ORIGINAL ARTICLE

ECHO STUDY IN PATIENTS WITH CIRRHOSIS OF LIVER

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ABSTRACT

Objective: The objective of this study was to evaluate cardiac function with echocardiography in liver cirrhosis.

Methods: With purposive sampling 75 cases and 25 controls were taken in these studies who hadpresence of signs of free fluid on physical examination, and presence of signs of cirrhosis on USG abdomen in a tertiary care hospital during June 2006 to August 2007. All 100 patients were studied for the clinical profile, laboratory investigation, imaging study and 2D echocardiography.

Results: Mean pulse rate of the study group is 99/min where as 72.8/min for the control group. Mean blood pressure of the study group is 90.42mmHg where as 109.81mmHg for the control group. There is significantly higher QTc interval in cirrhosis cases as compared to control. The echocardiography results obtained in this study show significantly higher right & left atrial diameter in cirrhotic patients as compared to the control group.

Conclusion: In our study, no significantly difference is noted for ME, MA, ME/MA,DTC(mitral) between case and control group.(P>0.05)

Key Words: ECHO, Cirrhosis, Liver, cardiac function

INTRODUCTION

Liver Cirrhosis is one of the important health problems in India as well as in Western countries. Chronic alcoholism remains an important etiology for that. Cirrhosis is a chronic liver disease characterized by diffuse fibrosis and regenerating nodules following hepatocellular necrosis of liver. It is a disease with characteristic clinical findings and diagnosis with histopathology.

Heart disease can affect the liver with development of cardiac cirrhosis and liver disease can affect the heart with development of Cirrhotic Cardiomyopathy. Abelman in 1945 had first claimed that cardiac functions are impaired in cirrhosis and were the first to define Hyperdynamic circulation provided by increased cardiac output and heart rate, decreased systemic vascular resistance in patients with alcoholic cirrhosis.

Subsequent studies show that nitric oxide and other endothelial dependent factor causes peripheral vasodilatation. Similar cardiac contractile function disorders found in non-alcoholic cirrhosis and in animal model under stress condition have suggested that it is independent from alcohol intake and was named as cirrhosis cardiomyopathy (CMP).

METHODS

Study Design: This was a Cross sectional study conducted in a tertiary care hospital.

Sample Size:

Purposive sampling technique used with sample size of 75 cases and 25 controls in these studies who had presence of signs of free fluid on physical examination, and presence of signs of cirrhosis on USG abdomen at a tertiary care hospital during June 2006 to August 2007.

Data Collection methods:

The information collected using interview technique facilitated by the guidelines (questionnaire) prepare for asking questions. The information noted in the questionnaire form.

Data Management and Analysis: `After the completion of data collection, data entry was done into Excel data file.Data analysis was done by Epi_info version 6.04 software. All variables in the stud were qualitative , so student t test was used to calculate p value. 95% confidence interval was considered significant. ($p < 0.05$)

RESULTS

In present study 75 cases having cirrhosis and 25 control without cirhhosis were included in the study. FOllowing table shows comparision of case group with the control goups.

Table 1: Comparison of study variable sin case and control groups

	Case	Control	P value
Mean PULSE(min)	99	72.8	0.044
MEAN BP(mmhg)	90.43	109.81	0.049
Right Arterial Diameter	2.81	2.65	0.002
Left Arterial Diameter	3.63	3.41	0.030
Left diastolic diameter	3.98	4.21	0.064
Right diastolic diameter	3.303	3.1	0.061
TA(msn)	0.59	0.46	0.009
TE(msn)	0.59	0.52	0.036
TE/TA(msn)	1.048	1.42	0.001
DT(tricus)	181.49	177.36	0.043
Ejection Fraction (%)	63.12	63.62	0.868
Left ventricular Wall thickness	0.91	0.79	0.044
IVS(cm)	0.93	0.77	0.020
Pulmonary Arterial Pressure	32.6	19.6	0.020

DISCUSSION

The study group had significantly higher value (P-0.0438 < 0.05)Mean blood pressure of the study group is 90.42mmHg whereas 109.81mmHg for the control group.The study group had significantly lowerblood pressure value (p=0.0493<0.05).The systemic circulation in patient with cirrhosis is Hyperdynamic and characterized by increase heart rate and cardiac output (CO) & decreased systemic vascular resistance with low arterial blood pressure. Among the factors that may increase the pulse rate is increased sympathetic nervous activity, increased Blood volume (increased preload) and the presence of arteriovenous communications.[1-3]

The echocardiography results obtained in this study show **significantly higher** right & left atrial diameter in cirrhotic patients as compared to the control group. (P<0.05)This dilatation can be perceived as an adaptation of cardiac hemodynamics to changes in the peripheral circulation. [2, 4]

Left & right ventricular diastolic diameters were not **significantly** different in both cases and controls. (P>0.05)

The impairment was manifested as non-significant increase in the E wave velocity, amarked increase in the A wave velocity, a marked increase in deceleration time or E wave and marking reduction in E/A ratio.

A shift in the Doppler profile to a lower E wave and a higher atrial contribution to ventricular filling, along with an increased E/A ratio are considered the typical noninvasive patterns of diastolic dysfunction. This data indicate the left ventricular diastolic function is altered in cirrhosis. (P < 0.05) this alteration is more marked in the presence of ascitis. Presumably, because the increased intrathoracic pressure and bulging of diaphragm induced by intra-abdominal fluid collections interfere-with diastolic expansion of the ventricles. It is present however even in absence of ascitis suggesting that non mechanical factors are involved as well. We can speculate that one of these factors in an increase in cardiac diameters that leads to a reduced ability of the ventricular wall to distance further.

Patchy fibrosis and increased heart weight may affect the stiffness of the myocardial wall and result impaired left ventricular filling and diastolic dysfunction [4-6] expanded blood volume may increase in the cardiac preload with overloading and impaired cardiac contractility as outcome. (Graph 4)

In our study systolic function – EF is not significantly-different in both cases and control. (P>0.05) Both in the presence and in the absence of clinical signs and symptoms of the heart disease alcoholic cirrhosis has been associated with systolic dysfunction, a phenomenon that was absent in our patient, moreover alcoholic heart disease is characterized by a different echo graphic pattern that includes a mark reduction of systolic function.

In our study, we did not find difference of degree of impairment in ventricular function as far as systolic function is concerned in alcoholic and non-alcoholic cirrhotic patients.

Thus we observed that participation of alcohol to the cardiac derangement cannot exclude, but other factors must be involved. These factors can be endotoxin, [7]bile acids, [8] tumor necrosis factor – A[9] and catecholamines[10]which depress cardiac functions and are frequently elevated in advanced cirrhosis.

There is significantly increase in LVWT in cases as compared to control group. (P<0.05)The increase in-Left ventricular wall thickness (LVPCO + IVS) that was seen in our cirrhoticpatient was because of the cardiac hypertrophy and was accompanied by a deranged distensability of cardiac walls in a variety of pathological condition.[2, 11, 12]

The mechanisms that increase left ventricular wall thickness are not clarified by our study. However, we can speculate that the renin-angiotensin-aldosterone[13] are involved because angiotensin II, E, NE enhance cardiac tissue growth both in vitro & vivo.

Another important finding that drew attention is the significantly higher pulmonary arterial pressure in the case group. (P<0.05) In fact, pulmonary vascular resistance was tended to decrease in cirrhotic patient. [14-15]The mechanism of increased PAP is not fully understood, but previous studies suggested the increased level of vaso active substance in pulmonary circulation and probable toxic effect of this substance on endothelial cell. It has been suggested that microthrombi can migrate to pulmonary vascular bed along Porto systemic shunts and cause increased in vascular resistance.

CONCLUSION

In our study, no significantly difference is noted for ME, MA, ME/MA,DTC(mitral) between case and control group.(P>0.05)

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