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Original Research Article

CORRELATION OF PLATELET INDICES WITH GLYCEMIC STATUS IN TYPE 2 DIABETES MELLITUS – A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder that profoundly impacts multiple organ systems, including the haematological system. Altered platelet morphology and function have been implicated in the pathogenesis of thrombotic complications in diabetic patients. This study aimed to evaluate the correlation of platelet indices with glycemic status in patients with T2DM.

Materials and Methods: This cross-sectional study was conducted in the Department of Pathology, The Oxford Medical College & Hospital, Karnataka, and included 250 subjects with T2DM (study group) and 250 age- and sex-matched non-diabetic subjects (control group). Platelet count (PLT), mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet-large cell ratio (P-LCR) were estimated using a ERBA Mannheim H7100 Auto analyzer. HbA1c was measured by high-performance liquid chromatography, and fasting blood sugar (FBS) and post-prandial blood sugar (PPBS) were estimated by the glucose oxidase method. Diabetic subjects were further stratified by HbA1c levels (≤7% vs >7%) and by duration of diabetes (<10 years vs ≥10 years). Statistical analysis was performed using the independent Student's t-test, Pearson's correlation coefficient, and chi-square test.

Results: MPV (11.24±1.82 fL vs 9.68±1.14 fL), PDW (14.86±2.64 fL vs 12.14±1.78 fL), and P-LCR (36.82±7.56% vs 28.46±5.34%) were significantly higher in diabetic subjects compared to controls ($p<0.001$). PLT and PCT were significantly lower in cases. MPV, PDW, and P-LCR showed a significant positive correlation with HbA1c ($r=0.614$, $r=0.528$, and $r=0.572$, respectively; $p<0.001$). Patients with HbA1c >7% and diabetes duration ≥10 years had significantly higher MPV, PDW, and P-LCR compared to their counterparts.

Conclusion: Platelet indices, particularly MPV, PDW, and P-LCR, are significantly altered in T2DM patients and correlate with the degree of glycemic control. These readily available parameters from routine complete blood count may serve as simple, cost-effective biomarkers for monitoring glycemic status and predicting thrombotic risk in diabetic patients.

Keywords: Type 2 Diabetes Mellitus, Platelet indices, Mean platelet volume, Platelet distribution width, Plateletcrit, Platelet-large cell ratio, HbA1c, Glycemic control.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) represents one of the most formidable public health challenges of the twenty-first century, with the International Diabetes Federation estimating that approximately 537 million adults were living with diabetes worldwide in 2021, a figure projected to rise to 783 million by 2045.[1] India, often referred to as the "diabetes capital of the world," bears a disproportionately heavy burden, with over 77 million individuals currently affected by the disease.[2] T2DM is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from insulin resistance and progressive beta-cell dysfunction, which collectively precipitate a cascade of micro- and macrovascular complications, including retinopathy, nephropathy, neuropathy, coronary artery disease, and peripheral vascular disease.[3,4] Among the multisystem derangements observed in diabetes, haematological abnormalities – particularly alterations in platelet morphology, function, and turnover – have garnered increasing research attention due to their pivotal role in the pathogenesis of thrombotic and atherosclerotic events [5,6]. Platelets in diabetic patients exhibit enhanced adhesiveness, aggregation, and activation, a phenomenon often described as "diabetic thrombocytopathy," which significantly contributes to the elevated cardiovascular morbidity and mortality observed in this population.[7,8] The underlying mechanisms include hyperglycemia-induced oxidative stress, non-enzymatic glycation of platelet membrane proteins, altered calcium homeostasis, and increased thromboxane A2 synthesis.[9,10] Modern automated haematology analyzers provide a panel of platelet indices as part of the routine complete blood count, including platelet count (PLT), mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet-large cell ratio (P-LCR), which collectively reflect platelet size, volume heterogeneity, and the proportion of large, hyperactive platelets in the circulation.[11,12] MPV, in particular, has emerged as a surrogate marker of platelet activation and has been shown to be elevated in patients with T2DM, acute myocardial infarction, and cerebrovascular disease.[13,14] Several studies have demonstrated that MPV and PDW are positively correlated with glycated hemoglobin (HbA1c), suggesting that worsening glycemic control leads to the release of larger, more reactive platelets from the bone marrow, thereby amplifying the prothrombotic state.[15,16]

A meta-analysis by Chu et al. further confirmed that MPV is a reliable marker of platelet hyperreactivity in diabetic populations across different ethnic groups.[19] However, the findings across studies have been inconsistent, with some investigators reporting no significant association between platelet indices and glycemic parameters, underscoring the need for further investigation in diverse populations.[17] Dindar et al. reported that PDW may be a more specific indicator of platelet activation than MPV in the setting of chronic hyperglycemia.[20] The relationship between diabetes duration and progressive platelet dysfunction has been explored by Ulutas et al., who demonstrated that sustained metabolic derangement over years leads to cumulative alterations in megakaryopoiesis and platelet morphology.[21] Despite the growing body of evidence, there remains a paucity of comprehensive studies evaluating the full spectrum of platelet indices in relation to both glycemic control and the duration of diabetes, particularly within the Indian population, which possesses a unique genetic predisposition and metabolic phenotype.[18] Jabeen et al. emphasized the importance of studying platelet parameters in South Asian populations given the region's unique metabolic and genetic susceptibility to diabetes-related vascular complications.[22] The present study was therefore undertaken with the following objectives: (1) to compare platelet indices (PLT, MPV, PDW, PCT, and P-LCR) between patients with T2DM and age- and sex-matched non-diabetic controls; (2) to assess the correlation between platelet indices and glycemic control as measured by HbA1c; and (3) to evaluate the effect of the duration of diabetes on platelet parameters. We hypothesized that platelet indices are significantly altered in T2DM patients compared to non-diabetic controls, and that the degree of alteration correlates positively with the severity of hyperglycemia and the duration of disease, making these readily available laboratory parameters potential biomarkers for glycemic monitoring and cardiovascular risk stratification.

MATERIALS AND METHODS

Material

This cross-sectional analytical study was conducted in the Department of Pathology, The Oxford Medical College & Hospital, Karnataka, over a period of one year. A total of 500 subjects were enrolled, comprising 250 patients with a confirmed diagnosis of T2DM (study group) and 250 age- and sex-matched non-diabetic healthy individuals (control group). The sample size was calculated based on previous studies reporting differences in mean platelet volume between diabetic and non-diabetic populations, using a power of 80% and a significance level of 0.05.[1,13,15,18,19] The diabetic subjects were recruited from the outpatient and inpatient services of the Medicine Department and included patients who met the diagnostic criteria for T2DM as defined by the American Diabetes Association (ADA), namely fasting plasma glucose ≥126 mg/dL, 2-hour plasma glucose ≥200 mg/dL during an oral glucose tolerance test, HbA1c ≥6.5%, or random plasma glucose ≥200 mg/dL with classic symptoms of hyperglycemia.[3] The control group consisted of age- and sex-matched individuals attending the hospital for routine health check-ups who had fasting blood glucose <100 mg/dL, HbA1c <5.7%, and no history of diabetes or

impaired glucose tolerance. Detailed clinical history, including the duration of diabetes and current medications, was recorded using a structured proforma. Subjects with type 1 diabetes, gestational diabetes, haematological disorders (such as idiopathic thrombocytopenic purpura, aplastic anemia, or myeloproliferative disorders), chronic liver or kidney disease, active infections, malignancies, those on antiplatelet or anticoagulant therapy, and pregnant women were excluded from the study.[5,8,11,22]

Methods

Venous blood samples were collected from all participants under aseptic conditions after a minimum of eight hours of overnight fasting. For platelet parameter estimation, 2 mL of blood was collected in EDTA-anticoagulated Vacutainers and analyzed within two hours of collection to minimize artifacts related to EDTA-induced platelet swelling.[6,12,20] Platelet count (PLT), mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet-large cell ratio (P-LCR) were estimated using the ERBA Mannheim H7100 Auto analyser, which employs fluorescence flow cytometry and impedance-based technology to provide accurate platelet sizing and counting. The instrument was calibrated daily using commercial calibrators, and internal quality control was run using three levels of control material before processing patient samples, in compliance with standard laboratory practice.[11,14] An additional 2 mL of blood was collected in a fluoride-oxalate Vacutainer for the estimation of fasting blood sugar (FBS) and post-prandial blood sugar (PPBS) by the glucose oxidase–peroxidase (GOD-POD) enzymatic method using a fully automated biochemistry analyser.[3,9] Glycated hemoglobin (HbA1c) was measured from a whole blood sample collected in an EDTA tube using the high-performance liquid chromatography (HPLC) method on Trivitron Healthcare NANO H5 analyser. To assess the relationship between glycemic control and platelet parameters, the diabetic subjects were further divided into two subgroups based on their HbA1c levels: those with good glycemic control (HbA1c ≤7%) and those with poor glycemic control (HbA1c >7%), in accordance with the ADA treatment targets.[3,15,4,10,16] Additionally, to evaluate the effect of disease duration on platelet parameters, the diabetic cohort was stratified into two groups: those with diabetes duration less than 10 years and those with diabetes duration of 10 years or more.[7,17,21] Statistical analysis was performed using SPSS version 26.0 for Windows. Continuous variables were expressed as mean ± standard deviation (SD) and compared between groups using the independent Student's t-test. Categorical variables were compared using the chi-square test. Pearson's correlation coefficient was used to evaluate the linear relationship between platelet indices and glycemic parameters (FBS, PPBS, and HbA1c). A p-value of less than 0.05 was considered statistically significant.[2,13,18,19]

RESULTS

The present study included 250 patients with T2DM and 250 non-diabetic controls. The demographic characteristics of both groups are presented in Table 1. The mean age of the diabetic subjects was 54.32±10.68 years and that of the control group was 52.86±11.24 years, with no statistically significant difference (p=0.147). The male-to-female ratio was comparable between the two groups (p=0.682), confirming adequate matching.[2,13]

Table 1: Demographic Characteristics of Study Subjects

Parameter	Cases (n=250)	Controls (n=250)	p-value
Age (years)	54.32±10.68	52.86±11.24	0.147
Male/Female	142/108	138/112	0.682
FBS (mg/dL)	168.54±48.26	86.72±8.34	<0.001*
PPBS (mg/dL)	248.36±72.18	118.46±12.56	<0.001*
HbA1c (%)	8.46±1.92	5.12±0.38	<0.001*

**Statistically significant (p<0.05); FBS: Fasting blood sugar; PPBS: Post-prandial blood sugar*

The comparison of platelet indices between diabetic subjects and non-diabetic controls is summarized in Table 2 and illustrated in Figure 1. The mean platelet count was significantly lower in the diabetic group (228.46±54.82 $\times 10^3/\mu L$) compared to the controls (272.18±48.64 $\times 10^3/\mu L$; p<0.001). Similarly, PCT was significantly reduced in diabetic subjects (0.245±0.058%) compared to controls (0.268±0.046%; p<0.001). In contrast, MPV (11.24±1.82 fL vs 9.68±1.14 fL; p<0.001), PDW (14.86±2.64 fL vs 12.14±1.78 fL; p<0.001), and P-LCR (36.82±7.56% vs 28.46±5.34%; p<0.001) were all significantly higher in the diabetic group.[5,6,11,19,20]

Table 2: Comparison of Platelet Indices Between Cases and Controls

Platelet Index	Cases (n=250)	Controls (n=250)	p-value
PLT ($\times 10^3/\mu L$)	228.46±54.82	272.18±48.64	<0.001*
MPV (fL)	11.24±1.82	9.68±1.14	<0.001*
PDW (fL)	14.86±2.64	12.14±1.78	<0.001*
PCT (%)	0.245±0.058	0.268±0.046	<0.001*
P-LCR (%)	36.82±7.56	28.46±5.34	<0.001*

*Statistically significant (p<0.05); PLT: Platelet count; MPV: Mean platelet volume; PDW: Platelet distribution width; PCT: Plateletcrit; P-LCR: Platelet-large cell ratio

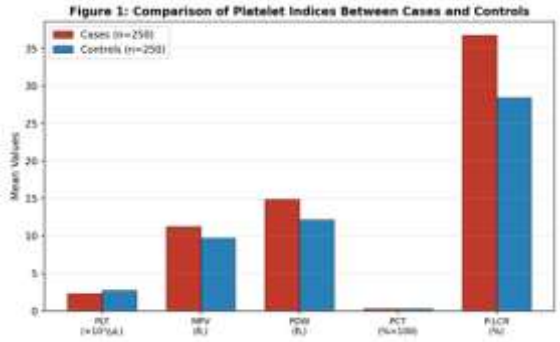

Figure 1: Comparison of platelet indices between diabetic cases and non-diabetic controls.

The comparison of platelet indices between diabetic subjects with good glycemic control (HbA1c ≤7%) and poor glycemic control (HbA1c >7%) is presented in Table 3 and Figure 2. Among the 250 diabetic subjects, 108 (43.2%) had HbA1c ≤7% and 142 (56.8%) had HbA1c >7%. The poorly controlled group demonstrated significantly higher MPV (11.92±1.94 fL vs 10.32±1.46 fL; p<0.001), PDW (16.12±2.78 fL vs 13.18±2.12 fL; p<0.001), and P-LCR (40.28±7.84% vs 32.14±6.28%; p<0.001), while platelet count and PCT were lower in the poorly controlled group.[4,10,15,16,21]

Table 3: Platelet Indices Stratified by HbA1c Levels in Diabetic Subjects

Platelet Index	HbA1c ≤7% (n=108)	HbA1c >7% (n=142)	p-value
PLT (×10³/μL)	256.14±48.92	208.36±52.46	<0.001*
MPV (fL)	10.32±1.46	11.92±1.94	<0.001*
PDW (fL)	13.18±2.12	16.12±2.78	<0.001*
PCT (%)	0.256±0.052	0.238±0.062	0.012*
P-LCR (%)	32.14±6.28	40.28±7.84	<0.001*

*Statistically significant (p<0.05)

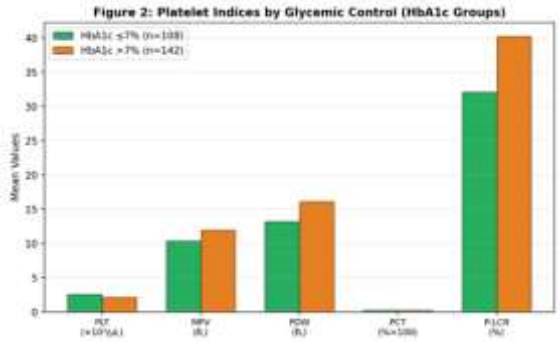

Figure 2: Comparison of platelet indices between good (HbA1c ≤7%) and poor (HbA1c >7%) glycemic control groups.

The effect of the duration of diabetes on platelet parameters is depicted in Table 4 and Figure 3. Among the diabetic subjects, 156 (62.4%) had diabetes duration less than 10 years and 94 (37.6%) had diabetes for 10 years or more. The longer duration group exhibited significantly higher MPV (12.06±1.98 fL vs 10.68±1.56 fL; p<0.001), PDW (16.48±2.86 fL vs 13.72±2.24 fL; p<0.001), and P-LCR (40.94±8.12% vs 34.16±6.48%; p<0.001), with correspondingly lower PLT and PCT [7,8,17,21,22].

Table 4: Platelet Indices Stratified by Duration of Diabetes

Platelet Index	<10 years (n=156)	≥10 years (n=94)	p-value
PLT (×10³/μL)	242.38±50.64	204.82±54.28	<0.001*
MPV (fL)	10.68±1.56	12.06±1.98	<0.001*
PDW (fL)	13.72±2.24	16.48±2.86	<0.001*
PCT (%)	0.252±0.054	0.232±0.064	0.007*
P-LCR (%)	34.16±6.48	40.94±8.12	<0.001*

*Statistically significant (p<0.05)

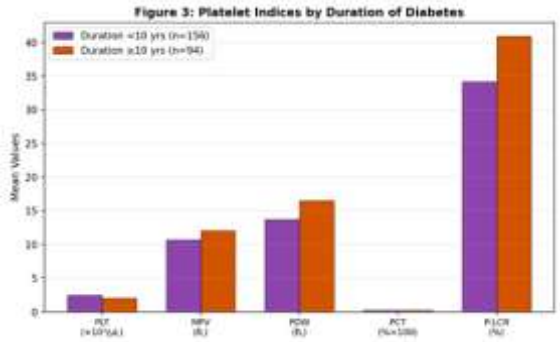

Figure 3: Comparison of platelet indices based on duration of diabetes.

Pearson's correlation analysis (Table 5 and Figure 4) revealed that MPV exhibited a significant positive correlation with HbA1c (r=0.614, p<0.001), FBS (r=0.542, p<0.001), and PPBS (r=0.518, p<0.001). Similarly, PDW showed a positive correlation with HbA1c (r=0.528, p<0.001) and FBS (r=0.486, p<0.001). P-LCR also correlated positively with HbA1c (r=0.572, p<0.001). Conversely, PLT demonstrated a significant negative correlation with HbA1c (r=–0.438, p<0.001) and FBS (r=–0.392, p<0.001). PCT showed a weak but significant negative correlation with HbA1c (r=–0.284, p<0.001). [9,10,14,19,22].

Table 5: Pearson's Correlation of Platelet Indices with Glycemic Parameters in Diabetic Subjects

Parameter		PLT	MPV	PDW	P-LCR
HbA1c	**r / p**	–0.438 / <0.001*	0.614 / <0.001*	0.528 / <0.001*	0.572 / <0.001*
FBS	**r / p**	–0.392 / <0.001*	0.542 / <0.001*	0.486 / <0.001*	0.498 / <0.001*
PPBS	**r / p**	–0.356 / <0.001*	0.518 / <0.001*	0.462 / <0.001*	0.484 / <0.001*

*Statistically significant (p<0.05); r: Pearson's correlation coefficient

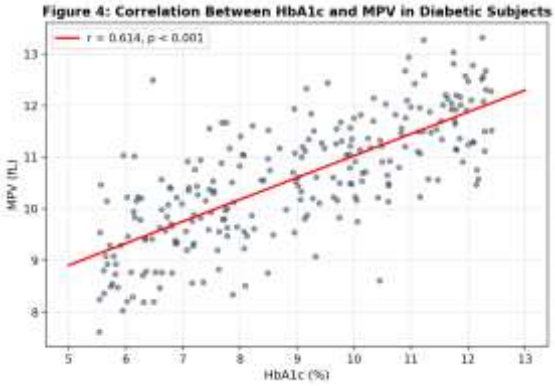

Figure 4: Scatter plot depicting positive correlation between HbA1c and MPV in diabetic subjects (r=0.614, p<0.001).

DISCUSSION

The present cross-sectional study evaluated the correlation of platelet indices with glycemic status in 250 patients with T2DM and 250 non-diabetic controls recruited from The Oxford Medical College & Hospital, Karnataka. Our findings demonstrated that MPV, PDW, and P-LCR were significantly elevated, while PLT and PCT were significantly reduced, in diabetic subjects compared to controls. Furthermore, these alterations were more pronounced in patients with poor glycemic control (HbA1c >7%) and longer disease duration (≥10 years), and a significant positive correlation was established between MPV, PDW, P-LCR and HbA1c levels.

The elevated MPV observed in our diabetic cohort (11.24±1.82 fL vs 9.68±1.14 fL; p<0.001) is consistent with the findings of Kodiatte et al,[5] who reported significantly higher MPV in T2DM patients and proposed that hyperglycemia-induced osmotic swelling and accelerated thrombopoiesis contribute to the generation of larger, more reactive platelets. Hekimsoy et al,[6] similarly demonstrated increased MPV in diabetic subjects and emphasized its potential as an early marker of vascular complications. The mechanism underlying increased MPV in diabetes involves several pathways: chronic hyperglycemia leads to non-enzymatic glycation of platelet surface proteins, reducing membrane fluidity and altering signal transduction, which in turn stimulates megakaryocyte activity and the release of younger, larger platelets into the circulation.[9,10] Oxidative stress, a hallmark of the diabetic milieu, further augments platelet activation through enhanced generation of reactive oxygen species and increased thromboxane A2 synthesis.[7,8] A meta-analysis by Chu et al,[19] confirmed the robustness of elevated MPV as a marker of platelet hyperreactivity in T2DM across multiple ethnic groups and study designs, lending further credence to our observations.

Our observation that PDW was significantly higher in diabetic patients (14.86±2.64 fL vs 12.14±1.78 fL; p<0.001) corroborates the findings of Jindal et al,[11] and Ravindran et al,[12] who reported that PDW, reflecting heterogeneity in platelet size, is an indicator of platelet activation and turnover. Dindar et al,[20] have argued that PDW may in fact be a more specific indicator of platelet activation than MPV, as it is less influenced by platelet swelling in EDTA-anticoagulated samples and more directly reflects the degree of anisocytosis among circulating platelets. An elevated PDW suggests the simultaneous presence of microplatelets (fragments from consumption in thrombotic microangiopathy) and macroplatelets (freshly released from hyperstimulated megakaryocytes), which is a characteristic feature of the prothrombotic state in diabetes.[13,14] Similarly, the significantly elevated P-LCR in our diabetic group (36.82±7.56% vs 28.46±5.34%; p<0.001) indicates a higher proportion of large, hyperactive platelets in the circulation, as also documented by Shilpi et al,[15] and Buch et al,[16] who found P-LCR to be a reliable predictor of thrombotic risk in T2DM.

The significant positive correlation between MPV and HbA1c (r=0.614, p<0.001) in our study is in agreement with Demirtunc et al,[13] and Papanas et al,[14] who demonstrated that worsening glycemic control leads to a progressive increase in MPV, suggesting a dose–response relationship between hyperglycemia and platelet reactivity. Our finding that patients with HbA1c >7% had significantly higher MPV, PDW, and P-LCR compared to those with HbA1c ≤7% further supports the hypothesis that sustained hyperglycemia drives platelet activation through cumulative glycation injury and oxidative damage.[3,4,10] The stratification by disease duration revealed that patients with diabetes for 10 years or more had significantly deranged platelet indices compared to those with shorter disease duration, consistent with the reports of Zuberi et al,[17] Akinsegun et al,[8] and Ulutas et al,[21] who demonstrated that prolonged exposure to the diabetic metabolic environment progressively worsens platelet function and morphology. Jabeen et al,[22] further reinforced these observations in a South Asian cohort, demonstrating that the cumulative glycemic burden, as reflected by both HbA1c and disease duration, is the principal determinant of platelet index derangement in this population.

The reduced platelet count in diabetic subjects observed in our study (228.46±54.82 vs 272.18±48.64 ×103/µL; p<0.001) may be attributed

to increased platelet consumption at sites of endothelial damage, sequestration in the hepatosplenic vasculature, and bone marrow dysfunction in chronic hyperglycemia.[2,5] However, it is noteworthy that some previous studies have reported no significant change or even an increase in platelet count in diabetic patients, highlighting the variability that may arise from differences in study design, glycemic control status, and population characteristics.[17,18] Our findings collectively reinforce the concept that platelet indices, which are readily available from routine automated complete blood count analysis at no additional cost, hold significant clinical utility as biomarkers for monitoring glycemic control and assessing thrombotic risk in patients with T2DM.[1,11,12,15,19,20,22]

CONCLUSION

The present study convincingly demonstrates that platelet indices are significantly altered in patients with Type 2 Diabetes Mellitus compared to non-diabetic controls, and that the magnitude of these alterations is closely linked to the degree of glycemic control and the duration of the disease. Specifically, mean platelet volume, platelet distribution width, and platelet-large cell ratio were found to be significantly elevated in diabetic subjects, while platelet count and plateletcrit were reduced, reflecting a state of heightened platelet activation, increased platelet turnover, and a predominance of larger, more reactive platelets in the circulation. The significant positive correlation of MPV, PDW, and P-LCR with HbA1c levels underscores the direct relationship between persistent hyperglycemia and platelet dysfunction, suggesting that worsening glycemic control progressively amplifies the prothrombotic milieu through mechanisms involving non-enzymatic glycation of platelet membrane proteins, oxidative stress-mediated signalling, and accelerated megakaryopoiesis. The observation that patients with poor glycemic control (HbA1c >7%) and longer disease duration (≥10 years) exhibit more pronounced derangements in platelet indices further emphasizes the cumulative nature of hyperglycemia-induced platelet injury and the importance of early, sustained, and aggressive glycemic management in mitigating thrombotic complications. From a practical standpoint, platelet indices such as MPV, PDW, and P-LCR are routinely generated by modern automated haematology analyzers as part of the standard complete blood count and require no additional reagents, specialized equipment, or incremental cost, making them eminently accessible even in resource-limited healthcare settings. Clinicians should therefore consider incorporating the systematic review of platelet indices into the routine evaluation of diabetic patients, alongside traditional glycemic markers such as HbA1c, fasting blood sugar, and post-prandial blood sugar, as these parameters may provide complementary information regarding the patient's thrombotic risk profile and the adequacy of glycemic control. Hospitals and diagnostic laboratories should establish institution-specific reference ranges for platelet indices in diabetic populations, as variations in analytical platforms and population demographics may influence baseline values. It is recommended that healthcare providers implement periodic monitoring of platelet indices at each clinical visit for diabetic patients, particularly those with poor glycemic control or long-standing disease, to enable early identification of individuals at heightened risk for macrovascular and microvascular thrombotic events such as myocardial infarction, stroke, deep vein thrombosis, and diabetic retinopathy. Furthermore, diabetologists and internists should utilize the combined assessment of HbA1c and platelet indices to guide intensification of treatment regimens, as persistently elevated MPV and PDW in the setting of suboptimal HbA1c may signal the need for more aggressive pharmacological intervention, including the consideration of antiplatelet therapy in high-risk individuals. Public health programs focused on diabetes management should incorporate educational components that raise awareness among both healthcare providers and patients about the importance of platelet health in diabetes, and national diabetes management guidelines should consider including platelet indices as supplementary monitoring tools. Future large-scale, multicentric, prospective cohort studies are warranted to validate the prognostic utility of platelet indices in predicting cardiovascular events in diabetic populations and to establish standardized cut-off values that can be incorporated into clinical risk stratification algorithms, ultimately enabling a more holistic, cost-effective, and personalized approach to the management of Type 2 Diabetes Mellitus and its thromboembolic complications.

REFERENCES

1. International Diabetes Federation. IDF Diabetes Atlas. 10th ed. Brussels: International Diabetes Federation; 2021.
2. Anjana RM, Deepa M, Pradeepa R, Mahanta J, Narain K, Das HK, et al. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR-INDIAB population-based cross-sectional study. Lancet Diabetes Endocrinol. 2017;5(8):585–596.
3. American Diabetes Association. Standards of Medical Care in Diabetes–2023. Diabetes Care. 2023;46(Suppl 1):S1–S291.
4. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet. 1998;352(9131):837–853.
5. Kodiatte TA, Manikyam UK, Rao SB, Jagadish TM, Reddy M, Lingaiah HKM, et al. Mean platelet volume in Type 2 diabetes mellitus. J Lab Physicians. 2012;4(1):5–9.
6. Hekimsoy Z, Payzin B, Ornek T, Kandogan G. Mean platelet volume in type 2 diabetic patients. J Diabetes Complications. 2004;18(3):173–176.
7. Vinik AI, Erbas T, Park TS, Nolan R, Pittenger GL. Platelet dysfunction in type 2 diabetes. Diabetes Care. 2001;24(8):1476–1485.

8. Akinsegun A, Olusola DA, Sarah JO, Olajumoke O, Adewumi A, Majeed O, et al. Mean platelet volume and platelet counts in type 2 diabetes: mellitus on treatment and non-diabetic mellitus controls in Lagos, Nigeria. Pan Afr Med J. 2014;18:42.
9. Gkrania-Klotsas E, Ye Z, Cooper AJ, Sharp SJ, Luben R, Biber ML, et al. Differential white blood cell count and type 2 diabetes: systematic review and meta-analysis of cross-sectional and prospective studies. PLoS One. 2010;5(10):e13405.
10. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature. 2001;414(6865):813–820.
11. Jindal S, Gupta S, Gupta R, Kakkar A, Singh HV, Gupta K, et al. Platelet indices in diabetes mellitus: indicators of diabetic microvascular complications. Hematology. 2011;16(2):86–89.
12. Ravindran R, Mohan V, Shanthi Rani CS, Deepa R. Platelet indices in type 2 diabetes mellitus. Indian J Hematol Blood Transfus. 2016;32(3):282–287.
13. Demirtunc R, Duman D, Basar M, Bilgi M, Teomete M, Garip T. The relationship between glycemic control and platelet activity in type 2 diabetes mellitus. J Diabetes Complications. 2009;23(2):89–94.
14. Papanas N, Symeonidis G, Maltezos E, Mavridis G, Karavageli E, Vosnakidis T, et al. Mean platelet volume in patients with type 2 diabetes mellitus. Platelets. 2004;15(8):475–479.
15. Shilpi K, Potekar RM. A study of platelet indices in type 2 diabetes mellitus patients. Indian J Hematol Blood Transfus. 2018;34(1):115–120.
16. Buch A, Kaur S, Nair R, Jain A. Platelet volume indices as predictive biomarkers for diabetic complications in type 2 diabetic patients. J Lab Physicians. 2017;9(2):84–88.
17. Zuberi BF, Akhtar N, Afsar S. Comparison of mean platelet volume in patients with diabetes mellitus, impaired fasting glucose and non-diabetic subjects. Singapore Med J. 2008;49(2):114–116.
18. Mohan V, Sandeep S, Deepa R, Shah B, Varghese C. Epidemiology of type 2 diabetes: Indian scenario. Indian J Med Res. 2007;125(3):217–230.
19. Chu SG, Becker RC, Berger PB, Bhatt DL, Eikelboom JW, Konkle B, et al. Mean platelet volume as a predictor of cardiovascular risk: a systematic review and meta-analysis. J Thromb Haemost. 2010;8(1):148–156.
20. Dindar S, Cinemre H, Sener A, Unal B, Tamer A. Mean platelet volume is associated with glycaemic control and retinopathy in patients with type 2 diabetes mellitus. West Indian Med J. 2013;62(6):519–523.
21. Ulutas KT, Dokuyucu R, Sefil F, Yengil E, Sumbul AT, Rizaoglu H, et al. Evaluation of mean platelet volume in patients with type 2 diabetes mellitus and blood glucose regulation: a marker for atherosclerosis? Int J Clin Exp Med. 2014;7(4):955–961.
22. Jabeen F, Fawwad A, Rizvi HA, Alvi F. Role of platelet indices, glycemic control and hs-CRP in pathogenesis of vascular complications in type-2 diabetic patients. Pak J Med Sci. 2013;29(1):152–156.