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STUDYING THE CHARACTERISTICS OF STRUCTURAL AND GEOMETRIC PARAMETERS OF THE LEFT VENTRICLE IN PATIENTS WITH CHRONIC KIDNEY DISEASE AND ARTERIAL HYPERTENSION IN THE BACKGROUND OF OBESITY

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Abstract:
The results of a survey of patients with CKD and metabolic syndrome indicate that the identified disorders of diastolic renal function worsened as the functional state of the kidneys worsened and the metabolic syndrome developed. The identified data support the consideration of kidney function parameters as one of the earliest markers of increased risk of cardiovascular complications in metabolic syndrome, as well as in determining treatment tactics for this category of patients.

Keywords: chronic kidney disease, degree of obesity, arterial hypertension, left ventricular remodeling

Currently, all over the world there is a rapid increase in the population with overweight, obesity and type 2 diabetes. Chronic kidney disease (CKD), like cardiovascular complications, is the most common, severe and prognostically unfavorable complication of metabolic syndrome. The results of large population studies indicate that in individuals with kidney disease and especially those with renal failure, the risk of cardiovascular disease with the formation of cardiorenal syndrome is much higher than in the general population [1].

The concept of cardiorenal syndrome implies a commonality of mechanisms for the development of cardiovascular and renal complications [2–5]. The contribution of renal pathology to the development of cardiorenal syndrome has been most studied in T2DM [6]. However, the problem of the formation of cardiorenal syndrome in obesity remains not fully resolved. Thus, at the moment there is no doubt about the influence of obesity on the course of nephropathies of various origins and the processes of remodeling of the left ventricular myocardium (LVM) [7, 8].

The relationship between the development of early stages of CKD, myocardial remodeling and obesity requires clarification. The significance of the hormonal activity of adipose tissue and the role of metabolic and hemodynamic disorders associated with obesity in the formation of cardiorenal syndrome in patients with CKD have not been clarified.

This condition can last for decades, gradually worsening and developing into obvious pathology, manifested by clinical markers of chronic renal failure and decompensation of renal function. Therefore, it is especially important for clinicians to identify the initial period of renal dysfunction, when aggressive tactics of prescribing medications can slow down the process of destruction of the renal glomerulus and change the future fate of the patient [54].

GOAL OF THE WORK

The purpose of this study was to study the relationship between left ventricular myocardial (LV) remodeling processes in patients with hypertension and the development of CKD in various obesity phenotypes.

MATERIALS AND METHODS

A total of 96 patients with hypertension were selected according to the inclusion criteria (stage I–II hypertension, grades 1–2, absence of renal pathology (history of kidney disease, structural changes in the parenchyma and vessels of the kidneys during ultrasound examination, changes in urinary sediment and urine density, proteinuria, decreased GFR less than 60 ml/min/1.73 m²) at the age of 46-58 years (average age - 50.34±4.6 years).

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Systolic blood pressure (SBP) was 140-180 mmHg. Art., diastolic (DBP) - 90-110 mm Hg. Art., lipid levels in the blood serum: total cholesterol (TC) ≥5.4 mmol/l, triglycerides (TG) ≥ 1.7 mmol/l, low-density lipoproteins (LDL) ≥3 mmol/l, atherogenic coefficient (AC) ≥ 3.5. Abdominal type of obesity - ratio of waist circumference (WC) to hip circumference (HC) - WC/HC ≥0.80; body mass index (BMI) ≥ 30 kg/m2, hyperinsulinemia (IRI) ≥ 18 mk/ml, insulin resistance (IR) as the ratio of glucose to basal insulin ≤ 6. A metabolically healthy obesity phenotype was considered the presence of obesity without hypertension, dyslipidemia (DLP) , hyperglycemia and IGT.

Exclusion criteria for the study were: secondary forms of obesity and hypertension, stage III hypertension, diabetes, autoimmune diseases, bronchial asthma, cancer, neurological pathology, mental disorders, cardiovascular pathology (IHD, chronic heart failure (CHF), heart rhythm disturbances), occlusive peripheral artery disease, severe liver dysfunction (increased transaminases more than two reference values), pregnancy, lactation, 13 constant use of antihypertensive drugs, statins, allergic reactions to taking ACE inhibitors, refusal of the study.

To assess the functional state of the kidneys, we examined the level of serum creatinine (CC), glomerular filtration rate (GFR), protein excretion from 30 to 300 mg in daily urine was considered microalbuminuria (MA).

All study patients (n = 96) were divided into 3 study groups: group 1 – patients with hypertension without obesity (BMI less than 30 kg/m²) (n = 32); 2nd group of patients with hypertension and obesity (BMI ≥ 30 kg/m²), but without metabolic changes (i.e. without IGT, hyperglycemia, DLP) - (n = 33) and 3rd group of patients with metabolically complicated obesity, who, in addition to obesity (BMI ≥30 kg/m²) and hypertension, had impaired glucose tolerance (IGT), dyslipidemia (n = 32)

Blood pressure (BP) monitoring was carried out by fully automatic measurement systems for 24 hours at intervals of 15 minutes during wakefulness and 30 minutes at night. The average parameters of SBP and DBP per day, daytime, nighttime were determined; time index (TI), variability in different periods of the day, degree of night-time decrease (SI) in SBP and DBP.

An echocardiographic study (EchoCG) was performed on a Mindray device (China) using the transthoracic method in the supine position and on the left side in M - and B - modes in accordance with the recommendations of the American Association of Echocardiography (ASE). At the same time, we assessed: end-diastolic and end-systolic dimensions of the LV (EDD and ESD), thickness of the posterior wall of the LV (PLW) and interventricular septum (IVS), size of the left atrium (LA) [9, 12]. The value of mean hemodynamic blood pressure (BPav.) was calculated using the HiKem formula. The indicators were calculated - end systolic and diastolic volumes (ESV and EDV), LV ejection fraction (EF), stroke volume (SV) - as the difference between EDV and ESV. LV myocardial mass (LVMM) was calculated using the formula of Devereux R.B. [204]; LV myocardial mass index (LVMI) - as the ratio of LVMI to body area; LVMI ≥134 in men and ≥110 g/m2 in women was taken as a criterion for LV hypertrophy. The relative LV wall thickness (LVW) was calculated..

$$АДср = \frac{АДсис - АДдиас}{3} + АДсис, \quad мм\ рт.ст.$$

$$ОТС = \frac{ТМЖП + ТЗСЛЖ}{КДР}$$

$$ММЛЖ = 1{,}04 \cdot \left[(КДРлж + ТМЖП + ЗСЛЖ)^3 - КДРлж^3\right] - 13{,}6, \quad г$$

$$ИММЛЖ = \frac{ММЛЖ}{S\text{тела}}, \quad г/см^2$$

$$S\text{тела} = М^{0{,}425} \cdot P^{0{,}725} \cdot 0{,}007284, \quad г/см^2$$

Diastolic function was assessed using pulsed Doppler echocardiography. The maximum flow rate of the late filling period (A, cm × s-1), the early filling rate (E, cm × s-1), and the E/A ratio were determined.

The research results were processed using parametric and nonparametric statistics using the Microsoft Excel statistical software package

RESEARCH RESULTS AND DISCUSSION

The general characteristics of the patients and the distribution of cardiometabolic risk factors are presented in Table 1**.**

Table 1.
General characteristics of patients and distribution of cardiometabolic risk factors

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Index	1- group (n=32)	2-group (n=33)	3- group (n=31)
Age, years	27,7 ±4,5	30,3±5,1	36,8±7,4**∞
Men, abs (%)	10 (58,8%)	9 (50%)	11 (57,8%)
Body mass index, kg/m2	21,4±2,2	28,4±2,7**	31,8±4,8 ***∞
SBP, mmHg Art.	114,8±12,4	138,3±11,9**	146,1±10,1***∞
DBP, mmHg Art.	76,1±7,5	89,3±5,4**	98,1±6,8***∞
Blood glucose, mmol/l	4,96±0,4	5,3±0,6	5,7±0,6**
IR, HOMA-IR	1,7±0,4	2,4±0,6*	4,17±1,16***∞
BHC, mmol/l	4,4±0,6	5,36±0,6**	6,34±1,1***∞
LDL cholesterol, mmol/l	2,2±0,6	2,6±0,7	3,82±1,1***∞
HDL cholesterol, mmol/l	2,2±0,5	1,98±0,5	1,54±0,5**
TG, mmol/l	0,8±0,3	0,99±0,2	1,6±0,8**∞
Leptin, ng/ml	10,9±4,5	25,7±7,2***	33,8±9,4*** ∞
Adiponectin, µg/ml	9,1±2,2	7,5±2,4*	6,49±2,92**
CC, mmol/l	133,26±10,73	149,15±8,91**	165,3±11,24***∞
GFR ml/min/m2	76,61±11,26	62,27±12,44*	51,84±12,64**∞
MAU, g/l	21,12±7,85	33,72±7,41**	42,56±8,11***∞
Al/cr, mg/mol	3,0±0,01	3,9±0,01*	4,5±0,01**∞

Note: **(p<0.01), ***(p<0.005) in relation to the data of the 1st group; ∞ (p<0.05) between the 2nd and 3rd study groups.

According to WHO criteria, all patients had a moderate degree of increase in blood pressure, where SBP averaged 114.8 ± 12.4 mm Hg for group I. Art. and DBP - 76.1±7.5 mm Hg. Art., in the second group - 138.3±11.9 and 89.3±5.4 mmHg, respectively. Art. (p<0.01), and in the 3rd group 146.1±10.1 and 98.1±6.8 mmHg. respectively (p<0.005) in relation to the data of the 1st study group.

Elevated levels of serum creatinine were significantly more common in obese hypertensive patients. Daily albumin excretion was also higher in patients of the second and third groups, but was significant only in hypertension in combination with a metabolically unhealthy obesity phenotype (p<0.005).

Analysis of the functional state of the kidneys showed that patients in the 2nd and 3rd study groups differed in the level of MAU, blood creatinine, Al/creatinine ratio and, accordingly, the level of GFR (Table 1). In the 2nd group of patients, there were significantly high levels of MAU (p<0.05), AL/cr (p<0.05) and blood creatinine levels by 16.0% (p<0.01), a decrease in GFR by 20 .6% (p<0.01). In the 3rd group

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of patients, it was characterized by more pronounced disorders of the functional state of the kidneys: there were significantly high levels of MAU (p <0.01), AL/cr (p <0.005) and blood creatinine levels in 25.6% (p <0.005), a decrease in GFR by 29.9% (p <0.005) compared to the 1st study group.

The results of the analysis of the structural and functional state of the myocardium by echocardiography showed that in group 2 patients there was a significant difference in the parameters of the ventricular ventricle, LVAD, and CSR compared with the data of patients of group 1. It was found that differences in the structural parameters of EchoCG, because TMZH, TZSLZh of the 1st and 2nd study groups were 15.2% and 15.9%, respectively (p <0.05). According to the differences in these parameters, there were also significant differences in LVMM between these groups of 15.6%, which was also statistically significant (p <0.05).

Table2.
Indicators of structural and geometric parameters of the left ventricle among patients of the study groups

Index	1-group (n=32)	2- group (n=33)	3- group (n=31)
LP, cm	3,68±0,021	4,04±0,028**	4,15±0,028**8
TZSLZh, cm	1,09±0,01	1,14±0,012*	1,16±0,032**
TMZHP, cm	1,11±0,012	1,17±0,013*	1,20±0,033**
CDR, cm	5,34±0,4	5,61±0,3	5,99±0,34**
DAC, cm	3,66±0,2	3,82±0,14	4,26±0,46**
EDV, ml	133,3±10,55	141,88±11,96	152,79±12,75**
ESR, ml	57,3±3,55	68,6±5,34	76,8±5,22**
OTS, st unit	0,39±0,008	0,41±0,007	0,43±0,007*
LVMM, g	233,6±3,92	256,72±3,5*	278,37±4,12**
LVMI, g/m²	120,35±4,3	128,35±3,3	132,76±3,44**
PV, %	56,98±1,62	54,83±2,53	52,01±2,33*
IVRT, mc	67,65±3,75	78,65±1,75**	86,65±1,75*** ∞
RE, m/s	0,50±0,012	0,46±0,012*	0,41±0,012**
RA, m/s	0,41±0,020	0,44±0,020	0,48±0,020*
E/A	1,01±0,74	1,04±0,74	1,11±0,74*
Heart rate,beats per minute	79,66±5,02	85,92±9,14	86,26±8,74**

Note * p<0.05; ** p<0.01; *** p<0.005 relative to 1 subgroup

Analysis of echocardiographic parameters characterizing the structural and geometric properties of the LV showed that in patients with CKD and obesity, statistically comparable data were observed for the LA, PVSD, LVAD and ESR (p>0.01) and ESR (p<0.05) between 2 and 3 group of the study in contrast to the data of the 1st group. When assessing the dependence of LVH on the severity of MS in patients of the 3rd group, significant differences in LVMM were determined (278.37±4.12 versus 233.6±3.92 (p<0.01)), which was confirmed by a significant difference in LVMI (132 .76±3.44 versus 120.35±4.3) (p<0.01). There was also a significant difference in the relative wall thickness of the LV myocardium (RTW) 0.43±0.007 versus 0.39±0.008 (p<0.05).

However, the results of the analysis of the hemodynamic parameters of the LV using the echocardiography method showed comparable data on EDR, EDV, ESV and, accordingly, SV, MOC and LVEF in patients of the 2nd and 3rd study groups (p>0.05).

Thus, regardless of the severity of MS, patients with CKD and hypertension are characterized by impaired LV remodeling, increased LVAD, LVTD and, accordingly, LVMM and LVMI. These structural changes in the LV in

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patients with CKD and various obesity phenotypes were aggravated as the disturbances in the metabolic profile of patients increased.

It was noted that the maximum rate of early diastolic filling of the LV (PE) in patients of the 2nd group was significantly different from the indicators of the 1st group of hypertension and CKD without obesity ($p < 0.05$). The flow rate into atrial systole (AS) increased slightly and the E/A ratio decreased, but the difference with group 1 also did not reach statistical significance ($p > 0.05$). However, based on the dynamics of the increase in isovolumic relaxation time (IVRT), we can talk about the formation of LV diastolic dysfunction in them ($p < 0.01$).

It was noteworthy that as the signs of metabolic syndrome increased in patients with CKD and hypertension (group 3), the isovolumic relaxation time (IVRT) increased ($p < 0.005$), in contrast to the data of group 1. At the same time, a significant decrease in PE was detected ($p<0.005$ in the 3rd group of patients with CKD in the context of metabolically unhealthy obesity. The dynamics of parameters characterizing LV diastolic function (RA ($p<0.005$), PE/RA ($p<0.005$)) was statistically significant.

Thus, the results of examination of patients with CKD and metabolic syndrome indicate that even with preserved renal function, they may have impaired LV diastolic function. The identified disorders of diastolic renal function worsened as the functional state of the kidneys worsened and metabolic syndrome developed.The identified data support the consideration of renal function parameters as one of the earliest markers of increased risk of cardiovascular complications in hypertension and in determining treatment tactics for this category of patients.

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