

SHORT COMMUNICATION

Development of early choroidal neovascular membrane in a young myope after LASIK

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PURPOSE. To describe development of early choroidal neovascular membrane (CNVM) after laser-assisted *in situ* keratomileusis (LASIK) in a young myopic patient with initial differential diagnosis of central serous chorioretinopathy (CSCR) that was successfully managed by photodynamic therapy (PDT).

METHODS. A retrospective interventional case report.

RESULTS. A 20-year-old woman with myopic astigmatism underwent an uneventful LASIK surgery resulting in best-corrected visual acuity (BCVA) of 20/20 bilaterally. One week later, the patient presented with decreased VA in the right eye and was diagnosed initially by her referring ophthalmologist with CSCR, and was treated by systemic corticosteroids without any improvement. Two weeks following LASIK, the patient's VA was found to be 20/200 and clinical evidence of early CNVM was confirmed by fluorescein angiography, indocyanine green angiography, and optical coherence tomography (OCT). PDT resulted in regression of her CNVM with improvement in her VA and OCT findings.

CONCLUSIONS. LASIK-induced early CNVM may be misdiagnosed as CSCR. PDT may be effective in the resolution of LASIK-induced early CNVM. (*Eur J Ophthalmol* 2007; 17: 262-5)

KEY WORDS. LASIK, CSC, CNVM, Corticosteroids

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INTRODUCTION

Laser-assisted *in situ* keratomileusis (LASIK) is an effective and widely used surgical technique for the correction of myopia and hyperopia (1). Vitreoretinal complications associated with these procedures are infrequent and may include retinal breaks, retinal detachment, vitreous hemorrhage, and subretinal macular hemorrhage (2, 3). Choroidal neovascular membrane (CNVM) after LASIK has rarely been reported (4, 5). We describe a myopic patient who underwent LASIK in both eyes and later presented with early CN-

VM in the right eye while being treated with systemic corticosteroids that responded well to photodynamic therapy (PDT).

Case report

A 20-year-old woman with myopic astigmatism was referred for evaluation of decreased visual acuity (VA) after an uneventful LASIK procedure in both eyes at a local clinic. There was no significant past ocular or medical history and family history was noncontributory. Her best-corrected VA (BCVA) had been 20/20

Fig. 1 - Clinical photograph of the right fundus in a 20-year-old woman 1 week after uneventful laser-assisted in situ keratomileusis surgery (A); fluorescein angiography of the right fundus (mid-phase), showing hot spot of hyperfluorescein with late staining and leakage around the optic disc (B, C); in addition, there is a localized area of hyperfluorescein at the temporal macula; optical coherence tomography of the right macula shows minimum thickness at the retinal pigment epithelium level (D).

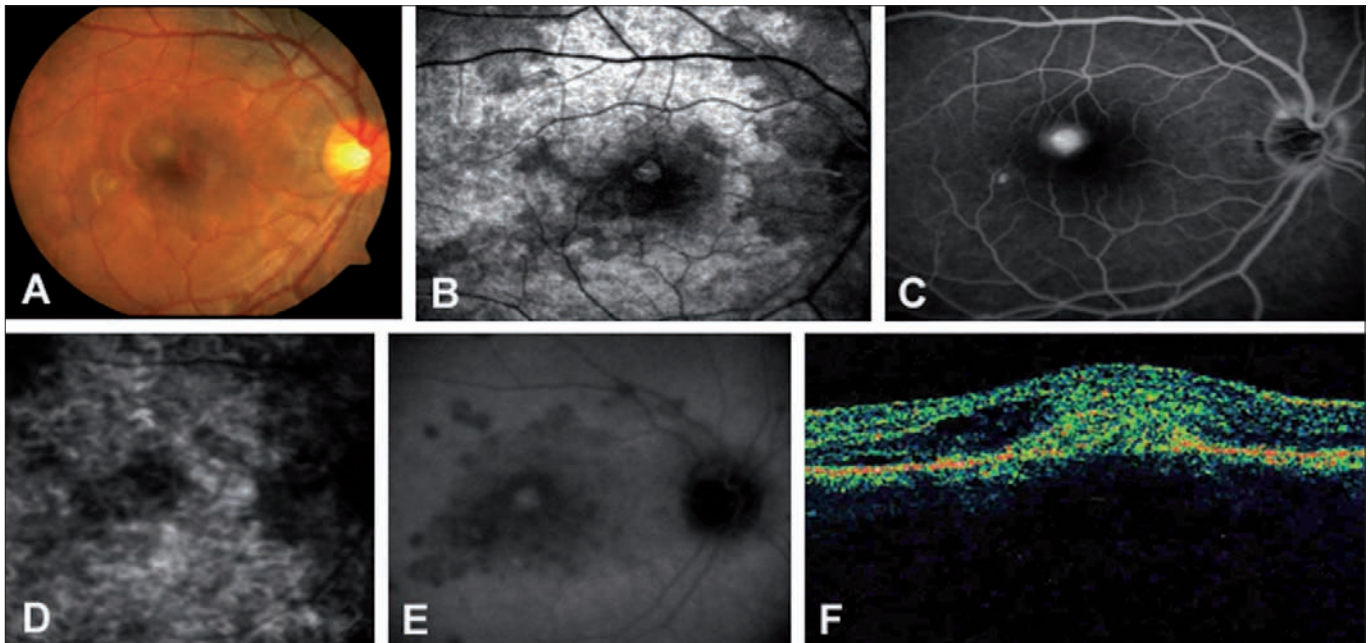
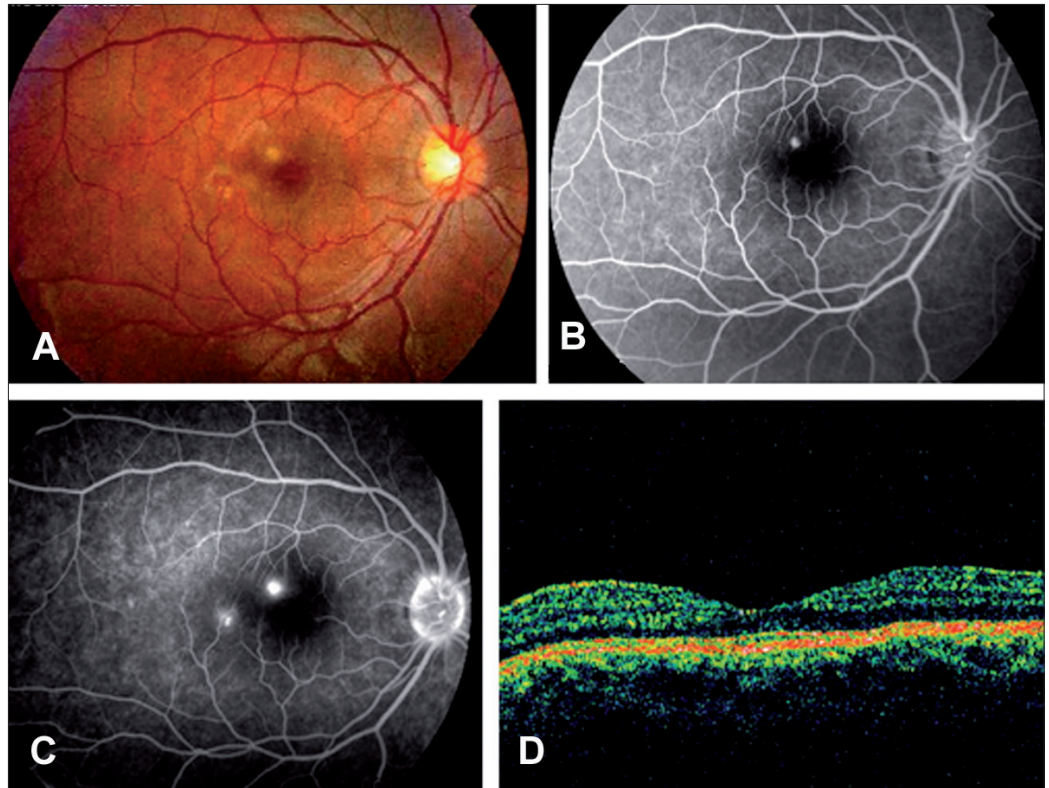


Fig. 2 - Fundus photograph of the right eye showing subretinal turbid fluid under the central macula (A); on fluorescein angiography (FA), there is retinal pigment epithelium (RPE) detachment with central area of choroidal neovascular membrane (CNVM) (B, C); on indocyanine green angiography, there are multiple areas of central serous chorioretinopathy with areas of CNVM that correlated with FA findings (D, E); optical coherence tomography of the right macula shows thick fibrovascular membrane at the RPE level to inner surface of retina (F).



Fig. 3 - Fundus photograph of the right eye showing cicatricial changes at the place of choroidal neovascular membrane (CNVM) after photodynamic therapy (A); fluorescein angiography of the right macula showing minimum staining at the place of CNVM with no obvious leakage (B); optical coherence tomography showing absence of fibrovascular membrane (C).

bilaterally with refractive errors of $-7.00+1.00 \times 90$ in the right eye and $-7.00+1.00 \times 105$ in the left. Dilated fundus examination of both eyes had revealed normal flat myopic fundi. After LASIK procedure, the patient had uncorrected VA of 20/20 bilaterally. One week later, the patient complained about decreased VA in the right eye and initially was diagnosed with CSCR based on fluorescein angiography (FA) (Fig. 1, A–C), which was not established by optical coherence tomography (OCT) (Fig. 1D). She had been started on systemic corticosteroids. One week later, the patient was referred for further treatment after no improvement in her VA was noted. On presentation to our hospital, her BCVA was 20/200 in the right eye and 20/20 in the left with normal intraocular pressures. Slit lamp examination bilaterally showed clear corneas with flaps in place and normal anterior segments with clear lenses. Fundus examination showed clear vitreous bilaterally and localized neurosensory retinal elevation, turbid subretinal fluid with retinal pigment epithelial (RPE) detachment in the right macular area (Fig. 2A), and normal retina in the left eye. Clinically, early CNVM was suspected in the right macula. Intravenous FA showed juxtafoveal CNVM with additional findings of multiple areas of CSCR on ICG (Fig. 2, B–E). OCT of the right macular area showed subretinal fibrovascular lesion with central macular thickness of $343 \mu\text{m}$ (Fig. 2F). She was advised to discontinue the use of systemic corticosteroids. The patient agreed to undergo PDT, which resulted in significant regression of her CNVM as documented by IVFA, ICG, and OCT (Fig. 3, A–C). Her BCVA improved to 20/60 in the

right eye and remained 20/20 in the left at her last follow-up at 6 months.

DISCUSSION

CSCR is a localized serous detachment of the posterior pole of the retina, occurring as a result of a focal defect in the RPE, which leads to fluid of choroidal origin leaking into the subretinal space, causing a detachment of the neurosensory retina in the macular area. The diagnosis can be confirmed by FA, which reveals one or more leakage points at the level of the RPE. Many conditions, including emotional stress, type A personality, pregnancy, autoimmune diseases, and Cushing's syndrome, have been associated with an increased incidence of CSCR (6). Increased levels of endogenous cortisol have been demonstrated to be present in patients with CSCR (7). It is hypothesized that increased endogenous glucocorticoids may act as the inciting or precipitating factor for the development of CSCR because of their ability to affect the RPE and the choroidal vasculature. Several reports have indicated worsening or precipitation of CSCR in patients started on systemic corticosteroid therapy and chronic complicated cases may develop CNVM (6, 8).

Our patient developed symptoms that might be initially related to CSCR or early subtle CNVM that progressed in 2 weeks after LASIK while being on systemic corticosteroids that responded well to PDT. To our knowledge, this is the first report in which a young myopic patient developed CNVM within 2 weeks of

LASIK. No cause-effect relationship between LASIK and CNVM has been proven; however, the formation of CNVM in early postoperative period could possibly link LASIK technique as a causal role in the development of CNVM (9). Sobha et al observed development of CSCR 1 month after hyperopic LASIK surgery that progressed to CNVM 1 month later (9). We believe that our patient developed early CNVM as an initial stage post-LASIK that rapidly progressed to advanced CNVM as a second stage that could be related to CSCR complicated by administration of systemic corticosteroids. The initial stage could be due to RPE disturbance and defect by mechanical ocular stress in conjunction with shock waves during LASIK procedure rather than due to Bruch's membrane damage especially in less myopic degenerative changes. It is well known that LASIK causes stress on the eye globe during the procedure. In particular, a long and rapid increase in intraocular pressure occurs during the application of a suction ring. Further, the laser ablation process itself can send acoustic shock waves to the posterior pole of the eye globe. In a study of 2955 post-LASIK patients, 3 (0.1%) developed evidence of CNVM. Despite argon laser treatment or submacular surgery, all 3 patients had poor visual outcome (4). CNVM development after LASIK is not lim-

ited to myopic patients alone but can occur in hyperopic patients after LASIK (2, 9). Unlike myopic eyes, however, hyperopic eyes do not appear to have the risk factors related to the Bruch's membrane breaks. CNVM in the setting of myopic degeneration can occur, and is believed to be multifactorial and could be related to stretching and disturbances of RPE and choriocapillaries in the macular region. However, CNVM due to myopic degeneration in a young patient is an uncommon phenomenon (10).

This case report emphasizes that patients undergoing LASIK should be warned about the possible risk of early CNVM. The use of systemic corticosteroids in patients with clinical suspicion of early CNVM should be discouraged. PDT may be effective in the management of LASIK-induced early CNVM and subsequent progression.

None of the authors has any propriety interest in this article.

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