Original Research Article

Study of liver biochemical profiles in congestive heart failure patients in Government Dharmapuri Medical College, Dharmapuri

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Abstract

Introduction: Heart failure causes a number of pathophysiological affects which, alone or in combination result in liver cell damage. As a consequence, liver function abnormalities are so common in heart failure. Liver dysfunction in heart failure is usually mild and asymptomatic and often detected incidentally on routine liver biochemical investigations.
Aim of The Study: To study the influence of congestive heart failure on liver biochemical profiles.
Materials and methods: Among cases admitted with heart failure in the medical wards, government general hospital, sixty patients who had met the inclusion and exclusion criteria were taken up for study.
Results: Among the total heart failure cases, 40% were due to rheumatic heart disease. Dilated cardiomyopathies represent about 23% of cases. Heart failure secondary to coronary artery heart disease is seen in about 13% of cases. We have found five cases with acute heart failure and three cases with hypotension. Present study revealed a strong correlation between liver function derangements and the above cases.
Conclusion: The study observed 20% of cases with jaundice. Among sixty cases liver enlargement was seen in 63% of cases. Increased liver size is strongly correlated with hyperbilirubinemia. Though the conjugated fraction of bilirubin is also elevated, the levels of unconjugated fraction were higher. Serum aminotransferases were elevated in 78% of cases unlike serum alkaline phosphatase which is increased only in 25% of cases. There found to be a significant correlation between rise in unconjugated bilirubin and elevation of serum aminotransferases.

Key words

Liver dysfunction, Heart failure, Hyperbilirubinemia, Serum alkaline phosphatase, Unconjugated bilirubin, Serum aminotransferases.

Introduction

Liver, the largest gland in the body has many complex functions. For the liver to perform its primary functions, high rates of blood flow and close contact between sinusoids and hepatocytes are essentials [1]. As a result of its complex vascular supply and high level of metabolic activity, the liver is uniquely vulnerable to a broad spectrum of circulatory disturbances [2]. Heart failure causes a number of pathophysiological affects which, alone or in combination result in liver cell damage [3]. As a consequence, liver function abnormalities are so common in heart failure. Liver dysfunction in heart failure is usually mild and asymptomatic and often detected incidentally on routine liver biochemical investigations [4]. Present study was undertaken, mainly to emphasize the importance of early identification of liver serum biochemical markers in heart failure patients. Liver biochemical tests are found to be very useful in assessing the duration and severity of heart failure. Early and adequate treatment of the underlying cause of heart failure reverts liver function derangements to normal and prevents permanent liver damage [5].

Materials and methods

The study was conducted in Medical wards, Government Dharmapuri Medical College Hospital, Dharmapuri. Present study was single Centre, cross-sectional and analytical study. Sixty patients with various etiologies of heart failure were observed. Period of Study was from July 2016 to March 2017. Informed consent was obtained in all cases. Congestive heart failure in all age groups was included in the study.

Exclusion criteria

- History of alcoholism.
- Past history of jaundice.
- Recent intake of hepatotoxic and cholestatic drugs.
- Presence of HBs Antigen and Anti HCV Antibodies.
- Pregnancy.

Selection of controls

Among patients who attended medicine outpatient department, government general hospital, for general health check-up, twenty persons were taken up as controls. Controls and study group were matched according to the age and gender. They are excluded from diseases which are thought to influence the study by appropriate investigations [6].

Laboratory investigations done in the study

- Serum total bilirubin (direct and indirect).
- Serum aspartate and alanine aminotransferases.
- Serum alkaline phosphatase.
- Serum proteins.
- Serum albumin.
- Prothrombin time.

Results

Gender distribution in heart failure was as per **Table – 1**. Percentage of jaundice was as per **Table – 2**. Percentage of hepatomegaly was as per **Table – 3**. Biochemical values in cardiac failure patients were as per **Table – 4**.

Table – 1: Gender distribution in heart failure.

SEX	NO. OF CASES
MALE	36
FEMALE	24
TOTAL	60

MALE AND FEMALE RATIO = 1.5: 1.

Discussion

To characterize the incidence and severity of liver function abnormalities in patients with heart

failure, the present study analyzed liver biochemical profiles in sixty cases with heart failure of varied etiologies and mechanisms [7]. Innumerable references about the clinical and biochemical parameters about the hepatic circulation, hepatic function and the overall disturbance in the liver function secondary to heart failure of varied etiologies have been correlated with the present study of sixty cases. Sincere effort has been made to correlate the results. Liver dysfunction in congestive heart failure is usually mild and asymptomatic and often detected incidentally on routine liver biochemical testing [8]. When symptomatic, it may present as mild jaundice. In patients with chronic, severe heart failure, jaundice may be so deep as to suggest biliary obstruction. When jaundice is accompanied by significant AT elevation in patients presenting with acute cardiac decompensation, the clinical picture may simulate that of acute viral hepatitis .Congestive heart failure results in a broad range of liver biochemical abnormalities [9]. Serum bilirubin is mildly elevated with a high unconjugated fraction [10]. Multiple factors are thought to be contributory including hepatocellular dysfunction, hemolysis, pulmonary infarction, canalicular obstruction secondary to distended hepatic veins, medications and superimposed sepsis [11]. Jaundice of right sided heart failure seems to be clinically and pathophysiological distinct from that of ischemic hepatitis [12]. Bilirubin level falls quickly with resolution of failure within three to seven days. Even in the presence of deep jaundice, the serum alkaline phosphatase level is usually normal or only minimally elevated a finding that helps distinguish cardiac from obstructive jaundice [13]. It returns to normal in a week after treatment of failure. Serum AT levels are mildly elevated usually two to three times normal levels, in up to one third of patients with chronic heart failure. In patients with severe, acute heart failure, however, the AT levels may be extremely elevated, simulating those of viral hepatitis, especially when the heart failure is complicated by hypotension [14]. However, high serum AT levels has been described in the setting of heart failure with minimal or no hemodynamic instability. These probably reflect ischemic injury secondary to decreased cardiac output because the degree of AT elevation correlates with the extent of zone 3 necrosis on liver biopsy specimens. After the treatment of heart failure [15], AT levels return to normal within five to ten days. Serum albumin levels are decreased in 30% to 50% of patients with cardiac decompensating. The incidence and degree of change appear to be similar in acute and chronic failure [16]. The serum albumin level does not correlate with the degree of histological damage to the liver Causes of low albumin probably include decreased hepatic synthesis, leakage from a congested intestine, and poor nutrition. With treatment, albumin level tends to rise over a period of one or more months. Prolonged prothrombin times are found in 80% to 90% of patients with acute and chronic heart failure due to decreased hepatic synthesis of vitamin–k dependent clotting factors. It returns to normal in two to three weeks following treatment of the heart failure [17].

Table – 2: Percentage of jaundice.

GENDER	TOTAL NO. OF CASES	NO. OF CASES WITH JAUNDICE
MALE	36	7
FEMALE	24	5
TOTAL	60	12

PERCENTAGE OF JAUNDICE = 20%

Table – 3: Percentage of hepatomegaly.

GENDER	TOTAL NO. OF CASES	NO. OF CASES WITH HEPATOMEGALY
MALE	36	22
FEMALE	24	16
TOTAL	60	38

An uncommon complication of sustained chronic congestive cardiac failure is so–called cardiac sclerosis. The pattern of liver fibrosis is mostly centrilobular [18]. The damage rarely fulfils the

criteria for the diagnosis of cirrhosis, but the historically sanctified term cardiac cirrhosis cannot easily be dislodged.Cardiac sclerosis was the most common form of hepatic fibrosis, occurring in 48% of cases. Cardiac fibrosis was quite frequent but frank cirrhosis was rarely found. Garvin et al, in 1943, found 1.7 per cent of 407 patients were thought to have cirrhosis due to heart diseases. In an another study, Koletsky et al, in 1944, found 35 (4.4 per cent) were considered to have cardiac cirrhosis at postmortem among 790 patients dying of heart diseases.Sherlock et al, 1951, described the clinical and biochemical features of zone 3 necrosis in heart failure. He found lack of correlation between right atrial pressure and the degree of zone 3 necrosis in patients with cardiac decompensating [19]. This is followed by many studies which proved cardiac output is the major determinant of hepatic ischemia which is the main contributing factor for liver function derangements. Sherlock found jaundice and hepatic synthetic function were no worse in patients with cardiac cirrhosis than in those with simple passive congestion. Bang, et al. in 1959 have found serum liver enzymes elevation in congestive heart failure patients. Killip et al, in 1960, described massive elevations of serum AT levels secondary to cardiogenic shock [20].

Table – 4: Biochemical values in cardiac failure patients.

VARIABLES	GROUP				P VALUE
	STUDY GROUP		CONTROL GROUP		
	MEAN	SD	MEAN	SD	
EF (%)	51.10	7.83	61.44	4.00	<0.01**
Liver Size (cm)	14.53	2.51	12.84	1.43	0.002**
Total Serum Bilirubin (mg/dl)	1.81	1.16	0.88	0.08	<0.01**
Conjugated Bilirubin (mg/dl)	0.18	1.17	0.05	0.06	<0.01**
Unconjugated Bilirubin (mg/dl)	1.63	1.01	0.83	0.07	<0.01**
SGOT (U/l)	113.55	77.68	42.44	21.67	<0.01**
SGPT (U/l)	99.92	64.97	37.48	19.36	<0.01**
SAP (U/l)	128.12	56.34	126.8	52.2	0.920
Serum Protein (g/dl)	6.55	0.53	6.94	0.67	<0.005**
Serum Albumin (g/dl)	3.27	0.56	4.04	0.35	<0.01**
A/G Ratio	1.02	0.26	1.43	0.27	<0.01**
Prothrombin time (sec.)	14.55	4.07	11.24	0.44	<0.01**

Conclusion

The present study undertaken shows significant changes in liver biochemical profiles in patients with congestive heart failure. These changes are found to be useful in assessing the duration and severity of heart failure. The liver performs a diverse array of biochemical, synthetic, and excretory functions, and as a result, no single biochemical test is capable of providing an accurate global assessment of hepatic function. Hence, clinical evaluation, a complete biochemical profile, the underlying cause and radiologic imaging are necessary to interpret the liver function tests. The altered liver functions in heart failure patients are often reversible. The present study suggests an early detection of liver function abnormalities in heart failure patients. Treatment should mainly be focused on the underlying heart disease. Regression in liver derangements occurs after successful treatment of heart failure in most of the cases. Present study could not follow up all sixty cases to assess the regression of liver function abnormalities due to practical concerns.

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