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Original Research

Association of Complete Blood Count-Derived Inflammatory Indices with Cardiovascular Risk Factors in Patients with Metabolic Syndrome: A Cross-Sectional Observational Study.

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Abstract
Background: Metabolic syndrome is characterized by clustering of central obesity, dysglycemia, hypertension, and atherogenic dyslipidemia, with chronic low-grade inflammation contributing to cardiovascular risk. Complete blood count-derived indices offer inexpensive measures of systemic inflammatory activity. *Objectives:* To evaluate the association of neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII) with cardiovascular risk factors in patients with metabolic syndrome. *Methods:* This hospital-based cross-sectional observational study included 80 adults with metabolic syndrome at Malla Reddy Institute of Medical Sciences, Hyderabad, Telangana, India, from January to July 2025. Demographic, anthropometric, blood pressure, glycemic, lipid, and complete blood count parameters were recorded. NLR, PLR, MLR, and SII were calculated, and their associations with hypertension, diabetes mellitus, smoking, hypertriglyceridemia, low HDL-C, and cumulative risk-factor burden were analyzed. *Results:* Mean age was 52.8 ± 10.6 years; 56.3% were male. Mean NLR, PLR, MLR, and SII were 2.31 ± 0.86, 124.7 ± 38.9, 0.27 ± 0.10, and 612.8 ± 268.4 $\times 10^3/\mu L$, respectively. NLR and SII were significantly higher in participants with hypertension, diabetes, smoking, and low HDL-C. SII also increased with hypertriglyceridemia. NLR correlated positively with systolic blood pressure, fasting glucose, triglycerides, and waist circumference and inversely with HDL-C. All four indices increased progressively with greater cardiovascular risk-factor burden. *Conclusion:* CBC-derived inflammatory indices, particularly NLR and SII, were associated with clustering of cardiovascular risk factors in metabolic syndrome and can provide low-cost adjunctive information on systemic inflammatory burden.

Keywords: *Metabolic syndrome; neutrophil-to-lymphocyte ratio; platelet-to-lymphocyte ratio; monocyte-to-lymphocyte ratio; systemic immune-inflammation index; cardiovascular risk; inflammation.*

Introduction

Metabolic syndrome (MetS) represents a clinically important constellation of central adiposity, elevated blood pressure, impaired glucose regulation, hypertriglyceridemia, and reduced high-density lipoprotein cholesterol (HDL-C). Current harmonized criteria recognize the syndrome when at least three of five metabolic abnormalities are present, with population-specific waist circumference thresholds used for central obesity [1]. The syndrome is clinically relevant because its components interact rather than simply coexist, creating a metabolic environment that accelerates type 2 diabetes and atherosclerotic cardiovascular disease. Contemporary guidance therefore considers MetS a practical framework for recognizing individuals with clustered cardiometabolic risk [2]. A large systematic review and meta-analysis demonstrated that MetS is associated with approximately a twofold increase in cardiovascular outcomes, emphasizing the need for accessible markers that refine risk characterization within this already high-risk population [3].

Chronic low-grade inflammation is a central biological feature linking visceral adiposity, insulin resistance, dyslipidemia, hypertension, and vascular injury. Adipose tissue functions as an active endocrine and immune organ, releasing cytokines and adipokines that promote oxidative stress, endothelial dysfunction, and prothrombotic signaling [4,5]. Prospective data have shown that inflammatory biomarkers provide additional information beyond conventional metabolic traits. Ridker et al. observed a graded increase in C-reactive protein with increasing numbers of MetS components and demonstrated added prognostic information for cardiovascular events [6]. Similar findings from the Framingham Offspring Study supported a relationship between inflammatory burden, MetS, and future cardiovascular events [7]. These observations strengthen the rationale for evaluating inflammatory measures that can be obtained during routine clinical care.

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Complete blood count (CBC)-derived inflammatory indices have gained attention because they integrate different cellular components of innate immunity, adaptive immunity, and thrombosis without requiring specialized assays. The neutrophil-to-lymphocyte ratio (NLR) is a simple marker reflecting the balance between neutrophil-predominant inflammation and lymphocyte-mediated immune regulation and has been associated with cardiovascular disease severity and prognosis [8]. In patients with MetS, higher NLR has been linked with the presence and increasing severity of the syndrome [9], although other studies have reported weaker associations in obesity and MetS populations [10]. The platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII) extend this concept by incorporating platelet and monocyte responses. SII, calculated from platelet, neutrophil, and lymphocyte counts, has shown associations with cardiovascular disease [11], subclinical atherosclerosis in hypertension [12], and cardiovascular mortality [13]. Recent evidence also links higher SII with blood pressure and adverse lipid profiles [14].

Against this background, the present study was undertaken to examine whether inexpensive CBC-derived indices reflect the cardiovascular risk profile of patients with established MetS in a tertiary-care Indian setting. The objectives were to determine NLR, PLR, MLR, and SII in patients with MetS; compare these indices across major cardiovascular risk factors; assess their correlations with anthropometric, hemodynamic, glycemic, and lipid parameters; and evaluate whether inflammatory indices increase with cumulative cardiovascular risk-factor burden.

METHODOLOGY

Study design and setting: This hospital-based cross-sectional observational study was conducted at Malla Reddy Institute of Medical Sciences, Hyderabad, Telangana, India, from January 2025 to July 2025. Adult patients fulfilling diagnostic criteria for metabolic syndrome were evaluated during the study period. Eighty eligible participants were included by consecutive sampling.

Study participants and eligibility criteria: Adults aged 18 years or older with metabolic syndrome were eligible. Metabolic syndrome was defined using the harmonized criteria of the International Diabetes Federation, National Heart, Lung, and Blood Institute, American Heart Association, and collaborating organizations [1]. Patients with acute febrile illness or clinically evident infection, active inflammatory or autoimmune disease, malignancy, hematological disorders affecting leukocyte or platelet counts, recent major surgery or trauma, pregnancy, or current systemic corticosteroid or immunosuppressive therapy were excluded because these conditions could alter CBC-derived inflammatory indices.

Clinical and anthropometric assessment: Demographic characteristics, smoking status, history of hypertension and diabetes mellitus, current treatment, and relevant clinical information were recorded using a structured proforma. Body weight and height were measured by standard methods, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Waist circumference was measured at the midpoint between the lowest rib and iliac crest. Blood pressure was recorded after adequate rest with an appropriately sized cuff.

Definition of metabolic and cardiovascular risk factors: Metabolic syndrome was diagnosed when at least three of the following were present: increased waist circumference using South Asian cut-offs; triglycerides ≥150 mg/dL or treatment; HDL-C <40 mg/dL in men or <50 mg/dL in women or treatment; blood pressure ≥130/85 mmHg or antihypertensive treatment; and fasting plasma glucose ≥100 mg/dL or treatment for hyperglycemia [1,2]. For risk-factor analyses, hypertension and diabetes mellitus were based on established clinical diagnosis or ongoing treatment. Current smoking was obtained from participant history.

Laboratory assessment and inflammatory indices: Venous blood was collected under standard aseptic conditions. Complete blood count included total leukocyte, neutrophil, lymphocyte, monocyte, platelet, and hemoglobin measurements using an automated hematology analyzer. Fasting plasma glucose and lipid profile were measured by routine laboratory methods. NLR was calculated as neutrophils/lymphocytes, PLR as platelets/lymphocytes, MLR as monocytes/lymphocytes, and SII as platelet count × neutrophil count/lymphocyte count [8,11].

Statistical analysis: Continuous variables were expressed as mean ± standard deviation and categorical variables as number and percentage. Group comparisons were performed across cardiovascular risk-factor categories. Pearson correlation assessed relationships between inflammatory indices and continuous cardiometabolic variables. One-way analysis of variance compared indices across cumulative risk-factor categories. Multivariable models adjusted selected associations for age, sex, and BMI. A two-sided p-value <0.05 was considered statistically significant.

Ethical considerations: The study was conducted in accordance with institutional ethical requirements and the Declaration of Helsinki. Necessary Permissions were obtained before starting the study.

Results

A total of 80 patients with metabolic syndrome were included in the final analysis. The mean age of the study population was 52.8 ± 10.6 years, and 45 (56.3%) participants were male. The mean body mass index (BMI) was 29.1 ± 4.2 kg/m², while the mean waist circumference was 101.6 ± 9.8 cm. Hypertension was present in 55 (68.8%) participants, diabetes mellitus in 39 (48.8%), and current smoking in 18 (22.5%). Hypertriglyceridemia and low HDL-C were observed in 59 (73.8%) and 54 (67.5%) patients, respectively. The baseline demographic, clinical, and cardiovascular risk characteristics are presented in Table 1.

Table 1. Baseline demographic and cardiovascular risk characteristics of patients with metabolic syndrome (n = 80)

Characteristic	n (%) / Mean ± SD
Age, years	52.8 ± 10.6
Age group, years	
<40	10 (12.5)
40-49	22 (27.5)
50-59	27 (33.8)
≥60	21 (26.3)
Sex	
Male	45 (56.3)
Female	35 (43.8)
BMI, kg/m²	29.1 ± 4.2
Waist circumference, cm	101.6 ± 9.8
Systolic blood pressure, mmHg	138.7 ± 16.9
Diastolic blood pressure, mmHg	85.4 ± 10.2
Hypertension	55 (68.8)
Diabetes mellitus	39 (48.8)
Current smoking	18 (22.5)
Hypertriglyceridemia	59 (73.8)
Low HDL-C	54 (67.5)
Fasting plasma glucose, mg/dL	128.6 ± 38.5
Triglycerides, mg/dL	189.4 ± 67.2

HDL-C, mg/dL	39.2 ± 8.4
LDL-C, mg/dL	118.7 ± 32.6

Data are presented as mean ± standard deviation or number (percentage), as appropriate. BMI: body mass index; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol.

The mean total leukocyte count was 7.82 ± 1.76 ×10³/μL, with a mean absolute neutrophil count of 4.72 ± 1.39 ×10³/μL and lymphocyte count of 2.18 ± 0.63 ×10³/μL. Among the CBC-derived inflammatory indices, the mean NLR was 2.31 ± 0.86, PLR was 124.7 ± 38.9, and MLR was 0.27 ± 0.10. The mean SII was 612.8 ± 268.4 ×10³/μL. Hematological parameters and derived inflammatory indices are summarized in Table 2.

Table 2. Complete blood count parameters and derived inflammatory indices (n = 80)

Hematological parameter	Mean ± SD
Hemoglobin, g/dL	13.4 ± 1.6
Total leukocyte count, ×10³/μL	7.82 ± 1.76
Neutrophil count, ×10³/μL	4.72 ± 1.39
Lymphocyte count, ×10³/μL	2.18 ± 0.63
Monocyte count, ×10³/μL	0.56 ± 0.18
Platelet count, ×10³/μL	267.4 ± 61.8
Neutrophil-to-lymphocyte ratio (NLR)	2.31 ± 0.86
Platelet-to-lymphocyte ratio (PLR)	124.7 ± 38.9
Monocyte-to-lymphocyte ratio (MLR)	0.27 ± 0.10
Systemic immune-inflammation index (SII), ×10³/μL	612.8 ± 268.4

NLR = neutrophil count/lymphocyte count; PLR = platelet count/lymphocyte count; MLR = monocyte count/lymphocyte count; SII = platelet count × neutrophil count/lymphocyte count.

CBC-derived inflammatory indices were compared according to major cardiovascular risk factors. Patients with hypertension had significantly higher NLR (2.48 ± 0.88 vs. 1.94 ± 0.69; p = 0.007) and SII (663.5 ± 276.8 vs. 501.2 ± 213.5 ×10³/μL; p = 0.012) than patients without hypertension. Participants with diabetes mellitus showed higher NLR, MLR, and SII values than those without diabetes. Current smokers also demonstrated significantly elevated NLR and SII. Patients with hypertriglyceridemia had higher PLR and SII, whereas those with low HDL-C showed significantly higher NLR and SII. The comparative findings are presented in Table 3.

Table 3. Association of CBC-derived inflammatory indices with major cardiovascular risk factors

Cardiovascular risk factor	NLR, Mean ± SD	PLR, Mean ± SD	MLR, Mean ± SD	SII, ×10³/μL Mean ± SD
Hypertension				
Present (n = 55)	2.48 ± 0.88	130.1 ± 39.6	0.28 ± 0.10	663.5 ± 276.8
Absent (n = 25)	1.94 ± 0.69	112.8 ± 34.9	0.24 ± 0.09	501.2 ± 213.5
p-value	**0.007**	0.064	0.091	**0.012**
Diabetes mellitus				
Present (n = 39)	2.57 ± 0.89	132.6 ± 41.4	0.30 ± 0.11	681.4 ± 287.6
Absent (n = 41)	2.06 ± 0.76	117.2 ± 35.1	0.24 ± 0.08	547.5 ± 232.1
p-value	**0.008**	0.076	**0.009**	**0.024**
Current smoking				
Yes (n = 18)	2.73 ± 0.91	139.8 ± 42.3	0.30 ± 0.11	726.9 ± 301.8
No (n = 62)	2.19 ± 0.81	120.3 ± 37.2	0.26 ± 0.09	579.7 ± 251.0
p-value	**0.016**	0.059	0.107	**0.039**
Hypertriglyceridemia				
Present (n = 59)	2.40 ± 0.87	131.1 ± 39.6	0.28 ± 0.10	651.9 ± 270.7
Absent (n = 21)	2.04 ± 0.79	106.7 ± 30.7	0.25 ± 0.09	503.0 ± 228.6
p-value	0.101	**0.013**	0.232	**0.026**
Low HDL-C				
Present (n = 54)	2.46 ± 0.87	129.5 ± 39.4	0.28 ± 0.10	657.2 ± 274.1
Absent (n = 26)	2.00 ± 0.76	114.7 ± 36.2	0.25 ± 0.09	520.6 ± 226.7
p-value	**0.024**	0.113	0.184	**0.031**

Bold p-values indicate statistical significance at p < 0.05. HDL-C: high-density lipoprotein cholesterol; NLR: neutrophil-to-lymphocyte ratio; PLR: platelet-to-lymphocyte ratio; MLR: monocyte-to-lymphocyte ratio; SII: systemic immune-inflammation index.

Correlation analysis demonstrated significant relationships between inflammatory indices and several continuous cardiovascular risk parameters. NLR showed positive correlations with systolic blood pressure (r = 0.38, p < 0.001), fasting plasma glucose (r = 0.35, p = 0.002), triglycerides (r = 0.29, p = 0.009), and waist circumference (r = 0.27, p = 0.016). Conversely, NLR was inversely correlated with HDL-C (r = -0.31, p = 0.005). SII showed similar associations, with significant positive correlations with systolic blood pressure, fasting plasma glucose, triglycerides, waist circumference, and BMI, and an inverse association with HDL-C. PLR demonstrated a modest positive correlation with triglycerides, whereas MLR correlated significantly with systolic blood pressure and fasting plasma glucose. Detailed correlation coefficients are shown in Table 4.

Table 4. Correlation between CBC-derived inflammatory indices and cardiovascular risk parameters

Cardiovascular risk parameter	NLR, r (p)	PLR, r (p)	MLR, r (p)	SII, r (p)
BMI	**0.22 (0.049)**	0.16 (0.158)	0.18 (0.111)	**0.25 (0.027)**
Waist circumference	**0.27 (0.016)**	0.19 (0.093)	0.20 (0.075)	**0.31 (0.005)**
Systolic blood pressure	**0.38 (<0.001)**	0.21 (0.061)	**0.24 (0.033)**	**0.40 (<0.001)**
Fasting plasma glucose	**0.35 (0.002)**	0.18 (0.112)	**0.30 (0.007)**	**0.36 (0.001)**
Triglycerides	**0.29 (0.009)**	**0.28 (0.012)**	0.20 (0.074)	**0.33 (0.003)**
HDL-C	**-0.31 (0.005)**	-0.19 (0.092)	-0.21 (0.061)	**-0.34 (0.002)**
LDL-C	0.17 (0.132)	0.14 (0.216)	0.12 (0.289)	0.19 (0.091)

Bold values indicate statistically significant correlations. r: Pearson correlation coefficient; BMI: body mass index; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol.

To assess cumulative cardiovascular risk burden, participants were categorized according to the number of major cardiovascular risk factors present, including hypertension, diabetes mellitus, current smoking, hypertriglyceridemia, and low HDL-C. Twenty-two (27.5%) participants

had two or fewer risk factors, 34 (42.5%) had three risk factors, and 24 (30.0%) had four or more risk factors. A progressive increase in inflammatory indices was observed with increasing cardiovascular risk-factor burden. Mean NLR increased from 1.82 ± 0.60 among patients with ≤2 risk factors to 2.28 ± 0.72 among those with three risk factors and 2.80 ± 0.91 among those with ≥4 risk factors ($p < 0.001$). SII showed a corresponding increase from 462.6 ± 183.5 to 604.8 ± 224.7 and 766.9 ± 292.4 ×10³/μL, respectively ($p < 0.001$) (Table 5).

Table 5. CBC-derived inflammatory indices according to cardiovascular risk-factor burden

Inflammatory index	≤2 risk factors (n = 22)	3 risk factors (n = 34)	≥4 risk factors (n = 24)	p-value
NLR	1.82 ± 0.60	2.28 ± 0.72	2.80 ± 0.91	**<0.001**
PLR	107.8 ± 31.6	123.5 ± 34.7	142.0 ± 42.8	**0.008**
MLR	0.22 ± 0.07	0.27 ± 0.09	0.32 ± 0.11	**0.002**
SII, ×10³/μL	462.6 ± 183.5	604.8 ± 224.7	766.9 ± 292.4	**<0.001**

Comparison was performed using one-way analysis of variance. Bold p-values indicate statistical significance.

On multivariable analysis after adjustment for age, sex, and BMI, higher NLR remained significantly associated with hypertension ($\beta = 0.29$, $p = 0.011$) and fasting plasma glucose ($\beta = 0.25$, $p = 0.024$). SII remained significantly associated with systolic blood pressure ($\beta = 0.31$, $p = 0.006$) and triglyceride concentration ($\beta = 0.27$, $p = 0.017$). Overall, the findings demonstrated a graded relationship between systemic inflammatory activity and cardiovascular risk burden. Patients with multiple cardiovascular risk factors consistently exhibited higher NLR, PLR, MLR, and SII values, with the strongest and most consistent associations observed for NLR and SII.

Discussion

The present study identified a consistent relationship between CBC-derived inflammatory indices and cardiovascular risk burden among patients with metabolic syndrome. NLR and SII showed the most reproducible associations: both were higher in participants with hypertension, diabetes mellitus, smoking, and low HDL-C, while SII was also elevated in hypertriglyceridemia. In addition, NLR correlated positively with systolic blood pressure, fasting plasma glucose, triglycerides, and waist circumference and inversely with HDL-C. A graded rise in NLR, PLR, MLR, and SII across increasing numbers of cardiovascular risk factors further suggests that routine hematological indices can reflect the cumulative inflammatory milieu accompanying cardiometabolic clustering.

These findings are biologically plausible because MetS is characterized by chronic low-grade inflammation driven by visceral adiposity, insulin resistance, oxidative stress, endothelial dysfunction, and altered adipokine signaling [4,5]. Earlier prospective studies using C-reactive protein demonstrated that inflammatory activity rises with increasing MetS components and adds cardiovascular prognostic information beyond conventional risk factors [6,7]. CBC-derived indices provide a simpler approach because they can be calculated from routinely available cell counts without additional assays. NLR has already been associated with cardiovascular disease severity and adverse outcomes [8]. Buyukkaya et al. reported that NLR increased with the presence and severity of MetS [9], which is concordant with the progressive increase observed across risk-factor categories in the present study. However, Bahadir et al. found that NLR was less informative than leukocyte count and high-sensitivity C-reactive protein in obese individuals with and without MetS [10]. Differences in participant selection, diabetes status, obesity distribution, and inflammatory comorbidity could account for this variability.

The association observed for SII is particularly relevant because this index incorporates neutrophils, lymphocytes, and platelets and therefore captures inflammatory, immune, and thrombotic activity simultaneously. A systematic review and meta-analysis by Ye et al. demonstrated that higher SII was associated with increased cardiovascular risk across multiple disease categories [11]. Cirakoglu and Yilmaz found SII to be independently associated with increased carotid intima-media thickness in hypertensive patients, linking the index with subclinical atherosclerosis [12]. Population-level evidence has also connected elevated SII with cardiovascular mortality [13]. More recently, Aljuraiban et al. reported associations between higher SII, hypertension, and adverse lipid measures, closely paralleling the present findings regarding blood pressure and dyslipidemia [14].

PLR and MLR showed less uniform associations than NLR and SII, although both increased significantly with greater cumulative risk burden. This pattern suggests that composite indices incorporating multiple hematological pathways could be more informative when interpreted alongside established cardiometabolic factors rather than as isolated diagnostic markers. Because the study was cross-sectional, the observed relationships indicate association rather than temporal or causal direction. Nevertheless, the findings support further prospective evaluation of inexpensive CBC-derived indices as adjuncts to cardiovascular risk assessment in MetS, particularly in resource-constrained clinical settings.

LIMITATIONS

This study has several limitations. Its single-center cross-sectional design prevents assessment of temporal relationships and limits external generalizability. The sample size was modest, and inflammatory indices were measured at a single time point, so intra-individual biological variation was not captured. Residual confounding from medications, diet, physical activity, and unmeasured inflammatory conditions also remains possible. Larger multicenter prospective studies with repeated measurements are required to validate these associations.

Conclusion

In patients with metabolic syndrome, complete blood count-derived inflammatory indices were associated with several established cardiovascular risk factors and with the overall burden of cardiometabolic abnormalities. NLR and SII demonstrated the strongest and most consistent relationships, showing significant associations with hypertension, diabetes mellitus, smoking, dyslipidemia, blood pressure, glycemic status, and central adiposity. The progressive increase in inflammatory indices across higher risk-factor categories supports the presence of greater systemic inflammatory activity as cardiovascular risk factors accumulate. Because these indices are inexpensive and derived from routine hematological testing, they could serve as practical adjunctive markers for cardiovascular risk characterization. Prospective multicenter studies should establish reproducibility, clinically useful thresholds, and incremental predictive value beyond conventional risk assessment.

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