Pak Heart J

IN HOSPITAL MORTALITY PREDICTORS OF PATIENTS WHO UNDERGO PRIMARY PERCUTANEOUS CORONARY INTERVENTION FOR ST SEGMENT ELEVATED MYOCARDIAL INFARCTION

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Date Received: May 25, 2019

Date Revised: Aug 22, 2019

Date Accepted: Sep 14, 2019

Contribution

BBK conceived the idea, designed the study, did data analysis and also did the final review.

All authors declare no conflict of interest.

This article may be cited as: Karasu BB. In hospital mortality predictors of patients who undergo primary percutaneous coronary intervention for ST segment elevated myocardial infarction. Pak Heart J 2019; 52 (04):349-55

ABSTRACT

Objective: To assess the in hospital mortality predictors of patients who underwent primary PCI in our hospital and to compare the results with similar studies in the literature .

Methodology: This cross-sectional study included patients who underwent primary PCI with the diagnosis of STEMI in department of cardiology Etimesgut Government Hospital, Ankara. Patients who survived and patients who died during their follow up in the hospital after primary PCI were assigned into 2 groups. Clinical and angiographic characteristics were compared between two groups. All statistical analyses were conducted using SPSS version 17.0 results were considered as significant if the p value was <0.05.

Results: Total of 85 patients were included. To groups were made on the bases of mortality. Group one had 58 patients who survived while group two had 25 patients who died during followup. Time interval between hospital admission and wire crossing (Door to balloon time) didn't differ among two groups ($88,64\pm32,47$ versus $86,72\pm38,33$, $p>0.05$) where as the patients who died had a longer symptom onset to hospital admission time ($8,72 \pm 7,68$ versus $3,19\pm2,82$, $p=0.001$). PCI performed during off hours wasn't found to be related with increased mortality ($p=0.830$).

Conclusion: Because the implementation of primary PCI in our hospital fulfil the quality of care and performance indicators recommended in the guidelines throughout the whole day, patient related factors become more likely to be associated with in hospital mortality compared to PCI related factors. Additional decline in the mortality rate can be achieved by raising the conciousness of community and improving transfer policies that could minimize the patient related factors including prehospital delay.

Key Words: ST segment elevated myocardial infarction; Primary percutaneous coronary intervention; In hospital mortality.

INTRODUCTION

ST-elevation myocardial infarction (STEMI) is one of the leading causes of death worldwide.[1] It is a cardiac emergency requiring immediate diagnosis and rapid therapeutic management. Although advances in cardiovascular pharmacotherapy and effective timely reperfusion by greater use of percutaneous coronary intervention (PCI) have considerably improved the prognosis related with STEMI, mortality remains substantial with a reported in hospital mortality rate of 6-14%. Notably, such rates may vary significantly across subsets of patients, underlining the presence of populations at higher or lower risks.[2] So, early and accurate risk stratification is preferred to predict the clinical outcome of patients with STEMI. In this manner, several studies were conducted to develop an applicable risk score system. Herein, we attempted to evaluate the clinical characteristics of patients with STEMI who died in a primary PCI center in Turkey. We also aimed to find which of the present risk score systems will mostly fit to represent our results.

With regard to the patients who have non ST elevated myocardial infarction or stable coronary artery disease needing revascularization, many risk scoring systems have been developed for identification of the high risk population.[8-13] However, in the context of STEMI, many of the studies examining the predictors of mortality were conducted through incorporation and validation of the predetermined risk scoring systems developed primarily for the patients with non ST elevated myocardial infarction or stable coronary artery disease.[14] Likewise, many of the remaining studies related with STEMI were conducted in the fibrinolytic era and validated by later studies for patients undergoing primary PCI.[15-19]

METHODOLOGY

This case control study included patients who underwent primary PCI with the diagnosis of STEMI in Department of Cardiology, Etimesgut Government Hospital. Of these, patients who survived after primary PCI during the hospitalization period were enrolled as the control group and patients who died after primary PCI during the index hospitalization period were enrolled as the study group (mortality group). Exclusion criteria included the patients who referred to hospital more than 24 hours after symptom onset, died before coronary intervention, who were treated with fibrinolytic therapy before PCI and whose data was lack of clinical information regarding the exact time of symptom onset; admission time to the hospital and experienced time delay in the hospital until stent implementation (Door to balloon time). Demographic, anthropometric, clinical and angiographic data of the patients in the registry were all recorded. The study was implemented in accordance with Guidelines for Good Clinical Practice laid down in Declaration of Helsinki. The study protocol was approved by the Hacettepe University Hospital ethics committee.

STEMI was diagnosed based on ischemic symtoms with ST segment elevation of > 1mm in at least two contiguous leads or >2 mm in leads V1 through V3 or new left bundle branch block and elevation of troponin levels.3 Prehospital delay (Patient related delay) was defined as the time interval between onset of the chest pain and presentation to the emergency department of the hospital. In hospital delay time (System related delay) was defined as the time from diagnosis of STEMI in the emergency department until stent implementation. “On hour” admission was described as the admission time to the hospital between 08:00 a.m and 17:00 p.m on weekdays. “Off hour” admission was described as the admission time to the hospital on weekends or between 17:00 p.m and 08:00 a.m on weekdays.

Treatment with antihypertensive drugs or a known diagnosis was defined as hypertension. Diabetes mellitus was diagnosed by fasting blood glucose >126 mg/dl, blood glucose >200 mg/dl at any time or a history of diabetes mellitus. Hypercholestrolemia was defined as a baseline cholestrol level >200 mg/dl and/or a low-density lipoprotein level >130 mg/dl or previously diagnosed and treated hypercholesterolemia. Cardiogenic shock was defined as persistent systolic blood pressure < 90 mmHg with clinical signs of poor organ perfusion in the absence of hypovolemia. Chronic renal failure was defined as a creatinine clearance less than 60 mL/min calculated by Cockroft formula.

Angiography procedures were carried out by the standart technique. The decision about treatment strategy was made by the operators. Angiograms were analyzed by two experienced physicians. Thrombolysis in myocardial infarction (TIMI) flow grade were determined from the angiographic films.

Continuous variables with normal distibution were expressed as mean ± SD and compared by Independent Samples T test. Categorical variables were described by frequencies or percentages and Pearson Chi- Square and Fisher's Exact test were used to analyse the data. Odds ratios and confidence intervals are presented in the tables. Kaplan-Meier method and Cox Regression analysis were performed to measure the effects of the variables and prepare the survival curves of the patients. All statistical analyses were conducted using SPSS version 17.0 for Windows. (SPSS Inc. Chicago, Illinois, USA) and results were considered as significant if the p value was <0.05.

RESULTS

Total of 85 patients were included. To groups were made on the bases of mortality. Group one had 58 patients who survived while group two had 25 patients who died during followup. All baseline demographics and clinical characteristics are described in Table 1.The mean age was 57.40±12.63 years in the control group and 65.24±10.90 years in the mortality group. The patients in the mortality group were older than those in the control group ($p<0.05$). There were no statistically significant differences between two groups in terms of gender distribution, history of hypertension or smoking. However, previous history of diabetes mellitus, chronic renal failure, coronary artery disease and myocardial infarction were found to be significantly more common among the mortality group population ($p<0.001$, $p<0.001$, $p=0.020$, $p=0.001$, $p=0.024$, respectively).

With respect to the localization of STEMI, there was no significant difference between two groups ($p>0.05$). The rate of the development of ventricular tachycardia/fibrillation was higher in the mortality group whereas the rates of development of atrioventricular block or atrial fibrillation were similar between two groups ($p=0.003$, $p=0.736$, $p= 0.377$, respectively). Presentation after cardiac arrest, high Killip class (Killip > 2), low

systolic blood pressure and high heart rate on admission were more likely to be seen in the mortality group compared to the control group with a statistical significance of p<0,001, p<0.001, p<0.001 p=0.004, respectively.

Time interval between symptom onset and hospital admission (pre hospital delay) was longer in the mortality group (8,72 ± 7,68 versus 3,19±2,82, p=0.001) but system related delay was found comparable between two groups (88,64±32,47 versus 86,72±38,33, p>0.05). In means of on-hour or off-hour admission times, there was no significant difference between two groups.

Mortality group had higher baseline levels of white blood cell (WBC) count, serum creatinine and glucose (p<0.001, p<0.001, p=0.012,respectively). Maximal CK-MB level was also higher in the mortality group (p=0.015). Whereas hemoglobin and hematocrit levels on admission were found lower in the mortality group (p=0.012, p=0.036). Baseline troponin value was slightly higher in the mortality group but the difference was not statistically significant (p=0.180).

The distribution of the type of the infarct related artery was similar between two groups. The rate of the existence of two or multivessel disease was more likely to be seen in the mortality group (p=0.029). Chronic total occlusion on non-infarct related artery was seen in a similar rate between two groups. TIMI 3 flow was less frequently obtained in the mortality group (p<0.001).In table 2, the clinical and angiographic characteristics are compared between two groups.

Table 1: Baseline Clinical Characteristics

Variables	Control Group (n=58)	Mortality Group (n=25)	P-Value
Age (years)	57.40±12.63	65,24±10,90	0.008
Male, n (%)	44 (75.86)	19 (76)	0.989
Hypertension, n (%)	27(46.55)	14 (58.33)	0.332
Diabetes mellitus, n (%)	12(20.69)	16 (66,67)	<0.001
Chronic renal failure, n (%)	1 (1.72)	10 (41.67)	<0.001
Smoking, n (%)	57 (98,28)	20 (86,96)	0.067
History of CAD, n (%)	2 (3.45)	8 (33.33)	0.001
History of MI, n (%)	1 (1.72)	4 (16.67)	0.024
History of CVA, n (%)	0 (0)	3 (15)	0.020
KILLIP class (%, 1/2/3/4)	(84.48)/(13.79)/(1.72)/(0)	16/24/4/56	<0.001
Systolic BP <100 mmHg, n (%)	6 (10.34)	19 (76)	<0.001
Heart rate >100/minute, n (%)	9 (15.52)	12 (48)	0.004
Development of AF, n (%)	4 (6.9)	4 (16)	0.377
Development of AV Blok, n (%)	4 (6.9)	3(12)	
Development of VT/VF, n (%)	4 (6.9)	9 (36)	0.003
Admission afterarrested, n (%)	0 (0)	7 (28)	<0.001
Symptom onset to admission (h)	3.19±2.82	7.68±8.72	0.001
Admission to wire crossing (min)	86.72±38.33	88.64±32.47	0.828
Off hour admission	27 (46.55)	11 (44)	0.830
Two or multivessel disease, n (%)	25 (43.1)	18 (72)	0.029
CTO on non-infarct related artery	6 (10.34)	6 (24)	0.20
TIMI-3 flow,n (%)	55 (94.83)	18 (72)	<0.001
Glucose (mg/dl)	165.09±88.24	290.25±171.38	<0.001
Creatinine (mg/dl)	1.00±0.00	1.67±0.87	<0.001
Hb (g/dl)	14.53±2.09	13.25±1.94	0.012
WBC ($10^3/mm^3$)	11.40±3.45	16.60±6.73	<0.001
Maximal CK-MB (IU/L)	161.40±151.43	285.74±293.94	0.015
Initial Troponin (ng/ml)	10.06±6.89	21.70±11.74	0.18

CAD, coronary artery disease; MI, myocardial infarction; CVA, cerebrovascular accident; BP, blood pressure; AF, atrial fibrillation AV, atrioventricular; VT, ventricular tachycardia; VF, ventricular fibrillation; CTO, chronic total occlusion; TIMI, thrombolysis in myocardial infarction

Table 2: Effects of Variables on the risk of In-Hospital Mortality

Variables		Odds Ratio	95% CI	P Value
Age > 65		1.75	0.65-4.69	0.266
Diabetes mellitus		7.67	2.66-22.13	<0.001
Chronic renal failure		40.71	4.8-345.07	<0.001
History of CAD		14.0	2.69-72.61	0.002
History of MI		11.4	1.20-108.13	0.034
History of CVA		23.4	1.15-475.19	0.040
KILLIP class 2 (compared to 1)		9.19	2.11-39.93	0.031
KILLIP class 3 (compared to 1)		12.25	0.64-234.82	0.096
KILLIP class 4(compared to 1)		319.0	16.21-6279.4	<0.001
KILLIP class >2 (compared to= 2)		85.6	10.13-721.56	<0.001
Systolic BP (< 100 mmHg)		27.44	7.89-95.56	<0.001
Heart rate (> 100/min.)		5.03	1.74-14.49	0.003
Development of VT/VF		7.56	2.06-27.95	0.002
Admission after arrested		47.43	2.58-870.80	0.009
Symptom onset to admission (> 1 hour)	(1.3)	1.3	0.48-3.51	0.240
Two or multivessel disease		3.39	1.23-9.38	0.018
TIMI < 3 flow (%)		7.13	1.66-30.50	0.008
Hyperglycemia mg/dl,compared to=140 mg/dl)	(>140	11.0	2.367-51.123	0.002
WBC (>10x10³/mm³ compared to=10x10³/mm³)		2.22	0.503-9.808	0.292
WBC (>15x10³/mm³ compared to=10x10³/mm³)		7.4	1.635-33.57	0.009

DISCUSSION

Cardiovascular diseases are the leading cause of death worldwide.[4] Of these, STEMI becomes prominent with a high short-term mortality due to its rapid disease progression rate.[5] Although survival after STEMI has considerably improved over decades due to the strategies enhancing early recognition and effective timely re perfusion particularly by primary PCI procedure, mortality related with STEMI remains substantial due to the disease, patient or procedure related factors.[6,7] So, early risk assessment at the time of first medical contact and/or in the hospital becomes important for predicting the clinical outcome of STEMI.

To date, Zwolle and CADILLAC are the risk scores that were generated and implemented to predict the 30 day mortality for STEMI patients undergoing primary PCI.[20,21] And, to our knowledge, there is only one risk score (ACTION-GWTG) that has been primarily developed to predict the in hospital mortality in patients with STEMI undergoing primary PCI.2,[22] Of these scores, Zwolle and CADILLAC have proven themselves as useful indexes for risk stratification after primary PCI, however ACTION-GWTG currently needs to be integrated and externally validated in further studies.[2,23]

In the highlight of the above risk scores, we attempted to establish the presence of any probable clinical, laboratory and angiographic characteristics that may be related with in hospital mortality in patients who underwent to primary PCI with the diagnosis of STEMI at our primary PCI center in Turkey. The following variables including age, history of diabetes mellitus, chronic renal failure, coronary artery disease, myocardial infarction, presence of two or multivessel disease, high Killip class, low systolic blood pressure, high heart rate, long symptom onset to hospital admission time (patient related prehospital delay), development of VT/VF, low TIMI flow grade, high levels of WBC, glucose, creatinine, CK-MB and lower hemoglobin levels were found to be more commonly encountered in patients who died in the index hospitalization period after primary PCI.

As well as the results of this study overlap with the previous risk score systems, there appeared to be some differences. Like in both Zwolle and CADILLAC risk indexes; advanced age, high Killip class, presence of two or multivessel disease and low TIMI flow grade were found to be related with mortality. Differently from

Zwolle and likely in the CADILLAC; lower hemoglobin levels and chronic renal failure were found to be associated with mortality whereas total ischemia time until reperfusion was longer in the mortality group likely in Zwolle.

With regard to ACTION-GWTG model, eight of the overall nine variables including age, heart rate, systolic blood pressure, diagnosis of STEMI, creatinine level, presentation after cardiac arrest, in cardiogenic shock or heart failure were found to be related with mortality in our study. Of these nine variables, troponin values were not shown to be higher in the mortality group of our study. We think that this was because the troponin samples were mostly taken only on admission. So, measured troponin levels didn't reflect the maximal troponin values. However, in order to recognize a reinfarction, the CK-MB levels were monitored as reflecting the maximal values and appeared to be significantly higher in our mortality group. Consistently, CK-MB assessment was previously shown to be a good predictor of infarct size on STEMI patients treated with PCI.[1]

Each of the remaining variables that were revealed to be related with mortality in our study were proven to be a predictor of mortality in previous different studies. Of these variables, DM was assigned to be a risk factor in TIMI risk score model which was built in the fibrinolytic era but soon after validated in patients undergoing primary PCI. On the other hand, high blood glucose level at admission, regardless of the presence of DM was shown to be closely associated with increased mortality in previous studies.[4] This finding seemed to be consistent with the results of our study stating the presence of higher glucose levels in the mortality group.

In patients with STEMI treated with fibrinolytic therapy, an independent association between leukocytosis and mortality was revealed by TIMI10A and TIMI10B studies.[24,25] A more recent sub study of HORIZONS-AMI trial involving patients with STEMI undergoing primary PCI detected an independent association between leukocytosis and 1 year mortality.[26] Similarly, high WBC count represented a higher risk of in hospital mortality in the present study.

The prevalence and impact of early ventricular tachycardia/fibrillation occuring in the setting of STEMI treated with primary PCI are poorly understood.[27,28] Although earlier investigations did not find a significant impact of ventricular tachycardia/fibrillation on prognosis, some later trials demonstrated an increased in hospital mortality in patients with ventricular tachycardia/fibrillation.[29] Never thless, short-term prognostic significance of early ventricular tachycardia/fibrillation in the first 48 hours of STEMI remains a ontroversial topic of discussion. In the present study, higher prevalence of ventricular tachycardia/fibrillation have been observed in the mortality group although none of the patients had died due to untreated VA.

The above mentioned variables were evaluated individually or in groups in many trials to analyse their impact on short and long term mortality among patients with STEMI. However, the results vary significantly across subsets of patients that mainly represent a selected population. So, their importance as an independent risk factor for mortality remains controversial and only some of them can be assigned as a measure in risk score systems which differ in nature from each other.

On the other hand, it's well established that there's a strong link between duration of myocardial ischemia and the survival rate, thereby achieving myocardial reperfusion as soon as possible is a priority. Total myocardial ischemia time until reperfusion is determined by patient related and system related factors.[30] In our study, the experienced time delay from the admission to the hospital until stent implementation (system related delay) was not found to be longer in the mortality group compared to the control group. In accordance with this finding, the number of patients who were admitted during off hours wasn't higher in the mortality group. The on- hour and off- hour disparity of in hospital mortality didn't occur. However, pre hospital delay (patient related delay) was shown to be significantly longer in the mortality group. On this basis, we propose that total myocardial is chemia time related with mortality is not attributable to the internal organization of the hospital sources. (system related delay). However, the attitude of the patients (patient related delay) plays the crucial role in determining the total ischemic time.

CONCLUSION

Patients with STEMI comprise a heterogeneous population with respect to the risk of adverse events. Thus, their correct stratification becomes essential to evaluate their prognosis. On this background, many variables have been analysed to generate risk scores. In the maze of plenty of risk scores, our present study evaluating patients from the real world correlated well with the ACTION-GWTG model. Beyond the measures in the ACTION-GWTG model, many other variables were also found to be related with mortality. We propose that more comprehensive, accurate but simpler and easily applicable risk score systems should be constructed for patients with STEMI undergoing primary PCI in the real world.

REFERENCES

1. Ahmed N, Carberry J, Teng V, Carrick D, Berry C. Risk assessment in patients with an acute ST-elevation myocardial infarction. J Comp Eff Res 2016;5(6):581-93.
2. Buccheri S, Capranzano P, Condorelli A, Scalia M, Tamburino C, Capodanno D.Risk stratification after ST-segment elevation myocardial infarction. Expert Rev Cardiovasc Ther 2016;14(12):1349-60.
3. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018 Jan 7;39(2):119-77.
4. Wang R, Mei B, Liao X, Lu X, Yan L, Lin M, et al. Determination of risk factors affecting the in-hospital prognosis of patients with acute ST segment elevation myocardial infarction after percutaneous coronary intervention.BMC Cardiovasc Disord 2017;17(1):243.
5. McManus DD, Gore J, Yarzebski J, Spencer F, Lessard D, Goldberg RJ, et al.Recent trends in the incidence, treatment,

and outcomes of patients with STEMI and NSTEMI. Am J Med 2011;124(1):40-7.

6. Widimsky P, Wijns W, Fajadet J, de Belder M, Knot J, Aaberge L, et al. Reperfusion therapy for ST elevation acute myocardial infarction in Europe: description of the current situation in 30 countries.Eur Heart J 2010;31(8):943-57.
7. Fox KA, Carruthers KF, Dunbar DR, Graham C, Manning JR,De Raedt H, et al. Underestimated and under-recognized: the late consequences of acute coronary syndrome (GRACE UK-Belgian Study).Eur Heart J 2010;31(22):2755-64.
8. Nashef SAM, Roques F, Michel P, Gauducheau E, Lemeshow S, Salamon R. European System for Cardiac Operative Risk Evaluation (EuroSCORE). Eur J Cardiothorac Surg 1999;16:9-13.
9. Nashef SA, Roques F, Sharples LD, Nilsson J, Smith C, Goldstone AR, et al.EuroSCORE II.Eur J Cardiothorac Surg 2012;41(4):734-44.
10. Rogers WJ, Canto JG, Lambrew CT,Tiefenbrunn AJ, Kinkaid B, Shoultz DA, et al. Temporal trends in the treatment of over 1.5 million patients with myocardial infarction in the US from 1990 through 1999: the National Registry of Myocardial Infarction 1, 2 and 3. J Am Coll Cardiol 2000;36(7):2056-63.
11. The GRACE Investigators. Rationale and design of the GRACE (Global Registry of Acute Coronary Events) Project: a multinational registry of patients hospitalized with acute coronary syndromes. Am Heart J 2001;141:190-9.
12. Sianos G, Morel MA, Kappetein AP, Morice MC, Colombo A, Dawkins K, et al. The SYNTAX Score: an angiographic tool grading the complexity of coronary artery disease. EuroIntervention 2005;1(2):219-27.
13. Garg S, Sarno G, Garcia-Garcia HM, Girasis C, Wykrzykowska J, Dawkins KD, et al. A new tool for the risk stratification of patients with complex coronary artery disease: the Clinical SYNTAX Score.Circ Cardiovasc Interv 2010;3(4):317-26.
14. Synetos A, Georgiopoulos G, Pylarinou V, Toutouzas K, Maniou K, Drakopoulou M, et al. Comparison of prognostic risk scores after successful primary percutaneous coronary intervention.Int J Cardiol 2017;230:482-7.
15. Califf RM, Pieper KS, Lee KL, Van De Werf F, Simes RJ, Armstrong PW, et al.Prediction of 1-year survival after thrombolysis for acute myocardial infarction in the global utilization of streptokinase and TPA for occluded coronary arteries trial.Circulation 2000;101(19):2231-8.
16. Morrow DA, Antman EM, Charlesworth A, Cairns R, Murphy SA, de Lemos JA, et al. TIMI risk score for ST-elevation myocardial infarction: a convenient, bedside, clinical score for risk assessment at presentation: an intravenous nPA for treatment of infarcting myocardium early II trial substudy.Circulation 2000;102(17):2031-7.
17 Amin ST, Morrow DA, Braunwald E, Sloan S, Contant C, Murphy S, et al.Dynamic TIMI risk score for STEMI. J Am Heart Assoc 2013;2(1):e003269.
18. Morrow DA, Antman EM, Giugliano RP, Cairns R, Charlesworth A, Murphy SA, et al. A simple risk index for rapid initial triage of patients with ST-elevation myocardial infarction: an InTIME II substudy. Lancet 2001;358(9293):1571-5.
19. Stone GW, Grines CL, Browne KF, Marco J, Rothbaum D, O'Keefe J, et al. Predictors of in-hospital and 6-month outcome after acute myocardial infarction in the reperfusion era: the Primary Angioplasty in Myocardial Infarction (PAMI) trail. J Am Coll Cardiol 1995;25(2):370-7.
20. De Luca G, Suryapranata H, van 't Hof AW, de Boer MJ, Hoorntje JC, Dambrink JH, et al.Prognostic assessment of patients with acute myocardial infarction treated with primaryangioplasty: implications for early discharge. Circulation 2004;109(22):2737-43.
21. Stone GW, Grines CL, Cox DA, Garcia E, Tcheng JE, Griffin JJ, et al. Comparison of angioplasty with stenting, with or without abciximab, in acute myocardial infarction. N Engl J Med 2002;346(13):957-66.
22. McNamara RL, Kennedy KF, Cohen DJ, Diercks DB, Moscucci M, Ramee S, et al. Predicting ın-hospital mortality in patients with acute myocardial ınfarction.J Am Coll Cardiol 2016;68(6):626-35.
23. Díez-Delhoyo F, Valero-Masa MJ, Velásquez-Rodríguez J, Devesa-Cordero C, Sousa-Casasnovas I, Juárez M, et al. Very low risk ST-segment elevation myocardial infarction? It exists and may be easily identified. Int J Cardiol 2017;228:615-20.
24. Bagai A, Schulte PJ, Granger CB, Mahaffey KW, Christenson RH, Bell G, et al. Prognostic implications of creatine kinase-MB measurements in ST-segment elevation myocardial infarction patients treated with primary percutaneous coronary intervention. Am Heart J 2014;168(4):503-11.
25. Gollop ND, Dhullipala A, Nagrath N, Myint PK. Is periprocedural CK-MB a better indicator of prognosis after emergency and elective percutaneous coronary intervention compared with post-procedural cardiac troponins?Interact Cardiovasc Thorac Surg 2013;17(5):867-71.
26. Abid AR, Mallick NH, Shahbaz A, Tarin SA. In-hospital outcome of acute myocardial infarction (st segment elevation type) in diabetics and non-diabetics.J Coll Physicians Surg Pak 2005;15(9):524-7.
27. Straumann E, Kurz DJ, Muntwyler J, Stettler I, Furrer M, Naegeli B, et al.Admission glucose concentrations independently predict early and late mortality in patients with acute myocardial infarction treated by primary or rescue percutaneous coronary intervention.Am Heart J 2005;150(5):1000-6.
28. Barron HV, Cannon CP, Murphy SA, Braunwald E, Gibson CM. Association between white blood cell count, epicardial blood flow, myocardial perfusion, and clinical outcomes in the setting of acute myocardial infarction: a thrombolysis in myocardial infarction 10 substudy.Circulation 2000;102(19):2329-34.
29. Seropian IM, Sonnino C, Van Tassell BW, Biasucci LM,

Abbate A. Inflammatory markers in ST-elevation acute myocardial infarction.Eur Heart J Acute Cardiovasc Care 2016;5(4):382-95.

30. Palmerini T, Mehran R, Dangas G, Nikolsky E, Witzenbichler B, Guagliumi G.Impact of leukocyte count on mortality and bleeding in patients with myocardial infarction undergoing primary percutaneous

coronary interventions: analysis from the Harmonizing Outcome with Revascularization and Stent in Acute Myocardial Infarction trial.Circulation 2011;123(24):2829-37.

31. Kosmidou I, Embacher M, McAndrew T, Dizon JM, Mehran R, et al. Early ventricular tachycardia or fibrillation in patients with ST elevation myocardial ınfarction undergoing primary percutaneous coronary ıntervention and ımpact on mortality and stent thrombosis (from the Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction Trial). Am J Cardiol 2017;120(10):1755-60.

32. Medina-Rodríguez KE, Almendro-Delia M, García-Alcántara Á, Arias-Garrido JJ, Rodríguez-Yáñez JC. Prognostic implication of early ventricular fibrillation among patients with ST-elevation myocardial infarction.Coron Artery Dis 2017;28(7):570-6.

33. Masuda M, Nakatani D, Hikoso S, Suna S, Usami M, Matsumoto S, et al. Clinical ımpact of ventricular tachycardia and/or fibrillation during the acute phase of acute myocardial ınfarction on ın-hospital and 5-year mortality rates in the percutaneous coronary ıntervention era. Circ J 2016;80(7):1539-47.