

**RELATIONSHIP BETWEEN BLOOD SUGAR LEVEL AND INTRAOCULAR
PRESSURE**

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**A PROJECT SUBMITTED TO THE FACULTY OF OPTOMETRY,
UNIVERSITY OF BENIN, BENIN CITY, IN PARTIAL FULFILMENT OF
THE REQUIREMENT FOR THE AWARD OF DOCTOR OF
OPTOMETRY (OD) DEGREE.**

CERIFICATION

This is to certify that this research project titled **RELATIONSHIP BETWEEN BLOOD SUGAR LEVEL AND INTRAOCULAR PRESSURE** was carried out by **OLADEJI MERCY OMOTOLA** in the Faculty of Optometry, University of Benin in partial fulfilment of the requirement for the Doctor of Optometry degree in the 2024/2025 Academic Session.

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DEDICATION

I dedicate this project to God Almighty, my late grandmother and also my parents Mr. and Mrs. Olusegun Oladeji for their love, and unwavering moral and financial support.

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ABSTRACT

Diabetes mellitus has been increasingly linked with ocular complications, particularly elevated intraocular pressure (IOP), a key risk factor for glaucoma. Understanding the relationship between blood sugar level and Intraocular pressure is critical for early detection and prevention of vision-threatening conditions. This study aimed to determine the relationship between fasting blood sugar (FBS) levels and intraocular pressure and assess the influence of age and gender on these parameters among adults in Benin City, Nigeria. A cross-sectional study was conducted among 100 adults aged 41–80 years with the mean age (61.5 years) attending St. Teresa Specialist and Laser Eye Center, Benin City. Fasting blood sugar was measured using a glucometer, and intraocular pressure was assessed using an iCare tonometer. Statistical analyses were performed using SPSS version 22, applying t-tests, ANOVA, and Pearson correlation. The findings revealed a strong positive correlation between fasting blood sugar and intraocular pressure ($r = 0.658$, $p = 0.01$). Males exhibited higher mean IOP (17.44 mmHg) than females (15.49 mmHg; $p = 0.047$), while older age groups (61–80 years) showed significantly higher FBS (126.84 mg/dl) and IOP (18.72 mmHg) values compared to younger participants ($p < 0.05$). It is concluded from this study that elevated fasting blood sugar is significantly associated with increased intraocular pressure, with age and gender acting as modifying factors. Routine IOP screening is recommended for individuals with elevated blood glucose, regardless of diabetic status.

CHAPTER ONE

1.0 INTRODUCTION

Intraocular pressure (IOP) refers to the fluid pressure inside the eye and is a key parameter in maintaining ocular health. Elevated IOP is a major risk factor for glaucoma, a leading cause of irreversible blindness globally (Tham *et al.*, 2015). Concurrently, blood sugar level measured as either fasting glucose or glycated hemoglobin (HbA1c) is critical in the management of diabetes mellitus, a metabolic disorder marked by chronic hyperglycemia (ElSayed *et al.*, 2023). The interplay between glycemic regulation and IOP has garnered research interest due to emerging evidence suggesting that dysregulated blood glucose may influence IOP homeostasis (Cohen *et al.*, 2017).

The global prevalence of diabetes has increased markedly, with ocular complications now recognized as significant comorbidities (Ogurtsova *et al.*, 2017). While the pathophysiology of diabetic retinopathy is well understood, the relationship between systemic glycemic status and IOP remains less clearly defined. Moreover, conflicting findings in current literature particularly regarding whether diabetics exhibit consistently higher IOP underscore a key gap in knowledge. This study aims to address this ambiguity by comparing IOP patterns in blood glucose level.

1.1 BACKGROUND INFORMATION

1.1.1 Intraocular Pressure (IOP) and Its Clinical Significance

Intraocular pressure (IOP) refers to the fluid pressure inside the eye, primarily maintained by the balance between aqueous humor production and outflow. Normal IOP ranges from 10 to 21 mmHg and is crucial for maintaining the eye's shape and proper optical function. Elevated IOP is

a major risk factor for glaucoma, a group of eye diseases characterized by optic nerve damage and progressive vision loss (Blumberg *et al.*, 2015). Open-angle glaucoma, the most common form, often develops slowly without noticeable symptoms until significant vision loss has occurred.

Monitoring IOP is essential in the early detection and management of glaucoma. Regular eye examinations, including tonometry to measure IOP, can help identify individuals at risk and initiate timely interventions to prevent irreversible vision loss. Factors influencing IOP include age, genetic predisposition, systemic health conditions like hypertension and diabetes, and lifestyle factors such as smoking (Weih *et al.*, 2001). Understanding the dynamics of IOP and its role in ocular health is vital for developing effective strategies to combat glaucoma and other pressure-related eye conditions.

1.1.2 Diabetes mellitus and Its Clinical Significance

Diabetes mellitus is a chronic metabolic disorder characterized by sustained hyperglycemia due to defects in insulin secretion, insulin action, or both, resulting in long-term damage to the heart, blood vessels, eyes, kidneys, and nerves (Sarkar *et al.*, 2019). It comprises several types, most notably type 1 diabetes, an autoimmune destruction of pancreatic β -cells leading to absolute insulin deficiency (Roep *et al.*, 2021), and type 2 diabetes, characterized by insulin resistance and a relative insulin secretory defect (Lebovitz, 2001). Globally, approximately one in nine adults live with diabetes, with age-standardized prevalence rising from 7% in 1990 to 14% in 2022, affecting an estimated 828 million people in 2022, and projected to exceed 700 million by 2045 (Wang *et al.*, 2024). Chronic hyperglycaemia underlies both microvascular complications such as retinopathy, nephropathy, and neuropathy and macrovascular complications including

coronary artery disease, stroke, and peripheral arterial disease (Fowler, 2011). Diagnosis is based on fasting plasma glucose ≥ 126 mg/dL, 2-hour plasma glucose ≥ 200 mg/dL during an oral glucose tolerance test, or HbA_{1c} $\geq 6.5\%$ (Karnchanasorn *et al.*, 2016). Management combines lifestyle modification including medical nutrition therapy, physical activity, and diabetes self-management education with pharmacotherapy tailored to type and comorbidities, such as insulin for type 1 diabetes and metformin, GLP-1 receptor agonists, and SGLT2 inhibitors for type 2 diabetes (Fujiwara *et al.*, 2019).

1.1.2 Glycemic Control and Intraocular Pressure

Glycemic control, a cornerstone of diabetes management, denotes the maintenance of blood glucose levels within target ranges to mitigate the progression of diabetes-associated complications. Clinically, this is quantified through glycated hemoglobin (HbA_{1c}), a biomarker reflecting average plasma glucose concentrations over approximately 8–12 weeks, offering a retrospective assessment of glycemic regulation (ElSayed *et al.*, 2023). Optimal glycemic control is imperative for minimizing systemic sequelae, including retinopathy, neuropathy, and nephropathy, as well as ocular pathologies such as glaucoma, which is closely linked to dysregulated intraocular pressure (IOP).

Emerging epidemiological and clinical evidence underscores a significant positive correlation between suboptimal glycemic control and elevated IOP, a critical modifiable risk factor for glaucoma and optic nerve damage. In a cross-sectional analysis by Cohen *et al.* (2017), individuals with elevated fasting serum glucose levels demonstrated a dose-dependent increase in IOP, independent of traditional risk factors such as age, body mass index, or hypertension. This association was corroborated in a large-scale population-based study by (Cui *et al.*, 2019),

which identified a robust, independent relationship between higher HbA1c levels and increased IOP among Chinese adults, even after adjusting for confounding variables. These findings collectively suggest that chronic hyperglycemia may exert direct or indirect influences on ocular hemodynamics and aqueous humor dynamics.

The pathophysiological interplay between hyperglycemia and IOP elevation remains multifactorial. Proposed mechanisms include osmotic fluid shifts secondary to hyperglycemia, which may transiently increase vitreous chamber volume and intraocular pressure. Chronic hyperglycemia also induces microvascular dysfunction and oxidative stress, impairing the structural and functional integrity of the trabecular meshwork, a critical regulator of aqueous humor outflow (Zhao *et al.*, 2015). Additionally, hyperglycemia-driven advanced glycation end products (AGEs) may contribute to trabecular sclerosis and reduced outflow facility, while insulin resistance and compensatory hyperinsulinemia could alter aqueous humor production via sympathetic nervous system activation. Such mechanisms collectively predispose individuals with prolonged glycemically dysregulated to sustained IOP elevation, amplifying glaucoma risk.

Despite these insights, critical gaps persist in understanding the temporal and causal relationships between glycemically control and IOP modulation. Existing studies are predominantly cross-sectional, limiting inferences about causality. Longitudinal investigations are warranted to delineate whether improved glycemically management attenuates IOP elevation or reduces glaucoma incidence, particularly in ethnically diverse cohorts with varying diabetes phenotypes. Furthermore, the role of glucose variability, an emerging metric of glycemically instability in influencing IOP fluctuations remains unexplored.

1.1.3 Blood Glucose Fluctuations and Intraocular Pressure:

Beyond chronic hyperglycemia, glycemic variability (GV), short-term blood glucose fluctuations has emerged as an independent risk factor for diabetic complications, including ocular pathologies. GV encompasses postprandial spikes and hypoglycemic dips, contributing to oxidative stress and endothelial dysfunction, even in individuals with controlled HbA1c (Monnier et al., 2008). Growing evidence links acute glycemic excursions to transient intraocular pressure (IOP) elevation. Pimentel *et al.* (2015) observed significant IOP increases following postprandial hyperglycemia in type 2 diabetics, likely due to osmotic fluid shifts into ocular compartments or autonomic dysregulation affecting aqueous humor dynamics. Similarly, Gopi *et al.* (2025), reported greater IOP variability in patients with frequent glycemic spikes, particularly those with poor glycemic control (HbA1c >8%), suggesting cumulative metabolic stress on trabecular meshwork function.

Proposed mechanisms include hyperglycemia-induced osmotic expansion of vitreous volume, ROS-mediated endothelial dysfunction, and inflammatory pathways impairing aqueous outflow. Repeated glucose spikes may exacerbate trabecular fibrosis and reduce outflow capacity, while autonomic neuropathy could disrupt ciliary body perfusion. However, evidence remains limited by cross-sectional designs, small cohorts, and inconsistent GV quantification (e.g., intermittent vs. continuous glucose monitoring CGM).

Longitudinal studies integrating CGM with IOP telemetry are needed to clarify real-time interactions and causality. Future research should evaluate whether GV reduction strategies (e.g., alpha-glucosidase inhibitors) mitigate IOP instability and glaucoma risk. Addressing these gaps

could refine diabetic care protocols, emphasizing dual targeting of chronic hyperglycemia and acute glucose fluctuations to optimize ocular outcomes.

1.1.4 Comparison of Intraocular Pressure in Diabetic and Non-Diabetic Populations

Individuals with diabetes mellitus (DM) consistently demonstrate higher intraocular pressure (IOP) than non-diabetic populations, a difference attributed to diabetes-specific physiological disruptions (Cohen *et al.*, 2017). Chronic hyperglycemia drives structural and functional changes in ocular tissues, particularly in the trabecular meshwork, a key regulator of aqueous humor outflow. Prolonged hyperglycemia promotes glycation of trabecular proteins, stiffening the meshwork and reducing its ability to drain fluid, thereby elevating IOP (Hymowitz *et al.*, 2016). Concurrently, osmotic effects of elevated blood glucose may transiently increase aqueous humor production, compounding pressure buildup.

Population studies highlight this disparity, with diabetic cohorts showing IOP elevations of 1–2 mmHg on average compared to non-diabetics (Chun *et al.*, 2015). These differences persist even after adjusting for variables like age and BMI, underscoring hyperglycemia's direct role. However, interpreting IOP in diabetics is complicated by confounding factors. For example, antihypertensive medications (e.g., thiazides) may lower IOP, while diabetes-associated corneal thickening can artificially inflate tonometry readings. Additionally, diurnal IOP fluctuations, often more pronounced in diabetics may lead to inconsistent measurements if not standardized.

This elevated IOP in diabetes translates to a clinically significant risk: diabetic individuals face over twice the likelihood of ocular hypertension, a precursor to glaucoma (Chun *et al.*, 2015). These findings emphasize the importance of regular IOP monitoring in diabetes care, alongside glycemic management, to mitigate glaucoma risk. Future research must address gaps in

understanding long-term IOP trajectories in diabetes and refine measurement protocols to account for confounders, ensuring accurate risk stratification.

1.2 STATEMENT OF PROBLEM

Glaucoma, a leading cause of blindness, is primarily linked to elevated intraocular pressure (IOP). However, up to 40% of cases occur with normal IOP, suggesting other contributing factors. Emerging evidence indicates that blood glucose fluctuations may influence IOP through osmotic shifts, vascular dysfunction, and autonomic regulation. While studies have explored this relationship in diabetic populations, there is limited research directly comparing glucose-IOP dynamics in the general population.

1.3 AIM AND OBJECTIVES OF STUDY

1.3.1 AIM

To determine the relationship between blood sugar levels and intraocular pressure (IOP) in the general population.

1.3.2 OBJECTIVES OF STUDY

1. To determine the gender difference In Intraocular Pressure (IOP) levels.
2. To determine the difference in Intraocular (IOP) levels among the different age ranges.
3. To determine the gender difference in Fasting Blood Sugar (FBS) levels.
4. To determine the difference in Fasting Blood Sugar levels (FBS) among the different age ranges.
5. To determine the correlation between Fasting Blood Sugar (FBS) levels and Intraocular pressure (IOP).

1.4 HYPOTHESIS

1.4 HYPOTHESIS

Null Hypotheses (H_0)

1. There is no significant relationship between blood glucose levels and intraocular pressure (IOP) in the general population.
2. There is no significant difference in the relationship between blood glucose levels and intraocular pressure (IOP) among the two genders.
3. There is no significant difference in the relationship between blood glucose levels and intraocular pressure (IOP) among different age groups.

Alternate Hypotheses (H_1)

1. There is a significant relationship between blood glucose levels and Intraocular pressure in the general population.
2. There is a significant difference in the relationship between blood glucose levels and intraocular pressure (IOP) among the two genders.
3. There is a significant difference in the relationship between blood glucose levels and intraocular pressure (IOP) among different age groups.

1.5 SIGNIFICANCE OF THE STUDY

- 1) The study examined how blood glucose levels affected intraocular pressure in the general population.

- 2) It provided evidence on IOP changes in individuals with varying glycemic levels, including non-diabetics.
- 3) It clarified whether subclinical hyperglycemia contributed to elevated IOP.
- 4) It supported the need for earlier IOP screening in people with raised fasting blood sugar.
- 5) It filled a gap in research by comparing glucose-IOP relationships across different age and gender groups.

1.6 DEFINITION OF TERMS

Intraocular Pressure (IOP) – The pressure within the eye, regulated by aqueous humor production and outflow, measured in millimeters of mercury (mmHg).

Blood Glucose Levels – The concentration of glucose in the bloodstream, expressed in mg/dL or mmol/L, regulated by dietary intake, insulin function, and hepatic metabolism.

Oxidative Stress – An imbalance between reactive oxygen species (ROS) and antioxidant defenses, leading to cellular damage, particularly relevant in glaucoma pathophysiology.

Autonomic Nervous System (ANS) – The neural network regulating involuntary physiological processes, including vascular control and aqueous humor secretion.

CHAPTER TWO

2.0 LITERATURE REVIEW

Several studies have investigated the intricate link between blood sugar levels and intraocular pressure (IOP), as this relationship plays a critical role in understanding the pathophysiology of glaucoma and ocular hypertension. Diabetes mellitus, characterized by chronic hyperglycemia, has been associated with increased IOP through mechanisms involving osmotic changes, autonomic dysfunction, and alterations in aqueous humor dynamics. Khalaj *et al.* (2015) examined the relationship between diabetes and intraocular pressure in patients at Avicenna Hospital compared to healthy individuals. Their study of 400 participants, including both diabetic and non-diabetic groups, revealed a statistically significant elevation of IOP in diabetic patients (mean 16.71 ± 1.96 mmHg) compared to non-diabetics (mean 12.86 ± 1.45 mmHg, $p = 0.001$). The findings supported the hypothesis that metabolic disturbances in diabetes contribute to elevated intraocular pressure, thereby predisposing individuals to ocular complications such as glaucoma.

Similarly, Cohen *et al.* (2017) assessed the correlation between serum glucose levels and IOP among 18,406 subjects in a large cross-sectional study. They found that mean IOP values were 13.1 mmHg in normoglycemic subjects, 13.7 mmHg in those with impaired fasting glucose, and 14.3 mmHg in those with diabetes, with a clear positive linear correlation between fasting glucose and IOP across both genders. This study provided compelling epidemiological evidence that even mild elevations in serum glucose could exert measurable effects on IOP, reinforcing the notion that hyperglycemia, whether chronic or transient, has a systemic impact on ocular physiology. The results also highlight the importance of glycemic regulation in preventing subtle

but cumulative increases in intraocular pressure that may contribute to glaucomatous damage over time.

Ojha *et al.* (2022) further examined the association between intraocular pressure and blood sugar levels in patients with type 2 diabetes mellitus compared with healthy controls. Conducted in a tertiary care center in Northern India, the study revealed that variations in glucose levels were directly reflected in changes in IOP, with significant differences observed between fasting and postprandial measurements among diabetic patients. These findings indicate that fluctuations in glycemic control, rather than just chronic hyperglycemia, can transiently influence IOP, suggesting that both acute and long-term metabolic instability play a role in ocular pressure regulation.

In another investigation, Samal *et al.* (2021) compared intraocular pressures among diabetic patients at varying stages of retinopathy and in non-diabetic individuals. Their study of 664 patients revealed a consistent pattern of higher IOP readings in diabetic eyes, with pressure increasing progressively with the severity of diabetic retinopathy from 15.0 mmHg in those without retinopathy to 20.9 mmHg in proliferative stages. This progressive trend strongly suggests a dose-dependent relationship between chronic hyperglycemia and intraocular pressure, possibly mediated by microvascular and structural changes within the trabecular meshwork and optic nerve head.

Supporting these observations, Vidhya *et al.* (2016) conducted a comparative study among diabetic and non-diabetic patients and reported significantly higher mean IOP values in diabetics (16.4 ± 1.32 mmHg) compared to non-diabetics (12.9 ± 1.09 mmHg). The researchers emphasized that diabetic patients should undergo regular ocular pressure monitoring to detect

early-onset glaucoma, as prolonged exposure to elevated glucose levels can compromise aqueous humor outflow resistance and increase optic nerve susceptibility.

Singh *et al.* (2017) also investigated the effects of type II diabetes mellitus on intraocular pressure in Central India. They found that diabetic participants consistently exhibited higher IOP across all age groups (mean = 17.61 mmHg) compared to non-diabetic counterparts (mean = 14.08 mmHg). The study further revealed that patients with longer disease duration and poor glycemic control had significantly higher IOP values, suggesting a cumulative effect of prolonged hyperglycemia on ocular tissues. Chronic exposure to elevated glucose levels likely induces glycosylation of collagen fibers in the trabecular meshwork, reducing aqueous outflow and thereby increasing intraocular pressure.

A large-scale study by Biswas *et al.* (2010) provided additional insights into factors influencing IOP among subjects with type 2 diabetes in India. Involving 1,377 participants aged over 40 years, the study revealed that IOP was significantly higher in individuals with hypertension, elevated pulse rates, and thicker central corneal thickness. Interestingly, women with elevated glycated hemoglobin (HbA1c) levels exhibited higher IOPs, reinforcing the link between poor glycemic control and ocular hypertension. Even after adjusting for confounding factors, the findings demonstrated that systemic metabolic and vascular alterations contribute to IOP dysregulation among diabetic patients.

Further evidence connecting acute glycemic changes and IOP comes from Yildiz *et al.* (2024), who investigated hyperglycemia's effects on intraocular pressure, corneal biomechanics, and corneal topography during the oral glucose tolerance test in non-diabetic individuals. The study found significant increases in IOP during peak hyperglycemia, accompanied by transient changes

in corneal thickness and elasticity. These findings demonstrate that even in individuals without diabetes, sudden elevations in blood glucose can temporarily alter ocular biomechanics and intraocular pressure, emphasizing the dynamic responsiveness of ocular structures to systemic metabolic fluctuations.

Similarly, Pimentel *et al.* (2015) explored the association between glucose variation and IOP fluctuation in both diabetic and non-diabetic subjects. They observed that postprandial IOP increased significantly compared to fasting measurements in both groups, but the magnitude of change was more pronounced among diabetics. Their multivariable analysis confirmed that glucose level variation remained independently associated with IOP change, even after controlling for age, gender, ancestry, and baseline IOP. These results underscore that glycemic variability not just chronic hyperglycemia plays a significant role in modulating intraocular pressure.

Expanding on this relationship, Liu *et al.* (2024) analyzed data from the UK Biobank to examine the association between glycemic control and intraocular pressure among a large general population. The study demonstrated that individuals with diabetes had higher mean IOP and a greater prevalence of ocular hypertension compared to non-diabetic participants. After adjusting for confounding variables, HbA1c remained positively associated with IOP, with every 10 mmol/mol increase in HbA1c corresponding to a 0.20 mmHg rise in IOP. These findings indicate that chronic hyperglycemia contributes to persistent IOP elevation, increasing the risk of optic nerve damage and glaucoma development over time.

Research on IOP in diabetic patients across various populations has also provided important insights. Apreutesei *et al.* (2014) analyzed glaucoma progression in patients with diabetes and

found that those with coexisting diabetic retinopathy showed greater visual field deterioration and required more medications to control IOP than those without retinopathy. Although the differences were not always statistically significant, the trend supported the hypothesis that diabetes may exacerbate glaucomatous changes by impairing optic nerve perfusion and trabecular meshwork function. Similarly, Matsuoka *et al.* (2012) examined Japanese diabetic patients and found significantly higher mean IOP in diabetics (15.5 mmHg) compared to non-diabetics (14.0 mmHg). Moreover, IOP was positively correlated with HbA1c levels, particularly in patients with diabetic retinopathy, confirming that poor glycemic control aggravates ocular hypertension.

The long-term implications of these findings are evident in meta-analyses such as that conducted by Zhao *et al.* (2017), which quantitatively summarized the relationship between diabetes and glaucoma risk. Their review of seven prospective cohort studies found that diabetes increased the risk of glaucoma by 36%, indicating a substantial epidemiological association. Similarly, Zhou *et al.* (2014) reported from 13 studies that diabetic individuals had a 40% higher likelihood of developing primary open-angle glaucoma than non-diabetics. Both reviews demonstrated consistent evidence that diabetes, through sustained hyperglycemia and vascular dysregulation, predisposes individuals to optic nerve damage secondary to elevated intraocular pressure.

More recently, Chauhan *et al.* (2024) provided contemporary evidence of the interconnected risks between primary open-angle glaucoma and diabetic retinopathy. Their global retrospective cohort analysis involving over 44,000 diabetic patients with glaucoma and more than four million without glaucoma revealed that those with glaucoma had a significantly higher 10-year risk of developing diabetic retinopathy and proliferative diabetic retinopathy. These findings

indicate a bidirectional relationship between ocular hypertension and diabetic microvascular damage, suggesting that elevated IOP and chronic hyperglycemia may mutually reinforce each other's pathological effects on the eye.

Collectively, these studies illustrate a consistent and biologically plausible relationship between blood sugar levels and intraocular pressure across diverse populations. Chronic hyperglycemia leads to osmotic imbalance, structural stiffening of ocular tissues, and microvascular compromise, all of which elevate IOP and heighten glaucoma risk. Meanwhile, acute glucose fluctuations can transiently influence corneal biomechanics and aqueous humor dynamics, showing that both long-term and short-term glycemic changes affect ocular pressure regulation. These findings underscore the clinical importance of maintaining optimal glycemic control in diabetic and pre-diabetic individuals to prevent ocular hypertension and reduce the likelihood of glaucoma progression.

CHAPTER THREE

3.0 MATERIALS AND METHOD

3.1 RESEARCH DESIGN

A cross-sectional study design was employed to assess the relationship between blood sugar level and Intraocular pressure in general population.

3.2 RESEARCH LOCATION

This study was conducted at St Teresa Specialist and Laser Eye Center, Benin City, Edo State, Nigeria.

3.3 STUDY POPULATION

This study population included individuals within the age range of 41- 80 years old who attended the selected Eye Clinic in Benin City, Edo State.

3.4.1 SAMPLING TECHNIQUE

A convenient sampling technique was used to select participants.

3.4.2 SAMPLE SIZE

A study by Olamoyegun *et al.* (2024) mentioned that the prevalence of Type II Diabetes Mellitus among adults in Nigeria was 7%. The sample size was determined by adopting the standard formula which is the Fisher's formula.

$$n = \frac{Z^2 p(1-p)}{d^2}$$

$$d^2$$

Where,

n = the sample size to estimate a single population proportion.

d = precision desired

p = maximum estimated prevalence

z = statistic level of confidence

$p = 7\% = 0.07$

$d = 0.05$ (absolute precision)

$n = \frac{(1.96)^2 \times 0.07(1-0.07)}{(0.05)^2}$

$n = 100$

Therefore, a total of 100 participants were used for this study.

3.5 RESEARCH MATERIALS

1. iCare-Tonometer: A rebound tonometer that measures intraocular pressure

2. Glucometer (Blood Glucose Meter): A handheld device that measures blood glucose concentration using a drop of blood placed on a test strip.

3. Snellen Chart: A standardized eye chart with letters of progressively smaller sizes, utilized to measure visual acuity at 6 meters.

4. Occluder: A handheld paddle or patch applied to cover one eye during monocular VA testing.

5. Blood Glucose Test Strips: Chemically treated strips that react with blood samples and provide electrical signals read by the glucometer to determine glucose levels.

5. Lancets: Small disposable, sterile needles used to puncture the skin to collect a blood sample for glucose testing.

6. Control Solution: A glucose-containing liquid used to verify the accuracy of a glucometer and test strips by providing a known reference value.

7. Alcohol Swabs or Antiseptic Wipes: Used to clean the finger before lancing to prevent infection and contamination.

8. Cotton Balls or Gauze Pads: Used to stop minor bleeding after obtaining a blood sample.

3.6.1 INCLUSION CRITERIA

- Adults aged 41-80 years.
- Healthy individuals.
- Willingness to undergo blood glucose, Intraocular Pressure and Visual acuity testing.

3.6.2 EXCLUSION CRITERIA

- Individuals aged <41 years.
- Pregnant individuals (due to hormonal influences on IOP).
- Participants with severe visual impairment (<6/60 Snellen acuity) or ocular comorbidities (e.g., cataracts, corneal opacities) that could confound IOP or VA measurements.

3.7 DESCRIPTION OF PROCEDURE

1. Administration of biodata

Participants were first informed about the purpose of the study, explaining that the research aimed to investigate the relationship between blood sugar levels and intraocular pressure. After providing this explanation and addressing any questions, participants were asked to provide their biodata, including age, gender, and relevant medical history. This information was collected using a structured questionnaire and recorded.

2. Visual Acuity (VA) Measurement Protocol

Preparation:

- The Snellen chart was placed at 6 meters.
- Participants were instructed to wear their habitual corrective lenses (if any).

Testing:

- An occluder was used to cover the left eye; the right eye was tested first.
- Participants were asked to read aloud the smallest line they could discern.
- The process was repeated for the left eye.
- VA was recorded as the smallest line optotypes correctly identified (e.g., 6/6, 6/12).

Documentation:

- VA for each eye (e.g., OD: 6/6, OS: 6/9) and use of corrective lenses were documented.

3. Blood Glucose Measurement Protocol

Fasting Blood Sugar (FBS) measurement was carried out following a standardized procedure to ensure accuracy and consistency. The glucometer was calibrated according to the manufacturer's instructions, and a new test strip was inserted into the device. The participant's fingertip was then cleaned thoroughly with an alcohol swab and allowed to air-dry to prevent dilution or contamination of the blood sample. A sterile lancet was used to make a small puncture on the fingertip, and the first drop of blood was wiped away using sterile gauze to eliminate potential interference from interstitial fluid. The second drop of blood was collected onto the preloaded test strip, and the glucometer automatically displayed the FBS value, which was recorded in mg/dL. After the measurement, pressure was applied to the puncture site with a cotton ball or gauze to stop bleeding, and all used materials were properly discarded in a biohazard container in line with infection control protocols.

4. Intra-Ocular Pressure Measurement Protocol

Before beginning the measurement, we ensured that the tonometer was properly calibrated and in working condition. The device would be inspected for cleanliness, and a new disposable probe would be inserted into the probe holder to maintain hygiene and prevent cross-contamination. The patient was seated comfortably with their head positioned straight and stable. The procedure would be explained to the patient to ensure cooperation and reduce anxiety. Since the iCare tonometer does not require topical anesthesia, the patient can keep their eyes open naturally without discomfort. The iCare tonometer would be held steadily in front of the patient's eye, aligning it with the center of the cornea. The tonometer would be held at a distance of approximately 4-8 mm from the cornea, ensuring that the probe is perpendicular to the eye's surface.

Once the tonometer is properly aligned, the measurement button was pressed to release the probe. A typical measurement sequence involves six consecutive readings, after which the device calculates an average IOP value.

After obtaining the IOP reading, the value is displayed on the digital screen of the tonometer. The measurement for both eyes were recorded, ensuring proper notation of the right eye (OD) and left eye (OS). If there is significant variability between readings, additional measurements may be taken to confirm accuracy. Once the readings are recorded, the disposable probe is removed and discarded appropriately to maintain hygiene and prevent contamination.

4. Data Recording

All readings were entered into a structured data collection sheet. Values were verified for accuracy and checked for inconsistencies.

3.8 DATA ANALYSIS

The data obtained from this study was analyzed using the Statistical Package for Social Sciences (SPSS) version 22.0. Descriptive statistics (frequencies, percentages, mean, and standard deviation) summarized the variables. Variations in glucose levels within individuals were analyzed. Statistical methods (t-tests, regression analysis and ANOVA) were used to determine the significance of differences.

3.9 ETHICAL CONSIDERATION

Ethical clearance was obtained from the Department of Research and Ethics Committee of the Department of Optometry, University of Benin, Benin City, under the tenets of the Declaration of Helsinki. This ensures that all procedures performed on each subject will not be against the public interest or inflict unnecessary harm to them.

Informed consent of all participants was obtained before any data was collected from them to ensure their full cooperation.

CHAPTER FOUR

RESULTS AND DATA ANALYSIS

This chapter presents the findings of the study on the relationship between fasting blood sugar (FBS) and intraocular pressure (IOP) among adults attending St. Teresa Specialist and Laser Eye Center, Benin City. The results are organized under descriptive statistics, correlation analysis, and comparisons based on gender and age. Tables and figures are provided to support the findings, with explanatory notes summarizing key trends.

4.1 Descriptive Statistics of Study Variables

Table 4.1 Distribution of Age by Gender

		Gender		
		Male	Female	Total
Age Range	41-50	10	12	22
	51-60	8	11	19
	61-70	18	18	36
	71-80	11	12	23
Total		47	53	100

Table 4.1 shows that the majority of participants were in the 61-70 years age range, followed by those aged 71–80 and 41-50 years. The fewest participants were in the 51-60 years age bracket.

Table 4.2 **Mean Intraocular Pressure (IOP) among different age ranges**

Age Range	Male					Female					Total mean	SD
	Mean IOP (mmHg) OD	SD	Mean IOP (mmHg) OS	SD	N	Mean IOP (mmHg) OD	SD	Mean IOP (mmHg) OS	SD	N		
41-50	14.60	4.502	13.90	4.067	10	13.83	4.239	13.25	2.989	12	13.864	3.6390
51-60	18.50	7.445	17.75	5.600	8	15.55	3.328	16.27	4.361	11	16.842	4.8564
61-70	17.89	5.212	17.17	5.090	18	17.00	5.931	17.39	4.767	18	17.361	4.6868
71-80	19.55	6.832	19.82	6.194	11	14.83	4.218	14.17	2.517	12	16.978	5.3735

Gender Differences in Intraocular Pressure (IOP)

Table 4.3: Comparing Mean IOP by Gender

Gender	N	Mean IOP (mmHg)	SD
Male	47	17.436	5.4330
Female	53	15.491	4.02010

Males had a higher mean IOP of 17.436mmHg (± 5.4330) compared to females with a mean IOP of 15.491mmHg (± 4.0201).

Table 4.4: Independent Samples t-test Comparing Mean Intraocular Pressure (IOP) by Gender

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	T	Df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Mean IOP	Equal variances assumed	5.600	0.020	2.050	98	0.043	1.9456	0.9490	0.0623	3.8289
	Equal variances not assumed			2.014	84.007	0.047	1.9456	0.9660	0.0247	3.8665

An independent samples t-test was conducted to compare mean Intraocular pressure (IOP) between males and females. Levene’s test indicated unequal variances ($F = 5.600$, $p = 0.020$), therefore the “equal variances not assumed” result was used. The test revealed a significant gender difference in mean IOP, $t(84.01) = 2.014$, $p = 0.047$. Males had a higher mean IOP compared to females (mean difference = 1.95 mmHg). There is a significant gender difference in IOP.

Differences in Intraocular Pressure (IOP) Across Age Ranges

Table 4.5: One-way ANOVA showing differences in mean Intraocular Pressure (IOP) across age ranges

Source of Variation	Sum of Squares	Df	Mean Square	F	p-value
Between Ranges	186.186	3	62.062	2.828	0.043
Within Ranges	2106.662	96	21.944		
Total	2292.848	99			

Table 4.5 reports the ANOVA test, which revealed a statistically significant difference in mean IOP across age ranges, $F(3,96) = 2.828$, $p = 0.043$. This indicates that at least one age range had a mean IOP that differed from the others. However, the ANOVA does not specify which ranges differed, so post-hoc analysis was conducted below to identify the specific age groups with significant differences.

Table 4.6: One-way ANOVA Showing Differences in Mean Intraocular Pressure (IOP) Across Age Ranges

Age Ranges Compared	Mean Difference (MD)	Std. Error	p-value
41–50 vs 51–60	-2.9785	1.4671	0.184
41–50 vs 61–70	-3.4975	1.2677	0.034
41–50 vs 71–80	-3.1146	1.3970	0.123
51–60 vs 61–70	-0.5190	1.3284	0.980
51–60 vs 71–80	-0.1362	1.4523	1.000
61–70 vs 71–80	0.3829	1.2505	0.990

The one-way ANOVA with Tukey HSD post-hoc test showed that intraocular pressure varied across age ranges, but only one pairwise comparison was statistically significant. Participants aged 61–70 years had a significantly higher mean IOP than those aged 41–50 years (mean difference = -3.50 , $p = 0.034$). This indicates that IOP tends to rise between the 51–60 vs 61–70. The differences between all other age ranges, including 41–50 vs 71–80, 51–60 vs 61–70, and 61–70 vs 71–80, were not statistically significant ($p > 0.05$), suggesting that IOP levels in these comparisons were broadly similar.

Table 4.7 Mean Fasting Blood Sugar (FBS) among different Age Ranges

Age Range	Male			Female			Total mean	SD
	Mean FBS (mg/dl)	SD	N	Mean FBS (mg/dl)	SD	N		
41-50	104.80	18.588	10	95.67	9.727	12	99.82	14.809
51-60	121.25	31.905	8	109.55	24.197	11	114.47	27.502
61-70	121.78	37.849	18	134.61	39.701	18	128.19	38.778
71-80	138.64	34.926	11	112.83	35.270	12	125.17	36.744

Gender Differences in Fasting Blood Sugar (FBS)

Table 4.8 Comparing mean Fasting blood sugar (FBS) by Gender

Gender	N	Mean FBS (mg/dL)	SD
Male	47	122.02	33.878
Female	53	115.66	33.725

Males had a higher mean FBS of 122.02mg/dL (± 33.878) compared to females with a mean FBS of 115.66mg/dL (± 33.725).

Table 4.9: Independent Samples t-test Comparing Fasting blood sugar (FBS) by Gender

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	T	Df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
FBS	Equal variances assumed	0.073	0.788	0.939	98	0.350	6.361	6.772	-7.077	19.799
	Equal variances not assumed			0.939	96.469	0.350	6.361	6.773	-7.083	19.805

An independent samples t-test was conducted to compare fasting blood sugar (FBS) levels between males and females. Levene’s test indicated equal variances assumed ($F = 0.073$, $p = 0.788$). The result showed no significant gender difference in FBS levels, $t(98) = 0.939$, $p = 0.350$. Although males had a higher mean FBS (mean difference = 6.361 mg/dL), this was not statistically significant.

Differences in Fasting blood sugar (FBS) Across Age Ranges

Table 4.10: One-way ANOVA showing differences in mean Fasting blood sugar (FBS) across age ranges

Source of Variation	Sum of Squares	df	Mean Square	F	p-value
Between Ranges	12391.797	3	4130.599	3.944	0.011
Within Ranges	100552.953	96	1047.427		
Total	112944.750	99			

Table 4.10 reports the ANOVA test, which revealed a statistically significant difference in mean FBS across age ranges, $F(3,96) = 3.944$, $p = 0.011$. This indicates that at least one age range had a mean FBS that significantly differed from the others. However, the ANOVA does not specify which ranges differed, so post-hoc analysis was conducted below to identify the specific age groups with significant differences.

Table 4.11: One-Way ANOVA Comparing Mean Fasting Blood Sugar (FBS) Across Age Ranges

Age Ranges Compared	Mean Difference (MD)	Std. Error	p-value
41–50 vs 51–60	-14.656	7.055	0.186
41–50 vs 61–70	-28.376	7.193	0.001
41–50 vs 71–80	-23.356	8.287	0.023
51–60 vs 61–70	-13.721	9.032	0.434
51–60 vs 71–80	-10.700	9.925	0.705
61–70 vs 71–80	3.021	10.024	0.990

Table 4.11 presents the results of a one-way ANOVA comparing mean FBS levels across the four age ranges. Post-hoc analysis using Games-Howell indicated that participants aged 61–70 years (MD = -28.38, $p = 0.001$) and those aged 71–80 years (MD = -23.36, $p = 0.023$) had significantly higher mean FBS compared with those aged 41–50 years. No other pairwise comparisons were significant ($p > 0.05$). This suggests that older participants tended to have higher FBS levels.

Correlation Between Fasting blood Sugar (FBS) and Intraocular Pressure (IOP)

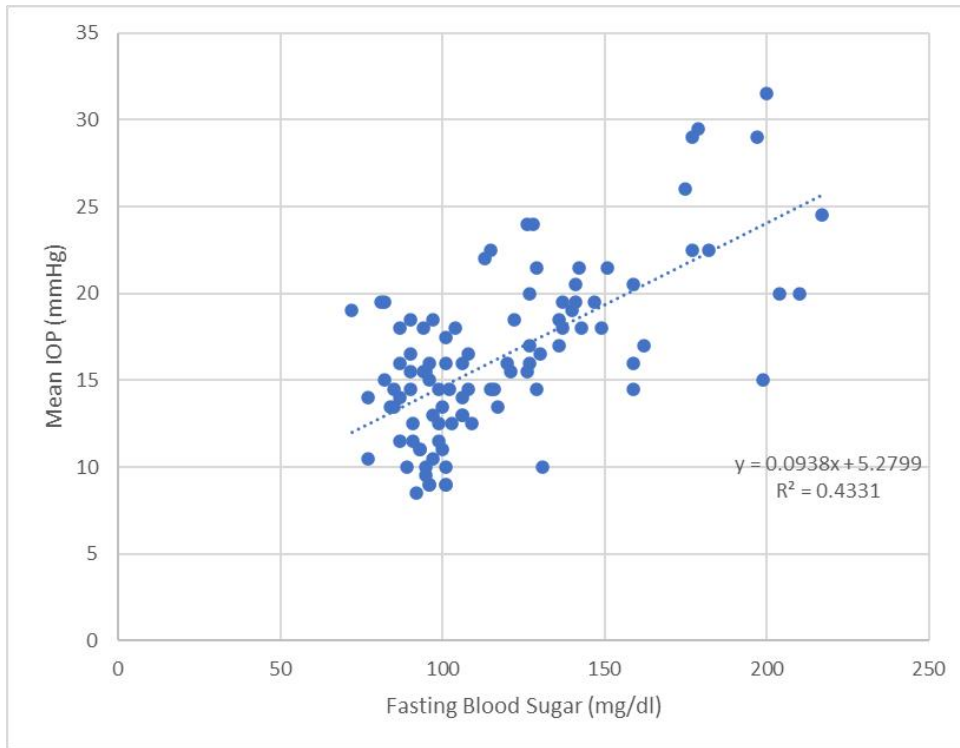


Figure 4.12: Scatterplot of Fasting blood sugar (FBS) vs Mean Intraocular Pressure (IOP) (with regression line)

Figure 4.12 shows the scatterplots of FBS against mean IOP with a regression line. The upward trend demonstrates that as fasting blood sugar increases, mean IOP with a regression line. The clustering data points along the regression line supports the significant positive relationship reported in figure 4.12

Table 4.13: Pearson’s Correlation Between FBS and Mean IOP (Overall Sample)

Variables	R	p-value
FBS vs Mean IOP	0.658	0.01

A Pearson’s correlation between fasting blood sugar (FBS) and mean intraocular pressure (IOP) was conducted to examine the relationship between fasting blood sugar (FBS) and mean intraocular pressure (IOP). A strong, positive, and statistically significant correlation was found ($r = 0.658$, $p = 0.01$), suggesting that higher FBS levels are associated with higher IOP.

CHAPTER FIVE

5.0 DISCUSSION

The study examined the relationship between fasting blood sugar (FBS) and intraocular pressure (IOP) and assessed whether differences existed across gender and age groups in Intraocular pressure and Fasting Blood Sugar.

The analysis in Table 4.13 demonstrated a strong, positive, and statistically significant correlation between fasting blood sugar and mean intraocular pressure. Participants with higher blood sugar levels recorded higher IOP values. This finding supports earlier reports by Cohen et al. (2017), who showed that intraocular pressure rises in a dose-dependent manner with increasing fasting glucose, and by Cui et al. (2019), who identified a significant relationship between glycated hemoglobin and IOP in Chinese adults. The consistency between this study and previous work strengthens the conclusion that glycemic status influences intraocular pressure regulation and may contribute to the development of ocular hypertension and glaucoma. The null hypothesis of no significant relationship between FBS and IOP was therefore rejected.

Gender differences in intraocular pressure were significant. Males recorded a higher mean IOP compared to females as seen in Table 4.3 and 4.4 showing the difference reached statistical significance. This outcome is noteworthy because it diverges from findings by Biswas *et al.*, 2010, who reported higher intraocular pressure in women, partly explained by systemic influences such as central corneal thickness and hypertension. The present study therefore highlights a discrepancy in the literature. The higher IOP observed among men in this sample may be explained by biological variation, hormonal influences in females, or other unmeasured

factors. This unexpected result emphasizes the need for further investigation into sex-specific determinants of intraocular pressure. The null hypothesis of no gender differences in IOP was rejected.

Furthermore, intraocular pressure varied significantly with age as shown in Table 4.5. Participants in the 61–70 years age group had significantly higher IOP than those in the 41–50 years group as shown in Table 4.6. These results support Singh *et al.*, (2017), who reported higher IOP values in diabetic individuals across all age groups, and they are consistent with the established understanding that structural and functional changes in the trabecular meshwork with age reduce aqueous humor outflow, leading to increased pressure. The null hypothesis relating to age differences in intraocular pressure was therefore rejected.

In contrast to IOP, gender differences in fasting blood sugar were not significant. Male participants had higher mean FBS values than females as shown in Table 4.8, but the difference was not statistically meaningful as shown in Table 4.9. This suggests that gender is not an independent determinant of fasting blood sugar in this population. Previous study by Chan *et al.*, (2019) has also reported no consistent pattern of gender variation in fasting glucose, and the present result aligns with those findings. The null hypothesis for gender differences in fasting blood sugar was retained

However, age differences were evident in fasting blood sugar. Participants aged 61–70 years and 71–80 years recorded significantly higher fasting blood sugar values compared to those aged 41–50 years as shown in Table 4.7. This aligns with global epidemiological evidence, including the findings of Wang *et al.*, (2024), which demonstrate that impaired glucose regulation and diabetes

are more prevalent in older adults. The null hypothesis relating to age differences in fasting blood sugar was therefore rejected.

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

The purpose of this study was to investigate the relationship between fasting blood sugar (FBS) and intraocular pressure (IOP), and to determine whether gender and age influenced these variables. A total of 100 adults aged 41–80 years were examined; There was a significant relationship between FBS and IOP as the analysis revealed a strong, positive, and statistically significant correlation between FBS and mean IOP ($r = 0.658$, $p = 0.01$). This indicates that higher blood sugar levels are associated with higher intraocular pressure.

There was no significant difference in Fasting blood sugar (FBS) between males and females. The independent samples t-test showed no significant gender difference in FBS (male mean = 122.02 mg/dL, female mean = 115.66 mg/dL, $p = 0.350$), confirming that fasting blood sugar levels did not vary significantly between genders in this sample.

There was a significant difference in Intraocular Pressure (IOP) between males and females. The independent samples t-test revealed a statistically significant difference, with males having a higher mean IOP (17.44 mmHg) compared to females (15.49 mmHg), $p = 0.047$. This demonstrates that gender does influence intraocular pressure.

There was a significant difference in Fasting blood sugar (FBS) across age groups. ANOVA and post-hoc analysis showed that participants aged 61–70 years ($p = 0.001$) and 71–80 years ($p = 0.023$) had significantly higher FBS compared to the 41–50 years age group. This result indicates that fasting blood sugar increases significantly with age.

There was a significant difference in IOP across age groups. ANOVA revealed a significant difference in mean IOP across the four age groups, $F(3,96) = 2.828$, $p = 0.043$. Post-hoc analysis further indicated that participants aged 61–70 years had significantly higher IOP than those aged 41–50 years ($p = 0.034$).

6.2 Recommendations

Based on the findings of this study, the following recommendations are made.

1. Routine intraocular pressure screening should be implemented for individuals with elevated fasting blood sugar levels, irrespective of whether a formal diagnosis of diabetes has been established.
2. Male gender should be considered a risk factor for higher intraocular pressure during clinical assessments.
3. Ophthalmological vigilance is required for individuals aged 61–70 and 71–80 years, as these groups demonstrated significantly higher fasting blood sugar and intraocular pressure levels.

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APPENDIX

Descriptives – FBS by Gender

Gender	N	Mean	Std. Deviation	Minimum	Maximum
Male	47	122.02	33.878	77	210
Female	53	115.66	33.725	72	217
Total	100				

Descriptives – FBS by Age Group

Age Group	N	Mean	Std. Deviation	Minimum	Maximum
41–50	22	99.82	14.809	77	142
51–60	19	114.47	27.502	72	179
61–70	36	128.19	38.778	77	217
71–80	23	125.17	36.744	85	200
Total	100				

Descriptives – IOP by Gender

Gender	N	Mean	Std. Deviation	Minimum	Maximum
Male	47	17.436	5.4330	9.0	31.5
Female	53	15.491	4.0210	8.5	29.0

Total	100				
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Descriptives – IOP by Age Group

Age Group	N	Mean	Std. Deviation	Minimum	Maximum
41–50	22	13.864	3.6390	8.5	21.5
51–60	19	16.842	4.8564	9.0	29.5
61–70	36	17.361	4.6868	9.0	29.0
71–80	23	16.978	5.3735	9.5	31.5
Total	100				

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
Age	100	41	80	61.37	11.294
FBS	100	72	217	118.65	33.777
IOP(OD)	100	7	33	16.52	5.449
IOP(OS)	100	8	30	16.29	4.871
Mean IOP	100	8.5	31.5	16.405	4.8125
Valid (listwise)	N100				

T-Test

Group Statistics

	Gender	N	Mean	Std. Deviation	Std. Error Mean
Mean IOP	Male	47	17.436	5.4330	.7925
	Female	53	15.491	4.0210	.5523

Independent Samples Test

	Levene's Test for Equality of Variances		t-test for Equality of Means						
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
								Lower	Upper
Mean IOP	5.600	.020	2.050	98	.043	1.9456	.9490	.0623	3.8289
Equal variances assumed									
Equal variances not assumed			2.014	84.007	.047	1.9456	.9660	.0247	3.8665

Group Statistics

	Gender	N	Mean	Std. Deviation	Std. Error Mean
FBS	Male	47	122.02	33.878	4.942
	Female	53	115.66	33.725	4.632

Independent Samples Test

	Levene's Test for Equality of Variances		t-test for Equality of Means						
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
								Lower	Upper
FBSEqual variances assumed	.073	.788	.939	98	.350	6.361	6.772	-7.077	19.799
Equal variances not assumed			.939	96.469	.350	6.361	6.773	-7.083	19.805

Descriptives

FBS

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
41-50	22	99.82	14.809	3.157	93.25	106.38	77	142
51-60	19	114.47	27.502	6.309	101.22	127.73	72	179
61-70	36	128.19	38.778	6.463	115.07	141.31	77	217
71-80	23	125.17	36.744	7.662	109.28	141.06	85	200
Total	100	118.65	33.777	3.378	111.95	125.35	72	217

Test of Homogeneity of Variances

FBS

Levene Statistic	df1	df2	Sig.
6.416	3	96	.001

ANOVA

FBS

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	12391.797	3	4130.599	3.944	.011
Within Groups	100552.953	96	1047.427		
Total	112944.750	99			

Multiple Comparisons

Dependent Variable: FBS

Games-Howell

(I) Age in 10-year	(J) Age in 10-year	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
41-50	51-60	-14.656	7.055	.186	-33.98	4.67
	61-70	-28.376*	7.193	.001	-47.50	-9.25
	71-80	-25.356*	8.287	.023	-47.92	-2.79
51-60	41-50	14.656	7.055	.186	-4.67	33.98
	61-70	-13.721	9.032	.434	-37.75	10.31
	71-80	-10.700	9.925	.705	-37.31	15.91
61-70	41-50	28.376*	7.193	.001	9.25	47.50
	51-60	13.721	9.032	.434	-10.31	37.75
	71-80	3.021	10.024	.990	-23.64	29.68
71-80	41-50	25.356*	8.287	.023	2.79	47.92
	51-60	10.700	9.925	.705	-15.91	37.31
	61-70	-3.021	10.024	.990	-29.68	23.64

*. The mean difference is significant at the 0.05 level.

Descriptives

Mean IOP

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean	Minimum	Maximum

					Lower Bound	Upper Bound		
41-50	22	13.864	3.6390	.7758	12.250	15.477	8.5	21.5
51-60	19	16.842	4.8564	1.1141	14.501	19.183	9.0	29.5
61-70	36	17.361	4.6868	.7811	15.775	18.947	9.0	29.0
71-80	23	16.978	5.3735	1.1205	14.655	19.302	9.5	31.5
Total	100	16.405	4.8125	.4812	15.450	17.360	8.5	31.5

Test of Homogeneity of Variances

Mean IOP

Levene Statistic	df1	df2	Sig.
.509	3	96	.677

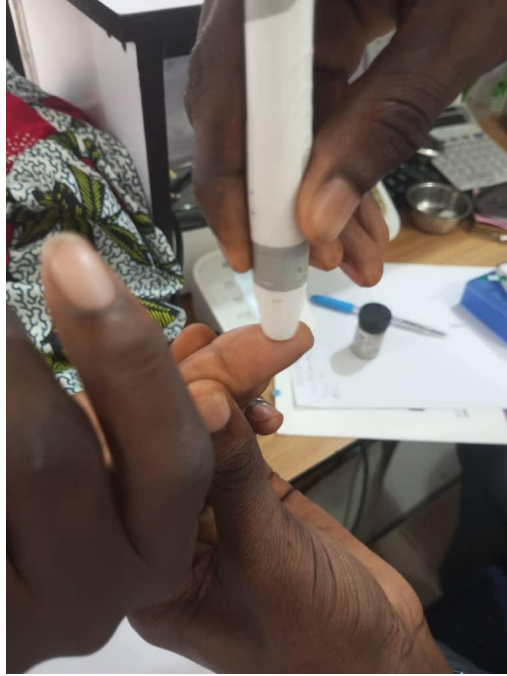
ANOVA

Mean IOP

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	186.186	3	62.062	2.828	.043

Within Groups	2106.662	96	21.944		
Total	2292.848	99			





5

NAME: Lyovnu Holtskule
AGE: 56
GENDER: Please tick Male Female
OCCUPATION: Tax Controller
VISUAL ACUTY: OD 9/15 OS 9/9
RANDOM BLOOD SUGAR: 95 mg/dL
INTRAOCULAR PRESSURE (MMHG): OD 17 mmHg OS 14 mmHg

57

NAME: Joseph Nielsen
AGE: 69
GENDER: Please tick Male Female
OCCUPATION: _____
VISUAL ACUTY: OD 6/6 OS 6/6
RANDOM BLOOD SUGAR: 100 mg/dL
INTRAOCULAR PRESSURE (MMHG): OD 15 mmHg OS 16 mmHg

56

NAME: Georgi Andreev
AGE: 64
GENDER: Please tick Male Female
OCCUPATION: Referee
VISUAL ACUTY: OD 6/12 OS 6/12
RANDOM BLOOD SUGAR: 81 mg/dL
INTRAOCULAR PRESSURE (MMHG): OD 20 mmHg OS 19 mmHg

NAME:

NAME: Chava Mathews
AGE: 60
GENDER: Please tick Male Female
OCCUPATION: Bus driver
VISUAL ACUTY: OD 6/4 OS 6/8
RANDOM BLOOD SUGAR: 187 mg/dL
INTRAOCULAR PRESSURE (MMHG): OD 20 mmHg OS 19 mmHg

6
NAME: Musa Ojino
AGE: 65
GENDER: Please tick Male Female
OCCUPATION: Teacher
VISUAL ACUTY: OD 6/24 OS 6/18
RANDOM BLOOD SUGAR: 120 mg/dL
INTRAOCULAR PRESSURE (MMHG): OD 11 mmHg OS 16 mmHg

NAME: Ada Nyoku
AGE: 68
GENDER: Please tick Male Female
OCCUPATION: Business Woman
VISUAL ACUTY: OD 6/60 OS 6/36
RANDOM BLOOD SUGAR: 141 mg/dL
INTRAOCULAR PRESSURE (MMHG): OD 27 mmHg OS 14 mmHg

4
NAME: Esther Edebor
AGE: 60
GENDER: Please tick Male Female
OCCUPATION: Teacher
VISUAL ACUTY: OD 6/9 OS 6/18
RANDOM BLOOD SUGAR: 151 mg/dL
INTRAOCULAR PRESSURE (MMHG): OD 1 mmHg OS 2.5 mmHg

3
NAME: Pulomenu Ekwana David
AGE: 62
GENDER: Please tick Male Female
OCCUPATION: Bus driver
VISUAL ACUTY: OD 6/24 OS 6/12
RANDOM BLOOD SUGAR: 130 mg/dL
INTRAOCULAR PRESSURE (MMHG): OD 14 mmHg OS 19 mmHg

S/N	Initials	Age	Gender	VA (OD)	VA(OS)	FBS	IOP(OD)	IOP(OS)
1	H.O		62 F	6/36	6/60	162mg/dl	15mmHg	19mmHg
2	O.W		71 F		6/24 6/36	87mg/dl	16mmHg	16mmHg
3	P.E		62 M		6/24	6/12 130mg/dl	14mmHg	19mmHg
4	E.E		60 F		6/9	6/18 151mg/dl	18mmHg	25mmHg
5	U.A		56 M	6/12-2		6/9 95mg/dl	17mmHg	14mmHg
6	M.O		65 F		6/24	6/9 120mg/dl	16mmHg	16mmHg
7	A.N		68 F	6/60	6/36	141mg/dl	27mmHg	14mmHg
8	C.M		80 M		6/9	6/18 137mg/dl	20mmHg	19mmHg
9	O.L		67 M		6/18	6/9 127mg/dl	27mmHg	13mmHg
10	I.M		41 F		6/24	6/6 84mg/dl	14mmHg	13mmHg
11	O.G		70 M		6/9	6/9 85mg/dl	15mmHg	14mmHg
12	E.O		67 M		6/9	6/12 93mg/dl	12mmHg	10mmHg
13	E.L		75 M		6/12	6/12 140mg/dl	18mmHg	20mmHg
14	A.J		50 M	6/9+2		6/12 116mg/dl	17mmHg	12mmHg
15	E.P		62 M	6/12+3	6/18+1	147mg/dl	20mmHg	19mmHg
16	O.J		65 M		6/9	6/6 128mg/dl	24mmHg	24mmHg
17	I.A		73 M		6/12	6/9 106mg/dl	15mmHg	11mmHg
18	E.O		67 F		6/9	6/12 106mg/dl	18mmHg	14mmHg
19	A.O		70 M		6/12	6/12 77mg/dl	10mmHg	11mmHg
20	G.J		79 F	6/60	6/36	126mg/dl	17mmHg	14mmHg
21	E.J		75 M		6/12	6/18 141mg/dl	18mmHg	21mmHg
22	U.P		66 M		6/9	6/6 101mg/dl	10mmHg	08mmHg
23	R.O		80 F	6/36	6/60	89mg/dl	07mmHg	13mmHg
24	FA		67 F		6/18	6/12 143mg/dl	19mmHg	17mmHg
25	RA		45 F		6/6	6/6 104mg/dl	22mmHg	14mmHg
26	O.G		55 F		6/6	6/9 96mg/dl	08mmHg	10mmHg
27	O.C		41 F		6/6	6/6 77mg/dl	14mmHg	14mmHg
28	Y.M		49 M		6/6	6/9 90mg/dl	15mmHg	14mmHg
29	SA		64 M		6/12	6/9 129mg/dl	20mmHg	23mmHg
30	T.C		80 F	6/36	6/36	131mg/dl	09mmHg	11mmHg
31	O.E		51 M		6/6	6/18 108mg/dl	14mmHg	15mmHg
32	M.S		61 M		6/9	6/9 101mg/dl	19mmHg	13mmHg
33	O.A		47 M		6/6	6/9 106mg/dl	15mmHg	11mmHg
34	O.M		74 F	6/36+1		6/12 199mg/dl	21mmHg	09mmHg
35	O.B		62 F	6/60		6/18 204mg/dl	21mmHg	19mmHg
36	I.M		58 M	6/36	6/36	179mg/dl	29mmHg	30mmHg
37	O.E		61 M		6/9	6/12 149mg/dl	16mmHg	20mmHg
38	A.N		42 M		6/6 6/6+2	100mg/dl	10mmHg	12mmHg
39	A.E		68 F		6/9 6/36	121mg/dl	17mmHg	14mmHg
40	J.S		41 M		6/6	6/6 82mg/dl	22mmHg	17mmHg
41	O.F		42 F		6/6	6/6 92mg/dl	08mmHg	09mmHg
42	E.V		43 F		6/5	6/6 94mg/dl	18mmHg	18mmHg
43	I.P		52 M		6/9	6/9 99mg/dl	10mmHg	15mmHg
44	U.V		70 F		6/12	6/18 97mg/dl	09mmHg	12mmHg
45	I.P		69 F		6/12	6/9 136mg/dl	15mmHg	19mmHg
46	I.M		75 M		6/18 6/36	108mg/dl	17mmHg	16mmHg
47	A.A		65 M		6/9	6/9 94mg/dl	15mmHg	16mmHg
48	O.F		67 M		6/9	6/9 87mg/dl	14mmHg	14mmHg
49	O.F		68 F		6/12	6/12 102mg/dl	10mmHg	19mmHg
50	E.A		64 M		6/9	6/9 177mg/dl	20mmHg	25mmHg
51	S.E		75 F	6/36	6/36-1	159mg/dl	17mmHg	15mmHg
52	E.M		47 M		6/6 6/12+1	142mg/dl	20mmHg	23mmHg
53	E.M		60 M		6/9	6/6 159mg/dl	14mmHg	15mmHg
54	A.G		69 F		6/12	6/12 217mg/dl	24mmHg	25mmHg
55	A.J		57 F	6/9+1	6/6-2	129mg/dl	16mmHg	13mmHg
56	O.A		64 M		6/12	6/9 81mg/dl	20mmHg	19mmHg

57	J.V	67	F		6/6	6/6	90mg/dl	15mmHg	16mmHg
58	D.O	46	M	6/6+1		6/6+3	101mg/dl	09mmHg	09mmHg
59	M.M	72	F		6/18		6/12 85mg/dl	12mmHg	15mmHg
60	O.J	70	M		6/9		6/12 175mg/dl	28mmHg	24mmHg
61	A.U	73	M	6/36			6/24 115mg/dl	21mmHg	24mmHg
62	J.S	56	F		6/6		6/9 82mg/dl	17mmHg	13mmHg
63	I.S	75	M		6/12		6/12 127mg/dl	14mmHg	18mmHg
64	M.O	80	M	6/60		6/36	197mg/dl	30mmHg	28mmHg
65	R.E	71	F		6/18		6/12 90mg/dl	19mmHg	14mmHg
66	R.A	73	M		6/12		6/12 95mg/dl	09mmHg	10mmHg
67	F.E	72	F		6/18		6/9 90mg/dl	18mmHg	19mmHg
68	E.T	54	F		6/9		6/9 122mg/dl	19mmHg	18mmHg
69	T.B	64	F		6/9		6/12 87mg/dl	11mmHg	12mmHg
70	V.P	43	F		6/6		6/6 96mg/dl	16mmHg	14mmHg
71	E.I	70	M		6/9		6/9 210mg/dl	21mmHg	19mmHg
72	O.R	43	F	6/12+1			6/9 99mg/dl	11mmHg	12mmHg
73	A.A	57	F		6/9	6/36	136mg/dl	19mmHg	18mmHg
74	G.E	52	M	6/36			6/24 113mg/dl	24mmHg	20mmHg
75	E.M	54	F		6/18	6/36	72mg/dl	17mmHg	17mmHg
76	T.U	65	F		6/9		6/18 100mg/dl	13mmHg	14mmHg
77	A.F	46	F		6/6		6/6 97mg/dl	13mmHg	13mmHg
78	A.L	49	F		6/18		6/18 117mg/dl	14mmHg	13mmHg
79	W.H	41	M		6/12		6/9 93mg/dl	10mmHg	12mmHg
80	O.I	42	M		6/9	6/9+2	127mg/dl	17mmHg	17mmHg
81	O.S	71	M		6/12		6/12 159mg/dl	20mmHg	21mmHg
82	O.E	72	F		6/18		6/24 96mg/dl	16mmHg	16mmHg
83	I.P	46	F		6/9		6/9 95mg/dl	10mmHg	10mmHg
84	A.J	76	F	6/60		6/36	103mg/dl	11mmHg	14mmHg
85	M.F	47	F		6/24		6/6 97mg/dl	18mmHg	19mmHg
86	H.E	67	F		6/12		6/9 182mg/dl	22mmHg	23mmHg
87	K.U	60	F		6/9		6/9 87mg/dl	17mmHg	19mmHg
88	M.O	71	F	6/36			6/24 99mg/dl	15mmHg	14mmHg
89	T.M	68	M		6/12		6/12 101mg/dl	17mmHg	18mmHg
90	K.K	72	M		6/12		6/12 200mg/dl	33mmHg	30mmHg
91	A.N	41	M		6/9		6/6 91mg/dl	11mmHg	12mmHg
92	I.C	52	F		6/6	6/6-2	106mg/dl	14mmHg	14mmHg
93	G.A	60	M		6/18		6/12 91mg/dl	12mmHg	13mmHg
94	O.M	45	F		6/6		6/6 96mg/dl	08mmHg	10mmHg
95	O.O	57	M		6/9		6/9 126mg/dl	28mmHg	20mmHg
96	U.P	66	F		6/24		6/24 101mg/dl	07mmHg	13mmHg
97	F.E	67	F		6/12		6/9 137mg/dl	19mmHg	17mmHg
98	H.K	59	F		6/9		6/9 115mg/dl	14mmHg	15mmHg
99	A.F	51	F		6/9		6/6 109mg/dl	12mmHg	13mmHg
100	O.C	70	F		6/12		6/12 177mg/dl	28mg/dl	30mmHg