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Development of Nephropathy in Chronic Heart Failure

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Introduction

Chronic heart failure (CHF) is a clinical syndrome characterised by the inability of the heart to provide adequate blood flow to meet the metabolic needs of the body. One of the most significant and frequent complications of CHF is the development of chronic kidney disease (CKD), which together form the so-called cardiorenal syndrome. The relationship between cardiac and renal dysfunction is bidirectional and reflects the complex pathophysiological interdependence of these organs.

Nephropathy in CHF develops due to haemodynamic disturbances as well as neurohormonal and inflammatory activation. Deterioration of renal function in patients with CHF is directly associated with increased risk of hospitalisation, worse prognosis and increased mortality. Therefore, understanding of pathogenesis, timely diagnosis and optimal therapy of renal dysfunction in patients with CHF is an important clinical task.

The aim of this paper is to provide an overview of current data on the mechanisms of nephropathy development in CHF, diagnostic approaches and therapeutic options, as well as to outline the key directions in the research and clinical management of this pathology.

Chronic heart failure is the end stage of various diseases of the cardiovascular system, including ischaemic heart disease, arterial hypertension, heart defects and cardiomyopathies. According to the European Society of Cardiology (ESC), the prevalence of CHF in developed countries is about 1-2% in the adult population and increases to 10% in people over 70 years of age.

Clinical manifestations of CHF include dyspnoea, rapid fatigue, lower limb oedema and decreased exercise tolerance. The pathogenesis of CHF includes decreased left ventricular ejection fraction or impaired myocardial relaxation. Compensatory mechanisms, such as activation of the sympathoadrenal system and the renin-angiotensin-aldosterone system (RAAS), initially maintain cardiac output but subsequently lead to deterioration of cardiac function.

Chronic kidney disease (CKD) is characterised by a progressive decrease in glomerular filtration rate and structural and functional changes in the kidneys. According to KDIGO, the diagnosis of CKD is made when the glomerular filtration rate is <60 ml/min/1.73 m² for more than 3 months or when there are structural signs of kidney damage. The main pathogenesis links include inflammation, fibrosis, activation of apoptosis, and hyperfiltration in the remaining nephrons leading to their exhaustion and death.

Cardiorenal syndrome is a pathological condition in which acute or chronic dysfunction of the heart or kidneys causes dysfunction of the second organ. There are 5 types of the syndrome depending on the primary lesion. Type II, chronic CHF causing progressive deterioration of renal function, is most common in CHF. Pathogenesis includes renal hypoperfusion, venous stasis, activation of RAAS, sympathetic nervous system and inflammation.

Main pathogenetic mechanisms: - Decreased renal perfusion and tissue hypoxia; - Increase in central venous pressure; - Activation of RAAS and sympathetic nervous system; - Oxidative stress, cytokine cascade;

- ✓ Involvement of FGF-23, endothelin-1, inflammatory markers

Diagnosis is based on the determination of CKD-EPI, creatinine, cystatin C, albuminuria. Central venous pressure, echocardiography, urinalysis are taken into account. Markers: NGAL, KIM-1, β2-microglobulin.

Main lines of therapy:

- ✓ Fluid volume control (diuretics)
- ✓ RAAS blockers (IAPPs, BRAs)
- ✓ Sodium-glucose cotransporter type 2 inhibitors (SGLT2 inhibitors)
- ✓ BP and hyperglycaemia control
- ✓ Maintenance of adequate renal perfusion

Special attention is paid to drugs that improve prognosis and protect the kidneys at the same time: dapagliflozin, empagliflozin.

The combination of CHF and CKD significantly worsens the prognosis. There is an increased risk of mortality, repeated hospitalisations, progression of CHF to the terminal stage with the need for replacement therapy. Combination therapy with SGLT2 and RAAS-blockers improves survival and organ function.

Conclusion

The development of nephropathy in CHF is a frequent and prognostically unfavourable complication that requires timely detection and a comprehensive approach. Modern methods of diagnosis, pharmacotherapy and multidisciplinary patient management can slow the progression of renal dysfunction and improve the quality of life of patients with CHF.

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