

Acute Kidney Injury Overlying Chronic Kidney Disease: Clinical Features and Predictors for Renal Recovery of Patients and Inpatient Outcome

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ABSTRACT

Background: The combination of acute kidney injury (AKI) with chronic kidney disease (CKD) has been found to have negative implications for renal as well as overall prognosis. However, there is little evidence available on renal recovery and factors influencing it in patients of AKI-on-CKD admitted in Indian hospitals. **Aims:** The aims of this study were to describe the clinical profile of patients with AKI-on-CKD, determine the factors associated with renal recovery, and their in-hospital outcomes. **Materials and Methods:** A retrospective cohort study was done on adult patients having existing CKD complicated by AKI, admitted to the hospital during the year 2025. Data on demographic, clinical, biochemical, and treatment variables were collected through the electronic medical record system. Renal recovery at discharge served as the primary outcome measure. **Results:** A total of 312 patients were recruited, of whom slightly more than half achieved renal recovery. Mortality in-hospital was 17.3% and 41.0% of patients needed renal replacement therapy (RRT). Diabetic nephropathy was the commonest etiology of the underlying CKD, while sepsis was the commonest trigger of AKI. Baseline better renal function was associated with higher chances of renal recovery. However, the presence of severe AKI, sepsis-related AKI, RRT, hypoalbuminemia, and high comorbidities burden were independent predictors of renal non-recovery. **Conclusion:** AKI-on-CKD was associated with significant mortality and incomplete renal recovery. The following variables have been identified as independent predictors of recovery from renal failure: Baseline renal function, AKI severity, sepsis, nutrition, burden of comorbidity, and RRT. Identifying such characteristics might assist in early identification of patients with high-risk renal complications.

Keywords: Acute kidney injury, Acute kidney injury-on-chronic kidney disease, Chronic kidney disease, Prognostic factors, Renal recovery, Renal replacement therapy

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INTRODUCTION

There is a gradual increase in the recognition of the connection between acute kidney injury (AKI) and chronic kidney disease (CKD), which were conventionally seen as separate pathological conditions. The individuals having CKD before the onset of acute disease will have a higher probability of developing AKI, and vice versa; and the development of AKI will hasten the progress of CKD. This bidirectional process has been termed as AKI-CKD Continuum.^[1,2] The presence of AKI along with CKD, known as AKI-on-CKD, is therefore unique and different from that of AKI without any prior CKD.^[3]

The decreased nephron mass and the inability of functional reserve seen in CKD make the kidney susceptible to further damage in the form of sepsis, nephrotoxicity, dehydration, and obstructive uropathy.^[4] The small amount of further insult in a kidney that already lacks functional reserve results in an exaggerated fall in the glomerular filtration rate (GFR), and the prognosis after such an event is usually worse than AKI in patients not having CKD.^[5,6] Multiple large cohort studies have shown that AKI on the basis of CKD is linked with greater risk of incomplete renal recovery, progression to end stage kidney disease, and higher short- and long-term mortality than AKI alone.^[7,8] Despite the fact that AKI-on-CKD is widely recognized, it is under-researched, particularly in low- and middle-income countries where the epidemiology, etiology, and access to renal replacement therapy (RRT) is different compared to the higher income nations.

The worldwide burden of CKD has increased in a consistent fashion during the past two decades due to the rising prevalence of diabetes mellitus, hypertension, and an aging population, but

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India is not immune to this pattern. In the Indian subcontinent, CKD commonly overlaps with a unique range of underlying conditions that precipitate AKI, such as tropical infections, sepsis from different sources, nephrotoxic use of herbal or over-the-counter drugs, and late referral to tertiary centers, which can affect the incidence and outcome of superimposed AKI. Furthermore, the clinical distinction between AKI on CKD and progression of CKD alone may be difficult in many cases, especially in the absence of information regarding pre-existing renal function, an issue that is even more problematic in cases with fragmented outpatient records.^[9,10]

The KDIGO consensus has led to formulation of standard diagnostic and staging criteria for AKI, which are being increasingly

used for patients with pre-existing CKD if the baseline creatinine levels are known.^[11,12] Recovery of renal function post-AKI, defined by various studies as normalization of serum creatinine levels to near baseline values or absence of need for dialysis at the time of hospital discharge, has been recognized as an important outcome measure in the context of short- and long-term prognosis of AKI.^[13] Identification of risk factors for recovery of kidney function post-AKI, including the severity of AKI, use of RRT, sepsis, and status of CKD, is very relevant to patient management, but there is lack of evidence from tertiary care hospitals in South Asia.^[14]

The existing studies regarding AKI in CKD have been conducted primarily in patients from North America and Europe where the etiology, comorbid conditions, and health care infrastructure are quite different from that in Indian teaching institutions. The information about this condition is lacking in Indian institutions and the few studies available have been marred by small sample size, non-uniform criteria of defining this entity, or focusing merely on one of the components (AKI or CKD). A knowledge about this condition would help us to recognize the condition early, avoid unnecessary use of nephrotoxic drugs, and prognosticate the outcome of the patient.^[15]

In view of this, the objective of our retrospective observational cohort study is to report the clinical profile of patients with AKI with CKD in a tertiary multispecialty teaching hospital of India. The main aims of the study included defining the etiological profile of the background CKD as well as the superimposed AKI, finding out the prevalence of patients with renal recovery at the time of discharge and identification of independent predictors of renal recovery. Additional aims of the study included characterization of the requirement of RRT, duration of stay in the hospital, and mortality rate among these patients to obtain locally applicable data.

MATERIALS AND METHODS

Study Design and Setting

The present study was an observational cohort study conducted in a tertiary care hospital in India. The study adhered to the STROBE criteria for observational cohort studies in epidemiology.^[1] The tertiary care hospital is a referral center for nephrology, internal medicine, critical care, and surgery and receives admissions from both urban and semi-urban catchment areas.

Study Period and Data Source

Patients admitted to the hospital from January 1, 2025, to December 31, 2025, who met the case definition for AKI with underlying CKD were retrieved using the patient records maintained in the electronic database of the hospital and the registration records from the nephrology department. The data were collected retrospectively from the medical records, laboratory reports, and discharge summary notes independently by two investigators.

Case Definition

CKD was categorized using the KDIGO definition as reduction in kidney function and evidence of damage that was present for a period of more than 3 months before hospitalization, determined through estimated GFR (eGFR), creatinine, radiological findings, or

medical history.^[15] The diagnosis of AKI was made where there was an increase of ≥ 0.3 mg/dL in serum creatinine levels within 48 h or by ≥ 1.5 times in the first 7 days.^[16] Baseline creatinine was taken as the lowest value documented in the past 3 months, and in its absence, 75 mL/min/1.73 m² eGFR from MDRD was assumed.^[7,17]

Participants

Those who had a diagnosis of CKD before superimposing AKI during their index hospitalization or those who came to hospital admission with AKI based on a pre-existing condition of CKD were included in the study. The participants whose CKD status was stage 5 under maintenance dialysis, kidney transplant recipients, lack of adequate baseline creatinine information to determine the stage of AKI, were pregnant, and lacked discharge information were excluded from the research. Those patients who died or left the hospital against medical advice after 24 h of admission were excluded for evaluation of the condition of their kidneys.

Variables

The information collected from this cohort includes demographic characteristics, co-morbidities, cause of underlying CKD, precipitating cause of AKI, KDIGO staging of AKI, vitals and laboratory results at the time of admission and discharge/death, need and modality of RRT, hospital stay, and in-hospital outcomes. The research outcome variable, which is renal recovery, is described as the return of serum creatinine to normal levels of 0.3 mg/dL or 25% of baseline values at the time of discharge without dialysis dependence, as was done in previous researches.^[18,19] Non-recovery subjects include those patients who did not fulfill the above-mentioned conditions at the time of discharge from the hospital.

Bias

Selection bias was minimized by reviewing all consecutive eligible patients as opposed to a sample of these patients. Retrospective information bias resulting from the collection of data was mitigated by use of a well-prepared and pre-tested case record form and data abstraction by two people. For patients whose baseline creatinine was unavailable, the back calculation approach was used as opposed to exclusion to avoid selection of severe or well-documented cases.

Study Size

Sample size calculation for the study was not done formally due to its exploratory nature, and all patients meeting the inclusion criteria during the 1-year study period were included, which led to a total number of 312 patients.

Statistical Methods

For continuous variables, normality was assessed through the Shapiro-Wilk test, and they were reported as mean $\pm$ standard deviation or median (interquartile range). Comparisons among groups were done using the t-test or Mann-Whitney U-test. For categorical variables, they were reported as counts (%), and comparisons among groups were done using Chi-square or Fisher's exact test. All variables with $P < 0.10$ in univariable analysis

were selected to be entered into multivariable logistic regression, where results were expressed as adjusted odds ratios with 95% confidence intervals (CIs). Model performance was measured through the Hosmer–Lemeshow test and receiver operating characteristic-area under the curve (ROC-AUC).

Ethical Considerations

The study was approved by the Institutional Ethics Committee before the start of the study and consent from the subjects was not required as the data analysis was retrospective and de-identified. Confidentiality of patient data was ensured by anonymizing all patient data at the time of data extraction.

RESULTS

Study Population

From the medical records and the nephrology admission list, there were 398 patients with an initial diagnosis of AKI-on-CKD throughout the study period. With the use of the exclusion criteria, 312 patients were eligible for inclusion in the analysis. Common reasons for patient exclusion include patients receiving maintenance dialysis before admission, insufficient baseline serum creatinine or discharge documentation, death or discharge against medical advice within 24 h, kidney transplantations, and pregnant patients. Patients were categorized by their renal function recovery at the time of discharge. Patients who survive and do not fulfill the criteria of renal recovery at discharge and dead patients during the stay in the hospital are considered to be non-recovery. Hence, the final group had patients with renal recovery or non-recovery at discharge.

Baseline Characteristics

Table 1 presents the baseline characteristics of the study participants. Compared to the non-recovery group, patients with renal recovery were younger. In addition, the patients who had renal recovery had better baseline renal function as evident by their higher eGFR before admission and fewer cases of advanced CKD (stage 4 and 5).

In the overall population, diabetes mellitus and hypertension were the most prevalent comorbidities but were not significantly different depending on whether they were in the renal recovery or non-recovery groups. On the other hand, an increased number of comorbidities was significantly associated with the non-recovery patients using the Charlson comorbidity index. There was no

statistically significant difference in the time of diagnosis of CKD between the two groups. Thus, poorer baseline renal function and an increased number of comorbidities were more related to failure of renal recovery than metabolic and cardiovascular comorbidities.

Etiology of underlying CKD and precipitating AKI

The causes of CKD and those of AKI are shown in Table 2 and Figures 1 and 2. Diabetic nephropathy was the most common form of underlying kidney disorder, followed by nephrosclerosis and chronic glomerulonephritis. Other forms of CKD were less commonly seen including chronic interstitial nephritis, obstructive/vesico-ureteric reflux nephropathy, autosomal dominant polycystic kidney disease, and CKD of unknown etiology.

Among the acute causes, sepsis or infection was the most common cause of AKI followed by volume depletion and pre-renal azotemia, as well as nephrotoxic drugs. The rest of the causes were cardiorenal syndrome, obstructive uropathy, and contrast-induced kidney injury.

The incidence of the severity of AKI was significantly different depending on whether recovery had occurred. While all three KDIGO stages were present among the population studied, individuals with severe AKI were more commonly found in the non-recovery group than in any other. In other words, stage 3 AKI occurred more often among those who did not have a successful recovery, while mild stages were more common among those who experienced recovery [Figure 3].

Laboratory Findings

Laboratory measurements on admission are provided in Table 3. In comparison with patients who achieved renal function recovery, patients in the non-recovery group showed signs of more severe impairment of renal function and greater systemic and metabolic disturbances.

Peak creatinine level on admission was significantly higher in the non-recovery group in addition to a higher level of baseline creatinine. Peak blood urea nitrogen level was also higher in this group of patients. On the other hand, the concentration of albumin and hemoglobin in the blood of patients in the non-recovery group was lower, which indicates the presence of greater nutritional and systemic disease burden.

Some significant differences could be noticed in terms of metabolic disorders markers. Non-recovery patients had higher levels of serum potassium and lower levels of bicarbonate in their blood. The level of C-reactive protein was also higher in

Table 1: Baseline demographic and clinical characteristics stratified by renal recovery status

Variable	Overall (n=312)	Recovery (n=181)	Non-recovery (n=131)	P-value
Age, years, mean±SD	58.4±13.2	56.9±12.8	60.5±13.5	0.014
Male sex, n (%)	196 (62.8)	118 (65.2)	78 (59.5)	0.29
Diabetes mellitus, n (%)	168 (53.8)	93 (51.4)	75 (57.3)	0.30
Hypertension, n (%)	245 (78.5)	139 (76.8)	106 (80.9)	0.38
Coronary artery disease, n (%)	74 (23.7)	37 (20.4)	37 (28.2)	0.10
Prior stroke, n (%)	28 (9.0)	13 (7.2)	15 (11.5)	0.19
Baseline CKD stage 4–5, n (%)	162 (51.9)	81 (44.7)	81 (61.8)	0.002
Baseline eGFR, mL/min/1.73 m ² , mean±SD	28.4±11.6	31.2±12.0	24.6±9.8	<0.001
Charlson comorbidity index≥4, n (%)	146 (46.8)	70 (38.7)	76 (58.0)	<0.001
Duration of known CKD, years, median (IQR)	3.0 (1.5–5.5)	2.8 (1.4–5.0)	3.5 (1.8–6.2)	0.06

CKD: Chronic kidney disease, IQR: Interquartile range, eGFR: Estimated glomerular filtration rate, SD: Standard deviation

Table 2: Etiology of underlying CKD and precipitating AKI

CKD etiology	n (%)	AKI etiology	n (%)
Diabetic nephropathy	132 (42.3)	Sepsis/infection	118 (37.8)
Hypertensive nephrosclerosis	78 (25.0)	Pre-renal/volume depletion	66 (21.2)
Chronic glomerulonephritis	42 (13.5)	Nephrotoxic drug exposure	46 (14.7)
Chronic interstitial nephritis	16 (5.1)	Cardiorenal syndrome	26 (8.3)
Obstructive/reflux nephropathy	18 (5.8)	Obstructive uropathy	24 (7.7)
ADPKD	12 (3.8)	Contrast-associated	20 (6.4)
Undetermined etiology	14 (4.5)	Other (NSAID, herbal remedies)	12 (3.8)
Total	312 (100)	Total	312 (100)

AKI: Acute kidney injury, CKD: Chronic kidney disease, ADPKD: Autosomal dominant polycystic kidney disease

Table 3: Laboratory parameters at admission stratified by renal recovery status

Parameter	Recovery (n=181)	Non-recovery (n=131)	P-value
Hemoglobin, g/dL, mean±SD	9.8±1.7	9.1±1.8	0.001
Baseline serum creatinine, mg/dL, mean±SD	1.9±0.6	2.4±0.8	<0.001
Admission serum creatinine, mg/dL, median (IQR)	3.9 (2.9–5.2)	4.8 (3.4–6.6)	<0.001
Peak serum creatinine, mg/dL, mean±SD	4.9±1.9	6.4±2.4	<0.001
Blood urea nitrogen, mg/dL, mean±SD	68.4±24.1	84.2±28.6	<0.001
Serum albumin, g/dL, mean±SD	3.3±0.5	2.9±0.6	<0.001
Serum potassium, mEq/L, mean±SD	4.6±0.8	5.1±1.0	<0.001
Serum bicarbonate, mEq/L, mean±SD	19.8±3.4	16.9±3.8	<0.001
C-reactive protein, mg/L, median (IQR)	24 (10–52)	58 (24–110)	<0.001

IQR: Interquartile range, SD: Standard deviation

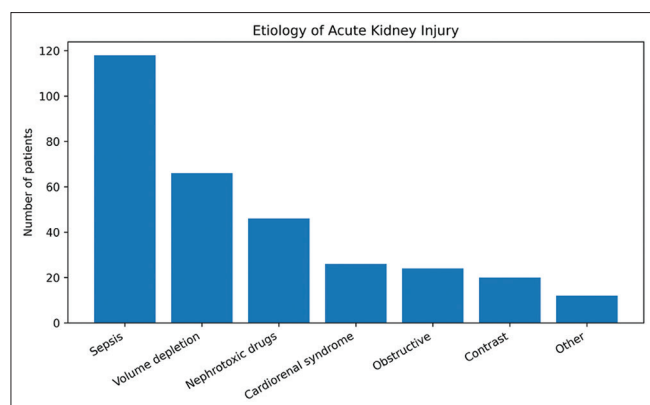


Figure 1: Distribution of underlying chronic kidney disease etiology among the study cohort (n = 312), expressed as percentage of total patients

non-recovery patients, which suggests greater inflammatory activity.

RRT, Hospital Course, and Outcomes

RRT, course during hospitalization, and outcomes during hospital stay are described in Table 4. RRT was needed in a large percentage of the cohort, and its frequency was higher significantly among subjects who did not recover from renal failure. In those patients who were on RRT, a significant number recovered sufficiently to become dialysis-free and satisfy renal recovery criteria on discharge.

The kind of RRT used was similar in both the recovered and unrecovered subjects, with intermittent hemodialysis being the most common. Therefore, need for RRT appears to be more relevant to renal outcome than kind of therapy.

The patients in the non-recovery group also experienced a more complex hospital stay. They needed more organ

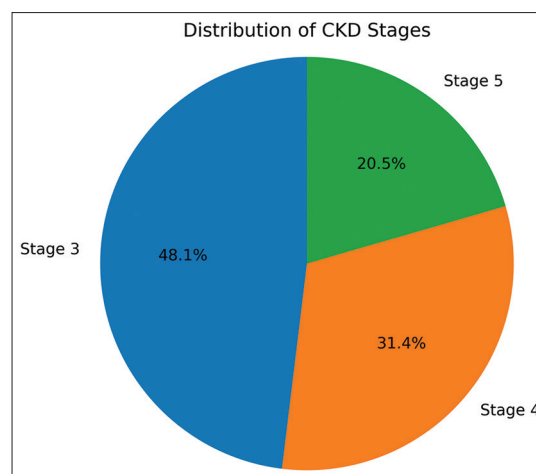


Figure 2: Distribution of precipitating acute kidney injury etiology among the study cohort (n = 312), expressed as percentage of total patients

support and stayed longer in the hospital compared to those in the recovery group. Mortality in the hospital occurred only among the patients in the non-recovery group, which speaks to the significant severity of renal recovery failure. It can be stated, therefore, that renal recovery failure not only correlated with significant renal impairment but also correlated with the increased need for organ support, more critical illness, and prolonged hospitalization.

Multivariable Predictors of Renal Recovery

Variables that were statistically significant on univariable analysis in relation to recovery from renal failure at the predetermined level of statistical significance were selected for multivariable logistic regression. Variables included demographic variables, baseline renal function, severity and etiology of AKI, burden of

comorbidities, need for RRT, serum albumin levels, and peak creatinine.

Despite adjustment for other possible confounders, several independent risk factors emerged in relation to recovery from renal failure [Table 5]. Baseline kidney function was found to be positively associated with the possibility of renal recovery; however, severe AKI, sepsis-related AKI, need for RRT, hypoalbuminemia, and high burden of comorbidities negatively predicted renal recovery.

The relationship between baseline eGFR and recovery persisted in the adjusted models, reflecting the role of existing renal reserve in determining the likelihood of recovery from AKI-on-CKD. In contrast, the presence of KDIGO stage 3 AKI on admission was significantly associated with much reduced chances of recovery relative to milder degrees of AKI.

AKI resulting from sepsis was an independent predictor of lower likelihood of recovery, demonstrating the negative impact of systemic infection and inflammation on renal outcomes. In addition, patients needing RRT had lower odds of renal recovery;

however, it is important to interpret this finding as a sign of disease severity rather than causative factor of dialysis *per se*.

In the absence of recovery, hypoalbuminemia continued to be an independent predictor while increased Charlson comorbidity index was associated with decreased likelihood of recovery, indicating the role of nutrition status and comorbidity. Although age had an inverse relationship with recovery, it failed to attain statistical significance.

Finally, the multivariate model proved its calibration to be adequate as tested by Hosmer–Lemeshow goodness-of-fit test and performed well on ROC analysis, suggesting the ability of the model to separate between patients who recovered and who did not recover [Table 5 and Figure 4]. Hosmer–Lemeshow goodness-of-fit: $\chi^2 = 6.12$, $P = 0.63$; ROC AUC = 0.79 (95% CI 0.74–0.84).

DISCUSSION

In this retrospective cohort study involving 312 cases of AKI on top of CKD, there was renal recovery in slightly more than half of patients at the point of discharge, while in-hospital mortality reached nearly one out of six cases. The factors associated with lack of renal recovery were increased AKI severity, sepsis as the cause of AKI, need for RRT, hypoalbuminemia, and a higher prevalence of co-morbid conditions, while an increased baseline eGFR acted as a protection factor. The results are generally in line with, and extend, the current body of literature on AKI on top of CKD, but provide information about South Asia specifically.

Our observed recovery rate in this patient population is slightly below the range of 60–70% reported in some Western studies of AKI on CKD, although this is consistent with data from Indian and other South Asian centers, where late presentation, greater occurrence of infection-related AKI, and the lack of pre-admission outpatient follow-up by nephrologists may all play a role in poorer renal outcomes.^[2,7,16] The relationship between a lower eGFR baseline level and hence greater CKD severity stage at baseline with decreased odds of renal recovery is consistent with the idea of reduced functional capacity of the kidneys, in which the smaller number of functioning nephrons means that the remaining

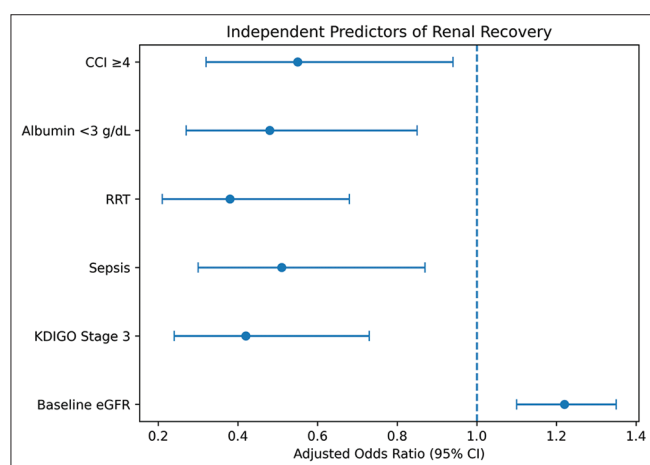


Figure 3: Distribution of KDIGO acute kidney injury (AKI) stage at presentation stratified by renal recovery status, illustrating the higher proportion of stage 3 AKI in the non-recovery group

Table 4: RRT, hospital course, and in-hospital outcomes

Variable	Overall (n=312)	Recovery (n=181)	Non-recovery (n=131)	P-value
RRT required, n (%)	128 (41.0)	58 (32.0)	70 (53.4)	<0.001
Intermittent hemodialysis, n (%)	96 (75.0 of RRT)	46 (79.3)	50 (71.4)	0.31
CRRT/SLED, n (%)	32 (25.0 of RRT)	12 (20.7)	20 (28.6)	0.31
ICU admission, n (%)	134 (43.0)	55 (30.4)	80 (61.1)*	<0.001
Length of stay, days, median (IQR)	9 (6–14)	8 (6–11)	12 (8–18)	<0.001
In-hospital mortality, n (%)	54 (17.3)	0 (0)	54 (41.2)	—

*Non-recovery ICU admissions calculated among 131 patients (77 survivors without recovery+54 deaths). IUC: Intensive care unit, RRT: Renal replacement therapy, IQR: Interquartile range, CRRT: Continuous renal replacement therapy, SLED: Sustained low-efficiency dialysis

Table 5: Multivariable logistic regression analysis of independent predictors of renal recovery

Variable	Adjusted OR	95% CI	P-value
Baseline eGFR (per 5 mL/min/1.73 m ² increase)	1.22	1.10–1.35	<0.001
KDIGO AKI stage 3 (vs. stage 1–2)	0.42	0.24–0.73	0.002
Sepsis-associated AKI	0.51	0.30–0.87	0.014
Requirement for RRT	0.38	0.21–0.68	0.001
Serum albumin <3.0 g/dL	0.48	0.27–0.85	0.012
Charlson comorbidity index ≥ 4	0.55	0.32–0.94	0.029
Age (per year)	0.98	0.96–1.00	0.06

eGFR: Estimated glomerular filtration rate, RRT: Renal replacement therapy, AKI: Acute kidney injury, OR: Odds ratio, CI: Confidence interval

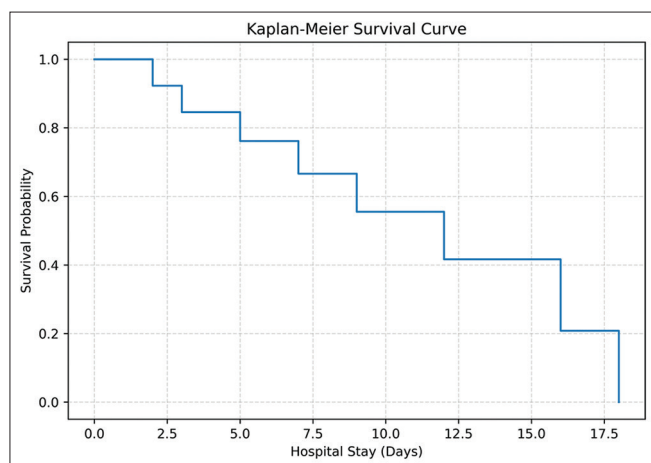


Figure 4: Receiver operating characteristic curve for the multivariable logistic regression model predicting renal recovery (area under the curve 0.79, 95% confidence interval 0.74–0.84)

margin for tolerance to further damage before reaching a level of irreversible loss of function is smaller, as discussed previously in relation to baseline renal function and AKI outcomes.^[3,5,17]

Sepsis not only formed the commonest cause for AKI in our study but also emerged as an independent predictor of failure to recover, and this reflects the findings in several large multinational studies where septic AKI has been shown to have poor renal and overall prognosis as compared to AKI due to other causes.^[8,18,19] The reasons behind this phenomenon are multi-factorial, with impaired microcirculation, inflammation and hemodynamic instability playing their role on top of the already existing renal dysfunction due to underlying chronic diseases rather than any specific unifying pathogenesis.^[4,9,20] This higher prevalence of sepsis-associated AKI in our patients as compared to some of the Western series is reflective of the higher prevalence of infections in tertiary care centers in the Indian subcontinent.^[12,21,22]

The link between higher AKI stage based on KDIGO criteria and decreased risk of renal recovery is well known and proven to be true in our multivariate analysis, when stage 3 AKI had significantly lower probability of recovery compared with less severe stages.^[16,18] In the same way, need for RRT was an independent predictor of non-recovery; however, it is worth mentioning that almost half of patients requiring RRT during their hospital stay recovered and became dialysis-free by the time of discharge from the ICU, meaning that need for RRT in this patient group cannot be considered the indicator of end-stage kidney disease and should be re-assessed.^[17,19]

Hypoalbuminemia on admission was independently associated with decreased odds of renal recovery in our study population; this association has been found in other cohorts of AKI patients and is believed to reflect not only a causal effect but also the interplay between inflammation, nutritional depletion, and changes in fluid distribution.^[6,23] Similarly, increased Charlson comorbidity index was associated with poor renal outcome, as in many other studies showing the link between the number of comorbidities and AKI severity/recovery, indicating that the outcome of AKI-on-CKD depends on general disease burden as well as on kidney dysfunction *per se*.^[10,24]

Age was significantly related to non-recovery in univariate analysis, but it was no longer significant when adjusted for other covariates, implying that the influence of age on non-recovery in this study group was largely mediated by its association with comorbidity burden and baseline kidney function, rather than being a biological effect of age, which has been seen before in other multivariate studies of AKI recovery.^[11,25]

A number of important limitations are worth considering. First, the nature of the retrospective, single-center design itself implies limited generalizability and creates the risk of potential uncontrolled confounding and information bias associated with the use of existing medical charts. Second, baseline serum creatinine concentration was missing for some of the screened patients and was calculated for others using the back-calculation formula – although it is a commonly used tool, it might have resulted in misclassification of AKI stage, both in one or another direction. Furthermore, due to the incomplete hourly records, urine output criteria could not be used; thus, there might have been some patients who met the criteria for AKI based on urine output and were not included in the analysis due to this reason. Third, only hospital discharge was considered as the time point at which renal recovery was defined, whereas long-term follow-up data needed to confirm if the patients classified as non-recovered eventually recovered would not have been accessible.

Despite these constraints, this study contributes valuable local information pertaining to the clinical features and determinants of recovery from AKI-on-CKD from a tertiary care center from India, a region that has considerably less available evidence than developed country cohorts. The availability of clinically and laboratory-based predictors of recovery, such as AKI severity, sepsis, requirement of RRT, serum albumin level, and burden of co-morbidities, can help clinicians in stratification of the risk of their patients on admission as well as set realistic expectations for renal outcomes. A prospective multicenter study with follow-up after discharge as well as new tubular damage biomarker, if possible, will validate these results.

CONCLUSION

Among this group of patients with AKI occurring in the context of CKD who were managed in a tertiary-care teaching hospital in India, renal recovery occurred in slightly more than 50% of patients, whereas in-hospital mortality rate was considerable. Decreased baseline eGFR, increased AKI severity, sepsis as the inciting cause, need for RRT, hypoalbuminemia, and increased morbidity load independently predicted lower chances of renal recovery. These results highlight the importance of early detection of AKI among patients with underlying CKD, prudent avoidance of nephrotoxic and septic hits when feasible, and careful prognostication of renal recovery in such patients.

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