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Original Article

Correlation of Serum Creatinine with Child-Pugh Score among Patients with Decompensated Cirrhosis of Liver

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Shamim Ahmed[1], AJ M Emrul Kayesh[2], Wahiduzzaman[3], Miraz Mahmud[4], Golam Kibria[5], Farhad Ahmed[6], Bibek Halder[7]

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ABSTRACT

Introduction: *Child–Pugh score can reliably predict disease severity among patients with decompensated liver cirrhosis, but a cheaper and readily available screening method like serum creatinine could lead to faster prediction of change in disease severity and better prognosis.* ***Objectives:*** *Present study aims to determine if there is any correlation of serum creatinine with Child-Pugh score among patients with decompensated cirrhosis of liver.* ***Methods & Materials:*** *This cross-sectional study was conducted at Sher-E-Bangla Medical College Hospital, Barisal, from July 2015 to December 2015, on 100 patients suffering from decompensated cirrhosis of liver. Adult patients of either sex, age ≥ 18 years, were enrolled in the study through purposive sampling technique.* ***Results:*** *Out of the 100-study population, 34% were from age group 51 – 60 years. Mean age was 58.49 ± 6.74 years. Study population was male (76%) predominant. HBV was responsible for liver cirrhosis among 42% of the population. During admission, 43% had tense ascites, followed by 21% with encephalopathy and 19% with variceal bleeding. Ascites was the most common (69%) first sign of decompensation, followed by variceal bleeding at 31%. Majority (44%) of the study population was classified as class C, followed by 37% as class B and 19% as class A. No statistically significant ($p > 0.05$) relation between serum creatinine level and Child-Pugh score was found.* ***Conclusion:*** *Most patients of*

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1. *Medical Officer (OPD), Sher-E-Bangla Medical College Hospital, Barishal.*
2. *Assistant Professor & Department Head of Medicine, Sher-E-Bangla Medical College, Barishal.*
3. *Assistant Professor, Dept. Of Pediatrics, Patuakhali Medical College, Patuakhali.*
4. *RMO, National Institute of Diseases of The Chest and Hospital (NIDCH), Mohakhali, Dhaka.*
5. *Dialysis Medical Officer, National Institute of Kidney Diseases and Urology, Dhaka.*
6. *IMO, Kurmitola General Hospital, Dhaka.*
7. *Emergency Medical Officer, Mymensingh Medical College Hospital, Mymensingh.*

decompensated cirrhosis have shown some degree of renal impairment, but the degree of renal impairment did not show significant relationship with Child-Pugh score. Degree of renal impairment cannot be used as a predictor for severity of liver disease.

Keywords: *Child-Pugh Score, Cirrhosis, Serum Creatinine.*

INTRODUCTION

Liver cirrhosis has become a major public health concern in recent years with high morbidity and mortality, and currently the 14th most common cause of death globally with an estimated 1.03 million deaths per year [1]. The prevalence of liver cirrhosis is often underestimated, because patients at the early phase of liver cirrhosis are often asymptomatic, and most of patients with liver cirrhosis are admitted due to its related complications [2]. The 1-year mortality of liver cirrhosis varies greatly from 1% to 57% according to the complications [3]. These complications include varices, ascites, hepatic encephalopathy (HE), hepatopulmonary hypertension, hepatocellular carcinoma, hepatorenal syndrome, spontaneous bacterial peritonitis, and coagulation disorders [4].

Acute deterioration in liver function in patients with cirrhosis is defined as decompensated cirrhosis and is characterized by jaundice, ascites, hepatic encephalopathy, hepatorenal syndrome or variceal hemorrhage [5]. Child–Pugh score has been widely used to assess the severity of deterioration in liver function. It was firstly proposed by Child and Turcotte to predict the mortality in cirrhosis patients. The primary version of Child–Pugh score included ascites, hepatic encephalopathy (HE), nutritional status, total bilirubin, and albumin [6]. The Child–Pugh classification was later updated by adding prothrombin time or international normalized ratio (INR) and removing nutritional status [7]. it broke down patients into three categories: A - good hepatic function, B - moderately impaired hepatic function, and C - advanced hepatic dysfunction. Child class A patients have an estimated 10% mortality rate; Child class B patients have an estimated 30% mortality rate, and Child class C patients have an estimated 70 to 80% mortality rate [8,9]. The Child-Pugh score can help predict all-cause mortality risk and development of other complications from liver dysfunction, such as variceal bleeding, as well [10].

Patients with cirrhosis and portal hypertension develop circulatory dysfunction characterized by disturbances in systemic and renal hemodynamics [11]. Renal dysfunction is the renal failure in patients with advanced liver failure (acute or chronic) in the absence of any identifiable causes of renal pathology, the systemic hemodynamics are normal in the upright position, but become hyperdynamic in the supine position that is, cardiac output increases and systemic vascular resistance decreases. The renal circulation is frequently vasodilated with glomerular hyperfiltration [12]. With disease progression, circulatory dysfunction worsens, manifested as vasodilatation and relative intravascular underfilling [13]. There is increased activity of the sympathetic nervous and renin-angiotensin systems in order to maintain hemodynamic stability. Peripheral edema and ascites can occur as a result of worsening sodium retention despite an increase in natriuretic substances. As systemic vasodilation progresses, the systemic arterial pressure

falls. Renal perfusion also decreases, leading to decreased renal blood flow. The renal circulation also becomes hypersensitive to the vasoconstrictive effects of various activated hormonal systems. When the vasoconstrictors overwhelm the compensatory effects of the various renal vasodilators, renal vasoconstriction occurs and GFR falls [14]. Renal dysfunction occurs in the latest phases of cirrhosis and is considered the extreme expression of circulatory dysfunction. Renal dysfunction is a common and serious problem in patients with advanced liver disease.

Serum creatinine measurement is still the most useful and widely accepted method for estimating renal function. Renal function should be routinely monitored in all patients with decompensated cirrhosis of liver. Patients with cirrhosis shows low serum creatinine due to reduced muscle mass, masking true hepato-renal impairment. While Child–Pugh score can reliably predict disease severity among patients with decompensated liver cirrhosis, it is costly and time consuming. A cheaper and readily available screening method to assess these patients is needed and serum creatinine has the potential to act as a biomarker for patients with decompensated liver cirrhosis. Present study aims to determine if there is any correlation of serum creatinine with Child-Pugh score among patients with decompensated cirrhosis of liver.

METHOD & MATERIALS

This was a cross sectional study conducted at Sher-E-Bangla Medical College Hospital, Barisal, from July 2015 to December 2015, on 100 patients suffering from decompensated cirrhosis of liver, under the strict supervision of the institute authority and only after receiving the ethical clearance from the institutional review board. Adult patients of either sex were included in this study. Admitted patients of different Medicine units of Sher-E-Bangla Medical College Hospital, Barisal were approached for the study. Severely ill patients and patients with compensated cirrhosis of liver, previous hepatocellular carcinoma and significant electrolyte disorder were excluded from the study. Aims and objectives of the study along with its procedure, risks and benefits of the study were explained to the respondent in easily understandable local language. Data were collected through face-to-face interview using a semi-structured questionnaire and data collection tools, only after Informed written consent was taken from the respondents. As per the selection criteria, 100 patients of both gender, age $\geq$ 18 years, were enrolled in the study through purposive sampling technique.

Study population underwent detail history taking, physical examination and relevant investigations. Relevant scoring and staging were carried out as Child-Pugh score Class A, B and C. Then serum creatinine was observed. Blood samples were collected from the participants and allowed to clot for half an hour and then the samples were centrifuged for 15 minutes and serum was stored in ultra-deep freezer at -800C until analysis. Statistical analysis was performed using Windows® based software program Statistical Packages for Social Sciences 25 (SPSS-25) (Chicago, IL, USA). After collection, all the data were checked and cleaned. Quantitative data were expressed as percentage, mean and standard deviation and qualitative data were expressed as frequency distribution and percentage. To determine statistical

significance, chi square test was considered according to applicability. P value of < 0.05 was considered statistically significant.

RESULT

Out of the 100 study population, 34% were from age group 51 – 60 years of age, followed by 31% from 61 – 70 years of age (Table I). Mean age was 58.49 ± 6.74 years. Study population was male (76%) predominant. As for socio-economic status, 55% were from middle class, followed by 39% from lower class. HBV was responsible for liver cirrhosis among 42% of the population, followed by HCV with 38%. During admission, 43% had tense ascites, followed by 21% with encephalopathy and 19% with variceal bleeding.

Table I: Descriptive statistics of the study population (n = 100).

Variables		Value
Age (years)		58.49 ± 6.74
Age group (years)		
	20 – 30	3 (3%)
	31 – 40	9 (9%)
	41 – 50	13 (13%)
	51 – 60	34 (34%)
	61 – 70	31 (31%)
	> 70	10 (10%)
Sex		
	Male	76 (76%)
	Female	24 (24%)
Socio-economic status		
	Lower class	39 (39%)
	Middle class	55 (55%)
	Higher class	6 (6%)
Aetiology of cirrhosis		
	HBV	42 (42%)
	HCV	38 (38%)
	Alcohol	5 (5%)
	PBC/PSC	7 (7%)
	Unknown	8 (8%)
Clinical features during admission		
	Tense ascites	43 (43%)
	Encephalopathy	21 (21%)
	Variceal bleeding	19 (19%)
	Jaundice	9 (9%)
	Fever	15 (15%)
	Other	11 (11%)

Data presented as n (%).

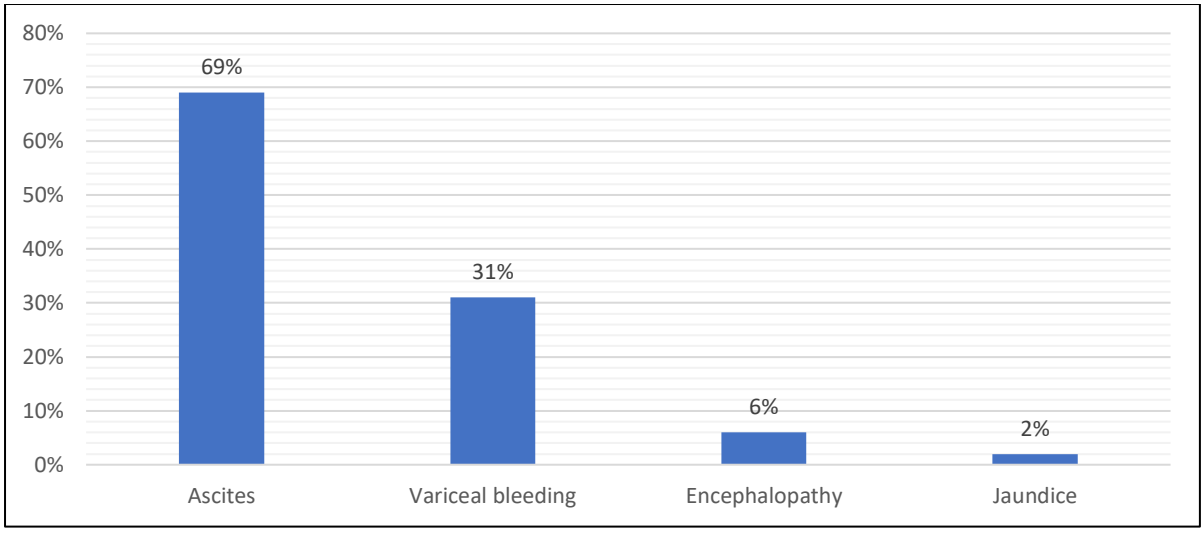

Figure 1: Distribution of study population according to first sign of decompensation (n = 100).

Study population was classified using the first sign of decompensation (Figure 1). Ascites was the most common (69%) first sign of decompensation, followed by variceal bleeding at 31% and encephalopathy at 6%. Biochemical profile of the study population was done (Table II). Mean hematocrit was 32.79 ± 4.38%, hemoglobin was 11.16 ± 2.41 g/dl, white blood cell count was 5.82 ± 5.24 $\times 10^9$/L, platelet count was 104.82 ± 78.59 $\times 10^9$/L, prothrombin time was 17.68 ± 7.81 second, INR was 1.3 ± 0.89, serum creatinine was 1.3 ± 0.97 mg/dL, bilirubin was 2.6 ± 5.16 mg/dL, AST was 72.42 ± 36.87 IU/L, ALT was 45.28 ± 41.57 IU/L and alkaline phosphatase was 107.27 ± 67.52 U/L.

Table II: Biochemical profile of the study population (n = 100).

Characteristics		Mean ± SD	Range (Min - Max)
	Hematocrit (%)	32.79 ± 4.38	17 - 48
	Hemoglobin (g/dL)	11.16 ± 2.41	5 - 16
	White blood count ($\times 10^9$/L)	5.82 ± 5.24	1.2 - 23.9
	Platelet count ($\times 10^9$/L)	104.82 ± 78.59	19 - 394
	Prothrombin time (s)	17.68 ± 7.81	11 - 35
	INR	1.3 ± 0.89	0.9 - 3.3
	Creatinine (mg/dL)	1.3 ± 0.97	0.5 - 3.7
	Bilirubin (mg/dL)	2.6 ± 5.16	0.3 - 33.9
	AST (IU/L)	72.42 ± 36.87	20 - 610
	ALT (IU/L)	45.28 ± 41.57	11 - 433
	Alkaline phosphatase (U/L)	107.27 ± 67.52	42 - 487

Data presented as mean ± SD and range.

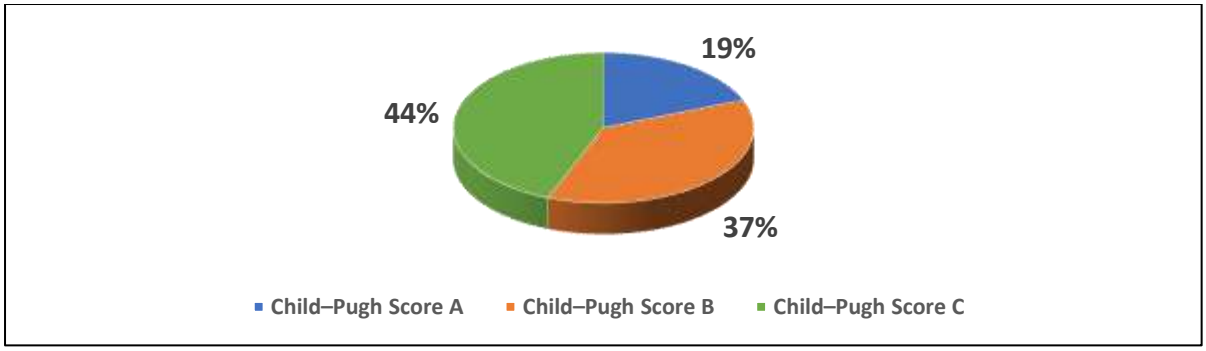

Figure 2: Distribution of study population according to Child–Pugh score (n = 100).

Study population was classified using Child–Pugh score (Figure 2). Majority (44%) of the study population was classified as class C, followed by 37% as class B and 19% as class A. Table III shows relation between serum creatinine level with Child-Pugh score and no statistically significant ($p > 0.05$) relation between serum creatinine level and Child-Pugh score was found.

Table III: Relation between serum creatinine level with Child-Pugh score (n = 100).

Characteristics		Child-Pugh score			P-value*
		A (n=19)	B (n=37)	C (n=44)	
Serum creatinine level (mg/dL)	< 0.6	3 (15.8%)	2 (5.4%)	1 (2.3%)	> **0.05**[a]
	0.6 - 1.2	6 (31.6%)	13 (35.1%)	19 (43.2%)	
	> 1.2	10 (52.6%)	22 (59.5%)	24 (54.5%)	

Data are presented as n (%).

[a] – Chi squared test was done

P value less than 0.05 was considered statistically significant

DISCUSSION

Present study showed a mean age of 58.49 ± 6.74 years with 76% male population and 65% of the study population had age between 51 to 70 years. These findings are consistent with prior studies among similar population groups [15,16]. Present study showed most common cause of cirrhosis to be HBV, HCV, alcohol and PBC/PSC which were 42%, 38%, 5% and 7% respectively. Previous studies showed a slightly reduced percentage for HBV and HCV, but increased percentage for alcohol [17]. This could be due to the difference in alcohol habit between the two study groups, current study being among a population with limited alcohol consumption habit. Tense ascites was found to be the leading cause of admission at 43% in present study, which is slightly higher than Papatheodoridis et al., 2005 study showing tense ascites to be the leading cause for admission for 36% of the population [17]. Ascites was found to be the first sign of

decompensation for 69% of the respondents in present study, which is consistent with the findings from prior studies [17–20].

Present study showed mean serum creatinine to be 1.3 ± 0.97 mg/dL for study population, which is lower compared to prior studies, showing the prevalence of higher than 1.5 mg/dL serum creatinine among patients with decompensated cirrhosis [21,22]. Present study shows majority (44%) of the population to be classified as Child–Pugh score class C, which is lower than Zatoński et al., 2010 study showing only 24.3% patients being classified as Child–Pugh score class C [23]. This could be due to the patients delaying in seeking treatment among study population. Although Zakim and Boyer, 2003 study shows 45% population to be classified as Child–Pugh score class C, which is consistent with the findings of present study [24]. Present study shows relation between serum creatinine level with Child-Pugh score and no statistically significant ($p > 0.05$) relation between serum creatinine level and Child-Pugh score was found. This finding is consistent with Rahman et al., 2013 study from Dhaka showing no statistically significant change in serum creatinine level among patients with different Child-Pugh score [25]. Although other studies have shown statistically significant change of creatinine with Child-Pugh score [26,27].

CONCLUSION

Most of the patients of decompensated cirrhosis have shown some degree of renal impairment, but the degree of renal impairment did not show significant relationship with Child-Pugh score. Based on the findings of this study, degree of renal impairment cannot be used as a predictor for severity of liver disease. But this was a single center study, and a multi-center study with larger sample size will provide more in-depth understanding on the relation between degree of renal impairment and Child-Pugh score among patients with decompensated cirrhosis of liver.

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