Stratification of cardiometabolic risk factors in patients with arterial hypertension and diabetes mellitus type 2

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Purpose: to study the components of the cardiometabolic syndrome and their role in the end-organ damage (EOD) in patients with arterial hypertension (AH) and type 2 diabetes mellitus (DM-2).

Methods. 45 patients (17 men and 28 women) with AH of 2nd stage were examined. Mean age - 54,5 ± 4,5 years. Clinical examination included assessment of anthropometry, lipid profile, carbohydrate metabolism, HOMA-IR index, serum levels of adiponectin (AN), echocardiography, ultrasound of general carotid artery, including measurement of the intima-media thickness of the common carotid artery (CCA IMT). The patients were divided into two groups: group 1 (n = 25) with DM- 2, group 2 (n = 20) without DM-2.

Results. 49.8% of patients in group 1 were diagnosed with abdominal obesity (AO) of 2nd degree. Left ventricular hypertrophy was found in 68.2% of patients in group 1 and in 43.2% in group 2 ($p < 0.01$). HbA1c level in patients of group 1 was significantly higher than in control group and patients of group 2 ($p<0.001$). Lipid disorders were characterized by hypercholesterolemia (68.4%), hypertriglyceridemia (42.0%), a reduction in HDLP (33.1%), which were more pronounced in patients of group 1 ($p < 0.001$). HOMA-IR index was 2.4 times higher than in patients of group 1 ($p < 0.001$). AN level in group 2 was 1.4 times higher than in control group ($p < 0.05$). Atherosclerotic plaques in the carotid arteries were detected in 49.7% of patients in group 1 ($p < 0.05$). CCA IMT in patients of group 1 was 1.2 times greater than in the control group ($p < 0.01$) and correlated with the level of total cholesterol ($r = 0,34$; $p < 0.01$), HOMA-IR ($r = 0,36$, $p < 0.01$).

Conclusions. AO and IR have decisive importance for the progression of cardiometabolic disorders and formation of EOD in patients with AH and DM-2. The mentioned above changes are caused by dyslipidemia, hypoadiponectinemia and favor the development of left ventricle hypertrophy and progression of atherosclerosis.