

Study of Cardiac Function in Alcoholic Cirrhotic Patients

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ABSTRACT

Introduction: Cardiac dysfunction in patients with cirrhosis occurs in the setting of a circulatory dysfunction characterized by a marked splanchnic arterial vasodilation. Circulatory changes can lead to the cardiac dilatation of the left chambers and the development of functional changes in the heart. The present study is intended to assess cardiac functions in patients of liver cirrhosis.

Material and methods: It was a cross sectional study conducted among 74 diagnosed cases of liver cirrhosis, admitted to department of general medicine, KIMS Hospital during the 6 months study duration.

Results: 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.

Conclusion: Left ventricular diastolic dysfunction is commonly associated with advancement of hepatic dysfunction while systolic function is maintained till advanced hepatic failure.

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

INTRODUCTION

Cirrhosis is a fatal condition. Although mild cirrhosis can be associated with prolonged survival, most diseases that induce cirrhosis progress, at variable rates, to end-stage liver failure. Deaths from hepatic failure, variceal bleeding and infection are common in advanced cirrhosis, and even the rate of sudden unexplained death is increased compared with that in a normal population. Moreover, patients with cirrhosis are well known to be fragile, and do poorly after invasive or stressful procedures.¹

Patients with end-stage liver disease manifest a hyperdynamic circulation characterized by a decrease in the systemic vascular resistance and arterial pressure, and an increase in the heart rate and cardiac output. The clinical manifestations of hyperdynamic circulations include warm skin, spider angioma, palmer erythema, and bounding pulse. These cardiovascular changes were described over 50 years ago by Kowalski and Abelmann in a group of alcoholic cirrhotic patients.² These findings were then confirmed in multiple experimental models of portal hypertension and in patients with cirrhosis. Initially it was thought that these changes were a manifestation of latent alcoholic cardiomyopathy, however future studies confirmed the same circulatory dysregulation in cirrhotic patients with different underlying diseases.^{3,4,5}

Different pathophysiological mechanisms including neurogenic, humoral, and vascular dysregulations are implicated in the pathogenesis of these cardiovascular

changes. The hyperdynamic circulation is most likely initiated by splanchnic and peripheral vasodilatation, leading to reduction in the effective arterial blood volume. This leads to a diminished renal blood flow in cirrhotic patients, which in turn stimulates the rennin-angiotensin-aldosterone system (RAAS), sympathetic nervous system, and antidiuretic hormone resulting in renal artery vasoconstriction, sodium retention, and volume expansion. Worsening liver disease results in progressive vasodilatation, making the hyperdynamic circulation and renal artery vasoconstriction more pronounced.⁶⁻⁸

Cardiac dysfunction in patients with cirrhosis occurs in the setting of a circulatory dysfunction characterized by a marked splanchnic arterial vasodilation. At the initial stages of cirrhosis, the circulatory dysfunction is compensated by the development of a hyperdynamic circulation. These circulatory changes can lead to the cardiac dilatation of the left chambers and the development of functional changes in the heart.⁹

The present study was intended to assess cardiac functions in patients of liver cirrhosis.

MATERIAL AND METHODS

A descriptive Cross-Sectional Study was conducted for 6 months in Department of Medicine, KIMS, Karad.

Study participants

Diagnosed cases of alcoholic liver cirrhosis, admitted to department of general medicine were enrolled in this study

Sample size

Considering prevalence of cardiac dysfunctions among liver cirrhosis patients as 70% as per reference study conducted by Sunil Dadhich et al. At 95% confidence interval, 85% power, 5% level of significance, The sample size can be calculated as follows:

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$$N = \frac{(1.96 \times 1.96) p \times q}{d^2} = \frac{(1.96 \times 1.96 \times 0.7 \times 0.3)}{(0.105 \times 0.105)} = \frac{0.798}{0.011} = 74$$

Hence, 74 diagnosed cases of liver cirrhosis, admitted to department of general medicine, KIMS Hospital during the period of 6 months were selected consecutively, after the approval of institutional ethical committee.

Outcome indicators

- Liver profile
- ECG findings
- 2D ECHO findings
- Renal profile
- Ejection fraction

Inclusion criteria: All the diagnosed cases of liver cirrhosis, admitted to department of general medicine, KIMS Hospital during the study duration were enrolled in this study.

Exclusion criteria: Patients with nonalcoholic cirrhosis, active variceal bleeding, acute alcohol abuse, alcoholic delirium, or undergoing emergency treatment with TIPS were excluded.

Patients with a history suggestive of, and/or electrocardiogram results or echocardiographic changes indicating, concomitant heart disease—for example, valvular heart diseases, ischaemic heart disease were also be excluded.

Study procedure: Cases of liver cirrhosis fulfilling the inclusion criteria were selected in the present study after obtaining their informed written consent. Detailed medical history, presence of other co-morbid conditions, general and systemic examination findings was recorded with the help of pre-validated, semi-structured case record proforma.

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

Investigations

- Complete hemogram
- Liver profile
- Renal profile
- ECG
- ECHO

STATISTICAL ANALYSIS

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. Descriptive statistics will be derived in the form of tables and charts for frequency analysis.

RESULTS

The present study was conducted in 74 subjects and consisted of alcoholic liver cirrhosis. All the cases were carefully selected after ruling out exclusion criteria. Majority of the cases belonged to age group of 46-55 years (47.29%), followed by 56-65 years (22.97%). The mean age of the study subjects was 48.7 years. (Table 1)

In the present study we assessed liver profile parameters of the study subjects, the mean serum albumin level was 2.93± 0.27 (2.5 – 3.8), mean Serum bilirubin level was 2.5 ±1.23

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%

Table-1: Age distribution of study subjects

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

Table-2: Distribution of liver profile parameters

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

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-4: Distribution of cardiac parameters

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

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

(0.81-3.83), Mean INR was 1.63± 0.3 (1.1 – 2.8), mean Serum AST level was 65.7± 29.3 (19-110) and the mean Serum ALT level was 61± 27.15 (21-102) (Table 2).

Morphological parameters 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. 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.

DISCUSSION

In the present study, we assessed the alcoholic cirrhotic patients for morphological and functional cardiac dysfunction. The present study was conducted among 74 cases of alcoholic liver cirrhosis, admitted in KIMS, Karad, for the period of 6 months after the approval of institutional ethical committee. The mean age of the study subjects was 48.7 years.

In the present study we assessed liver profile parameters of the study subjects, the mean serum albumin level was 2.93 ± 0.27 (2.5 – 3.8), mean Serum bilirubin level was 2.5 ± 1.23 (0.81-3.83), Mean INR was 1.63 ± 0.3 (1.1 – 2.8), mean Serum AST level was 65.7 ± 29.3 (19-110) and the mean Serum ALT level was 61 ± 27.15 (21-102) (Table 2).

Morphological parameters 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.

Diastolic dysfunction appears to be more prevalent in cirrhotic patients, indeed some authorities contend that some degree of diastolic dysfunction is present in virtually every patients with cirrhosis. In most of the studies performed in the recent past, diagnosis of LVDD was based on E/A ratio <1 using 2-D Doppler echocardiography. Valeriano et al¹⁰ also found a similar lower mean E/A ratio in both left and right ventricle in ascitic subgroup than in non-ascitic subgroup. Pozzi et al¹¹ showed that removal of ascitic fluid by rapid total paracentesis reduced the A wave velocity and increased the E/A ratio to the values similar to those of cirrhotic patients without ascites, but still abnormal as compared to healthy controls.

However, E/A ratio have several limitations as it is strongly dependent on preload and often requires age correction. Unlike transmitral valve Doppler flow, TDI directly measures the velocity of myocardial displacement as the LV expands in the diastole and therefore is independent of volume status and left atrial pressure. The ASE has included TDI parameters in the definition of LVDD. A recent study by Ruiz del Arbol et al showed LVDD in 37/80 (46.2%) with TDI in cirrhotic patients. They also found LVDD occurs simultaneously with other changes in cardiac structure and function and is associated with an impairment of effective arterial blood volume. LVDD was a sensitive marker of advanced cirrhosis, type 1 hepatorenal syndrome development, and mortality.

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.^{12,13}

In our study LVDD (Type I and II) was seen in eighty percent of cirrhotic patients. Parameters regarding left ventricular systolic performance were within normal range. Further studies are required to assess the prognostic impact of left ventricular diastolic dysfunction in patients with cirrhosis and the cut off parameter to taper or abandon beta blocker.

CONCLUSION

Left ventricular diastolic dysfunction is commonly associated with advancement of hepatic dysfunction while systolic function is maintained till advanced hepatic failure. Peak early diastolic wave velocity, deceleration time and E/e' ratio for diastolic dysfunction are accurately assessed by pulsed TDI.

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