Late-onset diffuse lamellar keratitis after blunt trauma without epithelial or flap damage

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A 29-year-old man presented to our clinic with pain, decreased vision, and hyphema in his left eye after being hit by a football. Laser in situ keratomileusis had been performed 6 years earlier. No damage was noted on the epithelium or flap structure. The intraocular pressure was 22 mm Hg. Stage III to IV diffuse lamellar keratitis (DLK) developed gradually over the next 4 days. Intensive topical steroid treatment had no effect on the DLK, and interface irrigation was performed on the sixth day. The corneal edema and DLK resolved following the irrigation.

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Diffuse lamellar keratitis (DLK) is a known complication of laser in situ keratomileusis (LASIK) surgery. It causes the formation of diffuse granular or powdery inflammatory haze at the level of the interface, which resembles waves of sand when viewed through the slitlamp. Hence, it is referred to as “sands of Sahara.” Although the exact cause of DLK is not known, its etiology is generally considered to be multifactorial.

Diffuse lamellar keratitis is marked by the presence of diffuse or multifocal neutrophilic infiltrates confined to the LASIK interface. The infiltrates are culture negative, and the etiology is thought to be noninfectious. Most cases of DLK occur within the first week or 2 following surgery. However, there are also reports of late DLK up to 12 years after LASIK. In some cases, DLK developed spontaneously; in some, it was associated with traumatic flap displacement; and in some, it was due to corneal epithelial damage. Late DLK development has also been reported in association with viral keratoconjunctivitis.

CASE REPORT

A 29-year-old man presented with pain and decreased vision in his left eye after being hit by a football. He had no complications after LASIK surgery 6 years earlier. The uncorrected distance visual acuity (UDVA) in the left eye was hand motions (HM), and the intraocular pressure (IOP) by applanation tonometry was 22 mm Hg. Biomicroscopic examination showed a 3.0 mm hyphema, red blood cells (RBCs) in the anterior chamber, and some corneal edema (Figure 1). No damage to the epithelium or the flap structure was noted. Intensive topical steroid treatment had no effect on the DLK, and interface irrigation was performed on the sixth day. The corneal edema and DLK resolved following the irrigation.

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Figure 1. Corneal edema, RBCs in the anterior chamber, and a 3.0 mm hyphema after blunt trauma.
flap structure was detected. The central corneal thickness (CCT) was 572 μm, and a fine layer of inflammatory opacities was noted on the Scheimpflug images. The patient was started on topical antibiotics, corticosteroids, and a cycloplegic agent every 6 hours.

Four days after presentation, stage III to IV DLK with a CCT of 611 μm was noted in the left eye (Figure 2, A). Significant inflammatory granular opacities were noted at the flap interface on the Scheimpflug images (Figure 2, B). No fluid was present at the flap interface. Topical corticosteroid treatment was increased to hourly drops with no significant improvement.

Two days later, the LASIK flap was irrigated and the corneal edema and DLK resolved. Mild folds were still visible in the flap after the irrigation (Figure 3). On day 54, the CCT had decreased from 611 μm to 446 μm. There were no opacities at the flap interface (Figure 4, A and B), and the UDVA had improved from HM to Snellen 0.8.

**DISCUSSION**

In the differential diagnosis of stromal opacification after trauma that occurs long after LASIK surgery, the 3 most important diagnoses to be considered are infectious keratitis, DLK, and more rarely, interface fluid syndrome. In our case, after blunt trauma there were no visible epithelial defects but there were RBCs and an inflammatory reaction in the anterior chamber; therefore, we did not consider infection as a cause of the inflammation.

We considered interface fluid syndrome, which is caused by fluid accumulation in the interlamellar space secondary to traumatic or steroid-induced IOP elevation and results in opacification of the flap interface and corneal edema. Interface fluid syndrome can be seen late after LASIK, with a misleading clinical picture that simulates DLK or infectious keratitis. Interface fluid syndrome was excluded as the diagnosis because the Scheimpflug images on day 6 (Figure 2, B) showed inflammation but no fluid accumulation in the interface.

Clinically, our case presented more like DLK, which usually occurs when foreign substances, including endotoxins, are introduced into the interface between the corneal flap and underlying stroma. However, to our knowledge, an epithelial defect or trauma to the flap...
epithelium is usually responsible for late-onset DLK and we did not observe either in this case.

In the treatment of this case of late-onset DLK following blunt trauma, rapid improvement in the patient’s symptoms, visual acuity, and slitlamp biomicroscopic findings occurred only after flap irrigation.

Diffuse lamellar keratitis can occur as a result of blunt trauma to the eye with no visible structural flap or epithelial defect as late as 5 years after LASIK. Scheimpflug imaging is complementary to slitlamp examination in the clinical observation of DLK. Interface irrigation can provide significant improvement in vision.

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