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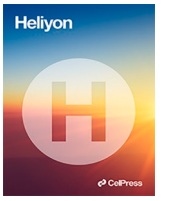

Interface fluid syndrome after small incision lenticule extraction surgery secondary to posner schlossman syndrome - A case report

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ABSTRACT

Purpose: This report describes a case of interface fluid syndrome (IFS) secondary to Posner Schlossman Syndrome (PSS) following small incision lenticule extraction (SMILE) surgery.
Case presentation: A 19-year-old male was diagnosed with IFS secondary to PSS in his left eye 1 month after undergoing SMILE. Detailed patient history and clinical findings, auxiliary examination results, and short-term follow-up are reported. In this patient, the IFS was caused by elevated intraocular pressure (IOP) due to PSS. Treatment with topical steroids in combination with anti-glaucoma drops led to complete regression of the fluid, and there was no recurrence during a 7-month follow-up period.
Conclusion: IFS is a potential complication of SMILE, and anterior segment ocular coherence tomography (AS-OCT) can definitively diagnose the condition. This case demonstrates that the treatment for IFS should be based on the underlying cause, and requires prompt and vigorous management for resolution.

1. Introduction

Interface fluid syndrome (IFS) is a rare complication of lamellar refractive surgeries such as laser-assisted in situ keratomileusis (LASIK) and small incision lenticule extraction (SMILE), and is characterized by fluid accumulation in the intrastromal space [1,2]. It usually occurs in the early postoperative period. IFS is commonly caused by a steroid induced elevation of intraocular pressure (IOP), endothelial decompensation, and uveitis [3–11]. Cases of IFS caused by Posner Schlossman Syndrome (PSS) after LASIK have been reported [12]. However, to our knowledge this is the first reported case of IFS caused by PSS after SMILE surgery.

2. Case presentation

A 19-year-old man underwent SMILE in both eyes to correct a myopic refractive error. Preoperative best corrected distance visual acuity (BCVA) was 20/20 in both eyes (OU), with a subjective refraction of −3.75/-0.25 × 180 in the right eye (OD) and −4.50 in the left eye (OS). Slit-lamp examination (SLE) demonstrated a clear cornea without opacities. The anterior chamber was deep without signs of inflammation OU. The dilated fundoscopy examination was unremarkable, and showed normal optic nerves, macula, and peripheral retinas. His IOP was 19.1 mm Hg OD, and 19.3 mm Hg OS, with a central corneal thickness (CCT) of 543 mm OD and 546 mm OS. Postoperative medications included gatifloxacin eye gel QID for 15 days, tobramycin-dexamethasone (Tobradex) eye drops

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QID for 7 days, deproteinized calf blood extract eye drops, and artificial tears 4 times daily OU.

At the day 1 and week 1 postoperative visits, examination of both eyes revealed corneal incisions that were healing well, and clear caps without anterior chamber inflammation. His uncorrected distance visual acuity (UDVA) was 20/20 OD and 20/25 OS. His IOP at 1 week was 14.5 mm Hg OD and 13.1 mm Hg OS. He was told to stop using the topical steroid drops, and return for a 1-month follow-up visit.

On postoperative day 32 he presented with left eye pain and redness. He stated the symptoms began 3 days prior, and he attributed them to staying up late and drinking. He denied any history of systemic diseases, allergies, and similar episodes. The UDVA was 25/20 OD and 20/40 OS, and IOP was 13.5 mm Hg OD and 35 mm Hg OS. Slit-lamp examination revealed left eye ciliary congestion, corneal edema, mild stromal haze, and several medium-sized keratic precipitates (KPs) on the corneal endothelium (Fig. 1a). Anterior segment ocular coherence tomography (AS-OCT) also showed edema of the cornea and interface fluid accumulation in the left eye (Fig. 2a). The anterior chamber angle of both eyes was open, with normal visual fields and optic discs. Based on the clinical presentation and AS-OCT results, the preliminarily diagnosis was IFS secondary to PSS. He was treated with a combination of 2 types of eye drops to the left eye to control the IOP (brinzolamide-timolol and brimonidine 0.2 %, twice daily), and 1 % prednisolone acetate to treat the PSS.

Three days after beginning treatment, he reported less pain and considerable improvement in vision in his left eye. The IOP was reduced to 13.1 mm Hg OD and 6.9 mm Hg OS, and the UDVA in his left eye had recovered to 20/25. Slit-lamp examination showed shrinkage of the KPs (Fig. 1b), and AS-OCT showed regression of corneal edema and absorbed interface fluid (Fig. 2b). Corneal endothelial densities measured by specular microscopy were 3428 cells/mm (OD) and 3600 cells/mm (OS). The 1 % prednisolone and brimonidine 0.2 % were discontinued, and he was begun on 0.02 % fluorometholone.

One month later (2 months postoperative), the IOP was reduced to within the normal range, and there was resolution of the anterior segment inflammation and corneal edema (Figs. 1c and 2c). The UDVA was 25/20 OD and 20/20 OS. The IOP was 13 mm Hg OD and 8.3 mm Hg OS. At this time he was off all topical medications. There was no recurrence during a 7-month follow-up period, and the IOP had stabilized. The patient was very pleased with his result.

3. Discussion

Posner Schlossman Syndrome (PSS) represents a specific type of anterior uveitis characterized by recurrent episodes of unilateral, acute elevations of IOP, that are out of proportion to the mild anterior chamber inflammation. PSS is associated with herpes simplex virus (HSV), varicella zoster virus (VZV), and cytomegalovirus (CMV) infections, with CMV infection being the leading cause [13,14].

CMV infection may manifest as PSS and Fuch's uveitis syndrome (FUS)-like anterior uveitis, and there tends to be a delay in the diagnosis of a viral etiology. Unlike in PSS, the inflammation tends to recur when topical corticosteroids are tapered in CMV [15,16]. FUS was ruled out in view of the acute symptoms of severe pain with ocular redness, and the absence of characteristic stellate KPs, iris changes, or vitritis. The differential diagnosis also included an infection with other viruses that commonly cause corneal endotheliitis, for example HSV and VZV as mentioned above.

It is important to distinguish a viral infection from a nonviral cause, because treatment with steroids alone without appropriate antiviral therapy can increase the risk of corneal endotheliitis [17]. However, in order to make definitive diagnosis of a viral etiology aqueous humor testing is required, which was not approved by our patient.

We did not treat our patient with antiviral drugs; instead he was treated with topical steroids and anti-glaucoma eye drops, and the inflammation was quickly controlled and his IOP remained stable. There was no corneal endothelial cell loss and no recurrence after stopping all medications. Therefore, our diagnosis of PSS is based on clinical manifestations and treatment outcome. In managing our patient, there were no diagnostic tests performed that could help identify anterior uveitis due to a viral infection, such as polymerase chain reaction (PCR) and confocal microscopy. Additionally, given the short follow-up period, should PSS recur in the future, it will be necessary to conduct laboratory tests to rule out other potential causes.

The relation between refractive surgery and PSS is not clear. Moshirfar et al. compared the incidence of uveitis in patients with LASIK and non-LASIK eyes, and reported that LASIK did not increase the incidence of uveitis [18]. Suarez et al. reported there is no cause-effect relation between LASIK and anterior uveitis [19]. As such, there is likely no relation in our patient between the occurrence of PSS and the SMILE procedure. However, it is possible that the inflammation in our patient could have been due to another cause,

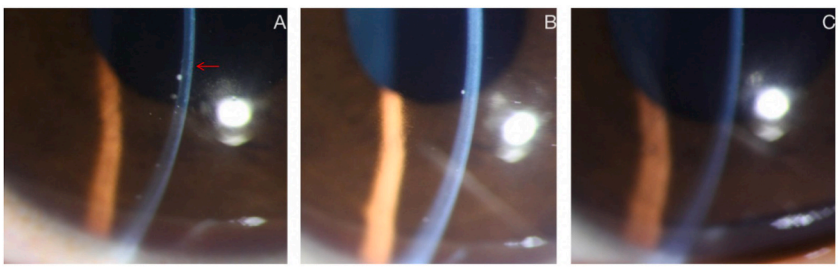

Fig. 1. Anterior slit-lamp images at different times postoperatively. A) Edematous cornea with mutton fat keratic precipitates were visible on 2022-07-11. B) A reduction of corneal edema and keratic precipitates was noted on 2022-07-14. C) Complete resolution of corneal edema and anterior chamber inflammation was noted on 2022-07-21.

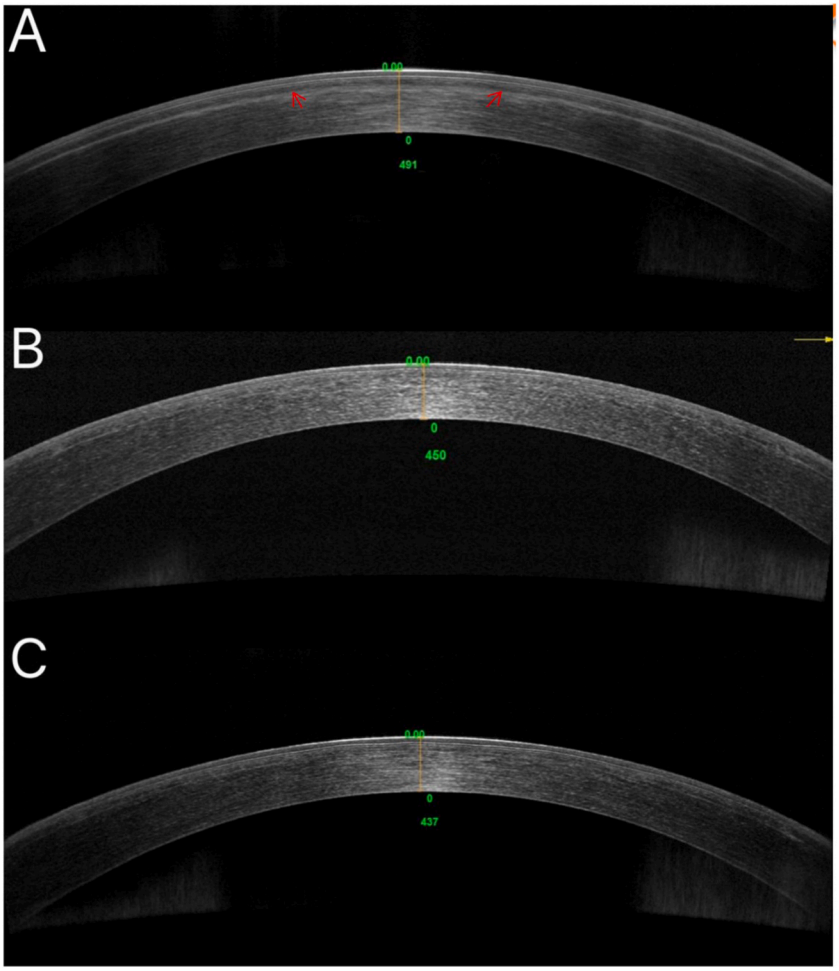

Fig. 2. Anterior segment optical coherence tomography (AS-OCT) images at the same time points as in Fig. 1. A) A hyporeflective area (corresponding to the area of fluid accumulation at the interface) and stromal hyper-reflectively are noted. B) Resolution of the interface fluid and decreased stromal hyper-reflectively. C) Substantial improvement in corneal edema is noted.

such as a viral infection, which was not identified in our examination and work-up. An elevated IOP at the onset of PSS after refractive surgery can lead to complications such as Interface fluid syndrome (IFS) and corneal ectasia [20].

IFS is a relatively uncommon, vision-threatening, flap-related complication of LASIK, characterized by accumulation of aqueous humor in the flap interface. The reported incidence of an interface fluid collection after LASIK is 1 in 6,000, and it usually occurs in the early postoperative period [1,21]. However, cases of IFS occurring several years after LASIK have been reported [4]. As such, IFS is considered to be a potential lifelong complication after LASIK, that can occur at any time after surgery [12]. With the widespread use of SMILE, cases of IFS after SMILE are being reported [21–24]. The connection between the corneal cap and the stromal bed is loose after SMILE, creating a potential space where fluid can accumulate. Thus, the incidence of IFS after SMILE may be much greater than indicated by the number of published reports.

Clinically, mild IFS is similar to diffuse lamellar keratitis (DLK) and is thus easily misdiagnosed. DLK is self-limiting, exhibits a good response to steroids, and can be diagnosed by Anterior segment ocular coherence tomography (AS-OCT) [25]. Conventional slit-lamp examination cannot detect mild interlayer fluid, yet mild interlayer fluid can be timely diagnosed by AS-OCT and treatment can be started promptly.

IOP elevation is a distinctive feature of most cases of IFS, and the IOP can vary markedly. In patients with severe IFS with significant fluid accumulation, the IOP tends to be low due to thinning of the central cornea and the buffering effect of the fluid [26]. In the case presented herein, the patient had moderate corneal edema and mild interface fluid accumulation. Dawson et al. described 3 stages of IFS, and the findings in our patient are consistent with stage 2 IFS as described by Dawson et al. [27]. However, the patient's IOP was still relatively high.

Common causes of IFS include steroid-induced ocular hypertension, primary or secondary glaucoma, uveitis, and corneal endothelial damage due to other causes [3–11]. Steroid-induced ocular hypertension is the most common cause of IFS, and symptoms can be relieved by cessation of topical corticosteroids and treatment with medications to lower the IOP. In our patient, postoperative steroid

administration did not result in IOP elevation. Instead, the inflammation led to excessive secretion of aqueous humor, and the exudate blocked the aqueous humor outflow channels resulting in increased IOP. Additionally, direct damage to the corneal endothelium from inflammation may cause IFS.

IFS should be treated based on the cause of the condition [28]. In this case, treating the primary condition was key for treating IFS, as IFS was triggered by PSS. Aggressive anti-inflammatory treatment with corticosteroids combined with medications to lower the IOP is the primary treatment for PSS. However, there have been reports of IFS caused by treating recurrent PSS with corticosteroids. Therefore, it is necessary to use steroids cautiously, or use non-steroidal anti-inflammatory drugs (NSAIDs) to suppress the inflammation. Moreover, close observation and long-term follow-up are required.

4. Conclusion

PSS is self-limiting with good prognosis. Present treatments can only control the symptoms of PSS, and cannot completely prevent a recurrence of PSS. After lamellar refractive surgery, increased IOP or corneal endothelial dysfunction from any cause can trigger corneal edema and lead to IFS. Determining the cause of the disease helps to make an early diagnosis, and timely treatment can lead to good results. Clinicians should pay more attention to the IOP of patients who have received refractive surgery, maintain good communication with them, and emphasize the importance of long-term follow-up.

Ethics statement

Review and approval by an ethics committee was not needed for this study because of the retrospective nature of the report.

Consent for participate

Informed consent was obtained from patient involved in the study.

Consent for publication

The patient provided informed consent for the anonymous publication of case details and images.

Data availability statement

Data will be made available on request.

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CARE guidelines

The work has been reported in line with the CARE-checklist-English-2013.

CRediT authorship contribution statement

Dilinigeer Mokumu: Writing – review & editing, Writing – original draft. **Wenfei Hu:** Resources, Data curation. **Ailifeire Damaola:** Resources. **Junshu Wu:** Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Not applicable.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/ j.heliyon.2023.e21863.

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