Original Research Article

Clinical evaluation of liver function in congestive heart failure in Cuddalore District

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Abstract

Background: Abnormal liver enzymes and liver function in congestive cardiac failure has long been recognized and occurs quite frequently in acute and chronic failure. Heart failure (HF) is characterized by the inability of systemic perfusion to meet the body's metabolic demands and is usually caused by cardiac pump dysfunction and may occasionally present with symptoms of a non-cardiac disorder such as hepatic dysfunction. The primary pathophysiology involved in hepatic dysfunction from HF is either passive congestion from increased filling pressures or low cardiac output and the consequences of impaired perfusion.

Aim of the study: The present study was undertaken to identify alterations in the liver biochemical profiles in relation to congestive heart failure and also to show their significance with respect to the duration of heart failure.

Materials and methods: Totally Sixty cases with heart failure of various etiologies and twenty healthy controls who got admitted at medicine ward of Sri Rajah Mutaiah Medical College and Hospital in the year 2019 March-August were included in the study. The higher number of cases was found within the age group of 40 to 50 years followed by the age group of 50 to 60 years. Laboratory tests measured only a limited number of these functions. No one test enabled the clinician to accurately assess the liver's total functional capacity. To increase both the sensitivity and specificity of laboratory tests in the detection of liver disease, it is best to use them as a battery.

Results: Present study has found a strong relationship between liver function derangements and heart failure cases. 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 was also elevated, the levels of unconjugated fraction were higher.

Serum aminotransferases were elevated in 78% of cases, unlike serum alkaline phosphatase which was increased only in 25% of cases. There found to be a significant correlation between rise in unconjugated bilirubin and elevation of serum aminotransferases.

Conclusion: Study revealed marked alterations in liver function with acute heart failure and during hypotension when compared to chronic heart failure. Low serum proteins and serum albumin levels are seen in many of the cases. Various factors play in causing these changes which have already been discussed. Serum prothrombin time, though prolonged in 88% of cases, the changes were only mild.

Key words

Liver Function Test, Congestive Heart Failure, Smoking, Alcoholism.

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. 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 [1]. Heart failure (HF) is a clinical syndrome characterized by the inability of systemic perfusion to meet the body's metabolic demands and is usually caused by cardiac pump dysfunction [2]. HF is subdivided into systolic and diastolic HF. Systolic failure presents reduced cardiac contractility whereas diastolic failure exhibits impaired cardiac relaxation with abnormal ventricular filling [3]. HF can result from several structural or functional congenital and acquired cardiac disorders that impair the ability of the ventricle to fill with or eject blood. Clinically, HF may present with a syndrome of decreased exercise tolerance due to dyspnea and/or fatigue-related to impaired cardiac output or may present with a syndrome of fluid retention from elevated filling pressure [4]. A spectrum of hepatic derangements can also occur in HF particularly in the setting of right heart failure (RHF). Any cause of right ventricular dysfunction can be associated with severe hepatic congestion; patients with hepatic congestion are usually asymptomatic and this entity may be suggested only by abnormal liver function tests (LFTs) during routine laboratory analysis [5]. Bridging fibrosis or cardiac cirrhosis can result from prolonged hemodynamic abnormalities, resulting in an impaired hepatic function with impaired coagulation, decreased albumin synthesis, and alteration in the metabolism of several cardiovascular drugs, which can lead to unwanted toxicity [6]. Heart failure, causes a number of pathophysiological effects which, alone or in combination result in liver cell damage. As a consequence, liver function abnormalities are so common in heart failure [7]. Liver dysfunction in heart failure is usually mild and asymptomatic and often detected incidentally on routine liver biochemical investigations [8].

Materials and methods

Totally Sixty cases with heart failure of various etiologies and twenty healthy controls who got admitted at medicine ward of Sri Rajah Mutaiah Medical College and Hospital in the year 2019 March-August were included in the study. The higher number of cases was found within the age group of 40 to 50 years followed by the age group of 50 to 60 years. Laboratory tests measured only a limited number of these functions. No one test enabled the clinician to accurately assess the liver's total functional capacity. To increase both the sensitivity and specificity of laboratory tests in the detection of liver disease, it is best to use them as a battery.

Inclusion criteria: Congestive heart failure in all age groups.

Exclusion criteria:

- History of alcoholism.
- Past history of jaundice.
- Recent intake of hepatotoxic and cholestatic drugs.

- Presence of HBs Antigen and Anti HCVAntibodies.
- Pregnancy.

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. 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 age and gender. They are excluded from diseases which are thought to influence the study by appropriate investigations.

Liver function tests: A large number of tests have been proposed, but many provide similar information.

- Tests used as indices of uptake, conjugation, and excretion of anionic compounds-Serum total bilirubin (and direct and indirect).
- Tests that reflect damage tohepatocytes-Serum aspartate and alanineaminotransferases.
- Tests that reflect cholestasis-Serum alkalinephosphatase.
- Tests that measure biosynthetic functions of the liver (Serum proteins, Serum albumin, Prothrombin time)

Results

Male and female ratio = 1.5: 1 as per **Table – 1**.

Table – 1: Gender distribution in heart failure.

Sex	No. of cases
Male	36
Female	24
Total	60

Etiology of heart failure was as per **Table – 2**. **Table - 3** shows total serum bilirubin level more than 0.9 mg/dl was taken as hyperbilirubinemia according to our study. Serum hyperbilirubinemia was seen in 77% of heart failure patients in the present study which was considered as highly significant (P = < 0.01) in comparison with control cases. The present study found an unusually high total serum bilirubin level in patients presented with hypotension. A significant (P = 0.001) correlation was found in those cases. Bilirubin levels were higher even than in acute heart failure. SGOT level of more than 42 U/l and SGPT level of more than 37 U/l was taken as elevated levels according to our study. The present study revealed 78% cases with elevated liver serum aminotransferases in sixty heart failure patients which were considered as highly significant (P = < 0.01). Serum alkaline phosphatase level more than 127 U/l was taken as higher than normal according to our study. The present study observed elevation of serum alkaline phosphatase in 15 cases (25%) among 60 heart failure cases which are considered to be not significant (P = 0.920). Low serum proteins level was found in 72% of cases and low albumin values were found in 93% of cases with sixty heart failure patients which were considered to be highly significant (P = < 0.01). The ratio of serum albumin to globulin below 1.43 was considered evidence for low A/G ratio according to the present study. The present study observed low A/G ratio in 93% of cases which was highly significant (P = < 0.01). Serum prothrombin time was often prolonged in congestive heart failure. The present study found prolonged prothrombin time in 88% of cases with sixty heart failure patients which were considered highly significant (P = < 0.01).

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 [9]. Clinical jaundice was present in 12 cases among total sixty cases. The present study showed 20% cases with jaundice in heart failure patients. We have found no correlation between the presence of jaundice and the duration of heart failure whereas heart failure patients with hypotensive

episodes showed a strong correlation with clinical jaundice. Jaundice was present in all three cases of heart failure with low blood pressure [10]. Lautt WW, et al. observed only eight (1.2%) cases with jaundice in their retrospective study on jaundice as a presentation of heart failure in 661 cases. All eight patients had severe cardiac dysfunction. He found jaundice due to heart disease tends to be mild and the most common mechanism is hepatic passive congestion. Liver size more than 13 cm by ultrasonography is defined as hepatomegaly in our study. An enlarged liver is seen in 63% of cases which is considered to be highly significant (P = 0.002) in heart failure patients. Total serum bilirubin level more than 0.9 mg/dl is taken as hyperbilirubinemia according to our study. Serum hyperbilirubinemia is seen in 77% of heart failure patients in the present study which is considered as highly significant (P = < 0.01) in comparison with control cases. The maximum level goes up to 5.2 mg/dl, which is seen in two patients [11].

Table – 2: Etiology of heart failure.

Diagnosis	Male	Female	Total	Percentage
RHD	13	11	24	40%
DCM	8	6	14	23.3%
CAHD	7	1	8	13.3%
Cor pulmonale	3	2	5	8.3%
Others	5	4	9	15%
Total	36	24	60	100%

Table - 3: Liver function test analysis between control group and cases.

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**

Note: **P Value ≤ 0.01 = Significant at 1% level, *P Value 0.011 to 0.05 = Significant at 5% level, P Value >0.05 = Not Significant at 5% level

Present study found an unusually high total serum bilirubin level in patients presented with hypotension. A significant (P = 0.001) correlation was found in those cases. Bilirubin

levels are higher even than in acute heart failure. Virtually any cause of shock or hemodynamic instability can result in ischaemic injury to the liver. SGOT level of more than 42 U/l and SGPT level of more than 37 U/l is taken as elevated levels according to our study. The present study revealed 78% cases with elevated liver serum aminotransferases in sixty heart failure patients which are considered as highly significant (P = <0.01) [12]. Moussavian SN, et al. found though both aminotransferases were high in his study on alterations in liver function in congestive heart failure with particular reference to serum enzymes, SGPT levels were less marked than, those of SGOT levels [13]. In the present study, ratio between SGOT and SGPT were found to be not significant in comparison with control cases (P = 0.984). Whereas there is a significant (P = < 0.01) correlation (r = 0.92) between elevation of SGOT and SGPT levels in the study group. Serum alkaline phosphatase level more than 127 U/l is taken as higher than normal according to our study. The present study observed elevation of serum alkaline phosphatase in 15 cases (25%) among 60 heart failure cases which are considered to be not significant (P = 0.920) [14]. In most of the cases the level is less than twofold rise. The present study has taken serum level less than 6.9 g/dl as evidence for decreased serum proteins level and serum level less than 4.0 g/dl as evidence for hypoalbuminemia. Low serum proteins level was found in 72% of cases and low albumin values were found in 93% of cases with sixty heart failure patients which are considered to be highly significant (P = < 0.01). The ratio of serum albumin to globulin below 1.43 is considered evidence for low A/G ratio according to the present study. The present study observed low A/G ratio in 93% of cases which is highly significant (P = < 0.01) [15]. Richman SM, et al. found decreased serum albumin level in 26% of cases. Serum prothrombin time is often prolonged in congestive heart failure. Present study found prolonged prothrombin time in 88% of cases with sixty heart failure patients which are considered highly significant (P = < 0.01). PT more than 11 seconds is taken as abnormal value according to the study [16]. Safran AP, et al. found prolonged PT in 80 to 90% of cases with acute and chronic heart failure. The present study observed there is only a mild elevation in prothrombin time. Caution should, therefore, be exercised when treating patients in heart failure with oral anticoagulants [17].

Conclusion

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. The 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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