Kardiologia Polska 2015; 73, 12: 1310–1316; DOI: 10.5603/KP.a2015.0098 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Relation of neutrophil-to-lymphocyte and platelet-to-lymphocyte ratio with coronary artery disease severity in patients undergoing coronary angiography

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Abstract

Background: Atherosclerosis is a chronic systemic inflammatory disease. Neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are systemic inflammatory markers that are correlated with poor cardiovascular outcomes.

Aim: To explore the relation of NLR and PLR with severity of coronary artery disease (CAD).

Methods: The study population consisted of 180 consecutive patients who underwent elective coronary angiography (CAG). While 100 patients (22 female, mean age: 60.6 ± 12.6 years) had abnormal CAG, 80 patients (44 female, mean age: 57.2 ± 10.9 years) had normal CAG. NLR and PLR were calculated as the ratio of neutrophil count to lymphocyte count and as the ratio of platelet count to lymphocyte count, respectively.

Results: Although age distribution was similar between the two groups ($p = 0.073$), female gender was significantly higher in the normal CAG group ($p < 0.001$). Patients with abnormal CAG had significantly higher NLR and PLR when compared to patients with normal CAG (3.7 ± 2.6 vs. 2.2 ± 1.7, $p < 0.001$ and 125.9 ± 72.3 vs. 102.6 ± 33.8, $p = 0.027$, respectively). NLR and PLR were significantly correlated with SYNTAX score and GENSINI score. In logistic regression analyses, only NLR (odds ratio: 1.576, confidence interval: 1.198–2.072, $p = 0.001$) was an independent predictor of CAD. An NLR of 2.3 or higher predicted the CAD with a sensitivity of 66% and specificity of 70%.

Conclusions: NLR and PLR seem to be a simple method to predict severity of CAD in patients undergoing elective CAG, and it may be part of cardiovascular examination before CAG.

Key words: neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, GENSINI, SYNTAX, coronary artery disease

Kardiol Pol 2015; 73, 12: 1310–1316

INTRODUCTION

Coronary artery disease (CAD) is the leading cause of morbidity and mortality throughout the world [1, 2]. It has a complex pathophysiology, and inflammation seems to play an important role in CAD [3]. Previous studies have shown that higher levels of inflammatory markers are associated with the severity of CAD and worse cardiovascular outcome [4]. Although endothelial damage has been known as the triggering factor for the formation of atherosclerotic plagues, inflammatory process is responsible in the initiation and progression of the atherosclerosis [3]. Inflammatory markers, including white blood cell (WBC), C-reactive protein (CRP), and homocysteine, have been used for the prediction of cardiovascular events in asymptomatic patients [5]. The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have recently been investigated as new predictors for worse cardiovascular outcome [6, 7]. Previous studies have shown that NLR is associated with morbidity and mortality in many cardiovascular diseases such as hypertension, heart failure, infective endocarditis, and acute coronary

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Received: 14.06.2014 **Accepted:** 12.03.2015 **Available as AoP:** 14.05.2015

syndromes (ACS) [5–8]. Although previous data have shown that NLR is associated with severity of CAD [4], its relation with PLR remains unclear. Increased platelet activation plays an important role in the initiation and progression of atherosclerosis. Recent studies have demonstrated that PLR is associated with adverse cardiovascular outcome [7, 9]. Despite the importance of NLR on severity of CAD, the association of PLR with severity of CAD is less well established. The aim of this study was to explore the relation of both NLR and PLR with severity of CAD in patients undergoing elective coronary angiography (CAG), which has not been studied previously.

METHODS

Study population

Between June 2013 and October 2013, 215 consecutive patients who were scheduled for elective CAG were enrolled in the study. Patients with systemic disease and those using of medical treatment to affect the WBC counts, such as the following: haematopoietic system disorders, history of malignancies, and/or treatment with chemotherapy, evidence of any concomitant inflammatory disease, acute infection, and chronic inflammatory status, ACS, and percutaneous coronary intervention within the past six months, history of using glucocorticoid therapy within the past thee months, secondary hypertension, history of chronic renal or hepatic disease, and cerebrovascular disease, were excluded from the study. After exclusion criteria (five patients with malignancy, eight patients with concomitant inflammatory disease, 22 patients with ACS), the remaining 180 patients were included into the study. This study was designed prospectively. All of the study population were evaluated in terms of eligibility for inclusion and exclusion criteria. Patients who were eligible for enrolment gave written informed consent before participating. Clinical data and blood samples were obtained after written informed consent of patients. Subsequently, all patients underwent CAG. According to the results of the CAG, patients were divided into two groups: abnormal or normal CAG. The abnormal CAG group was defined as patients with coronary artery stenosis equal to or greater than 50% and/or slow flow detected in coronary arteries. The normal CAG group was defined as patients with normal coronary arteries or stenosis less than 50% [10].

The investigation complies with the principles outlined in the Declaration of Helsinki. The study was approved by the Local Ethics Committee.

Assessment of severity of coronary artery disease

The severity of CAD was evaluated with both the SYNTAX and the GENSINI scoring systems. The SYNTAX scoring system is an angiographic index used for grading the severity of CAD. Each coronary lesion with a diameter stenosis of at least 50%, in vessels of at least 1.5 mm, was scored. The most recently updated online version (2.1) was used for the calculation of the SYNTAX scores (www.syntaxscore.com) [11]. The GENSINI scoring system was used to determine the severity of CAD [12]. This method defines narrowing of the lumen of the coronary arteries as 1 for 1% to 25% stenosis, 2 for 26% to 50%, 4 for 51% to 75%, 8 for 76% to 90%, 16 for 91% to 99%, and 32 for total occlusion. The score is then multiplied by a factor representing the importance of the lesion's location in the coronary artery system. For the location scores, 5 points were given for left main lesion; 2.5 for proximal left anterior descending (LAD) or left circumflex (LCX) artery; 1.5 for the mid-segment LAD and LCX; 1 for the distal segment of LAD and LCX, first diagonal branch, first obtuse marginal branch, right coronary artery, posterior descending artery, and intermediate artery; and 0.5 for the second diagonal and second obtuse marginal branches. Both scoring systems were calculated by two cardiologists blind to the participants. Then the mean of the two measures was calculated. CAD risk factors including fasting glucose, cholesterol levels, blood pressure, smoking, and socio-demographic data of all patients were recorded.

Laboratory findings and evaluation of cardiovascular risk factors

Complete blood counts, which included total WBC, neutrophils, and lymphocytes, were obtained at the time of admission. NLR was calculated as the ratio of neutrophil count to lymphocyte count, and PLR was calculated as the ratio of platelet count to lymphocyte count. All patients were evaluated for the presence of cardiovascular risk factors such as hypertension, hyperlipidaemia, diabetes mellitus, and smoking status. Hypertension was defined as systolic blood pressure ≥ 140 mm Hg and/or diastolic blood pressure ≥ 90 mmHg, previously diagnosed hypertension, or use of any antihypertensive medications. Hyperlipidaemia was defined as serum total cholesterol ≥ 240 mg/dL, serum triglyceride ≥ 200 mg/dL, low-density lipoprotein cholesterol ≥ 130 mg/dL, previously diagnosed hyperlipidaemia, or use of lipid-lowering medication. Diabetes mellitus was defined as fasting plasma glucose levels more than 126 mg/dL in multiple measurements, previously diagnosed diabetes mellitus, or use of antidiabetic medications such as oral anti-diabetic agents or insulin. Smoking status was defined as the history of tobacco use at admission or in the six months prior to visit. A family history of CAD was considered to be a history of documented CAD or sudden cardiac death in a first-degree relative before the age of 55 years for men and 65 years for women.

Statistical analysis

Statistical analyses were performed using SPSS 20.0 statistical package for Windows. Continuous data were expressed as mean ± standard deviation, while categorical data were presented as percentages. The $\chi 2$ test was used for comparison of categorical variables, while student t-test or Mann-Whitney

Table 1. Baseline characteristics and clinical data of the study population

	Patient group (n = 100)	Control group (n = 80)	P
Age [years]	60.6 ± 12.6	57.2 ± 10.9	0.073
Female gender	22 (22%)	44 (55%)	**< 0.001**
Systolic blood pressure [mm Hg]	135.9 ± 23.0	141.3 ± 18.9	0.051
Diastolic blood pressure [mm Hg]	74.5 ± 11.9	78.2 ± 18.9	**0.005**
Heart rate [bpm]	73.3 ± 13.8	74.4 ± 10.6	0.139
Smoking	51 (51%)	23 (29%)	**0.003**
Alcohol use	10 (10%)	7 (9%)	0.776
SYNTAX score	11.5 ± 7.7	0.5 ± 3.1	**< 0.001**
GENSINI score	51.0 ± 31.8	4.8 ± 13.9	**< 0.001**
ACEI user	21 (21%)	19 (24%)	0.659
ARB user	12 (12%)	15 (19%)	0.208
Calcium antagonist user	8 (8%)	15 (19%)	**0.032**
Beta-blocker user	30 (30%)	19 (24%)	0.349
Statin user	16 (16%)	19 (24%)	0.192
Antidiabetic drug user	19 (19%)	25 (31)	0.057
Diuretic user	1 (1%)	2 (2.5%)	0.435
Antiaggregant drug user	35 (35%)	26 (32%)	0.725

Data are expressed as mean ± standard deviation or as number and percentages; ACEI — angiotensin converting enzyme inhibitor; ARB — angiotensin receptor blocker

U test were used to compare parametric and nonparametric continuous variables, respectively. Normal distribution was assessed by Kolmogorov-Smirnov test. Correlation analysis was performed by Pearson or Spearman's correlation test. Logistic regression analysis was performed to determine the independent predictors of abnormal CAG. Receiver operating characteristic (ROC) curve analysis was performed to determine the cut-off level of NLR to predict the CAD. A value of $p < 0.05$ was considered statistically significant.

RESULTS

The study population consisted of 180 consecutive patients undergoing elective CAG. All members of the study population were divided into two groups according to the results of CAG. While 100 patients had abnormal CAG, 80 patients had normal CAG. Baseline characteristics and clinical data are shown in Table 1. Although age distribution was similar between two groups ($p = 0.073$), female gender was significantly higher in the normal CAG group ($p < 0.001$). While diastolic blood pressure was significantly higher in the normal CAG group ($p = 0.005$), smoking status was significantly higher in the abnormal CAG group ($p = 0.003$). The laboratory findings, NLR, and PLR values are shown in Table 2. Patients with abnormal CAG had significantly higher NLR and PLR when compared to patients with normal CAG ($p < 0.001$, $p = 0.027$, respectively). Patients with abnormal CAG had significantly higher creatinine and aspartate amino transferase levels and significantly lower high-density lipoprotein (HDL), when compared to patients with normal CAG. In correlation analysis, NLR and PLR were significantly correlated with SYNTAX score and GENSINI score (Table 3). We included NLR, PLR, age, HDL, and creatinine into multivariate analysis to determine the independent predictors of CAD. In logistic regression analyses, NLR and HDL were independent predictors of CAD (Table 4). ROC analysis was performed to determine the cut-off value of NLR to predict the CAD. A NLR of 2.3 or higher predicted the CAD with a sensitivity of 66% and specificity of 70% (Fig. 1).

DISCUSSION

In the present study, we demonstrated that patients with abnormal CAG had significantly higher NLR and PLR compared to patients with normal CAG. Moreover, NLR and PLR were correlated with severity of CAD. However, only NLR was an independent predictor of CAD. Our study is unique in its design, in that it combines SYNTAX and GENSINI score in one study.

It is clear that inflammation plays a pivotal role in atherosclerosis [3]. It causes not only cardiovascular disease, but also numerous chronic diseases such as cancers, diabetes mellitus, hypertension, connective tissue disease, and chronic kidney disease [7, 13–15]. In the initiation of atherosclerotic plaque formation, inflammatory molecules are released from endothelial cells of vessels and cause vascular damage [3]. Besides the early phase of atherosclerosis, inflammatory molecules also play an important role in the formation of

Table 2. Comparison of laboratory findings of patient and control groups

	Patient group (n = 100)	Control group (n = 80)	P
Leukocytes [mm^3]	9.16 ± 2.93	7.88 ± 2.28	**0.001**
Neutrophils [mm^3]	6.20 ± 2.85	4.62 ± 2.01	**< 0.001**
Lymphocytes [mm^3]	2.03 ± 0.75	2.45 ± 0.81	**0.001**
Platelets [10^3/mm^3]	220.4 ± 48.3	234.4 ± 65.8	0.233
Haemoglobin [g/dL]	13.5 ± 1.8	13.4 ± 1.5	0.354
Neutrophil-to-lymphocyte ratio	3.7 ± 2.6	2.2 ± 1.7	**< 0.001**
Platelet-to-lymphocyte ratio	125.9 ± 72.3	102.6 ± 33.8	**0.027**
Fasting glucose [mg/dL]	122.6 ± 49.5	117.6 ± 41.9	0.760
Creatinine [mg/dL]	0.98 ± 0.30	0.87 ± 0.35	**< 0.001**
Total cholesterol [mg/dL]	205.6 ± 47.5	212.3 ± 50.2	0.249
Triglycerides [mg/dL]	178.8 ± 79.0	189.1 ± 84.0	0.557
Low-density lipoprotein [mg/dL]	130.8 ± 36.4	127.2 ± 38.8	0.550
High-density lipoprotein [mg/dL]	40.3 ± 9.2	45.9 ± 12.2	**0.001**
AST [U/L]	31.7 ± 18.5	23.8 ± 12.7	**0.002**
ALT [U/L]	25.5 ± 11.8	23.2 ± 11.3	0.130
Sodium [mmol/L]	138.5 ± 4.9	140.6 ± 3.3	**0.001**
Potassium [mmol/L]	4.4 ± 0.5	4.5 ± 0.4	0.056

Data are expressed as mean ± standard deviation; AST — aspartate amino transferase; ALT — alanine amino transferase

Table 3. Correlation of neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio with severity of coronary artery disease

	SYNTAX score		GENSINI score	
	r	p	r	p
Neutrophil-to-lymphocyte ratio	0.407	**< 0.001**	0.413	**< 0.001**
Platelet-to-lymphocyte ratio	0.167	**0.025**	0.164	**0.028**

Table 4. Logistic regression analysis to determine the independent predictors of abnormal coronary angiography

	Odds ratio	95% confidence interval	P
Age [years]	1.023	0.993–1.054	0.134
Neutrophil-to-lymphocyte ratio	1.514	1.145–2.002	**0.004**
Platelet-to-lymphocyte ratio	0.999	0.990–1.008	0.836
High-density lipoprotein	0.951	0.920–0.983	**0.003**
Creatinine	2.537	0.858–7.503	**0.092**

thrombosis due to plaque rupture in ACS [5, 6]. Therefore, it is not surprising that inflammatory markers increase in patients with cardiovascular disease. Although numerous inflammatory markers including CRP, tumour necrosis factor alpha (TNF-α), interleukin (IL)-1, and IL-6 are known as indicators of inflammatory process, recent studies have shown that WBC count and its subtypes are also useful for predicting the inflammatory process in cardiovascular diseases [16]. Leukocytes play an important role in inflammatory processes, and neutrophils are the most abundant type of WBC.

The NLR, which can be derived from the WBC count, is a common, cheap, and reproducible test worldwide. Previous studies have shown that NLR is associated with poor clinical outcomes in various cardiovascular diseases [4–8]. In a study that included 300 patients who were admitted to hospital with ACS, higher NLR (median: 6.03 [4.48–7.9]) was associated with higher 30-day in-hospital mortality [17]. Another study reported by Cho et al. [18] showed that use of combination of NLR and haemoglobin provided valuable information for early risk stratification in patients with ST

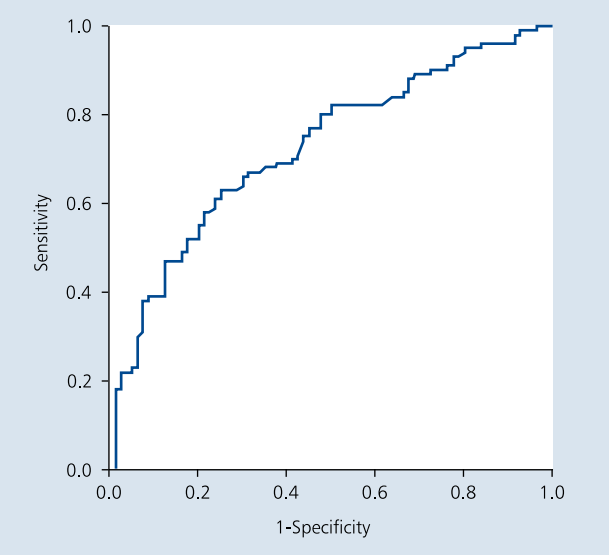

Figure 1. Receiver operating characteristic analysis for neutrophil-to-lymphocyte ratio to predict abnormal coronary angiography (area under curve is 0.726)

elevation myocardial infarction (STEMI). Patients with higher NLR and anaemia had higher mortality rate at six months (hazard ratio 5.6, 95% confidence interval 1.1–27.9, p = 0.036) compared to patients with lower NLR and without anaemia. After coronary artery bypass surgery, NLR is useful to predict saphenous vein graft disease [19]. Recently published trials established that NLR was associated with increased mortality and poor prognosis in ACS, especially STTRMI [5, 6, 17, 18]. Previous studies have shown that severity of CAD is correlated with higher NLR value. Kaya et al. [20] showed that NLR is a predictor of severe atherosclerosis, which may be useful for cardiac risk stratification in patients with CAD. They showed significant correlation between NLR and GENSINI score. In another study, Sönmez et al. [4] showed that NLR is a strong clinical laboratory value that is associated with the presence and complexity of CAD. They showed significant correlation between NLR and SYNTAX score. In our study we showed that NLR is significantly correlated with not only SYNTAX score but also GENSINI score.

Although numerous clinical trials have showed a relation between NLR and poor cardiovascular outcomes in cardiovascular disease, its relation with PLR remains unclear. PLR is also an inflammatory marker of atherosclerosis. Previous studies have shown that higher platelet and lower lymphocyte counts were associated with adverse clinico-pathologic features in some malignancies [21]. There are few clinical trials that demonstrate an association between PLR and poor cardiovascular outcomes. Yildiz et al. [9] showed that higher PLR was associated with poor prognosis in patients with CAD. High PLR (> 160) was an independent predictor of no-reflow in patients presenting with STEMI. Azab et al. [22] showed that higher value of PLR was a predictor of long-term mortality in patients with non-STEMI. Acar et al. [23] demonstrated that patients who had poor collateral circulation had higher PLR. PLR was also studied to evaluate critical limb ischaemia in peripheral arterial occlusive disease. Patients with PLR > 150 had more frequent limb ischaemia than patients with PLR < 150 [24]. In a recent study, we showed that both NLR and PLR were significantly higher in patients with non-dipper hypertension, and only PLR more than 107 was an independent predictor of dipper and non-dipper status [7]. In another recent study, Yüksel et al. [25] reported that PLR level was independently associated with GENSINI score together with WBC, age, and low HDL level in multivariate analysis. However, they did not provide any data about NLR.

Our study has important clinical implications. Similar to CRP, an inflammatuar marker that demonstrates an increased risk of cardiovascular morbidity and mortality, NLR and PLR might be used as promising markers in the prediction of severity of CAD in patients undergoing CAG. These simple, cheap, and readily available biomarkers could help identify individuals at risk for an advanced CAD, who might be candidates for an aggressive therapeutic approach to control cardiovascular risk factors. Although previous studies have used SYNTAX or GENSINI scores separately to evaluate severity of CAD, in our study we used both of them. Additionally, previous studies have only shown significant correlation between NLR and severity of CAD, we also showed significant correlation between PLR and severity of CAD. An important finding of our study is that NLR but not PLR was an independent predictor of abnormal CAG. To the best of our knowledge, our study is the second in the literature, which evaluates the relationship between the PLR and severity of CAD in patients undergoing elective CAG.

Limitations of the study

Our study has several limitations. Firstly, this study was designed as a cross-sectional study. It would be better if we could follow-up the patients and explored the relation between adverse cardiac events and inflammatory parameters. Secondly, we did not evaluate the prognostic value of the NLR and PLR. We did not evaluate the inflammatory markers including TNF-α, IL-1β, IL-6, and highly sensitive CRP in our study population. If we had that data, we could have been compared the NLR and PLR with these inflammatory markers. Other cardiovascular risk factors, including sedentary lifestyle, overweight/obesity (waist circumference and body mass index), known atherosclerosis present in the carotid artery, femoral arteries and aorta, carotid intima media thickness, and ankle-brachial index of patients, were not evaluated in our study population. These factors may also contribute to increased inflammatory process. Finally, our study had a small sample size. Although we showed significant correlation between PLR and severity of CAD, it was not a predictor of

CAD. Therefore, further large-scale, prospective studies are needed to gain more information to predict CAD.

CONCLUSIONS

Patients with CAD had significantly higher NLR and PLR compared to patients with normal CAG. Moreover, NLR and PLR were correlated with severity of CAD. Only NLR of 2.3 or higher was an independent predictor of CAD. NLR and PLR seem to be simple parameters to evaluate severity of CAD in patients undergoing elective CAG, and it could be part of cardiovascular evaluation before coronary angiography.

Conflict of interest: *none declared*

References

1. Lopez AD, Mathers CD, Ezzati M et al. Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. Lancet, 2006; 367: 1747–1757.
2. Onat A, Yazici M, Sari I et al. The Risk Factor Survey of 2003 in Western Turkey Indicates Trend to Declining Coronary Mortality and Urban Overall Mortality. Arch Turk Soc Cardiol, 2003; 31: 762–769.
3. Libby P, Ridker PM, Hansson GK. Inflammation in atherosclerosis: from pathophysiology to practice. J Am Coll Cardiol, 2009; 54: 2129–2138.
4. Sönmez O, Ertaş G, Bacaksız A et al. Relation of neutrophil-to-lymphocyte ratio with the presence and complexiety of coronary artery disease: anobservational study. Anadolu Kardiyol Derg, 2013; 13: 662–667.
5. Guasti L, Dentali F, Castiglioni L et al. Neutrophils and clinical outcomes in patients with acute coronary syndromes and/or cardiac revascularisation. A systematic review on more than 34,000 subjects. Thromb Haemost, 2011; 106: 591–599.
6. Tamhane UU, Aneja S, Montgomery D et al. Association between admission neutrophil to lymphocyte ratio and outcomes in patients with acute coronary syndrome. Am J Cardiol, 2008; 102: 653–657.
7. Sunbul M, Gerin F, Durmus E et al. Neutrophil to lymphocyte and platelet to lymphocyte ratio in patients with dipper versus non-dipper hypertension. Clin Exp Hypertens, 2014; 36: 217–221.
8. Uthamalingam S, Patvardhan EA, Subramanian S et al. Utility of the neutrophil to lymphocyte ratio in predicting long-term outcomes in acute decompensated heart failure. Am J Cardiol, 2011; 107: 433–438.
9. Yildiz A, Yuksel M, Oylumlu M et al. The utility of the platelet-lymphocyte ratio for predicting no reflow in patients with ST-segment elevation myocardial infarction. Clin Appl Thromb Hemost, 2015; 21: 223–228.
10. Sharaf BL, Pepine CJ, Kerensky RA et al. Detailed angiographic analysis of women with suspected ischemic chest pain (pilot phase data from the NHLBI-sponsored Women's Ischemia Syndrome Evaluation [WISE] Study Angiographic Core Laboratory). Am J Cardiol, 2001; 87: 937–941.
11. Sianos G, Morel MA, Kappetein AP et al. The SYNTAX Score: an angiographic tool grading the complexity of coronary artery disease. Euro Intervent, 2005; 1: 219–227.
12. Gensini GG. A more meaningful scoring system for determining the severity of coronary heart disease. Am J Cardiol, 1983; 51: 606.
13. Lee S, Choe JW, Kim HK et al. High-sensitivity C-reactive protein and cancer. J Epidemiol, 2011; 21: 161–168.
14. Pitsavos C, Tampourlou M, Panagiotakos DB et al. Association between low-grade systemic ınflammation and type 2 diabetes mellitus among men and women from the ATTICA study. Rev Diabet Stud, 2007; 4: 98–104.
15. Okyay GU, Inal S, Oneç K, Er RE et al. Neutrophil to lymphocyte ratio in evaluation of inflammation in patients with chronic kidney disease. Ren Fail, 2013; 35: 29–36.
16. Horne BD, Anderson JL, John JM et al. Intermountain Heart Collaborative Study Group. Which white blood cell subtypes predict increased cardiovascular risk? J Am Coll Cardiol, 2005; 45: 1638–1643.
17. Muhammed Suliman MA, Bahnacy Juma AA, Ali Almadhani AA et al. Predictive value of neutrophil to lymphocyte ratio in outcomes of patients with acute coronary syndrome. Arch Med Res, 2010; 41: 618–622.
18. Cho KH, Jeong MH, Ahmed K et al. Value of early risk stratification using hemoglobin level and neutrophil-to-lymphocyte ratio in patients with ST-elevation myocardial ınfarction undergoing primary percutaneous coronary ıntervention. Am J Cardiol, 2011; 107: 849–856.
19. Dogan M, Akyel A, Cimen T et al. Relationship between neutrophil-to-lymphocyte ratio and saphenous vein graft disease in patients with coronary bypass. Clin Appl Thromb Hemost, 2015; 21: 25–59.
20. Kaya H, Ertas F, Islamoglu Y et al. Association between neutrophil to lymphocyte ratio and severity of coronary artery disease. Clin Appl Thromb Hemost, 2014; 20: 50–54.
21. Asher V, Lee J, Innamaa A et al. Preoperative platelet lymphocyte ratio as an independent prognostic marker in ovarian cancer. Clin Transl Oncol, 2011; 13: 499–503.
22. Azab B, Shah N, Akerman M et al. Value of platelet/lymphocyte ratio as a predictor of all-cause mortality after non-ST-elevation myocardial infarction. J Thromb Thrombolysis, 2012; 34: 326–334.
23. Acar G, Kalkan ME, Avci A et al. The relation of platelet-lymphocyte ratio and coronary collateral circulation in patients with stable angina pectoris and chronic total occlusion. Clin Appl Thromb Hemost, 2015; 21: 462–468. doi: 10.1177/1076029613508599.
24. Gary T, Pichler M, Belaj K et al. Platelet-to-lymphocyte ratio: a novel marker for critical limb ischemia in peripheral arterial occlusive disease patients. PLoS One, 2013; 8: e67688.
25. Yüksel M, Yıldız A, Oylumlu M et al. The association between platelet / lymphocyte ratio and coronary artery disease severity. Anadolu Kardiyol Derg, 2015; 15: 640–647. doi: 10.5152/akd.2014.5565.

Cite this article as: 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: 1310–1316. doi: 10.5603/KP.a2015.0098.

Zależność między współczynnikami neutrofile/limfocyty i płytki krwi/limfocyty a zaawansowaniem choroby wieńcowej u chorych poddanych planowej koronarografii

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Streszczenie

Wstęp: Miażdżyca jest przewlekłą chorobą ogólnoustrojową o charakterze zapalnym. Stosunek liczby neutrofili do limfocytów (NLR) i stosunek liczby płytek krwi do limfocytów (PLR) to wskaźniki układowego stanu zapalnego związane z powikłaniami sercowo-naczyniowymi.

Cel: Celem badania była ocena zależności między współczynnikami NLR i PLR a stopniem ciężkości choroby wieńcowej (CAD).

Metody: Badana populacja obejmowała 180 kolejnych pacjentów poddanych koronarografii (CAG) w trybie planowym. U 100 chorych (22 kobiety, średnia wieku: 60,6 ± 12,6 roku) stwierdzono nieprawidłowości w CAG, natomiast u pozostałych 80 chorych (44 kobiety, średnia wieku: 57,2 ± 10,9 roku) obraz w CAG był prawidłowy. Obliczono współczynniki NLR i PLR jako odpowiednio stosunek liczby neutrofili do liczby limfocytów and stosunek liczby płytek krwi do liczby limfocytów.

Wyniki: Mimo że rozkład płci był podobny w obu grupach ($p = 0,073$), w grupie z prawidłowym obrazem w CAG odsetek kobiet był istotnie większy ($p < 0,001$). U chorych z nieprawidłowościami w CAG wartości współczynników NLR i PLR były znamiennie wyższe niż w grupie z prawidłowym obrazem w CAG (odpowiednio 3,7 ± 2,6 *vs.* 2,2 ± 1,7; $p < 0,001$ i 125,9 ± 72,3 *vs.* 102,6 ± 33,8; $p = 0,027$). Współczynniki NLR i PLR korelowały istotnie z punktacją w skali SYNTAX i skali GENSINIEGO. W analizach regresji logistycznej tylko współczynnik NLR (iloraz szans: 1,576; przedział ufności: 1,198–2,072; $p = 0,001$) był niezależnym czynnikiem predykcyjnym CAD. Współczynnik NLR wynoszący 2,3 lub więcej pozwalał prognozować wystąpienie CAD z czułością 66% i swoistością 70%.

Wnioski: Współczynniki NLR i PLR umożliwiają w prosty sposób prognozować o stopniu ciężkości CAD u chorych poddanych koronarografii w trybie planowym i mogą stanowić element badania układu sercowo-naczyniowego przed CAG.

Słowa kluczowe: stosunek liczby neutrofili do limfocytów, stosunek liczby płytek krwi do limfocytów, GENSINI, SYNTAX, choroba wieńcowa

Kardiol Pol 2015; 73, 12: 1310–1316

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Praca wpłynęła: 14.06.2014 r. **Zaakceptowana do druku:** 12.03.2015 r. **Data publikacji AoP:** 14.05.2015 r.