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Previous Statin Therapy Improves Clinical Outcome of Patients with ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention

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Abstract

Background: Statin treatment has been shown to reduce the risk of coronary artery disease and improve the outcome of patients with acute myocardial infarction. However, the effects of previous statin treatment on the clinical course of subsequent acute myocardial infarction remain unclear. This study was designed to investigate whether previous statin therapy influences the clinical outcome of patients with ST-Segment Elevation Myocardial Infarction (STEMI) treated with primary Percutaneous Coronary Intervention (PCI).

Methods: We evaluated the clinical outcome of 350 patients with STEMI undergoing primary PCI, of which 91 received previous statin treatment (statin group) and 259 did not (non-statin group). Myocardial perfusion, infarct size, inflammatory responses, and Major Adverse Cardiovascular Events (MACE) were evaluated.

Results: The frequency of MACE at 1 month after PCI was significantly lower in the statin group than the non-statin group (4.4% vs. 13.9%, p=0.014). Post-PCI peak creatine kinase was significantly lower in the statin group median, (interquartile range): (1246 [504-3301] vs. 2235 [952-4083] IU/ml; p=0.002), whereas peak high-sensitivity C-reactive protein did not significantly differ between the two groups (p=0.287). The frequency of ST-segment resolution after PCI was significantly higher in the statin group (90.1% vs. 76.8%; p=0.006), as was the frequency of Thrombolysis in Myocardial Infarction grade 3 coronary flow (p=0.008). Myocardial blush grade was similar in both groups (p=0.839). Multivariate logistic regression analysis revealed previous statin treatment, hs-CRP, blood glucose, and age to be independent predictors of MACE.

Conclusion: Previous statin therapy enhances coronary flow, reduces infarct size, and improves clinical outcome of STEMI patients treated with primary PCI.

Keywords: Statin treatment; STEMI; MACE

Abbreviations

STEMI: ST-Segment Elevation Myocardial Infarction; PCI: Percutaneous Coronary Intervention; MACE: Major Adverse Cardiovascular Events; hs-CRP: High-sensitivity C-Reactive Protein; Statin: 3-hydroxy-3-methylglutaryl Coenzyme A Reductase Inhibitor; ACS: Acute Coronary Syndrome; ECG: Electrocardiographic; CK: Creatine Kinase; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; HbA1c: Glycated Haemoglobin; eGFR: Estimated Glomerular Filtration Rate; TIMI: Thrombolysis in Myocardial Infarction; MBG: Myocardial Blush Grade; CABG: Coronary Artery Bypass Graft; ACEIs: Angiotensin-Converting Enzyme Inhibitors; ARBs: Angiotensin II Receptor Blockers; OR: Odds Ratio

Introduction

Lipid-lowering therapy is an important component of a comprehensive strategy to reduce the risk of cardiovascular disease. The benefits of 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor (statin) therapy for primary and secondary prevention of coronary artery disease have been firmly established by large-sample, randomized, controlled clinical trials [1-6].

Statin treatment was also reported to improve clinical prognosis of patients with Acute Coronary Syndrome (ACS) and the beneficial effect of statin was more prominent in aggressive rather than moderate lipid lowering [7,8]. Moreover, pretreatment with statin 12 hours prior to Percutaneous Coronary Intervention (PCI) was associated with a significant reduction in adverse clinical outcomes after coronary intervention in patients with ACS [9]. However, it is less clear whether long-term statin treatment before the onset of ischemic events improves the clinical outcome of patients with ST-Segment Elevation Myocardial Infarction (STEMI).

In this study we compared the frequency of adverse events and the parameters of coronary perfusion, myocardial necrosis, and inflammation between patients with STEMI who were taking statins prior to the event and those without the history of statin use.

Material and Methods

Study population

The study population consisted of patients with STEMI admitted to Kansai Medical University Hirakata Hospital between January 2006 and December 2012. All patients underwent coronary reperfusion therapy by primary PCI. The diagnosis of STEMI required the presence of the

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following three criteria: (1) Typical angina symptoms lasting more than 30 minutes, (2) Typical electrocardiographic (ECG) changes (i.e., ST-segment elevation >0.1 mV in at least 1 standard or 2 precordial leads), and (3) Creatine kinase (CK) greater than three times the normal upper limit. Patients with cardiopulmonary arrest during transportation to hospital were excluded from this study. Other exclusion criteria were comorbid malignant, infectious, chronic inflammatory or autoimmune diseases.

Study design

The ethics committee of Kansai Medical University Hirakata Hospital approved the study protocol. We divided the patients into two groups according to their status of statin treatment as determined by detailed interviews and/or medical records. Additional data including age, gender, body mass index, blood pressure, heart rate, smoking history, and medications other than statins were recorded. Blood samples were obtained on admission to measure baseline Low Density Lipoprotein (LDL) cholesterol, High Density Lipoprotein (HDL) cholesterol, Triglycerides, CK, high-sensitivity C-Reactive Protein (hs-CRP), Glucose, Glycated Hemoglobin (HbA1c), Creatinine, estimated Glomerular Filtration Rate (eGFR), Hemoglobin (Hb), and white blood cell counts. Hypertension was defined as systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg, or the current use of antihypertensive agents. Diabetes mellitus was defined as a fasting blood glucose ≥ 126 mg/dl and/or a 2-hour blood glucose ≥ 200 mg/dl, HbA1c ≥ 6.5%, or the current use of anti-diabetic medication (oral hypoglycemic drugs or insulin injection). Pre-infarction angina was defined as cardiac symptoms lasting <30 minutes within 48 hours of the onset of STEMI. Cardiogenic shock was defined as persistent hypotension (systolic blood pressure <90 mmHg) that required inotropes or intra-aortic balloon pumping to maintain blood pressure to be >90 mmHg.

Major Adverse Cardiovascular Events (MACE) were a composite of death, non-fatal myocardial infarction, new or worsening congestive heart failure, and non-fatal stroke up to 1 month. Re-infarction was defined as typical angina symptoms lasting more than 30 minutes associated with CK greater than three times the normal upper limit and/or cardiac troponin I greater than 0.04 ng/mL. Heart failure was defined as dyspnea and/or edema associated with pulmonary congestion that required specific treatment. In addition, ST-segment resolution on electrocardiography, postprocedural final Thrombolysis in Myocardial Infarction (TIMI) flow grade, Myocardial Blush Grade (MBG), peak CK, and peak hs-CRP were evaluated after PCI. ST-segment resolution was defined as ST elevation <50% of the initial value on presentation. We recorded electrocardiography on admission as well as during and immediately after PCI to evaluate ST-segment resolution after reperfusion. Flow in the stenotic vessel(s) after PCI was graded using the TIMI flow classification. An experienced interventional cardiologist determined MBG by visual assessment of contrast opacification within the myocardial territory of the stenotic vessel(s) on the final angiographic examination. CK was measured every 6 hours and hs-CRP was measured every 24 hours.

Percutaneous coronary intervention

All patients received chewable aspirin (100 mg) and a loading dose of oral clopidogrel (300 mg) or ticlopidine (200 mg) before coronary angiography. Some patients received intravenous infusion of nicorandil at 6 mg/h for 24 h after admission at the operator's discretion. Before coronary angiography, 3000 U heparin was administered. When the stenotic coronary artery was identified, 7000 U heparin was administered and PCI was initiated. Additional heparin was administered to maintain an activated clotting time >300 s during the procedure. Experienced interventional cardiologists performed all primary PCI procedures. The type of stent implanted (bare-metal or drug-eluting) was determined by the operator. After PCI, patients were monitored in the coronary intensive care unit until clinical status stabilized. All patients were prescribed aspirin (100 mg/day), clopidogrel (75 mg/day) or ticlopidine (200 mg/day), and statins. If necessary, elective PCI or Coronary Artery Bypass Graft (CABG) surgery was performed before hospital discharge. Coronary lesions were evaluated in at least two nonforeshortened angiographic views at end-diastole. Stenosis was defined as 75% or greater reduction in lumen diameter.

Statistical analysis

Data are presented as mean ± standard deviation or median (interquartile range) depending on the distribution. The Welch's t-test or Wilcoxon test was used to compare group means or medians as appropriate. Categorical variables are expressed as number (percentage) and were compared using χ^2 test or Fisher's exact test as appropriate. Independent predictors of MACE were identified using multivariate logistic regression analyses. In this model, age, gender, and factors with a P value <0.05 in univariate analysis were included (except those that may introduce multicollinearity). All statistical analyses were performed using JMP 10.0.2 for Windows (SAS Institute Inc., Cary, NC, USA). A two-sided P value <0.05 was considered as statistically significant.

Results

Of 358 patients meeting all inclusion criteria and no exclusion criteria (257 men and 93 women; mean age, 67.4 ± 11.7 years), 91 had been treated with statin (statin group), 259 were statin-naïve (non-statin group), and the status of statin use could not be determined on admission in 8 patients (who were excluded from the analysis). In the statin group, 36% were taking pravastatin, 24% rosuvastatin, 23% atorvastatin, 8% pitavastatin, 6% simvastatin, and 3% fluvastatin.

In baseline clinical characteristics, mean age, proportion of females, prevalence of diabetes mellitus, prevalence of hypertension, histories of PCI/CABG, and proportions receiving aspirin, angiotensin-converting–enzyme inhibitors (ACEIs), angiotensin II receptor blockers (ARBs), beta-blockers, or calcium channel blockers were significantly higher in the statin group. The proportion of current or past smokers was lower in the statin group (Table 1). In baseline laboratory data, LDL cholesterol levels were significantly lower and HbA1c levels were significantly higher in the statin group. Other baseline laboratory data were similar between the two groups (Table 2). In angiographic observations and treatment procedures, there was no statistically significant difference between the statin group and the non-statin group (Table 3).

During the 1-month monitoring period, MACE occurred in 4.4% of statin group patients and 13.9% of non-statin group patients, and chronic statin treatment prior to STEMI significantly reduced the risk of MACE (p=0.014) (Figure 1). The incidence rates of MACE during follow-up are summarized in Table 4. In the parameters of coronary perfusion and myocardial necrosis, the proportion of patients with ST segment resolution or TIMI grade 3 flow was significantly higher (p=0.006 and p=0.008, respectively), and post-procedural peak CK levels were significantly lower in the statin group (1246 [504-3301] vs.

	Statin group (n=91)	Non-statin group (n=259)	P
Male (%)	56 (61.5)	201 (77.6)	0.003
Age (years)	69.7 ± 10.7	66.6 ± 11.9	0.019
Body mass index	23.6 ± 3.4	23.9 ± 3.6	0.582
Smoking (%)	55 (60.4)	183 (71.8)	0.045
Hypertension (%)	69 (75.8)	166 (64.1)	0.040
Diabetes mellitus (%)	45 (49.5)	78 (30.1)	0.001
Post PCI (%)	27 (29.7)	24(9.3)	<0.001
Post CABG (%)	5 (5.5)	1(0.4)	0.001
Heart rate (min)	76.1 ± 19.4	78.7 ± 20.7	0.298
Systolic blood pressure (mmHg)	125.1 ± 33.4	130.3 ± 32.7	0.193
Diastolic blood pressure (mmHg)	68.0± 19.8	75.9 ± 20.9	0.001
Preinfarction angina (%)	53 (58.2)	127 (49.0)	0.131
Reperfusion time (h)*	4 [3−6]	4 [3−6]	0.807
Killip class (%)	-	-	0.136
I	79 (86.8)	233 (90.0)	-
II	3 (3.3)	1 (0.4)	-
III	4 (4.4)	6 (2.3)	-
IV	5 (5.5)	19 (7.3)	-
Bradyarrhythmia (%)	12 (13.2)	35 (13.5)	0.937
VT/Vf (%)	5 (5.5)	2 (1.0)	0.006
Medication on admission Aspirin (%)	37 (40.7)	24 (9.3)	<0.001
ACEI/ARB (%)	35 (41.2)	45 (18.7)	<0.001
Beta-blocker (%)	19 (22.4)	12 (5.0)	<0.001
Calcium blocker (%)	34 (40.0)	41 (17.0)	<0.001

Data presented as number of patients (%), mean (standard deviation), or median (IQR) as appropriate. PCI: Percutaneous Coronary Intervention; CABG: Coronary Artery Bypass Grafting; VT/Vf: Ventricular Tachycardia/Ventricular Fibrillation; ACEI/ARB: Angiotensin Converting Enzyme Inhibitor/Angiotensin II Receptor Blocker
*Onset of chest pain to reperfusion time

Table 1: Baseline clinical characteristics.

	Statin group (n=91)	Non-statin group (n=259)	P
LDL cholesterol (mg/dl)	99 [80–120]	124 [106–150]	<0.001
HDL cholesterol (mg/dl)	46 [37–51]	43 [36–53]	0.372
Triglyceride (mg/dl)	117 [85–168]	111 [77–178]	0.732
Hemoglobin (g/dl)	13.0 [11.9–14.1]	13.8 [12.4–15.0]	0.006
Hs-CRP (mg/dl)	0.13 [0.06–0.38]	0.16 [0.07–0.70]	0.123
White blood cell (/μl)	9400 [7500–12000]	9700 [7600–12100]	0.817
Creatinine (mg/dl)	0.81 [0.65–1.04]	0.82 [0.68–1.00]	0.923
eGFR (%)	64 [50–84]	69 [53–83]	0.423
Glucose (mg/dl)	164 [131–220]	153 [125–203]	0.104
HbA1c (%)	6.0 [5.4–7.0]	5.5 [5.2–6.4]	<0.001

Data expressed as median (IQR). LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; Hs-CRP: High Sensitivity C-Reactive Protein; eGFR: Estimated Glomerular Filtration Rate; HbA1c: Glycated Hemoglobin

Table 2: Baseline laboratory data.

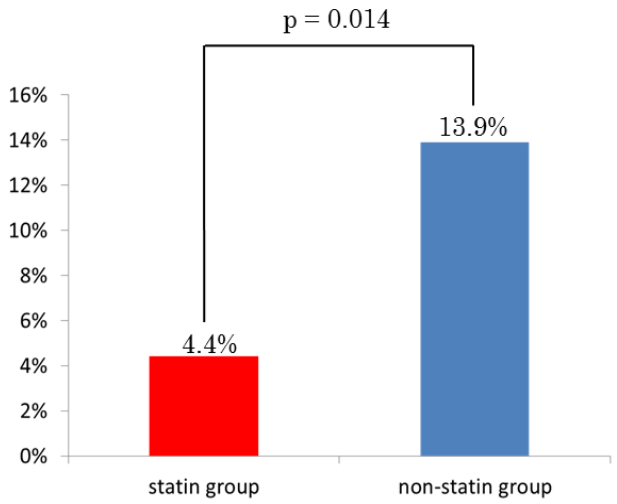

Figure 1: Comparison of the frequency of MACE between the statin group and the non-statin group. The frequency of MACE was lower in the statin group (4.4% vs. 13.9%; p=0.014).

2235 [952-4083] IU/ml, p=0.002). There was no statistically significant difference in post-procedural MBG and hs-CRP between the two groups (Table 5).

Baseline characteristics of patients with or without MACE during a 1-month follow-up period are shown in Table 6. Age, hs-CRP, creatinine, blood glucose, and proportions of ACEI/ARB/beta-blocker users were significantly higher and Hb, eGFR, and the proportion of previous statin treatment were significantly lower in the MACE group. Table 7 shows the results of multivariate logistic regression analysis of variables with P<0.05 on univariate analysis. eGFR was excluded from multivariate analysis due to collinearity with creatinine concentration. Previous statin treatment was an independent predictor of lower MACE frequency (Odds Ratio [OR], 0.13; 95% CI, 0.03-0.40; P<0.001). Older age, higher hs-CRP, and higher blood glucose were also associated with increased MACE (OR, 95% CI, age: 1.04, 1.01-1.09, p=0.023; hs-CRP: 1.14, 1.05-1.24, p=0.002; glucose: 1.01, 1.00-1.01, p=0.012).

Discussion

In the present study, we examined the effect of previous statin treatment on myocardial perfusion and necrosis after primary PCI and the frequency of MACE during a 1-month follow-up period in patients with STEMI. Our results indicate that previous statin treatment was associated with better myocardial perfusion, smaller infarct size, and reduced frequency of short-term MACE in STEMI patients treated with primary PCI.

Previous studies have shown that short-term statin treatment improves prognosis in patients with stable angina or ACS. The ARMYDA-ACS trial reported that statin treatment 12 hours before PCI in patients with non-STEMI resulted in an 88% overall reduction in MACE at 1 month [9], and the ARMYDA-RECAPTURE trial reported that reloading statin 12 hours before PCI in patients with stable angina/non-STEMI who were already receiving chronic statin therapy resulted in an 82% overall reduction in MACE at 1 month [10]. However, it is less clear whether chronic statin therapy before the onset of STEMI has beneficial effect on the outcome of patients. Yun et al. reported that chronic statin treatment reduced periprocedural MI and in-hospital MACE in patients with unstable angina/non-STEMI treated with PCI [11]. Lev et al. reported that STEMI patients treated with primary PCI who received previous statin therapy had a lower 30-day mortality than those without previous statin therapy and that chronic statin use was an independent negative predictor of short-term mortality [12]. In our present study, previous statin treatment was associated with reduced frequency of short-term MACE. Since the benefits of statin treatment for the primary and the secondary prevention of coronary artery disease are well established [1-6], these results collectively suggest that statin treatment both reduces ACS risk and exerts cardioprotective effects following ACS.

Although the mechanistic basis for the beneficial effects of previous statin therapy in patients with STEMI is not clear at present, one possible explanation is the improved coronary perfusion and the reduced infarct size. In our present study, chronic statin therapy was associated with higher frequency of ST-segment resolution and TIMI grade 3 flow and lower peak CK levels. Consistently, Oduncu et al. reported that previous statin therapy in patients with STEMI was associated with higher frequency of ST-segment resolution and lower peak CK-MB levels [13], and Ishii et al. also reported essentially the same results in patients with AMI [14]. Thus, chronic statin therapy may improve the outcome of STEMI patients by enhancing the restoration of coronary flow and reducing the infarct size. To our knowledge, this study is the first to show that previous statin treatment enhances myocardial perfusion, reduces infarct size, and improves clinical outcome following primary PCI in the same patient population with STEMI.

In addition to well documented lipid-lowering effects, statins also improve endothelial function, stabilize plaques, and reduce inflammation [15-17]. It was also reported that statin pretreatment is associated with reduced postprocedural hs-CRP in ACS patients treated with PCI [11]. However, we found no such reduction in baseline or periprocedural serum CRP in statin group patients. Kai et al. reported that pravastatin (a hydrophilic statin) decreased CRP after switching from simvastatin (a lipophilic statin) [18]. Moreover, aggressive lipid-lowering treatment with atorvastatin reduced CRP compared to pravastatin in patients with coronary artery disease [19]. Thus, pharmacologic lipophilicity and dose of the statin appears to influence CRP levels. The patients enrolled in this study were treated with a variety of different statins at different doses, and this may be one of the reason why we did not observe reduced CRP levels in the statin group.

Our study has several limitations. First, this is not a prospective randomized study and the sample size is small. Second, the decision for coronary intervention was made by multiple physicians, and some bias may be inevitable. Third, there were differences in the baseline characteristics between the two groups, which may have influenced the outcome. It should be noted, however, that patients in the statin group appear to be at higher risk for coronary artery disease.

In conclusion, previous statin treatment enhanced coronary perfusion, reduced infarct size, and improved short-term clinical outcome after primary PCI in patients with STEMI. Chronic statin therapy is therefore effective both for the primary/secondary prevention of STEMI and cardioprotection in STEMI patients.

	Statin group (n=91)	Non-statin group (n=259)	P
Multi-vessel disease (%)	42 (46.2)	104 (40.2)	0.318
Target vessel (%)	-	-	0.437
LMT	2 (2.2)	1 (0.4)	-
LAD	42 (46.2)	125 (48.3)	-
LCX	9 (9.9)	28 (10.8)	-
RCA	38 (41.8)	105 (40.5)	-
TIMI flow grade before PCI (%)	-	-	0.084
TIMI 0	43 (47.3)	161 (62.2)	-
TIMI 1	13 (14.3)	22 (8.5)	-
TIMI 2	26 (28.6)	55 (21.2)	-
TIMI 3	9 (9.9)	21 (8.1)	-
Collateral flow grade before PCI (%)	-	-	0.730
Collateral flow 0	60 (65.9)	159 (61.4)	-
Collateral flow 1	21 (23.1)	62 (23.9)	
Collateral flow 2	6 (6.6)	27 (10.4)	-
Collateral flow 3	4 (4.4)	11 (4.3)	-
Use of drug-eluting stent (%)	10 (11.0)	27 (10.4)	0.880
Nicorandil before PCI (%)	78 (85.7)	228 (88.0)	0.556
Intra-aortic balloon pump (%)	8 (8.8)	23 (8.9)	0.980
Temporary pacemaker (%)	18 (19.8)	65 (25.1)	0.305

Date expressed as number of patients (% of group). LMT: Left Main Trunk; LAD: Left Anterior Descending; LCX: Left Circumflex Artery; RCA: Right Coronary Artery; TIMI: Thrombolysis in Myocardial Infarction; PCI: Percutaneous Coronary Intervention

Table 3: Coronary angiographic and procedural characteristics.

	Statin group (n=91)	Non-statin group (n=259)	P
MACE (%)	4 (4.4%)	36 (13.9%)	0.014
Death (%)	2 (50%)	17 (47.2%)	-
Non-fatal MI (%)	0 (0%)	5 (13.9%)	-
Heart failure (%)	2 (50%)	13 (36.1%)	-
Non-fatal stroke (%)	0 (0%)	1 (3%)	-

Table 4: Comparison of the frequency of major adverse cardiovascular events (MACE) between the statin group and the non-statin group.

	Statin group (n=91)	Non-statin group (n=259)	P
ST resolution (%)	82 (90.1)	199 (76.8)	0.006
Final TIMI flow grade 3 (%)	87 (95.6)	220 (84.9)	0.008
MBG (%)	53 (58.2)	154 (59.5)	0.839
Peak CK (U/L)	1246 [504–3301]	2235 [952–4083]	0.002
Peak CRP (mg/dl)	7.4 [3.7–11.1]	8.0 [4.9–11.8]	0.287

PCI: Percutaneous Coronary Intervention; TIMI: Thrombolysis in Myocardial Infarction; MBG: Myocardial Blush Grade; CK: Creatine Kinase; CRP: C-Reactive Protein

Table 5: Parameters of coronary perfusion and infarct size after PCI.

	Event group (n=40)	Non-event group (n=310)	P
Male (%)	26 (65.0)	231 (74.5)	0.200
Age (years)	72.2 ± 12.8	66.8 ± 11.4	0.014
Body mass index	23.7 ± 3.9	23.8 ± 3.5	0.880
Smoking (%)	24 (63.2)	214 (69.5)	0.427
LDL cholesterol (mg/dl)	110 [82–136]	117 [98–143]	0.099
HDL cholesterol (mg/dl)	44 [37–56]	44 [36–53]	0.673
Triglyceride (mg/dl)	101 [62–137]	115 [79–179]	0.139
Hemoglobin (g/dl)	12.1 [10.7–13.5]	13.8 [12.6–14.8]	<0.001
Hs-CRP (mg/dl)	0.89 [0.21–6.51]	0.14 [0.06–0.45]	<0.001
White blood cell (μl)	9600 [7530–14200]	9650 [7600–12000]	0.298
Creatinine (mg/dl)	1.01 [0.79–1.62]	0.8 [0.66–0.98]	<0.001
eGFR (%)	53 [28–67]	69 [55–85]	<0.001
Glucose (mg/dl)	189 [144–251]	154 [124–204]	0.004
HbA1c (%)	5.7 [5.3–7.5]	5.6 [5.2–6.6]	0.110
Hypertension (%)	30 (75.0)	205 (66.1)	0.261
Diabetes mellitus (%)	17 (42.5)	106 (34.2)	0.300
Post PCI (%)	9 (22.5)	42 (13.6)	0.131
Post CABG (%)	1 (2.5)	5 (1.6)	0.684
Preinfarction angina (%)	18 (45.0)	162 (52.3)	0.387
Multi-vessel disease (%)	20 (50.0)	126 (40.7)	0.259
Onset of chest pain to reperfusion (h)	4 [2.5–7]	4 [3–6]	0.840
Medication on admission			
Statin (%)	4 (10.0)	87 (28.1)	0.014
Aspirin (%)	11 (27.5)	50 (16.1)	0.074
ACEI/ARB (%)	16 (43.2)	64 (22.2)	0.005
Beta-blocker (%)	7 (18.9)	24 (8.3)	0.038
Calcium blocker (%)	13 (35.1)	62 (21.5)	0.063

Table 6: Baseline characteristics of patients who developed Major Adverse Cardiovascular Events (MACE) and those free of MACE.

	Odds ration	95% CI	P
Age	1.04	1.01−1.09	0.023
Hemoglobin	0.83	0.67−1.03	0.085
Hs-CRP	1.14	1.05−1.24	0.002
Creatinine	1.18	0.95−1.46	0.140
Glucose Statin	1.01 0.13	1.00−1.01 0.03−0.40	0.012 <0.001
ACEI/ARB	1.96	0.80−4.71	0.141
Beta-blocker	2.90	0.85−9.51	0.088

CI: Confidence Interval

Table 7: Multivariate logistic regression analysis: Independent predictors of major adverse cardiovascular events within 30 days.

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