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Research Article

CARDIAC AND PULMONARY DYSFUNCTION IN CHRONIC LIVER DISEASE PATIENTS

Sumit Rungta[1]., Shweta Kumari[3]., Ajay Kumar[1]., Ghulam Akhtar[1] and Amar Deep[1,2]

[1]Department of Medical Gastroenterology, King George's Medical University, Lucknow, India
[2]Eeperimental and Public Health Lab, Department of Zoology, University of Lucknow, India
[3]Department of Biochemistry, King George's Medical University, Lucknow

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ABSTRACT

Patients with cirrhosis and portal hypertension (PHT) such as such as as cites, bleeding from esophagealvarices, and encephalopathy exhibit characteristic cardiovascular and pulmonary hemodynamic changes. Pulmonary abnormalities and symptoms are common in patients with chronic liver disease. If questioned, up to 70% of cirrhotic patients undergoing evaluation for liver transplantation complain of dyspnea. The diagnosis of liver cirrhosis was based on clinical, biochemical and ultrasound criteria. The aim of our study was to evaluate cardiac and pulmonary dysfunction in chronic liver disease.

INTRODUCTION

Chronic liver disease known to effect other system like cardiovascular and pulmonary system. Patients with chronic liver disease exhibit characteristic cardiac and pulmonary hemodynamic changes. Pulmonary abnormalities and symptoms are common in patients with chronic liver disease. If questioned, up to 70% of cirrhotic patients undergoing evaluation for liver transplantation complain of dyspnea[1].

Table 1 Causes of Pulmonary Abnormalities in Chronic Liver Disease

INTRINSIC CARDIOPULMONARY DISEASE
Chronic obstructive pulmonary disease
Congestive heart failure
Pneumonia
Asthma
SPECIFIC TO LIVER DISEASE
Associated with specific liver diseases
Panacinar emphysema: alpha-1 antitrypsin deficiency
Fibrosingalveolitits, pulmonary granulomas: primary biliary cirrhosis
Fluid retention complicating portal hypertension
Ascites
Hepatic hydrothorax
Pulmonary Vascular abnormalities
Hepatopulmonary syndrome
Portopulmonary hypertension

A variety of causes for pulmonary dysfunction in liver disease have been identified and include the table 1. A vasodilatory state and a hyperdynamic circulation affecting cardiac and pulmonary function, dominate the circulation.

Cardiac dysfunction characterised by systolic dysfunction, diastolic dysfunction and electromechnical abnormality, termed as cirrhotic cardiomyopathy. Cirrhotic cardiomyopathy is defined as subnormal ventricular response to stress in the face of high cardiac output.

Criteria of Cirrhotic Cardiomypathy

Cirrhotic cardiomyopathy is defined by chronic cardiac dysfunction typified by impaired cardiac contractility in response to stress and altered diastolic relaxation with electrophysiological abnormalities in cirrhotic patients with no known cardiac diseases [2-4]. A group of experts, at the 2005 World Congress of Gastroenterology at Montreal [Møller S, *et al*, 2008], proposed diagnostic and supportive criteria for cirrhotic cardiomyopathy as follows: (1) systolic dysfunction: blunted increase in cardiac output on exercise, volume challenge or pharmacological stimuli or resting ejection fraction <55 %, (2) diastolic dysfunction: the ratio of early to late (atrial) phases of ventricular filling or E/A ratio <1.0 (age-corrected), prolonged deceleration time (>200 ms), or

Corresponding author:* **Sumit Rungta
Department of Medical Gastroenterology, King George's Medical University, Lucknow, India

prolonged isovolumetric relaxation time (>80 ms), (3) 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 [2]. There is limited data regarding the prevalence of cirrhotic cardiomyopathy, it is because the disease usually remains silent with near normal cardiac function unless the patients are exposed to stress. It has been estimated that as many as 50 % of patients undergoing liver transplantation developed some signs of cardiac dysfunction [5] and about 7–21 % of patients died from heart failure in the post liver transplantation period [6].

Criteria of Hepatopulmonary Syndrome

1. Presence of chronic liver disease and portal hypertension.
2. Absence of intrinsic pulmonary disease-with normal chest X-ray, absence of pleural effusion, absence of intrinsic cardiac disease.
3. Pulmonary gas exchange abnormality ie. Arterial hypoxemia (PaO_2<80 mmHg) and alveolar arterial gradient (P_AO_2-PaO_2>20mmHg).
4. Intrapulmonary Vasodilation.

Pulmonary dysfunction involves diffusion abnormality with development of the hepatopulmonary syndrome and portopulmonary hypertension. Etiology of pulmonary dysfunction can be due to heavy smoking, chronic obstructive pulmonary disease, tense ascites, cardiac dysfunction, hepatopulmonary syndrome and portopulmonary hypertension. Patients in cirrhosis having compromised lung function with reduced diffusion capacity, ventilation and perfusion abnormalities and arterial hypoxemia.

MATERIAL AND METHODS

Total 66 patients with chronic liver disease and were enrolled in our study at the OPD of Department of medicine of Gandhi Memorial and associated Hospital, K.G.M.U. Lucknow. Clinically the patients having feature of CLD along with USG finding of coarse ecotexture of liver, dilated portal vein with and without splenomegaly were subjected to endoscopic examination. Those having oesophageal varices on endoscopy were included in the present study. We have done clinical evaluation, general examination and routine investigations of all patients. The data obtained and analysed by univariate analysis for refractory group and different subgroups of patients, responsive to diuretic. The data was reported as mean ± SD and student't' test and chi square test was used on appropriate to determine the significance of the data. The 'p' value <0.005 was taken as significant.

RESULTS

A total of 66 patients were enrolled in the present study on the basis of inclusion and exclusion criteria. The mean age of study group was 48.65 ± 13.74 years. The maximum number of patient in the study group were in the age group of 40-60 yrs (51.52%), With the 49 male (74.24%) and 17 female (25.76%). Patients are classified on the basis of symptoms. Ascites is most common symptoms present in 66.67 % of cases fallowed by jaundice (51.52 %), dysponia (53.03) and splenomegaly (33.33 %). Platyponia, orthodeoxia, spider angioma, clubbing and cyanosis were less common presentation. We excluded patients which are having tense ascites and active upper GI bleed during evaluation. Maximum no. of patients in our study having either alcoholism (30.30%) or hepatitis B (22.73 %) as their etiology. Etiology is not known in 34.85% of cases. Maximum no. of patients in our study having CTP class B (53.03%) and CTP class C (46.97%). Maximum number of patients in the study group had portal vein diameter between 12-16 mm (57.58%). Thus Maximum number of patients having portal hypertension on ultrasonography. On upper GI endoscopy maximum number of patient in our study had Grade II/III oesophageal varices (43.94%). The patients who were not had oesophageal varices were excluded from the study.

The main age of the study group was 48 ± 13.74 years. The maximum number of patient in the study group were in the age group of 40-60 yrs (51.52%). Out of 66 patients in the study group 49 patients (25.76 %) were female. So there is male predominance in the study. Patients are classified on the basis of symptoms. Ascites is most common symptoms present in 66.67 % of cases fallowed by jaundice (51.52 %), dysponia (53.03) and splenomegaly (33.33 %). Platyponia, orthodeoxia, spider angioma, clubbing and cyanosis were less common presentation. We excluded patients which are having tense ascites and active upper GI bleed during evaluation. Maximum no. of patients in our study having either alcoholism (30.30%) or hepatitis B (22.73 %) as their etiology. Etiology is not known in 34.85% of cases. In our study patients are usually of late age of disease. All patients enrolled in study were having either child pugh stage B or C. No patients in our study had child pugh stage A. Maximum number of patients in the study group had portal vein diameter between 12-16 mm (57.58%). Thus Maximum number of patients having portal hypertension on ultrasonography. On upper GI endoscopy maximum number of patient in our study had Grade II/III oesophageal varices (43.94%). The patients who were not had oesophageal varices were excluded from the study.

On UGIE maximum number of patients in our study had grade II/III oesophageal varies (43.94 %). The patients who were not had oesophageal varies were excluded from our study.Out of 66 patients 53 patients (80.30%) were found to have cardiac dysfunction. Out of 53 patients who were had cardiac dysfunction 45 patients (84.90%) had only cardiac dysfunction and 8 patients had both systolic and diastolic dysfunction and no patients had only systolic dysfunction. Out of 66 patients 38 patients (55.58%) having normal P_aO_2. Hypoxia (P_aO_2<80) was observed in 42.42% of the patients. Normal gradient were present in 39.39% of cases and increased gradient were observed in 60.61% of cases. Maximum number of patients with cardiac dysfunction were having alcoholism or unknown etiology and there is no significance difference between groups with or without cardiac dysfunction. Out of 35 patients with CTP stage 'B' 28 patients (80%) had cardiac dysfunction and out of 31 patients with CTP stage 'C' 25 patients (80.65%) had cardiac dysfunction. Comparison of both groups did not show significant different ('p'=1.000), so stage of liver disease did not have difference in prevalence of cardiac dysfunction in our study.

Table 2 Comparison of Means % of Saturation of O_2, P_aO_2 and $P_{(A-a)}O_2$ with Cardiac Dysfunction

Parmeter	Cardiac Dysfunction		P
	No	Yes	
% Saturation	92.98 ± 5.12	90.66 ± 8.30	0.419
P_aO_2	87.89 ± 17.87	83.80 ± 15.96	0.422
$P_{(A-a)}O_2$	23.83 ± 13.66	25.46 ± 20.19	0.783

In comparison patients with cardiac dysfunction had less value of saturation of oxygen in blood, $P_aO_2$and had Alveolar-arterial oxygen gradient but this was not statistically significant (p>0.05).

Table 3 Comparison of Means of Blood Pressure, Pulse rate and QTc Interval with Cardiac Dysfunction

Parameter	Cardiac Dysfunction		P
	No	Yes	
Systolic BP	122.92 ± 12.62	127.54 ± 9.12	0.679
Diastolic BP	78.60 ± 11.70	78.56 ± 9.50	0.987
Pulse Pressure	44.30 ± 6.62	48.98 ± 9.49	0.06
Mean BP	93.38 ± 11.61	94.89 ± 8.24	0.589
Pulse Rate	86.30 ± 13.21	85.62 ± 9.27	0.708
QTc	0.38 ± 0.053	0.39 ± 0.049	0.719

Comparison of means of systolic blood pressure, diastolic blood pressure and pulse rate in patients with or without cardiac dysfunction was not found significant. QTc interval comparison between patients with or without cardiac dysfunction was also found insignificant.

Table 4 Comparison of Means of Systolic Blood Pressure in different Child Pugh Stages

Child Pugh Group	n	Mean ± SD	P_{50}
B	35	128.40 ± 8.18	128
C	31	124.67 ± 11.49	124
Total	66	126.63 ± 9.97	128

Table 5 Comparison of Means of Diastolic Blood Pressure in different Child Pugh Stages

Child Pugh Group	n	Mean ± SD (mm/Hg)
B	35	81.14 ± 6.47
C	31	75.67 ± 11.97
Total	66	78.57 ± 9.87

'p'=0.020

Systolic blood pressure in CTP stage B patients was higher in comparison to patients in CTP stage C but this was not statistically significant and same in case of diastolic blood pressure but statistically significant (p=0.020).

Table 6 Comparison of Distribution of Symptom and signs with Hepatopulmonary Syndrome

	Hepatopulmonary Syndrome				Total		P
	No		Yes				
	n	%	n	%	n	%	
Ascites	39	88.64	5	11.36	44	66.67	0.655
Jaundice	30	80.24	4	11.76	34	51.52	0.673
Splenomegaly	19	86.36	3	13.64	22	33.33	0.392
History of UGIB	19	79.17	5	20.83	24	36.36	0.060
Orthodeoxia	1	16.67	5	83.33	6	9.09	0.000
Dyspnoea	30	85.71	5	14.29	35	53.03	0.202
Platypnoea	1	16.67	5	83.33	6	9.09	0.000
Spider Angioma	1	33.33	2	66.67	3	4.55	0.020
Clubbing	4	66.67	2	33.33	6	9.09	0.040
Cyanosis	0	0.00	3	100	3	4.55	0.002

It is seen that mean blood pressure in CTP stage B patients was higher in comparison to patients in CTP stage C and this was statistically significant (p=0.03). The mean pulse pressure in CTP stage B patients was nearly similar to patients in CTP stage C and it was also not statistically significant (p=0.341). It was found that pulse rate in CTP stage B patients was lower in comparison to patients in CTP stage C and this was statistically significant (p=0.018).

The commonest presenting symptoms in patients with hepatopulmonary syndrome upper GI bleed, ascites, platypnoea, orthodeoxia, dyspnoea. Orthodeoxia, platypnoea, spiderangioma and cyanosis was having statistically significant association with hepatopulmonary syndrome (p<0.05).

In our study there is no significant difference observed between biochemical andhematological parameters in patients with or without hepatopulmonary syndrome.

Table 7 Comparison of Mean of Saturation of O_2, P_aO_2 and $P_{(A-a)}O_2$ with Hepatopulmonary Syndrome

Parameter	Hepatopulmonary Syndrome		P
	No	Yes	
	Mean ± SD	Mean ± SD	
% Saturation	91.44 ± 7.08	87.86 ± 13.57	0.631
P_aO_2	86.62 ± 15.15	64.55 ± 14.49	0.001
$P_{(A-a)}O_2$	23.15 ± 18.19	45.09 ± 16.23	0.006

Patients with hepatopulmonary syndrome had lower mean value of saturation of oxygen in blood, $P_aO_2$and higher alveolar-arterial oxygen gradient in comparison to the patients without hepatopulmonary syndrome.

Table 8 Comparison of Hepatopulmonary Syndrome with Cardiac Dysfunction

Hepatopulmonary Syndrome	Hepatopulmonary Dysfunction			
	No		Yes	
	n	%	n	%
No	11	18.33	49	81.67
Yes	2	33.33	4	66.14
Total	13	19.70	53	80.30

'p'=0.337

Out of 6 patients with hepatopulmonary syndrome 4 patients (66.14%) had cardiac dysfunction and 2 patients (33.33%) did not have cardiac dysfunction. This was also not statistically significant.

CONCLUSION

The commonest presenting symptom in patients with cardiac dysfunction was ascites, jaundice, dyspnoea present in 79.55%, 67.65% and 80% cases respectively. Orthodeoxia, platypnoea. Spider angioma, clubbing and cyanosis were less common presentation. But all symptoms did not have any significant association with cardiac dysfunction. There was no significant difference observed in the biochemical and haematological parameters in patients with or without cardiac dysfunction.In our study there is no significant difference observed between biochemical andhematological parameters in patients with or without hepatopulmonary syndrome.

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