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Investigation of the relationship between modified Glasgow prognostic score and no-reflow phenomenon in patients undergoing primary percutaneous coronary intervention for ST-elevation myocardial infarction

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ABSTRACT

Objectives: No-reflow phenomenon (NRP) is a complication associated with poor clinical outcome in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (pPCI). The modified Glasgow prognostic score (mGPS) is a novel immune-inflammatory index, derived from C-reactive protein (CRP) and serum albumin levels and has been shown to be associated with prognosis in heart disease. In this study we aimed to investigated the relationship between mGPS and NRP in patients undergoing pPCI for STEMI.

Methods: A total of 379 patients (aged 59 ± 9.9 years; 54.9% male) were enrollled. The patients were divided into 2 groups:no-reflow (n = 72) and reflow (n = 307). No-reflow was defined as thrombolysis in myocardial infarction (TIMI) ≤ 2 flow. The mGPS of all patients was calculated from blood samples at admission. Logistic regression analysis was performed to determine the independent predictive factors for NRP.

Results: Mean age, pain to balloon duration, troponin T, white blood cell (WBC), Syntax score, neutrophil to lymphocyte ratio (NLR), glucose level, C-reactive protein level (CRP), diabetic and female patient ratio were higher, while left ventricular ejection fraction, ST segment resolution ratio at 60 min, and serum albumin level were lower in the NRP group.Logistic regression analysis showed that WBC count [Hazard ratio (HR): 0.816, 95% confidence interval (CI): 0.728-0.914, $p < 0.001$], NLR (HR: 0.482, CI: 0.355-0.654, $p < 0.001$), pain-to-balloon time (HR: 0.976, CI:0.960-0.991, $p = 0.002$) and mGPS (HR: 3.213, CI: 1.643- 6.283, $p = 0.001$) were independent predictive factors for NRP.

Conclusions: Modified GPS is an independent predictive factor for NRP in patients undergoing pPCI for STEMI.

Keywords: ST-segment elevation myocardial infarction, modified Glasgow prognostic score, no-reflow phenomenon, primary percutaneous coronary intervention

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The aim of treatment of ST-segment elevation myocardial infarction (STEMI) is to achieve rapid and permanent reperfusion as soon as possible. Currently, the most effective and widely used reperfusion therapy is primary percutaneous coronary intervention (pPCI) [1]. No-reflow phenomenon (NRP) is defined as a tissue-level perfusion defect despite restoration of epicardial coronary arteries during percutaneous coronary intervention (PCI). NRP is frequently observed in patients undergoing PCI for acute myocardial infarction [2]. Hypertension, smoking, dyslipidemia, diabetes, renal failure and inflammatory parameters have been shown to be risk factors for NRP in many studies. In addition, a recent study has also demonstrated an association between increased inflammatory status and NRP in elderly individuals [3].

The Modified Glasgow Prognostic Score (mGPS) is a scoring system based on elevated C-reactive protein (CRP) and low serum albumin (SA) levels among inflammatory parameters and has been shown to be effective in prognosis mainly in patients with malignancy [4]. In addition, recent studies have shown an association of mGPS with prognosis in patients with heart failure and acute coronary syndrome [5, 6]. There are also substantial studies demonstrating the association of CRP and albumin, components of mGPS, with NRP [7, 8]. However, according to our literature, there is no study investigating the relationship between NRP and mGPS in patients undergoing pPCI for STEMI and this study was performed to demonstrate this relationship.

METHODS

Patient Population

A total of 379 patients aged 18 to 80 years who underwent pPCI for STEMI between November 2022 and March 2023 were included in this retrospective cross-sectional study. STEMI was diagnosed with symptoms of myocardial ischemia and ST-segment elevation ≥ 1 mm in the two adjacent inferior leads or ≥ 2 mm in the precordial leads, or the presence of newly developed left bundle branch block and elevated cardiac markers. Patients with previous history of coronary artery disease, dysrhythmia, cardiogenic shock, pain for more than 12 hours, fibrinolytic therapy, active infection or chronic autoimmune disease, hematological disease, end-stage renal or hepatic failure, and known malignancy were excluded from the study. The study protocol was approved by the Ethics Committee of Bilecik Şeyh Edebali University Faculty of Medicine in accordance with the Declaration of Helsinki. Informed written consent was obtained from all participants.

Coronary Angiography

All patients diagnosed with STEMI received 300 mg acetylsalicylic acid with 600 mg clopidogrel or 180 mg ticagrelor. Intravenous bolus unfractionated heparin at a dose of 50-70 units/kg was administered to patients who were decided to undergo pPCI. Angiographic evaluations were performed by two experienced invasive cardiologists blinded to the study design using the Artis Zee Floor (Siemens Medical Solution, Erlingen, GERMANY). NRP was visually assessed according to Thrombolysis In Myocardial Infarction (TIMI) flow grade after pPCI.Accordingly, TIMI 0: no flow after the responsible lesion, TIMI 1: opaque is present after occlusion but fails to fill the entire vessel, TIMI 2: opaque fills the entire vessel but with a slower than normal flow, and TIMI 3: normal coronary flow is present. No-reflow was defined as flow in the responsible artery (IRA) ≤ TIMI 2 in the absence of dissection, thrombus or spasm. Patients with residual stenosis below 20% and TIMI 3 flow on IRA were defined as the reflow group.

Laboratory Measurements

Routine biochemical and hematologic parameters of the patients were obtained from the results of analysis of venous blood samples obtained from the antecubital region before coronary angiography. Creatinine kinase MB (CK-MB) and cardiac troponin T (Tn-T), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglyceride, platelet count, neutrophil count, lymphocyte count, serum creatinine, blood glucose, CRP, SA level and other biochemical and hemogram parameters were analyzed. Neutrophil to lymphocyte ratio (NLR) was calculated as the ratio of neutrophil count to lymphocyte count. Glomerular filtration rate was calculated using the Cockcroft-Gault equation.

Definition of Cardiovascular Risk Factors

Hypertension was diagnosed if systolic blood

pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg, or both; or if the individual was taking antihypertensive medication. Patients were defined as having diabetes mellitus (DM) if they were taking anti-diabetic medication or had a fasting glucose level ≥ 126 mg/dL or HbA1c value ≥ 6.5% in at least two measurements. Patients who smoked at least 1 cigarette per day for at least 1 year were defined as smokers. Familial coronary artery disease was defined as the presence of coronary artery disease in a first-degree female relative aged < 65 years or a first-degree male relative aged < 55 years. Hyperlipidemia was diagnosed if total cholesterol >200 mg/dL, LDL-cholesterol > 130 mg/dL, triglycerides > 150 mg/dL or if the patient was on lipid-lowering medication.

Modified Glasgow Prognostic Score (mGPS):

The modified GPS score was calculated as score 0: SA level ≥ 3.5 g/dL and CRP ≤ 1 mg/dL, score 1: SA level ≥ 3.5 g/dL and CRP > 1 mg/dL, score 2: SA level < 3.5 g/dL and CRP > 1 mg/dL.

Electrocardiographic Analysis

A 12-lead standard electrocardiogram (ECG) was performed in all patients at admission and 60 minutes after pPCI. ECG evaluation was performed by two expert cardiologists blinded to the other data of the patients. ST segment elevation was measured in millivolts 20 ms after the J point. Total ST segment elevation in leads DI, aVL, V1-V6 was calculated for noninferior infarction and total ST segment elevation in leads D2, D3, aVF, V5, V6 was calculated for inferior infarction. ST resolution (STR) was calculated as a percentage by calculating the total depression in ST elevation at the specified localizations 60 min after pPCI and proportioning it to the total ST elevation at baseline. Accordingly, patients were classified according to STR as complete STR (≥ 50%) and incomplete (STR < 50%) [9].

Statistical Analysis

SPSS 24.0 version software package (Chicago, IL, USA) was used to analyze the data obtained. A p value < 0.05 was accepted for statistical significance. Visual histogram and Kolmogorow-Smirnow test variables were used for normal distribution assessment. Levene's test was used for homogeneity of variances. Mean ± standard deviation and median and interquartile ranges (25^{th}-75^{th} percentiles) were used for normally distributed continuous variables and abnormally distributed continuous variables, respectively. Categorical variables were expressed as percentages and Chi-square test was used for comparison. Student's t-test was used for the comparison of normally distributed continuous variables and Whitney U test was used for variables that did not fit normal distribution. Univariate regression analysis was performed to determine the variables associated with no-flow and the factors that were significant were included in multivariate regression analysis to determine the independent predictors of no-reflow.

RESULTS

A total of 379 patients, 72 in the NRF group and 307 in the reflow group, were included in the study. The mean age of the patients was 59.9 ± 9.9 years and the male proportion of the population (n = 208) was 54.9%. Demographic characteristics and baseline clinical data of the patients are shown in Table 1. Patients in the NRF group had a higher mean age [62 (58-67) vs. 59 (51-67), $p = 0.002$], diabetes (54.2 % vs. 38.4 %, $p = 0.015$) and female (55.6 % vs. 42.7 %, $p = 0.048$) ratio. The history of drug use was similar in both groups. Pain-to-balloon time was observed to be longer in the NRP group [80 (60-90) vs. 60 (45-80), $p < 0.001$]. When both groups were compared in terms of laboratory and admission physical examination findings, peak TnT [4567 (3031-6536) vs. 3654 (1745-5231), $p < 0.001$], WBC count [13.8 (11.4-15.4) vs. 11.2 (10.2-13), $p < 0.001$], Syntax score [20 (16-24) vs. 18 (12-21), $p < 0.001$], proportion of patients with < 50% STR at 60 min (86.1% vs. 18.9%, $p < 0.001$), NLR [3.2 (2.5-4.2) vs. 2.1 (1.5-2.8), $p < 0.002$], glucose level [187 (146-219) vs.155 (127-200), $p = 0.001$], CRP level [4 (0.8-6) vs. 1.8 (0.7-4.3), $p < 0.001$] and Killip class ≥ 2 (9.7 % vs. 2.6 %, $p = 0.005$) were higher, while LVEF [43 (40-45) vs. 45 (40-50), $p = 0.008$] and SA level [4.0 (3.8-4.1) vs. 4.2 (4.1-4.3), $p < 0.001$] were lower in the NRP group. Modified GPS 1 (50 % vs. 20.8%) and mGPS 2 (9.7% vs. 2.3%) rates were higher in the NRP group (Table 1).

Patients were further categorized as mGPS =0 (group 1) and mGPS ≥ 1 (group 2). Group 2 patients had higher age [62 (55-68) vs. 60 (52-66), $p = 0.037$],

Table 1. Baseline characteristics of the study population

Variable	No-reflow (n = 72)	Reflow (n = 307)	*p* value
Age (years)	62 (58-67)	59(51-67)	0.002
Gender (female), n (%)	40(55.6 %)	130(42.7%)	0.048
BMI (kg/m2)	26.9(24.4-29.4)	27.4(24.8-30.1)	0.420
Diabetes mellitus, n (%)	39(54.2%)	118(38.4%)	0.015
Hypertension, n (%)	28(38.9%)	95(30.9%)	0.195
Current smoking, n (%)	19(26.4 %)	86(28 %)	0.453
ASA/P2Y12-inh, n (%)	18(25%)	80(26.1%)	0.492
ACEI/ARB, n (%)	23(31.9%)	92(30.0%)	0.743
BB, n (%)	18(25%)	76(24.8%)	0.966
CCB, n (%)	6(8.0%)	22(7.1%)	0.756
Statin, n (%)	15(20.8%)	62(20.2%)	0.904
SKB (mmHg)	132(110-140)	130(110-140)	0.089
DKB (mmHg)	85(76-91)	80(70-90)	0.153
Heart rate, (beat/min)	80(70-86)	78(68-86)	0.706
Pain-to-balon time (sec)	80(60-90)	60(45-80)	**< 0.001**
LVEF (%)	43(40-45)	45(40-50)	**0.008**
SYNTAX score	20(16-24)	18(12-21)	**< 0.001**
Peak troponın T (ng/L)	4567(3031-6536)	3654(1745-5231)	**<0.003**
WBC Count (10^3/μL)	13.8(11.4-15.4)	11.2(10.2-13)	**< 0.001**
NLR	3.2(2.5-4.2)	2.1(1.5-2.8)	**< 0.001**
Hemoglobin (g/dL)	14(13-15)	14.1(13-15)	0.474
Platelet count ($\times10^9$/L)	261(157-361)	257(165-401)	0.244
Creatinine (mg/dL)	0.93(0.8-1.1)	0.9(0.7-1.0)	0.850
Glucose (mg/dL)	187(146-219)	155(127-200)	**0.001**
Total cholesterol (mg/dL)	200(186-212)	199(181-213)	0.721
Triglycerides (mg/dL)	179(158-198)	178(158-199)	0.893
HDL-C (mg/dL)	37(33-39)	36(33-40)	0.820
LDL-C (mg/dL)	149(133-160)	142(127-157)	0.107
CRP (mg/L)	4.0(0.8-6)	1.8(0.75-5.2)	**< 0.001**
Albumin (mg/dL)	4.0(3.8-4.1)	4.2(4.1-4.3)	**< 0.001**
mGPS, n (%)			
0	29(40.3%)	236(76.9%)	**< 0.001**
1	36(50.0%)	64(20.8%)	**< 0.001**
2	7(9.7%)	7(2.3%)	**< 0.001**
STR (%)			
≥ 50	10(13.9%)	249(81.1%)	**< 0.001**
< 50	62(86.1%)	58(18.9%)	**< 0.001**
Killip class, n(%)			
Class 1	65(90.3%)	299(97.4%)	**0.005**
Class ≥2	7(9.7%)	8(2.6%)	**0.005**

CCB = Calcium channel blocker, ACEI = Angiotensin-converting enzyme inhibitor, ARB = Angiotensin receptor blockers, ASA = acetylsalicylic acid, BB = Beta-blocker, BMI = Body Mass Index, CRP = C-reactive protein, HDL-C = High density lipoprotein cholesterol, LDL-C = Low density lipoprotein cholesterol, LVEF = Left ventricle ejection fraction, WBC = white blood cell, STR = ST-segment resolution, SBP = systolic blood pressure, DBP = diastolic blood pressure, SYNTAX = Synergy between PCI with Taxus and Cardiac Surgery, NLR = Neutrophıl lymphocyte ratıo, mGPS = modified Glasgow prognostic score

Table 2. Baseline characteristics according to the mGPS groups

Variable	mGPS ≥ 1 (n = 82)	mGPS = 0 (n = 297)	*p* value
Age (years)	62 (55-68)	60 (52-66)	**0.037**
Gender (female), n (%)	45 (54.9%)	126 (42.4%)	**0.045**
BMI (kg/m^2)	27.2 (24.2-28.7)	27.3 (24.9-30.1)	0.177
Diabetes mellitus, n (%)	40 (48.8%)	117 (39.4%)	0.127
Hypertension, n (%)	28 (34.1%)	95 (32.0%)	0.712
Current smoking, n (%)	26 (31.7 %)	79 (26.6 %)	0.218
ASA/P2Y12-inh, n (%)	19 (23.2%)	79 (26.6%)	0.317
ACEI/ARB, n (%)	24 (29.3%9	91 (30.6%)	0.811
BB, n (%)	21 (25.6%)	73 (24.6%)	0.848
CCB, n (%)			
Statin, n (%)	18 (22%)	59 (19.9%)	0.678
SKB (mmHg)	130 (110-140)	131 (110-142)	0.820
DKB (mmHg)	81 (70-90)	80 (72-90)	0.676
Heart rate (beat/min)	79 (66-88)	78 (68-86)	0.484
Pain-to-balon time (sec)	70 (50-90)	60 (50-80)	**0.045**
LVEF (%)	44.5 (40-48)	45 (40-48)	0.281
SYNTAX score	18 (14.7-23)	19.8 (12-21.5)	**0.036**
Peak troponın T (ng/L)	4696 (2504-6605)	3654 (1980-5231)	**0.002**
WBC Count (10^3/μL)	12.6 (11-15)	11.3 (10.1-13.4)	**< 0.001**
NLR	2.8 (1.7-3.9)	2.1 (1.6-2.9)	**0.002**
Hemoglobin (g/dL)	14 (13-15)	14.1 (13-15)	0.706
Platelet count (×10^9/L)	265 (168-401)	268 (165-420)	0.765
Creatinine (mg/dL)	0.97 (0.8-1.12)	0.9 (0.8-1.04)	0.130
Glucose (mg/dL)	180.5 (143-209)	156 (126-200)	**0.002**
Total cholesterol (mg/dL)	199.5 (187-213)	200 (181-213)	0.818
Triglycerides (mg/dL)	178.5 (160-197)	178 (158-199)	0.795
HDL-C (mg/dL)	36.9 (33-41)	37 (33-40)	0.641
LDL-C (mg/dL)	142 (125-156)	144 (127-158)	0.582
CRP (mg/L)	5 (4-6)	1.78 (0.8-4.3)	**<0.001**
Albumin (mg/dL)	4.0 (3.8-4.2)	4.1 (4-4.3)	**0.001**
No-reflow (%)	35 (42.7%)	37 (12.5%)	<0.001
STR (%)			
≥ 50	33 (40.2%)	226 (76.1%)	**< 0.001**
< 50	49 (59.8%)	71 (23.9 %)	
Killip class, n (%)			
Class 1	78 (95.1%)	286 (96.3%)	0.629
Class ≥2	4 (4.9%)	11 (3.7%)	

CCB = Calcium channel blocker, ACEI = Angiotensin-converting enzyme inhibitor, ARB = Angiotensin receptor blockers, ASA = acetylsalicylic acid, BB = Beta-blocker, BMI = Body Mass Index, CRP = C-reactive protein, HDL-C = High density lipoprotein cholesterol, LDL-C = Low density lipoprotein cholesterol, LVEF = Left ventricle ejection fraction, WBC = white blood cell, STR = ST-segment resolution, SBP = systolic blood pressure, DBP = diastolic blood pressure, SYNTAX = Synergy between PCI with Taxus and Cardiac Surgery, NLR = Neutrophıl lymphocyte ratio, mGPS = modified Glasgow prognostic score

Table 3. Univariate and multivariate logistic regression analysis of the association between the no-reflow phenomenon and multiple parameters

	Univariate analysis		Multivariate analysis	
Variables	**OR (95% CI)**	***p value***	**OR (95% CI)**	***p value***
Gender (female)	1.670 (0.993-2.808)	0.053		
Age	0.961 (0.935-0.987)	**0.004**	1.020 (0.981-1.060)	0.323
Diabetes mellitus	0.528 (0.315-0.886)	**0.016**	0.814 (0.409-1.620)	0.557
ASA/P2Y12-inh	1.051 (0.588-1.879)	0.867		
WBC Count	0.732 (0.652-0.821)	**< 0.001**	0.816 (0.728-0.914)	**< 0.001**
Glucose	0.991 (0.985-0.996)	**0.001**	0.997 (0.990-1.005)	0.491
NLR	0.424 (0.332-0.543)	**0.013**	0.482 (0.355-0.654)	**< 0.001**
Pain-to-balloon time	0.963 (0.950- 0.976)	**< 0.001**	0.976 (0.960-0.991)	**0.002**
SYNTAX score	0.926 (0.887-0.968)	**< 0.001**	0.975 (0.920-1.034)	0.396
CRP	0.729 (0.653-0.813)	**< 0.001**	0.892 (0.772-1.031)	0.123[a]
Albumin	16.416 (5.613-48.016)	**< 0.001**	2.353 (0.557-9.942)	0.244[a]
mGPS	5.233 (2.998-9.132)	**< 0.001**	3.213 (1.643-6.283)	**0.001**[b]

WBC = White blood cell, NLR = Neutrophil lymphocyte ratio, CRP = C-reactive protein, SYNTAX = Synergy between PCI with Taxus and Cardiac Surgery, mGPS = modified Glasgow prognostic score.

[a]The variables (Age, Diabetes mellitus, Glucose, Pain-to-balloon time, SYNTAX score, CRP and albumin) were tested in a multivariable analysis.

[b]The variables (Age, Diabetes mellitus, WBC Count, Glucose, NLR, Pain-to-balloon timeSYNTAX score and mGPS) were tested in a multivariable analysis

female ratio [54.9% vs. 42.4%, $p = 0.045$], Syntax score [18 (14.7-23) vs. 19.8 (12-21.5), $p = 0.036$], pain to balloon time [70 (50-90) vs. 60 (50-80), $p = 0.045$], peak TnT level [4696 (2504-6605) vs. 3654 (1980-5231), $p = 0.002$], WBC count [12.6 (11-15) vs. 11.3 (10.1-13.4), $p < 0.001$], NLR [2.8 (1.7-3.9) vs. 2.1 (1.6-2.9), $p = 0.002$], glucose level [180.5 (143-209) vs. 156 (126-200), $p = 0.002$], no-reflow rate (42.7% vs. 12.5%, $p < 0.001$) and CRP level [5 (4-6) vs. 1.78 (0.8-4.3), $p < 0.001$], while SA level [4.0 (3.8-4.2) vs. 4.1 (4-4.3), $p < 0.001$], and the proportion of patients with ≥ 50% STR at 60 min (40.2% vs. 76.1%, $p < 0.001$) were lower (Table 2).

The results of the multivariate analysis performed to determine the predictive factors for NRP based on demographic, clinical and procedural parameters that were found to be significantly associated with NRP according to univariate analysis are shown in Table 3. According to the results of this analysis, WBC count [Hazard ratio (HR): 0.816, 95% confidence interval (CI): 0.728-0.914, $p < 0.001$], NLR (HR: 0.482, CI: 0.355-0.654, $p < 0.001$), pain to balloon time (HR: 0.976, CI: 0.960-0.991, $p = 0.002$) and mGPS (HR: 3.213, CI: 1.643-6.283, $p = 0.001$) were found to be significant and independent predictive factors for NRP.

DISCUSSION

NRP is a serious complication associated with mortality and morbidity frequently seen during pPCI [10]. Although it has been an area of interest for invasive cardiologists in recent years, its pathophysiology is still unclear. However, vasoconstriction, platelet and leukocyte activation, oxygen radicals released after reperfusion, endothelial damage, and dysfunction are the parameters blamed in pathophysiology [11]. The presence of an invisible microthrombus during PCI may also contribute to the development of NRP, the pathophysiology of which is unknown. Therefore, tests to identify patients who may be at risk for NRP before the procedure becomes even more important. In this study, we investigated the relationship between

mGPS, an inexpensive and easy-to-calculate scoring system, and NRP and demonstrated that mGPS is an independent and important predictor factor for NRP.

Diabetes, hypertension, smoking, dyslipidemia, renal insufficiency, age, and inflammatory parameters are accepted cardiovascular risk factors for NRP [3]. In our study, in parallel with these findings, it was observed that NRP group patients were older, female, and had a higher proportion of diabetic patients.

It is thought that endothelial damage, leukocyte and platelet accumulation at the microvascular level, oxygen radicals released after reperfusion and the inflammatory process play an important role in the pathophysiology of NRP [12]. There are also studies demonstrating the association of CRP and SA, components of modified GPS, with NRP [7, 8]. Recent data clearly demonstrate the relationship between inflammation and atherosclerosis. Therefore, inflammatory biomarkers are frequently used in both the diagnosis and prognosis of coronary artery disease. Among these, CRP is the most commonly used parameter. Increased CRP is associated with the risk of myocardial infarction and stroke in asymptomatic individuals and with recurrent coronary events, morbidity, and mortality in patients with stable coronary artery disease and acute coronary syndrome [13]. It is thought that increased CRP level decreases vasodilation at the microvascular level directly and by affecting endothelium-dependent mediators [14]. CRP is also thought to contribute to vasoconstriction at the microvascular level in which NRP is also thought to be involved. CRP is also thought to play a role in endothelial damage and activation of lymphocytes and platelets accumulated in this region [15]. In this study, SA level, the other component of mGPS, was found to be lower in the NRP group. Kurtul *et al*. [7] also demonstrated that low SA was a risk for NRP in patients undergoing pPCI for STEMI and this finding is in parallel with the results of our study. The pro-inflammatory state caused by plaque rupture may contribute to the development of NRP after PCI by increasing platelet aggregation. In addition, albumin inhibits platelet aggregation by increasing PGD2 production, which has direct and indirect antiaggregant effects [16, 17]. On the other hand, hypoalbuminemia is thought to cause endothelial damage by increasing both blood viscosity and free lysophosphatidylcholine concentration [18]. The results of this study support the mechanisms mentioned above and are thought to be involved in the pathophysiology of NRP.

In this study, pain-balloon duration was also shown to be an independent factor for NRP. Early after AMI, the thrombus at the site of the responsible lesion contains platelet-rich, erythrocyte-rich, and red fibrin. Thrombus of this character is usually resolvable with antiaggregant therapy such as tirofiban and abciximab. However, as time progresses, the thrombus becomes more robust and it is thought that fragmented fragments after ballooning and stenting performed during pPCI disrupt microvascular perfusion in the distal region and cause NRP [19].

In this study, another finding supporting the relationship between inflammation and NRP was that increased WBC count and NLR were shown to be predictor factors for NRP. Leukocytes accumulate in small vessel beds in the infarct zone, which plays a key role in the pathophysiology of NRP. The increase in neutrophil adhesion molecules in this region leads to the activation of leukocytes and monocytes. Aggregation of activated leukocytes in the capillary region may directly disrupt blood flow [20]. In addition, leukocyte aggregation is thought to cause additional vascular injury by leading to edema in the endothelial bed, increased permeability and oxygen radicals [21]. Consistent with these mechanisms, WBC count is usually high in patients with acute myocardial infarction. Increased WBC count is associated with poor prognosis and risk of developing NRP, as shown in previous studies [22, 23].

Limitations

Single centers and a small number of patients are the main limitations of the study.

CONCLUSION

The aim of STEMI treatment is to achieve revascularization as effectively and as early as possible. However, NRP, defined as impaired myocardial perfusion despite epicardial coronary revascularization, is frequently seen during pPCI and is also associated with mortality. In this study, mGPS based on CRP and SA levels, which are routine laboratory parameters, was

shown to be an independent predictor factor for NRP. For patients undergoing pPCI for STEMI, this score may help identify individuals at risk for NRP before the procedure and determine adjuvant treatment. However, larger studies are needed before mGPS can be routinely used to predict NRP.

Authors' Contribution

Study Conception: MK, KT; Study Design: MK, KT; Supervision: MK, KT; Funding: N/A; Materials: N/A; Data Collection and/or Processing: MK, RA, CA; Statistical Analysis and/or Data Interpretation: MK, KT; Literature Review: MK, RA, CA; Manuscript Preparation: MK and Critical Review: MK, RA, CA.

Conflict of interest

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