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Refractive change and photorefractive keratectomy treatment for a late traumatic flap loss

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A flap loss is rare and serious among various flap-related complications of LASIK. It can occur intra-operatively, early post-operatively or late post-operatively and can be classified aetiologically as iatrogenic or traumatic. Here, we report a case of late traumatic flap loss caused by a scratch from a cat's paw and successfully treated by PRK. A review of the literature indicates that this is the first case of late traumatic flap loss, for which the patient received a PRK re-treatment. With the increasing popularity of LASIK, the incidence of either iatrogenic or traumatic flap loss may increase and the awareness of this complication and the importance of its management should not be overlooked.

CASE REPORT

A 28-year-old woman underwent bilateral wavefront LASIK on 17 February 2007. The preoperative refractions were -6.00/-2.25 × 170 in the right eye and -8.00/-2.50 × 165 in the left eye for a visual acuity of 6/6 in both eyes. The keratometric values were 43.25@165/45.5@75 and 43.25@165/45.25@75 in the right and left eyes, respectively. The preoperative pachymetric readings were 562 μm for the right eye and 559 μm for the left eye. An automated microkeratome (Moria M2, Moria, Anthony, France) was used to create the flap with a superior hinge, followed by ablation with an excimer laser (VISX STAR S4 Inc, Santa Clara, CA) with the CustomVue software (VISX Inc, Santa Clara, CA). The attempted correction was -6.11/-1.94 × 169 in the right eye and -8.15/-1.98 × 165 in the left eye. The diameters of ablation zone were 8.0 mm in both eyes and the calculated ablation depth was 119 μm in the right eye and 151 μm in the left eye. The surgical course was uneventful. Six months after LASIK, her unaided vision was 6/6 in both eyes.

On 20 January 2008, 11 months after LASIK, the left eye of this patient was scratched by a cat's paw, while she was playing with the cat in a pet store. The patient suffered from severe ocular pain and she noted a sudden decrease in visual acuity. She was sent to an emergency service, where a LASIK flap loss was diagnosed. A bandage contact lens was inserted with topical medications.

Two days later, she came to our clinic. On examination, the unaided vision of the left eye was 6/150 and the VA was 6/15 with a manifest refraction of -4.50/-1.25 × 75. A central corneal epithelial defect was found; it was 8 × 8 mm in size and corresponded to the area of the previous LASIK flap (Figure 1). Topical 0.3% ciprofloxacin hydrochloride (Alcon Laboratories Inc, Fort Worth, TX), 0.1% fluorometholone (Alcon Laboratories Inc, Fort Worth, TX) and artificial tears were prescribed with a bandage contact lens for the affected eye. Four days after the flap loss, the epithelial defect healed and a moderate corneal haze presenting at the nasal and temporal sides remained, however, it spared the central visual axis (Figure 2). Three months after the flap loss, the unaided vision of the left eye was 6/18 and the VA was 6/9 with a refraction of -1.50/-0.75 × 68. The topography showed an irregular astigmatism at that time (Figure 3).

Eight months after the flap loss, the pachymetry was 425 μm for the right eye and 335 μm for the left eye. The unaided vision of the left eye was 6/30 and the VA was 6/7.5 (-1.00/-1.25 × 75). The keratometric reading of the right eye was 37.50@170/38.75@80, and 39.00@20/40.25@110 for the left eye. Topography revealed a decreased irregular astigmatism. Her vision still felt blurry without spectacles and she had a sensation of glare, which

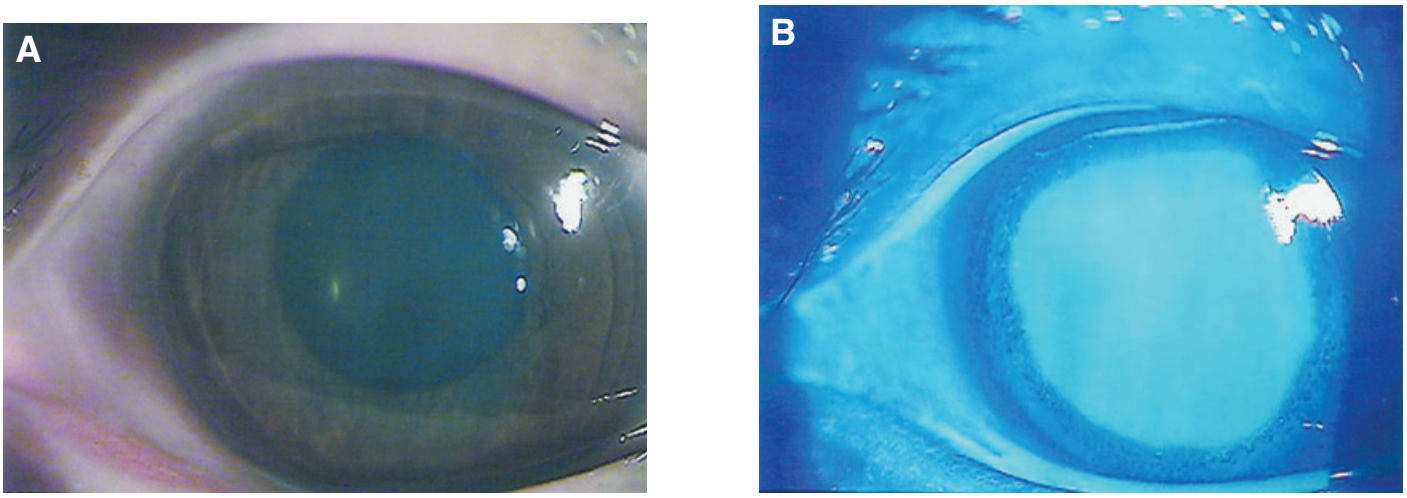

Figure 1. Photographs of LASIK flap loss at day 3. Figure 1A shows the clear cornea without a LASIK flap; Figure 1B shows a central staining by fluorescein in corneal epithelial defect corresponding to the area of the flap loss.

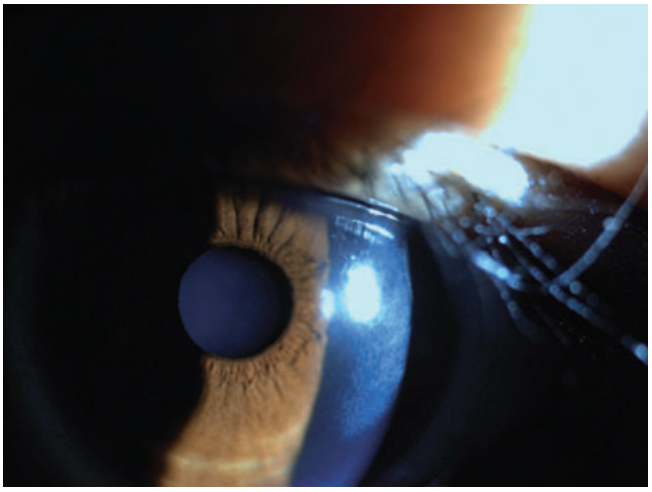

Figure 2. Photograph of the left eye eight months after flap loss. A mild corneal haze was located at the temporal side with sparing of the central visual axis.

deteriorated at night. PRK was suggested and performed with the same excimer laser. The attempted correction was -1.50/-0.65 × 55 with the ablation zone diameter 6.0 mm. Subsequent to laser ablation, intra-operative topical 0.02% mitomycin-C (Kyowahakk O Kogyo Co Ltd, Shizuoka, Japan) was used for one minute to reduce the corneal haze. The calculated ablation depth was 19 µm. Post-operatively, a bandage contact lens was inserted and 1% prednisolone acetate (Alcon Laboratories Inc, Fort Worth, TX) and 0.5% moxifloxacin hydrochloride (Alcon Laboratories Inc, Fort Worth, TX) were administered four times per day for one week. The steroids were tapered to twice per day in the second month and once per day in the third month. Eight months after PRK, her unaided vision was 6/7.5 and the VA was 6/6 with -0.25/-0.75 × 40. She felt satisfied with the visual outcome. The ultrasonic pachymetry was 322 µm. The follow-up topography revealed a regular astigmatism without the ectatic pattern (Figure 3).

DISCUSSION

Regarding the refractive change after a flap loss, there are few publications mentioning this issue. The refractive changes of traumatic and iatrogenic flap loss in the literature are summarised in Table 1. Sridhar, Rapuano and Cohen[1] described a case of early post-operative flap loss during removal of a contact lens 10 days after LASIK surgery. VA was 6/21 with -1.5/+0.50 × 167 at that time. No long-term follow-up of visual acuity or refractive change was published in that report. Tetz and colleagues[2] presented a case of late traumatic flap loss caused by a contact sport accident 3.5 years after the initial LASIK surgery. Sixteen weeks after the injury, the VA was 6/12 with -1.75/-0.50 × 30. Only eye-drops were prescribed for their case and no further surgery was performed.

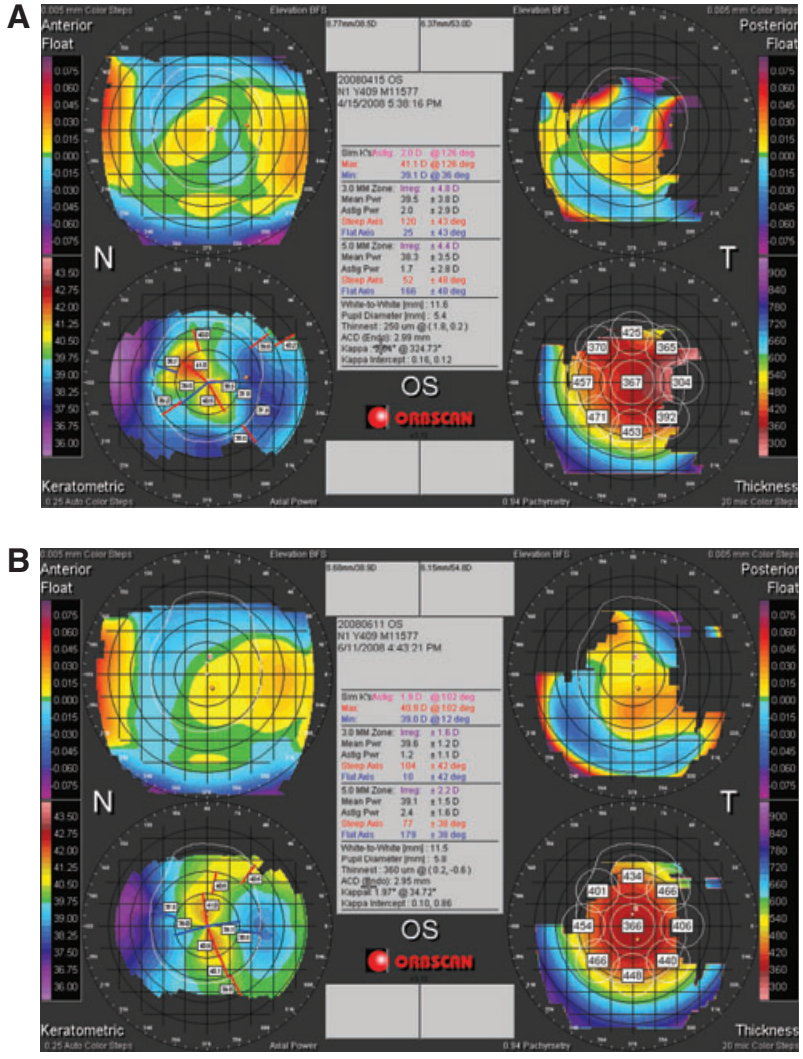

Figure 3. Evolution of the corneal topography of the left eye after the flap loss. Figure 3A shows an irregular astigmatism three months after injury; figure 3B shows the topography eight months after the flap loss. Note the marked reduction of irregular astigmatism.

Epstein and co-workers[3] described six eyes in six patients, whose corneal flaps were removed after LASIK due to intra-operative flap complications. Two eyes had free caps and both received photo-therapeutic keratectomy (PTK) to reduce the surface irregularity and PRK to reduce the refractive errors. Two eyes had buttonholes. One underwent PRK twice, while the other developed an epithelial in-growth and received a PTK to remove the flap, followed by PRK and astigmatic keratotomy. Two eyes of flap removal were due to epithelial in-growths. One was treated with PTK to remove the irregular flap; the other agreed to receive flap removal manually, followed by PTK and PRK. All of these eyes showed a myopic shift with some irregular astigmatism. After treatments, the mean VA gain of the six eyes was 2.2 ± 1.2 lines, compared to the mean value at times of the initial complications. From refractive changes of these cases and our case, we reason that the myopic changes after flap loss or amputation might be attributed to the flap created by the conventional microkeratome, which is thinner in the centre than in the periphery (a minus meniscus flap). This results in a steeper curvature of the exposed stromal bed relative to that of the flap surface. When the stromal bed is covered by the flap, this effect is masked, however, after a flap loss, the exposed steeper curvature results in a myopic change. This assumption is also supported by comparison of keratometric readings of the right and left eyes in our case, before and after flap loss. Before flap loss, the two eyes had similar keratometric values; however, after flap loss the corneal curvature of the left eye became steeper than in the unaffected eye.

Another possible contributing factor to the myopic change is epithelial hyperplasia. The relationship between epithelial hyperplasia and myopic regression after PRK has been widely suggested. It has been postulated that smaller ablation zones are associated with the development of myopic regression. In smaller ablation zones, the slope of the wound is steeper than in larger ablation zones. Clinically, regression is less common in eyes treated with 6.0 mm zones or larger.[4,5] In our case, the diameter of the ablation zone in the previous LASIK was 8.0 mm. But we still cannot exclude this possibility since we did not know the detailed wound healing process after surface photoablation, in which the epithelial thickness of the central and mid-peripheral cornea after flap loss should be measured and verified to confirm this process.

After flap loss, the corneal surface would be re-epithelialised (similar to the healing process after PRK) and theoretically, the irregular astigmatism and dysphotopsia should be minimal in the absence of significant scarring. Abbas and Hersh[6] found that, after PRK, corneal smoothing occurs in three to 12 months, as a result of stromal healing and remodelling. The irregular astigmatism compromises the visual rehabilitation after flap loss. This finding can be attributed to the course of flap creation. The flap thickness might not be uniform, which results in an

	Time after injury	Refraction
Sridhar et al[1]	10 days	-1.5 + 0.75 × 167
Tetz et al[2]	16 weeks	-1.75 - 0.50 × 30
Epstein et al[3]	Case 1, 6.5 months	+3.75 - 4.00 × 150
	Case 2, 4 months	-2.00 - 4.00 × 75
	Case 3, 7 months	-8.75 - 0.50 × 165
	Case 4, 6 months	-1.50 - 1.50 × 100
	Case 5, 14 days	-2.50 - 4.25 × 5
	Case 6, 4 months	-5.50 - 1.50 × 65
Eggink et al[12]	Case 1, 6 months	0 - 5.00 × 22
	Case 2, 6 months	-2.50 - 0.75 × 75
	Case 3, 6 months	-0.50 - 2.00 × 15
McLeod et al[13]	9 months after flap amputation	-1.50 + 0.25 × 171
	18 months after flap amputation	-3.75 + 3.75 × 97
Our case	11 months	-1.00 - 1.25 × 75

Table 1. Summary of the refractive changes after flap loss or amputation reported in the literature

uneven stromal surface. After reapposition of the flap, the rugged stromal surface might be masked by the 'lock and key effect'. Once the flap is removed the uneven stromal surface is exposed and irregular light reflection is observed. In our case, we found that the corneal surface became smoother and irregular astigmatism declined, as documented during monthly topographic examinations. Nine months after the flap loss, at which time the corneal surface had stabilised, we performed PRK at the patient's request. As the corneal haze did not block the pupil entrance, the ultimate visual outcome was better than those in other reported cases.

Majmudar and associates[7] described the first human series of eyes to receive treatment with mitomycin-C for post-radial keratotomy and PRK corneal haze. Carones and colleagues[8] confirmed that the prophylactic use of mitomycin-C may be safe and effective in preventing haze in high myopia after PRK. In contrast, Nassiri and co-workers[9] reported higher endothelial cell loss in eyes that received intraoperative topical mitomycin-C and the rate of cell loss was correlated with the duration of mitomycin-C exposure. Neither Carones and colleagues[8] nor Nassaralla and co-workers[10] observed a statistically significant difference in the endothelial cell count after application of a single dose of intraoperative mitomycin-C for two minutes. Nevertheless, serious ocular complications after use of mitomycin-C in pterygium surgery have been reported, including corneal oedema, corneal perforation, glaucoma and scleral thinning.[11] The toxicity can be reduced by shortening exposure time or reducing mitomycin-C concentration. In our case, as the residual stromal was thin, we shortened the exposure time of 0.02% mitomycin-C to one minute to prevent these possible toxicities. During application, mitomycin-C was confined to the central avascular cornea and contact with the limbal area was avoided. After application, copious irrigation with balanced salt solution was performed to remove residual mitomycin-C.

According to our experience, when there is sufficient residual stromal thickness, PRK should be considered as an alternative procedure for improvement of unaided vision during rehabilitation after traumatic flap loss; however, the irregular astigmatism produced by the flap loss might limit the improvement of VA. This limitation can be compensated by re-epithelialisation or by PTK to remove the surface irregularity. In our experience, a waiting period of nine months after the flap loss was required to attain stabilisation of the corneal surface before performance of PRK.

DECLARATION

The authors have no financial and propriety interests in any aspects of this study.

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