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Original Research Article

A prospective evaluation of hepatic parameters in congestive heart failure patients

C. Sri Krishna Appaji, P. Ravinder*

Department of General Medicine, Konaseema Institute of Medical Sciences and Research Foundation, Amalapuram, Andhra Pradesh, India

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***Correspondence:**
Dr. P. Ravinder,
E-mail: p_ravinder @gmail.com

ABSTRACT

Background: Heart failure is heterogeneous and complex clinical syndrome. Passive congestion of liver is associated with increase in direct and indirect serum bilirubin and elevated liver enzyme. Impaired perfusion due to decreased cardiac output is responsible for acute hepatocellular necrosis and serum aminotransferases will be elevated. Present study has been designed to study the prevalence of liver function abnormalities in heart failure patients, pattern of elevation of liver enzymes and correlation of liver function tests with etiology, duration and of heart failure.
Methods: 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.
Results: Serum bilirubin was 3.76±1.62 mg/dl in class IV and least in class I that is 1.125±0.30 mg/dl. Serum AST was highest in class IV 154.64±24.96 IU and least in class I that is 36.21±12.65 IU (p=0.001). Serum ALT was highest in class IV 192.24±34.44 IU and least in class I that is 33.34±11.460 (p=0.001).
Conclusions: From present study we can conclude that heart failure was common in fifth and sixth decade of life and there was male predominance. 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: Heart failure, Hepatic parameters, Liver enzymes

INTRODUCTION

Heart failure is heterogeneous and complex clinical syndrome. It can be defined as a complex clinical syndrome that result from structural or functional impairment of ventricular filling of ejection of blood which in turn leads to the cardinal clinical syndrome of dyspnoea and fatigue and signs of heart failure.[1,2] According to resent data current worldwide prevalence of is estimated at 64.34 million cases. It is expected that by 2030 there will be 8 million patients alone in US which accounts for 46% increase in prevalence.[3,4] Felder et al has concluded in his paper in circulation in 1950 that there is definite impairment of liver function in congestive heart failure. The exact basis for this change remains unknown. The pathophysiology of hepatic dysfunction due to heart failure include passive congestion due to increased filling pressures or low cardiac output and impaired perfusion from decreased cardiac output.[6,7] Passive congestion of liver is associated with increase in direct and indirect serum bilirubin and elevated liver enzyme. Impaired perfusion due to decreased cardiac output is responsible for acute hepatocellular necrosis and serum aminotransferases will be elevated.[8-10] But some time liver dysfunction may be the cause of heart failure like, cirrhosis of liver leading to Cirrhotic cardiomyopathy, non-alcoholic fatty liver disease is associated with coronary artery disease and structural damage to heart leads to left ventricular diastolic dysfunction, which represents a key contributor to the development of heart failure.[11-14]

Fouad et al has concluded that heart failure is associated with manifestations of liver failure and laboratory data specific to ischemic hepatitis or congestive hepatopathy.[15] Auer et al has reported that elevated liver enzymes are common in patients with HF.[16] Saner et al has concluded that congestive heart failure should always be considered as a possible cause of acute liver failure.[17]

From above discussed literature it is clear that hepatic abnormalities are associated with heart failure. With this view present study has been designed to study the prevalence of liver function abnormalities in heart failure patients, pattern of elevation of liver enzymes and correlation of liver function tests with etiology, duration and of heart failure.

METHODS

This is a prospective, cross sectional and analytic study conducted in the department of general medicine and cardiology Konaseema institute of medical science Amalapuram India from March 2017 to April 2020.

Present study is approved by the institutional ethics committee. Written informed consent was obtained from all patients or relatives of patients before enrolling them for study.

Selection of patients during the study period clinically and echocardiographically diagnosed cases of heart failure was enrolled for this study based on following inclusion and exclusion criteria.

Inclusion criteria included patients with heart failure all age and both sexes.

Exclusion criteria excluded pre-existing hepatic disorder, Use of hepatotoxic drug, Chronic alcoholic

Sample size based on exclusion and inclusion criteria 60 patients with heart failure were enrolled for this 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.[18,19]

Data were recorded in excel sheet and statistical Analysis was done with software SPSS-14 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 60 patients with various class and duration of heart failure were enrolled for this study for evaluation of changes in hepatic parameters.

Table 1: Demography of patients with heart failure.

Variables		Number	Percentage (%)
Age (mean 58.32±12.65 year)	Less than 30	2	3.33
	31 to 50	10	16.67
	51 to 70	34	56.6
	More than 70	14	23.33
Sex	M	46	76.66
	F	14	23.33
NYSA class	Class I	12	20
	Class II	28	46.67
	Class III	16	26.67
	Class IV	8	13.34
Duration of disease	Less than 1 year	6	10
	1 to 5	42	70
	More than 5	12	20

In our study as per table 1 mean age of patient was 58.32±12.65 years. Number of patients less than 30 years was 2 (3.33%), from 31 to 50 years were 10 (16.67%). Maximum number of patients was from 51 to 70 years of age that is 34 (56.6%). Number of patients above 70 years of age was 14 (76.66%). There was male predominance (46/14). As per NYSA classification maximum number of cases were class II (46.67%) followed by class III (26.67%). Percentage of patients with class I were 20% and class IV were 13.34%.

Regarding duration of disease 10% patients have disease since less than one year. Maximum number of patients has disease from to 5-year duration that is 70%. Duration of disease was more than 5 year in 20% patients.

Table 2: Clinical presentation of patients with heart failure.

Clinical variables	N (n=60)	Percentage (%)
Jaundice	16	26.67
Hepatomegaly	28	46.67
Ascites	18	30
Congested hepatomegaly in USG	24	40

Regarding clinical presentation of patient's jaundice was present in 26.67%, hepatomegaly which was most commonly present that was 40%, ascites was present in 30% and congested hepatomegaly in USG (40%).

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 3.76±1.62 mg/dl in class IV and least in class I that is 1.125±0.30 mg/dl. Serum AST was highest in class IV 154.64±24.96 IU and least in class I that is 36.21±12.65 IU (p=0.001). Serum ALT was highest in class IV 192.24±34.44 IU and least in class I that is 33.34±11.460 (p=0.001). Serum ALP was highest in class IV 60.23±16.45 IU and least in class I that is 41.12±9.35 (p=0.02). Serum total protein (g/dl) was decreased as the heart failure progressed least in class IV 3.74±1.54 g/dl and highest in class I that is 6.47.12±1.98 gm/dl (p=0.04). Serum albumin (g/dl) was least in class IV 2.89±0.79 g/dl and highest in class I that is 2.89±0.79 gm/dl (p=0.034). Prothrombin time (sec) was highest in class IV 22.67±6.32 sec and least in class I that is 12.54±3.21 sec (p=0.01).

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

Variable	Class I	Class II	Class III	Class IV	P value
Serum bilirubin (mg/dl)	1.125±0.30	1.70±0.58	2.14±0.66	3.76±1.62	0.001
Serum AST IU	36.21±12.65	49.42±21.45	87.25±14.45	154.64±24.96	0.001
Serum ALT IU	33.34±11.46	46.22±10.21	86.38±12.44	192.24±34.44	0.0001
Serum ALP IU	41.12±9.35	44.21±11.56	54.78±12.47	60.23±16.45	0.02
Serum total protein (g/dl)	6.47.12±1.98	5.45±2.05	5.24±2.14	3.74±1.54	0.04
Serum albumin (g/dl)	2.89±0.79	3.14±0.86	3.01±0.45	2.89±0.79	0.034
Prothrombin time (sec)	12.54±3.21	14.23±9.12	17.23±4.35	22.67±6.32	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)	1.09±0.4	1.98±0.56	2.97±1.25	0.02
Serum AST IU	39.12±9.12	48.11±6.14	114 ±24.54	0.001
Serum ALT IU	32.45± 6.24	79.65± 8.45	164.45± 25.12	0.000
Serum ALP IU	39.12± 5.45	44.85±10.14	59.42±9.85	0.04
Serum total protein (g/dl)	6.59±1.45	5.94±1.89	4.69±2.45	0.034
Serum albumin (g/dl)	3.65±0.98	3.01±1.22	2.86±1.66	0.12
Prothrombin time (sec)	14.21±2.54	15.76±3.45	19.54±3.44	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 2.97±1.25 mg/dl was significantly higher than the patients with duration of disease less than 5 year significantly (p=0.02). Serum AST was highest with duration of disease more than 5 year 114±24.54 IU and least in patients with duration of disease less than 5 year that is 39.12±9.12 IU (p=0.001). Serum ALT was highest with duration of disease more than 5 year 164.45±25.12 IU and least in patients with duration of disease less than 5 year that is 39.12±9.12 IU (p=0.001). Serum ALP IU was highest with duration of disease more than 5 year 59.42±9.85 IU and least in patients with duration of disease less than 5 year that is 39.12±5.45 IU (p=0.001). Serum total protein (g/dl) was least with duration of disease more than 5 year 3.65±0.98 g/dl and normal in patients with duration of disease less than 5 year that is 6.59±1.45 g/dl (p=0.034). Serum albumin (g/dl) was least with duration of disease more than 5 year 2.86±1.66 g/dl and normal in patients with duration of disease less than 5 year that is 3.65±0.98 g/dl (p=0.12). Prothrombin time (sec) was highest with duration of disease more than 5 year 19.54±3.44 sec and least in patients with duration of disease less than 5 year that is 14.21±2.54 sec (p=0.01).

DISCUSSION

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.[16,17] Shah et al has concluded that hepatic injury as a consequence of heart failure is common but less recognized syndrome.[20]

In present study we have observed that mean age of patient was 58.32±12.65 years and maximum number of patients was from 51 to 70 years of age. There was male predominance. This finding is supported by Van Deursen et al.[21] Most of the patients were in class III and class IV group and duration of disease was from 1 to 5 years. This corroborates with the work of Allen et al.[9]

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.[7] 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.[22] Samsky et al has reported that severity of hepatic damage increases with duration of disease which supports our study.[23] 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.[25]

CONCLUSION

From present study we can conclude that heart failure was common in fifth and sixth decade of life and there was male predominance. 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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