CORNEAL ENDOTHELIAL MORPHOLOGY AND BARRIER FUNCTION FOLLOWING EXCIMER LASER PHOTOREFRACTIVE KERATECTOMY

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ABSTRACT

To investigate the effect of deep stromal excimer laser ablation on corneal endothelial morphology and barrier function, excimer laser photorefractive keratectomy (PRK) was performed to obtain residual corneal thickness between 90–250 μm in NZW rabbit corneas (N=50). Corneal endothelium was stained with Alizarin red S for 2 minutes three days after excimer laser ablation, and analyzed morphometrically. Five groups of PRK were performed to obtain three residual corneal thicknesses of 150, 175, and 200 μm in one eye of NZW rabbits (N=30), and also to ablate -6 D and -12 D of correction (N=10). The paired corneas were used as control. Three days after PRK, corneal endothelial permeability was measured according to the method of Watsky et al. and compared to control. Corneas with residual thickness of 90–130 μm showed severe corneal endothelial damage. In 130–200 μm of residual thickness, the damage was inversely proportional to the residual corneal thickness (p<0.05). In corneas of residual thickness over 200 μm, endothelial damages were rarely seen. Corneal endothelial PaC (mean ± SD) three days following -6 and -12 diopter of PRK were 3.21 ± 0.76, and 3.25 ± 1.16 x 10^-4 cm/min which were similar to control (p>0.1). Three days following PRK, corneal endothelial PaC with residual corneal thickness of 200 μm was 3.28 ± 0.55 x 10^-4 cm/min which was also similar to control, whereas PaC with residual thickness of 175 μm and 150 μm were 3.68 ± 0.82, and 3.97 ± 0.58 x 10^-4 cm/min, which were significantly different from control (p<0.05). EM showed an intact monolayer of hexagonal endothelial cells, intact intercellular junctions, and normal subcellular organelles, but amorphous granular material ap-
peared within posterior Descemet's membrane in all excimer laser treated corneas suggesting that the endothelial cells were stimulated to secrete. The results of this study showed that corneal endothelial morphology and barrier function were maintained if ablation level did not go beyond 200 μm of residual corneal thickness. Deeper stromal ablation caused both a morphologic changes and impaired barrier function.

INTRODUCTION

Photorefractive keratectomy (PRK) offers good predictability, efficacy, and safety. However, its potential risk to the corneal endothelium is still unknown. Many studies have been undertaken to assess the effect of excimer laser ablation on the corneal endothelium. Previous animal studies have shown that excimer laser incision could cause endothelial damage if the ablation incision depth was within 40 μm of Descemet's membrane. Other authors have demonstrated that excimer laser keratotomies performed in rabbits could induce endothelial changes such as cellular edema and formation of an incisional ridge, but did not affect the endothelial cell density. Clinically, it has been demonstrated that PRK caused no damage to the corneal endothelium.

Corneal endothelial cells may be affected in various ways by excimer laser ablation. Fluorescence from and scattering of the incident laser beam can result in long wavelength radiation which can be absorbed by deep corneal layers. In addition, acoustic and shock waves are generated by the ultrashort energy pulses and by target recoil as tissue is ablated from the corneal surface. Pulse repetition may result in resonance at the posterior corneal surface. Thus high repetition rates (over 80 Hz) used during excimer laser corneal surgery may cause irreversible damage to the corneal endothelium.

Shallow excimer laser ablation appears to be free of corneal endothelial damage. However, deeper ablation is needed to correct higher myopia. In particular, in excimer laser assisted in situ keratomileusis (LASIK), laser ablation is closer to the corneal endothelium. Endothelial cells close to the photoablative process may be directly or indirectly damaged by the laser beam. Therefore, it is important to quantify the maximal ablation depth at PRK without causing endothelial damage. The purpose of this study was to investigate if deep stromal excimer laser ablation can affect the corneal endothelial cells morphometrically and functionally, and to establish the depth of excimer laser ablation that will not cause the endothelial damage.

MATERIALS AND METHODS

Endothelial Morphology

Fifty New Zealand white rabbits (2–3 kg) were anesthetized intramuscularly with ketamine chloride (5 mg/kg) and xylazine hydrochloride (95 mg/kg). Topical 0.5% proparacaine hydrochloride was instilled and the eyelids were held open with a wire speculum. Central corneal thickness was measured using an ultrasonic pachymeter, and the depth to be ablated was calculated. 193 nm excimer laser (ExciMed UV 200LA, Summit Technology, Inc.; Waltham, MA, USA) photorefractive keratectomy (PRK) was performed randomly to obtain various residual central corneal thicknesses calculated within a range of 90–250μm. The corneal epithelium was not removed before PRK. The repetition rate was 10 Hz, and the pulse energy density was 180 mJ/Cm². The ablation zone diameter