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ORIGINAL ARTICLE

Relationship between low-density lipoprotein levels on admission and 1-year outcome in patients with acute ST-segment-elevation myocardial infarction

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Acute ST segment myocardial infarction (ASTEMI);
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Statins

Abstract This study assessed the relationship between low-density lipoprotein cholesterol (LDL-C) levels on admission and the incidence of major adverse cardiovascular events (MACE) in patients with acute ST-segment-elevation myocardial infarction (ASTEMI). Patients with ASTEMI who had a lipid profile tested within 24 hours of symptom onset were enrolled. They were stratified into high and low LDL-C groups according to whether their LDL-C was above ($n = 501$) or below ($n = 575$) the median level, respectively. The incidence of MACE, cardiovascular death, non-fatal MI, revascularization, and stroke was compared between the groups at 1 month, 6 months, and 1 year. Survival analysis and Cox proportional hazard analysis were performed. In-hospital use of beta blockers was better in the high than in the low LDL-C group (76.6% vs. 69.7%, $p = 0.01$). Statin use was significantly higher in the high than in the low LDL-C group during follow-up (86.8% vs. 80.0%, $p = 0.003$ at1 month; 71.6% vs. 62.4%, $p = 0.002$ at 6 months; 67.8% vs. 61.2%, $p = 0.03$ at 1 year). The incidence of MACE on follow-up at 1 month was higher in the low than in the high LDL-C group (12.0% vs. 8.1%, $p = 0.04$). At 1 year, survival was not significantly different between the groups. Cox proportional hazards analysis indicated that the incidence of MACE was significantly associated with hypertension, current smoking, high-density lipoprotein cholesterol (HDL-C), in-hospital use of beta blockers, and statin use on follow-up ($p < 0.01$). LDL-C levels on admission in patients with ASTEMI had no significant effect on the 6-month and 1-year incidence of MACE, but the incidence of MACE was significantly higher in the low LDL-C group at 1 month. It would be relevant to further investigate the HDL-C level on admission, in-hospital use of beta blockers, and statin use during follow-up in relation to MACE.
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Introduction

It is well known that elevated low-density lipoprotein cholesterol (LDL-C) is a major risk factor for cardiovascular disease [1]. A number of randomized controlled clinical trials, including 4S [2], CARE [3], LIPID [4], WOSCOP [5], and AFCAPS/TexCAPS [6], confirmed that a reduction in LDL-C by statins reduced the risk of cardiovascular events in patients with coronary heart disease and cardiovascular risk factors. It appears that lower LDL-C levels are associated with better prognosis. In practice, many patients still have a high risk of major adverse cardiovascular events (MACE) despite aggressive statin therapy that lowers LDL-C [7—9] and contributes to achievement of the LDL-C target level [2—5]. Mouaz et al. found that a lower 3-year survival in patients with non-ST-segment-elevation myocardial infarction (NSTEMI) was associated with lower LDL-C on admission [10]. Hypocholesterolemia is associated with poorer clinical outcomes in patients with high cardiovascular risk and coronary heart disease, and in patients with some other diseases, such as chronic kidney disease and malignancy [11—18]. Low LDL-C on admission to hospital and LDL-C-lowering with statins may not have similar effects in reducing MACE. However, few studies have assessed the impact of LDL-C levels on admission in patients with acute ST-segment-elevation myocardial infarction (ASTEMI). In the present study we investigated the clinical characteristics, in-hospital management, treatment after discharge, and outcome of patients with ASTEMI.

Methods

Study population

A prospective, multicenter study conducted on patients with ASTEMI in LiaoNing Province in northeast China included 1429 consecutive patients who were admitted to 20 hospitals between May 2009 and May 2010. ASTEMI was defined according to the American College of Cardiology/American Heart Association ASTEMI criteria [19]. Patients were enrolled if they had a specific time of symptom onset, with symptoms including chest pain, dyspnea, and syncope, and if they were admitted to the hospital and had their lipid profile measured within 24 hours of symptom onset. Patients who did not meet these criteria, who were diagnosed with NSTEMI, or who had incomplete data were excluded. Finally, a total of 1076 patients were enrolled. The protocol was approved by the ethics committee of the study hospitals, and all patients gave written informed consent.

Study design

The patients were stratified into two groups according to their LDL-C level on admission: one group with LDL-C below the median for all patients, and the other with LDL-C above the median. Patients received emergency percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG), thrombolysis, and medical care according to their conditions and the availability of services in the admitting hospital. Basic demographic and clinical data were collected, including age, gender, body mass index (BMI; kg/m^2), smoking history, history of disease and medication, blood analysis [LDL-C, high-density lipoprotein cholesterol (HDL-C), total cholesterol (TC), triglyceride (TG), peak troponin, and creatinine]. In addition, patient status on admission (cardiogenic shock, ventricular arrhythmia, and Killip class) and methods of treatment were determined.

All subjects attended follow-up visits at 1 month, 6 months, and approximately 1 year after hospital discharge. Information on drug use and the incidence of MACE were recorded. In the present study, MACE was defined as cardiovascular death, non-fatal MI, revascularization, or stroke. The primary endpoint was the rate of 1-year MACE, and the secondary endpoints were the rate of 1-month and 6-month follow-up MACE.

Statistical analysis

Continuous data are expressed as mean $\pm$ standard deviation and were analyzed using the Student t test. Categorical data are expressed as n (%) and were analyzed using the χ^2 test or the Fisher exact test. Survival curves were prepared using the Kaplan—Meier method with the log-rank test. Univariate analysis of age, gender, BMI, current smoking, diabetes, hypertension, LDL-C, HDL-C, TC, TG, and other factors was performed to determine the predictors for MACE. A multiple Cox proportional hazard model was used to determine associations between significant factors identified in the univariate analysis and those identified in the MACE. A p value <0.05 was considered statistically significant. All analyses were performed using SPSS version 13.0 (SPSS Inc., Chicago, IL, USA).

Results

The median LDL-C level for all subjects on admission was 2.94 mmol/L; 501 patients had LDL-C $>$2.94 mmol/L (high LDL-C group), and 575 patients had LDL-C $\leq$2.94 mmol/L (low LDL-C group). The median follow-up duration was 274.07 $\pm$ 135.36 days. During the follow-up period, three patients died, one of a serious infection, one due to a traffic accident, and one due to gastrointestinal bleeding. In addition, 37 patients were lost to follow-up. The follow-up rate for the high and low LDL-C groups was 96.6% and 96.0%, respectively.

Baseline characteristics

Baseline characteristics are summarized in Table 1. BMI and the prevalence of diabetes were significantly higher in the high than in the low LDL-C group (25.30 $\pm$ 3.09 vs. 24.77 $\pm$ 3.40, $p < 0.001$ and 21.0% vs. 16.3%, $p = 0.04$, respectively). The proportion of men was significantly higher in the low than in the high LDL-C group (78.3% vs. 67.5%, $p < 0.001$). There were no significant differences with regard to age, history of disease, and modes of treatment between the groups.

Table 1 Baseline characteristics of the study patients.

Variable	Low-density lipoprotein cholesterol		*p*
	>2.94 mmol/L	≤2.94 mmol/L	
Patients	501	575	
Age (y)	62.07 ± 12.57	63.20 ± 12.01	0.13
Age ≥ 65 y	221 (44.1)	269 (46.8)	0.38
Male	338 (67.5)	450 (78.3)	<0.001
Body mass index (kg/m^2)	25.30 ± 3.09	24.77 ± 3.40	<0.001
Previous angina pectoris	131 (26.1)	166 (28.9)	0.32
Previous myocardial infarction	29 (5.8)	40 (7.0)	0.44
Previous stroke	52 (10.4)	82 (14.3)	0.06
Chronic heart failure	5 (1.0)	4 (0.7)	0.59
Diabetes	105 (21.0)	93 (16.3)	0.04
Medication for control of blood sugar[a]	82 (16.4)	74 (12.9)	0.10
Hypertension	231 (46.1)	254 (44.2)	0.53
Lipid-lowering therapy	17 (3.4)	14 (2.4)	0.35
Chronic renal failure	6 (1.2)	12 (2.1)	0.26
Current smoker	223 (44.5)	266 (46.3)	0.57
Family history of coronary heart disease	102 (20.4)	124 (21.6)	0.63
Previous percutaneous coronary intervention	12 (2.4)	26 (4.5)	0.06
Previous coronary artery bypass grafting	0	0	0.99

Data are presented as *n* (%) or mean ± SD.
[a] Hypoglycemic medication or insulin therapy.

Clinical characteristics, treatment, and laboratory findings on admission

The clinical characteristics, treatment, and laboratory findings on admission are summarized in Table 2. There were no significant differences in cardiogenic shock and ventricular arrhythmia on admission, Killip class, thrombolysis, thrombolytic reperfusion, emergency and selective PCI, vessel with PCI, CABG, peak troponin, TC, creatinine, and ejection fraction between the groups. Levels of HDL-C and TG on admission were significantly higher in the high than in the low LDL-C group (1.31 ± 0.41 vs. 1.23 ± 0.39 mmol/L, $p = 0.001$ and 2.02 ± 1.58 vs. 1.66 ± 1.49 mmol/L, $p < 0.001$, respectively).

Medications

The medications used in the hospital and after 1-month, 6-month, and approximately 1-year follow-up are summarized in Table 3. Use of aspirin, clopidogrel, statins, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers (ACEI/ARB), glycoprotein IIb/IIIa inhibitors, and low-molecular-weight heparin in hospital did not significantly differ between the groups. The in-hospital use of beta blockers was greater in the high than in the low LDL-C group (76.6% vs. 69.7%, $p = 0.01$). During follow-up, there were no significant differences in the use of aspirin, clopidogrel, ACEI/ARB, or beta blockers between the groups. However, statin use was significantly higher in the high than in the low LDL-C group during follow-up (86.8% vs. 80.0%, $p = 0.003$ at 1 month; 71.6% vs. 62.4%, $p = 0.002$ at 6 months; 67.8% vs. 61.2%, $p = 0.03$ at 1 year).

MACE in hospital and during follow-up

The incidence of MACE in the hospital and during 1-year follow-up is summarized in Table 4. The incidence of MACE was significantly higher in the low than in the high LDL-C group at 1-month follow-up (12.0% vs. 8.1%, $p = 0.04$). The incidence of MACE in the hospital and at 6-month and 1-year follow-up did not significantly differ between the groups.

Survival curves and Cox proportional hazard analysis

Survival curves are depicted in Fig. 1. At 1 year, the survival rate did not significantly differ between the groups. According to a univariate analysis, the independent predictors of MACE were age, gender, diabetes, hypertension, current smoking, HDL-C, statin use, and beta blocker use in hospital (Table 5). A multiple Cox proportional hazard model was prepared to identify independent predictors of MACE using factors that were significant in the univariate analysis. The independent predictors were hypertension [hazard ratio (HR) 1.517, 95% confidence interval (CI) 1.061—2.170, $p = 0.022$], current smoking (HR 2.440, 95% CI 1.599—3.725, $p < 0.001$), HDL-C (HR 0.306, 95% CI 0.173—0.541, $p < 0.001$), beta blocker use in hospital (HR 0.66, 95% CI 0.457—0.955, $p = 0.027$), and statin use (HR 0.602, 95% CI 0.423—0.856, $p = 0.005$) (Table 6).

Discussion

Our results demonstrate that LDL-C levels on admission in patients with ASTEMI were not significantly associated with the 1-year incidence of MACE. However, the incidence of

Table 2 Clinical characteristics, treatment, and laboratory findings for the study patients.

Variable	Low-density lipoprotein cholesterol		*p*
	>2.94 mmol/L	≤2.94 mmol/L	
Patients	501	575	
Cardiogenic shock on admission	11 (2.2)	21 (3.7)	0.16
Ventricular arrhythmia on admission	51 (10.2)	73 (12.7)	0.20
Killip class			0.21
I	426 (85.0)	468 (81.4)	
II	43 (8.6)	65 (11.3)	
III	21 (4.2)	21 (3.7)	
IV	11 (2.2)	21 (3.7)	
Thrombolytic therapy[a]	137 (27.3)	136 (23.7)	0.17
Thrombolytic reperfusion[b]	118 (23.6)	112 (19.5)	0.10
Urgent PCI[c]	142 (28.3)	185 (32.2)	0.17
Selective PCI[d]	56 (11.2)	55 (9.6)	0.39
Vessel with PCI			0.58
Left main artery	1 (0.50)	2 (0.8)	
Left anterior descending artery	90 (44.8)	125 (50.6)	
Left circumflex artery	32 (15.9)	38 (15.4)	
Right coronary artery	78 (38.8)	82 (33.2)	
Coronary artery bypass grafting	1 (0.2)	0	0.99
Peak troponin (mmol/L)	20.45 ± 39.03	22.37 ± 27.58	0.35
High-density lipoprotein cholesterol (mmol/L)	1.31 ± 0.41	1.23 ± 0.39	0.001
Total cholesterol (mmol/L)	5.69 ± 1.00	5.07 ± 19.1	0.47
Triglyceride (mmol/L)	2.02 ± 1.58	1.66 ± 1.49	<0.001
Creatinine	78.37 ± 35.05	81.06 ± 33.11	0.2
Ejection fraction (%)	50.69 ± 8.15	50.93 ± 8.94	0.64

Data are presented as *n* (%) or mean ± SD.
PCI = percutaneous coronary intervention.
[a] The thrombolytic protocol was 1.5 mU urokinase over 30 minutes or an 8-mg bolus of recombinant tissue-type plasminogen activator, followed by 42 mg over 90 minutes.
[b] Thrombolytic reperfusion was assessed by standard invasive and non-invasive methods. In the invasive method, angiographic findings were assessed according to the thrombolysis in myocardial infarction (TIMI) criteria. TIMI grades II or III were defined as successful reperfusion. The non-invasive methods included (1) rapid ST-segment normalization defined as a fractional change ≥50% at 2 hours; (2) rapid relief of chest pain at 2—3 hours after the start of thrombolytic treatment; (3) occurrence of reperfusion arrhythmia; (4) peak time of creatine kinase-MB/creatine kinase advanced to 14—16 hours after the onset of acute myocardial infarction (AMI). Reperfusion was considered to have been achieved for patients for whom two items of (1)—(4) applied, unless the two items were (2) and (3). If the start of thrombolytic treatment was within 6—12 hours after AMI onset, (4) did not apply.
[c] Intervention in the culprit vessel within 12 hours after the onset of chest pain or other symptoms, without previous (full or concomitant) thrombolytic or other clot-dissolving therapy.
[d] Intervention in the culprit vessel within 3—24 hours after thrombolysis therapy.

MACE was significantly higher in the low than in the high LDL-C group at 1-month follow-up. A few epidemiological studies have shown a relationship between lipid levels and the incidence of MACE, but these included only patients undergoing angiography and PCI [10,20]. In China, not all patients with ASTEMI are able to have emergency PCI treatment; therefore, our study included all patients undergoing various treatments that reflect the situation in the real world. The treatment methods in hospitals did not significantly differ between the groups.

Some studies have reported that an acute decrease in LDL-C (usually accompanied by an increase in TG), which happens over a few days, is associated with a larger MI [13,14,16]. However, in the present study, TG was significantly lower in the low LDL-C group during the first 24 hours, and the size of the MI, according to peak troponin and left-ventricle ejection fraction, did not significantly differ between the groups. There were no differences between the groups with regard to Killip classification, shock, ventricular arrhythmia, and location of the lesion; therefore, these confounding factors should not have a bearing on the difference in outcome between the groups. The study did reveal an association between LDL-C on admission and 1-month outcome in patients with ASTEMI.

The incidence of MACE at 1-month follow-up was significantly higher in the low than in the high LDL-C group. Al-Mallah et al. observed that low LDL-C on admission was associated with a decreased 3-year survival in patients with NSTEMI, but the authors did not provide information on the drugs used during follow-up [10]. The medications used in our study at follow-up were fully recorded. Data analysis revealed that the greatest difference between the groups during the follow-up period was the use of statins. Many reports have suggested that LDL-C-lowering by statins can reduce the incidence of MACE [2—6]. In addition, the

Table 3 Medications used during treatment.

Medication	Low-density lipoprotein cholesterol		*p*
	>2.94 mmol/L	≤2.94 mmol/L	
In hospital			
Aspirin	492/501 (98.2)	558/575 (97.0)	0.22
Clopidogrel	438/501 (87.4)	506/575 (88)	0.77
Statins	485/501 (96.8)	555/575 (96.5)	0.8
ACEI/ARB	372/501 (74.3)	413/575 (71.8)	0.37
Beta blockers	384/501 (76.6)	401/575 (69.7)	0.01
Glycoprotein IIb/IIIa inhibitors	46/501 (9.2)	74/575 (12.9)	0.06
Low-molecular-weight heparin	478/501 (95.4)	545/575 (94.8)	0.64
At 1-mo follow-up			
Aspirin	438/494 (88.7)	496/566 (87.6)	0.61
Clopidogrel	328/494 (66.4)	364/566 (64.3)	0.48
Statins	429/494 (86.8)	453/566 (80.0)	0.003
ACEI/ARB	271/494 (54.9)	306/566 (45.9)	0.80
Beta blockers	303/494 (61.3)	315/566 (55.7)	0.06
At 6-mo follow-up			
Aspirin	399/489 (81.6)	432/558 (77.4)	0.10
Clopidogrel	250/489 (51.1)	280/558 (50.2)	0.76
Statins	350/489 (71.6)	348/558 (62.4)	0.002
ACEI/ARB	220/489 (45.0)	252/558 (45.2)	0.96
Beta blockers	254/489 (51.9)	271/558 (48.6)	0.28
At 1-y follow-up			
Aspirin	380/484 (78.5)	425/552 (77.0)	0.56
Clopidogrel	210/484 (43.4)	246/552 (44.6)	0.70
Statins	328/484 (67.8)	338/552 (61.2)	0.03
ACEI/ARB	203/484 (41.9)	233/552 (42.2)	0.93
Beta blockers (%)	241/484 (49.8)	263/552 (47.6)	0.49

ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker.

ASTEROID [21], ESTABLISH [22], and JANPAN-ACS [23] studies showed regression of atherosclerotic plaques after a reduction in LDL-C with statins. The regression of atherosclerosis was associated with a marked reduction in the incidence of MACE [24]. In those studies, statins might have provided the maximal benefit of lowering lipid levels with long-term treatment. In the acute phase of ASTEMI, the lipid level may not be the most important factor influencing this process, and inflammation may be dominant. Many recent studies have shown that short-term treatment with statins has beneficial pleiotropic cardiovascular effects that are independent of their lipid-lowering effects [25,26]. These effects include antiplatelet and anti-inflammatory activities [27], protection of the endothelium [28], dilation of coronary microvessels [29], reduction of thrombogenesis [30], and significant lowering of intercellular adhesion molecule-1 and E-selectin levels [31]. In the present study, the rate of statin use was significantly higher in the high LDL-C group. Although LDL-C levels during follow-up were not available, the lower incidence of MACE at 1-month follow-up in the high LDL-C group may be related to the pleiotropic cardiovascular effects of statins, independent of their lipid-lowering effects. At 6-month and 1-year follow-up, statin use was still significantly higher in the high LDL-C group, but the incidence of MACE was not significantly different between the groups. After the acute phase of ASTEMI, plaques become more stable and the major function of statins may be to lower lipid levels. LDL-C-lowering by statins can reduce the incidence of MACE; thus, a reduction in LDL-C is likely to reduce MACE. This could be a rational explanation for the lack of significant difference in MACE incidence at 6-month and 1-year follow-up between the groups.

The prevalence of risk factors (high BMI, diabetes) was significantly higher in the high than in the low LDL-C group. A Cox proportional hazard model analysis revealed that the incidence of MACE after ASTEMI was not associated with age, gender, BMI, or diabetes, but was significantly correlated with hypertension, current smoking, HDL-C, beta blocker use in hospital, and statin use. Higher HDL-C levels, greater beta-blocker use in hospital, and statin use may have reduced the incidence of MACE in the high LDL-C group. Matsumoto et al. found that the LDL-C/HDL-C ratio had an impact on outcome in patients undergoing PCI, and that levels of HDL-C and LDL-C appeared to be equally important in secondary prevention [20].

There are some limitations in the present study. Informed consent was required for 1-year follow-up on MACE and medications; therefore, LDL-C levels during follow-up and statin doses were not available for both groups. Data on the inflammation marker high-sensitivity C-reactive protein (Hs-CRP) were missing; since many hospital laboratories cannot measure Hs-CRP, it was not analyzed. In addition, since treatment methods differed among

Table 4 MACE incidence in hospital and during 1-year follow-up.

	Low-density lipoprotein cholesterol		*p*
	>2.94 mmol/L	≤2.94 mmol/L	
In hospital			
Cardiovascular death	29/501 (5.8)	40/575 (7.0)	0.44
Nonfatal myocardial infarction	1/501 (0.2)	3/575 (0.5)	0.63
Revascularization	2/501 (0.4)	2/575 (0.4)	0.99
Stroke	1/501 (0.2)	1/575 (0.2)	0.99
MACE	33/501 (6.6)	46/575 (8.0)	0.38
At 1-mo follow-up			
Cardiovascular death	34/494 (6.9)	51/566 (9.0)	0.20
Nonfatal myocardial infarction	2/494 (0.4)	7/566 (1.2)	0.19
Revascularization	3/494 (0.6)	8/566 (1.4)	0.20
Stroke	1/494 (0.2)	2/566 (0.4)	1.00
MACE	40/494 (8.1)	68/566 (12.0)	0.04
At 6-mo follow-up			
Cardiovascular death	40/489 (8.2)	57/558 (10.2)	0.26
Nonfatal myocardial infarction	3/489 (0.6)	9/558 (1.6)	0.13
Revascularization	24/489 (4.9)	22/558 (3.9)	0.45
Stroke	3/489 (0.6)	5/558 (0.9)	0.73
MACE	70/489 (14.3)	93/558 (16.7)	0.30
At 1-y follow-up			
Cardiovascular death	56/484 (11.6)	72/552 (13.0)	0.47
Nonfatal myocardial infarction	8/484 (1.7)	16/552 (2.9)	0.18
Revascularization	55/484 (11.4)	62/552 (11.2)	0.95
Stroke	5/484 (1.0)	11/552 (2.0)	0.21
MACE	124/484 (25.6)	161/552 (29.2)	0.20

Data are presented as *n* (%).
MACE = major adverse cardiovascular event (cardiovascular death, non-fatal myocardial infarction, revascularization, and stroke).

patients, treatment bias might be present. This could affect the incidence of MACE. Furthermore, the sample in our study was small, so larger-scale trials are needed to confirm the results.

In spite of this, the finding that low LDL-C on admission was associated with poorer short-term outcome in ASTEMI is very salient. Patients with low LDL-C may also need aggressive risk modification with statins. The result suggests that it is equally important that clinicians take into consideration low LDL-C as well as high LDL-C in patients admitted with ASTEMI.

In summary, the LDL-C level on hospitalization in patients with ASTEMI had no significant effect on the 6-month and 1-year incidence of MACE. However, at 1-month

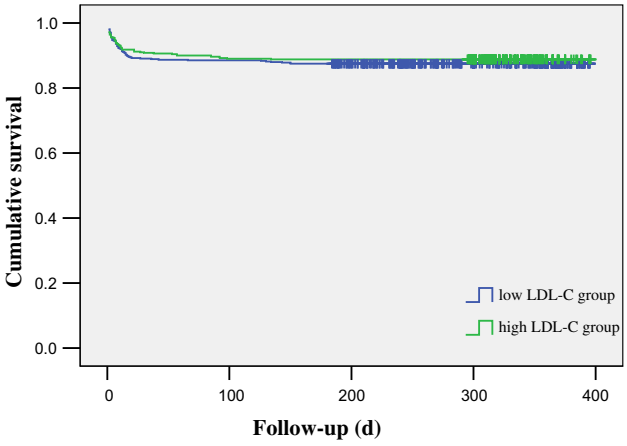

Figure 1. Survival curves for the two groups.

Table 5 Univariate analysis for prediction of 1-year major adverse cardiovascular event.

	Hazard ratio	95% confidence interval	*p*
Age	1.068	1.051–1.086	<0.001
Gender	1.758	1.231–2.511	0.002
Body mass index	1.033	0.984–1.084	0.196
Diabetes	1.578	1.062–2.344	0.024
Hypertension	1.449	1.023–2.05	0.037
Current smoker	2.858	1.899–4.302	<0.001
Low-density lipoprotein cholesterol	0.905	0.743–1.103	0.322
High-density lipoprotein cholesterol	0.376	0.219–0.647	<0.001
Total cholesterol	0.967	0.829–1.129	0.673
Triglyceride	0.868	0.74–1.017	0.08
Beta blocker use in hospital	0.564	0.395–0.806	0.002
Statin use during follow-up	0.562	0.397–0.793	0.001

Table 6 Multiple Cox proportional hazard analysis.

	Hazard ratio	95% confidence interval	*p*
Age	0.907	0.601—1.369	0.642
Gender	1.014	0.998—1.030	0.084
Diabetes	1.458	0.836—2.541	0.184
Hypertension	1.517	1.061—2.170	0.022
Current smoker	2.440	1.599—3.725	<0.001
High-density lipoprotein cholesterol	0.306	0.173—0.541	<0.001
Beta blocker use in hospitals	0.66	0.457—0.955	0.027
Statin use	0.602	0.423—0.856	0.005

follow-up, the incidence of MACE in the low LDL-C group was markedly higher than in the high LDL-C group. The analysis indicates that the LDL-C level on admission in patients with ASTEMI is not an independent predictor of MACE. It would be relevant to further investigate HDL-C level on admission, beta-blocker use in hospitals, and statin use during follow-up in relation to MACE. These findings suggest that in the early stage of ASTEMI, all patients may benefit from statin treatment, regardless of their LDL-C level.

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