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The relationship between neutrophil percentage-to-albumin ratio and infarct related artery patency in patients with acute coronary syndrome

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ABSTRACT

Aims: One of the leading causes of death and disease burden globally is coronary heart disease (CHD). The primary cause of acute coronary syndrome (ACS) is atherosclerotic plaque rupture and thrombus development. The prognosis of ACS patients is also linked to atherosclerotic plaques, inflammatory cell infiltration (lymphocytes, monocytes, and neutrophils), and inflammation indicators. Low serum albumin (SA) levels have been linked to death in ACS patients in earlier research. In our study, we aimed to investigate the association of neutrophil percentage-to-albumin ratio (NPAR) with infarct-related adverse cardiac events (MACE) and infarct-related coronary artery patency (IRA).

Methods: The NPAR ratio was calculated based on the past laboratory findings of patients admitted with ACS who underwent coronary angiography (CAG), which were registered in the data system at the time of admission. A total of 87 patients were included in the study. Of these patients, 62 (71%) were non-patent and 25 (29%) were patent IRA patients.

Results: NPAR was significantly higher in the non-patent group (19.22±3.14 and 17.14±2.78 p=0.004). In multivariable logistic regression analysis, NPAR [p=0.027, odds ratio (OR): 0.787, 95% confidence intervals (CIs): 0.637-0.974] levels were found to be independent predictors of patent IRA. As revealed by the ROC curve analysis, the cut-off value of 17.88 for NPAR predicted the non-patent IRA with a sensitivity of 64% and specificity of 64% (AUC: 0.681; CIs: 0.588-0.809; p=0.008) NPAR was significantly higher in the MACE group (22.83±3.85 and 17.95±2.49 p<0.001).

Conclusion: In conclusion, inflammatory markers have been and are being used as predictive parameters for cardiovascular diseases in many studies. In our study, we focused on neutrophils and albumin. These findings revealed that NPAR is a independent predictor of IRA patency and long term mortality. We also indicated that NPAR may have a predictive role for mortality in the long-term follow-up of ACS patients.

Keywords: Neutrophil, albumin, infarct related artery, acute coronary syndrome

INTRODUCTION

One of the leading causes of death and disease burden globally is coronary heart disease (CHD). One severe form of CHD with a high morbidity and mortality rate is ACS. The primary cause of ACS is atherosclerotic plaque rupture and thrombus development. The prognosis of ACS patients is also linked to atherosclerotic plaques, inflammatory cell infiltration (lymphocytes, monocytes, and neutrophils), and inflammation indicators. In recent years, neutrophil/lymphocyte ratio (NLR) is one of the most important and widely used inflammatory markers in the prognosis of ACS. A prognostic biomarker for cardiovascular, viral, and cancer conditions, NLR is an indication of inflammation.[1,2] It has been demonstrated that NLR, an indicator of inflammation, can forecast in-hospital death in patients with ACS.[3]

Serum albumin (SA) is linked to both acute and chronic inflammatory reactions, and elevated inflammation lowers SA levels in addition to suggesting nutritional status.[4] Low SA levels have been linked to death in ACS patients in earlier research.[5]

Neutrophil percentage-to-albumin (NPAR), calculated as neutrophil percentage numerator divided by serum albumin concentration, can amplify the changes of these two accessible evaluation parameters. According to one study, the NPAR at admission was an independent predictor of in-hospital mortality for patients with acute ST segment elevation myocardial infarction (STEMI).[6] In critically sick patients with coronary artery disease (CAD), a higher NPAR level was strongly associated with higher 30-day, 90-day, and 365-day all-cause death, according to another study.[7]

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In our study, we aimed to investigate the association of NPAR with infarct-related adverse cardiac events (MACE) and infarct-related coronary artery patency (IRA) in patients aged <65 years.

METHODS

Ethics

The study was carried out with the permission of Bandırma Onyedi Eylül University Health Sciences Non-interventional Researches Ethics Committee (Date: 22.04.2024, Decision No: 2024-4). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Design and Population

We retrospectively evaluated patients with ACS from Bandırma Research and Training Hospital database who were admitted to the department of cardiology. STEMI was defined as typical symptoms (chest pain >30 minutes) and ST elevation (greater than or equal to 1 mm in at least consecutive 2 leads on ECG) and NSTEMI was defined patients with chest pain, high cardiac troponin (cTn) levels and ST changes on ECG. Exclusion criteria were active infectious disease, active carcinoma, hematologic proliferative diseases, chronic inflammatory disease, coronary artery bypass graft (CABG), ACS and ischemic stroke in the last 3 months, active hepatobiliary diseases, steroid treatment for autoimmune disease, pulmonary embolism, pulmonary hypertension, malignancy, age >65 years and patients with inaccessible laboratory parameters. Finally, a total of 87 patients were included in the study (Table 1).

Laboratory Analysis and NPAR Calculation

Peripheral blood samples obtained from patients at the time of admission were used. These data were obtained retrospectively from the hospital automation system. NPAR was calculated by dividing the neutrophil percentage share (i.e. 66% was recorded 66) by albumin using the same blood samples taken at admission. Total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides and high-sensitivity C-reactivity protein levels were obtained from fasting blood samples taken the morning after admission. Serum levels of cardiac markers such as cTn and creatinine kinase-MB (CK-MB) were measured at admission and every 24 hours until peaks occurred, but values taken at the time of admission were used. Demographic, clinical and laboratory data of the patients were obtained from the hospital registry system.

Data Collection and Definition

Demographic information and cardiovascular risk factors such as previous CHD, hyper blood pressure, diabetic mellitus, dyslipidemia and smoking history were retrospectively collected from medical records. Systemic blood pressure, diastolic blood pressure, heart rate and Killip class were defined as the initial data recorded at admission. Simpson's method was used to calculate left ventricular ejection fraction (LVEF) for quantitative assessment of left ventricular systolic function before discharge. On 365-days, major adverse cardiac events (MACE) were defined as mortality due to reinfarction, stroke, malignant arrhythmia and heart failure.

Table 1. Characteristics of study patients according to the IRA

Variables	All (87)	IRA non-patent (62)	IRA patent (25)	p
Age	61.1±11.6	61.2±11.0	60.9±13.1	0.907
Male gender, n (%)	65 (74.7)	46 (74.2)	19 (76.0)	0.861
Hypertension, n (%)	46 (52.9)	30 (48.4)	16 (64.0)	0.187
Diabetes mellitus, n (%)	20 (23.0)	16 (25.8)	4 (16.0)	0.325
HL	39 (44.8)	29 (46.8)	10 (40.0)	0.565
CAD	30 (34.5)	21 (33.9)	9 (36.0)	0.850
LVEF	45.0 (40.0-55.0)	45.0 (35.0-55.0)	50 (45.0-55.0)	0.168
WBC	10.9 (9.0-13.4)	10.9 (9.3-14.2)	9.9 (7.3-11.9)	0.024
Hgb	13.7±2.1	13.7±2.0	13.8±2.3	0.881
Neutrophil count, x1000/ul	8.09 (5.98-10.18)	8.71 (6.62-10.70)	6.61 (4.75-8.80)	0.009
Lymphocyte count, x1000/ul	1.93 (1.42-2.57)	1.98 (1.44-2.62)	1.64 (1.26-2.53)	0.558
PLT	244.5 (204.0-279.0)	245.0 (201.0-280.0)	241.5 (212.0-268.0)	0.488
Albumin	4.03 (3.81-4.30)	4.00 (3.70-4.30)	4.10 (4.00-4.40)	0.131
CRP	0.75 (0.20-2.01)	1.05 (0.30-2.80)	0.30 (0.10-0.70)	0.001
Creatinine, mg/dl	0.94 (0.85-1.08)	0.95 (0.85-1.10)	0.92 (0.87-1.05)	0.666
Total cholesterol	189.0 (158.5-224.0)	198.5 (165.0-227.0)	176.0 (141.0-190.0)	0.028
Trigliserid	125.5 (104.0-176.0)	125.5 (104.0-216.0)	127.0 (105.0-163.0)	0.498
LDL	116.1±39.3	120.3±39.5	104.7±37.2	0.105
Troponin	6750.11	9059.69	1022.35	0.000
NPAR	18.62±3.17	19.22±3.14	17.14±2.78	0.004

IRA: Infarct-related coronary artery patency, HL: Hodgkin lymphoma, CAD: Coronary artery disease, LVEF: Left ventricular ejection fraction, WBC: White blood cell, Hgb: Hemoglobin, PLT: Thrombocyte, CRP: C-reactive protein, LDL: Low-density lipoprotein, NPAR: Neutrophil percentage-to-albumin ratio

Revascularization Procedure and Medications

Prior to percutaneous coronary intervention (PCI), a loading dose of antiplatelet drugs (aspirin 300 mg, clopidogrel 300 to 600 mg or ticagrelor 180 mg) was used according to established guidelines. PCI was performed via the femoral or radial route at the discretion of the attending physician, using standard techniques and appropriate strategies according to guidelines. No thrombolytic therapy was administered in the study population. During hospitalization and after discharge, antiplatelets, statins, b-blockers and angiotensin-converting enzyme inhibitors were administered to all patients according to guidelines unless contraindicated.

Statistical Analysis

IBM SPSS Statistics 23 package was used for statistical analysis. Categorical variables are presented as percentages, continuous variables are presented as mean±standard deviation if normally distributed and median interquartile range (IQR) if not. Two independent groups with normal distribution were compared using student's T test, while those without normal distribution were compared using Mann Whitney U test. The chi-square test will be used to compare categorical variables. Receiver operating characteristic curve (ROC) analysis was used to define the area under the curve to estimate the optimal cutoff level of NPAR and IRA patency. Regression analysis was performed for possible parameters in predicting IRA patency, evaluated in univariable analysis, and those with p-value <0.05 were evaluated in multivariable analysis. Statistical significance is defined as p-values less than 0.05.

G-power (version 3.1.9.7) was used to determine the minimum sample size. Accordingly, T tests, means: difference between two independent means (matched pairs), a priori: compute required sample size-given a, power, and effect size were selected. Accordingly, when α err prob=0.05, power (1-β err prob)=0.80, and effect size=0.6, it was determined that at least 86 participants should participate in the study for each group (actual power=80.3%).

RESULTS

A total of 87 patients with 62 non-patent and 25 patent IRA were included in the study. The responsible artery was the left anterior descending artery (LAD) in 29 patients, right coronary artery (RCA) in 29, circumflex artery (CX) in 12, saphenous grafts in 2, and diagonal artery or obtuse marginal artery in the rest. 65 (74%) of the patients were male and 22 (26%) were female (Table 1). NPAR was also significantly higher in the non-patent group (19.22±3.14 and 17.14±2.78 p=0.004). Troponin values were also significantly higher in the non-patent group (9059.69 and 1022.35 p<0.001). NPAR was also significantly higher in the MACE group (22.83±3.85 and 17.95±2.49 p<0.001). Albumin was also significantly higher in the MACE group (4.07 ± 0.35 and 3.50 ± 0.65 p<0.001). WBC values were compared between the two groups, the non-patent group was found to be significantly higher (10.9 and 9.9 p=0.02). CRP values were significantly higher in the non-patent group (1.05 and 0.3 p=0.001) (Table 2).

In multivariable logistic regression analysis, NPAR [p=0.027, odds ratio (OR): 0.787, 95% confidence intervals (CIs): 0.637-0.974] levels were found to be independent predictors of patent IRA. WBC (p= 0.049, OR: 0.848, 95% CIs: 0.717-0.999) levels were found to be independent predictors of patent IRA. But CRP (p=0.049, OR: 0.656, 95% CIs: 0.414-1.040). Univariable and multivariable regression analyses of potential predictive factors in determining IRA patency are shown in Table 3.

Table 2. Characteristics of study patients according to the MACE

Variables	Non-MACE (75)	MACE (12)	p
Hypertension, n (%)	38 (50.6)	8 (66.6)	0.308
Diabetes mellitus, n (%)	16 (21.3)	4 (33.3)	0.100
HL	34 (45.3)	5 (41.6)	0.815
CAD	26 (34.6)	4 (33.3)	0.929
LVEF	46.84±9.91	43.33±11.34	0.268
WBC	11.97±4.21	10.77±3.29	0.350
Hgb	14±1.86	12±2.68	0.002
Neutrophil count, x1000/ul	8.78 (6.62-10.70)	8.59 (4.75-8.80)	0.859
Lymphocyte count, x1000/ul	2.14 (1.44-2.62)	1.51 (1.26-2.53)	0.048
PLT	257.2 (201.0-280.0)	216.0 (212.0-268.0)	0.101
Albumin	4.07±0.35	3.50±0.65	0.000
CRP	3.10	2.70	0.114
Total kolesterol	190.64 (165.0-227.0)	189.75 (141.0-190.0)	0.949
Trigliserid	146.98 (104.0-216.0)	190.25 (105.0-202.0)	0.093
LDL	116.34±39.5	114.62±41.6	0.889
Troponin	5661.23	13555.70	0.083
NPAR	17.95±2.49	22.83±3.85	0.000

MACE: Major adverse cardiovascular events, HL: Hodgkin lymphoma, CAD: Coronary artery disease, LVEF: Left ventricular ejection fraction, WBC: White blood cell, Hgb: Hemoglobin, PLT: Thrombocyte, CRP: C-reactive protein, LDL: Low-density lipoprotein, NPAR: Neutrophil percentage-to-albumin ratio

Table 3. Univariate and multivariate logistic regression analysis for the risk factors in predicting the IRA patency

	Univariate	Multivariate
	OR (95% confidence interval)	OR (95% confidence interval)
NPAR	0.771 (0.637-0.934, p=0.008)	0.787 (0.637-0.974, p=0.027)
WBC	0.822 (0.698-0.969, p=0.019)	0.848 (0.717-0.999, p=0.049)
CRP	0.590 (0.360-0.968, p=0.037)	0.656 (0.414-1.040, p=0.073)

IRA: Infarct-related coronary artery patency, OR: Odds ratio, NPAR: Neutrophil percentage-to-albumin ratio, WBC: White blood cell, CRP: C-reactive protein

As revealed by the ROC curve analysis, the cut-off value of 17.88 for NPAR predicted the non-patent IRA with a sensitivity of 64% and specificity of 64% (AUC: 0.681; CIs: 0.588-0.809; p=0.008 (Figure).

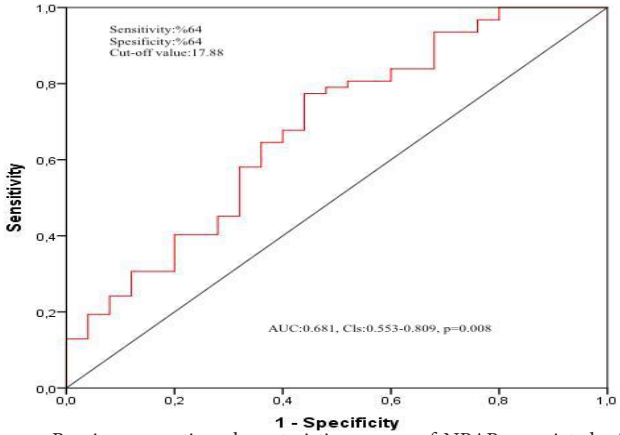

Figure. Receiver operating characteristics curves of NPAR associated with IRA patency

NPAR: Neutrophil percentage-to-albumin ratio, IRA: Infarct-related coronary artery patency

DISCUSSION

In our study, we investigated the relationship between NPAR and IRA patency and the group with MACE in ACS patients. The main finding of our study was that significantly higher NPAR levels were associated with non-patent IRA patients and MACE group and were independent predictors.

Coronary artery disease is one of the leading causes of death in the world and in our country. In recent years, the role of inflammation and biomarkers reflecting inflammatory status in CAD and its relationship with adverse events have been investigated in many studies. Numerous indicators of inflammation have been investigated. The condition and degree of stimulation of the inflammatory response in our bodies have been examined using a variety of biomarkers, including cytokines, adhesion molecules, white blood cells, and acute phase reactants. White blood cell (WBC) count, one of the most basic cells of inflammation, and its subtypes have been investigated in adverse events in cardiovascular diseases and used as a marker of inflammatory status. One study showed that increased neutrophil levels in ACS were associated with the extent of myocardial damage and short-term prognosis.[8]

Inflammation, the atherosclerotic process and the occurrence of CAD are tightly linked through several complex pathophysiological pathways. Neutrophils, an important member of the WBC, together with cytokines and phospholipids, play an important role in triggering the inflammatory reaction, coronary atherosclerosis and acute myocardial infarction (AMI).[9]

It has long been believed that albumin is a sign of nutritional well. According to the majority of data, alterations in acute phase proteins such SA and prealbumin may be linked to inflammation and the severity of the illness rather than reflecting inadequate nutritional condition.[10] In patients with ACS, low SA levels have been shown to be an independent predictor of both in-hospital mortality.[11] Reduced SA levels, or hypoalbuminemia, also raise blood viscosity and impair endothelium. Ischemic heart disease incidence is inversely correlated with SA levels.[12] Additionally, a significant correlation between low SA levels and long-term mortality was shown in STEMI patients undergoing PCI, as well as in patients with unstable angina pectoris (USAP) and NSTEMI.[13] In our study, albumin levels were significantly lower in the group with MACE. This suggests that low SA levels may be a predictive factor for long-term mortality after ACS, as in previous studies.

The ratio of neutrophil percentage to albumin count is expressed using a new metric called NPAR. In cases of severe sepsis or other clinical events such septic shock, acute renal injury, and cardiogenic shock, prior research has shown the predictive usefulness of NPAR.[14-16] In a study by Cai et al.[17] NPAR was found to be an independent predictor of 365-day mortality in patients followed in the coronary intensive care unit. Another study by Cui et al.[18] showed that NPAR was an independent predictor of in-hospital mortality after STEMI. In our study, the NPAR was significantly higher in the group with MACE compared to the group without MACE. This suggests that higher NPAR on admission may be independently associated with death from causes such as 365-day reinfarction, stroke, malignant arrhythmia and heart failure in patients with ACS. Furthermore, in our analysis, the NPAR was significantly higher in the group with non-patent IRA compared to the group patent IRA. This implies that NPAR may also serve as a predictor of IRA patency.

Preserving the IRA patent at an early stage is the primary objective of treatment for patients with AMI. Poor clinical results have been linked to off-patent IRA at presentation. Particularly in patients with STEMI, early IRA patency is crucial to maintaining cardiac function and lowering the likelihood of mechanical and lethal arrhythmias.[19] The TIMI flow rate is used to determine IRA patency. Good clinical results have been linked to monitoring the TIMI-3 flow rate at admission.[20] Another crucial sign of post-procedural patency is IRA patency at admission. For all of these reasons, the prognosis depends on the early assessment and restoration of IRA patency. The function of various indicators in forecasting IRA patency at presentation has been examined in earlier study. Doğan et al.[21] reported IRA patency in STEMI patients was predicted by the neutrophil-to-lymphocyte (N/L) ratio, another inflammatory marker. In another study, hematologic parameters were analyzed in STEMI patients undergoing primary angioplasty, and WBC count, which plays a vital role in inflammation, was associated with IRA patency.[22] In our study, WBC levels were significantly higher in patients with non-patent IRA compared to patent IRA group, which was similar to previous studies. It showed that the WBC values during hospitalization can be used as an indicator of IRA patency.

Troponin is the most commonly used biochemical parameter in the diagnosis of AMI. In previous studies, troponin values were found to be significantly higher in patients with non-patent IRA compared to patients with patent IRA.[23] In our study, similar to these studies, troponin values were found to be significantly higher in patients with non-patent IRA. Troponin was considered as a method that can be used for both diagnosis and patent IRA.

In everyday practice, it is simple to determine inflammation parameters. Their use for practitioners is expanded because they will be obtained using blood biomarkers that should be regularly observed in patients undergoing or planned CAG. The murky regions in this regard are still being clarified by several studies conducted in recent years. ACS continues to rank among the world's major causes of death. Our task will be made easier if there are more parameters available for usage, particularly in the follow-up of ACS patients. Thus, metrics that may make predictions without wasting a lot of time, like inflammatory parameters, come to the fore. We believe that our study demonstrates the importance of NPAR, simply calculated using neutrophils and albumin, for mortality prediction. Additionally, we discovered that NPAR was important in demonstrating IRA patency. One computation technique that is simple to apply in day-to-day work is NPAR.

Limitations

Since our study was single-center and retrospective, not all patients had albumin values and this led to a limitation in the number of patients. In addition, since inflammation parameters may be affected in patients older than 65 years of age, the age of the patients included in the study was limited to 65 years of age, considering that this would not lead to incorrect results.

CONCLUSION

In conclusion, inflammatory markers have been and are being used as predictive parameters for cardiovascular diseases in many studies. In our study, we focused on neutrophils and albumin. We have shown that NPAR may be pathway markers for IRA. We also indicated that NPAR may have a predictive role for mortality in the long-term follow-up of ACS patients. In addition, we think that easily calculable markers such as WBC and troponin may be instructive for IRA patency. According to the results of multivariate regression analysis, we think that CRP can also be used, albeit weakly.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of Bandırma Onyedi Eylül University Health Sciences Non-interventional Researches Ethics Committee (Date: 22.04.2024, Decision No: 2024-4).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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