Silent Predictors of Chronic Total Occlusion in Patients with Stable Angina Pectoris: The Role of Inflammatory and Metabolic Biomarkers

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Abstract

Aim: Atherosclerosis and inflammation play central roles in the pathogenesis of coronary artery disease (CAD). Early identification of chronic total occlusion (CTO) in patients presenting with stable angina pectoris is clinically important for guiding treatment strategies. Therefore, evaluating the diagnostic utility of novel inflammatory and metabolic biomarkers has gained increasing relevance. This study aimed to assess the predictive value of the systemic inflammatory index (SII), uric acid/HDL ratio (UHR), monocyte/HDL ratio (MHR), and triglyceride–glucose (TyG) index for detecting the presence of CTO.

Methods: In this retrospective study, 96 patients who underwent coronary angiography between January 1, 2024 and April 1, 2025 at Mersin City Training and Research Hospital were included. Thirty-six patients with at least one coronary artery showing 100% occlusion constituted the CTO group, while 60 patients without CTO formed the control group. Demographic characteristics and laboratory parameters were recorded, and SII, UHR, MHR, and TyG values were calculated and compared between groups.

Results: There were no significant differences between the groups regarding SII ($p = 0.118$), UHR ($p = 0.887$), or TyG ($p = 0.333$). However, MHR levels were significantly higher in the CTO group ($p < 0.001$). ROC analysis identified an MHR cutoff value of > 8.7 as a meaningful threshold for predicting CTO.

Conclusions: Among the biomarkers evaluated, only MHR demonstrated a significant association with the presence of CTO. Reflecting increased monocyte activity and reduced anti-inflammatory HDL capacity, MHR appears consistent with the chronic inflammatory nature of CTO. Its simplicity, low cost, and availability from routine laboratory tests further support MHR as a practical biomarker for assessing CTO risk in clinical practice.

Keywords: Atherosclerosis; stable angina; coronary artery disease; chronic total occlusion; inflammation; monocyte/HDL ratio; biomarkers

1. Introduction

Stable angina pectoris is an ischemic syndrome that may arise from atherosclerotic or structural abnormalities of the epicardial coronary arteries, functional endothelial dysfunction, or structural disorders within the microvascular system.[1] Inflammation and atherosclerotic plaque formation constitute the fundamental pathological mechanisms of coronary artery disease (CAD).

Chronic total occlusion (CTO) is a complex form of CAD characterized by complete obstruction of a coronary artery for at least three months, usually accompanied by the development of collateral circulation.[2] CTO is detected in approximately 20% of patients undergoing diagnostic coronary angiography, and this rate increases up to 40% among patients with heart failure or diabetes mellitus.[3]

To better elucidate the inflammatory mechanisms underlying CAD, several novel biomarkers such as the systemic inflammatory index (SII), monocyte-to-HDL cholesterol ratio (MHR), uric acid-to-HDL cholesterol ratio (UHR), and triglyceride–glucose index (TyG) have been investigated. Elevated SII has been associated with increased CAD severity and a higher risk of major adverse cardiovascular events (MACE).[4]

Monocytes constitute up to 10% of circulating white blood cells and play an active role in inflammation. HDL cholesterol, through its anti-inflammatory effects on monocytes, may reduce inflammation in atherosclerosis. Therefore, the monocyte-to-HDL cholesterol ratio (MHR) has been proposed as a novel indicator of inflammatory activity. [5,6]

The uric acid-to-HDL cholesterol ratio (UHR) has gained attention as a biomarker reflecting the interaction between pro-inflammatory and anti-inflammatory components.[7]

The TyG index is recognized as a reliable surrogate marker of insulin resistance and is closely associated with the pathophysiological basis of cardiovascular events.[8]

In this exploratory, hypothesis-generating study, we aimed to investigate the potential predictive value of inflammatory and metabolic biomarkers for the presence of chronic total occlusion in patients with stable angina pectoris.

2. Materials and Methods

### Study Design and Objective

This study aimed to determine whether novel inflammatory and metabolic biomarkers can serve as independent predictors of CTO in patients presenting with stable angina pectoris. Given the retrospective design, this study employed an exploratory analysis to generate hypotheses regarding the association between inflammatory and metabolic biomarkers and the presence of chronic total occlusion.

Patients who underwent coronary angiography (CAG) for stable angina pectoris between January 1, 2024, and April 1, 2025, at Mersin City Training and Research Hospital were retrospectively reviewed. Data from patients with CTO in at least one coronary artery and those with significant stenosis or non-critical stenosis associated with ischemia confirmed by functional imaging were analyzed retrospectively using hospital information systems, patient files, and imaging archives [Figure 1].

Figure 1

Study Flowchart
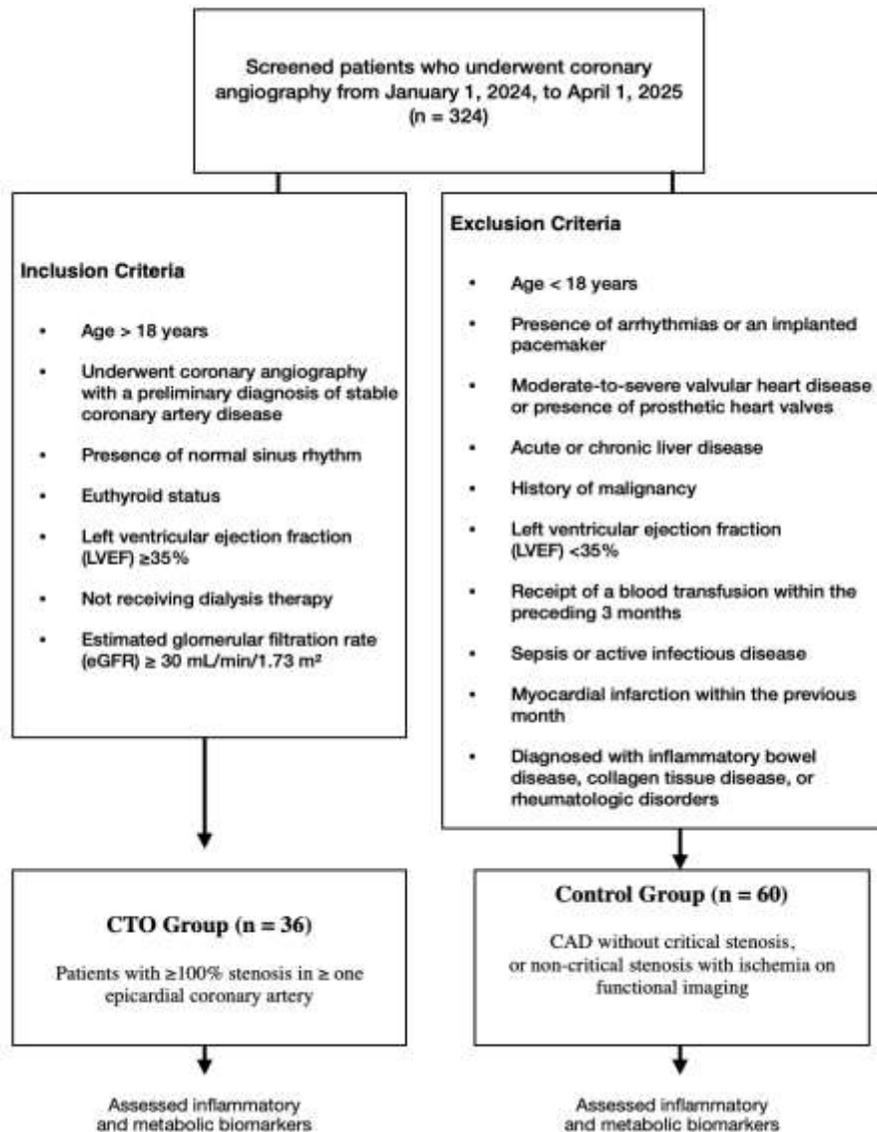
CAD: Coronary Artery Disease; CTO: Chronic Total Occlusion; eGFR: Estimated Glomerular Filtration Rate; LVEF: Left Ventricular Ejection Fraction]

Inclusion Criteria

Patients were eligible for inclusion if they met all of the following criteria:

1. Age> 18 years,
2. Underwent coronary angiography with a preliminary diagnosis of stable coronary artery disease,
3. Presence of normal sinus rhythm,
4. Euthyroid status,
5. Left ventricular ejection fraction (LVEF) ≥ 35%,
6. Not receiving dialysis therapy,
7. Estimated glomerular filtration rate (eGFR) ≥ 30 mL/min.

Exclusion Criteria

Patients were excluded if any of the following conditions were present:

1. Age <18 years,
2. Presence of arrhythmias or an implanted pacemaker,
3. Moderate-to-severe valvular heart disease or prosthetic heart valves,
4. Acute or chronic liver disease,
5. History of malignancy,
6. Left ventricular ejection fraction (LVEF) <35%,
7. Receipt of a blood transfusion within the preceding 3 months,
8. Sepsis or active infectious disease,
9. Myocardial infarction within the previous month,
10. Diagnosed inflammatory bowel disease, collagen tissue disease, or rheumatologic disorders.

Endpoints

The primary endpoint of the study was to evaluate how well these biomarkers can predict the presence of CTO. The secondary endpoint was to assess whether any of these biomarkers could act as independent predictors of CTO development.

Study Population

A total of 324 patients who underwent CAG during the study period were screened, and 96 patients who met the predefined inclusion criteria were enrolled. The CTO group consisted of 36 patients with 100% stenosis in at least one epicardial coronary artery. Patients were included consecutively based on predefined inclusion and exclusion criteria to minimize selection bias.

The control group consisted of 60 patients who underwent CAG for stable angina pectoris, had no evidence of CTO, and met the same inclusion and exclusion criteria as the CTO group, including patients with non-critical coronary stenosis or ischemia confirmed by functional imaging. The groups were compared with respect to inflammatory and metabolic biomarkers.

Table 1

Comparison of demographic, clinical, and laboratory characteristics between patients with and without chronic total occlusion (CTO)

Variables	CTO (+) (n = 36)	CTO (−) (n = 60)	p-value
Age (years)	63.83 ± 10.34	63.23 ± 9.20	0.768
Female sex, n (%)	4 (11.1)	8 (13.3)	0.750
BMI (kg/m^2)	27.04 ± 4.05	27.85 ± 4.28	0.363
Diabetes mellitus, n (%)	18 (50)	19 (31.7)	0.074
Hypertension, n (%)	23 (63.9)	41 (68.3)	0.655
Hyperlipidemia, n (%)	23 (63.9)	20 (33.3)	0.004
LVEF (%)	50 [45–55]	55 [55–60]	<0.001
Aspirin (ASA)	1.36 ± 0.49	1.60 ± 0.49	0.023
Clopidogrel	1.72 ± 0.45	1.88 ± 0.32	0.068
Statin	1 [1–2]	2 [2–2]	<0.001
ACE/ARB	1.5 [1–2]	1 [1–2]	0.875
Oral antidiabetic agents	2 [1–2]	2 [1–2]	0.091
Insulin	2 [2–2]	2 [2–2]	0.129
Hemoglobin (g/dL)	13.66 ± 1.95	14.34 ± 1.19	0.067
Hematocrit (%)	41.07 ± 5.68	42.75 ± 3.29	0.112
WBC (×10^3/μL)	7594 ± 2632	7855 ± 1992	0.584
Platelets (×10^3/μL)	274111 ± 85219	277066 ± 201050	0.933
Neutrophils (×10^3/μL)	5162 ± 1552	4775 ± 1657	0.260
Lymphocytes (×10^3/μL)	2220 [1665–2545]	2200 [1820–2552]	0.964
Triglycerides (mg/dL)	121.20 [92.25–190]	162.5 [110.25–219.5]	0.041
Total cholesterol (mg/dL)	163 [144.25–206.5]	191.5 [169.25–209.75]	0.005
HDL (mg/dL)	40.81 ± 9.74	42.85 ± 11.38	0.371
LDL (mg/dL)	105.17 ± 35.83	116.55 ± 31.83	0.109
Glucose (mg/dL)	119 [102.25–162.75]	113 [98.25–127.75]	0.128
Urea (mg/dL)	36.61 ± 21.90	33.55 ± 10.21	0.355
Creatinine (mg/dL)	0.8 [0.7–1.0]	0.9 [0.8–1.07]	0.073
GFR (mL/min)	88.42 ± 17.53	82.27 ± 19.59	0.126

Values are expressed as mean ± standard deviation, median [interquartile range], or number (percentage). BMI: body mass index; DM: diabetes mellitus; HT: hypertension; HL: hyperlipidemia; LVEF: left ventricular ejection fraction; ASA: acetylsalicylic acid; ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; WBC: white blood cell count; HDL: high-density lipoprotein; LDL: low-density lipoprotein; GFR: glomerular filtration rate; CTO: chronic total occlusion.

Laboratory Measurements

Blood samples were collected between 08:00 and 10:00 AM after 12 hours of fasting. A complete blood count and comprehensive metabolic panel were analyzed. Biomarkers were calculated as follows:

- Systemic Inflammatory Index (SII): platelet × neutrophil / lymphocyte
- Uric Acid/HDL Ratio (UHR)
- Monocyte/HDL Ratio (MHR)
- TyG Index: $\log_{10}$ [triglyceride × glucose / 2]

Ethical Approval

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Mersin City Training and Research Hospital and Mersin University Clinical Research Ethics Committee (Date: 28.05.2025, Decision No: 2025/614).

Statistical Analysis

All statistical analyses were performed using SPSS version 26.0 for Windows (SPSS Inc., Chicago, IL, USA). The normality of the distribution was assessed using the Shapiro–Wilk test. Continuous variables with a normal distribution are presented as mean ± standard deviation, whereas variables without a normal distribution are presented as median and interquartile range. Categorical variables are expressed as counts and percentages. Comparisons between groups were performed using the Student's t-test for normally distributed variables, the Mann–Whitney U test for non-normally distributed variables, and the Chi-square test for categorical variables.

Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the discriminative performance of SII, UHR, MHR, and the TyG index for predicting the presence of chronic total occlusion. Univariable logistic regression analysis was performed to examine the association between each individual clinical and laboratory variable and the presence of CTO. Results of the univariable analyses are presented as odds ratios (ORs) with corresponding 95% confidence intervals (CIs). Correlation analyses were conducted using Spearman's rank correlation coefficients to evaluate relationships between inflammatory biomarkers and relevant clinical variables. A p-value < 0.05 was considered statistically significant.

Table 2

Comparison of inflammatory and metabolic parameters between CTO and non-CTO groups

Parameters	CTO (+) (n = 36)	CTO (–) (n = 60)	p-value
SII (systemic immune-inflammation index)	678.72 ± 367.84	574.82 ± 274.18	0.118
UHR (uric acid-to-HDL ratio)	0.15 ± 0.07	0.15 ± 0.06	0.887
MHR (monocyte-to-HDL ratio)	13.81 ± 5.43	6.89 ± 6.15	<0.001
TyG index (triglyceride-glucose index)	8.99 ± 0.65	9.11 ± 0.52	0.333

SII: systemic immune-inflammation index; UHR: uric acid-to-HDL ratio; MHR: monocyte-to-HDL cholesterol ratio; TyG index: triglyceride-glucose index; CTO: chronic total occlusion. Values are expressed as mean ± standard deviation (SD).

Table 3

Diagnostic performance of inflammatory and metabolic biomarkers for predicting chronic total occlusion (CTO)

Score	Cut-off	AUC (95% CI)	p-value	Sensitivity (%)	Specificity (%)
MHR	>8.7	0.799 (0.705–0.874)	<0.001	80.6	66.7
SII	>511	0.563 (0.458–0.664)	0.298	63.9	45.0
TyG index	≤8.5	0.551 (0.446–0.652)	0.416	25.0	86.7
UHR	≤0.13	0.511 (0.407–0.615)	0.854	47.2	60.0

SII: systemic immune-inflammation index; UHR: uric acid-to-HDL ratio; MHR: monocyte-to-HDL cholesterol ratio; TyG index: triglyceride-glucose index; CTO: chronic total occlusion. AUC: area under the receiver operating characteristic (ROC) curve; CI: confidence interval; Higher AUC values indicate better discriminatory performance.

Table 4

Univariable Logistic Regression Analysis for Predictors of Chronic Total Occlusion (CTO)

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Age (years)	1.01	0.96 – 1.05	0.766
Sex (male)	1.23	0.34 – 4.42	0.750
Diabetes mellitus	0.46	0.20 – 1.08	0.076
Hypertension	0.88	0.55 – 1.42	0.608
MHR	1.20	1.11 – 1.31	<0.001
Ejection Fraction (%)	0.81	0.74 – 0.90	<0.001
UHR	1.63	0.001– 1239	0.885
TyG index	0.69	0.33 – 1.44	0.330
SII	1.00	0.9997 – 1.002	0.123

Abbreviations: MHR = monocyte-to-HDL cholesterol ratio; UHR = uric acid-to-HDL ratio; TyG index = triglyceride–glucose index; SII = systemic immune-inflammation index; CTO = chronic total occlusion.

Table ST1

Spearman Correlation Analysis Among Biomarkers and Clinical Variables

Variable 1	Variable 2	Spearman r	p-value
MHR	Ejection Fraction (%)	-0.248	0.015
MHR	SII	0.272	0.007
MHR	UHR	0.205	0.045
MHR	TyG index	0.029	0.779
Ejection Fraction (%)	TyG index	0.258	0.011
Ejection Fraction (%)	SII	0.022	0.829
SII	UHR	0.004	0.973
UHR	TyG index	0.236	0.021

Abbreviations: MHR = monocyte-to-HDL cholesterol ratio; SII = systemic immune-inflammation index; UHR = uric acid-to-HDL ratio; TyG index = triglyceride–glucose index.

3. Results

A total of 96 patients who met the inclusion criteria were analyzed, including 36 patients with CTO and 60 without CTO. The baseline demographic, clinical, and laboratory characteristics of the study population are presented in Table 1.

There were no significant differences between groups with respect to age, sex distribution, BMI, diabetes mellitus, hypertension, hemoglobin levels, renal function parameters, or most hematologic indices (all p> 0.05). However, hyperlipidemia was significantly more frequent in the CTO group (63.9% vs. 33.3%, p = 0.004), and LVEF was significantly lower among patients with CTO (median 50% vs. 55%, p <0.001). Triglyceride levels were lower in the CTO group (p = 0.041), whereas total cholesterol levels were also significantly lower in patients with CTO (p = 0.005). Aspirin use was more common in the non-CTO group (p = 0.023), and statin use was significantly higher among non-CTO patients (p <0.001). Comparison of inflammatory and metabolic biomarkers is shown in Table 2.

Mean SII, NLR, and TyG index values were similar between the CTO and non-CTO groups (all p> 0.05). In contrast, MHR levels were markedly higher in patients with CTO (13.81 ± 5.43 vs. 6.89 ± 6.15, p <0.001), the only inflammatory/metabolic marker to differ significantly between the two groups. The diagnostic performance of these biomarkers for predicting CTO is summarized in Table 3. MHR demonstrated the strongest discriminative ability, with an AUC of 0.799 (95% CI: 0.705–0.874; p <0.001), as shown in the ROC analyses in Figure 2. Using a cut-off value of>8.7, MHR yielded

Figure 1

ROC Curve Analysis of Variables for Predicting CTO Groups
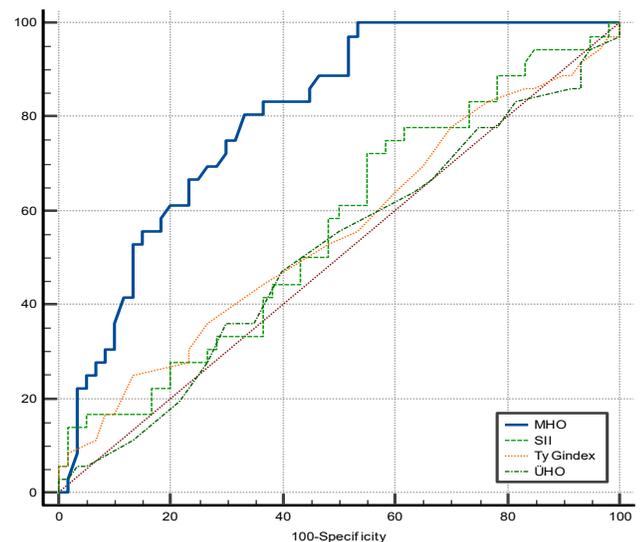
The ROC curves illustrate the discriminative performance of the evaluated variables in predicting CTO patient groups. Among these, the monocyte-to-HDL cholesterol ratio (MHR) demonstrated the strongest predictive ability, yielding an AUC of 0.799 (95% CI: 0.705–0.874; p <0.001). Sensitivity and specificity values corresponding to the optimal cut-off points are presented for each variable.

[ROC: Receiver Operating Characteristic; AUC: Area Under the Curve; CTO: Chronic Total Occlusion; HDL: High-Density Lipoprotein; MHR: Monocyte-to-HDL Cholesterol Ratio.]

80.6% sensitivity and 66.7% specificity. SII, NLR, and the TyG index showed limited predictive accuracy, with AUC values of 0.563, 0.511, and 0.551, respectively.

Univariable logistic regression analysis was performed to evaluate the association between clinical variables and the presence of CTO. In this analysis, a higher MHR was significantly associated with an increased likelihood of CTO (OR 1.20, 95% CI 1.11–1.31, p <0.001), whereas a higher ejection fraction was significantly associated with a reduced likelihood of CTO (OR 0.81, 95% CI 0.74–0.90, p < 0.001). Diabetes mellitus showed a borderline association (OR 0.46, 95% CI 0.20–1.08, p = 0.076), whereas age, sex, hypertension, UHR, TyG index, and SII were not significantly associated with CTO in univariable analysis (Table 4).

Correlation analyses revealed a modest inverse correlation between MHR and ejection fraction (r = –0.248, p = 0.015). MHR showed weak but statistically significant positive correlations with SII (r = 0.272, p = 0.007) and UHR (r = 0.205, p = 0.045), while no significant correlation was observed between MHR and the TyG index. No strong correlations were identified among the evaluated biomarkers (Supplementary Table 1).

4. Discussion

In this study, the roles of novel inflammatory and metabolic biomarkers in predicting the presence of CTO among patients presenting with stable angina pectoris were evaluated, and the findings were interpreted in the context of the current literature.

Previous studies have reported that the SII is positively associated with the severity of CAD and reflects atherosclerotic burden;[9,10] however, in our study, SII did not significantly predict the presence of CTO. This may be due to the largely completed chronic inflammatory process in CTO, as well as the suppressive effects of statin/aspirin therapy on inflammatory markers.[11,12]

Similarly, although UHR has been shown to reflect systemic inflammation and cardiovascular risk,[13,14] its discriminatory ability for CTO was limited in our cohort. Moreover, while the TyG index has been associated with metabolic risk and atherosclerotic burden in prior studies,[15,16] our findings suggest that TyG may not be sufficiently specific for CTO. The higher triglyceride and total cholesterol levels observed in the non-CTO group may also have reduced its discriminatory capacity.

On the other hand, strong evidence links the MHR with CAD severity, multivessel disease, SYNTAX score, and major adverse cardiac events.[17–19] Additionally, higher mortality rates have been reported among CTO patients with elevated MHR.[20] Several studies have also demonstrated that MHR may serve as a risk factor for CTO development and that certain cutoff values can predict CTO.[21–23]

The association between MHR and CTO likely arises from the combined effect of enhanced monocyte-driven inflammation and the diminished protective function of HDL. Following endothelial dysfunction, monocytes migrate into the vessel wall, transform into macrophages, accumulate oxidized LDL, and contribute to foam cell formation—processes that accelerate local inflammation and plaque progression. Over time, these inflammatory mechanisms promote fibrotic and calcified plaque formation, creating the substrate for CTO.[21,24] HDL, in contrast, exerts anti-inflammatory and antioxidative effects by inhibiting monocyte activation and oxidative stress; thus, reduced HDL levels facilitate inflammation-driven plaque advancement. Consequently, elevated MHR reflects the coexistence of heightened inflammation and impaired antiatherogenic capacity, thereby indicating increased CTO risk.[25] Therefore, the observed association between MHR and CTO should be interpreted as cross-sectional rather than causal.

In, our study, the strong association between high MHR levels and the presence of CTO, along with its superior discriminatory performance compared with other biomarkers, supports MHR as a clinically valuable screening tool. However, given its moderate discriminatory performance, MHR should be considered a supportive risk marker rather than a standalone clinical decision-making tool. Furthermore, its ease of calculation and low cost may enhance its practicality for CTO risk assessment in routine clinical practice.

Limitations
This study has several limitations. First, its retrospective, single-center design may introduce selection bias and limit how applicable the results are to other settings. In addition, the marked reduction from the initially screened population to the final study cohort and the definition of the control group may have further contributed to selection bias. Also, the relatively small sample size might have lowered the statistical power and contributed to the lack of significant links for some biomarkers. Inflammatory and metabolic markers were measured only once, which prevents analysis of how they vary over time. The possible influence of medications such as statins and aspirin on inflammatory biomarker levels could not be fully controlled and may represent residual confounding. Additionally, the exploratory nature of the study and the absence of a predefined primary hypothesis should be taken into account when interpreting the results. Finally, because the study is observational and cross-sectional, and biomarkers were measured at a single time point, it cannot establish causality.

5. Conclusion

In this exploratory analysis, our findings suggest that the MHR may be a useful biomarker associated with the presence of chronic total occlusion in patients with stable angina pectoris. In contrast, SII, UHR, and the TyG index did not offer meaningful diagnostic value for identifying CTO.

Our findings are consistent with previous studies that have linked MHR to the severity of coronary artery disease, suggesting that MHR could serve as a useful additional marker in the evaluation of CTO. These findings should be considered hypothesis-generating and require confirmation in larger, prospective studies.

Statement of ethics
The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Mersin City Training and Research Hospital and Mersin University Clinical Research Ethics Committee (Date: 28.05.2025, Decision No: 2025/614).

genAI
No artificial intelligence-based tools or generative AI technologies were used in this study. The entire content of the manuscript was originally prepared, reviewed, and approved by both authors.

Conflict of interest statement
The authors declare that they have no conflict of interest.

Availability of data and materials
The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Author contributions
Both authors contributed equally to the conception, writing, and revision of the manuscript. Both authors read and approved the final version of the manuscript.

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