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Aortic stenosis severity has no influence on automatically measured standard platelet parameters — other cardiovascular risk factors play crucial role

Brak wpływu stopnia zwężenia zastawki aortalnej na standardowe parametry płytek krwi — kluczowa rola innych czynników ryzyka sercowo-naczyniowego

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Abstract

Introduction. Calcification of the aortic valve, the main component of degenerative aortic stenosis (AS), results in turbulent blood flow, higher shear stress and may have an effect on the platelet (PLT) parameters. Platelet function and size, which are easily measured automatically during a complete blood count, are proposed as markers of platelet reactivity and risk factors for cardiovascular diseases.

Material and methods. 143 patients with AS (mean age: 70 ± 13 y., males 76/53%) were enrolled into the study and divided according to the AS severity: severe AS (n = 89; males 43/48.3%; age IQR: 70 [63–75] y.) and non-severe AS (n = 54; males 63/61.1%; age IQR: 70 [62–76] y.). The clinical data were collected and analyzed with special attention being paid to the cardiovascular risk factors, concomitant diseases (coronary artery disease [CAD], diabetes mellitus, arterial hypertension, obesity). Laboratory tests and echocardiography were assessed in all subjects. Routine admission complete blood cell count was obtained — PLT count, mean PLT volume (MPV), PLT distribution width (PDW) and percentage of giant PLT (giant PLT%) were analyzed.

Results. There were no differences between the PLT count, MPV, PDW and giant PLT% when comparing the group with severe AS to the non-severe AS group. Multivariate analysis showed a significant effect of CAD coincidence (β = –0.2, SE = 0.09, p = 0,03) and active smoking (β = –0.2, SE = 0.09, p = 0.03) on the PLT count; obesity (β = 0.2, SE = 0.09, p = 0.03) and CAD (β = –0.2, SE = 0.09, p = 0.03) on MPV; obesity (β = 0.21, SE = 0.09, p = 0.02), thienopyridines (β = 0.19, SE = 0.09, p = 0.03) and LMWH intake (β = 0.21, SE = 0.09, p = 0.02) on PDW; and similarly, obesity (β = 0.23, SE = 0.09, p = 0.01), thienopyridines (β = 0.18, SE = 0.09, p = 0.046) and LMWH intake (β = 0.23, SE = 0.09, p = 0.01) on giant PLT%.

Conclusions. Aortic stenosis severity has no effect on PLT count and morphology that are automatically measured. The coincidence of standard cardiovascular risk factors and the CAD effects on the PLT parameters that are established during a standard complete blood count.

Key words: aortic stenosis, platelet parameters, platelet count, platelet distribution width, percentage of giant platelets, mean platelet volume

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Introduction

Platelets originate in the bone marrow during megakaryopoiesis and are constantly released into the bloodstream. Morphologically, they are devoid of nuclei cells that are filled with granules of vasoactive and prothrombotic factors (including thromboxane A_2, serotonin and ATP). While circulating in the blood, platelets may differ, not only in number, shape or size, but also in their potential to maintain the continuity of the vascular system through complex hemostatic functions, which are regulated by agonists such as ADP, collagen or epinephrine. Their activation causes an increased expression of platelet membrane adhesive molecules such as P-selectin, GP Ib and IIb/IIIa receptors [1–3].

Calcification of the aortic valve, which is the main component of degenerative aortic stenosis (AS), is connected with alterations in shape and the valve orifice, which results in a turbulent blood flow, higher shear stress and may finally have an effect on the platelet parameters. The morphological platelet parameters, which are easily measured automatically during a routine complete blood count, are proposed as markers of platelet reactivity and also as risk factors for cardiovascular diseases.

The sudden and abrupt changes in the direction of blood flow, which occur in AS, are connected with the activation of platelets, hence they induce an increased tendency to form blood clots (as a result of atherothrombotic disease). Peripheral thrombosis is one of the major complications that is associated with aortic valve stenosis [4, 5].

The greater volume of platelets that is observed in patients with AS is associated with the increased metabolic and enzymatic activity of platelets, and this fact is supported by the literature [6]. However, there is still a lack of reliable, evidence-based scientific reports that analyze all of the platelet parameters in patients with aortic stenosis, who are often burdened with both cardiovascular risk factors as well as with coronary heart disease itself. The assessment of platelet parameters remains an undervalued test in routine clinical practice in spite of the wide availability and low assay cost possibly due to the difficulties with their full and comprehensive interpretation. The aim of the study was to assess the affect of AS severity on the PLT count and morphology and to observe whether other clinical factors play important roles in determining the PLT parameters in patients with AS.

Material and methods

One hundred and forty-three patients with newly or previously diagnosed AS who were hospitalized in the Department of Cardiology between 2007 and 2011 were enrolled into the study (mean age: 70 ± 13 y., males 76/53%). The study population was divided into two groups: 1) group 1: patients with severe aortic stenosis (n = 89, males 43/48.3%, age IQR: 70 [63–75] years); 2) group 2: patients with non-severe (mild and moderate) aortic stenosis (n = 54, males 63/61.1%, age IQR, 70 [62–76] years).

All of the necessary clinical data were assessed in all subjects on admission: determination of any concomitant diseases, as well as a physical examination, laboratory tests and echocardiography were performed. The routine complete blood count on admission was obtained with special attention being paid to the platelet parameters.

The standard exclusion criteria that are used in our lab include: a history of liver disease, paraproteinemia, myeloproliferative disorders, myelodysplastic syndrome, end stage renal failure (GFR < 15 ml/min/1,73 m^2), previously diagnosed antiplatelet antibodies, disseminated intravascular coagulation, having undergone cardiopulmonary bypass (in the preceding one month), acute infection, drug intake before hospitalization: nonsteroidal anti-inflammatory drugs (except aspirin), beta-lactam antibiotics, GP IIb/IIIa receptor antagonists, psychotropic drugs and herbal supplements.

Informed consent was obtained from each patient. The study protocol was approved by the Bioethics Committee of the Medical University of Silesia and performed according to the ethical guidelines of the 1975 Declaration of Helsinki.

Clinical data

A detailed medical history was collected from each subject that included: the current course of the disease, the main symptoms (classified in NYHA and CCA class), any concomitant diseases (including coronary artery disease [CAD], diabetes type 2 [DM], arterial hypertension [HA], hyperlipidaemia), family history, precise pharmacotherapy (especially aspirin, thienopyridines, low molecular weight heparins), alcohol and coffee intake and active tobacco smoking. We also collected physical examination parameters including: weight, height, body mass index (BMI), body surface area (BSA) and arterial blood pressure.

Coronary artery disease

In the study we included patients with a history of acute coronary syndrome and/or atherosclerosis of coronary vessels that was confirmed by coronary angiography.

Diabetes type 2

Patients with diagnosed diabetes (new or prior to hospitalization) were taken into consideration.

Obesity

Obesity was defined as BMI ≥ 30 kg/m^2.

Active smoking

Patients who continue to smoke cigarettes.

Arterial hypertension

Arterial hypertension that was diagnosed prior to or during hospitalization was based on the 2007 guidelines for the Management of Arterial Hypertension of the European Society of Hypertension and of the European Society of Cardiology [7].

Laboratory tests

Blood for the laboratory tests was collected from a peripheral vein using EDTA preservative (disodium edetate) between 6:00 and 8:00 a.m. after at least an eight-hour fast. The blood samples were delivered to the laboratory where tests were performed using a Sysmex SF-3000 apparatus within 30 minutes. The time from the receipt of the blood to the final tests did not exceed more than one hour in order to avoid any effect of the EDTA preservative on the platelet parameters. We routinely evaluated the following parameters: total blood platelet count (PLT count), mean platelet volume (MPV), platelet distribution width (PDW) and percentage of giant platelets (giant PLT%). Thrombocytopenia was defined as a PLT count < 150 × 10^3/ml.

Echocardiography

On admission, ECG-gated transthoracic echocardiography was performed in all patients, and in some cases transesophageal echocardiography, in order to visualize the aortic valve more accurately.

An experienced physician took all of the measurements using the same investigation protocol and techniques in order to reduce inter- and intra-observer variability in accordance with the European Society of Cardiology guidelines, especially the European Association of Echocardiography/ /American Society of Echocardiography recommendations on the echocardiographic assessment of valve stenosis [8].

In order to assess the severity of AS the following parameters were assessed: maximal (Pmax) and mean (Pmean) transvalvular pressure gradients, maximal blood velocity (Vmax), aortic valve area (AVA) using planimetry, which were then indexed depending on BSA. Patients with severe aortic stenosis were classified according to the European Society of Cardiology guidelines on the treatment of valvular heart disease (version 2012) [9].

The AS severity was established according to combination of following parameters with cut-off values:

- AVA < 1 cm^2 (most likely when < 0.8 cm^2);
- AVA/BSA < 0.6 cm^2 (preferred in patients with small BSA);
- transvalvular Vmax > 4 m/s;
- transvalvular Pmean > 40 mm Hg.

The parameters of left ventricular (LV) geometry and function, as well as the function of other valves were collected using the two-dimensional left parasternal long-axis view: LV end-diastolic and end-systolic volume, LV ejection fraction, stroke volume, stroke volume index, interventricular septum, LV mass and left ventricular mass index. The investigation was performed using Toshiba Aplio equipment with a 2.5 MHz sector ultrasound transducer and 2–7 MHz transesophageal transducer.

Statistical analysis

Statistical analysis was performed using STATISTICA for Windows (a data analysis software system). All data was collected in a Microsoft Office Excel spreadsheet. The U-Mann-Whitney test was used to compare two non-normally distributed variables, while the t-Student or Cochran-Cox tests were used for the comparison of normally distributed continuous variables when the variances were not homogeneous as tested using the Levene test. A p value of less than 0.05 was considered to indicate statistical significance.

The distribution of variables was tested using the Shapiro-Wilk test and continuous variables are reported as the mean with the standard deviation or the median with the interquartile range (25–75 Q) for non-normal distributions, while categorical variables are reported as absolute numbers and percentages.

Stepwise multivariate analysis was performed with platelets parameters and concomitant diseases, aortic stenosis degree, addictions and drugs as the dependent variables. The following variables were included: age, gender, concomitant diseases/risk factors such as obesity, CAD, HA, DM, current smoking, severe AS presence, aspirin, thienopyridines and low molecular weight heparin intake.

Results

Clinical characteristic

Demographic data, initial clinical assessment, concomitant diseases/risk factors, drugs, platelet and erythrocyte parameters, as well as the echocardiographic data that characterized both groups are included in Tables 1, 2 and 3. The analyzed groups were statistically similar according to age, gender, BSA, BMI, LV ejection fraction, systolic and diastolic blood pressure, frequency of antiplatelet and antiaggregant drug intake. Concomitant diseases/risk factors (HA, DM, CAD, smoking and obesity) were reported equally frequently in both groups, regardless of the degree of aortic stenosis.

Depending on the severity of AS, PLT count, MPV, PDW and giant PLT% were statistically similar when comparing group with severe AS to the non-severe AS group. Active smoking was connected with a decreased PLT count (median [IQR]: 126.5 [115–133] vs 164 [135–197], p < 0.001) and more frequent thrombocytopenia (n/%: 10/100% vs 47/36.4%, p < 0.001) compared to that of non-smokers. Obese patients had a statistically higher giant PLT% (mean ± SD: 35.5 ± 5.8 vs 31.8 ± 8.1, p = 0.004), PDW (median [IQR]: 13.7 [12.8–15.3] vs 12.6 [11.8–14.6], p = 0.002) and MPV (median [IQR]: 11.4 [10.7–11.8] vs 10.6 [10.1–11.3], p = 0.002) than non-obese subjects; similarly, the coincidence of DM was associated with a significantly

Table 1. Clinical characteristic of groups

	Severe AS n = 89	Non-severe AS n = 54	Measure	p value
Age, years	70 (63–75)	70 (62–76)	med. (IQR)	1
Male gender	43/48.3%	33/61.1%	n/%	0.1
Weight [kg]	79.4 ± 13.3 51–115	80.4 ± 12.8 55–114	mean ± SD min–max	0.68
Height [m^2]	166.2 ± 8.8 145–185	167.4 ± 8.3 149–184	mean ± SD min–max	0.44
BMI [kg/m^2]	28.6 ± 4.1 18.5–40	28.7 ± 4.5 19.8–44.5	mean ± SD min–max	0.89
BSA [m^2]	1.9 ± 0.2 1.5–2.4	1.9 ± 0.2 1.5–2.4	mean ± SD min–max	0.61
AVA [cm^2]	0.7 (0.57–0.87)	1.35 (1.0–1.6)	med. (IQR)	< 0.001
AVA plan. [m^2]	0.8 ± 0.3 0.2–1.5	1.3 ± 0.3 0.8–2.0	mean ± SD min–max	< 0.001
AVA/BSA [cm^2/m^2]	0.34 (0.28–0,46)	0.65 (0,50–0,84)	med. (IQR)	< 0.001
Vmax [m^2]	4.5 ± 0.8 2.2–6.8	3.1 ± 0.6 1.7–4.3	mean ± SD min–max	< 0.001
Pmax [mm Hg]	83 ± 29.2 19–180	40.4 ± 14.7 10.7–76	mean ± SD min–max	< 0.001
Pmean [mm Hg]	49 (37–60)	23.5 (16–27)	med. (IQR)	< 0.001
LVEF [%]	58 (48–63)	60 (53–67)	med. (IQR)	0.12
SBP [mm Hg]	129.4 ± 18.3 92–170	129.1 ± 15.7 100–150	mean ± SD min–max	0.95
DBP [mm Hg]	80 (72.5–80)	80 (70–85)	med. (IQR)	0.78

n — number; AS — aortic stenosis; med. — median; IQR — interquartile range; SD — standard deviation; ; min–max — minimal-maximal range; BMI — body mass index; BSA — body surface area; AVA — aortic valve area; AVA plan. — planimetric aortic valve area; AVA/BSA — indexed aortic valve area; Vmax — maximal blood velocity; Pmean — maximum and transvalvular pressure gradient; Pmax — maximum transvalvular pressure gradient; LVEF — left ventricular ejection fraction; SBP — systolic blood pressure; DBP — diastolic blood pressure

Table 2. Characteristic of groups depending on type of taken drug and concomitant disease/risk factor

	Severe AS n = 89	Non-severe AS n = 54	Measure	p value
Obesity	29/36.5%	19/35.9%	n/%	1
Coronary artery disease	52/58.4%	29/53.7%	n/%	0.6
Hypertension	67/75.3%	47/87%	n/%	0.09
Diabetes type 2	23/25.8%	13/24%	n/%	0.8
Smoking	5/5.6%	5/9.6%	n/%	0.5
ASA	74/87%	47/87%	n/%	1
Thienopyridines	7/8.3%	10/18.5%	n/%	0.07
LMWH	10/11.8%	4/7.4%	n /%	0.4

AS — aortic stenosis; n — number; ASA — acetylsalicylic acid intake; LMWH — low molecular weight heparin intake

Table 3. Characteristic of groups depending on laboratory tests

	Severe AS n = 89	Non-severe AS n = 54	Measure	p value
Leukocytes [× 10^3/μl]	6.6 (5.8–8.1)	6.3 (5.5–7.7)	med. (IQR)	0.26
Erythrocytes [× 10^3/ml]	4.5 (4.3–4.8)	4.4 (4.1–4.8)	med. (IQR)	0.4
Hb [g/dl]	13.5 ± 1.5 9–16.5	13.5 ± 1.6 9.5–17.2	mean ± SD min–max	0.94
Ht [%]	41 (37.8–43.4)	40.1 (37.6–42.7)	med. (IQR)	0.56
PLT [× 10^3/μl]	160 (132–188)	163 (129–201)	med. (IQR)	0.9
Thrombocytopenia	36/41.4%	22/40.8%	n/%	0.94
PDW [fl]	13.3 (12.3–15.3)	12.6 (12.1–14)	med. (IQR)	0.1
MPV [fl]	11.1 (10.4–11.8)	10.6 (10.3–11.4)	med. (IQR)	0.1
Giant PLT% [%]	33.8 ± 8 17.8–57.6	32.1 ± 6.8 20–49.8	mean ± SD min–max	0.3

n – number; AS – aortic stenosis; med. – median; IQR – interquartile range; Hb – hemoglobin; SD – standard deviation; min–max – minimal-maximal range; Ht – hematocrit; PLT – blood platelet count; PDW – platelet distribution width; MPV – mean platelet volume; giant PLT% – percentage of giant platelets

increased giant PLT% (mean ± SD: 35.5 ± 6.7 vs 32.4 ± ± 7.7, p = 0.03) and PDW (median [IQR]: 13.6 [12.6–15.9] vs 12.9 [11.9–14.7], p = 0.04). Patients with AS and CAD had a decreased PLT count (median [IQR]: 144 [129–180] vs 170 [151–203], p = 0.003) with a higher prevalence of thrombocytopenia (n/%: 43/53% vs 15/25%, p < 0.001) and increased MPV (median [IQR]: 11.1 [10.4–11.9] vs 10.6 [10.2–11.1], p = 0.02) and giant PLT% (median [IQR]: 34.5 [28.7–39.7] vs 30.8 [27.1–34.8], p = 0.02). The coincidence of HA has no impact on platelets parameters.

Multivariate analysis

Multivariate analysis showed a significant affect of CAD coincidence (β = –0.2, SE = 0.09, p = 0.03) and active smoking (β = –0.2, SE = 0.09, p = 0.03) on the PLT count; obesity (β = 0.2, SE = 0.09, p = 0.03) and CAD (β = –0.2, SE = 0.09, p = 0.03) on MPV; obesity (β = 0.21, SE = 0.09, p = 0.02), thienopyridines (β = 0.19, SE = 0.09, p = 0.03) and LMWH intake (β = 0.21, SE= 0.09, p = 0.02) on PDW, and similarly, obesity (β = 0.23, SE = 0.09, p = 0.01), thienopyridines (β = 0.18, SE = 0.09, p = 0.046) and LMWH intake (β = 0.23, SE = 0.09, p = 0.01) on giant PLT%. We found only trend to significance when analyzing the impact of diabetes mellitus on the giant PLT% (β = 0.15, SE = 0.08, p = 0.07), with no impact on the other parameters.

Discussion

The paper is one the first on broad analyses of platelet characteristics in aortic stenosis depending on its severity and cardiovascular comorbidities. No affect on the analyzed parameter was observed when the entire group with a stenotic aortic valve was assessed, MPV, PLT count, giant PLT% and PDW did not differ. The analyzed group, regardless of aortic stenosis severity, severe or non-severe, was homogeneous as to age, gender, BMI, BSA and any anti-platelet agents they were taking. A similar incidence of additional analyzed risk factors and cardiovascular diseases such as CAD, DM, obesity, smoking and HA was observed.

In the majority of papers that were focused on platelet parameters in patients suffering from particular cardiovascular disease without aortic stenosis, a significant affect of anti-platelet agents, which can interfere with the an accurate measurement of platelet characteristics, thereby causing dysfunction in the secondary platelets, was avoided [10]. Among the analyzed platelet parameters, a lower PLT count in individuals that had been treated with thienopyridines, a greater giant PLT% in those that had been treated with thienopyridines as well as patients who had been treated with LMWH and a greater PDW count in individuals who had been treated with thienopyridines were observed.

The available studies broadly assess platelet characteristics in patients with acute coronary syndrome in comparison with patients without CAD or with stable CAD. Greater values of MPV were described [11]. Other papers reported a significantly reduced PLT count [12]. A greater MPV with a normal PLT count were observed in individuals with stable CAD.

In the analyzed patient subpopulation with AS and CAD, a greater MPV and lower PLT often resulting in an abnormally low PLT count as well as a significantly increased giant PLT% were observed. Moreover, diabetes type 2 predisposed individuals to a significantly greater giant PLT% and PDW. Numerous papers report increased MPV and PDW in individuals with isolated type 2 diabetes [13]. However in analyzed population of patients, diabetes type 2 was no significant independent parameter influencing on platelets parameters, simultaneously this excludes interfering impact of the most potent factor on the statistical analysis of other factors. It is worth to mention, that performed stepwise multivariate

analysis revealed tendency to significant influence of diabetes type 2 on giant PLT%.

An increased giant PLT%, MPV and PDW with no affect on PLT count in comparison to normal body mass individuals were observed in obese patients. Papers that analyze the effect of body mass on platelets report a greater MPV in obese patients followed by its normalization after weight loss [14].

There is evidence of a greater MPV with subsequent decrease in the PLT count with no PDW change in patients who smoke. After smoking cessation, MPV and PLT normalizes after three months [15]. Due to contradictory reports, the effect of smoking on MPV remains unclear [16, 17]. Our analysis confirms a significant PLT decrease as a result of smoking in patients with AS.

We did not observe any significant affect of HA coincidence on the variability of platelet characteristics. Available data regarding an isolated HA effect is inconsistent — some papers deny that one exists [1], while others describe it as significant [18].

Platelet size may be correlated with their activation and escalated function. Increased MPV was described in diseases such as type 2 diabetes, hypercholesterolemia, obesity, AMI, ischaemic stroke, metabolic syndrome, preeclampsia or renal artery stenosis, which that are known to be risk factors, or as a result of atherosclerosis [1, 19].

It is worth mentioning that MPV is known to be an adverse prognostic factor for MI, coronary stent restenosis, as well as preeclampsia. There are also numerous rationales that suggest that MPV may be treated as an independent risk factor of cardiovascular diseases [1, 19]. A greater MPV is correlated with increased platelet activity and a decreased bleeding time [4, 19]. New parameters are actively being researched. PDW has recently been postulated to be a more specific platelet activity marker. The change in shape from disk to sphere with subsequent pseudopodia formation from a cell membrane probably takes place during platelet activation, which results in a change in PDW [20].

It is worth to mention that high percentage of patient taking aspirin resulted from extended indications, according to guidelines valid in year 2004 [21].

Basic platelet characteristics appear to be genetically regulated during megakaryopoiesis [22]. Reactivity, activation state and finally inter-individual variability in healthy subjects can disrupt measurements and proper deduction. In clinical practice, repeated measurements and assessments of all of the available platelet parameters may result in better knowledge about the function and activation of platelets.

Limitations of the study

The wide range of platelet parameter values that can be observed in the literature may be caused by the factors that are listed or the dysfunction of platelets [10] as well as a lack of a proper laboratory measurement standardization [23, 24]. The risk factors were not isolated in the analyzed group. Many factors, which appear to have key roles, are correlated as risk factors and the result of atherosclerosis.

Conclusions

1. In patients with AS, the severity of stenosis has no affect on the platelet count and morphology. 2. The coincidence of standard cardiovascular risk factors and the CAD in patients with AS effects on the PLT parameters that are established during a standard complete blood count.

Conflict of interest(s)

The authors declare no conflict of interest.

Streszczenie

Wstęp. Kalcyfikacje zastawki aortalnej, będące głównym komponentem degeneracyjnego zwężenia zastawki aortalnej (AS), skutkują turbulentnym przepływem krwi, co z kolei może wpływać na parametry płytek krwi (PLT). Funkcja i wielkość PLT, łatwo oznaczane podczas rutynowej morfologii krwi obwodowej, mogą być markerami aktywności PLT i potencjalnymi czynnikami ryzyka chorób układu sercowo-naczyniowego.

Materiał i metody. Do badania włączono 143 pacjentów z AS (średni wiek: 70 ± 13 l., mężczyźni 76/53%) i podzielono na dwie podgrupy zależnie od ciężkości AS — z ciężką AS (mediana wieku: 70 [63–75] l., n = 89, mężczyźni 43/48,3%) oraz niecięższą AS (mediana wieku: 70 [62–76] l., n = 54, mężczyźni 63/61,1%). U wszystkich chorych wykonano przezklatkowe badanie echokardiograficzne oraz zebrano wywiad lekarski, ze szczególnym uwzględnieniem czynników ryzyka chorób układu sercowo-naczyniowego i schorzeń współistniejących (choroby wieńcowej [CAD], cukrzycy typu 2, nadciśnienia tętniczego, otyłości, palenia tytoniu). W wykonanej rutynowo morfologii krwi obwodowej analizie poddano następujące parametry płytkowe: liczbę płytek (PLT *count*), średnią objętość płytek (MPV), wskaźnik anizocytozy płytek (PDW), odsetek płytek olbrzymich (*giant* PLT%).

Wyniki. Nie obserwowano istotnej różnicy pod względem PLT *count*, MPV, PDW ani *giant* PLT%, porównując grupy pod względem ciężkości AS. W przeprowadzonej analizie wieloczynnikowej wykazano istotny wpływ współwystępowania CAD (β = –0,2; SE = 0,09; p = 0,03) i palenia tytoniu (β = –0,2; SE = 0,09; p = 0,03) na PLT *count*; otyłości (β = 0,2; SE = 0,09; p = 0,03) i CAD (β = –0,2; SE = 0,09; p = 0,03) na MPV; otyłości (β = 0,21; SE = 0,09; p = 0,02), stosowania pochodnych tienopirydyn (β = 0,19; SE = 0,09; p = 0,03) oraz heparyn drobnocząsteczkowych (β = 0,21; SE = 0,09; p = 0,02) na PDW; i podobnie, otyłości (β = 0,23; SE = 0,09; p = 0,01), stosowania pochodnych tienopirydyn (β = 0,18; SE = 0,09; p = 0,046) oraz heparyn drobnocząsteczkowych (β = 0,23; SE = 0,09; p = 0,01) na *giant* PLT%.

Wnioski. Stopień AS nie wpływa na automatycznie mierzoną liczbę i morfologię PLT. Współwystępowanie czynników ryzyka chorób układu sercowo-naczyniowego oraz CAD wpływa na rutynowo oznaczane w morfologii krwi obwodowej parametry płytkowe.

Słowa kluczowe: zwężenie zastawki aortalnej, liczba płytek krwi, wskaźnik anizocytozy płytek krwi, odsetek płytek olbrzymich, średnia objętość płytek krwi

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