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REVIEW

Involvement of systemic venous congestion in heart failure☆

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KEYWORDS
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Abstract Systemic venous congestion has gained significant importance in the interpretation of the pathophysiology of acute heart failure, especially in the development of renal function impairment during exacerbations.

In this study, we review the concept, clinical characterization and identification of venous congestion. We update current knowledge on its importance in the pathophysiology of acute heart failure and its involvement in the prognosis. We pay special attention to the relationship between abdominal congestion, the pulmonary interstitium as filtering membrane, inflammatory phenomena and renal function impairment in acute heart failure. Lastly, we review decongestion as a new therapeutic objective and the measures available for its assessment.

PALABRAS CLAVE
Insuficiencia cardíaca;
Congestión venosa sistémica;
Inflamación;
Deterioro de la función renal

Implicación de la congestión venosa sistémica en la insuficiencia cardíaca

Resumen La congestión venosa sistémica ha cobrado mucha importancia en la interpretación de la fisiopatología de la insuficiencia cardíaca aguda, y muy especialmente en el desarrollo del deterioro de la función renal durante las agudizaciones.

En el presente trabajo se revisa el concepto, la caracterización clínica y la identificación de la congestión venosa. Se actualiza el conocimiento sobre su importancia en la fisiopatología de la insuficiencia cardíaca aguda y su implicación en el pronóstico. Se presta especial atención a la relación entre la congestión abdominal, el intersticio pulmonar como membrana filtrante, los

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fenómenos inflamatorios y el deterioro de la función renal en la insuficiencia cardíaca aguda. Por último, se revisa la descongestión como un novedoso objetivo terapéutico y los medios disponibles para su evaluación.

Concept of venous congestion

Taking into account strictly hemodynamic criteria, systemic venous congestion is defined as an increase in pulmonary wedge pressure >12 mm Hg or an increase in left ventricular diastolic pressure >16 mm Hg, measured through catheterization of the right-sided heart chambers.[1] However, the definition is of little practical use due to the methods' invasiveness. Systemic venous congestion in heart failure (HF) results from the increase in intravascular volume caused by an increase in ventricular diastolic pressure. Moreover, the increase in ventricular end-diastolic pressure is the direct result of 2 conditions: direct myocardial damage (as in ischemic heart disease or cardiomyopathies) and the increase in preload associated with prior ventricular dysfunction, which causes an increase in central venous pressure (CVP). HF is produces in both conditions, and, depending on the left ventricular ejection fraction (LVEF), we talk of HF with preserved ejection fraction for LVEF >50%; intermediate for 40–49% and reduced if LVEF <40%.[2]

Identifying congestion in acute HF (AHF) is of primary importance. Up to 66% of patients in the Acute Decompensated Heart Failure National Registry (ADHERE)[3] presented some degree of systemic congestion at admission.

Symptomatology of congestion

Classically, congestion is detected *via* a physical examination revealing the presence of the following classic signs: jugular venous distention, hepatomegaly, superficial collateral venous circulation, ascites and edema in dependent regions. Pulmonary congestion is defined mainly by the increase in respiratory effort or rate and the auscultation of end-inspiratory crackles.[4]

The signs of congestion and the degree of skin perfusion configure the 4 clinical phenotypes of patients with AHF (Fig. 1), with clear connotations for the prognosis and treatment.[5] Patients who present more signs of congestion during the examination have higher wedge pressure in the pulmonary artery and a poorer prognosis.[3–7] However, despite its usefulness, the physical examination has little sensitivity for detecting or ruling out the presence of congestion, especially the subclinical grades.[4] Quantifying or measuring the degree of congestion is difficult to determine using the physical examination.

In recent years and thanks to advances in technology, more objective complementary examinations have been developed to detect and measure venous

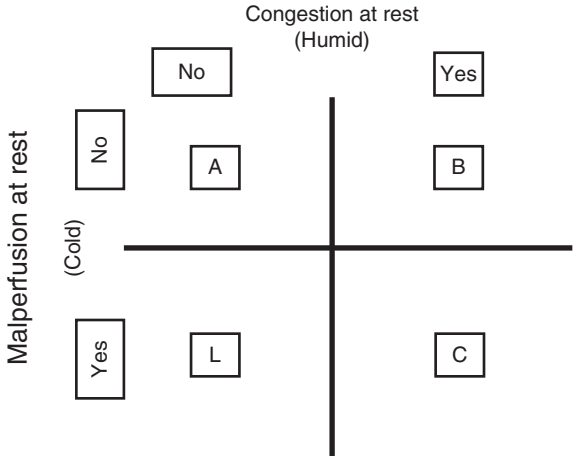

Figure 1 Description of the 4 patient phenotypes according to the degree of congestion and the degree of skin perfusion. *Source*: Adapted from Stevenson.[5]

congestion in HF, such as impedance audiometry,[8] pulmonary ultrasonography[9,10] and the measurement of the diameter and collapse of the inferior vena cava (IVC)[11] (Fig. 2). Thus, systemic congestion is considered when the ultrasound diameter of the IVC is >21 mm in inspiration, and the collapse is <50% of the baseline diameter.[11] Patients with systemic congestion estimated by this method have a poorer prognosis at 6 months of discharge from an episode of AHF.[12–14] Another ultrasound technique that assesses pulmonary congestion is pulmonary ultrasonography to visualize the Kerley B lines (sign of the comet tail).[15] This new approach to the ultrasound examination of the lungs has high sensitivity (94%) and specificity (92%) for detecting interstitial edema and helps differentiate between cardiogenic and respiratory dyspnea. The approach also helps quantify the intensity of the congestion, because there is a correlation between the number of artifacts in the various lung fields and NT-proBNP concentrations and the echocardiographic quotient E/e' (of vital importance for assessing HF with preserved LVEF).[9,10,16] Lastly, pulmonary impedance audiometry, built into pacemaker technology, has helped reduce the rate of readmissions of patients with chronic HF, as shown by the IMPEDANCE-HF study.[17]

Despite these techniques, determining a patient's congestive state remains difficult. A study published in 2007 with 134 patients hospitalized for HF (New York Heart Association functional class III) who were previously telemonitored in their homes through vital sign monitoring and