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Evaluating Cardiac Function in Patients with Liver Cirrhosis

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Abstract

Background and Aim: Cirrhotic cardiomyopathy affects heart health, while cardiac cirrhosis affects liver function. Both circumstances can occur simultaneously. Aim of the study was to evaluate cardiac function in patient of liver cirrhosis. ***Material and Methods:*** This study was carried out in the department of medicine, SMIMER hospital, Surat, Gujarat from June 2012 to august 2013. 50 patients, fulfilling the inclusion criteria. A thorough clinical history and examination were conducted for every patient presenting with liver cirrhosis. Fifty cirrhotic patients and were hospitalized and their pulse, blood pressure, ECG were obtained; blood was taken for liver function test, renal function test, Prothrombin time and complete blood count. Simultaneously 2D-echocardiography was performed in all 50 patients and various cardiac parameters were measured. ***Results:*** There Was a rise in both the LVPW and IVS components. The patients we had exhibited a higher PAP. Among fifty patients, thirty-three were identified as having ME/MA. Consequently, around 66% of patients, which equates to 33 out of 50, were identified as having diastolic dysfunction. Our findings indicate that there is no significant difference between our study and the previous one regarding LVDD, RVDD, EF%, ME, MA, ME/MA, and DT (mitral). There is significantly higher QTc interval in cirrhotic patients. significant diastolic dysfunction was noted in cirrhotic patients (66% of total patients). ***Conclusion:*** In individuals with cirrhosis, the QTc interval is significantly prolonged compared to those who are healthy. Individuals with cirrhosis experienced a significant enlargement of both their right and left atriums. Research indicated that individuals with cirrhosis did not demonstrate any notable alterations in the size of their left and right ventricles.

Keywords: ***Cardiac Function, Cirrhosis, Diastolic Dysfunction, Echocardiography.***

Introduction

Westerners and Indians suffer from liver cirrhosis, a serious health issue. Chronic alcoholism still plagues this condition. Cirrhosis is a chronic liver disease with proliferating nodules and extensive fibrosis. Due to liver cell death. This illness has distinct clinical symptoms and can be diagnosed histologically. Cirrhotic cardiomyopathy affects heart health, while cardiac cirrhosis affects liver function. Both circumstances can occur simultaneously [1,2].

Abelman pioneered the claim that cirrhosis causes cardiac dysfunction. He discovered hyperdynamic circulation, which increases cardiac output and heart rate and decreases systemic vascular resistance, in alcoholic cirrhosis patients. Later research showed that nitric oxide and other endothelium-dependent substances are essential for peripheral vasodilation. Cirrhosis cardiomyopathy (CMP) is a disease that affects heart contractile function in nonalcoholic cirrhosis in stressed animal models. These problems suggest they may exist without alcohol [3,4].

Cirrhosis usually causes hemodynamic alterations like higher cardiac output, lower systemic vascular resistance, and low arterial pressure. Even though resting cardiac output is high, cirrhotic cardiomyopathy reduces ventricular contractile response to stimuli. Cirrhotic cardiomyopathy prevalence is unknown. It stays inactive until stress, such as physical exercise, drugs, blood loss, or surgery, occurs. Cirrhotic cardiomyopathy rarely causes noticeable heart failure.

Warm skin, spider angioma, palmar erythema, and bounding pulse are hyperdynamic circulation symptoms. The mechanisms of these cardiovascular alterations include neurogenic, humoral, and vascular dysregulations. Widening of the splanchnic and peripheral vessels may cause hyperdynamic circulation by reducing arterial blood volume [5,6].

Recent media reports call this hyperdynamic state "stealing phenomenon, from the splanchnic to the peripheral circulation." Cirrhosis reduces kidney blood flow. This activates the RAAS, sympathetic nervous system, and antidiuretic hormone, which constricts renal artery walls, retains sodium, and increases volume. Circulatory irregularities can cause hepatorenal syndrome, ascites, spontaneous bacterial peritonitis, gastro-esophageal varices, and hepatopulmonary syndrome. Despite conflicting data, cirrhosis patients appear to have diastolic heart dysfunction. These patients also have left ventricular hypertrophy, left atrial enlargement, extended IVRT, and a poor E/A ratio [7,8].

Both systolic and diastolic structural, histological, electrophysiological, and functional abnormalities characterize this syndrome. Experimental animal studies show that cirrhosis involves numerous variables. These include unique membrane biophysical properties, compromised -adrenergic receptor signaling pathways,

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and increased C-GMP-influenced negative isotropic pathway activity [9,10]. Current guidance recommends broad management strategies. Therapy trials are scarce, causing this. Further research is needed. This study examined liver cirrhosis patients' heart function. Aim of the study was to evaluate cardiac function in patient of liver cirrhosis.

Material and Methods

From July 2012 to August 2013, this study was carried out by the Department of Medicine at SMIMER in Surat, Gujarat. SMIMER serves as a tertiary care facility located in South Gujarat. A thorough clinical history and examination were conducted for every patient presenting with liver cirrhosis. Furthermore, two-dimensional echocardiography was employed to evaluate the heart function of the patients.

Inclusion criteria encompass all individuals diagnosed with liver cirrhosis as determined by an abdominal ultrasound.

Exclusion criteria consist of individuals with cirrhosis, minors under 18 years old, and those who decline to participate.

Each patient received a thorough explanation of the study, and all provided their verbal consent to participate. A total of fifty patients who satisfied the inclusion criteria were part of the study. A thorough assessment of the clinical profile, laboratory tests, imaging studies such as computed tomography and abdominal ultrasound, along with two-dimensional echocardiography, was conducted for each patient.

All patients were admitted to the hospital, where their heart rates and blood pressure were assessed. The serum was analyzed for albumin, total bilirubin, direct bilirubin, and indirect bilirubin, all collected simultaneously. Tests include RBS, SGOT, SGPT, ALP, Blood Urea, and serum electrolytes. A one-step rapid qualitative immunoassay test was conducted to detect the hepatitis B surface antigen, serving as a viral marker for hepatitis B. A rapid visual spot test was conducted to qualitatively detect antibodies to HCV in human serum or plasma, specifically for the viral marker of hepatitis C. Both of these assessments were identified. Ultrasound imaging of the abdomen was performed on all patients. Based on the history of significant alcohol intake, the diagnosis of alcoholic liver cirrhosis was established.

Clinical evidence indicated portal hypertension, evidenced by splenomegaly, along with abnormalities observed during the ultrasound examination. An investigation was conducted on abnormal liver function tests in patients diagnosed with cirrhosis. Furthermore, we were unable to perform metabolic investigations, autoimmune serological tests, and other hepatitis markers due to a lack of necessary resources at our institute. The diagnosis of cryptogenic cirrhosis was established when the HbsAg and Anti-HCV antibody tests returned negative results, alongside a lack of significant alcohol consumption history.

All patients who received an echocardiogram underwent both M-mode and comprehensive two-dimensional echocardiography. The patients were evaluated while positioned supine and also in a partially reclined position on their left side, at an angle of 30 degrees between the left and right sides. M-mode echocardiographic measurements of the following aspects of left ventricular function were conducted following the guidelines set forth by the American Society for Echocardiography.

Measurements of the internal components of the left ventricle:

The end of systolic dimension (ESD) is measured in centimeters.

This represents the conclusion of the diastolic dimension, recorded in centimeters.

The proportion of left ventricular ejection fraction (LVEF) determined through a refined application of Simpson's method.

The "rule of disks" or the Simpson rule is the most commonly used method for calculating the volume of the ventricles. This approach requires capturing an apical view, either in four or two chambers, allowing for the delineation of the endocardial border during both end-diastole and end-systole phases. The ventricle is subsequently divided along its long axis into a series of disks, each maintaining the same height. This procedure is carried out multiple times. The volume of each individual disk is calculated by taking the height of the disk and dividing it by the area of the disk.

Here, the height is defined as the total length of the left ventricular long axis divided by the number of segments within the disk. The surface area of each disk can be determined by utilizing the diameter of the ventricle at that specific location. The volume of the ventricle is determined by summing the volumes of each disk, which are uniformly arranged along the ventricle's long axis. If the ventricle is contracting symmetrically, the actual ventricular volume is typically represented by either the four-chamber or the two-chamber view.

In practical application, the apical two-chamber view is often examined in a tangential approach. The volume derived from this perspective might underestimate the true volume of the left ventricle. The foreshortening of the ventricular apex can result in an inaccurate assessment of the left ventricular ejection fraction, often leading to an overestimation of the EF, no matter the viewpoint taken. Due to the previously discussed factors, the precision of a single plane image may be compromised if there is any asymmetry in the geometry of the ventricles or an irregularity in the motion of the systolic wall. In this specific situation, the accuracy of the volume measurement will be enhanced by utilizing a biplane.

Doppler studies involved conducting Doppler echocardiography on all patients to evaluate left ventricular diastolic function. This was done alongside 2-D echocardiography visualization of cardiac anatomy in the apical four-chamber view. A Doppler sample was strategically placed within the inflow area of the ventricle, just below the mitral valve annulus, near the tips of the mitral valve and aligned parallel to the presumed axis of blood flow. This positioning aimed to reduce the potential impact of transducer angulations. The Doppler sampling volume was adjusted in various planes until the maximum diastolic flow velocities were recorded or until the optimal spectral pattern was achieved.

To assess the diastolic function of the left ventricle (LV), the following pulsed wave Doppler indices of ventricular function were documented from the waveform of the transmitral velocity obtained.

1. - Peak E velocity (E) represents the initial filling velocity, indicating the early filling measured in meters per second per second.

- The later filling velocity, indicative of the atrial contribution, corresponds to the peak A velocity (A) measured in meters per second.

The calculated ratio of Peak E to Peak A (E/A) is below 1, suggesting a reduced level of left ventricular compliance.

2. The IVRT

3. Length of slowing down (MV)

Investigation of continuous wave Doppler, assessment of RVSP through TR jet, and application of color Doppler techniques.

Results

In this study, out of fifty patients with cirrhosis of the liver, 38 were male and 12 were female. The majority of individuals fall within the age range of 41 to 50 years old. A significant portion of the patients, totaling 48, falls within the younger age group, specifically between 21 and 40 years old.

In the study, forty-nine out of fifty patients presented with jaundice, forty-nine experienced ascites, one reported dyspnea, two showed signs of encephalopathy, and seven had melena. In the study group, a total of 36 patients reported alcohol consumption, whereas only 14 patients abstained from alcohol use. Throughout the trial, the symptoms that were most commonly observed included icterus at 66% and pedal edema at 42%.

The average pulse rate observed in this study is 100.54 beats per minute, surpassing the findings of similar studies conducted previously. The average blood pressure observed in this study is 90.35 mm/hg, indicating a lower reading compared to previous similar studies, and aligns with their results. The study revealed a mean QTc interval of 0.41 seconds, indicating a higher level than previous comparable studies and in comparison, to individuals without any abnormalities.

Echocardiography was conducted on all fifty cirrhotic patients involved in this study.

A comparison of our study's results with similar studies reveals significantly larger values in RA and LA diameter, along with a significant decrease in the ratio of TE to TA. Furthermore, there is a rise in both the LVPW and IVS components. The patients we had exhibited a higher PAP. Among fifty patients, thirty-three were identified as having ME/MA. Consequently, around 66% of patients, which equates to 33 out of 50, were identified as having diastolic dysfunction. Our findings indicate that there is no significant difference between our study and the previous one regarding LVDD, RVDD, EF%, ME, MA, ME/MA, and DT(mitral).

Table 1: Symptoms present in the patients

Symptoms	No. of patients
Ascitis	49
Encephalopathy	2
Malena	7
Dyspnea	1
Jaundice	39

Table 2: investigations results obtained in the study

Lab investigations	Mean value obtained (SD)
Hemoglobin	9.2
TC	7144
Blood urea	38.4
S. Albumin	1.0
S. Creatinine	2.9
S. Bilirubin (T)	6.1
S. Bilirubin (D)	4.35
S. Bilirubin (I)	1.8

Discussion

This study was conducted within the department of medicine over a period of about one year. A total of fifty patients who fulfilled the inclusion criteria were invited to take part in the trial. Individuals with cirrhosis experience hyperdynamic systemic circulation, marked by elevated heart rate and cardiac output, alongside reduced systemic vascular resistance and low arterial blood pressure. Increased sympathetic nerve activity, elevated blood volume (due to increased preload), and the existence of arteriovenous connections are factors that can contribute to a rise in pulse rate.

The study revealed a mean QTc interval of 0.41 seconds, indicating a higher level than that observed in previous comparable studies and in normal individuals. Patients with cirrhosis often experience a prolonged Q-T interval. The severity of the condition tends to escalate alongside the progression of the disease, which can have important implications for prognosis. Cirrhosis negatively affects the permeability of the plasma membrane and disrupts the function of its ion channels. Cirrhosis in humans leads to a decreased ability to regulate vascular tone through potassium and calcium channels across various cell types. These modifications lead to electrophysiological abnormalities in cardiac excitation and an extension of the QTc interval. Subsequent research has shown that the QTc interval remains unaffected by the underlying cause of cirrhosis, and it is linked to the Child Pugh score and liver tests. Individuals with a QTc exceeding 0.44 seconds (440 milliseconds) experienced a notably lower survival rate compared to those with a normal QTc [11,12].

In comparison to the results of the earlier study, our research showed no variations regarding LVDD, RVDD, EF%, ME, MA, ME/MA, or DT (mitral). Our research revealed that the enlargement of both atria can be seen as a response of the heart's hemodynamics to alterations in peripheral blood circulation. The impairment was shown by a slight increase in the speed of the E wave, a significant rise in the speed of the A wave, a notable increase in the time taken for the E wave to slow down, and a considerable decrease in the ratio of the E wave to the A wave. The typical noninvasive indicators of diastolic dysfunction include a change in the Doppler profile, showing a reduced E wave, an increased contribution from the atrium to ventricular filling, and a heightened E/A ratio. These traits are considered indicative of the condition. Cirrhosis is marked by alterations in the diastolic function of the left ventricle, as shown by these findings. When ascites occurs, this alteration becomes more evident than it typically would be.

The likely reason for this is that the expansion of the ventricles during diastole is obstructed by the heightened pressure within the thoracic cavity and the diaphragm's bulging, both of which result from fluid buildup in the abdominal area. Nonetheless, it is evident even in the absence of ascites, indicating that there are additional factors at play that are not purely mechanical. It can be inferred that one potential cause is an increase in heart diameters, leading to a reduced capacity for the ventricular wall to expand further. The stiffness of the myocardial wall may be influenced by uneven fibrosis and an increase in heart weight, potentially resulting in reduced filling of the left ventricle and diastolic dysfunction [13].

Patchy fibrosis could potentially play a role in this situation. An increase in cardiac preload can occur due to expanded blood volume, potentially resulting in overload and a decrease in cardiac contractility. Alcoholic cirrhosis has been linked to systolic dysfunction, regardless of the presence of clinical signs and symptoms of heart disease. This phenomenon was not observed in our patient. Additionally, alcoholic heart disease is defined by a distinct echocardiographic pattern, which features a significant reduction in systolic function [14].

The results of our research indicated that there was no noticeable difference in the level of impairment in ventricular function, specifically concerning systolic function, between patients with cirrhosis who consumed alcohol and those who did not. Consequently, we reached the understanding that the role of alcohol in cardiac issues cannot be dismissed; instead, it is essential to consider other contributing factors. Examples of these factors

include endotoxin, bile acids, tumor necrosis factor, and catecholamines.

Various factors can significantly impair cardiac functions and are commonly elevated in cases of advanced cirrhosis. The cardiac hypertrophy noted in our cirrhotic patient led to an increase in left ventricular wall thickness (LVPCO + IVS), which was also associated with impaired distensibility of the cardiac walls across different clinical scenarios. This observation was made following the patient's diagnosis of cirrhosis. Our research does not shed light on the mechanisms responsible for the increase in the thickness of the left ventricular wall. Conversely, it can be inferred that renin, angiotensin, and aldosterone play a role, as angiotensin II, E, and NE have been shown to promote the growth of heart tissue in both in vitro and in vivo environments [15].

Indeed, patients with cirrhosis exhibited a reduction in pulmonary vascular resistance. While the exact mechanism behind increased PAP remains unclear, previous studies have indicated a heightened presence of vasoactive substances in the pulmonary circulation, along with a potential detrimental impact of these substances on endothelial cells. There is a hypothesis suggesting that microthrombi may migrate to the pulmonary vascular bed through Porto systemic shunts, potentially leading to an increase in blood vessel resistance.

Conclusions

In individuals with cirrhosis, the QTc interval is significantly prolonged compared to those who are healthy. Individuals with cirrhosis experienced a significant enlargement of both their right and left atriums. Research indicated that individuals with cirrhosis did not demonstrate any notable alterations in the size of their left and right ventricles. A significant prevalence of diastolic dysfunction was noted among individuals with cirrhosis, representing 66% of the total patient population in our study. In both cases of cirrhosis, there was no observable indication of significant left ventricular systolic failure. Cirrhotic individuals exhibited significantly elevated levels of LVPW, IVS, and PAP compared to those who are healthy. We lacked the capability for tissue Doppler assessment of diastolic dysfunction in the ventricle, and we also did not have access to three-dimensional echocardiography.

List of Abbreviations

ECG: electrocardiogram
LVPW: left ventricular posterior wall
IVS: interventricular septum
LVDD: left ventricular diastolic dysfunction
RVDD: Right ventricular diastolic dysfunction
EF: ejection fraction
RBS: Random Blood Sugar
ALP: alkaline phosphatase
HCV: Hepatitis C virus
HbsAg: Hepatitis B surface antigen
LVEF: left ventricular ejection fraction
RVSP: right ventricular systolic pressure
PVP: pulmonary alveolar proteinosis

Declarations

Availability of data and material

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Conflict of Interest

The authors declared that there are no conflicts of interest

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Not applicable

Authors ‘contributions

K.P.K., & C.V. V., design the concept; K. M. D., involved in the recruitment of cases; P.A.K. involved in the data acquisition; K.P.K., K. M. D., were involved in the data analysis; K.P.K., & C.V. V., were involved in the manuscript preparation. All the authors have reviewed the manuscript.

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