

Haemodynamic Congestion in Chronic Heart Failure Patients

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Abstract

Fluid balance is critical in heart failure patients. Fifty percent of heart failure patients are re-hospitalised within six months of discharge from hospital despite best available guidelines. Chronic heart failure patients are in compensated state due to several adaptive changes in the body and physical exam may not correlate to pathophysiological changes occurring within the body leading to haemodynamic congestion. Physical exam and biomarkers have been less reliable to predict development of acute decompensation. Ultrasound evaluation of inferior vena cava diameter and inferior vena cava collapsibility index (IVCCI) has played a remarkable role in estimating and grading hemodynamic congestion in patients with heart failure. We believe echocardiographic assessment of inferior vena cava could predict future risk of HF decompensation and should be used as routine along with standard assessment in chronic heart failure patients.

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Abbreviations: HF: Heart Failure; IVC: Inferior vena cava; IVCCI: Inferior Vena Cava Collapsibility Index

Background

One of the greatest challenges in the management of patients with chronic heart failure is the monitoring and predicting acute worsening leading to hospitalization. Despite best clinical practice, including management within a heart failure assessment unit and close follow-up with the help of virtual consults via telephone with HF patient, the rate of re-hospitalisation is approximately 50% among heart failure patients over six months [1]. The most significant risk factor for this unacceptably high event rate, is failure to understand and acknowledge the dissonance between clinical signs of congestion and hemodynamic parameters [2].

When patients with chronic heart failure are in a compensated state, there is balance between intravascular and extravascular hydrostatic pressure, oncotic pressure, and lymphatic drainage. Due to neurohormonal activation and other mechanisms, intravascular hydrostatic pressure can exceed extravascular hydrostatic pressure to a level that cannot be compensated for by the lymphatic system. When this occurs, patients develop severe hypervolemia with decompensated heart failure. Clinical congestion occurs when the elevation in the left atrial filling pressure. It presents itself via signs and symptoms such as dyspnoea, rales, oedema, and weight gain. The increase of left atrial filling pressure without clinical congestion has been termed as hemodynamic congestion. It has been shown to precede clinical congestion by days or even weeks [3].

The rate at which clinical congestion occurs is connected to the rapidity of the rise, duration, and size of the left atrial pressure change. Thus, clinical congestion is only the tip of the iceberg of the hemodynamic derangements that herald the condition worsening [4-10]. The clinical methods available to spot the beginning of hemodynamic congestion and also the shift from the compensated to the decompensated state, both rely on physical examination and daily weights. Furthermore, serial biomarker measurements are used, such as B-type natriuretic peptide. However, these techniques have been shown to have an insufficient level of sensitivity and specificity to reliably predict the development of acute decompensation [4]. Hemodynamic congestion is linked to the development of HF by activating neurohormones and by triggering subendocardial ischaemia. This in turn causes myocardial necrosis, apoptosis and/or additional mitral insufficiency by its impacts on LV geometry [5].

Ultrasound evaluation of inferior vena cava size (IVCD) and inferior vena cava collapsibility index (IVCCI) has played a remarkable role in estimating and grading hemodynamic congestion in patients with heart failure [6]. There have been many large, well conducted studies about using this method to identify any

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changes in respiratory pattern of IVC indicating fluid imbalance (that hemodynamic congestion and intravascular depletion). Based on well-established knowledge, wide respiratory changes of IVCCI would certainly show that intravascular deficiency is likely. Whereas, reduced values of this specification would suggest high chance of hemodynamic congestion [7,8,9]. In chronic heart failure, the human body undergoes several adaptive pathophysiological changes, such as increase in alveolar capillary membrane thickness, increased lymphatic drainage and/ pulmonary hypertension [11], so that the body can tolerate

fluid imbalance with no clinical signs of congestion, until a trigger provokes acute decompensation of chronic heart failure.

Echocardiographic assessment of inferior vena cava could predict future risk of HF decompensation in a clinically euvolemic patients. We propose that routine physical exam should include echocardiographic assessment of IVC along with standard assessment of chronic heart failure patients. These patients with haemodynamic congestion could be followed more closely and in order to facilitate diuretic therapy optimisation.

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