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PLATELET-TO-LYMPHOCYTE RATIO AS A PREDICTOR OF CARDIOVASCULAR RISK IN TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Cardiovascular disease is a major cause of morbidity and mortality in type 2 diabetes mellitus (T2DM). Chronic inflammation, endothelial dysfunction, and platelet activation contribute to accelerated atherosclerosis. The platelet-to-lymphocyte ratio (PLR), derived from a routine complete blood count, may provide a simple marker of cardiovascular-risk burden.

Objective: To evaluate the association between PLR and estimated 10-year cardiovascular risk among adults with T2DM and to assess the ability of PLR to identify patients with higher cardiovascular risk.

Materials and Methods: This multicentre cross-sectional analytical study was conducted in the Departments of General Medicine at Geetanjali Institute of Medical Sciences, Jaipur, and Mahatma Gandhi Medical College and Hospital, Jaipur, from 1 July 2025 to 1 July 2026. A total of 200 adults aged 40–74 years with T2DM and without established cardiovascular disease were included. PLR was calculated from the platelet count and absolute lymphocyte count obtained from the same complete blood-count sample. Ten-year cardiovascular risk was estimated using the 2019 World Health Organization laboratory-based cardiovascular disease risk charts for the South Asia region. One-way analysis of variance, Spearman correlation, multivariable logistic regression, and receiver operating characteristic analyses were performed.

Results: The mean age of the 200 participants was 56.5 ± 8.1 years; 119 (59.5%) were men. Mean PLR was 128.8 ± 32.9. PLR increased from 106.7 ± 28.6 in the <5% cardiovascular-risk category to 156.9 ± 30.9 in the ≥30% category (one-way ANOVA $F(4,195)=17.19$, $p<0.001$). PLR correlated positively with estimated 10-year cardiovascular risk (Spearman $\rho=0.511$, $p<0.001$). Fifty-one participants (25.5%) had an estimated risk ≥20%. PLR identified this higher-risk group with an area under the ROC curve of 0.778 (95% CI 0.702–0.844); the optimal cutoff was 134.5, with sensitivity of 78.4% and specificity of 68.5%. Each 10-unit increase in PLR was associated with higher odds of estimated risk ≥20% after adjustment for HbA1c, BMI, and eGFR (adjusted OR 1.39, 95% CI 1.19–1.62, $p<0.001$).

Conclusion: PLR showed a graded association with increasing estimated 10-year cardiovascular risk in patients with T2DM and demonstrated moderate discriminatory ability for identifying participants with risk ≥20%. PLR may be useful as an inexpensive adjunct to conventional cardiovascular-risk assessment, but prospective studies using incident cardiovascular events are required before it can be used as a stand-alone prediction marker.

KEYWORDS: type 2 diabetes mellitus; platelet-to-lymphocyte ratio; cardiovascular risk; inflammation; atherosclerosis; WHO cardiovascular risk score

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major global health problem and is closely linked to premature cardiovascular morbidity and mortality. Atherosclerotic cardiovascular disease remains a leading cause of adverse outcomes among people with diabetes, in whom hypertension, dyslipidaemia, obesity, smoking, renal dysfunction, and metabolic abnormalities frequently cluster. Contemporary diabetes guidance therefore emphasizes systematic cardiovascular-risk assessment and aggressive management of modifiable risk factors [1].

The pathogenesis of cardiovascular disease in T2DM extends beyond conventional risk factors. Chronic hyperglycaemia, oxidative stress, endothelial dysfunction, low-grade inflammation, altered immune responses, and increased platelet reactivity jointly accelerate atherosclerosis and thrombosis. The worldwide epidemiology of T2DM and its vascular complications highlights the need for practical risk markers that can be incorporated into routine clinical evaluation [3].

The platelet-to-lymphocyte ratio (PLR) is calculated by dividing the platelet count by the absolute lymphocyte count. It combines two biologically relevant components: platelets, which participate in thrombosis and inflammatory signalling, and lymphocytes, whose relative reduction can reflect systemic inflammatory or

physiological stress. PLR has been described as an inexpensive and readily available inflammatory marker associated with adverse outcomes across several cardiovascular conditions [4].

Previous studies have shown that elevated PLR is associated with greater coronary atherosclerotic burden and complexity [6,7], while a meta-analysis in acute coronary syndrome found higher PLR to be associated with mortality and cardiovascular events [8]. In T2DM specifically, PLR has been linked to glycaemic control [9], diabetic microvascular complications [10], and adverse outcomes after percutaneous coronary intervention [5].

However, much of the available cardiovascular literature concerns patients with established coronary disease or acute coronary syndromes. Fewer studies have evaluated the relationship between PLR and estimated primary-prevention cardiovascular-risk burden among Indian adults with T2DM. The present study therefore evaluated PLR in relation to estimated 10-year cardiovascular risk among patients with T2DM attending two tertiary-care institutions in Jaipur, Rajasthan.

AIM AND OBJECTIVES

Aim

To evaluate platelet-to-lymphocyte ratio as a predictor of cardiovascular risk among patients with type 2 diabetes mellitus.

Primary Objective

To determine the association between PLR and estimated 10-year cardiovascular risk among adults with T2DM.

Secondary Objectives

i. To compare PLR across categories of estimated 10-year cardiovascular risk.
ii. To evaluate correlations of PLR with age, duration of diabetes, blood pressure, glycaemic indices, lipid parameters, and renal function.
iii. To determine whether PLR was associated with higher cardiovascular risk after adjustment for relevant variables not embedded in the WHO cardiovascular-risk score.
iv. To assess the discriminatory performance and an optimal cutoff of PLR for identifying patients with an estimated 10-year cardiovascular risk ≥20%.

MATERIALS AND METHODS

Study Design and Setting

A multicentre cross-sectional analytical study was conducted in the Department of General Medicine, Geetanjali Institute of Medical Sciences, Jaipur, and the Department of General Medicine, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India.

Study Period

The study was conducted from 1 July 2025 to 1 July 2026.

Study Population

Adults with established T2DM attending the General Medicine services of the participating institutions during the study period were screened for eligibility. Patients fulfilling the predefined inclusion and exclusion criteria were enrolled in the study.

Sample Size Calculation

The sample size was calculated to detect a modest correlation between PLR and estimated 10-year cardiovascular risk. Assuming an expected correlation coefficient of r=0.20, a two-sided α of 0.05, and a statistical power of 80%, the required sample size was calculated using Fisher's Z transformation:

$$n = [(Z_{1-\alpha/2} + Z_{1-\beta}) / \{0.5 \times \ln((1+r)/(1-r))\}]^2 + 3$$

With $Z_{1-\alpha/2}=1.96$, $Z_{1-\beta}=0.84$, and r=0.20, the calculated minimum sample size was 193.75, which was rounded to 194 participants. The final sample size was increased to 200 participants to improve precision and permit exploratory multivariable regression and receiver operating characteristic analyses.

Sampling Technique

Consecutive eligible participants were enrolled until the required sample size of 200 participants was achieved.

Inclusion Criteria

1. Age 40–74 years.
2. Established T2DM.
3. Clinically stable at the time of assessment.
4. Complete clinical and laboratory information required for calculation of cardiovascular risk and PLR.
5. Written informed consent according to the approved study protocol.

Exclusion Criteria

1. Previous myocardial infarction, stroke, coronary or peripheral revascularization, or established peripheral arterial disease.
2. Acute coronary syndrome at presentation.
3. Acute infection or febrile illness during the preceding two weeks.
4. Active inflammatory or autoimmune disease.
5. Active malignancy.
6. Known hematological disorder affecting platelet or lymphocyte counts.

7. Current systemic corticosteroid or immunosuppressive therapy.
8. Recent major surgery or trauma.
9. Pregnancy.
10. End-stage kidney disease or dialysis.
11. Incomplete clinical or laboratory data required for the primary analyses.

Clinical and Laboratory Assessment

Demographic and clinical information, including age, sex, smoking status, duration of diabetes, history of hypertension, anthropometric measurements, and blood pressure, was recorded using a structured case-record form. Laboratory investigations included complete blood count, fasting plasma glucose, glycated haemoglobin (HbA1c), serum creatinine, estimated glomerular filtration rate (eGFR), total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides. Platelet count and absolute lymphocyte count obtained from the same complete blood-count sample were used for calculation of PLR.

Calculation of Platelet-to-Lymphocyte Ratio

PLR = Platelet count / Absolute lymphocyte count

PLR was evaluated as a continuous variable for the principal analyses.

Cardiovascular-Risk Assessment

Ten-year cardiovascular risk was estimated using the 2019 World Health Organization laboratory-based cardiovascular disease risk charts for the South Asia region [2]. The model incorporated age, sex, smoking status, systolic blood pressure, diabetes status, and total cholesterol and estimated the 10-year probability of a fatal or nonfatal myocardial infarction or stroke. Participants were categorized into <5%, 5–<10%, 10–<20%, 20–<30%, and ≥30% estimated risk. For ROC and exploratory logistic regression analyses, higher cardiovascular risk was defined a priori as an estimated 10-year risk ≥20%.

Statistical Analysis

Continuous variables were summarized as mean ± standard deviation, and categorical variables were expressed as frequencies and percentages. PLR values across cardiovascular-risk categories were compared using one-way analysis of variance. Associations between PLR and estimated 10-year cardiovascular-risk percentage, as well as other clinical and biochemical variables, were assessed using Spearman correlation analysis. Receiver operating characteristic analysis was performed to assess the ability of PLR to discriminate participants with an estimated 10-year cardiovascular risk ≥20%, and the optimal PLR cutoff was identified using the Youden index. Because age, sex, smoking status, systolic blood pressure, diabetes, and total cholesterol were already components of the WHO cardiovascular-risk model, these variables were not re-entered into the exploratory regression model to minimize mathematical coupling. The multivariable logistic regression model was adjusted for HbA1c, body mass index, and eGFR. A two-sided p value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics

A total of 200 patients with T2DM were included in the study. The mean age of the participants was 56.5 ± 8.1 years. Of the 200 participants, 119 (59.5%) were male and 81 (40.5%) were female. The mean duration of diabetes was 7.2 ± 4.1 years, while the mean body mass index was 27.1 ± 3.4 kg/m². Hypertension was present in 85 (42.5%) participants, and 26 (13.0%) were current smokers.

The mean systolic and diastolic blood pressures were 134.1 ± 12.9 mmHg and 80.2 ± 7.9 mmHg, respectively. Mean HbA1c was 8.09 ± 1.21%. The mean total cholesterol, LDL cholesterol, HDL cholesterol, and triglyceride levels were 187.9 ± 30.7 mg/dL, 134.7 ± 32.7 mg/dL, 44.5 ± 9.5 mg/dL, and 168.7 ± 65.9 mg/dL, respectively. Mean serum creatinine was 0.97 ± 0.21 mg/dL, while mean eGFR was 88.0 ± 12.1 mL/min/1.73 m². Mean platelet count was 267.4 ± 52.0 ×10⁹/L, mean absolute lymphocyte count was 2.21 ± 0.69 ×10⁹/L, and mean PLR was 128.8 ± 32.9. The mean estimated 10-year cardiovascular risk was 14.1 ± 10.8%.

Table 1. Baseline demographic, clinical, and laboratory characteristics of the study participants

Variable	Value (n=200)
Age, years	56.5 ± 8.1
Male, n (%)	119 (59.5)
Female, n (%)	81 (40.5)
Duration of diabetes, years	7.2 ± 4.1
BMI, kg/m²	27.1 ± 3.4
Current smokers, n (%)	26 (13.0)
Hypertension, n (%)	85 (42.5)
Systolic BP, mmHg	134.1 ± 12.9
Diastolic BP, mmHg	80.2 ± 7.9
HbA1c, %	8.09 ± 1.21
Total cholesterol, mg/dL	187.9 ± 30.7
LDL cholesterol, mg/dL	134.7 ± 32.7

Variable	Value (n=200)
HDL cholesterol, mg/dL	44.5 ± 9.5
Triglycerides, mg/dL	168.7 ± 65.9
Serum creatinine, mg/dL	0.97 ± 0.21
eGFR, mL/min/1.73 m²	88.0 ± 12.1
Platelet count, ×10⁹/L	267.4 ± 52.0
Absolute lymphocyte count, ×10⁹/L	2.21 ± 0.69
Platelet-to-lymphocyte ratio	128.8 ± 32.9
Estimated 10-year CVD risk, %	14.1 ± 10.8

Distribution of Estimated Cardiovascular Risk

According to the WHO 10-year cardiovascular-risk classification, 42 (21.0%) participants had a risk of <5%, 55 (27.5%) had a risk of 5–<10%, 52 (26.0%) had a risk of 10–<20%, 31 (15.5%) had a risk of 20–<30%, and 20 (10.0%) had a risk of ≥30%. Overall, 51 participants (25.5%) were classified as having an estimated 10-year cardiovascular risk ≥20%.

Table 2. Distribution of participants according to estimated 10-year cardiovascular risk

WHO cardiovascular-risk category	n	%
<5%	42	21.0
5–<10%	55	27.5
10–<20%	52	26.0
20–<30%	31	15.5
≥30%	20	10.0
Total	**200**	**100.0**

Platelet-to-Lymphocyte Ratio Across Cardiovascular-Risk Categories

A progressive increase in PLR was observed with increasing cardiovascular-risk category. The mean PLR was 106.7 ± 28.6 among participants with a risk of <5%, 120.5 ± 27.2 in those with a risk of 5–<10%, 131.4 ± 27.9 in the 10–<20% group, 151.3 ± 30.5 in the 20–<30% group, and 156.9 ± 30.9 among participants with an estimated risk of ≥30%. The between-group difference was statistically significant (one-way ANOVA $F(4,195)=17.19$, $p<0.001$).

Table 3. Platelet-to-lymphocyte ratio according to cardiovascular-risk category

Cardiovascular-risk category	n	PLR, mean ± SD
<5%	42	106.7 ± 28.6
5–<10%	55	120.5 ± 27.2
10–<20%	52	131.4 ± 27.9
20–<30%	31	151.3 ± 30.5
≥30%	20	156.9 ± 30.9

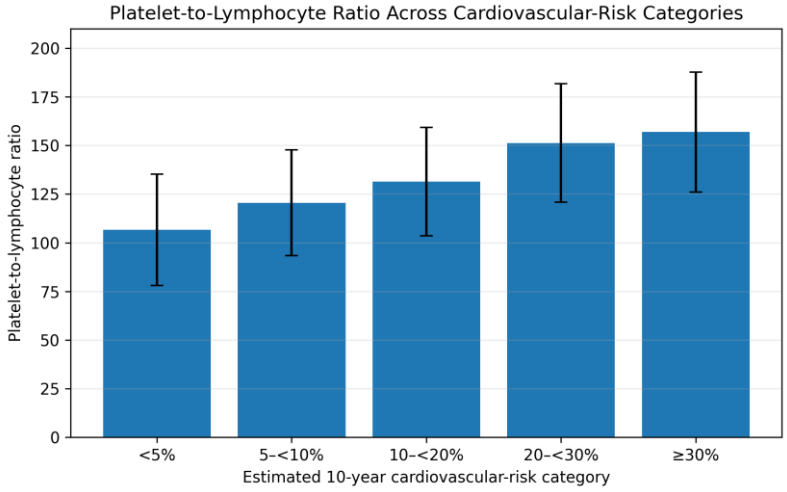

Figure 1. Mean platelet-to-lymphocyte ratio across estimated 10-year cardiovascular-risk categories. Error bars represent standard deviation.

Correlation of PLR with Cardiovascular Risk and Other Variables

PLR showed a moderate positive correlation with estimated 10-year cardiovascular risk (Spearman $\rho=0.511$, $p<0.001$). Significant positive correlations were also observed between PLR and age ($\rho=0.393$, $p<0.001$), systolic blood pressure ($\rho=0.240$, $p<0.001$), HbA1c ($\rho=0.215$, $p=0.002$), and total cholesterol ($\rho=0.150$, $p=0.034$). PLR showed a significant inverse correlation with eGFR ($\rho=-0.296$, $p<0.001$). No statistically

significant correlations were observed between PLR and duration of diabetes, BMI, LDL cholesterol, HDL cholesterol, or triglycerides.

Table 4. Correlation of PLR with clinical and biochemical variables

Variable	Spearman ρ	p value
Estimated 10-year cardiovascular risk	0.511	<0.001
Age	0.393	<0.001
Duration of diabetes	0.058	0.416
BMI	−0.099	0.165
Systolic blood pressure	0.240	<0.001
HbA1c	0.215	0.002
Total cholesterol	0.150	0.034
LDL cholesterol	0.123	0.083
HDL cholesterol	−0.038	0.589
Triglycerides	0.023	0.748
eGFR	−0.296	<0.001

Multivariable Logistic Regression Analysis

Multivariable logistic regression analysis was performed to evaluate factors associated with an estimated 10-year cardiovascular risk ≥20%. After adjustment for HbA1c, BMI, and eGFR, PLR remained significantly associated with higher cardiovascular risk. For every 10-unit increase in PLR, the odds of having an estimated cardiovascular risk ≥20% increased by 39% (adjusted OR 1.39, 95% CI 1.19–1.62, p<0.001). eGFR was independently and inversely associated with higher cardiovascular risk (adjusted OR 0.91, 95% CI 0.88–0.95, p<0.001). HbA1c and BMI were not significantly associated with higher cardiovascular-risk classification in the adjusted model.

Table 5. Multivariable logistic regression analysis for estimated cardiovascular risk ≥20%

Variable	Adjusted OR	95% CI	p value
PLR, per 10-unit increase	1.39	1.19–1.62	<0.001
HbA1c, per 1% increase	1.03	0.74–1.44	0.845
BMI, per 1 kg/m² increase	0.99	0.87–1.12	0.844
eGFR, per 1 mL/min/1.73 m² increase	0.91	0.88–0.95	<0.001

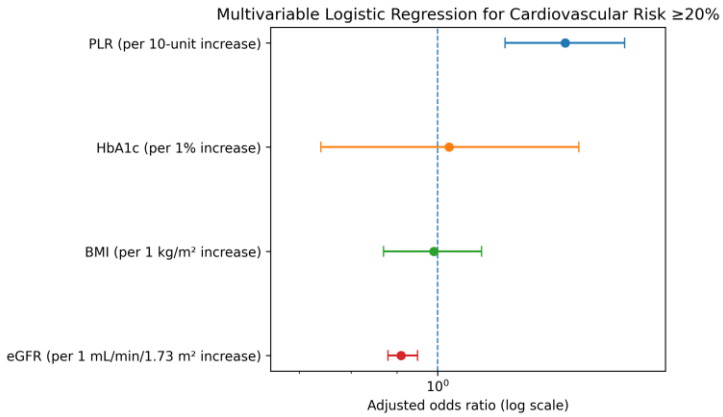

Figure 2. Adjusted odds ratios and 95% confidence intervals from the multivariable logistic regression model for estimated 10-year cardiovascular risk ≥20%.

Receiver Operating Characteristic Analysis

Receiver operating characteristic analysis was performed to assess the ability of PLR to identify participants with an estimated 10-year cardiovascular risk ≥20%. PLR demonstrated an area under the curve of 0.778 (95% CI 0.702–0.844), indicating moderate discriminatory ability. The optimal PLR cutoff was 134.5, which provided a sensitivity of 78.4% and a specificity of 68.5%. The corresponding Youden index was 0.469.

DISCUSSION

The present study evaluated the relationship between PLR and estimated 10-year cardiovascular risk among patients with T2DM. The principal finding was a progressive increase in PLR with increasing cardiovascular-risk category. PLR increased from 106.7 ± 28.6 in participants with an estimated cardiovascular risk <5% to 156.9 ± 30.9 in those with a risk ≥30%, and the overall between-group difference was statistically significant. In addition, PLR showed a moderate positive correlation with estimated 10-year cardiovascular risk (ρ=0.511, p<0.001). These findings suggested that increasing PLR was associated with a progressively greater burden of conventional cardiovascular risk among patients with T2DM.

The observed relationship between PLR and cardiovascular risk is biologically plausible. T2DM is characterized by chronic low-grade inflammation, oxidative stress, endothelial dysfunction, and increased platelet reactivity, all of which contribute to accelerated atherosclerosis and thrombotic complications. PLR integrates two hematological components relevant to these processes. An increased platelet count may reflect enhanced platelet activation and thrombo-inflammatory activity, whereas a relatively reduced lymphocyte count may reflect systemic inflammatory stress. Consequently, a higher PLR may represent an imbalance toward a more pro-inflammatory and prothrombotic state. Previous cardiovascular literature has similarly described PLR as a readily obtainable systemic inflammatory marker associated with cardiovascular disease severity and adverse outcomes [4].

The progressive rise in PLR across cardiovascular-risk categories observed in the present study was consistent with previous studies evaluating PLR in coronary artery disease. Kurtul et al. reported that higher PLR was associated with greater severity and complexity of coronary artery disease among patients with acute coronary syndromes [6]. Akboga et al. similarly demonstrated an association between elevated PLR, inflammatory activity, and more severe coronary atherosclerosis in patients with stable coronary artery disease [7]. A systematic review and meta-analysis by Li et al. also showed that higher PLR was associated with adverse outcomes among patients with acute coronary syndromes [8]. Although these studies predominantly involved patients with established cardiovascular disease, their findings support the concept that PLR reflects cardiovascular inflammatory and atherosclerotic burden.

The association of PLR with T2DM has also been reported in previous studies. Atak et al. demonstrated higher PLR among patients with T2DM and reported positive correlations between PLR and markers of glycaemic control [9]. In the present study, PLR showed a statistically significant positive correlation with HbA1c ($\rho=0.215$, $p=0.002$), suggesting that poorer glycaemic control may be associated with greater systemic inflammatory activity. This relationship may be explained by the effects of persistent hyperglycaemia on oxidative stress, endothelial dysfunction, platelet activation, and inflammatory signalling.

PLR was also significantly correlated with age ($\rho=0.393$, $p<0.001$), systolic blood pressure ($\rho=0.240$, $p<0.001$), and total cholesterol ($\rho=0.150$, $p=0.034$). These findings indicated that higher PLR tended to occur in participants with a greater burden of established cardiovascular-risk factors. However, no significant association was observed between PLR and duration of diabetes, BMI, LDL cholesterol, HDL cholesterol, or triglycerides. This suggested that PLR may not simply reflect overall metabolic disturbance but may predominantly represent inflammatory and thrombotic pathways that overlap only partially with conventional lipid and anthropometric risk factors.

An important finding was the inverse association between PLR and renal function. PLR demonstrated a significant negative correlation with eGFR ($\rho=-0.296$, $p<0.001$), and eGFR remained independently associated with higher cardiovascular-risk classification in multivariable analysis. Renal dysfunction is an established marker of increased cardiovascular burden in patients with diabetes and is itself associated with systemic inflammation and endothelial dysfunction. Li et al. also reported associations of PLR with diabetic microvascular complications, including diabetic nephropathy and retinopathy [10]. Therefore, the inverse relationship between PLR and eGFR observed in the present study may reflect the interaction between systemic inflammation, diabetic renal involvement, and cardiovascular-risk burden.

The multivariable analysis provided further support for the association between PLR and cardiovascular risk. After adjustment for HbA1c, BMI, and eGFR, each 10-unit increase in PLR was associated with a 39% increase in the odds of having an estimated 10-year cardiovascular risk ≥20% (adjusted OR 1.39, 95% CI 1.19–1.62, $p<0.001$). This finding suggested that the relationship between PLR and higher cardiovascular-risk classification was not explained solely by glycaemic control, obesity, or renal function. Nevertheless, the association should be interpreted cautiously because the study was cross-sectional and the outcome represented calculated cardiovascular risk rather than prospectively observed cardiovascular events.

The present findings were also broadly consistent with those reported by Song et al., who evaluated PLR in a large prospective cohort of patients undergoing percutaneous coronary intervention. That study demonstrated an association between elevated PLR and adverse cardiovascular and cerebrovascular events, with notable associations among patients with T2DM [5]. The current study differed in an important respect: individuals with established cardiovascular disease were excluded, and PLR was examined in relation to estimated primary-prevention cardiovascular-risk burden. This approach is clinically relevant because PLR can be calculated from a routine complete blood count without requiring an additional test or specialized laboratory facility.

ROC analysis demonstrated that PLR had moderate discriminatory ability for identifying participants with an estimated 10-year cardiovascular risk ≥20%, with an AUC of 0.778 (95% CI 0.702–0.844). A PLR cutoff of 134.5 provided a sensitivity of 78.4% and specificity of 68.5%, with a Youden index of 0.469. These findings indicated that PLR could help distinguish patients with a relatively higher cardiovascular-risk burden. However, the observed sensitivity and specificity were insufficient to support the use of PLR as an independent cardiovascular-risk prediction tool. Rather, PLR may have potential as an adjunctive marker alongside established cardiovascular-risk assessment methods.

The WHO cardiovascular-risk model used in the present study was appropriate for evaluating cardiovascular-risk burden in this population because it incorporated routinely available clinical variables, including age, sex,

smoking status, systolic blood pressure, diabetes status, and total cholesterol [2]. Because these variables were already components of the calculated WHO risk score, re-entering them into a regression model in which the WHO risk category was the dependent outcome could have resulted in mathematical coupling. The adjusted model therefore included HbA1c, BMI, and eGFR rather than variables already incorporated within the risk equation.

From a clinical perspective, PLR has several practical advantages. It is inexpensive, readily available, and can be calculated from a routinely performed complete blood count without additional blood sampling or laboratory expenditure. In resource-constrained settings, these characteristics may make PLR useful as a supplementary marker for identifying patients with T2DM who have a greater inflammatory and cardiovascular-risk burden. Nevertheless, PLR should not replace established cardiovascular-risk scores because it does not directly incorporate several major determinants of cardiovascular disease.

Strengths

- Multicentre design involving two tertiary-care institutions.
- Use of a low-cost biomarker available from a routine complete blood count.
- Use of a regionally calibrated WHO cardiovascular-risk framework.
- Evaluation of PLR as a continuous variable and across ordered cardiovascular-risk categories.
- Use of correlation, adjusted regression, and ROC analyses to examine association and discrimination from complementary perspectives.

LIMITATIONS

- The cross-sectional design could demonstrate association but could not establish temporality or causality.
- Estimated cardiovascular risk was a model-derived surrogate rather than prospectively observed myocardial infarction, stroke, or cardiovascular death.
- PLR may be influenced by transient inflammatory states, medications, occult infections, and unrecognized hematological conditions.
- A single PLR measurement may not represent long-term inflammatory status.
- Tertiary-care recruitment may limit generalizability to community populations or other geographical regions.

CONCLUSION

PLR increased progressively with estimated 10-year cardiovascular risk among patients with T2DM and remained independently associated with an estimated risk ≥20% after adjustment for HbA1c, BMI, and eGFR. Its moderate discriminatory performance suggested that PLR may serve as a simple and inexpensive adjunct to conventional cardiovascular-risk assessment. However, prospective longitudinal studies evaluating incident cardiovascular events are required to determine whether PLR provides incremental prognostic value beyond established cardiovascular-risk models.

DECLARATIONS

Informed Consent

Written informed consent was obtained from all participants according to the study protocol.

Consent for Publication

Not applicable.

Funding

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Conflict of Interest

The authors declared no conflict of interest.

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