

Is Contrast-Enhanced Imaging in the Emergency Department a Risk Factor for Contrast-Associated Acute Kidney Injury?

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Purpose: We aimed to determine the predisposing factors for contrast-associated acute kidney injury (CA-AKI) among patients exposed to contrast media during emergency department visits and follow-ups in internal medicine, and to evaluate subsequent outcomes such as mortality and intensive care unit (ICU) admission.

Patients and Methods: A total of 1483 patients who presented to the internal medicine clinic from the emergency department or outpatient clinic and underwent contrast-enhanced computed tomography (CT) were retrospectively analyzed. A total of 382 cases with kidney function tests available for the study before contrast exposure and at 24, 72, and 120 hours after exposure were included in the study. An increase in serum creatinine by 25% or more from the baseline or an absolute increase of 0.5 mg/dL (44.20 µmol/L) or more after one hour was considered as contrast nephropathy.

Results: Contrast-associated acute kidney injury was observed in 17% of the study population, with the majority of cases emerging within the initial 24-hour period following contrast exposure. In 54% of these patients, contrast media had been administered in the internal medicine department. Among them, 13.9% subsequently required admission to the intensive care unit (ICU). Furthermore, individuals with a history of hypertension were found to have a 2.31-fold increased risk of developing CA-AKI compared to those without hypertension.

Conclusion: The likelihood of contrast-associated nephropathy appears to be markedly increased in hypertensive individuals undergoing contrast-enhanced CT in emergency departments. Prophylactic intravenous hydration has also been demonstrated to play a crucial role in reducing CA-AKI incidence.

Keywords: contrast nephropathy, acute kidney failure, emergency service, hypertension

Introduction

Acute kidney injury (AKI) manifests as an abrupt deterioration in renal performance, typically emerging within a short timeframe ranging from several hours to a few days. This condition is frequently associated with impaired elimination of metabolic by products, fluid imbalance, and electrolyte disturbances.^{1,2} It is widely accepted that AKI can be classified into three main categories: pre-renal AKI, renal AKI, and post-renal AKI.³ Contrast-associated acute kidney injury (CA-AKI) is a frequently reversible cause of renal AKI and occurs shortly after the administration of contrast agents. It can be diagnosed only after all other causes of AKI have been excluded.^{4,5} The most widely accepted definition of CA-AKI is an increase in serum creatinine of 25% or more from baseline, or an absolute increase of 0.5 mg/dL (44.20 µmol/L) or more, occurring 24 to 120 hours after contrast administration.⁶ Currently, there is no sensitive or specific biomarker for CA-AKI. The onset typically occurs 24 to 48 hours after contrast exposure, peaks between days 7 and 10, and resolves within a few days. Typically, no clinical signs are observed in cases of contrast nephropathy.⁷⁻⁹

Preventive strategies are essential for the proper management of patients and for identifying individuals at high risk of developing contrast-associated nephropathy. The main risk factors for CA-AKI include diabetes mellitus (DM), advanced

age, chronic kidney disease (CKD), anemia, and congestive heart failure (CHF). Additionally, the clinical severity of AKI, the use of nephrotoxic drugs, the amount of contrast used, and the need for intensive care have also been suggested as contributing risk factors.^{6,10–12} Currently, there is no specific treatment protocol for contrast-associated nephropathy. Clinicians should identify high-risk patient populations and plan preventive treatment accordingly.¹³ Ensuring sufficient intravascular volume and preferring low-osmolar contrast agents have been shown to provide substantial protection against nephrotoxic effects.¹²

This study aimed to identify the risk factors for the development of contrast-associated nephropathy in patients exposed to contrast agents, both at the time of admission to the emergency department and during follow-up in the internal medicine department. Additionally, it sought to determine the mortality rate and the need for intensive care unit (ICU) admission in patients with and without contrast-associated nephropathy.

Materials and Methods

A total of 1483 patients who presented to the Internal Medicine Department of Izmir Katip Celebi University Atatürk Training and Research Hospital from the emergency department or outpatient clinic and underwent contrast-enhanced computed tomography between January 2017 and January 2021 were retrospectively analyzed. The study population consisted of 382 individuals with eligible kidney function tests, who were included in the study before contrast exposure and at 24, 72, and 120 hours after exposure. All procedures were conducted in accordance with the ethical standards of the committee responsible for human experiments (institutional and national) and the 1975 Helsinki Declaration, revised in 2013. Ethical approval was obtained from our hospital's ethics committee on 21.04.2022, under protocol number 0213. All contrast media were administered intravenously, using Iohexol at a concentration of 300 mgI/mL.

The medical history, laboratory results (Blood Urea Nitrogen (BUN), creatinine, glomerular filtration rate (GFR), total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides), imaging, and clinical follow-up of the patients included in the study were retrospectively obtained from the hospital information management system. The GFR and serum creatinine values of the patients were measured before contrast exposure and at 24, 72, and 120 hours after contrast exposure. In this study, an increase in serum creatinine of 25% or more from baseline, or an absolute increase of 0.5 mg/dL (44.20 µmol/L) or more within one hour, was considered as contrast-associated nephropathy.

Statistical Analysis

The patient data collected in the study were analyzed using IBM Statistical Package for the Social Sciences (SPSS) for Windows 26.0 (IBM Corp., Armonk, NY). Descriptive values were presented as frequency and percentage for categorical data, and as mean and standard deviation for continuous data. For inter-group comparisons, the “Independent Samples *t*-test” was used for two groups, and the “Pearson Chi-Square Test” was used for the comparison of categorical variables. The normality assumption was checked using the Shapiro–Wilk test. The Independent Samples *T*-test was performed to compare the means of two independent groups with normal distribution. Binary Logistic Regression analysis was used to model the categorical dependent variable and evaluate statistically significant effects on contrast-associated nephropathy. Results were considered statistically significant when the *p*-value was less than 0.05.

Results

Out of 1,483 retrospectively reviewed patient records, 382 individuals who fulfilled the inclusion criteria were selected for analysis. Table 1 provides an overview of the demographic features of the study population. Hypertension was the most common condition, and diuretics were the most frequently used medication. Contrast-associated nephropathy developed in 17% of the cases, predominantly on the first day. More than half of the cases (54%) received contrast media in the internal medicine department, and 13.9% of this subgroup subsequently required admission to the intensive care unit (Table 1).

No gender differences were observed in cases that developed contrast-associated nephropathy. However, it was found that the likelihood of exposure to contrast agents in the emergency department was significantly higher in cases that developed contrast-associated nephropathy. Patients who experienced contrast-associated acute kidney injury had significantly higher rates of intensive care unit admission and mortality; however, this association may reflect the greater

Table 1 Basic Demographic and Descriptive Characteristics of the Study Population

Gender (n,%)	
Female	173 (45.3)
Male	209 (54.7)
Age (mean±SD, year)	64.0±16.8
Comorbid Diseases (n,%)	
Hypertension	164 (42.9)
Chronic renal failure	22 (5.8)
Diabetes Mellitus	106 (27.7)
Atrial fibrillation	17 (4.5)
Hyperlipidemia	30 (7.9)
COPD ¹	26 (6.8)
Congestive heart failure	84 (22.0)
Medication	
ARB ²	28 (7.3)
Statins	28 (7.3)
NSAIDs ³	32 (8.4)
Diuretics	85 (22.3)
ACEI ⁴	58 (15.2)
Contrast Media Administration (n,%)	
Emergency Department	178 (46)
Internal Medicine Clinic	204 (54)
Contrast Nephropathy (n,%)	
Development of Contrast Nephropathy Day (n,%)	65 (17)
First Day	26 (40)
Third day	20 (30.8)
Fifth day	19 (29.2)
Hemodialysis (n,%)	
Permanent hemodialysis	11 (2.9)
Temporary hemodialysis	17 (4.5)
Hospitalization duration (mean±SD, days)	13.9±9.9
Transfer to intensive care unit (n,%)	53 (13.9)
Discharge (n,%)	311 (81.4)
Mortality (n,%)	69 (18.1)

Notes: Values are presented as mean ± standard deviation (SD) or n (%), as appropriate.

Abbreviations: COPD¹, Chronic Obstructive Pulmonary Disease; ARB², Angiotensin Receptor Blocker; NSAIDs³, non-steroidal anti-inflammatory drug; ACEI⁴, Angiotensin- Converting Enzyme Inhibitor.

baseline severity of illness and comorbidity burden in these patients, which could predispose them to both sign admission and the development of CA-AKI. Patients who experienced contrast-associated nephropathy exhibited significantly elevated rates of intensive care unit admission and mortality. When examining additional treatments administered before and after contrast exposure, it was found that patients who received isotonic fluid therapy before or after contrast exposure had significantly fewer cases of contrast-associated nephropathy (Table 2).

It was found that contrast-associated nephropathy was higher in all chronic diseases; however, this increase was statistically significant only in cases of hypertension (HT), diabetes mellitus (DM), hyperlipidemia, and congestive heart failure (CHF) ($p=0.001$; $p=0.034$; $p=0.013$; $p=0.011$, respectively). When evaluating the relationship between long-term medication use and contrast-associated nephropathy, it was determined that contrast-associated nephropathy was significantly more frequent in patients using lipid-lowering drugs and diuretics (Table 3).

Table 2 Comparison of Contrast Nephropathy and Site of Contrast Media Administration

	Contrast Nephropathy		p-value	Contrast Media Administration		p-value
	Yes n=65	No n=317		Emergency Service n=178	Clinic Service n=204	
Gender n (%)						
Female	30 (17.3)	143 (82.7)	0.878	89 (51.4)	84 (48.6)	0.084
Male	35 (16.7)	174 (35.0)		89 (43.6)	120 (57.4)	
Contrast Media Administration n (%)						
Emergency Department	41 (23.0)	137 (77.0)	0.003	-	-	-
Internal Medicine Clinic	24 (11.8)	180 (88.2)		-	-	
Hemodialysis n (%)						
Permanent hemodialysis	4 (45.5)	6 (54.5)	<0.001	8 (72.7)	3 (27.3)	0.078
Temporary hemodialysis	10 (58.8)	7 (41.2)	0.025	14 (82.4)	3 (17.6)	0.002
Referral to intensive care unit n (%)	15 (28.3)	38 (71.7)	0.018	29 (54.7)	24 (45.3)	0.202
Discharge n (%)	45 (14.5)	266 (85.5)	0.006	141 (45.3)	170 (54.7)	0.302
Mortality n (%)	19 (27.5)	50 (72.5)	0.010	35 (50.7)	34 (49.3)	0.448
Before Contrast Administration n (%)						
N-acetylcysteine Treatment	9 (13.0)	60 (87.0)	0.327	5 (7.2)	64 (92.8)	<0.001
Sodium Bicarbonate Treatment	2 (20)	8 (80)	0.682	2 (20)	8 (80)	0.114
Isotonic Therapy	15 (6.6)	212 (93.4)	<0.001	90 (39.6)	147 (60.4)	0.001
After Contrast Administration n (%)						
N-acetylcysteine Treatment	11 (14.7)	64 (85.3)	0.546	10 (13.3)	65 (86.7)	<0.001
Sodium Bicarbonate Treatment	4 (33.3)	8 (66.7)	0.129	4 (33.3)	8 (66.7)	0.349
Isotonic Therapy	23 (8.6)	245 (91.4)	<0.001	128 (47.8)	140 (52.2)	0.484

Notes: Values are presented as n (%). Italicised values indicate statistically significant differences ($p < 0.05$).

When laboratory results were compared, it was found that in patients with contrast-associated nephropathy, pre- and post-contrast BUN, post-contrast creatinine, and pre-contrast uric acid levels were significantly higher (Table 4). The location of contrast administration was grouped as the emergency department and internal medicine department. When comparing gender, age, temporary and permanent hemodialysis status, discharge status, intensive care unit (ICU) status, and mortality, it was found that only patients receiving temporary hemodialysis were significantly more likely to be in the emergency department ($p=0.002$). In terms of pre- and post-contrast treatments, it was observed that patients who received N-acetylcysteine before contrast administration and those who received N-acetylcysteine after contrast administration were significantly more likely to be followed up in clinical services (Table 2). When comparing laboratory results based on the location of contrast administration, all laboratory values, except for pre-contrast uric acid, triglycerides, and LDL, were significantly higher in the emergency department cases (Table 4).

In the Binary Logistic Regression analysis conducted to evaluate the statistically significant effects on contrast-associated nephropathy, the Odds Ratio (OR) for the hypertension variable was calculated as 2.315. The likelihood of developing contrast-associated nephropathy was 2.315 times higher in individuals with hypertension compared to those without hypertension. The Odds Ratio for the location of contrast administration (emergency department) was calculated as 2.485. The likelihood of developing contrast-associated nephropathy was 2.485 times higher when contrast was administered in the emergency department compared to when it was administered in the internal medicine clinic. Additionally, the OR for pre-contrast isotonic fluid therapy was calculated as 0.366, and for post-contrast isotonic fluid therapy as 0.262. (Table 5).

Discussion

The development of CA-AKI is associated with patient-related risk factors, physical and chemical characteristics, and the amount of contrast material used. The majority of existing literature on CA-AKI has focused on patients undergoing percutaneous coronary interventions. In contrast, limited data are available regarding its occurrence in emergency

Table 3 Contrast Nephropathy Associated with Chronic Disease and Medications

Chronic disease, n (%)	Contrast Nephropathy		p-value
	Yes n=65	No n=317	
Hypertension			<i>0.001</i>
Yes	40 (24.4)	124 (75.6)	
No	25 (11.5)	193 (88.5)	
Chronic kidney disease			0.066
Yes	7 (33.3)	14 (66.7)	
No	58 (16.10)	303 (83.9)	
Type 2 Diabetes			<i>0.034</i>
Yes	25 (23.6)	81 (76.4)	
No	40 (14.5)	236 (85.5)	
Atrial Fibrillation			1,000
Yes	3(23.6)	14 (82.4)	
No	62 (17)	303 (83)	
Hyperlipidemia			<i>0.013</i>
Yes	10 (33.3)	20 (66.7)	
No	55 (15.6)	297 (84.4)	
COPD ¹			0.416
Yes	6 (23.1)	20 (76.9)	
No	59 (16.6)	297 (83.4)	
Congestive Heart Failure			<i>0.011</i>
Yes	22 (26.2)	62 (73.8)	
No	43 (14.4)	255 (85.6)	
Medications n (%)			
ARB ²			0.292
Yes	7 (25)	21 (75)	
No	58 (16.4)	296 (83.6)	
Statins			<i>0.023</i>
Yes	9 (36)	16 (64)	
No	56 (15.7)	301 (84.3)	
NSAIDs ³			0.785
Yes	6 (18.8)	26 (81.3)	
No	59 (16.9)	291 (83.1)	
Diuretics			<i>0.005</i>
Yes	23 (17.1)	62 (72.9)	
No	42 (14.1)	255 (85.9)	
ACEI ⁴			0.668
Yes	22 (26.2)	62 (73.8)	
No	54 (16.7)	270 (83.3)	

Notes: Values are presented as n (%). Italicised values indicate statistically significant differences ($p < 0.05$).

Abbreviations: COPD¹, Chronic Obstructive Pulmonary Disease; ARB², Angiotensin Receptor Blocker; NSAIDs³, non-steroidal anti-inflammatory drug; ACEI⁴, Angiotensin- Converting Enzyme Inhibitor.

department settings and subsequent follow-up periods. CA-AKI may develop at higher rates in the emergency department compared to clinical services due to various factors such as high patient load, limited follow-up duration, and concerns for potentially life-threatening conditions.

Table 4 The Relationship Between the Administration of Contrast Media and the Results of Laboratory Tests

	Contrast Nephropathy		p-value	Contrast Media Administration		p-value
	Yes n=65	No n=317		Emergency Service n=178	Clinic Service n=204	
BUN¹ before contrast media (mean±SD, mmol/L)	10.75± 6.39	8.50± 6.64	<i>0.012</i>	10.17 ± 8.10	7.71±4.78	<i><0.001</i>
BUN¹ after contrast media (mean±SD, mmol/L)						
First Day	12.42±7.50	7.42±5.42	<i><0.001</i>	9.92±7.64	6.82±3.82	<i><0.001</i>
Third Day	12.14±7.92	6.39±4.60	<i><0.001</i>	8.50±7.21	6.35±3.75	<i><0.001</i>
Fifth Day	13.60±8.42	6.21±4.60	<i><0.001</i>	8.46±7.39	6.57±4.53	<i>0.003</i>
Creatinine before contrast media (mean±SD, µmol/L)	132.6±150.2	97.2±114.9	0.087	123.7±159.1	79.5±70.7	<i>0.001</i>
Creatinine after contrast media (mean±SD, µmol/L)						
First Day	159.1±159.1	88.4±97.2	<i><0.001</i>	123.7±150.2	79.5±61.8	<i><0.001</i>
Third Day	159.1±150.2	79.9±97.2	<i><0.001</i>	114.9±150.2	79.5±53.0	<i>0.002</i>
Fifth Day	176.8±167.9	79.5±79.5	<i><0.001</i>	114.9±141.4	79.5±53.0	<i>0.003</i>
Uric acid before contrast media (mean±SD, mmol/L)	0.36 ±0.18	0.30±0.14	<i>0.021</i>	0.32±0.16	0.30±0.14	0.233
Cholesterol before contrast media (mean±SD, mmol/L)	4.33±2.66	4.33±2.66	0.053	3.96±2.13	3.55±1.42	<i>0.035</i>
Triglyceride before contrast media (mean±SD, mmol/L)	2.84±8.32	1.64±1.82	0.284	2.15±5.44	1.57±0.86	0.185
LDL² before contrast media (mean±SD, mmol/L)	2.28±1.18	2.06±1.16	0.195	2.15±1.05	2.04±1.25	0.355

Notes: Values are presented as mean ± SD. Italicised values indicate statistically significant differences ($p < 0.05$).

Abbreviations: BUN¹, blood urea nitrogen; LDL², Low Density Lipoprotein.

Table 5 Logistic Regression Analysis for Contrast Nephropathy Status

	p-value	OR ¹	95% CI ²	
			LL ³	UL ⁴
Hypertension	<i>0.021</i>	2.315	1.132	4.732
Diabetes Mellitus	0.826	0.922	0.449	1.896
Hyperlipidemia	0.980	0.975	0.786	4.778
Congestive heart failure	0.437	1.356	0.629	2.924
Emergency Department	<i>0.003</i>	2.485	1.417	4.256
Statins	0.544	1.836	0.258	13.054
Diuretics	0.597	1.295	0.614	2.734
Isotonic Therapy (pre-contrast)	<i>0.038</i>	0.366	0.142	0.945
Isotonic Therapy (post-contrast)	<i>0.004</i>	0.262	0.106	0.649

Notes: Italicised values indicate statistically significant differences ($p < 0.05$).

Abbreviations: OR¹, odds ratio; CI², confidence interval; LL³, Lower Limit; UL⁴, Upper Limit.

In our study, the development of CA-AKI was more common in the emergency department (23%) compared to the internal medicine department (11.8%). There is considerable heterogeneity in the reported incidence of CA-AKI. In a study conducted by Mitchell et al, involving 174 patients who underwent contrast-enhanced computed tomography (CT) scans in the emergency setting due to suspected pulmonary embolism, the rate of CA-AKI was identified as 14%. In another study, Türedi et al reported an incidence of 23.7% in 257 patients who presented to the emergency department with suspected pulmonary embolism and underwent contrast-enhanced CT.^{14,15}

Some studies have suggested that female gender may be an independent risk factor for contrast-associated kidney injury, with a higher incidence observed in women within 72 hours of contrast exposure.^{16,17} No significant difference in the incidence of CA-AKI was observed between male and female patients, possibly due to a similar distribution of major CA-AKI risk factors across sexes in our cohort. In our study, consistent with previous literature, a significant association was observed between advanced age and the development of contrast-associated nephropathy.^{18–20} Palli et al reported

a significantly higher prevalence of nephropathy in patients over 65 years of age (38.64%) compared to younger individuals ($p=0.015$).²¹ Age-related structural and hemodynamic renal changes—including nephron loss, glomerulosclerosis, reduced vasodilatory response, and increased vasoconstriction—may collectively impair renal perfusion and increase susceptibility to nephrotoxic exposure in the elderly.^{22–25} These mechanisms likely contribute to the heightened risk of nephropathy with increasing age. Our findings support the notion that age is an independent risk factor for contrast-associated kidney injury.

When the relationship between the comorbidities observed in our patients and contrast nephropathy was analyzed, hypertension (HT) was found to be the most frequently associated condition, and a significant association was also found with diabetes mellitus (DM). In the logistic regression analysis, HT was associated with 2.315-fold increased risk of developing contrast-associated nephropathy. Rihal et al and Mehran et al reported that HT is a risk factor for CA-AKI.^{6,20} Many studies have identified DM as an independent risk factor for CA-AKI in multivariate analyses.^{6,20,26,27} Davidson and colleagues reported that diabetic patients without nephropathy did not develop contrast nephropathy. The incidence of CA-AKI in diabetic patients varies depending on kidney function and has been reported to range from 5% to 30%.²⁶ The reason why DM is a risk factor may be due to its associated microvascular complications. Regarding hypertension, further studies are needed to determine whether its effect on the kidneys is due to the condition itself or the antihypertensive medications used by these patients. In particular, the use of diuretics and renin–angiotensin–aldosterone system (RAAS) inhibitors may exacerbate hemodynamic alterations, reduce renal perfusion in susceptible patients, and thereby contribute to the risk of CA-AKI. Interestingly, despite CKD being a well-established risk factor for contrast-associated nephropathy in previous literature, our study did not find a statistically significant association. This may be attributed to factors such as the limited number of patients with advanced CKD, the routine use of protective measures in this population, or potential misclassification due to the retrospective nature of the data. Further prospective studies are warranted to explore this observation.

We found that the risk increased in patients using statin group antihyperlipidemic drugs and diuretics; however, while these variables were statistically significant in the univariate analysis, they did not remain significant in the logistic regression model. Toso et al demonstrated that statin use was associated with a notably lower risk of developing CA-AKI in patients who underwent coronary angiography.²⁸ In contrast, in the study by Nozou et al, the development of CA-AKI was more common among patients using statins, and the difference was statistically significant.²⁹ These conflicting results may be related to differences in patient populations, contrast exposure, and procedural settings. In our study, the widespread use of statins among patients with hyperlipidemia and the relatively small sample size may have influenced the statistical outcome. In the study by Kiski et al, diuretic use was associated with an increased risk of CA-AKI.¹⁷ In another study, angiotensin-converting enzyme inhibitors (ACEIs) have been associated with an increased risk of CA-AKI.²⁰ However, Palli et al found no significant relationship between the use of either ACEIs or NSAIDs and the development of CA-AKI.²¹ In our study, we also observed no significant association between these drug groups and contrast-associated nephropathy. The number of patients using NSAIDs was low, and for this reason, we believe that we may not have been able to detect any statistical significance.

Our findings did not reveal a statistically significant association between pre-contrast patients' lipid levels and the incidence of CA-AKI. However, lipid levels were found to be higher in patients who developed CA-AKI. Current literature includes only a small number of investigations exploring the potential role of hyperlipidemia as a risk factor for CA-AKI. Rihal et al found that hyperlipidemia is not a risk factor for CA-AKI.²⁰ Hypercholesterolemia and oxidized low-density lipoprotein (LDL) are known to impair endothelium-dependent vasodilation by reducing nitric oxide (NO) production and synthase activity, while promoting vasoconstriction through increased free oxygen radical formation.^{30–32} Therefore, it is thought that these cases may be at increased risk of developing contrast-associated acute kidney injury.

Our study revealed that patients who developed CA-AKI had significantly elevated baseline uric acid levels prior to contrast administration compared to those who did not. Some studies have investigated the relationship between pre-contrast exposure uric acid levels and the development of CA-AKI, and similar to our findings, uric acid levels were found to be statistically significantly higher in patients who developed CA-AKI.^{33,34}

In our study, fluid therapy administered before and after contrast administration was found to be effective in preventing contrast-associated acute kidney injury (CA-AKI). In the logistic regression analysis, pre-contrast isotonic fluid therapy was associated with a 0.366-fold risk (OR: 0.366), and post-contrast therapy with a 0.262-fold risk (OR: 0.262) of developing CA-AKI. These findings suggest that intravenous hydration, both before and after contrast

exposure, significantly reduces the likelihood of nephropathy and supports its role as a key preventive measure in at-risk patients. Sodium bicarbonate and N-acetylcysteine treatments, however, did not show a significant effect in preventing CA-AKI when given before and after contrast exposure. In a randomized controlled study conducted by Maioli et al, fluid therapy alone was compared with sodium bicarbonate infusion combined with fluid therapy administered before and after primary percutaneous coronary intervention. It was shown that hydration therapy with sodium bicarbonate before and after the procedure was more effective than hydration with isotonic saline administered only after the procedure.³⁵ According to the European Society of Intensive Care Medicine, current evidence does not adequately support the use of N-acetylcysteine (NAC) for the prevention of contrast-associated acute kidney injury (CA-AKI). In contrast, the American Thoracic Society recommends NAC supplementation alongside intravenous hydration in high-risk patients, despite limited supporting data.³⁶ A review comparing all treatments for CA-AKI prophylaxis in emergency departments found that theophylline, bicarbonate, and ascorbic acid were appropriate, while NAC treatment was less effective.³⁷ Although standard precautions are taken in our hospital when using contrast agents, the observation time in the emergency department may be insufficient for adequate intravenous hydration. Some patients were admitted to the internal medicine ward without receiving optimal fluid therapy in the emergency setting, which may have contributed to the observed differences.

In our study, the majority of the 14 patients who required hemodialysis (HD) due to contrast-associated acute kidney injury needed temporary HD. 21% of those who developed contrast nephropathy required acute HD, while 6% required permanent HD. In the study by Maioli and colleagues, 14% of the patients required temporary HD after developing contrast nephropathy, and 2% of these patients required permanent HD.⁸

In our study, the mortality rate and the rate of admission to the intensive care unit (ICU) were higher in patients who developed contrast nephropathy. In their study, Rihal et al found that the in-hospital mortality rate reached 22% among patients who developed acute kidney injury following percutaneous coronary intervention, while it remained at 1.4% in those without AKI.²⁰ In our study, the mortality rate in patients who developed CN was 29.2%. The higher mortality rate in our study compared to the literature may be due to the fact that patients presented to the hospital within a certain time frame and had comorbidities.

This study has several limitations. First, its retrospective design and single-center setting may limit the generalizability of the findings. The relatively small sample size may have reduced the statistical power to detect certain associations, and the lack of randomization introduces potential selection bias. Moreover, the absence of long-term follow-up data prevented us from assessing delayed renal outcomes. In the emergency department setting, acute kidney injury (AKI) is often multifactorial, and other clinical conditions such as sepsis, dehydration, hypotension, or the use of nephrotoxic agents may have contributed to renal dysfunction. As clinical adjudication was not performed, some cases of contrast-associated AKI may have been misclassified, potentially leading to an overestimation of the contrast-related risk. Future prospective, multicenter studies with standardized adjudication and more comprehensive clinical data are needed to clarify the independent role of contrast media in AKI development. Additionally, due to the retrospective design of the study, complete and reliable urine output measurements were not available for all patients. Hourly urine output was not routinely recorded for patients in the general internal medicine ward, and in many cases, these data were incomplete or missing. Therefore, the definition of CA-AKI in this study was based solely on changes in serum creatinine, consistent with approaches used in similar retrospective studies.

Though our study did not involve predictive modeling, recent research in other clinical contexts—such as patients undergoing coronary artery bypass surgery or percutaneous coronary interventions—has demonstrated the potential of such models for anticipating adverse outcomes, including acute kidney injury and bleeding events.³⁸ Applying similar predictive approaches to patients undergoing contrast-enhanced imaging in emergency and internal medicine settings could be a valuable avenue for future research. Such models might help identify high-risk individuals earlier and guide targeted preventive strategies, ultimately improving patient outcomes.

Conclusion

The results demonstrate that the diagnosis of HT in patients, and especially exposure to contrast agents in the emergency department, effectively predict the development of contrast-associated acute kidney injury (CA-AKI) and increase the risk by 2.3 times. The higher incidence of CA-AKI among patients undergoing contrast-enhanced imaging in the emergency department may, in part, reflect the presence of concurrent high-risk conditions such as sepsis, volume depletion, use of

nephrotoxic agents, and hemodynamic instability, which were not fully excluded due to the retrospective design. This underscores the importance of comprehensive clinical assessment before contrast administration, particularly in critically ill patients. Moreover, administering intravenous fluids both prior to and following contrast exposure appears to play a protective role against the onset of contrast-associated nephropathy. This preventive effect is particularly evident in patients undergoing contrast-enhanced CT imaging.

Abbreviations

ACEI, Angiotensin-Converting Enzyme Inhibitors; AKI, Acute kidney injury; BUN, Blood urea nitrogen; CA-AKI, Contrast-associated acute kidney injury; CHF, Congestive heart failure; CKD, Chronic kidney disease; CN, Contrast nephropathy; CT, Computerized tomography; DM, Diabetes mellitus; GFR, Glomerular filtration rate; HD, Hemodialysis; HT, Hypertension; ICU, Intensive care unit; LRA, Logistic Regression Analysis; NAC, N-Acetyl Cysteine; NO, Nitric Oxide; NSAIDs, Non-steroidal anti-inflammatory drugs; OR, Odds Ratio; SPSS, Statistical Package for the Social Sciences.

Institutional Review Board Statement

Ethical approval for this retrospective study was obtained from the Institutional Ethics Committee of İzmir Atatürk Education and Research Hospital (protocol code 0213 and date of 21.04.2022). The committee waived the requirement for individual patient consent because the study involved only a retrospective review of existing medical records, posed no additional risk to patients, and did not require any direct patient contact. All data were anonymized before analysis, and patient confidentiality was strictly maintained in accordance with the Declaration of Helsinki.

Informed Consent Statement

Patient consent was waived due to the retrospective nature of the study, in accordance with institutional and national research ethics guidelines.

Data Sharing Statement

The data supporting the findings of this study are not publicly available due to institutional data protection policies and patient confidentiality agreements. However, anonymized data may be made available from the corresponding author upon reasonable request and with permission from İzmir Katip Celebi University Atatürk Education and Research Hospital, in accordance with ethical and privacy guidelines. In accordance with journal policies, authors have ensured that the raw data are properly preserved and are ready to be provided to editors or peer reviewers upon request. Authors have also taken appropriate measures to maintain the complete storage of the raw data for a reasonable period after publication.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The author(s) report no conflicts of interest in this work.

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