Original Research Article

Cardiac changes in hepatic cirrhosis in Government Dharmapuri Medical College Hospital, Dharmapuri

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Abstract

Introduction: Cirrhosis liver, is characterized by diffuse destruction and regeneration of hepatic parenchymal cells leading to deposition of connective tissue with resulting disorganization of the lobular and vascular architecture. Despite the remarkable regenerative capacity of the liver, once hepatic parenchymal reserve is exceeded, clinically overt or decompensated cirrhosis ensues. Portal hypertension develops due to resistance to blood flow through the liver resulting increase in portal venous pressure leading to diversion of blood flow through low resistance portosystemic collaterals thereby bypassing the liver. The current study was designed to precisely evaluate the cardiovascular system in a group of patients with hepatic cirrhosis based on clinical examination.

Aim of the study: To clinically evaluate patients with hepatic cirrhosis with respect to changes in heart rate, blood pressure, mean arterial pressure, ECG jugular venous pressure and precordial examination.

Materials and Methods: 50 patients of cirrhosis liver were selected for the study. These patients were admitted in the general medical wards. All patients were subjected to routine investigations. All patients were subjected to ultrasound scan abdomen to confirm the diagnosis of cirrhosis. Patients with ascites underwent abdominal paracentesis and fluid was analyzed for protein content and cells. All patients were then subjected to electrocardiography, chest X-ray and M-mode 2-Dimensional echocardiography.

Results: Out of the 50 patients studied 35 (70%) were males and 15 (30%) were females. The age of the patients ranged from 19 years to 75 years. 5 Patients (10%) were alcoholics, 14 patients (28%) had past history of jaundice or, 8 patients (16%) presented with haemetemesis. Among this 4 patients were

HbsAG+ (8%) and 2 patients were diabetics 4%. All patients had sonographic evidence of cirrhosis.with portal hypertension. Out of 50 patients, 3 patients had elevated blood pressure. Previous studies show that the systolic blood pressure more than 160 mmHG and diastolic blood pressure more than 95 mmHg are the range for hypertension in cirrhotic patients. The electro cardiac gram showed an average heart rate of 82/ m. The low QRS voltage in chest leads and limb leads were found in 10 patients. T wave inversion was found in chest leads (V1 to V3 / V6) in 4 (8%) patients, in II, III avf in 7 patients (14%). The chest roentgenogram showed Hepatic Hydrothorax in 5 patients (10%). Cardio megaly was evident in chest X – ray in 11 patients (22%).

Conclusion: The result of this study clearly showed that a large number of patients with hepatic cirrhosis are asymptomatic (40%) about cardiovascular system, have evidence of cardiac involvement in electrocardiography and echo cardiograph. cardiac decompensation in cirrhosis is rare despite the high output state and its presence as indicated by left ventricular systolic dysfunction.

Key words

Cirrhosis liver, Jaundice, Cardiac decompensation, Portal hypertension.

Introduction

Cirrhosis liver reflects irreversible chronic injury to the hepatic parenchyma characterized by extensive fibrosis, in association with the formation of regenerative nodules with resulting disorganization of the lobular and vascular architecture. Decompensated cirrhosis leads to portal hypertension and hepatocellular failure. Hyperdynamic circulation more directly suggests the presence of portal hypertension and hepatocellular failure. Various studies have been carried out over the years to evaluate the cardiac and hemodynamic changes in cirrhosis of the liver [1]. Clinical features of cirrhosis derive from the morphologic alterations and after reflecting the severity of liver damage rather than the etiology of the underlying liver disease. Loss of functioning hepatocellular mass may lead to jaundice, edema, coagulopathy, spider telangiectasia, palmar erythema, parotid and lacrimal gland enlargement, nail changes, Dupuytren's contractures, gynaecomastia, ascites, testicular atrophy as well as confusion and asterixis suggesting hepatic encephalopathy. Distorted vasculature leads to portal hypertension [2]. Portal hypertension develops when resistance to blood flow through the liver, is increased and resulting an increase in portal venous pressure lead to diversion of blood flow through low resistance portosystemic collaterals thereby bypassing the liver. Hyperdynamic circulation, caput medusa, splenomegaly and gastro oesophageal varies more directly suggest the presence of portal hypertension. Ascites is usually preceded by abdominal distension edema of the legs is frequently seen [3]. The liver may be enlarged and firm or contracted and impalpable. The spleen may be palpable and firm. Hematological manifestations of cirrhosis include anemia, leukopenia, and thrombocytopenia which may result from splenomegaly and hypersplenism. The fundamental hemodynamic abnormality is an increased resistance to portal blood flow. This may be intra hepatic as in cirrhosis or due to obstructed portal vein due to thrombosis [4]. As the portal venous pressure is lowered by the development of collaterals deviating portal blood into systemic veins, the portal hypertension is maintained by increasing the blood flow in the portal system which thus becomes hyperdynamic. Resistance to portal blood flow is exerted along both the hepatic and portal collateral circulation and appears to be modified by vasoactive agents. Portal hypertension is defined as portal venous pressure exceeding 12 mm Hg. Ultrasonography has proved to be a useful non-invasive and inexpensive method to establish the presence of and etiology of portal hypertension [5]. A normal ultrasound shows the liver to have mixed echogenicity. In cirrhosis of the liver, the edge of the liver may be irregular

and the liver shows coarse echo pattern. It has a finely stippled echogenicity due to increased acoustic attenuation. In end stage cirrhosis, the liver is small and very echogenic. Chronic liver diseases like cirrhosis produce high cardiac output states [6]. The mechanisms are uncertain but have been attributed to increased blood volume, intrahepatic arteriovenous shunts, mesenteric arteriovenous shunts and defects in inactivation of circulating vasodilators'-Mode 2-Dimensional echocardiography is a useful – invasive method of studying the various morphological and functional parameter [7]. In patients with cirrhosis liver, prior studies have shown that right ventricular end diastolic volume and right ventricular end systolic volume were significantly reduced in patients whereas left ventricular end diastolic volume and left ventricular end systolic volume and left atrial volume were normal or slightly increased [8]. The right ejection fraction was significantly increased and the left ejection fraction was slightly decreased. Stroke volume was significantly greater. There is also evidence of myocardial contractile function impairment and ventricular hypo responsiveness to pharmacological or physiological stress [9]. Diastolic dysfunction was found to be 35% in prior studies and more common in alcoholic than in non-alcoholics. These changes are reversed following liver transplantation. Pericardial effusion has been demonstrated in a significant number of patients and is an echo free zone surrounding the heart and if large the whole heart can be seen swinging into it [10].

Materials and methods

The present study was conducted in the Government Dharmapuri Medical College Hospital from October 2016 to May 2017. 50 patients with cirrhosis liver were selected for the study. These patients were admitted to the general medical wards. Criteria followed for selection of patients included:

- Only patients with clinical, biochemical and sonographic evidence of cirrhosis liver were selected.
- Patients with previously detected heart disease were excluded from the study.
- Patients with inter current illness and those who were critically ill were excluded from the study.
- Patients with cardiac cirrhosis were excluded from the study.

A detailed history was elicited form the patient with special reference to cardiovascular symptoms. A thorough physical examination was performed in the patients and a special note was made regarding heart rate and rhythm, blood pressure, jugular venous pulse, and pressure and precordial examination. All patients were subjected to routine investigations viz, Blood urea, sugar, complete hemogram, serum cholesterol and liver function tests. All patients were subjected to ultrasound scan abdomen to confirm the diagnosis of cirrhosis. Patients with ascites underwent abdominal paracentesis and fluid was analyzed for protein content and cells. All patients were then subjected to electrocardiography, chest X-ray, and M-mode 2-Dimensional echocardiography.

Results

3 Patients (6%) had a congestive cardiac failure. The average pulse rate was 84 and it ranged from 54/m to 120/m. The jugular venous pressure was elevated in 6 (12%) patients. The systolic blood pressure from ranged 90mmHg to 160 mmHg., the average being 110.mmHg. The diastolic blood pressure ranged from 50 mmHg to 100 mmHg., the average being 70 mmHg. The mean arterial pressure ranged from 70 mmHg to 110 mmHg the average being 86 mmHg Functional high output systolic flow murmur was detected in 3 (6%) patients. Out of 50 patients, 3 patients had elevated blood pressure. Previous studies show that the systolic blood pressure more than 160mmHG and diastolic blood pressure more than 95 mmHg are the range for hypertension in patients. The electro cardiac gram showed an average heart rate of 82 / m. The low QRS voltage in chest leads and limb leads were found in 10 patients. T wave inversion was found in

chest leads (V_1 to V_3 / $V_{6)}$ in 4 (8%) patients, in II, III avf in 7 patients (14%). Regarding Hemiblock 2 (4%) cases were observed in the study. qs complex in anterior and Inferior leads is seen in 4 patients (8%).The chest roentgenogram showed Hepatic Hydrothorax in 5 patients (10%). Cardio megaly was evident in chest X – ray in 11 patients (22%) .M-Mode 2 Dimensional Echocardiographic studies, done showed abnormality in 15 (30%) patients. Pericardial effusion was detected in 4(8%) patients. Regarding enlargement of cardiac chambers – all 4 chambers were enlarged in 3 (6%) patients. With global hypokinemia, a nd left ventricular hypertrophy in 1 (2%) patient. One case of Porto pulmonary HT was observed in our study (2 %). Akinesia of the inferior and anterior wall was seen in 4 (8%) patients and hyperdynamic flow due to anemia was observed in 2 patients (4 %).

Chest roentgenograms showed that the elevated hemidiaphragm were the commonest abnormality detected and all these patients had ascites and the elevated hemidiaphragm probably reflecting increased intra-abdominal pressure. Cardiomegaly was detected in 11 patients. Hepatic Hydrothorax was found in 5 patients (**Table – 1 to 4**).

Table – 1: Cardiovascular symptoms and signs among cirrhosis patients.

SYMPTOMS PRESENT		RANGE
PULSE RATE	84/ min	54 to 120/min
JVP ELEVATION	12 %	-
BLOOD PRESSURE		
SYSTOLIC	110 mmHg	90 to160 mmHg
DIASTOLIC	70 mmHg	50 to 100 mmHg
MAP	84 mmHg	70 to 110 mmHg
HEART MURMURS	6%	-
CONGESTIVE CARDIAC FAILURE	6 %	-

Table – 2: Electrocardiography changes among cirrhosis patients.

ECG ABNORMALITIES	INCIDENCE (N=24)	%
LOW VOLTAGE COMPLEXES	10	20
CAHD / INFARCTION	4	8
ISCHEMIC CHANGES	10	20

Table – 3: Chest roentgenograms evaluation of patients under study is as follows.

X- RAY CHANGES	**INCIDENCE (N = 16)**	**%**
CARDIOMEGALY	11	22
HEPATIC - HYDROTHORAX	5	10

Discussion

The etiology of cirrhosis in a patient under study showed a slightly higher incidence of post hepatic or post necrotic cirrhosis as compared to western studies. About the cardiovascular evaluation, the average heart rate in the present study was 84±2 beats per minute. Other studies showed the average heart rate as follows [11]. Thus the present study confirms that there is an increase in heart rate in cirrhosis liver as compared to the average heart rate of healthy subjects, reflecting a hyper dynamic circulatory state. The average systolic blood pressure,

diastolic blood pressure and mean arterial pressure in the present study were 110 mm/Hg and 70 mm/Hg and 86mmHg respectively. In the other studies, they were as follows: The present study shows that the mean arterial pressure is comparable with another study but the diastolic pressure is within the normal range. The elevation of the jugular venous pulse in 12% of patient reflects an increase in the plasma volume and fluid over load. Significant correlation was demonstrated between the heart rate and mean arterial pressure both of which indicate a hyper dynamic circulation and serum albumin and serum bilirubin levels both of which are indicators of liver dysfunction [12]. The present study clearly demonstrated that hyperdynamic circulation progressively increase with the severity of the liver dysfunctions. One study concludes that the severity of cirrhosis is closely related to the degree of hyperkinetic circulatory state and portal hypertension. Significant positive correlation was noted between decreased MAP in 36 % and increased HR in 54 %, similarly decreased serum albumin by 32 % and increased serum bilirubin in 44%.The present study shows that out of 50 patients 3 patients are hypertensive 6%.Regarding the electrocardiographic findings not many studies are available showing the various electrocardiographic abnormalities in cirrhosis liver [13]. It has been said that cardiac arrhythmias in cirrhosis liver are always due to a definable precipitating event such as hypo or hyperkalemia, acidosis, hypoxia or cardiac irritation due to insertion of lines, although in older patients the possibility of ischemic heart disease must not be ignored. One study demonstrated that cardiac arrhythmias is common during surgery, the commonest arrhythmias being a premature ventricular contraction [14]. The current study shows that low voltage QRS complexes were present in 10 patients. Out of which 4 had pericardial effusion probably reflecting the presence of occult pericardial effusion. T wave inversion was noted in the precordial leads and limb leads in 11 patients and CAHD changes in 4 Patients had no symptoms referable to the cardiac system [15].

Table – 4: Echocradiographic evaluation of patients under study is as follows.

ECHO ABNORMALITIES	INCIDENCE (N = 15)	%
Cardiomyopathy Global hypokinemia and chamber enlargement present	3 (Out of 3, One patient was Alcoholic)	6
Pericardial Effusion	4	8
CAHD changes	5	10
Porto Pulmonary HT	1	2
Hyper dynamic Flow due to anemia	2	4
LV Dysfunction (LVEF<49%)	3	6

Conclusion

The result of this study clearly show that a large number of patients with hepatic cirrhosis are asymptomatic (40%) about cardiovascular system, have evidence of cardiac involvement in electrocardiography and echo cardiograph. cardiac decompensation in cirrhosis is rare despite the high output state and its presence as indicated by left ventricular systolic dysfunction. The incidence of hypertension in cirrhosis patients, our study shows 3 patients (6 %). Electrocardiographic abnormalities include low voltage complexes due to pericardial effusion nonspecific T wave abnormalities and CAHD changes. A cardiac evaluation is a pre-requisite in patients with cirrhosis undergoing stress like surgery because the presence of cardiac involvement adds to the morbidity and mortality.

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