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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Chronic statin treatment is a predictor of pre-interventional infarct-related artery patency in patients with ST elevation myocardial infarction treated with percutaneous coronary intervention

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Abstract

Background: Beyond lipid-lowering effects, early statin treatment has beneficial effects on prognosis after acute coronary syndrome. Infarct-related artery (IRA) patency before percutaneous coronary intervention (PCI) is known to be a strong predictor of improved clinical outcome.

Aim: We aimed to investigate the effects of chronic statin treatment before admission on IRA patency after myocardial infarction.

Methods: In this study, 938 ST elevation myocardial infarction (STEMI) patients admitted to the hospital within the first 12 h of symptom onset were prospectively enrolled (male, n = 682; female, n = 256; mean age 58.6 ± 12.4 years). All patients underwent emergent primary PCI. Patients were divided into two groups based upon angiographic IRA patency. Impaired IRA patency was defined as Thrombolysis In Myocardial Infarction (TIMI) grade 0 and 1 flow (non-patent IRA group). Angiographic IRA patency was defined as TIMI 2 and 3 flow (patent IRA group).

Results: Previous statin usage was more frequent in the patent IRA group (n = 138; 71.9%), than in the non-patent IRA group (n = 110; 14.7%; p < 0.001). Pre-PCI IRA patency was independently associated with body mass index (odds ratio [OR] = 1.087, 95% confidence interval [CI] 1.005–1.176, p < 0.001), previous chronic statin use (OR 0.065, 95% CI 0.043–0.098, p = 0.039), ejection fraction (OR 1.041, 95% CI 1.018–1.064, p < 0.001), and SYNTAX score (OR 0.927, 95% CI 0.899–0.957, p < 0.001) in multivariate logistic regression analysis.

Conclusions: Chronic pre-treatment with statins is a significant predictor of the IRA patency in patients with STEMI.

Key words: statin, myocardial infarction, TIMI flow, infarct-related artery patency

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INTRODUCTION

Early restoration of coronary flow in infarct-related artery (IRA) improves ventricular performance and decreases mortality in patients with ST elevation myocardial infarction (STEMI) [1]. The Thrombolysis In Myocardial Infarction (TIMI) flow grade score is a scoring method for assessing coronary blood flow [2]. Basal TIMI flow in IRA is crucial for patients with STEMI undergoing primary percutaneous coronary intervention (PCI). Patients with a patent IRA have lower rates of heart failure and cardiogenic shock, improved early and late ejection fraction, and reduced short- and long-term mortality [1]. Moreover, pre-PCI patency of IRA in patients with STEMI is a major determinant of post-PCI TIMI 3 flow, which is associated with improved clinical outcome [3].

Previous large clinical trials have demonstrated that statins reduce mortality and morbidity associated with cardiovascular disease, especially the incidence of myocardial infarction (MI) [4]. It was assumed that by lowering serum cholesterol levels,

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statins cause regression and/or stabilisation of the atherosclerotic plaque [5]. In addition to their potent effect on serum lipid levels, statins influence several other cellular pathways, including inflammatory, oxidative, and thrombotic processes [6]. Statins have also an effect on endothelial function and nitric oxide production, independent of changes in serum low-density lipoprotein cholesterol (LDL-C) levels [6].

We hypothesised that the above-mentioned "pleiotropic" effects could contribute to pre-PCI IRA patency in patients with STEMI. Furthermore, statins are positively effective on post-PCI IRA patency [7, 8]. However, it is unclear whether previous chronic statin use affects the pre-PCI IRA patency in patients with STEMI. Therefore, we aimed to investigate the effects of chronic statin treatment before admission on the IRA patency after MI.

METHODS

Study population

In this study, 938 STEMI patients within 12 h of symptom onset, in whom primary PCI was performed in our cardiology clinic between June 2016 and February 2017, were prospectively included (male, n = 682; female, n = 256; mean age 58.6 ± 12.4 years). Pretreatment with aspirin, clopidogrel, and heparin was administered at the catheter laboratory. Urgent diagnostic angiography was followed by primary PCI using standard techniques; femoral approach was used. Radial approach was not used because of lack of operator expertise and technical equipment. STEMI was defined as: typical chest pain > 30 min duration with ST elevation > 1 mm in at least two consecutive leads on the electrocardiogram or new onset left bundle branch block. Glycoprotein IIa/IIIb antagonists were used only in case of no reflow phenomenon and high thrombus burden after primary PCI, as suggested by the latest STEMI guidelines.

Those with a recent history of MI, PCI, coronary artery bypass graft, infectious or inflammatory disease, severe liver or renal disease, malignancy, or haematological disorders were excluded from our study. STEMI was the first clinical manifestation of clinically significant cardiovascular disease in our study population. All patients were free from any clinically significant peripheral arterial disease, heart failure, or stroke. Statins were exclusively used for primary prevention. Hyperlipidaemia was the main indication in all patients using statin therapy. Atorvastatin and rosuvastatin were used by the patients for hyperlipidaemia.

Patients who presented later than 12 h after the onset of symptoms were not included. The study was conducted according to the recommendations set forth by the Declaration of Helsinki on Biomedical Research Involving Human Subjects. The Institutional Ethics Committee approved the study protocol, and each participant provided written, informed consent.

Baseline characteristics of patients with STEMI were recorded, such as age, gender, body mass index, smoking at admission, history of hyperlipidaemia, hypertension, diabetes, family history of cardiovascular disease, previous medications (statins, angiotensin converting enzyme [ACE] inhibitors, angiotensin receptor blockers, beta-blockers, oral antidiabetic drugs, acetylsalicylic acid), systolic blood pressure, diastolic blood pressure, and heart rate (bpm). Also, the type of MI, target vessel, and chest pain time were recorded in all patients.

Blood samples and echocardiography

In all patients, antecubital venous blood samples for laboratory analysis were drawn upon admission into the emergency room. Biochemical markers were measured with an automated chemistry analyser.

Transthoracic echocardiography was performed for each patient immediately after primary PCI in a coronary intensive care unit (Vivid 7® GE Medical System, Horten, Norway). Ejection fraction was determined using the Simpson's method, according to the recommendations of the American Society of Echocardiography [9].

Coronary angiography (TIMI flow grade)

All patients underwent selective coronary angiography using the Judkins technique. Primary PCI procedures were performed with standard femoral approach with a 7-French guiding catheter. To achieve maximal dilatation each coronary angiogram was preceded by intracoronary injection of 100 μg nitroglycerine. TIMI flow grade was assessed in consensus by three experienced interventional cardiologists who did not have knowledge of the clinical and laboratory data. Pre-PCI TIMI flow grade was documented for each patient. Patients were divided into two groups based on the TIMI flow grade [2]: the impaired IRA patency group was defined as TIMI grade 0 and 1 flow (non-patent IRA group), and the patent IRA group was defined as TIMI 2 and 3 flow before intervention [10].

Statistical analysis

All analyses were conducted using SPSS 17.0 (SPSS for Windows 11.5, Chicago, IL). Normally distributed continuous variables were expressed as mean ± standard deviation, non-normally distributed variables were expressed as median (minimum–maximum), and categorical variables were expressed as percentages. Comparisons of categorical and continuous variables between the two groups were performed using the χ^2 test and independent samples t-test, respectively. A p-value of < 0.05 was considered significant for univariate analysis. Binary logistic regression analysis was used to determine the independent predictors of IRA patency. Variables with a p value lower than 0.25 in univariate analysis were entered in multivariate analysis.

RESULTS

Univariate analysis

Clinical characteristics, echocardiographic and laboratory findings of patients were shown in Table 1. Patients in the

Table 1. Comparison of baseline characteristics, risk factors, and laboratory findings between the groups

Variables	Patent IRA group (n = 192)	Non-patent IRA group (n = 746)	p
Baseline characteristics:			
Age [years]	58.6 ± 12.5	58.4 ± 11.9	0.842
Gender (male)	129 (67.2%)	553 (74.1%)	**0.035**
BMI [kg/m^2]	26.6 ± 2.5	26.2 ± 2.6	0.054
Systolic BP [mmHg]	118.8 ± 16.7	119.6 ± 20.9	0.631
Diastolic BP [mmHg]	73.0 ± 13.7	71.5 ± 11.4	0.178
Heart rate [bpm]	89.6 ± 13.1	90.5 ± 14.8	0.854
Ejection fraction [%]	48.0 ± 9.8	45.0 ± 9.3	**< 0.001**
Risk factors:			
Diabetes	66 (34.4%)	207 (27.7%)	**0.044**
Hypertension	85 (44.3%)	279 (37.4%)	**0.049**
Hyperlipidaemia	138 (71.9%)	124 (16.6%)	**< 0.001**
Smoking	64 (33.3%)	308 (41.3%)	**0.026**
Laboratory findings:			
Glucose [mg/dL]	150.2 ± 83.8	159.1 ± 86.5	0.196
Total cholesterol [mg/dL]	192.3 ± 45.3	189.2 ± 45.9	0.648
Triglyceride [mg/dL]	158.2 ± 102.7	145.1 ± 112.8	0.157
HDL-C [mg/dL]	37.9 ± 9.6	38.1 ± 9.0	0.860
LDL-C [mg/dL]	122.8 ± 40.3	122.1 ± 36.2	0.808
Creatinine [mg/dL]	0.95 ± 0.48	1.05 ± 0.68	**0.057**
Haemoglobin [g/dL]	13.2 ± 1.8	13.5 ± 2.0	0.071
White blood cell count [10^3/μL]	9.9 ± 4.0	10.2 ± 4.1	0.256
CK-MB [ng/mL]	29.70 (0.80–300)	49.70 (0.50–500)	**0.001**
Troponin [ng/mL]	6.76 (0.01–100)	14.17 (0.01–100)	**< 0.001**

Data are shown as mean ± standard deviation, number (percentage) or median (minimum–maximum). Students t test, Kruskal Wallis and χ^2 tests were used. BMI — body mass index; BP — blood pressure; CK-MB — creatine kinase muscle and brain; HDL-C — high-density lipoprotein cholesterol; LDL-C — low-density lipoprotein cholesterol; IRA — infarct-related artery

patent IRA group were more likely to be male and have diabetes, hypertension, and hyperlipidaemia. Smoking habit was more frequent in the non-patent IRA group. Troponin and creatinine kinase MB levels were higher in the non-patent IRA group. Left ventricular ejection fraction was higher in the patent IRA group than in the non-patent IRA group ($p < 0.05$, for all; Table 1).

Medications used before MI are shown and compared between groups in Table 2. Pretreatment with statin was higher in the patent IRA group compared with the non-patent IRA group (138 [71.9%] vs. 79 [10.6%], $p < 0.001$). Atorvastatin and rosuvastatin usage was also higher in the patent IRA group compared with the non-patent IRA group ($p < 0.001$ for both). Statin usage duration was similar between groups (2.02 ± 1.04 vs. 1.97 ± 1.01 years, $p = 0.728$). Beta-blocker and oral antidiabetic drugs were used more frequently in the patent IRA group ($p < 0.05$, for all).

Our data were collected from 465 (49.6%) patients with anterior STEMI, 398 (42.4%) with inferior STEMI, 63 (6.7%) with inferior posterior STEMI, and 12 (1.3%) with high lateral STEMI. The prevalence of inferior MI in the patent IRA group was larger than the non-patent IRA group ($p = 0.05$). Anterior MI in the patent IRA group occurred less frequently than in the non-patent IRA group ($p = 0.009$). Also, the SYNTAX score was higher in the non-patent IRA group than in the patent IRA group (15.9 ± 6.8 vs. 12.1 ± 7.0, $p < 0.001$). The pain onset to first medical contact and door-to-balloon time were similar between groups (4.6 ± 5.1 vs. 5.1 ± 6.0 h, $p = 0.254$ and 32.6 ± 6.9 vs. 31.9 ± 7.4 min, $p = 0.281$; Table 3).

Multivariate logistic regression analysis

Infarct-related artery patency was independently associated with body mass index (odds ratio [OR] 1.090, 95% confidence interval [CI] 1.011–1.175, $p = 0.025$), previous chronic statin use (OR 0.067, 95% CI 0.046–0.099, $p < 0.001$), and SYNTAX score (OR 0.919, 95% CI 0.892––0.947, $p < 0.001$) in multivariate logistic regression analysis (Table 4).

Table 2. Comparison of medications used before primary percutaneous coronary intervention between groups

Variables	Patent IRA group (n = 192)	Non-patent IRA group (n = 746)	p
Previous medications:			
Statin use (total)	138 (71.9%)	110 (14.7%)	**< 0.001**
Atorvastatin use	95 (49.5%)	79 (10.6%)	**< 0.001**
Atorvastatin 10 mg	55 (28.6%)	36 (4.8%)	
Atorvastatin 20 mg	23 (11.9%)	18 (2.4%)	
Atorvastatin 40 mg	12 (6.2%)	14 (1.8%)	
Atorvastatin 80 mg	5 (2.6%)	11 (1.4%)	
Rosuvastatin use	43 (22.4%)	31 (4.2%)	**< 0.001**
Rosuvastatin 10 mg	31 (16.1%)	24 (3.2%)	
Rosuvastatin 20 mg	12 (6.2%)	7 (0.9%)	
Statin usage duration [years]:	2.02 ± 1.04	1.97 ± 1.01	**0.728**
ACE-I use	50 (26%)	168 (22.5%)	0.175
ARB use	35 (18.2%)	137 (18.4%)	0.530
Beta-blocker use	27 (14.1%)	62 (8.3%)	**0.013**
ASA use	40 (20.8%)	124 (16.6%)	0.104
OAD use	79 (41.1%)	207 (27.7%)	**<0.001**

Data are shown as mean ± standard deviation or number (percentage). χ^2 test was used. ACE-I — angiotensin converting enzyme inhibitor; ARB — angiotensin receptor blocker; ASA — acetylsalicylic acid; IRA — infarct-related artery; OAD — oral antidiabetic drug

Table 3. Comparison of angiographic findings between groups

Variables	Patent IRA group (n = 192)	Non-patent IRA group (n = 746)	p
Angiographic findings:			
SYNTAX score	12.1 ± 7.0	15.9 ± 6.8	**< 0.001**
LMCA involvement	1 (0.5%)	13 (1.7%)	0.184
LAD involvement	79 (41.1%)	372 (49.9%)	**0.019**
RCA involvement	78 (40.6%)	269 (36.1%)	0.139
CX involvement	22 (11.4 %)	71 (9.5%)	0.274
Other (diagonal, ramus) involvement	12 (6.2%)	21 (2.8%)	0.124
Type of infarct:			
Anterior MI	80 (41.7%)	385 (51.6%)	**0.009**
Inferior MI	92 (47.9%)	306 (41.0%)	**0.051**
High lateral MI	5 (2.6%)	7 (0.9%)	0.078
Inferior posterior MI	15 (7.8%)	48 (6.4%)	0.347
Pain onset to FMC time [h]	4.6 ± 5.1	5.1 ± 6.0	0.254
Door-to-balloon time [min]	32.62 ± 6.97	31.97 ± 7.47	0.281
Killip class 2–4	6 (3.1%)	68 (9.1%)	**0.03**
Cardiogenic shock	2 (1.04%)	9 (1.2%)	0.449

Data are shown as mean ± standard deviation or number (percentage). χ^2 and Kruskal Wallis tests were used. CX — circumflex artery; FMC — first medical contact; IRA — infarct-related artery; LAD — left anterior descending artery; LMCA — left main coronary artery, MI — myocardial infarction; RCA — right coronary artery

DISCUSSION

The present study evaluated the impact of previous treatment with statins and other treatments such as ACE inhibitor, beta-blocker, and oral antidiabetic drug use on pre-PCI IRA patency of patients with STEMI treated with primary PCI.

Chronic pretreatment of patients with acute coronary syndromes with statins has been associated with a decrease in risk of cardiovascular events, including mortality and recurrent MI [4, 11, 12]. Statin pre-treatment protects against myocardial damage during coronary intervention [13].

Table 4. Multivariate relationships of infarct-related artery patency

Variables	B	95% CI	p
Body mass index [kg/m^2]	1.090	1.011–1.175	0.025
SYNTAX score	0.919	0.892–0.947	< 0.001
Statin use	0.067	0.046–0.099	< 0.001

R^2 = 0.391. Binary logistic regression test was used. CI — confidence interval

Also, statin pre-treatment reduces the rate of MI after PCI [14]. These beneficial effects of statins are associated with anti-inflammatory as well as plaque-stabilising effects [15].

In previous studies it was shown that pretreatment with statins decreased the risk of no reflow phenomenon [7, 8]. Prior statin use has been shown to preserve coronary microvascular integrity during cardiac stress in experimental hypercholesterolaemia, independent of lipid lowering [16]. In addition, chronic administration of statins has cardioprotective effects in ischaemia/reperfusion injury despite unaltered cholesterol levels [17]. Celik et al. [7] reported that prior statin use may improve coronary blood flow after primary PCI in patients with STEMI. In another study, Iwakura et al. [8] enrolled 293 consecutive patients with STEMI undergoing successful primary PCI and found that patients receiving chronic statin treatment before admission had lower incidence of no-reflow than those without it (9.1% and 34.6%). Several mechanisms have been proposed for this beneficial effect. Pre-PCI chronic statin treatment may reduce embolic events during coronary intervention by decreasing lipid content and increasing the thickness of the fibrous cap of the atherosclerotic plaque [15]. The mechanism for achieving better myocardial perfusion may be related to inhibitory effects on platelets and blood coagulation, improvement in endothelial function, plaque-stabilising effects, and reduction in inflammatory response by statins [18, 19]. In patients with acute coronary syndrome, statins reduce monocyte adhesion molecules and serum P-selectin levels and inhibit the adhesion of leukocytes to the endothelium. These changes result in reduced capillary obstruction caused by platelet and leukocyte aggregates, which in turn results in preserved coronary microvascular permeability and reduced intracellular oedema after reperfusion. Statins reduce the expression of monocyte adhesion molecules, inhibit the adhesion of leukocyte to endothelia cells [20], and lower serum P-selectin levels in patients with acute coronary syndrome [21]. Statin treatment preserves the coronary microvascular permeability, which might lead to the reduction of intracellular oedema after reperfusion [16]. Statins have beneficial effects on endothelial function and nitric oxide (NO) production, independent of changes in serum LDL levels [6, 22], which might be responsible for the prevention of no-reflow phenomenon in acute coronary syndromes [8].

Present study showed that pretreatment with statin was associated with a patent IRA upon presentation in STEMI-patients. Also, in the present study, the relationship between pretreatment statin use and pre-PCI IRA patency was independent of the LDL-C levels. To our knowledge, there are not enough data regarding the effects of prior statin use on pre-PCI patency of IRA in patients with STEMI undergoing primary PCI. In our study, we found that patients with normal flow in IRA had higher incidence of prior statin use. In the present study, the positive impact on IRA patency was independent of the type of statins. The "pleiotropic" effects of statins could contribute to pre-PCI IRA patency as well as lower risk of no-reflow in patients with STEMI. The beneficial effects related with statin use, such as inhibitory effects on platelets and blood coagulation, improvement in endothelial function, NO production, plaque-stabilising effects and reduction in inflammatory response, may be responsible mechanisms for pre-PCI IRA patency [6, 18, 19]. Also, statins upregulate endothelial NO synthase activity [6, 23], inhibit platelet CD40 ligand mediated thrombin generation [24], and increase fibrous cap thickness and plaque stability [19]. In a study conducted on patients with the first STEMI, infarct-related coronary plaques were assessed with intravascular ultrasound and statin treatment before the onset of MI was associated with coronary plaque morphology with less necrotic core and greater fibrous and fibrofatty components at the culprit lesion in the IRA [25]. In a study by Robinson et al. [10], as the number of medications including statins, ACE inhibitors, acetylsalicylic acid, and beta-blockers increased, the likelihood of IRA patency increased. However, that study did not investigate the separate effects of drugs on pre-PCI IRA patency. In the present study, the frequency of pretreatment with beta-blocker was higher in the IRA patency group compared with the non-patent IRA group. The IRA patency was not independently associated with previous beta-blocker use. Similarly, although the frequency of pretreatment with oral anti-diabetic drug use was higher in the pre-PCI patent IRA group, it was not related to pre-PCI IRA patency in logistic regression analysis. Finally, only statin usage was independently associated with pre-PCI IRA patency in the present study.

Limitations of the study

Subjects participating in our study were enrolled prospectively, and statins were used before enrolment, which makes our study a retrospective cohort study in terms of the cause-effect relationship. Therefore, our results are subject to the weaknesses of retrospective design. Prospective enrolment in our study helps to avoid significant bias.

CONCLUSIONS

In our study, chronic statin therapy before the onset of acute MI was independently associated with IRA patency. Therefore,

statin therapy in patients free from cardiovascular disease may improve clinical outcomes of the first STEMI by improving pre-interventional IRA patency.

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References

1. Stone GW, Cox D, Garcia E, et al. Normal flow (TIMI-3) before mechanical reperfusion therapy is an independent determinant of survival in acute myocardial infarction: analysis from the primary angioplasty in myocardial infarction trials. Circulation. 2001; 104(6): 636–641, doi:10.1161/hc3101.093701, indexed in Pubmed: 11489767.
2. TIMI Study Group. The Thrombolysis in Myocardial Infarction (TIMI) trial. Phase I findings. N Engl J Med. 1985; 312(14): 932–936, doi:10.1056/NEJM198504043121437, indexed in Pubmed: 4038784.
3. Mehta RH, Harjai KJ, Cox D, et al. Primary Angioplasty in Myocardial Infarction (PAMI) Investigators. Clinical and angiographic correlates and outcomes of suboptimal coronary flow inpatients with acute myocardial infarction undergoing primary percutaneous coronary intervention. J Am Coll Cardiol. 2003; 42(10): 1739–1746, doi: 10.1016/j.jacc.2003.07.012, indexed in Pubmed: 14642681.
4. Nagashima M, Koyanagi R, Kasanuki H, et al. Heart Institute of Japan, Department of Cardiology (HIJC) Investigators. Effect of early statin treatment at standard doses on long-term clinical outcomes in patients with acute myocardial infarction (the Heart Institute of Japan, Department of Cardiology Statin Evaluation Program). Am J Cardiol. 2007; 99(11): 1523–1528, doi: 10.1016/j.amjcard.2007.01.024, indexed in Pubmed: 17531574.
5. Libby P. Molecular bases of the acute coronary syndromes. Circulation. 1995; 91(11): 2844–2850, doi: 10.1161/01.cir.91.11.2844, indexed in Pubmed:7758192.
6. Mason RP, Walter MF, Jacob RF. Effects of HMG-CoA reductase inhibitors on endothelial function: role of microdomains and oxidative stress. Circulation. 2004; 109(21 Suppl 1): II34–II41, doi: 10.1161/01.CIR.0000129503.62747.03, indexed in Pubmed: 15173061.
7. Celik T, Demirkol S, Celik M, et al. Statins and coronary microvascular dysfunction in patients with acute ST segment elevation myocardial infarction. Int J Cardiol. 2012; 155(3): 480–481, doi: 10.1016/j.ijcard.2011.12.073, indexed in Pubmed: 22245479.
8. Iwakura K, Ito H, Kawano S, et al. Chronic pre-treatment of statins is associated with the reduction of the no-reflow phenomenon in the patients with reperfused acute myocardial infarction. Eur Heart J. 2006; 27(5): 534–539, doi: 10.1093/eurheartj/ehi715, indexed in Pubmed: 16401674.
9. Schiller N, Shah P, Crawford M, et al. Recommendations for Quantitation of the Left Ventricle by Two-Dimensional Echocardiography. J Am Soc Echocardiogr. 1989; 2(5): 358–367, doi: 10.1016/s0894-7317(89)80014-8, indexed in Pubmed: 2698218.
10. Robinson CR, Martin JL, Zhang L, et al. Infarct-related coronary artery patency and medication use prior to ST-segment elevation myocardial infarction. Am J Cardiol. 2006; 97(1): 7–9, doi: 10.1016/j.amjcard.2005.07.103, indexed in Pubmed: 16377273.
11. Pedersen TR, Kjekshus J, Berg K, et al. Scandinavian Simvastatin Survival Study Group. Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). Lancet. 1994; 344(8934): 1383–1389, doi: 10.1016/s0140-6736(94)90566-5, indexed in Pubmed: 7968073.
12. Sacks FM, Pfeffer MA, Moye LA, et al. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. Cholesterol and Recurrent Events Trial investigators. N Engl J Med. 1996; 335(14): 1001–1009, doi: 10.1056/NEJM199610033351401, indexed in Pubmed: 8801446.
13. Pasceri V, Patti G, Nusca A, et al. ARMYDA Investigators. Randomized trial of atorvastatin for reduction of myocardial damage during coronary intervention: results from the ARMYDA (Atorvastatin for Reduction of MYocardial Damage during Angioplasty) study. Circulation. 2004; 110(6): 674–678, doi: 10.1161/01.CIR.0000137828.06205.87, indexed in Pubmed: 15277322.
14. Chan AW, Bhatt DL, Chew DP, et al. Early and sustained survival benefit associated with statin therapy at the time of percutaneous coronary intervention. Circulation. 2002; 105(6): 691–696, doi: 10.1161/hc0602.103586, indexed in Pubmed: 11839623.
15. Doo YC, Han SJ, Han SW, et al. Effect of preexisting statin use on expression of C-reactive protein, adhesion molecules, interleukin-6, and antioxidized low-density lipoprotein antibody in patients with unstable angina undergoing coronary stenting. Clin Cardiol. 2005; 28(2): 72–76, doi:10.1002/clc.4960280206, indexed in Pubmed: 15757077.
16. Bonetti PO, Wilson SH, Rodriguez-Porcel M, et al. Simvastatin preserves myocardial perfusion and coronary microvascular permeability in experimental hypercholesterolemia independent of lipid lowering. J Am Coll Cardiol. 2002; 40(3): 546–554, doi: 10.1016/s0735-1097(02)01985-x, indexed in Pubmed: 12142124.
17. Endres M, Laufs U, Huang Z, et al. Stroke protection by 3-hydroxy-3-methylglutaryl (HMG)-CoA reductase inhibitors mediated by endothelial nitric oxide synthase. Proc Natl Acad Sci U S A. 1998; 95(15): 8880–8885, doi: 10.1073/pnas.95.15.8880, indexed in Pubmed: 9671773.
18. März W, Wieland H. HMG-CoA reductase inhibition: anti-inflammatory effects beyond lipid lowering? Herz. 2000; 25(2): 117–125, doi:10.1007/pl00001949, indexed in Pubmed: 10829251.
19. de Lorenzo F, Feher M, Martin J, et al. Statin therapy-evidence beyond lipid lowering contributing to plaque stability. Curr Med Chem. 2006; 13(28): 3385–3393, doi: 10.2174/092986706779010324, indexed in Pubmed: 17168712.
20. Serrano CV, Yoshida VM, Venturinelli ML, et al. Effect of simvastatin on monocyte adhesion molecule expression in patients with hypercholesterolemia. Atherosclerosis. 2001; 157(2): 505–512, doi: 10.1016/s0021-9150(00)00757-7, indexed in Pubmed: 11472753.
21. Murphy RT, Foley JB, Mulvihill N, et al. Impact of preexisting statin use on adhesion molecule expression in patients presenting with acute coronary syndromes. Am J Cardiol. 2001; 87(4): 446–8, A6, doi: 10.1016/s0002-9149(00)01400-4, indexed in Pubmed: 11179531.
22. Sakabe K, Fukuda N, Wakayama K, et al. Lipid-altering changes and pleiotropic effects of atorvastatin in patients with hypercholesterolemia. Am J Cardiol. 2004; 94(4): 497–500, doi: 10.1016/j.amjcard.2004.04.067, indexed in Pubmed: 15325939.
23. Laufs U, La Fata V, Plutzky J, et al. Upregulation of endothelial nitric oxide synthase by HMG CoA reductase inhibitors. Circulation. 1998; 97(12): 1129–1135, doi: 10.1161/01.cir.97.12.1129, indexed in Pubmed: 9537338.
24. Sanguigni V, Pignatelli P, Lenti L, et al. Short-term treatment with atorvastatin reduces platelet CD40 ligand and thrombin generation in hypercholesterolemic patients. Circulation. 2005; 111(4): 412–419, doi: 10.1161/01.CIR.0000153810.81187.7D, indexed in Pubmed: 15687128.
25. Hikita H, Kuroda S, Oosaka Y, et al. Impact of statin use before the onset of acute myocardial infarction on coronary plaque morphology of the culprit lesion. Angiology. 2013; 64(5): 375–378, doi: 10.1177/0003319712449196, indexed in Pubmed: 22679133.

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