

# Gadolinium-Associated Acute Kidney Injury: A 10-Year Single-Center Retrospective Cohort Study

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## Abstract

**Background:** In studies conducted in Europe and the United States, gadolinium-associated acute kidney injury (GA-AKI) has been reported in high-risk patients after receiving gadolinium-based contrast agents (GBCAs). Currently, there is no data available on Asian populations to confirm these findings.

**Methods:** A retrospective cohort study of chronic kidney disease patients with an estimated glomerular filtration rate (eGFR)  $\leq 60$  mL/min/1.73m<sup>2</sup> who received GBCAs from 2013 to 2023 at a tertiary care hospital. The outcomes were the incidence of GA-AKI and the associated risk factors.

**Results:** Among the 182 patients, the incidence of GA-AKI was 4.4%. Although the GA-AKI group had significantly higher age ( $> 65$  years) (OR 6.374; 95% CI 0.720-56.426,  $p=0.096$ ), diastolic blood pressure  $> 80$  (OR 6.148; 95% CI 1.111-34.008,  $p=0.037$ ) and eGFR  $\leq 30$  mL/min/1.73m<sup>2</sup> (OR 7.920; 95% CI 1.642-38.196,  $p=0.010$ ). The ROC curve analysis for predicting GA-AKI scored 2 out of 3, with a sensitivity of 87.5% and a specificity of 78.2%.

**Conclusions:** The incidence of GA-AKI was low. The associated factors included older age, higher diastolic blood pressure, and eGFR  $\leq 30$  mL/min/1.73m<sup>2</sup>.

**Keywords:** Gadolinium; AKI; acute renal failure; GA-AKI; CKD; chronic kidney disease

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# ภาวะไตวายเฉียบพลันจากสารทึบรังสีชนิดแกโดลิเนียม: การศึกษาย้อนหลังเป็นเวลา 10 ปี

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## บทคัดย่อ

**บทนำ:** การศึกษาที่ผ่านมาพบการเกิดความเป็นพิษต่อไตในผู้ป่วยความเสี่ยงสูงหลังได้รับยาฉีดแกโดลิเนียมในบางการศึกษาของยุโรปและอเมริกา ซึ่งปัจจุบันยังไม่มีข้อมูลการศึกษาในประชากรเอเชียที่สามารถยืนยันผลการศึกษาดังกล่าว

**ระเบียบวิธีวิจัย:** การศึกษานี้รวบรวมข้อมูลผู้ป่วยโรคไตเรื้อรังที่มีค่าการทำงานของไตน้อยกว่าหรือเท่ากับ 60 มล./นาที/1.73 ม<sup>2</sup> ที่ได้รับยาฉีดแกโดลิเนียมร่วมในการตรวจวินิจฉัยด้วยเครื่องสแกนแม่เหล็กไฟฟ้า ระหว่างปี พ.ศ. 2556 ถึง พ.ศ. 2566 จากฐานข้อมูลโรงพยาบาลตติยภูมิ เพื่อศึกษาความชุก และปัจจัยที่มีความสัมพันธ์กับการเกิดความเป็นพิษต่อไต

**ผลการวิจัย:** จากผู้ป่วยทั้งหมด 182 ราย พบอุบัติการณ์การเกิดความเป็นพิษต่อไตจากยาฉีดแกโดลิเนียมที่ร้อยละ 4.4 จากการวิเคราะห์ข้อมูลแบบหลายตัวแปรพบปัจจัยที่มีความสัมพันธ์ คือ อายุมากกว่า 65 ปี ที่ 6.374 เท่า ความดันโลหิตตัวล่างมากกว่า 80 มิลลิเมตรปรอท ที่ 6.148 เท่า และ ค่าการทำงานของไตที่น้อยกว่า 30 มล./นาที/1.73 ม<sup>2</sup> ที่ 7.920 เท่า และกราฟทำนายการเกิดความเป็นพิษต่อไตด้วยปัจจัยเสี่ยงดังกล่าว พบคะแนนที่ 2 จาก 3 มีความไวร้อยละ 87.5 ความจำเพาะร้อยละ 78.2

**สรุป:** การศึกษาพบอุบัติการณ์การเกิดความเป็นพิษต่อไตจากยาฉีดแกโดลิเนียมค่อนข้างต่ำ โดยปัจจัยที่มีความสัมพันธ์ ได้แก่ อายุและความดันโลหิตตัวล่างที่สูง และค่าการทำงานของไตที่น้อยกว่า 30 มล./นาที/1.73 ม<sup>2</sup>

**คำสำคัญ:** เครื่องสแกนแม่เหล็กไฟฟ้า; โรคไตเรื้อรัง; ไตวายเฉียบพลัน; ไตวาย; ไตเสื่อม

## Introduction

Radiological diagnostic tools have been used for diagnosing and monitoring treatment outcomes in medicine for a long time. These tools have been continuously developed to reduce side effects caused by the radiation and iodine-based contrast agents associated with acute kidney injury. This development led to the creation of magnetic resonance imaging (MRI) machines. In 1988, the Food and Drug Administration (FDA) approved the use of gadolinium injection in conjunction with MRI diagnostics to enhance diagnostic efficiency<sup>1</sup>. Gadolinium is primarily eliminated through the kidneys. According to the guidelines

of the European Society of Urogenital Radiology (ESUR) in 2018<sup>2</sup> and the American College of Radiology (ACR) in 2024<sup>3</sup>, the risk of nephrotoxicity from gadolinium injection is low if the appropriate dosage is used, i.e., less than or equal to 0.3 millimoles per kilogram of body weight. However, some studies support the occurrence of nephrotoxicity from gadolinium injections in high-risk patients, such as those with heart disease, diabetes, or hypertension. Most studies on the nephrotoxicity of Gadolinium-Based contrast agents (GBCAs) have been conducted in Europe and the United States<sup>4-7</sup>. There is a lack of research on Asian populations to confirm these

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findings. Therefore, the present study investigated the prevalence of acute kidney injury (AKI) associated with GBCAs and the associated risk factors.

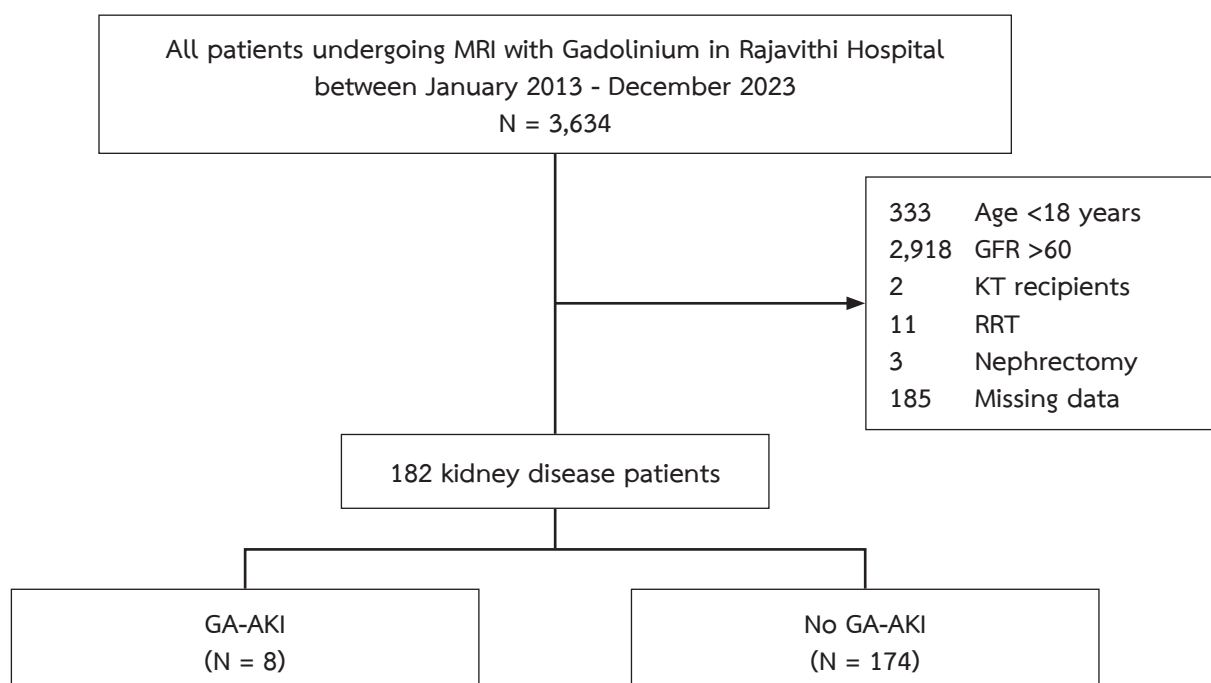
## Materials and methods

### Study design and population

We extracted data from a retrospective single-center cohort using electronic medical records ( $n = 3,634$ ). Chronic kidney disease (CKD) patients with an eGFR of 60 mL/min/1.73m<sup>2</sup> or below who received GBCAs from 2013 to 2023 at Rajavithi Hospital, a tertiary care hospital in Bangkok, Thailand, as shown in **Figure 1**. This study was approved by the Institutional Review Board of the

Research Ethics Committee of Rajavithi Hospital (IRB No. 004/2568). Written informed consent was waived.

The inclusion criteria were as follows: (1) non-dialysis CKD with an eGFR  $\leq 60$  mL/min/1.73m<sup>2</sup> for more than 3 months, (2) adults aged 18 years or older, and (3) those who received GBCAs. The exclusion criteria included: (1) post-kidney transplant status, (2) receiving dialysis, (3) history of nephrectomy or having a single kidney, (4) administration of GBCAs within the past 7 days, and (5) lack of available inpatient or outpatient data. GBCAs refer to contrast agents used in conjunction with MRI. They are classified into two types based on their structure: linear GBCAs and macrocyclic GBCAs.



**Figure 1** Study flow diagram

GA, Gadolinium; AKI, acute kidney injury; RRT, renal replacement therapy; KT, kidney transplant

### Outcomes

The outcomes of the present study were the incidence of gadolinium-associated acute kidney injury (GA-AKI), characterized by an increase in serum creatinine of  $\geq 0.3$  mg/dL or  $\geq 1.5$  times from baseline within 48–72 hours following GBCA administration<sup>8</sup>, and the identification of associated risk factors.

### Sample size calculation

The sample size for this study was calculated based

on a previous study by Takahashi et al. (2018), which reported a 14.8% incidence of gadolinium-induced nephrotoxicity in pre-dialysis chronic kidney disease patients. With a significance level of 0.05 and a margin of error not exceeding 20% of the incidence rate, the required sample size was determined to be 536. To account for an estimated 20% rate of missing data, the final target sample size was adjusted to 644 participants<sup>9</sup>.

### Statistical analysis

Categorical variables were compared using the Chi-square or Fisher's exact test, while continuous variables were analyzed using the independent t-test. Univariate and multivariate logistic regression analyses were performed to identify factors associated with gadolinium-associated acute kidney injury (GA-AKI), with results reported as adjusted odds ratios and 95% confidence intervals. Selected variables were used to develop a predictive scoring system for GA-AKI, and its performance was evaluated using receiver operating characteristic (ROC) curve analysis. All statistical analyses were conducted using IBM SPSS Statistics version 26, and a p-value of less than 0.05 was considered statistically significant.

### Results

A total of 3,634 CKD patients with  $\text{eGFR} \leq 60 \text{ mL/min/1.73m}^2$  received GBCAs between 1 January 2013 and 1 January 2023. Three thousand four hundred fifty-two patients were excluded due to the following reasons:  $\text{eGFR} \geq 60 \text{ mL/min/1.73m}^2$  (n=2,918), kidney transplant status (n=2), renal replacement therapy (n=11),

nephrectomy or single kidney disease (n=3), age <18 years (n=333), and missing data (n=185), resulting in 182 patients finally recruited (**Figure 1**).

Baseline characteristics are shown in **Table 1**. The mean age was  $65.53 \pm 14.62$  years, with a significantly higher age in the AKI group ( $70.13 \pm 4.94$  years) compared to the non-AKI group ( $65.32 \pm 14.89$  years,  $p=0.037$ ). The mean  $\text{eGFR}$  was  $44.83 \pm 11.04 \text{ mL/min/1.73m}^2$ , with a significantly higher percentage of  $\text{eGFR} \leq 30 \text{ mL/min/1.73m}^2$  in the AKI group (50%) compared to the non-AKI group (10.3%,  $p=0.001$ ). A higher percentage of patients in the AKI group had diastolic blood pressure above 80 mmHg (85.5%) compared to the non-AKI group (53.1%,  $p=0.028$ ). There were no statistically significant differences between the two groups in terms of underlying diseases, potential nephrotoxic drugs received concurrently (ACEi, ARB, metformin, SGLT2i, diuretic, aminoglycoside, NSAIDs), or types of GBCAs.

The incidence of GA-AKI was 4.4%, as shown in **Table 2**. The  $\text{eGFR}$  pre- and post-administration of gadolinium-based contrast agents declined by  $12.75 \pm 11.08$  in the AKI group, compared to an improvement of  $11.89 \pm 18.23$  in the non-AKI group ( $p < 0.001$ ).

**Table 1** Baseline characteristics and laboratory data of all patients

Characteristics	Total (n=182)	AKI (n=8)	Non-AKI (n=174)	p-value
Female, n (%)	100 (54.9%)	5 (62.5%)	95 (54.6%)	0.732
Age, years	$65.53 \pm 14.62$	$70.13 \pm 4.94$	$65.32 \pm 14.89$	0.037*
• Age > 65 years, n (%)	100 (54.9%)	7 (87.5%)	93 (53.4%)	0.058
Body weight, kg	$60.98 \pm 13.24$	$59.5 \pm 12.92$	$61.03 \pm 13.3$	0.821
Height, cm	$159.32 \pm 13.15$	$157.33 \pm 8.74$	$159.37 \pm 13.26$	0.792
Body mass index, $\text{kg/m}^2$	$23.77 \pm 4.44$	$24.79 \pm 8.56$	$23.75 \pm 4.36$	0.690
Systolic blood pressure, mmHg	$133.2 \pm 24.41$	$147.87 \pm 30.2$	$132.5 \pm 23.99$	0.082
Diastolic blood pressure (DBP), mmHg	$77.03 \pm 14.81$	$87.12 \pm 18.47$	$76.55 \pm 14.51$	0.048*
• DBP > 80 mmHg, n (%)	67 (36.8%)	6 (75%)	61 (35.1%)	0.028*
<b>Underlying diseases, n (%)</b>				
• Diabetes mellitus	44 (24.2%)	2 (25%)	42 (24.1%)	1
• Diabetic nephropathy	8 (4.4%)	1 (12.5%)	7 (4%)	0.307
• Diabetic retinopathy	5 (2.7%)	1 (12.5%)	4 (2.3%)	0.203
• Peripheral arterial disease	3 (1.6%)	0 (0%)	3 (1.7%)	1

**Table 1** Baseline characteristics and laboratory data of all patients (Continued)

Characteristics	Total (n=182)	AKI (n=8)	Non-AKI (n=174)	p-value
• Hypertension	82 (45.1%)	4 (50%)	78 (44.8%)	1
• Cirrhosis	9 (4.9%)	1 (12.5%)	8 (4.6%)	0.339
• Heart failure	4 (2.2%)	0 (0%)	4 (2.3%)	1
• Urological cancer	6 (3.3%)	0 (0%)	6 (3.4%)	1
• Non-urological cancer	37 (20.3%)	0 (0%)	37 (21.3%)	0.363
<b>Medications, n(%)</b>				
• ACEi	13 (7.1%)	0 (0%)	13 (7.5%)	1
• ARB	12 (6.6%)	1 (12.5%)	11 (6.3%)	0.427
• Metformin	15 (8.2%)	0 (0%)	15 (8.6%)	1
• SGLT2i	1 (0.5%)	0 (0%)	1 (0.6%)	1
• Diuretics	17 (9.3%)	0 (0%)	17 (9.8%)	1
• Aminoglycoside	2 (1.1%)	0 (0%)	2 (1.1%)	1
• NSAIDs	12 (6.6%)	1 (12.5%)	11 (6.3%)	0.427
<b>Types of gadolinium, n (%)</b>				
• Linear non-ionic	15 (8.2%)	2 (25%)	13 (7.5%)	0.133
• Linear ionic	111 (61%)	4 (50%)	107 (61.5%)	0.713
• Cyclic non-ionic	10 (5.5%)	0 (0%)	10 (5.7%)	1
• Cyclic ionic	46 (25.3%)	2 (25%)	44 (25.3%)	1
Blood urea nitrogen, mg/dL	24.11±14.9	19.38±9.98	24.34±15.08	0.359
Creatinine, mg/dL	1.53±0.72	2.46±2.01	1.49±0.59	0.213
<b>CKD Stage, n (%)</b>				
• CKD stage 3a	112 (61.5%)	2 (25%)	110 (63.2%)	0.056
• CKD stage 3b	51 (28%)	3 (37.5%)	48 (27.6%)	0.693
• CKD stage 4	13 (7.1%)	2 (25%)	11 (6.3%)	0.133
• CKD stage 5	2 (1.1%)	1 (12.5%)	1 (0.6%)	0.086
eGFR, mL/min/1.73m <sup>2</sup>	44.83±11.04	33.63±17.51	45.34±10.44	0.101
GFR ≤ 30 mL/min/1.73m <sup>2</sup> , n (%)	22 (12.1%)	4 (50%)	18 (10.3%)	0.001*
Serum bicarbonate, mmol/L	23.13±4.39	24.43 ± 4.61	23.07±4.38	0.425
Serum phosphorus, mmol/L	3.75±1.24	3.63±0.9	3.75±1.26	0.843
Serum albumin, g/L	3.64±0.82	3.69±0.41	3.64±0.83	0.875
Hemoglobin, g/dL	11.16±2.53	12.57±2.07	11.1±2.53	0.165
Fasting blood sugar, mg/dL	145.48±118.85	107±21.21	146.27±119.92	0.646

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; CKD, chronic kidney disease; eGFR, glomerular filtration rate estimated by Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation; NSAIDs, nonsteroidal anti-inflammatory drugs; SGLT2i, sodium glucose co-transporter subtype 2 inhibitors

**Table 2** Laboratory data pre- and post-administration of gadolinium-based contrast agents

Parameters	All (n=182)	AKI (n=8)	Non-AKI (n=174)	p-value
Pre-contrast BUN	24.11±14.9	19.38±9.98	24.34±15.08	0.359
Post-contrast BUN	19.93±11.35	23.63±10.23	19.75±11.4	0.348
• Change in BUN	-4.74±11.42	4.25±8.4	-5.2±11.39	0.022*
Pre-contrast Cr	1.53±0.72	2.46±2.01	1.49±0.59	0.213
Post-contrast Cr	1.34±0.7	3.16±1.97	1.25±0.45	0.029*
• Change in Cr	-0.19±0.56	0.71±0.67	-0.23±0.52	<0.001*
Pre-contrast eGFR	44.83±11.04	33.63±17.51	45.34±10.44	0.101
Post-contrast eGFR	55.64±21.03	20.88±10.78	57.24±19.99	<0.001*
• Change in eGFR	10.81±18.66	-12.75±11.08	11.89±18.23	<0.001*

BUN, blood urea nitrogen; Cr, creatinine; eGFR, glomerular filtration rate estimated by Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation

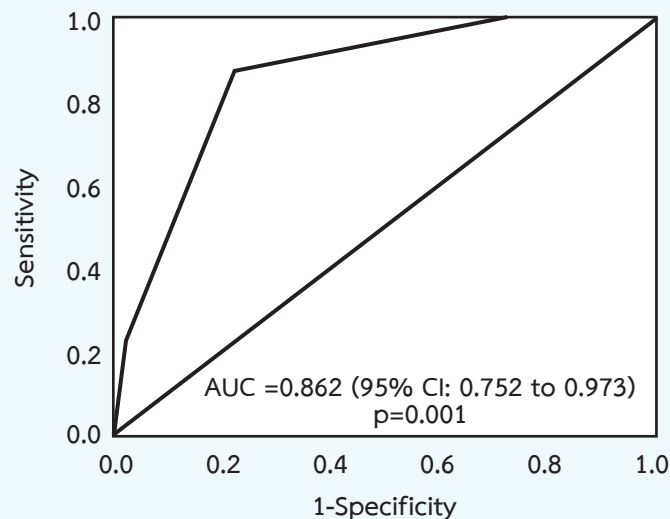
Multivariate logistic regression analysis was conducted to identify risk factors associated with GA-AKI. Statistically significant factors identified included age >65 years, diastolic blood pressure (DBP) >80 mmHg, and eGFR  $\leq 30$  mL/min/1.73m<sup>2</sup>, as shown in Table 3. The accuracy of the risk score was tested using the ROC analysis and evaluating the area under

the curve, as shown in **Figure 2**. The risk score comprised three variables: age >65 years, DBP < 80 mmHg, and eGFR  $\leq 30$  mL/min/1.73m<sup>2</sup>, with a value of 1 if the factor was present and 0 if it was absent. A score of 2 out of 3 predicted GA-AKI with a sensitivity of 87.5% and a specificity of 78.2% (**Table 4**).

**Table 3** Multivariate analysis of factors associated with gadolinium-associated acute kidney injury

	Adjusted Odds Ratio	95% Confidence Interval		p-value	Score (0-3)
		Lower	Upper		
Age >65	6.374	0.720	56.426	0.096	1
DBP >80	6.148	1.111	34.008	0.037*	1
eGFR $\leq 30$	7.920	1.642	38.196	0.010*	1

DBP, diastolic blood pressure; eGFR, glomerular filtration rate



**Figure 2** Receiver operating characteristic curve analysis  
AUC, area under the curve; CI, confidence interval

**Table 4** Predicted values at different cut-off points

Cut-off	Sensitivity	Specificity	PPV	NPV	Accuracy	LR+	LR-	Youden index
0	100.0%	0.0%	4.4%	N/S	4.4%	1.00	N/A	0.000
1	100.0%	27.0%	5.9%	100.0%	30.2%	1.37	0.00	0.270
2	87.5%	78.2%	15.6%	99.3%	78.6%	4.01	0.16	*0.657
3	25.0%	97.7%	33.3%	96.6%	94.5%	10.88	0.77	0.227

PPV, positive predictive value; NPV, negative predictive value; LR, likelihood ratio

## Discussion

This study found that the incidence of GA-AKI was 4.4%, which aligns with previous studies in Europe and America, where the reported range is from 3.5% to 28%<sup>10-14</sup>. The incidence of GA-AKI is lower than in the previous study, likely due to a higher baseline eGFR. However, it can be observed that in the group that developed GA-AKI, the eGFR values were similar to those in the previous study. Several associated factors are described in earlier studies, both for iodine-based and GBCAs. In the subgroup analysis, we first focused on patient factors, including sex, age, underlying diseases, and current medications. Second, we examined the characteristics of GBCAs, such as drug structure. In our study, patients only received gadolinium via the venous route, so we could

not identify differences associated with the arterial route. According to the ESUR 2018 and ACR 2024 guidelines, a low incidence of GA-AKI is observed with a gadolinium dose lower than 0.3 millimole per kilogram of body weight. In our center, we used a dose range of 0.1-0.2 millimole per kilogram of body weight. Third, we analyzed baseline laboratory data before contrast administration, including creatinine, estimated glomerular filtration rate, serum bicarbonate, serum phosphate, serum albumin, hemoglobin level, and proteinuria.

In the univariate analysis, we found that age >65 years, diastolic blood pressure >80 mmHg, and an eGFR ≤30 mL/min/1.73m<sup>2</sup> are factors associated with the development of GA-AKI. This observation suggests that functional renal dysfunction may be attributed to the physiologic



consequence of aging and the use of nephrotoxic drugs in the past in older patients, which are not used in the present. Upon reviewing previous studies on blood pressure and changes in renal function, elevated systolic blood pressure and/or pulse pressure, indicative of arterial stiffness, were associated with a more rapid decline in kidney function. In contrast, this association was not observed in the isolated high diastolic blood pressure group. As in previous literatures<sup>13</sup>, a greater reduction in eGFR is associated with a higher risk of GA-AKI. However, there was no significant difference in chronic kidney disease risk factors, including diabetic nephropathy and hypertension. Ihsan Ergün et al. retrospectively studied 91 patients with chronic kidney disease stages 3 and 4, with a mean eGFR of 33 mL/min/1.73m<sup>2</sup>, and demonstrated GA-AKI in 11 of 91 patients (12.1%), defined by an increase in creatinine level of more than 0.5 mg/dL within 24-72 hours. They identified risk factors including advanced age, diabetic nephropathy, low hemoglobin levels, and low serum albumin levels. In contrast to our study, we could not demonstrate an association between GA-AKI and baseline hemoglobin or serum albumin levels. To date, the American College of Radiology divides gadolinium-based contrast agents into Group I, Group II, and Group III agents according to their molecular structure, distinguishing between cyclic and linear forms. Group II agents are recommended for use due to their lower nephrotoxicity<sup>15</sup>. However, our study could not find statistically significant results, possibly due to the limitation that linear agents are not currently in use.

This study has several limitations inherent to its retrospective design. Notably, the absence of a control group and the failure to achieve the calculated sample size may compromise the robustness of the findings. The insufficient sample size was primarily due to substantial missing data. Despite these constraints, our analysis identified factors associated with GA-AKI in patients of advanced age, those with moderate to severe renal insufficiency, and those with elevated diastolic blood pressure. Based on these associations, we developed a risk score incorporating three variables: age >65, DBP >80 mmHg, and eGFR ≤ 30 mL/min/1.73m<sup>2</sup>. A score of

2 out of 3 was predictive of GA-AKI. However, the occurrence of only eight AKI events limits the study's statistical power and may preclude a reliable multivariable analysis.

In conclusion, our study demonstrated an incidence of GA-AKI of 4.4%. Subgroup analysis revealed that age >65 years, DBP >80 mmHg, and an eGFR ≤30 mL/min/1.73m<sup>2</sup> were associated with a higher likelihood of developing GA-AKI. The predicted risk score may be applicable in clinical practice, and one of the three modifiable factors associated with GA-AKI is elevated diastolic blood pressure (above 80 mmHg). Better control of diastolic blood pressure is therefore likely to provide more benefits than risks for patients. However, given the limitations of this dataset, particularly the relatively small number of patients who developed GA-AKI in the study, the scoring system may be prone to overprediction. A well-designed and longer prospective study may be required to validate this hypothesis.

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