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CORRESPONDENCE

The heart–kidney axis in cirrhosis: rethinking hepatorenal and cardiorenal syndromes

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Recently, Nadim et al. [1] redefined hepatorenal syndrome-acute kidney injury (HRS-AKI) in patients with cirrhosis, highlighting the distinct pathophysiological complexities compared to AKI in non-cirrhotic patients. These alterations include hemodynamic disturbances in the arterial circulation, portal hypertension, heightened activity of endogenous vasoactive systems, and systemic inflammation, all which worsen with cirrhosis disease progression. In addition, as cirrhosis advances, the transition from a hyperdynamic circulatory state to progressive decline in cardiac reserve owing to cirrhotic cardiomyopathy results in decreased cardiac output, which may contribute to renal hypoperfusion and, thus, precipitate HRS–AKI [2]. Nadim et al. [2] further note that hypoperfusion from hypovolemia accounts for approximately half of AKI cases in patients with cirrhosis, intrinsic causes (e.g., acute tubular necrosis) account for around 30%, and HRS accounts for 15–20%, with < 1% attributable to postrenal obstruction. However, cirrhotic cardiomyopathy—the predominant cardiac manifestation in these patients—often overlaps with congestive heart failure and pulmonary hypertension, which affect up to 60% of patients with cirrhosis [3, 4]. This overlap presents diagnostic challenges, as both reduced arterial perfusion and venous congestion contribute to decreased kidney function, though the role of congestion in HRS remains less defined. For example, a study of 127 patients with HRS–AKI undergoing right heart catheterization showed that ~ 60% had elevated right atrial pressures (> 10 mmHg), elevated pulmonary capillary wedge pressure (> 15 mmHg), or both [4]. This finding challenges current guidelines for HRS, which often assume volume depletion and recommend albumin administration and diuretic discontinuation.

Point-of-care ultrasound (PoCUS) has gained interest as a tool for noninvasive hemodynamic assessment, offering the ability to estimate elevated right and left heart filling pressures. In addition to ruling out hydronephrosis and assessing chronic kidney disease (CKD) severity through parenchymal evaluation, ultrasound can also be used to identify pulmonary or venous congestion (indicating fluid intolerance) and assess fluid responsiveness by estimating stroke volume and cardiac output through the velocity-time integral of the left ventricular outflow tract [5]. These measurements are particularly valuable for distinguishing between low stroke volume or high cardiac output states, as seen in cirrhotic cardiomyopathy [5].

A recent study found that 21% of cirrhotic patients with AKI, clinically considered adequately volume-repleted and classified as HRS-AKI, showed evidence of intravascular volume overload on PoCUS [6]. This finding suggests that in a significant number of patients with AKI and cirrhosis, congestion observed via PoCUS, or chest imaging may indicate an acute cardiorenal syndrome physiology rather than HRS–AKI (Fig. 1). These imaging modalities could be invaluable for characterizing AKI phenotypes in patients with combined cardiac, hepatic, and renal dysfunction, and for guiding individualized treatment approaches, including decongestion through negative fluid balance, paracentesis, or optimized heart failure/pulmonary hypertension-directed therapies

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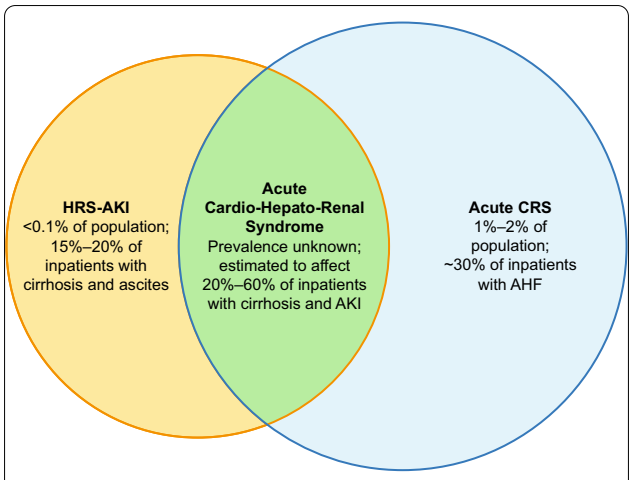

Fig. 1 Prevalence and overlap of acute kidney injury (AKI) phenotypes in patients with cirrhosis and cardiorenal syndrome. This figure illustrates the estimated prevalence of hepatorenal syndrome-AKI (HRS-AKI), acute cardio-renal syndrome (CRS), and acute cardio-hepato-renal syndrome. While the prevalence of HRS-AKI [S1, 2] and CRS [S2–S4] is relatively well-defined, the exact prevalence of acute cardio-hepato-renal syndrome remains unknown, though it is estimated to affect 20–60% of inpatients with cirrhosis and AKI [4, S5], highlighting the diagnostic challenges across these overlapping syndromes

(Fig. S1). Notably, targeted decongestion, aimed at restoring renal perfusion pressure, may reverse kidney dysfunction, providing insights into AKI reversibility, enhancing the likelihood of renal recovery, mitigating AKI-to-CKD transition, and ultimately improving clinical outcomes.

Supplementary Information

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Author contributions

FH-S and GR-G conceived the outline and drafted the initial version and the figure. JR, CR, and MHR reviewed and revised the manuscript and the figure for critical intellectual content and actively participated in shaping up the final document.

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Declarations

Conflicts of interest

CR acts as an advisory board member for ASAHI, Baxter, GE, Jafron, and Medtronic, and has received speaker fees from Astute, bioMérieux, B. Braun, CytoSorbents, ESTOR, FMC, and Toray, all unrelated to this work. MHR has received consultant fees from Baxter Healthcare; serves as on the Data Safety Monitoring Boards of clinical trials sponsored by Reata, Travere, and Astra Zeneca, all unrelated to this work. FH-S, JR, and GR-G have no conflicts of interests to disclose.

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