

LETTER TO THE EDITOR

Evaluation of rapid changes in haemodynamic status by Point-of-Care Ultrasound: a useful tool in cardioneurology

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Haemodynamic assessment of venous congestion is a fundamental and challenging task in the management of patients with right heart failure. Carbohydrate antigen 125 (CA 125) has recently emerged as a useful biomarker of right heart failure that can guide decongestion in patients with cardiorenal syndrome [1, 2]. However, CA 125 is not able to track rapid haemodynamic changes given its prolonged half-life (5–12 days) [3]. Recently, CA 125 but not NT-ProBNP was shown to have a strong correlation with renal congestion evaluated by Doppler ultrasound [4]. Backwards transmission of pressure from the right heart to the abdominal organs results in altered patterns of venous flow that can be quantified at the bedside using Point-of-Care Ultrasound (POCUS) [5, 6]. Both renal and splanchnic congestion occur in the setting of right heart failure and show good agreement with each other [7, 8]. Splanchnic congestion, evaluated by portal Doppler ultrasound, is an independent predictor of acute kidney injury (AKI) in cardiac surgery patients and can track decongestion in patients with cardiorenal syndrome [8, 9]. Changes in right atrial pressure should lead to almost instantaneous changes in the flow pattern of splanchnic veins. This could be particularly relevant for the evaluation of patients with rapidly changing haemodynamics. We present evidence from two cases showing that portal venous Doppler can detect rapid changes in haemodynamic status.

Portal venous Doppler was performed on two patients with right heart failure and splanchnic congestion. Hepatic vein Doppler was also obtained. A phased array probe with the predefined abdominal settings was used. The complete method for portal and hepatic Doppler has been previously published

[8]. Splanchnic congestion was graded by means of the portal vein pulsatility fraction (PF): $= 100 \times [(V_{\max} - V_{\min})/V_{\max}]$. A PF <30% is considered normal and a PF >50% is considered severe [8]. All measurements were repeated two to three times. The Institutional Review Board approved this study (NMM-3897-21-21-1) and written informed consent was obtained.

Case 1 (Figure 1): a 68-year-old female with morbid obesity and pulmonary hypertension was evaluated for oliguric AKI. Serum creatinine was 3.43 mg/dL and BNP was 1375 pg/mL. POCUS revealed a non-collapsible IVC of 2.4 cm and a portal vein PF of 140%. Echocardiogram showed right ventricular dysfunction and a preserved LVEF. Intermittent haemodialysis was started. During the first session, UF volume was 3000 mL at a rate of 1000 mL/h (8.3 mL/kg/h). Portal vein PF decreased to 55%, 34% and 31% during the first, second and third litre of volume removed, respectively. After the third litre, she developed rapid atrial fibrillation and UF was stopped. Eight hours later, portal vein PF increased to 65%, which was attributed to vascular refilling. Similar results were observed the following day (UF rate 4 mL/h). Urine output improved, kidney function returned to baseline and portal vein flow became non-pulsatile.

Case 2 (Figure 2): a 50-year-old female with lupus nephritis and ESRD. A brachiocephalic AV fistula had been placed 2 years prior after which the patient developed symptomatic heart failure. BNP was 6037 pg/mL. POCUS revealed a dilated non-collapsible IVC and a portal vein PF of 77%. Echocardiogram revealed severe pulmonary hypertension, dilated right heart and torrential tricuspid regurgitation. LVEF was preserved. Portal

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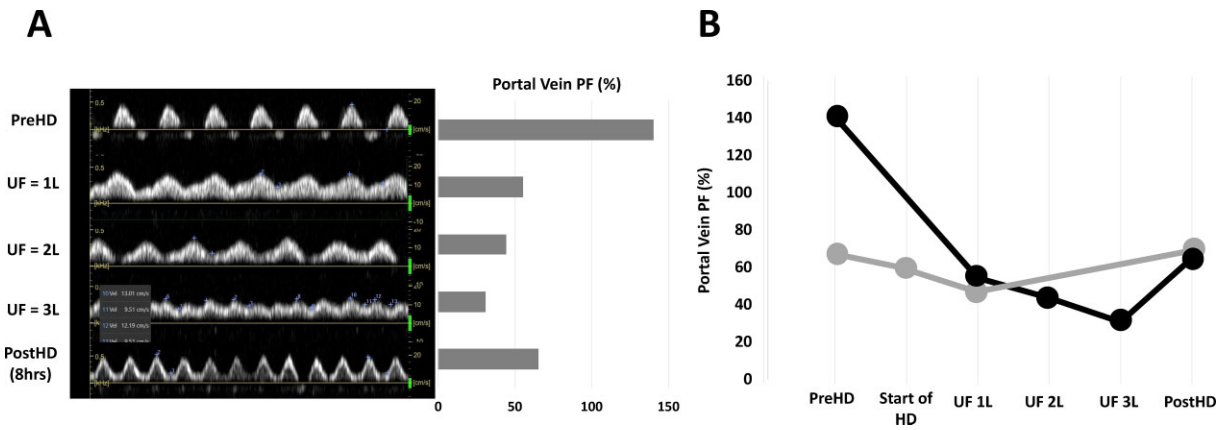


FIGURE 1: (A) Portal vein Doppler obtained before, during each hour and after the first haemodialysis session in Case 1. Grey bars show the portal vein pulsatility fraction at each timepoint. (B) Portal vein PF at each timepoint during the first (dark line) and second (grey line) haemodialysis sessions.

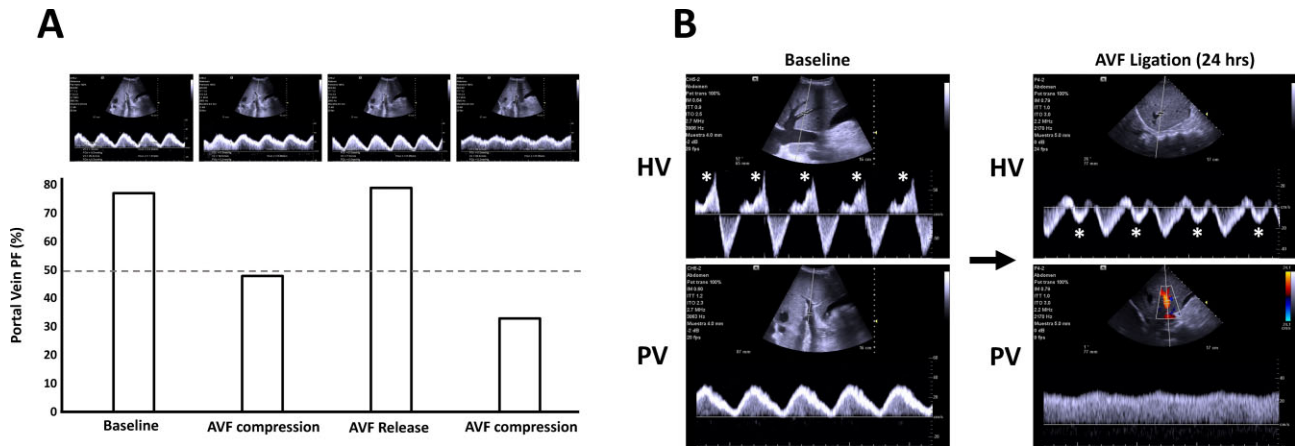


FIGURE 2: (A) Portal vein pulsatility fraction obtained at baseline and during AV fistula compression. This manoeuvre was repeated twice. (B) Hepatic vein (HV) and portal vein (PV) Doppler before and after AV fistula ligation. Asterisks show hepatic vein 'S' wave reversal before ligation (severe tricuspid regurgitation), which was reversed after AV fistula ligation.

vein PF measured seconds after manual compression of the AV fistula decreased to 48% and 33% after repeating the manoeuvre. The patient was transitioned to peritoneal dialysis and AV fistula was ligated. IVC and portal vein flow normalized and only moderate tricuspid regurgitation remained. Patient showed marked symptomatic improvement.

In the first case, rapid decongestion during ultrafiltration displayed a real-time improvement in splanchnic congestion. Given rapid volume removal is linked to intradialytic adverse events, the first case raises the possibility that the rate of splanchnic decongestion could serve as a predictor of ultrafiltration-related complications in patients with right heart failure. The second case illustrates that the effect of manually compressing the AV fistula on splanchnic congestion can be detected within seconds. Manual compression of a high-flow AV fistula leads to an immediate reduction in RV preload and an increase in LV afterload [10]; the ability to detect these rapid changes suggests this manoeuvre could have a role in predicting significant improvement of cardiac dysfunction after fistula ligation. These speculations warrant further study as accurate haemodynamic monitoring in patients with right heart failure is a pressing issue for the field of cardioneurology. These cases illustrate how rapid haemodynamic changes can

be assessed using portal vein Doppler. This assessment could become complementary to that provided by other congestion biomarkers with a longer half-life of elimination such as CA 125 in a similar fashion to how serum glucose and haemoglobin A1C are currently used in the management of diabetes.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

There are no new data associated with this article.

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