Original Article

# The Importance of Monocyte-to-High Density Lipoprotein-Cholesterol Ratio in Predicting Severity of Coronary Artery Disease in Acute Coronary Syndrome

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**Abstract**

**Background:** Evaluating the association between coronary artery disease (CAD) severity and new inflammatory markers in acute coronary syndrome (ACS) is critical for managing the therapy procedure as well. **Aim:** The primary goal of this study was to investigate the association between the monocyte-to-high density lipoprotein-cholesterol ratio (MHR), a novel inflammation marker, and the severity of CAD in patients with ACS. **Methods:** The study was performed on ACS patients who were hospitalized for coronary angiography (CAG) in the coronary intensive care unit and was conducted with a retrospective design. The study comprised 344 patients (mean age 60.49 ± 12.23 years) with ACS who had CAG and laboratory testing. There were 212 patients with mild CAD according to the Synergy between percutaneous coronary intervention (PCI) with Taxus and Cardiac Surgery (SYNTAX) score (SYNTAX score ≤22) and 132 patients with severe CAD (SYNTAX score >22). The association between SYNTAX score, MHR, uric acid, the neutrophil-to-lymphocyte ratio (NLR), and other markers were assessed. All analyses were performed using SPSS 26.0. **Results:** A modestly linear association was observed between MHR and SYNTAX score ($r$ = 0.522, $P$ < 0.001). Multivariate logistic regression analysis found male gender, high uric acid, high MHR, and NLR as possible individual predictors of SYNTAX score >22 in ACS. The receiver operating characteristic (ROC) analysis revealed that MHR 15.64 (AUC = 0.794; $P$ < 0.001) could predict SYNTAX score >22 with higher sensitivity (81.8%) and specificity (78.3%). **Conclusions:** The higher MHR independently predicts the severity of CAD in ACS. It may be a better parameter than the higher NLR and uric acid levels to predict CAD severity in ACS patients.

**Keywords:** *Coronary, monocyte, neutrophil-to-lymphocyte ratio, SYNTAX score, uric acid*

## Introduction

Coronary artery disease (CAD) constitutes the main cause of morbidity and mortality around the world. One of the most significant prognostic factor for CAD patients is the complex nature of the disease.[1] The complexity of CAD is reflected in many scores.[2,3] The Synergy between percutaneous coronary intervention (PCI) with Taxus and Cardiac Surgery (SYNTAX) score not only helps clinicians to decide on the type of coronary intervention to be performed, but also indicates the patient's short and long-term prognosis based on lesion location, severity and characteristics.[2,4]

An inflammatory and lipid-accumulating chronic disease of the vascular wall is called atherosclerosis.[5]

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There are many inflammatory mediators that correlate with the extent of atherosclerosis and the degree of inflammation.[6] Monocytes are one of the major cell types present in the atherosclerotic plaque. And monocytes secrete proinflammatory and prooxidant cytokines that cause the progression of atherosclerosis.[7] High density lipoprotein-cholesterol (HDL-C), a kind of plasma lipid, exhibits anti-inflammatory and anti-atherosclerotic properties.[8] The monocyte-to-high density lipoprotein-cholesterol ratio (MHR) is hypothesized to be correlated with oxidative stress and inflammation.[9]

There are studies showing that MHR is associated with increased disease burden and complexity in stable CAD and ST elevation myocardial infarction (STEMI).[10,11] Increased MHR has also been shown to be associated with poor outcomes in these patient groups.[12]

Our research aimed to determine whether CAD severity and MHR were related in acute coronary syndrome (ACS) patients. Secondly, we aimed to evaluate the effect of MHR in predicting the severity of CAD in ACS compared to other inflammatory markers.

## Subjects and Methods

Retrospective research was carried out on 356 consecutive patients hospitalized to Siirt Education and Research Hospital with an ACS diagnosis between January 2022 and January 2023. Patients with stable angina pectoris (SAP), patients with contraindications to coronary angiography (CAG), and patients with inflammatory and chronic infectious diseases were excluded. Finally, 344 patients were participated in the study. The research adhered to the norms of the Declaration of Helsinki and was granted approval by Siirt University's Ethics Committee.

### Blood sampling

Cardiovascular risk factors and comorbidities were determined from medical history and medical records. At enrolment, all patients had a complete blood count (CBC) and routine biochemistry. The CBC was analyzed on a DXH-800 automatic analyzer in the biochemistry laboratory and the biochemical parameters were analyzed on a Beckman Coulter AU5800 clinical chemistry analyzer. After analyzing the CBC data, the MHR ratio was computed by dividing the monocyte count by HDL-C, and the neutrophil-to-lymphocyte ratio (NLR) was manually obtained by dividing the neutrophil count by the lymphocyte count. Patients were classified according to tertiles of MHR: low (<11.09), intermediate (11.09–18.92) and high (>18.92) MHR groups.

### Angiographic analysis

The patients were taken to CAG unit within 48 hours of the diagnosis of ACS based on clinical risk stratification. All invasive procedures were done using the Judkins procedure and a biplane angiography system (*Artis Zee, Siemens, Germany*) via the femoral or radial approach. *Opaxol®* (Ioheksol 350 mgI/mL), a non-ionized low-osmolar agent of contrast was utilized. All coronary arteries were evaluated and documented in several images taken from various angles.

After the CAG, two specialists in cardiology were blind to the patient's clinical and laboratory results, evaluated all angiographic images and calculated each patient's SYNTAX score. The study included patients with coronary artery lesions that were above 1.5 mm in diameter and caused vessels to narrow by more than 50%. Each lesion's score was calculated separately, and the total SYNTAX score was determined using *Syntax Score Calculator v2.02 (www.syntaxscore.com)*.[13] After SYNTAX score calculation, two groups were defined: mild CAD (SYNTAX score ≤ 22) and severe CAD (SYNTAX score > 22).

### Statistical analysis

The data collected for the research was examined using the *SPSS 26 application (IBM Corporation, Armonk, NY, USA)*. Continuous variables were defined as mean ± standard deviation (SD) or median (minimum-maximum values), and categorical variables as percentages. After normality distribution was evaluated with the Kolmogorov-Smirnov test, student *t*-test was used to compare two groups in normally distributed data, and One-way ANOVA test was applied to compare three groups. *Post hoc* analysis was conducted using the Tukey test. When the data lacked a normal distribution, the Mann Whitney U test was used for two group comparisons, and the Kruskal Wallis test for three group comparisons. Categorical data was compared using the Pearson Chi-square test. Pearson's correlation test was used to conduct the correlation analysis. Logistic regression analyzes were done to find out independent determinants of SYNTAX score > 22. Significant predictors were analyzed using the receiver operating characteristic (ROC) analysis. All comparisons were deemed statistically noteworthy with a $P$ value of < 0.05.

## Results

The study group had a mean age of 60.49 ± 12.23 years and 191 (55.5%) were male. Among the patients, 182 (52.9%) had hypertension (HT), 94 (27.3%) had diabetes mellitus (DM), 182 (52.9%) had dyslipidemia, 152 (44.2%) were smokers, 124 (36%) had a family

**Table 1: Comparison of mild versus severe CAD groups in terms of demographic, clinical and biochemical characteristics**

| Parameters | Total (*n*=344) Mean±SD, Median (Min - Max) or *n* (%) | Mild CAD (*n*=212) Mean±SD, Median (Min - Max) or *n* (%) | Severe CAD (*n*=132) Mean±SD, Median (Min - Max) or *n* (%) | *P* |
|---|---|---|---|---|
| Age (years) | 60.49±12.23 | 60.94±11.83 | 59.77±12.86 | 0.389 |
| Male sex (%) | 55.5 (191) | 49.5 (105) | 65.2 (86) | 0.005* |
| HT (%) | 52.9 (182) | 49.1 (104) | 59.1 (78) | 0.070 |
| DM (%) | 27.3 (94) | 21.7 (46) | 36.4 (48) | 0.003* |
| Dyslipidemia (%) | 52.9 (182) | 44.3 (94) | 66.7 (88) | <0.001* |
| Smoking (%) | 44.2 (152) | 42.5 (90) | 47 (62) | 0.412 |
| Family history (%) | 36 (124) | 33 (70) | 40.9 (54) | 0.138 |
| Previous MI (%) | 50.6 (174) | 49.1 (104) | 53 (70) | 0.473 |
| UAP (%) | 32 (110) | 44.3 (94) | 12.1 (16) | <0.001* |
| NonSTEMI (%) | 33.1 (114) | 37.7 (80) | 25.8 (34) | <0.001* |
| STEMI (%) | 34.9 (120) | 17.9 (38) | 62.1 (82) | <0.001* |
| 1 vessel disease (%) | 56.4 (194) | 71.7 (152) | 31.8 (42) | <0.001* |
| 2 vessel disease (%) | 30.8 (106) | 27.4 (58) | 36.4 (48) | <0.001* |
| 3 vessel disease (%) | 12.8 (44) | 0.9 (2) | 31.8 (42) | <0.001* |
| Diastolic TA (mmHg) | 80.85±5.96 | 81.30±5.99 | 80.12±5.83 | 0.074 |
| Systolic TA (mmHg) | 123.89±13.66 | 125.16±13.08 | 121.85±14.30 | 0.028* |
| BMI (kg/m$^2$) | 27.75±4.28 | 27.19±4.49 | 28.65±3.76 | 0.020† |
| FBG (mg/dl) | 127.22±41.43 | 113.76±34.36 | 148.82±42.77 | <0.001† |
| Creatinine (mg/dl) | 1.00±0.30 | 0.97±0.33 | 1.05±0.25 | 0.027† |
| Uric acid (mg/dl) | 5.40±1.37 | 5.05±1.07 | 5.95±1.65 | <0.001† |
| Urea (mg/dl) | 33.16±9.29 | 31.43±8.66 | 35.93±9.62 | <0.001† |
| GFR (ml/min) | 75.79±24.06 | 77.43±24.99 | 73.15±22.32 | 0.109 |

*Chi-square test; †Student's *t*-test; ‡Mann Whitney *U*-test; $P<0.05$. CAD=coronary artery disease, HT=Hypertension, DM=Diabetes Mellitus, MI=Miyocardial infarction, UAP=Unstable angina pectoris, STEMI=ST elevation myocardial infarction, BMI=Body mass index, FBG=Fasting blood glucose, GFR=Glomerular filtration rate

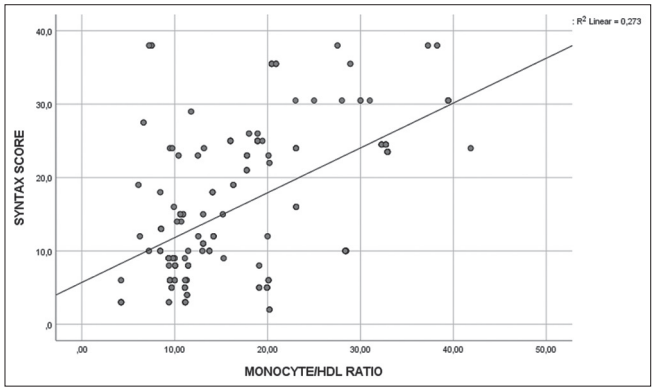


**Figure 1:** Pearson correlation analysis between the MHR and SYNTAX score

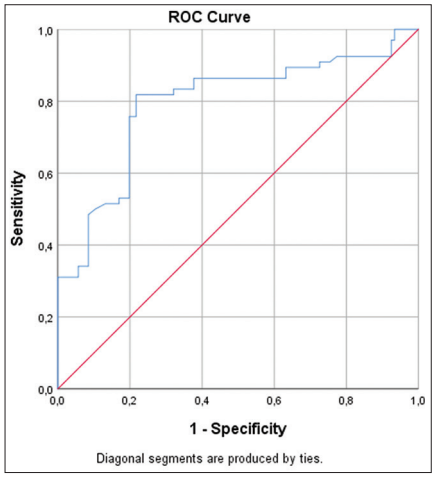


**Figure 2:** ROC analysis graph of MHR in predicting severe CAD

history of cardiovascular disease, and 174 (50.6%) had a history of myocardial infarction (MI). Of all patients, 110 (32%) were hospitalized for unstable angina pectoris (UAP), 114 (33.1%) for NonSTEMI, and 120 (34.9%) for STEMI. Table 1 illustrates the baseline characteristics of the study participants.

Patients were divided into mild and severe CAD groups according to SYNTAX score and a comparison was made between the two groups. The severe CAD group had drastically higher rates of male gender (65.2% vs. 49.5%; $P = 0.005$), DM (36.4% vs. 21.7%; $P = 0.003$), and dyslipidemia (66.7% vs. 44.3%; $P < 0.001$) [Table 1]. The mild CAD group had significantly higher rates of UAP (44.3% vs. 12.1%; $P < 0.001$) and NonSTEMI (37.7% vs. 25.8%; $P < 0.001$). STEMI rates were dramatically higher in the severe CAD group

**Table 2: Comparison of mild versus severe CAD groups in terms of SYNTAX score, hemogram, lipid, inflammatory and cardiac biomarker levels**

| Parameters | Total (*n*=344) Mean±SD, Median (Min - Max) or *n* (%) | Mild CAD (*n*=212) Mean±SD, Median (Min - Max) or *n* (%) | Severe CAD (*n*=132) Mean±SD, Median (Min - Max) or *n* (%) | *P* |
|---|---|---|---|---|
| Troponin (ng/mL) | 0.56 (0-25) | 0.21 (0.03-5.94) | 4.93 (0-25) | <0.001‡ |
| Hemoglobin (g/dl) | 13.94±1.64 | 14.08±1.39 | 13.72±1.97 | 0.071 |
| Rbc ($10^6$/µL) | 4.86±0.50 | 4.91±0.47 | 4.78±0.54 | 0.022† |
| Platelet ($10^3$/mm$^3$) | 248±65 | 251±71.9 | 245±52.3 | 0.439 |
| Wbc ($10^3$/µL) | 8.92±3.17 | 7.59±1.60 | 11.06±3.83 | <0.001† |
| Neutrophil ($10^3$/µL) | 5.72±2.31 | 4.90±1.20 | 7.04±2.97 | <0.001† |
| Lymphocyte ($10^3$/µL) | 2.09±0.78 | 1.96±0.59 | 2.29±0.99 | <0.001† |
| Monocyte ($10^3$/µL) | 0.62±0.28 | 0.52±0.15 | 0.79±0.36 | <0.001† |
| Total cholesterol (mg/dl) | 189.62±47.05 | 185.27±45.7 | 196.61±48.36 | 0.030† |
| LDL (mg/dl) | 117.92±34.07 | 106.81±29.58 | 135.78±33.29 | <0.001† |
| HDL (mg/dl) | 41.93±8.76 | 43.60±9.37 | 39.25±6.90 | <0.001† |
| Triglyceride (mg/dl) | 168.94±71.72 | 155.83±70.45 | 190±68.92 | <0.001† |
| AIP | 0.21±0.21 | 0.15±0.20 | 0.29±0.19 | <0.001† |
| CRP (mg/dl) | 8 (1.09-135.1) | 4.7 (1.09-41.7) | 18.9 (1.6-135.1) | <0.001‡ |
| HgA1c (%) | 6.01±0.99 | 5.86±0.93 | 6.23±1.04 | 0.010† |
| NLR | 3.01±1.49 | 2.80±1.53 | 3.34±1.37 | 0.010† |
| MHR | 16.55±8.58 | 12.97±5.65 | 22.30±9.35 | <0.001† |
| SYNTAX score | 15.89±10.06 | 8.96±4.55 | 27.01±5.48 | <0.001† |

†Student's *t*-test; ‡Mann Whitney *U*-test; *P*<0.05. CAD=Coronary artery disease, Rbc=Red blood cell, Wbc=White blood cell, LDL=Low density lipoprotein, HDL=High density lipoprotein, AIP=Atherogenic index of plasma, CRP=C-reactive protein, HgA1c=Hemoglobin A1c, NLR=Neutrophil-to-lymphocyte ratio, MHR=Monocyte-to-high density lipoprotein-cholesterol ratio, SYNTAX=SYNergy between PCI with TAXUS and Cardiac Surgery

**Table 3: Comparison of SYNTAX score, CRP, troponin-I levels and STEMI, 3-vessel disease frequencies according to MHR tertile groups**

| Parameters | MHR 1. tertil (*n*=118) Mean±SD, Median (Min - Max) or *n* (%) | MHR 2. tertil (*n*=112) Mean±SD, Median (Min - Max) or *n* (%) | MHR 3. tertil (*n*=112) Mean±SD, Median (Min - Max) or *n* (%) | *P* |
|---|---|---|---|---|
| SYNTAX score | 11.46±8.40 | 15.26±8.20 | 20.97±11.04 | <0.001**,††,‡‡ |
| CRP (mg/dl) | 3.8 (1.5–35.2) | 7.3 (1.09–28.5) | 41.6 (4.7–135.1) | <0.001**,††,‡‡ |
| Troponin-I (ng/ml) | 0.26 (0–14.08) | 0.437 (0.01–25) | 1.25 (0.06–24.55) | <0.001††,‡‡ |
| STEMI (%) | 28 (%23.3) | 30 (%25) | 62 (%51.7) | <0.001* |
| 3-vessel disease (%) | 12 (%27.3) | 6 (%13.6) | 26 (%59.1) | <0.001* |

ANOVA, post hoc Tukey test. **$P$<0.05 between 1. and 2. tertil, ††$P$<0.05 between 1. and 3. tertil, ‡‡$P$<0.05 between 2. and 3. tertil. *Chi-square test $P$<0.05. SYNTAX=SYNergy between PCI with TAXUS and Cardiac Surgery, CRP=C-reactive protein, STEMI=ST elevation myocardial infarction, MHR=Monocyte-to-high density lipoprotein-cholesterol ratio

(62.1% vs. 17.9%; $P < 0.001$). Furthermore, body mass index (BMI) ($P = 0.020$), fasting glucose ($P < 0.001$), creatinine ($P = 0.027$), urea ($P < 0.001$) was higher in the severe CAD group [Table 1].

In addition, troponin-I ($P < 0.001$), C-reactive protein (CRP) ($P < 0.001$) and Hemoglobin A1c (HbA1c) ($P = 0.010$) were notably higher in the severe CAD group. While there was no significant difference between the two groups in terms of CBC parameters hemoglobin and platelet ($P > 0.05$); other parameters white blood cell (Wbc), neutrophil, lymphocyte and monocyte values were significantly higher in the severe CAD group ($P < 0.001$). The red blood cell (Rbc) value was significantly lower in the severe CAD group ($P = 0.022$). Among the lipid parameters; total cholesterol ($P = 0.030$), low density lipoprotein (LDL) ($P < 0.001$), and triglyceride ($P < 0.001$) values were significantly higher in the severe CAD group, while HDL-C value was significantly lower in the severe CAD group ($P < 0.001$). In addition, the atherogenic index of plasma (AIP) value was observed to be higher in the severe CAD group ($P < 0.001$) [Table 2].

Furthermore, the severe CAD group had a significantly higher MHR when compared to the other group (12.97 ± 5.65 vs. 22.30 ± 9.35; $P < 0.001$). For

**Table 4: Univariate and multivariate logistic regression analysis for severe CAD**

| Parameters | Univariate | | Multivariate | |
|---|---|---|---|---|
| | O.R. (95%CI) | *P* | O.R. (95%CI) | *P* |
| DM (%) | 0.485 (0.299–0.785) | 0.003* | - | 0.059 |
| Male (%) | 1.905 (1.217–2.982) | 0.005* | 0.332 (0.147–0.751) | 0.008** |
| Dislipidemia (%) | 2.511 (1.598–3.946) | <0.001* | - | 0.454 |
| Uric acid (mg/dl) | 1.665 (1.391–1.993) | <0.001* | 2.405 (1.719–3.367) | <0.001** |
| MHR | 1.173 (1.130–1.218) | <0.001* | 1.262 (1.178–1.352) | <0.001** |
| NLR | 1.277 (1.101–1.482) | <0.001* | 1.480 (1.192–1.837) | <0.001** |
| BMI (kg/m$^2$) | 1.083 (1.029–1.140) | 0.002* | - | 0.594 |
| HgA1c (%) | 1.446 (1.162–1.799) | <0.001* | - | 0.092 |
| LDL (mg/dl) | 1.029 (1.021–1.038) | <0.001* | - | 0.090 |
| AIP | 28.529 (8.976–90.675) | <0.001* | - | 0.220 |

Univariate logistic regression. **P*<0.05. Multivariate logistic regression. ***P*<0.05. CAD=coronary artery disease, DM=Diabetes Mellitus, MHR=Monocyte-to-high density lipoprotein-cholesterol ratio, NLR=Neutrophil-to-lymphocyte ratio, BMI=Body mass index, HgA1c=Hemoglobin A1c, LDL=Low density lipoprotein, AIP=Atherogenic index of plasma

**Table 5: ROC analysis to predict severe CAD**

| Parameters | AUC (95% CI) | Cut-off | *P* | Sensitivity (%) | Specifity (%) |
|---|---|---|---|---|---|
| NLR | 0.655 (0.590–0.720) | 2.935 | <0.001* | 71.2 | 70.8 |
| MHR | 0.794 (0.741–0.846) | 15.64 | <0.001* | 81.8 | 78.3 |
| Uric aside (mg/dl) | 0.664 (0.603–0.726) | 5.47 | <0.001* | 63.6 | 67.9 |

Receiver Operating Characteristic (ROC) analysis; AUC=Area under the curve. **P*<0.05. CAD=coronary artery disease, NLR=Neutrophil-to-lymphocyte ratio, MHR=Monocyte-to-high density lipoprotein-cholesterol ratio

NLR, the severe CAD group had a much higher ratio than the mild CAD group. (2.8 ± 1.53 vs. 3.34 ± 1.37; $P = 0.01$). Moreover, the severe CAD group had a higher uric acid level (5.95 ± 1.65 vs. 5.05 ± 1.07; $P < 0.001$) [Tables 1 and 2, respectively].

The MHR stratified into tertiles. The study group comprised 118 patients in the first tertile of MHR, 114 patients in the second tertile of MHR, and 112 patients in the third tertile. Upon comparison, it was observed that SYNTAX score, CRP, troponin-I, STEMI, and 3-vessel disease significantly increased with increasing tertiles [Table 3].

A modestly positive association ($r = 0.522$, $P < 0.001$) between MHR and SYNTAX score seen in the Pearson correlation analysis [Figure 1]. The study found that male gender, high uric acid level, high MHR and NLR ratios were possible independent predictors of SYNTAX score >22 in ACS patients. Table 4 summarizes the outcomes of the regression analyzes. Table 5 shows the results of the ROC analysis, which indicate that the MHR cut-off of 15.64 has a sensitivity of 81.8% and a specificity of 78.3% for predicting SYNTAX score >22 (AUC = 0.794 (0.741-0.846), $P < 0.001$). Figure 2 illustrates the ROC curve for the MHR.

## Discussion

Our research found a substantial correlation between MHR and the severity of CAD in patients with ACS, as measured by the SYNTAX score. Additionally, increasing MHR is linked to more severe disease.

Atherosclerosis causes inflammation, and the migration of pro-inflammatory cells, including monocytes, is an important stage in the growth of plaque.[14] Monocytes take on a key function in plaque development and inflammation progression. In contrast, HDL-C has anti-inflammatory and anti-atherosclerotic properties.[15]

Several studies have indicated that MHR is an inflammatory marker and is related with poor outcomes in both cardiac and non-cardiac disorders.[10,16] Additionally, some studies have found a link between MHR and the presence of atherosclerosis.[17]

Studies have found a relationship between MHR and the level of deterioration of CAD. Akboga *et al*.[10] discovered that MHR is a significant indicator of high SYNTAX score (OR = 1.083, $P < 0.001$). Çetin *et al*.[12] reported that MHR is strongly associated with increased SYNTAX score in STEMI patients. Additionally, the incidence of major adverse cardiac events (MACE) increases with MHR tertile in the same study. Villanueva *et al*.[18] conducted a meta-analysis that demonstrated a positive correlation between higher MHR and higher MACE. Çağdaş *et al*.[19] found that MHR was correlated with SYNTAX score and SYNTAX score II in patients with STEMI. Arisoy *et al*.[11] demonstrated that MHR correlates with thrombus burden, a higher MHR being a reliable indicator of high thrombus burden

as characterized by the Thrombolysis in Myocardial Infarction (TIMI) grade. Our study found a strong relationship between MHR and SYNTAX score. Increasing MHR tertile groups showed a significant increase in SYNTAX score, STEMI rate, and critical 3-vessel disease rates, which is consistent with findings in the literature.

It has been emphasized that MHR is also associated with adverse outcomes after ACS. In research by Eyyupkoca and colleagues,[20] 231 patients with a history of STEMI were evaluated for adverse cardiac remodeling and found that high maximum heart rate was an important indicator of adverse cardiac remodeling, which could be assessed by the increase in left ventricular end-diastolic volume acquired by cardiac magnetic resonance imaging (MRI).

There have been studies on the association between MHR and stable CAD and STEMI. However, there has been limited study on the relationship between MHR and the seriousness of CAD in patients with UAP, NonSTEMI, and STEMI. Kalyoncuoglu *et al*.[21] found a link between high MHR and coronary slow flow/no-reflow in NonSTEMI patients after PCI. MHR has also been studied in the UAP population for stent restenosis. Tok *et al*.[22] found that MHR >14 significantly predicts stent restenosis in patients with stable CAD and UAP treated with bare metal stents.

Çağdaş *et al*.[19] also noted that MHR is a better indicator of CAD severity in STEMI patients compared to NLR and CRP. They emphasized that an MHR value of 13.9 can predict SYNTAX score > 22 with 76% sensitivity and 74% specificity (AUC = 0.786; $P < 0.001$). Our study found that MHR (AUC = 0.794; $P < 0.001$) was a better indicator of CAD severity than NLR (AUC = 0.655; $P < 0.001$) and uric acid levels (AUC = 0.664; $P < 0.001$) in all ACS groups, not just STEMI patients. Furthermore, MHR 15.64 was shown to predict SYNTAX score > 22 with higher sensitivity (81.8%) and specificity (78.3%) (AUC = 0.794; $P < 0.001$). Mohanty *et al*.[23] highlighted that MHR is a more effective predictor of CAD severity in ACS patients than the monocyte-to-lymphocyte ratio (MLR) and NLR.

### Limitations

However, our study had limitations. This study was implemented as a retrospective, single-center with a limited sample size. Additionally, the association between MHR and myocardial infarction with nonobstructive coronary arteries (MINOCA) syndrome caused by non-obstructive CAD was not included in the analysis. Advanced imaging techniques such as optical coherence tomography (OCT) and intravascular ultrasound (IVUS) were not used, only visual evaluation of CAD.

## Conclusions

The higher MHR is a better predictor of CAD severity and complexity in ACS patients than the higher NLR and uric acid levels. It is inexpensive and easy to apply, making it useful for ACS risk stratification in cardiology practice.

### Key messages

The higher MHR may be a better parameter than the higher NLR and uric acid levels to predict CAD severity in ACS patients.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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