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Assessment of renal dysfunction and the effect of gliflozins on chronic heart failure developed due to rheumatic heart defects

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Annotation: Chronic heart failure (CHF) is a disease in which the heart is unable to pump enough blood to supply the body with oxygen. It can occur as a result of many diseases of the cardiovascular system, among which the most common are coronary heart disease, hypertension, rheumatoid heart defects, and endocarditis.

Key words: Chronic heart failure, cystatin C, KIM-1.

Introduction

Chronic heart failure (CHF) is one of the most common clinical syndromes among the world's population and one of the main causes of mortality and disability as the final stage of a number of cardiovascular and other organ diseases. According to confirmed data, 1.0 - 2.6% of the population of Europe, 2.2% of the population of the United States of America and 7% of the population of the Russian Federation have SES, and this figure is shown to increase with age. 5% of all hospitalized patients in European countries are diagnosed with chronic heart failure. In the Russian Federation these figures are 16.7%.

CHF is the leading cause of hospitalization for the majority of the population over 65 years of age. Despite the widespread use of drugs whose effectiveness has been proven in its treatment, mortality among patients during the last 5 years after diagnosis is 60% in men and 45% in women. The mortality rate of patients with CHF is 4.0-10.3 times higher than that of the healthy population of this age. In recent years, a number of observations have confirmed that an increase in serum creatinine and impaired renal function with a decrease in glomerular filtration rate (GFR) worsen the outcome of CHF. Therefore, this condition is considered a risk factor for worsening the progression of CHF. Epidemiological and population studies have confirmed that early, even subclinical, renal dysfunction leads to a sharp deterioration in the condition of patients with CHF. According to a number of authors, renal dysfunction in CHF is determined in 32-60% of cases according to criteria such as creatinine, creatinine clearance, CFT, cystatin C, microalbinuria. Adding CKD to renal dysfunction dramatically increases hospitalizations, readmissions, and mortality. In recent years, several observations have emerged on the use of cystatin-C as an alternative marker for assessing renal function and cardiovascular risk. Its amount, unlike creatinine, does not depend on muscle mass and makes it possible to more accurately check CFT in children, the elderly, patients with diabetes, and pregnant women with non-standard muscle mass. Cystatin-C was first discovered in 1961 in cerebrospinal fluid, later in urine during tubular proteinuria, and in 1962 in blood serum and other biological fluids. Cystatin-C is a polypeptide

consisting of 120 amino acid chains with a molecular weight of 13.4 kDa. Its amount in the blood serum is an indicator of CFT, and indeed, in 1985, when this polypeptide was determined using an exogenous marker, it was confirmed that there is a very high correlation between its level and glomerular filtration capacity. After numerous trials, cystatin-C has been shown to be a very sensitive marker of renal function compared to creatinine, especially in cases where the fraction is slightly reduced, where the CFT is 90-60 ml per minute per 1.73 m2 of body surface area. .

Cystatin-C has also been shown to be one of the strongest and independent risk factors predicting cardiac death in patients with severe JES, regardless of renal functional status. In recent years, detection of the transmembrane protein Kiney Injury Molecule-1 (KIM-1), which contains mucin and immunoglobulin domains, has been recommended for early assessment of renal tubular changes.

Experimental models have shown that increases in KIM-1 are associated with ischemic effects on the kidney and are not always accompanied by increases in blood creatinine levels. Based on a number of observations, this protein is considered an early and reliable marker of renal tubular damage. Femke Vanaders et al noted that in patients without diabetes but with proteinuria, compared with controls, it was directly correlated with proteinuria levels, and this relationship was reduced by angiotensin-converting enzyme inhibitors (ACEIs). K.G. Jungbauer et al showed significantly higher levels of KIM-1 in patients with CHF compared to healthy controls. It has also been found to predict hospital readmission due to increased left ventricular ejection fraction, all-cause mortality, and increased chronic heart failure. K.G. Jungbauer et al suggested that renal tubular changes could be detected by KIM-1 in patients with normal performance status. Based on the above, detection of IMT-1 can be considered as a reliable marker of cardiorenal changes and an examination method that has additional prognostic value. It has also been found to predict hospital readmission due to increased left ventricular ejection fraction, all-cause mortality, and increased chronic heart failure. K.G. Jungbauer et al suggested that renal tubular changes could be detected by KIM-1 in patients with normal performance status. Based on the above, detection of IMT-1 can be considered as a reliable marker of cardiorenal changes and an examination method that has additional prognostic value. In particular, in an observation conducted by J. McMurray, a famous scientist dealing with the problems of chronic heart failure in Scotland, and co-authors, it was found that 8% of patients due to rheumatic heart developed chronic heart failure. defects. J.R. Terlink et al analyzed 31 cases and found that valvular changes resulted in HF in 4% of cases. In the Hillingdon study, 7% of heart defects detected by non-invasive diagnostic methods were caused by chronic heart failure. Another Framingham Heart Study found that heart defects were the etiological cause of HF in 9% of men and 16% of women. In a large study conducted in the United Kingdom between 1994 and 2001, 5.4% of 963 patients with chronic heart failure presenting to a physician with normal left ventricular ejection fraction (LVEF) had valve changes. In addition, during a prospective observation of 229 hospitalized patients with chronic heart failure, heart defects were found in 23.5%. Among heart defects, mitral valve insufficiency and aortic stenosis in most cases lead to chronic heart failure. Rheumatic heart defects are one of the leading causes of chronic heart failure in the Russian Federation.

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