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Coexistence of Chronic Heart Failure and Chronic Kidney Disease

Bobojonov Izzat Shonazar ugli
Tashkent Medical Academy Faculty №.-2 and Department of Hospital Therapy, Nephrology and Hemodialysis Department "Internal Diseases" second year clinical coordinator

Summary. Chronic heart failure (CHF) and chronic kidney disease (CKD) often coexist, presenting a significant challenge in healthcare. This article aims to explore the relationship between CHF and CKD in more detail, including their shared risk factors, pathophysiological mechanisms, impact on each other, and management strategies. By understanding these aspects, healthcare professionals can better manage individuals with both conditions and improve their outcomes.

Keywords: heart failure (HF); chronic kidney disease (CKD); GFR; ejection fraction; renin–angiotensin–aldosterone system inhibitors (RAAS-I); mineralocorticoid receptor antagonists (MRAs)

Chronic heart failure is a progressive condition characterized by the heart's inability to pump blood efficiently, leading to symptoms such as fatigue, shortness of breath, and fluid retention. Chronic kidney disease, on the other hand, is a progressive loss of kidney function, resulting in the accumulation of waste products and fluid imbalances in the body. Both conditions are prevalent worldwide and are associated with significant morbidity and mortality.

Several risk factors contribute to the development of both CHF and CKD. Hypertension, diabetes mellitus, obesity, and advanced age are common risk factors shared by these conditions. These risk factors contribute to the development of atherosclerosis, inflammation, and oxidative stress, which can damage both the heart and the kidneys. Additionally, medications used to treat one condition may have adverse effects on the other, further complicating the management of individuals with both CHF and CKD.

Heart failure (HF) is the leading CV complication in CKD patients and its prevalence increases with declining kidney function [4]. In the Atherosclerosis Risk in Communities (ARIC) study [5], a large, population-based study of US adults, the incidence of HF was 3-fold higher in individuals with an estimated glomerular filtration rate (GFR) <60 mL/min/1.73 m2, compared with the reference group with an estimated GFR ≥90 mL/min/1.73 m2. In dialysis patients, the presence of HF at the start of dialysis is a strong and independent predictor of short-term [6] and long-term mortality, in both hemodialysis (HD) [7] and peritoneal dialysis (PD) patients [8]. The median survival of dialysis patients with baseline HF has been estimated to be 36 months, in contrast with 62 months for those without baseline HF [7]. Over 80% of ESRD patients who are recently

diagnosed with HF are expected to die within only three years from the time of this diagnosis [9].

Myocardial hypertrophy is associated with a reduction in the capillary density [2], which creates an imbalance between oxygen demands and supplies, thus causing ischemia [3]. Ischemia promotes myocardial cell apoptosis, as well as extracellular matrix and collagen accumulation, leading to interstitial fibrosis, which, in turn, induces LV stiffness, increased LV filling pressure, impaired diastolic filling, and diastolic dysfunction [4, 5]. Moreover, myocardial fibrosis aggravates ischemia, by reduction of capillary density and coronary reserve [6], and considerably increases the risk of ventricular arrhythmias and sudden cardiac death [7–9]. Associated coronary artery disease—also, very common in patients with CKD and ESRD—further contributes to ischemia, myocardial cell damage, and fibrosis [1].

From a hemodynamic view, LVH is an adaptive remodeling process of the LV, which compensates the increase in cardiac work induced by an increased afterload (pressure overload), an increased preload (volume overload), or both. Increased afterload may result from arterial hypertension, arterial stiffness, or valvular aortic stenosis and typically leads to a concentric thickening of the LV wall (concentric hypertrophy), which is meant to boost the intraventricular systolic pressure. Increased preload may be due to hypervolemia, anemia, and (in HD patients) high blood flow arteriovenous fistula; volume overload leads to the development of LV dilatation (eccentric LVH), by accumulation of new myocardial sarcomeres in series [2]. Afterload and preload factors often coexist in various degrees and combinations, with an additive or synergistic effect, which explains why both patterns, as well as a mixed pattern of LVH, are commonly seen in CKD patients [1].

A large number of nonhemodynamic factors also contribute to the development of LVH and cardiomyopathy in CKD patients [2]. For example, hyperphosphatemia has been associated with high blood pressure (BP) [2], increased LV mass [3, 4], and diastolic dysfunction [5]. Excess angiotensin II can accumulate in the heart and promote myocyte hypertrophy, interstitial fibrosis, and microvascular disease, as well as cardiac conduction disturbances, QT prolongation, and arrhythmias [26]. High serum aldosterone, resulting from activation of renin-angiotensin system or other pathways, can induce myocardial fibrosis, possibly by release of transforming growth factor β [1, 7]. Sympathetic overactivity, which has been demonstrated in CKD, is also deleterious to the heart and may induce LV concentric remodeling [8].

The heart and kidneys play a major role in maintaining fluid homeostasis and normal blood pressure in the body. In physiological conditions, the cooperation between the heart and the kidneys enables a response to changes in renal perfusion, such as volume reduction or overload, which may cause ischemia or hyperperfusion injury [1]. On the other hand, sudden deterioration or chronic failure of one organ may be associated with decreased function of another. Renal salt and water excretion inability, improper renin secretion, and cardiomyopathic factors, such as myocardial infarction, left ventricular hypertrophy, and fibrosis, increase cardiac preload and afterload providing progressive volume and pressure overload [2,3]. Chronic existence of these abnormalities may lead to heart failure or aggravate an already existing HF. The common connection through the vascular bed, regulation by the sympathetic nervous system (SNS), and renin–angiotensin–aldosterone system (RAAS) cause stress on the renal nephrons. Further, insufficient kidney perfusion, due to low cardiac output, and renal venous congestion, caused by right heart failure, lead to kidney failure [2]. Furthermore, shared additional risk factors including diabetes mellitus, obesity, anemia, and iron deficiency or mineral disorder have an impact on the occurrence of HF in CKD and vice versa [3]. Common pathological mechanisms, shared risk factors, or systemic disorders may affect the heart and the kidneys, causing their simultaneous dysfunction (Figure 1). Moreover,

studies imply elevated intra-abdominal pressure as a risk factor for kidney failure [4,5].

In addition, the interaction between cardiac disease and renal dysfunction leads to diuretic resistance [6]. As a result of considerable disorder overlap, the differentiation of which disease is primary and which is secondary may be complicated and challenging. This distinction may be needed to develop new treatments to improve renal and cardiac function.

The presence of CKD in individuals with CHF is associated with worse outcomes. CKD is an independent risk factor for mortality in individuals with CHF, and the severity of kidney dysfunction is directly correlated with the severity of heart failure. The presence of CKD also affects the response to treatment, with individuals with both CHF and CKD having a lower response rate to medications such as angiotensin-converting enzyme inhibitors and beta-blockers.

Managing individuals with both CHF and CKD requires a multidisciplinary approach. Optimization of fluid balance, blood pressure control, and management of comorbidities such as diabetes and anemia are essential. Medications should be carefully selected, considering their effects on both the heart and the kidneys. In some cases, renal replacement therapy, such as dialysis or kidney transplantation, may be necessary.

Furthermore, lifestyle modifications, including a heart-healthy diet, regular exercise, smoking cessation, and weight management, play a crucial role in managing both CHF and CKD. These lifestyle changes can help control risk factors such as hypertension and diabetes, which are common to both conditions.

In conclusion, the coexistence of CHF and CKD presents a significant challenge in healthcare. Understanding the shared risk factors and pathophysiological mechanisms is crucial in managing individuals with both conditions. A multidisciplinary approach, focusing on optimizing fluid balance, blood pressure control, and managing comorbidities, is essential for improving outcomes in these individuals. Lifestyle modifications, including a heart-healthy diet and regular exercise, are also important. Further research is needed to explore novel treatment strategies and interventions to improve the prognosis of individuals with both CHF and CKD.

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