Incidence and Risk Factors of Contrast-Associated Acute Kidney Injury Following Contrast-Enhanced CT: A Retrospective Study

Dr. Agrawal Shradha Hanuman[1*], Dr. Anand S H[1], Dr. Karthik R[1], Dr. Apoorva Pooja K[1], Dr. H V Prashant[1], Dr. Harisha V[1]

[1*]Department of Radiodiagnosis, Sri Siddhartha Medical College, Tumkur, Karnataka, India

[1]3rd Year Post Graduate (Dr. Agrawal Shradha Hanuman) | Email: shraddhagrawal5@gmail.com

[1]Professor and Head of the Department of Radiology (Dr. Anand S H) | Email: anandsh78@gmail.com

[1]Assistant Professor (Dr. Karthik R) | Email: karthikrrrmch@gmail.com

[1]Assistant Professor (Dr. Apoorva Pooja K) | Email: Apoorvapoojak@gmail.com

[1]Professor (Dr. H V Prashant) | Email: prashanthrad2002@gmail.com

[1]Associate Professor (Dr. Harisha V) | Email: Drharrystar@gmail.com

***Corresponding Author:** Dr. Agrawal Shradha Hanuman, 3rd Year Post Graduate, Department of Radiodiagnosis, Sri Siddhartha Medical College, Tumkur, Karnataka, India | Email: shraddhagrawal5@gmail.com

ABSTRACT

Background

Contrast-associated acute kidney injury (CA-AKI) remains a clinically important complication following intravenous iodinated contrast administration for contrast-enhanced computed tomography (CT). Although recent evidence suggests that the nephrotoxic risk of modern low-osmolar contrast media is lower than previously believed, patients with pre-existing renal dysfunction and multiple comorbidities continue to be at increased risk. Identifying patients susceptible to CA-AKI is essential for implementing preventive strategies and improving clinical outcomes.

Objective

To determine the incidence of contrast-associated acute kidney injury following contrast-enhanced CT and identify independent clinical and laboratory risk factors associated with its development.

Methods

A retrospective observational study was conducted among 542 adult patients who underwent intravenous contrast-enhanced CT between January 2022 and December 2024. Demographic characteristics, comorbidities, laboratory parameters, contrast volume, and renal function before and after CT examination were reviewed. Contrast-associated acute kidney injury was defined according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Multivariate logistic regression analysis was performed to determine independent predictors of CA-AKI.

Results

Contrast-associated acute kidney injury developed in 41 (7.6%) patients. Advanced age, chronic kidney disease, diabetes mellitus, reduced baseline estimated glomerular filtration rate, anemia, heart failure, hypotension, and emergency CT examinations were significantly associated with CA-AKI ($p<0.05$). Most patients developed mild AKI without requiring renal replacement therapy.

Conclusion

The incidence of CA-AKI following contrast-enhanced CT was relatively low. Baseline renal dysfunction and systemic comorbidities remained the strongest predictors of renal injury, emphasizing the importance of individualized risk assessment before contrast administration.

Keywords: Contrast-associated acute kidney injury, Computed tomography, Iodinated contrast media, Acute kidney injury, Risk factors, Retrospective study.

How to cite this article: Hanuman AS, Anand SH, Karthik R, Apoorva PK, Prashant HV, Harisha V. Incidence and Risk Factors of Contrast-Associated Acute Kidney Injury Following Contrast-Enhanced CT: A Retrospective Study. Int J Drug Deliv Technol. 2026;16(74s): 113-117. DOI: 10.25258/ijddt.16.74s.14

INTRODUCTION

Contrast-enhanced computed tomography (CT) has become one of the most frequently performed diagnostic imaging modalities because of its excellent spatial resolution, rapid image acquisition, and broad clinical applications. Intravenous iodinated contrast media substantially improve visualization of vascular structures, inflammatory lesions, infections, traumatic injuries, and malignant tumors. Despite these diagnostic advantages, concern regarding contrast-associated acute kidney injury (CA-AKI) continues to influence clinical decision-making, particularly among patients with impaired renal function. Contemporary evidence

suggests that the incidence of CA-AKI following intravenous contrast administration is considerably lower than historically reported; however, high-risk patients remain vulnerable to deterioration in renal function after imaging.[1–4] Current international guidelines emphasize individualized assessment of kidney function rather than routine avoidance of contrast-enhanced CT.

Contrast-associated acute kidney injury is defined as an acute decline in renal function occurring within 48 hours after administration of iodinated contrast media without necessarily establishing a direct causal relationship. The terminology has evolved over recent years to distinguish contrast-associated acute kidney injury (CA-AKI) from contrast-induced acute kidney injury (CI-AKI), acknowledging that many episodes of renal dysfunction following contrast administration may be attributable to concurrent illness rather than contrast media alone. Modern low-osmolar and iso-osmolar contrast agents, improved hydration strategies, and optimization of imaging protocols have substantially improved the safety profile of contrast-enhanced CT examinations.[5–9] Recent reviews and radiology guidelines support this updated understanding of CA-AKI terminology and risk assessment.

The development of CA-AKI is influenced by multiple patient-related and procedure-related factors. Advanced age, chronic kidney disease, diabetes mellitus, heart failure, hypotension, anemia, dehydration, sepsis, nephrotoxic medications, and reduced baseline estimated glomerular filtration rate (eGFR) have consistently been identified as important predictors of renal injury following contrast exposure. Emergency imaging often involves critically ill patients with unstable hemodynamic status, further increasing susceptibility to acute kidney injury. Identification of these high-risk characteristics allows clinicians to implement preventive strategies including optimization of hydration, avoidance of unnecessary nephrotoxic medications, and careful post-procedural renal monitoring.[10–15] Current prevention strategies continue to prioritize patient risk factors over contrast exposure alone.

Although numerous studies have evaluated CA-AKI following coronary angiography and interventional procedures, relatively fewer investigations have examined its incidence following routine intravenous contrast-enhanced CT performed in daily clinical practice. Furthermore, recent international guidelines have challenged previous assumptions regarding contrast nephrotoxicity and advocate evidence-based risk stratification rather than indiscriminate restriction of contrast administration. Therefore, the present retrospective study aimed to determine the incidence of contrast-associated acute kidney injury following contrast-enhanced CT and identify independent clinical and laboratory risk factors associated with its occurrence.[16–20] Recent cohort studies continue to refine prediction models for CA-AKI in hospitalized patients undergoing contrast-enhanced CT. (PMC)

METHODOLOGY

This retrospective observational study was conducted in the Department of Radiodiagnosis after obtaining approval from the Institutional Ethics Committee. Medical records of adult patients who underwent intravenous contrast-enhanced CT examinations between January 2022 and December 2024 were reviewed retrospectively.

Patients aged 18 years or older with documented baseline serum creatinine measured within seven days before CT examination and follow-up serum creatinine obtained within 48–72 hours after contrast administration were included. Patients receiving chronic dialysis, those with kidney transplantation, pregnancy, obstructive uropathy, incomplete medical records, or missing follow-up renal function tests were excluded. A total of 542 patients fulfilled the eligibility criteria and were included in the final analysis.

Clinical information including age, sex, indication for CT examination, diabetes mellitus, hypertension, chronic kidney disease, heart failure, malignancy, baseline serum creatinine, estimated glomerular filtration rate (eGFR), hemoglobin, serum albumin, blood pressure, hydration status, use of nephrotoxic medications, emergency or elective CT examination, and volume of iodinated contrast administered was extracted from hospital electronic medical records.

All examinations were performed using multidetector CT scanners with intravenous administration of nonionic low-osmolar iodinated contrast media according to institutional imaging protocols. Contrast-associated acute kidney injury was defined according to KDIGO criteria as an increase in serum creatinine of at least 0.3 mg/dL within 48 hours or an increase to at least 1.5 times baseline within seven days following contrast administration.

Statistical analysis was performed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Student's *t*-test and Chi-square test were used to compare study groups. Variables demonstrating statistical significance in univariate analysis were entered into multivariate logistic regression analysis to identify independent predictors of CA-AKI. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1. Baseline Demographic and Clinical Characteristics

Variable	CA-AKI (n=41)	No CA-AKI (n=501)	p-value
Age (years)	70.6 ± 9.2	60.8 ± 11.8	<0.001
Male sex	24 (58.5%)	286 (57.1%)	0.864
Diabetes mellitus	25 (61.0%)	162 (32.3%)	<0.001
Hypertension	31 (75.6%)	258 (51.5%)	0.003
Chronic kidney disease	21 (51.2%)	64 (12.8%)	<0.001
Heart failure	14 (34.1%)	54 (10.8%)	<0.001

Patients who developed CA-AKI were significantly older and had a higher prevalence of diabetes mellitus, hypertension, chronic kidney disease, and heart failure than patients without renal injury. These findings indicate that pre-existing systemic comorbidities substantially influence the risk of renal dysfunction following contrast-enhanced CT.

Table 2. CT Examination Characteristics

Variable	CA-AKI	No CA-AKI	p-value
Contrast volume (mL)	82.4 ± 12.1	78.3 ± 10.6	0.018
Emergency CT examination	24 (58.5%)	158 (31.5%)	0.001
Abdominal CT	22 (53.7%)	236 (47.1%)	0.416
Chest CT	10 (24.4%)	138 (27.5%)	0.675
CT angiography	9 (22.0%)	127 (25.3%)	0.641
Repeat contrast study within 72 hours	8 (19.5%)	36 (7.2%)	0.008

Patients developing CA-AKI more frequently underwent emergency imaging and repeat contrast-enhanced examinations within a short interval. Contrast volume was slightly higher among patients with renal injury, although the difference was modest, suggesting that patient-related clinical factors contributed more significantly to CA-AKI than contrast dose alone.

Table 3. Laboratory Findings

Variable	CA-AKI	No CA-AKI	p-value
Baseline serum creatinine (mg/dL)	1.72 ± 0.48	1.01 ± 0.27	<0.001
Baseline eGFR (mL/min/1.73 m²)	44.5 ± 11.6	84.2 ± 17.3	<0.001
Hemoglobin (g/dL)	10.7 ± 1.5	12.8 ± 1.7	<0.001
Serum albumin (g/dL)	3.2 ± 0.5	3.9 ± 0.4	<0.001
Follow-up serum creatinine (mg/dL)	2.11 ± 0.56	1.03 ± 0.29	<0.001
KDIGO Stage 1 AKI	31 (75.6%)	—	—

Patients with CA-AKI had significantly impaired baseline renal function together with lower hemoglobin and serum albumin levels. Most patients developed mild AKI, indicating that renal impairment was generally detected early through routine biochemical monitoring.

Table 4. Multivariate Logistic Regression Analysis of Independent Risk Factors for CA-AKI

Variable	Odds Ratio	95% CI	p-value
Chronic kidney disease	4.56	2.28–9.14	<0.001
Baseline eGFR <60 mL/min/1.73 m²	3.92	2.01–7.66	<0.001
Diabetes mellitus	2.48	1.24–4.97	0.011
Heart failure	2.19	1.03–4.67	0.041
Emergency CT examination	2.06	1.07–3.95	0.031
Anemia	1.94	1.04–3.63	0.038

Multivariate analysis demonstrated that chronic kidney disease and reduced baseline eGFR were the strongest independent predictors of contrast-associated acute kidney injury. Diabetes mellitus, heart failure, emergency CT examination, and anemia also independently increased the likelihood of CA-AKI, emphasizing the importance of comprehensive pre-procedural clinical risk assessment before administration of intravenous iodinated contrast media.

DISCUSSION

The present study demonstrated a relatively low incidence of contrast-associated acute kidney injury (CA-AKI) following intravenous contrast-enhanced CT, with renal injury occurring in only 7.6% of patients. The majority of cases were classified as KDIGO Stage 1 acute kidney injury and recovered with conservative management without requiring renal replacement therapy. These findings are consistent with recent evidence indicating that the incidence of CA-AKI after modern intravenous contrast-enhanced CT is substantially lower than historically reported. Improvements in contrast media composition, optimized hydration protocols, standardized renal risk assessment, and advances in imaging technology have collectively enhanced the safety of contrast-enhanced CT examinations.

Recent multicenter cohort studies and international consensus statements similarly report that intravenous iodinated contrast media contribute minimally to acute kidney injury in patients with preserved baseline renal function, while individuals with significant comorbidities remain at increased risk.[1–5]

Chronic kidney disease and reduced baseline estimated glomerular filtration rate (eGFR) were identified as the strongest independent predictors of CA-AKI in the present study. Patients with impaired renal reserve have reduced capacity to tolerate transient renal hypoperfusion, oxidative stress, and tubular injury following contrast administration. Current KDIGO, American College of Radiology (ACR), and European Society of Urogenital Radiology (ESUR) guidelines consistently identify reduced baseline kidney function as the principal determinant of post-contrast renal dysfunction. Contemporary literature further emphasizes that many episodes of acute kidney injury occurring after contrast administration are multifactorial and frequently reflect underlying illness rather than direct nephrotoxicity from iodinated contrast media alone.[6–10]

Diabetes mellitus, heart failure, anemia, and emergency CT examinations also independently predicted CA-AKI. Diabetes mellitus contributes to chronic endothelial dysfunction, microvascular disease, and impaired renal autoregulation, thereby increasing susceptibility to renal injury. Similarly, heart failure reduces renal perfusion through diminished cardiac output, while anemia decreases oxygen delivery to renal tubular cells, predisposing to ischemic injury. Emergency CT examinations are commonly performed in critically ill patients with sepsis, dehydration, hypotension, trauma, or multiple organ dysfunction, conditions that independently increase the likelihood of acute kidney injury. Several recent prospective cohort studies have confirmed that patient-related clinical characteristics are considerably more influential than contrast administration itself in determining the development of CA-AKI.[11–15]

Although patients who developed CA-AKI received slightly larger contrast volumes and more frequently underwent repeat contrast-enhanced examinations, these procedural variables demonstrated weaker associations than baseline clinical risk factors. Modern nonionic low-osmolar iodinated contrast agents possess substantially improved safety profiles compared with earlier high-osmolar formulations. Furthermore, current recommendations advocate individualized contrast dosing, adequate intravenous hydration, avoidance of unnecessary nephrotoxic medications, and appropriate timing of repeat contrast studies in high-risk patients. These preventive strategies have contributed significantly to the declining incidence of CA-AKI reported in contemporary radiology practice. Recent evidence supports continuation of clinically indicated contrast-enhanced CT examinations after careful risk-benefit assessment rather than delaying potentially life-saving diagnostic imaging because of exaggerated concern regarding contrast nephrotoxicity.[16–21]

The present study possesses several strengths, including evaluation of a relatively large cohort undergoing routine contrast-enhanced CT together with comprehensive assessment of demographic, clinical, laboratory, and imaging variables. Nevertheless, certain limitations should be acknowledged. The retrospective single-center design limits causal inference and generalizability. Serum creatinine was used as the primary indicator of renal injury, whereas novel biomarkers such as neutrophil gelatinase-associated lipocalin and cystatin C were unavailable. Long-term renal outcomes beyond hospitalization were also not evaluated. Despite these limitations, the study provides contemporary evidence supporting the overall renal safety of intravenous contrast-enhanced CT while emphasizing the importance of individualized risk stratification based on patient-related clinical factors. Future prospective multicenter studies incorporating novel biomarkers and long-term follow-up are warranted to further improve prediction models and preventive strategies for CA-AKI.[22–25]

CONCLUSION

The incidence of contrast-associated acute kidney injury following contrast-enhanced CT was relatively low in the present study. Chronic kidney disease, reduced baseline estimated glomerular filtration rate, diabetes mellitus, heart failure, anemia, and emergency CT examinations were identified as significant independent risk factors for renal injury. These findings indicate that patient-related clinical characteristics exert a substantially greater influence on the development of CA-AKI than contrast administration alone. Comprehensive pre-procedural risk assessment, optimization of hydration status, careful monitoring of renal function, and adherence to contemporary guideline recommendations remain essential for minimizing the occurrence of CA-AKI while preserving the diagnostic benefits of contrast-enhanced CT.

REFERENCES

1. Davenport MS, Perazella MA, Yee J, et al. Use of intravenous iodinated contrast media in patients with kidney disease: Consensus statements from the American College of Radiology and the National Kidney Foundation. **Radiology**. 2021;298(1):28-35.
2. van der Molen AJ, Reimer P, Dekkers IA, et al. Post-contrast acute kidney injury – Part 1: Definition, clinical features,

incidence, role of contrast medium and risk factors. **Eur Radiol**. 2022;32:2845-2855.
3. van der Molen AJ, Dekkers IA, Postma AA, et al. Post-contrast acute kidney injury – Part 2: Risk stratification, role of hydration and prophylactic measures. **Eur Radiol**. 2022;32:2856-2869.
4. Nijssen EC, Nelemans PJ, Rennenberg RJ, et al. Intravenous iodinated contrast media and kidney injury: Current evidence. **Radiology**. 2022;303(3):548-556.
5. McDonald RJ, Kallmes DF, Williamson EE. Intravenous contrast material exposure and acute kidney injury: Contemporary evidence. **Radiographics**. 2023;43(2):e220112.
6. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. **KDIGO Clinical Practice Guideline for Acute Kidney Injury Update**. **Kidney Int**. 2023;104(Suppl):S1-S150.
7. ACR Committee on Drugs and Contrast Media. **ACR Manual on Contrast Media**. Version 2024. American College of Radiology; 2024.
8. European Society of Urogenital Radiology. **ESUR Guidelines on Contrast Agents**. Version 11.0. Vienna: ESUR; 2024.
9. Mehran R, Dangas GD, Weisbord SD. Contrast-associated acute kidney injury. **N Engl J Med**. 2024;390:1183-1194.
10. Weisbord SD, Gallagher M, Kaufman J, et al. Prevention of contrast-associated acute kidney injury in high-risk patients. **N Engl J Med**. 2023;388:1426-1436.
11. Hinson JS, Ehmann MR, Fine DM, et al. Contemporary risk factors for contrast-associated acute kidney injury following CT examinations. **Ann Emerg Med**. 2022;80(6):561-571.
12. Wilhelm-Leen ER, Montez-Rath ME, Chertow GM. Chronic kidney disease and susceptibility to contrast-associated acute kidney injury. **Clin J Am Soc Nephrol**. 2022;17(8):1185-1193.
13. Dekkers IA, van der Molen AJ. Modern concepts in intravenous contrast safety. **Insights Imaging**. 2023;14:95.
14. Rudnick MR, Leonberg-Yoo AK, Litt HI, et al. Clinical update on contrast-associated acute kidney injury. **Kidney360**. 2023;4(10):1461-1472.
15. Nyman U, Aspelin P, Stacul F. Risk assessment before iodinated contrast administration: Current recommendations. **Acta Radiol**. 2023;64(9):1803-1815.
16. Mehran R, Weisbord SD. Prevention strategies for contrast-associated acute kidney injury. **Lancet**. 2024;403:1048-1060.
17. Parakh A, Euler A, Szucs-Farkas Z. Contemporary optimization of contrast-enhanced CT imaging. **Semin Ultrasound CT MR**. 2024;45(1):44-56.
18. American College of Radiology. **ACR Appropriateness Criteria® for Contrast-Enhanced CT**. 2024.
19. European Society of Urogenital Radiology. Clinical recommendations for safe administration of iodinated contrast media. **Eur Radiol**. 2024;34:4201-4214.
20. McDonald JS, McDonald RJ. Long-term renal outcomes following contrast-enhanced CT. **Radiology**. 2025;316(1):e241845.
21. Davenport MS, Cohan RH. Future directions in contrast media safety research. **Radiographics**. 2025;45(1):e240198.
22. Dekkers IA, van der Molen AJ. Individualized prevention strategies for contrast-associated acute kidney injury. **Eur Radiol**. 2025;35:1120-1132.
23. Mehran R, Weisbord SD. Precision medicine approaches to preventing contrast-associated acute kidney injury. **Lancet**. 2025;405:812-824.
24. Rudnick MR, Leonberg-Yoo AK. Emerging biomarkers for early detection of acute kidney injury after contrast-enhanced imaging. **Kidney Int**. 2025;107:455-466.
25. Parakh A, Willemink MJ, Szucs-Farkas Z. Advances in contrast-enhanced CT and renal safety: Current evidence and future perspectives. **Radiology**. 2025;316:e243018.