Al-Azhar International Medical Journal

Volume 4 | Issue 1 Article 23

1-2023

Tissue Doppler Imaging Assessment for left ventricular systolic and diastolic function in Patients with Different Grades of Liver Cirrhosis

Mohamed Hassan Alnadry
Hepatology, Gastroenterology and infectious disease department, Faculty of Medicine, Al-Azhar University, Egypt

Mohamed Zakaria Abu-Amer
Hepatology, Gastroenterology and infectious disease department, Faculty of Medicine, Al-Azhar University, Egypt

Abdel Hamid Ismail Abu Rahhal
Cardiology department, Faculty of Medicine, Al-Azhar University, Egypt

Mohamed Abdel Hamid Helmy Hassan
M.B.B.CH, Egypt, kindbird1978@gmail.com

How to Cite This Article

Alnadry, Mohamed Hassan; Abu-Amer, Mohamed Zakaria; Rahhal, Abdel Hamid Ismail Abu; and Hassan, Mohamed Abdel Hamid Helmy (2023) "Tissue Doppler Imaging Assessment for left ventricular systolic and diastolic function in Patients with Different Grades of Liver Cirrhosis," *Al-Azhar International Medical Journal*: Vol. 4: Iss. 1, Article 23.
DOI: https://doi.org/10.58675/2682-339X.1643

ORIGINAL ARTICLE

Tissue Doppler Imaging Assessment of Left Ventricular Systolic and Diastolic Function in Patients with Different Grades of Liver Cirrhosis

Mohamed Hassan Alnadry [a,*], Mohamed Zakaria Abu-Amer [a], Abdel Hamid Ismail Abu Rahhal [b], Mohamed Abdel Hamid Helmy Hassan [c]

[a] Department of Hepatology, Gastroenterology and Infectious Diseases, Al-Azhar University, Cairo, Egypt
[b] Department of Cardiology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt
[c] Department of Hepatogastroenterology and Infectious Diseases, Al-Azhar University, Cairo, Egypt

Abstract

Background: **Chronic cardiac disease, known as cirrhotic cardiomyopathy dysfunction, in cirrhotic patients is characterized by altered diastolic relaxation and/or impaired contractile reactivity to stress in the absence of other recognized cardiac diseases.**

Aim and objectives: **The purpose of the study was to evaluate tissue Doppler in the left ventricle's systolic and diastolic characteristic performance in patients with liver cirrhosis.**

Patients and methods: **This cross-sectional case−control study was done at Al-Hussien Hospital, Al-Azhar University. The study included 75 patients with liver cirrhosis and 25 healthy participants, who were divided into four groups: the control group included 25 healthy volunteers, group 1 included patients with Child A liver cirrhosis, and groups 2 and 3 included patients with Child B and Child C cirrhosis. Participants were assessed using transthoracic echocardiography, tissue collection, and left lateral decubitus. We followed ASE's Doppler echocardiography guidelines for left ventricular systole function.**

Results: **There was a statistically significant higher mean value in patient groups compared with the control group regarding myocardial perfusion imaging posterior wall diameter, myocardial perfusion imaging tissue Doppler imaging, mitral valve E/É ratio, DT (ms), and isovolumic relaxation time (ms). The average values of the EF by Simpson, average S wave, and EF by M mode all differed significantly. When comparing the case groups with the control group, a significant difference regarding MV E/A ratio was seen. A *P* value of 0.05 was used to determine this difference.**

Conclusion: **Compared with the control group, patients with liver cirrhosis exhibited significantly worse left ventricular systolic and diastolic functioning, and the decompensated patients showed a greater difference than the compensated patients.**

Keywords: Cardiomyopathy, Cirrhotic, Electrophysiological abnormalities

1. Introduction

Cirrhotic cardiomyopathy, also known as cirrhotic heart disease, is defined by three characteristics: diastolic dysfunction, lowered stress-induced contractile reactivity, and electrophysiological anomalies with a prolonged QT interval.[1] Left ventricular diastolic dysfunction (LVDD), one of the three factors that cause cirrhotic cardiomyopathy, is a recognized early indicator of cardiac dysfunction in patients with LC.[2] In contrast with compensated LC, decompensated LC has a greater rate of LVDD.[3]

However, LVDD cannot be detected by an ECG or Child−Pugh scores.

In fact, the only way to detect LVDD is via echocardiography.[4]

Accepted 4 September 2022.
Available online 15 May 2023

* Corresponding author.
E-mail address: prof_nadry@yahoo.com (M.H. Alnadry).

https://doi.org/10.58675/2682-339X.1643

Exercise, pharmacological stress, and volume challenge all limit the increase in cardiac output, which is a sign of systolic dysfunction in cirrhotic individuals.[5] In addition, patients with cirrhosis suffer from severe chronotropic incompetence, which is characterized by a markedly diminished cardiac response to exercise as a result of autonomic dysfunction and a decreased sensitivity to sympathetic stimulation.[6] Thickened left ventricular wall, fibrosis, edema in the subendocardium, and altered collagen structure have all been linked to diastolic dysfunction in cirrhotic individuals, which eventually affects relaxation.[7]

The purpose of the study was to evaluate tissue Doppler for the left ventricle's systolic and diastolic characteristic performance in patients with liver cirrhosis.

2. Patients and methods

A cross-sectional case–control study was done at Al-Hussien Hospital, Al-Azhar University. A total of 75 patients with liver cirrhosis participated in the trial, along with 25 healthy volunteers who were matched with the patients regarding ages and sexes. The participants were divided into four groups: 25 patients with Child A hepatic cirrhosis made up group 1, 25 patients with Child B hepatic cirrhosis made up group 2, 25 individuals with Child C hepatic cirrhosis made up group 3, and 25 healthy volunteers made up group 4, as the control group.

Inclusion criteria were any adult suffering from liver cirrhosis who was older than 18 years.

Exclusion criteria were patients with inherited or acquired heart disorders, those who have diabetes or high blood pressure, those with anemia or pregnancy who have hyperdynamic circulation, patients receiving medications that influence heart functioning such as beta-blockers, patients with renal or pulmonary disorders, patients with atrial fibrillation, bundle branch blockers, ischemic heart disease, and postcardiac surgery.

The selected patients were subjected to the following:

(1) Informed written consent.
(2) Full clinical assessment, including full history taking (including name, age, sex, smoking habit, alcohol, education level, residential area (urban/rural), occupation, and drug history); clinical examination of cirrhotic liver and manifestation of hepatic decompensation including ascites, lower limb edema, and jaundice; and clinical examination of cardiomyopathy such as vital signs (blood pressure – pulse-heart rate), generalized edema, and dyspnea.
(3) Routine laboratory investigations, including complete blood count; erythrocytic sedimentation rate; kidney function tests such as blood urea and blood creatinine; liver function test such as serum albumin levels, transaminase levels [including alanine transaminase (ALT) and aspartate aminotransferase (AST)], prothrombin time (PT) and concentration, bilirubin level (total and direct), and alkaline phosphatase; and blood glucose levels after eating and after fasting.
(4) Ultrasonographic evaluation: the following parameters were assessed in ultrasonographic evaluation: liver size, liver texture, splenic size, and portal vein diameter.
(5) ECG.
(6) Echocardiography.

Participants underwent a transthoracic echocardiographic standard echocardiography and tissue collection. They were kept in a left lateral decubitus posture for assessment.

We adhered to the guidelines for Doppler echocardiography provided by the American Society of Echocardiography to assess the function of the left ventricle during systole.

Evaluation of left ventricular diastolic performance: to examine the diastolic function of the left ventricle in the apical four-chamber view, a Doppler beam oriented in the flow direction and a sample volume of 1–2 mm positioned between the mitral leaflets were used.

2.1. Sample size calculation

This study was based on a study carried out by Abdel-Gawad and colleagues. Epi Info STATCALC was used to calculate the sample size by considering the following assumptions: 95% two-sided confidence level, with a power of 80%, α error of 5%, and odds ratio calculated = 1.215. The final maximum sample size taken from the Epi- Info output was 67. Thus, the sample size was increased to 75 participants to assume any dropout cases during follow-up. We included 25 age-matched and sex-matched healthy population as the control group.

2.2. Analytical statistics

Utilizing the statistical programme for social sciences, version 23.0, recorded data were examined (SPSS Inc., Chicago, Illinois, USA). In terms of quantitative data, mean, SD, and ranges were reported. Qualitative variables were also shown as percentages and numbers. Using the Shapiro–Wilk

Test and Kolmogorov–Smirnov tests, data were examined for normality.

3. Results

According to the demographic data in Table 1, there were no statistically significant differences between the groups ($P > 0.05$ nonsignificant).

Table 2 shows the results of laboratory data. There was a highly statistically significant difference between groups regarding AST (μl/l), which was higher in group 1; ALT (μl/l), which was higher in subgroup 2; PT (s), which was higher in subgroup 3; creatinine (mg/dl), which was higher in group 3; albumin (g/dl), which was higher in control group; hemoglobin (Hb) (g/dl), which was high in control group; bilirubin (mg/dl), which was higher in group 3; and random blood sugar (RBS) (mg/dl), which was higher in group 1 ($P < 0.05$ significant) (Table 3).

Table 3 shows a statistically significant higher mean value in the patient group compared with the control group regarding MPI PWD, MPI TDI, MV E/É ratio, DT (ms), and IVRT (ms) ($P < 0.05$ significant), whereas EF by Simpson, average S wave, EF by M mode, and MV E/A ratio were significantly decreased in the patient group compared with the control group ($P < 0.05$ significant).

Table 4 shows a statistically significant difference between groups regarding echocardiographic findings such as MPI PWD, MPI TDI, EF Simpson, average S wave, EF M mode, MV E/E′, MV E/A, DT (ms), and IVRT (ms) ($P < 0.05$ significant).

Table 5 shows a statistically significant higher mean value in compensated group compared with decompensated group regarding EF by Simpson, average S wave, EF by M mode, and MV E/A ratio ($P < 0.05$, significant), whereas MPI PWD, MPI TDI, MV E/É ratio, DT (ms) and IVRT (ms) were significantly decreased in compensated group compared with decompensated group ($P < 0.05$ significant).

Table 1. Comparison between patients and controls according to demographic data.

Demographic data	Patients group ($N = 75$)	Control group ($N = 25$)	Test value	P value
Age (years)				
Range	42–74	42–72	$t = -0.610$	0.543
Mean ± SD	56.62 ± 10.42	55.17 ± 9.87		
Sex [n (%)]				
Male	45 (60)	16 (64)	$\chi^2 = 0.014$	0.906
Female	30 (40)	9 (36)		

t, independent sample t test; χ^2, χ^2 test.
P value more than 0.05 (NS).

Table 6 shows that there were seven (9.3%) patients with LV systolic dysfunction.

Table 7 shows that there were 49 (65.3%) patients with LVDD, and of them, eight (32%) patients had Child A, 16 (64%) patients had Child B, and 25 (100%) patients had Child C.

4. Discussion

Al-Hussien Hospital, part of Al-Azhar University, served as the site of this cross-sectional case–control study. A total of 75 patients with liver cirrhosis participated in the trial, along with 25 healthy volunteers who were matched with the patients regarding age and sex.

The participants were divided into four groups: group 1 consisted of 25 patients with Child A hepatic cirrhosis, group 2 consisted of 25 patients with Child B hepatic cirrhosis, group 3 consisted of 25 individuals with Child C hepatic cirrhosis, and 25 healthy volunteers made up the control group. According to demographic data, there was no statistically significant difference among groups ($P > 0.05$, nonsignificant). The current study was supported by Ashmawy et al.,[8] who performed a cross-sectional case–control study to assess the left ventricular function in cirrhotic liver patients using TDI. Of the 90 people with liver cirrhosis, 30 had Child A, 30 had Child B, and 30 had Child C cirrhosis. In addition, 45 healthy volunteers served as the study's control group. According to demographic information, there was no statistically significant difference between the groups. Laboratory data such as AST (l/l) levels were compared among the groups. It was discovered that there were statistically significant differences among them. ALT (μl/l), PT (s), creatinine (mg/dl), albumin (g/dl), Hb (g/dl), bilirubin (mg/dl), and RBS (mg/dl) had statistically significant results ($P < 0.05$ significant). This is in agreement with the current study. Ashmawy et al.[8] revealed that there was a statistically significant difference in Child grade groups A, B, and C regarding laboratory data, including AST, ALT, PT, creatinine, albumin, Hb, bilirubin, and RBS. The same results were reported by Hassan and Elden,[9] who demonstrated that the differences among the groups (Child grades A, B, and C) were statistically significant regarding laboratory data, including AST, ALT, albumin, and bilirubin. In the current study, comparison between patients and controls according to echocardiographic findings revealed that regarding MPI PWD, MPI TDI, and other metrics, there was a statistically significant greater mean value in the patient group compared with the control group. MV E/É ratio, DT (ms), and IVRT (ms) showed a

Table 2. Comparison between the study subgroups regarding laboratory data.

Laboratory data	Subgroup I ($N = 25$)	Subgroup II ($N = 25$)	Subgroup III ($N = 25$)	Control group ($N = 25$)	Test value	P value
AST (µl/l)	51.34 ± 17.69*	24.21 ± 4.42†	15.35 ± 2.79†	31.07 ± 6.63‡	$F = 61.053$	<0.001**
ALT (µl/l)	81.73 ± 30.58*	101.86 ± 40.73†	85.30 ± 17.11*	28.33 ± 6.73‡	$H = 34.66$	<0.001**
PT (s)	15.01 ± 0.91*	17.58 ± 1.16†	21.28 ± 1.89‡	12.28 ± 0.65§	$F = 23.7.9$	<0.001**
Creatinine (mg/dl)	0.88 ± 0.27*	1.02 ± 0.23†	1.05 ± 0.19†	0.92 ± 0.20*	$F = 3.215$	0.026*
Albumin (g/dl)	3.89 ± 0.31*	3.17 ± 0.20†	2.20 ± 0.33†	4.11 ± 0.29§	$F = 225.24$	<0.001**
Hb (g/dl)	13.68 ± 0.97*	12.17 ± 0.78†	12.22 ± 0.84†	13.83 ± 1.18*	$F = 22.355$	<0.001**
Bilirubin (mg/dl)	1.73 ± 0.24*	2.52 ± 0.36†	6.94 ± 2.93‡	0.88 ± 0.17§	$H = 82.79$	<0.001**
RBS (mg/dl)	130.69 ± 12.67*	117.77 ± 16.50†	112.51 ± 18.82†	116.23 ± 14.62†	$F = 6.249$	<0.001**

One way analysis of variance test was performed and multiple comparisons between groups through post-hoc test: Tukey's test.
Kruskal–Wallis was performed and multiple comparison between groups through Mann–Whitney test.
ALT, alanine transaminase; AST, aspartate aminotransferase; Hb, hemoglobin; PT, prothrombin time; RBS, random blood sugar.
Means that do not share same symbol are significantly different at P value ($P < 0.05$).
*P value less than 0.05 (S).
**P value less than 0.001 (HS).

Table 3. Comparison between patients and controls according to echocardiographic findings.

Echocardiographic findings	Patient group ($N = 75$)	Control group ($N = 25$)	Test value	P value
MPI PWD	0.44 ± 0.04	0.41 ± 0.02	$t = 3.594$	<0.001**
MPI TDI	0.52 ± 0.07	0.48 ± 0.03	$t = 2.075$	0.041*
EF by Simpson	62.61 ± 7.45	66.48 ± 4.28	$t = 2.460$	0.016*
Average S wave	11.55 ± 2.20	12.85 ± 1.30	$t = 2.791$	0.006*
EF by M Mode	62.66 ± 7.67	67.02 ± 4.00	$t = 2.715$	0.008*
MV E/É ratio	15.27 ± 3.12	5.42 ± 1.05	$t = 15.451$	<0.001**
MV E/A ratio	0.77 ± 0.20	1.38 ± 0.12	$U = 14.382$	<0.001**
DT (ms)	239.50 ± 19.28	197.04 ± 12.55	$t = 10.290$	<0.001**
IVRT (ms)	114.31 ± 13.79	76.78 ± 5.92	$t = 13.174$	<0.001**

IVRT, isovolumic relaxation time; MPI, myocardial perfusion imaging; PWD, posterior wall diameter; *t*, independent sample *t* test; TDI, tissue Doppler imaging; *U*, Mann–Whitney test.
P value more than 0.05 (NS).
**P* value less than 0.05 (S).
***P* value less than 0.001 (HS).

significant difference ($P < 0.05$ significant). A significant decrease in mean values for EF by Simpson, the patient group's average S wave, EF by M mode, and MV E/A ratio was noted compared with the control group, with a P value of 0.05. In harmony with the current study, Ashmawy et al.[8] reported a statistically significant difference between patient group and control group regarding MPI PWD, MPI

Table 4. Comparison between the study subgroups according to echocardiographic findings.

Echocardiographic findings	Subgroup I ($N = 25$)	Subgroup II ($N = 25$)	Subgroup III ($N = 25$)	Control group ($N = 25$)	Test value	P value
MPI PWD	0.42 ± 0.02*	0.44 ± 0.04†	0.45 ± 0.06†	0.41 ± 0.02*	$F = 5.556$	<0.001**
MPI TDI	0.47 ± 0.03*	0.51 ± 0.07†	0.54 ± 0.10†	0.47 ± 0.03*	$F = 6.936$	<0.001**
EF Simpson	65.21 ± 4.69*	62.76 ± 6.83*	59.83 ± 9.21†	66.47 ± 4.28*	$F = 4.995$	0.003*
Average S wave	12.58 ± 1.37*	11.81 ± 2.20*	10.25 ± 2.27†	12.85 ± 1.29*	$F = 10.08$	<0.001**
EF M mode	65.59 ± 3.99*	62.79 ± 7.12*	59.60 ± 9.76†	67.02 ± 3.99*	$F = 6.041$	<0.001**
MV E/E′	13.60 ± 4.54*	15.71 ± 1.88†	16.48 ± 1.03†	5.42 ± 1.03‡	$F = 97.85$	<0.001**
MV E/A	0.90 ± 0.29*	0.72 ± 0.10†	0.66 ± 0.08†	1.37 ± 0.12‡	$H = 90.0$	<0.001**
DT (ms)	230.09 ± 27.63*	243.30 ± 11.64†	244.80 ± 10.64†	197.03 ± 12.54‡	$F = 42.11$	<0.001**
IVRT (ms)	103.49 ± 17.60*	117.87 ± 6.91†	121.54 ± 6.14†	76.77 ± 5.92‡	$F = 95.94$	<0.001**

One way analysis of variance test was performed & multiple comparisons between groups through post-hoc test: Tukey's test.
Kruskal–Wallis was performed and multiple comparison between groups through Mann–Whitney test.
A, peak velocity of atrial filling; E, peak velocity of early filling; EF, ejection fraction; IVRT, isovolumic relaxation time; MV, mitral valve; MPI, myocardial perfusion imaging; PWD, posterior wall diameter; TDI, tissue Doppler imaging.
Means that do not share same symbol are significantly different at P value ($P < 0.05$).
*P value less than 0.05 (S).
**P value less than 0.001 (HS).

Table 5. Comparison of patients who were compensated against those who were not, as revealed by echocardiographic findings.

Echocardiographic findings	Compensated ($N = 50$)	Decompensated ($N = 25$)	Test value	P value
MPI PWD	0.42 ± 0.02	0.45 ± 0.05	$t = 4.296$	<0.001**
MPI TDI	0.48 ± 0.03	0.53 ± 0.09	$t = 4.195$	<0.001**
EF by Simpson	65.21 ± 4.72	61.30 ± 8.21	$t = 2.933$	0.004*
Average S wave	12.58 ± 1.38	11.04 ± 2.37	$t = 3.975$	<0.001**
EF by M mode	65.59 ± 3.99	61.20 ± 8.63	$t = 3.456$	<0.001**
MV E/É ratio	13.60 ± 4.54	16.10 ± 1.56	$t = 2.693$	0.008*
MV E/A ratio	0.91 ± 0.29	0.70 ± 0.10	$U = 3.541$	<0.001**
DT (ms)	230.39 ± 27.63	244.06 ± 11.04	$t = 2.404$	0.018*
IVRT (ms)	103.50 ± 17.60	119.72 ± 6.74	$t = 4.487$	<0.001**

t, independent sample *t* test; *U*, Mann–Whitney test.
A, peak velocity of atrial filling; E, peak velocity of early filling; EF, ejection fraction; IVRT, isovolumic relaxation time; MV, mitral valve; MPI, myocardial perfusion imaging; PWD, posterior wall diameter; TDI, tissue Doppler imaging.
P value more than 0.05 (NS).
**P* value less than 0.05 (S).
***P value* less than 0.001 (HS).

TDI, MV E/É ratio, DT (ms), and IVRT (ms), with higher values in the patient group (significant at 0.05). The mean EF by Simpson, Average S wave, EF by M mode, and MV E/A ratio between the case group and the control group could be clearly distinguished from one another ($P = 0.05$).

Therefore, compared with the control group, patients with liver cirrhosis had considerably worse LV systolic and diastolic function. Moreover, Hassan and Elden[9] reported that by conventional echocardiography, even if the EF via M-mode increased significantly ($P = 0.004$) in cirrhotic group versus control group, there was insignificantly increased LV end-diastolic dimensions, LV end-diastolic volume, and EF by 2D. Regarding diastolic function by conventional parameters, there were statistically higher significant values of E, A, and deceleration time in the cirrhotic group versus normal one. There was a significantly lower TDI systolic velocity and LV global longitudinal strain % for assessment of LV systolic function besides LVDD assessed by TDI in the cirrhotic group versus control.

Furthermore, comparison between the study groups according to echocardiographic findings of MPI PWD, average S wave, MPI TDI, EF Simpson, and EF M mode, the current study demonstrated that there were statistically significant differences between groups.

MV E/E′, MV E/A, DT (ms), and IVRT (ms) showed significant difference ($P < 0.05$). This comes in agreement with Ashmawy et al.,[8] who disclosed that the Child score for the severity of liver disease increased as a result of these LV dysfunctions. Because of echocardiographic findings in these results (MPI PWD, MPI TDI, EF Simpson, average S wave, and EF M mode); subgroups varied statistically significantly from one another in MV E/E 0, MV E/A, DT (ms), and IVRT (ms). In addition, Hassan and Elden[9] reported that all three groups had normal LV dimensions according to traditional echocardiographic settings. Both systolic and diastolic dysfunction was evident especially in Child B and C, and by TDI, they diagnosed diastolic dysfunction in 75% of all patient groups, indicating that TDI is more accurate in detecting early LV diastolic dysfunction.

Moreover, the study by Rani et al.[10] reported that echocardiographic alterations were statistically significant ($P < 0.05$) in cirrhotic patients related with Child–Pugh scoring-based disease severity.

Based on the Child–Pugh classification criteria, groups of cirrhotic patients were separated into groups that were compensated (Child–Pugh class A) and those that were not (Child–Pugh classes B and C). According to EF by Simpson's average S wave, the current investigation found that the mean value in the compensated group was statistically significantly greater than that in the decompensated group. EF by M mode and MV E/A ratio showed significant difference between compensated group and decompensated group ($P < 0.05$). MPI PWD, MPI TDI, MV E/É ratio, DT (ms), and IVRT (ms) showed significant decrease in the compensated

Table 6. Left ventricular systolic dysfunction distribution among patients group.

LV systolic dysfunction	*n* (%)
Yes	7 (9.3)
No	68 (90.7)

Table 7. Left ventricular diastolic dysfunction distribution among patients group.

Subgroups	Total	LV diastolic dysfunction [*n* (%)]
Child A	25	8 (32.0)
Child B	25	16 (64.0)
Child C	25	25 (100.0)
Total	75	49 (65.3)

group compared with the decompensated group ($P < 0.05$ significant). The current study showed that there were LV systolic dysfunction was seven (9.3%) patients. Also, we found that there were LVDD was 49 (65.3%) patients, out of them eight (32%) patients in Child A; 16 (64%) patients in Child B and 25 (100%) patients in Child C. This indicates that as liver cirrhosis severity increases the prevalence of were LVDD, means a positive correlation exist.

However, Bhuin and Mohanty[11] reported on the prevalence of cardiac dysfunction in nonalcoholic cirrhosis and showed that 37 (52.9%) patients had systolic dysfunction, whereas 57 (81.4%) patients had diastolic dysfunction. Our findings were supported by Hassan and Elden,[9] who showed a link between LV systolic failure and the severity of liver cirrhosis.

4.1. Conclusion

Standard Doppler echocardiography can detect right and left ventricle diastolic failure, whereas tissue Doppler is more capable of identifying diastolic dysfunction and provides a more accurate assessment of filling dynamics. Compared with the control group, patients with liver cirrhosis exhibited significantly worse left ventricular systolic and diastolic performance, and this difference was more prominent in decompensated patients than in compensated patients.

Ethical approval

The study was approved by the ethical committee of Faculty of Medicine, Al-Azhar University, Cairo. An informed consent was obtained from each patient.

Conflict of interest

There are no conflicts of interest.

References

1. Carvalheiro F, Rodrigues C, Adrego T, et al. Diastolic dysfunction in liver cirrhosis: prognostic predictor in liver transplantation? *Transplant Proc*. 2016;48:128–131.
2. Wiese S, Hove JD, Bendtsen F, Møller S. Cirrhotic cardiomyopathy: pathogenesis and clinical relevance. *Nat Rev Gastroenterol Hepatol*. 2014;11:177–186.
3. Falletta C, Filì D, Nugara C, et al. Diastolic dysfunction diagnosed by tissue Doppler imaging in cirrhotic patients: prevalence and its possible relationship with clinical outcome. *Eur J Intern Med*. 2015;26:830–834.
4. Ruiz-del-Árbol L, Serradilla R. Cirrhotic cardiomyopathy. *World J Gastroenterol*. 2015;21:11502–11521.
5. Wong F, Girgrah N, Graba J, Allidina Y, Liu P, Blendis L. The cardiac response to ex-ercise in cirrhosis. *Gut*. 2001;49:268–275.
6. Bernardi M, Caraceni P. Pathogenesisof ascites and hepatorenal syndrome: alteredhaemodynamics and neurohumoral systems. In: Gerbes A, Beuers U, Jüngst D, Pape G, Sackmann M, Sauerbruch T, eds. *FalkSymposium 117. Hepatology 2000. Symposium inHonour of Gustav Paumgartner, Munich, 6–8 May2000*. Dordrecht: Kluwer Academic Publishers; 2001:185–203.
7. Pozzi M, Carugo S, Boari G, et al. Evidence of functional and structural cardiac abnormalities in cirrhotic patients with and without ascites. *Hepatology*. 1997;26:1131–1137.
8. Ashmawy MM, Younis HA, Elbaset MA, et al. Evaluation of cardiac function in patients with liver cirrhosis using tissue Doppler study. *Egypt J Intern Med*. 2018;30:115–120.
9. Hassan AA, Elden NB. Assessment of left ventricular functions and its correlation to the severity of liver cirrhosis caused by hepatitis C virus by different echocardiography modalities (two-dimensional speckle-tracking echocardiography study). *Al-Azhar Assiut Med J*. 2019;17:154.
10. Rani K, Zaman B, Bai K. Echocardiographic alterations in liver cirrhosis. *Drug Resist TB Bone Marrow Stem Cells*. 2021;32:172–176.
11. Bhuin S, Mohanty R. Evaluation and correlation of cardiovascular dysfunction in nonalcoholic cirrhotic patients with its severity. *J Evol Med Dent Sci*. 2017;6:5200–5204.