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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Platelet distribution width and plateletcrit: novel biomarkers of ST elevation myocardial infarction in young patients

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Abstract

Background: Platelets play a central role in myocardial infarction, and platelet activity can be evaluated with platelet indices, including platelet distribution width (PDW) and plateletcrit (PCT). These indices have been demonstrated as markers of prothrombotic state in cardiovascular diseases.

Aim: Therefore, we aimed to investigate, the usefulness of these biomarkers in ST-elevation myocardial infarction (STEMI) in young patients.

Methods: This cross-sectional study consisted of 565 subjects who were classified into three groups: group 1 (168 young patients with STEMI), group 2 (173 non-young patients with STEMI), and group 3 (224 age-matched controls with angiographically normal coronary arteries). Male patients aged under 45 years and female patients aged under 55 years were defined as young STEMI.

Results: In group 1, PDW and PCT (17.2 ± 0.67, 0.249 ± 0.05, respectively) were significantly higher than the other groups (group 2, 16.4 ± 0.56, 0.231 ± 0.04; group 3, 15.1 ± 0.63, 0.227 ± 0.04). PDW and PCT had moderate negative correlation ($r = –0.305$, $r = –0.330$, respectively) with age and moderate positive correlation with peak creatine kinase MB ($r = 0.259$, $r = 0.320$, respectively). At multivariate analysis, adjusted for other factors, 1 fL increase in PDW levels was 13.5% more likely to be associated with young STEMI, and similarly, a 1% increase in PCT levels was 18.9% more likely associated with young STEMI.

Conclusions: Platelet distribution width and plateletcrit levels seem to be independent markers of STEMI in young patients and may reflect prothrombotic state in this specific population.

Key words: plateletcrit, platelet distribution width, ST elevation myocardial infarction, young patients

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INTRODUCTION

Platelets play a pivotal role in the atherosclerotic progression of acute coronary syndromes (ACS) [1–4]. Haematological determinants of platelets such as mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) can easily be evaluated in routine laboratory examination. Previous reports emphasise that elevated levels of MPV were associated with a high risk in ACS [5, 6]. PDW, which is a novel marker for coronary artery disease (CAD), measures the variability of platelet size [7]. Also, PCT — which is similar to haematocrit for erythrocytes — displays the number of platelets in a unit volume of blood, and an increased PCT level correlated with worse cardiovascular outcomes in CAD [8, 9].

Although myocardial infarction (MI) is mainly a disease of older ages, young people can suffer from it too [10]. Therefore, novel biomarkers of MI in young patients have gained some attention and, in this context, haematological determinants of platelets may provide useful data. In a recent study, elevated MPV was found to be an independent predictor of ST-segment elevation myocardial infarction (STEMI) in young patients [11]. However, to our knowledge, no studies have investigated other platelet parameters, namely PDW and PCT, in young patients with STEMI.

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We assessed the clinical value of PDW and PCT in young patients with STEMI in this particular study.

METHODS

Patient population

This cross-sectional study was conducted in Yuksek Ihtisas Training and Research Hospital in Turkey between January 2010 and January 2015. Consistent with the Declaration of Helsinki, this study was approved by the Institutional Ethics Committee. Informed consent was obtained from all participants. STEMI was diagnosed based on the criteria recommended by the American College of Cardiology and European Society of Cardiology guidelines [12, 13]. Exclusion criteria were active infection, haematological (including anaemia), oncological, or inflammatory diseases, renal or hepatic insufficiency, severe valvular disease, and hypo- and hyperthyroidism.

However, there were discrepancies in the cut-off age for the definition of young adults with MI, we determined the age cut off as 45 years for men and 55 years for women, with regard to previous literature [11]. We specified 168 patients as young patients with STEMI and attributed these patients as group 1. In total 173 non-young, sex-matched STEMI patients were selected from the rest of the patients with STEMI and included in group 2. Finally, group 3, an age- and sex-matched control group consisted of 224 patients who were admitted to our hospital with suspicion of stable angina pectoris and were found to have normal coronary arteries in coronary angiography.

Echocardiographic examination was performed to evaluate left ventricular ejection fraction (LVEF), left ventricular diameters, mechanical complications, right ventricular function, and valvular pathologies (Vivid 7 GE Medical System, Horten, Norway). Hypertension was defined as receiving antihypertensive therapy, having an arterial blood pressure (BP) > 140 mm Hg systolic and/or 90 mm Hg diastolic BP, or both. Diabetes mellitus (DM) was diagnosed with the use of an antidiabetic drug and a fasting blood glucose > 126 mg/dL. Hyperlipidaemia was considered as total cholesterol > 200 mg/dL, low-density lipoprotein-cholesterol (LDL-C) > 130 mg/dL, and triglyceride > 150 mg/dL or receiving lipid-lowering medication. Patients, who were smoking or had quit smoking within the last year were classed as smokers. A family history of premature CAD was defined as CAD in a parent or sibling diagnosed under the age of 55 years for men and 65 years for women.

Laboratory parameters

In all patients, blood samples were drawn at admission before starting any medication via atraumatic puncture of the antecubital vein. Dry tubes for biochemical tests and tubes with EDTA for the haematological test were used. Erythrocyte count, haemoglobin, haematocrit, and white blood cell count were measured using an automated haematology analyser Coulter Counter LH Series (Beckman Coulter Inc., Hialeah, Florida). The biochemical measurements were determined using an automated chemistry analyser (Abbott Aeroset, Abbott Laboratories, Abbott Park, IL, USA). Creatine kinase MB (CK-MB) was measured using an auto-analyser (COBAS MIRA, Roche, Switzerland).

Coronary angiography and primary percutaneous coronary intervention

In our tertiary heart centre, a staff cardiologist is always available in the emergency room. As soon as the diagnosis of STEMI was made, the patients were delivered to the catheter room. Our average door-to-balloon time was under 30 min. All patients underwent coronary angiography by the Judkins technique using left and right coronary catheters. Coronary angiography followed by primary percutaneous coronary intervention (PCI) with the conventional technique was performed to achieve adequate blood flow for infarct-related artery. The procedural decisions, including device selection and another adjunctive pharmacotherapy, were made at the discretion of the individual PCI operators. Intravenous unfractionated heparin 100 IU/kg, 600 mg clopidogrel, and 300 mg aspirin were given to all patients in the emergency department before PCI procedure. The post-intervention antiplatelet regimen included lifelong acetylsalicylic acid (75–150 mg/day) and clopidogrel (75 mg/day) for at least 12 months.

Statistical analysis

Continuous variables were tested for normal distribution by the Kolmogorov-Smirnov test. We reported continuous data as mean value and standard deviation, and categorical variables as percentages. We compared parametric variables using Student's t-test or the Mann-Whitney U-test between the groups. Categorical variables were compared with the χ^2 test. The correlations between PDW, PCT, and other clinical and laboratory parameters were performed with Pearson and Spearman correlation analysis. The effects of different variables on young MI were calculated in univariate analysis for each. Stepwise selection procedure with a level of 0.05 was used to identify important associations, and these associations were included in the multivariate logistic regression full model.

Multivariate logistic regression analysis was performed to evaluate the importance of platelet indices' in predicting STEMI in young patients. A p-value of < 0.05 was considered significant, and the confidence interval (CI) was 95%. All statistical analyses were performed with SPSS version 20 (SPSS Inc., Chicago, IL, USA).

RESULTS

Baseline characteristics and laboratory findings of groups are shown in Table 1. There were no significant differences in gender, the presence of DM, smoking, family history of CAD, LVEF, MI localisation, and peak CK-MB between group 1 and 2.

Table 1. Baseline characteristics and laboratory parameters of groups

Variables	Group 1 (n = 168)	Group 2 (n = 173)	Group 3 (n = 224)	p1 Group 1 and 2	p2 Group 1 and 3
Age [years]	41.5 ± 4.7	54 ± 8	43.4 ± 8.5	< 0.001	0.099
Gender (male)	122 (72.8%)	135 (78%)	146 (65%)	0.321	0.182
Diabetes mellitus	36 (21.4%)	42 (24.4%)	32 (14.3%)	0.506	0.132
Smoking	72 (42.9%)	75 (43.4%)	84 (41.9%)	0.921	0.457
Family history of CAD	59 (35.1%)	45 (26%)	38 (17%)	0.068	0.001
Hypertension	35 (20.8%)	61(35.5%)	17(15.2%)	0.003	0.232
Hyperlipidaemia	33 (19.6%)	53 (30.8%)	38 (17.0%)	0.018	0.572
BMI [kg/m^2]	23.8 ± 3.0	24.2 ± 3.8	23.1 ± 3.6	0.116	0.304
Glucose [mg/dL]	118 ± 51.2	119 ± 51.4	108.1 ± 40.8	0.881	0.123
Urea [mg/dL]	31.2 ± 33.5	31.9 ± 11.8	34.4 ± 21.9	0.800	0.393
Creatinine [mg/dL]	0.81 ± 0.21	0.86 ± 0.23	0.82 ± 0.16	0.042	0.724
Haemoglobin [mg/dL]	14.1 ± 1.78	14.0 ± 1.6	13.9 ± 1.3	0.890	0.321
WBC [10^3 μL]	12.9 ± 4.9	12.6 ± 4.6	7.2 ± 1.9	0.521	< 0.001
Neutrophil [10^3 μL]	9.7 ± 5.0	9.4 ± 4.1	4.4 ± 1.5	0.546	< 0.001
Lymphocyte [10^3 μL]	2.4 ± 1.7	2.1 ± 1.3	2.5 ± 0.7	0.132	0.015
Total cholesterol [mg/dL]	177.2 ± 38	178.9 ± 42.1	181.2 ± 43.5	0.713	0.463
LDL-C [mg/dL]	108.7 ± 36.4	112.8 ± 36.2	111.6 ± 32.5	0.332	0.501
HDL-C [mg/dL]	36.2 ± 9.5	36.5 ± 8.5	43.7 ± 12	0.788	< 0.001
Triglyceride [mg/dL]	170.4 ± 102.9	157.6 ± 95.7	150 ± 101.9	0.263	0.139
LVEF [%]	47.06 ± 8.8	46.98 ± 8.2	57.4 ± 2.4	0.933	< 0.001
Infarct-related artery:				0.005	
LAD	93 (56%)	77 (45.3%)		0.049	
RCA	59 (35.5%)	58 (34.1%)		0.784	
CX	14 (8.4%)	35 (20.6%)		0.002	
Peak CK-MB [IU/L]	71.4 ± 26.6	70.2 ± 26.8		0.059	
MPV [fL]	8.8 ± 0.98	8.6 ± 0.88	8.5 ± 0.71	0.022	0.003
PDW [%]	17.2 ± 0.67	16.4 ± 0.56	15.1 ± 0.63	< 0.001	< 0.001
PCT [%]	0.249 ± 0.05	0.231 ± 0.04	0.227 ± 0.04	< 0.001	0.001
Platelet count [10^3 μL]	278.1 ± 66.8	273.1 ± 62.9	272 ± 71.2	0.176	0.532
MI localisation:				0.240	
Anterior	89 (53%)	76 (43.9%)		0.118	
Inferior	71 (42%)	86 (49.7%)		0.168	
Lateral	8 (4.8%)	11 (6.4%)		0.521	

Data are expressed as mean ± standard deviation for normally distributed parametric variables and percentage for categorical variables; BMI — body mass index; CAD — coronary artery disease; CX — circumflex artery; HDL-C — high-density lipoprotein cholesterol; LAD — left anterior descending artery; LDL-C — low-density lipoprotein cholesterol; LVEF — left ventricular ejection fraction; MI — myocardial infarction; MPV — mean platelet volume; PCT — plateletcrit; PDW — platelet distribution width; RCA — right coronary artery; RDW — red cell distribution width; WBC — white blood cell

Age, the prevalence of hypertension, hyperlipidaemia, and creatinine levels were significantly higher in group 2 compared with group 1.

Compared with group 3, in group 1 the presence of family history of CAD, white blood cells (WBC), neutrophils, and lymphocytes were significantly higher than in group 3, and high-density lipoprotein (HDL), cholesterol, and LVEF were lower than in group 3.

The analysis of the platelet indices between groups is shown in Table 1 and Figure 1. In group 1, PLT count, MPV, PDW, and PCT were significantly higher than in both group 2 and 3. In the correlation analysis, PDW and PCT had a negative moderate correlation with age ($r = –0.305$, $p < 0.001$ and $r = –0.330$, $p < 0.001$, respectively, Fig. 2) and moderate positive correlation with peak CK-MB ($r = 0.259$, $p < 0.001$ and $r = 0.320$, $p < 0.001$, respectively, Fig. 3).

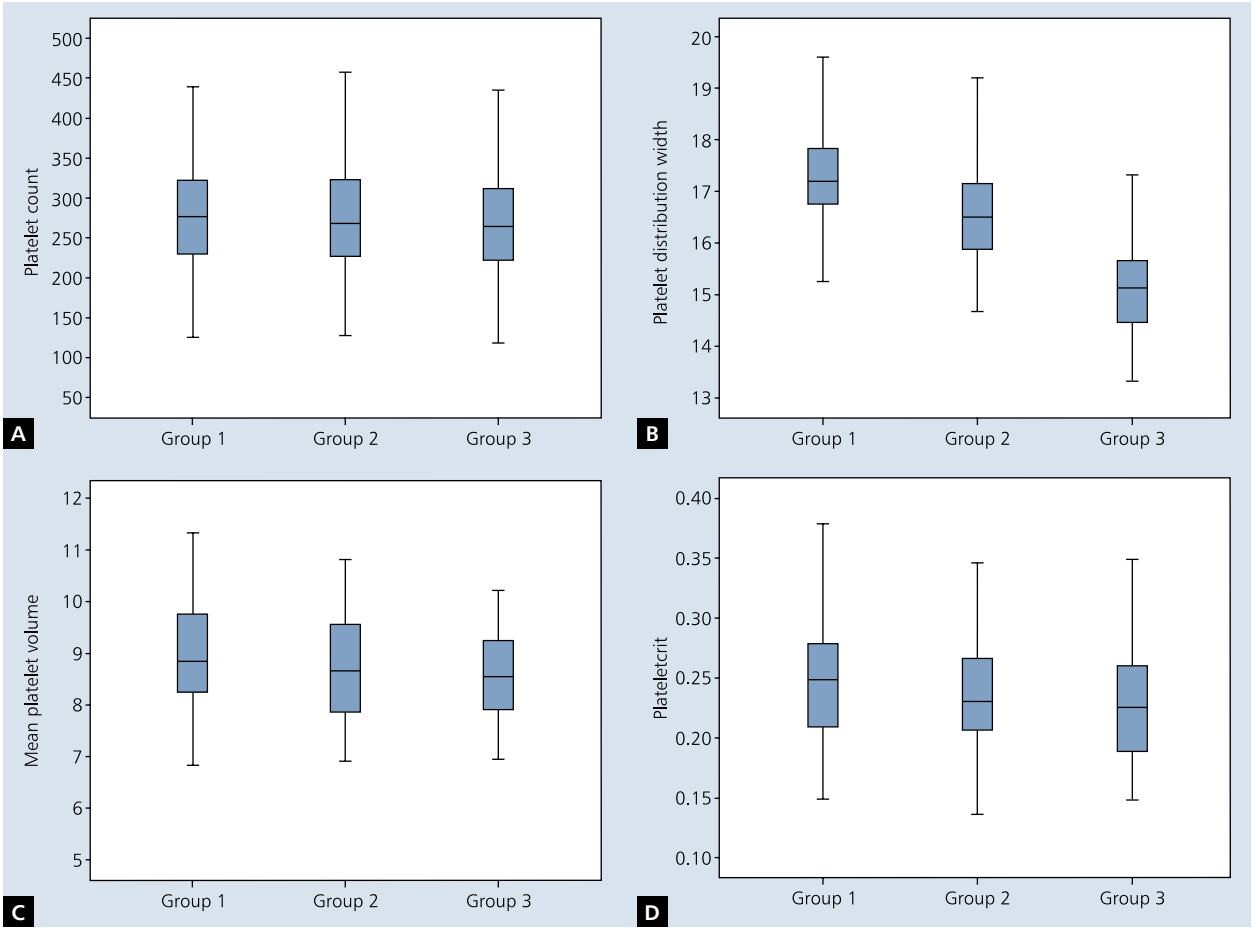

Figure 1. Box-plots showing the distributions of platelet count (**A**), platelet distribution width (**B**), mean platelet volume (**C**) and platelecrit (**D**) at groups 1, 2 and 3

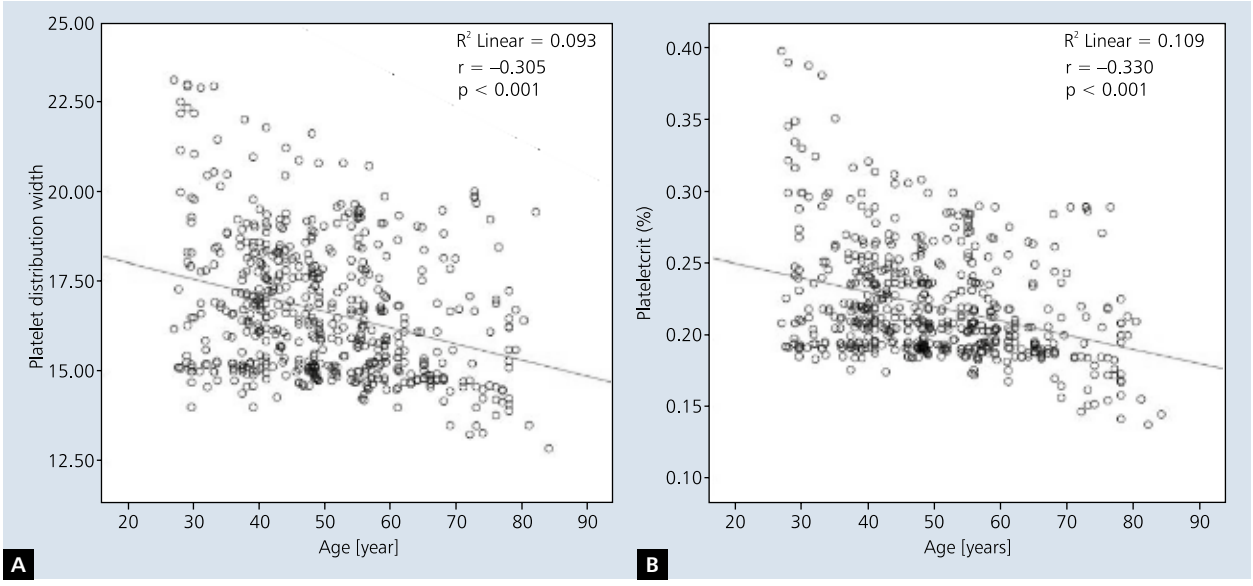

Figure 2. Scatterplots representing the relationships of platelet distribution width (**A**) and platelecrit (**B**) between age

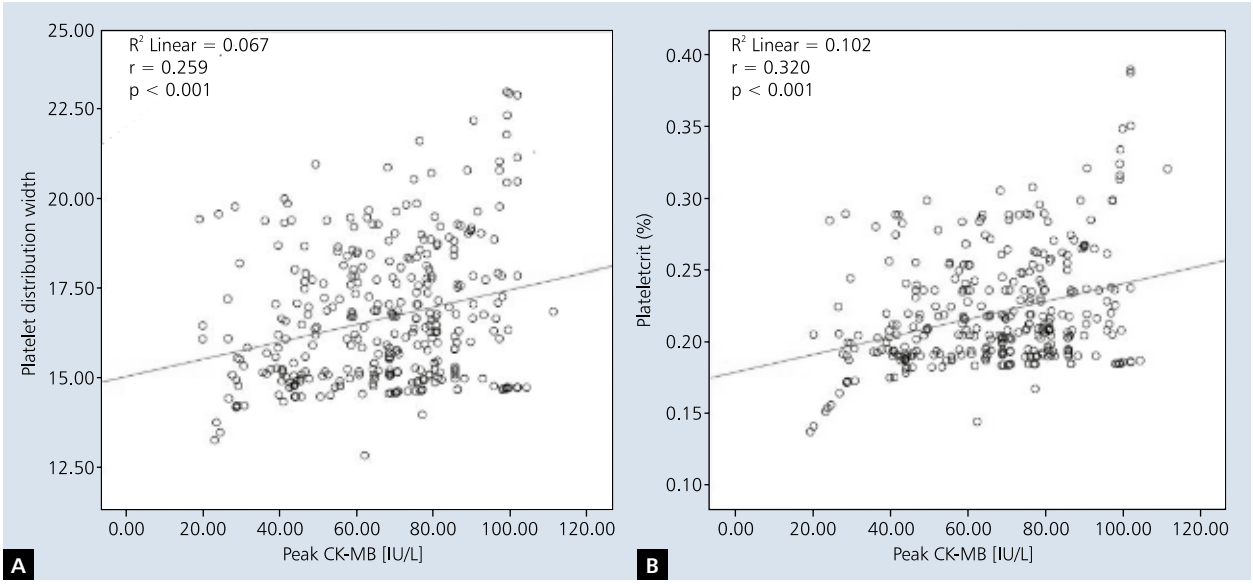

Figure 3. Scatterplots representing the relationships of platelet distribution width (**A**) and platelecrit (**B**) between peak creatine kinase-MB (CK-MB)

Table 2. Univariate and multivariate logistic regression analyses of variables on ST elevation myocardial infarction in young patients

	Unadjusted OR	95% CI	P	Adjusted OR	95% CI	P
Age [years]	2.105	1.766–2.510	< 0.001	2.155	1.734–2.679	< 0.001
Gender (male)	1.340	0.817–2.196	0.247			
Diabetes mellitus	1.185	0.714–1.966	0.512			
Smoking	1.020	0.665–1.567	0.926			
Family history of CAD	1.540	0.968–2.450	0.006	1.206	1.061–1.715	0.013
Hypertension	1.969	0.637–6.088	0.239			
Hyperlipidaemia	0.549	0.333–1.905	0.119			
Glucose	1.000	0.966–1.005	0.880			
Urea	0.959	0.992–1.010	0.815			
Creatinine	0.767	0.111–5.304	0.778			
Haemoglobin	0.991	0.868–1.131	0.893			
White blood cell	1.485	1.094–2.013	0.003	1.225	1.074–1.948	0.019
Neutrophil	0.986	0.941–1.032	0.539			
Lymphocyte	0.894	0.769–1.039	0.144			
Total cholesterol	1.001	0.995–1.007	0.715			
LDL-C	1.003	0.997–1.009	0.333			
HDL-C	1.494	1.179–2.029	0.012	1.158	1.098–1.365	0.036
Triglyceride	0.999	0.996–1.001	0.264			
LVEF	0.999	0.974–1.024	0.933			
Culprit lesion	1.098	0.365–3.298	0.868			
Peak CK-MB	1.000	0.999–1.001	0.831			
Platelet count	0.988	0.994–1.001	0.177			
MPV	1.786	1.363–2.965	0.001	1.281	1.153–2.364	0.006
PDW	1.411	1.073–1.752	< 0.001	1.135	1.055–1.221	0.001
PCT	1.329	1.114–1.925	< 0.001	1.189	1.017–1.679	< 0.001

Adjusted for age, family history of CAD, HDL-C, MPV, PDW, PCT; CAD — coronary artery disease; CI — confidence interval; HDL-C — high-density lipoprotein cholesterol; LDL-C — low-density lipoprotein cholesterol; MPV — mean platelet volume; PCT — plateletcrit; PDW — platelet distribution width

In multivariate analysis, adjusted for age, family history of CAD, HDL levels, WBC levels, 1 fL increase in PDW levels was 13.5% more likely to be associated with young STEMI (odds ratio [OR] 1.135, 95% CI 1.055–1.221, p = 0.001), and similarly, 1% increase in PCT levels was 18.9% more likely to be associated with young STEMI (OR 1.189, 95% CI 1.017–1.679, p < 0.001) (Table 2).

DISCUSSION

We assessed the usefulness of PDW and PCT in young patients with STEMI. We found that levels of these indices were higher in young patients with STEMI than in both of the other groups. To the best of our knowledge, this is the first study to evaluate PDW and PCT levels in young patients with STEMI

We postulated that, in this patient group, aggravated prothrombotic status and increased inflammatory activity may suggest the underlying mechanism in the elevations of PDW and PCT. Thrombosis and inflammation play key roles in the initiation and progression of MI. Larger, reactive platelets are more likely to contribute to the thrombotic process than the smaller ones [14]. Increased platelet mass may reflect augmented platelet activity that is accompanied by aggravated release of inflammatory mediators, and as a result of this augmented activity, increased platelet mass may result in destructive inflammatory response and prothrombotic status.

Platelet distribution width, which is variability in platelet size, is associated with increased platelet production and activity [15]. Vatankulu et al. [15] found a relationship between PDW and chronic total occlusion in stable CAD. Khandekar et al. [16] showed that MPV and PDW were higher in patients with acute MI. Ege et al. [17] revealed that in saphenous venous graft disease, PDW levels were higher. Conversely, De Luca et al. [18] showed that PDW was not associated with the extent of CAD and subclinical carotid atherosclerosis in a large prospective study. This inconsistent result may be partially explained by the fact that in our study we included a more homogeneous patient population than in the study by De Luca et al. [18]; our study consisted not only of young patients but also the STEMI end of the ACS spectrum. In light of these considerations, young people might have more active platelets that lead to more unstable coronary conditions like STEMI.

On the other hand, PCT is a parameter that gives data about the platelet mass, which can be obtained by the equation of PLT × MPV/10^7. Recently, Ugur et al. [19] reported the prognostic importance of PCT values to predict long-term cardiovascular mortality in patients with STEMI. Akpinar et al. [20] showed that increased PCT was significantly associated with saphenous venous graft disease, and PCT might be a useful predictor of coronary slow flow phenomenon [21]. Our findings are consistent with the results of these previous studies.

Platelet distribution width and plateletcrit parameters had a significant negative correlation with age in our study. In conjunction with that, in a former study, platelet activity was in a negative relationship with advancing age [22]. In young patients with STEMI, the increased level of PDW and PCT can be associated with high platelet activity. Also, PDW and PCT parameters had a significant positive correlation with peak CK-MB.

Limitations of the study

Our study had some limitations. Firstly, our study was a single-centre cross-sectional study. A follow-up study regarding the prognostic value of PDW and PCT may add value to the study. We had no data on inflammatory markers such as C-reactive protein, interleukin-6, or thromboxane A2. Assessment of these substances may strengthen the study. Determination of the temporal changes in PDW and PCT levels during follow-up would provide new aspects to the study.

CONCLUSIONS

Platelet distribution width and plateletcrit levels seem to be novel markers that can reflect STEMI in young patients and may reflect prothrombotic state in this specific population. Future prospective studies are needed to find the exact mechanisms of PDW and PCT levels on pathophysiology and prognosis.

Conflict of interest: *none declared*

References

1. Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction—summary article: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee on the Management of Patients With Unstable Angina). J Am Coll Cardiol. 2002; 40: 1366–1374.
2. Fitzgerald DJ, Roy L, Catella F, et al. Platelet activation in unstable coronary disease. N Engl J Med. 1986; 315(16): 983–989, doi:10.1056/NEJM198610163151602, indexed in Pubmed: 3531859.
3. Huczek Z, Filipiak KJ, Kochman J, et al. Prognostic significance of platelet function in the early phase of ST-elevation myocardial infarction treated with primary angioplasty. Med Sci Monit. 2008; 14: CR144–CR151, indexed in Pubmed: 18301358.
4. Huczek Z, Filipiak KJ, Kochman J, et al. Medium on-treatment platelet reactivity to ADP is favorable in patients with acute coronary syndromes undergoing coronary stenting. Platelets. 2011; 22(7): 521–529, doi: 10.3109/09537104.2011.568075, indexed in Pubmed: 21443410.
5. Martin JF, Kristensen SD, Mathur A, et al. The causal role of megakaryocyte–platelet hyperactivity in acute coronary syndromes. Nat Rev Cardiol. 2012; 9(11): 658–670, doi: 10.1038/nrcardio.2012.131, indexed in Pubmed: 22987055.
6. Lippi G, Filippozzi L, Salvagno GL, et al. Increased mean platelet volume in patients with acute coronary syndromes. Arch Pathol Lab Med. 2009; 133(9): 1441–1443, doi: 10.1043/1543-2165-133.9.1441, indexed in Pubmed: 19722752.
7. Herve P, Humbert M, Sitbon O, et al. Pathobiology of pulmonary hypertension. The role of platelets and thrombosis. Clin Chest Med. 2001; 22(3): 451–458, indexed in Pubmed: 11590840.
8. Bain BJ, Bates I. Basic haematological techniques. In: Lewis SM, Bain BJ, Bates I (eds.). Dacie and Lewis practical haematology. 9th ed. Churchill Livingstone, Edinburgh 2001: 19–46.

9. Thaulow E, Erikssen J, Sandvik L, et al. Blood platelet count and function are related to total and cardiovascular death in apparently healthy men. Circulation. 1991; 84(2): 613–617, indexed in Pubmed: 1860204.
10. Office of National Statistics. Weekly incidence of heart attacks. http://www.statistics.gov.uk/morbidity/cardiovascular diseases.
11. Ozkan B, Uysal OK, Duran M, et al. Relationship between mean platelet volume and atherosclerosis in young patients with ST elevation myocardial infarction. Angiology. 2013; 64(5): 371–374, doi: 10.1177/0003319712448834, indexed in Pubmed: 22669950.
12. Thygesen K, Alpert J, White H. Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Universal definition of myocardial infarction. J Am Coll Cardiol. 2007; 50(22): 2173–2195, doi: 10.1016/j.jacc.2007.09.011.
13. Thygesen K, Alpert J, Jaffe A, et al. Third universal definition of myocardial infarction. J Am Coll Cardiol. 2012; 60(16): 1581–1598, doi:10.1016/j.jacc.2012.08.001.
14. Karpatkin S. Heterogeneity of human platelets. VI. Correlation of platelet function with platelet volume. Blood. 1978; 51(2): 307–316, indexed in Pubmed: 620086.
15. Vatankulu MA, Sonmez O, Ertas G, et al. A new parameter predicting chronic total occlusion of coronary arteries: platelet distribution width. Angiology. 2014; 65(1): 60––64, doi: 10.1177/0003319713486339, indexed in Pubmed: 23636855.
16. Khandekar MM, Khurana AS, Deshmukh SD, et al. Platelet volume indices in patients with coronary artery disease and acute myocardial infarction: an Indian scenario. J Clin Pathol. 2006; 59(2): 146–149, doi: 10.1136/jcp.2004.025387, indexed in Pubmed: 16443728.
17. Ege MR, Guray U, Guray Y, et al. Platelet distribution width and saphenous vein disease in patients after CABG. Association with graft occlusion. Herz. 2013; 38(2): 197–201, doi: 10.1007/s00059-012-3668-z, indexed in Pubmed: 22955688.
18. De Luca G, Santagostino M, Secco GG, et al. Mean platelet volume and the extent of coronary artery disease: results from a large prospective study. Atherosclerosis. 2009; 206(1): 292–297, doi: 10.1016/j.atherosclerosis.2009.02.008, indexed in Pubmed: 19426979.
19. Uğur M, Ayhan E, Bozbay M, et al. The independent associatıon of plateletcrıt wıth long-term outcomes in patıents undergoıng prımary percutaneous coronary interventıon. J Crit Care. 2014; 29(6): 978–981, doi: 10.1016/j.jcrc.2014.07.001, indexed in Pubmed: 25124920.
20. Akpinar I, Sayin MR, Gursoy YC, et al. Plateletcrit. A platelet marker associated with saphenous vein graft disease. Herz. 2014; 39(1): 142–148, doi:10.1007/s00059-013-3798-y, indexed in Pubmed: 23575980.
21. Akpinar I, Sayin MR, Gursoy YC, et al. Plateletcrit and red cell distribution width are independent predictors of the slow coronary flow phenomenon. J Cardiol. 2014; 63: 112–118, doi: 10.1016/j.jjcc.2013.07.010.
22. Gilstad JR, Gurbel PA, Andersen RE. Relationship between age and platelet activation in patients with stable and unstable angina. Arch Gerontol Geriatr. 2009; 48(2): 155–159, doi: 10.1016/j.archger.2007.12.006, indexed in Pubmed: 18282622.

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Szerokość rozkładu objętości płytek i płytkokryt: nowe biomarkery zawału serca z uniesieniem odcinka ST u młodych pacjentów

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Streszczenie

Wstęp: Płytki krwi odgrywają podstawową rolę w zawale serca, a ich aktywność można ocenić na podstawie wskaźników płytkowych, takich jak wskaźnik rozpiętości rozkładu objętości płytek (PDW) i płytkokryt (PCT). Wykazano, że wskaźniki te są markerami stanu prozakrzepowego w chorobach sercowo-naczyniowych.

Cel: Celem pracy było zbadanie użyteczności tych wskaźników w zawale serca z uniesieniem odcinka ST (STEMI) u młodych pacjentów.

Metody: W tym przekrojowym badaniu uczestniczyło 565 osób, które podzielono na trzy grupy: grupa 1 — 168 młodych pacjentów ze STEMI, grupa 2 — 173 starszych chorych ze STEMI i grupa 3 — 224 dopasowanych pod względem wieku osób z prawidłowym obrazem tętnic wieńcowych w koronarografii (grupa kontrolna). Za młodych pacjentów ze STEMI uznawano mężczyzn w wieku poniżej 45 lat i kobiety poniżej 55 lat.

Wyniki: W grupie 1 wartości PDW i PCT (odpowiednio 17,2 ± 0,67; 0,249 ± 0,05) były istotnie wyższe niż w innych grupach (odpowiednio grupa 2: 16,4 ± 0,56; 0,231 ± 0,04; grupa 3: 15,1 ± 0,63; 0,227 ± 0,04). Wskaźniki PDW i PCT wykazywały umiarkowanie silną ujemną korelację (odpowiednio r = –0,305 i r = –0,330) z wiekiem i umiarkowanie silną dodatnią korelację z maksymalnym stężeniem frakcji MB kinazy kreatynowej (odpowiednio r = 0,259; r = 0,320). W analizie wieloczynnikowej, po skorygowaniu względem innych zmiennych, wykazano, że zwiększenie o 1 fl wartości PDW wiązało się z większym o 13,5% prawdopodobieństwem zawału w młodym wieku. Podobnie przy 1-procentowym wzroście wartości PCT prawdopodobieństwo STEMI u młodych osób zwiększało się o 18,9%.

Wnioski: Wskaźniki PDW i PCT są niezależnymi markerami STEMI u młodych osób i mogą być wyznacznikiem stanu prozakrzepowego w tej szczególnej populacji pacjentów.

Słowa kluczowe: płytkokryt, wskaźnik rozpiętości rozkładu objętości płytek, zawał serca z uniesieniem odcinka ST, młodzi pacjenci

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