Serial changes of mean platelet volume in relation to Killip Class in patients with acute myocardial infarction and primary percutaneous coronary intervention

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ABSTRACT

Introduction: Mean platelet volume (MPV) is related to the reactivity of platelets, and among survivors of acute myocardial infarction (MI), greater MPV is known to be associated with impaired reperfusion and higher mortality. The aims of the study is to investigate the dynamic changes of MPV and the relation between MPV and cardiac function in patients with acute MI and received primary percutaneous coronary intervention (PCI).

Materials and Methods: This retrospective cohort study included patients presented during January 2008 to March 2011 to Peking University Third Hospital with ST-segment elevation MI. All patients received successful PCI. MPV was measured serially from admission to day 7 after MI.

Results: In 375 patients, MPV reached its peak value (10.16 ± 1.05 fL) at the admission, and then reduced by 16% within the 24 hours. Patients with poorer ventricular function, estimated by high Killip Class (≥2, n=96), had higher MPV values at all time points studied. By logistic regression model and after adjusting for related confounders, high MPV remained as an independent predictor of Killip Class ≥2 (OR 1.873, CI 95% 1.373 - 2.673, *p*=0.001). Clopidgorel pre-usage resulted in significant MPV reduction on admission.

Conclusions: MPV undergoes rapid and dynamic changes during the acute phase of MI, and was higher in patients with high Killip Class, suggesting a predictive value of MPV in ventricular dysfunction and clinical outcome of acute phase of MI.

Keywords: Mean platelet volume, Acute myocardial infarction, Killip Class, Primary percutaneous coronary intervention, Clopidgorel

Introduction

Platelets play an important role in plaque rupture and thrombus formation leading to acute coronary syndrome (ACS) and myocardial infarction (MI). It has been shown that platelet size, measured as mean platelet volume (MPV), relates to their reactivity [1]. MPV is readily available with routine blood counts and is therefore an attractive index to investigate in clinical settings. Larger platelets are metabolically and enzymatically more active than smaller ones [2], and have greater prothombotic potential [3]. MPV is positively correlated with indicators of platelet activity including aggregation, release of thromboxane A2 or β-thromboglobulin and expression of glycoproteins Ib or IIb/IIIa [1,2,4,5]. A recent systematic review and meta-analysis showed that MPV was higher in patients with acute MI (9.24 fL) than in those without acute MI (8.48 fL). Elevated MPV has been regarded as an independent risk factor for MI [6]. Among survivors of MI, greater MPV is also associated with impaired reperfusion [2,7,8] and higher morbidity and mortality [2,6,7,9,10]. In patients with metabolic syndrome and ST-segment elevation MI (STEMI), increased MPV at the admission may be associated with the degree of left ventricular (LV) systolic dysfunction [11].

A majority of these studies on patients with MI only measured MPV from admission blood samples prior to drug administration. There are few reports on dynamic changes of MPV in patients with MI receiving primary percutaneous coronary intervention (PCI). Furthermore, despite the well-recognized association between MPV and clinical or angiographic outcomes in patients with MI, the relation between MPV and LV systolic dysfunction after PCI remains unclear. The aims of this study were to define the dynamic changes of MPV following MI and to determine the relationship between platelet indices and cardiac function, estimated by the Killip Class.

Methods

Patients

We conducted a retrospective study in patients who presented with STEMI during January 2008 to March 2011 to the Department of Cardiology, Peking University Third Hospital. Permission for the study was obtained by a local ethics committee. STEMI was diagnosed according to the American College of Cardiology/American Heart Association guideline in 2004. Digital angiograms were analyzed by two experienced interventional cardiologists, blind of MPV results. In order to assess coronary blood flow as a continuous variable, the corrected TIMI frame count (CTFC) was determined on the final angiogram, as previously described by Gibson *et al* [12]. All patients received successful PCI (defined as coronary angiography with optimized flow of TIMI grade 3). Of consecutive patients with STEMI, those who had the following conditions were excluded: 1) major kidney or hepatic disease; 2) malignancy; 3) infectious disease; 4) usage of clopidogrel prior to the onset; and 5) other causes of acute MI.

Killip classification

Killip Class was evaluated by three clinicians during day-1 after acute MI according to the classic article [13]. Class 1: no evidence of heart failure; Class 2: signs indicating mild to moderate degree of heart failure (e.g. S3 gallop, rales < half-way up lung fields or elevated jugular venous

pressure); Class 3: pulmonary edema, and Class 4: cardiogenic shock or hypotension. Patients were re-grouped according to Killip Class.

Blood sampling and haemotological analyses

In all cases, peripheral blood was drawn at the admission prior to administration of anti-platelet drugs or PCI, and then in the morning of the first, third and seventh day after hospitalization. Blood samples were collected into standardized tubes (INSEPACK ST serials, Beijing, China) containing EDTA as anticoagulant and stored in room temperature. All measurements were performed within 30 min after collection at the hospital clinical chemistry using a Sysmex XE2100 Haematology System (Sysmex Corporation, Kobe, Japan). The reference ranges are 9–13 fL for MPV, 9–17 fL for platelet distribution width (PDW), and 101–320 $\times 10^9$/L and 125–350 $\times 10^9$/L for platelet counts of women and men, respectively.

Adjunctive pharmacotherapy

All patients received routine medication that included aspirin at 300 mg before intervention, unfractioned heparin during PCI on routine basis, clopidogrel at a loading dose of 300 to 600 mg and a maintenance dose of 75 mg daily during hospitalization. The glycoprotein IIb/IIIa inhibitor (abciximab) was administered during PCI at 0.25 mg/kg in bolus followed by a 0.125 μg/kg/min 12-24 h infusion, at the discretion of the operator. Other drugs commonly prescribed were angiotensin-converting enzyme inhibitors or angiotensin II receptor blocker, β-blockers, statins and isonitrate.

Echocardiography

Each patient underwent echocardiography in the first or the second day after acute MI using a GE-Vivid 7 system with a 3.3-MHz multiphase array probe. Patients lying in the left decubitus position for acquisition of cardiac images. LV ejection fraction (LVEF) was determined using a modified biplane version of Simpson's method with apical two- and four-chamber views. Image acquisition and analyses were performed by experienced cardiologists.

Statistical Analysis

Results were presented as mean ± standard deviation (SD) or as percentages. Baseline clinical parameters between sub-groups were compared using Student's unpaired t-tests, One-way ANOVA, Chi-squared tests or Mann-Whitney Test. To test the normal distribution, the Kolmogorov-Smirnov test was used. One-way ANOVA followed by post-hoc analysis was used for comparison of hematological indices at various time-points. Repeated-measures ANOVA followed by post-hoc analysis was used for comparison of MPV values among different Killip Class groups and at various time-points or influence of abciximab use on MPV values. Spearman or Pearson correlation was used to identify the bivariate correlations. To assess the value of MPV in predicting the admission Killip Class score, logistic regression was performed to obtain odds ratio (OR) and 95% confidence interval (CI) with all confounders (including age, sex, fasting blood glucose levels, diabetes mellitus, hypertension, pre-infarction angina, time from onset to admission, serum creatinine; anterior MI, ST segment resolution after PCI, and CTFC) entered in the selection

procedure with a *p* level <0.05 staying in the model. Statistical significance was defined as $p<0.05$. All analyses were performed with SPSS for Windows version 15.0 (SPSS, Chicago, IL).

Results

Baseline characteristics of all study population

Of a total of 474 consecutive patients with STEMI and successful PCI, 99 patients were excluded for the following reasons: infectious disease (n=44), usage of clopidogrel prior to the onset (n=30, this group had taken clopidogrel 75mg daily continuously at least for one week), major renal or hepatic disease (n=17), other causes of acute MI (n=6) or malignancy (n=2). Finally, 375 patients were enrolled in the study. Baseline patient characteristics and medication are shown in Table 1. The mean interval from onset the symptom to hospital arrival was 4.8 ± 4.0 h and the door to PCI time was 95 ± 26 min. Of them, 279 (74%) had Killip Class 1 and higher score (≥2) was assigned to 96 cases (26%).

Changes in platelet indices after acute MI

Fig. 1 shows the time course of platelet related indices measured from admission through to day-7 after MI. MPV reached its peak value at admission (10.16 ± 1.05 fL), reduced by 16% within 24 hours ($p<0.0001$, Fig. 1) and then remained at this level during day-1 to day-7. Conversely, PDW level was the lowest at admission (12.66 ± 1.96 fL), and then increased from day-1 to day-7 ($p<0.0001$). Platelet counts were the lowest at day-3 and higher at the admission or day-7 ($p<0.0001$, Fig. 1).

Correlation among platelet indices

There was a moderate but significant inverse correlation between MPV levels and platelet counts at admission, day-1, -3 and -7 (r values were -0.339, -0.330, -0.387 and -0.473, respectively, all $p<0.0001$). Whilst MPV levels were highly correlated with PDW at the admission (Fig. 2A), such correlation become much weaker or disappeared during the subsequent time points studied (Fig. 2B-D). Notably, such a close correlation, seen at the admission, was maintained during days 1-7 in a small fraction of patients, and most of them had Killip Class ≥3 (Fig. 2B-D). MPV at the admission was weakly but significantly correlated with counts of white blood cell (r=0.153), neutrophils (r=0.147), monocytes (r=0.117) and blood glucose level (r=0.176, all $p<0.05$).

Baseline characteristics of patients with different Killip Class

The clinical characteristics and laboratory findings of three sub-groups with different Killip Class are summarized in Table 2. There was no significant difference between the three groups in receiving anti-platelet drugs during hospitalization. Risk factor profile and some baseline clinical parameters such as male percentage, body mass index (BMI), lipid profile and concomitant illness were comparable between the three groups. Compared with Killip Class-1 group, patients with higher Killip Class were older, more likely to suffer from diabetes mellitus as co-morbidity, had higher levels of high-sensitive C-reactive protein (hs-CRP), fasting glucose, N-terminal pro B-type

natriuretic peptide (Nt-proBNP) and CTFC. Moreover, the group with higher Killip Class had lower LVEF and more likely to have anterior infarction than those in the low Killip Class group (Table 2).

Relation between MPV and Killip Class or other clinical parameters

The admission MPV values correlated with impaired post-PCI myocardial reperfusion measured by CTFC (Fig. 3).

In all subgroups, peak MPV values occurred at the admission, then reduced within 24 hours and then remained at this level during days 1–7 ($p<0.0001$). Repeated-measures ANOVA showed greater MPV values in patients with high Killip Class at all the time points studied (Fig. 4, $p<0.0001$). After adjusting for common confounding factors, high MPV remained as an independent predictor for high Killip Class at the admission (OR 1.873, CI 95% 1.373 - 2.673, $p=0.001$).

The platelet counts were slightly but significantly lower in the sub-group with the higher Killip Class score in day-1 after MI. Although PDW was higher at the admission in patients with higher Killip Class, such difference was not evident during 1-7 days after MI with PDW values actually lower in the group with Killip Class ≥3 (Table 2).

MPV and anti-platelet medication

To evaluate influence of clopidogrel on early changes in platelet indices, we compared MPV, PDW and platelet counts between patients with (n=30, this group had the same inclusion criteria except clopidogrel pre-usage) and without (n=375) clopidogrel pre-usage. Risk factor profile, baseline clinical parameters and Killip classification percentages were comparable between the two groups (data not shown). At the admission, clopidogel pre-use was associated with a significant smaller MPV (9.27 ± 0.86 fL vs. 10.16 ± 1.05 fL, $p=0.027$) and lower PDW (11.6 ± 1.4 vs. 12.7 ± 2.0 fL, $p<0.01$), without change in platelet counts (220 ± 59 vs. 230 ± 66 $\times 10^9$/L, $p=0.41$).

Potential influence by Abciximab on MPV was examined by comparison of two subgroups with (n=117) and without (n=258) use of Abciximab at the discretion of higher risk patients by the operator. Compared with non-user counterparts, the Abciximab users had higher prevalence of family history of coronary heart disease (21.9 % vs. 15.3 %), larger infarct size estimated by peak values of creatine kinase (CK) (3189 ±2280 vs. 1879 ±1548 IU/L) and CK-MB (264 ±168 vs. 196 ±137 IU/L, both $p<0.0001$), and a poor reflow estimated by CTFC (35.7 ±20.5 vs. 30.6 ±18.7, $p=0.025$). Other indices were similar between both sub-groups. Repeated-measures ANOVA showed that the abciximab use had no effect on the levels of MPV measured at all the three time points studied ($p>0.05$, data not shown).

Discussion

To our knowledge, this is the first study showing that in patients with MI who had successful PCI with an average of 5 hours from symptom onset, MPV and other platelet indices undergo dynamic changes within the first 24 hours. Specifically, MPV reached its peak value at the admission, and then reduced sharply by 16% within 24 hours after acute MI and then maintained at this level afterwards. Importantly, MPV values were greater in patients with Killip Class ≥2 than

those with Killip Class-1.

Higher MPV has been associated with presence of cardiovascular risk factors such as diabetes, chest pain due to ACS, and adverse outcome after ACS [6,14]. Inflammatory conditions are also associated with enlarged MPV [6,14]. Clinical and experimental studies have shown that platelets contribute significantly to post-infarct inflammatory responses by binding to and activating circulating monocytes [15-17]. In our study, the peak MPV observed at the admission may be related to all these conditions. Indeed, a weak but significant correlation was observed between admission MPV and white blood cell counts. Platelet volume is determined at thrombopoiesis and increased platelet volume are correlated with higher DNA concentration in the nucleus of megakaryocytes [18]. There is evidence that larger platelets are functionally more active in terms of thrombotic and possibly inflammatory activities [18]. Collectively, higher MPV measured prior to or upon onset of MI represents a general pro-thrombotic state rather than an acute-phase reactant [19]. The source of large circulating platelets at the onset time of MI remains to be identified.

The sharp reduction in MPV within the first 24 hours is likely due to routine treatment including PCI, anti-platelet drugs or other drugs including contrast agents. Clopidogrel is known to improve reperfusion by block the $P2Y_{12}$ receptor that is critical in mediating platelet activation [20]. In the current study, all patients received aspirin and clopidogrel at the time of PCI, which likely contribute to such a rapid reduction in MPV. There is very limited data on the influence of MPV by anti-platelet drugs. A MPV-lowering effect of clopidogrel is indicated by our finding that patients who received clopidogrel prior to MI had smaller MPV at the admission compared to those who did not. We also observed that abciximab use per se seems to have no effect on changes in MPV during days 1-7 after MI, albeit this observation was made in the presence of clopidogrel. A transient reduction in platelet count was observed during days 1-3, and by day-7 platelet count returned back to the admission level. Anti-platelet medications, particularly clopidogrel may contribute to the transient reduction in platelet count seen at day-3. Consumption of platelets within the first days after MI may exert strong stimulus for increased platelets biogeneration and release by bone marrow megakaryocutes.

MPV and platelet count are known to be negatively correlated [21,22]. In our study, such an inverse correlation maintains throughout the first 7 days. Increases in both MPV and PDW are indicative of platelet activation [23]. Activation of platelets is associated with morphological changes including its spherical shape and formation of pseudopodia, which are more likely reflected by PDW [14,24]. Earlier studies showed that MPV and PDW were significantly raised in patients with acute MI compared with those with stable coronary heart disease or healthy subjects [25]. In contrast, our dynamic monitoring of platelet indices revealed PDW value at its lowest at the admission and then increased from around 24 hours onwards after MI. Although we did not have a matched healthy control group for comparison, PDW values measured at 1-7 days post MI are close to the upper limit of the normal reference range (9-17 fL). Interestingly, admission MPV and PDW were highly correlated, but such correlation largely disappeared at the other three time points studied. The reason for this phenomenon remains unclear based on this study, but might reflect turnover of population of platelets from different sources under conditions of MI and/or effects of anti-platelet treatment. Interestingly, differences in PDW among Killip Class subgroups are apparent during days 1-7, likely due to difference in the sensitivity of platelets to anti-platelet drugs. Further studies are warranted to investigate influence of anti-platelet agents on MPV and PDW.

In addition to coagulative activity [1,2,4,5], platelets have been shown to involve in inflammatory process that plays a crucial role in atherothrombosis [26] as well as post-MI myocardial inflammation and subsequent LV remodeling [14-17]. In addition, elevated MPV was a

simple surrogate for worse microvascular injury in patients with anterior wall MI [27]. It is important to address whether changes in MPV, seen at the time of hospital admission, are associated with adverse clinical consequences. In our cohort, patients with high Killip Class had higher CTFC indicating low- or no-reflow after restoration of epicardial arterial flow by PCI. To our knowledge, our study is the first to imply that platelet activity, estimated by MPV, is a contributing factor in post-MI functional impairment via mediating onset of no-reflow following PCI. Previous studies reported high MPV values being correlated with impaired reperfusion after both PCI and thrombolytic therapy [2,7,8]. The impaired reperfusion and microvascular injury are known to be associated with poor LV function. Patients with higher Killip Class are associated with increased mortality [28,29]. In a clinical study, MPV was higher in decompensated than stable heart failure patients, indicating that there may be some abnormalities of platelet activation during decompensated heart failure [30].

In patients with acute MI, presence of metabolic syndrome and advanced age are associated with poorer LVEF [11] and increased onset of no-reflow phenomenon following PCI [31]. In our study, while the patients with higher Killip Class were indistinguishable from those with Killip Class-1 in peak CK, hemodymanics, location of MI or time to admission, significant differences were observed in age, presence of diabetes, LVEF or CTFC. However, even taking all these factors into consideration, MPV remains to be an independent predictor for Killip Class. Thus, enhanced platelet activity may be an independent contributor for the development of acute pump failure in patients with acute MI.

Limitations

A major limitation of this retrospective study is a relatively small sample size and hence the subgroup analysis could not be performed in more detail. Further, MI patients received different loading doses of clopidogrel, and this factor was not analyzed when investigating the dynamic changes of the platelet indices due in part to the insufficient sample size. In addition, a prospective study with better control of other variables would be necessary to confirm the relationship between the platelet indices and LV dysfunction, and to address some question such as whether the extent of reduction of MPV during the 24 hours will lead to a change in clinical outcome and whether the subgroup of infarcted patients with elevated MPV warrants more aggressive treatment.

Conclusions

In patients with STEMI, platelet indices undergo dramatic changes from admission levels within the first week, particularly the first 24 hours, which may reflect pathophysiological nature of acute MI per se and the influence by therapeutic interventions. MPV at admission was higher in patients with higher Killip Class, suggesting an important role of MPV in predicting LV dysfunction after acute MI. MPV might therefore be a useful tool in monitoring the progression of patients with acute MI along with other biochemical markers.

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Conflict of interest statement

None.

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Figure legends

Fig. 1. The dynamic changes in the platelet indices in patients with STEMI who received successful primary PCI. Platelet indices were measured at the admission, day-1 (D1), day-3 (D3) and day-7 (D7) after MI (n=375). *p<0.05 compared with admission; $^{\#}p$<0.05 compared with D1; $^{\S}p$<0.05 compared with D3; $^{\dagger}p$<0.05 compared with D7. Results are mean ± SD.

Fig. 2. Relationship between MPV and PDW at the admission (A), day-1 (B), day-3 (C) and day-7 (D) in patients with STEMI who received successful PCI (n =375). The difference in the correlation coefficients between the admission and other time-points is statistically significant (p<0.05).

Fig. 3. Relationship between MPV at the admission and corrected TIMI frame count assessed on the final angiogram in patients with STEMI who received successful PCI (n=375).

Fig. 4. Comparison of MPV measured at different time points after MI among three sub-groups of patients with different Killip Class score (1, n=279; 2, n=61; 3, n=35). *p<0.05 compared with patients with Killip Class-1; $^{\#}p$<0.05 compared with patients with Killip Class-2; $^{\dagger}p$<0.05 compared with patients with Killip Class≥3. Results are mean ± SD.

Table 1
Baseline Characteristics of all Study Populations and Post-admission medications

Variable	Values (n=375)
Age (years)	61.6 ± 13.1
Male gender (%)	79.7
Hypertension (%)	52.5
Diabetes mellitus (%)	34.7
Hypercholesterolemia (%)	36.8
Current smoking (%)	49.3
Family history of coronary artery disease (%)	14.9
Body mass index (kg/m^2)	24.0 ± 4.5
Anterior infarction (%)	49.9
Prior angina (%)	62.9
Systolic blood pressure (mmHg)	135 ± 27
Diastolic blood pressure (mmHg)	77 ± 17
Heart rate (beats/min)	76 ± 17
On β-blocker treatment (%)	88
On ACEI or ARB treatment (%)	91
On statin treatment (%)	97
On acetylsalicylic acid treatment (%)	98
On clopidogrel treatment (%)	100
On abciximab treatment (%)	31
Creatinine (μmol/L)	86.9 ± 21.0
Peak creatine kinase (IU/L)	2288 ± 1903
Peak creatine kinase-MB (IU/L)	217 ± 150
ST-segment resolution ≥50% within 120 minutes after PCI (%)	83.2
Corrected TIMI frame count (CTFC)	31.3 ± 19.5

Values are represented as mean ± S.D. or the percentages. ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin II receptor blocker; PCI: primary percutaneous coronary intervention.

Table 2
Baseline Characteristics of Patients in Different Killip Classes

Variable	Killip Class			P value
	1	2	≥3	
Group size (n=, %)	279 (74.4%)	61 (16.2%)	35 (9.3%)	
Age (year)	60.0 ± 13.0$^{\#\dagger}$	65.6 ± 11.8*	66.6 ± 13.0*	0.001
Male sex (%)	82.1	73.8	71.4	0.151
Hypertension (%)	51.3	59.0	51.4	0.541
Diabetes mellitus (%)	29.0	54.1	45.7	<0.0001
Hypercholesterolemia (%)	36.2	37.7	40.0	0.896
Current smoking (%)	64.5	59.0	54.1	0.548
Family history of CAD (%)	16.1	8.2	17.1	0.269
Body mass index (kg/m^2)	24.7 ± 3.5	24.9 ± 5.0	22.3 ± 4.3	0.876
Anterior infarction (%)	46.8	54.1	68.6	0.041
Prior angina (%)	63.8	57.4	65.7	0.842
SBP (mmHg)	135 ± 27	136 ± 29	135 ± 20	0.945
DBP (mmHg)	78 ± 17	76 ± 19	78 ± 15	0.595
Heart rate (beat/min)	76 ± 16	76 ± 21	78 ± 22	0.811
LVEDd (mm)	49.4 ± 5.0	49.4 ± 5.0	52.0 ± 5.7	0.073
LVEF (%)	55.2 ± 8.0$^{\#\dagger}$	52.0 ± 7.3*†	50.1 ± 8.8$^{*\#}$	<0.0001
Time to admission (h)	4.8 ± 3.9	4.8 ± 3.9	5.6 ± 4.9	0.628
ST-segment resolution ≥50% within 120 min after PCI (%)	89.6	78.7	88.2	0.065
Corrected TIMI frame count (CTFC)	29.9 ± 17.8$^{\#\dagger}$	37.9 ± 22.6*	45.6 ± 23.0*	<0.0001
Peak CK (IU/L)	2206 ± 1849	2375 ± 1941	2784 ± 2217	0.181
Peak CK-MB (IU/L)	220 ± 145	230 ± 159	166 ± 168	0.111
White blood cell (×10^9/L)	9.91 ± 2.98	10.14 ± 2.78	10.41 ± 2.91	0.627
Neutrophils (×10^9/L)	7.31 ± 2.88	7.65 ± 2.90	8.10 ± 2.71	0.310
Monocytes (×10^9/L)	0.49 ± 0.24	0.46 ± 0.20	0.47 ± 0.29	0.737
Lymphocytes (×10^9/L)	2.06 ± 1.12	1.97 ± 0.89	1.77 ± 1.15	0.363
Hemoglobin (g/L)	145.4 ± 19.0	141.7 ± 16.3	139.2 ± 30.5	0.159
hs-CRP (mg/L)	16.58 ± 25.75	18.79 ± 23.95	25.82 ± 28.12	0.010
Creatinine (μmol/L)	85.8 ± 20.6	91.9 ± 21.3	87.6 ± 19.1	0.162
Nt-proBNP (pg/mL)	1644 ± 2249	2492 ± 3765	4329 ± 3963	<0.0001
Serum glucose (mmol/L)	6.29 ± 2.40$^{\#\dagger}$	7.63 ± 3.94*	7.82 ± 3.96*	<0.0001
Total cholesterol (mmol/L)	4.71 ± 0.98†	4.64 ± 0.93	4.29 ± 0.90*	0.048
Triglyceride (mmol/L)	1.81 ± 0.99	1.76 ± 1.16	1.49 ± 0.79	0.210
HDL-C (mmol/L)	0.95 ± 0.24	0.92 ± 0.20	0.96 ± 0.18	0.683
LDL-C (mmol/L)	3.03 ± 0.89	2.92 ± 0.66	2.70 ± 0.83	0.078
Platelet counts (×10^9/L)				
admission	223.7 ± 58.6	214.2 ± 66.0	197.7 ± 42.3	0.130
Day-1	207.6 ± 54.9$^{\#}$	191.5 ± 52.3*	190.7 ± 40.5	0.045

Day-3	190.1 ± 53.4	178.1 ± 51.4	178.9 ± 37.0	0.230
Day-7	222.8 ± 54.6	215.2 ± 56.6	200.8 ± 45.4	0.090
PDW （fL)				
admission	12.37 ± 1.68$^{\#\dagger}$	13.26 ± 2.18*	14.02 ± 2.77*	<0.0001
Day-1	16.6 ± 0.72†	16.7 ± 0.47†	15.5 ± 2.30$^{*\#}$	<0.0001
Day-3	16.7 ± 0.54†	16.8 ± 0.47†	16.0 ± 1.90$^{*\#}$	<0.0001
Day-7	16.6 ± 0.54†	16.8 ± 0.44†	16.2 ± 2.34$^{*\#}$	0.024

Values are represented as mean ± SD or the percentages. *p<0.05 compared with patients with Killip Class–1; $^{\#}p$<0.05 compared with patients with Killip Class–2; $^{\dagger}p$<0.05 compared with patients with Killip Class≥3.

SBP: systolic blood pressure; DBP: diastolic blood pressure; CAD: coronary artery disease; CK: creatine kinase; LVEDd: left ventricular end-diastolic dimension; LVEF: left ventricular ejection fraction; PCI: percutaneous coronary intervention; hs-CRP: high-sensitive C-reactive protein; Nt-proBNP: N-terminal pro B-type natriuretic peptide; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol; PDW: platelet distribution width.

Figure

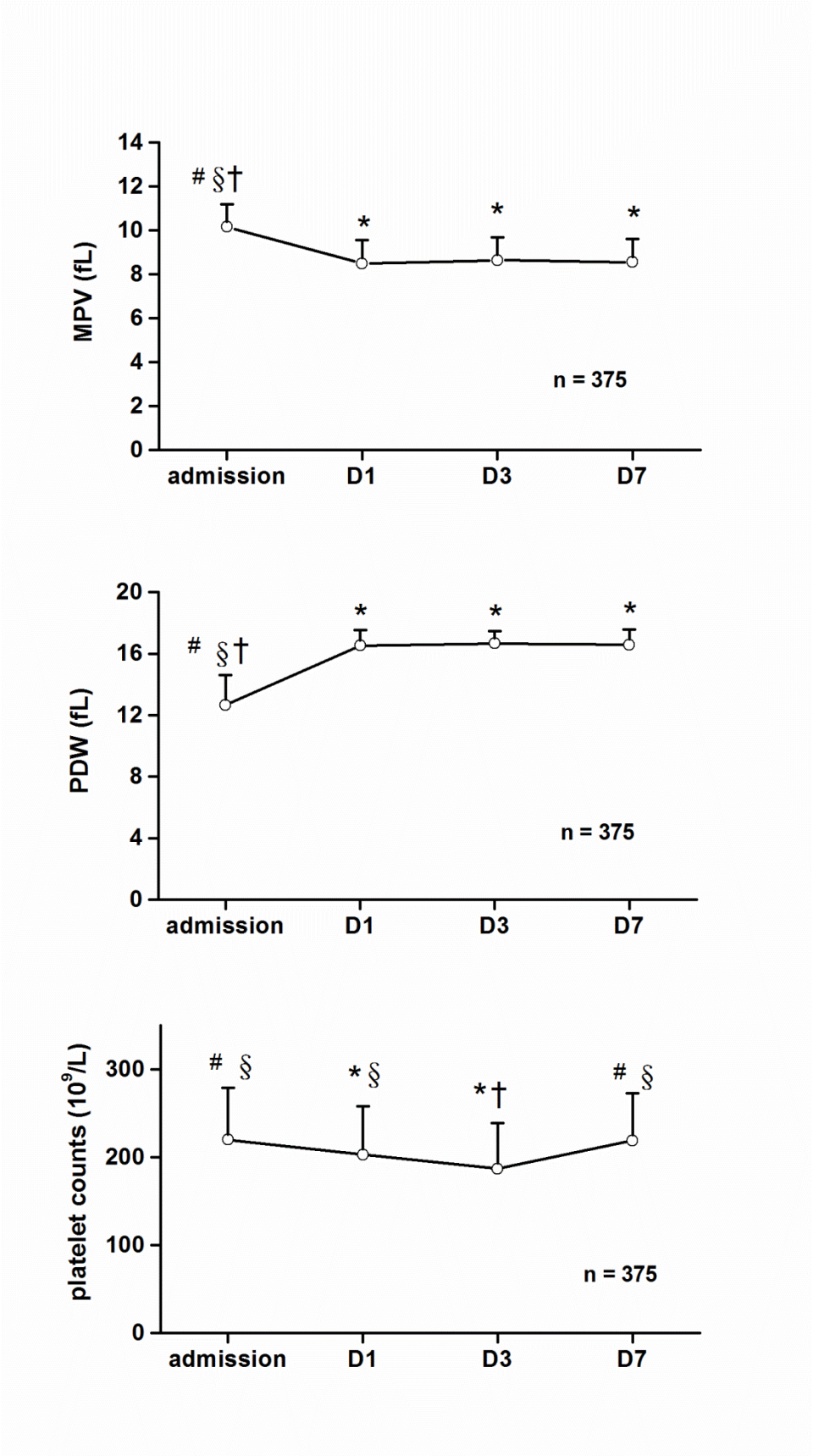

Figure 1

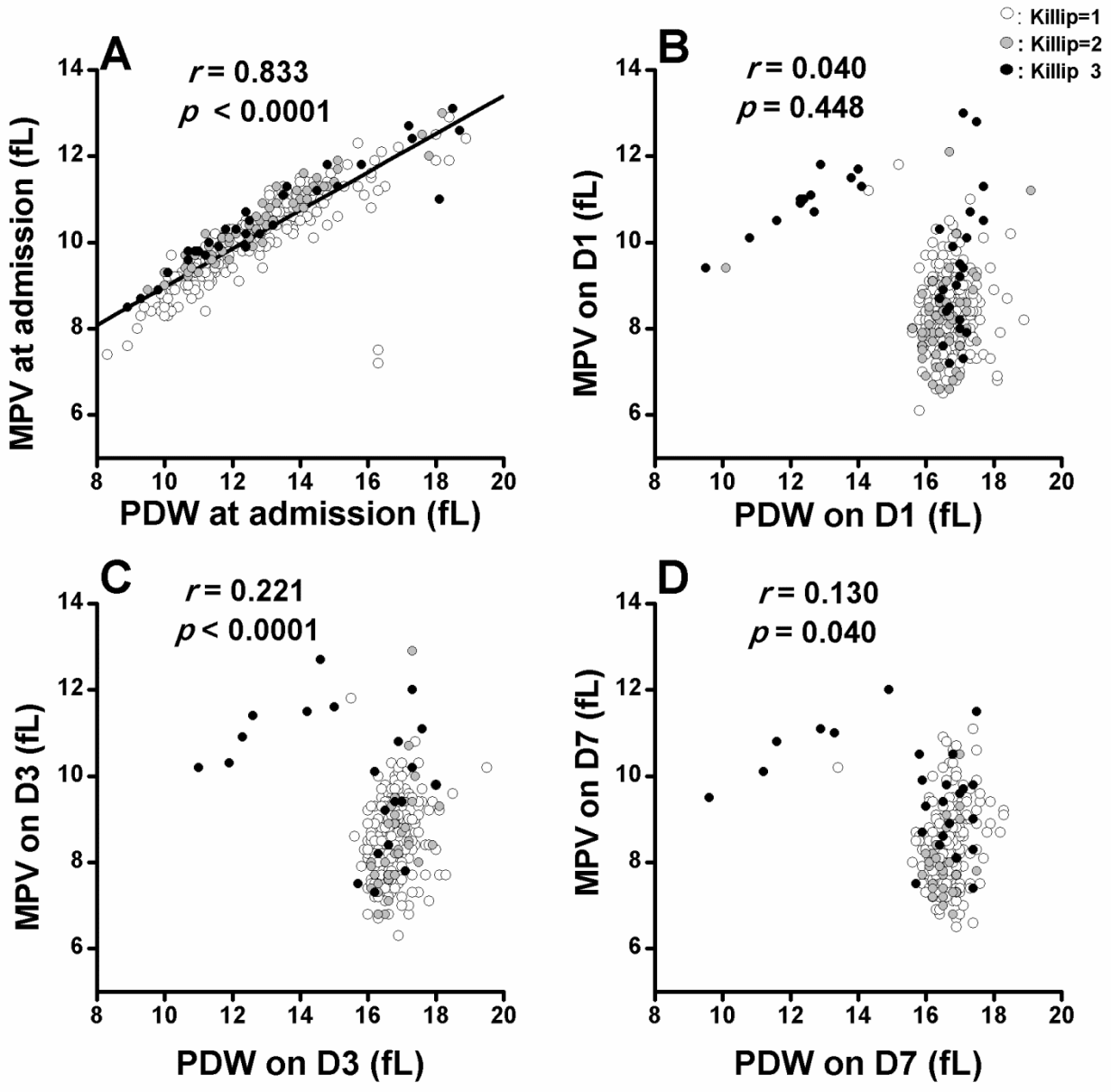
A
r = 0.833
p < 0.0001
MPV at admission (fL)
PDW at admission (fL)
B
r = 0.040
p = 0.448
MPV on D1 (fL)
PDW on D1 (fL)
: Killip=1
: Killip=2
: Killip 3
C
r = 0.221
p < 0.0001
MPV on D3 (fL)
PDW on D3 (fL)
D
r = 0.130
p = 0.040
MPV on D7 (fL)
PDW on D7 (fL)

Figure 2

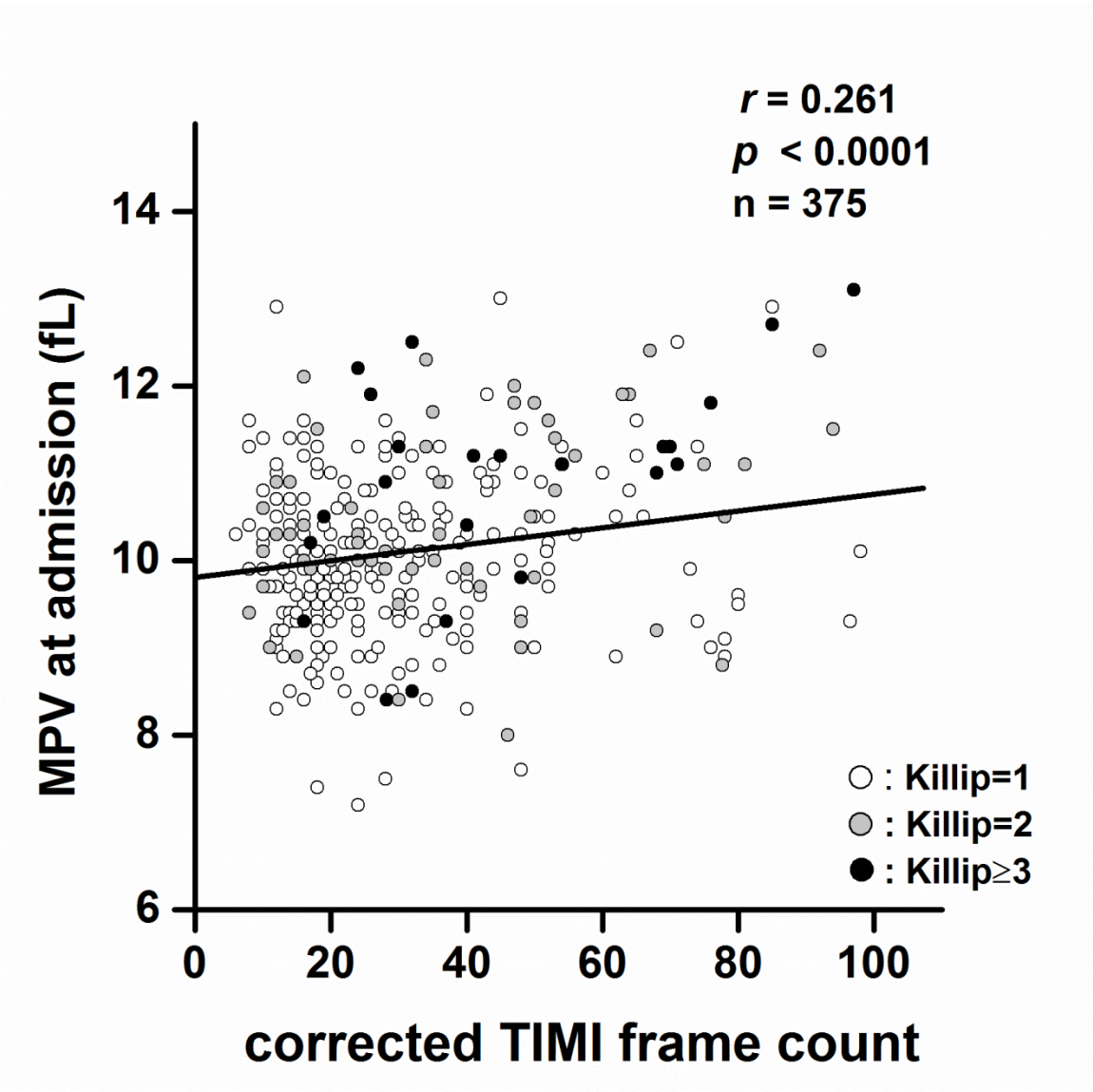
r = 0.261
p < 0.0001
n = 375
MPV at admission (fL)
14
12
10
8
6
0
20
40
60
80
100
corrected TIMI frame count
: Killip=1
: Killip=2
: Killip≥3

Figure 3

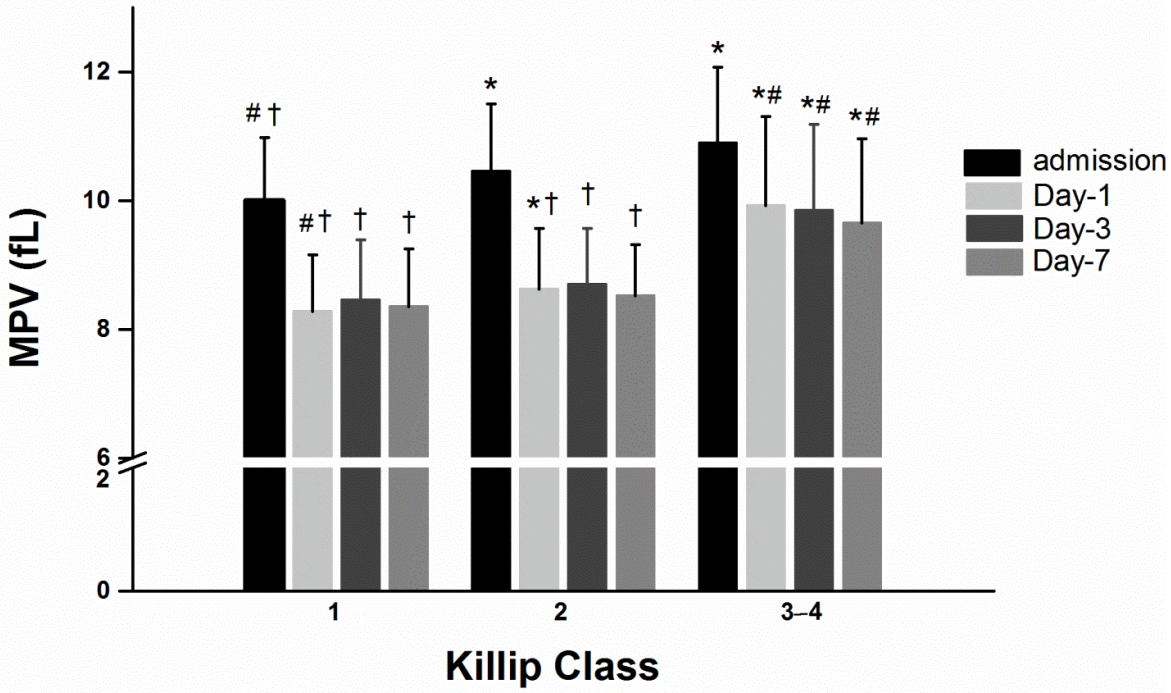

Figure 4

Comments

This study showed that in patients with acute myocardial infarction and received primary percutaneous coronary intervention, MPV reached the peak value at the admission, and was higher in patients with high Killip Class scores, suggesting a predictive value of MPV in ventricular dysfunction and in-hospital clinical outcome post-MI.

Highlights

MPV was higher in high Killip Class group and may predict ventricular dysfunction.

Conflict of Interest Form

Manuscript Name:	Serial changes of mean platelet volume in relation to Killip Class etc.
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Details of nature of conflict of interest:	None.