

Does CT Angiography for Suspected Acute Stroke Cause Acute Kidney Injury?

Şüpheli Akut İnme Hastalarında Yapılan BT Anjiyografi Akut Böbrek Hasarına Neden Olur mu?

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ABSTRACT

Aim: This study aimed to determine the incidence of contrast-associated acute kidney injury (CA-AKI) and identify its associated clinical determinants in patients presenting to the emergency department with suspected acute ischemic stroke who underwent contrast-enhanced cranio-cervical computed tomography angiography (CTA).

Material and Methods: This retrospective, single-center cohort study was conducted in the emergency department of a tertiary stroke center between March and September 2024, including consecutive patients who underwent cranio-cervical CTA for suspected stroke. Patients aged ≥ 18 years with available 7-day follow-up data were included; those requiring dialysis or with incomplete records were excluded. Patients were classified by glomerular filtration rate (GFR) into normal/mild chronic kidney disease (CKD), CKD III, and CKD IV groups. The primary outcome was AKI, defined as a serum creatinine increase of ≥ 0.3 mg/dL within 48 hours or ≥ 1.5 -fold from baseline within 7 days. Independent predictors were assessed using multivariable logistic regression.

Results: A total of 282 patients were analyzed (211 with normal/mild CKD, 61 with CKD III, 10 with CKD IV). AKI developed in 20 patients (7%). Patients who developed AKI differed significantly from those who did not in terms of ICU admission, hemorrhagic stroke diagnosis, use of inotropes, calcium channel blockers, antibiotics, steroids, and anticonvulsants, and the volume of intravenous fluids administered ($p < 0.05$ for all). Age, sex, and eGFR did not differ significantly between groups. On multivariable analysis, ICU admission (OR 4.72), inotrope use (OR 37.82), and IV fluid volume (OR 0.27) were independent predictors of AKI. No significant difference in AKI incidence was observed between patients with normal GFR and those with CKD III/IV ($p = 0.985$).

Conclusion: AKI may develop in patients undergoing cranio-cervical CTA for suspected stroke; however, no significant association between impaired GFR and AKI was demonstrated. Careful use of nephrotoxic medications and balanced intravenous fluid replacement may reduce AKI development. Fear of CA-AKI should not delay or preclude diagnostic CTA or endovascular procedures in acute ischemic stroke patients at risk of large vessel occlusion.

Keywords: Acute kidney injury, contrast agent, computed tomography angiography, ischemic stroke, chronic kidney disease

ÖZ

Amaç: Akut iskemik inme şüphesiyle acil servise başvuran ve kontrastlı kraniyoservikal bilgisayarlı tomografi anjiyografisi (BTA) uygulanan hastalarda kontrast madde ilişkili akut böbrek hasarı (KM-ABH) insidansını belirlemek ve bu duruma eşlik eden klinik belirleyicileri saptamaktır.

Gereç ve Yöntemler: Üçüncü basamak bir inme merkezinin acil servisinde, Mart-Eylül 2024 tarihleri arasında inme şüphesiyle kraniyoservikal BTA çekilen hastalar retrospektif, tek merkezli kohort olarak değerlendirildi. On sekiz yaş üstü, 7 günlük takip verisi bulunan hastalar dahil edildi; diyaliz hastaları ve eksik verisi olanlar dışlandı. Hastalar glomerüler filtrasyon hızına (GFH) göre normal/hafif KBH, KBH III ve KBH IV olarak sınıflandırıldı. Birincil sonlanım, serum kreatinininde 48 saat içinde $\geq 0,3$ mg/dL veya 7 gün içinde bazale göre $\geq 1,5$ kat artış olarak tanımlanan ABH gelişimiydi. Bağımsız risk belirleyicileri çok değişkenli lojistik regresyon ile incelendi.

Bulgular: Toplam 282 hasta analiz edildi (211 normal/hafif KBH, 61 KBH III, 10 KBH IV). ABH 20 hastada (%7) gelişti. ABH gelişen ve gelişmeyen hastalar arasında yoğun bakım ünitesine yatış, hemorajik inme tanısı, inotrop/kalsiyum kanal blokeri/antibiyotik/steroid/antikonvülzan kullanımı ve verilen intravenöz sıvı miktarı açısından anlamlı fark saptandı ($p < 0,05$). Yaş, cinsiyet ve eGFR gruplar arasında anlamlı farklılık göstermedi. Çok değişkenli analizde yoğun bakım yatışı (OR 4,72), inotrop kullanımı (OR 37,82) ve intravenöz sıvı miktarı (OR 0,27) bağımsız belirleyiciler olarak bulundu. Normal GFH ile KBH III/IV grupları arasında ABH gelişimi açısından anlamlı bir fark yoktu ($p = 0,985$).

Sonuç: İnme şüphesiyle başvuran ve kraniyoservikal BTA uygulanan hastalarda ABH gelişebilir; ancak bozulmuş GFH ile ABH gelişimi arasında anlamlı bir ilişki gösterilememiştir. Nefrotoksik ilaçların dikkatli kullanımı ve dengeli intravenöz sıvı replasmanı ABH gelişimini azaltabilir. KM-ABH korkusu, büyük damar tıkanıklığı riski taşıyan akut iskemik inme hastalarında tanısal BTA veya endovasküler girişimlerin geciktirilmesine veya atlanmasına neden olmamalıdır.

Anahtar Kelimeler: Akut böbrek hasarı, kontrast ajan, bilgisayarlı tomografi anjiyografi, iskemik inme, kronik böbrek hastalığı

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Introduction

Acute ischemic stroke is a serious clinical condition in which delays in diagnosis and treatment in emergency departments are directly related to permanent neurological damage and mortality (1,2). Therefore, the primary approach in the emergency department is to rapidly identify the occluded vessel and to initiate appropriate reperfusion therapy, either via intravenous thrombolysis (tPA) or mechanical thrombectomy (MER) (1,2). Contrast-enhanced computed tomography angiography (CTA) has a critical role in identifying patients who could benefit from mechanical thrombectomy (3). It rapidly and reliably demonstrates major vessel occlusions in the intracranial and cervical vessels. (4).

Contrast-associated acute kidney injury (CA-AKI) is a significant clinical concern, especially in time-pressured environments such as the emergency department, where it can influence decisions regarding contrast imaging (5). The incidence of acute kidney injury (AKI) among all patients has been reported to range from 3.3% to 14.5%, and CA-AKI has been reported to be the third most common etiology of hospital-acquired acute kidney injury (6,7). In the past, imaging procedures were delayed while awaiting laboratory results, due to concerns about CA-AKI (6,8). However, current literature and guidelines emphasize that in time-sensitive conditions, such as acute ischemic stroke, rapid diagnosis is of far greater importance than the risk to the kidneys, and that CTA should be performed without waiting for kidney function tests (1,9,10).

The risk of developing CA-AKI is closely related to both patient characteristics and accompanying clinical conditions; chronic kidney disease (CKD), especially an eGFR value below 30 mL/min/1.73 m², is considered the strongest independent risk factor (1,11). Comorbid conditions such as diabetes mellitus, older age, anemia, hypertension, and related cardiovascular diseases can further increase this risk by impairing renal perfusion or reducing renal reserve. Additionally, hypotension, congestive heart failure, low ejection fraction, and the use of nephrotoxic drugs are also among the significant factors contributing to the development of CA-AKI (2,7,12). Consequently, emergency department physicians often face clinical dilemmas when deciding on contrast imaging, such as the delay in diagnosis and treatment caused by waiting for kidney function tests, the challenge of establishing an accurate risk-benefit balance, and the lack of time for adequate prophylactic hydration (6,7,13)

This study aimed to investigate the relationship between intravenous contrast agent and the development of AKI by assessing the incidence of CA-AKI in patients presenting to the emergency department with suspected stroke who

underwent contrast-enhanced cranio-cervical CTA, as well as to identify the clinical determinants linked to this condition.

Material and Methods

Study Design and Setting

This study was designed as a retrospective observational single-center cohort study conducted in the emergency department (ED) of a tertiary care center. The ED functions as a large, comprehensive stroke center, with an annual total of approximately 1000 stroke activations. Before data collection, ethical approval was obtained from the institutional review board and relevant local committees (Marmara University Faculty of Medicine Ethics Committee for Non-Drug and Non-Medical Device Research; Ethical approval: 09.2025.25-0771).

Study population and sample

From March to September 2024, consecutive patients admitted to the ED of a tertiary stroke center who underwent cranio-cervical CTA for suspected acute stroke were included in the study. The standard dosing protocol for iohexol contrast agent at the local institution was 70 mL of 300 mg I/mL. In an effort to minimize confounding variables that could contribute to AKI acquired during the hospital visit, only patients suspected of having a stroke were specifically selected. Electronic medical records (EMR) were collected at admission, during hospitalization, and at discharge, and subsequently analyzed anonymously.

Inclusion and exclusion criteria

All consecutive patients meeting the following inclusion criteria were included in the study sample: (1) age ≥ 18 years, (2) performed cranio-cervical CTA in the ED with a suspicion of acute stroke, and (3) availability of 7-day follow-up data

Exclusion criteria were as follows: (1) need for hemodialysis or peritoneal dialysis treatment due to chronic kidney failure, (2) 7-day serum creatinine values were missing, (3) those who could not complete the examinations or left the hospital for any reason, and (4) incomplete electronic medical records.

Grouping

Considering that the risk of AKI increases in patients with low glomerular filtration rate, patients were classified into CKD categories based on previously established definitions. GFR ≥ 60 was classified as "normal and mild CKD," GFR 59–30 as CKD III, and GFR ≤ 30 as CKD IV (14).

Study process and data collection

Initial patient lists were retrieved from EMR based on activated stroke alerts for the specified date range.

The patient's; medical history (chronic diseases and the medications), demographic data (age and sex), vital signs (blood pressure, heart rate, temperature, and respiratory rate), NISHH and GKS score, laboratory findings (complete blood count, creatinine, electrolytes, albumin, INR, proteinuria in urine and arterial blood gas parameters), reperfusion treatment, hospital admission and following 7-day creatinine levels, medications received within the first 48 hours, and the amount of isotonic fluids administered intravenously along with the final diagnoses data were collected from the hospital's EMR system.

Outcomes

The primary outcome measure was the development of AKI, as previously defined (an increase in serum creatinine of 0.3 mg/dL or more from baseline within 48 hours or an increase in serum creatinine of 1.5 times or more from baseline within 7 days) (15). The serum creatinine levels were monitored within 48 hours and 7 days after the administration of intravenous contrast. Furthermore, a detailed examination of the individual charts of patients who developed AKI was conducted.

The secondary outcome was examined between AKI and the following determinants: type of stroke, comorbidities, patient age, admission to the Intensive Care Unit, amount of intravenous fluids given to the patient, medications, and treatments (including tPA and MER).

Statistical analysis

Statistical analyses were performed using Jamovi version 2.3.26 (The Jamovi Project, Australia). Continuous variables were reported as the mean, standard deviation, and 95% confidence interval (CI), or as the median and interquartile range (IQR; 25th–75th percentiles), depending on the distribution. Categorical variables were presented as counts and percentages. Continuous variables between the two groups were compared using the Mann-Whitney U test, while categorical variables were analyzed using the Chi-square test. A p-value <0.05 was considered statistically significant within a 95% confidence interval.

Independent predictors of AKI were identified using multivariable logistic regression analysis. The variables that were statistically significant in the univariate analysis ($p < 0.05$) were included in the multivariable model. The contributions of each variable included in the model, estimated using the hierarchical block method, were evaluated using the Akaike information criterion (AIC), Bayesian information criterion (BIC), and Nagelkerke R^2 . Regarding multicollinearity, all variables were assessed using correlation analyses, VIF, and tolerance values.

Residual and Cook's distance values were checked. The omnibus test confirmed the models' overall fit ($p < 0.01$). Ultimately, eight variables (ICU admission, IV fluids, inotropes, calcium channel blocker, antibiotics, steroids, anticonvulsants, hemorrhagic stroke) were included in the model. The overall model was significant ($\chi^2 = 50.5$, $df = 8$, $p = <0.001$; McFadden $R^2 = 0.409$)

Results

Within the selected time period, 378 patients with a stroke alert who underwent intravenous contrast cranio-cervical CTA were identified. After screening for exclusion criteria, a total of 282 patients were analyzed.

The patient population was classified as follows: 211 (74.8%) with normal kidney function/mild CKD, 61 (21.6%) with CKD III, and 10 (3.6%) with CKD IV. CKD V (end-stage renal disease) patients were excluded.

Age, gender, comorbidities, medications used by patients, recent diagnoses, and treatment procedures are described in Table 1, broken down by CKD classification. The most common comorbidities were hypertension (62.1%), diabetes (28%), and chronic heart failure (26.2%). 12.4% of the patients were admitted to the intensive care unit, 12.8% underwent MER, and 23% received tPA.

The proportion of patients with comorbid conditions was higher in groups with reduced renal function than in the normal group. However, the presence of any comorbidity did not result in a statistically significant difference in AKI development. Additionally, the percentage of patients using ACEI/ARB, diuretics, statins, and metformin was significantly higher in the CKD Stage III/IV groups. No statistically significant difference was observed among the CKD groups regarding intravenous fluid replacement (Kruskal-Wallis, $p = 0.800$) (Table 1).

AKI developed in 20 patients (7%) included in the study. Compared to patients who did not develop AKI, statistically significant differences were identified between the two groups regarding admission to the intensive care unit, occurrence of hemorrhagic stroke, medications used during treatment (inotropes, calcium channel blockers, antibiotics, steroids, and anticonvulsants), and the amount of intravenous fluid administered. Age, sex, eGFR, and all other examined parameters did not differ significantly between the two groups (Table 2). Multivariate logistic regression analysis revealed that three variables (ICU admission, intravenous fluid administration, and inotrope use) were significant independent predictors of AKI development (Table 3).

Characteristic	Overall n=282	Normal n=211	CKD III n=61	CKD IV n=10
Age (years)	68 (57-79)	65 (55-76)	76 (69-82)	81 (74-86)
Sex (female)	122 (43.3)	86 (40.8)	33 (54.1)	3 (30)
Comorbidities				
Diabetes	79 (28)	49 (23.2)	29 (47.5)	1 (10)
Hypertension	175 (62.1)	120 (56.9)	48 (78.7)	7 (70)
Chronic Heart Failure	74 (26.2)	42 (19.9)	26 (42.6)	6 (60)
Prior Kidney Injury	3 (1.1)	1 (0.5)	1 (1.6)	1 (10)
Cancer	17 (6)	14 (6.6)	2 (3.3)	1 (10)
Creatinine	0.88 (0.74-1.08)	0.8 (0.7-0.92)	1.27 (1.11-1.38)	2.06 (1.94-2.63)
Medication use				
Antiplatelets	103 (36.5)	72 (34.1)	23 (37.7)	8 (80)
Anticoagulants	44 (15.6)	31 (14.6)	11 (18)	2 (20)
ACEi/ARB	112 (39.7)	74 (35.1)	34 (55.7)	4 (40)
Diuretics	28 (9.9)	10 (4.7)	15 (24.6)	3 (30)
Statin	61 (21.6)	36 (17.1)	21 (34.4)	3 (30)
Metformin	49 (17.4)	30 (14.2)	19 (31.1)	0 (0)
Other	173 (61.4)	120 (56.9)	44 (72.1)	9 (90)
Last diagnosis				
Ischemic stroke	214 (75.9)	154 (73)	51 (83.6)	9 (90)
Hemorrhagic stroke	38 (13.5)	33 (15.6)	5 (8.2)	0 (0)
Other	30 (10.6)	24 (11.4)	5 (8.2)	1 (10)
ICU admission	35 (12.4)	27 (12.8)	7 (11.5)	1 (10)
IV fluids (ml)	2000 (1500-2250)	2000 (1500-2250)	1800 (1500-2500)	1900 (1550-2213)
Other procedures				
tPA	65 (23)	47 (22.3)	16 (26.2)	2 (20)
MER	36 (12.8)	25 (11.8)	10 (16.4)	1 (10)

Table 1. Baseline Characteristics of Patients Stratified by CKD Stage

Data are median (IQR 25th-75th percentile) or n (%). CKD; chronic kidney disease; ACEi/ARB, Angiotensin-converting enzyme inhibitors/ Angiotensin receptor blockers; ICU, intensive care unit; tPA, tissue plasminogen activator; MER, mechanical endovascular reperfusion

Characteristic	AKI n=20	No AKI n=262	P value
Age (years)	74 (64-80)	68 (57-79)	0.137
eGFR	79 (65-88)	83 (61-96)	0.365
ICU admission	12 (60)	23 (8.7)	<0.001
Medications Used In Treatment			
IV fluids (ml)	1625 (1500-2000)	2000 (1500-2340)	0.035
Inotropes	4 (20)	2 (0.007)	<0.001
Calcium Channel Blocker	14 (70)	122 (46.5)	0.043
Antibiotics	9 (45)	27 (10.3)	<0.001
Steroids	4 (20)	8 (3)	0.006
Anticonvulsants	12 (60)	48 (18.3)	<0.001
Last diagnosis			
Hemorrhagic stroke	9 (45)	29 (11)	<0.001
Other procedures			
tPA	2 (10)	63 (24)	0.179
MER	3 (15)	33 (12.5)	0.728

Table 2. Comparison of Clinical Variables Between Patients With and Without Acute Kidney Injury

Data are median (IQR 25th-75th percentile) or n (%). AKI, acute kidney injury; GFR, glomerular filtration rate; ICU, intensive care unit; tPA, tissue plasminogen activator; MER, mechanical endovascular reperfusion; IV, intravenous.

Patients who developed AKI were staged according to the KDIGO Acute Kidney Injury criteria, with 12 patients classified as Stage 1, 6 as Stage 2, and 2 as Stage 3. A total of 5 patients required hemodialysis. Mortality was observed in 9 patients, while renal function recovered to baseline levels in 9 of the 11 surviving patients.

While AKI developed in 15 (7.1%) patients with normal GFR and 5 (8.2%) patients with CKD III, no patients with CKD IV

developed AKI. When comparing the group with normal GFR to the group of patients with CKD III/IV, there was no statistically significant difference between the two groups in terms of AKI development ($p=0.985$) (Table 4).

In Table 5, the creatinine levels, as well as the median changes in creatinine levels at 24 and 48 hours, are reported.

Predictor	Estimate	Wald	P	Odds Ratio (95% CI)
Intercept	-2.3258	-2.25	0.024	0.098 (0.013-0.739)
ICU admission	1.5518	2.18	0.029	4.720 (1.170-19.04)
IV fluids (L)	-1.302	-2.50	0.013	0.272 (0.098-0.756)
Inotropes	3.6328	2.71	0.007	37.819 (2.747- 520.720)
Calcium Channel Blocker	1.3110	1.73	0.084	3.7099 (0.840-16.383)
Antibiotics	0.8895	1.29	0.198	2.4339 (0.629-9.421)
Steroids	0.8433	0.87	0.383	2.3241 (0.349-15.473)
Anticonvulsants	0.5658	0.77	0.440	1.7609 (0.418-7.415)
Hemorrhagic stroke	0.2934	0.36	0.717	1.3411 (0.274- 6.561)

Table 3. Multivariable logistic regression analysis of independent predictors associated with AKI

The statistical analysis employed the binomial logistic regression. Estimates represent the log odds of " presence of AKI" vs. " absence of AKI". CI, confident intervals; OR, odds ratio; AKI, acute kidney injury.

Discussion

In our study, AKI developed in 20 (7%) of 282 patients who underwent IV-contrast cranio-cervical CTA. This rate is consistent with the reported incidence range of 3.3%–14.5% in the broader AKI literature and aligns with findings from recent emergency department-based studies (1,7,13).

Many previous studies included all patients who were admitted to the hospital and underwent contrast-enhanced CT imaging for various reasons (trauma, sepsis, etc.) (9). This was one of the main issues with the studies. Because the use of contrast agents and related pathologies (e.g., hypotension and hypovolemia) can complicate the pathophysiology of AKI development. Additionally, it has included various CT imaging scans in the data sets without the use of standard and controlled amounts of intravenous contrast. Our study differs from these studies due to the use of a standard dose of contrast agent in a specific patient population.

For patients presenting to our emergency department with suspected acute stroke, the local stroke protocol is activated, and a cranio-cervical CTA is performed regardless of the patient's serum creatinine levels. Unlike other studies, this setting allowed us to work with a more homogeneous patient population, minimizing the risk of bias, rather than a cohort of patients with either normal or impaired GFR. Indeed, in our study, no significant difference was found in the incidence of AKI between the groups of patients with normal and low GFR (GFR<60 mL/min/1.73 m²) (p=0.985). This finding is consistent with a recent study by Imran Faruq et al. and a recent meta-analysis (1,9). Taken together, these findings are consistent with the growing body of evidence suggesting that, in patients with mild-to-moderate renal impairment (GFR 30–60 mL/min/1.73 m²), the independent nephrotoxic contribution of iodinated contrast agents may be more limited than historically assumed, and that a proportion of CA-AKI cases may in part reflect concurrent non-contrast-associated insults rather than direct tubular toxicity (9,16).

Characteristic	Overall n=282	Normal n=211	CKD III n=61	CKD IV n=10	p
AKI, n (%)					
No AKI	262 (93)	196 (92.9)	56 (91.8)	10 (100)	0.985*
AKI	20 (7)	15 (7.1)	5 (8.2)	0 (0)	

Table 4. AKI, defined as an increase in serum creatinine of 0.3 mg/dL or more from baseline within 48 hours or an increase in serum creatinine of 1.5 times or more from baseline within 7 days

Data are n (%). AKI, acute kidney injury; CKD, chronic kidney disease. *Normal versus CKD III/IV

Creatinine	Normal n=211	CKD III n=61	CKD IV n=10
Initial	0.8 (0.7-0.92)	1.27 (1.11-1.38)	2.06 (1.94-2.63)
48 hours	0.78 (0.64-0.9)	1.12 (0.93-1.33)	2.06 (1.72- 2.36)
7 days	0.81 (0.7-0.93)	1.21 (0.94-1.43)	2.18 (1.83-2.4)

Table 5. Creatinine Values Presented in mg/dL by CKD Classification

Data are median (IQR 25th-75th percentile). CKD, chronic kidney disease

In our study, we analyzed almost all the reported risk factors for AKI (comorbidities, impaired GFR, use of nephrotoxic drugs, ICU admission, etc.) (17) between the groups that developed AKI and those that did not. Additionally, we included data on intravenous fluid administration in our analysis to better characterize

patients' hydration status. On univariate analysis, ICU admission (60% vs. 8.7%, p<0.001), hemorrhagic stroke diagnosis (45% vs. 11%, p<0.001), inotrope use (20% vs. 0.7%, p<0.001), calcium channel blocker administration (70% vs. 46.5%, p=0.043), antibiotic use (45% vs. 10.3%, p<0.001), steroid use (20% vs. 3%, p=0.006), anticonvulsant use (60% vs. 18.3%, p<0.001), and the total volume of IV fluids administered within the first 48 hours were all significantly associated with AKI development. On multivariable logistic regression, three variables emerged as independent predictors: ICU admission (OR 4.72, 95% CI 1.17–19.04), inotrope use (OR 37.82, 95% CI 2.75–520.7), and IV fluid volume (OR 0.27, 95% CI 0.10–0.76). Given the retrospective and observational nature of the study, these

variables should be regarded as independent statistical associations rather than proven causal determinants of AKI. Age, sex, baseline eGFR, and comorbidities did not differ significantly between groups (1,18), a finding that may partly reflect the inherently older, comorbidity-laden nature of this stroke population in which such differences are less discriminating. The high odds ratio observed for inotrope use likely reflects the hemodynamic instability of patients requiring vasopressor support, which can independently compromise renal perfusion and precipitate AKI regardless of contrast exposure (7,19). The inverse association between IV fluid volume and AKI risk (OR 0.27) may suggest that more liberal hydration is associated with a lower likelihood of AKI in this setting. Given the retrospective, observational design of our study, however, this relationship should be interpreted as an association rather than evidence of a direct causal protective effect, and it may in part reflect confounding by indication, since hemodynamically unstable or critically ill patients are themselves at higher risk of AKI. Such an association is nonetheless mechanistically plausible, as volume expansion may reduce intratubular contrast concentration, attenuate renin–angiotensin-mediated renal vasoconstriction, and enhance medullary oxygenation, potentially lowering the risk of contrast-associated tubular injury (17). ICU admission was also a significant independent predictor of AKI in our multivariable model (OR 4.72), which is consistent with the well-established association between critical illness and AKI (7,20). In the neurocritical care setting, AKI complicates approximately 20% of acute brain injury cases and is associated with worsening outcomes, including prolonged ICU stay and increased mortality (20). In our cohort, the higher AKI rate among ICU patients most likely reflects the convergence of hemodynamic instability, nephrotoxic medication exposure, and systemic stress rather than contrast administration itself.

In the current study, no development of AKI was observed among patients with CKD IV (GFR < 30 mL/min/1.73 m²). This subgroup, however, comprised only ten patients with no observed AKI events, which provides very limited statistical power and precludes any reliable estimate of AKI risk in this population. Accordingly, this finding should not be interpreted as evidence that advanced CKD is not associated with an increased risk of AKI; rather, it most likely reflects the small sample size together with the increased clinical attention and proactive kidney-protective management afforded to these patients—including careful fluid balance, avoidance of nephrotoxins, and closer laboratory monitoring. Larger prospective studies are needed to characterize the risk of AKI in CKD IV patients undergoing emergency CTA (12).

A noteworthy finding in our cohort was the strong association between hemorrhagic stroke and AKI development (45% vs. 11%, $p < 0.001$) on univariate analysis, although this association did not retain statistical independence in the multivariable model. In intracerebral hemorrhage, AKI is known to arise through multiple pathways beyond contrast exposure, including hemodynamic instability, neurogenic inflammation, and iatrogenic nephrotoxin use (21). Consistent with this, hemorrhagic stroke patients in our cohort were more frequently admitted to the ICU and exposed to inotropes, anticonvulsants, and steroids, suggesting that AKI in this group reflects the overall burden of critical neurological illness rather than contrast nephrotoxicity (20,21).

Unlike the administration of intravenous contrast agent only for diagnostic cranio-cervical CTA, patients with acute ischemic stroke due to large vessel occlusion, treated with MER, may get a second and significant amount of extra intra-arterial contrast agent during the MER procedure. That theoretically increases the risk of CA-AKI. In their research, Ralph Weber et al. reported that the incidence of AKI did not increase in patients who underwent the MER procedure after CTA (2). In our study, we similarly found that the MER procedure did not create a significant difference between the groups.

Limitations

This study has some limitations:

First, because of the retrospective design of our study, we were unable to obtain 7-day creatinine data for some patients. In the future, conducting prospective, multicenter studies with larger cohorts will improve the generalizability of the results.

Second, the number of stage IV CKD patients in our study was limited. Studies with larger patient groups could allow for more comprehensive analyses of CA-AKI development in patients with stage IV CKD.

Third, our multivariable logistic regression model included eight covariates for only 20 AKI events, which corresponds to a low number of events per variable. Because of this, the model may be susceptible to overfitting, and some of the effect estimates should be interpreted with caution, as reflected in the wide confidence intervals observed for certain predictors such as inotrope use. We therefore regard these regression findings as exploratory and hypothesis-generating rather than definitive, and they warrant confirmation in larger cohorts.

Fourth, our patient cohort consists of a specific group of patients who presented with suspected acute ischemic stroke. Due to the nature of the disease affecting older individuals, the relationship between the development of AKI and age and comorbidities could not be clearly

established in our study. The results obtained in cohorts including different diseases and age groups may vary.

Fifth, patients' intravenous fluid admissions were calculated by reviewing patient orders. However, because of the retrospective study design, the amount of fluid administered to some patients via nasogastric or oral routes could not be determined with certainty. This condition was assumed to affect all groups randomly.

Finally, the volume of contrast agent administered for CTA may vary across institutions, and different imaging protocols may be employed. This variability could potentially influence the development of AKI, particularly in patients with CKD Stage III and IV.

Conclusion

In conclusion, AKI may develop in patients who present to the ED with a suspicion of stroke and are undergoing cranio-cervical CTA for this reason, and clinicians should take this into consideration. However, our study did not demonstrate a significant association between impaired GFR (GFR < 60 mL/min/1.73 m²) and the development of AKI. The secondary results obtained have shown that careful use of nephrotoxic drugs in this patient group, along with balanced and adequate IV fluid replacement, may be associated with a lower risk of AKI development. Particularly in patients admitted to the ICU and undergoing inotropic therapy, greater caution should be exercised.

Our results indicate that the fear of CA-AKI should not lead to the delay or omission of diagnostic CTA or endovascular procedures in patients with acute ischemic stroke who are at risk of large vessel occlusion.

Conflict of Interest: On behalf of all authors, the corresponding author states that there is no conflict of interest.

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not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature.

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