Impact of High-degree Atrioventricular Block on No-Reflow Phenomenon and Prognosis In Patients With ST-Segment Elevation Myocardial Infarction

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ABSTRACT

Introduction: Although it has been established that the high-degree atrioventricular block (HAVB) is associated with mortality in the course of ST segment elevation myocardial infarction (STEMI), the mechanisms by which the AV block causes mortality is not fully clear. In this study we aimed (i) to investigate relationship between HAVB and no-reflow which has been repeatedly shown to be associated with both short- and long-term mortality, (ii) to determine the effect of both HAVB and no-reflow on in-hospital and long-term mortality.

Materials and Method: A total of 1217 patients who underwent primary percutaneous coronary intervention (pPCI) were divided into two groups according to HAVB development, further according to no-reflow development. Independent predictors of no-reflow were investigated. Furthermore patients were compared in terms of in-hospital and long-term mortality.

Results: In present study, 47 patients (3.8%) had HAVB, and 150 patients (12.3%) had no-reflow. HAVB was an independent predictor of no-reflow (OR: 3.856, %95CI: 1.488-9.995; p=0.005). In survival analysis both HAVB and no-reflow was associated with in-hospital (19.1% vs. 3.2%; p<0.001 and 10.7% vs. 2.9%; p<0.001, respectively) and long-term (15.7% vs. 6.2%; p=0.037 and 14.1% vs. 5.5%; p<0.001, respectively) mortality.

Conclusion: In this study, we found that HAVB emerged with STEMI is associated with long-term mortality in addition to short-term. We presented the first study to examine the impact of HAVB on reperfusion success which is highly correlated with survival. We found HAVB is an independent predictor of no-reflow and patients that had no-reflow had a worse prognosis both in short- and long-term follow-up.

Keywords: ST segment elevation myocardial infarction, no-reflow, High grade AV block.

ST-Segment Yükselmeli Miyokard İnfarktüslü Hastalarda Yüksek Dereceli Atriyoventriküler Bloğun No-Reflow Fenomenine ve Prognoza Etkisi

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ÖZET

Giriş: ST segment yükselmeli miyokard enfarktüsü (STEMI) seyrinde yüksek dereceli atriyoventriküler bloğun (HAVB) mortalite ile ilişkili olduğu tespit edilmiş olsa da, AV bloğunun mortaliteye hangi mekanizmalarla neden olduğu, net olarak ortaya konulmamıştır. Bu çalışmada, (i) HAVB ile, hem kısa hem

de uzun dönem mortalite ile ilişkili olduğu defalarca gösterilen no-reflow arasındaki ilişkiyi (ii) hem HAVB hem de no-reflow'un hastane içi ve uzun dönem mortaliteye etkisini araştırmayı amaçladık.

Hastalar ve Metod: Primer perkütan koroner girişim (pPCI) yapılan toplam 1217 hasta, HAVB gelişip gelişmemesine, daha sonra da no-reflow gelişip gelişmemesine göre iki gruba ayrıldı. No-reflow'un bağımsız prediktörleri araştırıldı. Son olarak da hastalar, hastane içi ve uzun dönem mortalite açısından karşılaştırıldı.

Bulgular: Bu çalışmada, 47 hastada (%3.8) HAVB, 150 hastada (%12.3) no-reflow izlendi. HAVB, no-reflow'un bağımsız bir prediktörü olarak saptandı (OR: 3.856,% 95CI: 1.488-9.995; p=0.005). Sağ kalım analizinde HAVB ve no-reflow'da hastane içi mortalite (sırasıyla %19.1' e karşı %3.2; p<0.001 ve %10.7'ye karşı %2.9; p <0.001) ve uzun dönem mortalite (sırasıyla %15.7'ye karşı %6.2; p=0.037 ve %14.1' e karşı %5.5; p<0.001) daha yüksek olarak izlendi.

Sonuç: Bu çalışmada, STEMI ile ortaya çıkan HAVB' nin, kısa döneme ek olarak, uzun dönem mortalite ile de ilişkili olduğu tespit edildi. HAVB ile sağ kalımla ileri derecede korelasyonu olan reperfüzyon başarısının ilişkisini gösteren bu ilk çalışmada, HAVB' nin no-reflow için bağımsız bir prediktör olduğunu ve no-reflow'u olan hastaların hem kısa hem de uzun dönem izlemlerinde kötü prognoza sahip olduğunu ortaya koyduk.

Anahtar Kelimeler: Yüksek dereceli atriyoventriküler blok, ST segment yükselmeli miyokard infarktüsü, no-reflow.

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INTRODUCTION

Electrical conduction abnormalities are one of the major complications of acute ST segment elevation myocardial infarction (STEMI). Autonomic imbalance or ischemia and necrosis of the conduction system are known as the most likely mechanisms. High-degree atrioventricular block (HAVB) (defined as the presence of Mobitz type II second-degree or third-degree AV block) is, with a 1.5–6.9% incidence[1, 2], being dominant manifestation of AV conduction disturbance. Although primary percutaneous coronary intervention (pPCI) can ameliorate AV blocks complicated acute STEMI[3], HAVB continues to be associated with ominous outcomes, such as in-hospital death[4, 5].

In the setting of pPCI, no-reflow is defined as suboptimal myocardial reperfusion through a part of coronary circulation without angiographic evidence of mechanical vessel obstruction[6]. No-reflow is associated with lower left ventricular ejection fraction (LVEF), adverse left ventricular remodeling, increased number of mechanical complications and short- and long-term mortality[7-9].

Although many studies have been performed previously focus on association between HAVB and mortality, the mechanism by which the AV block causes mortality has not been clearly elucidated. In this

study we aimed to determine the impact of HAVB on reperfusion success and the effect of both HAVB and no-reflow on in-hospital and long term mortality.

PATIENTS and METHODS

Study Population

A total of 1303 patients with STEMI who underwent pPCI from January 2011 to June 2015 were retrospectively enrolled in the study. STEMI was defined based on the following criteria: a typical increase or decrease in cardiac biomarkers; ongoing ischemic symptoms (within 12 h of presentation); newly developed left bundle-branch block pattern, or a new ST elevation in two or more contiguous leads, with readings of at least 0.2 mV in leads V1, V2, and V3 or at least 0.1 mV in the remaining leads; or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality[10]. HAVB was defined as the presence of Mobitz type II second-degree or third-degree AV block and being have symptomatic of patients. All patients present with HAVB and symptomatic were applied a temporary pacemaker as soon as possible. A total of 86 patients who were treated noninvasively, patients under treatment of hemodialysis and had electrolyte imbalance and patients whose final diagnosis on discharge was other than STEMI (e.g., myocarditis, Kounis syndrome or Takotusubo cardiomyopathy) were excluded from the study. A total of remaining 1217 patients who were all treated with pPCI constituted the study population. Long-term follow-up data was obtained from hospital records and phone interviews. For patients we were not able to reach, we gathered information from the National Institute of Statistics, and the Registrar of Birth Records to determine whether they were deceased.

The study protocol was reviewed and approved by the local ethics committee of Kafkas University and was conducted in accordance with the Declaration of Helsinki.

Data collections

Baseline clinical and demographic characteristics, and patients' medical history data were obtained from the hospital records. Complete blood count and blood biochemical parameters had been measured in all patients on admission to the hospital and prior to pPCI. Blood samples were retested for troponin T and creatine kinase myocardial band (CK-MB) every 6 hours, until peak levels were detected. Afterwards, these tests, along with hemograms and creatinine tests, were repeated daily. Estimated glomerular filtration rate

(eGFR) was determined using Cockroft-Gault formula from blood sample obtained on admission. LVEF was defined as the post-procedural ejection fraction, and was assessed using a modified Simpson's method.

Angiographic analysis

The standard Judkins percutaneous transfemoral technique was used for all patients who were treated with pPCI by experienced interventional cardiologists. Coronary angiograms were recorded in digital media for quantitative analysis (Dicom-viewer; MedCom GmbH, Darmstadt, Germany). Digital angiograms were analyzed by two, independent and experienced, interventional cardiologists, who were blinded to all data. Patients' Syntax score was calculated using SS Calculator, version 2.1[11]. Acute transient or persistent coronary flow reduction [final thrombolysis in myocardial infarction (TIMI) flow grade <3 or final myocardial blush grade (MBG) <2] at the target vessel lesion in the absence of spasm, trombus, dissection and/or significant residual stenosis was defined as epicardial no-reflow[12].

Statistical analysis

The statistical analyses were performed using SPSS version 22.0 (SPSS Inc., Chicago, IL). With respect to data distribution and normality, the mean (± standard deviation) or median (0.25–0.75 percentiles) was used to express continuous variables, and a*t*-test or Mann–Whitney *U*-test was conducted to compare the variables. The categorical variables were presented as numbers (percentages) and compared using Fisher's exact test or an χ2-test. Multivariable logistic regression analyses were performed to identify the independent predictors of no-reflow, using variables that showed statistically significant association with no-reflow in the univariate analyses. Survival curves were calculated using the Kaplan-Meier method. Statistical significance was assessed using log-rank tests. A *p*-value < 0.05 indicated statistical significance.

RESULTS

The study population consisted of 1217 STEMI patients (mean age: 56 ± 12.3 years; 81.5% were males) who underwent pPCI. HAVB was observed in 3.86 % (n: 47) of the study population. Demographic, clinical, laboratory and coronary angiographic characteristics of all patients, patients with HAVB and without HAVB are listed in Table 1. Heart rate was significantly lower in patients with HAVB compared to patients without HAVB (35±5.3 vs. 79±13.9; p<0.001). No-reflow was significantly more frequent in patients with HAVB than those without (11.7% vs. 23.4%; p<0.001). Patients with HAVB were older, had a higher level of C-reactive protein (CRP) and had a higher percentage of infarct related artery (IRA) of RCA than those

without HAVB. Furthermore, compared to patients without HAVB, patients with HAVB had a lower systolic blood pressure and a lower level of hemoglobin, albumin and eGFR; a longer stent length and had more frequent ventricular arrhythmia. There was no difference between patients with and without HAVB, in terms of infarct size (detected by CK-MB), coronary artery disease (CAD) severity (determined by Syntax score), Killip class on admission, B-type natriuretic peptide, LVEF and total ischemia time.

During a mean follow-up of 43.3 months, 123 (9.9%) all-cause deaths were reported. The rate of in-hospital mortality was significantly higher in patients with HAVB than those without (n=9, 19.1% vs. n=38, 3.2%; p<0.001). The Kaplan–Meier survival curve of in-hospital is shown in Figure 1A. The rate of long-term mortality (the rate among the survivors (n=38 patients with HAVB, n=1132 patients without HAVB) beyond the discharge was still significantly higher for HAVB patients compared to without HAVB patients) (n=6, 15.7% vs. n=70, 6.2%; p=0.037 Figure 1B).

When the relationship between no-reflow and other variables was examined; it was seen that HAVB was more frequent in patient with no-reflow than those without. Patients with no-reflow were older, had a more frequent history of diabetes mellitus (DM) and hyperlipidemia, and had a higher percentage of smoking compared to those without no-reflow. Compared to patients without no-reflow, patients with no-reflow had a higher Killip class on admission; higher white blood cell (WBC) and neutrophil count; lower lymphocyte count; higher level of peak CK-MB and troponin, CRP, B-type natriuretic peptide (BNP), Fasting Blood Glucose (FBG); and lower hemoglobin, serum albumin, eGFR and LVEF. Furthermore patients with no-reflow had longer and smaller stent size; higher basal Syntax score; longer total ischemic time; more frequent thrombus grade≥3 and proximal/ostial lesion location than those without no-reflow (Table 2).

Multivariate regression analysis was used to determine the independent predictors of no-reflow by using parameters that were found to be associated with no-reflow in the univariate analysis(Table 3). HAVB (Odds ratio [OR]:3.127, 95% confidence interval [CI]: 1.215-9.056; p=0.006), Neutrophil-to-lymphocyte ratio (OR:1.048, 95% CI: 1.009-1.087; p=0.015), CRP (OR: 1.102, 95% CI: 1.008-1.202; p=0.027), LVEF (OR: 0.954, 95% CI: 0.898-0.976; p<0.001), total ischemia time (OR: 1.007, 95% CI: 1.005-1.009; p<0.001), thrombus grade≥3 (OR: 2.317 95% CI: 1.284-4.179; p=0.005), stent length (per 1 mm, OR: 1.054, 95% CI: 1.032-1.091; p<0.001), and stent diameter (per 1 mm, OR: 2.128, 95% CI: 1.186-4.354; p=0.011) were found to be independent predictors of no-reflow.

The rate of in-hospital mortality was significantly higher in patients with no-reflow than those without (n=16, 10.7% vs. n=31, 2.9%; p<0.001). The Kaplan–Meier survival curve of in-hospital is shown in Figure 2A. The rate of long-term mortality (the rate among the survivors (n=134 patients with no-reflow, n=1031 patients without no-reflow) beyond the discharge was still significantly higher for no-reflow patients compared to without no-reflow patients) (n=19, 14.1% vs. n=57, 5.5%; p<0.001 Figure 2B).

DISCUSSION

In the present study, we focused on the potential relationship between HAVB and the development of no-reflow in patients undergoing pPCI for STEMI. Our study demonstrated that STEMI patients with no-reflow had a higher frequency of HAVB. HAVB was shown to be an independent predictor of no-reflow development during pPCI and patients that had both HAVB and/or no-reflow, had a worse prognosis both in-hospital and in the long-term follow up.

HAVB with a slow escape rhythm is a potentially fatal event in the setting of STEMI if not detected and treated. Although a significant portion of HAVB is transient, rarely it may progress to irreversible and symptomatic block. To date, several clinical parameters related to the development of HAVB have been established in STEMI patients. Consistent with the results of previous studies[1, 4, 5]; we found that older age; infarct related artery of RCA and reduced eGFR were associated with increased likelihood of HAVB development. In accordance with previous studies LVEF, infarct size, total ischemic time, CAD severity (in this study determined by Syntax) were not different in patients with and without HAVB[1, 5, 13]. In the present study we also observed that patients with HAVB had a reduced hemoglobin level. Although there is no definitive relationship between anemia alone and HAVB, anemia could probably facilitate HAVB in STEMI patients through reduction in oxygen presentation and emergence of deeper tissue hypoxia[14].

A significant number of previous studies have shown that HAVB is associated with short-term mortality, but the results of long-term mortality seem contradictory[1, 4, 5, 13, 15]. In our study, which had one of the longest follow-ups to date, HAVB was associated with both in-hospital and long-term mortality. Although many studies have focused on the relationship between HAVB and mortality, the mechanism by which HAVB is caused by mortality, even in transient pacemaker-treated patients, has not been clearly demonstrated. Similar to our study, in a study conducted by Auffret et al. it has been established that the HAVB is associated with reduced post pPCI TIMI flow[16]. However for the first time in our study, HAVB found an independent predictor of no-reflow.

No-reflow is described as inadequate myocardial perfusion without evidence of vessel obstruction. The cause of no-reflow is complex and multifactorial. Despite not completely elucidated, multiple mechanisms have been put forward as the cause of no-reflow including tissue edema, distal embolization, spasm of microcirculation, platelet aggregation, neutrophilic plugging or a combination of these factors[17-19]. In STEMIs accompanied by HAVB, emergence of bradicardia and hypotension could decrease the cardiac output and perfusion pressure and increase intracoronary stasis possibility. Therefore HAVB may make more prominent the mechanisms responsible for the etiopathogenesis of no-reflow. More importantly, right ventricular (RV) apical pacing may not give the desired result in terms of haemodynamics. It has been previously established that RV apical pacing and dyssynchrony, even in acute phase, changes the electrical and mechanical activation pattern of the ventricles which result in changes in cardiac metabolism and perfusion, oxygen demand, haemodynamics, and mechanical function[20]. The increase in oxygen demand and the reduction in perfusion capacity may accelerate or embody the development of no-reflow.

Limitations

The present study has several limitations. First, although the data were acquired prospectively, the study had a retrospective design and was based on a registry analysis and number of patients with HAVB was low. Second, reperfusion success was evaluated only by visual assessment and a more specific and sensitive method such as coronary flow reserve, contrast echocardiography or cardiac magnetic resonance was not used. Finally, this study is the absence of exhaustive recording of permanent pacemaker implantation during the course of hospitalization and in the follow up. Namely, this study does not answer whether patients with HAVB in the setting of STEMI will be in need of permanent pacemaker in long-term.

CONCLUSION

This study showed that HAVB emerged with STEMI is associated with long-term mortality in addition to short-term. In this study which was the first study to examine the impact of HAVB on reperfusion success, we have shown that HAVB is significantly related to no-reflow, and is an independent predictor of no-reflow. Furthermore patients that had no-reflow had a worse prognosis both in short- and long-term follow-up. HAVB should be closely monitored because of its association with no-reflow, in addition to the relationship between HAVB and mortality.

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CONFLICT OF INTERESTS

The authors declare they have no conflicts of interest.

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		HAVB				
		All Patients (n:1217)		Patients without HAVB, n:1170		Patients with HAVB n:47		p value
Age		56	±12.3	56	12	61	±12.2	0.012
Male gender, n (%)		992	(81.5)	953	(81.5)	39	(83.0)	0.792
Diabetes, n (%)		280	(23.0)	268	(22.9)	12	(25.5)	0.675
Hypertension, n (%)		491	(40.3)	466	(39.8)	25	(53.2)	0.067
Hyperlipidemia, n (%)		538	(44.2)	518	(44.3)	20	(42.6)	0.816
COPD, n (%)		59	(4.8)	56	(4.8)	3	(6.4)	0.617
PAD, n (%)		205	(16.8)	198	(16.9)	7	(14.9)	0.716
Family history of CAD, n (%)		280	(23.0)	273	(23.3)	7	(14.9)	0.178
Smoking, n (%)		663	(54.5)	636	(54.4)	27	(57.4)	0.677
ASA, n (%)		25	(2.1)	24	(2.1)	1	(2.1)	0.971
b-Blocker, n (%)		86	(7.1)	82	(7.0)	4	(8.5)	0.694
ACEI/ARB, n (%)		238	(19.6)	230	(19.7)	8	(17.0)	0.655
Statin, n (%)		213	(17.5)	208	(17.8)	5	(10.6)	0.207
Insulin, n (%)		82	(6.7)	77	(6.6)	5	(10.6)	0.277
Arrest on admission		30	(2.5)	28	(2.4)	2	(4.3)	0.420
Killip class > 1 on admission (%)		195	(16.0)	186	(15.9)	9	(19.1)	0.551
Systolic blood pressure, mm Hg		132	±31.7	132	±30.9	118	±45.5	0.012
Heart rate, bpm		77	±16.1	79	±13.9	35	±5.3	<0.001
Hemoglobin (g/dL)		13.7	±1.8	13.7	±1.8	12.9	±2.0	0.009
WBC Count (/1000)		12.329	±3.9	12.301	±3.8	13.020	±5.4	0.587
Platelet Count (10000/microliter)		258	±68.0	259	±67.9	249	±70.9	0.301
Neutrophil Count (/1000)		9.573	±3.7	9.556	±3.6	9.991	±4.8	0.794
Lymphocyte count (/1000)		1.7	1.2-2.4	1.7	1.2-2.4	1.7	1.2-2.3	0.978
Neutrophil-to-lymphocyte ratio		5.3	3.4-8.2	5.3	3.4-8.2	4.9	3.5-7.3	0.639
FBG on admission (mg/dL)		127.0	105.0-170.5	127.0	105.0-170.0	139.0	109.0-203.0	0.131
C-Reactive protein (mg/dl)		10.0	5.6-17.6	9.9	5.6-17.3	12.1	7.8-24.1	0.045
Serum albumin (g/dl)		3.74	±0.48	3.75	±0.48	3.51	±0.42	<0.001
Estimated glomerular filtration rate		88.27	±25.56	88.73	±25.29	76.94	±29.71	0.016
Peak Creatine Kinase MB (ng/mL)		179.0	101.5-320.0	176.0	101.0-316.0	234.0	144.0-360.0	0.073
Peak Troponin I (ng/mL)		82.2	37.8-187.0	81.4	37.0-186.9	94.0	59.8-223.5	0.166
B-type natriuretic peptide pg/mL		73.7	35.8-137.1	72.0	35.0-134.6	87.0	49.9-174.3	0.113
Left Ventricular Ejection Fraction (%)		46.70	8.33	46.71	8.36	46.46	7.80	0.771
Total ischemia time, min		179.0	115.0-270.0	178.5	115.0-270.0	190.0	112.0-315.0	0.713
Infarct-related artery, n (%)	LAD	634	(52.1)	633	(54.1)	1	(2.1)	<0.001
	Cx	151	(12.4)	149	(12.7)	2	(4.3)	
	RCA	415	(34.1)	371	(31.7)	44	(93.6)	
	Other coronaries (Diagonal etc.)	17	(1.4)	17	(1.5)	0	(0.0)	
Proximal/ostial lesion for IRA, n (%)		702	(57.7)	677	(57.9)	25	(53.2)	0.525
Stent length, mm		21.85	±9.1	21.71	8.90	25.65	±12.7	0.003
Stent diameter, mm		3.11	±0.4	3.10	0.36	3.20	±0.5	0.146
No-reflow		150	(12.3)	131	(11.7)	19	(23.4)	<0.001
Ventricular tachycardia / fibrillation, n		67	(5.5)	61	(5.20)	6	(12.8)	0.026

(%)							
Basal syntax score	16.63	±4.5	16.61	±4.5	17.27	±3.8	0.107

Table 1 Demographic, clinical and laboratory characteristics of all patients, patients with and without HAVB, with p value. Abbreviations: HAVB: High degree atrioventricular block; COPD: Chronic Obstructive Pulmonary Disease; PAD: Peripheral Arterial Disease; CAD: Coronary artery disease; ASA: Acetylsalicylic acid; ACEI/ARB: Angiotensin converting enzyme inhibitor/Angiotensin receptor blocker; WBC: White Blood Cell; FBG: Fasting Blood Glucose; IRA: Infarct-related artery

	No-reflow				
	(-); n=1067		(+); n=150		p Value
Age	56	±12	61	±13	<0.001
Female gender, n (%)	874.0	(81.9)	118.0	(78.7)	0.338
Diabetes, n (%)	236.0	(22.1)	44.0	(29.3)	0.049
Hypertension, n (%)	415.0	(38.9)	76.0	(50.7)	0.006
Hyperlipidemia, n (%)	483.0	(45.3)	55.0	(36.7)	0.047
COPD, n (%)	49.0	(4.6)	10.0	(6.7)	0.268
PAD, n (%)	172.0	(16.1)	33.0	(22.0)	0.072
Family history of CAD, n (%)	250.0	(23.4)	30.0	(20.0)	0.350
Smoking, n (%)	594.0	(55.7)	69.0	(46.0)	0.026
ASA, n (%)	23.0	(2.2)	2.0	(1.3)	0.506
b-Blocker, n (%)	77.0	(7.2)	9.0	(6.0)	0.586
ACEI/ARB, n (%)	208.0	(19.5)	30.0	(20.0)	0.884
Statin, n (%)	204.0	(19.1)	9.0	(6.0)	<0.001
Insulin, n (%)	70.0	(6.6)	12.0	(8.0)	0.510
Arrest on admission	23.0	(2.2)	7.0	(4.7)	0.063
Killip class > 1 on admission (%)	157.0	(14.7)	38.0	(25.3)	0.001
Systolic blood pressure, mm Hg	131	±30	138	±43	0.063
Heart rate, bpm	77	±15	76	±21	0.976
Hemoglobin (g/dL)	13.7	±1.8	13.5	±2.0	0.131
WBC Count (/1000)	12.174	±3.752	13.434	±4.394	<0.001
Platelet Count (10000/microliter)	258	±68	258	±71	0.879
Neutrophil Count (/1000)	9.390	±3.570	10.872	±3.967	<0.001
Lymphocyte count (/1000)	1.800	1.264-2.400	1.500	1.000-2.100	0.001
Neutrophil-to-lymphocyte ratio	5.07	3.35-7.82	7.34	4.17-11.00	<0.001
FBG on admission (mg/dL)	150.10	±75.70	167.59	±84.97	<0.001
C-Reactive protein (mg/dl)	9.50	5.33-16.50	16.50	7.74-31.20	<0.001
Serum albumin (g/dl)	3.76	±0.48	3.65	±0.48	0.001
Estimated glomerular filtration rate	89.32	±25.18	80.84	±27.08	<0.001
Peak Creatine Kinase MB (ng/mL)	163.0	94.0-287.0	342.50	212.00-467.00	<0.001
Peak Troponin I (ng/mL)	77.00	34.00-165.00	181.82	77.00-276.00	<0.001
B-type natriuretic peptide pg/mL	65.35	33.40-121.30	133.50	85.85-279.80	<0.001
Left Ventricular Ejection Fraction (%)	47.65	±8.03	40.18	±7.44	<0.001
Total ischemia time, min	170.0	110.0-252.0	301.5	198.0-395.0	<0.001
Infarct-related LAD	558.0	(52.3)	76.0	(50.7)	0.808
LAD artery, n Cx	132.0	(12.4)	19.0	(12.7)	

(%)	RCA	360.0	(33.7)	55.0	(36.7)	
	Other coronaries (Diagonal etc.)	17.0	(1.6)	0.0	(0.0)	
Proximal/ostial lesion for IRA, n (%)		590.0	(55.3)	112.0	(74.7)	<0.001
Stent length, mm		21.02	±8.17	28.06	±12.57	<0.001
Stent diameter, mm		3.09	±0.34	3.23	±0.50	0.011
Basal syntax score		16.37	±4.54	18.53	±3.86	<0.001
High degree AV block		28.00	(2.6)	19.00	(12.7)	<0.001

Table 2 Demographic, clinical and laboratory characteristics of all patients, patients with and without no-reflow, with p value. Abbreviations: COPD: Chronic Obstructive Pulmonary Disease; PAD: Peripheral Arterial Disease; CAD: Coronary artery disease; ASA: Acetylsalicylic acid; ACEI/ARB: Angiotensin converting enzyme inhibitor/Angiotensin receptor blocker; WBC: White Blood Cell; FBG: Fasting Blood Glucose; IRA: Infarct-related artery

	Univariate analysis of no-reflow			Multivariate analysis of no-reflow		
	Odds ratio	95% C.I.	P value	Odds ratio	95% C.I.	P value
High Grade AV block	5.382	2.924-9.908	<0.001	3.856	1.488-9.995	0.005
Neutrophil-to-lymphocyte ratio	1.076	1.044-1.108	<0.001	1.051	1.011-1.093	0.012
C-reactive protein	1.039	1.028-1.050	<0.001	1.114	1.016-1.221	0.022
Left Ventricle Ejection Fraction	0.895	0.875-0.916	<0.001	0.926	0.892-0.962	<0.001
Total ischemia time, min	1.006	1.005-1.007	<0.001	1.006	1.004-1.008	<0.001
Stent length, (per 1 mm)	1.064	1.047-1.081	<0.001	1.068	1.041-1.095	<0.001
Stent diameter, (per 1 mm)	2.678	1.695-4.230	<0.001	2.361	1.247-4.471	0.008

Table 3 Univariate and multivariate logistic regression analysis of demographic, clinical, laboratory and coronary angiographic characteristics for no-reflow prediction

Figure Legends:

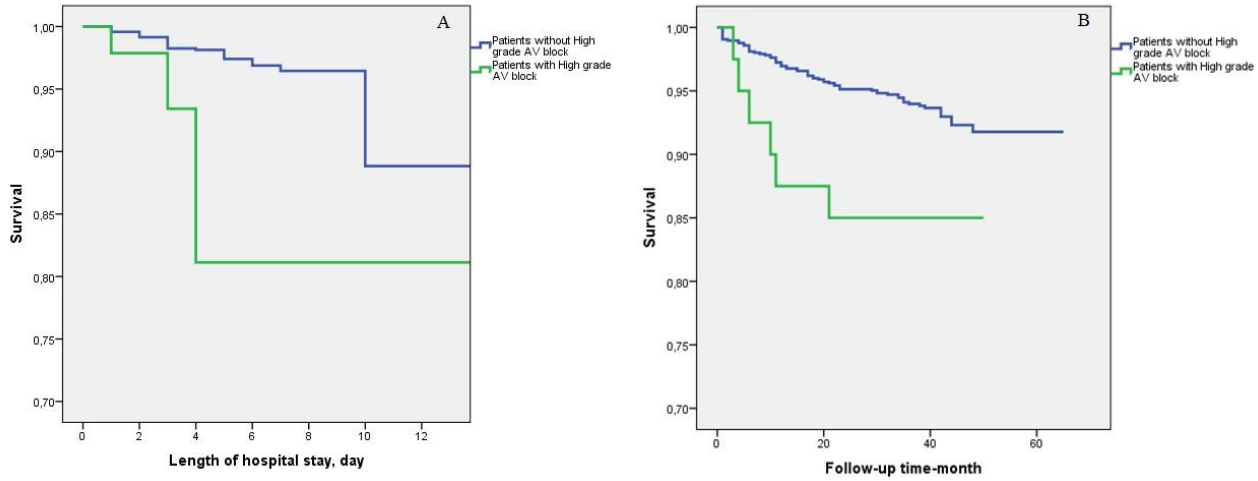

Figure 1. Kaplan meier survival analysis of in hospital(A) and long term mortality(B) in patients with and without high grade atrioventricular block.

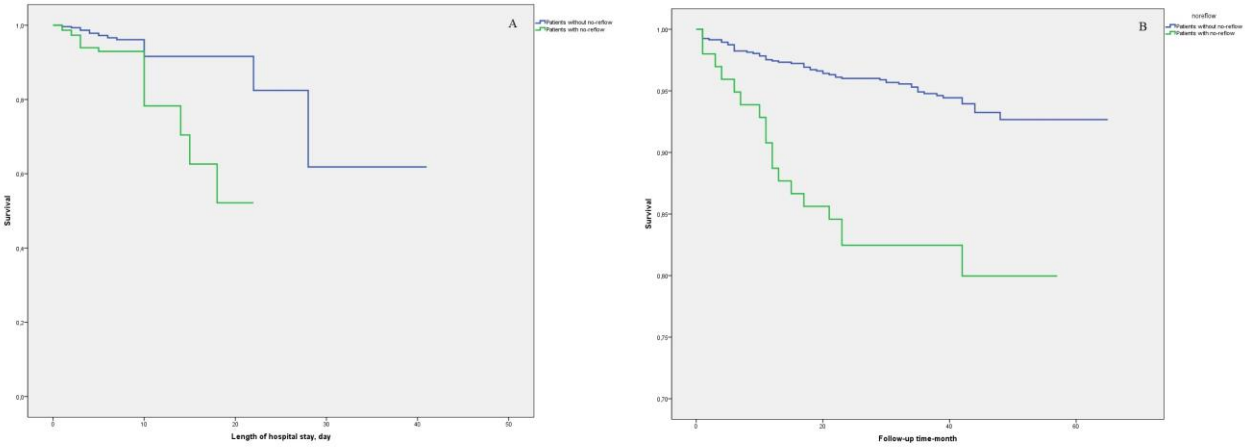

Figure 2. Kaplan meier survival analysis of in hospital(A) and long term mortality(B) in patients with and without no-reflow.