

# Congestion phenotypes in heart failure: The emerging role of POCUS

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## Dear editors:

I was very interested to read the article titled “Clinical ultrasound (POCUS) in patients with heart failure:

From diagnosis to discharge” (1) recently published in *Acta Médica Colombiana*, in which the authors provide an excellent review of the role of Point of Care UltraSound (POCUS) in the diagnosis of heart failure (HF) and the measurement of congestion in this scenario. However, I believe it is pertinent to share additional information to strengthen the importance of this tool in congestion phenotyping.

In HF, congestion is defined as the accumulation of fluid in the intravascular and extravascular compartments due to increased cardiac filling pressures (2). Congestion is not synonymous with volume overload (3). Although congestion has traditionally been considered a simple and uniform state of volume overload, today’s understanding emphasizes its complexity, differentiating between intravascular and interstitial or tissue congestion (4).

A distinctive trait of intravascular congestion is the rapid rise in pulmonary and cardiac filling pressures (4). Vascular redistribution is a key concept that proposes that increased venous tone induced by sympathetic activation following a triggering event can mobilize fluid from the splanchnic venous reservoir toward systemic circulation, elevating intravascular hydrostatic pressure without an actual increase in body water (3, 4).

Conversely, volume overload is a slower, more gradual phenomenon that results from sodium and water avidity in the renal tubule and an imbalance between hydrostatic and oncotic pressures in the intravascular and interstitial compartments, which, in turn, is mediated by neurohormonal activation and cardiorenal dysfunction (3, 4). As extracellular fluid volume expands, the resulting venous pressure elevation interrupts the Starling forces, favoring net capillary filtration. Once the lymphatic system’s ability to drain excess fluid is exceeded, it begins to accumulate in the interstitial space (4).

Although most patients with acute HF have a combination of both types of congestion, identifying the predominant phenotype could determine the most appropriate treatment strategy (3). It has been suggested that patients with intravascular congestion could benefit more from interventions aimed at venous tone modulation than from aggressive decongestive strategies. On the other hand, those with tissue congestion or volume overload would be more appropriate candidates for intensive diuretic treatment, as long as vascular filling is maintained. However, more studies are needed to support these assumptions (2).

A phenotype with predominant tissue congestion is evidenced in clinical findings like pulmonary crackles, pleural effusion, ascites and lower extremity edema; elevated biomarkers like CA 125, bioactive adrenomedullin and CD146; and through POCUS showing the presence of B lines and/or pleural effusion on the lung study. Meanwhile, the phenotypic profile of intravascular congestion is evidenced by clinical findings like orthopnea, bendopnea, jugular distention and a third heart sound; biomarkers like BNP or NT-proBNP, and sST2; and POCUS findings like a vena caval diameter  $\geq 21$  mm, VExUS score  $> 1$  or extended VExUS protocol data suggesting venous congestion (2, 3, 5).

Accurate congestion assessment continues to be a challenge in daily clinical practice. A comprehensive approach with multiple parameters including clinical findings, circulating biomarkers (many still under study) and imaging techniques, in which POCUS plays a leading role, is essential for characterizing the predominant congestion phenotype and determining the best treatment strategy for patients.

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## RESPONSE

We thank the author for his letter in response to our review article on POCUS for handling patients with acute heart failure. It is undoubtedly an open field in which research is essential to improve understanding of the potential role and weight of this intervention in treating this type of disease.

The letter's author is quite right in presenting the congestion phenotypes, which are undoubtedly important for decision making in internal medicine services. Using tissue markers like CA125 and the VExUS score provides more objectivity in evaluating congestion.

As we mentioned in our article, the prognostic impact of identifying subclinical congestion is crucial for determining whether to continue decongestion or intensify it as an inpatient. Unfortunately, we still do not have compelling evidence that an approach using POCUS or the phenotypes presented here has a positive impact on the treatment of these patients. There are two ongoing clinical trials which we hope will provide more clarity in “the role of POCUS-guided treatment in this clinical scenario.”

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