Alqalam Journal of Medical and Applied Sciences. 2026;9(5):1399-1402
https://doi.org/10.54361/ajmas.269539

Original article

Association Between Systemic Inflammatory Markers NLR and PLR in Patients with Type 2 Diabetes Mellitus

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Abstract
Type 2 diabetes is a metabolic inflammatory disease. However, Neutrophil-to-Lymphocyte ratio (NLR) and Platelet-to-Lymphocyte ratio (PLR) have emerged as potential inflammatory markers in patients with type 2 diabetes mellitus (T2DM). Therefore, its relationship with glycemic parameters remains controversial. This study aims to investigate the association between NLR, PLR, glycemic parameters, and hematological indices in patients with T2DM. A cross-sectional study was conducted on 65 patients with T2DM. Fasting blood sugar (FBS), glycated hemoglobin (HbA1c), and hematological indices were obtained from the medical record, including a complete blood count. NLR was calculated as the absolute neutrophil count divided by the absolute lymphocyte count, and PLR was calculated as the absolute platelet count divided by the absolute lymphocyte count. Data normality was assessed using the Shapiro-Wilk test. The data were non-normally distributed; Spearman's rank correlation was performed to investigate the associations between inflammatory markers and glycemic parameters. Statistical significance was set at $p < 0.05$. A total of 65 patients with T2DM were included (36 males, 55.4%; 29 females, 44.6%) with a mean age of 50±6.65 years. The mean level of FBS was 210±84 mg/dL, and the mean level of HbA1c was 9.34±2.16%. The study patient's cohort exhibited elevated systemic inflammatory markers. The mean NLR value was 3.0±2.89, and the mean PLR value was 153.08±130.89, both of which are higher than the standard reference ranges for healthy individuals. However, no statistically significant correlations were found between the inflammatory markers (NLR and PLR) and glycemic parameters. A strong and statistically significant positive correlation was observed between NLR and PLR (rho = 0.516, p < 0.001). NLR did not significantly correlate with FBS (rho = 0.025, p = 0.846) or HbA1c (rho = -0.044, p = 0.726). Similarly, no significant association was observed between PLR and FBS (rho = 0.100, p = 0.428) or HbA1c (rho = -0.048, p = 0.703).
Keywords. Diabetes Type 2, Systemic Inflammatory Markers, Neutrophil-to-lymphocyte Ratio, Platelets-to-lymphocytes Ratio, Glycemic Control.

Introduction

Type 2 diabetes mellitus is a complicated chronic disorder with hyperglycemia and high insulin levels. However, increased systemic inflammation markers indicate risk of microvascular and macrovascular damage linked to cardiovascular disease (CVD), diabetic retinopathy (DR), kidney disease, and diabetic neuropathy (DN) [1,2]. Recently, immunometabolism has highlighted the clinical utility of hemogram-derived inflammatory markers, which have been shown to have prognostic value for T2D disease progression and the severity of complications. Furthermore, non-invasive tools are low-cost for type 2 diabetes management [3]. The platelet-to-lymphocyte ratio (PLR) has been shown to track systemic inflammation and is related to other markers. PLR can be calculated by the ratio of the absolute platelet count to the absolute lymphocyte count. Thrombosis and atherosclerosis are linked to platelet activation, particularly in patients with T2DM [4]. Among these indices, the Neutrophil-to-Lymphocyte Ratio (NLR) and the Platelet-to-Lymphocyte Ratio (PLR) have emerged as independent predictors of the systemic inflammatory burden [3]. Elevated NLR levels reflect an imbalance between the innate immune response (neutrophils) and adaptive immunity (lymphocytes), while PLR serves as a surrogate marker for cytokine-induced thrombopoiesis and pro-thrombotic states associated with chronic inflammation [5,6]. In addition, the meta-analytical evidence has established a strong association between these ratios and the severity of diabetic complications, emphasizing their predictive value in clinical settings [2]. Furthermore, contemporary studies have demonstrated a significant positive correlation between these inflammatory markers and glycemic control, as measured by glycated hemoglobin (HbA1c), as well as the duration of the disease [7].

In patients with T2DM, a high inflammatory burden—often indicated by a synergistic rise in both NLR and PLR—has been linked to increased risks of subclinical atherosclerosis and peripheral neuropathy [8,9]. Despite the wealth of global data, there is a continuous need to evaluate these biomarkers within specific clinical cohorts to establish localized diagnostic cut-off points and predictive thresholds [10].

This study aims to investigate the association between NLR, PLR, and metabolic parameters in a group of 65 patients with T2DM. By analyzing the correlation between these hematological ratios and glycemic markers, this research seeks to reinforce the diagnostic potential of inexpensive CBC-derived indices in predicting the inflammatory status and potential vascular risks in diabetic patients.

Received*: 17-03-2026 -* ***Accepted****: 15-05-2026 -* ***Published****: 21-05-2026*

Methods

This cross-sectional study enrolled 65 patients with type 2 diabetes who were admitted to Derna Medical Center, a private clinic in Derna City, for routine follow-up analysis from January 2026 to March 2026.
For all these individuals, those with acute or chronic infections, autoimmune diseases, or hematological disorders (e.g., leukemia) were excluded. Additionally, the BC-5000 Mindray Automated Hematology Analyzer was used to collect venous whole blood and provide a complete blood count with differential. The ratios of neutrophils, platelets, and lymphocytes were used to construct the systemic inflammatory indices NLR and PLR, respectively. The RIELE Photometer 4040 Biochemistry Analyzer and the Mindray BS-240 were used to assess fasting blood sugar (FBS) and HbA1c%. Quantitative variables were expressed as Mean± mean±standard deviation (SD), while categorical ones were presented as frequencies and percentages.
The Shapiro-Wilk test was used to evaluate the normality of the data. Spearman's rank correlation was used to examine the relationships between inflammatory markers and hyperglycemia parameters because the data were not regularly distributed.
Frequencies were used to display categorical variables. The mean ± standard deviation was used to display continuous variables. The results with a P value <0.05 were considered statistically significant. SPSS statistical software (SPSS for Windows, version 26.0) was used for data analysis.

Results

(Table 1) presents the study population, which consisted of 36 males (55.4%) and 29 females (44.6%), with a mean age of 50 ± 6.65 years. Regarding glycemic status, the participants showed elevated mean levels of fasting blood sugar (210 ± 84 mg/dL) and glycated hemoglobin (9.34 ± 2.16%). Hematological analysis revealed a mean platelet count of 275.17 ± 77.72 × 10^3/μL and a mean WBC count of 8.32 ± 3.90 × 10^9/L. The inflammatory markers NLR and PLR were recorded at 3.08 ± 2.89 and 153 ± 130, respectively**.**

Table 1. Baseline clinical and hematological characteristics of the study populations (N=65)

Variables	Statistics
Gender Male Female	 36(55.4%) 29 (44.6%)
Age (years), Mean ± SD	50 ± 6.65
Fasting Blood Sugar (mg/dL)	210 ± 84
Glycated hemoglobin (HbA1c%)	9.34 ± 2.16
WBC (×109/L), Mean ± SD	8.32±3.90
Platelet count (103/μL) Mean±SD	275.17 ± 77.72
Neutrophil (%),Mean±SD	62.52 ± 12.70
Lymphocyte (%), Mean ± SD	29 ± 11.62
NLR	3.08 ± 2.89
PLR	153 ± 130

Table 2 illustrates the correlation between glycemic indexes and inflammatory markers (NLR and PLR). The Spearman's correlation analysis revealed that there were no statistically significant correlations between Fasting Blood Sugar (FBS) and either NLR (r = 0.025, p = 0.846) or PLR (r = 0.100, p = 0.428). Similarly, no significant association was observed between glycated hemoglobin (HbA1c) and the inflammatory ratios (p > 0.05). "However, a significant moderate positive correlation was observed between NLR and PLR (r = 0.516, p < 0.001). This indicates that as the Neutrophil-to-Lymphocyte Ratio increases, the Platelet-to-Lymphocyte Ratio tends to increase significantly within the study population."

Table 2. Spearman's Correlation Analysis Between NLR, PLR, and Glycemic Indexes

Variables	NLR		PLR	
	r	P-value	r	P-value
FBS	0.025	0.846	0.100	0.428
HbA1c	-0.044	0.726	-0.048	0.703
NLR	-	-	0.516	<0.001·

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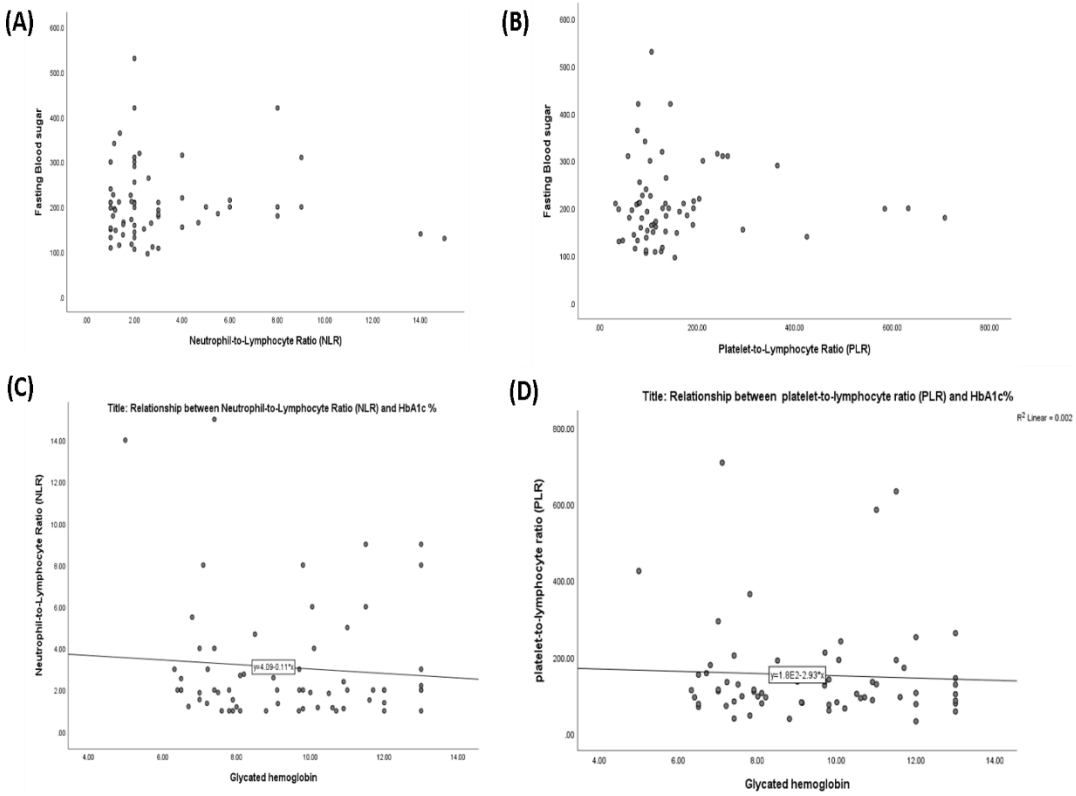

Figure 1. Scatter plots showing the correlation analysis between inflammatory markers and glycemic indices (n=65).
(A) Correlation between NLR and FBS; (B) Correlation between PLR and FBS; (C) Correlation between NLR and HbA1c; (D) Correlation between PLR and HbA1c. The results indicate no significant linear correlation between inflammatory ratios and glucose levels (P > 0.05).

Discussion

Type 2 diabetes mellitus (T2DM), which is caused by chronic hyperglycemia and its associated micro- and macrovascular problems. Achieving and maintaining glycemic control is still essential to reducing the burden of its complications [11]. The majority of participants in this cross-sectional study are men, middle-aged to older adults (≥ 50 years); NLR was significantly higher among the participants, which means the NLR value of 3.0±2.89. This finding suggests that while systemic inflammation is a hallmark of type 2 Diabetes Mellitus, rather than the degree of hyperglycemia itself, and an HbA1c value of ≤ 9.0%. However, an HbA1c level higher than 9 is one of the criteria for insulin therapy, which can be linked to the participants' poor glycemic control [12]. Higher NLR is an imbalance between lymphocyte apoptosis and neutrophil activation due to hyperglycemia [13]. PLR may help forecast the onset and degree of control of type 2 diabetes. However, more extensive prospective studies are required to confirm its association with HbA1c.

The present study investigated the correlation between inflammatory biometrics (NLR and PLR) and glycemic indices (FBS and HbA1c) in patients with diabetes. A primary finding of this research is the highly significant positive correlation between the Neutrophil-to-Lymphocyte Ratio (NLR) and the Platelet-to-Lymphocyte Ratio (PLR) ($r = 0.516$, $P < 0.001$). This strong association is consistent with previous studies [14, 15], confirming that both ratios serve as synchronized indicators of the systemic inflammatory response. Since both markers share the lymphocyte count as a common denominator, their simultaneous elevation reflects a coordinated shift in the innate and adaptive immune branches during diabetic inflammation [16].

Regarding glycemic control, the results showed no statistically significant correlation between NLR or PLR and either Fasting Blood Sugar (FBS) or HbA1c ($P > 0.05$). This lack of significance aligns with the findings of Demirtas et al. [17], who observed that while these markers are elevated in diabetic populations, they do not always mirror the fluctuations in glucose levels or "glycemic memory" represented by HbA1c. Instead, NLR and PLR may be more indicative of the presence of chronic microvascular complications rather than immediate metabolic control [18].

The absence of a significant link in this cohort could also be attributed to the modest sample size (n = 65), which may limit the statistical power to detect subtle linear relationships. Furthermore, as HbA1c represents a 3-month average, the dynamic nature of neutrophil and platelet counts might reflect acute physiological stressors not captured by long-term glucose monitoring. These findings highlight the importance of using

Received*: 17-03-2026 -* ***Accepted****: 15-05-2026 -* ***Published****: 21-05-2026*

NLR and PLR as independent markers of inflammatory status rather than direct substitutes for glycemic assessment.

Conclusion

This study concludes that there is a significant positive correlation between the Neutrophil-to-Lymphocyte Ratio (NLR) and the Platelet-to-Lymphocyte Ratio (PLR) among the study population, confirming their role as synchronized markers of systemic inflammation. However, the lack of a significant association with FBS and HbA1c suggests that these inflammatory indices may fluctuate independently of immediate glycemic control. These results highlight the clinical utility of NLR and PLR as independent tools for monitoring inflammatory status in diabetic patients. For future research, it is recommended to conduct longitudinal studies with larger sample sizes to further investigate the predictive value of these markers in the progression of diabetic complications.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this study.

Funding

This research did not receive any specific funding from public, commercial, or not-for-profit sectors.

Author contribution

All authors have contributed to the manuscript.

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Received*: 17-03-2026 -* ***Accepted****: 15-05-2026 -* ***Published****: 21-05-2026*