A 78-Year-Old Male With Acute on Chronic Kidney Injury

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A 78-year-old man with a medical history significant for heart failure with mid-range left ventricular (LV) ejection fraction, moderate aortic and tricuspid regurgitation (AR and TR), and normal right ventricular (RV) function, as well as chronic kidney disease and atrial fibrillation, was admitted to the hospital with nausea, abdominal pain, and dyspnea. He was found to have an acute-on-chronic kidney injury and elevated liver enzymes. He was admitted to the Internal Medicine service with a presumptive diagnosis of decompensated heart failure with a phenotype of right-sided failure and congestive hepatopathy, and he was placed on IV diuretics. During his hospital stay, his diuretic titration proved extremely challenging. His creatinine was labile, rising as high as 300 μM (3.39 mg/dL), from a baseline of 140 μM (1.58 mg/dL), and his diuretic dosing was frequently adjusted. His clinical volume examination was difficult given his known TR and body habitus.

Midway through his stay, he was reevaluated with the addition of whole-body point-of-care ultrasound (POCUS) for volume assessment. On the day of assessment, he was hemodynamically stable, with a BP of 108/46 mm Hg, heart rate of 60 beats/min, and oxygen saturation of 97% on ambient air.

Cardiovascular examination revealed a variable S1, a normal S2, and a grade II/VI holosystolic murmur best heard at the apex; as well as a grade II/VI diastolic murmur heard at the left upper sternal border. Jugular venous pressure was elevated at 5 cm above the sternal angle. Respiratory examination showed mildly decreased air entry bilaterally. There was no pedal edema. Relevant bloodwork included a creatinine of 229 μM (2.59 mg/dL)—up from 148 μM (1.67 mg/dL) 4 days prior—as well as a sodium of 124 mM (down from 132 mM 4 days prior). In the preceding days, his furosemide had been downtitrated significantly because of concerns about a soft BP, cool extremities, and possible hypovolemic hyponatremia.

POCUS was performed to augment the clinical volume assessment (Videos 1, 2).

Question 1: What changes does the initial cardiac POCUS reveal compared with his previously known heart disease (Video 1)?

Question 2: What do his inferior vena cava (IVC) and hepatic and portal venous profiles suggest about the cause of his ongoing kidney injury (Video 2)?
**Answer 1:** LV function is severely depressed, estimated ejection fraction 20% to 30%, with accompanying LV dilatation. RV systolic function is also newly impaired. There is qualitative evidence of severe AR, moderate-to-severe TR, and moderate mitral regurgitation. As a surrogate for cardiac output, note that his recorded LV outflow tract velocity-time integral (LVOT VTI) at this time was within the normal range at 21 cm, although this must be considered in the context of his severe AR (Fig 1).

**Answer 2:** The IVC is plethoric with no noted collapsibility and, furthermore, his hepatic veins are dilated, which argues against a hypovolemic state and suggests possible venous congestion. The hepatic venous waveform demonstrates reversal of the S wave on spectral Doppler, and portal vein Doppler likewise shows highly abnormal flow reversal (Figs 2, 3). These findings, in the presence of TR and RV failure, suggest severe venous congestion. His rise in creatinine, therefore, was thought to be primarily secondary to venous congestion caused by the combination of recently held diuretics and worsened biventricular function with valvular disease progression.

Given these findings, the patient was restarted on aggressive IV diuretics. Over the ensuing days, his creatinine decreased to 180 μM (2.04 mg/dL), and his sodium improved to 130 mM. However, the following week, he once again experienced a rise in his creatinine, this time to over 400 μM (4.52 mg/dL), while on aggressive diuresis for ongoing POCUS signs of organ congestion. It was hypothesized that, given his severely depressed LV function and severe AR, a degree of elevated LV end-diastolic pressure was actually necessary to preserve forward flow. LVOT VTI was not remeasured at this time but would have been a useful marker to corroborate the assumed physiology. His diuretics were therefore gently downtitrated, resulting in a gradual decrease in his creatinine back to his pre-admission baseline. The patient was doing well from this perspective and was being prepared for discharge by the allied health team. Unfortunately, he suffered an unwitnessed arrest in hospital, potentially arrhythmogenic given his underlying cardiac disease, and died before hospital discharge.

**Discussion**

The determination of volume status is one of the most challenging aspects of clinical medicine. Traditional noninvasive parameters such as physical examination of the jugular venous pressure are imperfect and limited in their utility. POCUS can greatly augment the clinical examination for volume overload; specifically, the IVC, heart (for signs of right or left heart failure), and thorax (for pleural effusions and B-lines suggestive of pulmonary edema). However, none of these techniques are without pitfalls, particularly for the assessment of systemic, rather than pulmonary, venous congestion. They must each be taken in context and with an awareness of the pitfalls of the examination. As such, there is increasing interest in the use of Doppler evaluation of the hepatic, portal, and renal vessels as additional markers of organ congestion. This is a relatively novel and very advanced POCUS technique. Although the evidence is still evolving, these techniques may be useful additions to a whole-body POCUS.

**Figure 1** – LVOT VTI is within normal limits at 21 cm, though this should be considered in the context of his severe AR. AR = aortic regurgitation; LVOT = left ventricular outflow tract; VTI = velocity-time integral.

**Figure 2** – Hepatic vein spectral Doppler shows abnormal flow with reversal of the S wave, consistent with severe congestion.
examination and may help to identify early signs of venous congestion and volume intolerance (Discussion Video).

Four vessels may be interrogated in this manner, which are ideally performed and interpreted in combination. A group has recently proposed a Venous eXcess Ultrasound Score grading system that has been validated in a post-cardiac surgery population as predictive of congestion-related acute kidney injury, using a combination of the hepatic, portal, and renal veins, in the setting of a plethoric IVC. In addition, studies support the examination of each of the aforementioned vessels individually in other clinical states. Specific to our case, an abnormal hepatic vein tracing is consistent with venous congestion, but it also can be seen in primary hepatic pathological conditions or stand-alone TR. Portal venous flow abnormalities have been associated with RV failure, increased central venous pressure, and liver congestion and have been shown to be helpful in titrating diuretics in right-sided heart failure. Cirrhosis, in the absence of cardiac disease, may result in increased portal venous pulsatility caused by arterial-portal shunting. Portal hypertension, in and of itself, does not contribute to pulsatility, but its cause may—such as cirrhosis, right heart failure, or TR. In our case, the renal vessels were not able to be interrogated because of very poor image quality.

Though an advanced application, POCUS users with a thorough understanding of spectral Doppler will find these techniques very similar to other applications involving the interrogation of blood vessels. Evaluation of the hepatic vein starts with B-mode identification of the vessel, usually in a subxiphoid position with the phased array probe (which allows for ECG gating if needed). Color Doppler is then used to confirm a strong signal in the vessel. Next, pulsed-wave Doppler is used to generate a spectral tracing, which can be interpreted on a spectrum ranging from normal to consistent with severe congestion, as in our case. A normal hepatic vein is triphasic (although it has four components), with its two primary components, the S wave (seen in systole) and the D wave (seen in diastole), both present below the baseline, representing blood flow toward the heart; with the magnitude of the S wave being greater than the D wave. In cases of severe congestion, the S wave becomes diminished, and, eventually, reversed (if tricuspid regurgitation exists), such as in this patient.

The portal vein may be likewise evaluated in a similar fashion. It is typically identified in a trans-hepatic plane using the curvilinear probe on the abdominal preset. Color, and then pulsed-wave Doppler, are applied, revealing a tracing that again follows a predictable progression from normal to severely congested. Normal portal venous blood flow is continuous, with phasic variation caused by respiration, and located above the baseline (heptopetal flow). In states of increasing congestion, the portal waveform becomes increasingly pulsatile, and, in severe instances, biphasic with reversed flow, as noted in our case.

In this patient, these techniques proved a useful adjunct to other POCUS and clinical parameters. However, it must be underscored that these examinations should not be interpreted in isolation; as our case demonstrates, many other factors must be considered when determining the optimal treatment regimen. Each tracing is also not without pitfalls and may be affected by other factors, such as intraabdominal pathology, thrombosis/stenosis, or TR. However, we believe that these techniques are particularly useful in the heart failure patient population, because these patients frequently suffer cardiorenal acute kidney injury, and determination of optimal volume status is often very challenging.

Reverberations

1. A whole-body approach to POCUS can augment the clinical volume examination, particularly in challenging patients.

2. The evaluation of abdominal vessels (including the hepatic, portal, and renal interlobular veins) with Doppler ultrasound is an advanced POCUS technique that can help identify signs of venous congestion, particularly in the heart failure population.

3. Care must be taken with these novel techniques to be aware of the limitations of each examination, and to integrate findings with other clinical and POCUS parameters.
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Additional information: To analyze this case with the videos, see the online article.

References