Original Article

Acute Kidney Injury in Chronic Liver Disease in Northwest India: Still a Battle to Conquer

Abstract

Background: Patients with cirrhosis are susceptible to development of acute kidney injury (AKI), which leads to poor outcome. We conducted a study to evaluate the spectrum of AKI in patients with cirrhosis. **Materials and Methods:** This study was conducted in consecutive cirrhotic patients with AKI admitted in a tertiary care center of India from April 2020 to December 2022. Details including history, examination findings, and results of laboratory investigations were recorded. **Results:** A total of 243 patients were enrolled in this study. The majority (91.3%) of the patients were males. The most common etiology of cirrhosis was alcohol in 58.4% (n = 142) followed by hepatitis B in 10.3% (n = 25) of patients. Pre-renal form of AKI was present in 54.4% (n = 132) of patients and hepatorenal syndrome (HRS) in 21.8% (n = 53) of patients. IgA nephropathy was the commonest (n = 6) glomerular pathology in nonresponders with intrinsic renal disease. Majority of the patients belonged to stage II (46.9%) and stage I AKI (37%), while only 16.1% had stage III AKI. Various stages of AKI showed a significant correlation ($P < 0.05$) with Child–Turcotte–Pugh (CTP) score and Model for End-stage Liver Disease (MELD)-Na score. The overall in-hospital mortality rate was found to be 18.5% (n = 45). **Conclusion:** Renal dysfunction is a frequent complication among cirrhotic patients. Pre-renal factors were the most common cause of AKI in cirrhotics. Stages of AKI showed significant correlation with liver prognostic scores. Renal biopsy should be considered in patients not responding to treatment, to guide further management.

Keywords: *Acute kidney injury, Biopsy, Cirrhosis, Ethanol, Glomerular*

Disha Saxena[1], Manoj Yadav[2], Tarun Kumar[1], Sanjeev Sharma[1], Pankaj Beniwal[1], Vinay Malhotra[1], Dhananjai Agarwal[1], Sandeep Nijhawan[2]

Departments of [1]Nephrology and [2]Gastroenterology, SMS Medical College and Hospital, Jaipur, Rajasthan, India

Corresponding author:
Sanjeev Sharma, Department of Nephrology, SMS Medical College and Hospital, Jaipur, Rajasthan, India. E-mail: sanjeevkamal71@gmail.com

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Introduction

Burden of liver cirrhosis is increasing globally as well as in Indian population.[1] This surge is probably because of increasing incidence of nonalcoholic steatohepatitis (NASH) and alcohol abuse.[1] Cirrhosis accounts for 1.16 million deaths per year worldwide, making it the 11th most common cause of death.[2] India accounts for one-fifth (18.3%) of all cirrhosis deaths globally.[3] Patients with cirrhosis are susceptible to renal dysfunction because of splanchnic vasodilation and substantial volume shift. In hospitalized patients, the prevalence of acute kidney injury (AKI) ranges from 20% to 50%.[4-6] AKI can occur in these patients because of pre-renal insult, intrinsic renal disease, postrenal factors, or due to hepatorenal syndrome (HRS-AKI).[7] Causes of intrinsic renal disease include acute tubular necrosis (ATN), glomerulopathies, tubulointerstitial pathologies, and vascular pathologies. Glomerular diseases commonly reported in cirrhotic patients include IgA nephropathy, membranous nephropathy, and membranoproliferative glomerulonephritis (MPGN). The exact frequency of each cause in patients with cirrhosis has not been described yet. Identification of underlying etiology is important as treatment differs substantially. Pre-renal AKI (PRA) responds well to plasma volume expansion, whereas HRS-AKI and ATN require specific treatment approaches and are associated with high mortality and morbidity.[7,8] The new criteria from the International Ascites Club provide a simple and prognostically relevant staging system for AKI in cirrhosis depending on the relative increases in serum creatinine.[7,8] As per these criteria, AKI in cirrhosis is defined by an increase in serum creatinine of 0.3 mg/dl (26.4 μmol/l) in <48 h, or a 50% increase in serum creatinine from a baseline within

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≤3 months. Furthermore, as per the International club of ascites ICA-AKI criteria, AKI has been classified into three stages (1–3) depending on the intensity of rise in serum creatinine.[9,10] This staging system correlates well with the prognosis in patients with cirrhosis, with stages 2 and 3 having a poor outcome compared to stage 1.[9,11] Majority of the studies have found pre-renal injury (70%) as the most common cause of AKI in cirrhotics, followed by intrinsic renal causes (30%) and postrenal factors (<1%).[12,13] History of ongoing gastrointestinal losses like diarrhea/vomiting responsible for hypovolemia, treatment history, and history to rule out infections like cellulitis, etc., should be obtained. Depending upon the history and careful assessment of patients, further management should be planned to prevent morbidity and mortality. The present study was conducted to evaluate the risk factors and spectrum of AKI among patients with decompensated cirrhosis.

Materials and Methods

We conducted a prospective, observational study on cirrhotic patients with AKI admitted in a tertiary care center of India from April 2020 to December 2022. The study protocol was reviewed and approved from the SMS Medical College and attached Hospitals, Jaipur-302004. Rajasthan (India), with the approval number 376 MC/EC/2022. All cirrhotic patients with AKI admitted during the study period with age >18 years were included in this study. Patients with previously known renal disease, post-organ transplant patients, and patients with malignancy were excluded. A total of 368 patients were assessed for eligibility and out of them, 243 were included for the final analysis [Figure 1]. Demographic and clinical details of the patients, along with results of laboratory investigations were collected [Table 1]. Child–Turcotte–Pugh (CTP) score and Model for End-stage Liver Disease (MELD) score of patients were also noted. The study protocol was according to the ethical guidelines and was approved by the institutional committee.

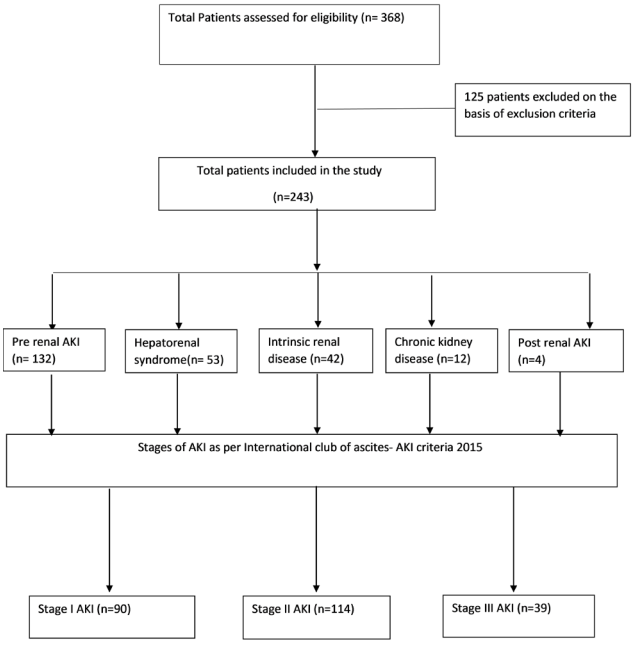

Figure 1: Consort flow diagram of the study. AKI: Acute Kidney Injury

Definitions

Cirrhosis of liver: Cirrhosis of liver was diagnosed by a gastroenterologist depending on the clinical features, laboratory parameters, liver imaging, endoscopy, and liver biopsy (if available).

AKI: The new 2015 ICA-AKI criteria[7] were used to diagnose AKI, which was categorized into three stages [Appendix 1].

Furthermore, patients with AKI were classified into PRA, HRS, postrenal AKI, and intrinsic renal disease (ATN/glomerular disease).

PRA: Patients of cirrhosis with AKI secondary to hypovolemia due to gastrointestinal bleeding, aggressive diuretic treatment, or lactulose-induced diarrhea are said to have PRA.

HRS: Patients were classified as having HRS based on 2015 ICA criteria for AKI,[7] which include the following: (1) presence of liver cirrhosis with ascites, (2) diagnosis of AKI based on the ICA-AKI criteria, (3) no improvement in serum creatinine after two consecutive days of plasma volume expansion with albumin 1 g/kg of body weight and withdrawal of diuretics, (4) absence of nephrotoxic drugs, (5) absence of shock, and (6) no macroscopic evidence of structural kidney damage, that is, absence of proteinuria (<500 mg/day), absence of

Table 1: Baseline demographic and laboratory characteristics of study participants

Characteristics	Total number of patients (*n* = 243)
Age (years)	47.85 ± 12.53
Male	222 (91.3%)
BMI (kg/m^2)	22.29 ± 1.84
Mid arm circumference (cm)	21.32 ± 1.38
Hemoglobin (g/dl)	8.10 ± 2.34
Urea (mg/dl)	89.67 ± 31.23
Creatinine (mg/dl)	2.59 ± 1.1
Serum protein (g/dl)	5.76 ± 1.09
Serum albumin (g/dl)	2.65 ± 0.42
Bilirubin (mg/dl)	7.34 ± 9.49
Serum lactate (mmol/l)	1.83 ± 1.32
Serum sodium (mEq/l)	131.87 ± 5.90
Serum potassium (mEq/l)	4.44 ± 0.88
Urine sodium (mEq/l)	54.79 ± 27.81
Urine potassium (mEq/l)	34 ± 14.18
CTP score	11.82 ± 2.37
MELD-Na score	30.19 ± 5.93

BMI: body mass index; CTP: Child Turcotte Pugh; MELD: Model for End-stage Liver Disease.

microhematuria (<50 red blood cells [RBCs]/high-power field), and normal findings on ultrasonography.

Intrinsic renal disease: Patients with intrinsic renal disease comprising glomerular disease and ATN were diagnosed after excluding PRA, HRS, and postobstructive cause of AKI. Diagnosis of AKI was reviewed and confirmed by nephrologists independently. Renal biopsies were performed in patients with active urinary sediments and nonresponders.

Statistical analysis

Normally distributed continuous variables were expressed as means and standard deviation, while non-normally distributed continuous variables were reported as median and interquartile range. Categorical variables were expressed as percentages. Significant parameters associated with the renal dysfunction were analyzed using binary logistic regression analysis. A *P*-value of less than 0.05 was considered statistically significant. Statistical Package for the Social Sciences (SPSS) 21 software was used for statistical analysis.

Results

A total of 243 patients were included, with 91.3% (n = 222) males, anda mean age of 47.85 years (±12.54). The most common etiology of cirrhosis was ethanol (58.4%), followed by chronic hepatitis B (10.3%) [Table 2].

The most common type of AKI was found to be PRA in 54.4% of patients, followed by HRS in 21.8%. Postrenal factors were bilateral ureteric obstruction in three patients and neurogenic bladder in one patient. Distribution of different types of AKI in this study is shown in Table 3 and Figure 2.

Table 2: Distribution of different etiologies of cirrhosis

Etiology of cirrhosis	Number (%)
Ethanol	142 (58.4%)
Hepatitis B	25 (10.3%)
Cryptogenic	23 (9.6%)
Ethanol + NASH	20 (8.2%)
NASH	18 (7.4%)
Ethanol + hepatitis B	6 (2.5%)
NASH + hepatitis B	3 (1.2%)
Hepatitis C	3 (1.2%)
Autoimmune hepatitis	2 (0.8%)
Wilson disease	1 (0.4%)

NASH: nonalcoholic steatohepatitis.

Table 3: Etiological classification of AKI

Type of AKI	Number (%)
Pre-renal AKI	132 (54.4%)
Hepatorenal syndrome	53 (21.8%)
Intrinsic renal disease	42 (17.3%)
Chronic kidney disease	12 (4.9%)
Postrenal AKI	4 (1.6%)

AKI: acute kidney injury.

Out of 42 patients with intrinsic renal disease, 26 patients recovered with conservative treatment and percutaneous renal biopsy was performed in 11 patients. Out of 11 patients [Figure 3], two patients were found to have severe ATN, two patients with hepatitis B had MPGN [Figure 4], and 1 had membranous nephropathy. The most common type of glomerular lesion was IgA nephropathy [Figure 5] in six patients. Renal biopsy could not be performed in rest of the nonresponders because of various contraindications.

Majority of the patients had stage II (46.9%) and stage I (37%) AKI, while only 16.1% of the patients were in stage III AKI. Table 3 shows the comparison of general characteristics, laboratory parameters, and outcome of patients with cirrhosis with different stages of AKI.

Mean values of total leukocyte count and serum creatinine were significantly different in various stages of AKI ($P < 0.05$). Stages of AKI showed a significant association ($P < 0.05$) with CTP score and MELD-Na score [Table 4].

Sixty-four patients (26.33%) required renal replacement therapy. Thirty-five patients received one or more sessions of intermittent hemodialysis, 22 patients underwent acute peritoneal dialysis, and sustained low efficiency dialysis was

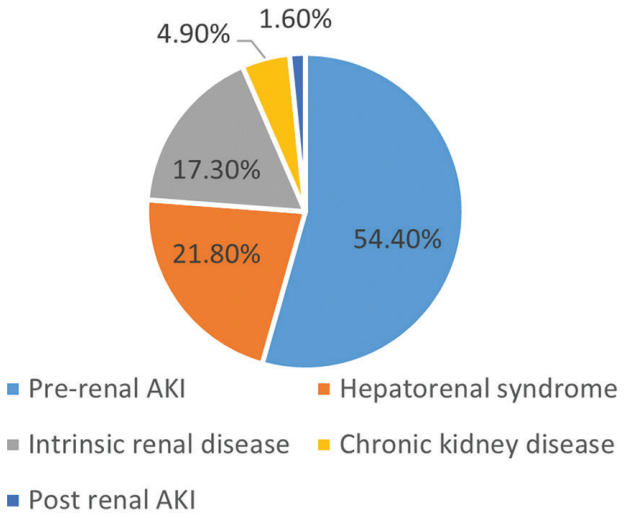

Figure 2: Etiological classification of cirrhosis. AKI: Acute kidney injury.

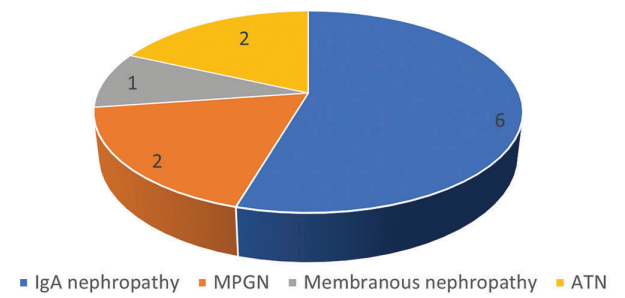

Figure 3: Histopathologic findings on renal biopsy of nonresponders. ATN: Acute tubular necrosis; MPGN: Membranoproliferative glomerulonephritis; IgA: Immunoglobulin A.

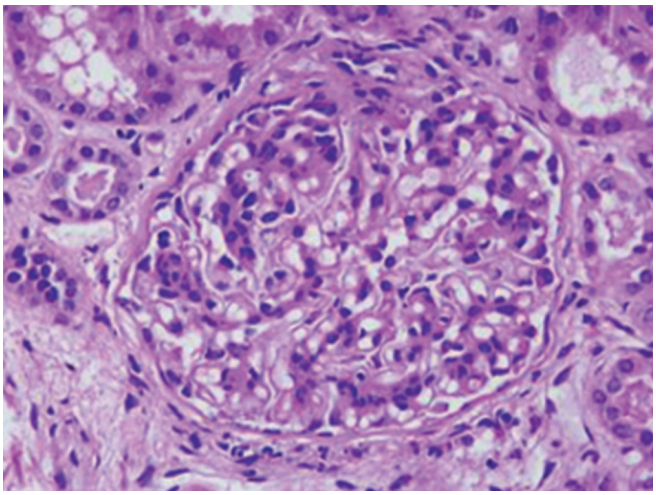

Figure 4: Membranoproliferative glomerulonephritis (periodic acid–Schiff stain, magnification ×400).

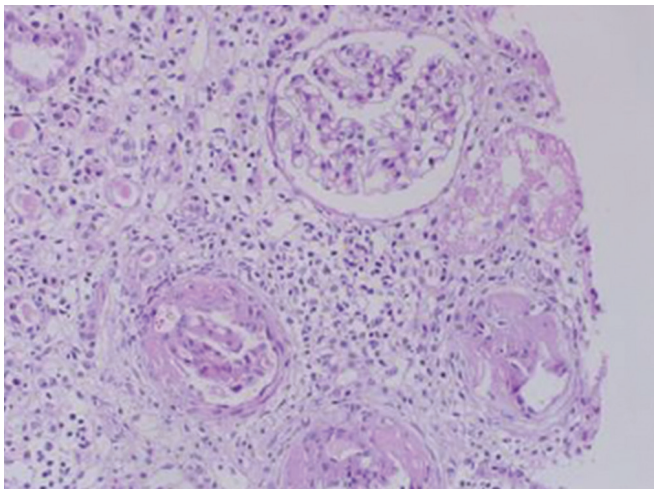

Figure 5: IgA nephropathy (hematoxylin and eosin stain, magnification ×100).

Table 4: Comparison of various parameters of patients with different stages of AKI[a]

Parameters	Severity of AKI			*P*
	Stage I (*n* = 90)	Stage II (*n* = 114)	Stage III (*n* = 39)	
Age (years)	50.06 ± 12.54	47.71 ± 13.55	48.15 ± 7.04	0.38
BMI (kg/m^2)	22.09 ± 2.05	22.22 ± 1.65	22.06 ± 1.75	0.83
MAC (cm)	21.52 ± 1.84	21.40 ± 1.96	21.80 ± 0.99	0.48
Hb (g/dl)	8.04 ± 2.35	8.39 ± 2.40	7.36 ± 2.06	0.05
Platelet count (×1000/mm^3)	88.40 ± 53.24	97.39 ± 47.59	91.07 ± 55.25	0.44
TLC (×1000/mm^3)	7.83 ± 3.03	11.26 ± 7.49	13.99 ± 8.36	<0.01
Serum creatinine (mg/dl)	1.82 ± 0.20	2.53 ± 0.43	4.54 ± 1.36	<0.01
Serum lactate (mmol/l)	1.65 ± 1.02	1.78 ± 0.89	1.67 ± 0.43	0.55
Serum sodium (mEq/l)	132.16 ± 5.11	131.60 ± 5.82	132 ± 7.88	0.79
Serum potassium (mEq/l)	4.43 ± 0.94	4.43 ± 0.84	4.50 ± 0.86	0.90
Serum albumin (g/dl)	2.65 ± 0.46	2.64 ± 0.38	2.60 ± 0.42	0.99
Serum AST (IU/l)[b]	69 (11–320)	91.5 (16–1150)	65 (30–179)	0.97
Serum ALT (IU/l)[b]	34.5 (10–410)	38 (12–220)	25 (12–66)	<0.01
Serum bilirubin (mg/dl)[b]	2.75 (0.4–21)	3.35 (0.5–35)	3.3 (0.9–44)	<0.01
Serum cholesterol (mg/dl)	107.04 ± 47.45	108.15 ± 44.51	106.46 ± 47.82	0.38
CTP score	11.02 ± 2.35	11.88 ± 2.37	12.58 ± 2.40	0.83
MELD-Na score	26.70 ± 4.53	31.63 ± 5.81	34.07 ± 5.04	0.48

[a]Data are mean ± standard deviation unless specified otherwise. [b]Data are median and range. AKI: acute kidney injury; ALT: alanine aminotransferase; AST: aspartate aminotransferase; BMI: body mass index; CTP: Child–Turcotte–Pugh; Hb: hemoglobin; MAC: mid arm circumference; MELD: Model for End-stage Liver Disease; TLC: total leukocyte count.

done in seven patients. The overall in-hospital mortality in our study population was 18.5% (*n* = 45).

Discussion

Renal dysfunction, especially HRS, has an unfavourable prognosis in patients with cirrhosis. Renal insufficiency is often associated with other accompanying cirrhosis complications like hepatic encephalopathy, variceal bleed, ascites, etc.[14] The first description of renal dysfunction in the setting of cirrhosis was from Europe and the United States in late 19th century.[15] Hecker and Sherlock[16] in 1956 gave the first detailed description of HRS. In the past, most of the clinical studies focused their attention on HRS, which is generally seen in advanced stages of cirrhosis.

In recent years, better understanding of pathophysiology of AKI has led to changes in diagnostic criteria, with specific treatment approaches responsible for improved outcome.[17]

Demographic parameters of patient population included in our study are in accordance with the previous studies from India. In studies conducted by Sethuraman and Balasubramanian[18] and Rathi *et al*.,[19] the mean age of the patients was 49.58 and 49.5 years, respectively. Mean age of the patients in our study was 47.85 years (±12.54). Majority of patients in our study were male, which is similar to previously conducted studies.[18,19] In the present study, the most common etiology of

cirrhosis was found to be ethanol followed by hepatitis B, which was replicated previously by a study from our center and other part of the country.[20,21] Similar results were replicated by a cohort study in Swedish population, with higher incidence of cirrhosis in men compared to women, and also, ethanol was the main culprit for cirrhosis (50.5%).[22]

The spectrum of renal dysfunction in our study was prerenal AKI in 54.4%, HRS in 21.8%, intrinsic renal disease in 17.3%, chronic kidney disease in 4.9%, and postrenal AKI in 1.6% of patients. Similar spectrum of renal dysfunction was seen in studies conducted by Arora *et al*.,[23] Keshav and Kumar,[24] and Soni and Nagpal.[25]

In patients with intrinsic renal disease with persistent renal dysfunction, the most common glomerular pathology was IgA nephropathy. In a study of liver transplant candidates with renal failure of undetermined etiology, the most common glomerular lesion on renal biopsy was found to be IgA nephropathy (45%).[26] In another study with 65 patients, the most common glomerular pathology was IgA nephropathy in 27 (41.5%) patients, followed by MPGN in 11 patients (16.9%).[27]

Majority of the patients in our study had stage II and stage I AKI as per the ICA-AKI criteria.[7] In a study from northern India, patients in stage I and stage II AKI constituted 77.4% and 19.7%, respectively.[17] Another study by Piano *et al*.[28] revealed that most of the patients had stage I AKI (52.5%) followed by stage III (31.2%).

Stages of AKI significantly correlated with the CTP and MELD-Na scores in our study; similar correlation was found with total leukocyte count. Identical results were replicated in a study conducted by Khatua *et al*.[29] and Shetty *et al*.[30] Overall in-hospital mortality in our study population was 18.5%; similarly, in the study by Khatua *et al*.,[29] mortality in hospitalized patients was 17.8%. But higher mortality rates were seen in the studies conducted by Shetty *et al*. (44.7%)[30] and de Carvalho *et al*. (52.7%).[10]

Our study results should be interpreted keeping in mind the limitations. This was a single-center study, and findings may not be generalizable to whole populations. However, no study has been published recently using the new 2015 Ascites Club Criteria in our population. Thus, we believe that results from the current study represent an important contribution on AKI patients with cirrhosis.

Conclusion

Renal dysfunction is a frequent cause of morbidity and mortality among cirrhotic patients. It was observed that PRA was the most common type of renal dysfunction. Patients with worst liver disease were found to have severe AKI with stage II and III. Authors suggest that screening of AKI should be done in all patients with decompensated cirrhosis and renal biopsy should be considered in patients not responding to treatment, for timely diagnosis and appropriate management.

Conflicts of interest

There are no conflicts of interest.

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