A VExUS Perspective on Cardiorenal Syndrome: Case Report and Literature Review

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Abstract

Cardiorenal syndrome has long been attributed to reduced cardiac output and renal hypoperfusion, but contemporary evidence increasingly identifies venous congestion as a primary driver of acute kidney injury, particularly in right-sided heart disease. The Venous Excess Ultrasound Score (VExUS) offers an innovative, non-invasive, organ-specific Doppler method to quantify systemic venous hypertension and better define the hemodynamic contributors of renal dysfunction. We report a case illustrating this paradigm, in which decompensated right-sided heart-failure led to high-grade venous congestion on VExUS assessment and resulted in acute kidney injury that improved with targeted decongestive therapy. This case underscores the growing relevance of VExUS as a practical bedside tool for diagnosing congestion-mediated renal injury and guiding individualized fluid management in cardiorenal patients.

Introduction

For decades, the prevailing explanation for cardiorenal syndrome centered on low cardiac output, systemic hypotension, and reduced renal perfusion, positioning acute kidney injury as a downstream consequence of forward flow failure. [1-4] More recent pathophysiologic understanding, however, has redefined this model by demonstrating that renal dysfunction, particularly in right-sided heart failure is more directly linked to *venous congestion* than to inadequate arterial supply. [2,5-7] In this updated framework, elevated right-sided pressures are transmitted retrograde into the renal venous system, increasing interstitial and intratubular pressure to the point of compressing the filtration apparatus, an effect increasingly described as "**renal tamponade"**. [1,6] Rather than a perfusion deficit alone, it is the sustained venous hypertension and resultant mechanical opposition to glomerular filtration that drive renal injury, marking a notable paradigm shift in the previously established pathophysiology of cardiorenal syndrome. [2,6]

In this context, VExUS (Venous Excess Ultrasound) has emerged as a ultrasound-based bedside method to directly quantify systemic venous congestion when traditional markers fail to capture organ-level pressure overload. [8-12] VExUS integrates measurement of inferior vena cava caliber with hepatic, portal, and intrarenal venous Doppler interrogation to determine the degree of backward pressure transmission affecting the liver, splanchnic vasculature, and kidneys. [12-14] By grading the severity of waveform alterations, including hepatic systolic reversal, portal pulsatility, and loss of continuous intrarenal venous flow, VExUS provides a structured and reproducible congestion index that outperforms single-parameter volume assessments. [12-16] As such, it represents an innovative clinical tool that not only diagnoses venous congestion more efficiently, but also enables more individualized and physiologically guided decongestive therapy. [11,15-20]

Herein, we present the case of a patient whose acute kidney injury was clarified by VExUS assessment, which identified severe venous congestion as the primary hemodynamic insult.

Case presentation

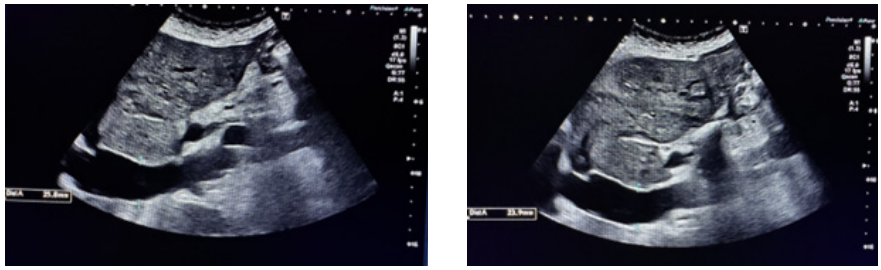
A 77-year-old woman presented with one week of progressive dyspnea, profound fatigue, asthenia, anorexia, nausea, and vomiting, with a marked decline in urine output becoming evident over the preceding forty-eight hours prior to admission. Her past medical history was significant for well-controlled type 2 diabetes mellitus, confirmed by an admission HbA1c of 5.5%, and longstanding severe tricuspid regurgitation. Initial laboratory evaluation revealed anemia

(hemoglobin of 9 g/dL), with no signs of ongoing inflammation (normal leukocyte count and C-reactive protein levels) and severe renal function impairment (urea of 322 mg/dL and creatinine of 8.6 mg/dL), accompanied by mild hyponatremia and hyperkalemia at 133 mmol/L and 5.8 mmol/L, respectively. She denied recent infections, new medications, or non-steroidal anti-inflammatory drug use.

On admission, the patient appeared volume overloaded but hemodynamically stable. Initial evaluation was consistent with acute kidney injury (AKI) and decompensated right-sided heart failure.

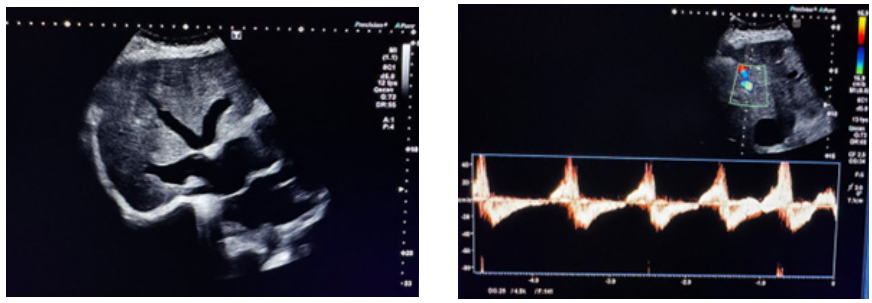
Point-of-care ultrasonography demonstrated a plethoric inferior vena cava measuring 25.8 mm, with minimal inspiratory collapse (23.9 mm), indicating markedly elevated right atrial and central venous pressure. Hepatic venous Doppler revealed systolic flow reversal, consistent with severe tricuspid regurgitation and backward pressure transmission into the hepatic venous network. Portal vein examination showed a pulsatility index greater than 50%, and intrarenal venous Doppler displayed discontinuous monophasic flow, a pattern that reflects elevated renal venous confluence pressures and interstitial congestion. No ascites, pleural effusion, or pericardial fluid was identified. Cardiac ultrasound confirmed preserved left ventricular systolic function but reiterated the presence of severe tricuspid regurgitation, reinforcing the systemic venous hypertension mechanism.

FIGURE 1 (a. and b.). Inferior vena cava (IVC) before (a.) and after (b.) inspiration. noting dilated IVC, with inspiratory collapse less than 50%.



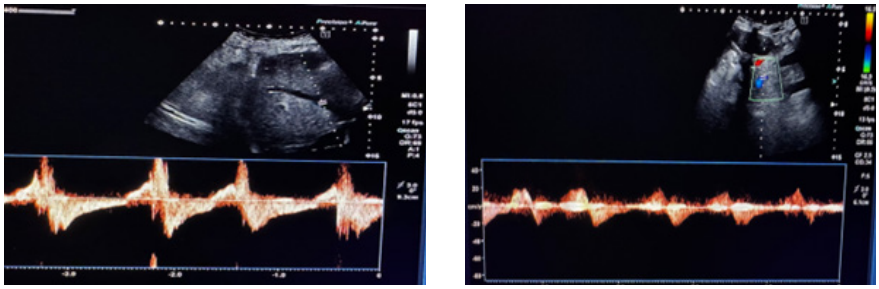
a. b.

FIGURE 2 (a. and b.). Dilated hepatic veins with systolic flow reversal



a. b.

Figure 3 (a. and b.). Portal vein (a.) showing a pulsatility index (PI)>50%, and intrarenal vein (b.) displaying discontinuous monophasic flow.



a. b.

COMPONENT	FINDING	CONGESTION GRADE
IVC	Dilated, non-collapsible	Severe
HEPATIC VEIN	S-wave reversal	Severe
PORTAL VEIN	PI > 50%	Severe
RENAL VEIN	Discontinuous flow	Severe

VExUS Score = 3, indicating high-grade systemic venous congestion.

The constellation of Doppler findings, including a dilated non-collapsing inferior vena cava, hepatic systolic flow reversal, highly pulsatile portal venous waveform, and discontinuous intrarenal venous pattern, was consistent with a VExUS grade 3 profile, denoting severe systemic venous congestion.

Management focused initially on decongestion using diuretics to improve volume overload alongside correction of electrolyte imbalance, with a particular focus on hyperkalemia. Serial reassessment of venous Doppler parameters enabled close monitoring of the trajectory of renal and systemic decongestion. With this conservative, diuretic-based approach alone, renal function gradually improved, and the patient did not require ultrafiltration.

Discussion

In recent years, the traditional paradigm that acute kidney injury (AKI) during acute decompensated heart failure (ADHF) is mainly a consequence of low cardiac output and renal hypoperfusion has been increasingly challenged. Instead, a growing body of evidence indicates that systemic and renal *venous congestion*, rather than just forward failure, may represent a principal pathophysiologic mechanism for what is often labeled as type 1 cardiorenal syndrome (CRS-1). [1-

7] In this conceptual framework, elevated right atrial pressure, transmitted via the inferior vena cava (IVC) to the hepatic, splanchnic, and renal veins, leads to renal venous hypertension, increased interstitial and intratubular pressure, and a dramatic reduction in effective glomerular filtration pressure. [1-7] This "backward failure" model is especially relevant in patients with right-sided heart failure, severe tricuspid regurgitation (TR), or valvular lesions that amplify transmission of venous pressure to end-organs. [2,6,7]

The physiologic plausibility of this mechanism has been strongly supported by experimental data. For example, animal studies using selective abdominal IVC constriction demonstrated that moderate increases in abdominal venous pressure, within ranges observed in human venous congestion, induced structural alterations in the kidney (glomerulomegaly, Bowman's space widening, renal interstitial edema) and liver (congestive hepatopathy), even with preserved cardiac output and without primary renal insult. [1] This suggests that venous hypertension alone can initiate and likely perpetuate organ damage. Such data challenge the notion that only low-output states or systemic hypotension lead to renal injury in heart failure, and underscore the need to consider venous hemodynamics when interpreting worsening kidney function. [2,6]

In clinical practice, however, detecting and quantifying venous congestion is not straightforward. Traditional markers such as elevated jugular venous pressure, peripheral edema, liver enlargement, ascites represent often late, relatively insensitive, or nonspecific findings. [6,7] Invasive measurement of central venous pressure (CVP) provides only limited insight and may not reflect organ-level congestion or renal venous hypertension. [6,7-10] Recognizing this gap, clinicians and researchers developed the VExUS protocol to provide a noninvasive, bedside, organ-based assessment of systemic venous congestion, combining IVC imaging with Doppler interrogation of hepatic, portal, and intrarenal veins. [12-15]

Originally described by William BeaubienSouligny and colleagues in a cohort of postoperative cardiac surgery patients, VExUS demonstrated that a high-grade congestion pattern (VExUS grade C/3), defined by a dilated IVC plus severely abnormal Doppler waveforms in two or more organ veins, was strongly associated with the subsequent development of AKI. [12] This seminal work validated the concept that ultrasound-based markers of venous congestion might be more predictive of renal injury than conventional parameters. [12,13] Subsequent studies, confirmed that VExUS scores ≥ 2 (i.e., moderate to severe congestion) were associated with a significantly increased risk of AKI, particularly in patients undergoing cardiac surgery. [8-11,13,15]

More recently, the VExUS protocol has been increasingly applied in heart failure and cardiorenal syndrome populations. A 2025 review of VExUS in the context of CRS emphasized that this multiparametric ultrasound approach offers real-time, noninvasive, organ-level insights into systemic venous congestion,

providing a more physiologically meaningful assessment than static pressure measurements or volume status estimates. [13,15,16] Another important advantage is that VExUS can serve not only as a diagnostic tool, but also as a dynamic guide for decongestive therapy: serial VExUS examinations can help clinicians titrate diuretics, or escalate to ultrafiltration when necessary, based on whether congestion at the organ level is truly improving. [8-11,13,15] In patients with right-sided HF and severe TR, Doppler patterns in the portal and intrarenal veins may be more informative than hepatic vein waveforms, because TR often disrupts hepatic venous flow independent of actual congestion, making hepatic Doppler less reliable in isolation. [14,16]

Despite its promise, VExUS is not without limitations. Its accuracy depends heavily on operator expertise, acoustic window quality, and correct interpretation of Doppler waveforms. [12,13] Some recent ICU studies documented low prevalence of high VExUS grades and failed to find a consistent association between admission VExUS and AKI or mortality, suggesting that context including underlying disease, patient population, timing matters greatly. [8,10,11] Moreover, VExUS does not simply reflect "volume overload"; rather, it captures a complex interplay between cardiac function, filling pressures, organ compliance, and venous return, meaning that a high VExUS may not always correspond to excess intravascular volume, but might instead reflect poor right heart function, decreased venous return, or altered venous compliance. [6,7,16]

Nonetheless, in a patient such as ours with decompensated right-sided heart failure, severe tricuspid regurgitation, systemic congestion, and rapid deterioration of renal function, the VExUS framework provides a compelling, mechanistically coherent explanation for AKI. [6,7,17-19] In this context, worsening creatinine may not reflect "pre-renal" hypoperfusion in the traditional sense, but rather "congestive nephropathy" driven by renal venous hypertension, elevated interstitial and intratubular pressure, and impaired filtration. [1,2,5,6,17] Using VExUS to monitor the extent of venous congestion and guide decongestive therapy, escalating and combining diuretics, or early initiation of ultrafiltration in diuretic-refractory congestion, could help restore renal perfusion gradients, and improve the chances of renal recovery. [8–11,13,15,20]

Thus, this case underscores a broader shift in our understanding of cardiorenal syndrome, from a model dominated by forward failure, to one in which backward failure via venous congestion plays a central, sometimes dominant, role. [2–7,17,19] The VExUS protocol, by visualizing and quantifying organ-level congestion in real time, offers a practical and physiologically grounded tool for both diagnosis and management of congestion-driven CRS-1. [12–16,20,21,22] As the literature continues to evolve, integrating VExUS into the routine assessment of acutely decompensated heart failure, especially right-sided and valvular subtypes, may facilitate more individualized and effective decongestive strategies, with the potential to improve renal and overall clinical outcomes.

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