Optic nerve head (ONH) in healthy subjects is not usually affected by LASIK surgery. Studies carried out with confocal scanning laser ophthalmoscopy, scanning laser polarimetry and optical coherence tomography (OCT) technologies found no evidence of retinal nerve fiber layer (RNFL) thinning or ONH morphology damage after LASIK. However, LASIK-induced optic neuropathy has been described as a rare complication after this procedure. It usually presents immediately after the surgery and may be related to the marked increase in intraocular pressure produced intraoperatively by the suction ring.

We report 2 cases of unusual acute optic neuropathies following LASIK, with a delay in the onset of symptoms and a different etiology to the previously mentioned intraoperative barotrauma.
6 hours and 3 days postoperatively, examination of the corneas was unremarkable; in the second visit, uncorrected distance visual acuity (UDVA) was 20/20 bilaterally. On the fifth day after surgery, the patient referred decreased UDVA in his right eye; UDVA was 20/32 and 20/20 in his left eye; IOP was 39 mmHg in the right and 25 mmHg in the left eye; slitlamp examination disclosed a hypertension induced corneal edema in the right eye and a normal cornea in the left. Tobradex was discontinued and Timolol maleate 0.5% was prescribed twice daily. Three days later, IOP was 11 mmHg in both eyes and corneal examination had returned to normal, but the patient complained of a right eye paracentral scotoma and UDVA was 20/30; eye fundus examination disclosed supero-nasal disk margins blurring (Figure 1,A) in his right eye. Right eye automated perimetry (Humphreys analyzer) disclosed an inferior arcuate scotoma, and OCT an increase in the superior peripapillar retinal fiber layer (RNFL) (Figure 2,A). Left eye examinations were within normal limits. Physical neurological examination, brain and orbits magnetic resonance (MR), lumbar puncture, blood test for infectious or autoimmune causes of optic neuropathies were all within normal limits.

He was finally diagnosed of an ischemic anterior optic neuropathy secondary to a severe acute steroid-induced ocular hypertension after a LASIK procedure. With no other treatment, after 15 months of follow-up, UDVA has returned to 20/20, but the patient complains of a persistent positive paracentral scotoma, disclosed in his visual field analysis; disk edema resolved but a mild superior optic disk atrophy established (Figure 1,B); in OCT, peripapillar RNFL progressively thinned in the superior quadrants to a subnormal final thickness (Figure 2, B,C).

Case 2

A 30-year-old man presented for refractive surgery; he reported no previous ocular or systemic conditions. Manifest refraction was −3.5 sph in the right eye and −3.75 sph in the left eye, achieving a CDVA of 20/20 in both eyes. Slitlamp examination, corneal topography, and eye fundus examination were all within normal limits. Preoperative IOP was 18 mmHg and US central corneal pachymetry 545 µm in both eyes. Uneventful bilateral sequential LASIK procedure was performed with a Technolas 217 Z excimer laser and the Moria LSK-One microkeratome with the 80 µm head. Topical tobramycin 0.1%/dexametasone 0.3% (Tobradex) was prescribed 4 times daily for 1 week.

6 hours and 6 days postoperative visits were unremarkable with UDVA 20/20 bilaterally; 20 days after the procedure, the patient complained of a mild decrease in UCVA in right eye, with retrobulbar discomfort increased with eye movements. The patient was emmetropic, with an UCDVA of 20/30 in right
eye and 20/20 in left eye. Anterior segment examination was unremarkable, but a subtle optic disk swelling and hyperemia in his right eye fundus was disclosed. Automated perimetry showed a diffuse visual field loss in his right eye, and OCT a superior and inferior thickening in right peripapillary RNFL. These tests were within normal limits for left eye.

Orbits and brain imaging showed multiple hyperintense lesions in the subcortical white matter and corpus callosum in the T2 and flair MR sequences.

The rest of neuro-ophthalmic examination and lumbar puncture were unremarkable.

The patient was then treated with intravenous methylprednisolone (1 g/day for 3 days) and then tapered with oral prednisone for 11 days.

Five months after the LASIK procedure right eye UCVA had returned to 20/20, OCT RNFL thickening had decreased to normal limits and milder diffuse visual defect persisted in the automated visual field examination. No new neurological symptoms have presented up to date.

The patient was finally diagnosed to have suffered an optic neuritis, as the first clinical manifestation of a demyelinating disease, incidentally occurring just after his refractive surgery.

**DISCUSSION**

Optic neuropathy is a rare but potentially vision-threatening complication described after LASIK with conventional microkeratomes, with femtolascer flap creation and also after epi-LASIK. Barotrauma or conventional microkeratomes, with femtolascer flap creation is the most described mechanism.

We routinely use in our LASIK procedures the Moria LSK-One microkeratome and we only need a very short time of suction (10-12 seconds) with it; to our knowledge we have not had any other case of clinical optic neuropathy after more than 300,000 procedures during the last 10 years at our institution.

Patients with «disk at risk» for this complication (optic nerve drusen or small cup disk) should be carefully evaluated before LASIK, specially when using devices for flap creation with long suction times; surface ablation surgery should be considered in these cases.

Other mechanisms can produce optic nerve damage after LASIK surgery. Steroid-induced ocular hypertension should be suspected when the onset of symptoms are not immediate after the surgery. Myopic patients with previous higher IOP are at higher risk to develop this drug-related adverse effect. Our first patient developed the ischemic optic neuropathy in his right eye, which had a much more severe steroid-induced IOP rise and a previous higher preoperative IOP than the left eye. A careful evaluation and a high index of suspicion are needed with these patients as their IOP will be underestimated with conventional tonometric techniques after the LASIK procedure.

Demyelinating disease should be also ruled out in these cases, as it is by far the most frequent cause of optic nerve damage in the general population between 20 and 45 years of age. A temporal coincidence with the LASIK procedure, as in our second patient, may exist.

These two cases of post-LASIK optic neuropathies and other previously described, illustrate the need of a careful examination of the optic nerve before and after the procedure.

Patients who have LASIK can rarely experience an acute optic neuropathy; this may be caused directly by the surgical procedure, but it could also be a secondary effect of the postoperative medication, as our first patient; finally, as in our second patient, the relationship between LASIK surgery and the onset of nerve damage can only be a casual temporal coincidence and not a causal effect. A thorough work-up is mandatory to disclose alternative etiologies if optic nerve damage is diagnosed after the procedure.

**REFERENCES**