ORIGINAL RESEARCH ARTICLE

Association of Renal Profile with Severity of Disease in Liver Cirrhosis Patients

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A B S T R A C T

Introduction: Liver fibrosis results from the perpetuation of the normal wound healing response resulting in an abnormal continuation of fibrogenesis (connective tissue production and deposition). Special care should be given for the recognition of the acute or chronic character of renal disease in liver cirrhosis patients. Hence; we planned the present study to assess the correlation of renal profile with the severity of disease in liver cirrhosis patients.

Material and methods: The present study included evaluation of liver cirrhosis patients and finding of correlation of severity of disease with the renal profile. A total of 40 liver cirrhosis patients were included in the present study. Complete demographic and clinical details of all the patients were obtained. We carried out all the haematological, hepatic and renal investigations in all the patients. Physical examination was done to look for any evidence of cardiac or renal involvement in each and every patient. Liver disease was staged according to Child-Pugh's score. All the results of etiological profile, evidence of cirrhosis, hepato-renal syndrome, and renal investigation were tabulated and compared. All the results were analyzed by SPSS software.

Results: A total of 40 liver cirrhosis patients were included in the present study. Out of 40, in 22 patients, alcohol was the etiologic factor. Renal profile of the liver cirrhosis patients was significantly associated with severity of disease in liver cirrhosis patients.

Conclusion: Renal profile is altered significantly with increase in severity of disease in liver cirrhosis patients.

Key words: Cirrhosis, Liver, Renal

INTRODUCTION

Liver is an interesting organ which receives all exiting circulation from the small and most of the large intestine, as well as spleen and pancreas, through the portal vein.[1] Liver fibrosis results from the perpetuation of the normal wound healing response resulting in an abnormal continuation of fibrogenesis (connective tissue production and deposition).[2] Fibrosis progresses in variable manner depending on the etiology of liver disease, host and environmental factors. Cirrhosis is an advanced stage of liver fibrosis that is accompanied by distortion of the hepatic vasculature.[3] A failing liver cannot remove toxins from the blood, so they eventually accumulate in the brain. The build-up of toxins in the brain is called hepatic encephalopathy. This condition can decrease mental function and cause stupor and even coma.[4,5] Physicians involved in the care of patients with cirrhosis recognize that the development of renal dysfunction is associated with significant morbidity and mortality. Special care should be given for the recognition of the acute or chronic character of renal disease; the causes of renal injury; the clinical conditions leading concomitantly to acute kidney injury and liver dysfunction, and the prognostic factors associated with the progression of acute kidney injury.[6-8]

Hence; we planned the present study to assess the correlation of renal profile with the severity of disease in liver cirrhosis patients.

MATERIAL AND METHODS

The present study was carried out in the department of general medicine. It included evaluation of liver cirrhosis patients and finding of correlation of severity of disease with the renal profile.

Informed consent was obtained after explaining in detail the entire research protocol.

A total of 40 liver cirrhosis patients were included in the present study.

Exclusion Criteria

- Patients of acute liver disease.
- Patients with chronic renal failure.
- Patients with rheumatic heart disease.
- Patients with collagen disease.
- Patients with any bleeding disorders.
- Patients having hepatocellular carcinoma/ any malignancy.
- Patients on any form of medicine or drug that modified the renal functioning

Methodology

Complete demographic and clinical details of all the patients were obtained. We carried out all the haematological,

Factor	1 point	2 points	3 points
Total bilirubin (mg/dL)	<2	2 – 3	>3
Serum albumin (g/L)	>3.5	2.8-3.5	<2.8
INR	<1.7	1.71-2.30	>2.30
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)

Table-1: Child-Pugh's score

Child Pugh Grade	Points
A	5- 6
B	7- 9
C	10- 15

Table-2: Child Pugh grading

Parameter	No. of patients	P- value
Alcohol	22	0.01*
NASH	6	
Hepatitis C	7	
Others	5	
Total	40	

*: Significant; NASH: Nonalcoholic steatohepatitis

Table-3: Distribution of subjects according to Etiology Code

Gender	No. of patients
Males	30
Females	10
Total	40

Table-4: Distribution of subjects according to gender

Parameter		N= 40
Blood urea	Normal	31
	Raised	9
	Total	40
Serum creatinine	Normal	31
	Raised	9
	Total	40

Table-5: Distribution of subjects according to renal parameters

hepatic and renal investigations in all the patients. Physical examination was done to look for any evidence of cardiac or renal involvement in each and every patient. Liver disease was staged according to Child-Pugh's score which is mentioned below.[9] (Table 1 and Table 2)

All the results of etiological profile, evidence of cirrhosis, hepato-renal syndrome, and renal investigation were tabulated and compared and correlated accordingly.

STATISTICAL ANALYSIS

The obtained data was analyzed by SPSS software. Chi-square test and student t test was used for assessment of level of significance. P- value of less than 0.05 was taken as significant.

RESULTS

A total of 40 liver cirrhosis patients were included in the present study. Out of 40, in 22 patients, alcohol was the etiologic factor, while in 7 patients, hepatitis C was the etiologic factor. Significant results were obtained while comparing the etiologic factors in patients of the present study (Table 3). Out of 40 liver cirrhosis patients, 30 were males and 10 females (Table 4). It was found that 31 normal and 9 raised Blood urea and simillar in Serum creatinine levels (Table 5) in all 40 patients. Table 6 is showing Distribution of subjects according to Child Pugh Score from all 40 subjects maaximum 24 comes in B Grade, 8-8 subjects comes in A and C grade respectively. Renal profile of the liver cirrhosis patients was significantly ($p<0.007^*$) associated with severity of disease in liver cirrhosis patients (Table 7 and 8).

Parameter		N= 40
Child Pugh Score	A	8
	B	24
	C	8
	Total	40

Table-6: Distribution of subjects according to Child Pugh Score

Blood urea	Child Pugh Score			Total	P- value
	A	B	C		
Normal	7	20	4	31	0.007*
Raised	1	4	4	9	
Total	8	24	8	40	

*: Significant

Table-7: Distribution of patients with Blood urea and severity of Cirrhosis of liver

Serum creatinine	Child Pugh Score			Total	P- value
	A	B	C		
Normal	7	20	4	31	0.007*
Raised	1	4	4	9	
Total	8	24	8	40	

*: Significant

Table-8: Distribution of patients with Blood urea and severity of Cirrhosis of liver

DISCUSSION

In the present study, we observed that renal manifestation was significantly associated with severity of disease in liver cirrhosis patients. Chayanupatkul M et al explained that the pathophysiology of cirrhotic cardiomyopathy is characterized by the impaired systolic response to physical stress, diastolic dysfunction as well as electrophysiological abnormalities especially prolongation of QT interval. The proposed mechanisms of systolic dysfunction consists of impairment of β-adrenergic receptor, the increase in endogenous cannabinoids, the presence of cardiosuppressants such as

nitric oxide and inflammatory cytokines. The impaired diastolic function results due to activation of cardiac renin-angiotensin system and salt retention which also leads to the development of cardiac hypertrophy. The derangement in membrane fluidity and ion channel defect leads to QT interval prolongation, which is found in 40-50% of cirrhotic patients. The increased identification of this disease will avert the complications of heart failure after hepative operations such as transjugular intrahepatic portosystemic shunt (TIPS) and liver transplantation.[10]

Peter G et al assessed whether prolongation of QT interval is an independent risk factor for development of hepatorenal syndrome in cirrhotic patients with acute variceal bleeding and found that hepatorenal syndrome developed in 17.9% patients. QT corrected by heart rate (QTc) prolonged at T1, returning towards baseline at T2 (mean ± SD; from 424.0±10.2 to 461.2±17.6 to 426.1±8.8ms, P 468 ms and sodium.[11] Nasr FM et al evaluated right and left ventricular systolic and diastolic functions in post hepatitis C liver cirrhosis patients using conventional echocardiography and tissue Doppler imaging. This study was conducted on 75 adults from inpatient and outpatient services of the Theodor Bilharz Research Institute (TBRI) hospital. They were divided into two groups: Group 1 included 50 patients with post hepatitis C liver cirrhosis; and Group 2 included 25 normal adults serving as a control group. All patients and normal volunteers were subjected to clinical examination, laboratory evaluation, abdominal ultrasonography and echocardiographic studies with tissue Doppler imaging for evaluation of left and right ventricular systolic and diastolic functions As a results The mitral flow showed significant increase in A wave velocity, as well as DT and IVRT with a significant decrease in E/A ratio in Group 1 compared to Group 2 ($P<0.01$). The tricuspid flow also showed a significant increase in A wave velocity ($P<0.01$) and DT ($P<0.05$) in addition to a significant decrease in E wave velocity and E/A ratio ($P<0.01$) in Group 1 as compared to Group 2. At the mitral annulus, we found a significant increase in average Aa velocity, E/Ea ratio and average systolic wave velocity S, in addition to a statistically significant decrease in the average Ea velocity and average Ea/Aa ($P<0.01$) in Group 1 as compared to Group 2. At the tricuspid annulus, there were significant increases in the average Aa velocity ($P<0.01$), S velocity ($P<0.01$) and E/Ea ($P<0.05$) together with a statistically significant decrease in the average Ea/Aa and average Ea velocity ($P<0.01$) in Group 1 compared to Group 2.[12]

Negru RD et al evaluated the extent of the QT interval prolongation, identify etiological and biochemical elements linked with it, and investigate the correlation with ventricular arrhythmic events in accordance to etiology and severity of liver cirrhosis and revealed that prolongation of QT interval is associated with moderate severity, alcoholic etiology, and plasmatic level of total proteins and triglycerides, however not with a higher incidence of ventricular arrhythmic events, except for hepatitis C virus etiology, favouring the novel hypothesis of a direct effect of this virus in cardiac arrhythmic effect.[13]

CONCLUSION

From the above results, the authors concluded that renal profile is altered significantly with increase in severity of disease in liver cirrhosis patients. However; future studies are recommended.

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