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Non-invasive imaging in acute decompensated heart failure with preserved ejection fraction

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Non-invasive imaging plays an increasingly important role in emergency medicine, given the trend towards smaller, portable ultrasound devices, the integration of ultrasound imaging across diverse medical disciplines, and the growing evidence supporting its clinical benefits for the patient. Heart failure with preserved ejection fraction (HFpEF) provides a compelling illustration of the impactful role that imaging plays in distinguishing diverse clinical presentations of heart failure with numerous associated comorbidities, including pulmonary, renal, or hepatic diseases. While a preserved left ventricular ejection fraction might misguide the clinician away from diagnosing cardiac disease, there are several clues provided by cardiac, vascular, and lung ultrasonography, as well as other imaging modalities, to rapidly identify (decompensated) HFpEF. Congestion remains the primary reason why patients with heart failure (irrespective of ejection fraction) seek emergency care. Furthermore, comprehensive phenotyping is becoming increasingly important, considering the development of targeted treatments for conditions exhibiting HFpEF physiology, such as cardiac amyloidosis. Timely recognition in such cases has lasting implications for long-term outcomes.

Introduction

Heart failure (HF) continues to be the foremost cause of hospitalization for individuals over 65 years of age. The proportion of HF with preserved ejection fraction (EF) (HFpEF) has now surpassed 50%.[1] Heart failure with preserved EF is characterized by advanced age and multiple comorbidities, contributing to diagnostic challenges in emergency settings. The manifestation of decompensated HFpEF varies from exertional dyspnoea with or without mild bimalleolar oedema to life-threatening scenarios such as frank pulmonary oedema. Across this spectrum, timely commitment to a precise diagnostic course remains crucial, particularly considering advancements in personalized treatment. European and American societies promptly advocate assessing the likelihood of HFpEF through scoring systems, respectively using the HFA-PEF and the H_2FPEF score, with cardiac ultrasonography (US) occupying a central role in both scores.[2,3] Lately, easy-to-use, portable ultrasound devices have emerged as invaluable time-saving instruments within the emergency department, serving as an extension of the conventional physical examination.[4] Further advancements in automated analyses utilizing artificial intelligence will facilitate the implementation of more comprehensive bedside analysis, although integrating this into the emergency setting remains challenging due to time pressure and often difficult imaging conditions.[5,6] Findings from US must be integrated into the clinical assessment to devise diagnostic and therapeutic strategies. This review concentrates on the non-invasive approach to a patient with suspected decompensated HFpEF, with particular emphasis on lung, vascular, and cardiac US.

Clinical presentation

The diagnosis of HF requires the presence of symptoms and/or signs of HF and objective evidence of cardiac dysfunction.[7] Relying solely on symptoms and signs for diagnosing HF is hindered by their limited sensitivity and specificity. Recognizing that not all cases of acute shortness of breath or oedema necessarily indicate HF is crucial. Other non-cardiac conditions, such as kidney and liver failure, anaemia, and lung disease, may mimic symptoms and are characteristic comorbidities in elderly HFpEF patients.[1] Congestion continues to be the foremost cause prompting patients with HF (regardless of EF) to seek emergency care.[8] This may vary from acute volume redistribution (e.g. flash lung

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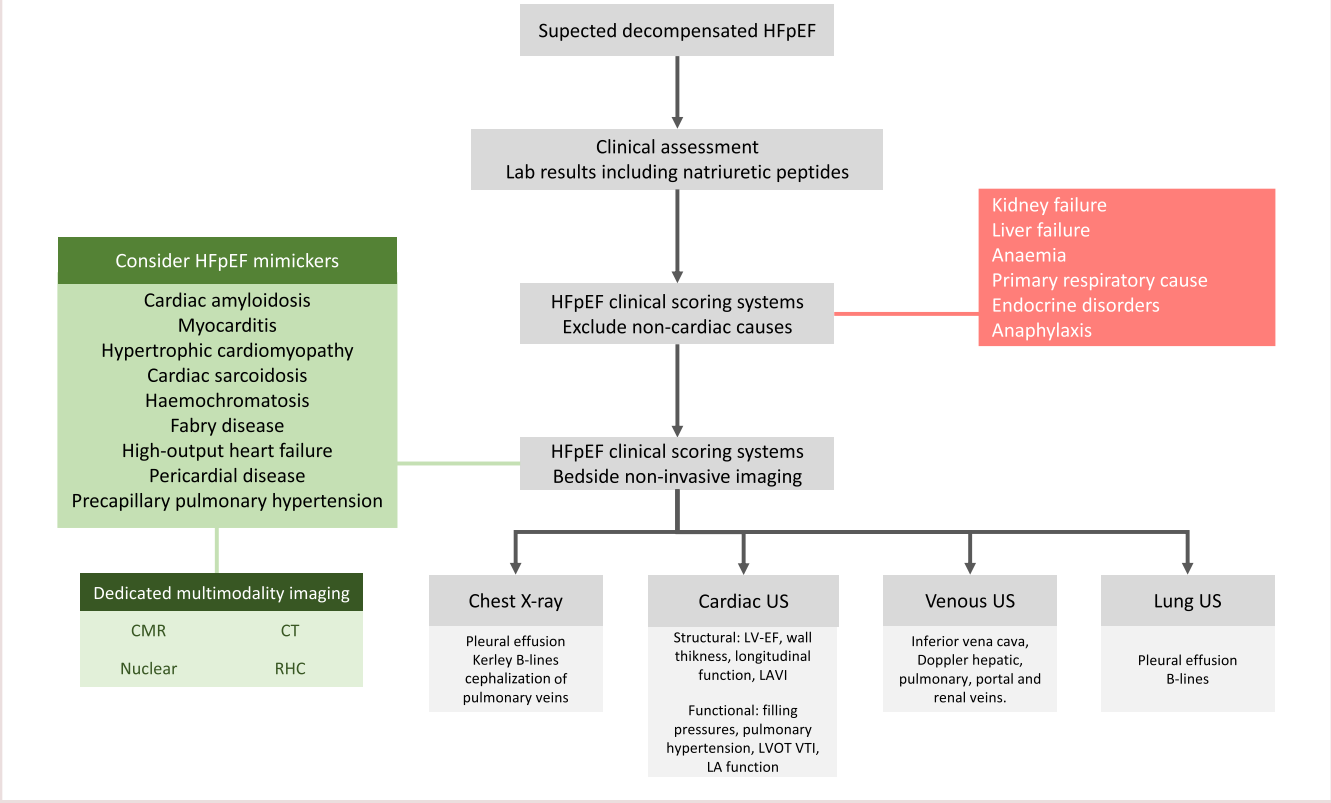

Figure 1 Proposed flowchart to approach patients with suspected decompensated heart failure with preserved ejection fraction (HFpEF). CMR, cardiac magnetic resonance; CT, computed tomography; LVEF, cardiac left ventricular ejection fraction; LAVI, left atrial volume index; LVOT VTI, left ventricular outflow tract velocity–time integral; RHC, right heart catheterization; US, ultrasound.

oedema) to the more gradual accumulation of fluid overload in subacute scenarios.[8] Imaging may aid in identifying congestion and clarifying its underlying cause and may hold particular value in cases where uncertainty exists. Adequate and rapid diagnosis of congestion significantly reduces the 'door to diuretics' time, while the aetiology also determines the short- and long-term management.[7,8] However, the significance of a medical history and clinical examination, including an electrocardiogram, remains undiminished.[7] *Figure 1* depicts a proposed flowchart to approach patients with suspected decompensated HFpEF.

Cardiac ultrasound

Assessment of structural heart disease

Focused cardiac US in life-threatening situations is primarily directed towards identifying specific conditions such as tamponade, interventricular septal flattening, acute valvular pathology, and/or severely impaired left ventricular (LV) systolic function. However, HFpEF may be easily overlooked in this context, and additional imaging insights are important to emphasize for its detection (*Figure 2*). The standard initial step with US involves the assessment of the LVEF as an indirect marker for contractility, which often employs the eyeballing method using two-dimensional imaging. Despite the arbitrary and debated division within the HF spectrum imposed by LVEF, its importance continues to be recognized as a crucial determinant in attaining long-term optimal management.[1] In acute settings, the presence of a preserved LVEF may inadvertently divert physicians from making a timely and accurate diagnosis of HF. Nevertheless, it is important to realize that many patients with HFpEF demonstrate more nuanced abnormalities in systolic function, in particular in the longitudinal shortening, despite preserved EF, and this is clearly linked with worse long-term outcome.[9] While less frequently employed in acute settings, speckle tracking strain has evolved as a reliable tool for assessing global longitudinal strain (GLS). This method offers prognostic information, indicates potential aetiologies, and is easily performed on contemporary machines, thereby avoiding the need for extensive post-processing work.[10] Reduced GLS is found in 50–60% of HFpEF cases, and absolute values below 16% serve as a predictive indicator for a worse prognosis, yet vendor dependence remains an issue.[11,12] Alternatively, LV longitudinal function can be evaluated through measures such as mitral annulus systolic ejection velocity (S′) using tissue Doppler and mitral annulus plane systolic descent (MAPSE) using the M-mode.[13,14]

Beyond EF, an assessment of global and regional LV wall thickness is recommended, taking into consideration the LV end-diastolic diameter. Patients with HFpEF commonly exhibit mild to moderate hypertrophy within the range of 11–16 mm, accompanied by concentric remodelling, typically attributable to increased afterload. Importantly, this is not an obligatory criterion for the diagnosis, and the LV wall was found to be normal in 46% of patients with HFpEF in the PARAGON-HF trial.[15] It is furthermore essential to exclude severe valvular heart disease, initially by colour Doppler and 2D structural imaging.[16] Of note, LVEF can be (falsely) preserved in the setting of significant mitral regurgitation given the reduced afterload.

Additionally, the structure and function of the left atrium (LA) should be assessed during the initial echocardiogram.[12] Abnormalities in LA function within HFpEF are more predictive of adverse outcomes than those of the LV.[17] Left atrial enlargement is indicative of a multifaceted interaction involving prolonged diastolic relaxation accompanied by

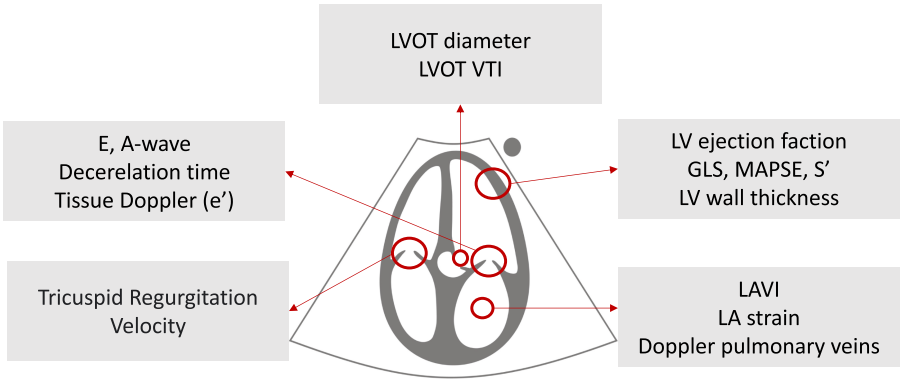

Figure 2 Cardiac ultrasound examination with focus on heart failure with preserved ejection fraction. GLS, global strain; LAVI, left atrial volume; LV, left ventricle; LVOT, left ventricular outflow tract; MAPSE, mitral annular plane excursion; VTI, velocity time integral.

elevated LV pressures, commonly associated with atrial fibrillation as a comorbidity, and coexisting mitral valve disease, with one in four patients with HFpEF presenting with (atrial functional) mitral regurgitation.[18,19] Left atrial volume should be calculated in biplane from dedicated apical four- and two-chamber views and indexed to body surface area [left atrial volume index (LAVI)]. Patients with HFpEF typically present with LAVI exceeding 34 mL/m^2, attributed to factors such as diastolic dysfunction, atrial fibrillation, and intrinsic atrial myopathy.

Functional assessment

The vast majority of acute patients with HFpEF have pulmonary hypertension, which starts as a passive result of downstream LA pressure elevation but, in many patients, ultimately leads to pulmonary vascular disease (PVD), characterized by a combination of vascular remodelling and vasoconstriction that affects the pulmonary veins, capillaries, and small arteries.[20] The estimation of systolic pulmonary pressure through Doppler echocardiography and the application of the Bernoulli formula are crucial for initial screening. The development of PVD is associated with an increased risk of RV dilation and dysfunction, secondary tricuspid valve regurgitation, and exaggerated right-sided venous congestion.[17] Mid-systolic notching in the RV outflow Doppler profile can be of value for diagnosing high pulmonary resistance in the context of PVD.

Utilizing US to measure LV outflow tract (LVOT) diameter and velocity–time integral (VTI) facilitates accurate calculations of stroke volume and cardiac output. Low-flow status in the context of HFpEF can arise from a combination of factors, including diminished longitudinal function, elevated heart rate, and impaired filling. Additionally, in a non-dilated left ventricle, subtle decreases in LVEF may lead to a significantly depressed cardiac output. A proposed cut-off value for LVOT VTI within HFpEF is 15.8 cm, serving to identify patients at an increased risk of adverse outcomes during subsequent hospitalization.[21]

Beyond LAVI, there is an increasing emphasis on LA function. It is important to note that this is intricately connected with diastolic and LV systolic (dys)function. Left atrial function can be assessed through the transmitral A-wave or the ratio of systolic to diastolic wave in the pulmonary veins (systolic blunting indicates LA pressure overload), but this cannot be disentangled from diastolic dysfunction. A more comprehensive approach involves calculating volumes throughout the cardiac cycle, to determine LA EF or utilizing LA strain. The latter is also recommended for gaining insights into LV filling pressures but often requires additional post-processing work and is less applicable in acute settings.[22] In a multicentric study where invasive LV filling pressure was used as a reference, LA reservoir strain < 18% was consistent with elevated LV filling pressure with excellent feasibility.[23]

A large amount of research has been devoted to the quantification of LV diastolic function, specifically ventricular stiffness and compliance. However, the presence of diastolic dysfunction is neither specific nor sufficient to diagnose HFpEF, resulting in the abandonment of the term diastolic HF.[1] The functional evaluation of diastolic function necessitates the integration of multiple parameters, such as the transmitral pulsed-wave Doppler signal (E- and A-waves, deceleration time), myocardial motion via tissue Doppler (e′-, s′-, a′-waves), as well as the impact on the LA, pulmonary circulation, and right heart. This Bayesian approach is complex and is not frequently employed in emergency settings.[12] In acute scenarios, one may prioritize insights into LV filling pressure over diastolic function (although interconnected), as the former can significantly impact acute treatment strategies. For this purpose, a summary of all US signs indicative of elevated filling pressures is compiled in *Table 1*. However, it is imperative to contextualize these findings within the patient's clinical presentation, and the likelihood of elevated filling pressures increases proportionally with the number of abnormal parameters present. The reliability and clinical utility of cardiac US for non-invasively assessing elevated pressures are commendably high, yet they do not match the precision of invasive methods.[24] Investigating beyond the cardiac realm appears promising for a more comprehensive evaluation of congestion and its underlying causes.

Lung ultrasound

Bedside application of lung US (LUS) is practical, quick, and easy to learn and has the potential to enhance outcomes.[25,26] Furthermore, dyspnoeic patients can undergo scanning in their comfortable position within 1–5 min. While the principal protocol entails a 6–8-region assessment, a rapid 4-region scan may prove adequate in emergency settings (*Figure 3*).[26–28] Among patients with acute dyspnoea, LUS has demonstrated excellent diagnostic accuracy for decompensated HF, with a sensitivity of 94–97% and a specificity of 97%, which has been shown to be superior to both clinical assessment and the chest X-ray, even when there is superimposed pneumonia.[29]

Lung US is mostly based on artefacts, with a central focus on the pleura. The highly reflective pleura that moves synchronously with respiration (lung sliding) gives rise to mirror reverberations, known as A-lines.[26] These A-lines manifest as horizontal, parallel lines equidistant from each other in normal aerated lungs. On the contrary, B-lines exhibit vertical alignment and represent comet tail artefacts arising from

Table 1 Ultrasound-based indicators of pressure and/or volume overload

Signs of pressure and/or volume overload			
Cardiac	**Lung**	**Venous**	**Chest X-ray**
E/A ratio > 2	Pleural effusion	IVC > 21 mm	Pleural effusion
E/e′ > 14	B-lines	Reduced IVC respiratory variation	Cephalization pulmonary veins
TRV > 2.8 m/s		Pulmonary veins S < D	Kerley B-lines
Deceleration time <160 ms		Hepatic veins S < D	
LAVI > 34 mL/m^2		S flow reversal	
LA reservoir strain < 18%		Portal vein pulsatility	

Cut-off values align with the European Association of Cardiovascular Imaging (EACVI) imaging expert consensus on HFpEF.[12]
IVC, inferior vena cava; LA, left atrium; LAVI, left atrial volume index; S, systole; D, diastole; TRV, tricuspid regurgitation velocity.

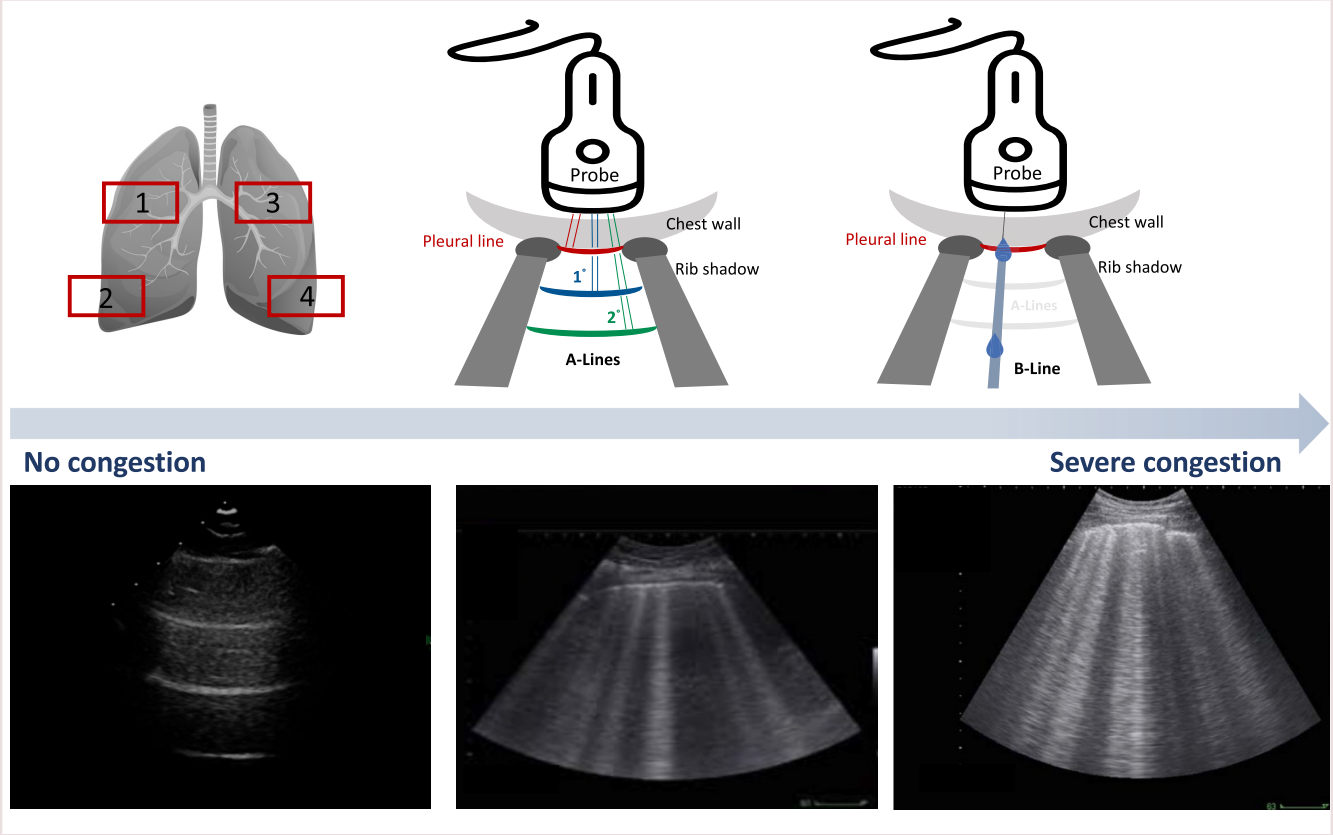

Figure 3 Lung ultrasound examination: the upper panel displays four-region scan and illustration on A and B-lines (which may coexist). The lower panel presents a patient example depicting a spectrum from no to severe lung congestion with corresponding B-line density.

the pleura line and extending towards the bottom of the US sector. These findings suggest heightened lung density, i.e. oedema and/or accumulation of inflammatory cells, and may occur alongside the presence of A-lines.[26,30] A pathological region is identified by the presence of three or more B-lines, and a positive examination for interstitial syndromes is established when two or more such positive areas are observed. The number of B-lines increases with the severity of congestion and facilitates monitoring of response to treatment.[31] When confluent, their numerical density can be approximated by assessing the proportion (%) of space they encompass on the screen beneath the pleural line, divided by 10.[26] For instance, if 80% of the screen below the pleural line is filled with B-lines, it would conventionally be quantified as 8 B-lines, up to a maximum of 10 per zone.

It is important to mention that interstitial syndromes (i.e. B-lines) may also result from interstitial pneumonia, diffuse parenchymal lung disease, and acute respiratory distress syndrome (ARDS) and do not specifically indicate HF. Certain sonographic characteristics can aid in distinguishing between these diverse causes. A grossly irregular pleural line with patchy, non–gravity-related distribution of B-lines suggests ARDS or intrinsic lung disease, whereas a thin and regular pleural line

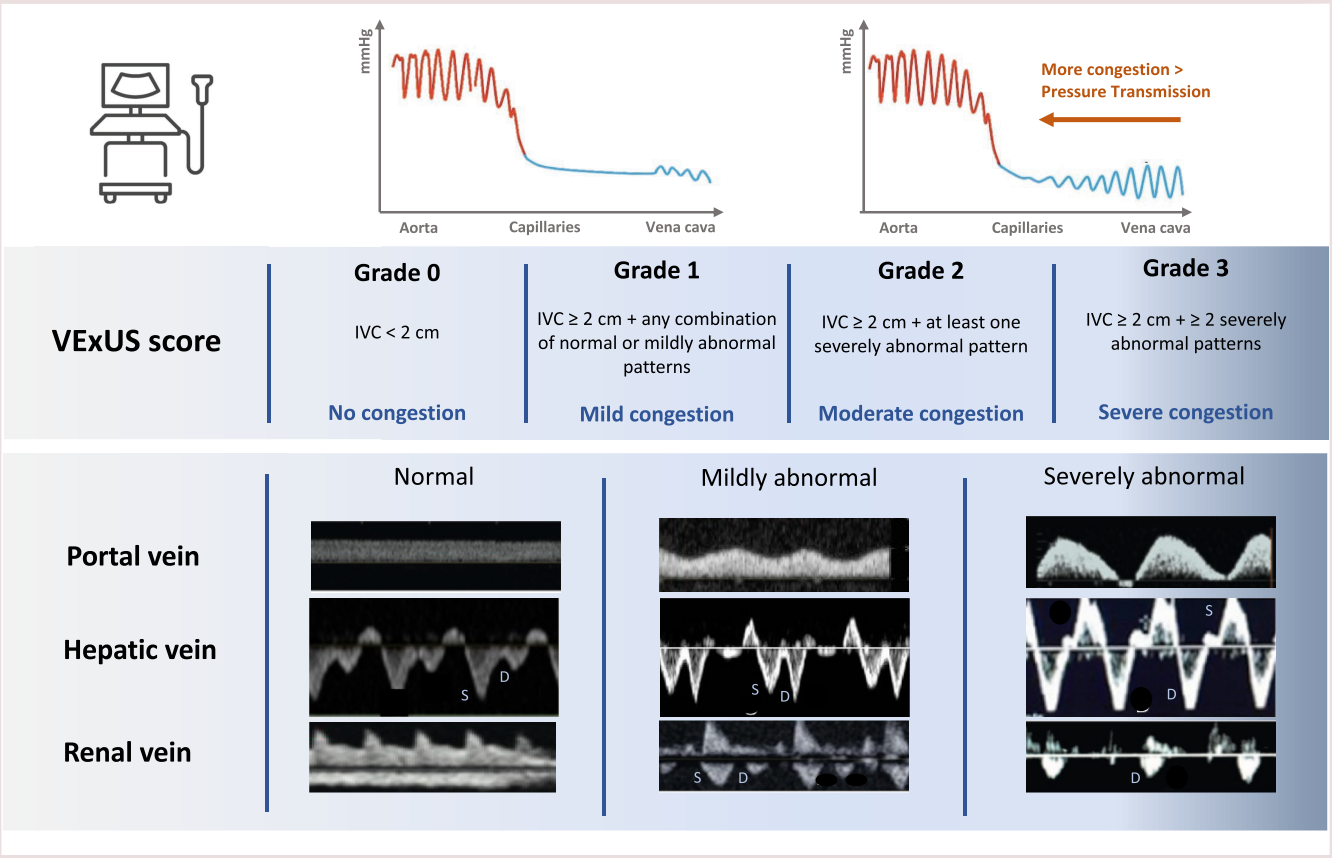

Figure 4 Vascular ultrasound examination: the upper panel illustrates increased pulsatility in the peripheral veins in setting of congestion. The lower panel presents patient examples depicting a spectrum from no to severe congestion. D, diastolic; IVC, inferior vena cava; S, systolic.

with homogeneous, gravity-related B-line distribution is indicative of cardiogenic pulmonary oedema.[26,30] Additionally, pleural effusion in the dependent zones, i.e. lateral and posterior chest wall (e.g. posterior axillary line), at the level of costophrenic angles further supports HF as the cause of B-lines (*Table 1*).[26]

Vascular ultrasound

In the normal circulatory system, pulsatility in the blood vessels increases as one approaches the heart. The pulsatile flow within the aorta is dampened resulting in continuous flow through the capillaries, thereby facilitating gas exchange. Venous return in compliant veins is initially continuous, but pulsatility increases as one approaches the right atrium due to the impact of systole and diastole, as well as respiratory swings. However, in the context of venous congestion, regulatory mechanisms and venous compliance are largely compromised, leading to an increase in venous pulsatility. Venous pressure, even more than impaired cardiac output, is a critical haemodynamic parameter affecting organ perfusion.[32] This prompted the utilization of mapping venous return for the grading of congestion using Doppler imaging in HF (HFpEF and HFrEF). As a general guideline, higher pulsatility in the peripheral veins correlates with increased congestion. The Venous Excess UtraSound (VExUS) score was proposed, which may serve to assist in both the diagnosis and monitoring of decompensated HFpEF. The VExUS system has four grades based on Doppler flow indices of the hepatic, renal, and portal vein in addition to inferior vena cava (IVC) assessment (*Figure 4*). A curvilinear transducer is preferred, although a phased array transducer is an acceptable alternative.[33]

Ultrasonography of the IVC is commonly used to estimate right atrial pressure (RAP), although the correlation with invasive catheterization is modest and there are limitations, for instances, in athletes and mechanically ventilated patients.[34] An IVC diameter larger than 21 mm with reduced respiratory variation (<50%) suggests increased RAP.[35] Examining venous flow in the hepatic veins (HV), along with the more static IVC assessment, offers insights into the mechanics of right heart filling patterns. Additionally, the flow pattern in portal and intra-renal veins yields information about the extent of pressure transmission to peripheral organs.[34]

The HV can easily be visualized from the subxiphoid position, as they drain into the IVC. Normal HV flow is pulsatile due to its proximity to the RA and consists of two antegrade (below the baseline) waves (S and D) and a retrograde (above the baseline) A-wave due to atrial contraction.[36] As within the pulmonary veins, the S-wave is normally larger than the D-wave. With increase in RAP, S-wave becomes blunted and smaller than the D-wave as the pressure gradient between the HV and the RA reduces. Further rise in RAP may lead to disappearance or reversal of the S-wave.[36] It is recommended to concurrently record an electrocardiogram trace along with HV waveform to enhance precise wave identification.

Visualizing the renal vessels requires a specific position, breath-hold, and preferably, a low-frequency probe, making it less practical in an acute setting. The portal veins (PV) are visualized by positioning the probe along the anterior axillary line, tilting the tip slightly to anterior.[36] They are easily distinguishable from HV due to the echogenic nature of the PV walls, the red appearance of flow (as opposed to the blue colour in HV), and their course of crossing the IVC. Normal portal and renal vein flow is continuous. Worsening venous congestion results in

Table 2 Overview of heart failure with preserved ejection fraction mimickers: clinical and echocardiographic clues alongside further diagnostic investigations

HFpEF mimicker	Clinical clues	Echocardiographic clues	Extra work-up
Cardiac amyloidosis	≥65 y male. Carpal tunnel syndrome. Ruptured biceps tendon. Spinal stenosis. Peripheral neuropathy. Low QRS voltage. Renal involvement. Stroke. GDMT intolerance. Orthostatic hypotension.	LV wall thickness ≥ 12 mm[45], most often diffuse. Thickening of the valve cusps. Low tissue Doppler velocities. Reduced GLS with apical sparing. Myocardial granular sparkling. Pericardial effusion.	Nuclear scintigraphy. Monoclonal protein screen. Biopsy. CMR with T1 mapping.
Myocarditis	Recent flu-like illness. Immunotherapy.	Regional wall motion abnormalities. Local thickening of the ventricular wall.	CMR. Endomyocardial biopsy.
Hypertrophic cardiomyopathy	Family history. Sudden cardiac death. Increased QRS voltage and deep T-waves.	Unexplained LV wall thickness of ≥15 mm[46], most often asymmetric. LVOT obstruction, reduced regional strain.	CMR. Genetic testing.
Cardiac sarcoidosis	Arrhythmias. Conduction block. Extracardiac disease (pulmonary, ocular, dermatologic).	Septal thinning. Pulmonary hypertension. Regional wall motion abnormalities.	CMR. FDG-PET scan. Tissue biopsy.
Haemochromatosis	Family history. Erectile dysfunction. Diabetes. Skin pigmentation. Liver disease. Frequent transfusions.	Biatrial enlargement. Restrictive filling. Decreased peak velocities LV lateral wall.	Ferritin and transferrin. HFE genetic testing, *T2*-weighted *CMR*.
Fabry disease	Angiokeratomas. Proteinuria. X-linked. Neuropathic pain. Hearing loss.	Concentric LV hypertrophy. Prominent papillary muscles. Valve leaflet thickening. Reduction in GLS, especially basal inferolateral.	Serum alpha-galactosidase level. GLA genetic testing. CMR. biopsy
High-output heart failure	Rendu–Osler–Weber. Fistulas. Excessive alcohol use.	Increased LVOT VTI, stroke volume, and cardiac output. Four-chamber enlargements.	Underlying causes: anaemia, AV malformations, cirrhosis, fistulas, thiamine deficiency.
Pericardial disease	Prior cardiac surgery. Chest radiation. Pericarditis.	Calcified pericardium. Septal bounce. Exaggerated respiratory variation of transvalvular flow. Annulus paradoxus. Prominent reversal of expiratory late diastolic flow in hepatic veins.	CT. CMR. Right heart catheterization.
Precapillary pulmonary hypertension	Family history. Connective tissue disease. Pulmonary embolisms. HIV. Anorexigenic exposure. Lung disease.	Pulmonary hypertension. Systolic notching RVOT. Interventricular septal flattening. RV dilatation. Pulmonary artery dilatation. No LA enlargement. E/A ratio < 1.	Chest CT. Nuclear imaging.

AV, arteriovenous; CMR, cardiac magnetic resonance; CT, computed tomography; GLA, galactosidase A; GDMT, guideline-directed medical therapy; HFE, haemochromatosis gene; HIV, human immunodeficiency virus; LA, left atrium; LV, left ventricle; LVOT, left ventricular outflow tract; RVOT, right ventricular outflow tract.

pulsatile flow. These alterations can be quantified using the PV pulsatility fraction (PVPF): $[(V_{max} - V_{min})/V_{max}] \times 100$. Portal vein pulsatility fraction > 30% is considered mildly abnormal, while PVPF > 50% is considered to indicate severe congestion. In post-cardiac surgery patients, increased PVPF has shown to be the most important predictor of acute kidney injury development.[33]

Understanding venous waveforms is crucial, yet certain conditions can independently alter them, introducing interpretation caveats. Increased PVPF may be seen in slender, healthy individuals. Cirrhosis may modify flow in both the HV and PV. Obstructive uropathy and chronic kidney disease can impact intra-renal flow. Additionally, the presence of right-sided valvular disease and arrhythmias like atrial fibrillation complicates interpretation. Importantly, venous congestion indicated by the VExUS score may not only arise from volume overload but also from pressure overload (e.g. pulmonary hypertension) or right HF (e.g. right ventricular infarction). In addition to the VExUS score, evaluating the internal jugular vein, for example, during central line insertion can be helpful. The jugular venous distensibility (JVD) ratio is defined as the maximal diameter during Valsalva to its resting diameter (end-expiratory phase), with values <4 indicating abnormality and severe congestion potentially reducing the ratio to <2.[34]

Multimodality imaging

Chest X-ray

The use of chest X-ray stands as a first-line diagnostic tool for patients experiencing acute dyspnoea. It serves not only in discerning pulmonary congestion but also in detecting alternative pathologies, with a particular focus on pneumonia, pleural effusion, and pneumothorax.[37] However, it is important to note that the absence of radiographic signs of congestion does not rule out HF, as its sensitivity ranges from 14% to 68%, which is lower than LUS.[38]

Pleural effusion becomes visible on a routine chest X-ray when the fluid volume reaches ~250–600 mL, often manifesting as blunting of the costophrenic angle.[39] The sensitivity of detecting fluid layering against the dependent parietal pleura is highest in a lateral decubitus projection, although this is seldom obtained in acute situations.

Conversely, supine projections can obscure large quantities of fluid.[40] Additionally, an increased cardiothoracic ratio, often reported as a marker for cardiomegaly, may mislead clinicians towards HFrEF with dilated LV in the acute setting, as it should not be measured on bedside anteroposterior radiographs where the heart appears artificially enlarged due to X-ray beam divergence.

Others radiographic signs that may indicate decompensated HFpEF are cephalization of the pulmonary veins and Kerley B-lines. Cephalization, also described as upper lobe pulmonary venous diversion, is present when the upper lobe veins are of the same or higher diameter than the lower lobe veins and reflect elevation of left atrial pressure. Kerley B-lines or septal lines are seen when the interlobular septa in the pulmonary interstitium become prominent due to oedema.[41] More recently, a new semiquantitative approach to pulmonary congestion has emerged; the congestion score index (CSI) as a semiquantitative approach based on a six-zone evaluation, scoring each zone from 0 (no congestion) to 3 (intense alveolar pulmonary oedema), can be used for risk stratification in patients with acute decompensated HF.[42–44]

Computed tomographyh and cardiac magnetic resonance

While computed tomography (CT) imaging is not the first-line modality in the acute setting, it may be employed to refine the differential diagnosis, particularly in cases where acute pulmonary embolism or severe lung involvement is suspected. When performed, the recognition of pericardial thickening, coronary calcium, dilated pulmonary artery, or enlarged chamber volumes can offer valuable indicators for an accurate diagnosis. It is noteworthy that CT imaging is pertinent in assessing pulmonary vein stenoses after AF ablation, which can mimic HFpEF.[9]

Abnormalities found in the baseline work-up should trigger potential further exploration by other (imaging) modalities. Cardiac magnetic resonance imaging has an important complementary role, especially in the setting with poor US windows or when underlying myocardial disease is suspected.[37] Cardiac magnetic resonance is the only imaging technique to provide detailed tissue characterization of the myocardium and assessment of fibrosis.[12] However, the applicability of cardiac CT and MR imaging in the emergency setting is constrained by several factors, including limited availability, extended examination times, higher costs, susceptibility to respiratory artefacts, and the lack of round-the-clock expertise.

Heart failure with preserved ejection fraction mimickers

Heart failure with preserved ejection fraction is acknowledged as an umbrella term characterized by significant heterogeneity in underlying diseases, which emphasizes the growing importance of meticulous phenotyping. Several distinct cardiac diseases closely mimic HFpEF physiology, and signs and symptoms yet require specific interventions.[17] This is particularly pertinent in younger patients (<55 years old) without risk factors such as obesity, diabetes, hypertension, and disease. An overview of potential HFpEF mimickers is provided in *Table 2*, accompanied by pertinent clues to facilitate diagnosis. This holds significance in acute settings, considering the therapeutic implications involved. For example, identification of hypertrophic cardiomyopathy necessitates vigilance in the management of LVOT obstruction, involving strategies such as avoiding dehydration, refraining from positive inotropes, or abstaining from the use of an intra-aortic balloon pump in deteriorating conditions.[47] Similarly, suspected precapillary pulmonary hypertension warrants consideration of the impact of administered drugs on the pulmonary circulation, influencing the selection of vasopressors and inotropes. While the therapeutic approach falls beyond this review, it is crucial to note that imaging extends beyond diagnosis, encompassing treatment and prognostication throughout the patient care continuum.

Conclusions

Given the increasing prevalence of HFpEF, it is essential to be familiar with its diagnosis in emergency settings. Ultrasonography imaging plays an important role in this respect and may guide important early treatment decisions. Restricting the evaluation to focused cardiac US only may be misleading, as HFpEF is associated with multiple comorbidities in other organs. Therefore, the integration of additional US modalities, such as vascular and lung ultrasound, into the clinical work-up appears promising for rapid diagnosis of congestion and its cause, enabling the swift integration of appropriate therapy.

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Data availability

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