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Association between baseline platelet count and severe adverse outcomes following percutaneous coronary intervention

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Abstract

Objective The aim is to evaluate the association between baseline platelet count (PC) and severe adverse outcomes following percutaneous coronary intervention (PCI) in current real-world practice. **Methods** A total of 18,788 patients underwent PCI with drug-eluting stents constituted the study population. Patients were categorized as having low (< 150 × 1000/μL), normal (150–300 × 1000/μL), and high (≥ 300 × 1000/μL) baseline PC. The primary endpoints included in-hospital and follow-up all-cause mortality. The secondary endpoint was major bleeding requiring a blood transfusion. **Results** In-hospital mortality rates for patients with low, normal, and high baseline PC were 0.6%, 0.4%, and 0.4%, respectively (P = 0.259). Similarly, mortality rates during long-term follow-up (median 23.8 months) for patients with low, normal, and high baseline PC were 0.9%, 0.6%, and 0.7%, respectively (P = 0.079). After multivariate adjustment, patients with low or high baseline PC tended to have similar risks for both in-hospital and follow-up mortality compared with the normal group. Subgroup analyses failed to demonstrate an independent prognostic value of baseline PC in specific population groups except patients who undwent transfemoral PCI. There was also no significant difference in the incidence of major bleeding requiring a blood transfusion in the low, normal, and high groups (0.5%, 0.3%, and 0.3%, respectively; P = 0.320). After multivariate adjustment, low or high baseline PC did not significantly increase the risk of major bleeding. **Conclusion** There is no significant association between baseline PC and severe adverse outcomes following PCI in current real-world practice.

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Keywords: Major bleeding; Mortality; Percutaneous coronary intervention; Platelet count

1 Introduction

Thrombosis and hemorrhage represent the main challenges of myocardial revascularization.[1,2] Platelets play a key role in the pathophysiological process of both thrombosis and hemorrhage.[3,4] An abnormal increase (thrombocytosis) or decrease (thrombocytopenia) in platelets may cause defective formation of hemostatic plugs and bleeding.[5,6] Accordingly, such patients were excluded from the vast majority of clinical trials, given the potentially increased risks. Few pooled post-hoc analyses[7–15] and cohort studies[13–15] drew inconsistent conclusions based on data mostly from thrombolysis or bare metal stents (BMS) era. Furthermore, contemporary treatment regimens have changed a lot over the last decade with common use of drug eluting stents (DES) and advances in adjunctive pharmacotherapy, but latest evidence is rare. The aim of this study is to evaluate the association between baseline platelet count (PC) and severe adverse outcomes following percutaneous coronary intervention (PCI) in current real-world practice both at short-term and long-term follow-up by analyzing data from the Beijing Heart and Metabolism Survey (BHMS).

2 Methods

2.1 Study design and patient population

BHMS is an investigator-initiated, multicenter cohort study conducted at five tertiary medical centers. The PC obtained at baseline, using a Coulter Counter method, was considered. From April 2004 to October 2010, a total of 21,620 consecutive patients receiving PCI were recruited. And only those implanted with DES were considered eligible for the study. To enhance homogeneity and ensure examination of a representative cohort in the context of contemporary treatment regimens, 843 (3.9%) patients receiving plain old balloon angioplasty without stent implantation

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and 1738 (8.0%) patients who underwent BMS implantation were excluded. Two hundred and eleven (1.0%) patients without completed baseline data and 40 (0.2%) with a terminal illness were also excluded. Thus a total of 18,788 patients constituted the cohort. Patients in our cohort were categorized as having low (< 150 × 1000/μL), normal (150–300 × 1000/μL), and high (≥ 300 × 1000/μL) baseline PC. In the overall cohort, the average length of in-hospital stay was 8 ± 6 days and the mean length of follow-up was about 2 years (25 ± 17 months). Figure 1 shows the study design and flow chart. The study protocol was reviewed and approved by the Ethics Committee of each participating institution.

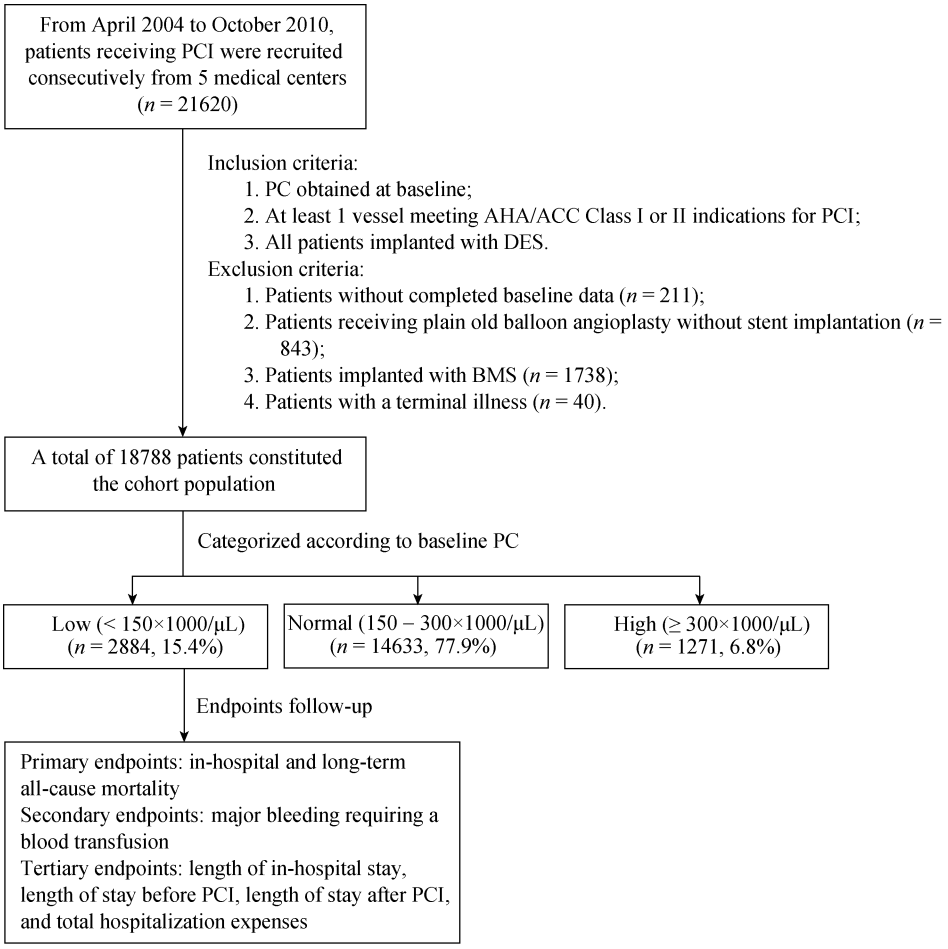

Figure 1. The study flow chart. PCI: percutaneous coronary intervention; PC: platelet count; AHA/ACC: American Heart Association/American College of Cardiology; DES: drug eluting stent; BMS: bare metal stent.

2.2 Clinical endpoints

The primary endpoints were in-hospital and follow-up all-cause mortality. The secondary endpoint was major bleeding requiring a blood transfusion. The tertiary endpoints included length of stay before PCI, length of stay after PCI, length of in-hospital stay, and the total hospitalization expenses.

2.3 Statistical analysis

Statistical analysis was performed using Statistical Package for Social Sciences (SPSS). All categorical variables were expressed as percentages and compared with Pearson chi-square test or Fisher exact test; continuous variables were expressed as median (interquartile range) and nonparametric tests were used (Kruskal-Wallis test for > 2 groups). Multivariate analyses with Cox proportional hazards methods derived the independent predictors of adverse events. Variables were selected for submission to the model if the univariate *P* value was < 0.25 or the variable was of known clinical significance but failed to meet the critical α level for inclusion.[7–15]

3 Results

3.1 Baseline demographic and clinical characteristics

As an overall cohort, the mean ± SD age was 60 ± 11

years with the median age of 60 (52–68) years. Male patients (n = 13,922) account for 74.1% of the cohort. There were 5384 (28.7%), 11,425 (60.8%), 2832 (15.1%), and 2915 (15.5%) patients had a history of diabetes mellitus (DM), hypertension, hyperlipidemia and prior myocardial infaction (MI), respectively. A total of 13,283 patients had the myocardial dysfunction with different New York Heart Association (NYHA) functional classes, among which class II, III and IV accounted for 54.6%, 12.8% and 3.4%, respectively. The prevalences of stable coronary artery disease (SCAD), unstable angina pectoris (UAP) and acute myocardial infarction (AMI) were 23.2%, 48.5% and 28.3%, respectively.

3.2 Distribution of baseline PC

The distribution of baseline PC was a skewed distribution with a median value of 2 × 1000/μL (165–240 × 1000/μL) and a mean value of (206 ± 60) × 1000/μL (Figure 2A). A majority of the baseline PC (14,633, 77.9%) were normally distributing in the range of 150–300 × 1000/μL (figure 2B). In addition, there were 2884 (15.4%), 1271 (6.8%) patients had their baseline PC < 150 × 1000/μL, and ≥ 300 × 1000/μL, respectively. Only 11 patients had their baseline PC lower than 50 × 1000/μL with the minimum value of 13 × 1000/μL (Figure 2C). And 4 patients in the high group had their baseline PC higher than 600 × 1000/μL with the maximum value of 664 × 1000/μL (Figure 2D).

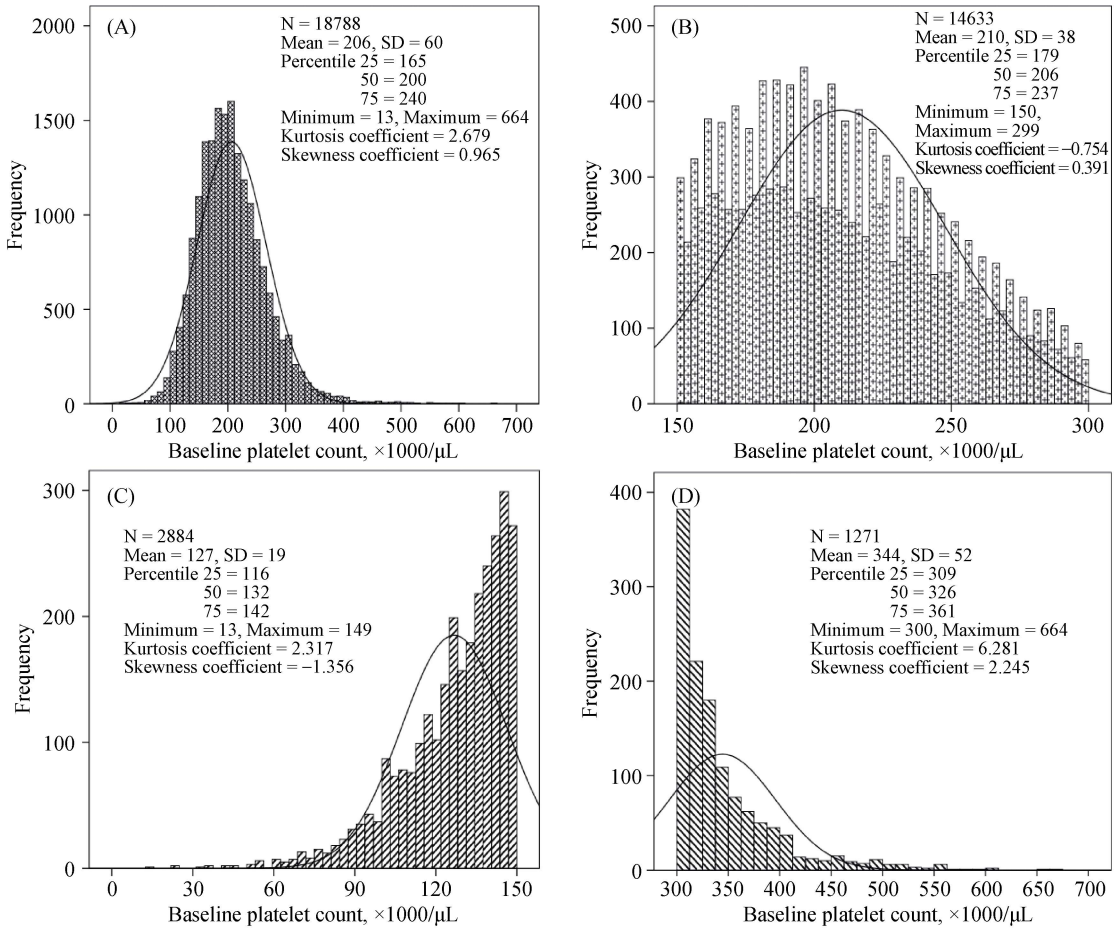

Figure 2. The distribution of baseline platelet count. (A): The distribution of baseline platelet count in the overall cohort; (B): the distribution of baseline platelet count in the normal group; (C): the distribution of baseline platelet count in the low group; (D): the distribution of baseline platelet count in the high group.

3.3 Comparison of baseline characteristics among groups

As detailed in Table 1, among the 3 groups there were major differences in baseline clinical characteristics, which in the low group were almost the opposite of the high group. From an angiographic and procedural viewpoint (Table 2), patients with lower baseline PC were more likely to have left main (LM) disease and left main multi-vessel disease (LMMVD). They were treated somewhat more frequently with single stent with shorter length and larger diameter. In addition, transradial approach PCI was more likely preferred

in the low group. Compared with the normal group, patients in the high group were also more likely to have LM disease and were treated more frequently with single stent with relatively shorter length and larger diameter.

With respect to medications used in hospital and upon discharge (Table 3), there were no significant differences

Table 1. Baseline clinical characteristics.

Characteristics	Low (*n* = 2884)	Normal (*n* = 14633)	High (*n* = 1271)	*P* value
Clinical variables				
Age, yrs	64 (55, 71)	59 (52, 68)	57 (49, 66)	< 0.001
Age ≥ 75, yrs	351 (12.2%)	1115 (7.6%)	75 (5.9%)	< 0.001
Female	519 (18.0%)	3906 (26.7%)	441 (34.7%)	< 0.001
Diabetes	901 (31.2%)	4146 (28.3%)	337 (26.5%)	0.001
Hypertension	1682 (58.3%)	8974 (61.3%)	769 (60.5%)	0.010
Hyperlipidemia	344 (11.9%)	2284 (15.6%)	204 (16.1%)	< 0.001
Prior myocardial infarction	374 (13.0%)	2328 (15.9%)	213 (16.8%)	< 0.001
Diagnosis				
SCAD	706 (24.5%)	3389 (23.2%)	273 (21.5%)	
UAP	1377 (47.7%)	7207 (49.3%)	521 (41.0%)	< 0.001
AMI	801 (27.8%)	4037 (27.6%)	477 (37.5%)	
NYHA functional classification				
Class 1	798 (27.7%)	4287 (29.3%)	421 (33.1%)	
Class 2	1551 (53.8%)	8092 (55.3%)	624 (49.1%)	
Class 3	409 (14.2%)	1800 (12.3%)	179 (14.1%)	0.073
Class 4	126 (4.4%)	454 (3.1%)	47 (3.7%)	
Laboratory variables				
Creatinine, umol/L	80.8 (69.0, 94.0)	77.0 (66.0, 90.1)	73.1 (61.1, 85.4)	< 0.001
FPG, mmol/L	6.0 (5.1, 7.9)	5.9 (5.1, 7.4)	5.8 (5.0, 7.2)	< 0.001
LDL-C, mmol/L	2.6 (2.0, 3.2)	2.8 (2.3, 3.5)	3.0 (2.4, 3.7)	< 0.001
HDL-C, mmol/L	0.9 (0.8, 1.1)	0.9 (0.8, 1.1)	0.9 (0.8, 1.1)	< 0.001
K^+, mmol/L	4.0 (3.8, 4.3)	4.1 (3.9, 4.4)	4.3 (4.0, 4.5)	< 0.001
LVEDD, mm	49 (46, 53)	48 (45, 52)	49 (45, 53)	< 0.001
LVEF, %	62 (54, 68)	62 (55, 68)	62 (55, 68)	0.001

Data were presented as median (interquartile range) for quantitative variables or as *n* (%) for qualitative variables. AMI: acute myocardial infarction; FPG: fasting plasma, glucose; HDL-C: high-density lipoprotein cholesterol; K^+: potassium ion**;** LDL-C: low-density lipoprotein cholesterol; LVEDD: left ventricular end diastolic dimension; LVEF: left ventricular ejection fraction; NYHA: New York Heart Association; SCAD: stable coronary artery disease; UAP: unstable angina pectoris.

Table 2. Angiographic and procedural characteristics.

Characteristics	Low (*n* = 2884)	Normal (*n* = 14633)	High (*n* = 1271)	*P* Value
Angiographic stenosis location				
LM	170 (5.9%)	745 (5.1%)	81 (6.4%)	0.045
LAD	2196 (76.1%)	1,1035 (75.4%)	974 (76.6%)	0.477
LCX	1455 (50.5%)	7163 (49.0%)	591 (46.5%)	0.060
RCA	1569 (54.4%)	7661 (52.4%)	662 (52.1%)	0.120
LM or multivessel disease	1717 (59.5%)	8369 (57.2%)	713 (56.1%)	0.039
Number of stents implanted	1 (1, 2)	1 (1, 2)	1 (1,2)	< 0.001
Single stent implanted	1866 (64.7%)	8531 (58.3%)	770 (60.6%)	< 0.001
Stent diameter, mm	3.0 (2.75, 3.5)	3.0 (2.5, 3.5)	3.0 (2.5, 3.5)	0.003
Stent diameter < 3.0 mm	1058 (36.7%)	6204 (42.4%)	520 (40.9%)	< 0.001
Stent length, mm	21 (18, 28)	24 (18, 29)	24 (18, 28)	< 0.001
Intraoperative GP IIb/IIIa inhibitor administration	326 (11.3%)	2283 (15.6%)	244 (19.2%)	< 0.001
Transradial approach	1568 (54.4%)	6848 (46.8%)	599 (47.1%)	< 0.001

Results are expressed as median (interquartile range) for quantitative variables or as *n* (%) for qualitative variables. GP: glycoprotein; LAD; left anterior descending; LCX: left circumflex; LM: left main; RCA: right coronary artery.

Table 3. Medications used in hospital and upon discharge.

Variable	Low (*n* = 2884)	Normal (*n* = 14633)	High (*n* = 1271)	*P* value
Aspirin				
Loading therapy				
Loading dose ≥ 300 mg/d	2466 (85.5%)	12862 (87.9%)	1098 (86.4%)	
Loading dose < 300 mg/d	176 (6.1%)	849 (5.8%)	75 (5.9%)	0.058
No loading dose	242 (8.4%)	922 (6.3%)	98 (7.7%)	
Maintenance therapy				
Maintenance dose ≥ 81 mg/d	2527 (87.6%)	12956 (88.5%)	1101 (86.6%)	
Maintenance dose < 81 mg/d	218 (7.6%)	849 (5.8%)	79 (6.2%)	0.010
No maintenance dose	139 (4.8%)	828 (5.7%)	91 (7.2%)	
Clopidogrel				
Loading therapy				
Loading dose ≥ 300 mg/d	2662 (92.3%)	13477 (92.1%)	1174 (92.4%)	
Loading dose < 300 mg/d	173 (6.0%)	922 (6.3%)	75 (5.9%)	0.162
No loading dose	49 (1.7%)	234 (1.6%)	22 (1.7%)	
Maintenance therapy				
Maintenance dose ≥ 75 mg/d	2788 (96.7%)	14169 (96.8%)	1229 (96.7%)	
Maintenance dose < 75 mg/d	61 (2.1%)	293 (2.0%)	26 (2.0%)	0.946
No maintenance dose	35 (1.2%)	171 (1.2%)	16 (1.3%)	
LMWH	2168 (75.2%)	11116 (76.0%)	925 (72.8%)	0.033
PPI	1358 (47.1%)	6266 (42.8%)	524 (41.2%)	< 0.001
Statin	2628 (91.1%)	13338 (91.2%)	1133 (89.1%)	0.055
ACEI	1460 (50.6%)	7435 (50.8%)	663 (52.2%)	0.625
ARB	506 (17.5%)	2567 (17.5%)	211 (16.6%)	0.695
CCB	744 (25.8%)	4272 (29.2%)	368 (29.0%)	0.001
β-blocker	1960 (68.0%)	10141 (69.3%)	832 (65.5%)	0.010

Data were presented as n (%) for qualitative variables. ACEI: angiotensin converting enzyme inhibitors; ARB: angiotensin receptor blockers; CCB: calcium channel blockers; LMWH: low molecular weight heparins; PPI: proton pump inhibitors.

regarding the use of clopidogrel among the 3 groups, whereas maintenance dose and loading dose of aspirin tended to be lower in the low group. The use of proton pump inhibitor (PPI) was more frequent in the patients with lower baseline PC than the others.

3.4 Association between baseline PC and clinical outcomes

3.4.1 Primary endpoints

In the overall cohort, 77 patients (0.4%) died in the hospital, and 120 patients (0.6%) died during the long-term follow-up (median 23.8 months). Compared with the normal group, both the low and high groups had the similar in-hospital and follow-up all-cause mortality (Table 4). In-hospital mortality rates for patients in the low, normal, and high group were 0.6%, 0.4%, and 0.4%, respectively; $P = 0.259$; and follow-up mortality rates for patients in the low, normal, and high group were 0.9%, 0.6%, and 0.7%, respectively; $P = 0.079$. After multivariable adjustment, patients with lower or higher baseline PC tended to have similar risks for both in-hospital and follow-up mortality compared with the normal group. As indicated in Table 5, hazard ratios (HRs) and 95% confidence intervals (CIs) of in-hospital death for the low and high group were 0.843 (95% CI: 0.412–1.723; $P = 0.639$) and 0.668 (95% CI: 0.236–1.890; $P = 0.447$), respectively; and the HRs of follow-up death for the low and high group were 1.204 (95% CI: 0.708–2.049; $P = 0.493$) and 0.942 (95% CI: 0.407–2.181; $P = 0.889$), respectively. Further subgroup analyses failed to demonstrate independent prognostic value of baseline PC in specific population groups except patients underwent transfemoral PCI (Figure 3). Kaplan–Meier curves

Table 4. Clinical outcomes at follow-ups.

Variable	Low (*n* = 2884)	Normal (*n* = 14,633)	High (*n* = 1271)	*P* value
Primary endpoints				
In-hospital mortality	17 (0.6%)	55 (0.4%)	5 (0.4%)	0.259
Follow-up mortality	27 (0.9%)	84 (0.6%)	9 (0.7%)	0.079
Secondary endpoints				
Major bleeding requiring a blood transfusion	13 (0.5%)	41 (0.3%)	4 (0.3%)	0.320

Table 5. Independent predictors of in-hospital and long-term mortality.

Variable	In-hospital mortality			Two-year mortality		
	HR	95% CI	*P* Value	HR	95% CI	*P* Value
Age ≥ 75 yrs	2.277	1.264–4.102	0.006	3.103	1.908–5.044	< 0.001
Female	2.240	1.321–3.800	0.003	1.577	1.002–2.480	0.049
Hypertension	0.825	0.484–1.404	0.478	0.792	0.514–1.219	0.289
AMI	3.024	1.736–5.267	< 0.001	2.296	1.473–3.581	< 0.001
Baseline PC						
Low	0.843	0.412–1.723	0.639	1.204	0.708–2.049	0.493
Normal	1.0 (Ref)	—		1.0 (Ref)	—	
High	0.668	0.236–1.890	0.447	0.942	0.407–2.181	0.889
Creatinine, umol/L	1.004	1.001–1.007	0.017	1.004	1.002–1.007	0.001
FPG, mmol/L	0.958	0.869–1.057	0.396	1.062	0.997–1.131	0.061
LDL-C, mmol/L	1.164	0.889–1.522	0.269	0.972	0.771–1.224	0.807
LVEF, %	0.975	0.954–0.996	0.021	0.973	0.955–0.991	0.003
LM or multivessel disease	1.830	0.980–3.419	0.058	1.655	1.031–2.655	0.037
Single stent implanted	0.854	0.396–1.460	0.183	0.924	0.650–0.998	0.035
Stent diameter < 3.0 mm	1.238	0.474–1.981	0.252	1.195	0.623–1.711	0.142
Stent length, mm	1.009	0.874–1.673	0.470	1.016	0.883–1.910	0.236
Intraoperative GP IIb/IIIa inhibitor administration	0.910	0.671–1. 158	0.086	0.974	0.683–2.265	0.391
Transradial approach	0.782	0.445–0.974	0.006	0.873	0.516–0.983	0.035
Aspirin	0.464	0.208–1.035	0.061	0.601	0.283–1.274	0.184
Clopidogrel	0.181	0.052–0.630	0.007	0.221	0.069–0.707	0.011

AMI: acute myocardial infarction; CI: confidence interval; FPG: fasting plasma, glucose; GP: glycoprotein; HR: hazard ratio; LDL-C: low-density lipoprotein cholesterol; LM: left main; LVEF: left ventricular ejection fraction; PC: platelet count.

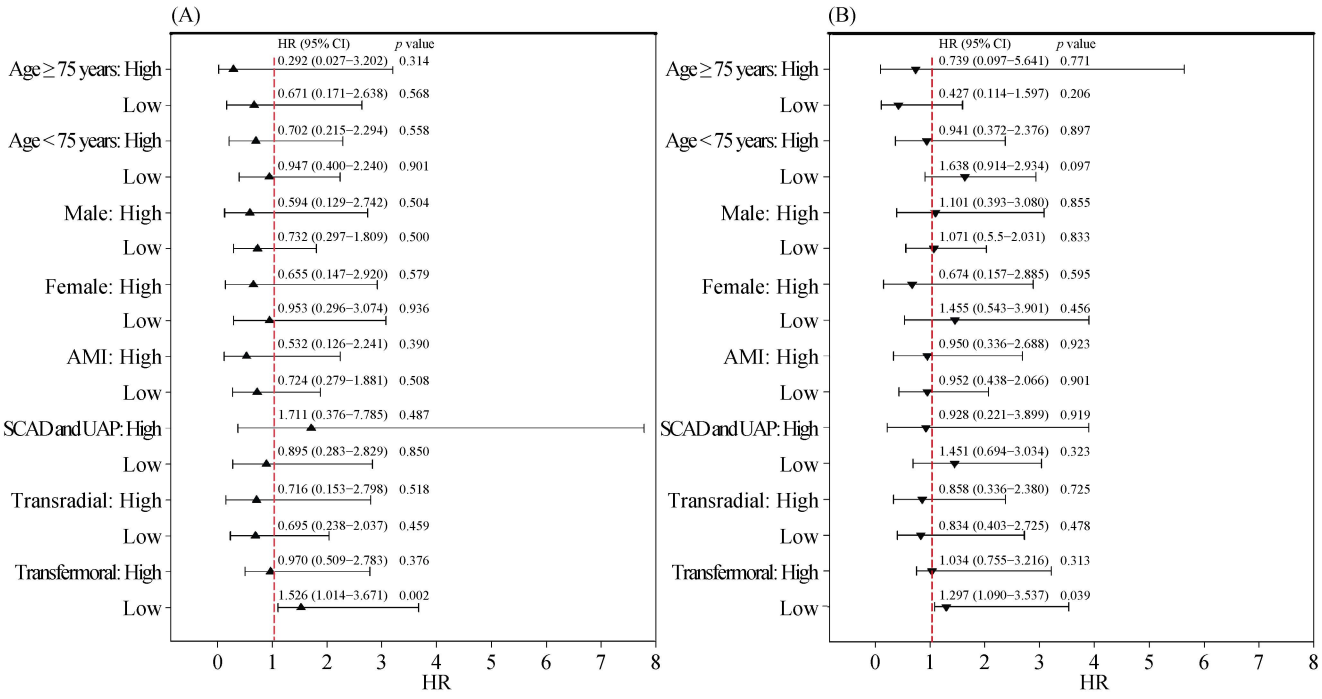

Figure 3. Subgroup analyses of the prognostic value of baseline platelet count. (A): Relationship between baseline platelet count and in-hospital mortality in subgroups; (B): relationship between baseline platelet count and follow-up mortality in subgroups. AMI: acute myocardial infarction; CI: confidence intervals; HR: hazard ratio; SCAD: stable coronary artery disease; UAP: unstable angina pectoris.

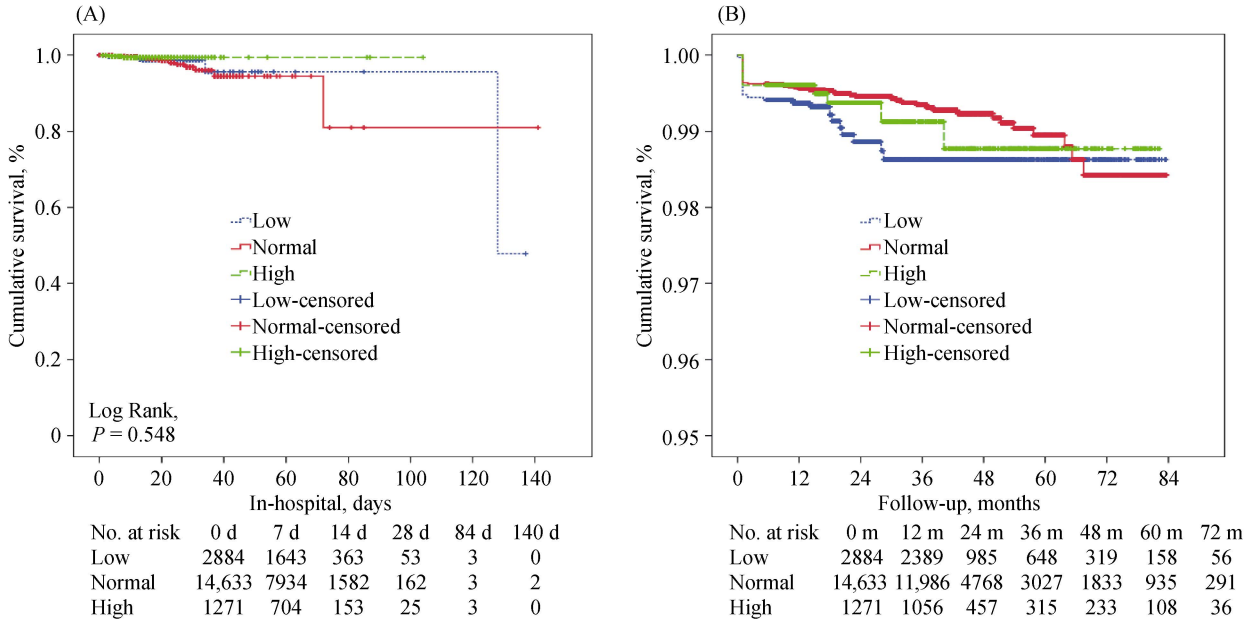

Figure 4. Kaplan–Meier curves of in-hospital and follow-up mortality. (A): Kaplan–Meier curves of in-hospital mortality; (B): Kaplan–Meier curves of follow-up mortality.

of in-hospital (Figure 4A) and follow-up mortality (Figure 4B) were presented in Figure 4. The cumulative survival rates in patients with low or high baseline PC continued to be similar to that in normal group (P = 0.548 and 0.082, respectively).

3.4.2 Secondary endpoint

There was also no significant difference in the incidence of hemorrhage among groups (major bleeding requiring a blood transfusion in the low, normal, and high group were 0.5%, 0.3%, and 0.3%, respectively; P = 0.320). After multivariable adjustment, low (HR: 1.978; 95% CI: 0.975–3.818; P = 0.052) or high baseline PC (HR: 1.264; 95% CI: 0.443–3.601; P = 0.662) did not significantly increase the risk of major bleeding (Table 6).

3.4.3 Tertiary endpoints

Although none of the tertiary endpoints were clinical adverse events, any of them could indirectly reflect the general incidence of severe adverse events. As indicated in Figure 5A, there was no significant difference in the total hospitalization expenses among groups (P = 0.342). There were statistically significant differences in the length of in-hospital stay (median value in three groups were all 7 days, P < 0.001) (Figure 5B), the length of stay before PCI (median value in three groups were all 2 days, P = 0.047) (Figure 5C), the length of stay after PCI (median value in 3 groups were all 4 days, P < 0.001) (Figure 5D), but the statistical differences did not translate into clinical importance.

Table 6. Independent predictors of major bleeding requiring a blood transfusion.

Variable	OR	95% CI	*P* Value
Age ≥ 75 yrs	0.862	0.306–2.430	0.779
Female	1.924	1.028–3.600	0.041
Hypertension	0.801	0.45 –1.423	0.449
History of stroke	1.409	0.430–4.616	0.571
AMI	2.228	1.234–4.024	0.008
Baseline PC			
Low	1.978	0.975–3.818	0.052
Normal	1.0 (Ref)	—	
High	1.264	0.443–3.601	0.662
Hematocrit, %	0.958	0.932–0.983	0.001
Creatinine, umol/L	1.006	1.002–1.009	0.001
FPG, mmol/L	1.009	0.914–1.113	0.862
LDL-C, mmol/L	0.994	0.732–1.350	0.968
LVEF, %	0.968	0.944–0.993	0.013
LM or multivessel disease	1.223	0.680–2.199	0.502
Intraoperative GP IIb/IIIa inhibitor administration	1.216	0.787–3.458	0.435
Transradial approach	0.762	0.391–0.960	0.030
Aspirin	1.669	0.826–2.570	0.558
Clopidogrel	1.125	1.029–1.532	0.005
PPI	0.893	0.455–0.996	0.062

AMI: acute myocardial infarction; CI: confidence interval; FPG: fasting plasma, glucose; GP: glycoprotein; LDL-C: low-density lipoprotein cholesterol; LM: left main; LVEF: left ventricular ejection fraction; OR: odds ratios; PC: platelet count; PPI: proton pump inhibitors.

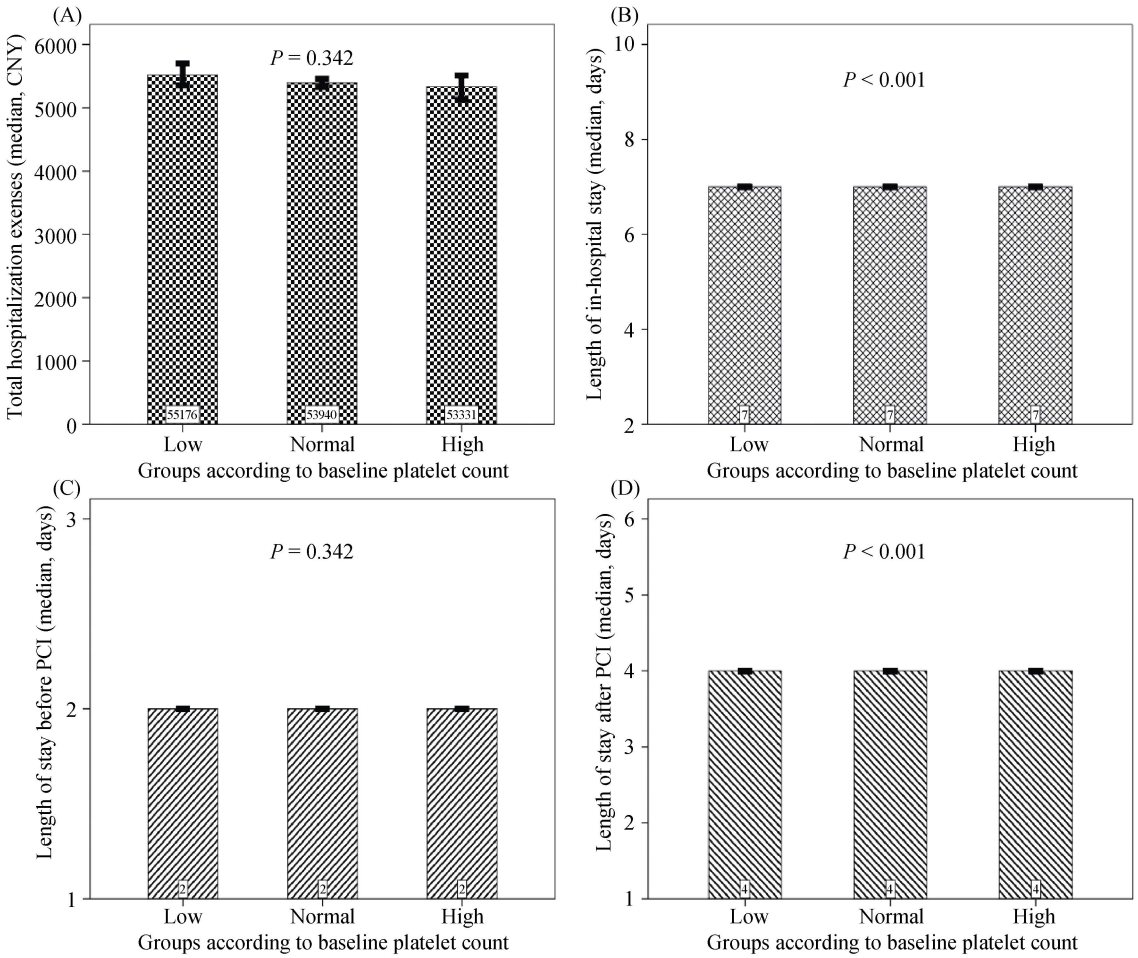

Figure 5. Comparisons of the tertiary endpoints among groups with different baseline platelet count. (A): Comparisons of the total hospitalization expenses among groups; (B): comparisons of the length of in-hospital stay among groups; (C): comparisons of the length of stay before PCI among groups; (D): comparisons of the length of stay after PCI among groups. CNY: Chinese Yuan; PCI: percutaneous coronary intervention.

4 Discussion

The PCI technique and the adjunctive pharmacotherapy have made great progress in the following several decades.[16] However, thrombosis and hemorrhage have always been the the major cause of morbidity and mortality in patients underwent PCI.[1,2] Therefore, patients with impaired quantity and quality of platelets were often excluded from prospective randomized controlled trials because of the potential increased risks of thrombosis and hemorrhage following PCI. Few pooled post-hoc analyses[7–15] and cohort studies[13–15] drew inconsistent conclusions based on data mostly from thrombolysis or BMS era.

Gibson, *et al.*[12] demonstrated that in patients with ST-elevation myocardial infarction (STEMI) who were treated with aspirin, high baseline PC was independently associated with increased rates of reinfarction at 30-day follow-up. However, clopidogrel therapy abolished this increase in the risk of reinfarction as PC increased. Iijima, *et al.*[7] argued that in patients underwent PCI after pre-treatment with 600 mg clopidogrel, high baseline PC was still independently associated with 30-day mortality. Others[9–11] agreed with that, high baseline PC was independently associated with an increased risk of adverse events following PCI. Whereas there was one more post-hoc analysis[8] revealing that low baseline PC in STEMI patients underwent PCI was strongly associated with 30-day adverse events but not with any 2-year adverse events. Similarly, a cohort study[15] showed that in-hospital death rate was higher in patients with low baseline PC due to an increased mortality in AMI patients underwent urgent but not elective PCI. In another cohort study,[14] baseline PC was not an independent predictor of 30-day mortality in AMI patients after adjustment of confounders. Interestingly, a U-shaped association between

baseline PC and long-term outcomes was also proposed.[13] Wu, *et al*.[17] demonstrated a significant association between baseline PC and clinical outcomes by meta-analysis of the above eight studies.[9–13,15–17] They confirmed a U-shaped relationship between baseline PC and the risk of mortality and adverse events. At 1-month follow-up, compared with the low PC group (< 150 × 1000/μL), the pooled relative risks of mortality and adverse events were 1.78 and 1.63 for the high PC group (> 350 × 1000/μL). At long-term follow-up, the pooled relative risks of mortality and adverse events were 1.48 and 1.28, respectively, for the high PC group.

However, the above studies have many limitations. Firstly, most of the patient population were AMI[9–12,14,16] or high-risk patients with acute coronary syndrome (ACS).[13,17] Even in the remainder one study,[15] the elective PCI accounted for less than 50%. Accordingly, conclusions drawn from the above studies could not be applicable to all CAD patients. Secondly, the sample sizes of such studies did not provide sufficient statistical power to detect low incidences of events in all prespecified groups according to clinical significance. Although equal interval classification[10,12,17] could increase statistical power, it might reduce the clinical significance of the cut-off points. Thirdly, not all patients in the above studies underwent PCI.[9,11–13,16,17] Lastly, and most important of all, some latest advances recommended by guidelines[18–20] were not reflected in the above studies, including DAPT, transradial approach for PCI, and PPI, etc.

Different from previous studies, we found that there were no significant differences among patients with different baseline PC in severe adverse outcomes, including in-hospital mortality, long-term follow-up mortality, and major bleeding requiring a blood transfusion. Although the exact mechanism is not fully understood, several factors with well-established benefits may be involved in the changing pattern between baseline PC and outcomes, including increased use of clopidogrel added to aspirin,[21–24] transradial approach PCI,[25–27] PPI,[28,29] and optimization of stent implantation.[30,31]

5 Conclusions

There is no significant association between baseline PC and severe adverse outcomes following PCI in current real-world practice.

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