www.c-jhs.com

ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

Calabar Journal of Health Sciences

Original Article

Diastolic versus systolic dysfunction in patients with chronic liver disease – A single center study in South-South, Nigeria

Uchenna Njideofor[1], Uchenna Okonkwo[1], Victor O Ansa[1], Clement O Odigwe[1]

[1]Department of Internal Medicine, University of Calabar, Calabar, Cross River, Nigeria.

***Corresponding author:**
Uchenna Okonkwo,
Department of Internal Medicine, University of Calabar, Calabar, Cross River, Nigeria.

ucsuizes@yahoo.co.uk

Received : 25 January 2021
Accepted : 20 May 2021
Published : 30 June 2021

DOI
10.25259/CJHS_5_2021

Quick Response Code:

ABSTRACT

Objectives: The term cirrhotic cardiomyopathy (CCM) has been used to describe the constellation of cardiovascular abnormalities including diastolic and systolic dysfunctions in patients with chronic liver disease (CLD). CCM contributes to morbidity and mortality associated with CLD. The aim of the study was to evaluate the left atrial and ventricular geometry, systolic and diastolic functions in patients with CLD.

Material and Methods: This was a cross-sectional analytical study that involved 80 patients with CLD seen at University of Calabar Teaching Hospital, Calabar, Nigeria, and 80 apparently healthy controls matched for age/gender. The participants were interviewed, examined and had resting transthoracic echocardiography. The data were analyzed using IBM SPSS version 20.0.

Results: A total of 160 subjects were recruited into the study with a male to female ratio of 2.8:1. There was no difference in the mean age of cases and controls (P = 0.115). Systolic function of the left ventricle was similar in the two arms. However, left ventricular diastolic dysfunction, left atrial enlargement, and increased left ventricular mass index (LVMI) were more prevalent among the patients with CLD compared to controls ($P < 0.05$).

Conclusion: The study demonstrated increased left atrial diameter, increased LVMI associated with diastolic dysfunction, and preserved systolic function at rest among CLD patients.

Keywords: Chronic liver disease, Diastolic dysfunction, Systolic dysfunction

INTRODUCTION

It has been recognized over the years that acute and chronic diseases of the heart and blood vessels can cause alteration in the structure and functions of the liver and can result in liver cirrhosis.[1] This condition has been termed cardiac cirrhosis or cardiac liver. More recently, chronic liver diseases (CLD) have also been noted to cause alterations in cardiac structure and function.[2]

CLD notably liver cirrhosis results in increasing resistance to portal venous outflow into the hepatic veins due to the fixed obstruction from the distortion of the hepatic micro-architecture and the variable obstruction from increased intrahepatic vasoconstriction due to increased elaboration of endothelin-1 and activation of the stellate cells.[3] The portal hypertension (PoHTN) that develops is associated with splanchnic vasodilatation caused by increased elaboration

of vasoactive agents including nitric oxide (NO), carbon monoxide (CO), endothelium derived hyperpolarizing factor, prostacyclin (PGI_2), adrenomedullin, glucagon, and hydrogen sulfide (H_2S).[3] The splanchnic vasodilatation results in a relative reduction in effective circulating blood volume which is sensed by the baroreceptor system and results in activation of the renin-angiotensin-aldosterone-sympathetic system (RAAS) and increased secretion of the antidiuretic hormone and subsequently the development of hyperdynamic circulation.[4] These neurohormonal substances also have deleterious effects on the heart and vasculature resulting in myocyte hypertrophy, myocyte apoptosis, alteration in the cardiac extracellular matrix, β-adrenoceptor dysfunction, and electrophysiologic abnormalities.[5] These cardiac alterations mark the development of cirrhotic cardiomyopathy (CCM). The earliest recognition of CCM was in patients with alcoholic liver disease who had increased cardiac output at rest.[6] CCM in patients with alcoholic liver disease is clinically and pathophysiologically distinct from alcoholic cardiomyopathy.[5] CCM has now been described in CLD of various etiologies.[7,8]

CLD is an important cause of morbidity and mortality particularly in regions where HBV is endemic.[9] Globally, it is ranked the 5th most common cause of early loss of life.[10] CCM contributes to poor effort tolerance, development of hepatorenal syndrome and can result in cardiovascular collapse following large volume paracentesis and during infection in CLD patients.[4] It may even lead to early mortality even after successful liver transplant.[11] There are very few studies from Africa on the impact of CLD on cardiac geometry and function, hence, this study which evaluated the left atrial and ventricular geometry, diastolic and systolic functions in CLD is intended to provide knowledge on the prevalence of this relatively obscure complication of CLD.

MATERIAL AND METHODS

This study was carried out on adult patients (18 years and above) with a diagnosis of CLD comprising chronic viral hepatitis, liver cirrhosis, and hepatocellular carcinoma seen at the Gastroenterology clinic, University of Calabar Teaching Hospital (UCTH), Calabar, Cross River State, Nigeria over a 10 months period from June 2018 to April 2019. The study was a cross-sectional analytical study. Diagnosis of CLD was based on clinical features, hepatitis B and C serology, abdominal ultrasound scan, triphasic CT scan, and histology where appropriate. Age- and sex-matched healthy controls were recruited from members of staff and relatives of patients with no symptoms or signs of CLD. The severity of the hepatic impairment in CLD was graded using MELD score calculated using the formula: MELD = 3.78 × ln (serum bilirubin [mg/dL]) + 11.2 × ln (INR) + 9.57 × ln (serum creatinine [mg/dL]) + 6.43. The patients were classified into three categories; Stage 1: MELD score ≤ 9, Stage 2: MELD score 10–20, and Stage 3: MELD score > 20. Controls were screened for CLD using abdominal ultrasound scan, Hepatitis B and C serology. Subjects with hypertension, diabetes mellitus, chronic kidney disease, and retroviral disease were excluded from the study. The weight was measured to the nearest 0.1 kg with a weighing scale with the patient wearing minimal clothing and without shoes. The height was measured with a stadiometer to the nearest 0.5 cm. The height was obtained without shoes or head gear with the patient standing erect with the feet together. The height was measured at the level of the vertex of the skull. The body surface area (BSA) was calculated using the Mosteller formula.[12]

$$BSA = \frac{\sqrt{weight\ (kg) \times height\ (cm)}}{3600}$$

The BSA was used in the determination of cardiac index (CI) and left ventricular mass index (LVMI). CI was calculated by dividing the cardiac output by the BSA and LVMI was calculated by dividing the left ventricular mass (LVM) by the BSA. LVM was also indexed to height.

All recruited individuals were evaluated using transthoracic two-dimensional echocardiography as well as Doppler and M-mode imaging with the use of commercially available Chison echocardiography machine model – LCD 1502 with D3P641 3.5 MHz transducer. Measurements were taken using standard guideline recommendations of the American Society of Echocardiography.[13] Parameters that were documented included left atrial diameter (LAD), interventricular septal thickness (IVST), left ventricular posterior wall thickness, left ventricular internal dimension (LVID), and LVM. Left ventricular systolic function was assessed by determination of the left ventricular ejection fraction (LVEF). LVEF was derived using the modified Simpson's method by tracing the endocardial borders of the left ventricle in apical 4-chamber and 2-chamber views at end-diastole and end-systole to determine end-diastolic volume (EDV) and end-systolic volume (ESV). LVEF = (EDV−ESV)/EDV. LVSD was considered to be present when the resting LVEF was < 55%. Fractional shortening (FS) was obtained using M-mode imaging at the mid-cavity of the left ventricle in parasternal long axis view to determine the LVID in diastole (LVIDd) and LVID in systole (LVIDs). FS = (LVIDd−LVIDs)/LVIDd. FS was considered abnormal if <25%. Left ventricular cardiac output was measured from the LV forward stroke volume multiplied by the heart rate. LV forward stroke volume was determined from the LV outflow tract (LVOT) area multiplied by the LVOT time velocity integral. CI was calculated by dividing the cardiac output by the BSA. LVM was obtained using the cubed formula by subtracting the volume of the left ventricular cavity from the volume enclosed by the epicardium to derive the volume of the left ventricular myocardium. The

myocardial volume was converted to mass by multiplying it by the myocardial density (1.04 g/ml). LVM = 0.8 × (1.04 [LVIDd + PWTd + IVSTd]^3 – LVIDd^3) + 0.6 g

Left ventricular diastolic function was assessed using pulsed wave Doppler study of transmitral inflow velocities to determine the early (E) and late (A) velocity ratio as well as the measurement of the deceleration time of the E wave. LV diastolic function was graded using the E/A ratio as well as the peak mitral E velocity.[13]

Sample size determination

This was calculated. Using Cochran's[14] formula and a prevalence rate of CLD of 4.2%[15], we arrived at a minimum sample size of 80 after accounting for attrition rate of 10%. This was applied to the study and control groups.

Ethical approval

Ethical approval was obtained from the Health Research Ethics Committee (HREC) of the UCTH, Calabar HREC approval number-UCTH/HREC/33/596.

Table 1: Comparison of age/anthropometric characteristics of cases and controls (*n*=160).

	Study group		*t*-test	*P*-value
	Cases (Mean±SD)	Controls (Mean±SD)		
Age (years)	42.0±12.6	40.8±9.9	1.264	0.208
Weight (kg)	66.0±12.7	71.4±11.9	2.791	0.006*
Height (m)	1.65±0.8	1.67±0.8	1.420	0.158
BSA (m^2)	1.73±0.2	1.8±0.2	2.827	0.005*
BMI (kg/m^2)	23.2±4.4	25.6±3.8	2.193	0.030*

BSA: Body surface area

RESULTS

A total of 160 individuals participated in the study out of which 80 were those with CLD while the remaining 80 were their age and gender matched healthy controls. The male-to-female ratio was 2.8:1. The mean age of the study population was similar to that of the control 42.0 ± 12.6 versus 40.8 ± 9.9 years. The leading etiologic agent for the CLD was HBV which accounted for about 73.75%, 16.25% had HCV, 8.75% had significant alcohol use, and 1.25% had HBV/HCV coinfection. Patients with CLD had lower BMI and BSA compared to the control group ($P < 0.05$) [Table 1].

The prevalence of LAD among the CLD patients was 36.8% compared to 11.3% in the controls. The mean LAD was significantly higher among CLD patients versus the controls ($P < 0.001$). CLD patients had a higher proportion of those with different grades of LAD with mild dilatation (26.6% vs. 7.5%), moderate dilatation (8.9% vs. 3.8%), and severe dilatation (1.3% vs. 0.0%). The prevalence of systolic dysfunction in CLD patients was 10% versus 7.5% in the control group. The difference was not statistically significant ($P = 0.576$). The prevalence of left ventricular diastolic dysfunction (40.0% vs. 13.8%) was higher among CLD patients compared with their controls. The difference was statistically significant ($P < 0.001$). The diastolic dysfunction found in both arms was Grade 1 "Table 2."

The mean IVST in diastole (IVSTd) was significantly higher in CLD patients compared to controls (1.20 ± 0.27 vs. 1.05 ± 0.21; $P < 0.001$). The mean posterior wall thickness in diastole (PWTd) was also higher among the CLD patients but it was not statistically significant ($P = 0.180$). Mean LV mass was not significantly different in the two arms; however, when LV mass was indexed to BSA and to height; the derived LVMI were significantly higher in patients with

Table 2: Comparison of LV systolic/diastolic dysfunction and left atrial diameter among cases and controls (*n*=160).

Variable	Cases *n*=80 *n* (%)	Controls *n*=80 *n* (%)	Total *n*=160 *n* (%)	Chi-square test	*P*-value
LV ejection fraction					
Normal LV systolic function (≥55%)	72 (80.0)	74 (92.5)	146 (91.3)	0.313	0.576
LV systolic dysfunction (<55%)	8 (10.0)	6 (7.5)	14 (8.8)		
LV diastolic dysfunction					
Mean E/A±SD	1.12±0.49	1.26±0.35		2.109	0.36
None	48 (60.0)	69 (86.2)	117 (73.1)		0.001*
Grade 1	32 (40.0)	11 (13.8)	43 (26.9)	14.025	
Left atrial diameter					
Normal	51 (63.8)	71 (88.8	122 (76.3)	FET,14.582	0.001*
Mild dilatation	21 (26.2)	6 (7.5)	27 (16.9)		
Moderate dilatation	7 (8.7)	3 (3.8)	10 (6.2)		
Severe dilatation	1 (1.3)	0 (0.0)	1 (0.6)		
Mean left atrial diameter±SD (cm)	3.8±0.5	3.5±0.4	3.6±0.5	*t*-test, 4.306	0.001*

SD: Standard deviation; *Statistically significant, FET: Fischer exact test, LV: Left ventricular

CLD compared to controls (P = 0.002). The mean LV stroke volume and estimated LV cardiac output were similar in the two arms but LV CI was significantly higher in the patients with CLD (P = 0.018). Aortic root and left ventricular outflow dimensions were similar in the two arms of the study. Similarly, the mean LVEF and FS were not significantly different between the cases and the controls Table 3.

Table 4 shows comparison of mitral valve E velocity deceleration time between cases and controls. Prolongation of deceleration time was found to be significantly more prevalent among those with CLD compared to their healthy controls, and the difference was statistically significant (P = 0.004).

The mean deceleration time was higher in cases than in controls (188.0 ± 51.6 ms vs. 160.2 ± 30.1 ms), and the difference was significant statistically (P < 0.001). Although mean LAD, LVMI, deceleration time, and prevalence of diastolic dysfunction increased with higher MELD categories, the difference did not reach statistical significance (P > 0.05), respectively, Table 5.

Table 3: Comparison of echocardiographic parameters of the left ventricle among cases and controls (n=160).

Variable	Study group				
	Cases (Mean±SD)	Controls (Mean±SD)	Total	*t*-test	*P*-value
PWTd (cm)	1.11±0.25	1.06±0.27		1.347	0.180
IVSTd (cm)	1.20±0.27	1.05±0.21		3.852	0.001*
LVOT (cm)	2.05±0.25	2.04±0.21		0.154	0.878
LVOT	19.41±4.82	19.26±3.21		0.233	0.816
TVI (cm)	63.8±19.2	63.3±14.5		0.205	0.838
Stroke volume (ml)					
AOD (cm)	3.17±0.4	3.10±0.4		1.083	0.281
LVIDd (cm)	4.60±0.69	4.63±0.52		2.282	0.763
LV cardiac output (L/min)	5.2±1.8	4.8±1.2		1.571	0.118
LV cardiac index (L/min/m^2)	3.0±1.0	2.6±0.70		2.400	0.018*
LVM (g)	197.7±63.1	176.8±52.2		2.282	0.24
LVMI (g/m^2)	114.5±36.9	97.7±29.1		3.210	0.002*
LVMI ($g/m^{2.7}$)	51.36±17	44.7±14.5		2.648	0.009*
LVFS (%)	38.4±7.6	39.1±7.1		0.557	0.578
EF (%)	67.6±9.6	68.3±8.3		0.526	0.599

SD: Standard deviation, *Statistically significant, AOD: Aortic root diameter, PWTd: Posterior wall thickness in diastole, IVSTd: Interventricular septal thickness in diastole, LVOT: Left Ventricular outflow tract, TVI: Time velocity integral, AOD: Aortic root diameter, LVIDd: Left ventricular internal diameter in diastole, LVM: Left ventricular mass, LVMI: Left ventricular mass index, LVFS: Left Ventricular fractional shortening, EF: Ejection fraction

DISCUSSION

The study showed that patients with CLD are mostly young males with the lower BMI and BSA than their healthy counterparts. The mean age of the CLD patients was 42.0 ± 12.6 and is similar to findings from other studies in Nigeria and West Africa.[16,17] In regions, where HBV is endemic, CLD affects mainly young and middle aged persons in the prime of their productive life because of vertical and childhood transmission of HBV in these regions.[17] The low weight of the CLD patients also resulted in reduction in the derived parameters of BSA and BMI when compared to the controls. Similar findings of reduced weight, BSA and BMI in CLD patients compared to healthy controls had also been reported by other authors.[7,18] Possible reasons for the weight loss in CLD include hyper-metabolism, reduced appetite, early satiety, malabsorption, endotoxinaemia, PoHTN, and porto-systemic shunting.[2-4]

The LAD was significantly higher among the CLD patients and it is similar to what had been reported by other authors.[7,18] A study in China had reported prevalence of the left atrial enlargement of 55% in patients with liver cirrhosis and also reported that the left atrial enlargement was the most common cardiac chamber enlargement in CLD.[19] Dadhich *et al*.[20] had similar findings of increased LAD in patients with liver cirrhosis compared to healthy controls which did not differ significantly between pre-ascitic cirrhosis compared to ascitic cirrhosis patients. The LAD in CLD patients is a reflection of the diastolic function of the left ventricle since there was no patient with mitral valve disease. As observed in this study, LAD had similar distribution with the left ventricular diastolic dysfunction.

The mean LVM was higher in the CLD patients and the difference became statistically when the LVM was indexed to the BSA and to the height. The mean IVSTd was significantly higher in the CLD patients. The IVS being a wall shared by both ventricles may reflect changes in either of the ventricles; thus, the increased IVST might have resulted from the hyperdynamic circulation and the pulmonary vascular changes of CLD. The LV PWTd in some CLD patients showed some degree of flattening in diastole as a result of compression by the diaphragm which had been pushed upward by hepatomegaly and ascites but the difference with the control group was not statistically significant. Similar findings have been reported in other studies.[18-20]

Table 4: Comparison of mitral E deceleration time between cases and controls.

Variable	Cases *n*=80 *n* (%)	Controls *n*=80 *n* (%)	Total *n*=160 *n* (%)	Chi-square test	*P*-value
Deceleration time					
Normal (≤200 ms)	55 (68.8)	70 (87.5)	125 (78.1)	8.229	0.004*
Prolonged (>200 ms)	25 (31.2)	10 (12.5)	35 (21.9)		
Mean deceleration time±SD (ms)	188.0±51.6	160.2±30.1	174.1±91.1	*t*-test, 4.169	0.001*

SD: Standard deviation, *Statistically significant

Table 5: Comparison of mean atrial diameter, left ventricular mass index, deceleration time, and diastolic dysfunction among different classes of MELD in patients with CLD (*n*=80).

Variables	MELD classes			ANOVA	*P*-value
	≤9 *n*=26 (%)	10–20 *n*=44 (%)	>20 *n*=10 (%)		
Left atrial diameter± SD (cm)	3.7±0.3	3.8±0.5	3.9±0.5	0.931	0.399
Left ventricular mass index±SD (g/m^2)	110.6± 23.7	114.2± 44.7	125.9± 26.0	0.609	0.546
Deceleration time±SD (ms)	183.0± 49.9	190.0± 57.3	192.1± 25.1	0.184	0.832
Diastolic dysfunction					
Present	8 (25.0)	18 (56.25)	6 (18.75)	FET, 2.560	0.319
Absent	18 (37.5)	26 (54.2)	4 (8.3)		

SD: Standard deviation, FET: Fischer exact test

Torregrosa *et al*.[21] demonstrated increased left ventricular wall thickness and LVMI in CLD patients which improved following liver transplantation. It is possible that increased LVMI, PWTd, and IVSTd in CLD patients are related to the activation of the RAAS and secondary hyperaldosteronism causing left ventricular remodeling and hypertrophy which has been demonstrated to be associated with mortality in CLD patients.[2,4-5]

Our study demonstrated preserved left ventricular systolic function in the CLD patients. EF and FS were similar in the two arms of the study. Similarly, LVOT velocity time integral and stroke volume did not vary significantly between the two arms of the study. Estimated left ventricular cardiac output showed no statistically significant difference between the cases and the controls but estimated left ventricular CI was significantly larger in the CLD group probably due to hyperdynamic circulation. Overall, systolic function appeared to be preserved at rest in CLD patients because of increased left ventricular stroke volume index, marked reduction in systemic vascular resistance, and increase in arterial compliance with resultant reduction in LV afterload and LV stroke work.[7,22] Barbosa *et al*.[23] in their study showed that stress studies particularly dobutamine stress echocardiography may be necessary before concluding on the normality or otherwise of systolic function in CLD patients. The factors that contribute to the systolic dysfunction in CLD during stress include defective adrenoceptor signaling, impaired second messenger coupling, alteration in cardiomyocyte membrane, and the negative inotropic effects of many humoral factors.[5]

Study of diastolic function told a different story. Diastolic dysfunction showed proportional increment in the prevalence with worsening hepatic function, although the differences did not reach statistical significance. This may be because of the relatively small population studied. The presence of diastolic dysfunction has been associated with mortality in cirrhotic patients; the probability of survival at 1 year was 95% in the absence of diastolic dysfunction and 79% with Grade 1 diastolic dysfunction and 39% with Grade 2 diastolic dysfunction.[24] This study did not have any CLD patient with Grade 2 diastolic dysfunction. Diastolic dysfunction has been reported to be more marked in patients with tense ascites and paracentesis resulted in reduction of the A velocity and increase of the E velocity but had no effect on the prolonged DT.[19,20] The use of beta blockers has been positively associated with the presence of diastolic dysfunction in a Greek study that had prevalence of diastolic dysfunction of 59.8%.[25] Sidmal *et al*.[26] in rural India found diastolic dysfunction of different grades in 91.6% of their cases and there was an association between the presence and severity of diastolic dysfunction and the severity of the liver disease. In Egypt, diastolic dysfunction was present in about 40% of their cirrhotic patient using pulse wave Doppler study of mitral inflow.[22] LVDD results from increased myocardial stiffness, altered cardiac extracellular matrix, myocardial fibrosis, and sub-endothelial edema.[2-4] Evidence of diastolic dysfunction in CLD should prompt institution of measures to control PoHTN.

CONCLUSION

CLD patients have abnormal cardiac structural changes which include interventricular septal hypertrophy, increased LAD, and increased LVMI. These changes are associated with cardiac dysfunction characterized by preserved systolic function at rest and diastolic dysfunction that progresses with worsening hepatic function.

Limitation

The small sample size and the lack of exclusion of CLD patients on beta blockers are major limitations to the study. The relationship between beta blockade and cardiac dysfunction in CLD is complex. While beta blockers are used to ameliorate PoHTN, which is the predominant pathophysiological mechanism underlying the development of cardiac dysfunction, their long-term use tend to impact negatively on diastolic function.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Møller S, Bernardi M. Interactions of the heart and the liver. Eur heart J 2013;34:2804-11.
2. Páll A, Czifra Á, Vitális Z, Papp M, Paragh G, Szabó Z. Pathophysiological and clinical approach to cirrhotic cardiomyopathy. J Gastrointestin Liver Dis 2014;23:301-10.
3. Martell M, Coll M, Ezkurdia N, Raurell I, Genescà J. Physiopathology of splanchnic vasodilation in portal hypertension. World J Hepatol 2010;2:208-20.
4. Yang Y, Lin H. The heart : Pathophysiology and clinical implications of cirrhotic cardiomyopathy. J Chine Med Assoc 2012;75:619-23.
5. Gassanov N, Caglayan E, Semmo N, Massenkeil G, Er F. Cirrhotic cardiomyopathy : A cardiologist's perspective. World J Gastroenterol 2014;20:15492-8.
6. Kowalski HJ, Abelman WH. The cardiac output at rest in Laennec cirrhosis. J Clin Investig 1953;32:1025-33.
7. Adebiyi AA, Ogah OS, Akere A, Otegbayo JA. Echocardiographic study of left ventricular structure and function in Nigerian patients with chronic liver disease. Nig J Cardiol 2017;14:26-30.
8. Ahmed B, Jean J, Seth K, Miriam K, Anoop S, Shashwatee B. Risk of cardiovascular disease due to chronic hepatitis C infection: A review. J Clin Transl Hepatol 2017;5:343-62.
9. Okonkwo U, Nwosu M, Bojuwoye B. The predictive values of the meld and child-pugh scores in determining mortality from chronic liver disease patients in Anambra state, Nigeria. Intern J Gastroenterol 2010;10:1-8.
10. Mokdad AA, Lopez AD, Shahraz S, Lozano R, Mokdad AH, Stanaway J, *et al*. Liver cirrhosis mortality in 187 countries between 1980 and 2010 : A systematic analysis. BMC Med 2014;12:1-24.
11. Scarlatescu E, Droc G, Tomescu D. The early stage of cirrhotic cardiomyopathy is a risk factor for postreperfusion syndrome during liver transplantation ? A retrospective observational study. J Transl Med 2016;21:52-6.
12. Mosteller RD. Simplified calculation of body-surface area. N Engl J Med 1987;317:1098-8.
13. Nagueh FS, Smiseth AO, Appleton PC, Byrd FB, Dokainish H, Edvardsen T, *et al*. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: An update from the American society of echocardiography and the european association of cardiovascular imaging. J Am Soc Echocardiogr 2016;29:277-314.
14. Charan J, Biswas T. How to calculate sample size for different study designs in medical research. Indian J Psychol Med 2013;35:121-6.
15. Ansa VO, Peters EJ, Ekott JU. Distribution of liver diseases among medical admissions in university of calabar teaching hospital. Mary Slessor J Med 2000;2:5-8.
16. Okonkwo UC, Nwosu MN, Ukah C, Okpala OC, Ahaneku JI. The clinical and pathological features of hepatocellular carcinoma in Nnewi, Nigeria. Niger J Med 2011;20:366-71.
17. Lemoine M, Eholie S, Lacombe K. Reducing the neglected burden of viral hepatitis in Africa: Strategies for a global approach. J Hepatol 2015;62:469-76.
18. Kamal N, Ashok G, Alpana P, Praffal K, Parikshit K. Evaluation of cardiac function in patients with liver cirrhosis: A hospital based cross sectional study. Natl J Med Res 2014;4:208-11.
19. Li X, Yu S, Li L, Han D, Dai S, Gao Y. Cirrhosis-related changes in left ventricular function and correlation with the model for end-stage liver disease score. Int J Clin Exp Med 2014;7: 5751-7.
20. Dadhich S, Goswami A, Jain VK, Gahlot A, Kulamarva G, Bhargava N. Cardiac dysfunction in cirrhotic portal hypertension with or without ascites. Ann Gastroenterol 2014;27:244-9.
21. Torregrosa M, Aguade S, Dos L, Segura R, Gonzalez A, Evangelista A, *et al*. Cardiac alterations in cirrhosis : Reversibility after liver transplantation. J Hepatol 2005;42: 68-74.
22. Ashmawy M, Younis H, Abd Elbaset M, Rahman A, Ashmway A, Shawky M, *et al*. Evaluation of cardiac function in patients with liver cirrhosis using tissue Doppler study. Egypt J Intern Med 2018;30:115.
23. Barbosa M, Guardado J, Marinho C, Rosa B, Quelhas I, Lourenco A, *et al*. Cirrhotic cardiomyopathy : Isn't stress evaluation always required for the diagnosis ? World J Hepatol 2016;8:200-6.
24. Finucci G, Desideri A, Sacerdoti D, Bolognesi M, Merkel C, Angeli P, *et al*. Left ventricular diastolic function in liver

cirrhosis. Scand J Gastroenterol 1996;31:279-284.
25. Papastergiou V, Skorda L, Lisgos P, Papakonstantinou N, Giakoumakis T, Ntousikos K, *et al*. Ultrasonographic prevalence and factors predicting left ventricular diastolic dysfunction in patients with liver cirrhosis: Is there a correlation between the grade of diastolic dysfunction and the grade of liver disease? Sci World J 2012;2012:1-6.
26. Preshant SS, 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.

How to cite this article: Njideofor U, Okonkwo U, Ansa VO, Odigwe CO. Diastolic versus systolic dysfunction in patients with chronic liver disease – A single center study in South-South, Nigeria. Calabar J Health Sci 2021;5(1):1-7.