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Corneal Stromal Thinning and Posterior Irregularity After DMEK: Clinical Observations and Biophysical Hypotheses

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Purpose: The aim of this study was to identify factors associated with significant postoperative stromal thinning in eyes undergoing Descemet membrane endothelial keratoplasty (DMEK).

Methods: This was a retrospective, multicenter interventional study. Eyes that underwent DMEK at Royal Liverpool University Hospital (UK) and ASST Spedali Civili di Brescia (Italy) were included. Eyes were stratified into 2 groups based on the final central corneal thickness (CCT): <500 μm and ≥500 μm. Demographic, clinical, and tomographic parameters were analyzed, including age, preoperative CCT, best corrected visual acuity (BCVA), posterior and total corneal power, and donor endothelial cell density (ECD). Hyperopic shift was defined as an increase of ≥+0.5 D in posterior corneal power or a decrease of ≤−1.0 D in total corneal power.

Results: Among 150 eyes (120 patients), those with a final CCT <500 μm were significantly older (mean [SD], 74.5 [9.9] vs. 68.7 [11.5] years; $P = 0.001$). Hyperopic shift occurred in 43% of eyes with complete tomographic data and correlated with a greater percentage reduction in CCT after DMEK (−25.5% [15.6%] vs. −16.4% [12.4%], $P = 0.02$). A larger proportional CCT reduction was observed in eyes with a final CCT <500 μm and was associated with the presence of preoperative posterior stromal ripples. No significant differences were observed in final BCVA, donor ECD, or treatment center.

Conclusions: Greater reductions in corneal thickness are associated with postoperative stromal thinning and hyperopic shift after DMEK. Preoperative stromal ripples are associated with greater reductions in corneal thickness after DMEK. Stromal remodeling appears influenced by endothelial recovery and preoperative biomechanical status, supporting emerging hypotheses on keratocyte loss and osmotic imbalance.

Key Words: endothelial keratoplasty, DMEK, corneal tomography, corneal edema, posterior stromal ripples

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Introduction

Descemet membrane endothelial keratoplasty (DMEK) has revolutionized the treatment of endothelial dysfunction,[1,2] offering rapid visual rehabilitation, reduced rejection rates, and excellent anatomical outcomes.[3] Despite its benefits, postoperative refractive unpredictability remains a significant clinical challenge, especially in a subset of patients who develop corneal stromal thinning and changes in posterior corneal contour.[4]

Recent studies and clinical experience suggest that the posterior corneal surface undergoes dynamic changes after DMEK surgery.[4–10] This is particularly relevant because posterior corneal irregularity can significantly affect higher order aberrations (HOAs), influencing contrast sensitivity and visual quality, even when best corrected visual acuity appears satisfactory.[4]

In this study, we retrospectively analyzed a multicenter cohort of DMEK patients, identified factors associated with significant stromal thinning, and interpreted our findings through the lens of emerging hypotheses in corneal cell biology and biomechanics.

MATERIALS AND METHODS

Study Design and Setting

This investigation was a retrospective observational study conducted at 2 tertiary referral centers: Royal Liverpool University Hospital (RLUH) in Liverpool, United Kingdom, and Spedali Civili di Brescia (SCB) in Brescia, Italy. The study complied with the principles of the Declaration of Helsinki. Institutional review board approval was obtained at

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both sites (protocol no. NP6210 at SCB and no. 12783 at RLUH), and the requirement for informed consent was waived because of the retrospective design.

Study Population

All patients who underwent uneventful Descemet membrane endothelial keratoplasty (DMEK) and had available preoperative and postoperative anterior segment optical coherence tomography (AS-OCT) scans, central corneal thickness (CCT) measurements, and best-corrected visual acuity (BCVA) data were considered eligible. Patients were excluded if AS-OCT imaging was not available preoperatively or postoperatively, or if they experienced intraoperative or early postoperative complications. These included, but were not limited to, prolonged pupillary block, dislocation of the air or gas bubble, inverted graft placement, complete graft detachment with a mobile graft in the anterior chamber, or a folded graft.

Data Collection and Variables

Clinical and demographic data were extracted from electronic medical records. Variables collected included patient age at surgery, gender, treatment center, and whether a combined DMEK and phacoemulsification procedure was performed. Preoperative data comprised CCT, BCVA (expressed in logMAR), posterior corneal power, total corneal power, and donor endothelial cell density (ECD). Postoperative data included BCVA, CCT, and the presence or absence of posterior stromal ripples (PSRs), which were identified by 2 corneal specialists via evaluation of the posterior corneal profile on AS-OCT images. AS-OCT scans and pachymetric and tomographic measurements were obtained using either the CASIA2 (Tomey Corporation, Nagoya, Japan) or ANTERION (Heidelberg Engineering, Germany) devices.

Outcome Measures and Stratification

For primary analysis, eyes were divided into 2 groups based on the final CCT measured at the last follow-up visit: less than 500 μm and 500 μm or greater.

A subgroup analysis was conducted on eyes with complete corneal tomography to evaluate postoperative refractive changes. A hyperopic shift was defined as either an increase of ≥+0.5 diopters in posterior corneal power or a decrease of ≤ −1.0 diopter in total corneal refractive power after surgery. This threshold of ≥ +0.5 D in posterior corneal power was chosen to remain consistent with previous tomographic studies assessing early postoperative refractive trends after DMEK,[11] in which subtle hyperopic shifts may already be optically significant, even if subclinical at the spectacle plane. A higher threshold would have reduced sensitivity for detecting moderate posterior curvature changes, particularly within our 3-month postoperative data window.[12]

Statistical Analysis

Descriptive statistics were used to summarize baseline demographic and clinical characteristics. Continuous variables were presented as mean ± SD or median (interquartile range [IQR]) as appropriate while categorical variables were presented as counts and percentages.

Independent-sample *t* tests and Pearson chi-squared tests were used as appropriate to assess associations between study groups. Locally estimated scatterplot smoothing (LOESS) curves and their 95% CIs were used to visually represent the longitudinal trends in visual acuity, CCT, and posterior and total corneal power for all eyes included up to 2 years after surgery.

Multivariable logistic regression analyses were conducted to identify factors associated with postoperative corneal thinning and the development of hyperopic refractive shift. Both models included age, gender, donor endothelial cell density, whether a combined phaco-DMEK was performed, indication for DMEK surgery, and the percentage change in central corneal thickness as covariates.

All statistical analyses were performed using R statistical software (version 4.4.0; R Foundation for Statistical Computing, Vienna, Austria). A *P* value of less than 0.05 was considered statistically significant.

RESULTS

Study Population

A total of 150 eyes from 120 patients met the inclusion criteria and were analyzed. Based on the final central corneal thickness (CCT) at the last follow-up, eyes were divided into 2 groups: those with CCT <500 μm (n = 57, 38%) and those with CCT ≥500 μm (n = 93, 62%).

Demographic and Clinical Characteristics

Baseline demographic and clinical details are summarized in Table 1. Patients in the <500 μm group were significantly older at the time of surgery than those in the ≥500 μm group (mean [SD], 74.5 [9.9] vs. 68.7 [11.5] years; $P = 0.001$). There was no difference in the proportion of cases treated at Spedali Civili di Brescia versus Royal Liverpool University Hospital ($P = 0.97$). Combined DMEK-phacoemulsification procedures were more common in the ≥500 μm group (44.1% vs. 24.6%, $P = 0.03$).

No statistically significant differences were observed in gender distribution (70.2% vs. 62.4% female; $P = 0.52$). Preoperative CCT and visual acuity were similar between groups (635.9 [110.2] μm vs. 672.9 [107.2] μm, $P = 0.08$, and 0.7 [0.5] vs. 0.6 [0.5] logMAR, $P = 0.20$, respectively). There were also no clinically significant differences between the 2 groups regarding donor characteristics: age of the donor, as well as the proportion of preloaded and surgeon-stripped grafts, was comparable (all $P > 0.05$), whereas donor ECD was slightly lower in the <500 μm group (mean [SD], 2582.9 [189.6] vs. 2653.7 [199] cells/mm^2, $P = 0.04$).

Longitudinal trends in visual acuity, CCT, and posterior and total corneal power after DMEK surgery are visualized via LOESS curves in Figure 1.

TABLE 1. Demographic Data and Donor Data From Included Eyes

	Final CCT <500	Final CCT ≥ 500	*P*
Eyes, n	57	93	
Female, n (%)	40 (70.2)	58 (62.4)	0.52
Age, mean (SD)	74.5 (9.9)	68.7 (11.5)	0.001
Location, n (%)			0.97
SCB	26 (45.6)	44 (47.3)	
RLUH	31 (54.4)	49 (52.7)	
Indication, n (%)			
FED	37 (64.9)	72 (77.4)	
PBK	12 (21.0)	13 (14.0)	
Failed graft	7 (12.3)	7 (7.5)	
HSV endotheliitis	—	1 (1.1)	
Congenital glaucoma	—	1 (1.1)	
Preoperative logMAR, mean (SD)	0.7 (0.5)	0.6 (0.5)	0.20
Preoperative CCT, mean (SD)	635.9 (110.2)	672.9 (107.2)	0.08
Combined phaco-DMEK, n (%)	14 (24.6)	41 (44.1)	0.03
Months of follow-up, median (IQR)	17.5 (10.4, 23.9)	15.5 (9.1, 21.9)	0.34
Donor data			
Donor age, mean (SD)	66.6 (10.6)	70.6 (7.9)	0.07
Donor ECD, mean (SD)	2582.9 (189.6)	2653.7 (199)	0.04
Preloaded grafts, n (%)	24 (42.1)	41 (44.1)	0.95
Surgeon-stripped grafts, n (%)	33 (57.9)	52 (55.9)	

Factors Influencing Final CCT

Factors correlating with final CCT lower than 500 μm are displayed in Figure 2 (top panel). Other than age at surgery, which was significantly higher in patients with eyes with lower final CCT, there was a small, albeit significant, difference in donor ECD (mean [SD] 2583 [190] vs. 2654 [199] cells/mm^2, $P = 0.03$), unlikely to be clinically relevant. The final best corrected visual acuity also did not differ significantly between groups (mean [SD], 0.16 [0.31] vs. 0.24 [0.43] logMAR, $P = 0.18$).

Absolute and percentage reductions from baseline in CCT after DMEK surgery were both associated with final CCT, with greater reduction in CCT correlating with a final CCT lower than 500 μm.

Factors Influencing Hyperopic Shift

Among eyes with complete posterior and total corneal tomographic data (n = 57), a total of 25 eyes (43%) exhibited a hyperopic shift postoperatively, defined as an increase of ≥+0.5 diopters in posterior corneal power or a decrease of ≤−1.0 diopter in total refractive corneal power. Comparison between eyes with and without a hyperopic shift revealed no significant differences in patient age, donor ECD, preoperative and final BCVA, or final CCT (Fig. 2, bottom panel). However, preoperative CCT was significantly greater in eyes that developed a hyperopic shift (mean [SD], 729.9 [157.7] μm vs. 636.6 [66.2] μm; $P = 0.01$). Absolute and percentage reductions in CCT after surgery were also associated with hyperopic shift, with greater reduction in CCT on average in eyes with hyperopic shift (mean [SD], −205.7 [159.9] vs. −108.0 [83.5], $P = 0.01$ and −25.5% [15.6%] vs. −16.4% [12.4%], $P = 0.02$, respectively). Eyes that underwent a hyperopic shift also had lower preoperative posterior corneal power (mean [SD], −6.5 [1.0] vs. −6.0 [0.5] D, $P = 0.03$) and, as expected, eyes with a hyperopic shift displayed lower final total corneal power (mean [SD], 41.4 [2.0] vs. 43.3 [2.7] D, $P = 0.003$), although there were no significant differences in preoperative total corneal power ($P = 0.76$).

Concordance of Refractive and Tomographic Shifts

Complete preoperative and postoperative subjective manifest refraction was available for 42 eyes. The mean (SD) preoperative spherical equivalent was −0.19 D (1.52 D). The mean (SD) postoperative spherical equivalent was 0.07 D (1.1 D). The mean (SD) change in spherical equivalent was 0.26 D (1.59 D). Of 42 eyes with complete preoperative and postoperative refraction, 14 eyes (33.3%) experienced a spherical equivalent change equal to or greater than +0.50 D.

Of the 42 eyes with preoperative and postoperative refraction, 32 also had complete preoperative and postoperative tomographic data. Of these 32 eyes, 13 (40.6%) did not experience either a subjective refraction hyperopic shift or a hyperopic shift as defined by tomographic criteria (an increase of ≥+0.5 diopters in posterior corneal power, or a decrease of ≤−1.0 diopter in total refractive corneal power). Five eyes (15.6%) had a concordance of both subjective and tomographic hyperopic shifts while 14 (43.7%) eyes experienced only either a tomographic shift (8 eyes [25%]) or a refractive shift (6 eyes [18.8%]).

Odds Ratios for Corneal Thinning and Hyperopic Shift

Odds ratios (ORs) from 2 multivariable logistic models for final CCT <500 μm and for tomographic hyperopic shift are displayed in Figure 3. In the models, a greater percentage decrease in central corneal thickness was the only significant predictor for both outcomes, increasing the odds of postoperative corneal thinning (OR [95% CI], 1.6 [1.1–2.33], $P = 0.02$) and hyperopic shift (OR [95% CI], 1.85 [1.09–3.14], $P = 0.02$). No other covariate was significantly associated with either outcome.

Preoperative Posterior Stromal Ripples

The presence of preoperative PSRs was associated with greater preoperative CCT (mean [SD], 710 [127] vs. 615 [61.9] μm, respectively, $P < 0.001$). The presence of preoperative PSRs was also significantly associated with both absolute and percentage reductions in CCT after DMEK surgery (mean [SD], −85.9 [73.3] vs. −187.8 [-130.7] micron and −13.6% [11.4%] vs. −24.6% [12.2%] micron, both $P < 0.001$). The relationship between preoperative PSRs

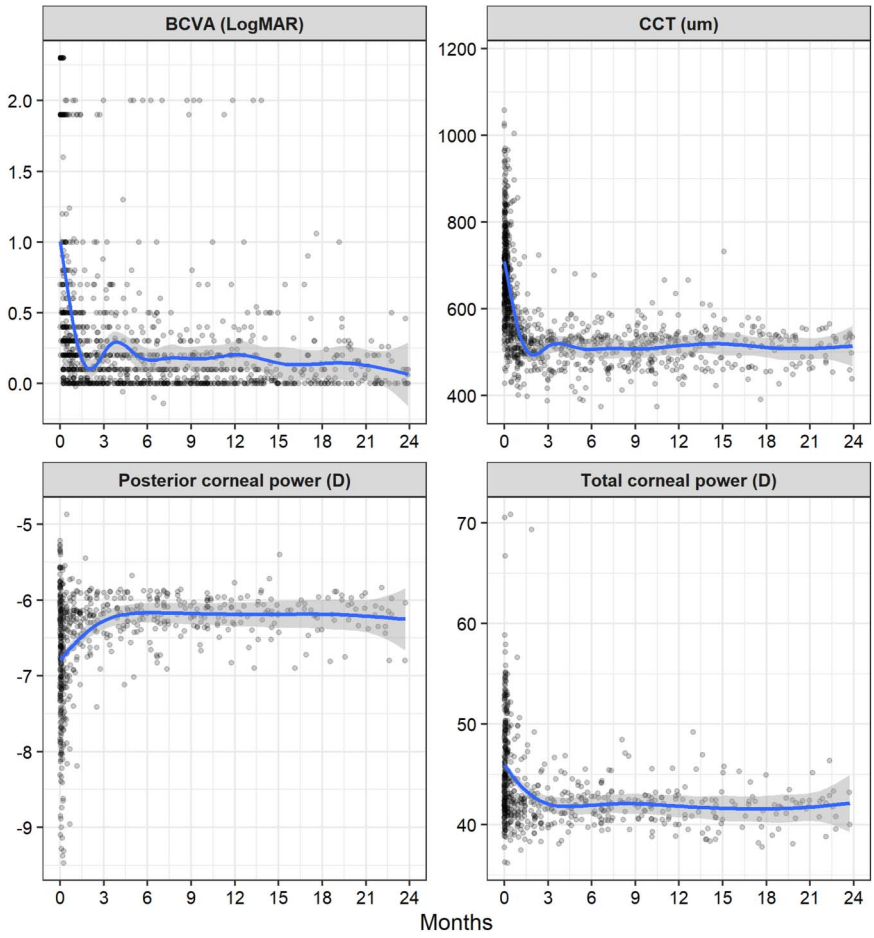

FIGURE 1. LOESS curves displaying longitudinal trends in vision, corneal thickness, and posterior and total corneal power for all eyes included up to 2 years after DMEK surgery. The gray-shaded ribbons represent the 95% CI. Dots represent individual observations.

and preoperative CCT, as well as absolute and percentage reductions in CCT, is displayed in Figure 4.

DISCUSSION

In this study, we found that a significant proportion of eyes undergoing DMEK incurred marked postoperative stromal thinning. In addition, we observed a substantial proportion of patients developing a hyperopic shift, an effect likely tied to both stromal volume changes and alterations in posterior corneal curvature.[12] These structural changes may result in posterior irregularity, which has important optical consequences.[4,10,11,13] Alterations in the back corneal surface can contribute to higher order aberrations that degrade the quality of vision even when Snellen acuity appears normal.[4] The increased variability in posterior corneal power also challenges refractive planning, especially in combined cataract-DMEK procedures.[12,14]

The underlying pathophysiology of corneal thinning after DMEK may extend beyond passive deturgescence. One compelling theory is that keratocyte loss occurs due to osmotic stress during the transient edema phase, likely reflecting transient osmotic imbalance during and after surgery while the donor endothelium reestablishes function. However, keratocyte degeneration may also result from prolonged exposure to chronic stromal edema in Fuchs endothelial dystrophy, which can drive nononcotic pathways such as metabolic stress, mitochondrial dysfunction, or apoptotic-like changes. These chronic alterations may render keratocytes more vulnerable to the acute osmotic shifts occurring during surgery. Thus, chronic and acute osmotic stress likely act cumulatively to drive stromal remodeling and thinning, although only the latter aligns with an oncosis-like mechanism. Oncosis is a form of pathological cell death characterized by cytoplasmic swelling and membrane breakdown.[15–17] The resulting cellular loss, particularly of keratocytes responsible for synthesizing proteoglycans (PGs), could reduce the stromal matrix's capacity to bind water, causing irreversible thinning even after endothelial function is restored.

This hypothesis is further supported by clinical findings and the observed rapid rate of thinning in certain eyes.[15,18,19] If keratocytes undergo oncosis due to prolonged swelling, especially in older or biochemically stressed corneas, the regenerative capacity of the stroma may be compromised. The collapse of fluid-filled stromal "lakes" formed during edema could also contribute mechanically to thinning, potentially producing microstructural damage or even lamellar microtears that weaken the cornea in the long term.[20,21]

Another complementary theory is that variability in endothelial pump function influences stromal homeostasis

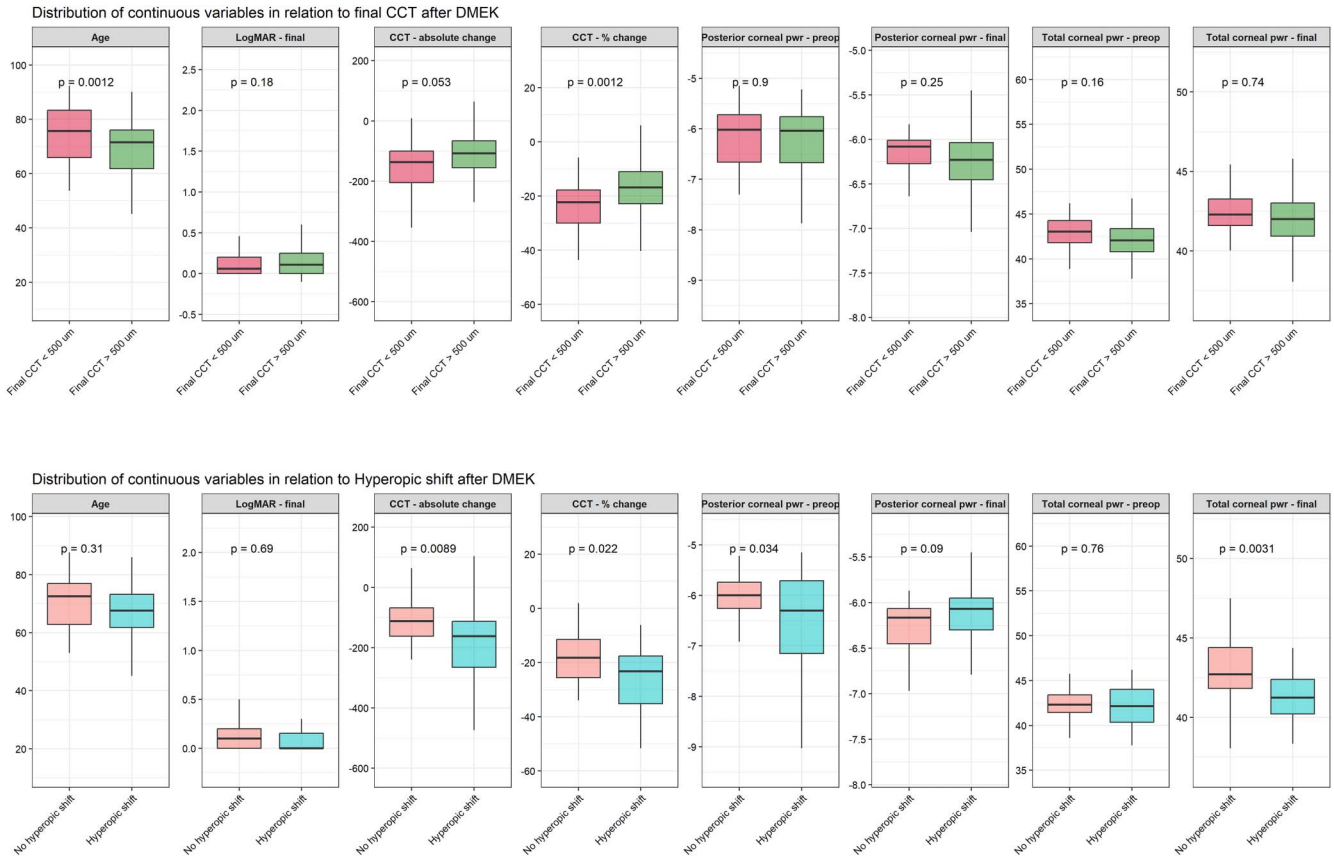

FIGURE 2. Boxplots displaying the distribution of continuous variables in relation to the final CCT lower than 500 μm (top row) and to hyperopic shift (lower row). *P* values from independent *t* tests are also shown in each panel.

differently across patients. Factors such as donor endothelial viability, surgical trauma, inflammation, donor age, and recipient age may all modulate the time it takes for the endothelial pump to fully reestablish osmotic equilibrium. In this transition period, uneven dehydration may occur in some cases, as suggested by the variability in posterior curvature recovery, and may create irregularities on the posterior corneal surface, contributing to posterior HOAs and refractive instability.

A greater hyperopic shift was observed in patients with higher preoperative central corneal thickness (CCT), and preoperative CCT was also significantly higher in those who developed preoperative posterior stromal ripples (PSRs), consistent with our findings of greater preoperative dysfunction in patients exhibiting preoperative PSRs in a previous study, where again PSRs were linked to greater preoperative CCT.[9] These observations support the interpretation that PSRs reflect more advanced endothelial dysfunction and greater preoperative stromal edema.

Although the presence of stromal ripples did not independently correlate with the refractive shift or a thin final CCT, they still represent a potential risk marker for postoperative refractive changes. Specifically, PSRs predicted greater proportional CCT reduction after surgery, likely due to the higher baseline edema that subsequently resolves once endothelial function is restored. In fact, stromal ripples were strongly correlated with both absolute and percentage CCT reductions after DMEK surgery. Absolute and percentage reductions in CCT were, in turn, associated both with a final CCT below 500 μm, with eyes exhibiting greater change being more likely to end up below the 500-μm threshold, and with the chance of experiencing a hyperopic shift after DMEK surgery. Eyes showing greater relative thinning were more likely to have a final CCT below 500 μm and to experience a hyperopic shift, suggesting a sequential relationship from preoperative PSRs to proportional thinning and then to refractive change. Of note, there was no significant difference in preoperative CCT between eyes with a final postoperative CCT lower than 500 μm and those with a final CCT greater than 500 μm. This finding highlights how CCT alone is an unreliable marker of endothelial dysfunction and cannot stratify patients at higher risk of marked stromal remodeling, resulting in low corneal thickness and refractive changes after DMEK. PSRs, therefore, appear to act as surrogate markers of preoperative stromal stress rather than a direct cause of thinning, identifying corneas more likely to undergo greater proportional deturgescence and associated refractive shift after DMEK. Multivariate models confirmed that thinning of the central cornea represented the principal driver of both structural and refractive postoperative changes in this cohort.

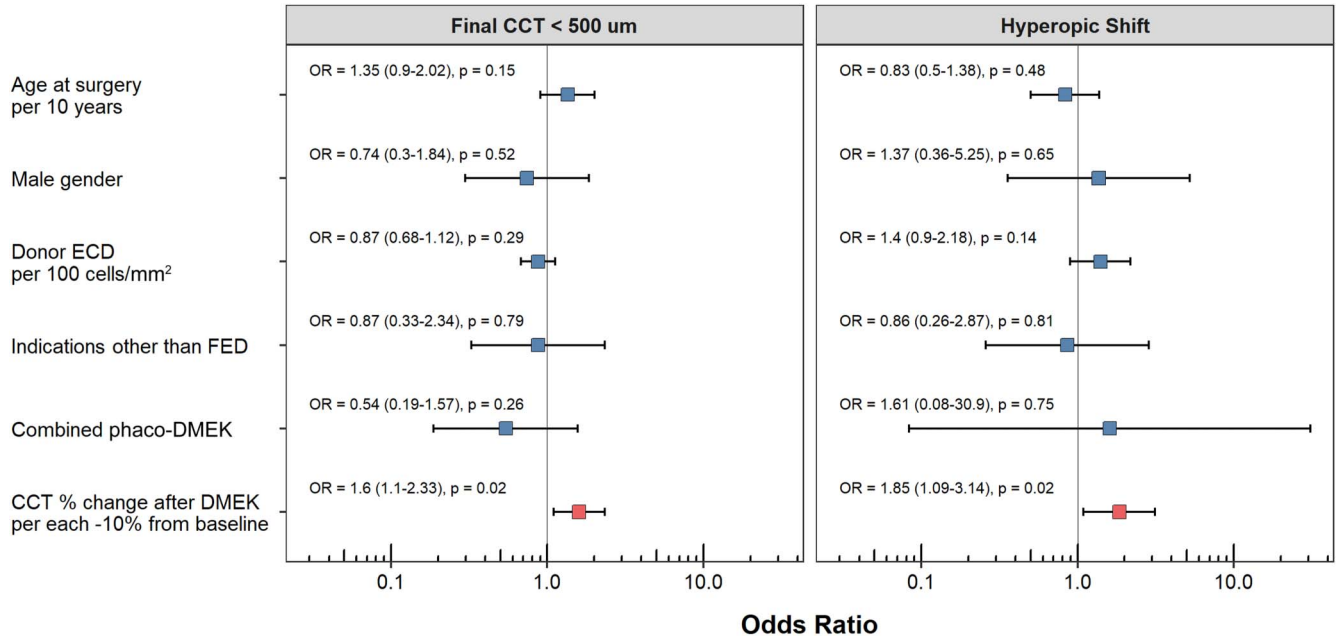

FIGURE 3. Forest plot of odds ratios for final CCT <500 μm (left panel) and for tomographic hyperopic shift (right panel) from a multivariate logistic proportional model. A hyperopic shift was defined as either an increase of ≥+0.5 diopters in posterior corneal power or a decrease of ≤ −1.0 diopter in total corneal refractive power after surgery. Covariates with significant *P* values are highlighted in red.

The clinical implication is that careful monitoring of stromal remodeling after DMEK is essential, especially in older patients or those showing features of marked endothelial dysfunction such as preoperative posterior stromal ripples. Integrating pachymetry and Scheimpflug-based posterior elevation maps in follow-up may help identify at-risk eyes early. From a research standpoint, there is a need for ultrastructural studies of explanted tissue, especially in eyes progressing to PK, to better understand the interaction between stromal cells, extracellular matrix, and endothelial recovery.

This study is limited by its retrospective design and incomplete documentation of preoperative edema duration,

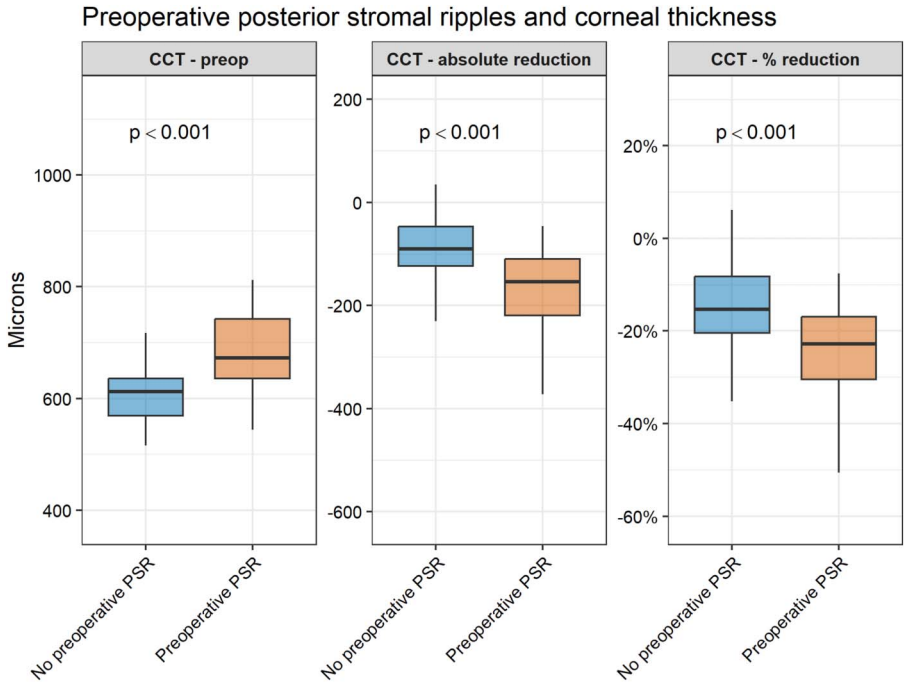

FIGURE 4. Boxplots displaying the distribution of preoperative CCT, absolute reduction in CCT after DMEK at the last visit available, and percentage reduction in CCT after DMEK in relation to the presence of preoperative posterior stromal ripples (PSRs). *P* values from independent *t* tests are also shown in each panel.

which precluded analysis of the impact of chronic stromal swelling on postoperative thinning. Histologic or in vivo confirmation of keratocyte loss and differentiation between oncotic and nononcotic mechanisms were also not possible. Despite these constraints, the consistency of associations across 2 centers supports the reliability of our findings and highlights the need for prospective studies incorporating standardized edema metrics and cellular imaging.

Postoperative corneal thinning and posterior irregularity after DMEK are more common than traditionally appreciated, and these changes likely reflect a combination of cellular loss, extracellular matrix remodeling, and variable endothelial pump recovery. While previous studies have characterized DMEK-induced refractive changes, our study uniquely links qualitative imaging features (posterior stromal ripples) to the quantitative magnitude of stromal remodeling. By integrating tomographic and clinical correlates, we provide a pathophysiological framework connecting edema severity, stromal biomechanics, and postoperative hyperopic shift. Understanding the interplay between these factors is crucial for optimizing refractive outcomes and long-term corneal health in DMEK patients.

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