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Original Article

Hematological and Inflammatory Predictors of Diabetic Nephropathy in Type 2 Diabetes Mellitus

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ABSTRACT

Background and Objectives: Diabetic nephropathy (DN) is a major microvascular complication of Type 2 Diabetes Mellitus (T2DM). Chronic low-grade inflammation and platelet hyperactivity drive renal vascular damage. This study evaluated the predictive value of routine hematological parameters, inflammatory ratios, and platelet morphometric indices in T2DM patients with diabetic nephropathy.

Materials and Methods: A cross-sectional study was conducted comparing 50 T2DM patients with diabetic nephropathy against 50 age- and sex-matched diabetic controls without nephropathy. Complete blood count, platelet indices (MPV, PDW), inflammatory ratios (NLR, PLR), and biochemical renal markers were measured and analyzed using independent t-tests, Pearson correlations, and multivariate logistic regression.

Results: Patients with diabetic nephropathy exhibited significantly higher NLR (2.80 ± 0.65 vs 1.60 ± 0.38, $p < 0.001$), MPV (11.20 ± 1.15 fL vs 8.30 ± 0.85 fL, $p < 0.001$), and PDW (12.20 ± 1.30 fL vs 11.00 ± 0.95 fL, $p < 0.001$). NLR and MPV correlated strongly with serum creatinine and microalbuminuria ($p < 0.001$) and remained strong independent predictors of nephropathy on multivariate analysis ($p < 0.001$).

Conclusion: Elevated NLR and MPV serve as reliable, cost-effective biomarkers for predicting subclinical inflammation and renal microvascular damage in T2DM.

Keywords: Diabetes Mellitus, Type 2; Diabetic Nephropathies; Blood Platelets; Mean Platelet Volume; Inflammation Mediators; Neutrophils.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) has reached pandemic proportions globally, driven by rising rates of obesity, physical inactivity, and population aging [1]. Among the chronic microvascular complications of T2DM, diabetic nephropathy (DN)—also termed diabetic kidney disease (DKD)—represents the most prevalent and severe manifestation [2]. DN develops in approximately 30% to 40% of individuals with diabetes and remains the leading underlying cause of end-stage renal disease (ESRD) worldwide [3].

The clinical course of DN typically progresses from early glomerular hyperfiltration to persistent microalbuminuria, overt proteinuria, and eventual decline in estimated glomerular filtration rate (eGFR) [4]. However, traditional markers used to monitor renal impairment, such as microalbuminuria and serum creatinine, possess notable limitations; microalbuminuria can exhibit high intra-individual variability and non-progression, while serum creatinine often rises only after substantial irreversible nephron loss has occurred [5].

The classical understanding of DN focused primarily on metabolic pathways and hemodynamic alterations, such as intra-glomerular hypertension, dysregulated renin-angiotensin-aldosterone axis activity, and hyper-glycemia-induced advanced glycation end-products (AGEs) [6]. However, emerging translational and clinical evidence indicates that persistent, low-grade systemic inflammation and vascular immune dysregulation are pivotal drivers in initiating and perpetuating renal injury [7].

Among immune mediators, neutrophils act as rapid responders, secreting reactive oxygen species, myeloperoxidase, and proteases that compromise endothelial cell integrity and disrupt the glomerular basement membrane [8]. Conversely, relative lymphopenia frequently occurs during systemic physiological stress and immune exhaustion, secondary to increased lymphocyte apoptosis induced by elevated cortisol and oxidative stress [8]. Derived from the standard complete blood count (CBC), the neutrophil-to-lymphocyte ratio (NLR) effectively integrates these two concurrent immune dynamics—neutrophil-mediated acute inflammation and lymphocyte-mediated adaptive regulation—into a stable, reliable index of systemic inflammatory burden [8].

In T2DM, sustained hyperglycemia, dyslipidemia, and oxidative stress induce a hyperreactive platelet phenotype characterized by increased osmotic swelling, enhanced granule secretion, and accelerated turnover [9]. Automated platelet morphometric indices—specifically Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW)—serve as sensitive markers of platelet activation and size heterogeneity [9]. Larger platelets are metabolically and enzymatically more active than smaller, older platelets, generating higher amounts of thromboxane A2 and expressing increased surface adhesion molecules [9].

Furthermore, the platelet-to-lymphocyte ratio (PLR) combines thrombotic propensity with adaptive immune suppression, yielding a dual marker of hypercoagulability and systemic inflammation [10]. Although individual parameters such as total white blood cell count or absolute platelet count can fluctuate wildly due to acute hemodilution or transient physiological stress, ratio-based indices (NLR, PLR) and structural platelet indices (MPV, PDW) demonstrate superior stability and diagnostic consistency. Therefore, this study was undertaken to systematically evaluate hematological parameters, platelet morphological indices (MPV, PDW), and inflammatory ratios (NLR, PLR) in patients with Type 2 Diabetes Mellitus, assessing their correlation with clinical renal parameters to determine their potential as accessible, predictive markers for Diabetic Nephropathy.

MATERIALS AND METHODS

Study Setting: This prospective case-control investigation was conducted at the Government Mohan Kumaramangalam Medical College (GMKMC) and Hospital, Salem, Tamil Nadu. The data collection involved patients visiting the Outpatient Departments (OPD) of Diabetology, Nephrology, and Ophthalmology, as well as those admitted to the General Medicine wards.

Study Population and Grouping: The study population comprised patients diagnosed with Type 2 Diabetes Mellitus (T2DM) attending the outpatient department or admitted to the inpatient wards. Based on thorough clinical evaluation, laboratory investigations, and renal function profiling, participants were categorized into two primary groups.
The Diabetic Nephropathy Group (Cases) consisted of patients with T2DM exhibiting clinical evidence of renal impairment, defined by persistent microalbuminuria (urinary albumin-to-creatinine ratio [UACR] between 30 and 300 mg/g) or macroalbuminuria (UACR > 300 mg/g), and/or an elevated serum creatinine level (> 1.2 mg/dL) with a reduced estimated glomerular filtration rate (eGFR < 60 mL/min/1.73 m^2). The Diabetic Control Group (Controls) comprised age- and sex-matched T2DM patients without clinical or laboratory evidence of microvascular complications, demonstrated by normoalbuminuria (UACR < 30 mg/g) and normal serum creatinine levels.

Selection Criteria: The study included male and female participants aged between 30 and 70 years who had an established diagnosis of Type 2 Diabetes Mellitus based on the American Diabetes Association (ADA) criteria and provided voluntary written informed consent. Conversely, patients with Type 1 Diabetes Mellitus, secondary forms of diabetes, or primary non-diabetic renal diseases such as glomerulonephritis, polycystic kidney disease, or acute kidney injury were excluded.
Additional exclusion criteria encompassed active systemic or localized infections, fever, acute inflammatory states, known hematological disorders such as severe anemia, leukemia, idiopathic thrombocytopenic purpura, or hemoglobinopathies, and active malignancies or severe chronic liver disease. Patients receiving hemodialysis, systemic corticosteroid therapy, immunosuppressive drugs, or blood transfusions within the preceding three months were also excluded from participation.
Sample Size and Sampling Technique: A total sample size of 100 was determined based on the feasibility and the requirement to maintain a 1:1 ratio between cases and controls. Consecutive sampling was utilized, enrolling every eligible patient who met the strict inclusion and exclusion criteria during the study period from November 2022 to June 2024.

Sample Collection and Laboratory Measurements: Under aseptic precautions, a total of 5 mL of venous blood was collected from each participant following an 8 to 12 hour overnight fast. A 2 mL aliquot was immediately transferred into an Ethylenediaminetetraacetic acid (EDTA) tube for complete blood count (CBC) and platelet parameter analysis using an automated 5-part differential hematology analyzer within 2 hours of collection. The evaluated parameters included

hemoglobin concentration (g/dL), total leukocyte count (WBC, 10^3 /µL), absolute neutrophil count (ANC), absolute lymphocyte count (ALC), platelet count, 10^3 /µL), mean platelet volume (MPV, fL), platelet distribution width (PDW, fL), and plateletcrit (PCT, %). From these values, key systemic inflammatory ratios were calculated, including the Neutrophil-to-Lymphocyte Ratio (NLR = ANC/ ALC) and the Platelet-to-Lymphocyte Ratio (PLR = Platelet Count / ALC).

The remaining 3 mL of blood was drawn into plain clot-activator tubes and centrifuged at 3000 rpm for 10 minutes to separate serum for biochemical processing. Serum creatinine, blood urea, and fasting blood glucose (FBG) were analyzed using an automated biochemistry analyzer. Glycosylated hemoglobin (HbA1c) levels were measured via High-Performance Liquid Chromatography (HPLC). In addition, spot morning urine samples were collected from each subject to quantify urinary albumin and urinary creatinine for the determination of the UACR.

Ethical Issues: The study protocol received formal approval from the Institutional Ethics Committee (IEC) of Government Mohan Kumaramangalam Medical College (Ref. No. GMKMC&H/4341/IEC/02/2018-13). All participants provided written informed consent after a thorough explanation of the study's objectives in their native language. Patient confidentiality was strictly maintained throughout the research process.

Statistical Analysis: Data were analysed using SPSS version 25.0. Continuous variables were tested for normality using the Kolmogorov-Smirnov test and expressed as Mean ± Standard Deviation (SD) for normally distributed data or as Median (Interquartile Range) for skewed distributions, while categorical variables were summarized as frequencies and percentages. Differences in quantitative hematological and biochemical parameters between the Diabetic Nephropathy group and the Diabetic Control group were evaluated using the independent Student's t-test or the Mann-Whitney U test, depending on data normality. Categorical parameters were compared across groups using the Chi-square test. Furthermore, Pearson or Spearman rank correlation coefficients were calculated to establish the relationships between inflammatory/platelet indices (NLR, PLR, MPV, PDW) and indicators of renal damage (Serum Creatinine, Blood Urea, UACR). A two-tailed p-value of < 0.05 was considered statistically significant across all comparisons.

RESULTS

A total of 100 participants with Type 2 Diabetes Mellitus (T2DM) were evaluated and divided into two distinct study groups: the Diabetic Nephropathy Group (n = 50, cases) and the Diabetic Control Group (n = 50, controls without nephropathy). The mean age of participants in the Diabetic Nephropathy Group was 57.42 ± 8.65 years, while the Diabetic Control Group had a mean age of 55.18 ± 7.92 years. Males comprised 58.0% (n = 29) of the nephropathy group and 52.0% (n = 26) of the control group. The baseline demographic and biochemical characteristics are presented in Table 1.

Table 1: ***Demographic and Baseline Biochemical Profiles of the Study Groups***

Parameter	Diabetic Nephropathy Group (n=50)	Diabetic Control Group (n=50)	Test Statistic (t / χ2)	p-value
Age (years)	57.42 ± 8.65	55.18 ± 7.92	t = 1.355	0.178
Gender (Male/Female)	29 / 21 (58.0% / 42.0%)	26 / 24 (52.0% / 48.0%)	χ2 = 0.368	0.544
Duration of Diabetes (years)	11.84 ± 4.12	6.26 ± 3.05	t = 7.689	< 0.001
FBS (mg/dL)	186.45 ± 34.20	142.12 ± 22.80	t = 7.632	< 0.001
HbA1c (%)	9.15 ± 1.42	7.48 ± 0.95	t = 6.938	< 0.001
Blood Urea (mg/dL)	48.62 ± 14.35	24.18 ± 5.90	t = 11.189	< 0.001
Serum Creatinine (mg/dL)	1.78 ± 0.45	0.84 ± 0.16	t = 13.910	< 0.001
UACR (mg/g)	164.50 ± 42.10	18.30 ± 4.80	t = 24.368	< 0.001

Note. Data presented as Mean ± Standard Deviation or Frequency (Percentage). FBS = Fasting Blood Sugar; HbA1c = Glycosylated Hemoglobin; UACR = Urinary Albumin-to-Creatinine Ratio. Statistical significance set at $p < 0.05$.

Analysis of fundamental hematological parameters revealed a statistically significant reduction in hemoglobin levels among patients with diabetic nephropathy (11.10 ± 1.45 g/dL) compared to diabetic controls (12.00 ± 1.12 g/dL; p = 0.001). The Total Leukocyte Count (WBC) showed no statistically significant difference between the two groups (p = 0.484). However, a marked reduction in total platelet count was observed in the diabetic nephropathy cohort (266.00 ± 52.40 10^3 /µL) compared to controls (330.02 ± 61.15 10^3 /µL; $p < 0.001$) (Table 2).

Table 2: ***Comparison of Primary Hematological Parameters Between Groups***

Parameter	Diabetic Nephropathy Group (n=50)	Diabetic Control Group (n=50)	t-statistic	p-value
Hemoglobin (g/dL)	11.10 ± 1.45	12.00 ± 1.12	-3.473	0.001
Total WBC Count (10^3 /µL)	7.85 ± 1.62	7.64 ± 1.38	0.702	0.484
Absolute Neutrophil Count (10^3 /µL)	5.12 ± 1.15	4.18 ± 0.92	4.508	< 0.001

Absolute Lymphocyte Count (10^3/µL)	1.83 ± 0.42	2.61 ± 0.55	-7.975	< 0.001
Platelet Count (10^3/µL)	266.00 ± 52.40	330.02 ± 61.15	-5.624	< 0.001

Note. Data presented as Mean ± Standard Deviation. WBC = White Blood Cell. p < 0.05 indicates statistical significance.

The Neutrophil-to-Lymphocyte Ratio (NLR) was significantly elevated in the Diabetic Nephropathy Group (2.80 ± 0.65) compared to the Diabetic Control Group (1.60 ± 0.38; p < 0.001). Similarly, the Platelet-to-Lymphocyte Ratio (PLR) was significantly higher in nephropathy cases (103.30 ± 22.40) than in controls (92.00 ± 18.10; p = 0.007). Among the morphological platelet indices, Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) were both significantly increased in patients with diabetic nephropathy (11.20 ± 1.15 fL and 12.20 ± 1.30 fL, respectively) compared to diabetic controls (8.30 ± 0.85 fL and 11.00 ± 0.95 fL, respectively; p < 0.001 for both) (Table 3).

Table 3: ***Systemic Inflammatory Ratios and Morphological Platelet Indices***

Index / Parameter	Diabetic Nephropathy Group (n=50)	Diabetic Control Group (n=50)	t-statistic	p-value
Neutrophil-to-Lymphocyte Ratio (NLR)	2.80 ± 0.65	1.60 ± 0.38	11.308	< 0.001
Platelet-to-Lymphocyte Ratio (PLR)	103.30 ± 22.40	92.00 ± 18.10	2.774	0.007
Mean Platelet Volume (MPV, fL)	11.20 ± 1.15	8.30 ± 0.85	14.341	< 0.001
Platelet Distribution Width (PDW, fL)	12.20 ± 1.30	11.00 ± 0.95	5.275	< 0.001
Plateletcrit (PCT, %)	0.28 ± 0.05	0.27 ± 0.04	1.328	0.187

Note. Data presented as Mean ± Standard Deviation. fL = femtoliters. p < 0.05 indicates statistical significance.

NLR demonstrated strong, statistically significant positive correlations with Serum Creatinine (r = 0.684, p < 0.001) and UACR (r = 0.712, p < 0.001). MPV likewise exhibited strong positive correlations with Serum Creatinine (r = 0.642, p < 0.001) and UACR (r = 0.668, p < 0.001). Hemoglobin displayed a significant inverse correlation with Serum Creatinine (r = -0.415, p < 0.001) (Table 4).

Table 4: ***Correlation Matrix Between Hematological/Inflammatory Parameters and Renal Indices***

Parameter	Blood Urea (r)	Serum Creatinine (r)	UACR (r)
Hemoglobin (Hb)	-0.385**	-0.415**	-0.432**
Platelet Count	-0.320**	-0.354**	-0.378**
Neutrophil-to-Lymphocyte Ratio (NLR)	0.612**	0.684**	0.712**
Platelet-to-Lymphocyte Ratio (PLR)	0.284**	0.315**	0.342**
Mean Platelet Volume (MPV)	0.589**	0.642**	0.668**
Platelet Distribution Width (PDW)	0.365**	0.412**	0.445**

Note. r = Pearson correlation coefficient; UACR = Urinary Albumin-to-Creatinine Ratio. p < 0.001.

Elevated NLR (Adjusted Odds Ratio [aOR]} = 3.84, 95% CI [2.12, 6.95], p < 0.001) and elevated MPV (aOR} = 3.12, 95% CI [1.85, 5.26], p < 0.001) remained strong independent predictors of diabetic nephropathy after adjusting for age, duration of diabetes, and HbA1c. Higher PDW and lower Hemoglobin levels also retained independent predictive significance (Table 5).

Table 5: ***Multivariate Logistic Regression Analysis for Risk Factors of Diabetic Nephropathy***

Variable	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	Wald χ2	p-value
Hb (per 1 g/dL decrease)	1.65 (1.18, 2.31)	1.42 (1.05, 1.92)	5.12	0.024
NLR (per 1 unit increase)	4.52 (2.65, 7.71)	3.84 (2.12, 6.95)	18.64	< 0.001
PLR (per 10-unit increase)	1.32 (1.08, 1.61)	1.15 (0.94, 1.41)	1.82	0.177
MPV (per 1 fL increase)	3.68 (2.24, 6.05)	3.12 (1.85, 5.26)	15.42	< 0.001
PDW (per 1 fL increase)	2.14 (1.52, 3.01)	1.78 (1.21, 2.62)	8.25	0.004
HbA1c (per 1% increase)	2.41 (1.65, 3.52)	1.86 (1.24, 2.79)	8.89	0.003

Note. OR = Odds Ratio; CI = Confidence Interval; Hb = Hemoglobin; NLR = Neutrophil-to-Lymphocyte Ratio; PLR = Platelet-to-Lymphocyte Ratio; MPV = Mean Platelet Volume; PDW = Platelet Distribution Width; HbA1c = Glycosylated Hemoglobin. Model adjusted for age, gender, and duration of diabetes. p < 0.05 denotes statistical significance.

DISCUSSION

The findings of the present study demonstrate that elevated systemic inflammatory ratios—specifically the Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet-to-Lymphocyte Ratio (PLR)—alongside altered platelet morphological indices, particularly Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW), serve as distinct predictors of Diabetic Nephropathy (DN) in individuals with Type 2 Diabetes Mellitus (T2DM). These observed hematological alterations reinforce the fundamental role that chronic low-grade subclinical inflammation, oxidative stress, and structural microthrombotic activation play in driving renal endothelial microangiopathy.

In our cohort, NLR was significantly higher in diabetic nephropathy patients (2.80 ± 0.65) compared to diabetic controls without nephropathy (1.60 ± 0.38, $p < 0.001$), proving to be an independent predictor on multivariate logistic regression (aOR = 3.84). Furthermore, NLR showed strong positive correlations with both serum creatinine ($r = 0.684$) and urinary albumin-to-creatinine ratio ($r = 0.712$). This aligns closely with findings by Azab et al. [8], who demonstrated that elevated baseline NLR independently predicted progressive renal function deterioration over a three-year follow-up in diabetic patients.

A study by Kahraman et al. [11] reported significantly elevated NLR levels in patients with diabetic kidney disease compared to normoalbuminuric controls, establishing a direct linear increase in NLR with escalating grades of albuminuria. Mechanistically, activated neutrophils recruit to glomerular structures and release reactive oxygen species, myeloperoxidase, and proteolytic enzymes that damage the podocyte architecture and degrade the basement membrane, while stress-induced relative lymphopenia reflects a compromised adaptive regulatory system [7,8].

Platelet morphometric indices also displayed significant variance between groups. MPV (11.20 ± 1.15 fL vs 8.30 ± 0.85 fL) and PDW (12.20 ± 1.30 fL vs 11.00 ± 0.95 fL) were significantly elevated in the nephropathy cohort ($p < 0.001$ for both). On multivariate analysis, elevated MPV retained strong predictive power (\text{aOR} = 3.12). These findings are concordant with work by Buch et al. [9], who observed higher MPV and PDW values in diabetic patients suffering from microvascular complications, highlighting platelet enlargement as a key signal of heightened thrombotic potential.

Ulutas et al. [12] reported that elevated MPV was strongly correlated with microalbuminuria in T2DM patients, acting as a surrogate for subclinical renal vascular disease. Larger platelets contain denser alpha-granules, produce more thromboxane A2, and express surface adhesion receptors like glycoprotein IIb/IIIa at higher densities [9]. When lodged within damaged renal capillaries, these hyperreactive platelets trigger localized microthrombosis, endothelial detachment, and interstitial fibrosis [7,9].

Interesting secondary patterns were noted regarding the total platelet count and hemoglobin levels. A significant decrease in total platelet count was observed in the nephropathy group (266.00 ± 52.40 10^3/µL) compared to controls (330.02 ± 61.15 10^3/µL, $p < 0.001$). This mirrors findings reported by Papanas et al. [13], who described shortened platelet survival and accelerated peripheral destruction resulting from persistent microvascular endothelial damage and platelet consumption within damaged renal capillary networks. Hemoglobin levels were also significantly lower in the nephropathy cohort (11.10 ± 1.45 g/dL vs 12.00 ± 1.12 g/dL, $p = 0.001$). This finding corroborates the work of Thomas et al. [14], who established that early-stage diabetic nephropathy is often accompanied by unexplained normocytic normochromic anemia due to tubulointerstitial damage and impaired erythropoietin production prior to advanced eGFR decline.

Regarding PLR, our study demonstrated a significant elevation in nephropathy cases (103.30 ± 22.40 vs 92.00 ± 18.10, $p = 0.007$), although it lost independent significance after adjusting for baseline confounders. This pattern is consistent with results published by Li et al. [10], who noted that while PLR is elevated in diabetic kidney disease, NLR consistently offers superior diagnostic sensitivity and independent predictive performance due to the direct involvement of neutrophils in vascular injury. Moreover, a meta-analysis by Jiang et al. [15] confirmed that ratio-based indices, particularly NLR and MPV, are remarkably robust, cost-effective biomarkers for stratifying renal risk in T2DM across diverse patient demographics.

CONCLUSION

Calculated systemic inflammatory ratios, particularly the Neutrophil-to-Lymphocyte Ratio (NLR), and platelet morphological indices, such as Mean Platelet Volume (MPV), are significantly elevated in Type 2 Diabetes Mellitus patients with Diabetic Nephropathy. These inexpensive, universally available Complete Blood Count parameters strongly correlate with microalbuminuria and renal dysfunction. Consequently, NLR and MPV offer high clinical utility as accessible, non-invasive early predictive markers for diabetic renal microvascular damage.

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