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CLINICAL CHANGES IN THE CARDIOVASCULAR SYSTEM IN KIDNEY DISEASES

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Annotation: During a clinical update conference convened by the Kidney Disease - Improving Global Outcomes (KDIGO), an international group of experts defined the current state of knowledge and the implications for patient care in important topic areas, including coronary artery disease and myocardial infarction, congestive heart failure, cerebrovascular disease, atrial fibrillation, peripheral arterial disease, and sudden cardiac death[1,2].
Although optimal strategies for prevention, diagnosis, and management of these complications likely should be modified in the presence of CKD, the evidence base for decision making is limited.
Key words: atrial fibrillation, heart failure, peripheral arterial disease, stroke

Relevance. Cardiovascular diseases (CVD) are the most common cause of death in chronic kidney disease (CKD), which is an independent risk factor (RF) for the development of CVD and death. Heart and kidney diseases have common traditional risk factors [arterial hypertension (AH), diabetes mellitus (DM), obesity, dyslipidemia, etc.], and when they are combined, non-traditional renal factors also act (overhydration, anemia, disorders of phosphorus-calcium metabolism, systemic inflammation, hypercoagulation), which can also affect the risk of development and pathogenesis of CVD.

Numerous prospective studies have shown that even modest declines in kidney function are associated with an increased risk of cardiovascular morbidity and death.

The central links of this model are the renin-angiotensin-aldosterone system (RAAS), endothelium-dependent factors, their antagonists - natriuretic peptides (NUP) and the kallikreinin system. When one of the organs is damaged due to activation of the RAAS and the sympathetic nervous system, the development of endothelial dysfunction and chronic systemic inflammation, a vicious circle is formed - a pathophysiological condition in which the combination of cardiac and renal dysfunction leads to an accelerated decline in the functional capacity of each organ.

The term "cardiorenal syndrome" has long been used to denote dysfunction of the intact kidney in the setting of cardiac dysfunction.

The modern concept of CRS covers the entire spectrum of cardiorenal relationships and, based on the characteristics of the pathophysiological process, temporal factors and causes of kidney or heart dysfunction, describes 5 types of this syndrome. 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, CRS includes various acute and chronic disorders in which the primary organ affected may be either the heart or the kidney.

Acute cardiorenal syndrome (CRS type I). Characterized by a sudden deterioration in cardiac function leading to acute kidney injury (AKI); occurs in ACS in 9–19%, in cardiogenic shock in 70% of cases. Acute decompensation of CHF is complicated by AKI in 24–45% of patients. AKI usually develops in the first days of hospitalization: in 50% - in the first 4 days, 70–90% - in the first 7 days. These patients often have a history of chronic renal dysfunction that predisposes them to the development of AKI. The development of AKI is associated with an increased risk of cardiovascular and overall mortality, longer hospitalization and re-hospitalization rates, progression of CKD (if present) to stages IV–V in acute coronary syndrome (ACS), myocardial infarction (MI), percutaneous coronary interventions (PCI) and coronary artery bypass grafting (CABG). The risk of

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adverse outcomes increases regardless of the transient or persistent nature of AKI; more severe AKI is associated with a higher risk of death[1,3].

The development of AKI in acute cattle is caused by impaired renal perfusion due to a decrease in cardiac output (CO) and/or a significant increase in venous pressure. Resistance to diuretic therapy often develops. The presence of AKI with or without hyperkalemia limits the use of ACEIs, ARBs, and aldosterone antagonists in patients with heart failure (HF) and MI, which may negatively affect disease outcomes.

Chronic cardiorenal syndrome (CRS type II). Characterized by the presence of chronic heart pathology [for example, chronic heart failure (CHF)], leading to the progression of CKD. Impaired renal function is widespread among patients with CHF (45–63.6%) and is an independent negative prognostic factor for the development of systolic and diastolic dysfunction of the left ventricle (LV), cardiovascular death, while a biological gradient between the severity of renal dysfunction and worsening clinical outcomes. One of the main factors of damage in chronic cattle disease is long-term renal hypoperfusion, which is preceded by micro- and microangiopathies [8]. The functional state of the kidneys may also deteriorate due to treatment for CHF.

Acute cardiorenal syndrome (CRS type III). It is characterized by a primary sudden impairment of renal function (for example, in acute glomerulonephritis or pyelonephritis, acute tubular necrosis, acute urinary tract obstruction), which leads to acute impairment of cardiac function (HF, arrhythmias, ischemia). The prevalence of AKI during coronary angiography (CAG) and cardiac surgery is 0.3–29.7% and is associated with high mortality. AKI affects cardiac function through several mechanisms, the hierarchy of which has not been established. Fluid overload can lead to the development of pulmonary edema, hyperkalemia contributes to arrhythmias and cardiac arrest, and the accumulation of uremic toxins reduces myocardial contractility and leads to the development of pericarditis. In addition, renal ischemia itself can provoke inflammation and apoptosis of cardiomyocytes [9].

Chronic cardiorenal syndrome (CRS type IV). The main causes of kidney damage in recent years are type 2 diabetes mellitus and hypertension; Atherosclerosis, CHF and obesity play a significant role. There is growing interest in the pathogenetic role of relative or absolute deficiency of erythropoietin in CKD, which can cause activation of apoptosis, fibrosis and inflammation in the myocardium, as well as the development of anemia.

Secondary cardiorenal syndrome (CRS type V). It is characterized by the presence of combined renal and cardiac pathology due to acute or chronic systemic disorders, while dysfunction of one organ affects the functional state of the other, and vice versa. Examples of such diseases are sepsis, diabetes, amyloidosis, systemic lupus erythematosus, sarcoidosis, systemic vasculitis. Sepsis is the most common and severe condition affecting heart and kidney function.

DIAGNOSIS OF CARDIORENALS SYNDROMES: Laboratory diagnostics. Detection of acute myocardial injury in cattle types I and III usually does not cause difficulties. Currently, natriuretic peptides (BNP, NT-proBNP) are recognized markers of acute heart failure and acute decompensation of heart failure. Troponins are used as sensitive markers of myocardial necrosis. However, an increase in troponin levels is also observed in patients with CKD without a clinical picture of ACS, and also has prognostic significance in type IV cattle. Cystatin C is superior to creatinine in diagnosing renal dysfunction and correlates with the duration and severity of AKI. An increase in serum cystatin C levels is a marker of decreased glomerular filtration rate (GFR), and in urine it indicates tubular dysfunction.

The earliest markers detected in the blood and urine of patients with AKI are NGAL (neutrophil gelatinase-associated lipocalin), KIM-1 (renal messenger molecule 1), IL-18 (interleukin-18) and NAG (lysosomal enzyme N-acetyl). -β-d-glucosaminidase), the appearance of which precedes the increase in creatinine levels in such patients by 48–72 hours. Early markers of chronic CRS (type II) are the appearance and persistence of risk factors for the development and progression

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of chronic cardiac and renal dysfunction (albuminuria, decreased LVEF and GFR, LVH). CVS in patients with CKD (type IV cattle) are associated with increased plasma levels of specific biomarkers such as troponins, ADMA (asymmetric dimethylarginine), PAI 1 (plasminogen activator inhibitor type I), homocysteine, NUP, C-reactive protein, serum amyloid protein A, albumin modified by ischemia.

Imaging diagnostic methods Currently, the most promising non-invasive methods are multiphoton microscopy of the kidneys, as well as phase-contrast magnetic resonance imaging (MRI), which allows one to assess blood flow in the vessels of the kidneys. The use of the bioimpedance vector analysis method in combination with the determination of certain biomarkers seems promising.

Acute kidney injury. Until recently, AKI was referred to as acute renal failure (ARF). The term "acute kidney injury" includes the entire spectrum of acute renal failure syndrome - from minor changes in the functional state of the kidneys to conditions requiring renal replacement therapy[9]. The risk of developing AKI increases both in the presence of risk factors for AKI (critical condition of the patient, sepsis, shock, trauma and burns, cardiac and major surgical interventions, nephrotoxic and X-ray contrast drugs, poisoning), and in the presence of conditions that increase the patient's susceptibility to risk factors for AKI (dehydration , older age, female gender, Negroid race, acute and chronic diseases, diabetes, cancer, anemia, therapy).

Chronic kidney disease. In 2012, a modification of the classification of chronic kidney disease (CKD) was proposed. CKD is defined as a change in kidney structure or function that persists for more than 3 months and affects a person's health status. In the case of preserved or increased GFR, as well as in patients with its initial decrease, the diagnosis of CKD requires the presence of signs of kidney damage:

- albuminuria >30 mg/day or urine albumin/creatinine ratio >30 mg/g (>3 mg/mmol);
- change in urine sediment;
- electrolyte disturbances;
- structural and morphological changes;
- history of kidney transplantation.

New recommendations suggest classifying CKD based on the categories of GFR (Table 2) and albuminuria (Table 3), which allows CKD patients to be stratified by risk of complications. To assess the functional state of the kidneys, except in special situations, it is recommended to use the CKD-EPI formula (2009) to calculate GFR, and to assess albuminuria, it is preferable to calculate the albumin/creatinine or protein/creatinine ratio in the morning urine. In CKD, the risk of complications is inversely related to GFR, so patients need regular monitoring of GFR and albuminuria to assess progression.

Summary. Chronic kidney disease in modern recommendations - CKD is considered as an independent risk factor for the development of CVD and as equivalent to coronary heart disease (CHD) in terms of the risk of complications. In accordance with national recommendations for cardiovascular prevention (2011), as well as the diagnosis and correction of lipid metabolism disorders for the prevention and treatment of atherosclerosis (2012), patients with CKD (determined by GFR

The renal section of the European recommendations on hypertension (2013) has been significantly changed. Thus, when stratifying patients by risk of cardiovascular disease in the headings "Target organ damage" and "Associated clinical conditions", diagnostic criteria for serum creatinine were excluded, and criteria for estimated GFR were replaced by the category of CKD. At the same time, CKD is divided into categories depending on the level of GFR: patients with CKD and GFR 30–60 ml/min/1.73 m2 (CKD stage III) are classified as having target organ damage, and patients with CKD and GFR <30 ml /min/1.73 m2 (CKD stages IV–V) – those with associated clinical conditions.

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In International recommendations 2012–2013. new target blood pressure levels in patients with CKD have been formulated. From the standpoint of evidence-based medicine, in patients with diabetic and nondiabetic nephropathy, the target systolic blood pressure level is <130 mmHg. subject to monitoring the dynamics of GFR. Target diastolic blood pressure is <90 mmHg.

Reducing proteinuria is recognized as a therapeutic goal. It has been convincingly shown that RAAS blockers are more effective in reducing albuminuria in patients with diabetic and non-diabetic nephropathy, CVD, and are also effective in preventing microalbuminuria.

Dosing of drugs is carried out taking into account GFR. At the same time, it is recommended to temporarily discontinue potentially nephrotoxic drugs in patients with GFR <60 ml/min/1.73 m2 (stages IIIa–V) with severe intercurrent diseases, primarily this applies to RAAS blockers, diuretics, NSAIDs, metformin, lithium preparations and digoxin.

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