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Original Research Article

THE STUDY OF PREVALENCE AND PATTERN OF RENAL DYSFUNCTION IN CIRRHOSIS: A CLINICAL CORRELATION WITH CTP AND MELD-NA SCORES

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ABSTRACT

Background: Renal dysfunction is a serious complication of liver cirrhosis, often contributing to significant morbidity and mortality. Early identification and classification of renal injury are vital for appropriate management. The objective is to determine the prevalence and patterns of renal dysfunction in patients with cirrhosis and to analyse their correlation with Child-Turcotte-Pugh (CTP) and MELD-Na scores.

Materials and Methods: A prospective observational study included 100 chronic liver disease (CLD) patients above the age of 18 years over a period of 18 months. Patients with acute hepatitis were excluded from the study. Renal dysfunction in CLD patients was classified into pre-renal AKI, intrinsic AKI, hepatorenal syndrome (HRS), and CKD. Severity of liver disease was evaluated using CTP and MELD-Na scores.

Results: Out of 100 CLD patients 88 were males 12were females. The mean age of the patients was 42.57 ± 13.45 years. Renal dysfunction was observed in 46% of patients. Among these, 22% had pre-renal AKI, 12% HRS, 6% intrinsic AKI, and 6% CKD. Significant correlations were found between renal dysfunction and higher CTP (Grade C: 75/92) and MELD-Na scores ($p < 0.001$). Mean serum creatinine was significantly elevated in patients with spontaneous bacterial peritonitis (SBP) and hepatic encephalopathy (HE) ($p < 0.01$).

Conclusion: Renal dysfunction is common in cirrhotic patients, most commonly pre-renal AKI. Severity of liver disease strongly correlates with renal injury. MELD-Na and CTP scores are useful prognostic tools in this population.

Keywords: Cirrhosis, Renal dysfunction, Hepatorenal syndrome, MELD-Na, CTP score, Acute kidney injury.

INTRODUCTION

Cirrhosis is defined as the morphological alteration observed in the end stage of a variety of chronic liver diseases (CLD), characterized by deranged hepatic angio-architecture, in which regenerative parenchymal nodules are encapsulated and separated by fibrotic septa.[1]

Cirrhosis may remain asymptomatic in its early stages but can progress to present with a range of complications, including ascites, spontaneous bacterial peritonitis (SBP), hepatorenal syndrome (HRS), variceal bleeding, hepatic encephalopathy (HE), hepatopulmonary syndrome, and hepatocellular carcinoma (HCC).[2]

Renal failure is a common and severe complication of decompensated cirrhosis and confers poor prognosis.[3] Acute kidney injury (AKI) occurs in approximately 19% of hospitalized patients with cirrhosis and chronic kidney disease (CKD) occurs in 1% of all patients with cirrhosis.[4] There is a spectrum of factors causing acute kidney injury (AKI) in liver cirrhosis including Prerenal AKI, Hepatorenal syndrome-type 1and 2, Intrinsic causes and postrenal causes.

The development of renal failure in patients with liver cirrhosis is associated with high morbidity and mortality and hence, timely and accurate diagnosis is essential to initiate therapy and improve clinical outcome. Renal dysfunction significantly influences prognosis in patients with liver cirrhosis with 50% of

patients with cirrhosis dying within 30 days of developing renal failure.[5]

Child Pugh score (CPS) has been widely utilized tool to assess liver dysfunction in the clinical practice.5 Renal dysfunction is heavily weighted in MELD (Model for End Stage Liver Disease) calculation as it has a strong impact on survival. The impact of the presence of renal insufficiency on survival has been the focus of several studies. MELD estimates that a one-unit increase in loge (creatinine) is linked with a 2.6-fold elevated risk of mortality.[4]

This study aims to assess the prevalence of renal dysfunction among patients with liver cirrhosis, to characterize the pattern of kidney disease namely acute kidney injury (AKI), hepatorenal syndrome (HRS), and chronic kidney disease (CKD) and to examine the correlation between the severity of renal impairment and liver disease severity as measured by the Child-Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores.

MATERIALS AND METHODS

Methods: A prospective observational study was conducted on 100 chronic liver disease (CLD) patients above the age of 18 years over a period of 18 months. patients with acute hepatitis were excluded from the study. Renal dysfunction in CLD patients was classified into pre-renal AKI, intrinsic AKI, hepatorenal syndrome (HRS), and CKD. Severity of liver disease was analysed using CTP and MELD-Na scores. MELD-Na and CTP scores calculated.

Renal dysfunction types identified as: pre-renal AKI, intrinsic AKI, CKD, or HRS.

The assessment of the patient was done by clinical examination, ultrasound, ascitic fluid analysis and blood investigations like RFT, LFT, Sr. electrolytes.

RESULTS

A prospective observational study was conducted on 100 chronic liver disease (CLD) patients above the age of 18 years over a period of 18 months from 1st June 2023 to 31st December 2024. The mean age of patients was 45.57 years and the majority belonged to 40-59 years (46.0%) followed by 20-39 years (36%). Out of 100 patients 88% were males and 12% were females with male to female ratio was 7.33:1. The majority had alcohol related liver disease (74%) followed by NAFLD (11%) and HBV (9%) related liver disease. Out of 100 patients ,24 had massive ascites ,48 had moderate and 20 had mild ascities.8 patients did not have ascities.23% of patients had SBP, 10% had hepatic encephalopathy and 88% had esophageal varices. Out of 100 cases 61% had grade C, 34% had grade B and 5 had grade A CTP score respectively. MELD score of 20-29,10-19,30-39 was seen in 41%, 34%, 21% patients respectively

Out of 100 patients 46.0% had renal dysfunction. Among 46.0%, 22.0% had pre-renal AKI, 12% had HRS, 6.0% had AKI secondary to renal cause and CKD respectively. The mean creatinine in CTPS grade-A was significantly low as compare to Grade-B and grade-C, the mean creatinine in Grade-B was significantly low as compare to Grade-C. This was statically highly significant (p=0.007).The mean MELD-sodium in grade-A was significantly low as compare to Grade-B and grade-C, the mean MELD-sodium in Grade-B was significantly low as compare to Grade-C.Which was statistically very highly significant (p=0.000).There was statistically highly significant difference of mean CTPS scores among liver cirrhosis with and without renal dysfunction (P<0.004) .There was statistically highly significant positive correlation between serum creatinine and MELD-Sodium score and Mortality rate (P<0.001). As serum creatinine values increased, MELD-Sodium score and Mortality rate also increased. As serum creatinine values increased, CTPS scores also increased. There was statistically significant positive correlation between serum creatinine and CTPS scores (P<0.05). Among 46 patients with renal dysfunction, 36 improved and 10 died. Among 54 patients without renal dysfunction, 48 improved and 6% died. This was highly significant (0.004).

Table 1: Age and Sex distribution of CLD patients

Age in years	No of males	No of females	Total Number	Percentage
20-30	06	-	06	6
30-40	30	-	30	30
40-50	46	8	54	38
50-60	06	4	10	08
Total	88	12	100	100
Mean ± SD			42.57± 13.45	

Table 2: Distribution of CLD patients according to etiology

Diagnosis	Number of cases	Percentage
Alcohol Related Cirrhosis	74	74
NAFLD Related Cirrhosis	11	11
HBV Related Cirrhosis	09	09
HCV Related Cirrhosis	01	01
Cryptogenic Cirrhosis	02	02
Autoimmune Cirrhosis	01	01
Wilson Related Cirrhosis	01	01
Cardiac Cirrhosis	01	01
Total	100.0	100.0

Table 3: Distribution of CLD patients according to CTP Scores

Grades of CTP Scores	CTP Scores	Number of cases	Percentage
A	5	01	01
	6	04	04
A Grade Total	---	05	05
B	7	06	06
	8	08	08
	9	20	20
B Grade Total	-----	34	34
C	10	15	15
	11	18	18
	12	14	14
	13	10	10
	14	03	03
	15	01	01
C Grade Total	----	61	61
Grand Total	-------	100	100

Table 4: Distribution of CLD patients according to MELD SODIUM Scores

MELD -NA Score	Number of cases	Percentage
Score ≥ 40	03	3.5
Score 30-39	21	21.0
Score 20-29	41	40.5
Score 10-19	34	34.0
Score ≤ 9	01	1.0
Total	100	100

Table 5: Pattern of Renal dysfunction In Liver Cirrhosis Cases

Types of Liver cirrhosis with renal dysfunction	Number of cases	Percentage
Pre-renal AKI	22	22.0
AKI Sec to renal cause	06	6.0
CKD	06	6.0
HRS	12	12.0
Total	46	46.0

Table 6: Comparison of CTPS scores with creatinine, sodium and MELD-sodium

Variables	Grades of CTPS			Correlation coefficient r & p-value	ANOVA-Test Value-Values and significance
	A	B	C		
	Mean ± SD	Mean ± SD	Mean ± SD		
Creatinine	0.98 ± 0.16	1.57 ± 1.23	2.06 ± 1.38	r = + 0.222 P<0.01	F= 5.11, P=0.007 HS
Sodium	137.5± 2.73	134.0 ± 5.10	131.6 ± 6.24	r = + 0.593 P<0.01	F= 6.98, P=0.004 HS
MELD-sodium	12.77 ± 1.93	18.34 ± 4.74	26.87 ± 7.06	r = - 0.256 P<0.01	F= 54.98, P=0.000 VHS

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

Table 7: Comparison of CTPS scores liver cirrhosis with renal dysfunction cases and liver cirrhosis without renal dysfunction cases

CTPS Grades	Liver cirrhosis with renal dysfunction cases (N=46) No (%)	Liver cirrhosis without renal dysfunction cases (N=54) No (%)	Total No (%)	Test-value, P-Values and significance
A	00 (0.0%)	05 (100%)	5	P = 0.004 HS
B	08(24.0%)	26 (76.0%)	34	
C	38(61.0%)	23 (39.0%)	61	
Total	46 (46.0%)	54 (54.0%)	100 (100.0%)	

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

Table 8: Comparison of study outcome between liver cirrhosis with renal dysfunction cases and liver cirrhosis without renal dysfunction cases

Study outcome	Liver cirrhosis with renal dysfunction cases (N=92) No (%)	Liver cirrhosis without renal dysfunction cases (N=108) No (%)	Total No (%)	Test-value, P-Values and significance
Improved	36 (42.8%)	48 (57.2%)	84 (100.0%)	$\chi2$=4.17 P =0.027, S
Death	10 (62.5%)	6 (37.5%)	16(100.0%)	
Total	46 (46.0%)	54(54.0%)	100 (100.0%)	

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

Table 9: Pearson coefficient for correlation (association) between serum creatinine and other variables

Variables	Correlation coefficient (r)	P-value and significance
MELD-Sodium	r = + 0.398	P = 0.002, HS
CTPS Scores	r = + 0.302	P = 0.019, S
Mortality	r = +0.372	P = 0.008, HS

DISCUSSION

In countries where the brunt of Alcoholic & viral hepatitis affected a significant percentage of its population, Chronic liver disease (CLD) and its complications continue to be a health problem.[6]

CLD is often accompanied by functional renal failure particularly in advanced stages of liver disease. Hemodynamic alterations with reduced effective arterial blood volume and peripheral vasodilation are followed by activation of vasoconstrictive hormones (rennin-aldosterone, vasopressin, endothelin) and neurohumoral systems (including increased activity of nervous system).[7]

Furthermore, infections, aggressive use of diuretics, repeated large volume paracenteses and gastrointestinal hemorrhage often contributes to the pronounced reduction in glomerular filtration rate (GFR). Nephrotoxic drugs such as nonsteroidal anti-inflammatory drugs or iodinated agents may also cause functional renal failure by inducing renal vasoconstriction.[8]

We took up this study to know the mechanisms of renal failure: pre renal, functional renal failure (HRS) and intrinsic renal failure in a patient with chronic liver disease. Functional renal failure is caused by renal hypoperfusion with no cell injury. Conversely, intrinsic renal failure may be due to glomerulonephritis or acute tubular necrosis. However, functional azotemia may in all cases lead to ischemic tubular injury and acute tubular necrosis. In this study we found that functional renal failure is the major mechanism of acute renal failure in patients with cirrhosis and has a poor outcome, with an in-hospital mortality of 16%.

In our study, the mean age of the patients was 45.57 years. Males mean age was 45.75 years and females mean age was 51.58 years. There was statistically significant difference between males and females (P<0.05). The mean age was 50.4 years in the study conducted by Florence et al.[9] This is comparable to Siregar and Gurning, where the mean age was 51.51 years. 4 However, according to another study, the mean age was 56.12 years.[10] This study showed male preponderance. The cross-sectional study conducted by Florence et al with 2346 patients also showed male preponderance (63.2%).[9] This was consistent with the study conducted by Siregar and Gurning, where 56.4% of males developed liver cirrhosis as compared with 43.6% females.[4] However, according to another study, males with liver cirrhosis comprised 68.8% of total patients. 10 In our study 88 % were male and 12% were female

In our study Alcohol, Hepatitis B, Hepatitis C and non-alcoholic fatty liver disease (NAFLD) were among the causes of cirrhosis. Out of 100, the majority of cases 74% was found to be alcohol induced liver cirrhosis followed by nonalcohol liver cirrhosis (11%), hepatitis B (9%), hepatitis C (1%) and 2% had unknown etiology. So, this study showed that alcohol intake was the leading cause of CLD followed by NAFLD. A study conducted by Parimal et al showed that 67% (n=134) had alcohol induced liver cirrhosis, 20% (n=40) had nonalcohol liver cirrhosis and 8% (n=16) had hepatitis B related cirrhosis, 3% (n=6) had hepatitis C related cirrhosis and 2%(n=4) cases had unknown etiology.[11] Florence et al also found alcohol consumption was the leading cause with prevalence of 63%.[9]

In the present study out of 100 chronic liver disease patients 46.0% of cases had renal dysfunction, whereas in case of Siregar and Gurning, 29.1% of patients developed RD.Florence et al study showed 46.8% (n=1099) liver cirrhosis with chronic kidney disease patients among 2346 patients.[9] Mohan et al reported 22% of patients with RD in liver cirrhosis.[12] Another study by Cheyron et al showed that AKI occurs in approximately 19% of hospitalized patients with cirrhosis.[13] and a study conducted by Fernandez-Seara J, the pprevalence rate of AKI in cirrhosis was 68%. Among 46.0% renal dysfunction cases in our study, 22% had pre-renal AKI, 12% of cases had HRS, each 6% cases had AKI sec to renal cause and CKD respectively. In the study of Fernandez-Seara J the prevalence of hepatorenal syndrome was 25%.[14] Another study showed that among patients with ascites, HRS developed in about 20% and 40% of the patients, at 1 and 5 years, respectively.[15]

CTP and MELD scores are two widely used scoring systems in CLD patients, not only to assess disease severity but also to estimate survival. MELD score is also used to prioritize patients on liver transplant waiting list. In our study, out of 100 cases 61% patients were in Child pugh turcotte score grade C, followed by 34% in grade'B' and 5% in grade A .The mean creatinine in grade-A was significantly low as compare to Grade-B and grade-C, the mean creatinine in Grade-B was significantly low as compare to Grade-C. which was was statistically highly significant difference of mean creatinine between the Grades of CTPS (P<0.01). A study by Hamza et al showed that a significant relationship between CPS and RD. RD was significant in patients with CP grade B and C (58%) as compared with those with grade A (0%).16 Siregar and Gurning also found that no one (0%) in grade A developed RD whereas 37.2% of patients with grade B or C developed renal failure. In this study there was statistically significant positive correlation between serum creatinine and CTPS scores (P<0.05). As serum creatinine values increased, CTPS scores were also increased. Siregar

and Gurning also reported a positive correlation between serum creatinine and CPS (r=0.359, p=0.007). 4 Choi et al also showed that RD was much more common in patients with higher severity of liver cirrhosis.[17] This shows that higher the severity of cirrhosis greater will be the risk of renal injury.

Low serum sodium has increasingly been recognized as a key determinant of outcomes in patients with ESLD, particularly those with hepatorenal syndrome and ascites.36–45 It has been shown to add to the prognostic value of the MELD score using the MELD-Na equation:46. In the study of jawaid iqbal, the mean MELD score was 21.75 ± 8.96. In 57 patients (80.3%), MELD score was > 15. The mean serum creatinine and mean serum sodium were 1.5 ± 1.1 mg/dl and 133.79 ± 6.9 mmol/L respectively. Statistically significant correlation was documented between MELD score and renal dysfunction (ɤ = 0.34, P = 0.004). Patients with higher MELD score were more prone to develop severe renal dysfunction. In our study the mean MELD-sodium in grade-A was significantly low as compare to Grade-B and grade-C, the mean MELD-sodium in Grade-B was significantly low as compare to Grade-Cwhich was statistically very highly significant (p=0.000). There was statistically highly significant positive correlation between serum creatinine and MELD-Sodium score and Mortality rate (P<0.001). As serum creatinine values increased, MELD-Sodium score and Mortality rate also increased. But in the study of Gomes et al. , serum creatinine in MELD score had no impact as a prognostic tool for acute kidney injury (AKI) in liver transplant patients, despite its high impact on MELD calculation.18 In-hospital mortality associated with AKI is also significantly higher in patients with cirrhosis, particularly in those with concomitant systemic inflammatory response syndrome, compared with those without liver disease (50%-68% vs 9%-11%, respectively).[3,19,20] Further deterioration in renal function is also frequent in hospitalized patients with cirrhosis and concomitant chronic kidney disease (CKD).[21]

In the study of florence wong, out of 1244 patients with cirrhosis C: 704(57%) were controls, 176 (14%) had acute kidney disease (AKD) and 364 (29%) had stage 1 Acute kidney injury (AKI).

AKD patients had similar baseline sCr to controls but had more hospital admissions in the past 6 months and higher peak sCr, though lower than that in stage 1 AKI patients (P < 0.0001). The in-hospital and 30-day mortality for controls was 4%, 9% for AKD and 16 % for AKI patients. In our study among 46 patients with renal dysfunction, 36 improved and 10 died. Among 54 patients without renal dysfunction,48 improved and 6% died. This was highly significant (P=0.004).

Comparison of creatinine with Hepatic Encephalopathy

We also found that there was statistical significance difference of mean serum creatinine between cases with HE and cases without HE (P<0.01).

The mean serum creatinine was significantly more in cases with HE as compared to cases without HE.

CONCLUSION

1. Renal dysfunction was prevalent in nearly half of cirrhotic patients Pre-renal AKI and HRS were predominant. Higher CTP and MELD-Na scores were strongly associated with renal dysfunction and mortality. Monitoring renal function and liver severity indices is crucial for early intervention. Early recognition and prevention of volume depletion helps in prevention of complication.

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