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Original Research

Hepatic parameters in congestive heart failure patients: A prospective study

Sunil Kumar Goel

Associate Professor, Department of General Medicine, Noida International Institute of Medical Sciences, Greater Noida, India

ABSTRACT:

Aim: To study the hepatic parameters in congestive heart failure patients. **Methods:** The prospective analytical study was conducted in the Department of General Medicine. Patients with heart failure all age and both sexes were included in this study. 100 patients with heart failure were included in this study. Various demographic parameters like age sex duration of disease were recorded on predesigned Performa. The hepatic biochemical parameters like serum bilirubin (direct, indirect and total), serum AST and ALT, Serum alkaline phosphatase, Serum proteins and Prothrombin time were estimated. **Results:** Mean age of patient was 57.87±9.69 years. Number of patients below than 30 years was 6 (6%), from 30 to 50 years were 17(17%). Maximum number of patients was from above 50 years of age that is 77 (77%). There was male predominance (79/21). As per NYSA classification maximum number of cases were class II (40%) followed by class III (30%). Percentage of patients with class I were 20% and class IV were 10%. Serum bilirubin was 3.65±1.74 mg/dl in class IV and least in class I that is 1.16±0.54 mg/dl. Serum AST was highest in class IV 159.24±21.26 IU and least in class I that is 38.88±10.87 IU (p=0.001). Serum ALT was highest in class IV 187.39±31.98 IU and least in class I that is 34.24±10.69 (p=0.001). Serum ALP was highest in class IV 59.89±14.59 IU and least in class I that is 40.27±8.69 (p=0.04). Serum total protein (g/dl) was decreased as the heart failure progressed least in class IV 3.69±1.84 g/dl and highest in class I that is 6.88.10±1.58 gm/dl (p=0.04). Serum albumin (g/dl) was least in class IV 2.86±0.85 g/dl and highest in class I that is 3.55±0.89 gm/dl (p=0.03). Prothrombin time (sec) was highest in class IV 21.65±5.85 sec and least in class I that is 11.88±3.78 sec (p=0.01). **Conclusion**: We conclude that Congested hepatomegaly was common presentation jaundice and ascites was also common. Change in biochemical parameters was increased with severity and duration of heart disease.

Keywords: Hepatic parameters, congestive heart failure

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Corresponding author: Sunil Kumar Goel, Associate Professor, Department of General Medicine, Noida International Institute of Medical Sciences, Greater Noida, India

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INTRODUCTION

Heart failure (HF) is a major public health problem, with frequent hospitalizations, impaired quality of life, and shortened life expectancy.[1] 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. HF can result from several structural or functional congenital and acquired cardiac disorders that impairs the ability of the ventricle to fill with or eject blood.[1] 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.[2] 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. 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. Passive hepatic congestion due to increased central venous pressure (CVP) may cause elevations of liver enzymes and both direct and indirect serum bilirubin. Impaired

perfusion from decreased cardiac output may be associated with acute hepatocellular necrosis with marked elevations in serum aminotransferases. Cardiogenic ischemic hepatitis ("shock liver") may ensue following an episode of profound hypotension in patients with acute HF.

Hepatic dysfunction due to passive congestion is particularly common in patients with right-sided HF with elevated right ventricular (RV) pressure. Any cause of right-sided HF can result in hepatic congestion, including constrictive pericarditis, severe pulmonary arterial hypertension (PAH), mitral stenosis, tricuspid regurgitation (TR), cor pulmonale, cardiomyopathy, and as a postoperative consequence of the Fontan procedure for pulmonary atresia and the hypoplastic left heart syndrome. TR is particularly prone to result in passive congestion because pressure from the RV is transmitted directly to the hepatic veins and sinusoids.[3] This increase in venous pressure caused by RV dysfunction leads to atrophy of the hepatocytes and causes perisinusoidal edema that can impair diffusion of oxygen and nutrients to the hepatocytes.[4,5] As a result from this hepatic congestion, mild jaundice, abnormalities in liver enzymes, and derangements in hepatic drug metabolisms ensues. On gross examination the congestive liver is enlarged, with a purple or reddish hue with prominent hepatic veins. The cut surface shows the classic nutmeg appearance, reflecting the alternating pattern of hemorrhage and necrosis of zone 3 with the normal or slightly steatotic areas in zones 1 and 2. Microscopically, the hallmark features of hepatic venous hypertension are prominence of the central veins, central vein hemorrhage, and sinusoidal engorgement.[3,6,7] Untreated, long-standing congestion can lead to cardiac fibrosis and, ultimately cardiac cirrhosis.[8] In contrast, low cardiac output (forward failure) is associated with some degree of perfusion abnormality that is not necessarily evident. Acute hypoxic hepatitis most commonly arises in the context of profound systemic hypotension from acute cardiopulmonary collapse after myocardial infarction, exacerbation of HF, or pulmonary embolism. In the absence of established hypotension ischemic hepatitis has been shown in instances of severe hypoxemia, such as obstructive sleep apnea, respiratory failure, and in conditions of increased metabolic demand, as seen in toxic/septic shock.[9,10]

MATERIAL AND METHODS

The prospective analytical study was conducted in the Department of General Medicine, after taking the approval of the protocol review committee and institutional ethics committee. 100 Patients with heart failure all age and both sexes were included in this study. patient with Pre-existing hepatic disorder, Use of hepatotoxic drug and Chronic alcoholic were excluded from the study.

All patients enrolled for this study was evaluated clinically and echocardiographically. Various demographic parameters like age sex duration of disease were recorded on predesigned Performa. The hepatic biochemical parameters like serum bilirubin (direct, indirect and total), serum AST and ALT, Serum alkaline phosphatase, Serum proteins and Prothrombin time were estimated. For estimation of above parameters ebra EM 200 biochemistry analyser was used. All parameters were compared based on NYSA classification and duration of disease.[11,12]

Data were recorded in excel sheet and statistical Analysis was done with software SPSS-22.0 version. Qualitative data were calculated as percentage and proportions and were analysed by chi-square test. Quantitative data were expressed as mean ± SD and these data were analysed by unpaired student t test. The p value less than 0.05 were taken as significant.

RESULTS

In present study 100 patients with various class and duration of heart failure were enrolled for this study for evaluation of changes in hepatic parameters.

Table 1: Demographic profile of the patients

Parameters		Number	Percentage (%)
	Less than 30	6	6
Age (mean	30 to 50	17	17
57.87±9.69 year)	Above 50	77	77
	M	79	79
Sex	F	21	21
	Class I	20	20
	Class II	40	40
NYSA class	Class III	30	30
	Class IV	10	10
Duration of	Less than 1 year	12	12
disease	1 to 5	67	67
	More than 5	21	21

In our study as per table 1 mean age of patient was 57.87±9.69 years. Number of patients below than 30 years was 6 (6%), from 30 to 50 years were 17(17%). Maximum number of patients was from above 50 years of age

that is 77 (77%). There was male predominance (79/21). As per NYSA classification maximum number of cases were class II (40%) followed by class III (30%). Percentage of patients with class I were 20% and class IV were 10%.

Regarding duration of disease 11% patients have disease since less than one year. Maximum number of patients has disease from to 5-year duration that is 69%. Duration of disease was more than 5 year in 20% patients.

Table 2: Clinical presentation of patients with heart failure

Clinical parameters	N =100	Percentage (%)
Jaundice	25	25
Hepatomegaly	47	47
Ascites	28	28
Congested hepatomegaly in USG	41	41

Regarding clinical presentation of patient's jaundice was present in 25%, hepatomegaly which was most commonly present that was 47%, ascites was present in 28% and congested hepatomegaly in USG (41%).

Regarding hepatic biochemical parameters there is significant variation in serum bilirubin (mg/dl) parameter as per progress in class of heart failure (p=0.001). Serum bilirubin was 3.65±1.74 mg/dl in class IV and least in class I that is 1.16±0.54 mg/dl. Serum AST was highest in class IV 159.24±21.26 IU and least in class I that is 38.88±10.87 IU (p=0.001). Serum ALT was highest in class IV 187.39±31.98 IU and least in class I that is 34.24±10.69 (p=0.001). Serum ALP was highest in class IV 59.89±14.59 IU and least in class I that is 40.27±8.69 (p=0.04). Serum total protein (g/dl) was decreased as the heart failure progressed least in class IV 3.69±1.84 g/dl and highest in class I that is 6.88.10±1.58 gm/dl (p=0.04). Serum albumin (g/dl) was least in class IV 2.86±0.85 g/dl and highest in class I that is 3.55±0.89 gm/dl (p=0.03). Prothrombin time (sec) was highest in class IV 21.65±5.85 sec and least in class I that is 11.88±3.78 sec (p=0.01).

Table 3: Liver biochemical parameters of patients in comparison with class of heart failure

Parameters	Class I	Class II	Class III	Class IV	P value
Serum bilirubin (mg/dl)	1.16±0.54	1.87±0.87	2.87±0.61	3.65±1.74	0.001
Serum AST IU	38.88±10.87	50.22±19.45	85±14.42	159.24±21.26	0.001
Serum ALT IU	34.24±10.69	45.87±9.39	84.48±11.44	187.39±31.98	0.0001
Serum ALP IU	40.27±8.69	45.88±10.98	52.88±11.47	59.89±14.59	0.04
Serum total protein (g/dl)	6.88 ±1.58	5.69±2.22	5.84±2.58	3.69±1.84	0.04
Serum albumin (g/dl)	3.55±0.89	3.28±0.77	3.21±0.51	2.86±0.85	0.03
Prothrombin time (sec)	11.88±3.78	13.33±8.98	18.38±4.29	21.65±5.85	0.01

Table 4: Liver biochemical parameters of patients in comparison with duration of heart failure

Parameters	Variable			P value
	less than 1 year	1 to 5 years	more than 5 years	
Serum bilirubin (mg/dl)	1.19±0.4	1.97±0.74	3.12±1.87	0.04
Serum AST IU	40.33±10.69	49.12±5.36	117±21.29	0.001
Serum ALT IU	40.35±8.77	77.12± 7.36	159.88±22.78	0.001
Serum ALP IU	40.12±5.77	46.18±12.14	61.46 ±10.03	0.04
Serum total protein (g/dl)	6.97±1.59	5.88±1.59	3.87±0.87	0.03
Serum albumin (g/dl)	3.97±0.59	3.21±1.09	2.67±1.44	0.15
Prothrombin time (sec)	13.87±2.29	14.86±4.09	20.16±4.03	0.01

Regarding comparison of liver biochemical parameters in patients with duration of heart failure as per table 4 it is clear that serum bilirubin was increased with the duration of disease. The mean value of serum bilirubin (mg/dl) in patients with duration of disease more than 5 year was 3.12±1.87mg/dl was significantly higher than the patients with duration of disease less than 5 year significantly (p=0.04). Serum AST was highest with duration of disease more than 5 year 117±21.29 IU and least in patients with duration of disease less than 5 year that is 40.33±10.69 IU (p=0.001). Serum ALT was highest with duration of disease more than 5 year 159.88±22.78 IU and least in patients with duration of disease less than 5 year that is 40.35±8.77 IU (p=0.001). Serum ALP IU was highest with duration of disease more than 5 year 61.46 ±10.03 IU and least in patients with duration of disease less than 5 year that is 40.12±5.77 IU (p=0.001). Serum total protein (g/dl) was least with duration of disease more than 5 year 3.87±0.87 g/dl and normal in patients with duration of disease less than 5 year that is 6.97±1.59 g/dl (p=0.03). Serum albumin (g/dl) was least with duration of disease more than 5 year 2.67±1.44 g/dl

and normal in patients with duration of disease less than 5 year that is 3.97±0.59 g/dl (p=0.15). Prothrombin time (sec) was highest with duration of disease more than 5 year 20.16±4.03 sec and least in patients with duration of disease less than 5 year that is 13.87±2.29 sec (p=0.01).

DISCUSSION

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 haemodynamic changes in patients.

Heart failure as a cause of acute liver failure is less documented and poorly understood condition. Auer et al have concluded that hepatic enzymes are elevated in heart failure patients. Pattern of change in hepatic enzyme differ as per in patients with chronic and acute decompensate HF and are surrogates of the type of hemodynamic alterations.[13,14] Shah et al has concluded that hepatic injury as a consequence of heart failure is common but less recognized syndrome.[15]

In our study as per table 1 mean age of patient was 57.87±9.69 years. Number of patients below than 30 years was 6 (6%), from 30 to 50 years were 17(17%). Maximum number of patients was from above 50 years of age that is 77 (77%). This finding is supported by Van Deursen et al.[16] There was male predominance (79/21). As per NYSA classification maximum number of cases were class II (40%) followed by class III (30%). Percentage of patients with class I were 20% and class IV were 10%. This corroborates with the work of Allen et al.[17]

We have observed that hepatic biochemical parameters were significantly elevated in patients with higher class of heart failure than class I. Serum total protein (g/dl) and albumin was significantly decreased in class III and class IV patients in comparison to class I and class II. Alvarez has concluded that may cause elevations of liver enzymes and both direct and indirect serum bilirubin and marked elevations in serum aminotransferases which support our study.[18] Nikolaou et al has concluded that Abnormal LFTs were present in about a half of patients presenting with heart failure which corroborates with our finding.[19] [2]Samsky et al has reported that severity of hepatic damage increases with duration of disease which supports our study.[20] Naschitz et al has concluded that the spectrum of heart diseases affecting the liver includes mild alterations of liver function tests in heart failure, cardiogenic ischemic hepatitis, congestive liver fibrosis, and cardiac cirrhosis which progress with the progress of disease which support our study. has reported that liver function abnormalities remain common in patients with congestive heart failure but are generally small in magnitude and not associated with clinically apparent hepatic disease which contradict our study.[21,22]

CONCLUSION

We conclude that Congested hepatomegaly was common presentation jaundice and ascites was also common. Change in biochemical parameters was increased with severity and duration of heart disease.

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