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Individualized Corneal Cross-linking With Riboflavin and UV-A in Ultrathin Corneas: The Sub400 Protocol

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Abstract

Purpose: To determine whether corneal cross-linking (CXL) with individualized fluence ("sub400 protocol") is able to stop keratoconus (KC) progression in ultrathin corneas with 12-month follow-up.

Design: Retrospective, interventional case series.

Methods: Thirty-nine eyes with progressive KC and corneal stromal thicknesses from 214 to 398 μm at the time of ultraviolet irradiation were enrolled. After epithelium removal, ultraviolet irradiation was performed at 3 mW/cm^2 with irradiation times individually adapted to stromal thickness. Pre- and postoperative examinations included corrected distance visual acuity (CDVA), refraction, Scheimpflug, and anterior segment optical coherence tomography imaging up to 12 months after CXL. Outcome measures were arrest of KC progression at 12 months postoperatively and stromal demarcation line (DL) depth.

Results: Thirty-five eyes (90%) showed tomographical stability at 12 months after surgery. No eyes showed signs of endothelial decompensation. A significant correlation was found between DL depth and irradiation time ($r = +0.448$, $P = .004$) but not between DL depth and change in K_{max} ($r = -0.215$, $P = .189$). On average, there was a significant change ($P < .05$) in thinnest stromal thickness ($-14.5 \pm 21.7 \mu\text{m}$), K_{max} ($-2.06 \pm 3.66 \text{ D}$) and densitometry ($+2.00 \pm 2.07 \text{ GSU}$). No significant changes were found in CDVA ($P = .611$), sphere ($P = .077$), or cylinder ($P = .915$).

Conclusions: The "sub400" individualized fluence CXL protocol standardizes the treatment in ultrathin corneas and halted KC progression with a success rate of 90% at 12 months. The sub400 protocol allows for the treatment of corneas as thin as 214 μm of corneal stroma, markedly extending the treatment range. The DL depth did not predict treatment outcome. Hence, the depth is unlikely related to the extent of CXL-induced corneal stiffening but rather to the extent of CXL-induced microstructural changes and wound healing.

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