European Journal of Ophthalmology / Vol. 17 no. 2, 2007 / pp. 259-261

SHORT COMMUNICATION

Ultrabiomicroscopy (UBM) in flap dislocation following LASIK: A case report

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PURPOSE. *To describe postoperative laser-assisted* in situ *keratomileusis (LASIK) flap dislocation occurred after trauma*
METHODS. *Ultrabiomicroscopy (UBM) is used to obtain a high-resolution imaging of the cornea.*
RESULTS. *The UBM results are presented and compared with histologic and confoscan findings.*
CONCLUSIONS. *The technique is useful and easy to perform, offering more opportunities to study the anatomical changes in LASK flap dislocation occurred after trauma. (Eur J Ophthalmol 2007; 17: 259-61)*

KEY WORDS. *LASIK, Flap dislocation, UBM*

Accepted: October 9, 2006

INTRODUCTION

Postoperative laser-assisted in situ keratomileusis (LASIK) flap dislocation is seen in approximately 1 to 2% of patients. This usually occurs within the first day or two after surgery and is due to eye rubbing in most cases (1). Late LASIK flap dislocations, defined as those that occur more than 1 week after surgery, are less common, but have been reported in association with high velocity, blunt ocular trauma as well as seemingly minor ocular injuries (2, 3). Appropriate management of the complications of corneal refractive surgery requires determination of the exact anatomic cause. Surface videokeratography only provides a graphical report of the presence and location of irregularity, not the underlying anatomic basis (4).
Pavlin and coworkers first described the use of a very high-frequency ultrasound system for high-resolution imaging of the cornea (5). Very high-frequency ultrasound enhanced by digital signal processing is able to resolve the epithelial layer from the stroma. It is also capable of detecting the stromal surgical interface despite its near-optical transparency at the slit lamp. The technique is therefore able to resolve flap from the residual stromal bed (5).

We report the case of a patient who sustained a traumatic partial LASIK flap dislocation 10 months after LASIK treatment.

Case report

A 27-year-old man underwent surgery for myopia and astigmatism only in his left eye, 10 months before presentation. In August 2002, he was examined at the ophthalmologic emergency department for trauma to this left eye following a car battery explosion (occurring 1 hour previously). His visual acuity was hand movement. Slit lamp examination revealed partial displacement and edema of the temporal part of the flap exposing the temporal third of the stromal bed, hyphema, and a localized iridodialysis (Fig. 1A). The flap was placed back in position and the patient was put on both systemic and local antibiotic and steroid therapy. Regular control examinations were carried out. At 1 week there was a reduction of the corneal edema and there seemed to be adhesion of the flap to the underlying corneal layer. The hyphema had cleared. There were posterior synechiae with irregularity of the pupillary margin (Fig. 1B).
UBM was carried out at 3 weeks. This showed a well-

 1120-6721/259-03$15.00/0

defined hyper-reflective area at the margin of the corneal hinge. Slight corneal edema, shown as a thin hyper-reflective band at the interface, was also observed. Hyper-reflective echoes were also evidenced in the superficial stroma (Fig. 2). At 2 months, the slight corneal edema had completely cleared, but the corneal margin alterations and those of the stroma persisted (Fig. 3). The uncorrected visual acuity was 6/20.

DISCUSSION

Several reports of late-onset flap dislocations indicate that significant adhesion between the flap and stromal bed had not developed weeks or even months after LASIK. Perez-Santonja and colleagues, in studies on rabbits, found that the normal LASIK procedure induces only minimal or no corneal fibrosis related to the central photoablation (6). The morphology of the keratocytes in the flap was comparable to quiescent keratocytes of unwounded stroma and showed no response to any of the photoablation used. Activated keratocytes are supposed to be the source of new extracellular matrix proteins such as cFgn (fibronectin) after LASIK, but in this case no expression of these proteins at the photoablation area was found. The wound healing process related to scar formation was found only in association with the epithelial plugs at the flap margins, suggesting the importance of the epithelial-stromal interaction for both stromal cell apoptosis and keratocyte activation. At 4 days after LASIK, the epithelial plugs were observed; EDA-cFn (cellular fibronectin) appeared in the periphery of the wound and as a narrow line at the flap interface. The adjacent kerato-

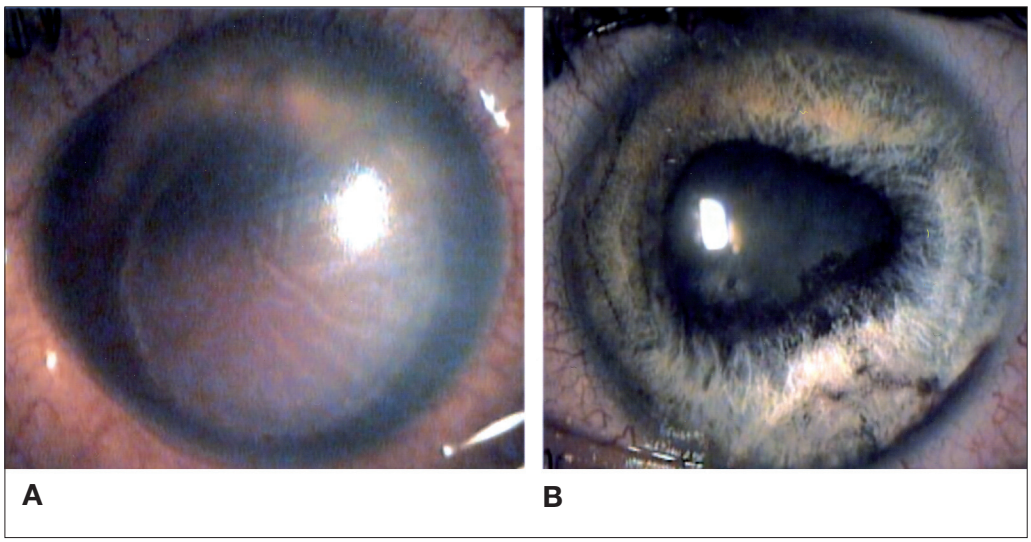

Fig. 1 - A) *Partial displacement and edema of the temporal part of the flap hyphema and a localizes iridodialysis.* **B)** *Posterior synechie with irregularity of the pupillary margin.*

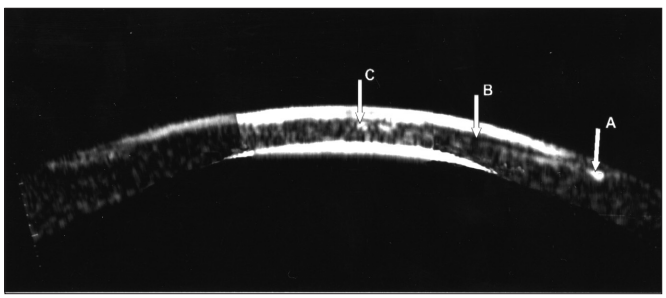

Fig. 2 - *Slight corneal edema* **(B)** *shown as a thin hyper-reflective echoes* **(C)** *were also evidenced in the superficial stroma starting from the margin of the flap* **(A)**.

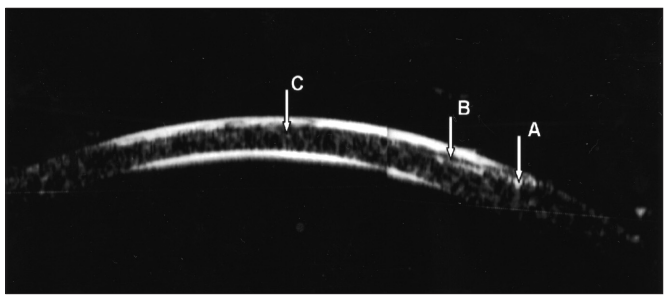

Fig. 3 - *At two months, the corneal edema had cleared, but the corneal margin alterations and those of the stroma persisted. In particolar, the hyper-reflective area at the margin of the flap is evident* **(A)**. *The thin hypo-reflective band at the interface correspond to the residual corneal edema* **(B)** *and the hyper-reflective to the underlying stroma* **(C)**.

cytes did not show any changes in their immunofluorescence for EDA-cFn. Tn (Tinascin) immunoreactivity also appeared in the periphery of the wound, but was not detected at the flap interface. At 5 months after LASIK, EDA-cFn immunoreactivity was occasionally observed as a distinct subepithelial band, probably at the level of the epithelial basement membrane. In the flap, excluding its margins, however, a normal immunoreaction (in keratocytes only) was observed. Immunoreaction for Tn appeared as a fluorescent area at the periphery of the flap, extending 700 to 1000 µm into the wound from the margin of it towards the center of the flap. No Tn immunoreaction was found in the central cornea at the flap interface. Five months after LASIK, the general pattern of both EDA-cFn and Tn immunoreactivities resembled that observed in 2.5-month specimens. Although rabbit corneas are different from human ones, the results were similar to those in human corneas. In vivo confocal microscopy has been used to study stromal changes in human corneas after LASIK. Keratocyte activity at the interface seems to peak in the early postoperative course (1–2 weeks), and by 6 months there appears to be a loss of keratocytes in the anterior portion of the flap (7, 8). Anderson and colleagues found active healing at 3 months after successful LASIK that virtually disappeared at 20 months (9). The wound repair response included altered collagen alignment, reactive keratocytes, and PAS-positive electron dense material in the wound interface. At 20 months postoperatively, there were occasional areas of separation between the flap and the interface, and only a few reactive keratocytes at the interface, most notably at the flap hinge.

Our results with UBM are similar to histologic and ultrastructural confoscan and UBM data reported in literature (10). In the case we report these changes were more evident due to the trauma. In particular, the hyper-reflective area at the margin of the flap could be the epithelial plug stimulated by fibronectin and tenascin (Fig. 3A). The thin hyporeflective band at the interface would correspond to the residual corneal edema (Fig. 3B). The underlying stromal hyper-reflectivity, on the other hand, could be due to the cellular hyper-reflectivity secondary to repair and regenerative processes (Fig. 3C). These aspects, with modest variations, were still evidenced in the following months. After 1 year, UBM showed quiescence of the various corneal layers.

The authors report no commercial or proprietary interest.

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