CARDIORENAL SYNDROME: PATHOPHYSIOLOGY, VERIFICATION

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Abstract. *The connection between cardiac and renal pathology has long attracted the attention of both cardiologists and neurologists. A high risk of cardiac death in terminal renal failure was noted soon after the introduction of hemodialysis into clinical practice, but only in the last decade it became clear that any kidney damage, both acute and chronic, is also associated with high general and cardiovascular mortality. These data, obtained in large randomized trials, became the basis of the concept of chronic kidney disease (CKD), developed under the auspices of the NKF (National Kidney Foundation, USA).*
Keywords: *hypoperfusion, hypovolemia.*

Recently, due to the increasing prevalence of cardiovascular pathology, an increase in the life expectancy of cardiological patients and the use of interventional methods of examination and treatment, the incidence of acute renal failure (ARF) has also been increasing [13]. Expert Group of Acute Dialysis Quality Initiative. The Group (ADQI) has developed the concept of acute renal injury (AKI) and defined it. For timely detection, assessment of the severity and treatment of renal dysfunction, a multi- level classification system RIFLE (Risk) was proposed, Injury, Failure, Loss of kidney function, End-stage kidney disease), further modified by the expert group Acute Kidney Injury Network (AKIN) [14, 15].

The need for early detection of kidney damage in cardiovascular pathology and diabetes mellitus for risk assessment, strategy development and tactics of patient management contributed to the emergence of such concepts as "cardiorenal syndrome" (CRS) (P. Ledoux, 1951), "cardiorenal anemic syndrome" (D.S. Silverberg, 2003) and " cardioreal continuum" (V.J. Dzau et al., 2005) [16-19]. This is evidenced by the change in diagnostic criteria and the assessment of the significance of renal dysfunction in the ESC/ESN recommendations [20]. However, the lack of an accurate definition and unified understanding of the pathophysiological mechanisms of cardiorenal interactions and clinical manifestations create difficulties for timely diagnosis and treatment. At the ADQI Conciliation Conference in Venice in 2008, C. Ronco et al. A classification was presented in which five types of cardiac syndrome were distinguished [24]. Definition. Cardiorenal syndrome is a pathophysiological disorder of the heart and kidneys, in which acute or chronic dysfunction of one of these organs leads to acute or chronic dysfunction of the other. Thus, cattle includes acute and chronic disorders, in which both the heart and the kidney can be the primary affected organ. A special feature was the declaration of the fundamental heterogeneity of the cardiorenal syndrome and the identification of its main five types depending on the presence of acute/chronic heart failure (HF), as well as the primary/ secondary occurrence of heart or kidney damage in relation to each other. Acute cattle (type 1) is characterized by an extraordinary deterioration of cardiac activity, leading to acute kidney injury (AKI), occurs in acute coronary syndrome (ACS) in 9-19% of cases [25, 26], and in cardiogenic shock - in 70% of cases [27]. Acute heart failure and acute decompensation of chronic heart failure are complicated by the development of AKI in 24-45% of patients [28-31]. OPP it usually develops in the first days of

hospitalization: 50% — in the first 4 days, 70-90% — in the first 7 days [32]. In case of OCD and ACS, the development of AKI is associated with higher general and cardiac mortality, longer hospitalization, frequency of rehospitalization and progression CKD if available. In HF, lethality is inversely proportional to the glomerular filtration rate (GFR), which is no less significant prognostic factor than the left ventricular ejection fraction (LVEF), and AKI is more severe in patients with reduced LVEF compared to patients with preserved LV [33-36]. The risk of adverse outcomes increases regardless of AKI resistance, even a slight increase in serum creatinine (by 0.3 mg/dl — 26.6 mmol/L) is associated with an increase in mortality, while more severe AKI is associated with a higher risk of death. The development of AKI in acute type 1 cattle is primarily caused by impaired renal perfusion due to a decrease in cardiac output and/or a significant increase in venous pressure. Resistance to diuretic therapy often develops: at the same time, the use of high doses or combinations of diuretics may be an additional iatrogenic mechanism for the progression of AKI. In hypervolemia resistant to diuretics, despite the optimization of cardiac output, it is possible to use extracorporeal ultrafiltration [37]. The presence of AKI with or without hyperkalemia limits the use of ACE inhibitors, angiotensin II receptor antagonists (ARA) and aldosterone antagonists among patients with HF and myocardial infarction, which can negatively affect the outcomes of the disease [38]. However, with careful monitoring of kidney function and potassium levels, the potential benefit of prescribing these drugs often outweighs the risk. Chronic cattle (type 2) is characterized by the presence of chronic cardiac pathology leading to the progression of CKD. Renal dysfunction is widespread among patients with chronic heart failure (CHF, 45-63,6 %) [39, 40] and is an independent negative prognostic factor in relation to the development of systolic and diastolic dysfunction LV, cardiovascular death, while a biological gradient was revealed between the severity of cardiac dysfunction and the deterioration of clinical outcomes [41, 42]. Prolonged renal hypoperfusion, preceded by micro- and macroangiopathy, is considered to be one of the main factors of injury in chronic cattle, primarily in CHF, although there is no direct link between a decrease in LVL and GFR, indicators of central hemodynamics and serum creatinine levels [43, 44].

Also, for Type 2 cattle are characterized by pronounced neurohormonal disorders: increased production of vasoconstrictors (adrenaline, angiotensin, endothelin), changes in sensitivity and/or release of endogenous vasodilators (natriuretic peptides, nitric oxide). The functional state of the kidneys may also worsen due to CHF therapy. The probability of developing renal dysfunction in patients with cardiac pathology is significantly higher than in the population, and the combination of any two factors of cardiovascular risk increases the likelihood of developing CKD by almost 4 times [8]. Arterial hypertension (AH) is a well—known and frequent cause of the development of CKD, while the development of hypertensive nephrosclerosis is significantly accelerated in the presence of metabolic disorders such as hyperuricemia, hyperglycemia and dyslipidemia [45, 46]. Even with an uncomplicated course of essential hypertension (according to the RIUMA study), a moderate decrease in GFR leads to a doubling of the risk of cardiac death, and blood pressure in the range of 130-139/85-89 mmHg is associated with an increase in the risk of microalbuminuria (MAU) more than twice as compared with the patient- mi with lower indicators. Acute renocardial syndrome (type 3 CATTLE) is characterized by primary, sudden deterioration of kidney function (for example, in acute glomerulonephritis or pyelonephritis, acute renal necrosis, acute urinary tract obstruction), which leads to acute cardiac dysfunction (HF, arrhythmias, ischemia). AKI is often observed in hospitalized patients and ICU

patients, reaching 9 and 35%, respectively. The prevalence of AKI in coronary angiography and cardiac surgery ranges from 0.3 to 29.7% [47, 48] and is associated with high mortality. AKI affects the functional state of the heart through several mechanisms [24]. Fluid overload can lead to the development of OSN, hyperkalemia - to the occurrence of arrhythmias and cardiac arrest, uremic intoxication reduces the inotropic function of the myocardium and leads to the development of pericarditis. Acidosis developing with renal insufficiency, contributing to the occurrence of pulmonary vasoconstriction and right ventricular insufficiency, has a negative inotropic effect and, in addition to electrolyte disorders, increases the risk of arrhythmias. In addition, renal ischemia can by itself provoke inflammation and apoptosis of cardiomyocytes. A special form of this type of cattle is renal artery stenosis [49]. Diastolic LV dysfunction associated with prolonged hypertension due to hyperactivation of the renin-angio- tensin-aldosterone system (RAAS), sodium and water retention on the background of impaired renal function and acute myocardial ischemia due to its increased oxygen demand on the background of peripheral- chesky vasoconstriction. RAAS blockade is an indispensable component of the therapy of such patients, however, with bilateral renal artery stenosis or artery stenosis of a single kidney, the use of these drugs can lead to decompensation of renal failure. In severe AKI requiring renal replacement therapy, hemodynamic instability may develop in the form of hypotension, rhythm and conduction disturbances, myocardial ischemia due to rapid movement of fluid and electrolytes during dialysis. Europe and Japan is 10-13%, reaching 20 in high-risk groups % [50, 51]. The main cause of kidney damage in recent years is type 2 diabetes mellitus (DM) and hypertension, atherosclerosis, CHF and obesity play a significant role, that is, diseases whose prevalence has been sharply increasing in recent decades. In patients with nondialysis CKD, the prevalence of cardiac pathology, general and cardiac mortality correlate with the severity of renal dysfunction [52]. There is growing interest in the pathogenetic role of relative or absolute erythropoietin deficiency in CKD, which can cause activation of apoptosis, fibrosis and inflammation in the myocardium [53]. Clinical studies have shown that the administration of erythropoiesis-stimulating drugs to patients with CHF, CKD and anemia leads to an improvement in the functional state of the heart, a decrease in LV size and a decrease in the level of cerebral natriuretic peptide [54]. Currently the primary importance of achieving a target blood pressure to slow the progression of renal dysfunction in patients with CKD has been proven, and the use of RAAS blockers in the maximum tolerated doses is recommended as the drugs of choice in these situations. Secondary cattle (type 5) is characterized by the presence of combined renal and cardiac pathology due to acute or chronic systemic disorders, while a violation of the function of one organ affects the functional state of another, and vice versa. Examples of such conditions are sepsis, diabetes, amyloidosis, systemic lupus erythematosus, sarcoidosis. Data on the prevalence of type 5 cattle are very scarce due to the large number of acute and chronic predisposing conditions. Sepsis is the most frequent and severe condition affecting the function of the heart and kidneys. It can lead to AKI, simultaneously causing deep myocardial depression, the mechanisms of development of these conditions are not fully understood. Prevalence AKI in sepsis is 11-64%, and the incidence of troponin is 30-80%, their combination is associated with an increase in mortality compared with the presence of only one of the conditions [58-60]. The development of functional myocardial depression and inadequate cardiac output lead to further deterioration of kidney function, as in type 1 Cattle, and AKI affects the functional state of the heart, as in type 3 cattle, resulting in a vicious circle that adversely affects the condition of both organs. In the treatment of this pathology, the same

principles are applied as in cattle 1st and 3rd types. Preliminary data on the use of intensive renal replacement therapy among patients with sepsis have shown that blood purification can play an important role in improving the functional state of the myocardium while ensuring optimal clearance, however, optimal schemes for the prevention and treatment of AKI in patients in critical condition have not yet been developed. Normal cardiorenal interactions are presented by A. Guyton (1990) in the form of a hemodynamic model in which the kidneys control the volume of extracellular fluid by regulating the processes of excretion and reabsorption of sodium, and the heart controls systemic hemodynamics [61]. The central links of this model are RAAS, endothelium-dependent factors and their antagonists — natriuretic peptides (NPS) and the kallikrein- kinin system. When one of the organs is affected, the RAAS and sympathetic nervous system are activated, endothelial dysfunction and chronic systemic inflammation develop, a vicious circle is formed in which a combination of cardiac and renal dysfunction leads to an accelerated decrease in the functional capacity of each of the organs, remodeling of the myocardium, vascular wall and renal tissue, an increase in morbidity and mortality. Thus, the direct and indirect effects of each of the affected organs on each other can lead to the appearance and preservation of combined heart and kidney disorders through complex neurohormonal feedback mechanisms [62]. Moreover, anemia, which is present in many patients with cattle, is included in this vicious circle, the frequency of its detection increases with an increase in the functional class of CHF according to NYHA, and the level of hemoglobin is inversely proportional to the size of the left ventricle of the heart and the severity of LVH [63, 64]. It is also important to remember about possible iatrogenic causes of cattle development. An uncontrolled increase in diuresis against the background of diuretic therapy can lead to hypovolemia and a decrease in preload, and the use of vasodilators can cause hypotension. In addition, nonsteroidal anti-inflammatory drugs, cyclosporine, ACE inhibitors and ARA II can also cause a decrease in renal perfusion.

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