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Original Article

Effect of loading dose atorvastatin on postoperative cardiovascular events in elderly patients with acute myocardial infarction undergoing percutaneous coronary intervention

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Abstract: Objective: To evaluate the effects of loading-dose atorvastatin on cardiac function, lipid metabolism, inflammatory response, and cardiovascular events in elderly patients with acute myocardial infarction (AMI) undergoing percutaneous coronary intervention (PCI). Methods: A retrospective analysis was conducted on 128 elderly patients with AMI (≥60 years) who underwent PCI. Patients were divided into a loading-dose group (group A; 40-80 mg loading dose, n=71) and a conventional-dose group (group B; 20 mg or without a loading dose, n=57). Cardiac functionindices, including cardiac output, left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD), lipid profiles (total cholesterol [TC], triglyceride [TG], low-density lipoprotein cholesterol [LDL-C], high-density lipoprotein cholesterol [HDL-C]), and inflammatory markers (C-reactive protein [CRP], procalcitonin [PCT]) were compared between the two groups before and after treatment. Major adverse cardiovascular events [MACEs] within 3 months after PCI and medication safety were also evaluated. Results: After treatment, cardiac output, LVEF, and HDL-C levels were significantly increased, while LVEDD, CRP, PCT, TC, TG, and LDL-C levels were significantly decreased in both groups, with significantly greater improvements observed in group A compared to group B (all $P<0.05$). The incidence of MACEs was significantly lower in group A than in group B (12.7% vs. 28.1%, $P=0.027$). No serious adverse drug reactions occurred in either group. Conclusion: Loading-dose atorvastatin can improve cardiac function, regulate lipid metabolism, suppress inflammatory responses, and reduce the incidence of short-term MACE in elderly AMI patients after PCI, while demonstrating good safety and clinical application value.

Keywords: Loading-dose, atorvastatin, percutaneous coronary intervention, acute myocardial infarction, cardiovascular events

Introduction

Clinical data show that acute myocardial infarction (AMI) represents the most severe clinical manifestation of coronary heart disease, characterized by acute onset, rapid progression, and high mortality [1, 2]. Currently, percutaneous coronary intervention (PCI) has become the preferred choice for elderly patients with AMI, as it can rapidly restore blood flow in the infarct-related artery and achieve myocardial reperfusion [3]. However, in clinical practice, the risk of postoperative cardiovascular events remains high in elderly patients due to factors such as multi-vessel coronary artery disease, multiple comorbidities, and impaired cardiac reserve function [4, 5]. Therefore, it is vital to optimize perioperative pharmacologic management for to improve the prognosis of elderly patients undergoing PCI.

Previous studies have demonstrated that statins exert not only lipid-lowering effects but also pleiotropic effects, including anti-inflammatory activity, plaque stabilization, and improvement of endothelial function, making them a first-line therapy for coronary heart disease [6]. Recent studies have shown that preoperative loading-dose atorvastatin before PCI can rapidly inhibit inflammatory responses, alleviate myocardial ischemia-reperfusion injury, and thus reduce the risk of perioperative myo-

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cardial infarction and major adverse cardiovascular events (MACEs) [7, 8]. However, most previous studies have focused on a younger population, and clinical evidence regarding the application of loading-dose atorvastatin in elderly patients with AMI remains insufficient. Also, the efficacy and safety of statin loading-dose therapy in elderly patients require further validation due to age-related liver and kidney dysfunction, altered pharmacokinetics, and complex medication regimens [9].

Therefore, this study retrospectively analyzed the clinical data of elderly patients with AMI who underwent PCI at our institution from January 2019 to December 2022. The effects of preoperative loading-dose (40-80 mg) and conventional-dose (20 mg or no loading dose) atorvastatin on postoperative cardiac function, lipid metabolism, inflammatory responses, and short-term MACE incidence, as well as medication safety, were compared between the two groups. This study aimed to provide clinical reference for optimizing statin therapy during perioperative period of PCI in elderly patients with AMI.

Patients and methods

Study population

The clinical data of elderly patients with AMI who underwent PCI at Sir Run Run Shaw Hospital Affiliated to Zhejiang University School of Medicine between January 2019 and December 2022 were retrospectively collected. According to the preoperative administration regimen of atorvastatin, patients were divided into a loading-dose group (group A; loading dose of 40-80 mg, n=71) and a conventional-dose group (group B; conventional dose of 20 mg or no loading dose, n=57). This study was approved by the Ethics Committee of Sir Run Run Shaw Hospital.

Inclusion and exclusion criteria

Inclusion criteria: (1) Age ≥60 years; (2) Diagnosis of AMI according to the diagnostic criteria specified in the Guidelines for the Diagnosis and Treatment of Acute ST-Segment Elevation Myocardial Infarction or the Guidelines for the Diagnosis and Treatment of Non-ST-Segment Elevation Acute Coronary Syndrome [10]; (3) Admission within 12 hours after symptom onset, with successful emergency PCI; (4) Complete clinical data available.

Exclusion criteria: (1) Previous history of coronary artery bypass grafting (CABG); (2) Severe hepatic or renal insufficiency (alanine aminotransferase >3 times the upper limit of normal or estimated glomerular filtration rate <30 $mL \cdot min^{-1} \cdot 1.73\ m^{-2}$); (3) Previous history of statin intolerance (such as myalgia, significant increase in muscle enzymes); (4) Severe infection, malignant tumor, or autoimmune diseases; (5) History of cardiogenic shock or pre-hospital cardiac arrest.

Treatment methods

All patients received standardized AMI treatment according to relevant clinical guidelines after admission, including antiplatelet therapy (aspirin, clopidogrel, or ticagrelor), anticoagulant therapy, and β-blockers when necessary, and angiotensin converting enzyme inhibitors (ACEIs)/angiotensin II receptor antagonists (ARBs). Emergency PCI was performed by experienced cardiovascular interventional physicians, and the procedural techniques followed standard PCI protocols [11]. Patients in group A received a loading dose of atorvastatin before PCI, whereas those in group B received a conventional dose or no loading dose. After PCI, both groups continued standard-dose statin treatment. The following indices were evaluated before and after treatment: cardiac function index, including cardiac output (CO), left ventricular ejection fraction (LVEF), and left ventricular end diastolic diameter (LVEDD); lipid metabolic indices, including total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C); and inflammatory makers, including C-reactive protein (CRP) and procalcitonin (PCT).

Observation indicators

Primary outcome measures: The occurrence of MACEs within 3 months after PCI was recorded, including cardiac death, non-fatal myocardial infarction, target vessel revascularization, and rehospitalization due to severe heart failure, among others.

Secondary outcome measures: Drug-related adverse reactions during treatment were

recorded, including abnormal liver function (alanine aminotransferase/aspartate aminotransferase elevation >3 times the upper limit of normal), myalgia or elevated creatine kinase, gastrointestinal symptoms, and other adverse events. Calculation of SAQ Scores: the Seattle Angina Questionnaire (SAQ) comprises 19 items across five dimensions: physical limitation, anginal stability, anginal frequency, treatment satisfaction, and disease perception. Each item was scored ordinally. For each dimension, the raw score was summed and then transformed to a 0-100 standardized score using the formula: Standardized Score = (Actual Raw Score - Minimum Possible Score)/(Maximum Possible Score - Minimum Possible Score) × 100.

Higher scores indicate better function, fewer symptoms, or greater treatment satisfaction. Specifically, for anginal frequency, scores of 0-30, 31-60, 61-99, and 100 correspond to daily, weekly, monthly, and no angina episodes, respectively.

Statistical methods

SPSS 26.0 statistical software was used for data analysis. Continuous variables conforming to a normal distribution were expressed as mean ± standard deviation (SD). Paired-samples t-test was used to compare variables before and after treatment within each group, and independent-samples t-test was used for comparison between groups. Categorical variables were expressed as the number of cases (percentage), and comparisons between groups were performed using the chi-square test or Fisher's exact test. A two-sided *P* value <0.05 was considered significant.

Results

Comparison of baseline characteristics between the two groups

The baseline characteristics of the two groups were well balanced and comparable. No significant differences were observed between two groups in demographic data, including age, sex, or BMI (P>0.05). Similarly, there were no significant differences in the history of hypertension, diabetes, hyperlipidemia, stroke, chronic kidney disease, smoking history, or alcohol consumption history (P>0.05). Furthermore, there were no significant differences between the two groups regarding AMI type, number of infarct-related vessels, number of coronary lesions, time from onset to PCI, number of implanted stents, or other disease-related baseline characteristics. Preoperative liver and kidney function, fasting blood glucose, and the use of concomitant medications, including β-blockers, ACEI/ARB, and antiplatelet agents, were also comparable between the two groups (P>0.05). Details are shown in **Table 1**.

Changes in cardiac function before and after treatment between the two groups

Before treatment, no significant differences were observed between the two groups in terms of cardiac output, LVEF, or LVEDD (all P>0.05). After treatment, cardiac output and LVEF were significantly increased, whereas LVEDD was significantly decreased compared with baseline values in both groups (P<0.05). Moreover, group A exhibited significantly greater improvements in these cardiac function indices after treatment (all P<0.05; **Table 2**).

Comparison of lipid profile changes before and after treatment between the two groups

Before treatment, no significant differences were observed between the two groups in terms of TC, TG, LDL-C, or HDL-C levels (all P>0.05). After treatment, the levels of TC, TG and LDL-C were significantly decreased, while HDL-C levels were significantly increased compared to baseline values in both groups (P<0.05), and these improvements were significantly greater in group A than that group B (P<0.05; **Table 3**).

Comparison of inflammatory markers before and after treatment between the two groups

Before treatment, no significant differences were observed between the two groups in inflammatory markers CRP and PCT (all P> 0.05). After treatment, the levels of CRP and PCT were significantly decreased compared with baseline levels in both groups (all P<0.05). Moreover, the levels of these two markers were significantly lower in group A than in group B (both P<0.05; **Figure 1**).

Comparison of the incidence of MACEs during postoperative follow-up between the two groups

During the 3-month follow-up period after PCI, the overall incidence of MACEs in group A was

Table 1. Comparison of baseline characteristics between the two groups

Indicator	Group A (loading dose 40-80 mg, n=71)	Group B (conventional/ non-loading dose, n=57)	statistic	*P* value
General Demographic Data	-	-	-	-
Age (years, $\bar{x} \pm s$)	68.5±5.2	69.1±5.5	0.642	0.522
Male, n (%)	42 (59.15)	32 (56.14)	0.121	0.728
Body mass index (kg/m^2, $\bar{x} \pm s$)	24.2±3.1	23.9±3.3	0.534	0.594
Age stratification, n (%)	-	-	0.145	0.703
60-74 years old	52 (73.24)	40 (70.18)	-	-
≥75 years old	19 (26.76)	17 (29.82)	-	-
Past medical history and living habits	-	-	-	-
History of hypertension, n (%)	48 (67.61)	39 (68.42)	0.009	0.924
History of type 2 diabetes, n (%)	26 (36.62)	21 (36.84)	0.001	0.992
History of hyperlipidemia, n (%)	32 (45.07)	24 (42.11)	0.115	0.734
History of ischemic stroke, n (%)	11 (15.49)	10 (17.54)	0.095	0.758
History of hemorrhagic stroke, n (%)	2 (2.82)	1 (1.75)	Fisher exact test	1.000
History of chronic heart failure, n (%)	8 (11.27)	7 (12.28)	0.032	0.858
History of chronic kidney disease, n (%)	10 (14.08)	9 (15.79)	0.071	0.790
Smoking history (current/past), n (%)	30 (42.25)	22 (38.60)	0.170	0.680
Drinking history (current/past), n (%)	25 (35.21)	19 (33.33)	0.048	0.826
The incidence and characteristics of coronary artery disease	-	-	-	-
Acute myocardial infarction type, n (%)	-	-	0.069	0.793
ST-segment elevation myocardial infarction (STEMI)	39 (54.93)	30 (52.63)	-	-
Non-ST-segment elevation myocardial infarction (NSTEMI)	32 (45.07)	27 (47.37)	-	-
Infarct-related artery, n (%)	-	-	0.158	0.983
The left anterior descending branch (LAD)	35 (49.30)	27 (47.37)	-	-
Right coronary artery (RCA)	20 (28.17)	17 (29.82)	-	-
Left circumflex branch (LCX)	12 (16.90)	10 (17.54)	-	-
Other/multiple infarct-related vessels	4 (5.63)	3 (5.26)	-	-
Number of coronary artery lesions, n (%)	-	-	0.070	0.965
Single-vessel disease	22 (30.99)	19 (33.33)	-	-
Double-vessel disease	31 (43.66)	24 (42.11)	-	-
Three-vessel disease	18 (25.35)	14 (24.56)	-	-
Time from onset to PCI)	12.3±8.5	13.1±9.2	0.521	0.603
Number of stent implantation (pieces)	1.8±0.7	1.7±0.8	0.748	0.456
Preoperative laboratory and medication baseline	-	-	-	-
Preoperative serum creatinine (μmol/L)	88.5±22.3	90.2±24.1	0.415	0.679
Preoperative alanine aminotransferase (U/L)	32.1±15.2	33.5±16.8	0.498	0.619
Preoperative aspartate aminotransferase (U/L)	35.2±18.5	36.8±20.1	0.472	0.637
Preoperative fasting blood glucose (mmol/L)	5.8±1.2	5.9±1.4	0.436	0.663
History of preoperative β-blocker use, n (%)	28 (39.44)	21 (36.84)	0.091	0.763
Preoperative ACEI/ARB drug use history, n (%)	30 (42.25)	23 (40.35)	0.047	0.829
Preoperative antiplatelet drug use history, n (%)	65 (91.55)	50 (87.72)	0.518	0.472
History of preoperative use of other statins, n (%)	12 (16.90)	10 (17.54)	0.009	0.924

12.7% (9/71), significantly lower than 28.1% (16/57) in group B (χ^2=4.891, P=0.027). The incidences of cardiac death, recurrent myocardial infarction and target vessel revascularization were lower in group A than in group B; however, the differences were not significant (all P>0.05; **Table 4**).

Comparison of hospitalization days and re-hospitalization rates between the two groups

The length of hospitalization days was significantly shorter in group A than in group B (8.5±2.3 days vs. 10.8±3.1 days; t=4.712, P<0.001). During the 3-month follow-up period

Table 2. Comparison of cardiac function indices before and after treatment between the two groups ($\bar{x} \pm s$)

Group	Time	Cardiac output (L/min)	Left ventricular ejection fraction (%)	Left ventricular end-diastolic diameter (mm)
Group A (n=71)	Before treatment	4.02±0.45	43.5±5.2	55.3±4.1
	After treatment	5.11±0.52*,#	54.6±4.8*,#	48.2±3.9*,#
Group B (n=57)	Before treatment	4.08±0.48	43.9±5.0	55.0±4.3
	After treatment	4.56±0.50*	48.3±4.9*	51.7±4.0*

Note: *P<0.05, compared with baseline value; #P<0.05, compared with group B after treatment.

Table 3. Comparison of blood lipid profiles before and after treatment between the two groups (x ± s, mmol/L)

Group	Time	Total cholesterol	Triglycerides	Low density lipoprotein cholesterol	High density lipoprotein cholesterol
Group A (n=71)	Before treatment	5.42±0.85	2.03±0.62	3.56±0.78	0.92±0.18
	After treatment	3.78±0.67*,#	1.45±0.48*,#	2.11±0.56*,#	1.24±0.20*,#
Group B (n=57)	Before treatment	5.38±0.82	2.06±0.59	3.52±0.75	0.93±0.19
	After treatment	4.45±0.73*	1.78±0.52*	2.78±0.63*	1.08±0.21*

Note: *P<0.05, compared to baseline value; #P<0.05, compared to group B after treatment.

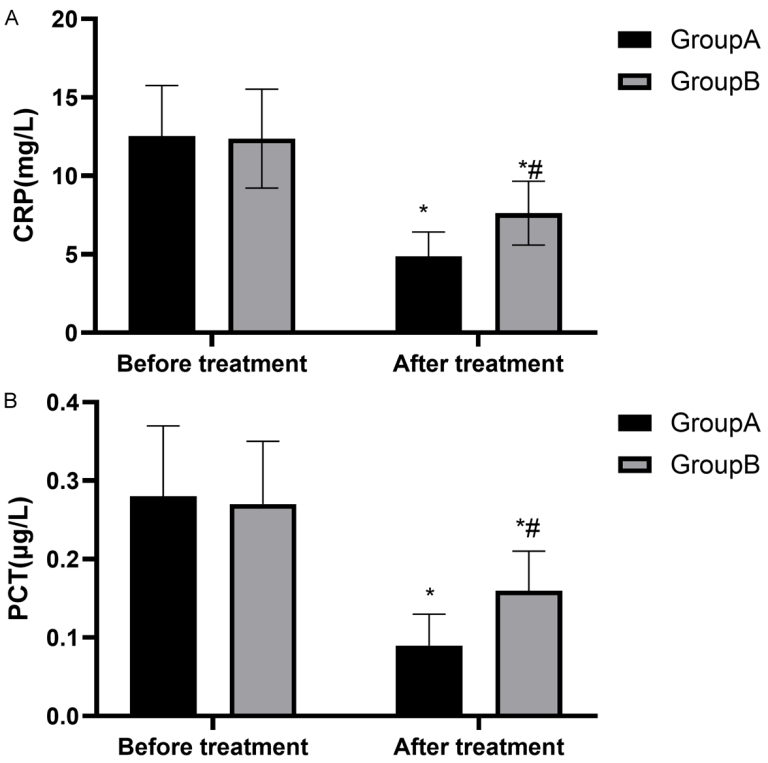

Figure 1. Comparison of serum CRP (A) and PCT (B) levels before and after treatment between the two groups. Notes: CRP, C-reactive protein; PCT, procalcitonin. *P<0.05, compared toh baseline value; #P<0.05, compared to group B after treatment.

after PCI, the rehospitalization rate in group A was 7.0% (5/71), significantly lower than 21.1% (12/57) in group B (χ^2=5.429, P=0.020; **Table 5**).

Comparison of adverse drug reactions between the two groups

No serious adverse drug reactions occurred in either group during the treatment period. The overall incidence of adverse reactions was 4.2% in group A and 3.5% in group B. No significant differences were observed between the two groups in the incidence of serious adverse reactions, general adverse reactions, or total adverse reactions (all P>0.05; **Table 6**).

Comparison of post-PCI quality of life between the two groups

After PCI, the scores of the five dimensions of the Seattle Angina Questionnaire (SAQ), including physical activity limitation, angina pectoris stability, angina frequency, treatment satisfaction, and disease perception, were significantly high-

Table 4. Comparison of the incidence of MACE within 3 months after operation between the two groups [n (%)]

Group	Number of cases	Cardiac death	Recurrent myocardial infarction	Target vessel revascularization	Total MACE
Group A	71	1 (1.4)	3 (4.2)	5 (7.0)	9 (12.7)
Group B	57	2 (3.5)	5 (8.8)	9 (15.8)	16 (28.1)
χ^2 value		0.613	1.164	2.533	4.891
P value		0.434	0.281	0.111	0.027

Note: MACE, major adverse cardiovascular events.

Table 5. Comparison of hospitalization days and rehospitalization rate between the two groups

Group	Number of cases	Hospitalization days (days, $\bar{x} \pm s$)	Rehospitalization rate [n (%)]
Group A	71	8.5±2.3	5 (7.0)
Group B	57	10.8±3.1	12 (21.1)
Statistical value		t=4.712	χ^2=5.429
P value		<0.001	0.020

er in group A than in group B (all P<0.001; **Table 7**), suggesting greater improvement in quality of life caused by loading-dose atorvastatin therapy.

Multivariate logistic regression analysis of MACEs

To adjust further for potential confounding factors, multivariate Logistic regression analysis was performed with the occurrence of MACE within 3 months after PCI as the dependent variable. After adjusting for intraoperative factors (operation time, contrast volume, number of implanted stents), postoperative concomitant medications (β-blockers, ACEI/ARB, antiplatelet drugs), and baseline characteristics (age, diabetes, hypertension), loading-dose atorvastatin remained an independent protective factor against MACEs (OR=0.42, 95% CI: 0.21-0.84, P=0.014) (**Table 8**).

Discussion

Recent epidemiologic studies have shown that AMI remains a common and life-threatening cardiovascular emergency among elderly population, and percutaneous coronary intervention (PCI) is currently an effective revascularization strategy and a major intervention for improving patient survival [12, 13]. However, in clinical practice, elderly patients with AMI often present with multiple vascular lesions, complex coronary atherosclerotic plaques, and reduced cardiac reserve. Despite successful PCI, they still face a considerable risk of MACEs and suboptimal recovery of cardiac function. Therefore, reducing the incidence of MACEs and improving clinical outcome are of great significance in such patients [14].

Several guidelines recommend statins as the cornerstone therapy for secondary prevention of coronary heart disease. However, the application value of loading-dose statin therapy during the perioperative period of PCI remained to be explored. Previous studies have shown that preoperative administration of high-load dose atorvastatin can rapidly enhance lipid-lowering effects, stabilize atherosclerotic plaques, and attenuate inflammatory responses. However, most of these studies have focused on the general population, and the clinical efficacy, anti-inflammatory effects, lipid-lowering effects, and safety profile of loading-dose atorvastatin in elderly AMI patients required further investigation [15, 16]. Therefore, optimizing the statin administration regimen before PCI in elderly patients with AMI may be of substantial clinical significance for improving postoperative cardiac function, reducing the incidence of MACEs, and enhancing quality of life.

Currently, the perioperative application strategies of atorvastatin in PCI mainly include conventional-dose maintenance therapy and preoperative loading-dose intensive therapy. Previous studies have demonstrated that a loading dose of atorvastatin (40-80 mg) can rapidly reduce LDL-C levels by competitively inhibiting HMG-CoA reductase and exert pleiotropic effects, including improvement in endothelial function, suppression of inflammatory responses, and stabilization of atherosclerotic plaques

Table 6. Comparison of the incidence of drug-related adverse reactions between the two groups [n (%)]

Group	Number of cases	Severe adverse reactions	General adverse reactions	Total incidence of adverse reactions
Group A	71	0 (0.0)	3 (4.2)	3 (4.2)
Group B	57	0 (0.0)	2 (3.5)	2 (3.5)
χ^2 value		-	0.042	0.042
P value		-	0.838	0.838

Note: Severe adverse reactions were judged by CTCAE ≥3. The common adverse reactions included mild gastrointestinal discomfort and transient abnormal liver function.

Table 7. Comparison of SAQ scores between the two groups ($\bar{x} \pm s$, points)

Group	Number of cases	Physical activity limitation (PL)	Angina stable state (AS)	Angina frequency (AF)	Treatment satisfaction (TS)	Disease awareness (DP)
Group A	71	78.5±8.2	82.4±7.6	79.3±8.5	81.6±7.9	80.2±8.1
Group B	57	68.3±9.1	71.5±8.4	68.7±9.2	70.8±8.6	69.5±8.9
t value		6.612	7.683	6.711	7.284	7.015
P value		<0.001	<0.001	<0.001	<0.001	<0.001

Note: SAQ, Seattle Angina Questionnaire.

Table 8. Multivariate Logistic regression analysis of factors affecting MACE within 3 months after PCI

Variable	β value	SE	Wald χ^2	OR value	95% CI	*P* value
Loading dose of atorvastatin (vs. conventional/no loading dose)	-0.868	0.352	6.082	0.42	0.21-0.84	0.014
Operation time (per 10 min increase)	0.156	0.089	3.074	1.17	0.98-1.39	0.080
Contrast volume (per 10 mL increase)	0.098	0.067	2.139	1.10	0.97-1.26	0.144
Number of stents (per 1 stent increase)	0.215	0.142	2.292	1.24	0.94-1.64	0.130
β-blockers (vs. no use)	-0.245	0.312	0.617	0.78	0.42-1.45	0.432
ACEI/ARB (vs. no use)	-0.312	0.298	1.096	0.73	0.41-1.31	0.295
Antiplatelet drugs (vs. no use)	-0.198	0.354	0.313	0.82	0.41-1.64	0.576
Age (per 1 year increase)	0.067	0.045	2.216	1.07	0.98-1.17	0.137
History of diabetes (vs. no)	0.356	0.298	1.427	1.43	0.80-2.56	0.232
History of hypertension (vs. no)	0.289	0.287	1.014	1.34	0.76-2.35	0.314

Note: MACE, major adverse cardiovascular events; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CI, confidence interval; OR, odds ratio. Variables with P<0.10 in univariate analysis were entered into the multivariate Logistic regression model using the Enter method. The dependent variable was MACE occurrence (1 = yes, 0 = no). The independent variable was loading dose of atorvastatin (1 = loading dose group, 0 = conventional/no loading dose group).

[17, 18]. Compared to a previous study that reported a similar magnitude of LDL-C and CRP reduction in elderly patients receiving high-dose atorvastatin, our findings showed a more pronounced improvement in LVEF, which may be attributed to the earlier administration of the loading dose and to differences in baseline inflammatory status [9]. Additionally, the reduction in MACE in our study was comparable to that reported in another study, though their cohort had a lower proportion of elderly patients.

In this study, cardiac function, lipid profiles, and inflammatory markers were improved after PCI in both groups, and patients in group A had greater improvements in these indices. The potential mechanisms may be explained as follows: conventional-dose atorvastatin mainly exerts long-term secondary preventive effects by inhibiting cholesterol synthesis and stabilizing atherosclerotic plaques, whereas its lipid-lowering and anti-inflammatory effects develop relatively gradually. In contrast, preoperative loading-dose atorvastatin can rapidly increase

plasma drug concentrations, promote greater reductions in TC, TG, and LDL-C, and attenuate inflammatory responses by suppressing the release of inflammatory mediators such as CRP and PCT. Our findings are consistent with previous studies demonstrating the cardioprotective effects of loading-dose atorvastatin administration [19, 20]. Moreover, the greater reductions in lipid and inflammatory markers observed in group A suggested that loading-dose atorvastatin can further enhance lipid-lowering and anti-inflammatory effects, improve myocardial microcirculation perfusion after PCI, and contribute to better postoperative cardiac function, supporting previous findings [21].

Previous studies have shown that the incidence of MACEs in elderly patients with AMI after PCI remains high, and it is closely associated with preoperative inflammatory status, lipid level, and plaque stability. High inflammatory burden and unstable atherosclerotic plaque may contribute to postoperative complications, including stent thrombosis, recurrent myocardial infarction, and target vessel revascularization, thereby adversely affecting patient prognosis and quality of life [22, 23]. Compared to a previous meta-analysis that reported a relative reduction in MACE risk with high-dose atorvastatin loading therapy in ACS patients, our study demonstrated a comparable reduction in MACE risk among elderly patients with AMI, suggesting that elderly patients may also benefit from loading-dose therapy. This may be partially explained by the higher baseline inflammatory burden and more complex coronary lesions typically seen in elderly patients, which may provide greater potential for therapeutic improvement after intensive statin therapy.

The results of this study showed that the incidence of MACEs within 3 months after PCI was 12.7% in group A, significantly lower than 28.1% in group B. Moreover, group A showed shorter hospitalization days and a lower re-hospitalization rate compared to group B. These findings suggest that preoperative loading-dose atorvastatin may reduce short-term MACE risk, shorten hospitalization time, and decrease re-hospitalization rates in elderly patients with AMI undergoing PCI, indicating clinical benefit of this treatment strategy [24]. The possible reasons are as follows: the occurrence of MACE is closely associated with plaque stability and residual inflammatory burden after PCI. Patients in group B exhibited relatively higher levels of LDL-C and CRP, which may contribute to an increased risk of adverse cardiovascular events. In contrast, patients in group A showed greater reductions in LDL-C, CRP, and PCT levels, which may contribute to coronary plaque stabilization, attenuation of inflammatory responses after PCI, and reduction of restenosis and stent thrombosis risk. Meanwhile, the greater improvement in cardiac function indices, including LVEF and cardiac output, may have contributed to a reduced risk of heart failure-related rehospitalization, which was consistent with previous findings [25]. In addition, the shorter hospitalization duration and lower rehospitalization rate observed in group A further supported the potential clinical value of this therapeutic regimen from the perspective of health economics.

Quality of life is an important endpoint for evaluating the comprehensive clinical efficacy of patients with coronary heart disease after PCI. SAQ comprehensively evaluates patients' quality of life from five dimensions, including physical limitation, angina pectoris stability, angina frequency, treatment satisfaction, and disease perception [26]. In this study, group A showed significantly higher SAQ scores in all domains than those of group B, suggesting that preprocedural loading-dose atorvastatin therapy is associated with greater improvement. Compared to a previous quality-of-life substudy that demonstrated improved SAQ scores following intensive medical therapy, our study observed greater improvements in the physical activity limitation and angina stability domains among patients receiving loading-dose atorvastatin. This may be related to the possible synergistic effects of rapid plaque stabilization and attenuation of inflammation during the perioperative period [27]. Several factors may contribute to these findings. Quality of life after PCI is affected by multiple factors, including angina pectoris symptom control, cardiac function, psychological status, and treatment compliance. The improvements in angina stability and frequency in group B were relatively limited, which may be a result of persistent inflammatory activity and residual lipid-related risk. Some patients may continue to experience symptoms such as chest tightness and fatigue during activity,

which limits their physical function and social participation. Through preoperative intensive lipid-lowering and anti-inflammatory treatment, patients in group A achieved better symptom control and cardiac functional recovery after PCI, which may reduce activity limitation caused by angina. Meanwhile, the lower MACE incidence and rehospitalization rate in group A may have reduced disease burden and psychological stress, thereby improving treatment satisfaction and disease perception and contributing to better SAQ scores. These findings are consistent with previous reports on the beneficial effects of atorvastatin therapy on quality of life in patients with coronary heart disease [27].

Treatment safety is an important consideration when selecting clinical treatment options. In particular, elderly patients with AMI are more likely to have impaired liver and kidney function multiple comorbidities, and complex medication regimens, which increase the risk of developing drug-related adverse reactions [28]. Compared to a previous study reporting a notable incidence of liver enzyme elevation among elderly patients receiving high-dose atorvastatin, our study observed a relatively lower incidence of adverse reactions, which may be related to the careful patient selection, dose management, and monitoring of hepatic and renal function in our center [9]. In this study, no serious adverse drug reactions occurred in either group during the treatment period. The most common adverse reactions included mild gastrointestinal discomfort and transient liver function abnormalities, and there was no significant difference in the incidence of adverse reactions between the two groups, suggesting that preoperative loading-dose atorvastatin (40-80 mg) and conventional-dose treatment have comparable safety profiles in elderly patients with AMI undergoing PCI, which is consistent with previous reports [29].

Several limitations should be acknowledged. First, this was a single-center retrospective study, and inherent selection and information bias cannot be completely excluded. Although multivariate regression was performed to adjust for potential confounders, residual confounding from unmeasured variables (e.g., lifestyle, adherence, socioeconomic status) may still exist. Second, the relatively limited sample size (n=128) limited the statistical power, and the control group consisted of patients receiving either conventional-dose atorvastatin (20 mg) or no loading-dose therapy, which may have introduced heterogeneity. Third, the short follow-up (3 months) precluded assessment of long-term clinical outcomes, including late-onset cardiovascular effects and delayed adverse effects. Fourth, plaque imaging (IVUS/OCT) and neuroendocrine markers were not examined, limiting mechanistic insights into plaque stabilization and the pleiotropic effects of statins. We relied solely on peripheral lipid profiles and inflammatory markers to indirectly reflect plaque stability, without providing *in situ* pathologic imaging evidence of vascular lesions. Therefore, plaque characteristics, including plaque burden, fibrous cap thickness, lipid core size, and other vulnerability features, could not be directly assessed. This represents a significant gap in direct observational data of the target lesions in vivo. Fifth, the broad loading-dose range (40-80 mg) may have introduced heterogeneity within the treatment group. More importantly, dose-stratified analyses comparing the efficacy of 40 mg versus 80 mg atorvastatin were not performed, making it difficult to determine the optimal loading-dose intensity. Such analyses were not feasible because of the limited sample size in each subgroup, which would have resulted in insufficient statistical power. Sixth, the study population consisted exclusively of elderly population from a single-center Chinese medical center, which may limit the generalizability of the findings to other populations with different genetic backgrounds, dietary habits, or healthcare systems. Seventh, multiple between-group and within-group comparisons were conducted without adjustment for multiplicity (e.g., Bonferroni correction), potentially increasing the risk of type I errors (false-positive findings). Given the exploratory nature of this study, no correction was applied, and this limitation should be considered when interpreting statistically significant findings. Eighth, the primary objective of this study was to compare clinical outcomes (MACE incidence) between loading-dose and conventional/no loading-dose atorvastatin regimens. The quantitative relationships between changes in inflammatory markers/lipid levels and subsequent MACE incidence were not further explored through correlation or regression analyses. Future studies should investigate the

dose-response associations between biomarker changes and clinical outcomes to better clarify the relationship between biological effects and prognosis. Ninth, this was a clinical observational study without *in vitro* experiments or *in vivo* animal model studies. Therefore, the molecular mechanisms underlying the potential effects of loading-dose atorvastatin, including regulation of inflammatory pathway and plaque stabilization, could not be directly elucidated. The lack of a complete translational evidence chain combining basic experimental validation with clinical observations represented a significant limitation. Future prospective multi-center studies with larger sample sizes, standardized dosing, dose-stratified analyses, extended follow-up periods, comprehensive imaging assessments (including IVUS/OCT), appropriate statistical correction for multiple comparisons, and further mechanistic investigations are warranted to validate these findings and further clarify the effects of loading-dose atorvastatin therapy in elderly patients with AMI undergoing PCI.

Conclusion

Preoperative loading-dose (40-80 mg) atorvastatin and conventional-dose treatment (20 mg or no loading dose) effectively improved cardiac function, blood lipid profiles, and inflammatory status in elderly patients with AMI after PCI. In addition, these strategies were also associated with reduced MACE incidence, shorter hospital stays, lower re-hospitalization rates, and improved SAQ-based quality of life, while demonstrating favorable safety profiles. Compared to conventional-dose therapy, loading-dose atorvastatin was associated with greater improvements in LVEF, greater reductions in LDL-C and CRP levels, a lower risk of MACEs, and enhanced quality of life. Therefore, loading-dose atorvastatin may represent a promising perioperative treatment strategy for elderly patients with AMI undergoing PCI.

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Disclosure of conflict of interest

None.

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