

Predictors of Mortality in Acute Kidney Injury Patients Admitted to Medicine Intensive Care Unit in a Rural Tertiary Care Hospital

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Abstract

Background: Acute kidney injury (AKI) is a challenging problem faced by intensive care clinicians worldwide, and it is associated with high morbidity and mortality, especially in critically ill patients. **Materials and Methods:** A hospital-based prospective, observational study was conducted in patients of AKI admitted to the Intensive Care Unit (ICU) of the Department of Medicine in a rural tertiary care hospital located in central India. Data of all consecutive AKI inpatients related to demographic variables, clinical profile, and laboratory investigations were collected from patient's medical records. **Results:** Of the total 229 AKI patients enrolled in this study, 65 (28.4%) patients died during their hospital stay. The presence of metabolic acidosis, hypotension, Glasgow coma scale (GCS) and Acute Physiologic Assessment and Chronic Health Evaluation (APACHE 2) score, advanced AKI stage, higher serum creatinine and blood urea levels on diagnosis of AKI and the peak rise in their level within 48 h of diagnosis of AKI, the use of mechanical ventilator, leukocytosis, and hyperkalemia were significantly associated with in-hospital mortality in AKI patients ($P < 0.05$). **Conclusion:** The overall in-hospital mortality in patients of AKI admitted to medicine-ICU was 28.4%. Sepsis was the most common cause of AKI (24.5%). The presence of metabolic acidosis, hypotension, GCS and APACHE 2 score, advanced AKI stage, higher serum creatinine, and blood urea levels on diagnosis of AKI and the peak rise in their level within 48 h of diagnosis of AKI, use of mechanical ventilator, leukocytosis, and hyperkalemia were associated with in-hospital mortality in AKI patients.

Keywords: Acute kidney injury, etiology, incidence, mortality, predictors

INTRODUCTION

Acute kidney injury (AKI) is characterized by rapid decline in kidney function over a period of hours to days resulting in retention of metabolic waste products.^[1,2] AKI is a challenging problem in low-resource settings due to the high burden of infectious diseases, diarrheal illnesses, snake bites, the over-the-counter availability of potentially nephrotoxic drugs, and medicinal herbs.

The previous studies showed that 3%–7% of hospitalized patients and 25%–30% of patients in the Intensive Care Unit (ICU) develop AKI, with 5%–6% of the ICU population requiring renal replacement therapy after developing AKI.^[3-5] AKI in ICU is associated with high mortality, longer hospital stay, and substantial health resource utilization.^[6,7] In addition, the previous studies have revealed that patients who recover from AKI are at increased risk of progressive chronic kidney disease (CKD) and development of end-stage renal disease (ESRD).^[8-10]

The identification of factors associated with mortality among patients hospitalized for AKI would help in establishing interventions or preventive measures that could improve survival in AKI patients. In view of the limited data, on hospitalized AKI patients, from resource-limited countries and its likely importance, this study was planned to assess the clinical profile and factors affecting the mortality in AKI patients in our setting, i.e., a rural tertiary care hospital located in central India.

MATERIALS AND METHODS

A prospective, observational study was conducted in patients of AKI admitted to the ICU of the Department of medicine in a

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rural tertiary care hospital located in a town in central India over a period of 18 months from November 1, 2014, to April 30, 2016.

Inclusion criteria

All consecutive patients of AKI, 18 years of age or older admitted to medical ICU with AKI, or developed AKI after hospitalization and fulfill the Kidney Disease Improving Global Outcomes definition.^[11] For this study, AKI is defined as an acute increase in serum creatinine by ≥ 0.3 mg/dL within 48 h, an increase of 1.5-fold or more in baseline serum creatinine level within the previous 7 days, or reduction in urine output < 0.5 mL/kg/h for 6 h.

Exclusion criteria

Patients with CKD or ESRD, patients with age < 18 years, withdrawal of treatment during the ICU stay, discharge against medical advice, AKI patients with incomplete data and AKI patients who did not wish to participate were not included in this study.

Demographic variables (age, gender, socioeconomic status, and education), clinical profile, diagnosis, comorbidities (hypertension [HTN] and diabetes mellitus [DM], ischemic heart disease [IHD]), alcohol intake, smoking history, the presence of hypotension, use of mechanical ventilator, the presence of sepsis, need for hemodialysis during hospitalization, urine output (oliguric or nonoliguric), outcomes (death/survived), and laboratory investigations-serum creatinine 1: serum creatinine on diagnosis of AKI, serum creatinine 2: difference between peak value of serum creatinine (within 48 hours of diagnosis of AKI) and serum creatinine on diagnosis of AKI, blood urea 1: blood urea on diagnosis of AKI, blood urea 2: difference between peak value of blood urea (within 48 hours of diagnosis of AKI) and blood urea on diagnosis of AKI, sodium, potassium, hemoglobin, total leukocyte count, and the presence of metabolic acidosis were collected from medical records of the AKI patients. Oliguria was considered to be present when the urinary volume was < 400 mL/day. ICU scores-Acute Physiologic Assessment and Chronic Health Evaluation (APACHE 2) score and Glasgow coma scale (GCS) scores of the AKI patients were also recorded. Sepsis was defined as two or more of the following four conditions: (1) Fever (oral temperature $> 38^{\circ}\text{C}$) or hypothermia ($< 36^{\circ}\text{C}$); (2) tachypnea (> 24 breaths/min); (3) tachycardia (heart rate > 90 beats/min); and (4) leukocytosis ($> 12,000/\mu\text{L}$), leukopenia ($< 4000/\mu\text{L}$), or $> 10\%$ bands, has a proven or suspected microbial etiology and sequential organ failure assessment score of ≥ 2 .

AKI was further classified into three stages according to the severity of kidney injury.

1. AKI Stage 1: increase in serum creatinine by ≥ 0.3 mg/dL or 1.5–1.9 times baseline
2. AKI Stage 2: increase in serum creatinine by 2.0–2.9 times baseline
3. AKI Stage 3: increase in serum creatinine of ≥ 3.0 times baseline or serum creatinine more than 4 mg/dL or initiation of renal replacement therapy.

In patients with shock or hypotension, point-of-care ultrasonography including rapid ultrasound in shock was used to look for inferior vena cava collapsibility, focused cardiac ultrasound with bedside clinical, and hemodynamic signs were used to assess volume status of the patients. We used crystalloid solution (normal saline) for fluid resuscitation.

We used sustained low-efficiency dialysis in few hemodynamically unstable AKI patients. We did not use continuous renal replacement therapy in any patient, as this facility is not available in our hospital.

Patient follow-up

The AKI patients were prospectively, monitored up to hospital discharge on a regular basis. At discharge, they were classified as nonsurvivors or survivors. Renal recovery in this study means dialysis independence at the time of hospital discharge and return of serum creatinine to within 20% of preAKI creatinine/baseline creatinine.

Statistical analysis used

Patients were categorized as survivors or nonsurvivors, and variables (demographic, clinical, and biochemical variables) were compared using bivariate analysis. Statistical analysis was performed using descriptive and inferential statistics. The Chi-squared test was used for the comparison of proportions of categorical variables and *z*-test for the difference between two means. Data were analyzed using the SPSS software 17.0 version (SPSS South Asia Private Limited, Bangalore, Karnataka, India). A value of $P < 0.05$ was considered as statistically significant.

RESULTS

We enrolled a total of 229 patients of AKI over an 18-month period. Three thousand five hundred eighty-four patients admitted to medicine ICU during this period. A total of 245 patients developed AKI during this period, out of which 16 patients were excluded as per the study exclusion criteria. The mean age was 49.76 ± 17.01 years ranging from 18 to 92 years. There were 36 (15.7%) AKI patients below the age of 30 years, 122 (53.3%) AKI patients between 30 and 60 years, and 71 (31%) AKI patients were above the age of 60 years [Table 1]. Of the total 229 patients of AKI, 71 (31%) were male, and 158 (69%) were female [Table 1]. The association of age of patients (< 30 years, 30–60 years, and > 60 years) and gender of the patients with the in-hospital mortality was not statistically significant ($P > 0.05$) [Table 1].

Of the 229 AKI patients, 86 (37.6%) had HTN, 63 (27.5%) had DM, and 43 (18.8%) had IHD. The association of comorbidities (HTN, DM, and IHD) with in-hospital mortality was not statistically significant [Table 2].

There were 34 (14.8%) current smokers and 41 (17.9%) alcohol drinkers of the total 229 AKI patients. The association of in-hospital mortality with either current smoking or alcohol drinking was not statistically significant [Table 2].

Table 1: Distribution of demographic variables among acute kidney injury patients with in-hospital mortality

Demographic variables	Nonsurvivors (n=65), n (%)	Survivors (n=164), n (%)	Total (n=229), n (%)	P
Age-groups (years)				
<30	10 (15.4)	26 (15.9)	36 (15.7)	0.75
30-60	37 (56.9)	85 (51.8)	122 (53.3)	
>60	18 (27.7)	53 (32.3)	71 (31)	
Gender				
Male	21 (32.3)	50 (30.5)	71 (31)	0.78
Female	44 (67.7)	114 (69.5)	158 (69)	

Figures in parenthesis represent the column percentage

Table 2: Distribution of comorbidities/habituall history among acute kidney injury patients with in-hospital mortality

Comorbidities/habituall history	Nonsurvivors (n=65), n (%)	Survivors (n=164), n (%)	Total (n=229), n (%)	P
HTN				
Present	25 (38.5)	61 (37.2)	86 (37.6)	0.85
Absent	40 (61.5)	103 (62.8)	143 (62.4)	
DM				
Present	18 (27.7)	45 (27.4)	63 (27.5)	0.96
Absent	47 (72.3)	119 (72.6)	166 (72.5)	
IHD				
Present	9 (13.8)	34 (20.7)	43 (18.8)	0.22
Absent	56 (86.2)	130 (79.3)	186 (81.2)	
Alcohol intake				
Present	9 (13.8)	32 (19.5)	41 (17.9)	0.31
Absent	56 (86.2)	132 (80.5)	188 (82.1)	
Current smokers				
Present	11 (16.9)	23 (14)	34 (14.8)	0.57
Absent	54 (83.1)	141 (86)	195 (85.2)	

Figures in parenthesis represent the column percentage. HTN: Hypertension; DM: Diabetes mellitus; IHD: Ischemic heart disease

Of the 229 AKI patients, 80 (34.9%) had Stage 1 AKI, 98 (42.8%) had Stage 2 AKI, and 51 (22.3%) had Stage 3 AKI [Table 3]. The association of staging of AKI patients (severity) with the in-hospital mortality was found to be statistically significant ($P < 0.05$) [Table 3].

One hundred and sixteen (50.7%) of 229 patients had oliguria. Hypotension was present in 90 AKI patients (39.3%). One hundred and seventy-six (76.9%) had metabolic acidosis. The invasive mechanical ventilator was used in 70 AKI patients (30.6%). Hemodialysis was done in 71 AKI patients (31%) [Table 4].

The association of urine output and the use of hemodialysis in AKI patients with in-hospital mortality was not statistically significant ($P > 0.05$) [Table 4]. The presence of hypotension and the use of an invasive mechanical ventilator in AKI patients were found to be significantly associated with in-hospital mortality ($P < 0.05$) [Table 4]. The mean number of hemodialysis sessions done in our AKI patients was 4.4 ± 1.3 (ranges from 2 to 8).

Fifty-three (23.1%) of the 229 AKI patients had GCS < 10 and 176 (76.9%) had GCS > 10 [Table 5]. Forty-four patients (19.2%) had APACHE 2 score ≤ 15 , 128 patients (55.8%) had APACHE 2 score between 16 and 30, and 57 patients

Table 3: Distribution of staging of acute kidney injury (severity) among acute kidney injury patients with in-hospital mortality

Stages of AKI	Nonsurvivors (n=65), n (%)	Survivors (n=164), n (%)	Total (n=229), n (%)	P
Stage 1	7 (10.8)	73 (44.5)	80 (34.9)	0.001
Stage 2	35 (53.8)	63 (38.4)	98 (42.8)	
Stage 3	23 (35.4)	28 (17.1)	51 (22.3)	

Figures in parenthesis represent the column percentage. AKI: Acute kidney injury

(25%) had APACHE 2 score ≥ 31 [Table 5]. Both GCS and APACHE 2 score were found to be significantly associated with in-hospital mortality ($P < 0.05$).

The mean serum creatinine value was 3.10 ± 1.69 and 3.82 ± 2.01 mg/dL among survivors and nonsurvivors, respectively. The mean blood urea was 101.83 ± 70.31 and 123.53 ± 73.86 mg/dL among survivors and nonsurvivors, respectively [Table 6].

Serum creatinine on diagnosis of AKI, peak rise in serum creatinine within 48 h of diagnosis of AKI, blood urea on diagnosis of AKI, and peak rise in blood urea within 48 h of diagnosis of AKI, and bicarbonate (HCO_3^-) levels were found to

Table 4: Distribution of clinical variables/interventions used among acute kidney injury patients with in-hospital mortality

Clinical variables/interventions used	Nonsurvivors (n=65), n (%)	Survivors (n=164), n (%)	Total (n=229), n (%)	P
UO				
Oliguric	40 (61.5)	76 (46.3)	116 (50.7)	0.11
Nonoliguric	25 (38.5)	88 (53.7)	113 (49.3)	
Hypotension				
Present	49 (75.4)	41 (25)	90 (39.3)	0.0001
Absent	16 (24.6)	123 (75)	139 (60.7)	
Invasive mechanical ventilator				
Used	49 (75.4)	21 (12.8)	70 (30.6)	0.0001
Not used	16 (24.6)	143 (87.2)	159 (69.4)	
Hemodialysis				
Used	22 (33.8)	49 (29.9)	71 (31)	0.55
Not used	43 (66.2)	115 (70.1)	158 (69)	

Figures in parenthesis represent the column percentage. UO: Urine output

Table 5: Distribution of Intensive Care Unit scores among acute kidney injury patients with in-hospital mortality

ICU scores	Nonsurvivors (n=65), n (%)	Survivors (n=164), n (%)	Total (n=229), n (%)	P
GCS score				
<10	30 (46.2)	23 (14)	53 (23.1)	0.001
11-15	35 (53.8)	141 (86)	176 (76.9)	
APACHE 2 score				
0-15	1 (1.5)	43 (26.2)	44 (19.2)	0.0001
16-30	22 (33.9)	106 (64.6)	128 (55.8)	
>31	42 (64.6)	15 (9.2)	57 (25)	

Figures in parenthesis represent the column percentage. ICU: Intensive Care Unit; GCS: Glasgow Coma Scale; APACHE: Acute Physiologic Assessment and Chronic Health Evaluation

Table 6: Comparison of laboratory parameters among survivors and nonsurvivors

Laboratory parameters	Status	n	Mean±SD	SEM	Z	P
Serum creatinine 1	Nonsurvivors	65	3.82±2.01	0.24	2.73	0.007
	Survivors	164	3.10±1.69	0.13		
Serum creatinine 2	Nonsurvivors	65	0.90±1.42	0.17	3.34	0.001
	Survivors	164	0.40±1.20	0.09		
Blood urea 1	Nonsurvivors	65	123.53±73.86	8.72	2.03	0.043
	Survivors	164	101.83±60.31	5.76		
Blood urea 2	Nonsurvivors	65	36.27±68.52	8.49	3.21	0.002
	Survivors	164	12.15±53.95	5.77		
Serum bicarbonate	Nonsurvivors	65	16.34±4.83	0.60	2.83	0.005
	Survivors	164	18.19±4.31	0.33		
Hemoglobin	Nonsurvivors	65	10.99±2.79	0.34	0.10	0.91
	Survivors	164	10.94±3.32	0.25		
Serum sodium	Nonsurvivors	65	132.03±9.99	1.036	0.24	0.67
	Survivors	164	131.6±9.24	0.948		

Serum creatinine 1: Serum creatinine on diagnosis of AKI; Serum creatinine 2: difference between peak value of serum creatinine (within 48 hours of diagnosis of AKI) and serum creatinine on diagnosis of AKI; blood urea 1: blood urea on diagnosis of AKI; blood urea 2: difference between peak value of blood urea (within 48 hours of diagnosis of AKI) and blood urea on diagnosis of AKI n: Number of AKI patients; SD: Standard deviation; SEM: Standard error of mean; AKI: Acute kidney injury

be significantly associated with in-hospital mortality ($P < 0.05$) [Table 6]. The mean HCO_3^- levels were 18.2 ± 4.3 and 16.3 ± 4.8 mEq/L among survivors and nonsurvivors, respectively [Table 6]. The mean Hb level was 10.9 g/dl (± 3.17) in both the survivors and nonsurvivors group. Hemoglobin of AKI patients was not related to in-hospital mortality ($P > 0.05$) [Table 6].

Hyperkalemia and leukocytosis were present in 66 AKI patients (28.8%) and 156 AKI patients (68.1%), respectively. Both hyperkalemia and leukocytosis were significantly associated with in-hospital mortality ($P < 0.05$) [Table 7]. The mean serum potassium level of AKI patients having hyperkalemia was 6.08 (± 0.65) which is mild (5.5–6.5 mEq/L).

Table 7: Distribution of laboratory parameters among acute kidney injury patients with in-hospital mortality

Laboratory parameters	Nonsurvivors (n=65), n (%)	Survivors (n=164), n (%)	Total (n=229), n (%)	P
Hyperkalemia				
Present	25 (38.5)	41 (25)	66 (28.8)	0.043
Absent	40 (61.5)	123 (75)	163 (71.2)	
Leukocytosis				
Present	54 (83.1)	102 (62.2)	156 (68.1)	0.002
Absent	11 (16.9)	62 (37.8)	73 (31.9)	

Figures in parenthesis represent the column percentage

The most common cause of AKI in our medicine ICU patients was sepsis (24.5%) followed by snake bite (16.2%), acute gastroenteritis (12.6%), cirrhosis (7%), congestive cardiac failure (5.2%), malaria (4.7%), CV stroke (4.4%), obstructive uropathy (4.4%), acute respiratory distress syndrome (3.5%), sickle cell disease (3.1%), dengue (3.1%), organophosphorus poisoning (2.6%), acute pancreatitis (2.2%), contrast-induced AKI (1.7%), scrub typhus (1.3%), HELLP syndrome (1.3%), hemolytic uremic syndrome (0.9%), bee sting (0.9%), and eclampsia (0.4%) [Table 8].

The primary source of sepsis was lung in 26 (46.4%) AKI patients followed by intraabdominal infections in seven (12.5%) patients, central nervous system infections in five (8.9%) patients, cellulitis in four (7.1%) patients, UTI in three (5.4%) patients, and unknown source in 11 (19.6%). Of the total 37 snake bite AKI patients, 29 had features of vasculotoxicity, five have features of both vasculotoxicity and neurotoxicity and three had features of neurotoxicity only. Of the vasculotoxic bites, mostly are attributed to Russell's viper bites (90%) and rest were due to saw scaled viper bites. This conclusion was based on the information obtained from description of the snakes by the patients or witnesses, the actual identification of the dead snake, and the clinical course. Of the total 11 malaria AKI patients, eight patients had falciparum malaria, two had mixed falciparum with vivax malaria, and one had vivax malaria.

Of the total 229 AKI patients enrolled in this study, 65 patients (28.4%) died during their hospital stay. AKI patients with sepsis had the highest in-hospital mortality (35.4%).

DISCUSSION

In this study, the overall in-hospital mortality among medicine ICU patients with AKI was 28.4%. AKI patients having sepsis had the highest in-hospital mortality (35.4%). In previous studies, a mortality rate of 35.9%, 38.5%, and 58% was reported by Peres *et al.*, Oluseyi *et al.*, and Hamzić-Mehmedbašić *et al.*, respectively.^[12-14] The variation in the mortality rate observed in these studies may be explained by the difference in the study populations, ICU settings, cause of ICU admission and underlying diseases, or by the nonuniform criteria used for defining AKI in different studies.

The association of age and gender with the in-hospital mortality was not found to be statistically significant in this study, which is in contrast to finding reported by Oluseyi

Table 8: Etiology wise distribution of acute kidney injury patients

Etiology	Frequency, n (%)
Sepsis	56 (24.5)
Snake bite	37 (16.2)
Acute gastroenteritis	29 (12.6)
Cirrhosis	16 (7)
CCF	12 (5.2)
Malaria	11 (4.7)
CV stroke	10 (4.4)
ARDS	8 (3.5)
Sickle cell disease	7 (3.1)
Obstructive uropathy	10 (4.4)
Dengue	7 (3.1)
OPP	6 (2.6)
Acute pancreatitis	5 (2.2)
Contrast - induced	4 (1.7)
Scrub typhus	3 (1.3)
HELLP	3 (1.3)
HUS	2 (0.9)
Bee sting	2 (0.9)
Eclampsia	1 (0.4)

n: Number of AKI patients; CCF: Congestive cardiac failure; ARDS: Acute respiratory distress syndrome; OPP: Organophosphorus poisoning; HUS: Hemolytic uremic syndrome; AKI: Acute kidney injury

et al., Poukkanen *et al.*, Kohli *et al.*, and Shiao *et al.*, where older age was associated with an increased mortality.^[13,15-17] Hamzić-Mehmedbašić *et al.* reported that female gender was related to increased mortality.^[14]

In this study, the association of comorbidities, i.e., HTN, DM, and IHD with in-hospital mortality was not statistically significant. DM and increased number of comorbidities were found to be related to mortality in AKI patients in the previous studies done by Sezer *et al.* and Samimagham *et al.*^[18,19]

The staging of AKI (severity) was related to in-hospital mortality in this study. Majority of the AKI patients among the nonsurvivors were having Stage 2 and 3 AKI. Similarly, the previous studies done by Oluseyi *et al.* also reported that advanced AKI or late presentation of AKI was related to increased mortality.^[13] Similar findings were also reported by Aboisaf *et al.* and Ostermann and Chang.^[20,21]

The presence of hypotension and the use of invasive mechanical ventilator in AKI were associated with mortality in this study.

This association might also reflect severity of illness in subset of our patients having other organ system failure leading to mortality. This finding is comparable with other studies done by Peres *et al.*, Krishnamurthy *et al.*, Liaño and Pascual, Uchino *et al.*, Kaul *et al.*, and Shiao *et al.*^[12,17,22-25]

GCS scores and APACHE 2 scores were associated with in-hospital mortality in our AKI patients which is consistent with the results of the previous studies done by Pan *et al.*, Lin *et al.*, Sezer *et al.*, Samimaghani *et al.*, Shiao *et al.*, and Friedericksen *et al.*^[17,18,26-28]

“Serum creatinine and blood urea levels on diagnosis of AKI” and “peak rise in creatinine and urea levels within 48 h of diagnosis of AKI” were associated with in-hospital mortality in our AKI patients. Similarly, Chertow *et al.* reported that modest changes in serum creatinine concentration were significantly associated with mortality.^[29] Serum creatinine >4 mg/dL was associated with increased in-hospital mortality in a study by Kaul *et al.*^[25] This finding is also in line with the results of Wilson *et al.* and Peres *et al.*^[12,30] The metabolic acidosis was associated with in-hospital mortality in AKI patients in this study. The AKI patients in this study having severe metabolic acidosis (≤ 7 mEq/L) had 2.4 times more risk of mortality in comparison to AKI patients having HCO₃⁻ levels in normal range. Metabolic acidosis was related to in-hospital mortality in studies done by Ostermann and Chang, and Peres *et al.*^[12,21]

Hyperkalemia and leukocytosis were related to in-hospital mortality in AKI patients in this study. It may be explained by the association of hyperkalemia and leukocytosis with an advanced AKI and sepsis, respectively, in our AKI patients. Hyperkalemia and the presence of sepsis were related to increased mortality in a study done by Dela Cruz *et al.*^[31]

The association of in-hospital mortality with the presence of albuminuria and oliguria was not significant in this study. The presence of oliguria was related to increased mortality in AKI patients in the previous studies reported by Kaul *et al.*, Liaño and Pascual, Dela Cruz *et al.* and Obialo *et al.*^[23,25,31,32] Similarly, oliguria was found to be an independent predictor of mortality or poor outcome in studies done by Teixeira C *et al.*, and de Mendonca *et al.*^[33,34]

CONCLUSION

The overall in-hospital mortality in patients of AKI admitted to medicine-ICU was 28.4%. Sepsis was the most common cause of AKI (24.5%). AKI patients having sepsis had the highest in-hospital mortality (35.4%). The presence of metabolic acidosis, hypotension, GCS and APACHE 2 score, advanced AKI stage, higher serum creatinine and blood urea levels on diagnosis of AKI and the peak rise in their level within 48 h of diagnosis of AKI, use of mechanical ventilator, leukocytosis, and hyperkalemia were associated with in-hospital mortality in AKI patients.

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Conflicts of interest

There are no conflicts of interest.

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