Prevalence and Associated Factors of Contrast-Associated Acute Kidney Injury in Kidney Disease: A Retrospective Cohort Study

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Abstract

Background: Research indicates that the intravascular administration of contrast agents during computed tomography (CT) procedures may lead to Contrast-Associated Acute Kidney Injury (CA-AKI). The prevalence and associated risk factors for CA-AKI vary among different populations, particularly in individuals with pre-existing kidney disease.

Methods: This retrospective cohort study included patients with an estimated glomerular filtration rate (eGFR) between 15 and 59 mL/min/1.73 m² who received intravenous contrast medium (CM) for CT between October 2021 and September 2024. The prevalence of CA-AKI in patients with kidney disease was evaluated, and associated risk factors were analyzed.

Results: A total of 655 patients met the inclusion criteria. Among them, 58 patients (8.58%) developed CA-AKI. Baseline demographic characteristics were not significantly different between the CA-AKI and non-CA-AKI groups. However, the CA-AKI group had significantly higher proportions of patients with chronic kidney disease (CKD) stage G3b and G4, current use of diuretics and vasopressors, multiple exposures to CM within 72 hours, and concurrent acute kidney injury (AKI). Multivariate analysis identified CKD stage G3b (odds ratio [OR] 2.75; 95% confidence interval [CI], 1.26–6.00; p = 0.011) and current AKI (OR 3.99; 95% CI, 1.89–8.42; p < 0.001) as significant factors associated with the development of CA-AKI.

Conclusions: CKD stage G3b and current AKI were significantly associated with an increased risk of CA-AKI. Therefore, caution is warranted when administering CM to patients with these conditions.

Keywords: CIN; renal failure; acute renal failure; kidney failure; contrast-induced nephropathy

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ความชุกและปัจจัยที่สัมพันธ์กับภาวะไตวายเฉียบพลันหลังการฉีดสารทึบรังสีในโรคไต: การศึกษาแบบย้อนหลัง

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บทคัดย่อ

บทนำ: การศึกษาก่อนหน้านี้พบว่า การฉีดสารทึบรังสีผ่านทางหลอดเลือดในการตรวจเอกซเรย์คอมพิวเตอร์ อาจส่งผลต่อการเกิดภาวะไตวายเฉียบพลันได้ ความชุกและปัจจัยเสี่ยงของภาวะไตวายเฉียบพลันหลังการฉีดสารทึบรังสี (Contrast-Associated Acute Kidney Injury; CA-AKI) มีความแตกต่างกันไปตามลักษณะของประชากร โดยเฉพาะในผู้ป่วยโรคไตเรื้อรัง

ระเบียบวิธีวิจัย: การวิเคราะห์ข้อมูลย้อนหลังในผู้ป่วยที่มีค่าประมาณอัตราการกรองของไตระหว่าง 15 ถึง 59 มล./นาที/1.73 ม^2 และได้รับสารทึบรังสีทางหลอดเลือดสำหรับการตรวจเอกซเรย์คอมพิวเตอร์ ตั้งแต่เดือนตุลาคม 2564 ถึง กันยายน 2567 เพื่อศึกษาความชุกและวิเคราะห์ปัจจัยที่เกี่ยวข้องกับการเกิดภาวะ CA-AKI

ผลการวิจัย: ผู้ป่วยที่เข้าเกณฑ์ทั้งหมด 655 ราย โดยในจำนวนนี้มีผู้ป่วย 58 ราย (ร้อยละ 8.58) ที่ได้รับการวินิจฉัยว่ามีภาวะ CA-AKI กลุ่มผู้ป่วยที่มีภาวะ CA-AKI มีสัดส่วนของผู้ที่มีโรคไตเรื้อรังระยะ 3b และ 4 การใช้ยาขับปัสสาวะหรือยากระตุ้นความดันโลหิต การได้รับสารทึบรังสีมากกว่าหนึ่งครั้งภายใน 72 ชั่วโมง และการเกิดภาวะไตวายเฉียบพลันขณะได้รับสารทึบรังสี สูงกว่ากลุ่มที่ไม่มีภาวะ CA-AKI ปัจจัยที่มีความสัมพันธ์กับการเกิดภาวะ CA-AKI อย่างมีนัยสำคัญทางสถิติ ได้แก่ โรคไตเรื้อรังระยะ 3b (odds ratio [OR] 2.75; 95% confidence interval [CI], 1.26–6.00; p = 0.011) และภาวะไตวายเฉียบพลันขณะได้รับสารทึบรังสี (OR %95 ;3.99 CI, 8.42–1.89; p < 0.001)

สรุป: ผู้ป่วยที่มีโรคไตเรื้อรังระยะ 3b และผู้ที่เกิดภาวะไตวายเฉียบพลันขณะได้รับสารทึบรังสี มีความเสี่ยงสูงต่อการเกิด CA-AKI ดังนั้นควรให้การป้องกันและติดตามอย่างใกล้ชิด หากจำเป็นต้องได้รับการฉีดสารทึบรังสี

คำสำคัญ: ไตวายเฉียบพลัน; ไตวาย; ไตเสื่อม; ไตบาดเจ็บ; ฉีดสี

Introduction

Currently, radiographic imaging with contrast medium (CM) through computed tomography (CT) scans is essential for diagnosing and planning the treatment of various diseases. Previous studies have shown that the injection of contrast agents through the bloodstream during CT scans can lead to acute kidney injury (AKI), known as Contrast-Associated Acute Kidney Injury (CA-AKI).[1,2] The incidence rate of CA-AKI ranges from 6% to 24%, varying across different studies and populations. The primary risk factor for CA-AKI is the estimated glomerular filtration rate (eGFR), with additive risk factors including advanced age, pre-existing kidney dysfunction, chronic diseases such as diabetes, chronic kidney disease (CKD), and hemodynamic instability before receiving contrast agents. The type and amount of CM or multiple CM administrations within 72 hours may increase the risk of CA-AKI.[3,4,5] There are currently numerous

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guidelines to prevent CA-AKI following the intravascular administration of contrast agents for CT scans.

Rajavithi Hospital is the largest hospital under the Department of Medical Services, Ministry of Public Health, Bangkok, Thailand. It receives referrals from provincial hospitals, resulting in a high volume of patients receivingintravenous CM for CT scans. Many patients have reduced kidney function based on blood tests before CM administration, putting them at high risk for CA-AKI. These patients must be assessed by nephrologists to prevent this condition before contrast administration.[1,2,4,6]

Previous studies have shown that AKI following the administration of contrast agents during CT scans is associated with an eGFR less than 30 ml/min/1.73 m².[7,8] However, some research shows no evidence of CA-AKI regardless of CKD stage.[9,10] Current guidelines recommend various measures to prevent CA-AKI in patients with CKD, but the specific eGFR threshold for this condition varies. The Kidney Disease Improving Global Outcomes (KDIGO) 2012 guidelines[1] recommend monitoring for CA-AKI when eGFR is less than 60 ml/min/1.73 m². However, the American College of Radiology (ACR) 2023 guidelines[2] and Japan's national guidelines suggest monitoring for CA-AKI when eGFR is less than 30 ml/min/1.73 m².[6] Additionally, the European Society of Urogenital Radiology (ESUR) 2018 guidelines recommend different thresholds for monitoring CA-AKI depending on the route of contrast administration.[4] For intra-arterial contrast administration or patients in critical care units, monitoring for CA-AKI is recommended when eGFR is less than 45 ml/min/1.73 m². For intravenous contrast administration, monitoring is recommended when eGFR is less than 30 ml/min/1.73 m².

From the differing guidelines and the large volume of patients at Rajavithi Hospital, many patients require consultation with nephrologists before administering contrast agents or might experience delays in necessary diagnostic procedures involving CT scans. Some patients may develop CA-AKI without receiving appropriate preventative measures. This study examines the factors influencing CA-AKI in patients with an eGFR of less than 60 ml/min/1.73 m^2 who received intravenous contrast agents for CT scans at Rajavithi Hospital. The goal is to develop appropriate guidelines to prevent CA-AKI in Rajavithi Hospital in the future.

Material and methods

This retrospective single-center cohort study was conducted at Rajavithi Hospital. It included patients with kidney disease defined as chronic kidney disease (CKD) stage III–IV and those with acute kidney injury who underwent computed tomography (CT) with iodinated contrast media between October 2021 and September 2024. The study was approved by the Ethics Committee of Rajvithi Hospital. Written informed consent was not required.

Study population

Eligible participants were adults aged 18 years or older with an estimated glomerular filtration rate (eGFR) between 15–59 ml/min/1.73 m². Patients were excluded if baseline serum creatinine data within 3 months or follow-up data 48–72 hours after contrast administration were unavailable, if they were undergoing kidney replacement therapy, or if they were pregnant.

Patients were classified as having AKI if their serum creatinine increased by ≥0.3 mg/dL from baseline during CM exposure. CKD was defined as a preexisting estimated glomerular filtration rate (eGFR) of 15–59 ml/min/1.73 m² within three months before CM exposure. Stable CKD was defined as a <0.3 mg/dL change in serum creatinine from baseline during CM exposure.

Outcomes

The primary outcome of the study was to determine the prevalence of contrast-associated acute kidney injury (CA-AKI) following intravascular contrast administration in patients with kidney disease. The secondary outcome was to identify factors associated with the development of CA-AKI in this population. CA-AKI was defined as serum creatinine increase ≥ 0.3 mg/dL within 48 - 72 hours after intravascular CM exposure

Contrast media

Contrast media used in the study included iso-osmolar contrast media (IOCM), such as iodixanol (Visipaque), and low-osmolar contrast media (LOCM), including iopamiro, ioversol (Optiray), and iohexol (Omnipaque). The amount of CM used for CT scans was based on the type of imaging and patient body weight. For CT of the brain, head, and neck, 50 ml of CM was administered. For CT of the chest, upper abdomen, or whole abdomen, 80 ml was used. In CT angiography (CTA) of the brain, neck, chest, or abdomen, patients weighing less than 80 kg received 80 ml, while those weighing 80 kg or more received 100 ml of CM.

Sample size calculation

The sample size was calculated using the estimated single proportion formula (Wayne WD, 1995),[11] based on a reported 21.2% prevalence of acute kidney injury after contrast media administration in patients with eGFR < 60 ml/min/1.73 m², as reported by McDonald et al. (2017). To ensure sufficient power and account for a 20% margin of error and potential missing data, the final target sample size was determined to be 436 patients.

Statistical analysis

Descriptive statistics were used to summarize baseline demographic data, with categorical variables presented as numbers and percentages. Continuous variables were expressed as mean and standard deviation for normally distributed data, and as median, minimum, maximum, and interquartile range (IQR) for non-normally distributed data.

For inferential statistics, categorical variables were analyzed using the Chi-square or Fisher's exact test. For continuous variables, the Paired t-test was used for normally distributed paired data, and the Wilcoxon Signed Rank test for non-normal paired data. For independent groups, the Student's t-test was applied for normally distributed data, and the Mann–Whitney U-test for non-normally distributed data. Pearson's correlation was used to assess relationships between continuous variables such as age, serum creatinine, and eGFR. Factors associated with CA-AKI were analyzed using multiple logistic regression, with results presented as adjusted odds ratios and 95% confidence intervals. A p-value of less than 0.05 was considered statistically significant, and all analyses were conducted using SPSS Statistics version 29.0.2.0.

Results

One thousand one hundred seventy-one patients met the inclusion criteria. After applying the exclusion criteria, 516 patients were excluded, including 321 patients with missing data, 176 patients who received kidney replacement therapy, 10 patients who had undergone kidney transplantation, and nine patients with end-stage kidney disease who had not yet undergone dialysis. Therefore, 655 patients were included in the final analysis. Among these, 58 patients were diagnosed with CA-AKI, accounting for 8.9% of the study population **(Figure 1)**.

The baseline characteristics between the AKI and no AKI groups showed no significant differences in age, sex, body mass index, blood pressure, preexisting diseases, hemoglobin, hematocrit, types of intravenous (IV) fluid, and N-acetylcysteine administration before CM exposure, and types of CM used. The proportions of patients with stable CKD stages G3b and G4, current use of diuretics and vasopressor agents during CM exposure, multiple CM exposures within 72 hours, current AKI, and admission to critical care units and emergency rooms during CM exposure, as well as baseline serum creatinine before CM exposure were significantly higher in the AKI group compared to the non-AKI group. Conversely, there was a substantially higher proportion of current use of angiotensin-converting enzyme inhibitors (ACEIs), metformin, stable CKD stage G3a, and admission in the outpatient departments, as well as higher serum albumin level in the non-AKI group **(Table 1)**.

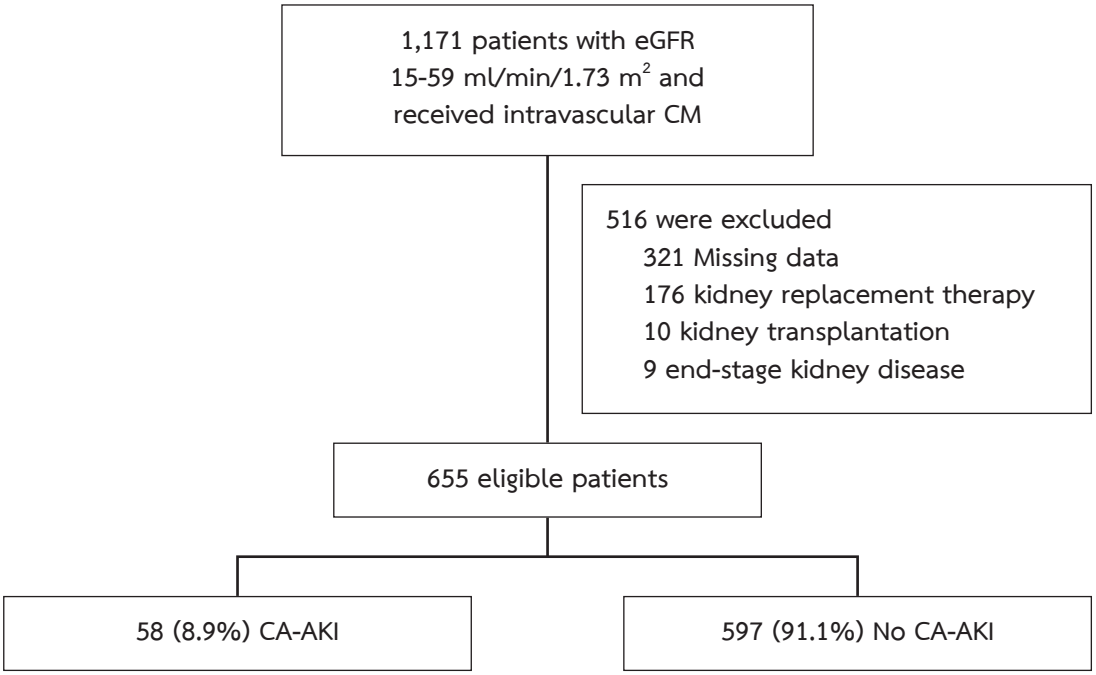

Figure 1 Study Flow Diagram

eGFR, estimated glomerular filtration rate; CM, contrast media; CA-AKI, contrast-associated acute kidney injury

Table 1 Baseline demographic and laboratory data of all patients

Parameters	AKI (n=58)	Non-AKI (n=597)	p-value
Age, year	66.93±14.86	68.76±12.26	0.288
Sex, n (%)			0.167
• Female	33 (56.9%)	283 (47.4%)	
• Male	25 (43.1%)	314 (52.6%)	
Body mass index (kg/m^2)	23.64±4.62	23.41±4.61	0.757
Systolic blood pressure (mmHg)	131.04±26.52	133.12±21.43	0.571
Diastolic blood pressure (mmHg)	74.63±16.8	74.89±13.29	0.909
Preexisting disease, n (%)			
• Hypertension	38 (65.5%)	371 (62.1%)	0.613
• Diabetes mellitus	20 (34.5%)	154 (25.8%)	0.153
• Solid malignancy	12 (20.7%)	143 (24%)	0.577
• Hematologic malignancy	3 (5.2%)	15 (2.5%)	0.237
• Cirrhosis	1 (1.7%)	15 (2.5%)	0.71
• Chronic kidney disease stages	43 (74.1%)	462 (77.4%)	0.574
- G3a	15 (25.9%)	297 (49.7%)	
- G3b	21 (36.2%)	122 (20.4%)	
- G4	7 (12.1%)	43 (7.2%)	

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

Parameters	AKI (n=58)	Non-AKI (n=597)	p-value
Current medications, n (%)	22 (37.9%)	170 (28.5%)	0.131
• Angiotensin-Converting Enzyme Inhibitor	2 (3.4%)	53 (8.9%)	0.031*
- Continued	1 (1.7%)	20 (3.4%)	
- Withdrew	1 (1.7%)	26 (4.4%)	
• Angiotensin II Receptor Blocker	7 (12.1%)	55 (9.2%)	0.960
- Continued	2 (3.4%)	23 (3.9%)	
- Withdrew	5 (8.6%)	24 (4%)	
• Metformin	2 (3.4%)	53 (8.9%)	0.031*
- Continued	0 (0%)	19 (3.2%)	
- Withdrew	2 (3.4%)	24 (4%)	
• Sodium-Glucose Cotransporter 2 Inhibitors	0 (0%)	3 (0.5%)	0.53
- Continued	0 (0%)	2 (0.3%)	
- Withdrew	0 (0%)	1 (0.1%)	
• Diuretics	13 (22.4%)	59 (9.9%)	0.026*
- Continued	8 (13.8%)	29 (4.9%)	
- Withdrew	5 (8.6%)	27 (4.5%)	
Wards, n (%)			<0.001*
• Outpatient department	10 (17.2%)	215 (36%)	
• Emergency room	15 (25.9%)	109 (18.3%)	
• Critical care unit	4 (6.9%)	6 (1%)	
• General ward	29 (50%)	267 (44.7%)	
IV fluid before CM exposure, n (%)			0.313
• No	19 (32.8%)	236 (39.5%)	
• Yes	39 (67.2%)	361 (60.5%)	
Types of IV fluid before CM exposure, n (%)			0.087
• Normal saline	25 (43.1%)	281 (47.1%)	
• Acetar/Ringer's lactate	7 (12.1%)	41 (6.9%)	
• Isotonic bicarbonate	1 (1.7%)	1 (0.2%)	
• 5% glucose in NSS/0.45% NSS	6 (10.3%)	38 (6.4%)	
N-acetylcysteine before CM exposure	30 (51.7%)	351 (58.8%)	0.297

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

Parameters	AKI (n=58)	Non-AKI (n=597)	p-value
Vasopressor during CM exposure	7 (12.1%)	9 (1.5%)	<0.001*
Multiple CM exposures within 72 hours	2 (3.4%)	3 (0.5%)	0.014*
Injection site, n (%)			0.065
• Intravenous	56 (96.6%)	592 (99.2%)	
• Intraarterial	2 (3.4%)	5 (0.8%)	
Types of Imaging, n (%)			0.006*
• Computed tomography	42 (72.4%)	519 (86.9%)	
• Computed tomography angiography	14 (24.1%)	73 (12.2%)	
• Angiogram	2 (3.4%)	5 (0.8%)	
Types of contrast media, n (%)			0.811
• Low osmolar	6 (10.3%)	56 (9.4%)	
• Iso-osmolar	52 (89.7%)	541 (90.6%)	
• Low osmolar			0.285
- Iopamiro	0 (0%)	5 (0.8%)	
- Iohexol (Omnipaque)	6 (10.3%)	39 (6.5%)	
- Ioversol (Optiray)	0 (0%)	12 (2%)	
Stable chronic kidney disease stages			<0.001*
• G3a	5 (8.6%)	262 (43.9%)	
• G3b	11 (19%)	101 (16.9%)	
• G4	5 (8.6%)	34 (5.7%)	
Current AKI during CM exposure	35 (60.3%)	151 (25.3%)	<0.001*
Hemoglobin (g/dL)	10.49±2.84	11.02±2.27	0.170
Hematocrit (%)	32.45±8.49	33.56±6.89	0.255
Serum albumin (g/dL)	3.21±0.9	3.63±0.74	0.001*
Serum creatinine before CM exposure (mg/dL)	1.8±0.57	1.52±0.51	<0.001*
eGFR before CM exposure (ml/min/1.73m^2)	35.91±11.28	44.08±11.34	<0.001*
Death within 30 days	23 (39.7%)	36 (6%)	<0.001*
RRT within 30 days	6 (10.3%)	1 (0.2%)	<0.001*

IV, intravenous; CM, contrast media; NSS, normal saline; AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; RRT, renal replacement therapy. *p-value < 0.05

Univariate analysis of associated factors of CA-AKI revealed CKD stages G3b/G4, current AKI, current use of diuretics and vasopressors, multiple CM exposures within 72 hours, types of imaging, admission wards, baseline serum albumin, creatinine, and eGFR before CM exposure as significant predictors for CA-AKI **(Table 2)**.

Table 2 Univariate analysis of associated factors for contrast-associated acute kidney injury

Parameters	OR 95%CI	p-value
Age, years	0.99 (0.97, 1.01)	0.288
Sex (%)		
• Female	Reference	1
• Male	0.68 (0.4, 1.18)	0.169
Body mass index (kg/m^2)	1.01 (0.95, 1.08)	0.756
Systolic blood pressure (mmHg)	1 (0.98, 1.01)	0.497
Diastolic blood pressure (mmHg)	1 (0.98, 1.02)	0.889
Underlying diseases		
• Diabetes mellitus	1.51 (0.86, 2.68)	0.155
• Solid malignancy	0.83 (0.43, 1.61)	0.577
• Hematologic malignancy	2.12 (0.59, 7.54)	0.247
• Hypertension	1.16 (0.66, 2.04)	0.613
• Coronary artery disease	0.84 (0.45, 1.55)	0.574
• Cirrhosis	0.68 (0.09, 5.25)	0.712
• Chronic kidney disease stages		
- G3a	Reference	1
- G3b	3.41 (1.7, 6.83)	0.001*
- G4	3.22 (1.24, 8.35)	0.016*
Stable chronic kidney disease stages		
• No chronic kidney disease	Reference	1
- G3a	0.1 (0.04, 0.27)	<0.001*
- G3b	0.59 (0.29, 1.2)	0.146
- G4	0.79 (0.29, 2.17)	0.654
Current AKI during CM exposure	4.49 (2.57, 7.85)	<0.001*
Current medications		
• Angiotensin-Converting Enzyme Inhibitor	0.22 (0.05, 0.98)	0.047*
• Angiotensin II Receptor Blocker	0.98 (0.38, 2.53)	0.960
• Metformin	0.22 (0.05, 0.98)	0.047*
• Diuretics	2.72 (1.1, 6.73)	0.031*
Wards		
• Outpatient department	Reference	1
• Emergency room	2.34 (1.11, 4.9)	0.025*
• Critical care unit	2.96 (1.29, 6.8)	0.011*
• General ward	14.33 (3.48, 59.01)	<0.001*
Intravenous fluid before CM exposure		
• No	Reference	1
• Yes	1.34 (0.76, 2.38)	0.314
N-acetylcysteine before CM exposure	0.75 (0.44, 1.29)	0.299
Vasopressor during CM exposure	8.97 (3.21, 25.08)	<0.001*
Multiple CM exposures within 72 hours	7.07 (1.16, 43.21)	0.034*
Route of contrast media		
• Intravenous	Reference	1
• Intraarterial	4.23 (0.8, 22.3)	0.089
Types of Imaging		
• Computed tomography	Reference	1
• Computed tomography angiography	2.37 (1.23, 4.55)	0.010*
• Angiogram	4.94 (0.93, 26.25)	0.061
Types of contrast media		
• Low osmolar	Reference	1
• Iso-osmolar	0.9 (0.37, 2.18)	0.811
Hemoglobin (g/dL)	0.91 (0.8, 1.02)	0.098
Hematocrit (%)	0.98 (0.94, 1.02)	0.254
Serum albumin (g/dL)	0.51 (0.36, 0.72)	<0.001*
Serum creatinine before CM exposure (mg/dL)	2.11 (1.41, 3.16)	<0.001*
eGFR before CM exposure (ml/min/1.73m^2)	0.95 (0.93, 0.97)	<0.001*

IV, intravenous; CM, contrast media; AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; OR, odds ratio; CI, confidence interval. *p-value < 0.05

For multivariate logistic regression analysis, those with a p-value < 0.1 from univariate analysis and other relevant factors, such as preexisting diseases, were included as covariates. It was found that baseline CKD stage G3b and current AKI status were significant predictors of CA-AKI. The adjusted odds ratios were 2.75 (95% CI 1.26-6.0, p=0.011) for CKD stage G3b and 3.99 (95% CI 1.89-8.42, p <0.001) for current AKI status. **(Table 3)**.

Table 3 Multivariate logistic regression analysis of associated factors for contrast-associated acute kidney injury

Parameters	Adjusted OR 95%CI	p-value
Preexisting diseases		
• Diabetes mellitus	1.5 (0.71, 3.17)	0.284
• Chronic kidney disease stages		
- G3a	Reference	1
- G3b	2.75 (1.26, 6)	0.011*
- G4	2.79 (0.99, 7.87)	0.053
Current AKI during CM exposure	3.99 (1.89, 8.42)	<0.001*
Current medication		
• Diuretics	1.81 (0.73, 4.45)	0.197
Vasopressor during CM exposure	2.15 (0.35, 12.99)	0.406
Multiple CM exposures within 72 hours	4.07 (0.25, 67.1)	0.327
Route of contrast media		
• Intravenous	Reference	1
• Intraarterial	1.11 (0.08, 15.42)	0.938
Types of Imaging		
• Computed tomography	Reference	1
• Computed tomography angiography	0.54 (0.23, 1.29)	0.168
• Angiogram	NA	1
Serum albumin (g/dL)	0.82 (0.5, 1.33)	0.412

CM, contrast media; AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; OR, odds ratio; CI, confidence interval. *p-value < 0.05.

In the subgroup analysis of stable CKD patients, univariate analysis identified diabetes mellitus, hematologic malignancy, CKD stages, current diuretic use, admission to the critical care unit, baseline serum creatinine, and eGFR before CM exposure as significant predictors of CA-AKI **(Table 4).** Multiple logistic regression showed that hematologic malignancy and CKD stage G3b were significant independent predictors for CA-AKI. Factors included in the analysis were diabetes mellitus, hematologic malignancy, CKD, ward status, and eGFR before CM exposure. The adjusted odds ratios were 11.3 (95% CI 1.5-85.2, p=0.019) for hematologic malignancy, and 4.49 (95% CI 1.02-19.78, p=0.047) for CKD G3b **(Table 5)**.

Table 4 Univariate analysis of associated factors for contrast-associated acute kidney injury in the subgroup of stable chronic kidney disease patients

Parameters	OR 95%CI	p-value
Age, year	1.01 (0.97, 1.05)	0.651
Sex (%)		
• Female	Reference	1
• Male	0.51 (0.2, 1.25)	0.138
Body mass index (kg/m^2)	0.92 (0.81, 1.05)	0.238
Systolic blood pressure (mmHg)	1 (0.98, 1.03)	0.652
Diastolic blood pressure (mmHg)	0.98 (0.94, 1.02)	0.252
Preexisting diseases		
• Diabetes mellitus	2.17 (0.89, 5.3)	0.089
• Solid malignancy	1.8 (0.73, 4.48)	0.204
• Hematologic malignancy	5.86 (1.14, 30.16)	0.034*
• Hypertension	2.09 (0.69, 6.35)	0.192
• Coronary artery disease	N/A	1
• Cirrhosis	1.75 (0.22, 14.27)	0.599
• Chronic kidney disease stages		
- G3a	Reference	1
- G3b	5.71 (1.93, 16.83)	0.002*
- G4	7.71 (2.12, 28)	0.002*

Table 4 Univariate analysis of associated factors for contrast-associated acute kidney injury in the subgroup of stable chronic kidney disease patients (continued)

Parameters	OR 95%CI	p-value
Current medications		
• Angiotensin-Converting Enzyme Inhibitor	N/A	0.998
• Angiotensin II Receptor Blocker	1.44 (0.38, 5.44)	0.587
• Metformin	N/A	0.998
• Diuretics	4.01 (1.47, 10.98)	0.007*
Admission wards		
• Outpatient department	Reference	1
• General ward	2.55 (0.87, 7.49)	0.088
• Emergency room	3.92 (0.89, 17.26)	0.071
• Critical care unit	39.2 (4.56, 337.06)	0.001*
Intravenous fluid before CM exposure		
• No	Reference	1
• Yes	1.47 (0.6, 3.56)	0.396
Types of IV fluid before CM exposure, n (%)		
• Normal saline	0.62 (0.07, 5.34)	0.662
• Acetar/Ringer's lactate	Reference	1
• Isotonic bicarbonate	N/A	0.998
• 5% glucose in NSS/ 0.45% NSS	N/A	1
N-acetylcysteine before CM exposure	0.93 (0.37, 2.36)	0.878
Multiple CM exposures within 72 hours	9.87 (0.86, 113.54)	0.066
Injection site, n (%)		
• Intravenous	Reference	1
• Intraarterial	N/A	0.999
Types of Imaging, n (%)		
• Computed tomography	Reference	1
• Computed tomography angiography	1.15 (0.33, 4.03)	0.831
• Angiogram	0 (0, 1)	0.999
Types of contrast media, n (%)		
• Low osmolar	Reference	1
• Iso-osmolar	0.72 (0.16, 3.25)	0.670

Table 4 Univariate analysis of associated factors for contrast-associated acute kidney injury in the subgroup of stable chronic kidney disease patients (continued)

Parameters	OR 95%CI	p-value
Current AKI during CM exposure	N/A	1
Hemoglobin (g/dL)	0.95 (0.77, 1.18)	0.656
Hematocrit (%)	1 (0.94, 1.08)	0.898
Serum albumin (g/dL)	0.63 (0.34, 1.14)	0.124
Serum creatinine before CM exposure (mg/dL)	2.17 (1.08, 4.36)	0.029*
eGFR before CM exposure (ml/min/1.73m^2)	0.94 (0.91, 0.98)	0.001*

IV, intravenous; NSS, normal saline; CM, contrast media; AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; OR, odds ratio; CI, confidence interval. *p-value < 0.05.

Table 5 Multivariate analysis of associated factors with CA-AKI in the subgroup of stable CKD patients

Parameters	Adjusted OR 95%CI	p-value
Preexisting disease		
• Diabetes mellitus	2.07 (0.75, 5.7)	0.161
• Hematologic malignancy	11.3 (1.5, 85.2)	0.019*
• Chronic kidney disease stages		
- G3a	Reference	1
- G3b	4.49 (1.02, 19.78)	0.047*
- G4	5.37 (0.4, 72.58)	0.206
Wards		
• Outpatient department	Reference	1
• General ward	2.3 (0.71, 7.44)	0.164
• Emergency room	4.21 (0.85, 20.76)	0.077
• Critical care unit	9.53 (0.76, 119.85)	0.081
eGFR before CM exposure, ml/min/1.73m^2	0.99 (0.92, 1.08)	0.887

CM, contrast media; eGFR, estimated glomerular filtration rate; OR, odds ratio; CI, confidence interval. *p-value < 0.05.

This study found that the 30-day mortality rate in the AKI group was 39.7%, predominantly affecting those with current AKI at 69.6%. Among patients with stable CKD who died, many had accompanying conditions such as cancer or sepsis. In contrast, the non-AKI group had a mortality rate of 6%. The study observed renal replacement therapy (RRT) within 30 days in the AKI group at a rate of 10.3%, with 50% of these patients having current AKI during CM exposure. In the stable CKD group, three cases requiring RRT were noted: The first case involved a patient with sepsis, indicated for hemodialysis due to volume overload, who subsequently died. The second case involved a patient with metastatic breast cancer, sepsis, and liver failure, indicated for hemodialysis due to severe metabolic acidosis. The third case involved a patient with CA-AKI along with NSAIDs, ciprofloxacin, and cotrimoxazole exposure, requiring hemodialysis due to uremia. This patient was able to discontinue hemodialysis within two weeks, with kidney function returning to baseline. In the non-AKI group, 0.2% of patients underwent RRT, with the patient also presenting with sepsis **(Table 6)**.

Table 6 Associations between stable chronic kidney disease stages and contrast-associated acute kidney injury with mortality and renal replacement therapy

Parameters	Dead within 30 days		RRT within 30 days	
	AKI (n=23)	Non-AKI (n=36)	AKI (n=6)	Non-AKI (n=1)
Stable CKD stages, n (%)	6 (26.1%)	16 (44.4%)	3 (50.0%)	1 (100%)
• G3a	1 (4.3%)	9 (25.0%)	2 (33.3%)	0 (0%)
• G3b	4 (17.4%)	4 (11.1%)	1 (16.7%)	1 (100%)
• G4	1 (4.3%)	3 (8.3%)	0 (0%)	0 (0%)
Current AKI before CM exposure, n (%)	16 (69.6%)	16 (44.4%)	3 (50.0%)	0 (0%)

CKD, chronic kidney disease; AKI, acute kidney injury; CM, contrast media

Discussion

The prevalence of CA-AKI currently demonstrates variability across different patient populations. In this study, the CA-AKI prevalence was 8.9%, encompassing patients with eGFR ranging from 15 to 59 ml/min/1.73 m², who underwent CT with CM exposure in outpatient and inpatient settings, including those with existing AKI. This finding closely aligns with the research by Kidoh in Japan, which reported a similar CA-AKI prevalence of 9.1% among patients with eGFR between 15 and 60 ml/min/1.73 m².[10]

The multivariate analysis revealed no significant associations between CA-AKI and baseline patient characteristics, including age, gender, and regular medications such as diuretics, metformin, and vasopressor agents. Previous CKD stage G3b (eGFR 30-44 ml/min/1.73 m^2) and current AKI emerged as statistically significant factors. These findings align with Davenport's study, which demonstrated that patients with eGFR < 30 ml/min/1.73 m^2 were significantly impacted by CA-AKI, while those with eGFR 30-44 ml/min/1.73 m^2 showed a trend towards significance.[7] In our study, 50% of patients with CKD stage G3b also had current AKI, and approximately 28% of those with CKD stage G4 experienced current AKI. This high comorbidity likely explains why only CKD stage G3b significantly influenced CA-AKI in our analysis.

Previous studies in patients in the ICU suggested that current AKI did not impact CA-AKI. Still, these studies potentially introduced selection bias by including both patients who received and did not receive CM. Patients might have been excluded from CM administration in cases of severely reduced kidney function.[12] Conversely,

our study found that current AKI significantly influenced CA-AKI occurrence. While the precise mechanism of kidney function deterioration after CM exposure remains unclear, pre-existing AKI warrants careful monitoring.

In a subgroup analysis of stable CKD patients, multivariate analysis revealed CKD stage G3b and hematologic malignancy as key factors affecting CA-AKI. Prior research has demonstrated that multiple myeloma may elevate CA-AKI risk, particularly in patients with compromised kidney function, hypercalcemia, or dehydration.[13,14]

A key strength of this study is its comprehensive data collection from a major referral center in Thailand. The research provides a more representative snapshot of clinical practice by including patients with reduced eGFR across both inpatient and outpatient settings, including those with existing AKI. This diverse patient population enhances the study's external validity, capturing a broader range of kidney function scenarios that more closely mirror real-world conditions.

However, the inclusion of patients with AKI prior to CM administration means that observed increases in serum creatinine after CT may be attributable to the underlying kidney injury rather than to the contrast agent itself. The high incidence of CA-AKI reported in this study is likely influenced by the focus on hospitalized patients, most of whom had documented serum creatinine measurements after contrast exposure. In contrast, follow-up data on kidney function are limited in the outpatient setting.

In conclusion, patients with CKD stage G3b and concurrent AKI are at particularly high risk for CA-AKI. Careful management and appropriate protective measures are essential when CM exposure is required in these individuals

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