International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Research Article

Evaluation of Renal Function in Chronic Liver Disease

Dr. Abhishek Kumar Verma[1], Dr. Anurag Vyas[2], Dr. Brijraj Mahida[3], Dr. Rangat Sharma[4]

[1]Associate Professor, Department of General Medicine, NCRIMS, Meerut, Uttar Pradesh, India
[2] Professor &Head, Department of General Medicine, NCRIMS, Meerut, Uttar Pradesh, India
[3]Junior Resident, Department of General Medicine, NCRIMS, Meerut, Uttar Pradesh, India
[4]Assistant Professor, Department of General Medicine, NCRIMS, Meerut, Uttar Pradesh, India

Corresponding Author:

Dr. Brijraj Mahida
Junior Resident, Department of General Medicine, NCRIMS, Meerut, Uttar Pradesh, India

Received: 01-08-2025
Accepted: 20-08-2025
Available Online: 07-09-2025

ABSTRACT

Background: Chronic liver disease (CLD) is a major cause of morbidity and mortality. Renal dysfunction frequently complicates CLD and worsens prognosis. Serum creatinine often underestimates renal impairment, while creatinine clearance provides a more accurate assessment.
Objectives: This study evaluated renal function in CLD patients using serum creatinine and creatinine clearance, and examined the influence of etiology on renal dysfunction.
Methodology: A cross-sectional study was conducted at NCRIMS, Meerut, including 50 CLD inpatients. Patients >60 years, with overt renal failure, diabetes, hypertension, or primary renal disease were excluded. Renal function was assessed by serum creatinine, 24-hour creatinine clearance, and Cockcroft–Gault formula.
Results: Mean age was 46.8 years; 70% were males. Alcoholic cirrhosis (58%) was most common. Although serum creatinine was normal in all patients, renal dysfunction was detected in 38% by 24-hour clearance and 42% by Cockcroft–Gault, more frequent in alcoholic CLD (48%) than hepatitis B (33%) and others (22%) ($p < 0.05$).
Conclusion: Serum creatinine underestimated renal dysfunction, while clearance methods were more sensitive. Alcoholic CLD showed the highest risk, supporting routine clearance-based monitoring.
Keywords: CLD, Renal dysfunction, Creatinine clearance, Alcoholic cirrhosis.

INTRODUCTION

Chronic liver disease (CLD) represents a spectrum of progressive hepatic disorders characterized by gradual destruction and regeneration of liver parenchyma leading to fibrosis, cirrhosis, and impaired liver function. It is a major global health problem and contributes significantly to morbidity and mortality, particularly in low- and middle-income countries. According to the World Health Organization, liver diseases account for nearly 2 million deaths annually, of which cirrhosis contributes to more than one million, placing CLD among the leading causes of global disease burden [1].

The interplay between the liver and kidney is complex, as hepatic dysfunction often affects renal hemodynamics and filtration capacity. Renal impairment is a frequent and serious complication of CLD, particularly in cirrhosis, and is associated with poor prognosis. Even mild alterations in renal function are known to adversely affect survival and increase the risk of complications such as hepatorenal syndrome and hepatic encephalopathy [2]. Traditionally, serum creatinine has been used as a standard marker of renal function; however, it often underestimates renal impairment in CLD due to factors such as reduced hepatic creatine synthesis, decreased muscle mass, and dilutional effects from fluid overload [3].

Creatinine clearance, measured by 24-hour urinary creatinine excretion or estimated through formulas such as the Cockcroft–Gault equation, provides a better estimate of glomerular filtration rate (GFR) and may serve as a more sensitive marker of renal dysfunction in CLD patients [4]. Several studies have highlighted discrepancies between serum

creatinine levels and true renal function in cirrhotic patients, emphasizing the need for more accurate methods of assessment [5]. Identifying early renal impairment in these patients is crucial for timely intervention and prognostic evaluation.

The etiology of chronic liver disease also appears to play a role in the occurrence and severity of renal dysfunction. Alcoholic cirrhosis, viral hepatitis, and non-alcoholic fatty liver disease are common causes of CLD in India, each with varying impacts on renal function. Studies from India have shown that patients with alcoholic cirrhosis are particularly prone to renal impairment due to combined effects of hepatotoxicity, malnutrition, and systemic hemodynamic changes [6]. Similarly, viral hepatitis–related cirrhosis has been associated with immune-mediated renal injury and glomerulonephritis [7].

India bears a substantial burden of CLD, with an estimated prevalence of cirrhosis ranging between 0.2–0.5% in the general population and accounting for nearly 10% of all hospital admissions in tertiary care centres [8]. Uttar Pradesh, with its high prevalence of alcohol consumption and viral hepatitis, contributes significantly to this burden. Evaluating renal function in CLD patients is particularly important in this setting, not only for clinical management but also for understanding the prognostic implications of different etiologies.

Given these considerations, this hospital-based cross-sectional study was undertaken at a tertiary care centre in Uttar Pradesh to evaluate renal function in patients with chronic liver disease using serum creatinine and creatinine clearance parameters, and to determine whether the etiology of CLD influences renal dysfunction.

METHODOLOGY

This study was designed as a hospital-based cross-sectional analytical study and was conducted in the Department of General Medicine at the National Capital Region Institute of Medical Sciences (NCRIMS), Meerut, Uttar Pradesh. The study was carried out over a period of one years, from July 2024 to June 2025, after obtaining approval from the Institutional Ethics Committee.

A total of 50 inpatients diagnosed with chronic liver disease (CLD) and admitted to the medical wards were included in the study. Diagnosis of CLD was based on a compatible clinical profile (signs of liver cell failure or reduced liver span) along with biochemical evidence (altered liver function tests, reversal of albumin-globulin ratio) or sonographic findings (altered echotexture of liver). Patients aged above 60 years, those with overt renal failure (serum creatinine >1.5 mg/dl), known primary renal disease, diabetes mellitus, hypertension, grade 4 hepatic encephalopathy, or recent gastrointestinal bleed were excluded.

Data regarding demographic variables such as age, sex, and weight, along with clinical features including presenting complaints (ascites, jaundice, encephalopathy, and history of alcoholism), and findings of liver cell failure were recorded using a structured proforma. All patients underwent a detailed clinical examination and baseline investigations. Diuretics were withheld for three days prior to laboratory assessments. Laboratory investigations included complete liver function tests, renal function tests, viral markers for hepatitis B, urine analysis, 24-hour urine volume, and urine creatinine.

Ultrasound examination of the abdomen was performed to assess liver echotexture, size, presence of splenomegaly, portal hypertension, ascites, and any renal pathology. Creatinine clearance was calculated using two methods:

1. **24-hour urine creatinine method** – by the formula:
(Urine creatinine × 24-hour urine volume) / Serum creatinine,
with the result expressed in ml/minute after dividing by 1440.
2. **Cockcroft–Gault formula (CGF)** – calculated as:
(140−Age)×Weight/(Serumcreatinine×72)(140 − Age) × Weight / (Serum creatinine × 72)(140−Age)×Weight/(Serumcreatinine×72),
with the value multiplied by 0.85 for female patients.

Serum creatinine levels were compared with creatinine clearance calculated by these two methods to determine their relative usefulness in detecting renal dysfunction in CLD patients. The relationship between the etiology of chronic liver disease (alcoholic, viral, or other causes) and the presence of renal dysfunction was also evaluated.

Data were entered into Microsoft Excel and analyzed using SPSS version 25. Descriptive statistics such as mean and standard deviation were used for continuous variables, while frequencies and percentages were used for categorical variables. Associations between categorical variables were assessed using chi-square test, and mean differences were analyzed using Student's t-test or ANOVA where appropriate. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 50 patients with chronic liver disease were included in the study. The mean age of the patients was 46.8 years, with the majority belonging to the 41–60 years age group. Males constituted 70% of the study population, and a history

of alcohol consumption was present in 58% of the cases. Viral hepatitis B infection was identified in 24%, while other etiologies such as non-alcoholic fatty liver disease accounted for 18%. Clinical features observed included ascites in 76% of patients, jaundice in 68%, splenomegaly in 54%, and hepatic encephalopathy in 22%.

With regard to renal function, the mean serum creatinine among the study population was 1.1 mg/dl, and none of the patients had overt renal failure at baseline. When creatinine clearance was calculated using the 24-hour urine method, 38% of patients showed reduced clearance values (<90 ml/min), whereas the Cockcroft–Gault formula identified 42% with reduced renal function. A significant discrepancy was noted between serum creatinine values and creatinine clearance estimates, indicating that reliance on serum creatinine alone underestimated the degree of renal dysfunction in patients with CLD.

When stratified by etiology, renal dysfunction was more common in alcoholic cirrhosis (48%) compared to viral hepatitis–related CLD (33%) and other causes (22%). Statistical analysis revealed a significant association between the etiology of CLD and the presence of renal dysfunction ($p < 0.05$). These findings suggest that both serum creatinine and creatinine clearance are useful in evaluating renal function, but clearance-based methods provide a more sensitive measure of impairment, particularly in alcohol-related liver disease.

Table 1: Socio-Demographic Characteristics of Patients with Chronic Liver Disease (n = 50)

Variable	Category	Frequency (n)	Percentage (%)
Age group (years)	≤30	6	12.0
	31–40	10	20.0
	41–60	22	44.0
	>60	12	24.0
Gender	Male	35	70.0
	Female	15	30.0
Etiology of CLD	Alcoholic	29	58.0
	Viral Hepatitis B	12	24.0
	Others (NAFLD, etc.)	9	18.0

Table 2: Clinical Profile of Patients with Chronic Liver Disease (n = 50)

Clinical Feature	Present (n)	Percentage (%)
Ascites	38	76.0
Jaundice	34	68.0
Splenomegaly	27	54.0
Hepatic encephalopathy	11	22.0
History of alcoholism	29	58.0

Table 3: Renal Function Parameters in Patients with Chronic Liver Disease (n = 50)

Renal Function Parameter	Normal n (%)	Abnormal n (%)	Mean ± SD
Serum Creatinine (<1.5)	50 (100.0)	0 (0.0)	1.1 ± 0.2 mg/dl
Creatinine Clearance (24 hr) ≥90 ml/min	31 (62.0)	19 (38.0)	88.6 ± 14.5 ml/min
Cockcroft–Gault Formula ≥90 ml/min	29 (58.0)	21 (42.0)	85.2 ± 16.3 ml/min
Renal dysfunction by etiology			
Alcoholic CLD	15 (51.7%)	14 (48.3%)	—
Viral Hepatitis CLD	8 (66.7%)	4 (33.3%)	—
Others	7 (77.8%)	2 (22.2%)	—

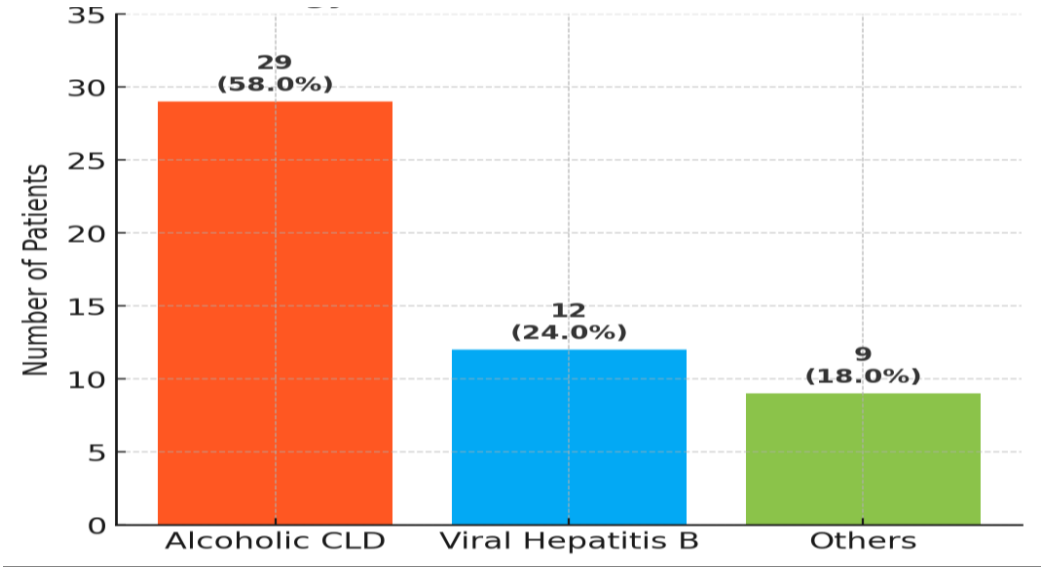

Figure 1: Etiology of Chronic Liver Disease (n= 50)

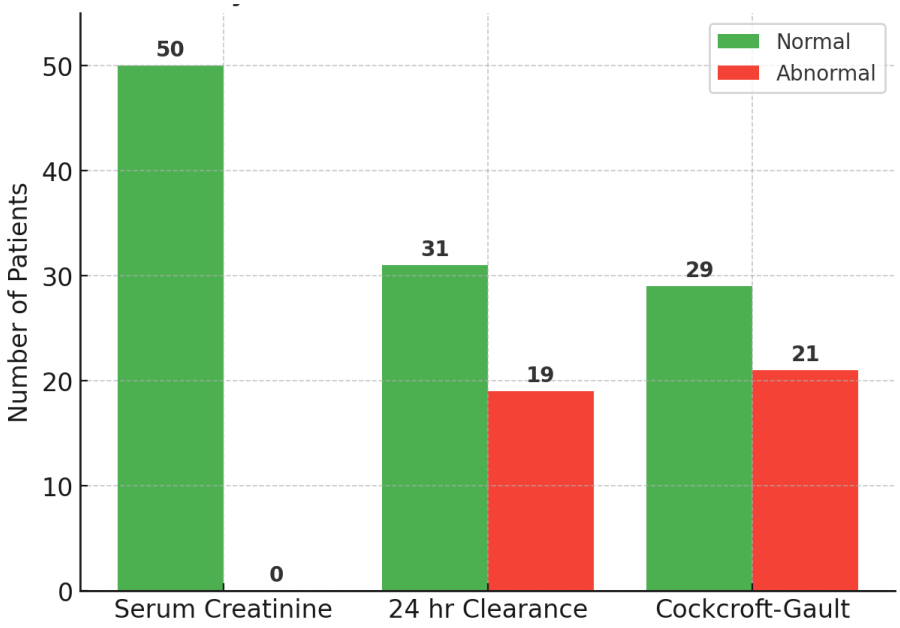

Figure 2: Renal Dysfunction Detection in CLD Patients (n= 50)

DISCUSSION

In this study, renal dysfunction was observed in 38% of CLD patients when assessed using 24-hour creatinine clearance and in 42% using the Cockcroft–Gault formula, whereas serum creatinine alone failed to detect abnormalities in most cases. This highlights the well-documented limitation of serum creatinine as a marker of renal impairment in patients with chronic liver disease. Several international studies have reported similar discrepancies. Sherman et al. demonstrated that serum creatinine substantially underestimates renal dysfunction in cirrhotic patients due to decreased muscle mass and impaired creatine synthesis [9]. Caregaro et al. also showed that GFR measured by creatinine clearance was more sensitive than serum creatinine in detecting subclinical renal impairment in cirrhotic patients [10]. These findings are consistent with the present study, underscoring the need for clearance-based methods in assessing renal function in CLD. The mean serum creatinine in this study was 1.1 mg/dl, despite nearly two-fifths of patients having reduced creatinine clearance. This phenomenon has been described by Angeli et al., who emphasized that even serum creatinine levels within the normal range may not exclude significant renal dysfunction in cirrhosis [11]. Similarly, a study conducted in South India by John et al. found that nearly 30% of cirrhotic patients with normal serum creatinine had reduced creatinine clearance, confirming the inadequacy of serum creatinine alone in such patients [12]. These findings align with the present results, indicating that reliance solely on serum creatinine may delay recognition of renal impairment.

In the current study, renal dysfunction was more prevalent in patients with alcoholic cirrhosis (48%) compared to those with viral hepatitis–related CLD (33%) and other causes (22%). This pattern has been previously reported in the

literature. In a study from Italy, Bernardi et al. observed higher rates of renal impairment among alcohol-related cirrhotics, attributing this to combined effects of hepatotoxicity, malnutrition, and systemic hemodynamic changes [13]. In India, a study by Sheth et al. also found that renal abnormalities were significantly more common in alcohol-related cirrhosis compared to viral etiologies [14]. Regionally, a study conducted in Uttar Pradesh by Srivastava et al. reported similar findings, with alcohol being the predominant factor associated with renal impairment in CLD [15]. These results suggest that etiology plays a contributory role in determining renal dysfunction in liver disease.

The presence of clinical features such as ascites (76%) and jaundice (68%) in the present study is consistent with decompensated liver disease, which is a known predictor of renal impairment. A study by Arroyo et al. demonstrated that the development of ascites and portal hypertension is closely linked to reduced effective arterial blood volume, precipitating renal dysfunction [16]. Indian studies by Singh et al. and Chawla et al. similarly reported that the presence of ascites and advanced Child-Pugh class correlated strongly with reduced renal clearance [17,18]. This aligns with the present study's observation that a significant proportion of patients with ascites had lower clearance values.

The findings of this study reaffirm that creatinine clearance, whether calculated by 24-hour urinary excretion or estimated by Cockcroft–Gault formula, is more sensitive than serum creatinine in detecting early renal dysfunction in CLD. Moreover, the association of renal impairment with alcoholic etiology highlights the importance of stratifying risk based on causative factors. Early identification and monitoring of renal function abnormalities are crucial for prognosis, as renal dysfunction is a major determinant of mortality in chronic liver disease and can complicate liver transplantation outcomes.

CONCLUSION

The present study demonstrated that serum creatinine alone is an unreliable marker for detecting renal dysfunction in patients with chronic liver disease, as it underestimated impairment compared to creatinine clearance measurements. While none of the patients had overt renal failure by serum creatinine criteria, nearly 40% were identified as having reduced renal function using 24-hour urinary creatinine clearance and the Cockcroft–Gault formula. This discrepancy underscores the importance of using clearance-based methods for early detection of renal impairment in cirrhotic patients.

Renal dysfunction was more prevalent in alcoholic cirrhosis compared to viral hepatitis and other etiologies, highlighting the contributory role of alcohol in worsening renal outcomes in CLD. Clinical features such as ascites and jaundice were common among patients with impaired clearance, supporting the close relationship between decompensated hepatic function and renal impairment. Early recognition and monitoring of renal abnormalities in CLD patients are crucial, as renal dysfunction significantly worsens prognosis and impacts survival.

LIMITATIONS AND RECOMMENDATIONS

This study was limited by its relatively small sample size and single-centre design, which may restrict the generalizability of the findings. The cross-sectional nature of the study prevented assessment of longitudinal changes in renal function or the prognostic implications of renal impairment over time. Measurement of glomerular filtration rate by gold-standard methods such as inulin clearance was not feasible, and hence creatinine-based estimates were relied upon.

Despite these limitations, the study highlights important clinical implications. It is recommended that in patients with chronic liver disease, renal function should be routinely evaluated not only with serum creatinine but also with creatinine clearance, either by 24-hour urine collection or Cockcroft–Gault estimation. Special attention should be given to patients with alcoholic cirrhosis, who are at greater risk of renal dysfunction. Larger multicentric prospective studies are needed to validate these findings and explore the prognostic significance of early renal impairment in CLD. Strengthening protocols for routine renal monitoring in cirrhotic patients may help in early detection of complications such as hepatorenal syndrome and improve overall outcomes.

REFERENCES

1. World Health Organization. Global status report on alcohol and health 2018. Geneva: World Health Organization; 2018.
2. Ginès P, Schrier RW. Renal failure in cirrhosis. N Engl J Med. 2009;361(13):1279–90.
3. Sherman DS, Fish DN, Teitelbaum I. Assessing renal function in cirrhotic patients: Problems and pitfalls. Am J Kidney Dis. 2003;41(2):269–78.
4. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron. 1976;16(1):31–41.
5. Caregaro L, Menon F, Angeli P, Amodio P, Merkel C, Bortoluzzi A, et al. Limitations of serum creatinine level and creatinine clearance as filtration markers in cirrhosis. Arch Intern Med. 1994;154(2):201–5.
6. Sheth RA, Nair S, Kamath PS. Renal dysfunction in cirrhosis: Etiology and impact on survival. Indian J Gastroenterol. 2005;24(5):221–5.

7. Lai KN, Li PK, Lui SF, Au TC, Tam JS, Tong KL, et al. Membranous nephropathy related to hepatitis B virus in adults. N Engl J Med. 1991;324(21):1457–63.
8. Duseja A. Spectrum and diagnosis of liver disease in India. Clin Liver Dis (Hoboken). 2014;3(4):140–2.
9. Sherman DS, Fish DN, Teitelbaum I. Assessing renal function in cirrhotic patients: Problems and pitfalls. Am J Kidney Dis. 2003;41(2):269–78.
10. Caregaro L, Menon F, Angeli P, Amodio P, Merkel C, Bortoluzzi A, et al. Limitations of serum creatinine level and creatinine clearance as filtration markers in cirrhosis. Arch Intern Med. 1994;154(2):201–5.
11. Angeli P, Merkel C. Pathogenesis and management of hepatorenal syndrome in cirrhosis. J Hepatol. 2008;48(Suppl 1):S93–103.
12. John M, Peter JV, Jacob CK, Cherian AM. Assessment of renal function in cirrhotic patients: Serum creatinine versus creatinine clearance. Indian J Gastroenterol. 1996;15(2):54–6.
13. Bernardi M, Di Marco C, Trevisani F, Gasbarrini G. Renal function abnormalities in cirrhosis: Relationship with alcoholic etiology and portal hypertension. Hepatology. 1991;14(2):263–9.
14. Sheth RA, Nair S, Kamath PS. Renal dysfunction in cirrhosis: Etiology and impact on survival. Indian J Gastroenterol. 2005;24(5):221–5.
15. Srivastava A, Pandey R, Tripathi M. Renal abnormalities in cirrhosis: A clinical study in a tertiary care centre in Uttar Pradesh. J Assoc Physicians India. 2014;62(7):550–4.
16. Arroyo V, Ginès P, Gerbes AL, Dudley FJ, Gentilini P, Laffi G, et al. Definition and diagnostic criteria of refractory ascites and hepatorenal syndrome in cirrhosis. Hepatology. 1996;23(1):164–76.
17. Singh R, Sharma P, Chawla Y. Clinical correlates of renal dysfunction in decompensated cirrhosis. Trop Gastroenterol. 2002;23(3):117–9.
18. Chawla YK, Tandon RK, Vasishta RK, Dilawari JB. Renal function in cirrhosis and its relation to severity of liver disease. Indian J Med Res. 1989;89:335–40.