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The influence of atorvastatin on endothelium dysfunction and combined course of ischemic heart disease, essential hypertension and chronic obstructive pulmonary disease

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Aim: To investigate the atorvastatin influence on severity of stable angina pectoris, blood pressure monitoring (BPM) and respiratory function indexes (RFE).

Materials and methods: 30 patients with ischemic heart disease in combination with essential hypertension (EH) and chronic obstructive pulmonary disease (COPD) with plasma concentration of the total cholesterol (TCH) more than 5.0 mmol/l and the cholesterol low density lipoproteins (LDL) more than 3.5 mmol/l before atorvastatin 20 mg per day treatment and three month after were surveyed. Blood pressure by BPM, tolerance to physical load by veloergometry, RF by computer spirograph, endothelium dysfunction by measuring a degree of the endothelium-dependent relaxation of a humeral artery with Doppler through a humeral artery diameter change at reactive hyperemia, C-reactive protein (CRP), interleukin-6 (IL-6), a tumor necrosis factor alpha (TNF- α) concentration, patients' quality of life were estimated. At realization of spyrography we estimated the forced vital capacity (FVC), the forced expiratory volume in 1 s (FEV₁), Tiffno index (FEV₁/FVC).

Results: After three month atorvastatin treatment the average TCH level has decreased from 5.89 \pm 0.13 mmol/l to 4.08 \pm 0.09 mmol/l, the cholesterol LDL level has decreased from 3.77 \pm 0.07 mmol/l to 2.10 \pm 0.09 mmol/l, the physical loading tolerance has increased at 24 patients (80%), on average, by 20.4 \pm 11.8 w. The interrogation has revealed reduction of angina pectoris' attacks numbers by 46.1%, and quantity of accepted nitroglycerine tablets for day by 3.7, on average. The average daily systolic BP (SBP) level has decreased by 9.1 mm Hg (p<0.05), diastolic BP (DBP) level has decreased by 7.3 mm Hg (p<0.05), SBP night decrease index has increased by 4.5 mm Hg (p<0.05). Morning DBP raising index has decreased by 5.7 mm Hg (p<0.05). After atorvastatin treatment the bronchial resistance has authentically decreased. No one surveyed patients has FVC level changed. It is demonstrated that obstructive syndrome in the examined patients caused by increased resistance both small and large airways. The obstruction of large airway correlates with degree of endothelial dysfunction. After atorvastatin treatment forced expiratory volume in 1 second (FEV1) has increased by 0.51 p/a (p<0.05) and occurred due to large bronchial tubes dilatation – by 11.2% (p<0.05). Tiffno index has increased by 6,4 \pm 1,1% (p<0,05), on average. Endothelial dysfunction in all examined patients decreased (a humeral artery diameter relaxation at reactive hyperemia has increased from 0.38 \pm 0.03 mm (8.2%) to 0.56 \pm 0.05 mm (13.3%) (p<0,05). Patients' quality of life is improved. The CRP level decreased by 0.79 pg/ml (p<0.05). IL-6 – by 1.7 pg/ml (p<0.05), TNF- α – by 17.6 % (p<0.05).

Conclusion: Atorvastatin inclusion in treatment of IHD in combination with EH and COPD promoted lipid exchange, systemic inflammation activity decrease and stimulation of synthesis of vaso- and bronchorelaxed nitric oxide. At this background the endothelial function is normalized, tolerance to physical load is increased, angina pectoris course, BP daily rhythm, RFE indexes and patients' quality of life is improved.

Keywords: atorvastatin, endothelial dysfunction, chronic obstructive pulmonary disease

Simvastatin influence on left ventricle diastolic function and endothelial function at patients with metabolic syndrome features

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Aim: To investigate simvastatin influence on left ventricle diastolic function (LVDF) and endothelium function in patients with metabolic syndrome features.

Materials and methods: 32 male patients with essential hypertension (EH) and metabolic syndrome features, such as low-density lipoproteins (LDL) cholesterol more than 3.0 mmol/l, high-density lipoproteins (HDL) cholesterol less than 1.0 mmol/l, abdominal obesity and impaired fasting glycaemia before simvastatin 20 mg per day treatment and four month after were surveyed. Endothelium function by measuring a degree of the endothelium-dependent relaxation of a humeral artery with Doppler through a humeral artery diameter change at reactive hyperaemia, LVDF by isovolumic relaxation time (IVRT), E/A ratio, epicardial adipose tissue thickness (EATT), LDL, HDL cholesterol and serum fasting glucose concentration.

Results: After four month simvastatin treatment LDL cholesterol level has decreased from 3.43 ± 0.15 mmol/l to 2.68 ± 0.07 mmol/l, HDL cholesterol level has increased from 0.89 ± 0.06 mmol/l to 1.12 ± 0.05 mmol/l ($p < 0.05$). Thus, 29 patients (90.6%) had normal lipoproteins ratio. Endothelial function in all examined patients improved (a humeral artery diameter relaxation at reactive hyperemia has increased from 0.36 ± 0.03 mm (8.1%) to 0.57 ± 0.04 mm (13.4%) ($p < 0.05$). IVRT has improved from 82.7 ± 3.2 ms to 63.1 ± 2.9 ms, E/A ration has significantly increased from 0.61 ± 0.04 to 0.98 ± 0.04 . Thus, 28 patients (87.5%) had improved diastolic function. There was not significant change in fasting glucose level. Before simvastatin management its level was 6.4 ± 0.33 mmol/l and after four month – 6.21 ± 0.21 mmol/l ($p = 0.53$). In addition, 4-month simvastatin treatment did not affect EATT: 0.43 ± 0.02 cm and 0.39 ± 0.04 cm ($p = 0.66$).

Conclusion: Simvastatin inclusion in treatment of patients with metabolic syndrome features promotes lipid exchange improve, but does not influence glycemic exchange. Four-month simvastatin treatment improves endothelial function and increases diastolic function if it is impaired.

Keywords: simvastatin, metabolic syndrome, left ventricle diastolic function, endothelial function