

SANDS of SAHARA: Post-laser *in situ* keratomileusis late-onset diffuse lamellar keratitis following sports injury



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ABSTRACT

A 27-year-old male presented with decreased vision and pain in his left eye after a boxing injury, 1 year post-LASIK. Examination showed a partially dislocated LASIK flap, leading to diffuse lamellar keratitis (DLK). Surgical realignment and treatment with topical steroids restored his visual acuity to 20/20 within 3 weeks. This case emphasizes the need for prompt intervention and long-term post-operative care for managing late-onset DLK following high-velocity blunt ocular trauma.

Key words: Lasik; Flap dislocation; Diffuse lamellar keratitis; Sports injury

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INTRODUCTION

The “Sands of Sahara” syndrome, also known as diffuse lamellar keratitis (DLK), is a rare, non-infectious inflammatory complication following laser *in situ* keratomileusis (LASIK) surgery. Its etiological factors remain largely unidentified, and it typically emerges during the early post-operative phase.¹ While some research has documented the occurrence of late-onset DLK after LASIK, these instances are often attributed to specific causes, such as trauma or epithelial defects.²⁻⁴ Notably, epithelial defects in the LASIK flap are significantly correlated with DLK, elevating its incidence from 2% to 56%.^{5,6} We present a case of DLK precipitated by traumatic partial flap dislocation 1 year post-LASIK.

CASE PRESENTATION

A 27-year-old male presented to the emergency department with a sudden decrease in vision and acute pain in his left eye, following trauma while boxing 1 h back. He had undergone uneventful bilateral myopic LASIK surgery a year prior and was not on any topical or systemic medications. Examination revealed an uncorrected visual acuity of 20/20 in the right eye and 20/200 in the left eye. Slit-lamp examination of the left eye showed diffuse conjunctival congestion and a partial dislocation of the temporal aspect of the nasally hinged flap (Figure 1), while the right eye remained unaffected. The temporal one-third of the stromal bed was exposed, with folding of the temporal portion of the flap under the central, non-displaced portion. Under an operating microscope,

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the infolded temporal flap segment was carefully separated from the stromal bed using a cannula. Sterile sponges were employed to realign the temporal flap and eliminate any adherent epithelial cells from the undersurface of the flap and stromal bed. The flap was repositioned on the stromal surface using the cannula, followed by extensive irrigation using a balanced salt solution beneath it. A bandage contact lens was applied, and a regimen of prednisolone acetate 1% and moxifloxacin 0.5% was initiated 4 times daily. By the first post-operative day, the left eye's visual acuity improved to 20/40, and while the flap edges were well-aligned, a typical "Sands of Sahara" diffuse dot-like granular haze became apparent at the interface (Figure 2). Consequently, the frequency of topical steroid application was increased to hourly. By day four post-surgery, the lamellar cellular infiltration showed signs of resolution, and notable visual acuity recovery in the patient's left eye, achieving 20/20. Prednisolone acetate was gradually tapered and moxifloxacin was discontinued. After 3 weeks, the patient regained uncorrected 20/20 visual acuity and prednisolone was discontinued.

DISCUSSION

LASIK flap dislocations occur in approximately 1–2% of cases, often within 24 h post-surgery, typically due to eye rubbing.⁷ In contrast, late flap dislocations, occurring

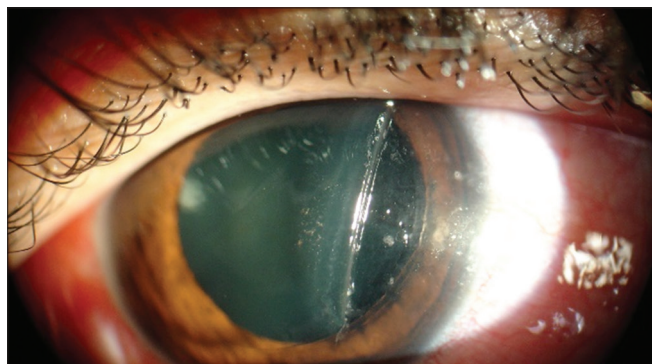


Figure 1: Dislocated LASIK flap in left eye



Figure 2: "Sands of Sahara" post-flap repositioning

more than a week post-surgery, are less prevalent and often linked to high-velocity blunt traumas or minor ocular injuries.⁸ DLK affects roughly 0.2–1.8% of LASIK patients, usually manifesting within the first post-operative week, and presents as granular whitish deposits beneath the flap, earning the "Sands of Sahara" nomenclature.^{7,9} Triggers for DLK include infections, endotoxins, eyelid secretions, talc from gloves, and sponge debris.¹ The majority of late-onset DLK cases, reported between 1 and 12 months post-operatively, were associated with traumatic or spontaneous epithelial defects. Some research suggests that epithelial injuries can alter corneal metabolism and oxygenation, facilitate inflammatory mediator diffusion from the tear film, or modify limbal vasculature permeability to inflammatory cells.^{10,11} Reports of late-onset flap dislocations and lamellar inflammation suggest inadequate adhesion between the flap and stromal bed weeks or months post-LASIK. Prompt and aggressive DLK treatment with topical steroids usually yields favorable visual outcomes with minimal sequelae.² Investigations into wound healing after LASIK, using immunohistochemical methods to detect wound-healing glycoproteins, tenascin, and cellular fibronectin, have shown that stromal remodeling occurs mainly at the flap edge.¹² Hence, appropriately positioned shearing forces may cause partial flap dislocation months post-surgery if strong enough to exceed the flap edge's lamellar cohesive forces.

CONCLUSION

This case highlights the potential for recreational activities to introduce such forces, underscoring the need for caution regarding traumatic flap dislocations due to high-velocity blunt ocular trauma or particular shearing injuries. It is crucial to inform LASIK candidates about this risk and emphasize long-term post-operative follow-up as these late complications can be resolved adequately if diagnosed in a timely fashion.

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