Short communication

Post-laser in situ keratomileusis refractive changes induced by glaucoma in the absence of keratectasia

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A R T I C L E  I N F O

Article history:
Received 2 November 2011
Accepted 24 January 2013
Available online 22 October 2014

Keywords:
Corneal ectasia
Glaucoma
LASIK
Myopic shift
Intraocular pressure

A B S T R A C T

Case report: We report the case of a 34-year-old man with progressive loss of visual acuity in his left eye, who was diagnosed with pigmentary glaucoma with an intraocular pressure of 32 mmHg. Eight years previously, the patient had undergone refractive surgery (LASIK). Hypotensive treatment led to a significant refractive change accompanied by flattening of the corneal curvature.

Discussion: We propose that a high intraocular pressure acting on a cornea weakened by refractive surgery can provoke corneal steepening, inducing refractive changes even in the absence of keratectasia.

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Cambios refractivos post-keratomileusis láser in situ inducidos por hipertensión ocular aguda sin signos de ectasia corneal

R E S U M E N

Caso clínico: Se presenta el caso de un varón de 34 años de edad, con pérdida progresiva de visión en el ojo izquierdo, que fue diagnosticado de glaucoma pigmentario con una presión intraocular de 32 mmHg. Ocho años antes, el paciente había sido intervenido con cirugía refractiva (lasik). El tratamiento hipotensor condujo a cambios refractivos significativos acompañados de aplanamiento de la curvatura corneal.

Discusión: Proponemos que una elevada presión intraocular, actuando sobre una córnea previamente debilitada por la cirugía refractiva, puede provocar alteraciones en la superficie corneal, induciendo cambios refractivos incluso en ausencia de ectasia corneal.

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* Please cite this article as: Martínez-de-la-Casa JM, Rodríguez-Uña I, González-Pastor E, Díaz-Valle D, García-Feijóo J. Cambios refractivos post-keratomileusis láser in situ inducidos por hipertensión ocular aguda sin signos de ectasia corneal. Arch Soc Esp Oftalmol. 2014;89:414-417.

** Presented as a panel communication at the 86th Congress of the Ophthalmology Society of Spain, September 2011, Oviedo, Spain.

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Introduction

The possible effects of high intraocular pressure (IOP) on a cornea which was previously submitted to Laser-assisted in situ keratomileusis (LASIK) have not been examined in depth. The authors present the case of a patient intervened with refractive surgery in which refractive and topographic changes were documented as the consequence of acute ocular hypertension without signs of corneal ectasia.

Clinic case

Male, 34, submitted to LASIK 8 years before for correcting a 6 diopter myopia. Seven years after surgery, the patient visited for a routine checkup referring gradual visual acuity (VA) reduction in the left eye (LE). Uncorrected VA was 0.1 and best corrected visual acuity (BCVA) was 0.8 (−2.00 to 0.75 at 40°).

Anterior pole examination evidenced pigment dispersion syndrome with iridial transillumination and Krukenberg spindle (Fig. 1). The corneal flap was adequately positioned without Hayes. Anterior chamber was broad and clear. Fig. 2 shows corneal topography at the time. IOP, assessed with applanation tonometry at the central cornea level was 32 mmHg. Corneal thickness, assessed by means of ultrasound pachymetry was of 527 μm. The left optic nerve papilla exhibited significant thinning of the neuroretinal ring at the inferior level. Treatment with latanoprost once a day was established on the basis of pigmentary glaucoma diagnostic.

In a visit 6 weeks later, LE VA had improved spontaneously. BCVA was 0.8 with −0.75 to 0.50 at 40°. Uncorrected VA was now of 0.6. Significant corneal thickness reduction was observed in comparison with the previous dissemination (ultrasound pachymetry: 465 μm) together with slight central corneal flattening (Fig. 3). Biomicroscopy revealed a cornea similar to that of the previous examination and IOP had diminished to 16 mmHg.

After suspending latanoprost, the refractive error reappeared and the topographic values (corneal curvature and thickness) returned to the pre-treatment values. Treatment was established with 0.5% timolol maleate, observing IOP reduction and corneal effects similar to those obtained with latanoprost: IOP was now at 18 mmHg and central corneal thinning and flattening was evidenced (Fig. 4). Corneal edema...
Fig. 3 – Corneal topography after 6 weeks treatment with latanoprost. Central corneal flattening and thickness reduction can be seen when comparing with the tomography taken before the hypotensor treatment.

Fig. 4 – Corneal topography after treatment with timolol, showing similar values to those obtained after treatment with latanoprost.
or fluid in the interface was not detected at any point during the entire follow-up period.

Discussion

The current literature contains a few isolated cases of refractive changes induced by IOP increases, mostly associated to corticoid treatment in the early post-surgery period.\(^1\) Similarly, refractive changes have been described in a corneal ectasia which were modified by anti-glaucoma treatment.\(^2\) However, the authors are not aware of the publication of previous studies describing refractive defects induced by raised IOP in corneae previously submitted to LASIK, without signs of corneal ectasia.

The present case did not exhibit increased IOP before the patient developed pigmentary glaucoma. This type of secondary glaucoma typically affects young myopic individuals and can cause very high IOP figures. The authors propose that, in response to a significant IOP increase, the cornea, which was already weakened by the surgery, developed a slight central curvature leading to a myopic refractive defect. In fact, a difference of 1 dpt was observed between the topography measures taken before and after the hypotensor treatment, and this could contribute to the observed refractive defect.

When considering the changes in central corneal thickness, the suggested first possibility was that these could be caused by the use of latanoprost as a hypotensor agent because the use of prostaglandin analogs has been related with changes in pachymetry.\(^3\) However, the fact that said changes reappeared after treatment with timolol prompted the authors to discard said possibility. Consequently, it was proposed that the corneal thickness variations in this case could be related to subclinic modifications in the corneal stroma hydration as the pressure thereof is inversely related to corneal thickness.\(^4,5\)

Accordingly, the corneal thickness produced during refractive surgery for myopia could produce diminished corneal hydration pressure, particularly in eyes submitted to LASIK where the corneal flap scarcely contributes to the maintenance of corneal architecture. In fact, no signs of ectasia were found in the 2 topographies of said patient.

On the basis of the above interpretations, the authors conclude that in patients submitted to LASIK surgery a significant increase of IOP, either induced or as a consequence of glaucoma, could give rise to subclinic changes in the corneal stroma hydration leading to an increase in corneal thickness and changes in its curvature. The consequences of said modifications are refractive changes, even in the absence of corneal ectasia.

Conflict of interests

No conflict of interests has been declared by the authors.

REFERENCES