

Hepatic Function Tests in Patients with Cardiac Failure.

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ABSTRACT

Background: Heart failure 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 noncardiac disorder such as hepatic dysfunction. **Aim:** To study the association of Liver function tests in patients with heart failure. **Methods:** Prospective cross-sectional study was conducted in Tertiary Medical College Hospital in heart failure patients. Complete biochemical investigations like plasma glucose, blood urea, serum creatinine, serum electrolytes, complete hemogram, liver function tests, urine analysis, x-ray chest, ultrasonography abdomen, ECG and echocardiography. **Results:** 50 heart failure patients were included in this study. In patients with ejection fraction, less than or equal to 40% or greater than 40%, bilirubin value shown statistical significance differences, other parameters are shown statistical insignificance. **Conclusion:** Increased bilirubin was independently associated with decreased ejection fraction in patients with heart failure.

Keywords: heart failure, Liver function test, ejection fraction.

INTRODUCTION

Heart failure is defined as impaired organ perfusion, also known as a forward failure, and high filling pressure and venous congestion, also known as a backward failure. HF is a systemic and chronic disease and as such involves many organs, not least the liver and kidney. The complex vascular system of the liver and its high metabolic activity render it vulnerable to circulation disturbances and trigger many molecular and hemodynamic changes in patients.^[1] A spectrum of hepatic derangements can also occur in heart failure particularly in the setting of right heart failure. 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 during routine laboratory analysis. The primary pathophysiology involved in hepatic dysfunction is either passive congestion from increased filling pressures or low cardiac output and the consequences of impaired perfusion.^[2,3] Abnormalities in liver function tests (LFTs) are not an uncommon finding in patients with HF. These abnormalities are a result of impaired perfusion or elevated right-sided cardiac pressures or are secondary to drug toxicity. Attempts at describing

the features of chronic liver damage secondary to HF have been ongoing since the early 20th century. Nevertheless, neither the pathophysiologic basis underlying these findings nor the clinical impact of impaired liver function on HF outcomes has been delineated.^[4,5]

Aim

To study the association of Liver function tests in patients with heart failure.

MATERIALS AND METHODS

A Prospective cross-sectional study was conducted in Tertiary care Medical College Hospital. Cases of congestive cardiac failure, as per Framingham criteria; of various age groups and etiologies such as Rheumatic valvular Heart Disease, Ischemic Heart Disease, Hypertensive Heart Disease, Congenital Heart Disease, Cardiomyopathies, Cor pulmonale, Congestive cardiac failure of varied presentation either acute or chronic. Patients with a history of jaundice, Presence of hepatitis B surface antigen and anti HCV antibody, History of alcoholism, Pregnancy, Recent intake of cholestatic or hepatotoxic drugs, Hemolytic disorders, Blood transfusion, Infectious hepatitis were excluded from the study. Clinical diagnosis of congestive heart failure was made in patients who met with the inclusion and exclusion criteria. The patient's completed demographic details with complete history were recorded. After hemodynamic stabilization, they were subjected to complete biochemical investigations like plasma glucose,

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blood urea, serum creatinine, serum electrolytes, complete hemogram, liver function tests, urine analysis, x-ray chest, ultrasonography abdomen, ECG and echocardiography.

RESULTS

50 patients with heart failure were included in the study. In this study, among 50 patients, 32 male and 18 female. There is no difference observed in the age group of the patients.

The present study revealed that among the total heart failure patients, 18% cases were dilated cardiomyopathies, 28% cases were rheumatic heart disease, 32% cases were Coronary Artery Heart Disease, 14% cases were cor pulmonale. Heart failure secondary to congenital heart disease is seen in 6% of cases. We have found 13 cases with acute heart failure and 6 cases with hypotension.

In patients with ejection fraction less than or equal 40%, 85% had increased bilirubin, 80% had increased SGOT, 75% had increased SGPT, and 25% had increased ALP. In patients with an ejection fraction of more than 40%, 37% had increase bilirubin, 80% had increased SGOT, 60% had increased SGPT, and 7% had increased ALP. Their significance difference observed in bilirubin value between ejection fraction group. No other parameters are shown significance.

Ejection fraction didn't correlate well with a serum protein, albumin, prothrombin time or liver size in our study.

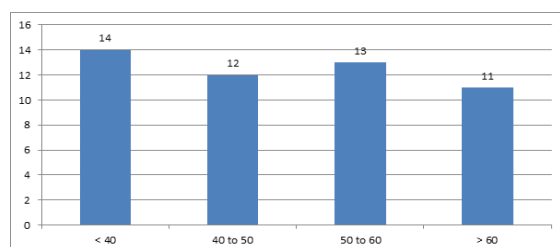


Figure 1: Age Distribution of Study Patients

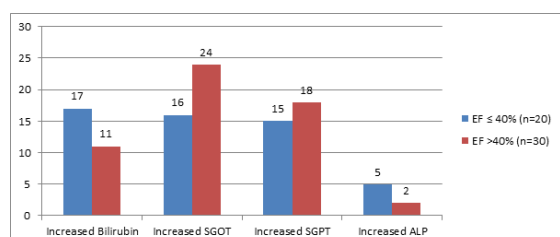


Figure 2: Distribution of Ejection Fraction on Liver Function Test

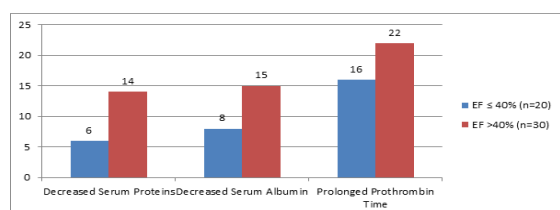


Figure 3: Distribution of Ejection Fraction on other Liver Parameters

DISCUSSION

Elevated transaminases are sensitive markers of liver injury, elevated bilirubin usually indicates cholestasis or extensive heme breakdown, and low albumin reflects impaired liver synthetic capabilities. It is worth noting that the magnitude of transaminases' elevation in different pathologic conditions vary dramatically and the cutoffs for clinical significance are usually arbitrary. We assumed that AST and ALT exceeding 3 times the upper normal limit would define marked alteration of transaminases. Surprisingly, the data on the prevalence, pathophysiology, and clinical significance of abnormalities of each LFT in AHF remain limited.^[6,7] The possible causes of low albumin in heart failure include malnutrition, inflammation, cachexia, hemodilution, liver dysfunction, protein-losing enteropathy, increased transcapillary escape rate, and nephrotic syndrome.^[8,9] Of these; inflammation, cachexia secondary to catabolic/anabolic imbalance, and nutritional deficiencies are probably the most important. One of the reasons behind poor nutrition in heart failure patients is edema of the intestinal wall and resulting poor absorption of oral nutrients. This may respond to appropriate diuresis. Clinically, the validation of elevated bilirubin as a strong independent predictor of outcome has potentially important value for risk stratification of chronic heart failure patients.^[10] Passive hepatic congestion due to increased central venous pressure is believed to cause elevations of both direct and indirect serum bilirubin ('congestive hepatopathy'), with nutmeg liver on pathology. Impaired perfusion from the decreased cardiac output is associated with acute hepatocellular necrosis ('hepatic ischemia') with elevations primarily in serum aminotransferases.^[11] Cardiogenic ischaemic hepatitis ('shock liver') may ensue following an episode of profound hypotension. Bridging fibrosis ('cardiac cirrhosis') can result from prolonged hemodynamic abnormalities, resulting in an impaired hepatic function with impaired coagulation and decreased albumin synthesis.

CONCLUSION

To conclude abnormal liver function tests are common in cardiac failure and may lead to patients with a bad outcome. Increased bilirubin is associated with decreased ejection fraction. Further research into the complex relationship between cardiac and hepatic function in heart failure may improve both understandings of pathophysiology and the clinical care of heart failure patients.

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