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Original article

The relation between mean platelet volume and coronary collateral vessels in patients with acute coronary syndromes

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ABSTRACT

Objective: Elevated mean platelet volume (MPV) has been proposed as a risk factor for coronary artery disease (CAD) and is associated with poor clinical outcome in acute coronary syndrome (ACS). We aimed to evaluate the association of MPV with presence of coronary collateral vessel (CCV) in patients with ACS.
Methods: A total of 417 patients with ACS were included in the study. All patients underwent coronary angiography on the first day after admission and patients with a greater than or equal to 80% obstruction in at least one epicardial coronary artery were included in the study. The CCVs are graded according to the Rentrop scoring system and a Rentrop grade 0 was accepted as no CCV development (Group 1), Rentrop Grade 1–2–3 were accepted as presence of CCV development (Group 2).
Results: The median of MPV was 9.1 ± 1.4 fl. Mean age was 60 ± 12 year. Group 1 consisted of 233 (55.9%) patients and Group 2 consisted of 184 (44.1%) patients. Presence of CCV was significantly associated with high levels of MPV ($p = 0.005$). Presence of CCV was also associated with presence of diabetes and systolic blood pressure.
Conclusion: High MPV on admission was associated with presence of CCV in patients with ACS.

Introduction

Platelets play an important role in the pathophysiology of atherosclerosis and prothrombotic events leading to ACS [1]. Mean platelet volume (MPV) is a useful biomarker of platelet activity [1,2]. Platelets are heterogeneous with respect to their size and reactivity. Large platelets are metabolically and enzymatically more active than small ones and have higher thrombotic potential [3]. Increase in MPV is associated with poor clinical outcome and impaired angiographic reperfusion in patients with myocardial infarction (MI) [4].

Coronary collateral vessels (CCVs) can provide a perfusion reserve in case of increased myocardial oxygen demand. Coronary collaterals can limit the myocardial ischemia and can protect the viable myocardium in patients with ACS [5–7]. Proliferation of CCV occurs in response to myocardial ischemia. The presence of CCV is correlated with well clinical outcomes in patients with ACS [8]. The complex mechanisms mediating the development of CCV in the heart are not well-understood. Some mediators (such as C-reactive protein, uric acid, circulating endothelial progenitor cells) were investigated to explain this relationship, but there is no conclusive evidence to explain this relationship [9,10]. Moreover, as increased MPV predicted the risk of coronary artery disease (CAD) and cardiac mortality, it may also be interesting to examine whether levels of MPV predict the presence of CCVs, one of the major predictors of mortality in patients with ACS. In the present study, we hypothesized that levels of serum MPV could be independently associated with the development of CCV after adjustment for traditional cardiovascular risk factors.

Methods

Patients

A total of 670 consecutive and unselected patients who were admitted to hospital with ACS were prospectively included in the study. Patients with a history of renal disease, a history of past coronary intervention or coronary artery bypass grafting, a history of inflammatory rheumatic disease, a history of chronic

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obstructive pulmonary disease, and taking aspirin/clopidogrel/warfarin/unfractionated heparin and low molecular weight heparin were excluded. The patients with less than 80% stenosis in the coronary angiography were also excluded from the study [11]. Finally, 417 patients were included in the study. According to coronary angiography results, patients were divided into two groups as Group 1 (no CCV) and Group 2 (presence of CCV). Informed consent was obtained from all patients. The study was approved by our local ethics committee. All demographic and clinical data were collected prospectively.

Laboratory analysis

In all cases, blood samples were drawn at admission before starting any medication (Thermo Clinical Lab system with Konelab 60 I kids, Helsinki, Finland). For assessment of MPV, blood samples were taken and put into tubes containing tripotassium ethylenediaminetetraacetic acid before clopidogrel, heparin, or tirofiban administration and studied within 20 min with an automated flow counter (Sysmex SE 9500, Roche, Mannheim, Germany). The expected values for MPV in our laboratory ranged from 7.0 to 10.5 fl.

Coronary angiography

Quantitative coronary angiography was performed by two experienced interventional cardiologists who had no knowledge of the patients' clinical information by the Judkins or Sones technique. Coronary vessel disease was described as 80% or greater degree of diameter stenosis in at least one coronary artery. Collateral circulation was graded according to the Rentrop classification. The collateral circulation was based on the injection that best opacified the occluded vessel: 0 = no visible filling of any collateral vessels; 1 = filling of side branches of the artery to be perfused by collateral vessels without visualization of the epicardial segment; 2 = partial filling of the epicardial segment by collateral vessels; and 3 = complete filling of the epicardial segment by collateral vessels [12]. Rentrop grade 0 accepted as no development of CCV and Rentrop grade $\geq$1 was accepted as presence of CCV (Group 2). Also, Rentrop grades 0–1 were accepted as bad CCV development, Rentrop grades 2–3 were accepted as well CCV development.

Statistical analysis

All analyses were performed using SPSS V 16.0 for windows (version 16.0, SPSS, Chicago, IL, USA). Quantitative variables were expressed as mean value ± SD for parametric variables and median and minimum–maximum levels for non-parametric variables. Comparison of parametric values between two groups was performed by means of independent-samples t test. Comparison of non-parametric values between two groups was performed by Mann–Whitney *U*-test. Categorical variables were compared by the chi-squared test. Pearson test used for correlation parametric variables and Spearman test was used for non-parametric variables. Two-tailed p-value < 0.05 was considered statistically significant. Multivariate logistic regression analysis was performed to evaluate the effects of gender, diabetes, systolic blood pressure, blood urea nitrogen (BUN), creatinine, hemoglobin, total protein, albumin, and MPV on development of good CCV. Receiver operating characteristics (ROC) analysis was performed. The best cutoff value was determined and the sensitivity and specificity at that point were determined.

Results

Table 1 shows baseline clinical characteristics of the patients. The mean platelet count was 229 ± 64 ($\times 10^3$) and the mean MPV was 9.1 ± 1.4 fl. Mean age was 60 ± 12 years. Rentrop 0, 1, 2, and 3 were determined in, respectively, 233 (55.9%), 84 (20.1%), 45 (10.8%), and 55 (13.2%) patients. Rentrop 1–2–3 (presence of CCV) were determined in 184 (44.1%) patients. Tables 2 and 3 show the relation between the presence of CCV and baseline clinical and laboratory characteristics of the patients. Presence of CCV was significantly associated with high levels of MPV ($p = 0.005$) (Table 3 and Fig. 1). Presence of CCV was also associated with presence of diabetes and low systolic blood pressure. Also the development of good CCV (Rentrop 2–3) was associated with high levels of MPV ($p = 0.016$) (Fig. 1).

Rentrop score was associated with MPV ($r = 0.181$ $p < 0.0001$), systolic blood pressure ($r = -0.178$, $p = 0.017$), and serum creatinine ($r = -0.126$, $p = 0.010$).

Table 1
Baseline clinical characteristics of patients.

Age (years)	60 ± 12
Female (*n*, %)	98 (23.5%)
Male (*n*, %)	319 (76.5%)
Smoking (*n*, %)	212 (50.8%)
Platelet count ($\times 10^3$/l)	229 ± 64
MPV (fl)	9.1 ± 1.4
Rentrop 0 (*n*, %)	233 (55.9%)
Rentrop 1 (*n*, %)	84 (20.1%)
Rentrop 2 (*n*, %)	45 (10.8%)
Rentrop 3 (*n*, %)	55 (13.2%)

MPV, mean platelet volume.

Table 2
Relation between presence of coronary collaterals and baseline characteristic of patients.

	Rentrop 0	Rentrop 1–2–3	*p*
Age (years)	59 ± 12	60 ± 12	0.171
Gender (male, %)	179 (76.8%)	140 (76.0%)	0.860
Smoking (*n*, %)	116 (49.7%)	96 (52.1%)	0.628
Hypertension (*n*, %)	40 (17.2%)	42 (22.8%)	0.149
Diabetes (*n*, %)	27 (11.6%)	37 (20.1%)	0.017
USAP (*n*, %)	73 (31.3%)	56 (30.5%)	0.844
NSTEMI (*n*, %)	81 (34.8%)	77 (41.8%)	0.139
STEMI (*n*, %)	79 (33.9%)	51 (27.7%)	0.140
Systolic BP (mmHg)	120 (90–190)	110 (80–210)	0.041
Diastolic BP (mmHg)	70 (50–110)	70 (50–130)	0.130

USAP, unstable angina pectoris; NSTEMI, non-ST elevation myocardial infarction; STEMI, ST elevation myocardial infarction; BP, blood pressure.

Table 3
Relation between presence of coronary collaterals and baseline laboratory findings of patients.

	Rentrop 0	Rentrop 1–2–3	*p*
MPV (fl)	8.9 ± 1.4	9.3 ± 1.4	0.005
Glucose (mg/dl)	106 (57–461)	108 (60–463)	0.392
WBC ($\times 10^9$/l)	10.1 ± 3.5	10.1 ± 3.2	0.926
BUN (mg/dl)	23 (5–47)	21 (9–46)	0.165
Creatinine (mg/dl)	0.9 (0.2–1.4)	0.9 (0.6–1.4)	0.169
Troponin (ng/ml)	0.4 (0–79)	0.7 (0–100)	0.299
CK-MB (IU/l)	25 (1–693)	33 (3–426)	0.306
Total cholesterol (mg/dl)	181.9 ± 40.7	185.1 ± 45.6	0.376
LDL cholesterol (mg/dl)	113.9 ± 35.7	119.7 ± 39.5	0.181
HDL cholesterol (mg/dl)	35 (16–89)	34 (20–124)	0.674
Triglyceride (mg/dl)	121 (39–675)	123 (22–849)	0.786
Hemoglobin (mg/dl)	14.4 (8.2–19.0)	14.6 (8.1–58.0)	0.366
Platelets ($\times 10^3$/l)	231.1 ± 61.2	226.7 ± 67.7	0.685
Total protein (g/dl)	6.6 ± 0.6	6.6 ± 0.6	0.353
Albumin (g/dl)	3.9 ± 0.4	3.8 ± 0.5	0.163

MPV, mean platelet volume; WBC, white blood cell; BUN, blood urea nitrogen; CK-MB, creatinine kinase MB fraction; LDL, low-density lipoprotein; HDL, high-density lipoprotein.