Original Article
DOI: https://doi.org/10.70749/ijbr.v3i7.1825

INDUS JOURNAL OF BIOSCIENCE RESEARCH
https://ijbr.com.pk
ISSN: 2960-2793/ 2960-2807

Frequency of Systolic and Diastolic Dysfunction in Patients Presenting with Liver Cirrhosis Secondary to Hepatis-C at Tertiary Care Hospital, Karachi

Lubna Gul[1], Rakshanda Jabeen[1], Ayesha Ejaz[1], Soniya[1], Guriya[1], Muhammad Zubair[1]

[1]Department of Medicine-Ward 6, Jinnah Postgraduate Medical Centre, Karachi, Pakistan

ARTICLE INFO

Keywords: Cirrhotic cardiomyopathy, systolic dysfunction, diastolic dysfunction, liver cirrhosis, echocardiography, Child-Pugh classification.

Correspondence to: Lubna Gul,
Department of Medicine Ward 6, Jinnah Postgraduate Medical Centre, Karachi, Pakistan.
Email: linawahab575@gmail.com

Declaration
Authors' Contribution: All authors equally contributed to the study and approved the final manuscript.

Conflict of Interest: No conflict of interest.

Funding: No funding received by the authors.

Article History
Received: 30-05-2025 Revised: 27-06-2025
Accepted: 03-07-2025 Published: 15-07-2025

ABSTRACT

Objective: To determine the frequency of systolic and diastolic dysfunction in patients presenting with liver cirrhosis secondary to hepatis C at Tertiary Care Hospital, Karachi. **Methods:** This cross-sectional study included 173 patients with liver cirrhosis admitted to a tertiary care hospital. Systolic and diastolic dysfunction were evaluated using echocardiography, with systolic dysfunction defined as an ejection fraction <55% and diastolic dysfunction identified by E/A ratio <1. Clinical parameters, including age, gender, cirrhosis duration, and Child-Pugh classification, were analyzed for their relationship with cardiac dysfunction. **Results:** Systolic dysfunction was present in 26.6% of patients, while 21.4% exhibited diastolic dysfunction. There were no significant associations between cardiac dysfunction and age, gender, cirrhosis duration, or Child-Pugh classification ($p > 0.05$). These findings suggest that cardiac dysfunction in cirrhosis develops independently of disease severity. **Conclusion:** Cirrhotic cardiomyopathy is common and may be underdiagnosed in cirrhotic patients, regardless of clinical severity. Routine cardiac screening, including echocardiography and ECG, should be considered for all cirrhotic patients to detect early dysfunction and prevent complications. Further research is needed to explore long-term outcomes and potential therapeutic strategies.

INTRODUCTION

Hyperdynamic circulation is a recognized clinical condition commonly observed in individuals with cirrhosis and portal hypertension.[1] It is defined by an elevated heart rate and cardiac output, along with decreased systemic vascular resistance and lower arterial blood pressure. [2] The primary driver of hyperdynamic circulation in patients with cirrhosis is the dilation of peripheral and splanchnic blood vessels. This occurs due to enhanced production and activity of vasodilatory agents, including nitric oxide (NO), carbon monoxide (CO), and endogenous cannabinoids, as well as a diminished vascular response to vasoconstrictive stimuli. [3]

In patients with cirrhosis, left ventricular ejection fraction (LVEF), an indicator of systolic function, has been observed to remain normal at rest. [4] In contrast, a diminished LVEF has been observed following various stressors, including physical exertion, sodium load, or an upright posture. This reduction is attributed to a weakened heart rate response to stress, decreased myocardial reserve, and impaired oxygen extraction by muscle tissue. [5-6] Studies in both human and animal models have reported a reduced preload reserve in response to different loading conditions in cirrhosis. Additionally, research has shown that diastolic dysfunction in patients with ascites improves following paracentesis. [7-8] Diastolic dysfunction in cirrhosis may result from cardiac hypertrophy, localized fibrosis, and subendothelial edema. On Doppler echocardiography, it is identified by a reduced E/A ratio (below 1), which represents the ratio of early to late atrial phases of ventricular filling. This decrease is particularly evident in cirrhotic patients, especially those with ascites. [9-10] Diastolic dysfunction, characterized by impaired passive and active filling of the left ventricle during diastole, results in an inadequate increase in stroke volume in response to various stimuli. This dysfunction may contribute to the onset of heart failure. [11] Diastolic dysfunction may develop before systolic dysfunction in cirrhosis and could be a key factor in the reduced physical activity observed in cirrhotic patients. Furthermore, it

likely plays a role in the development of fluid retention in these individuals. [12] Shaikh et al found systolic and diastolic dysfunction to be 33.8% and 20.3%. [13]

Among the factors that contribute to cirrhotic cardiomyopathy, systolic and diastolic dysfunction are known to be an early marker of cardiac dysfunction in Liver cirrhosis patients. Moreover, the prognosis of patients with cardiac involvement is poor. There is paucity of local literature on this subject. Given the fact that knowledge about systolic and diastolic dysfunction can impact the outcome of the patients and improve their quality of life gives us a strong rational to conduct this study it has not been well investigated. Hence data from this study would help us in formulating a preventive, screening and management plan.

MATERIAL AND METHODS

This descriptive cross-sectional study was conducted at the Department of Medicine, JPMC, Karachi, from 25 November 2024 to 25 May 2025 after approval from the College of Physicians and Surgeons Pakistan. The study aims to assess the prevalence of systolic and diastolic dysfunction in patients with liver cirrhosis. A total of 173 patients were included based on a calculated sample size, considering a diastolic dysfunction prevalence of 20.3%, a margin of error of 6%, and a 95% confidence level. The sampling technique employed was non-probability consecutive sampling.

Patients eligible for inclusion was liver cirrhosis for at least six months, confirmed by ultrasound findings such as shrunken liver with a longitudinal diameter of the right and left lobes measuring <90 mm and 70 mm, respectively, nodular liver surface, coarsened liver echo texture, ascites (>100 mL), or portal hypertension indicated by a portal vein diameter >13 mm. Participants were between 40 and 80 years of age, of either gender, and classified as Child-Pugh class A. Patients were excluded if they have a history of malignancy, nephropathy, valvular heart disease, acute coronary syndrome, asthma, COPD, chronic renal failure, congestive heart failure, stroke, hypo- or hyperthyroidism, HIV, or hepatitis B. Pregnant patients, identified through history and confirmed by dating scan, were excluded.

After obtaining approval from the institutional ethical review committee, informed consent was secured from all participants, ensuring confidentiality throughout the study. Demographic details, including age, gender, and cirrhosis duration, was documented. A consultant cardiologist with at least 10 years of echocardiography experience performed echocardiographic assessments. Systolic dysfunction was defined as an ejection fraction of ≤55%, while diastolic dysfunction was identified based on a reduced mitral E/A ratio of <1. The presence of systolic or diastolic dysfunction was recorded as per operational definitions. Data was collected using a structured proforma.

Statistical analysis was conducted using SPSS Version 20. Mean and standard deviation will be calculated for normally distributed quantitative variables such as age and cirrhosis duration, while median and interquartile range was reported for non-normally distributed data. Categorical variables, including gender, Child-Pugh classification (A/B/C), systolic dysfunction, and diastolic dysfunction, was presented as frequencies and percentages. Effect modifiers were controlled by stratifying data based on age, gender, Child-Pugh classification, and cirrhosis duration. Post-stratification analysis was performed using the chi-square test or Fisher's exact test, with a p-value of ≤0.05 considered statistically significant.

RESULTS

The study included 173 patients with liver cirrhosis. Most participants (68.2%) were between 61 to 80 years old, while 31.8% were between 40 to 60 years. Gender distribution was nearly equal, with 48% male and 52% female patients.

Among the participants, 26.6% had systolic dysfunction, while 73.4% had normal systolic function. Diastolic dysfunction was present in 21.4%, leaving 78.6% without it. Regarding cirrhosis duration, 64.2% had the disease for more than 12 months, while 35.8% had it for a year or less. Based on Child-Pugh classification, 31.8% were in Class A, 39.9% in Class B, and 28.3% in Class C, indicating that many patients had moderate to severe liver disease.

Among those with systolic dysfunction (n = 46), 28.8% were aged 61 to 80 years, and 21.8% were between 40 to 60 years. Patients without systolic dysfunction (n = 127) had a similar age distribution, and the difference was not statistically significant (p = 0.33). Men and women showed no significant difference in systolic dysfunction rates (p = 0.17). It affected 31.3% of males and 22.2% of females, while 68.7% of males and 77.8% of females had preserved systolic function. Cirrhosis duration also showed no significant association with systolic dysfunction (p = 0.21). Among those with the disease for more than 12 months, 29.7% had systolic dysfunction, compared to 21% of those with a shorter disease course. Child-Pugh classification did not significantly impact systolic dysfunction either (p = 0.68). Systolic dysfunction was present in 30.9% of Class A, 24.6% of Class B, and 26.6% of Class C patients, while the majority in each group did not develop it.

Among the 37 patients (21.4%) with diastolic dysfunction, 23.7% were between 61 to 80 years old, and 16.4% were 40 to 60 years old. The difference between age groups was not significant (p = 0.27). Men and women had similar rates of diastolic dysfunction (p = 0.30). It was present in 18.1% of males and 24.4% of females, while 81.9% of males and 75.6% of females had normal diastolic function. Cirrhosis duration had no clear link to diastolic dysfunction (p = 0.62). Among patients with cirrhosis for 12 months or less, 19.4% had diastolic dysfunction, compared to 22.5% of those with longer disease duration. Child-Pugh classification also showed no significant relationship (p = 0.95). Diastolic dysfunction was observed in 20% of Class A, 21.7% of Class B, and 22.4% of Class C patients, with most patients in each group remaining unaffected.

Systolic and diastolic dysfunction did not show significant associations with age, gender, cirrhosis duration, or Child-Pugh classification. These findings suggest that cardiac dysfunction in cirrhotic patients may develop independently of these factors, reinforcing the need for regular cardiovascular evaluation in this population.

Table 1
Distribution of Baseline Characteristics among the Study Participants.

Variables		n (%)
Age	40 to 60 years	55 (31.8)
	61 to 80 years	118 (68.2)
Gender	Male	83 (48)
	Female	90 (68.2)
Duration of Cirrhosis	≤ 12 months	62 (35.8)
	> 12 months	111 (64.2)
Child Pugh Score	A	55 (31.8)
	B	69 (39.9)
	C	49 (28.3)
Systolic dysfunction	Yes	46 (26.6)
	No	127 (73.4)
Diastolic dysfunction	Yes	37 (21.4)
	No	136 (78.6)
Total		173 (100)

Table 2
Distribution of Patient Characteristics according to the Systolic Dysfunction Groups.

Variables		Systolic dysfunction (Yes) n (%)	Systolic dysfunction (No) n (%)	P value
Age	40 to 60 years	12 (21.8)	43 (78.2)	0.33
	61 to 80 years	34 (28.8)	84 (71.2)	
Gender	Male	26 (31.3)	57 (68.7)	0.17
	Female	20 (22.2)	70 (77.8)	
Duration of Cirrhosis	≤ 12 months	13 (21)	49 (79)	0.21
	> 12 months	33 (29.7)	78 (70.3)	
Child Pugh Score	A	17 (30.9)	38 (69.1)	0.68
	B	17 (24.6)	52 (75.4)	
	C	12 (26.6)	37 (75.5)	

Table 3
Distribution of Patient Characteristics according to the Diastolic Dysfunction Groups.

Variables		Diastolic dysfunction (Yes) n (%)	Diastolic dysfunction (No) n (%)	P value
Age	40 to 60 years	09 (16.4)	46 (83.6)	0.27
	61 to 80 years	28 (23.7)	90 (76.3)	
Gender	Male	15 (18.1)	68 (81.9)	0.30
	Female	22 (24.4)	68 (75.6)	
Duration of Cirrhosis	≤ 12 months	12 (19.4)	50 (80.6)	0.62
	> 12 months	25 (22.5)	86 (77.5)	
Child Pugh Score	A	11 (20)	44 (80)	0.95
	B	15 (21.7)	54 (78.3)	
	C	11 (22.4)	38 (77.6)	

DISCUSSION

This study highlights the prevalence of cardiac dysfunction in cirrhotic patients and its connection to clinical characteristics. Cirrhotic cardiomyopathy, a well-documented but often overlooked condition, affects both systolic and diastolic function. [14] In our study, 26.6% of patients had systolic dysfunction, while 21.4% showed diastolic dysfunction, consistent with previous research. [15] We found no significant difference in cardiac dysfunction across age groups. This suggests that cirrhotic cardiomyopathy is not strictly age-dependent, a finding supported by earlier studies. [16] Similarly, gender had no impact on dysfunction prevalence, reinforcing the idea that cirrhosis-related heart issues are not gender-specific. [17]

Cirrhosis duration also showed no strong link to cardiac dysfunction. Some researchers have proposed that cirrhotic cardiomyopathy results from functional rather than structural changes, meaning heart impairment can occur at any stage. [18] Our findings support this, suggesting that hepatic dysfunction alone may be enough to trigger cardiac abnormalities. [19]

Surprisingly, we observed no clear connection between cirrhosis severity (Child-Pugh classification) and cardiac dysfunction. While some studies report higher cardiac dysfunction rates in advanced cirrhosis, our results do not confirm this pattern. [20] Differences in diagnostic methods, sample sizes, or patient demographics may explain these discrepancies. [21]

Cirrhotic cardiomyopathy often coexists with hepatorenal syndrome, where heart dysfunction worsens kidney function. [22] Acute kidney injury (AKI) is a frequent complication in cirrhotic patients, usually driven by sepsis, volume loss, and nephrotoxic drugs. Research has shown that renal and cardiac impairments often go hand in hand, creating a vicious cycle. [23] This underlines the need for early cardiovascular evaluation in cirrhotic patients, especially those at risk of kidney complications. [24]

Recent evidence suggests that inflammation, autonomic dysfunction, and hemodynamic changes drive cirrhotic cardiomyopathy. [25] Liver dysfunction increases nitric oxide and endotoxin levels, leading to vasodilation, reduced cardiac contractility, and QT prolongation. [26] These findings reinforce the idea that multiple systemic factors contribute to heart abnormalities in cirrhosis. [27]

Interestingly, our findings suggest that cardiac dysfunction may begin even in early-stage cirrhosis, before noticeable symptoms appear. This aligns with studies that recommend proactive screening for cardiac issues using echocardiography, ECG, and biomarkers like pro-BNP. [28]

Given the high prevalence of cardiac dysfunction in cirrhosis, routine cardiovascular screening should be standard practice. Physicians should use echocardiograms and ECGs, especially in patients undergoing TIPS procedures or liver transplantation. Identifying heart dysfunction early can improve treatment and overall outcomes. [27-28]

Limitations

This study has several limitations. Conducting it in a single tertiary care hospital may limit how well the findings apply to broader populations, especially those in primary care or rural settings, where patients might have different risk factors and disease patterns.

We assessed cardiac function at rest, which might not fully capture cirrhotic cardiomyopathy. Since many patients only show abnormalities during stress or exertion, our study may have underestimated the true prevalence of dysfunction. A more comprehensive evaluation, including stress echocardiography, could provide a clearer picture.

The study also lacks long-term follow-up, making it impossible to track how systolic and diastolic dysfunction progress over time or how they affect clinical outcomes. Future research should explore heart failure progression, mortality risk, and response to treatment in cirrhotic patients.

Additionally, we did not include biomarkers like high-sensitivity troponin, NT-proBNP, or inflammatory markers, which could have provided deeper insight into the mechanisms behind cirrhotic cardiomyopathy. These

markers could help in early diagnosis and risk assessment in future studies.

Certain confounding factors, such as diabetes, hypertension, obesity, medication use (beta-blockers, diuretics), and lifestyle habits, were not fully accounted for. These conditions may influence cardiac function independently of cirrhosis, and future research should carefully assess their role.

Despite these limitations, the study provides valuable insights into cardiac dysfunction in cirrhotic patients and highlights the importance of routine cardiovascular screening. Future multicenter, prospective studies with larger samples and advanced cardiac assessments can help refine these findings and improve patient care.

CONCLUSION

This study confirms that cardiac dysfunction is common in cirrhotic patients, regardless of age, gender, cirrhosis duration, or severity. These findings highlight the need for routine cardiovascular monitoring across all cirrhotic patients. Further research should explore underlying mechanisms and possible treatment strategies to reduce the burden of cirrhotic cardiomyopathy.

REFERENCES

1. Moller S, Henriksen JH. Cardiovascular complications of cirrhosis. Gut. 2008;57:268–78. https://doi.org/10.1136/gut.2006.112177
2. Sola E, Gines P. Renal and circulatory dysfunction in cirrhosis: current management and future perspectives. J Hepatol. 2010;53:1135–45. https://doi.org/10.1016/j.jhep.2010.08.001
3. Martell M, Coll M, Ezkurdia N, Raurell I, Genesca J. Physiopathology of splanchnic vasodilation in portal hypertension. World J Hepatol. 2010;2:208–20. https://doi.org/10.4254/wjh.v2.i6.208
4. Moller S, Krag A, Bendtsen F. Kidney injury in cirrhosis: pathophysiological and therapeutic aspects of hepatorenal syndromes. Liver Int. 2014;34:1153–63. https://doi.org/10.1111/liv.12549
5. Al-Hamoudi WK. Cardiovascular changes in cirrhosis: pathogenesis and clinical implications. Saudi J Gastroenterol. 2010;16:45–153. https://doi.org/10.4103/1319-3767.65181
6. Bosch J, Abraldes JG, Fernandez M, Garcia-Pagan JC. Hepatic endothelial dysfunction and abnormal angiogenesis: new targets in the treatment of portal hypertension. J Hepatol. 2010;53:58–567 https://doi.org/10.1016/j.jhep.2010.03.021
7. Garcia-Tsao G. Bacterial translocation: cause or consequence of decompensation in cirrhosis? J Hepatol. 2001;34:50–155. https://doi.org/10.1016/s0168-8278(00)00006-4
8. Fede G, Privitera G, Tomaselli T, Spadaro L, Purrello F. Cardiovascular dysfunction in patients with liver cirrhosis. Ann Gastroenterol. 2015 Jan-Mar;28(1):31-40. https://doi.org/10.1016/j.dld.2015.08.006
9. Stundiene I, Sarnelyte J, Norkute A, Aidietiene S, Liakina V, Masalaite L, Valantinas J. Liver cirrhosis and left ventricle diastolic dysfunction: Systematic review. World J Gastroenterol. 2019 Aug 28;25(32):4779-4795. https://doi.org/10.3748/wjg.v25.i32.4779
10. Carvalheiro F, Rodrigues C, Adrego T, Viana J, Vieira H, Seco C et al. Diastolic Dysfunction in Liver Cirrhosis: Prognostic Predictor in Liver Transplantation? Transplant Proc. 2016;48:128–31. https://doi.org/10.1016/j.transproceed.2016.01.010
11. Ruiz-del-Árbol L, Serradilla R. Cirrhotic cardiomyopathy. World J Gastroenterol. 2015;21:11502–521. https://doi.org/10.3748/wjg.v21.i41.11502
12. Fede G, Privitera G, Tomaselli T, Spadaro L, Purrello F. Cardiovascular dysfunction in patients with liver cirrhosis. Ann Gastroenterol. 2015;28:31–40. https://doi.org/10.1016/j.dld.2015.08.006
13. Shaikh S, Abro M, Qazi I, Yousfani A. Frequency of cirrhotic cardiomyopathy in patients with cirrhosis of liver: A tertiary care hospital experience. Pak J Med Sci 2011;27(4):44-748.
14. Naqvi R. Epidemiological trends in community-acquired acute kidney injury in Pakistan: 25 years experience from a tertiary care renal unit. Pak J Med Sci. 2021;37(2):312-319. https://doi.org/10.12669/pjms.37.2.3876
15. Arshad A, Ayaz A. Prevalence of risk factors of acute kidney injury in a tertiary care hospital in Pakistan. J Pak Med Assoc. 2020;70(8):1439-1444. https://doi.org/10.5455/jpma.20286
16. Bernardi M, Ricci CS, Sica G, et al. Cirrhotic cardiomyopathy: The relationship between liver and heart. World J Gastroenterol. 2020;26(21):2611-2624. https://doi.org/10.1002/cld.141
17. Wong F. Cirrhotic cardiomyopathy. Hepatol Int. 2009;3(4):515-519.
18. Henriksen JH, Moller S. Cirrhotic cardiomyopathy: Pathogenic mechanisms and clinical relevance. Nat Rev Gastroenterol Hepatol. 2014;11(3):177-186. https://doi.org/10.1038/nrgastro.2013.210
19. Baik SK, Fouad TR, Lee SS. Cirrhotic cardiomyopathy. Orphanet J Rare Dis. 2007;2:15.
20. Pozzi M, Carugo S, Boari G, et al. Evidence of functional and structural cardiac abnormalities in cirrhotic patients with and without ascites. Hepatology. 1997;26(5):1131-1137. https://doi.org/10.1053/jhep.1997.v26.pm0009362352
21. Moller S, Henriksen JH. Cardiovascular complications of cirrhosis. Gut. 2008;57(2):268-278.
22. Wong F, Girgrah N, Graba J, et al. The cardiac response to exercise in cirrhosis. Gut. 2001;49(2):268-275. https://doi.org/10.1136/gut.49.2.268
23. Liu H, Song D, Lee SS. Cirrhotic cardiomyopathy. Hepatol Int. 2008;2(3):294-304.
24. Myers RP, Lee SS. Cirrhotic cardiomyopathy and hepatorenal syndrome: The heart and kidney in liver failure. J Hepatol. 2005;42(2):19-24.
25. Zardi EM, Zardi DM, Dobrina A, et al. Cirrhotic cardiomyopathy: Pathophysiology and clinical implications. J Hepatol. 2010;53(5):982-990. https://doi.org/10.1007/978-1-4939-6377-5_35
26. Naschitz JE, Slobodin G, Lewis RJ, et al. Heart diseases affecting the liver and liver diseases affecting the heart. Am Heart J. 2000;140(1):111-120. https://doi.org/10.1067/mhj.2000.107177
27. Moller S, Bernardi M. Interactions of the heart and liver. Eur Heart J. 2013;34(36):2804-2811. https://doi.org/10.1093/eurheartj/eht246
28. Moller S, Henriksen JH. Cardiovascular dysfunction in cirrhosis. J Hepatol. 2010;53(5):719-729. https://doi.org/10.1136/gut.2006.112177