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Case Report

Post-LASIK interface fluid syndrome caused by steroid drops

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Abstract

Interface fluid syndrome after laser in situ keratomileusis (LASIK) is a rare but visually threatening postoperative complication. In this case series we present 8 post-LASIK eyes that developed interface fluid syndrome after prolonged steroid use. Patients presented with signs mimicking diffuse lamellar keratitis (DLK) that worsened with steroid treatment. Slit-lamp examination revealed corneal haze and an optically clear fluid-filled space between the flap and stroma. The IOP was high in all cases. Topical steroids were stopped and replaced with topical and systemic anti-glaucoma medications resulting in a dramatic improvement in visual acuity.

Keywords: Fluid interface, High intraocular pressure, LASIK, Myopia, Steroid drops

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Introduction

Laser in situ keratomileusis (LASIK) is the surgical procedure of choice for the correction of low to high myopia.[1–3] Steroid drops are typically prescribed after LASIK and continued for treatment of diffuse lamellar keratitis (DLK).[4,5] It is reported that 7–10% of the general population responds to corticosteroids with significant increase in intraocular pressure (IOP).[6]

Interface fluid syndrome is the most commonly used term to describe fluid collection in the flap interface.[7] It is a rare postoperative complication of LASIK and was first described in 1999 by Lyle[8] and subsequently in a number of case reports.[9–13] Others use the acronym PISK (pressure induced stromal keratitis).[14] Interface fluid syndrome is likely due to increased IOP.[15,16] Early diagnosis and differentiation from DLK is very important to avoid optic nerve damage and visual loss from the prolonged use of steroid drops.[11,17]

Case reports

Eight eyes of four patients with similar presentation after LASIK surgery with prolonged use of topical steroids are reported. Two patients underwent LASIK surgery in our centre, one patient in Egypt and one patient in Germany. All four patients presented three to four weeks after surgery with decreased vision, high IOP and fluid in the interface.

Case 1

A 34-year old female presented four weeks after LASIK complaining of gradually progressive blurring of vision. Her visual acuity was 20/200 in both eyes. Her preoperative myopic spherical equivalent was −4.50 D in both eyes and she had undergone LASIK with the NIDEK EC-5000 excimer laser (NIDEK Co. Ltd., Gamagori, Japan) using a Moria M2 microkeratome. The postoperative treatment regimen was topical steroid for one week and topical antibiotic for one

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week. The patient had a history of allergic disease and continued topical steroid past one week postoperatively.

On examination, there was moderate haze in both corneas. She was given artificial tears for one week and IOP was not measured. One week later the haze in both corneas was much worse. IOP was 32 mmHg in both eyes, measured with Goldmann applanation tonometry. Both optic discs were normal and perimetry results were normal. On slitlamp the flap was swollen but no interface fluid was noted. The patient was instructed to stop steroid use and asked to begin timolol and dorzolamide (Cosopt, MSD, UK) three times a day. The signs and symptoms improved dramatically, with visual blur disappearing, visual acuity returning to 20/22 and a clear cornea over 10 days of timolol and dorzolamide therapy. The final refraction was plano in both eyes.

Case 2

A 22 year old female presented one week after LASIK with signs similar to keratitis in both eyes. Visual acuity in the right eye was 20/50 and on the left 20/70. Her preoperative myopic spherical equivalent was −3.00 D in the right eye and −3.50 D in the left eye. The patient had undergone LASIK with the NIDEK EC-5000 excimer laser using a Moria M2 microkeratome. The postoperative treatment regimen was topical steroid for one week and topical antibiotic for one week. Both optic discs were normal. The patient was advised to increase the frequency of artificial tears and instill topical steroids twice daily. Ten days later, the keratitis persisted, vision started to decrease in both eyes. On slit-lamp examination fluid interface was noted in both corneas (Fig. 1). IOP with the Goldmann applanation tonometer was 26 mmHg in the right eye and 24 mmHg in the left eye. The patient was advised to stop topical steroid and start Cosopt three times daily and acetazolamide 250 mg twice daily. Within 4 days of therapy, the interface fluid decreased, the keratitis resolved and the vision improved to 20/20. The final refraction was plano in both eyes.

Case 3

A 23 year old male was referred to our centre because he was diagnosed as DLK and was unresponsive to the prescribed medications. The patient presented seven weeks after LASIK surgery in Egypt. His preoperative myopic spherical equivalent was −4.00 D in the right eye and −4.25 in the left eye. The patient had undergone LASIK with the Allegretto excimer laser (Alcon Inc., Fort Worth, TX. USA) using a mechanical microkeratome and his postoperative treatment regimen was the same as the previous two cases. Both optic discs were normal and the results of perimetry were within normal limits for both eyes. At presentation, the patient was taking topical steroids for keratitis but he was getting worse.

On slit-lamp examination, fluid interface was noted in both corneas and the IOP with Goldmann applanation tonometry was 28 mmHg in the right and 25 mmHg in the left. The patient was advised to stop topical steroids and was given Cosopt twice daily and acetazolamide for one week. The interface fluid improved within the first three days and the antiglaucoma medications were stopped after ten days. At final visit, both eyes were emmetropic.

Case 4

A 34 year old male had refractive surgery in Germany. His preoperative myopic spherical equivalent was −7.00 D in the right eye and −7.50 in the left eye. The patient had undergone LASIK with an Allegretto excimer laser using a laser keratome. His postoperative vision was excellent but began to decrease by the third week. The patient presented to our clinic seeking a second opinion and was using steroids on his own without medical advice. On examination, visual acuity was 20/50 in both eyes but there was haze in both corneas and had interface fluid on slitlamp examination. IOP with Goldman applanation tonometry was 33 mmHg in the right eye and 35 mmHg in the left eyes. Both optic discs

Figure 1. Slitlamp microscopy reveals interface fluid in case 2.

Table 1. Difference between diffuse lamellar keratitis and interface fluid syndrome.

	Diffuse lamellar keratitis	Interface fluid syndrome
History	Unknown	Prolonged use of steroid drops
Cause	Many causes such as marker pen, glove powders, meibomian gland secretions, povidone iodine solutions, bacterial wall endotoxins in autoclave reservoirs and cleaning solutions	Elevation of IOP
Inflammation	Yes	No
Onset	Early (2–5 days)	Late (2–4 weeks)
Pain	Yes	No
Vision	Decreased visual acuity	Decreased visual acuity
IOP	Normal	High (sometimes normal)
Refraction	Hypermetropic shift	Myopic shift
Treatment	Topical steroid	Topical and systemic anti-glaucoma, stop steroid
Visual recovery	Slow	Quick

were normal. The patient had a family history of glaucoma. The steroid drops were stopped and he was instructed to instill Cosopt three times daily and acetazolamide twice daily. All symptoms disappeared within 48 h of initiating therapy. His final refraction was −0.50 in both eyes.

Discussion

All four patients (eight eyes) underwent uneventful LASIK and had similar presentation with the exception of the first case which had haze but no obvious interface fluid seen on slitlamp. All patients used topical steroids for an extended period and there was a family history of glaucoma in one case. Two patients used topical steroid for three weeks, one patient used it for four weeks and one patient used it for seven weeks.

The slit-lamp findings and visual deterioration appeared identical to DLK in the four patients. All cases, however, presented outside of the first postoperative week and were not associated with any history of trauma. All cases failed to respond to high-dose topical steroids. Significant IOP elevations were noted in all cases, and the interface changes responded dramatically to the discontinuation of steroids and the lowering of IOP.

Goldmann applanation tonometry results showed ocular hypertension in all eyes. However in similar cases Goldmann applanation tonometry can be (inaccurately) low. No eye had inflammatory signs in the anterior chamber and no patient complained of pain. In all four patients, fluid was present in the interface that resulted from high ocular hypertension secondary to a hyper-response to steroids. All eight eyes had a normal optic disc. Visual field perimetry was performed in two patients and was normal in both cases. All patients recovered uncorrected visual acuity to their original post-LASIK levels.

This syndrome appears identical to post-LASIK DLK, but presents later and is associated with a significant elevation of IOP. Unlike DLK, this syndrome does not respond to steroids and resolves with lowering of the IOP (Table 1).

The increase of IOP causes the transudation of fluids through the endothelium from zones with high pressure to zones with lower pressure.[18] After LASIK, there is a virtual space between the flap and the stromal bed, the junction between the flap and the stroma is not able to bear the increase in pressure; therefore, the fluid gathers in the interface.[11]

Inaccurate applanation tonometry measurements post-LASIK due to the reduction in corneal thickness are well documented.[19] IOP measurement with Goldmann applanation tonometer can be low in some cases, and the diagnosis has to be confirmed by clinical exam and if available, by optical coherence tomography.[20] Reliable measurements of IOP are necessary after LASIK especially for glaucoma patients.

The measuring procedure and anatomical changes of the eye can lead to errors during applanation tonometry. In eyes with previous LASIK, Goldmann applanation tonometry measured IOP significantly lower than a pneumatonometer. IOP measurements with a pneumatonometer are independent of central corneal thickness. Hence, pneumatonometry is considered a more reliable method of IOP estimation than Goldmann applanation tonometry in eyes that have undergone LASIK.[21] Compared to Goldmann tonometry, the Tonopen (Reichert Technologies, Depew, NY, USA) appears to be less affected by the relative flattening of the cornea, thinning, and decreased rigidity of the central cornea following corneal refractive surgery.[22] The IOP measured on the peripheral cornea is closer to the actual IOP. Therefore, it is more accurate and reliable to measure IOP on the peripheral cornea than centrally after LASIK.[23]

Refractive surgeons should be cautious of cases of DLK that do not respond to steroid treatment. It has been reported that presumed late-onset diffuse lamellar keratitis of uncertain aetiology following uneventful LASIK can simulate or progress to interface fluid syndrome after treatment with topical corticosteroid.[13,24]

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