



## Clinico-pathological profile and short-term outcomes of renal dysfunction in patients with cirrhosis: A prospective observational study

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### Abstract

**Background & objectives:** Renal dysfunction commonly and seriously complicates cirrhosis. It increases morbidity, mortality, prolongs hospital stays, and raises the need for renal replacement therapy. It can present as acute kidney injury (AKI), chronic kidney disease (CKD), or hepatorenal syndrome (HRS). This study evaluated the clinico-pathological profile and short-term outcomes of renal dysfunction in patients with cirrhosis.

**Methods:** We conducted this prospective observational study in the Department of General Medicine, INHS Asvini, Mumbai, India, from October 2020 to September 2022. We enrolled 100 consecutive patients with cirrhosis and renal dysfunction. We recorded demographic, clinical, biochemical, and ultrasonographic parameters. We categorized renal dysfunction as AKI, CKD, or acute-on-chronic renal failure. We assessed dialysis requirement and in-hospital mortality as outcomes.

**Results:** The mean age of patients was  $47.72 \pm 7.99$  years. Males accounted for 55 percent of cases. Alcohol-related liver disease caused cirrhosis in 61 percent of cases. This was followed by hepatitis B infection (19 percent) and non-alcoholic steatohepatitis (14 percent). Most patients had advanced liver disease. Seventy-six percent belonged to Child-Pugh grade III. AKI was the most common renal dysfunction (70 percent), followed by CKD (20 percent), and acute-on-chronic renal failure (10 percent). Among AKI cases, pre-renal AKI was most frequent (40 percent), followed by acute tubular necrosis (24 percent), and HRS (6 percent). Dialysis was required in 12 percent of patients. Overall mortality was 24 percent. It was highest among cases of CKD (40 percent), HRS (33.3 percent), and acute tubular necrosis (29.2 percent). Mortality increased significantly with worsening Child-Pugh grade ( $P < 0.01$ ).

**Interpretation & conclusions:** Renal dysfunction often occurs in cirrhosis, with AKI being the leading cause. Preventable triggers like gastrointestinal bleeding, infections, and diuretic overuse were frequent. CKD, acute tubular necrosis, and advanced liver disease were linked to worse outcomes. Early detection and management may improve prognosis.

**Keywords:** Cirrhosis, acute kidney injury, chronic kidney disease, hepatorenal syndrome, mortality

### Introduction

Cirrhosis is the end stage of chronic liver disease. Causes include alcohol misuse, viral hepatitis, non-alcoholic fatty liver disease, autoimmune liver disorders, and metabolic diseases. Cirrhosis leads to high rates of illness and death worldwide. Progressive hepatic fibrosis causes portal hypertension and liver insufficiency. This makes patients prone to many systemic complications<sup>[1, 2]</sup>.

Renal dysfunction is one of the most severe complications of cirrhosis and is associated with poor survival. It may present as acute kidney injury (AKI), chronic kidney disease (CKD), or hepatorenal syndrome (HRS). Even minor increases in serum creatinine have been shown to adversely affect outcomes in cirrhotic patients<sup>[3, 4]</sup>.

The pathophysiology of renal dysfunction in cirrhosis is complex and involves many factors. Portal hypertension causes marked splanchnic vasodilatation and reduces effective arterial blood volume. This reduction activates the renin-angiotensin-aldosterone and sympathetic nervous systems. Resulting renal vasoconstriction worsens kidney function. Infections, gastrointestinal bleeding, excessive diuretic use, dehydration, and nephrotoxic drugs also cause kidney injury<sup>[5, 6, 7]</sup>.

Acute kidney injury is the most common form of renal dysfunction in hospitalized cirrhotic patients. Reported incidence ranges from 20 to 50 percent. Even small

increases in serum creatinine worsen outcomes. This highlights the need for early diagnosis and intervention<sup>[8]</sup>

Hepatorenal syndrome is a distinct type of renal failure seen in advanced cirrhosis with ascites. It features severe renal vasoconstriction but no structural kidney damage. HRS carries a high mortality if not treated. Vasoconstrictor therapy and albumin can improve outcomes, but liver transplantation is definitive<sup>[9, 10]</sup>.

In India, alcohol-related liver disease is a leading cause of cirrhosis. Many patients present late with decompensated disease. Prospective Indian data on the spectrum and short-term outcomes of renal dysfunction in cirrhosis are limited. This study evaluated clinico-pathological features and short-term outcomes in patients with cirrhosis admitted to a tertiary care center.

### Material and Methods

The Department of General Medicine at INHS Asvini, Colaba, Mumbai, conducted this prospective observational study from October 2020 to September 2022. We included 100 consecutive patients who presented with renal dysfunction in the setting of liver cirrhosis and gave informed written consent. We enrolled patients with chronic liver disease and compatible clinical features, such as signs of liver cell failure or reduced liver span, supported by

biochemical evidence (including altered liver function tests or reversal of the albumin-globulin ratio) and/or sonographic evidence of altered liver echotexture. Additional inclusion criteria included serum creatinine >1.3 mg/dL, acute kidney injury (AKI) as defined by KDIGO, urinary abnormalities such as hematuria, active urinary sediments or proteinuria >500 mg/day, and evidence of renovascular or structural kidney disease. We excluded patients with pre-existing chronic renal insufficiency with acute worsening of renal function, prerenal azotemia, acute glomerulonephritis, renal failure following renal transplantation, jaundice due solely to hemolysis, or acute renal and hepatic failure as part of multiorgan failure in intensive care unit patients.

After obtaining ethical committee approval, all participants were informed of the study's purpose and procedures and then provided written consent. Detailed demographic, clinical, and laboratory data were prospectively recorded in a predesigned proforma. Documented variables included age, sex, type and cause of renal dysfunction (e.g., volume depletion, hemorrhage, infection, or nephrotoxic drugs), etiology of liver disease, including viral markers, and severity of liver dysfunction (Child-Pugh grading). Other data included urine output, peak serum creatinine, urinary sodium, creatinine, osmolality, need for kidney or liver biopsy where indicated, treatment modalities (including dialysis requirement and type), associated complications, and final outcome, including mortality. All patients underwent abdominal ultrasonography to assess liver size, liver echotexture, splenomegaly, portal hypertension, ascites, and renal abnormalities.

Renal disorders were classified as acute renal failure, chronic kidney disease (including nephrotic and nephritic syndrome), and acute-on-chronic renal failure. Acute renal failure cases were further divided into prerenal failure, hepatorenal syndrome (HRS), and acute tubular necrosis (ATN). Acute renal failure was defined as an absolute increase in serum creatinine of  $\geq 0.3$  mg/dL, or as a  $\geq 50\%$  rise from baseline ( $\geq 1.5$ -fold), in the absence of pre-existing kidney disease. Chronic kidney disease was defined as evidence of kidney damage for at least 3 months. This could occur with or without a reduced glomerular filtration rate. Other features included persistent proteinuria, abnormal urinary sediment, abnormal imaging findings, or GFR  $< 60$  mL/min/1.73 m<sup>2</sup> for at least 3 months. Acute-on-chronic renal failure was defined as a  $\geq 50\%$  increase in serum creatinine from baseline in patients with known kidney disease. Nephrotic syndrome was defined as urinary protein excretion  $> 3.5$  g/day and serum albumin  $< 3$  g/dL. Hepatorenal syndrome was defined as serum creatinine  $> 1.5$  mg/dL in patients with cirrhosis and ascites. This diagnosis required no improvement after 48 hours of diuretic withdrawal and volume expansion, and the absence of shock, nephrotoxic drug exposure, or intrinsic renal disease. All data were analyzed with SPSS version 21.0. Microsoft Excel 2010 was used for making graphs. Qualitative variables were shown as frequencies and percentages. Associations were tested by Chi-square test. Quantitative variables were given as mean  $\pm$  standard deviation. The unpaired t-test was used for normally distributed data, and Mann-Whitney U test for non-normally distributed data. A p-value below 0.05 was statistically significant.

## Results

A total of 100 patients with chronic liver disease and renal dysfunction were included in the study. The mean age was  $47.72 \pm 7.99$  years. Most patients were 40 years old or younger (41 percent), 41–50 years old (30 percent), 51–60 years old (22 percent), and 61–70 years old (7 percent) (Table 1). Males were 55 percent of the population; females were 45 percent (Table 2).

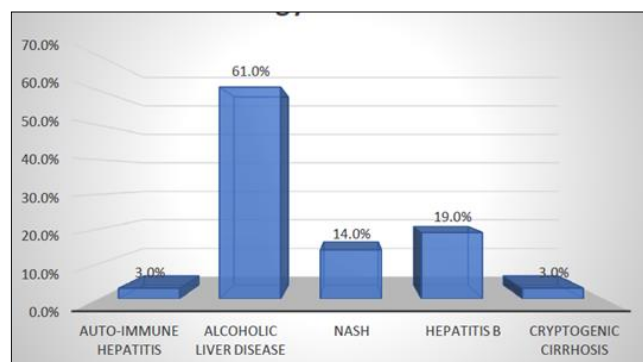
**Table 1:** Distribution of study groups as per age group

| Age Group                        | N   | %      |
|----------------------------------|-----|--------|
| <b><math>\leq 40</math></b>      | 41  | 41.0%  |
| <b>41-50</b>                     | 30  | 30.0%  |
| <b>51-60</b>                     | 22  | 22.0%  |
| <b>61-70</b>                     | 7   | 7.0%   |
| <b>Total</b>                     | 100 | 100.0% |
| <b>Mean Age - 47.72 +/- 7.99</b> |     |        |

**Table 2:** Distribution of study groups as per gender

| Gender        | N   | %      |
|---------------|-----|--------|
| <b>Female</b> | 45  | 45.0%  |
| <b>Male</b>   | 55  | 55.0%  |
| <b>Total</b>  | 100 | 100.0% |

Alcoholic liver disease was the most common cause of cirrhosis, seen in 61 percent of cases. Hepatitis B infection caused 19 percent of cases. Non-alcoholic steatohepatitis (NASH) caused 14 percent, autoimmune hepatitis caused 3 percent, and cryptogenic cirrhosis caused 3 percent (Figure 1).



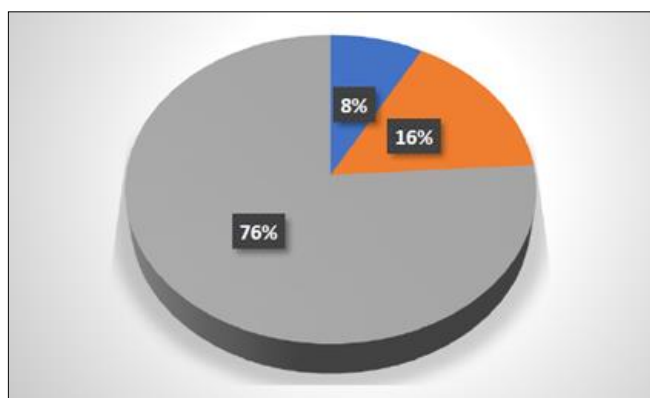
**Fig 1:** Etiology of Cirrhosis

The most frequent clinical features were pallor (70%), jaundice (52%), pedal edema (50%), malena (46%), hematemesis (43%), hepatic encephalopathy (23%), and bleeding per rectum in 20% of cases (Table 3).

**Table 3:** Distribution of study groups as per clinical features

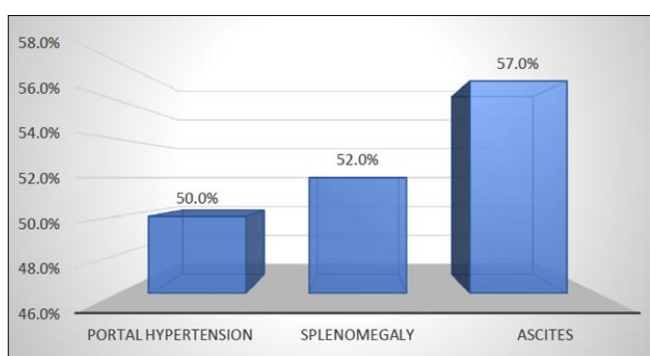
| Clinical Features      | N  | %     |
|------------------------|----|-------|
| Jaundice               | 52 | 52.0% |
| Pallor                 | 70 | 70.0% |
| Pedal edema            | 50 | 50.0% |
| Hematemesis            | 43 | 43.0% |
| Malena                 | 46 | 46.0% |
| Bleeding PR            | 20 | 20.0% |
| Hepatic Encephalopathy | 23 | 23.0% |

Based on Child-Pugh grading, 8% of patients were classified as Grade I, 16% as Grade II, and the majority (76%) as Grade III, indicating advanced liver disease in most patients (Figure 2).



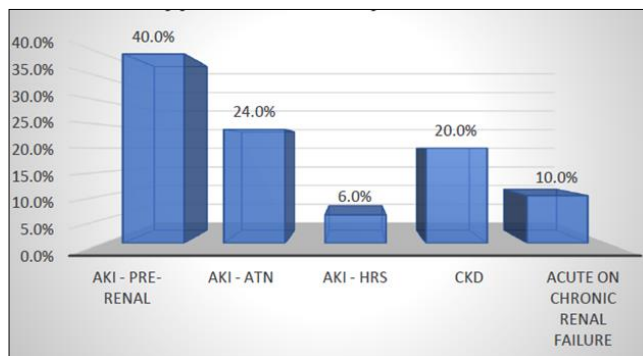
**Fig 2:** Distribution of study groups as per Child Pugh Grade

Ultrasonographic findings revealed ascites in 57% of patients, splenomegaly in 52%, and portal hypertension in 50% (Figure 3).



**Fig 3:** Distribution of study groups as per USG findings

Regarding renal dysfunction, acute kidney injury (AKI) was the most common presentation, observed in 70% of patients. Among these, pre-renal AKI was most common (40%), followed by acute tubular necrosis (ATN) in 24% and hepatorenal syndrome (HRS) in 6%. Chronic kidney disease (CKD) was present in 20% of patients, while acute-on-chronic renal failure was noted in 10% (Figure 4).



**Fig 4:** Type of Renal Dysfunction

Among patients with pre-renal AKI, the most common precipitating factors were gastrointestinal bleed (24 cases) and diuretic use (14 cases), while septic shock accounted for 2 cases. In patients with ATN, spontaneous bacterial peritonitis (SBP) was the most common cause (12 cases), followed by nephrotoxic drugs (5 cases), urinary tract infection (5 cases), and gastrointestinal bleed (2 cases). Among HRS patients, SBP was responsible in 5 cases, while 1 case occurred spontaneously. CKD was mainly due to diabetic nephropathy (7 cases), mesangio-proliferative glomerulonephritis (6 cases), membranoproliferative glomerulonephritis (2 cases), and unexplained causes (5 cases). In acute-on-chronic renal failure, gastrointestinal bleed was the leading precipitating factor (5 cases), followed by diuretic use (3 cases) and loose motions (2 cases) (Table 4).

**Table 4:** Precipitating factors for the development of renal dysfunction in cirrhosis cases

| Type of Renal Dysfunction             | Precipitating Factor/ Etiology | N  |
|---------------------------------------|--------------------------------|----|
| ARF - Pre-renal (n=40)                | GI Bleed                       | 24 |
|                                       | Diuretics                      | 14 |
|                                       | Septic shock                   | 2  |
| AKI - ATN (n=24)                      | GI Bleed                       | 2  |
|                                       | Nephrotoxic drugs              | 5  |
|                                       | SBP                            | 12 |
|                                       | UTI                            | 5  |
| ARF - HRS (n=6)                       | SBP                            | 5  |
|                                       | Spontaneous                    | 1  |
| CKD (n=20)                            | Diabetic Nephropathy           | 7  |
|                                       | MPGN                           | 2  |
|                                       | Mesangio-proliferative GN      | 6  |
|                                       | Unexplained                    | 5  |
| Acute on Chronic Renal Failure (n=10) | GI Bleed                       | 5  |
|                                       | Loose Motions                  | 2  |
|                                       | Diuretics                      | 3  |

Dialysis was required in 12% of patients. It was most commonly needed in patients with ATN (20.8%), acute-on-chronic renal failure (20%), CKD (15%), and pre-renal AKI (5%), while none of the HRS patients required dialysis (Table 5).

**Table 5:** Association of type of renal dysfunction and requirement of dialysis

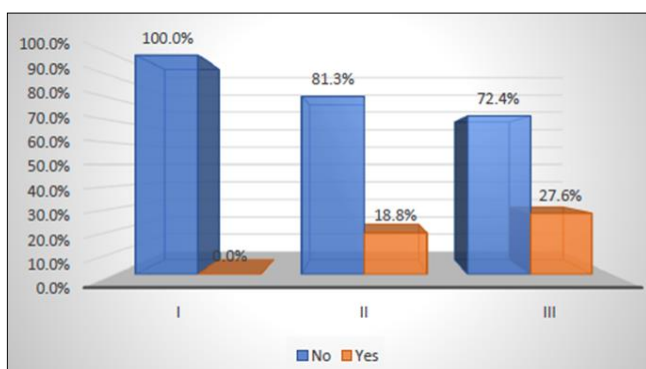
| Type of Renal Dysfunction      | N          | Dialysis  | %            |
|--------------------------------|------------|-----------|--------------|
| ARF - Pre-renal                | 40         | 2         | 5.0%         |
| AKI - ATN                      | 24         | 5         | 20.8%        |
| CKD                            | 20         | 3         | 15.0%        |
| Acute on Chronic Renal failure | 10         | 2         | 20.0%        |
| AKI - HRS                      | 6          | 0         | 0.0%         |
| <b>Total</b>                   | <b>100</b> | <b>12</b> | <b>12.0%</b> |

Overall mortality was 24% (Table 5). Mortality was highest among CKD patients (40%), followed by HRS (33.3%), ATN (29.2%), pre-renal AKI (15%), and acute-on-chronic renal failure (10%) (Table 6).

**Table 6:** Association of type of renal dysfunction and mortality

| Type of Renal Dysfunction      | N          | Mortality | %            |
|--------------------------------|------------|-----------|--------------|
| AKI - Pre-renal                | 40         | 6         | 15.0%        |
| AKI - ATN                      | 24         | 7         | 29.2%        |
| CKD                            | 20         | 8         | 40.0%        |
| Acute on Chronic Renal failure | 10         | 1         | 10.0%        |
| AKI - HRS                      | 6          | 2         | 33.3%        |
| <b>Total</b>                   | <b>100</b> | <b>24</b> | <b>24.0%</b> |

A statistically significant association was observed between Child-Pugh grade and mortality ( $p < 0.01$ ). Mortality rates increased with worsening liver disease severity, being 0% in Grade I, 18.8% in Grade II, and 27.6% in Grade III patients (Figure 5).



**Figure 5:** Association of Child Pugh grade and mortality

## Discussion

Chronic liver disease (CLD) is a major global health problem, and cirrhosis represents the final common pathway of multiple hepatic insults. Renal dysfunction is one of the most severe complications in cirrhosis and is associated with prolonged hospitalization, need for renal replacement therapy, and increased mortality. Early identification of renal impairment in cirrhotic patients is therefore clinically important [11, 12].

In the present study, the mean age of patients was  $47.72 \pm 7.99$  years, with male predominance (55%). Similar demographic findings were reported by Jai Prakash *et al.*, who observed that cirrhosis commonly affects middle-aged adults with higher prevalence among males, particularly in alcohol-related liver disease. The male predominance in our cohort may be attributed to the high burden of alcohol consumption among men in India [13].

Most patients in this study presented with pallor (70%), jaundice (52%), pedal edema (52%), ascites (50%), portal hypertension (57%), and splenomegaly (52%), suggesting advanced liver disease at presentation. A large proportion of patients belonged to Child-Pugh class C (76%), indicating decompensated cirrhosis. Similar observations have been reported in hospital-based studies in India, where delayed presentation and advanced disease are common. Jai Prakash *et al.* also demonstrated an increasing frequency of renal dysfunction with worsening Child-Pugh class [3].

Alcoholic liver disease was the commonest etiology in our study (61%), followed by hepatitis B infection (19%) and non-alcoholic steatohepatitis (14%). This is consistent with data from India, where alcohol has emerged as the leading cause of cirrhosis in several tertiary care centers. However, etiological patterns vary globally, with viral hepatitis being more common in some Asian and African populations [3].

Acute kidney injury (AKI) was the most frequent renal abnormality in our study (70%), followed by chronic kidney disease (20%) and acute-on-chronic renal failure (10%). Similar results were reported by Wong *et al.* and other investigators, who found AKI to be the predominant form of renal dysfunction in hospitalized cirrhotic patients [14].

Among AKI cases, pre-renal AKI was the most common subtype (40%), followed by acute tubular necrosis (24%) and hepatorenal syndrome (6%). Our findings differ slightly from Jai Prakash *et al.*, who reported ATN as the most common AKI subtype (44.4%), followed by pre-renal failure (36.4%) and HRS (19.2%). This variation may be related to differences in patient selection, severity of liver disease, sepsis prevalence, and timing of presentation [3].

The common precipitating factors for renal dysfunction in our study were gastrointestinal bleeding, diuretic use, and spontaneous bacterial peritonitis. These findings agree with previous literature showing that hypovolemia, infections, overdiuresis, and circulatory dysfunction are major triggers for AKI in cirrhosis [4].

Dialysis was required in 12% of patients, reflecting substantial renal morbidity. Overall mortality in our study was 24%, with highest mortality observed in CKD, hepatorenal syndrome, and ATN. Mortality also increased significantly with worsening Child-Pugh class. Similar associations between renal dysfunction, advanced cirrhosis, and poor survival have been documented by Jai Prakash *et al.* and other authors [3].

The present study highlights that renal dysfunction in cirrhosis is common and often precipitated by potentially preventable factors. Early recognition, prompt treatment of infections, careful use of diuretics, correction of hypovolemia, and close monitoring of high-risk patients may improve outcomes.

This study is limited by its single-center design, modest sample size, and short follow-up period. Larger multicentric prospective studies with long-term follow-up are required to validate these findings and assess long-term renal and survival outcomes.

## Conclusion

Renal dysfunction is frequent in cirrhosis, with AKI being the commonest presentation. Preventable triggers such as gastrointestinal bleeding, infections, and diuretic overuse were common. Advanced liver disease and intrinsic renal injury were associated with poor outcomes. Early recognition and timely intervention may improve survival.

## Acknowledgment

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## Financial support & sponsorship

Nil.

## Conflicts of Interest

None.

## Abbreviations

ADQI – Acute Dialysis Quality Initiative; AKI – Acute Kidney Injury; AKIN – Acute Kidney Injury Network; ALD – Alcoholic Liver Disease; ATN – Acute Tubular Necrosis; CKD – Chronic Kidney Disease; CLD – Chronic Liver Disease; CP – Child Pugh; CTP – Child Turcot Pugh; DM – Diabetes Mellitus; eGFR – Estimated Glomerular Filtration Rate; GFR – Glomerular Filtration Rate; GI Bleed – Gastrointestinal Bleed; GN – Glomerulonephritis; HBV – Hepatitis B Virus; HCC – Hepatocellular Carcinoma; HCV – Hepatitis C Virus; HE – Hepatic Encephalopathy; HRS – Hepato-renal Syndrome; HVPG – Hepatic Venous Pressure Gradient; INR – International Normalized Ratio; KDIGO – Kidney Diseases Improving Global Outcome; LT – Liver Transplant; MDRD – Modification of Diet in Renal Diseases; MPGN – Membranoproliferative Glomerulonephritis; NAFLD – Non-alcoholic Fatty Liver Disease; NASH – Non-alcoholic Steatohepatitis; OGD – Oro-gastro Duodenoscopy; PHTN – Portal Hypertension; RAAS – Renin Angiotensin Aldosterone System; SBP – Spontaneous Bacterial Peritonitis; sCr – Serum Creatinine; SIRS – Systemic Inflammatory Response Syndrome; SKLT – Simultaneous Kidney and Liver Transplant; TIPS – Trans-jugular Intrahepatic Portosystemic Shunt; USG – Ultrasonography; UTI – Urinary Tract Infection.

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