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ORIGINAL ARTICLE

The relationship between post-procedural platelet count and left ventricular aneurysm in patients with acute anterior ST-segment elevation myocardial infarction following primary percutaneous coronary intervention

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Abstract

Background: Left ventricular aneurysm (LVA) relates to worse prognosis in patients with myocardial infarction despite successful reperfusion treatment. There is no evidence that early detectable biomarkers can predict the risk for the future development of LVA.

Aim: The aim of our study was to investigate the possible predictive value of periprocedural haematological parameters for LVA.

Methods: A total of 281 patients with acute anterior ST-segment elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (pPCI) were enrolled. Haematological parameters were measured on admission before pPCI and between 8 and 12 h after pPCI, separately. The development of LVA was evaluated at one-year follow-up. The patients were then divided into two groups: an LVA group and a non-LVA group. Univariate and multivariate logistic regression analyses were performed to find the predictors of LVA.

Results: A total of 34 (12.1%) patients developed LVA at one-year follow-up after pPCI. Multivariate analyses revealed that a 10 × 10^9/L increase in platelet count 12 h after pPCI (odds ratio [OR] 1.092, 95% confidence interval [CI] 1.015–1.188, p = 0.039), peak cardiac troponin I (OR 1.107, 95% CI 1.003–1.215, p = 0.000), and left ventricular ejection fraction (OR 0.853, 95% CI 0.772–0.943, p = 0.002) were independent risk factors for LVA. For the prediction of LVA, platelet count 12 h after pPCI at a cut-off value > 197 × 10^9/L yielded a receiver operating characteristic-area under the curve (ROC-AUC) of 0.635 (82.3% sensitivity, 44.1% specificity).

Conclusions: Platelet count after pPCI was significantly associated with the development of LVA in anterior STEMI patients and may be available for early risk stratification of future LVA formation.

Key words: platelet count, left ventricular aneurysm, primary percutaneous coronary intervention, ST-segment elevation myocardial infarction

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INTRODUCTION

Left ventricular aneurysm (LVA) is a common complication of acute myocardial infarction (MI) and occurs in 10% to 38% of patients [1–3]. The occurrence of LVA is mostly due to a large MI area, resulting in damage or even loss of local myocardial contractility [4]. This pathophysiological process eventually leads to ventricular remodelling. Most of LVAs occur at the apex of the ventricle or in a few locations near the bottom of the posterior wall. Formation of LVA may damage ventricular geometry, leading to the limitation of systole and diastole ability [5], and may worsen the prognosis of MI, for instance by inducing angina [6], malignant arrhythmia [7], embolism [8], or

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even cardiac rupture [9] and death. LVA formation is divided into two periods: a period of extension of MI area (5–10 days after onset of MI) and a period of ventricular remodelling (2–4 weeks after MI). The necrotic myocardium is gradually replaced by fibrous tissue and the ventricular wall becomes thinner [6]. LVA progressively damages the function of residual surviving myocardium, so early diagnosis and appropriate treatment of ventricular aneurysm after MI are essential. Currently, the diagnostic techniques for LVA are progressing rapidly: electrocardiography, echocardiography, ventricular angiography, magnetic resonance imaging, radionuclide ventriculography, and myocardial perfusion imaging can be used for clinical diagnosis of ventricular aneurysm formation. Clinical risk factors for the development of LVA include anterior MI and poor collateral supply through smoking [10]. However, there is still no evidence of a reliable biomarker to predict the risk of future development of LVA, especially at the early phase of MI onset. Various haematological parameters can be easily and routinely available before primary percutaneous coronary intervention (pPCI) in ST-segment elevation myocardial infarction (STEMI) patients. High levels of neutrophil count have been proven to be associated with an increased risk of clinical adverse events after acute MI [11]. Therefore, we hypothesised that there might be some association between periprocedural haematological parameters and LVA formation. The purpose of our study was to investigate the possible predictive value of periprocedural haematological parameters for LVA.

METHODS

Study population

The initial study population of this retrospective cohort study was composed of 289 consecutive patients diagnosed with acute anterior STEMI, undergoing pPCI within 12 h from symptom onset, between September 2014 and March 2017, at the cardiology department of our hospital. Acute anterior STEMI was defined as typical chest pain lasting longer than 30 min, with new ST-segment elevation at the J point in at least two contiguous leads of > 2 mm (0.2 mV) in men or > 1.5 mm (0.15 mV) in women in leads V2–V3 and/or of > 1 mm (0.1 mV) in other contiguous anterior leads on admission electrocardiogram, and an increase in cardiac enzyme levels, which was defined as being above the 99th percentile cut-off point for cardiac troponin I (cTnI) or creatine kinase-MB fraction (CK-MB) [12]. Exclusion criteria included cardiogenic shock on admission, receipt of thrombolytic drugs in the previous 24 h, active infections, systemic inflammatory disease history, clinical evidence of autoimmune disease or haematological proliferative disorders, known malignancy, and liver disease. Patients with renal failure and patients who were lost to follow-up (e.g. death before the index echocardiography examination) were also excluded. A total of 281 patients were included in the present study. The study protocol was approved by the Beijing Shijitan Hospital Ethics Committee of Capital Medical University.

Definitions of clinical criteria

Left ventricular aneurysm was defined as outward bulging, with akinetic or dyskinetic motion, of the left ventricular wall during the entire cardiac cycle. Echocardiography at one-year (at 10–14 months from the onset of acute MI) follow-up was used for the diagnosis of LVA. Hypertension was defined as blood pressure > 140/90 mmHg or treatment for hypertension before admission. Hyperlipidaemia was defined as total cholesterol level > 5.72 mmol/L, low-density lipoprotein cholesterol level > 3.64 mmol/L, or treatment for hyperlipidaemia. Diabetes mellitus was defined as fasting plasma glucose level > 7.0 mmol/L, postprandial blood glucose level > 11.1 mmol/L, haemoglobin A1c level > 6.5%, or treatment for diabetes mellitus.

Coronary angiography and PCI procedure

Pharmacological treatment of all enrolled patients before pPCI included aspirin (300 mg loading dose), clopidogrel (600 mg loading dose), and an intravenous bolus of unfractionated heparin at a dose of 70 U/kg of body weight. Primary PCI was performed using the standard radial or femoral approach with a 6- or 7-French guiding catheter. The stent was deployed in all patients. The use of balloon pre-dilatation or post-dilatation, the type of stent (bare metal or drug-eluting), and the use of thrombus aspiration was left to the operator's decision. The glycoprotein IIb/IIIa receptor inhibitor tirofiban was given at the discretion of the operator and started during PCI procedure with 10 μg/kg intracoronary bolus followed by 0.15 μg/kg/min intravenous infusion. Technically successful stent implantation was defined as residual stenosis < 10% in the culprit lesion after the procedure, as visually assessed by angiography, without occlusion of a significant side branch, flow-limiting dissection, distal embolisation, or angiographic thrombus. Thrombolysis in Myocardial Infarction (TIMI) flow grades were evaluated by the consensus of two experienced interventional cardiologists, who were blind to the clinical and laboratory data, by using quantitative cardiovascular angiographic software. No-reflow after pPCI was defined as TIMI flow grade ≤ 2 after vessel recanalisation despite the absence of angiographic stenosis, spasm, dissection, or thrombosis. Normal reflow was defined as post-intervention TIMI grade 3 flow.

Laboratory analysis and echocardiography

In all patients, venous blood samples were drawn into standard EDTA-containing tubes on admission in the emergency room before pPCI and between 8 and 12 h (8–12 h) after pPCI. The haematological parameters such as neutrophil, platelet, and lymphocyte count were measured by an automated blood cell counter (XS-1000i; Sysmex Co, Kobe, Japan). The levels

Table 1. Haematological parameters of the study population

Variable	On admission before pPCI	8–12 h after pPCI	p
White blood cell count [$\times 10^9$/L]	9.76 ± 3.06	10.24 ± 3.09	0.065
Neutrophil count [$\times 10^9$/L]	7.17 ± 4.14	8.5 ± 4.62	0.008
Haemoglobin [g/L]	144.16 ± 20.72	138.86 ± 18.84	0.002
Platelet count [$\times 10^9$/L]	214.67 ± 58.58	211.02 ± 57.26	0.464
Haematocrit [%]	42.30 ± 4.84	40.86 ± 4.61	0.000
Mean platelet volume [fL]	10.33 ± 0.90	10.47 ± 1.11	0.129
Lymphocyte count [$\times 10^9$/L]	2.29 ± 1.15	1.67 ± 1.19	0.000
Monocyte count [$\times 10^9$/L]	0.54 ± 0.26	0.61 ± 0.31	0.040
Platelet distribution width [%]	12.38 ± 1.91	12.81 ± 2.16	0.017

Data are presented as mean ± standard deviation. pPCI — primary percutaneous coronary intervention

of creatinine and cardiac enzymes were also measured in all patients, with the use of standard methods. To determine the peak value of cardiac enzyme level, blood samples for troponin I (TnI) and CK-MB were obtained from a peripheral vein, after admission to the intensive coronary care unit, every 6 h during the first 48 h and every 12 h during the rest of the stay therein. Echocardiography investigation was routinely performed at 8–12 h after pPCI and at one-year (10–14 months from the onset of acute MI) follow-up, using a GE ViVidE7 ultrasound machine (GE Healthcare, Piscataway, NJ, USA) with a 3.5-MHz transducer. Left ventricular ejection fraction (LVEF) was measured by Simpson's method in the two-dimensional echocardiographic apical four-chamber view.

Statistical analysis

Continuous variables are presented as mean ± standard deviation or as median and interquartile range. The differences between groups were tested by independent samples t-test or Mann-Whitney U test. Categorical variables were summarised as percentages and compared with the χ^2 test. Multivariate logistic regression analysis, including covariates found to have a significant association with LVA in univariate analysis, was used to identify independent predictors of LVA. The receiver operating characteristic (ROC) curve was used to determine the cut-off value of platelet count to predict the LVA development. Statistical analysis was performed using the SPSS 22.0 Statistical Package Programme for Windows (SPSS Inc., Chicago, IL, USA). A two-sided p-value of < 0.05 was considered significant.

RESULTS

Our study included 281 patients (229 men; mean age, 62 ± 13 years) with acute anterior STEMI, who had undergone pPCI. Among all the subjects, 82.2% (n = 231) of patients had multi-vessel disease and 44.1% (n = 124) of patients had a proximal left anterior descending artery (LAD) lesion. Final procedural success was obtained in all patients. However, final TIMI flow grade ≤ 2 was found in 51 (18.1%) patients. In the current study, the incidence of LVA was 12.1% (n = 34) at one-year follow-up after pPCI.

Haematological parameters were acquired on admission before pPCI and 8–12 h after pPCI, separately. As shown in Table 1, neutrophil count, monocyte count, and platelet distribution width were significantly higher 8–12 h after pPCI than on admission before the procedure. However, lymphocyte count and haemoglobin and haematocrit levels detected 8–12 h after the procedure were significantly lower compared with their levels on admission (Table 2).

Study participants were divided into two groups: an LVA group and a non-LVA group, according to their echocardiographic result at one-year follow-up. Patients' baseline clinical parameters and procedural characteristics for LVA and non-LVA groups are shown in Table 2. Patients with LVA development at one-year follow-up had higher peak cTnI level (99.1 ng/mL vs. 29.6 ng/mL, p = 0.000), more frequent Killip class ≥ II (52.9% vs. 16.2%, p = 0.000), lower LVEF on admission (45.0% vs. 55.0%, p = 0.000), and a higher rate of proximal LAD lesion (79.4% vs. 50.6%, p = 0.002) than patients in the non-LVA group. The comparison of haematological parameters between the two groups on admission and 8–12 h after pPCI is presented in Table 3. White blood cell count was significantly higher in the LVA group than in the non-LVA group at both time points. Platelet count and platelet/lymphocyte ratio 8–12 h after pPCI were significantly higher in the LVA group than in the non-LVA group, but not at the time of admission before pPCI. No significant differences between the LVA and non-LVA groups at either time point were found in neutrophil count, haemoglobin level, lymphocyte count, monocyte count, platelet distribution width neutrophil/lymphocyte ratio, mean platelet volume-to-lymphocyte ratio, or lymphocyte-to-monocyte ratio (Table 3).

The results of the univariate logistic regression analyses for risk factors of the development of LVA are summarised in Table 4. Killip class ≥ II, peak cTnI, final TIMI flow grade ≤ 2,

Table 2. Baseline clinical and procedural characteristics of the study population divided according to left ventricular aneurysm (LVA) formation

Characteristics	Non-LVA (n = 247)	LVA (n = 34)	p
Age [years]	61.7 ± 13.7	64.8 ± 13.0	0.220
Male sex	202 (81.8%)	24 (70.6%)	0.164
Diabetes mellitus	71 (28.7%)	15 (44.1%)	0.074
Hypertension	149 (60.3%)	22 (64.7%)	0.709
Hyperlipidaemia	155 (62.8%)	23 (67.6%)	0.705
Active smokers	111 (44.9%)	13 (38.2%)	0.581
Killip class ≥ II	40 (16.2%)	18 (52.9%)	0.000
Peak cTnI [ng/mL]	29.6 (10.5–92.2)	99.1 (43.9–217.7)	0.000
Medication at hospital discharge:			
β-blockers	152 (61.5%)	24 (70.6%)	0.349
ACEIs or ARBs	137 (55.5%)	19 (55.8%)	0.963
Aldosterone receptor blockers	17 (6.9%)	11 (32.4%)	0.000
Statin	234 (94.7%)	30 (88.2%)	0.135
Thiazide or loop diuretic	44 (17.8%)	13 (38.2%)	0.011
Echocardiographic variables:			
Initial LVEDD [mm]	49.0 (45.0–52.0)	50.0 (48.0–56.0)	0.016
LVEDD one year later [mm]	50.0 (46.0–54.0)	60.5 (57.0–65.0)	0.000
Initial LVEF [%]	55.0 (50.0–60.0)	45.0 (40.8–49.0)	0.000
LVEF one year later [%]	57.0 (53.0–62.0)	40.0 (37.8–47.0)	0.000
Time from symptom onset to pPCI:			0.323
< 3 h	63 (25.5%)	10 (29.4%)	
3–6 h	98 (39.6%)	9 (26.5%)	
6–12 h	86 (34.9%)	15 (44.1%)	
Multi-vessel disease	204 (82.6%)	27 (79.4%)	0.253
Proximal LAD lesion	125 (50.6%)	27 (79.4%)	0.002
Stent length [mm]	30.0 (21.0–48.0)	30.5 (18.0–39.0)	0.117
Stent diameter [mm]	3.00 (2.75–3.50)	2.93 (2.50–3.19)	0.069
Stent max pressure [atm]	16.0 (12.0–8.0)	14.0 (10.0–16.0)	0.287
IABP support	15 (6.1%)	4 (11.8%)	0.263
Use of thrombus aspiration	67 (27.1%)	10 (29.4%)	0.838
Tirofiban use	151 (61.1%)	18 (52.9%)	0.358
Final TIMI flow grade ≤ 2	42 (17.0%)	9 (26.5%)	0.232

*Data are presented as mean ± standard deviation, number (percentage), or median (interquartile range). ACEIs — angiotensin-converting enzyme inhibitors; ARBs — angiotensin receptor blockers; cTnI — cardiac troponin I; IABP — intra-aortic balloon pump; LAD — left anterior descending; LVEDD — left ventricular end-diastolic diameter; LVEF — left ventricular ejection fraction; pPCI — primary percutaneous coronary intervention; TIMI — Thrombolysis In Myocardial Infarction

proximal LAD lesion, reduced LVEF, white blood cell count, platelet/lymphocyte ratio, and every 10 × 10^9/L increase in platelet count 8–12 h after pPCI were significantly associated with LVA. These variables were subsequently entered as independent variables in multivariate logistic regression analysis. In multivariate analyses, peak cTnI (odds ratio [OR] 1.107, 95% confidence interval [CI] 1.003–1.215, p = 0.000), LVEF (OR 0.853, 95% CI 0.772–0.943, p = 0.002), and every 10 × 10^9/L increase in platelet count 8–12 h after pPCI (OR 1.092, 95% CI 1.015–1.188, p = 0.039) were independent risk factors for LVA at one-year follow-up (Table 5). The most discriminative cut-off values of platelet count to predict LVA were 197 × 10^9/L, with a sensitivity of 82.3% and a specificity of 44.1% (area under curve 0.635, 95% CI 0.576–0.692; p = 0.004; as shown in Figures 1 and 2).

Table 3. Haematological parameters in left ventricular aneurysm (LVA) and non-LVA groups

Variable	No LVA (n = 247)	LVA (n = 34)	p
Haematological parameters on admission:			
White blood cell count [×10⁹/L]	9.6 ± 2.9	10.7 ± 3.8	0.046
Neutrophil count [×10⁹/L]	7.0 ± 3.3	8.2 ± 3.8	0.223
Haemoglobin [g/L]	144.6 ± 21.4	140.5 ± 13.8	0.316
Platelet count [×10⁹/L]	212.3 ± 47.4	233.6 ± 65.3	0.065
Haematocrit [%]	42.4 ± 4.9	41.3 ± 3.5	0.719
Mean platelet volume [fL]	10.3 ± 0.9	10.3 ± 1.0	0.681
Lymphocyte count [×10⁹/L]	2.26 ± 0.34	2.49 ± 0.44	0.500
Monocyte count [×10⁹/L]	0.54 ± 0.17	0.59 ± 0.21	0.301
Platelet distribution width [%]	12.4 ± 1.9	12.3 ± 2.1	0.719
Neutrophil/lymphocyte ratio	4.56 ± 2.69	5.65 ± 2.21	0.329
Platelet/lymphocyte ratio	124.6 ± 46.2	144.2 ± 48.3	0.189
Mean platelet volume-to-lymphocyte ratio	6.30 ± 3.09	6.86 ± 3.28	0.489
Lymphocyte-to-monocyte ratio	4.37 ± 1.80	5.64 ± 1.70	0.085
Haematological parameters 12 h after pPCI:			
White blood cell count [×10⁹/L]	10.1 ± 2.9	11.5 ± 3.9	0.013
Neutrophil count [×10⁹/L]	8.38 ± 3.92	9.39 ± 3.60	0.405
Haemoglobin [g/dL]	138.7 ± 20.9	136.1 ± 17.9	0.491
Platelet count [×10⁹/L]	207.0 ± 58.1	233.7 ± 56.8	0.012
Haematocrit [%]	40.98 ± 4.6	39.81 ± 4.5	0.702
Mean platelet volume [fL]	10.49 ± 1.16	10.30 ± 1.04	0.365
Lymphocyte count [×10⁹/L]	1.70 ± 0.54	1.49 ± 0.56	0.399
Monocyte count [×10⁹/L]	0.60 ± 0.21	0.65 ± 0.24	0.513
Platelet distribution width [%]	12.8 ± 2.1	12.7 ± 2.4	0.943
Neutrophil/lymphocyte ratio	6.60 ± 2.65	8.20 ± 2.46	0.122
Platelet/lymphocyte ratio	157.4 ± 59.1	195.8 ± 57.9	0.012
Mean platelet volume-to-lymphocyte ratio	8.22 ± 3.79	9.10 ± 4.45	0.325
Lymphocyte-to-monocyte ratio	2.98 ± 1.14	2.75 ± 1.17	0.195

Data are presented as mean ± standard deviation. Abbreviations — see Tables 1 and 2

DISCUSSION

The mortality rate in patients with LVA is up to six times higher than in patients without LVA [13]. In the present study, there were 34 (12.1%) acute anterior STEMI patients who developed LVA at one-year follow-up after pPCI. Multivariate analysis revealed that peak cTnI, LVEF, and platelet count 8–12 h after pPCI were significantly associated with the development of LVA. The formation of LVA was due to myocardial tissue necrosis, wall thinning, and loss of contractility of infarct area during healing processes, resulting in the akinesia (no movement) or dyskinesia (paradoxical ballooning) during ventricular systole. Various types of serious complications can occur because of LVA, particularly heart failure, ventricular arrhythmia, and thromboembolism. Early risk stratification to detect patients at high risk of developing LVA after pPCI is very important for the prevention and treatment of this condition. With the improvement of reperfusion strategies, such as PCI, the incidence of LVA has significantly decreased in recent years [14]. Mori et al. [15] found that in the pPCI era peak CK and final TIMI flow grade were significantly associated with the development of LVA in acute anterior STEMI patients undergoing pPCI. The association between elevated peak cardiac enzyme (cTnI) and LVA development was demonstrated in our study. Higher peak troponin level was revealed to be associated with larger infarct size. Shen et al. [13] found that LVA often results from a large infarct and subsequent severe global left ventricular dysfunction. The same pathophysiological relationship might also explain the significant association between initial LVEF and LVA development in our study. However, we did not identify the final TIMI flow grade ≤ 2 as an independent risk factor for LVA in one-year follow-up after acute anterior STEMI in the present

Table 4. Univariate logistic regression analysis to investigate the determinants of left ventricular aneurysm

Independent variables	OR	95% CI	p
Age	0.984	0.958–1.010	0.220
Male sex	0.963	0.455–2.310	0.730
Hypertension	0.824	0.390–1.741	0.612
Diabetes mellitus	0.504	0.242–1.047	0.066
Hyperlipidaemia	0.801	0.373–1.718	0.568
Active smokers	1.307	0.626–2.727	0.476
Killip class ≥ II	5.822	2.740–12.371	0.000
Time from symptom onset to pPCI (3-h increase)	0.915	0.576–1.452	0.706
Peak cardiac troponin I	1.008	1.005–1.012	0.000
Multi-vessel disease (vs. single-vessel disease)	0.729	0.324–1.638	0.444
Proximal LAD lesion (vs. distal LAD lesion)	3.765	1.580–8.967	0.003
Tirofiban use	0.715	0.348–1.470	0.362
Final TIMI flow grade ≤ 2	2.475	1.118–5.477	0.025
Left ventricular ejection fraction (initial)	0.869	0.826–0.915	0.000
White blood cell count on admission	1.109	1.000–1.230	0.051
White blood cell count 12 h after pPCI	1.134	1.024–1.256	0.016
Neutrophil count on admission	1.029	0.978–1.082	0.272
Neutrophil count 12 h after pPCI	1.017	0.976–1.059	0.423
Lymphocyte count on admission	1.062	0.889–1.268	0.507
Lymphocyte count 12 h after pPCI	0.659	0.369–1.175	0.157
Monocyte count on admission	1.943	0.550–6.869	0.303
Monocyte count 12 h after pPCI	1.262	0.623–2.554	0.519
Haemoglobin on admission	0.992	0.976–1.008	0.319
Haemoglobin 12 h after pPCI	0.994	0.979–1.010	0.491
Haematocrit on admission	0.013	0.101–12.713	0.252
Haematocrit 12 h after pPCI	0.105	0.100–9.962	0.165
Platelet count on admission	1.001	0.957–1.047	0.964
Platelet count 12 h after pPCI	1.079	1.016–1.147	0.014
Mean platelet volume on admission	0.912	0.591–1.409	0.679
Mean platelet volume 12 h after pPCI	0.880	0.667–1.162	0.368
Platelet distribution width on admission	0.963	0.783–1.184	0.718
Platelet distribution width 12 h after pPCI	0.994	0.841–1.175	0.943
Neutrophil/lymphocyte ratio on admission	1.025	0.973–1.078	0.352
Neutrophil/lymphocyte ratio 12 h after pPCI	1.039	0.988–1.093	0.138
Platelet/lymphocyte ratio on admission	1.003	0.998–1.008	0.191
Platelet/lymphocyte ratio 12 h after pPCI	1.005	1.001–1.008	0.017
Mean platelet volume-to-lymphocyte ratio on admission	1.031	0.946–1.124	0.489
Mean platelet volume-to-lymphocyte ratio 12 h after pPCI	1.033	0.968–1.103	0.326
Lymphocyte-to-monocyte ratio on admission	1.055	0.979–1.137	0.161
Lymphocyte-to-monocyte ratio (12 h after pPCI)	0.828	0.622–1.103	0.196

CI — confidence interval; OR — odds ratio; other abbreviations — see Tables 1 and 2

study, which is not consistent with an earlier study conducted by Mori et al. [14]. Different sample demographics and pPCI procedural characteristics may explain some of the differences between the two studies.

Various haematological parameters can be easily and routinely available before or after pPCI in STEMI patients. While there are several reports describing the predictive values of the haematological parameters for the prognosis of patients

Table 5. Multivariate logistic regression analysis to investigate the determinants of left ventricular aneurysm

Independent variables	OR	95% CI	p
Killip class ≥ II	1.856	0.433–7.957	0.405
Peak cardiac troponin I	1.107	1.003–1.215	0.000
Proximal LAD lesion	2.466	0.875–6.946	0.088
Final TIMI flow grade ≤ 2	1.986	0.694–5.683	0.201
Left ventricular ejection fraction	0.853	0.772–0.943	0.002
White blood cell count 12 h after pPCI	0.997	0.881–1.129	0.962
Platelet count 12 h after pPCI	1.092	1.015–1.188	0.039
Platelet/lymphocyte ratio 12 h after pPCI	1.001	0.996–1.006	0.613

Abbreviations — see Tables 1, 2 and 4

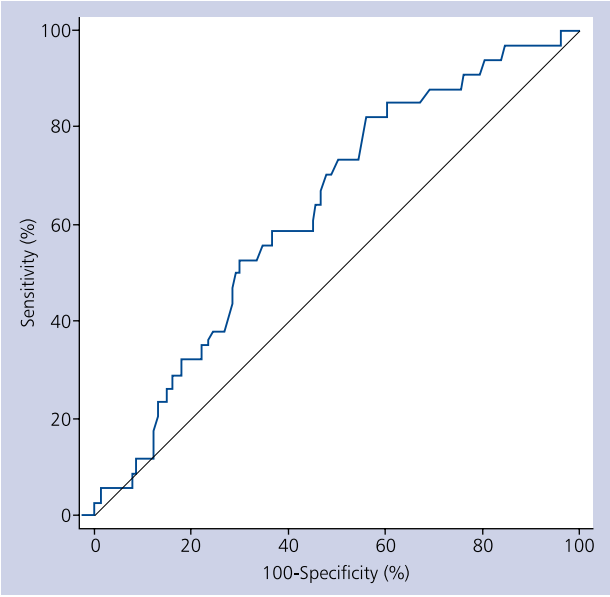

Figure 1. Receiver operating characteristic curve of platelet count 8–12 h after primary percutaneous coronary intervention for the prediction of left ventricular aneurysm formation

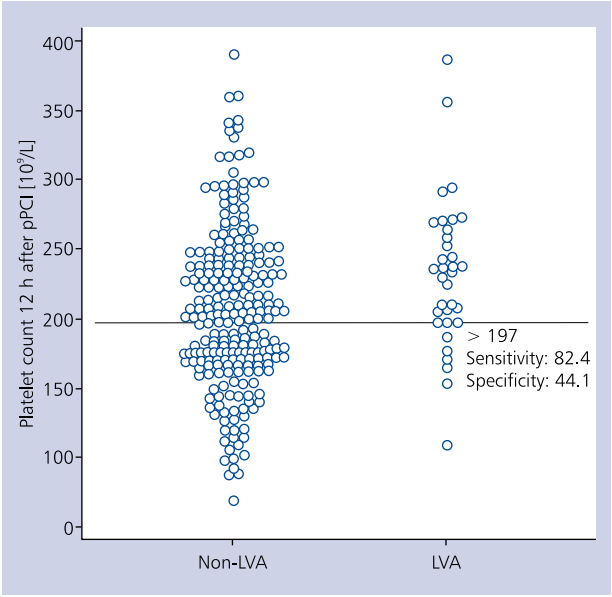

Figure 2. Dot plot distribution of platelet count 8–12 h after primary percutaneous coronary intervention (pPCI) for subjects with or without left ventricular aneurysm (LVA).

with acute MI undergoing pPCI, there are no reports showing the association between the haematological parameters and LVA development. The main purpose of the present study was to test the hypothesis that there may be some associations between periprocedural haematological parameters and future LVA formation. Although some of the complete blood count parameters, such as high neutrophil count [11], low lymphocyte count [16], mean platelet volume [17], neutrophil-to-lymphocyte ratio [18], platelet-to-lymphocyte ratio [19], and mean platelet volume-to-lymphocyte ratio [20], have been previously associated with poor cardiovascular outcomes after acute MI, we did not find any significant association between these parameters, acquired before or after pPCI, and LVA formation at one year follow-up after the procedure. After MI, inflammatory cells such as neutrophils and monocytes infiltrate the infarcted myocardium and secrete a variety of inflammatory cytokines. This healing process may weaken the myocardium and lead to infarct expansion under the ventricular stress, and finally to ventricular aneurysm formation [21]. In our study the white blood cell count was significantly higher in the LVA group than in the non-LVA group. Neutrophil counts on admission and 8–12 h after pPCI tended to be higher (although not significantly) in the LVA group than in the non-LVA group.

Mueller et al. [22] found that platelet count remained a significant independent predictor of mortality in patients with unstable angina and non-STEMI patients during long-term follow-up in a multivariate Cox regression analysis

adjusted for baseline demographic, clinical, and angiographic variables. Nikolsky et al. [23] found that high baseline platelet count in patients with acute MI is a powerful independent predictor of death and re-infarction within the first year after pPCI. The present study revealed that platelet count 8–12 h after pPCI was an independent risk factor for future LVA formation. Previous studies have found that platelets not only promote thrombus formation in the pathophysiological process of MI but may also trigger inadequate inflammatory response and subsequent ventricular remodelling [24]. Despite their participation in the circulatory inflammation response, activated platelets also bind to the wall of inflamed microvessels by attaching either directly to endothelial cells or to leucocytes that are already adherent to the vessel wall. Platelets can release markers of inflammation and directly activate other inflammatory cells, which can lead to the further release of inflammatory cytokines and amplify the inflammatory response in the microvessels [25]. The cross talk between platelets, leucocytes, and endothelial cells may impair the microvascular function in the infarct zone even in patients with successful mechanical reperfusion therapy. Yamamuro et al. [26] reported that even if successful reperfusion is achieved in epicardial coronary arteries, microvascular dysfunction, measured by coronary flow velocity, causes insufficient reperfusion of the infarcted myocardium, leading to left ventricular dysfunction and LVA formation.

The limitations of the present study lie in its single-centre, retrospective and observational design, and a small study population. Selection bias may be also considered a limitation because three patients who died before one-year follow-up were excluded from the final analysis. Inflammatory markers, such as C-reactive protein, were not routinely measured before or after pPCI; their measurement may have helped to elucidate the association between platelet count, inflammation response, and LVA development. Also, we did not collect detailed information about total periprocedural dosing of unfractionated heparin used in the study population, which may have influenced the post-procedural platelet counts. Further large prospective population-based studies, with a detailed examination of the signalling pathway and downstream targets of platelet activation, are needed to confirm the association between the platelet count and LVA in patients with STEMI receiving pPCI.

In conclusion, platelet count after pPCI was significantly associated with the development of LVA in anterior STEMI patients. Platelet count may be available for early risk stratification of future LVA formation in acute STEMI patients and might allow the optimisation of preventative therapy to improve the outcomes of patients with STEMI.

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