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Kidney Damage in Patients with Chronic Cardiac Insufficiency and Obesity

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Abstract
Chronic heart failure (CHF) and obesity are widespread in the population and often coexist, increasing the risk of complications in this category of patients. In patients with heart failure (HF), the prevalence of obesity is 32-49%. At the same time, most of the patients are pre-represented by persons with HF and a preserved LV ejection fraction. Obesity is an independent risk factor for heart failure. It is shown that as the body mass index increases for every 1 kg/m^2, the risk of developing HF increases by 7% in women and 5% in men. Chronic heart failure (CHF) and obesity are widespread in the population and often coexist, increasing the risk of complications in this category of patients. In patients with heart failure (CHF), the prevalence of obesity is 32-49%. At the same time, most of the patients are represented by persons with HF and a preserved LV ejection fraction. Obesity is an independent risk factor for heart failure. It has been shown that as the body mass index (BMI) increases for every 1 kg/m^2, the risk of developing HF increases by 7% in women and by 5% in men. Obesity has become a worldwide epidemic, and its prevalence is projected to increase by 40% in the next decade. The growing prevalence of obesity has an impact on the risk of developing diabetes mellitus, cardiovascular diseases, as well as chronic kidney disease (CKD). A high body mass index is one of the most significant risk factors for CKD. In obese individuals, compensatory hyperfiltration develops in response to an increase in the metabolic needs of overweight. An increase in intraclubular pressure can lead to kidney damage and an increased risk of developing CKD in the long term. In recent years, the incidence of glomerulopathy associated with obesity has increased 10-fold. In addition, obesity has been shown to be a risk factor for nephrolithiasis and a number of malignant neoplasms, including kidney cancer.

Key words: kidney function; chronic kidney disease; chronic heart failure; obesity; cardiovascular risk; leptin; adiponectin; insulin resistance.

Introduction

The prevalence of chronic heart failure (CHF) continues to increase, despite significant progress in the treatment of this disease. Obesity makes a significant contribution to the increase in the incidence and progression of CHF,the frequency of which is growing rapidly, including in the Uzbekistan. There is a direct relationship between obesity and the development of CHF. In patients with HF, the incidence of obesity is 32-49%. The presence of CHF and obesity is associated with the development of renal dysfunctions. A decrease in the functional state of the kidneys in patients with HF significantly worsens the prognosis of patient survival and is a predictor of an unfavorable clinical outcome. In turn, obesity is an in dependent risk factor for the development and progression of chronic kidney disease (CKD). Recent studies have proven a close relationship between obesity rates and the development/progression of CKD.

The high incidence of CKD in patients with CHF and obesity suggests the unity of the mechanisms of development of renal dysfunction in this comorbid pathology. Among these mechanisms, adipokines produced by adipose tissue cells occupy a special place.

The aim of the study was to assess the functional state of the kidneys and cardio—vascular risk in relation to the level of leptin and adiponectin in patients with CHF and obesity.

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Materials And Methods

The study included 116 patients with CHF of ischemic genesis of functional classes I – III (FC) aged 45-65 years with a history of myocardial infarction (6 to 12 months old). Depending on BMI, patients were divided into three groups comparable in functional class (FC) of CHF, age, gender, smoking frequency, AH experience, office blood pressure and heart rate: Group I (n = 34) is represented by persons with normal body weight (BMI = 18.5 – 24.9 kg/m^2), Group II (n = 40) — patients with CHF and overweight (BMI = 25 – 29.9 kg/m^2), group III (n = 42) — patients with CHF in combination with obesity of 1 – 2 degrees (BMI = 30 – 39.9 kg / m^2). Most patients had a preserved or intermediate ejection fraction (LV) of the left ventricle. CKD was determined in the presence of any markers of kidney damage persisting for 3 months or more.

The study did not include patients with primary pathology of the kidneys and urinary tract, acute coronary syndrome and acute cerebrovascular accident less than 6 months old, hemodynamically significant heart defects and rhythm disorders, endocrine, autoimmune, oncological pathologies, acute inflammatory diseases, type 1 and 2 diabetes mellitus, grade III obesity, chronic kidney disease

above stage 3b, any other diseases that could affect the results of the study. All patients received basic therapy of heart failure, the average dosages of drugs did not significantly differ.

The examination included an assessment of the general condition, determination of FC CHF by the test with 6-minute walking, measurement of blood pressure on both hands according to the standard method in the patient's sitting position, calculation of heart rate (HR). In addition, to diagnose and objectify the severity of CHF, the level of N-terminal cerebral natriuretic peptide (NT-proBNP) was determined by enzyme immunoassay. All subjects underwent anthropometry with the measurement of height, weight, waist circumference (FROM) and hip circumference (ABOUT), followed by the calculation of the ratio FROM/ABOUT and BMI. The body composition was studied by the bioelectric impedance method on an Omron monitor BF-508 — the percentage of subcutaneous and visceral fat in the body was analyzed. Abdominal obesity meant FROM ≥ 102 cm in men and FROM ≥ 88 cm in women, under visceral — excess visceral fat in the body ≥ 9%. The visceral fat index (VAI) was calculated.

The functional state of the kidneys was assessed by determining the excretion of albumin in urine — albuminuria (AU) by the ratio of albumin / creatinine in the morning portion of urine, blood creatinine with the calculation of glomerular filtration rate (GFR) according to the formula CKD-EPI. The stage of CKD was determined according to National recommendations on the level of GFR and albuminuria. The function of the proximal renal tubules was judged by concentration β2-microglobulins in urine determined by enzyme immunoassay (ELISA). The reference values were the level of β2- microglobulins in a spontaneous daily portion urine < 0.3 mg/l.

The combined risk of cardiovascular complications and CKD progression depending on GFR and AU was analyzed in all patients.

Table 1
Clinical and demographic characteristics of the patients included in the study (Me [Q1;Q2])

Indicator	I group CHF + normal body weight	Group II CHF + overweight	Group III CHF + obesity
Number of patients, n	34	40	42
Men/Women, (%)	65 / 35	60 / 40	62 / 38
Age,	58,0 [52,0; 64,0]	58,0 [53,0; 65,0]	57,0 [52,0; 63,0]
Presence of hypertension, %	85.3	95	97.6
Duration of AH, years	14 [7; 16]	15 [9; 17]	16 [8; 21]
BMI, kg/m^2	24,7 [21,3; 24,9]	27,5* [25,7; 29,3]	34,6*[31,9; 38,2]
Waist circumference, cm	86,0 [75; 86]	96,0*[84; 101]	106,0* [99; 111]
Waist circumference/Hip	0,82 [0,77; 0,86]	0,92 [0,86; 0,97]	0,96 [0,85; 0,99]

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circumference, cu.			
FC I **CHD**, %	29	25	9
FC II **CHD**, %	53	57	67
FC III **CHD**, %	18	18	24
FE,%	49,6 [44; 55]	49,7 [44; 56]	48,6 [43; 54]
T & M	362,3 [345; 378]	360,5 [336; 384]	358,3 [337; 380]
NT-proBNP, pg/ml	1337,2 [1234,5; 1432,6]	1340,7 [1245,3; 1428,7]	1351,3 [1249,5; 1434,1]
Number of patients with abdominal obesity, %	0	70*	78.6*
Subcutaneous fat, %	25,8 [24,2; 29,5]	36,7* [27,5; 43,7]	42,4*[39,3; 51,4]
Visceral fat, %	8,9 [6,0; 12,0]	12,0* [8,0; 18,0]	22,0*[12,0; 26,0]
VAI index units. #	2,8 [1,92; 3,14]	3,38 [1,89; 3,16]*,	3,93 [2,21; 4,15]*

Note: * — reliability of differences in comparison with group I at $p < 0.05$; # — reliability of differences between group II and III at $p < 0.05$. Abbreviations: AH — arterial hypertension, BMI — body mass index, OT — waist circumference, OT/ ABOUT — the ratio of waist circumference to hip circumference.

The level of leptin and adiponectin in the blood serum was determined by ELISA (Leptin kits, Diagnostics Biochem, Canada and Adiponectin, Mediagnost, GmbH, Germany). The reference values for leptin were 3.7 – 11.1 ng/ ml (for women — ≤ 27.6 ng/ml, for men — ≤ 13.8 ng/ml), for adiponectin — 8.2 – 19.1 ng/ml. To assess insulin resistance, the concentration of basal insulin was measured and the calculated HOMA-IR index was used. HOMA index equal to or greater than 2.27 was considered insulin resistance. Statistical analysis of the results was carried out using the package of built -in functions of the program “Microsoft Excel 2010” and

the program “STATISTICA 12.0” (StatSoft, Inc.). The data are presented in the form of Me [Q1;Q2], where Me is the median, [Q1;Q2] is the 25th and 75th percentiles, respectively, for qualitative values — frequency of occurrence (%).

Differences in average values and correlations were considered reliable at a significance level of $p < 0.05$. Independent samples were compared using the Kraskel–Wallis criterion. In the case of dichotomous indicators , statistical significance The differences in the shares were estimated usingthe exact Fisher method. To evaluate the relationship statistics, a correlation analysis was performed by Spearman. The study was conducted in accordance with the ethical principles set out in the Helsinki Declaration of the World Medical Organization

Association (2008), the Tripartite Agreement on Good Clinical Practice (ICH

GCP), the Constitution of the Russian Federation, Federal Law Of the Russian Federation No. 323-FZ "On the basics of public health protection in the Russian Federation" dated November 21, 2011. The clinical trial was approved by the Regional Ethics Committee. Informed consent was obtained from the study participants prior to the start of any research procedures.

Results

Among the examined patients, BMI naturally increased from group I to group III. Abdominal obesity was significantly more common among overweight and obese patients compared to those with normal body weight (78.6, 70 and 0%). A high percentage of visceral obesity was revealed among people with concomitant obesity/overweight, as well as in patients with normal body weight: 17.6% among people with normal BMI, 70% among overweight people and 100% among obese people (differences between I and II, I and III, II and III groups are reliable). Dysfunction of the adipose tissue the tissue assessed by the VAI indicator was significantly more pronounced among patients with CHF and obesity, overweight in comparison with those with "isolated" CHF.

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When analyzing the functional state of the kidneys, it is noteworthy that with an increase in BMI, the frequency of detection of persons with more severe stages of CKD significantly increased.

With the addition of obesity to CHF, a pronounced increase in AU was revealed. The differences in this indicator have reached the level of statistical significance between groups I and III. It should be noted a high percentage of individuals with AU/ Cr.> 30 mg/g in all the study groups. However, it was significantly lower in group I compared to groups II and III (71 vs 92.5 and 98%, respectively).

There were significant correlations between the severity of visceral obesity and AU (r = 0.58 and r = 0.42), GFR (r = -0.42 and r = -0.38), as well as VAI with the level of AU (r = 0.42 and r = 0.40), GFR (r = -0.38 and r = -0.37) in patients with CHF

in combination with obesity and overweight , respectively.

There was a significant increase in the level of β2- microglobulins in urine as obesity joined CHF. At the same time, reliable relationships between the concentration were established β2-microglobulins in urine and leptin levels (r = 0.62) and adiponectin (r = -0.88), HOMA-IR (r = 0.66) in the group of patients with CHF and obesity.

Table 2
Functional state of the kidneys in the patients included in the study (Me [Q1;Q2])

Indicator	Group I CHF + normal body weight (n = 34)	group II CHF + overweight (n = 40)	Group III CHF + obesity (n = 42)
GFR, mL / min / 1.73 m^2	73,2 [62,1; 86,3]	68,5 [54,9; 79,2]	61,3*[46,2; 67,1]
CKD C1, %	23.5	12.5	4.7*
CKD C2, %	41.2	55	35.7
CKD C3a,%	23.5	25	42.9*
CKD C3**b** %	0	7.5*	16.7*
β2-microglobulin, mcg/ml	0.29 [0,12; 0,50]	0,68 [0,27; 0,98]	1,12 * [0,84; 1,32]

When assessing the combined risk of CKD progression and the development of cardiovascular complications, depending on the degree of GFR reduction and the severity of albuminuria, a significant increase in high and very high risk was noted among patients with CHF and obesity. In addition, attention is drawn to the presence of significant differences not only in patients with isolated CHF, but also between patients with CHF and overweight and CHF in combination with obesity in terms of the frequency of occurrence of prognostically unfavorable categories of combined risk.

When studying the concentration of laboratory markers of visceral obesity, a significant decrease in the concentration of adiponectin and an increase in the level of leptin from group I to group III were found.

Correlation analysis showed the presence of statistically significant relationships between the concentration of leptin and GFR (r = -0.52), AU(r = 0.36), the c oncentration of adiponectin and GFR (r = 0.38), adiponectin and AU (r = -0.32) among people with CHF and obesity. Similar associations are observed in patients with CHF and overweight. An increase in body weight from group I to group III leads to negative changes in indicators characterizing the severity of insulin resistance.

There was a statistically significant increase in the degree and frequency of occurrence of insulin resistance in the group of patients with CHF with overweight and obesity. At the same time, attention is drawn to the increase in the HOMA index and the prevalence of IR to 17.6% in the group of patients with CHF and normal body weight. The progression of IR as BMI increases is also reflected in statistically significant correlations between the level of visceral fat in the body and HOMA-IR in patients with CHF and obesity and overweight (r = 0.76 and r = 0.72, respectively). In addition, among overweight and obese patients, there were

significant associations between the severity of AU and HOMA-IR (r = 0.28 and r = 0.34, respectively), GFR and HOMA-IR (r = -0.30 and r = -0.29, respectively).

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Discussion

The identification of a high percentage of patients with abdominal and visceral obesity in groups of individuals with normal and excessive BMI dictates the need to assess not only BMI in the diagnosis of obesity, but also the values of OT,

the ratio of OT / OB, as well as the content of visceral fat in the body. The results of the study confirm the important pathogenetic role of obesity in the development and progression of kidney dysfunction in patients with CHF. The addition of obesity to CHF leads to a significant decrease in the level of GFR in comparison with a group of patients with CHF without concomitant pathology. The data obtained are consistent with many studies that show that the presence of obesity is associated with an increase in the probability of a decreasein the calculated GFR < 60 ml/min/ 1.73 m^2 by 30%. As the BMI increases, the AU level increases statistically significantly (more than twofold when comparing patients with "isolated" CHF and patients with CHF and obesity). A significant increase in the number of patients with a clinically significant decrease in GFR (<60 ml/min/1.73

m^2) and an increase in AU indicates a progressive deterioration in the functional state of the kidneys in combination with CHF and obesity. A number of studies have noted that the incidence of albuminuria in patients with CHF significantly

exceeds its prevalence in the general population, in patients with diabetes and hypertension, amounting to 6.6 – 8.3%, 16 – 32% and 11 – 40%, respectively. Increased AU is an early highly sensitive marker of adverse kidney damage and may be a consequence of glomerular filter dysfunction, the development of intraclubular hypertension, and also reflects the presence of endothelial dysfunction. Probably, this is due to the high percentage of occurrence of persons with an AU level of more than 30 mg /g already among patients with "isolated"

CHF, which naturally increased with the addition of obesity to CHF. The decrease in GFR with the addition of obesity is due to damage to the kidneys by visceral fat and loss of nephrons.

The results of the study show that an increase in albuminuria and a decrease in GFR as BMI increases in patients with CHF significantly worsen the prognosis, which is reflected in an increase in the combined risk of CKD progression and the development of cardio-vascular complications.

The addition of obesity to CHF leads to a significant increase in the level of β2-microglobulin urine in patients with CHF when compared with a group of patients with CHF without comorbid pathology.

This indicates the presence of tubular dysfunction in this category of patients.

β2-microglobulin is a low molecular weight protein that passes freely through

the membrane of the renal glomeruli, then 99.8% of it is reabsorbed in the proximal part of the renal tubules. Impaired renal tubule function leads to an increase in urinary excretion of β2-microglobulin, therefore, the determination of this indicator in urine primarily reflects tubular dysfunction. In addition, β2-microglobulin is also a protein of inflammatory reactions. In patients with CHF

Significant associations have been established between the level of β2-microglobulin and leptin, adiponectin and obesity. It is known that leptin receptors are located in the cells of the tubular epithelium, which is probably due to an increase in the concentration of β2-microglobulins in conditions of leptin resistance in obesity.

The negative effects of obesity on the kidneys can be both a consequence of developing comorbid conditions, such as diabetes, hypertension, and the result of direct effects of adipose tissue on the kidneys associated with the endocrine activity of substances produced by adipocytes (adiponectin, leptin, etc.). The results obtained in this study reflect an increase in the concentration of leptin and a decrease in the concentration of serum adiponectin when joining CHF obesity. Recent studies confirm the existence of a close relationship between adipokines and CKD, especially in the presence of metabolic disorders. The level of leptin increases depending on the severity of renal dysfunction by 4 –7.5 times, and a decrease in the level of adiponectin is considered an independent predictor of deterioration of renal function in the initial stages of CKD. Hyperleptinemia also activates the proliferation of mesangiocytes, which increases their production of fibrogenesis mediators, has an anti-natriuritic effect and increases insulin resistance. In addition, it has been shown that in obesity, leptin can induce the production of type I collagen by mesangial cells and stimulate the proliferation of endotheliocytes and vascular smooth muscle cells, indirectly causing glomerular hypertrophy. On the other hand, obesity is associated with a low concentration of adiponectin.

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Adiponectin stimulates a number of enzyme reactions responsible for the production of NO and maintenance of normal endothelial function. Previous studies demonstrate the existence of an inverse relationship between albuminuria and adiponectin levels in the blood of obese individuals. The results of this study show the presence of close correlations between adiponectin concentrations and parameters reflecting the functional state of the kidneys. The nephroprotective effect of adiponectin can be explained by its ability to reduce the level of TNF-a expression, suppress its pro-inflammatory effect, increase endothelium-dependent and endothelium -independent vasodilation and improve endothelial function, inhibit endothelial cell hyperproliferation. There are works, in which it is shown that an increase in the level of adiponectin leads to a decrease in albuminuria, glomerular hypertrophy and a decrease in the inflammatory response in kidney tissue. A decrease in the level of AU leads not only to a slowdown in the progression of CKD, but also to a decrease in the risk of cardiovascular complications in patients with CHF and obesity.

Another of the main pathogenetic links in the progression of CKD is insulin resistance. It is known that insulin resistance leads to the development of systemic and local (organ and tissue vasoconstrictor) reactions, endothelial dysfunction and deficiency of the main vasodilator – nitric oxide NO. For the kidneys, this means spasms of glomerular arterioles, disorders of renal trophism, renal blood flow,
microcirculation and glomerular filtration, hypoxia and ischemia of the renal parenchyma. The data obtained confirm the important pathogenetic contribution of IR to the formation of renal dysfunction, as evidenced by the presence of a significant correlation between IR and albuminuria, β-2-microglobulin.

Conclusion

The results of the study indicate that the combination of CHF with obesity is accompanied by a significant deterioration in the functional state of the kidneys, an increase in the combined risk of progression of CKD and the development of cardiovascular complications.

Significant correlations between parameters characterizing kidney function and adipokines, HOMA-IR reflect an important pathogenetic contribution of hormonal activity of visceral adipose tissue and insulin resistance to the development and progression of renal dysfunction in patients with CHF and obesity.

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