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Original article

Spot Urine Sodium and Potassium Ratio as a Predictor of Acute Kidney Injury and Survival in Decompensated Cirrhosis

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Abstract

Background: Acute kidney injury (AKI) is associated with high mortality in decompensated cirrhosis, so early prediction is vital. Urinary sodium excretion may have an association with mortality in decompensated cirrhosis. The study aims to investigate the spot urinary Na+/K+ ratio & fractional excretion of sodium in patients admitted with decompensated cirrhosis and evaluate its relationship with AKI and short-term survival. ***Method:*** It was a prospective analysis of 200 patients with decompensated cirrhosis over 6 months. Urinary sodium excretion was analyzed by spot urine Na+/K+ ratio & FENa. Receiver operator curve (ROC) analysis was done to find cut-off values of Na+/K+ in predicting AKI at admission, during the hospital stay, and survival probability. P-value <0.05 was considered significant. ***Result:*** AKI of admission was observed in 36.5% of patients and associated with low urine Na+/K+ and low urine Na+. 24.5% of patients who had no AKI at admission progressed to AKI during the hospital stay and were associated with low spot urine Na+/K+ and low FENa+. Area under curve (AUC) for urinary Na+/K+ (cutoff, sensitivity/specificity) at admission was 68.6% (≤1.36, 75.5/59.1), during hospital stay was 73.4% (≤ 1.64,77.6/67.5) and of short-term mortality was 73.4% (≤ 1.34, 93.3% /58.8). ***Conclusion:*** Spot urine Na+/K+ ratio at admission is a simple, predictable tool to predict AKI during the hospital stay and 30 days mortality in decompensated cirrhosis.

Keywords: ***Fractional Excretion of Sodium (FENa), Child-Pugh Score (CTP), AKI and MELD-Na***

Introduction

Cirrhosis is a pathologic entity defined as diffuse hepatic fibrosis. Cirrhosis is one of the leading causes of mortality and morbidity, accounting for 2.4% of death worldwide in 2016 [1]. Most deaths in patients with cirrhosis occur due to hepatic decompensation leading to hepatic and extrahepatic organ failure [2]. The rate of decompensation is 5-7% per year [3]. The development of complications like variceal bleeding, ascites, and encephalopathy characterizes decompensated cirrhosis.

Renal dysfunction is another complication of cirrhosis and is associated with increased mortality, occurring in one of every 5 patients with cirrhosis (4). Acute kidney injury (AKI) is due to decreased renal perfusion and glomerular kidney function (GFR). The incidence of AKI ranges from 20%-50% in cirrhotic patients hospitalized for acute decompensation [4]. AKI in patients with chronic liver disease is three types as follows: (a) prerenal AKI which includes prerenal azotemia and hepato-renal syndrome (HRS AKI); (b) intrinsic or intrarenal AKI (mainly represented by acute tubular necrosis [ATN]); and (c) post-renal AKI. Prerenal azotemia is the most common cause of AKI (69%) [5]. Patients with AKI have high mortality than those without AKI, i.e., 52.7 % and 29.9%, respectively [6].

Patients with AKI in cirrhosis have high sodium retention, which may be secondary to hypovolemia in prerenal AKI. So low urinary Sodium excretion may be a useful indicator of renal dysfunction in cirrhosis. 24 hours urinary sodium measurement is ideal for sodium excretion assessment but is cumbersome. Na/K ratio in spot urine (Na/K_{ur}) is a useful tool for urinary Sodium excretion, which has a good correlation with 24-h urine sodium excretion and is used for managing patients with ascites due to cirrhosis.

Spot Na/K_{ur} ratio has a good correlation with renal dysfunction and survival in patients with cirrhosis admitted for evaluation for liver transplantation [9] and decompensation [10]. FENa correlates with survival among patients with cirrhosis and renal dysfunction [11]. FENa is a clinical tool that can differentiate structural AKI from prerenal AKI and HRS-AKI [12]. Therefore, this work aimed to investigate the spot Na/K_{ur} ratio and FENa in patients admitted for decompensated cirrhosis, evaluating its relationship with AKI and short-term survival in southern India.

Material and Methods

This study was conducted from October 2021 to March 2022 in the Department of Medical Gastroenterology, Madras Medical College, and Chennai. All the patients in this study had an age equal to 18

years. Informed consent was taken. Patients with prior renal disease, any hepatic or extrahepatic malignancy, or admitted for any elective procedure were excluded from the study.

The study protocol was under ethical principles. The study protocol was approved by the Institutional ethical committee EC Reg. No. ECR/270/Inst/TN/2013/RR-20

Patients were evaluated in detail within 48 hrs. of admission and were followed during their hospital stay for a minimum of 30 days. Patients discharged within 30 days were called by phone to find out short-term survival (30 days).

Acute decompensation was defined by the development of hepatic encephalopathy, Acute Variceal Bleeding, ascites, and coagulopathy. Hepatic encephalopathy was managed with lactulose and Rifaximin. All patients with Variceal bleeding are managed with intravenous octreotide, antibiotics and endotherapy i.e., either EVL or Glue. ACLF is defined according to APACL criteria [13]. MELD-Na and CTP scores are used to predict patients.

AKI in cirrhosis is defined per the ICA AKI/adapted KDIGO criteria as an acute increase in serum creatinine (sCr) of ≥0.3 mg/dL within 48 h or by 50% from a stable baseline sCr within 3 months (presumed to have developed within the past 7 days when no prior readings are available) [14]. AKI in cirrhosis is stratified into three stages of progressively increasing severity. Stage 1 AKI is defined by rather small changes in sCr, whereas Stages 2 and 3 AKI are defined by a two-fold and three-fold increase in sCr, respectively. Stage 1 further sub stratified into Stage 1a (sCr ≤1.5 mg/dL) and stage 1b (sCr ≥1.5 mg/dL). Baseline Creatinine was defined as the closest value obtained before the current hospitalization (preferably on an outpatient basis). The admission value was considered baseline in the absence of a previous exam.

Response to treatment is defined as no response if there is no decrease in sCr value, partial response if the regression of AKI stage with a reduction of sCr to ≥0.3 mg/dl above baseline value, Full response if the return of sCr to a value within 0.3 mg/dl of the baseline value.

Spot urine samples were obtained within 48 h of hospitalization and more than 24 h of diuretics withdrawal in case of previous use. Easily Analyzer was used to estimate urinary sodium (Na_{ur}) and potassium (K_{ur}) by the ISE method. C501 Cobas 6000 fully automated analyzer was used to estimate urinary Creatinine (Cr_{ur}) tests. The Na/K_{ur} ratio was calculated by simply dividing the result of the urinary Na and K. FENa by the serum and urinary parameters of sodium and Creatinine.

Data management and statistical analysis

Data were entered in MS Excel, and analysis was done using SPSS 21.0 version. Data were presented as mean and standard deviation or median and interquartile range (non-parametric) for continuous variables and as percentages for categorical variables. Unpaired t-test was done to compare two group means, and Mann Whitney U test was done to compare two group medians. Chi-square or Fisher exact test (when expected cell count is less than 5) was done to find out the association between categorical variables. ROC analysis was done to determine cut-off values of the Na/K_{ur} ratio in predicting acute kidney injury at admission, discharge, and survival probability. A P-value of less than 0.05 was considered significant.

Result

Patient characteristics

Two hundred patients were included in the study. The mean age was 49.3± 11 yrs. 82% were male. Alcohol was the most common cause of cirrhosis (64.5%). Mean CTP and MELD-Na scores were 10.5±2.1 and 21.9±6.5, respectively. At hospital admission and during the hospital stay, 73 (36.5%) and 49 (24.5%) developed AKI, respectively. Out of 73 patients who had AKI at admission, 20 (10%) patients had AKI 1a, 31(15.5%) patients had AKI 1b, 14(7%) patients had AKI 2, and 8 patients had AKI 3. At hospital admission, 62 patients were on diuretics use but had no impact on spot Na/K_{ur} (1.01 vs. 1.35, p=0.395). Thirty days mortality rate was 7.5%. (Table-1)

Table 1: Basic Data of Study population

Gender	
Male	164 (82%)
Female	36 (18%)
Age (Mean±SD) (range)	49.3±11.0 (22-80%)
Aetiology of Cirrhosis	
Cryptogenic	10 (5%)
Ethanol	129 (64.5%)
HBV	20 (10%)
HCV	8 (4%)
NAFLD	24 (12%)
Prior decompensation	102 (51%)
Complication	
Ascites	160 (80%)
Hepatic encephalopathy	135 (67.5%)
GI bleeding	110 (55%)
Infection	96 (48%)
Prior Propranolol	76 (38%)
Prior Diuretics	62 (31%)
Child Pugh Score	
Class A	7 (3.5%)
Class B	58 (29%)
Class C	135 (67.5%)
AKI at admission	73 (36.5%)
New AKI at Hospitalization	49 (24.5%)
Progression of AKI after hospitalization	
Worsened	83 (41.5%)
Improved	33 (16.5%)
ACLF	23 (11.5%)
Mortality	15 (7.5%)

Serum	
Creatinine at admission - Median (IQR)	0.9 (0.7-1.4%)
Creatinine at 48hr - Median (IQR)	1.1 (0.8-1.5%)
Creatinine at Hospitalization - Median (IQR)	1.3 (1-2.1%)
Na (Mean±SD)	132.6±5.3
K - Median (IQR)	4.0 (3.6-4.5%)
Albumin- Mean±SD	2.6±0.5
Urea - Median (IQR)	39.0 (22.5-60%)
Urine	
Na - Median (IQR)	33.8 (22.8-54.4%)
K - Median (IQR)	28.7 (21-36%)
Creatinine - Median (IQR)	126.0 (86.3-172.6%)
Na:K ratio - Median (IQR)	1.28 (0.87-2.0%)
FE Na - Median (IQR)	0.2 (0.1-0.4%)
CTP Score- Mean±SD	10.5±2.1
MELD score- Mean±SD	21.9±6.5

Parameters of patients who had AKI at admission:
Out of 200 patients admitted with decompensated cirrhosis, 73 (36.5%) had AKI at admission. AKI at admission significantly associated with high total leukocyte count (9700 vs. 7600; $p < 0.05$), high Urea (60 vs. 31; $p < 0.001$), high Creatinine (1.6 vs. 0.8; $p < 0.05$), high MELD-Na (25 vs. 20.2; $p< 0.05$), Low serum sodium (131.5 vs. 133.3; p=0.023), Low urine sodium (25.8 vs. 37.9; $p< 0.05$) and low urine Na/K (0.93 vs. 1.52; $p< 0.05$) in comparison to patients without AKI at admission. But not significantly associated with prior decompensation or with CTP score or FENa. (Table-2)

Table 2: Comparison of data among AKI at admission and No AKI at admission group

Investigations	**AKI at Admission (n=73)**	**No AKI at admission (n=127)**	**P value**
Age - Mean±SD	51.0±12.2	48.3±10.1	0.096
Prior Decompensation	40 (39.2%)	62 (60.8%)	0.416
Complications			
ASCITES	59 (36.9%)	101 (63.1%)	0.826
Hepatic encephalopathy	53 (39.3%)	82 (60.7%)	0.243
GI Bleeding	36 (32.7%)	74 (67.3%)	0.220
Infection	44 (45.8%)	52 (54.2%)	0.008
Prior Diuretics	26 (41.9%)	36 (58.1%)	0.285
Blood			
TLC - Median (IQR)	9700 (6800-13200)	7600.0 (5300-10200)	0.002
Serum			
Creatinine at admission - Median (IQR)	1.6 (1-2.5)	0.8 (0.6-1.1)	<0.001
Creatinine at 48hr - Median (IQR)	1.7 (1.4-2.7)	0.9 (0.8-1.1)	<0.001
Creatinine at Hospitalization - Median (IQR)	1.9 (1.2-3)	1.0 (0.8-1.7)	<0.001
Na- Mean±SD	131.5±6.3	133.3±4.6	0.023
K - Median (IQR)	4.1 (3.6-4.6)	4.0 (3.5-4.4)	0.274
Urea - Median (IQR)	60 (39-89)	31 (20-44)	<0.001
Urine			
Na - Median (IQR)	25.8 (20-42)	37.9 (25.8-60.9)	0.001
K - Median (IQR)	29.0 (22.8-36.3)	27.4 (20-34.4)	0.179
Creatinine - Median (IQR)	125.9 (81-198)	126.0 (94.7-161)	0.795
Na:K ratio - Median (IQR)	0.93 (0.75-1.36)	1.52 (0.93-2.1)	<0.001
Child-Pugh Class			0.117
Class A	0 (0.0%)	7 (100.0%)	
Class B	23 (39.7%)	35 (60.3%)	
Class C	50 (37.0%)	85 (63.0%)	
ACLF	11 (47.8%)	12 (52.2%)	0.230
Extra			
FE Na - Median (IQR)	0.3 (0.1-0.6)	0.2 (0.1-0.3)	0.013
CTP Score- Mean±SD	10.7±1.9	10.4±2.2	0.352
MELD score- Mean±SD	25±6.9	20.2±5.6	<0.001

**Fisher exact test p-value*

Parameters of patients who developed AKI during hospital stay:
Out of 127 patients with no AKI at admission, 49 (24.5%) progressed to AKI during the hospital stay. Progression to AKI during hospital stay is significantly associated with high serum Creatinine at 48 hours of admission (1 vs. 0.9, p= 0.023), Low platelet count (80,000 vs. 91,000; p=0.029), Low total protein (5.7 vs. 6.0; p=0.03), Low FENa (0.1 vs. 0.3; $p<0.05$) and Low Na/K (1.09 vs. 1.92; $p<0.05$). Patients who progressed to AKI during hospital stay had high CTP scores (10.5 vs. 10.3) and high MELD Na (20.7 vs. 79.9), but the difference was not statistically significant. (Table-3)

Table 3: Comparison of data among patients who developed AKI after hospitalisation and who had no AKI after hospitalisation

Investigations	AKI after Hospitalization (n=49)	No AKI after Hospitalization (n=78)	P value
Age - Mean±SD	49.1±10	47.9±10.2	0.515
Prior Decompensation	28 (45.2%)	34 (54.8%)	0.137
Complications			
ASCITES	40 (39.6%)	61 (60.4%)	0.641
Hepatic encephalopathy	35 (42.7%)	47 (57.3%)	0.200
GI Bleeding	30 (40.5%)	44 (59.5%)	0.592
Infection	21 (40.4%)	31 (59.6%)	0.728
Prior Diuretics	15 (41.7%)	21 (58.3%)	0.653
Blood			
TLC - Median (IQR)	7709.0 (5600-9400)	7500.0 (5300-10800)	0.676
Serum			
Creatinine at admission - Median (IQR)	0.8 (0.7-1.1)	0.7 (0.6-1.0)	0.248
Creatinine at 48hr - Median (IQR)	1.0 (0.8-1.2)	0.9 (0.7-1.1)	0.023
Creatinine at Hospitalization - Median (IQR)	1.9 (1.6-2.3)	1.0 (0.7-1.0)	<0.001
Na- Mean±SD	133.4±5.6	133.2±3.8	0.846
K - Median (IQR)	4 (3.5-4.3)	4 (3.6-4.4)	0.970
Urea - Median (IQR)	33 (21-50)	30 (19-42)	0.122
Urine			
Na - Median (IQR)	34.0 (25.8-57.8)	40.5 (25.0-65.9)	0.405
K - Median (IQR)	33.6 (23-42.5)	23.4 (16.7-31)	<0.001
Creatinine - Median (IQR)	133.0 (98-176.8)	123.5 (87.2-154)	0.319
Na:K ratio - Median (IQR)	1.09 (0.78-1.6)	1.92 (0.99-2.42)	<0.001
Child-Pugh Class			0.667
Class A	2 (28.6%)	5 (71.4%)	
Class B	12 (34.3%)	23 (65.7%)	
Class C	35 (41.2%)	50 (58.8%)	
ACLF	8 (66.7%)	4 (33.3%)	0.058*
Extra			
FE Na - Median (IQR)	0.1 (0.1-0.2)	0.3 (0.1-0.5)	<0.001
CTP Score- Mean±SD	10.5±2	10.3±2.3	0.598
MELD score- Mean±SD	20.7±6.2	19.9±5.2	0.430

**Fisher exact test p-value*

Comparison of parameters among Dead (30 days) and Alive patients

Total 15 patients died at 30 days from 200 patients. Expired patients had high MELD-Na than alive patients (26.9±7.2 vs 21.5±6.3; p<0.05). Similarly Expired patients had low urine Na/K ratio compared to alive patients (0.82 vs 1.36; p<0.05). (Table-4)

Table 4: Comparison of data among alive and dead patients

Investigations	Alive (n=185)	Dead (n=15)	P value
Age - Mean±SD	49.5±10.9	46.4±11.6	0.288
Prior Decompensation	10 (9.8%)	92 (90.2%)	0.207
Complications			
ASCITES	14 (8.8%)	146 (91.3%)	0.313*
Hepatic encephalopathy	14 (10.4%)	121 (89.6%)	0.041*
GI Bleeding	10 (9.1%)	100 (90.9%)	0.345
Infection	10 (10.4%)	86 (89.6%)	0.132
Prior Diuretics	7 (11.3%)	55 (88.7%)	0.244*
Serum			
Creatinine at admission - Median (IQR)	0.9 (0.7-1.4)	1.6 (0.7-2.9)	0.101
Creatinine at 48hr - Median (IQR)	1.1 (0.8-1.4)	1.9 (1.4-3.4)	0.001
Creatinine at Hospitalization - Median (IQR)	1.1 (1-1.9)	3.0 (2.3-4)	<0.001
Na- Mean±SD	133±5.2	127.7±5.1	<0.001
K - Median (IQR)	4 (3.6-4.5)	3.8 (3.6-4.4)	0.780
Urea - Median (IQR)	39 (22-60)	42 (32-111)	0.168
Urine			
Na - Median (IQR)	33.9 (22.88-56.73)	27.3 (16.95-34.84)	0.054
K - Median (IQR)	27.9 (20.9-35)	32.0 (26-41)	0.060
Creatinine - Median (IQR)	126.0 (86.5-173)	117.0 (86-161)	0.607
Na:K ratio - Median (IQR)	1.36 (0.9-2.03)	0.82 (0.6-1.13)	0.003
Extra			
FE Na - Median (IQR)	0.2 (0.1-0.4)	0.2 (0.1-0.5)	0.777

CTP Score- Mean±SD	10.4±2.1	11.4±2	0.082
MELD score- Mean±SD	21.5±6.3	26.9±7.2	0.002
Child-Pugh Class			0.244
Class A	0 (0.0%)	7 (100.0%)	
Class B	2 (3.4%)	56 (96.6%)	
Class C	13 (9.6%)	122 (90.4%)	
ACLF	7 (30.4%)	16 (69.6%)	<0.001*
AKI at admission	9 (12.3%)	64 (87.7%)	0.049
AKI at hospitalization	6 (12.2%)	43 (87.8%)	0.003*

**Fisher exact test p-value*

The area under the receiver operating characteristic curve (AUC) of urine Na/K ratio to predict AKI at admission was 0.686(p<0.05). Urine spot Na/K ratio ≤1.36 had sensitivity 75.3%, specificity 59.15%, positive predictive value (PPV) 51.4% and negative predictive value (NPV) 80.7% for predicting AKI at admission. (Table-5) (Figure-1)

Table 5: ROC analysis for Urine Na: K ratio

Variable	Na: K ratio Cut off	AUC	Sensitivity	Specificity	PPV	NPV	Accuracy	P-value
AKI at admission	</=1.36	0.686	75.3	59.1	51.4	80.7	65.0	<0.001
AKI at hospitalization	</=1.64	0.734	77.6	61.5	55.9	81.4	67.7	<0.001
Mortality	</=1.34	0.734	93.3	50.8	13.3	98.9	54.0	<0.001

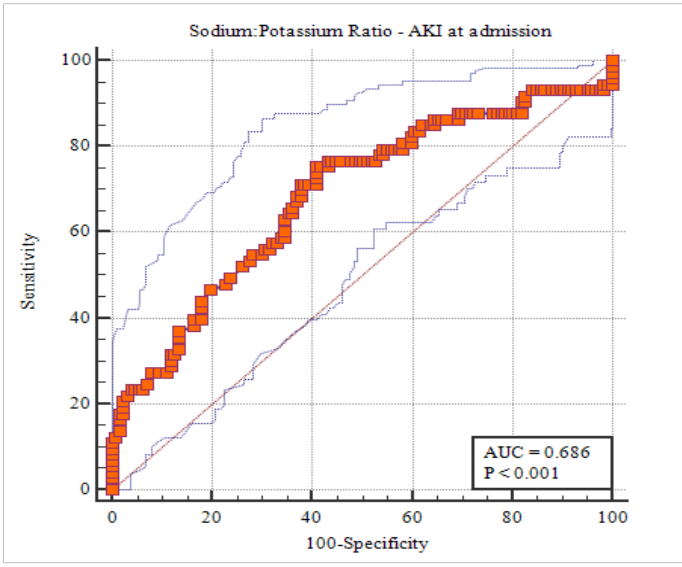

Figure 1: AUC of AKI at Admission

Similarly, the AUC of urine Na/K ratio to predict AKI progression during hospital stay was 0.734(p<0.001). Urine spot Na/K ratio ≤1.64 had a sensitivity of 77.6%, specificity of 61.5%, PPV 61.5%, and NPV 81.4%, with an accuracy of 67.7% for predicting AKI during a hospital stay. (Figure-2)

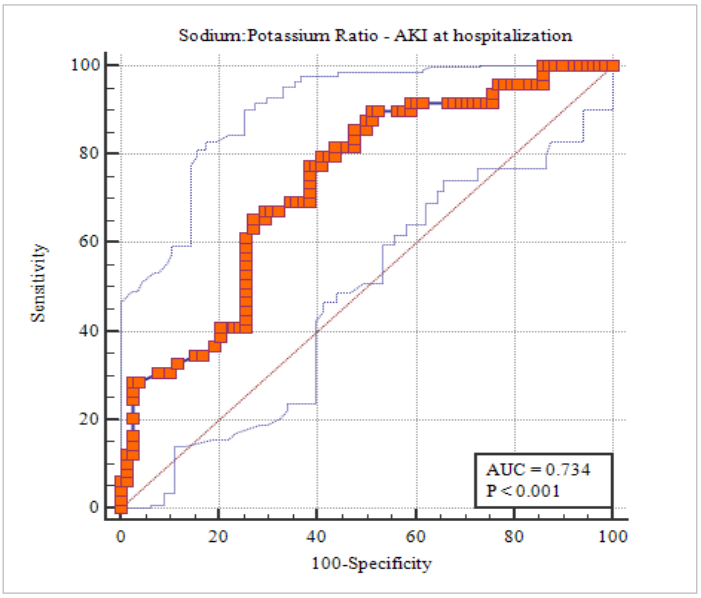

Figure 2: AUC of AKI at hospitalization

The AUC of urine Na/K ratio to predict survival at 30 days was 0.734(p<0.001). Urine spot Na/K ratio ≤1.34 had sensitivity 93.3%, specificity 50.8%, PPV 13.3% and NPV 98.9 % with accuracy of 54% for predicting short term mortality at 30days. (Figure-3)

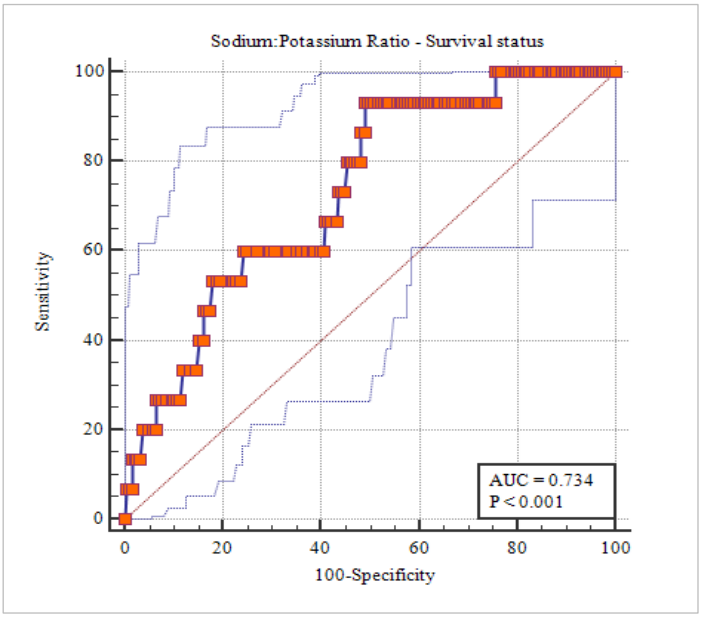

Figure 3: AUC of Mortality

Discussion

Prediction of risk of renal dysfunction in cirrhosis patients helps clinicians with a better management plan. Our study found that reduced sodium excretion, either as spot urine Na/K ratio or FENa, was related to more severe liver disease, i.e., high MELD-Na score or high CTP score. Similarly, Iqbal J et al. found that a high MELD score was associated with low urine Na/K ratio [15]. da Silva OM et al. found that poor sodium excretion group presented higher MELD score [16]. Morais EC et al. study observed that low urine Na/K and FENa were associated with high CLIF-SOFA, MELD score, and CTP score. In our study, AKI at admission is observed in 36.5% of patients, and those patients had low urine Na/K ratio but not FENa. But Morais EC et al. group. Patients with AKI at admission had both low urine Na/K and low FENa [10].

In our study, AKI at admission was observed in 36.5% of patients, and those patients had low urine Na/K ratio but not FENa. But Morais EC et al. found out that patients with AKI at admission had both low urine Na/K and low FENa [10].

Our study showed that patients who progressed to AKI had lower urine Na+/K+ ratio and FENa than those who didn't progress to AKI, which was statistically significant. Urine Na/K ratio ≤ 1.64 has high sensitivity and specificity to predict AKI with PPV of 61.5% and accuracy of 67.7%. Shankel T et al. found that urine Na+/K+ ratio having 100% sensitivity and specificity diagnostic test in AKI [17]. Morais Ec et al. study also showed similar results to our result, i.e., spot urine Na/K< 1 was associated with a high risk of AKI with a sensitivity of 71.8% and specificity of 66.7%, and high urine Na/K ratio (>2) was associated with low risk of AKI with sensitivity 89.7% and specificity 44.9% [10]. Cholongitas E et al. also stated that urine Na/K < 1 had high sensitivity and negative predictive value for the presence of GFR < 60 ml/min (79% and 87%, respectively) [9].

During the hospital stay, progression of decompensated cirrhosis to AKI was significantly associated with low spot urine Na/K ratio and low FENa, similar to Morais EC et al. study [10].

Prior history of decompensation was not significantly associated with the risk of progression of AKI or short-term mortality (30 days). Still, Morais EC et al. study showed that prior decompensation was associated with significant progression of AKI [10].

Spot urine Na/K ratio ≤ 1.34 predicts 30 days mortality with an accuracy of 54%. Morais EC et al. study showed that survival probability was 78.8% with spot Na/K_{ur} <1 [10]. Cholongitas E et al. found that spot Na/K_{ur} <1 was associated with a worse long-term prognosis in patients with decompensated cirrhosis [9].

The limitation of the study was the small sample size, so this study's result may not apply to a larger population. So, need of further studies. The second limitation was the lack of detailed information regarding dietary sodium intake, as urinary sodium excretion also depends upon dietary sodium intake. The third limitation was the lack of detailed information regarding diuretic dose and a group of diuretics. However, this study showed that diuretics have no impact on urinary spot Na/K excretion, but details of diuretics are needed. The fourth limitation was the lack of data regarding the different phenotypes of AKI, so the correlation of different phenotypes of AKI with urinary spot Na/K or FENa was not done.

Conclusion

So, in decompensated cirrhosis patients, the current study indicates that a low spot urine Na/K ratio can predict progression to AKI during hospitalization and short-term mortality. Spot urine Na/K ratio can be a simple objective tool compared to 24hr urine Na excretion to predict the progression of AKI and mortality. But this study needs validation in larger studies.

Statement of Ethics: This study was conducted after approval by the institutional ethical committee, Madras Medical College and performed in accordance with the Helsinki declaration. Written informed consent was obtained from all subjects.

Financial Support

Nil

Conflict of Interest

Nil

Lists of Abbreviations

ACLF: Acute on Chronic Liver Failure
AKI: Acute kidney injury

APACL: Asia-Pacific Association for the Study of Liver
AUC: Area Under the ROC Curve
CLIF-SOFA: Chronic liver failure sequential organ failure assessment
CTP: Child Turcotte Pugh
FE Na: Fractional Excretion of sodium
HBV: Hepatitis B Virus
HCV: Hepatitis C Virus
INR: International Normalized Ratio
ISE: Ion-selective electrode
IQR: Interquartile range
MELD: Model for End Stage Liver Disease
NAFLD: Non-alcoholic fatty liver disease
NPV: Negative Predictive Value
PPV: Positive predictive value
ROC: Receiver operating Characteristic
SD: Standard deviation

References

[1] Sepanlou SG, Safiri S, Bisignano C, Ikuta KS, Merat S, Saberifiroozi M, et al. (2020) The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol. Hepatol. 1;5(3):245-66.

[2] Sersté T, Cornillie A, Njimi H, Pavesi M, Arroyo V, Putignano A, et al. (2018) The prognostic value of acute-on-chronic liver failure during severe alcoholic hepatitis. J Hepatol.69(2):318-24.

[3] D'Amico G, Garcia-Tsao G, Pagliaro L. (2006) Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. J Hepatol. Jan;44(1):217-31.

[4] Garcia-Tsao G, Parikh CR, Viola A. (2008) Acute kidney injury in cirrhosis. Hepatology. 48(6):2064-77.

[5] Warner NS, Cuthbert JA, Bhore R, Rockey DC. (2011) Acute kidney injury and chronic kidney disease in hospitalized patients with cirrhosis. J Investig Med.59(8):1244-51.

[6] de Carvalho JR, Villela-Nogueira CA, Luiz RR, Guzzo PL, da Silva Rosa JM, Rocha E, et al. (2012) Acute kidney injury network criteria as a predictor of hospital mortality in cirrhotic patients with ascites. J Clin Gastroenterol.46(3):e21-26.

[7] Lee JW, Hwang JS, Chung WJ, Lee HJ, Park JG, Lee CH, et al. (2021) Diagnostic usefulness of the spot urine sodium/potassium ratio in cirrhotic patients with ascites. PLOS ONE.16(6):e0253886.

[8] da Silva OM, Bicca Thiele G, Fayad L, Lazzarotto C, Buzaglo Dantas-Corrêa E, de Lucca Schiavon L, et al. (2014) Comparative study of spot urine Na/K ratio and 24-hour urine sodium in natriuresis evaluation of cirrhotic patients with ascites. GE Port J Gastroenterol. 21(1):15-20.

[9] Cholongitas E, Goulis J, Arsos G, Birtsou C, Nakouti T, Papadopoulou S, et al. (2013) Association between sodium to potassium ratio in random urine samples and renal dysfunction and mortality in patients with decompensated cirrhosis. Clin Gastroenterol Hepatol. 11(7):862-7.

[10] Morais Rateke EC, Matiollo C, de Andrade Moura EQ, Andrigueti M, Maccali C, Fonseca JS, et al. (2021) Low sodium to potassium ratio in the spot urine sample is associated with progression to acute kidney injury and mortality in hospitalized patients with cirrhosis. Dig Liver Dis.53(9):1159-66.

[11] Ohashi N, Tsuji N, Naito Y, Iwakura T, Isobe S, Ono M, et al. (2013) Relationship between urinary fractional excretion of sodium and life prognosis in liver cirrhosis patients. Hepatol Res. 43(11):1156-62.

[12] Gowda YH, Jagtap N, Karyampudi A, Rao PN, Gujjarlapudi D, Sharma M, et al. (2022) Fractional Excretion of Sodium and Urea in Differentiating Acute Kidney Injury Phenotypes in Decompensated Cirrhosis. J Clin Exp Hepatol. 12(3):899-907.

[13] Hernaez R, Solà E, Moreau R, Ginès P. (2017) Acute-on-chronic liver failure. Gut. 66(3):541-53.

[14] Angeli P, Gines P, Wong F, Bernardi M, Boyer TD, Gerbes A, et al. (2015) Diagnosis and management of acute kidney injury in patients with cirrhosis: revised consensus recommendations of the International Club of Ascites. Gut. 64(4):531-7.

[15] Iqbal J, Khalid MA, Hanif FM, Mandhwani R, Laeeq SM, Majid Z, et al. (2018) Correlation Between MELD and UNa/K Ratio in Predicting Renal Dysfunction in Cirrhotic Patients. J Transl Int Med. 6(4):181-4.

[16] da Silva OM, Bicca Thiele G, Fayad L, Lazzarotto C, Buzaglo Dantas-Corrêa E, de Lucca Schiavon L, et al. (2014) Comparative study of spot urine Na/K ratio and 24-hour urine sodium in natriuresis evaluation of cirrhotic patients with ascites. J Port Gastrenterologia. 21:15-20.

[17] Shankel T, Shankel S. (2019) Urinary sodium/potassium ratio in acute kidney injury accurately differentiates prerenal azotemia from acute tubular necrosis. Acta Medica Marisiensis. 65:103-10.