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# Clinical profile of patients with AKI and liver cirrhosis attending tertiary care hospital

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**Abstract**
Despite improved understanding of precipitants and physiology of underlying AKI in cirrhosis, considerable confusion still continues to surround its diagnosis due to arbitrary use of creatinine which is effected by several factors like diet, volume status, protein intake, muscle mass, interference with assays of creatinine by elevated bilirubin level making traditional criteria of AKI with serum creatinine greater than 1.5 gm/dl not suitable as diagnostic criteria as it can delay the detection of AKI, further delaying treatment & prognosis. All participating patients of either gender admitted in department of GASTROENTEROLOGY with age >18 years with either diagnosed or newly diagnosed case of cirrhosis of liver (Including both compensated & decompensated cases) admitted with acute kidney injury diagnosed according to International Club of Ascites Classification were enrolled in this study. 64.9% of patients with AKI in the study had MELD score between 16-30 followed by 23.4% & 11.7% in MELD > 30 & MELD <= 15 group respectively.

**Keywords:** AKI, Liver Cirrhosis, MELD

## Introduction
Over the last several years consensus guidelines have evolved to aid in diagnosis of AKI in cirrhosis like Acute Dialysis Quality Initiative group for the Risk, Injury, Failure, Loss, End stage renal disease (RIFLE (2004), Acute Kidney Injury Network criteria (AKIN, 2007), Kidney Disease Improving Global Outcome (KDIGO 2012) to recently ICA-AKI criteria in 2015. As compared to ICA-AKI criteria, earlier classification also used urine output as component for renal failure with creatinine value used were more static in nature, however in ICA-AKI creatinine use is more dynamic in nature due to comparison with baseline creatinine with no use of urine output in diagnosis as most of the cirrhotic patients are often oliguric & urine measurement are mostly inappropriate, hence making ICA-AKI criteria more appropriate [1, 2].

Despite improved understanding of precipitants and physiology of underlying AKI in cirrhosis, considerable confusion still continues to surround its diagnosis due to arbitrary use of creatinine which is effected by several factors like diet, volume status, protein intake, muscle mass, interference with assays of creatinine by elevated bilirubin level making traditional criteria of AKI with serum creatinine greater than 1.5 gm/dl not suitable as diagnostic criteria as it can delay the detection of AKI, further delaying treatment & prognosis. Henceforth ICA AKI criteria is being validated & accepted for definition of AKI. The benefits of adopting the new, more sensitive definition are two - fold. First, lowering the threshold for a diagnosis of AKI will increase sensitivity and the association between even mild acute increases in creatinine and adverse outcomes has been well established. Second, the lower threshold of AKIN will identify those more severe case of AKI significantly earlier, thus facilitating earlier interventions and potentially improving prognosis [3, 4].

Despite these theoretical benefits of this new criteria the logical outcome of the new definition will be an increase in patients fulfilling criteria of AKI due to increased sensitivity and decrease in specificity causing patients to undergo numerous blood test and administration of treatments which are frequently both scarce (albumin) and expensive (albumin and terlipressin), and not without risk (excessive volume in patients with volume in ATN, ischemic complications of vasoconstrictors) causing concern that in some patients the

risks of treatment may outweigh the rewards [5].
Apart from use of urinary biomarkers different model had been used to predict the mortality in these patients using MELD, CLIF SOFA, however there is novel prognostic tool like Acute kidney injury - Chronic Liver Failure - Sequential Organ Failure- Assessment score (AKI CLIF SOFA score) is a novel prognostic score used to predict mortality which comprises of age, creatinine, bilirubin, lactate & use of vasopressin as variables & had been shown by Area under receiver operating characteristic curve (AUROC) that it has more discriminatory power of predicting of mortality upto 1 year than earlier used prognosis criteria [6].
Addressing the unique circumstances and needs of developing countries with lack of adequate health facilities and late presentation of cases where most cases can be due to infection which is potentially reversible if detected in early stage of AKI preventing its progression to kidney failure requiring dialysis is of paramount importance, hence this thesis deals with physiology predisposing cirrhotic patients to AKI, assesses the prevalence and outcomes of different causes of AKI and prognosis, and explores a new framework for understanding AKI in cirrhosis incorporating novel biomarkers of kidney injury.

## Methodology

**Study setting:** Study was conducted at department of gastroenterology.

**Type of study:** Longitudinal Prospective type of observational study.

**Sample size:** Total 94 patients were enrolled during this period (Calculated by sample size: Z 1-α /z 2 P (1-P) d2 z is standard normal variate (1.96 at 1% type 1 error) pi expected proportion in population based on previous studies(10%) d is absolute error or precision (5% for type 1 error)

**Inclusion criteria:** All participating patients of either gender admitted in department of gastroenterology with age >18 years with either diagnosed or newly diagnosed case of cirrhosis of liver (Including both compensated & decompensated cases) admitted with acute kidney injury diagnosed according to International Club of Ascites Classification were enrolled in this study.

### Exclusion criteria

Parenchymal kidney disease
Receiving renal replacement therapy/renal or liver transplant Pregnant or nursing patient refusal to participate in study.
Patient were followed for the time during hospital stay to determine outcome in form of improvement in creatinine level, need of renal replacement therapy & condition on discharged by means of either survival or death.

## Results

**Table 1:** Gender distribution

| Sex | Frequency | Percent |
|---|---|---|
| Female | 23 | 24.5 |
| Male | 71 | 75.5 |
| Total | 94 | 100.0 |

This table shows that in study of total 94 patients there are 23 female patients (24.5%) & 71 male patients (75.5%)

**Table 2:** Distribution of different cause of cirrhosis

| Cause of cirrhosis | Frequency | Percent |
|---|---|---|
| Alcohol | 27 | 28.7 |
| Autoimmune | 2 | 2.1 |
| Budd chiari syndrome | 1 | 1.1 |
| Cryptogenic | 19 | 20.2 |
| Hbv | 3 | 3.2 |
| Hcv | 3 | 3.2 |
| Nash | 39 | 41.5 |
| Total | 94 | 100.0 |

NASH is the most common cause of cirrhosis comprising of total 39 patients (41.5%) followed by Alcohol which were 27 in number (28.7%).

**Table 3:** Distribution of meld score in AKI patients

| Meld | Frequency | Percent |
|---|---|---|
| <=15 | 11 | 11.7 |
| 16-30 | 61 | 64.9 |
| >30 | 22 | 23.4 |
| Total | 94 | 100.0 |

64.9% of patients with AKI in the study had MELD score between 16-30 followed by 23.4% & 11.7% in MELD > 30 & MELD <= 15 group respectively.

**Table 4:** Distribution of urinary ngal

| Urinary ngal (ng/ml) | Frequency | Percent |
|---|---|---|
| <121 | 17 | 39.5 |
| >121 | 26 | 60.5 |
| Total | 43 | 100.0 |

Of 43 patients out of 94 whose Urinary NGAL were tested 60.5% (26 in number) had Urinary NGAL > 121ng/ml whereas 39.5% (17 in number) had Urinary NGAL value < 121 ng/ml.

## Discussion

Although useful of regular dynamic creatinine estimation can be useful in detection of AKI, but it may lag behind causing delay in diagnosis, treatment resulting into unfavourable prognosis. Henceforth there has been increasing utility of urinary & serum biomarkers like Neutrophil gelatinase-associated lipocalin (NGAL), IL18, Cystatin C which are raised much earlier than rise in creatinine level causing earlier and appropriate diagnosis of cause of AKI resulting into earlier institution of appropriate treatment and therefore favoring prognosis. As these are mostly increased in cases of ATN which has the worst prognosis, they help in early institution of hemodialysis resulting into prevention of valuable time frame caused due to misdiagnosis. This can be complemented with use of serial biomarkers measurement which is also helpful in predicting prognosis of patients.
Mean CTP score is 10.41+ 1.75 with 68.1% of patients developing AKI are categorized under child C followed by 29.8% & 2.1% in child B & child A respectively. This is similar to study conducted by Thabut *et al.* [7] where 74% patients have Child-Pugh class C with Mean CTP score of 11+ 2. Considering meld score, Mean meld score is 24.84 +

7.97 with 64.9% of patients with AKI in the study had MELD score between 16-30 followed by 23.4% & 11.7% in MELD > 30 & MELD <= 15 group respectively This is similar to Allegretti *et al.* [8] where mean MELD score is 24.

Of total 94 patients 38.3% has Stage 1 AKI, 29.8% has Stage 2 AKI & 31.9% has Stage 3 AKI and this is similar to study by Scott *et al.* [11] where they have 40% in Stage 1, 29.1% in Stage 2, 30.9% in Stage 3. When comparing Stage of AKI with multiple baseline variables, Age, MAP, creatinine at AKI, AKI CLIF sofa & lactate levels all of these shows statistically significant correlation ($p$<0.05) showing poorer value with increasing in the stage of AKI, worst in Stage 3, with 24% of patients belonging to child C category. 78.72% in my study had AKI before admission & 21.28% develop AKI during hospital admission which in belcher el al85 study was 60% & 40% respectively.

29.87% shows progression in AKI and 70.21% shows regression in AKI & in Belcher study 85, 60% shows no progression while 40% had progression in disease. In my study also maximum number of patients undergoing HD was in Stage 3 (56.66%) which is similar to Belcher *et al.* [10] study were maximum patients are in Stage 3 AKI (33.33%). Mortality in my study is 25.53% with maximum in Stage 3 AKI of about 41.66% which in Belcher *et al.* [10] study mortality is 84% reaching a peak of stage 3 showing increase in mortality with Stage of AKI as similar to my study.

Of total 94 patients, 43 patients underwent evaluation in form of Urinary NGAL of whom 17 patients (39.5%) has Urinary NGAL level <121 ng/ml & 26 patients (60.5%) has urinary NGAL level > 121 ng/ml where urinary NGAL of 121 ng/ml is the cut off value of laboratory investigations & by AUROC value of 111 ng/ml is used as the cut off value for hospital mortality which is 84% sensitive & 54% specific for mortality whereas as compared to Treeprasertsuk *et al.* [11] the AUROC of uNGAL in predicting mortality was 0.75 with a best cut-off level of 72 ng/mL providing 70.6% sensitivity and 69.2% specificity & among higher group 57.69% expired & 23.53% expired patient had value <121ng/ml. As urinary NGAL helps in predicting mortality and prognosis, so it was also correlated with other variables like MAP, creatinine at discharge / death, Lactate, AKI CLIF SOFA score, which all of these shows statistically significant correlation and is significantly poorer in patients with uNGAL > 121ng/ml as compared to uNGAL <121ng/ml.

Mean urinary NGAL value are 75.91, 160.99, 1340.91 ng/ml in PRA, HRS & ATN group which was similar as shown in Hamdy *et al.* [12] study where ATN group has the maximum mean NGAL level of 21.70 ±7.31, 115.53 ± 68.19 & 240.83 ±116.94 in PRA, HRS & ATN group respectively whereas similar results were shown regarding comparison with Stage of AKI where mean values are 228.44ng/ml, 552.49 ng/ml & 875.28 ng/ml in PRA, HRS & ATN group. Comparing my study & Hamdy *et al.* [12] study regarding level of urinary NGAL between survived & expired patients it is 547.82 ng/ml & 727. 49 ng/ml in my study whereas it is 73.46 & 104.96 in comparison study. In my study 53.69% of expired patients have NGAL value > 121ng/ml & only 23.53% of expired patients have value <121ng/ml ie at ratio of 2.28 whereas in study by Treeprasertsuk *et al.* [11] 86 the ratio is 3.42. Similar study conducted by Treeprasertsuk *et al.* [11], in my study also correlation between CTP group, liver biochemistry & NGAL level was not statistically significant.

## Conclusion

Mean age of patients were 56.79 + 11.74 yrs with majority of patients in age group of > 60 yrs (45.74%) with male predominance of 75.5%. Mean CTP score was 10.41 + 1.75 & meld score of 24.84 + 7.97 with 68.1% of patients in child C group & 64.9% in meld 16-30 group.

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