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# Original Research

## Assessment of clinical profile of patients with acute kidney injury and liver cirrhosis

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***ABSTRACT:***

**Background:** Acute kidney injury is a sudden and rapid deterioration in kidney function. It is a serious condition that occurs over a short period of time and can have a significant impact on overall health. The present study evaluated clinical profile of patients with acute kidney injury (AKI) and liver cirrhosis. **Materials & Methods:** 86 patients of cirrhosis of the liver and acute kidney injury of both genders were enrolled. Staging of AKI, and risk factors for cirrhosis was recorded. Patients were divided into three groups. Group I comprised of pre-renal azotemia, group II hepatorenal syndrome, and group III acute tubular necrosis. Child's and model for end stage liver disease (MELDs) scores, creatinine at admission, eGFR at admission and Child- Pugh score was recorded. Hospital stay were also recorded in all groups. **Results:** Group I had 16 males and 14 females, group II had 20 males and 15 females and group III had 11 males and 10 females. In group I, II and III, AKI stage 1 was seen in 7, 6 and 4, stage 2 in 8, 9 and 6 and stage 3 in 15, 20 and 11 respectively. Etiology for cirrhosis was alcohol in 12, 9 and 6, Hepatitis B in 10, 11 and 8, Hepatitis C in 9 , 8 and 4, cryptogenic in 4, 7 and 3 respectively. The difference was significant (P< 0.05). Creatinine at admission was 1.7 mg/dL, 3.4 mg/dL and 2.5 mg/dL. eGFR at admission was 39.4 mL/min/1.73 m2, 27.5 mL/min/1.73 m2 and 36.2 mL/min/1.73 m2. MELD score was 28.3, 21.5 and 32.8, Child- Pugh score was 10.5, 10.8 and 11.2. Hospital stay was 11.2 days, 10.1 days and 11.3 days in group I, II and III respectively. The difference was non- significant (P>0.05). **Conclusion:** Acute kidney injuries in liver cirrhosis are associated with high in-hospital mortality. MELD score and Child- Pugh score can be considered as indicator of AKI.

**Key words:** Acute kidney injuries, liver cirrhosis, MELD score

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## INTRODUCTION

Acute kidney injury is a sudden and rapid deterioration in kidney function. It is a serious condition that occurs over a short period of time and can have a significant impact on overall health.[1] AKI can range from mild to severe and may lead to a build-up of waste products and fluids in the body, which can be life-threatening if not treated promptly.[2] AKI is defined as abrupt (within 48 hours) increase in serum creatinine of 0.3mg/dl or increase in serum creatinine by 1.5 times or oliguria of < 0.5 ml/kg/h for > 6 hours.[3] Risk Injury Failure Loss and End stage renal disease (RIFLE) criteria are defined based on increase in serum creatinine / GFR and decrease in urine output. The AKIN criteria differ from the RIFLE criteria in several ways. The RIFLE criteria are defined as changes within 7 days while AKIN criteria suggest using 48 hours. AKIN criteria avoid using glomerular filtration rate as a marker in AKI.[4]

Acute kidney damage (AKI), a significant risk factor for early in-hospital mortality and of uttermost clinical and prognostic value, is seen often in hospitalized patients with liver cirrhosis. Patients with cirrhosis are more likely than noncirrhotic people to have AKI. One estimate for the prevalence of AKI in hospitalized cirrhotic patients ranges from 20% to 50%.[5] Pre-renal injury (PRI), acute tubular necrosis (ATN), and hepatorenal syndrome (HRS-AKI) are the three main causes of AKI in cirrhotic patients. Since treatments vary greatly, accurate distinction is essential. Plasma volume expansion often has a positive effect on pre-renal AKI, however HRSAKI

and ATN require alternative methods and are linked to increased in-hospital mortalit.[6]The present study evaluated clinical profile of patients with acute kidney injury (AKI) and liver cirrhosis.

## MATERIALS & METHODS

The present study consisted of 86 patients of cirrhosis of the liver and acute kidney injury of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. Parameters such as comorbid illnesses, staging of AKI, and risk factors for cirrhosis was recorded. Patients were divided into three groups. Group I comprised of pre-renal azotemia, group II hepatorenal syndrome, and group III acute tubular necrosis. Child's and model for end stage liver disease (MELDs) scores were recorded in all groups. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

## RESULTS

**Table I Distribution of patients**

| Groups | Group I | Group II | Group III |
|---|---|---|---|
| Status | PRA | ATN | HRS |
| M:F | 16:14 | 20:15 | 11:10 |

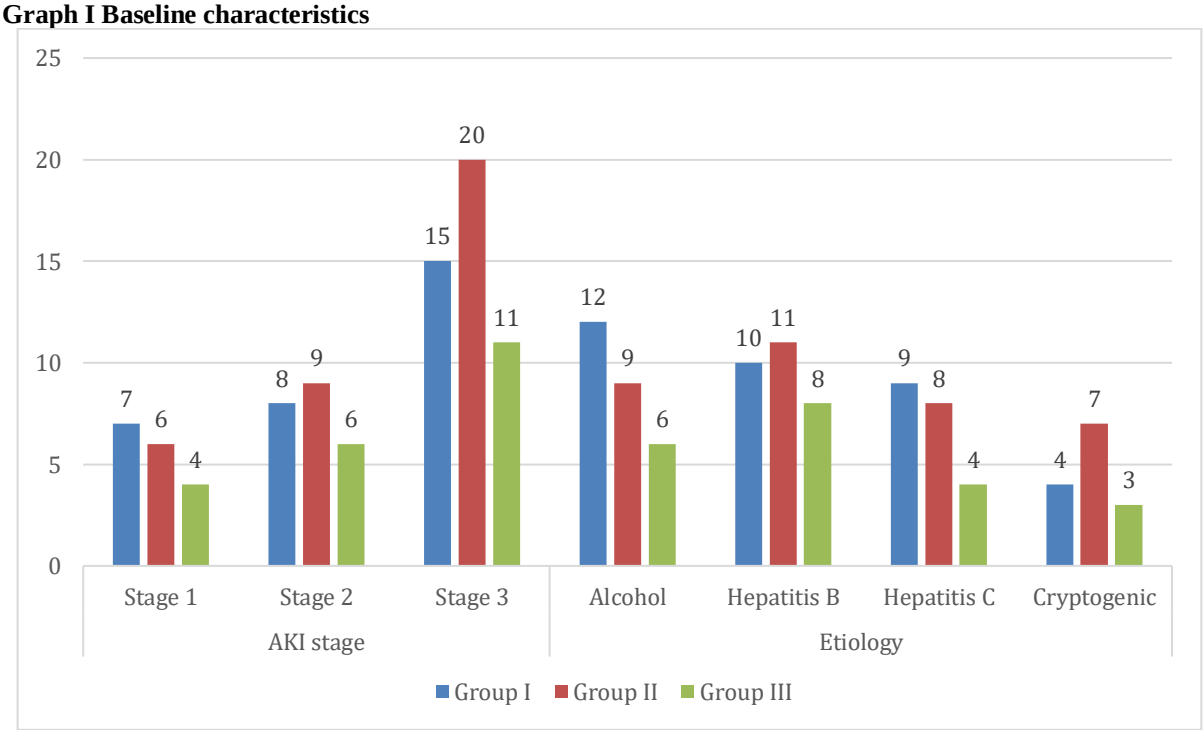
Table I shows that group I had 16 males and 14 females, group II had 20 males and 15 females and group III had 11 males and 10 females.

**Table II Baseline characteristics**

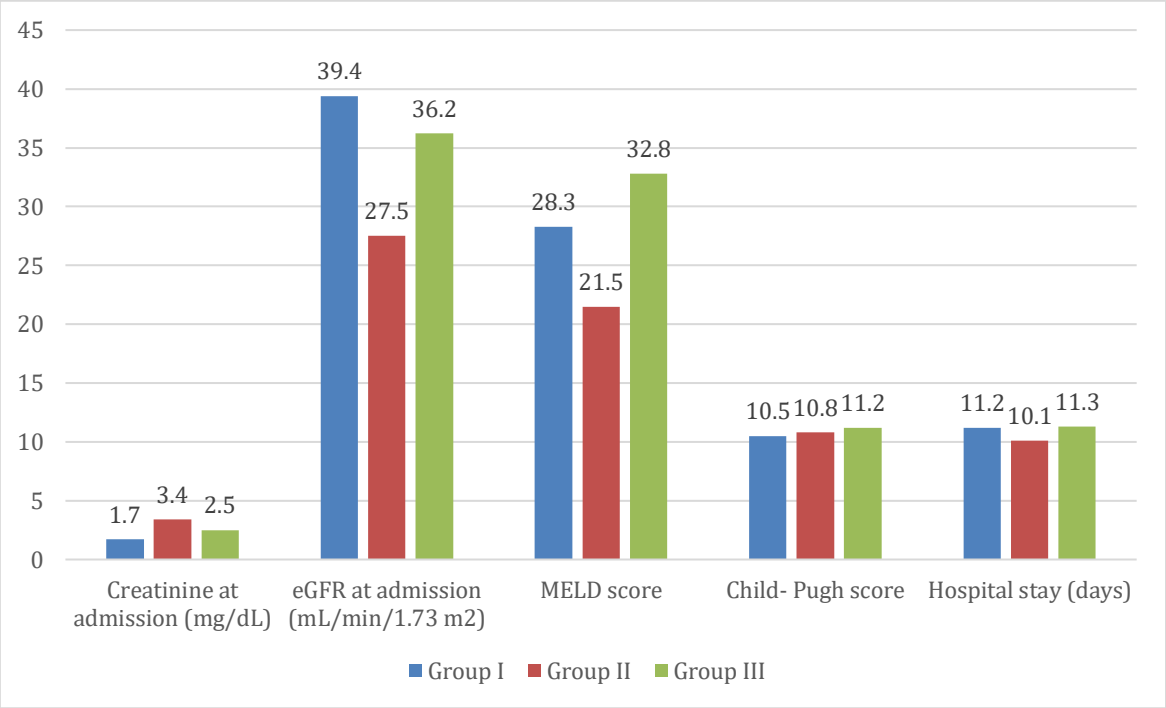
| Characteristics | Variables | Group I | Group II | Group III | P value |
|---|---|---|---|---|---|
| AKI stage | Stage 1 | 7 | 6 | 4 | 0.05 |
| | Stage 2 | 8 | 9 | 6 | |
| | Stage 3 | 15 | 20 | 11 | |
| Etiology | Alcohol | 12 | 9 | 6 | 0.82 |
| | Hepatitis B | 10 | 11 | 8 | |
| | Hepatitis C | 9 | 8 | 4 | |
| | Cryptogenic | 4 | 7 | 3 | |

Table II, graph I shows that in group I, II and III, AKI stage 1 was seen in 7, 6 and 4, stage 2 in 8, 9 and 6 and stage 3 in 15, 20 and 11 respectively. Etiology for cirrhosis was alcohol in 12, 9 and 6, Hepatitis B in 10, 11 and 8, Hepatitis C in 9, 8 and 4, cryptogenic in 4, 7 and 3 respectively. The difference was significant (P< 0.05).

**Graph I Baseline characteristics**



**Graph II Comparison of parameters**




Graph I shows that creatinine at admission was 1.7 mg/dL, 3.4 mg/dL and 2.5 mg/dL. eGFR at admission was 39.4 mL/min/1.73 m2, 27.5 mL/min/1.73 m2 and 36.2 mL/min/1.73 m2. MELD score was 28.3, 21.5 and 32.8, Child- Pugh score was 10.5, 10.8 and 11.2. Hospital stay was 11.2 days, 10.1 days and 11.3 days in group I, II and III respectively. The difference was non- significant (P>0.05).

## DISCUSSION

Traditional classifications of AKI causes include prerenal azotemia, intrinsic renal parenchymal disease, and post renal obstruction.[7] Although the underlying kidney function may be adequate in pre renal causes of AKI, decreased renal perfusion brought on by intravascular volume loss (caused, for example, by vomiting or diarrhea) or decreased arterial pressure (caused, for example, by heart failure or sepsis) leads to a lower glomerular filtration rate.[8] In an effort to keep the glomerular filtration rate constant, autoregulatory systems frequently can make up for some degree of diminished renal perfusion.[9] However, these systems are compromised in patients who already have chronic kidney disease, making them more prone to developing acute or chronic renal failure.[10] It adapted the AKI to represent acute renal dysfunction in cirrhosis and defined it by an increase in SCr of 0.3 mg/dL (26.4 μmol/L) in < 48 h, or a 50% increase in SCr from a baseline within ≤ 3 months. The severity of AKI is described by three stages similar to acute kidney injury network (AKIN) criteria for defining AKI in noncirrhotic patients.[11,12] The present study evaluated clinical profile of patients with acute kidney injury (AKI) and liver cirrhosis.

We found that group I had 16 males and 14 females, group II had 20 males and 15 females and group III had 11 males and 10 females. In group I, II and III, AKI stage 1 was seen in 7, 6 and 4, stage 2 in 8, 9 and 6 and stage 3 in 15, 20 and 11 respectively. We observed that etiology for cirrhosis was alcohol in 12, 9 and 6, Hepatitis B in 10, 11 and 8, Hepatitis C in 9, 8 and 4, cryptogenic in 4, 7 and 3 respectively. Arora et al[13] examined the clinical characteristics and risk factors for acute kidney damage (AKI) in 175 consecutive patients with decompensated cirrhosis. Prerenal AKI accounted for 67.6% of AKI prevalence, followed by postrenal AKI (1.4%), intrinsic renal AKI (7%) and hepato-renal syndrome (HRS) (23.8%). Mean arterial pressure (MAP), platelet count, and serum albumin were significantly lower and total leucocyte count (TLC), blood urea nitrogen, serum creatinine (SCr), total bilirubin, aspartate aminotransferase, international normalized ratio, Child-Turcotte-Pugh (CTP) score, and model for end-stage liver disease (MELD) score higher in cirrhosis patients with AKI than without AKI. In distinct forms of AKI, MAP, hemoglobin, TLC, and SCr were substantially different. AKI and CTP score were significantly correlated. Type of AKI had significant association with SBP, sepsis, and shock. Mortality occurred in 33.8% patients with AKI with 64.7% mortality in patients with HRS. Outcome had significant association with AKI, stage and type of AKI. Multivariate analysis showed SBP, sepsis, and shock as independent predictors of AKI.

We found that creatinine at admission was 1.7 mg/dL, 3.4 mg/dL and 2.5 mg/dL. eGFR at admission was 39.4 mL/min/1.73 m2, 27.5 mL/min/1.73 m2 and 36.2 mL/min/1.73 m2. MELD score was 28.3, 21.5 and 32.8, Child- Pugh score was 10.5, 10.8 and 11.2. Hospital stay was 11.2 days, 10.1 days and 11.3 days

in group I, II and III respectively. Patients with cirrhosis of the liver and AKI were investigated by Shetty et al[14] for their clinical characteristics and predictors of in-hospital mortality. Based on the updated 2015 Ascites Club Criteria, AKI staging was completed. Pre-renal azotemia (PRA), hepatorenal syndrome (HRS), and acute tubular necrosis (ATN) were the three forms of AKI that the patients were divided into. The majority of patients (57.3%) had stage 3 AKI. The two most common kinds of AKI were ATN (42.3%) and HRS (43.9), followed by PRA (13.8%). The overall in-hospital death rate was 44.7%. AKI stage 3 and those who required hemodialysis had the highest fatality rates, which rose with increasing AKI severity. Patients with ATN experienced a substantial increase in in-hospital mortality.

The shortcoming of the study is small sample size.

## CONCLUSION

Authors found that acute kidney injuries in liver cirrhosis are associated with high in-hospital mortality. MELD score and Child- Pugh score can be considered as indicator of AKI.

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