Rare disease

Post-LASIK edema-induced keratopathy (PLEK), a new name based on pathophysiology of the condition

Virgilio Galvis, Alejandro Tello, Mario Leandro Revelo, Paul Valarezo

Department of Ophthalmology, Centro Oftalmológico Virgilio Galvis, Floridablanca, Santander, Colombia

Corresponding to Dr Alejandro Tello, alejandrotello@gmail.com

Summary

A 33-year-old man who underwent uneventful laser in situ keratomileusis (LASIK) developed pressure-induced stromal edema resulting in an interface haze in both eyes and a pocket of fluid under the flap of the right eye 10 days after surgery, while receiving topical fluorometholone. Intraocular pressure by applanation tonometry was 16 mm Hg in his right eye (erroneous result due to the fluid in the interface) and 34 mm Hg in his left eye. After discontinuation of steroids and addition of ocular hypotensive medication, interface fluid collection disappeared in his right eye. Visual acuity improved and haze diminished in both eyes. This case illustrates that in the same patient a post-LASIK edema induced syndrome may be present with or without fluid in the interface, suggesting that both clinical pictures could be manifestations of a broad spectrum of the same condition. We suggest a new name for this non-inflammatory disorder: post-LASIK edema-induced keratopathy (PLEK).

BACKGROUND

Although this condition is rare, we consider very important that the ophthalmologists keep it in mind, since it can be confused with other more common inflammatory pathology and missing a proper diagnosis can lead to very serious visual consequences for the patient. The bottom line is that this possibility must not be forgotten and must be ruled out in any patient with a clinical picture of diffuse interlamellar keratitis (DLK), especially if the condition is of late-onset or has an atypical response to treatment.

CASE PRESENTATION

A 33-year-old man underwent sequential laser in situ keratomileusis (LASIK) for correction of myopic astigmatism using the Hansatome microkeratome and the Amaris Scwind excimer laser system. There were no intraoperative complications. The preoperative refraction was -5.00 ($-1.00 \times 150^\circ$) in the right eye and -4.75 ($-1.25 \times 15^\circ$) in the left eye, with a best-corrected visual acuity (BCVA) of 20/25 in both eyes. Preoperative intraocular pressure was 16 mm Hg in both eyes. After LASIK the patient was started on moxifloxacin, fluorometholone and tear substitutes.

One day after surgery, uncorrected visual acuity (UCVA) was for distance 20/20 in the right eye and 20/200 in the left eye, and for near vision 20/20 in both eyes. As expected, the right eye presented a manifest refraction of plano and the left eye -1.75 D sphere (monovision).

Ten days after surgery the patient consulted us referring blurred vision in both eyes. Uncorrected visual acuity was 20/60 in the right eye and 20/100 in the left eye, BCVA was 20/40 with refraction of plano $-2.00 \times 70^\circ$ and 20/20 with refraction of -2.00 ($-0.50 \times 80^\circ$), respectively.

Slit lamp biomicroscopy revealed mild interface infiltrate and mild haze limited to the anterior stroma in both

eyes; additionally, in the right eye, a fluid-filled space was identified in the lower third of the interface between the flap and the stromal bed (figure 1). Intraocular pressure was 16 mm Hg in the right eye and 34 mm Hg in the left eye measured by applanation tonometry. However digital estimation of the IOP was much higher in the right eye.

INVESTIGATIONS

Visante anterior segment OCT images confirmed the presence of an optically empty space in the interface between the LASIK flap and the stromal bed in the lower third of the right eye cornea, which diminished until disappearing in the superior third of the cornea (figure 1).

DIFFERENTIAL DIAGNOSIS

Differential diagnostic considerations in these cases include basically three conditions: DLK, post-LASIK interface fluid syndrome (IFS) and pressure-induced

Figure 1 Slit-lamp examination of the right eye (A) shows an optically empty space indicating interface fluid accumulation (white arrow) which corresponds with the Visante anterior segment OCT system image (B). There are features of early stage of interface fluid syndrome (similar to pressure-induced interlamellar keratitis) in the superior cornea (3A and 3B sections of the cornea) and more advanced IFS in the central and inferior cornea (1A, 2A, 1B and 2B sections of the cornea).

interlamellar keratitis (PISK).¹⁻⁷ DLK presents as granular inflammatory cell deposits within the LASIK interface, usually showing a wave-like appearance.¹ It most often presents during the first postoperative week, although late-onset DLK cases have been reported in post-LASIK cases related with trauma, epithelial defects or other inflammatory or infectious conditions. Intraocular pressure (IOP) is normal in DLK cases. Post-LASIK interface fluid syndrome (IFS) typical case is that of a patient who presents between 2 weeks and 2 months after LASIK with corneal edema simulating DLK and steroid-induced IOP increase, usually missed, since applanation tonometry is erroneously measured lower than it actually is, due to the presence of fluid in the interface.²⁻⁶

PISK appears to be identical to DLK, but is present at a later time frame and is associated with a significant elevation IOP.⁷

TREATMENT

Corneal edema related with corticosteroid-induced intraocular pressure elevation was considered as the cause of the problem, presenting as IFS in the right eye and as PISK in the left one. Immediately fluorometholone was discontinued and systemic acetazolamide and topical brimonidine tartrate/timolol maleate were initiated.

OUTCOME AND FOLLOW-UP

Two days later the patient reported better vision, UCVA was 20/60, in the right eye and 20/200 in the left eye. BCVA was 20/60 with refraction of plano ($-0.75 \times 70^\circ$) D and 20/40 with -2.25 D sphere, respectively. IOP was 8 mm Hg in both eyes measured with applanation tonometry (both centrally and close to the limbus). The slit lamp examination showed that both the interface infiltrate and the anterior stromal haze had diminished in both eyes; there was no evidence of fluid in the interface of the right eye.

One month later, the UCVA and BCVA were 20/25 with a refraction of plano in the right eye; in the left eye (treated for near vision), distance UCVA was 20/200 and the BCVA was 20/20 with a refraction of -2.00 D sphere. The patient did not report any symptom.

DISCUSSION

In 1999 Lyle reported the first case of post-LASIK IFS associated with what they thought was DLK.² In 2000, Najman-Vainer *et al*³ reported the case of a patient with IFS, referred to them 6 months after surgery, who finally reached a visual acuity of light perception alone and the optic nerve cup-to-disc ratio was 1.0.

In 2007, Dawson *et al*⁸ published an ex vivo experimental study on IFS. This elegant work made fundamental contributions to the understanding of the physiopathology of the condition. They demonstrated that both, endothelial cell damage and high-pressure, may cause swelling of the LASIK interface.

The presence of fluid in the interface causes the central Goldmann tonometer measurements to be grossly erroneous.²⁻⁶ In IFS cases, IOP measurements in the corneal

periphery using Goldmann tonometry have shown to be much more accurate than central measurements.⁴

PISK cases have been published since 2002.⁷ Reviewing the clinical pictures we found that IFS and PISK, considered independent by many, share indeed many features. In fact, the first IFS patient reported in literature,² initially presented PISK in the right eye and IFS in the left eye. Five months after LASIK, a small amount of interface fluid was noted in the right eye, consistent with IFS diagnosis. Thus the patient developed IFS in the right eye more than four months after having had a clinical picture of persistent PISK. We think that this sequence of events suggests some kind of relation between these two conditions, and that PISK might be the initial stage of IFS. In literature, three more cases having a similar course (initially presenting as PISK and later as IFS) have been reported.^{5 6}

According to the temporal relation between PISK and IFS suggested by those referred cases^{2 5 6} and taking into account that PISK was present in one eye and IFS in the fellow eye, in the patient we herein report, it seems to us that a condition initially diagnosed as PISK may merely correspond to early stages of IFS. In fact, in the right eye of our patient interface fluid was present only in the inferior cornea. Both, slit lamp examination and OCT Visante, demonstrated that the fluid filled space progressively diminished in the upper part of the cornea. On the contrary, slit lamp examination showed that stromal interface haze compromised the whole cornea (figure 1). Thus, while the inferior cornea of that particular eye presented advanced IFS, the superior cornea presented features of an early stage of IFS, similar to the so-called PISK. Based on Dawson's classification of IFS,⁸ we consider that PISK might correspond to stage 1 or early stage 2 of IFS.

We agree with Waring⁹ and with Tourtas and Cursiefen¹⁰ that PISK is not a keratitis. Waring,⁹ coauthor of Dawson's ex vivo study,⁸ suggested that the name IFS ties the concepts of interface haze and interface fluid, together. We agree with him, but since the interface fluid in stages 1 and 2 (Dawson's classification⁷) may not be visible during the slit lamp examination, we think that a more appropriate term for the condition ought to be more general. We propose a new name in which 'pressure-induced' is not included, since this condition may be caused not only by elevated IOP but also by any condition leading to post-LASIK corneal edema.^{11 12} Given that it is not really an inflammation, 'keratopathy' is more suitable than 'keratitis'.^{9 10} Since when examined at the slit lamp it does not always combine with evident interface fluid, we think the word 'fluid' may be misleading and we did not include it in the name. We propose the term: post-LASIK edema-induced keratopathy (PLEK).

Since five percent of the general population is high steroid responders with significant increases in IOP (more than 15 mm Hg above baseline),¹³ and since these medications are very frequently used in LASIK patients, elevated IOP is the first cause of what we would like to describe as PLEK, and refractive surgeons should be aware of this possibility.

Learning points

- ▶ If post-laser in situ keratomileusis (LASIK) conditions known as interface fluid syndrome (IFS) and pressure-induced interlamellar keratitis (PISK) are not detected, severe visual sequelae may occur due to optic nerve damage, since usually they are related with ocular hypertension.
- ▶ Always rule out IFS in a case of post-LASIK corneal edema and haze, simulating diffuse interlamellar keratitis (DLK). Look for fluid in the interface, and check the applanation tonometry in the corneal periphery.
- ▶ The basic cause of IFS and PISK is not ocular hypertension but the corneal edema secondary to it. For that reason a new name is proposed: post-LASIK edema-induced keratopathy (PLEK).
- ▶ -Post-LASIK edema-related disorders are not inflammatory, so steroids are not indicated, and may aggravate the condition.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Gritz DC**. LASIK interface keratitis: epidemiology, diagnosis and care. *Curr Opin Ophthalmol* 2011;**22**:251–5.
2. **Lyle WA**, Jin GJ. Interface fluid associated with diffuse lamellar keratitis and epithelial ingrowth after laser in situ keratomileusis. *J Cataract Refract Surg* 1999;**25**:1009–12.
3. **Najman-Vainer J**, Smith RJ, Maloney RK. Interface fluid after LASIK: misleading tonometry can lead to end-stage glaucoma. *J Cataract Refract Surg* 2000;**26**:471–2.
4. **Galal A**, Artola A, Belda J, *et al*. Interface corneal edema secondary to steroid-induced elevation of intraocular pressure simulating diffuse lamellar keratitis. *J Refract Surg* 2006;**22**:441–7.
5. **Hamilton DR**, Manche EE, Rich LF, *et al*. Steroid-induced glaucoma after laser in situ keratomileusis associated with interface fluid. *Ophthalmology* 2002;**109**:659–65.
6. **Shaikh NM**, Shaikh S, Singh K, *et al*. Progression to end-stage glaucoma after laser in situ keratomileusis. *J Cataract Refract Surg* 2002;**28**:356–9.
7. **Belin MW**, Hannush SB, Yau CW, *et al*. Elevated intraocular pressure-induced interlamellar stromal keratitis. *Ophthalmology* 2002;**109**:1929–33.
8. **Dawson DG**, Schmack I, Holley GP, *et al*. Interface fluid syndrome in human eye bank corneas after LASIK: causes and pathogenesis. *Ophthalmology* 2007;**114**:1848–59.
9. **Waring GO III**. Appropriate term for post-LASIK corneal edema. *J Cataract Refract Surg* 2009;**35**:1482–3; author reply 1483.
10. **Tourtas T**, Cursiefen C. “PISK-it is” or “PISK-opathy”? *Cornea* 2012;**31**:107.
11. **Ortega-Usobiaga J**, Martin-Reyes C, Llovet-Osuna F, *et al*. Interface fluid syndrome in routine cataract surgery 10 years after laser in situ keratomileusis. *Cornea* 2012;**31**:706–7.
12. **Han SB**, Woo SJ, Hyon JY. Delayed-onset interface fluid syndrome after laser in situ keratomileusis secondary to combined cataract and vitreoretinal surgery. *J Cataract Refract Surg* 2012;**38**:548–50.
13. **Razeghinejad MR**, Katz LJ. Steroid-induced iatrogenic glaucoma. *Ophthalmic Res* 2012;**47**:66–80.

Copyright 2012 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit

<http://group.bmj.com/group/rights-licensing/permissions>.

BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Galvis V, Tello A, Revelo ML, Valarezo P. Post-LASIK edema-induced keratopathy (PLEK), a new name based on pathophysiology of the condition. *BMJ Case Reports* 2012;10.1136/bcr-2012-007328, Published XXX

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow