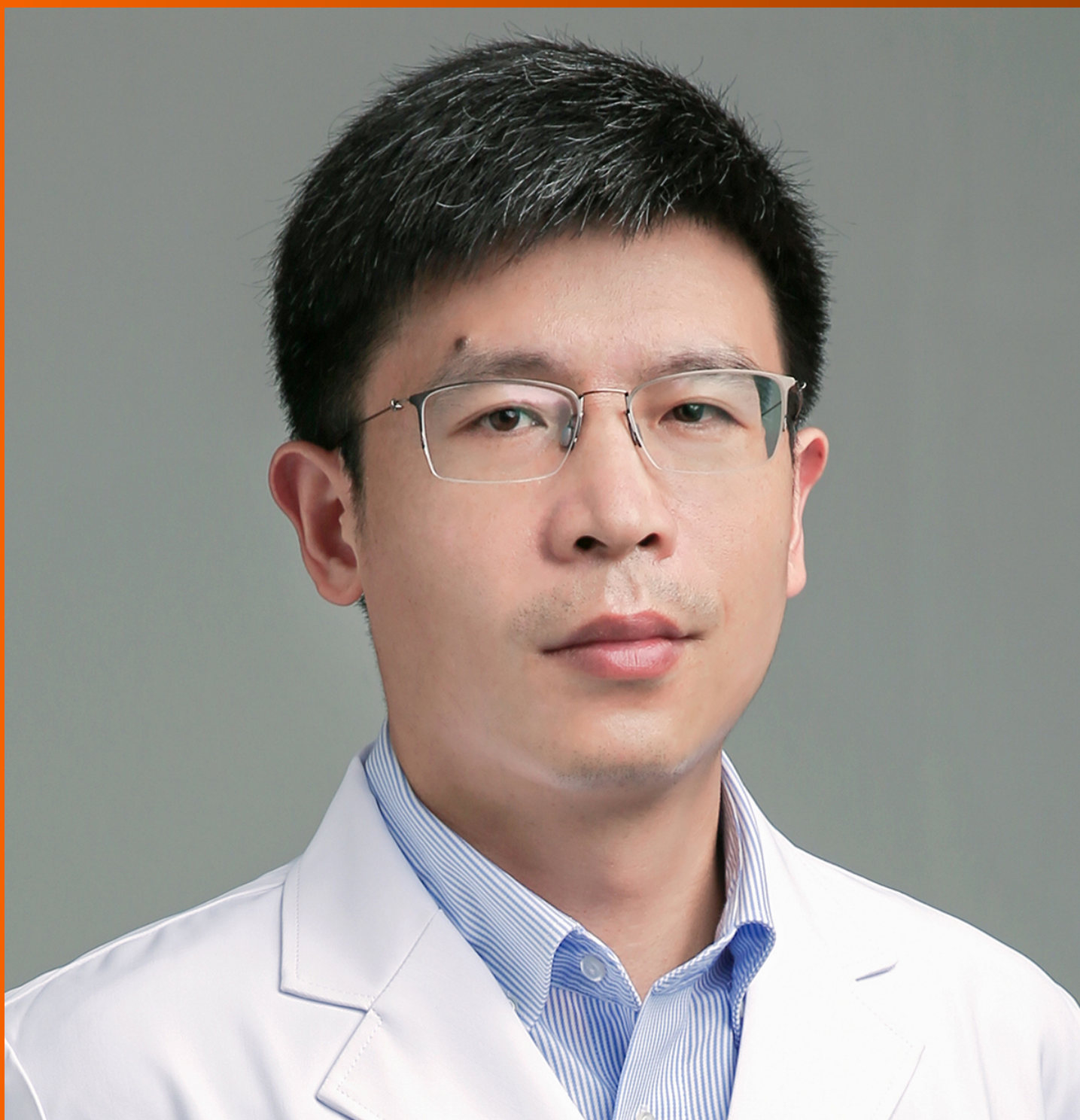


# World Journal of *Clinical Cases*

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## Delayed diffuse lamellar keratitis after small-incision lenticule extraction related to immunoglobulin A nephropathy: A case report

Ting-Ting Dan, Tai-Xiang Liu, Yi-Lu Liao, Zong-Ze Li

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### Abstract

#### BACKGROUND

Diffuse lamellar keratitis (DLK) is a complication of laser-assisted *in situ* keratomileusis (LASIK). This condition can also develop after small-incision lenticule extraction (SMILE) with a distinctive appearance. We report the case involving a female patient with delayed onset DLK accompanied by immunoglobulin A (IgA) nephropathy.

#### CASE SUMMARY

A 22-year-old woman was referred to our department for DLK and a decline in vision 1 mo after undergoing SMILE. The initial examination showed grade 2 DLK in the flap involving the central visual axis of the right eye. She was immediately administered with a large dose of a topical steroid for 30 d. However, the treatment was ineffective. Her vision deteriorated from 10/20 to 6/20, and DLK gradually worsened from grade 2 to 4. Eventually, interface washout was performed, after which her vision improved. DLK completely disappeared 2 mo after washout. Six months after SMILE, the patient was diagnosed with IgA nephropathy due to a 4-year history of interstitial hematuria.

#### CONCLUSION

DLK is a typical complication of LASIK but can also develop after SMILE. Topical steroid therapy was ineffective in our patient, and interface washout was required. IgA nephropathy could be one of the factors contributing to the development of delayed DLK after SMILE.

**Key Words:** Diffuse lamellar keratitis; Small-incision lenticule extraction; Immunoglobulin A nephropathy; Laser-assisted *in situ* keratomileusis; Case report

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**Core Tip:** Diffuse lamellar keratitis (DLK) is a typical complication of laser-assisted *in situ* keratomileusis but could also develop after small-incision lenticule extraction (SMILE). Topical steroid therapy was ineffective in our patient, and interface washout was required. Immunoglobulin A nephropathy could be one of the factors contributing to the development of delayed DLK after SMILE.

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## INTRODUCTION

A 22-year-old woman was referred to our department for diffuse lamellar keratitis (DLK) and a decline in vision 1 mo after undergoing small-incision lenticule extraction (SMILE). The initial examination showed grade 2 DLK in the flap involving the central visual axis of the right eye (OD). She was immediately administered with a large dose of a topical steroid for 30 d. However, the treatment was ineffective. Her vision deteriorated from 10/20 to 6/20, and DLK gradually worsened from grade 2 to 4. Eventually, interface washout was performed, after which her vision improved. DLK completely disappeared 2 mo after washout. Six months after SMILE, the patient was diagnosed with immunoglobulin A (IgA) nephropathy due to a 4-year history of interstitial hematuria.

## CASE PRESENTATION

### Chief complaints

A 22-year-old female patient presented with a 1-wk history of gradual vision loss.

### History of present illness

The patient had undergone bilateral SMILE 1 mo before and showed no other medical history.

### History of past illness

The patient's medications did not contribute to her vision loss.

### Personal and family history

The patient's personal and family history did not contribute to her vision loss.

### Physical examination

The preoperative best-corrected distance visual acuity (DVA) was 20/20 in both eyes (OU) with refraction of -7.25 diopter sphere (DS) -0.5 diopter cylinder (DC)\*20 OD and -6.50 DS -0.50 DC\*148 in the left eye (OS). The results of slit-lamp and dilated fundus examinations were normal, with no sign of dryness or superficial punctate keratopathy. The intraocular pressure (IOP) was 11.3 mmHg OD and 17 mmHg OS. Central corneal thickness was 650  $\mu$ m OD and 642  $\mu$ m OS. Specular microscopy revealed an endothelial cell density of 2756 cells/mm<sup>2</sup> OD and 2929 cells/mm<sup>2</sup> OS with a uniform endothelial cell pattern without any sign of abnormalities.

### Laboratory examinations

The laboratory findings, including those of routine blood examination and tests for HBV, HCV, HIV, and RPR, were unremarkable.

## FINAL DIAGNOSIS

DLK after SMILE.

## TREATMENT

The patient's postoperative regimen consisted of topical levofloxacin 0.3% and artificial tears 4 times a



day hourly OU, and topical fluorometholone 0.1% OU 4 times daily for the first week with the daily dose reduced by one drop every week. At her 1-wk postoperative visit, the uncorrected DVA (UDVA) was 16/20 OD and 16/20 OS. The manifest refraction at 1 wk was +0.5 DS OD and +0.25 OS. At this point, the IOP was 11.3 mmHg OD and 17 mmHg OS. She was told to continue tapering his medication regimen and return for her 1-mo follow-up visit.

However, 1 mo later, the patient presented with sudden worsening of symptoms; her vision was hazy and she did not experience pain or any other sensation, which she started experiencing 1 wk before the visit. The UDVA was 10/20 OD and 16/20 OS, and the manifest refraction was -1.25 DS = 16/20 OD. OD showed large focal infiltrates in the flap involving the central 6-mm visual axis (grade 2 DLK). OS was slightly infiltrated (grade 1 DLK). The IOP was 7.5 mmHg OD and 8.0 mmHg OS. A Pentacam assessment showed that the average optical density was 18.4 (Figure 1) OD and 13.8 OS. On the basis of these findings, she was diagnosed as showing DLK with myopia OD. She was advised to be treated with fluorometholone 0.1% OD 6 times daily 1 wk later. Her symptoms did not improve; therefore, she was prescribed 1% prednisolone acetate eye drops OD 8 times daily instead of Tobradex, the frequency of which was gradually reduced to 6 times daily for 1 wk. Two weeks later, *i.e.*, 2 mo after the operation, she presented with worsening of symptoms and cloudy vision OD. The UDVA declined to 6/20 OD. The manifest refraction was -1.50 DS = 14/20 OD with more large focal infiltrates in the flap involving the central 6-mm visual axis compared to before (grade 4 DLK). The IOP was 8.5 mmHg, and a Pentacam assessment showed that the average optical density was 19.5 D (Figure 2). Interface washout was eventually performed with a balanced salt solution and 0.1 mg dexamethasone. Her treatment regimen then consisted of topical fluorometholone 0.1% OD 4 times daily for the first week with the daily dose reduced by one drop every week. Thirteen days later, the UDVA was 10/20 -0.75 DS -0.75 DC\*38 = 16/20 OD, and there were few focal infiltrates in the flap involving the central 6-mm visual axis (grade 2 DLK). The IOP was 9 mmHg. The densitometry value was 18.4 D. A bacterial smear test showed no positive findings.

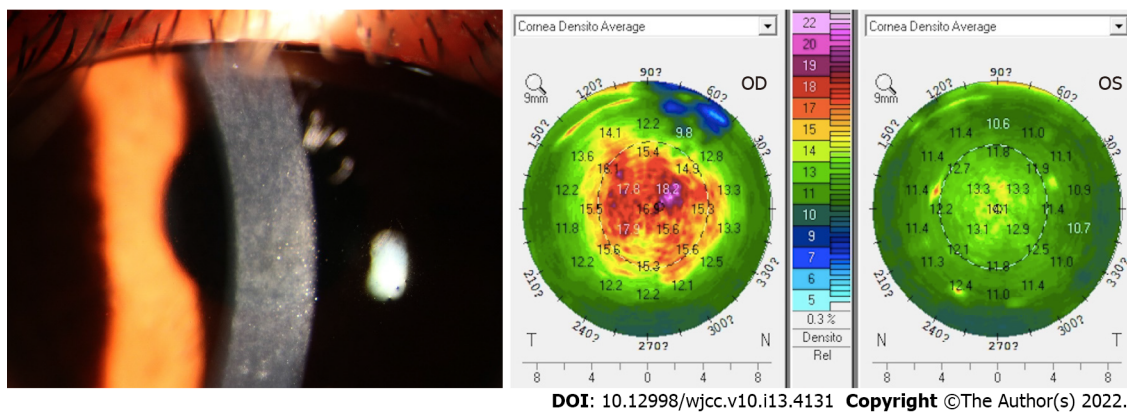
## OUTCOME AND FOLLOW-UP

Two months later, the patient reported a subjective improvement in VA; the UDVA was 16/20, and the DLK severity was grade 1. The average optical density was 19.6 D, and the IOP was 8.7 mmHg. Five months later, the UDVA was 16/20 and the DLK had completely disappeared (Figure 3). The densitometry value (12.6 D) was similar to that before the operation, 12 D. The patient was diagnosed as showing IgA nephropathy due to "interstitial hematuria for 4 years" at 6 mo after SMILE. Biochemical examination showed a serum albumin level of 30.0 g/L (reference value: 35-55 g/L). Routine urine examination showed the following findings: Urine albumin (+++) and occult blood (+++). Protein electrophoresis yielded the following findings: Albumin, 55.1% (reference value: 53.8%-66.1%);  $\alpha$ 1 globulin, 5% (reference value: 1.1%-3.7%);  $\alpha$ 2 globulin, 14.3% (reference value: 3.2%-6.5%). Renal biopsy showed aortic arteriosclerosis; IgM (++); IgA (++); C3d (+++); and C4d (+). The patient was eventually diagnosed as having postoperative SMILE OU, delayed DLK with myopia OD, and IgA nephropathy.

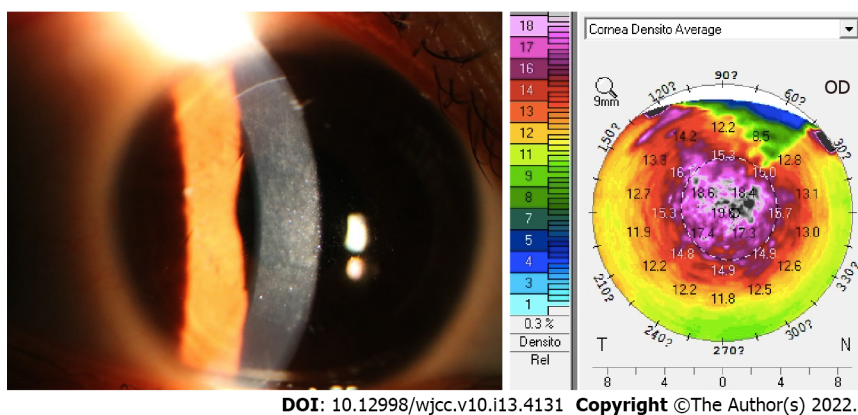
## DISCUSSION

DLK rarely occurs in SMILE surgery. The incidences of DLK after microkeratome laser-assisted *in situ* keratomileusis (LASIK), femtosecond laser-assisted LASIK, and SMILE range from 0.1% to 12.1% [1,2], 0.4% to 37.5% [3,4], and 0.2%-0.45% [5,6], respectively. The 2-4 mm corneal stroma incision in SMILE may reduce the risk of introduction of various foreign bodies into the corneal interface in comparison with that associated with the 20-mm corneal stroma incision in LASIK. However, SMILE may also have specific features that may lead to DLK: (1) The femtosecond laser bubbles increase antigen composition and space to stimulate inflammation [1]; (2) The excessive manipulation in thinner lenticules and the larger diameter may stimulate the secretion of inflammatory factors and increase the incidence of DLK [7]; (3) The higher laser energy may cause severe corneal stroma damage [8]; (4) The repeated use of negative pressure suction during surgery may cause epithelial damage leading to DLK; (5) Other agents such as meibomian gland secretions [9,10], oil, irrigating fluid, talc powder, traumatic flap dislocation, air dust, bacteria, and other stimulating inflammatory factors may be associated with DLK [11,12]; and (6) There are individual patient differences in sensitivity to lasers.

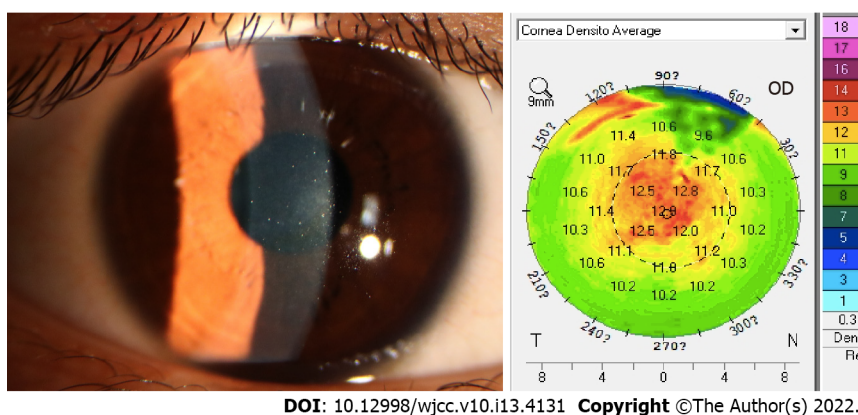
DLK usually occurs within 24 h and rarely appears 5 d after SMILE [6]. In this case, the surgical procedure was performed smoothly, and the patient's vision improved at 1 mo after the procedure, consistent with the findings reported by Lin *et al* [13]. DLK is highly sensitive to steroid therapy, and the majority of cases can be managed with aggressive topical steroid treatment [14,15]. Therefore, steroid therapy and topical steroid treatment are routinely administered. Previous data showed that interface washout may increase the risk of corneal melting associated with focal infiltrates [16]. Therefore, in our case, interface washout was eventually performed after 2 mo, not 1 mo, of hormone therapy. Thus, once topical steroid therapy showed no efficacy, interface washout should be performed.



**Figure 1** Slit-lamp photograph examination of grade 2 diffuse lamellar keratitis OD and Pentcam image of cornea density average OU at 1 mo after small-incision lenticule extraction.



**Figure 2** Slit-lamp photograph examination of grade 4 diffuse lamellar keratitis and Pentcam image of cornea density average OD at 2 mo after small-incision lenticule extraction.



**Figure 3** Slit-lamp photograph examination of grade 0 diffuse lamellar keratitis and Pentcam image of cornea density average OD at 6 mo after small-incision lenticule extraction.

In this case, DLK was delayed and lasted for more than 4 mo. In addition to treatment-related factors, the patient's systemic factors may have been responsible for the atypical presentation. The patient had been diagnosed with IgA nephropathy due to "interstitial hematuria for 4 years" at 6 mo after SMILE. IgA nephropathy is a form of glomerulonephritis characterized by deposition of the IgA antibody in the glomerular mesangial area, and the typical clinical presentation is gross hematuria. The specific etiology and mechanism of this condition are complex and include immune complex deposition, mucosal immunodeficiency, IgA molecular abnormalities, and microbial factors. Ocular involvement in patients with IgA nephropathy is rare; however, the most frequent association occurs with scleritis, episcleritis

keratoconjunctivitis, uveitis, and retinal vasculopathy[17]. To our knowledge, there is no study that directly shows delayed DLK after SMILE and IgA nephropathy. Tears contain a large number of SIgA and small amounts of IgA, and tear SIgA and serum IgA have the common antigenicity. IgA in tears is 3-8 times than that in the blood. IgA abnormalities in plasma affected by abnormal blood may result in sIgA and IgM abnormalities in tears to ocular corneal and conjunctival inflammation[18]. Unfortunately, we did not detect the IgA in tears and interface washing fluid. Li *et al*[19] reported a DLK occurring 4 years after SMILE because of trauma and it had cured with intensive topical corticosteroid eyedrops. As we know, IgA nephropathy needs a sufficient dose of glucocorticoid therapy, but in our case, glucocorticoid was too little to get curative effect and ocular surface drugs cannot reach the flap interface in early time. Until 2 years later, this patient still received a maintenance dose of glucocorticoid.

In addition to these factors, plasma albumin, complement, and inflammatory factors may be related to the presentation of DLK. Corneal nutrition is derived from aqueous humor, tears, and the limbal sclera, and laser postoperative healing may result in inflammatory cell-mediated interference *via* multiple antibiotic factors. Changes in the albumin, complement, and inflammatory factor content in the plasma can cause changes in the lymphocytes, plasma cells, complement, and inflammatory overexpression in the corneal epithelium, tears, and scleral edge as well, directly or indirectly triggering an indirect immune response to the corneal gland and resulting in DLK. It has been proven that the aggregation of inflammatory cells along with interface debris presence were detected by corneal confocal microscopy in DLK patient's cornea after SMILE[20].

## CONCLUSION

Autoimmune diseases are relative contraindications to SMILE. The hidden onset, the patient's lack of consciousness, and the absence of preoperative urine and kidney function examinations as routine tests led to a missed diagnosis. The patient had a 4-year history of hematuria, so her kidney disease was not controlled when she underwent surgery. Despite the long treatment process, she had good treatment and achieved satisfactory results in this special case. This case indicates that IgA nephropathy in patients may be one of the factors related to delayed DLK in patient after SMILE. Moreover, the preoperative history cannot be ignored, especially a history of autoimmune diseases.

## FOOTNOTES

**Author contributions:** Dan TT, Liu TX, Liao YL and Li ZZ collected the clinical information of the patient, and analyzed and interpreted the clinical data; Dan TT wrote the draft of this manuscript; Liu TX reviewed and edited the manuscript; all authors read and approved the final manuscript.

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