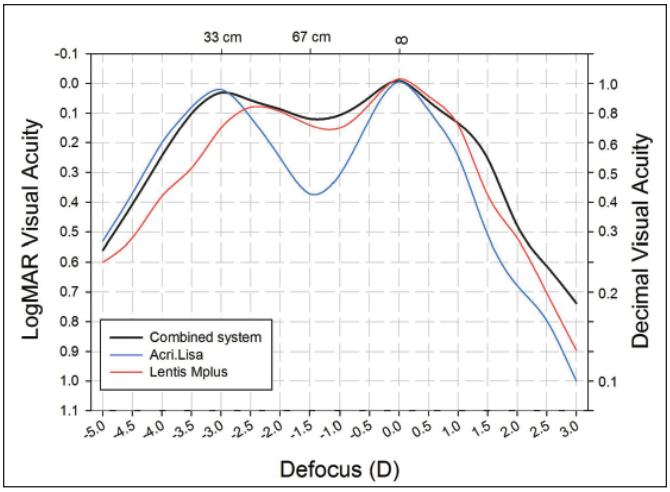

Figure. Mean binocular and monocular defocus curves with the combination of Lentis Mplus/Acri.Lisa multifocal intraocular lenses.

mance of the combined system. However, the presence of ocular dominance does not contraindicate the combination of MIOLs with different designs or additions as proved by previous studies.[6] It is noteworthy that the majority (82.5%) of patients in our study did not prefer the vision of one eye over the other when specifically asked about this item. Future studies should further address the issue of ocular dominance when combining refractive and diffractive MIOLs.

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Glaucomatous Damage from Pressure-induced Stromal Keratopathy After LASIK

Pressure-induced stromal keratopathy (PISK) after LASIK can cause significant visual loss if unrecognized and inappropriately treated.[1-5] The significant variability in reported clinical presentations may confuse practitioners, thus delaying or precluding appropriate diagnosis, if all clinical aspects are not considered. We herein describe a case of PISK in a young patient resulting in severe visual field loss in the absence of clinically definitive interface fluid and despite maximum measured intraocular pressure (IOP) <35 mmHg.

A 21-year-old woman, who had previously undergone LASIK and initial management at an outside facility, developed blurred vision and interface haze 2 weeks following uneventful LASIK. Preoperatively, corrected distance visual acuity (CDVA) was 20/20 bilaterally, refraction was −5.25 −0.50 × 150 in the right eye and −8.50 −1.25 × 028 in the left eye, pachymetry was 594 µm in the right eye and 631 µm in the left eye, and IOP was 23 mmHg in the right eye and 22 mmHg in the left eye. Optic nerves were normal with symmetrical 0.3 cup-to-disc ratios bilaterally.

The patient used prednisolone acetate 1% (Alcon Laboratories Inc, Ft Worth, Texas) four times daily in both eyes after LASIK. On postoperative day 1, uncorrected distance visual acuity (UDVA) was 20/20 bilaterally. Minimal debris was noted under the left LASIK flap, but the patient was asymptomatic. At 1 week, UDVA was 20/30 in the left eye, interface debris was still present, and the patient was still asymptomatic, but the decision was made to continue the prednisolone acetate four times daily in the left eye only. At 2 weeks, the patient reported "hazy" vision in the left eye without foreign body sensation or discomfort, and UDVA was 20/100. The patient was diagnosed with diffuse lamellar keratitis (DLK) in the left eye and prednisolone acetate was increased to every 2 hours. All subsequent follow-up and management occurred at a second outside facility.

At 3 weeks, there was no improvement, UDVA was 20/60, and IOP was 12 mmHg in the right eye and 31 mmHg in the left eye. A hazy granular interface was documented and the diagnosis of DLK was maintained. Over the next 5 months, the patient continued with topical steroids at varying doses, along with brimonidine 0.15% and timolol maleate 0.5% twice daily, for "atypical DLK." The patient's left cornea remained "hazy," and IOP fluctuated from 28 to 33 mmHg.

Six months after surgery, UDVA was 20/150 and IOP was 30 mmHg. The patient's left cornea was noted as "relatively clear" and topical steroids were discontinued. Four weeks later, UDVA was 20/100, IOP was

7 mmHg, and the patient reported seeing only parts of the letters.

Glaucoma evaluation documented UDVA 20/200 with eccentric viewing in the left eye, IOP was 10 mmHg in the right eye and 5 mmHg in the left eye, and a left relative afferent pupillary defect was noted with cup-to-disk ratio of 0.3 in the right eye and 0.8 in the left eye. Humphrey visual field testing revealed a dense superior paracentral scotoma with split fixation and a large inferior arcuate scotoma that has remained unchanged for 2 years.

This case demonstrates that severe glaucomatous damage can occur with PISK in the absence of obvious interface fluid and without excessively high measured IOP. Severe visual loss can occur over the course of a few months even in young healthy patients without ocular comorbidities if the appropriate diagnosis is not made, IOP lowering is not initiated, and steroids are not discontinued. This case appears to be unique in the combination of severe vision loss in a young patient without ocular comorbidities and without the presence of demonstrable interface fluid at any time point.

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