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Kidney dysfunction in chronic heart failure

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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%. The association of FC CHF with patient survival is recognized by almost all researchers. It seems obvious that the higher the HCN FC, the worse the prognosis. However, the linear relationship between CHF FC and the mortality of patients is not always traced. The results of a comparative study of the survival of patients with coronary heart disease (CHD) and symptoms of decompensation and without signs of CHF (n=1964), conducted showed that only the terminal stages (IV FC) of CHF play the role of an independent predictor of a poor prognosis (80% of mortality within 3 years), while with I-III FC survival rates are approximately the same: mortality is 38-42%. The immediate cause of decompensation of CHF may be various conditions that by themselves usually do not lead to CHF. 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. A number of retrospective studies have established a link between the course of CHF and DP, which is accompanied by a deterioration in the prognosis of patient survival. It is believed that the presence of DP in patients with CHF may be a predictor of an unfavorable clinical outcome. However, the degree of DP is not indicated in the diagnosis and its correction is not carried out.

Key words: chronic heart failure, glomerular filtration rate, chronic kidney disease

Introduction

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 m2, 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%. 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.

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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. That is, over time, a significant number of patients with CHF develop CRF. There is an opinion that a decrease in GFR is characteristic of the late stages of CHF, when there is a sharp decrease in renal blood flow and disruption of compensatory mechanisms.

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. D.L. Dries et al. , retrospectively analyzing the data from the studies of SOLV'D Treatment and SOLVD Prevention, confirmed that the calculated values of GFR are an important factor determining the survival of patients with CHF. 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 earliest marker of kidney damage is considered to be MAU. There is a hypothesis that MAU is a manifestation of generalized endothelial permeability disorder and increases the risk of complications from the cardiovascular system. Currently, MAU is considered as one of the important predictors of the risk of developing cardiovascular complications and can be used in the diagnosis of DP in CHF. As for the determination of GFR (by creatinine level), according to available data, an increase in serum creatinine does not always reflect changes in GFR. However, in addition to GFR (creatinine level), which depends on the anthropometric and gender data of patients, there are no other methods and markers for determining DP in the examination protocols. However, it is believed that cystatin C is a strong and independent predictor of cardiac mortality in patients with severe CHF and with normal or mildly impaired renal functions, it is considered more reliable in detecting glomerular filtration disorders, GFR is calculated by this indicator without taking into account anthropometric and gender data. A. Moran et al. it is believed that cystatin C levels are linearly associated with the risk of progression of systolic HF, and the rate of risk of rapid progression of diastolic HF can be judged by the appearance of high concentrations of cystatin C. Determination of alpha-1-microglobulin (A1M) protein in urine can also be considered as an early marker of DP in CHF. The content of A1M in urine reflects the severity and degree of damage to the renal tubules, an increase in its concentration in urine indicates moderate and reversible changes not associated with a violation of the histomorphological structure of the kidneys. A1M is recognized as an early sensitive marker of preclinical kidney pathology developing with a complication of coronary artery disease, its increased urinary excretion may precede an increase in serum creatinine levels. In one of the studies, a direct relationship was determined between the indicators of endothelial dysfunction, CHF FC and the severity of proteinuria measured by the content of A1M in urine. Therefore, A1M can also be considered as an indicator of endothelial dysfunction in patients with CHF. Obviously, to determine DP in patients with CHF, it is possible to use the determination of A1M in urine and cystatin C in serum, as well as GFR (by the level of cystatin C). The existing studies were conducted mainly with the participation of patients with comorbid pathology, and there are actually no studies on DP in patients with CHF in the absence of renal and endocrine pathology. Therefore, we decided to evaluate the indicators of renal function in patients with CHF in the absence of primary renal and/or endocrine pathology.

The aim of the study: to study cardiorenal relationships in patients with CHF.

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Materials and Methods

The study included 130 patients with the presence of clinical signs of CHF II, III FC according to NYHA of ischemic genesis with LV FV 50.9±7.68%. Of these, 73 (56.2%) patients were with CHF II FC (LVEF 52.2±5.09%) and 57 (43.8%) patients were with CHF III FC (LVEF 47.2±6.61%), the average age was 60.5± 7.2 years. The prescription of the transferred large–focal (with a Q wave) myocardial infarction is 4.06 ± 3.27 years. The number of hospitalizations over the past year averaged 1.05±0.32. The diagnosis of CHF and the assessment of its severity (FC) were carried out according to the recommendations of the European Society of Cardiology for the diagnosis and treatment of acute and chronic HF (2012). All studies were conducted with the informed consent of patients. Exclusion criteria: primary pathology of the kidneys and urinary tract, unstable angina pectoris, acute cerebrovascular accident suffered in the next 6 months, hemodynamically significant heart defects, severe liver dysfunction, arterial hypertension above grade 2, complex rhythm disturbances, permanent form of atrial fibrillation, diabetes mellitus. All patients had no pathological changes in urine tests and ultrasound of the kidneys. Drug treatment included beta-blockers, angiotensin converting enzyme inhibitors or angiotensin receptor antagonists, disaggregants, statins, diuretics. All patients underwent general clinical studies. Ultrasound of the heart was performed on the Vivid-7 device (GE, USA – Belgium) according to the standard procedure. Biochemical studies were performed on an Olympus analyzer: the level of cystatin C was determined using Randox laboratory kits (norm – 0.571.05 mg/L), creatinine concentration – using Weskmap kits (norm – 44.0110.0 mmol/L), the level of NT-proBNP in blood serum was determined using an automatic enzyme immunoassay analyzer mini Vivad (norm – up to 125 pg/ml). GFR by creatinine level was calculated using the Cockroft-Gault formula, GFR by cystatin C – by the formula GFR=-4.32+80.35/cystatin C. To determine the MAU (in the morning portion of urine >30 mg/l), an Olympus analyzer was used, the level of A1M in urine was a method of direct solid–phase enzyme immunoassay using a pair of monoclonal antibodies ELISA-A1M (norm – up to 10 mg/l). The vasomotor function of the endothelium was determined by high–resolution ultrasound using the D.S. Celermajer method, the pulse wave propagation velocity (CPV) was determined using the Impecard-M computer complex (the norm is up to 10.2 m/s). Statistical analysis was carried out using generally accepted methods of mathematical statistics using the statistical software package Statsoft Statistica 6.0 for Windows (USA), MS Excel XP. The results are presented in the form of arithmetic mean (M) and mean standard deviation (SD). To compare the quantitative indicators of two independent groups with a normal distribution of the trait, the Student's criterion (t) was used. The differences were considered significant at $p<0.05$.

Results

The levels of urea, blood glucose, and enzymes were within normal values in all examined patients. The level of NT-proBNP in patients averaged 268.07±92.16 pg/ml. The mean values of creatinine (99.80±11.67 mmol/L) and cystatin C (0.93±0.09 mg/L) in the examined group did not exceed normal values. However, the mean GFR values for creatinine and cystatin C levels are lower than normal values (83.12±12.78 and 84.25±11.87 ml/min/1.73 m2, respectively) and indicate that patients have a decrease in GFR and impaired glomerular filtration of the kidneys. A decrease in GFR (mild and moderate), determined by the level of cystatin C, was observed in 63.8% of patients. Consequently, most patients with ischemic CHF had chronic DP in the absence of primary renal pathology. A moderate decrease in GFR (in terms of cystatin C) was observed in 8.5% of patients – these patients have lesions of target organs in the absence of clinical manifestations. There were also elevated levels of A1M (15.4% of patients) and MAU (13.8% of patients). Also, 91.7% of patients had a violation of the vasomotor function of the endothelium, which was manifested by insufficient vasodilation in response to reactive hyperemia, as well as its absence (26.5% of patients), the presence of a vasoconstrictor reaction (18.9% of patients) and flow turbulence (11.4% of patients). The brachial artery sensitivity coefficient to shear stress was below normal ($p<0.05$). Endothelial dysfunction, characterized by a violation of endothelium-dependent vasodilation, is largely associated with increased arterial stiffness. The average CPV was 10.32±2.58 m/s, which is slightly higher than the normal level of this indicator. However, 39.4% of patients had an increased level of CPV, which may indicate reduced elasticity (increased stiffness) of arterial vessels. And, as is known, both the risk of cardiovascular diseases and arterial stiffness increase even with a slight decrease in kidney function. As a result of the correlation

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analysis, the relationship of the level of cystatin C with the severity of CHF (r=0.49, p<0.01), with the level of NT-proBNP (r=0.52, p<0.01), with the content of A1M in urine (r=0.50, p<0.01), with LV LV (i=-0.56, p<0.01), as well as between the level of cystatin C and the level of NT-proBNP (r=0.46, p<0.01). In the group of patients with CHF III FC, a decrease in GFR, determined by the level of cystatin C, was noted in 70% of cases. MAU was detected in 21% of patients, A1M – in 15.7%. In 96.5% of patients in this group, a violation of the vasomotor function of the endothelium was detected, in 52.6% – an increased level of SRPV. An increase in arterial stiffness may further lead to an increase in the cardiovascular risk associated with the development of DP in CHF. In CHF III FC, a relationship was revealed between CPV and the level of cystatin C (r=0.43, p<0.01), LV LV and the level of cystatin C (r=-0.86, p<0.001), the content of A1M and the coefficient of sensitivity of the brachial artery to shear stress (r=0.41, p<0.05), the concentration of A1M and the rate index of endothelial dysfunction (i=0.45, p<0.01), which confirms the relationship between the indicators of endothelial dysfunction, FC CHF and the severity of proteinuria measured by the content of A1M in urine. A close correlation was also revealed between the level of cystatin C and the level of NT-proBNP (r=0.51, p<0.01), which is consistent with the results of an earlier study [33], which mentioned the possibility of combined measurement of cystatin C and NT-proBNP to predict cardiovascular mortality in patients with HF.

Conclusion

According to previous studies, even a slight decrease in kidney function significantly worsens the course of the underlying cardiac pathology, increasing the frequency of complications and the risk of death. In our study, most patients with CHF of ischemic etiology have signs of DP in the absence of clinical manifestations. In CHF III FC, the signs of DP are determined against the background of endothelial dysfunction and increased arterial stiffness. It is possible that the close relationship of DP with the severity of the clinical condition of patients with CHF partially explains the role of DP as a factor in the progression of CHF, which emphasizes the importance of a comprehensive examination of the kidneys in patients with CHF and the inclusion of methods for assessing renal hemodynamics among the methods for diagnosing DP. Moreover, the Heart Outcomes Prevention Evaluation study showed that even a minor impairment of kidney function, regardless of other risk factors and treatment, is associated with an increase in cardiovascular events by 40%. According to the results of our study, the determination of the level of cystatin C, MAU and A1M makes it possible to detect the onset of renal dysfunction in patients with CHF. Cystatin C has the greatest sensitivity to the detection of early renal dysfunction and can be used as a marker of early DP in the absence of a clinical picture. Probably, cystatin C, MAU and A1M can be considered as early markers of DP in CHF, which are associated with the risk of developing systolic HF. Therefore, DP can be considered as a possible marker of the progression of CHF and tactics to prevent the progression of CHF should be directed to maintaining optimal kidney function.

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