

## Assessment of Cardiac Functions in Cirrhosis of Liver

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**Abstract:** Objective to Assess the Cardiac Functions in patients with Cirrhosis of Liver and Study the Prevalence of Cirrhotic Cardiomyopathy in these patients

**Methods:** A cross sectional observational study was undertaken where 32 patients with cirrhosis of liver were enrolled after excluding any known cardiac illness and known causes of cardiomyopathy. Patients were subjected to biochemical and sonographic evaluation to confirm Cirrhosis of liver. Cardiac assessment was performed non-invasively using electrocardiogram, transthoracic echocardiography. Results were interpreted using Chi-square/ Fisher Exact test. A  $P < 0.05$  was considered to be significant.

**Results:** Among the 32 patients very high prevalence of diastolic dysfunction was found among the study group. A significant correlation was found between Diastolic dysfunction and ascites ( $P < 0.038$ ). Alcohol consumption was not statistically associated with the cardiac findings. Cardiac abnormalities did not correlate with the severity of liver dysfunction.

**Conclusion:** Indian patients with cirrhosis of liver have a high prevalence of diastolic dysfunction. In the absence of any known cardiac causes it should be attributed to cirrhosis itself. Echocardiography can be routinely done in cirrhotic patients to detect cardiac abnormalities. However no correlation could be found between severity of liver dysfunction and cardiac changes.

**Key words:** Assessment of cardiac function in cirrhosis of liver, cirrhotic cardiomyopathy

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### I. Introduction

Cirrhosis of liver is a commonly encountered disease in clinical practice and is responsible for significant morbidity and mortality all over the world. This disease is progressive and chronic in nature, diffusely involves the liver and is associated with degeneration of liver cells, excess collagen deposition resulting in fibrosis, formation of nodules, and distortion of normal vascular architecture resulting in hemodynamic alterations.

The leading etiological factors in India are Hepatitis B and C; followed by alcohol and NALD.<sup>1</sup> Other causes include autoimmune hepatic diseases, Hepatic venous outflow tract obstruction, toxins, drugs, cardiac failure, metabolic abnormalities and genetic abnormalities. There is a significant group where etiology of cirrhosis of liver cannot be detected and this group is called "Cryptogenic Cirrhosis".

Complications of Cirrhosis of liver include hepatic encephalopathy, acute liver failure, ascites, portal hypertension, spontaneous bacterial peritonitis, bleeding varices, and hepatocellular carcinoma and hepatorenal syndrome.<sup>2</sup>

The systemic hemodynamic changes in cirrhosis of liver have been known for a very long time, but it was in 1950s that patients with alcoholic cirrhosis were found to have cardiovascular abnormalities which included hyperdynamic circulation, decreased peripheral resistance, low arterial blood pressure and increased cardiac output.<sup>3</sup> Studies done later revealed vascular hypo responsiveness to vasoconstrictors in alcoholic cirrhotic patients and these changes were attributed to the effects of alcohol on the heart and hence termed as alcoholic cardiomyopathy.<sup>4</sup> In 1989, Lee reported the reduced cardiac response was due to cirrhosis per se, rather than alcohol.<sup>5</sup> These findings led to the concept that cirrhosis itself triggers cardiac dysfunction and was termed as Cirrhotic Cardiomyopathy.<sup>6</sup>

It has been now recognized that the cardiovascular abnormalities result in the pathogenesis of several complications of liver diseases which contribute to significant morbidity and mortality in patients with cirrhosis. Therefore this study was done to evaluate the cardiovascular abnormalities in patients with cirrhosis of liver based on clinical signs and symptoms, relevant investigations, electrocardiography, roentgenography and echocardiography. The main objective of this study is to assess the Cardiac Functions in Cirrhosis of Liver and Study the Prevalence of Cirrhotic Cardiomyopathy in patients with Cirrhosis of Liver.

## II. Patients And Methods

This study was conducted from February 2012 - July 2013 in the Department of Medicine, Mahatma Gandhi Medical College and Research Institute. This study was started after getting clearance from the Institutional Human Ethics Committee ,MGMC&RI, and Pondicherry.

### Criteria For Selection Of Patient

1. Established cases of cirrhosis of liver.
2. Patients diagnosed with cirrhosis of liver by means of physical, biochemical and monographic evidence... Patients with prior history of cardiac diseases, any known causes of cardiomyopathy were excluded from the study.

### Study Design

A total of 32 patients were recruited for this study. A detailed history was elicited from the patients and a thorough physical examination was done.

Based on their clinical signs and symptoms patients underwent relevant investigations like complete hemogram, renal function tests, liver function tests, coagulation profile, blood sugar estimation, After that they were subjected to an ultrasound Abdomen to look for liver size, texture, presence of collaterals, dilatation of portal vein, presence of ascitis and splenomegaly.

Upper Gastrointestinal Endoscopy was done to look for the presence of any varices. Once a diagnosis of cirrhosis of liver was established they underwent further tests to determine their cardiac status.

Electrocardiogram was done and a special note was made for the presence of QT prolongation, low voltage complexes. It was followed up by a chest x-ray where presence of cardiomegaly was looked for. Echocardiography was done to assess the cardiac functions.

### Statistical Methods

Results on continuous measurements are presented on Mean  $\pm$  SD (Min-Max) and results on categorical measurements are presented in Number (%). Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups.

### Statistical software:

The Statistical software SPSS 15.0 was used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc.

## III. Results

This study was conducted over 18 months in Mahatma Gandhi Medical College & Research Institute, Pondicherry with the principle aim of assessing the cardiac functions in cirrhosis of liver.

Baseline data were collected from all patients. Age, gender, h/o alcohol consumption, symptoms, investigations like complete blood count, renal function test, liver function tests, coagulation profile, ultrasound abdomen, upper gastrointestinal endoscopy, chest x-ray, electrocardiography, echocardiogram were done.

The mean age for the study population was Mean  $\pm$ SD: 51.19  $\pm$  9.46. Of the 32 subjects in the study population 30 were males and 2 females. 75% of the study population was alcoholic whereas 25% were non-alcoholics

22 patients (68.75%) presented with abdominal distension whereas 59.4 % ( 19 patients) presented with jaundice. 25% (8 patients) presented with breathlessness and 25 % ( 8 patients) presented with chest pain

Mean pulse rate was 83.56  $\pm$  7.65, Systolic Blood pressure 110.31  $\pm$  8.22, diastolic blood pressure 70.99  $\pm$  6.40 and the Mean arterial pressure was found to be 83.75  $\pm$  5.14 (Table 1)

In the investigations studied the mean blood glucose levels were 88.22  $\pm$  9.56, Urea 37.87  $\pm$  31.33, creatinine 1.15  $\pm$  0.47, sodium 131.75  $\pm$  4.65, potassium 4.03  $\pm$  0.82, Chloride 103.75  $\pm$  6.44. Complete blood count had mean value of 8729.03  $\pm$  4749.46, hemoglobin 8.98  $\pm$  1.48, Platelet count 153750.00  $\pm$  92470.05

The coagulation profile done showed a mean of 27.9  $\pm$  15.4 for prothrombin time, 2.42  $\pm$  1.3 for INR, 3.48  $\pm$  3.83 for bleeding time, 4.77  $\pm$  1.90 for clotting time

Liver function test was done in which total proteins had a mean of 6.32  $\pm$  0.89, Albumin 2.80  $\pm$  0.75, Globulin 3.53  $\pm$  0.63, Bilirubin 7.31  $\pm$  8.95, AST 91.46  $\pm$  91.92, ALT 55.75  $\pm$  67.97, ALP 161.87  $\pm$  58.64, GGT 41.21  $\pm$  22.67

The coagulation profile done showed a mean of 27.9  $\pm$  15.4 for prothrombin time, 2.42  $\pm$  1.3 for INR, 3.48  $\pm$  3.83 for bleeding time, 4.77  $\pm$  1.90 for clotting time

Ultrasound abdomen reported Cirrhosis of liver in all the subjects. 56.25% (18 patients) had splenomegaly, ascitis in 68.75% (22 patients)%. Portal hypertension was seen in 50% (16 patients) (Figure 1). Upper Gastrointestinal endoscopy showed 28.1% (9 patients) had Grade 1 varices, 28.1% (9 patients) had Grade 2

varices, 15.6%(5 patients) had Grade 3 varices, 9.3 %(3 patients) had Grade 4 varices. 18.3%(6 patients) had normal study. QT prolongation was seen in 25%(8 patients) on ECG whereas 21.8%(7 patients) had low voltage complexes and 53.1%(20 patients) had a normal ECG(Table 2)

ECHO was done to look for cardiac dysfunction(Table 3 and 4).On analyzing the data 26 patients had diastolic dysfunction(81.2%). 65.6%(17 patients) had grade 1 diastolic dysfunction and 34.6%(9 patients) had grade 2 diastolic dysfunction( Figure 2).No systolic dysfunction was found in these patients.

Positive co-relation was found between ascites and diastolic dysfunction.( $p=0.038$ )(Table 5).No correlation was found between age ,gender,alcohol with the echo findings.

MELD was calculated. 7 patients had scores between 19-29,20 between 29-39 and 5 over 40.(Table 6 ) No co-relation was found between severity of hepatic dysfunction (table 7) and cardiac changes .No co-relation was found between portal hypertension and cirrhosis of liver(Table 8).

#### IV. Discussion

This study was done in Mahatma Gandhi Medical college & Research Institute Pondicherry over a period of 18 months Due to the lack of data relevant to the Indian population regarding the cardiac abnormalities detected in Cirrhosis of liver this study was undertaken to assess the cardiac functions in Cirrhosis of liver and find out the incidence of cirrhotic cardiomyopathy due to cirrhosis of liver and not due to any other cause of Cardiomyopathy.

Our study has shown an high prevalence of cardiac abnormalities in patients diagnosed with Cirrhosis of liver.Majority of the patients(81.2%) were found to have diastolic dysfunction in the absence of any known cardiac abnormality.However no evidence of systolic failure was found in these patients.

On studying the data no significant association could be found between age ,gender and alcohol to the cardiac changes detected.Alcohol consumption also did not co- relate with the cardiac changes further proving that cardiac changes in alcoholic cirrhosis is due to cirrhosis per se and not due to alcohol.J Alexander et al compared alcoholic and non alcoholic groups and no found no co- relation between alcohol and the cardiac findings.<sup>7</sup>Lee et al also stated that cardiac changes are due to cirrhosis per se rather than alcohol.<sup>5</sup>

Presence of hyperkinetic circulation has been well documented in cirrhosis of liver.In our study the mean pulse rate was  $83.56 \pm 7.56$  which is very high compared to normal subjects. Mashford ML, Mahon WA et al showed a mean heart rate of  $86 \pm 2.9$ .<sup>8</sup> There were many studies done later which also proved this finding namely Lenz K,Lieinberger G et al reported a mean heart rate of  $101 \pm 2$  Vs  $78$ .<sup>9</sup> McCormick P.A;Chin J et al reported  $101/\text{min} \pm 2$  vs  $78$ .<sup>10</sup>

The present study thus showed that cirrhotic patients have an hyperdynamic circulatory state compared to the average heart rate of healthy subjects. The diastolic pressure was calculated to be  $70.99 \pm 6.40$ . McCormick P.A;Chin J et al calculated it to be  $56$ .<sup>10</sup> .However it was normal in this study. The same study calculated the mean arterial pressure to be  $86$  .The mean arterial pressure calculated in our study was  $83.75 \pm 5.14$ .Mashford ML, Mahon WA et al demonstrated a mean arterial pressure of  $85 \pm 2.4$  in cirrhotic patients.<sup>8</sup>

The presence of an hyperdynamic circulation proves that there is an increased venous return to the heart, increased heart rate and contractility. The blood pressure also reduces along with systemic vascular resistance. Our study however reported a normal mean for the blood pressures calculated.As stated earlier it appears that portal hypertension plays an important role for the development of hyperkinetic circulation. In this study 50% of the patients had portal hypertension.

Cirrhotic cardiomyopathy is defined as chronic cardiac dysfunction in patients having cirrhosis, characterized by blunted contractile responsiveness to stress or altered diastolic relaxation with electrophysiological abnormalities in the absence of known cardiac disease.<sup>7</sup> To diagnose cirrhotic cardiomyopathy presence of one or more of the following is required.<sup>11</sup>

1. Baseline Increased Cardiac Output But Blunted Ventricular Response To Stimuli
2. Systolic And /Or Diastolic Dysfunction
3. Absence Of Overt Left Ventricular Failure At Rest
4. Electrophysiological Abnormalities Including Prolonged Q-T Interval In Electrocardiography And Chronotropic Competence.

Electrocardiogram taken showed 25% had QT prolongation whereas 21.9% had low voltage complexes. Bernardi M et al stated QT prolongation as the major ECG abnormality in Cirrhotic patients which parallels with the severity of liver disease.<sup>12</sup>The author showed a prevalence of 42.9% in alcoholic and 47.1% in non alcoholic cirrhosis. S Samuiullah et al reported 21.6 % of the study population had prolongation of QT interval which correlated with the severity of liver disease.<sup>13</sup> Trivesani F et al showed that acute gastrointestinal bleed further prolongs the QT interval which itself is an independent marker for mortality.<sup>14</sup>

However present study did not find any significant co- relation between QT prolongation and Cirrhosis of liver. A larger sample size could have given a clearer picture.Also could be due to the definitive lack of

evidence between QT prolongation and life threatening arrhythmias in cirrhotics and needs to be investigated further in the future. Chest x-ray done to look for any abnormality was normal in the study group.

Our study showed Diastolic dysfunction on Echocardiography. J Alexander et al studied Indian patients with cirrhosis and found evidence of diastolic dysfunction.<sup>7</sup> He attributed these cardiac changes to cirrhotic cardiomyopathy in the absence of any other risk factors for cardiac diseases. Shaikh S et al studied 74 cirrhotics in Pakistan and found diastolic dysfunction in 15 patients.<sup>13</sup> He also correlated it with severity of cirrhosis.

Sun et al demonstrated diastolic dysfunction in 48.8% of cirrhotics.<sup>15</sup> The author also associated the cardiac changes with MELD score. Ruiz-Del-Arbol et al also found diastolic dysfunction in patients with cirrhosis.<sup>16</sup> He found that grade 2 diastolic dysfunction had an increased mortality and higher risk of developing hepatorenal syndrome type 1. The present study showed 65.3% of the patients with diastolic dysfunction had grade 1 diastolic dysfunction whereas 34.3% had grade 2 diastolic dysfunction. Merli et al detected 64% to have diastolic dysfunction at rest.<sup>17</sup> But the author did not find any association between the cardiac abnormalities detected and cirrhosis of liver.

Diastolic dysfunction is an early marker for cardiac impairment and precedes systolic failure leading to subsequent heart failure.<sup>18</sup> Our study showed 81.2% (26 patients) had diastolic dysfunction. It has also been demonstrated that diastolic dysfunction could contribute to the progression to HRS Type 1.<sup>16</sup> There are very few studies pertaining to the Asian population regarding cardiac changes in cirrhosis of liver.

Valeriano et al inferred that patients with cirrhosis of liver along with ascites had higher incidence of diastolic dysfunction than patients without ascites.<sup>19</sup> The E/A ratio was decreased in patients with ascites compared to those without ( $0.9 \pm 0.2$  vs.  $1.3 \pm 0.4$ ,  $P < 0.05$ ). Similarly Pozzi M et al stated in the presence of ascites cirrhosis was associated with left ventricular diastolic dysfunction and increased wall thickness.<sup>20</sup> It was speculated whether the neurohumoral overactivity was causing the impaired ventricular relaxation.

Torregrosa M et al showed a significant relationship between ascites and diastolic dysfunction at rest and during stress compared to non ascitic patients.<sup>21</sup> Merli M et al demonstrated that diastolic dysfunction is prevalent in patients with ascites compared to non ascitic patients (77% vs 56%  $p = 0.04$ ).<sup>17</sup> An E/A ratio of less than 1 was a marker of slow clearance of ascites.<sup>22</sup>

Riuz-Del-Arbol et al compared diastolic dysfunction with ascites and elevated neurohumoral markers and ascites with normal neurohumoral markers.<sup>16</sup> On comparison the author found that severity of diastolic dysfunction was more when the neurohumoral markers were elevated along with ascites. Recent studies show that diastolic dysfunction is an independent marker for mortality.<sup>23</sup> In the absence of any known risk factors for cardiac illness diastolic dysfunction can be attributed to cirrhotic cardiomyopathy.

MELD score was calculated to find any association between cardiac abnormalities and severity of liver dysfunction.<sup>24,25</sup>

Sun et al showed a positive correlation between the two.<sup>15</sup> It was also suggested by the author that the scoring is useful in patients awaiting liver transplant. Riuz-Del-Arbol et al correlated association between LVDD (Left ventricular diastolic dysfunction) and portal hypertension and normal serum creatinine.<sup>150</sup> MELD score also had a significant correlation in the study. However there is still some controversy over the varicity of MELD score in predicting the severity of liver disease with the cardiac abnormalities detected. J Alexander et al did not find any association between the two parameters.<sup>7</sup> Merli et al did not find a positive relation between cardiac changes and severity of liver dysfunction.<sup>154</sup> Though the present study did not find any association between the two further Indian studies are required in order to establish an association between the two parameters especially for patients awaiting liver transplant in India. The Study sample size was not large enough to prove systolic failure in patients with Cirrhosis of liver. However the study proves an significant association between Cirrhosis of liver and diastolic dysfunction which can be attributed to cirrhosis itself. Due to lack of sufficient Indian data it requires further investigations. The study design was such that liver cirrhosis was diagnosed on the basis of ultrasound which still does not diagnose all cases of cirrhosis accurately. A liver biopsy is still the gold standard for diagnosing cirrhosis of liver. It is important to note that a normal echocardiogram does not rule out cardiac dysfunction in Cirrhosis of liver. Tissue Doppler imaging is a better diagnostic tool than ECHO to detect cardiac abnormalities. Blunted cardiac response to stress using pharmacological agents would be ideal to look for systolic failure in cirrhotics.

## V. Conclusion

1. This study showed a very high prevalence of Diastolic dysfunction in Cirrhosis of liver.
2. Diastolic dysfunction in Cirrhotics where no other known cardiac risk factors are present should be attributed to Cirrhotic Cardiomyopathy.
3. Echocardiography is a cheap and non invasive method to detect cardiac abnormalities and can be used for patients with cirrhosis of liver.
4. Very few studies at present are available on the treatment of cirrhotic cardiomyopathy and is an scope for further studies.

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Tables

Table 1 Distribution of Vital parameters of patients studied

Vital parameters	No. of patients (n=32)	%	Mean ± SD	
Pulse rate				
<60	0	0.0	83.56±7.65	
60-80	11	34.4		
80-100	21	65.6		
SBP				
<120	21	65.6	110.31±8.22	
120-139	11	34.4		
140-159	0	0.0		
160-200	0	0.0		
>200	0	0.0		
DBP				
<80	24	75.5		70.99±6.40
80-89	8	25.0		
90-99	0	0.0		
100-119	0	0.0		
120 & above	0	0.0		
MAP				

<70	0	0.0	83.75±5.14
70-100	32	100.0	
>100	0	0.0	

**Table 2** ECG findigns

ECG findings	No. of patients (n=32)	%
Normal	17	53.1
QT prolongation	8	25
LVC	7	21.8

**Table 3** ECHO findings

ECHO findings	No. of patients (n=32)	%
Normal	6	18.7
Diastolic Dysfunction	26	81.2
LV dysfunction	0	0.0

**Table 4** Diastolic dysfunction

Diastolic Dysfunction	No. of patients (n=30)	%
Grade I	17	65.6
Grade II	9	34.3

**Table 5** Comparison of Ascites and Diastolic dysfunction

	DIASTOLIC DYSFUNCTION		TOTAL
	YES	NO	
ASCITES NO	6	4	10
ASCITES YES	20	2	22
TOTAL	26	6	32

Ascites is statistically significant associated with ECHO findings with p=0.038

**Table 6** Meld Score

MELD SCORE	No. of patients	%
<9	0	0.0
9-19	0	0.0
19-29	7	21.9
29-39	20	62.5
>39	5	15.6
Total	32	100.0

**Table 7** Comparison of Meld Score and Echo findings

MELD SCORE	Total number of patients	Echo findings	
		Diastolic Dysfunction	Normal
<9	0	0	0
9-19	0	0	0
19-29	7	7(100%)	0
29-39	20	15(75%)	5(25%)
>39	5	4(80%)	1(20%)
Total	32	26(81.4%)	6(18.2%)

MELD SCORE is not statistically associated with ECHO findings with p=0.317

**Table 8** Comparison of Portal hypertension and Echo findings

Portal hypertension	Total number of patients	Echo findings	
		Diastolic Dysfunction	Normal
No	16	12	4
Yes	16	14	2
Total	32	26	6

Portal hypertension is not statistically associated with ECHO findings with p=0.59

Figure 1 Ultrasound findings

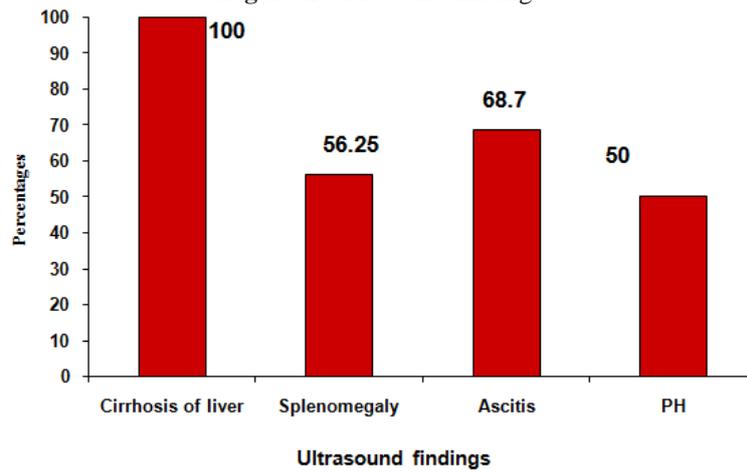


Figure 2 Echo findings

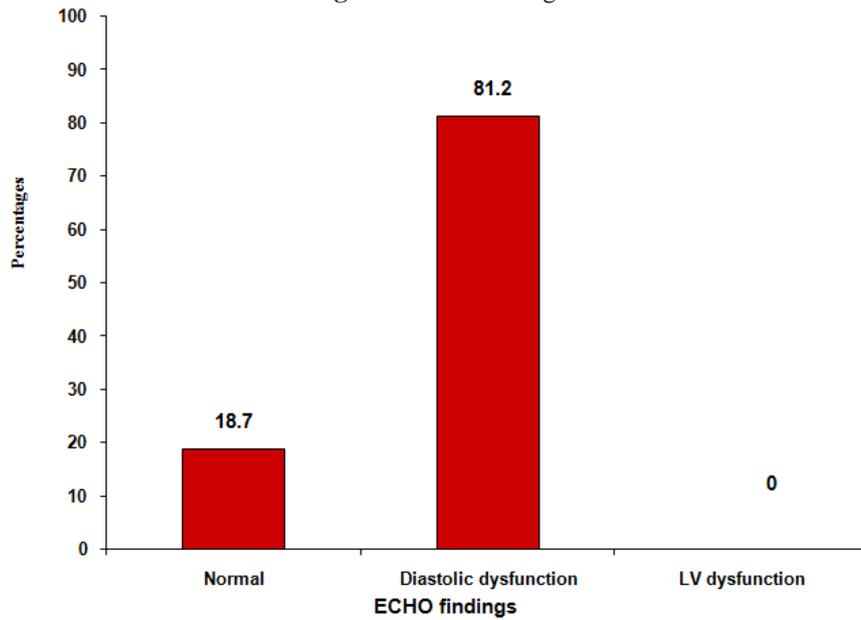


Figure 3 Grades of diastolic dysfunction

