Section: Miscellaneous

Original Research Article

# ASSOCIATION OF NEUTROPHIL–LYMPHOCYTE AND PLATELET–LYMPHOCYTE RATIOS WITH DIABETIC KIDNEY DISEASE IN TYPE 2 DIABETES MELLITUS

*Mohd Shaigan[1], Kartheek R Balapala[2], Sudeep Saran[3], Sumit Giri[4]*

[1]*Department of Community Medicine, AP Venkateshwara Institute of Medical Sciences, Gajraula, Uttar Pradesh, India.*
[2]*Doctoral Lecturer, MCS School of Medicine, Copperbelt University, Kitwe, Zambia*
[3]*Senior Physician and Director, Saran Hospital and Institute of Paramedical Sciences, Bareilly, Uttar Pradesh, India.*
[4]*Professor, Department of Pathology, NCRIMS, Meerut, India.*

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**Corresponding Author:**
**Dr. Mohd Shaigan,**
Department of Community Medicine, AP Venkateshwara Institute of Medical Sciences, Gajraula Uttar Pradesh, India.
Email: shaikhshaigan@gmail.com

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ABSTRACT

**Background:** Diabetic kidney disease (DKD) is one of the most common microvascular complications of type 2 diabetes mellitus (T2DM) and a leading cause of end-stage renal disease in India. Conventional biomarkers such as albuminuria and estimated glomerular filtration rate (eGFR) often identify DKD only after significant renal damage has occurred. Inflammation is increasingly recognized as a key mechanism in the pathogenesis of DKD. The neutrophil–lymphocyte ratio (NLR) and platelet–lymphocyte ratio (PLR), derived from routine complete blood counts, have emerged as novel inflammatory markers. This study aimed to evaluate NLR and PLR as potential indicators of DKD in patients with T2DM.

**Material and Methods:** A hospital-based cross-sectional study was conducted among 487 patients with T2DM attending a tertiary care hospital in central India. Participants were categorized into two groups: Group I – T2DM without DKD (n=243), and Group II – T2DM with DKD (n=244), based on urine albumin–creatinine ratio (UACR) and eGFR. Demographic, biochemical, and hematological parameters were analyzed, and NLR and PLR were calculated from complete blood counts. Correlations between these ratios and renal parameters were assessed using Pearson's correlation test.

**Results:** Patients with DKD were older and had longer diabetes duration, higher blood pressure, and poorer glycemic control compared to those without DKD ($p<0.001$). Mean serum creatinine and blood urea were significantly elevated, while eGFR was lower in the DKD group ($p<0.001$). Both NLR ($2.93 \pm 1.02$ vs. $2.06 \pm 0.74$) and PLR ($146.8 \pm 39.2$ vs. $118.4 \pm 31.5$) were significantly higher among DKD patients ($p<0.001$). NLR showed a strong negative correlation with eGFR ($r = -0.46$) and a positive correlation with UACR ($r = +0.52$), while PLR correlated inversely with eGFR ($r = -0.38$) and positively with UACR ($r = +0.44$) (all $p<0.001$).

**Conclusion:** Elevated NLR and PLR are significantly associated with the presence and severity of DKD in patients with T2DM. These easily obtainable, cost-effective inflammatory markers may serve as useful adjuncts for early detection and risk stratification of diabetic kidney disease, especially in resource-limited clinical settings.

**Keywords:** Type 2 diabetes mellitus; Diabetic kidney disease; Neutrophil–lymphocyte ratio; Platelet–lymphocyte ratio; Inflammatory markers.

## INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. Among its numerous microvascular complications, diabetic kidney disease (DKD) represents one of the most common and debilitating

outcomes, accounting for nearly 40% of end-stage renal disease (ESRD) cases worldwide.[1,2] The global prevalence of type 2 diabetes mellitus (T2DM) continues to rise, particularly in low- and middle-income countries such as India, where early detection of DKD remains a major challenge despite the availability of conventional markers like urinary albumin excretion and estimated glomerular filtration rate (eGFR).[3] These markers, while clinically valuable, often reflect renal injury only after substantial nephron damage has occurred, underscoring the need for simple, inexpensive, and reliable biomarkers for early identification of DKD.

Recent research has increasingly highlighted the role of inflammation in the pathogenesis and progression of DKD. Chronic low-grade inflammation contributes to endothelial dysfunction, mesangial expansion, and tubulointerstitial fibrosis, ultimately leading to renal impairment.[4] Among various inflammatory indices derived from routine complete blood count (CBC) parameters, the neutrophil–lymphocyte ratio (NLR) and platelet–lymphocyte ratio (PLR) have emerged as potential systemic inflammatory markers.[5,6] These ratios integrate the inflammatory and immune components of circulating leukocytes and platelets, providing a readily available and cost-effective reflection of the body's inflammatory status.

The NLR has been proposed as an indicator of subclinical inflammation and vascular injury in diabetic patients, showing correlations with insulin resistance, microvascular complications, and cardiovascular morbidity.[7] Similarly, the PLR has been associated with platelet activation and endothelial dysfunction—key mechanisms implicated in diabetic microangiopathy.[8] Several studies have suggested that both NLR and PLR are elevated in patients with T2DM, particularly those with nephropathy, and may correlate with albuminuria and declining renal function.[9-11] However, findings across studies have not been entirely consistent, and the utility of these indices as screening or predictive markers for DKD remains to be validated in diverse populations.

Given their simplicity, accessibility, and potential pathophysiological relevance, evaluating NLR and PLR as inflammatory markers in DKD may enhance early risk stratification and guide timely therapeutic interventions. Hence, the present study was undertaken with an aim to evaluate and compare the levels of neutrophil–lymphocyte ratio and platelet–lymphocyte ratio in patients with type 2 diabetes mellitus with and without diabetic kidney disease, and to explore their possible association with the severity of renal impairment.

## MATERIALS AND METHODS

### Study Design and Setting

This hospital-based cross-sectional study was conducted in the Department of Medicine, at a tertiary care teaching hospital in North India, over a period of 2 years between July 2023 to June 2025. The study aimed to evaluate the association of neutrophil–lymphocyte ratio (NLR) and platelet–lymphocyte ratio (PLR) with diabetic kidney disease (DKD) among patients with type 2 diabetes mellitus (T2DM). The study protocol was reviewed and approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrolment.

### Study Population

A total of 487 patients diagnosed with T2DM as per the American Diabetes Association (ADA) 2023 criteria were recruited from the outpatient and inpatient departments. The participants were aged between 35 and 75 years and had a diabetes duration of at least one year. Patients were stratified into two groups: Group I included T2DM patients without diabetic kidney disease (normoalbuminuria) (n=243), and Group II included T2DM patients with diabetic kidney disease (n=244), defined by the presence of albuminuria and/or reduced estimated glomerular filtration rate (eGFR).

### Inclusion and Exclusion Criteria

Inclusion criteria comprised patients with established T2DM, stable glycemic control over the past three months, and willingness to participate. Patients were excluded if they had acute or chronic infections, inflammatory or autoimmune disorders, hematological diseases, malignancies, hepatic dysfunction, recent surgeries, or were on medications known to alter leukocyte or platelet counts such as corticosteroids or immunosuppressants. Those with type 1 diabetes mellitus or other known causes of renal disease were also excluded to minimize confounding factors.

### Clinical and Biochemical Evaluation

Detailed demographic data, medical history, and anthropometric measurements including age, sex, duration of diabetes, body mass index (BMI), and blood pressure were recorded using a structured proforma. Venous blood samples were collected under aseptic precautions after an overnight fast of 8–10 hours. Routine biochemical investigations included fasting blood glucose, postprandial blood glucose, glycated hemoglobin (HbA1c), serum creatinine, lipid profile, and blood urea nitrogen. The eGFR was calculated using the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation.

Urine samples were obtained for assessment of albumin-to-creatinine ratio (UACR) using spot urine specimens. Based on UACR values, patients were classified as having normoalbuminuria (<30 mg/g), microalbuminuria (30–300 mg/g), or macroalbuminuria (>300 mg/g). DKD was defined as UACR ≥30 mg/g and/or eGFR <60 mL/min/1.73 m² persisting for at least three months.

### Hematological Parameters and Calculation of Ratios

Complete blood counts (CBC) were performed using an automated hematology analyzer. The neutrophil

count, lymphocyte count, and platelet count were recorded for each participant. The neutrophil–lymphocyte ratio (NLR) was calculated as the absolute neutrophil count divided by the absolute lymphocyte count, and the platelet–lymphocyte ratio (PLR) was obtained by dividing the platelet count by the lymphocyte count. All hematological parameters were measured on the same day as biochemical investigations to ensure comparability.

**Statistical Analysis**

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) as appropriate, while categorical variables were expressed as frequencies and percentages. The normality of data distribution was assessed using the Shapiro–Wilk test. Intergroup comparisons between T2DM patients with and without DKD were performed using the independent samples t-test or Mann–Whitney U test for continuous variables and the chi-square test for categorical variables. Correlation analyses between NLR, PLR, and renal parameters (UACR, eGFR) were performed using Pearson's or Spearman's correlation coefficients as applicable. A p-value <0.05 was considered statistically significant.

## RESULTS

Among the 487 participants studied, 243 (49.9%) had type 2 diabetes mellitus (T2DM) without diabetic kidney disease (Group I), while 244 (50.1%) had T2DM with DKD (Group II). The mean age of DKD patients was significantly higher (58.4 ± 10.1 years) compared to those without DKD (53.7 ± 9.8 years, $p < 0.001$). The duration of diabetes was also longer in the DKD group (median 11 [8–15] years vs. 6 [4–10] years, $p < 0.001$). Although body mass index did not differ significantly between the two groups ($p = 0.328$), both systolic and diastolic blood pressures were notably higher in DKD patients (138.8 ± 14.9 mmHg and 85.7 ± 9.1 mmHg, respectively; $p < 0.001$ for both). The prevalence of hypertension (64.3% vs. 37.9%, $p < 0.001$) and smoking (16.0% vs. 8.6%, $p = 0.018$) was significantly higher in DKD participants, suggesting an additive vascular risk burden in this group. [Table 1]

**Table 1: Baseline Demographic and Clinical Characteristics of Study Participants**

| Variable | Group I (T2DM without DKD, n=243) | Group II (T2DM with DKD, n=244) | p-value |
|---|---|---|---|
| | Frequency (%)/mean ± SD/median [IQR] | | |
| **Age (years)** | 53.7 ± 9.8 | 58.4 ± 10.1 | <0.001 |
| **Male:Female ratio** | 1.3:1 (138/105) | 1.6:1 (151/93) | 0.212 |
| **Duration of diabetes (years)** | 6 [4–10] | 11 [8–15] | <0.001 |
| **BMI (kg/m²)** | 26.7 ± 3.9 | 27.1 ± 4.2 | 0.328 |
| **Systolic BP (mmHg)** | 129.4 ± 12.6 | 138.8 ± 14.9 | <0.001 |
| **Diastolic BP (mmHg)** | 81.2 ± 8.3 | 85.7 ± 9.1 | <0.001 |
| **Smoking history** | 21 (8.6%) | 39 (16.0%) | 0.018 |
| **Hypertension** | 92 (37.9%) | 157 (64.3%) | <0.001 |

DKD – diabetic kidney disease; BMI – body mass index; BP – blood pressure

Biochemical analysis revealed that DKD patients exhibited poorer glycemic and renal profiles than their non-DKD counterparts. Mean fasting (156.5 ± 42.3 mg/dL) and postprandial (236.9 ± 59.2 mg/dL) glucose levels were significantly higher in DKD subjects compared with those without DKD ($p < 0.001$). Similarly, HbA1c was elevated in the DKD group (8.3 ± 1.1% vs. 7.5 ± 0.8%, $p < 0.001$), reflecting inadequate long-term glycemic control. Serum creatinine and blood urea levels were markedly increased in DKD (1.56 ± 0.43 mg/dL and 44.8 ± 13.5 mg/dL, respectively; $p < 0.001$ for both), with a corresponding decline in eGFR (52.7 ± 17.8 vs. 91.8 ± 14.3 mL/min/1.73 m², $p < 0.001$). Lipid profile assessment showed significantly higher total cholesterol, triglycerides, and LDL-C, along with lower HDL-C levels in DKD patients (all $p < 0.01$), consistent with the dyslipidemic pattern observed in diabetic microvascular disease. [Table 2]

**Table 2: Biochemical Parameters of the Study Population**

| Parameter | Group I (T2DM without DKD, n=243) | Group II (T2DM with DKD, n=244) | p-value |
|---|---|---|---|
| | mean ± SD | | |
| **Fasting plasma glucose (mg/dL)** | 138.2 ± 34.7 | 156.5 ± 42.3 | <0.001 |
| **Postprandial glucose (mg/dL)** | 204.7 ± 52.6 | 236.9 ± 59.2 | <0.001 |
| **HbA1c (%)** | 7.5 ± 0.8 | 8.3 ± 1.1 | <0.001 |
| **Serum creatinine (mg/dL)** | 0.89 ± 0.17 | 1.56 ± 0.43 | <0.001 |
| **eGFR (mL/min/1.73 m²)** | 91.8 ± 14.3 | 52.7 ± 17.8 | <0.001 |
| **Blood urea (mg/dL)** | 26.5 ± 6.2 | 44.8 ± 13.5 | <0.001 |
| **Total cholesterol (mg/dL)** | 184.3 ± 32.6 | 197.9 ± 37.1 | 0.002 |
| **Triglycerides (mg/dL)** | 152.6 ± 41.8 | 169.5 ± 45.3 | 0.001 |
| **HDL-C (mg/dL)** | 44.1 ± 7.8 | 41.3 ± 7.1 | 0.001 |
| **LDL-C (mg/dL)** | 108.2 ± 26.4 | 121.5 ± 29.8 | <0.001 |

HbA1c – glycated hemoglobin; eGFR – estimated glomerular filtration rate; HDL-C – high-density lipoprotein cholesterol; LDL-C – low-density lipoprotein cholesterol

Based on UACR categorization, all patients in Group I exhibited normoalbuminuria (<30 mg/g), confirming absence of DKD. In contrast, the DKD group showed 61.1% of patients with microalbuminuria (30–300 mg/g) and 38.9% with macroalbuminuria (>300 mg/g). This distribution highlights that a majority of DKD patients presented at the microalbuminuric stage, while nearly two-fifths had progressed to overt nephropathy. [Table 3].

**Table 3: Distribution of Urine Albumin–Creatinine Ratio (UACR) Categories**

| UACR Category | UACR (mg/g) | Group I (T2DM without DKD, n=243) | Group II (T2DM with DKD, n=244) |
|---|---|---|---|
| | | Frequency (%) | |
| **Normoalbuminuria** | **<30** | 243 (100%) | 0 (0%) |
| **Microalbuminuria** | **30–300** | 0 (0%) | 149 (61.1%) |
| **Macroalbuminuria** | **>300** | 0 (0%) | 95 (38.9%) |

UACR – urine albumin-to-creatinine ratio

The hematological profile demonstrated a significant inflammatory pattern among DKD patients. Total leukocyte and neutrophil counts were higher in Group II compared to Group I (8.24 ± 2.01 vs. 7.13 ± 1.78 $\times 10^9$ /L, $p < 0.001$; and 5.26 ± 1.43 vs. 4.31 ± 1.21 $\times 10^9$ /L, $p < 0.001$, respectively), while lymphocyte counts were lower (1.94 ± 0.58 vs. 2.27 ± 0.66 $\times 10^9$ /L, $p < 0.001$). Platelet counts showed a mild but significant elevation in DKD patients ($p = 0.042$). Both derived inflammatory indices—NLR and PLR—were markedly higher in the DKD group (NLR: 2.93 ± 1.02 vs. 2.06 ± 0.74; PLR: 146.8 ± 39.2 vs. 118.4 ± 31.5; $p < 0.001$ for both). [Table 4]

**Table 4: Comparison of Hematological Parameters and Inflammatory Ratios Between Groups**

| Parameter | Group I (T2DM without DKD, n=243) | Group II (T2DM with DKD, n=244) | p-value |
|---|---|---|---|
| | mean ± SD | | |
| **Total leukocyte count ($\times 10^9$ /L)** | 7.13 ± 1.78 | 8.24 ± 2.01 | <0.001 |
| **Neutrophil count ($\times 10^9$ /L)** | 4.31 ± 1.21 | 5.26 ± 1.43 | <0.001 |
| **Lymphocyte count ($\times 10^9$ /L)** | 2.27 ± 0.66 | 1.94 ± 0.58 | <0.001 |
| **Platelet count ($\times 10^9$ /L)** | 265.2 ± 56.3 | 277.9 ± 63.4 | 0.042 |
| **Neutrophil–lymphocyte ratio (NLR)** | 2.06 ± 0.74 | 2.93 ± 1.02 | <0.001 |
| **Platelet–lymphocyte ratio (PLR)** | 118.4 ± 31.5 | 146.8 ± 39.2 | <0.001 |

NLR – neutrophil–lymphocyte ratio; PLR – platelet–lymphocyte ratio

Correlation analysis demonstrated significant associations between inflammatory ratios and renal function indices. NLR showed a moderate negative correlation with eGFR ($r = -0.46$, $p < 0.001$) and a positive correlation with UACR ($r = +0.52$, $p < 0.001$). Similarly, PLR correlated inversely with eGFR ($r = -0.38$, $p < 0.001$) and positively with UACR ($r = +0.44$, $p < 0.001$). HbA1c and systolic blood pressure also exhibited significant correlations with both renal markers ($p < 0.001$), underscoring the multifactorial contribution of poor glycemic control and hemodynamic stress to kidney injury. [Table 5]

**Table 5: Correlation of NLR and PLR with Renal Function Parameters in the Study Population (n = 487)**

| Correlation Parameter | eGFR (r value) | p-value | UACR (r value) | p-value |
|---|---|---|---|---|
| **Neutrophil–lymphocyte ratio (NLR)** | −0.46 | <0.001 | 0.52 | <0.001 |
| **Platelet–lymphocyte ratio (PLR)** | −0.38 | <0.001 | 0.44 | <0.001 |
| **HbA1c** | −0.41 | <0.001 | 0.36 | <0.001 |
| **Systolic BP** | −0.33 | <0.001 | 0.31 | <0.001 |

r: Pearson's correlation; Negative correlation (−) indicates inverse relation with eGFR; positive (+) indicates direct relation with UACR; all correlations significant at $p < 0.001$.

## DISCUSSION

Diabetic kidney disease (DKD) remains a leading cause of chronic kidney disease and end-stage renal disease worldwide, with increasing prevalence in India due to rising rates of type 2 diabetes mellitus (T2DM).[12,13] Early identification of DKD is crucial to implement timely interventions that can slow progression, yet conventional markers such as albuminuria and estimated glomerular filtration rate (eGFR) often detect renal injury only after substantial nephron loss. This study aimed to evaluate neutrophil–lymphocyte ratio (NLR) and platelet–lymphocyte ratio (PLR) as potential inflammatory biomarkers for DKD among Indian T2DM patients.

Our findings indicate that patients with DKD were older, had a longer duration of diabetes, higher systolic and diastolic blood pressures, and greater prevalence of hypertension compared to those without DKD. These observations are consistent with previous Indian studies by Khandare et al., Chittawar et al., Gupta et al., and Sandhu et al., which demonstrate that aging, prolonged hyperglycemia, and hypertension collectively accelerate microvascular injury in T2DM.[14,15,16,17] Although body mass index did not differ significantly, the

higher prevalence of hypertension and smoking in DKD patients highlights the synergistic role of vascular risk factors in the pathogenesis of diabetic nephropathy.[17]

As expected, DKD patients exhibited poorer glycemic control, reflected by higher fasting and postprandial glucose levels and elevated HbA1c. Renal function parameters, including serum creatinine and blood urea, were significantly deranged in DKD, accompanied by a substantial decline in eGFR. Dyslipidemia, with higher total cholesterol, triglycerides, and LDL-C, as well as lower HDL-C, was also prominent. These findings align with previous Indian and global cohorts in the studies by Rai et al., Sisodia et al., Hirano et al., and Di Marco et al., emphasizing the interrelationship between metabolic dysregulation, endothelial dysfunction, and renal injury.[18,19,20,21]

A key finding of our study is the significant elevation of NLR and PLR among DKD patients. NLR increased from 2.06 ± 0.74 in non-DKD to 2.93 ± 1.02 in DKD patients ($p < 0.001$), while PLR rose from 118.4 ± 31.5 to 146.8 ± 39.2 ($p < 0.001$). Total leukocyte and neutrophil counts were elevated, and lymphocyte counts were reduced in DKD, reflecting a shift toward a proinflammatory milieu.[20] Platelet counts were also mildly increased, consistent with platelet activation in the context of endothelial dysfunction.[21] These data suggest that systemic inflammation, detectable through routine hematological parameters, plays a central role in the pathogenesis of DKD.[22]

Several studies support the utility of NLR and PLR as markers of diabetic microvascular complications.[23,24] A study by Swathi et al., reported significantly higher NLR in T2DM patients with nephropathy compared to those without.[23] Similarly, Jaaban et al., observed that PLR was elevated in diabetic nephropathy and correlated with albuminuria severity.[24] Our findings not only confirm these associations in an Indian population but also demonstrate that both NLR and PLR correlate significantly with renal function indices: NLR showed a moderate negative correlation with eGFR ($r = -0.46$) and a positive correlation with UACR ($r = +0.52$), while PLR showed inverse correlation with eGFR ($r = -0.38$) and positive correlation with UACR ($r = +0.44$), all statistically significant ($p < 0.001$). These results suggest that these inflammatory ratios could reflect the severity of kidney involvement.[25]

Chronic low-grade inflammation is a recognized contributor to DKD pathogenesis. Hyperglycemia induces oxidative stress, activates the renin-angiotensin-aldosterone system, and triggers endothelial dysfunction, leading to mesangial expansion and tubulointerstitial fibrosis.[26] Neutrophils contribute to tissue injury via reactive oxygen species and proinflammatory cytokines, whereas lymphopenia reflects impaired immunoregulation. Platelet activation further promotes microvascular thrombosis and inflammatory signalling.[27,28] Consequently, elevated NLR and PLR likely represent a cumulative effect of these pathological processes, integrating neutrophil-mediated inflammation, lymphocyte-mediated immunoregulation, and platelet-driven vascular injury.[27,28]

**Clinical Implications**

NLR and PLR, derived from routine complete blood counts, are inexpensive, readily available, and reproducible markers. Their significant correlation with renal dysfunction suggests that they may serve as early indicators of DKD, particularly in resource-limited settings where access to advanced biomarkers or frequent microalbuminuria testing may be challenging. Incorporating these indices into routine T2DM follow-up could allow clinicians to identify high-risk patients and implement early interventions, including glycemic optimization, blood pressure control, and renin-angiotensin system blockade.

**Limitations**

This study has some limitations. First, its cross-sectional design limits causal inference regarding the role of inflammation in DKD progression. Second, we did not assess other inflammatory biomarkers, such as C-reactive protein or interleukin-6, which could have provided complementary mechanistic insights. Third, the study was conducted at a single tertiary care center, which may limit generalizability to broader populations. Finally, factors such as subclinical infections, diet, or medication adherence, which can influence NLR and PLR, were not fully controlled.

## CONCLUSION

In conclusion, patients with T2DM and DKD exhibit significantly elevated NLR and PLR compared to those without DKD. These indices correlate with the severity of renal impairment and reflect systemic inflammation contributing to diabetic nephropathy. Given their simplicity, accessibility, and cost-effectiveness, NLR and PLR may serve as adjunctive markers for early detection and risk stratification of DKD in routine clinical practice, particularly in resource-constrained settings. Further prospective studies are warranted to validate their predictive value and assess their utility in guiding therapeutic interventions.

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