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CHRONIC KIDNEY DISEASE: IMPACTS ON MENTAL STATE, QUALITY OF LIFE AND CARDIOVASCULAR OUTCOMES

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Abstract: This review delves into the intricate relationship between chronic kidney disease (CKD), cardiorenal syndrome (CRS), and chronic heart failure (CHF), focusing on their collective impact on patients' mental health and quality of life. CKD is associated with a significantly elevated risk of cardiovascular diseases, with the prevalence being 64% higher in individuals with impaired renal function compared to those with normal kidney function. An inverse correlation has been documented between a glomerular filtration rate (GFR) below 60 ml/min/1.73 m^2 and an increased risk of mortality, cardiovascular complications, and hospitalizations. The progression of CKD amplifies cardiovascular risks, with stage 2 patients experiencing a 4.8% incidence of complications, nearly doubling by stages 3-4. Dialysis and kidney transplant recipients exhibit a tenfold higher likelihood of adverse cardiovascular events compared to the general population. Beyond physical complications, CKD imposes a substantial psychological toll, manifesting as depression, anxiety, and social withdrawal, which critically diminish quality of life. This review highlights the importance of comprehensive management strategies, including early intervention, cardiovascular risk mitigation, and psychosocial support, to improve outcomes for CKD patients.

Key words: cardiorenal syndrome, medical and social aspects, acute kidney injury (AKI).

SURUNKALI BUYRAK KASALLIGI: RUHIY HOLATGA, HAYOT SIFATIGA VA YURAK-QON TOMIR NATIJALARIGA TA'SIRI

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Annotatsiya. Mazkur maqolada surunkali buyrak kasalligi (SBK), kardiorenal sindrom (KRS) va surunkali yurak yetishmovchiligi (SYY) o'rtasidagi murakkab munosabatlar o'rganilib, ularning bemorlarning ruhiy salomatligi va hayot sifatiga bo'lgan umumiy ta'siri tahlil qilingan. SBK yurak-qon tomir kasalliklari xavfining sezilarli darajada oshishi bilan bog'liq bo'lib, buyrak funksiyasi buzilgan odamlarda buyrak funksiyasi normal bo'lganlarga nisbatan bu kasallikning tarqalishi 64% ga yuqori. Koptokcha filtratsiya tezligi (KFT) 60 ml/min/1,73 m^2 dan past bo'lsa, o'lim, yurak-qon tomir asoratlari va kasalxonaga yotqizish xavfining ortishi o'rtasida teskari bog'liqlik aniqlangan. SBK rivojlanishi yurak-qon tomir xavflarini oshiradi, 2-bosqichdagi bemorlarda 4,8% asoratlar kuzatiladi, bu esa 3-4 bosqichlarda deyarli ikki baravar oshadi. Dializ yoki buyrak transplantatsiyasini o'tkazgan bemorlarda yurak-qon tomir kasalliklari xavfi umumiy aholiga nisbatan 10 baravar yuqori. Jismoniy asoratlardan tashqari, SBK sezilarli psixologik bosim ko'rsatadi, bu esa depressiya, tashvish va ijtimoiy cheklanish shaklida namoyon bo'lib, hayot sifatini sezilarli darajada pasaytiradi. Ushbu maqolada SBK bilan og'rigan bemorlarni davolash natijalarini yaxshilash uchun erta aralashuv, yurak-qon tomir xavflarini kamaytirish va psixologik yordamni o'z ichiga olgan kompleks davolash yondashuvining muhimligi ta'kidlangan.

Kalit so'zlar: kardiorenal sindrom, tibbiy va ijtimoiy jihatlar, o'tkir buyrak yetishmovchiligi (OBY).

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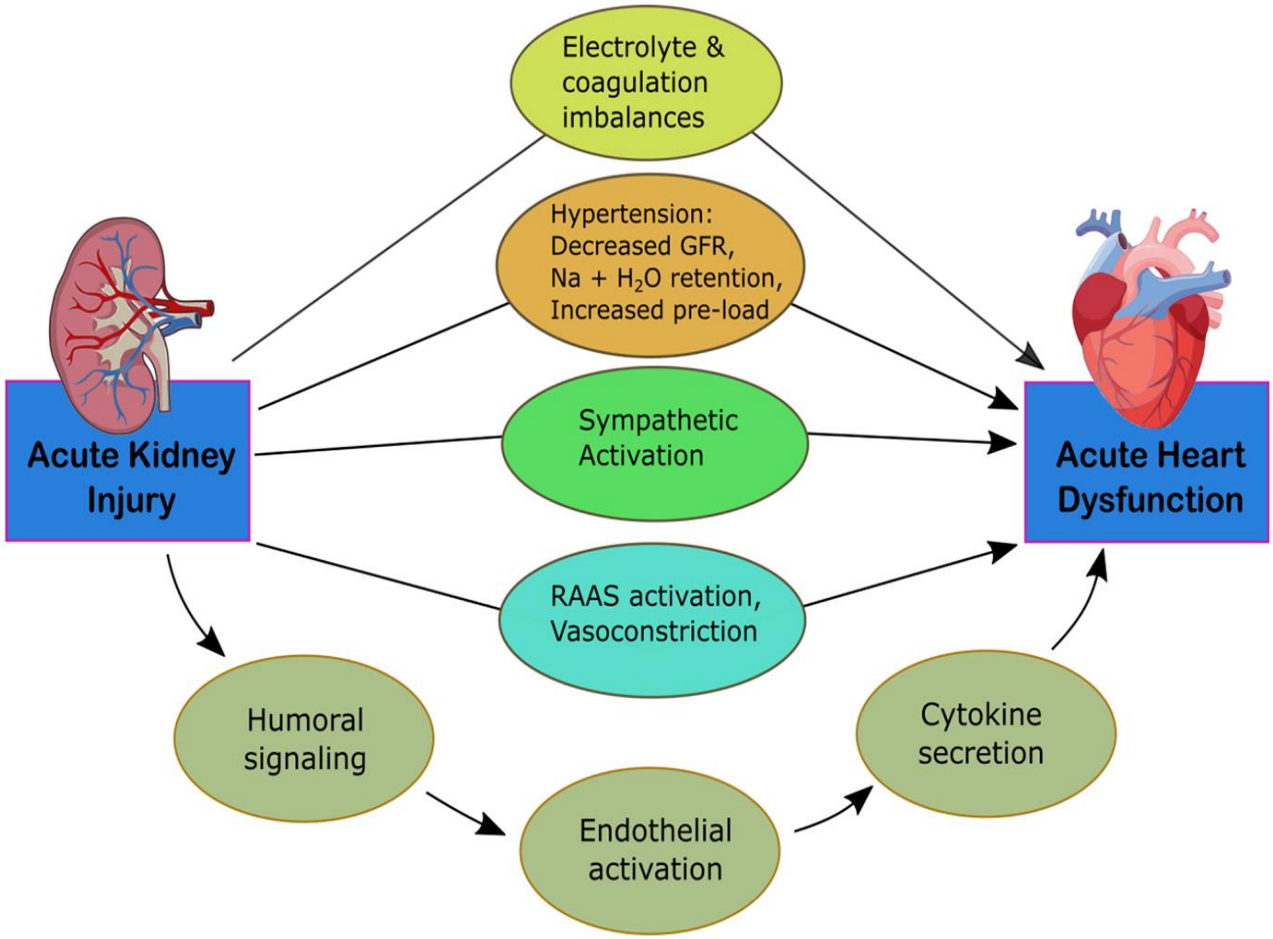
Currently, CRS is a pathological interdependent condition involving the heart and kidneys, developing as a result of acute or chronic dysfunction of one of the organs with

subsequent acute or chronic dysfunction of the other. The center of pathogenesis of cardiorenal syndrome is the activation of the renin-angiotensin-aldosterone system (RAAS) and sympathetic hyperactivation. It has long been known that the kidneys control the volume of extracellular fluid by regulating the processes of excretion and reabsorption of sodium, and the heart controls systemic hemodynamics. As studies have shown, when the heart and/or kidneys are damaged, chronic activation of the sympathetic nervous system and RAAS occurs, endothelial dysfunction and systemic inflammation develop, and a vicious circle is formed in which the combination of cardiac and renal dysfunction leads to an accelerated decrease in the functional capacity of each organ, myocardial remodeling, damage to the vascular wall and renal tissue ultimately lead to increased morbidity and mortality. In particular, a quarter drop in cardiac output leads to a two-fold decrease in renal perfusion. It should be emphasized that initially the glomerular filtration rate is relatively stable due to an increase in the filtration fraction through autoregulation and afferent vasoconstriction induced by angiotensin II. Therefore, a decrease in glomerular filtration rate is associated with a worse overall prognosis in patients with both cardiovascular diseases and renal dysfunction[1]. On the other hand, damage to the heart and kidneys is accompanied by hyperproduction of cytokines, increased migration of monocytes and neutrophils, which caused the progression of endothelial dysfunction and induced remote organ dysfunction.



[1] Шутов АМ, Серов ВА. Кардиоренальный континуум или кардиоренальный синдром? // Клиническая нефрология.2010;1:44-48.

In 2008, at the ADQI consensus conference in Venice, C. Ronco et al. proposed a definition and classification of cattle, in which they identified five types.

Type 1 – acute CRS. Acute cardiac dysfunction (cardiogenic shock, acute decompensation of chronic heart failure – CHF) significantly reduces cardiac output and increases venous pressure. Perfusion of the kidneys and their filtration capacity decrease, which leads to acute kidney injury (AKI) and subsequently to the development of chronic kidney disease (CKD).

Type 2 – chronic CRS. Characterized by the presence of chronic cardiac pathology, primarily CHF, leading to the development or progression of CKD. Renal dysfunction in patients with CHF is detected in 45.0-63.6% of cases. Systolic and diastolic dysfunction of the left ventricle (LV) leads to prolonged renal hypoperfusion against the background of micro- and macroangiopathies, severe neurohormonal disorders: increased production of vasoconstrictors (adrenaline, angiotensin II, endothelin), changes in the sensitivity and release of endogenous vasodilators (natriuretic peptides, nitric oxide). A combination of cardiovascular risk factors (arterial hypertension, dyslipidemia, hyperuricemia) increases the likelihood of developing CKD. The development of hypertensive nephrosclerosis in arterial hypertension (AH) is a common cause of CKD and is significantly accelerated by hyperuricemia, hyperglycemia and dyslipidemia. Moderate decrease in SCF in essential AH leads to a doubling of the risk of death. At different time periods of the disease, transformation of acute and chronic CRS (types 1 and 2) is possible.

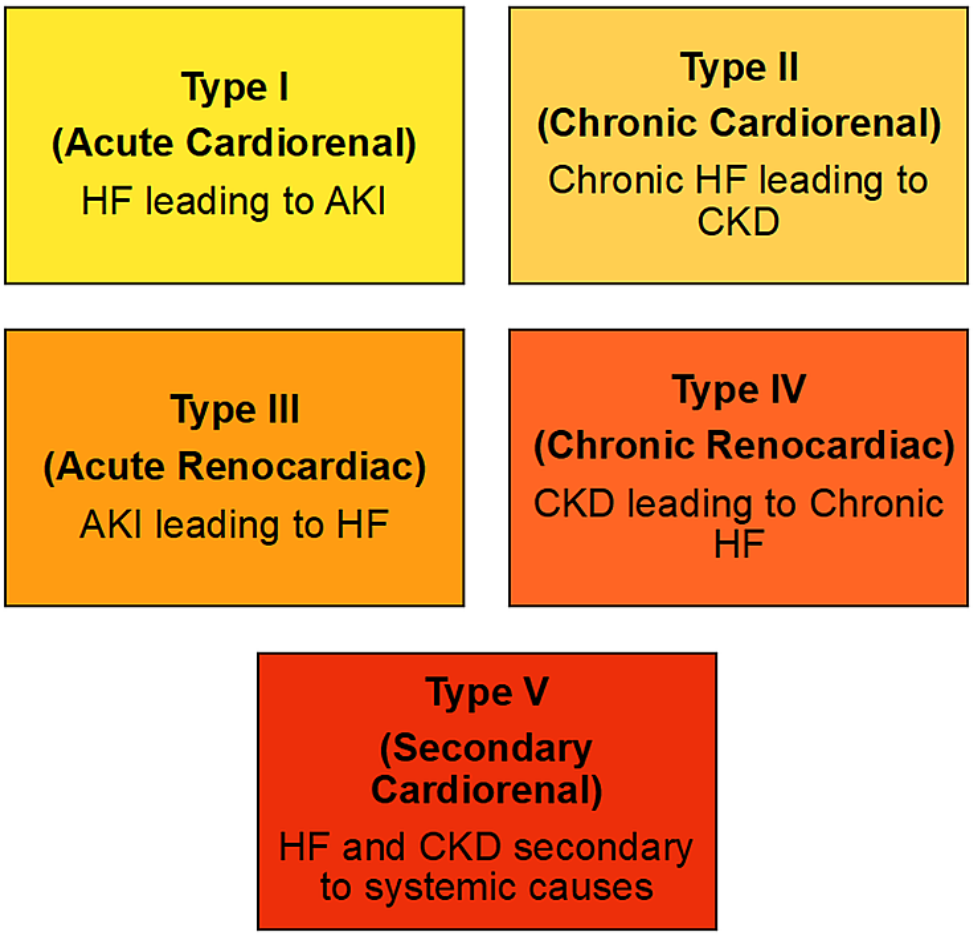
Type 3 CRS - acute renocardial syndrome. Characterized by a primary, sudden deterioration in renal function (for example, in acute glomerulonephritis or pyelonephritis, acute tubular necrosis, acute urinary tract obstruction), which leads to acute cardiac dysfunction (ACH, arrhythmia, ischemia). AKI (acute kidney injury) is frequently observed in hospitalized patients and in patients in the intensive care unit, in 9 and 35% of cases, respectively. The prevalence of AKI in coronary angiography (contrast-induced nephropathy) and cardiac surgery ranges from 0.3 to 29.7% and is associated with high mortality. Acidosis developing in renal failure, contributing to the development of pulmonary vasoconstriction and right ventricular failure, has a negative inotropic effect and, in addition to electrolyte disorders, increases the risk of arrhythmia. In addition, renal ischemia itself can provoke inflammation and apoptosis of cardiomyocytes.

A special form of this type of CRS is renal artery stenosis. Blockade of the renin-angiotensin-aldosterone system (RAAS) is a necessary component of therapy for such patients, however, in case of bilateral renal artery stenosis or stenosis of the artery of a single kidney the use of these drugs can lead to decompensation of renal failure. In severe AKI requiring renal replacement therapy (RRT), hypotension, rhythm and conduction disturbances, myocardial ischemia due to rapid fluid and electrolyte shifts during dialysis may develop.

Type 4 CRS – chronic renocardial syndrome. This is a situation where primary CKD leads to impaired cardiac function (ventricular hypertrophy, diastolic dysfunction or increased risk of adverse cardiovascular events). The main cause of kidney damage is diabetes mellitus (DM) type 2 and hypertension, a significant role is played by atherosclerosis, CHF and obesity. In patients with pre-dialysis CKD, the prevalence of cardiac pathology, overall and cardiac mortality correlate with the severity of renal dysfunction. The prevalence of cardiovascular disease in the population of patients with reduced renal function is 64% higher than in those

with preserved function. An independent inverse relationship was found between SCF <60 ml/min/1.73 m2 and an increased risk of death, cardiovascular complications, and hospitalization. The incidence of new cardiovascular complications is 4.8% in patients with stage 2 CKD and almost doubles in stages 3-4. The risk of developing adverse cardiovascular outcomes in patients on dialysis or in kidney transplant recipients is tens of times higher than in the general population .

Type 5 KRS - secondary CRS. Characterized by the presence of combined renal and cardiac pathology due to acute or chronic systemic diseases, while the dysfunction of one organ affects the functional state of another, and vice versa. Such diseases include sepsis, diabetes, amyloidosis, systemic lupus erythematosus, and sarcoidosis. Data on the prevalence of type 5 CRS are scarce due to the large number of acute and chronic predisposing conditions.



A persistent decrease in hemoglobin concentration below 120 g/l in women and below 130 g/l in men indicates anemic syndrome in chronic kidney disease. Accumulated clinical experience shows that anemia in chronic kidney disease develops long before the appearance of signs of uremia and serves as a cause of the progression of renal and cardiovascular complications. In conditions of anemia, the production of angiotensin II increases many times, which is accompanied by the development of fibrotic changes both in the heart and in the kidneys. Whereas, correction of renal anemia using erythropoietin preparations reliably slows the progression of chronic renal failure and significantly reduces the risk of cardiovascular diseases.

It was shown that in patients with chronic heart failure, hyperkalemia was detected in 11.2% of cases, and hypokalemia in 28.0% of cases. In the pre-dialysis stage of chronic kidney disease, persistent hyperkalemia serves as a predictor of a decrease in the glomerular filtration rate. Therefore, starting from the pre-dialysis stage of chronic kidney disease, it is necessary to ensure regular monitoring of blood potassium, especially in individuals taking drugs that block the activity of the RAAS.

In the study by I. T. Murkamilov and co-authors, bilateral cardiorenal relationships in nephrotic syndrome were analyzed taking into account gender characteristics[1]. It was shown that the average indicators of systolic, diastolic, pulse and mean arterial pressure are significantly higher in male patients with nephrotic syndrome. At the same time, they more often had supraventricular and ventricular ectopic activity. As the researchers emphasize, in the subgroup of women with nephrotic syndrome, sinus tachycardia, slowing of impulse conduction along the bundle branches of His, and impaired repolarization processes of the left ventricle were detected much more often.

In another study, which examined gender-specific characteristics of cardiorenal relationships and cytokine status in patients with chronic kidney disease, it was found that the diameter of the aortic outflow tract, the thickness of the intima-media complex of the carotid arteries, the size of the left atrium, the end-diastolic size of the left ventricle, the thickness of the interventricular septum and the mass of the myocardium of the left ventricle were significantly higher, while the ejection fraction of the left ventricle, on the contrary, was significantly lower in men compared to women[2].

Conclusion. Similarities in common risk factors, mechanisms of development and mutual aggravation of cardiovascular diseases and renal dysfunction substantiate the position of cardiorenal syndrome, which requires interdisciplinary care from both cardiologists and nephrologists, as well as health care organizers, in order to ensure a high level of medical (specialized) care with the maximum favorable outcome of therapy and a minimum of side effects associated with treatment

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