Chapter 8

Optic Neuropathy and Retinal Complications after Refractive Surgery

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Core Messages

- LASIK may be associated with uveitis.
- LASIK may be a safe and efficient option for treating refractive errors in eyes with previous retinal detachment surgery.
- Cryopexy, laser retinopexy, pneumatic retinopexy, or vitrectomy without a scleral band tend not to change the shape or length of the globe and should be preferred to repair RRD.
- Prophylactic treatment of vitreoretinal pathology before LASIK does not guarantee the prevention of post-LASIK vitreoretinal complications.
- It is very important to inform patients LASIK only corrects the refractive aspect of myopia, and vitreoretinal complications after LASIK although infrequent may occur.
- Reasons for poor VA after surgery for RRD post-LASIK include delayed referral to a vitreoretinal specialist.

8.1 Introduction

The prevalence of myopia in the United States ranges from 25 to 46.4% of the adult population [35–63]. In Asian populations, these proportions may be much higher and in African and Pacific island groups, much lower. The market for refractive surgery has a very large potential for people with low (less than −5.00 D) and moderate myopia (−5.01 to −10 D), and most patients fall into one of these two groups [63].

Refractive surgery has been accepted for correcting ametropias; however, this procedure may lead to complications. Hofman et al. [26], Sanders et al. [55], and Feldman et al. [20] have described cases of retinal detachment (RD) after radial keratotomy. Rodriguez and Camacho [45] reported on 14 eyes (12 patients) that had either asymptomatic or symptomatic retinal breaks, subclinical and clinical rhegmatogenous retinal detachment (RD), or both, 7 after automated lamellar keratoplasty (ALK), and 7 after radial keratotomy (RK). Rodriguez et al. [46], Barraquer et al. [11], and Ripandelli et al. [44] have reported retinal detachments af-
ter clear-lens extraction for myopia correction. Ruiz-Moreno and associates [48] reported the results of a clinically controlled study to investigate the rate of retinal detachment after implantation of phakic anterior chamber intraocular lenses (IOLs). The implantation of a phakic anterior chamber IOLs, as a correcting procedure, for severe myopia was followed by a 4.8% incidence of retinal detachment.

Laser-assisted in situ keratomileusis (LASIK) has become one of the most popular options for the correction of low to moderate myopia worldwide [41, 68]. However, complications including optic neuropathy [13], undercorrection and overcorrection [10], flap displacement [29], epithelial ingrowth [37], flap melting [5], keratitis [65], retinal tears [32], RDs [49], retinal phlebitis [29], corneoscleral perforations [3], retinal hemorrhages [3], macular hemorrhages [68], macular holes [15], serous macular detachments [60], choroidal neovascular membranes [3], reactivation of ocular toxoplasmosis [9], and irregular astigmatism have been reported.

The objective of this chapter is to review optic neuropathy and retinal complications that may occur after refractive surgery with an emphasis on LASIK.

8.2 Optic Neuropathy after LASIK

8.2.1 History and Mechanism of Optic Nerve Damage

Anterior ischemic optic neuropathy (AION), in most cases, is due to either arteriosclerosis or temporal arteritis. There are also large varieties of systemic, local vascular, and ocular disorders that can produce anterior ischemic optic neuropathy. The relationship between AION and LASIK was first reported by Lee et al. [30], with four cases of optic neuropathy and an onset of visual loss ranging from the day of surgery to 3 days after LASIK. Since that report, some studies have described the relationship between LASIK and the compromise of vascular supply of the posterior ciliary arteries such as in optic nerve ischemia (Fig. 8.1) [14], cilioretinal artery occlusion associated with ischemic optic neuropathy [1], appearance or progression of visual field defects in ocular hypertensive patients and normal tension glaucoma [13, 66], and choroidal infarcts [27].

Can all these conditions be explained by the same pathophysiology principle? In 1975, Hayreh [23] explained in detail that partial occlusion of the posterior ciliary arteries (due to any cause) is responsible for the development of AION because they supply the lamina cribosa, the prelaminar, and retrolaminar regions of the optic nerve. Anterior ischemic optic neuropathy, glaucoma, and low-tension glaucoma are manifestations of ischemia of the optic nerve head and retrolaminar optic nerve due to interference with posterior ciliary artery circulation because of an imbalance between perfusion pressure in the posterior ciliary arteries and intraocular pressure. If the process is sudden, then it produces anterior ischemic neuropathy with infarctions of the optic nerve head and retrolaminar region. If the process is chronic (as in glaucoma and low tension glaucoma), it produces slow degeneration of neural tissue in the optic nerve head and retrobulbar region, resulting in cupping of the optic disc and cavernous degeneration of the retrolaminar optic nerve.

When imbalance is produced between perfusion and intraocular pressure, by either lowering perfusion pressure or raising intraocular pressure, the susceptibility of intraocular blood vessels to obliteration varies considerably. The optic disc circulation is the first to compromise, then the peripapillary choroid, and finally the rest of the choroid. This explains the frequent presence of anterior ischemic optic neuropathy without a chorioretinal lesion. However, since a chorioretinal artery arises from a posterior ciliary artery, a cilioretinal artery occlusion may be associated with AION as described in the case reported by Ahmadieh and Javadi [1]. Finally, in AION the visual fields defects can be extremely variable and mimic many ocular and neurologic conditions. In fact, nerve fiber bundle defects with an arcuate scotoma may be seen in AION and simulate a glaucomatous de-

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**Fig. 8.1** A 39-year-old man had loss of vision on day one after bilateral LASIK. He described a hazy quality of vision in both eyes immediately after LASIK. His medical history had no other risk factors for optic neuropathy. Examination revealed a visual acuity of 20/20 in both eyes, normal color vision, increased cupping of the optic nerve, and a relative afferent papillary defect in the right eye (RE). a Fifty-degree fundus photo of the RE shows diffuse loss of retinal nerve fiber layer (RNFL) at the superior pole of the disc and early wedge defects in the RNFL at the inferior pole of the disc (arrows). b Visual field shows a dense inferior nerve fiber bundle–type scotoma and a moderate superior nerve fiber bundle–type scotoma corresponding to the disc and RNFL defects in the RE. c Fifty-degree fundus photo of the left eye (LE) shows diffuse loss of RNFL at the superior pole of the disc and a wedge defect in the inferotemporal RNFL corresponding to the notch in the inferior neuroretinal rim (arrows). d Visual field shows an early inferior nerve fiber bundle–type scotoma corresponding to loss of superior RNFL in the LE (modified and reprinted from [14], with permission from the American Academy of Ophthalmology)