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Identification and Quantification of Congestion in Heart Failure: a Work in Progress

Dyspnoea, fatigue, and peripheral oedema are the key symptoms and signs that characterize heart failure (HF). They are usually caused by water and salt retention or, to put it simply, congestion.¹ In routine clinical practice, assessment of signs and symptoms in patients with heart failure is often inaccurate and subjective as it requires considerable clinical skills and is based on the interpretation of reported patients' daily activities and medical history. Identification and quantification of haemodynamic, cardiac, or peripheral congestion is even more difficult. To overcome these limitations, more objective diagnostic tools have been developed and intensively investigated in the past couple of decades to improve management and outcomes of patients with heart failure. The Kansas City Cardiomyopathy Questionnaire (KCCQ) is a self-administered instrument that quantifies the impact of heart failure on patients' symptoms and quality of life. Findings from previous research suggest that KCCQ scores correlate with NYHA functional class, track changes in health-related quality of life associated with introduction of disease modifying heart failure therapies and are associated with prognosis.² Ultrasound can be used to assess cardiac function and to examine the lungs at the same time. By scanning de-aerated lung segments, vertical artefacts originating from the pleura (called B-lines) are frequently seen traversing the ultrasound screen. Lung ultrasound has been shown to be more sensitive for detecting pulmonary congestion than chest x-ray and physical examination in patients with suspected or overt heart failure.^{3,4} Assessment of pulmonary congestion by ultrasound requires a short period of training⁵, can be performed in a few minutes⁶ by a variety of health care providers, including sonographers and specialist heart failure nurses, and can be interpreted by physicians in real time. Ultrasound evidence of pulmonary congestion identifies patients with heart failure with higher natriuretic peptides and poorer outcomes, even when symptoms and signs are only mild or absent. Preliminary evidence suggests that treatment guided by lung ultrasound might improve heart failure management. While there is no current recommendation from U.S.-based heart failure societies, the European Society of Cardiology-HF guidelines recommend that lung ultrasound might be considered to assist with the detection of extravascular lung fluid during diagnostic workup of new onset acute heart failure.⁷

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In this article, Lee, Platz, and colleagues prospectively investigated the association between KCCQ-Total Symptom Score (TSS) and markers of congestion as assessed by lung ultrasound, chest x-ray, and physical examination among patients hospitalised with acute heart failure. Overall, they did not find a significant association between KCCQ-TSS and imaging markers of pulmonary congestion. However, they did find that a lower KCCQ-TSS was associated with a greater prevalence of peripheral oedema and worse NYHA class. There are several potential explanations for these discordant findings. Clinical symptoms and signs are late manifestations of congestion and are neither sensitive nor specific for heart failure. Shortness of breath could be confounded by old age or comorbidities such as anemia, chronic obstructive lung

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disease, anxiety or depression that frequently co-exist with heart failure.^{8,9} More importantly perhaps, fatigue, depression, and overall health perception uniquely contribute to dyspnoea¹⁰, and increase risk of a premature death in patients with heart failure.^{11,12}

Peripheral oedema is common in the elderly, particularly among those treated with dihydropyridine calcium antagonists (>20% in the current study). It should be also noted that many of those with long-standing heart failure are less likely to have dyspnoea at rest and pulmonary congestion, but more likely to have right ventricular dysfunction, peripheral edema, and worse prognosis¹³. Many obese patients may develop dysphoea even on mild exertion and peripheral oedema due to poor mobility. but do not necessarily have heart failure or worsening congestion as their precipitating symptom. Interestingly, higher body mass index (BMI) was associated with poorer KCCQ-TSS in this study; however, a higher BMI is also associated with a lower number of B-lines and a lower risk of death in patients with heart failure, suggesting that quantifying congestion of heart failure is difficult in obese patients with dyspnoea, particularly when left ventricular ejection fraction is not substantially impaired. Moreover, the number of B-lines on lung ultrasound might be increased by presence of lung fibrosis or infection, conditions that frequently complicate an episode of acute HF or might confound its diagnosis; therefore, in this setting, an increasing inferior vena cava (IVC) diameter might be a better marker of congestion than a high number of B-lines¹⁴. However, in this study, association between KCCQ-TSS and IVC diameter was not significant, potentially reflecting difficulties in visualizing correctly the IVC diameter and incurring in measurement errors when echoing patients who are acutely unwell. Unfortunately, novel ultrasound methods², such as jugular vein diameter (JVD) ratio (the ratio between the maximum diameter during Valsalva to that at rest), hepatic veins, portal vein and renal venous Doppler flow patterns, that can also guantify congestion in other peripheral organs and vessels, were not evaluated in this study.

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Acute dyspnoea might be a life-threatening condition and when heart failure is diagnosed, patients usually require aggressive doses of diuretics and vasodilators for a rapid relief of symptoms. It is plausible that the use of these therapies alleviated symptoms and pulmonary congestion quickly, with a slower effect on intravascular and peripheral edema and, indeed, KCCQ-TSS. By design, KCCQ scores reflect a recall period of two weeks prior to their administration, therefore, a likely explanation for the lack of associations between imaging markers of pulmonary congestion and KCCQ-TSS is that the this instrument might not capture the acute effects of medications that occur during the initial phases of a heart failure hospitalisation. If this was true, one would expect that a KCCQ-TSS might also fail to identify the initial stages of worsening congestion that eventually result in a heart failure hospitalisation. How to best monitor and identify worsening congestion at an early stage in patients with heart failure remains a highly relevant topic in contemporary research.

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