KIDNEY DYSFUNCTION IN CHRONIC HEART FAILURE

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ABSTRACT

The aim of the study was of the importance of impaired renal function in heart failure.

Material and methods. 42 patients who received inpatient treatment in 2017–FC II–IV, complicated by FC II —III CHF were examined.

Results. Urea levels and creatinine values (98.80±11.67 mmol/l) in both groups of the examined did not exceed normal values. Before the start of treatment, the level of Cystatin-C was 1.43 ± 0.06 mg / l in the main group of patients. In the control group, this indicator was 0.83 ± 0.01 mg / l. It was reported that the content of cystatin-C in the main group was 1.7 times higher than in the control group. After complex treatment, these values were 0.6 ± 0.01 mg / l in the main group and 0.81 ± 0.01 mg / l in the control group, respectively.

Conclusion. Therefore, cystatin C and β2 — MG can be considered as an early sign of tubulointerstitial dysfunction and fibrous changes in the kidneys. The presence of renal dysfunction worsens the course of CHF, and also worsens the quality of life of patients.

Key words: chronic renal failure, creatinine, chronic heart failure, beta-2-microglobulin.

INTRODUCTION

Chronic heart failure is a syndrome that develops as a result of a violation of the heart's ability to fill and/ or empty, occurring in conditions of imbalance of vasoconstrictor and vasodilating neurohormonal systems; accompanied by inadequate perfusion of organs and tissues of the body and manifested by a complex of symptoms: shortness of breath, weakness, palpitations, increased fatigue and fluid retention in the body. CHF can be caused by many diseases of the cardiovascular system, the most common of which include coronary heart disease, hypertension, endocarditis and rheumatoid heart defects. The weakening of the heart muscle leads to the inability to pump blood normally, as a result of which the amount of blood released into the vessels gradually decreases. Cardiorenal syndrome (CRS) is a pathological interdependent condition involving the heart and kidneys, developing as a result of acute or chronic dysfunction of one of the organs, followed by acute or chronic dysfunction of another organ. includes various acute and chronic disorders in which both the heart and the kidney may be the primary affected organ. The kidneys, being an organ involved in

important metabolic processes, regulate humoral The systems involved in microcirculation processes are susceptible to acute and chronic effects in various cardiovascular diseases (CVD) and affect the formation and progression of cardiovascular pathology. Renal dysfunction is associated with a higher incidence of recurrence of myocardial ischemia, myocardial infarction (MI), stroke, serious hemorrhagic complications, acute heart failure, atrial and ventricular fibrillation. Even a slight decrease in kidney function significantly worsens the course of the underlying cardiac pathology, at the same time. There are numerous data in the literature regarding the diagnostic values of biomarkers (creatinine, cystatin C) and the calculated index (creatinine clearance) for determining the functional state of the kidneys. The concentration of cystatin C is inversely correlated with the glomerular filtration rate (GFR), is a marker of glomerular dysfunction even if there is no increase in creatinine, and gives a more accurate approximation to the real values of GFR than the creatinine level. In, it was proved that cystatin C is a strong and independent risk factor for cardiac mortality in patients with severe heart failure with normal or slightly impaired renal function. There are also studies devoted to the study of beta-2-microglobulin (β^2MG) in patients with CHF. β^2MG is an independent predictor of mortality in the elderly, but the prognostic role of beta^2-microglobulin in heart failure has not been fully studied. In the kidneys, beta-2-microglobulin is filtered by glomeruli and reabsorbed back into the tubules. The detection of β^2 MG in urine indicates a violation of renal filtration. Therefore, it is used as a marker for the diagnosis and monitoring of tubulointerstitial kidney damage.

MATERIALS AND METHODS OF RESEARCH

The study was conducted in the cardiology departments of the multidisciplinary clinic of SamMU. 42 patients who received inpatient treatment in 2017–FC II–IV, complicated by FC II —III CHF were examined. The diagnosis of CHF was made on the basis of anamnesis, complaints, clinical and laboratory data and recommendations of the European Association of Cardiologists "ESC Recommendations for the diagnosis and treatment of acute and chronic heart failure 2016". The patients were divided into the main group of patients with CHF with renal dysfunction — 18 patients (women 7(38.8%), men 11 (61.1%), average age 65.3±8.2 years) and without dysfunction — 24 patients (women 10 (41.6%), men 14 (58.4%), average age 64.2±7.3 years). If we consider the comorbid conditions, the following changes were revealed, in the first group of CHF patients with renal dysfunction, stable tension stenocardia I - II FC - in 25.8%, III FC — in 18.5% of patients. According to the NYC classification, CHF I FC was observed in 2.3% of patients, II FC in 63.7%, III FC in 34.2% and IV FC in 4.9% of patients, hypertension was diagnosed in 96.4% of patients. Anemia of mild and moderate severity was observed in 65.6% of patients, hyperlipidemia in 64.3%. It is known from the life history of patients of the second group that stable

angina pectoris of tension I — II FC was observed in 22.3%, III FC in 19% of patients, and 90.2% suffered from hypertension. Anemia of mild degree was observed in 31.1%, hyperlipidemia - in 54.0% of cases. There were no changes in clinical urine tests and renal echosonography results indicating the presence of CKD. Patients with acute heart failure, acute myocardial infarction, and nesta were excluded from the study- severe angina pectoris, pericarditis, rheumatic heart diseases. All patients underwent general clinical (UAC, OAM), biochemical studies (ASAT, ALAT, total bilirubin, urine wines, creatinine, total protein, blood glucose d/e and n/e), cystatin C, the level of β2 — MG in urine, as well as instrumental methods of ECG and echocardiography, were used as an indicator of renal tubular functions in this study Statistical processing of the research results was carried out using the Microsoft Excel spreadsheet package 2019. The parameters were described as: arithmetic mean ± standard deviation (M±SD). To study the relationship between quantitative variables, correlation analysis was used with the calculation of Pearson's linear correlation coefficient or Spearman's rank correlation coefficient. The differences were considered significant at a significance level of $p<0.05$.

THE RESULTS AND THEIR DISCUSSION

Urea levels and creatinine values (98.80±11.67 mmol/l) in both groups of the examined did not exceed normal values. Before the start of treatment, the level of Cystatin-C was 1.43 ± 0.06 mg / l in the main group of patients. In the control group, this indicator was 0.83 ± 0.01 mg / l. It was reported that the content of cystatin-C in the main group was 1.7 times higher than in the control group. After complex treatment, these values were 0.6 ± 0.01 mg / l in the main group and 0.81 ± 0.01 mg / l in the control group, respectively. It was noted that GFR calculated using cystatin C revealed lower values (84.12±12.78 and 85.25±11.87 ml/min/1.73 m2, respectively), which indicated the presence of a decrease in GFR and impaired glomerular filtration function of the kidneys in patients. A decrease in GFR, determined in relation to cystatin C, was observed in 45.3% of patients. It follows from this that the majority of patients with CHF of ischemic origin had chronic renal dysfunction in the absence of primary renal pathology. In this regard, it is advisable to use it in assessing the functional state of the kidneys. Based on the conducted comprehensive studies, it was found that the content of β2 — MG in urine was significantly reduced (P0.001) to 6.4 ± 0.2 mg / ml in the main group. In the control group, that is, in patients with CHF without dysfunction, these values were 7.04 ± 0.2 mg/ml before treatment, and then 6.42 ± 0.2 mg /ml, and the values did not significantly differ from each other (P0.05). When studying the correlation between microglobulin β2 and cystatin-C, positive correlations of r = 0.59 (R0.05) were recorded in the main group, respectively. Thus, fibrous changes in the proximal tubules of the kidney accompanied by CHF with dysfunction enhance tubulointerstitial processes in it, which, in turn, leads to an increase in the content of

cystatin-C in the blood.

CONCLUSIONS

The results of the conducted studies have shown that even a slight decrease in kidney function exacerbates the course of the underlying cardiac pathology, increasing the frequency of complications. DP can be considered as a possible marker of CHF progression. It can also be concluded that the majority of patients with CHF of ischemic etiology have signs of renal dysfunction in the absence of clinical manifestations. Therefore, cystatin C and β2 — MG can be considered as an early sign of tubulointerstitial dysfunction and fibrous changes in the kidneys. The presence of renal dysfunction worsens the course of CHF, and also worsens the quality of life of patients.

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