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Clinical spectrum and outcomes of acute kidney injury: A prospective study from an intensive care unit of South India

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Abstract

***Introduction*: The prevalence of acute kidney injury (AKI) among hospitalized patients in the United States of America is around 5-7%, and that among Intensive Care Units (ICU) is 30%. The mortality and morbidity associated with AKI are immense, and the prevalence has been increasing over the past decades. However, the data on the prevalence, profile, and outcome of AKI in hospitalized patients from the developing world is meager.**

***Objective*: This study aimed at determining the common etiologies and clinical profile of AKI patients in the ICU of a tertiary care center, in South India and to assess the outcome of AKI in community-acquired and hospital-acquired AKI patients with respect to its etiology.**

***Materials and methods*: This study was designed to be a prospective observational study. We included 150 patients who were either directly admitted to ICU or transferred from ward to ICU, in our tertiary care center, who had AKI either at admission or during the course in the hospital. They were followed up until discharge/death and their clinical and biochemical data were studied.**

***Results*: The causes of AKI were grouped as acute tubular necrosis (56%), volume loss/hypoperfusion (26%), acute interstitial nephritis (12%), urinary tract obstruction (4.7%), and glomerulonephritis (1.3%). The major contributing illness for AKI was sepsis (including septic shock) (28.7%). The other common causes were nephrotoxins (24%), acute gastroenteritis (13.3%), pyelonephritis (7.3%), and urinary tract obstruction (4.7%). Among the subjects, 82.7% had community-acquired AKI and 17.3% had hospital-acquired AKI. Most of the patients (60%) recovered from AKI, whereas 36% had partial recovery, one person was dependent on renal replacement therapy (0.7%), and death occurred in 3.3% (n=5). The outcome between community-acquired and hospital-acquired AKI was statistically non-significant.**

***Conclusion*: Sepsis and nephrotoxins were the most common causes of AKI in our study. Community-acquired AKI was more prevalent than hospital-acquired AKI. The mortality rate of AKI patients in the ICU was less (3.3%).**

Key words: Acute kidney injury, AKI in developing countries, hospital-acquired AKI, community-acquired EMI.

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Introduction

Acute kidney injury is a huge issue worldwide and accounts for the bulk of mortality among hospitalized patients in critical care. In the United States of America, the prevalence of acute kidney injury (AKI) is around 5-7% in hospitalized patients, and that in Intensive Care Units is 30%. (1) In contrast to the developed countries, the etiology and outcomes of AKI in a developing country are different and predominantly community-acquired AKI (CAAKI). (2) Acute insult may be in the form of sepsis, hypoperfusion, toxin, obstruction, or parenchymal kidney disease. Background comorbidities in the form of old age, diabetes mellitus, chronic kidney disease, cardiac failure, liver failure, vascular disease, and nephrotoxic medications also contribute to the insult. (3) During the past decades, AKI was referred to as acute renal failure, and there are multiple definitions that created a difference in the calculation of its exact incidence. (4) The recent definitions by the Acute Dialysis Quality Initiative (ADQI) - risk, injury, failure, loss of kidney function, and end-stage kidney disease (RIFLE) classification in 2002, Acute Kidney Injury Network (AKIN) classification in 2004, and Kidney Disease: Improving Global Outcomes (KDIGO) definition in 2012 has made the diagnosis of AKI simple, universal, and easy to detect at an early stage, where reversibility is still possible.

When comparing the developed and developing regions of the world, there are multiple similarities and differences in the clinical spectrum of AKI and its public health implications. (5) In 2013, the International Society of Nephrology recognized AKI as a major mortality predictor and initiated the "0 to 25 initiative", which aimed at preventing deaths from treatable causes of AKI, worldwide. (6) Though 2 years away from the initiative target time, the prevalence of AKI in developing countries is still not only high but also variable, because of the scarcity of studies and data conveying the epidemiology and causes of AKI, lack of AKI registry for the country. (7) Moreover, the outcome of patients with AKI is directed by the etiology and background morbidity. This single-center study was aimed at determining the various etiologies causing AKI, its clinical spectrum of presentation, CAAKI vs hospital-acquired AKI (HAAKI), and outcomes based on that, in an intensive care unit (ICU) of a tertiary care center in South India.

Material and methods

This study was designed to be a prospective observational study. We included 150 adult patients admitted to the ICU or transferred from the ward to the ICU in our tertiary care center, Sri Ramachandra Institute of Higher Education and Research, Chennai, South India, who had AKI (as defined by KDIGO criteria), either at admission or during the course in the hospital. The study period was from November 2020 to October 2021. The patients were followed up until discharge or death whichever was earlier, and their clinical and biochemical data were studied.

Exclusion criteria

Patients less than eighteen years of age, pregnant females, and patients with chronic kidney disease were excluded.

Objective

The objective of the study was to determine the various etiologies causing AKI, its spectrum of presentation, and outcomes of AKI in community-acquired and hospital-acquired AKI patients and with respect to its etiology, in the ICU of a tertiary care center in South India.

Methodology

The patient's social, demographic, economic, and clinical details were recorded in the proforma sheet. The presenting complaints, history regarding urine output, and the duration of symptoms were recorded. Baseline clinical examination including vitals and hourly urine output of all selected patients were done. Baseline investigations including complete blood haemogram, blood urea nitrogen, creatinine, sodium, potassium, chloride, bicarbonate, and fasting and post-prandial blood sugar were noted at the time of diagnosis of AKI, and frequently repeated as required by the patient's condition, and at the time of discharge. USG abdomen was done for all patients to assess the kidney size and echogenicity to rule out chronic kidney disease. Other relevant investigations such as an anti-nuclear antibody, renal biopsy, and fever profile were done, as warranted by the patient's clinical condition. All patients were followed up till the time of discharge or death, whichever was earlier.

The most recent creatinine value available before the onset of AKI was taken as the baseline creatinine level. If no baseline creatinine was accessible, then the first creatinine value done during the current hospital admission was considered as the reference creatinine value. As per the AKI - KDIGO criteria, AKI was defined as an increase in serum creatinine of ≥0.3 mg/dl within 48 hours or, an increase in creatinine by ≥50% from the baseline/reference value within seven days or, if the urine output was less than 0.5 ml/kg/hour for six or more hours. (8) The data was collected in a Microsoft Excel data sheet and statistically analyzed by IBM SPSS version 21 (IBM Corporation). Descriptive statistics like mean, standard deviation, proportion, and percentage were used. The student's t-test and Pearson's chi-square test were used to test the significance. A p-value <0.05 was considered statistically significant.

The study was done in accordance with the protection of human subjects and was approved by the Institutional Ethical Review Committee. Written informed consent was collected from the study participants or their legal surrogates.

Results

During the one-year period, a total of 150 patients were identified with AKI in ICU. Out of 150, 60.7% (n=91) were males and 39.3% (n=59) were females.

The mean age of patients with AKI was 54.47±16.51 years (ranging from 19-90 years) with the majority from the age group of 41-50 years (21.3%). Based on the co-morbidities, 58.7% of them had type 2 diabetes mellitus, 47.3% had hypertension, 14% had coronary artery disease, 4.7% had stroke in the past, and 8.7% had dyslipidemia (**Figure 1**).

The frequency of the presenting complaints in decreasing order of frequency were fever 26.7%, loose stools 15.3%, oliguria 10%, headache and abdominal pain 9.3% each, altered sensorium 8%, anasarca 4.7%, cough 3.3%, lower urinary tract symptoms and chest pain 2.7% each, dyspnea, seizures and ascites 1.3% each, and back pain, bone pain, cerebrovascular accident, jaundice were all less than 1% each.

The various etiologies of AKI are presented in **Table 1**. All the causes of AKI among the 150 subjects were grouped under five sub-headings: Acute tubular necrosis (56%), Volume loss/hypoperfusion (26%), Acute interstitial nephritis (12%), Urinary tract obstruction (4.7%), and Glomerulonephritis (1.3%). The major contributing illness for AKI was sepsis (including septic shock), which was present in 28.7% (n=43). The other common causes were nephrotoxins (24%), acute gastroenteritis (13.3%), pyelonephritis (7.3%), and urinary tract obstruction (4.7%).

The etiology of sepsis and septic shock among 43 patients, in decreasing order of frequency, were: genito-urinary (n=19), pneumonia (n=11), soft tissue infections (n=4), gastroenteritis (n=3), spontaneous bacterial peritonitis (n=2), enteric fever (n=2), tuberculous meningitis (n=1), and cholecystitis (n=1).

The nephrotoxins that were found among the 36 subjects with AKI were amphotericin B (n=11), non-steroidal anti-inflammatory drugs (NSAID) (n=10), contrast agents (n=4), diuretics (n=3), rifampicin (n=2), native medicines (n=2), remdesivir (n=1), acyclovir (n=1), and vancomycin (n=1). AKI secondary to tropical fever was found in 13 individuals (8.7%), among which dengue (n=5) was the leading cause, followed by scrub typhus (n=4), leptospirosis (n=3), and malaria (n=1). Among the subjects, 82.7% (n=124) had CAAKI, and 17.3% (n=26) had HAAKI. Based on the anatomical classification, pre-renal AKI was found in 26.67% (n=40), renal AKI in 68.67% (n=103), and post-renal AKI in 4.67% (n=7).

Among the 150 AKI patients, 85.3% (n=128) had conservative management. There was a requirement for vasopressor in 13.3% (n=20), ventilator support in 8.7% (n=13), and renal replacement therapy in 8% (n=12). Most of the patients (60%) recovered from AKI, whereas there was a partial recovery (creatinine did not reach baseline at the time of discharge) in 36% of individuals, one person was dependent on renal replacement therapy (0.7%), and death occurred in 3.3% (n=5). The outcome between community-acquired and hospital-acquired AKI was compared (**Table 2**); the chi-square value was 4.218, and the p-value was 0.239, which was statistically non-significant.

The mortality rate was higher in the pre-renal cause of AKI (n=3) than in renal (2) and post-renal (n=0). Among the pre-renal AKI patients, the majority of them (75%) recovered completely. In the renal cause of AKI group, 56.3% recovered completely whereas 40.8% of individuals had only partial recovery at the time of discharge. Recovery from acute kidney injury was prolonged in the post-renal AKI group. Only 28.6% of subjects had complete recovery at the time of discharge in the post-renal AKI group, and 71.4% had partial recovery of AKI. The comparison of outcomes among pre-renal, renal, and post-renal AKI groups was found to be statistically significant (p=0.041) (**Table 3**). Out of the 12 individuals who required RRT in the hospital, eight of them partially recovered (66.7%), two individuals expired (16.7%), one had complete recovery (8.3%), and one was renal replacement therapy (RRT) dependent even at discharge (8.3%). The outcome among patients who required RRT in the hospital was compared, and the results were statistically significant (chi-square 27.667, p-value 0.001) (**Table 4**).

Discussion

The prevalence of AKI is poorly characterized, and its epidemiology varies according to the population and definition of AKI, thereby masquerading its burden in the community. (9,10) AKI in critically ill patients amplifies the risk of mortality, which is further heightened by older age groups and multiple comorbid conditions.

In our study, 60.7% of the 150 patients with AKI were males and 39.3% were females, with a male-to-female ratio of 1.5:1. Similar findings were made by Al-Homrany M in a study of AKI in the Saudi Arabian population, where male to female ratio was 1.4:1, indicating a male predilection to AKI. (11) The Program to Improve Care in Acute Renal Disease (PICARD) experience study with 618 patients found that 59% were males and 41% were females, yielding identical results to the current study. (12) In our study, the mean age of the AKI patients was 54.47±16.51 years. The most prevalent age category in both genders was 41-50 years. A similar observation was found in a study of 144 individuals with AKI in Marathwada, by Guradkar S. et al., where

the mean age was 48±18.24 years. (13) However, in a Saudi Arabian study, patients were marginally older with a mean age of 58.9±22.5 years with a predominance of elderly over 60 years of age. (11)
The most common co-morbid condition found in the study group was diabetes mellitus (58.7%), followed by hypertension (47.3%) and coronary artery disease (14%). Co-morbidities may have a negative impact on renal outcomes in the elderly, making them a high-risk group for AKI, thereby stressing the importance of early identification and treatment. (8) Surprisingly, in the PICARD study, hypertension was the most common co-morbidity (31.6%), followed by diabetes (24.2%), and heart failure (17.6%). (12) In Al-Homrany M, et al. study too, cardiac problems were more prevalent (16%) followed by diabetes (15.3%), which are all different from our study. (11)
The most common presenting symptom in the current study was fever (26.7%), followed by loose stools (15.3%), oliguria (10%), headache (9.3%), and abdominal pain (9.3%). Altered sensorium, anasarca, cough, lower urinary tract symptoms, chest pain, dyspnea, seizures, ascites, back pain, bone pain, cerebrovascular accident, and jaundice were among the other symptoms. However, a recent study by Nagamani R et al. found oliguria (80%) to be the common presenting symptom in AKI patients rather than fever, which was found in 58%. (14)
Broadly categorizing the etiology of AKI, the most common cause was acute tubular necrosis (ATN) accounting for 56% of the AKI patients in the ICU setting of our hospital. The other etiologies were volume loss (26%), acute interstitial nephritis (12%), urinary tract obstruction (4.7%), and glomerulonephrítis (1.3%) in decreasing order of frequency.
The most common etiology of AKI in our study was sepsis and septic shock, which contributed to 28.7% of the AKI. The other common causes were nephrotoxins (24%), acute gastroenteritis (13.3%), pyelonephritis (7.3%), and dengue fever (3.3%). The source of sepsis and septic shock in the majority of the cases were found to be from genitourinary tract infection (44.2%) and pneumonia (25.6%). A similar report was stated in Al-Homrany M. et al. study, in which the primary cause of AKI was ATN, found in 62.5%, in which sepsis accounted for 24.7%. (11)
In a study from a tertiary hospital in Puducherry, India, 22.4% of cases of AKI were secondary to sepsis. (15) According to a study in Marathwada, sepsis was the predominant cause of AKI (31.53%), (13) followed by acute gastroenteritis (16.21%), acute pancreatitis (14.41%), contrast-induced nephropathy (9%), hepatorenal syndrome (8.10%), drugs (6.30%), malaria (4.50%), and dengue fever (4.50%). A study by Sandeep Mahajan et al., demonstrated sepsis (73.2%), nephrotoxic drugs (53.2%), and dehydration (22.1%) as the major causes of AKI. (16) All these studies had sepsis as the prime cause of AKI, which was similar to our study.
In our present study, nephrotoxic drugs were responsible for 24% of the cases, with amphotericin B (used as an anti-fungal agent against mucormycosis, which was common during the COVID-19 pandemic) being the most common offending drug, followed by NSAIDs, contrast agents, rifampicin, diuretics, and herbal medicines. A prior study in a tertiary care hospital in Bangalore, India, found that nephrotoxic medications (38%) were the most common cause of developing renal failure in a hospital, followed by sepsis (35%). (17)
In our study population of 150, AKI secondary to tropical fever was found in 13 individuals (8.7%) among which dengue (n=5) was the leading cause followed by scrub typhus (n=4), leptospirosis (n=3), and malaria (n=1). In a study by Gopal Basu et al. on tropical fever causing AKI, scrub typhus led with 51.2%, followed by falciparum malaria (10.4%), enteric fever (8.7%), dengue (7.6%), mixed malaria (6.5%), leptospirosis (3.3%), and undifferentiated acute febrile illness (8.4%). (18)
In this study, the majority of AKI were caused by intrinsic renal insult (68.67%), while pre-renal factors and post-renal obstruction were responsible for 26.67% and 4.67% of the cases, respectively. Said R. reported a similar result in a study of 215 patients with AKI, in which intrinsic renal damage was responsible for 58% of cases, followed by pre-renal causes (28%) and post-renal obstruction (14%). (19) Kapadia MP et al. similarly documented intrinsic renal injury (66%) and pre-renal causes (26%) as common causes of AKI, with the remaining 8% attributable to post-renal obstruction, yielding comparable results to our study. (20)
Patients with AKI were divided into two groups: community-acquired AKI (CAAKI), those who had AKI at hospital admission, and hospital-acquired AKI (HAAKI), those who developed AKI during their course in the hospital; and compared with few key criteria. Analysis showed that 82.7% of patients had CAAKI, while the remaining 17.3% developed HAAKI, with male patients constituting the majority in both groups. The comparatively greater frequency of CAAKI in the current study could be attributed to the lower incidence of HAAKI as a result of the current advanced healthcare system and the availability of appropriate treatment modalities. Biradar et al. study also showed a similar frequency of 78.7% of CAAKI and 21.3% of HAAKI. (17)
Complete recovery from AKI was seen in 60% of

AKI patients, partial recovery in 36%, death in 3.3%, and 0.7% (n=1) was RRT dependent at discharge. Overall favorable outcome was seen in HAAKI, with complete recovery seen in 76.9% than CAAKI (56.5%). Partial recovery of renal function by discharge occurred in 23.1% of the HAAKI group as compared to 38.7% in CAAKI. There were no deaths in the HAAKI group as compared to 5 deaths in CAAKI (4%). In contrast to our findings, a study from Central India had a mortality rate of 51% in HAAKI patients compared to 20% in CAAKI patients. (2) Interestingly, in a study from the United Kingdom, the mortality among HAAKI was higher (62.9%) than that in the CAAKI group (45%).

In the current study, only 12 patients (8%) required RRT. Among the 12 patients, two expired (16.7%), one had complete recovery (8.3%), eight had partial recovery at the time of discharge (66.7%), and only one required RRT even at the time of discharge (RRT-dependent). Recent research on 500 patients with AKI found that 25% of the patients required RRT, with 30% of CAAKI patients and 19% of HAAKI patients requiring it. (2)

Summarising, our study addresses the clinical spectrum and etiology of AKI in the ICU setting of a tertiary care hospital in South India, compares the characteristics of CAAKI with HAAKI, and analyses the outcome of AKI with respect to its etiology. It is important to look into the etiology and clinical profile of critically ill AKI patients, which would be helpful in guiding us to plan strategies to prevent AKI, triage the patients, and prioritize the utilization of sparse and expensive therapeutic modalities. (16) Sensitising the clinicians on the judicial use of nephrotoxic drugs with vigilant monitoring to prevent AKI, is also important.

The limitation of this study is that the patients were followed up only till discharge, hence, the long-term residual effect of the disease on renal function is not known. Secondly, as this study was done in an ICU of a tertiary care hospital, there could be an inherent referral bias, hence, the data may not truly reflect that of an entire developing country. Since this study was done in a Medical ICU, fair representation of surgical and obstetric AKI patients is lacking.

Conclusion

In our study, AKI was more common in the male population, with diabetes and hypertension as the predominant comorbidity. Sepsis was the most common etiology of AKI, followed by nephrotoxins, which can be reduced with adequate renal protective measures and early intervention. Community-acquired AKI was more common than hospital-acquired one. There was an increased requirement for RRT in CAAKI when compared to HAAKI. The overall mortality among AKI patients was 3.3% and the outcome was better in HAAKI than CAAKI, with complete recovery seen in 76.9% of patients.

Table 1. Causes of acute kidney injury in the Intensive Care Unit

Etiology	Frequency	Percentage (%)
Volume loss/hypoperfusion	39	26
- Septic shock	4	2.6
- Cardiac	4	2.6
- Hepatorenal	1	0.6
- Acute gastroenteritis	20	13.3
- Dengue shock	5	3.3
- Acute pancreatitis	4	2.6
- Upper gastrointestinal bleed	1	0.6
Acute tubular necrosis	84	56
- Sepsis	36	24
- Nephrotoxin	36	24
- Rhabdomyolysis	1	0.6
- Accelerated hypertension	4	2.6
- Snake bite	1	0.6
- Malaria	1	0.6
- Multiple myeloma	2	1.3
- Paraquat poisoning	2	1.3
- Tumour lysis syndrome	1	0.6
Acute interstitial nephritis	18	12
- Scrub typhus	4	2.6
- Leptospirosis	3	2
- Pyelonephritis	11	7.3
Urinary tract obstruction	7	4.7
Glomerulonephritis	2	1.3

Table 2. The outcome of CAAKI patients in the Intensive Care Unit

Outcome		Clinical entities of AKI		Total
		CAAKI	HAAKI	
Death	Count	5	0	5
	%	4.0	0.0	3.3
Partial recovery	Count	48	6	54
	%	38.7	23.1	36.0
Complete recovery	Count	70	20	90
	%	56.5	76.9	60.0
RRT dependent	Count	1	0	1
	%	0.8	0.0	0.7
Total	Count	124	26	150
	%	100.0	100.0	100.0

Legend: CAAKI=community-acquired acute kidney injury; AKI=acute kidney injury; HAAKI=hospital-acquired acute kidney injury; RRT=renal replacement therapy.
Chi square=4.218, p-value=0.239.

Table 3. The outcome of acute kidney injury based on the anatomical classification

Outcome		Cause of AKI based on the anatomical classification			Total
		Post-renal	Pre-renal	Renal	
Death	Count	0	3	2	5
	%	0.0	7.5	1.9	3.3
Partial recovery	Count	5	7	42	54
	%	71.4	17.5	40.8	36.0
Recovery	Count	2	30	58	90
	%	28.6	75.0	56.3	60.0
RRT dependent	Count	0	0	1	1
	%	0.0	0.0	1.0	0.7
Total	Count	7	40	103	150
	%	100.0	100.0	100.0	100.0

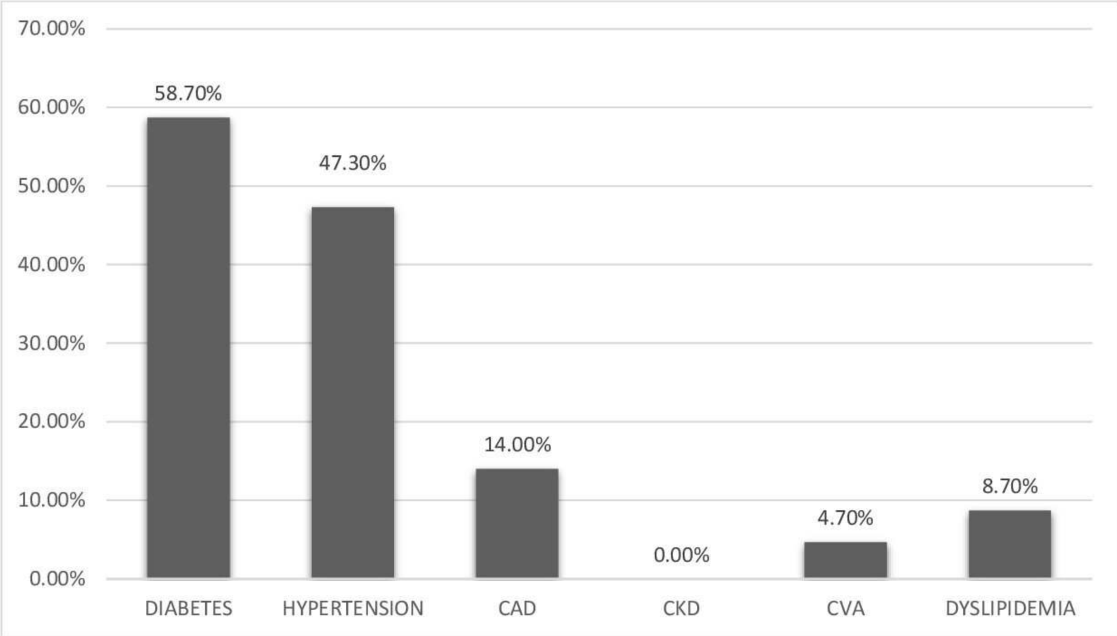
Legend: AKI=acute kidney injury; RRT=renal replacement therapy.
Chi square=13.154, p-value=0.041.

Table 4. The outcome of acute kidney injury in patients who required renal replacement therapy

Outcome		RRT needed		Total
		No	Yes	
Death	Count	3	2	5
	%	2.2	16.7	3.3
Partial recovery	Count	46	8	54
	%	33.3	66.7	36.0
Complete recovery	Count	89	1	90
	%	64.5	8.3	60.0
RRT dependent	Count	0	1	1
	%	0.0	8.3	0.7
Total	Count	138	12	150
	%	100.0	100.0	100.0

Legend: RRT=renal replacement therapy.
Chi square=27.667, p-value=0.001.

Figure 1. Distribution of co-morbidities among the AKI subjects



Legend: AKI=acute kidney injury; CAD=coronary artery disease; CKD=chronic kidney disease; CVA=cerebro vascular accident.

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