Risk factors analysis for adverse short-term prognosis in patients with contrast-induced acute kidney injury: a retrospective study

Yunzhen Deng
The Second Xiangya Hospital of Central South University
Yi He
The Second Xiangya Hospital of Central South University
Kaiting Zhuang
The Second Xiangya Hospital of Central South University
Yanlin Wen
The Second Xiangya Hospital of Central South University
Junxiang Chen (chenjxly@csu.edu.cn)
The Second Xiangya Hospital of Central South University

Research Article

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Posted Date: May 25th, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1642251/v1

Risk factors analysis for adverse short-term prognosis in patients with contrast-induced acute kidney injury: a retrospective study

Yunzhen Deng, MS[1], Yi He, MS[1], Kaiting Zhuang, MS[1], Yanlin Wen, MS[1], Junxiang Chen, MD[1,*]

Authors' affiliations

[1] Department of Nephrology, The Second Xiangya Hospital of Central South University, No. 139 Renminzhong Road, Changsha, Hunan 410012, China

[*] **Corresponding author information:**

Junxiang Chen, MD, Department of Nephrology, The Second Xiangya Hospital of Central South University, No. 139 Renminzhong Road, Changsha, Hunan 410012, China

E-mail: chenjxly@csu.edu.cn

Abstract

Aims: We aimed to evaluate risk factors influencing adverse short-term outcomes in contrast-induced acute kidney injury (CI-AKI) patients after coronary angiography (CA) or percutaneous coronary intervention (PCI).

Materials and methods: We retrospectively collected 64 consecutive CI-AKI patients after CA/PCI procedure from January 2014 to November 2019. The clinical outcomes were in-hospital mortality and persistent renal dysfunction (RD). Univariable and multivariable analyses were used to identify the risk factors for in-hospital mortality and persistent RD.

Results: The incidence of in-hospital mortality was 7.8% in CI-AKI patients. After adjusting potential confounders, cardiogenic shock (OR=40.5, 95% CI, 4.147–395.494, P=0.001) was the independent risk factor for in-hospital mortality. Persistent RD occurred in 35 (59%) of survival patients. After adjusting potential confounders, eGFR (OR=3.553, 95% CI, 1.497–25.416, P=0.027), duration of procedure (OR=1.037, 95% CI, 1.002–1.073, P=0.038) and contrast media category (OR=7.189, 95% CI, 1.202–42.982, P=0.031) were independent risk factors for persistent RD.

Conclusion: In-hospital mortality of CI-AKI patients was associated with severe systemic hemodynamic alteration. Patients with existing renal impairment before CA/PCI were more likely to develop persistent RD, while reducing CA/PCI procedure time and use of isotonic contrast media (IOCM) might help decreasing the risk.

Keywords: Contrast-induced acute kidney injury, risk factors, short-term prognosis

Introduction

Contrast-induced acute kidney injury (CI-AKI) is regarded as one of the most serious complications after coronary angiography (CA) and percutaneous coronary intervention (PCI) (1). Generally, CI-AKI is defined as an increase of creatinine in serum by ≥ 0.3 mg/dl within 48 h, or an increase to ≥ 50% within 7 days after contrast media (CM) administration (2). Previous studies have reported a variable incidence of CI-AKI following PCI ranged from 3.3% to 14.5% (3-5). Notably, in patients with advanced chronic kidney disease (CKD), the incidence of CI-AKI can be extremely high (> 25%) (6). Mostly, renal dysfunction (RD) in patients with CI-AKI is transient and reversible (7). However, CI-AKI patients are still at great risk of developing persistent renal impairment, renal adverse events, major adverse cardiac and cerebrovascular events (MACCE) and other comorbidities (8-10). In several recent studies, CI-AKI is demonstrated to be an independent risk factor for dialysis and mortality (3, 6, 11). Therefore, it is of great importance to reveal risk factors for CI-AKI associated adverse events to establish potential preventive strategies.

Several studies have revealed that multiple risk factors are associated with CI-AKI. Tsai et al. found that ST-segment elevation myocardial infarction (STEMI) presentation (odds ratio [OR]: 2.60; 95% confidence interval [CI]: 2.53 to 2.67), severe chronic kidney disease (OR: 3.59; 95% CI: 3.47 to 3.71) and cardiogenic shock (OR: 2.92; 95% CI: 2.80 to 3.04) were independent risk factors for CI-AKI (6). Mehran et al. demonstrated that older age (>75 years), diabetes, anemia, low ejection fraction and volume depletion were risk factors for the occurrence of CI-AKI (12). Nevertheless, since most patients with CI-AKI only have transient RD, more attention should be paid on the patients with short-term adverse events including in-hospital

mortality and persistent RD. However, as far as we are concerned, limited studies have investigated risk factors influencing short-term prognosis in CI-AKI populations. In this retrospective case-control study, we analyzed the incidence of short-term adverse events in CI-AKI patients and investigated independent risk factors for in-hospital mortality and persistent RD.

Materials and methods

Study design and population

This retrospective case-control study was conducted in the Second Xiangya Hospital of Central South University. From January 2014 to November 2019, a total of 1112 adult patients underwent CA or PCI and 72 patients who developed CI-AKI were selected for the present study. The exclusion criteria were as follows: (1) end stage renal disease requiring dialysis (2 patients); (2) exposure to contrast media within 7 days before CA/PCI procedure (3 patients); (3) lack of information to determine clinical outcomes or laboratory measurement of serum creatinine before or after procedure (3 patients). All the procedures were conformed to the ethical guidelines of 1964 Declaration of Helsinki and later amendments. The ethics committee of the Second Xiangya Hospital of Central South University reviewed and approved the study protocol. Since all the data in this retrospective study were anonymous, the formal consent from the patients were waived by the ethics committee.

CI-AKI is defined as an increase of creatinine in serum by ≥ 0.3 mg/dl within 48 h, or an increase to ≥ 50% within 7 days after CM exposure. The Patients who developed CI-AKI were divided into groups based on their short-term outcomes: in-hospital mortality and survival groups; the survival patients were further divided into persistent RD and transient RD groups. The definition of persistent or transient RD was based on whether the reduction of creatinine clearance was ≥ 25% at 3 months in comparison with baseline (13, 14).

Variables and end point

Hospital's electronic database was screened to collect democratic data and laboratory measurements data. Electronic medical records were also reviewed to obtain

medical/medication history information and CA/PCI procedure details. Serum creatinine concentration was assessed the day before angiography, immediately preprocedural on day 0, and on days 1, 2, 3, 5, 10, and 30 after the procedure. Serum creatinine was measured again at 3 months for all CI-AKI patients Mehran risk score was calculated to evaluate risk stratification. Creatinine clearance was calculated as previously described via Cockroft-Gault formula. The estimated GFR (eGFR) was calculated with the Modification of Diet in Renal Disease (MDRD) and the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula. The type of contrast media consisted of Low osmolality contrast media (LOCM) and isotonic contrast media (IOCM). LOCM included iohexol, iopamidol, iopromide and ioversol, while IOCM only contained iodixanol. The short-term clinical end points were in-hospital death and persistent RD. The follow-up events were monitored and recorded carefully by trained nurses with office visits or telephone interviews, and access to hospital electronic databases.

Statistical analyses

Continuous variables were displayed as mean ± standard deviation (SD) or median with interquartile range (25th and 75th centiles) and analyzed by t test or Mann-Whitney U test as appropriate. Categorical variables were displayed as frequencies (percentages) and analyzed with Chi-square test or Fisher's exact test as appropriate. Univariate and multivariate logistic regression analyses were performed to identify risk factors for in-hospital mortality and persistent renal dysfunction. A two-sided P value <0.05 was considered to be statistically significant. IBM SPSS statistical software version 22 (SPSS Inc. Chicago, Illinois, USA) was used to perform all analysis and calculation.

Results

Clinical and procedural characteristics of population

During the period, A total of 1112 patients were investigated, and 72 consecutive CI-AKI patients were initially identified. Eight patients were excluded according to exclusion criteria. Finally, 64 patients were enrolled in further evaluation. The

incidence of CI-AKI was 5.7%.

Demographic information, clinical baseline and procedural characteristics of in-hospital mortality and survival patients were shown in Table 1. The in-hospital mortality rate was 7.8% in CI-AKI patients. Generally, there was no significant difference in demographic characteristics, medical history, renal function, blood lipid, inflammation indicators, medication history and procedural characteristics among each group. Nevertheless, systolic blood pressure, platelets and CK-MB was significantly different among in-hospital mortality and survival groups (P=0.032, 0.028 and 0.006 respectively). The percentages of patients with cardiogenic shock and cardiac arrest were also significantly higher in in-hospital mortality group (P=0.002 and 0.045 respectively).

Persistent RD cases (n=35) and transient RD cases(n=24) accounted for 59% and 41% in survival patients respectively. Demographic information, clinical baseline and procedural characteristics of persistent and transient RD groups were shown in Table 2. Demographic characteristics, laboratory measurements, medical history, clinical presentation and medication history were generally well-balanced across groups. However, patients who developed persistent RD were more likely to have diabetes mellitus, worse baseline renal function, higher level of NT-proBNP and lower hemoglobin. The Mehran risk score was significantly higher in persistent renal dysfunction group(P=0.004). As for CA/PCI procedure, the volume of contrast media (P=0.035) and time of procedure (P=0.001) was significantly higher. As for contrast media category, there was also significant difference among two groups (P=0.016).

Risk factors for in-hospital mortality

Univariate logistic regression analysis identified that cardiac arrest, cardiogenic shock, CK-MB, platelets and prior COPD might serve as possible risk factors. After adjusting potential confounding risk factors, multivariate logistic regression analysis revealed that cardiogenic shock may serve as an independent risk factors for in-hospital mortality (OR=40.5, 95% CI, 4.147-395.494, P=0.001). The results of logistic regression were shown in Table 3.

Risk factors for persistent RD

Univariate logistic regression analysis identified that eGFR, duration of procedure, contrast media volume, category of contrast media, Mehran risk score and diabetes mellitus could serve as risk factors for persistent RD. In multivariate analysis, independent risk factors for persistent RD were: eGFR (OR=3.553, 95% CI, 1.497–25.416, P=0.027), duration of procedure (OR=1.037, 95% CI, 1.002–1.073, P=0.038) and contrast media category (OR=7.189, 95% CI, 1.202–42.982, P=0.031). The results of logistic regression were shown in Table 4.

Table 1 Demographic information, clinical baseline and procedural characteristics of in-hospital mortality and survival patients

Variables	In-hospital mortality(n=5)	Survival(n=59)	P value
Demographics			
Age (years)	71.20±8.41	66.83±9.36	0.317
Male, n (%)	4(80%)	43(73%)	1.000
Systolic blood pressure, mmHg	108.80±34.54	134.41±24,32	**0.032**
Diastolic blood pressure, mmHg	73.20±12.83	72.83±11.66	0.946
Medical History			
Active smoking, n(%)	4(80%)	26(44%)	0.177
Diabetes mellitus, n(%)	1(20%)	23(39%)	0.642
Hypertension, n(%)	4(80%)	45(76%)	1.000
COPD, n(%)	2(40%)	4(7%)	0.065
Prior myocardial infarction, n(%)	3(60%)	13(22%)	0.095
Prior cerebrovascular disease, n(%)	0(0%)	18(31%)	0.310
Clinical presentation			
STEMI, n(%)	3(60%)	16(27%)	0.150
Cardiogenic shock, n(%)	3(60%)	2(3%)	**0.002**
Cardiac arrest, n(%)	2(40%)	3(5%)	**0.045**
Laboratory measurements			
Scr, umol/L	155.1(151.1,183.1)	165.2(128.9,239.05)	0.733
Proteinuria, n(%)	4(80%)	26(44%)	0.177
eGFR<60ml/min/1.73m^2	4(80%)	43(73%)	1.000
Serum albumin, g/L	36.1(31.0,36.3)	33.9(30.4,37.2)	0.908
Hb, g/L	112.20±23.18	114.63±22.25	0.816
Total cholesterol, mmol/l	3.54(3.51,3.58)	3.95(3.30,4.42)	0.278
Triglycerides, mmol/l	1.37(1.27,1.60)	1.28(0.98,2)	0.658
LDL cholesterol, mmol/l	2.12±0.64	2.30±0.76	0.608
HDL cholesterol, mmol/l	0.90(0.85,0.98)	0.93(0.75,1.09)	0.720
Platelets, 10^9/L	131.40±52.61	191.59±57.68	**0.028**
Leukocytes, 10^9/L	11.10(9.94,12.76)	6.81(5.84,10.11)	0.070

CRP, mg/L	25.55(15.30,35.79)	9.23(4.24,32.52)	0.856
CK-MB, IU/L	143.90(81.00,177.70)	15.65(10.43, 37.2)	**0.006**
CK-MB>44 IU/L	4(80%)	10(17%)	**0.007**
NT-proBNP, pg/ml	14121(6638,16024)	5747(3483.61,8114.24)	0.216
NT-proBNP>300 pg/ml	4(80%)	31(53%)	0.366
Mehran score	9(5,11)	5(3,8)	0.178
Medication			
Statins	4(80%)	37(63%)	0.592
ACEI/ARB	1(20%)	13(22%)	1.000
Beta blocker	4(80%)	33(56%)	0.387
Diuretic, n(%)	4(80%)	35(59%)	0.640
Procedural characteristics			
contrast volume, ml	100(100,250)	100(50,150)	0.252
duration of procedure, min	92.40±61.29	69.02±47.31	0.303
IOCM, n(%)	2(40%)	27(46%)	1.000
IABP, n(%)	2(40%)	7(12%)	0.141
Artificial ventilation, n(%)	2(40%)	17(29%)	0.629
Hospital stays	18.5(9.25,37.5)	16(11,22.5)	0.571

Continuous variables were displayed as mean±SD or median and interquartile range. Categorical variables were displayed as No(%). COPD, chronic obstructive pulmonary disease; STEMI, ST-segment elevation myocardial infarction; Scr, serum creatinine; eGFR, estimated glomerular filtration rate; Hb, hemoglobin; LDL cholesterol, low density lipoprotein cholesterol; HDL cholesterol, high density lipoprotein cholesterol; CRP, C-reaction protein; CK-MB, creatine kinase isoenzyme MB; NT-proBNP, N-terminal pro brain natriuretic peptide; ACEI, Angiotensin Converting Enzyme Inhibitors; ARB, Angiotensin Receptor Blockers; IOCM, isotonic contrast media; IABP, intra-aortic balloon pump.

Table 2 Demographic information, clinical baseline and procedural characteristics of persistent and transient RD patients

Variables	Persistent Renal Dysfunction (n=35)	Transient Renal Dysfunction(n=24)	P value
Demographics			
Age (years)	68.17±8.54	64.88±10.32	0.186
Male, n (%)	23(66%)	20(83%)	0.233
Systolic blood pressure, mmHg	136.23±25.68	131.75±22.45	0.492
Diastolic blood pressure, mmHg	74.31±11.07	70.67±12.39	0.241
Medical History			
Active smoking, n(%)	13(37%)	13(54%)	0.286
Diabetes mellitus, n(%)	18(51%)	5(21%)	**0.029**
Hypertension, n(%)	28(80%)	17(71%)	0.536
COPD, n(%)	2(6%)	2(8%)	1.000
Prior myocardial infarction, n(%)	9(26%)	4(17%)	0.529
Prior cerebrovascular disease, n(%)	12(34%)	6(25%)	0.568
Clinical presentation			
STEMI, n(%)	12(34%)	4(17%)	0.233
Cardiogenic shock, n(%)	0(0%)	2(8%)	0.161
Cardiac arrest, n(%)	2(6%)	1(4%)	1.000
Laboratory measurements			

Scr, umol/L	187.80(159.70,265.35)	147.40(112.35,178.48)	**0.005**
Proteinuria, n(%)	14(40%)	12(50%)	0.594
eGFR, ml/min/1.73m^2			**0.002**
>90	5(14%)	8(33%)	
60-89	2(6%)	1(4%)	
30-59	8(23%)	12(50%)	
<30	20(57%)	3(13%)	
Serum albumin, g/L	34.32±7.06	34.98±3.65	0.677
Hb, g/L	109.86±25.32	121.58±14.68	**0.040**
Total cholesterol, mmol/l	4.03±1.00	3.88±0.85	0.503
Triglycerides, mmol/l	1.29(1.09,2.16)	1.26(0.86,1.80)	0.295
LDL cholesterol, mmol/l	2.38±0.75	2.18±0.78	0.317
HDL cholesterol, mmol/l	0.90±0.22	0.97±0.22	0.230
Platelets, 10^9/L	185.86±56.47	199.96±59.59	0.361
Leukocytes, 10^9/L	6.59(5.33,9.80)	7.22(5.90,10.09)	0.605
CRP, mg/L	6.15(3.20,20.13)	18.63(7.16,48.10)	0.866
CK-MB, IU/L	12.8(9.7,27.13)	22.80(14.5,45)	0.093
CK-MB>44 IU/L	6(17%)	4(17%)	1.000
NT-proBNP, pg/ml	6965.00(5349.79,11354.06)	3912.00(2034.14,4874.42)	**0.021**
NT-proBNP>300 pg/ml	22(63%)	9(38%)	0.068
Mehran score	6(4,11)	4(1.75,5)	**0.005**
Mehran risk score stratification			**0.004**
≤5	17(49%)	20(83%)	
6-10	7(20%)	3(13%)	
11-16	10(28%)	1(4%)	
≥16	1(3%)	0(0%)	
Medication			
Statins	24(69%)	13(54%)	1.000
ACEI/ARB	7(20%)	6(25%)	0.753
Beta blocker	22(63%)	11(46%)	0.286
Diuretic, n(%)	21(60%)	14(58%)	1.000
Procedural characteristics			
contrast volume, ml	100(50,200)	100(50,100)	**0.035**
duration of procedure, min	65(45,123)	45(29,55)	**0.001**
IOCM, n(%)	21(60%)	6(25%)	**0.016**
IABP, n(%)	5(14%)	2(8%)	0.689
Artificial ventilation, n(%)	8(23%)	9(38%)	0.254
Hospital stays	15(10,23)	18(13,23)	0.791

Continuous variables were displayed as mean±SD or median and interquartile range. Categorical variables were displayed as No(%). RD, renal dysfunction; COPD, chronic obstructive pulmonary disease; STEMI, ST-segment elevation myocardial infarction; Scr, serum creatinine; eGFR, estimated glomerular filtration rate; Hb, hemoglobin; LDL cholesterol, low density lipoprotein cholesterol; HDL cholesterol, high density lipoprotein cholesterol; CRP, C-reaction protein; CK-MB, creatine kinase isoenzyme MB; NT-proBNP, N-terminal pro brain natriuretic peptide; ACEI, Angiotensin Converting Enzyme Inhibitors; ARB, Angiotensin Receptor Blockers; IOCM, isotonic contrast media; IABP, intra-aortic balloon pump.

Table 3 Univariate and multivariate logistic regression analysis of in-hospital mortality of post-CA/PCI procedure AKI patients

Variables	Univariate analysis			Multivariate analysis		
	Odds ratio	95% confidence interval	p-value	Odds ratio	95% confidence interval	p-value
Prior myocardial infarction	5.308	0.800 to 35.200	0.084			
Prior COPD	8.667	1.107 to 67.864	0.040			
STEMI	4.031	0.616 to 26.392	0.146			
Cardiogenic shock	42.750	4.381 to 417.120	<0.001	40.500	4.147-395.494	<0.001
Cardiac arrest	12.440	1.474 to 105.050	0.021			
Systolic blood pressure	0.948	0.899 to 1.000	0.049			
Leukocytes	0.856	0.702 to 1.045	0.127			
Platelets	0.979	0.959 to 0.999	0.040			
CK-MB>44 IU/L	19.600	1.976 to 194.406	0.011			

COPD, chronic obstructive pulmonary disease; STEMI, ST-segment elevation myocardial infarction; CK-MB, creatine kinase isoenzyme MB.

Table 4 Univariate and multivariate logistic regression analysis of persistent renal dysfunction patients after CA/PCI procedure

Variables	Univariate analysis			Multivariate analysis		
	Odds ratio	95% confidence interval	p-value	Odds ratio	95% confidence interval	p-value
eGFR, ml/min/1.73m2			0.030			0.043
>90	1.000	Reference	-	1.000	Reference	-
60-89	3.200	0.227 to 45.192	0.389	3.247	0.180 to 58.552	0.425
30-59	1.067	0.255 to 4.463	0.930	0.223	0.026 to 1.880	0.168
<30	10.667	2.049 to 55.516	0.005	3.553	0.497 to 25.416	0.207
Scr	1.005	0.999 to 1.012	0.091			
Hb	0.974	0.949 to 1.000	0.052			
NT-proBNP>300 pg/ml	2.821	0.964 to 8.254	0.058			
Duration of procedure	1.033	1.009 to 1.059	0.008	1.037	1.002 to 1.073	0.038
Contrast volume	1.012	1.001 to 1.023	0.026			
IOCM	4.846	1.528 to 15.369	0.007	7.189	1.202 to 42.982	0.031
Mehran score	1.240	1.055 to 1.456	0.009			
Diabetes mellitus	4.024	1.227 to 13.191	0.022			

eGFR, estimated glomerular filtration rate; Scr, serum creatinine; Hb, hemoglobin; NT-proBNP, N-terminal pro brain natriuretic peptide; IOCM, isotonic contrast media; IABP, intra-aortic balloon pump.

Discussion

In the present study, we revealed that the incidence of in-hospital mortality was 7.8% in CI-AKI patients. After adjusting potential confounders, cardiogenic shock (OR=40.5, 95% CI, 4.147–395.494, P=0.001) was the independent risk factor for in-hospital mortality. Persistent RD occurred in 35 (59%) of survival patients. After

adjusting potential confounders, eGFR (OR=3.553, 95% CI, 1.497–25.416, P=0.027), duration of procedure (OR=1.037, 95% CI, 1.002–1.073, P=0.038) and contrast media category (OR=7.189, 95% CI, 1.202–42.982, P=0.031) were independent risk factors for persistent RD.

CI-AKI has become a frequent complication after CA/PCI procedure during hospitalization. Characterized by using iodinated radiocontrast agents, it is also called contrast-induced nephropathy (CIN). Many researchers have assumed that CI-AKI is associated with a great risk of short- or long-term mortality, renal adverse events, major adverse cardiac and cerebrovascular events (MACCE) (8-10).

The incidence of in-hospital death is the most severe short-term outcome of AKI. From our results, the in-hospital mortality rate in CI-AKI patients was 7.8%, which was consistent with other clinical trials (15, 16). However, the severity of AKI and degree of systemic hemodynamic alteration would dramatically impact the incidence of in-hospital death. The mortality rate increased to 31.8% and 50% in severe AKI and AKI with cardiogenic shock patients respectively (17, 18). Thus, we further investigated risk factors influencing mortality rate in AKI populations. Based on our results, the prevalence of cardiogenic shock and cardiac arrest were more frequent in death patients. However, there was no significant difference in admission renal function, contrast media application and clinical procedure between non-survival and survival patients. After adjusting confounders, cardiogenic shock was the only independent risk factor for in-hospital mortality. As a severe systemic hemodynamic dysfunction, cardiogenic shock would not only lead the reduction of renal blood flow and acute renal impairment, but also serve as a crucial prognostic indicator of short-term mortality (19, 20). Furthermore, in patients who suffered cardiogenic shock, the development of AKI also indicates poor prognosis (18, 21). These results from previous studies were consistent with our findings here. The clinical significance of our results is that more attention should be paid to CI-AKI patients who have experienced severe hemodynamic impairment because they are more likely to develop poor prognosis. In addition, CK-MB, an objective surrogate of myocardial ischemia

time (22), was significantly higher in inhospital-death patients. Although CK-MB was not an independent predictor, it also could indirectly reflect decreased cardiac output and the impact of systemic hypoperfusion in mortality.

Persistent RD is renal dysfunction lasting over a certain period. Although there is not a widely accepted consensus for the definition of persistent RD, it has been clearly demonstrated that patients with persistent RD are more likely to develop mortality, hemodialysis and major cardiovascular events compared with those with transient RD and those without CI-AKI (12, 23, 24). Recently, many researchers have demonstrated that patients with risk factors for CI-AKI are also more likely to develop persistent RD. In 2004, based on some important risk factors, Mehran et al. devised a simple score standard to stratify the risk of CI-AKI development, which has been widely applied in coronary artery disease (CAD) patients treated with CA/PCI (10, 12, 25). According to Mehran risk model, preexisting impaired renal function is one of the important risk factors for CI-AKI and has been demonstrated possibly relating to persistent RD. In 2013, Wi et al. reported that presence of high- or very high-risk group in Mehran risk score was a strong independent predictor for CI-AKI with persistent RD (26). The results of Kurogi et al. was also conformed to this view and further demonstrated CV(contrast volume)/eGFR performed similar to Mehran score as predictors for persistent RD (27). Our results also identified that eGFR was the independent risk factor of persistent RD. Although the mechanisms involving in the association between CI-AKI and progressive loss of kidney function have not been fully understood, patients with pre-existing renal function impairment are more susceptible to contrast nephrotoxicity and hemodynamic alterations compared with those absence of preexisting renal function impairment. Therefore, it is reasonable to identify pre-existing impaired renal function as independent risk factors for persistent RD. Moreover, patients with CKD are more likely to be influenced other comorbidities like hypertension, diabetes mellitus, proteinuria and cardiac insufficiency (23, 26). Those comorbidities can also contribute to the progression of renal dysfunction.

The volume and types of contrast media have been demonstrated to be associated with the development of CI-AKI. It has been clearly demonstrated that HOCM is more nephrotoxic than LOCM or IOCM because the osmolality of HOCM is comparatively high (28, 29). However, there is still controversy whether LOCM and IOCM is associated with decreased risk of CI-AKI. From prospective studies from Aspelin et al. and Jo SH et al., IOCM was better than LOCM for prevention of CIN in high-risk population (30, 31). Nevertheless, other studies showed that there was no difference in nephrotoxicity of IOCM and LOCM even in CKD or diabetes patients except some specific categories of LOCM (32-34). The osmolality of IOCM (290 mOsm/kg) is approximately equal to plasma but for the price of obviously increased viscosity (35). High viscosity would cause stasis in tubular fluid, flow resistance and increase of intratubular pressure, which would further promote the decrease of glomerular filtration, hypoperfusion and renal medulla hypoxia (36, 37). Our results showed that the percentage of IOCM use was higher in people who suffered persistent RD; however, the exposure time of contrast media was significantly longer than patients who developed transient RD. Increasing contact time of contrast medium would further amplify the cytotoxic effect to tubular epithelial cells (38). Moreover, contrast media volume was also significantly higher in patients who developed persistent RD, although it was not the independent risk factor in our model. Thus, reducing procedure time and careful selection IOCM may help improving renal outcome of CI-AKI patients. However, we still need more evidence to prove it.

There are still some limitations in our study. This is a single-center, retrospective observational study, results could be impacted by some bias. First, the repeated measurements of serum creatinine after CA/CPI procedure are absent in some patients. As a result, some CI-AKI patients may be neglected. Meanwhile, we cannot completely affirm that the serum creatinine at admission represents actual renal function because renal impairment may occur before CA/PCI. Second, owing to the restriction of retrospective design, small sample size, selection bias may affect the accuracy of our results. We have designed a prospective cohort study to further

validate our findings. Third, data regarding to long-term prognosis was not displayed or incomplete in most of patients. Therefore, we could just analyze risk factors for short-term prognosis. In the future, we would like to conduct a more comprehensive prospective study to further explore risk factors for unfavorable prognosis of CI-AKI.

Conclusions

In-hospital mortality of CI-AKI patients was associated with severe systemic hemodynamic alteration. Patients with existing renal impairment before CA/PCI were more likely to develop persistent RD, while reducing CA/PCI procedure time and usage of isotonic contrast media (IOCM) might help decreasing the risk.

Acknowledgements

We are grateful to the anonymous reviewers whose suggestions and comments contributed to the significant improvement of this paper.

Authors contributions

Junxiang Chen designed the study and finished the manuscript. Yunzhen Deng and Yi He acquired and analyzed the data. Kaiting Zhuang and Kaiting Zhuang reviewed and revised the manuscript. All authors read and approved the final version of the manuscript.

Funding

This study was funded by the National Natural Science Foundation of China (grant no. 81770692).

Availability of data and materials

All data are available from the corresponding author (chenjxly@csu.edu.cn) on reasonable request.

Ethical Statement

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The ethics committee of the Second Xiangya Hospital of Central South University reviewed and approved the study

protocol. Since all the data in this retrospective study were anonymous, the formal consent from the patients were waived by the ethics committee.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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