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The Importance of Liver Density on CT Scan in Heart Failure Patients

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Research Article

Keywords: Heart failure, liver density, computed tomography, prognosis, liver function

Posted Date: November 18th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11210516/v1

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Additional Declarations: No competing interests reported.

Abstract

Introduction

Heart failure (HF) is a major global health problem associated with substantial morbidity and mortality. Hepatic involvement is common in HF and may reflect the severity of systemic congestion and impaired hemodynamics. Although liver function tests and bilirubin levels have been investigated as prognostic markers in HF, the clinical significance of liver density measured by computed tomography (CT) has received less attention. This study aimed to evaluate the association between liver density on CT scans and clinical, laboratory, echocardiographic, and prognostic parameters in patients with HF.

Methods

This retrospective cross-sectional study included 145 patients with HF admitted to a tertiary heart center between 2020 and 2022. Adults with HF with reduced or mid-range ejection fraction confirmed by echocardiography were included. Demographic, clinical, laboratory, chest CT, and echocardiographic data were obtained from medical records. Statistical analyses were performed using SPSS version 24.0. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequencies. Associations were assessed using independent t-tests and chi-square tests. A p-value < 0.05 was considered statistically significant.

Results

The mean age was 42.8 ± 10.5 years, and the mean BMI was 27.06 ± 4.44 kg/m². Most patients were male. Edema and ascites were positively associated with liver density, whereas LVEDD (r = − 0.32) and SPAP (r = − 0.276) showed significant negative correlations. No significant association was observed between liver density and TAPSE (r = 0.056). Electrolytes, creatinine, and liver function tests were also associated with liver density. Patients with cardiac-related mortality had a mean liver density of 47.29 ± 7.1 HU, and this association was statistically significant (P = 0.029).

Conclusion

Liver density measured on chest CT may provide useful prognostic information in patients with HF and could serve as an additional imaging marker for clinical risk assessment.

Introduction

Heart failure (HF) is a major public health problem affecting approximately 1%–2% of adults and is associated with frequent hospitalization, impaired quality of life, and increased mortality. (1) Hepatic

involvement is common in patients with HF and may further influence disease severity, prognosis, and clinical management. (2) (Fig. 1).

The heart and liver have a complex and interdependent relationship, and systemic conditions such as alcohol misuse, certain medications, inflammation, autoimmune disorders, and infections can affect both organs (3). Liver disease may contribute to cardiac dysfunction even in the absence of other cardiovascular disorders (4, 5). Conversely, HF can cause hepatic complications through impaired cardiac output and increased systemic venous pressure. Cardiogenic liver injury, formerly referred to as “ischemic hepatitis,” is frequently observed in patients with HF who develop severe cardiogenic shock, in whom cardiac output becomes insufficient to meet the metabolic demands of the liver. (6)

Although hepatic involvement in HF is well recognized, the prognostic significance of radiologic measures of hepatic involvement remains insufficiently established. In particular, limited evidence is available regarding the clinical and prognostic relevance of liver density measured by computed tomography (CT) in patients with HF. CT provides a quantitative assessment of hepatic attenuation and may potentially reflect structural or functional hepatic alterations associated with systemic congestion and impaired hemodynamics. Therefore, further investigation is warranted to determine whether CT-derived liver density may provide clinically useful information regarding disease severity and prognosis in patients with HF. Previous studies have demonstrated that abnormalities in liver function tests (LFTs) are associated with poorer long-term outcomes in patients with chronic HF (3–5). Furthermore, markedly elevated total bilirubin levels have been associated with increased central venous pressure and reduced cardiac output (7). Abnormal liver test profiles have also been independently associated with compromised 180-day outcomes in patients with HF (8). The heart and liver have a complex and bidirectional relationship, often described as the heart–liver or cardio hepatic axis. (9–10) These findings highlight the potential importance of hepatic involvement in HF risk stratification. However, most previous investigations have focused primarily on biochemical markers, while relatively little attention has been given to radiologic parameters, particularly liver density assessed by CT.

Given the established relationship between hepatic dysfunction and adverse outcomes in HF, together with the limited evidence regarding radiologic assessment of hepatic involvement, this study aimed to investigate the association between liver density measured on CT scans and the prognosis of patients with HF. We sought to determine whether CT-derived liver density could provide additional prognostic information beyond conventional clinical and biochemical parameters in the assessment of patients with HF.

Methods

Study Design and Setting

This retrospective cross-sectional study was conducted at a tertiary cardiovascular center among adult patients admitted with heart failure (HF). A total of 145 patients were enrolled between 2020 and 2022.

Patients were followed until March 2022, which was considered the date of data censoring, or until death. Demographic, clinical, laboratory, echocardiographic, computed tomography (CT), and outcome data were obtained from hospital and clinical records. Routine non-contrast thoracic CT scans performed within six months of the clinical presentation for evaluation of HF were reviewed for assessment of hepatic parenchymal density. The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the institutional Ethics Committee.

Inclusion and Exclusion Criteria

Patients were eligible if they were aged ≥ 18 years, had been hospitalized with a diagnosis of HF, had HF with reduced or mid-range left ventricular ejection fraction, and had current or previous symptoms and clinical signs compatible with HF according to the New York Heart Association (NYHA) classification. Left ventricular dysfunction was confirmed by transthoracic echocardiography. Patients were included regardless of the underlying etiology of HF and received treatment according to established HF guidelines. Availability of the required clinical, laboratory, CT, echocardiographic, and follow-up data was also required. Patients were excluded if they had cirrhosis, cancer, thalassemia, malnutrition, or alcohol consumption, as these conditions may independently affect hepatic structure, liver density, or liver function and could confound the association between liver density and HF outcomes. Patients with incomplete clinical, laboratory, imaging, or outcome data were also excluded.

Data Collection

Demographic characteristics, medical history, clinical findings, laboratory results, CT findings, echocardiographic parameters, and clinical outcomes were collected from hospital and clinical records. Demographic variables included age, sex, weight, height, and body mass index (BMI). Medical history included diabetes mellitus, hypertension, dyslipidemia, and cigarette smoking. Laboratory variables included proBNP, albumin, uric acid, liver function tests, thyroid function tests, and creatinine, all measured by the hospital laboratory. Transthoracic echocardiographic data included left ventricular ejection fraction (LV-EF), right ventricular function, and systolic pulmonary artery pressure (SPAP). The primary outcome was all-cause mortality; cardiac-related mortality was also assessed, while re-admission was considered a secondary outcome. Patients were followed until March 2022 or until death. For patients lost to follow-up, outcome information was obtained through telephone interviews with the patients or their relatives.

Routine non-contrast thoracic CT scans obtained within six months of clinical presentation for evaluation of HF were reviewed by an experienced radiologist. Imaging was performed using a 16-slice SOMATOM Scope CT scanner (Siemens, Forchheim, Germany). Liver parenchymal density was defined as hepatic attenuation measured in Hounsfield units (HU) on non-contrast CT images. Normal liver parenchyma was considered to have an attenuation of approximately 60 HU, whereas severe hepatic steatosis was defined as liver attenuation ≤ 48 HU. Regions of interest (ROIs) were placed in both the right and left hepatic lobes while avoiding vascular structures, and the mean HU values were recorded (Fig. 2). ROIs

were positioned at the same anatomical locations across CT examinations to ensure measurement consistency. All scans were interpreted by an experienced radiologist, and inter-observer variability was assessed to evaluate the reliability of liver density measurements.

Statistical Analysis

Statistical analyses were performed using SPSS version 24.0 for Windows (SPSS Corp., Munich, Germany). The distribution of quantitative variables was assessed using the Kolmogorov–Smirnov test. Normally distributed continuous variables were compared using the independent-samples *t*-test, whereas the Mann–Whitney *U* test was used for non-normally distributed variables. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test, as appropriate. Correlation analysis was performed to evaluate the relationships between liver density and relevant clinical, laboratory, and echocardiographic variables. Logistic regression analysis was performed to identify independent predictors of mortality. All statistical tests were two-sided, and a *P*-value < 0.05 was considered statistically significant.

Results

A total of 145 patients with heart failure were included in the study. The mean age was 42.8 ± 10.5 years (range, 19–64 years), and 98 patients (67.6%) were men. The mean height, weight, and body mass index (BMI) were 169.9 ± 10.0 cm, 79.5 ± 16.7 kg, and 27.06 ± 4.44 kg/m², respectively (BMI range, 19–40 kg/m²). Regarding comorbidities, 29.7% of patients were cigarette smokers, 26.2% had diabetes mellitus, 30.3% had hypertension, 6.9% had dyslipidemia, and 23.4% had coronary artery disease. Chronic lung disease, rheumatological disease, and thyroid disease were reported in 1.4%, 0.7%, and 13.1% of patients, respectively. Clinically, dyspnea was reported in 84.8% of patients, whereas edema and ascites were present in 43.4% and 12.4%, respectively.

Echocardiographic assessment showed a mean left ventricular ejection fraction (LVEF) of 21.43 ± 12.01%. The mean left ventricular end-diastolic diameter (LVEDD), tricuspid annular plane systolic excursion (TAPSE), systolic pulmonary artery pressure (SPAP), and inferior vena cava (IVC) diameter were 6.27 ± 0.92 cm, 14.97 ± 3.87 mm, 40.99 ± 14.94 mmHg, and 19.23 ± 3.49 mm, respectively. The mean liver density measured by non-contrast CT was 51.49 ± 10.40 Hounsfield units (HU), with a range of 26–100 HU.

Liver density was significantly associated with several clinical and echocardiographic characteristics. Among the clinical variables, edema and ascites were significantly associated with liver density, with mean liver densities of 54.5 ± 9.3 HU and 52.64 ± 10.34 HU, respectively (P = 0.001 for both). No significant association was observed between dyspnea and liver density (P = 0.607). Echocardiographic analysis demonstrated a significant positive correlation between LVEF and liver density (r = 0.325, P = 0.001). In contrast, LVEDD was negatively correlated with liver density (r = − 0.320, P = 0.003). Similarly, SPAP and IVC diameter showed significant negative correlations with liver density (r = − 0.276, P = 0.002

and r = − 0.286, P = 0.001, respectively). No significant correlation was observed between TAPSE and liver density (r = 0.056, P = 0.514).

Several laboratory parameters were also significantly correlated with liver density. Negative correlations were observed with creatinine (r = − 0.214, P = 0.012), uric acid (r = − 0.186, P = 0.032), alkaline phosphatase (ALP) (r = − 0.205, P = 0.018), direct bilirubin (r = − 0.190, P = 0.026), and total bilirubin (r = − 0.296, P = 0.001). Positive correlations were observed with sodium (r = 0.317, P = 0.001), total cholesterol (r = 0.297, P = 0.001), and calcium (r = 0.178, P = 0.038). No statistically significant correlations were observed between liver density and hemoglobin, proBNP, albumin, vitamin D, alanine aminotransferase (ALT), aspartate aminotransferase (AST), international normalized ratio (INR), thyroid-stimulating hormone (TSH), iron saturation, ferritin, or blood urea nitrogen (BUN).

During follow-up, 25 patients (17.2%) died from cardiac causes, whereas 5 patients (3.4%) died from non-cardiac causes. Hospitalization occurred in 33 patients (22.8%) within 3 months and in 36 patients (24.8%) within 1 year. Liver density was significantly associated with cardiac mortality. Patients who died from cardiac causes had a mean liver density of 47.29 ± 7.1 HU, with a statistically significant association between liver density and cardiac mortality (P = 0.029). In contrast, no significant association was observed between liver density and non-cardiac mortality, for which the mean liver density was 49.0 ± 8.33 HU (P = 0.58). Similarly, liver density was not significantly associated with hospitalization within 3 months (mean liver density, 52.07 ± 9.81 HU; P = 0.243) or within 1 year (51.96 ± 9.44 HU; P = 0.340).

Overall, lower liver density was associated with markers of greater cardiac dysfunction, including lower LVEF, greater LVEDD, higher SPAP, and larger IVC diameter, and was significantly associated with cardiac mortality.

Discussion

The main finding of this study was that CT-measured liver density was significantly associated with several clinical and echocardiographic parameters reflecting heart failure (HF) severity. Liver density showed a positive correlation with left ventricular ejection fraction (LVEF) and negative correlations with left ventricular end-diastolic diameter (LVEDD), systolic pulmonary artery pressure (SPAP), and inferior vena cava (IVC) diameter. These findings suggest that lower liver density is associated with several markers of more advanced cardiac structural and hemodynamic abnormalities. However, these associations should be interpreted cautiously and do not establish a causal relationship between cardiac dysfunction and hepatic changes.

The interaction between the cardiovascular and hepatic systems, commonly referred to as the cardio hepatic relationship, is increasingly recognized in patients with HF. Reduced cardiac output and elevated systemic venous pressure may contribute to hepatic abnormalities through impaired hepatic perfusion and venous congestion. In the present study, the association of liver density with LVEF, LVEDD, SPAP, and IVC diameter supports the possibility that CT-derived hepatic changes may reflect the overall

hemodynamic burden of HF. Nevertheless, the present findings demonstrate association rather than causality and cannot determine whether reduced liver density is a consequence of cardiac dysfunction or reflects other underlying processes.

Interestingly, tricuspid annular plane systolic excursion (TAPSE), a commonly used parameter of right ventricular (RV) systolic function, was not significantly associated with liver density. This finding suggests that the observed relationship between hepatic density and HF-related abnormalities cannot be explained solely by RV systolic dysfunction. However, TAPSE represents only one aspect of RV performance and does not fully characterize RV–pulmonary artery coupling, tricuspid regurgitation, or systemic venous congestion. Therefore, the absence of an association between TAPSE and liver density should not be interpreted as evidence against a role for right-sided hemodynamics in hepatic involvement.

A further important finding was the association between liver density and clinical manifestations of congestion, particularly edema and ascites. Both edema and ascites were significantly associated with liver density, whereas dyspnea was not. Edema and ascites may reflect systemic venous congestion and increased hydrostatic pressure in patients with HF. Their association with liver density therefore provides additional support for a relationship between CT-derived hepatic changes and the hemodynamic state of patients with HF. The lack of association with dyspnea may be explained by the nonspecific nature of this symptom, which can be influenced by pulmonary, cardiac, and other systemic factors.

Liver density was also negatively correlated with total and direct bilirubin, whereas no significant associations were observed with AST or ALT. These findings suggest that CT-derived liver density and conventional biochemical markers may capture different aspects of hepatic involvement. Bilirubin levels may be particularly sensitive to hepatic congestion and impaired hepatic clearance in HF, whereas aminotransferases are more closely related to hepatocellular injury. Thus, structural or compositional hepatic changes detectable by CT may occur without a corresponding elevation in aminotransferase concentrations. Nevertheless, because of the observational nature of the study, these findings cannot establish whether the observed hepatic abnormalities were directly caused by HF.

The association between liver density and cardiovascular mortality is another clinically relevant finding. Patients who died from cardiac causes had lower mean liver density than the overall study population, and this association was statistically significant. However, this finding should be interpreted with caution. The present analysis does not establish liver density as an independent predictor of mortality because the observed association may be influenced by other clinical, laboratory, or echocardiographic factors. Demonstration of independent prognostic value would require an appropriately designed longitudinal analysis, preferably using survival analysis with adjustment for relevant potential confounders. Therefore, liver density should currently be regarded as an imaging parameter associated with cardiac outcomes rather than an established independent prognostic biomarker.

Our findings are broadly consistent with previous studies describing an association between HF and hepatic abnormalities. In severe circulatory failure, markedly reduced hepatic oxygen delivery may result

in hypoxic (ischemic) hepatitis, characterized by acute hepatocellular injury and marked elevations in aminotransferases (6, 11). Previous investigations have reported that elevated total bilirubin is associated with adverse outcomes in patients with HF (4, 5). Other studies have demonstrated relationships among severe tricuspid regurgitation, increased central venous pressure, hepatic congestion, and abnormalities in liver biochemical tests, particularly bilirubin and aminotransferases (9, 12–13). These observations provide a potential physiological framework for the association observed in the present study between liver density, bilirubin concentrations, and markers of congestion.

Longitudinal studies have also reported associations between abnormal liver function tests and mortality in patients with HF, with bilirubin and AST identified as potentially important prognostic markers (4–5, 12). The significant association between total bilirubin and liver density in our study is compatible with these observations. However, our study primarily evaluated cross-sectional associations between liver density and clinical, laboratory, and echocardiographic variables. Consequently, it remains uncertain whether liver density provides prognostic information beyond established HF markers. Further prospective studies are needed to determine its incremental prognostic value.

Previous studies have emphasized the bidirectional interaction between cardiac and hepatic dysfunction (3, 14). The present findings are consistent with this concept, suggesting that hepatic imaging abnormalities may accompany the structural and hemodynamic abnormalities observed in HF. However, the mechanisms underlying the association between liver density and HF severity remain uncertain. Potential mechanisms may include chronic hepatic congestion, altered hepatic perfusion, changes in hepatic tissue composition, and the systemic effects of advanced HF. These mechanisms require further investigation using longitudinal clinical, biochemical, and imaging assessments.

Some findings in our study differed from observations reported in previous investigations. In particular, previous studies have emphasized the importance of RV dysfunction, elevated central venous pressure, and tricuspid regurgitation in hepatic congestion and abnormal liver function tests (7, 12). In contrast, TAPSE was not significantly associated with liver density in our population. This discrepancy may be related to differences in study populations, disease severity, imaging techniques, or the specific parameters used to assess RV function and hepatic involvement. Moreover, TAPSE alone may not adequately characterize the complex interaction between RV performance, pulmonary pressures, venous congestion, and hepatic changes.

Similarly, although previous studies have reported associations between aminotransferase abnormalities and adverse outcomes in HF, AST and ALT were not significantly correlated with liver density in our study. This discrepancy may reflect the different biological information provided by CT-derived liver density and serum aminotransferases. Liver density may reflect structural or compositional characteristics of hepatic tissue, whereas aminotransferases primarily reflect hepatocellular injury. Hepatic steatosis is also associated with cardiovascular abnormalities, including subclinical coronary atherosclerosis and also influence CT-derived liver attenuation and may coexist with cardiovascular and metabolic abnormalities, providing a potential additional explanation for variation in liver density. (15–16). Previous

studies demonstrated that non-enhanced CT can be used to detect and quantify hepatic steatosis, with CT attenuation providing quantitative information regarding hepatic fat accumulation. (12, 17) Accordingly, CT-based assessment may provide complementary information rather than simply replicate conventional biochemical measures of hepatic dysfunction.

The present study has several limitations. First, the observational design limits the ability to establish temporal or causal relationships between liver density and HF severity. Second, the relatively small sample size and single-center setting may limit the generalizability of the findings. Third, although CT-derived liver density was assessed using standardized regions of interest, hepatic attenuation may be influenced by several factors, including technical characteristics of CT acquisition and underlying hepatic composition. Fourth, TAPSE and the available echocardiographic parameters may not fully characterize right-sided cardiac function or systemic venous congestion. Finally, although an association with cardiac mortality was observed, comprehensive multivariable survival analysis would be required to determine whether liver density provides independent prognostic information.

In conclusion, CT-measured liver density was associated with several clinical, laboratory, and echocardiographic markers of HF severity, including LVEF, LVEDD, SPAP, IVC diameter, edema, ascites, and bilirubin concentrations. Lower liver density was also associated with cardiac mortality. These findings suggest that CT-derived liver density may serve as a noninvasive imaging marker of cardiohepatic involvement in patients with HF. However, further prospective, multicenter studies with larger populations and adjusted longitudinal analyses are needed to determine the clinical significance and independent prognostic value of liver density in HF.

Implications

CT-derived liver density may serve as a complementary imaging marker of HF severity and systemic congestion. However, it should not yet be considered an established prognostic biomarker. Larger prospective studies are needed to determine whether liver density provides independent prognostic information beyond established HF risk factors.

Limitations

This study has several limitations. First, the cross-sectional design limits assessment of temporal relationships and causality; therefore, the observed associations do not establish liver density as an independent predictor of mortality. Second, the relatively small sample size and single-center setting may limit statistical power and generalizability. Third, all patients had COVID-19, which may independently affect cardiac function, hepatic status, inflammation, and clinical outcomes, potentially confounding the observed associations and limiting their applicability to non-COVID-19 HF populations. Fourth, residual confounding cannot be excluded, and TAPSE alone may not fully represent right ventricular function or systemic venous congestion. Finally, multiple associations were assessed, increasing the possibility of chance findings; therefore, the results should be considered hypothesis-generating and confirmed in larger prospective studies.

Conclusion

In conclusion, CT-measured liver density was significantly associated with several clinical, echocardiographic, and biochemical characteristics of HF, including LVEF, LVEDD, SPAP, IVC diameter, edema, ascites, and bilirubin. In contrast, no significant association was observed between liver density and TAPSE, AST, or ALT.

These findings suggest that liver density may be a potential imaging marker associated with HF severity and cardiohepatic involvement. However, the present study does not establish liver density as an independent prognostic factor because of its cross-sectional design and the absence of a dedicated longitudinal prognostic analysis.

The findings should also be interpreted in the context of the study population, as all participants had COVID-19, which may have influenced both hepatic and cardiovascular findings. Large, prospective, multicenter studies involving patients with and without COVID-19, with appropriate multivariable and survival analyses, are needed to determine whether liver density has independent prognostic value in HF.

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Rajaie Cardiovascular Institute, Tehran, Iran (Ethics Committee approval code: [IR.RHC.REC.1403.080]).

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly available due to privacy and confidentiality restrictions concerning the participants but are available from the corresponding author on reasonable request and with permission from the relevant ethics committee.

Competing interests

The authors declare that they have no competing interests.

Funding

The authors received no financial support for the research, authorship, or publication of this article.

Authors' contributions

M.S. contributed to the investigation, data curation, and writing of the original draft. M.M. was responsible for conceptualization, methodology, supervision, project administration, and writing – review and editing. N.N. contributed to the investigation, clinical management, and writing – review and editing. H.P. was responsible for the investigation, imaging analysis and interpretation, and writing – review and editing. P.G. contributed to the investigation and data curation. S.T., R.O., and H.G. contributed to the investigation, clinical management, and writing – review and editing. A.H.B. contributed to the investigation, data curation, and writing of the original draft.

All authors contributed to the study, critically reviewed the manuscript, and read and approved the final version of the manuscript.

Use of Large Language Models (LLMs)

ChatGPT (OpenAI) was used for language editing during manuscript preparation. The authors reviewed and approved the final manuscript and take full responsibility for its content.

Acknowledgment

The authors would like to acknowledge all colleagues involved in the clinical care of the patient at Rajaie Cardiovascular Institute.

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Tables

Table 1
Demographic, comorbidity, clinical, and treatment characteristics of the study population

Characteristic	Value
Demographic characteristics	
Age, years	42.8 ± 10.5
Height, cm	169.9 ± 10.0
Weight, kg	79.5 ± 16.7
BMI, kg/m²	27.06 ± 4.44
Sex, n (%)	
Male	98 (67.6)
Female	47 (32.4)
Comorbidities, n (%)	
Current smoking	43 (29.7)
Diabetes mellitus	38 (26.2)
Hypertension	44 (30.3)
Dyslipidemia	10 (6.9)
Coronary artery disease	34 (23.4)
Chronic lung disease	2 (1.4)
Rheumatologic disease	1 (0.7)
Thyroid disease	19 (13.1)
Clinical characteristics, n (%)	
Dyspnea	123 (84.8)
Edema	63 (43.4)
Ascites	18 (12.4)
Medications, n (%)	
Beta-blocker	117 (80.7)
Values are presented as mean ± standard deviation or number (percentage).	
BMI, body mass index; ACE, angiotensin-converting enzyme.	

Characteristic	Value
ACE inhibitor	79 (54.5)
Angiotensin receptor blocker	29 (20.0)
Angiotensin receptor–neprilysin inhibitor	10 (6.9)
Mineralocorticoid receptor antagonist	121 (83.4)
Allopurinol	37 (25.5)
Furosemide	123 (84.8)
Statin	48 (33.1)
Values are presented as mean ± standard deviation or number (percentage).	
BMI, body mass index; ACE, angiotensin-converting enzyme.	

Table 2

Correlations between liver density and echocardiographic and laboratory variables

Variable	Overall value*	P value	Correlation coefficient (r)
Liver density, HU	51.49 ± 10.40	—	—
Echocardiographic variables			
LVEF, %	21.43 ± 12.01	0.001*	0.325
LVEDD, cm	6.27 ± 0.92	0.003*	−0.320
TAPSE, mm	14.97 ± 3.87	0.514	0.056
SPAP, mmHg	40.99 ± 14.94	0.002*	−0.276
IVC diameter, mm	19.23 ± 3.49	0.001*	−0.286
Laboratory variables			
Creatinine, mg/dL	1.27 ± 0.73	0.012*	−0.214
Hemoglobin, g/dL	13.40 ± 2.13	0.075	0.150
Uric acid, mg/dL	7.21 ± 2.30	0.032	−0.186
ProBNP, pg/mL	5760.46 ± 8528.14	0.625	−0.079
Albumin, g/dL	3.80 ± 0.53	0.071*	0.161
Vitamin D, ng/mL	14.44 ± 8.16	0.967	0.011
ALP, IU/L	189.42 ± 96.90	0.018*	−0.205
ALT, IU/L	52.86 ± 101.67	0.281	−0.092
AST, IU/L	55.51 ± 130.70	0.396	−0.073
Direct bilirubin	0.83 ± 1.38	0.026*	−0.190
Total bilirubin	1.83 ± 1.90	0.001*	−0.296
Sodium, mEq/L	135.38 ± 5.23	0.001*	0.317
Phosphorus, mg/dL	3.90 ± 0.96	0.915	0.009
INR	1.53 ± 0.72	0.216	0.113

Overall values are presented as mean ± standard deviation. *P < 0.05, indicating a statistically significant difference.

ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; HU, Hounsfield units; INR, international normalized ratio; IVC, inferior vena cava; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; ProBNP, pro-B-type natriuretic peptide; SPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion; TSH, thyroid-stimulating hormone.

Variable	Overall value*	P value	Correlation coefficient (r)
TSH, mU/L	2.70 ± 2.15	0.872	−0.014
Total cholesterol, mg/dL	139.12 ± 42.79	0.001*	0.297
Iron saturation, %	18.14 ± 7.87	0.234	0.117
Ferritin, ng/mL	184.26 ± 189.91	0.912	−0.011
BUN, mg/dL	23.96 ± 19.21	0.082*	−0.150
Calcium, mg/dL	8.76 ± 0.55	0.038*	0.178
Overall values are presented as mean ± standard deviation. *P < 0.05, indicating a statistically significant difference.			
ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; HU, Hounsfield units; INR, international normalized ratio; IVC, inferior vena cava; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; ProBNP, pro-B-type natriuretic peptide; SPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion; TSH, thyroid-stimulating hormone.			

Table 3

Liver density according to clinical characteristics and outcomes

Variable	Liver density, mean ± SD (HU)	P value
Clinical characteristics		
Dyspnea	52.63 ± 7.20	0.607
Edema	54.50 ± 9.30	0.001*
Ascites	52.64 ± 10.34	0.001*
Clinical outcomes		
Other-cause mortality	49.00 ± 8.33	0.580
Cardiovascular mortality	47.29 ± 7.10	0.029*
3-month hospitalization	52.07 ± 9.81	0.243
1-year hospitalization	51.96 ± 9.44	0.340
SD, standard deviation; HU, Hounsfield units. *P-value < 0.05 was considered statistically significant, and the reported values represent comparisons of liver density according to the respective clinical characteristics or outcomes.		

Figures

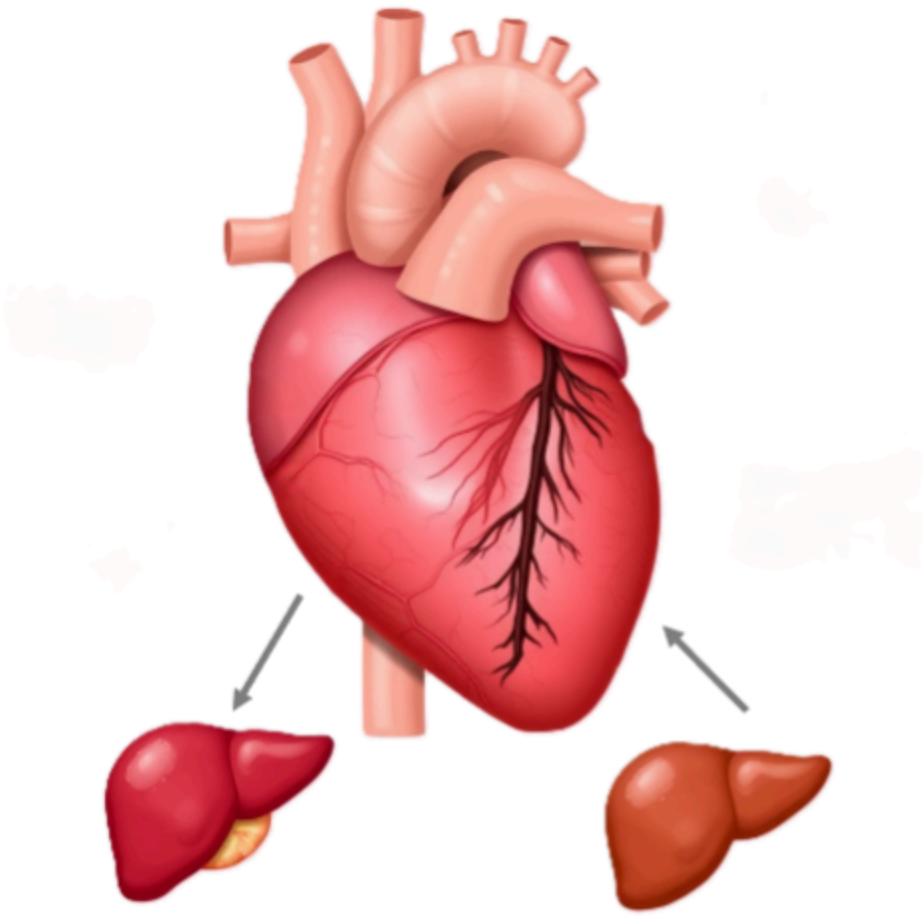


Figure 1

Bidirectional relationship between liver disease and cardiac dysfunction (Figure was created by AI).

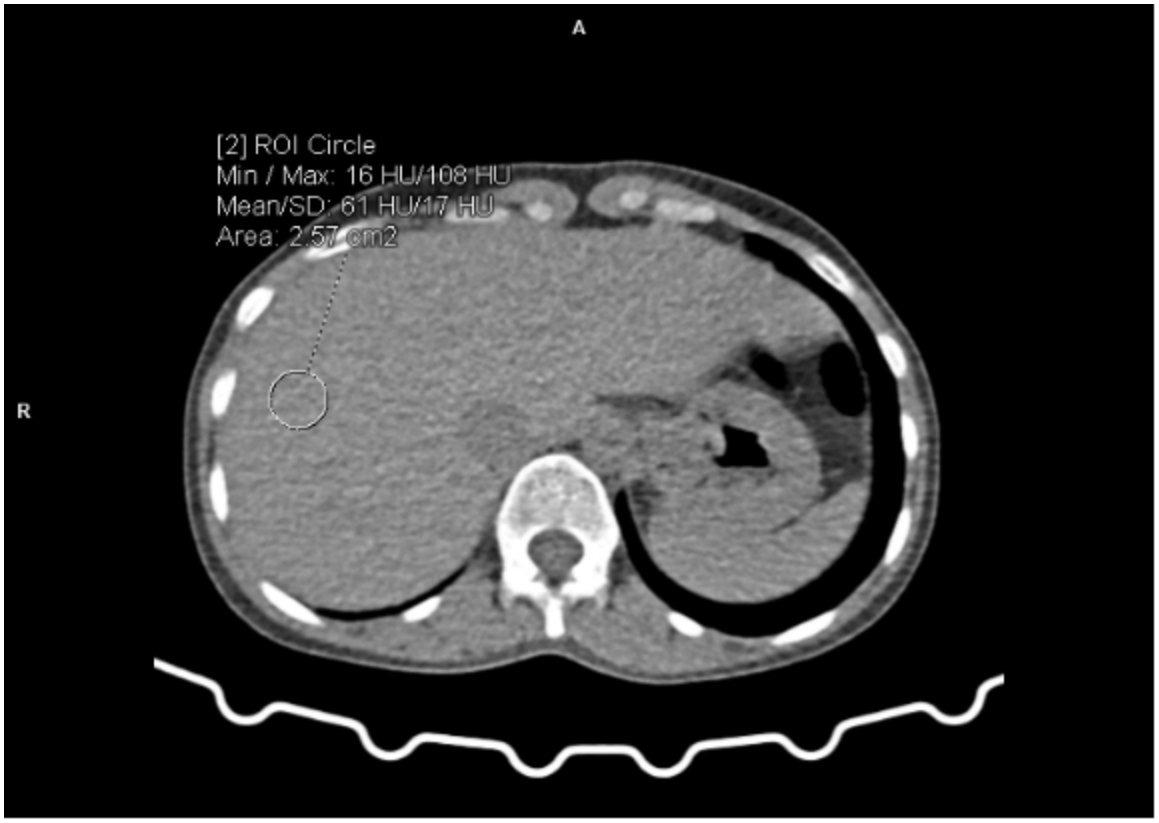


Figure 2

Non-contrast CT scan showing ROI placement for measurement of liver parenchymal density.