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How Does Left Ventricular Ejection Fraction Affect the Multimodal Assessment of Congestion in Patients with Acute Heart Failure? Results from a Prospective Study

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Abstract

The assessment of systemic congestion in acute heart failure (AHF) remains clinically challenging, particularly across different left ventricular ejection fraction (LVEF) phenotypes. This study aimed to evaluate whether differences exist in the degree of congestion, assessed through a multimodal approach including physical examination, biomarkers (NT-proBNP, CA125), and point-of-care ultrasound using the Venous Excess Ultrasound (VExUS) protocol, between patients with preserved (HFpEF) and reduced ejection fraction (HFrEF). We conducted a prospective observational study involving 90 hospitalized AHF patients, 80 of whom underwent a complete VExUS assessment. Although patients with HFrEF exhibited higher levels of NT-proBNP and CA125, and more frequent signs of third-space fluid accumulation such as pleural effusion and ascites, no statistically significant differences were found in VExUS grades between the two groups. These findings suggest that the VExUS protocol provides consistent and reproducible information on systemic venous congestion, regardless of LVEF phenotype. Its integration into clinical practice may help refine congestion assessment and optimize diuretic therapy. Further multicenter studies with larger populations are warranted to validate its diagnostic and prognostic utility and to determine its potential role in guiding individualized treatment strategies in AHF.

Keywords: heart failure; left ventricular ejection fraction; Venous Excess Ultrasound Score; congestion; biomarkers; NTproBNP; CA125

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1. Introduction

Heart failure (HF) is a clinical syndrome characterized by structural or functional cardiac abnormalities that impair ventricular filling or the ejection of blood [1], triggering a cascade of compensatory neurohormonal and inflammatory responses [2–4]. The estimated prevalence of HF is around 2% among adults in developed countries and exceeds 10% in

those over 70 years old, making it a major healthcare concern [3]. In Spain, the prevalence is similar [3] and HF remains a leading cause of hospitalization in older adults, primarily due to signs and symptoms of congestion [3,5].

To better understand the clinical course and guide treatment, HF is classified according to left ventricular ejection fraction (LVEF) into three main phenotypes: reduced (HFrEF, ≤40%), mildly reduced (HFmrEF, 41–49%), and preserved (HFpEF, ≥50%) [3]. This classification is clinically significant, as it reflects different underlying etiologies, comorbidities, and, most importantly, variable responses to treatment [5]. HFrEF is characterized predominantly by neurohormonal activation and structural remodeling, whereas HFpEF involves endothelial and microvascular dysfunction and presents a heterogeneous biological profile [5]. Despite these distinctions, decongestive treatment strategies in acute settings often do not differ across these phenotypes [5].

Accurate congestion assessment remains one of the most critical and challenging aspects of HF management. Persistent congestion after hospital discharge is strongly associated with poor prognosis [6]. Hemodynamic congestion, or elevated ventricular filling pressures, often precedes clinical signs by days to weeks [6]. Unfortunately, a reliable non-invasive method for determining volume status is lacking, posing a significant diagnostic challenge when physical examination and standard laboratory tests are inconclusive [7,8].

To address this issue, several tools have emerged, including congestion biomarkers, such as N-terminal pro-B-type natriuretic peptide (NT-proBNP) and Carbohydrate Antigen 125 (CA125) [9,10], and point-of-care ultrasound (POCUS), which has become increasingly integrated into clinical practice across internal medicine units worldwide. Bedside ultrasound has proven to be a valuable diagnostic tool that complements history and physical examination [11–14] and is now considered the fifth pillar of physical examination in HF [15].

Lung ultrasound (LUS) helps detect extravascular pulmonary water through B-lines, which correlate with worse prognosis [13,14]. Inferior vena cava (IVC) diameter assessment provides a simple estimate of right atrial pressure and is associated with changes in pulmonary capillary wedge pressure [12]. Therapy guided by IVC diameter in hospitalized HF patients has been shown to lead to reductions in rehospitalizations and mortality [11–14]. A recent pilot study (CAVAL US-AHF) combining a Doppler assessment of cardiac filling pressures, IVC, and LUS demonstrated that this approach is safe and reliable for guiding therapy [12].

In line with these advances, the recent consensus on the management of fluid overload in acute HF by Pau Llácer et al. [16] proposes two distinct congestion phenotypes and recommends a multiparametric approach that integrates clinical symptoms, biomarkers, and ultrasound findings to guide treatment.

To further refine the quantification of congestion, Beaubien-Souligny et al. proposed the Venous Excess Ultrasound (VExUS) score, a protocol based on Doppler flow patterns in three abdominal veins—hepatic, portal, and intrarenal—that classifies patients into three levels of congestion: none, mild, or severe [17].

The VExUS protocol begins by assessing the IVC. If the diameter is <2 cm, venous congestion is ruled out. If it is ≥2 cm, a further Doppler evaluation is performed. The hepatic vein waveform, portal vein pulsatility, and intrarenal vein pattern are analyzed and integrated into a severity score (grades 0–3). Higher VExUS grades correlate with increased venous pressure and worse outcomes [11,13,18–20].

Although the VExUS score is gaining attention as a tool for assessing systemic venous congestion, it has limitations. These include operator dependency, anatomical variability, and disparities in ultrasound equipment. Moreover, most studies have focused on critically

ill patients and those with HFrEF [18,20], leaving its performance across different LVEF phenotypes uncertain.

Some authors suggest that systemic congestion may be less pronounced or may follow a distinct pathophysiological pattern in HFpEF compared to HFrEF [21], which could impact the utility of VExUS in this population. Supporting this, a recent prospective study in HFpEF patients demonstrated that higher VExUS grades correlated with worse clinical outcomes [13,19]. Nevertheless, the pathophysiological characteristics of HFpEF—such as increased ventricular stiffness and diastolic dysfunction—might influence how congestion manifests and, consequently, how accurately VExUS captures it [20].

Although ultrasound is an emerging technique in HF management supported by growing evidence, it still presents certain limitations, including interobserver variability, patient anatomy, and differences in equipment [13,18,22]. Therefore, further studies are needed to explore multimodal congestion assessment strategies and to evaluate the integration of the VExUS protocol in heart failure patients, in order to support or refute its systematic use as a novel tool for patient management. Given these considerations, further research is warranted to determine whether VExUS offers consistent diagnostic value across the full spectrum of LVEF phenotypes.

This study, conducted by the Heart Failure Research Group of the Aragón Health Research Institute (GIIS-043), aims to assess the relationship between VExUS and LVEF and to evaluate the utility of this protocol in real-world clinical settings. Specifically, we aim to determine whether systemic venous congestion quantified by VExUS correlates with clinical, laboratory, and ultrasound findings and whether its diagnostic performance differs according to LVEF.

2. Materials and Methods

2.1. Study Design

This was a prospective, observational, single-center cohort study conducted at the Internal Medicine Department of Hospital Clínico Universitario Lozano Blesa, Zaragoza, Spain. This study included patients from the Cardio-Ren cohort (C.P.—COL21/003) during the 2023–2024 academic year. No pharmacological interventions were performed beyond routine clinical care.

2.2. Study Population

We included adult patients (≥18 years) admitted with a primary diagnosis of acute heart failure (AHF) or decompensated chronic heart failure, who presented with at least one clinical sign or symptom of congestion, such as dyspnea, orthopnea, bendopnea, peripheral edema, pulmonary crackles, ascites, or jugular vein distension, and an elevated NT-proBNP level (>1000 pg/mL) within the first 24 h of admission. This NT-proBNP cutoff was selected based on its frequent use in relevant clinical studies, such as the PARADIGM-HF trial [23], where it identifies patients with higher hemodynamic burden and clinical risk. These inclusion criteria were applied uniformly across all participants to ensure consistency and reduce potential selection bias. Written informed consent was required.

Exclusion criteria included admission from the intensive care unit or emergency observation area with stay > 24 h; refusal to participate or the absence of written consent; severe cognitive or functional impairment precluding outpatient follow-up; and advanced chronic kidney disease (CKD), renal transplant, or current dialysis treatment.

2.3. Data Collection and Variables

The aim of this study was to describe and compare the congestion profiles of patients with preserved versus reduced ejection fraction at the time of admission. Therefore, a

multimodal assessment—including physical examination, laboratory tests, and point-of-care ultrasound (POCUS)—was performed within the first 24 h of hospitalization.

We recorded demographic and clinical variables, including age, sex, comorbidities, New York Heart Association (NYHA) functional class, and chronic home medications. Clinical signs of congestion—such as dyspnea, orthopnea, bendopnea, peripheral edema, jugular vein distension, and pulmonary crackles—were systematically assessed. Laboratory tests included assessing the levels of NT-proBNP, CA125, creatinine, sodium, and potassium, and congestion indices such as the combined congestion algorithm (CAC) were calculated.

Echocardiographic evaluation included assessing inferior vena cava (IVC) diameter, the presence of B-lines, pleural or abdominal effusion, and the Venous Excess Ultrasound (VExUS) score, comprising Doppler patterns of hepatic, portal, and intrarenal veins. Congestion was also assessed using a clinical congestion score (range: 0–8) based on dyspnea, edema, and jugular venous distension [7].

Although the primary analysis focused on this baseline evaluation, all patients were monitored throughout hospitalization and prospectively followed for 90 days to assess therapeutic interventions (such as intravenous loop diuretics or hypertonic saline), in-hospital mortality, and post-discharge outcomes including mortality, readmission, and diuretic requirements.

2.4. Echographic Evaluation

All patients underwent standardized bedside ultrasound assessment using Lumify® (Philips Ibérica, Madrid, España) and Omega® (Esaote España S.A.U., Barcelona, Spain) devices, equipped with both phased-array and convex probes. Two trained physicians performed image acquisition after a calibration phase using ten representative cases, achieving interobserver agreement with a kappa coefficient of 0.80 (see Supplementary Materials).

LVEF was obtained from a standard transthoracic echocardiogram performed either during admission or within the previous 12 months. Patients were categorized as having preserved LVEF (HFpEF, $\geq$50%) or reduced LVEF (HFrEF, <50%).

Lung ultrasound was performed to assess B-lines in eight thoracic quadrants. Pleural effusion and ascites were evaluated via abdominal ultrasound. [IVC diameter was measured approximately 2 cm from its junction with the right atrium, and inspiratory collapsibility was recorded. A collapsibility > 50% was considered normal.].

The VExUS protocol was applied in patients with an IVC diameter $\geq$ 2 cm. It included a Doppler analysis of the hepatic, portal, and intrarenal veins:

- In the hepatic veins, waveforms were classified as normal, mildly, or severely altered based on the S/D wave ratio and flow direction.
- Portal vein pulsatility was categorized as <30%, 30–50%, or >50%, with higher pulsatility indicating greater congestion.
- Intrarenal venous flow was categorized as continuous, biphasic, or monophasic, with monophasic indicating severe congestion.

Based on these findings, a global VExUS score was assigned, classifying patients into four grades: grade 0 (no congestion), grade 1 (mild), grade 2 (moderate), and grade 3 (severe). For statistical analysis, this classification was dichotomized into low (grades 0 and 1) versus high (grades 2 and 3) systemic venous congestion.

2.5. Statistical Analysis

Qualitative variables were reported as percentages and quantitative variables as means or medians depending on the distribution (assessed by the Shapiro–Wilk test). Group comparisons employed a chi-square test, Student's *t*-test, ANOVA, or the Kruskal–Wallis

test, as appropriate. A two-tailed p-value < 0.05 was considered statistically significant. Analyses were performed using Jamovi software (version 2.3.28).

2.6. Ethical Considerations

This study complied with the principles of the Declaration of Helsinki (2013 revision). Approval was granted by the Ethics Committee of the Autonomous Community of Aragón (CEICA). No additional diagnostic procedures were required. Data were pseudonymized using coded identifiers accessible only to the research team. This study was conducted without external funding or participant compensation.

3. Results

A total of 90 patients with (acute heart failure) AHF were included, with a mean age of 84.5 ± 6 years; 48% were women. According to baseline NYHA functional classification, 15.6% were class I, 66.7% class II, 16.6% class III, and 1.1% class IV. Hypertension (86.7%) and atrial fibrillation (75.6%) were the most frequent comorbidities, followed by dyslipidemia (57.8%), CKD (35.6%), diabetes (34.4%), and chronic obstructive pulmonary disease (25.6%). The cohort was predominantly treated with loop diuretics (75.6%), beta-blockers (65.5%), and renin–angiotensin system blockers (53.3%) prior to admission. ISGLT2 use was present in only 28.9% of the sample at the time of the initial assessment. Sixty patients (67%) had HFpEF, and thirty (33%) had HFrEF.

At admission, 35% had orthopnea at rest, 28% with two pillows, and 28% with one. Peripheral edema was moderate-to-severe in 52%, and 83% had jugular vein distention > 6 cm (Figure 1). Clinical congestion scores were similar between groups (median 4 points in HFpEF vs. 5 points in HFrEF; p = 0.119).

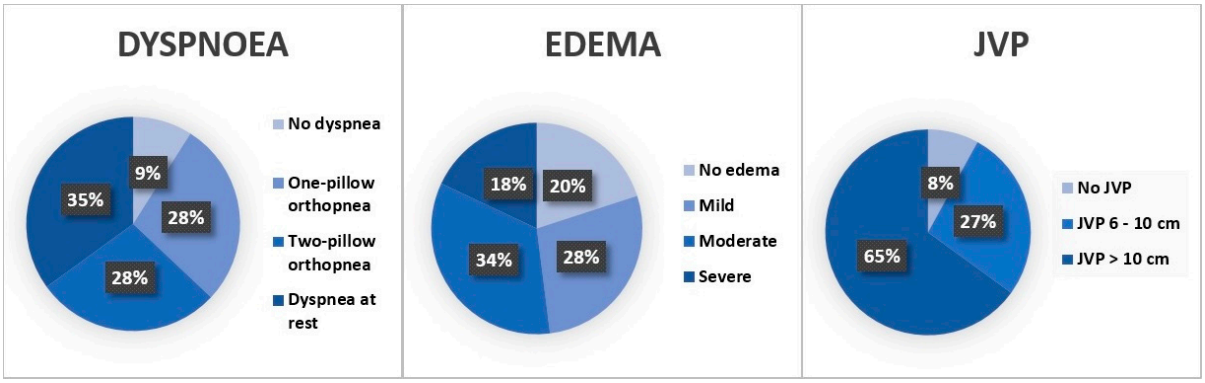

Figure 1. Degree of clinical congestion upon admission.

Lung ultrasound revealed the presence of B-lines in all evaluated patients. These were bilateral in 95% of patients with preserved left ventricular ejection fraction (HFpEF) and in 93% of those with reduced ejection fraction (HFrEF). Pleural effusion was observed in 52% of the HFpEF group, compared to 77% in the HFrEF group. Similarly, ascites was detected via abdominal ultrasound in 5% of HFpEF patients and 20% of HFrEF patients.

IVC measurements were comparable between groups. Patients with HFpEF had a mean IVC diameter of 22.4 mm ± 4.3 mm, with a collapse < 50% in 78% of cases. Those with HFrEF had a mean diameter of 22.8 mm ± 4.8 mm, and 70% exhibited a collapse < 50%.

It is important to highlight that, of the 90 patients included in our study, the VExUS protocol was successfully performed in 80 individuals. In the remaining 10 cases, inadequate acoustic windows made it technically impossible to obtain Doppler waveforms from one or more of the venous structures required for scoring—primarily the intrarenal veins.

Since complete evaluation is necessary for accurate classification, no VExUS grade was assigned when any component was missing.

Regarding hepatic vein Doppler assessment, no statistically significant differences were found (p = 0.242). Among HFpEF patients, 26% had normal flow, 41% showed moderate alterations, and 33% had severely abnormal flow. In the HFrEF group, these figures were 12%, 58%, and 30%, respectively. Similarly, no significant differences were observed in portal vein Doppler patterns (p = 0.355). In the HFpEF group, 35% had normal flow, 43% showed pulsatility < 50%, and 22% had pulsatility > 50%. In HFrEF patients, 24% had normal flow, 43% had pulsatility < 50%, and 43% showed pulsatility > 50%. As for intrarenal venous flow, the distribution was also similar (p = 0.839). In HFpEF patients, 55% exhibited continuous flow and 45% discontinuous flow (35% biphasic, 10% monophasic). In the HFrEF group, 63% had continuous flow and 37% discontinuous flow (30% biphasic, 7% monophasic) (Figure 2).

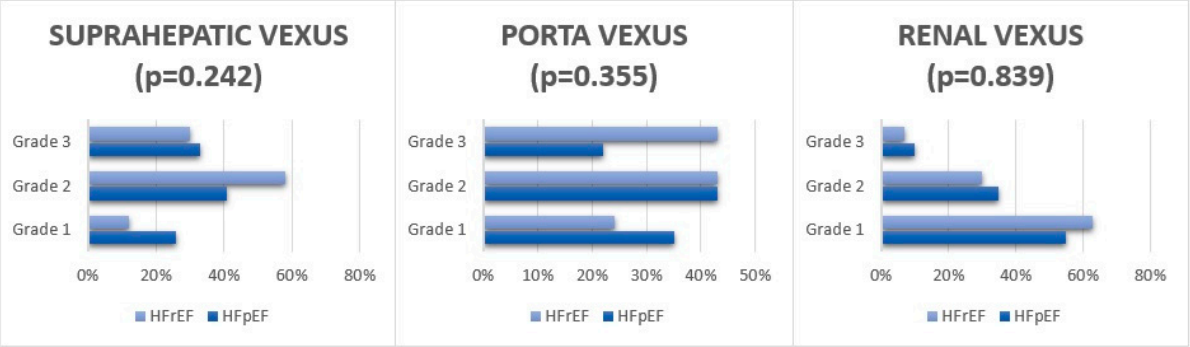

Figure 2. VExUS assessment. Suprahepatic, portal, and renal veins according to LVEF.

According to the VExUS classification, among patients with preserved ejection fraction (HFpEF), 20% were classified as grade 0 (no congestion), 39% as grade 1 (mild congestion), 26% as grade 2 (moderate congestion), and 15% as grade 3 (severe congestion). In the reduced ejection fraction group (HFrEF), 19% were classified as grade 0, 31% as grade 1, 38% as grade 2, and 12% as grade 3 (Figure 3).

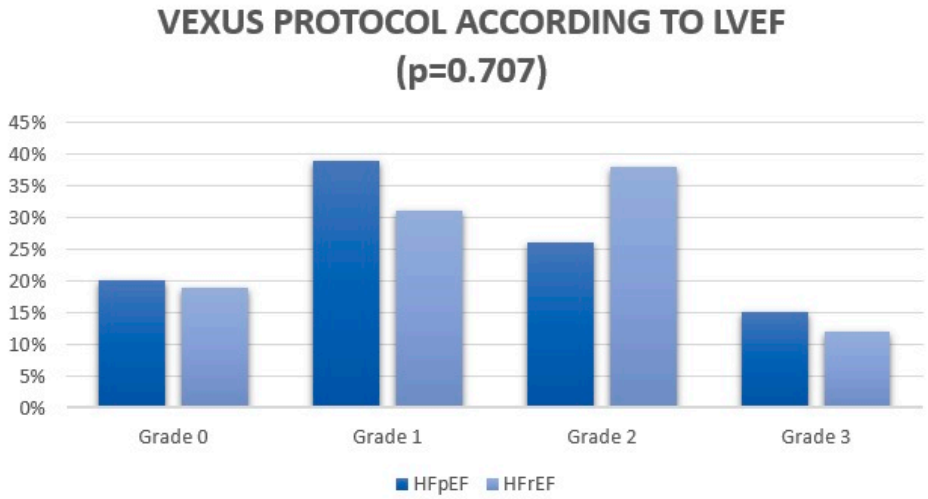

Figure 3. VExUS protocol according to LVEF.

When the cohort was dichotomized into low (VExUS grades 0–1) and moderate-to-severe (VExUS grades 2–3) congestion, 59% of HFpEF patients fell into the low-congestion group and 41% into the higher-congestion group. Among HFrEF patients, the distribution was evenly split (50% vs. 50%).

As previously described, there were no significant differences between the groups in terms of VExUS grading—neither in the four-grade classification ($p = 0.707$) nor in the dichotomized analysis ($p = 0.526$).

NT-proBNP and CA125 levels were significantly higher in HFrEF patients (median 11,278 vs. 4665 ng/L, $p < 0.001$ and 70.6 vs. 37.3 U/mL, $p = 0.032$, respectively). Although not statistically significant, HFrEF patients showed a lower estimated glomerular filtration rate and higher serum creatinine levels. Other parameters, including serum and urinary electrolytes, hemoglobin, and inflammatory markers, showed no significant intergroup differences (Table 1).

Table 1. Clinical, analytical, and ultrasound variables according to LVEF.

Variables	HFpEF (>50%)	HFrEF (<50%)	*p*-Value
Hypertension	52 (87)	26 (87)	1.000
Diabetes	20 (33)	11 (37)	0.754
Flutter/Atrial fibrillation	52 (87)	16 (53)	<0.001 *
Dyslipidemia	33 (55)	19 (63)	0.451
CKD	17 (28)	15 (50)	0.043 *
Congestion score (points)	4 ± 2	5 ± 2	0.119
Pleural effusion	31 (52)	23 (77)	0.022 *
Ascites	3 (5)	6 (20)	0.020 *
Inferior vena cava distension (mm)	22.4 ± 4.3	22.8 ± 4.8	0.693
VExUS grade 0–1	31 (59)	14 (50)	0.526
VExUS grade 2–3	21 (41)	14 (50)	0.526
Uric acid (mg/dL)	7.0 ± 3.8	7.2 ± 3.5	0.466
Urea (mg/dL)	66.7 ± 34	75.3 ± 23	0.223
Creatinine (mg/dL)	1.3 ± 0.5	1.5 ± 0.5	0.077
eGFR (mL/min/1.73 m^2)	52 ± 29	44 ± 24	0.270
Sodium (mmol/L)	141 ± 3.5	140 ± 5.1	0.097
Potassium (mmol/L)	4.02 ± 0.4	4.31 ± 0.9	0.056
Chloride (mmol/L)	100 ± 5	101 ± 6	0.820
Creatinine in urine (mg/dL)	37.5 ± 28.5	33.7 ± 21.3	0.564
Chloride in urine (mg/dL)	86 ± 59	87 ± 63	0.960
Sodium in urine (mg/dL)	93.5 ± 48.5	88 ± 61	0.438
Potassium in urine (mg/dL)	25.1 ± 10.1	27.4 ± 11.3	0.354
Urea in urine (mg/dL)	522 ± 412	536 ± 470	0.883
ACR in urine (mg/g)	92 (RIC: 237)	121 (RIC: 236)	0.406
NTproBNP (ng/dL)	4665 (RIC: 5662)	11,278 (RIC: 20,179)	<0.001 *
Ca 125 (U/mL)	37.3 (RIC: 51.6)	70.6 (RIC: 120)	0.032 *
Hemoglobin (g/dL)	12.4 ± 2	12 ± 2.4	0.368
Hematocrit (%)	38.2 ± 6.1	36.3 ± 7.5	0.215

Categorical variables are shown as number of patients (n) and percentage of total (%). Units are given for quantitative variables. * Variables with *p*-values <0.05 indicate significant variation between HF phenotypes.

There were no significant differences in intravenous furosemide doses or the use of hypertonic saline. However, higher VExUS scores correlated with a greater use of

intravenous diuretics. At discharge, guideline-directed medical therapy was underutilized, particularly in HFpEF patients.

There were no significant differences in in-hospital mortality (5% HFpEF vs. 10% HFrEF, $p = 0.370$), 3-month cardiovascular mortality (8% vs. 14%, $p = 0.514$), or all-cause readmission (14% vs. 26%, $p = 0.274$). Diuretic rescue was rare in both groups.

4. Discussion

In this single-center cohort study of patients hospitalized with AHF, the quantification of venous congestion at admission using the VExUS protocol showed no significant differences between those with HFpEF and those with HFrEF. Contrary to our initial hypothesis, this suggests that the VExUS score may serve as a reliable tool for assessing systemic congestion across the full spectrum of heart failure phenotypes. These findings are aligned with recent evidence highlighting the utility of multimodal congestion assessment irrespective of LVEF and support the broader clinical applicability of the VExUS protocol.

The baseline characteristics of our cohort mirrored those reported in national and international HF registries [24], with a predominance of HFpEF patients. Atrial fibrillation and anticoagulation therapy were more frequent in this group, in agreement with previous studies [24–26]. CKD was more prevalent in the HFrEF group, despite being typically associated with HFpEF [26]. Severe valvular disease and implantable cardioverter-defibrillators were also more common in HFrEF, reflecting the advanced structural damage characteristic of this phenotype and its specific therapeutic indications [27].

Regarding the degree of congestion assessed by physical examination, no significant differences were found between groups; however, tissue edema appeared more frequently in patients with reduced ejection fraction (HFrEF). This group also showed a higher prevalence of pleural effusion and ascites, likely reflecting volume overload-driven decompensation—typical of HFrEF—even though a mixed mechanism involving both vascular redistribution and volume expansion is increasingly recognized [28].

Van Aelst et al. [29] reported similar findings, noting that both the preserved (HFpEF) and reduced ejection fraction groups showed comparable levels of venous congestion in acute heart failure, despite the higher natriuretic peptide levels seen in HFrEF patients. Consistently, our study found increased levels of both intravascular (NT-proBNP) and tissue congestion markers (CA125) in HFrEF patients. However, recent studies have reported elevated CA125 levels in HFpEF patients compared to controls, suggesting a potential role for CA125 as a marker of congestion and prognosis in HFpEF [30,31]. The discrepancies between our findings and those of these studies may be attributed to differences in sample size, patient characteristics, or methodological approaches. These variations underscore the complexity of heart failure phenotyping and the need for further research to clarify the role of CA125 in HFpEF. Additionally, VExUS, as a direct ultrasound-based tool for assessing venous congestion, may provide complementary information to biomarker profiles, potentially enhancing our understanding of congestion pathways in HFpEF and HFrEF.

A recent meta-analysis by Palazzuoli et al. [32] emphasized the high prevalence of clinical and ultrasound signs of congestion across the heart failure spectrum, highlighting the importance of a multimodal approach to improve risk stratification. Our findings align with this conclusion, showing no differences in sonographic congestion patterns, which reinforces its value in tailoring therapy.

While physical signs of congestion were similarly distributed, pleural effusion and ascites were significantly more frequent in HFrEF, consistent with its greater volume overload tendency [28]. Nonetheless, no differences were observed in VExUS grading, supporting the findings from Van Aelst et al. [29] and a recent meta-analysis by Palazzuoli et al. [32], which emphasized the high prevalence and prognostic value of congestion

markers regardless of LVEF. Our data reinforce these conclusions and advocate for VExUS as a complementary tool in bedside evaluation.

Although some subgroup analyses, such as that of Bhardwaj et al. [33], suggest that VExUS-based congestion grading is independent of LVEF, further dedicated studies are needed—underscoring the relevance and future direction of our work.

Diuretic and hypertonic saline use did not differ by LVEF; higher VExUS scores correlated with increased intravenous diuretic requirements. This highlights its potential utility for tailoring depletive therapy. Clinical outcomes such as in-hospital mortality, three-month cardiovascular mortality, and readmissions showed no significant intergroup differences, possibly due to the limited sample size and follow-up duration [29–32,34].

Finally, we identified important gaps in baseline treatment. Many patients were underprescribed guideline-directed medical therapy upon admission despite prior LVEF classification, particularly regarding the use of disease-modifying therapies such as iSGLT2 inhibitors. This is concerning, considering current ESC guidelines which recommend these agents for both HFpEF and HFrEF patients [27]. Recent national data (RICA2 registry) showed a prescription rate of 68% for iSGLT2 inhibitors [35], highlighting a disparity that warrants further exploration.

The strengths of this study include its prospective design and comprehensive congestion assessment using multimodal tools. Limitations include the single-center setting, modest sample size, and the lack of long-term follow-up. Further multicenter studies are warranted to confirm these findings and explore the prognostic value of VExUS in diverse HF populations.

In summary, while HF phenotypes differ in traditional markers of congestion, VExUS provides consistent assessment across groups, supporting its utility as a universal tool for guiding therapy in AHF.

Study Limitations

This study has several limitations that should be acknowledged. First, it is a single-center observational study with a limited sample size, particularly within subgroups such as those with HFrEF, which may reduce statistical power and increase the risk of Type II error. As this was an exploratory study, no formal power calculation was performed. Second, challenges inherent to ultrasonographic interpretation exist: for example, IVC diameter can be elevated in the absence of true congestion due to factors like low body mass index, athletic conditioning, pulmonary hypertension, or severe tricuspid regurgitation. Additionally, other non-congestive factors such as liver disease, anemia, and obesity may also influence VExUS measurements and biomarker levels, potentially confounding their interpretation. Furthermore, while patients with advanced chronic kidney disease (CKD stages 4–5), dialysis dependence, or renal transplant were excluded, individuals with mild-to-moderate CKD (stages 1–3) or subclinical liver dysfunction were included, potentially confounding VExUS interpretation due to altered venous Doppler waveforms independent of volume status.

Ultrasound examinations were performed using two portable devices (Lumify® and Omega®), with Lumify® generally providing lower image quality. However, scans were conducted by trained sonographers using a standardized VExUS protocol, and reproducibility was substantial (Cohen's kappa 0.80), supporting consistent scoring despite device differences. Some variability related to device differences cannot be excluded.

Given these factors, the ultrasound findings must be interpreted cautiously within the broader clinical context, underscoring the importance of individualized assessment when guiding decongestive therapy. Future multicenter studies with larger samples are warranted to validate these findings and explore their prognostic significance.

5. Conclusions

In this study, no significant differences were found in the ultrasonographic assessment of venous congestion using the VExUS protocol between patients with acute heart failure (AHF) and preserved (HFpEF) versus reduced ejection fraction (HFrEF), despite notable differences in biomarkers and clinical signs of congestion. These findings support the applicability of the VExUS protocol as an objective and standardized tool for evaluating systemic congestion in AHF patients, regardless of LVEF phenotype. Its implementation may contribute to improved risk stratification and a more precise tailoring of decongestive therapy.

VExUS may represent a valuable addition to the clinical toolkit for the personalized management of acute heart failure.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/app15158157/s1, VExUS Scoring Reproducibility analysis.

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Institutional Review Board Statement: This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Aragón (CEICA) (protocol code COL21/003; date of approval: February 2023).

Informed Consent Statement: Informed consent was obtained from all subjects involved in this study.

Data Availability Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request. Due to privacy and ethical restrictions, raw data are not publicly available.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

HF	Heart failure
LVEF	Left ventricular ejection fraction
VExUS	Venous Excess Ultrasound
HFpEF	Heart failure with preserved ejection fraction
HFrEF	Heart failure with reduced ejection fraction
CA125	Carbohydrate antigen 125
NTproBNP	N-terminal pro-B-type natriuretic peptide
POCUS	Point-of-care ultrasonography
LUS	Lung ultrasound
IVC	Inferior vena cava
AHF	Acute heart failure
CKD	Chronic kidney disease
NYHA	New York Heart Association

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