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ORIGINAL ARTICLE

Serum Creatinine Levels Unrelated to Child-Pugh Status in Uncomplicated Cirrhosis of Liver with Ascites

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ABSTRACT

Introduction: Liver cirrhosis and renal failure are two diseases that may occur in conjunction due to mutually related pathological processes. Liver cirrhosis causes portal hypertension that ultimately causes splanchnic vasodilatation leading to hepatorenal syndrome. Hypovolemia is another cause of renal failure in liver cirrhosis. The study presented here was done to assess the renal functions in patients with liver cirrhosis with ascites.

Materials and methods: Sixty patients with uncomplicated cirrhosis of liver with ascites with normal renal function were included in this study. All patients were examined physically and biochemical. Main biochemical variables were serum bilirubin, serum albumin, serum creatinine and prothrombin time. Enrolled patients were categorized according to Child-Pugh class B and C. Serum creatinine levels were measured in all cases.

Results: Mean level of serum albumin, serum creatinine and prothrombin time among Child B and C classes of liver cirrhosis patients were 27.36 *vs* 26.84 gm/dl, 0.79 *vs* 0.93 mg/dl, 15.97 *vs* 19.26 seconds respectively. No statistically significant change in the serum creatinine level among Child B and C were noticed.

Conclusion: This study showed that the effect of liver cirrhosis on renal dysfunction would be minimal, if any.

Keywords: Serum creatinine, Renal failure, Liver cirrhosis.

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INTRODUCTION

World Health Organization (WHO) has defined cirrhosis of liver as a diffuse process characterized by fibrosis and conversion of normal liver architecture into structurally abnormal nodules. Most common complications of portal hypertension with cirrhosis of liver include hepatic encephalopathy, ascites, variceal bleeding, hepatorenal syndrome, spontaneous bacterial peritonitis and hepatocellular carcinoma.

In patients with renal failure, the major route of elimination of urea is not functional. Accordingly, there is increased blood urea leading to increased ammonia production. Renal failure is a challenging complication of cirrhosis of liver. Patients with cirrhosis of liver and renal failure are at high risk of mortality while awaiting for liver transplantation.[1,2] Renal failure in patients with cirrhosis of liver is primarily related to disturbance in circulatory function, reduction in systemic resistance due to primarily arterial vasodilatation in the splanchnic circulation triggered by portal hypertension.[3-5] Renal failure is common and particularly severe in patient with spontaneous bacterial peritonitis. In this case peritonitis is usually caused by Gram-negative bacteria due to bacterial translocation.[6,7] Nonsteroid anti-inflammatory drugs (NSAID) and diuretics may also cause renal failure in patient with liver cirrhosis since their kidney function is extremely dependent on renal prostaglandin.[8] Hepatorenal syndrome is common but severe complications are found in cirrhotic patients with ascites. about 20% of cirrhotic patients with ascites and normal renal function develop hepatorenal syndrome after 1 year and 39% after 5 years of ascites development.[9] The study presented here was planned to assess the effect of liver cirrhosis on renal function. We only enrolled patients with liver cirrhosis with uncomplicated state and avoided patients with liver cirrhosis with notable complications.

MATERIALS AND METHODS

A cross-sectional study to determine the scrum creatinine levels of uncomplicated cirrhosis of liver patients with ascites of Child-Pugh class B and C was planned. Study was done in the Department of Hepatology, Bangbandhu Sheikh Mujib Medical University, Dhaka, Bangladesh, during the period of January 2004 to December 2006. Sixty patients having uncomplicated cirrhosis of liver with ascites with normal renal function (serum creatinine <1.5 mg/dl) were enrolled in this study. Patients of cirrhosis with hepatorenal syndrome, spontaneous bacterial peritonitis, diuretic resistant ascites, renal impairment (serum creatinine >1.5 mg/dl), history of upper gastrointestinal bleeding, shock, sepsis and congestive cardiac failure were excluded from the study. The patients were adults of both sex having chronic liver diseases due to chronic hepatitis B or C or due to unknown etiologies. Detailed history and physical examination was done prior to inclusion of patients in this study. Ascites was detected clinically and was confirmed by ultrasonography. With all aseptic precaution 10 ml of venous blood was drawn from antecubital vein by disposable

syringe and blood was sent for assessment of bilirubin, albumin, prothrombin time and creatinine levels.

RESULTS

Baseline clinical data at the time of admission to hospital (Table 1) showed that the average age of patients was 49.3 years in Child B, 42.29 years in Child C. There were 37 patients of chronic hepatitis B, six having chronic hepatitis C and 17 having negativity of both viruses. They belong to either Child class B or C. They had clinically moderate to tense ascites.

The mean (±SD) of serum level of serum bilirubin in patients with Child-Pugh class B was 37.47 ± 33.40 and that for patients with Child-Pugh class C was 113.68 ± 107.21. The difference in mean bilirubin of the two Child-Pugh classes was statistically significant ($p < 0.01$). The patient with Child-Pugh C class had much higher level of serum bilirubin than that of Child-Pugh class B. The mean ± SD of serum level of albumin, creatinine and prothrombin time of patient with child class B were 27.36 ± 11.04 mg/dl, 0.79 ± 0.33 mg/dl and 15.97 ± 3.72 seconds, respectively, and those of Child-Pugh class C were 26.84 ± 7.41 mg/dl, 0.93 ± 0.31/dl and 19.26 ± 5.10 seconds, respectively (Table 2). The levels of serum creatinine levels did not show any significant differences between patients with Child-Pugh B and Child-Pugh C, respectively ($p > 0.05$).

DISCUSSION

Under normal physiological conditions the kidney is an organ of ammonia metabolism. Under normal post-absorptive condition 70% of total ammonia is released in renal vein and remainder is excreted in urine. However, during acute or chronic renal failure, this function is reversed. In clinical practice, serum creatinine measurement is still the most useful and widely accepted method for estimating renal function. Pinpointing the specific type of renal failure in cirrhosis of liver is important for both prognostic and therapeutic purposes. Differentiating the hepatorenal syndrome from acute tubular necrosis remains difficult. Granular cast can be observed in the urinary sediment in both condition but the presence of renal tubular epithelial cells favor the diagnosis acute tubular necrosis.[10] The occurrence of hypovolemia or septic shock immediately before and after renal failure favors the diagnosis of acute tubular necrosis. To date, most studies and consensus conferences have defined renal failure in cirrhosis as a serum creatinine concentration above 1.5 mg/dl (133 μmol per liter). In patients with cirrhosis due to low creatinine production because of reduced muscle mass, the low serum creatinine level results in an underestimation of the glomerular filtration rate (GFR). Thus, the current definition of renal failure in cirrhosis identifies only those patients with a severely reduced GFR (<30 ml per minute) and undoubtedly underestimates the prevalence of this clinical problem.

The present study depicting normal values of serum creatinine in Child B and C classes signifies normal function of kidneys in patients with liver cirrhosis. So any deterioration of renal function may be a sign of incipient hepatorenal syndrome or advancement of liver disease. Mortality among patients with cirrhosis and renal failure is very high, particularly among those with type 1 hepatorenal syndrome. Liver transplantation is an option that should be considered in all patients who have no contraindications to this procedure, and it should be performed as earliest, because severe renal failure is predictive of a poor outcome after transplantation.

The present study clearly depicts unchanged renal function even in uncomplicated cirrhosis of liver patients having ascites. So ascites *per se* is not related to impairment of renal function. The prognosis for patients with cirrhosis and renal failure is poor. The overall survival role is about 50% at 1 month and 20% at 6 months. Survival may vary according to type of renal failure. Usually renal function remains unchanged in cirrhotic patient. In patients with hepatorenal syndrome there may be functional impairment of renal function. Recently much works have occurred in the field of study for renal function in cirrhotic patients, but larger studies are required to approach the clinical intervention to prevent the consequence of hepatorenal syndrome and its management.

Table 1: Demographic data of the study group (n = 60)

Parameter	*Child B (12)*	*Child C (48)*	*p-value*
Male	10	43	
Female	2	5	
Age-range (years)	35-70	18-70	0.125
Mean	49.33	42.29	
HBsAg positive	6	31	
Anti-HCV positive	1	5	
Both negative	5	12	

Table 2: Biochemical parameters of patients of Child-Pugh classes

Parameter	*Child B (mean ± SD)*	*Child C (mean ± SD)*	*t-value*	*p-value*
Serum bilirubin (μmol/l)	37.47 ± 33.40	113.68 ± 107.21	4.157	<0.001
Serum albumin (gm/l)	27.36 ± 11.04	26.84 ± 7.41	0.191	0.849
Prothrombin time (sec)	15.97 ± 3.72	19.26 ± 5.10	2.014	0.05
Serum creatinine (mg/dl)	0.79 ± 0.33	0.93 ± 0.31	1.422	0.161

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