Intravenous Contrast Causes Nephropathy

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KEYWORDS

• CIN • Contrast • Contrast-induced nephropathy • Kidney injury • Kidney disease • AKI • CKD • GFR

KEY POINTS

- The long-standing belief that intravenous (IV) contrast causes significant kidney injury (contrast-induced nephropathy [CIN]) is unsupported by modern evidence; most studies linking contrast to acute kidney injury are flawed or inconclusive, and current data show minimal causal relationship.
- Early studies suggesting CIN were limited by small sample sizes, lack of controls, and outdated contrast agents, leading to persistent misconceptions despite evolving evidence.
- Recent large-scale, well-controlled studies indicate that IV contrast, especially with low-osmolar and iso-osmolar agents, is safe for most patients, including those in emergency settings, when used judiciously.
- Organizations like KDIGO and the American College of Radiology now recommend risk-based assessment using estimated glomerular filtration rate, discouraging routine preimaging renal testing in low-risk patients and emphasizing timely diagnosis over fear of contrast.
- Overestimating contrast nephrotoxicity leads to unnecessary delays and suboptimal care, risking missed or delayed diagnoses. Reframing contrast as a diagnostic tool rather than a toxin improves patient outcomes and aligns practice with current evidence.

INTRODUCTION: FRAMING THE MYTH

The concern that intravenous (IV) contrast may precipitate acute kidney injury (AKI)—has become deeply entrenched in clinical culture. This long-standing belief has shaped both clinician behavior and hospital policy, contributing to routine preimaging creatinine screening and protocol-related delays in obtaining contrast-enhanced studies.[1,2]

The origin of this belief can be traced to 1954, when the first report of kidney injury following IV pyelography was described.[3] Additional literature reinforced the

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Abbreviations	
ACR	American College of Radiology
AKI	acute kidney injury
CA-AKI	contrast-associated AKI
CI-AKI	contrast-induced acute kidney injury
CIN	contrast-induced nephropathy
CKD	chronic kidney disease
CT	computed tomography
eGFR	estimated glomerular filtration rate
ESRD	end-stage renal disease
GFR	glomerular filtration rate
ICU	intensive care unit
IV	intravenous
PCI	percutaneous coronary intervention
sCr	serum creatinine
STEMI	ST-elevation myocardial infarction

perception of contrast nephrotoxicity, despite methodological limitations such as absent control groups and unadjusted confounders.[4–6] Over time, the idea of "contrast-induced nephropathy" (CIN) became accepted as fact, despite inconsistency in definitions and lack of clinical proof. CIN is typically defined as an increase in serum creatinine by 0.3 mg/dL or percentage increase of 50% or greater ($\geq$1.5 fold above baseline) within 48 hours after contrast administration. This disease oriented outcome is of questionable clinical relevance. Additionally, similar fluctuations in creatinine are common among hospitalized patients and often occur for reasons unrelated to contrast exposure.

As newer data emerged, clinicians and researchers began to challenge the causality between IV contrast and kidney injury. Modern studies including those using propensity matching and well-controlled cohorts suggest that IV contrast is associated with AKI, but not causative in most cases.[7–17] This shift has prompted changes in terminology: many societies and journals now prefer the term "contrast-associated AKI" (CA-AKI), recognizing the distinction between association and causation.

This article critically appraises the use of peripheral IV contrast in emergency department (ED) patients, such as during contrast-enhanced computed tomography (CT) imaging for suspected pulmonary embolism, aortic dissection, or intra-abdominal pathology. This application is distinct from arterial contrast administration used in procedures like percutaneous coronary intervention (PCI) or diagnostic angiography, where patient populations, hemodynamic conditions, and contrast delivery routes differ substantially. The discussion aims to reassess the long-held belief that contrast-enhanced imaging in the emergency setting significantly contributes to AKI and to review the growing body of evidence supporting its safety in most patients. Emphasis is placed on facilitating evidence-based, timely diagnostic decision-making without unnecessary delays.

HISTORICAL CONTEXT: WHERE DID THE MYTH ORIGINATE?

The concept of CIN emerged from observational studies dating back to the 1950s to 1970s, which found temporal associations between contrast administration and increases in serum creatinine.[3,18–20] These early reports were often based on small, uncontrolled case series and involved contrast agents no longer in use—specifically high-osmolality ionic contrast media, which were more nephrotoxic than modern agents.

Throughout the 1990s and early 2000s, the literature continued to cite CIN as a common cause of hospital-acquired AKI. Yet these conclusions often stemmed from studies with serious methodological flaws. Emerging evidence suggested that these studies lacked appropriate control groups, failed to adjust for baseline kidney function or comorbidities, and/or used inconsistent definitions of AKI. In 2010, Katzberg and Newhouse[21] reviewed the available data and highlighted several methodological flaws, including the absence of control groups in much of the CIN literature. Additionally, it was noted that creatinine fluctuations can occur in patients not receiving contrast, calling into question the attribution of causality.[22]

The fear persisted, nonetheless, leading to the development of institutional protocols aimed at preventing contrast-related kidney injury. Hospitals began requiring routine serum creatinine testing prior to contrast-enhanced CT and implementing standardized hydration protocols, even as some studies began to question their necessity.

Studies using matched cohorts and modern statistical techniques began to tell a different story. Investigators such as McDonald, Hinson, and others showed that the incidence of AKI was similar between patients who received contrast and those who did not.[7,23] These findings challenged the long-standing assumption that IV contrast is a common cause of nephropathy.

PATHOPHYSIOLOGY: THEORETICAL BASIS FOR CONCERN

Several pathophysiological processes, including renal vasoconstriction, medullary hypoxia, and direct tubular toxicity have traditionally been proposed to explain CIN. These hypotheses were largely derived from early animal studies and in vitro experiments, providing biologic plausibility for contrast-related renal injury.[24,25] However, human clinical data supporting these mechanisms are limited and inconsistent, particularly with the use of modern low-osmolar and iso-osmolar contrast agents. In other words, studies have failed to demonstrate a consistent causal relationship between IV contrast exposure and measurable renal injury,[7,23,26] indicating that these theoretic mechanisms are likely of limited clinical significance with modern contrast agents.

CLARIFYING THE SCOPE: VENOUS VERSUS ARTERIAL CONTRAST

Some of the ongoing confusion around CIN stems from conflating 2 very different clinical scenarios: the use of venous contrast for diagnostic imaging and the use of arterial contrast during interventional procedures. Although both involve iodinated contrast agents, they differ markedly in the route of administration, patient population, and resulting physiologic effects.

Venous contrast is typically administered via peripheral IV access during CT angiography or contrast-enhanced CT of the chest, abdomen, or pelvis. These studies are commonly ordered in the ED for patients with suspected pulmonary embolism, aortic pathology, or intra-abdominal infection. Furthermore, these patients are often hemodynamically stable, and the contrast is delivered into the systemic circulation via a peripheral vein, passing through the right heart and lungs before entering the arterial system and ultimately reaching the kidneys. This route results in broader distribution before renal filtration, compared to the more direct and concentrated exposure of intra-arterial contrast. These differences in delivery route and concentration can potentially contribute to distinct renal hemodynamic effects and complicate direct comparisons between the two modalities.[27]

Arterial contrast is delivered directly into the arterial system during interventional procedures such as PCI or diagnostic angiography. Patients undergoing these

procedures, such as those with ST-elevation myocardial infarction (STEMI), represent a distinct, higher risk population with different hemodynamic and patient profiles. Arteriography is generally considered to carry a higher risk of kidney injury than a venous contrast load, primarily because it delivers a more concentrated bolus of contrast directly to the renal circulation and is performed in patients who typically have a higher baseline risk profile.[28] This higher risk context is exemplified in patients with STEMI, who often undergo emergent PCI and may later return for staged revascularization of additional vessels—situations that inherently involve repeated contrast exposure. These cases differ from typical ED or inpatient imaging cohorts, and findings from those populations should not be directly extrapolated.

Furthermore, rigorous study of arterial contrast exposure is inherently challenging because procedures such as PCI cannot be performed without contrast, precluding the existence of a true noncontrast control group. This methodological limitation complicates our ability to determine causality in the arterial contrast literature. Although true randomized comparisons are not feasible, some analyses have attempted to clarify this relationship.[29–31] In a cohort of 2025 STEMI patients undergoing primary PCI and 1025 noncontrast controls treated with fibrinolysis or conservative management, Caspi and colleagues reported no significant difference in AKI incidence, suggesting that contrast was not an independent determinant of renal dysfunction.

The 2024 American College of Radiology (ACR) Manual on Contrast Media acknowledges this ongoing uncertainty, emphasizing the need for individualized assessment in high-risk cardiovascular patients while reaffirming that, for most diagnostic contexts—including peripheral venous administration—modern IV contrast use remains safe, appropriate, and evidence-based.[32]

EVIDENCE FROM MODERN DATA

Venous Contrast in Emergency Department and Inpatients

Over the past decade, a growing body of evidence has challenged the presumed association between IV contrast administration and AKI in both ED and inpatient settings.

In a large, retrospective study of ED patients undergoing CT imaging, Hinson and colleagues[7] found no significant difference in the rate of AKI between patients who received IV contrast and those who did not. After adjusting for comorbidities and baseline renal function, the investigators concluded that IV contrast was not an independent risk factor for AKI. The findings contributed to a paradigm shift in ED imaging protocols, emphasizing individualized assessment of renal risk over indiscriminate avoidance of IV contrast.

Aycock and colleagues[8] performed a systematic review and meta-analysis of studies evaluating the risk of AKI following IV contrast administration in ED and hospitalized patients. Their findings demonstrated no significant association between IV contrast exposure and increased rates of AKI, dialysis, or mortality. The authors emphasized that when modern, low-osmolar contrast agents are used, and when appropriate hydration and attention to baseline renal function and comorbid risk factors are maintained, the risk of CIN is negligible. This analysis reinforced the growing consensus that contrast avoidance due to fear of CIN may lead to unnecessary diagnostic delays.

Building on prior research, Long and colleagues[9] reviewed multiple recent meta-analyses and large observational datasets, concluding that there remains no causal link between IV contrast and AKI in either ED or inpatient populations. They highlight that previously observed associations were likely due to confounding factors such as

underlying illness severity and hypotension, rather than contrast itself. Their synthesis supports current guideline trends encouraging the use of IV contrast when clinically indicated, rather than avoidance based on outdated concerns of CIN.

Critically Ill and Intensive Care Unit Patients

Recent evidence suggests that IV contrast administration is not independently associated with an increased risk of AKI in critically ill populations.[10] Lakhal and colleagues[11] had observed a relatively high incidence of AKI following contrast-enhanced imaging in intensive care unit (ICU) patients, but the study could not establish causality, as most patients had multiple concurrent risk factors for renal dysfunction. In a systematic review and meta-analysis, there was no significant difference in AKI incidence between contrast-exposed and unexposed critically ill patients, challenging the long-held belief that contrast media inherently causes nephrotoxicity in this high-risk group.[12] Similarly, Ehmann and colleagues[13] in 2023 analyzed more than 14,000 hospitalized patients, many of whom were in the ICU, and demonstrated that contrast exposure did not worsen renal recovery or increase the need for dialysis, even among those with pre-existing AKI. Most recently, in 2024, Hisamune and colleagues[14] analyzed more than 140,000 emergency and inpatient encounters, including ICU admissions, using a propensity-matched design. They found no increased risk of AKI or mortality among patients receiving IV contrast; in fact, adjusted analyses demonstrated slightly better outcomes in the contrast-exposed group. Collectively, these studies reinforce that in the era of low-osmolar contrast agents, the perceived risk of CIN in ICU patients is minimal, and contrast should not be withheld when clinically indicated for timely diagnosis or management.

CHANGING DEFINITIONS

KDIGO, an international nonprofit organization that creates and disseminates evidence-based guidelines in kidney disease, originally referenced the concept of "CIN" in prior recommendations.[33] As the evidence has evolved, KDIGO has moved away from the CIN construct and instead frames postcontrast renal injury within the broader definition of AKI, using the term "contrast-induced acute kidney injury" (CI-AKI) to better reflect current understanding of causation and risk. More recent literature considered by KDIGO indicates that the majority of acute kidney injuries following intravascular contrast is multifactorial in origin, reflecting contributions from hemodynamic instability, nephrotoxins, and existing comorbid conditions, rather than a direct causal effect from the contrast agent alone. For instance, a 2024 review noted that the term "CI-AKI" is reserved for cases where contrast is causally implicated, whereas the broader term "CA-AKI" captures any AKI after contrast exposure, without implying causality.[34] Thus, KDIGO's approach aligns with a shift away from rigid definitions of CIN toward an inclusive AKI framework, emphasizing that prevention and management should focus on overall AKI risk (baseline renal function, volume status, and nephrotoxin exposure) rather than only on the contrast administration event itself.

The ACR Manual on Contrast Media explicitly acknowledges the change in nomenclature from "contrast-induced acute kidney injury (CI-AKI)" to "contrast-associated acute kidney injury (CA-AKI)" to reflect that many cases of AKI after contrast are correlative rather than causally proven.[32] The manual states that in patients with stable estimated glomerular filtration rate (eGFR) of 30 mL/min or greater without other AKI risk factors, the risk of AKI attributable to IV contrast is very low. Thus, routine creatinine screening and prophylactic IV hydration are not routinely warranted and delays to contrast-enhanced imaging are usually unnecessary. Together, these changes reflect

an institutional shift away from blanket "CIN prevention" for all contrast exposures, toward a risk-stratified, judicious use of contrast.

AMERICAN COLLEGE OF RADIOLOGY RECOMMENDATIONS

EGFR, not serum creatinine, should guide renal risk assessment for contrast administration. Serum creatinine can be misleading because it varies with age, sex, muscle mass, and nutritional state, and may remain "normal" despite significant kidney impairment. EGFR incorporates these factors to better approximate true renal function and provides a more consistent measure across populations. Accordingly, current consensus statements—including those from the ACR and KDIGO—recommend defining risk thresholds using eGFR values, with 30 mL/min serving as the most evidence-supported cutoff for CA-AKI.[32,35]

When to Check Renal Function

According to the 2024 ACR Manual on Contrast Media,[32] preimaging serum creatinine or eGFR measurement is only indicated when the patient has one or more of the following risk factors:

- Known chronic kidney disease (CKD), especially eGFR less than 30 mL/min
- Recent or ongoing AKI
- Dialysis dependence
- History of kidney surgery or renal ablation
- Albuminuria
- Diabetes mellitus (optional depending on local policy)
- Current use of metformin or metformin-containing drug combinations (for post-imaging medication guidance)

For all other patients, routine laboratory testing is unnecessary and should not delay imaging.

Low-Risk Patients (Estimated Glomerular Filtration Rate ≥30)

Patients with preserved renal function rarely experience clinically significant CA-AKI. Routine screening or prophylaxis is not indicated, and contrast-enhanced imaging should proceed without delay.

Higher Risk or Known Kidney Disease (Estimated Glomerular Filtration Rate <30 or Recent Acute Kidney Injury)

In patients with advanced CKD (stage IV–V) or recent/ongoing AKI, the use of IV contrast should involve individualized risk–benefit assessment and potential collaboration with radiology. Contrast may still be administered when the diagnostic benefit outweighs potential harm, but documentation should reflect:

- The indication and expected diagnostic impact
- A brief discussion of risk with the patient when feasible
- Any planned hydration strategies

Hydration remains the most consistently supported preventive measure for patients at high risk, though the evidence for universal prophylaxis is weak and largely extrapolated from arterial contrast studies.[36–39] For the general population and for patients with fairly preserved renal function (eGFR ≥30), there is no evidence or guideline-based recommendation to provide prophylactic IV hydration before or after IV contrast administration.

Anuric End-Stage Renal Disease

Anuric patients with end-stage renal disease (ESRD) who lack a functioning transplant kidney are not at risk for CA-AKI, as their kidneys are nonfunctional. These patients may safely receive intravascular iodinated contrast without the risk of further renal injury. Contrast use in such cases should be guided primarily by diagnostic need rather than renal concerns.

Repeat Contrast Exposures

One risk factor for nephrotoxicity is multiple doses of intravascular contrast within a short time frame. Most low-osmolality iodinated contrast agents have a plasma half-life of approximately 2 hours, meaning near total elimination less than a day in patients with normal renal function. Historically, dosing intervals shorter than 24 hours have been discouraged, though the ACR notes that contrast should not be withheld when clinically necessary.

The decision to perform closely spaced contrast-enhanced studies is ultimately clinical and subjective. In general, patients with normal renal function can undergo repeat studies if needed, while high-risk individuals (eGFR <30, AKI) should be treated with greater caution (**Table 1**).

WHY THE MYTH PERSISTS—AND HOW IT CAN HARM CARE

The persistence of the belief that IV contrast routinely causes kidney injury reflects a combination of cognitive bias and institutional inertia rather than contemporary evidence. Once a clinical concept is labeled as a risk, it becomes psychologically anchored, making it difficult to unlearn. Clinicians may also fall prey to confirmation bias, recalling instances of creatinine rise after contrast while overlooking equally common rises in patients who never received it. Despite robust modern data disproving a causal link in most circumstances,[7–14] the cultural memory of CIN continues to influence policy and bedside decision-making. Defensive medicine and medicolegal caution further reinforce these outdated practices, perpetuating diagnostic delays and avoidance of appropriate imaging.

Modern literature highlights that kidney function can be assessed through several measures, including measured glomerular filtration rate (GFR) and eGFR, each with its own advantages and limitations.[35] EGFR formulas estimate renal function based on serum creatinine values adjusted for age, sex, and body size. These equations can offer a standardized approach to estimating renal function, but they are most reliable when serum creatinine (sCr) is in a steady state.[35,40,41]

Table 1
Summary of guidelines for contrast media use based on renal function and clinical scenario

Scenario	Recommendation
eGFR ≥30 mL/min	Proceed without delay or prophylaxis
eGFR <30 mL/min or recent AKI	Relative but not absolute contraindication. Consider alternatives (US/MRI/noncontrast CT) but if benefits outweigh risks proceed with contrast-enhanced study
Anuric ESRD	May receive contrast; no risk of additional renal injury
Repeat contrast <24 h apart	Generally safe in low-risk patients; use caution and clinical judgment in CKD stage IV–V or AKI

In clinical practice, most eGFR formulas assume a stabilized sCr concentration and, therefore, calculate kidney function using a single creatinine measurement taken. However, one study demonstrated that one-third of ED patients present with non–steady-state serum creatinine, meaning that kidney function may be significantly changing when rechecked.[41] Clinicians should, therefore, interpret eGFR with caution in acutely ill patients and consider the clinical trajectory, fluid status, and timing of the creatinine measurement before labeling a case as "CIN". Appropriate assessment of kidney function—rather than reflexive attribution of creatinine changes to contrast exposure—is essential to avoid perpetuating the historical misconception that IV contrast is a frequent cause of AKI.

Institutional protocols, often written decades ago, have also contributed to the inertia. Many hospitals still require routine preimaging creatinine checks or automatic hydration orders, even in low-risk patients. Such measures can inadvertently delay time-sensitive diagnostic studies. A recent ED root-cause analysis identified contrast-related institutional policies as modifiable contributors to delayed CT imaging, underscoring the operational cost of outdated safeguards.[1] Current ACR recommendations explicitly discourage these routine delays and advocate a selective screening approach, emphasizing the primacy of diagnostic urgency.[32]

The consequences of persistent contrast fear are not merely academic, they are clinical and systemic. In emergency medicine, delayed or substituted imaging can compromise patient outcomes. Deferral of CT angiography in suspected aortic dissection or pulmonary embolism can delay the diagnosis, which could lead to poorer outcomes. Likewise, non–contrast abdominal CT scans have been shown to be approximately 30% less accurate than contrast-enhanced studies, underscoring that important diagnoses may be missed when contrast is withheld.[42] Among patients with trauma, including those elderly, hypotensive, or with an ISS 25 or greater, IV contrast was not associated with an increased incidence of AKI.[43] Thus, again overreliance on noncontrast imaging can risk missed pathology and the need for repeat scans unnecessarily.

Ultimately, the persistence of the "contrast nephropathy" narrative reflects the slow pace of cultural change in medicine. The evidence now demonstrates that IV contrast is not an independent nephrotoxin in patients with eGFR 30 or greater. Modern guidelines from the ACR emphasize selective testing, patient-specific risk assessment, and timely diagnosis as the priorities. In the emergency setting, patient harm is more likely to result from delayed or missed diagnosis due to unwarranted avoidance of contrast than from the contrast itself.

SUMMARY

The belief that IV contrast routinely causes kidney injury has persisted far longer than the data supporting it. While early studies established biologic plausibility for renal toxicity, they relied on outdated agents and uncontrolled designs. Modern evidence demonstrates that contrast administration is associated with AKI but is rarely the direct cause. In most emergency populations, the risk of CIN is exceedingly low, especially with contemporary low-osmolar and iso-osmolar agents.

Guidelines from the ACR now reflect this reality, recommending against routine preimaging creatinine testing for low-risk patients. Instead, clinicians should focus on the greater danger of delayed diagnosis when contrast studies are withheld.

In the era of patient-centered care, involving the patient in this conversation is both ethical and evidence-based. For individuals with eGFR less than 30 or recent AKI, clinicians should briefly explain that the risk of worsening kidney function from IV

contrast is low and that the imaging test may be essential to avoid a missed or delayed diagnosis. Documentation should note both the clinical rationale and the risk–benefit discussion, which satisfies ACR guidance and supports defensible, transparent care.

Emergency physicians are uniquely positioned to bridge evidence and practice. Reframing contrast as a diagnostic ally rather than a nephrotoxin allows for timely, patient-centered care grounded in modern data rather than myth. Ultimately, the greater harm lies not in using contrast, but in failing to use it when the diagnosis may depend on it.

CLINICS CARE POINTS

- IV contrast rarely causes AKI. Most post-contrast creatinine rises are multifactorial and not directly attributable to modern low- or iso-osmolar agents.
- Use eGFR to guide decisions. If eGFR $\geq$ 30 mL/min and renal function is stable, proceed with contrast-enhanced imaging without delay or prophylactic hydration.
- eGFR < 30 mL/min or active AKI is a relative—not absolute—contraindication. Perform an individualized risk–benefit assessment, especially when the study is time-sensitive.
- Do not conflate venous CT contrast with arterial PCI contrast. Peripheral IV contrast used in diagnostic CT carries substantially lower renal risk.
- Delayed or missed diagnosis is often the greater harm. In suspected PE, aortic dissection, stroke, or intra-abdominal emergencies, withholding contrast may pose more risk than administering it.

DECLARATION OF AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this article, the author(s) used ChatGPT in order to proofread for spelling and grammatical errors and to optimize readability. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

DISCLOSURE

None.

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