MYOPIA: MORE THAN A REFRACTIVE ERROR – LASIK AND RETINAL DYSTROPHIES

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SUMMARY

Three patients who had undergone laser in situ keratomileusis (LASIK) correction for myopia were first seen because of suboptimal visual acuity (VA) and night blindness and/or photophobia. After a comprehensive examination including psychophysical and electrophysiological tests, two of the three patients were shown to suffer from a progressive cone-rod dystrophy. The third patient had retinitis pigmentosa. These cases illustrate the need for in depth preoperative evaluation in myopic patients about to undergo LASIK when signs or problems of night blindness and/or photophobia are present.

RÉSUMÉ

Trois patients sont présentés ayant été examinés pour une acuité visuelle sous-optimale et une héméralopie et/ou photophobie, après correction d'une myopie suivant la technique du laser in situ keratomileusis (LASIK). Sur base d'une évaluation élaborée, y inclus des tests psychophysiques et éléctrophysiologiques, un diagnostic de dystrophie des cônes et bâtonnets a été établi chez deux patients. Le troisième patient souffrait d'une rétinite pigmentaire. Ces cas soulignent la nécessité d'une évaluation préopératoire approfondie de patients myopes qui vont subir du LASIK quand ils présentent des signes ou problèmes d'héméralopie et/ou photophobie.

SAMENVATTING

Drie patiënten die een correctie van myopie hadden ondergaan met laser in situ keratomileusis (LASIK) werden onderzocht omwille van postoperatieve suboptimale visus en nachtblindheid en/of fotofobie. Na uitgebreid onderzoek met inbegrip van psychofysische en electrofysiologische testen werd een diagnose van progressieve kegeltjes-staafjesdystrofie gesteld bij twee patiënten. De derde patiënt leed aan retinitis pigmentosa. Deze gevallen illustreren de noodzaak van een doorgedreven preoperatief onderzoek bij myope patiënten die LASIK zullen ondergaan met klachten van nachtblindheid en fotofobie.

KEY WORDS

Retinal dystrophy, cone-rod dystrophy, retinitis pigmentosa, photophobia, night blindness, laser in situ keratomileusis, preoperative evaluation

MOTS-CLÉS

Dystrophie rétinienne, dystrophie de type cônes-bâtonnets, rétinite pigmentaire, photophobie, héméralopie, laser in situ keratomileusis, évaluation préopératoire

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INTRODUCTION

Laser in situ keratomileusis (LASIK) is a very popular and effective surgical technique to correct mild or moderate and even high myopia (20). As for any surgical procedure, several postoperative complications have been described. Although these complications are mostly related to the cornea (flap, interface, stromal bed) retinal and optic nerve problems have also been described after LASIK (1,27,29,32). Complications related to the cornea include dry eye syndrome, flap displacement, flap melting, diffuse lamellar keratitis, infection, epithelial ingrowth, keratectasia, corneascleral perforations and irregular astigmatism (4,27,29,30). Reported complications in fundo are peripheral retinal tears, retinal detachments, macular haemorrhages, central retinal vein occlusion, macular holes, choroidal neovascular membranes and ischemic optic neuropathy (1,2,12,14,18,22,23,25,27-29,32).

Retinitis pigmentosa (RP) is the term for a group of inherited disorders of the retina characterized by a progressive loss of photoreceptors (3). All types of RP lead to typical fundoscopic changes including outer retinal atrophy and intraretinal pigmentary changes more pronounced in the retinal periphery, secondary attenuation of the retinal vasculature and progressive atrophy of the nerve fiber layer leading to a waxy aspect of the optic nerve head (3). Symptoms include early night blindness, progressive loss of the peripheral visual fields and subsequent loss of central vision (3). The age of onset of RP as well as severity of disease is highly variable. The condition can be inherited as an autosomal dominant, an autosomal recessive or an X-linked trait (3). Knowledge of the molecular background of RP is expanding rapidly (7).

Cone-rod dystrophy is also a clinically and genetically heterogeneous progressive disorder first affecting the cones and subsequently also the rods, in which patients characteristically complain of reduced visual acuity, photophobia and impairment of colour vision (3,7). The age of onset is variable, but most patients present in the first two decades of life. Visual loss slowly progresses to a level of counting fingers in the later stages of the disease.

We report three patients who underwent LASIK correction for myopia and were subsequently examined by us because of suboptimal visual acuity and night blindness and/or photophobia. All three patients underwent a comprehensive ophthalmological, psychophysical and electrophysiological examination.

CASE REPORTS

Case 1:

A healthy 48-year-old man was examined in our department because of decreased visual acuity, photophobia, monocular diplopia and complaints of seeing halos around the lights, after an uneventful bilateral LASIK.
The preoperative best-corrected visual acuity (BCVA) was 7/10 in the right eye and 8/10 in the left eye. The refractive error prior to surgery was –8,0D(–1,50D)170° in the right eye and –5,25D(–1,75)165° in the left eye. Preoperative slit lamp examination and intraocular pressure were unremarkable in both eyes. Fundoscopy had apparently revealed pigment alterations in both macular areae.
He received a second LASIK treatment in the right eye because of deterioration of central vision thought to be related to a myopic refractive regression of 1,25D 1 week after initial treatment.
We saw the patient for the first time 17 months post LASIK treatment. He was referred to our department because of poor central vision. Daylight vision was satisfactory.
The BCVA in the right eye was 3/10 with –0,50D(–0,50D)90° and 6/10 without correction in the left eye.
Slit lamp examination revealed superficial stromal opacities, more pronounced in the right than the left eye. A slightly superiorly decentered ablation in the right eye and a regular ablation in the left eye were seen on corneal topography. Fundoscopy and conventional fluorescein angiography showed macular pigment alterations apart from myopic choroidosis (Figure 1). There was no sign of subretinal choroidal neovascularisation. Goldmann visual field (VA) analysis showed mild to moderate loss of central sensitivity and moderate concentric narrowing in both eyes. Colour vision examination

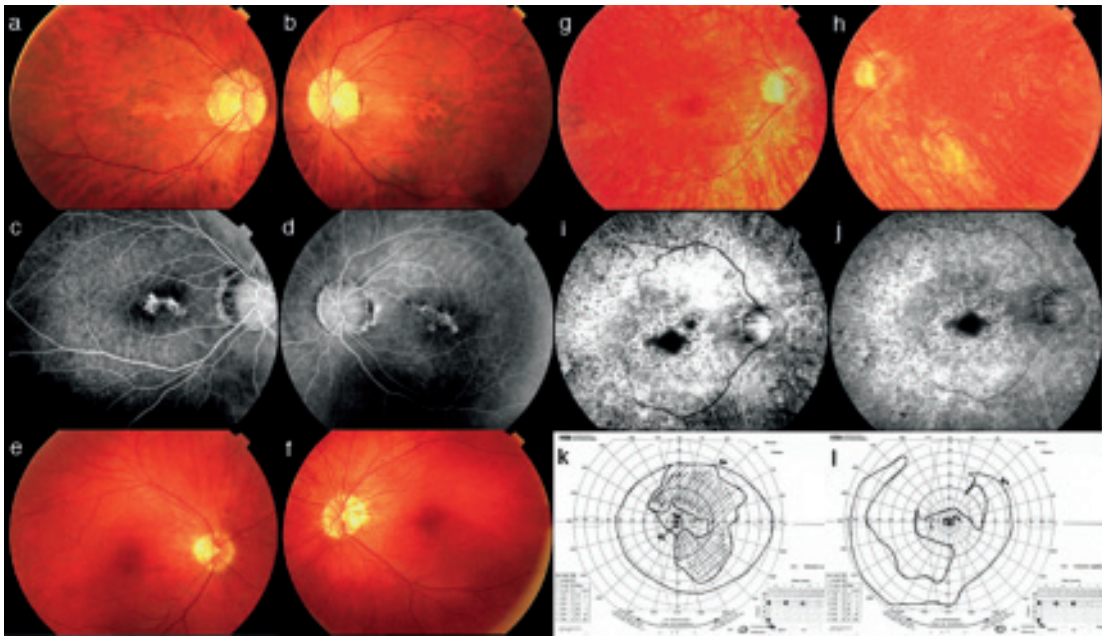


Figure 1. Composite of fundus pictures, fluorescein angiographies (FA) and Goldmann visual field analysis (GVF);
Panels a & b: RE & LE respectively of patient 1, illustrating retinal pigment epithelial alterations in central macular region;
Panels c & d: window defects give clearer view of changes in patient 1 on FA
Panels e & f: RE & LE respectively of patient 2, showing only myopic changes with tilted disc and temporal peripapillar chorioretinal atrophic crescent
Panels g & h: macular & nasal midperiphery respectively of patient 3 with evidence of myopic fundus, with bull's eye aspect in the macular area, small tilted optic disc with myopic conus, albinoid aspect of retina and attenuation of retinal vessels; a few areas of spicular intraretinal hyperpigmentation in nasal periphery
Panels i & j: early phase FA of RE better illustrates bull's eye maculopathy and atrophic aspect of both posterior pole and periphery
Panels k & l moderate constriction of visual fields and reduced sensitivity on Goldmann VF analysis, with large pericentral scotoma in RE and incomplete annular scotoma with temporal island in LE respectively

revealed a red-green defect in both eyes. ISCEV-standard electroretinography showed slightly subnormal scotopic responses. Oscillatory potentials were only residual, whilst combined maximal rod-cone responses showed amplitudes of about 3/5 of normal values. Both the photopic transient responses as well as the 30Hz flicker responses were reduced to 1/3 of normal values (Figure 2). Goldmann-Weekers dark adaptometry thresholds were elevated approximately 1,25 log units.
These results are compatible with a diagnosis of cone-rod dystrophy.

Case 2:

A healthy 48-year-old man was referred because of complaints of poor night vision and seeing halos around lights. The preoperative BCVA was 10/10 in both eyes. The refractive error before surgery was –2,50D(–2,50D)18° in the right eye and –5,0D in the left eye. Preoperative slit lamp examination and intraocular pressure were unremarkable in both eyes. Fundoscopy had shown myopic choroidosis.
He underwent his first LASIK treatment in the left eye. After this first treatment he complained of poor night vision. Because of this the LASIK in the right eye was postponed, but four years after his first treatment he underwent a first LASIK treatment in the right eye and a redo in the left eye.

The patient was referred for the first time to our department 4 1/2 years later after his first LASIK treatment because of poor central vision, com-

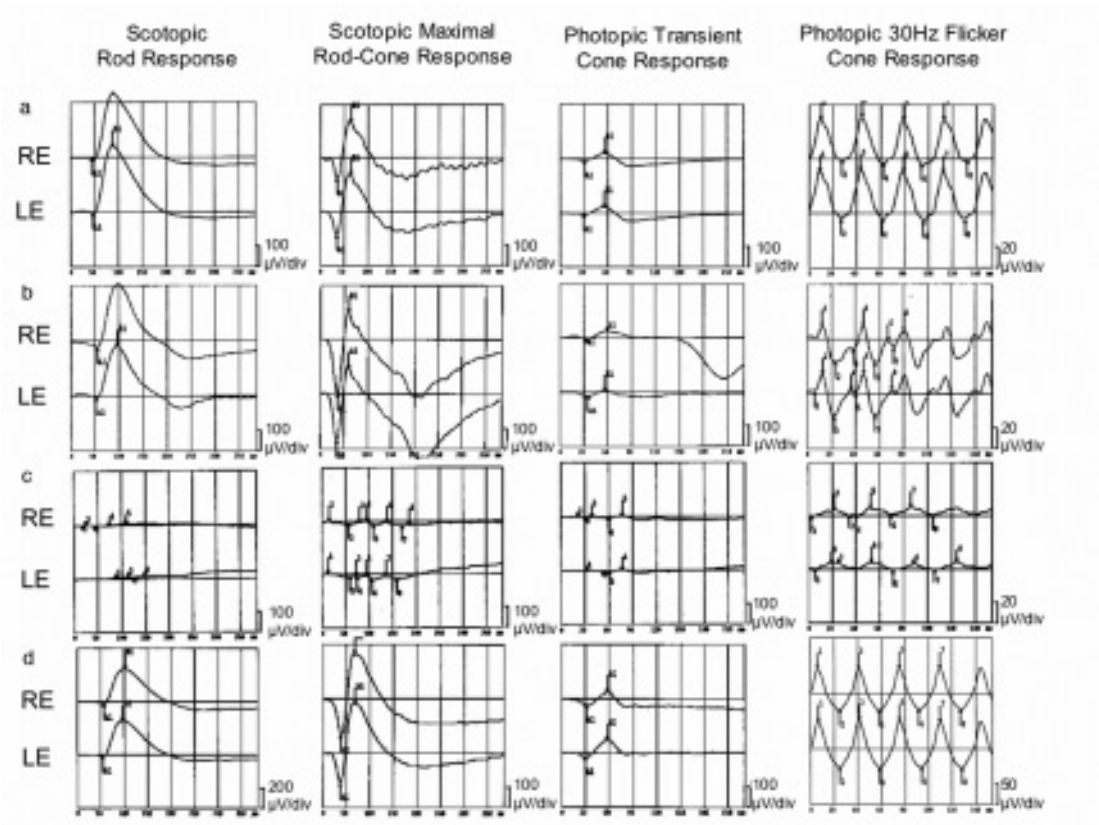


Figure 2.
ISCEV standard electroretinographies of patients; oscillatory potentials not shown; note differences in scaling;
Panel a: ERG of patient 1: slightly subnormal scotopic responses; combined maximal rod-cone responses show amplitudes of 3/5 of normal values; photopic transient responses and 30Hz flicker responses are reduced to 1/3 of normal
Panel b: ERG of patient 2: scotopic rod-specific responses with amplitudes of 3/5 of normal; amplitudes of combined maximal rod-cone responses are about 5/6 of normal, whilst transient photopic cone-specific responses were 1/3 of normal; 30Hz flicker responses are considerable delayed with amplitudes of 1/3 of normal
Panel c: ERG of patient 3: scotopic rod-specific responses are completely absent; amplitudes of photopic transient and 30Hz flicker cone-specific responses very severely reduced, with considerable latency delays
Panel d: normal control with amplitudes at 5th percentile

plaints of poor night vision and seeing halos around lights.
The postoperative BCVA was 5/10 in the right eye and 6/10 in the left eye. Slit lamp examination and intraocular pressure were unremarkable in both eyes. Corneal topography was unremarkable. Fundoscopy revealed a myopic fundus with pigment alterations in the macular area, a tilted disc and a crescent of atrophy temporal to the optic disc (Figure 1). Colour vision examination showed mild abnormalities in the red-green axis and very mild abnormalities in the blue-yellow axis. A bilateral decreased central sensitivity was illustrated by Humphrey automatic visual field.
ISCEV-standard electroretinography (Figure 2) showed scotopic rod-specific responses with amplitudes of 3/5 of normal. Amplitudes of combined maximal rod-cone responses were about 5/6 of normal values, whilst transient photopic cone-specific responses were 1/3 of the normal values. 30Hz flicker responses were considerable delayed with amplitudes of 1/3 of normal. Goldmann-Weekers dark adaptometry thresholds were elevated approximately 1 log unit. Contrast sensitivity was low for middle fre-

quencies and absent for high frequencies in both eyes.
On the basis of these results a diagnosis of progressive cone-rod dystrophy was made.

Case 3:

A healthy 24-year-old man was referred to our department because of decreased visual acuity, complaints of night vision and halos around lights after bilateral LASIK.
Preoperative BCVA was 7/10 in the right eye and 8/10 in the left eye. The refractive error prior to surgery was –7,50D(–1,5D)179° in the right eye and –8,75D(–1,75D)9° in the left eye.
Preoperative slit lamp examination had been unremarkable in both eyes. Fundoscopy was said to have only revealed myopic choroidosis in both eyes.
Subsequent to the first bilateral LASIK treatment, the patient complained of poor night vision and seeing halos around lights. Albeit subnormal, it is understood daylight vision was satisfactory.
During the following 4 years he had 3 repeated treatments in each eye in an attempt to correct for folds and hinges in the corneal flap, all with unsatisfactory results.
One year later he received a first lamellar corneal graft in the right eye in order to try and correct for the longstanding striae. Immediately afterwards he is understood to have developed a central haze, for which he subsequently had a second lamellar graft in the right eye three months later. Another five months after that, the diagnosis of retinitis pigmentosa was made in his first cousin. The same diagnosis was subsequently made in him.
When seen in our department one year later, the postoperative BCVA in the right eye was 3/10 with –14,0D(–0,50)70° and 6/10 with –0,75D(–0,75)157° in the left eye.
Corneal topography showed a slightly decentred lamellar graft superotemporal in the right eye and a large centred ablation zone in the left eye. Slit lamp examination revealed the circular scars of the lamellar graft in the right, and subepithelial horizontal striae in the left eye. A myopic fundus was seen, with annular depigmentation in the macular area, a small optic nerve head with myopic conus, albinoid aspect of the retina and attenuation of the retinal vessels. A few areas of spicular intraretinal hyperpigmentation could be seen in the periphery. Conventional fluorescein angiography of both eyes better revealed a bull's eye maculopathy and atrophic aspect of retinal periphery (figure 1). Moderate constriction of the visual fields (VF) and reduced sensitivity was noted on Goldmann VF analysis, with a large pericentral scotoma in the right eye and an incomplete annular scotoma with a temporal island in the left eye (Figure 1). Colour vision examination revealed a mild red green defect and a more pronounced blue yellow defect. Rod-specific responses were completely absent on ISCEV-standard electroretinography, whilst the photopic cone-specific responses were severely reduced (Figure 2). Goldmann-Weekers dark adaptometry (DA) thresholds were elevated approximately 1 log unit.
These results prompted a diagnosis of retinitis pigmentosa.

DISCUSSION

The common denominator in the three patients described here is a delay in the diagnosis of a retinal dystrophy for several years after a LASIK procedure, despite complaints that could have led to suspicion of retinal disease. The patients were all seen in our department seeking either a second or third opinion.
A number of factors in both the clinical history as well as the ophthalmological examination of the three patients reported here, might have led to a more thorough evaluation. Ideally this could have happened prior to LASIK, or at least after the first and prior to the second LASIK procedure.

These factors include fundus abnormalities, which are understood to have been noted in all three patients already prior to refractive surgery.

Furthermore, it is believed BCVA was already suboptimal in two of the three patients before surgery. Despite complaining of bad central vision after the initial LASIK procedure, the third patient underwent several additional treatments without assessment of retinal function. Unfortunately, in this case it was believed the pa-

tient's rather unspecific complaints were due to folds and hinges in the corneal flap.

Additionally, two patients complained of poor night vision and the third suffered from photophobia, but only after the initial LASIK procedure. Presumably, they had these complaints before undergoing LASIK, but may have failed to mention it prior to the first treatment. Despite these complaints, they all underwent at least one additional LASIK treatment without assessment of retinal function by electroretinography.

This may be because it is known that refractive surgery can induce complaints of poor night vision, which is probably related to decreased contrast sensitivity as well as to seeing halos around lights at night (11). The cause is probably multifactorial, and includes aberrations at the edge of the ablation zone, mild irregular astigmatism, flap striae and decentered ablations (8). In one study reporting several risk factors for complaints of night blindness after LASIK, the authors conclude that attempted degree of spherical correction, size of the optical zone, age of the patient and postoperative spherical equivalent play a major role in complaints of night blindness in the first postoperative year (21). This report further demonstrated a considerable decrease in the prevalence of poor night vision during the first year post LASIK from 25,6% at 1 month, to 4,7% at 12 months after LASIK (21).

Should the findings mentioned above have led to a more thorough assessment prior to LASIK surgery, a diagnosis of a retinal dystrophy would have been made before surgery. In this light it is important to keep in mind that the prevalence of myopia is considerably higher in patients with retinitis pigmentosa than in the normal population (24), which makes it more likely to encounter retinal dystrophy patients in a myopic, rather than in the general population.

It is therefore important to point out that, as advised by the American Academy of Ophthalmology, the preoperative evaluation prior to refractive surgery now consists of a complete medical and ophthalmological history as well as a comprehensive ophthalmologic examination, including dilated funduscopy (31).

It seems very unlikely that LASIK influences the evolution of a retinal dystrophy, albeit that effects of transient pressure rise during the placement of the suction ring on narrowed retinal vessels, are difficult to predict.
Indeed, when posterior segment complications related to LASIK are considered, several of its technical aspects need to be kept in mind.
The 193 nm wavelength used in excimer laser surgery is entirely absorbed at the level of the cornea (26,31), with no apparent risk for damage to the retina.
During the creation of the partial thickness corneal flap with the microkeratome, a suction ring is placed on the eye. It is known that, during this vacuum phase, intraocular pressure (IOP) peaks to more than 65 mmHg (19). Once the partial thickness corneal flap is made, IOP normalizes. The rapid change in intraocular pressure leads to changes in the vitreous that can cause retinal breaks and vitreous or retinal detachments (12,23,25,28,32). The underlying mechanism may be related to an increase in axial length with an anterior shift of the vitreous base and consequent traction on the peripheral retina (10). There are also some reports of ischemic optic neuropathy probably related to the extreme rise of IOP (5, 13,15). Other rare complications of the posterior segment that may be related to the transient rise and subsequent fall in intraocular pressure have also been reported. These include subretinal neovascularisation as well as macular and premacular haemorrhages (6,9,16-18,22,23).

In conclusion, underlying retinal dystrophies should be suspected in candidates for refractive surgery, when medical history reveals complaints of night blindness or photophobia, when BCVA is suboptimal, or when dilated fundus examination reveals pigment epithelial changes in the macula and/or retinal periphery. In such patients, it could be ethically acceptable to perform refractive surgery including LASIK, provided patients have been diagnosed using appropriate clinical, psychophysical and electrophysiological techniques. Patients should also be counselled on their visual prognosis and be extensively informed about the limitations of the potential results of refractive surgery.

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