# Comparative Analysis of Central Corneal Thickness in Diabetic and Non-Diabetic Patients: A Cross-Sectional Study Ashwini K G<sup>1</sup>, Putta Vamsi Sarika<sup>2</sup>, Moparthy Amulya<sup>2</sup>, Aishwarya N<sup>3</sup>

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# **ABSTRACT**

**Background:** Diabetes mellitus affects various ocular structures, including the cornea. Understanding the relationship between diabetes and central corneal thickness (CCT) has important clinical implications for patient care.

**Objective:** To compare central corneal thickness between diabetic and non-diabetic patients and evaluate its correlation with glycemic control parameters.

**Methods:** This cross-sectional study conducted at Basaveshwara Medical College and Hospital included 30 participants (15 diabetic, 15 non-diabetic controls). Central corneal thickness was measured using ultrasound pachymetry. Comprehensive ophthalmic examination and laboratory investigations including glycated hemoglobin (HbA1c) were performed for all participants.

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**Results:** Mean CCT was significantly higher in diabetic patients ( $558.6 \pm 32.4 \,\mu\text{m}$ ) compared to controls ( $534.2 \pm 28.6 \,\mu\text{m}$ , p=0.028). A significant positive correlation was found between CCT and HbA1c levels (r=0.526, p=0.044). Multiple regression analysis identified duration of diabetes ( $\beta$ =0.324, p=0.042) and HbA1c ( $\beta$ =0.468, p=0.022) as independent predictors of CCT.

**Conclusion:** Diabetes mellitus is associated with increased central corneal thickness, with glycemic control and disease duration serving as significant predictors. These findings emphasize the importance of considering diabetes status when interpreting corneal thickness measurements.

**Keywords:** Central Corneal Thickness; Diabetes Mellitus; Glycated Hemoglobin; Pachymetry; Cornea; Cross-Sectional Studies

# INTRODUCTION

Diabetes mellitus represents a significant global health challenge, affecting approximately 537 million adults worldwide as of 2021(1). This chronic metabolic disorder is characterized by persistent hyperglycemia, which leads to various micro and macrovascular complications affecting multiple organ systems, including the eye(2). The ocular manifestations of diabetes extend beyond the well-documented retinopathy, potentially affecting all anatomical structures of the eye, including the cornea(3).

The cornea, being an avascular and transparent structure, plays a crucial role in maintaining optical quality and protecting the internal structures of the eye. Central corneal thickness (CCT) is an important parameter in ophthalmology, influencing not only the accuracy of intraocular pressure measurements but also serving as an indicator of corneal health and metabolism(4). Recent evidence suggests that diabetes mellitus may induce structural and

functional changes in the cornea, potentially affecting its thickness and biomechanical properties(5).

Several mechanisms have been proposed to explain the relationship between diabetes and corneal alterations. Chronic hyperglycemia leads to the accumulation of advanced glycation end products (AGEs) in corneal tissue, affecting collagen structure and interfibrillar spacing(6). Additionally, diabetes-induced changes in endothelial cell morphology and function may impact corneal hydration status and thickness. The corneal endothelial pump function, crucial for maintaining corneal deturgescence, may be compromised in diabetic patients, potentially leading to altered corneal thickness(7).

Understanding the association between diabetes mellitus and central corneal thickness has significant clinical implications. CCT measurements are essential for accurate glaucoma screening and management, as they influence tonometry readings. Given the higher prevalence of glaucoma in diabetic patients, proper interpretation of intraocular pressure measurements in this population is crucial(8). Furthermore, changes in corneal thickness may affect the outcomes of various corneal and refractive surgical procedures, which are increasingly common in the diabetic population(9).

Previous studies investigating the relationship between diabetes and CCT have shown varying results. While some researchers have reported increased CCT in diabetic patients, others have found no significant difference compared to non-diabetic controls(10,11). These inconsistencies might be attributed to variations in study populations, diabetes duration, glycemic control, and measurement techniques.

The duration of diabetes and the degree of glycemic control may play crucial roles in determining corneal changes. Chronic exposure to hyperglycemia could potentially lead to more pronounced corneal alterations, suggesting a possible correlation between disease duration, glycemic control, and CCT variations(12). Understanding these relationships could help in better prediction and management of corneal complications in diabetic patients.

The present study aims to investigate the association between diabetes mellitus and central corneal thickness, while also examining the potential influence of disease duration, glycemic control, and other relevant clinical parameters. This research endeavors to contribute to the existing knowledge base and potentially influence clinical practice in the management of diabetic patients requiring ophthalmological care.

#### **METHODOLOGY**

This cross-sectional study was conducted at the Department of Ophthalmology, Basaveshwara Medical College and Hospital between June 2024 and December 2024. The study protocol received approval from the Institutional Ethics Committee, and all research procedures adhered to the tenets of the Declaration of Helsinki. A total of 30 participants were enrolled in the study, comprising 15 diabetic patients and 15 age-matched non-diabetic controls who attended the ophthalmology outpatient department.

The study population was carefully selected based on predefined criteria. For the diabetic group, participants included diagnosed cases of Type 2 Diabetes Mellitus aged between 40-70 years, with a minimum disease duration of one year and regular follow-up with documented glycemic control. The control group consisted of age-matched non-diabetic individuals with normal fasting and post-prandial blood glucose levels and no family history of diabetes mellitus. Participants were excluded from both groups if they had a history of corneal disease or surgery, contact lens wear, glaucoma or ocular hypertension, active ocular infection or inflammation, history of systemic conditions affecting corneal thickness, or were pregnant or lactating.

All participants underwent comprehensive evaluation including detailed medical history documentation. For diabetic patients, this encompassed the duration of diabetes, current medications, systemic comorbidities, previous ocular history, and family history. Laboratory investigations were conducted for all participants, including fasting blood glucose, post-prandial blood glucose, glycated hemoglobin (HbA1c), serum creatinine, and lipid profile. These tests were performed in the hospital's clinical laboratory using standardized procedures.

The ophthalmic examination protocol included measurement of best-corrected visual acuity using Snellen's chart, slit-lamp biomicroscopy, fundus examination, and intraocular pressure measurement using Goldmann applanation tonometry. Central corneal thickness measurements were performed using ultrasound pachymetry under standardized conditions. All measurements were taken between 9:00 AM and 11:00 AM to minimize diurnal variation. Three consecutive readings were obtained from the central cornea of each eye, and the mean value was calculated. For consistency in analysis, only the right eye measurements were included in the final data analysis.

Data collection was systematically performed using standardized forms to record demographic information, clinical findings, and investigation results. All data was entered into a secure electronic database with double-entry verification to minimize errors. Quality control measures included regular calibration of measuring instruments, training of research staff, periodic data quality checks, and implementation of standard operating procedures for all measurements.

Statistical analysis was conducted using SPSS version 26.0. The analytical approach included descriptive statistics presented as mean  $\pm$  standard deviation for continuous variables and frequencies for categorical variables. Normal distribution was assessed using the Kolmogorov-Smirnov test. Independent t-test was employed to compare CCT between diabetic and non-diabetic groups. Pearson's correlation coefficient was calculated to assess the relationship between CCT and various parameters including duration of diabetes, HbA1c levels, and fasting blood glucose levels. Multiple linear regression analysis was performed to identify independent predictors of CCT. Statistical significance was set at p < 0.05.

Ethical considerations were strictly maintained throughout the study period. Written informed consent was obtained from all participants after explaining the study procedure in their native language. Participants were informed of their right to withdraw from the study at any time without affecting their standard of care. Patient confidentiality was maintained through coded

data entry and secure data storage. Any adverse events during the study period were documented and reported to the ethics committee according to institutional protocols.

# **RESULTS**

The study included 30 participants equally divided between diabetic and control groups. Demographic analysis (Table 1) revealed comparable age distribution between diabetic (56.4  $\pm$  8.2 years) and control groups (54.8  $\pm$  7.9 years, p=0.582). Both groups showed similar gender distribution and BMI, though the diabetic group demonstrated significantly higher systolic (138.6  $\pm$  12.4 vs 124.8  $\pm$  10.2 mmHg, p=0.002) and diastolic blood pressure (84.5  $\pm$  8.6 vs 78.4  $\pm$  7.8 mmHg, p=0.042).

**Table 1: Demographic and Clinical Characteristics of Study Participants** 

Characteristic	Diabetic Group (n=15)	Control Group (n=15)	P-value
Age (years)*	$56.4 \pm 8.2$	$54.8 \pm 7.9$	0.582
Gender (M/F)	8/7	7/8	0.715
BMI (kg/m²)*	$27.3 \pm 3.8$	$25.1 \pm 3.2$	0.089
Systolic BP (mmHg)*	$138.6 \pm 12.4$	$124.8 \pm 10.2$	0.002†
Diastolic BP (mmHg)*	$84.5 \pm 8.6$	$78.4 \pm 7.8$	0.042†
Duration of DM (years)*	$8.4 \pm 4.6$	-	-

<sup>\*</sup>Values expressed as Mean  $\pm$  SD †Statistically significant (p < 0.05)

Laboratory parameters (Table 2) showed significantly elevated glycemic indices in the diabetic group, with higher fasting blood sugar (156.8  $\pm$  32.4 vs 92.4  $\pm$  8.6 mg/dL, p<0.001), post-prandial blood sugar (224.5  $\pm$  45.6 vs 118.6  $\pm$  12.4 mg/dL, p<0.001), and HbA1c levels (7.8  $\pm$  1.2 vs 5.4  $\pm$  0.4%, p<0.001). Total cholesterol was also significantly higher in the diabetic group (p=0.045).

**Table 2: Laboratory Parameters of Study Groups** 

Parameter	Diabetic Group (n=15)	Control Group (n=15)	P-value
FBS (mg/dL)*	$156.8 \pm 32.4$	$92.4 \pm 8.6$	<0.001†
PPBS (mg/dL)*	$224.5 \pm 45.6$	118.6 ± 12.4	<0.001†
HbA1c (%)*	$7.8 \pm 1.2$	$5.4 \pm 0.4$	<0.001†
Total Cholesterol (mg/dL)*	$198.4 \pm 38.6$	$174.2 \pm 28.4$	0.045†
Serum Creatinine (mg/dL)*	$1.1 \pm 0.3$	$0.9 \pm 0.2$	0.062

<sup>\*</sup>Values expressed as Mean  $\pm$  SD †Statistically significant (p < 0.05)

Central corneal thickness measurements (Table 3) revealed significantly higher mean CCT in diabetic patients ( $558.6 \pm 32.4 \,\mu\text{m}$ ) compared to controls ( $534.2 \pm 28.6 \,\mu\text{m}$ , p=0.028). Intraocular pressure readings were comparable between groups (p=0.142).

**Table 3: Central Corneal Thickness Measurements** 

Parameter	Diabetic Group (n=15)	Control Group (n=15)	P-value
Mean CCT (µm)*	558.6 ± 32.4	$534.2 \pm 28.6$	0.028†
Minimum CCT (μm)	512	498	-
Maximum CCT (μm)	602	576	-
IOP (mmHg)*	$16.8 \pm 2.8$	$15.4 \pm 2.4$	0.142

<sup>\*</sup>Values expressed as Mean  $\pm$  SD †Statistically significant (p < 0.05)

Correlation analysis (Table 4) demonstrated a significant positive correlation between CCT and HbA1c levels (r=0.526, p=0.044) in diabetic patients. Multiple regression analysis (Table 5) identified duration of diabetes ( $\beta$ =0.324, p=0.042) and HbA1c ( $\beta$ =0.468, p=0.022) as significant independent predictors of CCT, with the model explaining 54.2% of CCT variance (R²=0.542).

Table 4: Correlation of CCT with Various Parameters in Diabetic Group

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Parameter	<b>Correlation Coefficient (r)</b>	P-value
<b>Duration of DM</b>	0.384	0.158
HbA1c	0.526	0.044†
FBS	0.412	0.124
PPBS	0.438	0.102
Age	-0.186	0.507
BMI	0.224	0.422

†Statistically significant (p  $\leq$  0.05)

Table 5: Multiple Linear Regression Analysis for Predictors of CCT in Diabetic Group

Variable	β Coefficient	Standard Error	P-value
Age	-0.156	0.284	0.586
<b>Duration of DM</b>	0.324	0.156	0.042†
HbA1c	0.468	0.198	0.022†
FBS	0.246	0.168	0.148
BMI	0.188	0.224	0.406

†Statistically significant (p  $\leq$  0.05)

 $R^2 = 0.542$ , Adjusted  $R^2 = 0.486$ 

# **DISCUSSION**

The present study demonstrates a significant association between diabetes mellitus and increased central corneal thickness. Our findings of higher CCT in diabetic patients (mean difference  $24.4~\mu m$ ) align with several previous studies. Ozdamar Y et al.(13) reported a similar

increase in CCT among diabetic patients, attributing this to altered corneal hydration status and structural changes in the corneal stroma.

The significant correlation between CCT and HbA1c levels (r=0.526) observed in our study supports the findings of Zhao H et al.(14), who demonstrated that poor glycemic control contributes to corneal structural alterations. This relationship may be explained by the accumulation of advanced glycation end products (AGEs) in corneal tissue, as proposed by Lee et al.(6).

Our multiple regression analysis identified disease duration as an independent predictor of CCT, consistent with the findings of Storr-Paulsen et al.(7), who reported progressive corneal changes with increasing duration of diabetes. This temporal relationship suggests cumulative effects of chronic hyperglycemia on corneal structure.

The comparable IOP readings between groups despite CCT differences highlight the importance of considering CCT when interpreting tonometry readings in diabetic patients, as emphasized by Peters et al.(8). This has significant implications for glaucoma screening and management in the diabetic population.

The mechanism underlying increased CCT in diabetes likely involves multiple pathways. Wilson et al.(9) proposed that endothelial dysfunction and altered corneal hydration control contribute to these changes.

# **CONCLUSION**

This study demonstrates significantly increased central corneal thickness in diabetic patients compared to non-diabetic controls, with HbA1c levels and disease duration serving as independent predictors. These findings highlight the importance of considering diabetes status when interpreting corneal thickness measurements and emphasize the need for regular monitoring of corneal parameters in diabetic patients.

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