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Original Article

Correlation between neutrophil-to-lymphocyte ratio after percutaneous coronary intervention and the size of infarction in patients with acute myocardial infarction

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Abstract

Background
Inflammatory processes have a role in the pathogenesis of acute myocardial infarction (AMI), left ventricular remodeling, and repair. Infarct size (IS) is a strong predictor of prognosis following AMI. Several tools are used to predict IS, but most of them are expensive and inaccessible all the time. So, neutrophil-to-lymphocyte ratio (NLR) as a marker of inflammation is an easy and cheap tool to predict IS.

Aim
To assess if post-percutaneous coronary intervention (PCI) NLR can predict IS in patients with AMI.

Patients and methods
The study included 11 125 patients with AMI treated with primary PCI. We used cardiovascular magnetic resonance imaging following PCI to measure IS. We performed a full blood cell count within 1 day before PCI and after it. Patients were classified into two groups regarding NLR by using a cutoff value of 3.88. Patients were divided into high-NLR group ($n = 48$) or low-NLR group ($n = 77$). IS was our primary outcome.

Results
The high-NLR group had significantly higher IS than the low-NLR group (24.82 ± 11.33 vs. 17.20 ± 9.37, $P < 0.001$). We found that post-PCI NLR more than or equal to 3.88 was associated with large-sized infarction (odds ratio 2.997, 95% confidence interval 1.782–5.026, $P < 0.001$). The high-NLR group had significantly higher major adverse cardiac events risk than the low-NLR group ($P = 0.015$, 16.7 vs. 7.8%, hazard ratio 2.678, 95% confidence interval 1.246–5.768).

Conclusions
Post-PCI NLR may be used to predict IS and prognosis of patients with AMI revascularized by PCI.

Keywords: Acute myocardial infarction, infarct size, post-PCI NLR

Introduction

Inflammatory processes have a role in the pathogenesis of atherosclerotic coronary artery disease (CAD) [1,2] and acute myocardial infarction (AMI) [3]. They also affect left ventricular (LV) remodeling and repair. Several inflammatory markers have been investigated to predict cardiovascular outcomes of patients with CAD [4–8]. Neutrophil-to-lymphocyte ratio (NLR) was suggested by some studies as a prognostic factor of clinical outcomes [9–15]. Some studies found that post-percutaneous coronary intervention (PCI) NLR was better than pre-PCI NLR in prediction of the magnitude of myocardial damage [12–14].

Cardiovascular magnetic resonance imaging (CMR) is the golden standard test in measuring infarct size (IS), transmural

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extent, and assessment of microvasculature in AMI [16,17]. It quantifies the magnitude of the areas at risk and salvaged myocardium. IS Bhadran a strong predictor of systolic dysfunction and cardiovascular outcomes [18,19]. Although the association linking NLR to cardiovascular outcomes has been studied by some studies, the association linking NLR after PCI to myocardial injury markers obtained by CMR in patients with AMI is not known.

So, we wanted to examine the relation linking post-PCI NLR with IS in patients with AMI and also the relation between clinical outcomes and post-PCI NLR.

Patients and methods

This study was a prospective cohort study. It was conducted at Cardiology Department, National Heart Institute, Egypt. It was done during the period between September 2016 and August 2020. It included 125 patients with AMI who had primary PCI, had pre-PCI and post-PCI complete blood cells count (CBC), and CMR. The patients were recruited consecutively in the study.

Exclusion criteria of the study

The following were the exclusion criteria:

(1) Patients had not CBC performed within 1 day before and after PCI.
(2) Patients had not CMR performed after PCI.
(3) Patients with contraindications for coronary angiography.

Ethical consideration

Consent was obtained from every patient after explanation of the procedures. Medical research and ethics committee approved the study.

All participants were subjected to complete history taking, as well as full general and local cardiac examination. We performed for all the patients CBC, echocardiography, and CMR during hospitalization.

We followed up the patients at the outpatient clinic after discharge. We contacted the patients by telephone if needed. We recorded and analyzed the clinical events which took place in the 2-year follow-up.

Regarding treatment strategy, the patients were managed according to current guidelines [20–23]. They were given 10 000 IU unfractionated heparin (300 mg), aspirin, and 600 mg clopidogrel as loading doses. Glycoprotein IIb/IIIa inhibitors, balloon predilatation before, and thrombus aspiration were decided in every patient separately. After discharge, the patients were treated according to the relevant guidelines [20–23].

Cardiovascular magnetic resonance imaging

CMR imaging was done by using MR scanner with 1.5 T (MAGNETOM Aera; Siemensm, Siemens Medical Solutions USA, Inc., 40 Liberty Boulevard, Malvern, PA 19355, USA) machine, while the participant was supine, and image acquisition was ECG gated. Late gadolinium enhancement (LGE) images were used to measure IS and microvascular obstruction (MVO) and IS [16]. Cine images were acquired in multiple short axis (throughout the entire LV) and three standard cardiac long-axis views using a balanced steady-state free precession sequence. LGE images were performed using a segmented inversion-recovery gradient-echo sequence 10–15 min after contrast administration in the corresponding imaging planes as for the steady-state free precession images. Inversion delay time was set to null signals from normal myocardium [16]. IS was calculated as the sum of the areas of delayed hyperenhancement in each segment of the short-axis images. The resultant was then multiplied by thickness of the slices for covering the whole LV. Endocardial and epicardial borders were then summed to calculate LV myocardial volume using the same method. IS was expressed as a percentage of the affected LV myocardial volume. The 17-segment model was utilized to subdivide the LV. This CMR protocol for IS quantification has previously been defined and justified in details [16].

MVO was defined as the areas of hypoenhancement in bright segments of LGE images. It was measured by the same manner as previously mentioned [16]. We quantified area at risk (AAR) on T2-weighted images by use of a similar procedure as previously mentioned [16]. From previous parameters, myocardial salvage index (MSI) was calculated as follows: MSI = (AAR-IS)/AAR [16].

Outcomes and definitions

The primary outcome was IS measured by CMR following PCI. Our chief secondary outcome was the 2-year major adverse cardiac events (MACE, a composite of all causes of mortality, MI, and repeated revascularizations). Additional secondary outcomes were MSI, MVO, and AAR obtained by CMR. According to the Academic Research Consortium, every clinical outcome was defined [24]. Any death was considered cardiac except if a specific noncardiac reason could be explained. Recurrent MIs were defined as a rise in cardiac biomarkers more than the upper reference limit for the normal values with symptoms of ischemia or electrocardiographic findings indicating ischemia not associated with the index procedure. Repeated revascularization was needed if clinical or functional evidences of ischemia were found and/or coronary artery stenosis more than 50% in coronary angiography.

Statistical analysis

Recorded data were analyzed using the statistical package for the social sciences, version 20 (SPSS Inc., Chicago, Illinois, USA). The IS was considered large when infarction involved more than or equal to 20% of the total LV mass [25]. Numerical data were explored for normality by checking the data's distribution and using tests of normality (Kolmogorov–Smirnov and Shapiro–Wilk tests). Data were stated as mean, SD, median, and interquartile range values. For parametric data, Student *t* test was used to compare between the two groups. For nonparametric data, Mann–Whitney *U* test was used to compare between the two groups. χ^2 test

of significance was used to compare proportions between qualitative parameters. Linear regression analysis was used to test and estimate the dependence of a quantitative variable based on its relationship to one or more independent variables.

Kaplan–Meier estimates were used to calculate clinical outcomes' cumulative event rates, and level of significance was calculated by log-rank tests. The confidence interval (CI) and hazard ratio (HR) were estimated by Cox proportional hazard models to compare between the two groups regarding clinical outcomes which happened following the PCI. To adjust the baseline differences in the two groups, we used multivariable adjusted Cox proportional hazards regression and inverse-probability-weighted (IPW) analyses. After IPW adjustments, standardized mean differences were within ± 10% across all matched covariates, indicating getting the balance between both groups. The significance level was set at *P* value less than 0.05.

Results

We performed CMR for all the patients within 3.50 (interquartile range 2.78–4.74) days following PCI. We analyzed receiver operating characteristic curve to discover which leukocyte and differential count precisely predicted large IS (≥20% out of the whole LV mass). We compared total leukocyte count and NLR after and before PCI. NLR after PCI had the highest discriminative power in predicting a large IS with C-index (0.739, 95% CI 0.679–0.798), and NLR after PCI had an optimal cut off value = 3.88. Linear regression analysis found that NLR after PCI is significantly correlated to IS (r^2: 0.106, $P < 0.001$) (Fig. 1).

Clinical characteristics are shown in Table 1. Angiographic characteristics are shown in Table 2. Individuals of high-NLR group were elder and suffered from previous MI and PCI compared with individuals of the low-NLR group. Individuals of the high-NLR group had more frequently ST-segment elevation MI in comparison with those of the low-NLR group. There were no significant differences in other baseline characteristics.

Table 3 shows the results of CMR, echocardiography, and laboratory. The IS was notably higher in high-NLR group than low-NLR group ($P < 0.001$, 24.82 ± 11.33 vs. 17.20 ± 9.37), as well as AAR ($P < 0.001$, 41.92 ± 16.79 vs. 30.39 ± 15.55) and MVO [3 (0–9.58) vs. 0 (0–3.81), $P < 0.001$]. Other essential markers of infarction's severity were significantly greater in the high-NLR patients also. The high-NLR group had lower MSI than the low-NLR group ($P = 0.258$, 41.51 ± 17.41 vs. 43.98 ± 18.03), yet the differences were not significant statistically. Post-PCI NLR more than or equal to 3.88 was the most powerful predictor of large IS (odds ratio: 2.997, 95% CI: 1.782–5.026, $P < 0.001$) in multivariate logistic regression analyses (Table 4).

Median follow-up duration was 738 days. The high-NLR group had higher cumulative incidence of MACE than the low-NLR group ($P = 0.015$, 16.7 vs. 7.8%, HR 2.678, 95% CI 1.246–5.768) (Table 5 and Fig. 2). After adjusting baselines by IPW multivariable and Cox regression adjustment, post-PCI NLR more than or equal to 3.88 was consistently associated with a higher risk of MACE (Table 5). The higher risk of MACE in patients with AMI with high post-PCI NLR was mainly driven by the higher rates of hard end points, including all-cause death (4.2 vs. 0%, HR 8.724, 95% CI 01.020–74.685, $P = 0.052$) and MI (4.2 vs. 1%, HR 8.899, 95% CI 1.040–76.189, $P = 0.050$) (Table 5 and Fig. 2).

Discussion

We tested in this study the relationship linking post-PCI NLR with IS in individuals with AMI. We found the followings: first, NLR after PCI showed the greatest capability to distinguish large IS; second, high-NLR after PCI was correlated with

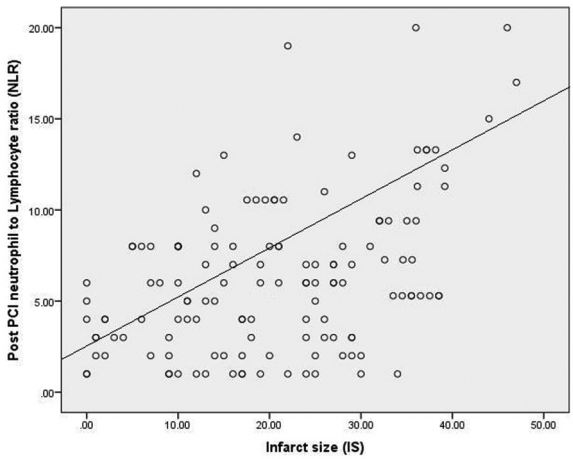

Figure 1: Post-PCI neutrophil-to-lymphocyte ratio (NLR) is correlated with infarct size (IS). ($r^2 = 0.106$; $P < 0.001$ HS). PCI, percutaneous coronary intervention.

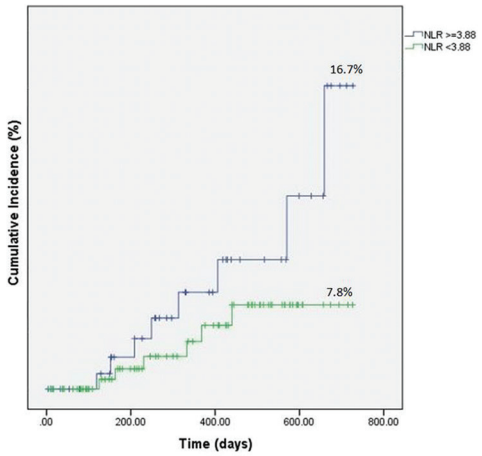

Figure 2: Cumulative incidence of major adverse cardiac events (MACE) within 2 years following PCI. A comparison between the cumulative incidence of MACE in the high-neutrophil-to-lymphocyte ratio (NLR) group and low-NLR group by use of Kaplan–Meier curves. PCI, percutaneous coronary intervention.

Table 1: Patients' baseline characteristics

Parameters	Total (*n*=125)	NLR ≥ 3.88 (*n*=48)	NLR < 3.88 (*n*=77)	*P*
Demographics				
Age (years)	52.74±12.67	55.72±14.01	51.60±11.64	0.049*
Male	102 (81.6)	39 (81.3)	63 (81.8)	0.774
Female	23 (18.4)	9 (18.7)	14 (18.2)	
BMI (kg/m^2)	25.34±3.50	24.72±3.81	25.65±3.19	0.026*
Cardiovascular risk factor				
Hypertensive	53 (42.4)	22 (45.8)	31 (40.3)	0.464
Diabetes mellitus	27 (21.6)	13 (27.1)	14 (18.2)	0.096
Dyslipidemia	21 (16.8)	7 (14.6)	14 (18.2)	0.298
History of myocardial infarction	6 (4.8)	4 (8.3)	2 (2.6)	0.022*
Previous PCI	9 (7.2)	6 (12.5)	3 (3.9)	0.012*
Previous cerebrovascular accidents	5 (4.0)	2 (4.2)	3 (3.9)	0.777
Laboratory results				
NT-proBNP (pg/ml)	215.89 (IQR: 57.58-831.73)	210.22 (IQR: 71.17-1042.67)	218.98 (IQR: 52.12-717.60)	0.268
Leukocytes (×10^3/µl)	10.20±3.19	11.74±3.50	9.27±2.68	<0.001**
Hemoglobin (g/dl)	13.91±1.96	13.60±2.06	14.11±1.85	0.014*
Platelet, (10^3/µl)	213.00±46.76	215.79±50.06	211.25±44.70	0.439
Clinical presentations				
Non-ST-elevation myocardial infarction	34 (27.2)	7 (14.6)	27 (35.1)	<0.001**
Time of door-to-balloon (min)	701.95 (IQR: 198.28-1326.13)	492.86 (IQR: 160.17-1283.38)	721.00 (IQR: 198.28-1347.55)	0.501
Symptoms-to-balloon time (min)	1334.88 (IQR: 642.72-1949.58)	1303.47 (IQR: 562.38-2542.56)	1378.66 (IQR: 709.16-1949.58)	0.788
ST-elevation myocardial infarction	91 (72.8)	41 (85.4)	50 (64.9)	<0.001**
Time of door-to-balloon (min)	72.10 (IQR: 54.59-84.46)	72.10 (IQR: 52.53-83.95)	72.10 (IQR: 55.11-84.46)	0.864
Symptoms-to-balloon time (min)	203.94 (IQR: 119.48-422.30)	208.06 (IQR: 104.55-424.88)	203.94 (IQR: 126.69-435.69)	0.628
Medications after percutaneous coronary intervention				
Aspirin	125 (100.0)	48 (100.0)	77 (100.0)	1.000
P2Y12 inhibitors	125 (100.0)	48 (100.0)	77 (100.0)	1.000
Beta-blocker	113 (90.4)	43 (89.6)	70 (90.9)	0.774
ACEI or ARBs	99 (79.2)	36 (75.0)	63 (81.8)	0.165
Statins	120 (96.0)	45 (93.8)	75 (97.4)	0.278

Data are presented as mean±SD and *n* (%). ACEI, angiotensin-converting enzyme inhibitors; ARBs, angiotensin receptor blockers; IQR, interquartile range; NLR, neutrophil-to-lymphocyte ratio; NT-proBNP, N-terminal pro-brain natriuretic peptide; PCI, percutaneous coronary intervention. *Significant, **Highly significant.

Table 2: Characteristics of the angiography and percutaneous coronary intervention of acute myocardial infarction patients

Variables	Total (*n*=125)	NLR ≥ 3.88 (*n*=48)	NLR < 3.88 (*n*=77)	*P*
Infarct related artery				
Left anterior descending coronary artery	57 (45.6)	25 (52.1)	32 (41.6)	0.312
Left circumflex coronary artery	20 (16.0)	7 (14.6)	13 (16.9)	
Right coronary artery	47 (37.6)	16 (33.3)	31 (40.3)	
Left main coronary artery	1 (0.8)	0	1 (1.3)	
Diseased vessels' number				
1	66 (52.8)	23 (47.9)	43 (55.8)	0.276
2	41 (32.8)	18 (37.5)	23 (29.9)	
3	18 (14.4)	7 (14.6)	11 (14.3)	
Multivessels disease	59 (47.2)	25 (52.1)	34 (44.2)	0.156
TIMI flow≤I pre-PCI	94 (75.2)	39 (81.3)	55 (71.4)	0.068
TIMI flow III post-PCI	117 (93.6)	44 (91.7)	73 (94.8)	0.389
Thrombus aspiration	67 (53.6)	28 (58.3)	39 (50.6)	0.205
Implanted stents' number	1.24±0.72	1.13±0.72	1.24±0.62	0.908
Stents diameter (mm)	3.30±0.52	3.19±0.52	3.30±0.62	0.191
Stents diameter < 3 mm	49 (39.2)	22 (45.8)	27 (35.1)	0.063
Stents length (mm)	32.55±17.61	31.83±17.41	32.96±17.72	0.675

Data are presented as mean±SD and *n* (%). NLR, neutrophil-to-lymphocyte ratio; PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction.

Table 3: IS assessment by using laboratory, echocardiographic, and cardiovascular magnetic resonance data

Variables	Total (n=125)	NLR ≥ 3.88 (n=48)	NLR < 3.88 (n=77)	P
Laboratory profiles following percutaneous coronary intervention				
Troponin I peak (ng/ml)	46.7 (IQR: 12.26-119.69)	77.6 (IQR: 22.97-191.07)	36.9 (IQR: 8.24-82.71)	<0.001**
Peak CK-MB (ng/ml)	144.5 (IQR: 49.23-256.37)	199.0 (IQR: 82.40-323.42)	103.8 (IQR: 40.48-217.23)	<0.001**
Echocardiography				
Ejection fraction	53.77±11.23	50.57±11.85	55.72±10.30	<0.001**
Wall motions score index	1.4 (IQR: 1.24-1.75)	1.5 (IQR: 1.24-1.85)	1.3 (IQR: 1.13-1.65)	<0.001**
Cardiac MRI				
Area at risk (% of LV)	34.81±17.00	41.92±16.79	30.39±15.55	<0.001**
Infarct size (% of LV)	20.09±10.82	24.82±11.33	17.20±9.37	<0.001**
Myocardial salvage index	43.05±17.82	41.51±17.41	43.98±18.03	0.258
Microvascular obstruction (per volume)	0.9 (IQR: 0.00-5.56)	3.0 (IQR: 0.00-9.58)	0.0 (IQR: 0.00-3.81)	<0.001**
LV EDV	152.75±38.32	154.19±42.75	151.93±35.33	0.667
LV ESV	75.50±33.89	82.19±41.51	71.38±27.50	0.014*
Mass of LV (g)	111.96±28.22	111.14±30.39	112.48±26.88	0.763
LV ejection fraction	53.66±11.12	50.26±12.05	55.83±9.99	<0.001**
LV stroke volume	77.04±17.41	72.00±16.48	80.24±17.20	<0.001**
LV cardiac output	5.25±1.13	5.15±1.13	5.36±1.13	0.187

Data are presented as mean±SD. CK-MB, creatine kinase-myocardial band; EDV, end diastolic volume; ESV: end systolic volume; IQR, interquartile range; NLR, neutrophil-to-lymphocyte ratio and SV, LV, left ventricle. *Significant, **Highly significant.

Table 4: Independent predictors of large infarct size (≥20%) in patients with acute myocardial infarction

	Odds ratio (OR) (95% CI)*
ST-segment elevation myocardial infarction	1.864 (1.051-3.337)
Post-PCI NLR ≥ 3.88	2.997 (1.782-5.026)
Male	2.039 (1.040-3.986)
BMI ≥ 25 kg/m^2	0.567 (0.340-0.948)

Adjusted variables were male, age, hypertension, diabetes mellitus, previous myocardial infarctions, previous PCI, ST-segment elevation myocardial infarctions, multivessel disease, anterior infarctions, BMI more than or equal to 25 kg/m^2, and post-PCI NLR more than or equal to 3.88. CI, confidence interval; NLR, neutrophil-to-lymphocyte ratio; PCI, percutaneous coronary intervention and TIMI, thrombolysis in myocardial infarction. *C-index of the logistic regression model for large infarct size was 0.739 (95% CI 0.679-0.798).

greater risks of MACE, bigger IS, and MVO. This correlation was strong after adjustment of baselines by use of IPW adjustments and multivariable adjusted Cox regression.

In spite of modern advances in revascularization treatment, AMI is still the most common cause of mortality and morbidity all over the world [26]. So, many clinical trials were done to predict prognosis and improve outcome following AMI [18,27,28]. Inflammatory processes play an important task in the pathogenesis of atherosclerotic CAD [1,2] and AMI [3]. Some studies tested the relations between different markers of inflammation and clinical outcomes in individuals with AMI [4–15]. An earlier study found that differential leukocytes count predicted outcomes better than total leukocytes count [29]. NLR exhibited the greatest power in predicting clinical outcomes among different leukocyte indices and differential count [29]. Park *et al.*[12] demonstrated that NLR after PCI was more significantly correlated with death than NLR before PCI. This can be explained by that the passed time after AMI is too brief for pre-PCI leukocytes profile to completely exhibit the magnitude of the damage of the myocardium. Moreover, temporal proximity may be a reason as CMR was performed following PCI. Furthermore, post-PCI leukocyte profile may also exhibit periprocedural damage of the myocardium [14]. Similar to previous studies [12,14,29], we found the risk of MACE was higher in patients with AMI with a high post-PCI NLR than patients having a low post-PCI NLR. The explanation of this correlation is that besides their importance in the repairing process of the infarcted myocardium, leukocytes have a role in expansion of infarction [29,30]. Following infarctions, neutrophils produce enzymes, superoxide radicals, and different metabolites that cause disruption of plaques and cause expansion of the infarction. Aggregation of neutrophils and platelets causes damage to microvessels, which promotes expansion of infarct [29]. Moreover, lymphocytes are sequestered and trapped within the microvasculature of the myocardium and produce mediators of inflammation, adding to further leukocyte infiltration and damage of the myocardium [31]. On this background, the results of the present study advocate that NLR after PCI can predict clinical outcomes following AMI. CMR is the golden standard test for evaluation of systolic performance and cardiac volumes [16]. Moreover, it is the most precise tool for visualization and quantification of postinfarction parameters in individuals with AMI [18]. Among the most important postinfarction parameter is IS. IS is significantly correlated to clinical outcomes [32]. Besides, it can offer an incremental prognostic value added to LV ejection fraction, a well-known predictor of AMI clinical outcomes [18]. Because ejection fraction is affected by both nonviable myocardium and viable stunned myocardium, parameters of CMR are specific markers in distinguishing the magnitude of irreversible damage of the myocardium [33]. Although some trials evaluated the

Table 5: Two-year clinical outcomes in patients with acute myocardial infarction

	NLR ≥ 3.88 (*n*=48)	NLR < 3.88 (*n*=77)	Unadjusted		Adjusted		IPW-adjusted	
			HR (95% CI)	*P*	HR (95% CI)	*P*	HR (95% CI)	*P*
MACE	8 (16.7)	6 (7.8)	2.678 (1.246-5.768)	0.015*	2.451 (1.040-5.325)	0.029*	1.741 (1.030-2.946)	0.049*
All-cause death	2 (4.2)	0 (0.0)	8.724 (1.020-74.685)	0.052	6.273 (0.721-54.363)	0.103	2.730 (0.989-7.478)	0.06
Myocardial infarction	2 (4.2)	1 (1.3)	8.899 (1.040-76.189)	0.050*	6.901 (0.773-61.491)	0.09	4.439 (0.906-21.815)	0.073
Any revascularization	4 (8.3)	4 (5.2)	1.833 (0.690-4.882)	0.255	1.545 (0.546-4.378)	0.454	1.339 (0.670-2.647)	0.465

Data are presented as *n* (%). Adjusted variables were age, sex, diabetes mellitus, prior myocardial infarction, ST-segment elevation myocardial infarction, anterior infarction, TIMI flow grade 0 before PCI. CI, confidence interval; HR, hazards ratio; IPW, inverse-probability-weighted; MACE, major adverse cardiac events; NLR, neutrophils-to-lymphocytes ratio; PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction. *Significant.

relationship between leukocytes and magnitude of infarct quantified by CMR [4,34–36], limited information is available about the relationship between IS and post-PCI NLR. So, we assessed if high post-PCI NLR values are correlated to larger IS obtained by CMR in AMI patients. The high post-PCI NLR was found to be correlated with bigger IS and MVO as obtained by many tools, like CMR, cardiac enzymes, and echocardiography in individuals with AMI treated with PCI. Although the MSI was lower in the post-PCI high-NLR group, the difference was not significant statistically. Our study recommends that post-PCI NLR can predict IS and prognosis in individuals with AMI treated by PCI. To validate prognostic implication of post-PCI NLR, we conducted a comprehensive assessment to find an association between post-PCI NLR and different markers of IS like echocardiographic and laboratory findings and also MACE. Moreover, the robustness of these results was supported by sensitivity analyses like multivariable regressions and IPW adjustments. Thus, our results recommend post-PCI NLR as a predictor of prognosis and IS in future studies that aim at IS reduction.

Conclusions

High values of post-PCI NLR were correlated with larger IS obtained by CMR. They are also correlated with the risk of MACE in individuals with AMI who were revascularized by PCI. Post-PCI NLR, which is a simply measured and widely available biomarker, can provide a useful risk stratification and prognostic information for patients with AMI revascularized by PCI.

Limitations

The major limitations of the present study are that it is based on a single-center experience and may be limited by the small number of patients.

Conflicts of interest

There are no conflicts of interest.

References

1. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH Inflammation, aspirin, and the risk of cardiovascular disease in apparently healthy men. N Engl J Med 1997; 336; 973–979.
2. Ross R. Atherosclerosis – an inflammatory disease. N Engl J Med 1999; 340:115–126.
3. Seropian IM, Toldo S, Van Tassell BW, Abbate A. Anti-inflammatory strategies for ventricular remodeling following ST-segment elevation acute myocardial infarction. J Am Coll Cardiol 2014; 63:1593–1603.
4. Song YB, Chung SM, Hahn JY, Choi SH, Choi JH, Lee SH, *et al*. Impact of white blood cell count on myocardial salvage and infarct size and clinical outcomes in patients undergoing primary percutaneous coronary intervention for ST-segment elevation myocardial infarction: a magnetic resonance imaging study. American Journal of Cardiology 2013; 111:6B-7B.
5. Shah B, Baber U, Pocock SJ, Krucoff MW, Ariti C, Gibson CM, *et al*. White blood cell count and major adverse cardiovascular events after percutaneous coronary intervention in the contemporary era: insights from the PARIS Study (Patterns of Non-Adherence to Anti-Platelet Regimens in Stented Patients Registry). Circ Cardiovasc Interv 2017; 10:e004981.
6. Dhillon OS, Narayan HK, Khan SQ, Kelly D, Quinn PA, Squire IB, *et al*. Pre-discharge risk stratification in unselected STEMI: is there a role for ST2 or its natural ligand IL-33 when compared with contemporary risk markers? Int J Cardiol 2013; 167:2182-2188.
7. Lindmark E, Diderholm E, Wallentin L, Siegbahn A. Relationship between interleukin 6 and mortality in patients with unstable coronary artery disease: effects of an early invasive or noninvasive strategy. JAMA 2001; 286:2107–2113.
8. Ørn S, Manhenke C, Ueland T, Damås JK, Mollnes TE, Edvardsen T, *et al*. C-reactive protein, infarct size, microvascular obstruction, and left-ventricular remodelling following acute myocardial infarction. Eur Heart J 2009; 30:1180-1186.
9. Duffy BK, Gurm HS, Rajagopal V, Gupta R, Ellis SG, Bhatt DL. Usefulness of an elevated neutrophil to lymphocyte ratio in predicting long-term mortality after percutaneous coronary intervention. Am J Cardiol 2006; 97:993-996.
10. Tamhane UU, Aneja S, Montgomery D, Rogers EK, Eagle KA, Gurm HS. Association between admission neutrophil to lymphocyte ratio and outcomes in patients with acute coronary syndrome. Am J Cardiol 2008; 102:653-657.
11. Azab B, Zaher M, Weiserbs KF, Torbey E, Lacossiere K, Gaddam S, *et al*. Usefulness of neutrophil to lymphocyte ratio in predicting short-and long-term mortality after non–ST-elevation myocardial infarction. Am J Cardiol 2010; 106:470-476.
12. Park JJ, Jang HJ, Oh IY, Yoon CH, Suh JW, Cho YS, *et al*. Prognostic value of neutrophil to lymphocyte ratio in patients presenting with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Am J Cardiol 2013; 111:636-642.
13. Sen N, Afsar B, Ozcan F, Buyukkaya E, Isleyen A, Akcay AB, *et al*. The neutrophil to lymphocyte ratio was associated with impaired myocardial perfusion and long term adverse outcome in patients with ST-elevated myocardial infarction undergoing primary coronary intervention. Atherosclerosis 2013; 228:203-210.
14. Bressi E, Mangiacapra F, Ricottini E, Cavallari I, Colaiori I, Di Gioia G, *et al*. Relation of neutrophil to lymphocyte ratio with periprocedural myocardial damage in patients undergoing elective percutaneous coronary intervention. Am J Cardiol 2016; 118:980-984.
15. Monteiro Júnior JG, Torres DD, da Silva MC, Martins CM, da Silva IK, do Nascimento ME, *et al*. Prognostic value of hematological parameters

in patients with acute myocardial infarction: Intrahospital outcomes. PloS one 2018; 13:e0194897.
16. Perazzolo Marra M, Lima JA, Iliceto S. MRI in acute myocardial infarction. Eur Heart J 2011; 32:284–293.
17. Wong DT, Richardson JD, Puri R, Nelson AJ, Bertaso AG, Teo KS, *et al*. The role of cardiac magnetic resonance imaging following acute myocardial infarction. Eur Radiol 2012; 22:1757-1768.
18. Eitel I, de Waha S, Wöhrle J, Fuernau G, Lurz P, Pauschinger M, *et al*. Comprehensive prognosis assessment by CMR imaging after ST-segment elevation myocardial infarction. J Am Coll Cardiol 2014; 64:1217-1226.
19. Larose E, Rodés-Cabau J, Pibarot P, Rinfret S, Proulx G, Nguyen CM, *et al*. Predicting late myocardial recovery and outcomes in the early hours of ST-segment elevation myocardial infarction: Traditional measures compared with microvascular obstruction, salvaged myocardium, and necrosis characteristics by cardiovascular magnetic resonance. J Am Coll Cardiol 2010; 55:2459-2469.
20. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, *et al*. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018; 39:119-177.
21. O'gara PT, Kushner FG, Ascheim DD, Casey DE, Chung MK, De Lemos JA, *et al*. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013; 61:e78-e140.
22. Amsterdam EA, Wenger NK, Brindis RG, Casey DE, Ganiats TG, Holmes DR, *et al*. 2014 AHA/ACC guideline for the management of patients with non–ST-elevation acute coronary syndromes: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014; 64:e139-e228.
23. Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, *et al*. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Kardiologia Polska (Polish Heart Journal) 2015; 73:1207-1294.
24. Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. Circulation 2007; 116:2634–2653.
25. Jang WJ, Yang JH, Song YB, Chun WJ, Oh JH, Park YH, *et al*. Clinical Significance of postinfarct fever in st-segment elevation myocardial infarction: A cardiac magnetic resonance imaging study. J Am Heart Assoc 2017;6:e005687.
26. Reed GW, Rossi JE, Cannon CP. Acute myocardial infarction. Lancet 2017; 389:197–210.
27. Morice MC, Serruys PW, Sousa JE, Fajadet J, Ban Hayashi E, Perin M, *et al*. A randomized comparison of a sirolimus-eluting stent with a standard stent for coronary revascularization. N Engl J Med 2002; 346:1773-1780.
28. Giugliano GR, Giugliano RP, Gibson CM, Kuntz RE. Meta-analysis of corticosteroid treatment in acute myocardial infarction. Am J Cardiol 2003; 91:1055–1059.
29. Horne BD, Anderson JL, John JM, Weaver A, Bair TL, Jensen KR, *et al*. Intermountain Heart Collaborative (IHC) Study Group. Which white blood cell subtypes predict increased cardiovascular risk? J Am Coll Cardiol 2005; 45:1638-1643.
30. Frangogiannis NG. The immune system and the remodeling infarcted heart: cell biological insights and therapeutic opportunities. J Cardiovasc Pharmacol 2014; 63:185–195.
31. Boag SE, Das R, Shmeleva EV, Bagnall A, Egred M, Howard N, *et al*. T lymphocytes and fractalkine contribute to myocardial ischemia/reperfusion injury in patients. J Clin Invest 2015; 125:3063-3076.
32. Stone GW, Selker HP, Thiele H, Patel MR, Udelson JE, Ohman EM, *et al*. Relationship between infarct size and outcomes following primary PCI: Patient-level analysis from 10 randomized trials. J Am Coll Cardiol 2016; 67:1674-1683.
33. Wu E, Ortiz JT, Tejedor P, Lee DC, Bucciarelli-Ducci C, Kansal P, *et al*. Infarct size by contrast enhanced cardiac magnetic resonance is a stronger predictor of outcomes than left ventricular ejection fraction or end-systolic volume index: prospective cohort study. Heart 2008; 94:730-736.
34. Palmerini T, Brener SJ, Genereux P, Maehara A, Della Riva D, Mariani A, *et al*. Relation between white blood cell count and final infarct size in patients with ST-segment elevation acute myocardial infarction undergoing primary percutaneous coronary intervention (from the INFUSE AMI trial). Am J Cardiol 2013; 112:1860-1866.
35. Husser O, Bodi V, Sanchis J, Nunez J, Mainar L, Chorro FJ, *et al*. White blood cell subtypes after STEMI: temporal evolution, association with cardiovascular magnetic resonance—derived infarct size and impact on outcome. Inflammation 2011; 34:73-84.
36. Reindl M, Reinstadler SJ, Feistritzer HJ, Klug G, Tiller C, Mair J, *et al*. Relation of inflammatory markers with myocardial and microvascular injury in patients with reperfused ST-elevation myocardial infarction. Eur Heart J Acute Cardiovasc Care 2017; 6:640-649.