ORIGINAL ARTICLE

EVALUATION OF CARDIAC FUNCTION IN PATIENTS WITH LIVER CIRRHOSIS: A HOSPITAL BASED CROSS-SECTIONAL STUDY

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ABSTRACT

Introduction: Cirrhosis is a pathologically defined as an entity associated with a spectrum of characteristic clinical manifestations like irreversible chronic injury of the hepatic parenchyma and include extensive fibrosis in association with the formation of regenerative nodules. Cardiac dysfunction in cirrhosis, a problem on the 'blind side of the heart', often remains ignored.

Methodology: This was ahospital based cross-sectional study, carried out under the department of medicine, SMIMER, Surat. All patients were informed about the study and their verbal consent was obtained. Patients fulfilling the inclusion criteria (n=50) were taken into study and all patients were studied for the clinical profile, laboratory investigation, imaging study like chest X-ray & Ultrasonography abdomen and 2D echocardiography. All patients were hospitalized and blood pressure and heart rate were measured.

Result: History of alcohol consumption was found in 36(72%) of the patients. Most common symptoms were ascitis, jaundice and malena**.** Mean total count was 7144(±1568). Mean blood urea level was 38.4(±19.78) while mean serum creatinin was 1.0(±0.72). Mean albumin level was 2.9 (±0.81). Mean serum billirubin total, direct and indirect was 6.1(±8.08), 4.35(±5.98), 1.8(±2.19) respectively. Diastolic dysfunction was present in about 66% (33 out of 50) of patients.

Conclusion: The patients of cirrhosis develop cirrhotic cardiomyopathy and this cirrhotic cardiomyopathy was not related to the etiology of liver cirrhosis**.**

Keywords: Liver Cirrhosis, cardiomyopathy, ascitis, cardiac function

INTRODUCTION

Cirrhosis is a pathologically defined as an entity associated with a spectrum of characteristic clinical manifestations like irreversible chronic injury of the hepatic parenchyma and include extensive fibrosis in association with the formation of regenerative nodules. These features result from number of factors like hepatocyte necrosis, collapse of the supporting reticulin network with subsequent connective tissue deposition, distortion of the vascular bed, and nodular regeneration of remaining liver parenchyma.[1]

Cardiac dysfunction in cirrhosis, a problem on the 'blind side of the heart', often remains ignored. However, cirrhosis is associated with a host of cardiovascular abnormalities including hyper dynamic circulation, portal hypertension, hepato-pulmonary syndrome and changes in several different vascular territories such as the renal and cerebral vasculature.[2]

Peripheral vasodilatation by reducing cardiac "after load" may prevent any clinical evidence of cardiac dysfunction; however, many studies have demonstrated that under physiological or pharmacological stress the ventricular systolic function may give an inadequate response.[2,3]

Cirrhosis is associated with an increased cardiac output and heart rate, as well as decreased systemic peripheral vascular resistance and blood pressure. Splancnic arterial vasodilatation and impaired autonomic activity play a role.[4]

Cirrhotic cardiomyopathy is a pathological condition defined as "a 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 known cardiac disease".[5]

In cirrhosis, cardiac output increases while systemic vascular resistance and arterial pressure decreases.[6,7]

Overall there seems to be a link between progression of liver function impairment, the development of portal hypertension and the degree of hyper dynamic circulation which is the hallmark of the deranged cardiovascular function in advanced liver disease.

Some cirrhotic patients have obvious features of cardiomyopathy. However most have a normal or supernormal ejection fraction as judged by echocardiography. Thus physicians often assume that cardiac function is often normal in these patients. However further investigations have uncovered multiple problems in cardiac performance that place these patients at risk of heart failure. Early cardiac decompensation is often missed because the cardiac workload is reduced by peripheral vasodilatation caused by liver failure.[8]

Thus cirrhotic patients are dismissed as having normal cardiac function. However when these patients are subjected to physiological or pharmacological stress they are prone to develop clinical signs of suboptimal perfusion including renal failure and acidosis. Most patients with liver disease have subtle defects in myocardial function that are not apparent on cursory examination and these defects become apparent only when these patients are exposed to stress.

METHODOLOGY

This was ahospital based cross-sectional study, carried out under the department of medicine, SMIMER, Surat, Gujarat, a tertiary health care center of South Gujarat from June 2012 to August 2013.

All patients with diagnosis of cirrhosis of liver on USG abdomen were included in study except cirrhosis case with, Age<18 years, patient with primary cardiac pathology, patientswith diseasesaffecting the heart like hyperthyroidism, thyrotoxicosis, patients not willing were excluded from the study.

All patients were informed about the study and their verbal consent was obtained. Patients fulfilling the inclusion criteria (n=50) were taken into study and all patients were studied for the clinical profile, laboratory investigation, imaging study like chest X-ray& Ultrasonography abdomen and 2D echocardiography. All patients were hospitalized and blood pressure and heart rate were measured.

Simultaneously serum was obtained and tested for albumin, total billirubin, direct billirubin and indirect billirubin, SGOT, SGPT, ALP, Blood Urea, serum electrolytes, RBS. Viral marker for hepatitis B was performed by one step rapid qualitative immunoassay test which can detect hepatitis B surface antigen; viral marker for hepatitis c was performed by rapid visual spot test for test for qualitative detection of antibody to HCV in human serum or plasma named. The diagnosis of alcoholic liver cirrhosis was based on H/O significant alcohol intake. Evidence of portal hypertension was diagnosed clinically by spleenomegaly and presence of USG findings. Abnormal liver function test in cirrhosis were studied. Other hepatitis markers, autoimmune serological test and metabolic studies were not performed due to unavailability of these tests at our institute. Cirrhosis was classified as cryptogenic when HbsAg and Anti HCV antibody were negative and there was no history of significant alcohol intake.

M-mode and detailed 2-D echocardiography were carried out in all patients. Patients were examined in supine position as well as lying partially on the left side at an angle of $30/45^0$.Doppler echocardiography was carried out in all the patients to assess the left ventriculardiastolic function under simultaneous 2-D echocardiography visualization of cardiacanatomy in apical four chamber view, a Doppler sample value was positioned within inflowarea of ventricle just below the mitral valve annulus(near the mitral valve tips and parallel topresumed axis of blood flow) to minimize the potential effect of transducer angulations theDoppler sampling volume was aligned in different planes until maximum diastolic, flowvelocities were recorded or till optimal spectral pattern was obtained.

RESULT

Out of 50 participants 38 were male and 12 were female. Most of patients (78%) are fall in age group of 20 to 50 years. History of alcohol consumption was found in 36(72%) of the patients.

Table 1: Clinical profile of the Patients (n=50)

Clinical profile	Frequency (%)
Symptoms	
Jaundice	39 (78)
Ascitis	49 (98)
Dyspnea	1 (2)
Encephalopathy	2 (4)
Malena	7 (14)
Sign	
Pallor	1 (2)
Icterus	33 (66)
Clubbing	0 (00)
Pedal edema	21 (42)

According to table 1, most common symptoms were ascitis, jaundice and malena**.** Out of 50, 33(66%) were presented with sign of icterus and 21(42%) with pedal edema.The mean pulse rate in study was 100.54(±9.59)/min while mean blood pressure was 90.35(±3.53)mm/hg.The mean QTc interval in this study was 0.41 sec.

Table 2: Laboratory profile of the patients (n=50)

Investigation	Mean±SD
Hemoglobin	9.2 ± 0.665
Total count	7144 ± 1568
Blood urea	38.4 ± 19.78
Serum creatinin	1.0 ± 0.72
Serum albumin	2.9 ± 0.81
Serum billirubin (T)	6.1 ± 8.08
Serum billirubin (D)	4.35 ± 5.98
Serum billirubin (I)	1.8 ± 2.19

According to table 2, the mean value of hemoglobin was 9.2 (±0.665) gm. Mean total count was 7144(±1568). Mean blood urea level was 38.4(±19.78) while mean serum creatinin was 1.0(±0.72). Mean albumin level was 2.9 (±0.81). Mean serum billirubin total, direct and indirect was 6.1(±8.08), 4.35(±5.98), 1.8(±2.19) respectively.

Table 3:Echocardiography finding of patients (n=50)

Echocardiographic Parameters	Mean (±SD)
IVS(cm)	095(0.232)
LVDD(cm)	4.07(0.591)
LVPW(cm)	0.97(0.321)
EF(%)	64.16(10.495)
LAD(cm)	3.58(0.455)
RAD(cm)	2.81(0.131)
PAP(mmhg)	29.18(7.258)
TE(m/sec)	0.58(0.141)
TA(m/sec)	0.57(0.166)
TE/TA	1.11(0.415)
DT(Tricuspid)msec.	183.48(7.404)
RVDD(cm)	3.30(0.564)

According to table 3, there are significant higher values in RA and LA diameter, marked decrease in TE/TA ratio.Also there is increase in LVPW and IVS dimensions. There was higher PAP in our patients. ME/MA <1 in echocardiography was present in 17 out of 50 patients and ME/MA >1 present in 33 out of 50 patients. Diastolic dysfunction was present in about 66% (33 out of 50) of patients.

DISCUSSION

The male to female ratio in the study was 3.16:1. Most of patients (78%) are fall in age group of 20 to 50 years. History of alcohol consumption was found in 36(72%) of the patients. This may be contributing factor for high male prevalence of liver disease. The most common etiology of cirrhosis was alcohol induced liver disease.

The most common clinical sign of liver failure was jaundice (n = 50). While on abdominal examination, the most common finding was presence of distended abdomen and shifting dullness (n = 50).

The mean QTc interval in this study was 0.41 sec.,which is on higher level as in previous similar studies compare to normal individuals. Q-T interval is frequently prolonged in cirrhosis; regardless the etiology of the disease worsens in parallel with the severity of the disease, and may have an important prognostic significance. The permeability of the plasma membrane and the function of its ion channels have been shown it be impaired in cirrhosis. An altered control of vascular tone by K+ &Ca++ channels in various cells in human cirrhosis. As result of these changes, there is electrophysiological abnormality in cardiac excitation & prolongation of QTc interval.Further studies shows that QTc interval is not influenced by the etiology of cirrhosis & related with Child Pugh score & liver tests. Patients with QTc longer than 0.44sec (440ms) had significantly lower survival rate than those with normal QTc.

When we compare outcome of our study with previous similar studies, we find almost similar result[9]. There are significantly higher values in RA and LA diameter, marked decrease in TE/TAratio. Also there is increase in LVPW and IVS dimensions.There was higher PAP in our patients. In our patients, ME/MA <1 in echocardiography was present in 17 out of 50 patients and ME/MA >1 present in 33 out of 50 patients. So diastolic dysfunction was present in about 66% (33 out of 50) of patients.

In our study there is no difference noted as compared to previous study in LVDD, RVDD, EF%, ME, MA, ME/MA, DT(mitral).

In our study,this dilatation of both atria can be perceived as an adaptation of cardiac hemodynamic to changes in the peripheral circulation.[10, 11]

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 Ewave 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. 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.

These factors can be endotoxin[12], bileacids[13], tumor necrosis factor[14]and catecholamine[15] which depress cardiacfunctions and are frequently elevated in advanced cirrhosis.

CONCLUSION

The most common etiology of cirrhosis was alcohol induced liver disease. The patients of cirrhosis develop cirrhotic cardiomyopathy and this cirrhotic cardiomyopathy was not related to the etiology of liver cirrhosis**.** There was no significantdifference indegree of impairment in ventricular function as far as systolic function is concerned in alcoholic and non-alcoholic cirrhotic patients, so other factors must be involved. Diastolic dysfunction was found in 33(66%) of the patient.

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