"PLATELET INDICES IN PATIENTS OF TYPE II DIABETES MELLITUS AT TERTIARY CARE TEACHING HOSPITAL – ONE YEAR CROSS SECTIONAL STUDY"

BY

REG NO: BG0122013

Dissertation

Submitted to

KAHER, Belagavi, Karnataka

In partial fulfilment
of the requirements for the degree of

M.D.
IN
GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE

J. N. MEDICAL COLLEGE
BELAGAVI - 590010. KARNATAKA

SEPTEMBER/OCTOBER 2025

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI

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With reference to the above, we wish to inform you that your proposed research project titled **"PLATELET INDICES IN PATIENTS OF TYPE II DIABETES MELLITUS 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.

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LIST OF ABBREVIATIONS

T2DM	-	Type 2 Diabetes Mellitus
MPV	-	Mean Platelet Volume
PDW	-	Platelet Distribution Width
P-LCR	-	Platelet Large Cell Ratio
PCT	-	Plateletcrit
IPF	-	Immature Platelet Fraction
HbA1c	-	Glycated Hemoglobin
UACR	-	Urine Albumin-Creatinine Ratio
NPDR	-	Non-Proliferative Diabetic Retinopathy
PDR	-	Proliferative Diabetic Retinopathy
CKD	-	Chronic Kidney Disease
CVD	-	Cardiovascular Disease
WHO	-	World Health Organization
DCCT	-	Diabetes Control and Complications Trial
UKPDS	-	UK Prospective Diabetes Study
SPSS	-	Statistical Package for the Social Sciences

ABSTRACT

Introduction: Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and impaired insulin secretion, leading to persistent hyperglycemia. The global prevalence of T2DM has surged, with India recognized as a major hub for diabetes cases. Microvascular complications such as diabetic retinopathy, nephropathy, and neuropathy significantly contribute to morbidity and mortality in diabetic patients. Indices of platelet, including Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW), have emerged as potential biomarkers for assessing microvascular complications and disease progression.

Methods: A hospital-based cross-sectional study was conducted with 125 patients diagnosed with T2DM. Clinical records, laboratory reports, and direct patient interviews formed the primary sources of data. Platelet indices, including MPV, PDW, and Platelet Large Cell Ratio (P-LCR), were measured and correlated with microvascular complications and HbA1c levels. Statistical analysis was performed using R version 4.4.0 and Microsoft Excel.

Results: Patients with microvascular complications exhibited significantly higher MPV, PDW, and P-LCR, with a marked correlation with disease severity. Higher values of MPV, PDW, Platelet LCR, IPF were seen with HbA1c $\geq$ 7. The study findings suggest that indices of platelet may serve as valuable indicators of diabetic complications, providing an opportunity for early diagnosis and intervention.

Conclusion: Indices of platelet can serve as cost-effective, non-invasive markers for early detection of diabetic complications, enabling better risk stratification and management. Further longitudinal research is needed to establish standardized

reference values and explore the mechanisms linking platelet dysfunction to microvascular damage.

Keywords: Type 2 Diabetes Mellitus, Platelet Indices, Microvascular Complications, Mean Platelet Volume, Platelet Distribution Width, HbA1c

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INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a metabolic condition caused by prolonged high blood sugar levels resulting from either resistance to insulin or insufficient insulin production..[1] It is a leading health issue globally, affecting > 422 million individuals, with India being recognized as the "diabetes capital of the world." The people with T2DM in India are alarmingly high, with over 77 million adults affected and an additional 25 million classified as pre-diabetic.[2] The growing issue of diabetes highlights, there is a critical demand for prompt identification and treatment to reduce its potential complications.

A significant worry regarding T2DM is its link to chronic complications that can lead to serious health problems and even death among those who suffer from the condition.[3] These complications are broadly classified into macrovascular and microvascular categories. Microvascular complications, including diabetic retinopathy, nephropathy and neuropathy, are caused due to chronic hyperglycemia-induced injury to tiny blood vessels. Detecting and managing these complications early is crucial as they often precede the onset of macrovascular diseases such as cardio-vascular disorders and stroke.[4]

Platelets are tiny blood cells without a nucleus that are essential for blood clotting and preventing excessive bleeding. In individuals with T2DM these platelets often show heightened activity and continuous activation leading to an increased risk of thrombosis. This impaired function of platelets is linked not only to the development of large blood vessel complications but also significantly contributes to issues affecting smaller blood vessels.[5] New research suggests that platelet

measurements including MPV and PDW are important indicators in understanding complications of Diabetes.[6]

Alterations in indices of platelet have been increasingly studied in patients with T2DM. There are studies which show that higher MPV and PDW levels are seen in those having diabetes as compared to non-diabetic. Elevated MPV and PDW have been associated with enhanced platelet activation and show a correlation which is positive and significant with HbA1c levels and the severity of microvascular complications.[7,8] These findings suggest the utility of indices of platelet as biomarkers for assessing the risk and progression of diabetic complications.[9]

While there is an increasing amount of evidence, there are still considerable gaps in our understanding of how indices of platelet contribute to the prediction of microvascular complications. Variability in study designs, sample sizes, and confounding factors such as coexisting medical conditions and medication use have led to inconsistent results. Many studies have been performed in restricted geographic locations, underscoring the necessity for research across varied populations, particularly in India, where the people present with uncontrolled diabetes and its complications.

This research focuses on assessing indices of platelet in individuals with T2DM and their links to microvascular complications, including diabetic retinopathy, nephropathy and neuropathy. By investigating the connection between indices of platelet and HbA1c levels, the study aims to uncover their potential role as early indicators of complications. The results from our study could enhance the stratification of risk and improve management approaches for those with T2DM, ultimately helping to lessen the impact of related health issues and mortality.

AIMS AND OBJECTIVES

1. To study Platelet Indices in patients with Type II Diabetes Mellitus.
2. To correlate Platelet Indices with Microvascular Complications of Type II Diabetes Mellitus
3. To correlate Platelet Indices with HbA1c.

REVIEW OF LITERATURE

Diabetes mellitus is a long-term metabolic condition recognized by WHO which causes elevated levels of glucose in blood. If not managed properly, it can lead to complications affecting various organs. Of all the diabetes cases, almost 90% of the cases are T2DM. This form of diabetes is marked by insufficient pancreatic beta cell production of insulin, resistance to insulin in different tissues, and an inadequate insulin response to address this resistance.[10,11] T2DM represents about 90% of all diabetes cases. In this condition, the body becomes less responsive to insulin, a phenomenon referred to as insulin resistance. To counteract this resistance, the body initially produces more insulin to regulate glucose levels effectively. However as time goes on, the ability to produce insulin diminishes resulting in the onset of T2DM. This form of diabetes is most frequently diagnosed in people over the age of 45.[1] Advancement of the disease results in insulin secretion's inability to regulate levels of sugar in blood effectively, leading to hyperglycemia. Individuals diagnosed with T2DM often exhibit characteristics of weight gain or a increased body fat composition, particularly concentrated in the abdominal region. In this context, adipose tissue contributes to insulin resistance through a variety of inflammatory mechanisms. This includes the enhanced release of free fatty acids (FFAs) and an imbalance in adipokine levels. The growing number of T2DM cases can be linked to several elements such as the global rise in obesity, lack of physical activity, consumption of high-calorie foods, and an older population. Collectively these factors have resulted in a fourfold increase in both the occurrence and prevalence of T2DM.[12,13] The development of T2DM involves several key organs, including the pancreas.[14] Recent findings suggest that dysfunction in adipokines, changes in gut microbiota, and issues with immune regulation and inflammation are critical

pathophysiological factors.[15] Recent studies on population health have pointed to worrying trends related to T2DM presenting a grim forecast for the future. In 2019, the IDF indicated that diabetes caused approximately 4.2 million deaths all over the world. Around 463 million adults with the age between 20 and 79 were living with the condition, and is estimated to reach 700 million by 2045. The estimated costs for diabetes-related healthcare reached at least 720 billion USD in 2019. The actual implications of T2DM could be even more profound, as nearly one-third of those affected are not diagnosed, amounting to about 232 million individuals. The majority of these undiagnosed cases are found among people aged 40 to 59. The frequency and effects of T2DM differ across regions, with over 80% of those impacted residing in low and middle income countries, making effective treatment more challenging. Individuals with T2DM are 15% more likely to die from any cause compared to those without the disease, with CVD being the foremost contributor to illness and death associated with T2DM.[16] The occurrence of T2DM is affected by a mix of genetic factors and environmental conditions. Genetic traits become more evident in individuals when they are exposed to lifestyles that encourage inactivity and diets rich in calories. While extensive studies across the genome have identified prevalent variants linked to T2DM, these variants account for only approximately 10% of the total variation in the disease indicating that less common genetic variants may also play a significant role.[17] People from different ethnic backgrounds may display unique physical traits that heighten their risk of developing combinations of CVD risk factors, including high BP, insulin resistance, and abnormal lipid levels.[18]

Pathophysiology of Microvascular Complications in T2DM

Complications affecting the small blood vessels in diabetes primarily result from long-term high blood sugar levels. Key research, which includes “UK

Prospective Diabetes Study (UKPDS") and the "Diabetes Control and Complications Trial (DCCT)", has shown a significant relationship between effective blood sugar management and the incidence of microvascular conditions.[19,20] These conditions are more likely to affect tissues which do not rely on insulin for glucose uptake, as kidneys, retina, and vascular endothelium, since these areas are directly influenced by blood glucose fluctuations.[21] Microvascular damage progresses due to various factors, including direct glucose-induced injury to blood vessels, oxidative stress from excessive superoxide production, and the formation of substances like sorbitol and advanced glycation end-products.[22] These disruptions lead to blood flow alterations, vessel permeability, and protein accumulation, which in turn contribute to organ dysfunction.[23]

a) Diabetic retinopathy, a big reason of vision loss in adults with diabetes, results from the damaging effects of hyperglycemia on the retinal microvasculature. When an injury occurs, it sets off a chain of changes in the tissue. This includes the basement membrane becoming thicker, capillary walls becoming more permeable, and the appearance of microaneurysms. These changes result in a decreased oxygen supply to the retina, which can cause the new capillaries to form which are susceptible to breaking and bleeding.[24] The buildup of fluid in the retina can cause macular edema, which can develop at any stage of retinopathy. A significant number of individuals with T2DM—up to 40% - are affected by retinopathy at the time of diagnosis. This statistic highlights the tendency for cases within this group to be identified later than preferable.[25] Risk factors for retinopathy include diabetes duration, blood sugar control, and blood pressure, with evidence suggesting that rapid improvements in blood sugar levels may temporarily worsen retinopathy in some cases. During

pregnancy, retinopathy may worsen, necessitating frequent retinal assessments to monitor for progression.[26]

b) Diabetic nephropathy, which found in 30–40% of individuals with diabetes, results from the combined effects of hyperglycemia and hypertension on the kidneys. The condition is characterized by changes like thickening of the basement membrane, kidney fibrosis, and arteriosclerosis, which initially lead to glomerular hyperfiltration and later to kidney function deterioration.[27] The progression to overt proteinuria is often marked by the presence of microalbuminuria, which signifies early kidney damage. Although not all patients with microalbuminuria progress to severe kidney disease, those who do face a high risk of cardiovascular events and kidney failure.[28] Managing cardiovascular risk factors aggressively is crucial for patients with microalbuminuria, and timely referrals to nephrology services are necessary to optimize care and prevent further renal damage.[29]

   DKD is a common microvascular complication of DM, affecting approximately 25% of the diabetic population.[30] Moreover, DM is the major cause of ESRD in the developed world, accounting for 50% of all cases.[31]

c) Diabetic neuropathy encompasses a variety of conditions that impact people with diabetes, particularly those with type 2 diabetes mellitus (T2DM), manifesting through different clinical symptoms. It is the most prevalent microvascular complication associated with T2DM, affecting nearly 50% of patients after a decade of the disease, with about 20% exhibiting symptoms at diagnosis. Many cases remain undiagnosed due to the asymptomatic nature of approximately half of the affected individuals. If untreated, diabetic neuropathy can lead to serious complications such as Charcot neuroarthropathy, foot ulcers, and foot amputations, significantly diminishing

quality of life and life expectancy.[32] The condition is classified based on factors like prevalence, distribution, clinical progression, and specific features, with diabetic peripheral neuropathies (DPN) traditionally divided into symmetric polyneuropathies, focal/multifocal neuropathies, and mixed forms. Recently, DPN has been further categorized into typical forms, particularly distal symmetric sensorimotor polyneuropathy (DSPN), which accounts for 75% of diabetic neuropathies and results from chronic hyperglycemia combined with cardiovascular risks. Currently, there is no effective treatment to reverse the damage caused by this condition, emphasizing the urgent need for preventive measures.[33]

Platelet Indices: Definitions and Clinical Significance

Platelets, which are small fragments produced by megakaryocytes, typically survive for 5 to 7 days. They are important for the regulation of bleeding and blood clotting. When there is injury to the endothelium, platelets get activated and stick to the exposed areas of the blood vessel's outer layer. This interaction leads to the creation of a platelet plug and contributes to the formation of a thrombus, which is vital for stopping excessive blood loss. In pathological conditions, platelets become essential for the formation of occlusive thrombi, making them a primary focus for preventing arterial thrombosis. Beyond their hemostatic role, platelets contribute to innate immunity and influence processes such as the regulation of tumor growth and vessel extravasations, underscoring their multifaceted functions in circulation. Understanding these diverse roles is essential for developing targeted interventions and treatments related to platelet function [34].

Indices of platelet are crucial metrics used in the assessment of platelet function and overall hematologic health. They help clinicians evaluate a patient’s

coagulation status, particularly in the context of various medical conditions such as clotting disorders, thrombocytopenia, and myeloproliferative diseases.(34) Understanding these indices can inform diagnostic processes and treatment strategies in various clinical settings. The primary indices of platelets include "Mean-Platelet-Volume (MPV), Platelet-Distribution-Width (PDW), Platelet-Large-Cell-Ratio (P-LCR), Plateletcrit (PCT), and Immature -Platelet- Fraction (IPF)".[35]

a) "Mean Platelet Volume (MPV)" reflects platelet size circulating in bloodstream, usually expressed in femtoliters (fL). It is the key platelet parameter examined and indicates the typical platelet size present in blood samples. In healthy individuals, MPV values generally fall between 7.2 and 11.7 fL.[36] An increased MPV suggests the presence of younger, more active platelets, which is frequently associated with conditions that involve heightened platelet turnover, such as inflammation or recovery from hemorrhage. On the other hand, a decreased MPV may point to older, less active platelets, potentially signaling chronic diseases or issues with bone marrow function.[36]

   The variations in MPV levels are not solely dependent on platelet count abnormalities; they are also influenced by the laboratory techniques employed during testing. Multiple factors—including ethnicity, age, tobacco use, alcohol consumption, and physical activity—can impact MPV readings.[36]

   Studies have investigated MPV as a potential prognostic biomarker, with many findings suggesting that elevated MPV correlates with poorer clinical outcomes. For example, study by Lembeck et al. showed a link with increased MPV levels and adverse prognoses with pancreatic adenocarcinoma patients, regardless of levels of other established prognostic indicators. This trend was

similarly noted in patients with myocardial infarction, who often had associations with worse clinical outcomes when presenting with higher MPV levels. Additionally, reduced MPV levels may indicate the presence of low-grade inflammation, as seen in conditions like rheumatoid arthritis.[37]

b) "Platelet Distribution Width (PDW)" quantifies the size variance of platelets , reflecting the degree of homogeneity or heterogeneity within the platelet population. An elevated PDW indicates greater size variability, which may be associated with increased platelet activation or the bone marrow's response to various factors, including inflammation or malignancy. In contrast, a reduced PDW suggests a more homogeneous platelet population, often seen in disorders that affect either platelet function or production.[38]

PDW values are typically expressed as percentages, with normal ranges generally falling between 9.6% and 15.9%. Variations in PDW can yield critical insights into the physiological processes influencing platelets. This measurement is recognized as an indicator of platelet anisocytosis—reflecting the size distribution of platelets formed by megakaryocytes—which tends to increase during platelet activation.[39]

While a review by Budak et al. proposes that PDW reference levels could range from 8.3% to 56.6%, evidence supporting such a broad range is scant in the literature. Studies examining healthy individuals have demonstrated that PDW typically falls between 10% and 18%. Furthermore, alterations in PDW have been documented in individuals suffering from various medical conditions, positioning it as a promising biomarker.[40]

Notably, PDW is often found to be proportionally related to Mean Platelet Volume (MPV) in healthy populations. However, under certain pathological conditions, such as threatened preterm labor, an increase in PDW may occur

alongside a decrease in MPV.(41) This divergence was also highlighted by Aydogan et al., who investigated MPV and PDW levels in patients diagnosed with both perforated and non-perforated acute appendicitis. Moreover, Amin et al. reported that elevated PDW in patients experiencing vaso-occlusive crises related to sickle cell disease may result from megakaryocyte hyperplasia.[42]

c) "Platelet-Large Cell Ratio (P-LCR)" determines percentage of bigger platelets relative to total number of platelets. A higher P-LCR often indicates increased platelet production or the body's reaction to various factors like bleeding or inflammation. This measurement is especially useful for tracking patients who are recovering from low platelet counts and for evaluating conditions such as essential thrombocythemia.[43]

   Analyzing P-LCR in conjunction with other indices of platelet is crucial, as it enriches the understanding of overall platelet functionality. While reference ranges may vary among different studies, P-LCR is commonly expressed as a percentage of the total platelet count, typically falling between 15% and 35%.[44]

   In the research conducted by Baig, a direct correlation was identified between P-LCR and other platelet metrics, including Platelet Distribution Width (PDW) and Mean Platelet Volume (MPV), while an inverse correlation with the total platelet count was noted in patients with thrombocytopenia. P-LCR appears to be more responsive to changes in platelet size compared to MPV, despite their observed correlation.([45]

d) Plateletcrit (PCT) is a quantitative metric that assesses the total volume of platelets in the bloodstream, analogous to how hematocrit evaluates red blood cell volume. This measurement is obtained by multiplying the platelet count

by the mean platelet volume (MPV). Elevated PCT levels may suggest thrombocytosis, reflecting an increased mass of platelets, whereas reduced PCT values can indicate thrombocytopenia, potentially signifying bone marrow dysfunction or acute medical conditions.[46]

The standard reference range for Platelet Crit (PCT) typically lies between 0.1% and 0.4%, but these values can differ based on individual health conditions and the clinical context. Generally, PCT values are commonly found within the range of 0.22% to 0.24%. This measurement is an important tool for screening potential irregularities in platelet levels. PCT has a nonlinear relationship with platelet count and holds similar clinical importance.[46]

In addition, a study by Tang et al. investigated possibility of using PCT as a novel marker for diagnosing Crohn's disease in individuals who have reduced levels of "high sensitivity C-reactive protein (hs-CRP)".[47]

e) "Immature Platelet Fraction (IPF)" reflects the ratio of new, immature platelets released, circulating in the blood, serving as a significant indicator of bone marrow function. Elevated IPF values indicate increased platelet production, which can be seen in conditions such as recovery from thrombocytopenia, an active response to infection, or bone marrow disorders. Conversely, a low IPF may suggest decreased production and can be associated with various conditions such as bone marrow suppression or chronic illnesses. Normal IPF values typically constitute a small percentage of the total platelet count, and variations can inform clinicians about a patient's hematologic health and bone marrow function.[48]

These platelet indices, when evaluated together, provide comprehensive insights into patient health and assist in making informed clinical decisions. Abnormal values may reflect underlying health issues that warrant further investigation or intervention, emphasizing the importance of these metrics in modern medical practice. Understanding the implications of these indices can significantly enhance patient care and improve health outcomes through tailored treatment approaches based on individual hematologic profiles.

The utility of indices of platelet in predicting vascular complications is gaining recognition, particularly among patients with diabetes and CKD. These indices reflect platelet activity and correlate to micro - vascular and the macro - vascular complications. Research has indicated that elevated indices of platelet correlate with glycemic control that is poor and an high risk of vascular complications, establishing their role as valuable predictive biomarkers.

In T2DM patients, higher levels of MPV, PDW and PCT are frequently observed, particularly in those with microvascular complications such as retinopathy and neuropathy. MPV is significantly elevated in individuals with diabetes compared to non-diabetics and demonstrates a correlation with HbA1c, FBS, and postprandial blood sugar levels.[49] This correlation suggests the potential of MPV as predictor of vascular complications. Furthermore, studies have shown that diabetic patients experiencing vascular complications exhibit significantly higher MPV, PDW, and P-LCR. This finding indicates these indices may serve to be cost-effective predictive parameters.

Association Between Indices of platelet and Microvascular Complications

The association between indices of platelet with that of microvascular complications in people with T2DM has emerged as a significant topic in diabetes research. Elevated levels of indices of platelet, including “mean – platelet - volume (MPV), platelet - distribution - width (PDW), and platelet -large cell - ratio (P-LCR)” , have been consistently correlated with the onset of microvascular complications such as retinopathy, nephropathy, and neuropathy.

Paul et al. (2024) and Khanna et al. (2024) proved that individuals with T2DM accompanied by microvascular complications demonstrated significantly elevated levels of MPV (mean value of 12.54 fl) and PDW (mean value of 19.05 fl) compared to their counterparts without complications, who exhibited values of 10.21 fl and 13.29 fl, respectively. This significant difference suggests that higher indices of platelet could be indicative of underlying pathological processes associated with diabetes.[50,51]

Gamage et al. (2024) and Meena (2023) has highlighted the relation between elevated indices of platelet and poor glycemic control in T2DM patients. Specifically, higher indices of platelet correlate with HbA1c levels above 7%, a threshold often associated with high risk for diabetes-related complications. The direct relationship between these biomarkers underscores the importance of monitoring indices of platelet as part of comprehensive diabetes management.[52,53]

The predictive capacity of indices of platelet for the early identification of complications has been examined in multiple studies recently. Khanna et al. (2024) and Meena (2023) suggest that these indices can serve as valuable, economical, and non-invasive tools for ongoing monitoring of patients with T2DM. Their ability to act

as reliable predictors may allow healthcare providers to implement preventative strategies early in the disease process, potentially mitigating the progression of complications.[51,53]

In conclusion, the relation between altered indices of platelet and that of microvascular complications in patients with T2DM highlights the need for further research into these parameters as potential biomarkers for disease progression and management. As our understanding of these relationships deepens, the integration of indices of platelet into practice may aid the ability to monitor and ultimately improve outcomes for individuals living with T2DM.

Indices of platelet and Glycemic Control

Hemoglobin A1c (HbA1c) serves as an important marker for assessing long-term blood sugar management in people with diabetes. It provides an estimate of average level of glucose from the previous three months by measuring the proportion of glycated hemoglobin in red blood cells. Standard diagnostic guidelines indicate that diabetes is well-managed when the HbA1c level is below 7%, which is associated with a decreased likelihood of developing diabetes-related complications.[54] Higher HbA1c levels show a poor glycemic control and are linked to an higher risk of complications as neuropathy, nephropathy, retinopathy, and CVD. Regular monitoring of HbA1c provides healthcare professionals with insights into a patient's adherence to their diabetic regimen, the effectiveness of treatment interventions, and necessary adjustments to management plans. Given its significance, HbA1c has become a cornerstone in the management of diabetes, guiding therapeutic decisions and lifestyle modifications.[55]

Recent studies indicate a significant relationship between HbA1c levels and changes in indices platelet. Elevated HbA1c levels in individuals with diabetes can lead to alterations in platelet function, characterized by increased platelet activation and aggregation. This relationship is critical because it suggests that chronic hyperglycemia can enhance platelet reactivity, contributing to pro-thrombotic states. High indices of platelet have been observed in poorly controlled diabetes, where the dysregulation of glucose metabolism may cause structural changes in platelets, leading to their increased size and activity.[56] Understanding the interplay between HbA1c and indices of platelet can provide valuable insights into the increased cardiovascular risk associated, underlining the importance of effective glycemic management.

Elevated indices of platelet in individuals with poorly controlled diabetes present significant clinical implications. Hyperglycemia can lead to various complications, including enhanced thrombotic activity due to increased platelet aggregation. This heightened platelet activity is related with an high risk of cardiovascular events, which are already elevated in the diabetic population. Moreover, patients with higher platelet counts and volume may experience greater systemic inflammation, further exacerbating the risk of complications.[57] Clinicians should consider monitoring not only HbA1c levels but also indices of platelet to assess the overall risk profile of diabetic patients. This comprehensive approach to patient management may facilitate timely interventions, such as antiplatelet therapy or more aggressive glycemic control strategies, ultimately improving patient outcomes and decreasing the burden of complications. Recognizing and addressing these correlations can enhance personalized treatment plans, aiming for better cardiovascular health in this vulnerable population.

Comparative Studies on Indices of platelet in Diabetic and Non-Diabetic Populations

Jindal et al. conducted a study with 75 diabetic patients, 50 of whom had 1 or more microvascular complications, alongside fifty control subjects from the hospital. They collected and analyzed anticoagulated blood using an automated cell counter to assess indices of platelet and total count. The findings revealed that diabetic patients exhibited more levels of MPV, PDW, and platelet-large cel- ratio compared to controls, with PDW being notably elevated in those with complications. Through a stepwise discriminant analysis, they successfully classified about 78.6% with diabetic complications using only PDW and MPV as key input variables, indicating that these indices of platelet are distinct between controls and diabetics, as well as among diabetics with varying complication statuses.[58]

Khanna et al. conducted a case-control study from 2021 to 2022, involving 200 participants divided into 2 groups of 100: one group of patients with a history of diabetes mellitus (DM) lasting over five years (cases) and a control group without diabetes. The diabetic group was further categorized into two subgroups: those with at least one complication (Group IA) and those without any complications (Group IB), which included conditions such as diabetic retino-pathy, nephron-pathy, and neuro-pathy. Hemoglobin levels, platelet counts, and indices were taken into accounts. The results revealed high MPV of 12.089 ± 1.450 fL in diabetics compared to 9.464 ± 1.424 fL in controls ($p = 0.001$). Additionally, PDW and P-LCR were notably elevated in cases, with values of 16.868 ± 2.352 fL and 34.975 ± 8.056%, respectively. These measurements were further differentiated within the diabetic cohort, showing that those with microvascular complications experienced a significant increase in MPV (12.5960 ± 0.95660 fL), PDW (17.1140 ± 2.58228 fL), and P-LCR

(35.408 ± 3.5490%) compared to those without complications. The statistical significance for these differences was confirmed with p-values of 2×10^{-3} for MPV and PDW, and 3.1×10^{-3} for P-LCR.[59]

Saha et al. conducted a study involving 160 patients, which included 80 individuals diagnosed with diabetes and 80 control participants. They meticulously documented the clinical histories and collected blood samples under sterile conditions for analysis with an automated hematology analyzer. The final data encompassed variables such as age, gender, Mean Platelet Volume (MPV), which were subsequently analyzed using SPSS software. The results indicated that India is on course to become the diabetes capital, currently reported at a 9.3% prevalence rate. Diabetes mellitus is a significant factor in global health issues, contributing to high rates of illness and mortality. The research indicated that levels of MPV and PCT were markedly higher in individuals with diabetes. Also patients with diabetes who experienced complications exhibited raised values in their platelet parameters. The findings of the study emphasize the link between elevated platelet volume and a tendency toward increased clotting risk in diabetes, suggesting that monitoring these platelet parameters might serve as a straightforward, efficient, and cost-effective approach to assess thrombotic risk in diabetic individuals.[60]

Walinjkar et al. conducted a study involving 125 people attending the diabetes outpatient department and those into the medicine department, alongside age- and sex-matched non-diabetic controls. They gathered comprehensive histories concerning the duration of diabetes, medications, and any previous instances of stroke, ischemic heart disease, and hypertension. The evaluation focused on T2DM patients for microvascular complications, and data on platelet indices, fasting and postprandial glucose levels, HbA1C, and creatinine were collected from blood

samples from the veins. The findings revealed that indices of platelet were elevated in individuals with T2DM. Additionally, individuals with complications showed even greater increases in these indices, and platelet dysfunction was positively correlated with HbA1C levels as well as with retinopathy, nephropathy, and neuropathy.[61]

Ahladas et al. conducted a study examining platelet and biochemical data from patients attending outpatient clinics. The research included 200 participants aged between 30 and 60 years, with 100 diagnosed with T2DM and 100 serving as a non-diabetic control group. The results revealed that patients with T2DM had significantly elevated levels of PCT, MPV, and PDW compared to the controls, indicating a more reactive platelet profile. Specifically, PCT values were 0.21% vs. 0.20%, MPV values were 8.69 fl vs. 8.27 fl, and PDW values were 17.8 fl vs. 17.5 fl, with all differences being statistically significant. Increased levels of these parameters were associated with complications related to T2DM. Among patients with macro-vascular complications, researchers found a relationship between glycated haemoglobin and both MPV and PDW. For those experiencing complications of microvasculature , there was a notable connection between total number of platelets and MPV in relation to glycated haemoglobin. The study suggests that the unique platelet features observed in T2DM patients indicate that their platelets may be more reactive. Therefore, assessing platelet characteristics could be a viable and economical method for identifying long-term complications in diabetic patients at an early stage.[62]

Previous studies have encountered several limitations, primarily characterized by small sample sizes, which hinder the generalizability of their findings. In particular, there is a notable need for further research that specifically targets microvascular complications within Indian populations, as this area remains underexplored. This study is designed to address these gaps by utilizing a larger, more

diverse sample and implementing a longitudinal approach to better understand the prevalence and impact of microvascular complications in this demographic. Through these efforts, we aim to contribute valuable insights that could inform clinical practices and policies in the management of such conditions.

The literature on Type II Diabetes Mellitus reveals a complex interplay between metabolic dysfunction, platelet activity, and microvascular complications. T2DM has insulin resistance and chronic hyperglycemia, leading to significant health burdens globally, particularly in regions like India where prevalence rates are alarmingly high.

There is growing evidence that platelet indices might be useful indicators for determining a patient's risk of microvascular problems in type 2 diabetes. Elevated levels of these indices correlate with poor glycemic control and the severity of complications, indicating their potential utility in clinical settings for risk stratification.

However, significant gaps remain in understanding the precise mechanisms linking platelet dysfunction to the pathogenesis of microvascular complications. Variability in study designs and populations highlights the need for further research, particularly in diverse demographic settings. Future studies should aim to establish standardized methodologies to evaluate indices of platelet across different populations, which could enhance our understanding of their prognostic value.

In conclusion, addressing the challenges associated with T2DM requires a multifaceted approach that includes not only improved glycemic control but also a deeper investigation into the role of indices of platelet as prognostic markers. By advancing our knowledge in this area, we can develop more effective strategies for early diagnosis and management of diabetic complications, ultimately reducing the load of this pervasive disorder on individuals and health care systems alike.

MATERIALS AND METHODS

Source of Data

The study utilized data from patients diagnosed with T2DM from Medicine OPD and IPD in the Tertiary Care Teaching Hospital between 1st April 2023 to 31st March 2024 over one year period. Clinical records, laboratory reports, and direct patient interviews formed the primary sources of data. Additional information was collected through validated questionnaires and diagnostic tests focusing on indices of platelet and microvascular complications.

Study Design

A hospital-based cross-sectional study.

Study Period

1st April 2023 to 31st March 2024 over one year period.

Sample Size

Formula used for sample size calculation is,

Sample size at 95% Confidence Interval,

At 18% Tolerable error , 5% Attrition (1.05)

$n = [(Z_{(1-\alpha/2)})^2 \times (SD)^2 / (18\% \text{ of } SD)^2] \times 1.05$

$n = 124.5 \sim 125.$

Hence, minimum sample size estimated is 125 participants.

Sampling technique

Participants were recruited using purposive sampling, targeting individuals with a confirmed diagnosis of T2DM who met the inclusion criteria. Efforts were made to ensure diversity in terms of age, gender, and duration of diabetes.

Inclusion Criteria and Exclusion criteria

Inclusion Criteria:

1. Age > 18 years
2. Patients diagnosed with T2DM

Exclusion Criteria:

1. History of Blood Transfusion in last 3 months
2. Patients on any Anti-Platelet Medications
3. Patients diagnosed with Chronic Kidney Disease
4. Pregnant Females

Data collection procedure

An informed written consent was obtained from all the study subjects.

- Detailed clinical history was taken for all patients including Demographic details, Duration of Diabetes with special emphasis on complications of diabetes.
- A thorough clinical examination and relevant investigations like Complete Hemogram, HbA1c and Urine was done for patients to assess the complications of diabetes.

- Diabetic Retinopathy was evaluated by using Direct Ophthalmoscopy.
- Diabetic Neuropathy was evaluated using a 10-g Monofilament for touch , pain sensation by pin prick, vibration by 128Hz Tuning Fork, Temperature sense and ankle jerk. Revised Neuropathy Disability Score (NDS) was used for assessment of Neuropathy. (Reference article https://doi.org/10.4103%2Fijem.IJEM_13_19)
- Diabetic Nephropathy was evaluated by Urine Albumin: Creatinine ratio for micro and macroalbuminuria.
- A predesigned and pre-tested Research Questionnaire was developed to collect data from all the study participants.

Data processing and analysis

The analysis of data is conducted using statistical software R version 4.4.0 along with Microsoft Excel. Categorical variables are represented through frequency tables, while that of continuous variables are expressed as Mean ± SD or Median (Min, Max). The Chi-square test is used to determine associations between categorical variables. The Shapiro-Wilk test and QQ plot are utilized to assess the normality of variables. If the data exhibit a normal pattern of distribution, parametric tests are considered ; otherwise, non-parametric tests are considered. To compare variable distributions concerning microvascular complications, a two-sample t-test is done , while the Mann-Whitney U test is done for non-parametric comparisons. Spearman's rank correlation test is considered to evaluate indices of platelet and HbA1c levels. A p-value of ≤ 0.05 is considered statistically significant.

RESULTS

Table 1: Distribution according to demographic details.

Variables	Sub Category	Number of subjects (%)
Age (years)	Mean ± SD	62 ± 12.43
	Median (Min, Max)	63 (32, 98)
Gender	Female	48 (38.4%)
	Male	77 (61.6%)

The mean age of subjects is 62 ± 12.43 years and a median age of 63 years (range: 32–98 years). Out of 125 subjects, 77 (61.6%) were male, and 48 (38.4%) were female, indicating a male predominance in the study population.

Figure 1: Distribution according to gender.

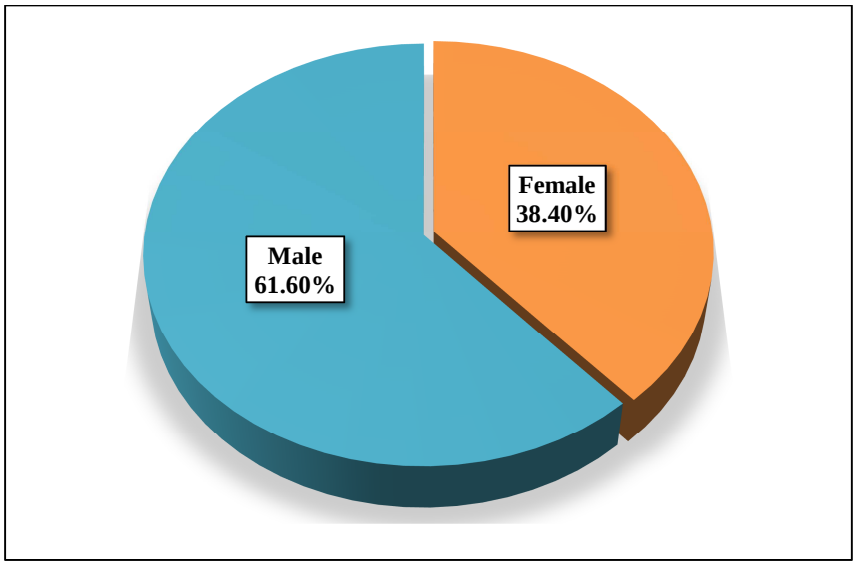

Table 2: Distribution according to HBA1C.

Variables	Mean ± SD	Median (Min, Max)
HBA1C	8.23 ± 2.07	7.6 (4.9, 16.7)

The mean HbA1c level in the study population was 8.23 ± 2.07%, with a median of 7.6% (range: 4.9–16.7%).

Table 3: Distribution according to hematological parameters.

Hematological Parameters	Mean ± SD	Median (Min, Max)
Hemoglobin	11.78 ± 2.4	12 (6.5, 17)
RBC Count	4.43 ± 0.88	4.5 (2.41, 6.53)
Hematocrit	38.71 ± 7.15	39.2 (22.3, 55.9)
MCV	87.96 ± 7.86	88.9 (64.8, 111.6)
MCH	26.73 ± 2.97	27.2 (16.5, 35.6)
MCHC	30.35 ± 1.71	30.6 (24, 36.7)
RDW	14.74 ± 2.31	14 (11.5, 23.9)
TLC	9.91 ± 5.56	8.4 (2.95, 33.14)

The mean hemoglobin level was 11.78 ± 2.4 g/dL, with a median of 12 g/dL (range: 6.5–17 g/dL). RBC count averaged 4.43 ± 0.88 million/µL, and the mean hematocrit was 38.71 ± 7.15%. Other indices showed mean values as follows: MCV, 87.96 ± 7.86 fL; MCH, 26.73 ± 2.97 pg; MCHC, 30.35 ± 1.71 g/dL; and RDW, 14.74 ± 2.31%. TLC had a mean of 9.91 ± 5.56 $x10^3$/µL.

Table 4: Distribution according to indices of platelet :

Platelet indices	Mean ± SD	Median (Min, Max)
Platelet Count	249.1 ± 92.2	253 (14, 495)
MPV	10.3 ± 1.07	10.2 (8.3, 13.5)
PDW	11.67 ± 2.52	11.4 (7.6, 20.5)
Plateletcrit	0.24 ± 0.08	0.24 (0.01, 0.42)
Platelet LCR	27.05 ± 8.33	26.2 (10.5, 48.5)
IPF	5.37 ± 3.24	4.7 (1, 18.2)

The platelet count had a mean of 249.1 ± 92.2 x10³/µL, with a median of 253 x10³/µL (range: 14–495 x10³/µL). MPV averaged 10.3 ± 1.07 fL, PDW was 11.67 ± 2.52%, plateletcrit was 0.24 ± 0.08%, and platelet LCR was 27.05 ± 8.33%. IPF had a mean value of 5.37 ± 3.24%.

Table 5: Distribution according to UACR.

Variables	Mean ± SD	Median (Min, Max)
UACR	204.6 ± 454.82	60.71 (0.29, 3114.2)

The mean UACR was 204.6 ± 454.82 mg/g, with a median of 60.71 mg/g (range: 0.29–3114.2 mg/g).

Table 6: Distribution according to diabetic nephropathy.

Diabetic Nephropathy	Number of subjects (%)
Macroalbuminuria	18 (14.4%)
Microalbuminuria	65 (52%)
Normal	42 (33.6%)

Among the subjects, 18 (14.4%) had macroalbuminuria, 65 (52%) had microalbuminuria, and 42 (33.6%) had normal albuminuria levels. This indicates that more than half of the population had some degree of diabetic nephropathy, predominantly microalbuminuria.

Figure 2: Distribution according to diabetic nephropathy

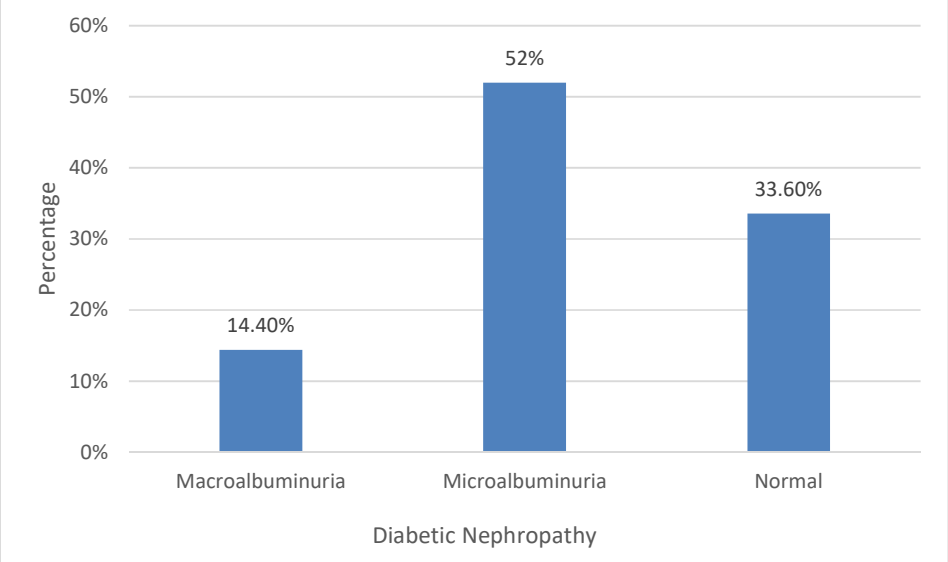

Table 7: Distribution according to duration of T2DM.

Variables	Mean ± SD	Median (Min, Max)
T2DM Years	5.12 ± 4.18	4 (0.1, 20)

The mean duration of Type 2 Diabetes Mellitus (T2DM) was 5.12 ± 4.18 years, with a median duration of 4 years (range: 0.1–20 years).

Table 8: Distribution according to diabetic retinopathy.

Diabetic Retinopathy	Number of subjects (%)
BE Fundi WNL	63 (50.4%)
BE Mild NPDR	39 (31.2%)
BE Moderate NPDR	17 (13.6%)
BE Moderate PDR	4 (3.2%)
BE Severe NPDR	1 (0.8%)
RE Mild NPDR	1 (0.8%)

A total of 63 (50.4%) subjects had BE Fundi WNL, while 62 (49.6%) had various degrees of diabetic retinopathy. Mild NPDR was observed in 39 (31.2%) subjects, moderate NPDR in 17 (13.6%), moderate PDR in 4 (3.2%), severe NPDR in 1 (0.8%), and RE mild NPDR in 1 (0.8%).

Figure 3: Distribution according to diabetic retinopathy.

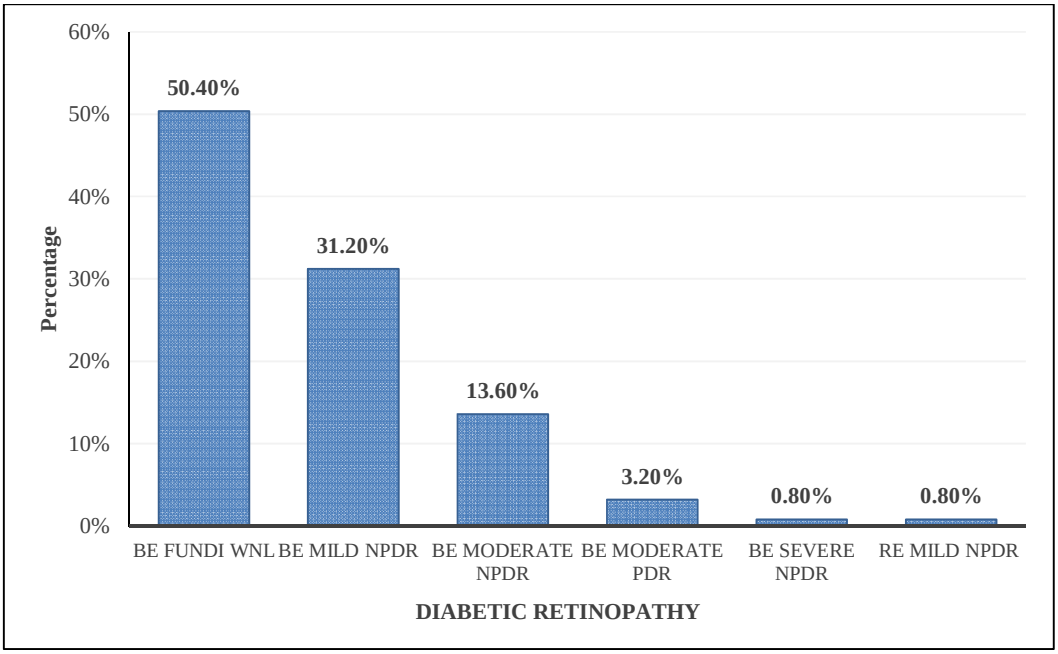

Table 9: Distribution according to Diabetic Neuropathy score.

Variables	Mean ± SD	Median (Min, Max)
DN Score	3.82 ± 2.94	2 (0, 9)

The mean DN (Diabetic Neuropathy) score was 3.82 ± 2.94, with a median of 2 (range: 0–9).

Table 10: Distribution according to microvascular complications.

Microvascular Complications	Sub Category	Number of subjects (%)
Diabetic Nephropathy	Absent	42 (33.6%)
	Present	83 (66.4%)
Diabetic Retinopathy	Absent	63 (50.4%)
	Present	62 (49.6%)
Diabetic Neuropathy	Absent	79 (63.2%)
	Present	46 (36.8%)

Microvascular complications were prevalent in the study cohort. Diabetic nephropathy was present in 83 (66.4%) subjects, diabetic retinopathy in 62 (49.6%), and diabetic neuropathy in 46 (36.8%).

Figure 4: Distribution according to microvascular complications.

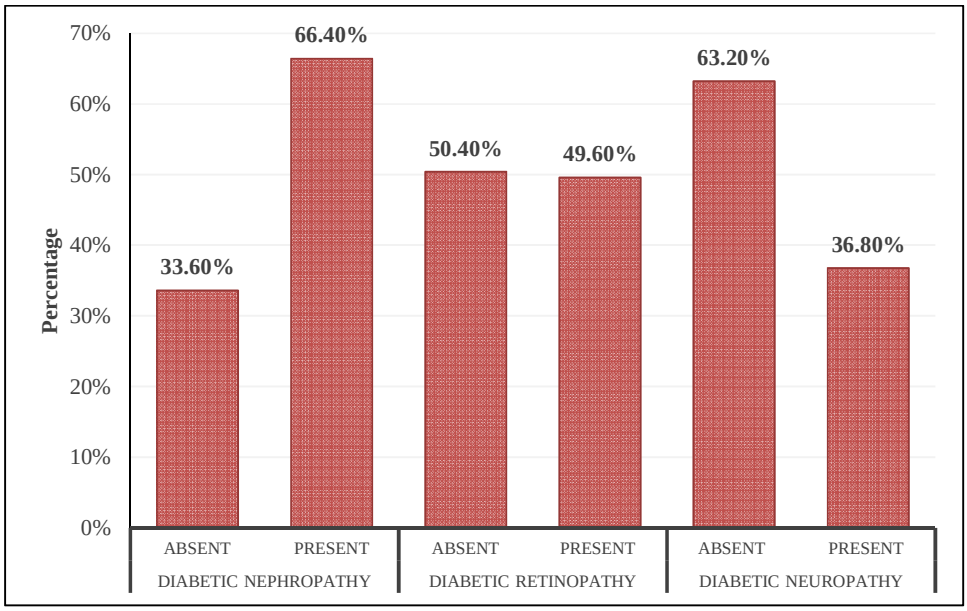

Table 11: Comparison of variables over diabetic retinopathy.

Variables	Sub Category	Diabetic Retinopathy		p-value
		Absent	Present	
Age (years)	Mean ± SD Median (Min, Max)	64.51 ± 12.43 66 (32, 98)	59.45 ± 12 60 (33, 86)	**0.0224[t]***
Gender	Female	27 (42.86%)	21 (33.87%)	0.3017[C]
	Male	36 (57.14%)	41 (66.13%)	
HBA1C	Mean ± SD Median (Min, Max)	7.9 ± 1.78 7.3 (5.5, 13.2)	8.56 ± 2.29 8 (4.9, 16.7)	0.1025[MW]
Hemoglobin	Mean ± SD Median (Min, Max)	11.52 ± 2.33 11.7 (6.5, 16.7)	12.04 ± 2.45 12.15 (6.9, 17)	0.2195[t]
RBC Count	Mean ± SD Median (Min, Max)	4.37 ± 0.8 4.5 (2.65, 6.03)	4.5 ± 0.95 4.52 (2.41, 6.53)	0.4063[t]
Hematocrit	Mean ± SD Median (Min, Max)	37.85 ± 6.96 38.8 (23.3, 53.7)	39.58 ± 7.29 39.75 (22.3, 55.9)	0.1778[t]
MCV	Mean ± SD Median (Min, Max)	86.94 ± 6.26 87.2 (66.9, 98.2)	89 ± 9.14 89.8 (64.8, 111.6)	0.0603[MW]
MCH	Mean ± SD Median (Min, Max)	26.43 ± 2.64 26.7 (16.5, 31.5)	27.05 ± 3.27 27.7 (18.1, 35.6)	0.0980[MW]
MCHC	Mean ± SD Median (Min, Max)	30.36 ± 1.89 30.6 (24, 36.7)	30.35 ± 1.52 30.6 (26.8, 33.3)	0.8939[MW]
RDW	Mean ± SD Median (Min, Max)	14.91 ± 2.46 14 (11.9, 22.6)	14.56 ± 2.15 14 (11.5, 23.9)	0.5615[MW]

TLC	Mean ± SD Median (Min, Max)	9.66 ± 4.9 8.15 (4.49, 33.14)	10.16 ± 6.19 8.59 (2.95, 32.93)	0.8280[MW]
Platelet Count	Mean ± SD Median (Min, Max)	281.08 ± 80.7 279 (113, 495)	216.6 ± 92.35 222.5 (14, 467)	**< 0.001[t]***
MPV	Mean ± SD Median (Min, Max)	9.53 ± 0.58 9.6 (8.3, 10.7)	11.08 ± 0.85 11.05 (9.6, 13.5)	**< 0.001[MW]***
PDW	Mean ± SD Median (Min, Max)	9.92 ± 1.18 9.8 (7.6, 12.7)	13.44 ± 2.28 12.75 (10.3, 20.5)	**< 0.001[MW]***
Plateletcrit	Mean ± SD Median (Min, Max)	0.25 ± 0.07 0.26 (0.1, 0.42)	0.23 ± 0.09 0.23 (0.01, 0.41)	0.2632[t]
Platelet LCR	Mean ± SD Median (Min, Max)	20.96 ± 4.62 21.4 (10.5, 30.4)	33.24 ± 6.48 32.25 (22.1, 48.5)	**< 0.001[MW]***
IPF	Mean ± SD Median (Min, Max)	3.32 ± 1.57 3.1 (1, 8.7)	7.46 ± 3.17 7.45 (2.1, 18.2)	**< 0.001[MW]***
T2DM Years	Mean ± SD Median (Min, Max)	2.46 ± 1.76 2 (0.16, 11)	7.82 ± 4.19 6 (0.1, 20)	**< 0.001[MW]***

*Abbreviation: "C – Chi square test, t – Two sample t test, MW – Mann Whitney U test, * indicates statistical significance."*

Subjects with diabetic retinopathy were younger on average (59.45 ± 12 years vs. 64.51 ± 12.43 years, with p-value = 0.0224) and had longer duration of Type 2 Diabetes Mellitus (7.82 ± 4.19 years vs. 2.46 ± 1.76 years, p-value < 0.001). Indices of platelet showed significant variation, with those having retinopathy exhibiting lower platelet counts (216.6 ± 92.35 vs. 281.08 ± 80.7, p-value < 0.001) and higher MPV (11.08 ± 0.85 vs. 9.53 ± 0.58, p-value < 0.001), higher PDW (13.44 ± 2.28 vs. 9.92 ± 1.18, p-value < 0.001), higher Platelet LCR (33.24 ± 6.48 vs. 20.96 ± 4.62, p-value < 0.001), and higher IPF (7.46 ± 3.17 vs. 3.32 ± 1.57, p-value < 0.001). Although not statistically significant, patients with retinopathy were observed to have higher levels of HbA1c.

There was no variation seen in gender, hemoglobin, RBC count, hematocrit, MCV, MCH, MCHC, RDW, TLC, or plateletcrit in those with and without retinopathy.

Figure 5: Mean plot of platelet counts over diabetic retinopathy.

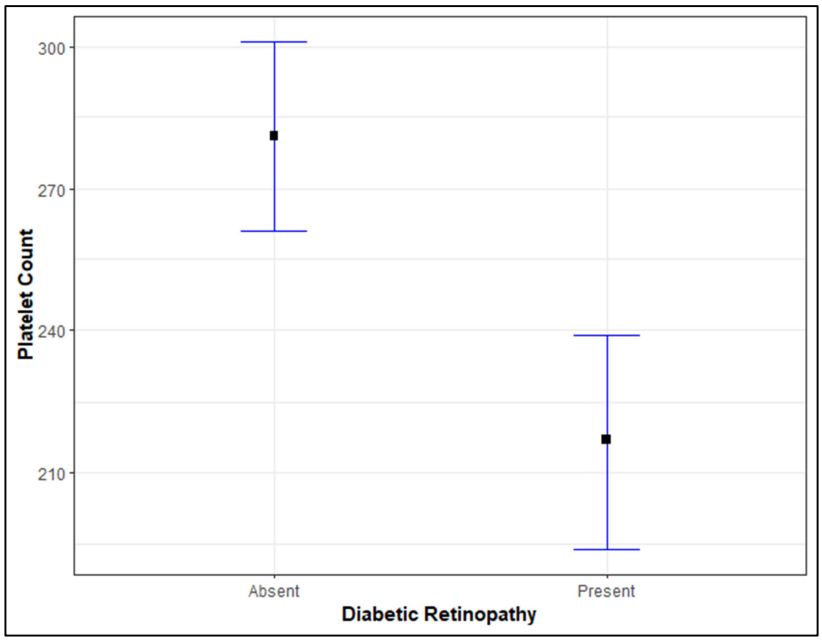

Figure 6: Mean plot of MPV over diabetic retinopathy.

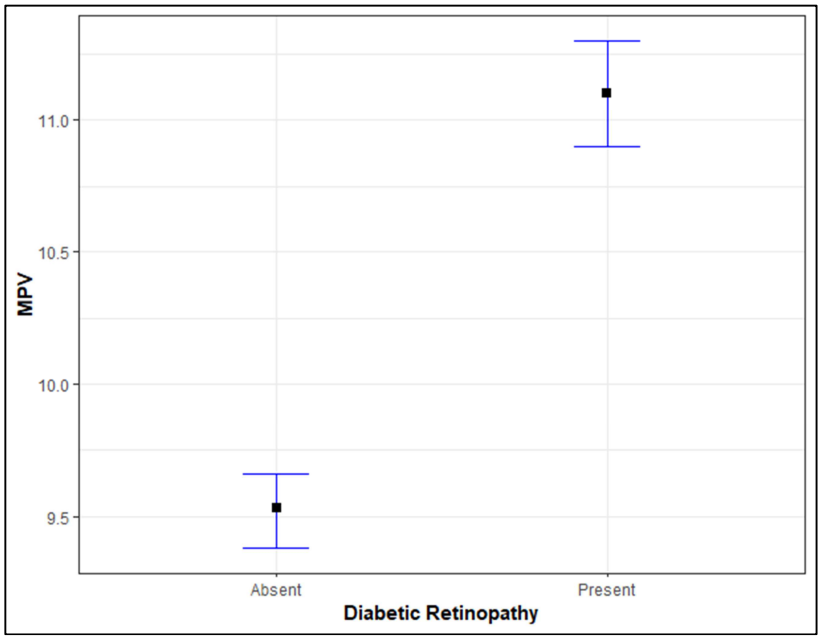

Figure 7: Mean plot of PDW over diabetic retinopathy.

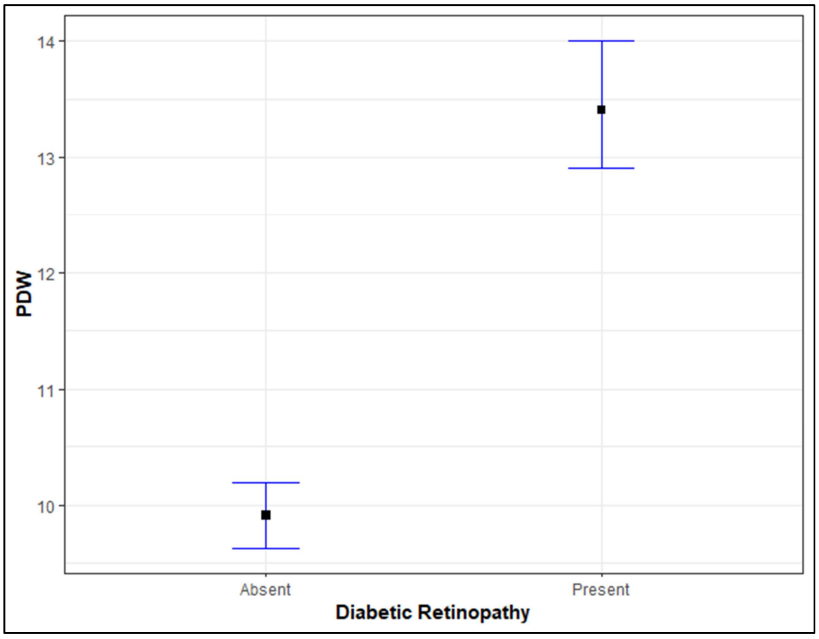

Figure 8: Mean plot of platelet LCR over diabetic retinopathy.

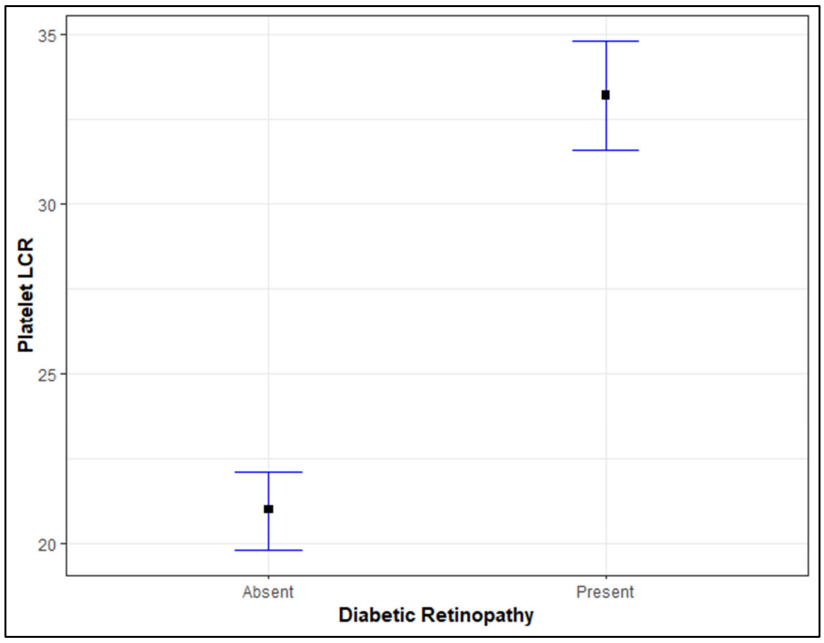

Figure 9: Mean plot of IPF over diabetic retinopathy.

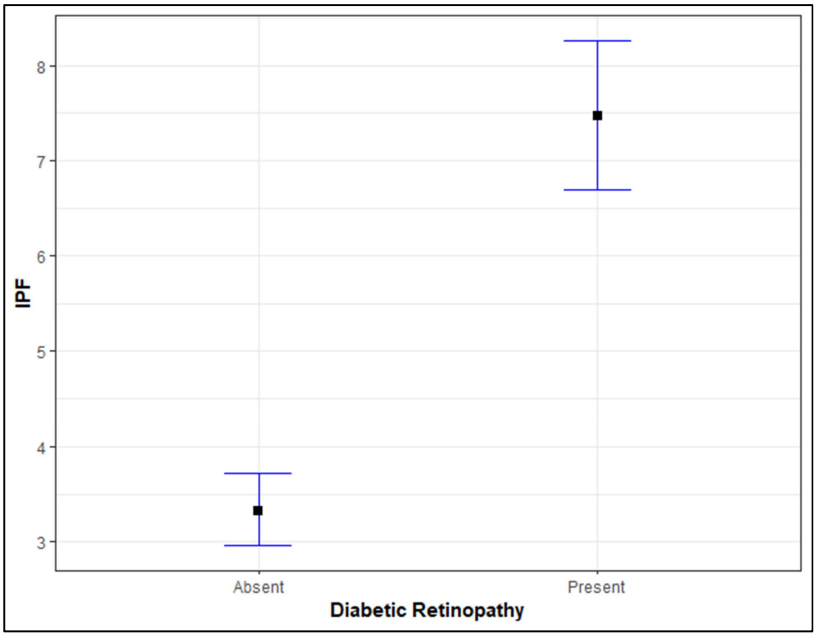

Figure 10: Mean plot of duration of T2DM over diabetic retinopathy.

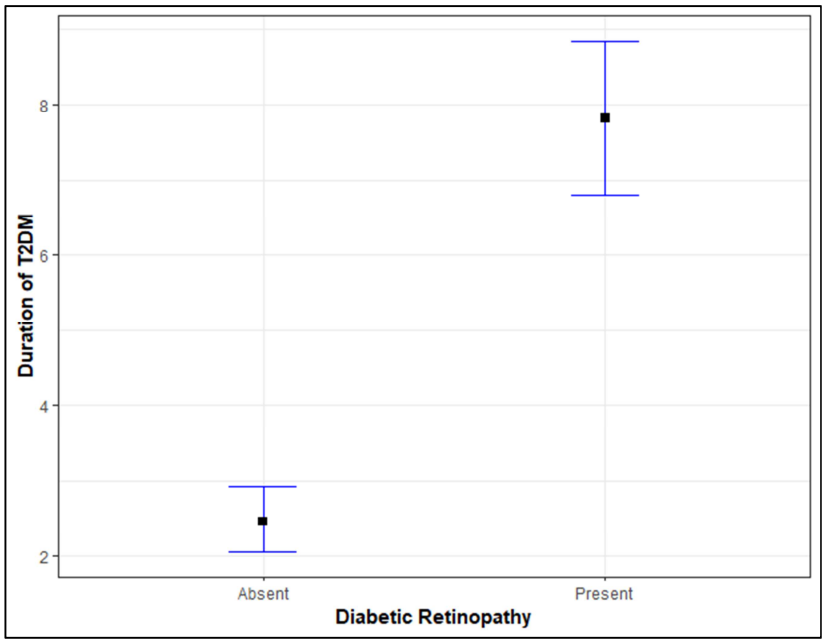

Table 12: Comparison of variables over diabetic neuropathy.

Variables	Sub Category	Diabetic Neuropathy		p-value
		Absent	Present	
Age (years)	Mean ± SD Median (Min, Max)	62.48 ± 12.63 63 (32, 98)	61.17 ± 12.18 63 (33, 86)	0.5728[t]
Gender	Female	36 (45.57%)	12 (26.09%)	**0.0308[C]***
	Male	43 (54.43%)	34 (73.91%)	
HBA1C	Mean ± SD Median (Min, Max)	7.99 ± 2.07 7.3 (5.3, 16.7)	8.63 ± 2.02 8.25 (4.9, 12.9)	0.0960[t]
Haemoglobin	Mean ± SD Median (Min, Max)	11.56 ± 2.34 11.8 (6.5, 16.9)	12.15 ± 2.47 12.3 (6.9, 17)	0.1807[t]
RBC Count	Mean ± SD Median (Min, Max)	4.41 ± 0.85 4.5 (2.65, 6.53)	4.46 ± 0.93 4.52 (2.41, 6.16)	0.7544[t]
Haematocrit	Mean ± SD Median (Min, Max)	38.11 ± 6.97 39 (23.3, 53.7)	39.73 ± 7.42 39.9 (22.3, 55.9)	0.2221[t]
MCV	Mean ± SD Median (Min, Max)	86.85 ± 7.71 87.2 (64.8, 106.6)	89.88 ± 7.83 90.45 (70.3, 111.6)	**0.0258[MW]***
MCH	Mean ± SD Median (Min, Max)	26.31 ± 2.92 26.7 (16.5, 34)	27.46 ± 2.96 28 (20, 35.6)	**0.0127[MW]***
MCHC	Mean ± SD	30.26 ± 1.79	30.52 ± 1.56	0.3204[MW]

	Median (Min, Max)	30.3 (24, 36.7)	30.7 (26.8, 33.3)	
RDW	Mean ± SD Median (Min, Max)	14.99 ± 2.54 14.4 (11.9, 23.9)	14.3 ± 1.78 13.9 (11.5, 19.7)	0.2039[MW]
TLC	Mean ± SD Median (Min, Max)	9.85 ± 5.25 8.31 (4.49, 33.14)	10.01 ± 6.12 8.59 (2.95, 32.93)	0.8719[MW]
Platelet Count	Mean ± SD Median (Min, Max)	274.28 ± 84.85 277 (61, 495)	205.85 ± 88.98 195.5 (14, 439)	**< 0.001[t]***
MPV	Mean ± SD Median (Min, Max)	9.73 ± 0.73 9.7 (8.3, 12.2)	11.27 ± 0.84 11.2 (9.5, 13.5)	**< 0.001[MW]***
PDW	Mean ± SD Median (Min, Max)	10.39 ± 1.5 10.3 (7.6, 15.7)	13.85 ± 2.43 13.3 (9.5, 20.5)	**< 0.001[MW]***
Plateletcrit	Mean ± SD Median (Min, Max)	0.25 ± 0.07 0.26 (0.07, 0.41)	0.23 ± 0.08 0.22 (0.01, 0.42)	0.1178[t]
Platelet LCR	Mean ± SD Median (Min, Max)	22.73 ± 5.8 22.5 (10.5, 41.8)	34.47 ± 6.62 33.7 (19.4, 48.5)	**< 0.001[t]***
IPF	Mean ± SD Median (Min, Max)	3.63 ± 1.67 3.2 (1, 8.7)	8.36 ± 3.12 8.15 (1.7, 18.2)	**< 0.001[MW]***
T2DM Years	Mean ± SD Median (Min, Max)	3.19 ± 2.5 2 (0.5, 15)	8.44 ± 4.41 10 (0.1, 20)	**< 0.001[MW]***

*Abbreviation:"C – Chi square test, t – Two sample t test, MW – Mann Whitney U test, * indicates statistical significance."*

Diabetic neuropathy was more prevalent among males (73.91% vs. 54.43% of males without neuropathy, p-value = 0.0308), longer duration of Diabetes (8.44 ± 4.41 years vs. 3.19 ± 2.5 years, p-value < 0.001).

Subjects with neuropathy had higher MCV (89.88 ± 7.83 vs. 86.85 ± 7.71, p-value = 0.0258) and MCH (27.46 ± 2.96 vs. 26.31 ± 2.92, p-value = 0.0127). Indices of platelet showed striking disparities, with those having neuropathy showing lower platelet counts (205.85 ± 88.98 vs. 274.28 ± 84.85, p-value < 0.001) and higher MPV (11.27 ± 0.84 vs. 9.73 ± 0.73, p-value < 0.001), higher PDW (13.85 ± 2.43 vs. 10.39 ± 1.5, p-value < 0.001), higher Platelet LCR (34.47 ± 6.62 vs. 22.73 ± 5.8, p-value < 0.001), and higher IPF (8.36 ± 3.12 vs. 3.63 ± 1.67, p-value < 0.001). It was observed that patients with neuropathy had higher levels of HbA1c.

There was no variation seen in age, hemoglobin levels, RBC count, hematocrit, MCHC, RDW, TLC, or plateletcrit between those with and without neuropathy.

Figure 11: Mean plot of platelet counts over diabetic neuropathy.

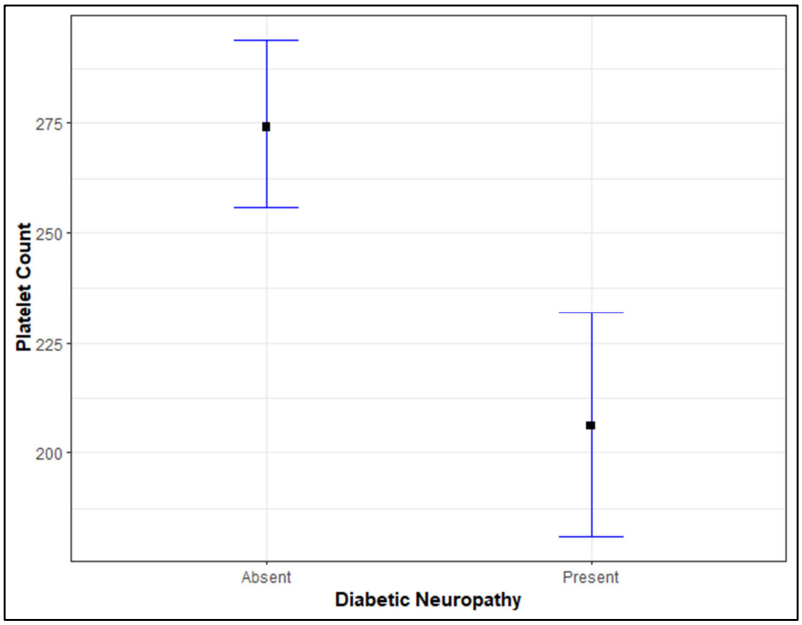

Figure 12: Mean plot of MPV over diabetic neuropathy.

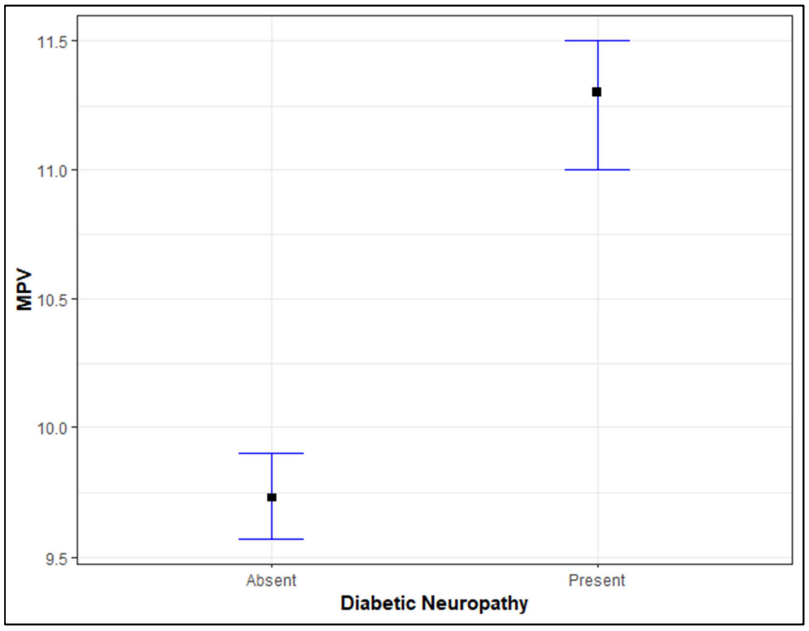

Figure 13: Mean plot of PDW over diabetic neuropathy.

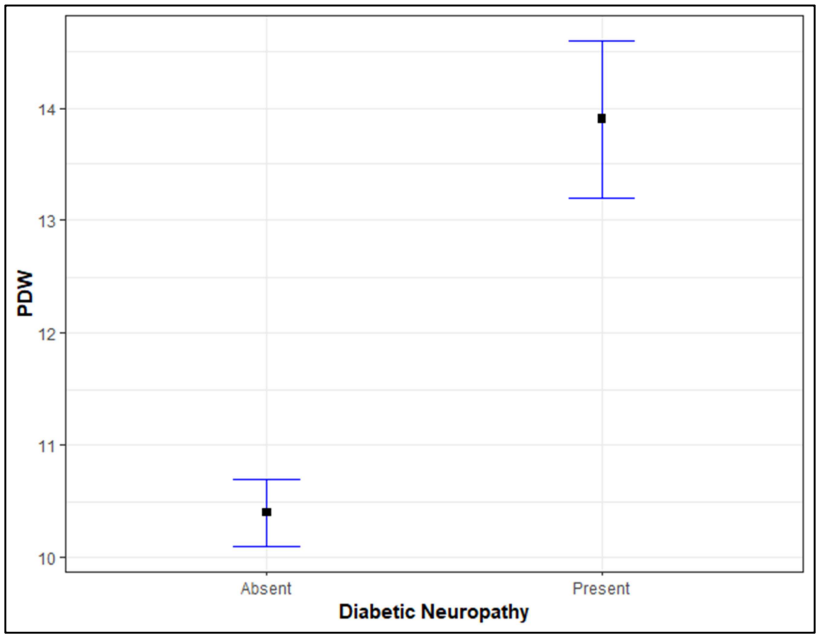

Figure 14: Mean plot of Platelet LCR over diabetic neuropathy.

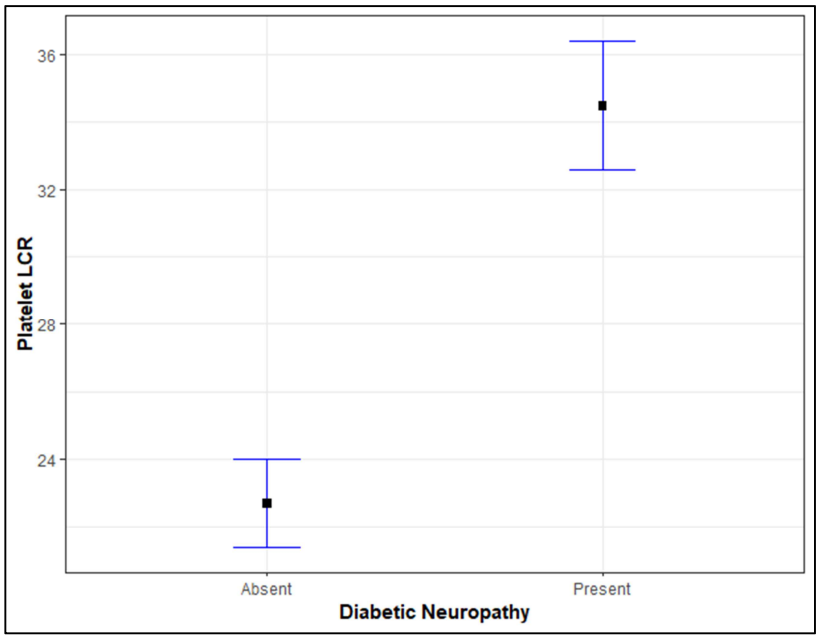

Figure 15: Mean plot of IPF over diabetic neuropathy.

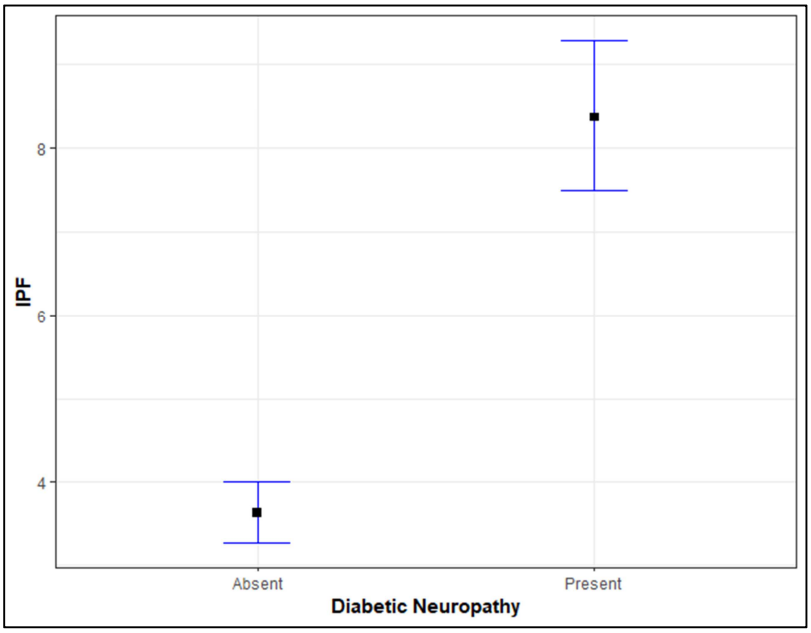

Figure 16: Mean plot of duration of T2DM over diabetic neuropathy.

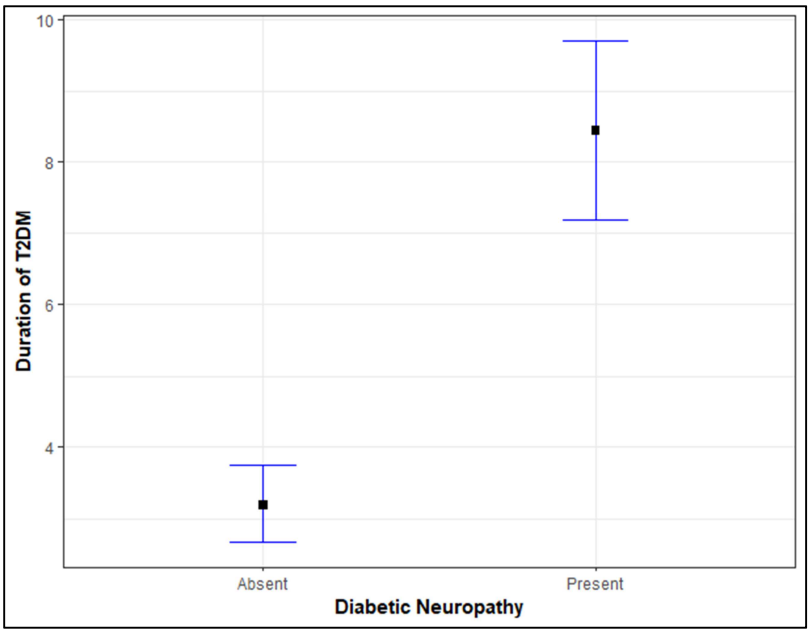

Table 13: Comparison of variables over diabetic nephropathy.

Variables	Sub Category	Diabetic Nephropathy		p-value
		Absent	Present	
Age (years)	Mean ± SD	59.19 ± 12.1	63.42 ± 12.43	0.0721[t]
	Median (Min, Max)	62 (33, 80)	64 (32, 98)	
Gender	Female	17 (40.48%)	31 (37.35%)	0.7342[C]
	Male	25 (59.52%)	52 (62.65%)	
HBA1C	Mean ± SD	7.89 ± 1.85	8.4 ± 2.16	0.2332[MW]
	Median (Min, Max)	7.45 (4.9, 12.9)	7.6 (5.5, 16.7)	
Hemoglobin	Mean ± SD	12.25 ± 2.16	11.54 ± 2.48	0.1190[t]
	Median (Min, Max)	12.3 (6.5, 17)	11.9 (6.6, 17)	
RBC Count	Mean ± SD	4.55 ± 0.69	4.37 ± 0.95	0.2761[t]
	Median (Min, Max)	4.54 (2.72, 5.94)	4.5 (2.41, 6.53)	
Hematocrit	Mean ± SD	40.18 ± 6.32	37.97 ± 7.46	0.1028[t]
	Median (Min, Max)	40.3 (23.3, 52.8)	39.1 (22.3, 55.9)	
MCV	Mean ± SD	88.33 ± 5.36	87.78 ± 8.89	0.7918[MW]
	Median (Min, Max)	88.85 (73.4, 98)	88.9 (64.8, 111.6)	
MCH	Mean ± SD	26.9 ± 2.44	26.65 ± 3.22	0.8405[MW]
	Median (Min, Max)	27.1 (20.1, 31.5)	27.3 (16.5, 35.6)	
MCHC	Mean ± SD	30.43 ± 1.84	30.32 ± 1.65	0.9229[MW]
	Median (Min, Max)	30.55 (26.8, 36.7)	30.6 (24, 33.5)	
RDW	Mean ± SD	14.23 ± 1.86	15 ± 2.48	0.1027[MW]
	Median (Min, Max)	13.9 (11.5, 20.3)	14.4 (11.9, 23.9)	
TLC	Mean ± SD	9.97 ± 5.92	9.88 ± 5.4	0.8898[MW]

	Median (Min, Max)	8.23 (4.91, 32.93)	8.44 (2.95, 33.14)	
Platelet Count	Mean ± SD Median (Min, Max)	265.4 ± 88.67 280 (14, 495)	240.84 ± 93.37 234 (36, 467)	0.1603^{t}
MPV	Mean ± SD Median (Min, Max)	10.08 ± 1.12 9.75 (8.3, 13.2)	10.41 ± 1.03 10.2 (8.3, 13.5)	0.0549^{MW}
PDW	Mean ± SD Median (Min, Max)	11.07 ± 2.47 10.3 (7.6, 20.5)	11.97 ± 2.51 11.8 (7.7, 20.4)	$\mathbf{0.0197^{MW}*}$
Plateletcrit	Mean ± SD Median (Min, Max)	0.25 ± 0.08 0.25 (0.01, 0.41)	0.24 ± 0.08 0.23 (0.09, 0.42)	0.4493^{t}
Platelet LCR	Mean ± SD Median (Min, Max)	25.35 ± 8.81 22.7 (10.5, 48.5)	27.91 ± 7.99 27.2 (11.9, 47.9)	0.0544^{MW}
IPF	Mean ± SD Median (Min, Max)	4.75 ± 3.27 3.4 (1.3, 18.2)	5.69 ± 3.2 5.3 (1, 16.5)	0.0634^{MW}
T2DM Years	Mean ± SD Median (Min, Max)	4.04 ± 3.38 3 (0.5, 15)	5.67 ± 4.44 5 (0.1, 20)	0.0508^{MW}

*Abbreviation: "C – Chi square test, t – Two sample t test, MW – Mann Whitney U test, * indicates statistical significance."*

There was a significant difference in PDW, which was higher and statistically significant in those with nephropathy (p-value = 0.0197). Although not statistically significant, we observed higher levels of HbA1c in patients with nephropathy.

No significant differences (p-value > 0.05) were observed for age, gender (p-value = 0.7342), hemoglobin levels, RBC count, hematocrit, MCV, MCH, MCHC, RDW, TLC , platelet count, MPV, plateletcrit, platelet LCR, IPF.

Figure 17: Mean plot of PDW over diabetic nephropathy.

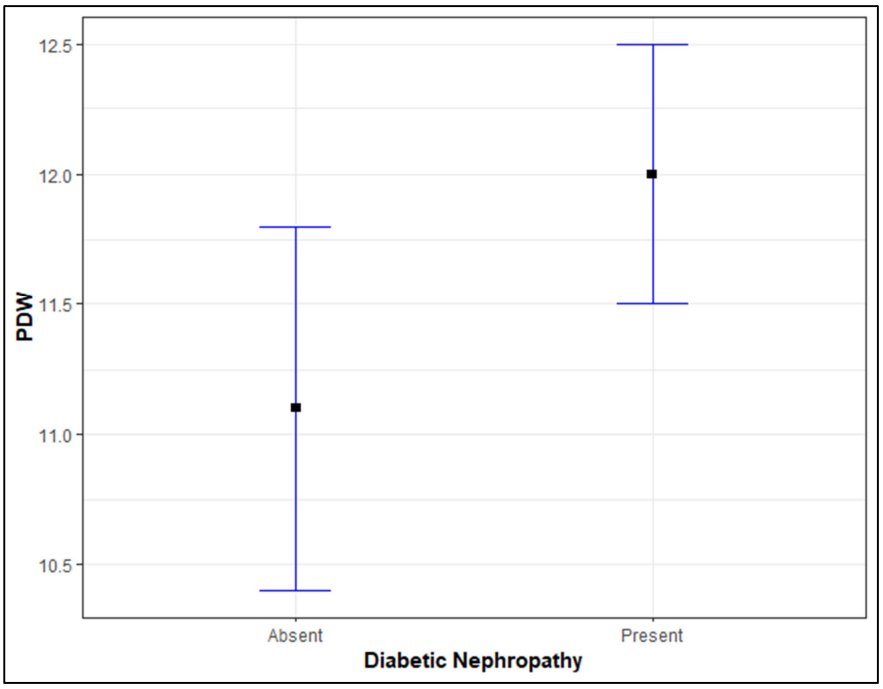

Table 14: Correlation of indices of platelet with HbA1c.

Platelet indices	HBA1C		p-value
	< 7	≥ 7	
Platelet Count	266.12 ± 98.97 267 (36, 495)	240.17 ± 87.74 241 (14, 467)	0.1356[t]
MPV	10.18 ± 1.16 10 (8.3, 13.5)	10.36 ± 1.02 10.2 (8.3, 13.2)	0.3767[t]
PDW	11.32 ± 2.72 10.8 (7.6, 20.4)	11.85 ± 2.41 11.4 (8.3, 20.5)	0.1635[MW]
Plateletcrit	0.25 ± 0.07 0.26 (0.07, 0.39)	0.24 ± 0.08 0.23 (0.01, 0.42)	0.2489[t]
Platelet LCR	25.84 ± 9.01 25.5 (10.5, 47.9)	27.69 ± 7.93 26.5 (14.4, 48.5)	0.2210[MW]
IPF	4.99 ± 3.3 4.1 (1, 14.4)	5.57 ± 3.21 5.05 (1.3, 18.2)	0.2102[MW]

Abbreviation: "t – Two sample t test, MW – Mann Whitney U test."

None of the platelet indices showed a statistically significant difference between the two HbA1c groups (p-values > 0.05). However, higher values of MPV, PDW, Platelet LCR, IPF were seen with HbA1c ≥ 7.

DISCUSSION

Our research provides an in-depth evaluation of indices of platelet in patients with T2DM, focusing on their relation with microvascular complications. These findings emphasize the potential of indices of platelet as markers for identifying and monitoring microvascular complications in T2DM.

The study observed that patients with early onset and longer duration of diabetes, had diabetic retinopathy, potentially reflecting the early onset of retinal damage in susceptible individuals. This variability considers age as potential modifier in the development of complications and the role of early screening and appropriate intervention especially in younger populations with poor glycemic control. We also observed patients with longer duration of diabetes had nephropathy and neuropathy.

Gender distribution revealed a male predominance in the study cohort, with men constituting 61.6% of the participants. Diabetic neuropathy was more common in males, highlighting potential gender-specific differences in the pathophysiology or risk factors associated with complications.[63] While the overall influence of gender on indices of platelet was not statistically significant, these findings suggest that further investigation into gender-related disparities in diabetes complications is warranted.

Hematological parameters, including hemoglobin levels, RBC count, and hematocrit, were evaluated as part of the study. Although no differences were seen in these parameters between patients with and without microvascular complications, subtle trends were noted. For example, patients with diabetic nephropathy had lower hemoglobin and hematocrit levels on average, possibly reflecting underlying anemia commonly associated with CKD. Additionally, higher MCV and MCH were observed in patients with neuropathy, suggesting alterations in erythrocyte indices that may

warrant further exploration. These findings, though not statistically significant, provide a broader context for understanding the systemic impact of T2DM and its complications.

The study revealed that patients who were having diabetic retinopathy had significantly higher levels of MPV, PDW, P-LCR, an IPF compared to those without retinopathy. Venkata et al. conducted a study that involved 100 diabetic patients who were from outpatient (OPD) and inpatient (IPD) settings. They also found higher MPV (0.001*), higher Plateletcrit (0.001*), PLCR(0.13), and PDW(0.067).[64] These findings highlight the heightened platelet activation and turnover in diabetic retinopathy, which likely contribute to retinal microvascular damage through mechanisms such as endothelial dysfunction, thrombus formation, and inflammation.[65] The marked elevation of these indices aligns with the pathophysiological changes observed in diabetic retinopathy, where chronic hyperglycemia induces oxidative stress and vascular injury, leading to capillary occlusion and neovascularization.[66] Such changes emphasize the role of dysfunction of platelets in the pathophysiology of retinopathy and its utility in monitoring disease severity.

A comparable trend was noted in individuals with diabetic neuropathy, in whom significantly higher levels of MPV, PDW, and P -LCR, along with a prolonged duration of diabetes. Qian et al showed that T2DM patients who experienced neuropathy had significantly lower platelet count (PLT) and plateletcrit (PCT) compared to healthy individuals, with a statistical significance of $P < 0.05$. The research revealed meaningful correlations between decreased PLT and PCT levels and a reduction in the amplitude and velocity Z-score, along with an increase in F-wave latency during nerve conduction evaluations. Additionally, lower PLT levels “(odds

ratio 2.268, 95% confidence interval 1.072-4.797; P < 0.05; PLT < 226 vs PLT ≥ 226) and PCT levels (odds ratio 2.050, 95% confidence interval 1.001-4.201; P < 0.05; PCT < 0.222 vs PCT ≥ 0.222)" in patients with T2DM were more prone for neuropathy. The duration of diabetes was also considered in this context.[67] Papanas et al. found that there was no observed association between mean platelet volume (MPV) and the severity of neuropathy in individuals with T2DM. Diabetes is known to disrupt various metabolic and vascular mechanisms, which lead to diabetic neuropathy. While raised MPV is associated with the presence of neuropathy, it does not correlate with its severity in patients with T2DM.[68] These findings suggest that prolonged exposure to hyperglycemia exacerbates platelet activation and contributes to microvascular ischemia, which plays a central part in the development of neuropathy. The relation between indices of platelet and neuropathy underscores the potential of these markers to identify patients at greater risk for nerve damage.[69] Furthermore, the higher Immature Platelet Fraction (IPF) in these patients may indicate increased bone marrow activity in response to ongoing vascular damage, reflecting the dynamic nature of platelet turnover in diabetic complications.[70]

Diabetic nephropathy presented a more nuanced relationship with platelet indices. While PDW was higher in patients with nephropathy, other indices such as MPV, P-LCR, and IPF did not show significant differences. The study population described by Sengupta et al. had a mean age of 61.7 ± 12.0 years. A significant portion, 62.1%, of the participants was over 60 years old. The average duration of diabetes among the participants was 8.0 ± 5.2 years. The mean platelet count was recorded at 236.4, with a standard deviation of 112.6. Additionally, the average values for various indices of platelet were as follows: MPV was 11.4 (± 1.7), PDW was 15.2 (± 3.8), PCT was 0.28 (± 0.11), and PLCR was 38.9 (± 11.8). Most participants

(63.1%) were in the early stages of nephropathy (stages 1-3). Significant differences in indices of platelet were found across the various stages of nephropathy, with Kruskal-Wallis p-values of 0.027 for MPV, 0.009 for PDW, 0.001 for PCT, and 0.007 for PLCR.[71] This finding suggests that platelet activation may contribute to kidney damage through mechanisms distinct from those in retinopathy or neuropathy. The absence of significant differences in other indices could also reflect the multifactorial nature of nephropathy, where factors such as hypertension, albuminuria, and systemic inflammation play pivotal roles.[72] Nevertheless, the elevated PDW highlights its potential as a marker to detect nephropathy earlier, particularly in identifying subtle changes in platelet heterogeneity linked to vascular injury. Wei et al study concluded that patients suffering from DKD had higher levels of PCT compared to the patients who were not suffering from DKD. Additionally it suggested that increased PCT and platelet count could act as risk factors independently for DKD and the urinary albumin-to-creatinine ratio (UACR).[73] "Taderegew et al. reported that 33.2% of the participants were experiencing at least one microvascular complication. The MPV , PDW , and P-LCR were higher in patients with complications compared to those without complications." All differences were significant.[74]

In our study although not statistically significant we found higher levels of indices of platelet i.e MPV, PDW, Platelet LCR and IPF in patients with HbA1c ≥ 7. The lack of correlation in this cohort could be attributed to population-specific factors, variations in sample size, or confounding variables such as medication use and co-morbid conditions. Despite this, the significant differences in indices of platelet among patients with microvascular complications reaffirm the role of chronic hyperglycemia in promoting platelet dysfunction, even if not directly reflected in HbA1c levels. This finding highlights the complexity of glycemic control and its

interplay with indices of platelet suggesting that these indices may reflect cumulative damage rather than immediate glycemic status.

The study's findings are in line with those of Khanna et al. (2024) and Saha et al., who reported elevated MPV, PDW, and P-LCR in diabetic populations, particularly in those with complications. Such consistency reinforces the role of indices of platelet as reliable markers for vascular risk.(59,60) Additionally, the inclusion of an Indian cohort in this study addresses a critical gap in the literature, as regional differences in genetics, diet, and socio-economic factors can significantly influence the prevalence and progression of T2DM and its complications. By focusing on an Indian population, this study provides valuable insights that could inform tailored screening and management strategies.

The implications of these findings are significant. Indices of platelet are cost-effective and readily available through routine hematological analyses, making them practical tools for early detection of diabetic complications. Integrating these into routine diabetes care could enable timely interventions, such as intensified glycemic control, antiplatelet therapy, or lifestyle modifications, to mitigate the progression of complications.

Future research should address these limitations through longitudinal studies that track changes in indices of platelet over time and their relationship with microvascular complications. Exploring the underlying mechanisms linking platelet-activation to vascular damage could provide deeper insights into the pathophysiology of diabetic complications and identify novel therapeutic targets. Furthermore, studies involving larger and more diverse populations would enhance the generalizability of findings and support the development of population-specific reference ranges for platelet indices.

Strengths

1. The study focuses on indices of platelet in an Indian cohort (North Karnataka) , addressing a significant gap in regional diabetes research.
2. Comprehensive evaluation of microvascular complications, provides valuable insights into the role of platelet dysfunction in T2DM.
3. Use of routine, cost-effective hematological parameters offers practical implementation in treatment of T2DM and prevention of microvascular complications.

Limitations

1. Small Sample size
2. Study design - Cross sectional study.
3. To get better outcomes, a prospective follow-up study should be done to identify the change in platelet indices with aggressive blood glucose control.

CONCLUSION

This study highlights the significant association between elevated indices of platelet and microvascular complications in T2DM, with MPV, PDW, and P-LCR emerging as potential markers for early detection and risk stratification. These findings underscore the value of integrating indices of platelet into routine diabetes care to improve outcomes for individuals living with this chronic condition. By addressing existing gaps through further research, the potential of indices of platelet as non-invasive, cost-effective tools for managing diabetes-related complications can be fully realized, ultimately reducing the load of microvascular complications.

SUMMARY

This is a cross sectional study carried out at a Tertiary Care Hospital which involved 125 patients diagnosed with T2DM. The objective of the study is to correlate the indices of platelet with microvascular complications and HbA1c.

The results of our study were as follows:

- Subjects with diabetic retinopathy were younger on average and had a longer duration of Type 2 Diabetes Mellitus. Indices of platelet showed statistically significant variation ($P < 0.01$), with those having retinopathy exhibiting lower platelet counts and higher MPV, higher PDW, higher Platelet LCR and higher IPF.
- Subjects with diabetic neuropathy were more prevalent among males and with longer duration of Diabetes. Indices of platelet showed statistically significant variation ($P < 0.01$), with those having neuropathy showing lower platelet counts, higher MPV, higher PDW , higher Platelet LCR, and higher IPF.
- Subjects with diabetic nephropathy had statistically significant higher PDW ($P < 0.01$). We found out that patients having nephropathy had higher values of MPV (P - 0.054), higher Platelet LCR (P - 0.054), higher IPF (P – 0.06) and longer duration of T2DM but was not statistically significant.
- In our study we found higher levels of MPV, PDW, Platelet LCR and IPF in patients with HbA1c ≥ 7, although was not statistically significant.

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BIBLIOGRAPHY

1. Type 2 Diabetes - StatPearls - NCBI Bookshelf [Internet]. [cited 2025 Jan 18]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK513253/
2. Jha RP, Shri N, Patel P, Dhamnetiya D, Bhattacharyya K, Singh M. Trends in the diabetes incidence and mortality in India from 1990 to 2019: a joinpoint and age-period-cohort analysis. J Diabetes Metab Disord. 2021 Jul 5;20(2):1725–40.
3. Epidemiology of Diabetes and Diabetes-Related Complications - PMC [Internet]. [cited 2025 Jan 18]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3870323/
4. Tomic D, Shaw JE, Magliano DJ. The burden and risks of emerging complications of diabetes mellitus. Nat Rev Endocrinol. 2022 Sep;18(9): 525–39.
5. Platelet Function in Patients with Diabetes Mellitus: From a Theoretical to a Practical Perspective - PMC [Internet]. [cited 2025 Jan 18]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3159301/
6. Senthil Nathan S, Varadaraj P, Nallusamy G, Reddy KSS. The Significance of Platelet Indices in the Evaluation of Thrombocytopenia. Cureus. 16(7):e65756.
7. Alhadas KR, Santos SN, Freitas MMS, Viana SMSA, Ribeiro LC, Costa MB. Are platelet indices useful in the evaluation of type 2 diabetic patients? J Bras Patol E Med Lab. 2016 Apr 26;52:96–102.
8. Shilpi K, Potekar RM. A Study of Platelet Indices in Type 2 Diabetes Mellitus Patients. Indian J Hematol Blood Transfus Off J Indian Soc Hematol Blood Transfus. 2018 Jan;34(1):115–20.

9. Association of platelet parameters with chronic complications in type 2 diabetes mellitus - IJPO [Internet]. [cited 2025 Jan 18]. Available from: https://www.ijpo.co.in/html-article/22756
10. Stumvoll M, Goldstein BJ, van Haeften TW. Type 2 diabetes: principles of pathogenesis and therapy. Lancet Lond Engl. 2005 Apr 9;365(9467):1333–46.
11. The natural history of insulin secretory dysfunction and insulin resistance in the pathogenesis of type 2 diabetes mellitus - PubMed [Internet]. [cited 2025 Jan 22]. Available from: https://pubmed.ncbi.nlm.nih.gov/10491414/
12. Chatterjee S, Khunti K, Davies MJ. Type 2 diabetes. Lancet Lond Engl. 2017 Jun 3;389(10085):2239–51.
13. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4.4 million participants. Lancet Lond Engl. 2016 Apr 9;387(10027):1513–30.
14. From the Triumvirate to the Ominous Octet: A New Paradigm for the Treatment of Type 2 Diabetes Mellitus - PMC [Internet]. [cited 2025 Jan 22]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC2661582/
15. Schwartz SS, Epstein S, Corkey BE, Grant SFA, Gavin JR, Aguilar RB. The Time Is Right for a New Classification System for Diabetes: Rationale and Implications of the β-Cell-Centric Classification Schema. Diabetes Care. 2016 Feb;39(2):179–86.
16. Gaede P, Vedel P, Larsen N, Jensen GVH, Parving HH, Pedersen O. Multifactorial intervention and cardiovascular disease in patients with type 2 diabetes. N Engl J Med. 2003 Jan 30;348(5):383–93.
17. Grarup N, Sandholt CH, Hansen T, Pedersen O. Genetic susceptibility to type 2 diabetes and obesity: from genome-wide association studies to rare variants and beyond. Diabetologia. 2014 Aug;57(8):1528–41.

18. Wong ND, Zhao Y, Patel R, Patao C, Malik S, Bertoni AG, et al. Cardiovascular Risk Factor Targets and Cardiovascular Disease Event Risk in Diabetes: A Pooling Project of the Atherosclerosis Risk in Communities Study, Multi-Ethnic Study of Atherosclerosis, and Jackson Heart Study. Diabetes Care. 2016 May;39(5):668–76.
19. Diabetes Control and Complications Trial Research Group, Nathan DM, Genuth S, Lachin J, Cleary P, Crofford O, et al. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med. 1993 Sep 30;329(14): 977–86.
20. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK Prospective Diabetes Study (UKPDS) Group. Lancet Lond Engl. 1998 Sep 12;352(9131):837–53.
21. Vithian K, Hurel S. Microvascular complications: pathophysiology and management. Clin Med. 2010 Oct;10(5):505–9.
22. Vascular complications of diabetes: mechanisms of injury and protective factors - PMC [Internet]. [cited 2025 Jan 22]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3546345/
23. Chronic hyperglycemia mediated physiological alteration and metabolic distortion leads to organ dysfunction, infection, cancer progression and other pathophysiological consequences: An update on glucose toxicity - ScienceDirect [Internet]. [cited 2025 Jan 22]. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0753332218322406

24. Shukla UV, Tripathy K. Diabetic Retinopathy. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2025 Jan 22]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK560805/

25. Macular Edema - StatPearls - NCBI Bookshelf [Internet]. [cited 2025 Jan 22]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK576396/

26. Bain SC, Klufas MA, Ho A, Matthews DR. Worsening of diabetic retinopathy with rapid improvement in systemic glucose control: A review. Diabetes Obes Metab. 2019 Mar;21(3):454–66.

27. Diabetic Nephropathy - StatPearls - NCBI Bookshelf [Internet]. [cited 2025 Jan 22]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK534200/

28. Weir MR. Microalbuminuria in Type 2 Diabetics: An Important, Overlooked Cardiovascular Risk Factor. J Clin Hypertens. 2007 May 25;6(3):134–43.

29. Microalbuminuria - StatPearls - NCBI Bookshelf [Internet]. [cited 2025 Jan 22]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK563255/

30. Zelnick LR, Weiss NS, Kestenbaum BR, Robinson-Cohen C, Heagerty PJ, Tuttle K, et al. Diabetes and CKD in the United States Population, 2009-2014. Clin J Am Soc Nephrol CJASN. 2017 Dec 7;12(12):1984–90.

31. Tuttle KR, Bakris GL, Bilous RW, Chiang JL, de Boer IH, Goldstein-Fuchs J, et al. Diabetic kidney disease: a report from an ADA Consensus Conference. Diabetes Care. 2014 Oct;37(10):2864–83.

32. Diabetic neuropathy - PMC [Internet]. [cited 2025 Jan 22]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7096070/

33. Anandhanarayanan A, Teh K, Goonoo M, Tesfaye S, Selvarajah D. Diabetic Neuropathies. In: Feingold KR, Anawalt B, Blackman MR, Boyce A, Chrousos G, Corpas E, et al., editors. Endotext [Internet]. South Dartmouth (MA):

MDText.com, Inc.; 2000 [cited 2025 Jan 22]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK279175/

34. Platelets - PubMed [Internet]. [cited 2023 Dec 28]. Available from: https://pubmed.ncbi.nlm.nih.gov/10801186/
35. Pogorzelska K, Krętowska A, Krawczuk-Rybak M, Sawicka-Żukowska M. Characteristics of platelet indices and their prognostic significance in selected medical condition – a systematic review. Adv Med Sci. 2020 Sep 1;65(2): 310–5.
36. Mean Platelet Volume (MPV): New Perspectives for an Old Marker in the Course and Prognosis of Inflammatory Conditions - PMC [Internet]. [cited 2025 Jan 22]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC6501263/
37. Large platelet size is associated with poor outcome in patients with metastatic pancreatic cancer - PubMed [Internet]. [cited 2025 Jan 22]. Available from: https://pubmed.ncbi.nlm.nih.gov/30307891/
38. Tzur I, Barchel D, Izhakian S, Swarka M, Garach-Jehoshua O, Krutkina E, et al. Platelet distribution width: a novel prognostic marker in an internal medicine ward. J Community Hosp Intern Med Perspect. 2019 Dec 14;9(6):464–70.
39. Definition of reference ranges for the platelet distribution width (PDW): a local need - PubMed [Internet]. [cited 2025 Jan 22]. Available from: https://pubmed.ncbi.nlm.nih.gov/19958208/
40. The use of platelet indices, plateletcrit, mean platelet volume and platelet distribution width in emergency non-traumatic abdominal surgery: a systematic review - PubMed [Internet]. [cited 2025 Jan 22]. Available from: https://pubmed.ncbi.nlm.nih.gov/27346963/

41. Ulkumen BA, Pala HG, Calik E, Koltan SO. Platelet distribution width (PDW): A putative marker for threatened preterm labour. Pak J Med Sci. 2014 Aug;30(4):745.

42. Aydogan A, Akkucuk S, Arica S, Motor S, Karakus A, Ozkan OV, et al. The Analysis of Mean Platelet Volume and Platelet Distribution Width Levels in Appendicitis. Indian J Surg. 2015 Dec;77(Suppl 2):495–500.

43. Babu E, Basu D. Platelet large cell ratio in the differential diagnosis of abnormal platelet counts. Indian J Pathol Microbiol. 2004 Apr;47(2):202–5.

44. Grotto HZW, Noronha JFA. Platelet larger cell ratio (P-LCR) in patients with dyslipidemia. Clin Lab Haematol. 2004 Oct;26(5):347–9.

45. Baig MA. Platelet indices- evaluation of their diagnostic role in pediatric thrombocytopenias (one year study). Int J Res Med Sci. 2015;3(9):2284–9.

46. Budak YU, Polat M, Huysal K. The use of platelet indices, plateletcrit, mean platelet volume and platelet distribution width in emergency non-traumatic abdominal surgery: a systematic review. Biochem Medica. 2016 Jun 10;26(2):178–93.

47. Tang J, Gao X, Zhi M, Zhou HM, Zhang M, Chen HW, et al. Plateletcrit: a sensitive biomarker for evaluating disease activity in Crohn's disease with low hs-CRP. J Dig Dis. 2015 Mar;16(3):118–24.

48. Grabek J, D'Elia N, Kelsey G. Immature platelet fraction as a predictor of platelet count recovery following allogeneic bone marrow transplantation. Pathology (Phila). 2021 Jun;53(4):493–7.

49. Taderegew MM, Woldeamanuel GG, Emeria MS, Tilahun M, Yitbarek GY, Zegeye B. Platelet Indices and Its Association with Microvascular Complications Among Type 2 Diabetes Mellitus Patients in Northeast Ethiopia:

A Cross-Sectional Study. Diabetes Metab Syndr Obes Targets Ther. 2021 Feb 25;14:865–74.

50. Paul A, Das S, Bose K, Karmakar R. Platelet Indices: Predicting Retinopathy in Patients with Type 2 Diabetes Mellitus. Med J Dr Patil Vidyapeeth. 2024 Nov;17(6):1231–5.
51. Khanna P, Salwan SK, Sharma A, Jr PK, Salwan SK, Sharma A. Correlation of Platelet Indices in Patients With Type 2 Diabetes Mellitus and Associated Microvascular Complications: A Hospital-Based, Prospective, Case-Control Study. Cureus [Internet]. 2024 Mar 11 [cited 2025 Jan 22];16(3). Available from: https://www.cureus.com/articles/232145-correlation-of-platelet-indices-in-patients-with-type-2-diabetes-mellitus-and-associated-microvascular-complications-a-hospital-based-prospective-case-control-study
52. Gamage V, Mohotti M, Wickramaratne K. Differences in platelet indices between type-2 diabetes mellitus patients with and without microvascular complications at Teaching Hospital, Karapitiya. Sri Lanka J Haematol. 2024 Apr 24;15:19–26.
53. Sangapur SM, S DR, Sharanabasappa, Meena B. Platelet indices and their correlation with HbA1c and association with microvascular complications in type-2 diabetes mellitus. Int J Res Med Sci. 2023 Apr 29;11(5):1594–9.
54. Hemoglobin A1C - StatPearls - NCBI Bookshelf [Internet]. [cited 2025 Jan 22]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK549816/
55. Significance of HbA1c Test in Diagnosis and Prognosis of Diabetic Patients - PMC [Internet]. [cited 2025 Jan 22]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC4933534/

56. Aktas F, Aktuglu MB. Evaluation of the relation between HBA1C and MPV, PDW levels of patients with Type 2 diabetes admitted in internal medicine polyclinics. North Clin Istanb. 2023 Sep 27;10(5):681–6.

57. Factors Contributing to Increased Platelet Reactivity in People With Diabetes - PMC [Internet]. [cited 2025 Jan 22]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC2660482/

58. Full article: Platelet indices in diabetes mellitus: indicators of diabetic microvascular complications [Internet]. [cited 2025 Jan 22]. Available from: https://www.tandfonline.com/doi/full/10.1179/102453311X12902908412110#abstract

59. Khanna P, Salwan SK, Sharma A. Correlation of Platelet Indices in Patients With Type 2 Diabetes Mellitus and Associated Microvascular Complications: A Hospital-Based, Prospective, Case-Control Study. Cureus. 16(3):e55959.

60. Saha A, Das R, Patra D, Chatterjee S. The role of platelet indices in diabetes mellitus - a comparative study of 160 cases among diabetic and non diabetic individuals in a tertiary care hospital in Eastern India. Int J Life Sci. 2023;12(4).

61. Walinjkar RS, Khadse S, Kumar S, Bawankule S, Acharya S. Platelet Indices as a Predictor of Microvascular Complications in Type 2 Diabetes. Indian J Endocrinol Metab. 2019 Apr;23(2):206.

62. Alhadas KR, Santos SN, Freitas MMS, Viana SMSA, Ribeiro LC, Costa MB. Are platelet indices useful in the evaluation of type 2 diabetic patients? J Bras Patol E Med Lab. 2016 Apr 26;52:96–102.

63. Aaberg ML, Burch DM, Hud ZR, Zacharias MP. Gender differences in the onset of diabetic neuropathy. J Diabetes Complications. 2008;22(2):83–7.

64. Venkata B, M SM. A Study of Platelets Indices in Patients with Diabetic Retinopathy and without Diabetic Retinopathy & the Effect of

Hyperglycemia on Platelet Indices. J Assoc Physicians India. 2022 Apr;70(4):11–2.

65. Endothelial Dysfunction in Diabetic Retinopathy - PMC [Internet]. [cited 2025 Jan 28]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7499433/
66. Oxidative stress and diabetic retinopathy: Molecular mechanisms, pathogenetic role and therapeutic implications - PMC [Internet]. [cited 2025 Jan 28]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7767789/
67. Qian Y, Zeng Y, Lin Q, Huang H, Zhang W, Yu H, et al. Association of platelet count and plateletcrit with nerve conduction function and peripheral neuropathy in patients with type 2 diabetes mellitus. J Diabetes Investig. 2021 Oct;12(10):1835–44.
68. Papanas N, Mavridis G, Karavageli E, Symeonidis G, Maltezos E. Peripheral neuropathy is associated with increased mean platelet volume in type 2 diabetic patients. Platelets. 2005 Jan 1;16(8):498–9.
69. Microvascular Dysfunction in Diabetes Mellitus and Cardiometabolic Disease - PMC [Internet]. [cited 2025 Jan 28]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7846151/
70. Immature platelet fraction in diabetes mellitus and metabolic syndrome - PubMed [Internet]. [cited 2025 Jan 28]. Available from: https://pubmed.ncbi.nlm.nih.gov/24140451/
71. Sengupta P, Priyadarshini A, Kumar Behera P, Padarabinda Tripathy K. Exploring Platelet Indices as Predictors of Nephropathy Severity in Type 2 Diabetes Mellitus: A Hospital-Based Cross-Sectional Analysis. Cureus. 2024 Oct;16(10):e71796.

72. Diabetic vascular diseases: molecular mechanisms and therapeutic strategies | Signal Transduction and Targeted Therapy [Internet]. [cited 2025 Jan 28]. Available from: https://www.nature.com/articles/s41392-023-01400-z

73. Wei S, Pan X, Xiao Y, Chen R, Wei J. The unique association between the level of plateletcrit and the prevalence of diabetic kidney disease: a cross-sectional study. Front Endocrinol [Internet]. 2024 Apr 25 [cited 2025 Jan 28];15. Available from: https://www.frontiersin.org/journals/endocrinology/articles/10.3389/fendo.2024.1345293/full

74. Taderegew MM, Woldeamanuel GG, Emeria MS, Tilahun M, Yitbarek GY, Zegeye B. Platelet Indices and Its Association with Microvascular Complications Among Type 2 Diabetes Mellitus Patients in Northeast Ethiopia: A Cross-Sectional Study . Diabetes Metab Syndr Obes. 2021 Feb 25;14:865–74.

ANNEXURE – I - INFORMED CONSENT FORM

" Platelet Indices in patients of Type II Diabetes Mellitus at Tertiary Care Teaching Hospital – One year Cross Sectional Study "

Name of Student/Principal Investigator:

Junior Resident, Department of General Medicine, JNMC Belagavi

Name of Guide/Co Investigators:

MD (GENERAL MEDICINE)

PROFESSOR AND HEAD OF DEPARTMENT

OF GENERAL MEDICINE, JNMC BELAGAVI

Introduction: The Patients who are having Type 2 Diabetes Mellitus are at increased risk of having microvascular (Diabetic Retinopathy, Diabetic Neuropathy, Diabetic Nephropathy) and macrovascular complications (Cardiovascular Disease, Stroke). Platelets play an important role in the pathophysiology of the complications. This study correlates the Platelet Indices with HbA1c and microvascular complications.

Explanation of procedure: Participant with Type 2 Diabetes will be selected and Detailed Clinical History with Complete Hemogram with Platelet Indices, Urine Albumin: Creatinine Ratio, HbA1c levels, Fundoscopy and Diabetic Neuropathy examination will be done.

Withdrawal from participation in the study: Participation in this study in 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 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 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 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 personal identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact:

If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Institutional Ethical Committee for Human Subjects Research 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 **" Platelet Indices in patients of Type II Diabetes Mellitus 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 - QUESTIONNAIRE

Title: "Platelet Indices in patients of Type II Diabetes Mellitus at Tertiary Care Teaching Hospital – One year Cross Sectional Study"

Investigator:

Guide:

DATA COLLECTION QUESTIONNAIRE

Note: All the data collected will be kept confidential. Your personal identity will not be revealed to anyone.

- **Demographic Details** Date of Interview: _____

1. Name of the patient : _____
2. IP Number: _____
3. Age: ____
4. Sex: Male / Female
5. Address: _____
6. Occupation: _____
7. Phone Number: _____
8. Income/ month: _____

Chief Complaints:	
Past History	
Personal History	
Treatment History	

- **Diabetes History**

9. When were you first diagnosed with diabetes? Year _____ Age_____
10. Please list all medication(s) you take, including dosage :
11. Do you take insulin, if Yes How many units:
12. Any History of Ischemic Heart Disease:
13. Any History of Stroke:
14. Risk Factors:

    Smoking Alcohol Hypertension

    Thyroid Disease Stroke Cardiovascular Disease

    High Cholesterol/Triglycerides Others:

- **Vitals:**

Temperature		Height	cm
Pulse	/min	Weight	Kg
Blood Pressure	/ mm Hg	BMI	
Respiratory Rate	/min		

- **Physical Examination:**

Index	Yes	No
Pallor		
Icterus		
Cyanosis		
Clubbing		
Lymphadenopathy		

- **Systemic Examination:**

CVS	
RS	
P/A	
CNS	

- **Ophthalmoscopy Examination:**

- **Neuropathy Examination: Revised Neuropathy Disability Score**

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

If score >6 is suggestive of Diabetic Neuropathy.

- **Nephropathy Examination**:

Urine examination - Urine albumin: Creatinine ratio

- **Lab Investigations:**

Investigations	**Values**	**Investigations**	**Values**
Hemoglobin		White Total Count	
RBC Count		Platelet Count	
Hematocrit		Mean Platelet Volume	
MCV		Platelet Distribution Width	
MCH		Platelet Crit	
MCHC		Platelet- Large Cell Ratio	
RDW		Platelet- LCC	
Urine Albumin: Creatinine Ratio		Immature Platelet Fraction	
HbA1c			

- **Microvascular Complications:**

Diabetic Retinopathy	
Diabetic Neuropathy	
Diabetic Nephropathy	

ANNEXURE – III

MASTER CHART

Sr No	IP Number	Age	Gender	HBA1C	Hemoglobin	RBC Count	Hematocrit	MCV	MCH	MCHC	RDW	TLC	Platelet Count	MPV	PDW	Plateletcrit	Platelet LCR	IPF	UACR	T2DM Years	Diabetic Retinopathy	Diabetic Retinopathy	Diabetic Neuropathy	DN Score(NDS)	Sample No
1	10021645	46	F	12.9	14.2	5.46	49.7	91	26	28.6	15	10.54	323	9.6	10.3	0.31	22.1	3.1	5.34	10	BE Mild NPDR	Present	Present	7	23574290
2	10018404	33	F	8	11.3	4.27	38.8	90.9	26.5	29.1	15.9	5.58	305	9.6	9.6	0.32	21.1	2.2	10.98	11	BE Fundi WNL	Absent	Absent	4	23574287
3	10049449	32	M	9.3	16.7	6.03	53.7	89.1	27.7	31.1	13.9	10.5	208	9.6	10.9	0.2	21.6	3.1	261.61	0.5	BE Fundi WNL	Absent	Absent	3	24166724
4	10049291	73	M	9.2	10.4	3.97	35.3	88.9	26.2	29.5	15.6	7.84	439	9.5	9.5	0.42	19.4	1.7	62.6	0.16	BE Fundi WNL	Absent	Present	7	24166723
5	10070179	66	F	11.8	6.9	2.61	22.3	85.4	26.4	30.9	18.3	14.23	174	12.2	17.6	0.22	44.5	12	420.72	15	BE Moderate NPDR	Present	Present	8	24372693
6	10084110	63	F	10.1	11.3	4.9	39.2	80	23.1	28.8	16.4	11.23	411	9	8.8	0.37	18	2.4	133.19	2	BE Fundi WNL	Absent	Absent	2	224656471
7	10021445	51	F	7.3	11.1	4.57	38.5	84.2	24.3	28.8	15.4	9.62	267	10.3	10.8	0.28	26.4	3.1	7.45	3	RE Mild NPDR	Present	Absent	3	23575008
8	10023753	33	M	10.6	14.6	5.1	44.4	87.1	28.6	32.9	14.6	5.85	123	11.4	14.3	0.14	36.6	7.2	28.75	3	BE Moderate NPDR	Present	Present	7	23601095
9	10024180	55	M	7.9	15.1	5.14	48.2	93.8	29.4	31.3	12.7	7.77	285	8.6	8.8	0.22	14.4	2	8.47	6	BE Fundi WNL	Absent	Absent	1	23601094
10	10023455	66	M	8.1	13.9	5	45.5	91	27.8	30.5	13.5	8.01	357	8.9	8.6	0.29	15.8	1.3	15.89	2	BE Fundi WNL	Absent	Absent	1	23601093
11	10024117	65	M	8.1	11.6	4.27	37.9	88.8	27.2	30.6	12.7	8.75	232	10.7	12	0.24	29.7	7.4	29.3	2	BE Mild NPDR	Present	Present	7	23601096
12	10021487	50	F	9.5	12	4.44	39	87	27	30.8	13.1	8.9	294	10.4	11.2	0.31	27.9	3.2	7.14	15	BE Moderate NPDR	Present	Absent	3	23575009
13	10021754	44	F	6.5	13.1	4.57	44.8	98	28.7	29.2	12.6	4.91	61	11.9	13.9	0.07	39.4	2.1	3.79	5	BE Mild NPDR	Present	Absent	1	23456633
14	10050697	65	M	7.5	10.9	4.63	35.7	77.1	23.5	30.5	15.2	11.1	359	10.6	12	0.38	29	5.3	172	10	BE Mild NPDR	Present	Present	7	24175010
15	10050836	75	M	9.2	13.1	4.6	40.8	88.7	28.5	32.1	16.9	19.2	251	9.9	11.8	0.26	28	7.3	185.94	3	BE Fundi WNL	Absent	Absent	2	24175008
16	10082659	62	M	6.3	16.9	6.49	52	80.1	26	32.5	16.1	11.68	257	10	11.9	0.26	25.6	5.1	538.04	5	BE Mild NPDR	Present	Absent	4	24454315
17	10051790	63	M	7.2	8.7	2.89	29.1	100.7	30.1	29.9	16.9	5.04	174	10.8	12.7	0.19	31.7	3.1	38.65	1	BE Mild NPDR	Present	Absent	2	24181691
18	10083251	58	M	10.1	14.3	5.4	45.9	85	26.5	31.2	12.5	5.72	260	11.2	12.8	0.26	24.2	5.3	131.67	10	BE Moderate NPDR	Present	Present	8	24465212
19	10067694	63	M	7.6	9.4	3.24	29.8	92	29	31.5	14.3	16.15	187	10.7	11.4	0.2	29.5	7.8	53.13	5	BE Mild NPDR	Present	Present	7	24335049
20	10068190	62	M	6.5	12	4.49	40	89.1	26.7	30	13.9	9.81	250	9.6	9.8	0.24	21.4	4.7	13.63	1	BE Fundi WNL	Absent	Absent	1	24335053
21	10065947	71	F	6.7	11.8	4.09	39.1	95.6	28.9	30.2	13.3	9.56	197	11.8	15.5	0.23	39.9	12.7	132.53	10	BE Mild NPDR	Present	Present	8	24335051
22	10069024	63	M	7.6	14.2	4.75	43.6	91.8	29.9	32.6	13.5	9.94	169	11	13.2	0.19	32.4	8.9	1.85	3	BE Mild NPDR	Present	Present	7	24335050
23	10068473	60	M	6.2	12.9	4.6	39.3	85.4	28	32.8	11.9	6.57	178	11.6	15.1	0.21	36.1	8.4	65.59	12	BE Mild NPDR	Present	Present	7	24331235
24	10069950	74	M	9.3	12.6	4.5	41	90.9	27.9	30.7	13.6	15.08	336	9.3	9.5	0.31	19.2	2.1	85.82	2	BE Fundi WNL	Absent	Absent	0	24336008
25	10071061	52	F	7.3	11.1	4.52	38.5	85.2	24.6	28.8	13.6	9.9	339	9.1	9.5	0.29	17.2	2.1	77.78	4	BE Fundi WNL	Absent	Absent	3	24352515
26	10069835	71	F	9.6	14.1	5.36	44.1	82.2	26.2	31.9	17.2	8.4	309	8.3	10.1	0.23	14.4	3.1	47.46	5	BE Fundi WNL	Absent	Absent	2	24335124
27	10071246	56	M	7.4	16.6	5.45	49.9	91.6	30.5	33.3	11.9	9.42	193	11.1	13.7	0.21	33.3	6.8	1973	6	BE Moderate NPDR	Present	Present	8	24352516
28	10070649	63	M	6.4	12.4	4.36	40.3	92.4	28.4	30.8	13.9	9.72	355	9.3	9.2	0.33	19	1.9	7.64	5	BE Fundi WNL	Absent	Absent	1	24342139
29	10069041	39	M	9.2	17	5.67	52.8	93.1	30	32.2	11.5	12.73	220	10.8	13	0.24	30.8	7.4	4.37	5	BE Moderate NPDR	Present	Present	7	24327972
30	10075959	44	F	6.2	11	4.02	38.4	95.5	27.4	28.6	13.9	8.15	267	9.8	9.8	0.24	22.3	3.2	27.39	4	BE Fundi WNL	Absent	Absent	2	24396465
31	10076443	56	F	7.9	10.2	3.85	34.2	88.8	26.5	29.8	16.1	7.42	325	10.8	12.7	0.33	31	5.2	80.86	10	BE Mild NPDR	Present	Absent	2	24396464
32	10073882	60	M	12.5	13.5	4.87	44.1	90.6	27.7	30.6	13.9	6.23	312	10.8	12.4	0.34	30.5	5.5	47.83	10	BE Mild NPDR	Present	Present	7	24396462
33	10076338	70	M	6.8	12.3	4.99	42.5	85.2	24.6	28.9	15.4	7.32	338	10.2	11.2	0.34	26.1	4.4	3.48	3	BE Fundi WNL	Absent	Absent	1	24400854
34	10075988	54	M	7.8	12.5	4.42	40.1	90.7	28.3	31.2	14	3.87	95	11.3	15	0.1	37.4	11.4	191.27	10	BE Mild NPDR	Present	Present	7	24396459
35	10075760	62	F	8.1	12.2	4.75	38.8	81.7	25.7	31.4	15.1	6.62	189	10.3	11.4	0.18	28.3	5.9	186.31	3	BE Fundi WNL	Absent	Absent	2	24396460
36	10076071	46	F	6.9	10.1	5.03	37.1	73.8	20.1	27.2	17.6	17.35	303	11.3	14.1	0.35	35.6	9.8	17.42	6	BE Mild NPDR	Present	Present	7	24396461
37	10075179	38	F	8.3	8.5	3.37	31.7	94.1	25.2	26.8	15.9	19.99	125	12.6	16.9	0.16	45	16.5	160.43	0.1	BE Moderate NPDR	Present	Present	8	24400852

38	10075212	76	M	9.2	8.4	3.47	27.7	79.8	24.2	30.3	20.3	15.36	290	8.8	8.3	0.21	15.4	3.5	3.54	1	BE Fundi WNL	Absent	Absent	0	24407392
39	10076774	65	F	6.1	6.5	2.72	23.3	85.7	23.9	27.9	16.4	18	291	9.7	10.1	0.26	21.2	4	10.37	0.5	BE Fundi WNL	Absent	Absent	1	24407402
40	10072209	86	M	7.4	7	2.41	25	103.7	29	28	13.1	14.87	274	11.1	12.4	0.3	34	5.3	357.75	6	BE Moderate NPDR	Present	Present	7	24405495
41	10076997	64	M	6.7	9.5	3.71	32.8	88.4	25.6	29	14.5	12.48	225	11	12	0.24	32.5	7.7	835.16	4	BE Mild NPDR	Present	Absent	2	24405497
42	10076981	54	M	7.8	12.4	5.94	43.6	73.4	20.9	28.4	15.4	5.7	201	10.8	15.4	0.22	33	8.7	15.83	6	BE Mild NPDR	Present	Present	8	24405504
43	10077129	68	M	8.2	12.9	4.23	40.3	95.3	30.5	32	13	7.09	283	9.9	11.4	0.26	24.3	4.5	10.4	2	BE Fundi WNL	Absent	Present	6	24405498
44	10076475	34	M	6.7	17	6.08	55.9	91.9	28	30.4	13.5	4.11	36	11.3	12.6	0.19	32.1	7.4	1686.44	2	BE Mild NPDR	Present	Present	7	24400855
45	10076665	78	M	5.5	15.1	5.28	50.2	95.1	28.6	30.1	13.2	6.15	175	11.7	16.3	0.19	37.9	13.1	60.05	8	BE Mild NPDR	Present	Present	7	24400853
46	10076396	63	F	6.3	12.8	5.1	42.8	83.9	25.1	29.9	13.9	10.1	285	10.3	11.9	0.29	27.2	5.1	86.32	5	BE Fundi WNL	Absent	Present	7	24405503
47	10076635	72	M	9.6	12.2	4.32	39.7	91.9	28.2	30.7	13.5	14.46	172	11.1	12.1	0.18	31.8	7.6	1309.72	14	BE Moderate NPDR	Present	Present	8	24400866
48	10075588	66	F	7.2	9.1	3.65	31.2	85.5	24.9	29.2	15.1	4.87	344	9.8	10.3	0.3	22.5	2.9	33.48	1	BE Fundi WNL	Absent	Absent	2	24400849
49	10075266	73	M	5.5	10.5	3.75	32.7	87.2	28	32.1	15.4	6.29	308	10.1	10.8	0.28	25.5	5.5	92.07	2	BE Fundi WNL	Absent	Absent	2	24405499
50	10071493	67	F	10	14.5	5.49	46.5	84.7	26.4	31.2	15.4	11.97	113	9.9	10.8	0.1	25.8	8.7	324.16	2	BE Fundi WNL	Absent	Absent	5	24400857
51	10077091	49	F	7.3	12.2	4.65	43.5	93.5	26.2	28	14.6	4.49	199	10.1	10.6	0.19	25.8	4.7	100.53	1	BE Fundi WNL	Absent	Absent	2	24405502
52	10076063	66	F	6.7	12.2	4.25	39.4	92.7	28.7	31	12.4	9.2	393	8.4	7.7	0.27	11.9	1	61.23	2	BE Fundi WNL	Absent	Absent	2	24400865
53	10076724	62	M	9.6	11.3	3.91	36.3	92.8	28.9	31.1	14	32.93	300	11.5	14.3	0.34	37	10.1	20.24	10	BE Moderate NPDR	Present	Present	7	24407394
54	10077493	63	M	6.4	12	4.55	39.1	85.9	26.4	30.7	13.4	7.13	380	8.9	8.4	0.3	15.2	3.3	8.5	1	BE Fundi WNL	Absent	Absent	1	24407399
55	10076717	69	M	4.9	13.1	4.52	42.7	94.5	29	30.7	14.6	12.06	165	11.8	15.2	0.19	40.1	9.5	3.54	6	BE Mild NPDR	Present	Present	7	24407401
56	10076505	60	F	11	8.3	3.06	28.8	94.1	27.1	28.8	15	10.56	115	10.2	11.2	0.11	25.7	2.9	46.72	2	BE Fundi WNL	Absent	Absent	0	24400856
57	10074226	62	M	5.8	12.1	3.79	37.6	99.2	31.9	32.2	13.1	6.57	228	10.9	12.7	0.24	31.9	8.7	46.72	5	BE Mild NPDR	Present	Present	7	24400861
58	10077497	70	M	7.6	10	3.4	33.4	98.2	29.4	29.9	21.2	7.54	179	9.4	9	0.15	19.3	4.6	139.53	2	BE Fundi WNL	Absent	Absent	2	24407403
59	10080356	65	M	9.6	13.1	4.24	41.7	98.3	30.9	31.4	12.5	12.33	144	11.7	14.7	0.17	39	8.4	44.85	20	BE Severe NPDR	Present	Present	9	24433628
60	10079854	56	M	10.3	14.1	5.1	43	84.3	27.6	32.8	12.5	5.92	296	9.2	10.1	0.24	19.2	3.5	11.22	2	BE Fundi WNL	Absent	Absent	0	24433627
61	10077633	45	M	6.2	8.4	3.05	25.1	82.3	27.5	33.5	13.4	6	224	9.5	9	0.19	18.9	2.1	1857.83	1	BE Fundi WNL	Absent	Absent	0	24415349
62	10077527	42	F	9	13.7	4.94	43.9	88.9	27.7	31.2	12	6.17	347	9.6	10.5	0.3	21.8	2.3	19.42	1	BE Fundi WNL	Absent	Absent	2	24415346
63	10078296	35	M	6	11.8	5.99	39.8	66.4	19.7	29.6	15.8	6.82	315	10	11.8	0.32	26.2	4.7	32.8	3	BE Mild NPDR	Present	Absent	3	24415342
64	10077656	73	M	5.7	7.3	2.61	23.4	89.7	28	31.2	14.3	15.55	73	13.5	20.4	0.1	47.9	14.4	60.71	16	BE Mild NPDR	Present	Present	7	24415344
65	10077796	65	M	9.8	8.3	3.97	26.9	67.8	20.9	30.9	18.6	25.75	467	9.8	10.5	0.41	25.4	7.5	37.98	5	BE Mild NPDR	Present	Absent	0	24415351
66	10076723	69	M	11.3	13.3	5	41.9	83.8	26.6	31.7	13.4	16.5	345	9.5	9.9	0.31	20.5	3.9	48.27	1	BE Fundi WNL	Absent	Absent	0	24415345
67	10080362	58	F	5.3	12.3	4.42	42.5	96.2	27.8	28.9	14.3	7.79	357	10.2	11.4	0.37	27.1	3.1	4.32	5	BE Mild NPDR	Present	Absent	1	24433630
68	10072420	62	M	11.3	9.4	3.55	30.2	85.1	26.5	31.1	13.1	6.45	154	9.4	9.8	0.13	21	2.6	7.64	1	BE Fundi WNL	Absent	Absent	2	24396458
69	10076316	84	F	12.3	10.4	3.9	36.4	93.3	26.7	28.6	14.6	7.81	225	9.9	9.8	0.21	22.7	1.6	363.92	4	BE Fundi WNL	Absent	Absent	2	24396463
70	10072126	70	M	6.6	8.4	3.28	29.5	89.9	25.6	28.5	15	19.85	495	9	8.5	0.39	16.4	2.4	0.29	1	BE Fundi WNL	Absent	Absent	0	24396457
71	10077749	74	M	11.5	13.3	5.27	45.6	86.5	25.2	29.2	14.8	16.99	203	10.4	12.6	0.21	28.6	5.8	129.89	14	BE Moderate NPDR	Present	Present	7	24409454
72	10076178	75	M	9.6	15.1	4.93	45.8	92.9	30.6	33	12.1	6.79	230	9	9.3	0.19	16.9	2.4	8.4	2	BE Fundi WNL	Absent	Absent	0	24409453
73	10077277	55	F	6.9	8.4	3.49	30.3	86.8	24.1	27.7	14.7	5.38	321	10.1	10.5	0.31	24.1	4	1135.28	5	BE Mild NPDR	Present	Absent	3	24409455
74	10076761	74	M	6.7	11.1	4.5	36.4	80.9	24.7	30.5	14.7	7.68	353	8.9	8.7	0.28	14.6	1.5	73.47	2	BE Fundi WNL	Absent	Absent	1	24409456
75	10073475	58	M	9.1	14.1	4.97	46.9	94.4	28.4	30.1	13.2	8.4	252	11.1	13.2	0.27	33.4	9	118.33	11	BE Moderate NPDR	Present	Present	8	24372696
76	10078537	72	F	5.7	10.3	3.91	36.5	93.4	26.3	28.2	15.5	9.63	172	9.7	10.3	0.15	22.1	3.6	20.06	2	BE Fundi WNL	Absent	Absent	2	24431180
77	10079866	80	F	6.8	12.7	5.01	42.8	85.4	25.3	29.7	13.9	5.78	281	9.5	9.8	0.26	20.9	3.1	24.92	5	BE Fundi WNL	Absent	Absent	1	24431183
78	10078928	72	M	5.8	12	4.26	39.8	93.4	28.2	30.2	13.3	8.56	370	9.1	9.1	0.3	16.8	1	121.1	2	BE Fundi WNL	Absent	Absent	1	24431185
79	10080104	76	M	7.2	10.1	2.84	31.7	111.6	35.6	31.6	13.7	8.44	249	11.1	11.8	0.27	32.1	4.9	1993.02	12	BE Mild NPDR	Present	Present	7	24431186
80	10079395	65	F	7	10.9	4.11	35.9	87.3	26.5	30.4	15.9	8.75	14	13.2	11.4	0.01	46.9	18.2	16	10	BE Moderate NPDR	Present	Present	7	24433625
81	10075509	75	M	6.8	13.5	4.88	43.8	89.8	27.7	30.8	13.2	9.71	304	9.7	11	0.29	23	6.8	337.7	5	BE Mild NPDR	Present	Absent	3	24386750
82	10077841	74	M	10	12.3	6.16	43.3	70.3	20	28.4	19.7	4.09	142	10.8	13.4	0.16	32.5	6.8	88.14	10	BE Moderate NPDR	Present	Present	8	24415348
83	10080413	72	F	7.6	9.7	4.76	32.7	68.7	20.4	29.7	15.2	6.93	220	9.3	9.3	0.18	18.8	3.6	33.37	1	BE Fundi WNL	Absent	Absent	0	24436187

84	10080092	61	M	11.5	9.1	3.14	28	89.2	29	32.5	13.4	23.29	230	9.1	8.6	0.17	16.8	2.9	167.41	3	BE Fundi WNL	Absent	Absent	1	24436185
85	10080376	79	F	7	8.1	2.73	29	106.2	29.7	27.9	16.6	7.57	177	11.8	13.7	0.2	37.5	8.1	123.86	15	BE Moderate PDR	Present	Present	8	24436186
86	10080613	60	F	9.1	11.8	6.53	42.3	64.8	18.1	27.9	23.9	9.81	253	9.7	13.4	0.25	24.9	4.6	100.6	5	BE Mild NPDR	Present	Absent	3	24442129
87	10080418	48	M	10.6	12	4.19	37.8	90.2	28.6	31.7	13.8	10.06	74	12	16.5	0.09	40.2	8.4	160.45	10	BE Moderate PDR	Present	Present	9	24433766
88	10080005	59	F	7.6	13.5	4.93	44.1	89.5	27.4	30.6	14.2	9.89	277	10.7	12.7	0.28	30.4	6.8	36.91	2	BE Fundi WNL	Absent	Absent	2	24431178
89	10081142	98	M	7.3	7.2	2.65	23.5	88.7	27.2	30.6	18.1	9.53	211	10.2	11.2	0.22	26.5	2.1	72.55	3	BE Fundi WNL	Absent	Absent	1	24442133
90	10080116	54	M	5.7	14.4	4.23	45.1	106.6	34	31.9	13.9	7.16	82	12.2	15.7	0.1	41.8	5.1	175.76	5	BE Mild NPDR	Present	Absent	2	24442136
91	10079411	48	F	11.4	8.3	3.25	29.2	89.8	25.5	28.4	16.7	9.41	194	10.5	12	0.21	28.3	9.4	488	10	BE Moderate PDR	Present	Present	8	24442148
92	10079899	70	F	8.1	13	4.87	40.8	83.8	26.7	31.9	13.9	28.67	395	10.7	11.8	0.41	30.2	7.2	12.07	10	BE Mild NPDR	Present	Present	7	24431184
93	10078930	61	M	6.3	14.5	5.42	46.3	85.4	26.8	31.3	13.2	7.06	254	10.6	12.7	0.26	29.2	6.1	139.29	4	BE Fundi WNL	Absent	Absent	2	24431187
94	10076755	75	M	7.8	12.2	4.42	38.4	86.9	27.6	31.8	13.9	10.69	264	9.7	9.5	0.26	21.8	2.5	3114.2	5	BE Fundi WNL	Absent	Absent	2	24407068
95	10077680	65	M	6.7	9.8	4.03	33.2	82.4	24.3	29.5	16.4	5.64	254	10.9	12	0.26	32	5.4	64.54	5	BE Mild NPDR	Present	Absent	1	24431188
96	10079022	53	F	6.8	11.9	4.54	36.8	81.1	26.2	32.3	13.6	9.28	438	9.9	10.8	0.39	23.8	4.6	38.6	3	BE Fundi WNL	Absent	Absent	0	24431179
97	10080392	52	M	9.3	13.4	5.18	44.4	85.7	25.9	30.2	14	5.26	169	9.6	11	0.16	23	3.2	0.94	2	BE Fundi WNL	Absent	Absent	6	24442143
98	10080640	60	M	7	14.1	5.06	43.9	86.8	27.9	32.1	15	5.42	279	9.8	10.2	0.27	22.6	2.4	6.7	2	BE Fundi WNL	Absent	Absent	0	24435214
99	10081110	76	F	16.7	12.6	4.52	42.1	93.1	27.9	29.9	14.7	19.12	286	10.1	11.5	0.29	26.5	5.4	115.71	10	BE Moderate NPDR	Present	Absent	4	24439528
100	10081457	75	F	5.8	11.7	4.39	37.5	85.4	26.7	31.2	13.5	12.55	241	10.3	10.2	0.25	26.3	4.1	7.4	1	BE Fundi WNL	Absent	Absent	0	24444716
101	10080082	51	F	10.4	13.9	4.79	46.4	96.9	29	30	11.9	23.54	363	10.1	11.5	0.37	25.3	3.1	72.28	5	BE Mild NPDR	Present	Absent	4	24430162
102	10083117	58	M	7.2	11.6	4.26	38.5	90.4	27.2	30.1	13.4	6.04	277	9.6	9.1	0.24	20.8	4.8	5.25	2	BE Fundi WNL	Absent	Absent	2	24469305
103	10083556	43	F	6.4	15.1	5.35	48.1	89.9	28.2	31.4	12.4	5.77	246	8.5	8.2	0.18	12.7	1.8	1.39	1	BE Fundi WNL	Absent	Absent	0	24465058
104	10082116	75	F	6.9	6.6	3.69	24.7	66.9	17.9	26.7	22.6	5.4	389	9.5	9.4	0.37	21.6	2.1	43.75	1	BE Fundi WNL	Absent	Absent	1	24455643
105	10081820	79	M	6.1	8.6	3	27.1	90.3	28.7	31.7	14.4	7.94	303	8.7	7.7	0.26	13.8	1.6	51.31	2	BE Fundi WNL	Absent	Absent	1	24455644
106	10078060	65	M	9.6	10.4	4.71	36.9	78.3	22.1	28.2	15.2	6.69	259	12.1	17.5	0.31	42.4	8.8	38.33	8	BE Mild NPDR	Present	Present	7	24412200
107	10082581	38	M	9.9	14.1	5.12	43.9	85.7	27.5	32.1	12.5	5.64	114	10.2	12.3	0.12	27.3	4.8	286.4	5	BE Mild NPDR	Present	Absent	4	24455648
108	10082425	85	F	7	7	2.69	25.6	95.2	26	27.3	16.6	5.74	191	9.9	10	0.18	23.1	4.1	484.18	3	BE Fundi WNL	Absent	Absent	2	24455646
109	10080917	49	M	8.7	14.4	4.87	44.2	90.8	29.6	32.6	14	4.63	92	11.9	18.2	0.11	39.8	7.8	388.56	10	BE Mild NPDR	Present	Present	7	24442140
110	10081468	76	M	6.8	8.6	4.01	32.1	80	21.4	26.8	19.7	11.75	334	9.7	10.3	0.3	22.8	3.1	18.13	2	BE Fundi WNL	Absent	Absent	1	24444715
111	10082565	78	M	11.1	13.3	4.42	42.3	95.7	30.1	31.4	12.3	7.56	255	11.3	14.3	0.29	35.5	8.9	190.39	12	BE Moderate NPDR	Present	Present	8	24453164
112	10081800	70	M	5.7	11.6	4.51	39.2	86.9	25.7	29.6	14.5	9.88	260	10.4	11.5	0.27	27.5	4.1	153.06	6	BE Fundi WNL	Absent	Absent	2	24453388
113	10082252	59	M	8.8	15.7	5.66	51.1	90.3	27.7	30.7	13.9	6.08	253	12.9	20.5	0.3	48.5	8.2	2.26	5	BE Mild NPDR	Present	Present	7	24451659
114	10081383	72	M	7.1	10.4	3.69	33	89.4	28.2	31.5	17.3	33.14	157	10.4	11.4	0.16	27.6	4.1	128.67	2	BE Fundi WNL	Absent	Absent	2	24443001
115	10082313	54	F	12.7	11.3	4.29	37.8	88.1	26.3	29.9	13.2	8.31	248	10.4	11.9	0.25	28	5.3	5.94	3	BE Mild NPDR	Present	Absent	2	24451658
116	10081960	54	M	11.2	12.3	4.51	39.7	88	27.3	31	12.9	2.96	122	11.7	14.9	0.16	38.1	8.6	920.75	14	BE Moderate PDR	Present	Present	9	24451657
117	10081543	58	F	6.6	12.1	4.33	38.8	89.6	27.9	31.2	12.2	2.95	149	11.2	13.2	0.17	34.4	8	60.83	5	BE Mild NPDR	Present	Present	7	24446829
118	10081230	58	F	13.2	14.3	4.98	45.9	92.2	28.7	31.2	12.4	7.44	203	10.2	11.3	0.2	25.9	5.4	34.68	1	BE Fundi WNL	Absent	Absent	5	24446827
119	10081501	72	M	6.8	10.5	3.33	28.6	85.9	31.5	36.7	11.9	8.42	300	8.3	7.6	0.21	10.5	2.8	3.03	2	BE Fundi WNL	Absent	Absent	2	24446828
120	10081265	42	F	8.2	10.7	4.34	36.6	84.3	24.7	29.2	12.8	10.79	412	9.7	10.3	0.38	21.9	3.2	183.53	3	BE Fundi WNL	Absent	Absent	2	24446227
121	10081372	52	F	12.1	12	4.61	40.6	88.1	26	29.6	15.1	4.73	188	11.7	13.4	0.22	38.8	8.7	45.23	6	BE Moderate NPDR	Present	Present	7	24446848
122	10078015	63	F	8.4	9.7	5.87	40.4	68.8	16.5	24	20.5	12.79	234	9.5	10.8	0.22	22.5	2.4	145.11	3	BE Fundi WNL	Absent	Absent	2	24415748
123	10079101	52	M	9.2	10.7	4.8	35.9	74.8	22.3	29.8	22.2	6.62	272	8.9	8.8	0.24	17.1	1.8	68.48	2	BE Fundi WNL	Absent	Absent	0	24401759
124	10083798	65	M	9.6	11.1	4.41	36.1	81.9	25.2	30.7	18	4.79	226	10.1	10.7	0.23	26.3	7.6	132.29	9	BE Mild NPDR	Present	Present	7	24485844
125	10083974	71	M	6.7	15	5.35	48.2	90.1	28	31.1	13.4	11.87	236	8.8	8.7	0.21	15.7	1.4	205.88	2	BE Fundi WNL	Absent	Absent	2	24465919