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STUDY OF CARDIAC PROFILE IN PATIENTS OF LIVER CIRRHOSIS

General Medicine

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ABSTRACT

Background: Liver cirrhosis is the production, in response to chronic liver injury, of degenerative nodules surrounded by fibrous band. Cirrhotic patients may also have renal, respiratory, hemodynamic and cardiac dysfunction, which increases morbidity and mortality in addition to liver damage. The prevalence of cirrhotic cardiomyopathy is estimated to be between 40 - 50 % in cirrhosis. Present study was aimed to study cardiac profile in patients of liver cirrhosis. **Material and Methods:** Present study was single-center, observational, cross-sectional study, conducted in patients of age > 18 years, diagnosed case of liver cirrhosis. Child-Pugh-Turcotte Score & MELD Score were calculated. **Results:** Present study included a total of 103 study participants. The mean age of the study participants was 46.66 ± 9.31 years, majority were males (90.29%) History of alcohol (85.44%) was the most common etiology seen followed by Hepatitis B (9.71%). According to their Child Pugh scores, most (88.35%) of the patients of liver cirrhosis belonged to class C, According to their MELD scores most (88.35%) of the patients of liver cirrhosis belonged to class 3. We found a statistically significant association between the Child Pugh's class and MELD class ($\chi 2$ (1, n=103) =52.956, p = 0.001). The patients of liver cirrhosis who participated in our study are disproportionately distributed in CP class C and MELD class 3 as compared to the CP class B and MELD class 2. **Conclusion:** Diastolic dysfunction is common in patients of liver cirrhosis irrespective of CP and MELD class. Patients of CP class C and MELD class 3 should be monitored for QT prolongation.

KEYWORDS

Cardiac dysfunction, cirrhosis of liver, CP score, MELD score

INTRODUCTION

Liver cirrhosis is the production, in response to chronic liver injury, of degenerative nodules surrounded by fibrous bands that may be caused by chronic alcohol consumption, hepatitis B or C infection, non-alcoholic steatohepatitis or other liver insults that may lead to end-stage liver disease[1].

Cirrhotic patients may also have renal, respiratory, hemodynamic and cardiac dysfunction, which increases morbidity and mortality in addition to liver damage[3]. Cirrhotic cardiomyopathy was first established as "cardiac dysfunction in patients with liver cirrhosis characterized by impaired contractile responsiveness to stress and/or altered diastolic relaxation with electrophysiological abnormalities in the absence of other known cardiac diseases".[2]

The prevalence of cirrhotic cardiomyopathy is estimated to be between 40 percent and 50 percent in cirrhosis, but this term has a substantial lack of strict criteria, while its production seems to be independent of the etiology of liver disease[4]. Most patients are diagnosed with diastolic heart failure and/or high- output heart failure during the clinical decompensation process of cirrhosis[5].

In liver cirrhosis, impaired cardiac function is sometimes undiagnosed, but this raises the risk of mortality in the sense of acute, decompensated cirrhosis[6]. Present study was aimed to study cardiac profile in patients of liver cirrhosis.

MATERIAL AND METHODS

Present study was single-center, observational, cross-sectional study, conducted in department of General Medicine, at NKPSIMS & RC medical college & Lata Mangeshkar hospital, Digdoh, Nagpur, India. Study duration was of 2 years (from November 2018 to November 2020). Study approval was obtained from institutional ethical committee.

Inclusion criteria

- Patients of age > 18 years, diagnosed case of liver cirrhosis, willing to participate in present study

Exclusion criteria

- Patients with known etiology of cardiovascular disease like hypertension, congestive cardiac failure, congenital heart disease, ischemic heart disease, valvular heart disease,
- Patients with severe anaemia (Hb<5), COPD, diabetes, Thyroid disorders,
- Patients with history of smoking

Study was explained to patients in local language & written consent was taken for participation & study. History was noted, followed by a detailed clinical examination and blood investigations (Complete Blood Count, Liver Function Test, Kidney Function Test, Prothrombin Time-International Normalized Ratio). Ultrasonography of abdomen was done for detection of cirrhosis. Further, a 12 lead ECG was done to study the ECG changes and calculate QT interval manually, and corrected QT (QTC) by using Bazett's formula

- Bazett's formula--QTC=QT Interval/√RR Interval
- Prolonged QT interval defined as value > 440 msec (0.44sec).

2D Echo was done with adult probe, on TOSHIBA make 2D Echo machine and was used to assess any cardiac anomaly with special reference to left ventricle end diastolic volume, I.V. septal thickness, left ventricular posterior wall thickness and to assess E/A ratio where E stands for early maximum left ventricular filling velocity, and A for late diastolic left ventricle filling velocity; left ventricle systolic function was calculated using ejection fraction (EF %) and diastolic function was assessed by E/A and if ratio was < 1, it was taken as diastolic dysfunction.

Operational definitions

CIRRHOSIS OF LIVER- Diagnosed by clinical signs (icterus, ascites, signs of liver cell failure) and symptoms (abdominal distension), biochemical markers (A/G reversal, raised bilirubin levels) and radiological evidence (Ultrasonography).

Cirrhoticcardiomyopathy[7]:

diagnostic and supportive criteria-

- Systolic dysfunction: Blunted increase in cardiac output on exercise, volume challenge or pharmacological stimuli or resting ejection fraction <55 %
- Diastolic dysfunction: The ratio of early to late (atrial) phases of ventricular filling or E/A ratio <1.0 (age-corrected), prolonged deceleration time (>200ms), or prolonged isovolumetric relaxation time (>80 ms)
- Supportive criteria: electrophysiological abnormalities, abnormal chronotropic response, electromechanical uncoupling/ dyssynchrony, prolonged QTc interval, enlarged left atrium, increased myocardial mass, increased brain natriuretic peptide (BNP) and pro-BNP, or increased troponin I.

Methods of measurement Child-Pugh-Turcotte Score

Measure	1point	2points	3points
Total bilirubin umol/L (mg/dL)	<34(<2)	34-50(2-3)	>50(>3)

Serum albumin	>35	28-35	<28
Prothrombin time OR	<4.0	4.0-6.0	>6.0
INR	<1.7	1.7-2.3	>2.3
Ascites	None	Mild	Moderate to severe
Hepatic encephalopathy	None	Grade I-II	Grade III-IV
Points	5-6	7-9	10-15
Class	A	B	C

MELD Score - MELD score was calculated by the formula-3.78 *ln [Serum bilirubin (mg/dL)] + 11.2*ln [INR] + 9.57*ln [serum creatinine (mg/dL)] + 6.43|

MELD Score	Class
<9	1
10-19	2
>20	3

Data was collected and compiled using Microsoft Excel, analyzed using SPSS 23.0 version. Frequency, percentage, means and standard deviations (SD) was calculated for the continuous variables, while ratios and proportions were calculated for the categorical variables. Difference of proportions between qualitative variables were tested using chi- square test or Fisher exact test as applicable. P value less than 0.05 was considered as statistically significant.

RESULTS

Present study included a total of 103 study participants. The mean age of the study participants was 46.66 ± 9.31 years. Most (38.83%) of our study participants were from the age group 41-50 years, followed by equal proportion (26.21%) of participants each from the 31-40 years and 51-60 years age group. 93 out 103 study participants were males (90.29%) with 10 females (9.71%).

Table 1- Age & Gender-wise distribution

Characteristics	No. of Cases	Percentage
Age (in years)		
< 30	2	1.94%
31-40	27	26.21%
41-50	40	38.83%
51-60	27	26.21%
Above 60	7	6.80%
Gender		
Male	93	90.29%
Female	10	9.71%

We found that most of the patients had abdominal distension (89.32 %) which was closely followed by the swelling over the body (87,38 %), yellowish discolouration of skin and sclera (66.02%), history of either hematemesis or melaena (32.04 %).

Table 2- Distribution of patients according to Symptoms

Symptoms	No.	%
Abdominal distension	92	89.32
Yellowish discolouration	68	66.02
Swelling	90	87.38
Hematemesis /Malena	33	32.04
Altered sensorium	8	7.77
Chest Pain	15	14.56
Cough	10	9.71
Breathlessness	13	12.62

According to the etiology, history of alcohol (85.44%) was the most common etiology seen followed by Hepatitis B (9.71%) while 4.85% of the patients had etiologies other than these.

Table 3. Etiology

Etiology	No.	%
H/o Alcohol	88	85.44
Hepatitis B	10	9.71
Wilson's Disease	1	0.97
Cryptogenic Cirrhosis	4	3.88

All the patients of liver cirrhosis had pallor (100.00%). Out of 103 patients of liver cirrhosis, 99 (96.12%) were afebrile, 91 (88.35%) had oedema on feet, and 70 (67.96%) had icterus. Around 7.77% of the patients had clubbing while 6.80% of the patients had loss of axillary and or pubic hair. Also, 4.85% of the patients had parotid swelling while 3.88% each had alopecia and gynaecomastia. Only 1 (0.97%) patient out of 103 had testicular atrophy. 89.32% of these patients had ascites. Palpable spleen was seen in 12.62% of the patients while flaps were appreciable in 7.77% of the patients.

Table 4. Clinical examination

General examination	No.	%
Pallor	103	100.00
Icterus	70	67.96
Clubbing	8	7.77
Oedema feet	91	88.35
Gynecomastia	4	3.88
Parotid swelling	5	4.85
Alopecia	4	3.88
Testicular atrophy	1	0.97
Loss of axillary/pubic hair	7	6.80
Systemic examination		
Ascites	92	89.32
Palpable spleen	13	12.62
Flapping tremors	8	7.77

According to their Child Pugh scores, scores were found in the range of 9 – 13 with a mean score of 11.10 ±1.05.Most (73.79%) of the patients had scores of 11 and 12. Most (88.35%) of the patients of liver cirrhosis belonged to class C while none of them could be categorized into class A.

Table 5. Distribution of patients according to CP score

CP score	No.	%
9	12	11.65
10	12	11.65
11	35	33.98
12	41	39.81
13	3	2.91
CP class		
B	12	11.65
C	91	88.35

According to their MELD scores, scores were found in the range of 14 – 35 with a mean score of 25.13 ± 4.43. According to the class as per the MELD scoring. Most (88.35%) of the patients of liver cirrhosis belonged to class 3 while none of them could be categorized into class 1.

Table 6. Distribution of patients according to MELD class

MELD class	No.	%
2	12	11.65
3	91	88.35

We found that 85 (82.52%) out of the total 103patients of liver cirrhosis had no cardiomegaly while 18 (17.48%) had cardiomegaly.

Table 7. Cardiomegaly on X-ray

Cardiomegaly	No.	%
Yes	18	17.48
No	85	82.52

The participants had a mean Left Ventricular mass of 163.92± 15.00 grams, and a mean Left ventricular ejection fraction of 62.49 ± 2.46%. Most (48.54%) of these patients showed features of diastolic dysfunction on 2D echo, which was followed by the presence of valvular abnormalities (25.24%) majority of which were mild mitral regurgitation and mild tricuspid regurgitation. 14 (13.59%) each out 103 patients had features chamber enlargement and mild pulmonary hypertension while 2 (1.94%) patients had pericardial effusion.

Table 8. Distribution of patients according to 2D Echo findings

Findings	No. / Mean ± SD	Range
LV mass (gm.)	163.92 ± 15.00	122–201
LVEF (%)	62.49±2.46	57– 70
Chamber enlargement	14	13.59
Diastolic dysfunction	50	48.54
Valvular abnormalities	26	25.24

Pulmonary hypertension	14	13.59
Pericardial effusion	2	1.94

A Pearson's chi-square test was calculated for comparing the frequency of patients in the CP class and the MELD class. We found a statistically significant association between the Child Pugh's class and MELD class ($\chi 2$ (1, n=103) =52.956, p = 0.001). The patients of liver cirrhosis who participated in our study are disproportionately distributed in CP class C and MELD class 3 as compared to the CP class B and MELD class 2.

Table 9. Association between CP class and MELD class

CP class	MELD class 2		MELD class 3	
	No.	%	No.	%
B	9	75.00	3	3.30
C	3	25.00	88	96.70
Total	12	100.00	91	100.00
Pearson chi 2 (1) = 52.9560 P=0.001(S)				

We found that there is no significant association of cardiomegaly, diastolic dysfunction, chamber enlargement and valvular abnormalities individually with either the CP classes or the MELD classes. Also, the association of EA ratio with either CP classes or the MELD classes was not significant. While comparing the different cardiac measurements, we found that there is a significant association of LA size with the CP classes (p<0.001) and separately with the MELD classes (p<0.001). The LV mass and IVSD are significantly associated individually with the CP classes (LV mass: p value <0.001; IVSD: p value = 0.022) and separately with the MELD classes (LV mass: p value <0.001; IVSD: p value <0.006). However, there was no significant association seen between the LVPWD and either the CP classes or the MELD classes. The LVED was significantly associated with the CP classes (p value < 0.001) and separately with the MELD classes (p value <0.001).

Table 10. Comparison of 2D ECHO parameters by CP class and MELD class

Parameter	CP class				MELD class			
	B (n=12)		C (n=91)		2 (n=12)		3 (n=91)	
	Yes	%	Yes	%	Yes	%	Yes	%
Cardiomegaly	2	16.67	16	17.58	0	0.00	14	15.38
	Pvalue=0.937,NS				Pvalue=0.144,NS			
Diastolic Dysfunction	4	33.33	46	50.55	4	33.33	46	50.55
	Pvalue=0.262,NS				Pvalue=0.262,NS			
Chamber Enlargement	0	0.00	14	15.38	0	0.00	14	15.38
	Pvalue=0.144,NS				Pvalue=0.144,NS			
Valvular abnormalities	1	8.33	25	27.47	1	8.33	25	27.47
	Pvalue=0.151,NS				Pvalue=0.151,NS			
EA ratio	Mean	SD	Mean	SD	Mean	SD	Mean	SD
	1.01	0.18	1	0.18	1.03	0.20	1	0.17
	Pvalue=0.8789,NS				Pvalue=0.5027,NS			
LA size	36	0.85	38.07	1.91	35.83	0.94	38.09	1.88
	Pvalue=00004, S				Pvalue=00001, S			
LVmass	144.5	13.60	166.48	13.26	147	13.06	166.15	13.83
	Pvalue=0.0001,S				Pvalue=0.0001,S			
IVSD	9.5	0.80	10.01	0.71	9.42	0.79	10.02	0.70
	Pvalue=0.0224,S				Pvalue=0.0065,S			
LVPWD	8.42	0.67	8.48	0.81	8.5	0.52	8.47	0.82
	Pvalue=0.7845,NS				Pvalue=0.9105,NS			
LVED	47.33	0.78	50.41	1.92	47.83	1.47	50.34	1.97
	Pvalue=0.0001,S				Pvalue=0.0001,vS			

DISCUSSION

A rarely mentioned complication of cirrhotic cardiomyopathy is identified as the presence in patients with liver cirrhosis of chronic cardiac dysfunction characterised by abnormal and blunted contractile responsiveness to physiological, pathological or pharmacological stress, in the absence of established cardiac disease and irrespective of the causes of cirrhosis[2].

The present study was conducted on 103 subjects over 2 years. In the present study the mean age of the patients was 46.66 ± 9.31 years & majority were in the age group of 41-60 years. This was similar to study conducted by Gosavi et al.,[8] where mean age was 48.7 years. In a study conducted by Karki et al.,[9] the mean age was 50.88 ±11.63 years with majority of patients in age group between 40-60 years.

In the present study, the study participants were predominantly males (90.29%) with 9.71% females. This is similar to study by Chandey M et al.,[10] with 91.1% male and 8.89 % female participants. In study by Punekar et al., [11] 72% were male and 28% were female. This can be attributed to more prevalence of alcohol consumption in males compared to females.

Alcohol (85.44%) was the most common etiology seen in the patients of liver cirrhosis which was followed by Hepatitis B (9.71%) while 4.85%of the patients had etiologies other than these. Similarly in study by Chandey M et al.,[10] and Punekar et al.,[11] alcohol was the most common etiology (75% and 57 % respectively). Karki et al.,[9] conducted a study on 85 subjects of which 72(84.70%) had history regular alcohol ingestion. This was followed by hepatitis C (12.94%) and hepatitis B (8.23%) associated cirrhosis.

It is known that in liver cirrhosis, portal blood flow is distorted which is accompanied by a decrease in hepatic clearance of bilirubin. In addition, portosystemic shunting as well as splenomegaly results in an increase in haemolysis and production of bilirubin. These together result in an increase in the concentration of unconjugated bilirubin in serum. In advanced cirrhosis, glucuronyl conjugation of bilirubin and biliary excretion of conjugated bilirubin are markedly impaired and jaundice appears. In a study by S Patil et al.,[5] mean serum bilirubin level in MELD class 1 was 1.19 ± 0.13 mg/dL, in MELD class 2 it was 2.36 ± 0.4 mg/dL and in MELD class 3 it is 7.22 ± 13.7 mg/dL inferring thereby a significant difference in serum bilirubin levels in each MELD groups (p<0.001). In study by Gosavi et al.,[8] liver profile parameters of the study subjects were assessed, mean Serum bilirubin level was 2.5 ±1.23mg/dl (0.81-3.83).

The mean serum albumin level in present study is 2.42 ± 0.37 gm/dl. There was statistically significant difference in serum albumin levels between CP Class B and C (2.78±0.19 and 2.37 ±0.36 respectively; P value=0.0002) and MELD class 2 and 3 (2.63±0.35 and 2.39±0.36 respectively; P value=0.0376). Most commonly, inadequate synthesis of albumin in the presence of increased catabolism due to significant systemic illness contributes to an overall hypoalbuminemia. Hypoalbuminemia is a feature of chronic and advanced hepatic cirrhosis. Significant and severe chronic hepatic impairment is needed before a noticeable decrease in plasma albumin is manifest. Hence the association of lower serum albumin level in a more severe liver cirrhosis patient is explainable.

In study by Gosavi et al.,[8] liver profile parameters of the study subjects were assessed, the mean serum albumin level was 2.93± 0.27gm/dl (2.5 – 3.8), however it did not study the serum albumin levels with severity. In a study by Abdel Monem et al.,[10] serum albumin(g/dl) level in CP class A, B and C were 3.79 ± 0.1666, 2.932 ± 0.070, 2.317 ± 0.224 (P value= 0.001) indicating that with the progression of liver disease from CP class A to CP class C there was significant decrease in serum albumin with the progression of liver disease.

In a study conducted by Chandey M et al.,[141] 6.66% were CP class A,43.33% were from class B and 50% were from class C. In another study by Abdel Mondem et al., (2020)[143] 30% belonged to class A , 30% to class B and 40 % to class C. In study by S Patil et al.,[5] 33.33% belonged MELD class 1 ,50 % belonged to MELD class 2 and 16.66 % to MELD class 3.

A higher Qtc was reported in the patients with a severe CP or MELD Class. In the present study Qtc in Class B was 0.40±0.03 seconds and class C was 0.44±0.03 seconds which was statistically significant (P value=0.0001). Even though the mechanism of the prolongation of QT intervals is not fully elucidated, it is attributed to autonomic changes caused by cirrhosis. The QT interval extends from the earliest activation of ventricular myocardium to the end of the latest ventricular repolarization. Therefore, QT intervals can be prolonged either because of a delay in the activation or because the complex process of repolarization is prolonged. Repolarization, per se, could be prolonged due to electrolytic abnormalities or previous cardiac

disease. Mozos et al.,[13] showed that corrected QT values were higher in Child-Pugh C cirrhotic patients (Child-Pugh A: 462 ±25 ms, Child-Pugh B: 493 ± 62 ms and Child-Pugh C: 520± 45 ms, p<0.001). In a study of 409 cirrhotic patients, Bal et al.,[15] reported higher corrected QT in the Child-Pugh C group than the Child-Pugh B group (451±43 vs 434 ± 31ms, p < 0.001). This, evidence and related literature strengthens the notion that Child-Pugh C or MELD class 3 patients should be monitored for QT prolongation.

The hyperdynamic circulatory changes can lead to the cardiac dilatation of the left chambers and the development of functional changes in the heart. In a study by S Patil et al.,[5] mean LA size in MELD 2 (37.09 ± 4.20mm) and MELD 3 (37.82±3.98 mm) were higher than MELD 1 (33.81± 2.41 mm) which is statistically important.

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.[5] The size of left ventricle at the end of diastole in present study participants was found to be in the range of 45 – 54 mm with a mean size of 50.05 ± 2.07 mm. There was statistically significant difference in LVED between CP Class B and C (47.33 ± 0.78 mm and 50.41 ± 1.92 mm respectively; P value=0.0001) and MELD class 2 and 3 (47.83 ± 1.47 mm and 50.34 ± 1.97 mm respectively; P value=0.0001). In a study by Shweta Patil et. Al.,[5], similar observations persisted for left ventricular end diastolic diameter which varied from 48.28±3.58 mm in MELD class 1 to 52.39±4.03 mm in MELD class 3 (p = 0.038), thereby inferring that higher is the degree of MELD score, worse is the left ventricular diastolic dysfunction. In a study by Silvestre et al.,[16] mean LVED is 49.26 ± 5.66 mm and it significantly correlated with MELD score. Patients with MELD≥16 (50.17 ±6.07mm) had significantly higher left- atrial diameter compared with patients with MELD scores <16 points (48.59 ± 5.25 mm; P value=0.05).

In the cirrhotic patients, due to the lengthening of systolic time periods affected by electromechanical coupling, such as electromechanical delay and pre-ejection duration, total electromechanical systole was prolonged, possibly due to a reduced response to the adrenergic drive[5]. It is known that the reductions in circulating blood volume and hyperdynamic circulation enhance sympathetic nervous system and renin- angiotensin- aldosterone system activities. The resulting increased cardiac output and reduced systemic vascular resistance may induce myocardial remodeling and left ventricular hypertrophy, causing systolic and diastolic dysfunction and, hence, cardiomyopathy.

Diastolic dysfunction is a frequent feature of cirrhotic cardiomyopathy, which is most commonly assessed by studying the diastolic transmitral flow pattern. An increased atrial contribution to late ventricular filling, leading to a reduced E wave to A wave ratio suggests Grade 1 diastolic dysfunction. Other features include prolonged IVRT and DT due to increased resistance to ventricular inflow.

CONCLUSION

Cardiac dysfunction may be an additional cause of morbidity and mortality in patients of cirrhosis of liver. Diastolic dysfunction is common in patients of liver cirrhosis irrespective of CP and MELD class. Other parameters on 2D echo like LVED, IVSd, LA size and LV mass correlate with the severity of liver cirrhosis. Patients of CP class C and MELD class 3 should be monitored for QT prolongation.

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