



## EVALUATION OF THE CLINICAL EFFECT OF SIMVASTATIN IN PATIENTS WITH UNSTABLE ANGINA AT A YOUNG AGE

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| Article history:   | Abstract:   |
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| <b>Received:</b> 11 <sup>th</sup> February 2023<br><b>Accepted:</b> 11 <sup>th</sup> March 2023<br><b>Published:</b> 17 <sup>th</sup> March 2023 | The clinical effect of rosuvostatin in patients with coronary heart disease (CHD) at young age was studied. We examined 120 patients with CHD aged 18 to 44 years (mean age 32.0±5.0 years). All patients underwent history taking, anthropometry, general clinical, laboratory, biochemical (lipid spectrum) and instrumental examinations. The obtained analysis of lipid profile in patients with IHD showed that simvastatin in the daily dose of 20 mg in 3 months showed hypolipidemic effect with good tolerability. |

**Keywords:** CHD, young age, atherosclerosis, lipoproteins, Rosuvastatin

**INTRODUCTION:** CHD is considered as a polyethylological disease manifested by acute or chronic mismatch of myocardial oxygen demand and its delivery through coronary arteries (CA). In young individuals coronary circulation disorder can be impaired due to atherothrombosis, spasm, CA obstruction and many other causes unrelated to CA atherosclerosis (pronounced CA tortuosity, endothelial dysfunction (ED), reduced local nitric oxide production, pathological susceptibility to heart pain, etc.). [1, 10, 25]. Often the pathogenesis of CHD is mixed, whereas along with atherosclerotic stenosis of coronary artery there are reversible changes of coronary blood flow associated with vascular tone (spasm or ED). In young age, according to coronary angiography (CAG) data, about 80% of acute myocardial infarction (AMI) cases occur against the background of atherosclerosis of CA, about 20% of cases it occurs against the background of unchanged or slightly altered vessels [4, 11, 20].

One of the main and most frequent etiological causes of CHD is stenotic atherosclerotic changes in the coronary arteries with lumen narrowing from 50 to 70%, which can go on for many years without clinical manifestations, until it leads to acute or chronic coronary events. Asymptomatic atherosclerotic changes in the coronary artery associated with LDL are detected at a young age and steadily progress over decades, even in middle age the frequency of detection of atherosclerotic changes in the coronary artery approaches 100% before they lead to the development of clinical manifestations of CVA [2, 3, 5, 8, 17]. Atherosclerotic changes in CA are based on lipoprotein metabolism disorders [7, 9, 10, 13, 15].

When destabilizing the course of CHD, the most important prognostic risk factor is considered to be lipid metabolism disorder (DLD), in which the concentration of lipids/lipoproteins in blood is observed to increase [6, 12, 14, 16]. Much attention has been paid for many years to the early detection, study, and adequate correction of elevated total cholesterol (TC) and low-density lipoprotein (LDL) levels, as they are atherogenic lipoproteins [2, 14, 15, 20]. Increased levels of LDL, amounts of small LDL particles, very low-density lipoprotein (VLDL) and decreased levels of high-density lipoprotein HDL are the three main high risk lipoproteins for CA atherosclerosis and the complications associated with them [6, 9, 10, 12, 18]. A 10% decrease in plasma levels of CHC contributes to a 25% reduction in the incidence of CHD in 5 years, and a decrease in LDL by 1 mmol/L is accompanied by a 20% reduction in cardiovascular disease (CVD) [5, 11, 13, 21]. Every fifth man is found to have a lower HDL level, every third man is found to have hypertriglyceridemia [6, 24, 25]. A sufficient number of studies have documented a high prevalence of lipid abnormalities in young individuals with CHD compared to the older age group [19, 22]. Lower HDL levels and higher triglyceride (TG) levels were observed among young patients with CHD, which once again states that LDL in young adults is one of the important risk factors in the development of CHD [16, 18, 23].

The development of early diagnostic methods, prevention and pathogenetic adequate correction of atherogenic DLD is considered to be one of the urgent problems of modern cardiology. The study of the problems associated with subclinical atherosclerosis is considered important because the detection and treatment of DLD in the early stages of the

pathological process, can be potentially reversible or significantly slow its progression. In this connection, the development of optimal diagnostic and therapeutic algorithms will help to effectively solve the problems associated with the atherosclerotic process.

**PURPOSE OF THE STUDY:** to evaluate clinical effect of simvastatin in patients with unstable angina pectoris at young age.

**MATERIALS AND METHODS:** this study was conducted in Samarkand regional branch of the Republican Specialized Scientific-Practical Medical Center of Cardiology (SRF RSPMC). 110 patients aged 18-44 years (mean age  $32.0 \pm 5.0$  years) admitted with the diagnosis of unstable angina pectoris were included in the investigation. All the patients had history taking, anthropometry, general clinical, laboratory, biochemical (lipid spectrum) and instrumental studies.

**RESULTS:** all patients with CHD depending on sex were divided into 2 groups: the 1st group included 65 (59,1%) male patients, the 2nd group included 45 (40,9%) female patients. The control group consisted of 52 volunteers .

Our study assessed the influence of LDL on the clinical course of CHD in order to determine the predictors of the prognosis of adverse outcomes. One of the objectives of the present study is to assess lipid status in patients with CHD, as a result, we studied the lipid spectrum among young patients. As the results of the study showed that levels of CHD, LDL, TG in both groups were elevated, CHD in group 2 was 0.33 mmol/l higher than in group 1 and was  $7.13 \pm 0.75$  mmol/l and  $6.8 \pm 0.86$  mmol/l respectively, ( $p < 0.001^*$ ), in the control group CHD averaged  $3.32 \pm 0.60$  ( $p < 0.001^*$ ).

There were no statistically significant differences between the groups in terms of HDL levels in group 1,  $1.0 \pm 0.15$  mmol/l, in group 2,  $0.97 \pm 0.16$  mmol/l, ( $p = 0.034^*$ ), although this index was lower than normal in the elderly group, in the control group this index was  $1.2 \pm 0.18$  mmol/l ( $p < 0.001^*$ ). LDL in group 1 was  $4.5 \pm 0.83$  mmol/l, in group 2  $4.32 \pm 0.62$  mmol/l, respectively ( $p = 0.038^*$ ), which shows impaired lipid metabolism in patients with CHD, in the control group LDL averaged  $2.96 \pm 0.83$  mmol/l ( $p < 0.001^*$ ). In patients in group 1, the TGs were significantly higher and were  $3.11 \pm 0.92$  mmol/l, and in group 2 it was  $2.87 \pm 0.81$  mmol/l, ( $p < 0.0001^*$ ), in the control group the TGs were  $2.21 \pm 0.74$  mmol/l ( $p < 0.001^*$ ). CA was elevated in both groups, which was  $5.92 \pm 1.26$  in group 1,  $6.52 \pm 1.2$  in group 2 in control group  $1.83 \pm 0.8$  ( $p = 0.03$ ).

All patients, starting from the first day of admission, were given dietary recommendations. On day 5, simvastatin at a dose of 20 mg per day was added to the diet therapy. After a 3-month course of treatment the patients' state, frequency of angina attacks and biochemical analysis to determine the lipid spectrum were re-evaluated. Under the influence of simvastatin against the background of basic therapy with nitrates and beta-blockers the number of angina attacks and doses of nitroglycerin taken improved. Treatment contributed to a significant decrease in the levels of CHC, LDL, TG already in a month after taking the drug. LDL decreased by 21%, the level of CHC decreased by 13% from the initial level. HDL increased by 14%, while TG level decreased only by 7%. The target LDL level of 3.0 was achieved only in 41% of patients, and levels less than 2.6 mmol/L in 5 patients. In the course of treatment, adverse events occurred in patients in the form of dyspeptic disorders.

**CONCLUSIONS:** thus, the results of a 3-month follow-up showed that, the use of simvastatin at 20 mg/day for 3 months a pronounced hypolipidemic effect, with good tolerability. In addition, simvastatin therapy not only reduces atherogenic lipoproteins in the blood, but also helps to increase the level of antiatherogenic lipoproteins and lead to "stabilization" of atherosclerotic plaque in the coronary arteries in patients with unstable angina pectoris correlates with an improvement in the subsequent course of the disease.

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