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ORIGINAL ARTICLE

Predictors of In-Hospital Mortality in Patients With ST-Segment Elevation Myocardial Infarction Complicated With Cardiogenic Shock

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Background ST-segment elevation myocardial infarction (STEMI) complicated with cardiogenic shock (CS) remains as an unresolved condition causing high morbidity and mortality despite advances in medical treatment and coronary intervention procedures. In the current study, we evaluated the predictors of in-hospital mortality of STEMI complicated with CS.

Methods In this retrospective study, we evaluated the predictive value of baseline characteristics, angiographic, echocardiographic and laboratory parameters on in-hospital mortality of 319 patients with STEMI complicated with CS who were treated with primary percutaneous coronary intervention. Patients were divided into two groups consisting of survivors and non-survivors during their index hospitalisation period.

Results The mortality rate was found to be 61.3% in the study population. At multivariate analysis after adjustment for the parameters detected in univariate analysis, chronic renal failure, Thrombolysis In Myocardial Infarction (TIMI) post percutaneous coronary intervention (PCI) $\leq$2, plasma glucose and lactate level, blood urea nitrogen level, Tricuspid Annular Plane Systolic Excursion (TAPSE) and ejection fraction were independent predictors of in-hospital mortality.

Conclusions Apart from haemodynamic deterioration, angiographic, echocardiographic and laboratory parameters have an impact on in-hospital mortality in patients with STEMI complicated with CS.

Keywords Myocardial infarction • Percutaneous coronary intervention • In-hospital mortality • Cardiogenic shock

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Introduction

Cardiogenic shock is defined as a severe myocardial contractile dysfunction causing failure of the left ventricle to provide sufficient cardiac output despite a normal or elevated preload. As a result of reduced pump capability, a vicious cycle comprising altered mental status, cold extremities and decreased urine output emerges.

It was estimated that the incidence of cardiogenic shock (CS) is 40,000–50,000 cases per year in the United States and 60,000–70,000 cases in Europe [1,2]. Despite serious advances in percutaneous coronary interventions (PCI), CS complicating ST segment elevation myocardial infarction (STEMI) remains as an unresolved condition in coronary care units. Before improvements in coronary interventions to STEMI patients, mortality rates for STEMI complicated by CS were as high as 70–80% [3]. Currently, the mortality rates still remain unacceptably high at 40.4–52.4% [4,5]. This unresolved issue necessitates evaluating STEMI complicated with CS patients not solely with haemodynamics but also from a different perspective. The perspective should begin with the characterisation of new predictors of in-hospital mortality which may affect the direction of new therapeutic pathways and progress in approaching this patient group.

The metabolic imbalance concomitant with decreased biventricular functions and failure in coronary intervention cause inevitable high morbidity and mortality. It is beneficial to increase current knowledge of prognostic predictors of in-hospital mortality in STEMI complicated with CS patients. Therefore, in the current study, we investigate the prognostic value of chronic renal failure, right and left ventricular functions, metabolic parameters including blood levels of lactate, glucose, blood urea nitrogen and thrombolysis in myocardial infarction (TIMI) flow after PCI for in-hospital mortality in 319 consecutive STEMI complicated with CS patients who were all treated with primary PCI within the first 12 hours from the onset of chest pain.

Material and Methods

In our hospital, in Istanbul, the reperfusion strategy of patients with STEMI is performed by primary PCI. First of all, STEMI patients are assessed by the cardiology Residents in emergency departments. After the verification of STEMI diagnosis, patients are directly transferred to the catheterisation laboratory. Therefore, in our tertiary centre, STEMI patients are admitted to the coronary care unit following primary PCI. Thrombolytic therapy is not preferred apart from exceptional circumstances in our institution because we have the opportunity to perform primary PCI throughout 24 hours.

Study Population

From September 2013 to June 2016, 5536 consecutive STEMI patients treated with primary PCI in our hospital were recorded into our electronic database. Inclusion criteria were (1) men or women, over 18 years old; (2) diagnosis of STEMI complicated by cardiogenic shock at emergency department admission; (3) transfer to the catheterisation laboratory within 12 hours after symptom onset; and (4) absence of any other possible aetiology causing hypotension such as severe fluid depletion or sepsis. Among 5536 patients with STEMI, 332 patients were diagnosed as CS on admission. Three patients were excluded from the study, because all of them were administered thrombolytic therapy. Therefore, our final study population included 329 (5.94%) consecutive patients who were diagnosed with STEMI complicated with CS and were admitted to coronary intensive care unit after PCI. The study population consisted of patients who were diagnosed as CS at emergency admission. All of the patients were treated with standard $P2Y_{12}$ inhibitors before the PCI. Standard two-dimensional transthoracic echocardiography (TTE) was performed using a Vivid 7 system (GE Vingmed Ultrasound AS, Horten, Norway) in all patients at the first 12 hours in coronary care unit and left ventricular ejection fraction (EF) was calculated by using a modified Simpson method [6]. The pulmonary arterial peak systolic pressure was calculated using the simplified Bernoulli equation [7]. Right ventricular function was determined by using tricuspid annular plane systolic excursion (TAPSE) which has been described as an accurate method for evaluation of right ventricular function [8].

Baseline characteristics, demographics (age, gender), risk factors (hypertension, diabetes, current smoking, hyperlipidaemia, family history), medical history (previous myocardial infarction, previous PCI, previous coronary artery bypass grafting, previous stroke, previous heart failure, previous peripheral arterial disease, previous chronic renal failure, previous chronic obstructive pulmonary disease) were all recorded from our electronic database. The Local Ethical Committee of our hospital approved the study protocol. Patients were retrospectively enrolled in our study. On coronary care unit admission the following parameters were recorded: glucose, haematocrit, haemoglobin, leucocyte, platelet, creatinine, blood urea nitrogen, potassium, sodium, alanine transaminase, aspartate transaminase, lactate dehydrogenase and arterial blood gas values. Intra-aortic balloon pump is the only mechanical circulatory support available in our centre. Mechanical circulatory support devices such as the Impella® Circulatory Support System (Abiomed), left ventricular assist device and extracorporeal membrane oxygenation are not routinely used in our centre.

Definitions

Acute STEMI was defined as acute myocardial infarction with ST-segment elevation of >0.1 mV in two or more contiguous leads. Right ventricular dysfunction was defined as TAPSE of ≤16 mm. Cardiogenic shock was defined according to the SHOCK trial [9] as a systolic blood pressure ≤90 mmHg for at least 30 minutes or vasopressors required to maintain blood pressure >90 mmHg, evidence of end-organ hypo-perfusion (e.g. urine output <30 mL or cold, diaphoretic extremities or altered mental status) and evidence of elevated filling pressures (e.g. pulmonary congestion on examination or chest radiograph). Treatment of CS was left at the discretion of the operator and included, besides primary PCI, fluid resuscitation, endotracheal intubation, placement of an intra-aortic balloon pump (IABP) and catecholamine administration. Coronary flow was graded angiographically by the operator according to the thrombolysis in myocardial infarction (TIMI) criteria (TIMI 0–3).

Statistical Analysis

Analyses were performed using Statistical Package for Social Sciences software, version 20.0 (SPSS; IBM, Armonk, New York, USA). Continuous variables were expressed as the mean ± standard deviation and Kolmogorov-Smirnov test was used for testing of normality. Continuous variables were expressed as mean± SD and compared using independent sample t test. Continuous variables with skewed distributions were compared using the Mann-Whitney U test. Comparisons between categorical variables between the groups were performed using the $\chi 2$ or Fisher's exact test. Independent sample t-test was used in the analysis of continuous variables. To analyse the prediction for in-hospital mortality, data from the admission parameters were employed as independent variables. The univariate relationship between baseline characteristics and in-hospital mortality were assessed by univariate hierarchical logistic regression analysis. Multivariate analysis by stepwise logistic regression models (backward elimination) tested variables that were significant at $p < 0.1$ in the univariate analysis. Statistical significance was defined as a p value <0.05.

Results

Baseline Characteristics

Baseline characteristics, divided into two groups according to survival, are summarised in Table 1. A total of 329 patients (mean age 69 ± 13; male gender 61.1%) with STEMI complicated with CS were included. The baseline demographics were similar in terms of hypertension, diabetes mellitus, hyperlipidaemia, current smoking status, previous MI,

Table 1 Baseline Characteristics.

	Non-survivors (n = 202)	Survivors (n = 127)	p value
Age, y	71 ± 13	67 ± 12	0.002
Male gender, n(%)	117 (57.9)	84 (66.1)	0.136
ICCU hospitalisation period, day	3.4 ± 4.4	6.1 ± 6.8	<0.001
Total hospitalisation period, day	4.3 ± 6.9	13.0 ± 13.5	<0.001
Current smoking status, n(%)	71 (35.1)	43 (33.9)	0.811
Hypertension, n(%)	102 (50.5)	56 (44.1)	0.258
Diabetes mellitus, n(%)	80 (39.6)	38 (29.9)	0.075
Hyperlipidaemia, n(%)	42 (20.8)	33 (26.0)	0.274
Previous MI, n(%)	38 (18.8)	21 (16.5)	0.600
Previous PCI, n(%)	24 (11.9)	17 (13.4)	0.687
Previous CABG, n(%)	18 (8.9)	12 (9.4)	0.869
Previous PAD, n(%)	11 (5.4)	7(5.5)	0.979
Previous CVA, n(%)	11 (5.4)	5 (3.9)	0.536
Chronic kidney disease, n(%)	49 (24.3)	17 (13.4)	0.017
Congestive heart failure, n(%)	31 (15.3)	24 (18.9)	0.401
COPD, n(%)	16 (8.3)	6(4.7)	0.201
CPA on admission, n(%)	69 (34.2)	29 (22.8)	0.029
Pain-balloon time (hour)	5.1 ±2.3	5.5 ± 2.5	0.141
Door-balloon time (minute)	26 ± 9	26 ± 7	0.835
VT/VF before CAG, n(%)	37 (18.3)	16 (12.6)	0.170
VT/VF after CAG, n(%)	26 (12.9)	17 (13.4)	0.893
Complete AV Block, n(%)	20 (9.9)	14 (11.0)	0.745
Anterior MI, n(%)	137 (67.8)	78 (61.4)	0.235
Inferior MI, n(%)	58 (28.7)	42 (33.1)	0.403
Systolic arterial pressure (mm Hg)	69 ± 10	70 ± 9	0.211
AF, n(%)	18 (8.9)	8 (6.3)	0.393

Continuous variables are presented as mean ± SD.
Nominal variables presented as frequency (%).
Abbreviations: ICCU, intensive cardiac care unit; MI, myocardial infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft; PAD, peripheral arterial disease; CVA, cerebrovascular accident; COPD, chronic obstructive pulmonary disease; CPA, cardiopulmonary arrest; VT, ventricular tachycardia; VF, ventricular fibrillation; CAG, coronary angiography; AV, atria-ventricular; AF, atrial fibrillation.

previous PCI, previous CABG, chronic lung disease, cerebrovascular disease and congestive heart failure. At admission, cardiopulmonary arrest was statistically more frequent in non-survivor group ($p = 0.029$). Pain to balloon time, door to balloon time, occurrence of VT/VF before or after the PCI and development of complete AV block were similar between groups. Types of myocardial infarction as anterior or inferior, systolic arterial blood pressure levels and the presence of atrial fibrillation on admission were similar between the groups.

Coronary Angiography Findings

The data regarding the coronary angiography are summarised in Table 2. TIMI flow in the culprit vessel before the PCI was similar between the groups. TIMI flow before PCI was 0 in 96.6% of patients and 1 in 3.4%. Left main coronary artery was the culprit lesion in 12 (3.6%) patients. The success of PCI, presented by TIMI 3 flow after PCI was 54.1%. Patients in the survivor group exhibited a higher incidence of TIMI 3 flow after PCI compared to non-survivors. (43.1% vs. 71.7%, $p < 0.001$) The intervened vessel was similar between the groups. Stent implantation was performed in 83.5%, tirofiban infusion in 21.5% and thrombus aspiration in 7.5%. Intra-aortic balloon pump was inserted in 88 (26.7%) patients. The IABP insertion was statistically higher in non-survivor group. (31.2% vs. 19.7%, $p = 0.022$)

Laboratory and Echocardiography Findings

Laboratory and echocardiographic data are summarised in Table 3. Haematocrit, haemoglobin, leucocyte and platelet were similar between groups. Plasma glucose and lactate, serum creatinine and blood urea nitrogen level on admission were notably higher in the non-survivor group ($p < 0.001$, $p < 0.001$, $p < 0.001$ and $p < 0.001$ respectively). Ejection fraction and right ventricular TAPSE were remarkably higher in survivors ($p < 0.001$ and $p = 0.003$ respectively).

Logistic Regression Analysis

During logistic regression analysis, the univariate predictors of in-hospital mortality were indicated in Table 4. Age, chronic renal failure, TIMI post PCI ≤2, glucose, creatinine, blood urea nitrogen, pH, lactate, bicarbonate, left ventricular end-systolic diameter (LVESD), left ventricular end-diastolic diameter (LVEDD), TAPSE, ejection fraction, IABP administration were defined as univariate predictors of in-hospital mortality. At multivariate analysis after adjustment for parameters detected in univariate analysis, chronic renal failure, TIMI post PCI ≤2, glucose, blood urea nitrogen, lactate, TAPSE and ejection fraction (EF) were still independent predictors of in-hospital mortality which is shown in Table 5.

Table 2 **Angiographic data of the patients.**

	Non-Survivors (n = 202)	Survivors (n = 127)	p value
TIMI flow in culprit before intervention			
TIMI 0, n(%)	195 (96.5)	123 (96.9)	0.877
TIMI 1, n(%)	7 (3.5)	4 (3.1)	0.877
TIMI flow in culprit after intervention			
TIMI 0, n(%)	71 (35.1)	23 (18.1)	0.001
TIMI 1, n(%)	38 (18.8)	11 (8.7)	0.012
TIMI 2, n(%)	6 (3.0)	2 (1.6)	0.424
TIMI 3, n(%)	87 (43.1)	91 (71.7)	<0.001
Intervened vessel			
LMCA, n(%)	6 (3.0)	6 (4.7)	0.409
LAD, n(%)	103 (51.0)	56 (44.1)	0.223
CX, n(%)	10 (5.0)	12 (9.4)	0.112
RCA, n(%)	37 (18.3)	35 (27.6)	0.048
Multivessel, n(%)	46 (22.8)	18 (14.2)	0.055
Stent, n(%)	162 (80.2)	115 (90.6)	0.012
Tirofiban inf, n(%)	47 (23.3)	24 (18.9)	0.348
Thrombus aspiration, n(%)	17 (8.4)	8 (6.3)	0.481
IABP, n(%)	63 (31.2)	25 (19.7)	0.022
Inotropic period(day)	3.0 ± 3.6	3.8 ± 3.3	<0.001
GIS bleeding n(%)	13 (6.4)	10(7.9)	0.618

Continuous variables are presented as mean ± SD.
Nominal variables presented as frequency (%).
Abbreviations: LMCA, left main coronary artery; LAD, left anterior descending; CX, circumflex; RCA, right coronary artery; IABP, intra-aortic balloon pump; GIS, gastrointestinal system; TIMI, thrombolysis in myocardial infarction.

Table 3 **Laboratory and echocardiographic data of the patients.**

	Non-survivors (n = 202)	Survivors (n = 127)	p value
Glucose (mg/dl)	142.6 ± 52.1	118.3 ± 31.3	<0.001
Haematocrit	37.8 ± 6.3	37.8 ± 5.8	0.748
Haemoglobin (g/dl)	12.8 ± 2.9	12.6 ± 1.9	0.823
Leucocyte (x10^3/μ/L)	15.8 ± 6.6	16.1 ± 11.9	0.614
Platelet (x10^3/μ/L)	254.2 ± 100.4	254.1 ± 89.6	0.718
Creatinine (mg/dl)	1.53 ± 1.09	1.17 ± 0.53	<0.001
BUN (mg/dl)	36.4 ± 16.9	24.8 ± 11.0	<0.001
Potassium (mEq/L)	4.2 ± 0.4	4.2 ± 0.5	0.128
Sodium (mEq/L)	135.8 ± 14.0	136.8 ± 3.9	0.273
ALT	95.9 ± 154.8	77.2 ± 151.6	0.914
AST	211.8 ± 290.0	170.0 ± 203.9	0.859
LDH	586.6 ± 536.2	429.7 ± 346.4	0.003
pH	7.27 ± 0.12	7.35 ± 0.09	<0.001
pCO_2	36.1 ± 9.5	34.1 ± 6.0	0.111
pO_2	99.7 ± 50.4	98.3 ± 47.2	0.778
sO_2	95.0 ± 5.6	95.7 ± 6.0	0.072
Lactate (mmol/l)	6.7 ± 4.3	3.3 ± 1.6	<0.001
HCO_3	17.5 ± 4.2	20.1 ± 3.3	<0.001
LVESD	4.4 ± 0.6	4.0 ± 0.6	<0.001
LVEDD	5.5 ± 0.5	5.3 ± 0.4	0.001
EF	29.2 ± 8.2	35.4 ± 8.4	<0.001
TAPSE	1.7 ± 0.2	1.8 ± 0.2	0.003
TPAP	26.9 ± 9.3	27.7 ± 8.3	0.177
MR ≥ + 3	39 (19.3)	16 (12.6)	0.112
TR ≥ +3	16 (7.9)	7 (5.5)	0.404

Continuous variables are presented as mean ± SD.
Nominal variables presented as frequency (%).
Abbreviations: BUN, blood urea nitrogen; ALT, alanine transaminase; AST, aspartate transaminase; LDH, lactate dehydrogenase; HCO3, bicarbonate; EF, ejection fraction; LVESD, left ventricular end systolic diameter; LVEDD, left ventricular end diastolic diameter; TAPSE, tricuspid annular plane systolic excursion; MR, mitral regurtitation; TR, tricuspid regurtitation.

Discussion

Despite considerable advances in the treatment of STEMI patients during recent years, the morbidity and mortality remains unacceptably high in STEMI patients complicated with CS [10]. Haemodynamic deterioration, occurrence of multi-organ dysfunction and development of systemic inflammatory response play a major role in mortality of CS patients [11]. Accordingly, not only haemodynamic derangements but also inflammatory, neurohumoral and metabolic parameters should be taken into consideration since they could be precursors for new therapeutic strategies [12]. In the current study, we assessed the prognostic effect of baseline, metabolic and echocardiographic parameters and percutaneous success on in-hospital mortality in a group of consecutive STEMI patients complicated with CS. Chronic renal failure (CRF), admission lactate, glucose, blood urea nitrogen levels, TIMI post PCI ≤2, EF and TAPSE were demonstrated to have a prognostic role in-hospital mortality in our study population.

Chronic renal failure was defined as a prognostic determinant in STEMI patients complicated with CS [13]. CRF is a predisposing factor for renal acidosis. Since severe hypotension deteriorates renal perfusion, it deepens the renal acidosis and has a prominent role in the vicious cycle encountered in CS patients. Furthermore the success of the PCI was also reported low in patients with CRF [14]. Apart from CS patients, in-hospital mortality remains high in patients with STEMI who had preliminarily underlying CRF [15]. We also detected CRF as a prognostic indicator for in-hospital mortality in STEMI complicated with CS patients.

After widespread utilisation of primary PCI, it occupies a key role in the treatment of STEMI patients. Successful reperfusion with final TIMI 3 flow is the preferred target of all PCIs. In the current STEMI treatment era, the main aim is to decrease the stunning period of the ischaemic myocardium by rapid reperfusion [16,17]. TIMI flow is one of the underlying factors effecting the stunning period of the ischaemic myocardium [18]. Moreover, major adverse cardiac events and cardiac death were recently demonstrated to occur more

Table 4 Univariable analysis of the variables.

	p value	Odds ratio	%95 CI
Age	0.010	1.02	1.00–1.04
Diabetes mellitus	0.075	1.53	0.95–2.46
CRF	0.018	2.07	1.13–3.79
TIMI post PCI ≤2	<0.001	3.34	2.07–5.37
Glucose	<0.001	1.01	1.00–1.01
Creatinine	0.001	2.00	1.30–3.07
BUN	<0.001	1.07	1.04–1.09
pH	<0.001	0.14	0.06–0.25
Lactate	<0.001	1.60	1.40–1.85
HCO_3	<0.001	0.83	0.77–0.89
LVEDD	<0.001	2.33	1.45–3.74
LVESD	<0.001	2.42	1.64–3.56
TAPSE	0.001	0.18	0.06–0.50
EF	<0.001	0.91	0.88–0.94
IABP administration	0.023	1.84	1.08–3.13

Abbreviations: GFR, glomerular filtration rate; CRF, chronic renal failure; BUN, blood urea nitrogen; HCO3, bicarbonate; LVESD, left ventricular end systolic diameter; LVEDD, left ventricular end diastolic diameter; TAPSE, tricuspid annular plane systolic excursion; EF, ejection fraction; IABP, intra-aortic balloon pulsation; TIMI, thrombolysis in myocardial infarction.

frequently in STEMI patients with TIMI post PCI ≤2 [19]. In our study, it is also demonstrated that TIMI post PCI ≤2 has a prognostic role for in-hospital mortality in STEMI complicated with CS.

In addition to haemodynamic derangements, CS patients should be analysed with metabolic and neurohumoral parameters. Regarding metabolic parameters, serum lactate level is considered to be a reliable indicator of tissue hypoperfusion [20]. Recently, lactate was found to be a prognostic indicator for early and late mortality after emergent coronary artery bypass grafting in myocardial infarction complicated with CS [21]. Lactate was also reported to have a prognostic significance for in-hospital mortality of STEMI patients complicated with CS [12]. The underlying pathophysiology for hyperlactataemia was demonstrated by Chiolero et al. [22]. Increased tissue lactate production was caused by hyperglycaemia and increased non-oxidative glucose utilisation in the context of insufficient oxygen supply to tissues [22]. Hyperglycaemia on admission was also reported to be associated with high mortality in STEMI patients [23]. In patients undergoing primary PCI, hyperglycaemia on admission, irrespective of the presence of diabetes, was associated with increased in-hospital mortality [24]. The close relationship between hyperglycaemia and hyperlactataemia was also confirmed in our study. Irrespective of the presence of diabetes mellitus, serum glucose level on admission was determined to be a prognostic predictor for in-hospital mortality in STEMI patients complicated with CS.

Table 5 Multivariable analysis of the variables.

	p value	Odds ratio	%95 CI
CRF	0.042	2.21	0.91–5.34
TIMI post PCI ≤2	0.005	2.57	1.32–5.00
Glucose	<0.001	1.02	1.01–1.03
BUN	<0.001	1.06	1.03–1.09
Lactate	<0.001	1.76	1.48–2.10
TAPSE	0.044	0.23	0.05–0.96
EF	<0.001	0.92	0.88–0.96

Abbreviations: CRF, chronic renal failure; BUN, blood urea nitrogen; TAPSE, tricuspid annular plane systolic excursion; EF, ejection fraction; TIMI, thrombolysis in myocardial infarction.

Another important parameter in CS patients is serum blood urea nitrogen (BUN) level. BUN level increases in severe hypotension. The prognostic value of BUN level was tested in STEMI patients complicated with CS following extracorporeal membrane oxygenation and found to be a prognostic predictor for 30-day mortality [25]. The impaired contractile capacity of the left ventricle results in deteriorated renal perfusion and high BUN level. Our study indicated BUN was an independent prognostic predictor of in-hospital mortality in STEMI patients with CS. Several factors could contribute to this condition: low left ventricular ejection fraction decreases renal arterial perfusion, low TAPSE increases renal venous pressure and a deterioration in renal neurohumoral balance.

Transthoracic echocardiography parameters have an impact on in-hospital survivors of STEMI patients complicated with CS. In a meta-analysis, right ventricular dysfunction was demonstrated to be a predictor of an adverse prognosis in STEMI without CS [26]. In 2010, it was also indicated that right ventricular dysfunction is an independent predictor for mortality in STEMI patients presenting with CS [27]. Otherwise, left ventricular ejection fraction is a well-known predictor in STEMI patients which was also included in mortality score systems such as ACEF (age, creatinine, ejection fraction) [28]. Among echocardiographic abnormalities, low ejection fraction and TAPSE on admission were found to be independent predictors for in-hospital mortality in our study population.

Current knowledge of prognostic predictors of in-hospital mortality in STEMI patients complicated with CS is dispersed and each study was constituted on a single parameter. Thus a study reuniting all of the parameters with a large cohort has been missing. Valente et al. reported a similar study with a limited study population in 2007 [12]. Daviervala et al. tested clinical, metabolic, angiographic and echocardiographic parameters for prediction of in-hospital mortality after coronary artery bypass grafting for myocardial infarction [21]. Despite a large cohort, not only STEMI patients, but, rather, all of the myocardial infarction patients were involved [21]. Thus, our study differentiates from other studies regarding a large cohort and multiple predictors derived from different modalities. The important hallmark of our study is all of the STEMI patients complicated with CS were revascularised within 12 hours from symptom onset according to the revascularisation guidelines [29]. Following primary PCI, metabolic and echocardiographic parameters were obtained during coronary care unit hospitalisation. As a result, metabolic parameters (serum lactate, glucose, blood urea nitrogen) and echocardiographic parameters (TAPSE, left ventricular ejection fraction) were able to predict the in-hospital mortality of our study population. Patients who may benefit more from mechanical support devices may be treated earlier after prognostic assessment with baseline characteristics, angiographic, echocardiographic and laboratory parameters. Whereas patients who are expected to have higher mortality risk after classification with those parameters may be solely treated with medical treatment due to insufficient cost-effectiveness of mechanical support devices.

Study Limitations

First of all, the retrospective and single centre nature of the study is an important limitation. Secondly, considering TTE only TAPSE was measured as a parameter representing right ventricular function. Follow-up was limited to in-hospital mortality. However, mortality data was cross-checked with the national death registry.

Conclusion

Our study was conducted on 329 consecutive STEMI patients complicated with CS who were treated with primary PCI within the 12 hours of symptom onset. The mortality rate was found to be 61.3% in our study population. The main finding of our study was CRF, serum lactate, glucose, blood urea nitrogen levels, on admission TIMI post PCI $\leq$2, EF and TAPSE were demonstrated to be independent predictors of in-hospital mortality in STEMI patients complicated with CS.

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