Clinical Features, Renal recovery and Predictors of Acute Kidney Injury in a Resource-Limited Country: A Prospective Study

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Abstract

Background

Recovery from acute kidney injury (AKI) in hospitalized patients is variable and persistent impairment of renal function at discharge is associated with long-term adverse outcomes. Understanding the risk factors for non-recovery AKI may help to avoid modifiable determinants and enhance recovery. However, data on renal recovery and its associated factors is limited, especially in resource-limited settings. This study aimed to assess the clinical features, renal recovery, and predictors of AKI among hospitalized patients in Ethiopia.

Methods

A prospective observational study was conducted involving all adult patients with age ≥18 years, and met the kidney disease: improving global outcomes criteria for AKI from April to July 2019. The main outcome variable was renal recovery at discharge. The logistic regression model was used to determine predictors of non-recovery from AKI. Statistical significance was considered at a p-value of less than 0.05 on multivariate analysis.

Results

Of the 169 patients included in the study over four months, 127(75.2%) were < 60 years, 121 (71.6%) had kidney disease on admission, almost one-third (33.1%) of them had stage 3 AKI, nearly half (50.29%) had prerenal AKI, and 32(19%) had exposure to nephrotoxins. Vomiting 31(18.34%), oliguria 42(24%), hematuria 15(25.4%), and proteinuria 26(40.6%) were common presenting features. Of the total study participants, most of them (68%) had complete renal recovery and fifty-four (32%) patients had non-recovery AKI at the time of hospital discharge. On multivariable analysis, proteinuria (AOR 6.2, CI 1.25-31.4, p=0.002), AKI stage III (AOR 4.7, CI 1.37-28.6, p=0.019), and nephrotoxin exposure (AOR 5.2, CI 2.1-14.89, p=0.007) were factors significantly associated with non-renal recovery.

Conclusions

Our study found that most of the patients had AKI on admission and one-third had a higher stage of AKI. A higher proportion of patients were found to have non-recovery AKI at hospital discharge. Renal non-recovery was significantly associated with the severity of AKI, nephrotoxic drug use, and proteinuria. Follow-up of serum creatinine and proteinuria, and careful drug use monitoring may help to identify patients with poor prognosis, initiate specific interventions, and improve renal recovery.

Introduction

Acute kidney injury (AKI) is a major complication of hospitalized patients and a predictor of poor short-and long-term outcomes. It occurred in over 1 of 5 adult hospitalizations, with pooled worldwide incidence rates

of 21.6% (1, 2, 3). About 13.3 million people are shown to develop AKI per year, 85% of them live in resource-limited countries (4).

Acute kidney injury is associated with a higher in-hospital to 6-month mortality, with a mortality rate ranging from 20–60% (5, 6). Moreover, the condition is associated with other adverse short-and long-term renal and non-renal outcomes including systemic complications, recurrent disease, progression to chronic kidney disease (CKD), end-stage renal disease (ESRD), cardiovascular events, length of hospital stay, hospital readmission, and cost to health care. Regardless of its severity, AKI is associated with a greater risk of morbidity and mortality (7, 8, 9, 10, 11).

The degree of recovery from subsequent episodes of AKI is shown to substantially affect these poor short- and long-term outcomes. Furthermore, age, hypertension, diabetes, preexisting CKD, and proteinuria are some of the factors found to be associated with a low rate of renal recovery. Recovery from AKI is a potentially modifiable event and measures taken to prevent the modifiable determinants can increase the probability of renal recovery (12, 13).

Understanding the risk factors for non-recovery after AKI may help for early initiation of specific interventions to enhance recovery, determine the duration of follow-up, and improve outcomes of AKI (13). However, despite persistent AKI at discharge has been known to be associated with poor long-term outcomes, data on renal recovery in surviving patients is sparse, especially in resource-limited countries. Therefore, this study aimed to assess the clinical features, renal recovery, and predictors of acute kidney injury among hospitalized patients admitted to a teaching hospital in Ethiopia.

Material And Methods

Study setting and period

This study was done parallel with other studies (14) from April 1 to July 30, 2019, at the medical ward of Jimma medical center, Southwest Ethiopia. Jimma medical center is the only teaching and referral hospital in the southwestern part of Ethiopia with an 800-bed capacity. Currently, it provides services for approximately 15,000 inpatient, 160,000 outpatient attendants, with a catchment population of around 15 million people. The internal medicine department of the hospital has two renal units and provides nephrologist-guided care to the patients.

Study design and Population

A four-month prospective observational study was conducted. All adult patients with age ≥18 years, and who met the kidney disease: Improving Global Outcomes (KDIGO) criteria for AKI were recruited consecutively. All patients who gave written informed consent were included in the study. Patients with underlying CKD, unable to give the required information, hospitalized for less than 48 hours, died, self-discharged, and referred to other institutions were excluded from the study.

Definition of AKI and outcomes

Definition of AKI and its severity was according to the KDIGO 2012 AKI criteria (1). Community-acquired AKI is AKI that is detected at admission, or within 48 hours of hospital admission. We used the serum creatinine obtained within seven days to three months of hospital admission, if unavailable, or the minimum and or most recent value of serum creatinine at the time of hospital admission was used as the baseline for community-acquired AKI(15,16,17). For patients with hospital-acquired AKI, the first documented serum creatinine on admission was used as the baseline(1). The discharge creatinine was the last creatinine measured as an inpatient. The duration of anuria was defined as the time in which urine output was less than 0.5 mL/kg/hr

The main outcome variable was renal recovery at hospital discharge. Recovery was defined as reversal at discharge or return to baseline kidney function. Reversal from the AKI episode was defined as no longer meeting AKI criteria. The pattern of renal recovery was defined as (I) early sustained reversal: recovery from AKI within two days, (II) late sustained reversal: recovery after two days and sustained through hospital discharge, (III) relapsing AKI with complete recovery, (IV) relapsing AKI without complete recovery, and (V) never recovery(18,19,20,21). The first three were considered as complete recovery and the last two as non-recovery AKI, defined as AKI not returned to baseline kidney function at discharge.

Data collection and statistical analysis

Data was collected by trained health professionals using a data abstraction checklist. Clinical and demographic characteristics including age, gender, clinical presentations, diagnosis, underlying comorbidities, laboratory results, and medications were collected from the patient's medical records prospectively. Serum creatinine and urine output results were reviewed daily until the patient's discharge from the hospital. Data was entered to Epi data version 4.4.2 and exported to SPSS version 26.0 for analysis. The data were presented as mean±(SD) and median(IQR, interquartile range) for continuous variables and frequency and proportions for categorical variables. The Chi-square test was used for the comparison of the proportions of categorical variables. The logistic regression model was used to determine predictors of non-recovery AKI at hospital discharge. Variables with a P value of less than 0.25 from the univariate analysis were included for multivariate analysis. Statistical significance was considered at a p-value of less than 0.05 on multivariate analysis.

Results

Baseline clinical characteristics of patients

The study included 169 AKI patients admitted to the internal medicine ward during the study period and fulfill the inclusion criteria. The mean($\pm$SD) age was 49.91$\pm$15.17(range 18-80) years and the majority of the patients (58.6%) were men. Of the patients, most of them (71.6%) had AKI on admission and almost one-third (33.1%) had KIDGO stage 3 AKI. The commonest clinical presentations among the study participants were vomiting 31(18.34%), anuria 11(6.5%), oliguria 42(24%), hematuria 15(25.4%), and proteinuria 26(40.6%). Concerning the cause of AKI, more than half of them (53%) had prerenal AKI, 28(17%) had intrinsic AKI, and 15(9%) had postrenal AKI. Hypertension (44.4%), heart failure (44.4%), and anemia (7.1%) were common underlying comorbidities identified. Of the patients, 32(19%) patients had exposure to

nephrotoxins, among these, non-steroidal anti-inflammatory drugs (NSAIDs) and Angiotensin-converting enzyme inhibitors (ACEIs) comprise the higher frequency with 17(10%), and 9(5.3%), respectively. Vancomycin 4(2.4%) and gentamycin 2 (1.2%) were the other nephrotoxins prescribed in AKI patients (**Table 1)**.

Laboratory values of patients

Urinalysis was done for 64 patients. Of this, 26(40.6%) had proteinuria and 15(25.4%) had hematuria. On the other hand, urine output was assessed for 103(60.94%) patients,11(10.7%) had anuria and 42(40.8%) had oliguria. Close to two-thirds (66.3%) of the patients had serum electrolyte assessment. Of this, 28(25%) had hyperkalemia, 32(28.6%) had hyponatremia, and 15(13.4%) had Hypocalcemia **(Table 2)**.

Table 1: Baseline clinical characteristics of AKI patients by renal recovery

Variables	Category	Total n (%)	Recovery (115)	Non-recovery (54)
Age	<60	127(75.2%)	92(54.4%)	35(20.8%)
	≥60	42(24.8%)	23(13.6%)	19(11.2%)
Sex	Male	99(58.6%)	66(39%)	33(19.5%)
	Female	70(41.4%)	49(29%)	21(12.5%)
Stage of AKI	Stage 1	73(43.2%)	61(36.1%)	12(7.1%)
	Stage 2	40(23.7%)	29(17.2%)	11(6.5%)
	Stage 3	56(33.1%)	25(14.8%)	31(18.3%)
Hypertension	Yes	75(44.4%)	42(24.8%)	33(19.6%)
	No	94(55.6%)	73(43.2%)	21(12.4%)
Diabetes	Yes	6(3.6%)	2(1.2%)	4(2.4%)
	No	163(96.4%)	113(66.8%)	50(29.6%)
Heart failure	Yes	75(44.4%)	44(26%)	31(18.4%)
	No	94(55.6%)	71(42%)	23(13.6%)
Anemia	Yes	12(7.1%)	10(5.9%)	2(1.2%)
	No	157(92.9%)	105(62.1%)	52(30.8%)
Nephrotoxins	Yes	32(19%)	15(9%)	17(10%)
	No	137(81%)	100(59%)	37(22%)
Serum potassium	Hyperkalemia	28(25%)	10(8.9%)	18(16.1%)
	Hypokalemia	11(9.8%)	7(6.3%)	4(3.5%)
Serum sodium	Hyponatremia	32(28.6%)	19(17%)	13(11.6%)
Serum calcium	Hypocalcemia	15(13.4%)	9(8%)	6(5.4%)
Chloride	Hyperchloremia	11(9.8%)	5(4.5%)	6(5.3%)
Urine output	Anuria	11(10.7%)	4(3.9%)	7(6.8%)
	Oliguria	42(40.8%)	27(26.21%)	15(14.56%)
Urinalysis	Proteinuria	26 (40.6%)	14(21.8%)	12(18.8%)
	Hematuria	15(25.4%)	4(6.8%)	11(18.6%)

Abbreviations: AKI, acute kidney injury,

Table 2: Laboratory values of AKI patients by renal recovery

Variables	Category	Recovery	Non-recovery
Baseline Scr	Mean ± SD (min, max)	1.14±0.40 (0.75, 2.44)	2.47± 1.02 (0.70, 4.76)
Admission Scr	Mean ± SD (min, max)	4.45±3.07(2.21, 13.80)	7.25±4.95 (1.97, 20.10)
Discharge Scr	Mean ± SD (min, max)	3.22± 2.61(0.91, 5.52)	5.84±3.85 (1.6, 15.10)
Admission BUN	Mean ± SD (min, max)	96.21±74.36 (23.5, 375)	123.29 ±85.53(40, 375)
Discharge BUN	Mean ± SD (min, max)	86.07±70.05(17.25, 525)	93.30±80.23 (32.10, 465)
Serum sodium	Mean ± SD (min, max)	135.02± 3.3(128,141)	133.17±9.37(119, 144)
Serum potassium	Mean± SD (min, max)	4.32±1.91(1.89,6.5)	4.80±2.45 (2.50, 7.10)
Calcium (ionized)	Mean ± SD (min, max)	1.06±0.16(0.67,1.33)	1.05 ±0.26 (0.60, 1.28)
Chloride	Mean ± SD (min, max)	107.76± 10.61(94,138)	111.55±9.95 (106, 140)
Duration of anuria (days)	Mean ± SD (min, max)	2.81± 0.72(2,6)	4.26±1.71(3, 11)
Duration of oliguria (days)	Mean ± SD (min, max)	1.94±0.89(1,5)	2.44±1.10(2,4)
Hemoglobin	Mean ± SD (min, max)	10.12 ±3.1(6.01, 13.5)	10.01±3.2 (5.5, 14.2)
WBC	Mean ± SD (min, max)	9.7± 5.4 (3.6, 18.00)	8.95 ± 3.40(4.5,16.0)
RBC	Mean ± SD (min, max)	3.69±1.03(2.55, 5.80)	3.68±1.05 (2.01, 5.32)
Platelet	Mean ± SD (min, max)	261.6± 98.40(126,462)	241.2±103.3 (139, 369)

Abbreviations: Scr; Serum creatinine, BUN; Blood urea nitrogen, Min; Minimum, Max; Maximum

Outcomes: Patterns of renal recovery

Among the study participants, more than two-thirds (68%) of the patients had complete renal recovery at hospital discharge. From this, 17(10%) had early sustained reversal, 78(46%) had a late sustained reversal, the other 20(12%) had relapsing AKI with complete recovery at discharge. Recovery was more likely in stage 1(36.1%) and stage 2 (17.2%) than in stage 3 AKI (14.8%). In addition, the median time from AKI diagnosis to complete renal recovery was increased with the stage of AKI. Patients with stage I had 3 days (IQR 2–7) while patients with stage II had 7 days (IQR 4–11), and patients with stage III had 12 days (IQR 10–22). Fifty-four (32%) patients had non-recovery AKI at discharge. From this, 23 (13.6%) patients had relapsing AKI

without recovery and 31(18.4%) patients never recovered during their hospital stay. The rate of non-recovery was proportional to the severity of AKI, as a higher rate of non-recovery was observed in stage 3 AKI (18.3%) than in stage II (6.5%), and stage I (7.1%) AKI (**Figure 1**). Moreover, a higher mean (±SD) baseline, admission, and discharge serum creatinine, and mean(±SD) duration of anuria and oliguria were observed in patients with non-recovery AKI. The mean(±SD) discharge serum creatinine was 5.97(±3.86) mg/dl which is higher than 3.22(±2.61) mg/dl in patients with complete recovery. The mean (±SD) duration of anuria 4.26±1.71 days (range 3-11 days) and oliguria 2.44±1.10 days (range 2-4days) were also higher in patients with non-recovery AKI (**Table 2**).

Predictors of renal non-recovery

Predictors associated with incomplete renal recovery are outlined in **Table 3**. On univariate logistic regression analysis, non-recovery was significantly associated with age ≥ 60 years, proteinuria, hypertension, heart failure, AKI stage II, and stage III, and exposure to nephrotoxins. On multivariable logistic regression analysis, patients with proteinuria had more than six times higher risks of renal non-recovery compared to patients without proteinuria (AOR 6.2, CI 1.25-31.4, p=0.002). Patients with AKI stage III disease had 4.7 times higher risks of renal non-recovery than patients with a less severe stage of AKI (AOR 4.7, CI 1.37-28.6, p=0.019), and patients with nephrotoxin exposure had more than 5 times increased risks for renal non-recovery than their counterparts (AOR 5.2, CI 2.1-14.89, p=0.007).

Table 3: Univariable and multivariable analysis of predictors of non-recovery AKI

ariables	Category	Outcome		OR (95%CI)	P-value	AOR (95%CI)	P-value
		Recovery	Non-recovery				
		115	54				
ge in years	< 60	92	35	Ref			
	≥ 60	23	19	2.1(1.05,4.46)	0.035	2.1(0.49,9.13)	0.315
roteinuria	Absent	30	8	ref			
	Present	14	12	3.2(1.22,11.4)	0.020	6.2(1.25,31.4)	0.002*
ypertension	No	73	21	Ref			
	Yes	42	33	2.7(1.4,5.31)	0.003	5.6(0.84,37.69)	0.074
eart failure	No	71	23	Ref			
	Yes	44	31	2.17(1.12,4.1)	0.021	3.7(0.69,19.7)	0.124
tage of AKI	Stage 1	61	12	Ref			
	Stage 2	29	11	3.2(1.36,7.86)	0.008	3.1(0.59, 15.2)	0.182
	Stage 3	25	31	6.3(2.79,14.2)	0.000	4.7(1.37, 28.6)	0.019*
ephrotoxins	No	100	37	Ref			
	Yes	15	17	3 (1.39, 6.75)	0.005	5.2(2.1,14.89)	0.007*

Ref= reference, *= stastically significant

Figure 2 shows the cumulative rate of renal recovery by the nephrotoxin exposure status of patients. Patients with nephrotoxic drug exposure had a low rate of renal recovery.

Discussion

Recovery from acute kidney injury (AKI) in hospitalized patients is variable and persistent impairment of renal function at discharge has been associated with increased mortality, resource utilization, progression to CKD, ESRD, and cardiovascular events. Even small changes in kidney function in hospitalized patients are associated with significant changes in short and long-term outcomes (1,22). Therefore, identifying risk factors leading to incomplete recovery after AKI, maximizing recovery, reducing the burden of CKD, and ESKD in the community should be the goal of any AKI prevention and treatment strategies (20,23).

This study, the first in Ethiopia, found a high prevalence (32%) of non-recovery AKI at hospital discharge. The degree of non-recovery was higher than findings from other studies, 15.2% in Cameroon(24), and 20.2% in South Africa(25). The high prevalence of non-recovery observed in this study could be due to the variation

with these studies in criteria used for renal recovery (dialysis independent vs return to baseline Scr), and difference in point in time of evaluation of recovery (at hospital discharge vs post-discharge). Studies defining recovery as independent from dialysis show higher rates of recovery than those defining recovery from AKI as a return to baseline serum creatinine (26). The rate of non-recovery in our study was lower than the results reported from previous studies, 47.3% in southwest Nigeria(27), 52.8% in Malawi(28), 60.7% in Cleveland, Ohio(29), and 61% in South Korea(30). The variation could be due to differences in sample size, length of follow-up, study design, study setting, patient population (surgical, medical, or mixed), and the inclusion/exclusion of patients with CKD. In addition, since our study is limited only to survivors at discharge, the exclusion of patients who died in the hospital may contribute to the low prevalence of non-recovery in this study. However, due to the absence of a uniform definition of renal recovery and the difference in the timing of assessment of recovery, the rate of renal non-recovery is different in studies published in the literature. Therefore, it is difficult to compare findings directly across studies (31,21).

Patients discharged with non-recovery AKI had a more severe stage of the disease and a higher mean discharge creatinine value, and mean duration of anuria and oliguria. Likewise, previous studies revealed that a more severe stage of AKI, a higher mean discharge creatinine, and a mean duration of anuria are common in patients with incomplete recovery (23,28,32,33). Interestingly, the severity of AKI was significantly associated with an increased likelihood of non-renal recovery. This is consistent with previous studies that demonstrated a significant association between the severity of AKI and incomplete recovery (19,29,30,34). Furthermore, declined renal function at hospital discharge and severity of AKI are found to be associated with the progression of AKI to CKD and mortality. Therefore, physicians should identify patients with a more severe stage of the disease and manage accordingly to enhance recovery and prevent these adverse outcomes. It is recommended that the intensity of therapeutic and preventive measures should be performed based on the severity of AKI (1,35).

Many studies found an association between exposure to nephrotoxic drugs and the risk of non-renal recovery after AKI. Prescription of nephrotoxic drugs is usually evaluated semi-quantitatively and thus associated with subsequent AKI and CKD, and worsening of lower severity of disease, morbidity, and mortality (36,37,38). This study found that the use of nephrotoxic drugs was associated with a significantly higher chance of non-renal recovery at discharge. The study highlights the need to minimize patients' exposure to nephrotoxic drugs to avoid persistent injury to the kidney, improve renal recovery and prevent subsequent poor short and long-term outcomes AKI. Measures taken to prevent AKI and protect kidney function, such as avoiding nephrotoxins and drug use monitoring can prevent persistent acute kidney injury(19).

One of the main findings of this study found a significant association between proteinuria with non-recovery AKI. As reported in previous studies preadmission proteinuria before an episode of AKI and in-hospital positive urine dipstick test are independent predictors of non-recovery at discharge(21,32,39). Therefore, close monitoring of proteinuria during the period of hospitalization may help to identify patients with persistent AKI and subsequent progressive kidney disease and initiate specific interventions to improve recovery.

The recovery of kidney function following AKI is shown to be an important determinant of morbidity and mortality. Even after distinct recovery, AKI is shown to be associated with long-term risk for CKD, even for less severe forms. Patients with one episode of mild AKI have significantly lower long-term survival rates than patients with no AKI. As a result, close medical follow-up of these patients is warranted (22,40,41). However, due to the lack of routine renal function tests in many hospitals in the country and periodic follow-up of patients post-discharge (42), the long-term outcomes include mortality, CKD, ESRD, and other adverse outcomes of AKI in our settings are not known.

Our study has some limitations. Firstly, this was a single-center study and exclude patients with underlying CKD. Secondly, urine output assessment was not done for some patients. Moreover, there was no monitoring of the patients after discharge. Therefore, the outcomes of AKI after discharge are not known. Despite these limitations, this is the first prospective observational study of adult medical admissions, which shed some light on the clinical features and recovery status of patients after AKI in resource-limited settings.

Conclusion

Our study found that most of the patients had AKI on admission and one-third had a higher stage of AKI. A higher proportion of patients were discharged with non-recovery AKI. Renal non-recovery was significantly associated with the severity of AKI, nephrotoxic drug use, and proteinuria. Efforts made to identify the clinical and medication use related factors with close monitoring of renal function tests, urinalysis, and careful drug use monitoring may help to identify patients with a poor renal prognosis, initiate specific interventions, and improve renal recovery at hospital discharge.

Abbreviations

ACEIs; Angiotensin-converting enzyme inhibitor(s), AKI Acute kidney injury, BUN; Blood urea nitrogen, CKD; Chronic kidney disease, ESRD; End-stage renal disease, ICU; Intensive care unit, JMC; Jimma Medical Center, KDIGO; Kidney Disease Improving Global Outcomes, NSAIDs; Non-steroidal anti-inflammatory drugs, RRT; Renal replacement therapy, Scr; Serum creatinine, UOP; Urine output.

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Declarations

Ethical approval and consent to participate

The study meets the ethical standards outlined in national and international guidelines and is conducted in accordance with the principles of the Declaration of Helsinki. The study was authorized by the ethical committee of Jimma University. Written informed consent was secured from all participants and collected data was kept confidential.

Consent for Publication

“Not applicable”

Availability of data and materials

All data used for this study will be made available by the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Author Contributions

A.A., K.K., and M.B. contributed to the conception and design of the study and discussion and interpretation of the results. A.B., Y.W., and E.A. contributed to the data collection, analysis, and drafting of the manuscript. All authors revised and approved the final manuscript.

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Figures

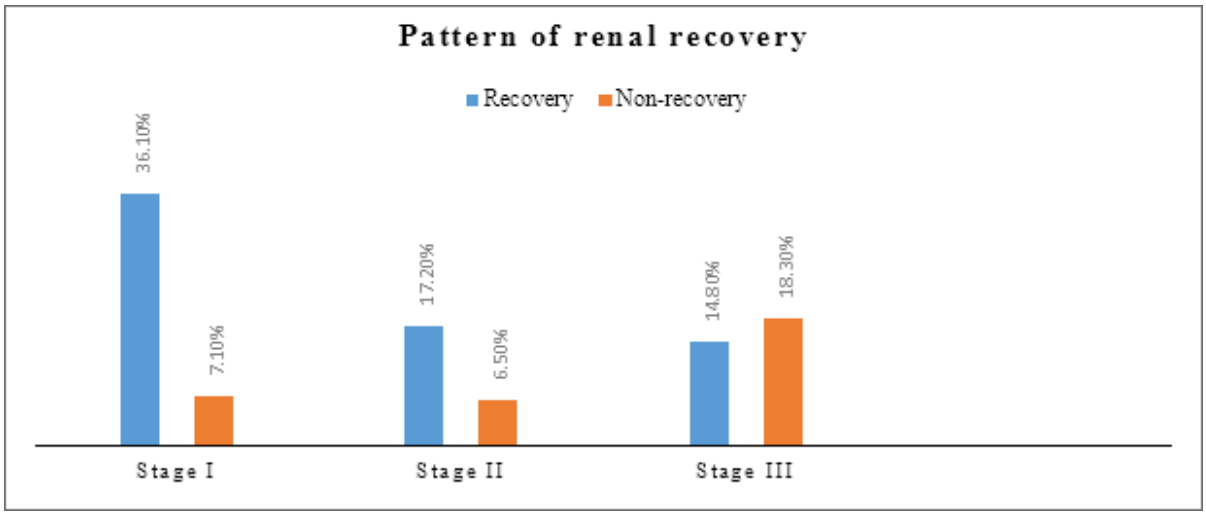

Figure 1

Pattern of renal recovery by stage of acute kidney injury

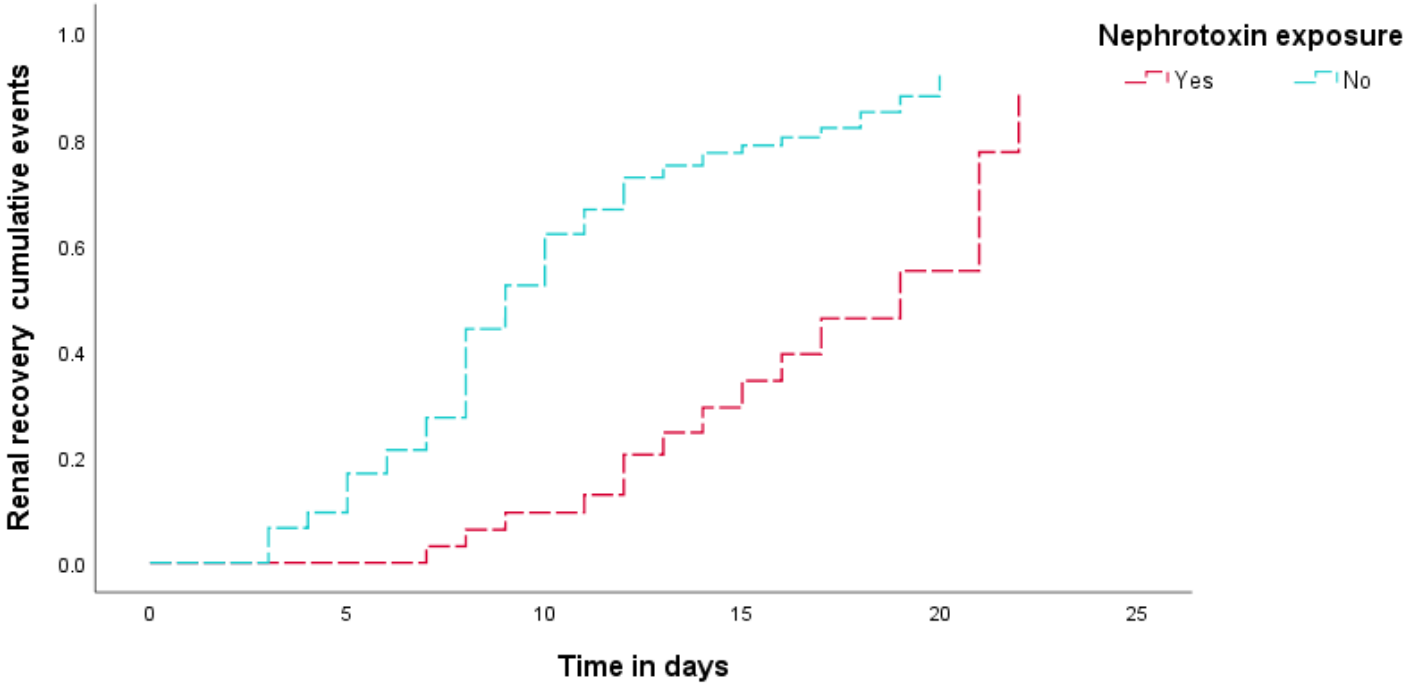

Figure 2

Kaplan-Meier curve of the cumulative renal recovery rate by nephrotoxin exposure