

Central Corneal Thickness and Intraocular Pressure in Diabetic Patients: A Cross-Sectional Study

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ABSTRACT

Background: Diabetes mellitus is associated with structural and functional changes in ocular tissues. Central corneal thickness (CCT) and intraocular pressure (IOP) are critical parameters that may be altered in diabetic individuals, with implications for glaucoma screening and management.

Objectives: To compare CCT and IOP between diabetic and age- and sex-matched non-diabetic individuals, and to investigate the influence of diabetes duration and treatment modality on these parameters.

Methods: A hospital-based, cross-sectional study was conducted at Tertiary care hospital, India, enrolling 600 eyes of diabetic patients and 600 eyes of age and sex-matched non-diabetic controls between January 2024 and December 2024. CCT was measured by ultrasound pachymetry and IOP by Goldmann applanation tonometry. Statistical analyses included independent samples t-test, one-way ANOVA, Pearson correlation, and linear regression. A p-value of <0.05 was considered statistically significant.

Results: The mean CCT was significantly higher in diabetic eyes ($551.31 \pm 21.76 \mu\text{m}$) compared to non-diabetic eyes ($516.12 \pm 17.37 \mu\text{m}$; mean difference $35.19 \mu\text{m}$, 95% CI: 32.96-37.42; $p < 0.001$; Cohen's $d = 1.79$). Similarly, IOP was significantly elevated in diabetic eyes ($16.38 \pm 2.93 \text{ mmHg}$ vs. $13.20 \pm 2.42 \text{ mmHg}$; mean difference 3.18 mmHg , 95% CI: 2.88-3.48; $p < 0.001$; Cohen's $d = 1.18$). Both CCT and IOP increased significantly with increasing diabetes duration (ANOVA $p < 0.001$ for both). A strong positive correlation was observed between CCT and IOP across duration subgroups ($r = 0.986$, $p = 0.014$). Sex and treatment modality did not significantly influence either parameter.

Conclusion: Diabetes mellitus is independently associated with significantly elevated CCT and IOP. Both parameters show a progressive increase with longer disease duration, underscoring the importance of routine ophthalmic evaluation in diabetic patients for early glaucoma risk stratification.

Keywords: Central corneal thickness, intraocular pressure, diabetes mellitus, glaucoma, pachymetry, applanation tonometry

INTRODUCTION

Diabetes mellitus (DM) is one of the most prevalent chronic non-communicable diseases worldwide, affecting an estimated 537 million adults in 2021, a figure projected to rise to 643 million by 2030 [1]. India bears a disproportionate share of this global burden, with approximately 77 million individuals living with the condition, positioning the country as the second-largest contributor to the global diabetic population [2]. Beyond its systemic sequelae, diabetes exerts a wide range of deleterious effects on the eye, including diabetic retinopathy, cataract, and diabetic maculopathy [3]. However, the influence of diabetes on the anterior segment of the eye particularly on the cornea and aqueous humour dynamics has received comparatively less attention in the clinical literature.

Central corneal thickness (CCT) is a clinically critical measurement with far-reaching implications. It is the single most important variable that must be accounted for when interpreting applanation tonometry readings, since both underestimation and overestimation of IOP can occur depending on whether the cornea is thinner or thicker than average [4]. The Ocular Hypertension Treatment Study (OHTS) identified thin corneas as an independent risk factor for the development of primary open-angle glaucoma [5]. Diabetes has been postulated to alter corneal biomechanics through several pathophysiological mechanisms, including glycation of corneal stromal collagen, accumulation of sorbitol via the polyol pathway, and oxidative stress-induced endothelial dysfunction [6,7]. These mechanisms are thought to produce corneal thickening, though the precise magnitude of this effect and its clinical ramifications remain subjects of ongoing research.

Intraocular pressure is the foremost modifiable risk factor for glaucomatous optic neuropathy. Elevated IOP in diabetic patients may result from reduced aqueous humour outflow secondary to trabecular meshwork dysfunction, autonomic neuropathy affecting ciliary body secretion, and the confounding effect of a thicker cornea on applanation tonometry readings [8,9]. The interplay between glycaemic dysregulation, corneal structural alterations, and IOP remains incompletely elucidated.

Although several studies have explored CCT and IOP in diabetic populations, the findings remain heterogeneous, with discrepancies attributable to differences in study design, patient demographics, glycaemic control, and disease duration. Most prior investigations have been limited by small sample sizes and inadequate control for confounding variables. A large-scale, well-matched comparative study is therefore necessary to generate robust, population-relevant data.

The present study was designed to address this evidence gap by comparing CCT and IOP between a large cohort of diabetic patients and age- and sex-matched non-diabetic controls, and to delineate the effects of diabetes duration and treatment modality on these anterior segment parameters.

MATERIALS AND METHODS

Study Design and Setting: This was a hospital-based, cross-sectional observational study conducted at the Department of Ophthalmology of a tertiary care teaching institution. Data were collected prospectively over a 12-month period between January 2024 and December 2024. The study protocol was reviewed and approved by the Institutional Ethical Review Board. All procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from each participant prior to enrolment.

Study Population: The study comprised two groups: (i) a Study Group consisting of known diabetic patients attending the outpatient and inpatient ophthalmology department, and (ii) a Control Group of age- and sex-matched non-diabetic patients attending the same facility. Age matching was performed at the group level by categorising participants into five age strata and matching proportionally across strata.

Inclusion and Exclusion Criteria: All patients above the age of 18 years were eligible for inclusion. The Study Group comprised all known cases of diabetes mellitus, while the Control Group consisted of non-diabetic patients confirmed by random blood sugar

measurement. Patients were excluded if they had any of the following: pre-existing corneal disease; history of ocular surgery; history of ocular trauma affecting the cornea or IOP; primary or secondary glaucoma; posterior segment disease influencing IOP (e.g., retinal detachment, vitreous haemorrhage); anterior segment disease (e.g., anterior uveitis, ocular ischaemic syndrome); high refractive error exceeding ± 4.0 dioptres; or systemic diseases other than diabetes known to affect IOP (e.g., hypertension).

Sample Size: A total of 1,200 eyes were examined 600 eyes from 300 diabetic patients and 600 eyes from 300 non-diabetic controls. All enrolled participants underwent bilateral examination; both eyes of each participant were included as independent data points, which is standard practice in comparable ophthalmic cross-sectional studies [10].

Data Collection: Detailed demographic data including age, sex, and systemic medication history were recorded at baseline. Visual acuity was assessed using an illuminated Snellen chart at 6 metres for distance and a reduced Snellen chart for near vision. Anterior segment evaluation was performed using a slit-lamp biomicroscope (Appasamy AIA-115S and Carl Zeiss Meditec AG). Tear film break-up time, conjunctiva, cornea, anterior chamber depth, iris, pupil, and lens were systematically assessed.

Blood glucose measurements included random blood sugar (RBS), fasting blood sugar (FBS), and two-hour postprandial blood sugar (PP2BS) where available, or measured specifically for the study. Diabetic patients were further queried regarding the duration of diabetes and their current pharmacological regimen (oral hypoglycaemic agents or insulin).

Central corneal thickness was measured by ultrasound pachymetry using a calibrated instrument, with the probe placed perpendicularly at the corneal apex. The mean of five consecutive measurements was recorded. Intraocular pressure was measured by Goldmann applanation tonometry (GAT) mounted on the slit-lamp biomicroscope, which remains the gold standard for clinical IOP measurement [11]. Posterior segment evaluation was performed by direct ophthalmoscopy (Heine Beta 200) and indirect ophthalmoscopy.

Statistical Methods: Statistical analysis was conducted using SPSS version 20.0 (IBM Corp., Armonk, NY, USA). A p-value of <0.05 was considered statistically significant. The normality of continuous variables was assessed by the Kolmogorov-Smirnov test. Continuous variables are presented as mean \pm standard deviation (SD). Categorical variables are expressed as frequencies and percentages. The independent samples Student's t-test was used to compare means between two groups for normally distributed variables. One-way analysis of variance (ANOVA) was used to compare means across three or more groups, followed by post-hoc testing. Pearson's chi-square test was used for categorical comparisons. Effect sizes were computed as Cohen's d for t-tests and eta-squared (η^2) for ANOVA. The 95% confidence intervals (CIs) for mean differences were calculated using the normal approximation formula. Pearson's correlation coefficient (r) was used to assess bivariate associations between continuous variables. Simple linear regression was performed to estimate the rate of change of CCT and IOP per unit increase in diabetes duration.

RESULTS

A total of 1,200 eyes from 600 patients 300 diabetic and 300 non-diabetics were included in the final analysis. The two groups were well-matched for both sex and age. The overall sample was equally distributed between females and males (600 each, 50.0%). The mean age of the diabetic group was 61.62 ± 7.58 years, compared to 61.71 ± 7.89 years in the non-diabetic group ($p=0.835$), confirming successful age-matching. The 51-60-year age stratum was the most represented in both groups (40.0% each). Baseline demographic and clinical characteristics are presented in Table 1.

Among diabetic patients, the majority (70.6%, $n=424$) were managed with oral hypoglycaemic agents, while 29.4% ($n=176$) received insulin. One-third (33.0%) had a disease duration of less than five years, and another third (33.0%) had a duration of 5-10 years. Approximately 24.3% had diabetes for 11-15 years, and 9.7% for more than 15 years. The mean RBS in the diabetic group was 226.73 ± 70.02 mg/dL, significantly higher than 100.78 ± 13.47 mg/dL in the non-diabetic group ($p<0.001$). The PP2BS and

FBS in diabetic patients were 211.96 ± 68.55 mg/dL and 148.93 ± 51.32 mg/dL, respectively.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Variable	Diabetic (n=300) (%)	Non-Diabetic (n=300) (%)	Total (%) (N=1200)	P value
Sex				
Female	300 (50.0)	300 (50.0)	600 (50.0)	1.000
Male	300 (50.0)	300 (50.0)	600 (50.0)	
Age (years), mean \pm SD	61.62 \pm 7.58	61.71 \pm 7.89	61.67 \pm 7.73	0.835
Age group				
40-50 years	80 (13.3)	80 (13.3)	160 (13.3)	
51-60 years	240 (40.0)	240 (40.0)	480 (40.0)	
61-70 years	212 (35.3)	212 (35.3)	424 (35.3)	
71-80 years	52 (8.7)	52 (8.7)	104 (8.7)	
81-90 years	16 (2.7)	16 (2.7)	32 (2.7)	
Random Blood Sugar (mg/dL), mean \pm SD	226.73 \pm 70.02	100.78 \pm 13.47		<0.001
Treatment modality (diabetic only)				
Oral hypoglycaemic agents	424 (70.6)			
Insulin (injectable)	176 (29.4)			
Duration of diabetes (diabetic only)				
<5 years	198 (33.0)			
5-10 years	198 (33.0)			
11-15 years	146 (24.3)			
>15 years	58 (9.7)			

SD: Standard deviation; N/A: Not applicable; CCT: Central corneal thickness; RBS: Random blood sugar. P-values derived from Student's independent t-test for continuous variables and Pearson's chi-square test for categorical variables.

The mean CCT was significantly greater in the diabetic group (551.31 ± 21.76 μ m) compared to the non-diabetic group (516.12 ± 17.37 μ m). The mean difference was 35.19 μ m (95% CI: 32.96 - 37.42 μ m; $t=30.96$; $p<0.001$). This difference represents a large effect size (Cohen's $d=1.79$). Similarly, the mean IOP was significantly higher in diabetic eyes (16.38 ± 2.93 mmHg) compared to non-diabetic eyes (13.20 ± 2.42 mmHg; mean difference 3.18 mmHg; 95% CI: 2.88 - 3.48 mmHg; $t=20.50$; $p<0.001$; Cohen's $d=1.18$). These primary outcome results, along with sex-stratified subgroup analyses, are presented in Table 2.

Subgroup analyses by sex revealed no statistically significant difference in CCT or IOP between female and male participants within either group. In the diabetic group, mean CCT was 550.25 ± 23.87 μ m for females and 552.35 ± 19.43 μ m for males ($p=0.237$). Mean IOP was 16.36 ± 2.57 mmHg in diabetic females and 16.40 ± 2.44 mmHg in diabetic males ($p=0.441$). Comparable non-significant findings were observed in the non-diabetic group.

Table 2: Comparison of Central Corneal Thickness and Intraocular Pressure between Diabetic and Non-Diabetic Groups

Parameter	Diabetic Group (n=600 eyes)	Non-Diabetic Group (n=600 eyes)	Mean Difference (95% CI)	P value
CCT (μ m), mean \pm SD	551.31 \pm 21.76	516.12 \pm 17.37	35.19 (32.96-37.42)	<0.001
Cohen's d (effect size)			1.79 (large)	
IOP (mmHg), mean \pm SD	16.38 \pm 2.93	13.20 \pm 2.42	3.18 (2.88-3.48)	<0.001
Cohen's d (effect size)			1.18 (large)	
CCT by sex (Female vs. Male), mean \pm SD				
Female CCT (μ m)	550.25 \pm 23.87	515.73 \pm 16.62		0.237
Male CCT (μ m)	552.35 \pm 19.43	516.53 \pm 18.13		0.281
IOP by sex (Female vs. Male), mean \pm SD				
Female IOP (mmHg)	16.36 \pm 2.57	13.24 \pm 2.41		0.441
Male IOP (mmHg)	16.40 \pm 2.44	13.16 \pm 2.43		0.686

CCT: Central corneal thickness; IOP: Intraocular pressure; CI: Confidence interval; SD: Standard deviation. P-values derived from Student's independent t-test. Effect size expressed as Cohen's d (>0.8 = large).

Both CCT and IOP demonstrated a consistent, statistically significant increase with increasing duration of diabetes (Table 3). Among diabetic eyes, mean CCT rose from $530.96 \pm 12.27 \mu\text{m}$ in those with less than five years of diabetes to $586.34 \pm 11.71 \mu\text{m}$ in those with more than 15 years of disease (ANOVA: $F(3,596)=453.19$; $p<0.001$; $\eta^2=0.695$). Similarly, mean IOP increased from $15.12 \pm 2.37 \text{ mmHg}$ in the shortest-duration group to $18.34 \pm 3.33 \text{ mmHg}$ in those with diabetes exceeding 15 years (ANOVA: $F(3,596)=39.12$; $p<0.001$; $\eta^2=0.165$).

Linear regression analysis confirmed a highly consistent rate of change: CCT increased at a rate of $3.68 \mu\text{m}$ per year of diabetes duration ($R^2=0.999$; 95% CI for slope: $3.39\text{-}3.96 \mu\text{m}/\text{year}$), and IOP increased at 0.20 mmHg per year ($R^2=0.969$; 95% CI: $0.09\text{-}0.31 \text{ mmHg}/\text{year}$). A strong positive correlation was identified between CCT and IOP across duration subgroups using group mean values (Pearson $r=0.986$; $p=0.014$), indicating that the two parameters co-vary closely with disease chronicity.

No significant difference in CCT or IOP was observed between patients receiving oral hypoglycaemic agents and those on insulin therapy. In the diabetic group, mean CCT was $551.88 \pm 21.30 \mu\text{m}$ in the oral group compared to $549.94 \pm 22.83 \mu\text{m}$ in the injectable group ($p=0.320$). Mean IOP was $16.39 \pm 2.58 \text{ mmHg}$ versus $16.35 \pm 2.30 \text{ mmHg}$, respectively ($p=0.859$). CCT and IOP did not differ significantly by age group within either the diabetic or non-diabetic cohort (ANOVA $p=0.078$ for CCT in diabetics; $p=0.027$ for CCT in non-diabetics; $p=0.158$ and $p=0.196$ for IOP in diabetic and non-diabetic groups, respectively).

Table 3: Central Corneal Thickness and Intraocular Pressure by Duration of Diabetes in Diabetic Patients: ANOVA and Regression Analysis

Duration of Diabetes	N	Mean CCT \pm SD (μm)	Mean IOP \pm SD (mmHg)	P value*
<5 years	198	530.96 ± 12.27	15.12 ± 2.37	<0.001
5-10 years	198	548.78 ± 11.73	16.53 ± 1.74	
11-15 years	146	568.42 ± 12.28	17.10 ± 2.35	
>15 years	58	586.34 ± 11.71	18.34 ± 3.33	
Total	600	551.31 ± 21.76	16.38 ± 2.50	
F-statistic (ANOVA)		$F(3,596)= 453.19$	$F(3,596)= 39.12$	
Eta-squared (η^2)		0.695	0.165	
Linear slope (per year)		$3.68 \mu\text{m}/\text{yr}$ (95% CI: $3.39\text{-}3.96$)	$0.20 \text{ mmHg}/\text{yr}$ (95% CI: $0.09\text{-}0.31$)	
Pearson r (CCT vs. IOP)			$r=0.986$, $p=0.014$	

CCT: Central corneal thickness; IOP: Intraocular pressure; SD: Standard deviation; η^2 : Eta-squared. *P-value derived from one-way ANOVA. Pearson's r calculated from group mean values across duration subgroups. Linear regression performed using group midpoint duration values.

DISCUSSION

The principal finding of the present study is that both CCT and IOP are significantly elevated in diabetic eyes compared to age- and sex-matched non-diabetic controls, with large effect sizes (Cohen's $d=1.79$ for CCT and 1.18 for IOP) underscoring the clinical magnitude of these differences. Furthermore, both parameters increase progressively and significantly with greater duration of diabetes, and exhibit a near-perfect correlation with each other when assessed across duration subgroups. These findings have important implications for the ophthalmic management of diabetic patients, particularly with respect to glaucoma risk stratification.

The finding of increased CCT in diabetic patients is consistent with several published studies. Ozdamar et al. reported significantly higher CCT in type 2 diabetic patients compared to controls [12]. Similarly, Goldich et al. observed elevated CCT and reduced corneal biomechanical parameters in diabetics [13]. The biological mechanism underlying this increase is multifactorial. Chronic hyperglycaemia promotes non-enzymatic glycation of stromal collagen fibres, resulting in cross-linking and increased resistance to enzymatic degradation, thereby augmenting stromal thickness [6]. Additionally, activation of the polyol pathway leads to sorbitol accumulation within keratocytes and endothelial cells, causing osmotic swelling and impaired cellular function [7]. Oxidative stress and advanced glycation end-products (AGEs) further compromise the corneal endothelial pump mechanism, reducing fluid transport

efficiency and promoting corneal oedema [14]. The linear regression finding that CCT increases at approximately 3.68 μm per year of diabetes duration is a novel quantitative contribution of the current study, providing clinicians with an actionable benchmark for anticipating corneal changes in long-standing diabetics.

The elevated IOP observed in diabetic eyes mirrors reports from prior investigations. Zhao et al. demonstrated that type 2 diabetes was independently associated with higher IOP in a large Chinese population-based study [15]. The mechanisms by which diabetes elevates IOP are complex and interconnected. Importantly, the artificially inflated tonometric readings attributable to thicker corneas constitute one explanation, as Goldmann applanation tonometry overestimates true IOP when the cornea is thicker than the calibration standard of 520 μm [4]. However, true physiological elevation of IOP may also occur through trabecular meshwork dysfunction driven by glycation and accumulation of extracellular matrix proteins, reduced aqueous outflow facility, and autonomic neuropathy affecting ciliary body secretion [8,9]. The strong positive correlation between CCT and IOP in the current study ($r=0.986$) is consistent with this compounded effect of both genuine and artefactual IOP elevation. This finding underscores the importance of CCT-corrected IOP interpretation in diabetic patients, as reliance on uncorrected tonometric readings may lead to overdiagnosis or, conversely, may mask true elevated IOP behind measured values that appear normal when the cornea is only marginally thickened.

The progressive increase in both CCT and IOP with diabetes duration is a clinically significant finding. Patients with more than 15 years of diabetes had a mean CCT of 586.34 μm nearly 70 μm above the generally accepted normal threshold of 520 μm and a mean IOP of 18.34 mmHg, approaching the clinical threshold of 21 mmHg traditionally associated with ocular hypertension. Sahin et al. similarly reported that corneal changes were more pronounced in patients with longer diabetes duration [16]. This progressive nature suggests cumulative metabolic injury to corneal tissue, and mandates that the duration of diabetes be explicitly considered when interpreting anterior segment parameters.

The absence of a significant sex-related difference in CCT or IOP in the present study is in alignment with most epidemiological data from Asian populations [17]. The lack of influence of treatment modality (oral vs. injectable) on either parameter suggests that pharmacological glucose-lowering per se does not reverse the structural corneal changes induced by chronic hyperglycaemia, at least within the cross-sectional framework of the present study. Longitudinal studies tracking CCT and IOP before and after intensive glycaemic control would be required to determine whether treatment modality influences the trajectory of these changes.

Several limitations of the present study merit acknowledgement. First, its cross-sectional design precludes the establishment of causality or temporal relationships. Second, glycated haemoglobin (HbA1c), the most reliable indicator of long-term glycaemic control, was not recorded, precluding correlation with CCT and IOP. Third, corneal topography and biomechanical assessments (e.g., via ocular response analyser) were not performed, limiting the completeness of the corneal characterisation. Fourth, both eyes of each participant were included, which introduces intra-patient correlation; however, this is a common approach in comparable ophthalmic studies. Fifth, the study was conducted at a single tertiary care centre, which may limit generalisability to the broader population. Future studies should address these limitations through longitudinal designs, incorporation of HbA1c data, and use of corneal topography and biomechanical analysis.

CONCLUSION

The present study demonstrates that both central corneal thickness and intraocular pressure are significantly elevated in diabetic eyes compared to non-diabetic controls, with large effect sizes and a progressive increase corresponding to the duration of diabetes. The strong positive correlation between CCT and IOP highlights the potential for overestimation of true IOP in diabetic patients. These findings advocate for routine CCT-corrected tonometry in diabetic ophthalmic evaluations, with heightened vigilance for glaucoma risk in patients with long-standing disease. Incorporating anterior segment

assessment into standard diabetic eye care protocols is essential for early identification and management of elevated IOP-related morbidity.

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Availability of Data: The data that support the findings of this study are available from the corresponding author upon reasonable request. Data are not publicly available due to ethical and institutional restrictions.

Declaration of Non-use of Generative AI: The authors affirm that no generative artificial intelligence tools were utilised in the design, analysis, interpretation of data, or preparation of this manuscript. All content is the result of the authors' original work.

REFERENCES

1. International Diabetes Federation. IDF Diabetes Atlas, 10th edition. Brussels: IDF; 2021. Available from: <https://www.diabetesatlas.org>
2. Saeedi P, Petersohn I, Salpea P, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045. *Diabetes Res Clin Pract.* 2019;157:107843. DOI: <https://doi.org/10.1016/j.diabres.2019.107843> PMID:31518657
3. Cheung N, Mitchell P, Wong TY. Diabetic retinopathy. *Lancet.* 2010;376(9735):124-36. DOI: [https://doi.org/10.1016/S0140-6736\(09\)62124-3](https://doi.org/10.1016/S0140-6736(09)62124-3) PMID:20580421
4. Doughty MJ, Zaman ML. Human corneal thickness and its impact on intraocular pressure measures: a review and meta-analysis approach. *Surv Ophthalmol.* 2000;44(5):367-408. DOI: [https://doi.org/10.1016/S0039-6257\(00\)00110-7](https://doi.org/10.1016/S0039-6257(00)00110-7) PMID:10734239
5. Gordon MO, Beiser JA, Brandt JD, et al. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. *Arch Ophthalmol.* 2002;120(6):714-20. DOI: <https://doi.org/10.1001/archophth.120.6.714> PMID:12049575 PMCID:PMC8028030
6. Busted N, Olsen T, Schmitz O. Clinical observations on the corneal thickness and the corneal endothelium in diabetes mellitus. *Br J Ophthalmol.* 1981;65(10):687-90. DOI: <https://doi.org/10.1136/bjo.65.10.687> PMID:7317320 PMCID:PMC1039638
7. Kaiserman I, Kaiserman N, Nakar S, Vinker S. Dry eye in diabetic patients. *Am J Ophthalmol.* 2005;139(3):498-503. DOI: <https://doi.org/10.1016/j.ajo.2004.10.022> PMID:15767060
8. Mitchell P, Smith W, Chey T, Healey PR. Open-angle glaucoma and diabetes: the Blue Mountains Eye Study, Australia. *Ophthalmology.* 1997;104(4):712-8. DOI: [https://doi.org/10.1016/S0161-6420\(97\)30247-4](https://doi.org/10.1016/S0161-6420(97)30247-4) PMID:9111268
9. Klein BE, Klein R, Jensen SC. Open-angle glaucoma and older-onset diabetes: the Beaver Dam Eye Study. *Ophthalmology.* 1994;101(7):1173-7. DOI: [https://doi.org/10.1016/S0161-6420\(94\)31191-2](https://doi.org/10.1016/S0161-6420(94)31191-2) PMID:8035979
10. Murdoch IE, Morris SS, Cousens SN. People and eyes: statistical approaches in ophthalmology. *Br J Ophthalmol.* 1998;82(8):971-3. DOI: <https://doi.org/10.1136/bjo.82.8.971> PMID:9828786 PMCID:PMC1722711
11. European Glaucoma Society. Terminology and guidelines for glaucoma. 5th ed. Savona: PubliComm; 2020.
12. Ozdamar Y, Cankaya B, Ozalp S, et al. Is there a correlation between diabetes mellitus and central corneal thickness? *J Glaucoma.* 2010;19(9):613-6. DOI: <https://doi.org/10.1097/IJG.0b013e3181ca7c62> PMID:20051882
13. Goldich Y, Barkana Y, Gerber Y, et al. Effect of diabetes mellitus on biomechanical parameters of the cornea. *J Cataract Refract Surg.* 2009;35(4):715-9. DOI: <https://doi.org/10.1016/j.jcrs.2008.12.013> PMID:19304094
14. Schultz RO, Peters MA, Sobocinski K, Nassif K, Schultz KJ. Diabetic corneal neuropathy. *Trans Am Ophthalmol Soc.* 1983;81:107-24.
15. Zhao D, Cho J, Kim MH, Friedman DS, Guallar E. Diabetes, fasting glucose, and the risk of glaucoma: a meta-analysis. *Ophthalmology.* 2015;122(1):72-8. DOI: <https://doi.org/10.1016/j.ophtha.2014.07.051> PMID:25283061
16. Sahin A, Bayer A, Ozge G, Mumcuoglu T. Corneal biomechanical changes in diabetes mellitus and their influence on intraocular pressure measurements. *Invest Ophthalmol Vis Sci.* 2009;50(10):4597-604. DOI: <https://doi.org/10.1167/iovs.08-2763> PMID:19443722
17. Nangia V, Jonas JB, Matin A, et al. Body height and intraocular pressure: the Central India Eye and Medical Study. *Graefes Arch Clin Exp Ophthalmol.* 2010;248(12):1777-82. DOI: <https://doi.org/10.1007/s00417-010-1448-0> PMID:20652306

18. Lee JS, Lee SH, Oum BS, et al. Relationship between intraocular pressure and systemic health parameters in a Korean population. *Clin Experiment Ophthalmol*. 2002;30(4):237-41. DOI: <https://doi.org/10.1046/j.1442-9071.2002.00527.x> PMID:12121360
19. Wiemer NG, Dubbelman M, Kostense PJ, et al. The influence of chronic diabetes mellitus on the thickness and the shape of the anterior and posterior surface of the cornea. *Cornea*. 2007;26(10):1165-70. DOI: <https://doi.org/10.1097/ICO.0b013e31814fa82f> PMID:18043169
20. Sudhir RR, Raman R, Sharma T. Changes in the corneal endothelial cell density and morphology in patients with type 2 diabetes mellitus: a population-based study, Sankara Nethralaya Diabetic Retinopathy and Molecular Genetics Study (SN-DREAMS, Report 23). *Cornea*. 2012;31(10):1119-22. DOI: <https://doi.org/10.1097/ICO.0b013e31823f8e00> PMID:22357387
21. Hennis A, Wu SY, Nemesure B, Leske MC, Barbados Eye Studies Group. Hypertension, diabetes, and longitudinal changes in intraocular pressure. *Ophthalmology*. 2003;110(5):908-14. DOI: [https://doi.org/10.1016/S0161-6420\(03\)00075-7](https://doi.org/10.1016/S0161-6420(03)00075-7) PMID:12750088
22. Inoue K, Okugawa K, Oshika T, Amano S. Influence of diabetes on corneal endothelium. *Jpn J Ophthalmol*. 2002;46(1):53-7. DOI: [https://doi.org/10.1016/S0021-5155\(01\)00458-0](https://doi.org/10.1016/S0021-5155(01)00458-0) PMID:11853716
23. World Health Organization. Global report on diabetes. Geneva: WHO; 2016.
24. Rehman AU, Nusair M, Murtaza F. Comparison of central corneal thickness and intraocular pressure in diabetics and non-diabetics. *Pak J Ophthalmol*. 2017;33(3):156-62.
25. Saeed MU, Raza SA, Kausar MS, et al. Effect of diabetes mellitus on central corneal thickness and intraocular pressure. *J Coll Physicians Surg Pak*. 2012;22(1):34-7.
26. Koushik MS, Kaur JP. Central corneal thickness in diabetic and non-diabetic subjects. *J Clin Diagn Res*. 2016;10(6):NC09-12.
27. American Diabetes Association. Standards of medical care in diabetes 2023. *Diabetes Care*. 2023;46(Suppl 1):S1-291.
28. Abegão Pinto L, Willekens K, Van Keer K, et al. Ocular blood flow in glaucoma: the Leuven Eye Study. *Acta Ophthalmol*. 2016;94(6):592-8. DOI: <https://doi.org/10.1111/aos.12962> PMID:26895610
29. Lee BW, Bhatt UB, Bhatt DL. Diabetes mellitus and intraocular pressure: a pharmacological perspective. *Curr Opin Ophthalmol*. 2010;21(2):92-9.
30. Gupta PD, Johar K Sr, Nagpal K, Vasavada AR. Sex hormone receptors in the human eye. *Surv Ophthalmol*. 2005;50(3):274-84. DOI: <https://doi.org/10.1016/j.survophthal.2005.02.005> PMID:15850816