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Original Article

Correlation between Ejection Fraction and Hepatic and Renal Functions in Heart Failure Patients

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Abstract

Introduction: Functioning of liver and kidney is highly dependent on cardiac output. Some degree of impairment in the liver and renal functions is normally expected in all case of heart failure. This is highly overlooked in most of the clinical scenarios.

Aim: To study the parallelism between degree of heart failure and impairment of liver and renal function in heart failure patients.

Materials and Methods: The prospective cross-sectional study was conducted in tertiary medical college hospital in heart failure patients. Complete biochemical investigations such as plasma glucose, blood urea, serum creatinine, serum electrolytes, complete hemogram, liver function tests, urine analysis, X-Ray chest, ultrasonography abdomen, electrocardiogram, and echocardiography.

Results: In patients with ejection fraction ≤40-85% had increased bilirubin, 92.5% had increased serum glutamic oxaloacetic transaminase, 92.5% had increased serum glutamic pyruvic transaminase, and 22.5% had increased alkaline phosphatase. In patients with ejection fraction, ≤40-57.5% had increased urea and 62.5% had increased creatinine.

Conclusion: The incidence of liver and renal involvement increased considerably with the degree of heart failure.

Key words: Ejection fraction, Hear failure, Liver function test, Renal function test

INTRODUCTION

Heart failure is a systemic clinical syndrome with a wide range of potential effects on all organ systems in our body like liver and kidney. Specific effects of heart failure on the different body organs depend on whether it occurs on the right or left side. After sometime, in either form of heart failure, the organs in the body will receive more oxygen supply, nutrients or body wastes are removed slowly due to poor cardiac output. Adequate blood supply is necessary for the liver to perform its various functions. As a result of high level of metabolic activity and its complex vascular supply, the liver is particularly vulnerable to a wide spectrum of circulatory disturbances. Heart failure causes a number of pathophysiological effects alone or in combination, resulting in liver damage. Congestive heart failure causes liver damage in 20% of patients and clinically evidence to jaundice. Jaundice correlates with the severity of heart failure as evidenced by pulmonary wedge pressure, right atrial pressure, and cardiac index.[1] However, hyperbilirubinemia is seen in 20-80% of patients with congestive heart failure. The raised serum bilirubin falls quickly with the improvement of heart failure usually becoming normal within 3-7 days. Serum bilirubin level mostly <3 mg/dl and it rarely rises more than 5 mg/dl 19. Extreme elevation of serum aminotransferases and serum bilirubin would suggest extensive hepatic necrosis.[2] Mild renal dysfunction is a common in heart failure patients. It is intimately associated with heart failure even in its earliest stage. Seattle heart failure model uses serum creatinine as a measure of renal function. Worsening of renal function mainly due to chronic venous congestion, hypoperfusion, or increased intra-abdominal pressure (≥8 mm Hg).[3] In heart failure cases, liver and renal dysfunctions are usually mild and asymptomatic. It is often detected incidentally on routine biochemical investigations. This study was

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undertaken particularly to emphasize the importance of early identification of liver and renal biochemical markers in heart failure patients. The renal and liver function tests are found to be a very useful in assessing the severity and duration of heart failure. Early identification and adequate treatment of the underlying cause of heart failure revert renal and liver derangements to normal and prevents permanent damage.

Aim

To study the parallelism between degree of heart failure and impairment of liver and renal functions in heart failure patients.

MATERIALS AND METHODS

A prospective cross-sectional study was conducted in tertiary care medical college hospital. Ethics committee approval and informed consent from the patients recruited were obtained. Cases of congestive cardiac failure, as per Framingham criteria,[4] 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 past history of jaundice, presence of hepatitis B surface antigen and anti-hepatitis C virus antibody, history of alcoholism, pregnancy, recent intake of cholestatic or hepatotoxic drugs, hemolytic disorders, blood transfusion, and 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. Patient's completed demographic details with complete history were recorded. After hemodynamic stabilization, they were subjected to complete biochemical investigations such as plasma glucose, blood urea, serum creatinine, serum electrolytes, complete hemogram, liver function tests, urine analysis, x-ray chest, ultrasonography abdomen, electrocardiogram, and echocardiography.

RESULTS

A total of 100 patients with congestive heart failure fulfilling the inclusion criteria were included in the study. In this study, among 100 total cases, 59 male and 41 female cases were present. The number of male cases was higher than female cases. The higher frequency of cases was found within the age group of <40 years followed by 50-60 years of age group (Table 1).

This study revealed that among the total heart failure patients, 20% cases were dilated cardiomyopathies, 32% cases were rheumatic heart disease, 28% cases were coronary artery heart disease, 12% cases were cor pulmonale. Heart failure secondary to congenital heart disease is seen in 3% of cases. We have found 12 cases with acute heart failure and 5 cases with hypotension.

In patients with ejection fraction ≤40-85% had increased bilirubin, 92.5% had increased serum glutamic oxaloacetic transaminase (SGOT), 92.5% had increased serum glutamic pyruvic transaminase (SGPT), and 22.5% had increased alkaline phosphatase (ALP). In patients with ejection fraction, more than 40-35% had increase bilirubin, 71.7% had increased SGOT, 61.7% had increased SGPT, and 5% had increased ALP.

Ejection fraction did not correlate well with a serum protein, albumin, prothrombin time or liver size in our study (Table 3).

In patients with ejection fraction ≤40-57.5% had increased urea and 62.5% had increased creatinine. In patients with ejection fraction, more than 40-15% had increase urea and 16.7% had increased creatinine (Table 4).

DISCUSSION

Serum hyperbilirubinemia is seen in 55% of heart failure patients in our study. Serum bilirubin level ranges

Table 1: Age distribution of study patients

Age	Number of cases
<40	27
40-50	24
50-60	26
More than 60	23
Total	100

Table 2: Influence of ejection fraction on liver function test

Liver function test	Ejection fraction		*P*
	≤ 40% (*n*=40)	>40% (*n*=60)	
Increased bilirubin	34	21	<0.0001
Increased SGOT	37	43	<0.0001
Increased SGPT	37	37	<0.0001
Increased ALP	9	3	0.008

SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, ALP: Alkaline phosphatase

Table 3: Influence of ejection fraction on liver parameters

Other liver parameters	Ejection fraction		*P*
	≤ 40% (*n*=40)	>40% (*n*=60)	
Decreased serum proteins	13	27	0.21
Decreased serum albumin	15	29	0.28
Prolonged prothrombin time	31	45	0.7

Table 4: Influence of ejection fraction on renal function test

Renal function tests	Ejection fraction		*P*
	≤ 40% (*n*=40)	>40% (*n*=60)	
Increased urea	23	9	<0.0001
Increased creatinine	25	10	<0.0001

between 0.78 and 3.9 mg/dl. Kubo *et al.* study shows the elevation of serum bilirubin level up to 2.5 mg/dl 20. Dunn *et al.* found mild elevation of serum bilirubin in 70% of heart failure patients.[5] Shovman *et al.* described ischemic hepatitis resulting from the acute onset of hypoxia in patients with poor EF.[6] This study also found elevated serum bilirubin level in patients with DCM and hypotension. The serum bilirubin level raises 3-4 folds the normal in ischemic hepatitis.[7] In our study, more than 3-fold rise in serum bilirubin seen with DCM patients. This study shows hepatomegaly in 74% of cases. Liver size ranges from 11 cm to 19 cm. Dunn *et al.* study shows 95% of hepatomegaly in heart failure patients.[5] Giallouratis *et al.* study found aminotransferase elevation is usually 2-3 times above the normal range in congestive heart failure. Degree of AT elevation correlates with hypotension and decrease hepatic perfusion.[8] This study shows 80% cases are associated with elevated SGOT 74% mostly the patients with reduce LV function shows the elevation of SGOT and SGPT levels which is highly significant ($P < 0.0001$). Our study reveals a significant correlation between SGPT and SGOT in heart failure patients. Richman *et al.* found both SGOT and SGPT were elevated in his study with reference to particular serum enzymes.[9] In this study, SGOT elevated in 80% and SGPT elevated in 74% mostly the patients with reduce LV function shows the elevation of SGOT and SGPT levels which is highly significant (Table 2). The SGOT/SGPT ratio is 1.2 in this study. This study revealed elevation of ALP in 12% cases among 100 heart failure cases. ALP ranges between 26 and 75 Sherlock *et al.*[8] and Kubo *et al.*[1] found increased serum alkaline phosphatase by 10-20% cases with heart failure. Richman *et al.* found high intrahepatic pressure may cause intrahepatic biliary obstruction leads to rise in ALP with congestive heart failure cases.[9] Our study revealed low serum proteins were found in 40% of cases, and low albumin values were found in 44% of cases among 100 heart failure patients. This study found low albumin/globulin ratio in 75% of cases. Naresh *et al.* found decreased serum albumin level in 39% of cases with heart failure.[10] This study found low serum albumin level in half of the patients. This low serum albumin level should be interpreted with caution because low albumin values were associated with reduced dietary intake of proteins, increased gastrointestinal loss or renal loss, altered vascular permeability, increased catabolism and over hydration. So changes in serum albumin level are not specific for liver disease. Our study revealed prolonged prothrombin time in 76% of cases among 100 heart failure patients, which is considered to be significant. White *et al.* observed prolonged PT in 80% of cases with acute and chronic heart failure.[11] Giallourakis *et al.*, 2002, study shows PT prolongation in 80% of cases with congestive heart failure. This is due to decreased synthesis of clotting factors II, V, VII, IX and X by liver.[8] Forman *et al.*, 2004, study found 27% cases associated with renal dysfunction. The renal dysfunction defined as elevated creatinine which correlates with left ventricular systolic function. Blood urea elevation is seen in 19.4% of patients. He found worsening of renal dysfunction in patients with reduced left ventricular ejection fraction (LVEF).[12] In our study also worsening of renal function present in 1/3rd of patients. Rusinaru *et al.* found renal dysfunction in 14% of cases with heart failure patients (LVEF more than or equal to 50). He described renal dysfunction due to heart failure is caused by intrinsic nephropathy, poor renal perfusion, vasoconstriction, and renal venous congestion.[13]

CONCLUSION

The incidence of liver and renal involvement increased considerably with the degree of heart failure. An early and prompt treatment of heart failure is necessary to prevent added morbidities caused by liver and renal involvement. Hence, the liver and renal function tests are warranted for congestive heart failure patients.

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