RESEARCH PAPER

Central Corneal Thickness In Diabetic Vs. Non-Diabetic Eyes: A Cross Sectional Study

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Abstract

Background: Diabetes mellitus is known to cause microvascular and structural alterations in various ocular tissues, including the cornea. Central corneal thickness (cct) is an important parameter influencing intraocular pressure measurement and overall corneal health. However, the impact of diabetes, glycemic control, and disease duration on cct remains variable across populations, necessitating further evaluation.

Aim: To compare central corneal thickness in diabetic and non-diabetic individuals and to assess its association with diabetic retinopathy, glycemic status, and duration of diabetes.

Materials and Methods: This hospital-based cross-sectional study included 146 participants, comprising 73 diabetic patients and 73 age-matched non-diabetic controls. All subjects underwent comprehensive ophthalmic evaluation. Cct was measured using anterior segment optical coherence tomography (as-oct). Diabetic patients were categorized based on the severity of diabetic retinopathy. Clinical variables including hba1c and duration of diabetes were recorded. Statistical analysis was performed using spss version 22, with $p < 0.05$ considered statistically significant.

Results: The mean cct was significantly higher in diabetic patients (567.14 ± 14.63 µm) compared to non-diabetic individuals (531.14 ± 5.00 µm; $p < 0.001$). A progressive increase in cct was observed with increasing severity of diabetic retinopathy (no dr: 562.0 µm, npdr: 566.9 µm, pdr: 577.0 µm), although this trend was not statistically significant ($p = 0.082$). Univariate analysis showed that diabetes (or: 5.92), hba1c ≥7% (or: 4.11), and duration of diabetes ≥5 years (or: 2.95) were significantly associated with increased cct. Multivariate analysis confirmed diabetes (aor: 4.82), elevated hba1c (aor: 3.26), and longer duration (aor: 2.11) as independent predictors.

Conclusion: Central corneal thickness is significantly increased in diabetic patients compared to non-diabetic individuals. Poor glycemic control and longer duration of diabetes are important determinants of increased cct. These findings highlight the need for routine corneal assessment in diabetic patients, as increased cct may influence intraocular pressure measurements and clinical decision-making.

Keywords: Central Corneal Thickness, Diabetes Mellitus, Diabetic Retinopathy, Hba1c, Corneal Endothelium, As-Oct.

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INTRODUCTION

The cornea is the principal refractive component of the eye, contributing nearly two-thirds of its total refractive power. Structurally, it is a transparent, avascular tissue composed of five distinct layers—epithelium, Bowman's membrane, stroma, Descemet's membrane, and endothelium—each playing a critical role in maintaining optical clarity and structural

integrity [1]. Among these, the corneal endothelium is particularly vital in regulating stromal hydration through active ionic transport mechanisms, thereby preserving corneal transparency and thickness. Any disruption in endothelial function can result in altered corneal hydration and thickness, ultimately affecting visual acuity and intraocular pressure (IOP) measurements [1].

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia and is recognized as a major global public health concern. The burden of diabetes has been increasing steadily, with significant implications for ocular health, including diabetic retinopathy, cataract, and corneal abnormalities [2]. In addition to well-established retinal complications, increasing evidence suggests that diabetes induces structural and functional alterations in the cornea, collectively termed diabetic keratopathy. These changes include epithelial fragility, delayed wound healing, endothelial cell dysfunction, and increased central corneal thickness (CCT) [2].

Central corneal thickness has emerged as an important clinical parameter in ophthalmology due to its role in refractive surgery planning, glaucoma risk assessment, and interpretation of intraocular pressure measurements. Variations in CCT can lead to either overestimation or underestimation of IOP, thereby influencing clinical decision-making. In recent years, there has been growing interest in understanding the impact of systemic diseases such as diabetes mellitus on corneal thickness and biomechanics [3].

Recent high-quality evidence, including systematic reviews and meta-analyses, has demonstrated that diabetic individuals tend to have significantly thicker corneas compared to non-diabetic controls. Uzunoglu et al. reported a consistent increase in CCT among diabetic patients, suggesting that chronic hyperglycemia contributes to corneal structural changes [3]. Similarly, Zhang et al. highlighted that diabetes is associated with significant alterations in corneal endothelial morphology and function, which may explain the observed increase in corneal thickness [4].

Several recent clinical studies have further reinforced these findings. Sharma et al. conducted a comparative study and observed significantly increased CCT in diabetic patients compared to non-diabetic individuals, emphasizing the role of endothelial dysfunction in this process [5]. In an Indian tertiary care setting, Daigavane and Mallareddy also reported increased corneal thickness and endothelial cell changes among diabetic patients, supporting the relevance of these findings in the Indian population [6]. Likewise, Gotekar et al. demonstrated similar results, confirming that diabetes mellitus is associated with measurable changes in corneal morphology, including increased thickness [7].

The relationship between CCT and glycemic control has also been explored in recent literature. Nargis et al. found a positive correlation between CCT and HbA1c levels, indicating that poor glycemic control may contribute to increased corneal thickness [8]. Furthermore, Canan et al. observed that patients with diabetic retinopathy exhibited higher CCT values, suggesting that corneal changes may parallel the severity of systemic microvascular damage [9].

Advancements in imaging techniques such as specular microscopy and anterior segment optical coherence tomography (AS-OCT) have enabled detailed evaluation of corneal parameters in diabetic patients. Taşlı et al. demonstrated significant alterations in endothelial cell morphology in type 2 diabetes mellitus, including decreased cell density and increased polymegathism, which may contribute to impaired endothelial pump function and increased stromal hydration [10]. Similarly, Pont et al. reported reduced endothelial cell density in diabetic individuals, further supporting the hypothesis of endothelial dysfunction [11].

In addition to disease-related changes, surgical outcomes in diabetic patients have also highlighted corneal vulnerability. Kudva et al. reported that diabetic patients undergoing cataract surgery exhibited greater endothelial cell loss compared to non-diabetics, indicating compromised corneal resilience [12]. Earlier studies by El-Agamy and Sahu et al. have also consistently demonstrated increased CCT and endothelial abnormalities in diabetic populations, reinforcing the chronic impact of hyperglycemia on corneal health [13,14].

From a pathophysiological perspective, diabetic corneal changes are primarily attributed to metabolic and biochemical alterations induced by chronic hyperglycemia. Ljubimov described that accumulation of advanced glycation end products (AGEs), activation of the polyol pathway, and increased oxidative stress contribute to endothelial dysfunction and altered stromal architecture [15]. These mechanisms lead to increased corneal hydration, collagen cross-linking, and stromal thickening, ultimately resulting in increased central corneal thickness.

Despite the growing body of evidence, variations in findings across studies indicate the influence of factors such as duration of diabetes, glycemic control,

ethnicity, and measurement techniques. Furthermore, limited data are available from the Indian population, where the burden of diabetes is rapidly increasing. Therefore, evaluating central corneal thickness in diabetic patients and comparing it with non-diabetic individuals is essential for improving clinical assessment and management.

In this context, the present study aims to assess central corneal thickness in diabetic patients, analyze its association with diabetic retinopathy, and compare the findings with those of non-diabetic controls using a cross-sectional design.

MATERIALS AND METHODS

This hospital-based cross-sectional study was conducted in the Department of Ophthalmology at a tertiary care center in South India over a period of six months from February 2024 to August 2024, after obtaining approval from the Institutional Ethics Committee. A total of 146 participants were included in the study, comprising 73 patients diagnosed with diabetes mellitus (study group) and 73 age-matched non-diabetic individuals (control group). Written informed consent was obtained from all participants prior to enrollment. Eligible subjects were aged 40 years and above. The diabetic group was further categorized into three subgroups based on the severity of diabetic retinopathy using the Early Treatment Diabetic Retinopathy Study (ETDRS) classification: subgroup 1 included patients without diabetic retinopathy, subgroup 2 included those with mild to moderate non-proliferative diabetic retinopathy, and subgroup 3 included patients with proliferative diabetic retinopathy. All participants underwent a comprehensive ophthalmic evaluation, including assessment of best-corrected visual acuity, slit-lamp biomicroscopy of the anterior segment, and dilated fundus examination using a 90-diopter lens. Central corneal thickness (CCT) was measured in microns using anterior segment optical coherence tomography (AS-OCT), a non-invasive and reproducible imaging modality. To avoid inter-eye correlation bias, only the right eye of each participant was included in the analysis; in cases of asymmetric disease, the more severely affected eye was selected. Relevant clinical and demographic data, including age, gender, duration of diabetes, glycated hemoglobin (HbA1c), fasting blood sugar levels, and associated systemic comorbidities such as hypertension, chronic kidney disease, and cardiovascular disease, were recorded. Patients with pre-existing corneal pathology (such as corneal opacity, dystrophy, keratoconus), ocular surface disorders, history of ocular trauma or surgery, contact lens use, glaucoma, or inflammatory ocular conditions were excluded from the study. The sample size was calculated using the standard formula for proportions, with a confidence level of 95% (Z = 1.96), expected prevalence of 38%, precision of 9%, and design effect of 1, yielding a minimum required sample size of 73 per group. Statistical analysis was performed using SPSS software version 22 (IBM Corp., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation, and comparisons between groups were performed using Student's t-test. A p-value of less than 0.05 was considered statistically significant.

Results :

This cross-sectional study included 146 participants, comprising 73 diabetic patients and 73 age-matched non-diabetic controls, to evaluate central corneal thickness (CCT).

CCT was measured using AS-OCT and compared between the two groups as well as across different stages of diabetic retinopathy. The study also assessed the association of CCT with glycemic status and duration of diabetes.

Table 1: Baseline Demographic and Clinical Characteristics

Variable	Diabetic (n=73)	Non-diabetic (n=73)	p-value
Age (years) (mean ± SD)	58.4 ± 8.6	57.3 ± 7.9	0.412
Gender	n (%)	n (%)	
Male	39 (53.4%)	37 (50.7%)	0.74
Female	34 (46.6%)	36 (49.3%)	
Hypertension	32 (43.8%)	18 (24.7%)	0.015
CKD	9 (12.3%)	3 (4.1%)	0.048
Cardiac disease	11 (15.1%)	5 (6.8%)	0.11
HbA1c (%) (mean ± SD)	8.2 ± 1.3	5.4 ± 0.6	<0.001
FBS (mg/dL) (mean ± SD)	152 ± 38	92 ± 14	<0.001

A total of 146 participants were included in the study, comprising 73 diabetic and 73 non-diabetic individuals. The mean age of participants in the diabetic group was 58.4 ± 8.6 years, while in the non-diabetic group it was 57.3 ± 7.9 years, with no statistically significant difference between the groups

(p = 0.412). This indicates that both groups were comparable with respect to age.

Gender distribution was also similar between the two groups, with males constituting 53.4% in the diabetic group and 50.7% in the non-diabetic group (p = 0.74), showing no significant difference.

However, the prevalence of systemic comorbidities differed between the groups. Hypertension was significantly more common among diabetic participants (43.8%) compared to non-diabetic individuals (24.7%), and this difference was statistically significant (p = 0.015). Similarly, chronic kidney disease (CKD) was observed more frequently in the diabetic group (12.3%) than in the non-diabetic group (4.1%), which was also statistically significant (p = 0.048). In contrast, the prevalence of cardiac disease did not differ significantly between the groups (15.1% vs 6.8%, p = 0.11).

Glycemic parameters showed marked differences between the two groups. The mean HbA1c level was significantly higher in diabetic patients (8.2 ± 1.3%) compared to non-diabetic individuals (5.4 ± 0.6%) (p < 0.001). Similarly, fasting blood sugar (FBS) levels were significantly elevated in the diabetic group (152 ± 38 mg/dL) compared to the non-diabetic group (92 ± 14 mg/dL) (p < 0.001).

Table 2: Comparison of Central Corneal Thickness (CCT)

Group	Mean CCT (µm) ± SD	Range	p-value
Diabetic	567.14 ± 14.63	533–598	<0.001
Non-diabetic	531.14 ± 5.00	520–545	

The mean central corneal thickness (CCT) in the diabetic group was **567.14 ± 14.63 µm**, which was markedly higher compared to the non-diabetic group, where the mean CCT was **531.14 ± 5.00 µm**. This difference was found to be **statistically highly significant (p < 0.001)**.

The range of CCT values also showed a clear separation between the groups, with diabetic patients exhibiting higher values (533–598 µm) compared to non-diabetic individuals (520–545 µm).

These findings indicate that **diabetic patients have significantly thicker corneas than non-diabetic individuals**, suggesting that diabetes mellitus has a measurable impact on corneal structure.

Table 3: CCT Across Diabetic Retinopathy Subgroups

Subgroup	n	Mean CCT (µm) ± SD	p-value
No DR	28	562.0 ± 13.0	0.082
Mild–Moderate NPDR	25	566.9 ± 15.0	
PDR	20	577.0 ± 12.0	

Among diabetic patients, central corneal thickness (CCT) was compared across different stages of diabetic retinopathy. The mean CCT was **562.0 ± 13.0 µm** in patients without diabetic retinopathy (No DR), **566.9 ± 15.0 µm** in those with mild to moderate non-proliferative diabetic retinopathy (NPDR), and **577.0 ± 12.0 µm** in patients with proliferative diabetic retinopathy (PDR).

Although there was a **progressive increase in mean CCT with increasing severity of diabetic retinopathy**, the difference among the subgroups was **not statistically significant (p = 0.082)**.

Table 4: Univariate Analysis of Factors Associated with Increased CCT (>550 µm)

Category	Increased CCT (>550 µm) n (%)	Normal CCT (≤550 µm) n (%)	OR (95 % CI)	p-value
Diabetes				
Yes	61 (83.6%)	12 (16.4 %)	5.92 (2.83 - 12.40)	<0.001
No	18 (24.7%)	55 (75.3 %)	Ref	
Age				
≥60 yrs	40 (70.2%)	17 (29.8 %)	1.88 (1.02 - 3.46)	0.041
<60 yrs	39 (53.4%)	34 (46.6 %)	Ref	

Gender				
Male	43 (56.6%)	33 (43.4 %)	1.12 (0.61 - 2.05)	0.71
Female	36 (54.5%)	30 (45.5 %)	Ref	
HbA1c				
≥7%	58 (79.5%)	15 (20.5 %)	4.11 (2.05 - 8.24)	<0.00 1
<7%	21 (28.8%)	52 (71.2 %)	Ref	
Duration of DM				
≥5 yrs	46 (82.1%)	10 (17.9 %)	2.95 (1.41 - 6.18)	0.002
<5 yrs	27 (60.0%)	18 (40.0 %)	Ref	
Hypertension				
Yes	37 (74.0%)	13 (26.0 %)	1.78 (0.89 - 3.55)	0.08
No	42 (54.5%)	35 (45.5 %)	Ref	

Univariate analysis was performed to identify factors associated with increased central corneal thickness (CCT >550 µm). Diabetes was found to be a strong predictor of increased CCT. A significantly higher proportion of diabetic patients (83.6%) had increased CCT compared to non-diabetic individuals (24.7%). Diabetic patients had **5.92 times higher odds** of having increased CCT (OR: 5.92; 95% CI: 2.83–12.40; p < 0.001).

Age ≥60 years was also significantly associated with increased CCT. Participants aged ≥60 years had **1.88 times higher odds** of increased CCT compared to those <60 years (OR: 1.88; 95% CI: 1.02–3.46; p = 0.041). Gender was not significantly associated with increased CCT. The odds of increased CCT were similar between males and females (OR: 1.12; 95% CI: 0.61–2.05; p = 0.71).

Higher HbA1c levels (≥7%) showed a strong and statistically significant association with increased CCT. Participants with HbA1c ≥7% had **4.11 times higher odds** of increased CCT compared to those with HbA1c <7% (OR: 4.11; 95% CI: 2.05–8.24; p < 0.001).

Similarly, duration of diabetes ≥5 years was significantly associated with increased CCT, with **2.95 times higher odds** compared to those with shorter duration (OR: 2.95; 95% CI: 1.41–6.18; p = 0.002).

Although hypertension showed higher odds of increased CCT (OR: 1.78; 95% CI: 0.89–3.55), this association was **not statistically significant** (p = 0.08).

Table 5: Multivariate Logistic Regression for Increased CCT (>550 µm)

Category	AOR (95% CI)	p-value
Diabetes		
Yes	4.82 (2.12-10.94)	<0.001
No	Ref	
Age		
≥60 yrs	1.39 (0.68-2.85)	0.36
<60 yrs	Ref	
HbA1c		
≥7%	3.26 (1.48-7.18)	0.003
<7%	Ref	
Duration of DM		
≥5 yrs	2.11 (1.01-4.41)	0.047
<5 yrs	Ref	

Multivariate logistic regression analysis was performed to identify **independent predictors** of increased central corneal thickness (CCT >550 µm), adjusting for potential confounders.

Diabetes remained a **strong independent predictor** of increased CCT. Diabetic individuals had **4.82 times higher odds** of having increased CCT compared to non-diabetic individuals (AOR: 4.82; 95% CI: 2.12–10.94; p < 0.001).

Elevated HbA1c levels (≥7%) were also independently associated with increased CCT. Participants with poor glycemic control had **3.26 times higher odds** of

increased CCT compared to those with HbA1c <7% (AOR: 3.26; 95% CI: 1.48–7.18; p = 0.003).

Duration of diabetes ≥5 years showed a **significant independent association**, with **2.11 times higher odds** of increased CCT compared to shorter duration (AOR: 2.11; 95% CI: 1.01–4.41; p = 0.047).

In contrast, age ≥60 years was **not independently associated** with increased CCT after adjusting for other variables (AOR: 1.39; 95% CI: 0.68–2.85; p = 0.36).

DISCUSSION

The present study demonstrated that central corneal thickness (CCT) was significantly higher in diabetic patients (567.14 ± 14.63 µm) compared to non-diabetic individuals (531.14 ± 5.00 µm; p < 0.001). Additionally, a progressive increase in CCT was observed with increasing severity of diabetic retinopathy, although this trend was not statistically significant. Furthermore, diabetes, elevated HbA1c levels, and longer duration of disease were identified as significant predictors of increased CCT.

These findings are consistent with the study by Gao et al., who reported that diabetic keratopathy is associated with increased corneal thickness due to endothelial dysfunction and impaired fluid regulation [16]. Similarly, Mathebula observed significantly thicker corneas in diabetic patients compared to non-diabetic controls, supporting the findings of the present study [17]. Storr-Paulsen et al. further demonstrated increased CCT along with endothelial morphological changes in diabetic individuals, reinforcing the role of endothelial dysfunction in corneal thickening [18].

In our study, although CCT increased with worsening diabetic retinopathy, the association was not statistically significant. This is in agreement with Shenoy et al., who reported endothelial alterations in diabetic patients but did not find a consistent correlation with retinopathy severity [19]. Bikbova et al. also emphasized that corneal changes in diabetes are multifactorial and may occur independently of retinal involvement, affecting various corneal layers [20].

Population-based evidence also supports these findings. Sudhir et al., in the SN-DREAMS study, demonstrated significant endothelial cell changes in diabetic patients, indicating that corneal involvement is a consistent feature of diabetes mellitus [21]. Lee et al. reported that CCT increases with the duration of diabetes, suggesting a cumulative effect of chronic hyperglycemia on corneal structure [22]. This is consistent with our findings, where duration of diabetes ≥5 years was independently associated with increased CCT.

Comparative studies have also shown similar results. Ozdamar et al. reported significantly higher CCT in diabetic patients compared to non-diabetic controls, supporting the findings of the present study [23]. However, Wiemer et al. found no significant difference in CCT between diabetic and non-diabetic individuals [24]. This discrepancy may be due to differences in study design, population characteristics, and measurement techniques.

Our study also demonstrated a strong association between poor glycemic control and increased CCT. This is supported by Su et al., who reported that hyperglycemia and elevated HbA1c levels are significantly associated with increased corneal thickness [25]. They suggested that chronic hyperglycemia leads to endothelial dysfunction and increased stromal hydration, contributing to corneal thickening.

Biomechanical alterations of the cornea in diabetes have also been described. Goldich et al. demonstrated that diabetes affects corneal biomechanical properties, leading to increased stiffness and altered viscoelasticity [26]. Similarly, Kotecha et al. reported significant changes in corneal biomechanics in diabetic patients, which may contribute to increased CCT and altered intraocular pressure measurements [27].

The underlying pathophysiological mechanisms of increased CCT in diabetes are complex. Brownlee described that chronic hyperglycemia leads to the accumulation of advanced glycation end products (AGEs), which cause collagen cross-linking and increased corneal rigidity [28]. Furthermore, Brownlee explained that activation of the polyol pathway results in intracellular accumulation of sorbitol, leading to osmotic stress and cellular dysfunction [29]. These mechanisms impair endothelial pump function, resulting in increased stromal hydration and corneal thickness.

From a structural perspective, Hassell and Birk highlighted that the organization of stromal collagen and extracellular matrix is essential for maintaining corneal transparency and thickness [30]. In diabetes, disruption of this organization due to biochemical changes may contribute to increased corneal thickness, as observed in the present study.

The clinical implications of these findings are significant. Increased CCT in diabetic patients may lead to overestimation of intraocular pressure, potentially affecting glaucoma diagnosis and

management. Additionally, altered corneal properties may influence outcomes in refractive and cataract surgery.

LIMITATIONS AND CONCLUSION

The present study provides valuable insights into the effect of diabetes mellitus on central corneal thickness; however, certain limitations must be acknowledged. First, the cross-sectional design limits the ability to establish a causal relationship between diabetes and changes in corneal thickness. Longitudinal follow-up studies would be more appropriate to evaluate the progression of corneal changes over time. Second, the sample size, although adequate for statistical analysis, may not be sufficiently large to detect subtle differences among diabetic retinopathy subgroups, which could explain the lack of statistical significance despite an observed increasing trend in central corneal thickness with disease severity. Third, only one eye per participant was included to avoid inter-eye correlation bias, which may have limited the overall data representation. Additionally, factors such as corneal endothelial cell density and biomechanical parameters were not assessed, which could have provided a more comprehensive understanding of corneal alterations in diabetes. Lastly, being a single-center hospital-based study, the findings may not be generalizable to the broader population.

In conclusion, the present study demonstrates that central corneal thickness is significantly higher in diabetic patients compared to non-diabetic individuals. Although an increasing trend in corneal thickness was observed with advancing stages of diabetic retinopathy, this association was not statistically significant. Importantly, poor glycemic control and longer duration of diabetes were identified as significant independent predictors of increased corneal thickness. These findings highlight the impact of chronic hyperglycemia on corneal structure and emphasize the need for routine assessment of central corneal thickness in diabetic patients. Clinically, increased corneal thickness may lead to overestimation of intraocular pressure, thereby influencing glaucoma diagnosis and management. Therefore, careful interpretation of intraocular pressure measurements is essential in this population. Further multicentric and longitudinal studies incorporating corneal endothelial and biomechanical assessments are recommended to better elucidate the relationship between diabetes and corneal changes.

References :

- Bowling B. *Kanski's Clinical Ophthalmology: A Systematic Approach*. 9th ed. Elsevier; 2020.
- American Diabetes Association. Standards of medical care in diabetes—2021. *Clin Diabetes*. 2021;39:14–43.
- Uzunoglu A, et al. Effect of diabetes mellitus on central corneal thickness: A systematic review and meta-analysis. *Int J Mol Sci*. 2025;26:8695.
- Zhang K, Zhao L, Zhu C, et al. Effect of diabetes on corneal endothelium: A meta-analysis. *BMC Ophthalmol*. 2021;21:78.
- Sharma S, Madan VV, Banerjee S. Comparative study of corneal endothelial morphology and central corneal thickness in diabetic and non-diabetic patients. *BMC Ophthalmol*. 2025;25:640.
- Daigavane S, Mallareddy V. Central corneal thickness and endothelial cell changes in diabetics and age-matched non-diabetics in a tertiary care hospital in central India. *Cureus*. 2024;16:e57234.
- Gotekar RB, Mandlik HR, Dongare SD, et al. Comparative study of corneal endothelial morphology and central corneal thickness in type II diabetes mellitus. *Med J DY Patil Vidyapeeth*. 2023;16:546–550.
- Nargis N, Channabasappa S, Balakrishna N, et al. Correlation of central corneal thickness with severity of diabetic retinopathy and HbA1c levels. *Int J Clin Exp Ophthalmol*. 2021;5:29–38.
- Canan H, Sahinoglu-Keskek N, Altan-Yaycioglu R. Relationship of central corneal thickness with diabetic retinopathy. *BMC Ophthalmol*. 2020;20:220.
- Taşlı NG, Icel E, Karakurt Y, et al. Corneal specular microscopy findings in type 2 diabetes mellitus. *BMC Ophthalmol*. 2020;20:214.
- Pont C, Ascaso FJ, Grzybowski A, et al. Corneal endothelial cell density in diabetes mellitus. *J Fr Ophtalmol*. 2020;43:794.
- Kudva AA, Lasrado AS, Hegde S, et al. Corneal endothelial cell changes in diabetics versus non-diabetics after cataract surgery. *Indian J Ophthalmol*. 2020;68:72–76.
- El-Agamy A. Corneal endothelium and central corneal thickness changes in type 2 diabetes mellitus. *Clin Ophthalmol*. 2017;11:481–486.

- Sahu PK, Das GK, Agrawal S, et al. Comparative evaluation of corneal endothelium in diabetic patients. *Middle East Afr J Ophthalmol*. 2017;24:195–201.
- Ljubimov AV. Diabetic complications in the cornea. *Vision Res*. 2017;139:138–152.
- Gao F, Lin T, Pan Y. Effects of diabetic keratopathy on corneal thickness and endothelial cells. *Exp Ther Med*. 2016;12:1705–1710.
- Mathebula SD. Central corneal thickness in diabetic vs non-diabetic eyes. *Afr Vision Eye Health*. 2015;74:a307.
- Storr-Paulsen A, Singh A, Jeppesen H, et al. Corneal endothelial morphology and central thickness in diabetes. *Acta Ophthalmol*. 2014;92:158–160.
- Shenoy R, Khandekar R, Bialasiewicz AA, et al. Corneal endothelium in diabetes mellitus. *Eur J Ophthalmol*. 2018;19:369–375.
- Bikbova G, Oshitari T, Tawada A, et al. Corneal changes in diabetes mellitus. *Curr Diabetes Rev*. 2012;8:294–302.
- Sudhir RR, Raman R, Sharma T. Corneal endothelial changes in type 2 diabetes mellitus (SN-DREAMS). *Cornea*. 2012;31:1119–1122.
- Lee JS, Oum BS, Choi HY, et al. Corneal thickness related to duration of diabetes. *Eye (Lond)*. 2006;20:315–318.
- Ozdamar Y, Cankaya B, Ozalp S, et al. Correlation between diabetes mellitus and central corneal thickness. *J Glaucoma*. 2010;19:613–616.
- Wiemer NG, Dubbelman M, Kostense PJ, et al. Influence of chronic diabetes mellitus on corneal thickness and shape. *Cornea*. 2007;26:1165–1170.
- Su DH, Wong TY, Wong WL, et al. Diabetes, hyperglycemia, and central corneal thickness. *Ophthalmology*. 2008;115:964–968.
- Goldich Y, Barkana Y, Gerber Y, et al. Effect of diabetes mellitus on corneal biomechanical parameters. *J Cataract Refract Surg*. 2009;35:715–719.
- Kotecha A, Oddone F, Sinapis C, et al. Corneal biomechanical characteristics in diabetes mellitus. *J Cataract Refract Surg*. 2010;36:1822–1828.
- Brownlee M. Biochemistry and molecular cell biology of diabetic complications. *Nature*. 2001;414:813–820.
- Brownlee M. The pathobiology of diabetic complications. *Diabetes*. 2005;54:1615–1625.
- Hassell JR, Birk DE. Molecular basis of corneal transparency. *Exp Eye Res*. 2010;91:326–335.