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Short communication

A plea for an early ultrasound-clinical integrated approach in patients with acute heart failure. A proactive comment on the ESC Guidelines on Heart Failure 2016

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ABSTRACT

Background: The European Association of Cardiology (ESC) Guidelines on the diagnosis and treatment of acute heart failure (AHF) indicate prompt therapy initiation and performance of relevant investigations as paramount. Specifically, echocardiography prior to treatment is advocated only with hemodynamic instability, and the evaluation of clinical signs of peripheral perfusion and congestion is suggested as guidance for early interventions. Given the growing body of evidence on the diagnostic/monitoring capabilities of bedside ultrasound (including focused cardiac ultrasound, comprehensive echocardiography, lung ultrasound), we discuss the potential benefit of an integrated clinical/ultrasound approach at the very early stages of acute heart failure.
Methods and Results: We proposed a narrative review of the current evidence on the clinical-ultrasound integrated approach to AHF, with special emphasis on the components of the early diagnostic-therapeutic workup where cardiac, inferior vena cava and lung ultrasound showed high diagnostic accuracy and the capability of substantially changing an exclusively clinically-oriented patient management. A proactive comment to the ESC guidelines is made, suggesting an integration of clinical and biochemical assessment, as defined by guidelines, with combined bedside ultrasound on may help in the definition of AHF pathophysiology and treatment.
Conclusion: A multi-organ integrated clinical-ultrasound approach should be advocated as part of the clinical-diagnostic workup at AHF very early phase. Whenever competence and technology available, bedside ultrasound, along with clinical and biochemical assessment, should target AHF profiling, identify the cause of AHF, and subsequently aid disease course and response to treatment monitoring.

Amongst the novelties introduced by the recently published Guidelines on the diagnosis and treatment of acute heart failure (AHF)[1], the importance of "time to therapy" approach is addressed as crucial in the diagnostic-therapeutic strategy: prompt initiation of appropriate therapy and deployment of relevant investigations is acknowledged as paramount.

Although the role of echocardiography is indicated as playing a pivotal role as diagnostic/monitoring tool in AHF in the guidelines, its performance prior to treatment initiation is advocated only in the setting of hemodynamic instability [1,2].

We believe that recent pervasive, cross-specialty, use of bedside multi-organ ultrasound in critical care and emergency department, supported by a growing body of evidence on its accuracy, accessibility, feasibility and cost-effectiveness [3–6], integrated with clinical and biochemical examination could represent a step forward in the management of the early phase of AHF. A multi-step approach using focused cardiac ultrasound (FoCUS), lung ultrasound (LUS) and comprehensive echocardiography, along with standard clinical and biochemical screening, in the early phase of patients admission may have a great potential impact on AHF diagnostic/therapeutic pathway.

FoCUS provides an initial essential screening regarding cardiac systolic function (normal vs markedly impaired), heart chambers

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(normal vs significantly abnormal) and gross valve morphology, and screening for severe hypovolemia [3,7]. It can hasten diagnosis, guide initial therapy or trigger emergency comprehensive echocardiography. FoCUS can narrow substantially the number of viable AHF mechanisms in differential diagnosis, and monitor the effects of early interventions (for example, by detecting changes in left and right systolic function upon initiation of pharmacological support, or in central volemia by following ventricular and inferior vena cava sizes over time) [3].

The role of comprehensive echocardiography is to further detail the FoCUS findings in the accurate assessment of cardiac anatomy and pathophysiology including regional wall motion abnormalities, diastolic function, valular pathology and left atrial pressure (LAP). As the correlation of echo with left ventricular filling pressure and natriuretic peptides is well established [6,8–11], less studies have been published in acute setting, however good correlation have been demonstrated [12,13]. Although some limitations exist, such as atrial arrhythmias and sinus tachicardia which are frequent in patients with AHF, the integration of different parameters to assess Diastolic function and LAP are necessary [6,14] (i.e. mitral inflow integrated with Tissue Doppler imaging , pulmonary venous flow and LA volume index - Table 1). The LAP assessment with Doppler echocardiography in patients with chronic HF (thus expected to be chronically elevated) may have limited value *per se*, but the LAP monitoring during and following therapy may give a precise clue of the HF underlying cause and may help in therapy titration.

Echocardiography estimation of stroke volume and cardiac output by means of left ventricular outflow tract (LVOT) PW Doppler flow sampling is a well-established technique, that allows reliable tracking of stroke volume changes [15].

A comprehensive echocardiographic exam using two-dimensional and Doppler techniques provides insight into the patient's volume status. Distension and small magnitude of inspiratory variations of inferior vena cava (IVC) suggest (instead of accurately diagnose) systemic venous congestion, although common pitfalls must always be taken into account [17]. Conversely, small size of IVC identifies severe hypovolemia, and its pronounced inspiratory size reduction predicts volume responsiveness with high specificity [3]. Moreover, LVOT VTI variations upon a passive leg raising maneuver [16] or a fluid challenge, are strong predictors of fluid responsiveness, allowing careful fluid titration when two-dimension-based echocardiographic indices may be misleading (such as with RV failure, chronic RV or LV failure, marked inspiratory efforts) [17].

Lung ultrasound (LUS) has been extensively studied in the past 15 years, and has been validated as an accurate non-invasive diagnostic tool for extra-vascular lung water (EVLW). It is now proposed as new standard for pulmonary congestion diagnosis and monitoring in patients with AHF [4,18] by looking at the presence and numbers of B-lines, indicating the loss of aeration and increase in density of the lung periphery. The number of B-lines has shown an excellent correlation with the amount of EVLW , it is a sensitive marker of changes in pulmonary congestion and it has a prognostic value in cardiology patients [19]. It is important to highlight that B-lines indicates increased EVLW (pulmonary congestion) but not necessarily a condition of high left ventricular filling pressure and high wedge pressure (pulmonary venous congestion) [18]. Indeed, a patient may show variable degrees of lung congestion for any given level of pulmonary artery wedge pressure, depending on the duration of his/her heart failure history, the speed of changes in pulmonary pressure, the characteristics of the alveolar-capillary membrane, oncotic pressure, and lymphatic drainage capacity [18]. LUS is remarkably accurate in identifying the cardiogenic origin of dyspnoea and in differentiating AHF syndromes from non-cardiac causes of acute dyspnoea even at a preclinical stage having high sensitivity and excellent specificity (94% and 92%, respectively) [20,21]. In a large multicenter study performed in the emergency department, a LUS-clinical integrated approach to dyspnoea proved to be more accurate than the traditional clinical work-up, chest X-ray, and natriuretic peptides [22].

B-lines may be found also in conditions as ARDS and fibrosis, where anyhow other pathognomonic LUS features allow to differentiate [4].

The integration of LUS with the available clinical information and with other diagnostic tools may greatly help a quick and correct diagnostic orientation. Interestingly, an integrated approach to dyspnoea combining FoCUS, LUS and IVC ultrasound shows higher accuracy in identifying AHF as a cause of respiratory failure, when compared to each method alone, with high sensitivity (94.3%), specificity (91.9%), negative predictive value (91.1%), and positive predictive value (94.3%) [23].

The last European Society of Intensive Care Medicine (ESICM)/Acute Cardiovascular Care (ACCV)/European Society of Cardiology (ESC) acute heart failure recommendations, highlight how therapeutic intervention in the first 60 min should be tailored to a patient's actual underlying pathophysiology, and not be driven by peremptory assumptions[2]. The limitations of the clinical exam in assessing the underpinning pathophysiology and the factors key to the management of respiratory failure/AHF (cardiac function, volume status, extravascular lung water) are well documented [14,24]. Postponing a more detailed understanding of these variables after the institution of treatment carries the risk of being detrimental to organ function and overall outcome. This may be particularly true in cases of AHF not associated with volume overload. It is well recognized that the accuracy of most clinicians in estimating and interpreting intravascular volume status seems to be suboptimal [24] and errors in estimating intravascular status may easily lead to incorrect treatment decisions [6,24]. In AHF pathophysiology volume loading conditions play a key role. Recent trials have demonstrated that the admission for AHF decompensation is preceded by an increase of right filling pressure rather than an increase in body weight [25–27]. The redistribution of blood volume from the splanchnic venous reservoirs in response to sympathetic stimulus lead

Table 1
Cut off and limitations of the main ultrasound parameters for the estimation of filling pressure[9] and systemic congestion.

Variable	Cut-off	Limitations
E/A ratio	>2	Sinus tachycardia or atrial arrhythmia
Deceleration time	<160 ms (in HFrEF) or normal (HFpEF)	Sinus tachycardia or atrial arrhythmia
E/e' average ratio	>14	Heavy annular calcification, mitral valve and pericardial disease; patients with CAD and regional dysfunction at the sampled segments.
LA maximum volume index	>34 mL/m^2	Severe bradycardia, high-output states, heart transplants with biatrial technique, atrial flutter/fibrillation, significant mitral valve disease, despite normal LV diastolic function.
TR jet	>2.8 m/s	severe TR and low systolic RV-RA pressure gradient; primary pulmonary hypertension
Pulmonary vein S/D ration	<1	Atrial arrhythmia, mitral valve disease and HCM.
Inferior vena cava	Dilated; <50% variation during respiratory cycle	Elevated pulmonary vascular resistance and intrathoracic pressure; respiratory effort; right side heart failure; high abdominal pressure; tamponade [10]
B-lines	Multiple/confluent	Sign of increased EVLW, not necessarily high LAP [11]

If more than half or all of the echocardiographic variables meet the cutoff values, then LAP is elevated and grade II diastolic dysfunction is present [9].
HFrEF: Heart failure with reduced ejection fraction; HFpEF: heart failure with preserved ejection fraction; LA: left atrium; TR jet: tricuspid regurgitation jet; S/D ratio: ratio between systolic and diastolic wave at pulmonary veins Doppler; RV-RA: right ventricle-right atrium; HCM; hypertensive cardiomyopathy; EVLW: Extravascular lung water; LAP: left atrial pressure.