Hypotension at Hospital Presentation and Post-contrast Acute Kidney Injury Following Computed Tomography with Contrast Media

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Abstract

Background: Post-contrast acute kidney injury (PC-AKI) is a major complication of contrast media usage, and risks for PC-AKI are generally evaluated before performing computed tomography (CT) with contrast at the emergency department (ED). Although persistent hypotension (systolic blood pressure [sBP] <80 mmHg for 1 h) is associated with increased PC-AKI incidence, it remains unclear whether tentative hypotension that is hemodynamically stabilised before CT is a risk for PC-AKI. We hypothesised that hypotension on ED arrival would be associated with higher PC-AKI incidence; even if CT with contrast was performed after patients are appropriately resuscitated.

Methods: This multicenter retrospective observational study was conducted at three tertiary care centres during 2013–2014. We identified 280 patients who underwent CT with contrast at ED and whose kidney function was subsequently evaluated. Patients were divided into two groups based on sBP on arrival (<80 vs $\geq$ 80 mmHg); hypotension was considered as tentative because CT with contrast has always been performed after patients were stabilised at participating hospitals. PC-AKI incidence was compared between the groups, and inverse probability weighting (IPW) was conducted to adjust background characteristics, including age, sex, comorbidities, anaemia, and acute physiology and chronic health evaluation II score.

Results: Eighteen patients were excluded due to chronic haemodialysis, cardiac arrest on arrival, or died within 72 h, and 262 were eligible for this study. PC-AKI incidence was higher in the tentative hypotension group than in the normotension group (7/27 [28.6%] vs 24/235 [10.2%], odds ratio [OR] 3.08 [95% confidence interval (CI) 1.18–8.03], $p = 0.026$), which was confirmed by IPW (OR 3.25 [95% CI 1.99–5.29], $p < 0.001$). Similar results were obtained in subgroups with an estimated glomerular filtration rate of <60 mL/min/1.73 m².

Conclusion: Tentative hypotension at ED was associated with PC-AKI development.

Background

Acute kidney injury (AKI) is a new-onset or worsening renal dysfunction that can be caused by variable factors damaging the kidney, such as cytotoxic agents, hypoxemic distress due to atherosclerosis and hypo perfusion due to hemodynamic instability.^{1–4} Post-contrast AKI (PC-AKI) is a subset of AKI following the intravascular administration of contrast media, previously known as contrast-induced AKI (CI-AKI). It has been extensively reported that PC-AKI is associated with increased short- and long-term mortality and prolonged hospital stay.⁵ Although various study groups have investigated the prevalence of PC-AKI, approximately 5%–7% of patients who underwent percutaneous coronary angiography using contrast media were found to develop PC-AKI.^{6,7}

Computed tomography (CT) is an important imaging modality, and more than 75.6 million CT scans are performed in the United States annually.⁸ It is frequently performed with contrast media particularly in the

emergency department (ED) with increasing popularity⁹ and sometimes followed by emergency interventional radiology that uses additional contrast media if major bleeding is detected. Although such increased exposure to contrast media in the ED has introduced PC-AKI among a considerable number of patients, no effective treatment for PC-AKI has been validated. The only strategy available for physicians would be to avoid unnecessary contrast media exposure during CT scan, particularly in patients with high risks for PC-AKI.

Although risk stratification of patients to predict the development of PC-AKI has been exclusively investigated, persistent hypotension, defined as systolic blood pressure (sBP) of <80 mmHg for at least 1 h requiring inotropic agents, has been suggested as one of the risk factors for PC-AKI.^{10,11} Nevertheless, it remains unclear whether tentative hypotension would affect the incidence of PC-AKI even after patients are hemodynamically stabilised. Considering that patients with hemodynamic instability in the ED would often undergo CT with contrast after stabilisation, we investigated whether CT scan using contrast media is associated with a high incidence of PC-AKI among patients who have a tentative hypotensive episode. Our hypothesis was that temporal hypotension, recognised on arrival at the ED and resuscitated adequately, is associated with PC-AKI following contrast-enhanced CT scan.

Methods

Study design and settings

This was a multicenter retrospective observational study conducted at three tertiary care centres between April 2013 and March 2014. The study protocol was reviewed and approved by the Institutional Review Board (IRB) for the Conduct of Human Research at each participating hospital (application number of IRB at Keio University School of Medicine: 20140447) and complied with the Declaration of Helsinki. Requirement for individual patient informed consent was waived by the IRB because of the retrospective design of this study.

During the study period, the indication for CT with contrast was decided by the treating physician and imaging was performed after sBP was increased to >90 mmHg by fluid resuscitation and/or vasopressor usage. The strategy of resuscitation before CT scan was also decided by the treating physician, considering possible diagnosis and hemodynamic status.

Study population

We retrospectively identified patients who arrived at the ED and were then admitted to each participating hospital during the study period. Inclusion criteria were as follows: 1) patients aged ≥ 18 years; 2) those who underwent a CT scan with contrast media in the ED and 3) those whose serum creatinine (sCr) levels were measured before and 1–3 days after the CT scan. Patients on haemodialysis, those who arrived with cardiac arrest and those who died within 72 h after admission were excluded.

Data collection and definitions

Data were collected from medical records by investigators, which included age, sex, comorbidities, vital signs on hospital arrival, sCr levels, estimated glomerular filtration rate (eGFR) before and after CT scan, haematocrit, acute physiology and chronic health evaluation (APACHE) II score on admission, renal replacement therapy (RRT) performed after admission, hospital length of stay (LOS) and survival status at discharge.

Temporal hypotension was defined as an sBP of <80 mmHg on hospital arrival or the initiation of vasopressors before CT scan; hypotension was considered to be tentative because all patients were resuscitated before the CT scan (sBP was increased to >90 mmHg) as mentioned earlier. PC-AKI was defined based on the most frequently published criteria, i.e. an absolute increase in the sCr level by ≥ 0.5 mg/dL or $\geq 25\%$ increase over baseline sCr level within 72 h after CT with contrast.¹² Anaemia was defined as a haematocrit value of <39% for men and <36% for women.¹⁰ Chronic kidney disease (CKD) was defined as an eGFR of <60 mL/min/1.73 m².

Outcome measures

The primary outcome was the development of PC-AKI. The secondary outcomes included in-hospital mortality, RRT within 30 days after admission and LOS.

Statistical analysis

Patient data were divided between temporal hypotension (sBP < 80 mmHg on hospital arrival) and normotension groups (sBP $\geq$ 80 mmHg on hospital arrival). To adjust covariates between the two groups, inverse probability weighting (IPW) analyses with propensity scores were performed to compare the primary outcome. The propensity score was developed using the logistic regression model to estimate the probability of being in the temporal hypotension group compared with the normotension group. Relevant covariates were carefully selected from known or possible AKI predictors based on previous studies, which included age, sex, presence of anaemia and comorbidities, including congestive heart failure, diabetes mellitus, hypertension, coronary artery disease (CAD), stroke, peripheral artery disease (PAD) and CKD.^{10,11,13} All these variables were entered into the propensity model, where patients with missing covariates were excluded from the propensity score calculation. The precision of discrimination and propensity score calibration were analysed using the c-statistic and Hosmer–Lemeshow goodness-of-fit test.¹⁴ IPW analyses were then performed to compare the primary and secondary outcomes between the two groups using chi-square test and ordinal logistic regression analysis.

Sensitivity analyses were conducted to validate the primary results. An IPW analysis with restriction was performed without using patient data with <0.1 or >0.9 of the propensity score to avoid extreme weights. Logistic regression analysis was also performed using the propensity score as covariate to confirm whether the results were independent of the IPW method.

Subgroup analyses were conducted to investigate the relationships between the effect of temporal hypotension on the development of PC-AKI and a baseline kidney function. Propensity score development

and IPW analyses on the primary outcome were repeated in each subgroup divided by the baseline eGFR.

Descriptive statistics were expressed as mean, median (interquartile range), or number (percentage) and were compared using unpaired *t*-tests, Mann–Whitney U tests, chi-square tests, or Fisher's exact tests, as appropriate. For testing all hypotheses, a two-sided α threshold of 0.05 was considered to be statistically significant. All statistical analyses were conducted using SPSS, version 26.0 (IBM, Armonk, NY), and Microsoft Excel (Microsoft, Redmond, WA, USA).

Results

After the screening process, 299 patients who underwent CT scan with contrast were identified during the study period. Although 280 patients satisfied all the inclusion criteria, 4 patients on haemodialysis and 11 with out-of-hospital cardiac arrest were excluded. Figure 1 summarises the patient flow diagram.

A total of 262 patients were eligible for this study, of whom 27 (10.2%) had temporal hypotension and 235 did not have temporal hypotension. The characteristics of the patients are summarised in Table 1. Patients in the temporal hypotension group had higher APACHE II scores on admission than those in the normotension group [15 (11–21) vs. 9 (6–14)]. Compared with the normotension group, more patients in the temporal hypotension group had CAD and PAD [22 (9.4%) vs. 1 (3.7%) and 5 (2.1%) vs. 0 (0.0%), respectively].

The propensity model predicting allocation to the temporal hypotension group was validated to have sufficient discrimination and calibration (c-statistic = 0.783 and Hosmer–Lemeshow goodness-of-fit p = 0.690), and no patients were excluded from IPW analyses due to missing covariates. The characteristics of the patients after IPW are summarised in Table 1, in which most covariates were successfully adjusted.

The incidence of PC-AKI was significantly higher among patients with temporal hypotension than among patients with normotension on ED arrival in the unadjusted analysis [7 (28.6%) vs. 24 (10.2%); odds ratio (OR), 3.08; 95% confidence interval (CI), 1.18–8.03; p = 0.026; Table 2]; these results were validated by the IPW analysis (27.8% vs. 10.6%; OR, 3.25; 95% CI, 1.99–5.29; p < 0.001; Table 2). Adjusted analyses for secondary outcomes showed that in-hospital mortality and RRT within 30 days after admission were significantly higher among patients with temporal hypotension than among patients with normotension (11.7% vs. 3.8%; OR, 3.34; 95% CI, 1.57–7.09; p = 0.001 and 8.5% vs. 1.5%; OR, 6.03; 95% CI, 2.02–18.00; p < 0.001, respectively; Table 2). The temporal hypotension on arrival was also associated with longer hospital stay (mean LOS = 24 vs. 14 days; coefficient = 11 days; 95% CI, 1–20 days; Table 2).

Sensitivity analyses were performed to assure that the primary results were not dependent on the propensity score. The IPW analysis, excluding patient data with <0.1 or >0.9 of the propensity score, similarly revealed that temporal hypotension was significantly associated with a higher incidence of PC-AKI (38.0% vs. 14.8%; OR, 3.54; 95% CI, 1.73–7.22; Table 3). Logistic regression with propensity score as a

covariate also validated the association between temporal hypotension and PC-AKI incidence (OR, 3.26; 95% CI, 1.99–5.33; Table 3).

Subgroup analyses demonstrated that the relationship between temporal hypotension on admission and PC-AKI incidence existed among patients with eGFR < 60 mL/min/1.73 m² [23.0% (95% CI, 14.1%–35.0%) in the temporal hypotension group vs. 1.2% (95% CI, –0.4% to 7.1%) in the normotension group; Table 4], but not among patients with eGFR ≥ 60 mL/min/1.73 m².

Discussion

This retrospective study demonstrated that tentative hypotension identified on hospital arrival was associated with a higher incidence of PC-AKI. This relationship was validated using IPW analysis, in which several AKI predictors were adjusted. Remarkably, the observed association was consistent across several sensitivity analyses, suggesting that the results were not dependent on the method of propensity score or the weighting.

Although the reason behind the relationship between tentative hypotension and higher PC-AKI incidence remains inconclusive, several pathophysiological mechanisms could be considered based on previous studies. First, ischaemic damages to the nephron caused by decreased renal blood flow during tentative hypotension would be persistent for considerable duration at least until contrast media was administered.¹⁵ Considering that CT with contrast has been reported to introduce PC-AKI only among patients with a history of higher stage CKD,^{16,17} clinically obvious AKI would have developed by contrast media in patients with preconditioning insults by tentative hypotension in this study. Second, a reduction in the outer medullary blood flow has been reported to be critical even for a shorter period of time because renal tissue injury would be extended after reperfusion. Although blood flow in most kidney tissues, cortex and inner medulla rapidly recovers after ischaemia, it takes more than 3 h in the outer medulla to be normalised after reperfusion.¹⁵ Given that contrast media is a potent vasoconstrictive agent,¹⁸ the contrast media used for CT would further reduce the outer medullary blood flow.

Although the results of this study suggested tentative hypotension as a risk factor for PC-AKI, the duration of hypotension that affects the kidney function before contrast media usage remains inconclusive. A retrospective study on 7230 patients undergoing PCI showed that transient hypotension for <1 h occurred more often in patients with PC-AKI than in those without PC-AKI.¹⁹ Another experimental study demonstrated a persistent reduction of blood flow in the outer medulla of rats after 30-min renal ischaemia.¹⁵ The degree of impact depending on the duration of tentative hypotension on the kidney function before CT with contrast needs to be further explored in a future study.

Although tentative hypotension would predispose patients to develop AKI after CT with contrast, the present study does not deny intravenous hydration. Several studies have reported that the incidence of PC-AKI was significantly lower in patients who received appropriate hydration than in those without

hydration,²⁰⁻²⁴ and intravenous fluid should be administered before and after the usage of contrast media, as a preventive strategy for PC-AKI.

Limitations

The results of this study must be interpreted within the context of the study design. Unfortunately, we did not record the amount of pre- or post-procedural fluid administration that would prevent AKI. However, patients with hypotension on ED arrival generally receive a considerable amount of fluid, which is probably more than that received by patients with normotension. Another limitation is that we did not analyse variables relating to the indication for CT with contrast in this study. If the indication for CT was a strong predictor for AKI, the results would be different. Moreover, we could not confirm the causal relationship between the contrast media and PC-AKI because of the lack of a control group, i.e. patients without contrast media usage. However, our results would at least indicate that tentative hypotension renders patients susceptible to kidney injury after contrast media usage. Furthermore, the IPW could not adjust all covariates and some residual differences existed in the adjusted analysis. To validate the results in this study, further study with a large sample size should be conducted. Finally, due to the retrospective study design, our results are not conclusive. Residual confounding and unmeasured AKI predictors preclude confirmation of our results.

Conclusion

To summarise, our results suggest that tentative hypotension in the ED was associated with the development of PC-AKI after the usage of contrast media. Therefore, a careful risk–benefit discussion must be performed before conducting CT with contrast for patients with hypotension on hospital arrival.

Abbreviations

PC-AKI: Post-contrast acute kidney injury

CT: Computed tomography

ED: Emergency department

sBP: Systolic blood pressure

IPW: Inverse probability weighting

OR: Odds ratio

CI: Confidence interval

AKI: Acute kidney injury

CI-AKI: Contrast-induced acute kidney injury

sCr: Serum creatinine

eGFR: Estimated glomerular filtration rate

APACHE: Acute physiology and chronic health evaluation

RRT: Renal replacement therapy

LOS: Length of stay

CAD: Coronary artery disease

PAD: Peripheral artery disease

CKD: Chronic kidney disease

Declarations

Ethics approval and consent to participate

The study protocol was reviewed and approved by the Institutional Review Board (IRB) for the Conduct of Human Research at each participating hospital (application number of IRB at Keio University School of Medicine: 20140447) and complied with the Declaration of Helsinki. Requirement for individual patient informed consent was waived by the IRB because of the retrospective design of this study.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and analysed during the current study are not publicly available due to privacy or ethical restrictions, but are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

J.Y. and R.Y. designed the study. J.Y., R.Y., K.H., H.K., K.S., Y.K. and T.F. performed data collection. J.S. managed quality control. J.Y., R.Y., K.H., H.K., K.S., Y.K., T.F. and J.S. performed data analysis, data

interpretation, writing and critical revision. All authors revised the manuscript.

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Tables

Table 1 Characteristics of patients who underwent contrast-enhanced computed tomography scan in the ED

	Before IPW			After IPW		
	Temporal	Normotension	p	Temporal	Normotension	p
	hypotension		value	hypotension		value
Case	27	235				
Age, median (IQR)	64 (51–76)	64 (48–77)	0.886	70 (54–78)	64 (49–77)	0.093
Male, n (%)	19 (70.4)	171 (72.8)	0.792	170 (76.2)	191 (72.6)	0.364
sCr (mg/dL), median (IQR)	0.87 (0.60–1.31)	0.79 (0.66–1.01)	0.331	0.81 (0.64–1.05)	0.80 (0.66–1.03)	0.934
eGFR (mL/min/1.73 m ²), median (IQR)	66 (45–95)	74 (55–91)	0.272	72 (48–95)	73 (54–91)	0.894
Anaemia, n (%)	12 (44.4)	111 (47.2)	0.783	100 (44.8)	124 (47.1)	0.611
Comorbidities, n (%)						
CHF	1 (3.7)	8 (3.4)	1.000	16 (7.2)	9 (3.4)	0.062
Diabetes	3 (11.1)	31 (13.2)	1.000	30 (13.5)	34 (12.9)	0.852
HTN	7 (25.9)	68 (28.9)	0.743	90 (40.4)	75 (28.4)	0.006
CAD	1 (3.7)	22 (9.4)	0.485	9 (4.0)	23 (8.7)	0.037
Stroke	3 (11.1)	24 (10.2)	0.748	22 (9.9)	27 (10.3)	0.884
PAD	0	5 (2.1)	1.000	0	5 (1.9)	0.065
APACHE II score, median (IQR)	15 (11–21)	9 (6–14)	<0.001	13 (9–17)	10 (6–15)	<0.001

APACHE II score = acute physiology and chronic health evaluation II score, CAD = coronary artery disease, CHF = congestive heart failure, eGFR = estimated glomerular filtration rate, HTN = hypertension, IPW = inverse probability weighting, IQR = interquartile range, PAD = peripheral artery disease and sCr = serum creatinine.

Table 2 Rates of post-contrast acute kidney injury and secondary outcomes

	Temporal hypotension	Normotension	p value	OR/coefficients	95% CI
Rates of PC-AKI					
Unadjusted analysis, n/total (%)	7/27 (28.6)	24/235 (10.2)	0.026	3.08	1.18–8.03
After IPW, % (95% CI)	27.8 (22.3–34.0)	10.6 (7.39–14.9)	<0.001	3.25	1.99–5.29
Secondary outcomes					
Emergent RRT, % (95% CI)	8.5 (5.5–13.0)	1.5 (0.50–4.0)	<0.001	6.03	2.02–18.00
In-hospital mortality, % (95% CI)	11.7 (8.0–16.6)	3.8 (2.0–6.9)	0.001	3.34	1.57–7.09
LOS, days, mean, median (IQR)	24.2, 14.0 (8.0–37.0)	13.8, 8.0 (5.0–16.0)		10.5	0.76–20.1

CI = confidence interval, IPW = inverse probability weighting, IQR = interquartile range, LOS = length of hospital stay, OR = odds ratio, PC-AKI = post-contrast acute kidney injury and RRT = renal replacement therapy.

Table 3 Rate of post-contrast acute kidney injury estimated using sensitivity analyses

	PC-AKI, % (95% CI)			
	Temporal hypotension	Normotension	OR	95% CI
IPW with restriction (0.1–0.9 of propensity score)	38.0 (29.1–47.8)	14.8 (8.7–23.8)	3.54	1.73–7.22
Propensity score as covariate			3.26	1.99–5.33

CI = confidence interval, IPW = inverse probability weighting, OR = odds ratio and PC-AKI = post-contrast acute kidney injury.

Table 4 Rate of post-contrast acute kidney injury estimated using subgroup analysis

	PC-AKI, % (95% CI)			
	Temporal hypotension	Normotension	OR	95% CI
eGFR < 60 mL/min/1.73 m ²	23.0 (14.1–35.0)	1.2 (–0.4 to 7.1)	24.43	3.11–191.68
eGFR ≥ 60 mL/min/1.73 m ²	17.9 (12.5–24.8)	14.6 (10.1–20.6)	1.27	0.71–2.29

CI = confidence interval, eGFR = estimated glomerular filtration rate, OR = odds ratio and PC-AKI = post-contrast acute kidney injury.

Figures

Figure 1

Patient flow diagram

Of the 299 patients who underwent CT scan with contrast, 262 were eligible for this study. Among them, 27 (10%) had tentative hypotension. CECT = contrast-enhanced computed tomography, IPW = inverse probability weighting and sCr = serum creatinine.