Aetiological factors, outcomes and mortality risk of acute kidney injury in hospitalized patients in a tertiary health centre in Nigeria: An eleven year review

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Abstract

Acute kidney injury is a major public health issue in Nigeria, and it is associated with an increase in mortality. The study's goal was to look at the most common precipitating factors, outcomes, and risk factors associated with mortality in our patients. This study examined the medical records of 11 years, of of hospitalized adult patients with confirmed Acute Kidney Injury (AKI). The patient record was searched for relevant information. The Kidney Disease Improving Global Outcomes (KDIGO) serum creatinine criteria were used to define AKI. The logistic regression analysis was used to determine the risk factors associated with mortality. A total of 399 patients were analysed. The overall mean age was 45.0 ± 17.3. The older age group (≥ 60years) compared to the younger group (<60 years) developed hospital acquired AKI (10% vs 5%). Pre-existing diseases like, hypertension (p<0.001), diabetes mellitus (p<0.001), anemia (p<0.001), stroke (p<0.001) and malignancy (p<0.001) were significantly higher in the older group. More of the older age group had more than 1 comorbidity (66 *vs*. 48%), were on diuretic and ACEI. The commonest causes/precipitants of AKI were septicaemia and other infections (62%) and hypovolaemia/hypoperfusion (45%). Overall mortality was 34%. The median length of stay was 11 (7.20) days, 25% had hemodialysis and 16% were admitted in the ICU. The risk factors for mortality identified were, hospital acquired AKI (OR: 6.59, 95% CI: 1.320-32.889, p=0.021), ICU admission (OR: 5.66, 95% CI: 2.061–15.512, p=0.001) and HIV infection (OR: 2.61, 95% CI: 1.063-6.424, p=0.036). The Commonest causes of AKI still remain infections and hypovolaemia and mortality from it was high in our patient population. Early identification of AKI and those at high risk of mortality and provision of adequate treatment are critical to improving outcomes in AKI patients.

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Introduction

Acute kidney injury (AKI) is a disease that has a global impact. The global prevalence in adults is approximately 21.6%, with 45% of sufferers coming from low and low-middle-income countries.[1,2] There are clear differences in the demographics and etiology of AKI patients. In developed countries, AKI primarily affects the elderly, whereas in developing countries, the majority of those affected are younger.[3-7] Several risk factors play a significant role in the pathogenesis of AKI. The most common factors in Sub-Saharan Africa are infections with septicaemia, hypovolemia, and glomerulonephritis.[3-5,8,9] Other causes include obstructive uropathy, plant nephrotoxins, and obstetrics and gynecology.[5,10-12] AKI is associated with development of Chronic Kidney Disease (CKD), progression of established CKD, increased mortality, increased Length Of hospital Stay (LOS), and high treatment cost.[13] AKI mortality is higher in the tropics than the rest of the world, with up to 32% of adults dying from AKI compared to a global rate of

23.3%.[5,14] Several studies have looked at the etiology and risk factors for AKI mortality in Nigeria, the majority of which are in southern Nigeria, but there are few studies in central Nigeria. As a result, identifying the causes and risk factors associated with increased mortality in this region is critical, as knowledge of these will drive efforts on detection, treatment, and prevention, in line with the ISN 0by25 mandate to eliminate AKI.[15] Using data collected over an eleven-year period, our goal was to examine the common predisposing/aetiological factors and short-term outcomes of AKI, as well as the risk factors most associated with mortality in our patients in the Federal Capital Territory. The findings of this study will help to improve strategies for effective medical care and public health on AKI.

Materials and Methods

From January 2010 to December 2020, we reviewed our records of patients seen at the adult nephrology unit at the University of Abuja Teaching Hospital. This included all patients referred to the unit by the medical, surgical, obstetrics and gynecology (Ob/Gyn), Intensive Care Unit (ICU), and emergency departments. The study included all patients aged 16 and up diagnosed with AKI. Patients with End-Stage Renal Disease (ESRD), on maintenance dialysis, renal transplant patients, patients without any identifying feature of AKI, and patients without any serum creatinine value at the time of admission were excluded. The data was entered by three research assistants. Members of the research team adjudicated and confirmed all AKI episodes to determine that AKI rather than CKD progression had been detected.

Definition of AKI

AKI was defined by KDIGO diagnosis and staging criteria as an increase in SCr greater than or equal to 26umol/L in 48 hours or 1.5 to 1.9 times baseline within 7 days.[16] Also used-for this study, was a urine output of less than 0.5 mL/hr for more than 6 hours or a history of oliguria or anuria for 24 hours and at least 1 creatinine, as well as any sign of acute renal disease (risk factors, normal kidney size, and normal hemoglobin level).

Variables/Definition of variables

Demographics, referral department, ICU admissions, clinical conditions, causes of AKI, pre-existing co morbidities that were either present or absent, exposure to medicines with nephrotoxic potential, herbal drug use as ever/never used within the 3-month prior admission, and pregnancy status in females were all collected. When available, creatinine values were obtained at baseline (any value within the previous one year), on admission, within 7 days of presentation, and within 3 months of discharge. Peak creatinine and potassium levels were measured and recorded. Peak potassium was high if it was >5.5 mmol/L and low if it was <3.5mmol/L. Serum bicarbonate was low if it was ≤10mmol/L. Dipstick urinalysis results for proteinuria and hematuria at presentation were obtained. Haemoglobin, at presentation, was also recorded and was categorized as ≥10 and <10g/dL.

Hypertension was self-reported or if patients were on blood pressure lowering medication prior to index presentation, with or without blood pressure ≥140/90 mmHg, while hypotension was systolic blood pressure of < 90mmHg and a diastolic BP of <60mmHg at presentation. Causes of AKI were abstracted from the case notes and adjudication was done based on presentation. Sepsis was defined as the presence of microbial infection in the presence of at least two of the following- temperature >38°C or <36°C, pulse rate >90/minute, respiratory rate >24cycles/minute, white cell count of >12,000 cells/mm^3 or <4000 cells/mm^3.[17] Hypovolaemia was defined as fluid loss with features of dehydration and changes in the cardiovascular system such as tachycardia and hypotension. Hypoperfusion was defined as conditions that cause reduced blood perfusion to the kidneys such as congestive cardiac failure, hepatorenal syndrome, and nephrotic syndrome. Hospital-acquired AKI was defined as AKI diagnosed after 7 days of admission to the hospital.

Outcomes

All-cause in-hospital mortality, haemodialysis, Length of hospital Stay (LOS) (days from admission to the day of discharge), and ICU admission were the patient outcomes. The renal outcomes were complete renal recovery defined as normalization of serum creatinine within 3 months of AKI (equal to or lower than baseline or index creatinine). Partial recovery occurred when serum creatinine was lower than the diagnosis value but not to baseline or reference without dialysis, or when there was no dialysis for more than 4 weeks, or when serum creatinine decreased and stabilized between 1.2-1.5 times baseline.[18] No recovery was defined as doubling of serum creatinine, starting maintenance RRT, or remaining on dialysis after 3 months.[18]

Statistical analysis

Continuous variables were presented as means with standard deviations, and if skewed, as medians with interquartile range. We presented data for categorical variables as frequencies with percentages. The patients were classified into two groups based on age: (age <60 years) and (age ≥60 years). For continuous variables, we used a two-sample t-test or the Mann-Whitney U test (for skewed data), and for categorical variables, we used the chi-square test. We divided patients into survivors and non-survivors and looked at risk factors for death. Variables with fewer than six events per variable were removed. We investigated the univariable relationship between risk factors and mortality. The multivariable logistic regression model included variables with known clinical relevance and a P value less than 0.25. The Odds Ratios (ORs) and Confidence Intervals (CIs) were displayed. The alpha level for statistical significance was set at 0.05. All data were entered into Excel and analyzed with Stata (version 16.1).

Results

We identified 653 patients with AKI but confirmed 399 patients for the final study, categorized as age <60 years or younger (n=299, 75%), and age ≥ 60 years or older (n=100, 25%).

The clinical features of participants are demonstrated in Table 1. The patients were more likely to be <60 years. There were significantly more males than females in each of the age groups (p=0.029). A higher proportion of patients with hospital-acquired AKI, were older (10 *vs* 5%) (p<0.029). A significantly higher proportion of the ≥60 years group had hypertension (p<0.001), diabetes (p<0.001), anaemia (p<0.001) malignancies (p<0.001), and stroke (p<0.001) than the <60 years group. They were more likely to have more than 3 comorbid diseases even as there were more tendencies of the younger group to have multiple causes of AKI. Medicine department admitted the largest proportion of patients in both age groups.

Table 2 displays the laboratory characteristics of the participants. The mean hemoglobin was significantly lower in the <60 age group. Proteinuria of 2+ and above was observed more in the younger age group. A higher proportion of the ≥60 age group had haematuria compared to the <60 group even though this was not significant (p=0.243), while a higher proportion of the <60 group had both hypo and hypernatremia, hyperkalaemia, and Low bicarbonate. The majority of the participants had stages 2 and 3 AKI with significantly more older patients with stage 2 AKI (p=0.015) and younger patients with stage 3 AKI (p=0.004).

Figure 1 displays, the causes and probable precipitants of AKI. Septicaemia/infection 247 (62%), and hypovolaemic/hypoperfusion (ischemic) 181 (45%), were the most common identified. For hypovolaemia/hypoperfusion causes, diarrhoea and vomiting 51 (28%), haemorrhage 39 (22%), and congestive cardiac failure 33 (18%) were the commonest observed. In infectious/septicaemic causes, the commonest focus of infection were gastrointestinal including typhoid fever 37 (15%), urosepsis 22 (9%), HIV related 47 (19%). The majority had sepsis with unknown focus 115 (47%). Malignant hypertension 8 (24%), and nephrotic syndrome 11 (33%) were prominent among the causes due to glomerulonephritis. Herbal concoction use 84 (60%), tenofovir 22 (16%), and NSAID use 28 (20%) were commonest with medication/drug category. Among obstructive causes, prostrate hypertrophy 22 (26%) was the commonest while preeclampsia/eclampsia 32 (64%) was commonest in the obstetric causes. Diseases like malaria,

Table 1. Demographic and clinical characteristics of AKI patients according to age.

Characteristics	Overall	Age <60 years	Age ≥60 years	p-value
Number	399	299	100	
Mean Age (years)	45.0 ± 17.3	37.2±11.5	68.5±7.4	
Sex (%)				
Male	230 (58)	163 (55)	67(67)	0.029
Female	169 (42)	136 (45)	33(33)	
AKI type (%)				
Hospital acquired	25 (6)	15 (5)	10 (10)	0.075
Admitting Department				
Surgery	41(10)	22(7)	19(19)	<0.001
Obs/Gyn*	50(30)	50(30)	0	-
Medicine/GOPD	265 (66)	187(63)	78(78)	0.005
ICU	63 (15)	56(19)	7(7)	0.005
Clinical characteristics (%)				
MAP	99.2±26.4	100.2±26.8	97.6±22.9	0.379
Systolic BP <90mmHg (%)	15(4)	10(3)	5(5)	0.543
Diastolic BP<60mmHg (%)	29(7.27)	21(7)	8(8)	0.745
Systolic BP ≥ 140 mmHg (%)	154(39)	112(38)	42(42)	0.419
Diastolic BP ≥90 mmHg (%)	149(38)	116(39)	33(33)	0.300
Oliguria (%)	213 (53)	162(54)	51(51)	0.581
Co-morbidities (%)				
Hypertension	156 (39)	94(31)	62(62)	<0.001
Diabetes	70 (18)	37(12)	33(33)	<0.001
Chronic Kidney Disease	58 (14)	42(14)	16(16)	0.631
Stroke	19	4(1.3)	15(15)	<0.001
Anemia	185 (48)	123(42)	62(63)	<0.001
Liver disease	24 (6)	17(6)	7(7)	0.632
Heart failure	33 (8)	21(7)	12(12)	0.118
Malignancies	15	3(1)	12(12)	<0.001
HIV	47(12)	44(15)	3(3)	0.001
HbsAg	28(10)	27(12)	1(1.5)	0.003
HCV	24(8.3)	15(7)	9(14)	0.196
Sickle cell disease	7	7(100)	0	
No of comorbidities (%)				
0	69(17)	60(20)	9(9)	0.011
1	120(30)	95(32)	25(25)	0.201
2	109(27)	81(27)	28(28)	0.860
3 and above	101(26)	63(21)	38(38)	0.001
No of causes (%)				
Single cause	230(60)	166(57)	63(68)	0.058
Multiple causes	156(40)	126(43)	30(32)	0.066
Medication/Drugs (%)				
ACEI or ARB	112 (28)	79(26)	33(33)	0.205
Diuretics	191 (48)	138(46)	53(53)	0.235

Abbreviations: AKI: acute kidney injury, GOPD: general out-patient department, ICU: intensive care unit, MAP: mean arterial blood pressure, HIV: human immunodeficiency virus, HCV: Hepatitis C virus antibody, HBsAg: Hepatitis B surface antigen, ACEI: angiotensin converting enzyme inhibitor, ARB: angiotensin receptor blocker. *only females.

haemolytic uremic syndrome, and rhabdomyolysis were some rare causes that were observed.

Table 3 displays the outcomes observed. There were 134 (34%) deaths in the overall cohort. Mortality did not differ significantly between the 2 age groups (p=0.698). The median length of stay was not different in the 2 groups also. A quarter of the patients had dialysis. Significantly more of the younger age group were admitted into ICU (p=0.005). For the renal outcomes, 36% completely recovered kidney function. There were not significant differences observed in all the renal outcomes between the 2 age groups.

The univariate and multivariable analysis of survivors versus non survivors are displayed in Table 4. Variables that were significant (p<0.25) in univariable regression were selected for the multivariable model and were as displayed in the table. The significant risk factors for mortality on adjusting were, hospital acquired AKI (AOR: 6.6, 95% CI: 1.320-32.889, p=0.021), ICU admission, (AOR:5.7, 95% CI: 2.061-15.512, p=0.001, presence of HIV, (OR: 2.6, 95% CI: 1.063-6.424, p=0.036).

Discussion

In this study, a higher proportion of patients were young (60 years old), males, and had community acquired AKI rather than hospital acquired AKI. Several authors[3,4,7] in Nigeria reported a younger age. AKI typically affects younger age groups who are considered healthier in developing countries, as opposed to patients in developed countries who are older and have more comorbidities.[10] Gender disparities were observed here, as in other reports, lending support to the validity of our findings.[19,20] It is possible that males are more at risk of AKI, despite having better access to health care than females in Nigeria.[21] In our study, community-acquired AKI was more common than hospital-acquired AKI. This matches the situation in low-income countries.[3,4,19] Halle *et al.*, from Cameroon, reported only 6.7% of their patients developed AKI while in hospital.[12] As reported by other authors in Sub-Saharan Africa,[3,4,12] two-thirds of our patients had stage 3 AKI. The late presentation could be due to a poor health-seeking

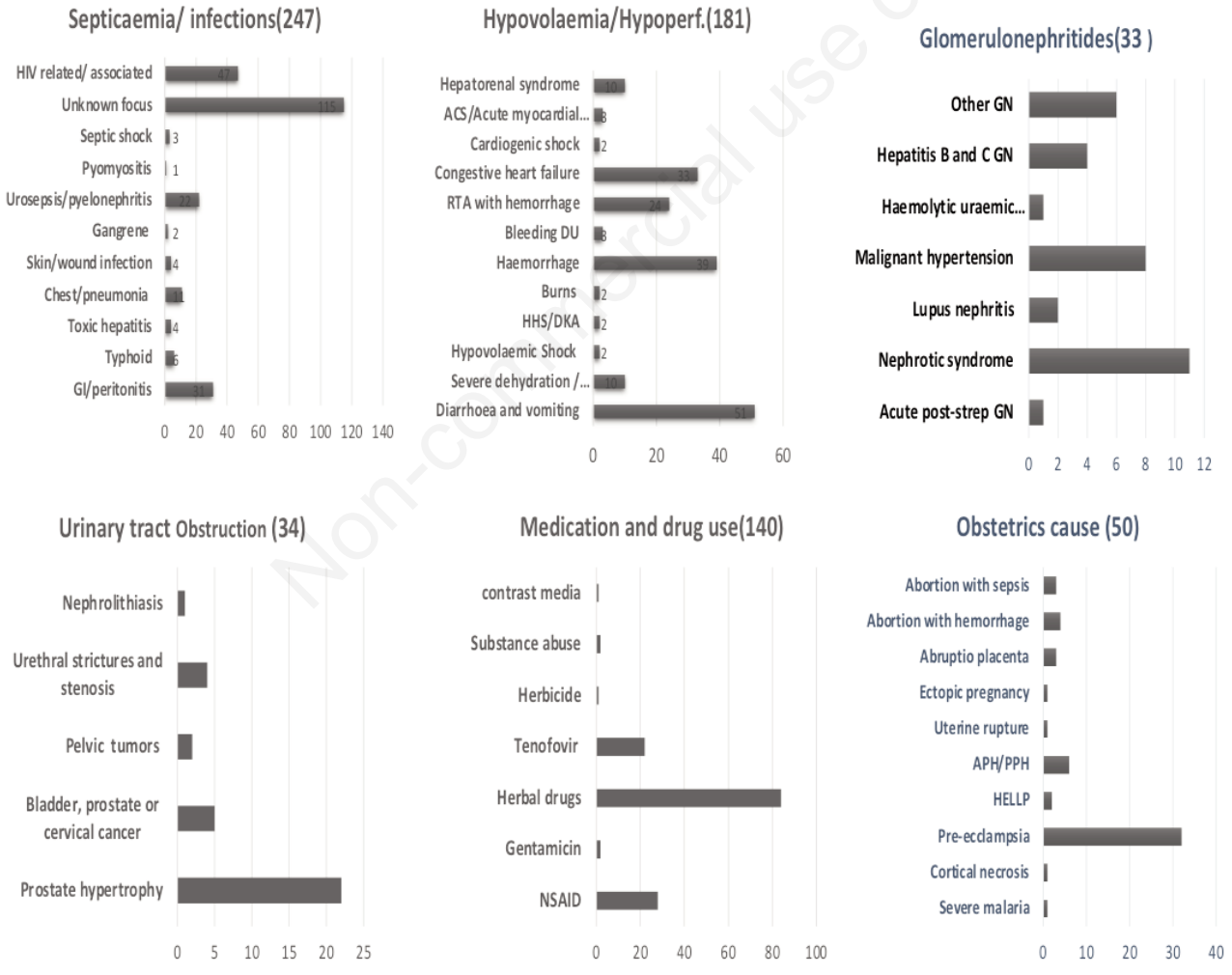

Figure 1. Identified risk factors/causes of acute kidney injury. Abbreviations: ACS: acute coronary syndrome, RTA: road traffic accident, DU: duodenal ulcer, HHS: hyperosmolar hyperglycaemic syndrome, DKA: diabetic keto acidosis, GN: glomerulonephritis, NSAID: nonsteroidal anti-inflammatory drugs, HELLP: haemolysis elevated liver enzymes and low platelets

habit, ignorance, or poverty. Many patients in our society seek help at religious or herbalist homes first.

The most common causes of AKI observed were septicaemia/infections (62% and 45%, respectively), and several authors had demonstrated similar findings.[3,4,7-12] The prevalence of septicemia and hypovolaemia as common causes of AKI reflects environmental conditions in developing countries where infections are still prevalent. This study, like another northern Nigerian study,[9] found frequent use of herbal drugs and traditional medicine remedies (35%). Halles *et al.* reported 32.3% use of herbal remedies,[12] while XU reported 40% in China.[22] Traditional herbal medicine is typically prescribed by traditional medicine practitioners who are unaware of the negative effects.[23,24] Obstetric/gynecological causes of AKI continue to be prevalent in Nigeria, accounting for 30% of our females. Makusidi *et al.* reported similar findings.[9,11] It was reported in 7.1% (38/536) of all AKI cases in Cameroon.[12] Unsafe home deliveries, illegal abortions, pre-eclampsia, haemorrhage from ante and postpartum haemorrhage, uterine rupture with hypotension, and sepsis are the causes. In 40% of our patients, there were multiple causes/precipitants. Multiple co-precipitants of AKI are common in developing countries, such as a patient with typhoid/sepsis who also has gastroenteritis and

Table 2. Laboratory characteristics of AKI patients according to age.

Variables	Overall 399	Age <60 years n=299	Age ≥60 years n=100	p-value
Hemoglobi ±SD(g/L)*	9.79±3.12	9.43±2.99	10.68±2.45	<0.001
WBC(IQR) cells/mm^3**	9.1(6.3,14.4)	9.75(6.4,15.3)	8.35(6,12.6)	0.171
Proteinuria				
None	156	109(38)	49(52)	0.026
1+	92	65(23)	27(29)	0.280
2+	76	65(22)	11(12)	0.011
3+ and above	54	46(15)	8(8)	0.065
Hematuria				
None	185(49)	144(51)	41(44)	0.214
Positive	190(51)	138(49)	52(56)	0.243
Peak creatinine (IQR)**	521(268,991)	591(282,1029)	352 (218,762)	0.001
AKI	26(7)	17(6)	9(9)	0.245
Stage 1				
Stage 2	110(27)	73(24)	37(37)	0.015
Stage 3	263(66)	209(70)	54(54)	0.004
Highest urea±SD mmol/L*	23.87±13.27	25.39±12.76	21.884±12.72	0.018
Sodium ±SD mmol/L*	137.85±11.17	137.75±11.19	138.13± 11.14	0.773
Sodium Normal(%)	183(46)	133(45)	50(50)	0.338
High >14 mmol/L(%)	122(31)	95(32)	27(27)	0.370
Low <13 mmol/L(%)	94(24)	71(24)	23(23)	0.879
Peak Potassium ±SD mmol/L*	4.7±1.053	4.81±1.11	4.71±0.85	0.420
Normal (%)	273(70.7)	195(67)	78(81)	0.017
>5.5 mmol/L(%)	82(21)	70(26)	12(13)	0.014
<3.5 mmol/L (%)	31(8)	25(9)	6(6)	0.445
HCO_3±SD mmol/L*	17.75±6.39	17.21±6.32	19.05±6.32	0.014
HCO_3 <=10 mmol/L	41(10)	34(11)	7 (7)	0.213

WBC white blood cells, HCO3 bicarbonate, IQR interquartile range, *mean **median (Mann-whitney U).

Table 3. Short term outcome of AKI according to age of participants.

Patient outcomes	Overall 399 n(%)	Age <60 (n=299) n(%)	Age ≥60 (n=100) n(%)	P-value
In-hospital mortality	134(34)	102(34)	32(32)	0.698
Median LOS (days)	11(7,20)	11(17,19)	11(17,21.5)	0.572**
Dialysis	99 (25)	81(27)	18(18)	0.068
ICU Admission	63 (16)	56(19)	7(7)	0.005
Renal outcomes				
Complete recovery	146(36)	105(35)	41(41)	0.337
Partial recovery	59(15)	44(15)	15(15)	1.000
No recovery/progression	15(4)	12(4)	3(3)	0.770
Unknown/LAMA	45(11)	36(12)	9(9)	0.468

LOS- length of stay. ** Mann-Whitney U, ICU intensive care unit, LAMA left against medical advice.

Table 4. Univariate and multivariable analysis of risk factors for mortality in Acute Kidney Injury.

Variables	Univariate			Multivariate		
	OR	95%CI	P-value	AOR	95%CI	P-value
Hospital acquired	4.67	1.959-11.122	0.001	6.59	1.320-32.889	0.021
ICU admission	5.79	3.233-10.364	<0.001	5.66	2.061-15.512	0.001
CKD	0.59	0.309-1.113	0.102	0.68	0.238-1.967	0. 482
SBP≥140mmHg	0.68	0.463-0.992	0.045	0.46	0.207-1.003	0.051
DBP >90mmHg	0.74	0.481-1.152	0.186	0.99	0.407-2.417	0.987
Hemoglobin<10g/dL	1.28	0.840-1.963	0.249	1.75	0.842-3.616	0.134
Sepsis	1.47	0.955-2.248	0.080	1.55	0.728-3.289	0.258
ACEI or ARB	0.38	0.223-0.633	<0.001	1.41	0.624-3.174	0.410
HIV	1.60	0.673-2.368	0.158	2.61	1.063-6.424	0.036
Proteinuria	0.74	0.481-1.143	0.176	0.82	0.409-1.653	0.580
Peak Cr >350	1.30	0.840-2.022	0.237	2.35	0.928-5.950	0.071
Hyponatremia	0.56	0.347-0.901	0.017	0.52	0.236-1.125	0.096
Hypernatremia	3.18	1.922-5.270	<0.001	1.30	0.492-3.430	0.059
HCO_3< 10mmol/L	1.80	0.924-3.501	0.084	1.82	0.711-4.674	0.211
hypoperfusion	0.62	0.310-1.234	0.173	1.39	0.498-3.876	0.529

Abbreviations: OR: odds ratio, AOR: adjusted odds ratio, ICU: intensive care unit, ACEI: angiotensin converting enzyme inhibitor, ARB: angiotensin receptor blocker, HIV: human immunodeficiency virus, Cr: creatinine, HCO_3: bicarbonate.

uses nephrotoxic herbal remedies.

This report revealed a high mortality rate of 34%. This finding was consistent with previous research in Nigeria and elsewhere.[3-5,10,19] Emem-Chioma *et al.* reported a higher rate of 44%, but their study only included patients in the severe stage of AKI using the RIFLE criteria,[19] whereas ours included patients in all stages using the KDIGO criteria.[16] The high mortality rate observed here could be attributed to a lack of funds for proper care, as well as late presentation to hospitals, among other factors.

We found that the median length of stay was 11 (IQR 7.20) days, which did not differ between age groups (p=0.572). Halle *et al.* observed a median length of stay of 8 (IQR 1.41) days in Cameroon,[12] whereas Chertow *et al.* reported that an increase in SCr >or=0.5 mg/dL was associated with a 3.5-day increase in The LOS.[13] Longer LOS is typically associated with higher costs for patients and their families.[13] A quarter of our patients received dialysis; Effa *et al.* reported 21.4%,[4] while Oluseyi *et al.* reported 62%.[3] We included patients with early-stage AKI who did not have a dialysis indication, and not all who needed dialysis could be dialyzed, accounting for the lower 25% observed. Many Nigerian patients still cannot afford dialysis and thus have a poor prognosis.

Mortality was 2.6 times likely to occur in HIV/AIDS patients compared to their counterparts with no HIV/AIDS. Even in the post-HAART era, Wyatt *et al.* found a mortality risk of 5.83 (95% CI, 5.11-6.65).[25,26] HIV-infected people are more likely to develop AKI and its complications. The cause of this observation in our patients is unknown, but it could be due to anti-retroviral naivety, severe immunosuppression (CD4 count, 200 cells/mm), and the presence of opportunistic infections. Another reason could be a late presentation. In this study, Hospital-Acquired (HA) AKI was a significant risk for mortality (AOR: 6.6, 95% CI: 1.320-32.889, p=0.021), and may result from nosocomial infection leading to sepsis, which was a common cause of AKI observed. Admission into ICU is another risk factor observed, (AOR:5.7, 95% CI: 2.061-15.512, p=0.001). In-hospital mortality is common in AKI severe enough to necessitate ICU admission.[27,28]

The study has limitations because it was conducted in a single center, was retrospective, and contained missing data, which may have influenced the results. Any conclusions derived from the data may require replication through hypothesis testing.

In conclusion, identifying the causes of AKI and risk factors for mortality is helpful in prioritizing patients at risk, increasing the chance of improving outcomes. Efforts must be increased to achieve ISN vision 0 by 2025.

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