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Incidence of contrast-induced acute kidney injury in patients with stroke who present to the Emergency Department in the central region of Saudi Arabia

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ABSTRACT

Background: Contrast Media (CM) imaging is a critical component of acute stroke management. Nevertheless, the risk of Post-Contrast Acute Kidney Injury (PC-AKI) in patients who undergo computed tomography angiography (CTA) or computed tomography perfusion (CTP) is a topic of controversy.

Aim: This study aimed to estimate the incidence and assess the predictors of PC-AKI after neurological imaging in adult patients with acute stroke.

Methods: This retrospective cohort study was conducted at the National Guard - Health Affairs, Riyadh, Saudi Arabia. Medical records of adult patients diagnosed with acute stroke who underwent CTA/CTP were reviewed. PC-AKI was defined as an increase in serum creatinine ≥0.3 mg/dl or ≥1.5-1.9 times the baseline level 48 hours after CM administration.

Results: The study included 741 consecutive patients. PC-AKI incidence was 1.8% (95% CI: 0.98%, 3.1%). Univariate logistic regression analysis showed that increased risk of PC-AKI was significantly associated with chronic kidney disease ($p < 0.001$), elevated baseline serum creatinine ($p = 0.006$), and decreased GFR ($p < 0.001$). Multivariate logistic regression showed that only decreased GFR <30 ml/minute/1.73 m^2 was an independent risk factor for developing PC-AKI.

Conclusion: Patients with acute stroke have a low incidence of PC-AKI. In order to facilitate the subsequent identification of patients who may develop acute kidney injury, baseline creatinine levels should be obtained without postponing the implementation of the appropriate imaging modalities. Close monitoring and protection against PC-AKI are necessary for patients with a baseline estimated glomerular filtration rate of less than 30 ml/minute/1.73 m^2, as the known benefits and potential hazards of contrast imaging must be considered.

Keywords: Acute kidney injury, contrast media, diagnostic imaging, intracranial hemorrhage, ischemic stroke.

Introduction

Acute stroke is a life-threatening medical condition that requires immediate intervention to mitigate the risk of long-term complications and mortality [1,2]. The diagnosis and treatment of patients with acute stroke are significantly influenced by early vascular imaging. A comprehensive computed tomography (CT) protocol is employed to evaluate acute stroke radiologically. This protocol comprises a non-contrast CT scan of the brain and CT angiography (CTA) of the head and neck vessels

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to determine which vessel is occluded. Furthermore, CT perfusion (CTP) is an additional imaging modality that can identify patients who would benefit from reperfusion treatment beyond the conventional time frame [3-6]. The use of CTP also aids in the assessment of the progression of the infarction and the identification of patients who may require decompressive surgery [7].

Nevertheless, the utilization of iodinated contrast media (CM) in CTA and CTP may result in renal toxicity, which can have a negative impact on renal function. According to the Contrast Media Safety Committee of the European Society of Urogenital Radiology, acute kidney injury (an increase in serum creatinine ≥0.3 mg/dl, or ≥1.5-1.9 times baseline) in the 48-72 hours following CM administration should be referred to as Post-Contrast Acute Kidney Injury (PC-AKI) in the absence of a control population [4]. Concurrently, the term "Contrast-Induced Acute Kidney Injury" is employed when a control population is present [4]. Research has demonstrated that PC-AKI is associated with extended hospital stays and an elevated risk of mortality [8,9].

Advanced age and the prevalence of vascular risk factors in the majority of stroke patients may increase the risk of developing PC-AKI. As a result, there is ongoing discussion regarding the use of iodinated CM in acute stroke patients and the actual risk of developing PC-AKI in these patients. In numerous centers, vascular imaging is typically delayed until the serum creatinine baseline is determined. Delaying the management of stroke patients may result in the loss of valuable time and the deterioration of neurological outcomes [1,10]. The justification for not conducting CTA or CTP in stroke patients, even in the presence of chronic kidney disease (CKD), is also a topic of controversy, as it is predicated on the reduction of the risk of PC-AKI [1].

In the interim, the prevalence of undiagnosed renal disease among stroke patients and the incidence of acute kidney injury (AKI) following contrast imaging remain to be determined [2,4]. Although numerous studies have been conducted, the overall risk following CTA/CTP remains ambiguous. Additionally, the current practice of waiting for baseline serum creatinine levels before imaging has not been altered at the majority of institutions [11,12].

The objective of the present investigation was to determine the prevalence and evaluate the predictors of PC-AKI following neurological imaging in patients with acute stroke who were treated at the National Guard-Health Affairs (NGHA) in Riyadh, Saudi Arabia.

Methods

Study design and settings

This retrospective cohort study was conducted by collecting the prospectively registered data of consecutive adult patients admitted to the NGHA in Riyadh, Saudi Arabia, and diagnosed with acute stroke during the period from 2018 to 2020.

Study population and eligibility criteria

This study included adult patients (age ≥ 18 years) with a diagnosis of stroke who underwent CTA and/or CTP and whose medical records recorded serum creatinine levels prior to contrast imaging.

We excluded patients with secondary referrals or in-hospital stroke or CKD requiring dialysis. Patients who died or were transferred/discharged within 48 hours of CTA/CTP were excluded.

Data collection

Hospital records of patients admitted to NGHA in Riyadh, Saudi Arabia, from 2018 to 2020 were reviewed. Data extracted included patient age, sex, medical history, admission serum creatinine, vascular imaging performed, hydration protocol, serum creatinine 48 hours after CM imaging, estimated glomerular filtration rate (eGFR), and patient outcome.

Study outcomes

The primary outcome was the incidence of PC-AKI. PC-AKI was diagnosed if there was an increase in serum creatinine ≥0.3 mg/dl or ≥1.5-1.9 times the baseline level at 48 hours after CM administration. Secondary outcomes included the identification of independent risk factors for PC-AKI.

Statistical analysis

Analyses were conducted using the R Statistical language version 4.4.0 [13], using the packages gtsummary version 1.7.2 [14]. Categorical variables were summarized as frequencies, and the associations with PC-AKI were tested using Pearson's chi-square test for independence of observation, Fisher's exact test, or the chi-squared test for Trend in Proportions as indicated. The Shapiro-Wilk test and the Q-Q plots were used to assess the distribution of numerical variables (i.e., age, serum creatinine). Variables that followed normal distribution were represented using the mean and standard deviation and compared using the unpaired T-test. The variables that did not follow the normal distribution were summarized using the median and interquartile range (25th - 75th percentiles) and compared between groups using the Mann-Whitney test (Wilcoxon rank sum test). The significance level for interpreting statistical significance was set at p-value < 0.05.

Results

During the study period, 1,800 patients were admitted into the NGHA in Riyadh, Saudi Arabia, of which 741 met the inclusion criteria. The patients' ages ranged between 23 and 109 years. Male patients accounted for 63%, and most were Saudi (91%). The type of stroke was ischemic in 74%, while intracranial hemorrhage was encountered in 3% only. The incidence of PC-AKI was 1.8% (95% CI: 0.98%, 3.1%) in all patients and 1.8% (95% CI: 0.9%, 3.5%) in those with a final diagnosis of acute ischemic stroke. However, the incidence was 8.6%

Table 1. Sociodemographic and outcome data (N = 741).

	All participants
Characteristic	***N* = 741**
Age (year)	
Median [IQR]	67.0 [58.0-78.0]
Range	23.0-109.0
Gender, *n* (%)	
Female	272 (37%)
Male	469 (63%)
Nationality, *n* (%)	
Saudi	676 (91%)
Non-Saudi	65 (9%)
Type of stroke, *n* (%)	
Ischemic	546 (74%)
Hemorrhagic	22 (3%)
TIA	173 (23%)
Diagnosis at discharge, *n* (%)	
Ischemic stroke	546 (74%)
Intracranial hemorrhage	22 (3%)
Transient ischemic attack	173 (23%)
Post-contrast acute kidney injury, n/N (%) (95% CI)	
All patients	13/741 (1.8%) (95% CI: 0.98%, 3.1%)
CKD patients	6/70 (8.6%) (95% CI: 3.5%, 18%)
Patients with ischemic stroke	10/ 546 (1.8%) (95% CI: 0.9%, 3.5%)

IQR: interquartile range; n: number within a category; N: total sample size.

in those with a history of CKD (95% CI: 3.5%, 18%; Table 1).

When comparing patients who developed PC-AKI to those who did not, PC-AKI was significantly associated with a higher percentage of patients with a CKD disease history (46% *vs.* 9%, $p < 0.001$; Table 2).

The comparison of the investigations at the time of admission showed that patients who developed PC-AKI had significantly higher baseline serum creatinine values (Median [IQR]: 1.3 [0.8-2.5] versus 0.8 [0.7-1.0] mg/dl, $p = 0.011$). Baseline GFR tended to show low values in patients who developed later PC-AKI. In addition, a significantly higher percentage of patients who developed PC-AKI underwent altering location at admission (54% *vs.* 19%, $p = 0.005$; Table 3).

Comparison of the details of intravenous fluid administration, radiography, treatment, and disposition was mostly comparable between the patients with and without PC-AKI ($p > 0.05$). The only significant comparison was the disposition from the Emergency Department, where a significantly higher percentage of the PC-AKI patients were admitted to the intensive care unit (23% *vs.* 4%, $p = 0.018$; Table 4).

Univariate logistic regression analysis was performed to identify risk factors for PC-AKI. Variables significantly associated with increased risk of PC-AKI included CKD (OR [95% CI]: 8.89 [2.79, 27.60], $p < 0.001$) and elevated baseline serum creatinine (OR [95% CI]: 1.68 [1.17, 2.56], $p = 0.006$). Decreased GFR was progressively and significantly associated with an increased risk of PC-AKI ($p < 0.001$; Table 5).

Multivariate logistic regression was performed to identify risk factors for PC-AKI using variables with $p < 0.1$ from univariate regression. Decreased GFR was an independent risk factor for the development of PC-AKI, and the risk increased progressively as the level of GFR decreased ($p < 0.05$; Table 6).

Discussion

The management of acute stroke is significantly influenced by contrast media imaging. Nevertheless, there is a disagreement regarding the risk of PC-AKI in patients who have undergone CTA orCTP [1]. The risk of developing renal dysfunction is typically elevated in patients with acute stroke due to the presence of numerous cardiovascular risk factors [1]. The objective of the current investigation was to determine the prevalence of PC-AKI and evaluate the predictors of this condition following neurological imaging in patients with acute stroke who were treated at NGHA, Riyadh, Saudi Arabia.

We discovered that the overall incidence of PC-AKI was 1.8% (95% CI: 0.98%, 3.1%) and 1.8% (95% CI: 0.9%, 3.5%) in individuals with a final diagnosis of acute ischemic stroke. Meanwhile, patients with a history of CKD exhibited a significantly higher incidence (8.6%, 95% CI: 3.5%, 18%). In accordance with our findings, two prior studies on patients with acute stroke reported an incidence of 1.3%-2% [2,15,16]. Nevertheless, other studies have documented higher rates of PC-AKI in acute stroke patients, with a range of 3%-5% [11,15,17-22]. The cumulative rate of AKI in stroke patients undergoing CTA/CTP was 3% (95% CI: 2%-4%), according to a meta-analysis of 14 studies [1]. However, a study on stroke patients who underwent thrombectomy following CTA/CTP reported a higher rate of 5.8% [23]. Furthermore, a meta-analysis that included 12 studies documented an 11.6% increase in the rate of stroke after all types (95% CI: 10.6%, 12.7%) Twenty-four.

The reported incidence rates of PC-AKI among the studies may be influenced by a variety of factors. Patients with CKD necessitating dialysis were excluded from certain investigations, such as the current one. Conversely, patients with end-stage renal disease who were undergoing dialysis may have been included in other studies. A retrospective case-control study conducted on a large scale demonstrated that the rate of nephrotoxicity following computerized tomography was significantly different between contrast-enhanced and non-enhanced imaging only in patients with baseline serum creatinine levels exceeding 1.5 mg/dl [24].

Furthermore, variations in the reported rates may arise as a consequence of the definition employed to diagnose PC-AKI. Serum creatinine levels were assessed 48 hours following the administration of CM in the current study. Nevertheless, serum creatinine levels may persist for an additional 5 days following exposure to the CM [25]. Consequently, the incidence of PC-AKI may be

Table 2. Characteristics of patients with and without post-contrast acute kidney injury (N = 741).

Characteristic	Overall, *N* = 741	No PC-AKI, *N* = 728	PC-AKI, *N* = 13	*p*-value
Age (year)				0.504[1]
Median [IQR]	67.0 [58.0-78.0]	67.0 [58.0-78.0]	72.0 [67.0-78.0]	
Range	23.0-109.0	23.0-109.0	46.0-85.0	
Gender, *n* (%)				0.777[2]
Female	272 (37%)	268 (37%)	4 (31%)	
Male	469 (63%)	460 (63%)	9 (69%)	
Nationality, *n* (%)				0.318[2]
Saudi	676 (91%)	665 (91%)	11 (85%)	
Non-Saudi	65 (9%)	63 (9%)	2 (15%)	
Smoking, *n* (%)				0.380[2]
No	660 (89%)	647 (89%)	13 (100%)	
Yes	81 (11%)	81 (11%)	0 (0%)	
Body mass index (kg/m^2)				0.986[1]
Median [IQR]	26.9 [23.4-31.3]	27.0 [23.4-31.2]	24.7 [22.5-32.5]	
Range	13.8-130.6	13.8-130.6	17.8-42.5	
Medical conditions, *n* (%)	660 (89%)	648 (89%)	12 (92%)	>0.999[2]
Hypertension, *n* (%)	548 (74%)	537 (74%)	11 (85%)	0.531[2]
Diabetes mellitus, *n* (%)	468 (63%)	460 (63%)	8 (62%)	>0.999[2]
Ischemic heart disease, *n* (%)	105 (14%)	101 (14%)	4 (31%)	0.098[2]
Congestive heart failure, *n* (%)	40 (5%)	38 (5%)	2 (15%)	0.152[2]
Chronic kidney disease, *n* (%)	70 (9%)	64 (9%)	6 (46%)	**<0.001*[2]**
Hyperlipidemia, *n* (%)	235 (32%)	231 (32%)	4 (31%)	>0.999[2]
Atrial fibrillation, *n* (%)	59 (8%)	58 (8%)	1 (8%)	>0.999[2]
Prior stroke, *n* (%)	197 (27%)	191 (26%)	6 (46%)	0.119[2]
Intracerebral hemorrhage, *n* (%)	12 (2%)	11 (2%)	1 (8%)	0.193[2]
Prior transient ischemic attack, *n* (%)	47 (6%)	47 (6%)	47 (6%)	>0.999[2]
Previous use of contrast media, *n* (%)	232 (31%)	230 (32%)	2 (15%)	0.365[2]
NSAIDs use, *n* (%)	329 (44%)	325 (45%)	4 (31%)	0.318[3]

PC-AKI: post-contrast acute kidney injury; IQR: interquartile range; NSAIDs: non-steroidal anti-inflammatory drugs; [1]Wilcoxon rank sum test; [2]Fisher's exact test; [3]Pearson's chi-squared test; *significant at $p < 0.05$.

Table 3. *Investigations of patients at the time of admission (N = 741).*

Characteristic	Overall, *N* = 741	No PC-AKI, *N* = 728	PC-AKI, *N* = 13	*p*-value
NIHSS at admission				0.810[1]
Median [IQR]	5.0 [2.0-10.0]	5.0 [2.0-10.0]	3.0 [2.0-11.0]	
Range	1.0-25.0	1.0-25.0	1.0-19.0	
Unknown	602	592	10	
Time since symptom onset (h)				0.655[1]
Median [IQR]	12.0 [3.0-48.0]	12.0 [3.0-48.0]	6.0 [1.0-24.0]	
Range	0.2-1,440.0	0.2-1,440.0	1.0-120.0	
Baseline serum creatinine (mg/dl)				**0.011*[1]**
Median [IQR]	0.8 [0.7-1.0]	0.8 [0.7-1.0]	1.3 [0.8-2.5]	
Range	0.4-11.2	0.4-11.2	0.6-4.8	
Glomerular filtration rate (ml/minute/1.73 m^2), *n* (%)				0.056[3]
≥90	337 (45%)	334 (46%)	3 (23%)	
89-60	306 (41%)	304 (42%)	2 (15%)	
59-30	76 (10%)	73 (10%)	3 (23%)	
29-15	14 (2%)	11 (2%)	3 (23%)	
<15	8 (1%)	6 (1%)	2 (15%)	
Hemoglobin (g/dl)				0.615[1]
Median [IQR]	13.9 [12.5-15.3]	13.9 [12.5-15.3]	14.7 [11.8-16.1]	
Range	6.8-19.9	6.8-19.9	7.9-17.8	

PC-AKI: post-contrast acute kidney injury; NIHSS: National Institutes of Health Stroke Scale; IQR: interquartile range; [1]Wilcoxon rank sum test; [2]Fisher's exact test; [3]Chi-squared Test for Trend in Proportions; *significant at $p < 0.05$.

Table 4. Administration of intravenous fluids and details of radiography, treatment, and disposition (N = 741).

Characteristic	Overall, *N* = 741	No PC-AKI, *N* = 728	PC-AKI, *N* = 13	*p*-value
Intake of IV fluid, *n* (%)	451 (61%)	443 (61%)	8 (62%)	0.960[1]
IV fluid time, *n* (%) (Total = 451)				0.614[2]
At admission	382 (85%)	374 (84%)	8 (100%)	
Before contrast	69 (15%)	69 (16%)	0 (0%)	
Type of fluid, *n* (%) (Total = 451)				>0.999[2]
Normal Saline	448 (99%)	440 (99%)	8 (100%)	
Ringer lactate	2 (0%)	2 (0%)	0 (0%)	
5% Dextrose	1 (0%)	1 (0%)	0 (0%)	
Radiological findings, *n* (%)	626 (84%)	614 (84%)	12 (92%)	0.704[2]
CT angiography, *n* (%)	741 (100%)	728 (100%)	13 (100%)	>0.999[2]
CT perfusion, *n* (%)	176 (24%)	172 (24%)	4 (31%)	0.520[2]
rtPA, *n* (%)	39 (5%)	39 (5%)	0 (0%)	>0.999[2]
Type of stroke, *n* (%)				0.319[2]
Ischemic	546 (74%)	536 (74%)	10 (77%)	
Hemorrhagic	22 (3%)	21 (3%)	1 (8%)	
TIA	173 (23%)	171 (23%)	2 (15%)	
Discharge diagnosis, *n* (%)				0.567[2]
Ischemic stroke	546 (74%)	536 (74%)	10 (77%)	
Intracranial hemorrhage	22 (3%)	21 (3%)	1 (8%)	
Transient ischemic attack	173 (23%)	171 (23%)	2 (15%)	
Disposition from ED, *n* (%)				**0.018*[2]**
Floor	707 (95%)	697 (96%)	10 (77%)	
ICU	34 (5%)	31 (4%)	3 (23%)	
Disposition from hospital, *n* (%)				>0.999[2]
Deceased	11 (1%)	11 (2%)	0 (0%)	
Home	730 (99%)	717 (98%)	13 (100%)	

PC-AKI: post-contrast acute kidney injury; DSA: digital subtraction angiography; [1]Pearson's chi-squared test; [2]Fisher's exact test; *significant at $p < 0.05$.

underestimated if the diagnosis is restricted to the initial 48 or 72 hours.

The form of stroke is also a significant factor, as previous research has demonstrated that the rate of PC-AKI is lower in patients with acute ischemic stroke than in those with intracranial hemorrhage. According to a meta-analysis, the prevalence of AKI was 12.9% (95% CI: 10.3, 15.5) in patients with acute ischemic stroke and 19% (95% CI: 8.3, 29.7) in patients with intracranial hemorrhage [26]. Furthermore, a study conducted by Frank et al. [22] showed that the rates of PC-AKI were 3% and 10.9% in patients with acute ischemic stroke and intracranial hemorrhage, respectively. This implies that intracranial hemorrhage is linked to an increased risk of developing PC-AKI, which can be attributed to the presence of chronic hypertension, hypertensive renal disease, and the administration of antihypertensive medications in those patients [27,28]. The current study was unable to calculate a discrete incidence rate of PC-AKI for patients with intracranial hemorrhage due to the low prevalence of this condition.

In the univariate logistic regression analysis conducted in the present study, CKD ($p < 0.001$), elevated baseline serum creatinine ($p = 0.006$), and decreased GFR ($p < 0.001$) were significantly associated with an increased risk of PC-AKI. Nevertheless, multivariate logistic regression demonstrated that the development of PC-AKI was solely influenced by a decreased GFR of less than 30 ml/minute/1.73 m^2. The independent risk factors for PC-AKI in stroke patients have been the subject of controversy in previous studies. Some studies, in agreement with our findings, reported that a GFR below 30 ml/minute/1.73 m^2 was significantly associated with a higher risk of PC-AKI [12,29].

In certain studies, the rate of PC-AKI did not exhibit a significant difference between patients with and without CKD (odds ratio = 0.63; 95% CI: 0.34, 1.12) [1]. Patients who underwent contrast-enhanced and non-contrast CT scans had a comparable risk of developing AKI, even after accounting for baseline renal function, according to a previous single-center study [30]. In the interim, a separate study found that contrast-enhanced CT was significantly associated with a higher risk of PC-AKI in patients with baseline serum creatinine levels above 1.5 mg/dl compared to patients undergoing unenhanced CT [24].

In a study of stroke patients who underwent thrombectomy after CTA/CTP, CKD, diabetes mellitus, and tandem occlusion were significantly associated

Table 5. *Univariate logistic regression for risk factors of post-contrast acute kidney injury.*

Characteristic	OR	SE	95% CI	*p*-value
Age (year)	1.01	0.02	0.97, 1.06	0.515
Male gender	1.31	0.61	0.42, 4.88	0.655
Body mass index (kg/m^2)	1.00	0.03	0.92, 1.05	0.984
Non-Saudi Nationality	1.92	0.78	0.29, 7.35	0.403
Smoking	0.00	1,195.00		0.990
Medical conditions	1.48	1.05	0.29, 27.20	0.708
Diabetes mellitus	0.93	0.58	0.31, 3.11	0.903
Hypertension	1.96	0.77	0.52, 12.70	0.386
Ischemic heart disease	2.76	0.61	0.74, 8.64	**0.096**
Congestive heart failure	3.30	0.79	0.50, 12.90	0.129
Chronic kidney disease	8.89	0.57	2.79, 27.60	**<0.001***
Hyperlipidemia	0.96	0.61	0.26, 2.97	0.941
Prior stroke	2.41	0.56	0.77, 7.34	0.118
Prior transient ischemic attack	0.00	1,569.00		0.992
Intracerebral hemorrhage	5.43	1.08	0.29, 31.50	0.119
Previous use of contrast media	0.39	0.77	0.06, 1.48	0.228
NSAIDs use	0.55	0.61	0.15, 1.71	0.325
Time since symptom onset (hour)	1.00	0.01	0.98, 1.00	0.679
Baseline serum creatinine (mg/dl)	1.68	0.19	1.17, 2.56	**0.006***
Glomerular filtration rate (ml/minute/1.73 m^2)				
≥90	Ref.	—	Ref.	
89-60	0.73	0.92	0.10, 4.45	0.734
59-30	4.58	0.83	0.83, 25.20	**0.066**
29-15	30.40	0.87	5.14, 181.00	**<0.001***
<15	37.10	1.00	4.32, 269.00	**<0.001***
Intravenous dye	769,239.00	1,319.00	0.00, NA	0.992
Recombinant tissue plasminogen activator	0.00	1,044.00		0.989

OR: odds ratio; SE: standard error; CI: confidence interval; NSAIDs: non-steroidal anti-inflammatory drugs; *significant at $p < 0.05$.

Table 6. *Multivariate logistic regression for risk factors of post-contrast acute kidney injury.*

Characteristic	OR	SE	95% CI	*p*-value
Ischemic heart disease				
No	Ref.	-	Ref.	
Yes	3.22	0.68	0.77, 11.80	0.084
Chronic kidney disease				
No	Ref.	-	Ref.	
Yes	0.72	1.06	0.08, 5.77	0.756
Baseline serum creatinine (mg/dl)	0.70	0.41	0.24, 1.35	0.390
Glomerular filtration rate (ml/minute/1.73 m^2)				
≥90	Ref.	-	Ref.	
89-60	0.77	0.92	0.10, 4.70	0.774
59-30	6.06	0.94	0.81, 39.00	0.056
29-15	72.40	1.36	4.85, 1,162.00	**0.002***
<15	208.00	1.97	4.28, 12,961.00	**0.007***

OR: odds ratio; SE: standard error; CI: confidence interval; *significant at $p < 0.05$.

with AKI in univariate analysis, but not in multivariable logistic regression analysis [23]. In stroke patients who underwent CTA, data from a tertiary center in Germany demonstrated that PC-AKI was substantially associated with CKD, elevated creatinine levels, reduced GFR, high NIHSS, and an altered level of consciousness at admission [22].

The controversial relationship between baseline creatinine levels and the relatively low incidence rates of PC-AKI, as observed in our study and other previous studies, indicates that delaying the indicated contrast-enhanced imaging in stroke patients due to concerns about PC-AKI may be superfluous. The outcomes of patients with acute stroke may be adversely affected by the delay in diagnostic work-up and intervention. The probability of a favorable outcome could be reduced by 10% as a result of a 45-minute delay in reperfusion, according to an estimate [31].

With an appropriate sample size, the current study offered real-world data and insights into the incidence and risk factors of PC-AKI in acute stroke from a tertiary-care center. Nevertheless, the investigation disclosed certain constraints. Physicians may have required CTA/CTP less frequently in patients with elevated baseline creatinine levels than in those with normal creatinine levels. Furthermore, serum creatinine levels were accessible for the diagnosis of PC-AKI at 48 hours, although some patients may encounter elevated levels as late as the fifth day following exposure to CM. As only four patients were identified in the cohort, we were unable to evaluate the additional impact of CTP following CTA on the incidence of PC-AKI. Additionally, the precision of the estimates and confidence intervals in multivariate logistic regression may be influenced by the low aggregate rate of PC-AKI. Finally, the generalizability of our findings is restricted by the fact that it is a retrospective single-center study.

Conclusion

In summary, the overall incidence of PC-AKI was 1.8% (95% CI: 0.98%, 3.1%), but it was significantly higher in patients with CKD (8.6%, 95% CI: 3.5%, 18%). In order to facilitate the subsequent identification of patients who develop AKI, baseline creatinine levels should be obtained without postponing the appropriate imaging modalities. Close monitoring and protection against PC-AKI are necessary for patients with a baseline eGFR of less than 30 ml/minute/1.73 m^2. In these patients, the patients' treating physicians may contemplate the utilization of magnetic resonance imaging and non-contrast CT. In order to evaluate the risk of PC-AKI between stroke patients who undergo non-contrast CT and various forms of contrast-enhanced imaging, stratified by baseline renal function, future randomized controlled trials are required.

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List of Abbreviations

CKD	Chronic Kidney Disease
CM	Contrast Media
CTA	Computed Tomography Angiography
CTP	Computed Tomography Perfusion
ED	Emergency Department
eGFR	Estimated Glomerular Filtration Rate
PC-AKI	Post-Contrast Acute Kidney Injury

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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None.

Consent to participate

Written informed consent was not required for this retrospective study.

Consent for publication

Not applicable.

Ethical approval

Ethical approval was granted by the Institutional Review Board of the National Guard Health Affairs (NGHA), Riyadh, Saudi Arabia, reference number [NRC21R/231/04], dated [06/03/2022].

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