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FEATURES OF THE COURSE OF CHRONIC HEART FAILURE IN PATIENTS WITH KIDNEY DYSFUNCTION

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Abstract: Chronic heart failure (CHF) is the outcome of many cardiovascular diseases. Despite significant advances in the field of medical and surgical treatment of such patients, the prevalence of CHF not only does not decrease, but continues to increase [12], especially among elderly and senile people [2]. The prognosis for patients with severe CHF is unfavorable. According to the Framingham study, after the onset of clinical symptoms of CHF, about 80% of men and 65% of women die within 6 years [5]. With CHF IV class (according to NYHA), mortality within six months reaches 44%.

Key words: cardiovascular diseases, glomerular filtration rate, chronic renal failure, chronic heart failure.

Kidney involvement in many diseases, initially not considered renal, makes it necessary to develop unified approaches to the management of patients with identified chronic renal failure (CHF), especially in terms of early warning and treatment of its complications: anemia, disorders of phosphorus-calcium metabolism, which significantly worsen the prognosis of other diseases. One of the pathogenetic mechanisms of CHF is the activation of a number of neurohumoral systems, among which special importance is attached to the sympathetic-adrenal, renin-angiotensin systems, vasopressin and the natriuretic peptide system. A decrease in glomerular filtration rate (GFR) is considered as a marker of poor prognosis for common diseases in the population, primarily CVD, which is fully consistent with the established concept of cardiorenal relationships.

The mechanism of development of kidney dysfunction in patients with CHF has not been fully studied. It is believed that renal dysfunction is caused mainly by a drop in cardiac output and neurohumoral activation. [63]. A fall in cardiac output leads to a decrease in renal blood flow. As a result, blood pressure on the walls of the afferent arterioles and sodium delivery to the ascending loop of Henle are reduced.

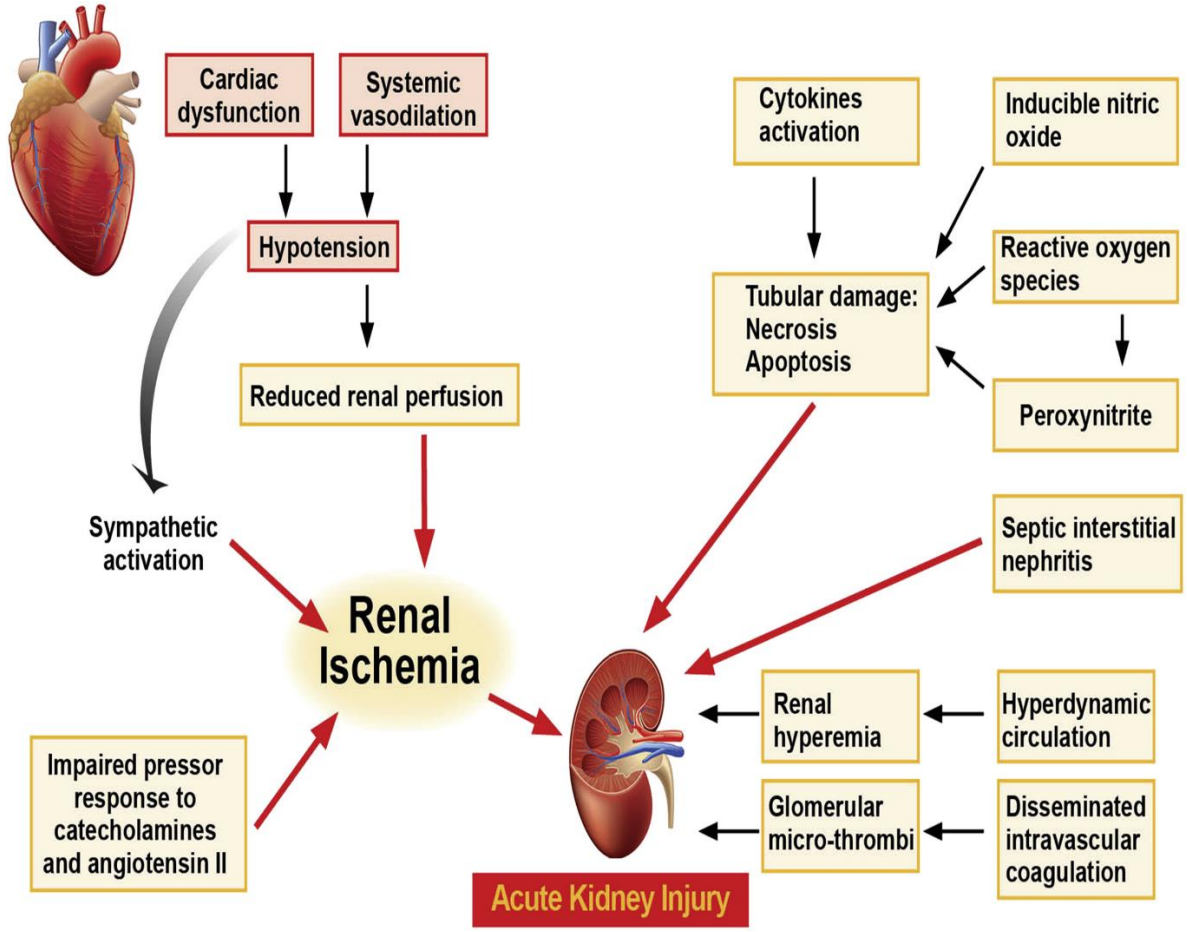
CHF is characterized by significant disturbances in renal hemodynamics, including an increase in renal vascular resistance, a decrease in GFR and a more significant decrease in renal plasma flow (RPF), which causes an increase in the filtration fraction (the ratio of GFR to RFP, expressed as a percentage) [4].

Renal blood flow (RBC) normally accounts for 15-20% (≈600 ml/min) of cardiac output and is very sensitive to changes in the systemic circulation. The reduction in cardiac output observed in systolic HF induces a redistribution of blood flow, one consequence of which is the development of renal vasoconstriction, which includes both pre- and post-glomerular arterioles, but with more significant constriction of the efferent arterioles. This adaptation at certain stages of CHF maintains GFR at normal levels and is characterized by an increase in the filtration fraction. Systemic and local adaptation processes are the result of activation of various systemic and local hormonal and humoral regulatory systems, such as

the SNS, RAAS, as well as arginine vasopressin, atrial and brain natriuretic peptides, nitric oxide, prostaglandins, endothelin, etc.

The term "cardiorenal" has been used in the medical literature for decades to describe the hemodynamic and neurohumoral connection between the heart and kidneys. In recent years, the term “cardiorenal syndrome” (CRS) has become widespread, the essence of which is considered ambiguously by various authors [7,66]. Some authors believe that this term should be used to characterize patients with a combination of severe heart and renal failure [3], others recommend using the level of serum Cr as one of the Cr criteria [8].

Definition of CRS “as a pathophysiological disorder of the heart and kidneys, resulting in acute or chronic dysfunction, chronic activation of vasoconstrictor systems can directly lead to damage to myocardial structures and cardiac remodeling, in particular under the influence of angiotensin II and aldosterone, promoting vascular remodeling, tissue fibrosis, oxidative stress and inflammation [1].



Pathophysiological mechanisms of development of chronic cardiorenal syndrome

In order to characterize the functional state, according to the recommendations of VNOK (2008), it is proposed to use the following terms (National Kidney Foundation. 2007; Russian recommendations second revision, 2008).

Chronic kidney disease (CKD) reflects the presence of kidney damage and/or GFR characteristics.

CKD criteria:

- Kidney damage ≥ 3 months, with decreased GFR or
- GFR < 60 ml/min/1.73 m2 ≥ 3 months, with or without kidney damage.

Kidney damage is structural or functional abnormalities of the kidneys. They may initially appear when GFR is normal, but may lead to a decline over time. Markers of kidney damage include abnormalities in the blood or urine and in the results of imaging studies.

Classification of CKD is based on GFR, calculated using the MDRD formula, and the presence of renal damage (Table 1). Calculation of GFR using the MDRD formula is recommended as a classifying indicator of the functional state of the kidneys.

1) the MDRD formula is the most reliably validated for estimating GFR in adults,

2) to calculate GFR, this method uses readily available data (elementary demographic data and serum CR),

3) the indicator can be calculated automatically and presented in the laboratory report.

Stages of Chronic Kidney Disease		GFR*	% of Kidney Function
Stage 1	Kidney damage with **normal** kidney function	90 or higher	
Stage 2	Kidney damage with **mild loss** of kidney function	89 to 60	
Stage 3a	**Mild to moderate** loss of kidney function	59 to 45	
Stage 3b	**Moderate to severe** loss of kidney function	44 to 30	
Stage 4	**Severe** loss of kidney function	29 to 15	
Stage 5	Kidney **failure**	Less than 15	

**Your GFR number tells you how much kidney function you have. As kidney disease gets worse, the GFR number goes down.*

In the development of chronic renal failure, the most important point is the slow, hidden impairment of all renal functions, which the patient is usually unaware of. However, modern examination methods make it possible to identify the hidden stage, since the changes that occur in the body when the functional capacity of the kidneys is impaired are now well known. With chronic renal failure, one of the most important functions of the kidneys is disrupted - maintaining water-salt balance. Along with disturbances in the water-electrolyte balance in chronic renal failure, the catabolism of many body proteins changes, which leads to a delay in the body of urea, calcium, uric acid, indole, guanidine, organic acids and other products of intermediate metabolism. Regardless of the disease that led to renal failure, the morphological picture in the kidneys boils down to the development of fibroplastic processes with the replacement of nephrons with connective tissue, hypertrophy of existing nephrons.

Conclusion.

Despite the large number of studies devoted to studying the effect of CKD on the prognosis of CHF, published in recent years, the influence of the functional state of the kidneys on the clinical features of CHF,

as well as the features of the course of CHF associated with kidney dysfunction, remain not fully studied.

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