

Impact of Electrolyte Disturbances on Mortality and Hospitalization Among Patients with Chronic Kidney Disease: A Retrospective Observational Study

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Abstract:

Background: Disturbances of electrolytes and acid-base balance are common among CKD patients and usually exacerbate during hospitalization. These disturbances are known to be associated with poor clinical outcomes; nevertheless, information regarding their cumulative effect on mortality, hospitalization, KRT, and renal recovery in hospitalized Indian CKD patients is rather sparse.

Objectives: In order to estimate the prevalence and pattern of electrolyte imbalance among admitted CKD patients, explore their relation with inpatient mortality, hospital stay, and need for KRT, and establish independent predictors of renal recovery in AKI-on-CKD patients.

Methods: This study is a retrospective observational study in which adult patients with pre-existing CKD hospitalized at the multispecialty teaching hospital between January and December 2025 were included. Demographic, clinical, laboratory, and management parameters of the patients were collected through electronic medical records. Electrolyte imbalance was assessed through admission biochemical parameters. Parameters predicting mortality and survival for patients with CKD were determined by multivariate logistic regression analysis. Evaluation of the model was done through ROC curve and Hosmer-Lemeshow goodness-of-fit test.

Results: Among 312 patients, at least one electrolyte disturbance was observed in 92.3%, while 36.5% had three or more concurrent abnormalities. Hyperphosphataemia (54.5%) and metabolic acidosis (49.0%) were the most frequent disturbances. In-hospital mortality was 12.5%, and 28.5% required KRT. Mortality, prolonged hospitalization, and KRT requirement increased progressively with a greater electrolyte disturbance burden. Hyponatraemia was an independent predictor of mortality during hospitalization (adjusted odds ratio [aOR], 2.63; 95% confidence interval [CI], 1.17 to 5.93; P=0.020). In patients with AKI-on-CKD, metabolic acidosis (aOR, 0.40; 95% CI, 0.18 to 0.91), three or more electrolyte abnormalities.

Conclusions: Electrolyte disorders were extremely common in hospitalized patients with CKD and were linked to poor in-hospital outcomes. Higher electrolyte load was related to greater risk of mortality, prolonged hospital stay, need for KRT, and decreased renal recovery. Evaluation of electrolyte disorders could help identify risks at an early stage.

Keywords: Chronic Kidney Disease; Electrolyte Disturbances; Hyponatraemia; Metabolic Acidosis; Renal Recovery; Kidney Replacement Therapy.

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Introduction

Chronic kidney disease (CKD) has become one of the most significant non-communicable diseases of the present era. As per the findings of the Global Burden of Disease reports, around 700 million people were suffering from CKD in 2017, which led to about 1.2 million deaths, and CKD became one of the twelve major causes of death worldwide.[1] The rate of the burden and deaths caused by this disease has increased tremendously

over the last few decades. Furthermore, the burden of this disease is greater in low- and middle-income countries owing to its late diagnosis and unavailability of renal replacement therapy due to various other health issues.[1,2] India is also burdened by the disease. In the adult population, the prevalence rate of the disease by community screening tests was 13% to 17%, and hospital-based prevalence showed a relatively younger age

group with a higher undiagnosed rate than the West.[3,4,5]

Apart from the progressive inability of the kidneys to eliminate waste products, the diseased organ eventually loses its ability to maintain the internal environment in terms of chemicals. Potassium balance, sodium balance, hydrogen ion balance, calcium and phosphate balance are dependent on normal function of the tubules and normal glomerular filtration, so that electrolyte and acid-base disorders are amongst the first and most prevalent complications of CKD.[6] Electrolyte and acid-base disturbances are increasingly common as the estimated glomerular filtration rate gets lower. Electrolyte and acid-base disorders are easily revealed or worsened in the event of acute disease due to the combination of low oral intake, sepsis, medications and AKI in the hospital setting.[6]

The individual electrolyte disturbances have all been associated with poor outcomes. In the CKD Prognosis Consortium meta-analysis, both low and elevated serum potassium levels have been found to predict mortality regardless of the degree of kidney dysfunction, thus establishing the typical U-shaped relationship.[7] More recent studies based on registry and hospital cohorts have confirmed that hyperkalaemia is associated with increased mortality and rapid worsening of kidney dysfunction.[8] Sodium imbalances behave the same way; hyponatraemia and hypernatraemia have independently been found to predict mortality in the Chronic Renal Insufficiency Cohort, while dysnatraemia has also predicted progression to kidney failure.[9] Metabolic acidosis, which is characterized by serum bicarbonate values under 22 mmol/L, develops in quite a large proportion of CKD patients and has been linked to rapid deterioration of renal function, malnutrition, and higher rates of mortality.[10,11] The disorders in calcium-phosphate metabolism and the resulting mineral imbalance contribute to the higher risk of heart diseases in these patients.[6]

With this increasing body of evidence, there still exist some gaps that need to be addressed in terms of practical medicine. Most of the available literature uses ambulatory cohorts in Western countries, where individual electrolytes are studied; however, the sick patient normally suffers from multiple imbalances at once, each contributing to his acute stress state. The contribution of the overall load on electrolytes and acid-base state to in-hospital mortality, in-hospital stay and the need for renal replacement therapy needs to be explored more, especially in South Asian populations. Recovery of renal function is another important indicator as it affects the need for dialysis, readmission rates and the patients' quality of life, but the connection between admission electrolyte

and acid base imbalance and recovery needs further exploration in Indian hospital cohorts.

An observational study involving retrospective analysis of electrolyte and acid base disturbances in adults with CKD admitted in a multispecialty teaching hospital in India over the course of one year was hence conducted. There were three objectives of the study including describing the pattern and incidence of electrolyte and acid base disturbance at admission, examining the correlation between electrolyte and acid base disturbances and in-hospital mortality, prolonged hospital stay and need for renal replacement therapy; and identification of independent factors predicting renal recovery. In view of the patients being admitted in India and having regard for electrolyte disorders as a whole and not in isolation, we endeavored to gather the evidence which may be useful in recognizing those at risk and prioritizing correction of the disorders.

Materials & Methods

Study Design and Setting: The study was conducted on a retrospective observational cohort basis in a multispecialty tertiary care teaching hospital in India. All adults suffering from CKD admitted to the hospital during the period from 1st January 2025 to 31st December 2025 were included in the study. The hospital is equipped with facilities to serve both urban and semi-urban communities in all aspects of nephrology from acute cases to those on maintenance hemodialysis. This study complies with the recommendations for observational studies as per the STROBE guidelines.[15]

Participants: For inclusion in the study, participants were required to be aged 18 years or more and to suffer from a condition known as CKD (chronic kidney disease), according to KDIGO definition, that means that their estimated glomerular filtration rate was 60 ml/min/1.73 m² or below with signs of kidney damage for three months or longer prior to admission. [12] For the calculation of eGFR, 2021 CKD-EPI creatinine equation was applied, and the stages of CKD were determined to be G1-G5.[13] Those on chronic dialysis, having a kidney transplant, being pregnant, and those with the admission duration of less than 24 hours or incomplete biochemistry on admission were excluded. Only one index hospitalization of each patient was taken into account, and, if there were several admissions within the study period, only the first one was analyzed.

Variables and Definitions: The principal exposures included electrolyte and acid-base disturbances as determined through laboratory tests conducted within the first 24 hours of admission to

the hospital. Electrolyte disturbances were referred to as hyperkalemia if the serum potassium level was greater than 5.5 mmol/L and hypokalemia if the level was below 3.5 mmol/L. The sodium disturbances were regarded as hyponatremia if the serum sodium level was below 135 mmol/L and hypernatremia if it was above 145 mmol/L. The metabolic acidosis was indicated by the serum bicarbonate level below 22 mmol/L while hypocalcemia was associated with serum albumin-corrected calcium levels below 8.5 mg/dL. Hyperphosphatemia was characterized by serum phosphate levels above 4.5 mg/dL. In order to capture the burden of biochemical disturbances regardless of the effect of individual electrolyte disturbances, an electrolyte disturbance score was computed.

The first outcome measured was in-hospital mortality from any cause. Additional outcomes that were evaluated included hospital stay lasting more than seven days; need for KRT in hospital stay; and renal recovery at discharge. Acute kidney injury with CKD (AKI-on-CKD) was determined using serum creatinine KDIGO criteria where baseline serum creatinine was the minimum value measured over the last three to twelve months, or calculated based on previous values in case there is no information about it.[14] Recovery of the kidneys was measured only in patients with AKI-on-CKD and was determined when serum creatinine became 25% higher than baseline and patient became independent from dialysis. Covariates included age, sex, comorbid conditions like hypertension, heart failure, diabetes mellitus, coronary artery disease, baseline stage of CKD, CKD etiology, AKI etiology and the presence of sepsis at admission.

Data sources and Measurement: The data were collected from the hospital EMR and the laboratory information system using a case-record form in a retrospective manner. Biochemical analysis was carried out at the central hospital laboratory that participates in the external quality assurance programme; serum electrolytes and creatinine were measured using the ion-selective electrode method and modified Jaffe kinetic assay, respectively. The data were independently collected by two investigators and any discrepancies were solved by consulting a nephrologist.

Study Size and Bias: Given that the study involved including all admissions eligible for inclusion within a specified one-year period, there was no prior sample size calculation. In terms of the multivariate analysis, the number of predictor variables included was limited in order to ensure an adequate ratio of outcome events to predictors to avoid over-fitting. The selection bias was reduced through consecutive enrolment of all eligible admissions, while information bias was controlled through the application of pre-defined objective

laboratory criteria as well as double data abstraction. Confounding cannot be ruled out because of the observational study design.

Statistical Analysis: For continuous variables, normality tests were done using Shapiro-Wilk, while categorical variables were described as proportions and frequency. For between-group comparison, continuous variables were tested using t-test or Mann-Whitney U-test, while categorical variables were tested using chi-square or Fisher's exact test. Univariate logistic regression analysis was done prior to multivariate model building, and only those variables that met $P < 0.10$ were used. Also, all clinically important covariates were added to the model. The odds ratios (ORs) and 95% confidence interval (CI) were provided. The performance of the logistic model was assessed using the Hosmer-Lemeshow goodness-of-fit test and ROC-AUC. The statistical analysis was two-sided, with $P < 0.05$ being statistically significant. All analyses were done using IBM SPSS statistics version 27.

Ethical Considerations: The project was approved by the Institutional Ethics Committee, which issued a waiver on the need for individual informed consent based on its non-interventional nature and retrospective design with anonymized information. The study was carried out following the principles of the Declaration of Helsinki.

Results

Participants: In the year 2025, 419 patients admitted with CKD were evaluated for their inclusion in the study. On using the established criteria for excluding patients from the study, 312 patients formed the sample size of the study. Patients who were on maintenance dialysis prior to admission, patients having undergone renal transplantation, pregnant women, patients staying less than 24 hours in the hospital, and patients without complete records of biochemical parameters at the time of admission were considered out of the study group. The study group was then analyzed based on their in-hospital survival rates.

Baseline Characteristics: Demographic and clinical baseline data are shown in Table 1. The majority of the study group were middle-aged and elderly with slight male predominance. Hypertension was the most common co-morbidity followed by diabetes mellitus, coronary artery disease, and heart failure. At admission sepsis was seen in about one-third of the cohort.

Advanced CKD was common, where G4 and G5 disease contributed substantially to the study population. The prevalence of CKD staging was G2 in 31 patients, G3 in 94, G4 in 96, and G5 in 91 patients. The mean baseline eGFR highlighted the

severity of renal disease. The main causes of CKD included hypertensive nephrosclerosis, diabetic nephropathy, CKD of uncertain cause, chronic glomerulonephritis, and obstructive/other renal diseases (Figure 1).

In comparison of survivors and non-survivors, non-survivors were found to be significantly older. The proportion of AKI on top of CKD was much higher in the patients who succumbed during the course of

their hospital stay. However, other factors such as gender, diabetes, hypertension, coronary artery disease, heart failure, stage of CKD, and eGFR did not demonstrate statistically significant difference between the two survival groups. It appears that while there was an extensive presence of advanced CKD and comorbid conditions in the entire study population, age and presence of an AKI element formed the key baseline variables.

Table 1: Baseline demographic and clinical characteristics of the cohort, overall and by in-hospital survival.

Characteristic	Overall (n = 312)	Survivors (n = 273)	Non-survivors (n = 39)	P-value
Age, years, mean $\pm$ SD	53.8 $\pm$ 14.0	52.8 $\pm$ 14.0	60.5 $\pm$ 12.4	<0.001
Male sex, n (%)	183 (58.7)	164 (60.1)	19 (48.7)	0.241
Diabetes mellitus, n (%)	153 (49.0)	131 (48.0)	22 (56.4)	0.416
Hypertension, n (%)	234 (75.0)	203 (74.4)	31 (79.5)	0.621
Coronary artery disease, n (%)	59 (18.9)	52 (19.0)	7 (17.9)	1.000
Heart failure, n (%)	49 (15.7)	42 (15.4)	7 (17.9)	0.860
Sepsis at admission, n (%)	96 (30.8)	79 (28.9)	17 (43.6)	0.095
AKI-on-CKD, n (%)	168 (53.8)	134 (49.1)	34 (87.2)	<0.001
CKD stage G4–G5, n (%)	187 (59.9)	160 (58.6)	27 (69.2)	0.275
Baseline eGFR, mL/min/1.73 m ² , mean $\pm$ SD	30.4 $\pm$ 20.9	30.8 $\pm$ 20.8	27.6 $\pm$ 21.1	0.378

CKD stage distribution: G2, 31 (9.9%); G3, 94 (30.1%); G4, 96 (30.8%); G5, 91 (29.2%).

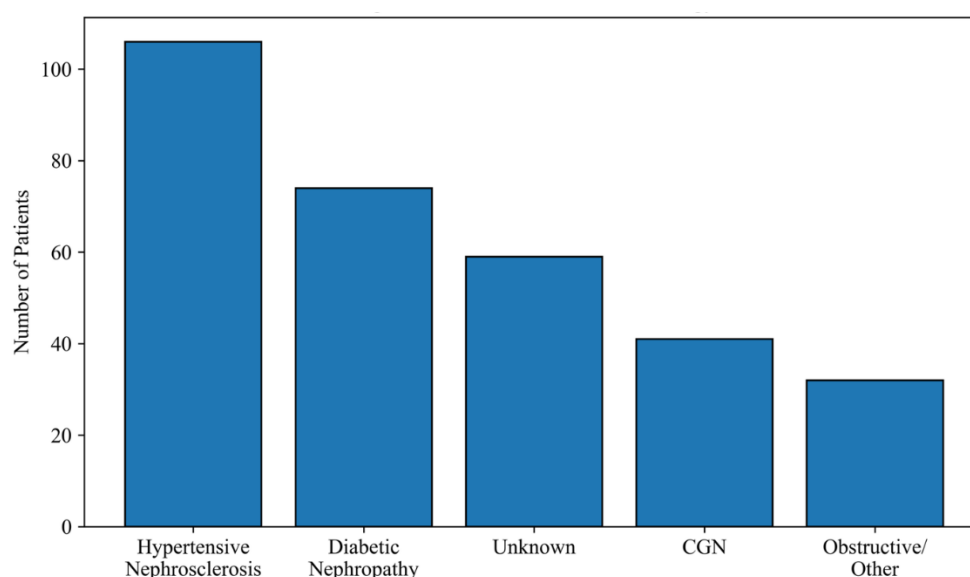


Figure 1: Distribution of assigned CKD aetiology.

Laboratory findings and electrolyte disturbances: Findings from the admission laboratory tests and the frequencies of electrolyte and acid–base disturbances are shown in Table 2 and Figure 2. Both electrolyte disturbances and acid–base disturbances were very prevalent in the studied population, and a large group of patients had several abnormalities at once.

Hyperphosphataemia and metabolic acidosis were the most common disturbances. Hyponatraemia and hypocalcaemia were also quite common, while

hyperkalaemia was rare. Hypernatraemia and hypokalaemia were relatively rare (Figure 2). Comparing according to survival showed that there were significant differences in some biochemical markers. Non-survivors had lower levels of sodium and bicarbonate in their blood than the survivors did. Also, the frequency of hyponatraemia was much higher in non-survivors, and this disturbance was the most prevalent one in terms of mortality.

However, for potassium, corrected calcium, phosphate, and haemoglobin, there was no

statistically significant difference noted between the survival and non-survival groups. Likewise, the prevalence rates of hyperkalemia, hypokalemia, hyponatremia, metabolic acidosis, hypocalcemia, and hyperphosphatemia did not show any

statistically significant difference individually. Hence, while numerous electrolyte imbalances were common among the subjects, hyponatremia seemed to be especially associated with negative hospital outcomes.

Table 2: Admission laboratory parameters and prevalence of electrolyte and acid–base disturbances, overall and by in-hospital survival.

Parameter	Overall (n = 312)	Survivors (n = 273)	Non-survivors (n = 39)	P-value
Sodium, mmol/L, mean $\pm$ SD	137.0 $\pm$ 5.3	137.2 $\pm$ 5.3	135.1 $\pm$ 5.0	0.020
Potassium, mmol/L, mean $\pm$ SD	4.9 $\pm$ 0.8	4.9 $\pm$ 0.8	5.1 $\pm$ 0.8	0.179
Bicarbonate, mmol/L, mean $\pm$ SD	22.3 $\pm$ 3.7	22.4 $\pm$ 3.7	21.2 $\pm$ 3.6	0.046
Corrected calcium, mg/dL, mean $\pm$ SD	8.8 $\pm$ 0.8	8.8 $\pm$ 0.8	8.7 $\pm$ 0.7	0.167
Phosphate, mg/dL, mean $\pm$ SD	4.7 $\pm$ 1.3	4.6 $\pm$ 1.3	4.9 $\pm$ 1.3	0.306
Haemoglobin, g/dL, mean $\pm$ SD	9.3 $\pm$ 1.6	9.3 $\pm$ 1.6	9.3 $\pm$ 1.5	0.890
Hyperkalaemia, n (%)	73 (23.4)	61 (22.3)	12 (30.8)	0.337
Hypokalaemia, n (%)	11 (3.5)	11 (4.0)	0 (0.0)	0.371
Hyponatraemia, n (%)	105 (33.7)	82 (30.0)	23 (59.0)	<0.001
Hypernatraemia, n (%)	23 (7.4)	21 (7.7)	2 (5.1)	0.751
Metabolic acidosis, n (%)	153 (49.0)	129 (47.3)	24 (61.5)	0.134
Hypocalcaemia, n (%)	105 (33.7)	91 (33.3)	14 (35.9)	0.892
Hyperphosphataemia, n (%)	170 (54.5)	146 (53.5)	24 (61.5)	0.439

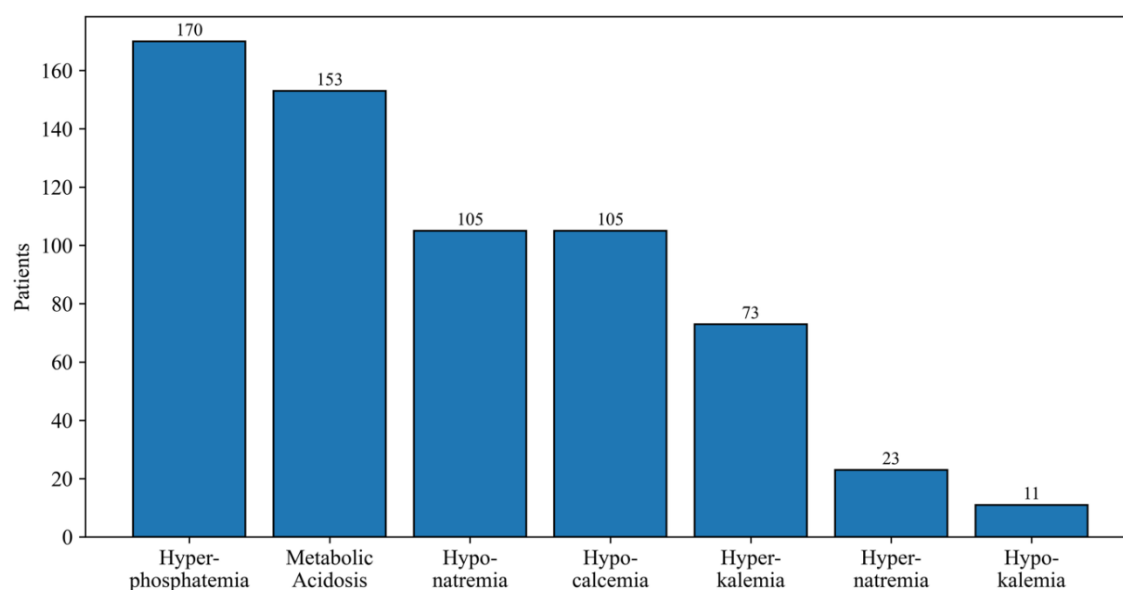


Figure 2: Prevalence of electrolyte and acid–base disturbances at admission.

Clinical Course, Kidney Replacement Therapy and Outcomes: Among the patients who suffered from AKI associated with CKD, sepsis was the most common causative agent. Other important causes included volume depletion, nephrotoxicity or medication-induced injury, contrast nephropathy or other factors, and obstructive mechanisms.

Quite a significant number of AKI associated with CKD patients received renal replacement therapy while in the hospital. The average length of stay in the hospital was quite short; however, the length of stay among those who died was quite significantly longer than the rest. The rate of mortality in the hospital was quite low. Among patients suffering

from AKI associated with CKD, less than half were able to recover renal function by discharge.

Among the AKI superimposed on CKD patients, the main precipitating cause was sepsis. Other factors contributing to AKI were pre-renal/volume depletion conditions, nephrotoxic/drug-related insults, contrast and other causes, and obstruction.

A significant trend existed between the burden of coexisting electrolyte and acid-base disturbances and poor clinical outcomes. As the burden increased, there was a trend towards higher mortality and requirement for renal replacement therapy, and prolonged hospitalization (Table 3). There was also a trend towards higher lengths of

stay as the burden increased. The trend was very obvious among those with three or more concurrent disturbances, who had the highest burden of

adverse events. The dose-response relation indicates that the burden of metabolic disturbances is more predictive than an individual disturbance.

Table 3: In-hospital outcomes according to the number of coexisting electrolyte and acid–base disturbances at admission.

Number of disturbances	n	In-hospital mortality, n (%)	Kidney replacement therapy, n (%)	Prolonged stay >7 days, n (%)	Median LOS, days
0	24	1 (4.2)	2 (8.3)	2 (8.3)	5
1	84	6 (7.1)	12 (14.3)	21 (25.0)	5
2	90	10 (11.1)	22 (24.4)	26 (28.9)	6
≥3	114	22 (19.3)	53 (46.5)	49 (43.0)	7
P (across groups)		0.034	<0.001	0.002	—

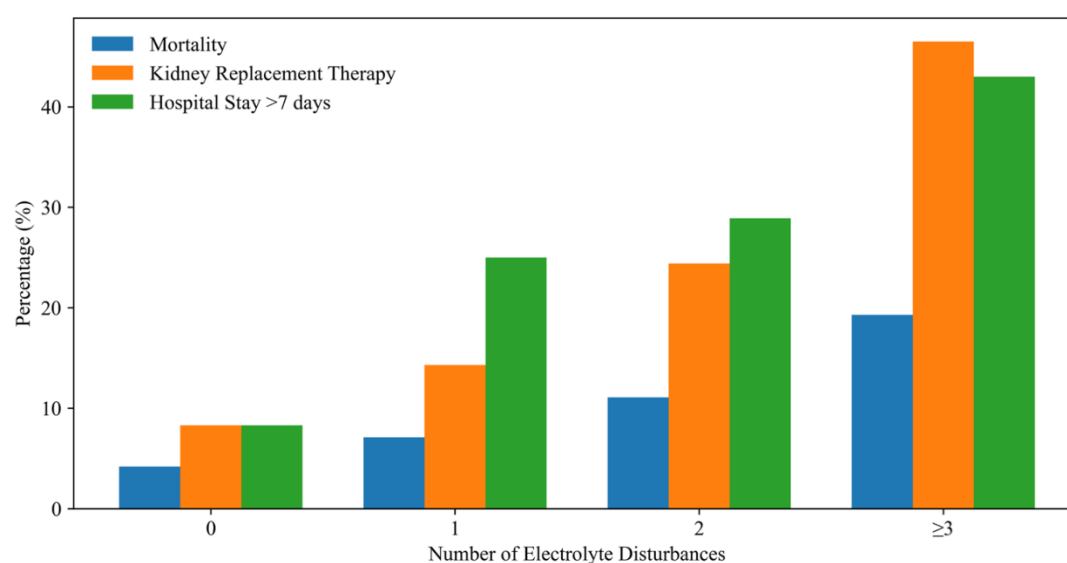


Figure 3: Gradient of adverse in-hospital outcomes across increasing electrolyte disturbance burden (0, 1, 2, ≥3 disturbances).

Association of Electrolyte Disturbances with Mortality: Logistic regression analysis of single variables showed a number of variables associated with in-hospital mortality (see Table 4). Increasing age was found to be associated with higher mortality risk, whereas AKI-on-CKD had a very strong correlation with mortality. Hyponatraemia was also found to be correlated with mortality; moreover, the risk of death was increasing with each electrolyte or acid-base disturbance.

Unlike sepsis on admission, this factor did not have a statistically significant effect in the univariable model. Metabolic acidosis and hyperkalaemia were positively correlated with mortality; however, the latter two factors did not have a statistically significant effect on mortality. Moreover, advanced stage of CKD was not independently associated with mortality either.

Table 4: Univariable logistic regression for in-hospital mortality.

Predictor	Odds ratio (95% CI)	P
Age (per year)	1.04 (1.02–1.07)	0.002
Sepsis at admission	1.90 (0.96–3.76)	0.067
AKI-on-CKD	7.05 (2.68–18.58)	<0.001
Hyperkalaemia	1.54 (0.74–3.23)	0.248
Hyponatraemia	3.35 (1.68–6.67)	<0.001
Metabolic acidosis	1.79 (0.90–3.55)	0.098
CKD stage ≥ G4	1.59 (0.77–3.27)	0.208
Electrolyte disturbance count (per additional)	1.53 (1.13–2.06)	0.005

Multivariable Predictors of Renal Recovery: Among the patients who had AKI-on-CKD, multivariable logistic regression analysis was used

to determine independent predictors of renal recovery at discharge. Table 5 provides information about these findings. Metabolic acidosis was an

independent predictor of non-renal recovery when other predictors were adjusted for in the model. Likewise, the presence of three or more electrolyte disturbances was also an independent predictor of non-recovery, thus reinforcing the finding that the degree of biochemical derangement had been associated with renal recovery.

Need for renal replacement therapy was another independent predictor of non-recovery. In all probability, this was because the patients requiring such treatment had severe insults to their kidneys and more severe illnesses overall. Age showed a trend towards poorer renal recovery with increasing age; however, it failed to achieve statistical significance. Also, the group with CKD G5 had a trend towards non-recovery, although again, this did not achieve statistical significance.

Sepsis-related AKI or hyperkalaemia was not independently associated with recovery in the

multivariable analysis. On the contrary, pre-renal or volume depletion AKI had a trend toward being more likely to have renal recovery; however, this relationship was not statistically significant after adjustment. The resulting model provided satisfactory discriminatory power, indicated by area under the ROC curve, meaning its ability to discriminate between patients who achieved renal recovery and those who did not recover well. The calibration of the model was also satisfactory, according to the Hosmer-Lemeshow goodness-of-fit test.

In summary, multivariable analysis results confirm the importance of the severity and accumulation of electrolyte and acid-base disorders, along with the requirement for kidney replacement therapy in achieving renal recovery in patients with AKI on CKD.

Table 5: Multivariable logistic regression: independent predictors of renal recovery among patients with AKI-on-CKD (n = 168).

Predictor	Adjusted odds ratio (95% CI)	P
Age (per year)	0.97 (0.94–1.00)	0.054
Sepsis-associated AKI	0.99 (0.45–2.16)	0.981
Metabolic acidosis	0.40 (0.18–0.91)	0.029
Hyperkalaemia	1.13 (0.43–2.95)	0.810
≥3 electrolyte disturbances	0.39 (0.17–0.89)	0.026
CKD stage G5	0.42 (0.17–1.06)	0.066
Required kidney replacement therapy	0.40 (0.17–0.98)	0.044
Prerenal/volume-depletion AKI	1.93 (0.79–4.70)	0.147

Model performance: area under the ROC curve 0.808; Hosmer–Lemeshow $\chi^2 = 6.12$, df = 8, P = 0.634.

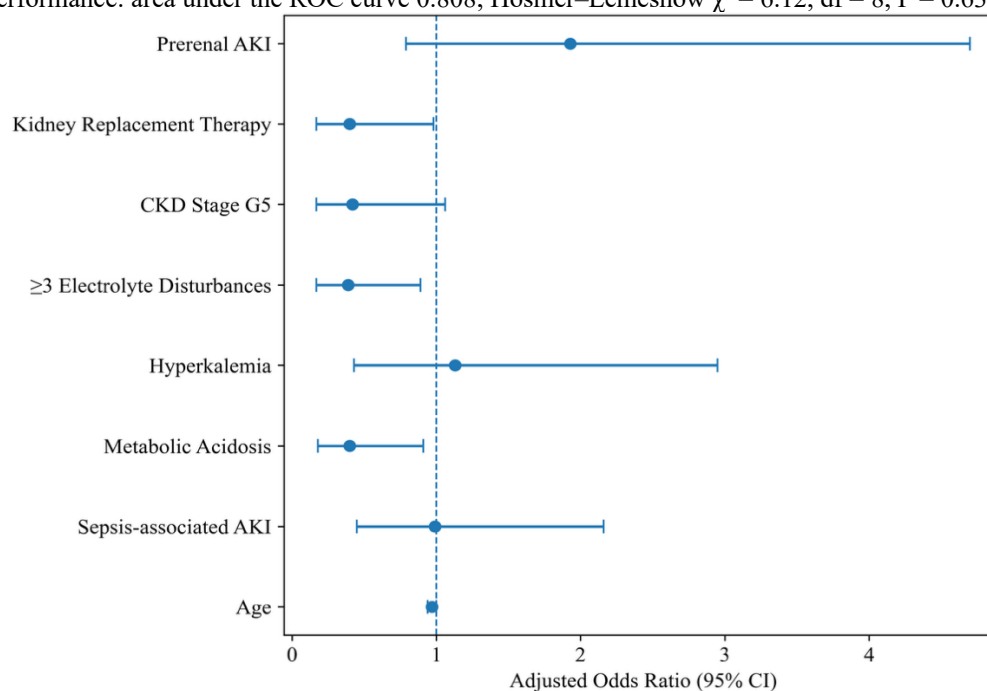


Figure 4. The Receiver operating characteristic curve for the multivariable model predicting renal recovery among patients with AKI-on-CKD.

Discussion

In this single centre retrospective study involving 312 hospitalised adults with CKD, electrolyte and acid-base disorders on admission were very common, occurring in over nine out of every ten patients, with three or more co-existing disorders seen in more than one third of the cohort. The cumulative burden of electrolyte disorders was related in a dose-response fashion with poor in-hospital outcome parameters – mortality, need for kidney replacement therapy and longer length of stay all increased incrementally with increasing number of electrolyte disorders. Amongst individual disorders, hyponatraemia was associated independently with mortality, while metabolic acidosis, having three or more disorders and the need for kidney replacement therapy each independently were associated with decreased probability of renal recovery in those with AKI on top of CKD. These observations build upon those that have largely been established in an ambulatory and Western cohort.

The role of hyponatremia in predicting mortality is consistent with the current scientific data. Both hyponatremia and hypernatremia were identified as independent risk factors for mortality among patients from the Chronic Renal Insufficiency Cohort Study, while dysnatremia was revealed to correlate with the development of end-stage renal disease.[9] In turn, outside the group of CKD patients, hospitalization because of mild hyponatremia correlates with higher rates of mortality.[16] Whether hyponatremia is an independent risk factor for mortality or merely an indicator of severity of the primary disorder has not been elucidated, but, in any case, our cross-sectional admission measure allows for differentiating between these two aspects.

Metabolic acidosis was found to have an independent association with the non-recovery of kidneys rather than death. This pattern is in line with literature on the link between low bicarbonate levels and progression of CKD [10,17,18] and also with findings of trials and cohort studies that show that acidosis is a modifiable risk factor in the progression of CKD. Acidosis in the acute phase is not only due to loss of renal buffers but also due to the effects of the illness, and thus there exists biological plausibility in this association with slow recovery. However, this is observational data and hence cannot imply causality with recovery of acidosis, which needs to be tested in prospective manner.

No association was observed between hyperkalemia and in-hospital mortality, in spite of the known significance of this laboratory marker as an independent predictor of mortality. However, the existence of a U-shape relationship between

serum potassium concentration and mortality rate has been observed in numerous large-scale studies in the context of varying degrees of renal function impairment [7,19], while the existence of hyperkalemia has also been regarded as an independent predictor of hospitalization and rapid progression of renal dysfunction [17,20]. It may be assumed that there are several explanations for the low evidence of the relation in our study group, such as lack of repeated tests, fast normalization of serum potassium level during the period of hospitalization, low frequency of events and impact of the simultaneous development of metabolic acidosis and acute kidney injury. Our results could thus be regarded as an illustration of the prognostic significance of electrolytes' total condition at the moment of hospitalization and not as an independent pathology.

The most important clinical observation from this study could be the clear dose-dependent risk gradient related to the total number of disturbances. The patients having three or more abnormalities showed a considerably higher need for dialysis, prolonged stay in the hospital, and significantly decreased likelihood of regaining normal kidney function. This dose-response relationship is similar to the observations in acutely ill and elderly patients, where a combined electrolyte disturbance was associated with increased mortality and increased length of stay compared to any single abnormality, and it provides support to the concept of using the electrolyte panel as an easily calculated composite marker of physiological dysfunction. Since the measured variables are available at admission, their total number could have some practical value for assessing risks.

These associations between AKI-on-CKD and mortality, as well as the role of the need for kidney replacement therapy and the stage of CKD, in influencing renal recovery, are consistent with the current literature on AKI and recovery.[19,20] Over half of the cohort had superimposed AKI, predominantly sepsis-related, emphasizing that the hospitalized CKD patient within this context often has an acute, and possibly reversible insult, added to their underlying chronic condition. The finding that renal recovery occurred in less than half of patients with AKI-on-CKD demonstrates an outcome that carries significant prognostic and economic implications and is influenced by the biochemical environment at admission.

A number of limitations are worth highlighting. The single-centre, retrospective study design reduces the possibility of making any generalisations or establishing any causality; possible confounding by other variables, such as renin-angiotensin system blockers, diuretics, and potassium binders and alkalis, is unavoidable. Because exposures were determined by a single

admission biochemistry test, we could neither measure any changes that occurred during the hospitalisation period nor differentiate between transient and persistent derangements. The baseline creatinine levels were derived using back calculation for some participants, which can lead to misclassification of AKI-on-CKD. The renal recovery was evaluated during discharge and not over a prolonged period, as the outcomes were only for the index episode, hence mortality and dialysis dependency could not be captured after discharge. The paucity of hypokalemia and hypernatremia makes it difficult for any statistical power for such subgroups, while the high prevalence of CKD whose cause is unknown represents a recognized issue among Indians but restricts any etiological inference.

The above-mentioned limitations are offset by several strengths, namely consecutive enrollment of all eligible admissions over a specified time period, use of pre-specified objective laboratory cutoffs, independent double data extraction, STROBE compliance, and taking into account the burden of electrolyte derangement as well as each one independently. The multivariate analyses demonstrated excellent discrimination and satisfactory calibration, which adds support to the validity of the main findings. Notably, the research provides inpatient data from a lower middle-income country, where prevalence of CKD is high, the disease tends to occur earlier and the cause of the high burden of disease is not known.[1,4,5]

The electrolyte and acid-base disorders were prevalent in the hospitalized patients with CKD and were significantly associated with poor in-hospital outcomes, with a dose-dependent effect of the number of co-occurring disorders on the hazards of death, dialysis and increased duration of hospital stay. Hyponatremia at admission was independently associated with mortality, while acidosis and electrolyte disorder burden were independently associated with lower rate of recovery of kidney function. The assessment of the patient's electrolyte state at admission, in its entirety, can help identify at-risk patients; however, further research is required to prove the effectiveness of systematic screening and correction.

Conclusion

In this retrospective observational study involving hospitalized patients with chronic kidney disease, electrolyte and acid-base disorders were very common at presentation and showed significant correlation with various hospital-related outcomes. Having multiple electrolyte abnormalities at the same time was associated with increasing incidence of death, longer hospital stay, and need for renal replacement therapy, which implies that the sum

total of electrolyte disorders can be clinically significant as an additional prognostic factor. The following factors were significantly associated with reduced chances of recovery of renal function among CKD patients who develop AKI: metabolic acidosis, the occurrence of three or more electrolyte disturbances, and need for renal replacement therapy. On the other hand, hyponatraemia was independently predictive of in-hospital mortality among these patients. These results indicate that it is essential to perform the evaluation of the total electrolyte status upon admission for all hospitalized patients with CKD as part of the diagnostic process. Even though it is not possible to make any causal conclusions due to the observational nature of the study, the observed associations might be helpful in early identification and monitoring of patients at increased risk for unfavourable outcomes. Further multicentre longitudinal studies are needed to confirm the current results and explore whether early correction of electrolyte imbalance can contribute to better patient outcomes.

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