DEVELOPMENTAL MEDICO-LIFE-SCIENCES

ISSN (P): 3007-2786, (E): 3007-2794 DOI: https://doi.org/10.69750/dmls.03.08.0224

ORIGINAL RESEARCH ARTICLE Open Access

Association of Systemic Inflammatory and Metabolic Biomarkers with Major Adverse Cardiovascular Events in Patients with Type 2 Diabetes: A Prospective Cohort Study

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ABSTRACT

Background: Cardiovascular complications continue to occur in type 2 diabetes mellitus (T2DM) despite treatment of established risk factors. Biomarkers reflecting immune activation and metabolic disturbance may reveal clinically relevant residual risk.

Objective: To examine whether routinely obtainable inflammatory and metabolic indices are related to subsequent major adverse cardiovascular events (MACE) in adults with T2DM.

Methods: We prospectively followed 120 adults with established T2DM at Rashid Latif Khan University Medical College, Lahore, Pakistan, from June 2025 to April 2026. Baseline testing comprised high-sensitivity C-reactive protein (hs-CRP), neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), glycated hemoglobin (HbA1c), lipid measures, and the triglyceride-glucose (TyG) index. The prespecified composite outcome was the first occurrence of cardiovascular death, nonfatal myocardial infarction, or nonfatal ischemic stroke. Time-to-event associations were estimated with Cox regression, and predictive discrimination was explored using receiver-operating characteristic analysis.

Results: Twenty-four participants (20.0%) reached the composite endpoint. Event-positive participants had less favorable inflammatory and metabolic profiles, characterized by higher hs-CRP, NLR, SII, HbA1c, triglycerides, and TyG index and lower HDL-C. After covariate adjustment, hs-CRP (aHR 1.13 per 1 mg/L), SII (aHR 1.09 per 100 units), HbA1c (aHR 1.29 per 1%), and TyG index (aHR 1.71 per unit) remained associated with MACE. Their combined model yielded an AUC of 0.86, with 83.3% sensitivity and 78.1% specificity.

Conclusion: A combined inflammatory–metabolic profile identified patients with T2DM who experienced subsequent cardiovascular events. hs-CRP, SII, HbA1c, and TyG index may provide complementary information when used alongside standard cardiovascular risk assessment.

Keywords: Type 2 diabetes mellitus; MACE; systemic inflammation; hs-CRP; systemic immune-inflammation index; triglyceride-glucose index; cardiovascular risk.

Received: 10/05/2026
Revised: 26/07/2026
Accepted: 24/08/2026
Published: 31/08/2026

INTRODUCTION

Type 2 diabetes mellitus (T2DM) confers a substantial excess risk of atherosclerotic cardiovascular disease and its major clinical consequences, including myocardial infarction, ischemic stroke, heart failure, and cardiovascular death. This burden is only partially accounted for by conventional risk factors like hypertension, dyslipidemia, obesity, smoking, and chronic hyperglycemia. Current evidence, however, suggests a general pathobiological scenario where insulin resistance, oxidative damage, endothelial dysfunction, and chronic low-grade inflammation act in concert to promote vascular disease in T2DM [1–3].

Within this framework, inflammation is not merely a bystander but an active component of diabetic vascular injury. Chronic inflammatory signalling can disrupt

endothelial homeostasis, elevate oxidative stress, promote monocyte trafficking, promote plaque progression, and promote thrombogenic potential. High-sensitivity C-reactive protein (hs-CRP) has been extensively investigated in this context and has shown associations with cardiovascular outcomes beyond standard metabolic and clinical variables [4,5].

Even simple blood tests could also reflect clinically significant immune activity. Neutrophil-to-lymphocyte ratio (NLR) derived from a basic complete blood count (CBC) represents a measure of the relative importance of inflammation with neutrophilic predominance and of lymphocytes. A higher NLR has been linked to negative cardiovascular outcomes and mortality in individuals with T2DM, which may imply its potential as a cost-effective risk marker [6,7].

A more comprehensive cell-based measurement is the systemic immune-inflammation index (SII) which incorporates platelets, neutrophils, and lymphocytes. The SII may reflect multiple aspects of biology since these cells populations are involved in inflammation, thrombosis, and immune regulation. The SII is a simple index that has been shown to be related to cardiovascular events and mortality in diabetes populations [8–10].

Other indicators of metabolism offer a supplementary view. HbA1c is a marker of chronic glycemic exposure, and triglycerides and high-density lipoprotein cholesterol (HDL-C) are markers of crucial aspects of diabetic dyslipidemia. The triglyceride-glucose (TyG) index, which is derived from fasting glucose and triglyceride levels, has been shown to be an easy-to-access surrogate of insulin resistance and has been linked to cardiovascular outcomes in multiple high-risk cohorts [11,12].

There is a biological interaction between inflammatory and metabolic abnormalities. Insulin resistance increases oxidative and inflammatory signaling, and systemic inflammation decreases insulin function, vascular function, lipid handling. For this reason, a multiparametric approach taking into account hs-CRP, NLR, SII, HbA1c, lipid variables and TyG index may characterize the cardiovascular vulnerability more completely than any one of these parameters [9,11–13].

Evidence from prospective cohorts evaluating these routinely available measures in combination remains limited. It is also uncertain whether joint inflammatory–metabolic profiling adds useful discrimination beyond conventional clinical variables. We therefore investigated the relationship of hs-CRP, NLR, SII, HbA1c, triglycerides, HDL-C, and TyG index with incident MACE in adults with T2DM and assessed the discrimination of a combined biomarker model [1,6,8,11,13].

MATERIALS AND METHODS

We conducted a prospective cohort study at Rashid Latif Khan University Medical College, Lahore, Pakistan, between June 2025 and April 2026. Using consecutive non-probability recruitment, 120 adults aged 30–75 years with T2DM of at least one year's duration were enrolled and followed for incident MACE. We excluded patients with active infection; autoimmune or other chronic inflammatory disorders; malignancy; major surgery, acute myocardial infarction, or stroke within the preceding three months; pregnancy; chronic corticosteroid or immunosuppressive treatment; hematologic conditions capable of altering leukocyte or platelet counts; dialysis-dependent end-stage renal disease; incomplete baseline investigations; or unavailable follow-up data.

At baseline, trained study personnel documented age, sex, body mass index (BMI), duration of diabetes, smoking status, hypertension, dyslipidemia, previous cardiovascular disease, family history of cardiovascular disease, and current medications using a structured form. Blood pressure was recorded after at least five minutes of seated rest with a calibrated automated device. BMI was expressed as body weight in kilograms divided by height in meters squared.

Following an overnight fast of 8–12 hours, venous blood was collected for fasting plasma glucose, HbA1c, total cholesterol, LDL-C, HDL-C, triglycerides, creatinine, hs-CRP, and complete blood count. Measurements were performed with calibrated automated analyzers according to routine laboratory procedures. NLR was calculated as absolute neutrophil count divided by absolute lymphocyte count. SII was derived as platelet count × neutrophil count / lymphocyte count. TyG index was calculated as ln [fasting triglycerides (mg/dL) × fasting plasma glucose (mg/dL) / 2].

Participants were followed through scheduled assessments, review of hospital documentation, and telephone contact when necessary. The primary endpoint was time to first MACE, defined as cardiovascular death, nonfatal myocardial infarction, or nonfatal ischemic stroke. Potential events were verified using the available clinical record, including electrocardiographic findings, cardiac biomarkers, neuroimaging, discharge documentation, and mortality records, as applicable.

Analyses were performed in IBM SPSS Statistics version 26.0. Normally distributed continuous variables were summarized as mean ± standard deviation, whereas skewed variables were presented as median with interquartile range; categorical variables were expressed as frequencies and percentages. Group comparisons used the independent-samples t test, Mann–Whitney U test, chi-square test, or Fisher exact test according to variable type and distribution. Cox proportional-hazards models were used to estimate associations with MACE. Variables of clinical relevance and variables associated with outcome in univariable analyses were considered in adjusted models. Hazard ratios with 95% confidence intervals were reported. Receiver-operating characteristic curves were used to examine discrimination of individual biomarkers and combined models. Statistical significance was defined by a two-sided p-value < 0.05.

Ethical approval was granted by the Institutional Ethics Review Committee of Rashid Latif Khan University Medical College, Lahore, Pakistan (ERC No. RLKUMC/ERC/2025/061). Written informed consent was obtained before participation. Confidentiality was maintained throughout the study, and all procedures conformed to the ethical principles of the Declaration of Helsinki.

RESULTS

The analysis included 120 participants, comprising 66 men (55.0%) and 54 women (45.0%); mean age was 57.6 ± 9.1 years. During follow-up, 24 individuals (20.0%) experienced MACE, whereas 96 (80.0%) did not. Compared with the event-free group, participants with MACE were older, had lived with diabetes for longer, and more often had hypertension and previous atherosclerotic cardiovascular disease (ASCVD). The proportion of men and women was not significantly different between outcome groups (Table 1).

Outcome groups also differed across several laboratory measures. MACE was accompanied by higher HbA1c, fasting glucose, LDL-C, triglycerides, hs-CRP, NLR, SII, and TyG index, while HDL-C and eGFR were lower. The clearest separations between groups were observed for hs-CRP, SII, TyG index, and HbA1c (Table 2).

The 24 composite events consisted of 10 nonfatal myocardial infarctions (41.7%), 8 nonfatal ischemic strokes (33.3%), and 6 cardiovascular deaths (25.0%). Nonfatal myocardial infarction therefore accounted for the largest share of observed MACE (Table 3).

In unadjusted Cox models, older age, longer diabetes duration, previous ASCVD, higher HbA1c, hs-CRP, NLR, SII, and TyG index, and lower renal function were associated with MACE. Following adjustment for age, sex, diabetes duration, hypertension, smoking, previous ASCVD, LDL-C, renal function, and medication use, hs-CRP, SII, HbA1c, and TyG index retained independent associations with outcome. Sex did not show an independent association in the adjusted analysis (Table 4).

Discrimination of the individual markers ranged from moderate to good. The multibiomarker model incorporating hs-CRP, SII, HbA1c, and TyG index achieved an AUC of 0.86 (95% CI 0.78–0.93), with 83.3% sensitivity and 78.1% specificity (Table 5).

A model restricted to age, sex, hypertension, smoking, diabetes duration, previous ASCVD, LDL-C, and renal function produced an AUC of 0.74. Adding metabolic markers increased the AUC to 0.80, and subsequent inclusion of inflammatory markers increased it to 0.86. Overall, incident MACE was associated with a combined phenotype of stronger inflammatory activity, poorer glycemic control, and greater metabolic disturbance.

Table 1. Baseline Clinical Characteristics According to MACE Status

Variable	Overall (n=120)	No MACE (n=96)	MACE (n=24)	p-value
Age, years	57.6 ± 9.1	56.2 ± 8.7	63.0 ± 8.6	0.001
Male, n (%)	66 (55.0)	51 (53.1)	15 (62.5)	0.409
Female, n (%)	54 (45.0)	45 (46.9)	9 (37.5)	0.409
BMI, kg/m²	29.1 ± 4.3	28.8 ± 4.1	30.2 ± 4.7	0.151
Diabetes duration, years	9.3 ± 5.2	8.5 ± 4.8	12.5 ± 5.5	0.001
Hypertension, n (%)	79 (65.8)	58 (60.4)	21 (87.5)	0.013
Dyslipidemia, n (%)	73 (60.8)	55 (57.3)	18 (75.0)	0.113
Current smoking, n (%)	27 (22.5)	18 (18.8)	9 (37.5)	0.050
Previous ASCVD, n (%)	34 (28.3)	22 (22.9)	12 (50.0)	0.008
Statin use, n (%)	91 (75.8)	75 (78.1)	16 (66.7)	0.244
SGLT2 inhibitor use, n (%)	42 (35.0)	37 (38.5)	5 (20.8)	0.106

Table 2. Inflammatory and Metabolic Biomarkers According to MACE Status

Biomarker	No MACE (n=96)	MACE (n=24)	p-value
HbA1c, %	7.7 ± 1.1	8.7 ± 1.3	<0.001
Fasting glucose, mg/dL	153 ± 38	184 ± 46	0.001
LDL-C, mg/dL	94.1 ± 28.7	108.6 ± 32.4	0.033
HDL-C, mg/dL	44.2 ± 9.6	38.6 ± 8.4	0.010
Triglycerides, mg/dL	166 (132–209)	221 (174–279)	<0.001
hs-CRP, mg/L	2.5 (1.5–3.9)	5.1 (3.3–7.0)	<0.001
NLR	2.28 (1.76–2.90)	3.31 (2.54–4.11)	<0.001
SII, ×10⁹/L	512 (401–662)	748 (579–936)	<0.001
TyG index	8.93 ± 0.49	9.46 ± 0.56	<0.001
eGFR, mL/min/1.73 m²	79.3 ± 17.5	68.7 ± 19.1	0.010

Table 3. Distribution of Major Adverse Cardiovascular Events

Cardiovascular Event	n	Percentage of MACE
Nonfatal myocardial infarction	10	41.7%
Nonfatal ischemic stroke	8	33.3%
Cardiovascular death	6	25.0%
Total MACE	24	100%

Table 4. Cox Regression Analysis for Predictors of MACE

Predictor	Unadjusted HR (95% CI)	p-value	Adjusted HR (95% CI)	p-value
Age, per 10-year increase	1.58 (1.16–2.16)	0.004	1.42 (1.02–1.99)	0.039
Male sex	1.28 (0.57–2.88)	0.548	1.19 (0.50–2.82)	0.694
Diabetes duration, per 5 years	1.31 (1.08–1.59)	0.006	1.21 (1.01–1.46)	0.044
Previous ASCVD	2.41 (1.10–5.28)	0.028	1.86 (1.01–3.44)	0.047
HbA1c, per 1% increase	1.46 (1.16–1.83)	0.001	1.29 (1.03–1.62)	0.027
hs-CRP, per 1 mg/L increase	1.19 (1.09–1.30)	<0.001	1.13 (1.04–1.23)	0.004
NLR, per 1-unit increase	1.31 (1.09–1.57)	0.004	1.15 (0.94–1.41)	0.173
SII, per 100-unit increase	1.13 (1.06–1.21)	<0.001	1.09 (1.02–1.17)	0.011
TyG index, per 1-unit increase	2.01 (1.28–3.16)	0.002	1.71 (1.08–2.71)	0.022
eGFR, per 10-unit increase	0.82 (0.69–0.97)	0.020	0.88 (0.73–1.06)	0.177

Table 5. Predictive Performance of Biomarkers for MACE

Biomarker/Model	AUC	95% CI	Sensitivity	Specificity
hs-CRP	0.78	0.68–0.88	75.0%	72.9%
NLR	0.73	0.62–0.84	70.8%	69.8%
SII	0.80	0.70–0.89	79.2%	72.9%
HbA1c	0.75	0.65–0.85	70.8%	71.9%
TyG index	0.81	0.72–0.90	79.2%	74.0%
Combined biomarker model	0.86	0.78–0.93	83.3%	78.1%

DISCUSSION

The present cohort indicates that cardiovascular events in T2DM occurred in the setting of concurrent inflammatory and metabolic derangement rather than an isolated abnormality. Multivariable adjustment showed that hs-CRP, SII, HbA1c and TyG index all remained independently associated with MACE, suggesting that the immune-inflammatory and metabolic aspects provided complementary information for prognosis [1–3].

This hs-CRP finding is in line with the biological significance of low-grade vascular inflammation in diabetes [4]. Higher circulating hs-CRP has repeatedly been linked with increased cardiovascular risk across diabetic and other high-risk populations [6]. Mechanistically, chronic inflammatory activity can affect endothelial function, increase oxidative damage, increase recruitment of leukocytes, destabilize atherosclerotic plaques and increase the risk of thrombosis [5]. Thus, differences in hs-CRP between outcome groups are consistent with the presence of inflammatory risk.

NLR was higher in the group of participants who had MACE as a sign of increased inflammatory activity with neutrophils versus lymphocytes [7]. Previous studies have shown a positive association between increased NLR and myocardial infarction, stroke, cardiovascular death and cardiovascular composite outcomes [8]. After adjustment for traditional risk factors, a similar association has been reported in T2DM. However, when NLR was included in our adjusted model, its independent contribution weakened following the addition of hs-CRP and SII, suggesting that these inflammatory measures were overlapping [9].

SII was independently associated with the outcome and so added information to NLR in the multivariable model. As SII is a combination of platelet, neutrophil and lymphocyte counts, it could be a simultaneous representation of inflammatory activation, immune balance, and thrombotic potential [10]. In diabetic and prediabetic populations, there have been also studies that correlated increased SII levels with cardiovascular events and mortality [11]. Our results suggest that this inexpensive index needs to be further investigated as a risk marker based on routine haematology tests [12].

There were also significant differences between outcome groups on metabolic control. The participants with MACE had higher levels of HbA1c, fasting glucose, triglycerides, and TyG index, along with lower levels of HDL-C. Advanced glycation, oxidative stress, vascular inflammation, and endothelial dysfunction can be a consequence of persistent hyperglycemia [13]. Diabetic dyslipidemia also acts to promote atherogenesis by the production of triglyceride-rich lipoproteins, low HDL-C and an increase in the number of small dense LDL particles [14]. These mechanisms offer a plausible explanation as to why those with less favourable metabolic profiles have a higher number of events [15].

The association of the TyG index to MACE remained significant after adjustments. It is entirely based on fasting triglyceride and glucose levels, which is practical because it does not require any special assays to measure insulin resistance [16]. Sympathetic activation, dyslipidaemia, endothelial dysfunction, vascular inflammation, and prothrombotic signalling can be promoted by insulin resistance [17]. Previous studies have shown that higher TyG values are associated with myocardial infarction, ischemic stroke, cardiovascular death, and combined cardiovascular events [18]. Our data extend these observations to this cohort of adults with T2DM.

This increase in model discrimination with the addition of the inflammatory and metabolic variables have clinical significance. The four-marker model, which included hs-CRP, SII, HbA1c, and TyG index, had an AUC of 0.86, while the conventional clinical model had an AUC of 0.74. As metabolic dysfunction and inflammation are closely linked but not identical processes, their combined evaluation may be a more complete indicator of

cardiovascular risk than evaluation of any one domain alone [19]. Most of the components are already routinely assessed in the care of patients with diabetes and hs-CRP can be added by a relatively simple laboratory assay [6].

These biomarker results are not meant to replace existing prevention methods, but to support them. To date, diabetes management is still focused on the all-rounder treatment of blood pressure, lipids, glycemia, tobacco use, body weight, and kidney disease [1]. The therapies with proven cardiovascular benefit, such as SGLT2 inhibitors and GLP-1 receptor agonists, are also recommended in appropriate high-risk patients [20]. Biomarker profiling should thus only be viewed as an additional tool to the guideline-based clinical assessment [21].

There are a number of limitations that should be taken into account. This study was performed at one single centre with a relatively small patient population, so the results are only applicable to a limited extent. A total of only 24 MACE events were observed, limiting the use of multivariable modelling. Biomarkers were evaluated primarily at baseline with no evaluation of changes over time to assess inflammatory or metabolic status. The study was observational [3], and residual confounding is possible. The strengths are the inclusion of prospective follow-up, the measurement of prespecified cardiovascular endpoints, and the central measurement of multiple inflammatory and metabolic factors [2].

CONCLUSION

Among adults with T2DM, subsequent MACE was associated with a combined pattern of increased systemic inflammation and adverse metabolic status. hs-CRP, SII, HbA1c, and TyG index retained independent associations with events, and a model combining these measures discriminated outcomes better than conventional clinical variables alone. The tests are easily available and could be used as useful tools for normal risk assessment. Further studies in larger, multicenter cohorts and with longer follow-up are justified.

Conflict of Interest: The authors declare no conflicts of interest.

Funding: This research received no external funding.

Authors' Contributions: AA contributed to study conception, methodology, data interpretation, and manuscript drafting. YK contributed to data collection, analysis, and critical revision. MH contributed to investigation, literature review, and manuscript preparation. All authors reviewed and approved the final manuscript.

Acknowledgments: The authors acknowledge the clinical and laboratory staff involved in participant assessment and data collection.

Data Availability: The data are available from the corresponding author upon reasonable request.

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This Article May be cited as: Azhar A, Khan Y, Huzaifa M. Association of systemic inflammatory and metabolic biomarkers with major adverse cardiovascular events in patients with type 2 diabetes: a prospective cohort study. *Dev. Med. Life Sci.* 2026;3(8):4-9. doi:10.69750/dmls.03.08.0224.