Total corneal astigmatism magnitude and vector orientation in diabetic and non-diabetic Malay eyes

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ABSTRACT

Introduction: The influence of diabetes mellitus (DM) on total corneal astigmatism (TCA) remains incompletely understood. Using total keratometry (TK), this study characterised TCA magnitude and orientation in a Malay adult population and evaluated the influence of metabolic control and ocular parameters on TCA.

Materials and Methods: This cross-sectional study analysed 190 eyes (88 non-diabetic; 102 diabetic). TCA magnitude and axis were derived from TK obtained using swept-source optical coherence tomography (IOLMaster 700, Carl Zeiss Meditec, Germany) and decomposed into power-vector components; J0 (horizontal/vertical) and J45 (oblique). Corneal endothelial parameters were measured using specular microscopy (Topcon SP-1P, Japan). Group comparisons were performed using Welch's t-test. Within diabetic eyes, linear regression models identified independent predictors of TCA magnitude and orientation, adjusting for age, glycated haemoglobin (HbA1c), DM duration, cumulative metformin exposure, central corneal thickness, endothelial cell density, and white-to-white diameter.

Results: TCA magnitude did not differ significantly between diabetic and non-diabetic eyes (p = 0.066). Vector analysis demonstrated no significant between-group difference in J0 or J45, with substantial vector centroid overlap. In diabetic eyes, higher HbA1c was independently associated with greater TCA magnitude, while increasing age was independently associated with a shift towards more negative J0 values. DM duration and metformin exposure were not independently associated with TCA magnitude or vector components.

Conclusion: Diabetes status alone was not associated with systematic differences in TCA magnitude or orientation. Age and metabolic control were the strongest factors associated with TCA characteristics. Vector-based analysis provides a robust framework for astigmatism assessment in diabetic and non-diabetic eyes.

KEYWORDS:
Astigmatism, cornea, diabetes mellitus, metformin, keratometry, Malay population

INTRODUCTION

Diabetes mellitus (DM) is a systemic metabolic disorder that affects multiple ocular tissues, including the cornea, where it induces structural and biomechanical alterations involving endothelial cell density, stromal hydration, and connective tissue integrity.[1] This is particularly relevant in the Malay population, in whom the pooled prevalence of DM has been estimated at 15.25%, exceeding the Malaysian national average of 14.39%.[2] Such diabetes-related corneal changes may influence corneal curvature and astigmatism, with important implications for visual quality, refractive predictability, and surgical planning.[1,3-4]

Corneal astigmatism in diabetic eyes has predominantly been evaluated using anterior keratometry and conventional cylinder-axis notation.[1,5] However, these approaches incompletely characterise corneal optics, as they do not account for the posterior corneal curvature, which has been shown to meaningfully influence total corneal astigmatism (TCA).[5] The advent of swept-source optical coherence tomography (SS-OCT) has enabled measurement of total keratometry (TK), providing a more physiologically accurate representation of corneal astigmatism by incorporating both anterior and posterior corneal surfaces.[6-7]

Despite these advances, the relationship between diabetes and TCA remains incompletely defined. Prior studies have reported variable findings, with limited data specifically addressing astigmatism behaviour in diabetic populations and inconsistencies arising from differences in disease severity, metabolic control, and methodological approaches.[1,4] Moreover, most prior investigations have relied on separate analyses of astigmatism magnitude and orientation, without adopting vector-based methods.

Vector representation of astigmatism, first formalised by Thibos et al., is now widely recognised as the preferred framework for quantitative astigmatism analysis. This approach decomposes astigmatic error into two independent Cartesian components (J0 and J45), enabling magnitude and orientation to be analysed as continuous variables while avoiding axis-wrapping artefacts inherent to polar representations. Consequently, vector analysis permits valid averaging, hypothesis testing, and parametric modelling of astigmatism data.[8-9]

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In addition, data examining the relationship between metabolic control, DM duration, cumulative metformin exposure, and corneal structural parameters such as central corneal thickness (CCT), endothelial cell density (ECD), and white-to-white diameter (WTW) with TCA magnitude and vector components remain limited, particularly in Malay populations.

Therefore, this study aimed to compare TCA magnitude and orientation between diabetic and non-diabetic eyes in a Malay adult population, and to determine whether systemic factors (age, DM duration, glycated haemoglobin [HbA1c], and cumulative metformin exposure) and ocular parameters (CCT, ECD, and WTW) are independently associated with TCA characteristics within diabetic eyes using a vector-based analytical framework.

MATERIALS AND METHODS

Study Design and Population

Participants were recruited from patients undergoing routine ophthalmic assessment at the Ophthalmology Clinic, Hospital Sultan Zainal Abidin (HoSZA) between January and June 2025. Eligible participants were Malay adults aged ≥18 years with reliable SS-OCT biometry, ECD measurements, and complete systemic data. Exclusion criteria included corneal pathology, previous corneal or intraocular surgery, ocular trauma, and media opacity that could compromise measurement quality. Only right eyes were included to avoid inter-eye correlation.

Demography, Systemic and Ocular Variables

A total of 190 participants were included in the analysis, comprising 88 non-diabetic individuals and 102 patients with DM. Diabetes status was determined based on documented clinical diagnosis, supported by random blood glucose and HbA1c measurements, obtained from the HoSZA General Medical Clinic or affiliated primary healthcare clinics.

Demographic variables included age and sex. Diabetes-specific variables comprised DM duration in years, cross-referenced using medical records and patient self-report. Only HbA1c values obtained within three months of ophthalmic assessment were included. Cumulative metformin exposure was calculated as the total prescribed metformin dose (mg) since treatment initiation, based on medical records and patient-reported adherence. Information regarding other diabetic treatment modalities, including insulin or combination therapy, was inconsistently documented in shared medical records, particularly for participants co-managed in primary healthcare clinics. Consequently, other treatment modalities could not be reliably stratified and were not included as a covariate in the analysis.

Ocular parameters included TCA, derived from TK measurement obtained using SS-OCT biometry with the IOLMaster 700 (Carl Zeiss Meditec, Germany). The device utilises telecentric keratometry with a 950-nm light source and acquires measurements along the visual axis by projecting 18 measurement points across three corneal zones (1.5, 2.5, and 3.5 mm), allowing calculation of the flattest and steepest keratometry values and TK. Measurements were accepted only when the device quality indicator signalled successful acquisition. Participants were instructed to fixate on the internal target and to blink immediately before each measurement to ensure tear-film stability. CCT and WTW were obtained during the same measurement session.[10]

ECD was measured using a non-contact Topcon SP-1P specular microscope (Topcon, Japan). Images were automatically centred, focused, and captured from the central, nasal and temporal cornea with full corneal exposure. All measurements were performed by a single trained operator.

Astigmatism Vector Analysis

TCA was expressed as magnitude in dioptres (D) and decomposed into power-vector components using Thibos notation.11 The horizontal/vertical component was defined as $J0 = -(C/2) \times \cos(2\alpha)$, representing astigmatism along the 0°/90° meridians, corresponding to with-the-rule (WTR) and against-the-rule (ATR) orientation. The oblique component was defined as $J45 = -(C/2) \times \sin(2\alpha)$. In these expressions, C denotes cylinder power and α denotes astigmatism axis in degrees.

Data Analysis

All statistical analyses were performed using Jamovi (version 2.6.26), an open-source statistical platform built on the R statistical computing environment (The jamovi project, 2025, https://jamovi.org). Statistical significance was defined as $p < 0.05$. All statistical tests were two-sided.

Continuous variables were assessed for normality using the Shapiro-Wilk test and visual inspection of histograms. Descriptive statistics are presented as mean ± standard deviation for approximately normally distributed variables and median with interquartile range (IQR) for non-normally distributed variables.

Comparisons between diabetic and non-diabetic eyes were performed using Welch's independent-samples t-test, selected a priori to account for unequal sample sizes and potential variance heterogeneity. Categorical variables were analysed using the χ^2 test of independence.

Within diabetic eyes, simple linear regression was initially used to screen associations between clinical variables and TCA magnitude and vector components (J0, J45). Variables of clinical relevance were subsequently entered into multiple linear regression models to identify independent predictors, while adjusting for age, sex, HbA1c, DM duration, cumulative metformin exposure, CCT, ECD, and WTW. Regression assumptions including linearity, homoscedasticity, normality of residuals, and absence of multicollinearity were verified. Model performance was assessed using the coefficient of determination (R^2). Due to a right-skewed distribution, cumulative metformin exposure was log-transformed prior to regression analysis to improve homoscedasticity.

Double-angle vector plots of J0 and J45 were generated using the R Editor module in Jamovi to visualise astigmatism orientation distributions and centroids.

Ethics Approval

This cross-sectional observational study was approved by the Universiti Sultan Zainal Abidin Human Research Ethics Committee (UniSZA/UHREC/2024/666) and supported by a DPU 1.0 Grant (UniSZA/2023/DPU1.0/42[RD053]). The study was conducted in accordance with the tenets of the Declaration of Helsinki.

RESULTS

Study Population and Baseline Characteristics

A total of 190 right eyes from a Malay cohort were analysed, comprising 88 non-diabetic eyes (46.3%) and 102 diabetic eyes (53.7%). The overall mean age was 69.30 ± 8.50 years (range 38 - 87 years), with no significant difference in age between groups (p = 0.551), confirming that the cohorts were well-balanced for age-related corneal analysis.

Baseline demographic and ocular characteristics are summarised in Table I. The two groups were comparable in age, sex distribution, ECD, and WTW. Diabetic eyes demonstrated significantly thinner CCT compared with non-diabetic eyes (p = 0.026). Diabetes-specific variables, including DM duration, HbA1c, and cumulative metformin exposure, were further summarised descriptively within the diabetic cohort only.

TCA Magnitude and Orientation Profile

TCA magnitude demonstrated a trend towards lower values in diabetic eyes compared with non-diabetic eyes; however, this difference did not reach statistical significance (p = 0.066).

Vector-based analysis revealed negative mean J0 values in both diabetic and non-diabetic eyes, indicating a predominance of ATR astigmatism in this predominantly older population. Mean J45 values were also negative in both groups, reflecting centroids oriented towards the 135° oblique meridian.

No statistically significant differences were observed between diabetic and non-diabetic eyes for either horizontal/vertical astigmatism orientation (J0) or oblique orientation (J45) component (p = 0.960 and p = 0.590, respectively), indicating no group-level association between diabetes status and TCA orientation.

Relationship Between Clinical Variables and TCA Magnitude in Diabetic Eyes

Within diabetic eyes, simple linear regression identified several variables associated with TCA magnitude. After multivariable adjustment, increasing age and higher HbA1c levels remained independently associated with greater TCA magnitude.

Variables that were significant in crude analysis, including DM duration and ECD, were no longer significant after adjustment, suggesting that their initial associations were

Table I: Demographic Profile, Diabetic and Ocular Parameters (N=190)

Variables	Non-Diabetics (n=88)	Diabetics (n=102)	p value
Age (years), mean ± SD	69.70 ± 9.60	69.00 ± 7.50	0.551
Sex, n (%)			0.120
Male	47 (53.4)	43 (42.2)	
Female	41 (46.6)	59 (57.8)	
TCA (TK) Magnitude (D), mean ± SD	1.21 ± 0.66	1.04 ± 0.63	0.066
Vector J0 (D), mean ± SD	–0.22 ± 0.59	–0.22 ± 0.50	0.960
Vector J45 (D), mean ± SD	–0.02 ± 0.29	–0.04 ± 0.27	0.590
CCT (µm), mean ± SD	531.20 ± 36.95	519.70 ± 33.63	**0.026**
ECD (cells/mm²), mean ± SD	2683.00 ± 521.00	2627.00 ± 458.00	0.430
WTW (mm), mean ± SD	12.00 ± 0.37	11.90 ± 0.39	0.220
DM duration (years), median (IQR)	-	9.00 (5.00-14.80)	-
HbA1c (%), mean ± SD	-	7.90 ± 1.80	-
Cumulative metformin dose (mg), median (IQR) (3.88–12.50 × 10^6)	-	7.31 × 10^6	
Log cumulative metformin dose, mean ± SD	-	13.10 ± 5.90	-

Statistical significance was defined as $p < 0.05$

Values are presented as mean ± SD or median (IQR), as appropriate. Continuous variables between non-diabetic and diabetic groups were compared using Welch's independent-samples t-test. Sex distribution was compared using the χ^2 test. DM-specific variables were summarised descriptively within the diabetic group only

Abbreviations: DM= Diabetes mellitus, HbA1c = glycated haemoglobin, IQR = Interquartile range, TCA = Total corneal astigmatism, TK = Total keratometry, D = Dioptres, CCT = Central corneal thickness, ECD = Endothelial cell density, WTW = White-to-white corneal diameter

Table II: Relationship of Clinical Variables with TCA Magnitude

TCA Magnitude (D)	Simple Linear Regression		Multiple Linear Regression	
(n=102)	Crude b[a] 95% CI	p value	Adj. b[b] 95% CI	p value
Age (years)	0.035 (0.020, 0.050)	**<0.001**	0.033 (0.017, 0.049)	**<0.001**
Sex (M-F)	–0.041 (–0.290, 0.209)	0.746		
DM duration (years)	0.002 (0.0004, 0.0039)	**0.016**		
HbA1c (%)	0.084 (0.0069, 0.161)	**0.035**	0.084 (0.007, 0.161)	**0.033**
Log metformin cumulative dose	–0.007 (–0.028, 0.014)	0.502		
CCT (µm)	–0.002 (–0.0059, 0.0014)	0.227		
ECD (cells/mm²)	0.00029 (0.00002, 0.00055)	**0.034**		
WTW (mm)	–0.301 (–0.611, 0.010)	0.057		

[a]Crude regression coefficient
[b]Adjusted regression coefficient; Model R^2 = 28.6%, Adjusted R^2 = 22.4%
Linear regression assumptions, including linearity and homoscedasticity, were met
Statistical significance defined as $p < 0.05$
Abbreviations: TCA = Total Corneal Astigmatism, D = Dioptres, DM= Diabetes mellitus, HbA1c = glycated haemoglobin, CCT = Central corneal thickness, ECD = Endothelial cell density, WTW = White-to-white corneal diameter

Table III: Relationship of Variables with J0 Orientation

J0 Astigmatism Component (D)	Simple Linear Regression		Multiple Linear Regression	
(n=102)	Crude b[a] 95% CI	p value	Adj. b[b] 95% CI	p value
Age (years)	–0.019 (–0.031, –0.006)	**0.004**	-0.022 (-0.035, -0.008)	**0.002**
Sex (M-F)	–0.041 (–0.290, 0.209)	0.746		
DM Duration (years)	–0.00025 (–0.0017, 0.0012)	0.725		
HbA1c (%)	0.031 (–0.032, 0.095)	0.329		
Log metformin cumulative dose	–0.009 (–0.026, 0.007)	0.262		
CCT (µm)	–0.002 (–0.005, 0.001)	0.134		
ECD (cells/mm²)	0.00003 (–0.00017, 0.00024)	0.807		
WTW (mm)	0.284 (0.039, 0.530)	**0.024**		

[a]Crude regression coefficient
[b]Adjusted regression coefficient; Model R^2 = 17.2%, Adjusted R^2 = 10.0%
Linear regression assumptions, including linearity and homoscedasticity, were met
Statistical significance defined as $p < 0.05$
Abbreviations: D = Dioptres, DM= Diabetes mellitus, HbA1c = glycated haemoglobin, CCT = Central corneal thickness, ECD = Endothelial cell density, WTW = White-to-white corneal diameter

attributable to confounding rather than independent effects. Cumulative metformin exposure was not associated with TCA magnitude in either crude or adjusted models. The final multivariable model explained approximately one-quarter of the variance in TCA magnitude (adjusted R^2 = 22.4%), consistent with the multifactorial nature of corneal astigmatism. Detailed regression results are outlined in Table II.

Relationship Between Clinical Variables and TCA Orientation (J0 and J45) in Diabetic Eyes

For the J0 component, representing horizontal/vertical astigmatism orientation (WTR/ATR), age remained independently associated with a shift towards more negative J0 values on multivariable analysis (Adj. b = –0.022 D/year; p = 0.002), reflecting an age-related transition towards ATR astigmatism (Table III).

Although WTW showed an association with J0 in univariable analysis, this relationship was no longer significant after adjustment. No systemic or ocular variables including age, HbA1c, DM duration, CCT, ECD, or metformin exposure were independently associated with the J45 component in either crude or adjusted analyses (all p > 0.05), suggesting that oblique astigmatism exhibits greater variability and weaker systemic associations.

Vector Distribution and Centroid Analysis

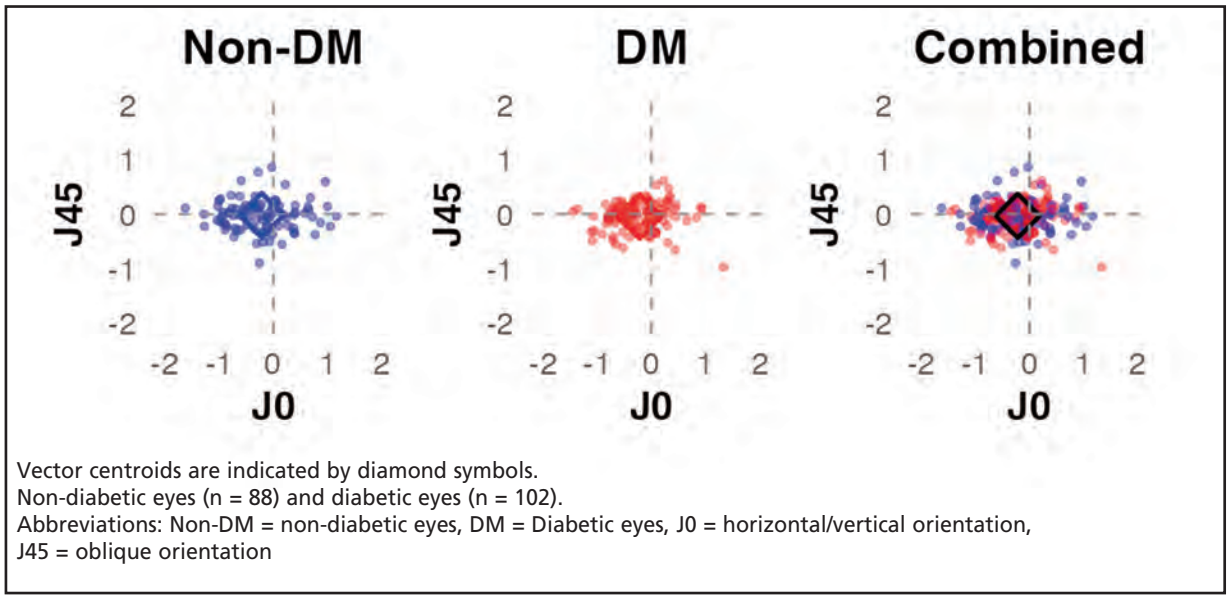
Double-angle vector plots demonstrated substantial overlap of J0 and J45 vector distributions and centroids between diabetic and non-diabetic eyes (Figure 1). This visual representation corroborated the statistical findings and confirmed the absence of a meaningful relationship between diabetes status and TCA orientation at a population level.

DISCUSSION

To our knowledge, this study is among the first to provide a vector-based characterisation of TCA in a Malay adult population, comparing diabetic and non-diabetic eyes. Our findings demonstrate that diabetes status alone does not systematically alter the TCA profile, while age and metabolic control (HbA1c) show statistically meaningful relationships with astigmatic orientation and magnitude, respectively. These findings suggest that corneal astigmatism is more strongly associated with age-related biomechanical remodelling and the prevailing metabolic environment than with the presence of diabetes itself.

Diabetes and TCA Magnitude

Diabetic eyes did not exhibit a statistically significant difference in TCA magnitude compared with non-diabetic eyes. This indicates that diabetes per se is less likely to be a dominant explanatory variable for TCA magnitude when



Vector centroids are indicated by diamond symbols.
Non-diabetic eyes (n = 88) and diabetic eyes (n = 102).
Abbreviations: Non-DM = non-diabetic eyes, DM = Diabetic eyes, J0 = horizontal/vertical orientation, J45 = oblique orientation

Fig. 1: Double Angle Plot of Non-Diabetic and Diabetic Eyes

assessed using TK. Importantly, this observation is consistent with prior work employing comparable keratometric methodologies, which similarly reported no meaningful magnitude difference between diabetic and non-diabetic eyes.[1]

Although earlier reports described higher anterior keratometric readings in diabetic patients, these often involved cohorts with poorer glycaemic control.[4,12] In contrast, the relatively well-controlled metabolic status of our diabetic cohort (mean HbA1c 7.9 ± 1.8%) and the use of TCA derived from TK, which incorporates posterior corneal power, may partly explain the absence of a significant magnitude difference. These findings suggest that any diabetes-related effect on TCA magnitude is likely modest and potentially masked by broader inter-individual variability.

HbA1c reflects integrated glycaemic exposure over the preceding three months and therefore represents the prevailing metabolic environment at the time of measurement. Elevated HbA1c levels are associated with hyperglycaemia-induced activation of polyol pathway, leading to intracellular sorbitol accumulation and osmotic stress within corneal tissues. In addition, accumulation of advanced glycation end products may alter collagen cross-linking changes in corneal biomechanics. These alterations may produce subtle curvature variability and contribute to astigmatic magnitude shift observed.[3,13]

Astigmatism magnitude exhibits substantial inter-individual dispersion in middle-aged and older populations, reflecting the combined effects of age-related corneal biomechanical remodelling and intrinsic anatomical heterogeneity.[5,14] This wide natural variability inflates error variance and reduces statistical power in magnitude-based cross-sectional comparisons, rendering them relatively insensitive to detecting subtle systemic influences such as diabetes-related effects on corneal toricity.[1] Accordingly, magnitude-only analyses may underestimate small but directionally specific corneal changes.

Although DM is frequently associated with increased CCT, our cohort demonstrated significantly thinner CCT in diabetic eyes. The median disease duration of approximately a decade suggests prolonged exposure to hyperglycaemia, which may induce degeneration of unmyelinated corneal nerves and reduce trophic support to the cornea, resulting in stromal and epithelial thinning.[4,13] Despite this structural difference, CCT was not independently associated with TCA magnitude or vector orientation after multivariable adjustment. This indicates that while diabetes-related corneal changes influence overall thickness, they do not meaningfully contribute to variability in astigmatic magnitude or orientation once age, metabolic control, and other ocular parameters are accounted for.

Collectively, these findings suggest that while TCA magnitude remains clinically relevant for refractive planning, it is not a discriminative marker for identifying diabetes-related corneal effects in cross-sectional analyses, where microstructural or transient metabolic influences may not translate into measurable differences in global corneal toricity.[3,4]

Vector Orientation of TCA (J0 and J45)

A major strength of this study is the application of vector-based astigmatism analysis, which overcomes the inherent limitations of conventional cylinder–axis notation for statistical modelling and group comparisons.

In both diabetic and non-diabetic eyes, mean J0 values were negative, indicating a predominance of ATR astigmatism consistent with the age profile of the cohort.[5,14] Crucially, no meaningful differences were observed between groups in either J0 or J45, and the substantial overlap of vector distributions and centroids on double-angle plots visually

corroborated these findings. This indicates the absence of systematic directional shift attributable to diabetes status.

These results indicate that diabetes does not systematically reorient TCA when assessed using TK and vector-based representation. This observed directional stability aligns with findings by Beato et al., who reported consistent keratometric behaviour between diabetic and non-diabetic eyes irrespective of diabetes duration or retinopathy stage.[1] Importantly, vector-based analysis further confirms that while apparent axis dispersion may be observed at the individual eye level, the population-level astigmatic centroid remains directionally stable across diabetes status, indicating preservation of the overall astigmatic phenotype.[8,14]

Relationships Between Clinical Variables and TCA in Diabetic Eyes

Within the diabetic cohort, HbA1c emerged as an independent variable associated with greater TCA magnitude, supporting the concept that current glycaemic control, rather than diabetes diagnosis alone, influences total corneal toricity. This relationship is biologically plausible, as fluctuations in glucose levels are known to induce transient refractive and topographic changes through alterations in stromal hydration and endothelial pump function.[3,13] This implies that while the presence of diabetes does not necessitate a change in surgical approach, the stability of glucose control at the time of biometry is paramount for refractive predictability.

In contrast, DM duration did not remain significant after multivariable adjustment, suggesting that its apparent effect is largely confounded by age and metabolic control. In the Malaysian context, delayed diagnosis and inconsistent longitudinal follow-up may further limit the precision of reported DM duration, reducing its statistical reliability as a predictor.[15]

Age remains the most consistent variable associated with vector orientation, independently related to a shift towards more negative J0 values. This finding reflects the well-established age-related transition towards ATR astigmatism, whereby age-driven biomechanical remodelling of the cornea shows a strong association with astigmatic orientation. As a result, the variance structure of vector profiles is largely governed by age effects, diminishing the relative contribution of disease-specific factors. Consequently, diabetes status alone did not differentiate vector profiles, as any potential diabetes-related influence appears modest when compared with the strong age-associated signal influencing corneal astigmatism orientation.[5,14]

No systemic or ocular variables were independently associated with the J45 component, consistent with the greater variability observed in oblique astigmatism. Unlike astigmatism magnitude or the J0 vector, J45 does not appear to follow a consistent biological pattern and demonstrates weak or absent associations with biometric and metabolic factors, as reported in prior studies.1,16 These findings support a cautious interpretation of J45 in both clinical assessment and epidemiological analysis in this Malay cohort.

Metformin Exposure and Corneal Astigmatism

Cumulative metformin exposure was not independently associated with TCA magnitude or vector components; however, short-term or transient corneal effects related to medication exposure cannot be excluded in a cross-sectional design. These findings reinforce the primacy of overall metabolic status, rather than specific pharmacological exposure in influencing corneal morphology.[3-4]

Implications for Cataract and Refractive Planning

From a cataract surgery perspective, these findings provide reassurance that TCA magnitude and orientation are broadly comparable between diabetes and non-diabetic eyes when measured using consistent technology. This supports the routine use of keratometric measurements without diabetes-specific adjustment. Nevertheless, the observed relationship between HbA1c and TCA magnitude highlights the importance of metabolic stability at the time of biometry acquisition to minimise transient refractive variability.[3,13]

LIMITATIONS

This study has several limitations. First, its cross-sectional design precludes causal inference between metabolic factors and corneal astigmatism behaviour. Systemic parameters were obtained at a single time point and may not fully capture temporal fluctuations in metabolic control that could influence corneal structure. Second, information regarding other diabetic treatment modalities, including insulin or combination therapy, was not consistently available across medical records, particularly for patients co-managed in primary care settings, and was therefore not included as a covariate.

Because medication modality was inconsistently documented, patients receiving insulin or combination therapy could not be reliably identified and treatment intensity was therefore not analysed. HbA1c was selected as the primary metabolic variable because it reflects integrated glycaemic exposure over the preceding three months and serves as a clinically relevant indicator of metabolic status independent of treatment regimen. Additionally, the modest R^2 values observed in the regression models indicate that a substantial proportion of variability in TCA remains unexplained, consistent with the multifactorial biomechanical nature of the cornea. As the diabetic cohort consisted predominantly of individuals with relatively well-controlled diabetes, inclusion of a larger proportion of poorly controlled diabetes may strengthen the observed associations. Finally, as the study population largely comprised older adults, the findings may not be generalisable to younger diabetic populations.

CONCLUSION

In this Malay adult population, diabetes status alone was not associated with systematic differences in TCA magnitude or orientation when assessed using TK and vector-based analysis. Age and metabolic control (HbA1c), rather than disease duration or metformin exposure, were the strongest factors associated with TCA characteristics. These findings support the use of vector-based analysis as a robust

framework for astigmatism assessment in both diabetic and non-diabetic eyes.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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