(e) ISSN Online: 2321-9599

(p) ISSN Print: 2348-6805

ORIGINAL ARTICLE

Assessment of incidence of renal dysfunction in liver cirrhosis patients

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ABSTRACT:

Background: The present study was conducted for evaluating incidence of renal dysfunction in liver cirrhosis patients. **Materials & methods:**A total of 40 individuals diagnosed with hepatic cirrhosis were recruited for the study. Comprehensive demographic and clinical profiles were documented for each subject. Biochemical assessments included quantification of total serum bilirubin, serum albumin concentrations, and prothrombin time. Evaluation of renal impairment was concurrently performed. The association between renal insufficiency and cirrhosis severity, as determined by the Child-Pugh grading, was subsequently analyzed. All collected data were tabulated in Microsoft Excel and subjected to statistical evaluation utilizing SPSS software. **Results:**The mean chronological age of the study population was 43.8 years. Elevated serum urea concentrations were observed in 17.5% (n = 7) of individuals, while increased serum creatinine levels, indicative of compromised glomerular filtration, were also documented in 17.5% (n = 7) of patients. These findings underscore the prevalence of renal involvement in hepatic cirrhosis.A statistically significant correlation was identified between renal dysfunction and the severity of hepatic decompensation as classified by the Child-Pugh scoring system. **Conclusion:**Liver cirrhosis is linked with considerable clinical morbidity and an elevated risk of mortality. Renal impairment frequently emerges during the advanced phases of cirrhotic progression and serves as a critical prognostic indicator of adverse clinical outcomes. Consequently, early detection and routine surveillance of renal biochemical parameters in cirrhotic individuals are imperative to facilitate timely therapeutic intervention and mitigate further deterioration of renal and hepatic function.

Key words: Renal, Cirrhosis, Liver

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This article may be cited as: Pandey JR, Kumar P. Assessment of incidence of renal dysfunction in liver cirrhosis patients. J Adv Med Dent Scie Res 2014;2(4):308-311.

INTRODUCTION

Hepatic cirrhosis has become a significant contributor to the global health burden. As reported by the Global Burden of Disease (GBD) 2010 analysis, cirrhosis accounted for approximately 31 million Disability-Adjusted Life Years (DALYs), representing 1.2% of the global DALY count, and was responsible for nearly one million fatalities, constituting 2% of total worldwide mortality in that year. The epidemiological patterns of hepatic cirrhosis have been extensively characterized in several high-income nations, particularly across Europe and the Americas.[1, 2] However, in low- and middle-income regions, especially within developing nations, there has been limited emphasis on mortality associated with cirrhotic disease, largely due to inadequate epidemiological surveillance and incomplete mortality data. International health agencies have progressively emphasized the mitigation of etiological factors contributing to cirrhosis, especially excessive ethanol consumption and persistent hepatitis B and C viral infections. In alignment with this objective, the World Health Organization (WHO) Western Pacific Region formulated a strategic goal in 2005 to achieve hepatitis B control by the year 2012.[3, 4]Renal dysfunction represents a frequent and clinically significant complication in individuals with hepatic cirrhosis, with an estimated prevalence of approximately one-fifth of hospitalized cirrhotic patients. This impairment in renal function may present acutely or may arise as an exacerbation of underlying chronic kidney disease (CKD). Irrespective of its temporal onset or etiology, renal impairment in the setting of cirrhosis is associated with substantially increased morbidity and elevated mortality risk.[5, 6] Hence; the present study was conducted for evaluating renal manifestations in liver cirrhosis patients.

MATERIALS & METHODS

A total of 40 individuals diagnosed with hepatic cirrhosis were recruited for the study. Comprehensive demographic and clinical profiles were documented for each subject. Biochemical assessments included quantification of total serum bilirubin, serum albumin concentrations, and prothrombin time. Additionally, the presence and extent of ascitic fluid accumulation and neuropsychiatric manifestations indicative of hepatic encephalopathy were systematically examined. These clinical and laboratory variables were collectively utilized to stratify the severity of hepatic cirrhosis using the Child-Pugh classification system. Evaluation of renal impairment was concurrently performed. The association between renal insufficiency and cirrhosis severity, as determined by the Child-Pugh grading, was subsequently analyzed. All collected data were

tabulated in Microsoft Excel and subjected to statistical evaluation utilizing SPSS software.

RESULTS

The mean chronological age of the study population was 43.8 years. A male predominance was noted, with 87.5% (n = 35) of the subjects being male, and 12.5% (n = 5) being female. Based on hepatic functional reserve stratification using the Child-Pugh scoring system, 30% (n = 12) of patients were categorized as Class A, 45% (n = 18) as Class B, and 25% (n = 10) as Class C, indicating a varied distribution of hepatic decompensation severity within the cohort.Renal impairment was identified in a subset of the study population. Elevated serum urea concentrations were observed in 17.5% (n = 7) of individuals, while increased serum creatinine levels, indicative of compromised glomerular filtration, were also documented in 17.5% (n = 7) of patients. These findings underscore the prevalence of renal involvement in hepatic cirrhosis.A statistically significant correlation was identified between renal dysfunction and the severity of hepatic decompensation as classified by the Child-Pugh scoring system. Among patients with elevated serum urea levels, 8.3% (n = 1) were in Class A, 16.7% (n = 2) in Class B, and 33.3% (n = 4) in Class C, with a p-value of 0.000 indicating high statistical significance. Similarly, for serum creatinine derangement, the distribution was identical—1 patient in Class A, 2 in Class B, and 4 in Class C—again yielding a p-value of 0.000. These findings indicate that renal impairment is more prevalent in individuals with advanced stages of cirrhosis and is significantly associated with worsening hepatic functional status.

Table 1: Demographic and clinical data of liver cirrhosis patients

Variable		Number	Percentage
Mean age		43.8 years	
Gender	Males	35	87.5
	Females	5	12.5
Child-Pugh Classification	Class A	12	30
	Class B	18	45
	Class C	10	25

Table 2: Incidence of renal dysfunction

Renal dysfunction	Number	Percentage
Abnormal serum urea levels	7	17.5
Abnormal serum creatinine levels	7	17.5

Table 3: Correaltion of renal dysfunction with severity grading of cirrhosis of liver

Renal dysfunction		Child-Pugh Classification			p-value
		Class A	Class B	Class C	
Serum urea	Normal	11	16	6	0.000 (Significant)
	Abnormal	1	2	4	
Serum creatinine	Normal	11	16	6	0.000 (Significant)
	Abnormal	1	2	4	

DISCUSSION

Cirrhosis represents a chronic hepatocellular disorder of substantial global prevalence, resulting from diverse etiological contributors such as adiposity-related hepatic steatosis, chronic ethanol-induced hepatotoxicity, persistent viral hepatitides (HBV and HCV), autoimmune hepatopathies, cholestatic syndromes, and hereditary metabolic disorders involving iron (hemochromatosis) or copper (Wilson's disease) overload. The underlying pathophysiological mechanism involves sustained hepatic inflammation leading to progressive parenchymal injury, characterized by extensive fibrosis and the formation of regenerative nodules, ultimately culminating in the development of portal hypertension. Renal dysfunction is a frequently encountered complication in cirrhotic patients, primarily driven by marked splanchnic arterial vasodilatation and systemic inflammatory activation, both of which contribute to altered renal perfusion and hemodynamic instability.[7-9]

The mean chronological age of the study population was 43.8 years. A male predominance was noted, with 87.5% (n = 35) of the subjects being male, and 12.5% (n = 5) being female. Based on hepatic functional reserve stratification using the Child-Pugh scoring system, 30% (n = 12) of patients were categorized as Class A, 45% (n = 18) as Class B, and 25% (n = 10) as Class C, indicating a varied distribution of hepatic decompensation severity within the cohort.Renal impairment was identified in a subset of the study population. Elevated serum urea concentrations were observed in 17.5% (n = 7) of individuals, while increased serum creatinine levels, indicative of compromised glomerular filtration, were also documented in 17.5% (n = 7) of patients. These

findings underscore the prevalence of renal involvement in hepatic cirrhosis.A statistically significant correlation was identified between renal dysfunction and the severity of hepatic decompensation as classified by the Child-Pugh scoring system. Among patients with elevated serum urea levels, 8.3% (n = 1) were in Class A, 16.7% (n = 2) in Class B, and 33.3% (n = 4) in Class C, with a p-value of 0.000 indicating high statistical significance. Similarly, for serum creatinine derangement, the distribution was identical—1 patient in Class A, 2 in Class B, and 4 in Class C—again yielding a p-value of 0.000. These findings indicate that renal impairment is more prevalent in individuals with advanced stages of cirrhosis and is significantly associated with worsening hepatic functional status. In a previous study conducted by Sort P et al, authors determined whether plasma volume expansion with intravenous albumin prevents renal impairment and reduces mortality in these patients. They randomly assigned 126 patients with cirrhosis and spontaneous bacterial peritonitis to treatment with intravenous cefotaxime (63 patients) or cefotaxime and intravenous albumin (63 patients). Cefotaxime was given daily in dosages that varied according to the serum creatinine level, and albumin was given at a dose of 1.5 g per kilogram of body weight at the time of diagnosis, followed by 1 g per kilogram on day 3. Renal impairment was defined as nonreversible deterioration of renal function during hospitalization.The infection resolved in 59 patients in the cefotaxime group (94 percent) and 62 in the cefotaxime-plus-albumin group (98 percent) (P=0.36). Renal impairment developed in 21 patients in the cefotaxime group (33 percent) and 6 in the cefotaxime-plus-albumin group (10 percent) (P=0.002). Eighteen patients (29 percent) in the cefotaxime group died in the hospital, as compared with 6 (10 percent) in the cefotaxime-plus-albumin group (P=0.01); at three months, the mortality rates were 41 percent (a total of 26 deaths) and 22 percent (a total of 14 deaths), respectively (P=0.03). Patients treated with cefotaxime had higher levels of plasma renin activity than those treated with cefotaxime and albumin; patients with renal impairment had the highest values.In patients with cirrhosis and spontaneous bacterial peritonitis, treatment with intravenous albumin in addition to an antibiotic reduces the incidence of renal impairment and death in comparison with treatment with an antibiotic alone.[10]Serum creatinine (SCr) has historically been utilized as a surrogate biomarker for evaluating renal function, based on the premise that its endogenous production remains relatively constant in individuals with stable body mass. Furthermore, it was presumed that SCr concentrations would be comparable among individuals matched for sex, age, and body weight. However, in individuals with advanced hepatic cirrhosis, this assumption does not hold true. Daily creatinine generation is notably diminished in this population due to two principal mechanisms: hepatic insufficiency impairs creatine synthesis, and concurrent malnutrition reduces the conversion of creatine to creatinine. These factors significantly undermine the reliability of SCr as an accurate index of renal function in cirrhotic patients.[11-13]Interpretation of SCr values in cirrhosis is further complicated by analytical interference. The Jaffe assay, commonly used for SCr measurement, is susceptible to false readings due to the presence of non-creatinine chromogenic substances in plasma, notably bilirubin. Kuster et al. demonstrated that even with compensation adjustments, the Jaffe method yielded an average underestimation of 6.14 μmol/L compared to enzymatic assays in cirrhotic individuals. This discrepancy led to a systematic overestimation of glomerular filtration rate (GFR) as calculated by the CKD-EPI equation, and correspondingly, an underestimation of the Model for End-Stage Liver Disease (MELD) score in those with SCr levels above 1 mg/dL.[14-17]Additionally, patients with impaired renal function—particularly those with advanced chronic kidney disease (CKD)—exhibit increased tubular secretion of creatinine, which further distorts the correlation between SCr and true GFR. Multiple investigations employing reference methods to directly quantify GFR have revealed that a considerable proportion of cirrhotic individuals with moderate to severe renal dysfunction exhibit either normal or only marginally elevated SCr concentrations. These findings collectively indicate that SCr lacks sensitivity and specificity as a renal biomarker in the cirrhotic population.[15-17]

CONCLUSION

Liver cirrhosis is linked with considerable clinical morbidity and an elevated risk of mortality. Renal impairment frequently emerges during the advanced phases of cirrhotic progression and serves as a critical prognostic indicator of adverse clinical outcomes. Consequently, early detection and routine surveillance of renal biochemical parameters in cirrhotic individuals are imperative to facilitate timely therapeutic intervention and mitigate further deterioration of renal and hepatic function.

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