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ORIGINAL PAPER

The relationship between atherogenic index of plasma and no-reflow in patients with acute ST-segment elevation myocardial infarction who underwent primary percutaneous coronary intervention

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Abstract

Because the phenomenon of no reflow has a poor prognosis in ST-segment elevation myocardial infarction (STEMI) patients and the atherogenic index of plasma (AIP) has been shown to be a strong predictor of coronary heart disease, we aimed to investigate the relationship between AIP and no-reflow in patients with acute STEMI who underwent primary percutaneous coronary intervention (PCI). A total of 763 consecutive STEMI patients (648 men; mean age 58 ± 12 years) who underwent primary PCI were recruited for this study. The patients were classified into a reflow group (n = 537) and a no-reflow group (n = 226) according to the postprocedural angiographic features of thrombolysis in the myocardial infarction flow of the infarct-related artery. The AIP value was significantly higher in the no-reflow group than in the reflow group [0.50 (0.38–0.65) vs. 0.39 (0.25–0.49) p < .001], and AIP was found to be an independent predictor of no-reflow development. The best cut-off value of AIP for predicting no-reflow was 0.54, with sensitivity of 46.02 and specificity of 84,73. In addition, the predictive power of AIP was greater than that of triglycerides and high-density lipoprotein cholesterol based on a receiver operator curve comparison. The AIP was independently associated with no-reflow in patients with STEMI after primary PCI. This might be a superior indicator compared to traditional lipid profiles.

Keywords No-reflow · Atherogenic index of plasma · St-segment elevation myocardial infarction · Percutaneous coronary intervention

Introduction

Primary percutaneous coronary intervention (PCI) is the gold standard therapy for ST-segment elevation myocardial infarction (STEMI). Although the aim of primary PCI is usually to provide successful myocardial reperfusion by opening the infarct-related artery (IRA), optimal blood flow may not be achieved in some patients. This situation which is the optimal blood flow could not be achieved through primary PCI is called no-reflow [1, 2]. This no-reflow phenomenon occurs at a frequency of 5% to 50% after primary PCI [3]. No-reflow is associated with reduced myocardial salvage, increased long-term mortality, and larger infarct size. Cardiogenic shock, severe arrhythmias, acute dyspnea due to heart failure, and sustained chest pain may be seen upon clinical evaluation [4]. Therefore, early detection, preventive measures, and treatment of no-reflow are crucial in order to change the prognosis of primary PCI.

Dyslipidemia is one of the well-known risk factors for cardiovascular disease (CVD) [5]. Dyslipidemia is associated with an elevated plasma concentration of triglycerides (TGs), total cholesterol (TC), and their blood-transporting lipoproteins low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), and high-density lipoprotein cholesterol (HDL-C) [6]. There

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is strong scientific evidence indicating the association of increased TGs and decreased HDL-C with increased cardiovascular risk [7, 8]. As a relatively new parameter, the atherogenic index of plasma (AIP) is calculated using the formula log(TG/HDL-C). This demonstrates the relationship between protective and atherogenic lipoproteins and the correlation with smaller LDL cholesterol particles and an increased fractional esterification rate for cholesterol in plasma [9]. AIP has been shown to be a strong predictor of atherosclerosis and coronary artery disease (CAD) [9, 10]. Higher AIP values in acute coronary syndrome (ACS) patients have been reported and have been used to evaluate the lipid-driven inflammation state [11].

Although higher AIP values have been shown to be associated with a poor clinical outcome in ACS patients, the relationship between AIP and no-reflow is unknown. Because the prognosis of the no-reflow phenomenon after primary PCI is poor, the aim of this study is to investigate the relationship between AIP and the no-reflow phenomenon in patients with acute STEMI.

Materials and methods

Study patients

We analyzed the data of 763 consecutive patients with STEMI who underwent primary PCI between January 2016 and April 2019. Patients who were admitted within 12 h of symptom onset were included in the study. Patients with end-stage liver and renal disorders, coagulopathy, malignancies or who were intolerant to dual antiplatelet drugs were excluded from the study. Patients who were being treated with thrombolytic and conservative methods and who were receiving statin treatment were also excluded.

A STEMI diagnosis was achieved using The Joint European Society of Cardiology definition of STEMI [12]. Coronary blood flow was defined according to TIMI flow grade. Blood flow less than TIMI flow III in the absence of dissection, vasospasm, and stenosis was defined as no-reflow. The restoration of TIMI flow III after primary PCI with less than 20% residual stenosis in epicardial coronary arteries was defined as reflow.

We used the Killip classification to categorize patients with an acute myocardial infarction based upon the presence or absence of simple physical examination findings that suggest heart failure. Killip class I stands for no evidence of heart failure. Killip class II stands for findings consistent with mild to moderate heart failure. Killip class III stands for overt pulmonary edema and Killip class IV stands for cardiogenic shock.

Written informed consent was obtained from all patients at the emergency department. The protocol of this study was approved by the local ethics committee of the institute in accordance with the Declaration of Helsinki and good clinical practice.

Blood analysis and echocardiography

Venous blood samples were obtained from peripheral veins after the provision of emergency service. Whole blood count parameters were measured using a Sysmex K-1000 autoanalyzer (Block Scientific, Bohemia, NY, USA) within 5 min of blood sampling. Blood chemistry and lipid parameters were measured using a Roche Cobas 8000 C502 autoanalyzer system (Roche Diagnostics, Indianapolis, IN, USA). The estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease Study (MDRD) GFR equation. The AIP was calculated using the formula log(TG/HDL-C). All patients underwent an echocardiographic evaluation before discharge. The left ventricular ejection fraction (LVEF) was measured using Simpson's method.

Percutaneous coronary intervention

Coronary angiography was performed to all study patients via the femoral or radial route using the Judkins technique. All primary PCIs were carried out using 6F or 7F guiding catheters. Digital media were used to document coronary angiograms for quantitative analysis (Dicom viewer; MedCom GmbH, Darmstadt, Germany). Two expert interventional cardiologists who were blinded to all clinical data analyzed the digital angiograms, and in the case of disagreement the consensus decision was chosen as the compromise. All the patients were given adequate antiaggregant and anticoagulant medication after the STEMI diagnosis based on recommended guidelines [13]. The attending operator decided on stent length and whether to perform balloon predilatation or postdilatation or primary stenting. The use of medications such as intracoronary tirofiban, nitroprusside, and adenosine was also left to the discretion of the operator.

Coronary blood flow was analyzed based on TIMI flow grade [14]. No-reflow was defined as less than TIMI flow 3 (Fig. 1). Reflow was defined as TIMI flow 3 (Fig. 2). The TIMI thrombus scale was used to evaluate the clot burden [15] We also limited the TIMI thrombus scale to two scores, a high thrombus grade (TIMI thrombus grade 4 or 5) and a low thrombus grade (TIMI thrombus grade 1, 2, or 3) [16].

Statistical analysis

Statistical analysis was performed using SPSS version 17.0 (SPSS Inc, Chicago, Illinois, USA). A Kolmogorov–Smirnov test was used to assess the normal distribution of continuous variables. Continuous variables

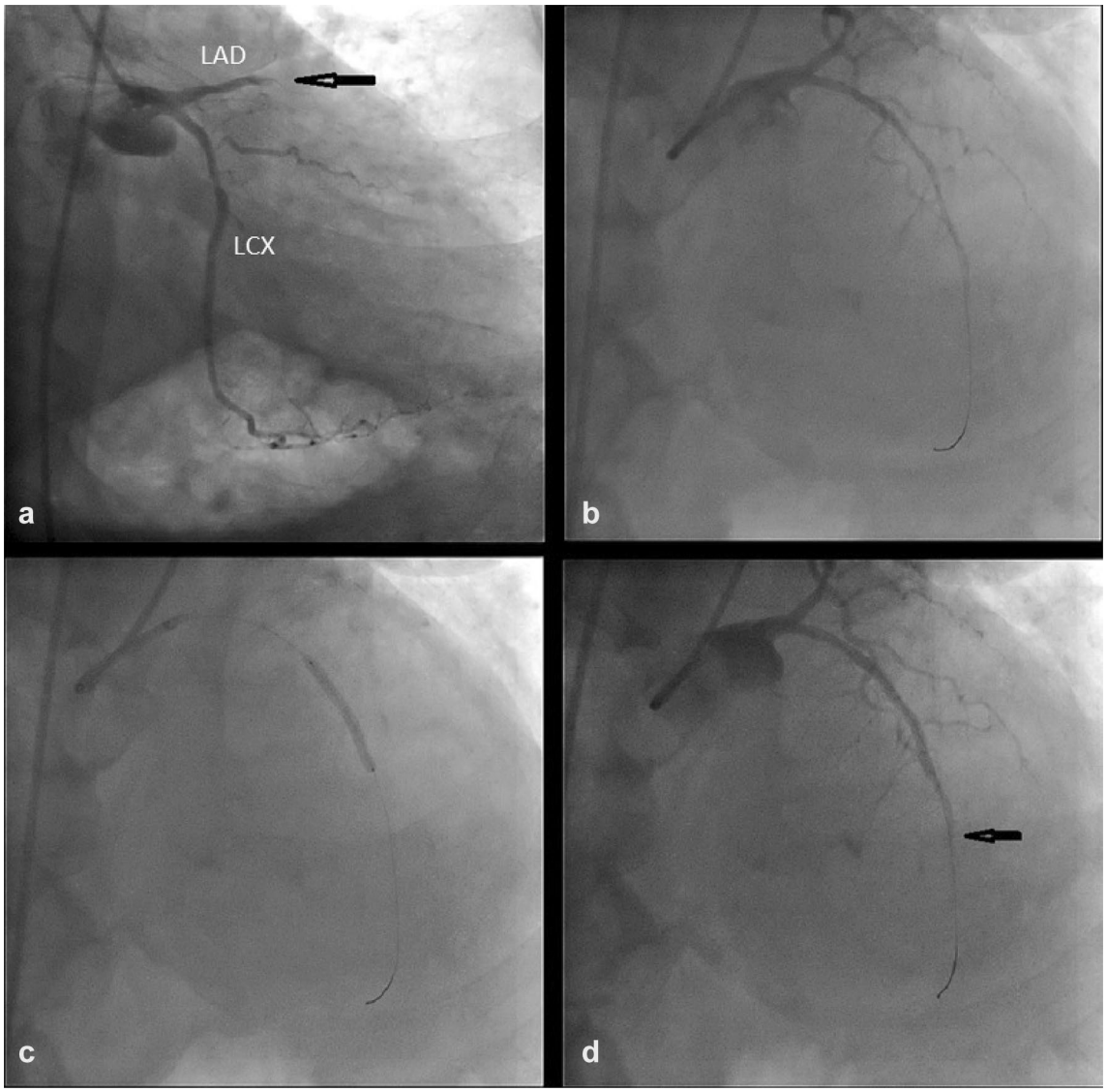


Fig. 1 72 y old male patient admitted with a diagnosis of acute anterior wall ST segment elevation myocardial infarction. His AIP value was calculated as 0.43. **a** black arrow shows thrombosis of proximal segment LAD artery. **b** Thrombotic segment was passed through with 0.014 inch guidewire. **c** 3.0×21 mm drug eluting stent was implanted to LAD. **d** Distal TIMI 1 flow (no-reflow) was obtained at LAD artery. *AIP* atherogenic index of plasma, *LAD* left anterior descending artery, *LCX* left circumflex artery, *TIMI* thrombolysis in myocardial infarction

with and without normal distribution were expressed as mean ± standard deviation and median with 25–75%, respectively. Categorical variables were expressed as percentages and compared using a Chi-square test. Correlations between continuous variables were tested using a Pearson correlation test, and noncontinuous variables were tested using a Spearman test. A Student t test and a Mann–Whitney U test were used to compare continuous and noncontinuous variables between the two groups. We investigated the effect of all variables on the no-reflow phenomenon by calculating odds ratios in a univariate analysis. All significant parameters obtained from the univariate analysis were selected for multivariate analysis, and forward conditional logistic regression analysis was used to determine the independent predictors of the no-reflow phenomenon. The odds ratio and 95% confidence interval of each independent variable were calculated. Receiver operating characteristic (ROC) curves and their corresponding area under the curve (AUC) were used to determine the accuracy and best cut-off values of AIP to predict no-reflow in STEMI patients undergoing primary PCI. A two-sided p value of < 0.05 was considered significant.

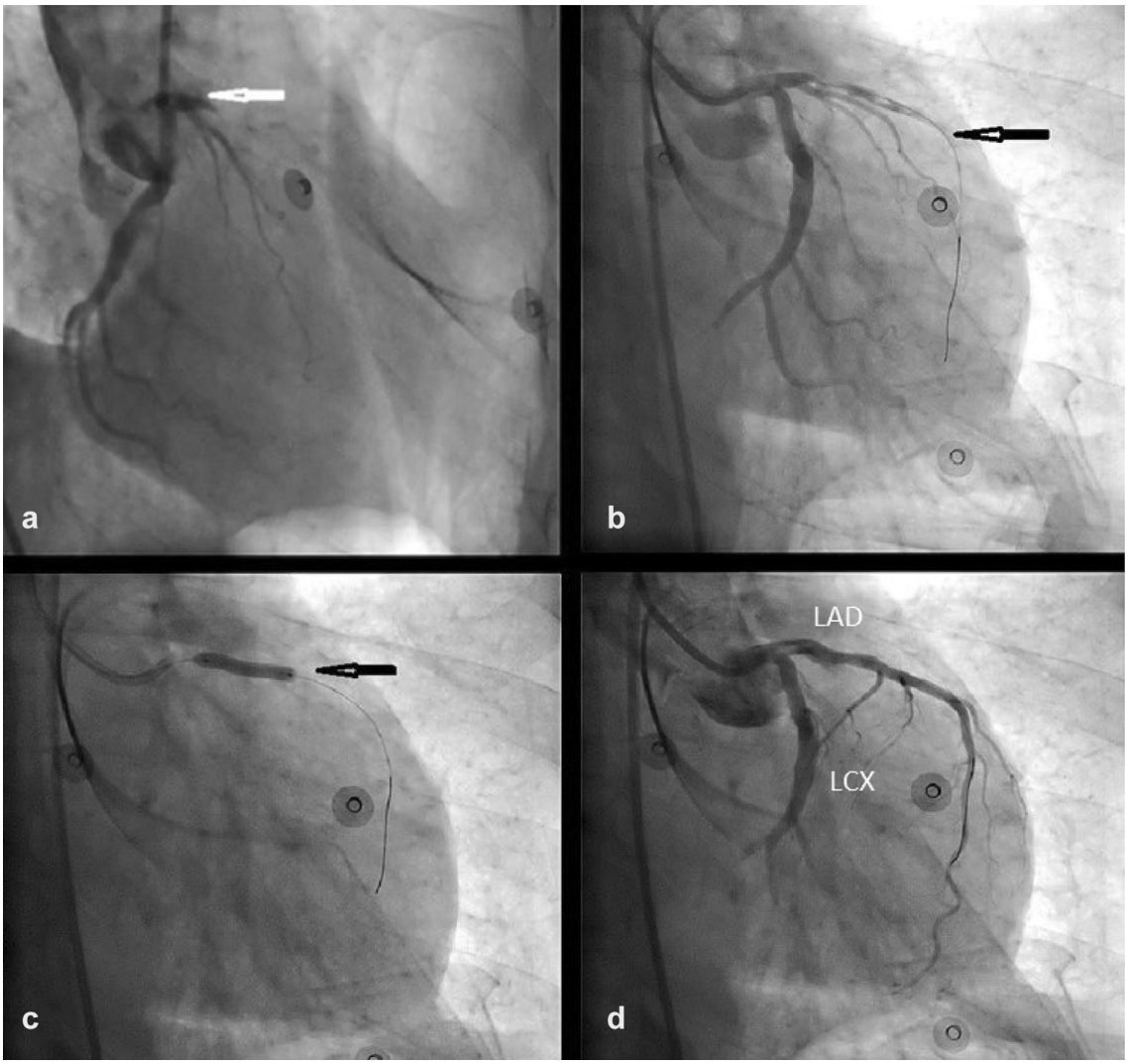


Fig. 2 67 y old male patient admitted with a diagnosis of acute anterior wall ST segment elevation myocardial infarction. His AIP value was calculated as 0.29. **a** white arrow shows thrombosis of proximal segment LAD artery. **b** Thrombotic segment was passed through with 0.014 inch guidewire. **c** 3.0×23 mm drug eluting stent was implanted to LAD. **d** Distal TIMI 3 flow was obtained at LAD artery. *AIP* atherogenic index of plasma, *LAD* left anterior descending artery, *LCX* left circumflex artery, *TIMI* thrombolysis in myocardial infarction

Results

A total of 763 patients with STEMI were analyzed in our study (648 men; mean age 58 ± 12 years). Study patients were divided into two groups based on TIMI flow grade after primary PCI. The no-reflow group comprised 226 patients (29.6%, mean age 59 ± 13 years), and the reflow group comprised 537 patients (70.4%, mean age 57 ± 12 years). According to the demographic parameters, the no-reflow group patients were older than the reflow group patients. Regarding the CAD risk factors, the frequency of diabetes mellitus (DM) and hypertension (HT) was higher in patients with no-reflow than in those with reflow. A family history of CAD and frequency of smoking were lower in patients with no-reflow. However, the number of patients with Killip class > 1 upon admission was much higher in the no-reflow group, and heart rates were also statistically higher in the no-reflow group.

The mean value of AIP was significantly higher in patients with no-reflow compared to patients with reflow [0.50 (0.38–0.65) vs. 0.39 (0.25–0.49)]. The white blood cell (WBC), fasting blood glucose, peak creatine kinase-myocardial band (CK-MB), TG, and C-reactive protein (CRP) levels were higher in patients with no-reflow than in those with reflow. Additionally, the HDL and eGFR levels were lower in patients with no-reflow than in those with reflow. Baseline demographical, hematological, and biochemical parameters are shown in Table 1.

LVEF values were significantly lower in patients with no-reflow than in those with reflow. In both groups, the

Table 1 The baseline characteristics and laboratory results of all patients and patients classified according to noreflow

	All patients (n: 763)	Patients without noreflow (n: 537)	Patients with noreflow (n: 226)	p value
Age, years	58 ± 12	57 ± 12	59 ± 13	0.019
Female gender, n(%)	115 (15.1)	79 (14.7)	36 (15.9)	0.668
Diabetes mellitus, n(%)	134 (17.6)	76 (14.2)	58 (25.7)	< 0.001
Hypertension, n(%)	286 (37.5)	183 (34.1)	103 (45.6)	0.003
Family history of CAD, n(%)	190 (24.9)	146 (27.2)	44 (19.5)	0.024
Smoking, n(%)	414 (54.3)	305 (56.8)	109 (48.2)	0.030
Heart rate, bpm	77 ± 16	77 ± 15	79 ± 19	0.040
Systolic blood pressure, mmHg	131 ± 31	132 ± 28	130 ± 39	0.734
Killip class > 1 on admission, n(%)	129 (16.9)	76 (14.2)	53 (23.5)	0.002
Haemoglobin, g/dL	13.5 ± 1.9	13.5 ± 1.7	13.5 ± 2.2	0.253
White blood cell count, 10^3/μL	12.48 ± 4.20	12.08 ± 3.84	13.47 ± 4.81	0.001
eGFR, ml/min × 1.73 m^2	87.34 ± 26.05	90.67 ± 24.60	79.45 ± 27.72	< 0.001
Fasting blood glucose, mg/dL	123 (103–155)	116 (101–146)	137 (114–189)	< 0.001
Non-HDL cholesterol, mg/dL	121 ± 26	120 ± 25	122 ± 28	0.381
LDL/HDL ratio	2.93 ± 1.11	2.89 ± 1.10	3.02 ± 1.13	0.124
Total cholesterol/HDL ratio	4.51 ± 1.29	4.40 ± 1.23	4.77 ± 1.39	0.001
Total cholesterol, mg/dL	158 ± 26	159 ± 25	157 ± 30	0.676
LDL cholesterol, mg/dL	101 ± 25	102 ± 24	99 ± 27	0.085
HDL cholesterol, mg/dL	38 ± 11	38 ± 11	35 ± 10	< 0.001
TG cholesterol, mg/dL	101 ± 36	95 ± 34	114 ± 37	< 0.001
Peak CK-MB, ng/mL	194 (109–342)	161(89–260)	327 (199–466)	< 0.001
C-Reactive protein, mg/dL	11.00 (5.60–19.00)	9.33 (5.12–17.30)	15.40 (8.10–26.80)	< 0.001
AIP	0.42 (0.29–0.53)	0.39 (0.25–0.49)	0.50 (0.38–0.65)	< 0.001

CAD coronary artery disease, *eGFR* estimated glomerular filtration rate, *LDL* low density lipoprotein, *HDL* high density lipoprotein, *TG* triglyceride, *CK-MB* creatine kinase-MB isoenzymes, *AIP* atherogenic index of plasma

most common IRA was the left anterior descending artery. The frequency of having proximal or ostial lesions of the IRA and of performing predilatation and postdilatation was higher in patients with no-reflow than in those with reflow. The patients in the no-reflow group had a higher thrombus burden and longer ischemic time than those in the reflow group. In addition, the patients in the no-reflow group had longer stent length compared to those in the reflow group. The echocardiographic and angiographic parameters of the groups are shown in Table 2.

Univariate and multivariate logistic regression analyses were used to analyze the effect of all variables and of clinically important variables on the no-reflow group (Table 3). In addition to AIP, the univariate analysis revealed that age, gender, DM, family history of CAD, smoking, Killip class > 1, heart rate, blood glucose, eGFR, CRP, LVEF, total ischemic time, proximal/ostial lesions on the IRA, baseline TIMI flow, high thrombus burden, predilatation, postdilatation, and stent length were associated with no-reflow development. The independent contributions of these variables were further analyzed using multivariate logistic regression. The AIP, eGFR, LVEF, baseline TIMI flow, and stent length remained independent predictors of no-reflow after primary PCI upon multivariate analysis. Adjusted odd ratios were 1.442 for AIP (95% CI 1.293–1.608, p < 0.001), 0.991 for eGFR (95% CI 0.983–0.999, p = 0.025), 0.921 for LVEF (95% CI 0.899–0.943, p < 0.001), 3.053 for baseline TIMI flow (95% CI 1.035–9.005, p = 0.043), and 1.052 for stent length (95% CI 1.028–1.076, p < 0.001).

An ROC curve analysis showed that the best cut-off value of AIP for predicting no-reflow was 0.54, with sensitivity and specificity of 46.02 and 84.73, respectively (AUC, 0.683; 95% CI 0.648–0.716). An ROC curve comparison analysis was performed to compare the AIP with TG and HDL-C levels. As shown in Fig. 3, the predictive power of AIP was found to be greater than that of TG and HDL-C levels (TG AUC, 0.648; 95% CI 0.613–0.682 and HDL-C AUC, 0.584; 95% CI 0.548–0.619).

Table 2 Angiographic and echocardiographic findings of all patients and patients classified according to noreflow

	All patients (n:763)	Patients without noreflow (n:537)	Patients with noreflow (n:226)	p value
LVEF,%	45.74 ± 8.43	47.72 ± 7.65	41.00 ± 8.33	< 0.001
Total ischemic time, min	185 (116–290)	172 (114–265)	215 (145–320)	< 0.001
Three vessel disease, n (%)	71 (9.3)	43 (8.0)	28 (12.4)	0.057
Infarct-related artery LAD, n (%)	399 (52.3)	277 (51.6)	122 (54.0)	0.545
Proximal/osteal lesion for IRA, n (%)	469 (61.5)	304 (56.6)	165 (73.0)	< 0.001
Baseline TIMI flow < 3, n (%)	705 (92.4)	480 (89.4)	225 (99.6)	< 0.001
High-grade thrombus burden,n (%)	543 (71.2)	359 (66.9)	184 (81.4)	< 0.001
Predilatation, n (%)	634 (83.1)	430 (80.1)	204 (90.3)	0.001
Stent length, mm	21.91 ± 8.80	20.61 ± 7.73	25.00 ± 10.30	< 0.001
Postdilatation, n (%)	215 (28.2)	138 (25.7)	77 (34.1)	0.019
In hospital death, n (%)	29 (3.8)	12 (2.2)	17 (7.5)	< 0.001

LVEF left ventricular ejection fraction, *LAD* left anterior descending artery, *IRA* infarct related artery, *TIMI* thrombolysis in miyocardial infarction

Table 3 Univariable and multivariable logistic regression analysis for the independent predictors of noreflo

	Univariate analysis		Multivariate analysis	
	p value	OR (95% CI)	p value	OR (95%CI)
AIP*	< 0.001	1.449 (1.321–1.591)	< 0.001	1.442 (1.293–1.608)
Family history of CAD	0.025	0.647 (0.443–0.947)	0.009	0.536 (0.335–0.857)
eGFR	< 0.001	0.983 (0.977–0.989)	0.019	0.991 (0.984–0.999)
LVEF	< 0.001	0.901 (0.881–0.922)	< 0.001	0.911 (0.889–0.934)
Baseline TIMI flow < 3	0.001	26.719 (3.676–194.179)	0.013	14.012 (1.749–112.287)
Stent length	< 0.001	1.057 (1.037–1.076)	< 0.001	1.442 (1.293–1.608)

AIP Atherogenic index of plasma, *CAD* coronary artery disease, *eGFR* estimated glomerular filtration rate, *LVEF* left ventricular ejection fraction, *TIMI* thrombolysis in myocardial infarction

*AIPx10 value

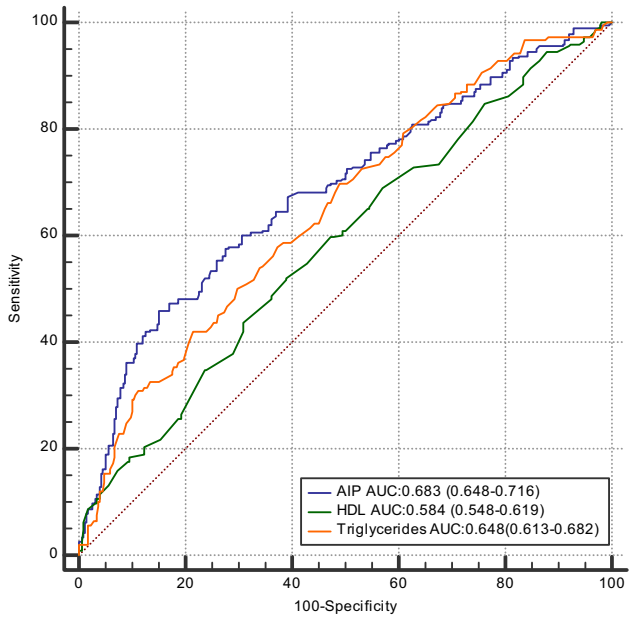


Fig. 3 ROC curve analysis for Atherogenix Index of Plasma, High Density Lipoprotein Cholesterol and Triglycerides

Discussion

To our knowledge, this study is the first to evaluate the relationship between AIP and the no-reflow phenomenon in STEMI patients who underwent primary PCI. We found that AIP is associated with no-reflow, is an independent predictor of no-reflow, and is a better predictor than TG and HDL-C levels.

The successful restoration of coronary blood flow in the IRA during STEMI does not always lead to improved myocardial perfusion. This no-reflow phenomenon occurs at a frequency of 5% to 50% (2) and is a serious prognostic sign. No-reflow can result in poor healing of the infarct and adverse left ventricular remodeling, thus increasing the risk of major adverse cardiovascular events (MACEs). To restore normal flow, factors associated with a high probability of no-reflow must be anticipated, and precautions should be taken to prevent its occurrence. Some pathophysiological mechanisms have been described as being related to no-reflow, including preexisting microvascular

dysfunction, distal thromboembolization, reperfusion injury, ischemic injury, and individual susceptibility [17]. Several previous studies have defined the relationship between the no-reflow phenomenon and various clinical, angiographical, and hematological factors [18–23]. In accordance with previous studies, in our study the frequency of DM, older age, history of HT, and a family history of CAD were found to be higher in the no-reflow group. In addition, heart rates; the Killip class; and the WBC, fasting blood glucose, CRP, and TG levels were higher. The eGFR and HDL-C levels were lower in the no-reflow group, consistent with previous studies. We also found that high AIP, low HDL-C, high TG, and high peak CK-MB levels had a significant effect on the no-reflow phenomenon.

Dyslipidemia is one well-known risk factor for CAD [5]. In particular, LDL-C has stronger impact than the other lipoproteins [24]. A study by Gentile et al. suggests a stronger association between small dense LDL-C (sdLDL) particles and early atherosclerosis compared to large LDL-C particles [25]. However, the measurement of sdLDL is limited and not easy to obtain in daily clinical practice due to its cost and complex detection method. AIP is described as the logarithmic transformation of the concentration of TG to HDL-C [log(TG/HDL-C)], and it is assumed the AIP level is inversely associated with the diameter of LDL-C particles and reflects sdLDL particle size [9]. At the ongoing studies based upon that effective correlation it has been demonstrated that AIP is strongly associated with CAD [26]. Moreover, AIP has been reported to be a powerful independent predictor of all-cause mortality in patients with ACS after coronary revascularization [27]. Similarly, elevated TG and lower HDL-C levels have been found to be independent predictors of long-term all-cause mortality and to be strongly associated with an increased risk of MACEs [28]. Based on these study findings, we hypothesized that AIP might be a predictor of no-reflow among STEMI patients. In our study, a multivariate logistic regression analysis showed that AIP was strongly associated with no-reflow after primary PCI.

The exact underlying processes between AIP and no-reflow in STEMI patients remained controversial. The inflammatory process is a well-known facilitating factor of CAD and of no-reflow [18]. Previous studies have shown that elevated CRP and lower HDL-C values are associated with no-reflow, referring to inflammation [18, 29]. Similarly, a systematic review concluded that the remnant particles formed by lipolysis of TG-rich lipoproteins show evidence for vascular wall inflammation [30]. Therefore, the association of AIP, which is a logarithmic transformation of TG to HDL-C, with no-reflow may be a consequence of that inflammatory process. No-reflow is also associated with an increased thrombus burden. The antithrombotic property of HDL-C has been previously defined [31, 32]. In our study, HDL-C levels were lower in patients with no-reflow. This may play another role in the relationship between AIP and no-reflow, given that increased AIP values are affected by low HDL-C.

Our study has some limitations. First, reperfusion success was assessed only by visual evaluation, and sensitive methods—such as coronary flow reserve, cardiac magnetic resonance imaging, the myocardial blush grade, and contrast echocardiography—were not used. Second, we did not have intravascular ultrasonography to further quantitatively evaluate the thrombus burden. Third, the study design is non-randomized and retrospective. Last, the study population was relatively small in sample size.

Conclusions

Our study investigated the relationship between AIP and the no-reflow phenomenon in relation to STEMI. We found that AIP is independently associated with the no-reflow phenomenon in STEMI patients undergoing primary PCI. We also found AIP to have better clinical value than TG and HDL levels in predicting no-reflow. Therefore, using AIP value which is feasible and easy to obtain may allow clinicians to make strategies based on risk estimation to improve the long-term outcomes of high-risk patients.

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Compliance with ethical standards

Conflict of interest The authorss declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this articles.

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