e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(6); 315-318

Original Research Article

Analysis of Cardiac Function in Patients with Alcoholic Cirrhosis

Sushil Kumar[1], Pankaj Mohan Shrivastava[2], Gopi Nath Dubey[3], Krishna Kumar Jha[4]

[1]Assistant Professor, Department of Medicine, Darbhanga Medical College and Hospital, Laheriasarai, Bihar

[2]Assistant Professor, Department of Medicine, Darbhanga Medical College and Hospital, Laheriasarai, Bihar

[3]Assistant Professor, Department of Medicine, Darbhanga Medical College and Hospital, Laheriasarai, Bihar

[4]Professor, Department of Medicine, Darbhanga Medical College and Hospital, Laheriasarai, Bihar

Received: 25-03-2024 / Revised: 23-04-2024 / Accepted: 26-05-2024

Corresponding Author: Dr. Gopi Nath Dubey

Conflict of interest: Nil

Abstract:

Background: Liver cirrhosis is a condition that is frequently seen in clinical settings and causes a considerable amount of morbidity and mortality worldwide. This is a slowly progressing, chronic illness that affects the liver in a diffuse manner. It is linked to nodule formation; excessive collagen deposition that causes fibrosis, liver cell degeneration, and abnormal vascular architecture that alters hemodynamics. The aim of the present study is to evaluate cardiac function in liver cirrhosis patients.

Methods: Between April 2023 and March 2024, 74 patients with a diagnosis of liver cirrhosis who were admitted to the Department of Medicine at the DMCH in Laheriasarai, Bihar, participated in a cross-sectional study.

Results: 59 cases (79.73%) in all had an LVDD diagnosis. Of them, Stage 1 LVDD (impaired relaxation) accounted for 47.29% of cases, Stage 2 LVDD (pseudo normal) for 31.08%, and Stage 3 LVDD (severe restrictive type) for just one patient.

Conclusion: While systolic function is preserved until severe hepatic failure, left ventricular diastolic impairment is frequently linked to the progression of hepatic dysfunction.

Keywords: Alcoholic Liver Cirrhosis, Cardiac Dysfunction, Diastolic Dysfunction, Hepatic Failure, Portal Hypertension.

Introduction

For a long time, having both liver cirrhosis and heart failure at the same time has been regarded as thrilling. Though the incidence of liver cirrhosis in cases of severe heart failure was widely reported a long time ago, the development of heart failure as a consequence of liver cirrhosis was not recognized until a few decades ago. With a baseline rise in cardiac output, a state of hyperdynamic circulation reduced peripheral vascular resistance and blunted systolic flow.

When we discuss the likelihood of cardiac dysfunction in patients with liver cirrhosis, we mean diastolic reactivity to physical and pharmacological stress, electrophysiological abnormalities, and subtle histomorphological changes in the setting of liver cirrhosis.[1] The pathophysiology of cirrhotic cardiomyopathy, however, may involve aberrant membrane biophysical characteristics, impaired adrenergic receptor signal transmission, and increased activation of unfavorable inotropic pathways mediated by cyclic guanosine monophosphate (cGMP).[2] Studies have connected cirrhosis to a number of heart issues. This includes an electrical abnormality, malfunction in the systolic and diastolic phases, and reduced cardiac contractility.[3,4] Heart failure during the diastolic and systolic phases point to a poor outcome in advanced cirrhosis. Diastolic dysfunction is associated with a higher risk of death after a TIPS procedure, while systolic dysfunction is connected to an increased chance of developing hepatorenal syndrome [5,6]. Reduced ascites clearance is also linked to diastolic dysfunction. Therefore, at this stage of cirrhosis, more impressive longevity is associated with intact systolic capacity and high cardiac output.

A disease called cirrhosis is identified histopathologically. It can manifest in a range of clinical manifestations and result in various issues,

some of which have the potential to be lethal.[7] Liver disease can result from cirrhosis of the heart, and cirrhosis of the liver can result in a cirrhotic cardiac defect when it spreads to the heart. These two ailments may have an impact on one another.[8] At first, cirrhotic cardiac abnormalities were considered to have little clinical significance; nevertheless, the detrimental effects of cardiac dysfunctions became apparent with the increasing usage of invasive procedures and liver transplants. As a result, opinions about the clinical importance of cirrhosis changed.

Numerous researchers have noticed alterations in the systemic hemodynamics and cardiovascular abnormalities in patients with cirrhosis.[9] An elevation in blood volume, a reduction in the systemic vascular resistance, and an increase in cardiac output are associated with a more advanced stage of liver cirrhosis.[10] Anecdotal observations suggest that coronary artery disease is a more common clinical issue than either alcoholic or cirrhotic cardiac abnormality, a condition in which the heart is damaged as a result of cirrhosis. There is a lack of data regarding the prevalence of various types of heart diseases in patients with liver cirrhosis, particularly in patients with alcoholic cirrhosis.[11]

Material and Methods

From April 2023 to March 2024, a cross-sectional study was carried out in the medicine department of Darbhanga Medical College and Hospital in Laheriasarai, Bihar. This study included all diagnosed cases of alcoholic liver cirrhosis that were admitted to the medicine department. Additionally excluded were patients receiving emergency treatment with TIPS, active variceal bleeding, acute alcohol abuse, alcoholic delirium, or a history suggestive of concomitant heart disease, such as valvular heart diseases or ischemic heart disease, as well as those with electrocardiogram results or echocardiographic changes indicating such conditions.

After gaining their informed written agreement, cases of liver cirrhosis that met the study's inclusion criteria were chosen. With the use of prevalidated, semi-structured case record proforma and these investigations—complete hemogram, liver profile, renal profile, ECG, and ECHO—a comprehensive medical history, the existence of additional co-morbid disorders, and the results of the general and systemic examination are recorded.

The severity of the cirrhosis was classified according to the Child-Pugh scale.

The collected data was coded and entered with the help of Microsoft Excel software. The data will be analyzed with the help of SPSS Version 22 statistical package.

Results

This investigation, which had 74 participants, focused on liver cirrhosis caused by alcohol. After excluding certain cases, each case was carefully chosen. The age group of 46–55 years old accounted for the majority of instances (47.29%), followed by 56–65 years old (22.97%). The study subjects' average age was 48.7 years. (Table 1)

Table 1: Age distribution of study subjects

Age group	Number of cases	Percentage
<35 years	3	4.05%
36-45 years	11	14.86%
46-55 years	35	47.29%
56-65 years	27	22.47%
>66 years	8	10.81%
Total	74	100%

The liver profile parameters of the study participants were evaluated in this investigation. The average serum albumin level was 2.93± 0.27 (2.5 – 3.8), the average serum bilirubin level was 2.5 ±1.23 (0.81-3.83), the average INR was 1.63± 0.3 (1.1 – 2.8), the average serum AST level was 65.7± 29.3 (19–110), and the average serum ALT level was 61± 27.15 (21–102). (Table 2)

Table 2: Distribution of liver profile parameters

Biochemical parameters	Mean value	Range
Serum albumin	2.93± 0.27	2.5 – 3.8
Serum bilirubin	2.5 ±1.23	0.81-3.83
INR	1.63± 0.3	1.1 – 2.8
Serum AST	65.7± 29.3	19-110
Serum ALT	61± 27.15	21-102

The three groups' heart rates mean arterial pressures, ejection fractions, and the sizes of each individual cardiac chamber were compared. The mean MAP level was 84.5± 2.97 (82-92), the mean heart

rate was 88.5± 4.85 (78-94), and the mean EF% was 72.3± 1.96 (65-74). A combination of the left ventricular diastolic dysfunction and the E/A ratio, eC value, E/eC ratio, and DT was used to evaluate it. Overall, 59 cases (79.73%) had an LVDD diagnosis. Of these, 47.29% of cases had Stage 1 LVDD (impaired relaxation), 31.08% had Stage 2 LVDD (pseudo normal), and just one patient had Stage 3 LVDD (severe restrictive type). (Table 3,4 and 5)

Table 3: Distribution of study subjects according to Child-Pugh scoring

CPT score	Number of cases	Percent
A (5-6)	18	24.32%
B (7-9)	53	71.62%
C (10-15)	3	4.05%
Total	74	100%

Table 4: Distribution of cardiac parameters

Cardiac parameters	Mean value	Range
Heart rate	88.5± 4.85	78-94
MAP	84.5± 2.97	82-92
EF%	72.3± 1.96	65-74

Table 5: Distribution of study subjects according to stages of Left ventricular dysfunction

Left ventricular dysfunction	Number of cases	Percentage
Stage 0	15	20.27%
Stage 1	35	47.29%
Stage 2	23	31.08%
Stage 3	1	1.35%
Total	74	100%

Discussion

We evaluated the individuals with cirrhosis due to alcohol for both morphological and functional heart impairment in the current investigation. The current study involved 74 cases of alcoholic liver cirrhosis, with a mean age of 48.7 years among the study participants.

The liver profile parameters of the study participants were evaluated in the current investigation. The average serum albumin level was 2.93± 0.27 (2.5 – 3.8), the average serum bilirubin level was 2.5 ±1.23 (0.81-3.83), the average INR was 1.63± 0.3 (1.1 – 2.8), the average serum AST level was 65.7± 29.3 (19–110), and the average serum ALT level was 61± 27.15 (21–102) (Table 2).

The cardinal cardiac parameters such as heart rate, mean arterial pressure, ejection fraction and the individual cardiac chamber size were compared between the three groups. We observed that the mean heart rate was 88.5± 4.85 (78-94), mean level of MAP was 84.5± 2.97 (82-92) and the mean EF% was 72.3± 1.96 (65-74).

The left ventricular diastolic dysfunction was assessed using the LAV, E/A ratio, e′ value, E/e′ ratio and DT. Cardiac dimension is enlarged in all the four chambers with increase in ejection fraction in cirrhotic patients with ascites.

Overall LVDD was diagnosed in 59 cases (79.73%). Out of which, 47.29% cases presented with Stage 1 (impaired relaxation) LVDD, Stage 2 LVDD (pseudo normal) among 31.08% and only one patient had severe restrictive type of (Stage 3) LVDD. While the left ventricular systolic function was preserved in all the studied patients. Patients with cirrhosis seem to have a higher prevalence of diastolic dysfunction; in fact, some experts argue that almost all cirrhosis patients have some degree of diastolic dysfunction. The majority of recent studies that used 2-D Doppler echocardiography to diagnose LVDD relied on an E/A ratio of less than 1. Similar lower mean E/A ratios were observed in the left and right ventricles of the ascitic subgroup compared to the non-ascitic subgroup by Valeriano et al. [12]. Rapid total paracentesis removal of ascitic fluid decreased A wave velocity and elevated E/A ratio to values equivalent to cirrhotic patients without ascites, but still aberrant when compared to healthy controls, as demonstrated by Pozzi et al. [13].

However, due to its significant reliance on preload and frequent need for age adjustment, the E/A ratio has a number of drawbacks. As the left ventricle expands during the diastole, the velocity of myocardial displacement is directly measured by TDI, which is independent of left atrial pressure and volume status, in contrast to transmitral valve Doppler flow. The definition of LVDD now includes TDI characteristics according to the ASE. According to a recent study by Ruiz del Arbol et al. [14], 37/80 (46.2%) of the cirrhotic patients with TDI had LVDD. They also discovered that LVDD is linked to a decrease in effective arterial blood vol-

ume and occurs concurrently with other modifications to heart structure and function. LVDD was a sensitive indicator of death, the onset of type 1 hepatorenal syndrome, and severe cirrhosis. Our study shows left ventricular diastolic dysfunction is present in most of the cirrhotic patients which was detected by TDI in 70% of cases. This rate is somewhat more than the 50- 60% found in recent study conducted by both TDI and Doppler echocardiography.[14,15] In our study, eighty percent of patients with cirrhosis had LVDD (Type I and II). The left ventricle's systolic performance parameters were within the usual range. To determine the cutoff point for tapering or stopping beta blockers and the prognostic significance of left ventricular diastolic dysfunction in cirrhosis patients, more research is necessary.

Conclusion

While systolic function is preserved until severe hepatic failure, left ventricular diastolic impairment is frequently linked to the progression of hepatic dysfunction. Pulsed TDI provides a reliable assessment of peak early diastolic wave velocity, deceleration time, and E/e′ ratio for diastolic dysfunction.

References

1. Milani A, Zaccaria R, Bombardieri G, Gasbarrini A, Pola P. Cirrhotic cardiomyopathy. Dig Liver Dis 2007; 39:507-15.
2. Baik SK, Fouad TR, Lee SS. Cirrhotic cardiomyopathy. Orphanet J Rare Dis 2007; 2:15.
3. Krag A, Bendtsen F, Mortensen C, Henriksen JH, Møller S. Effects of a single terlipressin administration on cardiac function and perfusion in cirrhosis. Eur J Gastroenterol Hepatol 2010; 22:1085-92.
4. Bernardi M, Maggioli C, Dibra V, Zaccherini G. QT interval prolongation in liver cirrhosis: Innocent bystander or serious threat? Expert Rev Gastroenterol Hepatol 2012; 6:57-66.
5. Cazzaniga M, Salerno F, Pagnozzi G, Dionigi E, Visentin S, Cirello I et al. Diastolic dysfunction is associated with poor survival in patients with cirrhosis with transjugular intrahepatic portosystemic shunt. Gut 2007; 56:869-75.
6. Krag A, Bendtsen F, Henriksen JH, Møller S. Low cardiac output predicts development of hepatorenal syndrome and survival in patients with cirrhosis and ascites. Gut 2010; 59:105-10.
7. Bacon RB. Cirrhosis and its complications. In: Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo J, editors. Harrison's Principals of Internal Medicine. 19th ed., Vol. 2. New York: McGraw Hill; 2015. p. 2058.
8. Dhangar V, Nayak K, Khaini P, Srivastav V. Echo study in patients with cirrhosis of liver. Natl J Med Res 2014; 4:241-3.
9. Patil S, Lal B, Pandey M, Haldia SS, Rishi JP. A clinical study of cardiovascular dysfunction in patients of cirrhosis of liver. Ann Int Med Den Res 2016; 2:212-5.
10. Venkateshwarlu N, Gandiah P, Indira G, Sivarajappa P, Prabhakar KK. Cardiac abnormalities in patients with cirrhosis. Indian J Life Sci 2013; 3:105.
11. Gupta R, Singhal A, Jatav O. Echocardiographic changes in alcoholic Liver disease. Int J Multidisc Res Dev 2015; 2:72-9.
12. Valeriano V, Funaro S, Lionetti R, Riggio O, Pulcinelli G, Fiore P. Modification of cardiac function in cirrhotic patients with and without ascites. Am J Gastroenterol 2000;95:3200-3205
13. Pozzi M, Carugo S, Boari G, et al. Evidence of functional and structural cardiac abnormalities in cirrhosis patients with or without ascites. Hepatology 1997; 26:1131-1137
14. Arbol LR, Achecar L, Serradilla R, et al. Diastolic dysfunction is a predicter of poor outcomes in patients with cirrhosis, portal hypertension and a normal creatinine. Hepatology 2013; 58:1732-1741.
15. Papastergiou V, Skorda L, Lisgos P, et al. Ultrasonographic prevalence and factors predicting left ventricular diastolic dysfunction in patients with liver cirrhosis: Is there a correlation between the grade of diastolic dysfunction and the grade of liver disease? Scientific World Journal 2012; 2012:615057.