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Original Article

Diastolic dysfunction characterizes cirrhotic cardiomyopathy

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ABSTRACT

Aim: Present study aims to study the occurrence of cirrhotic cardiomyopathy and its correlation to hepatorenal syndrome by assessing the cardiac status in patients with cirrhosis of liver and healthy controls.
Methods: Thirty alcoholic cirrhotic, thirty non-alcoholic cirrhotic and thirty controls were enrolled for the study. Cardiac parameters were assessed by color doppler echocardiography. Patients were followed up for twelve months period for development of hepatorenal syndrome.
Results: Mild diastolic dysfunction was present in 18 cirrhotic patients (30%): grade I in fifteen patients and grade II in three. Diastolic dysfunction was unrelated to age; sex and etiology of cirrhosis. Among all the echocardiographic parameters, only deceleration time was found to be statistically significant. Echocardiographic parameters in systolic and diastolic function were not different in compensated vs decompensated patients in different Child-Pugh classes or cirrhosis aetiologies.

At one year follow-up, no significant differences were found in survival between patients with or without diastolic dysfunction. Hepatorenal syndrome developed in only two patients and its correlation with diastolic dysfunction was not statistically significant.
Conclusions: Present study shows that although diastolic dysfunction is a frequent event in cirrhosis, it is usually of mild degree and does not correlate with severity of liver dysfunction. There are no significant differences in echocardiographic parameters between alcoholic and non-alcoholic cirrhosis. HRS is not correlated to diastolic dysfunction in cirrhotic patients. There is no difference in survival at one year between patients with or without diastolic dysfunction. Diastolic dysfunction in cirrhosis is unrelated to circulatory dysfunction, ascites and HRS.

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1. Introduction

The cardiovascular complications of cirrhosis have not received the attention they deserve. More than 50 years ago, increased cardiac output in alcohol-related cirrhosis was attributed to either impaired thiamine utilization or the presence of an endogenous vasodilator.[1] Later, it became clear that cirrhotic cardiac dysfunction is predominantly governed by peripheral vasodilatation. There is now substantial evidence that a hyperdynamic syndrome is due to impaired liver function and portal hypertension with splanchnic vasodilatation.[2] Subsequent studies described an impaired hemodynamic response to physiologic (exercise) and pharmacologic stress despite a high resting cardiac output. For a long time, these changes were thought to be the effect of alcohol and not cirrhosis. Animal models of alcoholic cirrhosis confirmed these findings. They were found to be related to decreased myocardial contractile function and were corroborated by additional human studies.[3]

Experimental and clinical studies have shown impaired myocardial contractility and electrophysiological abnormalities in cirrhosis, leading to a clinical entity called "cirrhotic cardiomyopathy".[4,5] This term denotes a chronic cardiac dysfunction. Blunted contractile responsiveness to stress and altered diastolic relaxation with electrophysiological abnormalities, such as prolongation of the QT interval occur in the absence of any other cardiac disease.[6,7] This may affect the prognosis of the patients and aggravate the course during invasive procedures such as surgery, insertion of transjugular intrahepatic portosystemic shunts, and liver transplantation.[8,9]

Cardiac failure has emerged as an important cause of mortality after liver transplantation and accounts for 7–21% of deaths in post orthotopic liver transplantation period.[10] Diastolic dysfunction has been proved to be an early marker of cardiac dysfunction occurring before systolic dysfunction at rest.[11] Also, there are some studies showing correlation between cirrhotic cardiomyopathy and development of hepatorenal syndrome (HRS).[12,13]

Left ventricular diastolic dysfunction is the cardiac abnormality most frequently investigated in cirrhosis. It is present in 30–50% of patients.[14] Diastolic function in majority of studies has been assessed by measuring mitral inflow by Doppler echocardiography, with few studies using the more modern Tissue Doppler Imaging (TDI) technique.[14,15]

There is paucity of data from South-east Asia and the Indian subcontinent on cirrhotic cardiomyopathy. Apart from a study on cardiac dysfunction in cirrhotic and non-cirrhotic portal hypertension[16] and another study from Mumbai[17] there is hardly any information on the status of cardiac abnormalities in Asian patients with cirrhosis.

We undertook this study with the primary objective to study cardiac status in patients with cirrhosis of liver in comparison to healthy controls to assess the occurrence of cirrhotic cardiomyopathy and its correlation to HRS. The secondary objective was to assess the correlation of echocardiographic parameters of cardiac dysfunction with the severity of liver dysfunction, and to assess whether or not there are significant differences in these parameters between patients with alcoholic and non-alcoholic cirrhosis. The prevalence of diastolic dysfunction and its relation to liver failure and prognosis were also investigated.

2. Patients and methods

Cirrhotic patients, either admitted in hospital or followed as out-patients, were considered for the study. Diagnosis of cirrhosis was established through histology and/or a combination of clinical, biochemical and imaging findings.

Patients were excluded from the study if they had evidence of cardiovascular disease, respiratory disease, renal disease or any other major systemic disease. Other exclusion criteria were: (1) cardiac arrhythmias or pulse rate > 100/min; (2) recent bleeding (<3 months); (3) Hemoglobin < 9 gm/dl; (4) serum creatinine > 1.5 mg/dl; (5) patients with hypertension, diabetes; (6) patients receiving drugs like spironolactone, beta blockers, nitrates, anti-hypertensive agents, and sympathomimetics at present or anytime in the past; (7) patients suffering from cirrhosis of mixed etiology (to observe any difference of diastolic dysfunction between alcoholic and non-alcoholic group and to avoid possible confounding factor) and (8) Age <18 and >75 years.

Finally, we included sixty cirrhotics. A third group, age and sex matched thirty healthy subjects on normal diet, served as controls.

Approval of the study protocol was obtained from the institutional ethical committee. Informed consent was obtained from all participants. Patients were followed up for a year.

2.1. *Clinical and biochemical evaluation*

Detailed clinical history was recorded. Patients underwent clinical examination and blood investigations. Liver function was quantified by Child-Pugh and MELD scores.

2.2. *Echocardiography*

Echocardiography was performed by an experienced operator in accordance with the recommendations of the American Society of Echocardiography.[18] From a long axis parasternal view, the left ventricular (LV) systolic and diastolic septal wall thickness (SWST and SWDT), posterior wall thickness (PWTs and PWTd) and the LV diameter (LVESD and LVEDD) were measured in M-mode. The LV mass and LV geometry were calculated accordingly. LV volumes and LV Ejection Fraction (LVEF) were estimated using Simpson's modified biplane method. An LVEF above 50% was considered normal. Pulsed Doppler examination of the LV inflow was performed with the sample volume placed between the mitral leaflet tips. The following parameters were recorded and measured: peak early (E wave) and atrial (A wave) flow velocities, their ratio E/A, and the E wave deceleration time. TDI was obtained from the four chamber apical view and tissue velocity was calculated. The myocardial peak systolic velocity (S′) was measured in lateral mitral annulus to define systolic function. Tissue velocities were also measured in the lateral mitral annulus