Kardiologia Polska 2016; 74, 10: 1160–1166; DOI: 10.5603/KP.a2016.0065

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Association of monocyte count on admission with angiographic no-reflow after primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction

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Abstract

Background: The no-reflow phenomenon during primary percutaneous coronary intervention (pPCI) in patients with ST-elevation myocardial infarction (STEMI) can lead to poor outcomes. It has been shown that the monocytes may be involved in the pathogenesis of coronary artery disease and associated with high risk of myocardial infarction.

Aim: To assess the relation between admission monocyte count and angiographic no-reflow after pPCI.

Methods: A total of 236 patients with acute STEMI, who underwent pPCI, were enrolled. The patients were divided into two groups (no-reflow and normal reflow) based on post-pPCI Thrombolysis in Myocardial Infarction (TIMI) flow grade. No reflow was defined as TIMI flow grades ≤ 2, and normal reflow was defined as TIMI 3 flow grade. The monocyte count and other laboratory parameters were measured on admission before pPCI.

Results: There were 43 (18.2%) patients in the no-reflow group and 193 (81.8%) patients in the normal-reflow group. Patients with no-reflow had significantly higher admission monocyte count ($0.76 \pm 0.48 \times 10^9$/L vs. $0.55 \pm 0.29 \times 10^9$/L, $p = 0.004$). Also, white blood cell and neutrophil counts were significantly higher while haemoglobin was significantly lower in the no-reflow group. In multivariate analysis, monocyte count remained an independent predictor of angiographic no-reflow phenomenon (odds ratio [OR] 2.665, 95% confidence interwal [CI] 1.102–6.445, $p = 0.030$) together with low haemoglobin concentration (OR 0.978, 95% CI 0.961–0.995, $p = 0.013$).

Conclusions: Monocyte count on admission and low haemoglobin concentration were independent clinical predictors of no-reflow following pPCI in patients with STEMI. Our findings suggest that admission monocyte count may be available for early risk stratification of no-reflow after pPCI and might allow the improvement of strategies to prevent this phenomenon.

Key words: monocyte count, no-reflow phenomenon, primary percutaneous coronary intervention, acute myocardial infarction

Kardiol Pol 2016; 74, 10: 1160–1166

INTRODUCTION

Early revascularisation with primary percutaneous coronary intervention (pPCI) after acute ST elevation myocardial infarction (STEMI) is associated with high success rates for Thrombolysis in Myocardial Infarction (TIMI) 3 flow attainment and improved prognosis [1]. However, impaired angiographic reflow is seen on some patients' angiographies despite opening up the epicardial coronary vessels in the setting of pPCI. This phenomenon is called no-reflow and is a predictor of poor outcomes [2]. The no-reflow phenomenon may be caused by multiple factors that eventually lead to distal microvascular obstruction and endothelial dysfunction. Recently, clinical research has focused on the predictive effects of blood cell-related biomarkers on admission and their usefulness in modifying the clinical approach of no-reflow phenomenon in patients with acute myocardial infarction (AMI). Recent investigations have suggested that monocytes may be involved in the pathogenesis of coronary artery disease [3] and high

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Received: 28.12.2015 **Accepted:** 11.04.2016 **Available as AoP:** 05.05.2016

total monocyte count is a strong predictor of high risk of myocardial infarction (MI) [4]. Previous studies have shown that interactions between monocytes and platelets probably play an important role in the pathogenesis of coronary microvascular obstruction [5]. Here, we aimed to investigate the relationship between on admission monocyte count and post-intervention no-reflow in patients receiving pPCI.

METHODS

Study population

We enrolled 236 consecutive patients with STEMI undergoing pPCI within 12 h from symptom onset, between September 2013 and May 2015 at the Cardiology Department of Beijing Shijitan Hospital. STEMI was defined as: typical chest pain > 30 min with ST elevation > 1 mm in at least two consecutive leads on the electrocardiogram or new onset left bundle branch block, and more than two-fold increase in serum cardiac markers. Exclusion criteria included cardiogenic shock on admission, active infections, systemic inflammatory disease history, known malignancy, and liver disease. The study protocol was approved by the Beijing Shijitan Hospital Ethics Committee, Capital Medical University, and written informed consent was obtained from patients.

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. Stent was deployed in all patients. The use of balloon predilatation or postdilatation, the type of stents (bare metal or drug eluting), and the use of thrombus aspiration were left to the operator's decision. The glycoprotein IIb/IIIa receptor inhibitor tirofiban was given by judgment of the operator and initiated 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. The TIMI flow grade was evaluated by consensus of two experienced interventional cardiologists who did not have knowledge of 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.

To evaluate clot burden, we performed TIMI thrombus scale in all patients [6]. In TIMI thrombus grade 0, no cine-angiographic characteristics of thrombus are present; in TIMI thrombus grade 1, possible thrombus is present with such angiographic characteristics as decreased contrast density, haziness, irregular lesion contour, or a smooth convex "meniscus" at the site of total occlusion suggestive but not diagnostic of thrombus; in TIMI thrombus grade 2, there is definite thrombus, with the largest dimensions ≤ 1/2 the vessel diameter; in TIMI thrombus grade 3, there is definite thrombus but with the largest linear dimension > 1/2 but < 2 vessel diameters; in TIMI thrombus grade 4, there is definite thrombus, with the largest dimension ≥ 2 vessel diameters; and in TIMI thrombus grade 5, there is total occlusion.

Laboratory analysis and echocardiography

In all patients, blood samples for measurements of white blood cell (WBC) count, monocyte count, and other biochemical parameters were drawn into standard EDTA-containing tubes on admission in the emergency room prior to the administration of aspirin and clopidogrel. Common blood counting parameters were measured by an automated blood cell counter (XS-1000i; Sysmex Co.). Glucose, creatinine, blood urea nitrogen, lipid profile, cardiac enzymes, and high-sensitivity C-reactive protein (hsCRP) were also measured in all patients determined by the standard methods. Echocardiography investigation was routinely performed on admission before pPCI, using a GE ViVidE7 ultrasound machine (GE Healthcare, America) with a 3.5-MHz transducer. Left ventricular ejection fraction was measured by Simpson's method in the two-dimensional echocardiographic apical four-chamber view.

Statistical analysis

Statistical analysis was performed by using the SPSS 21.0 Statistical Package Program for Windows (SPSS Inc., Chicago, IL, USA). Continuous variables are presented as mean ± standard deviation or as medians and interquartile ranges. The differences between groups were tested by independent samples t-test or Mann-Whitney U tests. Categorical variables were summarised as percentages and compared with the χ^2 test. A two-sided p value < 0.05 was considered significant. Potential independent variables associated with no-reflow in previous studies [7, 8] or significantly different between the no-reflow and normal reflow groups in our study were selected for univariate analysis. All the significant variables from the univariate analysis were entered into the multivariate logistic regression model to identify independent predictors for the development of the no-reflow phenomenon.

RESULTS

A total of 236 patients who underwent pPCI were enrolled in our analysis. Stent implantation was technically successful in all patients. The study population was divided into two groups according to the post-pPCI TIMI flow grade: normal-reflow group and no-reflow group. Normal-reflow was defined as post-PCI TIMI grade 3 flow. No-reflow was defined as post-PCI TIMI flow grade 0, 1, or 2. There were 43 (18.2%) patients in the no-reflow group (mean age 65.3 ± 12.7 years) and

Table 1. Baseline clinical characteristics of the study population

	Normal-reflow (n = 193)	No-reflow (n = 43)	P
Age [years]	61.0 ± 13.1	65.3 ± 12.7	0.055
Male sex	161 (83.4%)	31 (72.1%)	0.127
Diabetes mellitus	59 (30.6%)	16 (37.2)	0.469
Hypertension	118 (61.1%)	27 (62.8)	0.865
Hyperlipidaemia	137 (70.9%)	24 (55.8)	0.069
Active smokers	93 (48.2%)	17 (39.5)	0.317
Prior myocardial infarction	9 (4.7%)	1 (2.3%)	0.694
LVEF [%]	53.7 ± 8.7	51.9 ± 9.2	0.447
Creatinine [mg/dL]	0.90 ± 0.29	0.91 ± 0.30	0.800
hsCRP [mg/dL]	3.33 (0.16–645.00)	8.84 (0.67–139.00)	0.057

LVEF — left ventricular ejection fraction; hsCRP — high-sensitivity C-reactive protein

193 (81.8%) patients in the normal-reflow group (mean age 61.0 ± 13.1 years). The baseline clinical characteristics of study patients are listed in Table 1. There were no significant differences between the normal-reflow group and the no-reflow group in age, sex distribution, hypertension, diabetes mellitus, hyperlipidaemia, current smoking, prior MI, serum creatinine, and hsCRP level. No significant differences in the time from pain to intervention and other angiographic or procedural characteristics were observed between groups.

Common blood counting parameters are shown in Table 2. Platelet count, mean platelet volume, and lymphocyte count were not significantly different between the two groups. The platelet/lymphocyte ratio was higher in no-reflow group than that of the normal reflow group with a marginal significance (178.4 ± 115.2 vs. 150.7 ± 74.6, p = 0.050). WBC and neutrophil counts were significantly higher in the no-reflow group (p = 0.043 and p = 0.011, respectively). Haemoglobin and haematocrit levels were significantly lower in patients from the no-reflow group than in the normal-reflow group (p = 0.007 and p = 0.012, respectively). Patients with no-reflow had higher monocyte count (0.76 ± 0.48 × 10^9/L vs. 0.55 ± 0.29 × 10^9/L, p = 0.004) than patients with normal-reflow.

Univariate and multivariate logistic regression analyses of the association between the post-intervention no-reflow and multiple parameters are listed in Table 3. Age, gender, diabetes mellitus history, hsCRP, serum creatinine, WBC count, monocyte count, neutrophil count, lymphocyte count, platelet/lymphocyte ratio, and haemoglobin were analysed in a univariate analysis. On univariate logistic regression analysis, the monocyte count showed a significant association with no-reflow phenomenon (p = 0.032). The other univariate predictors of no-reflow were WBC count, neutrophil count, and haemoglobin. The identified significant predictors of no-reflow from univariate screen were included in a multivariate logistic regression analysis. In multivariate analyses, monocyte count was an independent predictor of no-reflow phenomenon (odds ratio [OR] 2.665, 95% confidence interval [CI] 1.102–6.445, p = 0.030), along with low haemoglobin concentration (OR 0.978, 95% CI 0.961–0.995, p = 0.013) (Table 4).

DISCUSSION

The goal of pPCI in STEMI is the rapid restoration of coronary blood flow to the jeopardised myocardium and to improve overall survival. However, in up to 12–39% of patients, myocardia tissue perfusion does not occur despite the presence of normal epicardial flow [9, 10]. This effect is known as the no-reflow phenomenon. In our study there were 43 (18.2%) patients who developed angiographic no-reflow. No-reflow phenomenon strongly affects the outcome of pPCI and may limit the benefits of recanalisation of the infarct-related artery. Early risk stratification in order to detect patients at high risk of no-reflow is very important for the prevention and treatment of this condition. In the current study, elevated monocyte count on admission and low haemoglobin concentration were found as independent predictors of no-reflow after pPCI in patients with STEMI.

The pathophysiology of no reflow phenomenon has not been fully understood; various mechanisms have been suggested in the development of no-reflow phenomenon. These factors include ischaemic endothelial damage, microvascular leukocytes plugging, reactive oxygen species, intravascular thrombus formation, and complex interactions between leukocytes and platelets induced by the inflammatory process. Monocytes comprise 10% of human blood leukocytes and are one of the major players of systemic inflammatory response. Monocytes can be recruited to inflamed sites and produce high levels of proinflammatory cytokines, such as tumour necrosis factor-α and interleukin-1β. In previous studies, high monocyte count was shown to be significantly associated with cardiovascular prognosis including mortality

Table 2. Angiographic and procedural characteristics of the study population according TIMI flow

	Normal-reflow (n = 193)	No-reflow (n = 43)	P
Time from symptom onset to pPCI:			0.765
< 3 h	32.6%	27.9%	
3–6 h	33.2%	32.6%	
6–12 h	34.2%	39.5%	
Pre-procedural TIMI-flow:			0.059
0	139 (72.0%)	36 (83.7%)	
1	11 (5.7%)	0 (0%)	
2	18 (9.3%)	6 (14.0%)	
3	25 (13.0%)	1 (2.3%)	
TIMI thrombus grade:			0.715
Grade 0	8 (4.1%)	1 (2.3%)	
Grade 1	22 (11.4%)	2 (4.7%)	
Grade 2	9 (4.7%)	2 (4.7%)	
Grade 3	7 (3.6%)	2 (4.7%)	
Grade 4	9 (4.7%)	1 (2.3%)	
Grade 5	138 (71.5%)	35 (81.3%)	
Anterior infarct location	102 (52.8%)	19 (37.2%)	0.317
Infarct-related coronary artery:			0.405
Left main	0 (0.0%)	0 (0.0%)	
Left anterior descending	101 (52.3%)	19 (44.2%)	
Left circumflex	19 (9.9%)	7 (16.3%)	
Right coronary artery	73 (37.8%)	17 ((39.5%)	
Stent type:			0.332
BMS	1 (0.5%)	1 (2.3%)	
DES	192 (95.5%)	42 (97.7%)	
Number of used stent*	2 (1; 2)	1 (1; 2)	0.201
Total stent length [mm]*	33.0 (23.0;48.0)	33.0 (24.0;59.5)	0.140
Stent diameter [mm]*	3.00 (2.75;3.50)	3.25 (2.78;3.50)	0.322
Use of thrombus aspiration	71 (36.8%)	22 (51.2%)	0.087
Tirofiban use	104 (53.9%)	30 (69.8%)	0.063

*Data are presented as the median value (25th, 75th percentiles); BMS — bare metal stent; DES — drug eluting stent; pPCI — primary percutaneous coronary intervention; TIMI — Thrombolysis in Myocardial Infarction

Table 3. Haematological parameters of the study population.

Variable	Normal-reflow (n = 193)	No-reflow (n = 43)	P
White blood cell count [$\times 10^9$/L]	9.52 ± 3.02	11.34 ± 3.99	0.043
Neutrophil count [$\times 10^9$/L]	6.61 ± 2.81	7.89 ± 3.65	0.011
Haemoglobin [g/dL]	14.64 ± 1.69	13.79 ± 2.49	0.007
Platelet count [$\times 10^9$/L]	211.60 ± 56.35	216.56 ± 66.93	0.615
Haematocrit [%]	43.92 ± 4.37	41.05 ± 4.54	0.012
Mean platelet volume [fL]	10.29 ± 0.91	10.51 ± 0.99	0.174
Lymphocyte count [$\times 10^9$/L]	2.26 ± 1.26	1.98 ± 1.32	0.200
Platelet/lymphocyte ratio	150.7 ± 74.6	178.4 ± 115.2	0.050
Monocyte count [$\times 10^9$/L]	0.55 ± 0.29	0.76 ± 0.48	0.004

Table 4. Independent predictors of no-flow phenomenon in patients with ST-elevation myocardial infarction in logistic regression analyses

Variable	Univariate		Multivariate	
	Odds ratio (95% confidence interval)	P	Odds ratio (95% confidence interval)	P
Age	1.025 (0.999–1.052)	0.056		
Sex	1.948 (0.905–4.193)	0.088		
Diabetes mellitus	1.346 (0.675–2.683)	0.399		
High-sensitivity C-reactive protein	1.000 (0.993–1.007)	0.993		
Creatinine	1.002 (0.989–1.014)	0.799		
White blood cell count	1.102 (1.001–1.214)	0.048	0.873 (0.652–1.169)	0.361
Neutrophil count	1.136 (1.025–1.259)	0.015	1.095 (0.976–1.228)	0.121
Haemoglobin	0.978 (0.961–0.996)	0.014	0.978 (0.961–0.995)	0.013
Lymphocyte count	0.825 (0.614–1.108)	0.201		
Platelet/lymphocyte ratio	1.004 (1.000–1.007)	0.057		
Monocyte count	2.665 (1.087–6.533)	0.032	2.665 (1.102–6.445)	0.030

in STEMI [11, 12]. Increased circulating platelet-monocyte aggregates were observed in patients with acute coronary syndromes [13, 14], which could induce expression and release of chemotactic factors, including monocyte-chemoattractant-protein-1 (MCP-1) and interleukin-8 (IL-8) from monocytes [15]. Besides activation of monocyte adhesion onto endothelial cells, MCP-1 induces the expression of tissue factor, superoxide anions, and exerts prothrombotic effects [16, 17]. Evidence of studies suggest that these factors may contribute to the development on no-reflow [18–20]. The other explanation for the relationship between monocytes and no-reflow may be related to microvascular obstruction. Extensive leucocyte plugging in the microvasculature within no-reflow areas were observed in the previous experiments [21]. In the first hours after onset of MI, monocytes dominate the neutrophil infiltration and accumulation by secreting chemokines, such as IL-8 [22, 23]. Thereafter, mechanical obstruction of the microvasculature by monocyte-induced neutrophils accumulation may contribute to the development of no-reflow. In addition, in the coronary arteries of patients undergoing pPCI for STEMI, the release of microparticles into coronary blood is accompanied by microvascular obstruction [24]. A study by Aleman et al. [25] showed that microparticles from monocytes are associated with prothrombinase activity and faster fibrin formation and might contribute to microvascular obstruction in no-reflow phenomenon.

Anaemia predicts a poorer outcome in patients with ischaemic heart disease, particularly in patients with acute coronary syndromes [26, 27]. Arbel et al. [28] found that men with STEMI in the lowest quartile of haemoglobin concentration had higher inflammatory biomarkers and inadequate bone marrow response. In our study, low haemoglobin level together with high level of monocyte count on admission in patients with no-reflow phenomenon might indicate the effect of inflammation process in no-reflow pathogenesis, as suggested by previous findings.

CONCLUSIONS

In the present study, we demonstrated that monocyte count on admission is independently associated with post-pPCI coronary no-reflow in patients with acute STEMI. Our results suggest that this cheap and easily measurable laboratory data may be available for the risk stratification of no-reflow phenomenon in patients with STEMI.

Sources of funding: *This work was supported by a grant from the Sunshine Cardiovascular Research Foundation of Chinese Medical Doctor Association (SCRFCMDA201101).*

Conflict of interest: *none declared*

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Cite this article as: Wang Z, Ren L, Liu N et al. Association of monocyte count on admission with angiographic no-reflow after primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction. Kardiol Pol, 2016; 74: 1160–1166. doi: 10.5603/KP.a2016.0065.

Związek między liczbą monocytów przy przyjęciu do szpitala a brakiem powrotu przepływu w angiografii po pierwotnej przezskórnej angioplastyce wieńcowej u chorych z zawałem serca z uniesieniem odcinka ST

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Streszczenie

Wstęp: Zjawisko braku przywrócenia przepływu (*no-reflow*) w trakcie pierwotnej przezskórnej interwencji wieńcowej (pPCI) u chorych z zawałem serca z uniesieniem odcinka ST (STEMI) może się wiązać z niekorzystnym rokowaniem. Wykazano, że monocyty mogą uczestniczyć w patogenezie choroby wieńcowej i wpływać na zwiększenie ryzyka zawału serca.

Cel: Badanie przeprowadzono w celu oceny zależności między liczbą monocytów przy przyjęciu do szpitala a zjawiskiem braku przepływu po pPCI obserwowanym w angiografii.

Metody: Do badania włączono ogółem 236 chorych z ostrym STEMI poddanych pPCI. Pacjentów podzielono na dwie grupy (grupa, w której nie przywrócono przepływu i grupa z prawidłowym przepływem) na podstawie oceny przepływu po pPCI w skali *Thrombolysis in Myocardial Infarction* (TIMI). Brak przywrócenia przepływu definiowano jako stopień przepływu w skali TIMI ≤ 2, a prawidłowy przepływ — jako stopień 3 w skali TIMI. Liczbę monocytów i inne parametry laboratoryjne określono przy przyjęciu do szpitala, przed pPCI.

Wyniki: W grupie z brakiem przepływu znalazło się 43 (18,2%) chorych, a grupa z prawidłowym przepływem liczyła 193 (81,8%) pacjentów. U osób, u których nie przywrócono przepływu, liczba monocytów przy przyjęciu do szpitala była istotnie wyższa ($0,76 \pm 0,48 \times 10^9$/l *vs*. $0,55 \pm 0,29 \times 10^9$/l; p = 0,004). W tej grupie chorych również liczba leukocytów i neutrofili była istotnie wyższa, natomiast stężenie hemoglobiny było znamiennie niższe niż w grupie z prawidłowym przepływem. W analizie wieloczynnikowej liczba monocytów pozostała niezależnym predyktorem zjawiska braku przepływu w angiografii (iloraz szans [OR] 2,665; 95% przedział ufności [CI] 1,102–6,445; p = 0,030), podobnie jak stężenie hemoglobiny (OR 0,978; 95% CI 0,961–0,995; p = 0,013).

Wnioski: Liczba monocytów przy przyjęciu do szpitala i niskie stężenie hemoglobiny były niezależnymi klinicznymi czynnikami predykcyjnymi zjawiska braku przywrócenia przepływu po pPCI u chorych ze STEMI. Obserwacje autorów sugerują, że liczba monocytów przy przyjęciu do szpitala może być parametrem służącym do wczesnej stratyfikacji ryzyka braku przepływu po pPCI, a w efekcie umożliwiającym poprawę postępowania terapeutycznego w celu zapobiegania temu zjawisku.

Słowa kluczowe: liczba monocytów, zjawisko *no-reflow*, pierwotna przezskórna interwencja wieńcowa, ostry zawał serca

Kardiol Pol 2016; 74, 10: 1160–1166

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Praca wpłynęła: 28.12.2015 r. **Zaakceptowana do druku:** 11.04.2016 r. **Data publikacji AoP:** 05.05.2016 r.