Abstract

Here, we report the case of a 28-year-old male patient who underwent uncomplicated laser-assisted in-situ keratomileusis (LASIK) in both eyes 10 days earlier and came with complaints of blurring of vision in the left eye with vision of 20/20. The patient gave a history of not tapering steroids as advised. His intraocular pressure (IOP) measured with rebound tonometer was 15 mmHg in the right eye and 25 mmHg in the left eye. On slit-lamp examination, the LASIK flap was intact in both the eyes, but the cornea appeared hazy in the left eye. Optical coherence tomography shows a central thickness of 524 µ in the left eye. The patient was diagnosed to have pressure-induced stromal keratopathy. The patient was switched to low-potency steroids, and anti-glaucoma drugs were given. On follow-up on slit-lamp examination, there was a significant decrease in haze in the left eye clear vision of 20/20 in both the eyes. It is important to recognize and treat PISK appropriately, as untreated elevated IOP can lead to optic nerve damage even over a brief period of several weeks.

Keywords: Laser-assisted in situ keratomileusis, optical coherence tomography, pressure-induced stromal keratopathy, rebound tonometry

INTRODUCTION

Interface fluid syndrome is a rare complication following laser-assisted in-situ keratomileusis (LASIK) surgery characterized by fluid collection in the flap interface. First described by Belin et al. in 2002. It is commonly reported 1 week to months after LASIK surgery. It can be misdiagnosed as diffuse lamellar keratitis (DLK), central toxic keratopathy, infectious keratitis, and epithelial ingrowth, among others. It has been reported secondary to raised intraocular pressure (IOP), endothelial decompensation, and uveitis. It is reported that a significant increase in IOP occurs in 5%–30% of steroid users. The elevated IOP can cause aqueous humor to enter the center of the cornea and collect at the interface under the corneal flap.

This case is unique as it can be misdiagnosed and untreated elevated IOP can lead to visual field and optic nerve damage even over a brief period of several weeks.

CASE REPORT

A 28-year-old male patient presented to the ophthalmology clinic with complaints of blurring of vision in the left eye for 1 day. His ocular history was significant for uncomplicated LASIK (flap thickness – 100 µ; ablation zone – 8 mm; total ablation depth – 80 µ; total number of pulses – 358 in both eyes; intralase ilasik machine was used to create the flap; and ablation was done with VISX Star S4 1R) 10 days earlier for a refractive error of 4.50 Sp1 in both the eyes and uncorrected visual acuity of 20/200 and best-corrected visual acuity of 20/20 in both the eyes, with a corneal thickness of 550 µ in both the eyes. He also gave a history of being on prednisolone eye drops four times a day following surgery which he did not taper as advised.

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did not have any history of uveitis, herpes keratitis, ocular hypertension, or glaucoma.

His uncorrected visual acuity was 20/20 in both the eyes. His IOP was measured with rebound tonometry (I Care) and was 15 mmHg in the right eye and 25 mmHg in the center and 35 mmHg at the junction of flap and the peripheral cornea in the left eye. His external examination was normal, with equally round and reactive pupils, full motility, and no orbital swelling or erythema. On slit-lamp examination, the LASIK flap was intact with superior hinge in both the eyes, with smooth and uniform corneal epithelium but appeared hazy at the interface in the left eye [Figure 1]. Optical coherence tomography showed a central thickness of 524 µ [Figures 2 and 3]. The anterior chamber was quiet; posterior segment examination was normal in both the eyes. The patient was switched from prednisolone eye drops to loteprednol 0.5% (L-Pred) eye drops twice a day for 2 days and then once a day for 2 days for both the eyes. For the left eye, dorzolamide 2%–timolol 0.5% (Dorzox T) eye drops twice a day and tablet acetazolamide 250 mg once a day for 3 days were prescribed. The patient was asked to come back for follow-up after 4 days. In the next visit, the patient reported a significant improvement of vision in the left eye with uncorrected visual acuity of 20/20. His IOP was 10 mmHg in the right eye and 12 mmHg in the left eye in the center and 10 mmHg at the junction of flap and the peripheral cornea in the left eye. On slit-lamp examination, there was a significant decrease in haze in the left eye, Lasik flap was intact, and the anterior chamber was quiet. Optical coherence tomography showed a thickness of 475 µ. The topical therapy of loteprednol eye drops once a day and dorzolamide and timolol eye drops were advised to continue for 2 days then to be stopped.

**Discussion**

The LASIK flap is known to be incomplete healing, retaining only 2%–28% of the original tensile strength for more than a decade after surgery. Therefore, the potential space between the flap and stroma is constant.\(^5\)

In pressure-induced stromal keratopathy, the amount of fluid might vary from a mild haziness to a stock of fluid that creates a gap between the anterior flap and the posterior stroma. In this case, the cornea showed diffuse edema and a pocket of fluid between the flap and residual stromal bed, as well as the history of topical steroids, which is characteristic for this entity.\(^3\)

Dawson *et al.* demonstrated histopathological evidence that edema can occur preferentially at the central and paracentral interface as they contain proteoglycans that can absorb water.\(^6\) Hamilton *et al.* reported that elevated IOP causes transudation of fluid across the endothelium which collects in the basal epithelium as microcystic edema.\(^3\)

In the post-LASIK cornea, the central stroma has a potential space; they hypothesized that the fluid collects in this potential space because it is easier to open the tight junctions between the corneal epithelial cells.\(^2\)

In the setting of PISK, the accurate measurement of IOP is challenging because of the accumulation of interface fluid which makes IOP measurement less reliable.\(^7\) Measuring the IOP in the corneal periphery, outside the area of interface fluid, will reveal a more accurate assessment of the true elevated IOP and aid in the diagnosis.\(^3\)

It is important to recognize and treat PISK appropriately because untreated elevated IOP can lead to visual field and optic nerve damage even over a brief period of several weeks.\(^7\)

It is important to differentiate from DLK as PISK worsens with steroids, the typical treatment for DLK.\(^7\)
We emphasize the importance of measuring IOP peripheral to the flap using hand-held applanation tonometer and rebound tonometer.[2] Alternative methods such as tonopen can be used.[8]

A long-term follow-up after routine LASIK surgery is probably the easiest and most necessary step to prevent PISK.[3]

**Declaration of patient consent**
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**
There are no conflicts of interest.

**REFERENCES**

1. Jia Z, Zhao S, Wei R, Huang Y, Zhang C, Yang R. Interface fluid syndrome: A potential lifelong complication after LASIK. A case report. Am J Ophthalmol Case Rep 2018;11:23-5.
2. Belin MW, Hannush SB, Yau CW, Schultze RL. Elevated intraocular pressure-induced interlamellar stromal keratitis. Ophthalmology 2002;109:1929-33.
3. Hamilton DR, Manche EE, Rich LF, Maloney RK. Steroid included glaucoma after laser in situ keratomileusis associated with interface fluid. Ophthalmology 2007;114;1848-59.
4. Gab-Alla AA. Incidence of interface fluid syndrome after laser in situ keratomileusis in Egyptian patients. Clin Ophthalmol 2017;11:613-8.
5. Bamashmus MA, Saleh MF. Post-LASIK interface fluid syndrome caused by steroid drops. Saudi J Ophthalmol 2013;27:125-8.
6. Dawson DG, Kramer TR, Grossniklaus HE, Waring GO 3rd, Edelhauser HF. Histologic, ultrastructural, and immunofluorescent evaluation of human laser-assisted in situ keratomileusis corneal wounds. Arch Ophthalmol 2005;123:741-56.
7. Lee V, Sulewski ME, Zaidi A, Nichols CW, Bunya VY. Elevated intraocular pressure-induced interlamellar stromal keratitis occurring 9 years after laser in situ keratomileusis. Cornea 2012;31:87-9.
8. Senthil S, Rathi V, Garudadri C. Misleading Goldmann applanation tonometry in a post-LASIK eye with interface fluid syndrome. Indian J Ophthalmol 2010;58:333-5.