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KIDNEY DAMAGE IN CHRONIC HEART FAILURE

Khusainova Munira Alisherovna
Samarkand State Medical University

Toirov Doston Rustamovich
Samarkand State Medical University

Uzokov Jurabek Baxtiyorovich
Samarkand State Medical University

Muhammadiyeva Dilnura Sherzodovna
Samarkand State Medical University

ABSTRACT

Despite the achievements of modern cardiology, chronic heart failure (CHF) is still a prognostically unfavorable condition. Mortality among patients with CHF is 4-8 times higher than in the general population, half of all patients die within 5 years after diagnosis. In patients with CHF of functional class IV (FC), mortality within six months reaches 44%. Heart and kidney lesions are widespread in the population and often coexist, increasing mortality and the risk of complications. The development of renal dysfunction (DP) is one of the most common comorbid conditions with CHF. A decrease in the contractility of the myocardium leads to a deterioration in the functional state of the kidneys, which, in turn, can cause the progression of CHF. DP in CHF may be associated with the addition of concomitant pathology of the kidneys and renal vessels, however, more than two thirds of patients with CHF without concomitant primary kidney pathology have chronic kidney disease (CKD), the prevalence of which among patients with decompensated CHF is 50-70%. DP significantly worsens the prognosis in people with CHF and low left ventricular ejection fraction (LVEF). Randomized studies of SOLVD and SAVE have shown an association between DP and mortality in patients with LV systolic dysfunction. With a decrease in glomerular filtration rate (GFR) <60 ml/min/1.73 m^2, the risk of mortality increased by 2.1 times, with reduced LV systolic function – by 3.8 times. It should also be noted that with a pronounced violation of the contractility of the LV myocardium, a decrease in GFR, as a rule, coincides with the appearance of another unfavorable predictor – an increase in the level of natriuretic peptides. In the meta–analysis, which included 80,098 patients with CHF, DP occurred in 63% of patients, and in 29% it was moderate or severe, mortality during the year among patients

without DP was 24%, in the presence of concomitant DP - 38%, with moderate or severe DP – 51%.

Keywords: kidney function; chronic kidney disease; chronic heart failure; cardiovascular risk;

INTRODUCTION

DP is an independent predictor of poor prognosis of CHF, although the pathogenesis of transient deterioration of renal function during decompensation of CHF remains not fully elucidated. On the one hand, patients with cardiovascular pathology develop DP as a consequence of cardiac pathology leading to the development of CKD. On the other hand, people with chronic kidney damage that has arisen against the background of a urinary tract disease develop damage to the cardiovascular system, aggravating the course of the underlying disease. Obviously, the primary nature of kidney and cardiovascular diseases is conditional (cardiorenal or renocardial syndrome), since the defeat of one organ invariably leads to a deterioration in the function of the other.Currently, the kidneys have been considered as an organ contributing not only to the formation of edematous syndrome, but also to the progression of myocardial dysfunction. This is due to the fact that the kidneys, by increasing preload, contribute to LV dilation, and by producing renin and activating the renin-angiotensin-aldosterone system, the development of myocardial hypertrophy and fibrosis.Over time, patients with CHF may develop DP, in some cases progressing up to chronic renal failure (CRF). Kidney function in CHF suffers mainly due to a drop in cardiac output and neurohumoral activation.

Previous studies have shown that in the early stages of CHF, the narrowing of the carrying arterioles prevails over the narrowing of the bearing ones. Nitric oxide, natriuretic peptides, prostaglandins E2 and E12 have a vasodilating effect on bringing arterioles. As a result, despite the decrease in renal blood flow, renal perfusion pressure and filtration fraction (FF) increase in the early stages of CHF, GFR does not change. With the progression of CHF, accompanied by a further drop in cardiac output, as well as depletion of local vasodilating systems, renal blood flow decreases so much that renal perfusion pressure, FF and GFR decrease and serum creatinine concentration increases.

The value of DP as a prognostic factor has been underestimated or ignored for a long time. Even the largest studies (CONSENSUS, SOLVD) did not consider the effect of DP on the survival of patients with CHF. For the first time, the prognostic value of serum creatinine concentration in patients with CHF was shown in the mid-1990s.

They showed that GFR is an independent predictor of overall and cardiovascular mortality, even stronger than LVEF and CHF FC according to NYHA. With GFR <44 ml/min, the relative risk of death was almost 3 times higher than with GFR >76 ml/min. Other studies have also shown that a decrease in GFR can serve as an independent predictor of cardiovascular mortality in CHF. According to some researchers, the kidney condition should be considered as a possible "mediator of CHF progression". There is evidence of a direct correlation between the severity of CHF and impaired renal function and that in CHF, the significance of DP as a predictor of an unfavorable prognosis is as great as LV FV and CHF FC.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.

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. 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

The course of BMI among patients from group I to group iii naturally increased. Abdominal obesity is characterized by significantly greater obesity compared to normal body weight and is very common in patients with normal body weight (78.6, 0 and 70%). Obesity can be detected among people with a high percentage of visceral obesity/overweight, as well as among patients with normal body weight: 17.6% among people with BMI, 70% among overweight people and 100% among obese people

(differences between groups I and II, I and III, II and III reliable). It was found that patients with obesity and prostate dysfunction are significantly more noticeable than the tissues assessed by the VAI index compared to the "discrete" prostate. Due attention is paid to the analysis of the functional state of the kidneys, that in the severe stage of detection in people with BMI, the frequency of CKD increases significantly. There were also hungry people who clearly added obesity throughout their lives. The statistical value for this indicator reaches values between groups I and III. > 30 mg/g in all studied groups. Group I and II, respectively, had significantly lower rates than group III (98 and 71 and 92.5%, respectively). Visceral obesity, fasting and body weight (r = 0.58 and r = 0.42), GFR (r = -0.42 and r = -0.38), as well as fasting levels with (R = 0.42 and r = 0.40), GFR (r = -0.38 and r = -0.37) in patients with CHF along with obesity and overweight. Due to obesity, CHF causes a significant increase in the level of 2 microglobulin detected in the urine. At the same time, in the group of patients with obesity and CHF, a significant correlation was maintained between the content of microglobulins in urine and the levels of leptin (r = 0.62) and adiponectin (r = -0.88), xoma-IR (r = 0.66). Patients have a high risk of CKD progression and development of cardiovascular complications and a very high degree of reduction in the risk of obesity in patients with CHF and GFR associated with albuminuria. In addition, the difference in views is often paid attention not only to the risk of obesity in certain categories of patients, but also prognostically in patients with hCG and hCG and in overweight patients. The study of markers of laboratory fattening revealed a significant decrease in the concentration of adiponectin and an increase in the level of leptin from group I to group III. Correlation analysis shows that there is a statistically significant relationship between the concentration of leptin and GFR (r = -0.52), AU (r = 0.36), adiponectin and GFR (r = 0.38), the concentration of adiponectin and AU (r = -0.32) in the concentration of mast hormone and adiponectin in serum. Similar associations are observed in a patient with CHF and overweight. Manifestations of insulin resistance group I is characterized by a negative increase in body weight, not accompanied by significant changes in the indicators of group III.

In the group of patients with CHF suffering from obesity and overweight, there is an increase in the rate and frequency of insulin resistance. At the same time, attention to the increase in the Homa index and the prevalence of IQ increased in the group of patients with CHF and normal weight to 17.6%. The increase in the male population is also reflected in statistically significant correlations between the levels of visceral fat and HOMA-IR in patients with obese CHF and an increase in overweight (r = 0.76 and r = 0.72, respectively). In addition, it was found that among overweight and obese patients:

The most important associations between AS and HOMA-IR (r = 0.28 and r = 0.34), GFR and HOMA-IR (R = -0.30 and r = -0.29), violence.

DISCUSSION

It should be noted that a high percentage of patients with abdominal and visceral obesity in groups of people with normal and excessive BMI indicates the need to assess not only BMI, but also its role in the diagnosis of obesity, the ratio of fat / Obi, as well as visceral fat in the body. The results of the studies confirmed the important role in the development of renal insufficiency and the progression of pathogenetic dysfunctions. The combination of CHF with water led to a significant reduction in the level of obesity compared to the group of patients with GFR pathology. Many studies receive information from those that show that the association with obesity is calculated with a decrease in the probability of GFR < < 60/min/m2 of 1.73. 30%. Regarding the increase in BMI, there are statistically significant AS (more than twice in "isolated" CHF patients and obese CHF patients). In patients with a significant decrease in GFR (<60 ml/min / 1.73m²) and AS, together with CHF, indicate a deterioration in the functional state of the kidneys. Data from some studies indicate the frequency of albuminuria in patients with CHF. 6.6-8.3%, 16-32% and 11-40%, respectively, its degree is increased with the general spread of hypertension and evenly with diabetes mellitus. A high level of intraglubular hypertension is reflected in the development of negative, also endothelial dysfunction, premature kidney damage and possible dysfunction of the glomerular market. Perhaps this is due to a higher percentage in people who have elevated au levels of more than 30 mg/g among "individual" patients CHF promotes natural fat gain by adding water. Along with the reduction of visceral harm, obesity is associated with fat loss and budding of GFR nephrons.

CONCLUSION

The results of the studies showed that in patients with BMI, albuminuria is further complicated by an increase in proliferation and a decrease in measures to reduce CHF GFR, which increases the risk of complications with mixed cardiovascular diseases, which affects the progression of CKD.

The contribution of CHF to obesity leads to a large group of patients with prostate pathology compared with the presence of increased CHF in other patients-the presence of microglobulin in the urine.

Patients of this category indicated concomitant dysfunction of the tubular channels.

usbular2 - microglobulin is a molecular protein that is free

of the renal membrane, from which 99.8% is then reabsorbed in the proximal part of the renal canal. Violation of the function of the renal channels leads to an increase in the excretion of 2-microglobulin in the urine, therefore, the determination of this indicator primarily reflects the dysfunction of the channel. In addition, uning2-microglobulin is a protein of inflammatory reactions. In patients with CHF 2 among the associations, there were more significant suppliers-the level of leptin and microglobulin, and adiponectin in obesity. As is known, water receptors are located on the epithelial cells of leptin, which is probably due to an increase in the concentration of leptin-2-microglobulin resistance in the blood.

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