Importance of EuroSCORE-II in the Development of Acute Ischemic Heart Failure After Acute Anterior ST Elevation Myocardial Infarction

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ABSTRACT

Introduction: We aimed to evaluate the importance of the EuroSCORE-II (ES-II) risk-scoring system in predicting the development of acute ischemic heart failure (AIHF) after acute anterior ST segment elevation myocardial infarction (A-STEMI).

Patients and Methods: A total of 261 patients (206 male; mean age, 63.5 ± 14.2 years) admitted to our centre with A-STEMI between April 2012 and January 2013 were included in the study. The patients were divided into two groups according to their clinical findings and were prospectively followed during the hospitalisation period for the development of cardiac morbidity and mortality. These groups were the AIHF group (n= 69) and non-AIHF group (n= 192). For the diagnosis of AIHF, we considered the recommendations of the European Society of Cardiology Guidelines on Heart Failure and the Framingham criteria.

Results: According to our results, the morbidity and mortality rates were higher in the AIHF group. Symptom-to-balloon time, ejection fraction (EF), glomerular filtration rate (gfr), no-reflow phenomenon, left main coronary artery disease and ES-II, which are the predictors of the development of AIHF, were determined via logistic regression analysis. ROC analysis revealed that symptom-to-balloon time > 209.5 min, EF < 36.5%, gfr < 68.5 mg/dL/1.73 m^2 and ES-II > 4.9% were the cut-off values in the development of AIHF.

Conclusion: Patients with A-STEMI complicated by AIHF have significantly high mortality rates. With the pre-estimation of the development of AIHF, complication rates can be reduced. For this purpose, ES-II score > 4.9% can be used as a predictor of AIHF after A-STEMI.

Key Words: Coronary artery disease; ST elevation myocardial infarction; EuroSCORE-II; acute ischemic heart failure

Akut Anterior Miyokart İnfarktüsü Sonrası Akut İskemik Kalp Yetersizliği Gelişimini Öngörmede EuroSCORE-II'nin Önemi

ÖZET

Giriş: Akut anterior ST yükselmeli miyokart infarktüsü (A-STEMİ) sonrası akut iskemik kalp yetersizliği (AİKY) gelişimini öngörmede EuroSCORE-II skorlama sisteminin önemini değerlendirmeyi amaçladık.

Hastalar ve Yöntem: Çalışmaya Nisan 2012-Ocak 2013 tarihleri arasında merkezimize A-STEMİ nedeniyle kabul edilen 261 hasta (206 erkek, ortalama yaş 63.5 ± 14.2) dahil edildi. Hastalar klinik bulgularına göre iki farklı gruba ayrıldı ve hastanede yatış periyodu süresince kardiyak morbidite ve mortalite gelişimi bakımından prospektif olarak takip edildi. Bu gruplar AİKY grubu ((n= 69) ve non-AİKY grupları (n= 192) idi. AİKY tanısı koyabilmek için Avrupa Kardiyoloji Cemiyeti kalp yetersizliği klavuzu ve Framingham kriterleri göz önünde bulunduruldu.

Bulgular: Çalışmamız sonuçlarına göre AİKY grubunda morbidite ve mortalite daha yüksekti. Regresyon analizleri sonuçlarına göre semptom-balon zamanı, ejeksiyon fraksiyonu, no-reflow fenomeni, sol ana koroner hastalığı, üç damar koroner arter hastalığı, SYNTAX skoru ve ES-II'nin AİKY gelişiminin prediktörleri oldukları belirlendi. ROC analizlerinin sonuçlarına gore semptom-kapı zamanının > 229.5 dakika, EF < %36.5, SYNTAX skoru > 27.75 ve EsII > %4.9 değerlerinin AİKY gelişimini öngördürebilecek sınır değerler oldukları belirlendi.

Sonuç: AİKY gelişen A-STEMİ'li hastalarda mortalite oranları artmaktadır. AİKY gelişiminin önceden öngörülmesi kardiyak komplikasyonları azaltabilir. Bu amaçla Es-II'nin > %4.9 olması A-STEMİ sonrası AİKY gelişimim öngördürücüsü olarak kullanılabilir.

Anahtar Kelimeler: Koroner arter hastalığı; acut anterior ST yükselmeli miyokart infarktüsü; EuroSCORE-II; akut iskemik kalp yetersizliği

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INTRODUCTION

Coronary artery disease (CAD) is one of the leading causes of mortality and morbidity[1,2]. The main reason for the development of CAD is atherosclerosis. Atherosclerotic plaques slowly increase over many years, resulting in luminal narrowing and decreased blood flow to the heart muscles. The formation of thrombus may block blood flow in coronary arteries causing ST segment elevation myocardial infarction (STEMI)[3-6]. The magnitude of myocardial necrosis after STEMI depends on the localization of the lesion in the infarct-related coronary artery. Cardiac functions directly affect the size of the necrotic tissues[7,8]. The greater the area affected, higher is the possibility of the development of a heart failure (HF)[9,10]. HF is defined as an abnormality in cardiac structure or function leading to failure of the heart to deliver oxygen at a rate commensurate with the requirements of the tissues, despite normal or increased filling pressures[7]. In total, 60% of the patients admitted to the hospital with HF had significant CAD[11]. Patients with HF have a worse prognosis if diagnosed with CAD. Patients with STEMI complicated by acute ischemic heart failure (AIHF) have significantly increased mortality rates compared with those without HF[12,13].

Various risk-scoring systems have been established in patients with acute coronary syndrome (ACS) to predict the mortality and morbidity rates before interventional operations. One of these systems is EuroSCORE (ES). ES was established to evaluate pre-operative mortality and morbidity rates in patients undergoing coronary artery bypass grafting (CABG)[14]. It was later determined that ES remained incapable to detect high-risk patients and, hence, logistic-ES was established[15,16]. It was further revealed that this score could be applied in detecting risks in patients undergoing percutaneous coronary intervention, in addition to the patients undergoing CABG[17,18]. However, it was found that logistic-ES predicts higher mortality than it actually is and, hence, EuroSCORE II (ES-II) was established[19-22].

There are no comprehensive trials in the literature for evaluating the relationship between ES-II and cardiac complication rates after acute anterior STEMI (A-STEMI). In this study, we aimed to evaluate the importance of EuroSCORE-II risk-scoring system in predicting the development of AIHF after A-STEMI.

PATIENTS and METHODS

Study Design

This observational study was conducted from April 2012 to February 2013. We evaluated 307 patients admitted to our centre with STEMI. Among those, 261 patients who met the criteria for admission and had acute A-STEMI for the first time were included in the study. The patients who had non-ST segment elevation myocardial infarction (non-STEMI), previous CAD and HF history before admission to the hospital were not included in the study.

The patients who had infection, myocarditis or heart muscle disease, chronic inflammatory disease, chronic lung or liver disease and severe valvular heart diseases were not included in the study. The patients whose symptom onset time was unknown or symptom time was longer than 12 h were excluded from the study. Data regarding the time from symptom onset to hospital arrival were obtained from either the patients or their relatives. Symptom-to-door time (StD) and door-to-balloon (DtB) time were calculated. Patients who did not agree to join the study or suffered from mental disorders were excluded from the study.

ST-segment elevation in acute anterior myocardial infarction, measured at the J point, was found to be in two contiguous leads. It was ≥ 0.25 mV in men < 40 years of age, ≥ 0.2 mV in men > 40 years of age, ≥ 0.15 mV in women in leads V2-V3 and/or ≥ 0.1 mV in other leads (in the absence of left ventricular hypertrophy or left bundle branch block).

Study Protocol and Medical Treatment

Patients' invasive treatment methods applied during primary percutaneous coronary intervention (PPCI), balloon and stent diameters and lengths were recorded. Blood samples were drawn from the vein of each subject immediately after presentation to the emergency department. Cardiac enzymes, liver function, kidney function, complete blood count and thyroid function were determined using the blood samples. Peak cardiac enzyme levels were identified by daily blood samples.

For medical treatment, the patients received 300 mg acetylsalicylic acid peroral therapy upon hospital admission. For maintenance therapy, 100 mg acetylsalicylic acid/day was continued. Patients aged > 75 years received 300 mg clopidogrel loading dose, whereas those aged < 75 years received 600 mg clopidogrel loading dose. Maintenance therapy of 75 mg/day was administered. All patients in the emergency department received 100 IU/kg intravenous (IV) bolus dose of unfractionated heparin. Additional dose was administered during coronary angiography when necessary. As a maintenance therapy, weight-adjusted low molecular weight heparin was subcutaneously administered to the patients. After coronary angiography, beta-blockers and angiotensin converting enzyme inhibitor/angiotensin receptor blocker therapy were started with a low dose and titrated to a suitable dose compatible with the hemodynamic stability of the patients. All patients in the intensive care unit were started on statin therapy. PCI dose of tirofiban infusion was administered to the patients with coronary thrombi. IV diuretics were given to the patients with signs and symptoms of AIHF. IV inotropes were started in patients with haemodynamic instability.

The patients were prospectively followed during the period of hospital stay. Informed consent was obtained from all subjects, and the investigation conforms to the principles outlined in the Declaration of Helsinki. The study protocol was approved by the Ethics Committee.

Acute Ischemic Heart Failure

The signs and symptoms of HF were evaluated according to the Framingham Criteria [Major criteria: paroxysmal nocturnal dyspnoea, neck vein distention, rales, radiographic cardiomegaly (increased heart size on chest radiography), acute pulmonary oedema, S3 gallop, increased central venous pressure (> 16 cm H_2O at right atrium), hepatojugular reflux and weight loss (> 4.5 kg in 5 days in response to treatment). Minor criteria: bilateral ankle oedema, nocturnal cough, dyspnoea on ordinary exertion, hepatomegaly, pleural effusion, decrease in vital capacity by one-third from maximum recorded and tachycardia (heart rate > 120 beats/min)][23]. The presence of two major or one major plus two minor Framingham Criteria was considered for the diagnosis of AIHF. Besides these criteria, Killip class 2-3-4 patients were diagnosed with AIHF after A-STEMI at admission to the hospital or during hospitalisation periods.

EuroSCORE-II

ES-II calculations (www.euroscore.org) of patients were performed with the help of a computer-assisted software program. Variables, such as age, gender, peripheral arterial disease, physical inactivity, neurological dysfunction, patient's history of past cardiac surgeries, creatinine level, history of endocarditis, critical preoperative state, diabetes wherein insulin is used, unstable angina, left ventricular dysfunction, history of myocardial infarction, pulmonary artery pressure, past thoracic aorta operations, post-infarction ventricular septal rupture, diabetes mellitus and available functional capacity, were entered into suitable areas in the software. ES-II scores, which were calculated based on these variables, were employed for study analyses.

Coronary Angiography and SYNTAX Score

SYNTAX score (STXs) was calculated to evaluate patients' CAD prevalence in our study. STXs was calculated using a dedicated software (version 2.11) (www.syntaxscore.org). Each coronary lesion producing ≥ 50% luminal obstruction in vessels with a diameter ≥ 1.5 mm was separately scored and added to provide the vessel STXs and then summed to provide the overall patient STXs. All coronary angiograms were scored by two experienced investigators who were blinded to the clinical data.

Echocardiography

Echocardiographic evaluations were performed using the Vivid 3 ultrasound system (GE Healthcare, Wauwatosa, WI, US) with a 2.5-MHz transducer. Echocardiographic evaluations were performed in all patients during admission to the hospital in the emergency department before coronary angiography. In addition to the basic measurements, left ventricular ejection fractions (EF) of patients with A-STEMI were calculated using the modified Simpson method. The average of three different measurements was used for this study.

Statistical Methods

Analysis was performed using the SPSS 17.0 software (SPSS, Chicago, Illinois, USA). Continuous variables were expressed as mean ± standard deviation, and categorical variables were expressed as percentages. Comparison of categorical variables between groups was performed using the chi-square test. Independent samples t-test was used for comparing continuous variables of multiple groups. To identify the correlations between AIHF and clinical or laboratory parameters, a univariate analysis was performed. A multivariate logistic regression analysis was used to assess the correlation among the parameters, whose statistical significance was demonstrated on a univariate analysis at a level of $p< 0.05$ and through previously well-known risk factors. Models were developed with stepwise techniques, for which the results were expressed as odds ratios (OR) with 95% confidence intervals (CI). For ROC analysis, the patients were divided into two groups: patients with and without AIHF. The area under the ROC curve analysis; cut-off values for ES-II, STXs, StD time and EF; 95% CI and sensitivity and specificity values were determined. The calculated p-values < 0.05 were considered statistically significant.

RESULTS

General Characteristics and Laboratory Results

We evaluated 307 patients admitted to our centre with A-STEMI. A total of 261 patients (176 male; mean age: 64.8 ± 14.2 years) who met the criteria were included in this study. There were no differences between groups according to the age, heart rate and DtB time means. StD time ($p= 0.021$), troponin-I ($p= 0.02$), postprandial blood glucose ($p= 0.013$) and creatinine levels ($p< 0.001$) were significantly higher in the AIHF group. Systolic and diastolic blood pressure means were significantly lower in the AIHF group. The average length of stay in the intensive care unit was 4.0 (± 2.5) days, whereas the length of total hospital stay was 6.9 (± 3.4) days. However, in the AIHF group, it was observed that the length of stay in the intensive care unit was 5.2 (± 4.5) days and the length of total hospital stay was 8.2 (± 5.0) days (Table 1).

Coronary Angiography and Echocardiography

The number of patients with left main ($p= 0.015$) or three-vessel ($p= 0.043$) CAD was significantly higher in the AIHF group. There were no differences between groups with respect to the number of implanted drug eluting stents (DES), stent diameter, stent length and left dominance. The ratio of no-reflow was significantly higher in the AIHF group ($p= 0.008$). The number of patients undergoing CABG after PPCI was higher in the AIHF group ($p= 0.013$). EF mean of the AIHF group was 33.2 (± 10.6)% and non-AIHF group was 39.5 (± 7.6)% ($p< 0.001$) (Table 1).

Cardiac Complications and AIHF

The number of patients with cardiogenic shock and diuretic treatment rates were significantly higher in the AIHF group. Furthermore, ventricular tachycardia, cardiac arrest and mortality rates were higher in the AIHF group than the other group (Table 1). Thirteen patients died during their hospital stay (4.2%), and all of them belonged to the AIHF group ($p< 0.001$).

In multivariate logistic regression analysis, ES-II [p< 0.001; OR, 7.2 (95% CI, 3.3-22.5)], STXs [p= 0.048; OR, 1.9 (95% CI, 0.7-4.8)], StD time [p= 0.033; OR, 2.3 (95% CI, 1.0-4.8)], LMCA lesion [p= 0.030; OR, 2.6 (95% CI, 0.8-11.5)], three-vessel CAD [p= 0.042; OR, 1.9 (95% CI, 0.8-3.1)], EF [p= 0.009; OR, 2.7 (95% CI, 1.3-5.6)] and no-reflow phenomenon [p= 0.050; OR, 2.1 (95% CI, 0.9-4.8)] were found to be independent predictors of AIHF (Table 2).

In the ROC analysis for the prediction of AIHF development, area under curve (AUC) was 0.850 and cut-off ES-II value was 4.90% [95% CI, 0.799-0.901; sensitivity, 61% and specificity, 89%] (Figure 1, Table 3). In the case of STXs before PPCI for the prediction of AIHF development, AUC was 0.642 and cut-off value was 27.75 [95% CI, 0.535-0.699; sensitivity, 47% and specificity, 65%]. In the case of EF before PPCI for the prediction of AIHF development, AUC was 0.715 and cut-off value was 36.5% [95% CI, 0.636-0.795; sensitivity, 63% and specificity, 73%]. For StD time for the prediction of AIHF development, AUC was 0.613 and cut-off value was 229.5 min [95% CI, 0.536-0.689; sensitivity, 72% and specificity, 55%] (Table 3).

DISCUSSION

In this study, we investigated the relationship between AIHF development and ES-II after A-STEMI. The patients were divided into two groups according to their clinical findings (AIHF or non-AIHF) and were prospectively followed during hospitalisation period for the development of cardiac morbidity and mortality.

The etiological factor is CAD in approximately 60% of the patients hospitalised because of HF[24,25]. Cardiac complications and mortality rates increase with increasing severity of CAD[26,27]. Approximately 10% of patients with ACS develop AIHF during hospitalisation period[28,29]. Among the evaluated A-STEMI patients in our study, a total of 69 patients (26.4%) admitted to the hospital and/or in hospital follow-up showed signs and symptoms of AIHF. The rate of AIHF development in A-STEMI patients in the present study was higher than those in previous ACS studies. There were unstable angina pectoris, non-STEMI, STEMI and sudden cardiac death in the ACS group. Rates of complications among the groups were different. A-STEMI occurs as a result of occlusion of left anterior descending artery (LAD), the largest coronary artery. All ACS do not occur because of the occlusion of the same coronary arteries. Little side branch lesions other than arterial occlusions may lead to ACS. The patients who only had A-STEMI were included in our study. The reason for higher AIHF development rates when compared with those of other ACS items is that LAD was the infarct-related coronary artery for infarction in all the patients.

When AIHF occurs after STEMI, morbidity and mortality rates increase significantly[30,31]. The present study showed that cardiogenic complications, such as cardiogenic shock, ventricular fibrillation, cardiac arrest, high degree atrioventricular block, mechanical complication and no-reflow phenomenon were significantly higher in the AIHF group. The AIHF group patients needed diuretic treatment more frequently. Intensive care unit and total hospital stays were longer in the AIHF group (Table 1). The reason for the extension of these periods could be the higher rate of cardiac complications in the AIHF group. As a result of our analysis, it was found that there were no significant differences between the two groups in terms of general characteristics. However, blood pressure and EF were lower in the AIHF group and creatinine, troponin-I, blood glucose and total ischemic time were higher in patients with AIHF. In previous studies, in-hospital mortality rates due to AIHF have been reported to be 4%-7%[32,33]. In our study, a total of 13 patients died due to AIHF (4.6%). All the patients who died (p< 0.001) belonged to the AIHF group.

It is important to know the factors that can predict AIHF development in patients with A-STEMI due to increased complication rates. ES-II is a risk score calculated before cardiac surgeries in order to determine the patients' risk levels. Capodanno et al. found that ES-II had better logistics than ES in predicting post PPCI 30-day mortality. Additionally, it is suggested to use ES-II in determining pre-PPCI risk[16-19]. ES-II values of patients in the AIHF group were higher than those of patients without AIHF (p< 0.001). Multivariate regression analysis was applied to variables with p< 0.05 in univariate regression analysis. ES-II was determined to be the most critical variable that can predict post A-STEMI (Table 2). ROC analysis was applied to numerical variables that were responsible for AIHF development. In ROC analysis performed for ES-II, cut-off value that could predict AIHF development was detected as 4.90% [AUC, 0.850 (95% CI, 0.799-0.901); sensitivity, 61% and specificity, 89%]. One of the predictors for post A-STEMI AIHF development other than ES-II was STXs. STXs, which was one of the indicators of the prevalence of CAD, was significantly higher in the AIHF group. There is an important relationship between the long-term survival rates of patients with HF and the severity of CAD[25,26]. STX's cut-off value that predicts AIHF development in patients with A-STEMI was determined as 27.75. It was observed that StD duration surpassing 229.5 min was one of the predictors for AIHF development. The AIHF predicting value for EF, which was calculated before PPCI with modified Simpson's method, was < 36.5% (Table 3). Our centre is a third-tier health institution, in which there is an angiography laboratory that is always open. Revascularization can be applied to patients rapidly after STEMI. This could be the reason why DtB duration was not among the variables that predict AIHF development in this study. The average DtB values were similar in the AIHF and non-AIHF groups. It was determined that the main delay before revascularization was related to StD time.

In conclusion, ES-II was seen to be the best predictor of AIHF development as a result of multivariate regression and ROC analyses (Table 2,3). Because parameters used to calculate ES-II are linked with clinical variables, it is comprehensible that ES-II's strength of predicting AIHF development is high. It can emerge due disorders in vitals in addition to AIHF coronary artery disease in patients with ACS. In particular, the level of kidney functions in patients with ACS is an important factor that can increase the

Table 1. Acute ischemic heart failure (AIHF) and non-AIHF patients' general characteristics and differences between groups

Variable	AIHF (n= 69)	Non-AIHF (n= 192)	p value
Age, years	66.2 (± 12.3)	64.1 (± 14.0)	0.187
Systolic blood pressure, mmHg	120 (± 34.5)	145.3 (± 21.2)	< 0.001
Diastolic blood pressure, mmHg	76.5 (± 24.9)	89.5 (± 13.3)	< 0.001
Heart Rate/min	86.6 (± 25.1)	84.3 (± 19.2)	0.424
Troponin-I, ng/dL	94.4 (± 86.3)	70.1 (± 67.7)	0.020
Postprandial blood glucose, mg/dL	167.1 (± 75.5)	143.3 (± 64.4)	0.013
Creatinine, mg/dL	1.11 (± 0.45)	0.88 (± 0.28)	< 0.001
Haemoglobin, g/dL	13.5 (± 2.1)	14.1 (± 1.7)	0.119
Platelets, 10^3/μL	232.5 (± 56.1)	240.3 (± 63.2)	0.367
EuroSCORE-II, %	7.69 (± 6.57)	2.69 (± 2.79)	< 0.001
SYNTAX score pre PPCI	27.6 (± 7.6)	24.8 (± 7.9)	0.011
Ejection fraction, %	33.2 (± 10.6)	39.5 (± 7.6)	< 0.001
Door-to-balloon time, min	20.7 (± 5.2)	21.7 (± 5.6)	0.191
Symptom-to-door time, min	270.3 (± 273.4)	221.6 (± 258.7)	0.021
Intensive care unit stay, days	5.2 (± 4.5)	3.5 (± 1.1)	< 0.001
Total hospital stay, days	8.2 (± 5.0)	6.3 (± 2.5)	< 0.001
No-Reflow, n, %	21 (30.4%)	30 (15.6%)	0.008
Cardiogenic shock (Killip class 4), n, %	23 (33.3%)	0 (0%)	< 0.001
Ventricular tachycardia, n, %	22 (31.9%)	10 (5.9%)	< 0.001
Atrioventricular block (High degree), n, %	5 (7.2%)	2 (1.0%)	0.015
Arrest, n, %	12 (17.4%)	4 (2.1%)	< 0.001
Mortality, n, %	13 (18.8%)	0 (0%)	< 0.001
CABG, n, %	8 (11.6%)	12 (6.3%)	0.013
Mechanical complication, n, %	6 (8.7%)	0 (0%)	< 0.001
Atrial fibrillation (Post-MI), n, %	5 (7.2%)	12 (6.3%)	0.483
Angiographic findings, n, %			
Left main coronary artery	9 (13.0%)	8 (4.2%)	0.015
One vessel disease	25 (36.2%)	86 (44.8%)	0.027
Two vessel disease	23 (33.3%)	67 (34.9%)	0.468
Three vessel disease	21 (30.4%)	39 (20.3%)	0.043
Left dominance, n, %	10 (14.5%)	21 (10.9%)	0.549
Drug eluting Stents, n, %	12 (17.4%)	38 (19.8%)	0.863
Total stent diameter, mm	3.11 (± 0.63)	3.08 (± 0.58)	0.750
Total stent length, mm	23.1 (± 7.5)	25.4 (± 11.9)	0.084
Medical Treatment, n, %			
Acetylsalicylic acid	67 (97.1%)	188 (97.9%)	0.501
Beta-blockers	57 (78.1%)	331 (93.2%)	< 0.001
Clopidogrel	66 (90.4%)	188 (97.9%)	0.385
Statins	63 (91.3%)	180 (93.2%)	0.006
RAAS inhibitors	56 (81.2%)	179 (90.4%)	0.045
Diuretic (IV)	69 (100%)	0	< 0.001
Diuretic (oral)	22 (31.9%)	8 (4.2%)	< 0.001
Glycoprotein IIb–IIIa inhibitor infusion	10 (14.5%)	23 (12.0%)	0.363

AIHF: Acute ischemic heart failure, CABG: Coronary artery by-pass surgery, IV: Intravenous, MI: Myocardial infarction, PPCI: Primary percutaneous coronary intervention, RAAS: Renin angiotensin aldosterone system.

Table 2. Univariate and multivariate analysis of risk factors determining acute ischemic heart failure

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p values	OR (95% CI)	p values
Age	1.1 (0.8-1.5)	0.048	0.7 (0.2-2.3)	0.552
No-reflow	2.3 (1.1-3.2)	< 0.001	2.1 (0.9-4.8)	0.050
Ejection fraction	7.3 (1.5-17.1)	< 0.001	2.7 (1.3-5.6)	0.009
Door-to-balloon time	1.0 (0.7-2.3)	0.045	0.8 (0.2-1.2)	0.145
Symptom-to-door time	3.2 (1.8-9.4)	< 0.001	2.3 (1.0-4.8)	0.033
LMCA lesion	2.7 (1.2-9.2)	< 0.001	2.6 (0.8-11.5)	0.030
Three vessel coronary artery disease	1.5 (0.8-4.3)	0.004	1.9 (0.8-3.1)	0.042
EuroSCORE-II	14.9 (6.3-20.4)	< 0.001	7.2 (3.3-22.5)	< 0.001
SYNTAX score	2.1 (1.7-7.6)	< 0.001	1.9 (0.7-4.8)	0.048
Drug Eluting Stent	0.9 (0.3-1.6)	0.242	-	-
Troponin-T	0.8 (0.2-1.3)	0.134	-	-
Haemoglobin	0.9 (0.1-1.5)	0.123	-	-
Creatinine	1.0 (0.4-1.6)	0.082	1.0 (0.4-1.7)	0.582

OR: Odds ratio, LMCA: Left main coronary artery, CI: confidence interval.

Table 3. ROC analysis of the acute ischemic heart failure predictors

Variables	AUC	95% Confidence interval		Cut-off	Sensitivity	Specificity
EuroSCORE-II	0.850	0.799	0.901	4.90	61%	89%
SYNTAX before PPCI	0.642	0.535	0.699	27.75	47%	65%
Ejection fraction	0.715	0.636	0.795	36.5%	63%	73%
Symptom-to-door time	0.613	0.536	0.689	229.5 min	72%	55%

AUC: Area under curve, PPCI: Primary percutaneous coronary intervention.

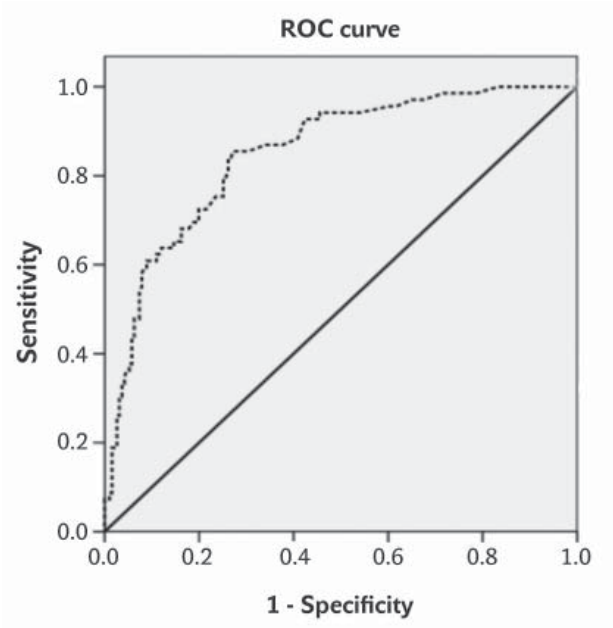

Figure 1. Receiver operator characteristic (ROC) curve of the acute ischemic heart failure (AIHF) patients. In the ROC analysis for the prediction of AIHF development, area under curve (AUC) was 0.850 and cut-off EuroSCORE-II value was 4.90% (95% CI, 0.799–0.901; sensitivity, 61% and specificity: 89%).

rate of complication. When parameters used to calculate ES-II are examined, it can be observed that patients' cerebral, inspiratory, echocardiographic and clinical states are evaluated. Changes in all these parameters can both raise ES-II values and ease AIHF development. Because of these reasons, we think that increased ES-II after A-STEMI may predict AIHF development.

Study Limitations

We analysed only the hospitalisation period of our patients in this study. With a long-term follow-up period, more accurate results could be obtained. Besides, we believe that it would be useful to determine the size of necrotic tissue with positron emission tomography, magnetic resonance imaging or scintigraphic methods and to assess the relationship of that with the ES-II.

CONFLICT of INTEREST

There are no conflict of interest for all authors.

AUTHORSHIP CONTRIBUTIONS

Concept/Design: İG, Aİ, MB
Analysis/Interpretation: İG, MZ, BSY
Data Acquisition: İG, MZ
Writing: İG, AÇA, BSY
Critical Revision: İG, MZ, Aİ, MB
Final Approval: All of authors

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