Original Article

LDL Cholesterol and 1-Year Mortality Rate after Acute ST-Elevation Myocardial Infarction

Osit Bambat MD[1], Wasan Udayachalerm MD[1],
Wacin Buddhari MD[1],Jakarpun Chaipromprasit MD[1], Vorarit Lertsuwunseri MD[1],
Siriporn Athisakul MD[1], Suphot Srimahachota MD[1]

[1] Cardiac Center and Division of Cardiovascular Medicine, Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

Background: Dyslipidemia is one of the risk factor of coronary artery disease but there have been reports showing paradoxical effect of LDL cholesterol (LDL-C) level which lower LDL-C level at the time of diagnosis of patients with acute coronary syndrome was associated with higher 1-year mortality. However, this relationship has not been established in Thai patients.

Objective: To evaluate LDL-C and 1-year all-cause mortality rate after acute ST-elevation myocardial infarction (STEMI) in Thailand.

Materials and Methods: The present study was a retrospective study of 1,542 consecutive patients with STEMI from 1999 to 2015 whom underwent percutaneous coronary intervention (PCI) either primary PCI or rescue PCI at a tertiary care hospital in Thailand. Patients were classified into 4 groups according to their initial LDL-C level: <70 (group 1), 70-99 (group 2), 100-129 (group 3) and ≥130 mg/dl (group 4). The primary end point was a 1-year all-cause mortality rate. T-test, Chi-square and survival time analysis were used to analyze.

Results: The rates of 1-year all-cause mortality were 23.0%, 21.2%, 12.3% and 9.2% in group 1, group 2, group 3 and group 4, respectively. Comparing with group 4, the hazard ratio (HR) was 2.72 (95 % confidence interval [CI] 1.62-4.57; *P*<0.001) for group 1, HR 2.45 (95% CI 1.64-3.66; *p*<0.001) for group 2 and HR 1.35 (95% CI 0.89-2.06; *p*=0.153) for group 3. In multivariate analysis, the predictors of 1-year all-cause mortality were Killip class IV (HR 2.40, 95% CI 1.78-3.25; *p*<0.001), cardiopulmonary resuscitation (HR 2.25, CI 1.64-3.09; *p*<0.001), left ventricular ejection fraction ≤40% (HR 2.07, 95% CI 1.57-2.72; *p*<0.001), culprit lesion at left main (HR 1.78, 95% CI 1.13-2.81; *p*=0.024), creatinine >1.5 mg/dl (HR 1.77, 95% CI 1.32-2.38; *p*<0.001), but not the LDL-C level (*p*=0.732)

Conclusion: In this single center experience, lower LDL-C level at time of diagnosis of STEMI was associated with higher 1-year all-cause mortality but the effect was not shown after multivariate analysis. This represent LDL-C was not the risk factor to predict 1-year all-cause mortality in STEMI but there were other factors that associated with the outcome. LDL-C was independent risk marker.

Keywords: LDL cholesterol, STEMI, 1-year mortality rate

J Med Assoc Thai 2018; 101 (6): 731-7
Website: http://www.jmatonline.com

Dyslipidemia is the harbinger of plaque formation in atherosclerotic disease. LDL cholesterol (LDL-C) progressively accumulates in intimal layer of coronary artery and turns into lipid core. Plaque becomes bigger and causing vessel stenosis. Acute coronary syndrome (ACS) occurs when plaque ruptures and stimulates clotting and coagulating cascade. If vessel was totally occluded, it would cause acute ST-elevation myocardial infarction (STEMI)[(1)]. The patients with STEMI had high mortality rate. In Thailand, there are 5% of the in-hospital death, the 12% of the 6-month mortality rate and the 14% of the 1-year mortality rate[(2)]. In China, there is 7.1% of the 1-year mortality rate[(3)]. The result of the relationship between LDL-C at time diagnosed of acute STEMI and mortality rate are surprising. Patients with lower LDL-C are associated with higher in-hospital death, 1-month mortality and 1-year mortality than higher LDL-C group[(4)]. However, this paradoxical phenomenon has not been established in Thai patients. The present study aimed to evaluate LDL-C level and 1-year all-cause mortality rate in acute STEMI patients underwent PCI in Thailand.

Correspondence to:

Srimahachota S. Division of Cardiovascular Medicine, Department of Medicine, Faculty of Medicine, Chulalongkorn University, 1873 Rama 4 Road, Pathumwan, Bangkok 10330, Thailand.
Phone: +66-2-256-4000 ext.80121-3, **Fax:** +66-2-256-4000 ext.80121-3,214.
Email: s_srimahachota@yahoo.co.th

How to cite this article: Bambat O, Udayachalerm W, Buddhari W, Chaipromprasit J, Lertsuwunseri V, Athisakul S, et al. LDL cholesterol and 1-year mortality rate after acute ST-elevation myocardial infarction. J Med Assoc Thai 2018;101:731-7.

Materiasl and Methods

Study design

The present study was a single-center, retrospective, descriptive study. The protocol was approved by the local Institutional Review Board.

Study population

The consecutive patients with acute STEMI from 1999 to 2015 were recruited in the study. STEMI was diagnosed by symptom of angina chest pain and electrocardiogram (EKG) showed J-point elevation more than 0.1 millivolt in 2 contiguous leads with or without reciprocal change that was ST-segment depression. Every patient underwent percutaneous coronary intervention (PCI), either primary PCI indication or rescue PCI indication for patients who failed to systemic thrombolysis.

The authors included patients who had admission lipid profile within 24 hours after diagnosed of acute STEMI. If there was not directed LDL-C available, estimated LDL-C would be calculated with Friedewald equation [LDL-C = total cholesterol (TC) – triglyceride (TG)/5 – HDL cholesterol (HDL-C)][5].

Evaluation of outcome

Patients were classified into 4 groups according to their initial LDL-C level; <70 (group 1), 70-99 (group 2), 100-129 (group 3) and ≥130 mg/dl (group 4), which adapted from the previous study[4]. The primary outcome is 1-year all-cause mortality rate. The mortality data were collected by using 13-digit of the Thai national identification number to find the patients' death at 1 year in the death registration database at the Civil Registration Section, Ministry of Interior. Secondary outcomes were finding the risk factors that predicted 1-year all-cause mortality rate.

Statistical analysis

Categorical data were expressed as frequencies and percentages and analyzed using the Chi-square test. Continuous variables were expressed as mean and standard deviations or medians and analyzed using independent t-test. The classified subgroup according to initial LDL-C level using one-way ANOVA was compared mean among groups. Cumulative of 1-year mortality event curves were constructed for the primary outcome with using Kaplan-Meier method. Crude event rates were determined from Kaplan-Meier rates and compared using log-rank test. Risk factors that associated with 1-year all-cause mortality rate were analyzed by using a Cox proportional-hazards regression model. A p-value <0.05 was cut-point of statistically significant. The authors used IBM SPSS Statistics for mac version 22 (Chicago, SPSS Inc.) for analyzed the data.

Results

Characteristics of enrolled patients

From January 1999 through December 2015, the total of 1,542 consecutive STEMI patients was enrolled in this study. There were 378 patients excluded because no admission lipid profile available within 24 hours after diagnosed STEMI.

Classified STEMI patients according to initial LDL cholesterol level

There were 1,164 patients were classified into 4 groups for primary outcome analysis. Group 1: there was more older age, more underlying diseases, fewer smokers, received more medications than other groups. Group 1 had less systolic blood pressure (SBP), less diastolic blood pressure (DBP), more heart rate (HR) and more Killip class IV than others (p<0.001) at time diagnosed STEMI. Group 1 had lower TC and TG, higher creatinine and glomerular filtration rate (GFR) than other groups, (Table 1).

In PCI data, group 2: there was longer door-to-balloon time than other groups; but onset-to-balloon time, number of vessel diseases, left main (LM) disease, culprit vessels, segment of culprit lesion and complete revascularization were not different among all groups. Group 4: there was highest stenting rate 89% (p<0.001), (Table 2).

Outcome

The rates of 1-year all-cause mortality were 23.0%, 21.2%, 12.3 and 9.2% in group 1, group 2, group 3, group 4, respectively. Comparing to group 4, hazard ratio (HR) was 2.72 (95% confidence interval [CI] 1.62-4.57; p<0.001) for group 1, HR 2.45 (95% CI 1.64-3.66; p<0.001) for group 2 and HR 1.35 (95% CI 0.89-2.06; p = 0.153) for group 3, (Figure 1). Group 1, there were more complications than other groups statistically significant such as: cardiopulmonary resuscitation (CPR), cardiogenic shock, need inotropic drugs, need intra-aortic balloon pump (IABP) and heart failure need mechanical ventilator. All groups had similar left ventricular ejection fraction (LVEF), (Table 3).

Table 1. Baseline characteristics of STEMI patients according to initial LDL cholesterol level

Characteristics	Group 1 LDL-C <70 mg/dl (n = 87)	Group 2 LDL-C 70-99 mg/dl (n = 212)	Group 3 LDL-C 100-129 mg/dl (n = 309)	Group 4 LDL-C ≥130 mg/dl (n = 556)	*p*-value
Male – n (%)	58 (67)	154 (73)	242 (78)	438 (79)	0.035
Age – yr	65±14	62±13	60±13	57±12	<0.001
Underlying diseases – n (%)					
Diabetes Mellitus	34 (39)	67 (32)	80 (26)	93 (17)	<0.001
Hypertension	51 (59)	121 (57)	149 (48)	219 (39)	<0.001
Dyslipidemia	37 (43)	90 (43)	111 (36)	184 (33)	0.060
Coronary artery disease	10 (12)	33 (16)	28 (9)	30 (5)	0.001
Smoking – n (%)	36 (41)	86 (41)	131 (42)	282 (51)	0.020
Previous Medications – n/total n (%)					
Anti-platelet	15/65 (23)	43/171 (25)	32/244 (13)	29/448 (6)	<0.001
Statin	21/65 (32)	60/171 (35)	40/244 (17)	31/448 (7)	<0.001
ACEi / ARB	16/65 (25)	37/171 (22)	40/244 (17)	35/448 (8)	<0.001
Beta-blocker	8/65 (12)	40/171 (23)	30/244 (12)	33/448 (7)	<0.001
Systolic blood pressure – mmHg	113±28	116±29	120±29	122±26	0.011
<90 mmHg – n (%)	15 (17)	27 (13)	28 (9)	37 (7)	0.003
Diastolic blood pressure – mmHg	66±17	69±17	72±16	74±16	<0.001
<60 mmHg – n (%)	24 (28)	48 (23)	48 (16)	67 (12)	<0.001
Heart rate – bpm	80±22	79±23	78±21	78±19	0.913
>100 bpm – n (%)	12 (14)	32 (15)	40 (13)	56 (10)	0.222
Killip class IV – n (%)	26 (30)	55 (26)	44 (14)	78 (14)	<0.001
Lipid profiles – mg/dl					
Total cholesterol	132±69	153±19	186 ± 21	241±43	<0.001
HDL cholesterol	52±49	53±41	53 ± 41	56±43	0.643
Triglyceride	125±137	111±81	134 ± 98	136±84	0.008
LDL cholesterol	55±11	86±8	115 ± 34	132±47	<0.001
Other laboratory					
Creatinine – mg/dl	1.74±1.60	1.37±1.12	1.15±0.72	1.03±0.40	<0.001
GFR – ml/min/1.73 m^2	58±29	73±29	80±28	87±24	<0.001
CK – IU/l	3,277±3,527	3,204±3,626	3,090±3,240	3,519±3,250	0.318
CK-MB – IU/l	369±392	328±303	313±307	359±445	0.053
HbA1C – %	6.3±1.3	6.8±1.9	6.7±2.0	6.8±2.1	0.466

STEMI = ST-elevation myocardial infarction; ACEi = angiotensin-converting-enzyme inhibitor; ARB = angiotensin receptor blocker; HDL = high-density lipoprotein; LDL = low-density lipoprotein; GFR = glomerular filtration rate; CK = creatinine phosphokinase; CK-MB = creatinine phosphokinase isozyme MB; HbA1C = hemoglobin A1C

Univariate and multivariate analysis

In univariate analysis, the predictors that most associated with 1-year all-cause mortality were Killip class IV HR 5.32 (95% CI 4.15-6.82; $p<0.001$), heart failure need mechanical ventilator HR 5.06 (95% CI 3.76-6.80; $p<0.001$) and CPR HR 4.82 (95% CI 3.71-6.80; $p<0.001$). In multivariate analysis, the predictors that most associated with 1-year all-cause mortality were Killip class IV HR 2.40 (95% CI 1.78-3.24; $p<0.001$), heart failure need mechanical ventilator HR 2.25 (95% CI 1.64-3.09; $p<0.001$) and CPR HR 2.07 (95% CI 1.57-2.72; $p<0.001$). LDL cholesterol <100 mg/dl was significant in univariate analysis HR 1.46 (95% CI 1.09-1.94; $p = 0.009$), but it was not showed statistically significant in multivariate analysis HR 0.94 (95% CI 0.70-1.27; $p = 0.732$) (Table 4). After

Table 2. Percutaneous coronary intervention data of STEMI patients according to initial LDL cholesterol level

PCI data	Group 1 LDL-C <70 mg/dl (n = 87)	Group 2 LDL-C 70-99 mg/dl (n = 212)	Group 3 LDL-C 100-129 mg/dl (n = 309)	Group 4 LDL-C ≥130 mg/dl (n = 556)	*p*-value
Door-to-balloon time – min	68±78	89±162	74±102	65±105	0.046
Onset-to-balloon time – min	187±257	177±284	165±343	210±343	0.243
>6 hour – n (%)	18 (21)	41 (19)	51 (17)	120 (22)	0.347
Strategy – n (%)					0.588
Primary PCI	79 (91)	198 (93)	291 (94)	512 (92)	
Rescue PCI	8 (9)	14 (7)	18 (6)	44 (8)	
Number of vessel diseases – n (%)					0.470
Single vessel disease	43 (49)	103 (49)	152 (49)	252 (45)	
Double vessel disease	19 (22)	51 (24)	85 (28)	168 (30)	
Triple vessel disease	25 (29)	58 (27)	72 (23)	136 (25)	
Left main disease – n (%)	5 (6)	4 (2)	6 (2)	15 (3)	0.182
Culprit lesions – n (%)					0.056
Left anterior descending artery	35 (40)	111 (52)	165 (53)	325 (58)	
Right coronary artery	45 (52)	74 (35)	112 (36)	184 (33)	
Left circumflex artery	6 (7)	24 (11)	27 (9)	42 (8)	
Left main	0 (0)	2 (1)	3 (1)	5 (1)	
Bypass graft	1 (1)	1 (1)	2 (1)	0 (0)	
Segment of culprit lesions – n (%)					0.156
Proximal part	37 (49)	96 (49)	137 (49)	251 (49)	
Mid part	24 (31)	72 (37)	105 (37)	195 (38)	
Distal part	15 (20)	24 (12)	38 (13)	65 (12)	
Left main	0 (0)	3 (2)	3 (1)	5 (1)	
Stenting – n (%)	63 (77)	171 (81)	255 (83)	492 (89)	0.001
Complete revascularization – n (%)					0.471
Yes – in this admission	36 (46)	103 (52)	141 (39)	258 (49)	
Yes – staged PCI	4 (5)	22 (11)	26 (9)	56 (11)	
Yes – CABG surgery	0 (0)	2 (1)	5 (2)	3 (1)	
Using glycoprotein IIb/IIIa – n (%)	53 (61)	148 (70)	215 (70)	395 (71)	0.302

STEMI = ST-elevation myocardial infarction; PCI = percutaneous coronary intervention; CABG = coronary artery bypass graft

adjusting for age and sex (Model 1), group 1 and 2 still had more 1-year all-cause mortality than group 4, but adjusting for more variables (Model 2 and 3), all groups of LDL-C were not statistical significant, (Table 5).

Discussion

The present study showed that patients with lower LDL-C level was associated with higher 1-year all-cause mortality rate in STEMI patients underwent PCI. In multivariate analysis, the predictors of 1-year all-cause mortality were Killip class IV, CPR, LVEF ≤40%, culprit lesion at left main, creatinine >1.5 mg/dl, triple vessel diseases, heart failure need mechanical ventilator and age. LDL-C did not predicted the outcome.

The STEMI patients with lower LDL-C had worse outcome than higher LDL-C, especially patients with LDL-C <70 mg/dl, (group 1) who had highest 1-year all-cause mortality. In this group 1 patients who were older age, had more underlying diseases, more unstable vital signs, and more unfavorable laboratory such as more impaired kidney function and more myocardial injury than patients in other groups which might lead to poor outcome. After revascularization, group 1 patients were associated with more complex lesions such as triple vessel diseases and LM disease, lower

Table 3. Outcomes and complications of STEMI patients according to initial LDL cholesterol level

Outcomes	Group 1 LDL-C <70 mg/dl (n = 87)	Group 2 LDL-C 70-99 mg/dl (n = 212)	Group 3 LDL-C 100-129 mg/dl (n = 309)	Group 4 LDL-C ≥130 mg/dl (N=556)	*p*-value
1-year mortality rate – %	23.0	21.2	12.3	9.2	
Hazard ratio (compare to group 4)	2.72	2.45	1.35	1	
95% confidence interval	1.62 to 4.57	1.64 to 3.66	0.89 to 2.06	reference	
p-value	<0.001	<0.001	0.153	-	
Complications – n (%)					
Cardiopulmonary resuscitation	20 (23)	25 (12)	21 (7)	52 (9)	<0.001
Life-threatening ventricular arrhythmia	11 (14)	18 (9)	18 (6)	36 (7)	0.125
Cardiogenic shock	32 (42)	63 (32)	53 (19)	104 (20)	<0.001
Need IABP	26 (30)	44 (21)	37 (12)	73 (13)	<0.001
Need inotropic drug					0.001
1 drug	20 (23)	48 (23)	49 (16)	95 (17)	
2 drugs	13 (15)	19 (9)	18 (6)	38 (7)	
3 drugs	5 (6)	13 (6)	9 (3)	12 (2)	
Heart failure need mechanical ventilator	11 (13)	16 (8)	15 (5)	29 (6)	0.031
Left ventricular ejection fraction – %	48±15	49±13	50±14	50±12	0.510
≤40%	26 (30)	55 (26)	75 (24)	116 (21)	0.176

STEMI = ST-elevation myocardial infarction; IABP = intra-aortic balloon pump

Table 4. Univariate and multivariate Cox regression for 1-year all-cause mortality

Variables	Univariate			Multivariate		
	HR	95% CI	*p*-value	HR	95% CI	*p*-value
Age	1.05	1.04 to 1.06	<0.001	1.04	1.03 to 1.05	<0.001
Killip class IV	5.32	4.15 to 6.82	<0.001	2.40	1.78 to 3.24	<0.001
Creatinine >1.5 mg/dl	4.71	3.66 to 6.21	<0.001	1.77	1.32 to 2.38	<0.001
LDL cholesterol <100 mg/dl	1.46	1.09 to 1.94	0.009	0.94	0.70 to 1.27	0.732
Left main disease	3.75	2.42 to 5.83	<0.001	1.78	1.13 to 2.80	0.013
Triple vessel disease	2.10	1.63 to 2.70	<0.001	1.43	1.57 to 2.72	<0.001
Cardiopulmonary resuscitation	4.82	3.71 to 6.26	<0.001	2.25	1.64 to 3.09	<0.001
Heart failure need mechanical ventilator	5.06	3.76 to 6.80	<0.001	1.50	1.08 to 2.08	0.015
Left ventricular ejection fraction ≤40%	3.81	2.98 to 4.88	<0.001	2.07	1.57 to 2.72	<0.001

LDL = low-density lipoprotein

rate of stenting, less complete revascularization and more complication e.g.: CPR, cardiogenic shock, need inotropic drugs, need IABP than other groups which leaded to higher mortality rate.

The lower LDL-C is the worse prognosis in cardiovascular disease, which is called cholesterol paradox, had been first describe in 2003. Rauchhaus M et al found that the lower serum TC in congestive heart failure (CHF) patients, both ischemic and non-ischemic in etiology, was independently association with the higher mortality. They proposed that lipoproteins may be protective effect in CHF due to modulation and reduction of lipopolysaccharide bioactivity which led to lower inflammatory cytokine[6]. In ACS situation, cholesterol paradox was reported in non-ST-elevation myocardial infarction (NSTEMI) patients with hypercholesterolemia (TC>200 mg/dl in known hypercholesterolemia patients or LDL-C ≥100 mg/dl in newly-diagnosed hypercholesterolemia patients) were associated with lower in-hospital mortality rate. Wang T Y et al proposed that the previous hypercholesterolemia patients had more visits to medical staff, more healthier and more treated with medication such as statin or anti-platelet before they

Table 5. Prognostic value of LDL cholesterol for 1-year all-cause mortality (Cox proportional-hazard model)

LDL cholesterol level	No adjusted		Model 1		Model 2		Model 3	
(mg/dl)	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
<70	2.72 (1.62 to 4.57)	<0.001	1.87 (1.25 to 2.83)	0.020	2.71 (0.50 to 14.70)	0.247	3.99 (0.63 to 25.07)	0.139
70-99	2.45 (1.64 to 3.66)	<0.001	1.88 (1.25 to 2.83)	0.002	3.29 (0.88 to 12.27)	0.076	3.12 (0.74 to 13.14)	0.120
100-129	1.35 (0.89 to 2.06)	0.153	1.19 (0.73 to 1.71)	0.603	1.59 (0.56 to 4.50)	0.383	1.78 (0.59 to 5.39)	0.304
≥130	1 (reference)	-	1 (reference)	-	1 (reference)	-	1 (reference)	-

Model 1; adjusted for age and sex.

Model 2; adjusted for underlying diseases (diabetes mellitus, hypertension, dyslipidemia, coronary artery disease), smoking, medications prior to STEMI (anti-platelet, statin, ACEi/ARB, beta-blocker), lipid profiles (total cholesterol, HDL cholesterol, triglyceride), other laboratories (creatinine, glomerular filtration rate, creatinine phosphokinase, creatinine phosphokinase subtype MB) and vital signs (systolic blood pressure, diastolic blood pressure, heart rate) with variables in Model 1.

Model 3; adjusted for percutaneous coronary intervention data (number of vessel diseases, left main disease, culprit lesions of vessel, stenting, complete revascularization, using glycoprotein IIb/IIIa), complications (cardiopulmonary resuscitation, life-threatening ventricular arrhythmia, cardiogenic shock, need intra-aortic balloon pump, number of inotropic drugs, heart failure) and Killip class with variables in Model 2

had ACS and could more tolerated the adverse outcome than normal lipid patients[7]. Unlike our study, the lower LDL-C patients took more medications than the higher LDL-C patients, which reflected that lower LDL-C group had more co-morbidities to treated e.g. diabetes mellitus (DM), hypertension (HT) or CAD.

The other explanation of the cholesterol paradox effect's could be the atherosclerosis that had multiple risk factors. When plaque ruptured and ACS occurred, there were not only lipid profile involved in the cascade[8]. There were other risk markers predicted outcome of ACS better than lipid, one them is C-reactive protein (CRP). When using the CRP level combine with Framingham risk score and LDL-C, the results showed increased CRP level was strong and dependent predictor of cardiovascular event[9]. In the present study, there were no CRP data to provide better predicted mortality outcome.

After ACS occurred, there was acute phase response which changed the plasma protein and lipoprotein[10]. TG increased by up to 50% but TC, LDL-C and HDL-C decreased by up to 47%, 39% and 11%, respectively[11]. Lipoprotein started to change within 24-48 hours and maximal change within 4-7 days after ACS occurred. These changes were associated with extent and severity of myocardial necrosis but were not associated with type of reperfusion therapy[12]. In subgroup analysis of LUNAR study, mean LDL-C was decreased from 136.2 to 133.5 mg/dl within median time 26 hours and 43 hours after ACS was occurred. Similar changes were observed for TC and HDL-C. These 2-5% change of lipid profile will rebound to increase to baseline level in day 4. The present study confirmed that lipid profile remained stable within first 4 days after ACS[13]. The authors' study, admission lipid profile was obtained within 24 hours after diagnosed STEMI and the result would be acceptable and might be little lower level than the baseline lipid profile at time the ACS occurred.

Conclusion

The lower LDL-C level at time diagnosed STEMI was associated with higher 1-year all-cause mortality rate but the effect did not show after multivariate

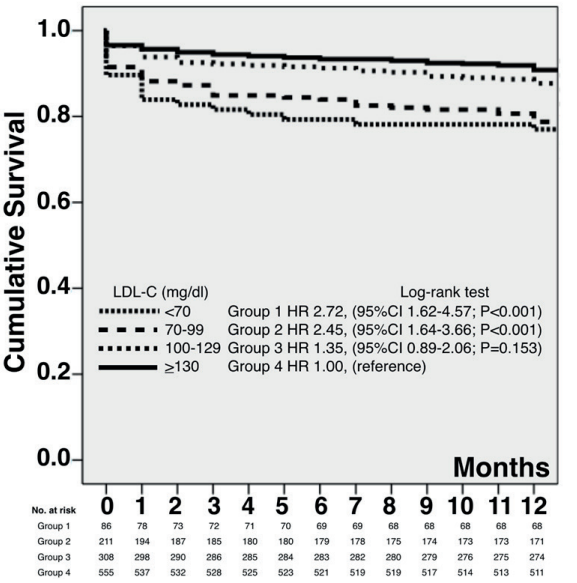

Figure 1. 1-year all-cause mortality rate of STEMI patients according to initial LDL-C level.

analysis. This represents LDL-C was not the risk factor to predict 1-year all-cause mortality in STEMI, but there were other factors that associated with the outcome. LDL-C was independent risk marker.

What is already known on this topic?

In the previous study showed the lower LDL-C level in ACS patients after PCI was associated with higher in-hospital mortality, 1-month and 1-year mortality[4]. However, there were no data in Thai population.

What is this study adds?

This is the first study of LDL-C and 1-year all-cause mortality after STEMI in Thailand, which confirmed the previous studies.

Acknowledgement

The authors wish to thank Jiranut Cholteesupachai, MD who shared the good comments in the present study and the Social Welfare Unit and the Computer Unit for providing the death registration database.

Potential conflicts of interest

The authors have no conflicts of interest.

References

1. Libby P. Current concepts of the pathogenesis of the acute coronary syndromes. Circulation 2001; 104: 365-72.
2. Srimahachota S, Boonyaratavej S, Kanjanavanit R, Sritara P, Krittayaphong R, Kunjara-Na-ayudhya R, et al. Thai Registry in Acute Coronary Syndrome (TRACS)--an extension of Thai Acute Coronary Syndrome registry (TACS) group: lower in-hospital but still high mortality at one-year. J Med Assoc Thai 2012; 95: 508-18.
3. Li J, Li X, Wang Q, Hu S, Wang Y, Masoudi FA, et al. ST-segment elevation myocardial infarction in China from 2001 to 2011 (the China PEACE-Retrospective Acute Myocardial Infarction Study): a retrospective analysis of hospital data. Lancet 2015; 385: 441-51.
4. Cho KH, Jeong MH, Ahn Y, Kim YJ, Chae SC, Hong TJ, et al. Low-density lipoprotein cholesterol level in patients with acute myocardial infarction having percutaneous coronary intervention (the cholesterol paradox). Am J Cardiol 2010; 106: 1061-8.
5. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972; 18: 499-502.
6. Rauchhaus M, Clark AL, Doehner W, Davos C, Bolger A, Sharma R, et al. The relationship between cholesterol and survival in patients with chronic heart failure. J Am Coll Cardiol 2003; 42: 1933-40.
7. Wang TY, Newby LK, Chen AY, Mulgund J, Roe MT, Sonel AF, et al. Hypercholesterolemia paradox in relation to mortality in acute coronary syndrome. Clin Cardiol 2009; 32: E22-E28.
8. Al Mallah MH, Hatahet H, Cavalcante JL, Khanal S. Low admission LDL-cholesterol is associated with increased 3-year all-cause mortality in patients with non ST segment elevation myocardial infarction. Cardiol J 2009; 16: 227-33.
9. Ridker PM, Rifai N, Rose L, Buring JE, Cook NR. Comparison of C-reactive protein and low-density lipoprotein cholesterol levels in the prediction of first cardiovascular events. N Engl J Med 2002; 347: 1557-65.
10. Balci B. The modification of serum lipids after acute coronary syndrome and importance in clinical practice. Curr Cardiol Rev 2011; 7: 272-6.
11. Rosenson RS. Myocardial injury: the acute phase response and lipoprotein metabolism. J Am Coll Cardiol 1993; 22: 933-40.
12. Pitt B, Loscalzo J, Ycas J, Raichlen JS. Lipid levels after acute coronary syndromes. J Am Coll Cardiol 2008; 51: 1440-5.
13. Miller M. Lipid levels in the post-acute coronary syndrome setting: destabilizing another myth? J Am Coll Cardiol 2008; 51: 1446-7.