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Original Article

A Comparative Study to Assess the Cardiovascular Complications in Patients of Liver Cirrhosis

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Abstract

Introduction: Cirrhosis is associated with a wide spectrum of characteristic clinical manifestations consisting of clinical and hemodynamic alterations. Involvement of the cardiovascular system is crucial during the course of cirrhosis. The most common cardiac abnormality that occurs among cirrhotic patients is the left ventricular diastolic dysfunction. Apart from increasing morbidity, it may also contribute to significant post-transplantation mortality. Therefore, this study was conducted to assess the cardiovascular complications of liver cirrhosis.

Materials and Methods: This case–control study was conducted on 100 ultrasound confirmed cases of cirrhosis of liver among males aged 20–60 years. A total of 40 controls were included in the study. Detailed histories were recorded. Physical examination was done. Echocardiographic findings assessing the systolic and diastolic functions were recorded and compared.

Results: The cirrhotics had the increased E/A ratio and isovolumic relaxation time and decreased deceleration time. The parameters relating to systolic function were similar in the cases and controls.

Conclusion: It can be effectively concluded from the present study that significant diastolic dysfunction is present in cirrhotics.

Keywords: Cardiovascular complications, Cirrhosis, Deceleration time, Diastolic dysfunction, E/A ratio

INTRODUCTION

Cirrhosis is associated with a wide spectrum of characteristic clinical manifestations consisting of clinical and hemodynamic alterations. Clinical features of cirrhosis derive from the morphological changes and often reflect the severity of hepatic damage rather than the etiology of the underlying liver disease. Progressive structural and functional impairment of the liver inevitably involve other organ systems such as central nervous system in the form of hepatic encephalopathy, kidneys in the form of hepato-renal syndrome, the cardiovascular system (CVS) in the form of cirrhotic cardiomyopathy, and respiratory system such as porto-pulmonary hypertension.[1]

Loss of functioning hepatocellular mass may lead to jaundice, edema, coagulopathies and a variety of metabolic abnormalities: Fibrosis and distorted vasculature leads to portal hypertension and its sequelae, including gastro-esophageal varices and splenomegaly. Involvement of the CVS is crucial during the course of cirrhosis due to its pathophysiological, clinical, and therapeutic relationships with the liver.

In 1953, Kowalski and Abelmann first documented that cirrhosis is associated with a hyperdynamic circulatory syndrome, characterized by an increase in cardiac output and a decrease in peripheral vascular resistance.[2] Since then, it has been realized that cardiovascular alterations are frequently observed in cirrhosis. Cirrhosis may result in subclinical latent cardiomyopathy with hyperdynamic circulation characterized by increased cardiac output and decreased peripheral resistance and volume overload state. It was previously reported that chronic alcoholism may have an effect on heart.[3] However, the exact pathogenetic mechanisms of these hemodynamic alterations remain uncertain.

The most common cardiac abnormality that occurs among cirrhotic patients is the left ventricular diastolic dysfunction (LVDD) related to the development of myocardial fibrosis,

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hypertrophy, and subendothelial edema.[4,5] Diastolic dysfunction occurs when the passive elastic traits of the myocardium are reduced due to the increased myocardial mass and changes in the extracellular collagen.[6] According to different studies, the prevalence of LVDD in cirrhotic patients ranges from 25.7% to as high as 81.4%.[7] Evidence suggests that patients with cirrhosis display primarily LVDD with normal systolic function at rest.[8] Diastolic dysfunction may progress to systolic dysfunction, although this has not been directly shown in cirrhotic patients.[9,10]

Apart from increasing the morbidity in the cirrhotic patients, cardiac failure has also emerged as an important cause of mortality after liver transplantation and accounts for 7–21% of deaths in post-orthotopic liver transplantation period.[11] Diastolic dysfunction has been proved to be an early marker of cardiac dysfunction occurring before systolic dysfunction at rest.[12] A stable cardiac status is important before the performance of interventional procedures or liver transplantation. It has been suggested that this cardiac dysfunction may be reversible after liver transplantation.[13]

Thus, cardiovascular complications are an important cause of morbidity in the patients with liver cirrhosis. However, studies in this regard, particularly in the Indian population, are scarce. Therefore, this study was conducted to assess the prevalence of cardiovascular diseases in patients with liver cirrhosis.

MATERIALS AND METHODS

This case–control study was conducted after the approval of Institutional Ethics Committee. This study was conducted on 100 cases of cirrhosis of liver (ultrasound confirmed) among males aged 20–60 years, was included in the study. A total of 40 age and sex matched controls were enrolled in the study. Cases and controls having comorbidities of systemic arterial hypertension, primary cardiac or pulmonary disease, hepatic encephalopathy, gross ascites, viral hepatitis, anemia, diabetes mellitus, thyroid dysfunction, collagen vascular diseases, and malignancies were excluded from the study. Patients having septicemia and patients not consenting to participate in the study were also excluded.

A written informed consent was obtained from each patient. Demographic details and relevant past and personal history were recorded. Physical examination was done. Color Doppler Echocardiogram (using Mindray M7 Echo machine) was done to assess the cardiac involvement. The echocardiographic findings were categorized and recorded as follows:

Systolic Function Indices

- Left ventricular internal dimension in systole (LVIDs)
- Left ventricular internal dimension in diastole (LVIDd)
- Interventricular septal diameter in diastole (IVSd)
- Left ventricular posterior wall thickness in diastole (LVPWd)
- Ejection fraction (EF)%.

Diastolic Function Indices

- Isovolumic relaxation time (IVRT)
- Mitral E deceleration time (DT)
- Mitral E and A velocity.

All the data were recorded and compared.

Statistical Analysis

The data were analyzed using the SPSS. The results were expressed as Mean ± SD. Quantitative data were analyzed by unpaired *t*-test. $P < 0.05$ was considered to be statistically significant.

RESULTS

The mean age of the patients with cirrhosis was 44.70 ± 8.65 years and of the controls was 46.02 ± 8.89 years. The difference was statistically insignificant with $P = 0.418$.

Table 1 shows the distribution of the systolic function indices among the cirrhotic cases and controls. The difference between the two groups was statistically insignificant ($P < 0.05$).

Table 2 shows the distribution of the diastolic function indices among the cirrhotic cases and controls. The E/A ratio and IVRT were significantly decreased in the cirrhotics ($P < 0.001$) and the DT was significantly increased ($P < 0.001$).

DISCUSSION

Cirrhosis of liver affects many organs and systems; hence, it may be considered as a systemic disease.[14] Cardiovascular changes have been realized to be a frequent

Table 1: Distribution of the systolic function indices among the cirrhotic cases and controls

Parameter	Controls	Cirrhotics	*P*-value	Significance
LVIDs (cm)	3.20±0.48	3.14±0.15	0.285	Not significant
LVIDd (cm)	4.79±0.50	4.71±0.35	0.282	Not significant
EF (%)	67.66±8.67	66.28±3.43	0.179	Not significant
IVSd (cm)	0.86±0.19	0.89±0.09	0.338	Not significant
LVPWd (cm)	0.88±0.26	0.85±0.15	0.357	Not significant

LVIDs: Left ventricular internal dimension in systole; LVIDd: Left ventricular internal dimension in diastole; IVSd: Interventricular septal diameter in diastole; LVPWd: Left ventricular posterior wall thickness in diastole; EF: Ejection fraction

Table 2: Distribution of the diastolic function indices among the cirrhotic cases and controls

Parameter	Controls	Cirrhotics	*P*-value	Significance
E/A	1.39±0.48	1.13±0.15	<0.001	Significant
DT	147.15±41.71	203.53±4.01	<0.001	Significant
IVRT	99.86±26.87	79.60±14.49	<0.001	Significant

IVRT: Isovolumic relaxation time; DT: Deceleration time

complication in the cirrhotics. LVDD can be seen as an early manifestation of many cardiovascular diseases since any change in myocardial structure may affect the function of the left ventricle causing an abnormal filling pattern. The cardiovascular changes may be attributed to the hyperdynamic circulation, characterized by increased cardiac output, and decreased peripheral vascular resistance and arterial pressure. These cardiovascular abnormalities have been suggested to induce or aggravate several complications of cirrhosis such as renal salt and water retention, variceal bleeding, hepatopulmonary syndrome, and increased cardiovascular fragility under stress.[15]

Thus, the knowledge of CVS involvement in a cirrhotic patient is important in planning the treatment and assessing the prognosis. However, very few studies have been conducted in this regard.

In the present study, the cardiovascular complications of cirrhosis of liver were assessed. Hundred patients diagnosed with liver cirrhosis were included in the study. A total of 40 healthy controls were included in the study. The mean age of the cirrhotics and the controls was almost similar ($P = 0.418$).

Systolic Dysfunction

In the present study, it was observed that there were no difference in the cirrhotics and the controls, in terms of the mean values of LVIDs, LVIDd, EF, IVSd, and LVPWd ($P < 0.05$). Thus, there was no significant systolic dysfunction in the cirrhotics and the controls.

In the study by Somani *et al.*,[16] they found no difference in the systolic function in the cirrhotics and the controls. Similar were the findings in the study by Sampaio *et al.*[17] and Dadhich *et al.*[18]

Thus, it can be effectively concluded that there is no systolic dysfunction in the cirrhotics.

Diastolic Dysfunction

In the present study, significant diastolic dysfunction was observed in the cirrhotics as compared to the controls as indicated by the decreased E/A ratio ($P < 0.001$) and IVRT ($P < 0.001$) and increased DT ($P < 0.001$).

In the study by Somani *et al.*,[16] they studied the cardiovascular complications of cirrhosis and included a total of 60 cases and 30 controls. They observed that the DT was significantly prolonged in cirrhotics compared to the controls ($P = 0.02$).

Similar were the findings of significantly decreased E/A ratio and increased DT in cirrhotics compared to controls in the studies by Sampaio *et al.*[17] and Dadhich *et al.*[18]

Diastolic dysfunction in cirrhosis was first reported in 1997.[19] The pathophysiological background of the diastolic dysfunction in cirrhosis is an increased stiffness of the myocardial wall, most likely because of a combination of mild myocardial hypertrophy, fibrosis, and subendothelial edema.[4] With Doppler echocardiography, Finucci *et al.*[20] found impaired left ventricular relaxation, decreased E/A ratio, and delayed early diastolic transmittal filling in patients with cirrhosis compared with controls.

In the study by Karagiannakis *et al.*,[21] they investigated 45 cirrhotic patients. They observed no difference in the left ventricular EF%. They concluded that diastolic dysfunction precedes systolic dysfunction in the cirrhotic patients. During the 2-year follow-up, they found that the presence of LVDD was a poor prognostic factor for the survival of the cirrhotic patients. They found that cirrhotic patients who died had lower E/A ratio and high DT.

Thus, diastolic dysfunction, in addition to being an early marker for cardiac involvement, may also be a prognostic factor for the long-term survival in the cirrhotics.

Limitations

The study was limited by the outpatient department attendance of the patients. Therefore, the results may not be generalized.

CONCLUSION

It can be effectively concluded from the present study that significant diastolic dysfunction is present in cirrhotics, as indicated by the impaired E/A ratio, DT, and IVRT. However, further studies need to be conducted to ascertain the role of diastolic dysfunction in the prognosis and survival. Furthermore, there was no systolic dysfunction in the present study. Therefore, diastolic dysfunction may indicate an early stage of cardiac involvement. Further studies need to be conducted to evaluate the patterns of cardiac involvement in cirrhotics and its role in the long-term prognosis.

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