International Journal of Research in Medical Sciences
Prashant S. Sidmal et al. Int J Res Med Sci. 2015 Sep;3(9):2316-2322
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20150623

Research Article

Pattern of diastolic dysfunction in alcoholic and non-alcoholic cirrhotic portal hypertensive patients with or without ascites in rural population in South India

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Received: 15 July 2015
Revised: 17 July 2015
Accepted: 10 August 2015

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ABSTRACT

Background: Abnormalities in cardiac function have been reported in liver cirrhosis, suggesting a latent cardiomyopathy in these patients. In this study, we evaluated the association between severity of diastolic dysfunction and severity of cirrhosis in alcoholic and non-alcoholic cirrhotic patients with or without ascites in rural population in South India.
Methods: This cross-sectional study was conducted on alcoholic and non-alcoholic cirrhotic patients coming from rural background admitted in a tertiary hospital in South India from January 2014 to March 2015. 60 patients were enrolled. Severity of cirrhosis was evaluated by Child-Pugh score. A 12-lead surface ECG and echocardiographic studies were performed.
Results: Seventy eight percent of patients were male. The mean age of patients was 58.98 ± 15.2 years. 21%, 37%, and 42% of patients were considered as child class A, B, and C, respectively. There was a significant relation between diastolic dysfunction and disease duration (P=0.001), age >60 years (P=0.004), and severity of cirrhosis (P=0.003).
Conclusions: Because of high prevalence of diastolic dysfunction in cirrhotic patients and risk of decompensation following invasive procedures, it could be suggested that all patients would be screened routinely by echocardiography before invasive procedures. According to the relation between Child-Pugh score and diastolic dysfunction, it is recommended for cardiac assessment, especially echocardiographic evaluation in all cirrhotic patients. In conclusion, left ventricular diastolic dysfunction is commonly associated with advancement of hepatic dysfunction while systolic function is maintained till advanced hepatic failure.

Keywords: Diastolic dysfunction, Alcoholic cirrhosis, Non-alcoholic, Portal hypertension

INTRODUCTION

Cirrhosis is a hepatic disease that presents in individuals aged 50-60 years, typically.[1,2] Patients with liver cirrhosis are reported to have a hyperdynamic circulation, which manifests as high cardiac output, decreased systemic vascular resistance, and widespread arterial vasodilatation, primarily.[3,4] Based on many previous studies traditionally, cirrhosis is associated with cardiovascular abnormalities.[5,6]

Cardiomyopathy is derived from the Greek roots: cardia (heart), mys (muscle) pátheia/páthesis (disease), that is, it is a condition affecting the heart muscles.

Cirrhotic cardiomyopathy is the term used to describe a collection of characters expressive of abnormal heart structure and function in patients with cirrhosis.[7,8] The term "cirrhotic cardiomyopathy" is generally defined by the following clinical criteria: (1) baseline increased cardiac output but blunted ventricular response to stimuli, (2) systolic and/or diastolic dysfunction, (3) absence of overt left ventricular failure at rest, and (4) electrophysiological abnormalities including prolonged QT interval on electrocardiography and chronotropic incompetence.[9-11] Many patients with cirrhosis exhibit various degrees of diastolic dysfunction. Diastolic relaxation is impaired in cirrhosis. In the majority of cases, diastolic dysfunction precedes systolic dysfunction. Cardiac response to physical exercise in cirrhotic patients is blunted, with subnormal responses in echocardiographic ejection fraction and contraction time.

Diastolic filling consists of two parts normally: rapid, early diastolic (active) relaxation and late diastolic (passive) filling. The first phase depends on the rate of ventricular relaxation, elastic ventricular recoil, the atrio-ventricular pressure gradient, and the passive elastic features of the left atrium and ventricle.[12,13] The second phase formed on the basis of the strength of left atrial contraction and the stiffness of the left ventricle. Diastolic dysfunction occurs when the passive elastic traits of the myocardium are reduced to increased myocardial mass and changes in the extracellular collagen secondarily.[14] This leads to stiffening and hypertrophy of the left ventricle with decreased compliance and higher diastolic pressures at each diastolic volume. So, relatively small increases in intravascular volume can lead to elevations in diastolic pressures. Shifting this pressure into the left atrium and pulmonary venous system can lead to pulmonary edema.[14-17]

Ventricular diastolic compliance and diastolic function can be assessed by measuring the velocity of blood flow from the left atrium to the left ventricle during early diastole (the E wave) and late diastole (the A wave) and calculating the E/A ratio by using the Doppler echocardiography. In other words, determinants of a diastolic dysfunction on a Doppler echocardiogram are decreased E/A ratio - the ratio of early to late (atrial) phases of ventricular filling and delayed early diastolic transmitral filling with prolonged deceleration and isovolumetric relaxation times.[18]

Many studies indicated that some level of diastolic dysfunction exists in most patients with cirrhosis.[18-23] Diastolic dysfunction may progress to systolic dysfunction, although this has not been directly shown in cirrhotic patients.[13,16,23]

Diastolic dysfunction can be graded as follows according to the diastolic filling pattern (Figure 1).

Grade 1 = impaired relaxation pattern with normal filling pressure
Grade 2 = pseudonormalized pattern
Grade 3 = reversible restrictive pattern
Grade 4 = irreversible restrictive pattern

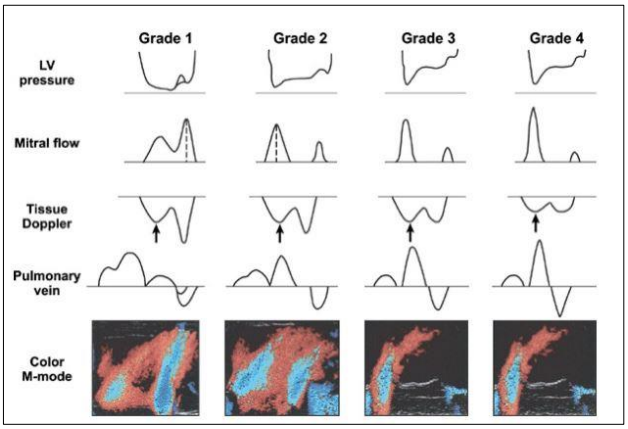

Figure 1: Grading of diastolic dysfunction.

Abnormal left ventricular (LV) filling patterns (grades 1 to 4). Grading of diastolic dysfunction and filling pattern based on mitral inflow, mitral annulus velocity, pulmonary vein velocity, and color M-mode of mitral inflow. Arrow, reduced early diastolic velocity (E') of mitral annulus in all stages of diastolic dysfunction (Courtesy Echo Manual, The, 3rd Edition, Oh, Jae K.; Seward, James B.; Tajik, A. Jamil)

METHODS

A total of sixty subjects, coming from rural background admitted in a tertiary care hospital in South India, were enrolled in this cross sectional case control study. A1coholic & nonalcoholic cirrhotic patients (with or without ascites) who were admitted from January 2014 to March 2015 were enrolled. Severity of the cirrhosis was evaluated by Child-Pugh criteria and divided in three groups: A (mild), B (moderate), and C (severe). This study project was approved by the ethics committee. A written informed consent was obtained from each patient. Subjects were counseled and explained about the objectives of the study by a qualified medical doctor. Detailed personal history was taken using a standard questionnaire.

Inclusion criteria comprised: 1) cirrhotic patients with or without ascites, diagnosis of cirrhosis was based on histopathological evidence (liver biopsy) or unequivocal clinical grounds (chronic liver disease stigmata, jaundice, ascites, esophageal varices), impaired liver function tests and ultrasonographic features consistent with cirrhosis (diffuse alteration and nodular transformation of liver parenchyma, and signs of portal hypertension).

Exclusion criteria comprised: 1) history or clinical evidence of cardiovascular disease; 2) major lung disease; 3) diabetes mellitus; 4) terminal liver failure; 5) major arrhythmias; 6) severe anemia (Hb <7 gm/dL); 7)

hepatic encephalopathy; 8) renal failure (serum creatinine >1.5 mg/dL); 9) history or sphygmomanometer evidence of hypertension according to JNC 7 criteria.[13]

Electrocardiography: A 12-lead surface EGG was obtained from all subjects in the supine position. ECG was recorded at a paper speed of 50 mm/s.

Echocardiography: Diastolic function of the heart was assessed and recorded. Echocardiographic studies were performed using a HDI 3000 (Philips ATL, Bothell, WA, USA) equipped with 2 to 4 MHz probes allowing M-mode, colour Doppler, two dimensional, and pulsed Doppler measurements. Echocardiography was performed according to the guidelines of American Society of Echocardiography.

Mitral inflow patterns: The normal E/A ratio is between 1 and 2.

Grade 1 diastolic dysfunction (Impaired myocardial relaxation)**:** The E/A ratio is <1, with a prolonged deceleration time (Dct) (>240 ms). In the tissue Doppler assessment, e' is also reduced with a resultant E/e' ratio (medial) <8, suggesting a normal LA pressure. The D wave of the pulmonary venous inflow is smaller than the S wave and the AR wave is normal.

Grade 2 diastolic dysfunction (Pseudonormalized pattern): When diastolic LV function deteriorates, LV compliance progressively decreases and there is an increase of LA pressure and the diastolic filling pressure. The transmitral E wave velocity progressively increases and the Dct decreases. As it does so, it goes through a phase that resembles a normal filling pattern. The E/A ratio is between 1 and 2 and the Dct between 160 and 240 ms. This pseudo-normal pattern is a transition pattern from impaired relaxation to restrictive filling and is a result of a moderately increased LA pressure superimposed on a relaxation abnormality. The following clues help distinguish this from a normal filling pattern E/e' ratio (medial) >15. Pulmonary venous flow AR >25 cm/sec and longer than transmitral A wave.

Grade 3 and 4 diastolic dysfunction (restrictive pattern): With more severe diastolic dysfunction, LV compliance reduces and LA pressures rise. The low compliance of the LV causes a rapid increase in the early LV pressure and a shortened inflow and DT. The E/A ratio is > 2. Dct is <160 ms. The high LA pressure manifests as an E/e' ratio >15 at the medial annulus. Forward diastolic pulmonary vein flow stops in mid-late diastole and during atrial contraction there is a significant flow reversal resulting in a prolonged AR. A reversal to grade 1 or 2 on reducing the preload by performing Valsalva manouvre or administering nitroglycerine suggests reversibility of the cardiac restriction and is termed grade 3. Diastolic filling should be graded as irreversible (grade 4) in the absence of such a reversal.

Statistical analysis: SPSS for Windows, version 16, was used for data analysis. The qualitative data were analyzed by chi-square, Fisher's exact test and the Student's t test for continuous variables. Continuous variables are presented as mean ± Standard Deviation (SD); categorical variables are presented as percentages. P value <0.05 was considered significant.

RESULTS

Socio-demographic

In total, mean age of patients were 53.6 (range: 35-68) years. Majority of patients were males (n=42, 70%).

Clinical and laboratory findings

Table 1 shows age, gender, and etiology of liver disease and child classification in our patients. Table 2 shows the range and mean values of some laboratory results of study patients.

Table 1: Baseline data of study patients.

Characteristics	
Age (mean ± SD)	53 ± 12
Sex (M/F)	42/18
Cause	
Cryptogenic	7
HBV	9
HCV	10
NAFLD	12
Alcoholic	22
Child Pugh classification	
Child A (5-6)	7
Child B (7-9)	23
Child C (10-15)	30

Table 2: Mean and standard deviation of some laboratory results in study patients.

Variables	
AST (U/dl)	
Mean ± SD	40.9 ± 8
Range	33-59
ALT (U/dl)	
Mean ± SD	58.6 ± 23.3
Range	19-103
Albumin (g/dl)	
Mean ± SD	2.75 ± 0.33
Range	22-3.6
Bilirubin (mg/dl)	
Mean ±SD	2.45 ± 1.41
Range	1-6
INR	
Mean ± SD	1.79 ± 0.44
Range	1.2-3.3

The most common cause of cirrhosis in study population as mentioned in Table 1 was alcoholic cirrhosis. The mean duration of diagnosis was 3.13 ± 4.02 years. There was a significant relation between diagnosis-duration and diastolic dysfunction.

Five percent of patients had normal diastolic function and 95% had diastolic dysfunction. There was an insignificant relation between gender and diastolic dysfunction and it was more frequent in male patients (Table 3).

Table 3: Distribution of diastolic function by gender.

Gender	Diastolic function					
	Normal function		Mild diastolic dysfunction		Moderate / severe dysfunction	
	No.	%	No.	%	No.	%
Male	2	3.3	14	23.3	26	43.3
Female	3	5	5	8.3	10	16.6
Total	5	8.3	19	31.6	36	

There was a significant relation between diastolic dysfunction and age (P=0.045). The age group of >60 years was the most frequent group of diastolic dysfunction. All cirrhotic patients in this group had some degree of diastolic dysfunction (Table 4). The relation between severity of cirrhosis and diastolic dysfunction was significant in this study (P=0.048) (Table 5).

Table 4: Distribution of diastolic dysfunction in various age groups.

Age group	Diastolic function					
	Normal function		Mild diastolic dysfunction		Moderate / severe dysfunction	
	No.	%	No.	%	No.	%
<40	3	5	3	5	6	10
40-60	2	3.3	7	11.6	12	20
>60	0	0	9	15	18	30
Total	5		19		36	

Table 5: Distribution of diastolic function between child groups.

Child class	Diastolic function					
	Normal function		Mild diastolic dysfunction		Moderate / severe dysfunction	
	No.	%	No.	%	No.	%
Child A	3	5	1	1.6	3	5
Child B	1	1.6	8	13.3	14	23.3
Child C	1	1.6	10	16.6	19	31.6
Total	5		19		36	

Table 6 shows the distribution of diastolic dysfunction severity between various causes of cirrhosis. Frequency of diastolic dysfunction in alcoholic group is more frequent than the other.

Table 6: Distribution of diastolic function between various causes of cirrhosis.

Causes	Diastolic function					
	Normal function		Mild diastolic dysfunction		Moderate / severe dysfunction	
	No.	%	No.	%	No.	%
Cryptogenic	1	1.6	2	3.3	4	6.6
HBV	1	1.6	3	5	5	8.3
HBC	1	1.6	2	3.3	7	11.6
NAFLD	0	0	4	6.6	8	13.3
Alcoholic	2	3.3	8	13.3	12	20
Total	5		19		36	

Overall LVDD was diagnosed in 91.6% of cirrhotic patients. Nineteen patients had mild (impaired relaxation) LVDD, while 36 had moderate/severe LVDD.

DISCUSSION

Diastolic dysfunction is a complex process that arises from numerous interrelated contributing factors such as pressure variations in the ventricle, cardiac preload and afterload, and ventricular relaxation and compliance.[24] The increased circulating blood volume, found in patient with cirrhosis, leads to a high cardiac preload and decreased peripheral vascular resistance with low cardiac afterload. The alteration in the diastolic function is likely due to an impaired ventricular relaxation. Diastolic dysfunction could be due to the stiffness of the ventricular wall as a result of cardiac hypertrophy described in cirrhotic cardiomyopathy.[7,25]

In this study, diastolic dysfunction in men was more than women. On the contrary, in the study of Redfield et al. they showed that heart failure with normal ejection fraction in any ages is more frequent in women rather than men.[26] In our study, diastolic dysfunction was increased significantly by increasing duration of disease (P=0.001). It was also seen in the study of Nasr et al.[27] So, they showed that diastolic dysfunction could be due to ascites or liver dysfunction. In the presence of ascites, intrathoracic pressure increased and diaphragm shifted up. This could prevent enough relaxation of ventricle.[27]

In our study, age group of >60 years had more diastolic dysfunction (P=0.045). The same results can be seen in other studies like El-Adi et al. that concluded cardiac changes is age related in cirrhotic patients in comparison with control group and showed that incidence of diastolic dysfunction was increased by age.[28] Increasing age can affect cirrhosis and can exacerbate cardiac dysfunction.

Severity of cirrhosis and diastolic dysfunction had a significant correlation (P=0.048). By increasing severity

of cirrhosis from Child A to Child C, diastolic dysfunction was seemed to worsen. In another study, by using Child-Pugh score and MELD score, moderate diastolic dysfunction and more severe cirrhosis are correlated significantly.[29] Achecar and Gonzalec-Tallon showed that diastolic dysfunction of left ventricle was exacerbated by liver disease.[30] The results of that study was similar to our study.

Prevalence of diastolic dysfunction in our study were 91.6% (31.6% mild, 60% moderate/severe). One study showed that 80%, 25%, and 24% of diastolic dysfunction were seen in patients who had severe, moderate, and mild liver fibrosis, respectively.[27] Another study showed that left ventricular diastolic dysfunction can be seen in 50% (25% grade I and 25% grade II) of cirrhotic patients.[30] In cirrhotic patients, patchy fibrosis may increase heart weight and also heart stiffness that affect ventricular filling and result in diastolic dysfunction.[31] The systemic circulation in patients with cirrhosis is hyperdynamic with an increased cardiac output and heart rate and a reduced systemic vascular resistance as the most pronounced alterations.[31] Expanding plasma volume increases cardiac preload and ultimately cardiac output in the presence of impaired cardiac contractility. Latent systolic and diastolic dysfunction with reduced work capacity is present and becomes in some patients manifested if the heart is challenged.[32] After liver transplantation both physical activity and cardiac function seem to improve.[33]

When the duration of cirrhosis was increased, liver cell failure was aggravated and cirrhosis becomes decompensated. In decompensated cirrhosis, there may be a further decrease in the arterial blood pressure owing to the unloading of baroreceptors and renal salt-water excretion is prolonged and incomplete.[34] Cardiac abnormalities such as heart stiffness due to fibrosis and circulatory problems may contribute to worsening of cardiac function in late stage of cirrhosis.

Diastolic dysfunction appears to be more prevalent in cirrhotic patients, indeed some authorities contend that some degree of diastolic dysfunction is present in virtually every patients with cirrhosis.[35,36] In most of the studies performed in the recent past, diagnosis of LVDD was based on E/A ratio <1 using 2-D Doppler echocardiography. Valeriano et al. also found a similar lower mean E/A ratio in both left and right ventricle in ascitic subgroup than in non-ascitic subgroup.[37] Pozzi et al. showed that removal of ascitic fluid by rapid total paracentesis reduced the A wave velocity and increased the E/A ratio to the values similar to those of cirrhotic patients without ascites, but still abnormal as compared to healthy controls.[38]

However, E/A ratio have several limitations as it is strongly dependent on preload and often requires age correction.[39] A recent study by Ruiz del Arbol et al. showed LVDD in 37/80 (46.2%) with Tissue Doppler Imaging (TDI) in cirrhotic patients. They also found LVDD occurs simultaneously with other changes in cardiac structure and function and is associated with an impairment of effective arterial blood volume. LVDD was a sensitive marker of advanced cirrhosis, type 1 hepatorenal syndrome development, and mortality.[40] Parameters regarding left ventricular systolic performance were within normal range in our study. Further studies are required to assess the prognostic impact of left ventricular diastolic dysfunction in patients with cirrhosis.

Limitations

Further studies with larger samples are recommended. Long term follow-up after discharge is needed to access the improvement of diastolic dysfunction with improvement in clinical status.

CONCLUSIONS

Cardiac dysfunction is a common complication of advanced cirrhosis that can make a variety of disturbances, including diastolic dysfunction. Duration of disease, increased age, and severity of cirrhosis can increase the severity of diastolic dysfunction. Because of high prevalence of diastolic dysfunction in cirrhotic patients and risk of decompensation following invasive procedures, it could be suggested that all patients would be screened routinely by echocardiography before invasive procedures.

According to the relation between Child-Pugh score and diastolic dysfunction, it is recommended for cardiac assessment, especially echocardiographic evaluation in all cirrhotic patients. In conclusion, left ventricular diastolic dysfunction is commonly associated with advancement of hepatic dysfunction while systolic function is maintained till advanced hepatic failure.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

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Cite this article as: Prashant S. Sidmal, Prashanthkumar BG, Shekarappa KC. Pattern of diastolic dysfunction in alcoholic and non-alcoholic cirrhotic portal hypertensive patients with or without ascites in rural population in South India. Int J Res Med Sci 2015;3:2316-22.