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PECULIARITIES OF HEMODYNAMIC DISORDERS IN HYPERTENSIVE PATIENTS DEPENDING ON BODY WEIGHT AND METABOLIC COMORBIDITY

Introduction. The problem of "non-infectious" epidemics is a burning issue in the world today. According to the WHO in 2018, non-communicable diseases (NCDs) dominant among the main reasons for the increase in morbidity and mortality in many countries in the XXI century. The main risk factors for NCDs globally are high blood pressure (BP) (over 18% of all cases), obesity, hyperglycemia and hyperlipidemia. Obese patients, even in the absence of diabetes mellitus (DM), hypertension (AH) and any other heart disease, develop hemodynamic changes leading to morphological and functional changes in the left ventricle (LV), even a small increase in arterial pressure (BP) causes a pronounced increase in its mass (LVM) [1, 2]. From a hemodynamic point of view, it is not clear why the thickening of the LV myocardial walls develops more rapidly and more pronounced in obesity than in isolated hypertension, why this combination often forms a concentric type of LV remodeling. One explanation may be that the

thickness of the LV walls in obesity increases not only in response to increased BP, but also as a side tissue reaction of the myocardium to hormonal and metabolic changes inherent in obesity [3, 4]. In recent years, a number of studies have been conducted that prove the mechanisms of lipotoxic damage to the myocardium in obesity, which changes both the structure of the myocardium and its functional state [5, 6]. The relationship between obesity and structural and functional changes in the heart, including LV hypertrophy, contractile dysfunction, apoptosis, cardiomyocyte fibrosis, has been shown. In conditions of combination of AH with obesity and diabetes, interstitial and perivascular fibrosis develops. Myocardial fibrosis leads to disorders of diastolic and systolic function and is associated with the development of adverse cardiovascular events [7-9]. The early stages of myocardial dysfunction are closely associated with the formation of diastolic disorders due to increased myocardial rigidity

The aim was to study the features of hemodynamic disorders in hypertensive patients depending on body weight and metabolic comorbidity.

Materials and Methods: We examined 340 patients with arterial hypertension: 200 patients with AH with class I - II obesity, 50 patients with AH and normal body weight, 50 patients with AH and overweight, 40 patients with AH, obesity and type 2 diabetes. The test group consisted of 30 healthy individuals. All patients aged 45-55. Anthropometric, biochemical, instrumental, statistical methods were used.

Results: In the course of the study, it was concluded that the degree of disorders of the structural and functional state of the heart and blood vessels increases with the increase of the body weight both in case of double (AH with obesity) and triple (AH, obesity and type 2 diabetes) comorbidity. In the absence of significant differences between overweight and normal weight patients, patients with obesity had significantly higher CIMT, cPWV, and aPWV values ($p < 0.05$ for all indicators). At the same time, the greatest severity of vascular remodeling was in patients with concomitant type 2 diabetes, which significantly distinguished this group from all the others, and the values of such indicators as CIMT, cPWV and aPWV - in particular, from hypertensive patients with obesity without type 2

diabetes ($p < 0.05$ for all indicators). Patients with obesity had a significantly lower level of EDVD than patients with normal body weight and overweight. An increase in body weight from normal to grade II obesity in patients with AH is accompanied by decreases in EDVD, an increase in LVESD and LVM, an increase in LAD and LV wall thickness ($p < 0.05$ for all indicators).

The main indicator of cardiac systolic function - EF - in patients with obesity was lower compared with patients with normal body weight (62.99 ± 3.32 % vs 64.42 ± 2.23 %, $p = 0.004$), despite the fact that only patients with preserved LVEF were included in the study. In the presence of obesity, the increase in pulmonary artery pressure according to Kitabatake distinguished this group from patients with normal body weight and overweight ($p = 0.000$ for both indicators), and the increase in integral diastolic function (E / e′) was significantly higher compared with hypertensive patients with normal body weight ($p < 0.05$), which confirms the progression of diastolic dysfunction in the case of weight gain.

In all groups of hypertensive patients most often diagnosed with a concentric type of remodeling with a predominance of concentric hypertrophy (AH + obesity - 71.5%, AH + normal BMI - 70%, AH + overweight - 62%, AH, obesity + type 2 diabetes - 72, 5%) as a prognostically unfavorable type of remodeling in the absence of significant differences between groups with different body weight ($p > 0.05$). The incidence of the normal type of transmitral blood flow in hypertensive patients with obesity was significantly lower than in hypertensive patients with normal body weight ($p = 0.019$). The distribution of normal and impaired transmitral blood flow in the groups of hypertensive patients was approximately the same, in the vast majority of patients there was the first degree of diastolic dysfunction in the form of impaired relaxation (AH + obesity – 76.5 %, AH + normal BMI – 60 %, AH + overweight – 76%, AH, obesity + type 2 diabetes – 70%). Maximum disorders of the structural and functional state of the heart and blood vessels were found in the group of patients with triple comorbidity ($p < 0.05$ for all indicators), where 7.5 % of patients had a more severe, pseudonormal, type of LV diastolic dysfunction.

Conclusions: The peculiarities of the structural and functional state of the heart and blood vessels in patients with hypertension with different body weight and the role of weight gain and metabolic comorbidity in the development and progression of cardiovascular remodeling are determined.

Keywords: arterial hypertension, obesity, cardiovascular remodeling, metabolic comorbidity.

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