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Mean platelet volume on admission is associated with further left ventricular functions in primary PTCA patients

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Abstract. – AIM: In ST elevation myocardial infarction (STEMI) patients, mean platelet volume (MPV) is associated with infarct related artery patency both before and after reperfusion. In anterior STEMI patients successfully treated with primary percutaneous coronary intervention (PCI), the relationship between left ventricular (LV) function and MPV on admission is unknown.

METHODS: 97 anterior STEMI patients successfully revascularizated with PCI between January 2010 and February 2011 are included. MPV on admission is recorded. All patients underwent transthoracic echocardiography within 3 days or before discharge. Patients were divided into two groups according to left ventricular ejection fraction (LVEF), as systolic dysfunction (LVEF < 50%, 1st group) and normal systolic functions (LVEF > 50%, 2nd group). The 1st group included 61 (47 males) patients and the 2nd group included 36 (35 males) patients.

RESULTS: MPV was; 9.5±1.1 femtoliter (fL) in the 1st and 8.8±0.8 fL in the second group. The difference between the groups was significant (p = 0.001). There was a significant difference in the Troponin I levels and white blood cell (WBC) counts on admission between two groups (30±29 vs 12.2±15.1 ng/mL, p = 0.001 and 12.3±3.8 vs 10.6±3.4 counts ×10^9/L, p = 0.027, respectively).

CONCLUSIONS: In anterior STEMI patients treated with percutaneous coronary intervention, increased MPV on admission is associated with impairment in left ventricular systolic function.

Key Words:
Ejection fraction, Myocardial infarction, Mean platelet volume, Primary percutaneous coronary intervention.

Introduction

Mean platelet volume (MPV) is an indicator of platelet activation and platelet functions[1]. Platelet aggregation plays an important role in the pathogenesis of myocardial infarction. MPV, an indicator of platelet activation, is found to be higher in patients with coronary artery disease than in normal subjects[2]. A high MPV is an independent risk factor for myocardial infarction[3]. In acute myocardial infarction patients treated with percutaneous coronary intervention, MPV is shown to be an independent predictor of poor angiographic reperfusion and six-month mortality[4]. In STEMI patients treated with fibrinolytic agents TIMI frame count was found to be greater in patients with higher MPV[5].

The relationship between MPV obtained from complete blood count during first admission to hospital and left ventricular systolic functions is unknown. In the present report we aimed to analyze the relationship between MPV on admission and left ventricular functions in anterior STEMI patients underwent primary PCI.

Materials and Methods

97 patients (82 male, 84.5%) admitted with anterior ST elevation myocardial infarction (MI) and revascularizated with primary percutaneous transluminal coronary angioplasty (PTCA) within 6 hours of symptom onset were included in this study. The diagnosis of acute anterior MI was considered when a patient had typical chest pain with more than 2 mm ST elevation in at least two consecutive anterior derivations.

Patients who refused angiography, and patient with cardiogenic shock before the procedure, resuscitated arrest and arrest under mechanical ventilation, inappropriate echocardiographic window that preclude evaluation of LV functions and who died before the third day were excluded. Ethics approval is obtained from Hospital Ethics Committee. All patients were informed about the study and written consent was taken.

For biochemical analyses 3 ml venous blood sample was taken into tubes containing dipotassium ethylene tetraacetic acid and examined within an hour. Beckman Coulter LH 750 Analyser (Miami, FL, USA) device was used for the process. Plasma LDL, HDL, glucose, troponin levels were also examined using the same sample.

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Patients who consented the procedure underwent standard angiography with a Siemens angiography device (Axiom Artis Zee, Siemens, Erlangen, Germany)**.** Primary percutaneous coronary intervention (PCI) was performed to infarct related artery. 100 Unit/kg unfractionated heparin, 300 mg clopidogrel loading dose and 300 mg aspirin was administered to all patients before the procedure. Use of glycoprotein IIb/IIIa inhibitors and thrombus aspiration catheter was left to operator's preference. Thrombolysis in Myocardial Infarction (TIMI) flows were estimated by two experienced cardiologist according to Gibson et al method[6]. Patients without TIMI III flow were excluded after the procedure.

2-Dimensional and M mode echocardiography (GE, Vingmed Vivid3 Expert, Horten, Norway) was performed to all patients between 3 and 5 days in the left lateral decubitis position as suggested by the American Society of Echocardiography[7]. LV functions were measured from the apical view using modified Simpson method. Left ventricular end systolic diameter (LVESD), left ventricular end diastolic diameter (LVEDD), left atrium diameter and ejection fraction (EF) were recorded. Patients were grouped according to their ejection fraction. The 1st group included patients with an EF < 50%, and the second group included patients with an EF ≥ 50%.

Statistical Analysis

All values were expressed as mean ± SD. Fitness to normal distribution was evaluated using Kolmogorov-Simirnov test. Analysis of variance (one way ANOVA) was made for continuous variables. Post Hoc analysis was made using Tukey and Tamhane tests. Ki-square (χ^2) was used for categorical variables. All statistical analyses were made using SPSS, 13.0 (SPSS Inc, Chicago, IL, USA). A p value less than 0.05 was considered statistically significant.

Results

Initial group characteristics are shown in Table I. Echocardiographic results are shown in Table II. 82 (84.5%) of the patients were male. The first group (EF < 50%) consisted of 61 patients (47 males, 77%), the second group (EF ≥ 50%) consisted of 36 patients (35 males, 97.2%). On comparison of the two groups, the first group was older (64.3±11.6 and 59.2±11.6 years, p=0.038). There were no differences in hemoglobin, platelet and blood glucose levels. There was no statistically significant difference in diabetes, hypertension and smoking status on admission between the groups. There was a significant difference in mean platelet volume (MPV) on admission between the groups (9.5 ± 1 vs 8.8 ± 0.7 fL p = 0.001). There was also a significant difference in troponin I levels (30±29 vs 12.2±15.1 ng/mL, p = 0.001) and WBC counts between the groups (12.3±3.8 vs10.6 ± 3.4 counts×10^9/L, p = 0.027).

Discussion

In our study, we observed a negative relation between admission MPV and subsequent EF. Compatible with previous studies, there was a significant relation between high white blood cells (WBC) count on admission and left ventricular EF.

Platelets play an important role in the pathogenesis of acute coronary syndrome. While platelets are spent during the MI, bone marrow produce and releases platelets to circulation. Mediators released from these rapidly produced immature platelets are more active. This has a negative effect on thrombotic process[8]. High MPV is a biochemical indicator of platelet activation and negative impacts on thrombotic process.

Left ventricular systolic functions after MI is the most important factor in morbidity and mortality[9]. There are several factors affecting left venricular remodeling after MI, like irreversible microvascular injury triggered by ischemic reperfusion, full-thickness (transmural) necrosis, wide infarct area, re-obstruction or re-stenosis of the

Table I.

	EF < 50 (n = 61)	EF ≥ 50 (n = 36)	p
Age (years)	64.3 ± 11.4	59.2 ± 11.6	0.038
Gender (Male %)	47 (77%)	35 (97.2)	0.008
DM	14 (23%)	6 (16.7%)	0.621
Smoking	31 (50.8%)	17 (47.2%)	0.834
HT	25 (41%)	14 (39%)	0.846
Glycemia (mg/dl)	154.3 ± 70.3	145.1 ± 76.4	0.552
MPV (fL)	9.5 ± 1	8.8 ± 0.7	< 0.001
Plt (10^9)	204 ± 48.3	221.7 ± 53.8	0.065
WBC (×10^9)	12.3 ± 3.8	10.6 ± 3.4	0.027
Hb (mg/dl)	14.1 ± 1.6	14.5 ± 1.4	0.193
TroponinT (ng/ml)	30 ± 29	12.2 ± 15.1	0.001
LDL	142.5 ± 35	131.1 ± 2 7.7	0.128

DM: diabetes mellitus, HT: hypertension, MPV: mean platelet volume, PLT: platelets, WBC: white blood cells, Hb: hemoglobin, LDL: low density lipoprotein.

Table II.

	EF < 50 (n = 61)	EF ≥ 50 (n = 36)	P
EF (%)	35.5 ± 6.3	58 ± 4.6	< 0.001
LVEDD (mm)	49.7 ± 5.4	47.1 ± 4.1	0.008
LVESD (mm)	34.7 ± 5.8	31.4 ± 4.4	0.005
Left atrium (mm)	39.2 ± 4.8	35 ± 3.1	< 0.001

EF: ejection fraction; LVEDD: left ventricular end-diastolic diameter; LVESD: left venricular end-systolic diameter.

infart related artery, left ventricular dyssynchrony, mitral regurgitation, inflammation and fibrosis, oxidative stress, neurohormonal activation (angiotensin II, aldosterone, BNP/ANP)[10].

MPV is a parameter showing platelet activation and function[1]. Elevated mean platelet volume in patients with MI is an important risk factor for recurrent infarction and death[10]. MPV is a strong, independent predictor for angiographic reperfusion and six month mortality in patients underwent primary PTCA[11].

According to previous studies, high MPV on admission have a negative effect on patency[12] and TIMI frame count[13] of the infarct related artery in MI patients treated with thrombotic therapy. In another study of patients with MI who underwent primary PTCA, the MPV was higher in patients with total occlusion of infarct related artery than in patients with partial occlusion[14].

We found that patients with a LVEF < 50% had a higher admission MPV than other group. Therefore, we proposed that, the increase in MPV may have caused recurrent thrombosis and worsen microvascular perfusion, and thereby affected left ventricular functions.

In conclusion, these findings, although do not prove a direct relation, may suggest an indirect role of MPV in LV remodelling. For this reason, in acute anterior STEMI patients, an high MPV may play an alerting role for possible LV dysfunction. Consequently, patients admitting to the Emergency Department with an acute anterior MI and a high MPV, are more likely to develop a ventricular dysfunction and therefore the role of more aggressive antithrombotic theraphy in this patient group should be evaluated. Further studies are needed on this issue.

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