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Study of correlation between mean platelet volume and HbA1c in type 2 diabetes mellitus with special reference to microvascular complications

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Abstract

Background: Type 2 diabetes mellitus (T2DM) is associated with platelet hyperreactivity and an increased risk of vascular complications. Mean platelet volume (MPV), a routinely reported platelet index, reflects platelet size and may indirectly indicate platelet reactivity. This study evaluated the relationship between MPV and HbA1c and compared MPV between patients with T2DM and non-diabetic controls, with particular reference to microvascular complications.

Methods: This comparative cross-sectional study was conducted at Dhiraj General Hospital from January 2024 to May 2025. One hundred patients with T2DM of more than five years' duration and 100 demographically matched non-diabetic controls were evaluated. Patients receiving antiplatelet drugs, those with specified anaemia, and participants meeting other predefined exclusion criteria were excluded. Clinical characteristics, blood pressure, fasting and postprandial blood glucose, HbA1c, serum creatinine, platelet count, and MPV were assessed. Diabetic participants were evaluated for microvascular complications. MPV was measured using an automated haematology analyser.

Results: Mean MPV was significantly higher in patients with T2DM than in controls (12.017 ± 1.227 fL vs 9.737 ± 1.365 fL; $p<0.001$), whereas platelet counts did not differ significantly ($p=0.201$). Patients with diabetes also had significantly higher HbA1c, fasting and postprandial glucose, serum creatinine, systolic blood pressure, and diastolic blood pressure. MPV was significantly higher among diabetic participants with microvascular complications ($p=0.0139$). In the subgroup analysis, HbA1c <6.5% showed $r=0.2645$ ($p=0.0350$), whereas HbA1c ≥6.5% showed $r=-0.428$ ($p=0.0426$).

Conclusion: MPV was significantly higher in patients with T2DM and was associated with the presence of microvascular complications. Because MPV is inexpensive and routinely available with complete blood count testing, it may have potential as an adjunctive marker of vascular risk. However, these findings do not establish causality or support MPV as a standalone diagnostic or prognostic marker. Larger prospective studies with standardised laboratory methods and adjustment for relevant confounding factors are warranted.

Keywords: mean platelet volume; HbA1c; type 2 diabetes mellitus; platelet indices; microvascular complications; glycaemic control.

Introduction

Diabetes mellitus is a common chronic metabolic disorder characterised by persistent hyperglycaemia and progressive disturbances in multiple physiological systems. Long-term hyperglycaemia is associated with microvascular and macrovascular complications that substantially contribute to morbidity, disability, and premature mortality. Diabetic retinopathy, nephropathy, and neuropathy are major microvascular complications and are influenced by duration of diabetes, glycaemic control, blood pressure, body weight, and other cardiovascular risk factors [1-4].

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In addition to abnormalities of glucose metabolism, diabetes is associated with endothelial dysfunction, oxidative stress, chronic low-grade inflammation, altered coagulation, and increased platelet reactivity. Platelets from patients with diabetes may demonstrate enhanced adhesion, activation, aggregation, and thromboxane production, contributing to the prothrombotic state observed in many individuals with diabetes [5,6].

Mean platelet volume is a routinely available platelet index reported by automated haematology analysers. It represents the average size of circulating platelets and is influenced by platelet production, maturation, and turnover. Larger platelets are generally younger and metabolically more active and contain greater amounts of dense granules and prothrombotic mediators. Consequently, increased MPV has been investigated as a surrogate marker of increased platelet reactivity and cardiovascular risk [7,8].

Several studies have reported increased MPV in patients with T2DM compared with individuals without diabetes [1,2,9]. A meta-analysis also reported higher MPV and platelet distribution width in patients with T2DM, while platelet counts did not consistently differ between diabetic and non-diabetic populations [10]. These observations suggest that qualitative or functional changes in circulating platelets may occur even when the absolute platelet count remains within the usual reference range.

The relationship between glycaemic control and platelet indices is of particular interest. HbA1c reflects average glycaemic exposure over approximately two to three months and is widely used for assessment of glycaemic control. Previous studies have examined whether higher HbA1c is accompanied by increased MPV, although findings have not been uniform [11,12]. Differences in patient characteristics, duration of diabetes, laboratory methods, medication use, and pre-analytical handling may contribute to these inconsistencies.

Microvascular complications provide an additional clinical context in which platelet indices can be evaluated. Increased platelet activation may contribute to endothelial injury and microvascular obstruction, while vascular disease itself may influence platelet turnover and activation. Studies have reported associations between MPV and diabetic retinopathy, nephropathy, and other vascular complications, although not all investigations have demonstrated a significant relationship [13-16].

MPV has potential advantages because it is obtained from a routine complete blood count without requiring an additional specialised assay. Nevertheless, its interpretation requires attention to the anticoagulant used, storage conditions, time between venepuncture and analysis, and analyser characteristics. MPV should therefore be interpreted with other clinical and laboratory findings rather than in isolation.

The present study was undertaken to assess the association between HbA1c and MPV in patients with T2DM, compare MPV between diabetic and non-diabetic participants, and determine whether MPV differs according to the presence of diabetic microvascular complications.

The primary objective was to assess the relationship between HbA1c and MPV in individuals with T2DM. Secondary objectives were to compare MPV between patients with T2DM and non-diabetic controls and to compare MPV between diabetic participants with and without microvascular complications.

Materials and methods

This hospital-based comparative study was conducted at Dhiraj General Hospital from January 2024 to May 2025. The study population consisted of adults with established T2DM and non-diabetic controls. Participants were selected according to predefined eligibility criteria, and relevant clinical and laboratory variables were recorded at evaluation.

The diabetic group comprised patients with a documented diagnosis of T2DM for more than five years. The control group comprised individuals without diabetes and without a known history of diabetic microvascular complications. The original sample-size calculation indicated a minimum requirement of 98 participants in each group; 100 participants were ultimately included in each group, giving a total study population of 200.

Participants in the diabetic group were eligible when they had established T2DM for more than five years. Control participants were required to have no history of diabetes and normal blood glucose values according to the study records.

Participants were excluded if haemoglobin was below 13 g/dL in males or below 12 g/dL in females, if non-diabetic participants had electrocardiographic evidence suggestive of coronary artery disease, or if diabetic participants had a disease duration of less than five years. Individuals receiving antiplatelet medications such as aspirin or clopidogrel were also excluded.

All participants underwent clinical assessment including relevant medical history, medication history, and evaluation for microvascular complications. Height

and weight were recorded and blood pressure was measured. Personal history of addiction including smoking, alcohol and tobacco chewing was recorded. Venous blood samples were collected for complete blood count, fasting blood glucose, postprandial blood glucose, HbA1c, and serum creatinine.

Samples for complete blood count and MPV assessment were collected in dipotassium EDTA tubes and analysed within one hour of collection to minimise changes associated with sample ageing. Blood samples for plasma glucose were collected in sodium fluoride-containing tubes. MPV and platelet count were obtained using an automated haematology analyser.

Diabetic participants were evaluated for diabetic retinopathy, nephropathy, and neuropathy using the clinical records and investigations available at the study centre. Diabetic nephropathy was diagnosed based on s.creatinine (eGFR) levels, creatinine >1.5mg/dL (eGFR<60ml/min/1.73m^2) was used as cut off for the purpose of the study. None of the patients underwent kidney biopsy. The study records indicated that 92 of the 100 diabetic participants had one or more microvascular complications. Because individual participants could have more than one complication, the reported percentages represent overlapping categories rather than mutually exclusive groups.

The sample size was estimated using previously reported MPV values of 7.5 ± 1.1 fL in diabetic participants and 7.1 ± 1.0 fL in non-diabetic participants. With a 95% confidence level and 80% power, the calculated minimum sample size was 98 participants per group. The final enrolled sample comprised 100 participants in each group.

Statistical analysis

Continuous variables were summarised using mean and standard deviation, while categorical variables were expressed as frequencies and percentages. Between-group comparisons were performed using the independent-samples t-test or Mann–Whitney U test, as appropriate. Correlation analysis was used to evaluate the relationship between HbA1c and MPV. A two-sided p-value <0.05 was considered statistically significant.

Results

A total of 200 participants were included in the final analysis, comprising 100 patients with T2DM and 100 demographically matched non-diabetic controls. The mean age was 62.37 years in the diabetic group and 61.26 years in the control group. There was no statistically significant difference in age between the groups (p=0.408). History of smoking was present in 12 participants in diabetic group and 13 in control group (p-NS).

Mean random blood glucose was 242.03 ± 108.32 mg/dL in the diabetic group compared with 118.70 ± 27.95 mg/dL in controls (p<0.001). Mean fasting blood glucose was 182.70 ± 87.65 mg/dL and 112.00 ± 20.25 mg/dL, respectively (p<0.001). Mean postprandial blood glucose was 211.83 ± 83.05 mg/dL and 133.04 ± 20.38 mg/dL, respectively (p=0.001). Mean HbA1c was 9.069 ± 2.65% in the diabetic group and 5.723 ± 0.880% in the control group (p<0.001).

Mean MPV was 12.017 ± 1.227 fL among diabetic participants compared with 9.737 ± 1.365 fL among controls (p<0.001). In contrast, platelet counts were similar between the groups, with mean values of 2.541 ± 0.837 and 2.433 ± 0.916, respectively (p=0.201).

Serum creatinine was higher in the diabetic group, with mean values of 1.707 ± 1.827 mg/dL compared with 0.892 ± 0.272 mg/dL in controls (p=0.009). Mean systolic blood pressure was 136.70 ± 17.33 mmHg in the diabetic group and 124.25 ± 7.34 mmHg in controls (p<0.001). Mean diastolic blood pressure was 81.74 ± 11.64 mmHg and 76.65 ± 4.95 mmHg, respectively (p<0.001).

Comparison of MPV according to microvascular complications showed significantly higher MPV among diabetic participants with microvascular complications. The reported mean (median) MPV in the affected subgroup was 12.35 (12.7) fL, with p=0.0139 (Table 1).

Relationship between HbA1c and MPV

For participants with HbA1c <6.5%, the correlation coefficient was r=0.2645 with p=0.0350, representing a moderate positive correlation. For participants with HbA1c ≥6.5%, the correlation coefficient was r=-0.428 with p=0.0426, representing a moderate negative correlation (Table 2). These subgroup results should be interpreted cautiously because correlation does not establish causality and may be affected by subgroup size and clinical heterogeneity.

Microvascular complications

Ninety-two of the 100 diabetic participants were reported to have one or more microvascular complications. Diabetic neuropathy was the most frequently reported complication, followed by retinopathy and nephropathy. The original study reported neuropathy in 83%, retinopathy in 57%, and nephropathy in 28% of diabetic participants. Four male participants were reported to have all three complications. Because more than one complication could occur in the same participant, the percentages are not expected to total 100%.

Table 1: Demographic and laboratory characteristics.

Variable	*Study mean ± SD*	*Control mean ± SD*	*Test*	*p-value*	*Remark*
Age (years)	62.37 ± 10.996	61.26 ± 11.576	t=0.78	0.408	NS
RBS (mg/dL)	242.03 ± 108.32	118.70 ± 27.95	U=1169.5	<0.001	HS
FBS (mg/dL)	182.70 ± 87.65	112.00 ± 20.25	U=2486.5	<0.001	HS
PPBS (mg/dL)	211.83 ± 83.05	133.04 ± 20.38	U=1934.5	0.001	HS
HbA1c (%)	9.069 ± 2.65	5.723 ± 0.880	U=727.0	<0.001	HS
MPV (fL)	12.017 ± 1.227	9.737 ± 1.365	U=1204.5	<0.001	HS
Platelet count	2.541 ± 0.837	2.433 ± 0.916	U=4938.0	0.201	NS
Creatinine (mg/dL)	1.707 ± 1.827	0.892 ± 0.272	U=4356.5	0.009	HS
Systolic BP (mmHg)	136.70 ± 17.33	124.25 ± 7.34	U=3375.5	<0.001	HS
Diastolic BP (mmHg)	81.74 ± 11.64	76.65 ± 4.95	U=4203.0	<0.001	HS

Abbreviations: NS: not significant; HS: highly significant; RBS: random blood sugar; FBS: fasting blood sugar; PPBS: postprandial blood sugar; MPV: mean platelet volume; BP: blood pressure.

Table 2: Pearson's correlation coefficient (r) indicates the strength and direction of the correlation between variables across HbA1c categories, with p.

HbA1c category	*r*	*p-value*	*Interpretation*
HbA1c <6.5%	0.2645	0.0350	Moderate positive correlation
HbA1c ≥6.5%	-0.428	0.0426	Moderate negative correlation

The association between increased MPV and microvascular complications is clinically relevant, but the study design cannot determine whether increased MPV precedes vascular disease, results from vascular injury, or reflects a combination of metabolic and vascular factors.

Discussion

The present study demonstrated that MPV was significantly higher in patients with T2DM than in non-diabetic controls, whereas platelet counts were not significantly different. The findings support the concept that diabetes may be associated with changes in platelet size and activity that are not necessarily accompanied by changes in the absolute number of circulating platelets. Similar observations have been reported in previous studies [1,2,9,10].

The biological basis of increased MPV in diabetes is likely multifactorial. Chronic hyperglycaemia can contribute to oxidative stress, endothelial dysfunction, non-enzymatic glycation, and alterations in platelet signalling. Insulin resistance may further influence platelet activation because platelets possess insulin receptors and insulin can modulate platelet responses to agonists [5,6,11].

Larger platelets contain more dense granules and are generally considered more metabolically active. They may release greater quantities of thromboxane A2, β-thromboglobulin, serotonin, and other mediators involved in platelet aggregation and vascular responses. Increased numbers of larger platelets may therefore represent increased platelet turnover or marrow production of younger platelets in response to peripheral consumption.

Our findings are broadly consistent with Kodiatte et al., who reported increased MPV in T2DM [1], and with Zuberi et al. [2]. Jindal et al. reported relationships between platelet indices and diabetic microvascular complications [13]. A meta-analysis by Zaccardi et al. also supported the association of platelet indices with diabetes while indicating that platelet count may not differ substantially between diabetic and non-diabetic individuals [10].

The absence of a significant difference in platelet count is important. Platelet count and platelet size represent different aspects of platelet biology. A stable platelet count does not necessarily indicate normal platelet function. Changes in platelet production, survival, turnover, and peripheral consumption may maintain platelet counts within a relatively narrow range while altering platelet size.

The higher serum creatinine observed in diabetic participants is compatible with renal involvement in a proportion of patients with long-standing diabetes. Previous work has examined the relationship between

MPV and microalbuminuria [17]. However, serum creatinine alone is not a complete assessment of diabetic kidney disease, and future studies should incorporate estimated glomerular filtration rate and urinary albumin-to-creatinine ratio.

The presence of microvascular complications was associated with higher MPV. Diabetic retinopathy, nephropathy, and neuropathy share pathways involving endothelial dysfunction, inflammation, oxidative stress, and metabolic disturbance. Increased platelet activation could potentially contribute to these processes by promoting microvascular thrombosis and the release of vasoactive and growth-promoting mediators.

The relationship between MPV and diabetic retinopathy is biologically plausible because activated platelets can interact with the vascular endothelium and contribute to local thrombotic and inflammatory processes. Platelet-derived mediators, including platelet-derived growth factor and vascular endothelial growth factor, have important roles in vascular remodelling and neovascular responses. However, the present study cannot establish whether increased MPV directly contributes to retinopathy.

Previous studies have not produced completely consistent findings. Papanas et al. reported increased MPV in patients with T2DM [14], while Demirtunc et al. investigated glycaemic control and platelet activity [12]. Other studies have not demonstrated significant relationships between MPV and particular complications. Differences may arise from patient selection, disease duration, glycaemic status, medication exposure, sample handling, and methods used to diagnose complications.

The HbA1c analysis requires cautious interpretation. The study showed different correlation directions in the two HbA1c categories. Therefore, the findings should not be simplified into a statement that MPV uniformly increases with HbA1c. Larger studies using regression models are needed to determine whether glycaemic exposure independently predicts MPV after adjustment for age, sex, diabetes duration, treatment, renal function, and other factors.

The diabetic group also had higher systolic and diastolic blood pressure. Hypertension is an established contributor to diabetic microvascular disease and may itself influence platelet activity. However, this study was not designed to determine an independent association between blood pressure and MPV, and the observed differences should therefore be interpreted descriptively.

An important practical advantage of MPV is its availability as part of the routine complete blood count. If standardised laboratory procedures are followed, it may provide an inexpensive additional parameter for research stratification and risk assessment. However, MPV is not disease-specific and can be influenced by inflammatory, cardiovascular, haematological, endocrine, and medication-related factors.

Pre-analytical handling is particularly relevant. Platelets can undergo time-dependent changes after collection, and MPV may vary according to anticoagulant, storage conditions, temperature, analyser characteristics, and the interval between collection and measurement. The present study attempted to reduce this source of variation by analysing EDTA-anticoagulated samples within one hour.

Treatment is another potential consideration. Antiplatelet drugs were excluded according to the protocol because aspirin and clopidogrel can influence platelet behaviour. Nevertheless, other medications used by patients with diabetes may influence metabolic status, blood pressure, renal function, and indirectly platelet indices. Future studies should record and adjust for major medication classes.

Duration of diabetes is also relevant. All diabetic participants had diabetes for more than five years, but the study did not provide a detailed analysis according to duration beyond this minimum criterion. Longitudinal studies could determine whether MPV changes with increasing disease duration and whether changes in MPV precede or accompany microvascular complications.

The Indian setting is important because diabetes and its complications represent major public health challenges, and inexpensive laboratory parameters may be useful in settings where advanced biomarker testing is not readily available. Nevertheless, this was a single-centre study, and the findings may not be generalisable to all Indian or international populations.

The study supports further investigation of MPV as an adjunctive research parameter rather than as an established diagnostic test. A clinically useful biomarker should provide information beyond conventional predictors and should demonstrate reproducibility across laboratories and populations. Future research should therefore focus on prospective validation, standardised measurement, and clinically meaningful outcomes.

Limitations: This study was limited by its comparative observational design, single-centre setting, and modest sample size, which may restrict generalisability. Multivariable analysis was not performed to assess independent associations after adjustment for potential

confounders. Correlation of MPV with glyco HBA1C < 6.5 or > 6.5 result with respect to smoker/nonsmoker and obese /nonobese individuals was not assessed. Direct platelet function and inflammatory or oxidative stress markers were not evaluated. Potential effects of comorbidities and other factors on MPV were not comprehensively assessed. Furthermore, the cross-sectional design prevents determination of whether elevated MPV preceded or followed the development of microvascular complications.

Conclusion

In this comparative study, patients with T2DM had significantly higher MPV than non-diabetic controls, while platelet counts were comparable between the groups. Higher MPV was also observed among diabetic participants with microvascular complications, suggesting a possible association between altered platelet indices and diabetic vascular abnormalities. As MPV is routinely available through complete blood count analysis, it may serve as a useful adjunctive parameter in clinical research and risk assessment. However, the present findings do not establish a causal relationship or confirm MPV as an independent predictor of diabetic complications. Larger prospective, multicentre studies with standardised MPV assessment and appropriate adjustment for confounding factors are required to validate these findings.

Conflict of interest

The authors declare that they have no conflict of interest.

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