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LONG-TERM KERATOMETRIC CHANGES AFTER PENETRATING KERATOPLASTY FOR KERATOCONUS AND FUCHS ENDOTHELIAL DYSTROPHY

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Abstract

Purpose—To compare long-term keratometric changes after penetrating keratoplasty (PK) for keratoconus and Fuchs' endothelial dystrophy.

Design—Retrospective, comparative case series

Methods—We retrospectively analyzed 168 corneas after PK for keratoconus (85 eyes of 63 subjects) and Fuchs' dystrophy (83 eyes of 60 subjects). Patients were examined after final suture removal at 12 months after PK to 30 years after surgery. Operations were performed by one surgeon using the same suturing technique in all cases. Eyes were excluded from further analysis after re-grafting or after relaxing incisions. Mean keratometric corneal power and astigmatism were measured by manual keratometry. Data were assessed by using generalized estimating equation models to determine change over time.

Results—Mean keratometric corneal power and astigmatism increased through 30 years after PK for keratoconus (P<.001 and P<.001), but did not change through 20 years after PK for Fuchs dystrophy (P=.55 and P=.55) The change in keratometric corneal power and astigmatism after PK in keratoconus patients only differed from the change in Fuchs dystrophy patients 10 or more years after PK (P=.002 and P=.003).

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- **a.** Design and Conduct of the study (MER, JCE, SVP, WMB)
- **b.** Collection, management, and analysis of data (MER, JCE, SVP, WMB)
- **c.** Interpretation of data (MER, JCE, SVP, WMB)
- **d.** Preparation of manuscript (MER, JCE, SVP, WMB)
- **e.** Review and approval of manuscript (MER, JCE, SVP, WMB)

The study protocol was reviewed and approved by the Institutional Review Board of the Mayo Clinic, Rochester, MN 55905

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Conclusions—Corneal curvature and regular astigmatism increase progressively after PK for keratoconus, but remain stable after PK for Fuchs dystrophy. Our data suggest that keratometric instability after PK for keratoconus is due to delayed, progressive ectasia in the host corneal rim.

INTRODUCTION

Keratoconus is a progressive, noninflammatory ectatic corneal disorder characterized by protrusion of the cornea and thinning of the corneal stroma, without neovascularization.[1] The estimated prevalence of keratoconus ranges from 0.04% to 0.22%.[2,3] Keratoconus typically progresses during the second and third decades of life, although the etiology and pathogenesis of keratoconus is unclear.[4]

The probability of recurrent ectasia after penetrating keratoplasty (PK) for keratoconus is estimated to be 6% – 11% at 20 to 25 years after surgery, based on clinical diagnosis and histopathologic examination. [5–10] The mean latency to recurrent ectasia is generally greater than 10 years, paralleling the natural evolution of keratoconus in adolescence.[8–13] Keratometric studies after PK for keratoconus are mixed, some reporting progression of astigmatism and corneal steepening [14,15] and others unable to detect a change.[16–20]

The purpose of this retrospective study was to measure and compare long15 term keratometric changes after PK for keratoconus and Fuchs dystrophy performed by one surgeon between 1976 and 1986 by using the same surgical technique.

METHODS

The study protocol was reviewed and approved by the Institutional Review Board of the Mayo Clinic. The cohort of our retrospective, comparative case series consisted of 181 consecutive subjects (216 eyes) who had penetrating keratoplasties PKs) for keratoconus and Fuchs dystrophy performed by one surgeon (W.M.B.) between 1976 and 1986. Excluded from the study were 9 subjects who withdrew research authorization, 8 subjects who did not have all sutures removed, and 41 subjects who did not have keratometry recorded or did not have adequate follow-up, leaving 168 eyes of 123 subjects having a PK for keratoconus (85 eyes of 63 subjects) and Fuch' dystrophy (83 eyes of 60 subjects). In the keratoconus group there were 40 (63 %) men and 23 (36 %) women, and age at PK was 36 ± 13 years (mean ± SD, range 16 – 80 years). In the Fuchs dystrophy group there were 6 (10 %) men and 54 (90 %) women, and the mean age at PK was 69 ± 8 years (range, 52 – 92 years). Nine subjects (10 eyes) were followed until re-grafting (6 keratoconus eyes, 1 Fuch' dystrophy eye) or relaxing incisions (3 keratoconus eyes) and thereafter were excluded from analysis.

The surgical technique has been described in detail previously.[21–23] Briefly, a manual trephine of Castroviejo was used to cut the host and donor tissue, with the donor tissue cut from the endothelial side. Donor diameter was 7.9 ± 0.4 mm (mean ± SD; range, 7.25 to 9.5 mm) for keratoconus and 7.9 ± 0.2 mm (range, 7.5 to 8.0 mm) for Fuch' dystrophy. Recipient diameter was 7.6 ± 0.4 mm (range, 7.0 to 9.0) for keratoconus and 7.5 ± 0.1 mm (range, 7.25 to 7.5) for Fuchs dystrophy. Donor buttons were sutured into the recipient by using a double-running technique, which consisted of 12 bites of a single 10-0 nylon suture and 12 bites of a single 11-0 nylon suture. All grafts were centered on the pupil. Postoperatively, the diagnosis of keratoconus and Fuchs dystrophy was confirmed by histopathology. Topical prednisolone acetate 1% was administered from the time of epithelial healing for 3 to 6 months, but rarely more than once daily after the second month. Suture removal for all patients was scheduled at 2 months (10-0 nylon) and 12 months (11-0) after surgery.

Mean keratometric corneal power and astigmatism were measured by using a Bausch & Lomb keratometer (Bausch & Lomb, Rochester, NY). Follow-up examinations were scheduled at 13 months (1 month after final suture removal), and at 5, 10, 15, 20, 25, and 30 years after PK. Follow-up examinations at other visits were included in the analysis if keratometry was recorded. Contact lenses, if worn, were removed immediately before examination. Central corneal thickness was measured by the same contact specular microscope at each examination.[21–23]

All statistical analyses were performed with SAS software (SAS Institute Inc., Cary, NC). Trends over time were investigated by using linear regression models. All models were completed by using generalized estimating equation models to account for the potential correlation within eyes over time and between fellow eyes of the same subject. These models were used to establish trends over time within each of the groups (keratoconus and Fuchs dystrophy) and for testing for differences between groups. Within each group, a linear model that related time to each of the measured parameters was completed. To test for differences between groups, a model was fit with a parameter for time and group, as well as an interaction between the time and group parameters. This modeling investigated keratometry versus time for differences in slope as well as intercept between the groups. Keratometric changes in astigmatism were evaluated by vector analysis and presented as doubled-angle plots.[24]

RESULTS

The Table shows the change in mean keratometric corneal power and astigmatism after PK for keratoconus and Fuchs dystrophy from baseline at 13 months (1 month after final suture removal) up to 30 years after PK. After PK for keratoconus, mean keratometric corneal power and astigmatism increased over the entire study period ($P<.001$ and $P<.001$; Figures 1 – 4). By contrast, after PK for Fuchs dystrophy the mean keratometric corneal power and astigmatism did not change for up to 20 years ($P=.55$; Figures 1 – 4). The increase in mean keratometric corneal power and astigmatism after PK for keratoconus only became significantly different than after PK for Fuchs dystrophy beyond the first decade after surgery ($P=.002$ and $P=.003$; Figures 1, 3).

Figures 5 shows the doubled-angled plot of the vectoral change in magnitude and axis of astigmatism for each subject between 13 months (1-month after final suture removal) and the last follow-up (if at least 15 years after surgery) after PK for keratoconus and Fuchs dystrophy. The cumulative distribution plot in Figure 6 shows the increase in mean keratometric corneal power and astigmatism after PK for keratoconus over the study period.

The mean difference between donor graft diameter and recipient diameter was .26 ± .09 mm in the keratoconus cohort and .43 ± .12 mm in the Fuchs dystrophy cohort. This difference was significant (P>.001), suggesting that more over-sized grafts were present in the Fuchs dystrophy cohort.

Mean central corneal thickness (CCT) was .53 ± .05 mm in keratoconus subjects and .53 ± . 05 mm in Fuchs dystrophy subjects at 1 year after PK. By 20 years after PK, CCT increased to .60 ± .05 mm in keratoconus subjects (P<.001) and .67 ± .07 mm in Fuchs dystrophy subjects (P<.001). The increase in CCT at 20 years after PK was not different between the two cohorts (P=.32)

Five (6 %) of the 85 PKs for keratoconus were eventually diagnosed with recurrent ectasia. The mean latency to diagnosis was 18 years (range, 16 to 19 years). Four (80%) of the 5 PKs had thinning of the recipient rim at the graft-host junction (Figure 7). Four of these 5

eyes had a repeat PK. Histologic characteristics consistent with keratoconus were not detected in any of the 4 donor buttons.

DISCUSSION

Our retrospective, comparative case series suggests a mechanism of delayed, but progressive corneal steepening and increasing regular astigmatism after PK for keratoconus that continues for up to 30 years. When compared to PK for Fuchs dystrophy, the keratometric instability after PK for keratoconus did not become statistically evident until 10 years after surgery. After PK for Fuchs dystrophy, we detected no significant keratometric changes through 20 years after surgery.

Recurrence of ectasia or keratoconus-like characteristics after PK for keratoconus has been reported in several studies.[5–15] Many provide histologic documentation of keratoconus in the donor button.[5–13] The latency to recurrence is generally not until 10 or more years after surgery.[8–15] It is hypothesized that subtle progression of corneal ectasia after PK for keratoconus may manifest itself as change in astigmatism and corneal curvature over time. Clinical studies after PK for keratoconus with follow-up limited to less than 12 years have failed to detect evidence of progressive astigmatism.[16–20] With longer follow-up, increasing regular myopic astigmatism beginning 7 or more years after final suture removal and progressing up to 25 years after surgery has been reported by de Toledo and coworkers[15]. De Toledo attributed their findings to longer follow-up which allowed the natural slow progression of keratoconus in the host corneal rim to progress into significant thinning and keratometric instability. Our long-term findings of keratometric instability after PK for keratoconus are consistent previous studies.[15–20] We were unable to detect a significant difference in keratometric change in the first 10 years when comparing PK for keratoconus with PK for Fuchs dystrophy. Beyond 10 years after surgery, corneal steepening and regular astigmatism progressively increased in the keratoconus cohort, remaining unchanged in the control Fuchs dystrophy cohort.

Although the etiology of late-onset keratometric instability after PK for keratoconus is unclear, our findings provide some insight. We detected no keratometric change after PK for Fuchs dystrophy, which served as the control group. Fuchs dystrophy was treated by PK during the same time period, by the same surgeon, and with the same trephination and suturing techniques as were used for PK to treat keratoconus. Therefore, it is unlikely that a normal age-related structural change in the corneal graft or the double-running suturing technique accounted for the delayed keratometric changes after PK for keratoconus. After final suture removal (13 months after PK), mean central corneal curvature was flatter in keratoconus patients than in Fuch' dystrophy patients. This might be explained by our finding of more over-sized grafts in the Fuchs dystrophy cohort. Alternatively, bulging of the presumably thinner keratoconus recipient rim allows the graft-host junction to steepen, as compared to corneas transplanted in Fuchs dystrophy patients, thus leaving the central cornea less curved.[25]

The etiology of recurrent keratoconus-like characteristics include recurrence of keratoconus in the donor graft, progression of the original disease in the host rim, or grafting with *forme-fruste* keratoconus donor corneas. Histopathologic findings in removed donor corneas many years after PK for keratoconus are sometimes identical to those in primary keratoconus corneas.[7–9,11] There is evidence that host keratocytes replace donor keratocytes after PK,[26] and some investigators have postulated that graft repopulation by recipient keratocytes is responsible for recurrent keratoconus characteristics.[9] Also, host epithelium eventually covers the donor cornea and chronic epithelial-stromal interactions may contribute to the

changes in Bowman's layer that lead to histopathologic findings that are consistent with recurrent keratoconus in the grafted cornea.

A continued natural progression of keratoconus in the host rim may induce thinning of both the host and donor stroma at the graft-host interface leading to wound slippage and progressive keratometric steepening and astigmatism. Lim[27] and de Toledo[15] found thinning of the host cornea 14 and 20 years after PK, and thinning preceded signs of recurrent ectasia. Histopathologic evidence shows that keratoconus changes exist in the peripheral cornea, as well as, the central cornea.[28] Persistent rubbing of the grafted eye in our study's ylounger keratoconus cohort could accelerate wound slippage and keratometric instability.[2,29] Reports of recurrent keratoconus after PK for keratoconus generally do not describe central stromal thinning or cone-like steepening in the graft cornea as seen in primary keratoconus.[9,14] In our study, mean central corneal thickness increased over the entire study period in grafts for both keratoconus and Fuchs' dystrophy. This finding is likely the result of chronic stromal thickening due to progressive endothelial cell loss.[21,23]

It has been suggested that some patients with apparent recurrent keratoconus were transplanted with donor corneas having *forme-fruste* keratoconus.[29] Current estimates of the prevalence of keratoconus in the general population range from 0.04% to 0.22%.[2,3] Although we recognize that the prevalence of *forme-fruste* keratoconus may be higher, the chances of patients receiving donor corneas with *forme-fruste* keratoconus is very small, making it unlikely to account for the estimated 7%–11% recurrence rate after PK for keratoconus.[10] Additional evidence against the donor cornea having *forme-fruste* keratoconus is the lack of reports of recurrent ectasia after PK for non-ectatic corneal disorders. If followed long enough, one would expect a small proportion of PKs for Fuchs dystrophy to develop ectasia if *forme-fruste* corneas were used for transplantation.

Although recurrence of keratoconus in the donor button after PK for keratoconus is well described, our findings are best explained by progression of the disease in the host rim with thinning and secondary structural instability at the graft-host junction. This is corroborated by the absence of clinical features of central keratoconus in the patients in our series and no detectable histopathologic evidence of keratoconus in donor buttons that required repeat PK.

In summary, our long-term data support previous observations that after PK for keratoconus there is delayed-onset keratometric instability that becomes manifest approximately 10 years after surgery and continues to progress for up to 30 years. Delayed refractive instability or recurrent ectasia should be discussed preoperatively as young grafted patients with keratoconus will live long enough to be at risk for this problem, whether they receive a PK or deep anterior lamellar keratoplasty. These findings should also be considered when contemplating further surgical refractive procedures in patients who previously have had PK for keratoconus.

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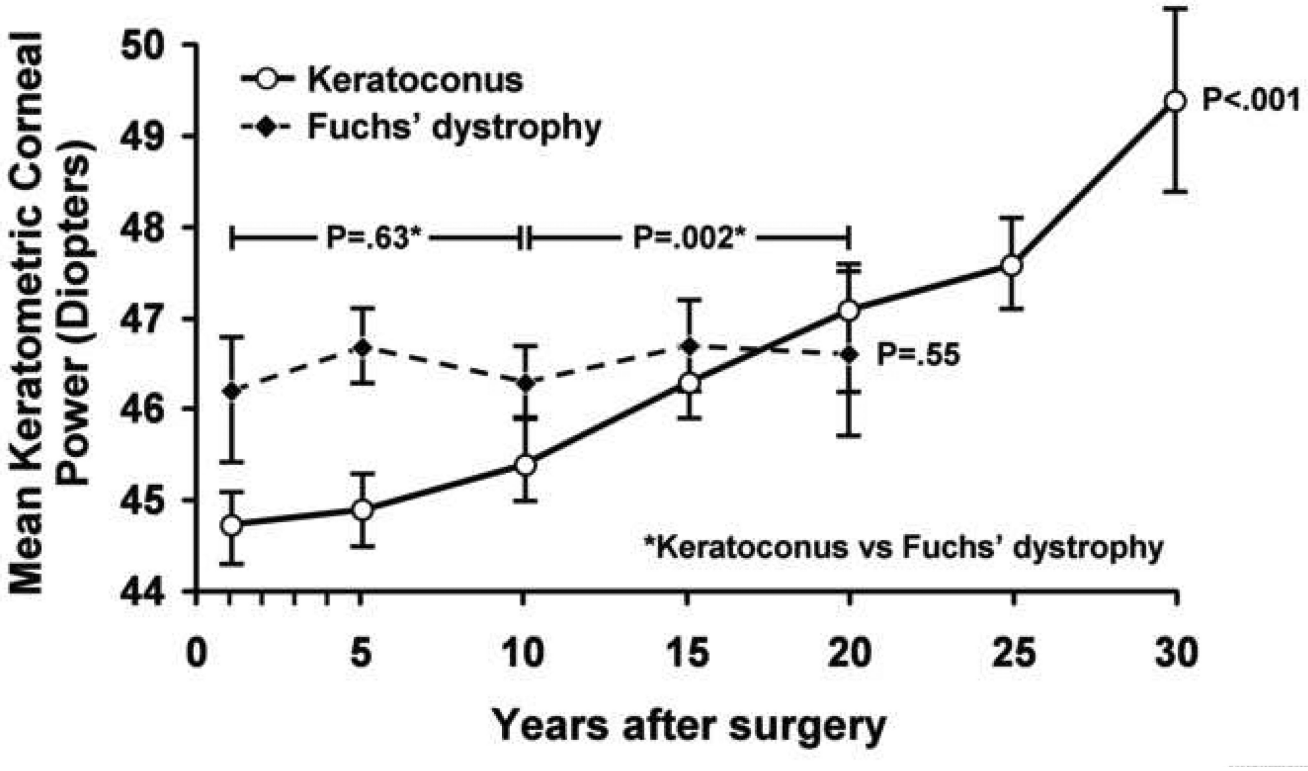


Figure 1.

Change in mean keratometric power (± standard error) after penetrating keratoplasty (PK) for keratoconus and Fuchs dystrophy. Over the study period, mean keratometric corneal power increased after PK for keratoconus (P<.001), but was unchanged after PK for Fuchs dystrophy (P=.55). When comparing keratoconus to Fuchs dystrophy,* the change in mean keratometric corneal power was not different before 10 years after PK (P=.63), but was greater in keratoconus beyond 10 years after PK (P=.002).

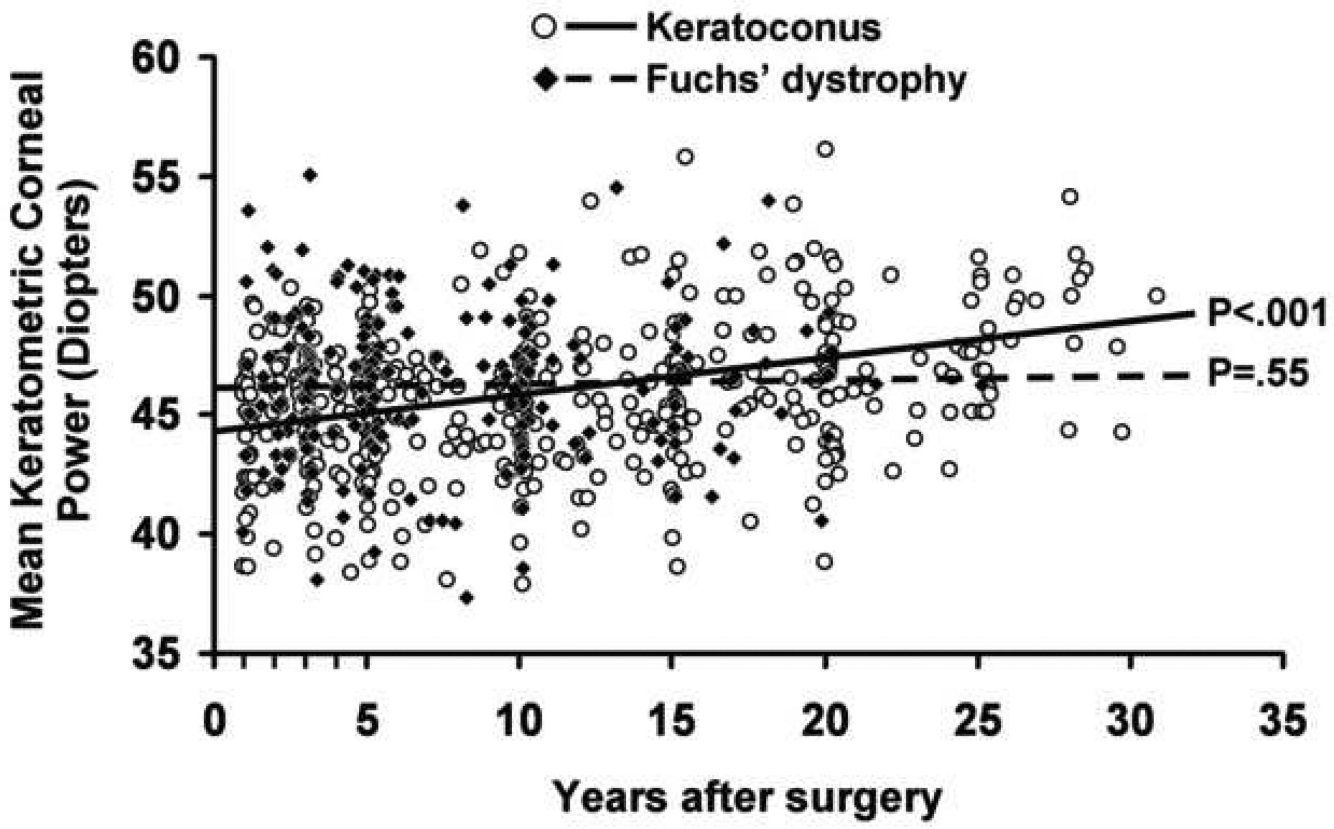


Figure 2.
Scatter-plot of mean keratometric corneal power in all cases at all examinations after penetrating keratoplasty (PK) for keratoconus and Fuchs dystrophy. Mean keratometric corneal power increased after PK for keratoconus (P<.001), but was unchanged after PK for Fuchs dystrophy (P=.55).

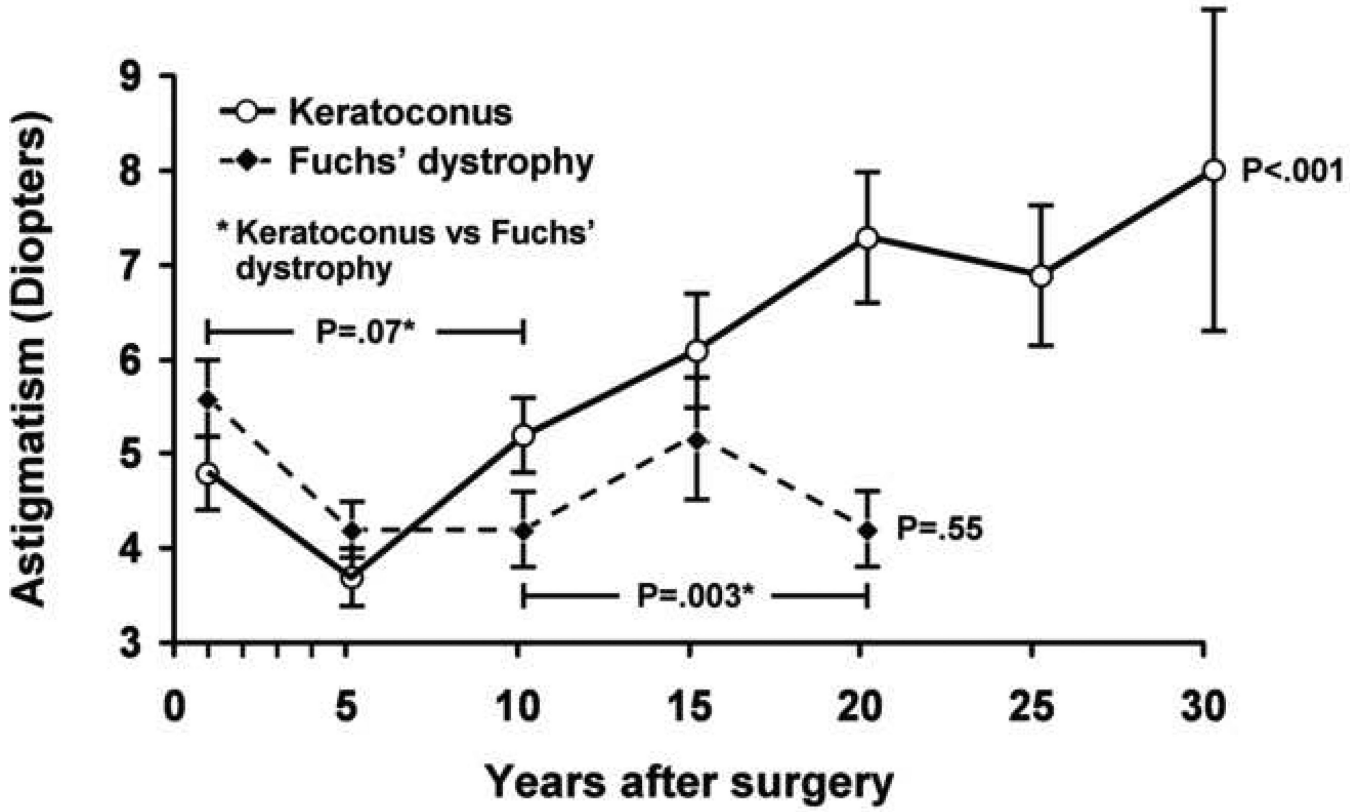


Figure 3.

Change in mean keratometric astigmatism (± standard error) after penetrating keratoplasty (PK) for keratoconus and Fuchs dystrophy. Mean keratometric astigmatism over the study period increased after PK for keratoconus (P<.001), but was unchanged after PK for Fuchs dystrophy (P=.55). When comparing keratoconus to Fuchs dystrophy,* the change in mean keratometric astigmatism was not different before 10 years after PK (P=.07), but was greater in keratoconus beyond 10 years after surgery (P=.003).

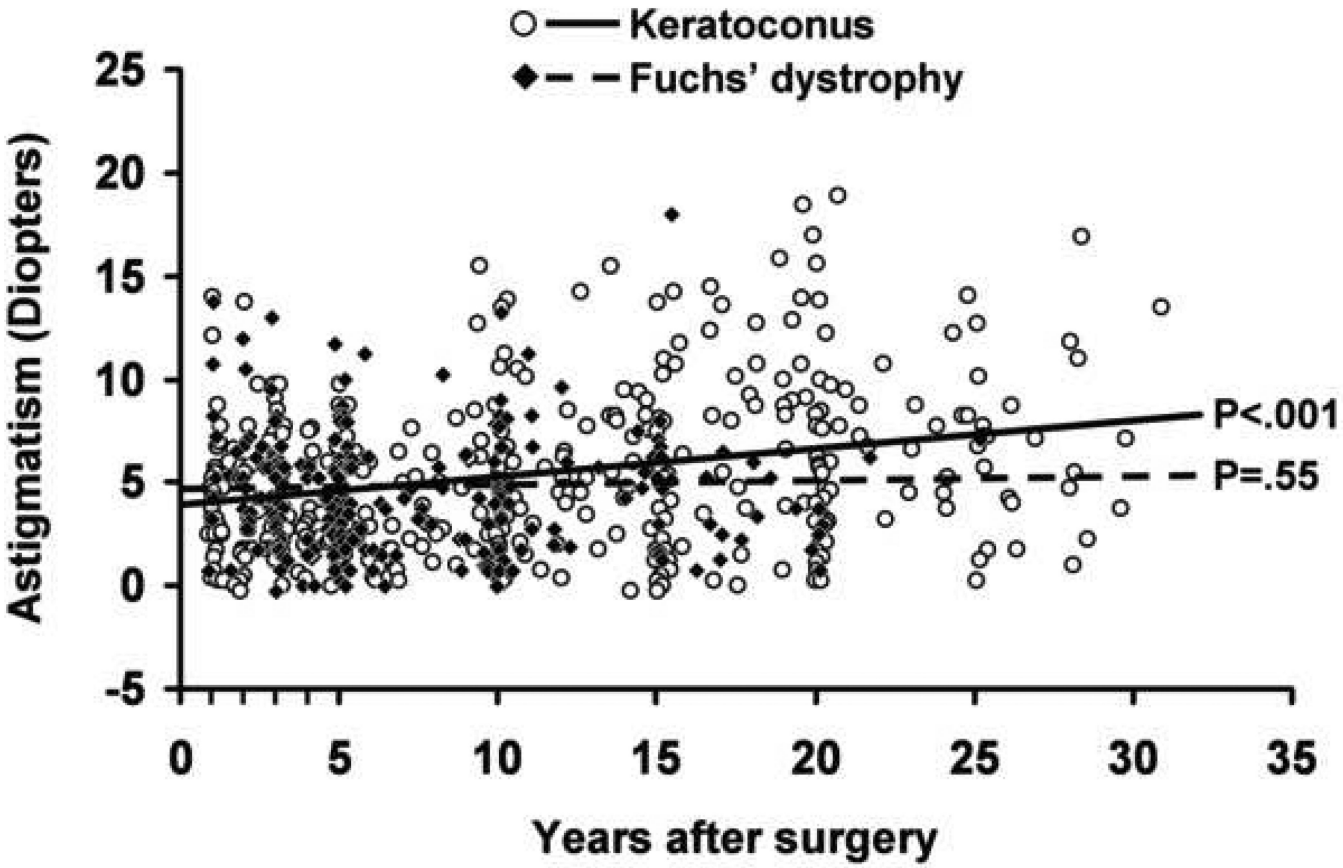


Figure 4.
Scatter-plot of mean keratometric astigmatism in all cases at all examinations after penetrating keratoplasty (PK) for keratoconus and Fuchs dystrophy. Keratometric astigmatism increased after PK for keratoconus ($P<.001$), but was unchanged after PK for Fuchs dystrophy ($P=.55$).

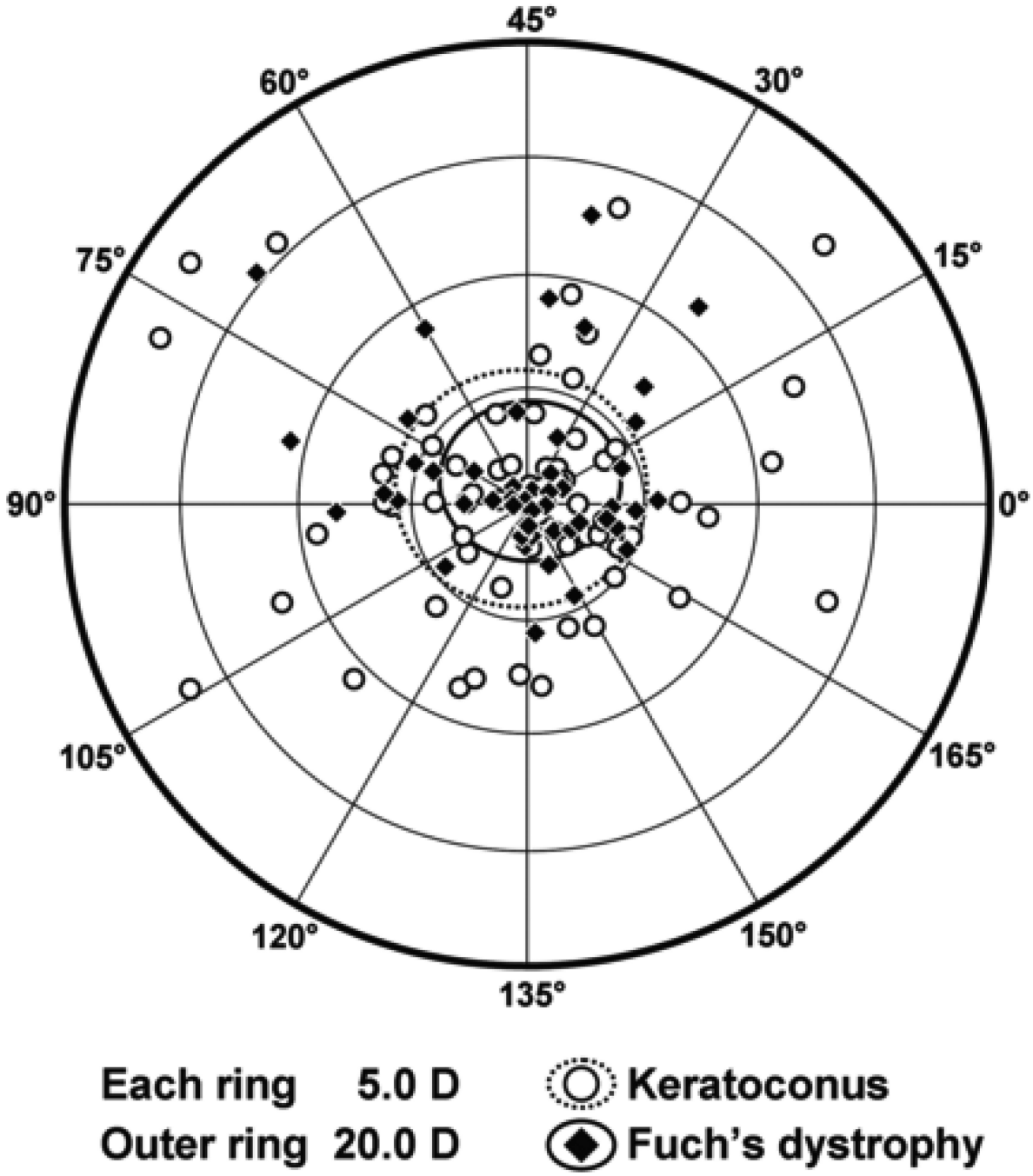
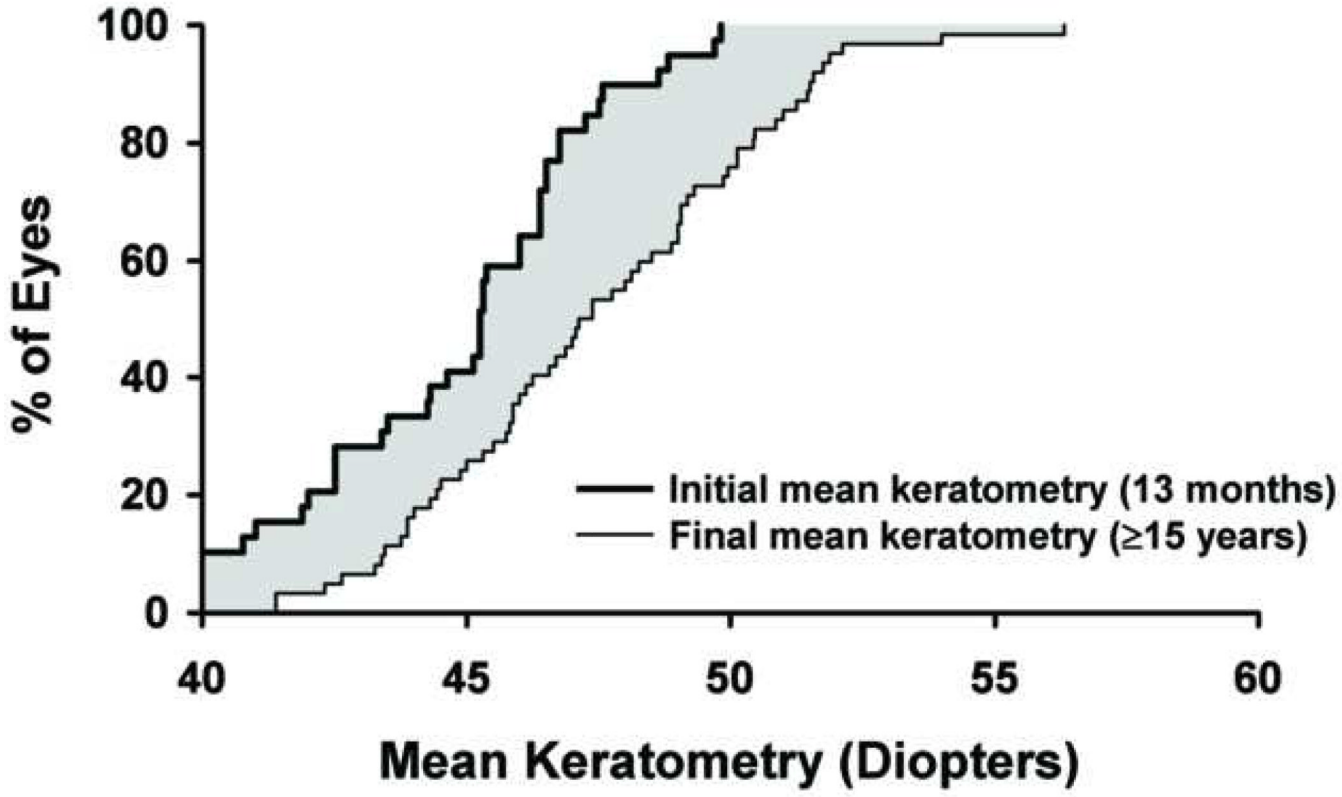


Figure 5.

Doubled-angle plot of the vectoral change (difference vector) observed in the astigmatism magnitude and axis for each subject between 13 months and last follow-up (if at least 15 years) after penetrating keratoplasty for keratoconus and Fuchs dystrophy. The refractive centroid was 0.9 D × 51 ± 5.5 D, and the shape factor was ρ=0.84 for keratoconus patients. The refractive centroid was 1.0 D × 44 ± 3.5 D, and the shape factor was ρ=0.83 for Fuchs dystrophy patients. This suggests no difference in astigmatic shift in any given axis direction when comparing PK for keratoconus with PK for Fuchs dystrophy.



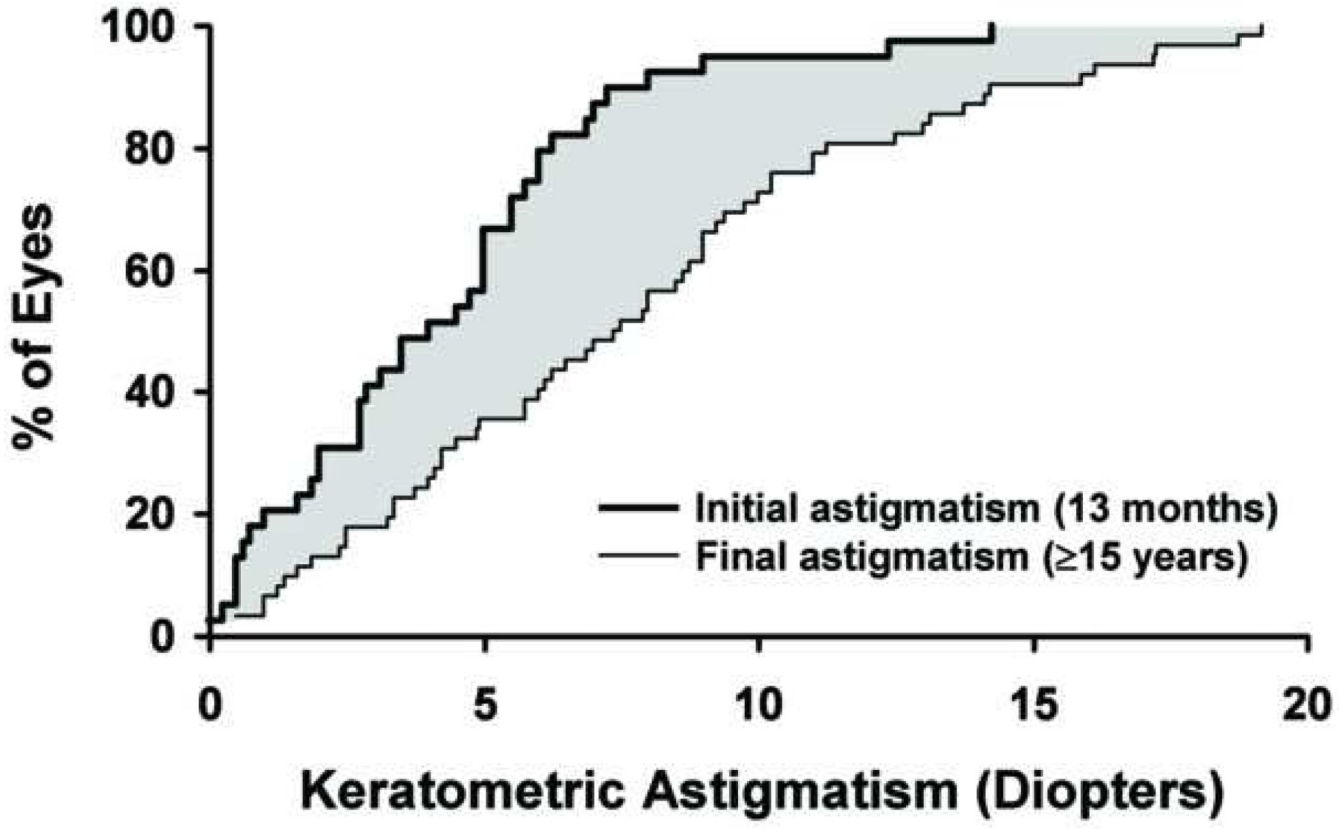


Figure 6.
Cumulative frequency distribution of (left) mean keratometric corneal power and (right) astigmatism at 1 month after suture removal (13 months after PK) and at last follow-up (if at least 15 years) after penetrating keratoplasty for keratoconus. The gray area between the two stepped lines represents the increase in postoperative corneal steepening or astigmatism.

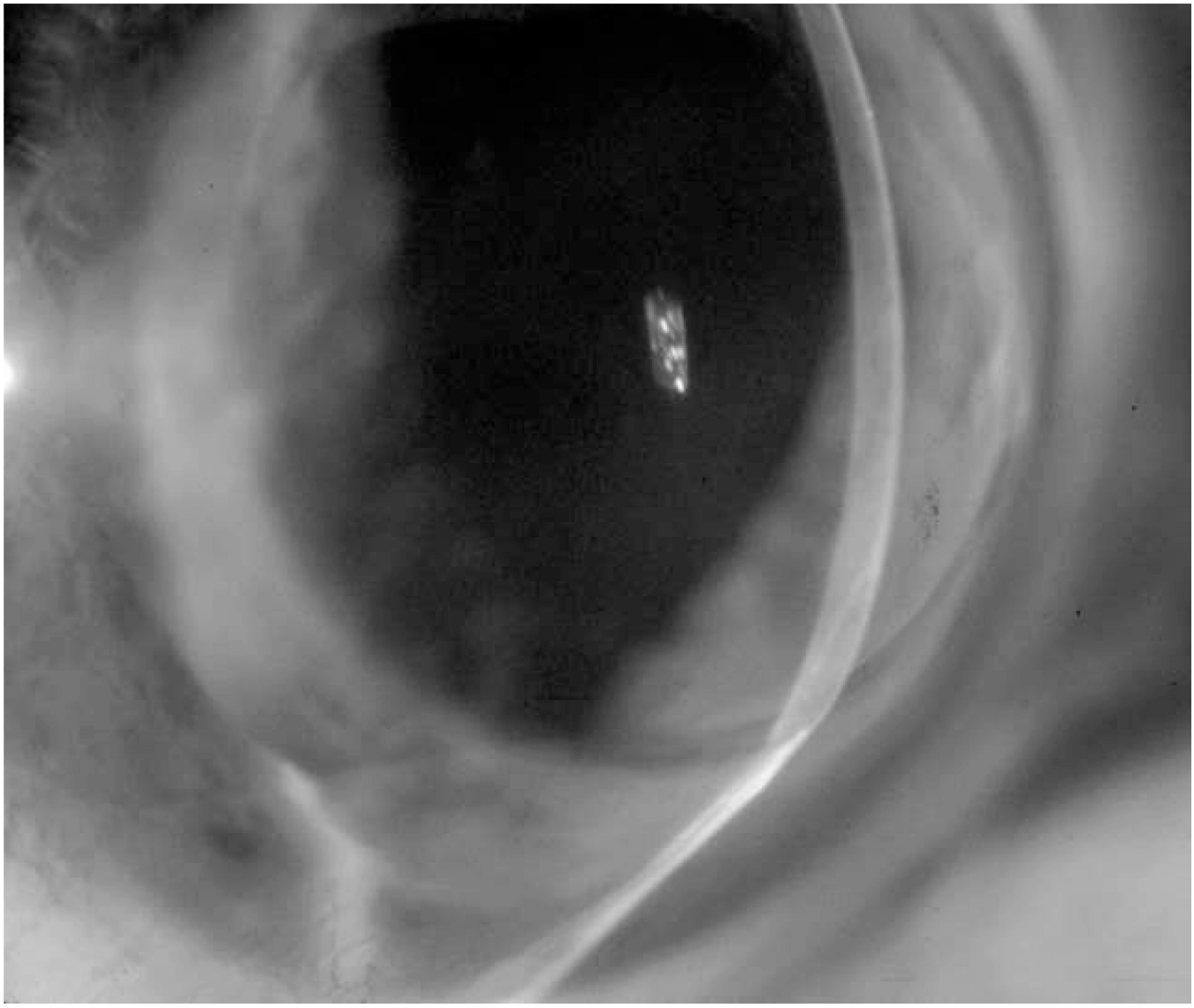


Figure 7.
Slit-lamp photograph showing thinning of the recipient rim at the inferior graft host junction 18 years after penetrating keratoplasty for keratoconus.

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Table

Mean Keratometric Corneal Power and Astigmatism (Diopters, mean ± standard deviation) after Penetrating Keratoplasty for Keratoconus and Fuchs' Dystrophy

	Time after Penetrating Keratoplasty							
	13 month	**5 years**	**10 years**	**15 years**	**20 years**	**25 years**	**30 years**	**P***
Keratometric Power								
Keratoconus	44.8±2.8	44.9±2.7	45.4±2.9	46.2±3.3	47.1±3.6	47.6±2.3	49.4±3.1	<.001
	(n=46)	(n=57)	(n=67)	(n=53)	(n=50)	(n=24)	(n=10)	
Fuchs' Dystrophy	46.1±4.1	46.7±2.9	46.3± 3.2	46.7±2.9	46.6±3.3	-	-	.55
	(n=39)	(n=58)	(n=52)	(n=28)	(n=23)			
Keratometric Cylinder								
Keratoconus	4.7±2.9	3.7±2.4	5.3±3.5	6.1±4.2	7.3±4.8	6.9±3.6	8.0±5.3	<.001
	(n=46)	(n=57)	(n=67)	(n=53)	(n=50)	(n=24)	(n=10)	
Fuchs' Dystrophy	5.6±2.9	4.3±2.6	4.4±3.0	5.3±3.4	4.2±2.2	-	-	.55
	(n=39)	(n=58)	(n=52)	(n=28)	(n=23)			

*Over entire study period, Generalized Estimating Equation Model