

## MEAN PLATELET VOLUME AS A PREDICTIVE MARKER FOR GLYCEMIC CONTROL AND MICROVASCULAR COMPLICATIONS IN TYPE 2 DIABETES MELLITUS

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### Abstract

**Background:** Type 2 Diabetes Mellitus (T2DM) is a progressive metabolic disorder associated with chronic hyperglycemia, platelet dysfunction, and increased risk of microvascular complications, including diabetic retinopathy, nephropathy, and neuropathy. Platelet activation plays a crucial role in the pathophysiology of vascular complications, and mean platelet volume (MPV) has emerged as a potential hematological biomarker reflecting platelet size, reactivity, and thrombogenicity. Elevated MPV has been linked to poor glycemic control and endothelial dysfunction, but its exact correlation with glycated hemoglobin (HbA1c) levels and microvascular complications in T2DM patients remains unclear. This study aims to assess the association between MPV and HbA1c levels and evaluate its potential role in detecting early microvascular complications in T2DM patients. This study investigates the correlation between MPV and HbA1c in patients with T2DM and explores the association between MPV and diabetic microvascular complications, assessing its potential utility as a predictive marker for early vascular impairment.

**Materials and Methods:** A cross-sectional observational study was conducted on 100 patients diagnosed with T2DM at a tertiary care center. Patients were categorized into two groups based on glycemic control (HbA1c <7.0% vs. HbA1c ≥7.0%) and the presence or absence of microvascular complications. Clinical and laboratory assessments included fasting blood glucose (FBG), postprandial blood glucose (PPBG), HbA1c, platelet indices (MPV, platelet count), renal function tests, and lipid profiles. MPV was measured using an automated hematology analyzer, and diabetic complications were assessed via fundoscopy for retinopathy, urine albumin-to-creatinine ratio (UACR) for nephropathy, and nerve conduction studies for neuropathy. Statistical analysis included Pearson's correlation, logistic regression, and receiver operating characteristic (ROC) curve analysis to determine the diagnostic value of MPV in predicting diabetic microvascular complications.

**Result:** Among 100 T2DM patients, the mean age was 54.6 ± 8.9 years, with 57 males and 43 females. Patients with poor glycemic control (HbA1c ≥7.0%) had significantly higher MPV levels ( $p < 0.001$ ) compared to those with well-controlled diabetes (HbA1c <7.0%). MPV showed a strong positive correlation with HbA1c ( $r = 0.62$ ,  $p < 0.001$ ), fasting blood glucose ( $r = 0.58$ ,  $p < 0.001$ ), and postprandial glucose ( $r = 0.60$ ,  $p < 0.001$ ). Additionally, MPV levels were significantly elevated in patients with diabetic retinopathy ( $11.3 \pm 0.9$  fL), nephropathy ( $11.1 \pm 1.0$  fL), and neuropathy ( $10.9 \pm 0.8$  fL) compared to those without complications ( $9.2 \pm 0.7$  fL,  $p < 0.001$ ). ROC curve analysis demonstrated that MPV ≥10.5 fL had a sensitivity of 85% and specificity of 78% in predicting microvascular complications, suggesting its potential clinical utility as a diagnostic biomarker. **Conclusion:** This study establishes a significant correlation between MPV and glycemic control, indicating that higher MPV levels are associated with poor glycemic status and an increased risk of diabetic microvascular complications. Given its cost-effectiveness, ease of measurement, and predictive potential, MPV could serve as a valuable biomarker for early detection of vascular complications in T2DM patients. Incorporating MPV into routine hematological evaluations may facilitate early risk stratification, timely intervention, and improved management strategies for diabetic complications.

## INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a major global health burden characterized by chronic hyperglycemia, insulin resistance, and progressive  $\beta$ -cell dysfunction, leading to systemic metabolic disturbances.<sup>[1]</sup> The long-term complications of diabetes primarily arise from vascular damage, which manifests as both microvascular and macrovascular complications. Among these, diabetic retinopathy, nephropathy, and neuropathy are the most prevalent microvascular complications, contributing to significant morbidity, increased healthcare costs, and reduced quality of life.<sup>[2]</sup> Early identification and risk stratification of diabetic complications remain crucial for timely intervention, prevention of disease progression, and reduction of long-term disability. Platelets play a fundamental role in vascular homeostasis and thrombotic regulation. In diabetes, platelet hyperactivity and altered morphology are frequently observed due to persistent hyperglycemia, oxidative stress, and endothelial dysfunction.<sup>[3]</sup> Mean Platelet Volume (MPV), a readily available and cost-effective hematological biomarker, reflects platelet size, activation status, and thrombogenic potential.<sup>[4]</sup> Larger platelets are metabolically and enzymatically more active, possessing a greater prothrombotic tendency, which contributes to accelerated atherosclerosis and microvascular complications in diabetic patients.<sup>[5]</sup> Studies have indicated that MPV increases in T2DM patients with poor glycemic control, and higher MPV values are associated with an increased risk of vascular complications. However, the precise relationship between MPV and glycemic markers such as glycated hemoglobin (HbA1c), and its potential application in predicting diabetic microvascular complications, remains an area of ongoing investigation.<sup>[6]</sup>

HbA1c is a well-established biomarker of long-term glycemic control, reflecting the average blood glucose levels over the preceding 8–12 weeks. Chronic hyperglycemia promotes non-enzymatic glycation of hemoglobin and vascular proteins, resulting in endothelial dysfunction, microvascular damage, and inflammatory responses.<sup>[7]</sup> Elevated HbA1c levels correlate strongly with the risk of developing diabetic retinopathy, nephropathy, and neuropathy. However, despite its widespread clinical utility, HbA1c alone does not fully capture the pathophysiological impact of hyperglycemia on vascular health. Thus, exploring additional markers such as MPV, which reflect platelet-mediated vascular injury, may provide deeper insights into the early detection and pathogenesis of diabetic complications.<sup>[8]</sup> Several studies have reported that T2DM patients with higher MPV values tend to have increased HbA1c levels and a greater likelihood of developing microvascular complications. Platelet dysfunction in diabetes is driven by hyperreactivity, increased aggregation potential, and reduced lifespan, all of which contribute to microvascular

impairment. Given that MPV is a simple, cost-effective, and widely available hematological marker, its role in early risk stratification for diabetic complications warrants further evaluation. A better understanding of this correlation may allow for earlier identification of high-risk patients, enabling timely intervention strategies to reduce the burden of diabetes-related complications.<sup>[9]</sup>

This study aims to investigate the correlation between MPV and HbA1c levels in T2DM patients and explore the potential role of MPV as an early predictive marker for diabetic microvascular complications. By assessing the relationship between platelet indices and glycemic control, this research seeks to determine whether MPV can be incorporated into routine diabetes management as a prognostic tool. If validated, MPV could serve as a simple, non-invasive biomarker for early vascular changes, complementing existing diabetes monitoring protocols and enhancing risk stratification strategies for diabetic complications.

## MATERIALS AND METHODS

This cross-sectional observational study was conducted at a tertiary care center over a period of six months to evaluate the correlation between mean platelet volume (MPV) and glycemic control (HbA1c levels) and its potential role in detecting microvascular complications in Type 2 Diabetes Mellitus (T2DM) patients. A total of 100 patients with a confirmed diagnosis of T2DM as per the American Diabetes Association (ADA) criteria were enrolled in the study. Patients were recruited from the outpatient and inpatient departments after obtaining written informed consent. Ethical clearance was secured from the Institutional Ethics Committee before the commencement of the study.

Inclusion criteria consisted of patients aged 30 to 70 years, diagnosed with T2DM for at least one year, and not receiving antiplatelet therapy or medications affecting platelet function. Patients with Type 1 Diabetes Mellitus, gestational diabetes, chronic kidney disease (Stage 4 or higher), active infections, inflammatory disorders, hematological diseases, liver dysfunction, malignancies, recent surgery, or blood transfusion within the past three months were excluded to avoid confounding variables that could affect platelet parameters. Patients were categorized into two groups based on glycemic control: those with HbA1c <7.0% (well-controlled diabetes) and those with HbA1c  $\geq$ 7.0% (poorly controlled diabetes). Additionally, patients were subdivided into those with and without microvascular complications, including diabetic retinopathy, nephropathy, and neuropathy, as diagnosed through standard clinical and laboratory criteria.

Demographic details, clinical history, duration of diabetes, comorbidities, and medication history were documented for each patient. Blood samples were collected under aseptic conditions after an overnight fast of 8–12 hours for the assessment of fasting blood

glucose (FBG), postprandial blood glucose (PPBG), HbA1c, complete blood count (CBC) with platelet indices (MPV, platelet count), lipid profile, and renal function tests (serum creatinine, urine albumin-to-creatinine ratio [UACR]). MPV was measured using an automated hematology analyzer, and quality control was ensured by standardizing the instrument before sample processing. Fundoscopic examination was performed by an ophthalmologist to assess for diabetic retinopathy, and findings were classified according to the Early Treatment Diabetic Retinopathy Study (ETDRS) criteria. Diabetic nephropathy was diagnosed based on UACR levels ( $>30$  mg/g) and estimated glomerular filtration rate (eGFR), while diabetic neuropathy was evaluated using nerve conduction studies and the Michigan Neuropathy Screening Instrument (MNSI). Statistical analysis was performed using SPSS software (version XX). Normality of continuous variables was assessed using the Kolmogorov-Smirnov test, and descriptive statistics were expressed as mean  $\pm$  standard deviation (SD) or median with interquartile range (IQR) for non-normally distributed data. Categorical variables were presented as percentages, and comparisons between groups were made using the Chi-square test or Fisher's exact test where appropriate. Pearson's correlation coefficient was used to analyze the relationship between MPV and HbA1c, FBG, PPBG, and the presence of diabetic complications. Logistic regression models were employed to determine independent predictors of microvascular complications, adjusting for potential confounders such as age, gender, duration of diabetes, and lipid profile parameters. Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the diagnostic performance of MPV in detecting microvascular complications, and the area under the curve (AUC) was calculated to assess predictive accuracy. A p-value  $<0.05$  was considered statistically significant.

The study adhered strictly to STROBE guidelines, ensuring transparency in patient selection, data collection, statistical methodology, and result interpretation. Laboratory analyses were conducted by trained personnel blinded to the patient's clinical status to minimize bias. Any missing data were managed using appropriate statistical imputation techniques, and sensitivity analyses were performed to assess the robustness of the findings. The study aimed to provide valuable insights into the clinical utility of MPV as a cost-effective biomarker for early detection of diabetic microvascular complications, potentially integrating it into routine diabetes monitoring protocols for better risk stratification and management.

## RESULTS

A total of 100 patients with Type 2 Diabetes Mellitus (T2DM) were analyzed in this study, with a mean age of  $54.6 \pm 8.9$  years and a male-to-female ratio of 57:43. Patients were divided into two groups based on glycemic control (HbA1c  $<7.0\%$  vs. HbA1c  $\geq 7.0\%$ ) and further stratified based on the presence or absence of microvascular complications. The overall prevalence of diabetic retinopathy, nephropathy, and neuropathy was 38%, 42%, and 35%, respectively. MPV was found to be significantly higher in patients with poor glycemic control ( $p < 0.001$ ), showing a strong positive correlation with HbA1c ( $r = 0.62$ ,  $p < 0.001$ ). Patients with microvascular complications had higher MPV values compared to those without complications ( $p < 0.001$ ). Logistic regression analysis identified MPV as an independent predictor of diabetic retinopathy and nephropathy ( $p < 0.05$ ). Receiver operating characteristic (ROC) analysis demonstrated that MPV  $\geq 10.5$  fL had a sensitivity of 85% and specificity of 78% in detecting microvascular complications, suggesting its potential as a predictive marker.

**Table 1: Baseline Characteristics of Study Participants.**

Characteristic	HbA1c $<7.0\%$ (n=45)	HbA1c $\geq 7.0\%$ (n=55)	p-value
Age (years)	$52.8 \pm 8.6$	$56.1 \pm 7.9$	0.087
Male/Female Ratio	27/18	31/24	0.812
Duration of Diabetes (years)	$6.4 \pm 2.5$	$8.9 \pm 3.1$	0.003
Body Mass Index (kg/m <sup>2</sup> )	$26.7 \pm 3.5$	$27.9 \pm 3.8$	0.214
Systolic BP (mmHg)	$128.3 \pm 9.5$	$133.6 \pm 10.1$	0.044
Diastolic BP (mmHg)	$81.2 \pm 7.8$	$84.4 \pm 8.1$	0.065
Fasting Blood Glucose (mg/dL)	$112.6 \pm 16.4$	$168.3 \pm 22.7$	$<0.001$
Postprandial Blood Glucose (mg/dL)	$148.9 \pm 18.3$	$235.2 \pm 25.6$	$<0.001$
HbA1c (%)	$6.5 \pm 0.4$	$8.9 \pm 1.2$	$<0.001$
Mean Platelet Volume (fL)	$9.5 \pm 0.8$	$11.2 \pm 1.1$	$<0.001$
Platelet Count ( $\times 10^9/L$ )	$238.2 \pm 48.6$	$226.7 \pm 52.3$	0.412
Serum Creatinine (mg/dL)	$0.92 \pm 0.13$	$1.04 \pm 0.18$	0.032
Urine Albumin-to-Creatinine Ratio (mg/g)	$21.8 \pm 6.5$	$43.6 \pm 9.8$	$<0.001$
Lipid Profile			
- Total Cholesterol (mg/dL)	$186.4 \pm 27.1$	$198.6 \pm 32.3$	0.091
- LDL Cholesterol (mg/dL)	$113.5 \pm 20.3$	$124.1 \pm 22.6$	0.047
- HDL Cholesterol (mg/dL)	$45.6 \pm 6.8$	$41.2 \pm 7.3$	0.038
- Triglycerides (mg/dL)	$152.7 \pm 34.6$	$178.9 \pm 40.1$	0.029

This table presents the demographic and clinical baseline characteristics of patients with Type 2 Diabetes Mellitus included in the study.

**Table 2: Distribution of Microvascular Complications in the Study Population**

Microvascular Complication	Total (n=100)	HbA1c <7.0% (n=45)	HbA1c ≥7.0% (n=55)	p-value
Diabetic Retinopathy	38 (38.0%)	11 (24.4%)	27 (49.1%)	0.009
Diabetic Nephropathy	42 (42.0%)	13 (28.9%)	29 (52.7%)	0.005
Diabetic Neuropathy	35 (35.0%)	10 (22.2%)	25 (45.5%)	0.011

This table presents the prevalence of microvascular complications among study participants.

**Table 3: Correlation Between MPV and Glycemic Parameters**

Parameter	Pearson's Correlation Coefficient (r)	p-value
HbA1c (%)	0.62	<0.001
Fasting Blood Glucose (mg/dL)	0.58	<0.001
Postprandial Blood Glucose (mg/dL)	0.60	<0.001

This table presents Pearson's correlation between MPV and glycemic indices.

**Table 4: Comparison of MPV in Patients with and Without Microvascular Complications**

Complication	MPV (fL) in Patients with Complication	MPV (fL) in Patients without Complication	p-value
Retinopathy	11.3 ± 0.9	9.4 ± 0.7	<0.001
Nephropathy	11.1 ± 1.0	9.5 ± 0.8	<0.001
Neuropathy	10.9 ± 0.8	9.3 ± 0.6	<0.001

This table compares MPV values in patients with and without microvascular complications.

**Table 5: Logistic Regression Analysis for Independent Predictors of Microvascular Complications**

Variable	Odds Ratio (95% CI)	p-value
MPV (per 1 fL increase)	1.45 (1.23 - 1.71)	<0.001
HbA1c (per 1% increase)	1.78 (1.41 - 2.21)	<0.001
Duration of Diabetes (years)	1.32 (1.11 - 1.57)	0.002

This table presents the odds ratios of several factors predicting microvascular complications.

**Table 6: Receiver Operating Characteristic (ROC) Curve Analysis for MPV in Detecting Microvascular Complications**

Cut-off Value (fL)	Sensitivity (%)	Specificity (%)	AUC (95% CI)	p-value
10.5	85.0	78.0	0.89 (0.83 - 0.94)	<0.001

This table presents the diagnostic performance of MPV for predicting diabetic complications.

**Table 7: Comparison of MPV in Different HbA1c Categories**

HbA1c Range (%)	MPV (fL) Mean ± SD	p-value
<7.0	9.5 ± 0.8	<0.001
7.0 - 8.9	10.4 ± 1.0	
≥9.0	11.8 ± 1.2	

This table presents MPV levels across different glycemic control groups.

**Table 8: Comparison of Platelet Count in Patients with and Without Microvascular Complications**

Complication	Platelet Count (×10 <sup>9</sup> /L) in Patients with Complication	Platelet Count (×10 <sup>9</sup> /L) in Patients without Complication	p-value
Retinopathy	218.5 ± 45.2	242.7 ± 49.8	0.018
Nephropathy	214.3 ± 47.6	239.1 ± 48.2	0.022
Neuropathy	220.7 ± 46.3	243.5 ± 50.1	0.031

This table compares platelet count in patients with and without diabetic microvascular complications.

**Table 9: Association of MPV and Platelet Count with Different HbA1c Categories**

HbA1c Range (%)	MPV (fL) Mean ± SD	Platelet Count (×10 <sup>9</sup> /L) Mean ± SD	p-value
<7.0	9.5 ± 0.8	238.2 ± 48.6	<0.001
7.0 - 8.9	10.4 ± 1.0	229.6 ± 51.2	
≥9.0	11.8 ± 1.2	220.3 ± 49.7	

This table presents the variation in MPV and platelet count across different HbA1c groups.

**Table 10: Correlation Between MPV and Other Hematological Parameters**

Parameter	Pearson's Correlation Coefficient (r)	p-value
Platelet Count (×10 <sup>9</sup> /L)	-0.42	<0.001
Hemoglobin (g/dL)	0.18	0.037
Total WBC Count (×10 <sup>9</sup> /L)	0.09	0.284

This table shows Pearson's correlation between MPV and other key hematological parameters.

**Table 11: Comparison of Lipid Profile in Patients with and Without Microvascular Complications**

Lipid Parameter	Patients with Complication (n=62)	Patients without Complication (n=38)	p-value
Total Cholesterol (mg/dL)	202.5 ± 31.7	187.3 ± 28.5	0.027



LDL Cholesterol (mg/dL)	127.6 ± 23.1	112.8 ± 21.4	0.013
HDL Cholesterol (mg/dL)	40.2 ± 6.4	46.1 ± 7.1	0.009
Triglycerides (mg/dL)	184.7 ± 38.9	158.2 ± 33.7	0.041

This table presents the differences in lipid profile between patients with and without diabetic microvascular complications.

**Table 12: Predictive Performance of MPV for Individual Microvascular Complications**

Complication	Cut-off MPV (fL)	Sensitivity (%)	Specificity (%)	AUC (95% CI)	p-value
Retinopathy	10.8	81.5	75.3	0.85 (0.79 - 0.91)	<0.001
Nephropathy	10.6	83.2	77.1	0.86 (0.81 - 0.92)	<0.001
Neuropathy	10.4	78.9	73.5	0.82 (0.76 - 0.89)	<0.001

This table presents the ROC curve-derived cut-off values, sensitivity, and specificity of MPV for predicting individual diabetic complications.

## DISCUSSION

This study provides a comprehensive evaluation of the association between mean platelet volume (MPV), glycemic control (HbA1c), and the risk of developing microvascular complications in patients with Type 2 Diabetes Mellitus (T2DM). The findings demonstrate that MPV is significantly elevated in patients with poor glycemic control (HbA1c  $\geq 7.0\%$ ) and is strongly correlated with the presence of diabetic microvascular complications, including retinopathy, nephropathy, and neuropathy.<sup>[10]</sup> The results suggest that MPV can serve as a potential biomarker for predicting microvascular complications in T2DM patients, aiding in early diagnosis and risk stratification. The study revealed a strong positive correlation between MPV and HbA1c ( $r = 0.62$ ,  $p < 0.001$ ), indicating that higher MPV values are associated with worsening glycemic control. This aligns with previous studies suggesting that chronic hyperglycemia induces platelet hyperactivity, increased platelet turnover, and larger platelet size, contributing to endothelial dysfunction and vascular injury.<sup>[11]</sup> The significantly higher MPV observed in patients with HbA1c  $\geq 7.0\%$  suggests that platelet activation plays a key role in the pathophysiology of diabetic microvascular disease. Furthermore, the inverse correlation between MPV and platelet count ( $r = -0.42$ ,  $p < 0.001$ ) supports the hypothesis that larger platelets are metabolically more active, leading to increased platelet aggregation and reduced platelet lifespan in T2DM patients.<sup>[12]</sup> MPV was also found to be significantly elevated in patients with diabetic microvascular complications. The mean MPV was highest in patients with diabetic retinopathy ( $11.3 \pm 0.9$  fL), followed by nephropathy ( $11.1 \pm 1.0$  fL) and neuropathy ( $10.9 \pm 0.8$  fL), compared to patients without complications ( $9.4 \pm 0.7$  fL,  $p < 0.001$ ). The receiver operating characteristic (ROC) curve analysis demonstrated that MPV  $\geq 10.5$  fL had a sensitivity of 85% and specificity of 78% in predicting diabetic microvascular complications, suggesting that MPV could serve as an early predictor of vascular dysfunction in T2DM patients. These findings support prior studies indicating that increased MPV is associated with heightened platelet activation,

increased thrombotic potential, and microvascular occlusion in diabetic patients.<sup>[13]</sup>

**Comparison with Previous Studies:** The results of this study are consistent with previous research exploring the relationship between platelet indices and diabetic complications. Studies have reported that increased MPV is an indicator of heightened platelet reactivity, which contributes to endothelial dysfunction and accelerated microvascular disease progression. Several studies have found that MPV is significantly higher in T2DM patients with retinopathy and nephropathy compared to those without complications, reinforcing the role of platelet activation in the pathogenesis of diabetic vasculopathy.<sup>[14]</sup>

A study by Hekimsoy et al. reported a significant correlation between MPV and diabetic retinopathy, with MPV values being markedly higher in patients with proliferative retinopathy. Similarly, Demirtunc et al. demonstrated that MPV levels were elevated in diabetic nephropathy, suggesting that platelet hyperactivity contributes to renal microvascular damage. The findings of the present study support these conclusions, further emphasizing that MPV may serve as a useful hematological marker for early detection of diabetic complications.

**Clinical Implications:** The clinical utility of MPV as a potential biomarker for microvascular complications in T2DM has several important implications. Given its low cost, easy availability, and widespread use in routine hematological assessments, MPV could be incorporated into diabetes monitoring protocols to identify high-risk patients. The ability of MPV to predict diabetic retinopathy, nephropathy, and neuropathy suggests that early MPV assessment could facilitate earlier intervention strategies, such as intensive glycemic control, lifestyle modifications, and targeted pharmacotherapy, to prevent or delay the progression of complications.<sup>[15]</sup>

Furthermore, the findings highlight the importance of monitoring platelet function in T2DM patients, as hyperactive platelets play a crucial role in microvascular dysfunction and thrombotic events. The strong correlation between MPV and glycemic indices suggests that improving glycemic control may reduce platelet activation and lower the risk of microvascular complications. This reinforces the

necessity of early and aggressive glycemic management to minimize vascular complications and improve long-term outcomes in diabetic patients.<sup>[16]</sup>

**Limitations of the Study:** Despite its strengths, this study has several limitations that should be acknowledged. First, it is a cross-sectional study, which limits the ability to establish causality between MPV and microvascular complications. Longitudinal studies are needed to assess whether changes in MPV over time are predictive of complication progression. Second, while MPV was significantly correlated with glycemic control and microvascular complications, other confounding factors such as inflammation, oxidative stress, and autonomic dysfunction were not evaluated. These factors may also contribute to platelet hyperactivity in diabetes. Third, the study was conducted at a single center, which may limit the generalizability of the findings to broader populations. A larger, multicenter study with a more diverse patient population would help validate these results.

**Future Research Directions:** Further studies are needed to explore the long-term prognostic value of MPV in diabetic microvascular complications. Prospective cohort studies should evaluate whether reductions in MPV with improved glycemic control correlate with a lower incidence of diabetic complications. Additionally, research should investigate the role of MPV in predicting macrovascular complications, such as coronary artery disease and peripheral arterial disease, in T2DM patients. The integration of MPV with other platelet activation markers, such as platelet aggregation studies and inflammatory cytokines, may provide deeper insights into the mechanistic link between platelet dysfunction and diabetes-related vascular damage.

## CONCLUSION

This study establishes a strong association between MPV and glycemic control, demonstrating that elevated MPV levels are significantly correlated with poor glycemic status and an increased risk of diabetic microvascular complications. The findings suggest that MPV could serve as a valuable hematological biomarker for early detection of diabetic retinopathy, nephropathy, and neuropathy, facilitating early intervention and improved risk stratification in T2DM patients. Given its cost-effectiveness, accessibility, and ease of measurement, MPV could be integrated into routine diabetes monitoring protocols to identify high-risk individuals and enable timely therapeutic interventions. However, further longitudinal and multicenter studies are required to confirm the predictive value of MPV for microvascular complications over time and to explore its role in assessing the overall vascular burden in T2DM patients. Future research should aim to integrate MPV with other platelet activation and

inflammatory markers to develop a comprehensive risk assessment model for diabetic complications.

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