Original Article

Urinary electrolyte parameters in sepsis-associated acute kidney injury: A prospective observational study

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ABSTRACT

Background and Aims: Sepsis-associated acute kidney injury (SA-AKI) significantly contributes to morbidity and mortality. Current biomarkers have limitations, necessitating the exploration of alternative indicators. This study aims to evaluate various urinary electrolyte parameters to predict SA-AKI. **Methods:** A prospective observational study included 111 sepsis patients within 24 h of admission. Urinary electrolyte samples were collected, and indices were calculated. Patients were monitored for 7 days to assess for acute kidney injury (AKI) according to Kidney Disease Improving Global Outcomes (KDIGO) definition criteria, mortality rates, and the need for renal replacement therapy. Mann-Whitney U test and Chi-squared test were used to analyse continuous and categorical variables, respectively. Receiver-operating characteristic (ROC) curves were constructed to determine to discriminatory ability of various parameters in predicting AKI. **Results:** Of 111 patients, 42.3% developed AKI, with a mortality rate of 59.5%. When evaluating urinary parameters, the product of urine sodium and urine creatinine exhibited the maximum full form [area under the receiver operating characteristic (AUROC): 0.66; 95%CI: 0.56, 0.77)], and the parameter of fractional excretion of potassium (FeK) exhibited an AUROC of 0.62 (95%CI: 0.51, 0.72). Furthermore, 2-hour excretion of potassium revealed a statistically significant correlation with 2-hour creatinine clearance ($r = 0.62$, $P < 0.001$). Logistic regression models, incorporating Sequential Organ Failure Assessment (SOFA) score, FeK, and urine sodium concentration as variables ($P = 0.020$, 0.044, and 0.033, respectively), achieved an AUROC of 0.751 in predicting AKI. **Conclusion:** Urine sodium levels and fractional potassium excretion moderately effectively predict AKI in sepsis patients. Urine potassium excretion correlates with glomerular filtration rate.

Keywords: Acute kidney injury, biomarker, creatinine clearance, electrolytes, glomerular filtration rate, potassium, ROC curves, sepsis, sodium

INTRODUCTION

Sepsis is defined as a life-threatening condition characterised by organ dysfunction triggered by an unregulated host response to infection.[1] Sepsis accounts for a significant proportion, ranging from 20% to 70%, of all cases of acute kidney injury (AKI) in critically ill patients.[2] Although there is currently no distinct definition for sepsis-associated AKI (SA-AKI), it is generally referred to as AKI occurring in patients with sepsis as per the Kidney Disease Improving Global Outcomes (KDIGO) definition.[3,4]

The wide-ranging definitions of sepsis and AKI have led to variable data, with SA-AKI mortality rates reported between 11% and 77%.[5,6] Given the high

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mortality risk, early and accessible identification of SA-AKI is essential to enable timely interventions. Although various biomarkers exist, their limited use due to cost, accessibility, and turnaround issues underscores the need for a simple, widely applicable early AKI detection test.[7]

The urine electrolyte index, such as fractional excretion of sodium (FeNa), differentiates pre-renal from renal AKI but lacks predictive value in SA-AKI.[8,9] Urinary potassium levels have yet to be extensively studied in patients with AKI. However, the excretion of potassium is a physiological response to kidney dysfunction. A higher fractional excretion of potassium (FeK) has been linked to persistent AKI, and elevated FeK levels often precede an increase in serum creatinine levels, making it a valuable early indicator of AKI.[9,10] Interestingly, it has been observed that higher urine output is associated with lower urinary potassium excretion, whereas lower urine output is associated with higher urinary potassium excretion.[10] In addition, 2-hour urine potassium excretion correlated with creatinine clearance with a moderate predictive ability for AKI in critically ill patients.[11]

This study was conducted to evaluate urinary electrolyte indices and their capacity to predict the onset of AKI. Our primary objective was to determine if 2-hour urinary potassium levels could predict AKI development in sepsis patients. Secondary objectives included: 1) assessing the predictive accuracy of FeK, FeNa, 2-hour sodium excretion, and the urine potassium-to-creatinine ratio for SA-AKI, and 2) evaluating the correlation between 2-hour creatinine clearance and the 2-hour excretion of sodium and potassium. We hypothesised that indices incorporating potassium levels would better predict AKI. Specifically, we postulated that lower 2-hour potassium excretion and a higher FeK would be superior in predicting the onset of AKI.

METHODS

This prospective observational study was conducted in the All India Institute of Medical Sciences, New Delhi intensive care units, from January 2022 to August 2023. Institute Ethics Committee approval was obtained for the research (vide approval number IECPG-726/25/25.11.2021 dated 16 December 2021), and the study was registered with the Clinical Trials Registry-India (vide registration number CTRI/2022/03/041334, www.ctri.nic.in) before recruitment of the study subjects. The study procedures follow the World Medical Association (WMA) guidelines and the Declaration of Helsinki, 2013. The study was conducted in accordance with the Good Clinical Practice guidelines. Written informed consent was obtained for participation in the study and use of the patient data for research and educational purposes.

Patients eligible for recruitment included those with clinically suspected sepsis or a Sequential Organ Failure Assessment (SOFA) score increase of 2 or more secondary to new-onset infection.[1] They were enroled within 24 h of admission or the onset of new sepsis. Excluded from the study were patients with pre-existing AKI (detected based on serum creatinine values measured at admission) or chronic kidney disease, those exposed to diuretics, pregnant women, individuals who had trauma to kidney or bladder surgery, and patients without indwelling catheters. Upon enrolment, urine samples were collected in the early morning, specifically between 6 am and 8 am, to measure urine concentrations of creatinine, sodium, and potassium. The 2-hour urinary potassium and sodium levels were calculated by multiplying the urine potassium and sodium concentrations by the volume of the collected samples, respectively. Simultaneously, blood samples were obtained and sent to the biochemistry to measure serum sodium, potassium, and creatinine levels. Based on these laboratory values, the study used various derived indices and creatinine clearance.

1. Creatinine clearance

$$= \frac{\text{urine creatinine}\left(\frac{\text{mg}}{\text{dL}}\right) \times \text{urine volume(ml)}}{\text{serum creatinine}\left(\frac{\text{mg}}{\text{dL}}\right) \times 120}$$

2. Fractional excretion of potassium(FeK)

$$= \frac{\text{urine potassium}\left(\frac{\text{mmol}}{\text{L}}\right) \times \text{serum creatinine}\left(\frac{\text{mg}}{\text{dL}}\right)}{\text{serum K}\left(\frac{\text{mmol}}{\text{L}}\right)} \times \text{urine creatinine}\left(\frac{\text{mg}}{\text{dl}}\right)$$

3. Fractional excretion of sodium (FeNa)=

$$\frac{\text{urine sodium}\left(\frac{\text{mmol}}{\text{L}}\right)\times\text{serum creatinine}\left(\frac{\text{mg}}{\text{dL}}\right)}{\text{serum sodium}\left(\frac{\text{mmol}}{\text{L}}\right)\times\text{urine creatinine}\left(\frac{\text{mg}}{\text{dL}}\right)}$$

4. Urine potassium to urine creatinine ratio (UK/UCr) $= \frac{\text{Urine potassium}\left(\frac{\text{mmol}}{\text{L}}\right)}{\text{Urine creatinine}\left(\frac{\text{mg}}{\text{dL}}\right)}$

5. Urine sodium and urine creatinine product (UNa*UCr) =

$$\text{Urine sodium}\left(\frac{\text{mmol}}{\text{L}}\right)\times\text{ Urine creatinine}\left(\frac{\text{mg}}{\text{dL}}\right)$$

All patients were closely monitored for 7 days following their enrolment in the study, during which a comprehensive set of data points was collected. This data encompassed the evaluation of AKI based on the KDIGO criteria within 7 days,[4] the requirement for renal replacement therapy (RRT), mortality, and microbiological evidence indicating sepsis. Furthermore, the data collection process included recording demographic information, Acute Physiology and Chronic Health Evaluation 2 (APACHE2) scores, SOFA scores, and other pertinent clinical details. AKI was diagnosed either based on an increase in serum creatinine by 0.3 within 48 h or by a decrease in urine output to less than 0.5 mL/kg/h for more than 6 h.

The sample size for this study was calculated using a web-based tool available at http://wnarifin.github.io. Building on the study by Burns *et al.*[11] to attain a sensitivity of 77% and a specificity of 88%, with a 5% alpha error and an absolute precision of 0.15, the total sample size was calculated to be 111 patients.

All data were tabulated in Google Sheets and subjected to statistical analysis by Jamovi (The Jamovi project (version 2.6.2, Sydney, Australia).[12] The normality of the data was evaluated using the Shapiro-Wilk test. All the variables except serum sodium had a skewed distribution. The skewed categorical variable (mortality) was analysed using the Chi-squared test, while the skewed continuous variables were assessed using the Mann-Whitney U test. Demographic data are expressed as median (IQR).

To evaluate the discriminatory ability of various parameters for AKI, receiver-operating characteristic (ROC) curves were generated, and the optimal cut-off values were determined based on Youden's index. Spearman's correlation analysis was employed to examine the relationships between creatinine clearance and the 2-hour excretion of potassium and sodium. Subsequently, logistic regression models were built using the data obtained from univariate analysis to predict the occurrence of AKI.

RESULTS

In this prospective study, 111 patients with suspected sepsis were enroled [Figure 1]. Among this cohort, the incidence of AKI was observed in 42.3% (n = 47) of the cases. Out of 47, 10 patients required renal replacement therapy. A comparison between patients who developed AKI and those who did not reveal that the former group had significantly higher SOFA ($P < 0.001$) and APACHE 2 scores ($P < 0.001$), lower levels of urine sodium ($P = 0.016$) and its derived parameters, and higher FeK values ($P = 0.027$). Other parameters (urine potassium, urine creatinine, serum sodium and potassium, creatinine clearance, FeNa, and 2-hour potassium excretion ratio of urine potassium to urine creatinine) showed no significant differences between the two groups. The overall mortality rate in the study was 34.2%. However, patients who developed AKI had a substantially higher mortality rate ($P < 0.001$) (59.5%) compared to those who did not (14.06%). A summary of all the parameters can be found in Tables 1 and 2.

ROC curves for various parameters were constructed to predict AKI in septic patients. The SOFA score emerged as the most effective predictor with the highest area under the ROC curve (AUROC) (0.69; 95%CI: 0.61, 0.79). When evaluating urinary parameters, the product of urine sodium and urine creatinine exhibited the maximum AUROC (0.66; 95%CI: 0.56, 0.77). Table 3 provides a comprehensive overview, presenting each parameter's AUROC values, corresponding confidence intervals, optimal cut-off values, sensitivity, and specificity [Figure 2].

Spearman's correlation analysis, which assessed the relationship between creatinine clearance and the 2-hour excretion of sodium and potassium, revealed a statistically significant correlation between 2-hour potassium values ($P < 0.001$), with an r-value of 0.62 [Table 4 and Figure 3].

In our analysis, we initially conducted a univariate analysis to identify variables that exhibited significant differences between patients with

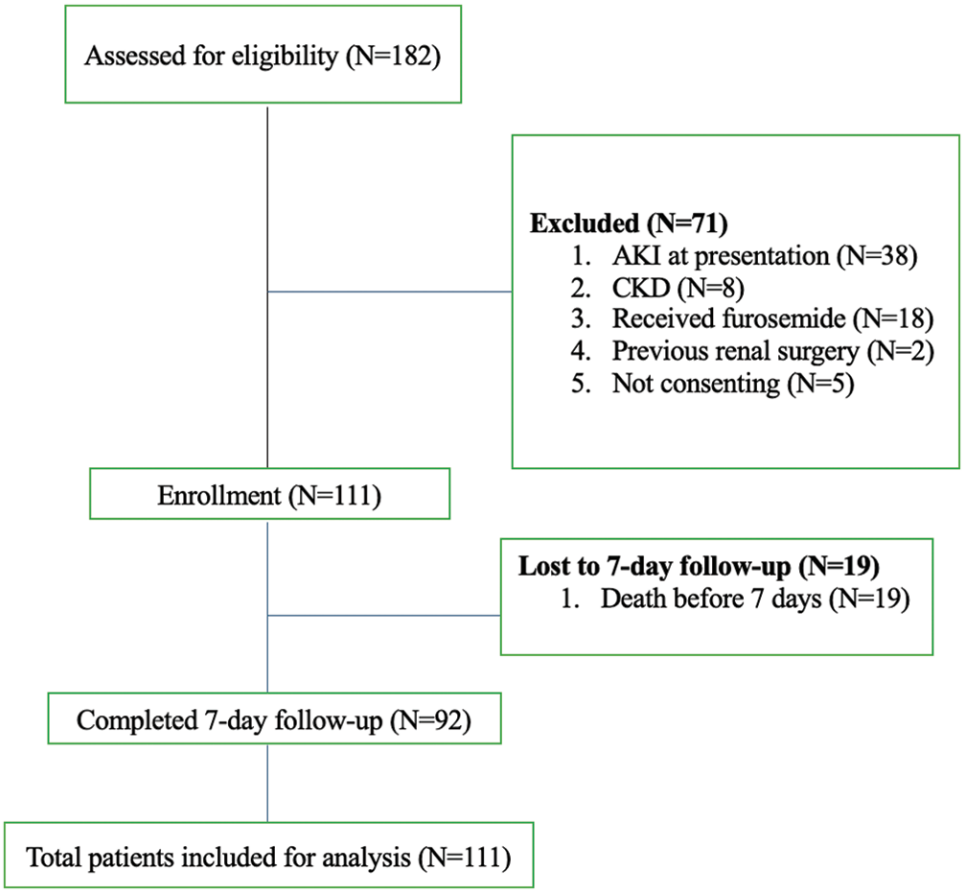

Figure 1: Flowchart of recruitment. AKI = Acute kidney injury, CKD = Chronic kidney disease

AKI and those without AKI. Subsequently, we constructed logistic regression models to predict the occurrence of AKI. To predict AKI, we integrated the variables SOFA score, FeK, and urine sodium concentration into the model. All three variables demonstrated significance, with P = 0.020, 0.044, and 0.033, respectively. The model achieved an AUROC of 0.751 [Table 5 and Figure 4].

DISCUSSION

This study found that fractional excretion of potassium and urine sodium levels had a moderate ability to predict AKI.

These findings of incidence of AKI and mortality rates associated with SA-AKI align with previous studies, where the incidence of SA-AKI ranged from 20% to 70%, and mortality rates associated with AKI varied widely, ranging from 11% to 77% in earlier reports.[2,5,6]

The univariate analysis showed that patients who developed AKI had lower urine sodium levels than those who did not. This discrepancy can be explained by patients with hypovolemia or reduced volume status who may experience decreased renal perfusion, leading to activation of the renin-angiotensin-aldosterone system. This parameter can serve as an indicator for predicting which patients may be at risk of developing AKI.[9,13,14] This suggests the presence of transient AKI, which can be corrected with appropriate fluid management. Out of the 47 patients studied, only 10 required renal replacement therapy, indicating that just 21.2% of patients had persistent AKI necessitating this intervention, while the remaining 78.8% experienced transient AKI.

It is worth mentioning that the urine electrolyte samples were collected during the first day of sepsis when fluid resuscitation was ongoing. This timing may have influenced the findings.

Other derived parameters of urine sodium, such as 2-hour sodium and the product of urine sodium and urine creatinine values, also showed significant differences between the two groups. This can be attributed to the considerable disparity in urine

Table 1: Comparison of Clinical and Laboratory Parameters in Patients With and Without Acute Kidney Injury (AKI) in Sepsis

Parameters	All patients (*n*=111) Median (IQR)	AKI (*n*=47) Median (IQR)	Non-AKI (*n*=64) Median (IQR)	*P*
Age (years)	31 (24-45.5)	35 (25-49)	29.5 (24-43.5)	0.209
Gender (M/F)	57/54	25/22	32/22	
SOFA	4 (2-6)	5 (4-7)	4 (2-5)	<0.001
APACHE II	11 (9-19)	17 (10.5-21)	11 (7.75-14)	<0.001
Mortality (*n* (%))	37 (34.2%)	28 (59.5%)	9 (14.06%)	<0.001
Urine Sodium (mmol/L)	64 (22-108)	36 (14.4,10)	81 (31-101)	0.016
Urine Potassium (mmol/L)	35.9 (23-59.1)	35.9 (24.9-65.6)	35.5 (22.9-55)	0.405
Urine Creatinine (mg/dL)	54.9 (33.5-88.2)	58.5 (29.6-92)	50 (35.5-86.3)	0.979
Serum Sodium (mmol/L)	136 (133-139)	136 (132-139)	136 (133-139)	0.517
Serum Potassium (mmol/L)	4.1 (3.6-4.45)	4.1 (3.6-4.7)	4 (3.5-4.3)	0.570
Serum Creatinine (mg/dL)	0.6 (0.5-0.8)	0.7 (0.6-1)	0.6 (0.4-0.7)	0.004
Urine Volume (mL)	80 (100-120)	100 (80-105)	100 (80-120)	0.453
Creatinine Clearance (mL/min)	71.2 (40.3-136)	64.1 (38.1-115)	75.2 (41-140)	0.200
2-hour Urine Potassium (mmol)	3.35 (2.23-6.06)	3.34 (2.45-6.48)	3.37 (2.15-5.88)	0.414
2-hour Urine Sodium (mmol)	5.58 (2.1-11.3)	3.3 (1.23-10)	7.32 (2.77-12.1)	0.029
FeNa (%)	0.41 (0.13-1.13)	0.40 (0.10-1.18)	0.42 (0.27-1.13)	0.347
FeK (%)	10.2 (7-15.2)	11.7 (7.91-17.3)	9.16 (5.76-12.7)	0.027
UK/UCr (mmol/mg)	0.66 (0.43-1.01)	0.73 (0.49-0.95)	0.61 (0.41-0.95)	0.213
UNa*UCr (mmol*mg/L^2)	2581 (1154-5890)	1968 (814-4124)	3618 (1649-6701)	0.003

Data expressed as median (interquartile range) or numbers. SOFA=Sequential Organ Failure Assessment, APACHE 2=Acute Physiology And Chronic Health Evaluation, FeNa=Fractional excretion of sodium, FeK=Fractional excretion potassium, UK/UCr=Ratio of urine potassium to urine creatinine, UNa*UCr=Product of urine sodium and urine creatinine

Table 2: Different Sources of Sepsis

Source of sepsis	*n* (%)
Pneumonia (CAP/HAP)	23 (20.7)
Bloodstream infection	22 (19.8)
Urinary tract infection	6 (6.3)
Intra-abdominal infections	7 (5.4)
Skin and soft tissue infection	20 (18)
Multiple sources (a combination of the above)	18 (13.5)
Others*	20 (16.2)

*Includes central nervous system infection, tropical fever syndromes, pleural space infection, and undetermined sources of infection

Table 3: Performance Characteristics of Parameters in Predicting Acute Kidney Injury (AKI) in Sepsis Patients

Parameters	AUROC (95%CI)	Best cut-off	Sensitivity	Specificity
SOFA score	0.69 (0.61, 0.79)	5	59.57	71.99
APACHE 2 score	0.67 (0.59, 0.79)	12	48.94	87.5
2-h K	0.54 (0.43, 0.65)	2.4	80.85	32.81
2-h Na	0.62 (0.52, 0.72)	1.7	38.3	90.62
FeK	0.62 (0.51, 0.72)	6.6	91.49	31.25
FeNa	0.55 (0.43, 0.66)	0.11	34.04	90.62
CrCl	0.57 (0.56, 0.77)	95.7	70.21	46.88
UK/UCr	0.56 (0.46, 0.67)	0.54	68.09	46.88
UNa*UCr	0.66 (0.56, 0.77)	2464	63.83	64.05

Data expressed as median (interquartile range) or numbers. SOFA=Sequential Organ Failure Assessment, APACHE 2=Acute Physiology And Chronic Health Evaluation, 2-h K=2-hour excretion of potassium, 2-h Na=2-hour excretion of sodium, FeNa=Fractional excretion of sodium, FeK=Fractional excretion potassium, CrCl=Creatinine clearance, UK/UCr=Ratio of Urine potassium to urine creatinine, UNa*UCr=Product of urine sodium and urine creatinine

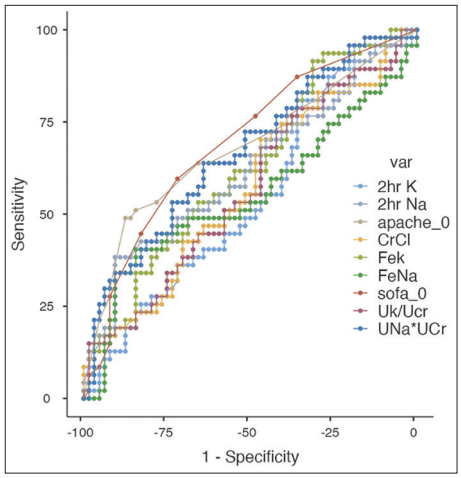

Figure 2: ROC curves showing prognostic performance of various parameters in predicting AKI. ROC = Receiver Operating Characteristic, AKI: Acute Kidney Injury, 2-h K: 2-hour potassium, 2-h Na: 2-hour sodium, APACHE_0 = Acute Physiology And Chronic Health Evaluation score at admission, CrCl = Creatinine Clearance, FeNa = Fractional excretion of sodium, FeK = Fractional excretion potassium, SOFA_0 = Sequential Organ Failure Assessment at admission, UK/UCr = Ratio of Urine potassium to urine creatinine, UNa*UCr = Product of urine sodium and urine creatinine

sodium concentration between the two groups, and these parameters are essentially derived from the same underlying difference. This is also why the product of urine sodium and urine creatinine gives the best AUROC for predicting AKI.

FeK was notably higher in patients who developed AKI than in those who did not. The kidneys handle potassium differently from sodium, particularly

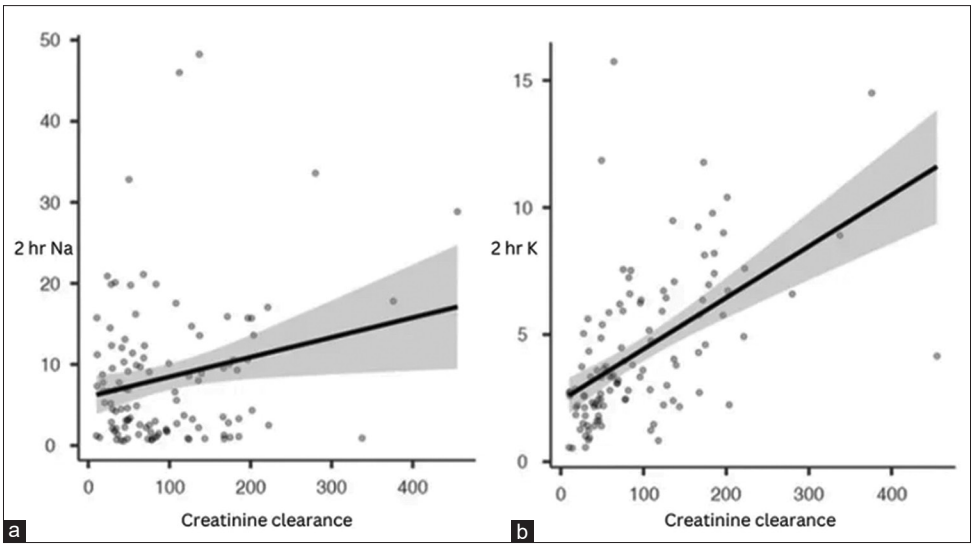

Figure 3: (a) Correlation between 2-hour sodium excretion and creatinine clearance (r = 0.04) and (b) Correlation between 2-hour potassium excretion and creatinine clearance (r = 0.64). 2-h K = 2-hour excretion of potassium, 2-h Na = 2-hour excretion of sodium

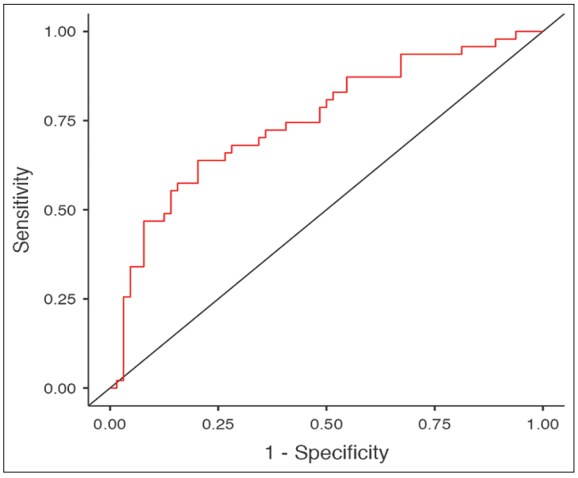

Figure 4: ROC curve of the logistic regression model to predict sepsis-associated acute kidney injury with an AUROC of 0.751. ROC = Receiver Operating Characteristic, AUROC = Area Under the Receiver Operating Characteristic

Table 4: Correlation Analysis Between 2-Hour Urine Electrolyte Excretion and Creatinine Clearance

Parameters	Spearman's rho	Significance (*P*)
2-h K	0.62 (0.38, 0.65)	<0.001
2-h Na	0.04 (0.03, 0.38)	0.641

Data expressed as median (interquartile range) or numbers. 2-h K=2-hour excretion of potassium, 2-h Na=2-hour excretion of sodium

Table 5: Multivariate Logistic Regression Model for Predicting AKI in Sepsis Patients

Parameters	Adjusted odds ratio (95%CI)	Significance (*P*)	AUROC for a given model
SOFA score	1.25 (1.03, 1.52)	0.020	0.751
FeK	1.04 (1.00, 1.08)	0.044	
Urine sodium	0.99 (0.98, 0.99)	0.023	

Data expressed as median (interquartile range) or numbers. SOFA=Sequential Organ Failure Assessment, FeK=Fractional excretion potassium

under stress conditions. In these patients, due to aldosterone secretion, potassium is excreted while sodium is absorbed, increasing the urinary potassium concentration.[9] A significant elevation of FeK has been observed in AKI patients.[10]

A hypothesis suggests that the increase in FeK may result from factors such as low glomerular filtration rate (GFR) or other influences such as aldosterone activity, as proposed by Burns *et al*.[11] Our study showed a strong correlation between 2-hour urinary potassium levels and creatinine clearance, indicating that elevated urinary potassium may directly reflect reduced GFR. However, the exact cause for increased FeK remains uncertain. FeK levels can be higher even when serum potassium and creatinine are within normal ranges if the urine potassium-to-creatinine ratio is elevated. However, this ratio showed no significant difference between groups. One finding that could partially explain higher FeK in our study was a slightly elevated baseline serum creatinine in the AKI group [0.7 (0.6, 1.0) vs 0.6 (0.4, 0.6); $P < 0.001$], a baseline difference not observed in studies where higher FeK levels were associated with AKI onset.[10,15] However, despite the strong correlation, 2-hour potassium excretion was not a robust predictor of AKI in our cohort, with an AUROC of 0.54 (95% CI: 0.43–0.65), in contrast to the findings in the study by Burns *et al*.[11]

Logistic regression model components, which included SOFA score, urine sodium, and FeK, demonstrated statistical significance with *P* values less than 0.05. However, it is important to note that

the effect size, measured in terms of odds ratio, was not clinically significant for urine sodium and FeK, with values of 0.99 and 1.04, respectively. Nevertheless, this model exhibited an impressive AUROC of 0.751. This high AUROC is attributed to the strategic selection of two components in the model, with one providing greater sensitivity and the other greater specificity.

Our study has several limitations. Firstly, we only had a single measurement of urinary electrolyte parameters to predict AKI. Continuous monitoring over time might provide more insight. Secondly, our study included patients in the resuscitation phase, during which significant fluid shifts can occur, potentially altering the urine electrolyte parameters. Thirdly, even though we included cases before the onset of AKI, our study population had statistically significant baseline creatinine levels, suggesting the beginning of AKI. This was due to the absence of baseline values at admission for comparison to detect an increase of 0.3 mg/dL in creatinine.

CONCLUSION

This prospective observational study was carried out to assess the urinary electrolyte parameters in predicting AKI in sepsis patients. Urine sodium levels and fractional excretion of potassium had a moderate ability to predict AKI in sepsis patients. Urine potassium excretion correlates with glomerular filtration rate.

Study data availability

De-identified data may be requested with reasonable justification from the authors (email to the corresponding author) and shall be shared after approval as per the authors' Institution policy.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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