

ORIGINAL ARTICLE

Correlation of platelet indices with microvascular complications in patients with type 2 diabetes mellitus: an analytical cross-sectional study.

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ABSTRACT... Objective: To compare platelet indices between T2DM patients with and without microvascular complications and to evaluate their correlation with diabetic retinopathy, nephropathy, and neuropathy. **Study Design:** Cross-sectional Observational study. **Setting:** Department of General Medicine, Federal Government Polyclinic Hospital, Islamabad. **Period:** April to December 2025. **Methods:** An analytical cross-sectional study of 220 T2DM patients (110 with microvascular complications, 110 without) was conducted at a tertiary care hospital in Islamabad, Pakistan. Platelet indices were measured on an automated hematological analyzer. Independent samples t-test and Spearman's rank correlation were used for analysis. **Results:** MPV (13.41 ± 2.10 vs. 12.00 ± 1.89 fL), PDW (16.81 ± 2.73 vs. $15.24 \pm 2.67\%$), and P-LCR (28.66 ± 7.00 vs. $24.00 \pm 6.71\%$) were significantly higher in the complication group (all $p < 0.001$). Spearman's correlation confirmed significant positive associations for MPV ($\rho = +0.343$), PDW ($\rho = +0.283$), and P-LCR ($\rho = +0.309$; all $p < 0.001$). Platelet count showed no significant differences or correlations. **Conclusion:** MPV, PDW, and P-LCR are significantly elevated and positively correlated with microvascular complications in T2DM. These routinely available, low-cost indices may serve as practical adjuncts for microvascular risk stratification, particularly in resource-constrained settings.

Key words: Blood Platelets, Diabetic Angiopathies, Diabetic Nephropathies, Diabetes Mellitus, Diabetic Retinopathy, Type 2, Mean Platelet Volume.

Article Citation: Hameed S, Dar SI, Nasir M, Awan M, Javed I, Zaheer K. Correlation of platelet indices with microvascular complications in patients with type 2 diabetes mellitus: an analytical cross-sectional study. Professional Med J 2026; 33(10):1938-1944.
<https://doi.org/10.29309/TPMJ/2026.33.10.10496>

INTRODUCTION

Diabetes mellitus (DM) has evolved into one of the defining health crises of the twenty-first century. According to the 11th IDF Diabetes Atlas, an estimated 589 million adults were living with diabetes in 2024 (approximately one in nine worldwide) projected to reach 853 million by 2050.¹ Type 2 diabetes mellitus (T2DM) accounts for over 90% of all cases and is driven by a complex interaction of insulin resistance, progressive beta-cell failure, chronic inflammation, and adverse lifestyle factors.²

Pakistan bears a disproportionate share of this burden; the IDF Atlas identifies it as having the highest age-adjusted prevalence globally, with approximately 33 million affected, the third largest diabetes population worldwide.^{3,4} A 2024 systematic review and meta-analysis estimated the pooled prevalence of T2DM in Pakistan at 14.60%.⁵ Suboptimal glycemic control, limited

healthcare access, poor treatment adherence, and low health literacy compound the public health challenge and make Pakistan particularly vulnerable to the downstream consequences of chronic hyperglycemia.⁶

Among the serious sequelae of long-standing T2DM, microvascular complications, diabetic retinopathy, nephropathy, and peripheral neuropathy, constitute the triad most directly attributable to sustained hyperglycemia and represent the principal drivers of diabetes-related morbidity. Retinopathy remains a leading cause of blindness in working-age adults; nephropathy is the single most common cause of end-stage renal disease; and neuropathy affects up to 50% of patients with long-standing disease, predisposing to foot ulceration, amputation, and cardiovascular autonomic dysfunction.^{7,8}

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Article received on:

31/03/2026

Accepted for publication:

03/06/2026



These complications share a common pathophysiological substrate: chronic hyperglycemia promotes the accumulation of advanced glycation end-products (AGEs), activates protein kinase C, and drives flux through the polyol pathway, collectively inducing endothelial dysfunction, basement membrane thickening, pericyte loss, and capillary occlusion; superimposed oxidative stress and a proinflammatory milieu amplify this cascade, culminating in progressive ischemia across retinal, glomerular, and neural vascular beds.^{8,9}

A dimension of this pathophysiology that has attracted increasing research attention is the role of platelet hyperactivity. Platelets in patients with T2DM demonstrate a distinctly prothrombotic phenotype: glycation of platelet membrane proteins, impaired insulin-mediated inhibition of platelet activation, and the hyperglycemia-associated increase in thromboxane A₂ and serotonin release collectively enhance platelet reactivity, aggregation, and adhesion.^{10,11} Larger, metabolically more active platelets, reflected in elevated Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW), are recruited from bone marrow stores under conditions of chronic vascular stress, and these morphologically altered platelets carry a greater thrombogenic burden than their smaller counterparts.^{11,12}

Platelet indices, MPV, PDW, and Platelet Large Cell Ratio (P-LCR), are automatically generated as part of a complete blood count on modern hematological analyzers and are therefore widely and inexpensively available, even in resource-limited settings. Several studies conducted across diverse populations have reported significant elevations in MPV and PDW among diabetic patients with microvascular complications compared to those without.¹³⁻¹⁵ Taderegew et al., in a cross-sectional study of 352 T2DM patients in Northeast Ethiopia, demonstrated that MPV, PDW, and P-LCR were significantly higher in patients with microvascular complications and proposed these indices as cost-effective prognostic markers.¹³ Buch et al. found MPV and PDW to be superior predictors of microvascular, as opposed to macrovascular, complications in T2DM.¹¹ Abd El-Ghany et al. reported that MPV and PDW correlated with the presence of retinopathy, nephropathy,

and neuropathy in Egyptian patients with T2DM, concluding that these indices merit consideration as early biomarkers.¹⁵

Despite this growing body of international evidence, data from Pakistan, a country carrying one of the world's highest burdens of T2DM and its complications, remain conspicuously limited. Studies from the region are predominantly restricted to small, single-complication cohorts. This gap is particularly relevant given the epidemiological, genetic, and healthcare delivery context that distinguishes South Asian T2DM patients from those in whom most existing evidence has been generated.^{5,6}

The present study therefore compares the mean platelet indices between T2DM patients with and without microvascular complications presenting to a tertiary care hospital in Islamabad, Pakistan, and to determine their correlation.

METHODS

This was a cross-sectional study conducted in the Department of General Medicine at a tertiary care hospital in Islamabad, Pakistan. The study was conducted from April to December 2025 after ethical approval was obtained from the Ethical Committee of the institution (ref no. 1/12/2024). The study was carried out in accordance with the Declaration of Helsinki, and written informed consent was obtained from all participants prior to enrolment.

Sample size was calculated using the OpenEPI online calculator. Using a published prevalence from Ahluwalia et al. at 80% power and $\alpha = 0.05$, the minimum required sample was 220 participants (110 per group).¹⁶

Non-probability consecutive sampling was employed. The study population was stratified into two groups based on the presence or absence of microvascular complications. Group 1 comprised patients with one or more confirmed microvascular complications (diabetic retinopathy, nephropathy, or neuropathy). Group 2 comprised patients with diagnosed T2DM but without any microvascular complication.

Inclusion criteria contained: Patients aged 30–70

years, with a confirmed diagnosis of T2DM for at least five years based on ADA criteria (HbA1c $\geq$ 6.5%, fasting plasma glucose $\geq$ 126 mg/dL, or two-hour plasma glucose $\geq$ 200 mg/dL on two separate occasions), and willing to provide informed consent. On the other hand, the exclusion criteria consisted of: patients with hematological malignancies, chronic liver disease, or other significant comorbidities that could confound platelet indices; patients receiving corticosteroids, antiplatelet therapy, or bone marrow suppressants; pregnant females; and patients with a history of blood transfusion within the preceding three weeks.

A structured, proforma was used to collect patient data via direct patient interview and review of medical records. Variables recorded included age, sex, BMI, place of residence, duration of diabetes, treatment modality, compliance, and comorbidities. HbA1c was measured at enrolment.

Microvascular complications were defined as: Diabetic retinopathy diagnosed on fundoscopy; diabetic nephropathy defined as albuminuria $>$ 30 mg/g or estimated glomerular filtration rate (eGFR) $<$ 60 mL/min/1.73 m²; and diabetic neuropathy based on clinical symptoms corroborated by nerve conduction studies. Venous blood samples were collected for complete blood count analysis, performed on a Sysmex 3-part and 7-part automated hematological analyzer. Platelet indices recorded were: Mean Platelet Volume (MPV, fL), Platelet Distribution Width (PDW, %), and Platelet Large Cell Ratio (P-LCR, %).

Data were analyzed using IBM SPSS version 26 (IBM Corp., Armonk, NY). Continuous variables (age, BMI, duration of diabetes, HbA1c, and platelet indices) were summarized as mean $\pm$ standard deviation (SD). Categorical variables (gender, residence, education, treatment modality, compliance, and comorbidities) were expressed as frequencies and percentages. Normality of continuous variables was assessed using the Shapiro–Wilk test. Normally distributed variables were compared between groups using the independent samples t-test; non-normally distributed variables were compared with the Mann–Whitney U test. Categorical variables were compared using the chi-squared (χ^2) test or Fisher's exact test as appropriate. Spearman's rank

correlation coefficient (ρ) was used to evaluate the relationship between platelet indices and the presence of microvascular complications. A p-value $<$ 0.05 was considered statistically significant.

RESULTS

A total of 220 T2DM patients were enrolled, 110 per group. The baseline characteristics of both groups are summarized in Table-I. Patients in Group 1 were significantly older and had a significantly longer duration of diabetes. Mean BMI was significantly higher in Group 1 as well. Furthermore, glycemic control was significantly worse in Group 1, as evidenced by a higher mean HbA1c, as was the prevalence of hypertension. Poor treatment compliance was more frequently observed in Group 1 (35.5% vs. 14.5%; $p <$ 0.001). However, gender distribution and place of residence did not differ significantly between groups thereby eliminating them as confounding factor in this study.

TABLE-I

Baseline demographic and clinical characteristics of study participants

Variable	Group 1 – With Comp.	Group 2 – No Comp.	P-Value
Demographic Characteristics			
Age, years (Mean $\pm$ SD)	58.3 $\pm$ 7.5	47.1 $\pm$ 11.3	$<$ 0.001
Gender: Male, n (%)	60 (54.5%)	58 (52.7%)	0.892
BMI, kg/m ² (Mean $\pm$ SD)	28.9 $\pm$ 3.9	27.6 $\pm$ 3.3	0.010
Residence: Urban, n (%)	67 (60.9%)	71 (64.5%)	0.676
Clinical Characteristics			
Duration of DM, years (Mean $\pm$ SD)	16.0 $\pm$ 5.1	10.2 $\pm$ 3.6	$<$ 0.001
HbA1c, % (Mean $\pm$ SD)	9.7 $\pm$ 1.3	8.0 $\pm$ 1.0	$<$ 0.001
Hypertension: Yes, n (%)	73 (66.4%)	43 (39.1%)	$<$ 0.001
Treatment: OHA only, n (%)	39 (35.5%)	61 (55.5%)	0.012
Treatment: Insulin only, n (%)	26 (23.6%)	17 (15.5%)	0.012
Treatment: Both, n (%)	45 (40.9%)	32 (29.1%)	0.012
Good compliance, n (%)	33 (30.0%)	54 (49.1%)	$<$ 0.001
Fair compliance, n (%)	38 (34.5%)	40 (36.4%)	$<$ 0.001
Poor compliance, n (%)	39 (35.5%)	16 (14.5%)	$<$ 0.001

Data expressed as mean $\pm$ SD for continuous variables and n (%) for categorical variables. Continuous variables compared by independent samples t-test; categorical variables by χ^2 test. $p < 0.05$ considered statistically significant.

Among the 110 patients in Group 1, neuropathy was the most prevalent complication (62.7%), followed by retinopathy (59.1%) and nephropathy (50.0%). The majority of Group 1 patients had two concurrent complications (46.4%), while 40.9% had a single complication and 12.7% had all three. The breakdown is presented in Table-II.

TABLE-II

Distribution of microvascular complications in group 1 (n = 110)

Microvascular Complication	n	%
Retinopathy only	65	59.1%
Nephropathy only	55	50.0%
Neuropathy only	69	62.7%
Single complication	45	40.9%
Two complications	51	46.4%
All three complications	14	12.7%

Percentages reflect proportion of Group 1 patients with the respective complication; categories are not mutually exclusive.

The primary outcomes comparing platelet indices between the two groups are presented in Table-III. MPV was significantly elevated in Group 1 compared to Group 2 (13.41 ± 2.10 fL vs. 12.00 ± 1.89 fL; $p < 0.001$). Similarly, PDW was significantly higher in Group 1 ($16.81 \pm 2.73\%$ vs. $15.24 \pm 2.67\%$; $p < 0.001$), as was P-LCR ($28.66 \pm 7.00\%$ vs. $24.00 \pm 6.71\%$; $p < 0.001$). In contrast, PCT did not differ significantly between the two groups ($0.227 \pm 0.053\%$ vs. $0.219 \pm 0.058\%$; $p = 0.266$). Platelet count was likewise comparable across groups ($252.9 \pm 48.6 \times 10^9/L$ vs. $261.6 \pm 52.9 \times 10^9/L$; $p = 0.418$).

Spearman's rank correlation analysis revealed statistically significant positive correlations between microvascular complications and MPV ($\rho = +0.343$; $p < 0.001$), PDW ($\rho = +0.283$; $p < 0.001$), and P-LCR ($\rho = +0.309$; $p < 0.001$). PCT ($\rho = +0.093$; $p = 0.171$) and platelet count ($\rho = -0.055$; $p = 0.419$) did not demonstrate significant correlations. These findings are detailed in Table-IV.

TABLE-III

Comparison of platelet indices between Group 1 (With Complications) and Group 2 (Without Complications)

Platelet Index	Group 1 Mean $\pm$ SD	Group 2 Mean $\pm$ SD	P-Value	Test
MPV (fL)	13.41 ± 2.10	12.00 ± 1.89	<0.001	Independent t-test
PDW (%)	16.81 ± 2.73	15.24 ± 2.67	<0.001	Independent t-test
P-LCR (%)	28.66 ± 7.00	24.00 ± 6.71	<0.001	Independent t-test
Platelet Count ($\times 10^9/L$)	252.9 ± 48.6	261.6 ± 52.9	0.418	Mann-Whitney U

Values expressed as mean $\pm$ SD. Normality assessed by Shapiro-Wilk test. Independent samples t-test used for normally distributed data; Mann-Whitney U test used for non-normal distribution (platelet count). Bold p-values indicate statistical significance ($p < 0.05$). MPV = Mean Platelet Volume; PDW = Platelet Distribution Width; P-LCR = Platelet Large Cell Ratio.

TABLE-IV

Spearman's Rank Correlation of Platelet Indices with Presence of Microvascular Complications (n = 220)

Platelet Index	Spearman's ρ	P-Value	Interpretation
MPV (fL)	+0.343	< 0.001	***
PDW (%)	+0.283	< 0.001	***
PCT (%)	+0.093	0.171	NS
P-LCR (%)	+0.309	< 0.001	***
Platelet Count ($\times 10^9/L$)	-0.055	0.419	NS

ρ = Spearman's rank correlation coefficient. *** $p < 0.001$; NS = Not Significant. Positive ρ indicates higher index values associated with presence of complications.

DISCUSSION

This cross-sectional study evaluated the relationship between platelet indices and microvascular complications in 220 patients with T2DM attending a tertiary care center in Islamabad, Pakistan. The principal findings are threefold. First, MPV, PDW, and P-LCR were each significantly higher in patients with one or more microvascular complications compared to those without ($p < 0.001$ for all three). Second, Spearman's correlation confirmed significant positive associations for these three indices (all $p < 0.001$), while PCT and platelet count showed no significant differences or correlations, implicating platelet morphology and heterogeneity, rather than platelet mass, as the key drivers of the microvascular phenotype.

The observed elevations in MPV in the complication group are consistent with a large and growing body of international evidence. Khanna et al., in a hospital-based prospective case-control study of 200 subjects, reported significantly higher MPV values in diabetics with microvascular complications versus in those without.¹⁷ Taderegew et al. similarly demonstrated significantly elevated MPV, PDW, and P-LCR in T2DM patients with microvascular complications in Northeast Ethiopia, with the magnitude of elevation increasing with advancing age and longer disease duration.¹³ Abd El-Ghany et al. specifically reported receiver-operating characteristic (ROC) thresholds for MPV (> 11.9 fL: sensitivity 80%, specificity 97.8%) and PDW (> 16.9%: sensitivity 74.5%, specificity 100%) as discriminating values for the presence of microvascular complications among Egyptian T2DM patients.¹⁵ Buch et al., whose study explicitly compared microvascular with macrovascular complications, concluded that MPV and PDW are more strongly predictive of microvascular than macrovascular disease; an observation that underscores the particular relevance of these indices in the context of diabetic retinopathy, nephropathy, and neuropathy.¹¹

The biochemical basis for these observations is well established. T2DM engenders a prothrombotic platelet phenotype through multiple converging mechanisms. Chronic hyperglycemia promotes non-enzymatic glycation of platelet membrane glycoproteins, impairing insulin-mediated inhibition of platelet activation and increasing intracellular calcium mobilization.¹⁸ Concurrently, oxidative stress (driven by reactive oxygen species (ROS) generated via glucose auto-oxidation, the polyol pathway, and, AGE receptor signaling, attenuates endothelial nitric oxide and prostacyclin production, thereby removing two of the principal physiological brakes on platelet aggregation.^{18,19} The net result is an upregulation of thromboxane A₂ (TXA₂) synthesis: larger platelets, which carry a greater density of dense granules and arachidonic acid-metabolizing enzymes, release proportionally more TXA₂ per activation event, amplifying thrombogenic signaling within the microcirculation.²⁰ Elevated MPV and PDW reflect the preferential recruitment of this younger, morphologically larger, and more

metabolically active megakaryocyte progeny from bone marrow reserves under conditions of sustained vascular stress.^{11,21} P-LCR, reflecting the proportion of platelets exceeding 12 fL, is a direct surrogate for this large-platelet dominance, corroborated by its significant correlation ($p = +0.309$) in our cohort. Reddy et al. specifically proposed MPV, PDW, and P-LCR as markers of subclinical platelet activation and suggested that automated cell counter-derived indices could serve as economical surrogates for monitoring glycemic sequelae even in primary care settings.²¹

The clinical characteristics of our two groups illuminate several confounders that must be considered when interpreting platelet index data. Patients with microvascular complications were significantly older (58.3 vs. 47.1 years), had a markedly longer disease duration (16.0 vs. 10.2 years), worse glycemic control (HbA1c 9.7 vs. 8.0%), higher BMI, greater prevalence of hypertension (66.4 vs. 39.1%), and poorer treatment compliance. Each of these variables is independently associated with platelet hyperactivation. Duration of diabetes and HbA1c levels, in particular, have been repeatedly shown to positively correlate with MPV and PDW.^{13,22} Aktas and Aktuglu demonstrated that patients with HbA1c > 10% exhibited significantly higher MPV and PDW than those with lower HbA1c, concluding that both parameters can be monitored as adjuncts to glycemic assessment even in primary healthcare facilities.²² Hypertension, highly prevalent in our complication group, is itself an independent stimulus for platelet activation via endothelial shear stress mechanisms, further compounding the thrombogenic burden.¹⁸ These clinical realities do not negate the discriminating value of platelet indices but underscore the necessity of multivariate adjustment in future studies designed to isolate their independent contribution to microvascular risk beyond conventional risk factors.

From a broader perspective, the findings of this study contribute to an evolving paradigm in which the hematological system is recognized as an active participant in diabetic microvascular pathogenesis. The traditional framework focused almost exclusively on metabolic derangements at the level of the capillary wall: basement

membrane thickening, pericyte dropout, and VEGF-driven neovascularization.^{8,9} Emerging evidence increasingly implicates platelet-megakaryocyte crosstalk, platelet-derived growth factor (PDGF) and VEGF release from alpha granules, and microvesicle shedding as proximate drivers of neovascularization and vascular remodeling in diabetic retinopathy and nephropathy.^{8,19} Mirghani, in a 2025 World Journal of Diabetes review, enumerated a broader range of clinical implications of platelet indices in diabetes, including their potential relevance to monitoring treatment response and identifying patients at highest risk of progressing to end-stage complications.²² In settings where specialist investigations such as fundoscopy, nerve conduction studies, and urinary albumin quantification may not be uniformly accessible, an elevated MPV or PDW on a routine CBC could serve as a low-threshold trigger for expedited specialist referral.

Several limitations of this study warrant acknowledgement. First, as a cross-sectional design, causality cannot be established. Second, although patients on antiplatelet agents were excluded, other medications, including metformin and statins, may exert modest independent effects on platelet morphology that were not formally assessed. Third, the study was conducted at a single tertiary care center in Islamabad, and findings may not be fully generalizable to community or rural populations where T2DM phenotype, nutritional status, and healthcare access differ substantially. Fourth, multivariate regression analysis to isolate the independent contribution of platelet indices from correlated variables such as HbA1c, disease duration, hypertension, and BMI were not performed; future studies incorporating logistic regression or propensity-score matching would substantially strengthen inferential quality. Finally, inter-analyzer variation in reference ranges for MPV and PDW may affect reproducibility of absolute values across centers.

CONCLUSION

Our findings confirm and support the clinical utility of routinely available platelet indices as low-cost, non-invasive adjuncts for microvascular risk stratification. Given Pakistan's extraordinary diabetes burden and the inequitable distribution of specialist diagnostic

services, integrating platelet index monitoring into standard diabetic follow-up offers a pragmatic, immediately implementable strategy to identify patients at elevated microvascular risk and trigger timely specialist referral. Prospective longitudinal studies incorporating multivariate adjustment and ROC-derived cut-off validation are now warranted to establish whether these indices can serve not only as risk markers but as surrogate endpoints for monitoring the efficacy of glycemic and antiplatelet interventions.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

SOURCE OF FUNDING

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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