# Steroid-induced Glaucoma after Laser In Situ Keratomileusis Associated with Interface Fluid

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***Purpose:*** To report the ocular manifestations and clinical course of eyes developing interface fluid after laser in situ keratomileusis (LASIK) surgery from a steroid-induced rise in intraocular pressure.

***Design:*** Retrospective, noncomparative interventional case series.

***Participants/Intervention:*** We examined six eyes of four patients who had diffuse lamellar keratitis develop after uneventful myopic LASIK surgery and were treated with topical corticosteroids.

***Principal Outcome Measure:*** Slit-lamp findings, intraocular pressure measurements, and visual field loss.

***Results:*** All eyes had a pocket of fluid develop in the lamellar interface between the flap and the stromal bed associated with a corticosteroid-induced rise in intraocular pressure. However, because of the interface fluid, intraocular pressure was normal or low by central corneal Goldmann applanation tonometry in all eyes. The elevated intraocular pressure was diagnosed by peripheral measurement in several cases after months of elevated pressure. All six eyes had visual field defects develop. Three eyes of two patients had severe glaucomatous optic neuropathy and decreased visual acuity develop as a result of undiagnosed steroid-induced elevated intraocular pressure.

***Conclusions:*** A steroid-induced rise in intraocular pressure after LASIK can cause transudation of aqueous fluid across the endothelium that collects in the flap interface. The interface fluid leads to inaccurately low central applanation tonometry measurements that obscure the diagnosis of steroid-induced glaucoma. Serious visual loss may result. *Ophthalmology 2002;109:659–665 © 2002 by the American Academy of Ophthalmology.*

The first report of interface fluid developing after laser in situ keratomileusis (LASIK) was reported by Lyle and Jin.[1] This case featured steroid-induced intraocular pressure elevation and falsely low intraocular pressure reading from applanation tonometry. The pocket of interface fluid, however, was believed to be related to the presence of epithelial ingrowth. Subsequently, we reported a case of interface fluid after LASIK surgery in a patient with diffuse lamellar keratitis without epithelial ingrowth who went on to have severe optic neuropathy develop from steroid-induced elevated glaucoma.[2] We hypothesized that the fluid resulted from transudation of aqueous across the stromal bed, resulting from steroid-induced elevated intraocular pressure. Fluid collected in the potential space between the flap and bed, causing erroneous applanation tonometry readings. Recently, Rehany et al[3] and Protellinha et al[4] reported similar cases. Since our first report (Case 1), we have collected three additional cases of fluid developing between the epithelial flap and stromal bed after LASIK in eyes treated with topical steroids for diffuse lamellar keratitis. In these cases, intraocular pressure measured by applanation tonometry suggested hypotony because of the collection of interface fluid. We believe this complication warrants early recognition and special attention to intraocular pressure measurement to avoid potentially devastating vision loss.

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## Case Reports

### Case 1

A 58-year-old man was referred to us in consultation after having undergone bilateral LASIK 6 months previously. The patient's preoperative cycloplegic refraction was $-10.00\ -0.25 \times 110$ in the right eye and $-8.25\ -1.50 \times 140$ in the left eye with best-corrected visual acuity of 20/20 in each eye. Preoperative intraocular pressure was 18 mmHg by applanation in both eyes. Funduscopic examination revealed pink optic nerves with cup-to-disc ratios of 0.4 in the right eye and 0.5 in the left eye. There was no prior history of glaucoma. Preoperative keratometry showed corneal curvatures of 44.50/44.50 in the right eye and 43.62/44.37 $\times$ 062 in the left eye. Corneal pachymetry was 575 $\mu$m in

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the right eye and 569 μm in the left eye. The procedure consisted of creating a 160-μm thick flap with a 6.0-mm ablation zone of 96 μm in central depth in the right eye and 76 μm in central depth in the left eye, leaving residual stromal beds of 319 μm in the right eye and 333 μm in the left eye.

The patient reported mild, intermittent discomfort in the left eye at the postoperative week 3 visit. The left eye was diagnosed with mild diffuse lamellar keratitis, characterized by diffuse granular haze in the interface, reducing uncorrected visual acuity to 20/200. The patient was started on fluorometholone acetate 0.1% (Flarex, Alcon Laboratories, Ft. Worth, TX) four times daily in the left eye. Vision decreased to the 20/400 to count fingers range by postoperative month 2. Keratometry readings were 41.50/37.75 × 115 in the left eye. The cornea revealed diffuse haze in the flap with microcystic edema at the edge. Intraocular pressure by applanation was 12 mmHg. The patient was started on prednisolone acetate 1% (Pred Forte, Allergan, Irvine, CA) four times daily but increased to every 2 hours 2 weeks later when there were no signs of improvement. At the 4-month postoperative visit, uncorrected visual acuity remained at 20/400 in the left eye with 2+ microcystic edema, and intraocular pressure was 7 mmHg by applanation. Topical steroids were increased to every hour in the left eye. One week later, pressure was found to be 25 mmHg by Kowa portable applanation tonometer (Kowa Co., Ltd., Tokyo, Japan) in the left eye. Steroids were reduced to four times daily, and betaxolol hydrochloride, 0.25% (Betoptic-S, Alcon Laboratories, Ft. Worth, TX) was started. At the 5-month postoperative visit, uncorrected visual acuity was 20/20 in the right eye and count fingers at 1 foot in the left eye with a 0.9 cup-to-disc ratio and a pressure of 10 mmHg by applanation tonometry. The patient was referred for consultation.

At our initial examination 6 months postoperatively, the patient complained of progressive decreased vision in his left eye since the surgery. On examination, the uncorrected visual acuity was 20/20 in the right eye and light perception in the left eye. There was a brisk afferent pupillary defect present in the left eye. Biomicroscopy revealed a nasally hinged flap with moderate epithelial edema and microcystic edema of the epithelium peripheral to the flap in the left eye. There was an optically clear fluid-filled space between the flap and the stromal bed in the left eye. Funduscopic examination showed a pale optic nerve with a deeply excavated, extremely thin circumferential rim and a cup-to-disc ratio of 0.99. Corneal topography demonstrated marked central steepening in the left eye: 44.25/45.73 × 002. Intraocular pressure measured using applanation tonometry was less than 5 mmHg. The pressure was 38 mmHg when measured peripherally using a Tono-Pen 2 (Automated Ophthalmics, Inc., Ellicott City, MD). The patient was diagnosed with steroid-induced elevation of intraocular pressure. Prednisolone was discontinued, oral acetazolamide, 250 mg four times daily, topical dorzolamide, 2% (Trusopt, Merck Laboratories, West Port, PA), and brimonidine, 0.25% (Alphagan, Allergan, Irvine, CA) were added to the betaxolol. Two weeks later, the interface fluid had cleared in the left eye, vision remained at light perception, and intraocular pressure was 12 mmHg by applanation and 13 mmHg by Tono-Pen peripherally. An automated visual field (Zeiss Humphrey Systems, Dublin, CA) obtained 7 months postoperatively revealed subtle superior and inferior arcuate changes in the right eye. The left eye could not undergo visual field testing because of poor visual acuity (Fig 1).

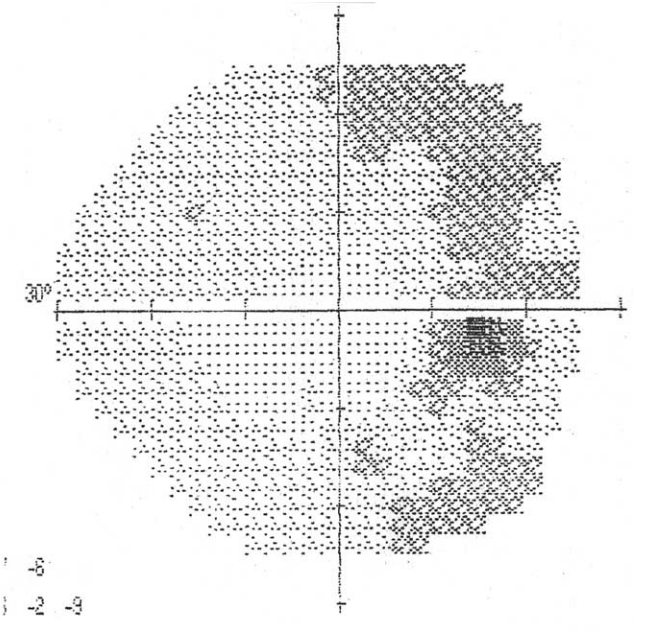


**Figure 1.** Case 1, right eye. Visual field 7 months after laser in situ keratomileusis surgery. There is a suggestion of early superior and inferior arcuate defects. The left eye could not undergo visual field testing because of light perception visual acuity.

## Case 2

A 31-year-old man was referred to us in consultation after having undergone bilateral LASIK 7 weeks previously. The patient's preoperative manifest refraction was −10.25 −0.75 × 007 in the right eye and −12.00 −2.00 × 140 in the left eye with best-corrected visual acuity of 20/30 in each eye. The patient noted mild irritation in the left eye 1 week postoperatively, was diagnosed with mild diffuse lamellar keratitis in both eyes, and was started on prednisolone acetate, 1%, every 2 hours in both eyes. An increase in the interface inflammation was noted in both eyes at the 3-week postoperative visit. Elevated intraocular pressure, measured to be 23 mmHg in both eyes, was treated with brimonidine tartrate, 0.2%, and timolol maleate, 0.5% (Timoptic, Merck Laboratories, West Port, PA) drops. The patient was placed on oral methylprednisolone (Medrol dose pack, Pharmacia & Upjohn, Peapack, NJ), and topical prednisolone was increased to every hour in both eyes to treat the increased interface inflammation. Best-corrected visual acuity had diminished to 20/200 in the left eye at the 1-month postoperative visit. The patient was started on oral prednisone, 80 mg once daily, with continued hourly topical prednisolone. The "inflammation" persisted, and a week later the patient underwent flap elevation and irrigation of the interface in the left eye without improvement in visual acuity. At 5 weeks postoperatively, best-corrected visual acuity was 20/60 in the right eye using a −6.50 rigid gas-permeable contact lens and 20/400 in the left eye. Oral steroids were tapered to 20 mg/day by the 6-week postoperative visit, topical steroids were continued hourly in both eyes, and the patient was referred for consultation.

On our initial examination 7 weeks postoperatively, the patient was unable to walk without assistance because of poor visual acuity and photophobia. His medications included oral prednisone, 10 mg once daily, topical prednisolone acetate four times daily in both eyes, and brimonidine and timolol twice daily in both eyes. Best-corrected visual acuity was 20/100 in the right eye and light perception in the left eye. Biomicroscopy revealed superiorly hinged flaps in both eyes with epithelial and microcystic edema peripheral to the flap in both eyes. An optically clear fluid-filled space between the flap and the stromal bed was present in both eyes (Fig 2). Corneal topography demonstrated marked central steepening in both eyes, left greater than right (Fig 3). Intraocular pressure measured with applanation tonometry was 0 mmHg in the

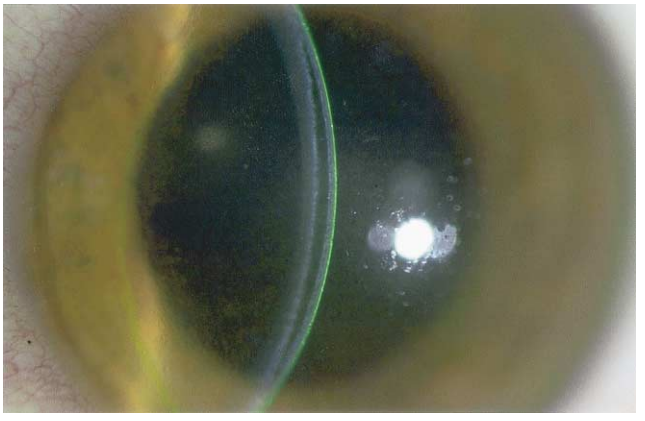

**Figure 2.** Case 2, left eye. An optically clear fluid-filled space is present between the flap and stromal bed. We hypothesize that it is caused by transudation of fluid across the endothelium from a steroid-induced elevation of intraocular pressure.

right eye and 1 mmHg in the left eye. The pressure was 30 mmHg in the right eye and 40 mmHg in the left eye when measured peripherally using a Tono-Pen 2.

The patient was instructed to stop the oral and topical steroid medications, continue brimonidine and timolol, and start oral acetazolamide, 250 mg four times daily. Best-corrected visual acuity had improved significantly at the 1-week follow-up visit to 20/40+2 in the right and 20/50-2 in the left. Trace interface fluid remained with marked improvement in corneal stromal and epithelial edema. Funduscopic examination revealed a cup-to-disc ratio of approximately 0.4 in both eyes with superior and inferior thinning in both eyes, right greater than left. Corneal topography demonstrated a significant decrease in central steepening in both eyes (Fig 4). Applanation tonometry had increased to 10 mmHg in both eyes. An automated visual field (Zeiss Humphrey Systems, Dublin, CA) obtained 4 months after the initial surgery demonstrated significant superior and inferior arcuate defects, right greater than left consistent with glaucomatous optic neuropathy in both eyes (Fig 5).

## Case 3

A 44-year-old woman underwent sequential LASIK for correction of mild myopic astigmatism using 180-$\mu$m flaps: $-1.00\ -1.25 \times 065$ in the left eye and $-1.00\ -2.25 \times 100$ in the right eye 1 week later. Preoperative pachymetry revealed a 543-$\mu$m thickness in the right eye and a 525-$\mu$m thickness in the left eye. Intraocular

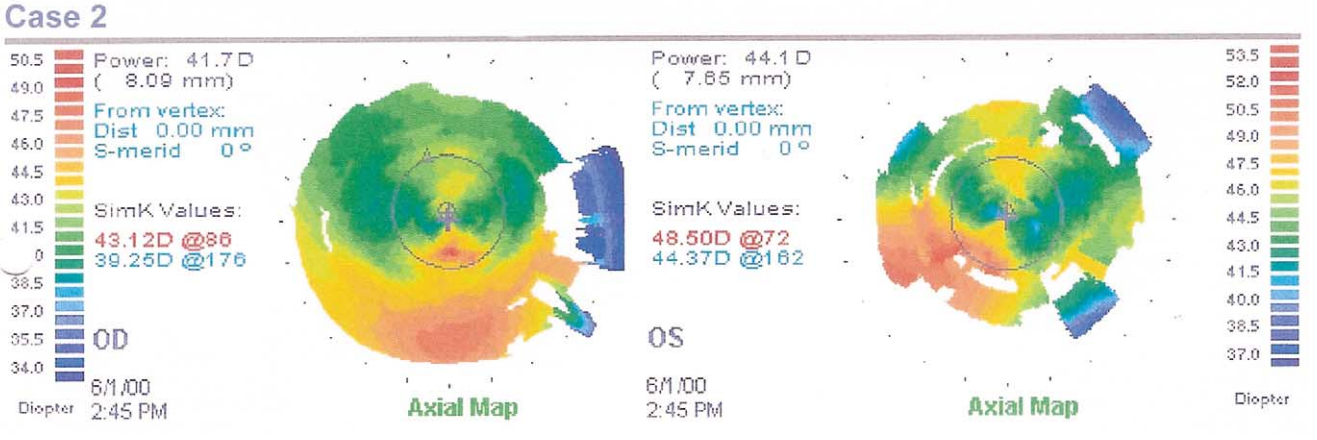


**Figure 3.** Case 2. Corneal topography 7 weeks after bilateral laser in situ keratomileusis. Both eyes demonstrate corneal steepening and marked astigmatism, left greater than right.

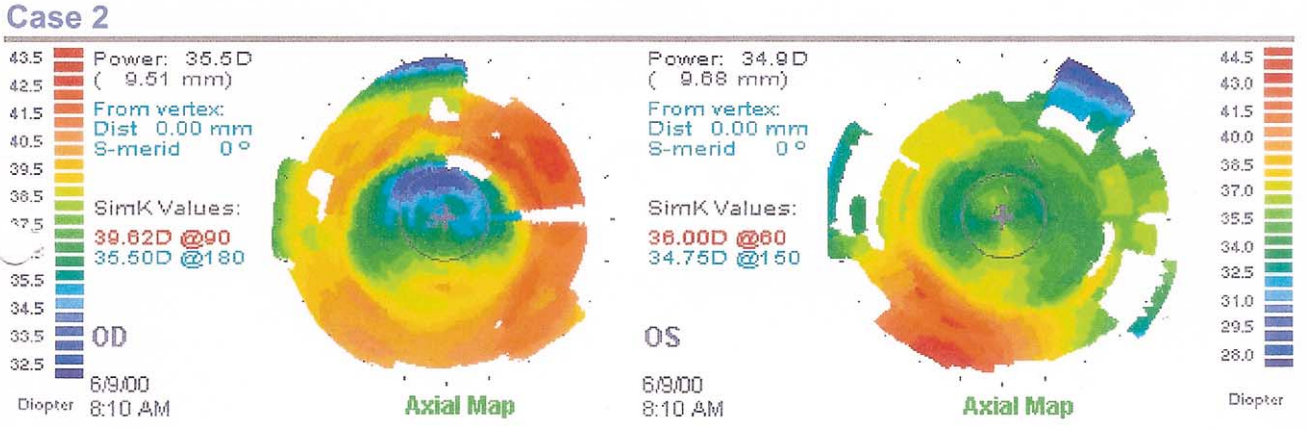


**Figure 4.** Case 2. Corneal topography 1 week after discontinuation of oral and topical steroids. Both eyes demonstrate a marked decrease in central corneal steepening consistent with resolution of interface fluid.

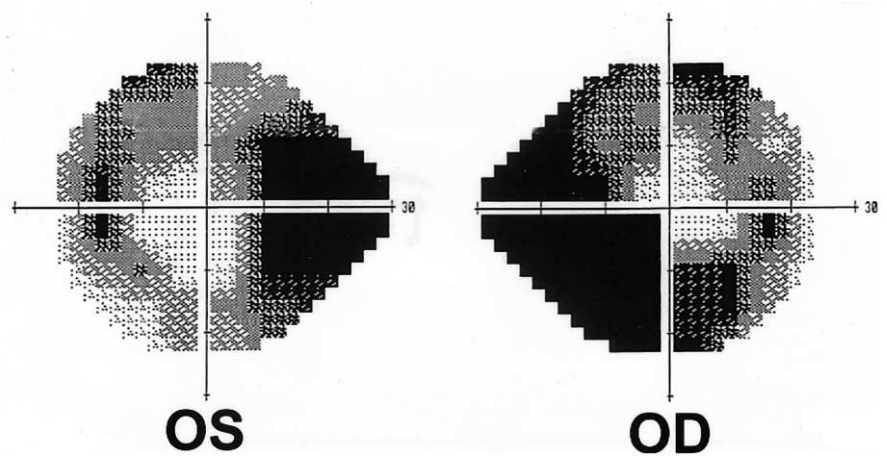


**Figure 5.** Case 2. Visual fields 4 months after laser in situ keratomileusis surgery. Both eyes demonstrate dense superior and inferior arcuate defects consistent with glaucomatous optic neuropathy.

pressure measured by Tono-Pen was 17 mmHg in the right eye and 19 mmHg in the left eye preoperatively. The postoperative course of the right eye was uneventful. Prednisolone acetate, 1%, and ciprofloxacin hydrochloride, 0.3% (Ciloxan, Alcon Laboratories, Ft. Worth, TX), eyedrops were started four times daily as part of the normal postoperative regimen. By 1 week postoperatively, trace interface debris inferotemporally and 1+ diffuse lamellar keratitis was present in the left eye. Uncorrected visual acuity was 20/20, and prednisolone was continued. At the postoperative week 3 visit, uncorrected visual acuity had decreased to 20/30-1, and the diffuse lamellar keratitis had worsened. Prednisolone drops were increased to every 2 hours around the clock. Five days later, the interface debris began to have the appearance of epithelium. There was no improvement on the higher steroid regimen, so the flap was lifted and irrigated at postoperative week 6. The day after the irrigation, uncorrected visual acuity was 20/25-1. Steroid drops were decreased to six times daily. One week later, it seemed the diffuse lamellar keratitis and epithelial ingrowth had recurred, and the patient was experiencing increasing pain. Steroid drops were increased to every 2 hours. Ten days later neither the clinical examination nor the symptoms had improved. The patient was started on oral prednisone 40 mg once daily with a rapid taper. Visual acuity had decreased to 20/400 3 days later. Slit-lamp biomicroscopy revealed an optically clear fluid-filled interface between the flap and stromal bed (Fig 6). Intraocular pressure measured peripheral to the flap using a Tono-Pen was 57 mmHg. Right eye pressure had risen to 27 mmHg. Oral acetazolamide, 250 mg four times daily, topical timolol maleate, 0.5%, and brimonidine tartrate, 0.25%, were given in the office, and the patient was sent home on dorzolamide hydrochloride 2%/timolol maleate 0.5% (Cosopt, Merck Laboratories, West Port, PA) twice daily. The patient had been using topical steroids every hour. These were tapered to every 2 hours, and the oral prednisone was decreased to 20 mg once daily. Best-corrected visual acuity had improved to 20/15 by the next day. The interface fluid had nearly resolved (Fig 7). Intraocular pressure using Tono-Pen was 12 mmHg centrally and 17 mmHg peripherally. Topical steroids were tapered, and oral prednisone was discontinued. Three months postoperatively, the patient's manifest refraction was $-1.25\ -0.50 \times 100$, yielding a visual acuity of 20/25. Visual field testing 6 months postoperatively revealed mild superior and inferior nasal field defects in the left eye (Fig 8).

## Case 4

A 19-year-old man with an ocular history of chronic open-angle glaucoma on dorzolamide hydrochloride 2%/timolol maleate

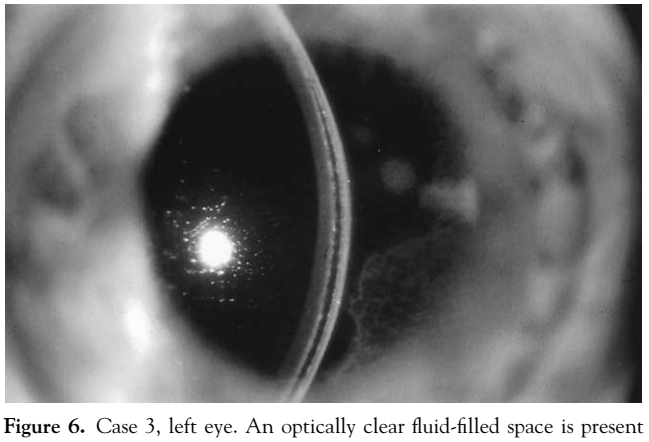

**Figure 6.** Case 3, left eye. An optically clear fluid-filled space is present between the flap and stromal bed.

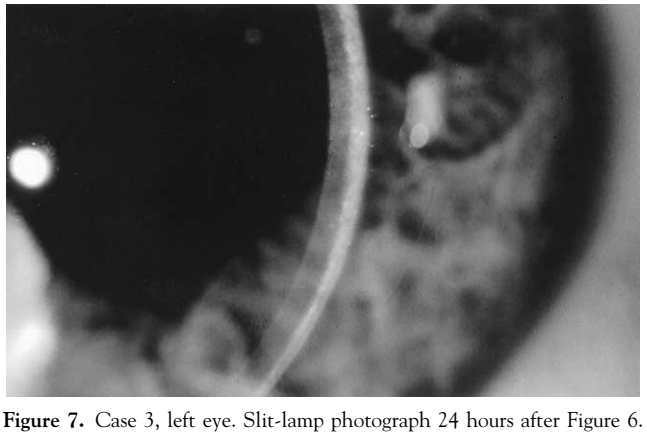

**Figure 7.** Case 3, left eye. Slit-lamp photograph 24 hours after Figure 6. Topical and oral steroids had been tapered, and intraocular pressure-lowering agents had been initiated. The interface fluid has nearly resolved.

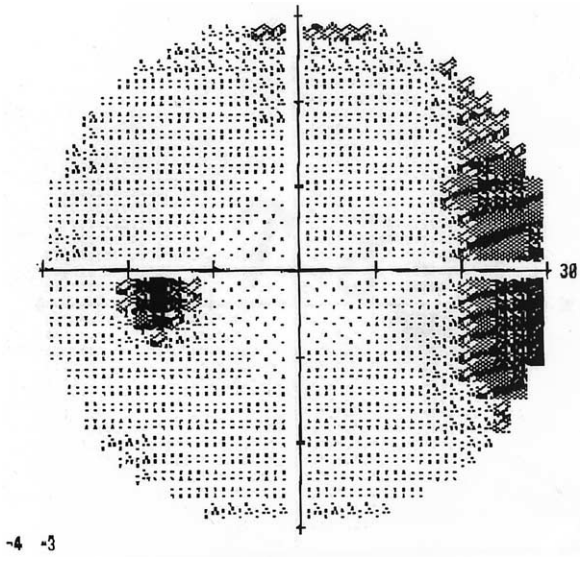


**Figure 8.** Case 3. Visual field in left eye 6 months after laser in situ keratomileusis surgery. There is a mild superior and inferior nasal step. The visual field in the right eye was within normal limits.

0.5%, brimonidine tartrate 0.25%, and latanoprost 0.005% (Xalatan, Pharmacia & Upjohn, Peapack, NJ) underwent bilateral LASIK using a flap thickness of 130 $\mu$m. Preoperative myopia was −12.25 sphere in the right eye and −13.00 in the left eye. Preoperative intraocular pressures using applanation tonometry were 22 mmHg in both eyes, and pachymetry revealed corneal thicknesses of 550 $\mu$m in the right eye and 547 $\mu$m in the left eye. Postoperative medications included topical ofloxacin, 0.3% (Ocuflox, Allergan, Irvine, CA), and topical fluorometholone acetate, 0.1%, four times daily in both eyes. At the postoperative week 1 visit, the patient noted eye pain and worsening vision. Intraocular pressure was 32 mmHg in the right eye and 38 mmHg in the left eye using applanation tonometry of the central cornea. A diffuse faint reticular haze in the flap interface of both eyes was noted, worse in the right eye. The patient's postoperative fluorometholone was increased to every 2 hours for presumptive diffuse lamellar keratitis. Oral acetazolamide, 500 mg twice daily, and pilocarpine hydrochloride, 2% four times daily, were added to the patient's existing glaucoma medications. The patient noted worsening vision at the follow-up visit. Slit-lamp biomicroscopy revealed an increase in flap edema and an optically clear fluid-filled space between the flap and stromal bed, more prominent in the right eye. Intraocular pressure was "unmeasurable" by applanation tonometry. Steroid eyedrops were immediately discontinued. At the 1-month postoperative visit, the patient's uncorrected visual acuity was count fingers in both eyes. Slit-lamp biomicroscopy revealed 3+ flap edema in the right eye, 2+ flap edema in the left eye, and persistence of interface fluid and extensive epithelial ingrowth in both eyes. Intraocular pressure continued to be elevated, measuring 42 mmHg in the right eye and 43 mmHg in the left eye by peripheral Tono-Pen. Both flaps were subsequently lifted and irrigated to remove epithelial ingrowth. The patient also underwent trabeculectomy in both eyes, resulting in reduced intraocular pressure to 14 mmHg in both eyes. Best spectacle-corrected visual acuity was 20/400 in both eyes at the most recent visit.

## Discussion

The eyes in our series share several key clinical features. All eyes had a steroid-induced rise in intraocular pressure associated with interface fluid, which was associated with an apparent glaucomatous optic neuropathy. In all eyes, elevated intraocular pressure was misdiagnosed, because the standard central measurement of intraocular pressure by Goldmann tonometry was erroneously very low.

Normally, highly elevated intraocular pressure causes transudation of fluid across the endothelium.[5] This fluid typically collects in the basal epithelium, presenting clinically as microcystic edema. In the post-LASIK cornea, however, the central stroma has a potential space created by the microkeratome incision. In contrast to the epithelium, which is tightly adherent, this potential space requires little force to open and is commonly opened iatrogenically to perform LASIK enhancement surgery.[6] We hypothesize that, as the fluid migrates forward through the stoma, it collects in this potential space, because it is easier to open than are the tight junctions between corneal epithelial cells. This fluid collects in the interface and expands it, causing the fluid that we observed clinically.[2] One might expect that microcystic edema would develop peripherally in these eyes, where a flap interface is not present. In some of the eyes, peripheral microcystic edema was seen, whereas in others it was not. It may be that in eyes without peripheral microcystic edema, the fluid that transudates peripherally migrates centrally along the corneal lamellae to collect in the fluid pocket rather than forcing its way between the tight junctions of the peripheral epithelium.

The role of endothelial function in developing interface fluid associated with elevated intraocular pressure is uncertain. We hypothesize that post-LASIK eyes with endothelial dysfunction may have interface fluid develop at a lower intraocular pressure than normal eyes. None of the eyes presented here had endothelial cell counts preoperatively, so their endothelial function is unknown. It is unnecessary, however, to postulate endothelial dysfunction to explain the interface fluid in these cases, because the pressures in each eye were high enough to cause microcystic edema in normal eyes.[5]

We hypothesize that the interface fluid caused the falsely low central Goldmann applanation tonometry measurement.[2] In effect, the applanation tonometer was measuring the pressure of the fluid in the interface, which was low, instead of the elevated pressure in the anterior chamber. Another possible explanation for the erroneous tonometry measurement can be considered. Goldmann applanation tonometry measurements decrease after myopic LASIK, typically by 2 to 3 mmHg. The reduction in measured intraocular pressure increases with increasing attempted correction and is approximately 3 mmHg for every 100 $\mu$m of ablation depth.[7,8] These changes likely relate to the decrease in corneal thickness after LASIK, because the Goldmann tonometer is calibrated to be most accurate on corneas 520-$\mu$m thick, a thickness at which the corneal rigidity just offsets the capillary attraction of the tear film to the tonometer head.[9] This small drop of 2 to 3 mmHg, however, is significantly smaller than the profound de-

creases in intraocular pressures seen in the cases we report here. Consequently, change in corneal thickness induced by LASIK is not a reasonable explanation for the erroneous Goldmann applanation tonometry measurements.

Although we believe the falsely low tonometry measurement occurs because the tonometer tip is measuring the pressure in the fluid-filled interface instead of the eye, we do not suggest that the measurements so obtained are an accurate measure of the pressure in the fluid-filled interface. The tonometer is measuring the pressure through a LASIK flap that is typically 160-$\mu$m thick. As discussed previously, the Goldmann applanation tonometer in theory will give a falsely low reading of the pressure of the fluid in the interface.

Epithelial ingrowth occurred in three of the eyes in this series as well as in one previous report.[1] Epithelial ingrowth is unlikely to be the cause of the interface fluid for several reasons. First, three of the eyes in this series did not have epithelial ingrowth, and four previously reported eyes also did not.[3,4] Second, in a large series of reported eyes with epithelial ingrowth, no cases of interface fluid were observed.[10] The common feature of all these eyes has been elevated intraocular pressure associated with corticosteroid treatment. Perhaps the presence of the interface fluid lifts the edge of the flap, exposing stroma onto which the epithelium can advance, leading to epithelial ingrowth.

All eyes presented here were placed on topical corticosteroids to treat interface inflammation. Diffuse lamellar keratitis occasionally follows LASIK[11] and is seen with diffuse mild inflammation confined to the lamellar interface. The incidence of diffuse lamellar keratitis may be as high as 1 in 25 in some centers.[11] The treatment of choice for diffuse lamellar keratitis remains topical corticosteroids. In all eyes corticosteroids were continued to treat long-term "inflammation." We believe that the apparent long-term inflammation was actually corneal edema related to the interface fluid rather than collections of intracorneal leukocytes. It is important to not only recognize diffuse lamellar keratitis and initiate appropriate treatment but to identify eyes with interface fluid. This syndrome, if undiagnosed, can lead to significant visual loss, as evidenced by the visual field defects sustained by all six eyes in this case series.

Elevation of intraocular pressure resulting from oral or topical corticosteroid use is relatively common. One study looking specifically at topical corticosteroid use found 4% to 6% of the population to be "high responders," manifesting elevations of greater than 15 mmHg and intraocular pressures greater than 31 mmHg.[12] Nearly one third of the population were found to be "moderate responders," with pressure rises between 6 and 15 mmHg and intraocular pressures between 20 and 31 mmHg. Among those at higher risk are individuals with preexisting glaucoma and those who are more likely to be seen in a refractive surgery practice, patients with high myopia.[13]

Case 4 involves a subject with preexisting glaucoma, making it difficult to identify what incremental damage can be attributed to the steroid-induced intraocular pressure elevation, especially because the pressure remained significantly elevated after steroids were discontinued. The subject did, however, have interface fluid develop, causing erroneous applanation tonometry measurements that were quickly recognized. This case highlights the importance of rapidly identifying interface fluid in patients with preexisting glaucoma.

In summary, early recognition of this glaucoma-induced interface fluid is essential to avoid complications associated with prolonged elevated intraocular pressure. Clinically, there are several clues that are useful to diagnose this entity.

1. Postoperative "hypotony." Intraocular pressure measurements by Goldmann applanation tonometry of less than 9 mmHg should be viewed with skepticism. Measurement of the intraocular pressure on the cornea peripheral to the flap using a Tono-Pen or applanation tonometer can be performed.
2. Clinical course of diffuse lamellar keratitis. First described in 1998,[11] diffuse lamellar keratitis typically is seen on postoperative day 1 to 3 and resolves by day 7 to 10. A patient who has been on topical steroids for treatment of diffuse lamellar keratitis and has not had resolution in 10 days should be carefully evaluated for interface fluid. In addition, a diagnosis of diffuse lamellar keratitis made for the first time more than 10 days after the surgery should prompt an examination for interface fluid.
3. Changes in corneal topography. Postoperative corneal topography after myopic LASIK surgery should demonstrate significant flattening of the treatment zone. The corneal topography of the eyes in this series demonstrated marked steepening of the treatment zone when the patients were initially seen with elevated intraocular pressure. The steepening reverted to the normal flattening after the pressure returned to normal. The steepening observed is most likely due to interface fluid pushing the central cornea anteriorly relative to the peripheral cornea.
4. Presence of interface fluid. Biomicroscopy readily reveals optically clear fluid in the flap interface. In addition, one should carefully examine the peripheral cornea for the presence of microcystic edema, an additional clue indicating the presence of elevated intraocular pressure.

It is unclear how much time is required for the flap interface to heal. Patients who require topical steroids to treat ailments unrelated to the primary LASIK procedure, such as uveitis, episcleritis, or keratitis, may be at risk for interface fluid developing for months after the initial surgery. In each of these situations, if interface fluid develops, intraocular pressure should be measured on the peripheral cornea, topical steroids should be tapered or discontinued if possible, and medications to lower intraocular pressure should be initiated. With proper attention and treatment, profound glaucomatous optic neuropathy can be avoided.

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