Plasma calprotectin was associated with platelet activation and no-reflow phenomenon in acute coronary syndrome

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Abstract

Background

No-reflow occurs in 3–4% of all percutaneous coronary interventions and has a strong negative impact on clinical outcomes of acute coronary syndrome. Therefore, the discovery of a biomarker that can early predict the occurrence of no-reflow has great clinical significance. Calprotectin is found to be a biomarker of plaque instability and is identified to be a novel diagnostic and prognostic biomarker of cardiovascular diseases. But the association of plasma calprotectin with platelet activation and no-reflow phenomenon in acute coronary syndrome is not clear.

Methods

In this study performed at Yantai Yuhuangding Hospital from 2017 to 2018, a total of 176 Chinese patients with acute coronary syndrome who had undergone percutaneous coronary interventions were recruited consecutively, aged from 30 to 88 years. The coronary angiography and percutaneous coronary intervention procedures were performed and angiographic no-reflow was defined as a thrombolysis in myocardial infarction scores grade less than 3. Blood samples were collected immediately at admission for detection of plasma calprotectin and platelet–monocyte aggregates formation. Statistical analysis was performed for the variable's comparisons between groups and for the prediction value of plasma calprotectin for no-reflow.

Results

In this study, we have demonstrated that acute coronary syndrome patients with higher plasma calprotectin had an elevated level of platelet activation and a higher incidence of no-reflow. Plasma calprotectin level was independently associated with platelet activation and no-reflow in patients with acute coronary syndrome. Despite that platelet activation biomarker platelet–monocyte aggregate was associated with no-flow, only plasma calprotectin and serum low density lipoprotein cholesterol acted as independent predictors of no-reflow in patients with acute coronary syndrome.

Conclusion

Plasma calprotectin was associated with platelet activation and may act as an early prediction biomarker of no-reflow in patients with acute coronary syndrome.

Background

No-reflow occurs in 3–4% of all percutaneous coronary interventions (PCIs) and has a strong negative impact on clinical outcomes of acute coronary syndrome (ACS). Indeed, patients with no-reflow exhibit a higher prevalence of mortality, heart failure, and early postinfarction complications (1-3). Consequently, early detection and appropriate prevention strategies of no-reflow have an important impact on the outcome of ACS.

Calprotectin, a heterotetramer of proteins S100A8 and S100A9, is identified to be a novel diagnostic and prognostic biomarker of cardiovascular diseases(4). Calprotectin increases in the high-risk unstable or vulnerable atherosclerotic plaques in coronary arteries(5) and has been shown to be significantly increased in ACS patients compared with stable ischemic heart disease patients, at both intraplatelet transcript and circulating levels(6). Increasing plasma calprotectin was associated with a higher risk of a recurrent cardiovascular event and significantly increased risk of cardiovascular death or myocardial infarction in ACS patients (7, 8). In addition, circulating calprotectin is associated with thromboxane-dependent platelet activation in ACS(9).

Despite these studies, the association of plasma calprotectin with platelet activation and no-reflow phenomenon in ACS is not clear. The objective of this study was to investigate the relationship between calprotectin and platelet activation and evaluate the value of plasma calprotectin in predicting the development of no-reflow phenomenon in ACS patients.

Methods

Study population

This study was performed at Yantai Yuhuangding Hospital. A total of 176 Chinese patients with ACS who had undergone PCI were recruited consecutively from 2017 to 2018, aged from 30 to 88 years. ACS diagnosis criteria was defined according to published guidelines(10, 11). Patients with a history of chronic kidney disease, inflammatory bowel disease, active malignancy, severe infection, significant hepatic dysfunction and auto-immune diseases were excluded. The study protocol was approved by the local institutional ethics committee. All patients provided written consent and received standard treatment according to the ACS management guidelines. Permission was granted to use data for analysis.

Data Collection and Variable definitions

Demographic and clinical data were captured for all patients. The coronary angiography and PCI procedures were performed according to the current standard practice (12). The Thrombolysis In Myocardial Infarction (TIMI) scoring systems were applied to evaluate the anterograde flow in the target culprit coronary artery and angiographic no-reflow was defined as a TIMI grade less than 3(13, 14).

Other outcomes of interest included in-hospital major adverse cardiac events (MACE), including death, non-fatal myocardial infarctions□acute heart failure, chest pain, complete atrioventricular block, ventricular fibrillation and ventricular tachycardia.

Biomarker Assays

Blood samples were collected immediately at admission. Whole blood was carefully drawn via peripheral venipuncture into sterile acid-citrate-dextrose vacutainer tubes. The first 5mls of blood were discarded. The remaining blood was immediately transported at room temperature to the laboratory for detection of

platelet–monocyte aggregates (PMA) formation with whole blood flow cytometry. The plasma was frozen at -80°C for further analysis. Plasma calprotectin were measured using an enzyme-linked immunosorbent assay kit (Biolegend, USA).

Statistical analysis

The 176 enrolled patients were divided into 2 groups (group 1 with lower plasma calprotectin level and group 2 with higher plasma calprotectin level) according to the median of calprotectin detected in our study (3681ng/ml).

The continuous variable data were tested for normality distribution with Kolmogorov-Smirnov test and presented as the mean ± standard deviation (SD). The independent sample t test and the Mann-Whitney U test were used for comparison of the study groups. Categorical variables were compared using Pearson's chi-square test or Fisher's test and presented as absolute counts and percentages. To determine the association among variable biomarkers and platelet activation, the Pearson correlation analyses and multivariate linear regression analyses were performed. The predictive parameters for no-reflow were assessed using logistic regression analysis, and the statistically significant variables were included in multivariate analysis for no-reflow by using a multiple logistic regression model. Discrimination was assessed using the area under the receiver operating characteristic curve (AUROC). The AUROC analysis calculated cut-off values, sensitivity and specificity. P value of <0.05 was considered statistically significant. Data analysis was performed using SPSS version 22 (SPSS Inc., Chicago, IL, USA).

Results

ACS patients with lower plasma calprotectin level versus ACS patients with higher plasma calprotectin level

A total of 176 ACS patients was included in this study. The 176 patients were divided into 2 groups according to the median of calprotectin detected in our study (3681ng/ml). There were 83 patients (mean age 63 ± 10 and 65.1% male) in lower calprotectin group (group 1) and 93 patients (mean age 65 ± 12 and 76.1% male) in higher calprotectin group (group 2). Baseline clinical characteristics and laboratory factors are shown in Table 1. Mean age was not significantly different. There were more ST segment elevation ACS (STE-ACS) patients (58 (63.0%) vs. 15 (18.1%), p□0.001) and higher grace scores (139.03±36.73 vs. 102.23±36.27, p□0.001) in group 2 compared with group 1. With respect to coronary risk factors, there was significant higher presence of diabetes mellitus (p = 0.012) in group 2 than in group 1. However, no significant difference were found in hypertension, smoking, hypercholesterolemia, body mass index and previous coronary artery disease (CAD) history between groups (all p□0.05).

With respect to baseline laboratory variables, the serum total cholesterol (TC) and low density lipoprotein cholesterol (LDL-c) were significantly higher in group 2 than in group 1 (p = 0.037 and p=0.007 respectively). Serum glucose on admission and fasting blood glucose were also significantly higher in group 2 (p = 0.009 and p = 0.018 respectively), while there were no significant differences in high density

lipoprotein cholesterol (HDL-c), triglyceride, blood hemoglobin, platelet, mean platelet volume (MPV)、platelet distribution width (PDW) between groups (all p＜0.05). Compared with group 1, high-sensitive cardiac troponin I (hs-cTnI) on admission and B-type natriuretic peptide (BNP) on admission were found to be significantly higher in group 2 (p＜0.001). In addition, the other admission parameters comprising serum Creatine, blood urea nitrogen, C-reactive protein (CRP), D-Dimer, white blood cell count (WBC) and neutrophil count were higher in group 2 than in group 1 (p =0.001, p = 0.026, p = 0.005, p = 0.002, p = 0.003 and p =0.001 respectively). As for echocardiography variables, significantly higher left ventricular diastolic diameter (LVDd) and lower left ventricle ejection fraction (LVEF) were found in group 2 (p =0.005 and p =0.004). However, previous medication history of variable drugs were not significantly different between groups (all p＞0.05). (Table 1)

Patients in group 2 seemed to have higher in-hospital mortality (4(4.3%) vs. 1(1.2%), p=0.217) and in-hospital MACE (8(8.6%) vs. 3(3.6%), p=0.172) compared with in group 1, but the difference was not significant. The no-reflow was much more frequent in group 2 than in group 1 (21(22.8%) vs. 1(1.2%), p＜0.001). (Table 1)

ACS patients with no-reflow versus ACS patients without no-reflow

Compared with group 1, platelet activation biomarker PMA was found to be significantly higher in group 2 (p＜0.001) (Table 1). ACS patients with no-reflow had higher plasma calprotectin and PMA compared with those without no-reflow (6062.9±999.8 vs 3625.7±1526.8 ng/ml, p<0.001; 47.58±12.30 % vs 36.73±12.55%, p<0.001, respectively) as shown in figure 1 (A and B).

Determinants of plasma calprotectin and PMA in ACS patients

The analyses of correlation demonstrated that either calprotectin or PMA were positively associated with hs-cTnI on admission, BNP on admission, glucose on admission, GRACE score, LDL-c, TC, CRP, WBC and neutrophil lymphocyte ratio (N/L). Calprotectin and PMA were negatively associated with LVEF (Table 2). Figure 2 showed that calprotectin was positively correlated with PMA (r=0.439, p<0.001). Only calprotectin and hs-cTnI on admission were found to be independently associated with PMA as shown in table 3.

Determinants of no-reflow in ACS patients

The analysis of univariate logistic regression revealed that no-reflow was associated with calprotectin, PMA, diabetes mellitus history, LDL-c, N/L ratio, glucose on admission and BNP on admission (all p<0.05). The multivariate logistic regression showed that only calprotectin and LDL-c were independent predictors of no-reflow (p< 0.001 and p=0.017 respectively) (Table 4). The ROC curves of calprotectin and LDL-c for predicting no-reflow were shown in figure 3. AUROC of calprotectin and LDL-c for predicting no-reflow were 0.898 and 0.779, respectively. The cut-off value of plasma calprotectin for no-reflow was 4748.77 ng/ml with a sensitivity of 0.95 and a specificity of 0.77. The cut-off value of LDL-c for no-reflow was 3.06 mmol/l with a sensitivity of 0.77 and a specificity of 0.69.

Discussion

In this study, we have demonstrated that ACS patients with higher plasma calprotectin had an elevated level of platelet activation and a higher incidence of no-reflow. Plasma calprotectin level was independently associated with platelet activation and no-reflow in patients with ACS. Despite that platelet activation biomarker PMA was associated with no-flow, only plasma calprotectin and serum LDL-c acted as independent predictors of no-reflow in patients with ACS as shown in the present study.

In humans, no-reflow may occur in emergency PCI or elective PCI for ACS. The occurrence of no-reflow after PCI decreased the efficacy of reperfusion therapy and is associated with worse clinical outcomes(15). Following primary PCI for acute myocardial infarction (AMI), no-reflow measured by angiography remarkably increases the short-term mortality risk at 30 days(2) and long-term mortality risk at 1 year(16, 17) and at 5 years respectively(18). Therefore, the discovery of a biomarker that can early predict the occurrence of no-reflow has great clinical significance.

Calprotectin is an inflammation-associated peptide with proinflammatory properties, mainly secreted from activated neutrophils and monocytes under various conditions(4). Calprotectin is traditionally thought to be involved in the pathophysiology of various inflammatory conditions such as rheumatoid arthritis (19). However, some recent studies implied that calprotectin may be implicated in the pathogenesis of cardiovascular and cardiometabolic diseases based on low-grade chronic inflammation(7, 20).

High levels of calprotectin were found in human atherosclerotic plaques and it is correlated with the characteristics of rupture-prone lesions(5). As a result, calprotectin is supposed to be a biomarker of plaque instability(21). Calprotectin is also found to be specifically expressed in neutrophils and macrophages in infarcted myocardium(22). Blood calprotectin level are markedly higher in ACS patients than in stable CAD or healthy subjects(20, 23) and plasma levels of calprotectin were significantly elevated in patients with AMI than in patients with unstable angina pectoris(22). Moreover, levels of calprotectin were also found to be higher in acute ST segment elevation myocardial infarction (STEMI) patients who died after a median 12 months follow-up compared to the STEMI patients who survived(8). Calprotectin has been associated with increased risk of cardiovascular death or myocardial infarction after an acute coronary syndrome(7). What's more, calprotectin is found to be correlated with first and recurrent cardiovascular events in middle-aged healthy individuals(24). Similarly, our study revealed that calprotectin was positively correlated with on admission cTnI, BNP and Grace score. A negative association between calprotectin and EF was also found in our study.

Despite the important roles of calprotectin in ACS, the role of calprotectin in no-reflow phenomenon of ACS patients has never been clarified. The pathophysiology of post-PCI no-reflow is complex and it involves inflammation, vasoconstriction, higher platelet reactivity and microcirculation embolization(25). Micro-embolization in distal coronary circulation may occur after plaque rupture or erosion and subsequent thrombosis. Thrombotic material from the originally occurred thrombus may move distally and embolize smaller arteries, thus causing no-reflow. Increased calprotectin concentration was found in

aspirated coronary artery blood distal to the culprit ACS lesion associated with thrombosis and is related to local leukocyte activation. Thus, calprotectin is supposed to be a biomarker of inflammation and thrombosis in ACS(26). In the current study, we firstly demonstrated that calprotectin was an independent predictor of no-reflow in ACS patients.

The essential role of platelets for the pathogenic thrombosis development in ACS is highlighted by a large body of evidence. There are increased plasma concentrations of indicators of platelet activation in patients with ACS compared to those with stable CAD or normal population(27, 28). Platelet magnify chronic inflammation and interaction of platelet with leukocytes, endothelial cells and macrophages promotes a proinflammatory and prothrombotic setting leading to plaque instability and subsequent intracoronary thrombosis(29). Platelets may induce inflammatory reactions directly and indirectly by promoting inflammation and recruitment of inflammatory cells. Platelets adhere to endothelium of small coronary arteries get activated and release several leukocyte recruitment molecules and vasoactive molecules(30). For these reasons, platelets contribute to ACS not only by inducing the intraluminal thrombosis but also by micro-embolization in distal coronary circulation, by local thrombosis and vasoconstriction in the microcirculation and by platelet-mediated inflammatory reactions (31). Accordingly, higher platelet reactivity and activation were found to be associated with elevated prevalence of no-reflow after PCI in ACS patients (32). Correlation between plasma calprotectin and thromboxane-dependent platelet activation has been demonstrated in ACS patients(9). In the current study, we also found that plasma calprotectin was positive correlated with platelet activation PMA in ACS patients and PMA was positive correlated with no-reflow in ACS patients.

White blood cell subtypes play crucial roles in modulating the inflammation in the atherosclerotic process and N/L ratio is thought to be an independent predictor of no-reflow after primary PCI(33). Some studies have confirmed neutrophil activation and accumulation in the myocardial area affected by acute coronary occlusion(34). This accumulation is further increased after reperfusion and is another potential source of free radicals(34). Interaction between activated neutrophils and damaged endothelium may induce endothelial dysfunction and vasoconstriction(35). Inhibition of selectin adhesion molecules influencing the interaction between activated neutrophils and damaged endothelium has been shown to limit infarct size in animal models(36). In the present study, we also found that with no-reflow. Moreover, WBC and N/L ratio were positive correlated with calprotectin and PMA.

LDL-c plays a fundamental role in the pathophysiology of CAD. By now, it is well known that the property of atherosclerotic plaques may determine their thrombogenicity (37). Vulnerable plaques like lipid-rich plaques with thin caps are more likely to form thrombus than stable plaques with thick caps and poor lipid cores(38) . Erosion or rupture of a vulnerable plaque directly activate platelets and causing thrombus formation by the exposure of thrombogenic materials including collagens and lipid-rich atheromatous core comprising of oxidized LDL particles and cholesterol sulfate. It has been confirmed by intra-coronary imaging that the lipid rich and necrotic core rich culprit plaques may act as an important predictor of distal embolization and no-reflow in ACS patients(39). Compared with normocholesterolemic rabbits, hypercholesterolemic rabbits demonstrated markedly increased no-reflow(40). Patients undergoing

elective PCI with preprocedural statin therapy have a decreased incidence of periprocedural myocardial infarction compared with that in patients with no statin therapy(41). In patients with AMI, long-term use of statins improved coronary flow and reduced the incidence of no-reflow(42). In this study, we found that LDL-c was an independent predictor of no-reflow with lower sensitivity and specificity compared with calprotectin in ACS patients.

In this study, we demonstrated that ACS patients with higher plasma calprotectin had a higher incidence of no-reflow and plasma calprotectin acted as independent predictors of no-reflow in patients with ACS. The mechanism of no-reflow seems to imply many pathways and probably only a part has been clarified. Further basic researches are needed to better understand the specific mechanism of calprotectin in the development of no-reflow.

Conclusion

Plasma calprotectin was associated with platelet activation and may act as an early prediction biomarker of no-reflow in patients with acute coronary syndrome.

Abbreviations

Percutaneous coronary intervention: PCI; Acute coronary syndrome; ACS; Thrombolysis In Myocardial Infarction: TIMI; Major adverse cardiac events: MACE; Platelet–monocyte aggregates: PMA; Standard deviation: SD; Area under receiver operating characteristic curve: AUROC; ST segment elevation ACS: STE-ACS; Coronary artery disease: CAD; Total cholesterol: TC; Low density lipoprotein cholesterol: LDL-c; High density lipoprotein cholesterol: HDL-c; Mean platelet volume: MPV; Platelet distribution width: PDW; High-sensitive cardiac troponin I :Hs-cTnI; B-type natriuretic peptide: BNP; C-reactive protein: CRP; White blood cell count: WBC; Left ventricular diastolic diameter: LVDd; Left ventricle ejection fraction: LVEF; Neutrophil lymphocyte ratio: N/L; Acute myocardial infarction: AMI; ST segment elevation myocardial infarction: STEMI.

Declarations

Acknowledgements

Not applicable.

Authors’ contributions

N.S. and X.Z. designed the study; L.L., L.Z., N.S., and H.W. participated in collecting the data, enzyme-linked immunosorbent assay and flow cytometry; X.Z. and N.S. carried out total data analysis and drafted the manuscript; Y.A. guided and reviewed this manuscript. All the authors approved the final version of the article.

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Availability of data and materials

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki, and was approved by the Human Research Ethics Committee of Yantai Yuhuangding hospital. All participants provided written informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing financial interests of this article.

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Tables

Table 1. Comparison of variables between groups divided according to the median of calprotectin.

variables	calprotectin ≤3681 ng/ml N=83	calprotectin >3681 ng/ml N=93	P value
Age (y)	63±10	65±12	0.228
Male, n (%)	54(65.1)	70(76.1)	0.109
Smoking, n (%)	18(21.7)	23(25.0)	0.605
Hypertension, n (%)	36(43.4)	53(57.6)	0.060
Diabetes mellitus, n (%)	12(14.5)	28(30.4)	0.012
Hypercholesterolemia, n (%)	24(28.9)	35(37.6)	0.221
STE-ACS, n (%)	15(18.1)	58(63.0)	<0.001
Body mass index (kg/m^2)	26.4±2.4	27.3±2.6	0.862
TC (mmol/L)	4.6±1.07	4.99±1.3	0.037
LDL-c (mmol/L)	2.65±0.88	3.04±1.02	0.007
HDL-c (mmol/L)	1.12±0.25	1.13±0.24	0.911
Triglyceride (mmol/L)	1.37±0.62	1.38±0.83	0.962
Cystin-c (mg/L)	0.92±0.17	1.04±0.36	0.092
Glucose on admission (mmol/L)	8.60±5.51	11.17±5.78	0.009
Hs-cTnI on admission (pg/ml)	3020.70±10864.68	12180.81±18134.70	<0.001
BNP on admission (pg/ml)	134.29±207.14	419.02±618.32	<0.001
Serum creatine (umol/L)	69.20±15.02	86.40±44.87	0.001
Blood urea nitrogen (mmol/L)	5.54±1.65	6.67±4.27	0.026
Fasting blood glucose (mmol/L)	6.60±2.97	7.66±2.85	0.018
CRP (mg/L)	8.00±8.52	38.63±55.03	0.005
WBC (*10^9/L)	7.79±3.82	9.57±3.88	0.003
Neutrophil count (*10^9/L)	5.23±3.45	7.00±3.65	0.001
N/L ratio	3.37±2.84	5.41±3.5	<0.001
Hemoglobin (g/L)	139.83±15.94	141.4±22.28	0.596
Platelet (*10^9/L)	230.85±87.43	231.87±71.82	0.933
MPV (fl)	9.88±1.81	9.51±2.60	0.280
PDW (fl)	11.88±3.51	11.57±1.98	0.479
D-Dimer (mg/L)	0.64±0.35	0.96±0.89	0.002
LVEF (%)	60.53±6.45	57.37±7.8	0.004
LVDd (mm)	44.9±6.06	47.97±5.53	0.005
Grace score	102.23±36.27	139.03±36.73	<0.001
PMA (%)	32.43±11.05	43.2±12.53	<0.001
Number of diseased vessels	1.46±1.03	1.68±0.96	0.062
Plasma calprotectin (ng/ml)	2489.72±747.58	5233.32±1125.62	<0.001
Discharge BNP (pg/ml)	123.44±185.55	485.55±601.63	<0.001
Albumin (g/L)	39.11±3.59	37.60±3.85	0.008
Previous CAD, n (%)	29(34.9)	32(34.4)	0.941
medications previous, n (%)			
Aspirin	28(33.7)	30(32.3)	0.835
Glycoprotein IIb/IIIa antagonist	10(12.0)	12(12.9)	0.864
ACEI	21(25.3)	24(25.8)	0.939
B-blocker	23(27.7)	18(19.4)	0.190

	Statin	31(37.3)	36(38.7)	0.853
	Nitrate	20(24.1)	27(29.0)	0.460
No-reflow, n (%)		1(1.2)	21(22.8)	<0.001
In-hospital MACE, n (%)		3(3.6)	8(8.6)	0.172
In-hospital mortality, n (%)		1(1.2)	4(4.3)	0.217

Values are expressed as mean ±SD (standard deviation) or number (%).
STE-ACS, ST segment elevation ACS; TC, total cholesterol; LDL-c, low density lipoprotein cholesterol; HDL-c, high density lipoprotein cholesterol; BNP, B-type natriuretic peptide; hs-cTnI, high-sensitive cardiac troponin I; CRP, C-reactive protein; WBC, white blood cell count; N/L ratio, neutrophil lymphocyte ratio; MPV, mean platelet volume; PDW, platelet distribution width; LVEF, left ventricle ejection fraction; LVDd, left ventricular diastolic diameter; PMA, platelet–monocyte aggregates; CAD, coronary artery disease; ACEI, angiotensin-converting-enzyme inhibitor; MACE, major adverse cardiac events

Table 2 Correlations of baseline laboratory factors with calprotectin and PMA.

variables	calprotectin		PMA	
	r	P	r	p
hs-cTnI on admission	0.335	<0.001	0.367	<0.001
BNP on admission	0.297	<0.001	0.236	0.002
GRACE score	0.445	<0.001	0.385	<0.001
LVEF	-0.240	0.001	-0.205	0.006
LDL-c	0.264	<0.001	0.252	0.001
TC	0.240	0.002	0.192	0.012
CRP	0.472	<0.001	0.248	0.030
WBC	0.358	<0.001	0.377	<0.001
N/L ratio	0.322	<0.001	0.295	<0.001
Glucose on admission	0.256	0.002	0.191	0.024

Table 3 Multivariate linear regression analysis of various laboratory factors and PMA.

model		Unstandardized Coefficients		Standardized Coefficients	T	P value
		B	Standard error	β		
1	calprotectin	0.0019	0.0007	0.25	2.78	0.006
	hs-cTnI on admission	0.00018	0.00007	0.24	2.70	0.008
	BNP on admission	0.003	0.0020	0.12	1.48	0.143
	LDL-c	1.36	1.01	0.10	1.35	0.180
	WBC	0.26	0.28	0.08	0.92	0.359
	Glucose on admission	0.23	0.18	0.10	1.29	0.199

Table 4. Univariate and multivariate logistic regression analyses of multiple variables and the no-reflow.

variables	Unadjusted OR	p value	Adjusted OR	95% CI	p value
calprotectin	1.001	0.001	1.001	1.001-1.002	0.001
PMA	1.064	0.001	0.988	0.929-1.049	0.685
Diabetes millitus	5.357	0.001	3.440	0.728-16.259	0.119
LDL-c	3.042	0.001	2.760	1.202-6.340	0.017
WBC	1.142	0.007	0.986	0.793-1.227	0.902
N/L ratio	1.169	0.008	1.059	0.815-1.377	0.667
Glucose on admission	1.083	0.025	1.034	0.904-1.182	0.630
BNP on admission	1.001	0.024	1.000	0.999-1.001	0.643
Age	0.990	0.621			
Male	0.865	0.768			
aspirine	0.663	0.369			
smoking	0.956	0.934			
hs-cTnI on admission	1.000	0.076			
CRP	1.010	0.057			
D-Dimer	1.101	0.749			
MPV	0.952	0.577			

Figures

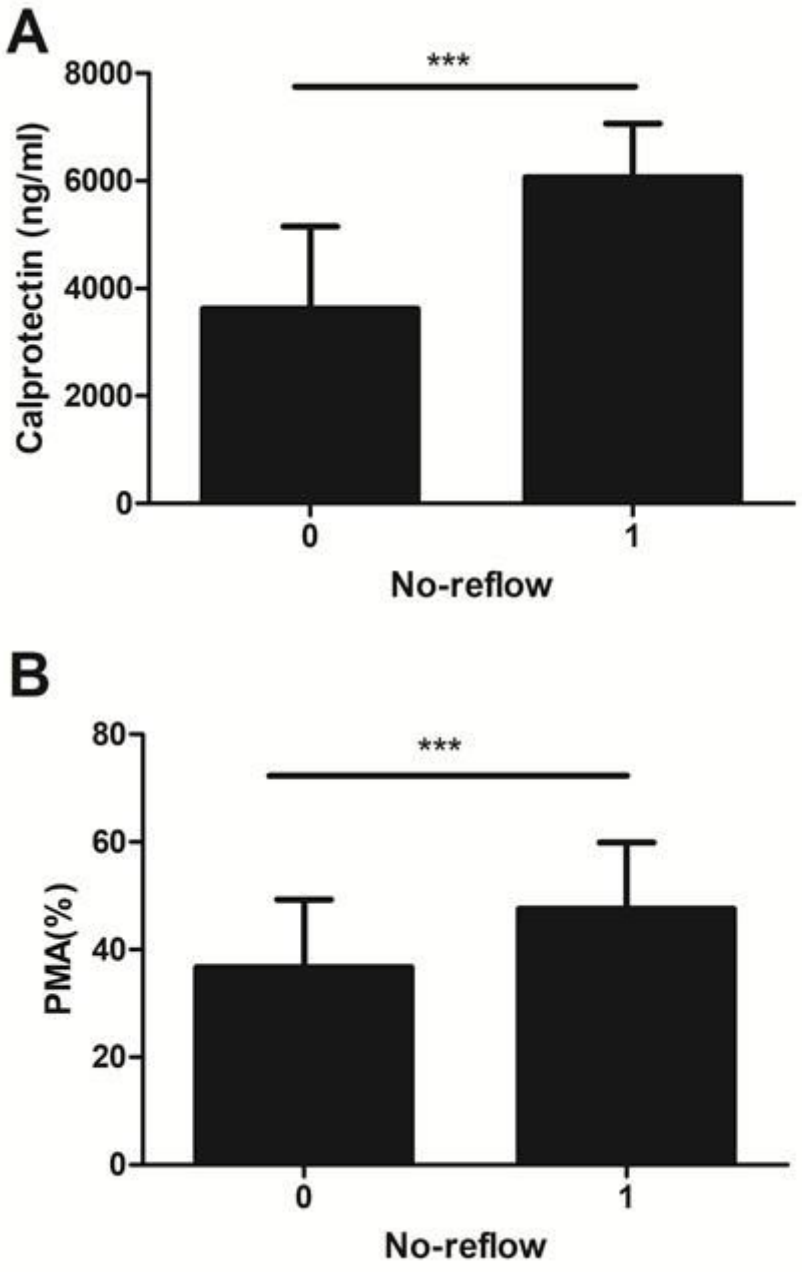

Figure 1

Comparison of calprotectin and PMA between ACS patients with and without no-reflow. (A) ACS patients with no-reflow had higher PMA (47.58±12.30 % vs 36.73±12.55%, ***p<0.001) and (B) higher plasma calprotectin (6062.9±999.8 vs 3625.7±1526.8ng/ml, ***p<0.001) compared with those without no-reflow. Data are means ± SD.

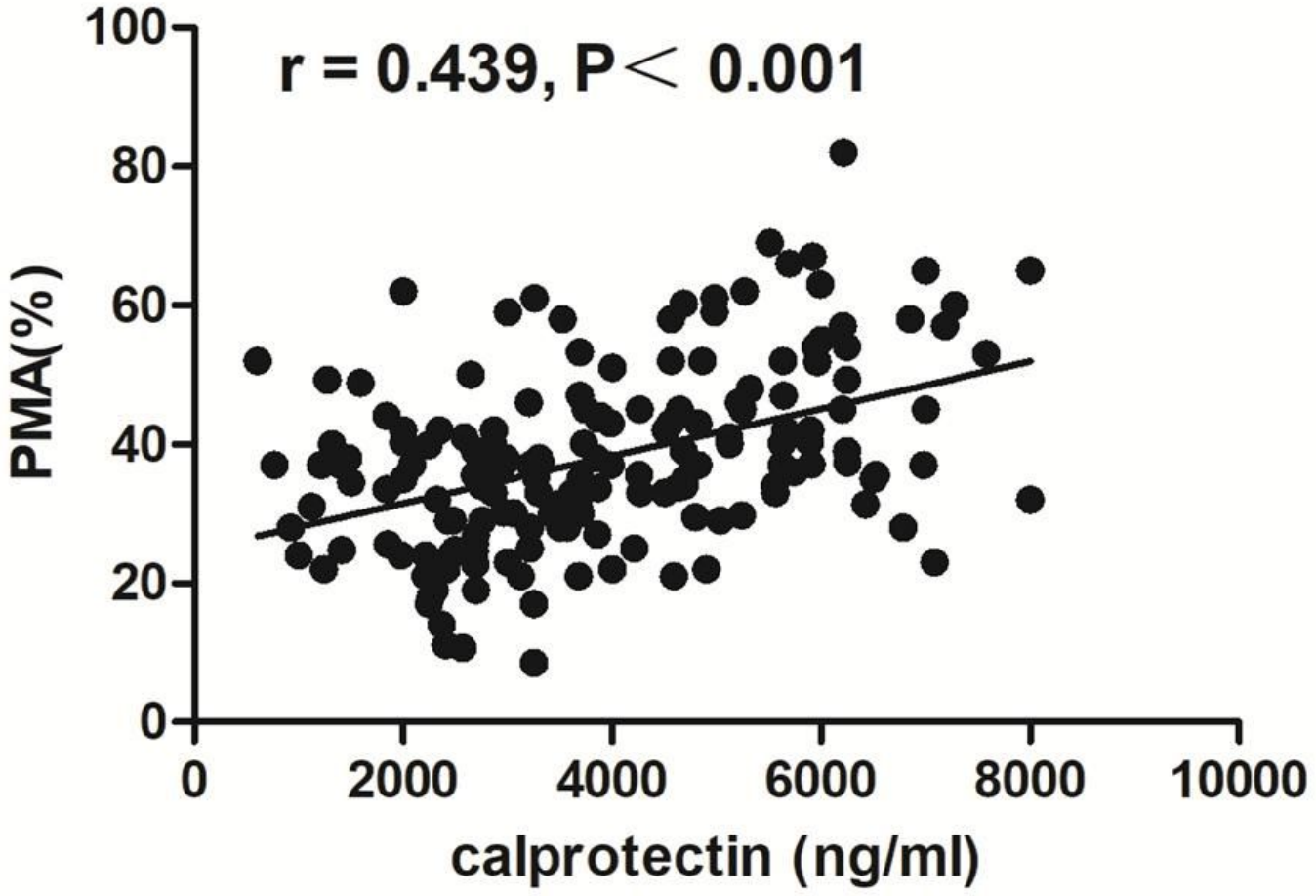

Figure 2

Correlation between plasma calprotectin and PMA in ACS patients. Plasma calprotectin was positively correlated with PMA (r=0.439, p<0.001).

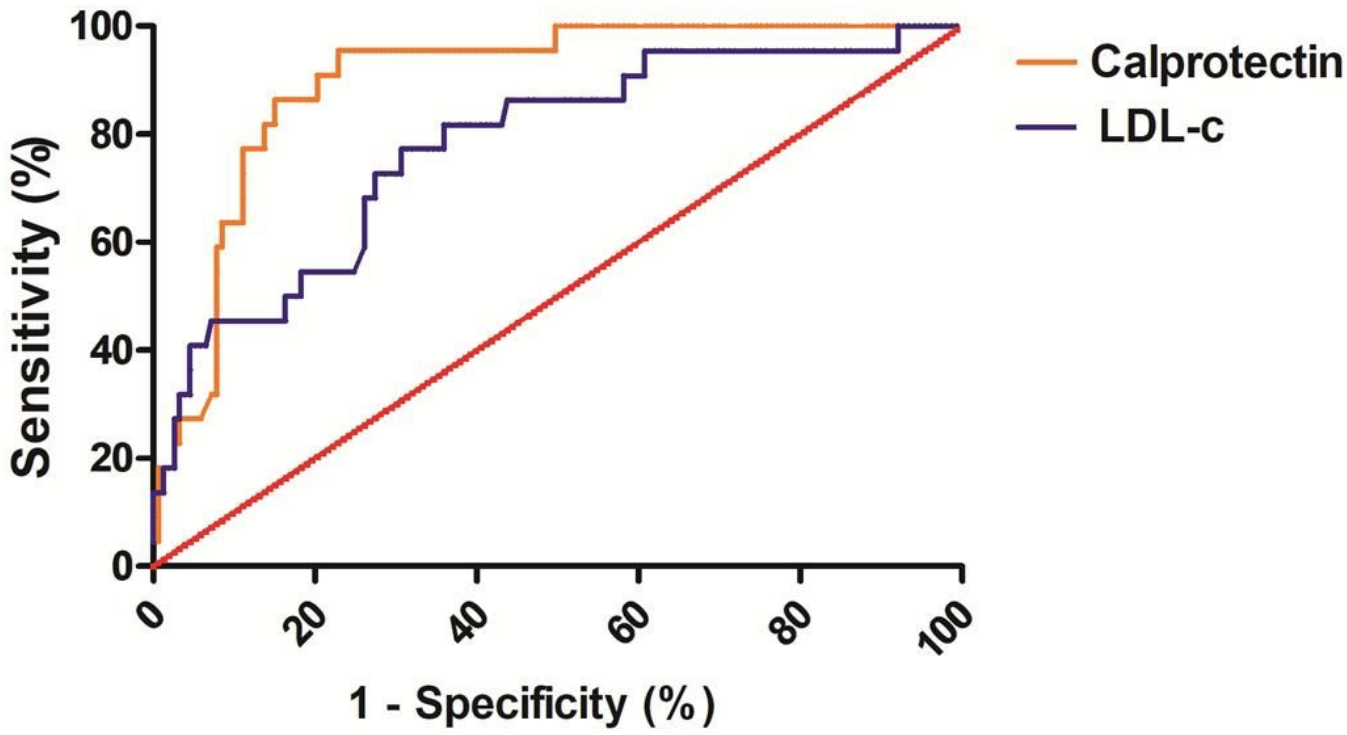

Figure 3

The ROC curves of calprotectin and LDL-c for predicting no-reflow in ACS patients. AUROC of calprotectin and LDL-c for predicting no-reflow were 0.898 and 0.779, respectively.