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CASE REPORT

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Utility of Doppler ultrasound derived hepatic and portal venous waveforms in the management of heart failure exacerbation

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Abstract

Doppler ultrasound assessment of hepatic and portal vein waveforms aids in the management of patients with heart failure by noninvasively monitoring the efficacy of decongestive therapy. In the right clinical context, these waveforms can be used as an adjunct to physical examination and inferior vena cava ultrasound.

KEYWORDS

Doppler, heart failure, hepatic vein, nephrology, POCUS, point-of-care ultrasound, portal vein

1 | INTRODUCTION

Obtaining an accurate assessment of the volume status and systemic hemodynamics is paramount in the management of patients with heart failure, particularly those with accompanying renal dysfunction. Though bedside clinical examination coupled with vital signs has traditionally been the standard in guiding fluid removal strategy,[1] there is accumulating evidence that these parameters are unreliable.[2] In the recent past, clinician performed point-of-care ultrasonography (POCUS) has emerged as a valuable bedside diagnostic tool.[3] It is intended to answer focused and often binary questions such as "is the inferior vena cava (IVC) full?", "is there a pericardial effusion?", "is the global cardiac function adequate?", etc In this context, Doppler ultrasound assessment of hepatic and portal veins is emerging as a marker for systemic congestion and end-organ dysfunction.[4] Though the data are limited at this time, this technique can be an important adjunct to IVC ultrasound, which is more commonly used as a surrogate for right atrial pressure and venous congestion. Herein, we present a case of heart failure with illustrative images of hepatic and portal venous waveforms before and after decongestive therapy.

2 | CASE DESCRIPTION

A 43-year-old man with a history of hypertension, diabetes mellitus type 2, congestive heart failure with reduced ejection fraction, and chronic kidney disease stage 3 with a baseline glomerular filtration rate of ~45 mL/min was admitted to the hospital for right lower extremity necrotizing fasciitis requiring below-knee amputation. Postoperatively, he developed cardiac arrest and was successfully resuscitated; however, he subsequently developed volume overload with pulmonary edema and acute kidney injury requiring renal replacement therapy. The echocardiogram showed severely reduced left ventricular ejection fraction of ~20% and a trace tricuspid regurgitation (TR). The IVC diameter was ~2.4 cm with minimal respiratory variation, portal vein Doppler revealed a pulsatile waveform with flow reversal, and the hepatic vein Doppler demonstrated retrograde flow during ventricular systole, all suggestive of systemic venous congestion. Hepatic and portal vein waveforms are shown in Figure 1. After a few days, his urine output improved, and renal replacement therapy was stopped. Repeat Doppler studies were performed approximately 2 weeks after the initial scan, which demonstrated significant improvement in portal vein pulsatility as

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well as return of antegrade flow during ventricular systole suggestive of resolving venous congestion (Figure 2). We continued with diuretic therapy and patient's renal function returned to near baseline at the time of discharge.

3 | DISCUSSION

In patients with acute decompensated heart failure, residual clinical congestion at hospital discharge is associated with worse outcomes.[5] While the reference standard for congestion assessment is measurement of right atrial pressure (RAP) and pulmonary capillary wedge pressure (PCWP) using pulmonary artery catheterization,[6] its invasive nature precludes routine use. Estimation of RAP using IVC ultrasound has emerged an attractive option, particularly with the advent of hand carried ultrasound devices. The venous circulation holds nearly 70% of the circulating blood volume.[7] In volume overload, the distention of the IVC is often seen, and this enlargement reflects transmission of pressure changes from the right atrium to end organs. In general, IVC diameter less than 21 mm with an inspiratory collapse of more than 50% is considered to indicate normal RAP in spontaneously breathing patients. High RAP is indicated by more than 21 mm diameter and less than 50% collapse, while anything in between (eg, less than 21 mm with less than 50% collapse) is correlated with intermediate RAP.[8] Interestingly, one study reported that the sensitivity for predicting RAP more than 10 mm Hg was 82% with IVC ultrasound and only 14% from jugular venous pulse inspection.[9] Nevertheless, IVC ultrasound is subject to numerous pitfalls limiting its utility to assess volume status when used in isolation. For example, the vessel is routinely measured in its long axis (anteroposterior diameter) but the inspiratory collapse can occur craniocaudally and also mediolaterally resulting in errors.[10] In addition, one must be vigilant about anatomic factors leading to false collapse such as diaphragm compressing the IVC.[11] Furthermore, the changes in the size of the IVC depend on variations in intrathoracic pressure and lung compliance. Vigorously breathing patients with large intrathoracic pressure swings can collapse their IVC despite the RAP being high. On the other hand, patients with poor inspiratory effort may not be able to collapse their IVC despite low RAP. Another common source of error is small-appearing IVC even with high RAP in patients with tense ascites due elevated intra-abdominal pressure.

In the light of all these pitfalls associated with IVC ultrasound, hepatic and portal vein waveforms can provide additive value in assessment of volume status as well as organ dysfunction. The normal portal venous waveform is described as "phasic" (Figure 3), with gentle undulation, primarily influenced by atrial contraction.[12] With increases in central venous pressure, RA pressure changes during the cardiac cycle can be transmitted to the portal circulation leading to a "pulsatile" flow pattern as in our case. Pulsatility can be quantified using pulsatility index (%) calculated as 100× [maximum velocity-minimum velocity/maximum velocity], which in turn can be used to monitor response to decongestive therapy. Normal pulsatility index is generally considered less than 30%. Portal vein Doppler assessment may be a surrogate for renal congestion as well, which is important in managing patients with heart failure and acute kidney injury. For example, in one study, portal waveform alterations were independently associated with acute kidney injury in postcardiac surgery patients.[13] Another study linked an increase in the portal vein pulsatililty index to abnormal liver tests in patients with volume overload.[14] More recently, Singh, et al described the utility of portal vein waveforms in accurate volume assessment and management of hypotonic hyponatremia.[15]

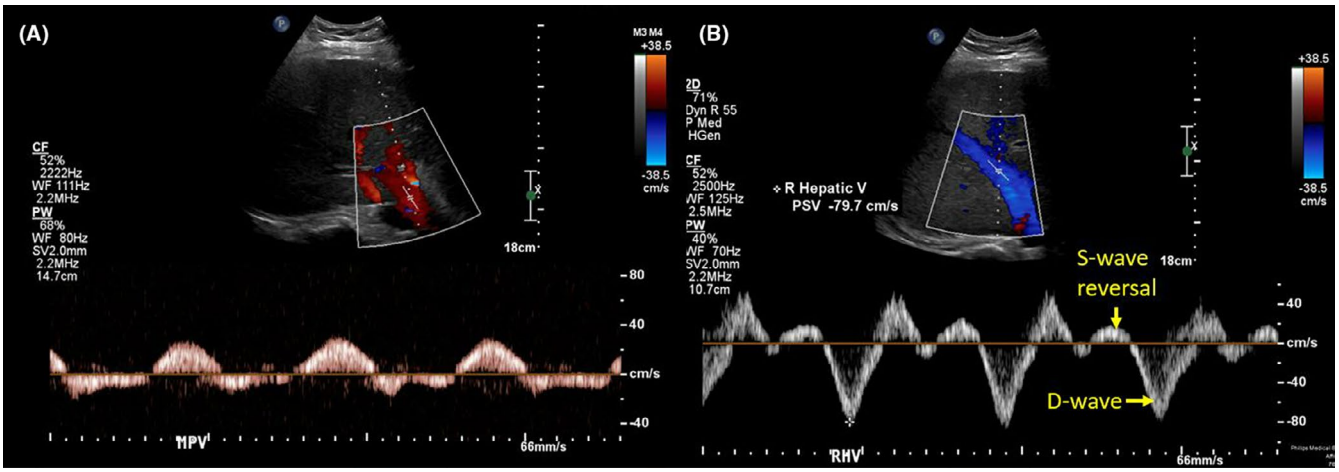

FIGURE 1 Portal (A) and hepatic (B) vein waveforms at the time of cardiac decompensation. Note the pulsatile portal vein and monophasic [D is the only anterograde wave] hepatic vein tracings

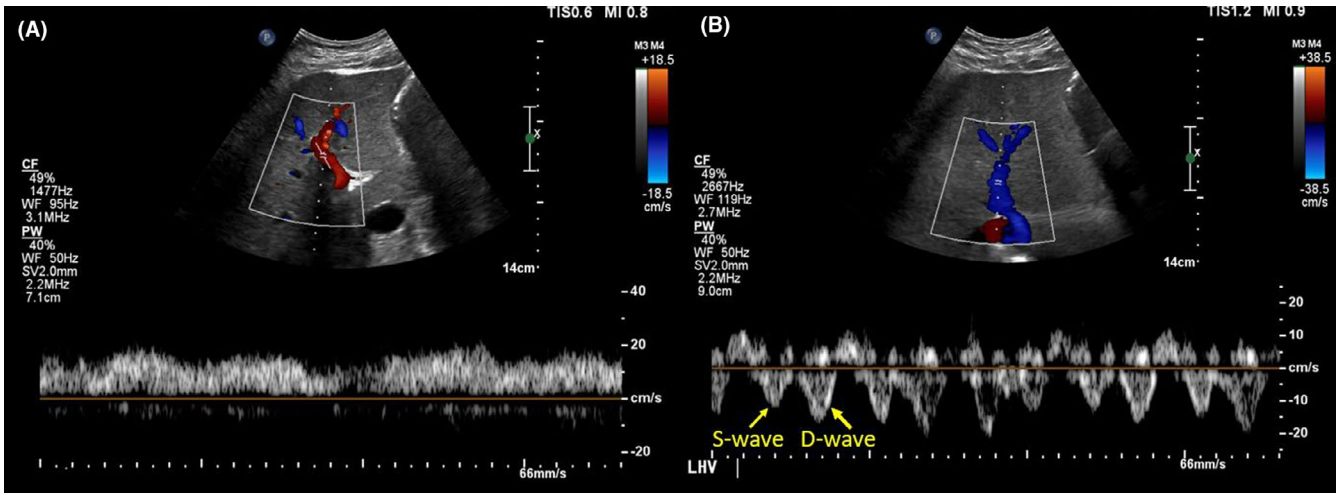

FIGURE 2 Portal (A) and hepatic (B) vein waveforms after decongestive therapy. Note improved pulsatility of the portal vein and return of the S-wave below the baseline on the hepatic vein tracing

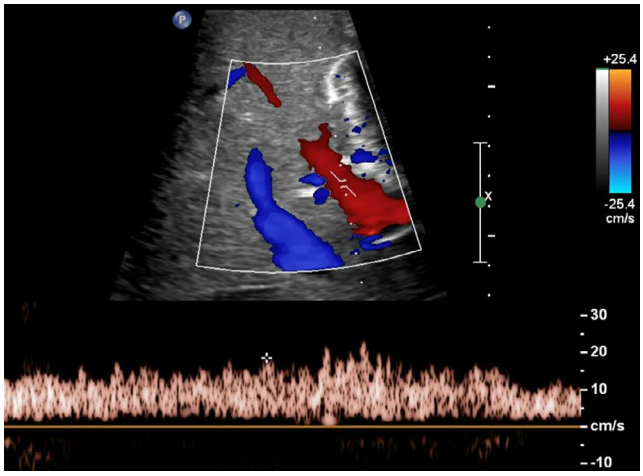

FIGURE 3 Normal portal vein waveform with minor phasic variation

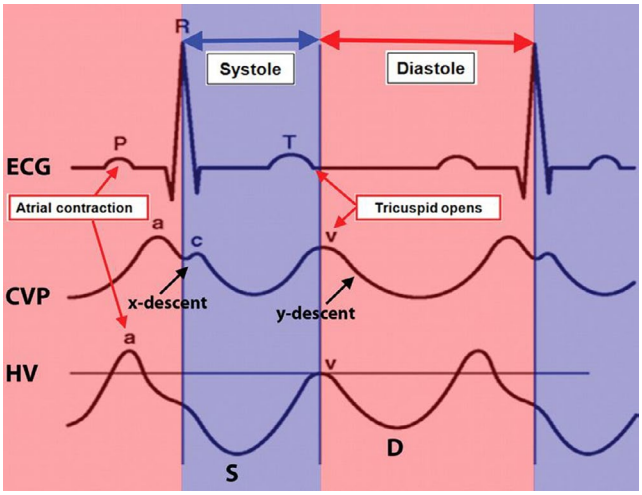

FIGURE 4 Illustration of normal time-correlated electrocardiographic (ECG) findings, central venous pressure (CVP) tracing, and hepatic venous (HV) waveform. The peak of the retrograde a wave corresponds with atrial contraction, which occurs at end diastole. The trough of the antegrade S-wave correlates with peak negative pressure created by the downward motion of the atrioventricular septum during early to mid-systole. The peak of the upward-facing v-wave correlates with opening of the tricuspid valve, which marks the transition from systole to diastole. The peak of this wave may cross above the baseline (retrograde flow) or may stay below the baseline (ie, remain antegrade). The trough of the antegrade D-wave correlates with rapid early diastolic right ventricular filling. The cycle then repeats. Adopted from reference 12 with kind permission of the Radiological Society of North America

Hepatic venous waveform is slightly complex compared with portal waveform. Most of the blood flow in hepatic vein is anterograde (displayed below the baseline) toward the heart and consists of multiple individual waves analogous to a jugular venous pulse tracing. Changes that increase RAP (eg, atrial contraction at end diastole) will cause above the baseline deflections and changes that decrease RAP with cause a downward deflection. The "a"-wave is the first wave and an upward (retrograde) deflection generated by increased RAP from atrial contraction at end diastole. The next wave is "S"-wave, an anterograde deflection caused by decreasing RAP during early mid-systole. But if the tricuspid valve is open (as in regurgitation), retrograde deflection can occur (reversal of S-wave). The third wave is the "V"-wave, which is a retrograde deflection due to increase in RAP caused by continued venous return toward end systole. The peak of the wave can be below or above the baseline. The last wave is the "D"-wave, another anterograde deflection caused by decreasing RAP from rapid early diastolic right ventricular filling. Figures 4 and 5 demonstrate components of the normal hepatic venous waveform. Normally, the S-wave is greater (or deeper) than the D-wave. As the RAP increases, D becomes deeper than S

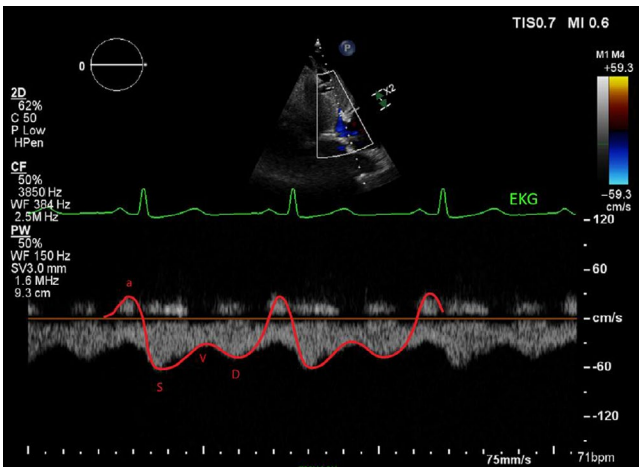

FIGURE 5 Normal hepatic vein waveform. Correlate with EKG findings as described in Figure 4

and then the S becomes retrograde leaving the D-wave as the only anterograde deflection (monophasic pattern).[16]

In our case, one can easily appreciate the "to and fro" pattern of the portal vein (100% pulsatility) and hepatic waveform with retrograde S-wave when the patient was congested. On the repeat scan, the portal vein pulsatility improved as well as the S-wave in the hepatic vein returned to below the baseline though not greater than D indicating improvement in RAP. As mentioned above, the patient did not have significant tricuspid regurgitation, eliminating a confounding factor for the retrograde S-wave.

4 | CONCLUSION

Evaluation of blood flow pattern in the portal vein and hepatic veins using Doppler ultrasonography adds one more data point to assessing volume status and the clinicians, particularly nephrologists and internists need to be comfortable with interpretation of these waveforms. Though it did not drastically change the management in our case, it can be immensely helpful in cases where physical examination and IVC ultrasound findings are discordant. Future research is needed to study whether adding this sonographic parameter to traditional physical examination and/or IVC ultrasound to guide therapy leads to improvement in outcomes. In addition, whether a personalized treatment strategy in the outpatient setting aimed at preventing or reversing portal and hepatic flow alterations has favorable impact on hospital admissions of heart failure patients would be an interesting research question.

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CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Both authors: made substantial contribution to the preparation of this manuscript; SS: drafted the initial version of the manuscript and performed literature search; AK: senior nephrologist, and reviewed and revised the manuscript for critically important intellectual content.

INFORMED CONSENT

Informed consent has been obtained from the patient for the publication of this case report.

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