

Evaluation of Intraocular Pressure in Fasting and Postprandial Glycemic Status in Type 2 Diabetes Mellitus Patients: An Analytical Comparative Study

Suresh Kumar S.¹, Sakthi Kesavan S.², Jakkidi Prathiba Reddy³, Karthikeyan R.⁴

¹Associate Professor, Departmental of Anesthesia, Fathima Institute of Medical Sciences, Kadapa, Mariyapuram, Andhra Pradesh.

²Assistant Professor, Departmental of Orthopedics, Meenakshi Medical College Hospital and Research Institute, Enathur, Kanchipuram, Tamil Nadu

³Associate Professor, Department of Ophthalmology, Dhanalakshmi Srinivasan Medical College and Hospital, Siruvachur Post, Perambalur District, Tamil Nadu.

⁴Associate Professor, Department of Psychiatry, Nandha Medical College and Hospital Erode District, Tamil Nadu.

Received: 27-11-2024 / Revised: 21-12-2024 / Accepted: 29-12-2024

Corresponding author: Dr. Karthikeyan R

Conflict of interest: Nil

Abstract

Purpose: To evaluate variations in intraocular pressure (IOP) during fasting and postprandial glycemic states among patients with Type 2 Diabetes Mellitus (T2DM), compare findings with non-diabetic controls, and assess the influence of central corneal thickness (CCT) and duration of diabetes on IOP.

Methods: A hospital-based analytical comparative study was conducted among 60 participants comprising 30 patients with T2DM and 30 age- and sex-matched healthy controls. Fasting blood sugar (FBS) and postprandial blood sugar (PPBS) were measured using standard biochemical methods. Intraocular pressure was assessed during fasting and postprandial states using calibrated tonometry, while central corneal thickness was measured using ultrasonic pachymetry. Demographic, clinical, and ocular parameters were recorded. Statistical analysis was performed using SPSS version 25.0. Independent sample t-test, paired t-test, Pearson correlation coefficient, and descriptive statistics were employed. A p-value <0.05 was considered statistically significant.

Results: Diabetic participants demonstrated significantly higher fasting and postprandial blood glucose levels compared with controls. Mean fasting IOP in diabetics was 16.3 ± 2.1 mmHg and increased significantly to 18.1 ± 2.3 mmHg in the postprandial state. In contrast, non-diabetic participants exhibited minimal variation in IOP. A significant positive correlation was observed between PPBS and IOP among diabetic individuals ($r = 0.610$, $p < 0.001$), whereas the correlation between FBS and IOP was weaker ($r = 0.274$, $p = 0.049$). Mean central corneal thickness was significantly greater among diabetics. Participants with thicker corneas demonstrated higher IOP values. Furthermore, a progressive increase in postprandial IOP was observed with increasing duration of diabetes.

Conclusion: Postprandial hyperglycemia is significantly associated with elevated intraocular pressure among patients with Type 2 Diabetes Mellitus. The influence of glycemic status on IOP is further modified by central corneal thickness and duration of disease. Routine assessment of postprandial IOP and corneal thickness may improve early identification of diabetic individuals at increased risk of ocular hypertension and glaucoma.

Keywords: Type 2 Diabetes Mellitus; Intraocular Pressure; Postprandial Hyperglycemia; Central Corneal Thickness; Ocular Hypertension; Glaucoma; Diabetic Eye Disease; Glycemic Variability.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Type 2 Diabetes Mellitus (T2DM) represents one of the most significant public health challenges of the twenty-first century [1]. According to the International Diabetes Federation, the global prevalence of diabetes continues to increase at an alarming rate, particularly in low- and middle-income countries. India is currently recognized as

one of the countries with the largest diabetic populations worldwide, and the burden is projected to increase substantially over the coming decades [2]. The chronic hyperglycemic state associated with diabetes contributes to widespread microvascular and macrovascular complications that significantly impair quality of life and increase

healthcare expenditure [3]. Ocular manifestations of diabetes have traditionally focused on diabetic retinopathy, which remains the leading cause of preventable blindness among working-age adults [4]. However, emerging evidence indicates that diabetes affects virtually every ocular structure, including the cornea, lens, retina, optic nerve, and aqueous humor drainage pathways. These changes may contribute to alterations in intraocular pressure (IOP), a critical determinant of glaucoma risk [5].

Glaucoma is the second leading cause of irreversible blindness globally. Elevated intraocular pressure remains the most important modifiable risk factor for glaucomatous optic neuropathy. Although glaucoma is a multifactorial disease, numerous epidemiological studies have demonstrated a positive association between diabetes mellitus and elevated IOP. The mechanisms underlying this relationship are complex and incompletely understood. Proposed explanations include increased aqueous humor production secondary to hyperglycemia-induced osmotic changes, impaired trabecular meshwork function, extracellular matrix remodeling, oxidative stress, endothelial dysfunction, and chronic low-grade inflammation [6-8].

Hyperglycemia may influence aqueous humor dynamics through multiple pathways. Elevated blood glucose levels can alter osmotic gradients between the plasma and intraocular compartments, resulting in changes in aqueous humor formation and drainage. Chronic hyperglycemia may also induce structural modifications within the trabecular meshwork, reducing aqueous outflow facility and subsequently elevating IOP. Furthermore, advanced glycation end-products (AGEs), which accumulate in diabetic tissues, have been implicated in extracellular matrix deposition and tissue stiffness, potentially affecting ocular biomechanics and aqueous humor circulation [9-10].

Most previous investigations evaluating the relationship between diabetes and intraocular pressure have relied on fasting glucose measurements or glycated hemoglobin (HbA1c) levels. However, postprandial hyperglycemia is increasingly recognized as an independent predictor of vascular complications and may exert distinct physiological effects compared with fasting hyperglycemia. Acute postprandial glucose excursions are associated with increased oxidative stress, endothelial dysfunction, inflammatory activation, and hemodynamic changes. These effects may have immediate consequences for ocular physiology and intraocular pressure regulation [11].

The clinical significance of postprandial IOP fluctuations remains underexplored. In routine

ophthalmic practice, intraocular pressure is often measured at a single time point without consideration of glycemic status. Consequently, transient elevations in IOP related to postprandial glucose surges may remain undetected. Such fluctuations could be particularly important among diabetic individuals who are already predisposed to glaucoma and other ocular complications [12].

Another important factor influencing the interpretation of intraocular pressure measurements is central corneal thickness (CCT). Goldmann applanation tonometry, considered the clinical gold standard for IOP assessment, assumes an average corneal thickness. Variations in corneal thickness can result in overestimation or underestimation of true intraocular pressure. Several studies have reported increased central corneal thickness among diabetic patients, possibly due to endothelial dysfunction, altered corneal hydration, and metabolic changes affecting corneal structure. Therefore, assessment of corneal thickness is essential for accurate interpretation of IOP values in diabetic populations [13].

The duration of diabetes may also play a critical role in determining ocular outcomes. Prolonged exposure to hyperglycemia contributes to cumulative tissue damage, microvascular dysfunction, and extracellular matrix alterations. These changes may progressively impair aqueous humor drainage mechanisms and lead to sustained elevation of intraocular pressure. Investigating the relationship between disease duration and IOP may therefore provide valuable insights into the long-term ocular consequences of diabetes [14].

Despite increasing evidence linking diabetes with elevated intraocular pressure, relatively few studies have simultaneously evaluated fasting and postprandial glycemic states, central corneal thickness, and duration of disease within a single analytical framework. Moreover, data from South Indian populations remain limited. Given the high prevalence of diabetes and the growing burden of glaucoma in India, understanding these relationships has important implications for clinical screening, risk stratification, and preventive ophthalmology.

The present study was therefore undertaken to compare fasting and postprandial intraocular pressure among patients with Type 2 Diabetes Mellitus and healthy controls, evaluate the influence of central corneal thickness on IOP measurements, and investigate the relationship between glycemic status and ocular pressure dynamics. By identifying factors associated with elevated IOP in diabetes, this study aims to contribute to improved ophthalmic surveillance and early detection of glaucoma risk among diabetic individuals.

Aim and Objectives

Aim: To evaluate intraocular pressure in fasting and postprandial glycemic states among patients with Type 2 Diabetes Mellitus and compare the findings with non-diabetic controls.

Objectives

1. To compare fasting intraocular pressure between patients with Type 2 Diabetes Mellitus and healthy controls.
2. To compare postprandial intraocular pressure between diabetic and non-diabetic individuals.
3. To assess the magnitude of intraocular pressure changes between fasting and postprandial states.
4. To determine the relationship between fasting blood glucose levels and intraocular pressure.
5. To determine the relationship between postprandial blood glucose levels and intraocular pressure.
6. To evaluate the influence of central corneal thickness on intraocular pressure measurements.
7. To assess the association between duration of diabetes and postprandial intraocular pressure.
8. To identify factors contributing to elevated intraocular pressure among patients with Type 2 Diabetes Mellitus.

Materials and Methods

Study Design: This hospital-based analytical comparative cross-sectional study was conducted to investigate the relationship between glycemic status and intraocular pressure among patients with Type 2 Diabetes Mellitus and healthy controls.

Study Setting: The study was carried out in the Departments of Ophthalmology and General Medicine of a tertiary care teaching hospital in South India over a one-year period from January 2023 to December 2023.

Study Population: The study population comprised adult patients attending outpatient services during the study period. Participants were categorized into a diabetic group and a non-diabetic control group.

Sample Size: A total of 60 participants were included in the final analysis, consisting of 30 patients diagnosed with Type 2 Diabetes Mellitus and 30 age- and sex-matched healthy controls.

Sampling Technique: Purposive sampling was employed for participant recruitment. Eligible participants who fulfilled the selection criteria and provided informed written consent were consecutively enrolled until the desired sample size was achieved.

Inclusion Criteria: Participants were eligible for inclusion if they fulfilled all the following criteria:

Diabetic Group

1. Adults aged between 30 and 70 years.
2. Confirmed diagnosis of Type 2 Diabetes Mellitus according to American Diabetes Association (ADA) criteria.
3. Duration of diabetes of at least one year.
4. Patients receiving oral hypoglycemic agents, insulin therapy, or a combination of both.
5. Ability and willingness to provide written informed consent.

Control Group

1. Adults aged between 30 and 70 years.
2. Age- and sex-matched healthy individuals.
3. Normal fasting and postprandial blood glucose levels.
4. No history of diabetes mellitus or impaired glucose tolerance.
5. Willingness to participate and provide written informed consent.

Exclusion Criteria

Participants were excluded if they had any of the following:

1. Previous diagnosis of glaucoma or ocular hypertension.
2. History of ocular surgery.
3. History of ocular trauma.
4. Corneal disorders including keratoconus, corneal dystrophies, or corneal scars.
5. Significant refractive error exceeding ± 5 diopters.
6. Active ocular infection or inflammation.
7. Current or recent corticosteroid therapy.
8. Systemic diseases known to affect intraocular pressure.
9. Neurological disorders affecting visual function.
10. Pregnancy or lactation.
11. Inability to cooperate with ophthalmic examination procedures.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Human Ethics Committee before initiation of participant recruitment. All procedures adhered to the ethical principles outlined in the Declaration of Helsinki. Participants received detailed information regarding the study objectives, methodology, benefits, and potential risks before enrollment. Written informed consent was obtained from all participants. Confidentiality of collected information was maintained throughout the study.

Clinical Evaluation: All participants underwent comprehensive clinical evaluation. A detailed medical history was obtained, including age, sex, and duration of diabetes, treatment history, systemic comorbidities, medication usage, and ocular history. General physical examination and

ophthalmic assessment were performed by trained investigators.

Demographic variables were recorded using a pre-designed data collection form. Duration of diabetes was documented from medical records and participant interviews. Body mass index and blood pressure measurements were recorded whenever available.

Biochemical Assessment

Fasting Blood Sugar (FBS): Participants were instructed to maintain an overnight fast of at least 8–10 hours. Venous blood samples were collected in the morning before breakfast.

Fasting blood glucose concentration was estimated using a standardized automated biochemical analyzer in the central clinical laboratory.

Postprandial Blood Sugar (PPBS): Following a standard breakfast, blood samples were obtained two hours after meal consumption. Postprandial blood glucose levels were measured using the same laboratory methodology employed for fasting samples to ensure consistency.

Ophthalmic Examination: All ophthalmic assessments were conducted under standardized environmental conditions by trained ophthalmologists.

Visual Acuity Assessment: Best-corrected visual acuity was assessed using Snellen's visual acuity chart under standard illumination conditions.

Anterior Segment Examination: Anterior segment evaluation was performed using slit-lamp biomicroscopy to exclude corneal pathology, active inflammation, or other ocular abnormalities that could influence intraocular pressure measurements.

Fundus Examination: Dilated fundus examination was performed whenever indicated to rule out advanced retinal pathology or optic disc abnormalities suggestive of glaucoma.

Intraocular Pressure Measurement: Intraocular pressure was measured using a calibrated non-contact tonometer under standardized conditions.

Measurements were performed at two distinct time points:

Fasting IOP Measurement: IOP was recorded during the fasting state before breakfast and before blood sample collection. Participants were seated comfortably for at least five minutes before examination.

Postprandial IOP Measurement: A second IOP measurement was obtained two hours after consumption of a standard breakfast, coinciding with postprandial blood glucose assessment. For each eye, three consecutive readings were obtained.

The average value was considered for statistical analysis. To minimize measurement variability, all assessments were performed by the same examiner whenever possible.

Central Corneal Thickness Measurement: Central corneal thickness was measured using ultrasonic pachymetry.

Participants were instructed to fixate on a distant target while measurements were obtained. Three central corneal readings were recorded for each eye and averaged for analysis.

Based on pachymetric values, participants were categorized into the following groups:

- CCT <530 μm
- CCT 530–560 μm
- CCT >560 μm

This categorization enabled assessment of the influence of corneal thickness on intraocular pressure measurements.

Outcome Measures

Primary Outcome

1. Difference in fasting and postprandial intraocular pressure among diabetic and non-diabetic participants.

Secondary Outcomes

2. Correlation between fasting blood glucose and intraocular pressure.
3. Correlation between postprandial blood glucose and intraocular pressure.
4. Influence of central corneal thickness on intraocular pressure.
5. Association between duration of diabetes and postprandial intraocular pressure.
6. Distribution of intraocular pressure changes after meals.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software version 25.0.

Continuous variables were expressed as mean \pm standard deviation (SD), while categorical variables were presented as frequencies and percentages.

The following statistical tests were applied:

1. Independent sample t-test for comparison of continuous variables between diabetic and non-diabetic groups.
2. Paired t-test for comparison of fasting and postprandial intraocular pressure within groups.
3. Pearson correlation coefficient for assessing relationships between glycemic parameters and intraocular pressure.

4. Chi-square test for categorical variables where appropriate.
5. Descriptive statistical analysis for demographic and clinical characteristics.

A p-value less than 0.05 was considered statistically significant.

Results

Baseline Characteristics of Study Participants:

A total of 60 participants were included in the study, comprising 30 patients with Type 2 Diabetes Mellitus and 30 age- and sex-matched healthy

controls. The demographic profiles of both groups were comparable, minimizing confounding due to age and sex. The mean age of diabetic participants was 56.2 ± 7.8 years, compared with 54.9 ± 6.5 years among controls. Male participants constituted 60.0% of the diabetic group and 56.7% of the control group. Both fasting and postprandial blood glucose levels were significantly higher among diabetic participants, confirming appropriate classification of study groups.

Table 1: Baseline Demographic and Clinical Characteristics

Variable	Diabetic Group (n=30)	Control Group (n=30)	p-value
Age (years)	56.2 ± 7.8	54.9 ± 6.5	0.462
Male (%)	60.0	56.7	0.792
Female (%)	40.0	43.3	0.792
FBS (mg/dL)	142.6 ± 18.9	89.3 ± 7.4	<0.001
PPBS (mg/dL)	216.4 ± 24.1	124.6 ± 10.2	<0.001

The study groups were well matched with respect to age and gender distribution. As expected, diabetic individuals exhibited significantly elevated fasting and postprandial blood glucose levels compared with controls, confirming the metabolic distinction between the groups.

Comparison of Intraocular Pressure in Fasting and Postprandial States: A significant increase in postprandial intraocular pressure was observed among diabetic participants.

Table 2: Comparison of Fasting and Postprandial Intraocular Pressure

Variable	Diabetic Group	Control Group	p-value
Fasting IOP (mmHg)	16.3 ± 2.1	14.9 ± 1.8	0.003
Postprandial IOP (mmHg)	18.1 ± 2.3	15.1 ± 2.0	0.001
Change in IOP (mmHg)	$+1.8 \pm 0.9$	$+0.2 \pm 0.4$	<0.001

Diabetic participants demonstrated significantly higher fasting and postprandial intraocular pressure compared with controls. The mean postprandial increase of 1.8 mmHg suggests that acute glycemic fluctuations exert measurable effects on ocular pressure regulation.

Influence of Central Corneal Thickness on Intraocular Pressure: Central corneal thickness

(CCT) was evaluated to determine its effect on measured intraocular pressure values. Diabetic participants demonstrated significantly thicker corneas than non-diabetic controls.

Furthermore, a progressive increase in IOP was observed with increasing corneal thickness in both groups.

Table 3: Central Corneal Thickness and Intraocular Pressure

CCT Category (μm)	Diabetic IOP (Mean \pm SD)	Control IOP (Mean \pm SD)	
<530	15.9 ± 1.8	13.7 ± 1.6	
530–560	17.2 ± 2.3	14.5 ± 1.9	
>560	18.1 ± 2.5	15.3 ± 2.0	
Variable	Diabetics	Controls	p-value
Mean CCT (μm)	556.2 ± 23.4	541.6 ± 20.8	0.011

The diabetic group exhibited significantly thicker corneas than controls. Increasing corneal thickness was associated with higher measured IOP values, emphasizing the importance of pachymetric correction during glaucoma screening in diabetic populations.

Correlation between Glycemic Status and Intraocular Pressure: Pearson correlation analysis was performed to determine the relationship between blood glucose levels and intraocular pressure.

Table 4: Correlation between Glycemic Parameters and IOP

Group	FBS vs IOP (r)	p-value	PPBS vs IOP (r)	p-value
Diabetic	0.274	0.049	0.610	<0.001
Control	0.102	0.301	0.180	0.212

Among diabetic participants, PPBS showed a strong positive correlation with IOP ($r = 0.610$), whereas FBS demonstrated only a weak correlation. These findings suggest that acute postprandial glycemic excursions may exert a greater influence on intraocular pressure than fasting glycemia.

Distribution of Intraocular Pressure Changes between Fasting and Postprandial States

To better understand the magnitude of postprandial IOP elevation, participants were categorized according to the degree of IOP change.

Table 5: Distribution of Postprandial IOP Elevation

IOP Elevation Category	Diabetic's n (%)	Controls n (%)
No change (<1 mmHg)	4 (13.3%)	24 (80.0%)
Mild increase (1–2 mmHg)	14 (46.7%)	6 (20.0%)
Significant increase (>2 mmHg)	12 (40.0%)	0 (0%)

Forty percent of diabetic patients demonstrated clinically significant postprandial IOP elevation greater than 2 mmHg, whereas none of the controls exhibited comparable changes. This observation highlights the impact of glycemic fluctuations on ocular pressure regulation.

Association between Duration of Diabetes and Postprandial IOP: The diabetic group was stratified according to duration of disease.

Table 6: Duration of Diabetes and Postprandial IOP

Duration of Diabetes	Mean Postprandial IOP (mmHg) \pm SD
<5 years	17.1 \pm 1.7
5–10 years	18.3 \pm 2.1
>10 years	19.0 \pm 2.4

Patients with longer duration of diabetes demonstrated progressively higher postprandial IOP values. Chronic hyperglycemia appears to exert cumulative effects on ocular tissues and aqueous humor drainage pathways.

Multivariate Assessment of Factors Associated with Elevated IOP

Table 7: Factors Associated with Elevated Postprandial IOP

Variable	Association with Elevated IOP
Postprandial blood glucose	Strong positive association
Duration of diabetes	Moderate positive association
Central corneal thickness	Moderate positive association
Age	Weak association
Gender	No significant association

Interpretation

Postprandial blood glucose emerged as the strongest determinant of elevated intraocular pressure, followed by duration of diabetes and central corneal thickness.

Discussion: The present analytical comparative study evaluated the influence of fasting and postprandial glycemic status on intraocular pressure among patients with Type 2 Diabetes Mellitus and healthy controls. The study demonstrated that diabetic individuals exhibit significantly higher fasting and postprandial IOP values than non-diabetic controls, with postprandial glycemia showing the strongest association with

elevated ocular pressure. A major finding of the study was the significant increase in IOP following meals among diabetic participants. The mean postprandial increase of approximately 1.8 mmHg contrasts sharply with the minimal change observed among controls. These findings suggest that acute metabolic fluctuations exert measurable effects on ocular physiology.

The association between diabetes and elevated IOP has been documented in several epidemiological investigations. Wu et al. reported that increasing postprandial glucose levels were independently associated with elevated intraocular pressure in a large Taiwanese population. Our findings are consistent with these observations and further

demonstrate that postprandial glycemia may be a stronger predictor of IOP than fasting glucose levels [12].

The strong correlation between PPBS and IOP observed in the present study may be explained by osmotic and hemodynamic mechanisms. Acute hyperglycemia alters plasma osmolarity and may influence aqueous humor formation. Additionally, glucose-induced oxidative stress and endothelial dysfunction can impair trabecular meshwork function, thereby reducing aqueous outflow and increasing intraocular pressure [13-14].

Pimentel et al. observed significant postprandial IOP elevation among diabetic individuals compared with non-diabetic controls. Similar results were obtained in the present study, strengthening evidence that glycemic fluctuations influence ocular pressure dynamics. These findings underscore the importance of considering the timing of IOP measurement in diabetic patients [15].

Central corneal thickness was significantly greater among diabetic participants. This observation agrees with studies by Pandey et al. and Chowdhury et al., who reported increased corneal thickness and endothelial dysfunction among patients with diabetes. Thicker corneas may result in overestimation of IOP when measured using applanation-based techniques. Therefore, routine pachymetry should be incorporated into glaucoma screening programs for diabetic individuals [16].

The present study also demonstrated a duration-dependent increase in postprandial IOP. Participants with diabetes duration exceeding ten years exhibited the highest IOP values. Chronic hyperglycemia may lead to cumulative structural changes within the trabecular meshwork, Schlemm's canal, and extracellular matrix. Such alterations reduce aqueous humor drainage efficiency and contribute to sustained ocular hypertension [17-18]. From a clinical perspective, these findings are highly relevant. Glaucoma remains a leading cause of irreversible blindness, and early detection of elevated IOP is critical. Routine ophthalmic examinations in diabetic patients should ideally include postprandial IOP assessment, particularly among those with long-standing disease or poor glycemic control [19-20].

The study also highlights the potential role of glycemic variability as an independent risk factor for ocular hypertension. While fasting glucose and HbA1c remain important markers of metabolic control, postprandial hyperglycemia may provide additional prognostic information regarding glaucoma risk.

Conclusion

The present study demonstrates that postprandial hyperglycemia is significantly associated with elevated intraocular pressure among patients with Type 2 Diabetes Mellitus. The relationship is strengthened by increasing duration of disease and greater central corneal thickness. These findings support the incorporation of postprandial IOP measurement and pachymetric evaluation into routine diabetic eye care. Early identification of ocular hypertension in diabetic patients may facilitate timely intervention and reduce the burden of glaucoma-related visual impairment.

References

1. Yin L, Zhang D, Ren Q, Su X, Sun Z. Prevalence and risk factors of diabetic retinopathy in diabetic patients: A community-based cross-sectional study. *Medicine (Baltimore)*. 2020;99(9):e19236.
2. Kaldırım H, Atalay K, Ceylan B, Yazgan S. Efficacy of hyperbaric oxygen therapy on central corneal thickness, intraocular pressure, and nerve fiber layer in patients with Type 2 diabetes. *Korean J Ophthalmol*. 2021;35(1):1–9.
3. Agrawal A, Ahuja S, Singh A, Samanta R, Mittal SK. Influence of glycated haemoglobin levels on intraocular pressure in patients with Type II diabetes mellitus. *Nepal J Ophthalmol*. 2019;11(21):19–23.
4. Singh W, Singh Salaria N, Pandey ML, Bhandari V, Singh S, Bhardwaj P. Prevalence and associated risk factors of primary open-angle glaucoma among patients with Type 2 diabetes mellitus. *Cureus*. 2022;14(9):e28908.
5. Pandey S, Singh A, Vannadil H, Agrawal M. Corneal parameters in diabetics versus non-diabetics and correlation with various blood sugar parameters. *Rom J Ophthalmol*. 2024;68(2):128–134.
6. Biswas S, Raman R, Koluthungan V, Sharma T. Intraocular pressure and its determinants in subjects with Type 2 diabetes mellitus in India. *J Prev Med Public Health*. 2011;44(4):157–166.
7. Chopra V, Varma R, Francis BA, Wu J, Torres M, Azen SP. Type 2 diabetes mellitus and risk of open-angle glaucoma: The Los Angeles Latino Eye Study. *Ophthalmology*. 2008;115(2):227–232.
8. Braha A, Simion A, Timar R, Timar B. Factors associated with increased intraocular pressure in Type 2 diabetes patients. *J Clin Med*. 2024;13(3):676.
9. Choi JA, Park YM, Han K, Lee J, Yun JS, Ko SH. Fasting plasma glucose level and risk of open-angle glaucoma: Nationwide population-based cohort study in Korea. *PLoS One*. 2020;15(9):e0239529.

10. Bekmez S, Kocaturk T. Higher intraocular pressure levels associated with lower hysteresis in Type 2 diabetes. *Open Ophthalmol J*. 2018;12:29–33.
11. Chowdhury B, Bhadra S, Mittal P, Shyam K. Corneal endothelial morphology and central corneal thickness in Type 2 diabetes mellitus patients. *Indian J Ophthalmol*. 2021;69(7): 1718–1724.
12. Ehlers N, Bramsen T, Sperling S. Applanation tonometry and central corneal thickness. *Acta Ophthalmol*. 1975;53(1):34–43.
13. Wu CJ, Fang WH, Kao TW, Chen YJ, Liaw FY, Chang YW, et al. Postprandial glucose as a risk factor for elevated intraocular pressure. *PLoS One*. 2016;11(12):e0168142.
14. Hanyuda A, Sawada N, Yuki K, Uchino M, Ozawa Y, Sasaki M, et al. Relationships of diabetes and hyperglycaemia with intraocular pressure in a Japanese population. *Sci Rep*. 2020;10:5355.
15. Pimentel LG, Gracitelli CP, da Silva LS, Souza AK, Prata TS. Association between glucose levels and intraocular pressure: Pre- and postprandial analysis in diabetic and nondiabetic patients. *J Ophthalmol*. 2015;2015:832058.
16. Baisakhiya S, Garg P, Singh S. Association between glycemic control and intraocular pressure in patients with Type II diabetes mellitus. *Natl J Physiol Pharm Pharmacol*. 2017;7(1):43–46.
17. Módis L Jr, Szalai E, Kertész K, Kemény-Beke A, Kettesy B, Berta A. Evaluation of the corneal endothelium in patients with diabetes mellitus Type I and II. *Histol Histopathol*. 2010;25(12):1531–1537.
18. Adam L, O'Connor C, Garcia AC. Evaluating the impact of diabetes self-management education methods on knowledge, attitudes and behaviours of adult patients with Type 2 diabetes mellitus. *Can J Diabetes*. 2018;42(5):470–477.
19. Buehler AM, Cavalcanti AB, Berwanger O, Figueiro M, Laranjeira LN, Zazula AD, et al. Effect of tight blood glucose control versus conventional control in Type 2 diabetes mellitus: A systematic review. *Cardiovasc Ther*. 2013;31(3):147–160.
20. Zapuskalov IV, Krivosheina OI, Tsyrov GI. Local mechanisms of relationship between colloid-osmotic pressure and ophthalmic tone in patients with diabetes mellitus. *Vestn Oftalmol*. 1998;114(2):43–44.