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CASE REPORT

Intrarenal venous flow in cardiorenal syndrome: a shining light into the darkness

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Abstract

The aim of this case report is to assess the potential role of intrarenal Doppler ultrasonography as a non-invasive method to evaluate intrarenal venous flow (IRVF) in acute heart failure (AHF) and concomitant renal dysfunction. We report a case of an 81-year-old woman with valvular heart disease (previous mitral valve replacement) that presented with acutely decompensated heart failure and concomitant worsening renal function (WRF). In addition to complete physical examination, laboratory analysis, and echocardiography, IRVF was assessed at baseline and 48 h after the administration of diuretic treatment. At admission, physical examination and echocardiography revealed signs of intravascular congestion (jugular venous distension and severely dilated inferior vena cava). In addition, a significant increase in serum creatinine from 1.23 to 1.81 mg/dL was noted without signs of hypoperfusion at clinical evaluation. At baseline, intrarenal Doppler ultrasonography showed a monophasic IRVF pattern indicating a severely elevated interstitial renal pressure. After aggressive decongestion, a dynamic behaviour was found in IRVF changing from monophasic to biphasic pattern in parallel with an improvement in clinical parameters and renal function (serum creatinine changed from 1.81 to 1.44 mg/dL). In this case of a patient with AHF and WRF, IRVF changed after aggressive decongestion in agreement with clinical evolution. According to these findings, this technique could provide valuable information for identifying patients with a 'congestion kidney failure' phenotype. Further studies are needed confirming this observation and evaluating the potential role of this technique for guiding decongestive therapy in patients with AHF and WRF.

Keywords Congestive heart failure; Diutretic treatment; Intrarenal venous flow in cardiorenal syndrome

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Introduction

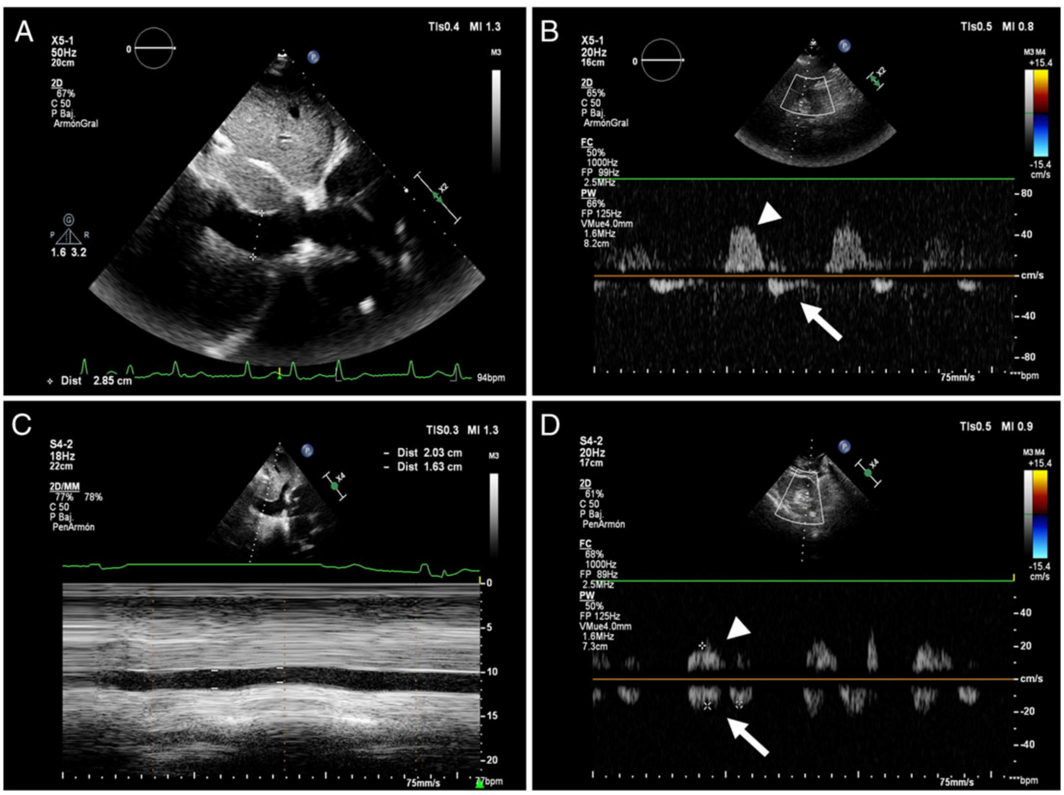
Worsening renal function (WRF) in acutely decompensated heart failure (ADHF) was classically attributed to reduced cardiac output and intravascular volume depletion secondary to the use of diuretics. However, recent evidence has challenged this notion and postulate venous renal congestion as a key component of the cardiorenal interaction in ADHF.[1,2] However, there is a lack of well-stablished tools for distinguishing between both haemodynamic phenotypes. Recently, intriguing data suggest the potential role of intrarenal Doppler venous flow patterns (IRVF) assessed by echography as an attractive tool to better understand intrarenal haemodynamics.[3,4]

Case report

We report a case of an 81-year-old woman with valvular heart disease (previous mitral valve replacement) that presented with increased dyspnoea, abdominal discomfort, and 1.5 kg weight gain. Ambulatory daily heart failure treatment was bisoprolol 1.25 mg, furosemide 160 mg, chlorthalidone 25 mg, acetazolamide 125 mg, and spironolactone 12.5 mg. No evident cause for clinical decompensation was noted. Vital signs at presentation were as follows: heart rate 69 bpm, arterial blood pressure 90/56 mmHg (usual values and no signs of hypoperfusion), respiration rate 19 per minute, and capillary Sa02 96%. The physical examination revealed abdominal and jugular venous distension, positive hepatojugular reflux, but no peripheral oedema nor rales. Echocardiography showed a normally functioning prosthetic mechanical mitral valve, preserved left ventricular ejection fraction, dilated right ventricle with preserved systolic function (tricuspid annulus plane systolic excursion of 20 mm), a moderate tricuspid regurgitation with an estimated pulmonary artery systolic pressure of 65 mmHg, and a severely dilated inferior vena cava (*Figure 1A*). Given that the patient had a mechanical mitral valve, echocardiographic assessment of left ventricular end diastolic pressures was not possible. The laboratory analysis showed an N-terminal prohormone of brain natriuretic peptide of 3205 pg/mL and WRF, with an increase in serum creatinine from 1.23 to 1.81 mg/dL (a decline in estimated glomerular filtration rate from 45 to 27 mL/min/1.73 m^2). A Doppler ultrasonography evaluating renal interlobar venous flow showed a monophasic pattern (*Figure 1B*), suggesting a severe elevation in intrarenal venous pressures.

In this case, according to physical and Doppler findings, renal venous congestion appeared to be casually linked to the occurrence of WRF. Accordingly, we started an aggressive intravenous diuretic treatment during the first 48 h (cumulative furosemide dose of 500 mg). At this time, we reassessed the echo-Doppler, registering a reduction of the diameter of inferior vena cava (*Figure 1C*) and a change from monophasic to biphasic venous flow pattern in the intrarenal ultrasonography (*Figure 1D*), indicating an improvement in venous flow and, therefore, a reduction of venous pressures. Importantly, this change occurred in parallel with clinical improvement (resolution of abdominal discomfort, 2 kg weight loss) and an improvement in renal function (serum creatinine and estimated glomerular filtration rate changed from 1.81 to 1.44 mg/dL and from 27 to 38 mL/min/1.73 m^2, respectively).

Figure 1 Images of echocardiography performed at admission and at 48 h visit. (A) Inferior vena cava measurement at admission showing a diameter of 2.85 cm. (B) Intrarenal venous flow* at admission showing a monophasic pattern. (C) Inferior vena cava measurement at admission at 48 h visit showing a maximum diameter of 2.03 cm with inspiratory collapse of <50%. (D) Intrarenal venous flow* at 48 h visit showing a biphasic discontinuous pattern. *Pulsed-wave Doppler signal of interlobar renal vessel recorded with the use of a commercially available system (Philips Healthcare) with a sector transducer frequency range of 2.5 to 5 MHz. The upward Doppler signal corresponds to arterial flow (arrow head) and the downward Doppler signal to venous flow (arrow).



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Changes in laboratory parameters after diuretic treatment are shown in Figure S1.

Discussion

Intrarenal venous Doppler ultrasonography has been used for years to evaluate non-cardiac conditions linked to elevated interstitial renal pressure such as obstructive uropathy or diabetic nephropathy. However, recent evidence suggests the potential application of this imaging modality for evaluating intrarenal haemodynamics in heart failure.

Iida *et al*. evaluated the characteristics of intrarenal Doppler ultrasonography and their prognostic implications in a cohort of stable, non-ischaemic heart failure patients.[3] The authors described three IRVF patterns (continuous, biphasic discontinuous, and monophasic discontinuous) and correlated each pattern with clinical, echocardiographic, and haemodynamic (measured by right heart catheterization) variables. Interestingly, the authors observed that the biphasic and monophasic discontinuous patterns were associated with increased right atrial pressure features and correlated strongly with clinical outcomes independent of conventional prognostic factors such as right atrial pressure levels and cardiac index. In the same line, Nijst *et al*. evaluated the variations in IRVF signals after volume loading (1 L fluid expansion over 3 h) and unloading in stable heart failure patients and control subjects.[4] Continuous IRVF pattern was the most common signal at baseline in both groups. However, after fluid administration, most of heart failure patients developed a biphasic pattern in contrast with control subjects where no variations were observed. Interestingly, 1 h after administration of intravenous diuretics, 70% of heart failure patients returned to the baseline situation (continuous flow pattern). Nevertheless, it is important to note that these two studies were carried out in stable and euvolemic patients.

To the best of our knowledge, this is the first report showing a change in IRVF following aggressive depletive treatment in a patient with ADHF and WRF on admission. Interestingly, Doppler findings were correlated with clinical decongestion and renal improvement.

Worsening renal function in the setting of ADHF has a complex and multifactorial pathophysiology, which explains why patients show mixed clinical response to treatment.[5] Based on the findings by Iida *et al*. and Nijst *et al*., IRVF patterns assessed by Doppler may have a role for identifying the renal haemodynamic disturbances in patients with heart failure. We speculate that biphasic and monophasic discontinuous venous flow patterns could help physicians to identify a phenotype of 'congestive renal failure' in which an aggressive decongestive therapy would be recommended. Conversely, a continuous venous flow pattern would identify those with normal renal venous pressures in which a more conservative diuretic strategy should be indicated. Moreover, the dynamic behaviour in response to therapy observed in our patient makes this imaging modality an attractive tool for monitoring treatment response.

Conflict of interest

None declared.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. Changes in laboratory parameters after diuretic treatment. (A) Glomerular filtration rate. (B) NT-proBNP. (C) Hemoglobin. (D) Hematocrit. eGFR: estimated glomerular filtration rate; NT-proBNP: amino-terminal pro-brain natriuretic peptide.

References

1. Hanberg JS, Sury K, Wilson FP, Brisco MA, Ahmad T, Ter Maaten JM, Broughton JS, Assefa M, Tang WH, Parikh CR, Testani JM. Reduced cardiac index is not the dominant driver of renal dysfunction in heart failure. *J Am Coll Cardiol* 2016; **67**: 2199–2208.
2. Mullens W, Abrahams Z, Francis GS, Sokos G, Taylor DO, Starling RC, Young JB, Tang WH. Importance of venous congestion for worsening of renal function in advanced decompensated heart failure. *J Am Coll Cardiol* 2009; **53**: 589–596.
3. Iida N, Seo Y, Sai S, Machino-Ohtsuka T, Yamamoto M, Ishizu T, Kawakami Y, Aonuma K. Clinical implications of intrarenal hemodynamic evaluation by Doppler ultrasonography in heart failure. *JACC Heart Fail* 2016; **4**: 674–682.
4. Nijst P, Martens P, Dupont M, Tang WHW, Mullens W. Intrarenal flow alterations during transition from euvolemia to intravascular volume expansion in heart failure patients. *JACC Heart Fail.* 2017; **5**: 672–681.
5. Nuñez J, Núñez E, Miñana G, Bayés-Genis A, Sanchis J. Worsening renal function in acute decompensated heart failure: the puzzle is still incomplete. *JACC Heart Fail* 2016; **4**: 232–233.

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