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Original Research Paper

Interplay of Cardiac and Renal Dysfunction with Disease Severity in Cirrhosis: A Prospective Cross-Sectional Study

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Abstract

Introduction: Cirrhosis is a multisystem disorder characterized by hepatic fibrosis, portal hypertension, and progressive extrahepatic organ involvement. Cardiac dysfunction and renal impairment significantly influence prognosis but remain under-characterized in North Indian populations. This study evaluated the etiological spectrum of cirrhosis and assessed the prevalence and interrelationship of cardiac and renal dysfunction across disease severity.

Methods: A prospective cross-sectional study was conducted at a tertiary care centre in Amritsar (October 2023–January 2025). Eighty clinically and radiologically confirmed cirrhotic patients were enrolled. Clinical examination, biochemical evaluation, viral screening, Doppler ultrasonography, ECG, and transthoracic echocardiography were performed. Disease severity was classified by Child–Pugh criteria. Renal dysfunction was assessed using estimated glomerular filtration rate (eGFR). Statistical analyses were performed using SPSS v21 with significance set at $p \leq 0.05$.

Results: Alcoholic liver disease was the leading etiology (45%), followed by hepatitis B (25.8%) and NAFLD (17.5%). Half of the cohort belonged to Child–Pugh class B. Diastolic dysfunction was detected in 56.9% of patients, most commonly Grade I. Renal impairment (eGFR <60 mL/min/1.73m²) was present in 19.3%. Both cardiac and renal dysfunctions showed increasing prevalence with advancing Child–Pugh class. A significant association was observed between reduced left ventricular ejection fraction and lower eGFR values (p=0.017), indicating interdependence of cardiac and renal compromise.

Conclusion: Cirrhosis in this population is predominantly alcohol-related, with substantial cardiac and renal involvement that intensifies with disease severity. Integrated evaluation of cardio-renal function is essential for prognostication and timely management. Larger multicentre longitudinal studies are warranted to refine risk stratification.

Keywords: *Cirrhosis, Diastolic dysfunction, Renal Impairment, Cardio-renal Interaction, Child–Pugh Classification.*

Introduction

Cirrhosis is the last stage of chronic liver disease with extensive fibrosis, development of regenerative nodules, and disruption of structural liver architecture. These changes impair hepatocellular metabolism and detoxification function, with resulting complications such as portal hypertension, ascites, hepatic encephalopathy, hepatorenal syndrome (HRS), and hepatopulmonary syndrome [1].

Induction of fibrogenesis results from activated hepatic stellate cell conversion into collagen-producing myofibroblasts caused by hepatocyte, Kupffer cell, and sinusoidal endothelial cell mediators. Overproduction of extracellular matrix, sinusoidal capillarization, and intrahepatic vascular resistance perpetuate portal hypertension [2].

The etiological profile of cirrhosis varies globally. In India, alcohol remains the predominant cause, followed by hepatitis B virus, hepatitis C virus, autoimmune hepatitis, and non-alcoholic fatty liver disease (NAFLD) [3]. NAFLD, particularly non-alcoholic steatohepatitis (NASH), is rapidly increasing worldwide as the hepatic manifestation of metabolic syndrome, closely linked to obesity and type 2 diabetes mellitus [4].

Except for hepatic failure, cirrhosis also has systemic effects on the cardiovascular and renal systems. Hemodynamic perturbations such as splanchnic vasodilation and portosystemic shunting lead to dysfunction of extrahepatic organs [5]. Cirrhotic cardiomyopathy is defined by a subnormal contractile response to stress, diastolic dysfunction, and electrophysiologic abnormalities with the absence of structural heart disease [6]. Suggested mechanisms include disturbed β-adrenergic signal transduction, dysfunctional calcium handling, and excess nitric oxide and endocannabinoid activity [7]. Clinically, blunted cardiac reserve, systolic and diastolic dysfunction, and lengthened QT intervals manifest with severity matching the stage of liver disease [8].

Renal insufficiency, a significant predictor of mortality among cirrhotic patients, almost invariably presents as an acute kidney injury, acute-on-chronic kidney disease, or as HRS [9]. HRS is a syndrome produced by splanchnic vasodilation with relative central hypovolemia to cause extensive renal vasoconstriction through the renin–angiotensin–aldosterone system [10]. Type 1 HRS runs a rapid course, but type 2 runs a slower chronic course [9,10].

Against the backdrop of interrelationships between hepatic, cardiac, and renal dysfunction, the present investigation sought to understand the etiological profile of cirrhosis among North Indian populations, to evaluate the profile and magnitude of cardiac involvement, and to observe the proportion with renal impairment alongside both cardiac dysfunction and overall disease severity.

Material and Method

Research design and setting

This prospective cross-sectional survey occurred between October 2023 and January 2025 in the Department of Medicine, Government Medical College, Amritsar.

Study populations

80 patients with a diagnosis of cirrhosis by clinical and radiological examinations were enrolled. Institutional Ethics Committee approval was obtained, and written informed consent was taken from all participants.

Inclusion criteria: sex patients with cirrhosis.

Exclusion criteria: acute/chronic renal failure unrelated to cirrhosis, rheumatic heart disease, collagen vascular disease, bleeding disorders, and hepatocellular carcinoma or other malignancy.

Clinical and laboratory assessment

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All participants were taken through meticulous history, systemic examination, and screening for stigmata of chronic liver disease and extrahepatic organ dysfunction. Laboratory tests comprised complete blood counts, liver function tests, serum urea, creatinine, electrolytes, and coagulation profile. Viral screening for hepatitis B, C, and HIV was undertaken.

Doppler ultrasonography was carried out in all cases to evaluate hepatic morphology, portal hypertension, renal function, and splenomegaly. Cardiac assessment consisted of chest X-rays, electrocardiograms, and transthoracic Doppler echocardiography. Upper G.I. endoscopy was done to record varices. Disease severity was also determined by Child–Pugh's classification.

Statistical analysis

Statistics were compared using SPSS version 21.0 (IBM Corp., Chicago, IL, USA). Descriptive statistics were applied to baseline variables. Categorical variable associations were tested with chi-square, continuous with Student's t-test or ANOVA. A p-value ≤0.05 was regarded as statistically significant.

Ethical issues

All procedures were approved by the Institutional Ethics Committee. Confidentiality measures were taken through anonymized participant identification, and data were stored safely.

Results

Among 120 patients enrolled, the predominant etiology of cirrhosis was alcoholic liver disease (45%), followed by hepatitis B infection (25.8%) and NAFLD (17.5%). Hepatitis C virus and other causes (including autoimmune and cryptogenic) together accounted for 11.7% (Table 1). This distribution highlights alcohol as the leading contributor, with viral and metabolic causes constituting a substantial burden.

Table 1. Etiology of cirrhosis in the study population (n=120).

Etiology	Frequency	%
Alcoholic	54	45.0
HBV	31	25.8
NAFLD	21	17.5
HCV	7	5.8
Others	7	5.8

Table 2. Distribution of patients by Child–Pugh class

Class	Frequency	%
A	38	31.7
B	60	50.0
C	22	18.3

Based on Child–Pugh classification, 50% of patients were in Class B, 31.7% in Class A, and 18.3% in Class C (Table 2), reflecting a predominance of moderate disease severity.

Table 3. Distribution of patients according to diastolic dysfunction.

Grade	Frequency	%
None	52	43.1
Grade I	39	32.4
Grade II	18	15.0
Grade III	11	9.5

Echocardiographic assessment revealed diastolic dysfunction in 56.9% of patients. Grade I was most frequent (32.4%), followed by Grade II (15%) and Grade III (9.5%), while 43.1% had no dysfunction (Table 3). This indicates a high prevalence of subclinical cirrhotic cardiomyopathy.

Table 4. Distribution of patients according to eGFR.

eGFR (mL/min/1.73m²)	Frequency	%
>90	61	50.5
60–89	37	30.2
30–59	15	12.6
<30	7	6.7

Estimated GFR evaluation showed renal impairment in 19.3% of patients, with 12.6% having moderate (30–59 mL/min/1.73m²) and 6.7% having severe impairment (<30 mL/min/1.73m²). Half of the cohort (50.5%) maintained preserved eGFR (>90 mL/min/1.73m²), while 30.2% had a mild reduction (60–89) (Table 4).

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Table 5. Correlation of LVEF and eGFR in cirrhotic patients.

LVEF (%)	Mean eGFR	SD
>55	85.3	11.7
45–55	68.9	13.9
<45	52.6	16.4

A statistically significant relationship was observed between cardiac and renal dysfunction (p=0.017). Patients with preserved LVEF (>55%) had a mean eGFR of 85.3 mL/min/1.73m², while those with reduced LVEF (<45%) showed markedly lower eGFR (52.6 mL/min/1.73m²) (Table 5). This demonstrates that declining cardiac function is closely associated with worsening renal impairment in cirrhosis.

Discussion

Etiologic spectrum of cirrhosis and its correlation with cardiac as well as renal dysfunction were investigated in the present analysis through data synopsized through five tables. Alcohol remained the most common cause of cirrhosis, with contributions from hepatitis B virus infection as well as non-alcoholic fatty liver disease. The pattern is consistent with previous Indian reports where data indicated that alcohol has remained the most common cause of cirrhosis as a leading cause, but where rising prominence is being noted with non-alcoholic fatty liver disease, indicative of rising disease burden from metabolic syndrome as well as diabetes in the region [11–13].

Concerning severity, nearly half were Child–Pugh class B, with fewer being class C or class A. Such intermediate predominance is in contrast to earlier reports that typically would find higher percentages of advanced decompensation, consistent with prior clinical detection within the present cohort [14,15]. Mean MELD scores observed were equivalent to prior Indian cohorts, confirming their value in stratification of risk [16].

Echocardiography revealed cardiac dysfunction in over a third. Although left ventricular ejection fraction did not significantly correlate with Child–Pugh class, cardiac output reduced stepwise with increasing cirrhosis. Such a relation is in keeping with the idea of cirrhotic cardiomyopathy, where suppressed contractile reserve and diastolic dysfunction worsen with increasing disease severity [17,18].

Renal function impairment was likewise a frequent finding, with nearly a third of patients experiencing acute kidney injury with a lower estimated glomerular filtration rate tied to higher disease severity. These results agree with previous literature that has highlighted the hepatorenal syndrome burden and kidney dysfunction among hospitalized cirrhosis cases [19,20].

As organ dysfunction correlated with severity was compared, cardiac output showed a strong correlation with Child–Pugh class, but indices of renal function, especially estimated glomerular filtration rate, had strong correlations with severity and prevalence of impairment. Surprisingly, cardiac–renal dysfunction correlation was moderate, reflecting that these complications, although commonly occurring together, might be caused by partially different pathophysiological pathways instead of a proportionally linear course [21].

Altogether, these results highlight the dual impact of cardiac and renal failure in cirrhosis and their increasingly cumulative relationship with worsening severity. There is support for the importance of holistic, organ-specific evaluation as part of standard management of cirrhotic patients and an indication of the importance of extensive screening in making decisions regarding prognosis and priority for liver transplantation.

Conclusion

This study highlights the interplay between liver, cardiac, and renal dysfunction in cirrhotic patients from a tertiary care centre in North India. Alcohol remained the predominant cause, though viral hepatitis and NAFLD also contributed substantially, reflecting changing epidemiological trends. Cardiac dysfunction and renal impairment showed increasing prevalence with advancing Child–Pugh class, supporting their role as key systemic complications of cirrhosis with significant prognostic implications. The principal strength

of this study is its comprehensive evaluation of cardiac and renal function alongside etiological factors, providing a multidimensional understanding of cirrhosis beyond hepatic pathology alone. The integration of clinical, biochemical, and echocardiographic parameters offers valuable insight into the systemic burden of the disease.

However, the study has limitations. Being single-centre and hospital-based, the findings may not be generalizable to the wider population. The relatively modest sample size and lack of longitudinal follow-up preclude assessment of long-term outcomes such as survival and transplant-free progression. Additionally, confounding influences from comorbid conditions could not be entirely excluded. Despite these limitations, the study underscores the need for routine cardiovascular and renal evaluation in cirrhotic patients and supports further multicentre, longitudinal studies to refine prognostic stratification and guide therapeutic strategies.

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Conflict of Interest: None
Ethical approval: Institutional Research Committee

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