E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.02.251

Original Research Article

Study of Renal Functions in Chronic Liver Disease Patients Admitted to a Tertiary Care Hospital, Mysuru

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Received: 14.04.2026 Revised: 18.05.2026 Accepted: 28.06.2026 Published: 03.07.2026

ABSTRACT

Background: Chronic liver disease is a major cause of morbidity and mortality worldwide, with renal dysfunction being one of its most serious complications. Hemodynamic alterations associated with portal hypertension and systemic vasodilation predispose patients to impaired renal perfusion, leading to acute kidney injury and hepatorenal syndrome. Early identification of renal dysfunction is essential to improve clinical outcomes. This study evaluated renal function abnormalities and their association with different etiologies of CLD (Chronic Liver Disease).

Methods: A hospital-based cross-sectional observational study was conducted at K.R. Hospital, MMC&RI, Mysuru, from April 2023 to April 2024. Adult patients (>18 years) diagnosed with chronic liver disease were enrolled. Patients with pre-existing kidney disease, diabetes mellitus, hypertension, or a history of nephrotoxic drug use were excluded. Clinical details and laboratory parameters were collected to assess renal function and determine the incidence of renal impairment across various etiologies of CLD.

Results: Among patients with chronic liver disease, renal impairment was observed in 26% of cases. The majority of affected patients were middle-aged males with alcohol-related cirrhosis. Renal dysfunction was predominantly functional in nature and attributed to portal hypertension and systemic circulatory disturbances. Serum creatinine demonstrated limitations as a reliable marker of renal function in cirrhotic patients, emphasizing the need for careful clinical evaluation and early recognition of renal impairment.

Conclusion: Renal dysfunction is a common and clinically significant complication of chronic liver disease, particularly in patients with alcohol-related cirrhosis. Early detection and timely management are crucial to reducing morbidity and improving patient outcomes. The study highlights the need for improved diagnostic strategies and further research to enhance the assessment and management of renal dysfunction in chronic liver disease.

Keywords: Chronic Liver Disease, Renal Dysfunction, Acute Kidney Injury, Hepatorenal Syndrome, Cirrhosis, Portal Hypertension, Serum Creatinine, Alcoholic Liver Disease, Renal Impairment.

INTRODUCTION

Chronic liver disease is a progressive disorder characterized by persistent hepatic injury, fibrosis, and eventual cirrhosis, leading to significant systemic complications. Among these, renal dysfunction is one of the most serious and life-threatening consequences, contributing substantially to increased morbidity and mortality. The close physiological relationship between the liver and kidneys plays a crucial role in maintaining homeostasis, and impairment of hepatic function can adversely affect renal perfusion and function through complex hemodynamic and neurohormonal mechanisms.[1]

Renal dysfunction in CLD encompasses a broad spectrum ranging from mild reductions in GFR (Glomerular Filtration Rate) to severe kidney failure. HRS (Hepatorenal Syndrome), a functional form of renal failure occurring in

advanced liver disease, represents one of the most severe manifestations. However, renal impairment in CLD is not limited to HRS and may also result from acute tubular necrosis, pre-renal azotemia, chronic kidney disease, or acute kidney injury precipitated by infections, hypovolemia, or nephrotoxic medications.[2–5]

The pathogenesis of renal dysfunction in CLD is multifactorial. Portal hypertension-induced splanchnic vasodilation activates the renin-angiotensin-aldosterone system, sympathetic nervous system, and antidiuretic hormone release, resulting in renal vasoconstriction, sodium and water retention, and progressive decline in renal perfusion. In addition, systemic inflammation, bacterial translocation, and infectious complications such as spontaneous bacterial peritonitis further aggravate renal injury and worsen clinical outcomes.[3,4]

Renal function is an important prognostic indicator in patients with CLD and forms a key component of the MELD (Model for End-Stage Liver Disease) score used for liver transplant prioritization. Nevertheless, serum creatinine, the conventional marker of renal function, may underestimate renal impairment in cirrhotic patients because of reduced muscle mass and altered creatinine metabolism, highlighting the need for careful assessment and newer biomarkers.[5] Early identification and appropriate management of renal dysfunction are essential to improve survival and optimize treatment outcomes, including timely consideration for liver transplantation.

Aims and Objectives

The aim of this study was to evaluate renal function in patients with chronic liver disease by assessing renal function tests and determining the incidence of renal failure. The objectives were to evaluate alterations in renal function among patients with chronic liver disease and to compare these changes across the different etiologies of chronic liver disease to identify any significant variations in renal impairment.

MATERIALS AND METHODS

Study Design

This descriptive, cross-sectional observational study was conducted in the Department of General Medicine at a tertiary care teaching hospital over 18 months, from January 2022 to June 2023. Clinical, laboratory, and demographic data were collected prospectively without any intervention in patient management. The study design enabled the evaluation of the association between thrombocytopenia severity and disease characteristics across different etiologies of chronic liver disease.

Inclusion and Exclusion Criteria

Patients aged 18 years and above with a confirmed diagnosis of chronic liver disease based on clinical, biochemical, and radiological findings, who were willing to provide informed consent, were included in the study. Patients with a known history of kidney disease, a history of nephrotoxic drug consumption, or a known diagnosis of diabetes mellitus or hypertension were excluded from the study.

Sample Size Calculation

The sample size was calculated utilizing the standardized epidemiological formula for cross-sectional studies (Lwanga & Lemeshow, 1991):

$$n = \frac{z^2 \times p \times q}{e^2}$$

Where:

- n represents the required sample size
- z denotes the standard normal deviate corresponding to the selected confidence level (1.96 for 95% confidence interval)
- p indicates the estimated prevalence (65%)
- q equals 100 - pp p (35%)
- e signifies the allowable error, calculated as 15% of pp p (15% of 65 = 9.75)

By substituting the values in the above formula, the sample size was calculated n = 95.7. (rounded off to 96)

So, finally the sample size was estimated to be 100 (rounded of for easy comparison).

Data Collection Procedure

After obtaining informed consent, all enrolled patients underwent a detailed clinical evaluation, including history taking and physical examination. Demographic details, clinical findings, laboratory investigations, and relevant radiological assessments were systematically recorded using a structured case record form designed specifically for the study. Baseline investigations were performed at enrolment, and renal function parameters along with other relevant biochemical tests were documented according to standardized institutional protocols. Data were collected prospectively by trained medical personnel under supervision, ensuring uniformity in clinical assessment and laboratory procedures.

Each participant was assigned a unique study identification number to maintain confidentiality. All collected data were entered into an electronic database and cross-verified for accuracy, completeness, and consistency before statistical analysis.

Statistical Analysis

The collected data were analyzed using SPSS version 25.0**.** Quantitative variables were expressed as mean ± standard deviation (SD), while qualitative variables were presented as frequencies and percentages. The Chi-square test was used for categorical variables, and the Student's *t*-test or one-way ANOVA was used for continuous variables, as appropriate. Correlation analysis was performed to assess the relationship between renal function parameters and chronic liver disease. A *p*-value of **<**0.05 was considered statistically significant.

RESULTS

Variable	Category	Frequency	Percentage
Age	25–35 years	6	6%
	35–45 years	33	33%
	45–55 years	51	51%
	>55 years	10	10%
Gender	Male	66	66%
	Female	34	34%

Table 1. Demographic Characteristics of the Study Population (N = 100)

Table 1 illustrates the demographic profile of the study participants. The majority of patients belonged to the 45–55-year age group (51%), indicating that chronic liver disease was most common among middle-aged adults. A clear male predominance (66%) was also observed.

Variable	Category	Frequency	Percentage
Alcohol history	Yes	72	72%
	No	28	28%
HBsAg	Positive	10	10%
	Negative	90	90%
HCV	Positive	0	0%
	Negative	100	100%
NAFLD	Positive	18	18%
	Negative	82	82%

Table 2. Etiological Profile of Chronic Liver Disease

Table 2 demonstrates the etiological factors associated with chronic liver disease. Alcohol consumption was the predominant risk factor (72%), whereas hepatitis B accounted for 10% of cases. No participant was positive for hepatitis C, and NAFLD contributed to 18% of cases.

Variable	Category	Frequency	Percentage
USG	Cirrhotic liver with dilated portal vein	93	93%
	Fatty liver with dilated portal vein	7	7%
Upper GI Endoscopy	Normal	51	51%
	Grade II varices	25	25%
	Grade IV varices	24	24%

Table 3. Clinical and Imaging Findings

Table 3 illustrates the radiological and endoscopic findings among study participants. Cirrhosis with portal vein dilatation was the predominant ultrasonographic finding (93%). Endoscopy showed esophageal varices in nearly half of the patients, indicating significant portal hypertension.

Parameter	Category	Frequency	Percentage
Urea	<40 mg/dL	61	61%
	40–100 mg/dL	39	39%
Creatinine	<1.4 mg/dL	58	58%
	1.4–2.4 mg/dL	14	14%
	2.4–3 mg/dL	28	28%
Albumin	<3 g/dL	97	97%
Globulin	>4 g/dL	16	16%
AST >100 IU/L	35	35%	
ALT >100 IU/L	60	60%	
ALP >100 IU/L	68	68%	
Total Bilirubin >2 mg/dL	52	52%	
Direct Bilirubin High	100	100%	
High MCV	60	60%	
TLC Normal	100	100%	
Platelets Low	100	100%	

Table 4. Biochemical and Hematological Profile

Table 4 summarizes the laboratory profile of the study population. Hypoalbuminemia, thrombocytopenia, elevated liver enzymes, and impaired renal function were common findings, reflecting advanced liver dysfunction and associated renal involvement.

Variable	p-value	Interpretation
Age	0.050	Borderline significant
Gender	0.010	Significant

Table 5. Association between Demographic Factors and Renal Failure

Table 5 observes that increasing age showed a borderline significant association with renal failure, while female gender demonstrated a statistically significant association with renal dysfunction among patients with chronic liver disease.

Variable	p-value	Significance
Alcohol history	0.002	Significant
HBsAg	0.553	Not significant
HCV	Not applicable	No positive cases
NAFLD	Descriptive only	No association tested

Table 6. Association between Etiological Factors and Renal Failure

Table 6 demonstrates that alcohol history showed a statistically significant association with renal failure, whereas hepatitis B infection did not. Because all patients were HCV negative, no statistical comparison could be performed.

Variable	p-value	Interpretation
USG findings	0.364	Not significant
Upper GI Endoscopy	<0.001	Highly significant

Table 7. Association between Clinical Findings and Renal Failure

Table 7 illustrates the relationship between clinical findings and renal failure. Endoscopic evidence of portal hypertension was significantly associated with renal dysfunction, whereas ultrasonographic findings alone were not predictive.

Laboratory Parameter	p-value	Interpretation
Urea	0.622	Not significant
Creatinine	<0.001	Highly significant
Albumin	0.130	Not significant
Globulin	0.132	Not significant

AST	0.022	Significant
ALT	0.424	Not significant
ALP	0.154	Not significant
Total Bilirubin	0.281	Not significant
Direct Bilirubin	Not applicable	Uniformly elevated
MCV	0.413	Not significant
TLC	Not applicable	No variation
Platelets	Not applicable	Uniform thrombocytopenia

Table 8. Association between Laboratory Parameters and Renal Failure

Table 8 summarizes the association between laboratory parameters and renal failure. Serum creatinine emerged as the strongest predictor of renal dysfunction ($p < 0.001$), followed by AST ($p = 0.022$). Other biochemical and hematological parameters did not show statistically significant associations.

DISCUSSION

The present study evaluated renal function among 100 patients with CLD. The majority of patients were aged 45–55 years (51%), followed by the 35–45-year age group (33%), with a marked male predominance (66%). These findings are consistent with the observations of Wang et al.[6] who reported that chronic liver disease predominantly affects middle-aged adults because of prolonged hepatic injury. Similarly, Marcellin and Kutala [7] highlighted the higher prevalence of CLD among males due to greater exposure to alcohol-related risk factors.

In the present study, alcohol-related liver disease (72%) was the leading cause of CLD, followed by hepatitis B infection (10%), while no cases of hepatitis C were detected. These findings are in agreement with Marcellin and Kutala [7] and Younossi et al.[8] who identified alcohol as one of the major etiological factors contributing to the global burden of chronic liver disease.

Ultrasonography revealed cirrhosis with dilated portal vein in 93% of patients, indicating advanced liver disease. Tsochatzis et al.[9] described cirrhosis as the end stage of chronic liver injury associated with portal hypertension, while Ginès and Schrier[10] explained that portal hypertension results in systemic vasodilatation and renal hypoperfusion, thereby predisposing patients to renal dysfunction. The high prevalence of esophageal varices observed in the present study further reflects advanced portal hypertension, which has been recognized by Biggins et al.[11] as an important risk factor for acute kidney injury.

The present study demonstrated that renal failure was observed in 26% of patients with chronic liver disease, indicating a considerable burden of renal impairment. Similar findings were reported by Ginès and Schrier,[10] who identified renal dysfunction as one of the most serious complications of cirrhosis. Furthermore, Belcher et al.[12] and Allegretti et al.[13] demonstrated that even mild deterioration in renal function significantly increases mortality and worsens clinical outcomes in patients with cirrhosis.

Renal dysfunction in the present study was predominantly observed in patients with advanced liver disease, ascites, jaundice, and portal hypertension. Bernardi et al.[14] and Ruiz-del-Arbol et al.[15] reported that splanchnic vasodilatation, activation of the renin–angiotensin–aldosterone system, and renal vasoconstriction play a central role in the development of hepatorenal syndrome. In addition, Angeli et al.[16] showed that infections such as spontaneous bacterial peritonitis further aggravate renal dysfunction through systemic inflammation.

The higher frequency of renal dysfunction among patients with alcohol-related liver disease in the present study supports the findings. Demonstrated that chronic alcohol exposure contributes to hepatic inflammation and increases susceptibility to renal injury. Likewise, Marcellin and Kutala[7] emphasized that alcohol-related liver disease continues to be a major global public health concern.

The present study also highlights the limitations of serum creatinine as a marker of renal dysfunction in cirrhosis. Angeli et al.[17] recommended that acute kidney injury should be identified using dynamic changes in serum creatinine because reduced muscle mass in cirrhotic patients may underestimate the severity of renal impairment.

The presence of renal dysfunction in 26% of patients has important prognostic implications. Alessandria et al.[18] demonstrated that MELD score is a strong predictor of survival in hepatorenal syndrome, while Simonetto et

al.[19] emphasized that early diagnosis and treatment with albumin and vasoconstrictors improve patient outcomes. Similarly, Solanki et al.[20] and Piano et al.[21] reported improved renal recovery with terlipressin and albumin therapy. In advanced cases, liver transplantation remains the definitive treatment, whereas renal replacement therapy serves as supportive management until transplantation becomes feasible, as described by Huelin et al.[22]

CONCLUSION

The present study confirms the close relationship between liver and kidney function in patients with CLD. The observed prevalence of renal dysfunction (26%) highlights that renal impairment is a common and clinically significant complication of CLD, with important implications for patient prognosis, therapeutic decision-making, and liver transplantation outcomes. These findings underscore the importance of regular assessment of renal function, early identification of kidney impairment, and timely multidisciplinary management to improve clinical outcomes. Furthermore, the study emphasizes the need for continued research to develop more sensitive diagnostic markers and effective therapeutic strategies for the prevention and management of renal dysfunction in this high-risk patient population.

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