

Contrast-Induced Renal Injury After Computed Tomography in Ischemic Stroke Patients Receiving Intravenous Thrombolytic Therapy

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Abstract

Aim: This study aimed to evaluate the incidence of contrast-induced nephropathy (CIN) and the factors influencing its development in patients with acute ischemic stroke who were admitted to the emergency department and received intravenous thrombolytic therapy along with intravenous contrast.

Materials and Methods: This retrospective observational study included acute ischemic stroke patients aged over 18 years who received intravenous thrombolytic therapy at the emergency department of a tertiary care training and research hospital, a major stroke center in its region. The study was carried out between 1 January 2024 and 1 January 2025. All patients underwent contrast-enhanced brain and supraaortic computed tomography angiography, after receiving a standard dose of intravenous contrast. CIN was defined as either an increase of more than 25% increase in baseline serum creatinine levels or an absolute increase of ≥ 0.50 mg/dL within 48-72 hours post-contrast administration.

Results: A total of 194 patients met the inclusion criteria, with a median age of 74 years, half of whom were female. CIN was observed in 14.9% of patients, although none required dialysis. Patients who developed CIN had significantly lower creatinine levels at admission compared to those who did not ($p=0.020$). No other parameters, rates, or scores at admission showed statistically significant differences between the groups.

Conclusion: The incidence of CIN in patients receiving intravenous thrombolysis for acute ischemic stroke was 14.9%. Patients who developed CIN exhibited significantly lower creatinine levels at admission.

Keywords: Acute kidney injury, contrast media, emergency medicine, glomerular filtration rate, nephropathy

Introduction

Ischemic stroke is a leading cause of morbidity and mortality worldwide, making the early detection and treatment of cerebrovascular occlusion critical for minimizing post-stroke disability (1). Current national and international guidelines for acute ischemic stroke recommend several non-invasive intracranial vascular imaging modalities for the initial evaluation of patients in the emergency department (ED) to guide decisions regarding medical or mechanical endovascular treatments (2,3). Among these imaging

techniques, computed tomography angiography (CTA) of the brain and great vessels is one of the most frequently utilized modalities in the acute setting (1). While CTA is generally considered safe and well-tolerated, contrast-induced nephropathy (CIN) remains a potentially serious complication associated with the intravenous (IV) contrast media used in such imaging studies. The incidence of CIN has been reported to be as high as 7% in some studies (4). Additionally, the reported incidence of CIN in acute ischemic stroke varies widely, ranging from 2% to 23% across different clinical contexts (5).



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There are also studies suggesting that CIN alternatively referred to as contrast-associated nephropathy or post-computed tomography (CT) acute kidney injury may have no significant difference in incidence between imaging with contrast and without contrast, and that its relevance is now primarily historical due to advancements in tomography and contrast agent technology, such as the development of lower-osmolality contrast materials (6,7). CIN is typically characterized by a sudden decline in renal function occurring within 48-72 hours of IV contrast administration (5,8). Although there is no universally agreed definition of CIN, the most widely accepted criterion, as proposed by the Kidney Disease Improving Global Outcomes guidelines, defines CIN as either a greater than 25% increase from baseline serum creatinine levels or an absolute increase of ≥ 0.50 mg/dL (9). Various factors, including renal vascular stenosis, direct renal tubular toxicity, decreased renal blood flow, oxidative stress, endothelial dysfunction, and pre-existing chronic kidney disease, have been implicated in the pathogenesis of CIN. However, the precise mechanisms underlying why some patients develop CIN after contrast administration while others do not remain unclear (6,10). Additionally, IV tissue plasminogen activator (t-PA) therapy, commonly used in the treatment of acute ischemic stroke, may contribute to the development of CIN (11).

Our ED and hospital serve as one of the primary centers for IV thrombolysis, neuroendovascular treatment, and neurointensive care for ischemic stroke in our province. It also functions as an on-call stroke center, for emergency medical and interventional endovascular treatment of ischemic stroke, about 7-8 days per month. In this study, our objectives were to evaluate (1) the incidence of CIN development, (2) the factors influencing this incidence, and (3) the impact of baseline creatinine levels, on the development of CIN in patients admitted to our ED with acute ischemic stroke, and treated with IV t-PA therapy.

Materials and Method

Patients and Study Design

This study was designed as a single-center, retrospective, and observational study. Patients who presented to the ED of University of Health Sciences Türkiye Ankara Training and Research Hospital from 1 January 2024 to 1 January 2025 with a preliminary diagnosis of acute ischemic stroke, who received thrombolytic therapy and underwent contrast-enhanced brain and supraaortic CTA, were evaluated.

Patients aged 18 years or older with documented creatinine and estimated glomerular filtration rate (GFR) values at admission and within 48-72 hours post-admission were included in the study. Exclusion criteria were as follows: patients with missing

laboratory data at admission, those on dialysis for chronic renal failure, those with a GFR < 30 mL/min/1.73 m² at admission, patients whose contrast-enhanced tomography was performed at another center, those without measured creatinine levels within 72 hours of admission, and patients transferred to another center within 48 hours. As all eligible patients meeting the inclusion criteria during the specified time period were enrolled, no sample size calculation was performed.

Study Protocol

Demographic data (sex, age), comorbidities (hypertension, diabetes mellitus, coronary artery disease, previous stroke, atrial fibrillation), laboratory parameters (hemoglobin, platelets, glucose, creatinine, urea, aspartate aminotransferase, alanine aminotransferase, international normalized ratio, and lactate levels), creatinine levels at 48 and 72 hours, and Mehran scores were recorded. The Mehran score, originally developed to predict the risk of CIN following percutaneous coronary intervention, is calculated based on variables such as blood pressure, age, intra-aortic balloon pump status, congestive heart failure, anemia, diabetes mellitus, contrast volume, and GFR values (12). It classifies patients into four risk categories: low risk (≤ 5), intermediate risk (6-10), high risk (11-15), and very high risk (16). Laboratory parameters, scores, and other variables (vital signs, comorbidities, etc.) studied for the patients were selected for their similarity to prior studies in the literature, and as much as retrospective data of the study allowed.

The diagnosis of contrast-associated nephropathy was defined as either an increase in serum creatinine level of ≥ 0.5 mg/dL within 72 hours after contrast administration or a $\geq 25\%$ increase in serum creatinine compared with baseline. Patients were classified in terms of CIN based on the difference between their creatinine levels at admission and at 48-72 hours post-contrast administration.

Patients included in this study had a pre-existing diagnosis of acute ischemic stroke and presented to the ED within the first 4.5 hours of symptom onset. At presentation, all patients underwent a non-contrast brain CT, and contrast-enhanced brain and supraaortic CTA, to assess eligibility for thrombolysis or mechanical thrombectomy. Contrast-enhanced studies were performed using a routine and standard injection of 60.4 g iohexol (Biemexol, 350 mg/ml, Biem® ilaç, Ankara) during CTA. The volume of contrast media was administered in standard doses, independent of the weight of the patients.

The present study was conducted in accordance with the principles of the Declaration of Helsinki. The Ethics Committee of Health Science University Türkiye, Ankara Training and Research Hospital approved the study (decision number: E25-396, date: 22.01.2025).

Statistical Analysis

Statistical analysis was conducted using SPSS version 26 (SPSS Inc., Chicago, Illinois, USA) and Jamovi version 2.6.2.0. The normality of data distribution was evaluated using histograms, Q-Q plots, and the Shapiro-Wilk test. Descriptive statistics were presented as mean ± standard deviation or median with interquartile range (IQR) for continuous variables, and as frequencies and percentages for categorical variables. Comparisons between paired groups were performed using the Mann-Whitney U test for continuous variables and the chi-Squared test for categorical variables. A p value of less than 0.05 was considered statistically significant.

Results

A total of 253 patients with acute ischemic stroke were admitted to the ED, underwent contrast-enhanced CT, and received thrombolytic therapy. Of these, 194 patients met the inclusion criteria and were included in the study (Figure 1). Among the included patients, 97 (50%) were male, and the median age was 74 years. The median creatinine level at admission was 0.88 mg/dL (IQR: 0.73-1.06), and the median creatinine level at 48-72 hours post-admission was 0.90 mg/dL (IQR: 0.74-1.17). CIN developed in 29 (14.9%) of the patients. Demographic and laboratory data for all patients are presented in Table 1.

Patients were divided into two groups based on the development of CIN. Demographic characteristics, laboratory results, and comorbidities were compared between the two groups (Table 2). In pairwise comparisons, the median creatinine level at presentation was 0.80 mg/dL (IQR: 0.70-0.94) in patients who developed CIN, compared to 0.89 mg/dL (IQR: 0.74-1.10)

in patients who did not develop CIN ($p=0.020$). The median creatinine level at 48-72 hours was 1.85 mg/dL (IQR: 1.59-2.42) in patients with CIN, compared to 0.86 mg/dL (IQR: 0.72-0.99) in patients without CIN ($p<0.001$).

No significant difference was observed in the median Mehran score, or between the subgroups based on Mehran scores in the groups with and without CIN ($p=0.050$). Additionally, there were no statistically significant differences between the two groups for any other parameters examined.

Table 1. The demographic characteristics of patient population

| Characteristics | Total n=194 |
|---|---------------------|
| Age, median (IQR) | 74 (64-81) |
| Gender, male, n (%) | 97 (50) |
| Systolic blood pressure, (mmHg), (IQR) | 160 (140-186) |
| Diastolic blood pressure, (mmHg), (IQR) | 85 (75-94) |
| Heart rate (/min) (IQR) | 79 (69-87) |
| Laboratory parameters, median (IQR) | |
| Hemoglobin (g/dL) | 13.1 (11.8-14.6) |
| Platelets ($\times 10^3/L$) | 219 (171-273) |
| INR | 1.05 (1.01-1.13) |
| Glucose (mg/dL) | 127 (108-170) |
| Admission creatinine (mg/dL) | 0.88 (0.73-1.06) |
| Creatinine at 48-72 hours (mg/dL) | 0.90 (0.74-1.17) |
| Admission GFR (mL/min) | 75.50 (59.25-90.75) |
| Urea (mg/dL) | 36 (28-47) |
| AST (U/L) | 19 (15-23) |
| ALT (U/L) | 13 (10-19) |
| Lactate (mmol/L) | 2.1 (1.6-2.7) |
| MEHRAN score, median (IQR) | |
| ≤5, n (%) | 82 (42.3) |
| 6-10, n (%) | 53 (27.3) |
| 11-15, n (%) | 50 (25.8) |
| ≥16, n (%) | 9 (4.6) |
| Comorbidities, n (%) | |
| Diabetes mellitus, n (%) | 97 (50) |
| Hypertension, n (%) | 148 (76.3) |
| Coronary artery disease, n (%) | 74 (38.1) |
| Atrial fibrillation, n (%) | 44 (22.7) |
| Previous stroke, n (%) | 52 (26.8) |
| Contrast induced nephropathy, n (%) | 29 (14.9) |
| IQR: Inter quantile range, INR: International normalized ratio, AST: Aspartate aminotransferase, ALT: Alanine transaminase, GFR: Glomerular filtration rate | |

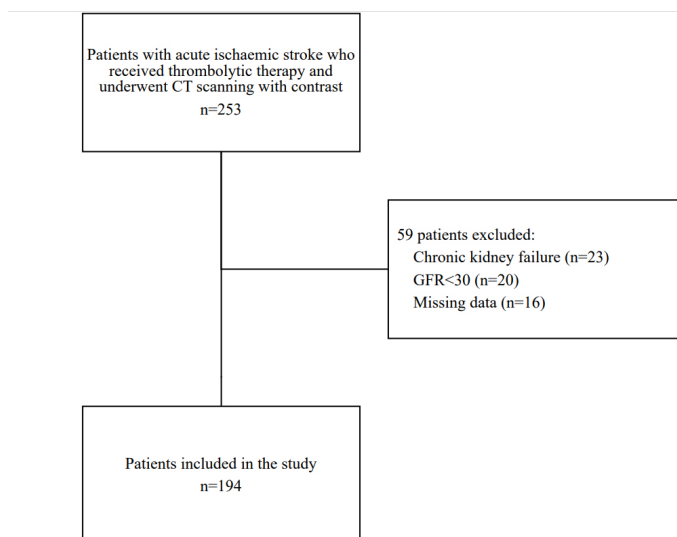


Figure 1. Patient flow chart

CT: Contrast-induced nephropathy, GFR: Glomerular filtration rate

Discussion

Contrast-enhanced CT plays a crucial role in the accurate and effective diagnosis of vascular emergencies, including aortic dissection, pulmonary embolism, and ischemic stroke, particularly in ED settings. The use of contrast media in CT has increased over the years (13,14). However, as the administration of contrast agents is associated with potential adverse events such as CIN, some centers may adopt policies that recommend waiting for baseline creatinine levels in acute ischemic stroke patients, or, if this is not feasible, opting for alternative imaging modalities such as ultrasound

(15). Nonetheless, in the context of ischemic stroke, waiting for these blood tests can have significant negative consequences on patient outcomes, particularly in terms of delaying treatment and impairing neurological recovery (16). Given that time is a critical factor in stroke management, emergency physicians often proceed with contrast-enhanced CT without waiting for certain routine laboratory results, provided the potential benefits outweigh the risks. This approach, while essential for timely diagnosis and intervention, also makes the ED one of the highest-risk areas in the hospital for the development of CIN (5).

Table 2. Comparison of demographic characteristics, laboratory parameters, comorbidities, and Mehran scores of patients with and without CIN

| Characteristics | CIN present n=29 | CIN absent n=165 | p value |
|--|---------------------|---------------------|------------------|
| Age, median (IQR) | 78 (69-83) | 73 (64-81) | 0.100 |
| Gender, male, n (%) | 13 (44.8) | 84 (50.9) | 0.546 |
| Systolic blood pressure, (mmHg), (IQR) | 145 (138-180) | 160 (140-187) | 0.292 |
| Diastolic blood pressure, (mmHg), (IQR) | 79 (75-90) | 85 (77-94) | 0.079 |
| Heart rate(/min) (IQR) | 75 (68-80) | 79 (69-88) | 0.127 |
| Laboratory parameters, median (IQR) | | | |
| Hemoglobin (g/dL) | 12.6 (10.3-13.8) | 13.2 (12-14.7) | 0.099 |
| Platelets (x10 ³ /L) | 211 (168-278) | 221 (174-271) | 0.979 |
| INR | 1.08 (0.98-1.14) | 1.05 (1.01-1.11) | 0.639 |
| Glucose (mg/dL) | 139 (118-189) | 126 (106-163) | 0.186 |
| Admission creatinine (mg/dL) | 0.80 (0.7-0.94) | 0.89 (0.74-1.1) | 0.020 |
| Creatinine at 48-72 hours (mg/dL) | 1.85 (1.59-2.42) | 0.86 (0.72-0.99) | <0.001 |
| Admission GFR (mL/min) | 77 (68-91) | 75 (57-90) | 0.201 |
| Urea (mg/dL) | 34 (26-44) | 37 (28-47) | 0.230 |
| AST(U/L) | 17 (15-21) | 19 (15-24) | 0.267 |
| ALT(U/L) | 12 (10-16) | 13 (9-19) | 0.442 |
| Lactate (mmol/L) | 1.7 (1.5-2.5) | 2.1 (1.6-2.7) | 0.148 |
| MEHRAN score, median (IQR) | 7 (4-12) | 7 (3-11) | 0.268 |
| ≤5, n (%) | 11 (37.9) | 71 (43) | 0.050 |
| 6-10, n (%) | 5 (17.2) | 48 (29.1) | |
| 11-15, n (%) | 13 (44.8) | 37 (22.4) | |
| ≥16, n (%) | 0 (0) | 9 (5.5) | |
| Comorbidities, n (%) | | | |
| Diabetes mellitus, n (%) | 12 (41.4) | 85 (51.5) | 0.314 |
| Hypertension, n (%) | 20 (69) | 128 (77.6) | 0.315 |
| Coronary artery disease, n (%) | 15 (51.7) | 59 (35.8) | 0.103 |
| Atrial fibrillation, n (%) | 6 (20.7) | 38 (23) | 0.781 |
| Previous stroke, n (%) | 10 (34.5) | 42 (25.5) | 0.311 |

IQR: Inter quantile range, INR: International normalized ratio, AST: Aspartate aminotransferase, ALT: Alanine transaminase, CIN: Contrast-induced nephropathy

Studies examining this high-risk situation in EDs report varying rates of CIN for different clinical conditions. For instance, a study by Turedi et al. (17) involving 257 patients with suspected pulmonary embolism, all of whom received prophylactic measures before and after contrast-enhanced CT, found CIN in nearly a quarter of the patient population. Similarly, a South African study on multi-trauma patients reported a high incidence of CIN (14.7%) following contrast-enhanced CT (18). In contrast, a study of trauma patients over the age of 55 found the incidence of CIN to be only 1.9% in the contrast group (19), while a study from our country reported a higher rate of 4.9% in patients with undifferentiated diagnoses who underwent contrast-enhanced CT for various reasons in emergency department (20). In addition to these varying rates, the literature presents differing perspectives on the clinical significance of CIN. Some studies (6,7) regard CIN as a “historical reality” that is now considered less clinically relevant due to advancements in contrast media and CT technology, and thus, recommend routine contrast-enhanced CT scans. Conversely, other studies (5,11,21) continue to view CIN as a high-incidence complication and urge caution in its management, particularly in EDs. In our study, we aimed to contribute to this ongoing debate by evaluating a cohort of patients in our ED who shared similar pathology, received uniform treatment, and underwent comparable physiological changes. Our findings reveal a CIN incidence of 15% among patients with acute ischemic stroke who received IV thrombolytic therapy, suggesting a significant concern for this patient population. In our study, the incidence of CIN was found to be as high as 15% in patients with acute ischemic stroke who received IV thrombolysis. However, it is noteworthy that none of our patients required temporary or permanent dialysis. This finding aligns with other studies (1,22) that prioritize neurovascular function and patient benefit over concerns about CIN in ischemic stroke patients receiving t-PA. When comparing the incidence of CIN in our study with other stroke-related literature, we find that it is significantly higher than some studies (6,7,8,23), slightly higher than others (11,22), but similar to very few (1). This disparity may be attributed to the heterogeneity of patient populations in other studies, which included patients with ischemic stroke, hemorrhagic stroke, and intracerebral hemorrhage, as well as variations in treatment approaches (mechanical thrombectomy, intravenous t-PA, and other anticoagulant therapies). In contrast, our study focused exclusively on ischemic stroke patients treated with intravenous t-PA and patients with bleeding were excluded. This made our study cohort more homogeneous, with similar pathology, identical treatment regimens, and comparable amounts of IV contrast administered, all triggered by similar physiological stimuli, while excluding other complicating factors such as vascular injury or embolism. We believe this homogeneity is a

significant strength of our study. In our review of the literature, we found no other studies investigating the development of CIN specifically in ischemic stroke patients receiving intravenous t-PA alone. Moreover, the relatively older age (median = 74 years) and higher comorbidity (at least 50% of the cohort had one or more comorbidities) compared to other studies, as well as the fact that all patients received intravenous t-PA, may have contributed to the higher incidence of CIN observed. In fact, one study has suggested that receiving intravenous t-PA in acute stroke is associated with a seven-fold increased risk of CIN (8).

Several studies have identified various factors that increase the risk of developing CIN, including an estimated GFR <30 mL/min (6,8,22), diabetes mellitus (1), hypertension (8), chronic kidney disease (6,22), and higher volumes of contrast (23). Other risk factors include elderly patients, those using non-steroidal anti-inflammatory drugs after contrast administration, and patients on angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, beta-blockers, statins, or insulin (22). Smoking (1) and intravenous t-PA therapy (8) have also been associated with an increased risk of CIN. In our study, patients with an initial GFR <30 mL/min were excluded, as they were deemed at high risk of developing severe chronic kidney disease (stage V). Although some studies suggest that CIN is clinically significant only in this high-risk population and report that CIN is less important in patients with a GFR >60 mL/min (6), the literature remains divided on this matter. In our cohort, no statistically significant differences were observed in the risk of CIN in relation to comorbidities or vital signs at presentation. The retrospective nature of our study, the loss of data, and the urgency of acute ischemic stroke management in patients receiving IV thrombolytics within the treatment window limited our ability to access detailed home medication lists during the stable period. Consequently, we were unable to evaluate the impact of specific medications on CIN development, which we consider a limitation of our study. Additionally, all patients in our study received intravenous t-PA, and were administered a standard dose of 60.4 g of non-ionic low-osmolality contrast material during CTA. Surprisingly, CIN did not develop in our patients with high Mehran scores. This may be due to two reasons: our patients with high Mehran scores are very few in number (9 patients), and this number may not be reflected in a statistical significance. Additionally, the score itself is more related to cardiological interventional contrast studies and higher contrast volumes (12,24). In our study, patients received 80 cc (60.4 g) of IV contrast material as a standard dose.

Several studies have identified potential independent predictors for the development of CIN, including total white blood cell count (23), the C-reactive protein/albumin ratio (10,21), gamma-glutamyl transferase (25), and erythrocyte distribution width

(26). However, in our patient cohort, none of the biochemical parameters, ratios, or scores evaluated at the time of presentation for ED were found to differ significantly between patients with and without CIN. Interestingly, only the creatinine level at presentation was significantly lower in patients who developed CIN ($p=0.020$). This finding is somewhat surprising, as the general literature suggests that creatinine levels at presentation are typically higher in patients who develop CIN (1,5,6). This discrepancy may be attributed to the more homogeneous nature of our patient cohort, with similar pathology, treatment protocols, and physiological responses. Furthermore, as all of our patients were monitored in intensive care following acute ischemic stroke treatment, we did not investigate the potential relationship between the development of CIN and intensive care unit admission, which could be another limitation of the study.

Study Limitations

The primary limitations of this study include its single-center, retrospective design and the relatively small sample size, which may limit the generalizability of the findings. Additionally, the inability to include patient medication data in the analysis represents another limitation, considering that routine medications play a crucial role in renal function. This is particularly relevant given the elderly nature of the study population, where comorbidities and polypharmacy are common. These factors could influence the development of CIN and warrant further investigation in future studies.

Conclusions

Our study found that the incidence of CIN in patients with acute ischemic stroke receiving intravenous t-PA treatment was 14.9% (29/194). Notably, none of the CIN patients required temporary or permanent hemodialysis. The median age of our patient cohort was relatively high, and many had multiple comorbidities. A statistically significant difference was observed between the groups with and without CIN in creatinine levels, measured at presentation with the CIN group showing lower creatinine levels. However, no significant differences were found between the groups with regard to any other parameter, rate, or score at presentation.

Ethics

Ethics Committee Approval: The present study was conducted in accordance with the principles of the Declaration of Helsinki. The Ethics Committee of Health Science University Türkiye, Ankara Training and Research Hospital approved the study (decision number: E25-396, date: 22.01.2025)

Informed Consent: This study was designed as a single-center, retrospective, and observational study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: D.Ü.K., C.U.T., Concept: D.Ü.K., S.G., S.C.Y., Design: D.Ü.K., C.U.T., Data Collection or Processing: S.G., S.C.Y., C.U.T, Analysis or Interpretation: S.G., S.C.Y., C.U.T, Literature Search: D.Ü.K., S.G., S.C.Y., Writing: D.Ü.K., S.G.

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