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CLINICAL AND PROGNOSTIC MARKERS OF CARDIORENAL SYNDROME PROGRESSION AND THEIR ROLE IN PERSONALIZED TREATMENT

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Abstract
Cardiorenal syndrome is a relevant problem in modern cardiology and nephrology, associated with the mutual deterioration of cardiac and renal function. This article discusses clinical and prognostic markers of cardiorenal syndrome progression, including reduced estimated glomerular filtration rate, elevated NT-proBNP levels, albuminuria, type 2 diabetes mellitus, and signs of venous congestion. It is shown that a comprehensive assessment of these parameters makes it possible to identify high-risk patients and justify a personalized approach to treatment. Individualized therapy contributes to reducing the frequency of heart failure decompensation and slowing the progression of renal dysfunction.

Keywords: Cardiorenal syndrome, chronic heart failure, chronic kidney disease, albuminuria, NT-proBNP, eGFR, personalized treatment.

Introduction

Cardiorenal syndrome is one of the most important interdisciplinary problems in modern cardiology and nephrology, as it is characterized by interrelated impairment of cardiac and renal function. The progression of this condition leads to worsening chronic heart failure, declining renal function, increased hospitalization rates, and a higher risk of adverse cardiovascular outcomes. Early identification of clinical and prognostic markers of cardiorenal syndrome progression is of particular importance. These markers include a reduced estimated glomerular filtration rate, elevated NT-proBNP levels, the presence of albuminuria, type 2 diabetes mellitus, and signs of venous congestion. A comprehensive assessment of these indicators makes it possible to identify high-risk patients in a timely manner and select an individualized treatment strategy. Due to the heterogeneity of the clinical manifestations of cardiorenal syndrome, standard treatment approaches do not always provide sufficient effectiveness. Therefore, the development of personalized therapy based on the clinical, laboratory, and pathogenetic characteristics of the patient is an important direction for optimizing the management of patients with cardiorenal syndrome.

Objective of the Study

To evaluate clinical and prognostic markers of cardiorenal syndrome progression and determine their role in selecting a personalized treatment strategy.

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Materials and Methods

The study included 108 patients with clinical signs of cardiorenal syndrome. The age of the examined patients ranged from 45 to 78 years, with a mean age of 64.2 ± 8.7 years. There were 66 men, accounting for 61.1%, and 42 women, accounting for 38.9%. Chronic heart failure of functional class II–III was diagnosed in 72.2% of patients, arterial hypertension in 67.6%, type 2 diabetes mellitus in 48.1%, and stage II–III chronic kidney disease in 70.4% of the examined patients. All patients underwent comprehensive clinical, laboratory, and instrumental examination. The assessed parameters included serum creatinine level, estimated glomerular filtration rate, albuminuria, NT-proBNP level, blood pressure, left ventricular ejection fraction, the presence of edematous syndrome, dyspnea, hepatomegaly, peripheral edema, and other signs of venous congestion.Patients were divided into two groups depending on the clinical course of the disease. The first group included patients with signs of cardiorenal syndrome progression, including recurrent decompensation of heart failure, deterioration of renal function, and increasing albuminuria. The second group included patients with a stable course of the disease. Differences were considered statistically significant at $p < 0.05$.

Results

The study found that progression of cardiorenal syndrome was more frequently observed in patients with reduced estimated glomerular filtration rate, elevated NT-proBNP levels, albuminuria, type 2 diabetes mellitus, and pronounced signs of venous congestion.

In patients with an eGFR below 60 mL/min/1.73 m², the frequency of recurrent heart failure decompensation was 56.4%, whereas among patients with an eGFR ≥ 60 mL/min/1.73 m² this rate was 29.7%. The differences were statistically significant ($p = 0.004$). This indicates the important prognostic role of reduced renal function in the development of an unfavorable course of cardiorenal syndrome.

The NT-proBNP level in patients with disease progression was significantly higher and amounted to 1760 ± 490 pg/mL, compared with 980 ± 360 pg/mL in patients with a stable course of the disease ($p < 0.001$). Elevated NT-proBNP reflected the severity of myocardial stress, volume overload, and heart failure severity.

Albuminuria greater than 30 mg/g was detected in 62.9% of patients with progression of cardiorenal syndrome and only in 34.2% of patients with a stable course of the disease ($p = 0.006$). This parameter may be considered a marker of renal injury, endothelial dysfunction, and increased cardiovascular risk.

Correlation analysis revealed a negative relationship between estimated glomerular filtration rate and NT-proBNP level: $r = –0.44$; $p < 0.001$ (Table 1). This indicates that deterioration of renal function is associated with greater severity of heart failure. A positive correlation was also found between the level of albuminuria and the severity of clinical signs of congestion: $r = 0.38$; $p = 0.002$. Type 2 diabetes mellitus was also associated with a higher risk of cardiorenal syndrome progression. Patients with diabetes more frequently had albuminuria, reduced eGFR, and recurrent episodes of heart failure decompensation. This confirms the role of metabolic disorders in the development and progression of cardiorenal impairment. Signs of venous congestion, including peripheral edema, dyspnea, liver enlargement, and congestive manifestations, were more common in patients with an unfavorable

course of the disease. The presence of clinically significant congestion was associated with increased NT-proBNP levels, reduced eGFR, and a higher frequency of hospitalizations.

Based on the obtained data, the main clinical and prognostic markers of cardiorenal syndrome progression were identified: eGFR below 60 mL/min/1.73 m², NT-proBNP level above 1000 pg/mL, albuminuria greater than 30 mg/g, presence of type 2 diabetes mellitus, and signs of venous congestion.

The use of a personalized approach, including optimization of diuretic therapy, blood pressure control, glycemic correction, nephroprotective therapy, and dynamic monitoring of renal function, reduced the frequency of recurrent heart failure decompensation from 33.3% to 17.6% during 12 weeks of follow-up ($p = 0.039$).

Table 1 Statistical indicators characterizing the progression of cardiorenal syndrome

Indicator	Patients with CRS progression	Patients with stable disease course	p-value
Frequency of recurrent decompensation in patients with eGFR < 60 mL/min/1.73 m²	56.4%	29.7% in patients with eGFR ≥ 60 mL/min/1.73 m²	0.004
NT-proBNP level, pg/mL	1760 ± 490	980 ± 360	<0.001
Albuminuria > 30 mg/g	62.9%	34.2%	0.006
Correlation between eGFR and NT-proBNP	r = –0.44	—	<0.001
Correlation between albuminuria and signs of congestion	r = 0.38	—	0.002
Frequency of recurrent decompensation after a personalized treatment approach	17.6%	33.3% before treatment	0.039

Table 1 presents statistical indicators characterizing the progression of cardiorenal syndrome.

Conclusions:

1. The progression of cardiorenal syndrome is associated with the combined influence of heart failure, renal dysfunction, venous congestion, neurohumoral activation, and metabolic disorders.
2. The main clinical and prognostic markers of an unfavorable course are a decrease in eGFR below 60 mL/min/1.73 m², elevated NT-proBNP levels, albuminuria greater than 30 mg/g, type 2 diabetes mellitus, and signs of venous congestion.
3. Reduced eGFR and elevated NT-proBNP levels are significantly associated with a higher frequency of recurrent heart failure decompensation and progression of cardiorenal syndrome.
4. A comprehensive assessment of clinical and laboratory markers makes it possible to identify high-risk patients and justify a personalized treatment approach aimed at slowing the progression of cardiac and renal dysfunction.

Discussion

The obtained results confirm that the progression of cardiorenal syndrome is determined by the combined influence of cardiac, renal, vascular, and metabolic factors. Cardiorenal syndrome is a complex clinical and pathogenetic condition in which impaired cardiac function contributes to the

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deterioration of renal function, while renal dysfunction, in turn, aggravates the course of cardiovascular pathology [1, 2]. In this regard, isolated assessment of only cardiac or only renal function does not allow a full evaluation of the patient's prognosis. The most informative approach is a comprehensive assessment that includes simultaneous evaluation of clinical signs of heart failure, renal function, biomarkers of myocardial stress, and markers of renal injury.

Current guidelines on chronic kidney disease emphasize the importance of combined assessment of estimated glomerular filtration rate and albuminuria, since these indicators have important diagnostic and prognostic value [3, 13]. In turn, heart failure guidelines highlight the need for comprehensive evaluation of clinical status, natriuretic peptides, renal function, and comorbid conditions [11, 12].

A decrease in estimated glomerular filtration rate is one of the most significant indicators of an unfavorable course of cardiorenal syndrome. Impaired renal filtration contributes to sodium and fluid retention, increased circulating blood volume, aggravated venous congestion, and progression of chronic heart failure. At the same time, cardiac decompensation worsens renal perfusion and activates the renin-angiotensin-aldosterone system and the sympathetic nervous system, leading to further renal tissue damage [1, 2, 9].

In the present study, a decrease in eGFR below 60 mL/min/1.73 m² was associated with a higher frequency of recurrent heart failure decompensation. This confirms that renal dysfunction in patients with cardiorenal syndrome is not only a laboratory finding but also an important clinical and prognostic marker. Similar data are consistent with studies in which impaired renal function is considered one of the key factors of poor prognosis in patients with cardiovascular pathology [4, 5, 8].

Elevated NT-proBNP levels reflect the degree of myocardial overload, the severity of heart failure, and the risk of recurrent decompensation. In combination with reduced eGFR, this indicator acquires particular prognostic significance, as it indicates simultaneous involvement of two target organs — the heart and kidneys. In our study, patients with an unfavorable course of cardiorenal syndrome had significantly higher NT-proBNP levels, confirming its role as a marker of myocardial stress and volume overload [11, 12].

Albuminuria is an important marker not only of renal damage but also of systemic endothelial dysfunction. Its presence in patients with cardiorenal syndrome indicates an increased risk of chronic kidney disease progression and cardiovascular complications. Therefore, assessment of albuminuria should be considered an essential component of the examination of patients with suspected cardiorenal syndrome [3, 13]. In the present study, albuminuria greater than 30 mg/g was more frequently detected in patients with disease progression, confirming its high prognostic significance.

The presence of type 2 diabetes mellitus is of particular importance. In such patients, cardiorenal syndrome develops against the background of chronic hyperglycemia, insulin resistance, microangiopathy, inflammation, endothelial dysfunction, and progressive damage to the glomerular apparatus of the kidneys. Data reported by Daminov B.T. et al. also indicate the important role of diabetic nephropathy in the formation of cardiorenal interactions and the progression of cardiac and renal dysfunction [5]. This substantiates the need for more careful monitoring and early initiation of cardio- and nephroprotective therapy in this category of patients.

Venous congestion is also an important pathogenetic mechanism of worsening renal function. Increased venous pressure leads to a decrease in the renal perfusion gradient, impaired glomerular

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filtration, and sodium and fluid retention. In this regard, patients with pronounced signs of congestion require individualized correction of diuretic therapy with mandatory monitoring of renal function and electrolyte balance [2, 11, 14].

The study results also show that personalized treatment of patients with cardiorenal syndrome should include not only standard therapy for heart failure and chronic kidney disease but also consideration of the leading clinical phenotype. In patients with predominant venous congestion, the main emphasis should be placed on effective decongestion. In the presence of significant albuminuria and diabetes mellitus, the nephroprotective strategy should be intensified. In patients with high NT-proBNP levels and frequent episodes of decompensation, optimization of heart failure therapy and more frequent dynamic monitoring are required.

Special attention should be paid to patients with ischemic heart disease undergoing invasive procedures with the use of contrast agents. According to Daminova L.T., Muminov Sh.K., and Nigmonov B.B., percutaneous coronary interventions may be accompanied by a risk of worsening renal function, especially in patients with baseline renal dysfunction, diabetes mellitus, and older age [7]. This further confirms the need to assess eGFR, creatinine, albuminuria, and the comorbid background when choosing management tactics for patients with cardiorenal syndrome.

Current data also confirm the importance of cardio- and nephroprotective drugs, including SGLT2 inhibitors, in patients with chronic kidney disease, type 2 diabetes mellitus, and heart failure [13, 15]. Therefore, a personalized approach should include not only assessment of prognostic markers but also selection of therapy based on the functional status of the heart and kidneys, the level of albuminuria, the risk of hyperkalemia, blood pressure, and the patient's comorbid profile.

Thus, clinical and prognostic markers make it possible not only to assess the risk of cardiorenal syndrome progression but also to determine the most rational treatment strategy for each individual patient. Comprehensive assessment of eGFR, NT-proBNP, albuminuria, type 2 diabetes mellitus, and signs of venous congestion allows identification of high-risk patients and substantiates a personalized treatment approach aimed at slowing the progression of cardiac and renal dysfunction.

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