European Review for Medical and Pharmacological Sciences 2017; 21: 4457-4463

Study on the effect of different doses of rosuvastatin on ventricular remodeling in patients with acute coronary syndrome after emergency percutaneous coronary intervention

J. GUO, W.-H. ZHANG, Q. ZHAO, J.-S. WO, S.-L. CAI

Department of Cardiology, The Affiliated Hospital of Qingdao University, Qingdao, Shandong Province, China

Abstract. – OBJECTIVE: The objective of the present study was to observe the effects of different doses of rosuvastatin on cardiac protection in patients with acute coronary syndrome (ACS) after stent implantation.

PATIENTS AND METHODS: A total of 137 patients with ACS were selected from March 2014 to January 2015 and randomly divided into: 1. The conventional treatment group: 45 patients were treated with conventional drugs such as aspirin, clopidogrel, nitrates, and a β-blocker; 2. The conventional rosuvastatin dose group: 45 patients received 10 mg/d rosuvastatin before sleep in addition to routine therapy; 3. The large rosuvastatin dose group: 47 patients received 20 mg/d rosuvastatin before sleep in addition to routine therapy. The course of treatment was 12 weeks. At 1, 6, and 12 week, ultrasound echocardiography, electrocardiogram (ECG), high-sensitivity C-reactive protein (hs-CRP), and pro-brain natriuretic peptide (pro-BNP) levels were tested to evaluate the therapeutic effects. The ultrasonic imaging criteria included left ventricular end diastolic diameter (LVEDD), left ventricular end systolic diameter (LVESD), left ventricular end diastolic volume (LVEDV), left ventricular end systolic volume (LVESV), and left ventricular ejection fraction (LVEF).

RESULTS: After 1 week, hs-CRP, pro-BNP, and echocardiography of the patients in the three groups showed no significant differences ($p>0.05$); after 6 and 12 weeks, the levels of hs-CRP, MMP-9, and pro-BNP in the large rosuvastatin dose group were significantly lower than in the conventional rosuvastatin dose group and conventional treatment group ($p<0.05$), and ultrasonic indexes changed significantly after 12 weeks ($p<0.05$). There were no significant differences in ultrasonic indexes after 6 weeks ($p>0.05$). No thrombosis or restenosis occurred during the follow-up period in each group.

CONCLUSIONS: Three months after emergency percutaneous coronary intervention, a high-dose of rosuvastatin can delay ventricular remodeling, effectively inhibit malignant remodeling of the heart, improve left ventricular systolic function, reduce the prevalence of adverse events, and significantly improve the long-term prognosis.

Key Words:
Acute coronary syndrome, Emergency percutaneous coronary intervention (PCI), Rosuvastatin, Ventricular remodeling.

Introduction

Recent studies have shown that the main pathological features of acute coronary syndrome (ACS) are: 1. Complete or incomplete rupture of atherosclerotic plaques; 2. Complete or incomplete thromboembolic obstruction; 3. Myocardial ischemia or acute insufficiency. Clinically, ACS is classified as acute myocardial infarction (AMI) or unstable angina (UA). AMI can be divided into ST segment elevation acute myocardial infarction (STEMI) and non-ST segment elevation acute myocardial infarction (NSTEMI), according to the elevation of the electrocardiographic ST segment of patients. Among them, NSTEMI and UA are also collectively referred to as non-ST elevation acute coronary syndrome (NSTE-ACS). For STEMI patients, early and rapid emergency thrombolysis and/or percutaneous coronary intervention (PCI) of the coronary artery and downstream coronary artery infarctions can significantly reduce mortality and improve prognosis. However, because of local myocardial necrosis caused by myocardial ischemia, and

Corresponding Author: Wenzhong Zhang, MD; e-mail: zhang_wenzhong1@163.com

hypoxia caused by acute coronary artery syndrome, heart enlargement and left ventricular systolic dysfunction often occur after emergency PCI, which affects the prognosis of patients. In the present study, oral administration of large doses (0.4 mg/kg/d) of rosuvastatin, a selective inhibitor of 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA), was adopted to treat ACS patients after PCI, and the effects were compared with patients who received a conventional dose of rosuvastatin and those who did not receive rosuvastatin treatment. The results are as follows.

Patients and Methods

Patients

A total of 137 patients with ACS were selected for inclusion in the study between March 2014 and January 2015. There were 78 males and 59 females, aged from 28-87 years old, with average age of 61 ± 4.7 years. Patients were divided into the conventional therapy group, the conventional rosuvastatin dose group, and the large rosuvastatin dose group according to numbers randomly generated from a computer. Age, sex, operative time, time from admission to surgery, intraoperative blood loss, days of hospitalization, and other basic parameters of the three groups of patients were not significantly different (p>0.05) (Table I).

Inclusion and Exclusion Criteria

The inclusion criteria were as follows: (1) Patients were in accordance with the ACS diagnostic criteria formulated by the American College of Cardiology (ACC)/American Heart Association (AHA). (2) All enrolled patients had typical and varying degrees of primordial pain or discomfort, and were diagnosed with ACS by electrocardiogram, cardiac index assessment, and measurement of troponin levels; (3)Patients underwent emergency PCI or emergency intravenous thrombolytic therapy; (4) Patients and their families agreed to the relevant laboratory examinations[1]; (5) Patients did not have other cardiovascular diseases.

All enrolled patients signed the informed consent and the study was approved by the Ethics Committee of The Affiliated Hospital of Qingdao University.

The exclusion criteria were as follows: (1) Patients who had taken lipid-lowering drugs within 2 months before admission; (2) Patients who were taking immunosuppressant drugs; (3) Patients with acute or chronic bacterial and/or viral infections; (4) Patients with autoimmune diseases; (5) Patients with connective tissue diseases; (6) Patients with malignant tumors; (7) Patients with disorders of the liver or kidney; (8) Patients with chronic myopathies; (9) Patients who were hypersensitive to rosuvastatin; (10) Patients with peripheral vascular diseases, chronic heart failure, thyroid disease, major trauma which occurred within half a year before admission to the study, and history of surgery; (11) Patients with myocardial infarction that occurred within 6 months before admission to the study, patients who underwent percutaneous transluminal coronary angioplasty, patients with history of coronary artery bypass graft, and patients taking corticosteroids or other immunomodulatory drugs; (12) Patients and families who were incompatible with the study and patients with a history of mental illness[2].

Therapeutic Regimen

After enrollment, the 45 patients in the conventional therapy group (the control group) were given aspirin (Yangze Pharma Co., Ltd., Taizhou, China), clopidogrel (Yangze Pharma

Table I. Basic parameters of 137 ACS patients who underwent emergency PCI.

Group	Sex		Age	Operative time (t/min) loss (V/ml)	Intraoperative blood	Hospitalization days (t/d)	Time from admission to surgery (t/min)
	Male	Female					
The control group	22	23	61.8±4.6	45.2±12.6	56.4±4.7	6.8±1.3	10.5±3.6
The large rosuvastatin dose group	20	27	57.8±6.4	50.3±11.9	58.3±3.9	7.4±1.2	12.4±2.1
The conventional rosuvastatin dose group	27	18	62.3±3.7	46.8±10.7	48.1±4.3	7.1±0.8	10.8±1.6
t	0.21	0.37	0.85	0.14	0.22	0.11	0.24
p	>0.05	>0.05	>0.05	>0.05	>0.05	>0.05	>0.05

Co., Ltd., Taizhou, China), nitrates (Yangze Pharma Co., Ltd., Taizhou, China), a β-blocker (Yangze Pharma Co., Ltd., Taizhou, China), and other conventional drugs without statins. The 45 patients in the conventional rosuvastatin dose group were given oral rosuvastatin (Crestor, AstraZeneca Pharmaceutical Co., Ltd., Berlin, Germany) at a dose of 20 mg per day before bedtime in addition to routine therapy. In the large rosuvastatin dose group, the 47 patients were given oral rosuvastatin at a dose of 40 mg per day before bedtime in addition to routine therapy[1]. The course of treatment was 12 weeks, during which dynamic changes of indicators of liver, renal function, and creatine kinase were closely monitored.

Observational Indexes

Heart rate: the heart rate of patients in each group at 1, 6, and 12 week after PCI was recorded using a Yuyue YX301 finger pulse oximeter (Yuyue Company, Shanghai, China). Patients were asked to remain in a calm and quiet state. Each count lasted for 1 min and after three measurements the average heart rate was recorded.

Echocardiographic indexes: on the first postoperative day and at the end of postoperative week 1, 6, and 12, all patients were analyzed with a Philips IE33 (with probe frequency of 2.5 MHz) color Doppler ultrasonic diagnostic apparatus (Philips, Eindhoven, The Netherlands) to measure left ventricular end diastolic diameter (LVEDD), left ventricular end systolic diameter (LVESD), left ventricular end diastolic volume (LVEDV), left ventricular end systolic volume (LVESV), and left ventricular ejection fraction (LVEF).

Serological measurements: on the first day and at 1, 6, and 12 week after surgery, 4-6 ml venous blood was drawn from all patients to determine the levels of biochemical indexes including high-sensitivity C-reactive protein (hs-CRP)[3,4] brain natriuretic peptide (pro-BNP)[3,5], and matrix metalloproteinase 9 (MMP-9)[3,6]. Patients fasted for 6-10 h before blood drawing. At the end of postoperative week 1, 6, and 12, 4-6 ml fasting blood from the ulnar vein was collected from all patients, among which 2 ml was used to measure the levels of plasma pro-BNP by rapid fluorescence immunoassay (Triage tester), and the remaining 2-4 ml was centrifuged for 10 min at 402.48 × g. Serum was extracted and stored at -80°C. Enzyme-linked immunosorbent assay (ELISA) was used to measure the levels of hs-CRP, pro-BNP, and MMP-9.

Statistical Analysis

SPSS19.0 software (Version X; IBM, Armonk, NY, USA) was used for data analysis. Qualitative data were analyzed with a χ^2-test. Data that did not meet the conditions on the 2 × 2 table adopted Fisher's exact probability test. The comparisons within qualitative data were tested by analysis of variance (ANOVA), and $p<0.05$ was taken as statistically significant.

Results

Comparison of Heart Rate, Echocardiography, and Biochemical Indicators

One week after PCI, there were no significant changes in the heart rate of patients in the control group, the conventional rosuvastatin dose group, and the large rosuvastatin dose group ($p>0.05$). LVEDD, LVESD, LVEDV, and LVESD of the three groups were all lower compared with before treatment, while LVEF increased compared with before treatment, although there were no significant differences between the three groups ($p>0.05$). 6 weeks after PCI, the heart rate changes of patients in the conventional rosuvastatin dose group and large rosuvastatin dose group were not significantly different ($p>0.05$). LVEDD, LVESD, LVEDV, and LVESD in the three groups were lower than those before treatment, LVEF increased compared with that before treatment, and the differences were not statistically significant ($p>0.05$). The levels of hs-CRP, pro-BNP, and MMP-9 of the three groups decreased significantly compared with before treatment, and the curative effects of the large rosuvastatin dose group were more significant ($p<0.05$). 12 weeks after PCI, there were no significant changes in the heart rates of patients in the control group, the conventional rosuvastatin dose group, and the large rosuvastatin dose group ($p>0.05$). The levels of hs-CRP, pro-BNP, and MMP-9 of the three groups decreased significantly compared with before treatment, and the curative effect in the large rosuvastatin dose group was more pronounced ($p<0.05$) (Table II).

Safety Comparison

Comparing safety in the conventional rosuvastatin dose group and the large rosuvastatin dose group, the liver and kidney functions of both groups were not significantly different ($p>0.05$). Therefore, treatment with the large dose of rosu-

Table I. Basic parameters of 137 ACS patients who underwent emergency PCI.

Group	Sex		Age	Operative time (t/min) loss (V/ml)	Intraoperative blood	Hospitalization days (t/d)	Time from admission to surgery (t/min)
	Male	Female					
The control group	22	23	61.8±4.6	45.2±12.6	56.4±4.7	6.8±1.3	10.5±3.6
The large rosuvastatin dose group	20	27	57.8±6.4	50.3±11.9	58.3±3.9	7.4±1.2	12.4±2.1
The conventional rosuvastatin dose group	27	18	62.3±3.7	46.8±10.7	48.1±4.3	7.1±0.8	10.8±1.6
t	0.21	0.37	0.85	0.14	0.22	0.11	0.24
p	>0.05	>0.05	>0.05	>0.05	>0.05	>0.05	>0.05

vastatin did not affect liver and kidney functions of patients, and was relatively safe (Table III).

Therapeutic Effects

During the treatment, except for a few cases of dizziness and palpitations, the patients in the three groups did not show adverse drug reactions or liver functional damage. After 1 week of treatment, hs-CRP and pro-BNP levels, and echocardiographic indexes of the patients in the three groups were compared, and the differences were statistically significant ($p<0.05$). After 6 weeks of treatment, hs-CRP and pro-BNP levels of patients in the large rosuvastatin dose group were significantly lower than those of the conventional rosuvastatin dose group ($p<0.05$) and the conventional therapy group ($p<0.05$); however, there were no significant differences in the changes of echocardiographic indexes ($p>0.05$). After 12 weeks of treatment, hs-CRP, pro-BNP, and cardiac troponin I (cTNI) levels of patients in the large rosuvastatin dose group were significantly lower than those of the conventional rosuvastatin dose group ($p<0.05$) and conventional therapy group ($p<0.05$), and there were significant differences in the changes of echocardiographic indexes ($p<0.05$). There were occurrences of restenosis, death, nonfatal myocardial infarction, and cardiogenic shock after patients in the three groups were followed-up for 1 year (Table IV).

Discussion

According to the latest guidelines[1-3], there should be a persistent lipid-lowering treatment period following PCI. Specifically: 1. ACS patients should be given cholesterol-lowering statins after PCI, maintaining low-density lipoprotein cholesterol (LDL-C) < 2.60 mmol/l (100 mg/dl) or even < 2.08 mmol/l (80 mg/dl); 2. Patients who also have diabetes should maintain LDL-C < 80 mg/dl, and even after reaching target lipid levels, they should not cease taking medicine and the dose should not be blindly reduced; 3. When LDL-C levels are not within acceptable range, cholesterol absorption inhibitors or other lipid-lowering drugs can be combined; 4. When LDL-C reaches standard levels and the levels of triglycerides continue to be over 2.26 mmol/l, fibrate or nicotinic drugs can be combined; 5. If triglycerides are > 1.70 mmol/l, and they remain high after 3 months of improving lifestyle and diet, fibrate or nicotinic lipid-lowering drugs should be used. Among lipid-lowering drugs, rosuvastatin[4-6] is a selective and competitive HMG-CoA reductase inhibitor. The main indications for use are patients with primary or mixed dyslipidemia whose lipids cannot be controlled even with dietary changes, exercise therapy, and reasonable weight control. The main pharmacological actions include 1. Inhibition of the ability of HMG-CoA to shift to mevalonic acid[7]; 2. Increases in the number of liver LDL cell surface receptors[8]; 3. The promotion of LDL absorption and catabolism, inhibition of very-low-density lipoprotein (VLDL) synthesis in the liver, and reduction of the total number of VLDL and LDL particles[9].

In our clinical practice, it was found that large doses of statins can improve the prognosis of patients after emergency PCI. The 137 enrolled patients with ACS were randomly divided into three groups, the conventional therapy group (the control group), the conventional rosuvastatin dose group, and the large rosuvastatin group. Postoperative weeks 1, 6, and 12 were selected as the time points to observe echocardiography, ECG, and blood biochemical indexes of patients, through which the dosage and curative effects of rosuvastatin after PCI were evaluated. It was concluded that 1 week after

Table II. Comparison of heart rate, echocardiography, and blood biochemical indexes of patients in the three groups before treatment ($\bar{x}$±s).

Index	Group	Number of cases	Before PCI	One week after PCI	Six weeks after PCI	Twelve weeks after PCI
HR (bpm)	Control group	45	134.1±6.5	95.6±4.5	78.6±2.4	77.5±3.2
	High-rosuvastatin dose group	47	126.3±10.3	82.4±8.2	75.3±1.8	78.3±2.5
	Conventional rosuvastatin dose group	45	131.7±4.6	89.3±3.8	77.2±3.5	76.4±1.3
	F	-	0.62	0.73	0.61	0.57
	p		>0.05	>0.05	>0.05	>0.05
LVEDD (mm)	Control group	45	59.3±5.3	58.2±2.0	56.3±5.1	54.7±4.8
	High-rosuvastatin dose group	47	58.7±3.5	59.0±1.8	51.2±2.6	48.6±3.7
	Conventional rosuvastatin dose group	45	58.2±2.8	58.6±2.5	54.2±1.5	51.3±5.5
	F	-	0.78	0.52	0.48	3.61
	p		>0.05	>0.05	>0.05	<0.05
LVESD (mm)	Control group	45	42.1±4.7	37.0±1.3	37.2±2.6	36.2±4.5
	High-rosuvastatin dose group	47	41.8±6.2	37.6±1.8	35.5±2.8	31.7±2.2
	Conventional rosuvastatin dose group	45	41.6±5.7	38.5±2.8	36.4±3.3	35.1±2.8
	F	-	0.72	0.46	0.82	2.7
	p		>0.05	>0.05	>0.05	<0.05
LVEDV (ml)	Control group	45	108.0±5.9	107.3±5.4	107.3±6.5	108.4±5.6
	High-rosuvastatin dose group	47	109.5±5.2	106.7±5.5	102.4±4.7	89.7±10.2
	Conventional rosuvastatin dose group	45	110.5±3.8	105.2±2.3	104.3±3.2	102.1±5.1
	F	-	0.65	0.56	0.76	8.6
	p		>0.05	>0.05	>0.05	<0.05
LVEF (%)	Control group	45	45.9±4.2	49.2±5.5	52.2±5.8	53.2±3.7
	High rosuvastatin dose group	47	42.4±4.6	52.3±4.8	58.5±2.6	61.2±3.5
	Conventional rosuvastatin dose group	45	46.3±8.5	51.4±4.6	54.3±6.4	56.4±4.3
	F	-	0.63	0.47	0.73	14.5
	p		>0.05	>0.05	>0.05	<0.05
Hs-CRP (μg/l)	Control group	45	16.8±6.8	10.8±6.4	9.25±4.3	9.02±2.5
	High-rosuvastatin dose group	47	15.2±7.2	4.7±3.8	3.5±2.3	2.37±3.6
	Conventional rosuvastatin dose group	45	13.6±5.3	6.2±3.2	5.8±1.6	4.8±3.2
	F	-	0.37	6.3	8.6	10.7
	p		>0.05	<0.05	<0.05	<0.05
Pro-BNP (ng/d)	Control group	45	487.8±21.8	257.2±16.8	198.7±23.1	117.8±10.2
	High-rosuvastatin dose group	47	597.4±64.5	121.7±31.5	98.2±16.5	65.4±19.2
	Conventional rosuvastatin dose group	45	512.6±85.3	209.6±17.8	106.9±10.5	97.6±10.8
	F	-	0.76	7.3	10.6	9.8
	p		>0.05	<0.05	<0.05	<0.05
MMP-9 (μg/l)	Control group	45	197.6±37.8	173.8±21.5	172.7±31.2	168.4±12.6
	High-rosuvastatin dose group	47	202.5±16.5	112.7±16.5	98.5±12.2	90.4±21.4
	Conventional rosuvastatin dose group	45	211.4±25.6	145.2±18.1	131.7±24.7	112.4±15.6
	F	-	0.82	6.4	9.6	12.8
	p		>0.05	<0.05	<0.05	<0.05

Notes: PCI, percutaneous coronary intervention; HR, heart rate; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter; LVEDV, left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; hs-CRP, high-sensitivity C-reactive protein; pro-BNP, pro-brain natriuretic peptide; MMP-9, matrix metalloproteinase 9.

Table III. Comparisons of liver and renal function indexes between the three groups ($\bar{x}$±s).

Program	Group	Number of cases	Before surgery	One week after PCI	Six weeks	F	*p*
Scr (μmol/l)	Control group	45	239.4±38.6	277.3±32.4	236.5±50.7	0.89	0.32
	High rosuvastatin dose group	47	218.3±21.8	223.4±19.5	204.3±10.4	0.77	0.35
	Conventional rosuvastatin dose group	45	197.4±18.4	193.6±21.5	208.3±22.4	0.69	0.49
	F	-	0.66	0.25	0.51	-	-
	p		0.33	0.72	0.45	-	-
BUN (mmol/l)	Control group	45	11.4±3.4	12.3±3.4	12.4±2.5	0.65	0.31
	High rosuvastatin dose group	47	10.7±2.4	12.6±2.2	11.9±2.8	0.33	0.41
	Conventional rosuvastatin dose group	45	10.5±2.4	11.7±1.4	13.2±3.3	0.28	0.54
	F	-	0.41	0.31	0.44	-	-
	p		0.32	0.45	0.55	-	-
TBIL (μmol/l)	Control group	45	56.4±21.8	57.8±23.5	58.4±12.7	0.48	0.31
	High rosuvastatin dose group	47	55.8±12.7	52.3±10.9	52.4±9.9	0.44	0.24
	Conventional rosuvastatin dose group	45	52.3±8.9	52.4±9.8	51.4±7.8	0.55	0.66
	F	-	0.24	0.43	0.55	-	-
	p		0.35	0.45	0.24	-	-
ALT (U/l)	Control group	45	51.4±21.5	50.4±13.3	47.3±12.8	0.28	0.51
	High rosuvastatin dose group	47	45.4±12.9	47.2±19.2	50.5±12.3	0.33	0.78
	Conventional rosuvastatin dose group	45	49.2±10.7	48.2±10.9	43.3±9.6	0.44	0.78
	F	-	0.39	0.87	0.42	-	-
	p		0.88	0.74	0.38	-	-

Notes: PCI, percutaneous coronary intervention; Scr, serum creatinine; BUN, blood urea nitrogen; TBIL, total bilirubin in serum; ALT, alanine aminotransferase.

Table IV. Comparison of postoperative follow-up data of patients in the three groups who underwent PCI [n(%)].

Programs	Follow-up number	The control group (n=45)	The conventional rosuvastatin dose group (n=45)	The large rosuvastatin dose group (n=47)	F	*p*
Restenosis occurred after 1 year follow-up	137	18 (0.40)	16 (0.36)	5 (0.11)	3.21	<0.05
Death occurred at the end of follow-up	137	6 (0.13)	2 (0.04)	1 (0.02)	2.25	<0.05
Non-fatal myocardial infarction occurred at the end of follow-up.	137	9 (0.20)	6 (0.13)	2 (0.04)	3.78	<0.05
Non-fatal cardiac shock occurred at the end of follow-up	137	1 (0.02)	0 (0.00)	0 (0.00)	7.82	>0.05
Target vessel revascularization at the end of the follow-up	137	11 (0.24)	21 (0.47)	39 (0.83)	0.82	<0.05

PCI, hs-CRP, pro-BNP, and MMP9 levels in fasting venous blood, and echocardiographic indexes of patients in the three groups were not statistically significant (p>0.05). After 6 weeks, hs-CRP and pro-BNP levels of patients in the large rosuvastatin dose group were significantly lower than those of the conventional rosuvastatin dose group (p<0.05) and conventional therapy group (p<0.05). Additionally, echocardiographic indexes were not significantly different (p>0.05). After 12 weeks of treatment, hs-CRP and pro-BNP levels of patients in the large rosuvastatin dose group were significantly

lower than those of the conventional rosuvastatin dose group ($p<0.05$) and conventional therapy group ($p<0.05$), and changes in echocardiographic indexes were significantly different ($p<0.05$). It was shown that oral administration of rosuvastatin following emergency PCI had a cardio-protective and dose-dependent effect. After 6 weeks, although the levels of hs-CRP, pro-BNP, and MMP9 were significantly improved, echocardiographic indexes of the patients were equal to or worse than those before surgery, which may have been attributed to the fact that cardiac remodeling caused by ACS is a chronic process during recovery. Regarding ischemic tissue, although PCI recanalization improves myocardial ischemia and hypoxia, the recovery of myocardial functions, and improvements in ventricular morphology require time. After the 1-year follow-up for emergency PCI, the occurrence of restenosis, death, non-fatal myocardial infarction, cardiogenic shock, and other adverse events in patients of the large rosuvastatin dose group were significantly lower than those of the conventional therapy group (control group) and conventional rosuvastatin dose group. It was shown that rosuvastatin can indirectly produce a significant effect on the long-term prognosis of patients after PCI by reducing blood fat and improving blood glucose. However, further follow-up observation is needed for tracking the long-term prognosis of patients.

Conclusions

For patients with ACS, taking a large dose of rosuvastatin before bedtime after emergency PCI, can delay the process of atherosclerosis, effectively inhibit malignant cardiac remodeling, improve left ventricular systolic function, reduce the occurrence of adverse events, and improve long-term prognosis. Its clinical effect is, therefore, significant.

Conflict of interest

The authors declare no conflicts of interest.

References

1) Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD. Third universal definition of myocardial infarction. Glob Heart 2012; 7: 275-295.
2) Braunwald E, Antman EM, Beasley JW, Califf RM, Cheitlin MD, Hochman JS, Jones RH, Kereiakes D, Kupersmith J, Levin TN, Pepine CJ, Schaeffer JW, Smith ER, Steward DE, Theroux P, Alpert JS, Eagle KA, Faxon DP, Fuster V, Gardner TJ, Gregoratos G, Russell RO, Smith SJ. ACC/AHA guidelines for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients with Unstable Angina). J Am Coll Cardiol 2000; 36: 970-1062.
3) Zamani P, Schwartz GG, Olsson AG, Rifai N, Bao W, Libby P, Ganz P, Kinlay S. Inflammatory biomarkers, death, and recurrent nonfatal coronary events after an acute coronary syndrome in the MIRACL study. J Am Heart Assoc 2013; 2: e3103.
4) Oduncu V, Tanalp AC, Erkol A, Sirma D, Dundar C, Akgun T, Turkyilmaz E, Kilicgedik A, Gozubuyuk G, Tigen K, Izgi A, Kirma C. Impact of chronic pre-treatment of statins on the level of systemic inflammation and myocardial perfusion in patients undergoing primary angioplasty. Am J Cardiol 2011; 107: 179-185.
5) Zu H, Li Q, Huang P, Wang X. Therapeutic value of blood purification and prognostic utilities of early serum procalcitonin, C reactive protein, and brain natriuretic peptide levels in severely burned patients with sepsis. Cell Biochem Biophys 2015; 72: 259-263.
6) Fu FY, Chen BY, Chen LL, Zhang FL, Luo YK, Jun F. Improvement of the survival and therapeutic effects of implanted mesenchymal stem cells in a rat model of coronary microembolization by rosuvastatin treatment. Eur Rev Med Pharmacol Sci 2016; 20: 2368-2381.
7) Gotoh K, Masaki T, Chiba S, Ando H, Fujiwara K, Shimasaki T, Tawara Y, Toyooka I, Shiraishi K, Mitsutomi K, Anai M, Itateyama E, Hiraoka J, Aoki K, Fukunaga N, Nawata T, Kakuma T. Effects of hydrophilic statins on renal tubular lipid accumulation in diet-induced obese mice. Obes Res Clin Pract 2013; 7: e342-e352.
8) Jones PH, Davidson MH, Stein EA, Bays HE, McKenney JM, Miller E, Cain VA, Blasetto JW. Comparison of the efficacy and safety of rosuvastatin versus atorvastatin, simvastatin, and pravastatin across doses (STELLAR* Trial). Am J Cardiol 2003; 92: 152-160.
9) Kelle I, Akkoc H, Uyar E, Erdinc M, Evliyaoglu O, Saribas S, Tunik S, Ozogul C. The combined effect of rosuvastatin and ischemic pre- or post-conditioning on myocardial ischemia-reperfusion injury in rat heart. Eur Rev Med Pharmacol Sci 2015; 19: 2468-2476.