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# Highly effective lipid-lowering therapy on local vascular stiffness and symptoms of chronic heart failure in patients with postinfarction cardiosclerosis

V. E. Oleynikov, A. A. Khromova, E. A. Abramova, L. I. Salyamova, A. V. Babina, N. A. Tomashevsky Penza State University, Penza, Russia

#### Corresponding author:

Valentin E. Oleynikov, Penza State University, 40 Krasnaya street, Penza, 440026, Russia. Phone: 8(841)254–82–29. E-mail: v.oleynikof@gmail.com

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# Abstract

**Objective.** To study the effect of achieving the target level of total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) on the parameters of local vascular stiffness and the development of chronic heart failure (CHF) in patients with ST-elevation myocardial infarction (STEMI). Design and methods. The study included 80 patients with myocardial infarction with ST segment elevation (STEMI) aged 35 to 65 years. On the 7–9th day of STEMI, at the 24th and 48th weeks of treatment with atorvastatin 40–80 mg clinical symptoms of heart failure were analyzed, the lipid profile and brain natriuretic peptide (BNP) were determined. Ultrasound of the common carotid arteries using high-frequency RF signal technology was performed. Depending on the effectiveness of treatment with atorvastatin, patients were retrospectively divided into 2 groups: 40 people who reached the target level of LDL-C (highly effective therapy, HET) and 37 patients who did not reach the target level of LDL-C (relatively effective therapy, RET). **Results.** In patients who reached the target LDL-C level, the initial BNP values were 115.2 pg/ml, with the regression by 34.5 % at follow-up (p = 0.03). There was no significant change in the RET group. In the HET group, a decrease in IMT, an improvement in distensibility parameters, a decrease in local PWV and stiffness index of common carotid arteries, were found. Based on the 6-minute walk test, only patients in HET group showed an increase in exercise tolerance after 24th week (p = 0.04). Also, individuals who did not reach the target LDL-C level, showed a decrease in the quality of life 27.4% (p = 0.03). Patients in the HET group showed no negative dynamics. Conclusion. We showed that at the long-term follow-up after STEMI, the achievement of the target LDL cholesterol values was accompanied by a significant improvement in the parameters of local rigidity of the main arteries, and a decrease in BNP level, as well as by more favorable course of CHF.

**Key words:** myocardial infarction, vascular stiffness, atorvastatin, heart failure, hypertension

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V. E. Oleynikov et al.

Влияние высокоэффективной липидснижающей терапии на локальную сосудистую ригидность и симптомы хронической сердечной недостаточности при постинфарктном кардиосклерозе

В.Э. Олейников, А.А. Хромова, Е.А. Абрамова, Л.И. Салямова, А.В. Бабина, Н.А. Томашевский Федеральное государственное бюджетное образовательное учреждение высшего образования «Пензенский государственный университет», Пенза, Россия

#### Контактная информация:

Олейников Валентин Эливич, ФГБОУ ВО ПГУ, ул. Красная, д. 40, Пенза, Россия, 440026. Тел.: 8(841)254–82–29. E-mail: v.oleynikof@gmail.com

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## Резюме

Цель исследования — изучить влияние достижения целевых значений общего холестерина (OXC) и XC липопротеинов низкой плотности (XC ЛПНП) на параметры локальной сосудистой ригидности и развитие симптомов хронической сердечной недостаточности (ХСН) у больных инфарктом миокарда с подъемом сегмента ST (ИМпST). Материалы и методы. В исследование включено 80 больных ИМпST в возрасте от 35 до 65 лет. На 7-9-е сутки ИМпST, 24-й и 48-й неделях лечения аторвастатином 40-80 мг пациентам определяли уровень липидного спектра и мозгового натрийуретического пептида (ВNР). Также проводили ультразвуковое исследование общих сонных артерий с применением технологии высокочастотного сигнала RF. С помощью различных методик анализировали клинические симптомы ХСН. В зависимости от эффективности лечения аторвастатином больные были разделены на 2 группы: 40 человек, достигших целевого уровня ХС ЛПНП (группа высокоэффективной терапии, ВЭТ), и 37 больных, не достигших целевых значений ХС ЛПНП (группа относительно эффективной терапии, ОЭТ). Результаты. У больных, достигших целевых значений ХС ЛПНП, исходные значения ВNP составили 115,2 пг/мл, в результате лечения выявлен регресс на 34,5 % (р = 0,03). В группе ОЭТ значимой динамики не было. При ВЭТ отмечено снижение толщины комплекса интима-медиа (ТКИМ), улучшение параметров растяжимости, уменьшение локальной скорости распространения пульсовой волны (СРПВ), а также индекса жесткости общих сонных артерий. По результатам теста с шестиминутной ходьбой только больные группы ВЭТ продемонстрировали увеличение толерантности к физической нагрузке уже с 24-й недели (р = 0,04). Также у лиц, не достигших целевого уровня ХС ЛПНП, отмечено снижение качества жизни на 27,4% (р = 0,03). У пациентов группы ВЭТ отрицательной динамики не выявлено. Заключение. В настоящем исследовании показано, что достижение целевых значений ХС ЛПНП у больных, перенесших ИМпЅТ, в отдаленном периоде сопровождалось не только значимым улучшением параметров локальной ригидности магистральных артерий и снижением уровня BNP, но и более благоприятным течением развившейся XCH.

**Ключевые слова:** инфаркт миокарда, сосудистая ригидность, аторвастатин, сердечная недостаточность, артериальная гипертензия

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#### Introduction

A comprehensive study of etiopathogenesis mechanisms of cardiovascular diseases (CVDs), including coronary heart disease (CHD), has allowed solving problems of their treatment and prevention successfully. However, according to prognoses, despite significant achievements, cardiovascular pathology will remain the main cause of death at the global level for the next 20 years [1]. The incidence of ST-segment elevation myocardial infarction (STEMI) in European countries varies from 58 to 144 people per 100 thousand of the population per year [2]. It is known that one of the most important components of STEMI treatment is lipid metabolism correction. Low-density lipoprotein (LDL) reduction leads to a directly proportional reduction of cardiovascular risk [3], and has a positive effect on adherence to treatment and longterm prognosis.

**Objective** to study the influence of achieving the target values of total cholesterol and low-density lipoproteins on the parameters of local vascular stiffness, and the development of symptoms of chronic heart failure (CHF) in patients with STEMI.

# Design and methods

An open, single-center, prospective, comparative, controlled clinical research included 80 patients with STEMI. Sixty-eight men (85%) and 12 women (15%) were examined, average age was  $50.9 \pm 8.4$  years, body mass index (BMI) was  $27.1 (24.5; 29) \text{ kg/m}^2$ , blood pressure values were 115 (110; 125) and 76 (70; 80) mmHg; 55% (69 patients) had a history of arterial hypertension (AH); 53% (66 patients) were smokers. The criteria for inclusion in the research were age of patients from 35 to 65 years; the 2nd-4th days of STEMI; the presence of hemodynamically significant stenosis (more than 50%) of only one coronary artery (infarct-related artery), confirmed by coronary angiography (CA).

Exclusion criteria were repeated and recurrent acute myocardial infarctions; hemodynamically related stenosis (more than 50%) of two or more coronary arteries, and of the left main stem (more than 30%);

severe and/or paroxysmal forms of disturbance of heart rate and conduction; CHF in the history of FC III–IV according to NYHA; uncontrolled AH; liver failure or an increase in the level of bilirubin more than 1.5 times from normal values; active liver diseases or an increase of transaminase activity by more than 3 times; chronic kidney disease higher of Stage 3A; individual intolerance of statins; severe concomitant diseases precluding participation in the study.

The research protocol and primary documentation were approved by the Local Ethics Committee (FS-BEI HE Penza State University). All patients signed an informed consent prior to inclusion in the program. Clinical trial identification number is NCT02590653 (clinicaltrials.gov).

Treatment within the framework of study was started in the first 24–96 hours from the onset of the disease, the duration of observation and treatment was 48 weeks. Patients took Atorvastatin-TEVA® 40–80 mg per day (Teva Pharmaceutical Industries Ltd., Israel).

A comprehensive clinical and instrumental examination was carried out on the 7th-9th day of STEMI, and in the 24th and the 48th weeks of treatment: measuring of alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatine phosphokinase (CPK), total cholesterol (TC), low-density lipids (LDL–C), high-density lipoprotein cholesterol (HDL–C), triglycerides (TG), brain natriuretic peptide (BNP). Biochemical parameters were evaluated using an OLYMPUS AU 400 device (OLYMPUS CORPORATION, Japan).

Local stiffness was evaluated with common carotid artery (CCA) ultrasound imaging using the RF technology. The study was carried out according to the guidelines of the Mannheim Protocol [4], using a high-frequency linear sensor (13–14 MHz) of the MyLab 90 ultrasound scanner (Esaote, Italy). The following parameters were determined: local systolic pressure (loc Psys), local diastolic pressure (loc Pdia), pressure at a local point (P (T1), amplification pressure (AP), compliance coefficient (CC), distensibility coefficient (DC),  $\alpha$  and  $\beta$  stiffness indices, augmentation index (Aix), local pulse wave velocity (PWV), and carotid intima-media thickness (CIMT) [5].

26(1) / 2020 77

Using the Minnesota Questionnaire, the progression of CHF symptoms and their impact on the quality of life on the 7th-9th days, and after 48 weeks have been studied [6].

The clinical symptoms of CHF in patients were analyzed using the clinical assessment scale (CAS, modification by Mareev V. Yu., 2000) on the 7th-9th days, and after 48 weeks. The results were corresponded to FC I ( $\leq$  3 points); FC II (4–6 points); FC III (7–9 points); and FC IV (> 9 points) [6].

CHF was also evaluated by tolerance to physical activity during the six-minute walk test every 12 weeks. According to the results of the covered distance, the functional class of CHF was set [6].

For statistical processing of the research results, a licensed version of the STATISTICA 10.0 program (StatSoft, Inc., USA) was used. With a parametric distribution, the values were presented in the form of mean value and standard deviation ( $M \pm SD$ ); the Student's t-test was used for the analysis of related and unrelated groups. With nonparametric distribution, the values were presented by the median (Me) and interquartile scatter in the form of the 25th and 75th percentiles. To test the null hypothesis, the Wilcoxon rank test for related and the Mann-Whitney test for unrelated groups were used.

Using the above statistical methods, the statistical reliability of the data was estimated using tables of critical values. The differences were considered statistically significant at p < 0.05.

### Research results

In the course of this research, three patients have dropped out for the following reasons: death on the 16th day due to a myocardial rupture; one patient died in the 10th month of observation due to acute heart failure; and one patient did not follow the schedule of visits of the research center.

Depending on the effectiveness of treatment with atorvastatin, namely, the achievement of the target values of LDL–C by patients to study the dynamics of individual clinical and laboratory parameters, and arterial stiffness indicators, the patients were divided into two groups. Forty patients who reached the LDL–C level of less than 1.8 mmol/L and/or reduction by 50% from the initial indicators were included in the highly effective therapy (HET) group, and 37 patients who did not reach the LDL–C target values were combined into the relatively effective therapy (RET) group. It should be noted that patients were comparable in age, gender, anthropometric and anamnestic data, as well as in concomitant therapy (Table 1).

Among the individuals who made up the HET group, 26 (65%) patients received the maximum daily dose of atorvastatin. The parameters of lipid metabolism and BNP dynamics in patients of both groups are presented in Table 2.

The TG indices in both groups were within normal values before the start of the research, and they did not significantly change by the completion of the observation. However, in the group with the achievement of the target values of low-density lipids, the level of

COMPARATIVE CHARACTERISTICS OF GROUPS OF PERSONS «HET» AND «RET»

Table 1

| Indicator                   | «HET» group<br>(n = 40) | «RET» group<br>(n = 37) | р  |
|-----------------------------|-------------------------|-------------------------|----|
| Age, years                  | $51.4 \pm 9.8$          | 52.2 (49; 62)           | NS |
| Men, n (%)                  | 33 (82.5%)              | 32 (86.5%)              | NS |
| Women, n (%)                | 7 (17.5%)               | 5 (13.5%)               | NS |
| Weight, kg                  | 82.3 (75; 91)           | $84.1 \pm 12.6$         | NS |
| Height, cm                  | $172.9 \pm 7.2$         | $171.3 \pm 9.2$         | NS |
| BMI, kg/m <sup>2</sup>      | 26.9 (25; 29)           | $28.2 \pm 4.3$          | NS |
| HTN, n (%)                  | 25 (62.5%)              | 22 (59.5%)              | NS |
| Burdened inheritance, n (%) | 12 (30%)                | 11 (29.7%)              | NS |
| Tobacco smoking, n (%)      | 33 (82.5%)              | 27 (72,9%)              | NS |
| SBP (mm Hg)                 | 114 (110; 120)          | $112.3 \pm 10.2$        | NS |
| DBP (mm Hg)                 | 75 (65; 75)             | 70 (65; 75)             | NS |
| HR                          | $67 \pm 7.8$            | 65 (60; 70)             | NS |

**Note:** HET — group of highly effective lipid-lowering therapy; RET — group of relatively effective lipid-lowering therapy; BMI — body mass index; HTN — hypertension; SBP — systolic blood pressure; DBP — diastolic blood pressure; HR — heart rate; p — significant difference between 7–9th day and 48th week, NS — non-significant differences.

Table 3

## THE LIPID PROFILE AND THE LEVEL OF BNP IN THE COMPARISON GROUPS

| Indicator     | «HET» group (n = 40) |                 | р    | «RET» group (n = 37) |                   | р    |
|---------------|----------------------|-----------------|------|----------------------|-------------------|------|
|               | 7–9 days             | 48 weeks        | Р    | 7–9 days             | 48 weeks          | P    |
| TC, mmol/l    | $6.2 \pm 1.4$        | $3.5 \pm 0.8$   | 0.01 | $5.7 \pm 1.1$        | 4.1 (3.7; 4.8)    | 0.01 |
| HDL-C, mmol/l | 1.2 (0.9; 1.4)       | 1.1 (1.0; 1.3)  | 0.01 | 1.2 (0.99; 1.4)      | $1.2 \pm 0.3$     | NS   |
| LDL-C, mmol/l | 4.3 (2.9; 5.2)       | $2.0 \pm 0.5$   | 0.01 | 3.5 (1.8; 4.0)       | 2.8 (2.5; 3.2)    | 0.01 |
| TG, mmol/l    | 1.2 (0.7; 1.7)       | 1.2 (0.9; 1.5)  | NS   | 1.1 (0.6; 1.6)       | $1.5 \pm 0.5$     | NS   |
| BNP, pg/ml    | 115.2 (42.8; 144.2)  | 75.4 (16.8; 89) | 0.03 | 101.8 (46; 137.5)    | 78.2 (20.2; 74.8) | NS   |

**Note:** HET — group of highly effective lipid-lowering therapy; RET — group of relatively effective lipid-lowering therapy; TC — total cholesterol; HDL — high-density lipoproteins; LDL — low-density lipoproteins; BNP — brain natriuretic protein; p — significant difference between 7–9th day and 48th week, NS — non-significant differences.

ДИНАМИКА ПОКАЗАТЕЛЕЙ ЛОКАЛЬНОЙ ЖЕСТКОСТИ ОБЩИХ СОННЫХ АРТЕРИЙ И ТОЛЩИНЫ КОМПЛЕКСА ИНТИМА-МЕДИА В ГРУППАХ СРАВНЕНИЯ

| Indicator      | «HET» group       |                    |        | «RET» group         |                    |        |
|----------------|-------------------|--------------------|--------|---------------------|--------------------|--------|
|                | 7–9 days          | 48 weeks           | р      | 7–9 days            | 48 weeks           | p      |
| CIMT, μm       | $724.3 \pm 125.3$ | $602.5 \pm 109.7$  | 0.0001 | 697 (581; 832)      | $681.2 \pm 134.9$  | NS     |
| DC, 1/кРа      | 0.02 (0.02; 0.03) | 0.03 (0.025; 0.04) | 0.02   | 0.02 (0.01; 0.025)  | 0.02 (0.02; 0.025) | NS     |
| CC, mm²/кPa    | $0.90 \pm 0.28$   | $0.93 \pm 0.31$    | ur     | 0.87 (0.69; 1.1)    | $0.98 \pm 0.42$    | NS     |
| Index α        | 4.2 (3.8; 5.4)    | 3.1 (2.5; 4.8)     | 0.01   | 5.1 (4.7; 6.2)      | 4.8 (4.5; 5.9)     | NS     |
| Index β        | 8.4 (7.6; 10.2)   | 7.4 (5.9; 8.1)     | 0.01   | 9.3 (7.1; 11.1)     | 8.6 (6.9; 10.9)    | 0.02   |
| PWV, m/s       | 7.1 (6.2; 7.9)    | 5.4 (4.3; 6.8)     | 0.03   | 7.6 (6.8; 8.7)      | 7.4 (6.5; 8.2)     | NS     |
| Loc Psys, mmHg | $104.4 \pm 9.2$   | 107.5 (102.3; 112) | NS     | $106.8 \pm 12.1$    | $109.2 \pm 11.7$   | NS     |
| Loc Pdia, mmHg | 70 (70; 80)       | 75 (70; 80)        | NS     | 70 (65; 80)         | 79.5 (75; 80)      | 0,03   |
| P (T1), mmHg   | 102.4 (95; 107.2) | $103.1 \pm 10.4$   | NS     | 100.4 (96.8; 107.5) | $104.8 \pm 11.6$   | NS     |
| AP, mmHg       | 1.7 (1.1; 3.5)    | 4.2 (2.8; 7.1)     | 0.0001 | 2.4 (1.2; 4.3)      | $6.1 \pm 2.9$      | 0.0001 |
| Aix, %         | 1.4 (-1.1; 2.9)   | 4.7 (3.2; 6.5)     | 0.0001 | $1.7 \pm 3.9$       | $5.1 \pm 2.9$      | 0.0003 |

**Note:** p — significant differences between the values of the indicators at 7–9 days and 48 weeks.

HDL–C reduced by 7.8% (p < 0.01). The BNP level in patients of the RET group was 101.8 pg/mL before treatment, and it was 78.2 pg/mL after 48 weeks; no reliable dynamics was found. In patients who reached the target values of LDL–C, the initial BNP values were 115.2 pg/mL, and 75.4 pg/mL after treatment. The reduction of this laboratory parameter was observed from the 24th week (p = 0.04), and there was a regression by 34.5% (p = 0.03) by the end of the observation.

It is proved that the CIMT value is an important indicator characterizing the risk of developing cardiovascular complications. The CCA ultrasound imaging using the RF technology allows conducting the measurement of this indicator with high accuracy due to high resolution and operator independence of the method.

The regression of CIMT in patients with target and lower values of atherogenic lipids during treatment is a logical and explainable result. The extensibility of the arterial wall, local PWV, as well as indices reflecting true vascular stiffness, had reliable dynamics only in the HET group (Table 3).

According to the results of the six-minute walk test, there was no change of the covered distance in the RET group, and patients of the HET group showed an increase of tolerance to physical activity from the 24th week (p = 0.04) (Figure 1).

**79** 

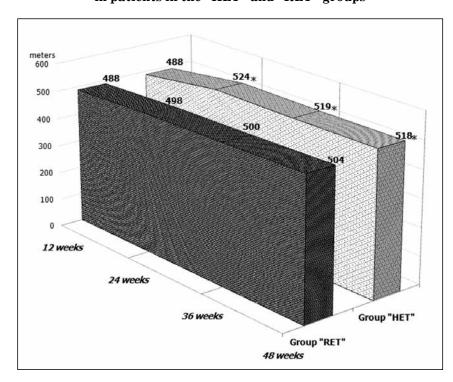


Figure 1. Distance covered according to 6 minute-walk test in patients in the "HET" and "RET" groups

**Note:** \* — p < 0.05 — differences between the values of the 7th–9th days and subsequent visits.

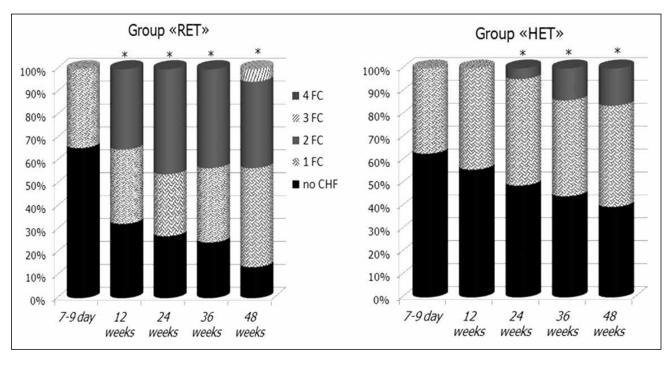


Figure 2. Evaluation of the CAS values in patients of the "HET" and "RET" groups

Note: \* — p < 0.05 — differences between the values of the 7th–9th days and subsequent visits.

When studying the results of the Minnesota Questionnaire, there was a decrease in the quality of life by 27.4% (p = 0.03) in patients who did not reach the target level of LDL–C. There was no negative dynamics in patients of the HET group.

When assessing the clinical symptoms of CHF by CAS, STEMI was revealed on the average of 1 (0; 1.5) point in the RET group on the 7th-9th day; it was 1 (0; 2) point (p > 0.05) after 24 weeks of observation, and by the end of therapy it was 2 (1; 3) points (p = 0.01). In patients of the HET group, the average level of points did not undergo significant dynamics, and initially it was 1 (0; 1) point; it was 1 (1; 2) point after 24 weeks, and by the 48th week of observation it was 1 (1; 2) point. A detailed description of the functional classes of CHF in the comparison groups is presented in Figure 2.

## Discussion

Cardiovascular diseases (CVDs) hold a leading position among the causes of mortality in the developed countries of the world. In Europe, more than four million people die from heart and blood vessel diseases, and their complications per year. Thanks to the introduction of modern methods of early diagnosis, and invasive and drug methods of treatment, many patients survive after the first episode of CVD, but are in the ultra-high-risk group of relapses for life. Data of multicenter research indicate that a decrease of TC and LDL–C level in patients of the above group is directly connected with a clinically and statistically significant reduction of the risk of death from cardiovascular pathology [7, 8]. This determines the methodology of this research with division of STEMI patients in groups depending on the effect of atorvastatin on LDL–C level. A meta-analysis of the results of 26 randomized clinical trials involving more than 170,000 patients indicates that with a decrease of LDL-C level by 1 mmol/L, there is a decrease of mortality: from all causes by 10%, from CVD by 20%, from the risk of coronary complications by 23%, and from stroke by 17% [9].

Initially, higher absolute values of atherogenic lipids have been revealed in the HET group (Table 2), and a more significant regression of these parameters was revealed after 12 months of treatment. In general, the LDL–C level decreased by 2.1 mmol/L (–52.5 %, p < 0.01) in the HET group. In patients of the RET group, the regression was 0.71 mmol/L (–20.4 %, p < 0.01). Our results indicate a dose dependent effect of statins on the level of LDL–C, since 65 % of patients have received the maximum daily dose of atorvastatin in the HET group. The above results are consistent

with recommendations confirming a direct proportional dependence of the dose, and the degree of decrease of LDL–C and cardiovascular risk [10].

Literature review data on changes in the concentration of HDL-C during treatment with HMG-CoA reductase inhibitors are controversial. A number of studies have shown that lovastatin and pravastatin increase HDL-C by 10%, simvastatin by 14%, atorvastatin by 2-4%, and may even lower HDL-C. It follows from some studies that statins that are less effective in lowering LDL-C (fluvastatin) have a more pronounced effect on the concentration of HDL-C. In accordance with this hypothesis, atorvastatin in low doses increases the level of HDL-C, but is able to reduce their concentration during intensive therapy [11, 12]. We have found a similar pattern: along with a significant decrease in total cholesterol and LDL, the level of HDL–C decreased by 0.09 mmol/L (-7.8%, p < 0.05), and the average values amounted to 1.07 (1.0; 1.29) mol/L in the HET group after 12 months of observation.

It is known that an increase in arterial stiffness, regardless of other factors, entails a risk of heart failure [4, 5]. Initially, with increasing stiffness of the vascular wall, cardiac afterload increases, resulting in diastolic dysfunction, left ventricular hypertrophy and diastolic heart failure. An increase in the rigidity of the great vessels leads to a shift of the reflected wave from diastole to late systole, which entails a decrease in diastolic pressure, and, consequently, deterioration in coronary perfusion [13]. The processes described above in a STEMI patient are only aggravated due to the inclusion of many compensation and activation mechanisms components of the renin-angiotensin-aldosterone system and hypersympathicotonia, in response to an acute decrease in stroke volume and cardiac output [14].

The research results suggest that most factors of cardiovascular risk realize their influence through a change in the elastic properties of the vascular wall [15]. Therefore, an assessment of the structural and functional properties of large arteries can give an idea of the effect of one or more factors on the course and prognosis of a post-myocardial infarction period.

It is customary to evaluate elastic properties of arteries by the PWV parameter. H. Tomiyama et al. showed that PWV is an independent predictor of cardiovascular complications in patients with acute coronary syndrome, measured upon admission to the hospital [16].

It was found that PWV in the carotid artery significantly decreased by 16% (p < 0.05) in STEMI patients with target LDL–C after 12 months.

The ARIC study [17] is devoted to the study of carotid stiffness parameters in coronary heart disease in 15,792 patients aged 45 to 64 years. It was proved that the stiffness of the carotid arteries was significantly worse in patients with coronary artery disease compared with healthy individuals of a similar age.

In this study, the parameters of local stiffness of the carotid arteries were investigated in detail. It was found that in all patients with a very high cardiovascular risk, regardless of the achievement of the target values of atherogenic lipids, the carotid artery extensibility improved assessed by DC, but the stiffness index  $\beta$ , as well as the local augmentation index and augmentation pressure in the carotid artery decreased.

The development of CHF in patients after STEMI determines the prognosis, duration and quality of life, as it increases morbidity and mortality by 3-4 times [6]. As part of the Framingham Heart Study, it was demonstrated that a high concentration of cholesterol in the blood is associated with a high risk of CHF [6, 18]. The administration of statins in patients with an existing coronary pathology led to a decrease in the incidence of CHF by 9-45% in addition to correction of lipid metabolism [19, 20]. The results of several prospective randomized trials indicate that more intensive therapy with HMG-Co-A reductase inhibitors reduced the hospitalization rate by 27% due to the development of heart failure symptoms in patients with the medical history of CHD without chronic heart failure [21].

In recent years, very controversial data on the benefits of statins in CHF have been published [22]. However, there are a number of effects that explain their impact on the pathophysiological mechanisms of CHF, and can affect the symptoms and prognosis. One of them is improving endothelial function, and as a result, restoring its barrier function, vasodilation, reducing ischemia of the heart and kidneys. In addition, antithrombotic, anti-inflammatory, and antiproliferative effects contribute to the regression of myocardial hypertrophy and slower atherogenesis [23].

To control the symptoms and severity of CHF in STEMI patients, BNP level analysis and the six-minute walk test were used. It was revealed that the achievement and maintenance of the target values of LDL–C probably caused a regression of the objective parameters of the chronic heart failure developed after STEMI. An effective lipid-lowering therapy was accompanied by improved quality of life for patients according to the Minnesota Questionnaire. The decrease in the quality of life in patients who did not reach the target values of LDL–C is associated with the dissatisfaction of patients with socio-economic aspects and their

participation in the life of family and friends, as well as a decrease in the positive emotional perception of life in these patients.

In the HET group, a significant decrease in BNP and an increase in exercise tolerance were registered after 24 weeks, while maintaining the achieved result in the 48th week of observation.

The PWV dependence on the CHF severity was investigated. The higher PWV values were found in patients with CHF compared with the control group of healthy individuals. In addition, a decrease in arterial extensibility and a progressive decrease in compliance were associated with an increase in the severity of CHF [21, 22].

In our study, the achievement of the target values of atherogenic lipids was accompanied by a significant improvement in the properties of the vascular wall. Apparently, the regression of local PWV and an increase in the extensibility of the main arteries indirectly affected the quality of life of STEMI patients, which is confirmed by the results of testing patients using the Minnesota Questionnaire and assessing the severity of CHF by the CAS.

A retrospective analysis of the CAS results in both groups showed almost the same ratio of patients with FC I CHF, and patients who did not have CHF symptoms (Figure 2). In the 24th week of observation, 50% RET group patients belonged to FC II, and there were less than 10% (p < 0.05) patients in the group with the achievement of target lipids. The number of RET group patients decreased to 38%, while, on the contrary, it rose to 20% in the HET group after two years treatment. However, in the group of less effective therapy, individuals (10%) appeared whose clinical picture of CHF corresponded to FC III. After 48 weeks, the proportion of patients who did not have manifestations of CHF in the group of highly effective therapy was 35%, while in patients with less effective statin therapy this parameter approached 10%.

# **Conclusions**

One of the most important tasks in the treatment of patients with STEMI is successful secondary maintenance therapy, which implies prescribing drugs that affect pathogenetic mechanisms of CHD, and the effective control of their use. In the present study, it was shown that the achievement of the target LDL–C values in patients who underwent STEMI was accompanied not only by a significant improvement in the parameters of local rigidity of the main arteries and a decrease in the BNP level, but also by a more favorable course of developed chronic heart failure.

Conflict of interest / Конфликт интересов
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# **Author information**

Valentin E. Oleynikov, MD, PhD, DSc, Professor, Head, Department of Therapy, Medical Institute, Penza State University, e-mail: oleynikof@gmail.com;

Angelina A. Khromova, MD, PhD, Senior Lecturer, Department of Therapy, Medical Institute, Penza State University, e-mail: hromova-a.a@yandex.ru;

Evgenia A. Abramova, MD, PhD, Senior Lecturer, Department of Therapy, Medical Institute, Penza State University, e-mail: melnikova1910@gmail.com;

Lyudmila I. Salyamova, MD, PhD, Associate Professor, Department of Therapy, Medical Institute, Penza State University, e-mail: l.salyamova@yandex.ru;

Anastasiya V. Babina, MD, Postgraduate Student, Penza State University:

Nikita A. Tomashevsky, Medical Student, Penza State University.

## Информация об авторах

Олейников Валентин Эливич — доктор медицинских наук, профессор, заведующий кафедрой терапии медицинского института ФГБОУ ВО «Пензенский государственный университет», e-mail: oleynikof@gmail.com.

Хромова Ангелина Анатольевна — кандидат медицинских наук, старший преподаватель кафедры терапии медицинского института ФГБОУ ВО «Пензенский государственный университет», e-mail: Hromova-a.a@yandex.ru;

Абрамова Евгения Александровна — кандидат медицинских наук, старший преподаватель кафедры терапии медицинского института ФГБОУ ВО «Пензенский государственный университет», e-mail: melnikova1910@gmail.com;

Салямова Людмила Ивановна — кандидат медицинских наук, доцент, доцент кафедры терапии ФГБОУ ВО «Пензенский государственный университет» e-mail: l.salyamova@yandex.ru;

Бабина Анастасия Вячеславович — аспирант ФГБОУ ВО «Пензенский государственный университет»;

Томашевский Никита Андреевич — студент ФГБОУ ВО «Пензенский государственный университет».