In-hospital Initiation of PCSK9 Inhibitor and Short-term Lipid Profile Alteration, as well as In-hospital Mortality and Readmission Rate in Patients with Acute Myocardial Infarction

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Abstract

Background

Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors have been shown to improve cardiovascular outcomes when added to conventional statin therapy. This study aims to investigate the efficacy and safety of in-hospital initiation of PCSK9 inhibitor among patients with acute myocardial infarction(AMI) based on real-world experience.

Methods and Results

Data were collected from the Biobank of the First Affiliated Hospital of Xi'an Jiaotong University between January 2016 and December 2020. 7556 AMI patients were screened for eligibility. Propensity Score Match(PSM) was employed and covariates were age, sex, admission blood pressure and lipid profiles. Eligible participants were (1) propensity-matched 1:2:2 of Statin plus Evolocumab (dual therapy) vs. Statin vs. Statin plus Ezetimibe. 95 Statin plus Evolocumab users achieved significant decreased LDL level (0.92 ± 0.62 mmol/L in the 1st month and 1.17 ± 0.73 in the 3rd month) and a promising attainment rate of LDL(79.5% in 1st month and 80.0% in the 3rd month) as compared to the other two groups. (2) propensity-matched 1:2:2 of Statin plus Ezetimibe Evolocumab (triple therapy) vs. Statin vs. Statin plus Ezetimibe. Similarly, 75 triple medication users achieved significantly decreased LDL levels and a promising attainment rate of LDL as compared to the other two groups. In-hospital mortality, readmission rate within 3 months were then analyzed and decreased readmission rate was observed through PCSK9i therapy.

Conclusions

Based on the present single-centre real-world PSM-adjusted study, PCSK9i has been effective in short-term lipid control among AMI patients. The long-term effectiveness for reducing major cardiovascular events among AMI patients based on real-world experience remains to be explored.

Trial registration: The study was registered at ClinicalTrials.gov, Unique Protocol ID: 82100477-1.

Introduction

Due to the high rate of mortality and morbidity, acute myocardial infarction (AMI) plays as one of the leading health-threatening problems worldwide^{1, 2, 3}. Apart from acute reperfusion therapy including fibrinolytic drugs, percutaneous intervention(PCI), and coronary artery bypass graft surgery, secondary prevention medication is still the foundation for AMI patients. Especially, lipid lowering drugs treatments are cornerstones for acute and chronic management in AMI patients⁴⁻⁶.

The advent of Proprotein Convertase Subtilisin-Kexin Type 9(PCSK9) has brought a breakthrough in the treatment of atherosclerotic disease. PCSK9 is a secreted serine protease synthesized by the liver, which

can bind to and degrade LDL receptors, thereby reducing the clearance of serum LDL⁷. It has been shown by previous clinical studies that patients with the atherosclerotic disease still have poor LDL-C level control under the conventional statin and ezetimibe treatment. One observational study has indicated that after receiving statin treatment for at least 3 months, the standard rate of LDL-C in patients with very high-risk dyslipidemia is only 23.9%⁸. Thus, the recommendation of LDL target values for ASCVD patients tend to be lower and lower; and currently, for patients at very high risk, LDL reduction of $\geq 50\%$ from baseline and an LDL goal of <1.4 mmol/L (<55 mg/dL) are suggested^{9, 10}. A growing number of clinical studies of PCSK9i have proved that high-risk atherosclerosis patients can achieve satisfied blood lipid (especially LDL-C) control by combining PCSK9i on the basis of statin treatment, thus further reducing the risk of myocardial infarction, stroke and other events¹¹⁻¹³.

However, most of the previous studies discussing the clinical effectiveness of PCSK9i were based on chronic lipids management. In addition, according to contemporary guidelines^{14, 15}, PCSKi should be initiated when the initiation of statin and ezetimibe has not met the targeted LDL-C levels. Thus, few studies are focusing on the effectiveness, safety and cardiovascular benefits of PCSK9i on LDL-C management when initiated during the acute phase of cardiovascular events. In the latest Chinese consensus on dyslipidaemia management in patients with high-risk atherosclerosis disease¹⁶, it is recommended that the individualized lipid-lowering therapy should be selected according to the patient's baseline LDL-C level and the expected reduction range of the lipid-lowering regime. As a result, some AMI patients with high baseline LDL-C levels might opt to receive the combined treatment of statins and PCSK9i in the early stage of AMI onset. Whereas, clinical evidence for the efficacy and safety of this treatment regime that combined PCSKi and statin could be initiated during the acute phase of AMI for high-risk patients with high baseline LDL-C levels based on real-world experience are still limited.

Therefore, this study aims to investigate the efficacy and safety of in-hospital initiation of PCSK9i in patients with AMI based on real-world experience. Through this single-centre real-world study, in-hospital initiation of PCSK9i has been effective in short-term lipid control among AMI patients. The long-term effectiveness for reducing major cardiovascular events remains to be explored.

Methods And Results

Study design and Clinical data collection

This was a single-centre, retrospective cohort study. 7556 consecutive patients admitted to the cardiology department of the First Affiliated Hospital of Xi'an Jiaotong University for AMI between January 2016 and December 2020 were enrolled. The inclusion criteria were confirmed admission diagnosis of AMI and were defined based on the universal definition criteria by the American Cardiology College². The exclusion criteria were: (1) severe noncardiac disease with an expected survival of less than 1 year and unwillingness to participate; (2) patients over the age of 80 years or living far away from the hospital's

catchment area. A patient could only be included once. In hospital, PCSK9i was initiated according to the Chinese consensus on dyslipidaemia management in patients with high-risk atherosclerosis disease¹⁶.

The medical records of the patients were collected from the Biobank of the First Affiliated Hospital of Xi'an Jiaotong University, which contains the identified data derived from raw medical records, information about patients' detailed medical histories, present medication, biochemical and echocardiography results. Follow-up information, including in-hospital mortality, readmission rate as well as short-term lipid profile alteration were obtained via biobank, telephone and questionnaires by the general practitioner (GP). Written informed consent was obtained from all study participants, with ethnic committee approval at the First Affiliated Hospital of Xi'an Jiaotong University. The study was registered at ClinicalTrials.gov, Unique Protocol ID: 82100477-1.

Study Cohorts and Treatments

Participants were allocated into four groups according to different lipid-lowering strategies: 1). Statin, 2).Statin plus Ezetimibe, 3).Statin plus Evolocumab and 4). Statin plus Ezetimibe plus Evolocumab, contingent on whether they began Evolocumab during hospitalization. Patients were initiated with Evolocumab(140 mg per two weeks, subcutaneous injection) during hospitalization either due to extremely high LDL level and/or large area myocardial infarction based on the Chinese consensus on dyslipidaemia management in patients with high-risk atherosclerosis disease¹⁶. The use of Statin(20mg atorvastatin or 10mg rosuvastatin per day) or Statin plus Ezetimibe(10mg per day) therapy was based on the present guidelines for the treatment of AMI¹⁷.

Propensity Score Match

Because patients receiving Statin, Ezetimibe and Evolocumab according to their admission LDL level and varied a lot on baseline characters, Propensity Score Match(PSM) was employed and covariates were age, sex, admission blood pressure and lipid profiles. All eligible participants were (1) propensity-matched 1:2:2 of Statin plus Evolocumab vs. Statin vs. Statin plus Ezetimibe, and (2) propensity-matched 1:2:2 of Statin plus Ezetimibe Evolocumab vs. Statin vs. Statin plus Ezetimibe. The propensity score was performed by R 3.6.0 and package MatchIt and calculated using the values of the covariates.

Statistics

All statistical analyses were performed by using SPSS for Mac 22.0 (SPSS Inc, Chicago, IL), Graphpad 9.0 Prism (GraphPad Software San Diego, CA) or R 3.6.0. Data were presented as frequencies or percentages for categorical variables and mean $\pm$ SD for continuous variables, unless otherwise indicated. Simple t-test was used to compare continuous variables which are in the normal distribution. Mann-Whitney U test was used to compare continuous variables which do not conform to the normal distribution. χ^2 test was used

to compare categorical variables. One-way ANOVA was used to compare continuous variables of three or more independent (unrelated) groups. A value of $P < 0.05$ was considered statistically significant.

Results

Study Cohort

A total of 7556 AMI patients from the biobank database between January 2016 and December 2020 were screened for eligibility. After excluding those without revascularization or Statin based therapy, the remaining 5802 Statin users, 801 Statin plus Ezetimibe users and 170 Statin plus Evolocumab users (including 95 users without and 75 users with Ezetimibe), were selected for this study. Then, 1st and 3rd-month follow-up data were collected and analysed, including in-hospital mortality, readmission rate and lipid profiles(Fig.1).

As admission LDL level and crowd size vary among different lipid-lowering strategy groups, propensity score match(PSM) was performed for further analysis. 1st PSM based on Statin plus Evolocumab therapy and of these, 95 users were successfully matched with 190 Statin users and 190 Statin plus Ezetimibe users respectively. 2nd PSM based on Statin plus Ezetimibe plus Evolocumab(triple) therapy and of these, 75 users were successfully matched with 150 Statin users and 150 Statin plus Ezetimibe users respectively. The matched groups were well-balanced in demographic and clinical characteristics(Appendix Table1 and 2, appendix Fig1 and 2).

Baseline Characteristics and short-term follow-up in the whole cohort.

In the whole cohort, the mean ages were 62.90 ± 11.91 , 58.73 ± 12.16 , 57.72 ± 11.07 and 54.38 ± 11.77 years among Statin, Statin plus Ezetimibe, Statin plus Evolocumab and triple therapy groups. The admission LDL was 2.25 ± 0.74 , 2.95 ± 1.03 , 3.24 ± 0.98 and 3.90 ± 1.45 mmol/L respectively(Table.1), which is also inconsistent with the basic strategy that higher LDL level requires intensive lipid-lowering therapy in AMI patients. According to 2019 ESC/EAS Guidelines for the management of dyslipidaemias⁹, for patients at very high cardiovascular risk, LDL reduction of $\geq 50\%$ from baseline and an LDL goal of <1.4 mmol/L (<55 mg/dL) are recommended. We further analysed the control situation of LDL(<1.4 mmol/L) among each group. On admission, the target rate was 11.8%, 4.0%, 3.2% and 1.3% among each group; In the 1st month follow-up, around 37.0%, 28.8%, 79.5% and 55.3% of the whole patients reached treatment goal among each group and after 3month, 40.6%, 29.3%, 80.0% and 43.8% respectively(Table 1). Despite higher LDL levels at admission, AMI patients achieved a promising control rate in the short-term PCSK9i treatment.

Around 1.9% AMI patients died with Statin alone therapy and 0.7%, 0.8%, 0.0% among Statin plus Ezetimibe, Evolocumab dual and triple group during the 3month follow-up. The readmission rate was

4.0% and 5.3% with PCSK9i treatment and more than 10%, about 10.4% in Statin users and 14.7% in Statin plus Ezetimibe users respectively.

Statin plus Evolocumab therapy(dual therapy) based PSM analysis

95 Statin plus Evolocumab users, 190 Statin users and 190 Statin plus Ezetimibe users were well matched for this PSM analysis. The mean age was 59.42 ± 11.66 , 58.62 ± 12.73 and 58.53 ± 10.63 among each group and the admission LDL was 3.11 ± 1.02 , 3.24 ± 1.13 , and 3.24 ± 0.98 mmol/L respectively after PSM adjustments.

In admission, 0.5%, 2.1% and 3.2% AMI patients reached target LDL level among Statin, Statin plus Ezetimibe, Statin plus Evolocumab group. In the 1st month follow-up, the target rate was 31.0%, 21.3%, 79.5% and 29.4%, 21.4%, 80.0% after 3month respectively(Fig3.A). The mean LDL level was significantly decreased in Statin plus Evolocumab users compared to other two groups, with 0.92 ± 0.62 , 1.58 ± 0.44 , 1.96 ± 0.82 mmol/L in 1st month and 1.17 ± 0.73 , 1.61 ± 0.49 , 2.10 ± 0.82 mmol/L in the 3rd month. (Table2 and Fig3.B). Also, a similar trend was observed in ApoB level, with 0.39 ± 0.20 , 0.64 ± 0.16 , 0.70 ± 0.22 g/L in 1st month and 0.46 ± 0.20 , 0.59 ± 0.15 , 0.75 ± 0.22 g/L in the 3rd month follow-up respectively. Around 4.0% AMI patients were re-hospitalized with Statin plus Evolocumab therapy, and 11.6%, 10.5% readmission rate in the other two groups during the short-term follow-up.

Statin plus Ezetimibe plus Evolocumab therapy(triple therapy) based PSM analysis

In the 7556 AMI patients, 75 patients received triple therapy (Statin plus Ezetimibe plus Evolocumab) and were well matched with 150 Statin users and 150 Statin plus Ezetimibe users. The mean age was 52.59 ± 11.92 , 53.57 ± 11.29 and 53.67 ± 11.86 among each group and the admission LDL was 3.59 ± 0.95 , 3.82 ± 1.20 , and 3.90 ± 1.45 mmol/L respectively after PSM adjustments.

Around 1.3%, 0% and 1.3% AMI patients reached target LDL level among Statin, Statin plus Ezetimibe, triple therapy group during admission. In the 1st month follow-up, the achieved rate was 10.7%, 28.6%, 55.3% and 0.0%, 17.9%, 43.8% after 3month respectively(Fig4.A). Also, the mean LDL level was significantly decreased in triple therapy patients compared to the other two groups, with 1.43 ± 1.06 , 1.96 ± 0.49 , 2.04 ± 0.81 mmol/L in the 1st month and 1.40 ± 0.50 , 2.06 ± 0.42 , 2.37 ± 1.13 mmol/L after 3-month follow-up(Table3 and Fig4.B). Additionally, a similar decrease was observed in ApoB level, triple medication users reached 0.59 ± 0.29 , 0.61 ± 0.20 g/L in 1st month and 3rd month, compared to 0.75 ± 0.17 , 0.80 ± 0.13 g/L in Statin group and 0.76 ± 0.20 , 0.83 ± 0.29 g/L in Statin plus Ezetimibe group. 5.3% AMI patients re-hospitalized with triple therapy, which was 6.7% and 6.0% lower than those in Statin and Statin plus Ezetimibe users.

Discussion

In this single-centre, retrospective, population-based study, we have found that in-hospital initiation of PCSK9 has been effective in short-term lipid control among AMI patients. In both PCSK9i plus statin therapy with or without ezetimibe cohort, the mean LDL levels decrease by more than 60%. In addition, more than 50% of patients of the above two groups reach the target LDL-C level after one-month treatment. PCSK9i plus statin therapy with or without ezetimibe exhibit a better lipid-lowering effect as compared to statin or statin plus ezetimibe therapy.

Previously, PCSK9 inhibitors have been shown to reduce major cardiovascular events in secondary prevention among patients with and without clinically significant atherosclerotic cardiovascular disease (ASCVD). EVOPACS study is the first study to evaluate the very early application of PCSK9i in ACS patients¹⁸. It is confirmed that compared with a high-intensity statin alone, the addition of evolocumab brings a very significant reduction in LDL-C levels. Also, the ODYSSEY study demonstrated that alirocumab added to intensive statin therapy reduced the occurrence of both Type 1 and Type 2 MI among patients after a recent ACS¹⁹. Our study is the first to the best of our knowledge to investigate the lipid profile alteration as well as the short-term outcomes of PCSK9i initiated together with statin among AMI patients focusing on the real-world evidence. Similar to EVOPACS study¹⁸, our results show that LDL is significantly decreased via PCSK9i treatment after 1-month and 3-month follow-up when added to either statin or statin plus ezetimibe therapy. It is also noteworthy that ApoB, the primary apolipoprotein of LDL particles, exhibited the same trend as LDL among each treatment group, further proving the efficiency of PCSK9i in lipid-lowering strategy.

Besides, our study shows decreased readmission rate in the short-term follow-up via PCSK9i therapy. Mechanistically, previous studies have proved that after adjusting for ASCVD risk factors (including LDL level), serum PCSK9 levels were linearly associated with the fraction and amount of necrotic core tissue in coronary atherosclerosis²⁰. And compared with healthy volunteers, a significantly higher concentration of PCSK9 was observed in patients with ACS²¹. Additionally, increased PCSK9 levels were associated with higher platelet reactivity and might be a possible predictor of ischemic events in ACS patients undergoing PCI²². Taken together, our results have provided further clinical benefits that PCSK9i might exert lasting cardiovascular benefits on AMI patients apart from the lipid control. The reduction of the readmission rate is only based on a 3-month follow-up. Long-term follow-up data are warranted to further validate the efficacy of the in-hospital initiation of PCSK9i²³.

Notably, in terms of lipid-lowering effect, we have found that triple therapy of PCSK9i, statin and ezetimibe is not superior to statin plus evolocumab treatment before PSM adjustment. Several potential mechanisms may account for this phenomenon: First, most patients with triple treatment have extremely high LDL levels on admission, which was even 20% higher than that of the dual therapy group. Second, different lipid-lowering mechanisms also influence the effect. Ezetimibe inhibits the absorption of cholesterol from the small intestine and decreases the amount of cholesterol normally available to liver cells; the lower levels of cholesterol in the liver cells lead them to absorb more cholesterol from circulation

and thus “indirectly” lowering the levels of circulating cholesterol. On the other hand, evolocumab inhibits PCSK9 from binding to LDL receptors on the liver surface, and there are more LDL receptors on the surface of liver cells to remove LDL-C from the blood directly. At last, only 75 triple therapy users were included in this study and probably the reduced compliance may also affect the result.

There are several limitations in the current work. First, this study is limited in its single-centre, retrospective and observational nature. Future multicenter studies based on longer follow-up are required. Also, although the dosage and frequency of statin (20mg atorvastatin or 10mg rosuvastatin per day) are fixed, the data on whether statin therapy was started during or before hospital admission, and whether statin was not allowed due to its side effects are inaccessible so that it is not possible to match this situation in the four groups. However, the initiation, dosage and frequency of Evolocumab were fixed among all the groups in this study.

Conclusions

In conclusion, through this retrospective, PSM-adjusted, real-world cohort study, we have found that PCSK9i has been effective in short-term lipid and readmission control among AMI patients. The long term effectiveness, as well as the specific mechanism of PCSK9i for reducing major cardiovascular events, remain to be explored.

Declarations

Ethics approval and consent to participate

Written informed consent was obtained from all study participants, with ethic committee approval at the First Affiliated Hospital of Xi'an Jiaotong University.

Consent for publication

All authors have reviewed the final version of the manuscript and approved it for publication.

Availability of data and materials

The datasets used or analysed during the current study are available from the corresponding author on reasonable request.

Competing Interests

Not applicable.

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Authors' contributions

JS, BL and ZY participated in the design of the study. JL, BL and HL collected the patients' data and did the follow-up. HL, YL and GTJ performed the statistical analysis. YL, HW, YW and CW finished the patients' follow-up. JS, JL and BL drafted the manuscript. All authors approved the final manuscript.

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Tables

Table 1

Distribution of Demographic and Clinical Characteristics According to different lipid-lowering strategies in AMI patients.

		Statin	Statin+ Ezetimibe	Statin+ Evolocumab	Statin+ Ezetimibe+ Evolocumab
Age(years)		62.90±11.91	58.73±12.16	57.72±11.07	54.38±11.77
Female Sex(%)		20.3	21.0	15.7	18.8
SBP(mmHg)		123.41±22.24	125.20±22.79	127.54±22.20	122.72±23.04
DBP(mmHg)		77.17±14.53	79.01±15.02	82.36±14.49	78.98±15.32
EF(%)		51.11±10.27	52.81±10.39	51.11±10.27	51.27±9.72
Pro-BNP(pg/mL)		2071.69	1334.93	2071.69	1135.26
HbA1c(%)		6.33±1.45	6.29±1.44	6.33±1.45	6.75±2.00
Cre(umol/L)		77.94	70.48	69.36	62.97
UA(umol/L)		333.91	340.59	330.43	330.08
TG(mmol/L)		1.48±0.99	1.94±0.66	1.57±0.74	1.92±1.02
HDL(mmol/L)		0.93±0.22	0.98±0.24	1.00±0.21	1.00±0.23
TCDL(mmol/L)		3.93±0.99	4.76±1.25	4.96±1.19	5.69±1.50
ApoA(g/L)		1.05±0.19	1.09±0.19	1.09±0.17	1.09±0.20
ApoB(g/L)		0.77±0.20	0.95±0.24	1.00±0.23	1.16±0.29
ApoE(mg/L)		35.64±14.04	41.59±18.67	41.96±12.92	46.68±19.88
Lp(a) (mg/L)		261.68	302.22	330.56	368.69
Death (%)		1.9	0.7	0.8	0.0
Readmission (%)		10.4	14.7	4.0	5.3
LDL (mmol/L)	Admission	2.25±0.74	2.95±1.03	3.24±0.98	3.90±1.45
	Under Target	11.8	4.0	3.2	1.3
	Follow-up(1m)	1.60±0.51	1.87±0.75	0.93±0.63	1.41±1.02
	Under Target	37.0	28.8	79.5	55.3
	Follow-up(3m)	1.60±0.54	1.90±0.82	1.13±0.67	1.40±0.50
	Under Target	40.6	29.3	80.0	43.8

Data were shown in mean±SD, median or n (%).

SBP, systolic blood pressure; DBP, diastolic blood pressure; EF, eject fraction; HbA1c, haemoglobin A1c; Cre, creatinine; UA, uric acid; TG, triglyceride; LDL, low density lipoprotein; HDL, high density lipoprotein; ApoA, apolipoprotein A; ApoB, apolipoprotein B; ApoE, apolipoprotein E; TCDL, total cholesterol lipoprotein. Lp(a), Lipoprotein(a)

Table 2

Short-term lipid profile alteration among Statin plus Evolocumab / Statin/ Statin plus Ezetimibe lipid-lowering strategies in AMI patients after PSM adjustment.

		Statin	Statin+ Ezetimibe	Statin+ Evolocumab	<i>p</i>1	<i>p</i>2
LDL (mmol/L)	Admission	3.11±1.02	3.24±1.13	3.24±0.98	0.303	0.968
	Follow-up(1m)	1.58±0.44	1.96±0.82	0.92±0.62	<0.001	<0.001
	Follow-up(3m)	1.61±0.49	2.10±0.82	1.17±0.73	0.048	<0.001
TG (mmol/L)	Admission	1.56±1.01	1.55±0.72	1.57±0.74	0.927	0.823
	Follow-up(1m)	1.33±0.67	1.53±0.70	1.29±0.65	0.121	0.038
	Follow-up(3m)	1.57±1.13	1.66±0.84	1.18±0.51	0.214	0.011
HDL (mmol/L)	Admission	0.98±0.22	1.02±0.22	0.99±0.21	0.593	0.364
	Follow-up(1m)	1.01±0.22	1.05±0.25	1.03±0.24	0.042	0.654
	Follow-up(3m)	1.00±0.18	1.00±0.25	1.14±0.32	0.145	0.078
ApoA (g/L)	Admission	1.08±0.20	1.11±0.20	1.09±0.17	0.785	0.240
	Follow-up(1m)	1.17±0.18	1.18±0.18	1.21±0.19	0.223	0.390
	Follow-up(3m)	1.20±0.17	1.15±0.20	1.25±0.24	0.525	0.122
ApoB (g/L)	Admission	0.99±0.21	1.00±0.26	1.00±0.23	0.676	0.974
	Follow-up(1m)	0.64±0.16	0.70±0.22	0.39±0.20	<0.001	<0.001
	Follow-up(3m)	0.59±0.15	0.75±0.22	0.46±0.20	0.038	<0.001
ApoE (mg/L)	Admission	41.83±18.16	41.60±16.61	41.96±12.92	0.950	0.853
	Follow-up(1m)	33.82±11.29	34.46±11.93	20.79±9.39	<0.001	<0.001
	Follow-up(3m)	32.83±10.80	30.44±9.50	21.59±8.18	0.018	0.013
LP(a) (mg/L)	Admission	331.77	310.66	330.55	0.975	0.679
	Follow-up(1m)	281.96	283.66	271.18	0.902	0.876
	Follow-up(3m)	358.29	370.88	345.92	0.951	0.867
TCDL (mmol/L)	Admission	4.97±1.19	5.03±1.30	5.00±1.16	0.799	0.884
	Follow-up(1m)	3.16±0.58	3.57±0.90	2.37±0.74	<0.001	<0.001
	Follow-up(3m)	3.18±0.56	3.72±1.02	2.79±0.72	0.074	<0.001

Data were shown in mean±SD, median or n (%). *p*1, Statin plus Evolocumab vs. Statin; *p*2, Statin plus Evolocumab vs. Statin plus Ezetimibe.

TG, triglyceride; LDL, low density lipoprotein; HDL, high density lipoprotein; ApoA, apolipoprotein A; ApoB, apolipoprotein B; ApoE, apolipoprotein E; TCDL, total cholesterol lipoprotein. Lp(a), Lipoprotein(a)

Table 3

Short-term lipid profile alteration among Statin plus Ezetimibe plus Evolocumab / Statin/ Statin plus Ezetimibe lipid-lowering strategies in AMI patients after PSM adjustment.

		Statin	Statin +Ezetimibe	Statin +Ezetimibe +Evolocumab	<i>p1</i>	<i>p2</i>
LDL (mmol/L)	Admission	3.59±0.95	3.82±1.20	3.90±1.45	0.060	0.666
	Follow-up(1m)	1.96±0.49	2.04±0.81	1.43±1.06	0.001	0.001
	Follow-up(3m)	2.06±0.42	2.37±1.13	1.40±0.50	<0.001	<0.001
TG (mmol/L)	Admission	2.08±1.12	1.92±1.04	1.93±1.01	0.311	0.948
	Follow-up(1m)	1.72±0.71	1.58±0.68	1.50±0.72	0.148	0.539
	Follow-up(3m)	1.53±0.64	1.82±0.74	1.74±1.17	0.534	0.796
HDL (mmol/L)	Admission	0.98±0.24	1.01±0.22	1.00±0.23	0.472	0.818
	Follow-up(1m)	0.99±0.24	0.99±0.21	0.94±0.21	0.253	0.176
	Follow-up(3m)	1.03±0.29	1.00±0.27	0.95±0.18	0.316	0.491
ApoA (g/L)	Admission	1.09±0.19	1.11±0.20	1.09±0.21	0.877	0.456
	Follow-up(1m)	1.15±0.20	1.14±0.19	1.10±0.18	0.850	0.336
	Follow-up(3m)	1.11±0.16	1.15±0.23	1.12±0.14	0.525	0.606
ApoB (g/L)	Admission	1.16±0.29	1.15±0.24	1.16±0.29	0.269	0.730
	Follow-up(1m)	0.75±0.17	0.76±0.20	0.59±0.29	0.004	0.001
	Follow-up(3m)	0.80±0.13	0.83±0.29	0.61±0.20	0.008	0.010
ApoE (mg/L)	Admission	46.68±19.88	46.90±21.08	46.68±19.88	0.825	0.939
	Follow-up(1m)	31.00±8.62	36.77±16.37	28.92±13.96	0.573	0.061
	Follow-up(3m)	37.91±10.10	34.56±14.43	30.26±14.06	0.265	0.466
LP(a) (mg/L)	Admission	331.77	375.71	433.87	0.982	0.248
	Follow-up(1m)	450.00	283.66	271.18	0.587	0.851
	Follow-up(3m)	435.08	427.27	595.43	0.502	0.332

	up(3m)					
TCDL (mmol/L)	Admission	5.55±1.14	5.72±1.30	5.79±1.42	0.181	0.739
	Follow-up(1m)	3.52±0.65	3.55±0.89	2.82±1.18	<0.001	<0.001
	Follow-up(3m)	3.65±0.74	3.93±1.39	2.97±0.64	0.009	0.003

Data were shown in mean±SD, median or n (%). *p*1, Statin plus Ezetimibe plus Evolocumab vs. Statin; *p*2, Statin plus Ezetimibe plus Evolocumab vs. Statin plus Ezetimibe.

TG, triglyceride; LDL, low density lipoprotein; HDL, high density lipoprotein; ApoA, apolipoprotein A; ApoB, apolipoprotein B; ApoE, apolipoprotein E; TCDL, total cholesterol lipoprotein. Lp(a), Lipoprotein(a).

Figures

Figure 1. Patients Selection, Propensity Score Matching and Follow-up.

Figure 1

Please See image above for figure legend.

Figure.2 LDL level and compliance rate on admission and short-term follow-up among different lipid-lowering strategies in AMI patients.

(A). LDL level and compliance rate among different lipid-lowering strategies on admission and 1st and 3rd month follow-up. Each point represent one AMI patient's lipid data. (B). LDL alteration among different lipid-lowering strategies on admission and 1st and 3rd month follow-up. Data were shown in mean±SEM LDL Target Level=1.4mmol/L.

Figure 2

Please See image above for figure legend.

Figure3. Lipid profile alterations and short-term follow-up among Statin plus Evolocumab / Statin/ Statin plus Ezetimibe lipid-lowering strategies in AMI patients after PSM adjustment.

(A). LDL level and compliance rate among different lipid-lowering strategies on admission and 1st and 3rd month follow-up. Each point represent one AMI patient's lipid data after PSM adjustment. LDL(B) and ApoB(C) alteration among different lipid-lowering strategies on admission and 1st and 3rd month follow-up. Data were shown in mean±SEM. (D).Readmission rate among each group.

95 Statin plus Evolocumab users, 190 Statin plus Ezetimibe users and 190 statin users were chosen after 1:2:2 propensity score matching. For statistical analysis one-way ANOVA followed by Sidak's multiple comparison test was applied, *p < 0.05, ***p < 0.001.

Figure 3

Please See image above for figure legend.

Figure4. Lipid profile alterations and short-term follow-up among Statin plus Ezetimibe plus Evolocumab / Statin/ Statin plus Ezetimibe lipid-lowering strategies in AMI patients after PSM adjustment.

(A). LDL level and compliance rate among different lipid-lowering strategies on admission and 1st and 3rd month follow-up. Each point represent one AMI patient's lipid data after PSM adjustment. LDL(B) and ApoB(C) alteration among different lipid-lowering strategies on admission and 1st and 3rd month follow-up. Data were shown in mean±SEM. (D).Readmission rate among each group. 75 Statin plus Ezetimibe plus Evolocumab users, 150 Statin plus Ezetimibe users and 150 statin users were chosen after 1:2:2 propensity score matching. For statistical analysis one-way ANOVA followed by Sidak's multiple comparison test was applied, *p < 0.05, **p < 0.01, ***p < 0.001.

Figure 4

Please See image above for figure legend.

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