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.