

Association of mean platelet volume with presence of non-viable myocardium in ischaemic cardiomyopathy

Semi Ozturk¹, Ahmed Seyfettin Gürbüz², Süleyman Çağan Efe³, Mehmet Fatih Yılmaz⁴, Özden Savaş Den Hartigh⁵, Ferahnaz Çınaral Sayın⁵, Cevat Kıрма⁴

¹Department of Cardiology, Haseki Education and Training Hospital, Istanbul, Turkey

²Department of Cardiology, Yunus Emre State Hospital, Eskisehir, Turkey

³Department of Cardiology, Agri State Hospital, Agri, Turkey

⁴Department of Cardiology, Kartal Kosuyolu Heart Training and Research Hospital, Istanbul, Turkey

⁵Department of Nuclear Medicine, Kartal Kosuyolu Heart Training and Research Hospital, Istanbul, Turkey

Abstract

Background: Parameters derived from complete blood count, such as mean platelet volume (MPV), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR), have recently been proposed as measures of inflammation in addition to C-reactive protein (CRP), a classical inflammatory marker. Significant association of these parameters with atherosclerosis and complications have increasingly been reported.

Aim: The aim of the study is to evaluate the relationship between these parameters and the presence of myocardial viability assessed with positron emission tomography (PET) in patients with ischaemic cardiomyopathy (ICM).

Methods: A total of 122 ICM patients who had undergone PET were enrolled in this study. The patients were dichotomised depending on the presence of transmural scar. Group 1 consisted of 21 patients who had transmural scar tissue only, who were accepted as the group having non-viable myocardium. Group 2 consisted of 101 patients who had hibernation and/or non-transmural scar, who were accepted as the group having viable myocardium. Haematological parameters within 30 days of PET imaging were retrospectively analysed.

Results: There were no significant differences between the two groups regarding values of white blood cell, neutrophil, lymphocyte, platelet, haemoglobin, red cell distribution width, CRP, PLR, and NLR. Patients with non-viable myocardium have significantly higher levels of MPV ($p = 0.002$). In multiple logistic regression analysis, MPV (odds ratio [OR] = 0.373, 95% confidence interval [CI] 0.20–0.69, $p = 0.002$), was identified as an independent predictor of non-viable myocardium. In receiver-operator characteristic (ROC) analysis, a cut-point of 8.19 identified patients with non-viable myocardium (area under curve: 0.72, 95% CI 0.60–0.84). An MPV value greater than 8.19 demonstrated a sensitivity of 76% and a specificity of 55%.

Conclusions: The present study showed that MPV is an inexpensive, clinical, and routinely measurable parameter that is associated with the presence of viable myocardium in ICM.

Key words: platelet, positron emission tomography, cardiomyopathy

Kardiol Pol 2017; 75, 10: 1013–1019

INTRODUCTION

Acute coronary syndromes are the worldwide leading cause of mortality and morbidity. Increased primary percutaneous intervention facilities decreased mortality; however, ischaemic cardiomyopathy (ICM) remains a cause of major morbidity even after successful revascularisation. The pres-

ence of viable myocardium is correlated with the outcomes after revascularisation in ICM. Different imaging modalities, including echocardiography, single photon emission computed tomography (SPECT), positron emission tomography (PET), and magnetic resonance imaging (MRI), are studied to determine viable myocardium. Moreover, the high diagnostic

Address for correspondence:

Dr. Semi Ozturk, Department of Cardiology, Haseki Education and Training Hospital, Istanbul, Turkey, e-mail: semi_ozturk@yahoo.com

Received: 06.11.2016

Accepted: 18.05.2017

Available as AoP: 07.07.2017

Kardiologia Polska Copyright © Polskie Towarzystwo Kardiologiczne 2017

value of PET in the assessment of myocardial viability is well documented [1].

White blood cell (WBC) subsets and their derived parameters such as the neutrophil-to-lymphocyte ratio (NLR) and the platelet-to-lymphocyte ratio (PLR) are accepted as the new inflammatory markers that may be associated with the atherosclerotic process [2]. Mean platelet volume (MPV) is an indicator of platelet activity, where larger platelets are more active in terms of aggregation and thrombogenicity. NLR and MPV are not only associated with cardiovascular events but also are associated with the severity of atherosclerosis [3].

These two parameters determine the outcome of cardiovascular events. Some studies have observed the relationship between NLR, MPV, and myocardial perfusion defects [4, 5]. However, to the best of our knowledge, the relation of these parameters with the presence of myocardial viability — which is the major determinant of outcome in ICM — has not been studied. Our aim is to evaluate this relationship between myocardial viability assessed with PET and haematological blood count parameters, such as WBC, neutrophil, lymphocyte, platelet, haemoglobin, red cell distribution width (RDW), MPV, NLR, PLR, and inflammatory marker C-reactive protein (CRP) in patients with ICM.

METHODS

Study population

Consecutive patients undergoing myocardial PET imaging between August 2011 and September 2013, who had a history of ICM were evaluated. All patients had a history of myocardial infarction. The time from myocardial infarction to PET changed from 3 to 38 months. PET imaging was performed in order to determine the presence of viable myocardium for stable angina pectoris after coronary angiography for all patients. Complete blood count and CRP levels within 30 days of imaging were retrospectively evaluated. Patients were included in the present study after the following exclusions: patients with hepatic and haemolytic disorders, concomitant inflammatory diseases and neoplastic diseases, and patients taking steroids, immunosuppressive drugs, or non-steroidal anti-inflammatory drugs (except for low-dose aspirin). A total of 122 patients (107 males and 15 females) were included in the study. The patients were dichotomised depending on the presence of transmural scar. Group 1 consisted of 21 patients who had transmural scar tissue only (19 males and 2 females). Group 2 consisted of 101 patients who had hibernation and/or non-transmural scar (88 males and 13 females). Forty patients had hibernation only. Sixty-four patients had hibernation and non-transmural scar, and three patients had non-transmural scar only. Histories, demographics, risk factors, biochemical results, and PET imaging findings of all patients were reviewed and noted.

Myocardial perfusion SPECT imaging

All patients scheduled for 18F-fluorodeoxyglucose (FDG) PET/CT for ICM should also undergo myocardial perfusion

assessment. Patients underwent Tc-99m MIBI myocardial perfusion scintigraphy with a two-day protocol. The treadmill exercise or pharmacological stress with dipyridamole was used. The treadmill test was performed using Bruce protocol. 20 mCi Tc-99m MIBI was injected at peak exercise level, and the patients continued the exercise at least for 90 s. For the dipyridamole stress test, 0.56 mg/kg of dipyridamole was infused intravenously over 4 min. Tc-99m MIBI (20 mCi) was injected 8 min after the beginning of injection of dipyridamole infusion. Tc-99m MIBI ECG gated SPECT imaging was performed 1 h after the 20 mCi tracer injection at stress and at rest.

Myocardial 18F-FDG PET imaging

The baseline blood sugar level was checked in the morning after at least 6 h of fasting. 50–100 g of glucose was loaded, and after 45–60 min the injection blood sugar level was rechecked. 444 MBq (12 mCi) of 18F-FDG was injected if the sugar level was lower than 140 mg/dL. 2, 3, and 5 U of insulin was injected for 140–160 mg/dL, 160–180 mg/dL, and 180–200 mg/dL of blood glucose, respectively, if the sugar level was higher than 140 mg/dL. About 45–60 min after 18F-FDG injection, myocardial 18F-FDG PET study was performed in a PET scanner in three-dimensional mode (Siemens Biograph 2 LSO DUO PET/CT, Germany). PET acquisition parameters were as follows: myocardium was covered in one bed position with a slice thickness of 5 mm, 10 min of emission time, 128 matrix, zoom 2.0, and iterative reconstruction (4 iterations, 8 subsets). Attenuation correction was performed by CT.

PET images were analysed by two nuclear medicine specialists. Perfusion and 18F-FDG PET images were interpreted side-by-side using conventional Siemens cardiac display software, and a comparison of myocardial perfusion to myocardial metabolism was included. Three major patterns were reported:

- Reduced myocardial perfusion with preserved FDG uptake, mismatch pattern, representing hibernating myocardium.
- Absent myocardial perfusion with absent FDG uptake, match pattern, representing transmural scar.
- Partially reduced myocardial perfusion with concordant FDG uptake, non-transmural match pattern, representing non-transmural scar.

The presence of the hibernation and/or non-transmural scar were accepted as the presence of viability. An example pattern of transmural scar is displayed in Figure 1. Since PET imaging was not performed with ECG gated and additional quantification was not available on the current software, we did not measure the extent or number of metabolic defects.

Biochemical and haematological parameters

The blood was collected from the antecubital vein. Complete blood count analysis was performed using a Beckman Coulter HMX-AL (Brea, CA, USA), and CRP was measured in serum

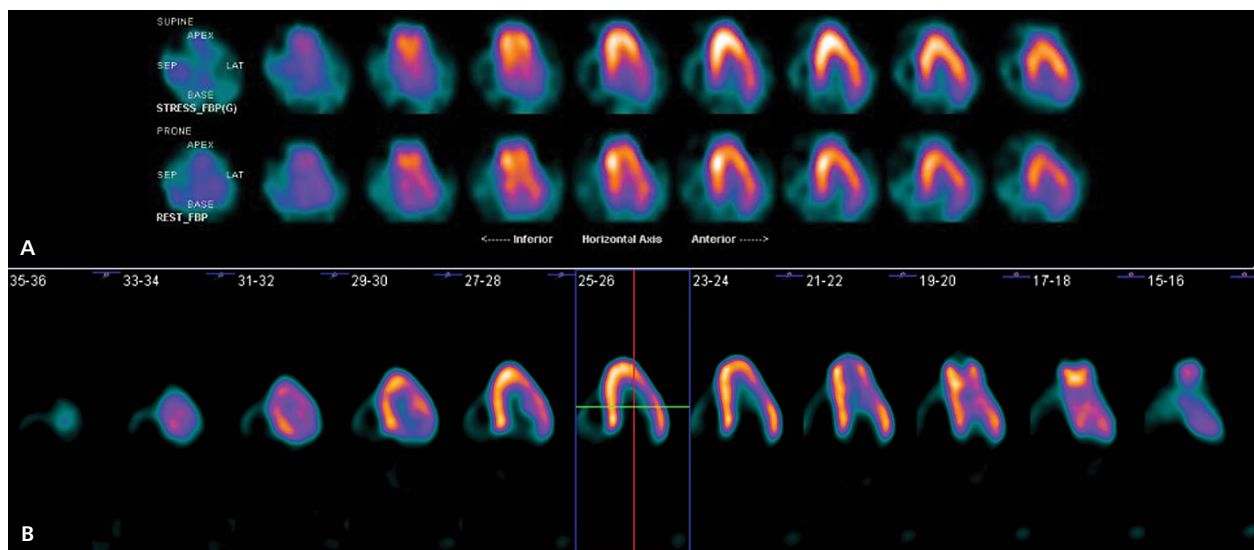


Figure 1. A patient with apical transmural scar; **A.** A perfusion both at rest and stress; **B.** Absence of fluorodeoxyglucose uptake

by enzyme immunoassay (EIA) (Immagine CRP EIA Kit; Beckman Coulter Inc., Brea, CA). WBC, neutrophil, lymphocyte, platelet, haemoglobin, RDW, and MPV were recorded, and NLR and PLR were derived from these parameters.

Statistical analysis

Statistical analyses were performed using SPSS 17.0 (Chicago). The Kolmogorov-Smirnov test was used to analyse the normality of the data. The continuous data was expressed as mean \pm standard deviation (SD) and the categorical data were expressed as percentages. The χ^2 test was used to assess the differences in categorical variables between the groups. The relationships among the parameters were assessed using Pearson's or Spearman's correlation analysis according to the normality of the data. Multiple logistic regression analysis was used to identify the independent predictors of viable myocardium. All variables showing significance values of less than 0.25 on univariate analysis (age, dyslipidaemia, hypertension, NLR, RDW, and MPV) were included in the model. The differences between patient subgroups were tested using Mann-Whitney U or Student's T testing, where an appropriate p-value < 0.05 was considered statistically significant.

RESULTS

In total 122 patients with ICM having myocardial PET were evaluated. Group 1 consisted of 21 patients with transmural scar in myocardium, who were accepted as the group having non-viable myocardium. Group 2 consisted of 101 patients with non-transmural scar and/or hibernation in myocardium, who were accepted as the group having viable myocardium.

The baseline characteristics of the groups are presented in Table 1. There were no differences between these two

groups in terms of age, hypertension, diabetes mellitus, and dyslipidaemia. In addition, no significant difference was observed regarding the double antiplatelet therapy (DAPT), beta-blocker, angiotensin converting enzyme (ACE) inhibitor, and mineralocorticoid receptor antagonist (MRA) use. There were no significant differences between the two groups regarding WBC, neutrophil, lymphocyte, platelet, haemoglobin, RDW, NLR, PLR, and CRP. Patients with viable myocardium have significantly lower levels of MPV ($p = 0.002$) (Table 2, Fig. 2). In multiple logistic regression analysis, MPV (odds ratio [OR] = 0.373, 95% confidence interval [CI] 0.20–0.69, $p = 0.002$) was identified as an independent predictor of myocardial viability (Table 3). In receiver-operator characteristic (ROC) analysis, a cut-point of 8.19 identified the patients with viable myocardium (area under curve [AUC]: 0.72, 95% CI 0.60–0.84) (Fig. 3). An MPV value less than 8.19 demonstrated a sensitivity of 76% with a specificity of 55%.

DISCUSSION

This study revealed that MPV is independently associated with the presence of viable myocardium. Our study showed that a cut-off value of MPV below 8.19 could predict the presence of viable myocardium in ICM.

The outcomes depend on the presence of viable myocardium in patients with ICM. The studies showed better prognosis, both in medically treated and revascularised patients with viable myocardium compared to non-viable [6]. Although many different imaging techniques were proposed, including echocardiography, SPECT, PET, and MRI, 18F-FDG PET is the most sensitive imaging modality for detecting viable myocardium. A meta-analysis of 24 studies reported a weighted sensitivity and specificity of 92% and 63%, respectively, with a positive and negative predictive value of 74% and 87%,

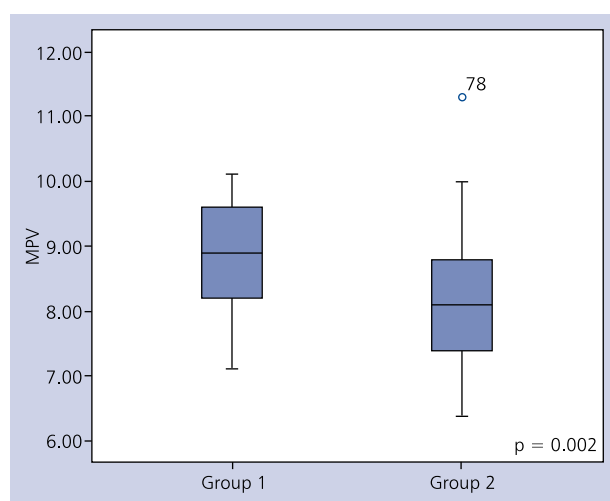
Table 1. Baseline characteristics

	Viable group (n = 101)	Non-viable group (n = 21)	p
Age	60.6 ± 10.6	57.7 ± 8.8	0.25
Sex, male	88 (87.1%)	19 (90.5%)	0.67
Hypertension	24 (23.8%)	8 (38.1%)	0.17
Diabetes mellitus	38 (37.6%)	8 (38.1%)	0.97
Dyslipidaemia	26 (25.7%)	9 (42.9%)	0.12
Ejection fraction	26.2 ± 8.0	27.6 ± 11.5	0.49
Double antiplatelet therapy	23 (22.8%)	4 (19.0%)	0.7
Beta-blocker	97 (96%)	21 (100%)	0.35
ACEI/ARB	92 (91.1%)	20 (95.2%)	0.53
MRA	41 (40.6%)	9 (42.9%)	0.85

ACE/ARB — angiotensin converting enzyme inhibitor/angiotensin receptor blocker; MRA — mineralocorticoid receptor antagonist

Table 2. Haematological parameters of groups

	Viable group (n = 101)	Non-viable group (n = 21)	p
White blood cell	7977 ± 2019	7789 ± 1520	0.68
Neutrophil	5085 ± 1482	4816 ± 1227	0.44
Lymphocyte	1939 ± 680	2202 ± 960	0.14
Neutrophil-to-lymphocyte ratio	2.92 ± 1.25	2.51 ± 1.04	0.17
Platelet	239 ± 67	233 ± 57	0.72
Platelet to lymphocyte ratio	138 ± 62	116 ± 34	0.23
Haemoglobin	13.3 ± 1.6	13.7 ± 1.6	0.3
Mean platelet volume	8.13 ± 0.94	8.89 ± 0.91	0.002
Red cell distribution width	15.9 ± 1.8	15.7 ± 2.3	0.25
C-reactive protein	1.07 ± 1.35	1.07 ± 1.36	0.41

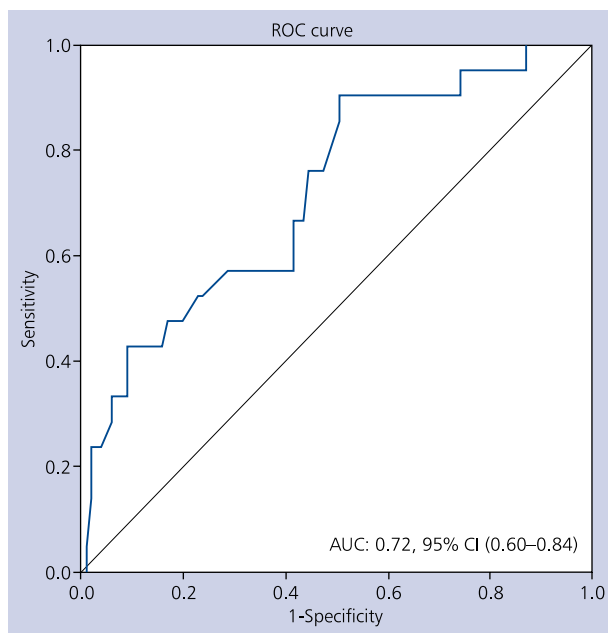
**Figure 2.** Mean platelet volume (MPV) values of groups; group 1 — non-viable; group 2 — viable

respectively, for the diagnosis of hibernating myocardium and the prediction of patient outcomes [6].

Mean platelet volume is a marker of platelet activity. Large platelets have denser granules containing mediators that play a role in inflammation, atherosclerosis, and eventually cardiac syndromes. Higher MPV is associated with increased mortality after both ST segment elevation myocardial infarction (STEMI) and non-STEMI [7, 8]. Higher MPV correlates with the reperfusion failure in STEMI [8, 9]. Higher MPV level may be associated with an impaired microvascular perfusion, even in successfully reperfused patients [10, 11]. Fabregat-Andrés et al. [11] showed the association of high MPV level with larger infarct size in STEMI by using a cardiac MRI, which is a very sensitive tool for the myocardial infarct assessment. Another study found a significant correlation between the degree of systolic depression and MPV in STEMI patients [12, 13]. MPV level predicts transmural involvement of myocardium in patients with STEMI [14]. Beside these studies, long-term

Table 3. Multivariate analyses of viable myocardium

	p	Exp (B)	CI % for EXP (B)	
			Lower	Upper
Age	0.722	1.01	0.955	1.069
Dyslipidaemia	0.174	2.188	0.708	6.76
Hypertension	0.237	1.938	0.647	5.809
Neutrophil-to-lymphocyte ratio	0.063	1.596	0.975	2.614
Red cell distribution width	0.848	1.028	0.775	1.364
Mean platelet volume	0.002	0.373	0.201	0.689

**Figure 3.** Diagnostic accuracy of mean platelet volume in prediction of viable myocardium; AUC — area under curve; ROC — receiver-operator characteristic

patency of infarct-related artery in follow-up is less in patients with higher MPV [15]. Left anterior descending artery (LAD) involvement is associated with high MPV level in patients with non-STEMI, and this may be another factor causing large infarct size in addition to the microvascular obstruction [16]. Although contradictory data exists, some studies showed a correlation of MPV with severity of coronary artery disease [17, 18]. Sarikaya et al. [5] showed MPV levels were higher in diabetic patients with myocardial perfusion defects than in those without. Thus, more extensive micro- and macrovascular diseases may result in a less perfused and less viable myocardium.

We did not observe any correlation between NLR, PLR, and CRP with myocardial viability. Evidently, low-grade inflammation contributes to the progression of atherosclerosis in all stages. Since our study included patients with stable

coronary artery disease with prior myocardial infarctions, it is plausible that the inflammatory parameters like NLR and CRP are all alike between the groups. Besides, platelet aggregation is an important stage that has a significant influence on the outcomes. Platelet aggregation ultimately determines the infarct size, transmuralty of infarct, and systolic functions. Aurigemma et al. [19] showed an increased persistence of platelet activity after one month in patients with STEMI and microvascular obstruction. Kiris et al. [20] compared MPV levels of non-STEMI patients on admission and at the 24th h; they found an association between total mortality and an early increase in MPV. The increased platelet activity after coronary events continues for a long period. Thus, more severely affected myocardium may be the result of the more persistent, extensive, and severe platelet activation. MPV was significantly higher in both ischaemic and non-ICM patients, compared with healthy controls in another study; this study demonstrated not only the correlation between high MPV levels and a decreased ejection fraction, but also the relationship between the high thromboembolic risk in cardiomyopathies and high MPV levels [21]. Since MPV is affected by several factors, such as increased levels of interleukins in cardiomyopathies, there may be a non-causal relationship between MPV and these factors. MPV may be a marker that reflects all stages of the atherosclerotic and thrombotic pathologies.

Limitations of the study

The retrospective nature of the study is the major limitation. The small number of patients is another limitation. Additionally, unavailability of quantitative data of PET patterns is a significant restriction. The findings of the study are yet to be generalised across all patients in clinical practice.

CONCLUSIONS

The present study showed that MPV is an inexpensive, clinical, and routinely measurable parameter that is associated with the presence of viable myocardium in ICM.

Conflict of interest: none declared

References

- Anagnostopoulos C, Georgakopoulos A, Pianou N, et al. Assessment of myocardial perfusion and viability by positron emission tomography. *Int J Cardiol.* 2013; 167(5): 1737–1749, doi: [10.1016/j.ijcard.2012.12.009](https://doi.org/10.1016/j.ijcard.2012.12.009), indexed in Pubmed: [23313467](https://pubmed.ncbi.nlm.nih.gov/23313467/).
- Sari I, Sunbul M, Mammadov C, et al. Relation of neutrophil-to-lymphocyte and platelet-to-lymphocyte ratio with coronary artery disease severity in patients undergoing coronary angiography. *Kardiol Pol.* 2015; 73(12): 1310–1316, doi: [10.5603/KP.a2015.0098](https://doi.org/10.5603/KP.a2015.0098), indexed in Pubmed: [25987404](https://pubmed.ncbi.nlm.nih.gov/25987404/).
- Uysal HB, Dağlı B, Akgüllü C, et al. Blood count parameters can predict the severity of coronary artery disease. *Korean J Intern Med.* 2016; 31(6): 1093–1100, doi: [10.3904/kjim.2015.199](https://doi.org/10.3904/kjim.2015.199), indexed in Pubmed: [27052265](https://pubmed.ncbi.nlm.nih.gov/27052265/).
- Williams BA, Merhige ME. Association between neutrophil-lymphocyte ratio and impaired myocardial perfusion in patients with known or suspected coronary disease. *Heart Lung.* 2013; 42(6): 436–441, doi: [10.1016/j.hrtlng.2013.07.013](https://doi.org/10.1016/j.hrtlng.2013.07.013), indexed in Pubmed: [23981470](https://pubmed.ncbi.nlm.nih.gov/23981470/).
- Sarikaya S, Sahin S, Akyol L, et al. Mean platelet volume is associated with myocardial perfusion defect in diabetic patients. *Cardiovasc J Afr.* 2014; 25(3): 110–113, doi: [10.5830/CVJA-2014-013](https://doi.org/10.5830/CVJA-2014-013), indexed in Pubmed: [25000440](https://pubmed.ncbi.nlm.nih.gov/25000440/).
- Schinkel AFL, Bax JJ, Poldermans D, et al. Hibernating myocardium: diagnosis and patient outcomes. *Curr Probl Cardiol.* 2007; 32(7): 375–410, doi: [10.1016/j.cpcardi.2007.04.001](https://doi.org/10.1016/j.cpcardi.2007.04.001), indexed in Pubmed: [17560992](https://pubmed.ncbi.nlm.nih.gov/17560992/).
- Wasilewski J, Desperak P, Hawranek M, et al. Prognostic implications of mean platelet volume on short- and long-term outcomes among patients with non-ST-segment elevation myocardial infarction treated with percutaneous coronary intervention: A single-center large observational study. *Platelets.* 2016; 27(5): 452–458, doi: [10.3109/09537104.2016.1143919](https://doi.org/10.3109/09537104.2016.1143919), indexed in Pubmed: [26939525](https://pubmed.ncbi.nlm.nih.gov/26939525/).
- Huczek Z, Kochman J, Filipiak KJ, et al. Mean platelet volume on admission predicts impaired reperfusion and long-term mortality in acute myocardial infarction treated with primary percutaneous coronary intervention. *J Am Coll Cardiol.* 2005; 46(2): 284–290, doi: [10.1016/j.jacc.2005.03.065](https://doi.org/10.1016/j.jacc.2005.03.065), indexed in Pubmed: [16022956](https://pubmed.ncbi.nlm.nih.gov/16022956/).
- Pereg D, Berlin T, Mosseri M. Mean platelet volume on admission correlates with impaired response to thrombolysis in patients with ST-elevation myocardial infarction. *Platelets.* 2010; 21(2): 117–121, doi: [10.3109/09537100903487599](https://doi.org/10.3109/09537100903487599), indexed in Pubmed: [20063988](https://pubmed.ncbi.nlm.nih.gov/20063988/).
- Sezer M, Okular I, Goren T, et al. Association of haematological indices with the degree of microvascular injury in patients with acute anterior wall myocardial infarction treated with primary percutaneous coronary intervention. *Heart.* 2007; 93(3): 313–318, doi: [10.1136/hrt.2006.094763](https://doi.org/10.1136/hrt.2006.094763), indexed in Pubmed: [16940390](https://pubmed.ncbi.nlm.nih.gov/16940390/).
- Fabregat-Andrés Ó, Cubillos A, Ferrando-Beltrán M, et al. Mean platelet volume is associated with infarct size and microvascular obstruction estimated by cardiac magnetic resonance in ST segment elevation myocardial infarction. *Blood Coagul Fibrinolysis.* 2013; 24(4): 424–427, doi: [10.1097/MBC.0b013e32835d9bca](https://doi.org/10.1097/MBC.0b013e32835d9bca), indexed in Pubmed: [23322274](https://pubmed.ncbi.nlm.nih.gov/23322274/).
- Yazici H, Poyraz F, Sen N, et al. Relationship between mean platelet volume and left ventricular systolic function in patients with metabolic syndrome and ST-elevation myocardial infarction. *Clin Invest Med.* 2011; 34(6): 330, doi: [10.25011/cim.v34i6.15892](https://doi.org/10.25011/cim.v34i6.15892).
- Acar Z, Aga MT, Kiris A, et al. Mean platelet volume on admission is associated with further left ventricular functions in primary PTCA patients. *Eur Rev Med Pharmacol Sci.* 2012; 16(11): 1567–1569, indexed in Pubmed: [23111972](https://pubmed.ncbi.nlm.nih.gov/23111972/).
- Kim D, Choi DH, Kim BB, et al. Prediction of infarct transmural extent from C-reactive protein level and mean platelet volume in patients with ST-elevation myocardial infarction: comparison of the predictive values of cardiac enzymes. *J Clin Lab Anal.* 2016; 30(6): 930–940, doi: [10.1002/jcla.21959](https://doi.org/10.1002/jcla.21959), indexed in Pubmed: [27075615](https://pubmed.ncbi.nlm.nih.gov/27075615/).
- Maden O, Kacmaz F, Selcuk MT, et al. Relationship of admission haematological indices with infarct-related artery patency in patients with acute ST-segment elevation myocardial infarction treated with primary angioplasty. *Coron Artery Dis.* 2007; 18(8): 639–644, doi: [10.1097/MCA.0b013e3282f0eech](https://doi.org/10.1097/MCA.0b013e3282f0eech), indexed in Pubmed: [18004115](https://pubmed.ncbi.nlm.nih.gov/18004115/).
- Liu Q, Wang T, Chen R, et al. Mean platelet volume predicts left descending artery occlusion in patients with non-ST-elevation myocardial infarction. *Platelets.* 2014; 25(4): 246–251, doi: [10.3109/09537104.2013.810332](https://doi.org/10.3109/09537104.2013.810332), indexed in Pubmed: [24102229](https://pubmed.ncbi.nlm.nih.gov/24102229/).
- Ekici B, Erkan AF, Alhan A, et al. Is mean platelet volume associated with the angiographic severity of coronary artery disease? *Kardiol Pol.* 2013; 71(8): 832–838, doi: [10.5603/KP.2013.0195](https://doi.org/10.5603/KP.2013.0195), indexed in Pubmed: [24049023](https://pubmed.ncbi.nlm.nih.gov/24049023/).
- De Luca G, Verdoia M, Cassetti E, et al. Mean platelet volume is not associated with platelet reactivity and the extent of coronary artery disease in diabetic patients. *Blood Coagul Fibrinolysis.* 2013; 24(6): 619–624, doi: [10.1097/MBC.0b013e328360c75a](https://doi.org/10.1097/MBC.0b013e328360c75a), indexed in Pubmed: [23689272](https://pubmed.ncbi.nlm.nih.gov/23689272/).
- Aurigemma C, Scalone G, Tomai F, et al. Persistent enhanced platelet activation in patients with acute myocardial infarction and coronary microvascular obstruction: clinical implications. *Thromb Haemost.* 2014; 111(1): 122–130, doi: [10.1160/TH13-02-0166](https://doi.org/10.1160/TH13-02-0166), indexed in Pubmed: [24085158](https://pubmed.ncbi.nlm.nih.gov/24085158/).
- Kırış T, Yazici S, Günaydin ZY, et al. The prognostic impact of in-hospital change in mean platelet volume in patients with non-ST-segment elevation myocardial infarction. *Angiology.* 2016; 67(7): 690–696, doi: [10.1177/0003319715627734](https://doi.org/10.1177/0003319715627734), indexed in Pubmed: [26787684](https://pubmed.ncbi.nlm.nih.gov/26787684/).
- Açıkgöz N, Ermiş N, Yağmur J, et al. The evaluation of mean platelet volume levels in patients with idiopathic and ischemic cardiomyopathy: an observational study. *Anadolu Kardiyol Derg.* 2011; 11(7): 595–599, doi: [10.5152/akd.2011.161](https://doi.org/10.5152/akd.2011.161), indexed in Pubmed: [21911318](https://pubmed.ncbi.nlm.nih.gov/21911318/).

Cite this article as: Ozturk S, Gürbüz AS, Efe SÇ, et al. Association of mean platelet volume with presence of non-viable myocardium in ischaemic cardiomyopathy. *Kardiol Pol.* 2017; 75(10): 1013–1019, doi: [10.5603/KPa2017.0136](https://doi.org/10.5603/KPa2017.0136).

Związek średniej objętości płytek ze stanem żywotności miokardium w kardiomiopatii niedokrwiennej

Semi Ozturk¹, Ahmed Seyfettin Gürbüz², Süleyman Çağan Efe³, Mehmet Fatih Yılmaz⁴, Özden Savaş Den Hartigh⁵, Ferahnaz Çınaral Sayın⁵, Cevat Kıрма⁴

¹Department of Cardiology, Haseki Education and Training Hospital, Istanbul, Turcja

²Department of Cardiology, Yunus Emre State Hospital, Eskisehir, Turcja

³Department of Cardiology, Agri State Hospital, Agri, Turcja

⁴Department of Cardiology, Kartal Kosuyolu Heart Training and Research Hospital, Istanbul, Turcja

⁵Department of Nuclear Medicine, Kartal Kosuyolu Heart Training and Research Hospital, Istanbul, Turcja

Streszczenie

Wstęp: Parametry uzyskane na podstawie morfologii krwi, takie jak średnia objętość płytki (MPV), stosunek liczby neutrofilów do limfocytów (NLR), stosunek liczby płytek do limfocytów (PLR) są ostatnio proponowane jako wskaźniki zapalenia, obok białka C-reaktywnego (CRP), klasycznego markera stanu zapalnego. Pojawia się coraz więcej doniesień na temat istotnych związków tych parametrów z miażdżycą i jej powikłaniami.

Cel: Celem niniejszej pracy była analiza zależności między tymi parametrami a cechami żywotności miokardium ocenianymi metodą pozytonowej tomografii emisyjnej (PET) u chorych z kardiomiopatią niedokrwinną.

Metody: Do badania włączono 122 chorych z kardiomiopatią niedokrwinną, u których wykonano PET. Pacjentów podzielono na dwie grupy w zależności od obecności pełnościennej blizny. Grupa 1 składała się z 21 osób, u których stwierdzono bliznę pełnościennej, i uznano ją za grupę z martwicą miokardium. Grupa 2 liczyła 101 chorych, u których stwierdzono hibernację miokardium i/lub bliznę niepełnościennej, co kwalifikowano jako zachowanie żywotności miokardium. Retrospektywnie przeanalizowano parametry hematologiczne w okresie 30 dni przez wykonaniem PET.

Wyniki: Nie stwierdzono istotnych różnic między grupami w odniesieniu do liczby leukocytów, neutrofilów, limfocytów, płytek krwi, stężenia hemoglobiny, dystrybucji erytrocytów czy wartości CRP, PLR i NLR. U chorych z martwicą miokardium wartości MPV były istotnie wyższe ($p = 0,002$). W analizie wieloczynnikowej regresji logistycznej wykazano, że MPV (iloraz szans [OR] = 0,373; 95% przedział ufności [CI] 0,20–0,69; $p = 0,002$) jest niezależnym czynnikiem predykcyjnym braku żywotności miokardium. W analizie krzywych ROC określono punkt odcięcia w celu identyfikacji chorych z martwicą miokardium wynoszący 8,19 (pole pod krzywą: 0,72; 95% CI 0,60–0,84). Wartości MPV powyżej 8,19 cechowały się wrażliwością wynoszącą 76% i swoistością równą 55%.

Wnioski: W niniejszej pracy wykazano, że MPV jest niedrogim, klinicznie użytecznym i mierzonym rutynowo parametrem związanym z obecnością żywotnego miokardium u pacjentów z kardiomiopatią niedokrwinną.

Słowa kluczowe: płytki krwi, pozytonowa tomografia emisyjna, kardiomiopatia

Kardiologia 2017; 75, 10: 1013–1019

Adres do korespondencji:

Dr. Semi Ozturk, Department of Cardiology, Haseki Education and Training Hospital, Istanbul, Turkey, e-mail: semi_ozturk@yahoo.com

Praca wpłynęła: 06.11.2016 r.

Zaakceptowana do druku: 18.05.2017 r.

Data publikacji as AoP: 07.07.2017 r.