

EVALUATION OF PLATELET INDICES IN TYPE 2 DIABETES MELLITUS PATIENTS

Dr. Vinaya V Ingavale^{1*}, Dr. Paresh Marathe², Dr. Sujata Agrawal³, Dr. Maya Parihar Malhotra⁴

¹Assistant Professor, Pathology, Topiwala National Medical College, Mumbai

Email ID – vinayaingavale@gmail.com

Orchid ID- 0009-0009-2812-0497

²Associate Professor, Pathology, Bombay Hospital Institute of Medical Sciences

Email ID – bhims90@reddiffmail.com

Orchid ID- 0000-0002-9254-3621

³Senior resident, Pathology, Institute of Medical Sciences, BHU

Email ID – dr.sujata555@gmail.com

Orchid ID- 0009-0005-0598-6141

⁴Associate Professor, Pathology, Bombay Hospital Institute of Medical Sciences

Phone Number: 9820302628

Email ID – bhims90@reddiffmail.com

***Corresponding author: Dr Vinaya V Ingavale, Email ID: vinayaingavale@gmail.com**

ABSTRACT

Background: Type 2 diabetes mellitus is characterized by a connection with increased risk of vascular complications due to enhanced platelet activation. Platelet-related measures like mean platelet volume (MPV) and platelet distribution width (PDW), and plateletcrit (PCT) are simple and cost-effective markers of platelet activity.

Objective: To evaluate platelet profile alterations in patients with type 2 diabetes mellitus and compare them with non-diabetic controls.

Materials and Methods: This hospital-based case-control study included 200 subjects, comprising One hundred patients suffering from type 2 diabetes mellitus and 100 age- and sex-matched healthy controls. Assessment of platelet parameters was carried out using an automated hematology analyzer. Glucose profile variables such as fasting blood glucose (FBS), postprandial blood sugar (PPBS), and HbA1c were also assessed. Data analysis was conducted statistically using unpaired t-test.

Results: Mean platelet volume was significantly higher in diabetic patients compared to controls ($p=0.031$). No substantial modification was observed in PDW, PCT, or platelet count. Glycemic parameters (FBS, PPBS, HbA1c) were significantly elevated in diabetic patients ($p<0.001$). **Conclusion:** Increased MPV in type 2 diabetes mellitus reflects enhanced platelet activation and may serve as a simple, cost-effective marker for early detection of vascular complications.

Keywords: Type 2 diabetes mellitus, Mean platelet volume, Platelet indices, PDW, HbA1c

How to cite this article: Ingavale VV, Marathe P, Agrawal S, Malhotra MP., Evaluation of Platelet Indices in Type 2 Diabetes Mellitus Patients. Int J Drug Deliv Technol. 2026;16(7): 1499-1503; DOI: 10.25258/ijddt.16.7.161

INTRODUCTION

Diabetes mellitus represents a long-standing metabolic illness characterized by A metabolic condition involving persistent high blood sugar resulting from defective insulin production or action, or both.¹ It represents a major and rapidly growing universal wellbeing problem. Rendering to recent estimates, the pervasiveness of diabetes has mounted vividly over the last 10yrs, with type 2 diabetes mellitus (T2DM) accounting for nearly 90-95% of all cases.² The disease imposes a significant burden on healthcare systems due to its long-term complications and associated indisposition and death.

The chronic hyperglycemic state in diabetes leads to a variety of complications, broadly categorized into microvascular complications and macrovascular complications including cerebrovascular disease, and peripheral vascular disease.³ Among these, cardiovascular complications remain the one of the leading causes of death in patients with T2DM.⁴ The underlying

mechanisms responsible for these complications are complex and multifactorial, involving metabolic, hemodynamic, and inflammatory pathways.^{3,4}

One of the key contributors to the advancement of vessel related diseases complications in diabetes is platelet dysfunction. Platelets have a key role in hemostasis; however, in diabetic individuals, they exhibit increased reactivity and aggregation tendencies. This enhanced platelet activity contributes to a prothrombotic state, predisposing patients to thrombotic events.⁵ Several factors such as hyperglycemia, insulin resistance, oxidative stress, and endothelial dysfunction have been implicated in altering platelet behavior in diabetes.⁶

Hyperglycemia induces non-enzymatic glycation of platelet surface proteins, leading to structural and functional alterations. It also adds oxidative strain, which further enhances platelet activation. Insulin normally exerts an inhibitory effect on platelet aggregation; however, in insulin-resistant states, this protective mechanism is impaired, resulting in increased platelet

aggregation.⁵ Additionally, endothelial dysfunction reduces the production of antithrombotic factors such as nitric oxide and prostacyclin, further promoting platelet activation.⁷

In recent years, there has been growing interest in the use of platelet indices as markers of platelet activation. Platelet indices, which include MPV, PDW and PCT are routinely available as part of automated complete blood count analysis.⁸ These guides provide ancillary data about platelet geomorphology and activity and can be obtained without additional cost or specialized testing.

MPV is considered one of the most reliable indicators of platelet activation.⁸ Bigger platelets are considered younger, extra metabolically active, and contain higher levels of prothrombotic substances such as thromboxane A2.⁹ Consequently, raised MPV has been correlated with increased peril of vascular snags in various clinical conditions, including diabetes. PDW reflects variability in platelet size and is an indicator of platelet anisocytosis, while plateletcrit (PCT) represents the total circulating platelet mass. Although MPV has been extensively studied, the clinical significance of PDW and PCT in diabetes remains less clearly defined.¹⁰

Several studies have reported an association between elevated MPV and poor glycaemic regulator, together with an elevated risk of diabetic complications.¹¹⁻¹³ However, the findings regarding PDW and PCT have been inconsistent, and further research is required to establish their clinical utility. Moreover, variations in research inhabitants and methodologies necessitate additional studies to validate these factors in different clinical settings.

Given the increasing burden of diabetes and its complications, there is a need for simple, reliable, and cost-effective biomarkers that can aid in early detection and risk assessment. Platelet indices fulfill these criteria and may serve as useful tools in routine clinical practice.⁹ Their evaluation could help identify patients at higher risk of developing vascular complications, thereby facilitating early intervention and improved management.³

Despite the growing body of evidence on platelet indices in diabetes, there remains inconsistency in findings, particularly regarding PDW and plateletcrit (PCT). Moreover, limited data are available from the Indian population, especially in hospital-based settings where early identification of thrombotic risk is crucial. The current experiment aims to address these gaps by comprehensively evaluating multiple platelet indices in patients with type 2 diabetes mellitus and comparing them with well-matched non-diabetic controls, thereby contributing to the existing literature and assessing their potential utility as simple and cost-effective markers in routine clinical practice.

Therefore, the ongoing experiment was undertaken to evaluate platelet indices in patients with type 2 diabetes mellitus and to compare them with non-diabetic controls. The study also aims to assess the potential role of these indices as markers of platelet activation and their association with glycemic status.

MATERIALS AND METHODS

Study Design and Setting:

This was a hospital-based observational case-control study operated in the Department of Pathology in collaboration with the Departments of Medicine and Biochemistry at a tertiary care centre. The study was carried out over a period of two years from 2016 to 2018.

Ethical Considerations:

Approval for the study protocol was obtained from the Institutional Ethics Committee (BH-EC-013 Approval number) prior to commencement. Written informed consent was obtained from all participants included in the study.

Study Population:

A total of 200 subjects were enrolled and divided into two groups:

- **Group A (Cases):** 100 patients diagnosed with type 2 diabetes mellitus
- **Group B (Controls):** 100 apparently healthy, non-diabetic individuals

The sample size was calculated based on detecting a significant difference in mean platelet volume (MPV) between patients with type 2 diabetes mellitus and healthy controls. Using data from previous studies (Hekimsoy et al.), the mean MPV in diabetic patients and controls was assumed to be 8.9 ± 1.2 fL and 8.3 ± 1.0 fL, respectively.

The study sample size was computed using the formula for comparison of two means:

$$n = 2 \times (Z\alpha/2 + Z\beta)^2 \times \sigma^2 / d^2$$

Where:

$$\begin{aligned} Z\alpha/2 &= 1.96 && \text{(for 95\% confidence interval)} \\ Z\beta &= 0.84 && \text{(for 80\% power)} \\ \sigma &= \text{pooled standard deviation (assumed as 1.1)} \\ d &= \text{expected mean difference (0.6 fL)} \end{aligned}$$

Substituting these values, the least size of the sample required in each group was approximately 85 subjects. Considering possible variability and to increase the power of the study, a total of 100 subjects were included in each group, making the overall sample size 200.

Controls were selected to be age- and sex-matched with the cases to minimize confounding variables.

Inclusion Criteria:

Patients were included in the diabetic group based on the following diagnostic criteria for type 2 diabetes mellitus:

- Fasting blood sugar (FBS) ≥ 126 mg/dL, or
- Postprandial blood sugar (PPBS) ≥ 200 mg/dL, or
- Glycated hemoglobin (HbA1c) $\geq 6.5\%$, or
- Presence of classical symptoms of diabetes with random blood glucose ≥ 200 mg/dL

For the control group, healthy individuals with:

- FBS between 70-100 mg/dL
- PPBS ≤ 120 mg/dL
- HbA1c $< 6.5\%$

were included.

Exclusion Criteria

The study excluded individuals with the following conditions: Anemia

- Leukopenia or leukocytosis
- Thrombocytopenia or thrombocytosis
- Known hematological disorders
- Chronic infections such as HIV, Hepatitis B, and Hepatitis C
- Autoimmune disorders such as systemic lupus erythematosus
- History of thrombosis
- Pregnancy
- Smoking

Additionally, patients with comorbid conditions such as hypertension, hypercholesterolemia, malignancy, acute febrile illness, and ischemic heart disease were excluded to avoid confounding effects on platelet indices.

Data Collection:

A thorough medical history was recorded from all participants using a structured proforma. Demographic details including age and gender were recorded. Relevant scientific and test site parameters were documented.

Sample Collection and Processing:

Venous blood was collected from the participants under aseptic precautions after an overnight fast of 10-12 hours. Samples were collected in ethylenediaminetetraacetic acid (EDTA) tubes for hematological analysis and in appropriate tubes for biochemical investigations.

All samples were processed within 2 hours of collection to ensure accuracy and reliability of results.

Laboratory Analysis

• Hematological Parameters:

Platelet indices including MPV, PDW, PCT, and platelet count were measured using an automated hematology analyzer (Beckman Coulter LH 750), which operates on the principle of electrical impedance and VCS (Volume, Conductivity, and Scatter) technology.

Peripheral blood smear examination was performed in selected cases to validate platelet counts and to rule out spurious results due to platelet clumping or presence of giant platelets.

2. BIOCHEMICAL PARAMETERS

- Fasting blood sugar (FBS) and postprandial blood sugar (PPBS) were estimated using the glucose oxidase method.
- Glycated hemoglobin (HbA1c) was measured using high-performance liquid chromatography (HPLC).
- Serum creatinine was estimated using a spectrophotometric method.
- Total cholesterol was analyzed using an automated biochemical analyzer.

Quality Control:

All laboratory investigations were carried out following standard operating procedures. Internal quality control measures were maintained to ensure accuracy and precision of results.

Statistical Analysis:

Data obtained from the study were entered into Microsoft Excel and analyzed using appropriate statistical software. Quantitative variables were expressed as mean $\pm$ standard deviation (SD).

The difference between the two groups was analyzed using the unpaired Student's t-test for normally distributed data. For non-normally distributed data, the Mann-Whitney U test was applied.

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

RESULTS

A total of 200 subjects were included in the present study, comprising 100 patients with type 2 diabetes mellitus (Group A) and 100 healthy non-diabetic controls (Group B). The baseline demographic and laboratory parameters of both groups were analyzed and compared.

Demographic Characteristics

The mean age of patients in Group A was **45.44 $\pm$ 8.07 years**, while in Group B it was **44.13 $\pm$ 8.04 years**. The difference in mean age between the two groups was statistically not significant ($p=0.25$), indicating that both groups were comparable in terms of age.

With respect to gender distribution, Group A consisted of **56 males and 44 females**, whereas Group B included **52 males and 48 females**. The gender distribution between the two groups was comparable. **Table 1** summarizes the demographic characteristics of the study population.

Demographic characteristics of study population

Parameter	Group A (Cases)	Group B (Controls)	p-value	Significance
Age (years)	45.44 $\pm$ 8.07	44.13 $\pm$ 8.04	0.25	NS
Gender (M:F)	56:44	52:48	—	—

Glycemic Parameters

The mean fasting blood sugar (FBS) level in Group A was **165.4 $\pm$ 33.17 mg/dL**, compared to **100.53 $\pm$ 12.75 mg/dL** in Group B. The difference was statistically significant ($p<0.001$).

Similarly, the mean postprandial blood sugar (PPBS) level was **212.96 $\pm$ 46.35 mg/dL** in diabetics and **118.32 $\pm$ 12.58 mg/dL** in controls, which was also statistically significant ($p<0.001$).

The mean glycated hemoglobin (HbA1c) was **8.566 $\pm$ 1.795%** in Group A and **5.62 $\pm$ 0.34%** in Group B, showing a highly significant difference ($p<0.001$).

These findings confirm poor glycemic control in the diabetic group.

Comparison of glycemic parameters

Parameter	Group A (Cases)	Group B (Controls)	p-value	Significance
FBS (mg/dL)	165.4 $\pm$ 33.17	100.53 $\pm$ 12.75	<0.001	Significant
PPBS (mg/dL)	212.96 $\pm$ 46.35	118.32 $\pm$ 12.58	<0.001	Significant
HbA1c (%)	8.566 $\pm$ 1.795	5.62 $\pm$ 0.34	<0.001	Significant

Biochemical Parameters

The mean total cholesterol level in Group A was **163.95 $\pm$ 22.44 mg/dL**, whereas in Group B it was **158.22 $\pm$ 22.74 mg/dL**. The difference was not statistically significant ($p=0.074$).

The mean serum creatinine level was **0.843 $\pm$ 0.192 mg/dL** in diabetics and **0.771 $\pm$ 0.146 mg/dL** in controls. This difference was statistically significant ($p=0.0032$).

Comparison of biochemical parameters

Parameter	Group A (Cases)	Group B (Controls)	p-value	Significance
Total Cholesterol (mg/dL)	163.95 $\pm$ 22.44	158.22 $\pm$ 22.74	0.074	NS
Serum Creatinine (mg/dL)	0.843 $\pm$ 0.192	0.771 $\pm$ 0.146	0.0032	Significant

Platelet Parameters

The platelet count in Group A was **271.9 $\pm$ 76.75 $\times 10^9/L$** , while in Group B it was **273.7 $\pm$ 60.31 $\times 10^9/L$** . The difference was not statistically significant ($p=0.8536$).

in diabetic patients compared to **8.416 $\pm$ 0.985 fL** in controls. This difference was statistically significant ($p=0.031$).

Platelet distribution width (PDW) was **16.82 $\pm$ 0.6046 fL** in Group A and **16.75 $\pm$ 0.6142 fL** in Group B. The difference was not statistically significant ($p=0.4656$).

Mean platelet volume (MPV) was found to be **8.795 $\pm$ 1.437 fL**

Plateletcrit (PCT) was $0.2359 \pm 0.0665\%$ in diabetics and $0.24184 \pm 0.059\%$ in controls, which was also not statistically significant ($p=0.51$).

Comparison of platelet indices

Parameter	Group A (Cases)	Group B (Controls)	p-value	Significance
Platelet Count ($\times 10^9/L$)	271.9 ± 76.75	273.7 ± 60.31	0.8536	NS
MPV (fL)	8.795 ± 1.437	8.416 ± 0.985	0.031	Significant
PDW (fL)	16.82 ± 0.6046	16.75 ± 0.6142	0.4656	NS
PCT (%)	0.2359 ± 0.0665	0.24184 ± 0.059	0.51	NS

DISCUSSION

Type 2 diabetes mellitus is a chronic metabolic disorder associated with significant morbidity and mortality, primarily due to its vascular complications.² Both microvascular complications such as retinopathy, nephropathy, and neuropathy, and macrovascular complications including coronary artery disease and stroke, contribute substantially to disease burden.^{3,4} The pathogenesis of these complications is complex and involves persistent hyperglycemia, oxidative stress, endothelial dysfunction, and inflammatory processes.⁵

Chronic hyperglycemia plays a central role in the development of vascular complications by inducing biochemical and structural alterations in the vascular endothelium. Increased production of reactive oxygen species, formation of advanced glycation end products, and activation of protein kinase pathways contribute to endothelial damage.¹⁴ This results in impaired vasodilation, increased vascular permeability, and a prothrombotic state.

In addition to endothelial dysfunction, alterations in blood components also play a crucial role in the pathophysiology of diabetes. Among these, platelets have gained considerable attention due to their role in thrombosis and hemostasis.¹⁵ In diabetic patients, platelets exhibit increased reactivity, enhanced aggregation, and altered adhesion properties. These changes contribute significantly to the prothrombotic milieu observed in diabetes and are implicated in the development of vascular complications.⁶

Platelet activation in diabetes is influenced by several factors, including hyperglycemia, insulin resistance, and oxidative stress. Hyperglycemia leads to non-enzymatic glycation of platelet proteins and membrane lipids, resulting in structural and functional changes.⁷ Insulin resistance reduces the inhibitory effect of insulin on platelet aggregation, while oxidative stress further enhances platelet activation. As a result, platelets in diabetic patients tend to be larger, more reactive, and more prone to aggregation.^{7,16}

In the present study, mean platelet volume (MPV), which is a marker of platelet size and activity, was found to be significantly increased in patients with type 2 diabetes mellitus compared to non-diabetic controls. This finding suggests the presence of larger and more metabolically active platelets in diabetic individuals. Larger platelets contain more dense granules and produce higher levels of prothrombotic substances such as thromboxane A₂, thereby contributing to increased thrombotic risk.^{15,17}

The findings of increased MPV in this study are in accordance with previous studies. Hekimsoy et al.¹⁸ reported significantly elevated MPV values in diabetic patients, indicating increased platelet activation. Similarly, Demirtunc et al.¹⁹ demonstrated a positive association between MPV and glycemic control, with higher MPV observed in patients with poor glycemic control. These observations support the concept that platelet activation is closely linked to hyperglycemia and metabolic status in diabetes.^{18,19}

The possible mechanisms underlying increased MPV in diabetic patients include increased platelet turnover and release of larger, younger platelets from the bone marrow. Hyperglycemia-induced osmotic changes may also contribute to platelet swelling.

Additionally, chronic metabolic stress may alter megakaryocyte function, leading to the production of larger platelets.²⁰

In contrast, platelet distribution width (PDW) and plateletcrit (PCT) did not show statistically significant differences between diabetic patients and controls in the present study. PDW reflects variability in platelet size and is considered a marker of platelet anisocytosis, whereas PCT represents the total platelet mass. The lack of significant difference in these parameters suggests that platelet activation in diabetes may be more closely related to changes in platelet size rather than overall platelet mass or variability.

Similarly, platelet count did not differ significantly between the two groups, indicating that quantitative platelet changes are less prominent than qualitative alterations in platelet function in diabetes. These findings emphasize that platelet activation in diabetes is primarily functional rather than numerical.

The glycemic parameters, including fasting blood sugar, postprandial blood sugar, and HbA_{1c}, were significantly elevated in diabetic patients, confirming poor glycemic control. Although correlation analysis was not performed, previous studies have demonstrated a positive association between HbA_{1c} levels and platelet indices, particularly MPV. This suggests that poor glycemic control may contribute to increased platelet activation.²¹

The present study highlights the potential utility of MPV as a simple and cost-effective marker of platelet activation in type 2 diabetes mellitus. Since MPV is routinely available as part of complete blood count analysis, it can serve as a convenient tool for identifying patients at increased risk of thrombotic complications.

However, certain limitations should be considered. The study was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. Furthermore, correlation of platelet indices with duration of diabetes and presence of complications was not assessed. Future studies with larger populations and longitudinal follow-up are needed to further establish the predictive value of platelet indices.

Overall, the findings of this study support the role of platelet activation in the pathophysiology of type 2 diabetes mellitus and underscore the clinical significance of platelet indices, particularly mean platelet volume, as potential markers for assessing thrombotic risk in these patients.

CONCLUSION

The present study demonstrates that mean platelet volume is significantly increased in patients with type 2 diabetes mellitus, indicating enhanced platelet activation. In contrast, platelet distribution width, plateletcrit, and platelet count did not show significant differences between diabetic and non-diabetic individuals.

These findings suggest that qualitative changes in platelet function, rather than quantitative alterations, play a key role in the prothrombotic state associated with diabetes. Mean platelet volume, being a simple, cost-effective, and routinely available parameter, may serve as a useful marker for early detection of platelet activation and assessment of thrombotic risk in patients with type 2 diabetes mellitus.

Further large-scale and longitudinal studies are recommended to validate the clinical utility of platelet indices in predicting vascular complications in diabetes.

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