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A Study on cardiac dysfunction in non diabetic, non hypertensive patients with liver cirrhosis

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Abstract

Background: Cirrhotic cardiomyopathy (CCM) represents a subclinical cardiac dysfunction observed in patients with liver cirrhosis, often unmasked only during physiological stress. Despite its clinical relevance, particularly in the context of liver transplantation, the characterization of cardiac dysfunction among cirrhotic patients devoid of conventional cardiovascular comorbidities remains limited.

Objectives: To evaluate the prevalence and patterns of cardiac dysfunction in non-diabetic, non-hypertensive patients with liver cirrhosis using electrocardiographic and echocardiographic parameters, and to explore its association with liver disease severity based on the Child-Pugh classification.

Methods: This cross-sectional study included 100 cirrhotic patients without diabetes or hypertension. Detailed clinical evaluation, ECG, and transthoracic echocardiography were conducted. Parameters such as QTc interval, ejection fraction (EF), E/A ratio, and left atrial (LA) volume were assessed. The Child-Pugh score was used to stratify liver disease severity.

Results: Cirrhotic cardiomyopathy was present in 35% of the cohort, with a significantly higher prevalence in Child-Pugh class B compared to class A. The mean QTc interval was 443.13 ± 26.87 ms. Diastolic dysfunction, as defined by an E/A ratio < 1, was seen in 35% of cases. Systolic dysfunction (EF < 60%) was found in 71% of patients. These abnormalities correlated significantly with advancing Child-Pugh class.

Conclusion: Cardiac dysfunction in cirrhosis is a direct hepatic consequence and independent of systemic comorbidities. Routine cardiovascular evaluation in cirrhotic patients is recommended, especially to enhance pre-transplant risk stratification.

Keywords: Cirrhotic cardiomyopathy; Liver cirrhosis; QTc prolongation; Echocardiography; Diastolic dysfunction; Child-Pugh classification; Non-diabetic; Non-hypertensive

1. Introduction

Chronic liver disease (CLD) is defined by a progressive decline in hepatic function lasting over six months, commonly resulting in fibrosis and cirrhosis due to persistent inflammation and tissue regeneration. Globally, CLD affects approximately 1.5 billion people, with non-alcoholic fatty liver disease (NAFLD) accounting for 59% of cases, followed by hepatitis B (29%), hepatitis C (9%), and alcoholic liver disease (2%), while other etiologies make up about 1%[1]. NAFLD, the most prevalent chronic liver condition, encompasses a spectrum from simple steatosis to steatohepatitis,

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cirrhosis, and hepatocellular carcinoma[2]. It is closely linked to metabolic syndrome[3] and is recognized as an independent risk factor for coronary and cerebrovascular disease[4].

CLD also impacts cardiac function even in the absence of pre-existing heart disease[5,6]. In advanced cardiac or hepatic disease, laboratory abnormalities can serve as prognostic markers, emphasizing the need for integrated hepatic and cardiac evaluation[7]. Cirrhosis, regardless of cause, increases risk for complications and reduces life expectancy, largely due to its subtle effects on the cardiovascular system[8,9], with cardiac function deteriorating as liver disease advances[10].

The association between liver disease and cardiovascular abnormalities, first described by Kolwaski et al.[11], is now well established[12,13]. Cirrhotic cardiomyopathy (CCM) is defined by cardiac dysfunction in cirrhosis, characterized by hyperdynamic circulation, systolic and diastolic impairment, and electrophysiological changes. Pathophysiology involves splanchnic vasodilation, sympathetic activation, β-adrenergic receptor downregulation, and increased myocardial stiffness, resulting in both systolic and diastolic dysfunction and QT interval prolongation[14–16]. Echocardiographically, systolic dysfunction appears as reduced ejection fraction, while diastolic dysfunction is evident through an E/A ratio <1.0, prolonged deceleration time, and increased isovolumetric relaxation time[17].

Cardiac structural and functional abnormalities, particularly left ventricular hypertrophy and diastolic dysfunction, have been observed even in non-diabetic, non-hypertensive NAFLD patients. However, few studies have specifically assessed cardiac dysfunction in non-diabetic, non-hypertensive cirrhotic patients, despite the recognized influence of liver-related metabolic and inflammatory factors on cardiovascular remodeling. The primary objectives of this study were to evaluate cardiac dysfunction in non-alcoholic patients diagnosed with cirrhosis of the liver and to explore its relationship with the severity of cirrhosis. Specifically, the study aimed to assess the prevalence of cirrhotic cardiomyopathy and to determine how the degree of liver dysfunction correlates with the presence and extent of cardiac involvement in this population.

2. Methodology

This was a cross-sectional prospective study conducted over 18 months, from July 2023 to December 2024, at Al-Ameen Medical College Hospital, Vijayapur, Karnataka. The study population comprised patients with non-alcoholic liver cirrhosis attending the outpatient and inpatient departments of the Medicine Department at Al-Ameen Medical College and Hospital, Vijayapur. Inclusion criteria were adults aged over 18 years, non-alcoholic patients diagnosed with cirrhosis, and those willing to participate in the study. Exclusion criteria included patients with hypertension, diabetes, a history of alcohol use, severe ascites, coronary artery disease, heart failure, other risk factors for cardiomyopathy apart from cirrhosis, recent upper gastrointestinal bleeding, and severe anaemia.

100 Eligible patients were recruited from the OPD and IPD settings. Data collection was performed using a structured proforma (see Appendix), capturing detailed history and thorough clinical examination. Echocardiographic parameters assessed included the E/A ratio and ejection fraction. In this study, an E/A ratio less than 1 was used as a diagnostic criterion for diastolic dysfunction, and an ejection fraction of 60% was considered the mean normal value for systolic function.

All collected data were entered and compiled in Microsoft Excel. Descriptive statistics were used to present the data. Statistical analysis was performed using SPSS software (version 26.0), with a significance level set at 5% ($\alpha = 0.05$). Qualitative variables were expressed as frequencies and percentages, while quantitative variables were presented as mean and standard deviation. The chi-square test was employed to compare proportions between variables, and Student's t-test and ANOVA were used to compare mean values.

3. Results

The study population comprised 100 participants aged 31 to 70 years. The largest age group was 41–50 years (39%), followed by 51–60 years (34%). The 31–40 and 61–70 age groups represented 14% and 13% of the cohort, respectively. Males predominated (67%), while females made up 33% of the population. Hepatitis B Virus (HBV) infection was the most frequent underlying etiology (61%), with Hepatitis C Virus (HCV) accounting for 30%. Idiopathic causes and primary biliary cholangitis (PBC) contributed to 8% and 1% of cases, respectively. Regarding liver function, 60% of participants were classified as Child-Turcotte-Pugh (CTP) Class A, and 40% as CTP Class B (Table 1).

The mean QTc interval was 443.13 ms (SD: 26.87), with 62% exhibiting QTc prolongation. The E/A ratio—an indicator of diastolic function—was less than 1 in 35% of subjects, corresponding to early diastolic dysfunction, and more than 1

in the remaining 65%. This aligns with the diagnosis of diastolic dysfunction in 35% of participants. Cirrhotic cardiomyopathy (CCM) was present in 35% of cases, reflecting the prevalence of cardiac involvement in this population (Table 2).

Table 1 Demographic and Clinical Characteristics of Study Population (N = 100)

Characteristic	Category	Frequency	Percentage (%)
Age Group (years)	31–40	14	14.0
	41–50	39	39.0
	51–60	34	34.0
	61–70	13	13.0
Sex	Male	67	67.0
	Female	33	33.0
Etiology	HBV	61	61.0
	HCV	30	30.0
	Idiopathic	8	8.0
	PBC	1	1.0
Child-Turcotte-Pugh (CTP)	A	60	60.0
	B	40	40.0

Table 2 Cardiac and Echocardiographic Findings in the Study Population (N = 100)

Parameter	Category	Frequency	Percentage (%)
QTc Interval	Normal QTc	38	38.0
	Prolonged QTc	62	62.0
E/A Ratio	<1	35	35.0
	>1	65	65.0
Diastolic Dysfunction	Yes	35	35.0
	No	65	65.0
Cirrhotic Cardiomyopathy	Yes	35	35.0
	No	65	65.0
End Diastolic Volume (EDV)	<90 mL	41	41.0
	>90 mL	59	59.0
End Systolic Volume (ESV)	<38 mL	16	16.0
	>38 mL	84	84.0
Ejection Fraction (EF)	<60%	71	71.0
	>60%	29	29.0

In terms of ventricular volumes, end diastolic volume (EDV) exceeded 90 mL in 59% of patients (mean: 100.84 ± 27.93 mL), while end systolic volume (ESV) was greater than 38 mL in 84% (mean: 58.51 ± 18.07 mL). Ejection fraction (EF), a measure of systolic function, was below 60% in 71% of the cohort (mean: 53.79 ± 8.19%), indicating that reduced systolic performance was common (Table 2).

A cross-tabulation of CTP class and presence of cirrhotic cardiomyopathy revealed a striking association. In CTP Class A patients, 91.7% were free of CCM while only 8.3% had it. In contrast, among CTP Class B patients, 85.7% had CCM and 14.3% did not. The Pearson Chi-square value was 14.835 with a p-value of < 0.001, indicating a statistically significant association between worsening liver function (CTP Class B) and the presence of cirrhotic cardiomyopathy (Figure 1).

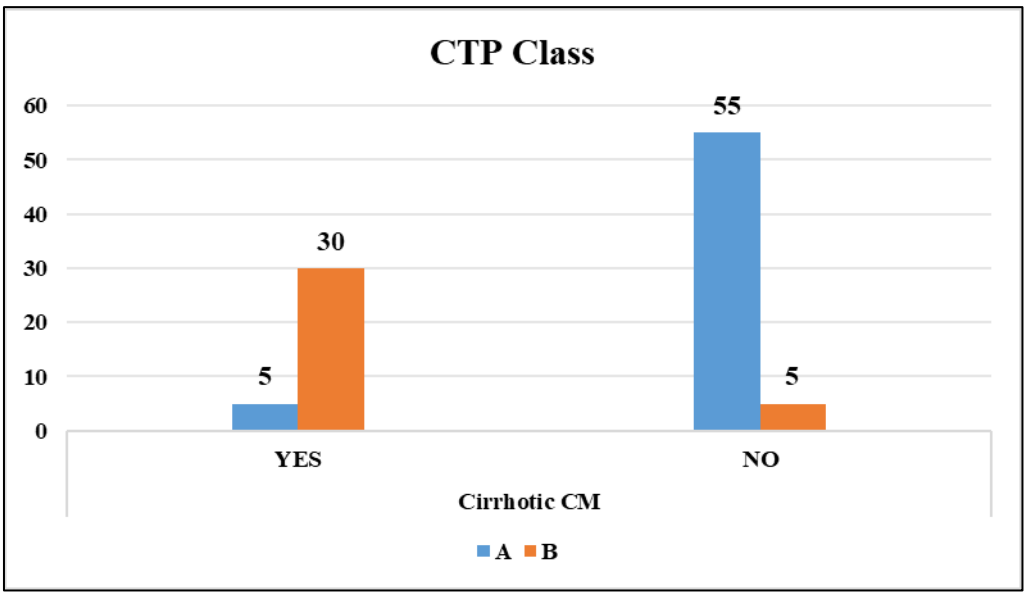

Figure 1 CTP Class vs Cirrhotic Cardiomyopathy Analysis

4. Discussion

Cirrhotic cardiomyopathy (CCM) remains a frequently under-recognized entity in patients with chronic liver disease, especially in its compensated stages. With the advancement of non-invasive diagnostics like echocardiography and electrocardiography, more attention has been paid to the subclinical cardiac alterations associated with cirrhosis. Our study sought to evaluate the prevalence of CCM and associated functional and structural cardiac abnormalities in cirrhotic patients and to assess their correlation with liver disease severity. The results of our study are discussed here in comparison with the findings of other relevant studies to validate patterns, highlight differences, and underscore key implications.

4.1. Demographic Profile

In our study, the most commonly affected age group was 41–50 years (39%), followed by 51–60 years (34%), with a mean age in the early 50s, indicative of middle-aged predominance. This pattern mirrors Shweta Patil et al.,[18] where 70% were aged 40–60 years (mean 55.82 ± 7.44), and Seleem et al.,[19] who reported a mean age of 53.6 ± 12.1 years. Similarly, Farid Ud Din et al.[20] found that 61.96% of patients were between 25 and 45 years (mean 42.92 ± 8.72), while Behera et al.[21] reported a mean age of 44.86 ± 15.90 years. Pimentel et al.[22] also included a relatively young cohort (mean age ≈50 years), and Dadhich et al[23]. noted comparable age distributions.

Males comprised 67% of our study population, reflecting a consistent trend across literature. Chandey et al. [24]reported 91.1% males, Shweta Patil et al.[18] had 68.33% male patients, Behera et al.[21]had 66%, Farid Ud Din et al[20]. had 58.7%, and Pimentel et al.[22] also showed male predominance (56%). This consistent finding may reflect lifestyle-related risk exposures, especially alcohol use, contributing to cirrhosis.

4.2. Etiology of Cirrhosis

In our study, HBV was the most common etiology (61%), followed by HCV (30%). In contrast, Chandey et al.[24] identified alcohol as the leading cause (75.6%), followed by HCV (37.8%). Behera et al. [21]reported alcohol-induced cirrhosis in 41.4%, cryptogenic (15.6%), hepatitis-related (13.28%), and NASH in 7.82%. Pimentel et al.[22] found that 69.8% had non-alcoholic liver disease, while Seleem et al.[19] did not emphasize etiology, and Mansoor et al[25]. highlighted HCV as significantly associated with systolic dysfunction (p = 0.007).

4.3. Prevalence of Cirrhotic Cardiomyopathy

In our study, CCM was found in 35% of cirrhotic patients. This closely aligns with Mansoor et al. [25](34%) and Farid Ud Din et al. [20](41.3%). Behera et al.[21]found a higher prevalence at 68%, with clear progression by Child class: 28.6% in Class A, 58.3% in Class B, and 94.7% in Class C ($p < 0.001$). Our own data followed a similar trend: 20% in Child A and 57.5% in Child B ($p < 0.001$). Pimentel et al.[22]reported that 57% had echocardiographic signs of diastolic dysfunction (24% had $E/e' > 8$, 8.5% had $E/A < 0.8$, and 37% had DT > 240 ms), though only 5 patients had systolic dysfunction.

4.4. Echocardiographic Functional and Structural Changes

In our study, 71% had EF < 60% (mean EF: 53.79 ± 8.19%), and 35% had E/A ratio < 1, indicative of diastolic dysfunction. Shweta Patil et al.[18] reported abnormal E/A in 48.33% of patients, increasing to 70% in MELD Group III. Behera et al.[21] also found lower E/A ratios in cirrhotics (0.89 ± 0.22 vs. 1.096 ± 0.08 in controls, $p = 0.0001$) and significantly lower in CCM (0.785 ± 0.16 vs. 1.11 ± 0.19, $p < 0.05$). Dadhich et al.[23] diagnosed LVDD in 70% of cirrhotics (60% pre-ascitic, 80% ascitic), mostly stage I and II. Pimentel et al.[22]noted that although LA volume differed significantly with Child class, other parameters like E/e' were higher in Child C (42%) but without statistical significance ($p = 0.18$).

In terms of structural changes, Al Atroush et al.[26] and Seleem et al.[19] reported elevated LA volume index (patients: 39.13 ± 4.6 ml/m^2 vs. controls: 21.53 ± 3.85, $p < 0.001$), and LV mass index (patients: 69.33 ± 12.78 g/m^2 vs. controls: 56.25 ± 8.65, $p < 0.001$). Dadhich et al.[23] highlighted that all four cardiac chambers were significantly enlarged in ascitic cirrhotics, with left atrial enlargement being the most prominent ($p < 0.0001$).

4.5. QTc Interval and Electrophysiological Abnormalities

QTc interval had a mean of 443.13 ± 26.87 ms and prolonged in 62% in our study. Behera et al.[21]reported a mean QTc of 474 ± 66 ms, significantly longer in CCM (0.49 ± 0.05 s) vs. non-CCM (0.432 ± 0.07, $p = 0.0016$). Chandey et al.[24] showed that QTc was progressively prolonged with worsening liver disease: Class A (425 ± 20.97 ms), Class B (437.35 ± 42.6 ms), and Class C (479.71 ± 29.48 ms, $p = 0.04$). They also demonstrated higher QTc with bilirubin >3 mg/dL (477.75 ± 32.32 ms, $p = 0.000$) and hypoalbuminemia <2.8 g/dL (461.87 ± 41.94 ms, $p = 0.001$). Pimentel et al[22]. found a mean QTc of 445 ± 29 ms in cirrhotics vs. 429 ± 19 ms in controls ($p = 0.04$), with significantly prolonged values in Child B and C vs. A ($p = 0.003$, $p = 0.02$).

4.6. Correlation with Liver Disease Severity

Our study confirmed significant correlations between worsening liver function and cardiac abnormalities. EF, E/A ratio, and structural parameters deteriorated progressively across Child-Pugh classes. Similar trends were seen in Chandey,[24]Behera [21], Shweta Patil [18], and Pimentel [22], all of whom demonstrated that higher MELD or Child-Pugh scores were strongly associated with increased prevalence of CCM, QTc prolongation, or diastolic dysfunction. Farid Ud Din et al.[20] and Mansoor et al.[25] also emphasized that although conventional comorbidities (diabetes, HTN) did not significantly influence CCM, severity of hepatic dysfunction did.

5. Conclusion

Our study demonstrates a significant association between worsening liver function and the prevalence of cirrhotic cardiomyopathy in a cohort specifically free from common cardiovascular comorbidities such as diabetes and hypertension. Key findings include QTc prolongation, reduced ejection fraction, E/A ratio alterations, and left atrial dilation—all of which correlated with advancing Child-Pugh class. These results affirm that cardiac dysfunction in cirrhosis is a direct hepatic consequence, not merely a reflection of systemic comorbidity. Early recognition and routine cardiac evaluation in cirrhotic patients, even those without conventional risk factors, are essential for comprehensive disease management and improving pre-transplant risk stratification.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Approved by the Institutional Ethical Committee of Al-Ameen Medical College & Hospital (Ref No: IEC/AMC/2023/103).

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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