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Research Article

Integrated Echocardiographic Phenotyping of Cirrhotic Cardiomyopathy: A Severity-Dependent Multi-Domain Analysis

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Abstract

Background: Cirrhotic cardiomyopathy is a recognized complication of chronic liver disease characterized by subclinical myocardial dysfunction despite preserved conventional systolic parameters. Advanced echocardiographic techniques may improve the identification of cardiovascular involvement and clarify its relationship with liver disease severity.

Objectives: To characterize cardiac involvement in cirrhosis using an integrated multi-domain echocardiographic approach and evaluate its association with liver disease severity assessed by MELD-Na score.

Methods: This cross-sectional observational study included 50 patients with established cirrhosis and 50 age- and sex-matched healthy controls. Comprehensive transthoracic echocardiography, including speckle-tracking strain imaging, diastolic function assessment, atrial mechanics, and right ventricular–pulmonary coupling evaluation, was performed. Patients with cirrhosis were stratified according to MELD-Na categories (≤9, 10–19, ≥20). Multivariable regression analyses adjusted for age, heart rate, mean arterial pressure, and hemoglobin were performed.

Results: Left ventricular ejection fraction was preserved in patients with cirrhosis; however, left ventricular global longitudinal strain was significantly reduced compared with controls (−17.4% *vs* −20.1%), as was right ventricular free-wall strain (−18.2% *vs* −22.0%). Left atrial reservoir strain was lower in cirrhotic patients (26.1% *vs* 34.2%), while average E/e' was higher (11.9 *vs* 8.5). Across increasing MELD-Na categories, there was a progressive worsening of left ventricular global longitudinal strain (−18.8%, −17.2%, −15.9%), right ventricular free-wall strain (−20.1%, −18.0%, −16.2%), left atrial reservoir strain (30.4%, 25.8%, 21.6%), and TAPSE/PASP ratio (0.71, 0.55, 0.41), accompanied by rising E/e' (9.6, 12.1, 14.3). On multivariable linear regression analysis, increasing MELD-Na score remained independently associated with worsening LV global longitudinal strain (β = 0.28, p = 0.002), impaired RV free-wall strain (β = 0.24, p = 0.004), reduced left atrial reservoir strain (β = −0.31, p = 0.003), lower TAPSE/PASP ratio (β = −0.015, p = 0.008), and higher E/e' ratio (β = 0.19, p = 0.005) after adjustment for age, heart rate, mean arterial pressure, and hemoglobin level.

Conclusions: Cirrhosis is associated with a severity-dependent multi-domain cardiovascular phenotype involving ventricular mechanics, diastolic abnormalities, atrial dysfunction, and impaired RV–pulmonary interaction despite preserved conventional systolic function. Integrated echocardiographic phenotyping may improve cardiovascular assessment in advanced liver disease.

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Introduction

Cirrhosis is a systemic disorder associated with profound hemodynamic and neurohumoral alterations extending beyond hepatic dysfunction. The hyperdynamic circulatory state characteristic of advanced cirrhosis is associated with increased cardiac output, reduced systemic vascular resistance, and altered ventricular loading conditions. Despite these abnormalities, conventional systolic parameters such as Left Ventricular Ejection Fraction (LVEF) often remain preserved, potentially masking underlying myocardial dysfunction [1, 2].

Cirrhotic cardiomyopathy is traditionally characterized by impaired cardiac contractile responsiveness, diastolic dysfunction, and electrophysiological abnormalities in the absence of known structural heart disease [3]. However, increasing evidence suggests that cardiovascular involvement in cirrhosis extends beyond isolated systolic or diastolic dysfunction and includes abnormalities in myocardial deformation, atrial mechanics, and right ventricular–pulmonary coupling [4,5].

Speckle-tracking echocardiography has emerged as a sensitive technique for detecting subclinical myocardial dysfunction in cirrhosis. Global Longitudinal Strain (GLS) can identify subtle impairment despite preserved ejection fraction and may correlate with liver disease severity [6,7]. Nevertheless, strain measurements are influenced by loading conditions and therefore require careful interpretation within the altered hemodynamic environment of cirrhosis.

The etiology of cirrhosis may additionally influence cardiovascular phenotype. Alcohol-related liver disease may overlap with alcohol-induced myocardial dysfunction, whereas Metabolic dysfunction-Associated Steatotic Liver Disease (MASLD) and viral hepatitis may contribute through systemic inflammation and metabolic dysregulation. We hypothesized that cirrhotic cardiomyopathy represents a multi-domain echocardiographic phenotype that progresses in parallel with liver disease severity. Therefore, integrated cardiovascular phenotyping in cirrhosis requires assessment across multiple echocardiographic domains.

The present study aimed to evaluate ventricular mechanics, diastolic function, atrial performance, and RV–pulmonary interaction in patients with cirrhosis and to examine their relationship with liver disease severity.

Methods

Study design and population

This cross-sectional observational study was conducted at a tertiary care academic center after institutional ethics committee approval. Consecutive adult patients with established cirrhosis were prospectively enrolled. Age- and sex-matched healthy controls without evidence of liver disease or structural cardiovascular disease were included for comparison. Cirrhosis was diagnosed based on clinical, biochemical, ultrasonographic, and endoscopic findings.

Patients with known ischemic heart disease, prior myocardial infarction, reduced LVEF (<55%), congenital heart disease, primary cardiomyopathy, significant valvular heart disease, significant tricuspid regurgitation, significant pericardial effusion, persistent atrial fibrillation/flutter, bundle branch block, chronic lung disease, active sepsis, and those with poor echocardiographic windows were excluded.

Clinical assessment

Detailed demographic, clinical, and biochemical data were systematically recorded for all study participants at the time of enrollment. Demographic assessment included age and sex distribution. Clinical evaluation comprised assessment of liver disease etiology, Child–Turcotte–Pugh (CTP) classification, MELD-Na score, resting heart rate, systolic and diastolic blood pressure, and calculation of mean arterial pressure. Laboratory investigations included hemoglobin level, serum creatinine, serum sodium, serum bilirubin, and International Normalized Ratio (INR).

The etiology of cirrhosis was categorized into alcohol-related liver disease, chronic viral hepatitis, Metabolic dysfunction-Associated Steatotic Liver Disease (MASLD), autoimmune liver disease, and cryptogenic cirrhosis. Patients were further stratified according to MELD-Na severity categories for subgroup analysis of echocardiographic parameters and cardiovascular involvement.

Echocardiographics assessment

A comprehensive transthoracic echocardiography was performed using the GE Vivid E95 ultrasound platform equipped with phased-array transducers. Image acquisition and analysis were carried out in accordance with the recommendations of the American Society of Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI) [7]. Standard parasternal long-axis, short-axis, apical four-chamber, two-chamber, and three-chamber views were acquired for all participants.

Conventional echocardiographic measurements, including chamber dimensions, ventricular volumes, wall thickness, Left Ventricular Ejection Fraction (LVEF), Doppler assessment, tissue Doppler imaging, and pulmonary artery systolic pressure estimation were obtained using the integrated vendor platform software available on the GE Vivid E95 system.

Two-dimensional speckle-tracking strain analysis was performed offline using EchoPAC software (GE Healthcare), which was available with the echocardiographic platform. Left Ventricular Global Longitudinal Strain (LVGLS) analysis was derived from apical four-chamber, two-chamber, and three-chamber views using automated function imaging with manual adjustment of endocardial borders where required.

Right ventricular free-wall strain analysis was performed from RV-focused apical four-chamber views using dedicated right ventricular strain analysis modules within EchoPAC software. Only RV free-wall segments were included in strain calculations to minimize septal influence.

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Left atrial and right atrial reservoir strain analyses were performed using dedicated atrial strain analysis software modules integrated within the EchoPAC platform. Atrial strain measurements were obtained using ECG-gated apical four-chamber acquisitions, and reservoir strain values were calculated during ventricular systole.

For deformation imaging, frame rates between 50 and 80 frames/s were maintained to optimize speckle-tracking quality. Three consecutive cardiac cycles were acquired during quiet respiration and averaged for final analysis. All measurements were performed offline by experienced echocardiographers blinded to clinical and laboratory data.

Reproducibility analysis

Intra-observer and inter-observer variability analyses were performed in a randomly selected subgroup. Measurements were repeated by independent blinded observers and assessed using intraclass correlation coefficients.

Statistical analysis

Continuous variables are expressed as mean ± standard deviation. Comparisons between groups were performed using Student's t-test or Mann–Whitney U test where appropriate. Comparisons across MELD-Na categories were performed using ANOVA or Kruskal-Wallis testing. Multivariable linear regression analyses adjusted for age, heart rate, mean arterial pressure, and hemoglobin level were performed to evaluate the independent association between MELD-Na score and echocardiographic abnormalities. A p-value <0.05 was considered statistically significant.

Results

Patient characteristics

Baseline characteristics are summarized in Table 1. Cirrhotic patients exhibited lower systemic blood pressure, higher resting heart rates, anemia, and a wide distribution of MELD-Na scores, allowing severity-based analysis.

Multi-domain echocardiographic abnormalities

A summary of ventricular, atrial, right ventricular-pulmonary, and diastolic parameters is presented in Tables 2 and 3. The integrated echocardiographic domains assessed in this study and their relationship with liver disease severity are summarized schematically in Figure 1. Although left ventricular ejection fraction was preserved, patients with cirrhosis exhibited significantly impaired left ventricular global longitudinal strain and right ventricular free-wall strain compared with controls (Figure 2). Right ventricular-pulmonary coupling, assessed by TAPSE/PASP ratio, was significantly reduced, indicating early RV-pulmonary interaction abnormalities.

Atrial function analysis revealed impaired left and right atrial reservoir strain, often preceding marked atrial enlargement. Diastolic assessment demonstrated reduced mitral annular e′ velocities, elevated E/e′ ratios, and a high prevalence of grade I or indeterminate diastolic dysfunction. Left atrial reservoir

Table 1: Baseline clinical characteristics of the study population.

Variable	Cirrhosis (n = 50)	Controls (n = 50)	*p* value
Age, years	48.6 ± 9.1	47.9 ± 8.7	0.68
Male sex, n (%)	34 (68)	33 (66)	0.84
Heart rate, beats/min	86.9 ± 12.5	72.8 ± 9.4	<0.001
Mean arterial pressure, mmHg	79.1 ± 10.2	91.6 ± 9.8	<0.001
Hemoglobin, g/dL	9.7 ± 1.8	13.3 ± 1.1	<0.001
Ascites present, n (%)	28 (56)	–	–
MELD-Na score	16.8 ± 7.1	–	–

Table 2: Ventricular mechanics and right ventricular–pulmonary interaction.

Parameter	Cirrhosis	Controls	*p* value
LVEF, %	61.4 ± 5.2	62.1 ± 4.9	0.42
LV global longitudinal strain, %	−17.4 ± 2.6	−20.1 ± 2.4	<0.001
RV free-wall strain, %	−18.2 ± 3.1	−22.0 ± 3.0	<0.001
TAPSE, mm	18.9 ± 3.2	22.1 ± 3.1	<0.001
PASP, mmHg	33.6 ± 7.4	25.8 ± 5.6	<0.001
TAPSE/PASP ratio, mm/ mmHg	0.56 ± 0.15	0.86 ± 0.19	<0.001

Demonstrates subclinical systolic impairment and early RV–pulmonary uncoupling, despite preserved EF.

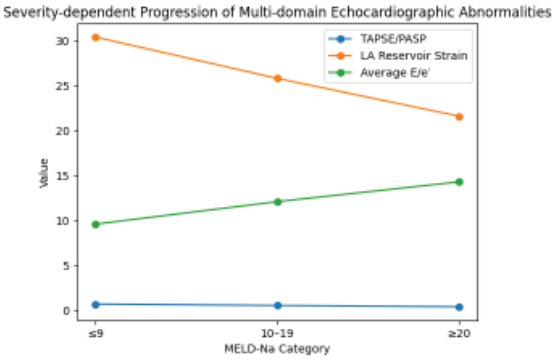

Figure 1: Integrated multi-domain echocardiographic abnormalities across MELD-Na strata. With worsening liver disease severity, there was a progressive reduction in right ventricular–pulmonary coupling (TAPSE/PASP ratio) and atrial reservoir function, accompanied by rising left ventricular filling pressures (E/e'), supporting a severity-dependent, multi-domain cardiovascular phenotype in cirrhosis.

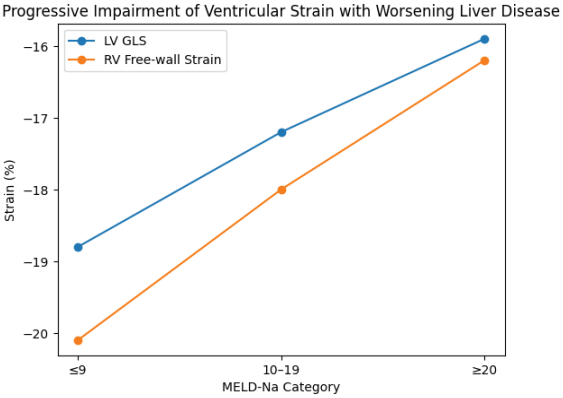

Figure 2: Progressive worsening of left ventricular global longitudinal strain and right ventricular free-wall strain across increasing MELD-Na categories. Despite preserved left ventricular ejection fraction, both left and right ventricular myocardial deformation deteriorated with advancing liver disease severity, indicating a severity-dependent subclinical systolic impairment.

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strain was significantly reduced and average E/e′ was higher in patients with cirrhosis compared with controls, indicating impaired atrial compliance and elevated filling pressures (Figure 3).

Integrated phenotype and liver disease severity

When echocardiographic abnormalities were analyzed across MELD-Na categories, a clear severity-dependent clustering emerged (Table 4). Patients with lower MELD-Na scores demonstrated isolated abnormalities in myocardial strain or atrial mechanics, whereas those with advanced liver disease exhibited concurrent involvement across multiple domains, including ventricular mechanics, atrial dysfunction, RV-pulmonary interaction, and diastolic abnormalities. An integrated heatmap analysis demonstrated a graded, severity-dependent clustering of echocardiographic abnormalities across ventricular, atrial, diastolic, and RV-pulmonary domains (Figure 4).

Multivariable regression analysis

On multivariable linear regression analysis adjusted for age, heart rate, mean arterial pressure, and hemoglobin level, increasing MELD-Na score remained independently associated with worsening myocardial deformation and diastolic functional abnormalities (Table 5). Higher MELD-Na scores demonstrated a significant independent association with impaired left ventricular global longitudinal strain (β = 0.28, 95% CI: 0.11 to 0.44, p = 0.002) and right ventricular free-wall strain (β = 0.24, 95% CI: 0.08 to 0.39, p = 0.004).

Similarly, worsening liver disease severity was independently associated with reduced left atrial reservoir strain (β = −0.31, 95% CI: −0.52 to −0.10, p = 0.003) and impaired RV-pulmonary arterial coupling as reflected by lower TAPSE/

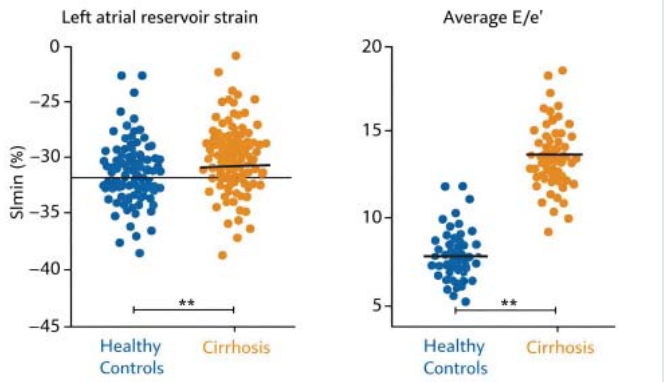

Figure 3: This figure compares left atrial (LA) reservoir strain and average E/e' ratio between healthy controls and patients with cirrhosis, using distinct colors to differentiate the two groups.Left panel – Left atrial reservoir strain (%): Healthy controls (blue) demonstrate higher (more negative) LA reservoir strain values, indicating preserved atrial compliance and reservoir function. In contrast, patients with cirrhosis (orange) show a significant reduction in LA reservoir strain, reflecting impaired atrial deformation and reduced atrial compliance. This finding is consistent with early structural and functional atrial involvement in cirrhotic cardiomyopathy. Right panel – Average E/e' ratio: Healthy controls (blue) exhibit lower E/e' ratios, consistent with normal left ventricular filling pressures. Patients with cirrhosis (orange) show significantly higher E/e' values, indicating elevated left ventricular filling pressures and impaired diastolic relaxation.

Table 3: Atrial and diastolic echocardiographic parameters.

Parameter	Cirrhosis	Controls	*p* value
Left atrial volume index, mL/m²	36.4 ± 8.2	28.7 ± 6.0	<0.001
LA reservoir strain, %	26.1 ± 6.5	34.2 ± 7.0	<0.001
RA reservoir strain, %	29.4 ± 7.1	38.0 ± 7.6	<0.001
Septal e', cm/s	6.0 ± 1.4	8.4 ± 1.6	<0.001
Lateral e', cm/s	8.1 ± 1.7	10.7 ± 2.0	<0.001
Average E/e'	11.9 ± 3.1	8.5 ± 2.0	<0.001

This table anchors the HFpEF-like diastolic phenotype and atrial dysfunction.

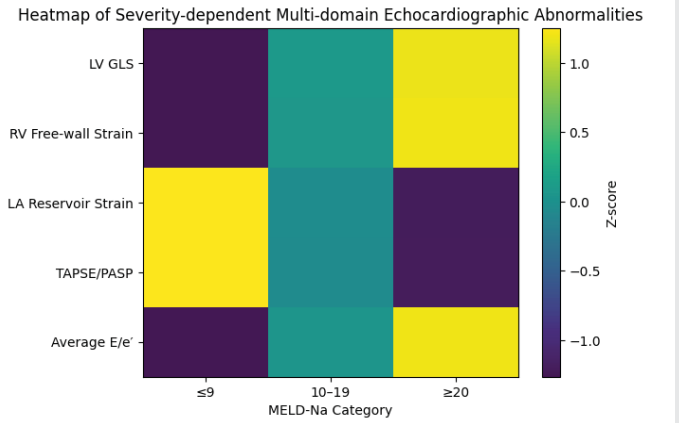

Figure 4: Heat map illustrating multi-domain cardiovascular involvement in cirrhosis. Legend: Heatmap depicting severity-dependent multi-domain echocardiographic abnormalities across MELD-Na categories. Values are expressed as row-wise z-scores to allow comparison across echocardiographic domains. Progressive worsening of ventricular deformation, atrial reservoir function, right ventricular–pulmonary coupling, and left ventricular filling pressures is observed with increasing liver disease severisty, highlighting the integrated and continuous nature of cirrhotic cardiomyopathy.

Table 4: Integrated echocardiographic phenotype across MELD-Na categories.

Parameter	MELD-Na ≤9 (n=22)	MELD-Na 10–19 (n=12)	MELD-Na ≥20 (n=16)	*p* for trend
LV GLS, %	−18.8 ± 2.1	−17.2 ± 2.3	−15.9 ± 2.6	<0.01
RV free-wall strain, %	−20.1 ± 2.8	−18.0 ± 2.9	−16.2 ± 3.1	<0.01
TAPSE/PASP ratio	0.71 ± 0.16	0.55 ± 0.14	0.41 ± 0.12	<0.001
LA reservoir strain, %	30.4 ± 5.9	25.8 ± 5.4	21.6 ± 5.1	<0.001
Average E/e'	9.6 ± 2.1	12.1 ± 2.6	14.3 ± 3.0	<0.001
≥2 domains abnormal, n(%)	5 (23)	7 (58)	13 (81)	<0.001

This table visually proves that cardiac involvement progresses with liver.

Table 5: Multivariable linear regression analysis.

Dependent Variable	β Coefficient	95% Confidence Interval	Adjusted *P* value
LV GLS	0.28	0.11 to 0.44	0.002
RV Free-Wall Strain	0.24	0.08 to 0.39	0.004
LA Reservoir Strain	−0.31	−0.52 to −0.10	0.003
TAPSE/PASP Ratio	−0.015	−0.026 to −0.004	0.008
Average E/e'	0.19	0.07 to 0.31	0.005

Adjusted covariates: age, heart rate, mean arterial pressure, and hemoglobin level. MELD-Na score remained independently associated with worsening ventricular deformation and elevated filling indices after adjustment.

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PASP ratio (β = −0.015, 95% CI: −0.026 to −0.004, p = 0.008). In addition, MELD-Na score remained significantly associated with a higher average E/e′ ratio (β = 0.19, 95% CI: 0.07 to 0.31, p = 0.005), suggesting progressive worsening in filling dynamics with advancing hepatic dysfunction.

These findings indicate that the observed abnormalities in ventricular mechanics, atrial function, and diastolic indices persisted even after adjustment for baseline hemodynamic and hematologic differences, supporting an independent relationship between liver disease severity and cardiovascular dysfunction.

Discussion

Cirrhosis is increasingly recognized as a multisystem disorder in which cardiovascular dysfunction plays a central but often underappreciated role. The hyperdynamic circulatory state of cirrhosis, characterized by increased cardiac output, reduced systemic vascular resistance, and chronic plasma volume expansion, has been well documented for decades [1,2]. Despite these profound hemodynamic changes, cardiac involvement frequently remains clinically silent due to preserved resting systolic function, leading to underdiagnosis of cirrhotic cardiomyopathy in routine practice.

The present study demonstrates that cirrhotic cardiomyopathy is best understood not as a single functional abnormality but as a multi-domain cardiovascular phenotype involving ventricular mechanics, atrial function, right ventricular–pulmonary interaction, and diastolic reserve. By integrating echocardiographic findings across these domains, we show that cardiac involvement in cirrhosis is widespread, severity-dependent, and detectable even in the absence of overt heart failure or reduced ejection fraction.

Pathophysiological basis of multi-domain cardiac involvement in cirrhosis

The cardiovascular abnormalities observed in cirrhosis arise from a complex interplay of neurohumoral activation, systemic inflammation, myocardial remodeling, and altered loading conditions. Chronic activation of the sympathetic nervous system and renin-angiotensin-aldosterone system, along with increased circulating cytokines and nitric oxide, leads to myocardial hypertrophy, interstitial fibrosis, and impaired excitation-contraction coupling [2-4]. These changes collectively impair myocardial reserve while preserving resting systolic indices. Our findings of impaired ventricular strain despite preserved ejection fraction support prior experimental and clinical observations that myocardial contractility in cirrhosis is intrinsically abnormal and becomes apparent only with sensitive techniques or physiological stress [3,5].

Importantly, the involvement of both left and right ventricular mechanics underscores the global nature of myocardial dysfunction in cirrhosis rather than isolated left-sided disease.

Alcohol-related cirrhosis demonstrated relatively greater ventricular deformation abnormalities. This finding is clinically important because chronic alcohol exposure itself may contribute to myocardial remodeling and systolic dysfunction. However, similar abnormalities were also observed in non-alcoholic etiologies including viral hepatitis and MASLD, supporting the broader concept of cirrhotic cardiomyopathy beyond isolated alcohol-mediated myocardial injury.

Right ventricular-pulmonary interaction: Relevance to portal hypertension

Right ventricular-pulmonary coupling represents a particularly relevant domain for hepatologists, given the close relationship between portal hypertension, pulmonary vascular changes, and right heart function. Portopulmonary hypertension and subclinical increases in pulmonary vascular resistance are well-described complications of advanced liver disease [6]. In this study, reduced TAPSE/PASP ratios suggest early impairment in right ventricular–pulmonary interaction, even in patients without overt pulmonary hypertension.

This finding has important clinical implications, as right ventricular dysfunction is a key determinant of outcomes during liver transplantation and in critically ill cirrhotic patients [7,8]. Early identification of impaired RV-pulmonary coupling may therefore enhance cardiovascular risk stratification in advanced liver disease.

Relevance of global longitudinal strain in cirrhosis

In the present study, patients with cirrhosis demonstrated significantly impaired left ventricular Global Longitudinal Strain (GLS) despite preserved conventional systolic function. This finding highlights the potential utility of myocardial deformation imaging for detecting subclinical cardiovascular involvement that may not be apparent on routine ejection fraction assessment. GLS has emerged as a sensitive marker of early myocardial dysfunction and may identify subtle abnormalities in longitudinal fiber mechanics before overt systolic impairment becomes clinically evident [6,9].

However, interpretation of GLS in cirrhosis requires careful consideration of the unique hemodynamic milieu associated with chronic liver disease. Myocardial deformation parameters are influenced by preload, afterload, systemic vascular resistance, and hyperdynamic circulation, all of which are frequently altered in advanced cirrhosis. Consequently, impaired GLS should not be interpreted solely as evidence of intrinsic myocardial contractile dysfunction. Rather, strain abnormalities likely reflect a complex interaction between altered loading conditions, neurohumoral activation, endothelial dysfunction, and myocardial structural remodeling [1,4].

Importantly, the persistence of GLS abnormalities after multivariable adjustment for age, heart rate, mean arterial pressure, and hemoglobin level, along with their progressive worsening across increasing MELD-Na categories, supports the presence of clinically relevant cardiovascular involvement accompanying advanced liver disease. These findings suggest that GLS may serve as a valuable adjunctive marker

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for identifying early cardiac dysfunction and improving cardiovascular risk stratification in patients with cirrhosis [3,6].

Atrial dysfunction as an integrative marker of chronic circulatory stress

Atrial remodelling and dysfunction reflect the cumulative burden of chronic volume overload and elevated filling pressures. In our cohort, both left and right atrial strain were impaired, often preceding marked atrial enlargement. Atrial strain provides a sensitive marker of reduced atrial compliance and reservoir function and integrates information from ventricular diastolic properties, volume status, and pulmonary pressures [9,10].

From a hepatology perspective, atrial dysfunction may help explain the propensity of cirrhotic patients to develop arrhythmias, exercise intolerance, and hemodynamic instability during acute illness. Importantly, atrial dysfunction represents a potentially reversible stage of myocardial involvement, particularly in the context of liver transplantation, as suggested by prior studies demonstrating partial normalization of cardiac abnormalities after transplantation [11].

HFpEF-like diastolic phenotype in cirrhosis

Although LVEF remained preserved, significant impairment in GLS and diastolic parameters was observed. This highlights the limitations of relying solely on conventional systolic indices in cirrhosis. Diastolic dysfunction has long been considered a central feature of cirrhotic cardiomyopathy [3]. However, its clinical recognition remains challenging due to the confounding effects of hyperdynamic circulation and variable loading conditions. In this study, the combination of impaired relaxation, elevated filling pressure surrogates, atrial dysfunction, and preserved systolic function closely mirrors the pathophysiological substrate of Heart Failure with preserved Ejection Fraction (HFpEF).

We deliberately describe this entity as a HFpEF-like phenotype, rather than diagnosing HFpEF, to avoid misclassification, since the present study did not include NT-proBNP assessment, invasive hemodynamic evaluation, exercise testing, or direct validation of filling pressures. Therefore, the term "HFpEF-like phenotype" should be interpreted only as a conceptual echocardiographic framework rather than a definitive clinical diagnosis. This conceptual framework is particularly useful for hepatologists, as it explains why cirrhotic patients often tolerate resting conditions but decompensate during stressors such as infection, aggressive fluid resuscitation, transjugular intrahepatic portosystemic shunt placement, or surgery [6,12]. Recognition of this phenotype emphasizes the importance of cautious volume management and cardiovascular monitoring in advanced liver disease.

Severity-dependent progression and clinical relevance

A key strength of the present study is the demonstration of a graded relationship between echocardiographic abnormalities and liver disease severity, as assessed by MELD-Na score. Patients with early cirrhosis exhibited isolated abnormalities in myocardial strain or atrial mechanics, whereas those with advanced disease demonstrated concurrent involvement across multiple cardiovascular domains. This severity-linked progression supports the concept that cirrhotic cardiomyopathy evolves in parallel with hepatic dysfunction rather than representing a static complication [13,14].

For hepatologists, this finding reinforces the importance of cardiovascular assessment as liver disease advances. Multi-domain echocardiographic phenotyping may provide valuable information during transplant evaluation, risk stratification, and longitudinal follow-up.

Clinical implications for hepatology practice

From a practical standpoint, our findings suggest that reliance on left ventricular ejection fraction alone is insufficient for cardiovascular assessment in cirrhosis. Comprehensive echocardiography incorporating strain imaging, atrial function, and right ventricular-pulmonary interaction offers a more accurate depiction of cardiac involvement. This integrated approach may help identify patients at higher risk of cardiovascular decompensation during acute illness or invasive interventions.

Limitations

This study is limited by its cross-sectional design and lack of outcome data. Invasive hemodynamic validation was not performed. While strain parameters are load-dependent, consistent trends across multiple domains and severity strata support the robustness of the findings. Invasive hemodynamic validation, natriuretic peptide assessment, and exercise testing were not performed. The contribution of alcohol-related myocardial dysfunction cannot be completely excluded. Prospective studies are needed to determine the prognostic value of this integrated phenotype and its reversibility following liver transplantation.

Conclusions

Cirrhotic cardiomyopathy represents a multi-domain cardiovascular phenotype involving ventricular mechanics, atrial dysfunction, right ventricular-pulmonary interaction, and diastolic reserve. For the practicing hepatologist, these findings emphasize that cardiac dysfunction in cirrhosis is often subclinical, multifactorial, and progressive, and may not be captured by conventional echocardiographic parameters alone. Recognition of this phenotype provides a comprehensive framework for cardiovascular evaluation in patients with cirrhosis and may inform clinical decision-making in advanced liver disease.

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Author contributions

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- Data curation: Sameer Purra, Sahanawaz Shah,
- Formal analysis:, Sheikh Jan Mohammad, Qayoom Yousuf
- Investigation: Sameer Purra, Sheikh Jan Mohammad
- Writing – original draft: Sameer Purra, Shahnawaz Shah,
- Writing – review & editing: Sheikh Jan Mohammad, Qayoom Yousuf
- All authors approved the final manuscript.

Ethical approval

The study protocol was reviewed and approved by the Institutional Ethics Committee of Sher-i-Kashmir Institute of Medical Sciences, Srinagar (Approval No: #48/2024). The study was conducted in accordance with the Declaration of Helsinki.

Consent to participate

Written informed consent was obtained from all participants or their legally authorized representatives prior to enrollment.

Patients' consent form

Consent for participation and use of anonymized clinical data for research and publication purposes was obtained from all patients.

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