To our knowledge, orbital myositis has only once been previously reported. In conclusion, we present a case of superior rectus muscle myositis due to CSS. The concurrent association with an eosinophilic conjunctival nodule further illustrates the heterogeneity of the ocular manifestations of CSS.

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Relapsing Diffuse Lamellar Keratitis After Laser In Situ Keratomileusis Associated With Recurrent Erosion Syndrome

Diffuse lamellar keratitis (DLK) is a well-described complication of laser in situ keratomileusis (LASIK) that generally occurs within the first week after surgery. Late-onset cases of DLK have been reported to occur many months after surgery and are sometimes associated with recurrent erosions.1,3 We describe 3 patients who had intraoperative epithelial defects and who subsequently developed DLK multiple times in the same location of the same eye, always following an episode of recurrent erosion.

Report of Cases. Case 1. A 33-year-old woman underwent bilateral LASIK in May 2001 for high myopia. Preoperative evaluation revealed clear corneas with no evidence of anterior basement membrane dystrophy. The procedure was uneventful in the right eye. In the left eye, however, a 2.0 × 2.0-mm corneal epithelial defect was noted in the superior paracentral location after creation of the flap, and the epithelium surrounding the defect was noted to be generally poorly adherent to the Bowman layer. A bandage soft contact lens was placed on the left eye, and the patient was instructed to use a combination of 0.1% fluorometholone and 0.3% ofloxacin eyedrops, 4 times daily, in both eyes. On the first postoperative day, the patient’s uncorrected visual acuity (UCVA) was 20/70 OS. On ophthalmic examination, the contact lens was in place, and there was mild flap edema, but no epithelial defect or lamellar keratitis was noted. The soft contact lens was removed, and on the next day, the patient manifested acutely with reports of decreased vision, foreign body sensation, photophobia, tearing, and pain in the left eye. Visual acuity was still 20/70 OS, and a recurrent 2.0 × 1.9-mm epithelial defect was found in the same location as when the defect had first been noted. No lamellar keratitis was found, and a bandage soft contact lens was placed on the eye. On the third postoperative day, the patient’s visual acuity was decreased to 20/400 OS, and moderately severe DLK was found. Treatment with 1% prednisolone acetate was instituted every hour around the clock in place of the 0.1% fluorometholone. By 2 days later, the DLK began to recede, and the epithelial defect was healed. Eight months postoperatively, the patient’s best-spectacle-corrected visual acuity (BSCVA) was 20/20 OS.

At this time, the patient underwent an uneventful LASIK re-treatment (with relifting of the original flaps). Five months after the re-treatment, while the patient was away on vacation, she went to an emergency department with reports of decreased vision, foreign body sensation, photophobia, tearing, and pain in the left eye. She was diagnosed as having a corneal abrasion and prescribed 0.3% ofloxacin, 4 times daily. On seeing an ophthalmologist 2 days later, her visual acuity was 20/60 OS, and she had a healing epithelial defect in a location corresponding to that of her previous 2 epithelial defects, as well as interface inflammation consistent with DLK. The patient was instructed to use both 1% prednisolone, every 2 hours, and 0.3% ofloxacin, 4 times daily. Two days later, the patient returned to our care and was found to have responded well to topical corticosteroid therapy in the left eye with only trace DLK noted and with a UCVA of 20/30 OS.

Ten and a half months after retreatment, the patient was seen with reports of discomfort in her left eye for 2 days and had a BSCVA of 20/15 OD and 20/25 OS. Once again, an area of epithelial irregularity was found in the same area of the superi or cornea in which epithelial defects had been previously. An underlying moderate DLK was present in the superior flap interface. Treatment with 1% prednisolone acetate was used every 2 hours initially, with a rapid tapering course. The patient responded well to therapy, and the BSCVA is 20/25 OS.

Case 2. A 54-year-old woman underwent bilateral sequential LASIK in September 2001 for moderate myopia. Because the left eye sustained an intraoperative epithelial defect in the inferotemporal para-central location, a bandage soft contact lens was placed on the eye, and the patient was instructed to use both 0.1% fluorometholone and 0.3% ofloxacin eyedrops, 4 times daily. The next day, UCVA was 20/80 OS. Although there was still a 2.0 × 2.0-mm epithelial defect present, no interface inflammation was noted. The defect was healed by the third postoperative day with a UCVA of 20/20 OS.

Two and a half months later, the patient was seen with a 2-day his-
tory of redness, photophobia, and foreign body sensation in the left eye. The UCVA was still 20/20 OS, but a 2.0 × 2.5-mm epithelial defect was found in the same location as previously noted intraoperatively, and moderately severe DLK was found in the interface (Figure 1). A bandage soft contact lens was placed on the eye, and the patient was instructed to use both 1% prednisolone acetate and 0.3% ofloxacin eye drops, 4 times daily. Clinical improvement was then noted after 2 days, and the frequency of instillation of the corticosteroid eye drops was tapered. The UCVA remained at 20/20 OS.

Sixteen months post-LASIK, the patient returned with a second episode of redness, foreign body sensation, and irritation in the left eye. The UCVA was 20/25 OS. Slitlamp examination revealed a 2.0 × 2.0-mm patch of loose epithelium in the same location of the previous epithelial defects, but no interface inflammation was noted. A bandage soft contact lens was placed, and 0.3% ciprofloxacin eyedrops, 4 times daily, were prescribed. Three days later, although the epithelial defect was almost healed, moderately severe DLK was noted (Figure 2). Treatment with 1% prednisolone eye drops was used hourly with rapid resolution of the inflammation. The patient ultimately elected to proceed with anterior stromal micropuncture in an attempt to treat the recurrent erosions that led to her episodes of DLK. Five months after the micropuncture procedure was performed, the patient’s UCVA remained at 20/20 OS, and she has not had any further episodes of DLK.

Case 3. A 34-year-old man was referred for recurrent corneal erosions in the right eye following bilateral LASIK performed in February 2003. According to the patient, the right eye developed an epithelial defect intraoperatively, for which a bandage soft contact lens was placed for 1 day. No DLK was reported in the immediate postoperative period. Although UCVA recovered to 20/20–1 OU, the 6-month period following the LASIK procedure was punctuated with 3 episodes of recurrent erosion, all documented to have occurred in the same inferior paracentral area of the right cornea. Each episode was treated with lubrication and topical antibiotic eye drops, but not with topical corticosteroids. The patient was referred to our institution during the third episode of recurrent erosion, 4 days after onset of symptoms of blurred vision and irritation of the right eye.

On ophthalmic examination, the UCVA was 20/40 OD and BSCVA was 20/30 OD. In the right eye, a 1.5-mm patch of irregular epithelium was noted in the inferior paracentral cornea, surrounded by a 3-mm area of flap edema, flap haze, and mild interface inflammation with leukocyte clumping. The patient was diagnosed as having recurrent corneal erosion associated with DLK, the latter of which was probably in the early-resolution phase. The right eye was treated with...
nonpreserved artificial tears and 1% prednisolone every 4 hours. One week later, UCVA and BSCVA had improved to 20/20 – 1 OD. The area of previously noted epithelial irregularity was smooth, but in its place were a few intraepithelial cysts, which had an appearance consistent with anterior basement membrane dystrophy. Despite complete resolution of the flap edema and DLK, a mild haze persisted within the flap and the LASIK interface.

Comment. While most cases of DLK occur within the first postoperative week, an increasing number of cases have been reported to occur months after surgery. Most of these late cases of DLK follow an event that resulted in disruption of the corneal epithelium.1-3 This association of late DLK with epithelial defects implies the release of inflammatory stimulants from a site other than the interface itself. In these late-onset cases of DLK, cytokines and inflammatory mediators released by the injured epithelium may contribute to the development of DLK through keratocyte injury and chemotaxis of polymorphonuclear cells.4 These cytokines can also be released during flap formation, causing early-onset DLK.5 Thus, irritants intrinsic to the flap formation, causing early-onset toxines can also be released during early-onset DLK.6

Intraoperative epithelial defects have been associated with 38.9% of the early-onset DLK cases in 1 series.7 Chang-Godinich et al1 also described a woman who had recurrent erosions who suffered DLK associated with an intraoperative epithelial defect, and who then had a second episode of DLK in the same eye 2 months postoperatively. Our first patient also had recurrent erosions and DLK associated with an intraoperative epithelial defect, but our case was unique because she developed 2 separate episodes of late-onset erosion-associated DLK. Our second and third patients also developed separate episodes of late-onset erosion-associated DLK, but without the initial episode of early-onset DLK despite the initial intraoperative epithelial defect. The fellow eye of the woman described by the patient also had recurrent erosion-associated DLK even in the absence of perioperative DLK.

Based on our article, it appears that recurrent DLK can occur not only in patients with overt preoperative evidence of anterior basement membrane dystrophy or in patients with a known history of recurrent erosion, as described previously, but also in asymptomatic patients who develop an intraoperative epithelial defect attributable to a suspected underlying subclinical anterior basement membrane dystrophy. Furthermore, these recurrent episodes of DLK can occur many months following the primary LASIK or re-treatment procedure, even when no DLK was documented during the initial perioperative period. Given that intraoperative epithelial defects occur in approximately 3% to 9.7% of LASIK cases,7,8 the number of patients at risk for late-onset erosion-associated DLK could be high. Thus, consideration should be given to advising potentially at-risk patients who have undergone LASIK (eg, patients with intraoperative epithelial defects) of the need to seek consultation from an ophthalmologist should symptoms of recurrent erosion occur. Treatment in such circumstances should not only address the recurrent erosion, but also should include the use of topical corticosteroids to prevent or treat associated DLK. Furthermore, primary eye care providers and emergency physicians should also become familiar with this potential complication. This subgroup of post-LASIK patients may initially have signs and symptoms of acute erosion and DLK-related inflammation that can readily be mistaken for a case of corneal abrasion or infectious keratitis. The clinical presentation of diffuse interface inflammation in the setting of mild epithelial disruption may be particularly confusing if a prior history of LASIK is not elicited. In patients 1 and 2, prompt and appropriate treatment of each episode of recurrent DLK with frequent administration of topical corticosteroids resulted in good outcomes, with no adverse effects on final visual acuity. However, DLK that is severe or that is not treated aggressively, as in patient 3, may result in a poorer outcome due to complications such as stromal haze, scarring, epithelial ingrowth, and irregular astigmatism.

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