

Corneal Changes after laser in situ keratomileusis(LASIK)

A protocol of an essay

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INTRODUCTION

Laser in situ keratomileusis (LASIK) is the most commonly performed refractive surgery. LASIK is a relatively new procedure that is technically a variation of PRK. The lasik procedure involves the use of microkeratome to create a hinged flap in the cornea underneath which an excimer laser is used to remove micro amounts of stromal tissue to correct the particular refractive error (1).

In laser in situ keratomileusis (LASIK), the photoablation is performed in the corneal mid-stroma, below a hinged tissue-flap. Post-operative problems include a gradual loss of the refractive effect (regression), and complications related to the LASIK-flap. The corneal wound repair seems to play a role for development of these complications (2).

After LASIK, active stromal wound healing in the central cornea results in the production of a hypocellular primitive stromal scar, whereas secondary tissue adjustments seem to cause the Bowman's layer undulations and the subsequent epithelial cell modifications. Most of the interface particles revealed by confocal microscopy in the region of the stromal scar are organic in nature and presumably innocuous to the cornea (3).

Diffuse lamellar keratitis (DLK) usually occurs in the early post-LASIK period. In most cases, it develops within the first month. Late-onset DLK has been reported 2 to 14 months after LASIK associated with trauma, epithelial defects, recurrent corneal erosions and iritis (4).

Permanent pathologic changes were present in all post-LASIK corneas. These changes were most prevalent in the lamellar interface wound. These changes along with other pathologic

alterations in post-LASIK corneas may change the functionality of the cornea after LASIK (5).

Characteristic changes in central corneal morphology were observed during the first 8 weeks after LASIK-scraper. The changes included an initial, massive loss of keratocytes in the anterior stroma followed by gradual repopulation of the acellular region by highly reflecting, migratory fibroblasts. By the third week, repopulation was complete and corneal morphology gradually normalized (6).

Both subbasal and stromal corneal nerves in LASIK flaps recover slowly and do not return to preoperative densities by 3 years after LASIK. The numbers of subbasal nerves appear to decrease between 2 and 3 years after LASIK. The orientation of the regenerated subbasal nerves remains predominantly vertical (7).

Epithelial ingrowth is often detected within 1 month of primary surgery. It can, however, occur in the later stages(8). It has been demonstrated that in the majority of cases, about 90%, ingrowing epithelial cells are continuous with the epithelial cells of the corneal surface, hence the name 'epithelial ingrowth'. The cells may, however, originate from separate cells implanted during surgery. The natural history of ingrowing cells is essentially benign in that 55% disappear with time, 24% diminish and 10% remain unaltered. This observation is supported by histopathological findings (9).

Increased forward shift of the posterior corneal surface is common after myopic LASIK and correlates with the residual corneal thickness and the ablation percentage per total corneal

thickness. An excessively thin residual corneal bed or a large ablation percentage may increase the risk of iatrogenic complications (10).

The change in corneal thickness and curvature affects the estimation of IOP with Goldmann applanation tonometry after excimer laser photorefractive surgery. The amount of reduction in IOP reading might be influenced by the specific laser surgical procedure. This is of clinical importance in the evaluation of any future glaucoma in the increasing number of patients who undergo photorefractive laser surgery (11).

LASIK induces a shift in corneal polarization axis which is responsible for inaccuracies in retinal nerve fibre layer (RNFL) thickness measurements. A customised compensation for corneal polarimetric changes after LASIK allows normalisation some of the thickness parameters (12).

LASIK changed total and higher –order corneal aberrations, these changes in spherical – like aberrations were dependent on the achieved correction (13).

Qualitative and quantitative changes in endothelial cell morphology demonstrate that LASIK does induce an acute effect on the corneal endothelium that may represent transient endothelial cell edema (14).

Aim of the work

The aim of our study To review the literature on corneal changes that may happen after laser in situ keratomileusis (LASIK)

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