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Original Research Article

COMPARATIVE EVALUATION OF CENTRAL CORNEAL THICKNESS IN CONTROLLED AND UNCONTROLLED TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Diabetes mellitus is a major global health concern associated with several ocular complications, including structural and functional changes in the cornea. Chronic hyperglycemia can alter corneal endothelial function, increase central corneal thickness (CCT), and influence intraocular pressure (IOP) measurements, potentially affecting glaucoma assessment and the planning of anterior segment surgeries. The present study aimed to compare CCT among healthy individuals, controlled Type 2 diabetes mellitus (T2DM) patients, and uncontrolled T2DM patients, and to evaluate its association with glycemic control.

Materials and Methods: This cross-sectional observational study was conducted in the Department of Ophthalmology at Sri Guru Ram Das Institute of Medical Sciences & Research, Amritsar, from July 2024 to December 2025. A total of 300 participants were enrolled and divided into three groups: healthy controls (n = 150), controlled T2DM (HbA1c ≤7%; n = 75), and uncontrolled T2DM (HbA1c >7%; n = 75). Statistical analysis was carried out using SPSS, with $p < 0.05$ considered statistically significant.

Results: Mean central corneal thickness increased progressively from 525.8 ± 18.7 µm in healthy controls to 548.6 ± 21.4 µm in controlled diabetics and 566.9 ± 24.5 µm in uncontrolled diabetics ($p < 0.001$). Similarly, mean intraocular pressure was significantly higher in diabetic patients (14.2 ± 2.1, 16.0 ± 2.4, and 17.8 ± 2.8 mmHg, respectively; $p < 0.001$). HbA1c showed a significant positive correlation with CCT ($r = 0.58$, $p < 0.001$) and IOP ($r = 0.46$, $p < 0.001$). Multiple regression analysis identified HbA1c as the strongest independent predictor of CCT, while age and gender were not significant predictors.

Conclusion: Patients with Type 2 diabetes mellitus, particularly those with poor glycemic control, exhibit significantly increased central corneal thickness and intraocular pressure compared with healthy individuals.

Keywords: Type 2 diabetes mellitus; central corneal thickness; intraocular pressure; HbA1c; corneal endothelium; glycemic control.

INTRODUCTION

Diabetes mellitus (DM) is one of the most prevalent chronic metabolic disorders worldwide and constitutes a major public health challenge because of its rapidly increasing incidence and associated systemic and ocular complications. It is characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both, leading to disturbances in carbohydrate, fat, and protein metabolism.[1] According to the International Diabetes Federation (IDF), hundreds of millions of adults are currently living with diabetes globally, with Type 2 diabetes mellitus

(T2DM) accounting for approximately 90–95% of all cases. The disease imposes a substantial burden on healthcare systems due to its long-term microvascular and macrovascular complications affecting the retina, kidneys, peripheral nerves, cardiovascular system, and ocular surface.[2]

Among the ocular manifestations of diabetes, diabetic retinopathy has received considerable attention because it is a leading cause of visual impairment. However, increasing evidence suggests that diabetes also affects the anterior segment of the eye, particularly the cornea. The cornea is a transparent, avascular tissue responsible for approximately two-thirds of the refractive power of the eye and plays a critical role in maintaining visual quality. Its structural integrity depends on the coordinated function of the corneal epithelium, stroma, Descemet's membrane, and the corneal endothelium. Alterations in any of these layers may compromise corneal transparency and visual function.[3]

Chronic hyperglycemia induces several biochemical and cellular changes within the cornea. Persistent elevation of blood glucose promotes the accumulation of advanced glycation end products (AGEs), oxidative stress, activation of the polyol pathway, and chronic low-grade inflammation, all of which adversely affect corneal metabolism and endothelial cell function.[4] Since corneal endothelial cells possess limited regenerative capacity, prolonged metabolic insult may impair endothelial pump function, resulting in stromal hydration and an increase in central corneal thickness (CCT). Diabetes has also been associated with reduced corneal sensitivity, delayed epithelial wound healing, recurrent corneal erosions, endothelial polymegathism and pleomorphism, and increased susceptibility to postoperative corneal edema.[5]

Central corneal thickness is an important ophthalmic parameter because it influences the accuracy of intraocular pressure (IOP) measurement obtained by Goldmann applanation tonometry. A thicker cornea may lead to overestimation of IOP, whereas a thinner cornea may result in underestimation. Consequently, CCT has become an essential component in the diagnosis, monitoring, and management of glaucoma and other ocular disorders. Furthermore, accurate assessment of CCT is indispensable before refractive surgery, cataract surgery, and corneal transplantation, making it an important clinical biomarker in routine ophthalmic practice.[6]

Several studies have reported that patients with diabetes exhibit significantly greater CCT than non-diabetic individuals. Population-based evidence from the Singapore Malay Eye Study demonstrated a positive association between diabetes, elevated blood glucose levels, and increased central corneal thickness, suggesting that metabolic abnormalities may directly influence corneal structure.[7] Nevertheless, published findings remain inconsistent, as some investigators have observed only modest increases or no statistically significant differences after controlling for confounding variables such as age, duration of diabetes, and associated systemic diseases. These discrepancies may reflect differences in study populations, glycemic control, duration of disease, measurement techniques, and sample characteristics.[8]

Glycemic control is one of the most important determinants of diabetic complications. Glycated hemoglobin (HbA1c) serves as a reliable indicator of long-term blood glucose control and is widely used to classify patients into controlled and uncontrolled diabetes. Poor glycemic control is associated with greater oxidative stress, endothelial dysfunction, basement membrane thickening, and accelerated tissue damage. It is therefore biologically plausible that uncontrolled T2DM may produce more pronounced alterations in corneal morphology, including increased central corneal thickness, than well-controlled disease.[4,8]

Despite the growing recognition of diabetic keratopathy, relatively fewer studies have specifically compared central corneal thickness between controlled and uncontrolled Type 2 diabetes mellitus. Such comparisons are clinically important because changes in CCT may influence glaucoma assessment, surgical planning, postoperative outcomes, and overall ocular management in diabetic patients. Identifying differences in corneal thickness according to glycemic status may also provide additional insight into the impact of chronic hyperglycemia on corneal health and reinforce the importance of optimal metabolic control.

Therefore, the present study aims to comparatively evaluate central corneal thickness in patients with controlled and uncontrolled Type 2 diabetes mellitus. The findings may contribute to a better understanding of diabetes-related corneal alterations and assist ophthalmologists in improving clinical decision-making, particularly in the interpretation of intraocular pressure measurements and the preoperative assessment of diabetic patients.

MATERIALS AND METHODS

Study Design and Setting: This hospital-based cross-sectional observational study was conducted in the Department of Ophthalmology, Sri Guru Ram Das Institute of Medical Sciences and Research, Amritsar, Punjab, India, over a period of 18 months from July 2024 to December 2025. The study adhered to the principles outlined in the Declaration of Helsinki. Approval was obtained from the Institutional Ethics Committee before commencement of the study, and written informed consent was obtained from all participants prior to enrollment.

Study Population: A total of 300 participants were recruited consecutively from the ophthalmology outpatient and inpatient services. The study

population consisted of 150 patients diagnosed with Type 2 diabetes mellitus and 150 age-matched healthy individuals without diabetes who served as controls. Diabetic participants were further stratified according to glycemic control based on glycated hemoglobin (HbA1c) levels into:

- Controlled Type 2 diabetes mellitus (HbA1c ≤7%)
- Uncontrolled Type 2 diabetes mellitus (HbA1c >7%)

Only one averaged value per participant was included in the final statistical analysis to avoid inter-eye correlation.

Sample Size Calculation: The sample size was estimated for comparison of two independent means using the following formula:

$$n = \frac{(Z_{\alpha/2} + Z_{\beta})^2 (SD_1^2 + SD_2^2)}{(M_1 - M_2)^2}$$

where Zα/2 was 1.96 for a 95% confidence interval, Zβ was 0.84 corresponding to 80% study power, SD represented the standard deviation of central corneal thickness in each group, and $M_1 - M_2$ represented the anticipated mean difference.

Based on previous published data demonstrating an expected difference of approximately 10 µm with a standard deviation of 30 µm, the minimum required sample size was calculated as 142 participants per group. After considering a potential 10% attrition rate, 150 participants were included in each group, yielding a total sample size of 300 participants.

Eligibility Criteria

Inclusion Criteria

Participants were eligible if they fulfilled the following criteria:

- Adults diagnosed with Type 2 diabetes mellitus according to the treating physician with HbA1c ≥6.5%.
- Age-matched healthy individuals without diabetes serving as controls.

Exclusion Criteria

Participants were excluded if they had any condition known to influence corneal thickness or intraocular pressure, including:

- Previous ocular trauma
- Previous ocular surgery
- Refractive surgery
- Active ocular infection
- Corneal opacity, dystrophy, degeneration, or scarring
- History of glaucoma surgery or glaucoma medication
- Thyroid disorders
- Hypertension treated with systemic beta-blockers
- Any other ocular pathology affecting corneal integrity.

Clinical Evaluation: A detailed demographic and clinical history was obtained from each participant. Information regarding age, sex, duration of diabetes, medical history, and HbA1c levels was recorded. All participants underwent a comprehensive ophthalmic examination including best-corrected visual acuity assessment, slit-lamp biomicroscopy, fundus examination, and measurement of intraocular pressure and central corneal thickness.

Measurement of Central Corneal Thickness: Central corneal thickness (CCT) was measured using a NIDEK CEM-530 non-contact specular microscope (NIDEK Co., Ltd., Japan).

Participants were seated comfortably with the chin positioned on the chin rest and forehead against the support. They were instructed to fixate on the internal target while maintaining steady gaze. Proper alignment of the optical axis was ensured before image acquisition. Five consecutive measurements were obtained from the central cornea of each eye, and the average value was recorded for analysis. Images that were poorly focused or demonstrated inadequate endothelial visualization were discarded and repeated until acceptable image quality was obtained.

The same calibrated instrument was used throughout the study, and all measurements were performed by a single experienced examiner to minimize inter-observer variability.

Measurement of Intraocular Pressure: Intraocular pressure (IOP) was measured using a Goldmann applanation tonometer, regarded as the clinical gold standard.

One drop of 0.5% proparacaine hydrochloride topical anesthetic was instilled into each eye followed by fluorescein staining using a sterile fluorescein strip. Measurements were obtained under cobalt-blue illumination using slit-lamp biomicroscopy. The tonometer prism was disinfected before each examination according to institutional infection-control protocols.

Three consecutive IOP measurements were obtained for each eye, and their mean value was considered for statistical analysis. All measurements were performed by the same ophthalmologist under standardized environmental conditions to minimize measurement variability.

Outcome Measures: The primary outcome was comparison of central corneal thickness between participants with controlled and uncontrolled Type 2 diabetes mellitus.

Secondary outcomes included comparison of intraocular pressure among study groups and evaluation of the relationship between glycemic control and corneal thickness.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics software (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro–Wilk test. Normally distributed variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages.

Comparisons between two independent groups were performed using the independent samples Student's t-test. Comparisons among more than two groups were performed using one-way analysis of variance

(ANOVA) followed by appropriate post hoc testing. Categorical variables were analyzed using the Chi-square test or Fisher's exact test where appropriate. Pearson's correlation coefficient was used to evaluate associations between HbA1c levels, central corneal thickness, and intraocular pressure. A two-tailed p value <0.05 was considered statistically significant.

RESULTS

A total of 300 participants were included in the study, comprising 150 healthy controls, 75 patients with controlled Type 2 diabetes mellitus, and 75 patients with uncontrolled Type 2 diabetes mellitus. The majority of participants belonged to the 40–60-year age group, with comparable age and gender distribution across the three groups. Central corneal thickness and intraocular pressure were subsequently compared among the study groups.

[Table 1] presents the baseline demographic characteristics of the study participants. A total of 300 individuals were included, comprising 150 healthy controls, 75 patients with controlled Type 2 diabetes mellitus (HbA1c ≤7%), and 75 patients with uncontrolled Type 2 diabetes mellitus (HbA1c >7%). The mean age was comparable among the three groups, with no statistically significant difference (p = 0.214). Similarly, the distribution of male and female participants did not differ significantly across the groups (p = 0.731). These findings indicate that the study groups were well matched with respect to age and gender, thereby minimizing the influence of demographic confounding factors on the comparison of central corneal thickness and intraocular pressure.

Table 1: Demographic characteristics of study participants

Variable	Controls (n=150)	Controlled T2DM (n=75)	Uncontrolled T2DM (n=75)	p-value
Age (years)	51.8 ± 10.4	53.2 ± 9.8	54.1 ± 10.7	0.214
Male, n (%)	82 (54.7)	43 (57.3)	45 (60.0)	0.731
Female, n (%)	68 (45.3)	32 (42.7)	30 (40.0)	

Table 2: Glycemic characteristics of diabetic participants

Variable	Controlled T2DM	Uncontrolled T2DM	p-value
Duration of diabetes (years)	6.4 ± 2.8	10.1 ± 4.5	<0.001
HbA1c (%)	6.58 ± 0.29	8.84 ± 1.12	<0.001

[Table 2] compares the clinical profile of participants with controlled and uncontrolled Type 2 diabetes mellitus. Patients in the uncontrolled diabetes group demonstrated a significantly longer duration of diabetes and markedly higher HbA1c levels than those with controlled diabetes (p < 0.001). These findings confirm clear separation between the two diabetic groups according to glycemic status and indicate poorer long-term metabolic control among participants with uncontrolled diabetes.

Table 3: Comparison of Central Corneal Thickness among study groups

Group	Mean CCT (µm)	SD	p-value
Controls	525.8	18.7	
Controlled T2DM	548.6	21.4	
Uncontrolled T2DM	566.9	24.5	<0.001
Post hoc analysis			
Comparison	**Mean Difference (µm)**		**p-value**
Controls vs Controlled T2DM	22.8		<0.001
Controls vs Uncontrolled T2DM	41.1		<0.001
Controlled vs Uncontrolled T2DM	18.3		<0.001

[Table 3] summarizes the comparison of central corneal thickness (CCT) among healthy controls, controlled Type 2 diabetes mellitus, and uncontrolled Type 2 diabetes mellitus. The mean CCT progressively increased from healthy controls to controlled diabetic patients and reached the highest values among patients with uncontrolled diabetes. One-way analysis of variance demonstrated a highly significant overall difference between the three groups (p < 0.001). Post hoc analysis further revealed statistically significant differences between each pair of groups. These findings suggest that worsening glycemic control is associated with increased central corneal thickness.

Table 4: Comparison of Intraocular Pressure among study groups

Group	Mean IOP (mmHg)	SD	p-value
Controls	14.2	2.1	
Controlled T2DM	16.0	2.4	
Uncontrolled T2DM	17.8	2.8	<0.001
Post hoc analysis			
Comparison	**Mean Difference**		**p-value**
Controls vs Controlled	1.8		<0.001

Controls vs Uncontrolled	3.6	<0.001
Controlled vs Uncontrolled	1.8	0.002

[Table 4] compares intraocular pressure (IOP) among the three study groups. The mean IOP was lowest in healthy controls, intermediate in patients with controlled diabetes, and highest among those with uncontrolled diabetes. Statistical analysis demonstrated a highly significant difference in mean IOP across the groups ($p < 0.001$). Pairwise comparisons also showed significant differences between controls and diabetic participants as well as between controlled and uncontrolled diabetes. The findings indicate that poor glycemic control is associated with higher intraocular pressure.

Table 5: Correlation of HbA1c with Central Corneal Thickness and Intraocular Pressure

Variable	Pearson's r	p-value
HbA1c vs CCT	0.58	<0.001
HbA1c vs IOP	0.46	<0.001

[Table 5] presents the correlation analysis between HbA1c levels, central corneal thickness, and intraocular pressure in diabetic patients. Pearson's correlation analysis demonstrated a moderate positive correlation between HbA1c and central corneal thickness ($r = 0.58$, $p < 0.001$), indicating that increasing HbA1c levels were associated with thicker corneas. A significant positive correlation was also observed between HbA1c and intraocular pressure ($r = 0.46$, $p < 0.001$). These findings suggest that poor glycemic control contributes to structural corneal changes and increased intraocular pressure.

Table 6: Multiple linear regression analysis for predictors of Central Corneal Thickness

Predictor	β Coefficient	Standard Error	p-value
Age	0.08	0.06	0.162
Gender	0.04	0.81	0.493
Duration of diabetes	0.29	0.09	0.003
HbA1c	0.51	0.07	<0.001

[Table 6] presents the results of multiple linear regression analysis performed to identify independent predictors of central corneal thickness. After adjustment for age and gender, HbA1c emerged as the strongest independent predictor of increased central corneal thickness ($p < 0.001$), followed by duration of diabetes ($p = 0.003$). In contrast, neither age nor gender showed a statistically significant association with central corneal thickness. These findings indicate that chronic hyperglycemia and prolonged duration of diabetes independently contribute to corneal thickening.

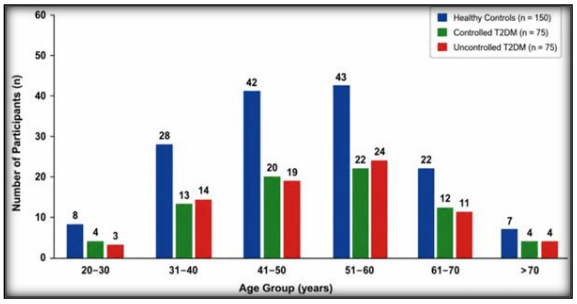

Figure 1: Age-wise distribution of healthy controls, controlled Type 2 diabetes mellitus, and uncontrolled Type 2 diabetes mellitus

[Figure 1] illustrates the age-wise distribution of participants across the three study groups: healthy controls, controlled Type 2 diabetes mellitus (HbA1c ≤7%), and uncontrolled Type 2 diabetes mellitus (HbA1c >7%). The majority of participants in all groups belonged to the 51–60 years age category, followed by the 41–50 years age group. Comparatively fewer participants were observed in the younger (20–30 years and 31–40 years) and older (>70 years) age categories. The age distribution was broadly comparable among the three groups, indicating that there was no significant difference in the age profile of the study population. This comparable distribution minimizes the potential influence of age as a confounding factor in the evaluation of central corneal thickness and intraocular pressure among healthy controls, controlled diabetics, and uncontrolled diabetics.

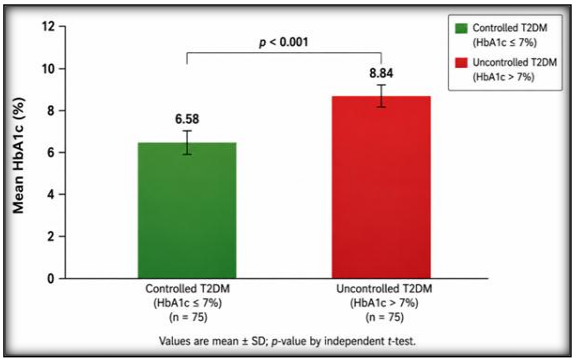

Figure 2: Comparison of mean HbA1c levels between controlled and uncontrolled Type 2 diabetes mellitus

[Figure 2] illustrates the comparison of mean glycated hemoglobin (HbA1c) levels between patients with controlled and uncontrolled Type 2 diabetes mellitus. The mean HbA1c level was 6.58

± 0.29% in the controlled diabetes group and 8.84 ± 1.12% in the uncontrolled diabetes group. Statistical analysis using the independent t-test demonstrated a highly significant difference between the two groups ($p < 0.001$). The markedly higher HbA1c values observed in the uncontrolled diabetes group indicate poorer long-term glycemic control compared with the controlled diabetes group. These findings confirm the appropriate classification of diabetic participants into controlled and uncontrolled groups based on HbA1c levels and provide the basis for subsequent comparisons of central corneal thickness and intraocular pressure.

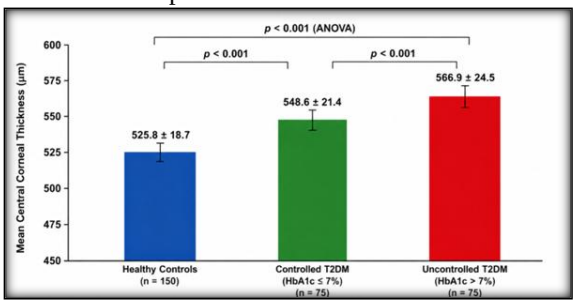

Figure 3: Comparison of mean central corneal thickness among healthy controls, controlled Type 2 diabetes mellitus, and uncontrolled Type 2 diabetes mellitus

[Figure 3] illustrates the comparison of mean central corneal thickness (CCT) among healthy controls, patients with controlled Type 2 diabetes mellitus, and patients with uncontrolled Type 2 diabetes mellitus. The mean CCT was 525.8 ± 18.7 µm in healthy controls, 548.6 ± 21.4 µm in the controlled diabetes group, and 566.9 ± 24.5 µm in the uncontrolled diabetes group. A progressive increase in central corneal thickness was observed from healthy controls to controlled diabetic patients, with the highest values recorded in patients with uncontrolled diabetes. Statistical analysis using one-way analysis of variance (ANOVA) demonstrated a highly significant difference among the three groups ($p < 0.001$). Post hoc analysis further confirmed significant differences between healthy controls and controlled diabetes, healthy controls and uncontrolled diabetes, as well as between controlled and uncontrolled diabetes (all $p < 0.001$). These findings indicate that central corneal thickness increases with worsening glycemic control and suggest that chronic hyperglycemia may contribute to structural alterations of the cornea in patients with Type 2 diabetes mellitus.

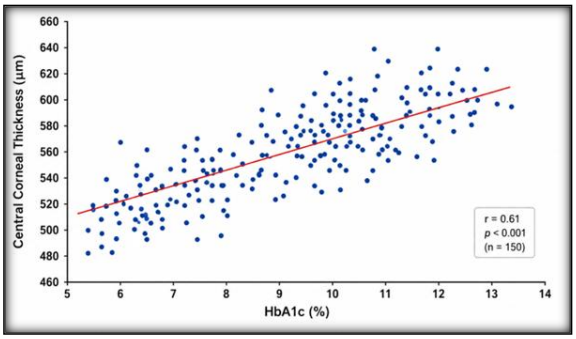

Figure 4: Scatter plot demonstrating the positive correlation between HbA1c and central corneal thickness in diabetic participants

[Figure 4] illustrates the relationship between glycated hemoglobin (HbA1c) levels and central corneal thickness (CCT) among diabetic participants. The scatter plot demonstrates a moderate positive correlation, indicating that central corneal thickness tended to increase with increasing HbA1c levels. Pearson's correlation analysis revealed a statistically significant positive association between HbA1c and CCT ($r = 0.58$, $p < 0.001$), suggesting that poorer long-term glycemic control is associated with greater corneal thickness. The fitted regression line shows an upward linear trend, further supporting the positive relationship between these variables. These findings indicate that chronic hyperglycemia may contribute to structural changes in the cornea, emphasizing the potential influence of glycemic control on corneal morphology in patients with Type 2 diabetes mellitus.

DISCUSSION

The present study was undertaken to comparatively evaluate central corneal thickness (CCT) in patients with controlled and uncontrolled Type 2 diabetes mellitus and to determine whether glycemic control influences corneal morphology. Diabetes mellitus is increasingly recognized as a disorder affecting not only the retina but also the anterior segment of the eye. Chronic hyperglycemia induces structural and functional changes in the corneal epithelium, stroma, endothelium, and corneal nerves through the accumulation of advanced glycation end products (AGEs), oxidative stress, chronic inflammation, and endothelial dysfunction. These alterations may ultimately result in increased central corneal thickness and inaccurate intraocular pressure measurements, thereby influencing glaucoma diagnosis and the planning of anterior segment surgeries.[9,10] The principal findings of the present study demonstrated that central corneal thickness and intraocular pressure increased progressively with worsening glycemic control, while HbA1c emerged as an independent predictor of increased corneal thickness. These observations support the concept that long-term metabolic control plays an

important role in maintaining corneal integrity in patients with Type 2 diabetes mellitus.

The demographic characteristics of the study population revealed that the three study groups were comparable with respect to age and sex distribution, with no statistically significant differences observed between healthy controls, controlled diabetic patients, and uncontrolled diabetic patients. Such homogeneity is important because age and sex have been reported to influence corneal characteristics and could potentially confound comparisons of central corneal thickness. The comparable demographic profile of the present study therefore strengthens the internal validity of the findings by minimizing the influence of these variables on the observed differences in corneal thickness. Similar age- and sex-matched study designs have been adopted in previous investigations evaluating diabetes-related corneal alterations, thereby allowing more reliable assessment of the independent effect of diabetes on corneal morphology.[11,12] Furthermore, a large population-based study conducted among multiethnic Asian adults demonstrated that, even after adjustment for demographic characteristics and other ocular variables, diabetes remained independently associated with greater central corneal thickness, emphasizing that metabolic status rather than demographic characteristics primarily explains the observed corneal changes.[9]

The present study also demonstrated a significant difference in HbA1c levels between controlled and uncontrolled diabetic participants, confirming the appropriate categorization of the study groups according to long-term glycemic control. Patients with uncontrolled diabetes exhibited markedly higher HbA1c values together with a longer duration of disease, indicating prolonged exposure to chronic hyperglycemia. Glycated hemoglobin is widely accepted as the most reliable indicator of average blood glucose concentration over the preceding two to three months and has consistently been associated with the development and progression of diabetic microvascular complications. Similar observations have been reported in previous clinical studies showing that poor glycemic control is associated with increased corneal thickness, endothelial dysfunction, reduced endothelial cell density, polymegathism, pleomorphism, and delayed epithelial wound healing.[10,13] The underlying mechanisms include increased intracellular sorbitol accumulation through activation of the polyol pathway, excessive production of reactive oxygen species, mitochondrial dysfunction, and accumulation of AGEs within the corneal extracellular matrix. These biochemical disturbances impair endothelial pump function, promote stromal hydration, and consequently increase central corneal thickness.[12,13]

The findings of the present study therefore reinforce the concept that HbA1c is not only a marker of systemic metabolic control but also an important indicator of diabetes-related ocular alterations. The significant difference in HbA1c observed between controlled and uncontrolled diabetic patients provided an appropriate framework for evaluating the influence of glycemic status on corneal morphology in the subsequent analyses of this study.

One of the principal findings of the present study was the significant increase in central corneal thickness (CCT) among patients with Type 2 diabetes mellitus, with the greatest values observed in the uncontrolled diabetic group. Mean CCT increased progressively from 525.8 ± 18.7 µm in healthy controls to 548.6 ± 21.4 µm in controlled diabetics and 566.9 ± 24.5 µm in uncontrolled diabetics, with the differences being highly significant ($p < 0.001$). This stepwise increase strongly suggests that deterioration in glycemic control is accompanied by measurable structural changes within the cornea. These findings are clinically important because increased CCT may lead to overestimation of intraocular pressure measured by Goldmann applanation tonometry, thereby affecting glaucoma diagnosis, risk stratification, and therapeutic decision-making.

The present findings are consistent with several previous investigations reporting thicker corneas in diabetic patients than in healthy individuals. Storr-Paulsen et al. demonstrated a significantly greater CCT in patients with Type 2 diabetes despite relatively good glycemic control, indicating that diabetes itself contributes to alterations in corneal morphology independent of overt corneal edema.[14] Likewise, El-Agamy and Alsubaie reported significantly increased CCT together with reduced endothelial cell density and increased endothelial pleomorphism in diabetic patients, suggesting that chronic metabolic stress adversely affects both corneal thickness and endothelial integrity.[14] More recently, Kim and Kim, in a large retrospective study involving over 500 diabetic patients, observed that increased CCT was particularly evident in patients with longer disease duration and HbA1c levels ≥7%, supporting the concept that poor metabolic control accelerates diabetes-related corneal changes.[15] These observations closely parallel the results of the present study, in which uncontrolled diabetes was associated with the greatest increase in central corneal thickness.

The pathophysiological basis for increased CCT in diabetes is multifactorial. Persistent hyperglycemia activates the polyol pathway, resulting in intracellular sorbitol accumulation, osmotic stress, oxidative injury, and mitochondrial dysfunction within corneal endothelial cells. In addition, excessive formation of advanced glycation end products (AGEs) promotes collagen cross-linking, reduces endothelial Na^+/K^+-ATPase pump activity, and impairs the cornea's ability to maintain stromal dehydration. Consequently, stromal hydration increases, producing a measurable rise in central corneal thickness. These structural

alterations become more pronounced with prolonged disease duration and inadequate glycemic control, explaining the progressive increase in CCT observed in uncontrolled diabetic patients.

The present study demonstrated a significant increase in intraocular pressure (IOP) among diabetic patients, with the highest mean IOP observed in the uncontrolled diabetes group. Since Goldmann applanation tonometry is influenced by central corneal thickness (CCT), the elevated IOP recorded in diabetic individuals may reflect not only true ocular hypertension but also diabetes-induced alterations in corneal biomechanics. Chronic hyperglycemia increases collagen cross-linking, corneal stiffness, and corneal thickness, thereby resulting in overestimation of IOP measurements. Ramm et al. reported that although Goldmann-measured IOP tended to be higher in diabetic patients, this increase was closely associated with changes in corneal thickness and biomechanical properties rather than a genuine elevation in intraocular pressure.[16] Therefore, assessment of CCT should be incorporated when interpreting IOP in diabetic patients, particularly in those being evaluated for glaucoma.[17]

Another important finding of the present study was the significant positive correlation between HbA1c and central corneal thickness ($r = 0.58$, $p < 0.001$), indicating that worsening glycemic control is associated with progressive corneal thickening. This finding supports previous evidence that chronic hyperglycemia contributes directly to structural changes within the cornea through accumulation of advanced glycation end products, oxidative stress, endothelial dysfunction, and impaired stromal fluid regulation. A systematic review by Wang et al. concluded that diabetes is consistently associated with increased corneal biomechanical parameters and thicker corneas, particularly in patients with poor metabolic control and longer disease duration.[18] Similarly, Mathew et al. emphasized that prolonged hyperglycemia adversely affects corneal endothelial function and stromal homeostasis, ultimately leading to increased corneal thickness and altered corneal biomechanics.[19]

In the present study, multiple linear regression analysis identified HbA1c as the strongest independent predictor of central corneal thickness, whereas age and gender did not demonstrate significant independent associations. This finding emphasizes that long-term glycemic control exerts a greater influence on corneal morphology than demographic variables. Clinically, these observations underscore the importance of incorporating pachymetry into routine ophthalmic evaluation of patients with Type 2 diabetes mellitus. Measurement of CCT not only improves the interpretation of intraocular pressure but may also serve as an indicator of diabetes-related corneal involvement. Early identification of corneal alterations in poorly controlled diabetes may facilitate more accurate glaucoma assessment, safer planning of refractive and cataract surgery, and closer ophthalmic surveillance, ultimately contributing to improved visual outcomes and individualized patient management.[20]

CONCLUSION

The present study demonstrated that patients with Type 2 diabetes mellitus have significantly greater central corneal thickness (CCT) and intraocular pressure (IOP) than healthy individuals, with the highest values observed among patients with uncontrolled diabetes. A progressive increase in CCT from healthy controls to controlled and uncontrolled diabetic patients highlights the adverse effect of chronic hyperglycemia on corneal structure. Furthermore, the significant positive correlation between HbA1c and CCT, together with multiple regression analysis identifying HbA1c as the strongest independent predictor of corneal thickness, indicates that long-term glycemic control plays a crucial role in determining corneal health.

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