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KIDNEY DYSFUNCTION IN CHRONIC HEART FAILURE

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✓ ***Resume***

In the 21st centure, chronic heart failure remainsone of the medical and social problems of society, since its prevalence, development, negative consequences and economic costs are high. Chronic heart failure is an important medical, social and economic problem, representing a new epidemic of cardiovascular disease, covering more than 23 million people worldwide. Numerous studies have shown that impaired renal function is strongly correlated with a prognosis of heart failure in patients with systolic dysfunction of the left ventricle. Moreover, renal dysfunction is a strong independent risk factor for the adverse course of coronary heart disease and mortality in patients with advanced heart failure. Given the close relationship between the work of the heart and kidneys, researchers recently increasingly use the term cardiorenal syndrome (CRS). Shlipak (2004) defines CRS as the simultaneous presence of heart and kidney dysfunction in a patient. Along with numerous methods for assessing the functional ability of the kidneys, the study of intrarenal blood flow is of particular importance in order to early identify its dysfunction. One of these is the ultrasound dopplerographic examination of the renal arteries.

Key words: chronic heart failure, cardiorenal syndrome, chronic kidney disease.

СУРУНКАЛИ ЮРАК ЕТИШМОВЧИЛИГИДА БУЙРАК ФУНКЦИЯСИНИНГ БУЗИЛИШИ

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✓ ***Резюме***

21-асрда сурункали юрак етишмовчилиги жамиятнинг тиббий ва ижтимоий муаммоларидан бири бўлиб қолмоқда, чунки унинг тарқалиши, ривожланиши, салбий оқибатлари ва иқтисодий харажатлари юқори. Сурункали юрак етишмовчилиги муҳим тиббий, ижтимоий ва иқтисодий муамммо бўлиб, юрак қон томир касалликларининг янги эпидемияси ҳисобланиб, дунё аҳолисидан 23 млн кишини қамраб олган. Кўплаб изланишлар буйраклар фаолиятини бузилиши чап қоринча систолик дисфункцияси мавжуд беморларда юрак етишмовчилиги ривожланиши билан чамбарчас боғлиқлигини кўрсатмоқда. Бундан ташқари юрак ишемик касаллигининг нохуш кечиши ва зўрайиб борувчи юрак етишмовчилигидан беморлар ўлимида буйрак дисфункцияси кучли мустақил хавф омили ҳисобланади. Юрак ва буйракнинг узвий боғлиқлигини ҳисобга олиб охирги йилларда изланувчилар кардиоренал синдром (КРС) тушунчасидан фойдаланмоқдалар. Shlipak (2004) КРСни беморда бир вақтнинг ўзида юрак ва буйраклар дисфункцияси мавжудлиги сифатида баҳолайди. Буйракларнинг функционал имкониятларини баҳоловчи кўплаб услублар билан бир қаторда интраренал қон оқимини, унинг дисфункциясини эрта аниқлаш мақсадида ўрганиш муайян аҳамиятга эга. Буйрак томирларини доплерографик текшируви ана шундай усуллардан бири ҳисобланади.

Калит сўзлар: сурункали юрак етишмовчилиги, кардиоренал синдром, сурункали буйрак касаллиги, допплерографик текширув.

НАРУШЕНИЕ ФУНКЦИИ ПОЧЕК ПРИ ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТИ

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✓ ***Резюме***

Хроническая сердечная недостаточность (ХСН) остается одной из медицинских и социальных проблем общества в 21 веке с ее распространенностью, прогрессированием, неблагоприятными последствиями ее течения и высокими экономическими расходами. Хроническая сердечная недостаточность (ХСН) является финальной стадией различных заболеваний сердца, характеризующейся истощением резервных возможностей миокарда и системных компенсаторных механизмов. ХСН представляет собой новую эпидемию сердечно-сосудистых заболеваний. Одним из главных нерешенных вопросов остается ранняя диагностика повреждений, которая позволяет предотвратить органные повреждения или замедлить прогрессирование дисфункции сердца и почек. Учитывая значительный вклад ХСН в структуру заболеваемости и смертности населения, необходимо разработать стратегию своевременной диагностики хронической болезни почек у больных и разработать нефропротективные стратегии для лечения. Такая тактика позволит предупредить обострение прогрессирования этих патологий, что приведет к уменьшению повторных госпитализаций, продлению продолжительности и улучшению качества жизни больных с КРС.

Ключевые слова: хроническая сердечная недостаточность, сопуствующая патология, хроничесая болезнь почек, кардиоренальный синдром

Relevance

Despite the achievements of modern cardiology, chronic heart failure (CHF) still remains 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 functional class IV (FC), mortality within six months reaches 44% [1-3].

The link between FC CHF and patient survival is recognized by almost all researchers. It seems obvious that the higher the CHF FC, the worse the prognosis. However, a linear relationship between the FC of CHF and mortality of patients is not always observed. The results of a comparative study of the survival of patients with ischemic heart disease (IHD) and symptoms of decompensation and without signs of CHF (n = 1964), conducted by R. Califf et al. [4], showed that only terminal stages (FC IV) of CHF play the role of an independent predictor of a poor prognosis (80% of mortality within 3 years), while in FC I-III, the survival rates are approximately the same: mortality is 38-42 %. Various conditions, which by themselves usually do not lead to CHF, can be the immediate cause of CHF decompensation [5].

Heart and kidney disease 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 conditions comorbid with CHF. A decrease in myocardial contractility leads to a deterioration in the functional state of the kidneys [6, 7], which, in turn, can cause the progression of CHF.

A number of retrospective studies have established a relationship between the course of CHF and DP, which is accompanied by a deterioration in the prognosis of patient survival [8, 9]. It is believed that the presence of DP in patients with CHF may be a predictor of an unfavorable clinical outcome [1]. However, the degree of DP is not indicated in the diagnosis and is not corrected.

DP in CHF may be associated with the addition of concomitant pathology of the kidneys and renal vessels, however, more than two-thirds of CHF patients without concomitant primary renal pathology have chronic kidney disease (CKD) [10], the prevalence of which among patients with decompensated CHF is 50-70 % [11-13]. DP significantly worsens the prognosis in people with CHF and low left ventricular ejection fraction (LVEF).

The randomized trials SOLVD and SAVE have shown an association between LTP and mortality in patients with LV systolic dysfunction [14]. With a decrease in the glomerular filtration rate (GFR) <60 ml / min / 1.73 m2, the risk of mortality increased by 2.1 times, with a reduced systolic LV 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 [15]. In a 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% [16].

LTP is an independent predictor of poor prognosis of CHF, although the pathogenesis of transient deterioration of renal function during decompensation of CHF remains unclear. 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, in persons with chronic kidney damage, which has arisen against the background of a disease of the urinary system, damage to the cardiovascular system develops, aggravating the course of the underlying disease. Obviously, the primary nature of diseases of the kidneys and the cardiovascular system is conditional (cardiorenal or renocardial syndrome), since damage to one organ invariably leads to a deterioration in the function of another.

Currently, the kidneys have come to be regarded as an organ contributing not only to the formation of edema syndrome, but also to the progression of myocardial dysfunction. This is due to the fact that the kidneys, by increasing the preload, promote LV dilatation, and by producing renin and activating the renin-angiotensin-aldosterone system - the development of hypertrophy and myocardial fibrosis.

Over time, patients with CHF may develop DP, in some cases progressing to chronic renal failure (CRF) [3]. 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 efferent arterioles prevails over the narrowing of the efferent arterioles. Nitric oxide, natriuretic peptides, prostaglandins E2 and E12 have a vasodilating effect on the bringing arterioles. As a result, despite a decrease in renal blood flow, in the early stages of CHF, renal perfusion pressure and filtration fraction (FF) increase, 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 the concentration of serum creatinine increases.

That is, over time, a significant number of patients with CHF develop chronic renal failure. 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 a breakdown of compensatory mechanisms [1, 3, 17].

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 the serum creatinine concentration in patients with CHF was shown in the mid-1990s. In 2000, Hillege et al. [18] calculated GFR using the Cockroft-Gault formula in patients with CHF III-IV FC according to NYHA and LVEF <35% included in the PRIME-II study. They showed that GFR is an independent predictor of general and cardiovascular mortality, even stronger than LVEF and heart failure 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. [19], having retrospectively analyzed the data from the SOLV'D Treatment and SOLVD Prevention studies, confirmed that the calculated GFR values are an important factor determining the survival rate of patients with CHF.

Other studies have also shown that a decrease in GFR can be an independent predictor of cardiovascular mortality in CHF [20, 21]. According to some researchers [3, 19, 22], the state of the kidneys 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 [1] and that in CHF, the significance of LTP as a predictor of an unfavorable prognosis is as great as LVEF and FC CHF.

In 2001 L.M. Ruilope et al. [23] proposed two main criteria for the diagnosis of LTP: an increase in serum creatinine concentration or a decrease in GFR and the presence of microalbuminuria (MAU) or macroalbuminuria (proteinuria).

The earliest marker of kidney damage is MAU. There is a hypothesis that MAU is a manifestation of a generalized violation of endothelial permeability and increases the risk of complications from the cardiovascular system. Currently, MAU is considered as one of the important predictors of the risk of cardiovascular complications and can be used in the diagnosis of DP in CHF [24, 25]

As for the determination of GFR (by the level of creatinine), according to the available data, an increase in the level of creatinine in the blood serum does not always reflect changes in GFR. However, apart from GFR (in terms of creatinine level), which depends on the anthropometric and gender data of patients, there are no other methods and markers for determining LTP 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 [26-28]; anthropometric and gender data. A. Moran et al. [29] believe that levels of cystatin C are linearly related to 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 renal pathology that develops with complications of coronary artery disease; its increased urinary excretion may precede an increase in serum creatinine levels [30].

In one of the studies [31], a direct relationship was determined between the indicators of endothelial dysfunction, CHF FC and the severity of proteinuria measured by the A1M content in urine. Therefore, A1M can also be considered as an indicator of endothelial dysfunction in patients with CHF. Obviously, to determine LTP in patients with CHF, one can use the determination of A1M in urine and cystatin C in the blood serum, as well as GFR (by the level of cystatin C).

Existing studies were carried out mainly with the participation of patients with comorbid pathology, and in fact there is no work on the study of 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.

Thus, the available scientific and clinical data indicate that the kidneys play an important role in the progression and prognosis of CHF. At the same time, it is noted that even a slight decrease in renal function significantly aggravates the course of the underlying cardiac pathology, while increasing the frequency of complications and the risk of death, and, on the contrary, the deterioration of the contractile function of the myocardium on the work of the kidneys is reflected in the most negative way [35].

Thus, the development of renal dysfunction in patients with CHF should be considered the cardiorenal syndrome, the main manifestations of which are a decrease in GFR and an increase in EAM (micro- or macroalbuminuria). The serum creatinine concentration does not sufficiently characterize renal function, therefore, the algorithm for examining patients with CHF should include an assessment of GFR.

Determination of GFR using the CKD EPI (Kidney Disease Epidemiology Collaboration) formula is the main routine method for assessing renal impairment in patients with CHF. A decrease in GFR is consistently associated with a worse prognosis in patients with CHF. Cardiovascular risk increases significantly with GFR less than 60 ml / min / 1.73 m2 [36].

In addition, in all patients with CHF, daily urinary albumin excretion should be determined. With a single detection of micro- or macroalbuminuria, one can suspect the presence of renal dysfunction and recommend re-examination. Microalbuminuria (MAU) is considered an early marker of kidney damage and may be associated with decreased GFR or present in isolation, as previously discussed in the CRS criteria. Standard albumin excretion is assessed by the ratio of albumin to urine creatinine: below 30 mg / g is defined as a normal indicator, 30-299 mg / g - MAU more than 300 mg / g - macroalbuminuria. There was a significant increase in the incidence of MAU and macroalbuminuria in patients with CHF according to the CHARM and GISSI-HF sub-studies, which was significantly associated with unfavorable outcomes [41].

Cystatin C is one of the key biomarkers reflecting the renal filtration capacity, as it freely passes through the basement membrane of the renal glomeruli, then is reabsorbed, but not secreted in the tubules. Many studies have demonstrated the superiority of the formula with cystatin C over creatinine in assessing the estimated GFR, especially in patients with moderate renal dysfunction [42].

The role of cystatin C for the diagnosis of cattle in patients with CHF continues to be studied, while for the diagnosis of cattle type 1 in acute heart failure, the marker has 90% sensitivity and 77% specificity [39].

One of the main unresolved issues is the early diagnosis of damage, which allows you to prevent organ damage or slow the progression of heart and kidney dysfunction. Changes in the functional state of the kidneys in CHF, which have a high prognostic value, in practice are detected mainly by clinical and laboratory parameters.Through the renal artery, the kidneys receive about 15-25% of the blood ejected by the left ventricle. Doppler indices of blood flow in the arteries of the kidneys using the example of the resistance index and the maximum blood flow velocity reflect not only local intrarenal processes, but also systemic hemodynamic processes [38]. The change in the renal artery resistance index is considered as a prognosis of the progression of CHF. Ultrasound methods allow expanding the diagnostic capabilities to identify subclinical changes in this contingent of patients [40]. This study of renal vessels in chronic heart failure in dynamics is quite informative for assessing the patient's condition.

In patients with HF, especially those with pre-existing signs of renal dysfunction, a progressive increase in serum creatinine levels is often observed after administration of RAAS blockers against the background of diuretic therapy, as well as in combination with X-ray contrast agents, nephrotoxic antibiotics. It is the impaired renal function that most restricts the use of these classes of drugs in patients with CHF, this always leads to a significant decrease

in the effectiveness of treatment of the latter, especially from the standpoint of improving the long-term prognosis. At the same time, it should be borne in mind that in most patients with heart failure, deterioration of renal function during initiation of ACE inhibitors or ARB therapy is expected, usually insignificant, and in most cases should not be considered as a reason for cancellation [37].

In addition, many drugs with the renal route of excretion (digoxin, insulin) can accumulate in patients with reduced renal function, and therefore it is necessary to adjust the dose of these drugs taking into account the level of GFR and sometimes control their concentration in the blood plasma.

Many studies have shown that the progression of CKD is slowed down by strict control of blood pressure and glycemia, the use of angiotensin converting enzyme inhibitors (unless contraindicated) or angiotensin II receptor antagonists

Considering the significant contribution of CHF to the structure of morbidity and mortality in the population, the unfavorable prognostic value of the development of CKD in this contingent of patients, it is necessary to develop a strategy for the timely diagnosis of CKD in patients with CHF. This tactic will prevent the exacerbation of the progression of these pathologies, which will lead to a decrease in re-hospitalizations, an extension of the duration and an improvement in the quality of life of patients with cattle.

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