Long-term prognostic significance of admission plateletcrit values in patients with non-ST elevation myocardial infarction

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The plateletcrit has been investigated as a new predictor of cardiovascular risk. The objective of our study was to investigate the role of admission plateletcrit in predicting long-term cardiovascular mortality in patients presenting with non-ST-segment elevation myocardial infarction (NSTEMI). We enrolled 296 patients with NSTEMI (mean age 59.2 ± 11.8 years; 228 men, 68 women) in this study. The study population was divided into tertiles on the basis of admission plateletcrit values. A high plateletcrit ($n = 98$) was defined as a value in the upper third tertile (plateletcrit >0.23), and a low plateletcrit (n = 198) was defined as any value in the lower two tertiles (plateletcrit ≤ 0.23). The median follow-up time was 38 months. In multivariate analyses, a significant association was noted between high plateletcrit values and the adjusted risk of long-term mortality (odds ratio = 12.15, 95% confidence interval = 1.78–82.77; $P < 0.001$). In the Kaplan–Meier survival analysis, the long-term mortality rate was 20.4% in the high plateletcrit group versus 4.5% in the low plateletcrit group ($P < 0.001$). Long-term major advanced cardiac events (MACE), hospitalization for heart failure and reinfarction were significantly higher in patients with high plateletcrit. Admission plateletcrit is a strong and independent predictor of long-term cardiovascular mortality in patients with NSTEMI. *Blood Coagul Fibrinolysis* 27:696–701

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Introduction

Acute coronary syndrome (ACS) is a set of signs and symptoms caused by the rupture of an arterial plaque, which provokes the formation of a platelet-rich coronary thrombus. ACS remains a leading cause of morbidity and mortality. Platelets play critical roles in inflammation, thrombosis and cardiovascular physiopathology [1]. Platelet indices have received increasing attention as possible markers of inflammation and of a greater risk for adverse cardiovascular outcomes [2,3]. Although easily detected platelet products with otomatic counter such as platelets count and mean platelet volume (MPV) are used in the prediction of major adverse cardiovascular events, the plateletcrit has been investigated as a new predictor of cardiovascular risk [4,5].

The plateletcrit is a part of the routine complete blood count and is an indicator of total platelet mass (plateletcrit = platelet count × MPV/10^7). There are few studies investigating the relationship between plateletcrit (PCT) and coronary artery disease in the literature. Akpınar *et al.* [6,7] demonstrated a clinical utility of PCT in both coronary slow flow phenomenon and saphenous vein grafting disease. Recently, Murat *et al.* found that high plateletcrit values on admission are independently associated with long-term adverse outcomes in patients with ST-segment elevation myocardial infarction (STEMI) who undergo primary angioplasty [8]. In this study, we analysed the relationship between high plateletcrit values and long-term cardiovascular mortality and adverse cardiovascular outcomes in patients with non-STEMI (NSTEMI).

Materials and methods

The retrospective study population consisted of 296 patients (mean age 59.2 ± 11.8 years; 228 men, 68 women) who were admitted because of NSTEMI to the emergency department from February 2009 to December 2009. All patients presented to the emergency department with acute chest pain. Initially, 402 patients were screened, but 320 patients were found to be eligible to the study. Twenty-four patients were excluded due to loss of follow-up, and the study was completed with 296 patients. The exclusion criteria included patients with unknown admission PCT, haematologic proliferative diseases, active or chronic inflammatory or autoimmune diseases, clinical evidence of active infection, malignant neoplasia, steroid therapy or chemotherapy, and unavailable complete blood cell count (CBC).

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Patients who were lost to long-term follow-up were also excluded. The local ethics committee approved the study.

Blood samples and laboratory assay

On admission, venous blood was obtained from all patients. Platelets, haemoglobin, red blood cell distribution width (RDW), white blood cell (WBC) count, MPV and PCT were measured as part of the automated CBC using a Coulter LH 780 Hematology Analyser (Beckman Coulter Ireland Inc., Mervue, Galway, Ireland). Troponin I, creatine kinase-MB (CK-MB) and other biochemistry measurements were carried out by our biochemistry department using standard methods. Twelve-hour fasting serum levels of total cholesterol, triglyceride, low-density lipoprotein (LDL) and high-density lipoprotein cholesterol (HDL) were measured with standard enzymatic methods.

Data analysis and definitions

NSTEMI is defined by electrocardiographic prominent T-wave inversion or and/or positive biomarkers of necrosis (e.g. troponin, CK-MB) in the absence of ST segment elevation and in an appropriate clinical setting (anginal equivalent or chest discomfort). The basic drug treatment for NSTEMI includes antiplatelet agents, beta-blockers, angiotensin-converting enzyme inhibitor/angiotensin receptor blockers, and statins. The thrombolysis in myocardial infarction (TIMI) risk score was calculated from the initial clinical history, ECG and laboratory values collected on admission. The Global Registry of Acute Coronary Events (GRACE) risk score based on clinical history, ECG and laboratory values was calculated at first arrival to the coronary care unit (CCU) or the acute medical admissions unit [9].

At admission, medical history and a special questionnaire on lifestyle and risk factors were taken from each patient. Patients with diabetes mellitus were identified on admission as those with documented diabetes mellitus using either oral hypoglycemic agents or insulin treatment. Hypercholesterolemia was defined as total cholesterol at least 200 mg/dl. A positive family history for coronary artery disease (CAD) was defined as documented evidence of CAD in a parent or sibling before the age of 60 years. Hypertension was defined as the previous use of antihypertensive medications, a systolic pressure more than 140 mmHg or a diastolic pressure more than 90 mmHg for at least two separate measurements. Smoking was defined as the current regular use of cigarettes. BMI was calculated as the weight in kilograms divided by the square of the height in meters. Three-vessel disease was defined as stenosis more than 50% in three major epicardial coronary arteries. Quantitative assessment of left ventricular systolic function was performed using the modified biplane Simpson's method to calculate the left ventricular ejection fraction [10]. The admission glomerular filtration rate (GFR) was estimated with the simplified Modification of Diet in Renal Disease (MDRD) Eq. [11]. For the diagnosis of metabolic syndrome at baseline, we followed the NCEP-ATP III criteria [12]. Anaemia was defined as a baseline haemoglobin concentration of less than 13 mg/dl in men and less than 12 mg/dl in women. Patients were also evaluated according to the Killip clinical examination classification [13].

Cardiovascular mortality was defined as unexplained death, death from myocardial infarction (MI), heart failure and arrhythmia. Major advanced cardiac events (MACE) were defined as cardiovascular mortality, reinfarction or target vessel revascularization (TVR). Reinfarction was defined as an increase in the CK-MB enzyme levels of twice the upper limit of normal and ST-segment re-elevations. Hospitalization for heart failure was defined by the presence of new symptoms of dyspnoea with pulmonary venous congestion on radiograph with interstitial or alveolar oedema requiring hospitalization. Follow-up data were obtained from hospital records or by interviewing (directly or by telephone) patients, their families or their personal physicians.

Statistical evaluation

The study population was divided into tertiles on the basis of the admission plateletcrit values. A high plateletcrit ($n = 98$) was defined as a value in the upper third tertile (plateletcrit >0.23), and a low plateletcrit ($n = 198$) was defined as any value in the lower two tertiles (plateletcrit ≤ 0.23). Statistical analysis was performed using SPSS for Windows (version 17.0; SPSS Inc, Chicago, Illinois, USA). A P value less than 0.05 was considered statistically significant. Qualitative variables were expressed as percentages (%), and quantitative variables were expressed as mean value $\pm$ standard deviation (SD). The parametric values were compared between the two groups with the two-tailed Student's t-test. Categorical variables were compared with the likelihood-ratio chi-square or Fisher's exact test. A backward stepwise multivariate Cox regression analysis, which included variables with P value less than 0.1, was performed to identify independent predictors of long-term cardiovascular mortality. The cumulative survival curve for long-term cardiovascular mortality was constructed using the Kaplan–Meier method, with differences assessed using log-rank tests.

Results

The demographic and clinical characteristics of the individual groups are listed in Table 1. Diabetes mellitus, hypertension, renal failure and metabolic syndrome were more common in the high PCT group. Admission Killip class more than 1, length of hospital stay and TIMI risk score were higher in the high plateletcrit group, but male sex was higher in the low plateletcrit group. Other

Table 1 Baseline characteristics of study patients

	Low PCT group (PCT ≤ 0.23), *n*: 198	High PCT group (PCT > 0.23), *n*: 98	*P*
Age (years)	59.2 ± 11.6	59.3 ± 12.4	0.96
Male	162 (81.8)	66 (67.3)	**0.005**
BMI (kg/m^2)	28,6 ± 4.3	28.0 ± 4.6	0.31
Waist circumferences (cm)	97.7 ± 9	97.0 ± 8.7	0.52
Diabetes mellitus	48 (24.2)	39 (39.8)	**0.006**
Hypertension	112 (56.6)	72 (73.5)	**0.005**
Hyperlipidaemia	98 (49.5)	54 (55.1)	0.20
Family history for CAD	62 (31.3)	31 (31.6)	0.96
Current smoker	90 (45.5)	41 (41.8)	0.56
Alcohol	25 (12.6)	7 (7.1)	0.15
Renal failure	3 (1.5)	8 (8.2)	**0.007**
Admission anaemia	62 (31.3)	40 (40.8)	0.11
Peripheral arterial disease	6 (3)	8 (8.2)	0.08
Cerebrovascular disease	6 (3)	4 (4.1)	0.74
Previous coronary surgery	24 (12.1)	8 (8.2)	0.30
Previous coronary angioplasty	24 (12.1)	17 (17.3)	0.22
Previous myocardial infarction	58 (28.3)	29 (29.6)	0.96
Metabolic syndrome	79 (39.9)	60 (61.2)	**0.001**
LVEF	53.0 (9.3)	51.0 (10.8)	0.14
ST-segment depression >1 mm	47 (23.7)	31 (31.6)	0.25
GRACE risk score	112.7 (27.6)	120 (33.6)	0.17
TIMI risk score	3.3 (1.4)	3.7 (1.4)	**0.03**
Killip class >1	5 (2.5)	9 (9.2)	**0.02**
Treatment (PCI/ACBG)	115 (55.6)	45 (45.9)	0.12
Three-vessel disease	67 (37.6)	27 (31.8)	0.17
Time of hospital stay (days)	8.0 (4.8)	9.7 (6.6)	**0.03**

Mean values (standard deviation) and % (*n*) are reported for continuous and categorical variables, respectively. CABG, coronary artery bypass grafting; CAD, coronary artery disease; CIN, contrast-induced nephropathy; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention. Bold indicates that *P*-value is statistically significant between the study groups.

baseline characteristics were not statistically different between the two groups.

The laboratory data for the patients are listed in Table 2. Platelet counts, WBC, RDW, MPV and plateletcrit were higher in the high plateletcrit group, but haemoglobin levels were higher in low plateletcrit group. Other laboratory characteristics were not statistically different between the two groups.

Tables 3 and 4 present the in-hospital and long-term adverse outcomes. In-hospital advanced heart failure was more frequent in the high PCT group. The other in-hospital outcomes were similar between groups. The median follow-up time was 38 months. Long-term cardiovascular mortality, hospitalization for heart failure, reinfarction and MACE were significantly higher in patients with high PCT. In the Kaplan–Meier survival analysis, the long-term mortality rate was 20.4% in the high plateletcrit group versus 4.5% in the low plateletcrit group ($P < 0.001$) (Fig. 1).

We performed a univariate and multivariate analysis to determine the factors associated with long-term cardiovascular mortality (Table 5). Plateletcrit more than 0.23, age, hypertension, diabetes mellitus, left ventricular ejection fraction (LVEF), GRACE risk score, metabolic

Table 2 Laboratory findings of patients

	Low PCT group (PCT ≤ 0.23), *n*: 198	High PCT group (PCT > 0.23), *n*: 98	*P*
PCT	0,19 ± 0.3	0.26 ± 0.3	**<0.001**
Haemoglobin (g/dl)	13.3 ± 1.9	12.9 ± 2.0	**0.03**
WBC (×10^9/l)	8.5 ± 2.7	10.4 ± 3.8	**<0.001**
Platelet counts (×10^9/l)	216.3 ± 46.3	296.3 ± 68.2	**<0.001**
MPV (fL)	8.9 ± 1.1	9.2 ± 1.4	**0.03**
RDW (%)	13.7 ± 1.3	14.2 ± 1.6	**0.007**
Admission blood glucose (mg/dl)	131.4 ± 61.2	145.2 ± 75.1	0.12
Admission blood creatinine (mg/dl)	1.1 ± 0.56	1.2 ± 0.50	0.12
Admission GFR (MDRD)	86.2 ± 33.1	79.7 ± 38.7	0.14
Total cholesterol (mg/dl)	185.7 ± 48.7	194.6 ± 47.9	0.14
Triglyceride (mg/dl)	183.4 ± 128.0	190 ± 148	0.71
LDL cholesterol (mg/dl)	112.2 ± 38	117.7 ± 34.7	0.23
HDL cholesterol (mg/dl)	39.3 ± 10	41.8 ± 10.4	0.47
CK-MB peak (U/l)	65.3 ± 35.4	74.5 ± 39.5	0.11
Troponin I (ng/ml)	2.4 ± 1.8	2.8 ± 1.8	0.14

Mean values (standard deviation) and % (*n*) are reported for continuous and categorical variables, respectively. CK-MB, creatinine kinase-MB; GFR, glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MDRD, modification of diet in renal disease; PCT, plateletcrit; RDW, red blood cell distribution width; WBC, white blood cell. Bold indicates that *P*-value is statistically significant between the study groups.

Table 3 **In-hospital cardiac events and complications**

	Low PCT group (PCT ≤ 0.23), *n*: 198	High PCT group (PCT > 0.23), *n*: 98	*P*
In-hospital mortality	2 (1)	2 (2)	0.60
Reinfarction	4 (2)	2 (2)	1.0
Target-vessel revascularization	1 (0.5)	1 (1)	0.60
MACE	6 (3.0)	3 (3.1)	1.0
Stroke	3 (1.5)	0 (0)	0.21
Cardiopulmonary resuscitation	2 (1)	3 (3.1)	0.34
Renal failure requiring dialysis	1 (0.5)	1 (1)	0.56
Serious ventricular arrhythmia	2 (1)	3 (3.1)	0.34
Advanced heart failure	6 (3)	9 (9.2)	0.04
Inotrope use	1 (0.5)	3 (3.1)	0.11
Cardiogenic shock	0 (0)	2 (2)	0.56
New atrial fibrillation	11 (5.6)	4 (4.1)	0.17
Complete atrioventricular block requiring transient pacemaker	2 (1)	0 (0)	1.0
Gastrointestinal bleeding	0 (0)	1 (1)	0.33
Pseoanevrizma	5 (2.5)	3 (3.1)	0.49
Haematoma	6 (3.0)	4 (4.1)	0.36
Arteriovenous fistula	1 (0.5)	0 (0)	0.30
Blood transfusion	6 (3)	8 (8.2)	0.07

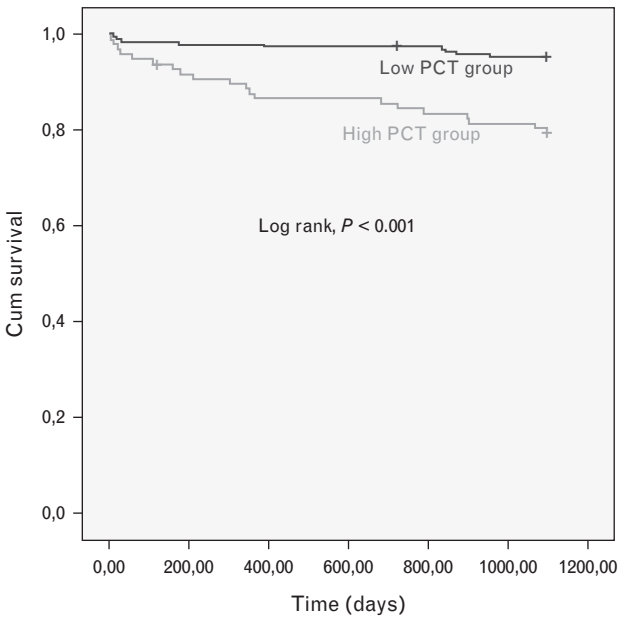
Mean values (standard deviation) and % (*n*) are reported for continuous and categorical variables, respectively. MACE, major adverse cardiac events (cardiovascular death, reinfarction and target-vessel revascularizaton); NLR, neutrophil to lymphocyte ratio.

Table 4 **Long-term cardiac events of study patients**

	Low PCT group (PCT ≤0.23), *n*: 198	High PCT group (PCT >0.23), *n*: 98	*P*
Cardiovascular mortality	9 (4.5)	20 (20.4)	**<0.001**
Hospitalization for heart failure	14 (7.1)	17 (17.3)	**0.007**
Stroke	6 (3.0)	2 (2)	1.0
Reinfarction	19 (9.6)	28 (28.6)	**<0.001**
Target-vessel revascularization	10 (5.1)	10 (10.2)	0.096
MACCE	28 (14.1)	40 (40.8)	**<0.001**

Mean values (standard deviation) and % (*n*) are reported for continuous and categorical variables, respectively. MACE, major adverse cardiac events (cardiovascular death, reinfarction and target-vessel revascularizaton). Bold indicates that *P*-value is statistically significant between the study groups.

Fig. 1



Kaplan–Meier curves for long-term survival according to plateletcrit groups. The long-term cardiovascular mortality was 20.4% in the high PCT group versus 4.5% in the low PCT group ($P < 0.001$).

syndrome, TIMI risk score, Killip class more than 1, GFR, peripheral arterial disease, admission anaemia, treatment (PCI/ACBG), admission anaemia and troponin I were analysed using the multivariate Cox regression model. In the multivariate analyses, a significant association was noted between high plateletcrit values and the adjusted risk of long-term mortality [odds ratio = 12.15, 95% confidence interval (CI) = 1.78–82.77; $P < 0.001$].

Discussion

The primary finding of this study is that higher plateletcrit is associated with an increased risk of long-term cardiovascular mortality in patients with NSTEMI. Additional analysis revealed that higher plateletcrit value was associated with increased risk of long-term MACE, reinfarction and hospitalization for heart failure in patients with NSTEMI.

The clinical significance of easy measurement and the evaluation of haematological parameters in patients with ACS have been shown in previous studies [14,15]. Assessment of platelet indexes such as platelet count, MPV, RDW and PCT is very easy and practical. Platelets release many mediators such as thromboxanes that may lead to increased inflammation [16]. In addition, increased platelet activation plays a significant role in the initiation and progression of atherosclerosis [17].

Table 5 The independent predictors of long-term cardiovascular mortality

	Univariate			Multivariate		
	OR	CI	*P*	OR	CI	*P*
PCT > 0.23	5.39	2.35–12.35	**<0.001**	12.15	1.78–82.77	**0.011**
Age	1.08	1.04–1.12	**<0.001**	0.97	0.88–1.08	0.63
Hypertension	4.25	1.44–12.54	**0.009**	1.78	0.22–14.35	0.59
Diabetes mellitus	3.40	1.56–7.42	**0.002**	0.66	0.14–3.19	0.60
LVEF (%)	0.90	0.87–0.94	**<0.001**	0.90	0.84–0.98	**0.012**
GRACE risk score	1.04	1.02–1.05	**<0.001**	1.03	0.98–1.07	0.23
Metabolic syndrome	4.03	1.66–9.76	**0.002**	5.36	0.95–30.14	0.06
TIMI risk score	1.65	1.25–2.18	**<0.001**	0.78	0.33–1.83	0.56
Killip class >1	16.57	5.26–52.24	**<0.001**	4.75	0.25–89.05	0.30
GFR (MDRD)	0.97	0.95–0.98	**<0.001**	0.98	0.95–1.01	0.14
Peripheral arterial disease	4.11	1.20–14.07	**0.024**	2.59	0.19–35.13	0.47
Treatment (PCI/ACBG)	4.84	1.91–12.27	**0.001**	0.45	0.91–2.23	0.33
Admission anaemia	3.03	1.39–6.63	**0.005**	0.84	0.15–4.67	0.84
Troponin I (ng/ml)	1.29	1.08–1.53	**0.004**	1.49	1.05–2.11	**0.027**

CABG, coronary artery bypass grafting; CI, confidence interval; CK-MB, creatinine kinase-MB; GFR, glomerular filtration rate; LVEF, left ventricular ejection fraction; MDRD, modification of diet in renal disease; OR, odds ratio; PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction. Bold indicates that *P*-value is statistically significant between the study groups.

Large hyperactive platelets play an important role in intracoronary thrombus formation and acute thrombotic events [18]. It has been shown that platelet size, when measured as the MPV, is a marker of platelet function and is positively associated with indicators of platelet activity. An increased MPV, an indicator of larger and more reactive platelets, has been associated with adverse cardiovascular outcomes in ACS [2,19]. The plateletcrit is part of the routine CBC and provides more comprehensive data about total platelet mass because the plateletcrit is the product of the platelet count and the MPV [5]. Plateletcrit has been assumed to indicate the number of circulating platelets in a unit volume of blood, analogous to the haematocrit for erythrocytes.

As the knowledge about plateletcrit is very limited in the literature, the plateletcrit has been investigated as a new predictor of inflammation and cardiovascular risk. Sahin *et al.* [20] demonstrated that the plateletcrit is correlated with C-reactive protein (CRP) levels in patients with chronic inflammatory diseases such as tuberculosis. Akpınar *et al.* [6] showed that the plateletcrit provides reliable data regarding the total platelet mass and may be a useful predictor of slow coronary flow (SCF) phenomenon. Akpınar *et al.* [7] found that more than 50% stenosis in at least one saphenous vein graft was more highly correlated with high plateletcrit than with low plateletcrit values. In another study, the plateletcrit was found to be a predictor of ineffective international normalized ratio levels in nonvalvular atrial fibrillation [21]. In addition to stable vascular disease, plateletcrit was also associated with prognosis in ACS. For instance, Murat *et al.* demonstrated the importance of the admission plateletcrit value in patients undergoing primary angioplasty for STEMI [8]. In this study, after adjustments for potentially confounding factors, a high plateletcrit value (>0.237) was one of the independent predictors of long-term cardiovascular mortality ($P = 0.03$). In our study, concordant with Murat *et al.* study, the independent risk for long-term cardiovascular mortality was found to be higher in the high plateletcrit group (>0.230). The cut-off values of PCT were 0.200 and 0.188 in the CSF phenomenon and saphenous vein grafting disease, respectively [6,7]. It can be hypothesized that, from the plateletcrit levels in some studies, inflammation and platelet activity are increased in the setting of ACS. Furthermore, platelet count, MPV, RDW and WBC were higher in the high plateletcrit group.

Cardiovascular risk factors such as diabetes mellitus, hypertension, renal failure and metabolic syndrome were more common in patients with high plateletcrit. In addition, the length of the hospital stay and the TIMI risk score were higher in the high plateletcrit group. Myocardial injury markers such as troponin and creatine kinase-MB and admission glucose and creatinine levels were elevated in the high plateletcrit group, which was not statistically significant. However, haemoglobin and LVEF were lower in the high plateletcrit group. In our study, as it was expected, the risk factors and parameters for adverse cardiovascular events were found to be higher in the high plateletcrit group. Moreover, plateletcrit, troponin and LVEF were demonstrated to be significant independent predictors for long-term cardiovascular mortality in this study. Furthermore, short in-hospital follow-up period compared to long term, in-hospital cardiovascular events were not statistically different between two study groups.

Study limitations

Several limitations should be considered for this study. It was a single-centre study, and the patients had different treatment modalities (CABG/PCI versus medical treatment). However, PCT was significantly associated with long-term cardiovascular mortality independent of the treatment modality. We did not assess inflammatory markers such as high-sensitive C-reactive protein, B-type natriuretic peptide, tumour necrosis factor-a and

interleukin (IL)-6, and other proinflammatory cytokines or other markers of oxidative stress. Such inflammatory biomarkers are expensive and are not immediately available in everyday practice.

Conclusion

To the best of our knowledge, our study is the first to investigate the predictive value of the PCT in patients with NSTEMI. The admission PCT is a strong and independent predictor of long-term cardiovascular mortality in patients with NSTEMI. The higher PCT levels in patients with ACS mean that increased inflammation and thrombus burden raise with elevated total platelet mass. In patients with ACS, a higher PCT may be used as a marker for more aggressive antiplatelet treatment.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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