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POSTCONTRAST ACUTE KIDNEY INJURY AFTER COMPUTED TOMOGRAPHY PULMONARY ANGIOGRAPHY FOR ACUTE PULMONARY EMBOLISM

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☐ **Abstract—Background: Despite the widespread use of computed tomography pulmonary angiography with contrast media for the diagnosis of acute pulmonary embolism, high-quality evidence on risk factors for postcontrast acute kidney injury related to its use is lacking. Objective: The present study aimed to investigate whether the level of estimated glomerular filtration rate observed in the emergency department (ED) is significantly associated with the occurrence of postcontrast acute kidney injury in patients undergoing computed tomography pulmonary angiography. Methods: We performed a retrospective observational study using data automatically collected by a clinical data retrieval system from 1300 patients who underwent computed tomography pulmonary angiography for suspected acute pulmonary embolism in the ED. A total of 632 patients were selected for the study after exclusion. Univariate analyses were performed to identify significant risk factors for postcontrast acute kidney injury (the primary outcome). Multivariate logistic regression analysis was used to confirm the effect of estimated glomerular filtration rate in the ED on the occurrence of postcontrast acute kidney injury after adjustment for confounding variables. Results: The total incidence rate of postcontrast acute kidney injury was 6.49% (41/632 patients). No statistically significant association between estimated glomerular filtration rate and the risk of postcontrast acute kidney injury was observed. Conclusion: Our study findings could serve as useful reference for physicians who are concerned about performing computed tomography pulmonary angiography for fear of renal function deterioration.** © **2019 Elsevier Inc. All rights reserved.**

☐ **Keywords—renal function; acute kidney injury; postcontrast acute kidney injury; computed tomography pulmonary angiography; acute pulmonary embolism**

INTRODUCTION

Acute pulmonary embolism (APE) is a potentially life-threatening disease associated with high mortality, and requires early diagnosis and proper management in the emergency department (ED) (1,2). Due to its high sensitivity and specificity, rapid availability, and advantage in establishing alternative diagnoses, computed tomography pulmonary angiography (CTPA) is recognized as a standard diagnostic procedure for patients with suspected APE in the ED (3,4). Despite the advantages of CTPA, concerns about contrast exposure may lead physicians to hesitate to use CTPA in the ED due to concerns regarding the risk of postcontrast acute kidney injury (PC-AKI). Contrast exposure has been considered a common cause of hospital-acquired acute kidney injury (AKI) for a long time, and no effective treatment or preventative strategy for it exists (5–7).

Recent studies have reported that the risk of PC-AKI may have been overestimated by clinicians (8,9). With

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emerging clinical data refuting the causal relationship between contrast exposure and nephrotoxicity, high-quality evidence is required for clinical decision-making (9–12). In particular, there is a debate surrounding whether contrast exposure is associated with nephrotoxicity in patients with renal function impairment (13–17). No prospective randomized controlled trials have investigated this issue, and retrospective studies have limitations due to the difficulty in controlling confounders such as comorbidities and disease severity (8,9,18). Therefore, practical research is required to control confounders resulting from study population heterogeneity and to provide physicians with high-quality evidence for decision-making in the specific clinical setting (8,9). Therefore, the present study aimed to investigate whether the level of estimated glomerular filtration rate (eGFR) observed in the ED is significantly associated with the occurrence of PC-AKI in patients undergoing CTPA.

MATERIALS AND METHODS

This retrospective observational study was approved by the institutional review board of the clinical trial center (approval number 4-2018-0810) and conformed to the Strengthening the Reporting of Observation Studies in Epidemiology (STROBE) guidelines for reporting observational studies. The requirement for informed consent was waived owing to the study's retrospective design, and the study protocol followed the Declaration of Helsinki.

Study Setting and Population

The present study was performed in a Level I ED with approximately 80,000 annual visits at an urban tertiary teaching hospital located in the northwestern area of Seoul, Korea. On average, approximately 3000 computed tomography (CT) scans per month were performed in this ED. A bolus infusion of 60 mL iodinated contrast agent (100 mL/bottle; Xenetix 300; Guerbet, Villepinte, France) was intravenously administered at a rate of 4 mL/s using an automatic injector following our CTPA protocol. CT scans were performed using a 128-slice multidetector CT scanner (Revolution EVO; GE Healthcare, Chicago, IL). All consecutive patients who underwent CTPA in the ED between January 2015 and December 2017 were included in the study. Among these patients, those who either 1) had a history of end-stage renal disease or 2) did not undergo follow-up serum creatinine test within 48 h after contrast media administration were excluded from the analysis.

Data Source and Collection

Data in the present study were automatically collected using a clinical data retrieval system developed by the medical information department. This system allowed us to define a clinical data model with desired variables and conditions. We defined a clinical data model that included all consecutive patients who underwent CTPA in the ED within the study period; variables specified by us were automatically coded in this model and included patients' age, sex, previous medical history, use of nephrotoxic medications, laboratory results, time of renal replacement therapy (RRT) initiation, and mortality.

Definition of Exposure and Outcome

The study exposure was the last eGFR prior to the performance of CTPA in the ED. We used the Chronic Kidney Disease Epidemiology Collaboration equation to calculate eGFR in accordance with the current guideline (19). In our dataset, eGFR measured as a continuous variable was converted into a categorical variable in four steps. The primary outcome was PC-AKI, which was defined as an increase in serum creatinine concentration $\geq$ 0.3 mg/dL or a $\geq$ 1.5–1.9-fold increase from baseline (Kidney Disease: Improving Global Outcomes definition of AKI) within 48–72 h after contrast media administration (18).

Statistical Analysis

Categorical variables were reported as counts and percentages and were analyzed using chi-squared test or Fisher's exact test. Continuous variables were expressed as median and interquartile range and were compared between the two groups using the Mann–Whitney *U*-test because all continuous variables in our dataset violated normality by the Shapiro–Wilk test. Differences were considered statistically significant at $p < 0.05$. Univariate logistic analyses were used to assess the effect of the baseline characteristics of patients on PC-AKI, and variables with a univariate *p*-value of < 0.05 were included in the multivariate logistic regression analysis. Furthermore, clinically significant factors reported to be independent predictors of the risk for PC-AKI according to previous studies were added to the multivariate logistic regression analysis. All statistical analyses were performed using SAS software version 9.4 (SAS Institute, Cary, NC).

RESULTS

Within the study period, 1300 patients in total underwent CTPA for suspected APE in the ED. Among these patients, 639 patients were excluded because they did not

undergo serum creatinine test within 48 h after contrast media administration. Additionally, 29 patients were excluded owing to a history of end-stage renal disease. Thus, a total of 632 patients were included in the present study. In the study population, 11 (1.74%) patients had eGFR < 30 mL/min/1.73 m^2; 94 patients (14.87%), eGFR between 30 and 59 mL/min/1.73 m^2; 249 patients (39.4%), eGFR between 60 and 89 mL/min/1.73 m^2; and 278 patients (43.99%), eGFR ≥ 90 mL/min/1.73 m^2. The total incidence rate of PC-AKI in the present study was 6.49% (41/632), and the incidence rate in the subgroup classified according to the last eGFR prior to CTPA was 27.27% (3/11), 6.38% (6/94), 6.43% (16/249), and 5.76% (16/278), respectively (Figure 1).

In the univariate analyses, the risk of PC-AKI was significantly associated with serum hemoglobin level, history of heart failure, history of hematologic disease, Glasgow Coma Scale score, and insulin use after CTPA (Table 1). These factors were used as covariates in the multivariate logistic regression analysis, with patients' age, history of diabetes mellitus, and pH identified as independent risk factors for PC-AKI according to previous studies. In the present study, the last eGFR prior to CTPA was associated with PC-AKI in the univariate analysis; however, such association was not statistically significant in the multivariate analysis (Table 2). We confirmed that there was no multicollinearity problem among variables included in the multivariate model, as confirmed by the variance inflation factor value used, and the *p*-value for the Hosmer–Lemeshow test was 0.213, suggesting that our model was well calibrated.

Figure 2 shows the clinical flow related to renal function deterioration after CTPA. Among patients with PC-AKI in the present study, 5 (0.79%) patients received RRT. RRT was initiated in 3 of these patients owing to hypoxic brain damage and sepsis after cardiac arrest. One patient received RRT for metabolic acidosis due to intraperitoneal bleeding, whereas another patient underwent RRT due to pneumonia with septic shock.

DISCUSSION

APE is a serious clinical condition with a high mortality rate (9–31%) (20–22). In the International Cooperative Pulmonary Embolism Registry, the fatality rates of APE were approximately 15% and 58% in hemodynamically stable patients and unstable patients, respectively (23). Hence, a rapid and accurate diagnostic approach for APE is required in the ED. CTPA is the best modality suitable for this demand in the ED (3,4). However, in clinical practice, concerns about kidney injury resulting from contrast exposure hinder physicians from determining the progression of CTPA in all patients with suspected APE. Correspondingly, one of the emergency medicine textbooks recommends that pulmonary ventilation–perfusion scanning should be preferentially performed instead of CTPA for patients with eGFR < 60 mL/min/1.73 m^2 (24). However, confirming the causal relationship between contrast administration and AKI is very difficult because they are highly confounded, bidirectional, and unpredictable (8). Therefore, the Contrast Media Safety Committee recommended that the previous term "contrast-induced nephropathy" should be replaced with the term "PC-AKI" to refer to the presence of AKI after contrast exposure without causal relation between contrast exposure and renal function deterioration (18).

Recently, several studies have suggested that the clinical risk of PC-AKI has been overestimated (8,25,26). In particular, large retrospective studies that used propensity score matching to evaluate PC-AKI after contrast-enhanced CT and that categorized populations according to their baseline eGFR have reported that the risk of PC-

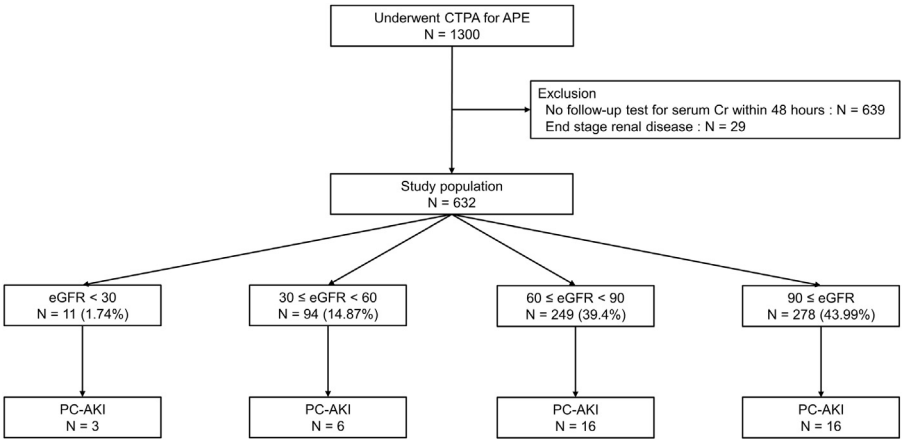

Figure 1. Study enrollment flow chart. CTPA = computed tomography pulmonary angiography; APE = acute pulmonary embolism; Cr = creatinine; eGFR = estimated glomerular filtration rate; PC-AKI = postcontrast acute kidney injury.

Table 1. Unadjusted Odds Ratios for PC-AKI

Variable	Odds Ratio (95% CI)	*p*-Value
Sex, male	1.112 (0.588–2.104)	0.744
Age	0.997 (0.976–1.019)	0.775
Baseline renal function		
eGFR < 30	Ref	
30 ≤ eGFR < 60	0.182 (0.038–0.867)	0.033
60 ≤ eGFR < 90	0.183 (0.044–0.757)	0.019
90 ≤ eGFR	0.163 (0.039–0.673)	0.012
Previous medical history		
Hypertension		
No	Ref	
Yes	0.771 (0.406–1.465)	0.427
Diabetes mellitus		
No	Ref	
Yes	1.300 (0.634–2.666)	0.473
Hyperlipidemia		
No	Ref	
Yes	2.042 (0.585–7.129)	0.263
Heart failure		
No	Ref	
Yes	4.050 (1.555–10.551)	0.004
Occlusive coronary artery disease		
No	Ref	
Yes	1.789 (0.760–4.209)	0.183
Atrial fibrillation		
No	Ref	
Yes	0.896 (0.207–3.877)	0.883
Hematologic disease		
No	Ref	
Yes	9.253 (2.131–40.181)	0.003
Malignancy		
No	Ref	
Yes	1.104 (0.580–2.100)	0.763
Liver disease		
No	Ref	
Yes	1.032 (0.237–4.489)	0.967
Laboratory results		
White blood cell	1.040 (0.979–1.105)	0.199
Neutrophil	1.016 (0.950–1.088)	0.641
Hemoglobin	0.856 (0.748–0.980)	0.025
Hematocrit	0.961 (0.917–1.007)	0.096
Red blood cell distribution width	1.121 (0.991–1.268)	0.069
Delta neutrophil index	1.031 (0.991–1.072)	0.127
Platelet	1.001 (0.998–1.003)	0.552
International normalized ratio	0.893 (0.439–1.815)	0.754
Blood urea nitrogen	1.020 (0.994–1.047)	0.140
Serum sodium	1.031 (0.968–1.098)	0.348
Albumin	0.642 (0.393–1.050)	0.077
C-reactive protein	1.002 (0.999–1.005)	0.284
Total cholesterol	0.992 (0.984–1.000)	0.053
Serum glucose	1.002 (0.998–1.005)	0.391
D-dimer	1.026 (0.995–1.058)	0.095
pH	0.770 (0.409–1.447)	0.417
Use of nephrotoxic medications before*		
No	Ref	
Yes	1.324 (0.695–2.521)	0.393
Use of nephrotoxic medications after†		
No	Ref	
Yes	0.867 (0.453–1.658)	0.666
Insulin before		
No	Ref	
Yes	1.319 (0.166–10.468)	0.794
Insulin after		
No	Ref	
Yes	4.684 (2.438–8.999)	<0.001
Glasgow Coma Scale score	0.799 (0.713–0.895)	0.001
Systolic blood pressure	1.009 (0.998–1.020)	0.110
Diastolic blood pressure	1.007 (0.988–1.027)	0.462
Confirmed APE	0.492 (0.223–1.086)	0.079
Confirmed sepsis	1.587 (0.462–5.459)	0.463

PC-AKI = post-contrast acute kidney injury; CI = confidence interval; eGFR = estimated glomerular filtration rate; APE = acute pulmonary embolism.
The effects of variables were analyzed using univariate logistic regression.
Nephrotoxic medications include nonsteroidal anti-inflammatory drug, angiotensin-converting enzyme inhibitor, angiotensin II receptor blocker, statin, aminoglycoside, and amphotericin B.
* Refers to medication use prior to contrast exposure.
† Refers to medication use after contrast exposure.

AKI in patients with eGFR ≥ 30 mL/min/1.73 m^2 was not significantly high; nonetheless, there remains conflicting evidence on whether patients with eGFR < 30 mL/min/1.73 m^2 are at increased risk of PC-AKI (27–29). The present study showed that the last eGFR prior to CTPA in patients with suspected APE in the ED was not associated with the occurrence of PC-AKI, even if the eGFR was < 30 mL/min/1.73 m^2. Moreover, only 5 patients who underwent CTPA required RRT after PC-AKI, and these cases were accompanied by other predisposing conditions aside from contrast exposure such as sepsis, multiorgan failure, and cardiac arrest that required RRT initiation for renal dysfunction. Alternative modalities of CTPA are inadequate to efficiently diagnose APE in various ways. Pulmonary ventilation–perfusion scanning alone is incapable of detecting APE, and the utility of echocardiography to confirm right ventricular dysfunction, which can be observed only in severe APE, is dependent on the operator's skill (30). Thus, when CTPA for APE is avoided, the risk of misdiagnosis could increase, leading to worse clinical outcomes (9,31). The present study shows that there is no need to choose imaging studies based on the patient's eGFR level; the risk–benefit ratio favors the performance of CTPA in the appropriate clinical situation.

All independent predictors of the risk for PC-AKI in our multivariate analysis were factors susceptible to the influence of AKI regardless of contrast exposure. Schefold et al. described how cardiac and renal diseases interact through numerous pathophysiological pathways in both acute and chronic settings (32). Furthermore, they reported that chronic heart failure leads to progressive renal impairment via accelerated renal cell apoptosis and replacement fibrosis (32). Hematologic diseases such

Table 2. Adjusted Odds Ratios for PC-AKI

Variable	Odds Ratio (95% CI)	*p*-Value
Baseline renal function		
eGFR < 30	Ref	
30 ≤ eGFR < 60	0.265 (0.034–2.037)	0.238
60 ≤ eGFR < 90	0.386 (0.057–2.615)	0.695
90 ≤ eGFR	0.373 (0.052–2.664)	0.650
Age	0.992 (0.966–1.020)	0.581
Hemoglobin	0.847 (0.728–0.984)	0.031
pH	0.901 (0.341–2.384)	0.834
Glasgow Coma Scale score	0.807 (0.700–0.931)	0.003
Diabetes mellitus		
No	Ref	–
Yes	0.448 (0.179–1.124)	0.087
Heart failure		
No	Ref	–
Yes	3.275 (1.04–10.316)	0.043
Hematologic disease		
No	Ref	–
Yes	9.197 (1.864–45.369)	0.006
Insulin after*		
No	Ref	–
Yes	5.591 (2.445–12.783)	<0.001

PC-AKI = postcontrast acute kidney injury; CI = confidence interval; eGFR = estimated glomerular filtration rate.
The effects of variables were analyzed using multivariable logistic regression.
* Refers to medication use after contrast exposure.

as myeloma and leukemia have been reported to be the malignancies associated with the highest 5-year incidence of AKI (33). Renal function deterioration may occur via cast nephropathy, hypercalcemia, and glomerulopathy in patients with myeloma (34–36). The risk of sepsis, volume depletion, and tumor lysis syndrome could contribute to AKI in patients with leukemia (37). Patients requiring insulin use after contrast exposure could be thought of as patients with diabetes who need stricter sugar control owing to disease severity. Normochromic normocytic anemia is observed in patients with reduced glomerular filtration rate as a result of the inability of the kidney to sufficiently synthesize erythropoietin (32). The level of consciousness could be reduced in patients with septic shock or other hemodynamically unstable conditions susceptible to AKI (38). In conclusion, the eGFR value observed in the ED could be explained as a marker of other significant predictors identified in the present study, which could elucidate why our results were contrary to those of previous studies that reported baseline renal function prior to contrast exposure as a risk factor for PC-AKI.

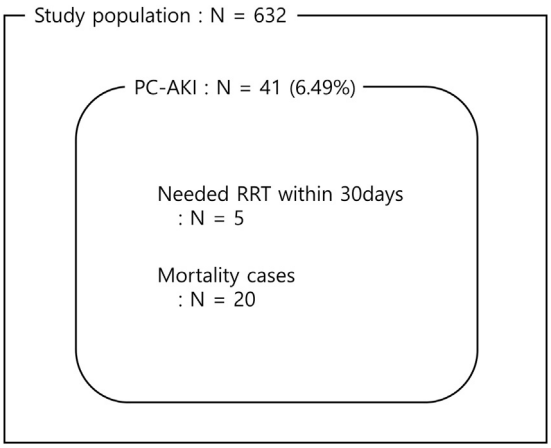

Figure 2. Clinical flow after computed tomography pulmonary angiography. PC-AKI = postcontrast acute kidney injury; RRT = renal replacement therapy.

Despite the rapid increase in the use of CTPA in clinical practice, there is a lack of standardized evidence supporting the strategic choice of CTPA decision (7). The considerable variability in outcomes of previous studies in relation to PC-AKI could be attributed to the difficulty of controlling appropriately for confounders (in both prospective and retrospective study designs) owing to population heterogeneity (6,9,39). In particular, previous studies did not take into account the differences in nephrotoxicity between angiocardiography and intravenous administration by conflating them (31). Therefore, for clinically useful evidence, it is necessary to design the study for targeted clinical setting. For this reason, we posed a research question to resolve the conflicts between PC-AKI development due to low eGFR in the ED and accurate diagnosis of APE through CTPA. We enrolled all patients with suspected APE in the ED who underwent CTPA to maintain study population heterogeneity consistent with the research question while attempting to eliminate the selection bias of previous studies that included patients diagnosed with only APE. Subsequent studies on PC-AKI should also be performed in the same manner to provide tailored evidence on specific clinical practice such as angiography for large-vessel occlusion or contrast-enhanced CT for pancreatitis.

Limitations

Our study has several limitations. The present study was retrospectively designed and prone to unidentified confounders. In addition, it is difficult to generalize our results because they were derived from a single center. Therefore, additional multicenter studies should be conducted to apply our results to clinical practice. In our study, a large number of predictors were used in the multivariate regression model, considering the number of primary outcomes that occurred. Therefore, the statistical modeling used in the present study does not exclude the possibility of overfitting. Lastly, our study focused on PC-AKI, and it may be more practical for physicians to demonstrate associations between CTPA and clinical outcomes such as death or the need for RRT rather than an intermediate endpoint such as PC-AKI itself.

CONCLUSIONS

The eGFR level in the ED is not significantly associated with the occurrence of PC-AKI in patients undergoing CTPA for APE. Thus, our study results suggest that physicians who are hesitant in deciding whether CTPA should be performed for fear of PC-AKI should not base their decisions on patients' eGFR.

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ARTICLE SUMMARY

1. Why is this topic important?

Despite the widespread use of computed tomography pulmonary angiography (CTPA) with contrast media for the diagnosis of acute pulmonary embolism, high-quality evidence on its use considering postcontrast acute kidney injury (PC-AKI) is lacking.

2. What does this study attempt to show?

This study attempted to determine whether the level of estimated glomerular filtration rate (eGFR) observed in the emergency department is significantly associated with the occurrence of PC-AKI in patients undergoing CTPA.

3. What are the key findings?

No statistically significant association between eGFR and the risk of PC-AKI was observed.

4. How is patient care impacted?

The findings suggest that physicians who are hesitant in deciding whether CTPA should be performed for fear of PC-AKI should not base their decisions on patients' eGFR.