

Cardiac changes in patients with chronic liver disease: A prospective descriptive study

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Abstract

Background: The systemic circulation in patients with cirrhosis is hyperdynamic with an increased cardiac output and heart rate and a reduced systemic vascular resistance as the most pronounced alterations. The concomitant cardiac dysfunction has recently been termed “cirrhotic cardiomyopathy”, which is an entity different from that seen in alcoholic heart muscle disease. **Objective:** To study cardiac changes in patients with chronic liver disease. **Methodology:** The present study was a prospective descriptive clinical study consisting of thirty five patients with cirrhosis who were inpatients in the department of Medicine, tertiary care Institute, from Oct 2011 to Oct 2013. The patients were evaluated for presence of cirrhotic cardiomyopathy. **Results:** In A group there is no patient, while in Child- Pugh Score- B group 14 subjects have cirrhotic cardiomyopathy out of 16 and in C group 18 have cirrhotic cardiomyopathy out of 19. We analyzed that there was no relation between severity of liver disease and cirrhotic cardiomyopathy (p value- 0.446). **Conclusion:** Indian patients with cirrhosis do have diastolic dysfunction. In the absence of other risk factors for cardiac disease, this dysfunction can be attributed only to cirrhotic cardiomyopathy.

Keywords: Cardiomyopathy, Cirrhosis, chronic liver disease

Introduction

The clinical picture of patients with cirrhosis is dominated by the classical complications such as ascites, bleeding from oesophageal varices, portal hypertension and encephalopathy. In addition, a considerable number of patients show signs of peripheral vasodilatation with palmar erythema and reddish skin, raised and bounding pulse, and a low systemic blood pressure indicating a hyperdynamic circulation. The hyperdynamic syndrome comprises an increased heart rate, cardiac output, and plasma volume, and a reduced systemic vascular resistance and arterial blood pressure. Increased cardiac output in cirrhosis was described more than 50 years ago and a hyperdynamic, hyporeactive circulation is today a well characterized element in the clinical appearance of these patients. In addition, patients with cirrhosis develop complications from a variety of organs including the heart, lungs, and kidneys, and other organ systems.

Besides the hepatorenal syndrome, this has led to the recognition of new clinical entities, such as cirrhotic cardiomyopathy and the hepatopulmonary syndrome. Cirrhotic cardiomyopathy was initially thought to be of little clinical relevance. However, with the frequent use of invasive procedures like surgical portocaval shunts, transjugular intrahepatic portosystemic shunt and liver transplantation, the adverse consequences of cardiac dysfunction became evident. These procedures put additional stress on the dysfunctional heart, precipitating overt cardiac failure. Unexpected deaths due to heart failure following these procedures became a cause of concern in centers where these procedures were regularly performed.

Past decades have seen the appearance of new techniques for the study of different aspects of cardiac function. Our knowledge of cardiovascular pathophysiology has improved considerably, including our understanding of cardiovascular complications of liver disease. Kowalski *et al* [1] were the first to report

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that patients with cirrhosis had abnormal cardiovascular function and a prolonged QT interval. The systemic circulation in patients with decompensated cirrhosis is hyperdynamic and characterised by increased heart rate and cardiac output (CO) and decreased systemic vascular resistance with low normal or decreased arterial blood pressure [2–4]. Among the factors that may increase the CO in patients with cirrhosis are increased sympathetic nervous activity, increased blood volume (increased preload), and the presence of arteriovenous communications [5–7].

Many of the patients present with dyspnoea, fluid retention, and limited exercise capacity [4,8,9]. There is paucity of data from the Indian subcontinent on cirrhotic cardiomyopathy. Apart from a study by Jacob Alexander et al, there is scanty information on the status of cardiac abnormalities in Indian patients with cirrhosis.

Just as the etiological spectrum of cirrhosis varies in different parts of the world, data from one population may not be valid in a different population.

We undertook to study cardiac status in patients with cirrhosis of liver at rest and stress to assess the occurrence of cirrhotic cardiomyopathy, to study if echocardiographic parameters of cardiac dysfunction correlate with the severity of liver dysfunction, and to appraise whether or not there are significant differences in these parameters between alcoholic and non-alcoholic cirrhosis.

Material Methods

Present prospective descriptive clinical study was approved by the institutional ethics committee and consent was obtained from each patient. Thirty five consecutive patients with cirrhosis of liver presenting to the Department of Medicine, tertiary care Institute, Bhopal from October 2011 to October 2013 were included in the study. Diagnosis of cirrhosis of the liver was made on clinical, biochemical, serological, and ultrasound imaging.

All patients were ambulatory and hemodynamically stable.

Inclusion criteria were: Diagnosed cases of chronic liver disease irrespective of age and sex. Exclusion criteria were K/C of CAD, hypertension, RHD, NIDDM,

cardiomyopathy. Known cases of hyperthyroidism, hypothyroidism.

Detail history was taken using Pre-designed and pretested questionnaire. Investigation included complete blood count, Liver and Renal function, Blood sugar, Urine- R/M, HBsAg, Lipid profile, Chest X- ray, USG, ECG and Echocardiography. Echocardiography was performed at rest and also after stress. From the 2D echocardiography ejection fraction was calculated by using Modified Simpson rule.

All valves of heart were seen in detail, Regional wall motion abnormalities was look in every patient. The M mode echocardiogram was used for the measurement of end diastolic and systolic dimensions. All the four chambers were look individually for chamber enlargement. For the stress echocardiography we performed 6MWT (6 minute walk test). In this test we advised to walk around 100ft. On flat hard surface. This test is easy to perform and well tolerated by the patients. Two-dimensional and M mode echocardiographic studies were performed by an experienced cardiologist using a commercially available cardiac ultrasound machine (GE- Vivid3). Echocardiographic images were obtained from the parasternal and apical windows with the patient reclining on the left side, according to the recommendations of American Echocardiography Committee. In Doppler echocardiography accompanied by electrocardiogram, peak early filling velocity (E wave), peak atrial systolic velocity (A wave), early and late mitral diastolic flow ratio (E/A), ratio of E and A velocity time integrals was measured. With M mode measurements, Isovolumetric relaxation time (IVRT) was measured.

Statistical Methods

The Statistical software namely SPSS 15.0, Stata 8.0, MedCalc 9.0.1 and Systat 11.0 were used for the analysis of the data and Microsoft word and Excel were used to generate graphs, tables. A p value of <0.05 was considered as significant. Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients, Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups Inter group analysis) and Student t test (two tailed, dependent) was used to find the significance of study parameters on continuous scale within each group.

Results

In the study thirty five patients were included, age group ranging from 30 to 70 yrs, which included 31 males and 4 females constituting 89% and 11%, Ratio of male /female was 7.75/1. Maximum age of the patient was 70 yrs and minimum was 30 yrs. Maximum patients come under the 50-59yrs age group (40%). The most common presentation was decrease appetite (88.5%), abdominal distension (88.5%), hematemesis and malena in 54.2%. There were no patients with advanced features of cirrhosis. Among the 35 patients common clinical signs were ascitis (94.2%), splenomegaly (80%) edema (68.5%), icterus (48.5%) and pallor (42.8%). Ascitis was minimal to gross was included in our study. Among the patients in the study majority of the patients had alcohol has etiology (51%). Non alcoholic etiologies were Hepatitis B (43%) and Hepatitis C (6%). None of the patients in the study had other etiologies like autoimmune, biliary causes. There were no patients of multiple etiologies. Severity of cirrhosis was calculated based on Child- Pugh criteria, Majority of the subjects were in severity group of Child B (54%) or Child C (46.0%). In our study we compare Child-Pugh Score with Cirrhotic cardiomyopathy. In A group there is no patient, while in B group 14 subjects have cirrhotic cardiomyopathy out of 16 and in C group 18 have cirrhotic cardiomyopathy out of 19. We analyzed that there was no relation between severity of liver disease and cirrhotic cardiomyopathy (p value- 0.446). **Table no. 1** In our study we compare the occurrence of LVH in cirrhotic cardiomyopathy patients. We found that LVH is present in 2 subjects (5.7%) and absent in 33 subjects (94.2%). P value was also insignificant i.e. 0.65. Ejection fraction was increase in 23 patients (65.2%) and decrease in 12 patients (34.2%) after stress. None of the patient had same ejection fraction after stress.

Table No 1: Comparison between presences of LVH in Cirrhotic cardiomyopathy

LVH	Cirrhotic cardiomyopathy		Total
	Present	Absent	
Yes	2	0	2
NO	30	3	33
Total	32	3	35

Discussion

The present study was a prospective descriptive clinical study consisting of thirty five patients with cirrhosis who were inpatients in the department of Medicine, tertiary care Institute, from Oct 2011 to Oct 2013. All patients were ambulant patients thus eliminating the effect of cardiac deconditioning due to bed rest.

The patients were evaluated for presence of cirrhotic cardiomyopathy. Cirrhotic cardiomyopathy is defined as chronic cardiac dysfunction in patients with cirrhosis, characterized by blunted contractile responsiveness to stress and/or altered diastolic relaxation with electrophysiological abnormalities in the absence of known cardiac disease. In our study, male patients outnumbered females (89% vs. 11%). This is due to the distribution of cirrhosis in between the genders as well as life style difference. The mean age of the patients in our study was around fifty four years and subjects in the age group 30-70 years were included. Majority presented with history of decrease appetite (88.5%), abdominal distension (88.5%) as chief complaint. From our 35 patients, 31 (approximately 88.5%) presented

with decrease appetite and 31 (88.5%) had presented with abdominal distension. Clinically ascitis was found in 33 of our patients (94.2%) and Edema was detected in 24 (68.5%), due to activated RAAS, hypoproteinemia and ascitis compressing the abdominal inferior venacava. Icterus was detected in 17 (48.5%) of the study population, this was due to the inclusive criteria used in the study. Cirrhotic patients of our study had liver dysfunction of intermediate & late severity (Sixteen patients (46%) were in Child Pugh class B, nineteen Patients (54%) were in severity class C. Detection of child class B were due to aggressive investigation and symptoms suggestive of liver dysfunction. Child C patients presented with features of gross ascitis or recent GI bleeding. Only ambulated patients were enrolled to eliminate the effect of cardiac decondition due to rest. In our study there was no significant association between the severity of hepatic dysfunction and cardiac changes was seen which is consistent with one published report from India [10]. But there are reports that cardiac changes parallel the severity of hepatic dysfunction.

In our study we found that left ventricular hypertrophy is present in 2 subjects (5.7%) and absent in 33 subjects (94.2%). So we can say there is no relationship between presences of LVH in cirrhotic cardiomyopathy and similar reports also published from India [10].

In most studies of patients with cirrhosis, the heart mass has been found to be within the normal range [11,12]. However, some have reported an increased left ventricular mass [13,14] and in a recent experimental study of portal hypertensive rats, left eccentric hypertrophy was found to correlate directly with the degree of hyperdynamic circulation [15].

The determination of heart volumes in patients with cirrhosis has given somewhat different results depending on the methods used. [16–18] In echocardiographic studies, Kelbæk *et al* [19] and Rector *et al* [17] found the size of the left ventricle to be normal and that of the left atrium enlarged [20,21]. Others, however, have reported increases in both the end diastolic and the end systolic volumes of the left ventricle [13,22–25]. Wong *et al* [16] used radionuclide angiography and they reported normal left ventricular systolic and diastolic volumes, including the stroke volume. Normal and increased right ventricular and atrial volumes have been found by echocardiography [17,20,21,26,27].

In contrast, magnetic resonance imaging has shown reduced right ventricular and atrial dimensions and slightly increased left ventricular and atrial volumes [12]. Reduced right heart volumes could reflect a general contraction of the central blood compartment in patients with cirrhosis, as previously suggested by other techniques [28].

The change in the left ventricular dimensions in cirrhosis is related to haemodynamic dysfunction. Thus, Lewis *et al* [28] found significant correlations between the left ventricular end diastolic diameters on one hand and CO, stroke volume, mean arterial blood pressure, and blood volume on the other.

Moreover, significant direct correlations between plasma atrial natriuretic peptide and left atrial volume and left ventricular end diastolic diameter have been reported [20,21].

Future pathophysiological and clinical research is needed to assess the prognostic implications of cirrhotic cardiomyopathy.

Conclusion

This study demonstrates that Indian patients with cirrhosis do have diastolic dysfunction. In the absence of other risk factors for cardiac disease, this dysfunction can be attributed only to cirrhotic cardiomyopathy. No correlation of cardiac status with severity of liver dysfunction was established. There were no significant differences in cardiac structural and functional parameters between alcoholic and non alcoholic cirrhosis. Echocardiography plays a significant role in detecting early cardiac changes in cirrhosis however these changes do not seem to be predictor of increased mortality in patients of cirrhosis.

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