Clinical Case Report

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Delayed onset of pressure-induced interlamellar stromal keratitis in a patient with recurrent uveitis

A case report

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Abstract

Introduction: Corticosteroid treatment for uveitis can lead to delayed-onset pressure-induced interlamellar stromal keratitis (PISK), even years after laser in situ keratomileusis (LASIK).

A 35-year-old man presented to our clinic after experiencing blurred vision in his left eye for 1 month. For the past month, he had been prescribed topical steroid and anti-glaucomatous medication. He had undergone LASIK for both eyes 5 years earlier, and had suffered uveitis attacks in his left eye over the last 2 years.

Slit-lamp examination revealed stromal haziness with interface fluid accumulation in the left eye. The left eye showed an intraocular pressure (IOP) of 35 mm Hg and visual acuity of 6/20. Anterior segment ocular coherence tomography (OCT) confirmed the diagnosis of PISK. Steroid treatment was tapered, and latanoprost treatment was started. One month later, the patient's symptoms resolved, with IOP reduced to 10 mm Hg and visual acuity increased to 6/6 in the left eye. Latanoprost treatment was discontinued to avoid potential uveitis reactivation, and the patient's visual field defect progressed and IOP rebounded. Due to evident glaucomatous damage, trabeculectomy was suggested but was refused.

Conclusion: Patients with PISK plus uveitis should be treated with a tailored regimen involving corticosteroid and antiglaucomatous medication or surgical intervention based on the individual condition. Early recognition and appropriate treatment may aid in preventing severe visual sequela in such patients.

Abbreviations: dB = decibel, DLK = diffuse lamellar keratitis, IOP = intraocular pressure, LASIK = laser in situ keratomileusis, mg = milligram, mm Hg = millimeter of mercury, PISK = pressure-induced interlamellar stromal keratitis.

Keywords: glaucoma, pressure-induced interlamellar stromal keratitis, steroid, uveitis

1. Introduction

Diffuse lamellar keratitis (DLK) is a form of diffuse infiltration or inflammation that is confined to the flap interface and can be focal or multifocal. It occurs as a rare complication after laser in situ keratomileusis (LASIK) surgery, typically presenting on postoperative days 1 to 3.[1] In most cases, DLK is mild and asymptomatic. However, severe cases often require hourly application of topical steroid drops or even surgical intervention. Without treatment, severe DLK can cause flap melting and necrosis.[2]

In 2002, Belin et al[3] described 4 cases presenting slit-lamp findings and visual degradation seemingly identical to DLK, but with onset beyond the first postoperative week and no response to high-dose topical steroids. All 4 cases exhibited significantly elevated intraocular pressure (IOP). The interface changes dramatically responded to both IOP-lowering medication and discontinuation or tapering of the topical steroids. They named this condition pressure-induced interlamellar stromal keratitis (PISK). As the 2 disorders share a similar clinical appearance but require different treatment, it is highly important to differentiate PISK from DLK.[4] Delayed treatment of PISK can lead to substantial glaucomatous field loss and optic atrophy.[5]

2. Case report

A 35-year-old man presented to our clinic after experiencing blurred vision in his left eye for 1 month. During the past month, he had been using 1% topical prednisolone, along with 250 mg acetazolamide 4 times daily, and 0.15% brimonidine and 0.5% timolol/2% dorzolamide twice daily to treat uveitis in his left eye. The patient had undergone LASIK for both eyes 5 years earlier, and had experienced uveitis attacks in the left eye over the past 2 years.

Upon presentation at our clinic, intraocular pressure measured by noncontact tonometry was 9 mm Hg in the patient's right eye and 35 mm Hg in his left eye. His best-corrected visual acuity was

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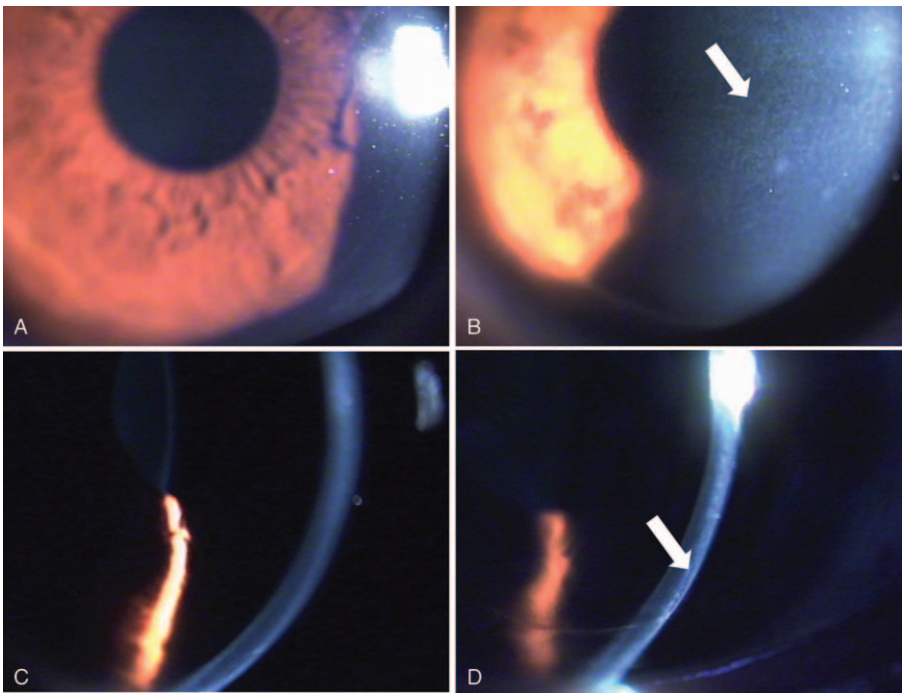

Figure 1. A and C, Slit-lamp examination revealed normal finding in the right eye. B and D, The left eye exhibited stromal haziness with fluid accumulation in the interface (white arrows).

6/6 in the right eye and 6/20 in the left eye. Slit-lamp examination revealed grossly normal in his right eye (Fig. 1A and C) and stromal haziness with fluid accumulation at the interface in his left eye (Fig. 1B and D). The anterior chamber exhibited no significant inflammation or sequelae of uveitis. Fundoscopic examination revealed an enlarged cup-to-disc ratio of about 0.8 in his left eye, without retinal lesion or vitreous opacity. The cup-to-disc ratio in his right eye was ~0.5. Anterior segment ocular coherence tomography (OCT) also showed interface fluid accumulation in the left eye (Fig. 2). The patient was diagnosed with steroid-induced glaucoma with elevated intraocular pressure-induced stromal keratitis. We shifted the patient from 1% topical prednisolone to 0.5% loteprednol 4 times a day with gradual tapering. Due to the uncontrolled IOP, the patient was also prescribed 0.005% latanoprost each night.

One month later, the interface fluid and haziness in the left eye were resolved. The intraocular pressure in the left eye was 10 mm Hg as measured with a Tono-Pen outside the LASIK flap area, and visual acuity with correction was 6/6 in both the eyes. Automated perimetry revealed a mean deviation of −1.31 dB in the right eye and −6.07 dB in the left eye (Fig. 3A). During the next month, OCT revealed reduced thickness of the superior and inferior retinal nerve fiber layer in the left eye (Fig. 4). Latanoprost treatment was discontinued to avoid potential

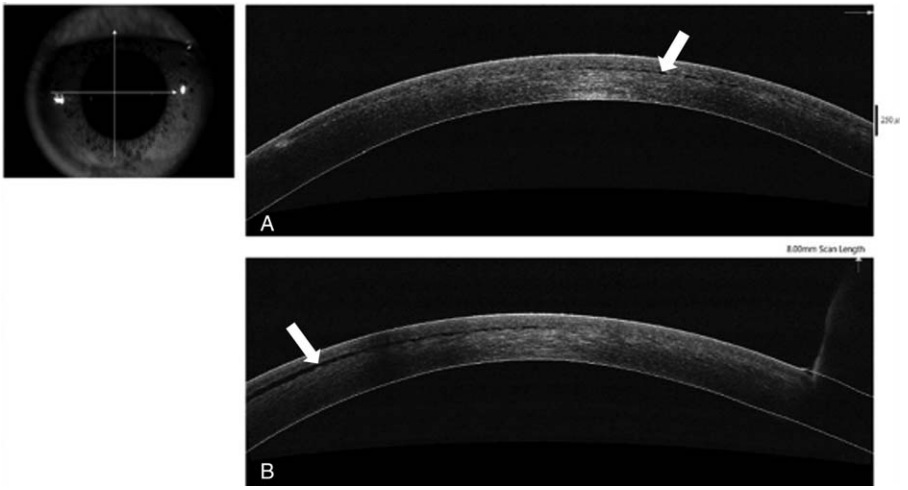

Figure 2. A and B, Anterior segment optical coherence tomography (horizontal and vertical scans) revealed obvious interface fluid accumulation (white arrows) in the left cornea.

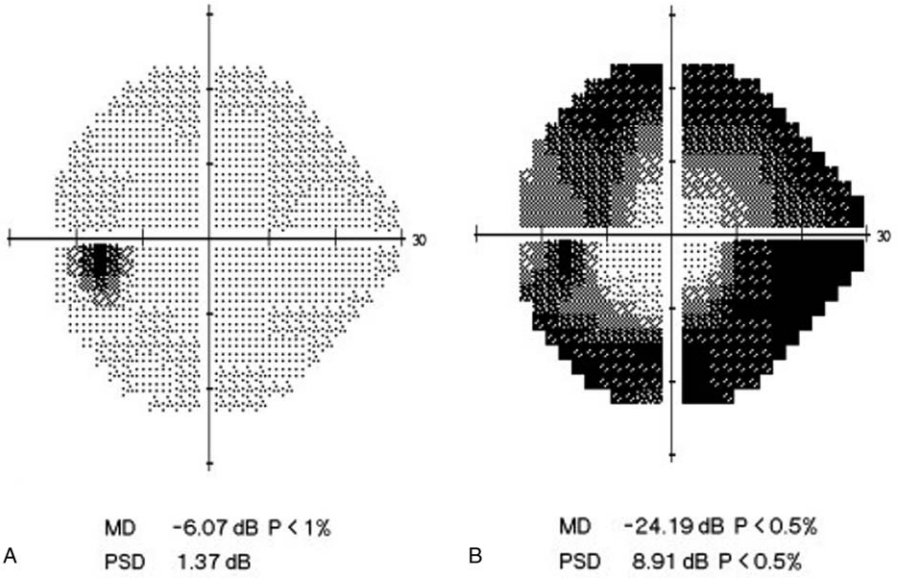

Figure 3. A, Automated perimetry showed a mean deviation of −6.07 dB in the left eye after resolution of the interface fluid in the cornea. B, Two months later, the mean deviation in the left eye had progressed to −24.19 dB.

uveitis reactivation. Two months later, the visual field revealed a central island in the left eye (Fig. 3B) and the intraocular pressure rebounded after latanoprost discontinuation; therefore, trabeculectomy was suggested. However, the patient refused surgery and did not return to our clinic. The patient's informed consent has been obtained thereafter.

3. Discussion

The clinical pictures of PISK and DLK are nearly identical, except that PISK often occurs beyond 1 week after LASIK and is accompanied by significantly elevated IOP. Nordlund et al[6] reported that the average onset of PISK was 17 days after LASIK. However, late-onset DLK in LASIK patients has also been described, and it is crucial to differentiate these 2 diseases since they require contradictory treatments.[7–9] Our patient suffered from PISK that started 5 years after undergoing LASIK. Lee et al[10] reported a case of PISK occurring 9 years after LASIK, but with atypical presentation. In contrast, our present case involved typical PISK presentation, including stromal haze, interface fluid accumulation, and high IOP following steroid treatment. Moreover, anterior segment OCT revealed space-occupying interface fluid accumulation, which can falsely lower IOP measurements in PISK patients.[4] For the assessment of IOP in PISK patients, pneumotonometry and Tono-Pen are favored over Goldmann applanation tonometry, since Goldmann applanation

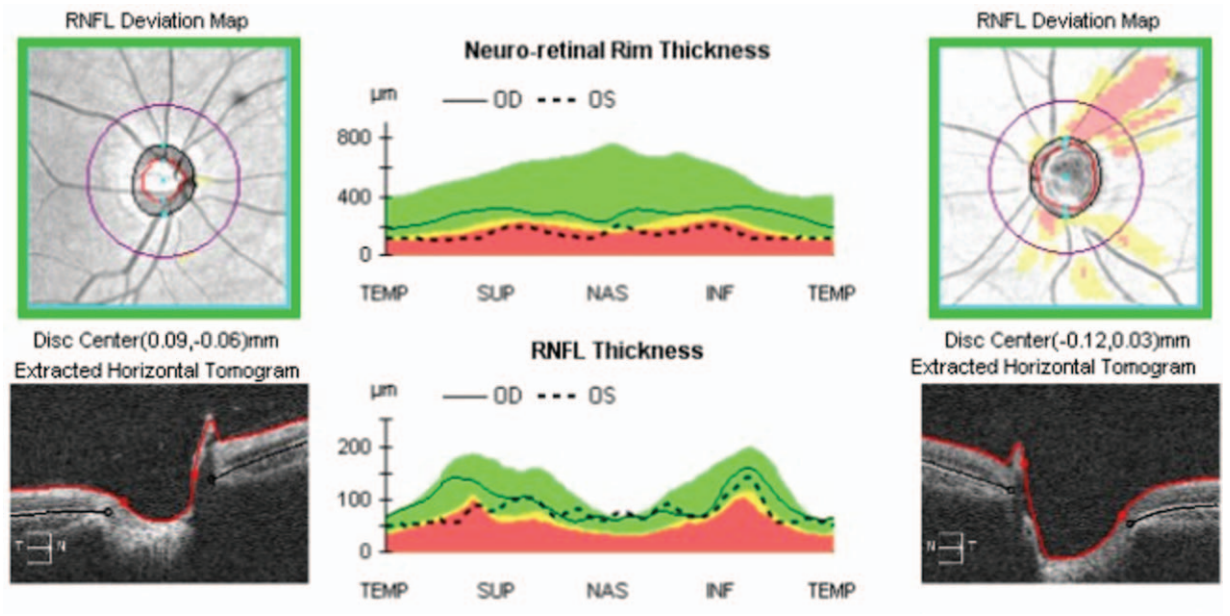

Figure 4. Optical coherence tomography revealed reduced thickness of the superior and inferior retinal nerve fiber layer in the left eye.

tonometry is more easily affected by cornea structural alterations, such as corneal thinning and space-occupying interface fluid accumulation.[4] It is postulated that the stromal edema and fluid accumulation in PISK results from increased IOP, which may impair corneal fluid dynamic function.[6,10] Common treatments include the administration of IOP-lowering agents and the cessation of steroid treatment.[10]

The patient in our case had previously suffered a uveitis attack in his left eye and had thus been prescribed topical steroid treatment before his presentation at our clinic. Posner-Schlossman syndrome was initially suspected due to the increased intraocular pressure; however, no typical keratic precipitate was detected on the endothelium and the intraocular pressure decreased only after discontinuing steroid treatment. We also performed anterior chamber tap and PCR for herpes simplex virus, cytomegalovirus, and varicella zoster virus, which yielded negative results. Thus, the cause of uveitis remains undetermined as the patient did not return for further examinations.

While corticosteroid treatment is recommended as first-line treatment for uveitis,[11] risks include glaucoma development and, as in our case, PISK development after LASIK. According to Panek et al,[12] frequent monitoring and adequate control of both intraocular pressure and intraocular inflammatory status are critical safety measures when treating uveitis with corticosteroids. Medical or surgical antiglaucomatous therapy should be administered in cases with pre-existing optic nerve damage, and in cases of continuously increasing or persistently high intraocular pressure, which may ultimately lead to optic nerve damage. If the intraocular pressure continues to rise or remains unchanged, corticosteroid treatment should be tapered to the lowest dosage that sufficiently controls the inflammation.[12] Overall, the treatment of PISK with uveitis remains difficult, and there are no clear guidelines available. We suggest that the treatment involving immunosuppressive drugs and antiglaucomatous therapy should be tailored to the individual case, based on the severity of inflammation, glaucomatous status, and PISK disease process. Further studies are needed to evaluate the treatment effects.

In summary, here we report a typical PISK case with onset 5 years after LASIK surgery. Regular intraocular pressure tracking is important in patients receiving long-term steroid medication for uveitis. Early recognition and appropriate treatment may aid in the prevention of severe visual sequela.

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