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ARAŞTIRMA / RESEARCH

The predictive value of GRACE risk score on the left ventricular ejection fraction after acute anterior ST-segment myocardial infarction

Akut anterior ST segment miyokard infarktüsü sonrası GRACE risk skorunun sol ventrikül ejeksiyon fraksiyonundaki prediktif değeri

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Abstract

Purpose: The aim of the study is to investigate the predictive value of GRACE score for left ventricular ejection fraction (LVEF) after acute anterior segment acute myocardial infarction (AMI). Despite rapid and complete reperfusion in AMI, inadequate recovery of left ventricular function may result in a decrease in LVEF.

Materials and Methods: We retrospectively analyzed 712 patients presented with AMI and 290 patients were included. Patients were divided into two groups according to LVEF and a value <50% was defined as depressed EF group (group 1), a value ≥50% was defined as preserved EF group (group 2). The GRACE risk scores of all patients were calculated.

Results: 132 patients were included in the group 1, 158 patients were included in the group 2. In-hospital death GRACE risk score and in-hospital death/MI GRACE risk score were higher in group 1. A significant negative correlation was found between risk scores and LVEF. In multivariate regression analysis, in-hospital death risk score, and in-hospital death/MI risk score were found to be independently predictors of depressed LVEF.

Conclusion: GRACE risk score has a clinically important role predicting depressed LVEF in acute anterior segment AMI patients treated with primary PCI.

Keywords: risk stratification, left ventricular function, acute myocardial infarction, heart failure

Öz

Amaç: Çalışmanın amacı, akut anteriyor miyokard infarktüsünden (AMİ) sonra sol ventrikül ejeksiyon fraksiyonu (SVEF) için GRACE risk skorunun prediktif değerini araştırmaktır. AMİ' da hızlı ve tam reperfüzyona rağmen, sol ventrikül fonksiyonunun yetersiz iyileşmesi SVEF' de azalmaya neden olabilir.

Gereç ve Yöntem: AMİ ile başvuran 712 hasta retrospektif olarak incelendi ve 290 hasta çalışmaya dahil edildi. Hastalar SVEF' ye göre iki gruba ayrıldı ve <% 50' den düşük bir değer düşük EF grubu (grup 1), ≥%50 değeri korunmuş EF grubu (grup 2) olarak tanımlandı. Tüm hastaların GRACE risk skorları hesaplandı.

Bulgular: Grup 1' e 132, grup 2' ye 158 hasta dahil edildi. Hastane içi mortalite GRACE risk skoru ve hastane içi mortalite/Mİ GRACE risk skoru grup 1' de daha yüksek bulundu. Risk skorları ile SVEF arasında anlamlı bir negatif korelasyon mevcuttu. Çok değişkenli regresyon analizinde hastane içi mortalite riski skoru ve hastane içi mortalite/Mİ risk skoru düşük SVEF' nin bağımsız belirleyicileri olarak bulundu.

Sonuç: GRACE risk skoru, primer perkütan koroner girişim ile tedavi edilen akut anteriyor AMİ hastalarında düşük SVEF' yi öngörmede klinik olarak önemli bir role sahiptir.

Anahtar kelimeler: Risk sınıflandırması, sol ventrikül fonksiyonu, akut miyokard infarktüsü, kalp yetmezliği

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INTRODUCTION

Ischemic cardiomyopathy is one of the variants of heart failure (HF) which almost develops as a complication after acute coronary syndromes (ACS)[1]. Improved reperfusion strategies including percutaneous coronary interventions (PCI) and evidence-based pharmacotherapies have provided a permanent diminution in the in-hospital case-fatality rates of ACS over recent decades[2]. Nevertheless, as a short and long term complication of ACS, HF still continues to be associated with mortality[3].

Even providing enough flow in the culprit vessels by rapid and complete reperfusion in acute myocardial infarction (AMI), poor recovery of left ventricular (LV) function and negative remodeling results in a decrease in LV ejection fraction (LVEF). As a parameter used for assessment of LV systolic functions LVEF is the most important predictor of prognosis in patients with AMI[4].

In the ACS patients, especially AMI patients, risk stratification at admission plays an important role as a benefit in reducing adverse outcomes. The Global Registry of Acute Coronary Events (GRACE) risk scores are the major scoring system that are recommended for routine use by current American Heart Association and European Society of Cardiology guidelines[5,6]. Given the poor prognosis took account of depressed LVEF after AMI, we planned and aimed to investigate the relationship and predictive effects of this risk score on the depressed LVEF in patients with anterior segment ST elevation AMI.

MATERIALS AND METHODS

Study groups

In this observational study, we retrospectively investigated the medical records of patients with acute anterior segment myocardial infarction (AAMI) who were admitted our emergency department and successfully treated with PCI within 6 hours from symptoms onset, between July 2011 and December 2015. Patients who underwent primary PCI for left anterior descending coronary artery (LAD) lesions and had post interventional thrombolysis in myocardial infarction (TIMI) 3 flow after primary PCI were enrolled in the study. The AAMI was defined as typical chest pain with a new onset ST-segment elevation from the J point ≥2 consecutive leads with at least 0.2 mV in leads V1-V4 or at least 0.1 mV in the remaining leads on the electrocardiography or new onset left bundle brunch block. Clinical history including age, sex, diabetes mellitus (DM), hypertension (HT), smoking was recorded from medical records. In-hospital death and in-hospital death/MI GRACE risk scores (which include age, creatinine, heart rate, systolic blood pressure, Killip class, cardiac arrest at admission, elevated cardiac markers, and ST-segment deviation) were recorded for every patients[7]. In hospital death score was accepted as the in-hospital 6 months mortality risk prediction, in-hospital death/MI score was accepted as in-hospital 1-year mortality and recurrent myocardial infarction risk prediction[7]. Killip classification was determined by the attending physician in the emergency department. Specifically, Killip class I patients had no evidence of HF; Killip class II patients had mild heart failure with rales involving one third or less of the posterior lung fields and systolic blood pressure of 90 mmHg or higher; Killip class III patients had pulmonary edema with rales involving more than one third of the posterior lung fields and systolic blood pressure of 90 mmHg or higher; Killip class IV patients had cardiogenic shock with any rales and systolic blood pressure lower than 90 mmHg[8]. DM was defined as a fasting glucose value >126 mg/dL, with or without hemoglobin A1c >6.5 %, or current use of medication for DM. HT was defined as having a systolic blood pressure >140 mmHg, and/or diastolic blood pressure >90 mmHg, as well as patient were receiving antihypertensive treatment. We retrospectively analyzed 712 patients presented with AAMI. 73 patients were excluded because of previous coronary artery disease; 62 patients were excluded because of subacute AAMI; 49 patients did not have TIMI 3 flow after primer PCI; echocardiography was not performed at the same hospitalization to 74 patients; 36 patients had never been done echocardiography; 13 patients had anemia (hemoglobin value <13 mg/dL in men, and <12 mg/dL in women); 17 patients were referred for surgical operation (coronary bypass); 3 patients had received fibrinolysis therapy at admission; 42 patients had received glycoprotein 2b/3a inhibitors; 21 patients had spent more than 6 hours before PCI; 32 patients were excluded other reasons (heart failure, valvular heart disease, congenital heart disease, atrial fibrillation, second or third degree atrioventricular block, clinical evidence of any

infection, malignancy, end-stage liver diseases and renal failure, pregnancy, dilated or other form of cardiomyopathies, systemic or autoimmune inflammatory diseases that cause depressed LVEF). The protocol design was approved by the local institutional Research Ethics Committees of our faculty of medicine (70904503/64, 2015).

Angiographic and echocardiographic evaluation

All patients received 300 mg acetylsalicylic acid and 300 mg clopidogrel loading doses before primary PCI. Coronary angiography was performed using percutaneous femoral approach. Unfractioned heparin (60-100 U/kg) was administered after the initial angiographic imaging. Primary PCI was performed to the LAD coronary artery in all patients. After primary PCI, all patients were followed in the coronary care unit with a treatment of 100 mg acetylsalicylic acid, 75 mg clopidogrel, unfractioned intravenous or low molecular weight subcutaneous heparin, captopril, and statin. TIMI flows were estimated according to the Gibson et al.'s[9] method and patients without TIMI 3 flow after primary PCI were excluded from the study.

A two-dimensional transthoracic echocardiography was performed to all AAMI patients with a GE Vivid 7 device (GE Healthcare Inc. Milwaukee, Wisconsin, USA) with 6T, 5 MHz probe lying supine in the left lateral position. LVEF was calculated using Simpson's biplane method. All the patients had undergone to echocardiographic evaluation in the same hospitalization. Patients were divided into two groups according to LVEF. Patients who had a LVEF value <50% was defined as depressed EF group (Group 1), patients who had a LVEF value ≥50% was defined as preserved EF group (Group 2).

Statistical analysis

SPSS software 21.0 for Windows (SPSS Inc., Chicago, IL, USA) was used for all statistical analysis. Categorical variables are presented as counts and percentages. Continuous variables were evaluated for normal distribution assumption using the Kolmogorov-Smirnov and Shapiro-Wilks tests and were reported as mean plus standard deviation in brackets or median with interquartile range. Spearman Rank correlation test was used for correlation analysis. Receiver operating curves (ROC) were generated to define cut-off values (upper left corner of ROC as point of maximum sensitivity and specificity) of in-hospital death and in-hospital death/MI for presence of lower LVEF in the study population. In addition, univariate and multivariate binary logistic regression analysis were performed to investigate independent correlates of depressed LVEF. Variables with a p value <0.10 in univariate analysis were included in multivariate regression analysis. All p-values were two-sided and considered statistically significant when they were <0.05.

RESULTS

Totally 712 patients had undergone primary PCI with the diagnosis of AAMI. 422 patients were excluded from the analysis due to exclusion criteria (Flow diagram, Figure 1). According to the LVEF value, 132 patients were included in the group 1 (LVEF<50%), 158 patients were included in the group 2 (LVEF ≥50%). Clinical, demographic and laboratory properties of the study are summarized in Table 1. On comparison of the two groups, group 1 patients were older than group 2 patients (60.2±13.8 vs. 55.6±10.9, p=0.002). Frequency of patients with HT was higher in group 1 (56.8% vs. 43%, p=0.025). Neutrophil counts were higher in group 1 (9.78 [7.3-11.0] vs. 7.79 [5.9-9.1], p=0.021), but lymphocyte (2.34 [1.5-3.1] vs. 3.0 [1.7-3.7], p=0.002) and platelet counts were lower in group 1 (239.1 [190.5-285.0] vs. 253.5 [213.5-312.0], p=0.02). There were no differences between groups in other demographic and laboratory properties.

LV end-systolic diameter (LVESD) (39.1 [35.0-42.0] vs. 31.4 [29.0-34.0], p<0.001), LV end-diastolic diameter (LVEDD) (51.1 [47.3-54.0] vs. 47.3 [45.0-50.0], p<0.001), and left atrial diameter (LAD) (39.4±4.6 vs. 36.9±3.3, p<0.001) were higher in group 1.

Pre-PCI TIMI 0 flow (64.4% vs. 41.8%, p=0.002) in the first angiographic imaging frequency was higher in group 1, but TIMI 1 (15.2% vs. 25.9%), p=0.002), TIMI 2 (18.9% vs. 28.5%, p<0.001) and TIMI 3 (1.5% vs. 3.8%, p<0.001) flow frequency were lower in group 1. Also, the patients who had Killip 2 (40.9% vs. 12.7%, p<0.001) and 3 (9.8% vs. 2.5%, p<0.001) score at the admission were higher in group 1 (Table 1).

In-hospital death GRACE risk score (165.5±28.3 vs. 136.6±26.7, <0.001) and in-hospital death/MI GRACE risk score (228.0 [198.0-252.0] vs. 203.2 [183.5-216.2], <0.001) were higher in group 1. Correlation analysis of the in-hospital death and in-hospital death/MI risk scores and the LVEF were

performed by the Spearman Rank Correlation test and presented by using Scatter Dot analysis. A significant negative correlation was found between risk scores and LVEF (rho: -0.518, p<0.001 and rho: -0.440, p<0.001, respectively) (Table 2, Figure 2).

Table 1. Basal characteristics and demographics features in the study groups

Variables	EF< 50 (132)	EF≥ 50 (158)	p value
Age (years)	60.2±13.8	55.6±10.9	0.002
Male n (%)	104 (78.8)	121 (76.6)	0.674
BMI (kg/m2)	27.2 (24.3-29.4)	26.9 (24.2-28.8)	0.733
DM n (%)	51 (38.6)	48 (30.4)	0.171
HT n (%)	75 (56.8)	68 (43.0)	0.025
HPL n (%)	47 (35.6)	51 (32.3)	0.618
Current smoker n (%)	55 (41.7)	74 (46.8)	0,407
Glucose (mg/dL)	169.7 (114.0-179.0)	155.7 (113.8-170.0)	0.436
Creatinine (mg/dL)	0.91 (0.8-1.0)	0.9 (0.8-1.0)	0.297
HB (g/dL)	14.9 (13.7-15.9)	14.8 (13.9-16.0)	0.949
HTC (%)	44.2±4.7	43.5±4.1	0.342
Neutrophil ($x10^9$/L)	9.78 (7.3-11.0)	7.79 (5.9-9.1)	0.021
Lymphocyte ($x10^9$/L)	2.34 (1.5-3.1)	3.0 (1.7-3.7)	0.002
Platelet ($x10^9$/L)	239.1 (190.5-285.0)	253.5 (213.5-312.0)	0.020
RDW (%)	13.7 (12.8-14.3)	13.5 (12.8-14.0)	0.159
MPV (fL)	8.3±1.2	8.0±0.9	0.061
LVEF (%)	41.2 (38.0-45.0)	57.5 (54.8-61.0)	<0.001
LVEDD (mm)	51.1 (47.3-54.0)	47.3 (45.0-50.0)	<0.001
LVESD (mm)	39.1 (35.0-42.0)	31.4 (29.0-34.0)	<0.001
LAD (mm)	39.4±4.6	36.9±3.3	<0.001
Lesion localization n (%)			
Proximal	97 (73.5)	100 (63.3)	0.258
Medial	34 (25.8)	55 (34.8)	0.218
Distal	1 (0.8)	2 (1.3)	0.108
Pre-PCI TIMI flow n (%)			
TIMI 0	85 (64.4)	66 (41.8)	0.002
TIMI 1	20 (15.2)	41 (25.9)	0.002
TIMI 2	25 (18.9)	45 (28.5)	<0.001
TIMI 3	2 (1.5)	6 (3.8)	<0.001
In-hospital death score	165.5±28.3	136.6±26.7	<0.001
In-hospital death/MI score	228.0 (198.0-252.0)	203.2 (183.5-216.2)	<0.001
KILLIP score n (%)			
1	63 (47.7)	131 (82.9)	<0.001
2	54 (40.9)	20 (12.7)	<0.001
3	13 (9.8)	4 (2.5)	<0.001
4	2 (1.5)	3 (1.9)	<0.001

Variables are presented as n (%), mean (SD), or median (range); EF: ejection fraction; BMI: body mass index; DM: diabetes mellitus; HT: hypertension; HPL: hyperlipidemia; HB: hemoglobin; HTC: hematocrit; RDW: red cell distribution width; MPV: mean platelet volume; LVEF: left ventricular ejection fraction; LVEDD: left ventricular end diastolic diameter; LVESD: left ventricular end systolic diameter; LAD: left atrial diameter; PCI: percutaneous coronary intervention; TIMI: thrombolysis in myocardial infarction score; in-hospital death score: in-hospital 6 months mortality risk; in-hospital death/MI score: in-hospital 1 year mortality and recurrent myocardial infarction risk

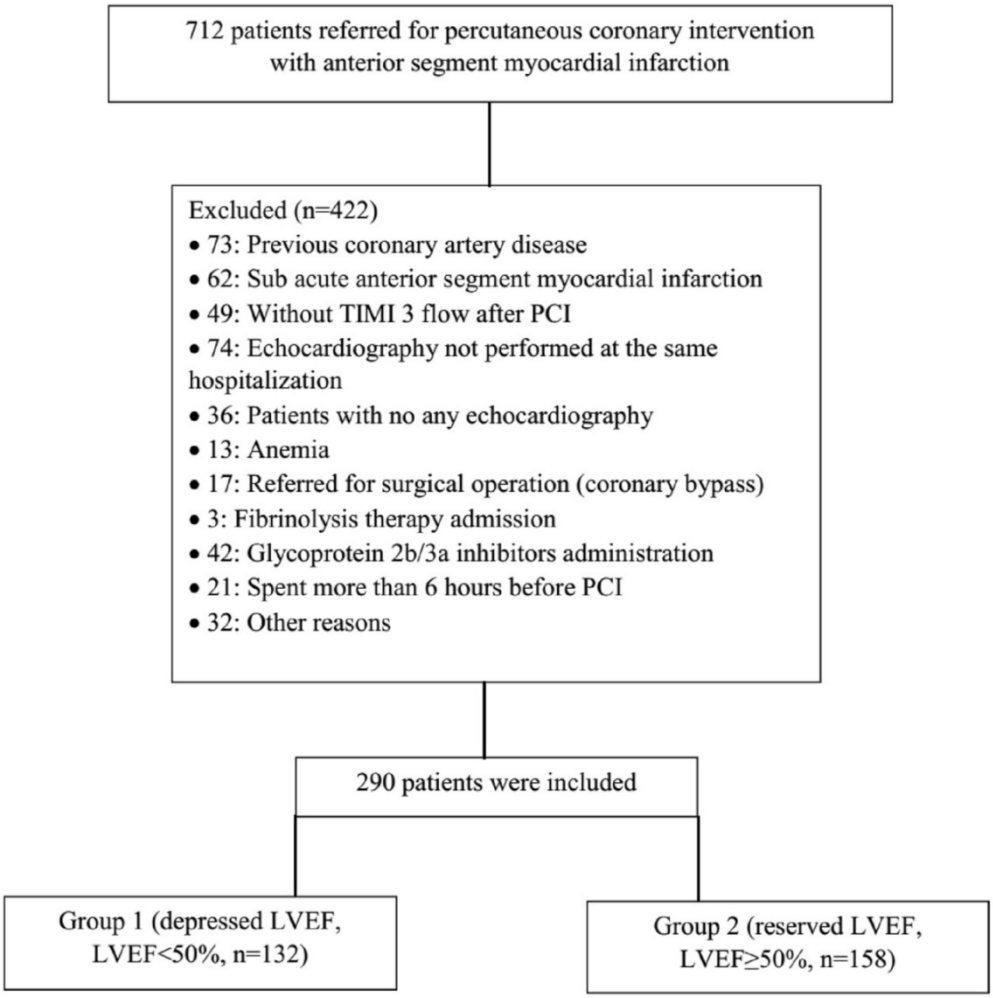

Figure 1. Flow diagram of patient selection.

Table 2. Spearman Rank Correlation (R) between LVEF and in-hospital death, in-hospital death/MI

Variables	R	p
LVEF and in-hospital death score	-0.518	<0.001
LVEF and in-hospital death/MI score	-0.440	<0.001

Abbreviations: LVEF: left ventricular ejection fraction; in-hospital death score: in-hospital 6 months mortality risk; in-hospital death/MI score: in-hospital 1 year mortality and recurrent myocardial infarction risk

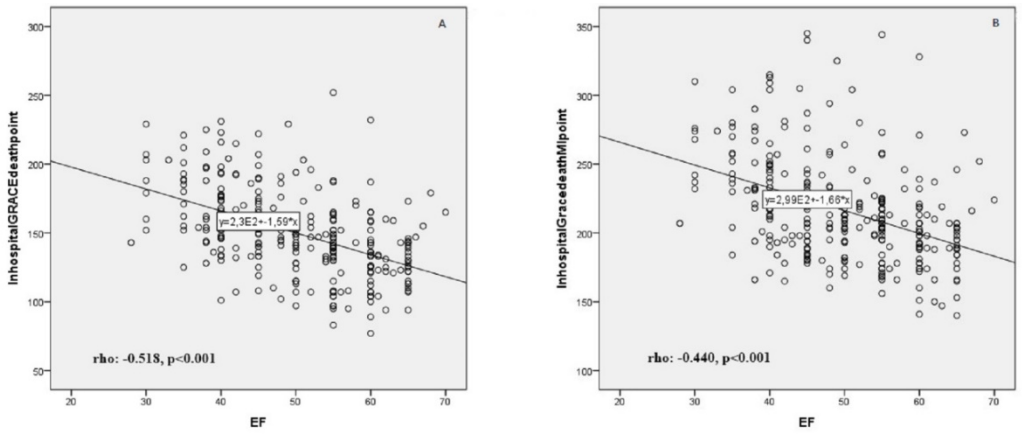

Figure 2. Correlation analysis of in-hospital death score and depressed EF (A), of in-hospital death/MI score and depressed EF (B) were performed by spearmen rank correlation analysis test and presented by using scatter dot analysis.

In ROC curve analysis, area under curve (AUC) of in-hospital death risk score was significantly higher compared with AUC of in-hospital death/MI risk score for discrimination of depressed LVEF in the study population (0.790 vs. 0.714; 95% CI: 0.738-0.842, p<0.001 vs. 0.654-0.775, p<0.001). Cut-off level of in-hospital death risk score >143 predicted depressed LVEF with sensitivity of 80% and specificity of 65%. Cut-off level of in-hospital death/MI risk score >196 predicted depressed LVEF with sensitivity of 78% and specificity of 60% (Figure 3).

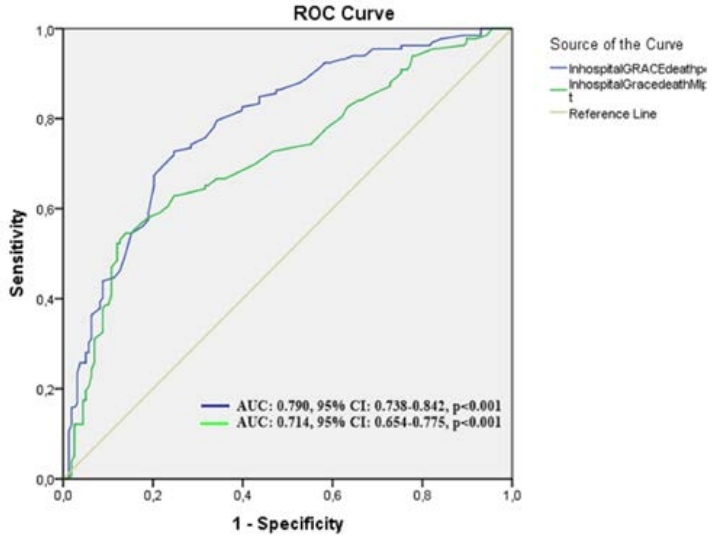

Figure 3. Receiver operating characteristic (ROC) curve analysis to identify depressed EF. The cut-off level of in-hospital death risk score was set at 143, the cut-off level of in-hospital death/MI risk score was set at 196.

Univariate and multivariate binary logistic regression analysis were performed to investigate independent correlates of depressed LVEF in the study population. In the multivariate model, age, hematocrit, lymphocyte, LVEDD, LVESD, in-hospital death risk score (OR: 1.10; 95% CI: 1.05-1.15; p<0.001), and in-hospital death/MI risk score (OR: 0.96; 95% CI: 0.92-1.00; p=0.032) were found to be independently predictors of depressed LVEF (Table 3).

Table 3. The predictors of low EF in binary logistic regression analysis.

Variables	Unadjusted OR (95 % CI)	p	Adjusted OR (95 % CI)	p
Age	1.03 (1.01-1.05)	0.002	0.94 (0.89-0.99)	0.015
HT	1.74 (1.9-2.78)	0.02	1.70 (0.72-3.99)	0.224
KILLIP score	2.76 (1.83-4.18)	<0.001	1.30 (0.45-3.78)	0.633
Pre-PCI TIMI flow	0.63 (0.48-0.82)	<0.001	0.79 (0.50-1.25)	0.313
In-hospital death score	1.02 (1.02-1.03)	<0.001	1.10 (1.05-1.15)	<0.001
In-hospital death/MI score	1.02 (1.02-1.03)	<0.001	0.96 (0.92-1.00)	0.032
HTC	1.06 (0.99-1.11)	0.053	1.12 (1.07-1.24)	0.023
MPV	1.23 (0.99-1.53)	0.059	1.09 (0.75-1.57)	0.663
Neutrophil	1.00 (1.00-1.00)	0.002	1.00 (1.00-1.00)	0.486
Lymphocyte	1.00 (1.00-1.00)	<0.001	1.00 (0.99-1.01)	0.001
LVEDD	1.15 (1.09-1.22)	<0.001	0.74 (0.63-0.88)	<0.001
LVESD	1.45 (1.33-1.58)	<0.001	1.87 (1.55-2.24)	<0.001
LAD	1.18 (1.10-1.25)	<0.001	1.00 (0.90-1.12)	0.94

EF: ejection fraction; OR: odds ratio; CI: confidence interval; HT: hypertension; PCI: percutaneous coronary intervention; TIMI: thrombolysis in myocardial infarction score; HTC: hematocrit; MPV: mean platelet volume; LVEDD: left ventricular end diastolic diameter; LVESD: left ventricular end systolic diameter; LAD: left atrial diameter; in-hospital death score: in-hospital 6 months mortality risk; in-hospital death/MI score: in-hospital 1 year mortality and recurrent myocardial infarction risk.

DISCUSSION

In this study, we evaluated the ability of GRACE risk score to predict depressed LVEF and determined the correlation between in-hospital death GRACE risk score, in-hospital death/MI GRACE risk score and depressed LVEF in AAMI patients. Also, these risk scores were found independently predictors of depressed LVEF value in multivariate binary logistic regression analysis.

The most common etiology of heart failure with reduced EF in the developed countries is ischemic heart disease, which is associated with more than 60% of diagnoses[10]. ACS associated with LV systolic dysfunction is a condition related poor prognosis[11, 12]. The degree of LV systolic dysfunction is a major determinant of long-term outcomes in ACS[13]. Among patients with ACS, depressed LVEF is associated with increased one year mortality or hospitalization for heart failure (HF)[14]. After an AMI, the patient is in a risk stage of developing HF. Atrial fibrillation, multi-vessel coronary diseases, hypertension, chronic renal diseases, diabetes mellitus, and anemia raise the risk for depressed LVEF in ACS[15-17].

Risk stratification of ACS patients to predict HF is useful to focus the in-hospital and outpatient coronary care. ACC/AHA and ESC guidelines emphasize the importance of estimating the level of risk and the use of risk scores[18]. GRACE risk score, which is based on clinical features, ECG changes, and biomarkers (troponin and creatinine) is one of the most frequently used risk score and has shown its utility in the setting of ACS, to predict in-hospital and follow-up mortality and re-infarction[19, 20]. The usefulness of GRACE risk score to predict heart failure was investigated in a previous study, but in this study the population consisted of patients with ST segment elevation ACS and non-ST segment elevation ACS[21]. Contrary to that study, our study population consisted only of anterior ST segment elevation ACS patients. In-hospital death and in-hospital death/MI risk score were higher in the depressed EF group. There were negative correlation between in-hospital death and in-hospital death/MI risk score and depressed EF. In predicting depressed EF in the first admission and after primary PCI of AAMI, the best discriminative value of in-hospital death score was 143, providing sensitivity of 80% and specificity of 65%, and in-

hospital death/MI score was 196, providing sensitivity of 78% and specificity of 60%. Also, we showed that both in-hospital death and in-hospital death/MI risk score were independent predictors of depressed LVEF.

It is very well known that several traditional risk factors such as hypertension, aging, diabetes mellitus, smoking, physical inactivity were identified in the general population and included in CV risk estimators[22]. A study concluded that age was an independent predictor of LV systolic dysfunction after ACS[23]. In our study, we found that patients in the depressed EF group were older, as well as more often hypertensive compare with patients in the preserved EF group, similarly, in line with other studies.

Killip class shows the degree of heart failure after AMI[24]. This score is helpful in terms of orientation treatment in the acute phase of AMI. A previous study showed that Killip class was an important prognostic factor for depressed LVEF[25]. Similarly, the patients in the depressed LVEF group had higher Killip score at admission in our study.

Among the types of ACS, STEMI was the strongest and most important prognostic factor for LVEF[25]. Diabetes mellitus, hypertension, anemia, prior heart failure, atrial fibrillation, other ACS forms were some of the other risk factors for predicting LVEF[21]. In the previous studies, investigators were focused on heart failure on admission, mortality after discharge, hospitalization, etc. Additionally, in most of the studies, the patients had been admitted with all forms of ACS. But, in our study, we kept our exclusion criteria wide, focused only on the GRACE risk score for predicting LVEF value and just included early-onset AAMI patients. All patients had TIMI III flow after primary PCI. We excluded the causes that could affect LVEF, except demographic characteristics. Therefore, we believe that our study is different from previous ones.

Some limitations of the present study should be taken into consideration. This study is a retrospective study of unicenter data. The data was lack of patients' treatment, socioeconomic and educational status. But, all the patients were under standard AMI treatment including acetylsalicylic acid, clopidogrel, unfractioned intravenous/low molecular weight subcutaneous heparin, captopril, and statin as a routine. Additionally, there were not any data about biomarkers with utility in depressed LVEF and HF (for example; natriuretic peptides). Another limitation is that GRACE score was developed and validated based on data from 1999 through 2003. Rate of PCI and use of clopidogrel were nearly 30% when this score was introduced. However, these treatments are nowadays used in approximately 90% of ACS patients.

LVEF is the most important indicator of HF. Most of the studies were focused on HF. But, we investigated and focused directly LVEF. Our findings suggest that in-hospital death and in-hospital death/MI risk scores were associated with depressed EF in AAMI patients and had a positive correlation with LVEF. Based on LVEF estimation, in-hospital death and in-hospital death/MI risk scores had independent predictive value with a good sensitivity and specificity. These significant findings could guide for clinical practice.

Yazar Katkıları: Çalışma konsepti/Tasarımı: VT, NK; Veri toplama: VT, NK; Veri analizi ve yorumlama: VT, NK; Yazı taslağı: VT, NK; İçeriğin eleştirel incelenmesi: ÜG; Son onay ve sorumluluk: VT, NK, AYK, ÜG; Teknik ve malzeme desteği: -; Süpervizyon: ÜG; Fon sağlama (mevcut ise): yok.
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