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EDITORIAL

Portal vein pulsatility: An important sonographic tool assessment of systemic congestion for critical ill patients

Stavros Dimopoulos, Michael Antonopoulos

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Abstract

In this editorial we comment on the article by Kuwahara *et al*, published in the recent issue of the *World Journal of Cardiology*. In this interesting paper, the authors showed a correlation between portal vein pulsatility ratio, examined by bedside ultrasonography, and prognosis of hospitalized patients with acute heart failure. Systemic congestion is being notoriously underdetected in the acutely ill population with conventional methods like clinical examination, biomarkers, central venous pressure estimation and X-rays. However, congestion should be a key therapeutic target due to its deleterious effects to end organ function and subsequently patient prognosis. Doppler flow assessment of the abdominal veins is gaining popularity worldwide, as a valuable tool in estimating comprehensively congestion and giving a further insight into hemodynamics and patient management.

Key Words: Systemic congestion; Organ perfusion; Hemodynamics; Central venous pressure; Point of care ultrasound; Venous excess ultrasound

Core Tip: Venous excess ultrasound score incorporates the assessment of the inferior vena cava and the Doppler flow patterns of hepatic, portal and renal veins. It can provide valuable information about volume status, guide fluid management decisions in the acutely ill and obviate the deleterious effects of congestion to the peripheral organs.

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INTRODUCTION

In this observational study[1], the authors included 56 patients who were hospitalized for decompensated acute heart failure (AHF) and 17 controls without AHF. They calculated the portal vein pulsatility ratio (PVPR) by ultrasonography on admission and on discharge. On admission, PVPR was significantly higher in patients with AHF compared with controls and was decreased at discharge after guideline directed medical therapy. The authors suggest this PVPR amelioration might reflect venous congestion and HF condition, as there was noted concomitant improvement in clinical, laboratory (total bilirubin, brain natriuretic peptide) and echocardiographic [inferior vena cava (IVC) diameter, tricuspid regurgitation pressure gradient] indices. They also showed that patients with a higher PVRP at discharge had worse prognosis (cardiac death and HF hospitalization) at 1 year of follow up, implying that PVRP could be a novel prognostic marker for hospitalized patients with AHF.

THE ROLE OF VENOUS CONGESTION

In acutely ill patients, the complex interplay between cardiac dysfunction (especially right heart failure), vascular tone dysregulation, systemic inflammation and neurohormonal activation, often leads to venous congestion. In this setting, the superimposed acute kidney injury with subsequent sodium and fluid retention, aggravates considerably clinical management. Systemic congestion is the hallmark of AHF, leads to peripheral organ dysfunction (liver, kidney, lungs, intestines) and is associated with dismal prognosis of the critically ill patients[2,3]. As in the current paper[1], there is a considerable proportion of HF patients with residual congestion after a recent hospitalization. These patients have a high risk (up to 60%) for death or HF hospitalization at 12 months, especially if they have untreated congestion[4,5]. In the DOSE trial[6] only 15% of patients were free from congestion at 72 h of diuretic therapy, which rose to 42% in the recent ADVOR trial[7]. Congestion is often underdiagnosed and undertreated and should be a key therapeutic target in hospitalized patients with AHF. It seems that reduction in congestion is more important even than improvement in cardiac output, in order to prevent further renal failure or mortality[8-10].

TRADITIONAL BEDSIDE ASSESSMENT OF VENOUS CONGESTION IS PROBLEMATIC

The presence of classic clinical findings (peripheral edema, JVP, third heart sound and rales) is useful but has low sensitivity for the detection of ongoing congestion[11], and its absence does not exclude congestion. The same is true for chest X ray, as 20% of patients with AHF in the emergency department have normal radiological findings[12]. Estimation of IVC size and respiratory variations with ultrasonography, although it may help with fluid tolerance, is a poor marker for intravascular volume and fluid responsiveness[13]. IVC size and collapsibility are influenced by intrathoracic and intraabdominal pressures, by cardiac pathology (tricuspid regurgitation, pulmonary hypertension, severe diastolic dysfunction) and by obstructive pathology (tension pneumothorax, tamponade, pulmonary embolism), where it reflects more pressure than volume burden[14]. Central venous pressure (CVP), measured at the bedside through a central catheter, is a static marker of cardiac preload which does not specify in which part of the Starling curve the patient is functioning. Except from extreme values, CVP has failed to guide fluid management[15] and should be interpreted together with cardiac output, as dictated by the Guytonian physiology, in order to comprise both cardiac and venous return function of the circulatory system[16]. CVP is also influenced by thoracic, abdominal and pericardial pressures, which makes its interpretation difficult in the complex clinical setting[17].

VENOUS EXCESS ULTRASOUND SCORE

Doppler evaluation of the venous system help us estimate the downstream effects of elevated Right atrial pressure (RAP). Venous excess ultrasound (VExUS) protocol was introduced by Beaubien-Souligny *et al*[18] to quantify systemic congestion in a comprehensive manner and was validated in cardiac surgery patients. It comprises ultrasonographic estimation of IVC size and collapsibility, along with the Doppler flow patterns of the hepatic, portal and renal parenchymal veins. Depending on flow alterations, a score is derived (grade 0 = no congestion, grade 3 = severe congestion), which indicates the estimated degree of splanchnic congestion. VExUS score severity is strongly associated with elevated CVP levels[19] and has a good sensitivity and specificity to predict an elevated RAP[20]. It may predict AKI in various settings[21-23] and has been used to guide various interventions (diuretic therapy, ascites drainage, hemodialysis)[24]. A key advantage of these Doppler waveforms is that they are dynamic and allow monitoring the

Table 1 Summary of main limitations of sonographic splanchnic vein flow assessment

Inferior vena cava	Cylinder effect (misaligned diameter measurement in long axis)
	Abdominal aorta misinterpreted as inferior vena cava
	Presence of thrombus or obstruction
	Normally distended in young individuals and athletes due to increased venus reserve
	Distended in mechanically ventilated patients (PEEP, mean airway pressure)
	Diameter influenced by respiratory effort in spontaneous breathing patients
	Collapsed in abdominal hypertension
	Distended in tricuspid regurgitation, pulmonary hypertension, severe diastolic dysfunction, cardiac tamponade, tension pneumothorax, pulmonary embolism
Hepatic vein	Difficult interpretation in presence of arrhythmias and pacing (ECG tracing imperative)
	In case of tricuspid regurgitation, does not alone reflect venous congestion
	Decreased venous phasicity in parenchymal liver disease (cirrhosis, occlusive disease)
Portal vein	Enhanced pulsatility in thin healthy individuals
	Unreliable in parenchymal liver disease (cirrhosis, severe steatosis, occlusive disease, arteriovenous fistulas)
	Unreliable in severe portal hypertension (stagnant/retrograde flow, low velocities)
Renal vein	Technically the most challenging
	Influence of body habitus and mechanical ventilation
	Results differ between cortical versus hilar vessel interrogation
	Unknown reliability in renal parenchymal diseases and kidney transplantation

response to decongestive therapy in real time[25]. In patients with AHF, VExUS was shown to predict AKI and have prognostic implications[23,26], as in the current paper by Kuwahara *et al*[1].

In particular portal vein Doppler, which was used by the authors of this paper, is less technically demanding and is affected only by parenchymal hepatic pathology. In a recent trial, the author's conclusions were similar as Kuwahara *et al* [1], portal vein pulsatility ≥ 50% was highly prevalent in a population of patients hospitalized with AHF and was most closely associated with RV dysfunction. After decongestive therapy, patients with abnormal PVPR at discharge had poorer long-term clinical outcomes when compared with those with normal PVPR pattern[27].

The term extended venous excess ultrasound score (E-VExUS) or extended VExUS has been proposed to include Doppler interrogation of additional veins such as internal jugular, splenic, and femoral veins[28], or even sonographic estimation of the intestinal wall[29].

PITFALLS OF VEXUS

Doppler evaluation of venous congestion comes with various limitations. It is operator dependent and this may affect image acquisition and interpretation. Ultrasound penetration and Doppler sensitivity depends also on the probe and ultrasound machine available. Severe obesity, supine position, drainage tubes, surgical wounds, pain and agitation, may hamper ultrasound examination of visceral venous blood flow in the acute setting.

IVC is influenced by thoracic, abdominal and pericardial pressures and particularly mechanical ventilation positive pressures. Hepatic vein flow may be altered by arrhythmias, pacing, tricuspid regurgitation and pulmonary hypertension, even without fluid overload. Renal vein flow is the most challenging to obtain and is affected in renal parenchymal disease[30]. Portal vein flow is more advantageous, as it remains less affected by arrhythmias, pacing, tricuspid regurgitation and pulmonary hypertension; however, it may be altered in patients with cirrhosis and portal hypertension and should be cautiously considered. In Table 1, we summarize the main limitations regarding ultrasound evaluation of splanchnic venous flow[13,18,31].

VExUS may quantify congestion but may not indicate the reason of congestion, nor differentiate between pressure or volume overload. This is why the physician should interpret Doppler findings in the clinical context and combine clinical assessment with information from laboratory tests, echocardiography or even right heart catheterization[25,28] in more challenging cases.

CONCLUSION

Congestion is often underdiagnosed and undertreated in the acutely ill patients, leading to excessive morbidity and mortality. Although congestion relieve should be a key therapeutic target, traditional bedside assessment is problematic. Ultrasound interrogation of the splanchnic veins emerge as an effective tool to quantify congestion and estimate effects of right atrial pressure to peripheral organs. It should be assessed along with other echocardiographic, hemodynamic and clinical indices to determine how much congestion contributes to organ dysfunction. Additional research is needed for splanchnic vein flow assessment as a valid diagnostic and prognostic tool to guide therapeutic decisions and improve prognosis.

FOOTNOTES

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