The Effect of Intensified Low Density Lipoprotein Cholesterol Reduction on Recurrent Myocardial Infarction and Cardiovascular Mortality

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Background: Lipid-lowering therapy plays an important role in preventing the recurrence of cardiovascular events in patients after acute myocardial infarction (AMI). This study aimed to assess the effect of intensified low density lipoprotein cholesterol (LDL-C) reduction on recurrent myocardial infarction and cardiovascular mortality in patients after AMI.

Method: The 562 enrolled AMI patients (84.2% male) were divided into two groups according to 3-month LDL-C decrease percentage equal to or more than 40% (n = 165) and less than 40% (n = 397). To evaluate the long-term efficacy of LDL-C reduction, the 5-year outcomes were collected, including time to the first occurrence of myocardial infarction and time to cardiovascular death.

Results: The baseline characteristics and complication rates were not different between the two study groups. The patients with 3-month LDL-C decrease $\geqq$ 40% had higher baseline LDL-C and lower 3-month, 1-year, 2-year, 3-year, 4-year and 5-year LDL-C than the patients with 3-month LDL-C decrease < 40%. In Kaplan-Meier analyses, those patients with 3-month LDL-C decrease $\geqq$ 40% had a higher rate of freedom from myocardial infarction (p = 0.006) and survival rate (p = 0.02) at 5-year follow-up. The 3-month LDL-C < 40% parameter was significantly related to cardiovascular death (HR: 9.62, 95% CI 1.18-78.62, p < 0.04).

Conclusions: After acute myocardial infarction, 3-month LDL-C decrease < 40% was identified to be a significant risk factor for predicting 5-year cardiovascular death. The patients with 3-month LDL-C decrease $\geqq$ 40% had a higher rate of freedom from myocardial infarction and lower cardiovascular mortality, even though these patients had higher baseline LDL-C value.

Key Words: Acute myocardial infarction • Cardiovascular death • Low-density lipoprotein cholesterol • Mortality • Statin

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INTRODUCTION

Acute myocardial infarction (AMI) is one of the major causes of morbidity and mortality in Taiwan and worldwide.[1-3] Furthermore, assessing the risk factor of clinical outcomes after AMI remains an important research topic.[4-11] Elevated serum levels of low-density lipoprotein cholesterol (LDL-C), a well-known risk factor for development and progression of coronary artery disease, contributes destabilization of atherosclerotic vascular disease and further significantly increases the risk

of AMI.[12-14] Lipid-lowering therapy plays a critical role in preventing the recurrence of cardiovascular events in primary or secondary prevention. Previous studies have demonstrated that lowering LDL-C levels with statins reduces the risk of recurrent cardiovascular events and improve survival in patients with AMI.[15-21]

National Cholesterol Education Program (NCEP) guidelines recommend that intensity of therapy should be sufficient to achieve at least a 30% to 40% reduction in LDL-C levels in high risk individuals.[22] Earlier studies in patients with stable angina showed that a significant positive correlation was identified between the percentage reduction in LDL-C during the first 3 months after coronary revascularization and the time until recurrence of cardiovascular events.[23] However, no prior publication compared the outcomes of reducing LDL-C $\geqq$ 40% and less than 40% in patients with AMI. Therefore, the aim of this study was to assess the effect of intensified LDL-C reduction on recurrent myocardial infarction and cardiovascular mortality in patients after AMI.

MATERIALS AND METHODS

Patients

A total of 661 consecutive patients diagnosed with acute myocardial infarction were enrolled in this study from Jan. 2005 to Dec. 2007. Diagnosis of AMI was made on the basis of typical angina lasting more than 30 minutes, new electrocardiographic change that included ST-segment elevation 0.2 mV in 2 contiguous electrocardiographic leads or other ST/T changes, biochemical evidence of peak creatine kinase more than 2 times the upper limit of normal, and wall motion abnormalities by echocardiography.[4,10,11] Criteria for exclusion were patients with LDL-C less than 70 mg/dl, loss follow-up and a diagnosis of chronic hepatitis or cirrhosis. Therefore, a total of 562 patients were included in the study. The Human Research Committee of our hospital approved the study protocol.

Data collection

The basic data was collected, including hypertension, diabetes mellitus, smoking history, uremia, chronic obstructive pulmonary disease, gout, family history of coronary artery disease and Killip class. The previous stroke or myocardial infarction events were also collected.

The vital signs, such as heart rate, systolic and diastolic blood pressure, were measured at the emergency department and discharge. The peripheral blood samples were drawn at the emergency room or after admission, including peak creatinine kinase (CK), CK-MB isoform, Troponin I, creatinine, total bilirubin and alanine aminotransferase. Serum total cholesterol, LDL-C, high density lipoprotein-cholesterol (HDL-C) and triglycerides were checked at fasting status. Three months after discharge, all patients received follow-up blood sampling, including creatinine, total bilirubin, alanine aminotransferase, total cholesterol, LDL-C, HDL-C and triglyceride.

The following complications were also recorded, including: cardiogenic shock, complete atrioventricular block, use of intra-aortic balloon pump, use of temporal pacemaker, ventricular septal defect, mitral regurgitation, ventricular tachycardia, ventricular fibrillation, atrial fibrillation, use of mechanical ventilator and post cardiopulmonary resuscitation.

Medication

All patients with AMI received aspirin 300 mg and clopidogrel 300 mg at the emergency room. Then, heparin, glycoprotein IIb/IIIa inhibitor and beta-blocker were prescribed if no contraindication. After admission, aspirin, clopidogrel, beta-blocker, angiotensin converting enzyme inhibitors (ACEI) or angiotensin receptor blocker (ARB) were prescribed if patients had no contraindication. The choice of statin use was dependent on the decision of attending physicians. Additionally. the medications administered at the emergency room and discharge were recorded.

Clinical evaluation and outcomes

To evaluate the long-term efficacy of LDL-C reduction, the 5-year outcomes were collected, including time to the first occurrence of myocardial infarction and time to cardiovascular death.

Statistics

The data were analyzed statistically at $p < 0.05$ using SPSS version 21.0 (SPSS Inc, Chicago, IL, USA). The continuous data was shown as mean and standard devia-

tion and the categorical data was shown as percentages. An independent t test was used for continuous variables and a Chi-square test was used to analysis categorical variables. Kaplan-Meier analyses were used to assess long term survival and freedom from myocardial infarction. The relations of LDL-C variables with cardiovascular mortality were examined using multivariable Cox proportional hazards regression.

RESULTS

Baseline characteristics (Table 1)

The patients (n = 562, 84.2% male) were divided into two groups according to whether the 3-month LDL-C decrease percentage was equal to or more than 40% (n = 165), or less than 40% (n = 397). The patients with 3-month LDL-C decrease ≧ 40% (group 1, n = 165) had similar age with those patients with 3-month LDL decrease < 40% (group 2, n = 397) (60.3 ± 13.3 vs. 61.3 ± 13.6, p = 0.43). The patients in group 1 were heavier than group 2 patients, although not significantly (81.0 ± 13.0 vs. 68.4 ± 13.8, p = 0.05). There were no differences between the two groups in body height, percentage of smoker, hypertension, diabetes, uremia, chronic obstructive pulmonary disease, previous infarction, previous stoke, family history of coronary artery disease and gout. The severity of AMI, shown as Killip Class, was not different between the 2 groups (1.8 ± 1.0 vs. 1.8 ± 1.0, p = 0.98). There was also no difference between the two groups in both length of hospital stay and vital signs at the emergency department or discharge.

Table 1. Baseline characteristics of two study groups

	3 month LDL-C decrease ≧ 40%* (Group 1) N = 165	3 month LDL-C decrease < 40%* (Group 2) N = 397	p value
Male	140 (84.8%)	333 (83.9%)	0.90
Age	60.3 ± 13.3	61.3 ± 13.6	0.43
Body weight (kg)	81.0 ± 13.0	68.4 ± 13.8	0.05
Body height (cm)	164.8 ± 8.0	163.8 ± 16.2	0.58
Current smoker	80 (48.5%)	179 (45.1%)	0.52
Ex-smoker	23 (13.9%)	68 (17.1%)	0.38
Underlying diseases			
Hypertension	92 (58.2%)	230 (57.9%)	0.64
Diabetes	59 (37.6%)	150 (37.8%)	0.63
Uremia	2 (1.2%)	6 (1.5%)	1.00
COPD	6 (3.6%)	12 (3.0%)	0.79
Previous infarction	7 (4.2%)	28 (7.1%)	0.25
Previous stroke	10 (14.9%)	22 (14.4%)	1.00
Family history of CAD	21 (12.7%)	40 (10.1%)	0.37
Gout	13 (7.9%)	27 (6.8%)	0.72
Menopause	6 (3.6%)	21 (5.3%)	0.52
Killip class	1.8 ± 1.0	1.8 ± 1.0	0.98
Hospital stay (days)	7.9 ± 7.0	9.7 ± 9.4	0.26
Vital sign at emergent department			
Heart rate (beats/minute)	81.2 ± 24.9	81.1 ± 25.6	0.96
SBP (mmHg)	139.7 ± 40.4	138.5 ± 53.4	0.80
DBP (mmHg)	80.5 ± 29.3	75.2 ± 30.8	0.07
Vital sign at discharge			
Heart rate (beats/minute)	67.4 ± 24.3	68.6 ± 25.0	0.60
SBP (mmHg)	117.7 ± 22.6	117.7 ± 20.7	0.99
DBP (mmHg)	64.4 ± 26.2	61.7 ± 29.7	0.31

CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; DBP, diastolic blood pressure; LDL-C, low density lipoprotein-cholesterol; SBP: systolic blood pressure.

Complication rates

Whether the patients with 3-month LDL-C decrease ≧ 40% or < 40%, the 2 groups had similar complication rates, including cardiogenic shock, complete atrioventricular block, ventricular septal defect, mitral regurgitation, ventricular tachycardia, ventricular fibrillation, atrial fibrillation and post cardiopulmonary resuscitation. There was also no difference in use of mechanical ventilator, intra-aortic balloon pump and temporal pacemaker between the 2 groups.

Laboratory data (Table 2)

There were no significant differences in peak creatinine kinase, peak CK-MB isoform, peak Troponin I, creatinine, total bilirubin and alanine aminotransferase between the 2 groups. In the lipid profile, the patients with 3-month LDL-C decrease ≧ 40% had higher baseline total cholesterol (204.2 ± 40.7 vs. 177.7 ± 44.1, p < 0.001), LDL-C (129.6 ± 29.3 vs. 109.8 ± 28.9, p < 0.001) and HDL-C (37.7 ± 9.7 vs. 36 ± 9.3, p = 0.046) compared to the patients with 3-month LDL-C decrease < 40%. However, there was no difference in triglyceride levels between the 2 groups (121.6 ± 129.2 vs. 121.2 ± 87.6, p = 0.97).

Medication at emergent department and discharge (Table 3)

Regarding medications administered at the emergency department, there was no significant difference in the percentage of aspirin, beta-blocker, clopidogrel, glycoprotein IIb/IIIa inhibitor and heparin use between group 1 and group 2 AMI patients.

The patients with 3-month LDL-C decrease ≧ 40% had a similar percentage of drug use at the time of discharge with those patients with 3-month LDL-C decrease < 40%, including: aspirin, beta-blocker, clopidogrel and ACEI or ARB use.

Table 2. Complication rates and laboratory data of two study groups

	3 month LDL-C decrease ≧ 40%* (Group 1) N = 165	3 month LDL-C decrease < 40%* (Group 2) N = 397	p value
Complication rates			
Cardiogenic shock	16 (9.7%)	40 (10.1%)	1.00
Post IABP	6 (3.6%)	21 (5.3%)	0.52
CAVB	3 (1.8%)	10 (2.5%)	0.77
Post temporal pacemaker	2 (1.2%)	12 (3.0%)	0.37
Ventricular septal defect			
Mitral regurgitation	3 (1.8%)	10 (2.5%)	0.77
Ventricular tachycardia	5 (3.0%)	11 (2.8%)	1.00
Ventricular fibrillation	5 (3.0%)	6 (1.5%)	0.31
Atrial fibrillation	3 (1.8%)	9 (2.3%)	1.00
Post mechanical ventilator	12 (7.3%)	30 (7.6%)	1.00
Post cardiopulmonary resuscitation	2 (1.2%)	5 (1.3%)	1.00
Laboratory data			
Peak CK (U/L)	2605.7 ± 2558.9	2400.0 ± 2568.7	0.40
Peak CK-MB(U/L)	223.0 ± 204.3	200.4 ± 216.7	0.27
Peak Troponin I (mg/dL)	112.3 ± 133.7	117.4 ± 155.1	0.70
Creatinine (mg/dL)	1.2 ± 1.1	1.3 ± 1.2	0.37
Total bilirubin (mg/dL)	0.8 ± 0.3	0.8 ± 0.4	0.81
GPT (U/L)	43.5 ± 31.7	49.5 ± 116.5	0.53
Total cholesterol (mg/dL)	204.2 ± 40.7	177.7 ± 44.1	< 0.001
HDL-C (mg/dL)	37.7 ± 9.7	36 ± 9.3	0.046
LDL-C (mg/dL)	129.6 ± 29.3	109.8 ± 28.9	< 0.001
Triglyceride (mg/dL)	121.6 ± 129.2	121.2 ± 87.6	0.97

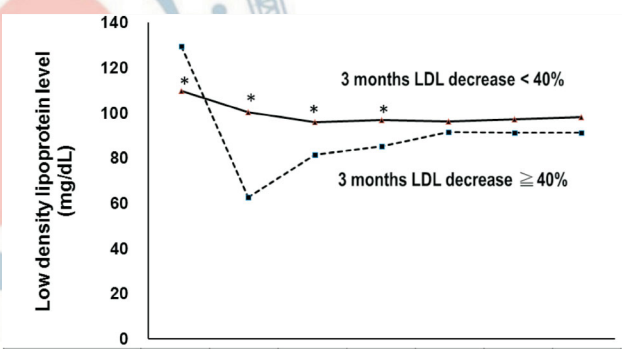
CAVB, complete atrioventricular block; CK, creatinine kinase; GPT, alanine aminotransferase; HDL-C, high density lipoprotein-cholesterol; IABP, intra aortic balloon pump; LDL-C, low density lipoprotein-cholesterol.

Table 3. Medications of two study groups

	3 months LDL-C decrease ≧ 40%* (Group 1) N = 165	3 months LDL-C decrease < 40%* (Group 2) N = 397	p value
Medication at emergency department			
Aspirin	162 (100.0%)	382 (98.7%)	0.33
Beta-blocker	89 (75.4%)	195 (68.9%)	0.11
Clopidogrel	160 (97.0%)	378 (95.2%)	0.49
Glycoprotein IIb/IIIa inhibitor	19 (11.5%)	42 (10.6%)	0.77
Heparin	161 (99.4%)	378 (98.2%)	0.45
Medications at discharge			
Aspirin	152 (93.8%)	348 (89.2%)	0.11
Beta-blocker	107 (64.8%)	226 (57.9%)	0.09
Clopidogerl	160 (98.2%)	369 (98.1%)	1.00
ACEI or ARB	154 (93.3%)	364 (91.7%)	0.51
Statin use at discharge			
No statin	13 (7.8%)	195 (49.1%)	< 0.001
Rosuvastatin	115 (69.7%)	134 (33.8%)	< 0.001
Atorvastatin	29 (17.6%)	57 (14.4%)	0.37
Simvastatin + Ezitimide	6 (3.6%)	6 (1.5%)	0.12
Other statins	2 (1.2%)	5 (1.3%)	1.00

ACEI, angiotensin converting enzyme inhibitors; ARB, angiotensin receptor blocker; LDL-C, low density lipoprotein-cholesterol.

Regarding statin use at the time of discharge, the patients with 3-month LDL-C decrease ≧ 40% had a higher percentage use of statin than those patients with 3-month LDL-C decrease < 40% (91.5% vs. 50.9%, p < 0.001), especially use of rosuvastatin (69.7% vs. 33.8%, p < 0.001). There was no difference in use of atorvastatin, simvastatin with ezitimide and other statins between these 2 groups.



	Baseline	3 month	1 year	2 year	3 year	4 year	5 year
3 months LDL decrease ≧40%	129.6±29.3	62.6±15.4	81.5±30.5	85.4±29.7	91.8±26.6	91.4±20.2	91.3±20.7
3 months LDL decrease < 40%	109.8±28.9	100.4±28.9	96.0±33.9	96.9±30.5	96.4±30.5	97.2±23.9	98.3±21.9

Figure 1. *Fiver year follow-up low density lipoprotein-cholesterol (LDL-C) data between two groups. The patients with 3-month LDL-C decrease ≧ 40% had higher baseline LDL-C and lower 3-month, 1-year, 2-year, 3-year, 4-year and 5-year LDL-C than the patients with 3-month LDL-C decrease < 40%. * Indicates statistical significance (p < 0.05).*

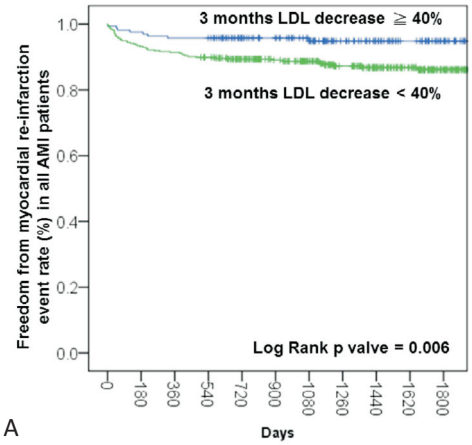
Follow-up laboratory data (Figure 1)

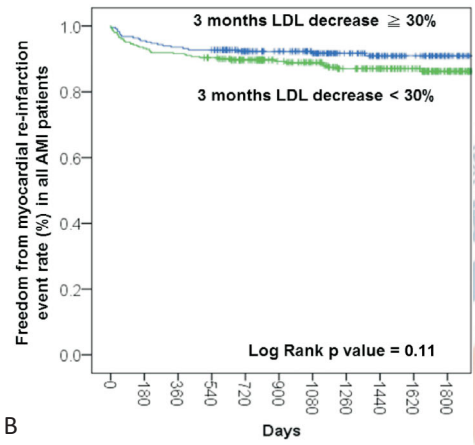
In Figure 1, the patients with 3-month LDL-C decrease ≧ 40% had a higher baseline LDL-C and a lower 3-month, 1-year, 2-year, 3-year, 4-year and 5-year LDL-C than the patients with 3-month LDL-C decrease < 40% (Figure 1).

Long-term outcomes (Figures 2, 3 and 4)

In Kaplan-Meier analyses for freedom from myocardial infarction (Figure 2), the patients with 3-month LDL-C decrease ≧ 40% had a higher rate of freedom from myocardial infarction at 5-year follow-up than the patients with 3-month LDL-C decrease < 40% (log rank p value = 0.006). However, there were no significantly different rates of freedom from myocardial infarction at 5-year follow-up between patients with 3-month LDL-C decrease ≧ 30% and < 30% (log rank p value = 0.11) or between patients with 3-month LDL-C decrease ≧ 20% and < 20% (log rank p value = 0.32) (Figure 2).

In Kaplan-Meier analyses for cardiovascular mortal-





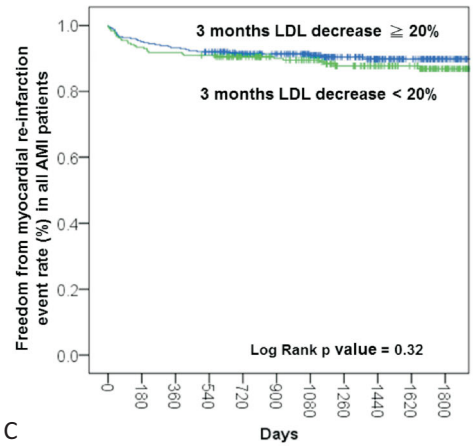
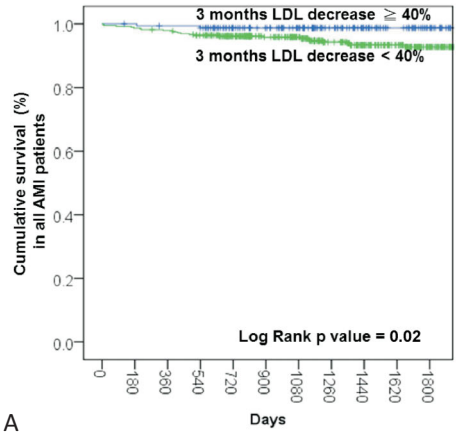
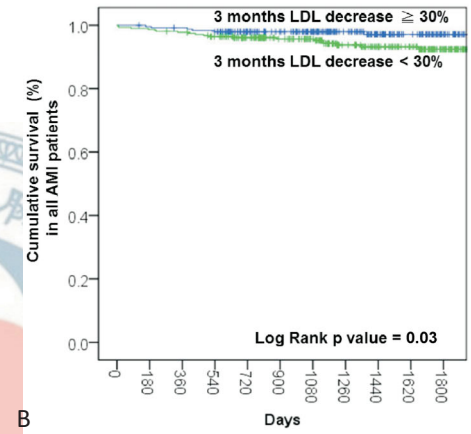


Figure 2. *Kaplan-Meier analyses for freedom from myocardial infarction in all AMI patients. Panel A showed the patients with 3-month low density lipoprotein-cholesterol (LDL-C) decrease ≧ 40% had higher rate of freedom from myocardial infarction at 5-year follow-up than the patients with 3-month LDL-C decrease < 40% (long Rank p value = 0.006). Panel B and C showed there were no significantly different rates freedom from myocardial infarction at 5-year follow-up between patients with 3-month LDL-C decrease ≧ 30% and < 30% (long Rank p value = 0.109) or between patients with 3-month LDL-C decrease ≧ 20% and < 20% (long Rank p value = 0.321).*





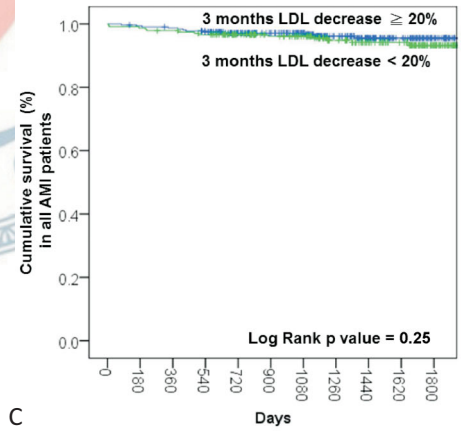


Figure 3. *Kaplan-Meier analyses for cardiovascular mortality in all AMI patients. Panel A showed the patients with 3-month low density lipoprotein-cholesterol (LDL-C) decrease ≧ 40% had higher 5-year survival rate than the patients with 3-month LDL-C decrease < 40% (log rank p value = 0.016). Panel B showed the patients with 3-month LDL-C decrease ≧ 30% had higher 5-year survival rate than the patients with 3-month LDL-C decrease < 30% (long Rank p value = 0.027). Panel C revealed there were no significantly different 5-year survival rates between patients with 3-month LDL-C decrease ≧ 20% and < 20% (log Rank p value = 0.245).*

ity (Figure 3), the patients with 3-month LDL-C decrease ≧ 40% had a higher 5-year survival rate than the patients with 3-month LDL-C decrease < 40% (log rank p value = 0.02). Besides, the patients with 3-month LDL-C decrease ≧ 30% had a higher 5-year survival rate than the patients with 3-month LDL-C decrease < 30% (log rank p value = 0.03). However, there were no significantly different 5-year survival rates between patients with 3-month LDL-C decrease ≧ 20% and < 20% (log rank p value = 0.25) (Figure 3).

In further subgroup analysis of patients received statin therapy, Kaplan-Meier analyses showed the patients with 3-month low density lipoprotein-cholesterol (LDL-C) decrease ≧ 40% had higher 5-year survival rate than the patients with 3-month LDL-C decrease < 40% (log rank p value = 0.01) (Figure 4).

Table 4 showed that several LDL-C parameters had an insignificant risk of cardiovascular death, including baseline LDL-Cl > 100 mg/dl, 3rd month LDL-C > 70 mg/dl, 3rd month LDL-C > 100 mg/dl, 3rd month LDL-C decreased < 20%, and 3-month LDL-C decreased < 30%. Only 3-month LDL-C < 40% parameter was significantly related to cardiovascular death (HR: 9.62, 95% CI 1.18-78.62, p < 0.04).

DISCUSSION

The strengths of this study include the 5-year long-term follow-up and the detailed information on clinical risk factors. To the best of our knowledge, this is the first analysis of the role of 3-month LDL-C decrease ≧ 40% to assess long-term outcomes in patients after AMI. This 5-year follow-up study demonstrated that AMI patients with 3-month LDL-C decrease ≧ 40% had lower 3-month, 1-year, 2-year, 3-year, 4-year and 5-year LDL-C values than the patients with 3-month LDL-C decrease < 40%. Only 3-month LDL-C decrease < 40% was found to be significantly related to cardiovascular death. Furthermore, those AMI patients with 3-month LDL-C decrease ≧ 40% had a higher rate of freedom from myocardial infarction and lower cardiovascular mortality, even though these patients had higher baseline LDL-C values.

Statins were shown to play a critical role in secondary prevention of cardiovascular events.[19,24-26] However, how long the LDL-C should be followed and how soon the LDL-C level should be reduced in daily practice of secondary prevention remains unknown. A previous study showed the 3-month percentage of LDL-C reduction after coronary revascularization had a substantial impact in preventing or delaying cardiovascular event recurrence.[23] Even though LDL-C exceeded the guideline-recommended goal, cardiovascular event recurrence can be delayed with sufficient reduction of LDL-C

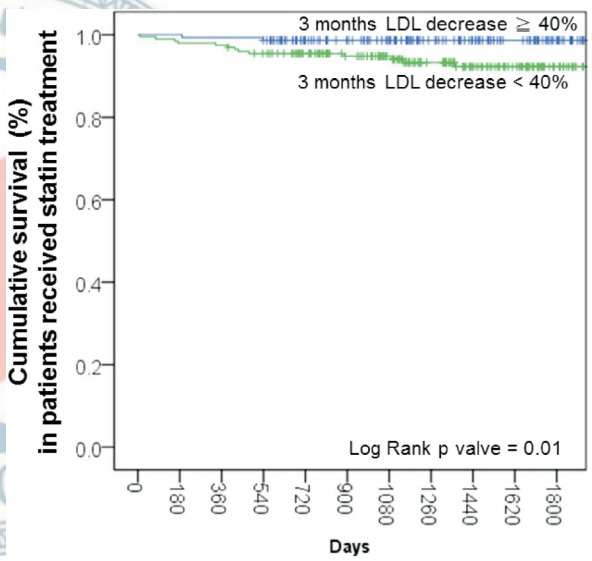


Figure 4. *Kaplan-Meier analyses for cardiovascular mortality in patients received statin therapy. The figure showed the patients with 3-month low density lipoprotein-cholesterol (LDL-C) decrease ≧ 40% had higher 5-year survival rate than the patients with 3-month LDL-C decrease < 40% (log rank p value = 0.01).*

Table 4. Hazard ratio for cardiovascular mortality

Variable	Hazard ratio	p value
Baseline low density lipoprotein-cholesterol > 100 mg/dl	0.76 (0.27-2.15)	0.60
3-month low density lipoprotein-cholesterol > 100 mg/dl	1.24 (0.52-2.18)	0.52
3-month low density lipoprotein-cholesterol > 70 mg/dl	1.38 (0.55-4.74)	0.22
3-month low density lipoprotein-cholesterol decrease < 20%	0.49 (0.14-1.70)	0.58
3-month low density lipoprotein-cholesterol decrease < 30%	1.54 (0.50-7.09)	0.18
3-month low density lipoprotein-cholesterol decrease < 40%	9.62 (1.18-78.62)	0.04

during the early period after coronary revascularization.[23] However, no previous publication mentioned the association between 3-month improvement of LDL after AMI and long-term outcome. The results of the present study suggest that LDL-C decrease < 40% at 3-months could be a useful predictor for cardiovascular death.

Statin use has been proven to reduce the risk of recurrent cardiovascular events and improve survival in patients with AMI.[15-21] However, in this study, 49.1% of patients in the group of LDL-C reduction less than 40% were not given statins at discharge. However, only 7.8% of patients with 3-month LDL-C decrease ≧ 40% did not receive statins. The beneficial effect, as shown above, may be related to the pleiotropic effects of statin or achieved lower LDL-C level in ≧ 40% group. Therefore, subgroup analysis of patients received statin therapy was done. After exclusion of the patient without statin use at discharge, Kaplan-Meier analyses still showed that patients with 3-month low density lipoprotein-cholesterol (LDL-C) decrease ≧ 40% had higher 5-year survival rate than patients with 3-month LDL-C decrease < 40% (log rank p value = 0.01) (Figure 4). This result further supported lower LDL-C level ≧ 40% play an important role in long-term outcomes in patients after AMI.

In this study, only the 3-month LDL-C < 40% parameter was shown to be significantly related to cardiovascular death (HR: 9.62, 95% CI 1.18-78.62, p < 0.04), but not other LDL-C parameters, including baseline LDL-C > 100 mg/dl, 3rd month LDL-C > 70 mg/dl, 3rd month LDL-C > 100 mg/dl, 3rd month LDL-C decrease < 20% and 3-month LDL-C decrease < 30%. These results also confirmed that patients with 3-month LDL-C decrease ≧ 40% had better 5-year outcomes than the patients with 3-month LDL-C decrease < 40%, even though a higher baseline LDL-C was present in these groups (Figure 1). A recent ESC guideline proposed LDL-C < 70 mg or a reduction >50% in high-risk patients.[27] However, this topic has not been examined in an Asian population. The CREDO-Kyoto Registry Cohort-2 trial showed, in patients post first coronary revascularization, the risk for MACE was significantly higher in the LDL-C ≥ 120 mg/dl group.[28] Whereas, there were insignificant differences of MACE in the LDL-C = 100-119 mg/dl group and the LDL-C < 80 mg/dl group.[28] Sakamoto and Ogawa also mentioned about "Just make it lower" is an alternative strategy of lipid-lowering therapy with statins in Asian patients.[29] Furthermore, the beneficial effects of statin therapy were shown to vanish when LDL-C is below a certain level in AMI patients.[30] These previous researches[28-30] supported our findings in this study.

Study limitations

There were several limitations to our study. First, our sample size was relatively small. Furthermore, this was a single center study. It might be necessary to further confirm the results of this study by another large cohort multi-center study. Whereas, this study was an observational study and the differences among groups might influence the results. However, most of the baseline characteristics, complication rates, baseline laboratory data and medications between these two study groups were shown to be insignificant different (Tables 1, 2 and 3). Furthermore, the adherence of statin and effect of different statins were not investigated in this study.

CONCLUSIONS

After AMI, 3-month LDL-C decrease < 40% was identified to be a significant risk factor to assess 5-year cardiovascular death. The patients with 3-month LDL-C decrease ≧ 40% had a higher rate of freedom from myocardial infarction and lower cardiovascular mortality, even though these patients had higher baseline LDL-C value.

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