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Acute kidney injury after successful cardiopulmonary resuscitation; risk factors and prognosis: a retrospective cross-sectional study

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Abstract: **Objective:** Acute kidney injury (AKI) is an independent risk factor in critically ill patients. This study aimed to evaluate the prevalence of AKI in resuscitated cardiac arrest (CA) patients, its potential risk factors, and outcomes of AKI in cardiac arrest survivors.

Methods: A hundred and forty-nine cases of post-CA patients that survived for at least 24 hours, admitted to three hospitals between 2016 and 2020, were studied. AKI was defined by the RIFLE (Risk, Injury, Failure, Loss, and End-stage) criteria. Baseline demographic data, resuscitation variables, the prevalence of AKI, in-hospital and six-month mortality were collected. Logistic regression evaluated the factors associated with AKI occurrence and mortality.

Results: AKI occurred in 59 (39.6%) of the patients. Of these, 9 patients (15.3%) required renal replacement therapy (RRT) during their hospital stay. There were 47 (52.2%) in-hospital deaths in patients without AKI and 41 (69.5%) in patients with AKI (P=0.036). Post-CA AKI was significantly associated with six-month mortality (OR=1.65; 95% CI: 1.39,2.88; P=0.029). Older age, the higher cumulative dosage of epinephrine during cardiopulmonary resuscitation, post-CA shock, in-hospital CA, initial pulseless electrical activity (PEA) or asystole rhythm, longer duration of cardiac arrest, as well as higher admission creatinine and lactate levels were independently associated with AKI; in contrast, higher admission base excess level was negatively associated with AKI.

Conclusion: AKI occurred in nearly 40% of CA patients. AKI was associated with a higher in-hospital and six-month mortality rates.

Keywords: Acute Kidney Injury; Cardiac Arrest; Cardiopulmonary Resuscitation; Emergency Department

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1. Introduction

Despite significant improvements in post-cardiac arrest (CA) care, mortality rates still remain high after sudden CA, even in patients with return of spontaneous circulation (ROSC), following cardiopulmonary resuscitation (CPR) (1,2). CA patients must receive immediate CPR to achieve ROSC. These patients will eventually die because of post-CA shock and brain injury (3). Post-CA syndrome can be caused by post-CA ischemia-reperfusion injury, including multiple organ failure (MOF) (3,4).

In particular, brain injury has been recognized as the major cause of post-CA death and disability (5). However, less attention has been paid to the prevalence and prognostic value of extra-cerebral organ dysfunctions.

Acute kidney injury (AKI) has been reported as an independent risk factor for mortality in critically ill patients during their intensive care unit (ICU) stay (6). AKI may occur in 12%-81% of cardiac arrest survivors and becomes clinically evident median 1-2 days after the CA (7,8). About 4-33 % of patients with AKI will need renal replacement therapy (RRT) during their hospital stay, but only a small proportion of them will be RRT dependent at 30 days after their CA (5,6).

The AKI occurrence is related to the previous renal disease

and post-CA hypoperfusion (9). In most patients, however, it is not yet clear whether AKI is due to systemic or renal hypoperfusion, circulating nephrotoxins, or additional insults (9). AKI is transient in most patients, but in some cases persistent AKI occurs (10). Knowing about these predictions of persistent AKI in resuscitated CA patients could help to eliminate or modify risk factors and allocate appropriate resources for hemodialysis in these patients.

The current study aimed to evaluate the prevalence of AKI in resuscitated CA patients, as well as its potential predictors, and outcomes of AKI among CA survivors.

2. Methods

2.1. Study design and setting

This multi-center retrospective, cross-sectional study performed in 3 referral hospitals in Isfahan, Iran, from January 1, 2016, to December 31, 2020. The Ethics Committee of Isfahan University of Medical Sciences approved the study (ID: IR.MUI.MED.REC.1399.129), and informed consents were waived, because the study was retrospective and observational. Cardiopulmonary resuscitation (CPR) and post-CA care were done according to the guidelines of the American Heart Association (AHA).

2.2. Study population

Adult patients (age ≥18 years old), who achieved ROSC after in-hospital CA (IHCA) or out-of-hospital CA (OHCA) were eligible. Inclusion criteria included the patients who survived for at least 24 hours after ROSC, and had at least four recorded levels of serum creatinine (Cr). Exclusion criteria were pregnancy, transfer from another hospital, missing data, AKI before the occurrence of CA in IHCA, receiving vasoactive agents before CA, and history of chronic renal failure. Given the prevalence of 50% for AKI in a previous study (11), the estimation error of 10%, and the 95% confidence interval (CI), 97 cases were needed.

2.3. Definitions

Cardiac arrest was defined as cardiorespiratory collapse and abrupt loss of heart function, pulse, and spontaneous circulation. CA could be caused by cardiac arrhythmia such as ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), pulseless electrical activity (PEA), or asystole rhythm.

AKI was defined by the RIFLE (Risk, Injury, Failure, Loss, and End-stage) criteria (12). The RIFLE criteria classify AKI into three groups (Risk, Injury, and Failure) based on relative serum Cr level and urine output (Table 1). The most recent serum Cr level from medical records within one year of the CA was used as the baseline. If no previous records were available, the admission serum Cr level was used as the baseline for patients with OHCA and IHCA. The post-resuscitation shock was defined as vasoactive drugs infusion for more than 6 hours to stabilize patient's hemodynamics, despite adequate fluid loading (6).

2.4. Data collection

The patient demographics and resuscitation variables were recorded. Baseline demographic characteristics included age, sex, drug history (beta-blockers, angiotensin-converting enzyme inhibitors (ACEs), and angiotensin receptor blockers (ARBs)), smoking, and underlying diseases such as diabetes mellitus, cardiac diseases, hypertension, end-stage renal disease, hyperlipidemia, and prior cerebrovascular disease or stroke. Resuscitation variables included primary monitored rhythm of CA, the presence of witnesses, IHCA or OHCA, the total dosage of epinephrine received during resuscitation, time interval from collapse to chest compression, and duration of CPR (time from chest compression to ROSC). In addition, data on AKI occurrence and in-hospital mortality rate were collected. Serum level of Cr, hemoglobin (Hb), sodium (Na), potassium (K), serum lactate, serum bicarbonate (HCO3-), and base excess (BE), were measured at the time of admission to the emergency department. Also, Serum Cr levels were investigated at 12, 24, 48 and 72 hours after admission. Serum Cr level changes were not taken into account in this study. However, the diagnosis of AKI at any time during hospitalization was considered, and this is the reason for the repeated creatinine level measurements. The medical records and telephone follow-ups provided statistics on mortality, and survival rates.

2.5. Statistical analysis

IBM SPSS software version 22.0, was used to analyze the data. The categorical data were reported as numbers and percentages, and the chi-square test was used to compare groups. The continuous variables were declared as means with standard deviations (SD), and independent t-tests or the Mann-Whitney U test were used to compare groups. To determine the normality of continuous data, the Kolmogorov-Smirnov test was used. Predictive factors associated with AKI development were evaluated in a logistic regression analysis initially; Then variables related with a higher univariate association with AKI (P<0.1) were introduced in multivariable analysis. Odds ratios (OR) and 95% confidence intervals (CI) were estimated. The significance level was considered as P-value<0.05.

3. Results

3.1. Patient profiles

Among 198 patients successfully resuscitated after OHCA or IHCA, 39 were excluded, because 13 of them had missing data on serum Cr level or urine output, 10 had chronic kidney disease and 16 patients did not survive for 24 hours after resuscitation. Finally, 149 patients were included in the analysis (Figure 1).

Baseline characteristics are reported in table 2. The mean age of the patients was 61.3 ± 18.1 (23–98) years, and 103 patients were male (69.1%). Cardiac arrest occurred in hospital in 52 (34.9%) cases, and the initial rhythm of VF or pulseless VT

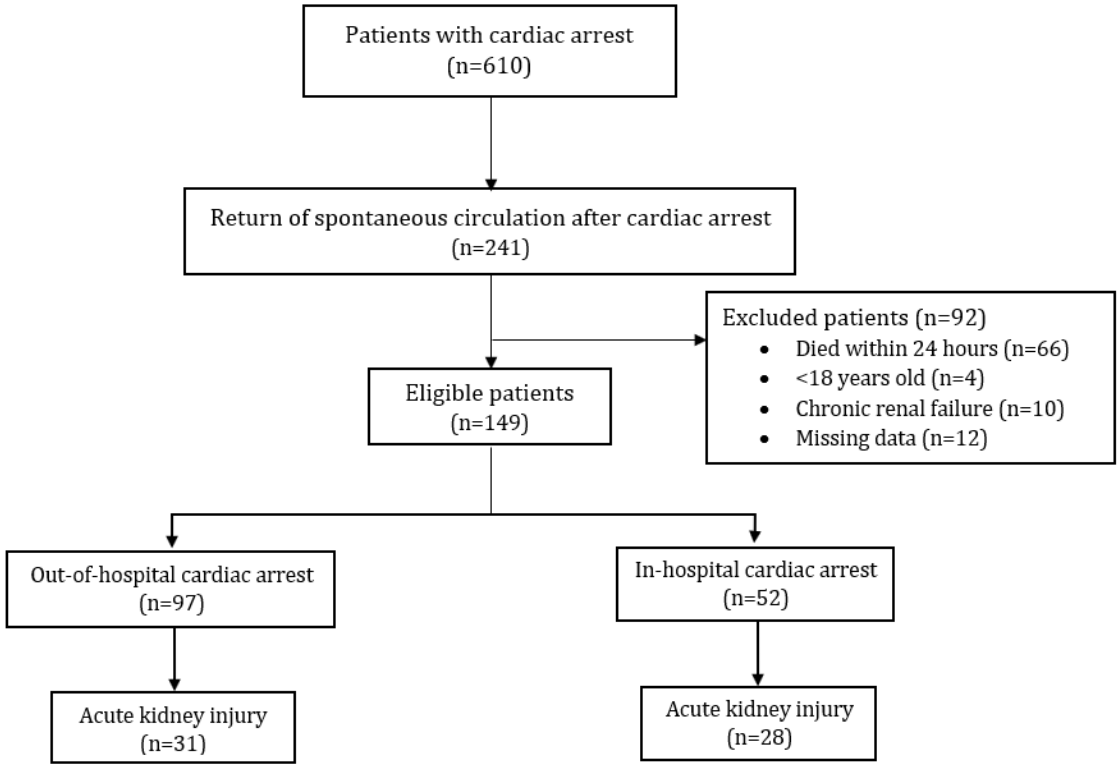

Figure 1 Study flow chart

Table 1 RIFLE Classification for acute kidney injury

Stage	Glomerular filtration rate (GFR)	Urine output
Risk	Increased serum creatinine × 1.5 or decreased GFR >25%	<0.5 mL/kg/h × 6 h
Injury (AKI)	Increased serum creatinine ×2 or decreased GFR >50%	<0.5 mL/kg/h × 12 h
Failure (ARF)	Increased serum creatinine × 3 or decreased GFR >75% or if baseline serum creatinine ≥4 mg/dL after rise of >0.5 mg/dL	<0.3 mL/kg/h × 24 h or anuria × 12 h
Loss	Complete loss of kidney function >4 weeks	
ESRD	Complete loss of kidney function >3 months	

ESRD: End-stage renal disease; ARF: Acute renal failure; AKI: Acute kidney injury; H: hour

were seen in 82 (55.0%) subjects. Post-CA shock occurred in 75 (50.3%) cases. None of the included patients underwent targeted temperature management (TTM). Overall hospital survival was 59.1%, with 52 patients (34.9%) surviving after six months.

3.2. Prevalence of AKI and renal outcome on discharge

In total, AKI class injury/ failure (I/F) developed in 59 of 149 patients (39.6%) after a median (first and third quartile) of 2 days (2,3), during the observation period following CA. Of these, nine patients (15.3%) required RRT during their hospital stay; all of them underwent hemodialysis. In patients who survived, the mean serum Cr level on discharge was 2.1 ± 1.7 mg/dL, and the mean difference between their discharge

Table 2 Baseline characteristics and clinical data of the patients

Variable	All patients (N= 149)	No AKI (N = 90)	AKI (N = 59)	P-value
Age (year), mean ± SD	61.2 ± 17.7	58.8 ± 16.6	64.8 ± 18.8	0.011#
≤40, n (%)	20 (13.4)	15 (16.7)	5 (8.5)	
41 to 50, n (%)	20 (13.4)	15 (16.7)	5 (8.5)	
51 to 60, n (%)	32 (21.5)	24 (26.7)	8 (13.6)	0.012
61 to 70, n (%)	29 (19.5)	15 (16.7)	14 (23.7)	
> 70, n (%)	48 (32.2)	21 (23.3)	27 (45.8)	
Gender, n (%)				
Male	103 (69.1)	57 (63.3)	46 (78.0)	0.059
Female	46 (30.9)	33 (36.7)	13 (22.0)	
Place of cardiac arrest, n (%)				
Out-of-hospital	97 (65.1)	66 (73.3)	31(52.5)	0.009
In-hospital	52 (34.9)	24 (26.7)	28 (47.5)	
Initial cardiac rhythm, n (%)				
VF/VT	82 (55.0)	56 (62.2)	26 (44.1)	0.029
Asystole/PEA	67 (45.0)	34 (37.8)	33 (55.9)	
Time from collapse to CPR (min), mean ± SD	2.5 ± 2.5	2.0 ± 1.1	3.1 ± 2.9	0.001*
Time from CPR to ROSC (min), mean ± SD	25.0 ± 14.6	22.2 ± 11.2	29.2 ± 18.1	0.004*
Total epinephrine given (mg), mean ± SD	7.2 ± 2.6	6.3 ± 2.3	8.5 ± 3.1	0.012*
ACEIs/ARBs use on admission, n (%)	45 (30.2)	25 (27.8)	20 (33.9)	0.619
Baseline co-morbidities, n (%)				
Chronic heart failure	30 (20.1)	17 (18.9)	13 (22.0)	0.158
Hypertension	71 (47.7)	40 (44.4)	31 (52.5)	0.333
Coronary artery disease	49 (32.9)	30 (33.3)	19 (32.2)	0.886
Diabetes mellitus	40 (26.8)	20 (22.2)	20 (33.9)	0.116
Others	74 (49.7)	41 (45.6)	33 (55.9)	0.215
Admission lab tests				
Creatinine level (mg/dL), mean ± SD	1.9 ±1.8	1.1 ± 0.3	2.7 ± 2.1	<0.001*
Hemoglobin (mg/dL), mean ± SD	12.1 ± 3.3	12.5 ± 3.4	11.7 ± 3.2	0.204#
Bicarbonate level (mEq/L), mean ± SD	21.8 ± 6.9	23.3 ± 7.4	20.1 ± 5.9	0.026*
Base excess (mEq/L), mean ± SD	-3.7 ± 2.3	-2.0 ± 1.9	-5.8 ±4.7	0.029#
Potassium level (mg/dL), mean ± SD	4.4 ± 0.8	4.8 ± 0.7	4.9 ± 0.8	0.895*
Sodium level (mg/dL), mean ± SD	137.1 ± 12.1	138.6 ± 5.1	134.8 ± 17.7	0.070*
Lactate level (mmol/L), mean ± SD	3.8 ± 2.1	2.1 ± 1.8	5.6 ± 4.3	0.008*
Post-cardiac arrest shock, n (%)	75 (50.3)	35 (38.9)	40 (67.8)	0.001
In-hospital mortality, n (%)	88 (59.1)	47 (52.2)	41 (69.5)	0.036
Six-month mortality, n (%)	97 (65.1)	53 (58.9)	44 (74.7)	0.049

*Analysis by Mann-Whitney U test; # Analysis by t-tests

VF: Ventricular fibrillation; VT: Ventricular tachycardia; PEA: Pulseless electrical activity; CPR: Cardiopulmonary resuscitation; ROSC: Return of spontaneous circulation; ACEIs: Angiotensin converting enzyme inhibitors; ARBs: Angiotensin II receptor blockers; AKI: Acute kidney injury; SD: Standard deviation; Min: Minutes

serum Cr and admission serum Cr levels was 0.22 mg/dL (95% CI: -0.6,0.1; P=0.237). Finally, two patients (3.4% of the AKI patients) needed RRT at the time of hospital discharge.

AKI patients were significantly older than those without AKI (P=0.011). AKI patients had more significantly higher proportion of initial asystole or PEA rhythm (55.9% vs. 37.8%; P=0.029) and IHCA (47.5% vs. 26.7%; P=0.009). Subjects who developed AKI had a longer time from collapse to chest compression (difference in means 1.11 minutes (95% CI: 0.63,2.15; P=0.001), a longer duration of CPR (difference in means 7.01 minutes (95% CI: 2.26,11.76; P=0.004), and received more epinephrine during CPR than patients without AKI (P=0.012). Subjects with AKI had higher admission serum creatinine level (P<0.001), serum lactate level (P=0.008), and had lower admission serum bicarbonate (P=0.026) and base excess levels (P=0.029), compared to patients without AKI.

3.3. Predictors of AKI

The predictors of AKI after cardiac arrest are listed in table 3. In multivariate regression analysis, older age (OR=1.02; P=0.043), higher cumulative dosage of epinephrine during CPR (OR=1.20; P=0.008), post-cardiac arrest shock (OR=2.35; P=0.011), IHCA (OR=2.48; P=0.027), initial PEA or asystole rhythm (OR=2.09; P=0.010), longer duration of cardiac arrest (OR=1.08; P=0.004), and higher admission serum Cr level (OR=10.76; P<0.001) and serum lactate level (OR=1.11; P=0.012) were independently associated with AKI class I/F,

Table 3 Predictors of acute kidney injury after cardiac arrest

Variables	Univariable analysis		Multivariable analysis	
	OR (95% CI)	P-value	OR (95% CI)	P-value
Age (year)	1.04 (1.02-1.06)	0.004	1.02 (1.00-1.04)	0.043
Female	0.421 (0.196-.0.902)	0.026	0.488 (0.231-1.034)	0.061
In-hospital cardiac arrest	2.31 (1.34-3.98)	0.012	2.48 (1.24-4.96)	0.027
Asystole/PEA rhythm	2.09 (1.07-4.08)	0.010	2.11 (1.09-4.10)	0.009
Post-cardiac arrest shock	2.62 (1.34-5.13)	0.009	2.35 (1.41-4.18)	0.011
ACEIs/ARBs use on admission	0.815 (0.364-1.826)	0.619	-	-
Chronic heart failure	1.12 (0.72-1.69)	0.211	-	-
Hypertension	1.11 (0.71-1.81)	0.345	-	-
Coronary artery disease	1.08 (0.68-1.71)	0.823	-	-
Diabetes mellitus	1.07 (0.66-1.73)	0.150	-	-
Admission creatinine level (mg/dL)	5.31 (2.58-10.92)	0.001	10.76 (4.02-18.62)	<0.001
Admission base excess level (mEq/L)	0.94 (0.89-0.99)	0.033	0.91 (0.84-0.98)	0.016
Admission serum lactate level (mmol/L)	1.12 (1.08-1.16)	0.010	1.11 (1.07-1.15)	0.012
Admission hemoglobin level (mg/dL)	0.92 (0.81-1.05)	0.207		
Admission bicarbonate level (mEq/L)	0.93 (0.87-0.99)	0.048	0.947 (0.89-1.01)	0.089
Admission potassium level (mg/dL)	1.006 (0.915-1.107)	0.894	-	-
Admission sodium level (mg/dL)	0.952 (0.897-1.010)	0.105	-	-
Total epinephrine given (mg)	1.19 (1.06-1.36)	0.004	1.20 (1.05-1.37)	0.008
Time from collapse to chest compression (min)	1.02 (1.00-1.05)	0.030	1.05 (1.01-1.09)	0.004
Duration of cardiac arrest (min)	1.12 (1.02-1.20)	0.012	1.08 (1.03-1.13)	0.004

All the statistically significant results have been highlighted by Bold
OR: Odds ratio; CI: Confidence interval; PEA: Pulseless electrical activity; ACEIs: Angiotensin converting enzyme inhibitors; ARBs: Angiotensin II receptor blockers; Min: Minutes

whereas higher admission serum BE level (OR=0.91; P=0.016) was negatively associated with AKI (Table 3).

3.4. Patients' outcomes

Finally, there were 47 (52.2%) in-hospital deaths in patients without AKI and 41 (69.5%) in patients with AKI (P=0.036). The overall six-month mortality rate was 65.1% among included patients. Six-month mortality rate was higher in cases with AKI than in patients without AKI (53/90 [58.9%] vs. 44/59 [74.7%]; P=0.049), and in univariate analysis, post-cardiac arrest AKI was also significantly associated with six-month mortality (OR=1.65; 95% CI:1.39,2.88; P=0.029). AKI subjects with and without RRT had comparable six-month mortality rate (7/9 [78.8%] vs. 37/50 [74.0%]; P=0.811).

4. Discussion

The current study evaluated the prevalence and risk factors of AKI among IHCA and OHCA survivors. We identified several predictors of AKI and investigated that AKI was an independent predictor of in-hospital and six-month mortality.

The current study showed that AKI is common in patients resuscitated from CA (39.6%). It was higher than what was investigated in previous epidemiological studies about AKI in general ICU patients (6,13,14). The incidence of AKI after CA in previous studies ranged from 12% to 81% (6-9). This can be attributed to several factors, including study population and the definition of AKI.

Similar to the current study, IHCA was shown to be an independent predictor of AKI (6,7,15). The definition of AKI across previous studies was different. In these studies, AKI was defined according to RIFLE, acute kidney injury network (AKIN), or kidney disease improving global outcomes (KDIGO) criteria. In the study by Para et al. (11), AKI was diagnosed in 66.1% of patients according to KDIGO criteria, while 46.7% according to RIFLE criteria.

In this study, 15.3% of cases with AKI needed RRT during the hospital stay, which is higher than 5.7% in the general ICU population (16). The reported need for RRT in AKI patients after CA has been 4%-33% (6,8,15). Sandroni et al. (5), in systematic review and meta-analysis, showed that 33% of patients with AKI (range from 18% to 60%) were treated with RRT. In addition, 3.4% of the AKI patients who survived to discharge required RRT, which was comparable to previous CA studies (6,15). In other studies, none of the survivors required RRT after hospital discharge (9,17,18). As the results showed, AKI was associated with the recovery of renal function in most survivors after CA. Similar results have been demonstrated in large cohort studies on AKI in general ICU patients (13,19).

AKI patients were significantly older than those without AKI, due to reduced physiological function of the kidneys. This result was similar to previous studies (6,9,11,15), but was not confirmed by some studies (7,17,18). In the current study, subjects with AKI not only had a longer time from collapse to CPR and longer duration of CPR but also had more fre-

quently post-CA shock and had received more epinephrine during CPR. Similar to the current study, the duration of CA was shown as a predictor of AKI in previous studies (5,6, 9,15). But, Kim et al. and Domanovits et al. investigated that duration of CA was not independently associated with AKI (17,18). AKI could be due to a sudden decrease in renal blood flow after cardiac arrest and could be related to the inflammatory storm caused by the massive release of pro-inflammatory cytokines. In the current study, the post-CA shock was the significant independent predictor of post-arrest AKI, as it had been in other studies (6,7,9,15). Consistent with the current study, the presence of an initial asystole/PEA rhythm, was found to be an independent predictor of AKI (5,6,9,15,18). Contrarily, Dutta et al. found that non-shockable rhythms was not associated with AKI (7). The association between total epinephrine given during CPR and post-CA AKI was shown in previous studies (9,15,17).

Admission serum Cr and lactate levels were significantly higher in AKI patients than without AKI. A meta-analysis including eight studies showed that higher admission serum Cr levels and more elevated serum lactate levels were significant predictors of AKI (5). These results were consistent with other studies (6,9,18).

Consistent with the current study, post-CA AKI was associated with increased mortality in a systematic review and meta-analysis (5). Vanston et al. (20) and Para et al. (11) investigated that AKI was independently associated with mortality after cardiac arrest. Contrarily, Yanta et al. (8) did not find any association between AKI and mortality rate. It is not clear, whether AKI is directly associated with mortality rate in patients after ROSC or if it should be considered an indicator of ischemic-reperfusion injury severity or post-CA shock.

AKI subjects with and without the need for RRT had comparable six-month mortality. Similarly, AKI with the need for RRT was not associated with poor outcomes in recent studies (6,8,15), but Nielsen et al. (21) and Ghoshal et al. (22) investigated that needing RRT in AKI patients was an independent predictor of a bad outcome.

5. Limitations

There are some limitations. Firstly, the present study was retrospective. Secondly, we used admission serum Cr level of patients who had no previous laboratory data as baseline Cr and it might not be the same as the serum level of Cr before CA. Finally, we could not evaluate the impact of potential AKI prevention strategies, such as the use of TTM on the occurrence of AKI after cardiac arrest, since patients did not undergo TTM.

6. Conclusion

This study found that AKI occurs in 39.6% of patients during the observation period following CA, and it was associated with increased in-hospital and six-month mortality rate. The risk factors for AKI include the older age, higher cumulative dosage of epinephrine during CPR, post-cardiac arrest shock, IHCA, initial PEA/ asystole rhythm, longer duration of cardiac arrest, and more elevated admission serum Cr and lactate levels. AKI is usually transient among survivors after cardiac arrest, and 15.3% of patients with AKI required RRT during their hospital stay.

7. Declarations

7.1. Acknowledgment

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7.2. Authors' contributions

FH, MNI, AAM, FFM, JS, and STH contributed to the conception, study design, and data collection and evaluation. FH and AAM contributed to the statistical analysis, and interpretation of data. FH and AAM were responsible for overall supervision. FH, AAM, and MNI drafted the manuscript, revised by FFM, JS and STH. All authors performed editing and approved the final version of this paper for submission, also participated in the finalization of the manuscript and approved the final draft.

7.3. Conflict of interest

The authors declare no conflict of interest.

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