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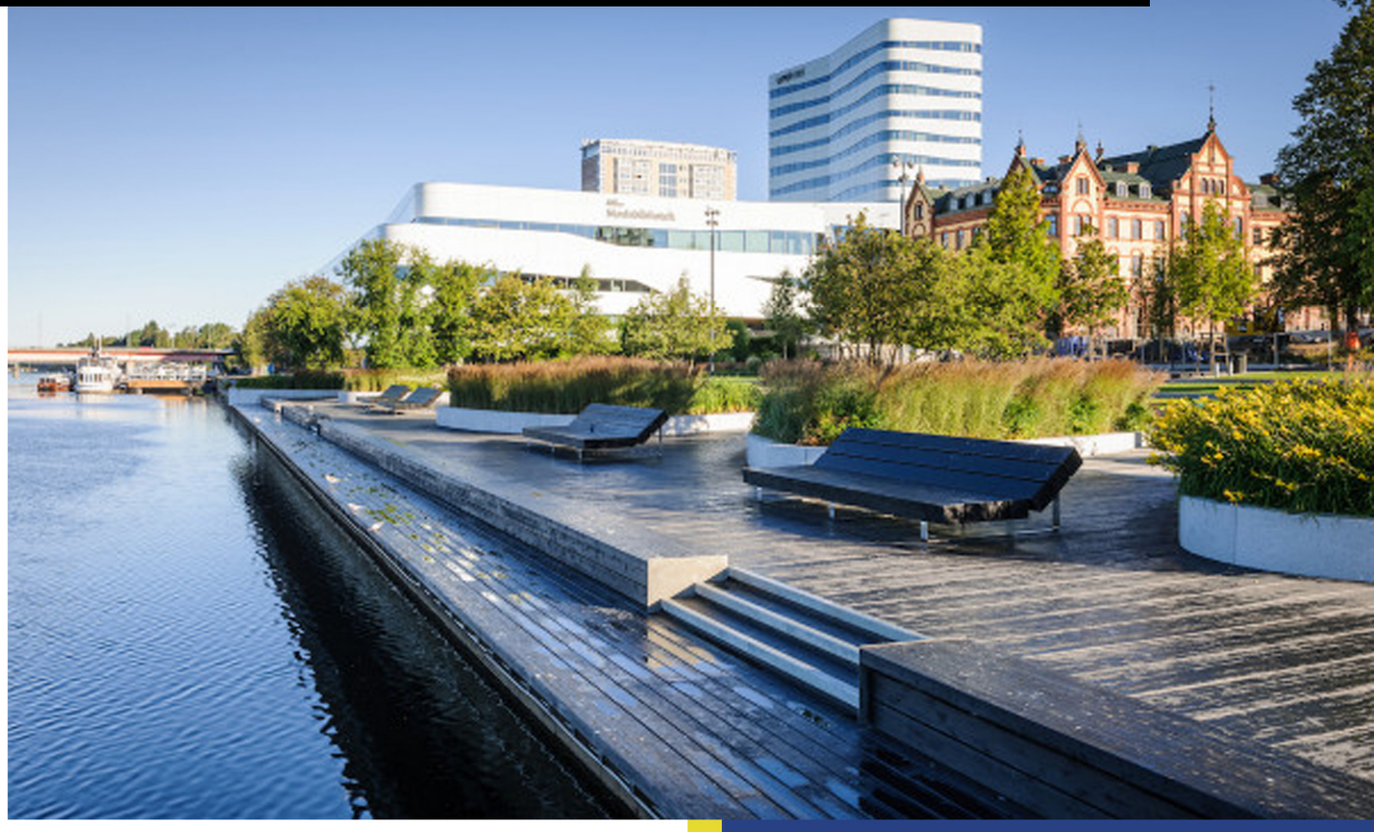

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INDICATORS OF CENTRAL CARDIOHEMODYNAMICS IN PATIENTS WITH ARTERIAL HYPERTENSION WITH POSTINFARCTION CARDIOSCLEROSIS AND DIABETES MELLITUS TYPE 2

Abstract. *Central hemodynamic parameters in 40 patients with arterial hypertension, postinfarction cardiosclerosis and type 2 diabetes were evaluated. Based on the study, it is proved that type 2 diabetes mellitus is a predictor of concentric LV myocardial hypertrophy, which can be considered as a reaction of the heart to prolonged increased load and disruption of myocardial microcirculation, contributes to complex structural and functional reorganization of the heart with a tendency to reduce myocardial contractility. Comorbidity of hypertension, postinfarction cardiosclerosis and type 2 diabetes increases the incidence of signs of left ventricular diastolic dysfunction type 1 (type of relaxation disorder), which leads to a worsening of the prognosis of cardiovascular complications.*

Keywords: *arterial hypertension, postinfarction cardiosclerosis, type 2 diabetes mellitus, left ventricular diastolic dysfunction.*

Numerous epidemiological studies suggest that the risk of coronary complications in patients with type 2 diabetes mellitus (DM2) without coronary heart disease (CHD) is equal to the risk in those who already have clinical manifestations of coronary heart disease. Thus, the frequency of cardiovascular events in patients with diabetes mellitus in men with myocardial infarction (MI) was 50.2%, while in men without diabetes with a history of myocardial infarction - 29% [1]. The risk of coronary death in patients with diabetes and coronary heart disease was almost 3 times higher than in people with coronary heart disease or diabetes mellitus2 alone. According to a meta-analysis of 37 retrospective studies, the risk of coronary death in women associated with diabetes is 50% higher than in men [2].

It is also known that DM2 is one of the main risk factors for chronic coronary syndrome, and more than 40% of patients with acute coronary syndromes have DM2 [3,4].

According to long-term comparative studies in patients with diabetes with acute coronary syndrome compared with patients with normal glucose tolerance test, the incidence of MI is 1.4 times higher than the results of a two-year postinfarction period and 1.8 times higher mortality [5].

In recent years, researchers have become increasingly interested in the prognostic value of myocardial remodeling and diastolic left ventricular dysfunction (DLVD) in patients with hypertension (AH) after MI with and without carbohydrate metabolism disorders [6-10].

The above data determined the feasibility of the study, which aimed to assess the state of central hemodynamics and diastolic function of the left ventricle in patients with hypertension with postinfarction cardiosclerosis and type 2 diabetes.

Materials and methods: a comprehensive retrospective study of 64 patients (men and women in equal numbers of 32 people) with hypertension who suffered from MI, had concomitant diabetes mellitus2 (group 1). The mean age of patients was: 62.26 ± 1.04 years.

The study did not involve patients with severe comorbidities of the respiratory system, digestive system, kidneys, cancer. The comparison group (group 2) consisted of 40 people with coronary heart disease who had MI without diabetes. The average age of patients was 63.35 ± 1.08 years, men were 22 (55%), women - 18 (45%). The groups were represented by age and sex.

Diagnosis of hypertension was established according to the recommendations of the International Society of Hypertension (ISH), 2020. Diagnosis of diabetes mellitus2 was established on the basis of clinical, instrumental, biochemical criteria in accordance with the recommendations of WHO experts, the European Society of Endocrinologists.

The diagnosis of MI was determined according to the criteria of the European Society of Cardiology / American College of Cardiology / American Heart Association / World Heart Federation (ESC / ACCF / AHA / WHF) (2012) (Thygesen K. et al., 2012).

All patients were examined clinically and instrumentally. Echocardiographic examinations were performed according to the standard method on an ultrasound device RADMIR (Ultima PRO 30) (Kharkiv, Ukraine). The following LV parameters were determined in the M-mode: final diastolic size (FDS), cm; final systolic size (FSS), cm; the thickness of the posterior wall of the LV (TLVPW), cm; the thickness of the interventricular septum (IVST), see final diastolic volume (FDV) and final systolic volume (FSV), ml of LV was calculated by the method of Simpson (1991), and then calculated the fraction of LV ejection (LVEF), %.

Left Ventricular Mass (LVM) has been calculated by the formula by R. Devereux et al.:

$$1.04x[(a+b+c)^3 – c^3] – 13.6;$$

where a – IVST; b – TLVPW; c – EDD.

The left atrium (LA), cm, has also been determined by the size between the posterior wall of LA and the posterior aortic wall on a parasternal image along the long axis.

Statistical processing of the obtained data was carried out using the Statistica 13.0 statistical software package (StatSoft Inc, USA), Microsoft Windows 7. Quantitative characteristics with normal distribution have been shown as the mean ± standard error of the mean (M±m); the Student's t test has been used to compare the means of the two samples. For all types of analysis, the differences have been considered statistically significant at $p<0.05$.

Results and its discussion. When comparing the obtained cardiohemodynamic parameters in comparative groups of patients with AH, postinfarction cardiosclerosis (PICS) in the presence and absence of type 2 diabetes mellitus (NIDD), it was determined that the relative interventricular septum thickness (RIVST), the relative thickness of the left ventricle posterior wall (RTLVPW), the left ventricular myocardium mass index (LVMMI), and the thickness index of left ventricular myocardium wall (TILVMW) ($p<0,05$) significantly differed in the groups of examined patients (Table 1). Thus, TLVPW was 8% higher, IVST–by 9.5%, RIVST–by 24%, RTLVPW–by 30%, LVMMI–by 11%, and TILVMW–by 15%.

Table 1

Results of cardiohemodynamic parameters in the examined patients

Parameters	Study group of patients, n=68	Experimental group, n=40	P
SBP, mm Hg	159.4±3.0	158.2±3.1	>0.05
DBP, mm Hg	92.4±1.9	91.6±2.1	>0.05
HR, bpm	76.8±2.1	76.4±2.3	>0.05
Pulse, bpm	76.6±1.9	76.3±2.3	>0.05
EDV, ml	180.36±3.4	178.26±3.1	>0.05
ESV, ml	82.8±1.7	81.4±1.8	>0.05
EDD, cm	6.54±0.09	6.41±0.11	>0.05
ESD, cm	3.16±0.07	3.11±0.08	>0.05
LVEF, %	47.63±0.8	48.12±0.9	>0.05
RIVST, cm	0.67±0.05	0.51±0.06	>0.05
RTLVPW, cm	0.54±0.03	0.38±0.04	>0.05
TLVPW, cm	1.52±0.02	1.40±0.02	>0.05
IVST, cm	1.47±0.03	1.33±0.01	>0.05
LA, cm	4.01±0.07	3.95±0.12	>0.05
Aorta, cm	3.31±0.05	3.29±0.06	>0.05
LVM, g	263.28±6.9	251.35±6.7	>0.05
TILVMW	0.53±0.02	0.45±0.01	>0.05
LVMMI, g/m^2	142.68±4.69	127.25±5.01	>0.05

SBP–systolic blood pressure; DBP–diastolic blood pressure; HR–heart rate.

The data obtained suggest that type 2 diabetes mellitus (NIDD) is a predictor of the development of concentric LV myocardial hypertrophy in patients with AH and postinfarction cardiosclerosis (PICS), which is a heart response to prolonged exercise, as well as a violation of the microcirculation processes in the myocardium, which contributes to the complex structural and functional restructuring of the heart with the occurrence of predominantly concentric remodeling and concentric LV hypertrophy with a tendency to decrease the myocardial contractility. This understanding coincides with the opinions of other researchers [11-12]. A significant number of patients with type 2 diabetes mellitus (NIDD) have LV hypertrophy, which dictates the need to study the prevalence of LV diastolic dysfunction in this category of patients.

The analysis of indicators of diastolic function of LV myocardium in patients with AH, PICS, and type 2 diabetes mellitus (NIDD) has also been carried out. It was found that in such patients, there are differences in the indicators of LV diastolic function depending on the presence of type 2 diabetes mellitus (NIDD) (Table 2).

Table 2

Indicators of the myocardium diastolic function in the examined patients

Parameters	Study group of patients, n=68	Experimental group, n=40	P
E, mm/s	63.47±2.8	57.92±2.6	<0.05
A, mm/s	71.14±1.6	62.28±1.7	<0.05
IVRT, ms	107.6±2.1	103.9±1.9	<0.05
DT, ms	234.5±9.8	221.4±8.7	<0.05
E/A, units	0.89±0.06	0.93±0.05	<0.05

Thus, the maximum early filling rate (E) in the examined patients with concomitant type 2 diabetes mellitus (NIDD) was significantly higher ($p<0.05$). The maximum late atrial filling rate (A) was also significantly higher in case of diabetes mellitus. The ratio of E/A peaks is important in determining LV diastolic dysfunction (LVDD). In patients with AH with PICS and type 2 diabetes mellitus (NIDD), this indicator was 0.89±0.05 and without type 2 diabetes mellitus (NIDD)– 0.93±0.04 ($p<0.05$), which indicates slow relaxation of the LV.

According to the results of this fragment of the study, it was found that a low E/A ratio took place in patients with comorbid pathology (a type of relaxation disorder), which in combination with normal values of LVEF, EDD, ESD, EDV, ESV indicates the advantage of LVDD in the hypertrophic type in this category of patients. The comorbidity of AH, PICS, and type 2 diabetes mellitus (NIDD) increases the incidence of type 1 LVDD symptoms, which further contributes to a worsening prognosis of cardiovascular complications.

Conclusions:

1. It has been established that in patients with AH, PICS, and type 2 diabetes mellitus (NIDD), it is type 2 diabetes mellitus (NIDD) that is a predictor of the development of concentric LV myocardial hypertrophy, which can be considered as a heart reaction to a prolonged increased load and disturbances in the processes of microcirculation in the myocardium, contributes to the complex structural and functional restructuring of the heart with a tendency to decrease the myocardium contractility.

2. The comorbidity of AH, PICS, and type 2 diabetes mellitus (NIDD) increases the incidence of type 1 LVDD symptoms (a type of relaxation disorder), which leads to a worsening prognosis for the development of cardiovascular complications.

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