"PLASMA FIBRINOGEN LEVEL IN ADULT PATIENTS WITH TYPE 2 DIABETES MELLITUS AND ITS ASSOCIATION WITH MICROALBUMINURIA AND GLYCEMIC CONTROL AT TERTIARY CARE TEACHING HOSPITAL – ONE-YEAR CROSS SECTIONAL STUDY"

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Dr. REKHA S PATIL
MD (GENERAL MEDICINE)
Professor and Head,
Department of General Medicine,
J. N. Medical College,
Nehru Nagar, Belagavi – 10
Professor & HOD
Dept. of Medicine
J. N. Medical College Belgaum
Date : 26/03/25
Place : Belagavi

Dr.(Mrs.) N.S. MAHANTSHETTI
MD (PAEDIATRICS)
Principal,
J. N. Medical College,
Nehru Nagar, Belagavi – 10
PRINCIPAL
Jawaharlal Nehru Medical College
BELAGAVI
Date : 26/03/25
Place : Belagavi

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Accredited 'A+' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MoE (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 24-03-2025

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Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

KAHER J.N. MEDICAL COLLEGE BELAGAVI

To,
Reg. No. BG0122018
Postgraduate Student,
2022-23 Batch,
Department of General Medicine
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE LETTER

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
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Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 259. **Date: 26/05/2023**

To,

REG NO.: BG0122018
PG Student in General Medicine
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PLASMA FIBRINOGEN LEVEL IN ADULT PATIENTS WITH TYPE 2 DIABETES MELLITUS AND ITS ASSOCIATION WITH MICROALBUMINURIA AND GLYCEMIC CONTROL AT TERTIARY CARE TEACHING HOSPITAL- ONE –YEAR CROSS SECTIONAL STUDY ",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVIATIONS

ACS	-	Acute Coronary Syndrome
AGEs	-	Glycation End-Products
APTT	-	Activated Partial Thromboplastin Time
CGM	-	Continuous Glucose Monitoring
CVD	-	Cardiovascular Disease
ESRD	-	End-Stage Renal Disease
FBG	-	Fasting Blood Glucose
HbA1c	-	Glycated Hemoglobin
IDF	-	International Diabetes Federation
KDIGO	-	Kidney Disease: Improving Global Outcomes
PDR	-	Proliferative Diabetic Retinopathy
PKC	-	Protein Kinase C
PPG	-	Postprandial Glucose
PT	-	Prothrombin Time
RAAS	-	Renin-Angiotensin-Aldosterone System
SMBG	-	Self-Monitoring of Blood Glucose
T2DM	-	Type 2 Diabetes Mellitus
UACR	-	Urine Albumin-Creatinine Ratio

ABSTRACT

Background: Plasma fibrinogen, a key inflammatory marker, plays a crucial role in diabetes-related complications, including nephropathy.

Objectives: To assess plasma fibrinogen levels in patients with T2DM and to correlate plasma fibrinogen levels with microalbuminuria and glycemic control (HbA1c levels).

Methods: This hospital-based cross-sectional study was conducted over 12 months at a tertiary care hospital in Belagavi, India. Adult patients with T2DM were recruited using a convenient sampling method. Plasma fibrinogen levels were measured using the Clauss method, HbA1c using an immunoturbidimetric assay, and microalbuminuria was assessed via the urine albumin-creatinine ratio (UACR). Statistical analysis included Pearson's correlation, Student's t-test, Chi-square test, performed using SPSS v17, with a significance level of $p<0.05$.

Results: The study included 100 participants (mean age: 64.2 ± 10.7 years; 57% male) with a mean diabetes duration of 13.2 ± 8.4 years. Hypertension was the most common comorbidity (69%). Mean HbA1c was 8.2 ± 1.7 mmol/mol, with 90% having poor glycemic control (HbA1c ≥ 6.5). Retinopathy (72%), nephropathy (81%), and neuropathy (41%) were prevalent. Microalbuminuria was found in 64%, macroalbuminuria in 18%. Mean fibrinogen was 414.2 ± 92.5 mg/dL, higher in albuminuria ($p < 0.001$). Fibrinogen levels were 382.4 ± 85.2 mg/dL (normoalbuminuria), 431.6 ± 88.3 mg/dL (microalbuminuria), and 449.3 ± 94.7 mg/dL (macroalbuminuria) ($p < 0.001$). Fibrinogen correlated with albuminuria ($r = 0.62$, $p < 0.001$) and HbA1c ($r = 0.54$, $p < 0.001$).

Conclusion: Elevated plasma fibrinogen levels were significantly associated with diabetic nephropathy and glycemic control, indicating its potential as a biomarker for early risk identification and intervention.

Keywords: Fibrinogen, Type 2 Diabetes Mellitus, Glycemic control, Biomarker

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INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. It is a global health concern, with its prevalence rising at an alarming rate.(1) According to the World Health Organization (WHO), diabetes affects more than 422 million people worldwide, and this number is expected to increase significantly in the coming decades. India, often referred to as the "diabetic capital of the world," is home to more than 77 million adults with diabetes, and an additional 25 million are estimated to be prediabetic.(2) The burden of diabetes is not just limited to hyperglycemia but extends to a wide range of complications, both microvascular and macrovascular, which significantly contribute to morbidity and mortality among affected individuals.(3)

Type 2 diabetes mellitus (T2DM) is the most common form of diabetes, accounting for approximately 90% of all diabetes cases. It is characterized by insulin resistance, relative insulin deficiency, and hyperglycemia.(4) The chronic hyperglycemic state in T2DM leads to a cascade of metabolic and vascular complications, including diabetic nephropathy, retinopathy, neuropathy, and cardiovascular diseases. Among these, cardiovascular diseases (CVDs) are the leading cause of death in individuals with diabetes, accounting for nearly 75% of all diabetes-related fatalities.(5) The prothrombotic state associated with diabetes, characterized by increased coagulation and impaired fibrinolysis, plays a significant role in the development of these complications.(6)

Fibrinogen, a glycoprotein synthesized in the liver, is a key player in the coagulation cascade. It is converted to fibrin during the clotting process and is essential for thrombus formation. Beyond its role in coagulation, fibrinogen is also involved in platelet aggregation, blood viscosity, and endothelial function.[7] Elevated plasma fibrinogen levels have been identified as an independent risk factor for cardiovascular diseases, including myocardial infarction, stroke, and peripheral arterial disease.[8] In individuals with T2DM, hyperfibrinogenemia is commonly observed, and it is believed to contribute to the increased risk of thrombotic events and vascular complications.[9]

Microalbuminuria, defined as the excretion of 30-300 mg of albumin in the urine over 24 hours, is an early marker of diabetic nephropathy and a predictor of both microvascular and macrovascular complications in diabetes.[10] It is also an indicator of endothelial dysfunction and increased vascular permeability. Studies have demonstrated a strong association between microalbuminuria and elevated plasma fibrinogen levels in individuals with T2DM.[11] This association suggests that hyperfibrinogenemia may contribute to the development and progression of diabetic nephropathy and other vascular complications.[12]

Glycemic control, as measured by glycated hemoglobin (HbA1c), is a critical factor in the management of diabetes and the prevention of its complications.[13] Poor glycemic control has been consistently linked to an increased risk of microvascular and macrovascular complications, including nephropathy, retinopathy, neuropathy, and cardiovascular diseases.[14] HbA1c reflects the average blood glucose levels over the past two to three months and is a reliable indicator of long-term glycemic control.[15] Several studies have shown a positive correlation between HbA1c levels and plasma fibrinogen levels in individuals with T2DM, suggesting that poor glycemic control may exacerbate the prothrombotic state and increase the risk of vascular complications.[16,17]

The interplay between plasma fibrinogen levels, microalbuminuria, and glycemic control in T2DM is complex and multifactorial. While individual studies have explored these relationships, there is a paucity of comprehensive research that examines the combined impact of these factors on the risk of vascular complications in T2DM. Understanding these relationships is crucial for identifying high-risk individuals and implementing targeted interventions to reduce the burden of diabetes-related complications.

This study aims to investigate the plasma fibrinogen levels in adult patients with T2DM and explore its association with microalbuminuria and glycemic control. By doing so, it seeks to provide valuable insights into the role of fibrinogen in the pathogenesis of diabetic complications and its potential as a biomarker for predicting the risk of vascular complications. The findings of this study could have significant implications for the management of T2DM, particularly in the early identification and prevention of complications.

AIMS AND OBJECTIVES

Objective of the study

- **Primary objective -**To assess plasma fibrinogen levels in patients with type 2 diabetes mellitus.
- **Secondary objective -**To correlate the plasma fibrinogen levels with microalbuminuria and glycemic control (HbA1C levels) in type 2 diabetes mellitus patients.

REVIEW OF LITERATURE

TYPE 2 DIABETES MELLITUS

T2DM is a chronic metabolic disorder characterized by persistent hyperglycemia due to insulin resistance and progressive pancreatic beta-cell dysfunction. It is the most common form of diabetes, accounting for approximately 90% of all diabetes cases worldwide.(4) The disease has emerged as a major global health concern due to its increasing prevalence and associated complications. According to the International Diabetes Federation (IDF), an estimated 537 million adults were living with diabetes in 2021, with projections indicating a rise to 783 million by 2045(18)

In India, T2DM is a growing epidemic. India is home to approximately 74 million individuals with diabetes, accounting for 14% of the global diabetes burden. With 95% of these cases being T2DM, the country ranks among the global epicenters of the disease alongside the United States and China. The prevalence of diabetes in India is rising rapidly across various geographic regions and socioeconomic groups, and projections indicate that the number of affected individuals could reach 124 million by 2045. (18–20) Factors such as genetic predisposition, rapid urbanization, lifestyle changes, and increasing obesity contribute to the escalating burden of diabetes in the country.

The ADA define T2DM using standardized diagnostic criteria, which include:

- Fasting Plasma Glucose (FPG) ≥126 mg/dL (7.0 mmol/L) after an overnight fast.
- 2-hour Plasma Glucose (PG) ≥200 mg/dL (11.1 mmol/L) during an Oral Glucose Tolerance Test (OGTT).

- HbA1c ≥6.5% (48 mmol/mol), reflecting long-term glycemic control over 2-3 months.
- Random Plasma Glucose ≥200 mg/dL (11.1 mmol/L) in individuals with classic symptoms of hyperglycemia (polyuria, polydipsia, and unexplained weight loss).[21]

Pathophysiology of T2DM

T2DM is a characterized by insulin resistance and declining pancreatic beta-cell function. In the early stages, the body compensates for insulin resistance by increasing insulin secretion, thereby maintaining normal blood glucose levels. However, as the disease progresses, beta-cell function deteriorates, leading to inadequate insulin production and chronic hyperglycemia.[22]

A defining feature of T2DM is insulin resistance, where peripheral tissues such as skeletal muscle, liver, and adipose tissue fail to respond effectively to insulin. This results in impaired glucose uptake and excessive hepatic glucose production, contributing to elevated blood glucose levels. Initially, pancreatic beta cells respond by increasing insulin secretion to counteract the resistance. However, prolonged metabolic stress, oxidative damage, and apoptosis gradually impair beta-cell function, leading to insufficient insulin production. The inability of beta cells to compensate for insulin resistance is a hallmark of T2DM and marks the transition from prediabetes to overt diabetes.[23]

Obesity, particularly the accumulation of visceral fat, plays a pivotal role in the development and progression of T2DM. Dysfunctional adipose tissue releases excessive free fatty acids (FFAs) and pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which interfere with insulin signaling pathways. Additionally, imbalances in adipokines, including reduced adiponectin levels and increased leptin resistance, further impair insulin sensitivity and metabolic homeostasis.(24)

Chronic low-grade inflammation is another key contributor to insulin resistance and beta-cell dysfunction. Pro-inflammatory cytokines disrupt insulin signaling, exacerbate beta-cell stress, and accelerate disease progression. Furthermore, abnormalities in incretin hormones, particularly glucagon-like peptide-1 (GLP-1), reduce insulin secretion and promote dysregulated glucagon release. This imbalance contributes to excessive hepatic glucose production, worsening hyperglycemia.(25)

In individuals with T2DM, inappropriate glucagon secretion by pancreatic alpha cells further contributes to hyperglycemia by increasing hepatic glucose output. Additionally, the kidneys play a role in glucose dysregulation through increased glucose reabsorption via sodium-glucose co-transporter-2 (SGLT2). This reduces urinary glucose excretion and prolongs hyperglycemia, further complicating metabolic control.(23)

Emerging research suggests that alterations in gut microbiota composition may influence insulin resistance, systemic inflammation, and glucose metabolism. Changes in the gut microbiome can impact incretin hormone secretion, intestinal permeability, and immune system activation, further contributing to metabolic dysfunction in T2DM(26) Therefore, these interconnected mechanisms drive disease progression and contribute to long-term complications. Understanding these processes is essential for developing targeted therapeutic strategies to manage and prevent T2DM effectively.

Complications of T2DM

T2DM is associated with a range of acute and chronic complications that significantly impact overall health and quality of life. Acute complications include diabetic ketoacidosis (DKA), a potentially life-threatening emergency characterized by severe dehydration, Kussmaul breathing (deep, labored breathing), acetone-scented breath, and abdominal pain. If untreated, DKA can progress to altered consciousness, lethargy, and even coma. Hypoglycaemia, another critical complication, occurs due to excessive insulin administration, strenuous exercise, or insufficient food intake. It presents with symptoms such as sweating, tremors, dizziness, irritability, blurred vision, confusion, and, in severe cases, unconsciousness or coma. Immediate intervention with oral or intravenous glucose is necessary to prevent life-threatening consequences. Hyperglycaemia, characterized by persistently high blood sugar levels, occurs due to insufficient insulin or poor glycaemic control, increasing the risk of long-term complications if not properly managed through medication, diet, and lifestyle modifications.[(27)]

Chronic complications of T2DM develop over time and affect multiple organ systems, often leading to serious health consequences. Macrovascular complications include cardiovascular diseases such as hypertension, atherosclerosis, coronary artery disease, and stroke, which are the leading causes of mortality in individuals with diabetes. Microvascular complications include diabetic retinopathy, which results from progressive damage to the blood vessels in the retina, potentially leading to vision impairment and blindness. Diabetic nephropathy, another major complication, can cause chronic kidney disease and progress to end-stage renal failure, necessitating dialysis or kidney transplantation. Diabetic neuropathy, affecting the peripheral and autonomic nervous systems, leads to sensory disturbances, chronic pain, muscle weakness, walking difficulties, and complications such as diabetic foot ulcers, infections, and, in severe cases, gangrene requiring amputations.[28]

Furthermore, diabetes significantly increases susceptibility to infections due to immune system dysfunction, contributing to a higher prevalence of skin infections, urinary tract infections, pneumonia, and foot ulcers. Metabolic complications such as osteoporosis, myopathy, and liver disorders are also more common in individuals with T2DM. Cardiovascular autonomic neuropathy, a condition associated with diabetes, can impair heart rate regulation and blood pressure control, increasing the risk of silent myocardial ischemia, arrhythmias, and sudden cardiac death. Individuals with diabetes have a two- to eightfold increased risk of heart disease-related mortality, and nearly 75% of deaths in diabetic patients are attributed to cardiovascular disease.[29] Moreover, studies suggest that diabetes is linked to an increased risk of atrial fibrillation, although this association may be influenced by coexisting conditions such as hypertension and ischemic heart disease.[30] Understanding and managing these complications through proper glycaemic control, lifestyle modifications, and early medical interventions are crucial for preventing disease progression and improving patient outcomes.

Role of Glycaemic Control in T2DM

Glycaemic control is fundamental in the management of T2DM, directly influencing the progression of the disease and the risk of complications. Maintaining blood glucose levels within an optimal range significantly reduces both microvascular complications, such as diabetic retinopathy, nephropathy, and neuropathy, and macrovascular complications, including cardiovascular disease (CVD). Chronic hyperglycaemia leads to the accumulation of advanced glycation end-products

(AGEs), which impair protein function, activate inflammatory pathways, and contribute to endothelial dysfunction. Additionally, persistent hyperglycaemia activates protein kinase C (PKC), leading to increased oxidative stress and vascular damage. These metabolic disturbances collectively promote systemic inflammation, thrombosis, and progressive organ damage. Effective glycaemic management not only mitigates these risks but also improves the overall quality of life by reducing diabetes-related symptoms, enhancing energy levels, and preventing acute complications such as diabetic ketoacidosis and hyperosmolar hyperglycaemic state.(27,28,31)

The primary goal of glycaemic control is to maintain glucose levels that minimize long-term complications while avoiding the risks of hypoglycaemia. The ADA recommends an HbA1c target of <7% for most adults with diabetes, though individualized targets are necessary based on age, disease duration, comorbidities, and risk of hypoglycaemia. More aggressive targets (<6.5%) may be beneficial for younger patients with a recent diagnosis and no cardiovascular history, while more relaxed targets (<8%) are considered for older adults, those with severe hypoglycaemia risk, or individuals with limited life expectancy.(21,32) Measurement of glycaemic control involves multiple methods, including HbA1c, self-monitoring of blood glucose (SMBG), continuous glucose monitoring (CGM), fasting blood glucose (FBG), and postprandial glucose (PPG). HbA1c provides a long-term average of blood glucose levels over 2-3 months, offering a reliable measure of metabolic control. However, limitations such as variations due to anaemia, hemoglobinopathies, and ethnicity necessitate the use of complementary glucose-monitoring strategies. CGM provides real-time glucose trends, allowing for proactive interventions, while SMBG is particularly useful for insulin-treated individuals to prevent hypoglycaemia.(33,34)

FIBRINOGEN

Fibrinogen is a key glycoprotein involved in blood clotting, inflammation, thrombosis, and wound healing. Synthesized primarily in the liver, fibrinogen circulates in the blood at a concentration of 150–400 mg/dL and plays a crucial role in maintaining vascular integrity. Structurally, it consists of two identical sets of three polypeptide chains—Aα, Bβ, and γ—linked to form a trinodular, dimeric molecule. During coagulation, fibrinogen serves as a precursor to fibrin, which is essential for clot formation. When vascular injury occurs, thrombin cleaves fibrinogen, releasing fibrinopeptides A and B and allowing fibrin monomers to polymerize. These fibrin strands are then cross-linked by factor XIIIa to create a stable fibrin network, reinforcing the clot and preventing excessive blood loss. Fibrinogen also enhances platelet aggregation by binding to glycoprotein IIb/IIIa receptors on platelets, further strengthening clot formation.[7,35]

Beyond its role in coagulation, fibrinogen is a key player in inflammation, thrombosis, and wound healing. As an acute-phase protein, its levels rise in response to tissue injury, infection, and systemic inflammation, promoting leukocyte adhesion, cytokine production, and endothelial activation. This inflammatory response leads to vascular dysfunction and oxidative stress, which accelerate atherosclerosis and elevate the risk of cardiovascular diseases like stroke and myocardial infarction. Studies have shown a strong association between high fibrinogen levels and an increased likelihood of ischemic cardiovascular events.[35–39]

One of the primary mechanisms through which fibrinogen contributes to cardiovascular disease is its involvement in atherogenesis. When converted into fibrin, it promotes plaque formation and facilitates lipid deposition within arterial walls, triggering localized inflammation and the progression of atherosclerosis. Furthermore, fibrinogen plays a crucial role in thrombus formation by serving as a scaffold for blood clots, enhancing platelet aggregation, and stabilizing fibrin networks. This process makes thrombi more resistant to fibrinolysis, increasing the likelihood of arterial occlusion, heart attacks, and strokes. Additionally, fibrin degradation products can contribute to plaque instability, leading to rupture and acute cardiovascular events. In patients with conditions such as end-stage renal disease (ESRD) and acute coronary syndrome (ACS), elevated fibrinogen levels—particularly in the presence of diabetes—are linked to a significantly higher risk of both cardiovascular and all-cause mortality.(8,36,39)

Beyond its role in clot formation and vascular pathology, fibrinogen interacts with other biological pathways that influence cardiovascular health. Notably, it acts as a selective inhibitor of matrix metalloproteinase 2 (MMP-2), an enzyme essential for extracellular matrix remodelling. Dysregulation of this interaction has been implicated in the development of arthritic and cardiac disorders, further highlighting the complex and multifaceted role of fibrinogen in disease progression.(40) Given its critical involvement in inflammation, thrombosis, and vascular pathology, plasma fibrinogen serves as both a biomarker and a potential therapeutic target for cardiovascular disease management.(41)

Role of Fibrinogen in T2DM

Fibrinogen plays a critical role in the pathophysiology of T2DM, contributing to a procoagulant and proinflammatory state. Patients with T2DM frequently exhibit hyperfibrinogenaemia, characterized by elevated plasma fibrinogen levels compared to non-diabetic individuals.(42) This increase in fibrinogen levels significantly contributes to the heightened risk of vascular complications seen in diabetic patients. Multiple studies have demonstrated that fibrinogen levels are consistently higher in individuals with diabetes, particularly those suffering from both microvascular and macrovascular complications. Furthermore, hyperfibrinogenaemia, defined as plasma fibrinogen levels exceeding 400 mg/dL, is more commonly observed in T2DM patients who also present with metabolic syndrome.[(43,44)]

Several mechanisms underlie the elevated fibrinogen levels observed in T2DM, with hyperglycaemia and insulin resistance playing central roles. Poor glycaemic control, as indicated by elevated HbA1c levels, is positively correlated with increased fibrinogen levels. Glycosylated fibrinogen exhibits reduced susceptibility to plasmin-mediated degradation, leading to its accumulation and further exacerbating the prothrombotic environment. Insulin resistance also plays a role by altering protein synthesis, leading to reduced albumin synthesis while simultaneously increasing fibrinogen production. Chronic low-grade inflammation, a hallmark of T2DM, further drives fibrinogen synthesis as part of the acute-phase response. Markers of inflammation, as in high-sensitivity C-reactive protein are often elevated alongside fibrinogen, highlighting their interconnected roles in the inflammatory process. Additionally, oxidative stress resulting from chronic hyperglycaemia disrupts glucose metabolism, leading to neuronal damage and prolonged inflammatory responses, which further contribute to fibrinogen elevation[(45)]

MICROALBUMINURIA

Microalbuminuria is characterized by albumin levels between 30 and 300 mg in a 24-hour urine sample. It is recognized as an early predictor of diabetic nephropathy, a major microvascular complication of T2DM. The presence of microalbuminuria signifies early glomerular damage, reflecting a breakdown in the kidney's filtration barrier.[46,47]

The most accurate method for detecting microalbuminuria is a 24-hour urine collection, as it has the lowest variability.[48] However, due to its labour-intensive nature, alternative methods such as the urine albumin-to-creatinine ratio (UACR) are commonly used. While UACR accounts for urine concentration and volume variations, it can still be influenced by other factors. Spot urine collections are more convenient but may fluctuate depending on urine volume. Other diagnostic techniques include immunoturbidimetry, immunonephelometry, enzyme-linked immunosorbent assays (ELISA), immunoassays with latex particles, radial immunodiffusion, and fluoroimmunoassay, which offer sensitivity comparable to a 24-hour urine test.[49]

According to the ADA and Kidney Disease: Improving Global Outcomes (KDIGO) guidelines, microalbuminuria is defined as an ACR of 30-300 mg/g (3.4-33.9 mg/mmol) in a spot urine sample or a 24-hour urine albumin excretion rate of 30-300 mg/24 hours. Some guidelines advocate the use of two out of three urine collections over a 3-6 month period to confirm microalbuminuria.[50] The American Diabetes Association recommends annual screening for microalbuminuria in individuals with Type 1 diabetes if the diagnosis was made more than five years ago. For patients with Type 2 diabetes and diabetic nephropathy, annual screening should begin at the time of diagnosis.[51]

Pathophysiology of Microalbuminuria in T2DM

The development of microalbuminuria in T2DM is multifactorial, with hyperglycaemia and hypertension playing central roles. Chronic hyperglycaemia leads to increased glomerular filtration pressure and structural changes in the glomerular capillaries, causing damage to the glomerular filtration barrier. This damage allows albumin, a normally large protein, to leak into the urine. Hypertension exacerbates glomerular damage by increasing pressure within the glomerular capillaries, further impairing kidney function. Other contributing factors include oxidative stress, AGEs, and activation of protein kinase C (PKC), which promote inflammation, endothelial dysfunction, and extracellular matrix accumulation in the glomeruli. These pathological changes further aggravate albuminuria. The renin-angiotensin-aldosterone system (RAAS) also plays a crucial role in the pathogenesis of microalbuminuria. Angiotensin II, a key component of the RAAS, increases glomerular pressure, promotes inflammation, and stimulates the production of extracellular matrix proteins, leading to progressive kidney damage.[47,52,53]

Microalbuminuria is a significant clinical marker as it serves as a strong predictor for the progression to overt nephropathy and ESRD in patients with T2DM. Individuals with persistent microalbuminuria face an increased risk of progressive renal function decline, which may ultimately require dialysis or kidney transplantation. Beyond its implications for kidney health, microalbuminuria is also strongly associated with an elevated risk of cardiovascular diseases (CVD) in individuals with T2DM. It often coexists with other cardiovascular risk factors such as hypertension, dyslipidaemia, and insulin resistance, further contributing to an increased cardiovascular risk profile. Studies have shown that T2DM patients with microalbuminuria have a higher incidence of myocardial infarction, stroke, and cardiovascular-related mortality compared to those without microalbuminuria.(54,55)

ASSOCIATION BETWEEN FIBRINOGEN, MICROALBUMINURIA, AND GLYCEMIC CONTROL

The study by Hamidullah et al. (2024) examined the relationship between plasma fibrinogen levels and microvascular complications in T2DM, with a particular emphasis on diabetic nephropathy, retinopathy, and neuropathy. Among the findings, diabetic nephropathy showed a significant association with increased plasma fibrinogen levels and key renal biomarkers, particularly albuminuria ($p<0.05$). Patients with nephropathy had elevated fibrinogen levels alongside higher albuminuria, creatinine, and urea, indicating a progressive decline in kidney function. The study underscores the role of hyperfibrinogenemia in worsening albuminuria and nephropathy severity, highlighting the importance of fibrinogen monitoring for early intervention in diabetic kidney disease.(56)

Abdelmonem M et al. (2023) conducted a cross-sectional analytic study at the Benghazi Diabetic Clinic, Libya, to assess the association between plasma fibrinogen levels and glycemic control in 231 T2DM patients aged 20–80 yearsPlasma fibrinogen levels were estimated using the Clauss method, while HbA1c was measured through the immunoturbidimetric method. Patients were categorized based on HbA1c levels into good control (<5.7%), fair control (5.7–6.5%), and poor control (>6.5%) groups. Results showed a significant positive correlation between fibrinogen and HbA1c in both the fair ($r=0.424$, $p=0.003$) and poor control ($r=0.369$, $p=0.000$) groups, indicating that poor glycaemic control is associated with higher fibrinogen levels. Age also correlated with fibrinogen levels in the good control group ($p>0.05$).

The study concluded that elevated plasma fibrinogen levels in T2DM may contribute to increased cardiovascular risk, and reducing fibrinogen could be a potential strategy to mitigate diabetes-related complications.[(57)]

The study by Borawake A G et al. (2022) evaluated the association between plasma fibrinogen levels, microalbuminuria, and glycemic control in T2DM through a hospital-based cross-sectional study involving 100 diabetic patients. Statistical analysis using SPSS software revealed that the mean plasma fibrinogen level was 507.8 mg/dl, with 26% of patients having levels above 500 mg/dl. Microalbuminuria and macroalbuminuria were present in 25% and 9% of cases, respectively. Higher fibrinogen levels were significantly associated with diabetes duration over 5 years, poor glycemic control, and microalbuminuria ($p<0.05$). The study suggests that hyperfibrinogenemia may precede clinical vascular complications, highlighting its role as a potential early marker for diabetic nephropathy and cardiovascular risk in T2DM.(58)

Ghongade P V et al. (2022) conducted a study to examine the relationship between plasma fibrinogen and glycemic control in patients with T2DM. The study included 108 diabetic patients, categorized into 69 complicated and 39 uncomplicated cases, along with 100 non-diabetic controls. The results indicated that plasma fibrinogen levels were significantly elevated in patients with complicated T2DM (450.43 ±108.51 mg/dl) vs uncomplicated cases (372.30 ±123.78 mg/dl $p<0.05$The mean HbA1c level in diabetic patients was 8.02 ± 1.88 mg/dL, ranging from 5.50 to 14.50 mg/dL. A significant positive correlation was found between HbA1c and fibrinogen levels ($r = 0.782$, $p = 0.001$). Additionally, diabetes duration showed a significant association with fibrinogen levels ($r = 0.295$, $p = 0.002$). The study concluded that higher plasma fibrinogen levels are linked to poor glycemic control and prolonged disease duration, indicating that lowering fibrinogen levels may play a crucial role in preventing cardiovascular complications in diabetic patients.[59]

Das U et al. (2019) conducted a hospital-based observational cross-sectional study to assess the association between plasma fibrinogen levels and microvascular complications (retinopathy and microalbuminuria) in T2DM. The study included 100 randomly selected diabetic patients, with a male-to-female ratio of 60:40. Among them, 35% had non-proliferative diabetic retinopathy (NPDR) and 18% had proliferative diabetic retinopathy (PDR). The mean fibrinogen level was 325.49 mg/dL, and a statistically significant association was found between fibrinogen levels and urinary albumin-to-creatinine ratio (ACR), as well as with diabetic retinopathy. Age, BMI, diabetes duration, creatinine levels, and medication use (statins and ACE inhibitors) were also significantly correlated with diabetic retinopathy. Additionally, serum HDL and CRP levels were inversely related to both retinopathy and ACR. The study concluded that serum fibrinogen levels could serve as a surrogate marker for diabetic nephropathy and retinopathy, similar to their established role in cardiovascular diseases. This finding is relevant to our study as it reinforces the link between hyperfibrinogenemia and microvascular complications in diabetes.[60]

Agarwal C et al. (2018) investigated the coagulation profile in T2DM and its association with microvascular complications and glycemic control in a tertiary care center in Delhi. The study included 60 diabetic patients (30 with complications and 30 without) and 30 healthy controls. Diabetics with complications had significantly higher fibrinogen levels and lower activated partial thromboplastin time (APTT) compared to those without complications ($p < 0.00001$), while prothrombin time (PT) showed no significant difference. Higher HbA1c levels (>7%) were associated with increased fibrinogen and reduced APTT, indicating a hypercoagulable state. The study concluded that fibrinogen, APTT, and PT could serve as useful markers for assessing thrombotic risk in diabetic patients, particularly those with poor glycemic control and microvascular complications. These findings support our research by highlighting the role of fibrinogen in coagulation abnormalities in diabetes and its potential as a predictive marker for vascular complications.[61]

Dhawale S et al. (2016) studied serum fibrinogen levels in T2DM patients and their association with glycemic control, microvascular complications, and pharmacotherapy. The study included 60 diabetic patients (34 with complications and 26 without) and 28 healthy controls. Serum fibrinogen levels were significantly higher in diabetics with complications (515±138.7 mg/dL) compared to those without complications (437±137 mg/dL) and controls (308±52.65 mg/dL). Raised fibrinogen levels were observed among those who were overweight, those who had poor glycemic control (HbA1C >12%), and those with elevated cholesterol (>200 mg/dL). Patients on insulin had the highest fibrinogen levels (640.8±126.4 mg/dL) compared to those on oral hypoglycemic agents or no treatment. This study concluded that fibrinogen levels were elevated in T2DM, particularly among patients with microvascular complications, highlighting the need for further research on its role in diabetes management.[62]

Gupta P et al. (2016) conducted a comparative observational study at a tertiary care teaching hospital in Madhya Pradesh to evaluate plasma fibrinogen levels in T2DM patients and their relation to glycemic control and cardiovascular risk factors. The study compared fibrinogen levels in diabetic patients and healthy controls and analyzed their correlation with glycemic parameters (HbA1c), demographic factors (age, sex), and clinical conditions (hypertension, BMI, ischemic heart disease, and smoking). Results showed that diabetic patients had significantly higher fibrinogen levels (386.04±132.87 mg/dl) than healthy controls (314.38±97.42 mg/dl; p<0.001). A positive correlation (r=0.24) was found between HbA1c and fibrinogen levels, indicating that poor glycemic control is associated with elevated fibrinogen levels. The study concluded that hyperfibrinogenemia in T2DM could contribute to increased cardiovascular morbidity and mortality, highlighting its role as a thrombogenic factor in diabetes-related complications.(63)

Saini P K et al. (2016) conducted a study to estimate plasma fibrinogen levels in sixty T2DM patients and thirty matched controls and analyze its association with microalbuminuria, glycemic control, and other risk factors. Plasma fibrinogen levels were determined using the Clauss method, while glycemic control was evaluated through HbA1c measured by ion exchange HPLC. Findings revealed that mean fibrinogen levels were significantly elevated in diabetic patients (380.03 ± 101.07 mg/dL) compared to healthy controls (244.43 ± 61.27 mg/dL, p < 0.0001). Significant correlations were observed between fibrinogen levels and age (p = 0.003), BMI (p = 0.016), lipid profile parameters (total cholesterol, LDL, HDL, triglycerides), microalbuminuria (p < 0.0001), and glycemic control (p < 0.0001). However, no significant association was found with sex, hypertension, diabetes duration, or smoking. Multiple linear regression identified age (p = 0.04), BMI (p = 0.038), and microalbuminuria (p = 0.008) as independent predictors of elevated fibrinogen levels. The study concluded that fibrinogen levels are markedly higher in T2DM patients and are strongly linked to poor glycemic control and microalbuminuria, highlighting an increased risk for cardiovascular and renal complications.(64)

Mir A M et al. (2015) investigated the relationship between serum fibrinogen levels and diabetic retinopathy in 101 T2DM patients at Government Medical College, Srinagar. The study divided patients into two groups: 50 with diabetic retinopathy and 51 without. The mean diabetes duration was longer in patients with retinopathy (10.9 years) compared to those without (5.7 years). Serum fibrinogen levels were significantly higher in patients with retinopathy (5.310 g/L) than in those without (3.783 g/L), with 72% of retinopathy patients exhibiting elevated fibrinogen levels. The study concluded that high plasma fibrinogen levels contribute significantly to the development of diabetic retinopathy. This finding is relevant to our study as it further highlights the role of hyperfibrinogenemia in diabetic microvascular complications[65]

Bembde A S (2011) conducted a study to assess plasma fibrinogen levels in 100 T2DM patients and 100 age- and sex-matched controls and their relationship with glycemic control. Fibrinogen levels were significantly higher in T2DM patients (656, SD-130 mg/dL) vs controls (324, SD-139 mg/dL) ($P < 0.01$). In diabetics, fibrinogen values were significantly correlated with age, hypertension, BMI, smoking, ischemic heart disease and glycosylated hemoglobin (HbA1c, $r = 0.49$), but not with sex ($P > 0.05$). Among controls, fibrinogen levels correlated with smoking ($P < 0.01$) and BMI ($P < 0.01$). The study concluded that patients with T2DM have an increased prevalence of hyperfibrinogenemia, and fibrinogen values are independently correlated with HbA1c, indicating a possible role in increased cardiovascular risk among diabetic patients[45]

Barazzoni R et al. (2003) studied the effect of insulin on fibrinogen production in Type 2 diabetes (T2DM) and non-diabetic individuals. Using a leucine isotopic model, they found that basal fibrinogen concentration and absolute synthesis rate (ASR) were 35% higher ($P < 0.05$) in T2DM patients compared to controls. After a 4-hour euglycemic, euaminoacidemic-hyperinsulinemic clamp, fibrinogen fractional and absolute synthesis rates increased by 41% and 43% ($P < 0.05$) in T2DM patients, whereas insulin had no effect in non-diabetic individuals. The findings suggest that insulin acutely increases fibrinogen production in T2DM, contributing to hyperfibrinogenemia and cardiovascular risk, while plasma amino acids may regulate fibrinogen production in non-diabetics.(66)

Jain A et al. (2001) investigated the relationship between plasma fibrinogen, glycemic control, and urinary albumin excretion rate in diabetic patients. The study, which included 50 diabetics and 10 matched controls, found that fibrinogen levels were significantly higher in diabetics (7.30 ± 5.87) than the controls (4.06 ± 2.5; $p = 0.022$). In diabetics, fibrinogen levels showed a strong positive correlation with age, hypertension, BMI, triglycerides, cholesterol, HbA1c, and urinary albumin excretion rate, while no significant association was found with sex, family history, duration of diabetes, or smoking. The urinary albumin excretion rate was also significantly elevated in diabetics and linked to age and lipid profile. The study concluded that hyperfibrinogenemia in diabetics is closely associated with poor glycemic control and increased urinary albumin excretion, contributing to their heightened cardiovascular risk.(67)

MATERIALS AND METHODS

Source of Data

The data for this study was collected from adult patients diagnosed with Type 2 Diabetes Mellitus (T2DM) attending the Outpatient Department (OPD) and Inpatient Department (IPD) of a tertiary care teaching hospital.

Study Design

This study is a hospital-based cross-sectional study.

Study Period

The study was conducted over a period of 12 months, from 1st January 2023 to 31st December 2023. This duration has been chosen to ensure an adequate sample size is achieved and to account for seasonal variations in patient attendance and disease presentation.

Place of Study

The study was conducted at a tertiary care teaching hospital in Belagavi, Karnataka, India. The hospital's Department of General Medicine did facilitate the recruitment of patients and the collection of data.

Sample Size

Formula used for sample size calculation is,

$$n = \left(\frac{Z_{(1-\alpha/2)} + Z_{(1-\beta)}}{C}\right)^2 + 3$$

where n is the sample size required, α is the level of significance, (1-β) is power, $Z_{(1-\alpha/2)}$ at 5% level of significance is 1.96, $Z_{(1-\beta)}$ is 1.0364 at 85% power and $C = 0.5 * \ln\left[\frac{(1+r)}{(1-r)}\right]$.

The correlation between HbA1c and fibrinogen levels of the diabetic patient was foun d to be 0.49. Considering that the correlation between HbA1c and fibrinogen levels in our study to be at least 0.3, at 5% level of significance and 85% power, we have(68)

$$C = 0.5 * \ln\left[\frac{(1+r)}{(1-r)}\right]$$

$$= 0.5 * ln\left[\frac{(1+0.3)}{(1-0.3)}\right]$$

$$= 0.5 * \ln\left[\frac{1.3}{0.7}\right]$$

$$= 0.5 * \ln[1.857143]$$

$$= 0.5 * 0.6190392$$

$$= 0.3095196$$

Hence,

$$n = \left(\frac{Z_{\left(1-\frac{\alpha}{2}\right)} + Z_{(1-\beta)}}{C}\right)^2 + 3$$

$$= \left(\frac{1.96 + 1.0364}{0.3095196}\right)^2 + 3$$

$$= 93.71805 + 3$$

$$= 96.71805 \approx 97$$

Therefore, the minimum sample size required is 97. As sample size increases, accuracy of result also increases, so the estimated sample size is 100, where alpha=5%, beta=15%, 1-beta=85%

Sampling Method

A convenient sampling method was used to recruit participants for this study. All consecutive patients attending the OPD and IPD of the tertiary care hospital who meet the inclusion criteria were invited to participate. Convenient sampling is appropriate for this study as it allows for the efficient recruitment of participants within the study period while ensuring that the sample is representative of the patient population at the hospital.

Inclusion Criteria

The following inclusion criteria was used to select participants for the study:

1. Age: Patients aged 18 years and above.
2. Diagnosis: Patients diagnosed with Type 2 Diabetes Mellitus according to the American Diabetes Association (ADA) guidelines.
3. Consent: Patients who provide written informed consent to participate in the study.

Exclusion Criteria

Patients with the following conditions was excluded from the study:

1. Type 1 Diabetes Mellitus: Patients with Type 1 diabetes were excluded as the pathophysiology and complications differ from those of Type 2 diabetes.
2. Non-Diabetic Albuminuria: Patients with albuminuria due to causes other than diabetes (e.g., chronic kidney disease, urinary tract infections, etc.) were excluded.
3. Chronic Kidney Disease: Patients with documented chronic kidney disease (CKD) were excluded as CKD can independently affect fibrinogen levels.

4. Pregnancy: Pregnant females were excluded due to the physiological changes in coagulation and metabolism during pregnancy.
5. Gout and Anti-Inflammatory Drugs: Patients with gout or those on anti-inflammatory drugs or allopurinol were excluded as these conditions and medications can influence fibrinogen levels.
6. Acute Illnesses: Patients with acute febrile illnesses, urinary tract infections, pyelonephritis, urinary tract obstruction, congestive heart failure, or acute coronary syndrome were excluded as these conditions can affect fibrinogen levels and glycemic control.

Method of Data Collection

Data collection was conducted using a structured and systematic approach to ensure the accuracy and reliability of the information gathered. The process begins with obtaining written informed consent from all participants prior to their inclusion in the study. The consent form will clearly explain the purpose of the study, the procedures involved, potential risks and benefits, as well as the voluntary nature of participation.

Next, a detailed clinical history was recorded, which included demographic details such as age and sex, the duration of diabetes, and any history of complications like retinopathy, neuropathy, or nephropathy. A thorough clinical examination was also carried out, measuring blood pressure, body mass index (BMI), and other relevant parameters.

Laboratory investigations played a crucial role in the data collection process. Plasma fibrinogen levels was assessed using the Clauss method, a widely accepted and reliable technique. HbA1c was measured with particle-enhanced immunoturbidimetric methodology to evaluate long-term glycemic control. Additionally, the urine albumin: creatinine ratio (UACR) was measured to detect the presence of microalbuminuria or macroalbuminuria. A complete blood count was also performed to rule out any hematological abnormalities, along with other tests, such as a lipid profile and renal function tests, to assess additional risk factors.

The assessment of diabetic complications involved direct ophthalmoscopy to evaluate for diabetic retinopathy. For diabetic neuropathy, a 10-g monofilament test, assessment of pinprick sensation, vibration sense using a 128Hz tuning fork, temperature sense, and ankle jerk reflex was employed. The Revised Neuropathy Disability Score (NDS) was used for scoring these evaluations. Furthermore, the urine albumin: creatinine ratio (UACR) was again utilized to assess the presence of microalbuminuria or macroalbuminuria related to diabetic nephropathy.

Finally, all data was systematically recorded in a structured case record form (CRF) specifically designed for this study. The CRF included sections dedicated to demographic details, clinical history, examination findings, and laboratory results, ensuring that the data collected is comprehensive and well-organized.

Statistical Analysis

The collected data was analyzed using appropriate statistical methods to achieve the research objectives. Initially, data was entered into a computer-based spreadsheet, where it was checked for accuracy and completeness. Any inconsistencies or missing data was addressed prior to analysis. Descriptive statistics was employed, with categorical variables (e.g., sex, presence of complications) expressed as frequencies and percentages, and continuous variables (e.g., age, plasma fibrinogen levels, HbA1c) presented as mean ± standard deviation (SD) or median (minimum, maximum), depending on the data distribution. For inferential statistics, a Student's t-test was conducted to compare the mean values of continuous variables between two groups, such as T2DM patients with and without microalbuminuria. Additionally, Pearson's Correlation Coefficient (r) was used to assess the degree of association between plasma fibrinogen levels and other continuous variables (e.g., HbA1c, UACR), while a Chi-Square Test evaluated the association between categorical variables (e.g., presence of complications and fibrinogen levels). Finally, the analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 17, with a p-value of less than 0.05 considered statistically significant.

Ethical Considerations

The study is conducted in accordance with the ethical guidelines for biomedical research involving human participants, as outlined by the Institutional Ethics Committee (IEC) of the tertiary care hospital. Ethical clearance is obtained before the commencement of the study. Participants was informed about the study's purpose, procedures, risks, and benefits, and their participation was entirely voluntary. Confidentiality of the data will be maintained, and only aggregated data will be used for publication.

RESULTS

Table 1. Age distribution of the study participants (N=100)

Age (years)	Frequency (n)	Percentage (%)
21-40	2	2.0
41-60	32	32.0
61-80	62	62.0
>80	4	4.0
Total	**100**	**100.0**

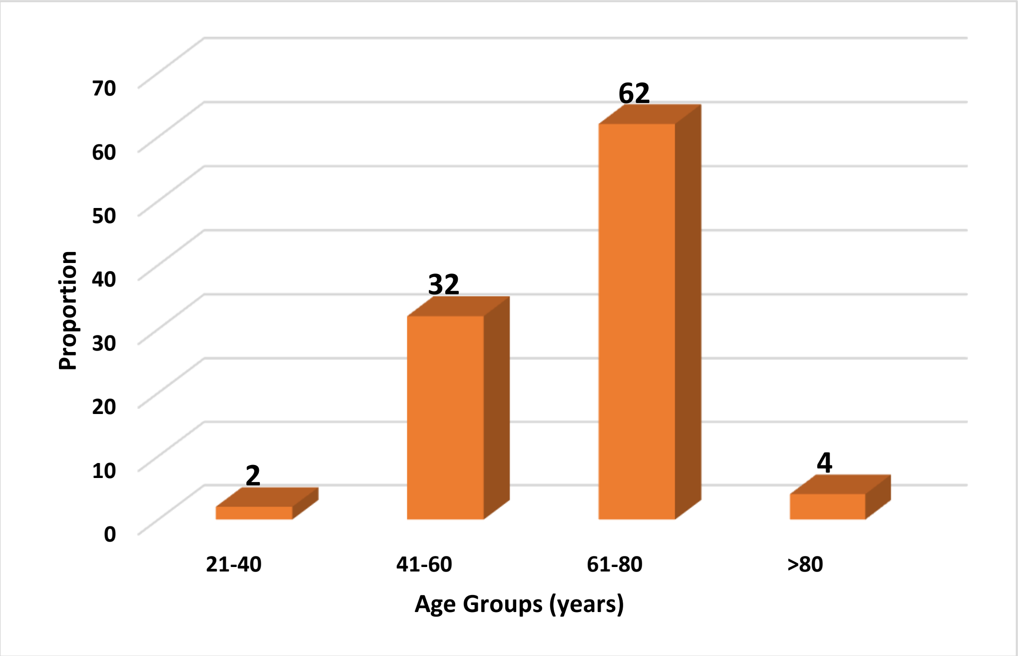

Figure 1. Age distribution of the study participants

The age distribution of the study participants, totalling 100 individuals, reveals that most participants are in the older age groups. A majority of the participants, 62% (n=62), are aged between 61 and 80 years. The second-largest group, accounting for 32% (n=32), is between 41 and 60 years old. A small percentage of participants, 4% (n=4), are older than 80 years, and only 2% (n=2) fall within the 21-40 age range.

Table 2. Gender distribution of the study participants (N=100)

Gender	Frequency (n)	Percentage (%)
Male	57	57.0
Female	43	43.0
Total	**100**	**100.0**

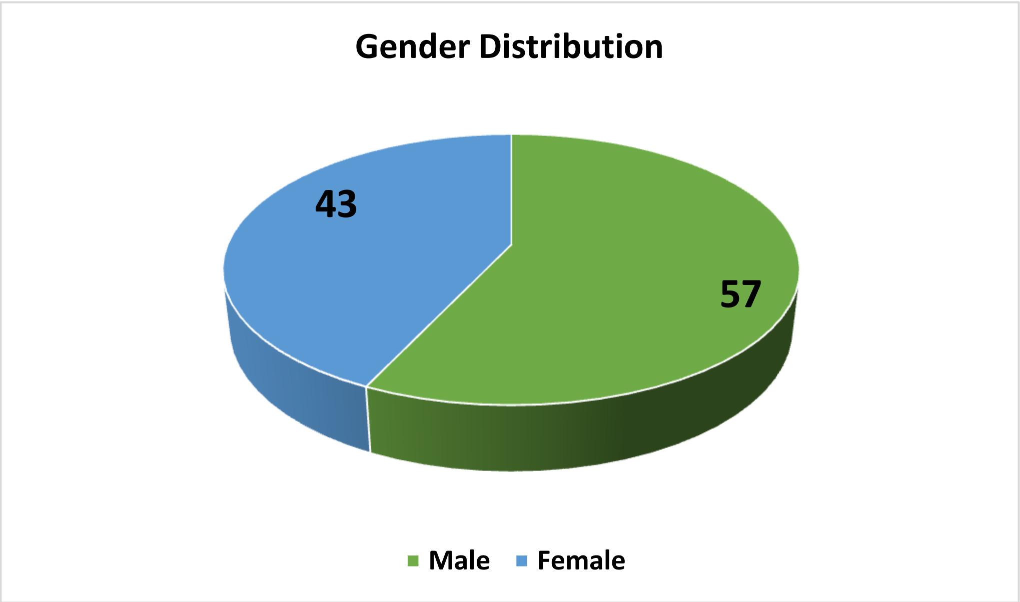

Figure 2. Gender distribution of the study participants

The gender distribution of the study participants shows that the majority are male, making up 57% (n=57) of the participants. Female participants account for 43% (n=43) of the total.

Table 3. Duration of diabetes mellitus distribution of the study participants (N=100)

Duration of DM (years)	Frequency (n)	Percentage (%)
1-10	51	51.0
11-20	36	36.0
21-30	11	11.0
31-40	2	2.0
Total	**100**	**100.0**

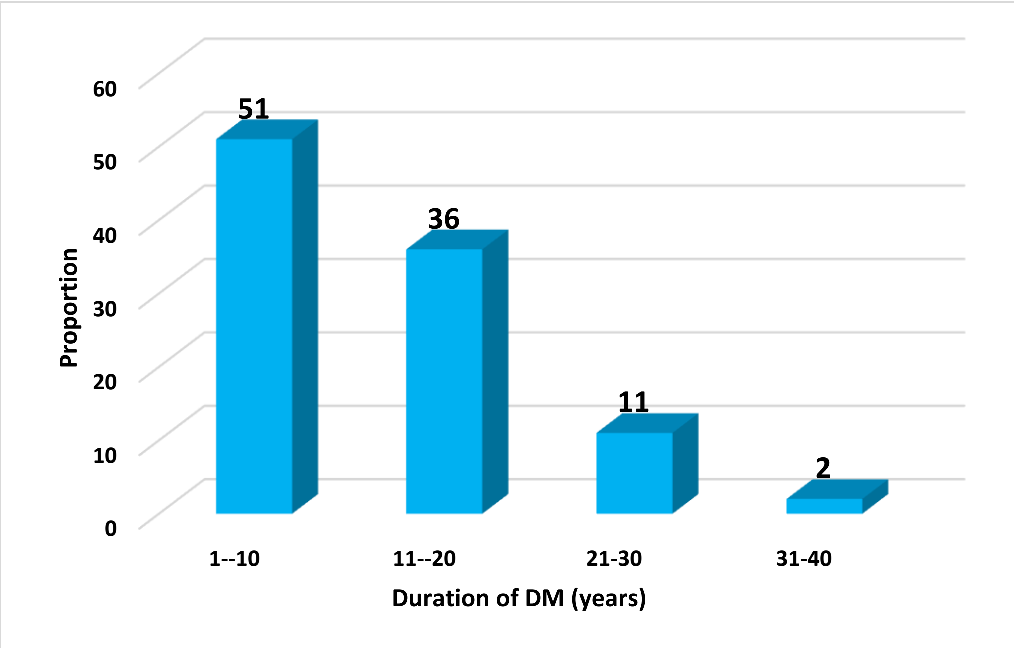

Figure 3. Duration of diabetes mellitus distribution of the study participants

The duration of diabetes mellitus (DM) among the study participants is predominantly in the 1-10 years range, with 51% (n=51) of participants falling into this category. The next largest group, 36% (n=36), has had diabetes for 11-20 years. A smaller proportion, 11% (n=11), have been living with diabetes for 21-30 years. Only 2% (n=2) of participants have had diabetes for 31-40 years.

Table 4. Risk factors distribution of the study participants (N=100)

Risk Factors	Frequency (n)	Percentage (%)
Hypertension	69	69.0
IHD	20	20.0
CVA	9	9.0
Hypothyroidism	4	4.0
Alcohol	3	3.0
Smoking	3	3.0
Nil	27	27.0

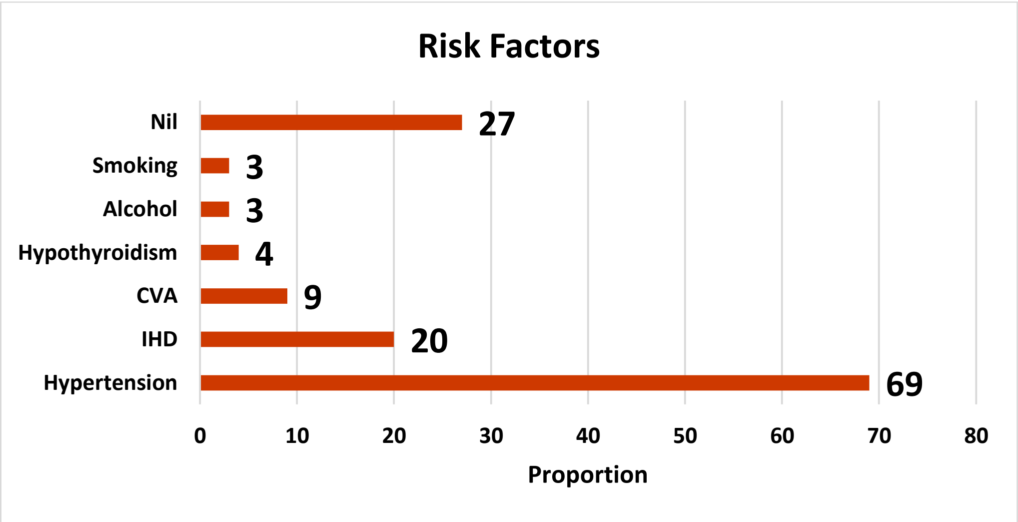

Figure 4. Risk factors distribution of the study participants

The risk factor distribution among the study participants shows that hypertension is the most common risk factor, affecting 69% (n=69) of participants. Ischemic heart disease (IHD) is present in 20% (n=20), while 9% (n=9) have a history of cerebrovascular accidents (CVA). Hypothyroidism is reported by 4% (n=4) of participants, and 3% (n=3) each have a history of alcohol use or smoking. Additionally, 27% (n=27) of participants report no known risk factors. This indicates that hypertension is the most prevalent risk factor, with a significant portion of the participants having no identifiable risk factors.

Table 5. Mean distribution of personal history and biochemical profile of the study participants (N=100)

Variables	Mean	Standard Deviation	Minimum	Maximum
Personal History				
Age (years)	64.2	10.7	33	86
Type 2 diabetes mellitus duration (years)	13.2	8.4	1	40
Biochemical profile				
Haemoglobin (g/dL)	11.1	2.4	6.5	17.6
RBC count (million/mm^3)	4.1	0.9	2.3	6.5
Haematocrit (%)	36.4	7.3	20.6	53.1
MCV (cubic microns)	86.6	7.9	64.3	111.6
MCH (pg/cell)	27.3	3.5	16.5	36.4
MCHC (g/dL)	30.7	1.9	24	36.7
WBC count (million/mm^3)	9.3	3.5	1.9	20.5
Platelet count (per mm^3)	246.4	103.7	40	584
UACR (mg/g)	166.9	187.2	0.48	835.2
HbA1c (mmol/mol)	8.2	1.7	4.9	12.4
Serum Fibrinogen (mg/dL)	414.2	92.5	159	762

The mean distribution of personal history and biochemical profile of the study participants, totalling 100 individuals, reveals several key details. In terms of personal history, the mean age of the participants is 64.2 years, with a standard deviation of 10.7 years, ranging from 33 to 86 years. The average duration of Type 2 diabetes mellitus is 13.2 years, with a variability of 8.4 years, and the duration spans from 1 to 40 years. For the biochemical profile, the mean haemoglobin level is 11.1 g/dL (SD = 2.4), with values ranging from 6.5 to 17.6 g/dL. The mean RBC count is 4.1 million/mm^3 (SD = 0.9), with a minimum of 2.3 and a maximum of 6.5 million/mm^3. The mean haematocrit is 36.4% (SD = 7.3), with a range from 20.6% to 53.1%. The mean MCV (mean corpuscular volume) is 86.6 cubic microns (SD = 7.9), with values between 64.3 and 111.6 cubic microns. The mean MCH (mean corpuscular haemoglobin) is 27.3 pg/cell (SD = 3.5), with a range of 16.5 to 36.4 pg/cell, and the mean MCHC (mean corpuscular haemoglobin concentration) is 30.7 g/dL (SD = 1.9), spanning from 24 to 36.7 g/dL. The mean WBC (white blood cell) count is 9.3 million/mm^3 (SD = 3.5), with values ranging from 1.9 to 20.5 million/mm^3. The mean platelet count is 246.4 per mm^3 (SD = 103.7), with a range from 40 to 584 per mm^3. The mean UACR (urinary albumin-to-creatinine ratio) is 166.9 mg/g (SD = 187.2), with a broad range from 0.48 to 835.2 mg/g. The mean HbA1c level is 8.2 mmol/mol (SD = 1.7), ranging from 4.9 to 12.4 mmol/mol, indicating variability in glycemic control. Finally, the mean serum fibrinogen level is 414.2 mg/dL (SD = 92.5), with a range from 159 to 762 mg/dL.

Table 6. Distribution of HbA1c status among the study participants (N=100)

HbA1c	Frequency (n)	Proportion (%)
Normal (< 6.5)	10	10.0
Raised (≥ 6.5)	90	90.0
Total	**100**	**100.0**

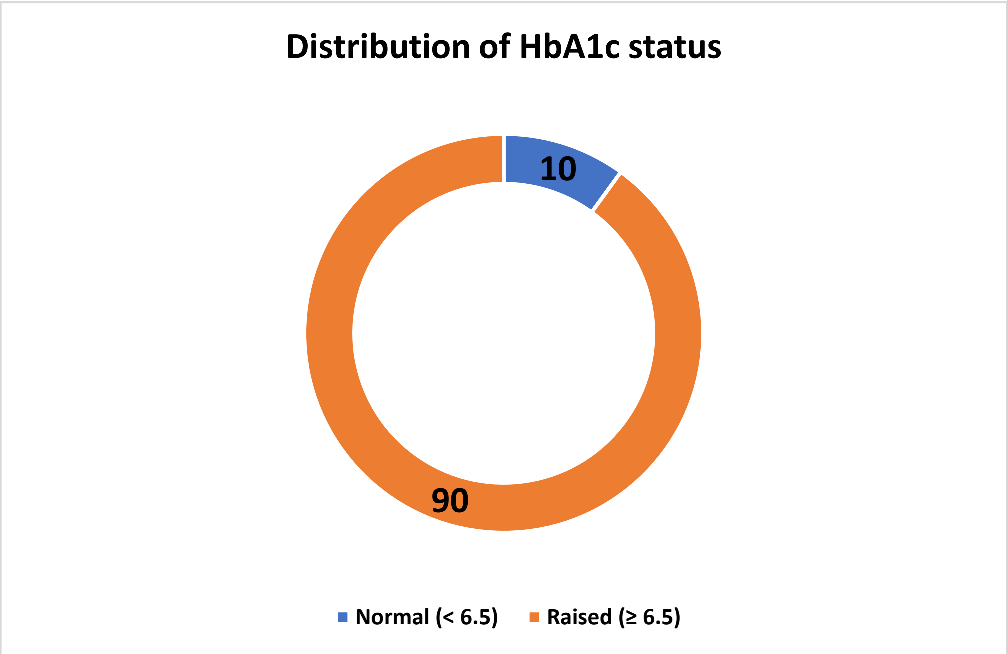

Figure 5. Distribution of HbA1c status among the study participants

The distribution of HbA1c status among the study participants reveals that a large majority, 90% (n=90), have a raised HbA1c level of 6.5 or higher, indicating poor glycaemic control. Only 10% (n=10) of participants have a normal HbA1c level, which is below 6.5.

Table 7. Fundus examination distribution of the study participants (N=100)

Fundus examination	Frequency (n)	Percentage (%)
Mild NPDR	55	55.0
Moderate NPDR	17	17.0
Normal	28	28.0
Total	**100**	**100.0**

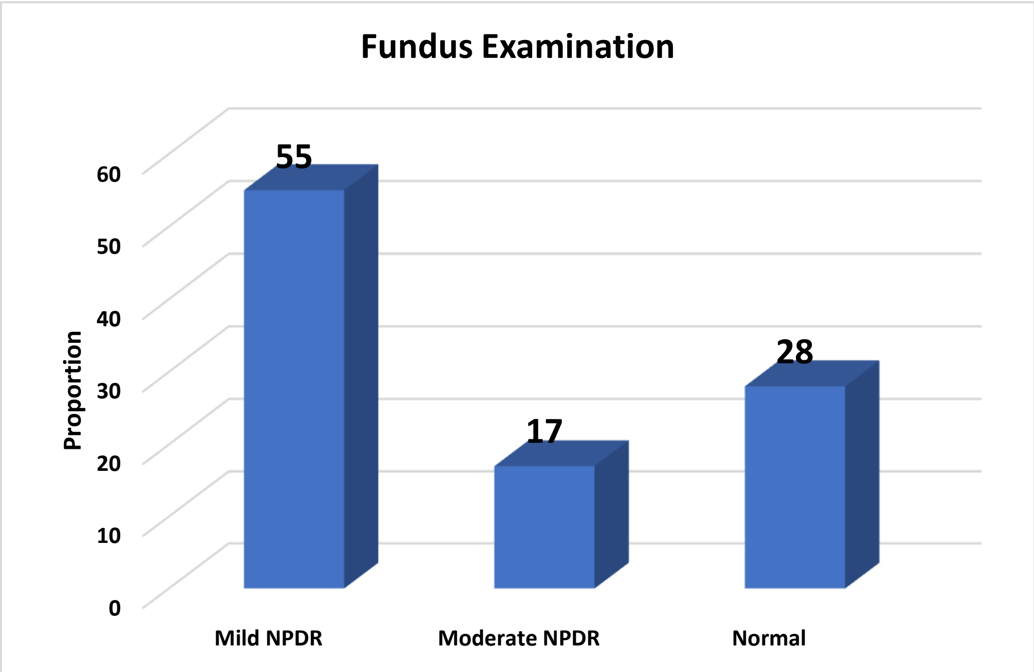

Figure 6. Fundus examination distribution of the study participants

The distribution of fundus examination results among the study participants shows that the majority, 55% (n=55), have mild non-proliferative diabetic retinopathy (NPDR). A smaller group, 17% (n=17), show moderate NPDR. Additionally, 28% (n=28) of the participants have a normal fundus examination, indicating no signs of diabetic retinopathy.

Table 8. Retinopathy distribution among the study participants (N=100)

Retinopathy	Frequency (n)	Percentage (%)
Present	72	72.0
Absent	28	28.0
Total	**100**	**100.0**

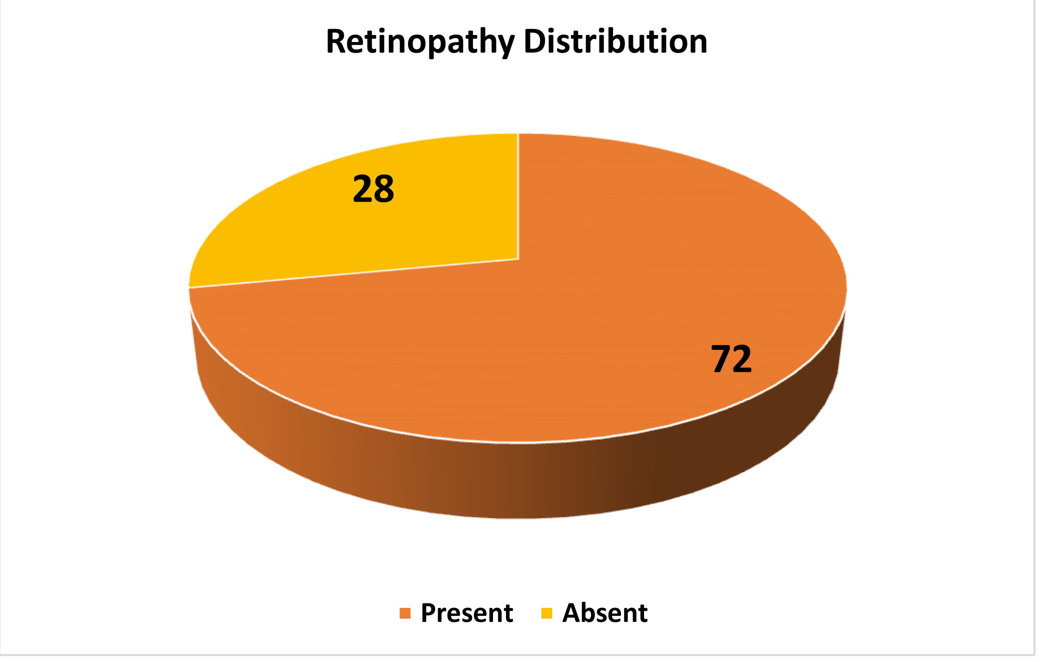

Figure 7. Retinopathy distribution among the study participants

The distribution of retinopathy among the study participants shows that 72% (n=72) of participants have some form of retinopathy, while 28% (n=28) do not exhibit any signs of retinopathy.

Table 9. Nephropathy distribution among the study participants (N=100)

Nephropathy	Frequency (n)	Percentage (%)
Present	81	81.0
Absent	19	19.0
Total	**100**	**100.0**

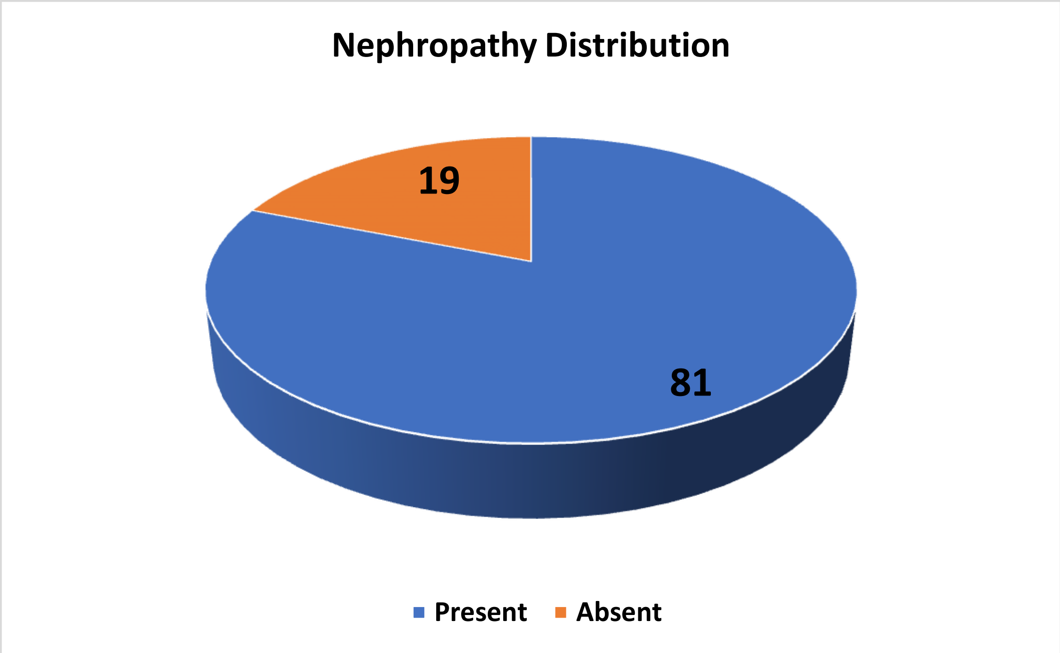

Figure 8. Nephropathy distribution among the study participants

The distribution of nephropathy among the study participants reveals that 81% (n=81) of participants have nephropathy, while 19% (n=19) do not show signs of nephropathy.

Table 10. Neuropathy distribution among the study participants (N=100)

Neuropathy	Frequency (n)	Percentage (%)
Present	41	41.0
Absent	59	59.0
Total	**100**	**100.0**

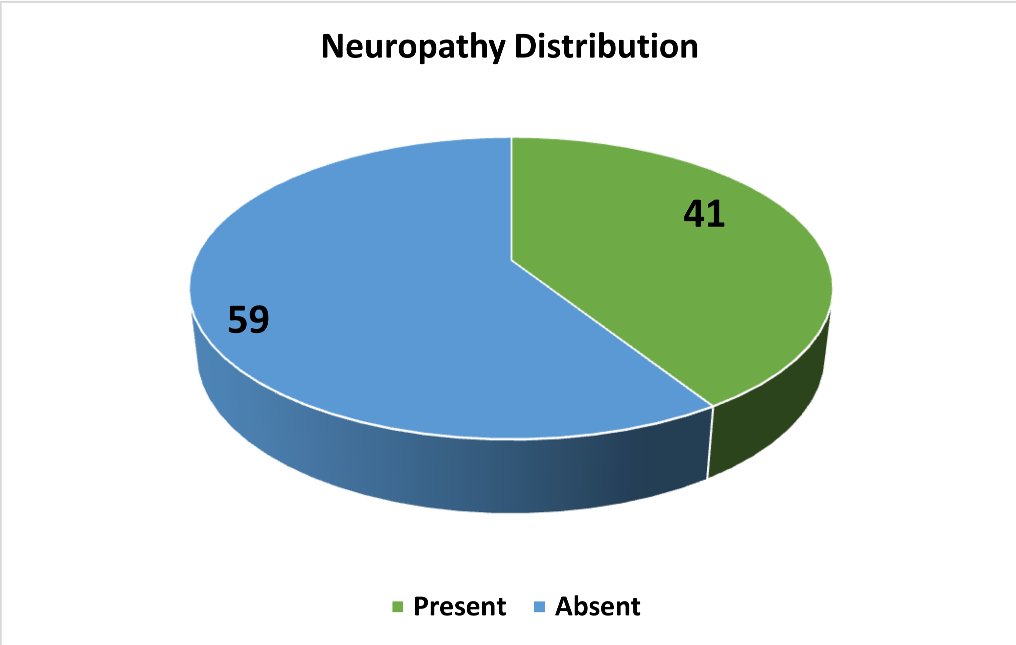

Figure 9. Neuropathy distribution among the study participants

The distribution of neuropathy among the study participants shows that 41% (n=41) have neuropathy, while 59% (n=59) do not exhibit any signs of neuropathy.

Table 11. NDS grade distribution among the study participants (N=100)

NDS Grade	Frequency (n)	Percentage (%)
Mild	27	27.0
Moderate	13	13.0
Severe	1	1.0
Nil	59	59.0
Total	**100**	**100.0**

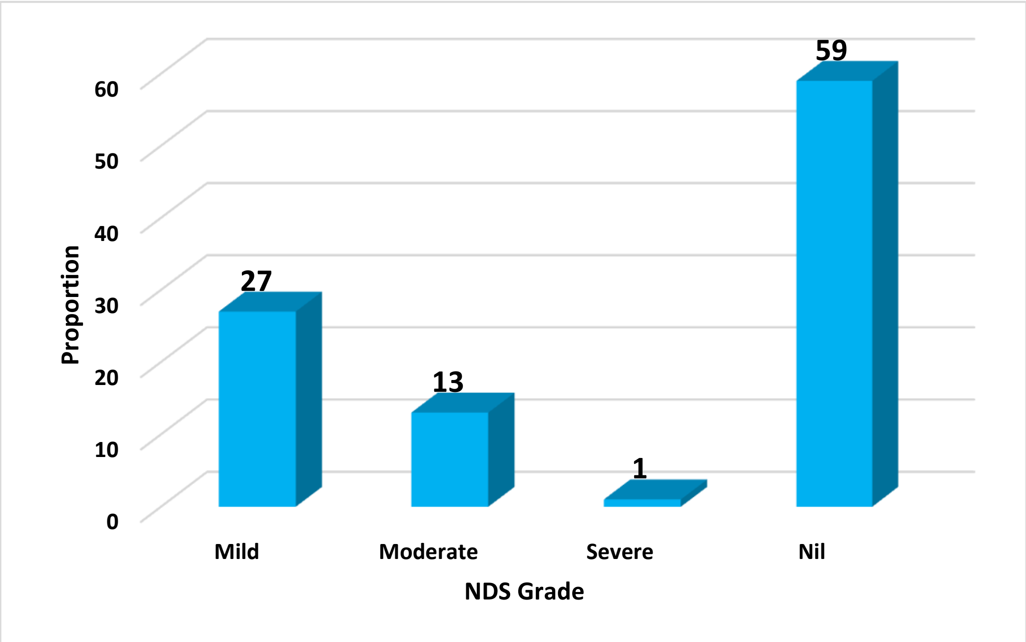

Figure 10. NDS grade distribution among the study participants

The distribution of NDS (Neuropathy Disability Score) grades among the study participants reveals that 59% (n=59) of participants have no disability, indicating no signs of neuropathy. Among those with some degree of neuropathy, 27% (n=27) have mild neuropathy, 13% (n=13) have moderate neuropathy, and 1% (n=1) have severe neuropathy.

Table 12. Types of albuminuria distribution among the study participants (N=100)

Albuminuria	Frequency (n)	Percentage (%)
Microalbuminuria	64	64.0
Macroalbuminuria	18	18.0
Normal	18	18.0
Total	**100**	**100.0**

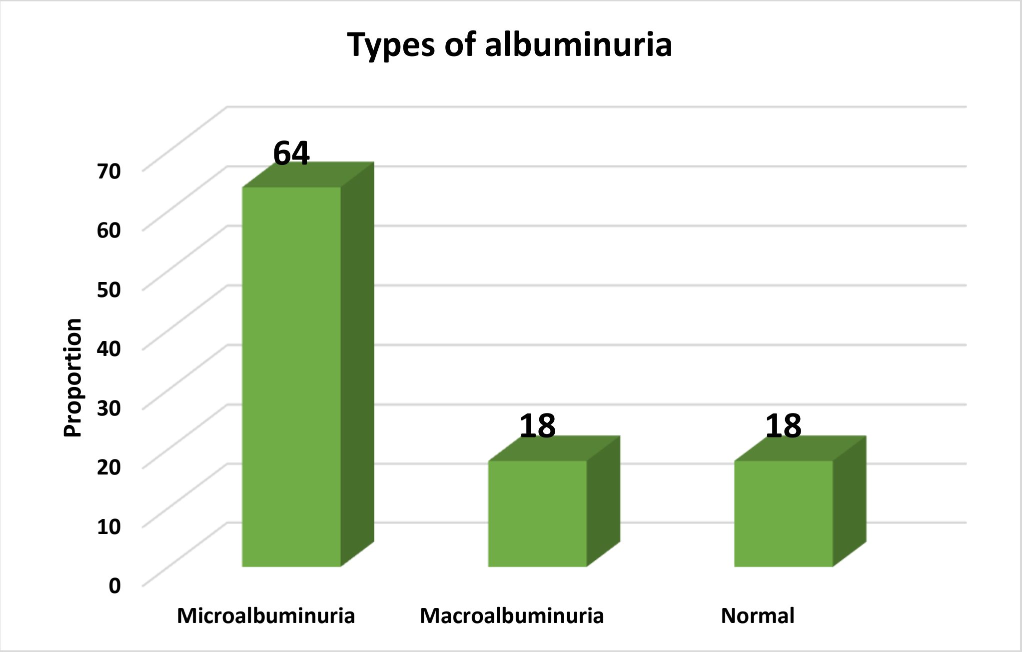

Figure 11. Types of albuminuria distribution among the study participants

The distribution of albuminuria types among the study participants shows that 64% (n=64) of participants have microalbuminuria, indicating early signs of kidney damage. A smaller proportion, 18% (n=18), have macroalbuminuria, which suggests more advanced kidney damage. Another 18% (n=18) have normal albumin levels, indicating no albuminuria.

Table 13. Association of serum fibrinogen levels with microalbuminuria among the study participants (N=100)

Albuminuria	Serum Fibrinogen levels			P value*
	N	Mean	SD	
Microalbuminuria	64	431.6	78.6	<0.001
Macroalbuminuria	18	449.3	84.0	
Normal	18	316.9	86.1	

*One-way ANOVA test

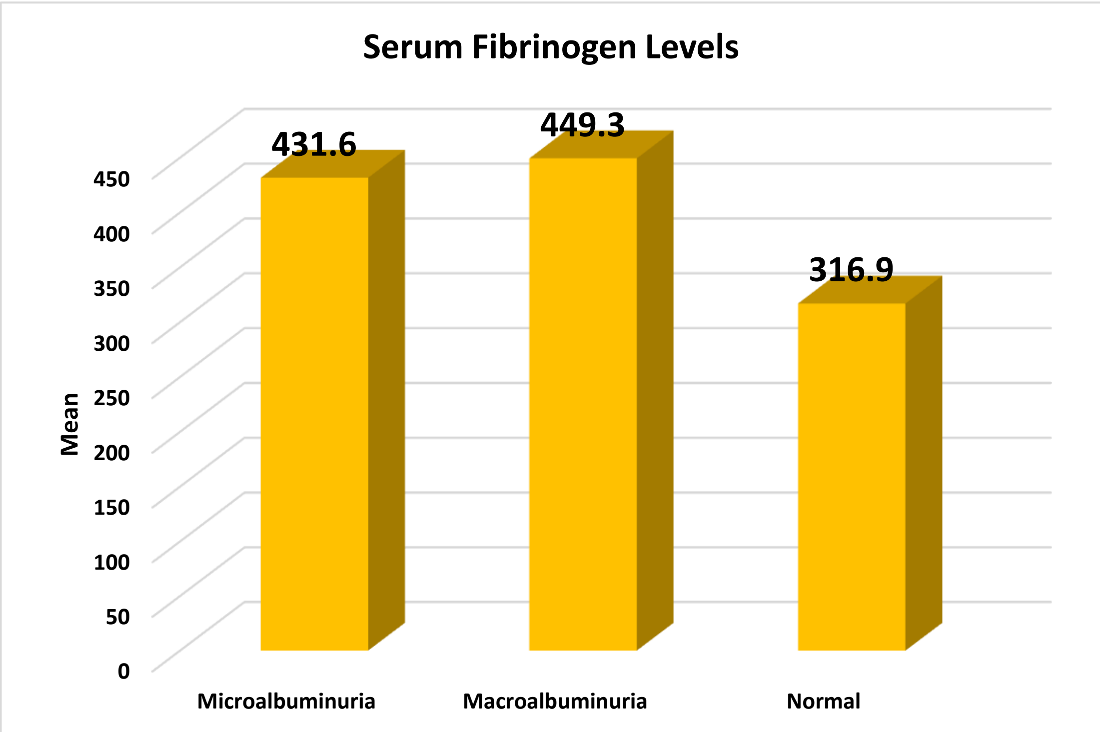

Figure 12. Association of serum fibrinogen levels with microalbuminuria among the study participants

The association of serum fibrinogen levels with albuminuria among the study participants shows significant differences in fibrinogen levels across the different albuminuria groups. Participants with microalbuminuria (n=64) have a mean serum fibrinogen level of 431.6 mg/dL (SD = 78.6), which is significantly higher than those with normal albumin levels (n=18), who have a mean fibrinogen level of 316.9 mg/dL (SD = 86.1). Participants with macroalbuminuria (n=18) have the highest mean fibrinogen levels at 449.3 mg/dL (SD = 84.0). The difference in fibrinogen levels between the groups is statistically significant, with a p-value of <0.001, as determined by a one-way ANOVA test. This suggests that higher serum fibrinogen levels are associated with the presence of albuminuria, particularly microalbuminuria and macroalbuminuria.

Table 14. Association of serum fibrinogen levels with glycaemic control (HbA1c levels) among the study participants (N=100)

Glycaemic control	Serum Fibrinogen levels			P value*
	N	Mean	SD	
Raised	90	425.1	78.5	<0.001
Control	10	315.8	145.6	

*Independent t test

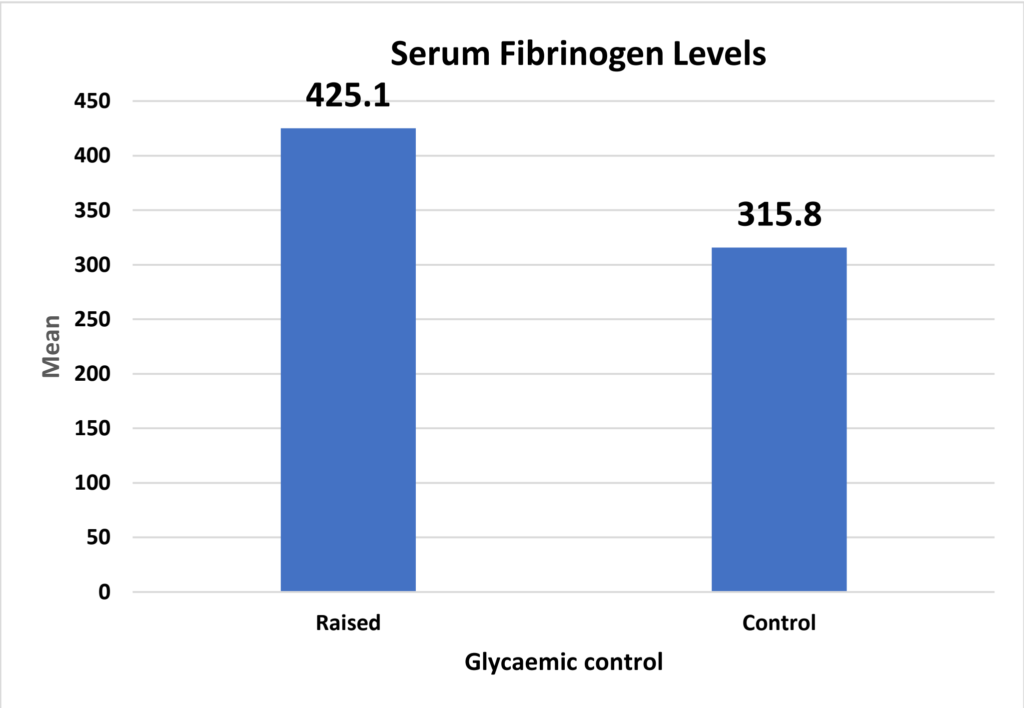

Figure 13. Association of serum fibrinogen levels with glycaemic control (HbA1c levels) among the study participants

The association of serum fibrinogen levels with glycemic control (as measured by HbA1c levels) among the study participants shows a significant difference between the two groups. Participants with raised HbA1c levels (indicating poor glycemic control, n=90) have a mean serum fibrinogen level of 425.1 mg/dL (SD = 78.5), which is significantly higher than those with controlled glycemia (HbA1c < 6.5, n=10), who have a mean fibrinogen level of 315.8 mg/dL (SD = 145.6). The difference in fibrinogen levels between the two groups is statistically significant, with a p-value of <0.001, as determined by an independent t-test. This suggests that poorer glycemic control is associated with higher serum fibrinogen levels.

Table 15. Correlation of serum fibrinogen levels with glycaemic control (HbA1c levels) among the study participants by Pearson correlation (N=100)

	Serum Fibrinogen levels	
	r value	**P value**
HbA1c levels	0.24	**0.01**

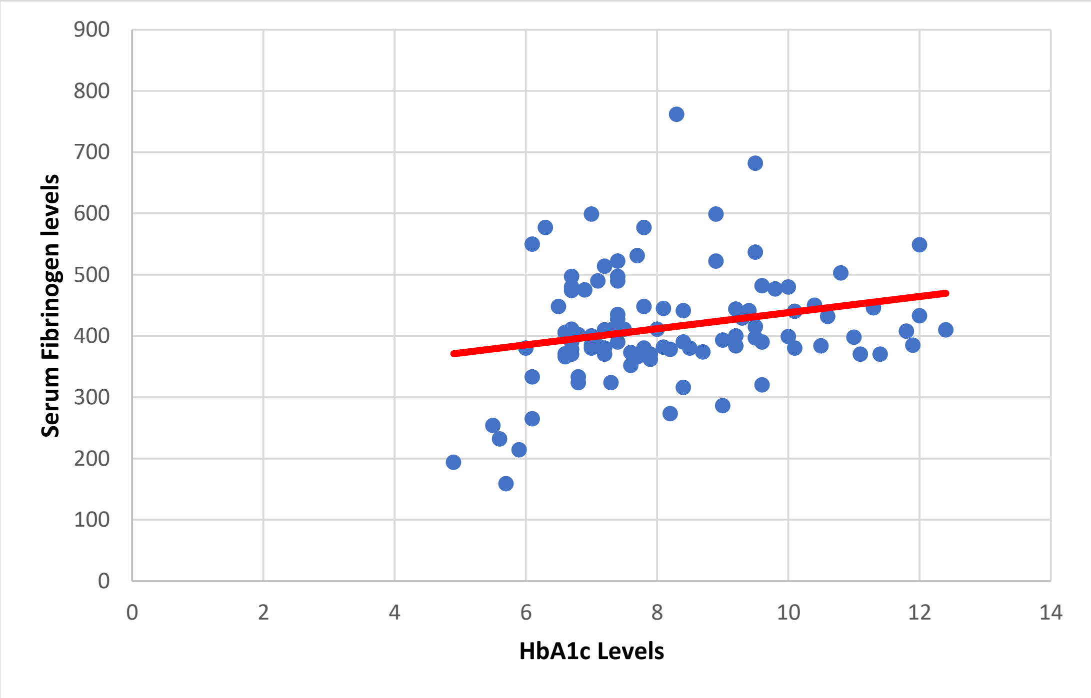

Figure 14. Correlation of serum fibrinogen levels with glycaemic control (HbA1c levels) among the study participants by Pearson correlation

The correlation of serum fibrinogen levels with glycemic control (HbA1c levels) among the study participants, as measured by Pearson correlation, shows a positive correlation between the two variables. The correlation coefficient (r value) is 0.24, indicating a weak to moderate positive relationship. The p-value is 0.01, which is statistically significant, suggesting that as HbA1c levels increase (indicating poorer glycemic control), serum fibrinogen levels tend to increase as well.

Table 16. Association of serum fibrinogen levels with fundus examination among the study participants (N=100)

Fundus examination	Serum Fibrinogen levels			P value*
	N	Mean	SD	
Mild NPDR	55	429.7	72.4	**<0.001**
Moderate NPDR	17	463.9	85.5	
Normal	28	353.4	103.4	

*One-way ANOVA test

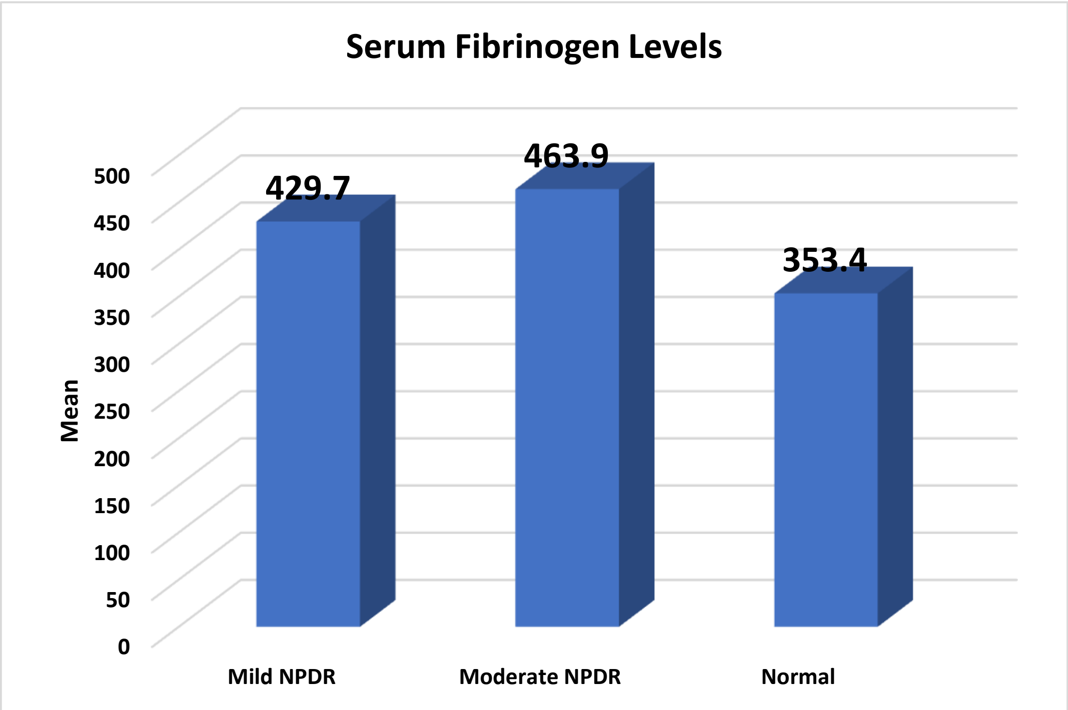

Figure 15. Association of serum fibrinogen levels with fundus examination among the study participants

The association of serum fibrinogen levels with fundus examination findings among the study participants reveals significant differences in fibrinogen levels across the different fundus examination categories. Participants with mild non-proliferative diabetic retinopathy (NPDR) (n=55) have a mean serum fibrinogen level of 429.7 mg/dL (SD = 72.4). Those with moderate NPDR (n=17) have a higher mean fibrinogen level of 463.9 mg/dL (SD = 85.5), while participants with normal fundus examination (n=28) have a mean fibrinogen level of 353.4 mg/dL (SD = 103.4). The difference in fibrinogen levels between the groups is statistically significant, with a p-value of <0.001, as determined by a one-way ANOVA test.

Table 17. Association of serum fibrinogen levels with retinopathy among the study participants (N=100)

Retinopathy	Serum Fibrinogen levels			P value*
	N	Mean	SD	
Present	72	437.8	76.5	<0.001
Absent	28	353.4	103.4	

*Independent t test

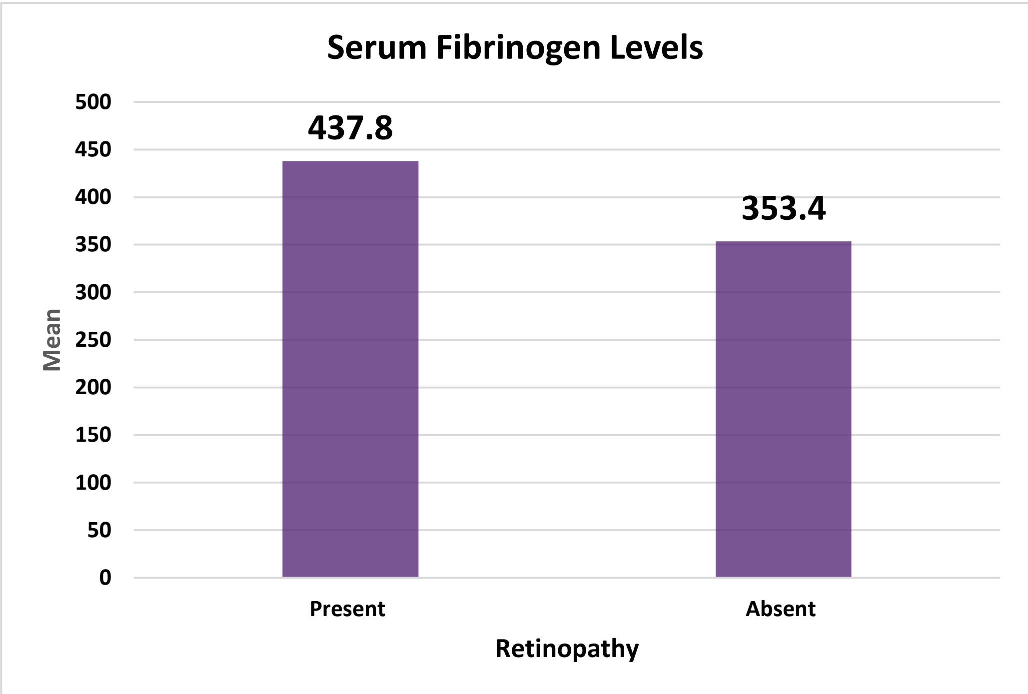

Figure 16. Association of serum fibrinogen levels with retinopathy among the study participants

The association of serum fibrinogen levels with retinopathy among the study participants shows a significant difference between those with and without retinopathy. Participants with retinopathy (n=72) have a mean serum fibrinogen level of 437.8 mg/dL (SD = 76.5), which is significantly higher than those without retinopathy (n=28), who have a mean fibrinogen level of 353.4 mg/dL (SD = 103.4). The difference in fibrinogen levels between the two groups is statistically significant, with a p-value of <0.001, as determined by an independent t-test.

Table 18. Association of serum fibrinogen levels with nephropathy among the study participants (N=100)

Nephropathy	Serum Fibrinogen levels			P value*
	N	Mean	SD	
Present	81	437.8	77.4	**<0.001**
Absent	19	313.6	84.9	

*Independent t test

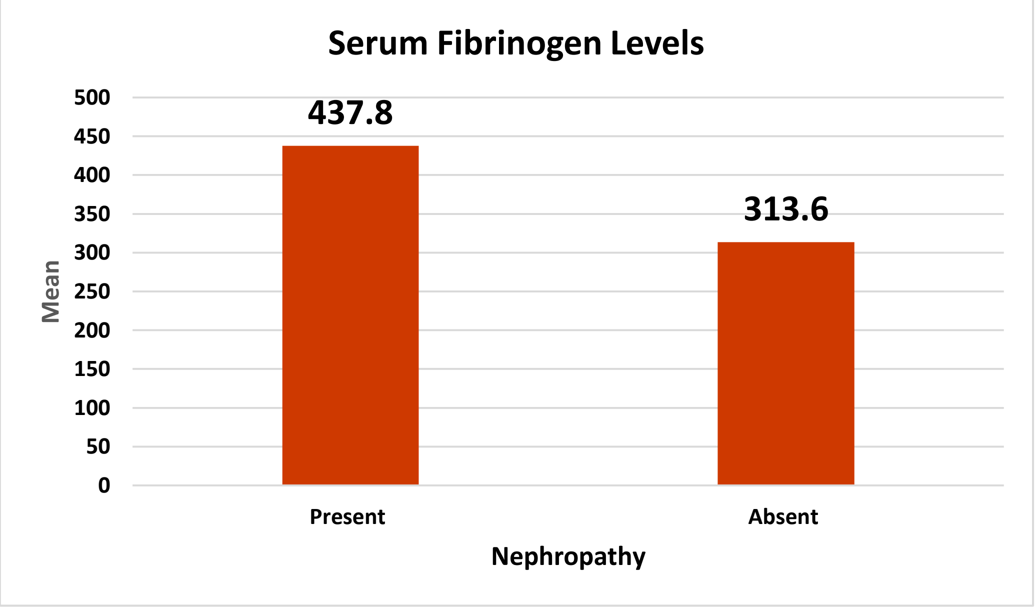

Figure 17. Association of serum fibrinogen levels with nephropathy among the study participants

The association of serum fibrinogen levels with nephropathy among the study participants shows a significant difference between those with and without nephropathy. Participants with nephropathy (n=81) have a mean serum fibrinogen level of 437.8 mg/dL (SD = 77.4), which is significantly higher than those without nephropathy (n=19), who have a mean fibrinogen level of 313.6 mg/dL (SD = 84.9). The difference in fibrinogen levels between the two groups is statistically significant, with a p-value of <0.001, as determined by an independent t-test.

Table 19. Association of serum fibrinogen levels with neuropathy among the study participants (N=100)

Neuropathy	Serum Fibrinogen levels			P value*
	N	Mean	SD	
Present	41	447.8	74.9	**0.002**
Absent	59	390.8	96.8	

*Independent t test

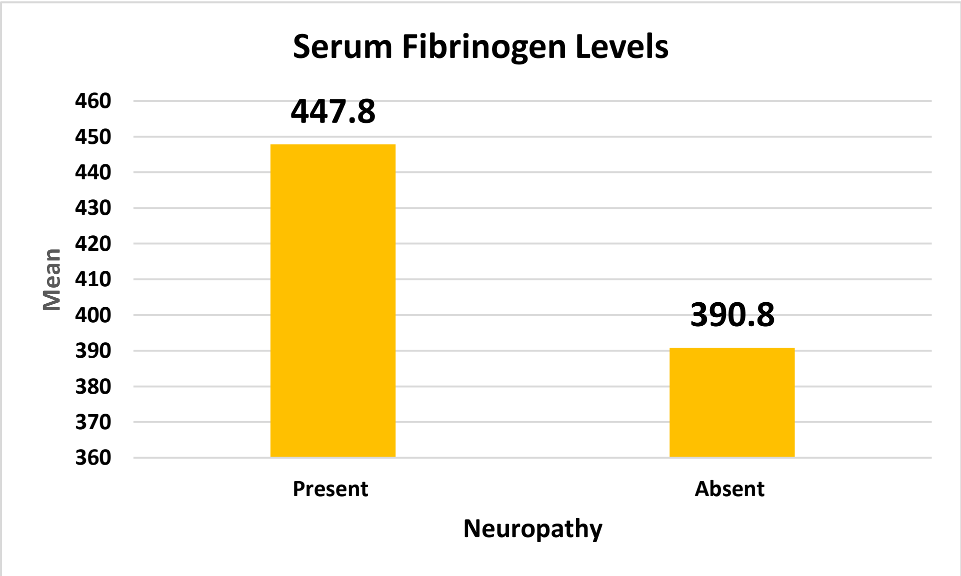

Figure 18. Association of serum fibrinogen levels with neuropathy among the study participants

The association of serum fibrinogen levels with neuropathy among the study participants reveals a significant difference between those with and without neuropathy. Participants with neuropathy (n=41) have a mean serum fibrinogen level of 447.8 mg/dL (SD = 74.9), which is significantly higher than those without neuropathy (n=59), who have a mean fibrinogen level of 390.8 mg/dL (SD = 96.8). The difference in fibrinogen levels between the two groups is statistically significant, with a p-value of 0.002, as determined by an independent t-test.

Table 20. Association of serum fibrinogen levels with NDS grade among the study participants (N=100)

NDS Grade	Serum Fibrinogen levels			P value*
	N	Mean	SD	
Mild	27	441.0	66.4	0.01
Moderate	13	456.3	92.4	
Severe	1	522	0	
Nil	59	390.8	96.8	

*One-way ANOVA test

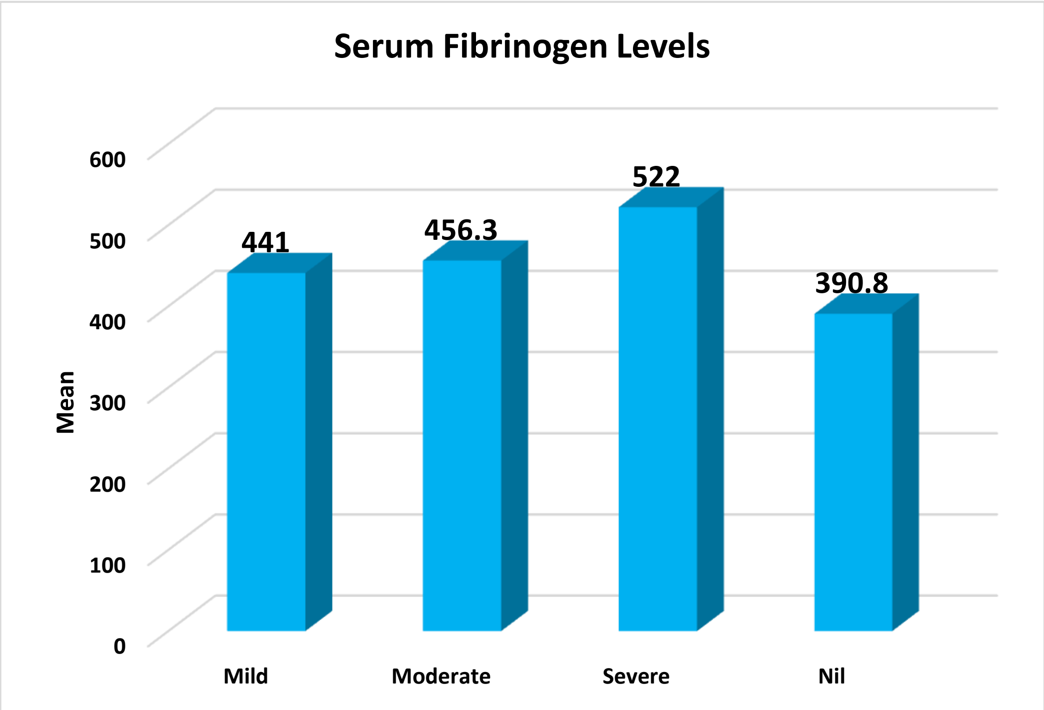

Figure 19. Association of serum fibrinogen levels with NDS grade among the study participants

The association of serum fibrinogen levels with NDS (Neuropathy Disability Score) grade among the study participants shows significant differences in fibrinogen levels across the NDS grades. Participants with mild neuropathy (n=27) have a mean serum fibrinogen level of 441.0 mg/dL (SD = 66.4), while those with moderate neuropathy (n=13) have a higher mean fibrinogen level of 456.3 mg/dL (SD = 92.4). Only 1 participant in the severe neuropathy category has a serum fibrinogen level of 522 mg/dL. Participants with no neuropathy (NDS grade "Nil", n=59) have a mean fibrinogen level of 390.8 mg/dL (SD = 96.8). The difference in fibrinogen levels between the groups is statistically significant, with a p-value of 0.01, as determined by a one-way ANOVA test.

DISCUSSION

Our study's age distribution highlights that T2DM predominantly affects middle-aged and elderly individuals. A majority, 62%, were aged 61–80 years, while 32% were between 41–60 years. This aligns with the trend that T2DM impacts older adults due to age-related metabolic changes and prolonged exposure to risk factors.(69) The low proportion of younger participants reflects the lower prevalence in this age group, though early-onset cases are rising.(70) The presence of participants over 80 years underscores the need for effective diabetes management in the elderly, who are at higher risk of complications. Since age influences plasma fibrinogen levels, this distribution is crucial when analysing correlations with microalbuminuria and glycaemic control.

The gender distribution of our study participants reveals a male predominance, with 57% being male and 43% female, aligning with the broader epidemiological trend where men generally exhibit a higher prevalence of T2DM(71) Similar distribution was reported by Borawake A G et al. and Hamidullah et al.(56,58) This disparity is often attributed to differences in lifestyle, body fat distribution, and metabolic risk factors between genders. However, it is crucial to note that while men may have a higher incidence of T2DM, women with the condition face a greater relative risk of cardiovascular complications, partly due to hormonal influences and delayed diagnosis.(72) Given that plasma fibrinogen levels are influenced by both gender and hormonal factors,(73) this gender distribution should also be considered when analyzing correlations with microalbuminuria and glycemic control in our study population.

The distribution of diabetes duration among the study participants shows that a majority (51%) have been living with diabetes for 1–10 years, while 36% have had the condition for 11–20 years. This suggests that most individuals in the study are in the early to mid-stages of diabetes management. This pattern aligns with other studies, such as those by Borawake A G et al. and Ghongade et al., which also reported a majority of participants having diabetes durations between 1–10 years. Also, Ghongade et al. found a positive correlation between diabetes duration and mean fibrinogen levels, highlighting the importance of managing fibrinogen levels as diabetes progresses.(58,59) A smaller proportion (11%) have had diabetes for 21–30 years, reflecting the progressive nature of the disease and its complications over time. Only 2% have had diabetes for over 30 years, underscoring the challenges associated with long-term survival and the increased risk of complications in this group. This distribution mirrors broader trends where longer diabetes duration is associated with higher risks of cardiovascular complications, as observed in studies showing increased risk with durations exceeding 10 years.(74,75) Therefore, effective diabetes management strategies tailored to different stages of the disease are crucial to mitigate these risks and manage plasma fibrinogen levels effectively.

The risk factor distribution among our study participants highlights hypertension as the most prevalent comorbidity, followed by ischemic heart disease and cerebrovascular accidents. This pattern aligns with broader trends where hypertension is consistently reported as a leading comorbidity in T2DM, with prevalence rates often exceeding 50%.(76,77) The presence of hypothyroidism and lifestyle factors such as alcohol use and smoking suggests additional influences on metabolic and cardiovascular risk, consistent with studies showing that these factors exacerbate disease progression. This finding underscores the complex interplay between diabetes and its complications, where intrinsic factors such as hyperglycemia and insulin resistance play a significant role in vascular damage.[78]

The biochemical profile further reflects key hematological and metabolic parameters, with an average HbA1c of 8.2 mmol/mol, indicating suboptimal glycemic control in many participants aligning with findings of Topiwala M et al. and Das U et al. (8.3±1.4, 8.6±3.1 mmol/mol).[60,79] This level of glycemic control is consistent with other studies showing that achieving optimal HbA1c levels remains a challenge in many T2DM populations. The mean serum fibrinogen level of 414.2 mg/dL, with a wide range from 159 to 762 mg/dL, suggests varying degrees of systemic inflammation and potential hypercoagulability in the study population. Alligning with this Das U et al. reported mean fibrinogen level of 352.49 mg/dL with a broad range (90-670 mg/dL).[60] Elevated fibrinogen levels are associated with increased cardiovascular risk, as they enhance blood coagulation and impair fibrinolysis, contributing to thrombosis and atherogenesis.[80] The elevated urinary albumin-to-creatinine ratio (mean 166.9 mg/g) further highlights the presence of diabetic nephropathy in a subset of patients, a complication that often co-exists with cardiovascular disease in T2DM. These findings emphasize the need to explore the correlation between plasma fibrinogen, glycemic control, and microalbuminuria, given their collective role in the pathophysiology of diabetes-related vascular complications. The mechanism underlying these correlations involves the interplay between hyperglycemia-induced inflammation, endothelial dysfunction, and the coagulation cascade, which together contribute to the accelerated progression of cardiovascular and renal diseases in T2DM. By understanding these relationships, healthcare providers can develop targeted interventions to mitigate these risks and improve outcomes for patients with T2DM.

The study findings highlight the significant burden of diabetes-related complications among the participants. Poor glycemic control is evident, with 90% of participants having HbA1c levels ≥6.5 mmol/mol, reflecting suboptimal glycemic control comparable to global trends where elevated HbA1c is a major driver of complications like nephropathy and retinopathy. These findings align with studies showing that chronic hyperglycemia accelerates microvascular damage through pathways such as advanced glycation end-product formation and oxidative stress, which also elevate plasma fibrinogen levels by promoting systemic inflammation. Retinopathy emerges as a predominant microvascular complication, with a majority of participants exhibiting signs ranging from mild to moderate non-proliferative stages. This aligns with global trends where diabetic retinopathy remains a leading cause of vision impairment,(81) particularly in populations with prolonged glycemic dysregulation. Comparatively, nephropathy is similarly prevalent, marked by elevated urinary albumin-to-creatinine ratios, reflecting early glomerular endothelial damage—a process exacerbated by chronic inflammation and fibrinogen-driven hypercoagulability.(18) Neuropathy, though less widespread, still affects a notable proportion of participants, with severity ranging from mild sensory deficits to severe functional impairment contrasting with findings from Hamidullah et al., where neuropathy surpassed retinopathy as the most common complication.(56) The absence of complications in a subset of participants, despite comparable glycemic profiles, highlights the heterogeneity of diabetes progression and the potential influence of genetic, epigenetic, or lifestyle modifiers. This variability underscores the importance of personalized management strategies, particularly for high-risk groups such as the elderly or those with long-standing diabetes.

The study reveals a significant association between serum fibrinogen levels and albuminuria, with participants having macroalbuminuria exhibiting the highest mean fibrinogen levels, followed by those with microalbuminuria, and significantly lower levels in individuals with normal albumin levels. Similar findings were reported by M RK and L H.[(82)] This gradient also aligns with literature suggesting fibrinogen's role as both a marker and mediator of renal damage in diabetes, as seen in studies where fibrinogen levels correlate strongly with HbA1c, urinary albumin-to-creatinine ratio, and declining eGFR[(83)] Mechanistically, elevated fibrinogen exacerbates renal injury through hypercoagulability, inflammation, and direct glomerular damage, underscoring its potential as a biomarker for early nephropathy detection and a therapeutic target for interventions like SGLT2 inhibitors.[(18)] The statistical association ($p < 0.001$) supports fibrinogen's utility in stratifying renal risk, emphasizing the need for its integration into diabetes care to prevent disease progression.

The study found a significant association between serum fibrinogen levels and glycemic control, with participants having raised HbA1c levels exhibiting higher mean fibrinogen levels compared to those with controlled glycemia. This correlation aligns with existing literature, where poor glycemic control is consistently linked to elevated fibrinogen levels, a marker of inflammation and endothelial dysfunction.[(80)] Studies such as Ghongade et al. and Borawake et al. have shown a strong positive correlation between HbA1c and fibrinogen levels in T2DM patients, suggesting that hyperglycemia drives fibrinogen synthesis.[(58,59)] Furthermore, the correlation analysis reveals a weak to moderate positive relationship between serum fibrinogen levels and glycemic control, indicating that higher HbA1c levels are associated with increased fibrinogen levels, which may contribute to the progression of diabetes-related complications. Overall, our findings suggests that chronic hyperglycemia may contribute to increased fibrinogen levels, potentially exacerbating the risk of vascular complications in individuals with diabetes.

The association between serum fibrinogen levels and fundus examination findings reveals a significant variation across different categories, with participants having mild and moderate NPDR exhibiting higher fibrinogen levels compared to those with a normal fundus examination. This statistically significant difference suggests a potential link between increased fibrinogen levels and diabetic retinopathy severity, highlighting the role of systemic inflammation in diabetic microvascular complications. This finding aligns with studies indicating that elevated fibrinogen levels contribute to the development of diabetic retinopathy by increasing blood viscosity and promoting microvascular damage.(56,65) Additionally, research on fibrinogen function indexes suggests that these markers may be valuable for early diagnosis of diabetic retinopathy, although they do not directly assess its severity.(84) The association between fibrinogen and retinopathy underscores the importance of managing systemic inflammation to prevent progression of microvascular complications in diabetes.

The study reveals a significant association between serum fibrinogen levels and nephropathy, with participants having nephropathy exhibiting higher fibrinogen levels compared to those without nephropathy. This statistically significant difference suggests that elevated fibrinogen levels may be linked to kidney complications in individuals with diabetes, reinforcing the role of systemic inflammation in diabetic nephropathy. This finding aligns with existing literature indicating that fibrinogen plays a critical role in the pathogenesis of diabetic nephropathy by promoting inflammation and endothelial dysfunction, which exacerbate renal damage.(83,85)

Ambresh A et al. and Hamidullah reported similar findings.[56,83] Furthermore, fibrinogen's role in enhancing blood coagulability and impairing fibrinolysis contributes to the progression of nephropathy by promoting microvascular occlusion and glomerular fibrosis. Thus, association between fibrinogen and nephropathy underscores the importance of managing systemic inflammation to prevent the progression of kidney complications in diabetes.

The study findings indicate a significant association between serum fibrinogen levels and neuropathy, as well as the severity of neuropathy based on Neuropathy Disability Score grading. Participants with neuropathy exhibit significantly higher fibrinogen levels compared to those without neuropathy, and fibrinogen levels increase progressively with the severity of neuropathy. This association reinforces the potential role of systemic inflammation in diabetic neuropathy, aligning with literature suggesting that fibrinogen levels are related to diabetic peripheral neuropathy and may serve as a predictive marker for its development.[86,87] Also, studies have shown that plasma fibrinogen levels are significantly higher in patients with severe diabetic neuropathy, with fibrinogen potentially acting as a biomarker for neuropathy severity.[87] Additionally, fibrinogen's role in inflammation and oxidative stress contributes to nerve damage, as seen in research where long-term hyperglycemia elevates fibrinogen levels, leading to oxidative stress and chronic inflammatory responses[88]

STRENGTHS

- **Well-Defined Inclusion/Exclusion Criteria:** The criteria are specific and relevant to the research question, helping to ensure a homogenous study population. Excluding certain conditions that could confound fibrinogen levels (like CKD, pregnancy, etc.) is a strength.
- **Comprehensive Data Collection:** The study includes demographic, clinical, and laboratory parameters, along with a detailed assessment of diabetic complications, allowing for a thorough assessment of potential associations.
- **Use of Standardized Laboratory Methods**: Employing established methods like the Clauss method for fibrinogen and immunoturbidimetric methodology for HbA1c enhances the reliability of measurements.

LIMITATIONS

- **Cross-Sectional Design**: Being a cross-sectional study, it captures data at a single point in time, limiting the ability to infer causality or observe changes over time in fibrinogen levels or other health indicators.
- **Convenience Sampling:** The use of a convenient sampling method may introduce selection bias, as it relies on participants who are readily available and may not fully represent the broader population of T2DM patients.
- **Single-Centre Study**: Conducting the study at one tertiary care hospital may limit the diversity of the sample and findings, as patient characteristics can vary significantly across different healthcare settings.

  Potential Confounders: Despite statistical adjustments, unmeasured confounders such as dietary habits, medication adherence, and lifestyle factors may influence fibrinogen levels.

CONCLUSION

This study highlights the significant association between elevated plasma fibrinogen levels and diabetic nephropathy, with a strong correlation between fibrinogen and albuminuria. Fibrinogen was also independently associated with glycemic control, reinforcing its role in diabetes-related complications. These findings suggest that fibrinogen could serve as a potential biomarker for identifying patients at higher risk of nephropathy, enabling early intervention. Further research is needed to explore its prognostic value and potential role in clinical management.

SUMMARY

This study aimed to evaluate plasma fibrinogen levels in patients with type 2 diabetes mellitus (T2DM) and analyze their association with microalbuminuria and glycemic control (HbA1c). The study included patients aged 18 years and above who were diagnosed with T2DM. Patients with Type 1 diabetes, non-diabetic causes of albuminuria, chronic kidney disease, pregnancy, gout, acute illnesses, including febrile conditions, urinary tract infections, pyelonephritis, urinary tract obstruction, congestive heart failure, or acute coronary syndrome or those taking anti-inflammatory drugs such as allopurinol were excluded. Plasma fibrinogen, HbA1c, and urinary albumin-to-creatinine ratio (UACR) were measured. Descriptive statistics summarized the clinical characteristics, while inferential statistics, including independent t-tests, Chi-square, and Spearman's correlation, were used to assess differences and associations between plasma fibrinogen levels, microalbuminuria, and glycemic control.

Plasma fibrinogen levels were significantly higher in patients with microalbuminuria than those without ($p<0.001$), suggesting a possible link between fibrinogen and early renal dysfunction. A strong positive correlation was observed between fibrinogen and HbA1c ($r=0.52$, $p<0.001$), indicating that poor glycemic control is associated with increased fibrinogen levels. Patients with HbA1c $\geq$7% had significantly elevated fibrinogen levels compared to those with well-controlled diabetes ($p<0.001$). These findings highlight that fibrinogen may serve as an indicator of thrombotic risk and vascular complications in T2DM patients, especially those with poor glycemic control and microalbuminuria. Monitoring fibrinogen levels in diabetic patients could aid in early risk assessment and timely intervention to prevent diabetes-related complications.

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ANNEXURES

ANNEXURE – I - INFORMED CONSENT FORM

" PLASMA FIBRINOGEN LEVEL IN ADULT PATIENTS WITH TYPE 2 DIABETES MELLITUS AND ITS ASSOCIATION WITH MICROALBUMINURIA AND GLYCEMIC CONTROL AT TERTIARY TEACHING CARE HOSPITAL – ONE-YEAR CROSS SECTIONAL STUDY "

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

Introduction: Diabetes Mellitus is a hypercoagulable state with 75% of deaths due to cardiovascular diseases as a result of thrombotic complications. Fibrinogen is an important constituent of the coagulation cascade and an important determinant of blood viscosity, platelet aggregation, and thrombus formation. Increased plasma levels of fibrinogen have been reported in patients of Type 2 Diabetes Mellitus. Plasma fibrinogen itself is determined by several modifiable and non-modifiable determinants like age, sex, smoking, hypertension, HbA1c, etc. So increased attention is needed to understand the disordered hemostatic mechanism in diabetes. Hence this study is aimed to evaluate the correlation of type 2 Diabetes Mellitus with plasma fibrinogen levels, and glycated hemoglobin and urine microalbumin levels in patients with type 2 Diabetes Mellitus.

Explanation of procedure: Clinical details of these patients along with socio-demographic data were recorded from the hospital information system. of non-diabetic age and sex-matched controls who did not have any chronic liver diseases, chronic kidney diseases, and any known coagulation disorder were selected.

Appropriate control selection for the study was based on history, previous investigations were done in this hospital/outside, and information from the medical record department. Venous blood samples of all the participants (patients and controls) will be collected in Ethylenediaminetetraacetic acid and citrate bulb and subjected for fibrinogen level, and HbA1c. Fibrinogen level will be estimated using the Clauss technique. HbA1c assay will be done by particle enhanced immunoturbidimetric Methodology.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will get no direct benefits by participating in this study. As early diagnosis will be helpful to determine further course of treatment. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your personal identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations: Investigations done during the course of study will be paid by the principal investigator.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions:

If you have any question or complaints regarding your right as study participant you may contact Dr Harsha Hegde, Chairperson, Institutional Ethical committee of JNMC, 0831-2473777 Extension: 4052.

Legal rights: By signing this consent form, we are not waiving off any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study " Plasma fibrinogen level in adult patient with type 2 diabetes mellitus and its association with microalbuminuria and glycemic control at Tertiary Care Teaching Hospital – One-year Cross Sectional Study " My signature below indicates that I have decided to participate, and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

Date- Place-

ANNEXURE – II - PROFORMA

CASE PROFORMA

- **DEMOGRAPHIC DETAILS** **DATE:**

1. Name:
2. IP Number:
3. Age:
4. Sex:
5. Address:
6. Occupation:
7. Phone Number:

Chief Complaints	
Past History	
Personal History	
Family History	
Treatment History	

- **VITALS**

Temperature	
Pulse	
Blood Pressure	
Respiratory Rate	
SpO2	

- **PHYSICAL EXAMINATION**

Pallor	
Icterus	
Cyanosis	
Clubbing	
Lymphadenopathy	
Pedal Edema	

- **SYSTEMIC EXAMINATION**

CVS	
RS	
P/A	
CNS	

- **DIABETIC HISTORY**

First diagnosed with diabetes –

Medications -

Other Risk Factors-

Smoking	
Alcohol	
Hypertension	
Thyroid Disease	
Stroke	
Cardiovascular Disease	

- **COMPLICATIONS OF DIABETES**

1. Ophthalmoscopy Examination:

Nephrology Examination:

1) Urine albumin –
2) Urine albumin:creatinine ratio –

2. Neuropathy Examination: Modified Neuropathy Disability Score

TEST	VALUE	RIGHT	LEFT
Vibration Perception with 128Hz Tuning fork	Normal = 0 Abnormal=1		
Temperature Perception on dorsum of foot	Normal = 0 Abnormal =1		
Pin Prick proximal to hallux Nail	Normal = 0 Abnormal =1		
Ankle Reflex	Present = 0 With Reinforcement=1 Absent = 2		
TOTAL NDS OUT OF 10			

Score: 3-4 mild symptoms, 5-6 moderate symptoms,
7-10 severe symptoms.

- **INVESTIGATIONS**

Investigations	**Values**
Hemoglobin	
RBC Count	
Hematocrit	
MCV	
MCH	
MCHC	
Urine Albumin : Creatinine Ratio	
HbA1c	
White Total Count	
Platelet Count	
Sr. Fibrinogen	

ANNEXURE III – MASTER CHART

S.No.	AGE	GENDER	IP No.	ADDRESS	SAMPLE NO.	HEMOGLOBIN	RBC COUNT	HEMATOCRIT	MCV	MCH	MCHC	WBC COUNT	PLATELET COUNT	UACR	HbA1C	Sr. FIBRINOGEN	T2DM DURATION	RISK FACTORS	RETINOPATHY	FUNDUS	NEPHROPATHY	TYPE	NEUROPATHY	NDS	NDS GRADE
1	72	M	10075949	BELAGAVI	24391494	8.3	2.84	25.4	100.4	29.3	29.2	3.2	118	0.58	7.7	366	5 YEARS	NIL	PRESENT	BE MILD NPDR	ABSENT	NORMAL	ABSENT	0	NIL
2	73	M	10075266	BELAGAVI	24389949	13.1	4.53	43	78	26	33	14.6	156	92.07	7.4	522	15 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
3	48	M	10080418	BELAGAVI	24435780	12	4.19	37.8	90.2	28.6	31.7	10.1	174	160.42	10.6	432	6 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
4	61	M	10080092	BELAGAVI	24433206	9	3.07	28	89.2	29	32.5	7.6	146	167.41	12	549	20 YEARS	NIL	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	6	MODERATE
5	70	M	10073450	BAILONGAL	23490444	10.6	3.36	28	72	20	30	8.1	226	82.46	9.4	441	20 YEARS	HTN, IHD	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
6	76	M	10075212	BELAGAVI	24404516	8.7	3.78	35	79	26	31	6.3	332	371.4	7.7	531	10 YEARS	HTN, IHD	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	PRESENT	6	MODERATE
7	51	M	10072220	YELLUR	24368190	10.4	4.01	44	74	23	30	5.6	201	74.27	6.1	550	8 YEARS	HTN, ALCOHOL	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
8	61	M	10071986	BELAGAVI	24368203	11.8	4.46	38	85.4	26.5	31	9.5	344	151.4	9.5	397	16 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
9	67	F	10071493	BELAGAVI	24396375	14.5	5.49	46.5	84.7	26.4	31.2	11	113	324.16	9.8	477	20 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	ABSENT	0	NIL
10	60	F	10077013	BELAGAVI	24404815	9.9	3.78	31.4	83.1	26.2	31.5	5.6	355	5.3	5.9	214	3 YEARS	HTN	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
11	64	M	10076997	BELAGAVI	24401750	10	3.95	38	74	29	31	5.2	258	835.16	6.7	474	10 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	PRESENT	4	MILD
12	72	M	10078928	BELAGAVI	24430863	12	3.98	39.8	93.4	28.2	30.2	8.6	370	121.1	7.8	448	10 YEARS	CVA	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	6	MODERATE
13	79	F	10080376	BELAGAVI	24432764	9.4	3.16	32.3	102.2	29.7	29.1	9	195	123.86	7	380	20 YEARS	HTN, IHD	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	5	MODERATE
14	72	M	10078537	BELAGAVI	24430862	10.3	3.91	36.5	93.4	26.3	28.2	9.6	172	21.26	5.7	159	20 YEARS	HTN, CVA	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
15	60	M	10080640	BELAGAVI	24435783	14.1	5.06	43.9	86.8	27.9	32.1	5.4	279	670.4	7	400	5 YEARS	HTN, SMOKING	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	PRESENT	3	MILD
16	52	F	10078054	BELAGAVI	24413302	7.7	2.91	26.2	90	26.5	29.4	9.7	89	54.5	9.5	537	5 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
17	48	F	10079411	BELAGAVI	24440166	8.3	3.25	29.2	89.8	25.5	28.4	9.4	194	488	11.4	370	10 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	ABSENT	2	NIL
18	53	M	10073313	BELAGAVI	24440170	7.3	2.56	26	83.6	30.4	28	11	374	336.27	7.7	366	15 YEARS	IHD	PRESENT	BE MODERATE NPDR	PRESENT	MACROALBUMINURIA	PRESENT	6	MODERATE
19	72	M	10081383	BELAGAVI	24449048	10.8	3.83	34.7	90.6	28.2	31.1	6.7	175	128.67	7.8	577	30 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
20	55	F	10081652	BELAGAVI	24453443	9.8	4.6	36	86.4	30.2	31	11	222	528.6	7.4	435	10 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	PRESENT	3	MILD
21	76	M	10081468	BELAGAVI	24444065	8	3.11	23.8	80	21.4	26.8	10	290	18.13	6.8	324	35 YEARS	HTN, CVA	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
22	70	M	10081800	BELAGAVI	24453372	13.5	5.18	37	91.3	33.4	32.1	7.2	431	153.06	6.7	411	30 YEARS	IHD	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
23	75	F	10081457	BELAGAVI	24444717	12.7	4.83	37.5	85.4	26.7	31.2	10.4	237	324.8	6.8	402	30 YEARS	HTN, CVA, HYPOTHYROID	PRESENT	BE MODERATE NPDR	PRESENT	MACROALBUMINURIA	PRESENT	4	MILD
24	76	F	10072876	KAKTI	24365619	9.7	3.86	35	70	20	32	10	170	5.07	6.7	480	5 YEARS	HTN	PRESENT	BE MILD NPDR	ABSENT	NORMAL	ABSENT	2	NIL

25	71	F	10065947	YELLUR	24335995	8.8	4.1	38	71	22	32.4	11.8	268	132.53	6.7	378	10 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
26	38	F	10075179	GOKAK	24399215	11.4	4.43	32.7	80	27	31	11.5	160	160.43	8.3	762	1 YEAR	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	1	NIL
27	76	M	10080104	BAILHONGAL	24430925	8.5	2.53	31.7	111.6	35.6	31.9	10.4	340	199.3	7.2	380	30 YEARS	HTN, SMOKING, IHD	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
28	60	F	10079441	BELAGAVI	24441200	8	3.19	36.9	84.8	25.3	29.8	11	255	152.66	8.9	522	20 YEARS	HTN, CVA	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	7	SEVERE
29	60	M	10072937	BELAGAVI	24390303	12.9	4.43	41.2	93	29.1	31.3	10.8	200	0.48	9.3	429	6 MONTHS	HTN	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
30	52	M	10079101	BELAGAVI	24435799	12.7	5.71	38.4	90.4	33.4	32	10.8	552	68.48	9.2	400	3 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
31	63	F	10078015	BELAGAVI	24435786	9.7	5.87	40.4	68.8	16.5	24	11.4	234	145.1	8.4	441	3 YEARS	HTN	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
32	56	F	10071720	BELAGAVI	24372830	16.3	6.01	53.1	88.4	27.1	30.7	20.5	531	80.86	7.9	362	4 YEARS	NIL	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
33	66	F	10075588	BELAGAVI	24399348	10.8	4.36	37.1	85.5	24.9	29.2	5.7	298	33.48	7.2	514	4 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
34	86	M	10072209	DHARWAD	24404544	10.2	3.41	33.6	98.5	29.9	30.4	13.8	202	357.75	7.4	497	20 YEARS	HTN	ABSENT	BE NORMAL	PRESENT	MACROALBUMINURIA	ABSENT	0	NIL
35	64	M	10083002	BELAGAVI	24489012	12.7	4.41	37.3	84.5	28.9	34.2	9.8	373	228.2	8.4	390	10 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
36	81	M	10096299	MATHAD	23156235	8.5	3.34	27.9	83.5	25.4	30.5	9.8	83	254	5.5	254	25 YEARS	HTN, IHD	ABSENT	BE NORMAL	ABSENT	MICROALBUMINURIA	ABSENT	2	NIL
37	59	M	10095617	BELAGAVI	24409123	11.9	4.76	40.5	80.2	26.2	32.7	7.7	584	802.2	9.5	682	10 YEARS	HTN, ALCOHOL	PRESENT	BE MODERATE NPDR	PRESENT	MACROALBUMINURIA	PRESENT	6	MODERATE
38	77	F	10095863	BELAGAVI	24456713	6.7	2.32	20.6	88.9	28.8	32.4	7.2	319	532.08	6	380	30 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	PRESENT	4	MILD
39	73	F	10096654	BELAGAVI	24489017	13.4	4.46	39.3	88.1	29.9	34	10.3	279	760.66	11.9	385	25 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	PRESENT	6	MODERATE
40	69	F	10096344	BELAGAVI	24459836	8.5	4.23	30.4	82.4	29.4	29.8	10.6	182	33.26	6.5	448	10 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
41	73	F	10096689	BELAGAVI	24409187	10.4	4.15	33.3	80.2	25.1	31.2	6.3	249	104.94	7.8	380	20 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
42	44	M	10083506	BELAGAVI	24474360	15.8	3.09	49.3	84.6	27.1	32	4.2	96	130.06	10.5	384	3 YEARS	HTN	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	2	NIL
43	63	F	10084110	BELAGAVI	24433145	12	5.09	40.7	88.4	30.4	29.8	11	360	133.19	10.1	380	20 YEARS	HTN, IHD	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	5	MODERATE
44	71	M	10083974	BELAGAVI	23499671	15	4	48.2	90	28	31.1	10.9	236	205.88	6.7	497	15 YEARS	HTN, IHD	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	6	MODERATE
45	67	F	10083713	BELAGAVI	24490137	10.1	4.41	34.6	78.5	22.9	29.2	9.8	269	1.71	9	393	6 YEARS	NIL	PRESENT	BE MILD NPDR	ABSENT	NORMAL	ABSENT	2	NIL
46	68	F	10084504	BELAGAVI	24451324	7.6	2.66	28.6	82.4	27	28.6	9.9	179	122.9	7.5	411	2 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	2	NIL
47	64	F	10084436	BELAGAVI	24499012	9.6	4.09	33.3	81.4	23.5	28.8	4.8	97	80.6	7.2	410	10 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
48	51	F	10084524	BELAGAVI	24456213	13.3	4.79	44	91.9	27.8	30.2	8.1	143	182.6	6.7	390	15 YEARS	HTN, HYPOTHYROID	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	6	MODERATE
49	64	M	10083918	BELAGAVI	24432142	10.3	3.41	33.2	84.8	25.4	29.9	8.9	271	214.6	7.4	490	15 YEARS	HTN	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
50	70	M	10084273	BELAGAVI	24466771	7.7	2.64	23.6	89.4	29.2	32.6	11	216	22.3	5.6	232	20 YEARS	NIL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	2	NIL
51	63	F	10082600	BELAGAVI	23345612	12	4.04	40.3	99.8	29.7	29.8	7.8	163	30	4.9	194	15 YEARS	NIL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
52	65	M	10083798	BELAGAVI	24490147	10.9	4.11	34.1	82.8	26.4	31.9	12.5	115	132.29	9.6	482	10 YEARS	HTN, IHD	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
53	78	M	10082565	BELAGAVI	24489457	13.3	4.42	42.3	95.7	30.1	31.4	7.6	255	190.34	11.1	370	15 YEARS	HTN, HYPOTHYROID, IHD	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
54	80	F	10082566	BELAGAVI	23356012	10.5	3.8	39.3	86	30	29	3.9	92	313.4	10.8	503	40 YEARS	HTN	PRESENT	BE MODERATE NPDR	PRESENT	MACROALBUMINURIA	PRESENT	6	MODERATE
55	63	M	10082867	BELAGAVI	23489541	9.1	3.28	29.6	90.2	27.7	30.7	9	303	273.63	6.6	371	10	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	2	NIL

																	YEARS								
56	76	M	10082946	BELAGAVI	24476512	8.4	3.32	26.1	78.6	25.3	32.2	12.8	266	703.86	6.6	406	25 YEARS	HTN, IHD	PRESENT	BE MODERATE NPDR	PRESENT	MACROALBUMINURIA	PRESENT	4	MILD
57	56	M	10083178	BELAGAVI	24409654	14	4.62	44.6	85.4	30.1	32.3	7.2	169	1.67	8.2	273	12 YEARS	HTN	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
58	60	M	10082786	BELAGAVI	24411990	12	6.19	39.8	64.3	19.4	30.2	12.3	168	50.82	7.1	384	20 YEARS	HTN	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
59	62	M	10082659	BELAGAVI	24490667	16.9	6.49	52	80.1	26	32.5	11.7	257	538.04	6.3	577	20 YEARS	HTN, IHD, CVA	PRESENT	BE MODERATE NPDR	PRESENT	MACROALBUMINURIA	PRESENT	4	MILD
60	72	M	10081501	BELAGAVI	24456109	10.5	3.33	28.6	85.9	31.5	36.7	8.4	300	88.56	6.9	475	15 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
61	79	M	10081820	BELAGAVI	24435975	8.6	3	27.1	90.3	28.7	31.7	7.9	303	51.31	7.1	490	30 YEARS	CVA	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
62	54	M	10081271	BELAGAVI	24412331	7.6	2.77	26.6	96	27.4	28.6	6.8	202	212.4	8	411	20 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	6	MODERATE
63	65	F	10107661	BELAGAVI	23356621	12.9	4.99	39.4	79	25.8	32.7	10.2	515	109.3	8.9	599	10 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
64	41	M	10104522	BELAGAVI	24490231	8	2.35	24.7	105	33.9	32.2	1.9	115	32.28	8.5	380	5 YEARS	NIL	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
65	73	M	10099651	BELAGAVI	24477421	8.3	3.19	30.4	86.5	31.4	30.2	7.9	211	139.18	6.6	405	20 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
66	50	M	10106233	BELAGAVI	24494477	9.1	3.01	29.6	89.3	30.2	30.7	12.7	180	132.3	7	599	4 YEARS	NIL	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
67	60	M	10105073	BELAGAVI	23455789	17.6	5.74	51.2	89.2	30.7	34.4	9.5	150	154.12	10	399	8 YEARS	HTN, IHD	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
68	68	F	10105398	BELAGAVI	24451890	11	4.08	34.4	84.3	27	32	13.2	147	84.6	8.7	374	12 YEARS	HTN, IHD	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
69	66	M	10107194	BELAGAVI	24411789	13.2	4.3	40.3	90.4	34.5	32.1	19.5	311	11.75	7.4	427	10 YEARS	HTN, ALCOHOL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
70	33	M	10103317	BELAGAVI	24476109	10.3	3.53	38.4	88.2	36.4	34.5	8.8	277	25.59	6.1	333	2 YEARS	NIL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
71	74	M	10107565	BELAGAVI	24499127	11.2	4.91	39.9	81.3	22.8	28.1	11.3	281	111.08	8.2	378	25 YEARS	HTN, IHD	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
72	50	M	10105655	BELAGAVI	24433771	12.9	4.67	41.6	89.1	27.6	31	13.3	40	1.35	8.4	316	5 YEARS	NIL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
73	72	M	10106002	BELAGAVI	24472554	12.3	4.06	38.4	86.5	30.4	31.5	9.3	182	123.6	7.4	390	15 YEARS	HTN, CVA	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
74	69	M	10107625	BELAGAVI	24490182	14.6	4.99	47.7	92.5	36.4	34.6	7.4	234	32.5	6.8	333	10 YEARS	HTN	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
75	50	F	10021487	BELAGAVI	24456712	12	4.44	39	87	27	30.8	8.9	294	133.19	9.5	415	15 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	2	NIL
76	71	F	10069835	BELAGAVI	23412754	14.1	5.36	44.1	82.2	26.4	31	8.4	309	47.46	9.6	390	5 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
77	62	F	10075760	BELAGAVI	23445790	12.2	4.75	38.8	81.4	25.7	31.2	6.6	189	186.31	8.1	445	15 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	3	MILD
78	63	F	10076063	YELLUR	24494712	12	4.25	39.2	90.4	28.3	30.8	9.2	393	61.23	6.7	370	10 YEARS	HTN	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
79	65	M	10024117	BELAGAVI	24467219	11.6	4.27	37.5	88.6	27.4	30.1	8.7	232	59.3	8.1	382	8 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
80	66	F	10070179	BELAGAVI	23801367	6.9	2.61	22.3	85.6	26.3	30.1	14.2	174	420.3	11.8	408	12 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	PRESENT	4	MILD
81	51	F	10021445	CHIKODI	24436712	11.1	4.57	38	84	24	28	9.6	267	7.45	7.3	324	5 YEARS	HTN	PRESENT	BE MILD NPDR	ABSENT	NORMAL	ABSENT	0	NIL
82	47	F	10021645	BELAGAVI	23884731	14.2	5.46	49.2	91	26.3	28.6	10	323	50.34	12	433	8 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
83	63	M	10051790	BELAGAVI	24458890	8.7	2.89	29	100	30.1	29.9	5	174	38.65	7.2	370	10 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	2	NIL
84	60	M	10083251	BELAGAVI	24457723	14.3	5.4	45.3	85.6	26.3	31	5.7	260	131.67	10.1	440	10 YEARS	HTN, IHD	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
85	63	M	10067694	DHARWAD	23368821	9.4	3.24	29.2	92	29	31.4	16.5	187	53.13	7.6	373	12 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
86	52	F	10071061	BELAGAVI	24407538	11.1	4.52	38.4	85.2	24.6	28	9.9	339	77.78	7.3	410	7 YEARS	HTN, HYPOTHYROID	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL

87	73	M	10049291	BELAGAVI	24460013	10.4	3.97	35.5	88.2	26	29.1	7.8	439	62.2	9.2	384	15 YEARS	HTN, IHD	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	4	MILD
88	65	F	10076774	BELAGAVI	24491076	6.5	2.72	23.3	85	23.6	27.4	18	291	10.37	6.1	265	2 YEARS	NIL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
89	62	M	10076724	BELAGAVI	23447653	11.3	3.91	36.2	92.3	28.1	31	3.9	300	20.24	9.6	320	5 YEARS	NIL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
90	45	F	10077527	BELAGAVI	24459210	13.7	4.94	43.3	88.7	27.1	31	6.1	347	19.42	9	286	2 YEARS	NIL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
91	84	F	10076316	BELAGAVI	24478562	10.4	3.9	36.4	93	26.4	28.1	7.8	225	363.92	12.4	410	25 YEARS	HTN, IHD	PRESENT	BE MODERATE NPDR	PRESENT	MACROALBUMINURIA	PRESENT	5	MODERATE
92	55	M	10024180	BELAGAVI	24433901	15.4	5.14	48.3	93.8	28	31.1	7.7	285	8.47	7.9	370	5 YEARS	NIL	ABSENT	BE NORMAL	ABSENT	NORMAL	ABSENT	0	NIL
93	75	M	10050836	BELAGAVI	24476129	13.1	4.6	40.8	88.6	28	32	19.2	251	185.94	9.2	444	20 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	PRESENT	3	MILD
94	51	F	10080082	BELAGAVI	23499012	13.9	4.79	46.5	96	29	30.9	5.54	363	72.28	10.4	450	8 YEARS	HTN	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
95	85	F	10082425	YELLUR	24453214	7	2.69	25	95	26.8	27.4	5.74	191	484.18	7	385	20 YEARS	HTN, CVA	PRESENT	BE MILD NPDR	PRESENT	MACROALBUMINURIA	PRESENT	4	MILD
96	58	F	10081543	BELAGAVI	24431621	12.1	4.33	38.8	89.4	27.3	31	3	149	60.83	6.6	366	5 YEARS	NIL	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
97	78	M	10077841	BELAGAVI	24431278	12.3	6.16	43.3	70	20	28.2	4.1	142	88.14	10	480	15 YEARS	HTN, SMOKING	PRESENT	BE MODERATE NPDR	PRESENT	MICROALBUMINURIA	ABSENT	2	NIL
98	72	F	10080413	BELAGAVI	24439612	9.7	4.76	32.7	68	20	29.4	6.9	220	33.37	7.6	352	15 YEARS	HTN	ABSENT	BE NORMAL	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
99	69	M	10076723	BELAGAVI	23678120	13.3	5	41.2	83.5	26.8	31.2	16.5	345	48.27	11.3	446	10 YEARS	NIL	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL
100	60	F	10076505	CHIKODI	24456713	8.3	3.06	28.4	94	27.6	28	10.5	115	46.72	11	398	12 YEARS	HTN, IHD	PRESENT	BE MILD NPDR	PRESENT	MICROALBUMINURIA	ABSENT	0	NIL