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Contrast-induced acute kidney injury in patients followed at the intensive care unit after aneurysmal subarachnoid hemorrhage (Fisher grade IV) surgery: A Retrospective study

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ABSTRACT

Background Contrast-enhanced imaging studies are widely used to diagnose and follow up acute cerebrovascular diseases. Exposure to contrast media may lead to nephropathy. This study investigated the incidence of contrast-induced acute kidney injury during intensive care follow-up of patients who underwent aneurysmal subarachnoid haemorrhage surgery and the impact of this condition on patient outcomes.

Material and Methods Patients >18 years of age with no known renal injury and admitted to the intensive care unit after Fisher Grade IV aneurysmal subarachnoid haemorrhage and surgery between January 2017 and June 2022 were retrospectively analysed. Renal injury was defined as a renal injury occurring within 48 hours of exposure to contrast media in line with the Kidney Disease Improving Global Outcomes criteria.

Results Among the 85 patients with subarachnoid haemorrhage who received at least one contrast medium, the mean age was 55, and 40% were female. 11.8% of the patients were found to have early acute kidney injury and were non-oliguric. At 48 hours, six, three, and one patients had Stage 1, 2, and 3 injuries, respectively. None of the patients required renal replacement therapy. Patients received a mean of 2 mL/kg/h saline infusion after contrast media administration and had a mean arterial pressure of 93.6 mmHg. There was no association between acute kidney injury and comorbidities, Glasgow coma scale, or APACHE II scores.

Conclusions The study found that the incidence of contrast-induced acute kidney injury was low and transient in patients followed at the ICU after aneurysmal subarachnoid haemorrhage (Fisher Grade IV) surgery. Adequate hydration and hemodynamic stability were found to be effective in reducing acute kidney injury in these patients.

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INTRODUCTION

Aneurysmal subarachnoid haemorrhage (aSAH) is a devastating condition with high mortality and morbidity.[1] Acute brain injury causes changes in hemodynamic functions due to catecholamine fluctuations and other neurohumoral changes. It increases the risk of acute kidney injury (AKI).[2] The risk of renal dysfunction may be higher for patients with hypertension, diabetes mellitus, and those on nephrotoxic drugs. AKI occurring after aSAH contributes to increased mortality and morbidity.[3]

Computed tomography (CT) is an important imaging modality to diagnose and follow up various diseases. In some cases, intravenous contrast media (IVCM) administration may be required to improve image quality.[4] Contrast agents used in imaging studies to diagnose and follow up intracranial aneurysms are thought to cause an increased risk of renal injury.[5]

IVCM administration has been reported to be the third most common cause of hospital-acquired AKI.[6,7] Increasing contrast media (CM) use has led to growing interest in contrast-induced nephropathy in recent years.6 Contrast-induced AKI (CI-AKI) is an acute decline in renal function after intravascular administration of contrast medium without an alternative cause.[8] The pathophysiological basis of CI-AKI has yet to be fully understood. Still, it is thought to be related to an interaction of hemodynamic changes, an increase in free oxygen radicals, and direct toxic effects on renal tubular cells.[9] IVCM used in imaging is thought to increase the risk of AKI, especially in the high-risk population admitted to the intensive care unit (ICU) after aSAH.[10] This study sought to determine the incidence of AKI according to Kidney Disease Improving Global Outcomes (KDIGO) criteria and its effects on outcomes among patients who had undergone IVCM-enhanced imaging during postoperative care at the ICU after aSAH.

MATERIAL AND METHODS

After obtaining approval from the ethics committee (2011-KAEK-25 2022/06-06), the authors scanned hospital records to identify adult patients aged ≥ 18 years who had received at least one contrast-enhanced cranial radiologic imaging during follow-up at the ICU after surgery for aSAH by the same surgical team between January 2017 and June 2022. Patients were excluded if they had a history of chronic kidney disease, AKI, need for dialysis at the time of hospitalisation, repeated hospitalisations (referral from different department, readmission to intensive care within one month), and missing medical records (incomplete creatinine data in the first 48 hours after IV contrast agents [specimen rejection]). The study was conducted in accordance with the Declaration of Helsinki. This study used the AKI definition published in 2012 by the KDIGO working group (Table 1).11 This definition was based on 24 and 48-hour levels after contrast exposure.

Clinical data were retrospectively analysed using the hospital information system database and patient medical records. The authors recorded demographic variables (age and sex), comorbidities, the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, the Glasgow Coma Score (GCS), IV fluid therapy, urine output, haemoglobin and serum creatinine levels before contrast-enhanced CT (CECT), serum creatinine levels at 24 and 48 hours after CECT, additional risk factors for AKI after ICU admission including sepsis, hemodynamic fluctuations (mean arterial pressure below 60 mmHg and/or need for high vasoactive drugs [> 0.1 microgram/kg/min]), presence of potential nephrotoxic drugs (aminoglycosides, glycopeptides, trimethoprim-sulfamethoxazole, loop diuretics, etc.), n-acetylcysteine (NAC) therapy, contrast agent used and its quantity, the requirement for renal replacement therapy (RRT) within 48 hours of CECT, length of ICU stay, need for permanent renal support at discharge from ICU and ICU discharge status.

Statistical analysis

Patient data collected for the study were analysed using the IBM Statistical Package for the Social Sciences version 23.0 for Windows (IBM Corp., Armonk, NY). Descriptive values were expressed in frequency and percentage for categorical data and median, minimum, and maximum for continuous data. Intergroup comparisons were performed using the Mann-Whitney U test, and categorical variables were compared using the Chi-squared or Fisher's exact test. Statistical significance was set at $p < 0.05$.

RESULTS

The study included 85 patients who were followed at the general ICU after aSAH surgery between January 2017 and June 2022 and met the inclusion crite-

Table 1. KDIGO AKI definition.[11]

AKI stage	Serum creatinin	Urine output
I	1.5-1.9 times baseline or ≥ 0.3 mg/dL increase	< 0.5 mL/kg/h for 6-12 hours
II	2.0-2.9 times baseline	< 0.5mL/kg/h for ≥ 12 hours
III	3.0 times baseline or increase in SCr to ≥ 4.0 mg/dL or initiation of RRT or in patients < 18 years, decrease in eGFR to < 35 mL/min/1.73 m^2	< 0.3 mL/kg/h for ≥ 12 hours or anuria for ≥ 12 hours

KDIGO: Kidney Disease Improving Global Outcomes, AKI: acute kidney injury, SCr: serum creatinine, RRT: renal replacement therapy, eGFR: estimated glomerular filtration rate.

ria (Figure 1). One patient who developed meningitis after aSAH surgery, one who was shocked at the imaging time, and one with only one kidney were excluded. Patients who underwent CECT imaging at our institution during the study's time frame received iohexol (omnipaque, GE Healthcare, USA) and iopromide (ultravist, Bayer Healthcare, Germany) at 0.5-0.75 mL/kg.

According to the Fisher classification system, all patients in the study were diagnosed with Grade IV aSAH.[12] The anterior cerebellar aneurysm was present in 76 patients (89.4%), and the posterior cerebellar aneurysm was present in 9 (10.6%). Seventy-two patients (84.7%) had external ventricular drainage systems in the postoperative period.

Thirty-four patients (40%) were female, and 51 (60%) were male. The median age was 55 years and ranged 21-86 years. Seven patients (8.2%) were smokers, and 48 (56.5%) had at least one comorbid condition. Hypertension was the most common comorbidity in patients with comorbidities, with 51.8% (n: 44). The patients had a median APACHE II score of 26 (5-67.2) and 8 (3-15) median GCS. The mean haemoglobin level was 11.8 g/dL (8.3-16).

Table 2 showed stages of AKI that developed within the first and second day, as well as clinical and laboratory parameters. At 48 hours, six, three, and one patient had Stage 1, Stage 2, and Stage 3 injuries, respectively. None of the patients required RRT. The length of stay at the ICU ranged from 1 to 180 days, with a median length of stay of 5 days. 57.6% (n: 49) of the patients were transferred from the ICU to the ward, and 42.4% (n: 36) died in the ICU. The distribution of demographic and clinical characteristics of the patients by occurrence of AKI within the first and second day was given in Tables 3 and 4. There was no statistical difference between patients with and without AKI regarding comorbidities, amount of

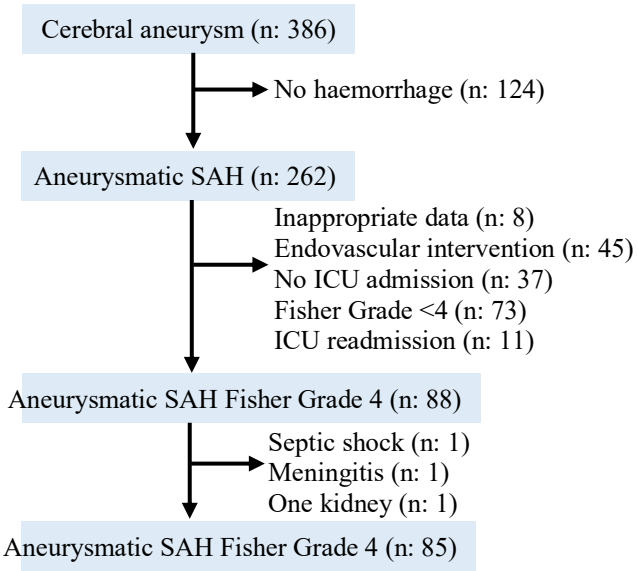

Figure 1. Flow chart of the study. SAH: subarachnoid haemorrhage, ICU: intensive care unit.

Table 2. Distribution of acute kidney injury and clinical characteristics of patients (n: 85).

Age (year)	55 (21:86)
Gender (female/male)	34 (40)/51 (60)
Smoking	7 (8.2)
Comorbidities	48 (56.5)
Hypertension	44 (51.8)
Diabetes mellitus	7 (8.2)
Heart disease	4 (4.7)
Lung disease	3 (3.5)
Other	3 (3.6)
Glasgow coma scale (GCS)	8 (3:15)
APACHE II	26 (5:67.2)
Before CECT	
Haemoglobin	11.8 (8.3:16)
Hematocrit	35.1 (25.3:48.7)
Creatinine	0.8 (0.4:1.8)
BUN	15.8 (4.7:35)
eGFR	96.4 (37.2:164.4)
After CECT first day	
Creatinine	0.8 (0.5:2.1)
BUN	16.9 (5.6:41.0)
eGFR	91.2 (11.3:158.7)
1. day acute kidney injury	
non-AKI patients	75 (88.2)
Stage I	7 (8.2)
Stage II	2 (2.4)
Stage III	1 (1.2)
After CECT second day	
Creatinine	0.8 (0.4:4.4)
BUN	18.3 (7.0:61.2)
eGFR	95.9 (9.8:158.5)
2. day acute kidney injury	
non-AKI patients	75 (88.2)
Stage I	6 (7.1)
Stage II	3 (3.5)
Stage III	1 (1.2)
Sepsis	0
Nephrotoxic drug after CECT (48 hours)	26 (30.6)
Intravenous saline (cc/h; 48 hours)	141.9 (71.4:287.5)
Blood/blood product (48 hours)	6 (7.1)
N-acetylcysteine	12 (14.1)
Vasoactive drug use (48 hours)	17 (20)
RRT (within 48 hours)	0
Length of stay in ICU (day)	5 (1:180)
RRT after discharge	0
ICU discharge	
Exitus	36 (42.4)
Ward	49 (57.6)

APACHE II: acute physiology and chronic health evaluation, CECT: contrast-enhanced computer tomography, BUN: blood urea nitrogen, eGFR: estimated glomerular filtration rate, RRT: renal replacement therapy, ICU: intensive care unit.
The values were expressed as n (%) or median (minimum: maximum).

hydration, hemodynamic parameters, and use of nephrotoxic drugs.

DISCUSSION

This study analysed the KDIGO-defined incidence of AKI within the first 48 hours of contrast-enhanced imaging during ICU stay after aSAH surgery. This study also examined potential risk factors and effects on patient outcomes. Results indicate that 11.76% of the patients had AKI after exposure to CM. These patients did not require RRT during their stay at the ICU. RRT was neither required for seven patients with AKI who were discharged to the ward. The incidence of AKI after exposure to CM is unclear and was reported in one meta-analysis, which ranged from 1% to 20%.[13] These differences have been attributed to differences in the definition of AKI, patient populations and procedures, timing of patient follow-up, and possible changes in patient hydration status.[14] The incidence of CI-AKI reported in the literature varies markedly depending on the definition. A systematic review published by the National Institute for Health and Care Excellence reported a need for full agreement on the definitions for detecting AKI and predicting its outcomes.[15] A prospective study by Jabara[16] used four different definitions to determine CI-AKI and reported that the incidence could vary between 3.3% and 10.5% depending on the definition used.

This study aimed to determine the incidence of CI-AKI according to the criteria adopted by the KDIGO in 2012, which was used in the publications of Laforcade *et al*.[17] in 2021 and included in the latest guidelines of the French Societies of Nephrology and Radiology.[18] Therefore, the incidence reported in our series may not be comparable with studies using different definitions. McDonald *et al*.[19] reported an AKI rate of 9.9% in the group that received IVCM. However, since their study included patients undergoing abdominal, pelvic, and thoracic CT angiography and diagnostic or interventional cardiac catheterisation, they reported that heterogeneity between procedures and patient groups may affect the results. Clec'h *et al*.[20] reported a CI-AKI rate of 16.78% in patients admitted to the medical-surgical ICU. They noted that RRT was required in 29.16% of patients, increased risk of death in the ICU, and prolonged length of ICU stay. They also stated that the populations included in the study had multiple risk factors for the develop-

Table 3. Distribution of demographic and clinical characteristics by occurrence of acute kidney injury (day 1).

Variables	Acute kidney injury (day 1)		*P*-value
	Absent	Present	
Age (year)	56 (30:86)	52 (21:70)	0.336
Gender (female/male)	30 (40)/45 (60)	4 (40)/6 (60)	1.000
Comorbidities	43 (57.3)	5 (50)	0.741
Hypertension	41 (54.7)	3 (30)	0.186
Diabetes mellitus	7 (9.3)	0 (0)	0.592
Glasgow coma scale	8 (3:15)	9 (3:14)	0.626
APACHE II	26 (5:63.9)	32.2 (11.3:67.2)	0.214
Pre-CECT			
Haemoglobin	11.7 (8.6:16)	12.1 (8.3:13.7)	0.364
Hematocrit	35.1 (25.8:48.7)	36.2 (25.3:40.1)	0.433
Creatinine	0.8 (0.4:1.7)	0.9 (0.5:1.8)	0.093
BUN	15.8 (4.7:35)	14.5 (6.9:28.5)	0.854
eGFR	97.6 (37.2:164.4)	87.1 (42.4:154.7)	0.481
Day 1 after CRT			
Creatinine	0.8 (0.5:1.6)	1.5 (1.0:2.1)	< 0.001
BUN	16.4 (5.6:41)	21.7 (8.9:39.3)	0.035
GFR	96.7 (11.3:158.7)	59.1 (28.9:98.3)	< 0.001
Mean arterial pressure (first 24 h)	92.5 (66.6:121.8)	87.5 (70.3:118.9)	0.364
Urine output (hours cc/h)	130.2 (48.8:307)	134.8 (45:200)	0.978
N-acetylcysteine	12 (16)	0 (0)	0.344
Length of stay at the ICU (day)	5 (1:180)	8 (2:105)	0.228
Exit from the ICU			0.737
Ward	44 (58.7)	5 (50)	
Exitus	31 (41.3)	5 (50)	

APACHE II: acute physiology and chronic health evaluation, CECT: contrast-enhanced computer tomography.
The values were expressed as n (%) or median (minimum: maximum).

ment of AKI. The present study was conducted with a patient group (Fisher Grade IV aSAH), which we thought would have fewer confounding factors. We tried to determine the direct effect of CM on the kidney by minimising potential clinical confounders and found that CM had a minimal and transient impact on renal function.

The American College of Radiology reported that AKI after exposure to CM may be associated with the patient's underlying comorbidities rather than with CM.[4] Patients diagnosed with aSAH are usually relatively healthy young individuals. These patients have a low prevalence of comorbidities such as diabetes mellitus, hypertension, and heart failure.[21] Although the patient group in this study was relatively young (a mean age of 55), 48 (56.5%) had at least one comorbid condition that could affect the kidney. In addition, six patients who developed AKI had at least one comorbidity. A history of premorbid hypertension is a known risk factor for aSAH.[22] Although hypertension was present in 51.8% of the patients in this study, it had no significant association with AKI. However, the definition and duration of premorbid hypertension are unknown.

The incidence of CI-AKI in patients with mild-to-moderate renal impairment and diabetes mellitus has been reported to range from 9% to 50%.23 Unlike McCullough's series[24], which included 24.8% diabetic patients and reported that AKI might be associated with diabetes mellitus, the series in the present study had a lower incidence of diabetes (8.2%). While none of the patients who developed AKI on day 1 had diabetes mellitus, 10% of those who developed AKI had diabetes mellitus. Although one study reported that CM injection may increase susceptibility to AKI in patients with diabetes mellitus [25], the present study found that the presence of diabetes mellitus did not increase the risk of AKI. This difference may be due to

Table 4. Distribution of demographic and clinical characteristics by occurrence of acute kidney injury (day 2).

Variables	Acute kidney injury (day 2)		*P*-value
	Absent	Present	
Age (year)	55 (23:86)	57 (21:72)	0.978
Gender (female/male)	29 (38.7)	5 (50)	0.512
	46 (61.3)	5 (50)	
Smoking	7 (9.3)	0 (0)	0.592
Comorbidities	42 (56)	6 (60)	1.000
Hypertension	39 (52)	5 (50)	1.000
Diabetes mellitus	6 (8)	1 (10)	1.000
Glasgow coma scale (GCS)	8 (3:15)	13 (3:15)	0.422
APACHE II	26.2 (5:58)	21 (8.7:67.2)	0.881
Pre-CECT			
Haemoglobin	11.8 (8.6:16)	11.8 (8.3:13.4)	0.662
Hematocrit	35.1 (25.8:48.7)	35.6 (25.3:39.4)	0.521
Creatinine	0.8 (0.4:1.8)	0.8 (0.6:1.3)	0.854
BUN	15.8 (4.7:35)	14.8 (11.0:34.1)	0.989
eGFR	96.4 (37.2:154.7)	92.4 (42.4:164.4)	0.761
CECT 48 hours			
Creatinine	0.8 (0.4:2.0)	1.4 (0.9:4.4)	<0.001
BUN	17.8 (7:43)	26.5 (16.8:61.2)	0.001
eGFR	100.5 (36.9:158.5)	47 (9.8:101.6)	<0.001
Mean arterial pressure (first 48 hours)	93.6 (64.8:174.6)	95.2 (73.2:107.1)	0.956
Nephrotoxic drug after CECT	23 (30.7)	3 (30)	1.000
Hourly IV fluid (48 hours)	141.9 (71.4:287.5)	136.3 (92.5:265)	0.723
Urine (cc/h)	132.7 (48.8:307)	115.4 (45:238.2)	0.544
Blood/blood product (48 hours)	6 (8)	0	1.000
N-acetylcysteine	12 (16)	0	0.344
Vasoactive drug use	14 (18.7)	3 (30)	0.411
Length of stay at the ICU (day)	5 (1:115)	7 (2:180)	0.317
Exit from ICU			1.000
Ward	43 (57.3)	6 (60)	
Exitus	32 (42.7)	4 (40)	

APACHE II: acute physiology and chronic health evaluation, CECT: contrast-enhanced computer tomography, BUN: blood urea nitrogen, eGFR: estimated glomerular filtration rate, IV: intravenous, ICU: intensive care unit.

The values were expressed as n (%) or median (minimum: maximum).

the relatively younger age of the patients or the shorter disease duration, which may influence the development of micro- and macrovascular complications of diabetes mellitus. Kellum *et al*.[26] reported that even small increases in serum creatinine may affect survival in critically ill patients. Zhang *et al*.[27] reported that the development of AKI after intracranial aneurysm clipping surgery was associated with poor prognosis. Retrospective studies by McDonald *et al*.[28] and Davenport *et al*.[29] compared clinically similar patients who underwent CT scanning with or without contrast enhancement. Both studies achieved results similar to the present study; exposure to CM did not affect clinical outcomes related to AKI in patients with normal baseline renal functions.

Serum creatinine levels and glomerular filtration rate are important parameters that provide information about renal function.[30] This study minimised confounding factors by recruiting patients with normal baseline glomerular filtration rate and creatinine levels. Furthermore, good baseline renal functions may reduce the risk of AKI after IVCM exposure in aSAH patients who are relatively young and have few comorbidities. A single-centre retrospective study by Clec'h *et al*.[20] reported a CI-AKI incidence of 16.8% in adult patients who underwent CECT imaging for emergency diagnosis. However, they noted that they were unable to investigate how the incidence of AKI

was affected by the presence of coexisting risk factors for kidney injury, such as sepsis, nephrotoxic drugs, and hemodynamic impairment, or how it was affected by CM volume and implementation of prophylactic measures. Hydration, a prophylactic measure against CI-AKI, is important in preventing ischemic conditions.[31] A prudent approach for all patients receiving CM is to ensure adequate hydration before and after imaging.[32] Urine viscosity increases in proportion to the volume of CM injected. Adequate hydration has been shown to decrease urine viscosity.[33] To better characterise patients regarding hydration status, this study analysed intravenous fluid administered to the patients. The guidelines published by the European Society of Cardiology in 201434, which contains important preventive strategies against CM-associated AKI, recommend peri-procedural hydration with intravenous saline at a rate of 1-1.5 mL/kg/h 3-12 hours before CM administration and 1-1.5 mL/kg/h 12-24 h after the procedure. The patient population in the present study received a mean saline infusion of 2 mL/kg/h within the first 48 hours of CM administration. This fluid regimen may have played a role in the transience of AKI. The same guideline recommends using iso-osmolar or low-osmolar CM in addition to hydration.[34] The contrast agents administered to the patients in this study were nonionic iso-osmolar iohexol (omnipaque, GE Healthcare, USA) and iopromide (ultravist, Bayer Healthcare, Germany). Some authors doubt whether modern iodinated (iso-osmolar) contrast is nephrotoxic.[35] Several studies have evaluated the renal safety of nonionic contrast agents in patients with impaired renal function after intra-arterial contrast injection only during percutaneous coronary angiography (PCA).[35] However, few publications have evaluated the incidence of CI-AKI after intravenous injection of iso-osmolar CM and found a low risk.[36,37] Although CI-AKI found in this study was higher than in previous studies, it was transient and did not lead to permanent kidney damage or need for renal support. The amount of iso-osmolar CM used for patients in this study was similar, approximately 0.5-0.75 mL/kg and may be relatively low compared to doses used in interventional intra-arterial applications such as PCA.

N-acetylcysteine (NAC) is most commonly used as a mucolytic in ICUs to facilitate ciliary activity.[38] It is also used for the treatment of paracetamol toxicity[39] and hepatic failure[40], acute lung injury,[41] sepsis, renal failure, and carbon monoxide poisoning.[42] Tepel *et al*.[43] reported that IV or oral administration of NAC was nephroprotective in preventing CI-AKI by eliminating free radicals. On the other hand, Suva *et al*.[44] emphasised that the use of NAC to prevent CI-AKI ended with inconsistent and uncertain results. There is no routine protocol used to avoid CM damage in our unit. However, NAC has been reported to be a potential prophylactic treatment against CI-AKI.[45] In the present study, NAC was used to increase mucolytic activity in 14.1% of the patients in the ICU. Analysis of the prophylactic role of NAC against CI-AKI in this study did not yield a statistical difference. However, none of our patients who developed AKI had received NAC therapy, suggesting it might be clinically important. This effect can be determined in future studies with a larger sample size.

Many studies suggested that most cases of AKI following contrast administration may be related to incidental nephrotoxic exposures (e.g., hypovolemia, cardiac dysfunction, and infection) that were present at the time of CM exposure.[46,47] CI-AKI prevention requires discontinuing nephrotoxic drugs and adjusting hemodynamic parameters.[48] In the present study, 30.6% of the patients were on nephrotoxic medications, and there was no significant difference between patients with and without AKI. The short hospital stays among the patients in this study may have played a role in preventing the occurrence of AKI caused by nephrotoxic drugs.

Zhang *et al*.[28] reported that preoperative aneurysm rupture was a risk factor for AKI. The existence of a link mediated by cytokines in this process is yet to be investigated.[49,50] All of the patients in this study were patients with ruptured aSAH. Patients with above-normal creatinine levels during postoperative follow-up at the ICU were excluded from the study to try to rule out the possibility of an early contribution of the procedure to the development of AKI. However, cytokine release may also have an effect on AKI during the later period. Although the combination of induced hypertension, hypervolemia and hemodilution (triple-H therapy) is often utilised to prevent and treat cerebral vasospasm after aSAH, its efficacy and precise role in managing the acute phase remains unclear.[51] In this study, triple-H therapy is hypothesised

to impact CI-AKI outcomes. Hemodynamically, this hypothesis seems supported by mean arterial pressures as high as 93.6 (64.8-174.6) mmHg.

GCS is widely used to assess patients with head trauma or other types of acute brain injury to guide early treatment. Lee *et al*.[5] investigated the incidence and clinical effect of CI-AKI after coil embolisation in patients with aSAH. They found that 50% of the patients in the CI-AKI group had a GCS below [8], compared to 19.7% in the group without CI-AKI. They reported that a low baseline GCS score may be associated with CI-AKI. Patients followed during postoperative ICU in the present study had a mean baseline GCS score of [8]. There was no significant correlation between GCS and AKI observed at 24 and 48 hours after CM exposure. Compared with other types of AKI (such as ischemic AKI), contrast-induced AKI is usually characterised by a relatively rapid recovery of renal functions. Serum creatinine levels that increase in CI-AKI return to baseline within 3-7 days.[52] Since AKI is typically mild, most patients (except those with moderate-to-severe chronic kidney disease) are non-oliguric.[53] This suggests that treatments to maintain hemodynamic stability and adequate hydration are effective.

In the present study, the mortality rate was 36%, and transient AKI that developed early during hospital stay was not associated with mortality. Ehrlich *et al*.[54] reported that renal functions were not affected after contrast-enhanced CT in patients presenting with acute stroke, and CT angiography offered additional clinical value. This result is in line with the present study, which showed that renal injury was transient and reversible in patients with normal renal function during diagnosis and treatment.

Limitations

The major limitation of this retrospective study was that patients with aSAH who received CECT were not compared with those who did not. Thus, the isolated effect of aSAH on renal function could not be investigated. However, this can be overlooked since the primary aim of this study was not to determine the absolute toxicity of IVCM but to determine the incidence of CI-AKI and to identify potentially dangerous conditions that should be considered before exposing patients to IVCM. Also, the small number of patients with CI-AKI may increase the possibility of error. Therefore, a larger-scale study may improve the statistical reliability of the associated findings. Finally, this study excluded patients with AKI and thus did not investigate the adverse effect of CM on renal function in patients with aSAH and AKI or CI-AKI.

CONCLUSIONS

This study found that young patients with mild comorbidities and without aggravating factors such as severe anaemia, hypovolemia, and hypotension might have reversible fluctuations in renal function that did not affect clinical outcomes. This means that clinically required imaging studies should not be avoided due to fear of CI-AKI.

Conflict of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical Approval

The protocol of the study was approved by the Medical Ethics Committee of Healt of Sience University, Bursa Training and Research Hospital, Bursa, Turkey. (Decision number: 2011/KAEK-25, date: 06.06.2022).

Authors' Contribution

Study Conception: HAK, IC; Study Design: HAK, IC; Literature Review: RA; Critical Review: IC; Data Collection and/or Processing: HAK,; Analysis and/or Data Interpretation: IC; Manuscript preparing: HAK, RA, IC.

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