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Admission Monocyte/HDL Ratio Predicts Adverse Cardiac Remodeling After ST-Elevation Myocardial Infarction

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ABSTRACT

Background: Inflammation plays a critical role in cardiac remodeling after myocardial infarction (MI). Monocyte to high-density lipoprotein-cholesterol (HDL-C) ratio (MHR) has emerged as a potential indicator of inflammation. **Objectives:** The study aimed to investigate the prognostic role of MHR at the time of hospital admission in late cardiac remodeling and subsequent 1-year mortality in an academic training and research hospital. **Methods:** This prospective multicenter study included 231 patients with acute ST-elevation MI. Left ventricular (LV) functions and volumes were assessed by cardiac magnetic resonance (CMR) imaging at 2 weeks and 6 months post-MI. The definition of adverse cardiac remodeling (AR) was based on the increase of LV end-diastolic volume by ≥ 12% at 6 months post-MI. All patients were followed for survival for 1 year after the second CMR imaging measurements. **Results:** At 6 months post-MI, 20 patients (23.8%) exhibited AR. The median MHR was higher in the AR group compared to the group without AR (2.2 vs. 1.5, $p < 0.001$). A positive correlation was found between MHR and infarct size in the groups with and without AR. High MHR was an independent predictor of AR (OR: 3.21, $p = 0.002$). The cut-off value of MHR in predicting AR was found to be >1.6 with 92.7% sensitivity and 70.1% specificity (AUC ± SE: 0.839 ± 0.03, $p < 0.001$). Mortality risk was 5.62-fold higher in the group with MHR of >1.6 (HR: 5.62, $p < 0.001$). **Conclusions:** These results indicate that admission MHR is a useful tool to predict patients with AR who are at risk of progression to heart failure and mortality after MI. (REV INVEST CLIN. 2022;74(2):104-12)

Keywords: Biomarker. Cardiac remodeling. Monocyte to high-density lipoprotein-cholesterol ratio. Myocardial infarction.

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INTRODUCTION

Cardiac remodeling after acute myocardial infarction (MI) is related to the balance between the extent of ischemia, inflammation, and oxidant radicals and the levels of defense systems against these pathological conditions[1-3]. Inflammation is not only a local response but it is also a systemic process that follows the elevation of inflammatory mediators. While extremely elevated inflammation or oxidant radicals are associated with adverse cardiac remodeling (AR), elevated defensive systems (anti-inflammatory and antioxidant balance) are associated with reverse or adaptive cardiac remodeling[4,5].

Monocytes, which are the main players of innate immunity, extravasate to inflammatory tissues after the leukocyte recruitment cascade during inflammation[6]. Lipid-loaded macrophages, which are produced due to the activation of monocytes in the area of inflammation, are of critical importance in the inflammatory response[7]. Oxidation of high-density lipoprotein-cholesterol (HDL-C) mainly occurs in inflammatory microenvironments. HDL-C plays a role in suppressing the activation of monocytes and the proliferation and differentiation of monocyte progenitor cells[8]. Thus, accumulation of monocytes and decrease in HDL-C are associated with atherosclerosis and cardiovascular diseases. Recent studies show that the monocyte to HDL-C ratio (MHR), which is an easily calculable measure, may be a new indicator of inflammation[9]. However, we could not find any studies in the literature exploring the relationship between post-MI cardiac remodeling and MHR. MHR was reported to be an important predictor of the slow-flow/no-reflow phenomenon in MI patients[10]. The no-reflow phenomenon was a strong predictor of infarct size and early cardiac remodeling[11]. We assumed that MHR might be an important indicator in cardiac modeling after MI because of the relationship between high monocyte counts and low levels of HDL-C in inflammatory responses. In the present study, we aimed to investigate the prognostic role of MHR at the time of hospital admission in late cardiac remodeling and subsequent 1-year mortality.

METHODS

This study was planned as a multicenter prospective study between June 2015 and June 2020, including Ankara Dr. Nafiz Korez Sincan State Hospital, Ankara Diskapi Training and Research Hospital, Yildirim Beyazit University Atatürk Training and Research Hospital, and Ankara Numune Training and Research Hospital. Assuming an alpha value of 0.05, power of 0.90, and 25% estimated AR rate in line with previous reports[12], the estimated sample size was at least 157 patients in total. The study was performed in accordance with the Declaration of Helsinki and approved by the Ankara Yildirim Beyazit University Faculty of Medicine's Non-Drug Clinical Research Ethics Committee on 24 June 2013 under Decision No. 2013/106. Written informed consent was obtained from all patients.

This research received financial support from the Ministry of Health of the Republic of Turkey (Department of Research, Development, and Health Technology Evaluation) through project 2015/SAGEM-2/001.

Study population

A total of 231 patients older than 18 years of age who were admitted to the hospital with ST-elevation MI (STEMI) for the first time ever and who underwent primary percutaneous coronary intervention within 12 hours after the onset of chest pain was evaluated in this study. STEMI was defined according to the third universal definition of MI[13] and was managed according to the latest guidelines of the European Society of Cardiology[14]. Patients with any mechanical complications (ventricular septal rupture, ventricular free wall rupture or cardiac tamponade, and papillary muscle rupture), cardiogenic shock, or need for an intra-aortic balloon pump and those with a history of silent ischemia/infarct or right coronary artery occlusion, with any kind of systemic inflammatory disease or autoimmune disease, with a history of chronic corticosteroid or anti-inflammatory drugs, with pregnancy or delivery within the past 90 days or currently breastfeeding, and with emergency or elective coronary artery bypass graft scheduled after the angiography procedure were excluded from the study.

Clinical, demographic, laboratory, and radiological findings were recorded in patient files during follow-up in a timely manner. After inclusion of patients in the study, follow-up cardiac magnetic resonance (CMR) imaging was performed at baseline (2 weeks) and 6 months after the index event. All patients were

followed for 1 year for survival after the 6-month CMR imaging. The Global Registry of Acute Cardiac Events (GRACE) risk score was calculated using the official calculator (www.gracescore.org).

Laboratory parameters

Venous blood samples were taken at first admission and analyzed for complete blood count and lipid and cardiac biomarkers. Collected blood samples were centrifuged at 1500 rpm for 10 minutes to measure the determined parameters. Complete blood count parameters were measured with a Sysmex XN-1000 hematology analyzer (Sysmex Corporation, Kobe, Japan). Hemoglobin was measured photometrically. Biochemical parameters were measured with a 7600-120 model automatic biochemical analyzer (Hitachi High Technologies, Tokyo, Japan). Lipid parameters were determined by the homogenous enzymatic colorimetric method with a Hitachi Modular P800 autoanalyzer (Roche Diagnostics Corp., Indianapolis, IN, USA). The following inflammatory indices were calculated: leukocyte count to HDL-C ratio (WHR), monocyte count to HDL-C ratio (MHR), neutrophil count to HDL-C ratio (NHR), platelet count to lymphocyte count ratio (PLR), and neutrophil count to lymphocyte count ratio (NLR).

CMR imaging

All CMR imaging studies were performed with a 3-T scanner (MAGNETOM Skyra, Siemens Medical Systems, Erlangen, Germany). The imaging protocol included the acquisition of 4- and 2-chamber views and cine short-axis sections from the base to the apex of the heart (slice thickness of 6 mm at 10-mm intervals). The indices of left ventricular (LV) systolic function were assessed using a retrospective electrocardiogram-gated turbo-fast low-angle shot (turbo-FLASH) sequence with the following settings: echo time (TE) 1.42 ms, repetition time (TR) 39 ms, flip angle 57°, voxel size 1.67 × 1.67 × 6 mm. Cardiac function and volumes were measured using syngo.via imaging software (Siemens). LV end-diastolic and end-systolic volumes (LVEDV, LVESV) were calculated with short-axis-based planimetry from the basal to the apical level. Stroke volume was calculated as LVEDV minus LVESV, and LV ejection fraction (LVEF) was calculated as follows: LVEF = [(LVEDV - LVESV)/LVEDV] × 100. The definition of AR was based on an increase of LVEDV by ≥ 12% at 6 months post-MI[12,15].

Statistical analysis

The STATA program (StataCorp LLC, College Station, TX, USA) was used for data analysis. Normality testing was performed with the Shapiro–Wilk test. Normal distributions were shown as mean ± standard deviation and non-normal distributions as median (interquartile range [IQR]: 25^{th}-75^{th} percentile). Categorical variables were expressed as numbers and percentages. Student's *t*-test or the Mann-Whitney *U* test were used to compare numerical variables between the groups with and without AR. Chi-square, Yate's correction, and Fisher exact chi-square tests were used for comparisons of categorical data. The relationships between numerical variables were evaluated by Pearson and Spearman's correlation analysis. Changes of CMR parameters were evaluated with paired sample t-tests or Wilcoxon test. The difference of these changes between the groups was evaluated by mixed model repeated measures analysis. Multivariable logistic regression analyses (backward method) were conducted to establish any possible independent predictors of AR. The optimal threshold value of MHR in predicting AR was evaluated by Youden index method in ROC curve analysis. Survival plots were analyzed by Kaplan-Meier method. Values of $p < 0.05$ (*) were considered significant in the statistical analysis.

RESULTS

A total of 231 patients were included in the CMR imaging analysis. The mean age was 53.9 ± 8.7 years and patients were mostly male (88.7%) with a representative risk profile for cardiovascular disease. At 6 months post-MI, 20 patients (23.8%) exhibited AR. The median cardiac troponin I (57 ng/L vs. 46.5 ng/L, p = 0.035), median white blood cell count (11.6 × 10^9/L vs. 10.4 × 10^9/L, p = 0.038), mean monocyte count (0.8 ± 0.2 × 10^9/L vs. 0.7 ± 0.2 × 10^9/L, p = 0.003), median high-sensitivity C-reactive protein (26.4 mg/L vs. 18.9 mg/L, p = 0.045), median WHR (31.8% vs. 27.8%, p = 0.027), median NHR (20.8% vs. 18.3%, p = 0.034), and median MHR (2.2% vs. 1.5%, p < 0.001) levels were higher in the AR group compared to the group without AR. GRACE scores were also higher in the AR group (Table 1).

Table 1. Demographic and clinical findings

Variables	All population (n = 231)	Adverse Cardiac Remodeling		p
		No (n = 176)	Yes (n = 55)	
Gender, n (%)				
Female	26 (11.3)	22 (12.5)	4 (7.3)	0.338
Male	205 (88.7)	154 (87.5)	51 (92.7)	
Age, years	53.9 ± 8.7	54.0 ± 9.2	53.7 ± 7.1	0.846
BMI, kg/m^2	26.8 ± 4.1	26.4 ± 4.1	27.8 ± 4.2	0.314
Smoking, n (%)	122 (52.8)	93 (52.8)	29 (52.7)	0.999
Hypertension, n (%)	99 (42.9)	74 (42.0)	25 (45.5)	0.656
Diabetes mellitus, n (%)	53 (22.9)	40 (22.7)	13 (23.6)	0.889
SBP, mm Hg	124 ± 18.1	124.6 ± 17.5	122.5 ± 19.9	0.528
DBP, mm Hg	76.3 ± 12.4	76.5 ± 12	75.7 ± 13.7	0.730
HR, beat per minute	76.6 ± 16.1	75.9 ± 16.9	78.7 ± 13.4	0.272
LVEF, %	46.7 ± 8.9	46.5 ± 8.6	47.6 ± 9.7	0.423
Door-to-balloon time, min	42.7 ± 8.7	42.3 ± 9.7	43.8 ± 5.4	0.459
Symptom-to-balloon time, min	293.8 ± 52.8	298.1 ± 53.1	282.8 ± 52.5	0.361
Grace score	121 (100-144)	120 (99-141)	136 (101-169)	0.033
IRA, n (%)				
LAD	151 (65.4)	116 (65.9)	35 (63.6)	0.748
Cx	80 (34.6)	60 (34.1)	20 (36.4)	
Pre-PCI TIMI flow				
0	152 (65.8)	113 (64.2)	39 (70.9)	0.432
1	23 (10.0)	16 (9.1)	7 (12.7)	
2	27 (11.7)	22 (12.5)	5 (9.1)	
3	29 (12.6)	25 (14.2)	4 (7.3)	
Post-PCI TIMI flow >2, n (%)	222 (96.1)	170 (96.6)	52 (94.5)	0.585
cTn-I, ng/L	46.8 (38.5-59.7)	46.5 (38-59)	57 (45-68.3)	0.035
CK-MB, IU/L	55 (21-75)	53.5 (20-70)	58 (23.5-75)	0.631
Glucose, mg/dL	112.5 (96-146)	112 (96-139)	115 (97-163)	0.393

(Continues)

Table 1. Demographic and clinical findings *(continued)*

Variables	All population (n = 231)	Adverse Cardiac Remodeling		*p*
		No (n = 176)	Yes (n = 55)	
Pre-PCI TIMI flow				
Hemoglobin, g/dL	14.1 ± 1.5	14.1 ± 1.4	14.1 ± 1.7	0.949
WBC, × 10^9/L	10.8 (8.8-13.5)	10.4 (8.5-12.8)	11.6 (10.1-14.1)	0.038
Neutrophils, × 10^9/L	7.6 (6.2-9.3)	7.5 (6.1-8.6)	7.6 (6.8-9.4)	0.853
Lymphocytes, × 10^9/L	2.3 (1.8-3.1)	2.5 (1.8-3.0)	2.4 (1.7-3.1)	0.514
Monocyte, × 10^9/L	0.7 ± 0.2	0.7 ± 0.2	0.8 ± 0.2	0.003
Platelets, × 10^9/L	280.4 ± 70.7	275.7 ± 65.8	295.1 ± 83.4	0.119
Total cholesterol, mg/dL	197.6 ± 47.5	195.6 ± 45.1	203.9 ± 54.2	0.262
LDL, mg/dL	136 (110-161)	135 (105-157)	142 (119-172)	0.159
HDL, mg/dL	41.9 ± 9.6	42.9 ± 9.6	38.7 ± 8.9	0.005
Triglycerides, mg/dL	142.5 (101-185)	151.5 (104.5-183)	120 (91-198.5)	0.184
Creatinine, mg/dL	1.0 ± 0.2	1.0 ± 0.2	1.0 ± 0.2	0.127
hs-CRP, mg/L	20 (12.3-28.6)	18.9 (10.7-26.8)	26.4 (20-32)	0.045
WHR, %	28.9 (20.7-35.4)	27.8 (20.0-34.7)	31.8 (26.3-39.5)	0.027
NHR, %	18.8 (13.3-23.9)	18.3 (13.0-22.6)	20.8 (13.4-28.0)	0.034
MHR, %	1.7 (1.2-2.1)	1.5 (1.1-2.0)	2.2 (1.7-2.9)	< 0.001
NLR	3.3 (2.5-4.2)	3.3 (2.6-4.1)	3.5 (2.3-4.4)	0.613
PLR	114 (90-153.0)	111.1 (87.4-154.2)	115.5 (97.0-152.3)	0.561
Discharge therapy				
ACE/ARB	225 (97.4)	171 (97.2)	54 (98.2)	0.999
Beta blockers	222 (96.1)	169 (96.0)	53 (96.4)	0.999
Statins, n (%)	227 (98.3)	173 (98.3)	54 (98.2)	0.999

Data are mean ± standard deviation, median (IQR), or number (%). ACE: angiotensin-converting enzyme; ARB: angiotensin II receptor blocker; BMI: body mass index; Cx: circumflex artery; cTn-I: cardiac troponin I; DBP: diastolic blood pressure; HDL: high-density lipoprotein; HR: heart rate; hs-CRP: high-sensitivity C-reactive protein; IRA: infarct-related artery; LAD: left anterior descending artery; LDL: low-density lipoprotein; MHR: monocyte to HDL ratio; NHR: neutrophils to HDL ratio; NLR: neutrophils to lymphocytes ratio; PCI: percutaneous coronary intervention; PLR: platelets to lymphocytes ratio; SBP: systolic blood pressure; TIMI: thrombolysis in myocardial infarction; WBC: white blood counts; WHR: WBC to HDL ratio.

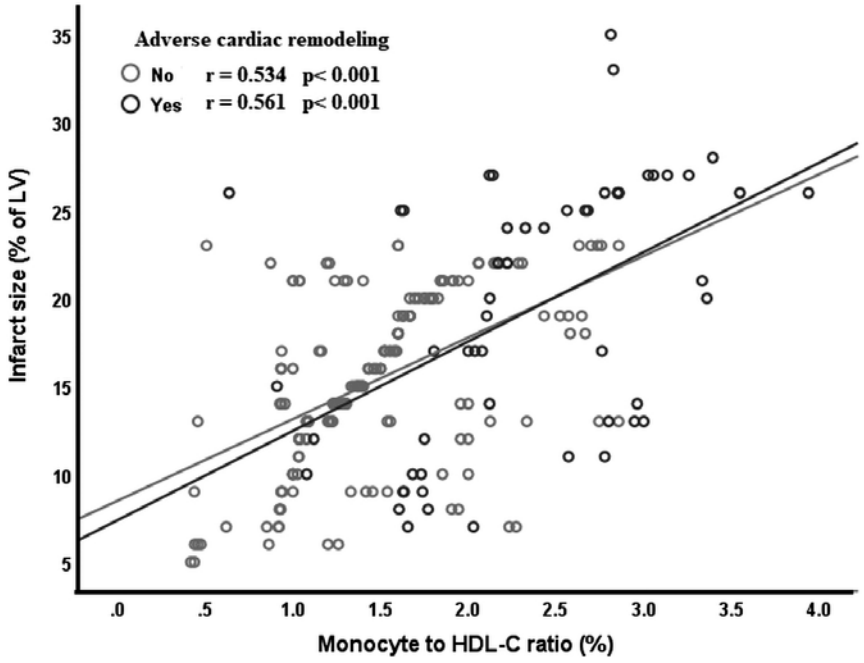
At 2 weeks post-MI, median infarct size was larger in the AR group compared to the group without AR (20% vs. 15% of LV, p < 0.001), with no differences for other baseline CMR imaging parameters. At 6 months post-MI, mean LVEF was lower in the AR group compared to the group without AR (45.6 ±

Table 2. Results of cardiac magnetic resonance imaging

Variables	2 weeks post-MI			6 months post-MI			Δp
	Adverse Cardiac Remodeling		p	Adverse Cardiac Remodeling		p	
	No	Yes		No	No		
	(n = 176)	(n = 55)		(n = 176)	(n = 55)		
LVEF, %	48.3 ± 9.4	49.3 ± 10.1	0.501	51.7 ± 9.2	45.6 ± 10.2	< 0.001	< 0.001
LVEDV, mL	145 (127-165)	158 (129.7-181)	0.247	125 (119-150)	175 (140-214)	< 0.001	< 0.001
LVESV, mL	73 (57-94)	78 (58-105)	0.508	60 (51-80)	87 (65.2-117)	< 0.001	< 0.001
Stroke volume, mL	72.4 ± 17.3	74.4 ± 15.7	0.445	75.9 ± 16.6	73.5 ± 16.4	0.349	0.109
CO, mL/min	4.5 ± 1.1	4.7 ± 1.0	0.231	4.8 ± 1.1	4.6 ± 1.2	0.251	0.144
CI, mL/min/m^2	2.5 ± 0.6	2.6 ± 0.5	0.264	2.7 ± 0.6	2.6 ± 0.5	0.264	0.092
Infarct size, % of LV	15 (12-20)	20 (13-26)	< 0.001	13 (10-18)	18 (10-23)	< 0.001	0.467

Data are mean ± standard deviation or median (IQR). CI: cardiac index; CO: cardiac output; LVEF: left ventricular ejection fraction; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume.

Figure 1. The relationship between MHR and the size of infarct.



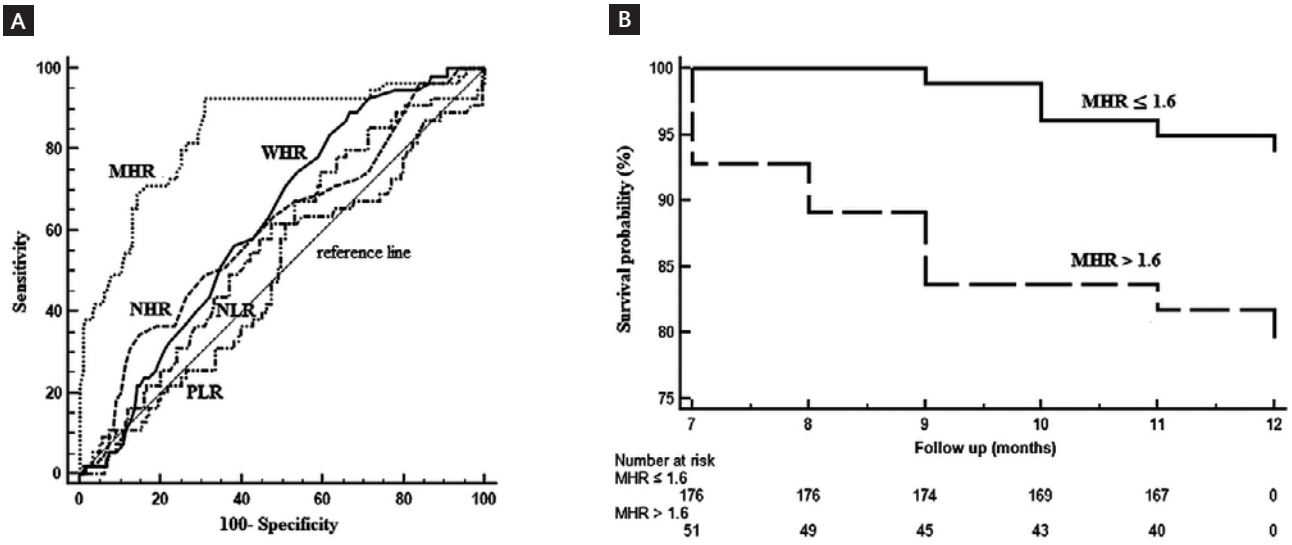
10.2% vs. 51.7 ± 9.2%, p < 0.001), while the median LVEDV (175 mL vs. 125 mL, p < 0.001), median LVESV (120 mL vs. 72 mL, p = 0.003), and median infarct size (18% vs. 13% of LV, p < 0.001) were higher in AR group (Table 2).

A positive correlation was found between MHR and infarct size in groups with and without AR (Fig. 1). MHR (OR: 3.21, p = 0.002) and infarct size (OR: 1.09, p = 0.001) were determined as independent predictors of AR. Accordingly, a 1% increase in MHR

Table 3. Independent predictors of adverse remodeling

Variables	Univariable regression			Multivariable regression		
	OR	95% CI	p	OR	95% CI	p
Infarct size	1.08	1.03-1.13	< 0.001	1.09	1.02–1.15	0.001
Grace score	1.02	1.01-1.04	0.037	–	–	–
cTn-I	1.10	1.01-1.20	0.030	–	–	–
WBC	1.03	1.01-1.05	0.037	–	–	–
Monocyte	17.7	4.43-70.92	0.005	–	–	–
HDL	0.95	0.92-0.98	0.007	–	–	–
WHR	1.02	1.01-1.04	0.031	–	–	–
NHR	1.05	1.01-1.09	0.036	–	–	–
MHR	3.01	1.95-4.65	< 0.001	3.21	1.51-84	0.002
hs-CRP	1.05	1.01-1.10	0.024	–	–	–
				Nagelkerke R^2 = 0.371; p < 0.001		

Figure 2. Diagnostic performance of MHR in predicting AR **(A)** and mortality risk **(B)**. MHR: monocyte to HDL ratio; NHR: neutrophils to HDL ratio; NLR: neutrophils to lymphocytes ratio; PLR: platelets to lymphocytes ratio; WBCs: white blood cells; WHR, WBC to HDL ratio.



increased the risk of AR by 3.21-fold (Table 3). MHR showed superior diagnostic performance compared to other indices in predicting AR and the threshold value of MHR was found to be >1.6% with 92.7% sensitivity and 70.1% specificity (AUC ± SE: 0.84 ± 0.03, 95% CI: 0.78-0.88, PPV: 46.8%, NPV: 96.7%, p < 0.001) (Fig. 2A). Mortality risk was 5.62-fold higher in patients with MHR of >1.6 (HR: 5.62, 95% CI: 2.01-15.70, log-rank p < 0.001) (Fig. 2B).

DISCUSSION

To the best of our knowledge, this is the first study to investigate the role of MHR in cardiac remodeling after MI. Admission MHR values were higher among patients who developed AR. A positive correlation was detected between MHR and infarct size in patients with and without AR. It was also determined that MHR is an independent predictor of AR and has high diagnostic performance in predicting AR.

The recovery process after MI includes a complex array of molecular, cellular, and physiological responses that directly affect the pathological and structural changes in the heart and thus the prognosis. A multistage recovery process initiated by the immune system follows acute MI[16]. Inflammation is of critical importance in cardiac healing during this recovery process[17]. The increased levels of leukocytes, neutrophils, and monocytes and low level of anti-inflammatory HDL-C are important indicators of inflammation. Furthermore, HDL-C can inhibit the activation and migration of leukocytes[18]. Following MI, increased leukocyte migration from the spleen to the heart tissue was associated with an increase in specialized pro-resolving lipid mediator production in the myocardium[19]. Neutrophils, the first line of defense against inflammation, gather in the ischemic zone to scavenge dead cell debris following MI. The release of reactive oxygen species, granular components, and pro-inflammatory mediators by neutrophils may contribute to myocardial injury[20]. It has been suggested that the inhibition of monocytes that are recruited in the infarcted myocardium may improve ventricular function[21]. Monocytes are activated by binding to adhesion molecules expressed during the inflammation process[22,23]. Activated monocytes transform into macrophages, which engulf oxidized LDL-C molecules and turn into foam cells. These foam cells facilitate the release of chemokines, cytokines, and growth factors[24]. HDL-C reduces the activation and adhesion of monocytes, regulates the release of endothelial adhesion molecules, reverses the effects of oxidized LDL-C, and causes vasodilation via the release of nitric oxide[8,25]. This series of events plays an important role in determining the macroscopic structure and geometry of the scarring and has significant effects on cardiac remodeling[16]. Therefore, an index generated by leukocytes and their subtypes and HDL-C may be a more important indicator of cardiac healing after MI.

High values of WHR, NHR, and MHR at admission have been demonstrated to be independent inflammatory markers of thrombus burden, prognosis, and cardiovascular events[26-29]. The prognostic significance of MHR in terms of mortality is consistent with current findings. However, we could not find any study evaluating the relationships between WHR, NHR, and MHR and AR. Current findings indicate that MHR has better diagnostic performance than WHR and NHR in predicting AR. Suzuki et al.[30] suggested that the amount of CD14++CD16+ cells in circulation is higher in patients with atrial fibrillation, and this can be associated with left atrial remodeling. Canpolat et al.[31] suggested that MHR may be an important marker for left atrial remodeling. Values of admission MHR and baseline infarct size were higher in patients with AR, reflecting higher inflammation in the acute phase in infarct areas, but infarct size showed no characteristic healing differences between the groups with and without AR. Nevertheless, there was a positive correlation between infarct size and MHR in both groups and MHR predicted AR regardless of infarct size. These findings might be related to an extremely increased inflammatory response in patients with AR. This is consistent with higher levels of C-reactive protein in patients with AR. In the event of an extremely increased inflammatory response, besides the above mechanisms, higher monocyte counts might negatively affect cardiac recovery[22]. HDL-C molecules can also prevent the activation and propagation of monocytes while inhibiting activated monocytes[32].

Blood parameters are the most commonly available laboratory data during the early period of hospital admission, and they are present universally for the first hour of admission[14]. A biomarker that can be easily evaluated in clinical practice would be of critical importance in the classification of high-risk patients, such as those with AR, which is an important predictor of heart failure and poor prognosis. The threshold value of MHR had high diagnostic performance in detecting patients who developed AR, and it was also determined to be an indicator of high risk in terms of mortality. On the other hand, the threshold value of MHR predicting AR is also consistent with the threshold values of MHR predicting mortality or major adverse cardiac events as shown in a previous meta-analysis[33]. Therefore, MHR, which is an index that does not increase the costs of patient care and is easy to evaluate in different hospitals that treat MI patients, may be a useful biomarker in cardiac remodeling and mortality risk stratification after MI.

One of the important limitations of this study is that MHR was calculated only at the time of admission. MHR levels after the acute phase of MI were not considered. Another important limitation is that the cytokines that play a role in both AR and inflammatory response were not analyzed. Finally, subtypes of monocytes were not evaluated. Doing so may provide

a better understanding of the role of monocytes in the pathophysiology of cardiac remodeling and reveal the prognostic value of MHR levels more clearly.

In conclusion, high admission MHR is an independent predictor of AR at the 6-month follow-up in STEMI patients who have undergone successful primary percutaneous coronary intervention. MHR offers high diagnostic performance for the classification of AR after MI from a prognostic point of view.

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