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Correlations between hematological indicators and other known markers in acute coronary syndromes

Mehmet Ertürk[1], Fatma N. Turhan Caglar[2], İsmail Bıyık[3], Nilgün Işıksaçan[4], Serkan Yazan[1], Mehmet Rıfat Yıldırım[1], Ömer Faruk Baycan[1], Begüm Özalp[1], Hüseyin Karakurt[1]

[1)] Department of Cardiology, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey
[2)] Department of Cardiology, Bakirkoy Dr. Sadi Konuk Education and Research Hospital, Istanbul, Turkey
[3)] Department of Cardiology, Usak University, School of Medicine, Education and Research Hospital, Usak, Turkey
[4)] Department of Biochemistry, Bakirkoy Dr. Sadi Konuk Education and Research Hospital, Istanbul, Turkey

Abstract

Introduction: Recently, many hematologic markers have identified as prognostic and diagnostic indicators in different acute coronary syndrome (ACS) patients. In particular, neutrophil / lymphocyte ratio (NLR) and platelet / lymphocyte ratio (PLR) are recognized as markers in the diagnosis and prognosis of ACS. In this study, our aim was to investigate the correlation between the diagnostic yield of PLR and NLR values and other markers such as troponin in all ACS patients.

Material and methods: 319 patients admitted to our hospital with ACS and 283 control patients were included in the study. Leukocyte, neutrophil, platelet, lymphocyte counts, PLR, NLR and high sensitive troponin I (HsTnI) measurements were taken.

Results: Leukocyte, neutrophil and platelet counts were significantly higher in the ACS group than the controls ($p < 0.001$). Lymphocyte count was significantly lower in the ACS group than the controls ($p < 0.001$). NLR and PLR were found to be significantly higher in the ACS group than the controls (4.0 ± 3.3 vs 2.1 ± 1.4 and 126.3 ± 68.9 vs 106.9 ± 49.4, $p < 0.001$, respectively). NLR was showed significant correlation with HsTnI, PLR, angina time, presence of ST elevation and T wave negativity ($p < 0.05$), and PLR revealed significant correlation with NLR, HsTnI, ST elevation and T wave negativity ($p < 0.05$).

Conclusion: Hematologic markers were found to be significantly higher in the ACS group than the controls. The difference also continued in subgroup analyzes. NLR and PLR correlated with many other ACS indicators.

Key words: Platelet lymphocyte ratio, neutrophil lymphocyte ratio, acute coronary syndrome, troponin.

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Introduction

Coronary artery disease (CAD) is a major cause of morbidity and mortality worldwide.[1] One of the most important causes of CAD is atherosclerosis and atherosclerosis is considered to be an inflammatory disease.[2] Previous studies have shown serious evidence that atherosclerosis is systemic rather than focal disease.[2] Recently, many studies have been published, which demonstrate that hematological markers measured in peripheral blood count such as platelet, neutrophil, lymphocyte are predictors in the diagnosis and the determination of the prognosis of acute coronary syndromes (ACS).[3, 4] Increased platelet activation has been reported to play a role in the onset and the progression of atherosclerosis.[5] It has been reported that increased platelet count is associated with platelet activation and may be associated with thrombotic tendency and major adverse cardiac events.[3, 5]

On the other hand, there are reports that low lymphocyte counts are associated with lymphocyte apoptosis in prolonged inflammation, decreased immune-reactivity, and may also cause major adverse cardiac events.[5-7] As a result, proliferation and relative thrombocytosis are seen in the megakaryocytic series with inflammation.[7] Thus, in some studies, high platelet / lymphocyte ratio (PLR) and neutrophil / lymphocyte ratio (NLR) are predicted to better reflect inflammation and pro-thrombotic states and may be predictors of prognosis in acute ACS patients.[7, 8] In our study, we aimed to investigate the diagnostic efficiency of PLR and NLR values and their correlation with other predictors such as high sensitivity troponin I in ACS patients admitted to our hospital.

Patients and Method

This single-center comparative study was conducted in a tertiary heart center. The study was approved by the local Ethics committee and informed consent of the patients and controls were obtained. 319 patients admitted to our hospital with the diagnosis ACS between 2015 and 2017 were included in the study. In the same time period, 283 healthy individuals who applied to our outpatient clinics were included in the study as a control group. Subsequently, the ACS group was divided into subgroups as unstable angina pectoris (USAP), non-ST elevation myocardial infarction (NSTEMI), and ST elevation myocardial infarction (STEMI). Patients with severe valve disease, decompensated heart failure, malignancy, hematological disease, systemic inflammatory disease, active infection, autoimmune disease, severe renal or hepatic insufficiency and those using steroids were excluded from the study.

The demographic and biochemical parameters of the patients and controls were obtained from the files. NLR and PLR were calculated from the neutrophil, platelet and lymphocyte counts obtained from the hemogram. PLR and NLR were calculated by dividing platelet count by lymphocyte count, respectively, by dividing the number of neutrophils by the number of lymphocytes. For the measurement of high sensitive troponin I (HsTnI) EDTA whole blood sample was used, HsTnI was measured by the method of the AQT90 FLEX cardiac Troponin I immunoassay (Radiometer Medical ApS, Copenhagen, Denmark), and the method is a one-step sandwich immunofluorometric assay based on the use of three monoclonal antibodies, two for the capture and one for the detection.[9]

Complete blood count (CBC) was measured from EDTA whole blood samples via BC 6800 auto analyzer (Mindray Medical International Limited, Shenzhen, China). Other biochemical measurements such as glucose, HDL, LDL, total cholesterol and triglycerides were made from serum samples via Cobas systems (Roche Diagnostic Basel, Switzerland) by using commercial kits (Roche Diagnostic Basel, Switzerland).[9] Two levels of internal quality controls were made for all devices.

Statistical analysis

Statistical analysis was performed using SPSS version 15.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were described by mean and standard deviation. Categorical variables were described in percent. Mann-Whitney U-test and student t-test were used for continuous variables, while chi-square test was used for categorical variables. Kruskal-Wallis test and Mann-Whitney U-test were used for subgroup analysis. Spearman test was used for correlation analysis. The

diagnostic accuracy of high sensitive troponin I, NLR and PLR for ACS was calculated by receiver operating characteristic (ROC) analysis.

Results

319 ACS patients (219, 68.7%, male) and 283 controls (181, 64%, male) were included in the study. There were 114 (35.7%) patients with USAP, 101 (31.7%) patients with NSTEMI and 104 (32.6%) patients with STEMI in the ACS subgroups. The demographic, clinical, electrocardiographic and biochemical parameters of the patients are shown in **Table 1**. Neutrophil and platelet counts were significantly higher in the ACS groups than in the control group ($p < 0.001$ respectively). Lymphocyte count was lower in the ACS group than in the control group ($p < 0.001$). NLR and PLR were significantly higher in the ACS group than in the control group ($p < 0.001$).

In the ACS subgroup analysis, NLR and PLR values were significantly higher in STEMI patients than in USAP and NSTEMI patients ($p < 0.001$) (**Table 2**). Correlation analyzes revealed that NLR correlated with HsTnI, PLR, ST elevation, ST depression, T wave negativity and angina duration (**Table 3**). PLR was correlated with NLR, HsTnI, ST elevation and T wave negativity (**Table 3**). Receiver operating characteristic (ROC) analysis results are given in **Table 4**. When the cut-off value for NLR was taken as 2.5, the sensitivity of NLR for the diagnosis of ACS was found 63.6%, specificity 80.2%, negative predictive value 66.1% and positive predictive value 78.6% (95% confidence interval 0.66-0.75). When the cut off value for PLR was taken as 101.2, the sensitivity of PLR for the diagnosis of ACS was determined as 54.9%, specificity, 54.4%, negative predictive value 51.7% and positive predictive value 57.6% (95% confidence interval 0.54-0.63).

Discussion

In recent years, NLR and PLR have become important markers easily measured from peripheral blood and have proven diagnostic and prognostic information in patients with ACS.[10] In our study, NLR and PLR values were found to be higher in all ACS patients than the control group in accordance with the literature. In subgroup analyzes, NLR and PLR values were significantly higher in STEMI patients than NSTEMI and USAP patients. NLR and PLR were correlated with each other and with other parameters which have proven prognostic information in ACS patients such as high sensitive troponin I (**Table 3**). Sezer et al. showed that the number of neutrophils and the mean platelet volume in patients with acute myocardial infarction were associated with reperfusion injury after infarct-related artery occlusion opened.[10] In many inflammatory conditions neutrophils are stimulated to release many cytokines and cytotoxic or proteolytic enzymes.[4]

These enzymes have adverse effects on the ischemic heart by damaging endothelial cells, stimulating the coagulation system, blocking the microvascular circulation, and increasing infarct expansion.[4] Lymphocytes, as parts of the adaptive immunity system, fight against inflammation and suppress inflammation.[2] Thus, low

Table 2. Subgroups comparisons of hematologic indicators and cardiac markers

	USAP (n=114, 35.7%)	NSTEMI (n=101, 31.7%)	STEMI (n=104, 32.6%)	
	Mean ± Standard Deviation	Mean ± Standard Deviation	Mean ± Standard Deviation	P value
HsTnI (µg/L)	0.0108 ± 0.0084	1.2659 ± 3.6638	5.5973 ± 7.8381	<0.001
NLR	2.61 ± 1.59	3.30 ± 2.36	6.13 ± 4.34	<0.001
PLR	99.27 ± 48.54	125.90 ± 65.59	156.37 ± 78.56	<0.001

HsTnI: *High sensitivity troponin I,* ***NLR:*** *neutrophil / Lymphocyte ratio,* ***PLR:*** *platelet / lymphocyte ratio*

Table 1. Demographic characteristics of patients and controls

	Patients (n=319)	Controls (n=283)	P value
Age (years)	56.6 ± 11.4	47.3 ± 13.6	<0.001
Male, n (%)	219 (68.7)	181 (64)	0.223
BMI, kg/m²	27.8 ± 4.2	27.5±4.3	0.697
History			
Known CAD, n (%)	109 (34.2)	54 (19.1)	<0.001
Previous PCI, n (%)	80 (25.1)	44 (15.5)	0.004
Previous AMI, n (%)	59 (18.5)	32 (11.3)	0.014
Previous CABG, n (%)	51 (16)	13 (4.6)	<0.001
Previous stroke / TIA, n (%)	17 (5.3)	7 (2.5)	0.074
Cardiovascular risk factors, n (%)			
Hypertension, n (%)	145 (45.5)	102 (36)	0.019
Diabetes mellitus, n (%)	57 (17.9)	39 (13.8)	0.172
Family history of CAD, n (%)	80 (25.1)	30 (10.6)	<0.001
Smoking, n (%)	156 (48.9)	127 (44.9)	0.323
Electrocardiographic findings in admission			
Normal, n (%)	87 (27.3)	247 (87.3)	<0.001
ST segment elevation, n (%)	100 (31.3)	0 (0)	
ST segment depression, n (%)	61 (19.1)	6 (2.1)	
T-wave inversion, n (%)	42 (13.2)	24 (8.5)	
Bundle branch block, n (%)	29 (9.1)	6 (2.1)	
Duration of angina, hours	7.9 ± 1.7	8.0 ± 1.8	0.886
Biochemical parameters			
Total cholesterol, mg/dL	202.2 ± 44.3	188.6 ± 42.7	<0.001
LDL cholesterol, mg/dL	140.0 ± 41.6	123.9 ± 37.8	<0.001
HDL cholesterol, mg/dL	44.2 ± 10.5	44.8 ± 14.1	0.321
Triglycerides, mg/dL	167.0 ± 103.1	188.8 ± 125.0	0.046
Glucose, mg/dL	137.4 ± 63.1	115.4 ± 47.1	<0.001
Creatinine, mg/dL	0.96 ± 0.5	0.88 ± 0.47	0.003
High sensitive Troponin I µg/L	2.2295 ± 5.4671	0.0102 ± 0.0054	<0.001
Hemogram parameters			
Hematocrit, %	41.4 ± 5.9	40.6 ± 5.1	0.422
WBC	10.4 ± 3.5	8.5 ± 2.3	<0.001
Neutrophil count 103 / µL	7.2 ± 3.3	5.0 ± 1.9	<0.001
Lymphocyte count 103 / µL	2.3 ± 1.1	2.7 ± 0.9	<0.001
Platelet count 103 / µL	244.5 ± 60.0	260.6 ± 64.8	<0.001
NLR	4.0 ± 3.3	2.1 ± 1.4	<0.001
PLR	126.3 ± 68.9	106.9 ± 49.4	<0.001

BMI: *body mass index,* ***CAD:*** *coronary artery disease,* ***AMI:*** *acute myocardial infarction,* ***CABG:*** *coronary artery bypass grafting,* ***TIA:*** *transient ischemic attack,* ***LDL:*** *low-density lipoprotein cholesterol,* ***HDL:*** *high-density lipoprotein cholesterol,* ***Htc:*** *hematocrit,* ***WBC:*** *white blood cell count,* ***NLR:*** *neutrophil / lymphocyte ratio,* ***PLR:*** *platelet / lymphocyte ratio.*

lymphocyte counts have been associated with atherosclerosis progression and poor clinical outcomes.[2] Zouridakis et al found that low lymphocyte counts in unstable angina pectoris patients are strongly associated with future cardiac events.[11]

Although the pathophysiological mechanism of this condition is not fully understood, lymphocyte count is thought to reflect the early response of myocardial ischemia to physiological stress and systemic immunodeficiency.[4] Conversely, high lymphocyte counts during

Table 3. Correlation analyzes for NLR and PLR in ACS patients

Spearman's rho	Correlation Coefficient Sig. (2-tailed)	NLR	PLR
NLR	r	1.000	0.733**
	p	.	0.000
	n	319	319
HsTnI	r	0.466**	0.475**
	P	0.000	0.000
	n	319	319
PLR	r	0.733**	1.000
	p	0.000	.
	N	319	319
ST depression	r	-0.183**	0.051
	p	0.001	0.361
	n	319	319
Bundle branch block	r	-0.017	-0.045
	p	0.762	0.422
	n	319	319
Duration of angina	r	0.145**	0.085
	p	0.010	0.129
	n	319	319
Location of infarction	r	-0.023	0.068
	p	0.677	0.227
	n	319	319
T inversion	r	-0.285**	-0.221**
	p	0.000	0.000
	n	319	319
ST segment elevation	r	0.466**	0.365**
	p	0.000	0.000
	n	319	319
Type of angina	r	-0.075	-0.023
	p	0.181	0.676
	n	319	319

HsTnI; *high sensitivity troponin I,* ***NLR;*** *neutrophil / Lymphocyte ratio,* ***PLR;*** *platelet / lymphocyte ratio,* ***ACS;*** *acute coronary syndrome*

ACS reflect a strong immune response and the prognosis of these patients is better.[4] Instead of examining neutrophils and lymphocytes separately, NLR has been shown to be more prognostic.[2] Wang et al. have shown that NLR is predictive of all-cause mortality and cardiovascular events in patients undergoing coronary angiography.[12] Likewise, Tamhane et al. found that NLR value in patients with percutaneous coronary intervention is related to in-hospital and 6-month mortality.[13] Ayca et al. have shown that NLR is associated with stent thrombosis.[14]

NLR has been studied by different investigators in relation to different scoring systems showing CAD severity and it has been shown to be related to NLR by SYNTAX, GRACE and TIMI scores.[15-17] In our study, similarly, the NLR values were higher in the ACS group than in the control group. Correlation analyzes showed that NLR correlated with high sensitive troponin I, PLR, ST elevation, ST depression, T wave negativity and angina duration. NLR was found to have a sensitivity of 63.6% and a specificity of 80.2% for ACS diagnosis. The role of platelets in ACS is versatile.[4] The platelet count is associated with the underlying inflammation because inflammatory mediators stimulate megakaryocytic proliferation and result in a prothrombotic state with relative thrombocytosis.[4, 5]

As platelet levels increase, more platelet rich thrombosis is observed in atherosclerotic plaques, and antiplatelet therapy response is reduced.[4] It is known that platelets are associated with endothelial activation as well as inflammatory functions.[2] Active platelets release pro-angiogenic mediators and regulate the microvasculature of blood vessels.[18] As essential components of coronary thrombus formation, platelets play an important role in ACS.[2] In the CADILLAC study, increased platelet counts were also associated with restenosis and stent thrombosis.[19] PLR is thought to better reflect both inflammation and coagulation pathways.[4] In their study using optical coherence tomography, Wang et al showed that high PLR is associated with sensitive plaque properties such as thinner fibrous cap, wider lipid load in non-target vessels in ACS patients.[20]

The authors suggested that the relation between PLR and sensitive plaques is associated with immunologic and inflammatory pathways.[20] The relationship between PLR and CAD severity is also shown in other publications.[21] For example, Kurtul et al. have shown that high PLR is associated with moderate to high SYNTAX score in patients with ACS.[21] In another study, high platelet counts have been shown to be associated with microvascular plug formation, thrombus formation, and vasoconstriction and no-reflow development.[8] PLR has been shown to be associated with mortality in STEMI and NSTMI patients.[22] Oylumlu et al noted that high PLR values in ACS patients were associated with in-hospital mortality.[4]

In their meta-analysis, Li et al. showed that increased PLR is associated with in hospital and long-term mortality and cardiovascular events in ACS patients.[5] Yıldız et al. have also shown that high PLR values are associated with no-reflow development after percutaneous interventions.[23] Prajapati et al. showed a relationship between

Table 4. Receiver operating characteristic (ROC) analyzes of HsTnI, NLR and PLR for diagnosis of ACS

	AUC	SE	P-value	CI%95		Specificity	Sensitivity	NPV	PPV	Cut off value
				Lower limit	Upper limit					
HsTnI	0.800	0.018	0.000	0.764	0.836	99.6	54.2	65.9	99.4	>0.0023
NLR	0.709	0.022	0.000	0.667	0.751	80.2	63.6	66.1	78.6	>2.5
PLR	0.586	0.023	0.000	0.541	0.632	54.4	54.9	51.7	57.6	>101.2

HsTnI; *high sensitivity troponin I,* ***NLR;*** *neutrophil / Lymphocyte ratio,* ***PLR;*** *platelet / lymphocyte ratio,* ***ACS;*** *acute coronary syndrome,* ***NPV;*** *negative predictive value,* ***PPV;*** *positive predictive value*

increased PLR and NLR and decreased HDL values in CAD patients.[18] The combined use of NLR and PLR suggests that more prognostic information will be obtained. In this context, Choi et al. found that combined NLR and PLR elevations were associated with long-term cardiovascular events (22). Hematological markers have also been shown in other publications as indicators of inflammation. Similar to other studies, in our study, NLR and PLR in patients with ACS were higher than in the control group. The difference of present study from the other studies is that all the ACS subgroups were included and examined in the same study and that the NLR and PLR were high in all the subgroups.

Conclusion

Inflammatory and thrombotic processes play a crucial role in the development of atherosclerosis, atherosclerotic plaque destabilization and clot formation after plaque rupture. In the diagnosis and prognosis of ACS, there are still needs for reliable, easy, inexpensive and fast detectable markers. PLR and NLR are cheap and easy to use markers in the diagnosis and the prognosis of ACS patients but definite cut off values are needed to use these markers. Large scale and comprehensive studies are needed to reveal absolute cut off values for these markers.

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Corresponding author:
Dr. İsmail Bıyık / (Tel:+905424173209)
Mail: ismailbiyikmd@yahoo.com

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