ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2024-6.875 ResearchBib IF: 8.848 / 2024

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-4, ISSUE-5

EFFECTS OF KIDNEY DYSFUNCTION ON DISEASE COURSE IN PATIENTS WITH CHRONIC HEART FAILURE.

Norboyev A.A.
Republican Specialized Scientific and Practical Medical Center of Cardiology, Tashkent, Uzbekistan.
Boqiyeva D. R.
Tashkent medical academy, Tashkent, Uzbekistan.
Xodjanova Sh. I.
Tashkent medical academy, Tashkent, Uzbekistan.

The functions of the heart and kidneys are closely interrelated. Their interaction can be defined as a complex biological relationships between distant organs, which are mediated through cellular, canlecular, nervous, endocrine and paracrineed factors. Under physiological conditions this connection.Helps maintain homeostasis and optimal functioning of the human body. Deterioration the function of one of these organs is caused by a vicious range of events leading to multi-organ failure

accuracy. Although it is well known about the dysfunction renal function in patients with heart disease, it remains unclear whether renal dysfunction is passive response to cardiac failure activity. In clinical practice throughout the for more than 13 years, the term “cardiorenal syndrome" (CDS), i.e. coexistence cardiac and renal nature in the same patient.

Key words: Chronic heart failure, cardiorenal syndrome, chronic kidney disease (CKD)

ВЛИЯНИЕ ДИСФУНКЦИИ ПОЧЕК НА ТЕЧЕНИЕ ЗАБОЛЕВАНИЯ У ПАЦИЕНТОВ С ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТЬЮ.

Норбоев А.А. - Республиканский специализированный научно-практический центр кардиологии, Ташкент, Узбекистан.
Бокиева Д.Р.- Ташкентская медицинская академия, Ташкент, Узбекистан.
Ходжанова Ш.И.-Ташкентская медицинская академия, Ташкент, Узбекистан.

Функции сердца и почек тесно взаимосвязаны. Их взаимодействие можно определить как сложные биологические взаимоотношения между отдаленными органами, которые опосредуются клеточными, канлекулярными, нервными, эндокринными и паракринными факторами. В физиологических условиях эта связь способствует поддержанию гомеостаза и оптимального функционирования организма человека. Ухудшение функции одного из этих органов вызвано порочным кругом событий, приводящих к полиорганной недостаточности. Хотя хорошо известно о нарушении функции почек у больных с сердечно-сосудистыми заболеваниями, остается неясным, является ли почечная дисфункция пассивным ответом на несостоятельность сердечной деятельность. В клинической практике на протяжении более 13 лет широко используется термин «кардиоренальный синдром» (КРС), т.е. сосуществование сердечной и почечной природы у одного и того же пациент.

Ключевые слова: Хроническая сердечная недостаточность, кардиоренальный синдром ,Хроническая болезнь почек (ХБП)

ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2024-6.875 ResearchBib IF: 8.848 / 2024

The problem of chronic heart failure (CHF), and especially CHF with reduced ejection fraction, is one of the most significant for modern healthcare systems. This is due to high mortality, reduced quality of life, frequent episodes of hospitalization and severe comorbidity of patients with this pathology. Involvement of the kidneys in the pathological process is one of the most common comorbid conditions in cardiovascular diseases. There are a large number of pathogenetic mechanisms of the mutually negative influence of heart failure and renal dysfunction, which are reflected in the concept of "cardiorenal syndrome".

There are five subtypes of cardiorenal syndrome:

Cardiorenal syndrome type 1 (acute RRS) is characterized by rapid deterioration of cardiac function, which leads to the development of acute kidney injury (AKI). It is observed in acute heart failure due to myocardial infarction, high blood pressure (BP), with rapid progression of chronic heart failure (CHF), cardiogenic shock. The cause of acute kidney injury in type 1 cattle is inadequate blood supply to the kidneys due to low cardiac output and/or increased venous pressure, which leads to congestion in the renal vasculature.

Cardio-renal syndrome type 2 (chronic RRS) is characterized by chronic dysfunction of the heart (for example, coronary heart disease, valvular disease), which leads to the progression of chronic renal failure. The incidence of kidney dysfunction in CHF is about 25%. The reason for the development of type 2 KRS is a long-term decrease in blood supply to the kidneys. Independent predictors of worsening renal function are older age, hypertension, diabetes mellitus, and acute myocardial infarction.

Cardiorenal syndrome type 3 (acute renocardial syndrome) is characterized by a sudden and primary deterioration of kidney function (eg, acute kidney injury or glomerulonephritis), leading to acute cardiac dysfunction (eg, arrhythmia, angina). AKI can affect cardiac function in several ways. Fluid retention in the body can cause acute decompensation of chronic heart failure and pulmonary edema, and hyperkalemia can cause arrhythmias. Untreated uremia reduces myocardial contractility, acidosis leads to narrowing of the pulmonary vessels, which worsens the course of right ventricular heart failure.

Cardio-renal syndrome type 4 (chronic reno-cardiac syndrome) is characterized by primary chronic kidney disease (for example, chronic glomerulonephritis), which leads to decreased heart function, hypertrophy of the heart chambers, and an increased risk of developing cardiovascular diseases.

Cardiorenal syndrome type 5 (secondary RRS) is characterized by the presence of combined dysfunction of the heart and kidneys as a result of acute or chronic systemic disease. Type 5 KRS is observed in sepsis, diabetes mellitus, systemic lupus erythematosus, amyloidosis and sarcoidosis.

Cardiorenal syndrome type 1 occurs with acute decompensation of cardiac function, leading to a decrease in glomerular filtration. Previously, researchers suggested that decreased cardiac output with decreased renal perfusion is the main cause of deterioration of renal function in cardiorenal syndrome types 1 and 2. However, recent studies have shown that increased central venous pressure is a more important factor. When patients develop fluid overload due to deteriorating cardiac function, venous pressure increases and is transmitted back to the efferent arterioles; this results in a net decrease in glomerular filtration pressure and kidney damage. Other factors involved in the pathogenesis of cardiorenal syndromes types 1 and 2 include increased intra-abdominal pressure, activation of the renin-angiotensin-aldosterone system (RAAS),

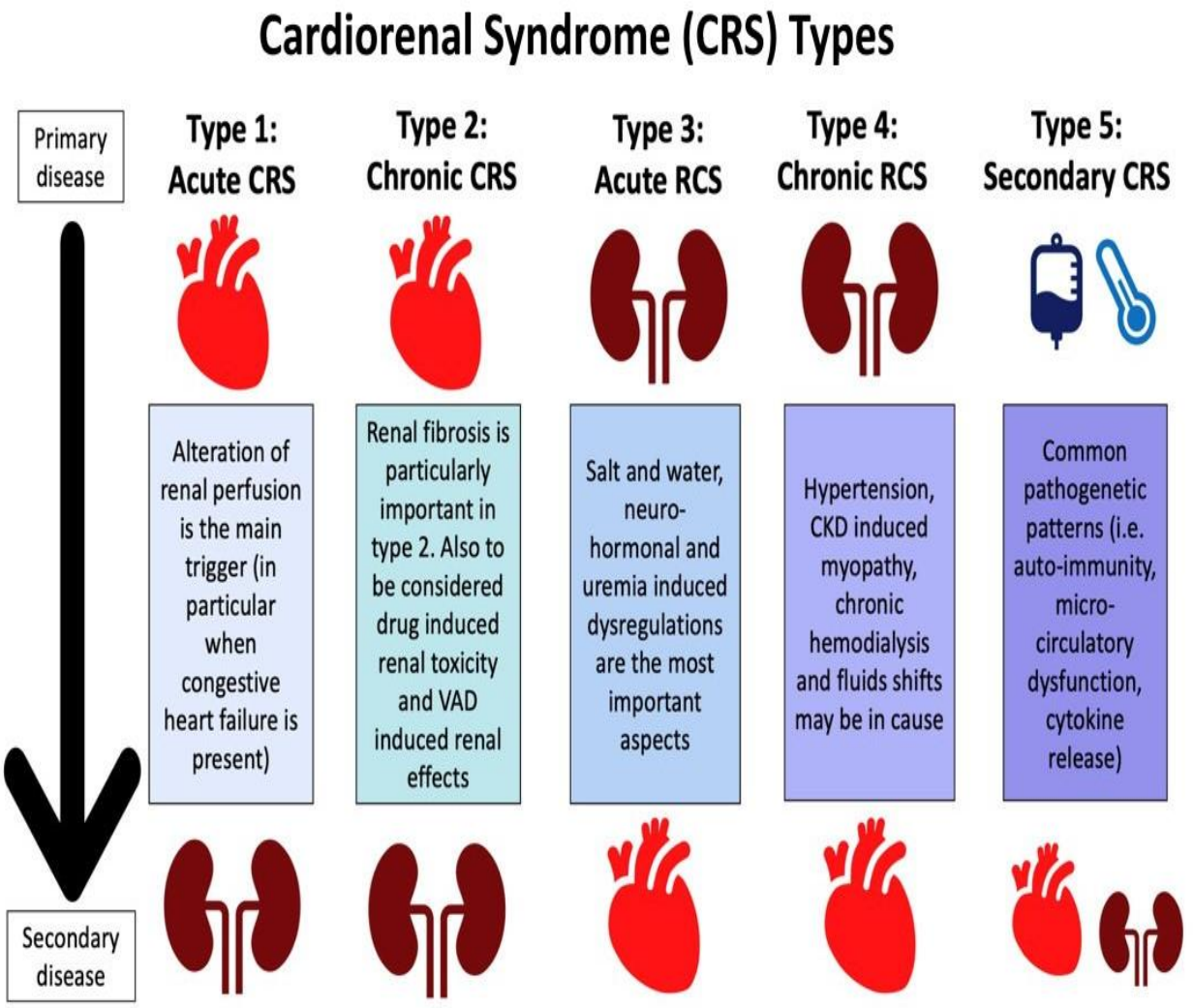
ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2024-6.875 ResearchBib IF: 8.848 / 2024

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-4, ISSUE-5

activation of the sympathetic nervous syndrome, and increased inflammatory kidney damage associated with heart failure.

Targeting this cycle is the basis of therapy for type 1 cardiorenal syndrome. Cardiorenal syndromes types 3 and 4 most often result from volume overload due to renal dysfunction, cardiac dysfunction secondary to metabolic disorders (such as acidemia), and neurohormonal changes accompanying kidney disease. Patients may develop cardiorenal syndrome type 5 due to sepsis, systemic lupus erythematosus (SLE), diabetes mellitus, decompensated cirrhosis, or amyloidosis; all these disorders can lead to diseases of both the heart and kidneys.



Ricci Z, Romagnoli S, Ronco C. Cardiorenal Syndrome. Crit Care Clin. 2021;37(2):335-347.

Schematic representation of the five Cardiorenal syndrome (CRS) types according to the organ direction (primary > secondary disease) and the time window (acute or chronic). According to this classification, two CRS (acute and chronic), two renocardiac (acute and chronic) syndromes, and one secondary CRS are depicted.
CKD, chronic kidney disease; RCS, renocardiac syndrome; VAD, ventricular assist device.

When discussing renal risk factors for cardiovascular disorders in CKD, it should be noted that what dyslipidemia and chronic inflammation contribute additional load on the myocardium and endothelium vessels [2]. In patients with impaired function

kidney and significant proteinuria lipid profile becomes atherogenic, in part due to dysfunction of high-density lipoprotein cholesterol (HDL-C) and excessive oxidation

ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2024-6.875 ResearchBib IF: 8.848 / 2024

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-4, ISSUE-5

low-density lipoprotein cholesterol (LC LDL). In addition, chronic inflammation

is one of the pathogenetic factors that can able to contribute to the development and progress the study of cardiovascular diseases, how is it has been confirmed in studies showing significant increase in C-reactive protein in patients with CKD with a significant positive correlation with the resistive index of the renal arteries and feedback with the glomerular filtration (GFR).

In recent years, special attention has been given to the role phosphate retention and related disorders, falling under the CKD-MCD section. In patients with renal dysfunction, deficiency often develops activity of vitamin D due to the lack of its pre-cause, disturbances in the activity of the renal enzyme 1α-hydroxylase, which converts this a precursor to the active hormone, or both. As a result, phosphorus-calcium is disrupted

metabolism in tissues and hyperphosphatemia occurs [12] .

Pulmonary hypertension in CKD may be associated with several risk factors such as

anemia, apnea, increased sympathetic activity pain, inflammation, vascular calcification and endothelial dysfunction, but pathogenesis remains unclear pulmonary arterial hypertension (PAH) in early these stages in patients with CKD[3].

In one study, TAPSE and ePASP scores were significantly different in patients with CKD from control group of healthy individuals. In addition, ePASP negatively correlated with GFR, showing it progressive increase with deterioration of function kidneys, at the same time there were no statistical significant differences between the two groups in terms of pulmonary artery wedge pressure and final but-diastolic volume of the RV. In fact, on experimental models (on dogs) was there is a connection between hyperparathyroidism and calcification pulmonary vessels and PAH, increased incidence of PAH, a relationship between PAH and hyperparathyroidism has been identified in predialysis and dialysis patients[5].

Thus, timely assessment of the bilateral influence of the heart and kidneys is a key point in understanding the severity of such pathology. The mechanisms leading to multiorgan changes during the development of renal dysfunction require further study, and the implementation of treatment and preventive measures should be carried out taking into account the multidisciplinary nature of the problems.

REFERENCES

1. ВЛИЯНИЕ ПОЧЕЧНОЙ ДИСФУНКЦИИ НА СЕРДЕЧНО-СОСУДИСТУЮ СИСТЕМУ. ВОЗМОЖНОСТИ РАННЕЙ ДИАГНОСТИКИ ПОЧЕЧНОЙ ДИСФУНКЦИИ . Б.Ж. Иманов1 , И.Т. Муркамилов*2,3 , И.С. Сабиров 3 , А.Ш. Сарыбаев

2. Krane V., Wanner C. Statins, inflammation and kidney disease. Nat Rev Nephrol.2011;7(7):385397.doi:10.1038/nrneph.2011.62https://dio:10.3389/fphys.2019.01108

3. Lai S., Ciccariello M., Dimko M.et al. Cardio-Renal Syndrome Type 4: The Correlation Between Cardiorenal Ultrasound Parameters. Kidney Blood Press Res. 2016;41(5):654-662. DOI:https://doi. org/10.1159/000447934.https://dio:10.1016/j.ijcard.2018.04.001

4. ,, Assosiation between renal function and cardiovascular structure and function in heart failure with preserved ejection fraction”. Gori M, Senni M, Gupta D., Chartyan D, et al (2014) Eur , Heart j. 35, 3442-3451 https://dio:10.1093/eurheartj/ehu254

ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2024-6.875 ResearchBib IF: 8.848 / 2024

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-4, ISSUE-5

5 Domenici A., Luciani R., & Principe F. Pulmonary hypertension in dialysis patients. Perit Dial Int. 2010; 30(2): 251-252. DOI:10.3747/ pdi.2009.00082.

6 ,, Chronic kidney disease induces left ventricular overexpression of the prohypertopic micro RNA -212 “ Sarkozy M, Gaspar R. Zvara A.Siska A. Kovari B. Szucs G.et al (2019) Sci . Rep. 9:1302

http://dio10.1038/s41598-018-37690-5

7 ,,Cardiovascular implications of proteinuria: an indicator of chronic kidney disease.’’ Agrawal, V., Marinescu, V., Agarwal, M., and McCullough, P. A. (2009) Nat. Rev. Cardiol. 6, 301–311. http://doi:10.1038/nrcardio.2009.11

8.,,Left ventricular hypertrophy in new hemodialysis patients without symptomatic cardiac disease.” Foley, R. N., Curtis, B. M., Randell, E. W., and Parfrey, P. S. (2010) Clin. J. Am. Soc. Nephrol. 5, 805–813. http://doi:10.2215/CJN.07761109

9. Testani JM, Chen J, McCauley BD, Kimmel SE, Shannon RP (2010) Potential effects of aggressive decongestion during the treatment of decompensated heart failure on renal function and survival. Circulation 122:265–272

10. Verbrugge FH, Dupont M, Steels P, Grieten L, Swennen Q et al (2014) The kidney in congestive heart failure: ‘are natriuresis, sodium, and diuretics really the good, the bad and the ugly?’. Eur J Heart Fail 16:133–142

11. Fudim M, Loungani R, Doerfler SM, Coles A, Greene SJ et al (2018) Worsening renal function during decongestion among patients hospitalized for heart failure: findings from the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial. Am Heart J 204:163–173

12. Милованов Ю.С., Фомин В.В., Милованова Л.Ю. Трудности коррекции гиперфосфатемии у больных хронической почечной недостаточностью. Место не содержащих кальций фосфорсвя-зывающих препаратов. Тер. Архив. 2016; 88(6): 95-100.