

Clinical spectrum and intermediate outcomes of community and hospital-acquired acute kidney injury: A single centre study

NAVEEN KUMAR MATTEWADA, DHANIN PUTHIYOTTIL, SREEJITH PARAMESWARAN, P.S. PRIYAMVADA

ABSTRACT

Background. There is minimal literature on the spectrum and long-term outcomes of acute kidney injury (AKI) from tropical countries.

Methods. Patients with AKI without underlying chronic kidney disease (CKD), were recruited from March 2017 to December 2018 to assess their outcomes. Survivors were followed for a year post-discharge. A linear model with fixed effects was created to compare the estimated glomerular filtration rate (eGFR) trajectories of patients with and without CKD at the end of follow-up.

Results. A total of 529 patients with AKI were recruited, of which 288 (54.4%) were hospital-acquired AKI. Infections and sepsis were the most common aetiologies for community-acquired AKI and hospital-acquired AKI. The overall mortality rate was 42.9% ($n=227$). The ICU stay (HR 1.78; 95% CI 1.08–2.93), mechanical ventilation (HR 1.98; 95% CI 1.09–3.54), and the requirement for inotropic support (HR 2.36; 95% CI 1.65–3.39) were independent risk factors of in-hospital mortality. Among 156 subjects with long-term follow-up, 70 (44.9%) developed CKD after a median follow-up of 12 months. Age ($p<0.001$) and hospital-acquired AKI ($p=0.014$) were significant predictors, whereas ICU stay and comorbid conditions did not influence the GFR trajectories. CKD patients showed a lower eGFR from the first follow-up ($p<0.001$).

Conclusions. AKI is associated with significant mortality. Even after an apparent recovery, around half the survivors progress to CKD at the end of 1 year.

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INTRODUCTION

Acute kidney injury (AKI) is a major public health concern, with a steadily increasing prevalence in recent decades. Its aetiology in low and middle-income countries (LMIC) is different from that

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of high-income countries (HIC).^{1,2} Even though 85% of the world's population resides in LMICs, data on the epidemiology and AKI outcomes from these areas are relatively scanty.³ The absence of national registries and non-uniform diagnostic criteria often results in a significant under-reporting of AKI events. Using the Kidney Disease: Improving Global Outcomes (KDIGO) or equivalent criteria, the pooled AKI estimates in LMICs are increasing and approaching those of the HICs.^{3,4} Patients from LMICs are often younger, with higher relative proportions of community-acquired AKI (CAAKI). The majority do not have diabetes, hypertension, or cardiovascular risk factors, which would make them susceptible to AKI. Major inequalities exist in access to healthcare, and late presentation to healthcare systems are common. Only minimal data exist on the prevalence and causes of hospital-acquired AKI (HAAKI) in tropical countries. Hospital-acquired AKI is less common than CAAKI and is associated with lower mortality. The long-term consequences of surviving an AKI in the tropics remain primarily unknown.

India is the most populous country globally, with a high AKI burden. The aetiologic profile also shows a wide variation across urban and rural areas.^{5–7} Even though conditions like acute diarrhoeal disease and cortical necrosis have decreased over the past few decades, infectious causes and envenomation remain important causes of AKI. Most data on the AKI spectrum in India stems from multiple single-centre studies. There is considerable heterogeneity in the epidemiology of AKI from different parts of India. Even in the same geographic region, important differences exist between urban and rural areas.^{5–10}

We studied the aetiology, in-hospital outcomes, and follow-up kidney function of AKI patients admitted to a tertiary care centre catering to a predominantly rural population.

METHODS

The study was done in a tertiary care centre in southern India. The recruitment period was from March 2017 to December 2018. All patients with AKI for whom Nephrology consultations were sought were enrolled, subject to the exclusion criteria. Patients with pre-existing chronic kidney disease (CKD) (estimated glomerular filtration rate [eGFR] <60 ml/minute/1.73m², urine albumin excretion >30 mg/day, or urine protein $\geq+$ on urine dipstick), glomerular disease, renovascular, or interstitial disease were excluded. Patients who did not have a baseline eGFR value were excluded if the ultrasound showed a bipolar length of the kidney <9 cm, loss of cortico-medullary differentiation, and cysts in the kidney. All survivors were called for follow-up visits at 2 weeks, 3 months, and 1 year after discharge from the

hospital. The demographic parameters, biochemical parameters, disease severity scores, and clinical details of AKI were recorded at initial hospitalization. The treatment decisions regarding dialysis were as per the physician's discretion. The three follow-up visits assessed the GFR using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) creatinine-based equation. A dipstick examination for urine albumin and blood pressure was recorded on each visit.

Case definitions

AKI was defined according to KDIGO 2012 guidelines. CAAKI was defined as AKI on admission or developing within 48 hours of entry to the hospital. HAAKI was defined as AKI occurring after 48 hours of hospitalization in a patient with baseline normal kidney function. For patients with CAAKI, any eGFR value in the 3 months preceding the onset of the illness was considered baseline. For HAAKI patients, eGFR on admission was considered the baseline. Delayed recovery (DR) was defined as eGFR values <60 ml/minute/1.73m² on the first follow-up visit, 2 weeks post-discharge. Sepsis was defined as per the third international consensus guidelines for sepsis.¹¹ CKD was defined as eGFR <60 ml/minute/1.73m² on the last visit. Adverse endpoints were defined as CKD, new-onset albuminuria, and new-onset hypertension. Ethical approval was obtained to establish the cohorts. Informed consent was obtained from all participants before recruitment.

Statistical analysis

All categorical variables were expressed as frequencies and percentages. The continuous variables were presented as mean with 95% confidence intervals (95% CI) or the median with interquartile range (IQR), depending on the distribution. The continuous variables between independent groups were compared using the Student *t*-test or the Mann-Whitney U test, depending on the normality of the data. The Chi-square test was used to compare the categorical variables. A Cox regression model was created to assess the independent predictors of in-hospital mortality. A linear mixed model with covariate adjustment was used to compare the temporal changes in eGFR among those with and without CKD at one year. All statistical analyses were done at a 5% significance level, and $p < 0.05$ was considered significant. The data were analysed with IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY: IBM Corp.

RESULTS

A total of 529 patients with AKI were recruited; 285 (53.5%) had KDIGO stage 3 AKI, 185 (23.6%) had stage 2, and 121 (22.9%) had stage 1; 288 (54.4%) had HAAKI. The AKI stages of patients with HAAKI and CAAKI have been given in Table 1. Most subjects were from rural areas (441; 83.4%). ICU admission was required in 329 (62.2%). Comorbid conditions were present in 148 (28%). A total of 195 (37.5%) required renal replacement therapy. The predominant comorbid conditions were hypertension (76, 14.4%), diabetes (72, 13.6%), and coronary artery disease (37, 7.0%). Baseline creatinine was 0.84 mg/dl (95% CI 0.82–0.85; available for 342 patients).

Aetiology of AKI

The predominant aetiologies responsible for hospitalization in the entire cohort were infections (147, 27.7%) and sepsis (36, 6.8%), together contributing 34.5% cases (Table 2). The most common aetiology for sepsis/infections was cellulitis and necrotizing soft tissue infections (62, 11.7%). Of these 62 individuals with skin and soft tissue infections, only 13 had diabetes.

The majority had more than one risk factor for AKI; the predominant risk factors included nephrotoxic medications (211, 39.9%) and hypoperfusion/hypotension (150, 28.4%). The nephrotoxic medications were NSAIDs and aminoglycosides administered at peripheral centres before the referral. A total of 114 (21.5%; 101 HAAKI and 13 CAAKI) patients underwent major surgical procedures (elective surgeries [61], emergency surgeries [44], cardiac surgeries [9]). During the hospital stay, catheter-associated urinary tract infections were documented in 65 (12.3%), and bloodstream infections in 45 (8.5%).

In-hospital outcomes

The HAAKI patients were sicker, had more comorbid conditions, and needed life support and ICU admissions. AKI was less severe in patients with HAAKI. The baseline characteristics of the CAAKI and HAAKI cohorts have been given in Table 1. The overall mortality was 42.9% ($n=227$). The characteristics of survivors and non-survivors have been given in Table 3. The mortality was higher for HAAKI ($n=145/288$) compared with CAAKI ($n=82/241$); unadjusted OR 1.96 (95% CI 1.38–2.79). ICU admissions, mechanical

TABLE 1. Clinical and biochemical characteristics of the acute kidney injury cohort

Parameter	Entire cohort ($n=529$)	CAAKI ($n=241$)	HAAKI ($n=288$)	p value
Median age (IQR)	49.7 (48.3, 60)	50 (48.1, 51.8)	49.3 (47.4, 51.2)	0.619
Male gender, n (%)	322 (60.9)	158 (65.3)	164 (57.3)	0.073
ICU admission, n (%)	329 (62.2)	123 (50.8)	206 (71.8)	<0.001
Vasopressors, n (%)	224 (42.3)	73 (30.2)	151 (52.6)	<0.001
Mechanical ventilation, n (%)	258 (42.8)	53 (22.3)	205 (71.1)	<0.001
Major surgical procedure, n (%)	114 (22.5)	13 (05.4)	101 (35.1)	<0.001
Comorbid conditions, n (%)	148 (27.9)	42 (17.4)	106 (36.8)	<0.001
Nephrotoxic medications, n (%)	211 (39.9)	39 (16.2)	172 (59.7)	<0.001
Median peak creatinine, mg/dl (IQR)	3.00 (1.80, 5.75)	4.77 (2.73, 7.27)	2.10 (1.50, 3.40)	<0.001
<i>Acute kidney injury stage, n (%)</i>				
1	121 (22.9)	27 (11.2)	94 (32.6)	
2	185 (23.6)	42 (17.4)	83 (28.8)	<0.001
3	283 (53.5)	172 (71.4)	111 (38.5)	
Renal replacement therapy, n (%)	195 (37.3)	123 (51)	72 (25)	<0.001

CAAKI community-acquired acute kidney injury HAAKI hospital-acquired acute kidney injury ICU intensive care unit

TABLE 2. Aetiology of acute kidney injury

Aetiology	n (%)
Infections	147 (27.7)
Sepsis	36 (6.8)
Cellulitis and NSTIs	62 (11.7)
Tropical infections–malaria/dengue/leptospirosis/others	31 (5.9)
Pneumonia	18 (3.4)
Urinary sepsis	11 (2.1)
Tuberculosis	12 (2.3)
Malignancy	82 (15.5)
Acute abdominal conditions (Appendicitis, cholecystitis, perforations, intestinal obstruction, etc.)	66 (12.5)
Acute pancreatitis	21 (4.0)
Snake envenomation	44 (8.3)
Poisoning	13 (2.4)
Cardiac causes	41 (7.8)
Obstructive uropathies	28 (5.3)
Obstetric causes	5 (0.9)
Diarrhoea	4 (0.7)

NSTI necrotizing soft tissue infection

ventilation, and need for inotropes were independent risk factors of in-hospital mortality (Table 4).

Follow-up

Among the 302 survivors, 180 came for the first follow-up visit scheduled 2 weeks post-discharge (median duration 24 days from AKI onset (IQR 20.3, 30 days). Among the 180 patients, 17 (9.4%; 10 with CAAKI, 7 with HAAKI) died in the first year following discharge. The kidney function had improved in all of them at discharge. Eight deaths occurred in patients with advanced malignancies. The cause of death was not evident in 9 patients, as it happened outside the hospital.

We had data for 156 patients beyond 3 months (135 finished all follow-up visits, and 21 patients were lost after 3 months of

follow-up). Among the 156 survivors (101 with CAAKI, 55 with HAAKI), infections ($n=41$, 26.3%), envenomation ($n=27$, 17.3%), surgical AKI ($n=24$, 15.4%), and obstructive uropathies ($n=16$, 10.3%) accounted for the majority. Recurrent AKI during the same hospitalization occurred in 4 patients. After a few months of recovery from the initial AKI, 2 patients had a repeat episode of AKI.

A total of 87/156 patients (55.7%) developed adverse kidney endpoints after a median follow-up of 12 months (IQR 12, 12; Table 5). Among the 10 patients with end-stage renal disease (ESRD), 5 did not recover from the initial insult and remained dialysis-dependent from the beginning. We did kidney biopsies on 3 patients with snake envenomation, suspecting cortical necrosis. Two patients had evidence of acute tubular necrosis, and 1 of thrombotic microangiopathy. The eGFR categories at the end of follow-up among the survivors have been shown in Fig. 1. The presence of HAAKI (33/55; 60%) was associated with higher odds of CKD compared to CAAKI (38/101; [37.6%]; OR 2.48; 95% CI 1.29–4.88). Those who progressed to CKD were older, with more comorbid conditions and ICU stay. Rather than the severity of kidney failure or need for dialysis, a lower eGFR on the first follow-up predicted CKD at 1 year (Table 6). A linear model with fixed effects was created to compare the eGFR trajectories of patients with and without CKD at the end of follow-up. Age, comorbid conditions, type of AKI, and ICU stay were incorporated into the model. Age ($p<0.001$) and HAAKI ($p=0.014$) were significant predictors, whereas ICU stay and comorbid conditions did not influence the eGFR trajectories. The adjusted model showed that those who developed CKD had lower eGFR from the first follow-up (Fig. 2); the recovery from the initial AKI episode was incomplete, with subsequent progression to CKD.

DISCUSSION

Our study comprised 529 patients with AKI, predominantly from rural backgrounds. In contrast to the existing data from

TABLE 3. Characteristics of survivors and non-survivors

Parameter	Survivors (n=302)	Non survivors (n=227)	p value
Median age (IQR)	50 (40,62)	50 (36,62)	0.739
Male gender, n (%)	182 (62.1)	141 (60.3)	0.367
ICU admission, n (%)	124 (41.1)	205 (90.3)	<0.001
Hypotension, n (%)	54 (17.9)	170 (74.9)	<0.001
Renal replacement therapy, n (%)	82 (27.2)	113 (49.8)	<0.001
Mechanical ventilation, n (%)	79 (26.2)	179 (78.9)	<0.001
Comorbid conditions, n (%)	86 (28.5)	62 (27.3)	0.845
Infections*, n (%)	108 (36.8)	111 (47.6)	<0.001
Malignancy, n (%)	24 (7.9)	51 (22.5)	<0.001
Community-acquired acute kidney injury	82 (34)	159 (66)	<0.001
Hospital-acquired acute kidney injury	145 (50.3)	143 (49.7)	
Acute kidney injury stage 3, n (%)	162 (53.3)	121 (53.6)	1.00
Both urine output and creatinine criteria, n (%)	154 (51.0)	164 (72.2)	<0.001
	Median (IQR)	Median (IQR)	
Albumin	3.0 (2.6, 3.6)	2.9 (2.4, 3.3)	0.113
Peak creatinine	2.9 (1.9, 5.2)	3.1 (1.8, 6.2)	0.429
Haemoglobin (g/dl)	10.6 (8.9, 12.1)	10.7 (8.9, 12.3)	0.659
White cell count (cmm)	11 460 (7504, 16 352)	12 390 (6960, 17 730)	0.430
Platelet count ($\times 10^9$)	2.12 (1.29, 2.68)	2.06 (1.29, 2.68)	0.837
Albumin (g/dl)	3.0 (2.5, 3.6)	2.9 (2.4, 3.4)	0.113
Aspartate aminotransferase (i.u./L)	25 (41, 77)	52 (28.7, 103.5)	0.012
Alanine aminotransferase (i.u./L)	29 (18, 54)	29 (20, 67.7)	0.208

* includes subjects admitted with sepsis and infections and hospital-acquired infections ICU intensive care unit

India, our study showed a higher prevalence of HAAKI, >50% of all AKIs. HAAKI is considered uncommon in India as well as

TABLE 4. Independent risk factors of mortality

Parameter	Hazard ratio (95% CI)
Mechanical ventilation	1.78 (1.08, 2.93)
Hypotension	2.36 (1.65, 3.39)
ICU admission	1.98 (1.09, 3.54)
Malignancy	1.24 (0.89, 1.73)
Infections	1.00 (0.76, 1.03)
Renal replacement therapy	1.03 (0.72, 1.45)
HAAKI	1.03 (0.72, 1.46)
Both creatinine and urine output criteria	1.26 (0.86, 1.83)

ICU intensive care unit HAAKI hospital-acquired acute kidney injury

TABLE 5. Adverse endpoints at the end of the follow-up* (n=156)

Category	n (%)
Chronic kidney disease	70 (44.8)
New onset albuminuria	4 (2.5)
New onset hypertension	(1 with eGFR >60 ml/minute/1.73 m ²)
	20 (12.8)
	(16 with eGFR >60 ml/minute/1.73 m ²)

* 87 patients had one or more adverse endpoints. 7 patients had more than one adverse endpoint eGFR estimated glomerular filtration rate

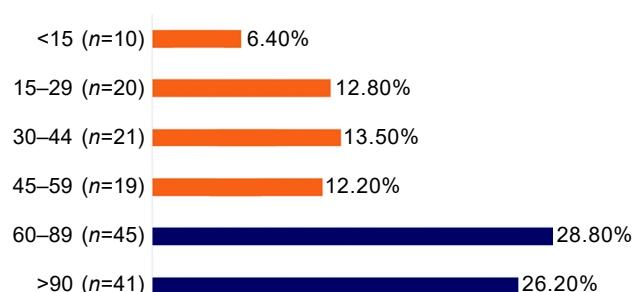


FIG 1. Estimated glomerular filtration rate (eGFR; ml/minute/1.73 m²) categories among survivors at the end of 1 year

TABLE 6. Baseline characteristics of individuals with and without chronic kidney disease on follow-up (n=156)

Parameter	No CKD (n=86)	CKD (n=70)	p value
Age	44.3 (32.7, 52.6)	55.3	<0.001
Gender	51 (59.3)	37 (52.9)	0.516
<i>Comorbid conditions*</i>	10 (11.8)	32 (35.5)	
Systemic hypertension	09 (10.4)	16 (22.8)	0.023
Diabetes mellitus	03 (03.4)	19 (27.1)	<0.001
Coronary disease	03 (03.4)	05 (07.0)	0.470
Stroke	01 (01.2)	01 (01.4)	1.00
ICU	28 (32.6)	30 (42.9)	0.002
Vasopressors	09 (10.5)	15 (21.4)	0.590
Mechanical ventilation	15 (17.6)	13 (19.4)	0.782
Acute kidney injury stage 3	49 (57)	46 (65.7)	0.266
Renal replacement therapy	28 (32.9)	22 (31.4)	0.841
eGFR <60 ml/minute/1.73 m ² on first follow-up	22 (25.5)	53 (75.7)	<0.001
	Median (IQR)	Median (IQR)	
Hospital stay	10 (6, 17)	10.5 (7, 16)	0.807
Baseline creatinine	0.8 (0.7, 0.95)	0.78 (0.85, 0.92)	0.372
Peak creatinine	3.6 (1.8, 6.6)	2.9 (2.1, 8.4)	0.446
Creatinine on first follow-up	1.02 (0.80, 1.28)	1.77 (1.33, 2.7)	<0.001
eGFR on first follow-up	77.6 (71.8, 83.4)	40.7 (35.2, 46.1)	<0.001

* more than one comorbid conditions in some patients, total numbers will not add up

other LMICs. Previous studies from different parts of India reported prevalence rates varying from 3.4% to 15.7%.^{5-7,10} HAAKI may often be underdiagnosed, especially in surgical settings where a marginal rise in creatinine or drop in urine output can be easily overlooked.¹² In our study, the median creatinine levels in the HAAKI group were much lower compared to CAAKI. Most previous studies from India have used a serum creatinine-based definition for AKI, which might lead to underdiagnosis of HAAKI. We believe that applying KDIGO criteria resulted in higher detection rates of AKI. As we recruited patients referred to nephrology, a referral bias between medical and surgical specialties could also be a factor. Approximately 70% of subjects of HAAKI in our study were admitted to ICU, which would have resulted in closer monitoring of urine output, kidney function, and resultant early referrals.

The predominant aetiologies responsible for hospitalization were problems specific to the tropics. However, a few major differences in epidemiology were observed compared to data from other parts of India. Sepsis has been reported as the dominant cause of AKI in a few centres in India.^{6,8,10,13} Others reported more classical tropical causes, such as acute diarrhoeal disease, envenomation, cortical necrosis, and infections like malaria, leptospirosis, and scrub typhus, as the leading

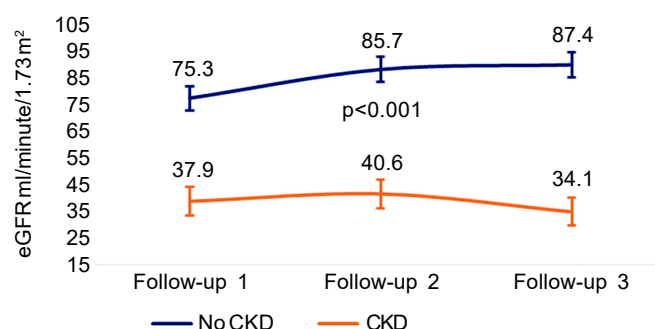


FIG 2. Age-adjusted estimated glomerular filtration rate (eGFR) trajectories over one year for those with and without chronic kidney disease (CKD)

ICU intensive care unit eGFR estimated glomerular filtration rate

causes.^{5,7,9,10} Even though sepsis/infections were the dominant cause, the spectrum was dominated by necrotizing soft tissue infections occurring in non-diabetic individuals. The rural community, whose livelihood depends on farming, the hot and humid climate, and the delay in accessing healthcare facilities, may contribute to the high prevalence of skin/soft tissue infections. Soft tissue infection-related sepsis as the predominant cause for AKI was reported in another study from Tamil Nadu.¹³ Envenomation/toxins were a major cause of AKI, similar to that reported by Jayakumar *et al.*⁹ and Vairakkani *et al.*,¹³ from adjacent geographic areas. Paraquat was the most common toxin responsible for AKI, with a 90% case fatality rate. Even though around 40% of patients had a history of use of nephrotoxic medication, it could not be considered AKI's sole cause. Most received it at the primary point of referral when other risk factors coexisted. AKI's contribution due to diarrhoea, malaria, typhus, dengue, and leptospirosis was significantly less. The AKI spectrum shows considerable variations across India, possibly due to region-specific infections, accessibility to healthcare systems, and referral policies of individual institutions.

The occurrence of AKI due to obstetric causes has steadily declined in India, but still accounts for around 1%–5% of all AKIs.^{14,15} Major regional variations in incidence are also seen. Despite being a predominantly rural population, the obstetric causes were considerably less, possibly due to referral bias. Our catchment area has a well-developed three-tier system for antenatal care, with most pregnancies being booked. Milder AKIs are often managed in secondary and tertiary care centres; those requiring life or organ support are only referred to higher centres. Internal referral bias might have contributed, as intensivists manage many obstetric AKIs, and only patients seeking a nephrology referral were included. The relatively lower proportion of diarrhoeal AKIs in our study also attests to good primary care facilities available for the public. Acute surgical conditions and malignancies were responsible for a high proportion of AKI, hitherto unreported from other single-centre studies from India.^{13,16} Even though HAAKI was more common, the demographics and epidemiology appeared different from the data from developed and other developing countries.^{1,17} Unlike the data from developed countries, where cardiac surgery is the most common cause of AKI, in our study, the majority underwent non-cardiac surgeries, mostly due to malignancies and emergencies.

Even though around 40% of patients had a history of use of nephrotoxic medications, the same could not be considered AKI's sole cause. Most of them received it at the primary point of referral when other risk factors for AKI also coexisted. The contribution of AKI due to diarrhoea, malaria, typhus, dengue, and leptospirosis was significantly less in our study.

The mortality of AKI reported from Indian studies ranges from 9% to 74%.^{7,10,13,18–20} The relatively higher mortality in our study might be due to a higher proportion of ICU patients. The mortality is comparable with data from other Indian centres (40%–47%) with similar proportions of patients who need life and organ support.^{6,7,13} Unlike other Indian studies on AKI, ours has a higher proportion of HAAKI; this might be another potential reason for a higher mortality. There is only minimal data on HAAKI from India; the reported mortality rates are around 40%–45%, whereas the mortality reported from general medical wards is much lower.^{20–22} CAAKI appeared to be associated with lower mortality compared to HAAKI, despite

having more patients with higher stages of AKI. CAAKI is reported to be associated with better in-hospital survival compared to HAAKI, despite having a more severe renal injury.^{23,24} There are only limited data comparing the outcomes of HAAKI and CAAKI from LMIC countries. A recent study from Brazil reported higher mortality rates for patients who acquire AKI in the ICU but not in the wards. Two recent meta-analyses confirmed a better outcome with CAAKI, despite having comparable kidney failure and dialysis needs.^{25,26} The higher mortality rates in HAAKI result from severe organ failure needing intensive care and life support rather than the severity of kidney failure per se.²⁵ We also observed that HAAKI is associated with higher mortality on univariate analysis. After adjusting for other covariates, ICU admissions, mechanical ventilation, and inotropic support were the independent determinants of mortality.

The post-discharge mortality in our study was 10%. The mortality rates tend to be higher following the recovery of AKI, with the reported mortality rates varying from 18% to 28%.^{27,28} In individuals who survive a critical illness complicated by AKI, the follow-up mortality rates can be as high as 60%.²⁹ The post-discharge mortality rates appear lower, possibly due to the younger age of the population, fewer comorbid conditions, different epidemiologic profiles, and considerable loss of follow-up.³⁰ To our knowledge, no data exist on post-discharge AKI mortality rates in India. In addition to increasing short-term mortality, AKI is associated with future progression to CKD, ESRD, and adverse cardiovascular outcomes. Meta-analyses have reported 23.4–25.8 CKD events/100 person-years in those who sustain AKI.^{31,32} However, the studies included in those meta-analyses did not represent the AKI profile in LMICs (most studies were from Europe and North America). The study population comprised predominantly those undergoing cardiovascular surgeries or AKI in selected subjects. The long-term outcomes of AKI in the tropics are hypothesized to be better than in the developed world due to the more favourable demographic profile: younger individuals with less comorbid conditions.³ However, only minimal data are available from LMICs to justify this conclusion. Follow-up studies are often limited to AKI resulting from a single aetiology. The reported CKD prevalence among snake envenomed patients is 22%–41%.³³ Another Sri Lankan study reported that 9% of subjects with leptospiral AKI progress to CKD.³⁴ In our study, 45% of subjects developed CKD on follow-up. Two-thirds of those who progressed to CKD had no pre-existing comorbid conditions and were at least a decade younger than the data from the developed world. The CKD prevalence in our study is somewhat higher than the reports from HICs.^{31,32} The higher CKD progression rates might have resulted from the referral nature of the centre, and receiving sicker patients with severe renal failure. A recent study from the state of Tamil Nadu reported that 20% of AKI survivors progress to higher stages; however, the proportion of patients who needed dialysis was considerably lower. The higher CKD prevalence is a matter of concern because only a quarter of the follow-up cohort had underlying risk factors. This underscores the importance of long-term periodic follow-up after AKI, which seldom happens in LMICs. We also observed that progression to CKD following envenomation was somewhat lower than other aetiologies. Schiffel *et al.* reported a similar observation: critically ill patients with nephrotoxic acute tubular necrosis (ATN) tend to recover better than ischaemic or mixed ATN.³⁵

We observed that rather than the severity of AKI, the recovery of kidney function acted as a determinant of long-term outcomes. Those who had a GFR >60 ml/minute/1.73 m² on the first follow-up visit had lower chances of progression to CKD at 1 year. There is accumulating evidence that initial renal recovery is an important predictor of long-term kidney function.^{36–38} The risk of developing CKD is the lowest when it recovers within 48 hours, whereas 58% progress to CKD once recovery is delayed beyond 30 days.³⁸ The prevalence of new-onset albuminuria was very low in our study. Recent literature highlights the role of increased urinary protein excretion after an apparent recovery of AKI. It has been reported that there is a 23% higher chance of worsening proteinuria following one year of AKI. In addition to the GFR, proteinuria after AKI is an independent contributor to disease progression.^{39,40} However, in those studies, 40%–60% of AKI survivors had pre-existing proteinuria at the time of admission with AKI. We excluded patients with baseline albuminuria and believe this might account for the low prevalence of proteinuria in our study.

The strengths of our study include prospective data collection from the time of follow-up until the end of 1 year. To our knowledge, no prior studies from India have assessed the long-term outcomes of mixed medical–surgical AKIs. The limitations include considerable loss of follow-up, especially for those with milder AKIs. There are limitations in extrapolating the data to the entire cohort, as those with milder AKI stages did not come for follow-up. Most patients were from rural areas with low connectivity; hence, logistical issues might stand in the way of scheduled follow-up visits. The study entry criteria and the intra-hospital referral policies might have influenced the aetiologic spectrum in our study. The inclusion criteria were set as patients with AKIs seeking nephrology referral. So, milder AKI cases managed by intensivists and physicians might have been excluded; this might account for the under representation of obstetric AKIs and pyelonephritis. Despite excluding patients with renal parenchymal and renovascular disorders, 44% showed evidence of CKD at 1 year. However, it might not be prudent to extrapolate these findings at community levels, as only 50% of the cohort turned up for follow-up.

We do not have biopsy data on all patients who progressed to CKD. As per the inclusion criteria, we excluded those with glomerular and interstitial pathology, so biopsies were not mandated at the time of diagnosis of AKI. We biopsied 3 patients with snake envenomation who did not recover, suspecting cortical necrosis. One patient had thrombotic microangiopathy, and the other had ATN. Those who eventually developed CKD showed partial improvement in kidney function at discharge; biopsies were not performed as the clinical setting was ATN. However, the renal parameters did not touch baseline on 1- and 3-month visits. We discussed the feasibility of biopsy in patients who did not show complete recovery, but patients did not consent, as it might not contribute to a change in treatment decisions. We do not have data on multidrug-resistant organisms contributing to sepsis. Baseline creatinine measurements were not available for all patients. Because of the poor socioeconomic conditions, people are not sensitized and seldom undergo periodic health check-ups. We attempted to exclude underlying CKD by ultrasound imaging for those without baseline kidney function. However, despite the best efforts, those with early CKD may still have been included. As subjects seeking a nephrology referral were only included, the recruitment is biased towards sicker patients with

multiorgan involvement. We believe this might partially be responsible for the relatively high mortality and CKD rates. The study is not sufficiently powered to examine whether the aetiology of AKI has any bearing on long-term outcomes.

Conflicts of interest. None declared

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