Cirrhotic Cardiomyopathy: Proportion and Correlation with Liver Fibrosis Severity

Juferdy Kurniawan[1*], Maela Rustiana Dewi[2], Dono Antono[3], Dicky Levenus Tahapary[2], Leonard Nainggolan[2], Gurmeet Singh[2], M. Syahrir Azizi[3], Rudi Putranto[2], Kaka Renaldi[2], Sukamto[2]

[1]Division of Hepatobiliary, Department of Internal Medicine, Faculty of Medicine Universitas Indonesia - Cipto Mangunkusumo Hospital, Jakarta, Indonesia.
[2]Department of Internal Medicine, Faculty of Medicine Universitas Indonesia - Cipto Mangunkusumo Hospital, Jakarta, Indonesia.
[3]Division of Cardiology, Department of Internal Medicine, Faculty of Medicine Universitas Indonesia - Cipto Mangunkusumo Hospital, Jakarta, Indonesia.

[*]***Corresponding Author:***
Juferdy Kurniawan, MD, PhD. Division of Hepatobiliary, Department of Internal Medicine, Faculty of Medicine Universitas Indonesia - Cipto Mangunkusumo Hospital. Jl. Diponegoro No. 71, Jakarta 10430, Indonesia. Email: juferdy.k@gmail.com.

ABSTRACT

Background: *Cirrhotic cardiomyopathy is characterized by an impaired systolic response to stress, diastolic dysfunction, and electrophysiological abnormalities, without underlying structural heart disease. Left ventricular diastolic dysfunction (LVDD) is the most common manifestation found in cirrhotic cardiomyopathy. Previous studies reported its correlation with a higher Child-Turcotte-Pugh (CTP) score in late-stage cirrhosis. To date, no studies have evaluated the correlation between liver fibrosis and LVDD parameters. This study aims to evaluate the correlation between liver fibrosis and LVDD parameters.* ***Methods:*** *We conducted a cross-sectional study on liver cirrhosis patients without a history of cardiac disease. Subjects were recruited consecutively from the Hepatobiliary Outpatient Clinic and Internal Medicine Inpatient Unit at Cipto Mangunkusumo National General Hospital. A total of 92 patients underwent 2-dimensional echocardiography with tissue Doppler imaging, transient elastography, and laboratory tests for routine hematology, ALT, and AST.* ***Results:*** *A total of 27.2% of subjects had diastolic dysfunction and 37% had normal diastolic function with an increased left atrial volume index. APRI and FIB-4 showed a positive correlation with left atrial volume index ($r = 0.302$; $p = 0.003$ vs $r = 0.401$; $p < 0.0001$, respectively). FIB-4 demonstrated a fair performance in estimating increased left atrial volume index (AUC 0.746; 95% CI 0.646–0.846), with a cut-off point of 4.38.* ***Conclusion:*** *Diastolic dysfunction is the most common early manifestation found in cirrhotic cardiomyopathy. Liver fibrosis, assessed by APRI and FIB-4, was positively correlated with increased left atrial volume index.*

Keywords: *Liver cirrhosis, cardiomyopathies, ventricular dysfunction, left, elasticity imaging techniques, echocardiography, doppler, atrial remodeling.*

INTRODUCTION

Liver cirrhosis represents the end-stage of progressive hepatic fibrosis, which is characterized by architectural distortion and the formation of regenerative liver nodules.[1] It is a major cause of morbidity and mortality in patients with chronic liver disease, accounting for approximately 2.4% of global deaths in 2019, making it the 11th leading cause of death worldwide.[2] The burden of liver

cirrhosis is expected to rise due to changing lifestyles, increased alcohol consumption, and global demographic shifts that influence the transmission of hepatotropic viruses.

A key complication of cirrhosis is portal hypertension, which may manifest as ascites, hepatorenal syndrome, portal hypertensive gastropathy, and gastroesophageal varices. Portal hypertension contributes to cardiovascular dysfunctions through the activation of the renin–angiotensin–aldosterone system (RAAS) and sympathetic nervous system, resulting in a hyperdynamic circulatory state. In cirrhotic patients, peripheral arteriovenous shunting and systemic vasodilation occur due to increased nitric oxide (NO) production, resulting in higher arterial blood flow and reduced systemic vascular resistance, contributing to elevated cardiac output. This leads to increased cardiac workload and subsequent myocardial remodeling, including hypertrophy, fibrosis, and increased stiffness.[3,4] The pathomechanisms contributing to cardiac dysfunction in liver cirrhosis include alterations in cholesterol/phospholipid composition of the cardiomyocyte plasma membrane, impaired β-adrenergic receptor function, cardiodepressant effects of endogenous cannabinoids, type I collagen deposition in cardiomyocytes, and reduced titin myofilament phosphorylation, which increases myocardial stiffness.[4,5]

Cardiovascular abnormalities in cirrhosis were first described by Kowalski and Abelmann in 1953, who reported an increased resting cardiac output and decrease in peripheral resistance among patients with chronic liver disease and alcoholism. These findings were initially attributed to alcohol toxicity, which was later proven to be present in nonalcoholic cirrhosis. The term cirrhotic cardiomyopathy was introduced in 1990 by Lee et al., which describes a subclinical cardiac dysfunction in cirrhotic patients, characterized by systolic and diastolic dysfunction, electrophysiological abnormalities, and cardiac chamber dilation and hypertrophy, in the absence of intrinsic heart disease.[4,6]

Among these features, diastolic dysfunction is considered an early manifestation of cirrhotic cardiomyopathy. The prevalence of cirrhotic cardiomyopathy among cirrhotic patients varies from 46% to 87.5%, with 34.3% in Indonesia.[7], 68.4% in India[8], and 46.2% in Spain[9]. Pozzi et al.[10] were the first to describe left ventricular diastolic dysfunction (LVDD) in cirrhosis in 1996, using an E/A ratio < 1 as the diagnostic criterion. Recent studies have identified septal e' velocity, E/e' ratio, and left atrial volume index (LAVI) as more sensitive markers of LVDD in this population.[11]

Previous studies have demonstrated the correlations between these diastolic parameters and the severity of liver dysfunction assessed with the Child-Pugh score.[7,8,12,13] However, the Child-Pugh score reflects hepatic dysfunction and clinical decompensation but does not directly reflect portal hypertension, which plays a central role in the pathogenesis of cardiac dysfunction in cirrhosis. Liver fibrosis markers, which better reflect the degree of intrahepatic resistance and portal pressure, may serve as more relevant parameters for assessing cardiovascular complications in liver cirrhosis.

Noninvasive liver fibrosis parameters such as transient elastography, APRI, and FIB-4 have been shown to correlate with the degree of liver fibrosis and portal pressure. Transient elastography provides high diagnostic accuracy for advanced fibrosis ($F \geq 3$) and cirrhosis (AUC 0.95 and 0.97, respectively).[14] APRI and FIB-4 are simple, inexpensive, and accessible scoring systems requiring only age, platelet count, and liver function tests.[15] This study aimed to investigate the correlation between liver fibrosis severity, as assessed by transient elastography, APRI score, and FIB-4, with parameters of LVDD in patients with liver cirrhosis.

METHODS

This cross-sectional study was conducted in the Hepatobiliary Outpatient Clinic and Inpatient Ward of Cipto Mangunkusumo National General Hospital from April to June, 2025. The sample size was calculated using a formula based on a correlation coefficient (r 0,5), with a minimum of 32 subjects. All subjects were recruited consecutively. This study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Indonesia (No. KET-1839/UN2.F1/ETIK/PPM.00.02/2024).

Inclusion and Exclusion Criteria

All patients with liver cirrhosis of any etiology aged ≥ 18 years who agreed to participate were included in the study. Subjects with coronary artery disease, history of valvular heart disease, or evidence of significant moderate to severe regurgitation or stenosis based on echocardiography results, heart failure, congenital heart disease, arrhythmias, cardiac tumors or paracardiac masses compressing the heart, and patients with grade 2 or 3 ascites were excluded.

Data Collection

Participants were screened for eligibility criteria. Baseline characteristics data such as age, gender, etiology of liver cirrhosis, duration of liver cirrhosis, comorbidities such as hepatocellular carcinoma, diabetes, hypertension, obesity, dyslipidemia, chronic kidney disease, and medication history including beta-blockers, aldosterone antagonists, and other drugs related to comorbid conditions were obtained.

Two-dimensional echocardiography with tissue Doppler imaging (GE Vivid T9®) was performed by a single examiner, which was subsequently validated by a cardiovascular consultant. LVDD was defined based on the Cirrhotic Cardiomyopathy Consortium 2019 criteria. Subjects with ≥ 3 criteria of the following: 1) Septal e' velocity < 7 cm/second; 2) E/e' ≥ 15; LAVI > 34 ml/m2; TR velocity > 2,8 m/second were considered to have severe diastolic dysfunction. Diastolic dysfunction grading was assessed based on the American Society of Echocardiography (ASE) 2016 guidelines.[16] Liver fibrosis was assessed by transient elastography (fibroscan®), APRI score, and Fibrosis-4 (FIB-4) score.[14] Transient elastography was performed by two examiners. Portal hypertension was reported based on the presence of ascites, portal hypertensive gastropathy, and gastroesophageal varices. All subjects were tested for laboratory parameters such as hemoglobin, platelets, AST, and ALT.

Data Analysis

Statistical analyses were performed using IBM SPSS. Numerical data were presented as mean (standard deviation) for normally distributed data and median (min-max) for skewed distributed data. Categorical data were presented in percentages. The correlation between transient elastography results, APRI, and FIB-4 score with LVDD was analysed using Pearson analysis for normally distributed data or Spearman analysis for skewed distributed data. Receiver Operating Characteristic (ROC) analysis was performed to determine the transient elastography, AST to Platelet Ratio Index (APRI), and FIB-4 cut-off point to detect LVDD. The optimal cut-off point was determined using Youden's Index method with sensitivity-(1-specificity) data. P value <0.05 was considered statistically significant.

RESULTS

Ninety-two participants were recruited and analyzed in this study, consisting of 60 outpatients (65.3%) and 32 inpatients (34.7%). The median age of the subjects was 57 (25–73) years, with 54 males (58.7%). A total of 78 out of 92 patients (84.8%) had portal hypertension. The most common cause of liver cirrhosis was chronic hepatitis B (67.4%), followed by chronic hepatitis C (26.1%). Patient comorbidities included diabetes mellitus (21.7%), hypertension (31.5%), and non-metastatic hepatocellular carcinoma (6.5%). Among the inpatients, 5 (5.4%) were admitted for hepatic encephalopathy, 4 (4.3%) for esophageal varices hemorrhage, and 1 for massive ascites, while the remaining were hospitalized for elective gastroscopy. (**Table 1 and 2**)

LVDD was identified in 25 subjects (27.2%), consisting of 14 subjects (15.2%) with grade 1 LVDD, 9 subjects (9.8%) with grade 2 LVDD, and 2 subjects (2.2%) with grade 3 LVDD. A total of 34 subjects (37%) had normal diastolic function but with an increased left atrial volume index. Overall, 47 subjects (51.1%) had an elevated left atrial volume index regardless of their diastolic function status.

APRI score showed a weak positive correlation with increased left atrial volume index ($r = 0.302$; $p = 0.003$). In addition, FIB-4 score demonstrated a moderate positive correlation with increased left atrial volume index ($r = 0.401$; $p < 0.001$). (**Table 5, Figure 1**)

Table 1. Baseline characteristics of the study participants

Parameters	N=92
Gender, n (%)	
Male	54 (58,7%)
Female	38 (41,3%)
Age, years; median (min – max)	57 (25-73)
Etiology of cirrhosis, n (%)	
Chronic hepatitis B	62 (67,4%)
Chronic hepatitis C	24 (26,1%)
Non-B non-C	6 (6,5%)
Nonalcoholic fatty liver	2 (2,2%)
Genetic*	1 (1,1%)
Unknown	3 (3,3%)
Cirrhosis duration, years; median (min – max)	2 (1 month – 15 years)
Comorbidities; n (%)	
Hepatocellular carcinoma	6 (6,5%)
Type 2 diabetes	20 (21,7%)
Hypertension	29 (31,5%)
Obesity	42 (45,7%)
Dyslipidemia	3 (3,3%)
CKD G3	4 (4,3%)
G6PD deficiency	1 (1,1%)
Other malignancy**	2 (2,2%)
Without comorbid; n (%)	41 (44,6%)
Antiviral therapy; n (%)	
Tenofovir	58 (63%)
Entecavir	4 (4,3%)
DAA	6 (6,5%)
History of DAA therapy	18 (19,5%)
Nonselective beta blocker; n (%)	
Propranolol	63 (68,5%)
Carvedilol	2 (2,2%)
Other drugs; n (%)	
Spironolacton	18 (19,6%)
Oral antidiabetic drugs	12 (13%)
Subcutaneous insulin	4 (4,3%)
Antihypertensive drugs	27 (29,3%)
Portal hypertension; n (%)	78 (84,8%)
***Child Turcotte Pugh* (CTP); n (%)**	
A	50 (54,3%)
B	31 (33,7%)
C	11 (12,0%)
Esophageal varises hemorrhage; n (%)	4 (4,3%)
Hepatic encephalopathy grade 1-3; n (%)	5 (5,4%)
Ascites grade 1; n (%)	16 (17,4%)
Massive ascites post-paracentesis; n (%)	1 (1,1%)
Mean arterial pressure, mmHg; mean (SD)	89,9 (11,2)
Resting heart rate, bpm; mean (SD)	73 (12,3)
Hemoglobin, g/dl; mean (SD)	12,1 (2,3)
Platelet count, (per mm^3); median (min – max)	132.000 (25.000 – 337.000)
AST, U/L; median (min – max)	38 (17 – 252)
ALT, U/L; median (min – max)	29 (8 – 197)
Transient elastography kPa); median (min – max)	16,8 (4,7 – 62,7)
APRI score; median (min – max)	1,0 (0,2 – 7,7)
FIB-4 score; median (min – max)	3,6 (0,6 – 19,0)

*Suspected Alagille syndrome; **1 prostate cancer, 1 laryngeal carcinoma

Table 2. Comparison of characteristics between outpatient and inpatient subjects

Parameters	Outpatient (N= 60)	Inpatient (N = 32)
Child Turcotte Pugh (CTP), n (%)		
A	36 (60%)	14 (43,8%)
B	21 (35%)	10 (31,2%)
C	3 (5%)	8 (25%)
Hemoglobin (g/dl)	12,5 (2,0)	11,2 (2,6)
Platelet count (per mm^3)	142.000 (40.000 – 337.000)	95.000 (25.000 – 291.000)
AST (U/L)	36,5 (19 – 105)	43,5 (17 – 252)
ALT (U/L)	28,5 (9 – 197)	30 (8 – 190)
Transient elastography (kPa)	16,5 (4,7 – 57,2)	17,8 (4,9 – 62,7)
APRI score	0,8 (0,2 – 5,7)	1,4 (0,2 – 7,7)
FIB-4 score	2,7 (0,8 – 18,8)	4,8 (0,6 – 19,0)

Table 3. Echocardiographic findings of the study participants

Parameters	N = 92
LVIDd (mm)	44,2 (6,0)
LVIDs (mm)	28,3 (4,0)
IVSd (mm)	9 (4 – 16)
LVPWd (mm)	9 (6 – 16)
Cardiac output (L/minute)	4,1 (2,12 – 6,97)
Ejection fraction (%)	66 (40 – 79)
Ejection fraction ≤ 50%; n (%)	2 (2,2%)
LAVI (mL/m^2)	33,18 (10,5)
LAVI ≥ 34 $mL/m^{2,}$ n (%)	47 (51,1%)
E/A ratio	1,1 (0,62 – 3,6)
Septal e' (cm/s)	8 (5 – 13)
Lateral e' (cm/s)	10 (6 – 16)
E/e' ratio	8,7 (2,0)
TRVmax (cm/s)	212,7 (35,8)

Table 4. Proportion of diastolic dysfunction between outpatient and inpatient subjects

	Outpatient (N=60)	Inpatient (N=32)
LVDD; n (%)	16 (26,7%)	9 (28,1%)
Grade 1	10	4
Grade 2	5	4
Grade 3	1	1
LAVI ≥ 34 ml/m^2; n (%)	25 (41,6%)	22 (68,6%)

Table 5. Correlation between liver fibrosis parameter and LVDD parameters

	E/e' ratio	e' septal	LAVI	E/A ratio
Transient elastography	r 0,113 p 0,284	r – 0,051 p 0,628	r 0,127 p 0,107	r -0,018 p 0,865
APRI score	r 0,08 p 0,45	r 0,225 p 0,051	r 0,302 p 0,003	r 0,193 p 0,065
FIB-4 score	r 0,12 p 0,253	r 0,134 p 0,204	r 0,401 p <0,001	r 0,120 p 0,256

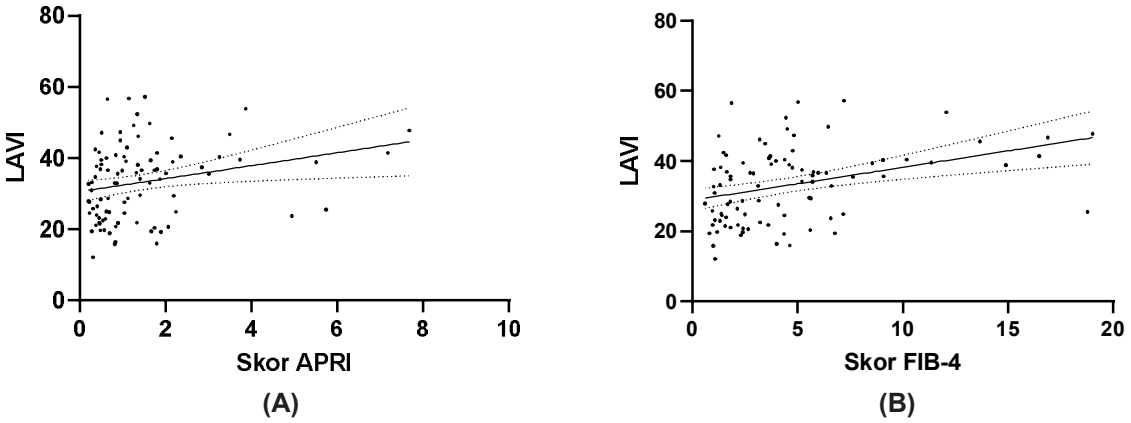

Figure 1. Scatter plot diagram showing the correlation between APRI score and LAVI (A), FIB-4 score and LAVI (B)

Further analysis regarding the estimation of LVDD showed that the liver fibrosis parameter had poor performance in estimating the event. Despite this, the FIB-4 score demonstrated fairly good performance in estimating the increased of left atrial volume index, with an AUC of 0.746 (95% CI: 0.646–0.846, p < 0.001) and a cutoff value of 4.38. (**Table 6, Figure 2**)

Table 6. Performance of liver fibrosis parameters in estimating the event of LVDD and increased LAVI

Parameters	LVDD		Increased LAVI	
	AUC (95% CI)	p value	AUC (95% CI)	p value
Transient elastography	0,491 (0,363 – 0,619)	0,897	0,599 (0,481 – 0,717)	0,06
APRI score	0,360 (0,230 – 0,490)	0,037	0,709 (0,603 – 0,815)	0,001
FIB-4 score	0,433 (0,305 – 0,561)	0,319	0,746 (0,646 – 0,846)	<0,001

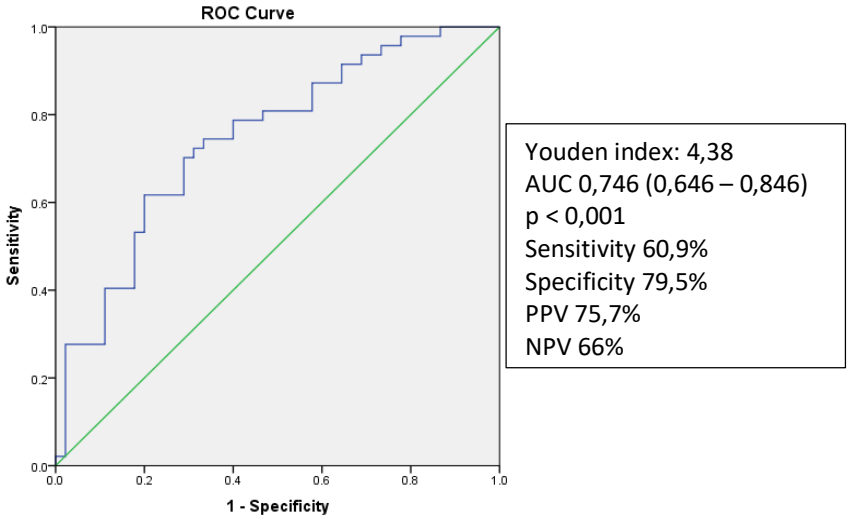

Figure 2. ROC curve of FIB-4 score in estimating elevated left atrial volume index

DISCUSSION

The prevalence of LVDD in our study (27.2%) was comparable to the findings of the previous study conducted in RSCM by Mondrowinduro et al.[7] (34%), and lower than other reported studies. A study by Solanki et al.[8] in India reported LVDD in 68,4% subjects, while Al Atroush et al.[12] in Egypt reported a prevalence of 87,5%. The lower prevalence of LVDD observed in our study may be due to the predominance of patients with compensated cirrhosis among the study subjects. Most cirrhotic subjects in this study were categorized as Child-Pugh (CP) A (54.3%), while CP B and C accounted for 33.7% and 12%, respectively. Since the majority of the study participants were recruited from the outpatient clinic, a greater proportion of CP A and B patients was expected. This CP classification pattern is consistent with a previous study that also enrolled patients from both outpatient and inpatient settings. For instance, Mondrowinduro et al.[7] reported CP A, B, and C distributions of 50%, 34.3%, and 15.7%, respectively. In contrast, studies focusing solely on hospitalized patients, such as the study by Al Atroush et al.[12]reported a higher proportion of advanced disease, with 45% classified as CP B and 55% as CP C, with no patients in the CP A category. Several factors may also explain this distribution, including earlier detection of cirrhosis, improved diagnostic capabilities, and the success of antiviral treatment programs.

Reduced ejection fraction (≤50%) was identified in 2 subjects only (2,2%). Nearly all subjects demonstrated preserved systolic function, with a median ejection fraction of 66% (40%–79%). This finding aligns with existing literature stating that patients with cirrhosis generally have normal or even slightly elevated ejection fractions at rest. Mondrowinduro et al.[7] also reported a normal mean ejection fraction of 70.43% (±7.01%). Due to systemic vasodilation and hyperdynamic circulation in cirrhotic patients—where ejection fraction tends to be normal or even elevated—additional markers are needed to assess subclinical systolic dysfunction. Currently, the American Society of Echocardiography (ASE) recommends the use of strain imaging (Global Longitudinal Strain/GLS) as a new objective method for evaluating myocardial contractility.[11] A higher incidence of systolic dysfunction was reported in studies using global longitudinal strain, with Mansoor et al.[17] reporting 28% and Razpotnik et al.[18] 53.3%.

In this study, subgroup analysis showed comparable LVDD prevalence between outpatients and inpatients (Table 4). Among inpatients, more than half had LVDD grades 2–3, whereas outpatients were predominantly LVDD grade 1 (62.5%). It was consistent with previous studies linking LVDD to cirrhosis severity.[8,12,19] Inpatient subjects of this study had higher median transient elastography, APRI, and FIB-4 scores; a greater proportion of Child Pugh C; and lower median platelet counts, indicating more severe portal hypertension.

This study also found that 51.1% of patients had an elevated left atrial volume index (LAVI) (≥34 mL/m^2). Increased LAVI in cirrhotic patients has been widely reported in previous studies, such as by Farouk et al.[20], who reported a mean LAVI of 41 (±12) mL/m², and Al Atroush et al.[12], who found a mean LAVI of 39 (±4.23) mL/m², both significantly higher than control groups ($p < 0.001$). LAVI reflects anatomical and volumetric changes in the left atrium due to chronic volume overload and elevated left ventricular filling pressures.[11] In the study by Cesari et al.[21], elevated LAVI emerged as one of the strongest predictors of mortality, alongside the E/e' ratio. Similarly, a cohort study by Merli et al.[22], which followed 90 cirrhotic patients over two years, reported a significant association between high LAVI and increased mortality risk ($p = 0.04$). Elevated LAVI is not only associated with cirrhosis-related morbidity and mortality but also with cardiovascular morbidity, as supported by cardiovascular literature linking increased LAVI to a higher risk of congestive heart failure, atrial fibrillation, and ischemic stroke.[23]

In our study, both APRI and FIB-4 scores showed a positive correlation with LAVI ($r = 0.302$, $p = 0.003$; $r = 0.401$, $p < 0.001$, respectively). One study by Mansoor et al.[17] explored the relationship between FIB-4 and cardiac dysfunction but found no statistically significant association ($p = 0.234$). To the best of our knowledge, no previous studies have

specifically investigated the correlation between cardiac dysfunction and liver fibrosis severity using transient elastography or APRI score. The weak correlation observed in this study may be attributable to the predominance of subjects with compensated cirrhosis and the fact that most patients were receiving beta-blockers.

In this study, both APRI and FIB-4 scores demonstrated fairly good performance in estimating elevated LAVI, with AUCs of 0.709 (95% CI: 0.603–0.815) and 0.746 (95% CI: 0.646–0.846), respectively. Using Youden's index, the optimal cut-off points for APRI and FIB-4 scores were determined to be 0.92 and 4.38, respectively. The FIB-4 cutoff value falls within the established range for diagnosing liver cirrhosis, suggesting its appropriateness for this purpose. However, the APRI cut-off value identified in our study was relatively low—even below the WHO-recommended lower cut-off for significant fibrosis (≥1)[24], which may indicate limited sensitivity of APRI in this specific context or reflect the influence of other clinical variables in our study population.

Diastolic dysfunction in cirrhosis has been reported to be associated with advanced cirrhosis, hepatorenal syndrome (HRS) development, and mortality.[8,9,13] It contributes to impaired chronotropic function, which reduces effective arterial blood volume, leading to decreased renal perfusion and then contributing to the pathogenesis of hepatorenal syndrome. In addition, asymptomatic cardiac dysfunction may manifest as heart failure during stress (e.g., surgery, liver transplantation, or TIPS procedures).[4,6,25] Early detection of cirrhotic cardiomyopathy is therefore crucial for prognostication, outcome prediction, and guiding therapeutic strategies.

This study is the first to evaluate the correlation between the degree of liver fibrosis and cardiac dysfunction in patients with liver cirrhosis. It also uses the most recent diagnostic criteria for cirrhotic cardiomyopathy, as defined by the Cirrhotic Cardiomyopathy Consortium in 2019, which is based on the 2016 guidelines of the American Society of Echocardiography and considered to be more practical and applicable than earlier definitions. The liver fibrosis parameters used in this study are non-invasive. While not all healthcare facilities have access to transient elastography, the two other fibrosis markers (APRI and FIB-4) are simple to calculate, relying only on age, platelet count, AST, and ALT—laboratory tests that are routinely performed even in peripheral or primary care settings. However, this study has some limitations. A large proportion of participants were on propranolol for either primary or secondary prevention of esophageal varices, which may have contributed to an underestimation of the true prevalence of left ventricular dysfunction and potentially weakened the strength of the correlation observed.

CONCLUSION

Diastolic dysfunction is the most common early manifestation found in cirrhotic cardiomyopathy. Liver fibrosis assessed by APRI and FIB-4 is positively correlated with increased left atrial volume index. FIB-4 score has a fairly good performance in estimating increased left atrial volume index, with an AUC of 0.746 (95% CI: 0.646–0.846, $p < 0.001$) and a cutoff value of 4.38.

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CONFLICT OF INTERESTS

The authors have no conflicts of interest related to this study.

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