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Original Research Article

A Study on Liver Function Abnormalities in Congestive Cardiac Failure in Government General Hospital, Siddipet

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Abstract:

Aim of the Study: To study the liver function tests in congestive cardiac failure and the relationship between liver function test and remission and exacerbation of congestive cardiac failure.

Material and Methods: All cases of congestive cardiac failure (100), of varied etiologies observed in patients from July 2020 to December 2021. This study is an observational study, comparing the liver functions between cases (various causes of heart failure) and between cases and controls.

Results: 24 patients with rheumatic heart disease,17 patients showed abnormal liver function (72%), Of the 18 patients with cor pulmonale 12 showed abnormal liver function (69%). In 6 patients with hypertensive heart disease 3 showed abnormal liver function (57%), whereas in 38 patients with coronary artery heart disease, 27 showed abnormal liver function (73%) 10 out of 14 patients with cardiomyopathy showed abnormal liver function.

Conclusion: Liver function abnormalities were mostly present in patients with coronary artery disease (73%) and rheumatic valvular heart disease (70%) developing heart failure.

Keywords: Liver Function Tests; Congestive Cardiac Failure.

Introduction

The liver has been called the custodian of milieu interior1. So any liver disorders will have far reaching consequences on body's homeostasis. Also, numerous pathologies of other systems can affect liver.

Both acute and chronic heart failure may result in abnormalities of liver. Liver receives 25% of cardiac output that a fall in cardiac output will result in hepatic hypoperfusion. Liver has the capacity to withstand changes in blood flow by vasoactive mechanisms and oxygen extraction from blood. However when critical levels are reached, hepatic injury ensues [2].

Both Right and left sided heart failure can result in liver injury. In right-sided heart failure, elevation of right heart pressure resulting in raised pressure in hepatic sinusoids, hepatic congestion and liver cell hypoxia. In left sided heart failure, decreased cardiac output results in hepatic hypo perfusion and hypoxia. The common pathway is centrilobular hepatocellular necrosis. Zone 3 of the liver lobule is most vulnerable to hypoxic injury due organization of hepatic blood flow [3,4,5].

In this study, the effects of congestive cardiac failure on liver and its function is analyzed in 100 patients compared with 25 healthy individuals. Various etiologies of congestive cardiac failure have been included and compared, based on their effects on liver functions [6,7,8,9].

Remissions and exacerbations have been tracked on the 7th day and the variations of liver function have been recorded and an attempt has been made to find whether liver function tests can be used as prognostic indicators of congestive cardiac failure [10,11]. Understanding anatomy and functions of liver, liver enzymes, causes and various forms of congestive cardiac failure and their manifestations, pathology of liver in heart failure are needed before evaluating abnormalities of liver function tests in congestive heart failure [12,13].

Aims of the Study

1. To observe any differences in liver function tests with the etiology of congestive cardiac failure.
2. To study the relationship between liver function test and remission and exacerbation of

congestive cardiac failure.

3. To study whether liver function tests can be used as a prognostic indicator in cases of congestive cardiac failure.

Material and Methods

All cases of congestive cardiac failure (100), of varied etiologies observed in patients. 20 healthy individuals were taken as controls. Liver function tests are performed to both controls and cases, serum bilirubin, Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Serum Amino phosphate (SAP), Serum proteins and Prothrombin time admitted at Government Hospital, Siddipet during the period from July 2020 to December 2021.

Inclusion Criteria

1. Rheumatic valvular heart disease
2. Ischemic Heart Disease
3. Hypertensive Heart Disease
4. Congenital Heart Disease46
5. Cardiomyopathies
6. Corpulmonale

Exclusion Criteria

1. Known alcoholic.
2. Past History of jaundice.
3. Recent intake of Hepatotoxic drugs or drugs causing raised liver parameters, such as Rifampicin, INH, Steroids, chlorpromazine, amiodarone, statins, hydralazine, phenytoin and valproate.
4. Positive viral markers.

Results

Table 1: Duration of illness

Years of Heart failure	No of cases	percentage
< 1yr	5	5%
1-5 yrs	44	44%
>5 yrs	50	50%

Table 2: Etiology of heart failure

Etiology	No of cases	Percentage
Rheumatic heart disease	24	24%
Coronary heart disease	38	38%
Cor pulmonale	18	18%
cardiomyopathy	14	14%
Hypertensive heart disease	6	6%

Table 3: Abnormal liver function tests as per Etiology

Etiology	No. of cases	No. of cases with abnormal liver function tests	Percentage
Rheumatic heart disease	24	17	70%
Coronary Atherosclerotic heart disease	38	27	73%
Cor pulmonale	18	12	66%
Cardiomyopathy	14	10	70%
Hypertensive heart disease	6	3	50%

Table 4: Serum Bilirubin with remission and exacerbation

No of cases with abnormal LFT	73 (On follow up, 50 showed remission; 12 showed exacerbation)
No of cases showing exacerbation	12
No of cases showing increase of Serum bilirubin on exacerbation	4-6- no change
Percentage	68%
No of cases showing remission	50
No of cases showing reduction of Serum biliruubin on remission	44; 6- showed increase
Percentage	93.6%
Controls	Serum bilirubin were normal in all the controls
p- Value is significant – 0.0212.	

Table 5: Liver function tests

S No	Test	Normal Range	Results		
			Range	No of patients	Percentage
			< 1.2	22	29.3%
1	Serum		1.2-3 mg/dl	43	57.3%
			3-5 mg/dl	7	9.3%
			>5 mg/dl	3	4%
2	AST	Up to 40 I.U	Normal range	36	48%
			Increased	39	52%
3	ALT	Up to 35 I.U	Normal range	44	58.6%
			Increased	31	41.3%
4	S.A.P.	3 – 13 KA Units	Normal range	65	86.6%
			Increased	10	13.3%
5	Serum albumin	> 3g%	Normal	57	76%
			Reduced	15	20%
			A:G reversal	3	4%
6	Prothrombin time	Control (12-14 sec), Test abnormal if 1 ½ times greater than control	Normal	33	44%
			Prolonged	42	56%
In controls, no abnormality of LFT was noted. 62					

Discussion

In this study, the clinical features and liver function test in congestive cardiac failure due to various etiologies in 100 patients have been compared within and with 20 controls and correlated with various studies in an effort to compare Indian scenario with global picture.

Hepatomegaly was seen in 68 patients out of 100 (68%). The liver enlargement varies from 1cm to 10cm below the RCM in 26% of cases. White et al14 (1956) found hepatomegaly in 95% of their cases of congestive cardiac failure and Sinha et al [15] (1960) found hepatic enlargement in 25.5% of cases. Dunn et al [16] (1973) have also described hepatomegaly in 95% of cases. Richman et al [17] (1961) have described hepatomegaly more than 5cm in as many as 50% of patients.

None of the controls did have hepatomegaly. Icterus was present in 16 (21%) cases. Alcohol induced jaundice and jaundice due to hepatotoxic drugs were ruled out by relevant history taking. Viral hepatitis was ruled out by serological tests for viral markers. Rheumatic heart disease produced the most number of cases with clinically detectable jaundice. 63 Icterus was least present in patients with Cor pulmonale and hypertensive heart disease. None of the controls had icterus. White et al have reported clinically apparent jaundice in 20% of cases. Gravin et al [18] and Kubo et al [19] have also described clinical jaundice in less than 20% of cases.

Hyperbilirubinemia was detected in 73 out of 100 cases. The etiologies most associated with hyperbilirubinemia were coronary artery heart disease (73%) and rheumatic valvular heart disease (70%).In majority of cases serum bilirubin did not exceed 3mg/dl.Kubo et al have reported that serum bilirubin is increased in 20 to 80% of patients with congestive cardiac failure; it rarely exceeds 5mg/d land is usually less than 3mg/dl. Zieve et al [20] has reported that unconjugated bilirubin is usually higher than conjugated bilirubin.

Sheila Sherlock et al [21] and Richman et al have also reported that levels usually range between 1mg/dl and 5mg/dl with the unconjugated form constituting the major fraction. Sherlock et al has reported that only rarely have levels exceeded 20 mg/dl in patients with severe right-sided heart failure.

Richman et al has observed that with improvement of the right-sided heart failure elevated serum bilirubin levels return back to normal quite rapidly over a period of 3-7 days.

S. No	Authors	% of cases with hyper bilirubinemia
1	Felder et al [22]	52%
2	Sherlock et al	68%
3	Evans et al	26%
4	White et al	40%
5	Wahi et al	45%
6	Naresh bhu et al	58%
7	Richman et al	31%
8	This study	68%

Marked increase in serum bilirubin was observed in rheumatic valvular heart disease in this study. This correlates with Sherlock's observation that the deep icterus has a correlation with valvular diseases of heart. The severity of failure and duration of failure correlate well with the elevation in serum bilirubin level. The elevated bilirubin level was less than 3mg/dl in 43 cases and more than 5mg/dlin 3 cases who had severe congestive cardiac failure. With remission of congestive cardiac failure, the serum bilirubin returned to normal in 47 cases which correlates with Richman et al.

In this study 71% of patients showed an abnormal increase in serum bilirubin levels of which 81% showed mild rise of bilirubin between 1-5mg/dl; which correlates with Kubo's et al observation.

Richman et al, Dunn et al and Sherlock et al have reported that elevation in serum aminotransferase levels are seen in 3-50% of patients with right sided heart failure. The wide range in incidence reflects the fact that elevations are seen more commonly in acute congestive heart failure (15-48%) than in chronic failure (3-5%).

Richman has reported that asparate transaminase levels are typically more marked than alanine transaminase levels, the former values ranging from 40-801I.U.This degree of marked elevation is seen in acute heart failure secondary to cor pulmonale or rheumatic heart disease with tricuspid insufficiency, or due to heart failure complicated by shock and hypertension.

In the present study one patient who had severe congestive cardiac failure with shock, showed elevation of asparate transaminase upto 925 I.U. which correlate with Richman's observation. In the present study 39 cases (52%) of cases showed elevated asparate transaminase levels and 31 cases (41%) of alanine transaminase levels. With remission 72% of raised asparate transaminase levels and 74% raised alanine transaminase levels returned to normal. Richman et al and Sherlock have reported elevation of serum alkaline phosphatase levels in 10-20% of patients with right sided heart failure. Dunn et al however reports that in most patients the levels are within normal limits, rarely do they exceed twice normal. Felders et al have also reported increased serum alkaline phosphatase in 10-20% of patients with congestive cardiac failure.

Elevation of serum alkaline phosphatase levels do not correlate with increases in serum bilirubin or aminotransferases. The highest elevations are usually seen in patients with marked liver enlargement. With improvement in the cardiac status serum alkaline phosohatase returns to normal in 1 week.

In the present study 10 cases (13%) showed elevation in alkaline phosphatase levels. With remission, in all cases the serum alkaline phosphatase levels returned to normal. In 51% of cases that had hepatomegaly, 20% showed elevated serum alkaline phosphatase levels which correlates with Richman et al's study. In controls, the values were normal.

The serum albumin was decreased in 30-50% of patients with congestive cardiac failure as per Richman et al.The degree of hypoalbuminemia was usually mild and the majority of patients exhibit levels between 2.5and 2.9 g/dl. Dunn et al reported that serum albumin concentrations below 1.5 g/dl are rarely observed and are often associated with marked ascites and edema. With resolution of the underlying cardiac disease, improvement in serum albumin usually occurs over a period of a few months.

According to Richman et al and Novel O et al [23], hyperglobuminemia occurs in 37-50% of patients with right sided heart failure and is more common in patients with acute than with chronic heart failure. The elevation tends to be mild, with levels between 3.5 and 4.1 g/dl in the majority of patients. In contrast to other liver tests, the hyperglobulinemia usually does not return to normal after successful treatment of the congestive cardiac failure. The increase in globulin levels and the decrease in albumin levels lead to reversal of Alubumin/Globulin ratio.

In the present study 15 cases showed decreased albumin (considering a cutoff 3mg%); 3 cases showed increased globulin. Albumin globulin reversal was noted in 3 cases. On remission one patient turned normal. Presence of albumin globulin and hypoalbuminemia were associated with a poor prognosis.

Prothrombin time is prolonged in 80-90% of patients with acute and chronic heart failure according to White et al. Richman et al and Dunn et al report that the prothrombin time returns to normal usually 2 to 3 weeks following the successful treatment of heart failure. In the present study 42 of 100 (42%) cases studied showed a prolonged prothrombin time. The prolongation in the prothrombin time ranged from 11/2 times to 2 times that of the control. Controls did not show any variation. Repeated prothrombin time at day 7 did not show any improvement on remission. As the prothrombin time could not be repeated at 3 weeks from remission changes might not have occurred; as suggested by other studies.

Conclusion

1. The commonest aetiology of cases presenting with features of congestive cardiac failure at Government General Hospital, Siddipet is coronary artery heart disease.
2. Liver function abnormalities were mostly present in patients with coronary artery disease (73%) and rheumatic valvular heart disease (70%) developing heart failure.

3. Liver function abnormalities were least in patient with hypertensive heart disease (50%) developing heart failure.
4. The serum bilirubin, serum alkaline phosphatase and serum transaminases returned to normal with remission. Serum protein values and the prothrombin time did not change with remission.
5. The serum bilirubin, serum enzymes and prothrombin time were elevated with exacerbation. Serum proteins did not show any change with exacerbation.
6. Severe congestive cardiac failure with hypotension leads to a gross elevation of serum asparate transaminase and alanine transaminase.
7. Serum alkaline phosphatase elevation correlated with the presence of hepatomegaly.
8. Serum bilirubin levels at presentation of more than 5mg, presence of hypoalbuminemia and albumin-globulin ratio reversal were associated with a poor prognosis.

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