The relationship between platelet indices and clinical features of coronary artery disease

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Abstract

Background: Platelets play a key role in the development and progression of cardiovascular disease. The degree of platelet activation may be assessed by platelet indices such as platelet count, mean platelet volume (MPV) and platelet distribution width (PDW).

Aim: To evaluate the relationship between platelet indices and clinical features of coronary artery disease (CAD).

Methods: Our population is represented by a total of 441 consecutive patients undergoing coronary angiography. Patients were divided into three groups according to their clinical presentation: Patients with stable angina (Group I), with acute coronary syndrome (Group II), and with a normal coronary angiogram (Group III). All demographic and clinical features were collected retrospectively. Platelet indices were measured in all patients.

Results: There was no statistical difference for platelet count, MPV and PDW values among the groups. Correlation analysis showed a positive association between platelet count and Gensini scoring (Kendall's tau b, r = 0.312, p = 0.037, two-tailed) and also age (Kendall's tau b, r = 0.518, p = 0.001, two-tailed) in patients with CAD. However, there was no significant correlation between Gensini scoring and MPV or PDW values in these patients.

Conclusions: PDW and MPV may not be related to the clinical features or presentation and extent of CAD. Our study findings add to the conflicting results of previous studies in this area. Prospective trials with longer follow-up periods and larger samples are warranted to conclusively define the role of platelet indices in CAD.

Key words: platelet indices, mean platelet volume, platelet distribution

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INTRODUCTION

Platelets play a key role in the development and progression of cardiovascular disease, with increased aggregation and activation occurring in patients with chronic stable angina and acute coronary syndrome (ACS) [1]. Circulating platelets are heterogeneous with respect to their size, density and reactivity. It is generally accepted that large platelets are metabolically and enzymatically more active than small ones [2, 3]. It has been postulated that large platelets may be an indicator of platelet activation, and thus be related to the extent and also clinical presentation of coronary artery disease (CAD) [4]. The degree of platelet activation may be assessed by platelet indices such as platelet count, mean platelet volume (MPV) and platelet distribution width (PDW). It is also unclear whether these parameters can be considered risk factors for CAD.

MPV and PDW are easily measured, and they increase during platelet activation. Elevated MPV has been proposed as a risk factor for CAD or acute myocardial infarction. However, it remains uncertain whether the increased MPV is the result or the cause of CAD [5]. Indeed, contrasting data has been reported on the relationship between MPV and the risk of CAD [4–7]. Although PDW represents the variability in platelet size and may potentially provide more information than MPV

in terms of number of platelets reactivity, limited data exists on the relationship between PDW and the extent of CAD [4].

Thus, the aim of this study was to evaluate the relationship between platelet indices and clinical features of CAD in a consecutive cohort of patients undergoing coronary angiography.

METHODS

Our population consisted of 441 consecutive patients undergoing coronary angiography between February and July 2012. Patients were divided into three groups according to their clinical presentation: Group I: Stable angina was defined as chest pain on effort relieved by rest or sublingual nitrates, and most of these patients had a positive treadmill test and/or thallium scintigraphy. Group II: ACS was classified as non-ST-segment elevation myocardial infarction (NSTEMI) [8] and unstable angina (UA) according to the Braunwald classification [9] or ST-segment elevation myocardial infarction (STEMI). The diagnosis of STEMI was based on typical symptoms and new ST-segment elevation at the J point in ≥ 2 contiguous leads (≥ 0.2 mV in V1 through V3 and ≥ 0.1 mV in other leads) [10]. Group III: Control group, the patients with a normal coronary angiogram and chest pain were classified as having non-cardiac chest pain.

All demographic and clinical features were collected retrospectively. Patients were excluded if they had atrial fibrillation, haemodynamically significant obstructive valvular disease and oral warfarin therapy. Hypertension was defined as systolic pressure > 140 mm Hg and/or diastolic pressure > 90 mm Hg or if the individual was taking antihypertensive medications. The diagnosis of diabetes was based on a previous history of diabetes treated with or without drug therapies. Hyperlipidaemia was defined by elevated total plasma cholesterol levels (> 200 mg/dL).

The study was approved by the local ethics committee. The investigation conformed to the principles outlined in the Declaration of Helsinki.

Biochemical measurements

Blood samples were drawn at admission in patients undergoing elective or urgent coronary angiography. We measured platelet indices in a blood sample collected in tripotassium EDTA (7.2 mg) tubes. These blood samples were analysed within 1 h of venipuncture by an automatic blood counter (ADVIA 2120i Haematology System, Siemens Healthcare Diagnostics, Deerfield, Il, USA) used for whole blood analysis.

Coronary angiography

Femoral, brachial or radial artery cannulation was used for arterial access site and Judkins technique using 5- or 6-French catheters was applied for cannulation of the left and right coronary arteries. All angiograms were evaluated by two experienced physicians blinded to the study. Angiograms with stenotic lesions in all major epicardial coronary arteries including left main, left anterior descending (LAD), left circumflex (LCx), and right coronary (RCA) arteries were assessed, and the severity of CAD was assessed by using the Gensini scoring system [11] which grades narrowing of the lumens of the coronary arteries as: 1 for 1–25% narrowing, 2 for 26–50% narrowing, 4 for 51–75% narrowing, 8 for 76–90% narrowing, 16 for 91–99% narrowing, and 32 for total occlusion. This score was then multiplied by a factor that takes into account the importance of the lesion's position in the coronary arterial tree: the left main coronary artery × 5; the proximal segment of LAD × 2.5; the proximal segment of the LCx × 2.5; the mid-segment of the LAD × 1.5; the RCA, the distal segment of the LAD, the posterolateral artery and the obtuse marginal artery × 1; and others × 0.5. Scoring was performed by two observers and averaged.

Statistical analysis

Statistical package for social sciences (SPSS, version 15) software was used to analyse the data. Continuous data was expressed as mean ± standard deviation and categorical data as percentage. Analysis of variance and the χ^2 test were used for continuous and categorical variables, respectively. Kendall's in tau-b correlation analysis was performed to evaluate the relationship between platelet indices, groups and Gensini scoring. A p value < 0.05 was considered statistically significant.

RESULTS

Four hundred and forty-one patients were enrolled in the study; 394 of them had CAD and 47 of them had a normal coronary angiogram (Group III). Among the patients, 312 of them had stable angina pectoris (Group I) and 82 had ACS (Group II). The prevalence of CAD was significantly higher in the male gender, as we had expected. The baseline characteristics of the study population are shown in Table 1.

There was no statistical difference for platelet count, MPV and PDW values among the groups (Table 2). Forty-three of the patients with ACS had STEMI and 39 patients had UA/NSTEMI; there was also no statistical difference for platelet count, MPV and PDW values between these subgroups: 242.64 ± 83.4 vs. 247.23 ± 61.9 (p > 0.05); 8.5 ± 0.6 vs. 8.34 ± 0.8 (p > 0.05); 53.88 ± 8.1 vs. 52.22 ± 8 (p > 0.05), respectively. Furthermore, there was no significant correlation between MPV value and troponin T levels (r = 0.83; p > 0.05) in Group II. The extent of CAD and the number of totally occluded vessels in Groups I and II are summarised in Table 3. Correlation analysis showed a positive association between platelet count and Gensini scoring (Kendall's tau b, r = 0.312, p = 0.037, two-tailed) in patients with CAD. There was also a positive association between platelet count and age (Kendall's tau b, r = 0.518, p = 0.001, two-tailed) in these patients (Fig. 1). However, there was no significant correlation between Gensini scoring and MPV or PDW values in patients with CAD. Diabetes mel-

Table 1. Baseline characteristics of the study population

	Group I (n = 312)	Group II (n = 82)	Group III (n = 47)	P
Age [years]	60.7 ± 11.0	59.9 ± 10.8	58.5 ± 11.9	NS*
Male gender	68.3%	76.8%	27.7%	< 0.05**
Hypertension	44.4%	30.5%	36.2%	NS**
Hyperlipidaemia	13.5%	15.9%	17.0%	NS**
Diabetes mellitus	27.7%	22.0%	21.3%	NS**
Previous MI	4.8%	7.3%	0.0%	NS**
Heredity for CAD	22.1%	17.1%	27.7%	NS**
History of PCI	8.7%	7.3%	0.0%	NS**
History of CABG	6.4%	2.1%	0.0%	NS**
History of CVD	2.8%	0.0%	0.0%	NS**

*ANOVA; **Fisher exact test; Group I — patients with stable angina pectoris; Group II — patients with acute coronary syndrome; Group III — control group, patients who had a normal coronary angiogram; MI — myocardial infarction; CAD — coronary artery disease; PCI — percutaneous coronary intervention; CABG — coronary artery bypass graft; CVD — cerebrovascular disease

Table 2. Platelet indices in the study groups

Platelet indices	Group I (n = 312)	Group II (n = 82)	Group III (n = 47)	P
Platelet count [× 10⁹/L]	237 ± 75	245 ± 73	233 ± 65	NS*
Platelet distribution width [fL]	54.6 ± 8.6	52.3 ± 7.1	52.3 ± 7.1	NS*
Mean platelet volume [fL]	8.7 ± 3.9	8.4 ± 0.7	8.9 ± 2.2	NS*

*ANOVA analysis; Group I — patients with stable angina pectoris; Group II — patients with acute coronary syndrome; Group III — control group, patients who had a normal coronary angiogram

Table 3. Extent of coronary artery disease (CAD) and number of totally occluded vessels in the study population

	Group I (n = 312)	Group II (n = 82)	P
Extent of CAD			
Single vessel CAD	71 (23%)	23 (28%)	NS*
Two vessel CAD	98 (31%)	24 (29%)	NS*
Three vessel CAD	143 (46%)	35 (43%)	NS*
LM stenosis (≥ 50%)	18 (6%)	3 (4%)	NS*
Total occlusions			
Single vessel	63 (20%)	49 (60%)	< 0.05*
Two vessel	34 (11%)	7 (9%)	NS*

*Fisher exact test; Group I — patients with stable angina pectoris; Group II — patients with acute coronary syndrome; LM — left main

litus was present in 115 (26%) patients in the study population. There were no differences in terms of platelet count, MPV and PDW values between diabetic and non-diabetic subgroups: 241.76 ± 66 vs. 237.28 ± 75.3 (p > 0.05); 8.55 ± 1.1 vs. 8.77 ± 3.9 (p > 0.05); 53.84 ± 7.9 vs. 54.01 ± 8.4 (p > 0.05), respectively. As expected, the Gensini score was higher in the diabetic subgroup than in the non-diabetics: 40.12 ± 4 vs. 26.1 ± 33.2 (p = 0.023). To determine a possible relationship between platelet indices and atherosclerotic burden, we analysed another more atherogenic subgroup identified as patients who had peripheral artery disease and/or cerebrovascular disease. Of 441 patients, 36 (8%) had peripheral artery disease (27 patients) and/or cerebrovascular disease (nine patients). Platelet count, MPV and PDW values were similar between this more atherogenic subgroup and other patients: 281.15 ± 103.6 vs. 236.61 ± 70.8 (p > 0.05); 8.45 ± 1 vs. 8.72 ± 3.5 (p > 0.05); 54.12 ± 5.6 vs. 54 ± 8.5 (p > 0.05), respectively. Nevertheless, Gensini score was higher in this subgroup: 43.7 ± 42.2 vs. 28.98 ± 35.15 (p = 0.017).

DISCUSSION

Platelets have an increasingly well-defined critical role in coronary artery thrombosis and in other common cardiovascular diseases, including stroke, peripheral vascular disease, and diabetes mellitus. Although the role of platelets in thrombosis is well characterised, platelets may also have a role in the pathogenesis of the underlying atherosclerotic process [12]. A large variability in baseline platelet reactivity has been observed that may potentially be due to variability in platelet size. Larger platelets have a greater mass and are both metabolically and enzymatically more active than smaller platelets [5]. Haemostatically reactive and larger platelets have more granules and adhesion receptors that have resulted in a decreased bleeding time showing increased activation [13]. They have a greater prothrombotic potential, with higher levels
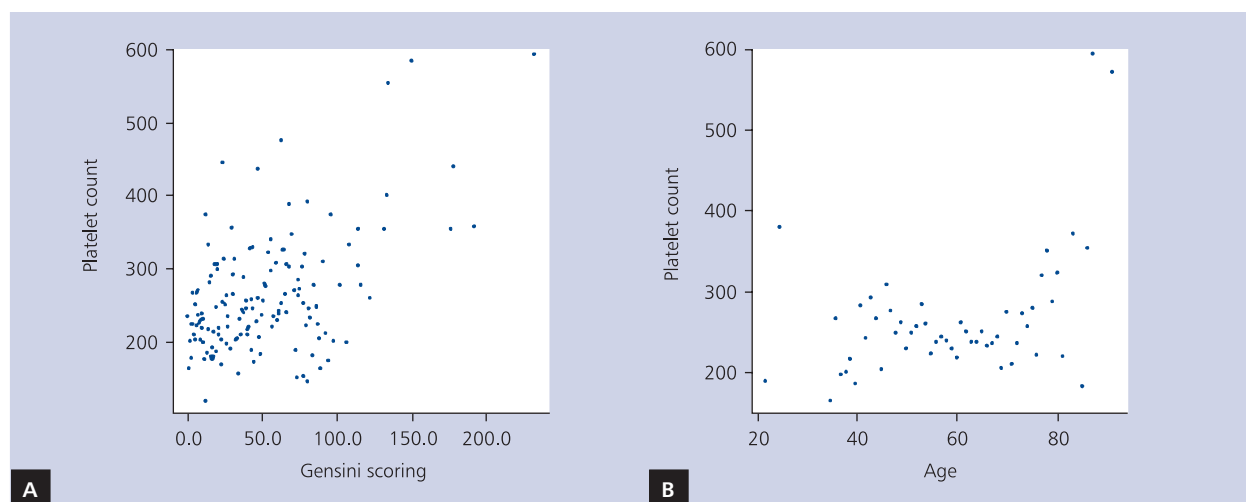
Figure 1. **A**. Correlation analysis between platelet count and Gensini scoring; **B**. Correlation analysis between platelet count and age

of intracellular thromboxane A_2 and beta thromboglobulin levels, as well as increased levels of procoagulant surface proteins [14].

In order to obtain a larger surface, platelets change in shape during activation from discoid to spherical. Platelet indices such as platelet count, MPV and PDW are easily measured, and they increase during platelet activation. PDW directly measures the variability in platelet size, and its high values could suggest larger production of larger reticulated platelets. Haematology analysers based on impedance technology measure platelet volume by the deformation of electrical field, which depends on the platelet vertical diameter. Analysers with laser optical technology determine the platelet volume according to the cross diameter of the platelet. Therefore, activated platelets seem larger independently of the principle of measurement [15].

Elevated platelet indices have been proposed as a risk factor for CAD or ACS. A stepwise decrease in MPV in subjects with chest pain in acute myocardial infarction, UA and non-cardiac chest pain has been observed in different ethnic groups [16, 17]. Lippi et al. [18] reported that Italian patients with ACS had significantly higher MPV values than patients without ACS. Furthermore, Huczek et al. [19] demonstrated that MPV is a strong, independent predictor of impaired angiographic reperfusion and six-month mortality in patients treated with primary percutaneous coronary intervention.

However, it remains uncertain whether increased platelet indices are the result or the cause of CAD. PDW represents the variability in platelet size and may potentially provide more information than MPV in terms of number of platelets reactivity [4]. Platelet production is governed by various agents. Thrombopoietin has been shown to be a major agent controlling platelet number, and changes in platelet production from megakaryocytes is probably modulated by a series

of cytokines [20–23] that are also involved in the inflammatory response. It has been shown that the presence of activated megakaryocytes in the bone marrow in atherosclerosis correlates with increased circulating levels of the inflammatory cytokine interleukin-6 found in atherosclerosis [24]. We therefore measured platelet count and size in patients with stable angina and ACS. Since atherosclerosis itself may influence the bone marrow megakaryocytes before platelet production [25, 26], we also assessed the extent of CAD in all patients by coronary angiography.

In the present study, we demonstrated that there is no statistical difference for platelet count, PDW and MPV values between patients with a normal coronary artery angiogram and those with coronary atherosclerotic disease. Therefore, we did not find any relationship between PDW and MPV with clinical presentation or extent of disease in 394 coronary atherosclerotic patients undergoing coronary angiography. In addition, there was also no statistical difference for platelet indices between subgroups of ACS. However, correlation analysis showed positive associations between platelet count with Gensini scoring and advanced age in patients with CAD.

In accordance with our data, several studies have reported that there is no relationship between MPV or PDW and the extent of CAD [4, 6, 27, 28]. MPV has been shown to correlate with both megakaryocyte ploidy and with the percentage of circulating reticulated platelets [29]. Furthermore, a positive correlation between thrombopoietin levels and MPV values has been demonstrated in CAD [30]. Thus, larger platelet volume may not imply higher platelet reactivity shown to be related to the extent and complexity of CAD [31]. Furthermore, it may even be associated with reduced aggregation, since larger platelets may be precursors of mature platelets. Platelet volume has been shown to be associated with other prognostic factors such as smoking [32], diabe-

tes [33], obesity [34] and hypertension [35]. However, these risk factors may primarily affect the extent of CAD and clinical outcome other than platelet volume.

Supporting our data, Würtz et al. [36] reported that platelet aggregation is significantly associated with platelet count even within the normal range in patients with CAD. Aliberti et al. [37] showed that there is a significant correlation between fibrinogen and platelet count in the patients with coronary heart disease. Fibrinogen and platelets are two of the major contributors to the pathogenesis and evolution of cardiovascular diseases. Higher platelet count may be a clue to the activated inflammatory process of atherosclerosis. However, the relationships between platelet count and the evolution of atherosclerosis/subclinical atherosclerosis needs to be clarified.

Limitations of the study

The limitations of this study are its retrospective nature and insufficient clinical data. Serum fibrinogen level was not measured in our patients. Thus, it was impossible to evaluate the relationship between fibrinogen levels and platelet count. Additionally, a higher percentage of patients were already taking aspirin on a daily basis in Group I than in Group II (98% vs. 72%; p < 0.05, respectively). However the percentages of patients who had been taking dual antiplatelet therapy were similar between the groups (44% vs. 54%, p > 0.05, respectively).

CONCLUSIONS

This study showed that PDW and MPV are not related to the clinical features or presentation or extent of CAD. Thus, these parameters cannot be considered as risk factors for CAD. However, there are positive associations between platelet count with Gensini scoring and advanced age in patients with CAD.

Conflict of interest: none declared

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Zależności między wskaźnikami płytkowymi a obrazem klinicznym choroby wieńcowej

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Streszczenie

Wstęp: Płytki krwi odgrywają ważną rolę w rozwoju i progresji chorób układu sercowo-naczyniowego. Stopień aktywacji płytek można ocenić na podstawie wskaźników płytkowych, takich jak liczba płytek krwi, średnia objętość płytek (MPV) i szerokość rozkładu płytek (PDW).

Cel: Celem badania była ocena zależności między wskaźnikami płytkowymi a obrazem klinicznym choroby wieńcowej (CAD).

Metody: Badaniem objęto 441 kolejnych pacjentów poddanych angiografii wieńcowej. Chorych podzielono na trzy grupy w zależności od charakterystyki klinicznej: osoby ze stabilną dławicą piersiową (grupa I), osoby z ostrym zespołem wieńcowym (grupa II) i osoby z prawidłowym obrazem w angiografii wieńcowej (grupa III). Wszystkie dane demograficzne i kliniczne zbierano w sposób retrospektywny. U wszystkich chorych zmierzono wskaźniki płytkowe.

Wyniki: Nie stwierdzono statystycznych różnic między grupami pod względem liczby płytek krwi i wartości MPV oraz PDW. Analiza korelacji wykazała dodatnie zależności między liczbą płytek krwi a punktacją w skali Gensiniego (współczynnik tau-b Kendalla r = 0,312; p = 0,037; test dwustronny) oraz wiekiem (współczynnik tau-b Kendalla, r = 0,518; p = 0,001; test dwustronny) u pacjentów z CAD. Nie wykazano jednak u tych chorych istotnych korelacji między punktacją w skali Gensiniego a wartościami MPV lub PDW.

Wnioski: Wartości PDW i MPV mogą się nie wiązać z obrazem klinicznym ani charakterem zmian miażdżycowych lub rozległością CAD. Wyniki te są rozbieżne z danymi uzyskanymi we wcześniejszych badaniach na ten temat. Należy przeprowadzić prospektywne badania z dłuższym okresem obserwacji i większą liczebnością próby, aby jednoznacznie ustalić znaczenie wskaźników płytkowych w CAD.

Słowa kluczowe: wskaźniki płytkowe, średnia objętość płytek, rozkład płytek krwi

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