



Refractive Surgical Case Report

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Case Report

A 32-year-old female deemed a good LASIK candidate experiences loose epithelium during her bilateral LASIK procedure. Past medical history was positive for ADHD and her past ophthalmic history was unremarkable. She currently takes Vyvanse for ADHD with NKDA.

Acuvue Oasys 8.4 bandage contacts (BCLs) were placed on each eye immediately following her surgery.

Day-1 Postoperative Visit:

Visual Acuity 20/30 OD and OS with mild diffuse lamellar keratitis (DLK) noted. BCLs were left in place to protect the loose epithelial cells. Prednisolone/Gatifloxacin drops were instilled every 2 hours along with hourly instillation of artificial tears.

Day-3 Postoperative Visit:

Visual Acuity 20/30 OD and OS with grade 2+ diffuse DLK noted. The treating surgeon was promptly notified, and the patient was scheduled for a bilateral interface irrigation.

The interface irrigation resulted in clearance of DLK inflammation, but the patient still had persistent loose epithelial cells OD and OS. A new Acuvue Oasys 8.4 BCL was placed on each eye. The patient was started on Durezol QID and Besivance QID OD and OS with hourly preservative free artificial tear usage. The combination medication containing Prednisolone/Gatifloxacin was discontinued.

Day-4 Postoperative Visit:

Visual Acuity 20/25 OD and OS with negative DLK noted. BCLs were left in place with no changes in medications.

Day-7 Postoperative Visit:

Visual Acuity 20/25 OD and OS with negative DLK noted. BCLs were removed. NaFl dye revealed small epithelial defects with loose epithelial cells. New BCLs were placed on each eye with a decrease in Durezol usage to BID and Besivance to TID OD and OS.

Day-11 Postoperative Visit:

Visual Acuity 20/20⁻³ OD and 20/25 with negative DLK noted. BCLs were removed. NaFl dye revealed negative epithelial defects. Durezol and Besivance medications were discontinued. The patient was instructed to instill sodium chloride 5% drops QID, sodium chloride ointment qhs, hourly preservative free tear instillation, and Restasis BID OD and OS.

Impression:

This patient developed bilateral postoperative DLK as a direct result of her corneal epithelial defects. Subclinical corneal anterior basement membrane dystrophy can often present itself during a LASIK procedure during flap creation and manipulation.

Disease Entity

Background

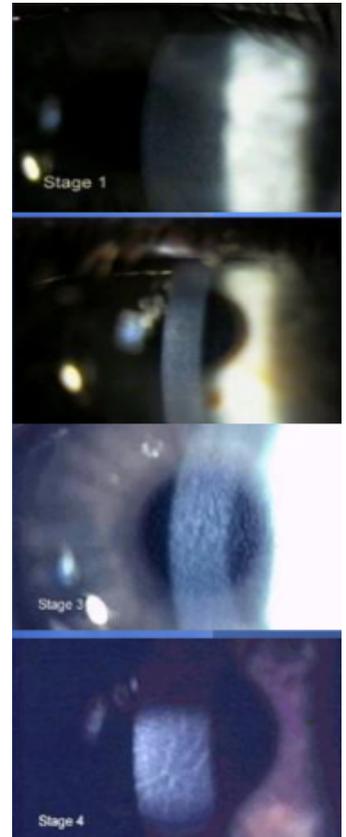
Diffuse lamellar keratitis (DLK) describes a rare, noninfectious complication of refractive surgery that is characterized by inflammatory infiltrates beneath the corneal flap interface. The characteristic accumulation of fine white infiltrates manifests clinically as a grainy corneal opacification that has led some to describe the condition as “sands of Sahara syndrome”.

Clinical Presentation

The characteristic clinical presentation in DLK develops 1 to 2 days following refractive surgery and typically resolves 5-8 days after the initiation of appropriate therapy. The post-surgical corneal opacification that accompanies DLK is typically nonlocalized and subepithelial.

DLK is divided into four stages according to the extent of corneal involvement.

- Stage 1 typically arises 1-2 days after refractive surgery. It is characterized by peripheral inflammatory infiltrates without central corneal involvement.
- Stage 2 typically arises on postoperative days 3-4 when inflammatory cells begin migrating from the periphery into the central cornea often comprising visual acuity.
- Stage 3 is characterized by further centripetal migration of inflammatory cells and the development of permanent corneal scarring. Stage 3 is often referred to as the “threshold” DLK subtype because of the likelihood that eyes in this stage of DLK will develop permanent scarring and resultant loss of visual acuity.
- Stage 4 describes the phase in which stromal melting and further corneal scarring occur. The significant epithelial destruction that ensues during this phase often results in a hyperopic shift.



Etiology

The exact pathophysiology of DLK remains uncertain. The accumulation of inflammatory infiltrates within the areas of surgical manipulation has led some investigators to postulate that manipulation of the corneal surface may instigate the inflammatory changes that characterize DLK. In fact, the presence of an epithelial defect following a LASIK procedure places the patient at a 24-times greater risk of developing DLK when compared to normal controls.

Intraoperative exposure to inflammatory instigators such as red blood cells, fine sponge fibers, or meibomian gland secretions have also been suggested as playing a role in disrupting corneal epithelial cells during the development of DLK.

Epidemiology

DLK affects between 2-4% of patients undergoing LASIK surgery.

Symptoms

Patients with DLK often exhibit pain, foreign body sensation, photophobia, and blurry vision.

Differential Diagnosis

- Central Toxic Keratopathy
- Infectious keratitis
- Interface fluid
- Corneal haze secondary to increased IOP
- Superficial punctuate keratitis
- Epithelial ingrowth
- Trauma

Management

Given its inflammatory nature, all stages of DLK typically respond well to corticosteroid therapy. In certain cases, irrigation beneath the flap followed by flap repositioning can remove subepithelial infiltrates and minimize complications associated with the condition.

Outcome

This patient ended up achieving a 20/20 uncorrected visual acuity OD and OS. Prompt recognition and treatment of DLK is important to secure an optimal postoperative visual acuity. Prolonged DLK can result in a hyperopic shift, corneal scarring, or possibly even melting of the superficial LASIK flap.

Important note that ADHD medications often result in dry eye disease. These patients require dry eye treatments to aid their ability to achieve comfort with contact lens wear and minimize their postoperative LASIK dryness.

