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Research Article

Evaluation of Inflammatory Markers as Predictors of Cardiovascular Risk in Patients with Type 2 Diabetes Mellitus

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ABSTRACT
Background: Type 2 diabetes mellitus (T2DM) is associated with an increased risk of cardiovascular disease, with chronic low-grade inflammation playing an important role in the development and progression of atherosclerosis. Inflammatory markers such as high-sensitivity C-reactive protein (hs-CRP), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) may provide additional information for cardiovascular risk stratification.
Methods: This cross-sectional observational study included 110 patients with T2DM. Demographic, clinical, glycemic, lipid, and inflammatory parameters were assessed. Cardiovascular risk was estimated using conventional risk parameters, and patients were categorized according to risk. The associations of hs-CRP, NLR, and PLR with cardiovascular risk were evaluated using correlation and multivariate logistic regression analyses.
Results: The mean age was 57.6 ± 9.8 years, and 56.4% of patients were male. Hypertension and dyslipidemia were present in 58.2% and 51.8%, respectively. Overall, 36.4% of patients were classified as having high cardiovascular risk. High-risk patients had significantly higher hs-CRP (5.2 vs 2.6 mg/L), NLR (3.54 vs 2.39), and PLR (158.6 vs 119.8) compared with the low-to-moderate risk group (all $p<0.001$). hs-CRP showed the strongest correlation with cardiovascular risk ($r=0.52$), followed by NLR ($r=0.46$) and PLR ($r=0.38$; all $p<0.001$). On multivariate analysis, hs-CRP ≥3 mg/L (AOR 2.74; 95% CI: 1.21–6.20) and NLR ≥3.0 (AOR 2.48; 95% CI: 1.10–5.60) independently predicted high cardiovascular risk.
Conclusion: Increased systemic inflammation was significantly associated with cardiovascular risk in patients with T2DM. hs-CRP and NLR may serve as simple and clinically useful adjunctive markers for identifying patients at increased cardiovascular risk.

Keywords: Type 2 Diabetes Mellitus, Cardiovascular Risk, Inflammation, High-Sensitivity C-Reactive Protein, Neutrophil-To-Lymphocyte Ratio, Platelet-To-Lymphocyte Ratio.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most prevalent metabolic disorders worldwide and represents a major public health challenge because of its progressive nature and associated microvascular and macrovascular complications.[1,2] Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality among patients with T2DM. Individuals with diabetes have a substantially increased risk of coronary artery disease, stroke, peripheral arterial disease, and cardiovascular mortality compared with individuals without diabetes.[3,4] Early identification of patients at increased cardiovascular risk is therefore an important component of diabetes management.The increased cardiovascular risk associated with T2DM cannot be explained solely by conventional risk factors such as hypertension, dyslipidemia, obesity, smoking, and poor glycemic control.[5] Chronic low-grade systemic inflammation has emerged as an important mechanism linking insulin resistance, hyperglycemia, endothelial dysfunction, and atherosclerosis.[6] Persistent hyperglycemia promotes oxidative stress and the formation of advanced glycation end products, leading to endothelial injury and activation of inflammatory pathways. These processes contribute to the initiation, progression, and destabilization of atherosclerotic plaques.[7]
Several circulating inflammatory biomarkers have consequently been investigated as

potential indicators of cardiovascular risk. High-sensitivity C-reactive protein (hs-CRP) is one of the most extensively studied markers of systemic inflammation and has been associated with endothelial dysfunction, atherosclerotic burden, and cardiovascular events.[8] Increased hs-CRP concentrations are frequently observed in patients with T2DM and may provide additional cardiovascular risk information beyond conventional metabolic risk factors.[9]

Inflammatory indices derived from routine complete blood counts have also attracted increasing attention because they are inexpensive, readily available, and easily calculated in clinical practice. The neutrophil-to-lymphocyte ratio (NLR) reflects the balance between neutrophil-mediated inflammatory activity and lymphocyte-mediated immune regulation.[10] Similarly, the platelet-to-lymphocyte ratio (PLR) represents interactions between inflammation, platelet activation, and thrombosis. Elevated NLR and PLR have been associated with atherosclerosis, coronary artery disease severity, and adverse cardiovascular outcomes in several patient populations.[10,11]

The coexistence of chronic inflammation, dysglycemia, dyslipidemia, and other cardiovascular risk factors in patients with T2DM provides a rationale for evaluating inflammatory markers as potential tools for cardiovascular risk stratification.[12] Simple biomarkers such as hs-CRP, NLR, and PLR could potentially identify high-risk individuals who may benefit from more intensive cardiovascular risk-factor modification. However, the relationship between these inflammatory markers and overall cardiovascular risk among patients with T2DM requires further evaluation, particularly in routine clinical populations.

Therefore, the present study was undertaken to evaluate inflammatory markers as predictors of cardiovascular risk in patients with type 2 diabetes mellitus and to determine their association with glycemic control, conventional cardiovascular risk factors, and estimated cardiovascular risk.

MATERIALS AND METHODS

A total of 110 patients with type 2 diabetes mellitus fulfilling the eligibility criteria were included in the study.

Inclusion Criteria

Patients were included if they:

1. Were aged ≥18 years.
2. Had an established diagnosis of type 2 diabetes mellitus according to accepted diagnostic criteria.
3. Were willing to participate and provided written informed consent.

Exclusion Criteria

Patients with the following conditions were excluded:

1. Acute infection or febrile illness at the time of evaluation.
2. Chronic inflammatory or autoimmune disorders.
3. Active malignancy.
4. Recent major surgery, trauma, or acute cardiovascular event.
5. Severe hepatic or renal dysfunction likely to significantly alter inflammatory markers.
6. Current treatment with systemic corticosteroids or other immunosuppressive medications.
7. Pregnancy.

Data Collection

Detailed demographic and clinical information was recorded using a structured study proforma. Data included age, sex, duration of diabetes, smoking status, alcohol consumption, history of hypertension, dyslipidemia, family history of cardiovascular disease, current medications, and other relevant comorbidities.

A detailed physical examination was performed. Height and weight were measured using standardized methods, and body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared (kg/m²). Blood pressure was measured after an appropriate period of rest using a calibrated sphygmomanometer.

Laboratory Investigations

Venous blood samples were collected under aseptic precautions after an overnight fast. Laboratory evaluation included:

- Complete blood count with differential leukocyte count
- Fasting blood glucose
- Postprandial blood glucose
- Glycated hemoglobin (HbA1c)
- Serum creatinine
- Total cholesterol
- Triglycerides
- High-density lipoprotein cholesterol (HDL-C)
- Low-density lipoprotein cholesterol (LDL-C)
- High-sensitivity C-reactive protein (hs-CRP)

All biochemical parameters were measured using standardized laboratory methods.

Assessment of Inflammatory Markers

The following inflammatory markers were evaluated:

Neutrophil-to-lymphocyte ratio (NLR) was calculated as:

NLR = Absolute neutrophil count / Absolute lymphocyte count

Platelet-to-lymphocyte ratio (PLR) was calculated as:

PLR = Platelet count / Absolute lymphocyte count

High-sensitivity C-reactive protein (hs-CRP) was measured using the standardized assay available in the institutional laboratory.

Assessment of Cardiovascular Risk

Cardiovascular risk was assessed using demographic characteristics, smoking status, blood pressure, lipid profile, glycemic parameters, and other conventional cardiovascular risk factors. An appropriate validated cardiovascular risk prediction score was used to estimate the cardiovascular risk of each eligible participant. Patients were subsequently categorized into cardiovascular risk groups according to the predefined categories of the selected risk model.

The relationship between hs-CRP, NLR, and PLR and estimated cardiovascular risk was evaluated. Their associations with age, duration of diabetes, BMI, blood pressure, HbA1c, and lipid parameters were also assessed.

Outcome Measures

The primary outcome was the association of inflammatory markers (hs-CRP, NLR, and PLR) with cardiovascular risk among patients with T2DM.

Secondary outcomes included the correlation of inflammatory markers with glycemic control, lipid parameters, duration of diabetes, obesity, hypertension, and other conventional cardiovascular risk factors and the identification of independent predictors of high cardiovascular risk.

Statistical Analysis

Data were entered into a spreadsheet and analyzed using SPSS.21statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range according to their distribution, whereas categorical variables were presented as frequencies and percentages.

The normality of continuous variables was assessed using the Shapiro–Wilk test. Differences in normally distributed continuous variables between groups were analyzed using the independent-samples Student's t-test, while the Mann–Whitney U test was used for non-normally distributed variables. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate.Correlations between inflammatory markers and cardiovascular/metabolic parameters were evaluated using Pearson or Spearman correlation coefficients, depending on data distribution. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the ability of hs-CRP, NLR, and PLR to discriminate patients with high cardiovascular risk. The area under the curve (AUC), optimal cut-off values, sensitivity, and specificity were calculated.Variables demonstrating clinically relevant or statistically significant associations were entered into multivariate logistic regression analysis to identify independent predictors of high cardiovascular risk. Adjusted odds ratios (AORs) with 95% confidence intervals were reported. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 110 patients with type 2 diabetes mellitus were included in the study. The mean age of the study population was 57.6 ± 9.8 years, and 62 (56.4%) patients were male. The mean duration of diabetes was 9.2 ± 5.7 years. Hypertension was present in 64 (58.2%) patients, dyslipidemia in 57 (51.8%), and 25 (22.7%) were current smokers. The mean BMI was 27.4 ± 4.1 kg/m². The mean HbA1c was 8.3 ± 1.6%, indicating suboptimal glycemic control in a substantial proportion of patients (Table 1).

Table 1. Baseline demographic and clinical characteristics of the study population (N=110)

Variable	Value
Age (years), mean ± SD	57.6 ± 9.8
Age ≥60 years, n (%)	48 (43.6)
Male sex, n (%)	62 (56.4)
Female sex, n (%)	48 (43.6)

Duration of diabetes (years), mean ± SD	9.2 ± 5.7
Diabetes duration ≥10 years, n (%)	49 (44.5)
BMI (kg/m²), mean ± SD	27.4 ± 4.1
Overweight/obesity, n (%)	73 (66.4)
Hypertension, n (%)	64 (58.2)
Dyslipidemia, n (%)	57 (51.8)
Current smoking, n (%)	25 (22.7)
Family history of CVD, n (%)	31 (28.2)
Systolic BP (mmHg), mean ± SD	136.8 ± 16.7
Diastolic BP (mmHg), mean ± SD	83.6 ± 9.5
HbA1c (%), mean ± SD	8.3 ± 1.6

The mean fasting blood glucose was 156.8 ± 48.5 mg/dL, while mean LDL-C and triglyceride levels were 116.4 ± 35.7 mg/dL and 173.5 ± 71.6 mg/dL, respectively. Regarding inflammatory parameters, median hs-CRP was 3.4 mg/L (IQR: 1.8–5.9), median NLR was 2.76 (IQR: 2.05–3.68), and median PLR was 132.5 (IQR: 103.4–168.7) (Table 2).

Table 2. Biochemical and inflammatory parameters among study participants (N=110)

Parameter	**Value**
Fasting blood glucose (mg/dL), mean ± SD	156.8 ± 48.5
Postprandial blood glucose (mg/dL), mean ± SD	228.6 ± 67.4
HbA1c (%), mean ± SD	8.3 ± 1.6
Total cholesterol (mg/dL), mean ± SD	194.7 ± 42.6
LDL-C (mg/dL), mean ± SD	116.4 ± 35.7
HDL-C (mg/dL), mean ± SD	42.3 ± 9.8
Triglycerides (mg/dL), mean ± SD	173.5 ± 71.6
Serum creatinine (mg/dL), mean ± SD	1.02 ± 0.24
Total leukocyte count (/µL), mean ± SD	7,840 ± 1,920
hs-CRP (mg/L), median (IQR)	3.4 (1.8–5.9)
NLR, median (IQR)	2.76 (2.05–3.68)
PLR, median (IQR)	132.5 (103.4–168.7)

Based on estimated cardiovascular risk, 31 (28.2%) patients were classified as low risk, 39 (35.5%) as moderate risk, and 40 (36.4%) as high risk. Thus, more than one-third of the study population belonged to the high cardiovascular-risk category (Table 3).

Table 3. Distribution of patients according to estimated cardiovascular risk (N=110)

Cardiovascular risk category	**n (%)**
Low risk	31 (28.2)
Moderate risk	39 (35.5)
High risk	40 (36.4)
Total	**110 (100.0)**

Patients with high cardiovascular risk were older and had a longer duration of diabetes than those in the low-to-moderate risk group. Hypertension, dyslipidemia, and smoking were also significantly more frequent among high-risk patients. Mean HbA1c was significantly higher in the high-risk group (9.0 ± 1.5% vs 7.9 ± 1.5%; p<0.001). Importantly, all three inflammatory markers were significantly elevated among patients with high cardiovascular risk. Median hs-CRP was 5.2 mg/L compared with 2.6 mg/L, median NLR was 3.54 compared with 2.39, and median PLR was 158.6 compared with 119.8 in the high-risk and low-to-moderate risk groups, respectively (all p<0.001) (Table 4).

Table 4. Comparison of clinical, metabolic, and inflammatory parameters according to cardiovascular risk

Variable	**Low–moderate risk (n=70)**	**High risk (n=40)**	**p-value**
Age (years), mean ± SD	53.9 ± 8.4	64.1 ± 8.6	<0.001

Male sex, n (%)	36 (51.4)	26 (65.0)	0.17
Diabetes duration (years), mean ± SD	7.5 ± 4.7	12.2 ± 6.1	<0.001
BMI (kg/m²), mean ± SD	26.8 ± 3.8	28.5 ± 4.4	0.04
Hypertension, n (%)	34 (48.6)	30 (75.0)	0.007
Dyslipidemia, n (%)	29 (41.4)	28 (70.0)	0.004
Current smoking, n (%)	10 (14.3)	15 (37.5)	0.005
HbA1c (%), mean ± SD	7.9 ± 1.5	9.0 ± 1.5	<0.001
LDL-C (mg/dL), mean ± SD	108.6 ± 31.8	130.1 ± 38.1	0.002
hs-CRP (mg/L), median (IQR)	2.6 (1.4–4.0)	5.2 (3.5–7.4)	<0.001
NLR, median (IQR)	2.39 (1.84–2.97)	3.54 (2.79–4.45)	<0.001
PLR, median (IQR)	119.8 (95.2–145.6)	158.6 (128.4–191.7)	<0.001

Correlation analysis demonstrated that hs-CRP, NLR, and PLR were positively correlated with estimated cardiovascular risk. The strongest correlation was observed for hs-CRP (r=0.52, p<0.001), followed by NLR (r=0.46, p<0.001) and PLR (r=0.38, p<0.001). HbA1c was positively correlated with hs-CRP (r=0.41, p<0.001) and NLR (r=0.34, p<0.001), suggesting an association between poorer glycemic control and increased systemic inflammation (Table 5).

Table 5. Correlation of inflammatory markers with cardiovascular and metabolic parameters

Parameter	hs-CRP, r (p-value)	NLR, r (p-value)	PLR, r (p-value)
Cardiovascular risk	0.52 (<0.001)	0.46 (<0.001)	0.38 (<0.001)
Age	0.31 (0.001)	0.29 (0.002)	0.20 (0.04)
Duration of diabetes	0.37 (<0.001)	0.32 (0.001)	0.25 (0.009)
BMI	0.28 (0.003)	0.19 (0.05)	0.14 (0.15)
HbA1c	0.41 (<0.001)	0.34 (<0.001)	0.26 (0.006)
LDL-C	0.30 (0.001)	0.24 (0.01)	0.21 (0.03)

On multivariate logistic regression analysis, after adjustment for potential confounding factors, hs-CRP ≥3 mg/L (AOR 2.74; 95% CI: 1.21–6.20; p=0.016) and NLR ≥3.0 (AOR 2.48; 95% CI: 1.10–5.60; p=0.029) remained independent inflammatory predictors of high cardiovascular risk. Age ≥60 years, hypertension, and HbA1c ≥8% were also independently associated with high cardiovascular risk. PLR ≥150 showed an increased odds of high cardiovascular risk but did not retain statistical significance after adjustment (Table 6).

Table 6. Multivariate logistic regression analysis of predictors of high cardiovascular risk

Predictor	Adjusted OR	95% CI	p-value
Age ≥60 years	3.12	1.34–7.26	0.008
Hypertension	2.39	1.04–5.50	0.040
HbA1c ≥8%	2.56	1.12–5.85	0.026
hs-CRP ≥3 mg/L	2.74	1.21–6.20	0.016
NLR ≥3.0	2.48	1.10–5.60	0.029
PLR ≥150	1.69	0.75–3.82	0.207

Overall, patients with higher cardiovascular risk demonstrated a greater inflammatory burden, poorer glycemic control, and a higher prevalence of conventional cardiovascular risk factors. Among the inflammatory biomarkers evaluated, hs-CRP showed the strongest relationship with cardiovascular risk, followed by NLR. Both hs-CRP and NLR remained independently associated with high cardiovascular risk after adjustment for other relevant clinical and metabolic factors.

DISCUSSION

The present study evaluated the association of inflammatory markers with cardiovascular risk among 110 patients with type 2 diabetes mellitus (T2DM). The principal finding was that patients with high cardiovascular risk had

significantly higher levels of hs-CRP, neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) compared with those in the low-to-moderate risk group. In addition, inflammatory markers demonstrated significant positive correlations with cardiovascular risk and several adverse metabolic parameters. On multivariate analysis, elevated hs-CRP and NLR remained independent predictors of high cardiovascular risk, suggesting that systemic inflammation may provide additional information for cardiovascular risk stratification in patients with T2DM.

In the present study, the mean age was 57.6 ± 9.8 years, 56.4% of patients were male, and the mean duration of diabetes was 9.2 ± 5.7 years. Hypertension and dyslipidemia were present in 58.2% and 51.8% of patients, respectively, while 66.4% were overweight or obese. These findings demonstrate the clustering of conventional cardiovascular risk factors commonly observed in T2DM. The Emerging Risk Factors Collaboration demonstrated that diabetes is independently associated with approximately a two-fold increased risk of coronary heart disease, ischemic stroke, and other vascular deaths, emphasizing the substantial cardiovascular burden associated with diabetes.[13]

Chronic low-grade inflammation has increasingly been recognized as a central component of both T2DM and atherosclerotic cardiovascular disease. Donath and Shoelson described the important contribution of inflammatory pathways to insulin resistance, β-cell dysfunction, and the development of diabetes-related complications.[14] Similarly, Libby et al. emphasized that inflammation participates at virtually every stage of atherosclerosis, from endothelial dysfunction and plaque formation to plaque destabilization and acute cardiovascular events.[15] The increased inflammatory burden observed among high-risk patients in the present study is therefore biologically consistent with current understanding of diabetic atherosclerosis.

The median hs-CRP level in the present study was 3.4 mg/L and was significantly higher among patients with high cardiovascular risk than among those with low-to-moderate risk (5.2 vs 2.6 mg/L; $p<0.001$). Furthermore, hs-CRP showed the strongest correlation with cardiovascular risk ($r=0.52$; $p<0.001$). These findings are supported by Ridker et al., who demonstrated that CRP provides important information regarding future cardiovascular risk and may complement conventional lipid-based risk assessment.[16] Kaptoge et al., in a large collaborative meta-analysis, also demonstrated continuous associations between circulating CRP concentrations and the subsequent risk of coronary heart disease, ischemic stroke, and vascular mortality.[17]

The present study additionally demonstrated a significant positive correlation between hs-CRP and HbA1c ($r=0.41$; $p<0.001$). This relationship suggests that poor glycemic control may contribute to enhanced systemic inflammatory activity. Hyperglycemia induces oxidative stress, endothelial dysfunction, advanced glycation end-product formation, and activation of pro-inflammatory signaling pathways, all of which may accelerate atherosclerosis.[18] Previous studies have similarly reported higher inflammatory marker concentrations among individuals with impaired glucose metabolism and poorly controlled diabetes, supporting a close interaction between metabolic dysregulation and inflammation.[19]

NLR was another important marker in the present study. Median NLR was significantly higher among patients at high cardiovascular risk than among patients with low-to-moderate risk (3.54 vs 2.39; $p<0.001$). NLR also showed a significant positive correlation with cardiovascular risk ($r=0.46$; $p<0.001$) and HbA1c ($r=0.34$; $p<0.001$). NLR integrates two components of the inflammatory response: neutrophilia, reflecting nonspecific inflammatory activation, and relative lymphopenia, which may represent physiological stress and altered immune regulation. Consequently, NLR has emerged as a readily available marker of systemic inflammation and cardiovascular risk.[20]

Balta et al. highlighted NLR as a simple and inexpensive inflammatory marker associated with several cardiovascular diseases and cardiovascular risk factors.[21] Similarly, Verdoia et al. reported an association between higher NLR and the presence and severity of coronary artery disease among patients undergoing coronary angiography.[22] These observations are consistent with the present findings and suggest that NLR may be particularly useful in clinical settings where more specialized inflammatory biomarkers are not routinely available.

PLR was also significantly elevated among high-risk patients in the present study (158.6 vs 119.8; $p<0.001$) and demonstrated a positive correlation with cardiovascular risk ($r=0.38$; $p<0.001$). PLR reflects both platelet activation and lymphocyte-mediated inflammatory

responses and may therefore represent the interaction between inflammation and thrombosis. Elevated PLR has previously been associated with the severity of atherosclerosis and adverse cardiovascular outcomes.[23] However, although PLR was significantly associated with cardiovascular risk on unadjusted analyses in the present study, PLR ≥150 did not remain an independent predictor after multivariate adjustment (AOR 1.69; p=0.207). This finding suggests that PLR may reflect the overall inflammatory and metabolic burden but may have less independent predictive value than hs-CRP and NLR.

A notable observation was the progressive relationship between inflammatory markers and other adverse metabolic characteristics. hs-CRP, NLR, and PLR were positively correlated with duration of diabetes, HbA1c, and LDL-C, although the strength of these associations varied. Longer exposure to hyperglycemia, insulin resistance, oxidative stress, and dyslipidemia may result in sustained endothelial and inflammatory activation. Low-grade vascular inflammation subsequently promotes lipid accumulation, plaque progression, and a prothrombotic state, providing a mechanistic explanation for the increased cardiovascular risk associated with longstanding T2DM.[24]

Multivariate analysis provided further support for the clinical significance of inflammation. hs-CRP ≥3 mg/L was independently associated with high cardiovascular risk (AOR 2.74; 95% CI: 1.21–6.20), while NLR ≥3.0 was associated with approximately 2.5-fold higher odds of high cardiovascular risk (AOR 2.48; 95% CI: 1.10–5.60). Age ≥60 years, hypertension, and HbA1c ≥8% were also independent predictors. These findings indicate that inflammatory markers should not be viewed in isolation but rather as potential complementary parameters alongside established cardiovascular risk factors.

The clinical importance of inflammation in cardiovascular disease has also been strengthened by therapeutic studies. The CANTOS trial demonstrated that targeting inflammation with canakinumab reduced recurrent cardiovascular events independently of lipid lowering, providing direct evidence for a causal role of inflammation in atherosclerotic disease.[25] More recently, studies involving colchicine have further demonstrated that modulation of inflammatory pathways can reduce cardiovascular events among appropriately selected patients with established coronary disease.[26] These findings reinforce the concept that inflammation is not merely a marker but an important contributor to cardiovascular disease pathogenesis.

From a clinical perspective, hs-CRP, NLR, and PLR have the advantage of being relatively accessible biomarkers. NLR and PLR can be calculated directly from routine complete blood counts without additional laboratory expenditure, while hs-CRP is widely available in clinical laboratories. Their incorporation alongside conventional cardiovascular risk assessment may potentially help identify patients with T2DM who have a greater underlying inflammatory burden. However, inflammatory biomarkers should complement rather than replace established risk factors and validated cardiovascular risk-prediction models.

CONCLUSION

The present study demonstrated that increased systemic inflammation was significantly associated with higher cardiovascular risk in patients with type 2 diabetes mellitus. Elevated hs-CRP and NLR were independent predictors of high cardiovascular risk, with hs-CRP showing the strongest association. These readily available inflammatory markers may serve as useful adjuncts to conventional cardiovascular risk assessment in patients with T2DM.

Limitations

The study was limited by its single-centre, cross-sectional design and relatively small sample size of 110 patients. The lack of longitudinal follow-up prevented assessment of the ability of inflammatory markers to predict actual future cardiovascular events. Additionally, residual confounding from medications, lifestyle factors, and other inflammatory conditions could not be completely excluded.

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