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Predictive value of oxidative, antioxidative, and inflammatory status for left ventricular systolic recovery after percutaneous coronary intervention for ST-segment elevation myocardial infarction

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SUMMARY

OBJECTIVE: This study aimed to evaluate the association between left ventricular ejection fraction recovery and the total oxidant status, total antioxidant capacity, and high-sensitivity C-reactive protein levels.

METHODS: A total of 264 ST-elevation myocardial infarction patients were classified into two groups according to baseline and 6-month follow-up left ventricular systolic function: reduced and recovery systolic function. Predictors of the recovery of left ventricular ejection fraction were determined by multivariate regression analyses.

RESULTS: Multivariable analysis indicated that oxidative status index, baseline left ventricular ejection fraction and peak creatine-kinase myocardial bundle level, and high-sensitivity C-reactive protein were independently associated with the decreased of left ventricular ejection fraction at 6-month follow-up.

CONCLUSION: Oxidative stress and inflammation parameters were detrimental to the recovery of left ventricular ejection fraction in patients with ST-elevation myocardial infarction.

KEYWORDS: Ventricular dysfunction. Oxidants. Antioxidants. ST elevation myocardial infarction.

INTRODUCTION

Primary percutaneous coronary intervention (p-PCI) is recommended as the preferred reperfusion strategy for acute ST-segment elevation myocardial infarction (STEMI) patients who are admitted within the first few hours after the initiation of symptoms[1]. Unfortunately, a significant proportion of patients undergoing STEMI remain with reduced left ventricular systolic function (LVSF)[2]. LVSF is the most important prognostic indicator of in-hospital and long-term mortality of patients with STEMI[3]. Therefore, early identification of these patients is vital because early interventions such as more intense anti-remodeling therapy, close follow-up, and implantation of automated cardioverter-defibrillator may be beneficial for these patients[3].

Several underlying mechanisms including local ischemia and myocardial cell death, oxidative stress and inflammation in the injured myocardial tissue, cardiodepressive effects of reactive oxygen species (ROS) and inflammatory cytokines, changes in the extracellular matrix in response to metalloproteinase activation, structural changes due to mechanical stress, and increased synthesis of collagen and myocardial fibrosis are responsible for the pathogenesis of LV remodeling[4-6]. These processes are interrelated and enable the advancement of the disease from acute to chronic. Furthermore, oxidative stress and inflammation play an essential role in the apoptotic and necrotic death seen in cardio-myositis[4,5]. In this study, we aimed to evaluate the association between total oxidative status (TOS), total antioxidative capacity (TAC), and high-sensitivity C-reactive protein (hs-CRP) in the development of left ventricular systolic dysfunction (LVSD) in patients presenting with STEMI.

METHODS

Study population

This cohort study initially recruited patients with a first STEMI. A total of 1980 adult patients presenting with STEMI between

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February 2010 and April 2016 were screened. Patients with a diagnosis of acute myocardial infarction (MI) based on clinical, electrocardiographic, and cardiac biomarker criteria[7] and an echocardiographic LVEF ≤0.40 were included. Transthoracic echocardiography was performed in each patient before randomization, and the echocardiographic LVEF was determined by the Simpson method. The inclusion criteria also included successful PCI (defined as Thrombolysis in Myocardial Infarction [TIMI] flow grade 3 and residual stenosis of the infarct-related artery 30%) performed 12 h after the onset of symptoms and informed consent to perform echocardiography at three predefined time points.

Exclusion criteria were defined as clinical signs of congestive heart failure or cardiogenic shock in the first week after infarction, other significant cardiac diseases, EF >0.40, life-limiting noncardiac disease, Killip class IV heart failure, prior MI, severe chronic obstructive pulmonary disease, and symptomatic peripheral arterial disease. Based on these criteria, 1716 patients were excluded: 1561 due to EF >0.40, 6 with Killip class IV heart failure, 15 due to the presence of a re-flow phenomena, 2 with severe chronic obstructive pulmonary disease, 10 because of prior heart failure, and 30 with previous MI. In addition, 5 patients died before randomization and 5 refused to participate, 47 patients were excluded due to noncompliance in the follow-up, 35 patients were excluded due to side effects or noncompliances of drugs. Therefore, a total of 264 patients (aged 23–91 years) were included in this study. At discharge, patients were administrated medical therapy according to contemporary guidelines[8,9]. Clinical and echocardiographic evaluations were repeated at 6 months according to the institutional guideline-based pre-hospital, in-hospital, and outpatient clinical care track protocol (MISSION!)[10]. Afterward, the patients were divided into two groups according to their LVEF at 6-month follow-up: LVEF ≤40% (nonrecovery) and LVEF >40% (recovery) (Figure 1). Clinical data were collected in the Cardiology Department (Microsoft access system) and hospital Information System (Enlilsoft®). The study complies with the Declaration of Helsinki of 1975, as revised in 1983. The Institutional Review Board approved the study, and written informed consent was obtained from all subjects.

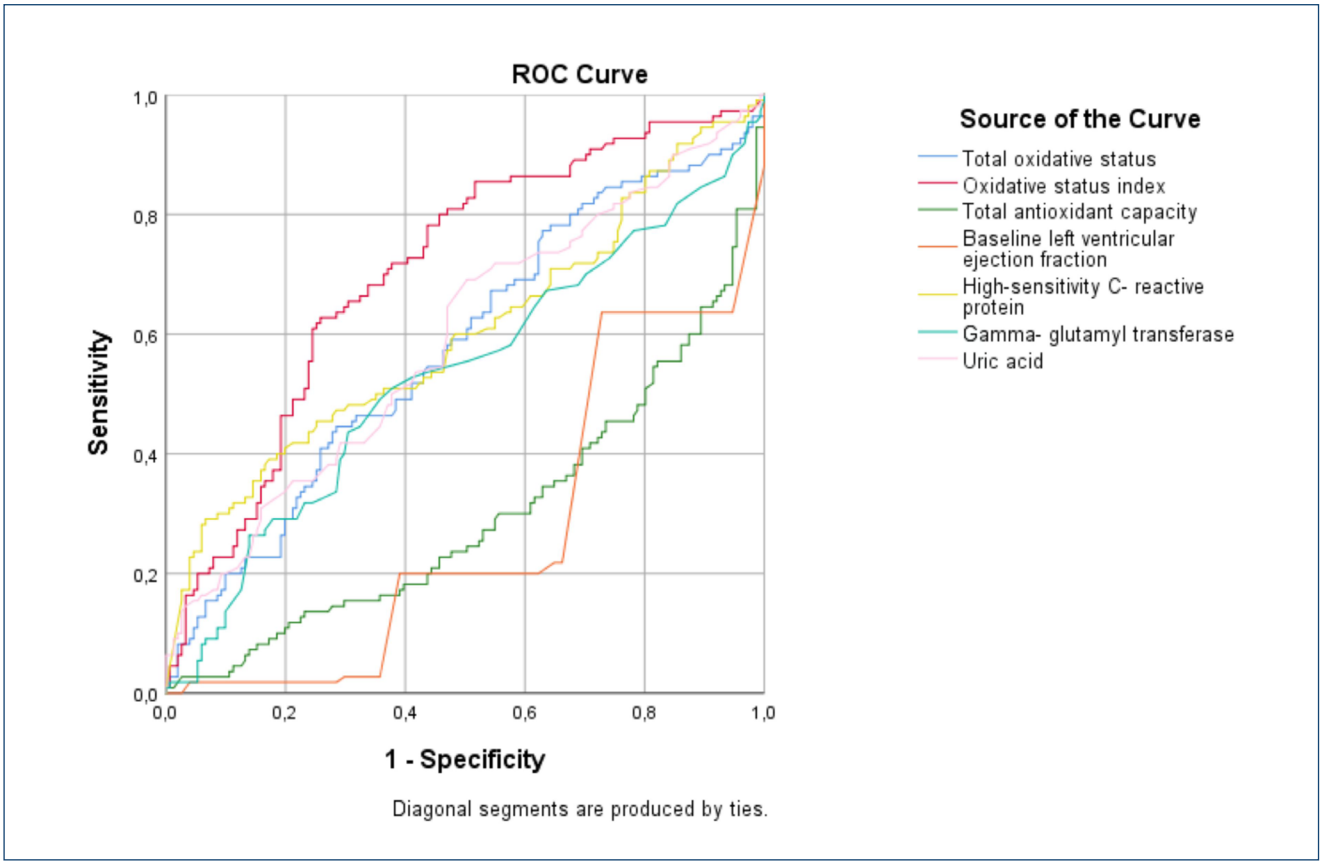

Figure 1. Receiver operating characteristic curve with calculated area under the curve and optimal cutoff point for oxidative status index, total antioxidant capacity, total oxidative status, baseline left ventricular ejection fraction, high-sensitivity C-reactive protein, gamma-glutamyltransferase, and uric acid to identify the recovery of left ventricular ejection fraction.

Transthoracic echocardiography protocol

Transthoracic echocardiography was performed within 12 h of admission and at the completion of the study procedures using a commercially available system (X5-1 probe, IE33, Philips, Andover, MA, USA) with a 3.5-MHz or M5S transducer from standard parasternal and apical transducer positions with two-dimensional frame rates of 60–100 frames/s and tissue Doppler frame rates >100 frames/s. Standard M-mode, 2D, color, pulsed, and continuous-wave Doppler images were acquired and stored digitally for subsequent off-line analysis (Xcelera, Phillips Healthcare) by two echocardiography specialists blinded to the study time point, treatment allocation, and oxidative and antioxidant status values. The LVEF was calculated in the apical four- and two-chamber views using Simpson's biplane method[11].

Laboratory analysis

Serum total oxidative stress (TOS), TAC levels, hs-CRP, gamma-glutamyltransferase (GGT), and uric acid (UA) levels were measured at baseline. Decreased TAC and increased TOS and UA levels were used as markers of oxidative stress, and increased hs-CRP was used as a marker of inflammation. Laboratory analysis was performed as stated in the previous study[12-14]. TAC and TOS levels were determined with a spectrophotometric kit (Rel Assay Diagnostics, Gaziantep, Turkey) and read in an auto-analyzer (Olympus AU2700; Olympus, Tokyo, Japan). The TAC and TOS levels were expressed as mmol Trolox equivalent/L and mmol H_2O_2 equivalent/L, respectively. The oxidative status index (OSI) is defined as the ratio of TOS to TAC levels, expressed as a percentage. For the calculation of OSI, TAC units were represented as mmol/L, and the OSI value calculated according to the following formula: OSI (arbitrary unit) = TOS (mmol H_2O_2 equiv./L)/TAC (mmol Trolox equiv./L)[12-14].

Statistical analysis

The SPSS version 16.0 software package was used for statistical analyses in this study. Categorical variables were expressed as frequency (%) and compared using the χ^2 test. Kolmogorov-Smirnov test was used to test the distribution of numeric variables; those with normal distribution were expressed as mean±standard deviation and were compared with Student's t-test. Data without normal distribution were expressed as median (interquartile range of 25–75% percentiles) and were compared with the Mann-Whitney U-test. If groups were more than two, continuous variables were compared using one-way ANOVA or the Kruskal-Wallis test. In all statistical analyses, p-value <0.05 was considered as statistically significant. Regression and receiver operating characteristics (ROC) curve analysis was performed as stated in the previous study[14].

RESULTS

Of the 1980 patients with acute STEMI examined, 264 patients with an LVEF ≤40% in admission were included. The mean age of the patient population was 62.08±12.8 years (range 23–91) and 81% were males (Table 1). In 143 (47.7%) patients, the only culprit lesion was in the left anterior descending coronary artery. All patients received the treatment considered appropriate by the current guidelines at discharge. Echocardiographic data obtained within 24 h of admission are presented in Table 1. The mean initial LVEF was 30.6±4.3%, while the mean follow-up LVEF was 42±8.2%. Moderate-to-severe mitral regurgitation was observed in 59 (23%) patients. At 6-month follow-up, 129 (48%) patients did not show any recovery of LVSF, and the LVEF remained ≤40%. The remaining 135 (52%) patients showed LVSF recovery (Table 1).

Univariate and multivariate analyses were performed to evaluate the correlates of reduced LVEF (<40%) at 6-month follow-up. Univariate analysis showed that peak troponin T, peak CK-MB, blood urea nitrogen (BUN), TOS, TAC, OSI, UA, hs-CRP levels, age, initial heart rate, and baseline LVEF were significantly correlated with LVEF recovery. Multivariate analysis showed that OSI (odds ratio [OR] 1.12, 95% confidence interval (CI) (1.06–1.18); p<0.001), baseline LVEF (OR 0.85, 95%CI 079–0.91; p=0.006), and peak CK-MB level (OR 1.004, 95%CI 1.002–1.006; p<0.001) were independently associated with normalization of LVEF (>40%) at 6-month follow-up (Table 2). ROC curve analysis showed that OSI (C-statistic 0.723; 95%CI 0.66–0.77, p<0.001), TOS (C-statistic 0.579; 95%CI 0.52–0.63, p<0.001), TAC (C-statistic 0.719; 95%CI 0.66–0.76, p<0.001), initial LVEF (C-statistic 0.734; 95%CI 0.68–0.78, p<0.001), hs-CRP (C-statistic 0.59; 95%CI 0.52–0.67, p=0.006), and UA (C-statistic 0.59; 95%CI 0.52–0.66, p=0.012) were significant predictors of LVEF recovery following STEMI (Figure 1). We calculated the cutoff point of 20 for OSI, 1.3 for TAC, 25 for TOS, 30 for initial LVEF, 54 for hs-CRP, and 5.88 for UA to estimate the LVEF recovery following STEMI, with a sensitivity of 64, 56, 76, 78, 29, and 68% and a specificity of 75, 78, 39, 67, 93, and 49%, respectively.

DISCUSSION

The main finding of this study is the association of oxidant/antioxidant status and inflammation parameters with the recovery of LV functions in patients presenting with acute STEMI.

Table 1. Demographic and clinical characteristics of patients with and without depressed left ventricular ejection fraction.

	Group I (n=128)	Group II (n=136)	p-value
Baseline characteristics			
Female gender, n (%)	27 (21.1)	25 (18.4)	0.345
Diabetes mellitus, n (%)	31 (24.2)	39 (28.7)	0.248
Hypertension, n (%)	65 (50.8)	55 (40.4)	0.059
Hyperlipidemia, n (%)	33 (25.8)	31 (22.8)	0.336
Smoking, n (%)	74 (57.8)	88 (64.7)	0.153
Obesity, n (%)	46 (35.9)	52 (38.2)	0.398
Age (years)	64.8±12.7	61.1±12.7	0.017
Systolic blood pressure (mmHg)	123.00±26.6	123.6±29.0	0.849
Diastolic blood pressure (mmHg)	74.4±15.8	73.5±14.1	0.634
Heart rate (bpm)	79.6±19.0	75.9±16.9	0.96
Weight (kg)	73.2±13.5	76.3±12.4	0.056
Length (cm)	165.64±7.5	167.1±7.5	0.108
BMI	26.2±4.0	26.8±3.9	0.235
Waist circumference (cm)	91.6±8.3	93.5±7.6	0.06
Previous treatment, n (%)			
RAS blockers	45 (18.0)	8 (16.0)	0.458
β-Blockers	25 (19.5)	17 (12.5)	0.082
Statins	19 (14.8)	14 (10.3)	0.176
Medication at discharge, n (%)			
ACEi/ARBs			
β-Blockers	125 (98)	134 (99)	0.481
Statins	121 (95)	127 (94)	0.483
Antiplatelet	121 (95)	131 (97)	0.430
Aldosterone antagonists	128 (100)	136 (100)	0.622
Diuretics	40 (31)	13 (10)	<0.001
Device therapy, n (%)			
Cardiac resynchronization therapy	20 (15)	4 (3)	<0.001
Implantable-cardioverter defibrillator	8 (7)	2 (2)	<0.001
Localization of MI, n (%)			<0.001
Anterior	80 (62.5)	47 (37.6)	
Nonanterior	48 (37.5)	89 (65.4)	
Infarct-related artery, n (%)			<0.001
LAD	70 (66.0)	46 (38.0)	
Cx	13 (12.3)	19 (15.7)	
RCA	23 (21.7)	56 (46.3)	
Duration of CCU stay (day)	2.2±0.8	2.0±0.5	0.02
Laboratory findings			
Total antioxidant capacity	1.30±0.2	1.51±0.2	<0.001
Total oxidant capacity	29.55±5.72	27.69±4.99	0.005
OSI	23.51±6.32	18.99±5.50	<0.001
hs-CRP (mg/L), median (IQR)	38.5 (8.7–60.00)	12.7 (6.72–15.00)	<0.001
Uric acid (mg/dL)	6.5±1.6	5.9±1.3	0.001

Continue...

Table 1. Continuation.

	Group I (n=128)	Group II (n=136)	p-value
LDL (mg/dL)	108.2±36.5	107.3±34.4	0.843
HDL (mg/L), median (IQR)	43.00 (35.00–48.00)	39.00 (33.00–44.00)	0.006
Trigliserit (mg/dL)	123.2±62.9	152.6±126	0.017
Total cholesterol (mg/dL)	175.8±41.6	176.1±42.4	0.959
Initial creatinine (mg/dL)	1.1±0.3	1.0±0.2	0.004
BUN	21.08±8.62	17.8±5.8	< 0.001
Initial glucose (mg/dL)	179.3±100	167.4±77	0.280
Hemoglobin (mg/dL)	14.3±2.6	14.2±1.7	0.802
Platelet count	237±80	233±66	0.659
Initial CK-MB (U/L)	36.5 (22.00–69.75)	29.00 (20.00–50.00)	0.009
Initial troponin (ng/mL)	0.18 (0.40–0.58)	0.09 (0.01–0.44)	0.037
Peak CK-MB (U/L)	250.00 (100.00–368.00)	135.00 (81.00–234.00)	<0.001
Peak troponin (ng/mL)	5.5 (2.8–9.4)	3.1 (1.1–5.9)	< 0.001
Echocardiographic parameters			
LV diastolic diameter (mm)	50.8±4,5	48.5±3.5	<0.001
LV systolic diameter (mm)	36.6±4.9	32.2±5.0	<0.001
IVS (mm)	11.6±1.1	11.3±1.1	0.07
Left atrial diameter (mm)	40.3±3.2	39.4±3.7	0.04
LV ejection fraction at baseline (%)	28.9±4.0	32.2±4.0	< 0.001
LV ejection fraction in the first year (%)	34.8±4.3	48.8±4.5	<0.001
LV mass index (g/m)	117±33	104±32	<0.001

BMI: body mass index; RAS: renin–angiotensin system; ACEi/ARBs: angiotensin-converting enzyme inhibitors/ angiotensin ii receptor blockers; MI: myocardial infarction; LAD: left anterior descending artery; Cx: circumflex; RCA: right common artery; CCU: coronary care unit; OSI: oxidative status index; hs-CRP: high-sensitivity C-reactive protein; LDL: low-density lipoprotein; HDL: high-density lipoprotein; IQR: interquartile range; BUN: blood urea nitrogen; CK-MB: creatine kinase myocardial bundle; LV: left ventricle; IVS: interventricular septum; GFR: glomerular filtration rate, BUN: blood urea nitrogen. Group I: patients with depressed left ventricle ejection fraction; Group II: Patients with recovery left ventricle ejection fraction after 1 year to the ST-segment elevation myocardial infarction. Data presented as mean±SD or number (%) of the patients.

Table 2. Univariate and multivariate regression analysis of predictors of left ventricular recovery in the study population.

	Unadjusted odds ratio	Confidence interval	p-value	Adjusted odds ratio	Confidence interval	p-value
TOS	1.05	1.00–1.09	0.017			
TAS	0.05	0.02–0.13	<0.001			
OSI	1.14	1.09–1.19	<0.001	1.12	1.06–1.18	<0.001
Age	1.03	1.01–1.04	0.002			
ACEi	3.48	1.39–8.67	0.007			
CK-MB peak	1.004	1.002–1.005	<0.001	1.004	1.002–1.006	<0.001
Troponin peak	1.06	1.01–1.12	0.014			
BUN	1.06	1.02–1.10	0.001			
Uric acid	1.27	1.07–1.50	0.005			
hs-CRP	1.01	1.01–1.02	<0.001	1.016	1.005–1.028	0.006
Heart rate	1.01	0.99–1.02	0.07			
LV ejection fraction (baseline)	0.80	0.75–0.85	<0.001	0.85	0.79–0.91	<0.001

TOS: total oxidative status; TAS: total antioxidative status; OSI: oxidative status index; ACEi: Angiotensin-converting enzyme inhibitors; CK-MB: creatine-kinase myocardial binding; BUN: blood urea nitrogen; hs-CRP: high-sensitivity C-reactive protein; LV: left ventricle.

The other findings of this study are OSI, baseline LVEF, and peak CK-MB level were independently associated with normalization of LVEF (>40%) at 6-month follow-up and ejection fraction alteration (ΔEF) at 6-month follow-up was positively correlated with TAC and negatively correlated with TOS, OSI, baseline LVEF, hs-CRP, and UA.

Consistent with our data, previous studies have suggested that oxidative stress, increased inflammation, and decreased antioxidant capacity are associated with poor cardiovascular outcomes[4]. In contrast, we evaluated a possible role of TAC, TOS, and inflammatory status in LV systolic recovery after a first STEMI. We found that increased inflammation and TOS and decreased TAC might pave the way for permanent myocardial dysfunction in patients with STEMI. This study results showed that depending on the underlying inflammation, oxidant and antioxidant status accompanying myocardial contractile dysfunction might contribute to LVSD in patients with STEMI.

Borekci et al.[15] have demonstrated that OSI, UA, and neutrophil-to-lymphocyte ratio were associated with spontaneous reperfusion in patients with STEMI. Similarly, Turan et al.[16] have reported that plasma TOS and OSI were associated with the complexity and severity of coronary artery disease in patients with acute coronary syndrome. Additionally, we have previously reported a positive association between the development of atrial fibrillation after STEMI and TAC, TOS, and OSI[17]. However, none of these studies addressed the relationship between LVSD and oxidative stress markers in patients with STEMI. Data from this study suggest that in STEMI patient population, plasma TAC, TOS, and OSI were increased in patients with LVSD when compared to those without LVSD. Thus, increased oxidative stress may contribute to pathogenesis in these patients.

LVSF is the most important predictor of in-hospital and long-term prognosis in patients with STEMI[3]; therefore, an estimate of which patients may develop LVSD is critical. Abou et al.[18] showed that smaller enzymatic infarct size, baseline LVEF, and absence of mitral regurgitation were independently associated with LVEF recovery at follow-up. Using the Korean Acute Myocardial Infarction Registry and Korean Myocardial Infarction Registry, Oh et al. showed that recovery of LVSD was observed in 51% of the subjects. The same study reported that moderate systolic dysfunction, Killip class I-II, lack of use of diuretics, non-STEMI, lower peak troponin I level, single-vessel disease, non-left anterior descending culprit lesion, and statin use were independent predictors of recovery of depressed LVEF[19]. In the PREDICTS study, Brooks et al. showed that EF >35% at presentation, length of stay, prior MI, lateral wall motion abnormality at presentation, and peak troponin were related to the recovery of LVSD[20]. Similarly, in this study, low EF at presentation, higher CK-MB, and hs-CRP levels were independently associated with depressed LVEF. Additionally, we report that oxidant and antioxidant parameters were associated with depressed LVEF. Additionally, it has been shown that other inflammatory parameters such as IL-6, IL-1RA, and resistin plasma levels at baseline have a good predictive value both as independent variables and as a group for the development of adverse LV recovery and major cardiovascular outcomes at 6-month follow-up after STEMI[21]. Our findings supported this study.

CONCLUSIONS

Studies have failed to clarify how guideline-based medications influence LVEF recovery at follow-up fully. Previous studies with STEMI patients undergoing p-PCI showed the beneficial effects of these therapies; however, these studies reported lower usage of ACEi, ARB-II, and β-blockers[22,23]. Furthermore, this study showed that some patients did not present with LVEF alteration even when they received contemporary guideline-based medications. Therefore, we suggest that the oxidant, antioxidant, and inflammatory status of the patient may also be implicated in the pathophysiology. This emphasizes the importance of a systematic approach in treatment regimens that includes lifestyle changes and antioxidant therapy.

AUTHORS' CONTRIBUTION

FA: Project administration, Writing – review & editing. **HAB:** Conceptualization, Data curation. **AB:** Investigation, Validation. **HBS:** Formal analysis. **MÖ:** Methodology, Project administration.

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