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Cardiorenal Lesions and Their Clinical and Immunological Characteristics in Patients With Gout

Tairov Doston Rustamovich, PhD
Samarkand State Medical University, department propaedeutics of internal diseases

Ass, Berdiev Doniyor Khasanovich
Samarkand State Medical University, department propaedeutics of internal diseases

Mannonova Zukhrakhon Akmalovna
Student at Samarkand State Medical University, No. 2, therapeutic work, group 328

Abstract: Cardiorenal syndrome encompasses a spectrum of disorders affecting both the heart and kidneys, wherein acute or chronic dysfunction in one organ can induce acute or chronic dysfunction in the other. This reflects the interconnected interactions between the heart and kidneys at multiple interfaces. These include hemodynamic cross-talk between a failing heart and the kidney's response, as well as changes in neurohormonal markers and molecular inflammatory signatures characteristic of its clinical phenotypes. The mission of this scientific statement is to describe the epidemiology and pathogenesis of cardiorenal syndrome within the context of the evolving clinical-pathological descriptions over the last decade. It also discusses diagnostic and therapeutic strategies applicable to cardiorenal syndrome, summarizes interactions between the heart and kidneys in special populations, such as diabetic patients and kidney transplant recipients, and highlights the role of palliative care for patients with cardiorenal syndrome. Finally, it emphasizes the need to establish a curriculum in cardiorenal therapy to guide future trials and address clinical and research needs in this important area.

Keywords: kidney disease, renal failure, chronic kidney disease, acute kidney injury, end-stage renal disease, albuminuria, congestive/myocardial/heart failure, cardiomyopathy, cardiorenal, pre-dialysis, ultrafiltration.

Introduction

Cardiorenal syndrome (CRS) refers to the interaction between the heart and kidneys, where dysfunction in one organ can lead to dysfunction in the other. The term encapsulates a wide spectrum of disorders characterized by hemodynamic and neurohormonal disturbances, inflammatory processes, and other pathological mechanisms. This concept has evolved significantly since its initial description by Robert Bright in 1836, who documented structural changes in the heart associated with progressive kidney disease. Over the past decades, numerous advances have been made in understanding the cardiorenal connection, including hemodynamic phenotypes, pathophysiology, therapeutic approaches, and clinical outcomes. The interplay between cardiovascular and kidney diseases encompasses a range of phenomena, including hemodynamic interactions during heart failure, the impact of atherosclerosis on both organ systems, neurohormonal activation, cytokine production, and biochemical disruptions along the anemia–inflammation–bone-mineral axis in chronic kidney disease (CKD). The formal definition of CRS was first proposed in 2004 by the National Heart, Lung, and Blood Institute Working Group. This definition emphasized the bidirectional interaction between the heart and kidneys, identifying CRS as a syndrome in which acute or chronic dysfunction in one organ induces acute or chronic dysfunction in the other. Further refinement of CRS classification came in 2008, segmenting the syndrome into five subtypes based on the severity and sequence of organ involvement. This work focuses on exploring the definition, pathophysiology, diagnostic approaches, and therapeutic strategies for CRS. It highlights the clinical phenotypes observed in special populations, such as diabetic patients

and kidney transplant recipients, and underscores the importance of comprehensive end points and a specialized curriculum in cardiorenal therapy to address future clinical and research needs.

Methodology

The authors were tasked with reviewing the current literature and developing a consensus summary on Cardiorenal Syndrome (CRS) based on expert opinions. A draft manuscript was prepared during an initial teleconference, with authors assigned specific sections according to their expertise. All contributors had continuous access to the working document to provide input, and section editors critically reviewed and revised the content. A comprehensive search of relevant articles was conducted in the MEDLINE database to identify additional pertinent studies. Furthermore, team members recommended articles outside the official search scope as needed. References were made to established guidelines where appropriate without duplicating recommendations already contained within those documents. Consensus-driven suggestions and considerations were included to provide guidance to the cardionephrology community. The strict conflict-of-interest policies of the American Association of Cardiology (AAS) were adhered to for all contributors. Each team member disclosed any relevant conflicts, ensuring that over 50% of the group remained free from related conflicts. Neither the chair nor the co-chair reported any industry-related conflicts. Contributors updated their electronic conflict-of-interest disclosures throughout the project and reported new conflicts at the start of each teleconference. Detailed information about individual disclosures is available in the authorship table.

Results

Heart failure (HF) is a complex mechanical and neurohormonal syndrome that results in blood congestion in the lungs and peripheral tissues, manifesting in cardinal symptoms such as exercise intolerance and edema. Diagnosing HF requires the presence of specific signs and symptoms, along with evidence of structural or functional abnormalities in the heart. In the context of Cardiorenal Syndrome (CRS), this extends to both the heart and kidneys. Several diagnostic tools are employed to establish the structural and functional abnormalities characteristic of CRS, including biomarkers, non-invasive imaging, invasive hemodynamic monitoring, and adjunctive volume measurement methods. Biomarkers of cardiac and renal injury offer valuable insights into the clinical context of CRS, enabling early detection of organ damage, monitoring recovery processes, and predicting long-term outcomes. Although cardiac injury biomarkers (troponins) and wall stress markers (BNP/NT-proBNP) are widely used in clinical practice, kidney injury biomarkers are becoming an additional dimension in diagnostic algorithms. Current definitions of acute kidney injury (AKI) rely on changes in creatinine levels or urine output, resulting in a delay of 24–48 hours in taking corrective measures. Emerging biomarkers specific to tubular damage in AKI are being evaluated for their role in identifying CRS phenotypes and guiding therapeutic strategies. Non-invasive imaging techniques, such as echocardiography, play a significant role in assessing congestion markers and impaired blood flow dynamics in CRS. These tools are clinically accessible at the bedside and help in identifying hemodynamic parameters such as central venous pressure (CVP), pulmonary capillary wedge pressure, and left atrial pressure. Echocardiography also provides prognostic value specific to CRS phenotypes.

Discussion and Conclusion

The interconnected cycle of cardiac and renal dysfunction manifests clinically through symptoms associated with volume overload and impaired cardiac pump function, such as dyspnea, fatigue, and chronic pain. In addition to being prevalent in populations with heart failure (HF) and chronic kidney disease (CKD), depression is a common symptom, significantly impacting quality of life. Furthermore, CKD-related bone and mineral disorders are associated with a high incidence of skeletal fractures following falls, exacerbating patient morbidity. Pain management in patients with CRS is critical yet challenging due to contraindications for common therapies like NSAIDs, which can worsen HF and CKD by inducing fluid retention and acute kidney injury (AKI). Opioid use is generally limited, with safer options such as hydromorphone, oxycodone, and fentanyl being preferred in advanced CKD and HF cases. Proper symptom management improves patient outcomes and quality of life. The multifactorial nature of dyspnea in CRS emphasizes the importance of endurance exercises and

volume regulation through tailored diuretic therapy. Innovations like peritoneal dialysis have shown benefits in managing refractory HF symptoms. Depression, as a common comorbidity in CRS, has been identified as an independent predictor of mortality, underscoring the need for comprehensive psychological support. The development of interdisciplinary care models focusing on early detection and targeted management of CRS is critical. Effective communication, advanced care planning, and appropriate integration of palliative care services play a vital role in addressing the complex needs of patients with severe CRS. Over the past decade, global advancements in interdisciplinary approaches to cardiorenal medicine have refined disease definitions, improved diagnostic accuracy, and guided effective treatment strategies. Despite these advancements, patients with concurrent heart and kidney disease continue to face unacceptably high rates of hospitalization, symptom burden, and mortality. The establishment of national quality benchmarks, targeted research funding, and specialized educational programs is essential to bridge existing gaps and enhance outcomes for patients with CRS. Future developments in biomarker research, non-invasive diagnostic tools, and personalized therapy are expected to further improve disease management and patient prognosis.

References

1. Tairov D.R., Berdiev D.KH. "Gout: Immunological and genetic characteristics of the disease." Volume: 3, Issue: 12, Dec-2024. journals.academiczone.net.
2. Tairov D.R., Berdiev D.KH. "Characteristics of Cardiohemodynamic Disorders in Gout Disease." SCIENTIFIC PROGRESS. Volume 2, Issue 3, 2021. ISSN: 2181-1601.
3. Tairov D.R., Berdiev D.KH. "Medication and Non-Medication Methods to Eliminate Hyperuricemia in Gout." "Science and Education" Scientific Journal. Impact Factor 3.848. May 2023, Volume 4, Issue 5.
4. Tairov D.R., Berdiev D.KH. "Characteristics of Heart Functional Disorders in Gout Disease." GALAXY INTERNATIONAL INTERDISCIPLINARY RESEARCH JOURNAL (GIIRJ). ISSN (E): 2347-6915. Vol. 10, Issue 5, May 2022.
5. Tairov D.R., Berdiev D.KH. "Cardiovascular Damage and Metabolic Syndrome Comorbidities in Patients with Gout Disease." 2nd International Congress on Multidisciplinary Studies. Indonesia. February 20th, 2021. conferencepublication.com.
6. Nasonov E.L., Karateev D.E., Satibaldiyev A.M., et al. (2015).
7. Adams P.F. "Current Estimates from the National Health Interview Survey, 1996." Vital Health Stat. Vol. 10, 1999.
8. Saag K.G., Mikuls T.R. "Recent Advances in the Epidemiology of Gout." Curr. Rheumatol. Rep. Vol. 7, 2005, pp. 235–241.
9. Terkeltaub R.A. "Clinical Practice. Gout." N. Engl. J. Med. Vol. 349, 2003, pp. 1647–1655.
10. Tairov E.S., Tairov D.R. "Metabolic Syndrome in Gout: Its Relationship with Renal Functional Disorders." Questions of Science and Education. No. 28 (77), 2019.
11. Wallace K.L., Riedel A.A., Joseph-Ridge N. "Increasing Prevalence of Gout and Hyperuricemia Over 10 Years Among Older Adults in a Managed Care Population." J. Rheumatol. Vol. 31, 2004, pp. 1582–1587.
12. Tairov D.R., Makhmudova H.D. "Cardiovascular Damage in Patients with Gout." Scientific Progress. Vol. 2, No. 2, 2021, pp. 242–249.
13. Yarmukhamedova S.KH., Kamolova D.ZH. "Study of Myocardial Geometry in Hypertensive Patients Using Echocardiography." Achievements of Science and Education. No. 12 (53), 2019.
14. Eliseev M.S., Barskova V.G., Nasonov V.A. "Clinical Significance of Metabolic Syndrome in Gout." Clinical Gerontology. Vol. 12, No. 2, 2006, pp. 29–33.

15. Tairov E.S., Tairov D.R., Solovyev S.K. "Gout: Diagnosis and Treatment." Tashkent, Uzbekistan, 2020.

16. Sowers M.J., Whaley-Connell A., Hayden M.R. "The Role of Obesity in Cardiorenal Syndrome." Cardiorenal Medicine. Vol. 1, 2011, pp. 5–12.

17. Hayden M., Tyagi S.K. "Uric Acid: A New Perspective on an Old Cardiovascular Risk Marker." Nutr Metab (London). Vol. 1, 2004, p. 10.

18. Jalal D.I., Chonchol M., Chen W., Targher G. "Uric Acid as a Therapeutic Target in CKD." Am J. Dis., 2013, Vol. 61, pp. 134–146.

19. Johnson R.J., Segal M.S., Sautin Y., Nakagawa T., Feig D.I., Kang D.H., Gersch M.S., Benner S., Sanchez-Lozada L.G. "Potential Role of Sugar (Fructose) in the Epidemics of Hypertension, Obesity, and Metabolic Syndrome." Am J Clin Nutr. Vol. 86, 2007, pp. 899–906.

20. Steele T. "Human Urate Secretion: Pyrazinamide Suppression Test." Ann Intern Med. Vol. 79, 1973, pp. 734–737.

21. Suliman M.E., Johnson R.J., Garcia-Lopez E., et al. "J-Shaped Relationship Between Uric Acid and Mortality in CKD." Am J. Dis. Vol. 48, 2006, pp. 761–771.

22. Kan D.H., Nakagawa T., Feng L., Watanabe S., Han L., Mazzali M., Truong L., Harris R., Johnson R.J. "The Role of Uric Acid in Kidney Disease Progression." J Am Soc Nephrol. Vol. 13, 2002, pp. 2888–2897.

23. Tseng C. "Correlation of Urinary Albumin Excretion Rate with Uric Acid Levels in Type 2 Diabetes." Kidney Int. Vol. 68, 2005, pp. 796–801.

24. Ono I., Hosoya T., Gomi H., et al. "Serum Uric Acid and Kidney Failure Prognosis in IgA Nephropathy Patients." Nephron. Vol. 87, 2001, pp. 333–339.

25. Thomas G., Sehgal A.R., Kashyap S.R., Srinivas T.R., et al. "Metabolic Syndrome and Kidney Disease: A Systematic Review and Meta-Analysis." Clin J Am Soc Nephrol. Vol. 6, 2011, pp. 2364–2373.

26. Wright A.F., Rudan I., Hastie N.D., Campbell H. "The Complexity of Urate Transporters." Kidney Int. Vol. 78, 2010, pp. 446–452.

27. Gutman A.B., Yu T.F. "Urate Nephrolithiasis." Am J Med. Vol. 45, 1968, pp. 756–779.