Severe diffuse lamellar keratitis after femtosecond lamellar keratectomy

A femtosecond laser microkeratome is becoming a popular device for performing lamellar cuts before laser ablation during laser-assisted in situ keratomileusis (LASIK) surgery, achieving good refractive outcomes and a low rate of complications.[1–5]

Inflammatory reactions at the interface after using a femtosecond laser for creating lamellar cuts have been communicated at ophthalmology meetings but not yet published in papers. We present a case of severe diffuse lamellar keratitis (DLK) after using IntraLase (IntraLase Corp, Irvine, CA) for performing LASIK surgery.

Case report

A 32-year-old Caucasian male patient attended our clinic for refractive surgery. He had nonrelevant medical or ocular history. The preoperative cicloplegic refraction was right eye, −1.25 sph, −1 cyl×80° visual acuity = 1 (decimal scale); left eye, −1.25 sph, −1.75 cyl×90° visual acuity = 1. Pachymetry and topography were adequate for excimer laser ablations. The rest of the ocular examination was unremarkable.

He underwent uneventful bilateral LASIK using IntraLase. Parameters used for cutting: 120 μm (depth of the cuts), 50° for superior hinge, 9 mm (diameters of the flap), 1.6 mJ of energy for the lamellar cut and 2.5 mJ for the side cut. An esiris excimer laser (Schwind, Frankfurt, Germany) was used for the refractive ablation. Standard postoperative treatment was prescribed: Tobradex (Alcon-Cusí, El-Masnou, Barcelona, Spain) every 6 h for 1 week and preservative-free tears (Vislube, Thea, Barcelona, Spain) every 12 h for 1 month.

Forty eight hours after surgery, the patient complained of misty vision in his right eye and had uncorrected visual acuities of 0.9 for the right eye and 1 for the left eye. Biomicroscopy showed a dense aggregate of clumped cells in the whole interface, including visual axis (fig 1) affecting the right eye and more scattered cells at the periphery of the flap in the left eye, with DLK stages III and I being diagnosed, respectively. A confocal microscopy examination (Tandem Scanning Confocal Microscope-165A; ASL, Reston, Virginia, USA) was performed (fig 2), and intensive topical corticosteroid treatment was started every hour.

Three weeks after surgery, the patient's uncorrected visual acuities were 0.5 for the right eye and 1 for the left eye, and the subjective refraction: right eye: +2 sph, −0.5 cyl×145° visual acuity = 0.7; left eye: −0.25 cyl×90°, visual acuity = 1.2. Biomicroscopy showed the appearance of stromal melting with moderate scarring and corrugated ''mud cracks'' in the right eye. The manifest refraction of the patient 3 months after LASIK was right eye: +2.5 sph, −0.5 cyl×115° visual acuity = 0.8; left eye: +0.5 sph −0.25 cyl×90°, visual acuity = 1.

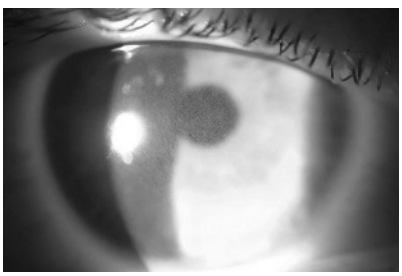

Figure 1 Biomicroscopy showing a dense aggregate of clumped cells in visual axis.

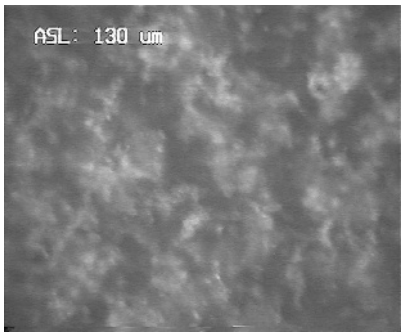

Figure 2 Confocal microscopical examination after surgery.

Discussion

DLK is a multi-aetiological syndrome characterised by an inflammatory response at the interface in patients operated on by LASIK.[6–8]

The ability of Nd-YAG photodisruption to evoke this syndrome after LASIK using IntraLase has not been published previously. The diagnosis of DLK in our case was based on the clinical features and the confocal microscopy images.[9] Before the presentation of this case, some mild inflammatory interface reactions (DLK I and II) after IntraLase cuts had been seen in our unit.[10] A posterior reduction in the levels of energy to 1.2 mJ (lamellar cut) and 1.4 mJ (side cut) was followed by the disappearance of the reaction.

A specific inflammatory-related complication after using femtosecond laser microkeratomes has been recently reported, the transient light sensitivity syndrome.[11] For this new syndrome, a positive correlation between its incidence and the energy settings has been found.

As the initial parameters of energy installed in our IntraLase unit were standard, and as the chance of evoking inflammation at the interface seems to be related to the levels of energy, these parameters should be carefully monitored at the installation and customised for each unit.

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References

1 **Ratkay-Traub I**, Ferincz IE, Juhasz T, *et al*. First clinical results with the femtosecond neodynium-glass laser in refractive surgery. *J Refract Surg* 2003;**19**:94–103.
2 **Binder PS**. Flap dimensions created with the IntraLase FS laser. *J Cataract Refract Surg* 2004;**30**:26–32.
3 **Principe AH**, Lin DY, Small KW, *et al*. Macular hemorrhage after laser in situ keratomileusis (LASIK) with femtosecond laser flap creation. *Am J Ophthalmol* 2004;**138**:657–9.
4 **Tran DB**, Sarayba MA, Bor Z, *et al*. Randomized prospective clinical study comparing induced aberrations with IntraLase and Hansatome flap creation in fellow eyes: potential impact on wavefront-guided laser in situ keratomileusis. *J Cataract Refract Surg* 2005;**31**:97–105.
5 **Durrie DS**, Kezirian GM. Femtosecond laser versus mechanical keratome flaps in wavefront-guided laser in situ keratomileusis: prospective contralateral eye study. *J Cataract Refract Surg* 2005;**31**:120–6.
6 **Smith RJ**, Maloney RK. Diffuse lamellar keratitis: a new syndrome in lamellar refractive surgery. *Ophthalmology* 1998;**105**:1721–6.
7 **Holland SP**, Mathias RG, Mork DW, *et al*. Diffuse lamellar keratitis related to endotoxins released from sterilizer reservoir biofilms. *Ophthalmology*. 2000;107: 1227–33; discussion 1233–, 1234.
8 **Shah MN**, Misra M, Wihelmus KR, *et al*. Diffuse lamellar keratitis associated with epithelial defects after laser in situ keratomileusis. *J Cataract Refract Surg* 2000;**26**:1312–18.
9 **Tervo T**, Moilanen J. In vivo confocal microscopy for evaluation of wound healing following corneal refractive surgery. *Prog Retin Eye Res* 2003;**22**:339–58.
10 **Javaloy J**, Vidal MT, Abdelrahman A, *et al*. Femtosecond and mechanical microkeratomes: a comparative quality control study using confocal microscopy. *J Refract Surg*, 2006.In press.
11 **Stonecipher KG**, Dishler JG, Ignacio TS, *et al*. Transient light sensitivity after femtosecond laser flap creation: clinical findings and management. *J Cataract Refract Surg* 2006;**32**:91–4.

Evidence for retinal remodelling in retinitis pigmentosa caused by *PDE6B* mutation

Retinitis pigmentosa is a genetically heterogeneous group of progressive retinal degenerations.[1] Autosomal recessive retinitis pigmentosa caused by mutations in the gene encoding the β-subunit of rod photoreceptor cyclic guanosine monophosphate-phosphodiesterase (PDE6B) was one of the first forms to be identified, and there are well-studied murine and canine animal models as well as proof-of-concept success of somatic gene therapy.[1–4] Rapid rod photoreceptor degeneration in the animal models is complicated by morphological changes involving the inner retina.[5 6] It is unknown, however, whether the human form of retinitis pigmentosa is also complicated by retinal remodelling; the answer could have implications for treatment potential. We used optical coherence tomography (OCT) to study the retina of a patient with retinitis pigmentosa with a known *PDE6B* null mutation,[7] and found there was abnormal laminar architecture suggesting retinal remodelling.

Case report

A 25-year-old woman with retinitis pigmentosa was homozygous for the Cys270X mutation in *PDE6B*. There was no rod function and only severely impaired cone function.[7]