

Steroid-Induced Glaucoma After Myopic LASIK

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CASE PRESENTATION

A 35-year-old white male presented to the Glaucoma Service at the Scheie Eye Institute in Philadelphia with a complaint of decreased vision in his right eye after undergoing bilateral LASIK for myopia 8 months earlier. Before undergoing refractive surgery, he had been diagnosed with ocular hypertension due to IOPs measuring in the upper teens and low twenties. His IOPs were maintained in the mid-to-upper teens with a topical beta blocker.

During the patient's pre-LASIK examination, his IOPs spiked to more than 30 mm Hg after pupillary dilation in both eyes. A complete evaluation that included a dilated fundus examination and gonioscopy was unremarkable. Preoperatively, the patient's central corneal thicknesses measured 527 μ m OD and 542 μ m OS. He had normal Humphrey visual fields (Carl Zeiss Meditec, Inc., Dublin, CA).

Postoperatively, the patient had blurred vision in his right eye due to folds in the corneal flap and some fluid in the stromal interface. Over the next 4 months, his flap was revised three times, and he was instilling prednisolone acetate 1% q.i.d in his right eye to treat fluid in the flap's interface. His IOPs were reportedly within the normal range during this time.

After the flap in the patient's right eye healed and he discontinued steroids, his vision remained blurry, and he stated that he could see only out of the superotemporal field. A subsequent examination showed cupping of the optic disc with corresponding visual field loss in his right eye only. An MRI was negative for an intracranial compressive lesion, and the patient had no hereditary, nutritional, or toxic factors associated with optic neuropathy.

The patient's past medical history was significant for hypertension and hypercholesterolemia that were well controlled on atenolol and simvastatin. He was using a topical beta blocker in both eyes at bedtime to control his previously diagnosed ocular hypertension. He had quit

smoking tobacco 7 years ago, acknowledged the social use of alcohol, and reported he did not use illicit drugs.

On initial evaluation, the patient's uncorrected vision was count fingers with eccentric fixation OD and 20/20 OS. He had full and intact extraocular motility, and his pupils measured 5 mm OD and 4 mm OS with a relative afferent pupillary defect in his right eye. Confrontational testing revealed only a residual superotemporal island of vision in his right eye and full visual fields in his left eye. The patient's IOP measured 12 mm Hg OD and 13 mm Hg OS.

Slit-lamp examination showed bilateral LASIK flaps with nasal hinges. Except for pigment adhering to the corneal endothelium in his right eye, his corneas were clear. The pigment did not show a typical Krukenberg spindle pattern. The anterior chambers were deep and quiet bilaterally. The patient's irides were blue without any transillumination defects, and he had a 2+ posterior subcapsular cataract in his right eye. The crystalline lens in his left eye was clear.

Gonioscopy showed wide, open angles bilaterally with an angle recess of 40° to 45° and visualization of a wide ciliary body band throughout. The patient's irides were concave peripherally (Figure 1), and he had a 3+ homogeneously pigmented trabecular meshwork (Figure 2).

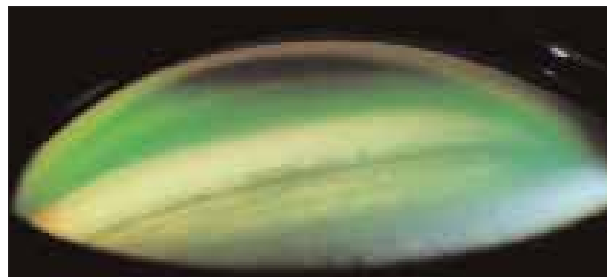


Figure 1. The patient's iris had a concave peripheral configuration on gonioscopy.

Funduscopy revealed cup-to-disc ratios of 0.9 OD and 0.4 OS. The optic nerve in the patient's left eye was pink (Figure 3). We deferred pupillary dilation due to his history of elevated IOP after this procedure.

We determined the patient had previously undiagnosed glaucoma in his right eye secondary to the use of topical steroids after multiple revisions of his LASIK flap. His refractive surgeon may have mistakenly interpreted his high post-operative IOPs as normal due to the presence of fluid in the stromal interface.

Although the patient did not have any transillumination defects in his right eye, he did have a very wide angle, a concave peripheral iris configuration, and pigment in the trabecular meshwork, all features of pigmentary dispersion syndrome.

HOW WOULD YOU PROCEED?

1. Would you recommend regular perimetric testing to monitor the patient for progressive glaucomatous visual field loss?
2. Initiate medical treatment to prevent the release of additional pigment from the iris?
3. Perform a laser peripheral iridotomy (LPI) to reduce the bowing of the peripheral iris?
4. Maintain the patient on ocular antihypertensive drugs?

SURGICAL COURSE

We decided to perform bilateral LPIs to flatten the patient's irides and reduce their contact with the lens zonules. Our goal was to decrease the dispersion of pigment and stabilize the IOP, as described by Karickhoff.¹

Forty-five minutes after the procedure, the patient's IOPs measured 8 mm Hg OD and 10 mm Hg OS. He was instructed to discontinue the topical beta blocker and to instill prednisolone acetate 1% in both eyes q.i.d. for 4 days. He was scheduled for follow-up in 1 week.

Four days after undergoing bilateral LPIs, the patient presented to the on-call resident with a complaint of decreased vision and halos in his left eye. He had never used the prednisolone in his right eye and discontinued it in his left eye 9 hours before his evaluation by the resident.

The patient's uncorrected vision was count fingers with eccentric fixation in his right eye and 20/30 (20/20 pin-hole) in his left eye. His IOPs measured 10 mm Hg OD and 32 mm Hg OS. Slit-lamp examination showed patent LPIs and trace inflammation in both anterior chambers.

We administered an alpha agonist, a topical carbonic anhydrase inhibitor, a beta blocker, and pilocarpine 1%, which lowered the IOP in the patient's left eye to

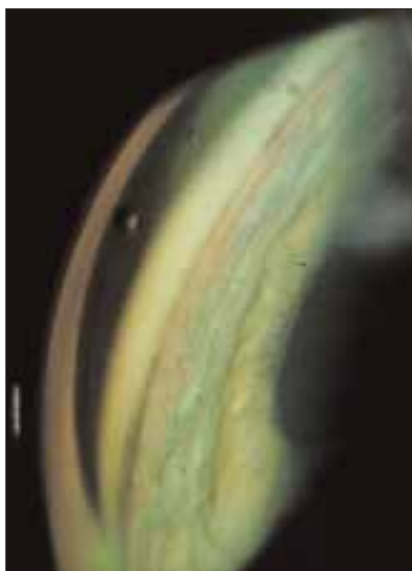


Figure 2. Gonioscopy showed a wide angle with heavy pigmentation of the trabecular meshwork consistent with pigmentary dispersion syndrome.

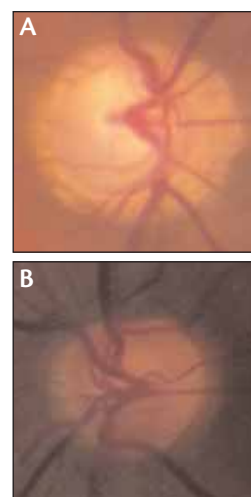


Figure 3. Optic disc photographs show almost total cupping in the patient's right eye (A) and a healthy disc in his left eye (B).

15 mm Hg. He was instructed to discontinue the steroid and continue using the IOP-lowering medications. When the patient returned for his previously scheduled follow-up 7 days after his bilateral LPIs, his IOPs measured 16 mm Hg OD and 7 mm Hg OS. The low IOP in his left eye at this time suggested that his pressure had risen postoperatively due to his use of the steroid. All of the ocular antihypertensive medications were discontinued at this time.

When the patient returned 2 weeks later, the uncorrected vision in his left eye had returned to 20/20, and his IOPs measured 13 mm Hg OD and 16 mm Hg OS. Gonioscopy showed that the bowing of the peripheral iris in both eyes had resolved. His IOPs after dilation was 17 mm Hg OD and 19 mm Hg OS. Pachymetry showed central corneal thicknesses of 430 μ m OD and 450 μ m OS. Because the patient's thin corneas resulted in the underestimation of his IOP using Goldmann applanation tonometry, we instructed him to resume the use of a topical beta blocker in both eyes.

OUTCOME

The documented spike in the patient's IOP after using prednisolone in his left eye for 3 days after LPI confirmed that he was a steroid responder. We also concluded that the elevated IOPs in the patient's eyes after LASIK were erroneously diagnosed as normal due to the presence of fluid in the stromal interface.

The presence of a posterior subcapsular cataract in only the patient's right eye further supported our hypothesis that

he was a steroid responder. Steroid usage is implicated in the development of posterior subcapsular cataracts. We postulated that cataractous development in the patient's right eye could have been accelerated by elevated IOPs secondary to his use of steroids. Gillies et al reported that elevated IOP after intravitreal triamcinolone might be implicated in the rapid progression of a posterior subcapsular cataract.²

We elected to maintain our patient on a topical beta blocker and strongly cautioned him to avoid periocular, inhaled, and systemic steroids, because these agents could elevate his IOP. On recent follow-up, the patient's IOPs measured 12 mm Hg, and his discs were stable in both eyes. 24-2 Humphrey Visual Field testing of his left eye was normal.

DISCUSSION

LASIK is currently the most popular and successful form of refractive surgery for correcting myopia. Compared with PRK, LASIK has a lower incidence of postoperative haze and a faster visual recovery.³ The formation of a corneal flap, however, introduces several possibilities for complications such as a free, wrinkled, or displaced flap; epithelial ingrowth; interface infectious keratitis; and non-infectious diffuse lamellar keratitis (DLK).^{4,5}

Several investigators have described steroid-induced glaucoma after LASIK complicated by the accumulation of fluid in the flap's interface. Lyle and Jin observed a patient who had fluid in his corneal interface 6 weeks after receiving steroids for postoperative DLK. The IOP in the eye with DLK measured 9 mm Hg with Goldmann applanation tonometry versus 30 mm Hg with the Tono-Pen (Reichert, Inc., Depew, NY).⁶

Fogla et al also described significant differences in IOPs measured with different tonometers in a patient who was treated aggressively with topical steroids for DLK after LASIK. In this case, the patient's IOP measured 3 mm Hg centrally with Goldmann applanation tonometry and 50 mm Hg peripherally with Schiotz tonometry.⁷ Hamilton et al⁸ reported a series of six cases wherein a false measurement of IOP masked elevated pressures. They postulated that a low endothelial cell count and an elevated IOP might exacerbate the accumulation of fluid in the corneal interface after LASIK.

Although most ophthalmologists are aware of the effects of LASIK on postoperative IOP, the recently described phenomenon of fluid in the flap's interface represents an important factor that can lead to grossly false IOP measurements. This change is distinct from the 2- to 5-mm Hg decrease in IOP that usually occurs after ablation and thinning of the cornea.

Brashford et al suggested that surgeons should monitor patients closely for steroid-induced glaucoma masked by fluid in the flap's interface after LASIK and should treat affected patients with the cessation of steroids and initiation of glaucoma medications.⁹ To help surgeons accurately identify the factors underlying elevated IOPs in glaucoma patients after LASIK, Brashford et al also suggested comparing a series of measurements obtained preoperatively with those measured after the eye has stabilized postoperatively.⁹ Surgeons can use the difference between the two sets of readings to determine a more accurate IOP after LASIK.

The patient described herein has an atypical case of open-angle glaucoma in his right eye and suspected open-angle glaucoma in his left eye. Although he did not demonstrate the full spectrum of features that accompany pigmentary dispersion syndrome, the presence of pigment in his angles on gonioscopy combined with bilaterally concave irides supported this diagnosis and his subsequent treatment with LPIs. □

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