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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Is mean platelet volume associated with the angiographic severity of coronary artery disease?

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Abstract

Background: Platelet activation and aggregation play key roles both in the pathogenesis of atherosclerosis and in the development of acute thrombotic events. Platelet volume is a marker of platelet activation and function, and is measured using mean platelet volume (MPV).

Aim: To determine the relationship between MPV and angiographic Gensini and SYNTAX scores, which give information about the severity and complexity of coronary artery disease (CAD).

Methods: This study included 435 consecutive patients undergoing elective coronary angiography. The complete blood count and biochemical examination of blood were obtained after 12 h of fasting. The independent association between MPV and the severity of CAD was statistically evaluated using PASW Statistics 18 for Windows.

Results: Mean age of the study population was 58.4 ± 9.3 years, of whom 196 were female (45.1%) and 239 male (54.9%). Of the patients, 63.2% had CAD, 31.7% had diabetes mellitus, 61.8% had hypertension, 56.6% had hyperlipidaemia, and 38.6% were smokers. Mean Gensini score was 20.7 ± 31.1. According to Gensini scores, 160 of the patients (36.8%) had normal coronary arteries (Gensini score: 0), 134 of the patients (30.8%) had minimal CAD (Gensini score: 1–19), and 141 of them (32.4%) had severe CAD (Gensini score ≥ 20). Mean MPV values were 8.4 ± 1.0 fL in the group that had no CAD, 8.7 ± 1.0 fL in the group with minimal CAD, and 9.3 ± 1.5 fL in the group with severe CAD. According to Spearman correlation analysis, the positive relationship found between MPV and Gensini score was statistically significant ($p < 0.001$, $r = 0.290$). Likewise, SYNTAX score was also associated with MPV ($p < 0.001$, $r = 0.504$).

Conclusions: We determined a positive correlation between MPV and Gensini and SYNTAX scores. Therefore, this simple haematology test can be used in determining cardiovascular disease burden besides other risk factors during routine clinical practice. For further information about this topic, large-scale studies are needed.

Key words: Gensini score, mean platelet volume, SYNTAX score

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INTRODUCTION

Coronary artery disease (CAD) is the commonest form of heart disease and is a leading cause of death worldwide. Many parameters and tests are used to determine atherosclerotic CAD. Platelets have an important role in the initiation of atherosclerotic lesions and subsequent complications [1]. Increased platelet activity is associated with increased mean platelet volume (MPV). Large platelets that contain more dense granules are metabolically and enzymatically more active than small platelets and have higher thrombotic potential [2, 3]. Platelets represent an important link between inflammation, thrombosis, and atherogenesis [4]. MPV is elevated in patients with acute coronary syndrome (ACS), and is used as an independent predictor of recurrent myocardial infarction (MI) and cardiac death [5]. In this study, we aimed to investigate the relationship between the angiographic severity and complexity of CAD and MPV.

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METHODS

The sample was derived from a population of 812 consecutive patients who underwent coronary angiography due to a positive noninvasive stress test result. In total, 377 of them were excluded because they met the exclusion criteria (n = 342) or did not fulfill the inclusion criteria (n = 35). Finally, 435 patients were enrolled (age 58.4 ± 9.3 years; mean ± SD), including 239 (54.9%) male and 196 (45.1%) female subjects. Our institutional review board approved the study, and we obtained informed consent from all patients. The inclusion criteria were: age greater than 18 years, a coronary angiogram clear enough to enable evaluation of the cause of stress-induced chest pain, and the patient's consent. The exclusion criteria were: current pregnancy, cardiomyopathy, previous MI or any revascularisation procedures (whether percutaneous transluminal coronary angioplasty or coronary artery bypass grafting), unstable angina pectoris, history of congenital heart disease, and any history of platelet disorders.

Selective coronary angiography was performed by the femoral approach using the Judkins technique and General Electric Innova 3100 angiographic system (Buc Cedex, France). Multiple views were obtained, with visualisation of the left anterior descending (LAD) and left circumflex coronary artery in at least four projections, and the right coronary artery in at least two projections. Coronary angiograms were recorded on compact discs in DICOM format. All angiograms were analysed by two cardiologists blinded to the clinical data. The extent and severity of the CAD were evaluated according to the Gensini score [6]. In this scoring system, a severity score is derived for each coronary stenosis based on the degree of luminal narrowing and its topographic importance. Reduction in the lumen diameter and the roentgenographic appearance of concentric lesions and eccentric plaques are evaluated. The severity and complexity of CAD was also evaluated by a new and validated scoring system, the SYNTAX score. This is calculated by a computer program consisting of sequential and interactive self-guided questions. The algorithm consists of 12 main questions. The total SYNTAX score was composed of the individual scores for each separate lesion with a diameter stenosis of ≥ 50% in a vessel of ≥ 1.5 mm in diameter by visual assessment, as previously reported [7]. The 435 patients were divided into four groups on the basis of SYNTAX scores; Control group (SYNTAX score: 0), low SYNTAX score group: Group I (SYNTAX score: 1–22), intermediate SYNTAX score group: Group II (SYNTAX score: 23–32), and high SYNTAX score group: Group III (SYNTAX score: ≥ 33). A complete blood count (CBC) and biochemical examination of blood were performed in all patients before the procedure. CBC analysis including MPV and platelet count was performed using a Beckman Coulter HMX-AL (Brea, CA, USA). Creatinine clearance was calculated according to the Modification of Diet in Renal Disease (MDRD) formula.

Statistical analysis

The data was analysed with the PASW Statistics version 18 software package. The normal distribution of variables was verified with the Kolmogorov-Smirnov test. Spearman's rho correlation was used when one or both of the variables were not normally distributed. Comparisons between the groups were done either with ANOVA when the distribution was normal, or with the Kruskal Wallis test and Mann-Whitney U test when the distribution was not normal. When needed, binary comparisons among the groups were done through Bonferroni correction ($p < 0.017$ was considered statistically significant). A χ^2 test was used to investigate whether distributions of categorical variables differed within groups. Moreover, logistic regression analyses were conducted according to age, sex, diabetes mellitus (DM), hypertension (HT), hyperlipidaemia (HL), smoking, uric acid, MPV, and total cholesterol (TC)/HDL ratio. Multiple regression analysis was performed in order to assess which proportion of variability in Gensini score is determined by MPV. The data is shown as mean ± SD for continuous variables and absolute numbers (%) for dichotomous variables. All analyses were stratified by severity of CAD. A p value of less than 0.05 was considered statistically significant.

RESULTS

Baseline characteristics and biochemical examinations are shown in Table 1. Of the 435 patients, 63.2% had CAD, 31.7% had DM, 61.8% had HT, 56.6% had HL, and 38.6% were current smokers. Mean Gensini scores were 7.1 ± 4.7 and 57.3 ± 30.9 in the minimal and severe CAD groups, respectively. Higher Gensini scores were calculated in men than in women (26.6 ± 33.1; 13.6 ± 26.9, respectively; $p < 0.001$). While there was no correlation between HT and severity of CAD ($p = 0.179$), diabetic and hyperlipidaemic patients and smokers had more severe CAD than the controls ($p = 0.006$; $p < 0.001$; $p < 0.001$, respectively). According to Gensini scores, 160 of the patients (36.8%) had normal coronary arteries (Gensini score: 0), 134 (30.8%) had minimal CAD (Gensini score: 1–19), and 141 (32.4%) had severe CAD (Gensini score ≥ 20). There was a statistically significant relationship between severity of CAD and MPV ($p < 0.017$ for all comparisons, after Bonferroni correction) (Table 2). According to Spearman correlation analysis, the positive relationship determined between MPV and Gensini score was statistically significant ($p < 0.001$, $r = 0.290$; $p < 0.001$, $r = 0.264$ in women; $p < 0.001$, $r = 0.318$ in men). Likewise, SYNTAX score was also associated with MPV ($p < 0.001$, $r = 0.504$; $p < 0.003$, $r = 0.352$ in women; $p < 0.001$, $r = 0.573$ in men) (Fig. 1). According to SYNTAX score, 217 of the patients (49.9%) were enrolled as controls (SYNTAX score: 0), 179 (41.1%) had low SYNTAX scores (SYNTAX score: 1–22), 15 (3.4%) had intermediate SYNTAX scores (SYNTAX score: 23–32), and 24 (5.5%) had high SYNTAX scores (SYNTAX

Table 1. Baseline characteristics for the severity of coronary artery disease

	Control group	Minimal CAD	Severe CAD	P
Age [years]	57.3 ± 9.9	59.3 ± 8.8	58.7 ± 8.9	0.181
LDL-C [mg/dL]	127.0 ± 35.6	130.7 ± 40.1	123.1 ± 35.3	0.239
HDL-C [mg/dL]	46.1 ± 12.7	43.6 ± 11.9	39.6 ± 10.1	**< 0.001**
TC [mg/dL]	197.7 ± 42.8	202.3 ± 45.1	189.2 ± 42.5	**0.043**
TG [mg/dL]	143.5 ± 89.3	163.7 ± 83.5	165.9 ± 95.9	**0.005**
TC/HDL	4.5 ± 1.5	4.9 ± 1.5	5.0 ± 1.6	**0.011**
MDRD Cr Cl [mL/min/1.73 m^2]	96.9 ± 29.3	91.9 ± 26.6	91.4 ± 23.0	0.134
Uric acid [mg/dL]	5.2 ± 1.5	5.6 ± 1.6	5.8 ± 1.4	**0.004**
GGT [U/L]	28.4 ± 18.3	30.0 ± 21.2	32.6 ± 29.8	0.863
Haemoglobin [g/dL]	13.8 ± 1.8	14.7 ± 8.9	14.2 ± 1.7	0.180
MPV [fL]	8.4 ± 1.0	8.7 ± 1.0	9.3 ± 1.5	**< 0.001**
Platelet count [× 10^3/μL]	259.2 ± 68.9	248.7 ± 67.9	237.6 ± 61.2	**0.008**

Severity of coronary artery disease (CAD) was determined by Gensini score. LDL-C — low density lipoprotein cholesterol; HDL-C — high density lipoprotein cholesterol; TC — total cholesterol; TG — triglyceride; MDRD Cr Cl — modification of diet in renal disease creatinine clearance; GGT — gamma-glutamil transferase; MPV — mean platelet volume

Table 2. Bonferroni correction for the severity of coronary artery disease groups

	Controls — minimal CAD (p)	Controls — severe CAD (p)	Minimal CAD — severe CAD (p)
MPV	**0.009**	**0.000**	**0.001**
TC/HDL	0.029	**0.004**	0.644
PLTc	0.055	**0.003**	0.201
HDL	0.072	**0.000**	**0.014**
TG	**0.003**	**0.001**	0.838

Bonferroni correction was performed for the binary comparisons among the groups. A p value of less than 0.017 was considered statistically significant. Severity of coronary artery disease (CAD) was determined by Gensini score; MPV — mean platelet volume; TC — total cholesterol; HDL-C — high density lipoprotein cholesterol; PLTc — platelet count; TG — triglyceride

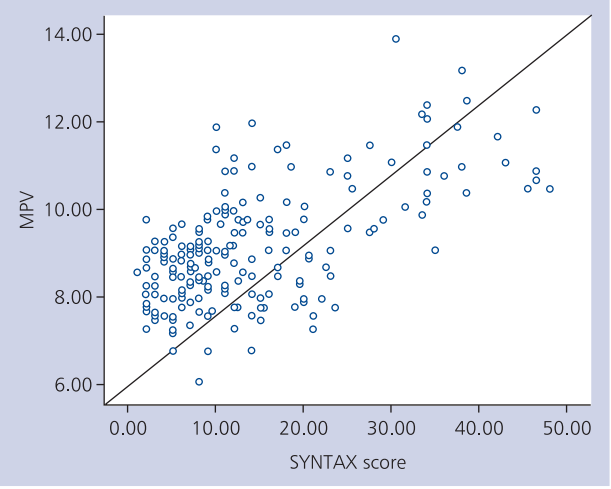

Figure 1. Correlation between SYNTAX score and mean platelet volume (MPV) (p = 0.001, r = 0.504); SYNTAX — Synergy between percutaneous coronary intervention with taxus and cardiac surgery

score ≥ 33). Mean MPV values were 8.4 ± 1.1 fL in the control group, 8.8 ± 1.0 fL in the group with low SYNTAX scores, 10.3 ± 1.4 fL in the group with intermediate SYNTAX scores, and 11.2 ± 1.0 fL in the group with high SYNTAX scores (Fig. 2) (p < 0.008 for all comparisons, after Bonferroni correction). Cutoff values of MPV for predicting the severity of CAD are shown in Figure 3. After adjustment according to traditional risk factors including age, sex, DM, HL, HT, and smoking status, the relationship between MPV and Gensini score maintained its significance (p = 0.009). No significant association was determined between MPV and DM, HT, HL, or smoking (p = 0.103, p = 0.297, p = 0.256, and p = 0.975, respectively). There was a statistically significant negative correlation between platelet count and the severity of CAD determined by Gensini score (p = 0.001, r = –0.166).

A significant inverse relationship was determined between Gensini score and HDL cholesterol (p < 0.001, r = –0.220). Moreover, triglyceride, uric acid levels, and TC/HDL ratio showed a positive correlation with Gensini score (p = 0.005, r = 0.135; p = 0.003, r = 0.156 and p = 0.001, r = 0.157, respectively). According to the Kruskal Wallis test, there were significant differences with regard to HDL, triglyceride, platelet count and TC/HDL ratio in groups with varying severity of CAD. Bonferroni correction was performed for the binary comparisons among the groups and the p value was set at 0.017 (Table 2). In the logistic regression analysis, age, sex, DM, HT, HL, smoking, uric acid, MPV, and TC/HDL ratio were

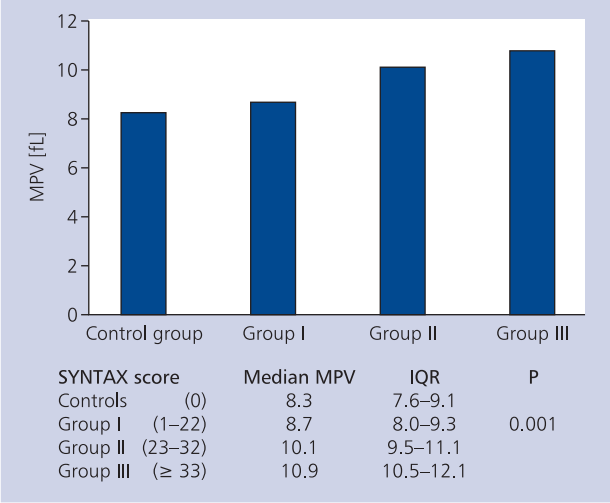

Figure 2. Relationship between the severity and complexity of coronary artery disease and mean platelet volume according to SYNTAX score; IQR — interquartile ranges; MPV — mean platelet volume

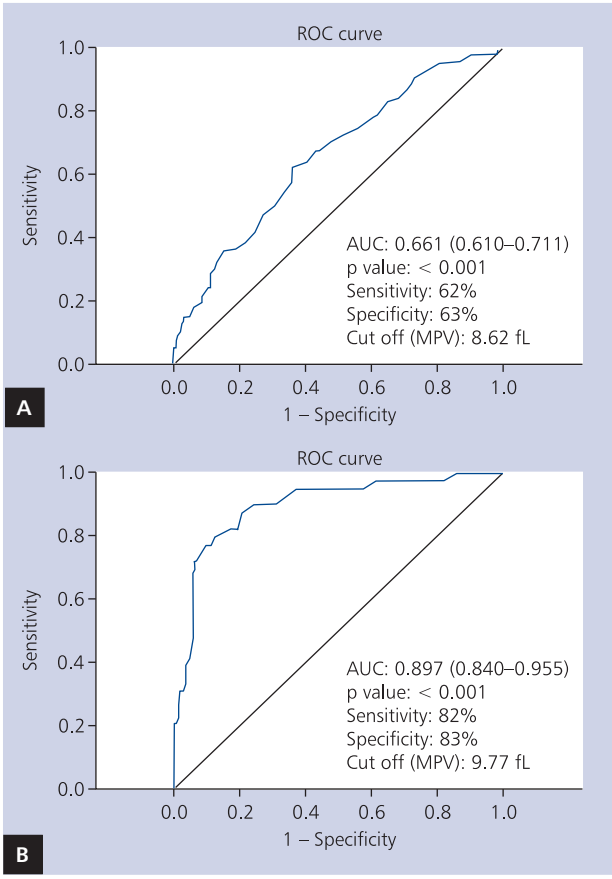

Figure 3. Cut-off values of mean platelet volume (MPV) for predicting the severity of coronary artery disease. Receiver-operating characteristic (ROC) curves for SYNTAX score ≥ 1 (**A**), and ≥ 33 (**B**), respectively

Table 3. Multivariate analysis of coronary artery disease (logistic regression model without interaction)

	P	OR	95% CI
Age	**0.028**	**1.034**	**1.004–1.064**
Sex (male vs. female)	**0.000**	**3.060**	**1.762–5.312**
Diabetes mellitus	**0.028**	**1.897**	**1.073–3.356**
Hypertension	0.130	1.526	0.883–2.639
Hyperlipidaemia	**0.011**	**1.907**	**1.163–3.128**
Smoking	**0.001**	**2.514**	**1.466–4.310**
Uric acid	0.352	1.087	0.912–1.297
MPV	**0.003**	**1.396**	**1.122–1.739**
TC/HDL	0.111	1.151	0.968–1.367

Coronary artery disease was defined as Gensini score > 1; *OR is statistically significant (CI does not include 1); OR — odds ratio; CI — confidence interval; MPV — mean platelet volume; HDL — high density lipoprotein-cholesterol; TC — total cholesterol

Table 4. Relationship between treatment modality and mean platelet volume [fL]; p < 0.001

Medical therapy	8.49 ± 1.04
Percutaneous coronary intervention	9.11 ± 1.22
Coronary artery bypass graft surgery	9.51 ± 1.63

the covariates. While MPV was found to be an independent predictor of CAD (CAD was defined as Gensini score > 1) (odds ratio: 1.396; 95% confidence interval [CI] 1.122–1.739), age, sex, DM, HL, and smoking were also found to affect the severity of CAD (Table 3). Well established risk factors for CAD such as age, male gender, DM, HL, HT, smoking, TC/HDL ratio, and uric acid were added to a multiple regression analysis model to verify whether MPV provides any additional diagnostic information beyond them. While the model was established by the risk factors other than MPV; gender, HL, and HT were found to be significant and 10.8% (R square) proportion of variability in Gensini score was determined by them. When MPV was added to this model, this ratio increased to 19.4% (R square). In this case, MPV provides 8.6% proportion of variability in Gensini score beyond the other risk factors. Furthermore, a statistically significant relationship was found between MPV and treatment modality after coronary angiography (p < 0.001) (Table 4).

DISCUSSION

The widespread availability of particle counters in clinical laboratories now permits routine accurate measurement of platelet volume. Platelets are small, irregularly shaped, clear cell fragments, 2–3 μm in diameter, derived from fragmen-

tation of precursor megakaryocytes. The average lifespan of a platelet is normally 5–9 days. They circulate in the blood of mammals and are involved in haemostasis, leading to the formation of blood clots. Platelets have a pivotal role in the development of CAD. The increase in platelet consumption at the site of the coronary atherosclerotic plaques causes larger platelets to be released from the bone marrow. The fact that the increase persists even after discharge from hospital reinforces the view that platelet volume is chronically larger in infarct patients [8]. MPV is a useful means of identifying larger platelets, which are haemostatically more active and a risk factor for developing coronary atherosclerosis, leading to MI.

It has been reported that elevated MPV is associated with acute MI, mortality following MI, unstable angina pectoris, congestive heart failure, and coronary artery ectasia. Furthermore, platelet volume is a predictor of a further ischaemic event and death when measured after MI [9]. MPV measured before percutaneous coronary intervention has been shown to correlate positively with subsequent restenosis after a successful procedure [10]. The correlation between platelet size and haemostatic reactivity suggests that large platelets have higher thrombotic potential; however, the mechanism underlying the phenomenon remains unclear.

There is some evidence that platelets are involved in atheroprogression. An increase in systemic platelet activation has been described for a variety of atherosclerotic diseases, including CAD [11], transplant vasculopathy [12], and carotid artery disease [13]. Moreover, platelet factor 4 [14] and other platelet-derived chemokines and growth factors [15] are found in human atherosclerotic plaques. Contrary to the literature, in our study there was a statistically significant relationship between severity of CAD and MPV values in patients with stable CAD [16, 17]. This suggests that the increased MPV contributes to the prothrombotic state in CAD, and that larger platelets may play a specific role in the formation of atherosclerotic plaques. Because larger platelets are haemostatically more active, the presence of larger platelets is probably a risk factor for developing coronary thrombosis and MI. Patients with higher MPV values can easily be identified during routine haematological analysis and it can be used in determining cardiovascular disease risk in addition to other risk factors during routine clinical practice.

In addition, to the best of our knowledge, this is the first study to demonstrate that elevated MPV is associated with treatment modality after coronary angiography. Accordingly, increased platelet volume was often associated with the choice of percutaneous coronary intervention and coronary artery bypass grafting surgery.

Therefore, MPV is an important, simple, effortless, and cost effective tool that should be used more extensively to predict the severity of CAD. It has been established that MPV is higher in type 2 diabetic patients than in non-diabetic patients. Among type 2 diabetic patients, MPV is higher in those who have microvascular complications [18]. In contrast to the abovementioned research, there was no relationship between DM and MPV in our study ($p = 0.117$). This may be explained by the fact that diabetic patients had good glycaemic control in our study.

Patients with very low platelet counts ($< 50 \times 10^9$/L) have higher rates of bleeding, whereas those with very high platelet counts ($> 600 \times 10^9$/L) are more likely to develop thrombosis [19]. However, the exact relationship between platelet count and clinical outcome in CAD remains to be elucidated. In previous platelet studies, a significant relationship between platelet count on admission and mortality or major adverse cardiac events (MACE) in patients with ACS has been shown. Ly et al. [20] showed that elevated platelet count on admission was associated with higher rates of MACE at 30 days in patients with ACS. In addition, Gibson et al. [21] demonstrated a significant relationship between high platelet count on admission and reinfarction in 3,491 patients with ST-elevation MI. In contrast to these findings, a statistically negative correlation was found between platelet count and severity of CAD in our study. According to our data, low platelet counts were associated with more severe CAD rather than controls. Some previous studies have reported results similar to ours. Wu et al. [22] demonstrated a U-shaped relationship between platelet count and the risk of mortality and MACE. A lower platelet count might correlate with adverse outcomes. McClure et al. [19] found that among 10,984 patients with ACS, 7% developed thrombocytopenia during hospitalisation. In multivariate analysis, patients with thrombocytopenia had an increased risk of non-fatal MI. The mechanism for the increased risk of bleeding in thrombocytopenia is clear, but the relationship between a low platelet count and increased incidence of MACE has not been fully elucidated. One possible explanation may be consumption of platelets in a thrombus. However, several reports have shown no significant relationship between platelet count and mortality or MACE [23]. Unlike the abovementioned studies, our study was performed in patients with stable CAD. Considering that there is no platelet consumption in stable CAD, an inverse relationship between platelet count and increased severity of the CAD needs to be verified and clarified in further studies.

The TC/HDL ratio is more indicative of cardiovascular disease than TC. High TC and low HDL cholesterol increase the ratio, and so that scenario is undesirable [24]. High serum uric acid level was significantly associated with the severity and complexity of CAD. It has been correlated with several cardiovascular risk factors, endothelial dysfunction, subclinical atherosclerosis, and the severity of coronary atherosclerotic plaques [25]. A long-standing association exists between elevated triglyceride levels and CAD [26]. In accordance with the literature, triglyceride, serum uric acid levels, and TC/HDL ratio showed a positive correlation with severity of CAD in our study. In addition, a statistically significant inverse relationship was determined between Gensini score and HDL cholesterol.

Limitations of the study

Our study has some limitations. First, the study population was relatively small. A larger study population would provide a higher statistical power. In the current study, the patients did not undergo intravascular ultrasonography to assess the coronary plaque burden. Another limitation was that platelet count was not determined visually by peripheral blood smear. Large scale prospective studies are needed to obtain further information.

CONCLUSIONS

Our study showed that elevated MPV values are associated with the severity and complexity of CAD evaluated by Gensini and SYNTAX scores in stable patients.

In regard to the association between severity of CAD and MPV values, this simple haematology test can be used to determine cardiovascular disease burden besides other risk factors during routine clinical practice.

Therefore, we conclude that MPV can be considered a marker of platelet reactivity or a risk factor for CAD. Large-scale prospective, randomised clinical trials are needed to test whether MPV and platelet counts obtained during routine testing are of greater value in terms of diagnosis, risk stratification, and treatment evaluation in patients with stable CAD.

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Czy istnieje związek między średnią objętością płytek krwi a zaawansowaniem choroby wieńcowej?

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Streszczenie

Wstęp: Aktywacja i agregacja płytek krwi odgrywają istotną rolę w patogenezie miażdżycy, a także w rozwoju ostrych zdarzeń zakrzepowo-zatorowych. Objętość płytek krwi, opisywana jako średnia objetość płytek (MPV), jest wyznacznikiem aktywności płytek i ich funkcji.

Cel: Celem badania było określenie zależności między MPV a wskaźnikiem Gensini i SYNTAX score, będącymi liczbową miarą stopnia zaawansowania choroby wieńcowej.

Metody: Badanie przeprowadzono u 435 chorych, u których wykonano planową koronarografię. Krew do badań morfologicznych i biochemicznych pobrano na czczo (12 h od ostatniego posiłku). Niezależny związek między MPV i stopniem nasilenia choroby wieńcowej oceniono za pomocą programu PASW Statistics 18 dla systemu Windows.

Wyniki: Średni wiek pacjentów wynosił 58,4 ± 9,3 roku; 196 (45,1%) osób stanowiły kobiety, a 239 (54,9%) mężczyźni. Wśród badanych u 63,2% zdiagnozowano chorobę wieńcową, u 31,7% — cukrzycę, u 61,8% — nadciśnienie tętnicze, a u 56,6% — hiperlipidemię. Wśród badanych 38,6% osób paliło tytoń. Średni wynik Gensini score był równy 20,7 ± 31,1. Według Gensini score 160 (36,8%) chorych miało prawidłowe tętnice wieńcowe (Gensini score: 0), u 134 (30,8%) pacjentów występowały minimalne oznaki choroby wieńcowej (Gensini score: 1–19), a u pozostałych 141 (32,4%) osób stwierdzono zaawansowaną chorobę wieńcową (Gensini score: ≥ 20). Wartości MPV u pacjentów bez choroby wieńcowej wynosiły średnio 8,4 ± 1,0 fl, w grupie z minimalnym zaawansowaniem choroby wieńcowej — 8,7 ± 1,0 fl, a w grupie z zaawansowaną chorobą wieńcową — 9,3 ± 1,5 fl. Analiza korelacji Spearmana pokazała pozytywny związek między MPV i Gensini score ($p < 0,001$; $r = 0,290$). Podobnie wynik SYNTAX score wiązał się z MPV ($p < 0,001$; $r = 0,504$).

Wnioski: Stwierdzona dodatnia korelacja między Gensini i SYNTAX score a MPV wskazuje, że w rutynowej praktyce lekarskiej proste badanie hematologiczne można wykorzystać do oceny obciążenia chorobami układu sercowo-naczyniowego, oprócz innych czynników ryzyka. Aby potwierdzić tę zależność i uzyskać dodatkowe informacje, należy przeprowadzić badanie w większej grupie chorych.

Słowa kluczowe: Genisini score, SYNTAX score, oznaczanie objętości płytek krwi

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