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ASSESSMENT OF CARDIAC FUNCTIONS IN CIRRHOSIS OF LIVER

Medicine

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ABSTRACT

INTRODUCTION

Cirrhosis of liver is a common sequele of so many chronic liver disorders and it has known complication like UGI bleed , ascites and portal hypertension. The aim of present study was to esteblish adverse effect of cirrhotic liver on cardiovascular system and presence of cirrhotic cadiomyopathy in such patients.

Objective: 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 50 patients with cirrhosis of liver were enrolled after excluding any known cardiac illness and known causes of cardiomyopathy.Patients were subjected to sonographic evaluation and fibroscan to establish the diagnosis of 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 50 patients very high prevalence of diastolic dysfunction was found among the study group. A significant correlation was found between Diastolic dysfunction and ascitis. Alcohol consumption was not statistically associated with the cardiac findings. Cardiac abnormalities did not correlate with the severity of liver dysfunction In our present study diastolic dysfunction was calculated by E/A ratio which was present in 26 patients (52%). ECG changes of long QTc interval was seen in 34 of patients. Significant statistical correlation between CP score and QTc interval,(p-value 0.0274) was seen in my study. In my study there was significant statistical correlation between CP class and LVEDD, (p-value 0.0328) and statistical correlation between CP class and LV mass.(p-value 0.0374) and statistical correlation between CP class and E/A ratio, (p-value 0.0451).

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.

KEYWORDS

Fine needle aspiration Cytology, FNAC, Cervical lymphadenitis, Histopathology.

INTRODUCTION

Cirrhosis is a chronic disease of the liver that leads to a number of complications, some of which may eventually prove fatal. It is a common hepatological disorder seen in clinical practice. Cirrhosis is a pathologically defined entity that is associated with a spectrum of characteristic clinical manifestations.

Cirrhosis results from different mechanisms of liver injury that lead to necroinflammation and fibrogenesis; histologically it's characterized by diffuse nodular regeneration surrounded by dense fibrotic septa with subsequent parenchymal extinction and collapse of liver structures, together causing pronounced distortion of hepatic vascular architecture.[1]

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.[2] 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[3] In 1989, Lee reported the reduced cardiac response was due to cirrhosis per se, rather than alcohol[3] These findings led to the concept that cirrhosis itself triggers cardiac dysfunction and was termed as Cirrhotic Cardiomyopathy[4]

Cardiac abnormalities have been studied by many researchers, of which Kowalski et al[2] were the first to report that patients with cirrhosis had abnormal cardiovascular function and a prolonged QT interval.

Cirrhotic cardiomyopathy is a form of 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 other known cardiac disease.

The main clinical features of cirrhotic cardiomyopathy include baseline increased cardiac output, attenuated systolic contraction or diastolic relaxation and electrical conductance abnormalities (prolonged QT interval). These effects are more pronounced in patients with ascites compared to those without.

Liver functions are affected in the course of cardiac diseases, and similarly, liver diseases affect cardiac functions. Advanced liver cirrhosis is associated with an increase in blood volume, a reduction in systemic vascular resistance, and an increase in cardiac output.[5]

Hence, this study will aim to evaluate the structural and functional abnormalities of cardiac function in patients with cirrhosis of liver.

AIM AND OBJECTIVES

To establish relation between cirrhosis of liver and its effect on cardiac function and structure.

MATERIALS AND METHODS

METHODOLOGY

PLACE OF STUDY:
Shri Ram MurtiSmarak Institute of Medical Science, Bareilly,Uttar Pradesh.

SAMPLE SIZE : Total of 50 patients
TYPE OF STUDY: Cross sectional study

INCLUSION CRITERIA
- Adult male and female age > 18 years from OPD/IPD of SRMS-IMS hospital, Bareilly.
- All Patient admitted for cirrhosis diagnosed by clinically, biochemically, radiologicaly (ultrasound abdomen) and fibroscan

EXCLUSION CRITERIA
- Age <18 years
- Known case of:
- Cardiomyopathy
- Coronary artery disease
- Hypertension,
- Heart failure
- Congenital heart disease,
- Valvular heart diseases
- Corpulmonale

All the patients of cirrhosis because of various etiologies will be evaluated thoroughly :

A. Routine Investigation.
a. Hb, TLC, PLATELET COUNT
b. SERUM BILURUBIN, SGPT, SGOT
c. UREA, CREATININE, SODIUM, POTASSIUM
d. URINE R/M
e. CHEST X- RAY
B. Child-Turcotte-Pugh (CTP) score

Child-Turcotte-Pugh Classification for Severity of Cirrhosis			
Clinical and Lab Criteria	Points*		
	1	2	3
Encephalopathy	None	Mild to moderate (grade 1 or 2)	Severe (grade 3 or 4)
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
Prothrombin time Seconds prolonged International normalized ratio	<4 <1.7	4-6 1.7-2.3	>6 >2.3
*Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points)			
Class A = 5 to 6 points (least severe liver disease)			
Class B = 7 to 9 points (moderately severe liver disease)			
Class C = 10 to 15 points (most severe liver disease)			

C. Electrocardiography (ECG)
D. Echocardiography (2D ECHO)
a. Left ventricular diastolic function (E/A ratio)
b. Systolic function (ejection fraction)
c. Wall thickness (left ventricle posterior wall thickness + interventricular septum thickness).
E. In my study cirrhosis of liver was taken when the Ultrasound abdomen shows coarse echotexture of liver and Fibroscan shows value >12.5 kpa.

A 12-lead surface ECG at speed of 25mm/s. All the parameters like heart rate, QRS axis, PR interval, QTc interval along with any other abnormal findings were noted. QT interval was corrected using the BAZET formula: QTc = QT/√(RR in seconds). The adjusted QT of 440 milliseconds or more was considered as prolonged QT interval.

In this study, 2D ECHO machinewas usedto assess the cardiac structure in special reference to left ventricular end diastolic diameter (LVEDD), interventricular septal thickness (IVS), left ventricular posterior wall thickness (LVPW) and E/A ratio, in which E velocity is early maximal ventricular filling velocity, and A velocity is late diastolic or atrial velocity. Echocardiography was performed according to guidelines of American Society of Echocardiography.

The left ventricular mass was calculated using the formula known as the 'ASE- cube formula'

LV Mass (g) = 0.8{1.04[([LVEDD + IVS + LVPW]3 - LVEDD3)]} + 0.6

Observations

Table 1 :Distribution of the study subjects according to symptoms present*

	CP CLASS				Total	
	B		C			
	N	%	N	%	N	%
Fatigue	1	14.3	9	20.9	10	20
Weakness	2	28.6	8	18.6	10	20
Abd Pain	2	28.6	11	25.6	13	26
Nausea/Vomitting	0	0	4	9.3	4	8
Icterus	4	57.1	32	74.4	36	72
Breathlessness	2	28.6	12	27.9	14	28
Total	7	100	43	100	50	100

*Multiple responses

Table 2 : Distribution of the study subjects according to QT interval

		CP CLASS				Total	
		B		C			
		N	%	N	%	N	%
Qtc	Normal (≤ 440ms)	5	71.4	11	25.6	16	32.0
	Prolonged (> 440ms)	2	28.6	32	74.4	34	68.0
Total		7	100	43	100	50	100

Table 3 : Distribution of the study subjects according to LVEDD

		CP CLASS				Total	
		B		C			
		N	%	N	%	N	%
LVEDD	< 56mm	7	100.0	23	53.5	30	60.0
	≥ 56mm	0	0.0	20	46.5	20	40.0
Total		7	100	43	100	50	100

Table 4 : Distribution of the study subjects according to LVEF

		CP CLASS				Total	
		B		C			
		N	%	N	%	N	%
LVEF	< 55	0	0.0	0	0.0	0	0.0
	≥ 55	7	100.0	43	100.0	50	100.0
Total		7	100.0	43	100.0	50	100.0

Table 5 : Distribution of the study subjects according to LV mass

LV Mass		CP CLASS				Total	
		B		C			
		N	%	N	%	N	%
Female	≤ 150	0	0.0%	0	0.0%	0	0.0%
	> 150	1	100.0%	6	100.0%	7	100.0%
	Total	1	100.0%	6	100.0%	7	100.0%
Male*	≤ 200	5	83.3%	14	37.8	19	44.2
	> 200	1	16.7%	23	62.2	24	55.8
	Total	6	100.0%	37	100.0%	43	100.0%

*p-value 0.0374

Table 6 : Distribution of the study subjects according to E/A Ratio

		CP CLASS				Total	
		B		C			
		N	%	N	%	N	%
E/A RATIO	≤ 1	6	85.7	18	41.8	24	48.0
	> 1	1	14.3	25	58.2	26	52.0
Total		7	100.0	43	100.0	50	100.0
p-value =		0.0451 (Fisher Exact's)					

DISCUSSION

In the present study it was observed that at the time of presentation 34 (68.0%) cases of cirrhosis had prolonged QT-interval of >440ms. Amongst patients of CP Class B, 2 (28.6%) while 32 (74.4%) cases of Class C had prolonged QT-interval of >440ms. This study showed that QTc prolongation is a frequent finding in patients with liver disease. The prolongation of QTc interval correlated with the severity of liver damage. Bernadi et al[6]and Bal JS et al[7]reported long QTc intervals in

46% and 40% cirrhotic patients respectively in their studies. Although Bernardi et al[6] showed that patients with prolonged QT interval had lower survival rate than the normal counterparts; this finding was not reproduced by a more recent study by Bal JS et al[7]. In Venkateshuw arlu et al study 46% (23) had ecg changes, of which 20 cases had prolonged QTc interval.[8]

As far as echo cardiographic findings are concerned ,LAD (left atrial diameter) was above normal limits in all 50 patients of this study group with mean LAD of 40 mm. Wong et al[9] reported mean LAD of 40mm and Pozzi et al[10] reported mean LAD of 41mm which directly reflected on the LV mass. Venkateshuwarlu et al study with a mean 39.96±1.34[8]. However LAD was increased in all patients of each of these studies.

In my study LVEDD was found to be abnormal (>56mm) in 20(40%) out of 50 patients. All these patients belong to CPscore C (100%). In the study of Wong et al [9] reported abnormal LVEDD in 41 of there patients.

The LVEF (left ventricular ejection fraction) was normal (≥55%) in all patients of the study group with mean LVEF of 61.1%. Wong et al[8] reported mean LVEF of 62.7% and Pozzi et al[10] reported mean LVEF of 63.4%.

In the current study, it was observed that all the females had a Left ventricular mass above normal i.e. more than 150gms. Amongst males, most of the patients (19, 44.2%) had normal LV mass of below 200gms while only (24, 55.8%) had a LV mass above normal. Similar pattern of LV Mass abnormality was seen in male patients of CP class-B (5, 83.3% normal and 1, 16.7% higher) and CP class-C (14, 37.8% normal and 23, 62.2% higher).LV mass (left ventricular mass) was more than the normal limits (male >200gm and female >150gm) in 49% of the patients, with a mean value of 169.9±31.50 gms. Wong et al[9] reported mean LV mass of 171.4±20.04gm. It may also be due to myocardial adaptation to a chronically elevated blood volume. Venkateshuwarlu et al study LV mass was significantaly raised in 66% of the study population with a mean value of 169.30±26.04gms.[8]

In the present study it was observed that at the time of presentation 24 (48.0%) cases of cirrhosis had abnormal E/A Ratio ≤ 1 while remaining 26 (52.0%) cases had E/A Ratio > 1.Amongst patients of CP Class B, 6 (85.7%) and18 (41.8%) cases of Class C had diastolic dysfuctionl E/A Ratio ≤ 1 while remaining patients, 1 (14.3%) of class-B and 25 (58.2%) of class-C had E/A Ratio > 1, mean value of E/A ratio of 0.918 was seen in our study. Wong et al[9] reported mean E/A ratio of 0.9±0.1gm. E/A ratio.[11]

SUMMARY

- In my study of 50 patients 86% were male and 14% were female. In my study 7 cases were in class B 14% and class C 43 patient 86%. No case were seen in class A .
- the diadnosis of cirrhosis was done in this study by non invasive technique of usg abdomen and fibroscan of liver , coarse echotexture of liver and fibroscan valiue > 12.5kpa was taken as cirrhosis.
- In my study majority of patients hadcame under the CP class C.
- Increase in CP score criteria score is directly proportional to the increase in risk of cardiac abnormalities.
- LV mass was abnormal in 21 of the patients
- In our present study it was absorved that at the time of presentation 24 (48.0%) cases of cirrhosis had normal E/A ratio <= 1 while remaining 26 cases had E/A ratio >=1.0
- ECG changes of long QTc interval was seen in 34 of patients.
- In my study there was significant statistical correlation between CP score and QTc interval,(p-value 0.0274).
- In my study there was significant statistical correlation between CP class and LVEDD ,(p-value 0.0328).
- In my study there was significant statistical correlation between CP class and LV mass.(p-value 0.0374).
- In my studty there was significant statistical correlation between CP class and E/A ratio,(p-value 0.0451).

CONCLUSION

All patients of cirrhosis of liver irrespective of cause must be screened for cardiovascular structural and functional abnormalities with the help of ECG and COLOUR DROPLER ECHO cardiograph .These changes shown significant rise with the severity of disease as per CP score. Altered hemodynamic in cirrhotic patients, progressive in nature may be suspected as a case of cirrhotic cardiomyopathy. Awareness of cardiac abnormalities may be helpful in management approach of cirrhotic patients.

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