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Case Report

Venous Excess Doppler Ultrasound: A Visual Guide to Decongestion in Cardiorenal Syndrome

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Keywords
Point-of-care ultrasound · Venous excess Doppler ultrasound · Cardiorenal syndrome · Nephrology

Abstract
Promptly recognizing congestion, both clinical and hemodynamic, is paramount in the management of patients with heart failure. The pathophysiology of congestion involves a complex interplay of absolute fluid gain, volume redistribution from venous capacitance beds to the central venous circulation, inadequate excretion due to renal dysfunction, salt and water retention, and endothelial dysfunction. While congestive nephropathy is gaining wider recognition as a distinct variant of hemodynamic acute kidney injury (AKI), there are limited bedside diagnostic tools for proper evaluation of these patients. In this manuscript, we describe a case of AKI where POCUS helped us diagnose clinically silent congestion as well as monitor the response to therapy. A patient with heart failure with mildly reduced ejection fraction was initially administered intravenous fluids for rise in serum creatinine attributed to volume depletion. However, POCUS demonstrated a completely different scenario with severe venous congestion. Both sonographic stigmata of congestion and serum creatinine improved with diuretic therapy. Furthermore, serial venous excess Doppler ultrasound scans facilitated the visualization of decongestion in real time.

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Introduction

Congestive nephropathy is a hemodynamic phenotype of acute kidney injury (AKI) that is being increasingly recognized in patients with heart failure and other fluid overload states. It is characterized by venous congestion and renal interstitial edema as a consequence of elevated right heart pressures [1]. It is well known that residual congestion at hospital discharge, particularly in conjunction with worsening renal function, portends worse outcomes in patients with heart failure [2]. As such, early detection of congestion and effectively addressing it are of utmost importance. Unfortunately, physical examination and other commonly used bedside parameters to detect congestion have significant limitations leaving a diagnostic void, especially in patients with improving symptoms but persistent hemodynamic congestion [3, 4]. Over the past few years, point-of-care ultrasonography (POCUS) has evolved as an adjunct to physical examination in various medical specialties including nephrology. It allows objective assessment of hemodynamic congestion at the bedside by the clinician who is taking care of the patient [5]. Moreover, it is a non-invasive tool and radiation free. Additionally, venous excess Doppler ultrasound (VExUS) is a grading system recently developed to quantify systemic venous congestion and monitor the response to decongestive therapy [6]. Herein, we describe a case of hemodynamic AKI where POCUS, in particular VExUS, helped us in arriving at a correct diagnosis as well as monitoring the decongestion process in real time.

Case Presentation

A 61-year-old man with a history of hypertension and heart failure with mildly reduced left ventricular ejection fraction (44%) was admitted for evaluation and management of nonspecific symptoms concerning for acute coronary syndrome. While acute coronary syndrome was ruled out, his serum creatinine increased to 1.65 mg/dL from a presentation value of 1.02 mg/dL (baseline 1–1.1 mg/dL) over 4 days. Patient reported reduced oral intake/loose stools prior to presentation, but there was no significant change in weight compared to baseline. Fractional excretion of sodium of <1% and the N-terminal pro-B-type natriuretic peptide (NT-proBNP) from a week ago was 311 pg/mL (close to normal). A renal sonogram excluded hydronephrosis. Review of chest radiograph did not demonstrate any pulmonary edema. Echocardiogram from 3 days prior reported normal left ventricular filling pressures and an inferior vena cava (IVC) diameter of 1.8 cm with more than 50% collapsibility consistent with a normal right atrial pressure (RAP) of 3 mm Hg. There were no major valvular abnormalities. Based on this information, he was thought to be volume depleted and intravenous fluids were administered. He received a total of 2 L of isotonic fluids over 2 days with documented blood pressures ranging from 105/65 mm Hg to 144/96 mm Hg and heart rate 80–92 bpm during this period.

However, nephrology consultation was requested as the serum creatinine only marginally improved to 1.6 mg/dL and the urine output decreased. Physical examination was unremarkable; there was no significant pedal edema. The blood pressure was 137/98 mm Hg with a pulse rate of 87 bpm. The documented urine output was only 350 mL in the previous 24 h, but the patient reported 2-3 unmeasured voids. Weight on the day of consult was 74 kg compared to 73.2 kg 4 days prior. Daily weights were not available in the interim. Because of the suspicion for hemodynamic AKI, POCUS-enhanced physical examination was performed by the nephrology team, which demonstrated following findings: (a) Lungs showed B-lines in the lateral lung zones suggestive of mildly increased extravascular lung water. (b) Maximal IVC diameter was 3.1 cm with minimal inspiratory collapse consistent with an elevated RAP of 15 mm Hg. (c) Doppler ultrasound of the systemic veins (i.e., VExUS scan) revealed severe

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congestion. Specifically, hepatic vein Doppler revealed diastolic wave larger than the systolic wave, portal vein Doppler showed 100% pulsatility, and intrarenal venous waveform demonstrated only the diastolic wave (i.e., venous flow only during the diastole). Based on these findings, acute congestive nephropathy was diagnosed, and the patient was started on intravenous diuretic. On follow-up examination, the IVC and venous waveforms showed consistent improvement (Fig. 1) and the patient was transitioned to oral diuretic. Lung B-lines resolved as well. Serum creatinine simultaneously improved to 1.16 mg/dL (1.6–1.4 to 1.25–1.16), confirming the diagnosis of congestive nephropathy.

Discussion

Congestive nephropathy denotes renal dysfunction due to venous congestion and consequent impaired organ perfusion. This term was recently coined [1], but several studies have previously shown that elevated RAP (or central venous pressure) is associated with worsening renal function even though the cardiac index is preserved [7]. This observation has pathophysiologic basis as the renal perfusion pressure is the difference between mean arterial pressure and RAP; if the RAP is elevated, the perfusion pressure drops leading to impaired renal perfusion. Further, activation of the renin angiotensin-aldosterone system, sodium and water retention, interstitial edema, endothelial dysfunction, and elevated intra-abdominal pressure all contribute to increased pressure within the encapsulated kidney (renal tamponade), leading to organ dysfunction. In addition, renal dysfunction can exacerbate the existing fluid overload, resulting in a vicious cycle. As such, it is imperative that objective assessment of fluid status is crucial for appropriate management of patients with fluid disorders [8]. Nevertheless, paucity of accurate bedside diagnostic tools to detect hemodynamic congestion has left the cardiorenal physicians with some precision guesswork to do in day-to-day practice. POCUS attempts to fill this void by providing information on important hemodynamic variables, thereby facilitating the diagnosis when interpreted in conjunction with the clinical-laboratory parameters. The diagnostic accuracy of POCUS is proven beyond doubt when compared to conventional physical examination parameters as well as chest radiography [9, 10]. Therefore, it is not surprising that our patient was found to have significant congestion despite normal physical examination findings and a normal appearing chest radiograph. Poor oral intake and diarrhea reported by the patient were confounding factors that favored volume depletion. Similarly, weight is subject to documentation and measurement errors and does not reflect congestion due to fluid redistribution. Laboratory parameters such as fractional excretion of sodium are too non-specific to discriminate various etiologies of AKI [11]. Furthermore, we often rely on "recent" laboratories and imaging, which are not necessarily reliable in patients with tenuous hemodynamic status. After all, hemodynamics are "dynamic." Our case nicely illustrates this point as the echocardiogram at the presentation reported normal RAP whereas POCUS (after fluid administration) revealed a plethoric IVC with substantial increase in size. While the recent NT-proBNP was near normal, we obtained a repeat value immediately after POCUS, which was significantly elevated at 48,670 pg/mL. It eventually improved to 537 pg/mL at 2-week follow-up in the primary care clinic.

Among the POCUS parameters, VExUS merits special mention in this context as it allowed us not only to diagnose congestive nephropathy with confidence but also ensure effective decongestion at discharge. VExUS grading system was devised to quantify systemic venous congestion (a reflection of RAP and venous compliance) and predict organ injury. In a cohort of cardiac surgery patients, presence of severe flow abnormalities in multiple Doppler patterns with a dilated IVC showed strong association with AKI (HR: 3.69 CI 1.65–8.24 p = 0.001),

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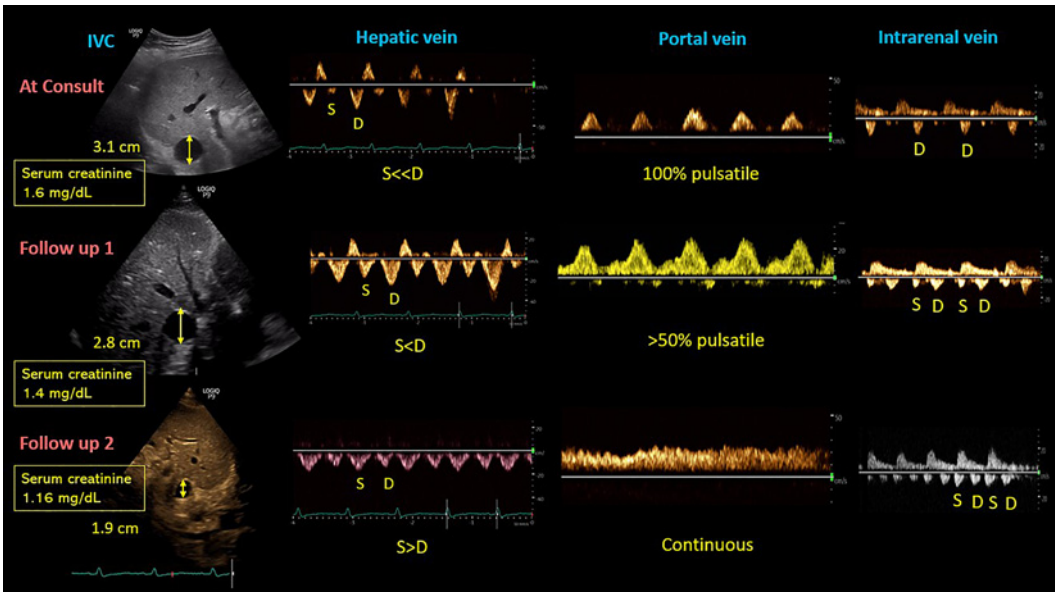

Fig. 1. IVC (transverse view) and VExUS (venous excess Doppler ultrasound) findings at the time of nephrology consultation and follow-up examinations.

outperforming isolated RAP measurement [12]. In normal state, hepatic vein Doppler has two below-the-baseline waves, S and D signifying venous return in systole and diastole. These are like X and Y descents of the central venous pressure tracing and the amplitude of the S-wave is greater than that of the D-wave. The waveform is considered mildly abnormal when the S-wave is smaller than the D-wave and severely abnormal when the S-wave is reversed. These changes occur as a result of elevated RAP, right ventricular dysfunction, and functional tricuspid regurgitation leading to reduced gradient between the right heart and venous return in the setting of fluid overload. Normal portal vein is relatively continuous unlike the hepatic vein as it is separated from the systemic venous circulation by hepatic sinusoids. When the RAP is elevated, it is increasingly transmitted to the portal vein leading to pulsatility. It is considered mildly abnormal when the pulsatility is 30–50% and severely abnormal when it is more than 50%. Intrarenal vein Doppler is normally continuous similar to that of portal vein, mildly abnormal when it is pulsatile with distinct S, D components (biphasic), and severely abnormal when it is monophasic with only D-wave below the baseline (i.e., S-reversal similar to that of hepatic vein). These waveform transitions are illustrated in Figure 2. In our patient, one can note the transition from severe congestion to near-normal state with intravenous diuretic therapy. At the time of discharge, RAP (estimated by IVC ultrasound), hepatic, and portal vein waveforms have normalized. The intrarenal waveform remained mildly abnormal with a biphasic pattern, but it is expected to improve slowly in parallel with resolution of the interstitial edema.

In summary, we demonstrate the utility of POCUS-enhanced physical examination in the management of cardiorenal syndrome. We believe our case adds to the emerging literature prompting physicians to think about fluid tolerance [13] before contemplating empiric intravenous fluid therapy. On a note of caution, POCUS should not be interpreted in isolation or considered a replacement for careful history taking or conventional physical examination. Above discussed POCUS parameters do have limitations. For example, B-lines in the lung can be seen in conditions such as pulmonary fibrosis, infection, contusion, etc. Hepatic vein Doppler interpretation is error prone without a simultaneous electrocardiogram, portal vein

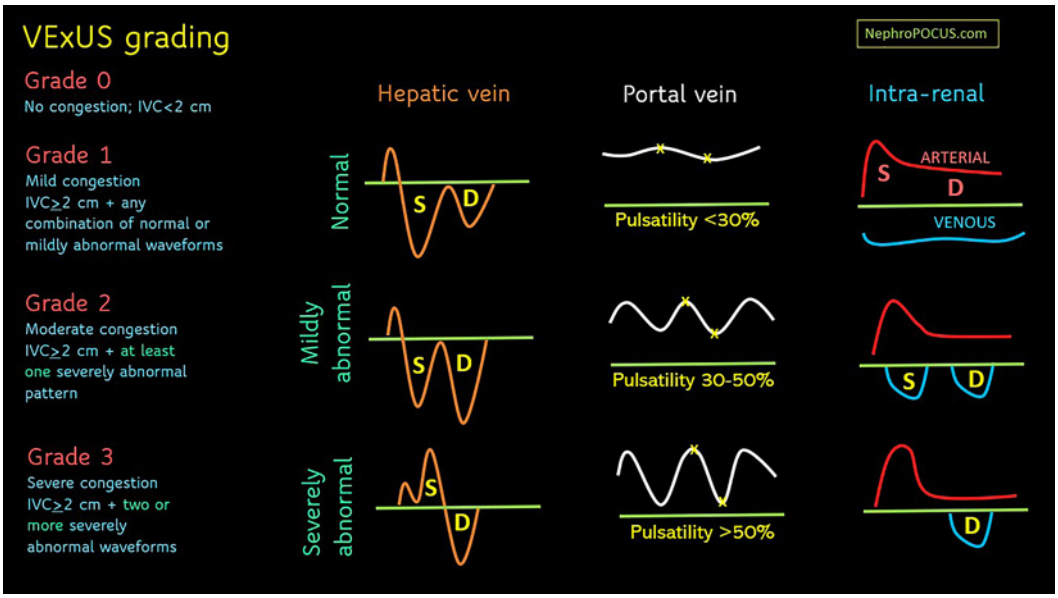

Fig. 2. VExUS grading for the quantification of systemic venous congestion. Please see the manuscript text for description of the waveforms (image courtesy of NephroPOCUS.com, obtained with permission).

waveform may not be trustworthy in cirrhosis, and intrarenal waveform is not well studied in patients with pre-existing chronic kidney disease. Therefore, clinical context must guide the interpretation of POCUS findings. Future studies should consider evaluating the impact of POCUS-guided management on measurable outcomes such as effective decongestion at discharge and hospital readmissions. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material at https://doi.org/10.1159/000531709.

Statement of Ethics

Ethical approval is not required for this study in accordance with local or national guidelines. Written informed consent was obtained from the patient for the publication of the details of their case study and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Sirisha Gudlawar drafted the initial version of the manuscript. Abhilash Koratala procured the ultrasound images, reviewed, and revised the manuscript for critical intellectual content. Both the authors have approved the final version for submission.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

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