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SHOULD CONTRAST-ENHANCED COMPUTED TOMOGRAPHY BE WITHHELD IN PATIENTS WITH IMPAIRED KIDNEY FUNCTION? A NARRATIVE REVIEW OF CURRENT EVIDENCE

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ABSTRACT

Intravenous iodinated contrast media improve the diagnostic performance of contrast-enhanced computed tomography (CECT), but concerns about acute kidney injury continue to influence its use in patients with impaired kidney function. This narrative review evaluates current evidence on the kidney safety of modern intravenous iodinated contrast media and examines whether clinically indicated CECT should be withheld in these patients. PubMed and professional guidelines were searched for controlled observational studies, propensity-adjusted analyses, systematic reviews, randomized prophylaxis trials and consensus recommendations concerning intravenous iodinated contrast administration. Evidence distinguishes contrast-associated acute kidney injury, which indicates a temporal relationship, from contrast-induced acute kidney injury, which implies causation. The additional risk attributable to modern intravenous iodinated contrast media appears minimal in patients with a stable estimated glomerular filtration rate (eGFR) ≥45 mL/min/1.73 m^2. Risk also remains low in most patients with eGFR 30–44 mL/min/1.73 m^2, although diabetes and concurrent kidney insults may increase susceptibility. Greater uncertainty persists in patients with eGFR <30 mL/min/1.73 m^2 or active acute kidney injury. These conditions should prompt individualized risk–benefit assessment rather than automatic exclusion from CECT. Routine prophylactic hydration is generally unnecessary when eGFR is stable at ≥30 mL/min/1.73 m^2 but may be considered in selected high-risk patients. When contrast enhancement is necessary to answer a clinical question, the potential kidney risk should be balanced against the consequences of delayed, missed or incomplete diagnosis. Impaired kidney function alone should therefore not determine whether clinically necessary CECT is performed.

Keywords: Acute Kidney Injury; Chronic Kidney Disease; Contrast-Associated Acute Kidney Injury; Contrast-Enhanced Computed Tomography; Contrast-Induced Acute Kidney Injury; Iodinated Contrast Media

1 INTRODUCTION

Contrast-enhanced computed tomography (CECT) plays a central role in the detection, characterization, staging and management of numerous diseases. Intravenous iodinated contrast media improve the visualization of organs, blood vessels and pathological enhancement and are particularly valuable in oncological, vascular, abdominal, infectious, traumatic and emergency imaging. Nevertheless, concern about acute deterioration in kidney function continues to influence decisions regarding contrast administration, especially in patients with chronic kidney disease (CKD) or acute kidney injury (AKI).

Historically, an increase in serum creatinine occurring shortly after iodinated contrast administration was frequently attributed directly to the contrast medium and described as contrast-induced nephropathy. This assumption led to the postponement or cancellation of CECT examinations, routine prophylactic hydration, arbitrary reduction of contrast doses and substitution with unenhanced or alternative imaging. Much of the early evidence, however, was derived from uncontrolled studies, older high-osmolar contrast media and intra-arterial procedures. Consequently, these studies could not reliably distinguish kidney injury caused by contrast from AKI resulting from the patient's underlying illness or other concurrent kidney insults [1,2].

Patients undergoing CT frequently have conditions that independently predispose them to AKI, including sepsis, hypotension, haemorrhage, volume depletion, cardiovascular disease, pre-existing CKD and exposure to potentially nephrotoxic medications. Contemporary terminology therefore distinguishes contrast-associated acute kidney injury (CA-AKI), which describes AKI occurring after contrast administration without establishing causation, from contrast-induced acute kidney injury (CI-AKI), in which the contrast medium is considered to have contributed causally to the kidney injury [1]. The European Society of Urogenital Radiology uses the related term post-contrast acute kidney injury (PC-AKI) for deterioration in kidney function occurring after intravascular contrast administration without necessarily attributing the change to contrast exposure [2].

Controlled observational studies and propensity-adjusted analyses have substantially changed estimates of the kidney risk associated with modern intravenous iodinated contrast media. Several studies have reported comparable rates of AKI among patients undergoing CECT and unenhanced CT, particularly when baseline kidney function is preserved or only moderately impaired [3,6–13]. These findings are clinically reassuring, but they should be interpreted cautiously because propensity-based methods cannot eliminate unmeasured confounding or selection bias. Patients considered to be at the greatest risk may preferentially undergo unenhanced CT, while those receiving contrast may differ in illness severity, clinical indication and preventive management.

More recent multi-centre evidence suggests that the additional risk attributable to contrast may not be uniform across all levels of kidney function. The risk appears minimal in patients with stable estimated glomerular filtration rate (eGFR) ≥45 mL/min/1.73 m^2 and remains low in most patients with eGFR 30–44 mL/min/1.73 m^2. However, diabetes and other concurrent kidney insults may increase susceptibility within the latter group. A clinically relevant risk becomes more plausible when eGFR is below 30 mL/min/1.73 m^2, although the available evidence remains conflicting and affected by residual confounding [13–15]. The incremental risk is also difficult to quantify in active AKI because serum creatinine is changing and eGFR estimates are unreliable in non-steady-state kidney function.

The decision to administer iodinated contrast media should therefore not be based on kidney function alone. Avoiding contrast exposure may prevent a potential adverse effect, but an unenhanced examination may be diagnostically inadequate for the clinical question. The resulting consequences may include missed or delayed diagnosis, repeat imaging, postponed treatment or inappropriate management. These risks are particularly important in suspected active haemorrhage, aortic and other vascular emergencies, bowel ischaemia, major trauma, complicated infection, organ infarction and malignancy.

This narrative review evaluates current evidence concerning CI-AKI following intravenous iodinated contrast administration for CECT in patients with impaired kidney function. It examines contemporary terminology, the strength and limitations of the available evidence, risk according to baseline kidney function, patient- and procedure-related risk factors, preventive measures and current professional recommendations. It also proposes a practical approach to balancing potential kidney injury against the diagnostic consequences of withholding clinically necessary contrast enhancement.

2 MATERIAL AND METHODS

2.1 Review design

This narrative review examined current evidence concerning acute kidney injury following intravenous iodinated contrast administration for contrast-enhanced computed tomography (CECT). The review focused on the distinction between contrast-associated acute kidney injury (CA-AKI) and contrast-induced acute kidney injury (CI-AKI), the influence of baseline kidney function, preventive measures and recommendations concerning the performance of CECT in patients with impaired kidney function. The review was conducted as a narrative synthesis and was not designed as a systematic review or meta-analysis.

2.2 Literature search

PubMed was searched for English-language publications from January 2010 to August 2026. The search combined relevant Medical Subject Headings and free-text terms, including:

"iodinated contrast media," "intravenous contrast media," "contrast-enhanced computed tomography," "contrast-associated acute kidney injury," "contrast-induced acute kidney injury," "post-contrast acute kidney injury," "contrast-induced nephropathy," "chronic kidney disease," "acute kidney injury," "estimated glomerular filtration rate," "hydration," "metformin" and "dialysis."

The terms were combined using the Boolean operators "AND" and "OR" as appropriate. Reference lists of relevant reviews, consensus statements and eligible primary studies were also examined to identify additional publications. Earlier landmark studies were retained when they were necessary to explain the historical development of current concepts or remained influential in clinical practice.

Guidelines and consensus documents were identified from the American College of Radiology, National Kidney Foundation and European Society of Urogenital Radiology. The most recent available versions were prioritised, while earlier guidance was included when it established definitions or recommendations that remain relevant.

2.3 Eligibility and selection of evidence

The review prioritised studies that evaluated intravenous iodinated contrast administration for CECT and included a clinically comparable unexposed or unenhanced CT group. Eligible evidence comprised controlled cohort studies, propensity-score-adjusted or propensity-matched analyses, multi-centre studies, systematic reviews, meta-analyses, randomized controlled trials of preventive interventions and professional guidelines or consensus statements.

Studies confined to intra-arterial contrast administration were not used to estimate the kidney risk of intravenous contrast-enhanced CT because the two routes involve different clinical circumstances and potential mechanisms of kidney injury. However, selected angiographic trials were included when they provided directly relevant evidence concerning preventive interventions, such as intravenous hydration, sodium bicarbonate or N-acetylcysteine. Their indirect applicability to routine intravenous CECT was explicitly acknowledged.

Case reports, small uncontrolled case series, animal studies, non-English publications and articles without clinically relevant kidney outcomes were excluded from the principal evidence synthesis. Studies involving older high-osmolar contrast media were used only to provide historical context.

2.4 Evidence appraisal and synthesis

The authors assessed potentially relevant publications according to study design, population, baseline kidney function, route of contrast administration, type of contrast medium, definition of AKI, comparator group, adjustment for confounding and reported clinical outcomes. Particular attention was given to whether studies distinguished temporal association from a causal relationship between contrast exposure and AKI.

Because substantial heterogeneity existed in patient populations, AKI definitions, contrast protocols and statistical adjustment, the evidence was synthesized narratively rather than pooled quantitatively. Greater interpretative weight was assigned to controlled multi-centre studies, adequately adjusted cohort analyses, systematic reviews, randomized prophylaxis trials and professional consensus statements. Residual confounding, selection bias, underrepresentation of patients with severe chronic kidney disease and the unreliability of eGFR during active AKI were considered when interpreting the findings.

No formal risk-of-bias instrument or certainty-of-evidence grading system was applied because this was a narrative review. Accordingly, descriptions such as “minimal,” “low,” “uncertain” or “clinically plausible” were used as qualitative interpretations of the available evidence rather than as formal evidence grades.

3 RESULTS AND DISCUSSION

3.1 Terminology and definitions: CA-AKI, PC-AKI and CI-AKI

Accurate terminology is essential when interpreting changes in kidney function following iodinated contrast administration. Historically, terms such as contrast-induced nephropathy (CIN) were commonly applied whenever serum creatinine increased after contrast exposure, even when a causal relationship had not been established. This practice contributed to overestimation of the nephrotoxic effect of intravenous contrast media [1,2].

Contrast-associated acute kidney injury (CA-AKI) refers to AKI occurring within a clinically relevant period after contrast administration, irrespective of whether the contrast medium caused the deterioration. The term describes a temporal association and includes kidney injury arising from other factors, such as sepsis, hypotension, haemorrhage, dehydration, cardiac dysfunction, medication exposure or progression of underlying kidney disease [1].

Post-contrast acute kidney injury (PC-AKI) is the term preferred by the European Society of Urogenital Radiology and is conceptually similar to CA-AKI. It describes a deterioration in kidney function following intravascular contrast administration without automatically attributing causation to the contrast medium [2]. The European Society of Urogenital Radiology defines PC-AKI as an increase in serum creatinine of at least 0.3 mg/dL (26.5 μmol/L) or at least 1.5 times the baseline value within 48–72 hours after intravascular contrast administration [2].

By contrast, contrast-induced acute kidney injury (CI-AKI) implies that iodinated contrast contributed causally to the kidney injury. Establishing CI-AKI requires more than demonstrating an increase in serum creatinine after CECT. Competing causes of AKI must be considered, ideally through comparison with clinically similar patients who did not receive contrast. Although every CI-AKI event occurs after contrast administration, only a proportion of CA-AKI or PC-AKI events can reasonably be attributed to contrast exposure.

The term CIN is increasingly discouraged because it presumes both nephropathy and causation. It may remain appropriate when discussing historical studies that originally used the term, but CA-AKI, PC-AKI and CI-AKI provide greater precision in contemporary scientific writing. In this review, CA-AKI is used for temporally associated kidney injury, CI-AKI for kidney injury considered attributable to iodinated contrast and PC-AKI when referring specifically to European Society of Urogenital Radiology terminology.

3.2 Historical perspective and the changing concept of contrast nephrotoxicity

Early estimates of contrast nephrotoxicity were derived largely from uncontrolled studies, older high-osmolar agents and intra-arterial procedures. Their relevance to contemporary intravenous CECT is limited because acutely ill patients commonly develop creatinine fluctuations from sepsis, hypotension, dehydration or medication exposure even without contrast [1,2].

Controlled cohorts and propensity-adjusted comparisons subsequently found similar AKI rates in many patients undergoing CECT and unenhanced CT, particularly when baseline kidney function was preserved or only moderately impaired [3,6–13]. These observational methods reduce measured confounding but cannot eliminate treatment-selection bias or unmeasured differences.

Modern intravenous iodinated contrast should therefore be regarded as carrying a graded rather than uniform kidney risk. Attributable risk appears small in most patients but remains uncertain in active AKI and severe CKD, where evidence is limited and conflicting [1,5,14–16].

3.3 Contemporary evidence for CI-AKI following intravenous CECT

The principal question is not how frequently AKI occurs after CECT, but whether intravenous iodinated contrast increases that frequency above the background rate in comparable patients who do not receive contrast. Randomized comparisons of CECT and unenhanced CT are rarely feasible because contrast administration is determined by the clinical indication. Most current evidence therefore comes from controlled observational studies using regression adjustment, propensity-score matching or weighting to reduce measured differences between exposed and unexposed patients.

McDonald and colleagues compared patients undergoing CECT with propensity-matched patients undergoing unenhanced CT and found no significant difference in the incidence of AKI [3]. A subsequent analysis of 21,346 propensity-matched patients found that intravenous contrast exposure was not independently associated with emergency dialysis or 30-day mortality [7]. Among patients with stage 3 or stage 4–5 CKD, another propensity-adjusted study reported no significant increase in AKI, emergency dialysis or short-term mortality following intravenous contrast administration [8].

Other clinical settings have produced similarly reassuring findings. Hinson and colleagues evaluated emergency-department patients who underwent CECT, unenhanced CT or no CT and found that intravenous contrast administration was not independently associated with AKI [10]. A propensity-adjusted study of intravenous iodixanol also found no significant increase in AKI, dialysis or mortality across the examined kidney-function strata [9]. In critically ill patients, however, no significant association was demonstrated when pre-CT eGFR exceeded 45 mL/min/1.73 m^2, whereas an increased incidence of AKI was observed among patients with eGFR ≤45 mL/min/1.73 m^2. This increase was not accompanied by a statistically significant rise in dialysis or mortality [11].

A five-centre propensity-weighted study of 68,687 emergency-department patients by Su et al. found a modest increase in AKI 48–72 hours after contrast exposure overall (odds ratio 1.16; 95% confidence interval 1.04–1.29), but no increase from 48 hours to 1 week. Among patients with eGFR <30 mL/min/1.73 m^2, CECT was associated with higher risks of AKI and haemodialysis, whereas no increased AKI risk was demonstrated at eGFR >45 mL/min/1.73 m^2 [13].

Systematic reviews have generally reported similar overall conclusions. A meta-analysis of controlled studies found comparable rates of AKI, dialysis and death between contrast-exposed and unexposed patients [6]. A later meta-analysis of CT studies similarly found no significant overall increase in AKI, renal replacement therapy or mortality following intravenous contrast administration [12]. These pooled findings are reassuring for the general population but may provide limited certainty for patients with severe CKD because such patients constituted a relatively small proportion of many included cohorts.

More recent studies suggest that clinically important heterogeneity exists across eGFR and diabetic subgroups. Lee and colleagues examined 75,328 patients and found that, compared with unenhanced CT, CECT was associated with higher odds of AKI among patients with eGFR <30 mL/min/1.73 m^2, both with and without diabetes. Among patients with eGFR 30–44 mL/min/1.73 m^2, increased odds were confined to those with diabetes. The reported odds ratios were 2.12 and 1.62 in patients with eGFR <30 mL/min/1.73 m^2 with and without diabetes, respectively, and 1.83 in patients with diabetes and eGFR 30–44 mL/min/1.73 m^2 [14]. Higher odds of 30-day dialysis were demonstrated only in patients with diabetes and eGFR <30 mL/min/1.73 m^2.

A 16-institution retrospective cohort study published in 2025 also found that the attributable risk of CI-AKI was minimal in the general CT population but advised greater caution in patients with CKD and eGFR below 45 mL/min/1.73 m^2 [15]. The study provides important contemporary multi-centre evidence, although its retrospective design remains susceptible to residual confounding and differences in clinical selection.

Evidence concerning patients with active AKI is more limited. A propensity-matched study of hospitalized patients with established AKI found no significant increase in subsequent deterioration of kidney function, new renal replacement therapy or mortality following intravenous contrast exposure [16]. This finding does not establish universal safety because serum creatinine is already changing in active AKI, eGFR is unreliable in non-steady-state kidney function, and clinical selection strongly influences whether contrast is administered.

Taken together, the available evidence supports a graded interpretation. The additional risk attributable to modern intravenous iodinated contrast appears minimal when kidney function is preserved and remains low in most patients with stable moderate impairment. Increased risk becomes more plausible in selected patients with eGFR 30–44 mL/min/1.73 m^2, particularly when diabetes or other concurrent kidney insults are present, and is of greatest concern when eGFR is below 30 mL/min/1.73 m^2. Active AKI remains a high-risk clinical state in which the independent contribution of contrast cannot be estimated reliably.

Table 2: Summary of major evidence concerning CI-AKI following intravenous CECT

Study or evidence group	Design and population	Principal finding	Important limitation
McDonald et al. [3,7]	Propensity-matched CT cohorts	No significant overall increase in AKI, emergency dialysis or 30-day mortality	Retrospective design and residual confounding
McDonald et al. [8]	Propensity-adjusted patients with CKD	No significant increase in AKI, dialysis or mortality in the examined CKD groups	Limited numbers in the most severe kidney-function categories
Hinson et al. [10]	Emergency-department cohort comparing CECT, unenhanced CT and no CT	Contrast was not independently associated with AKI	Single-centre observational design
McDonald et al. [9]	Propensity-adjusted study of intravenous iodixanol	No significant increase in AKI, dialysis or mortality	Contrast-agent and institutional specificity
McDonald et al. [11]	Propensity-adjusted intensive-care cohort	Increased AKI at eGFR ≤45 but no significant increase in dialysis or mortality	Critically ill population with substantial competing causes of AKI
Controlled meta-analyses [6,12]	Systematic reviews and meta-analyses	No significant overall increase in AKI, renal replacement therapy or mortality	Heterogeneity and underrepresentation of severe CKD
Lee et al. [14]	Large cohort stratified by diabetes and eGFR	Increased AKI risk below eGFR 30 and at eGFR 30–44 among patients with diabetes	Retrospective design and possible residual confounding
Su et al. [13]	Five-centre emergency-department propensity-weighted cohort	Higher AKI and haemodialysis risks at eGFR <30 mL/min/1.73 m^2; no increased AKI risk at eGFR >45 mL/min/1.73 m^2	Retrospective design with possible residual confounding and treatment-selection bias
Choi et al. [15]	Sixteen-institution retrospective cohort	Minimal general-population risk; greater concern below eGFR 45	Observational design and treatment-selection bias
Yan et al. [16]	Propensity-matched hospitalized patients with active AKI	No significant worsening of kidney outcomes after contrast exposure	eGFR unreliable during active AKI; strong possibility of selection bias

3.4 Kidney function and patient-related risk factors

Baseline kidney function is the most important measurable factor in assessing the likelihood of AKI following intravenous iodinated contrast administration. In patients with stable eGFR ≥45 mL/min/1.73 m^2, controlled studies have demonstrated little or no additional risk attributable to contrast [1,3–15]. Most patients with stable eGFR 30–44 mL/min/1.73 m^2 also appear to have a low attributable risk, although recent evidence indicates that risk may be higher in selected subgroups, particularly patients with diabetes or concurrent causes of kidney injury [14,15].

Patients with eGFR <30 mL/min/1.73 m^2 constitute the principal group in whom a clinically relevant contrast-related risk remains plausible. Evidence in this population is inconsistent because patients with severe CKD are frequently underrepresented in controlled studies and may be preferentially excluded from contrast administration. An eGFR below 30 mL/min/1.73 m^2 should therefore trigger an individualized risk–benefit assessment, but it should not be treated as an absolute contraindication to clinically necessary CECT [1,14,15].

Active AKI is another important high-risk state. Serum creatinine is changing during AKI, and equations used to calculate eGFR assume stable kidney function. The reported eGFR may therefore misrepresent the patient's current filtration capacity. Decisions concerning contrast administration in active AKI should be based on the clinical course, urine output, likely cause and reversibility of the kidney injury, haemodynamic status, urgency of imaging and expected diagnostic benefit rather than on a single calculated eGFR value [1,16].

Diabetes mellitus is best regarded as a risk modifier rather than an independent contraindication. Diabetes alone, in the absence of CKD or AKI, does not justify withholding intravenous contrast. However, Lee and colleagues found increased odds of AKI among patients with diabetes and eGFR 30–44 mL/min/1.73 m^2, suggesting that diabetes may identify a more susceptible subgroup within moderate CKD [14]. When eGFR was below 30 mL/min/1.73 m^2, increased risk was observed both in patients with and without diabetes.

Sepsis, hypotension, haemorrhage, volume depletion, heart failure and major surgery increase the background risk of AKI. These conditions also make it difficult to determine whether a subsequent deterioration in kidney function was caused by contrast or by the underlying illness. When clinically feasible, significant volume depletion and haemodynamic instability should be corrected before contrast administration, provided that doing so does not delay urgent or potentially life-saving imaging.

3.5 Contrast- and procedure-related factors

3.5.1 Osmolality and type of contrast medium

Modern CECT is performed predominantly with non-ionic low-osmolar contrast media, while iso-osmolar iodixanol is used in some institutions and clinical circumstances. High-osmolar contrast media are associated with more adverse physiological effects and are no longer routinely used for intravenous CECT.

Available evidence does not demonstrate a consistent, clinically meaningful kidney-safety advantage of iso-osmolar iodixanol over contemporary low-osmolar contrast media following intravenous administration [1,2,9]. The choice of agent should therefore consider diagnostic performance, patient history, local availability, cost and institutional protocol rather than an assumption that iso-osmolar contrast eliminates the possibility of kidney injury. The 2025 multi-centre study by Choi and colleagues also indicates that contrast osmolality should not be considered independently of baseline kidney function and clinical context [15].

3.5.2 Contrast dose and iodine delivery

Contrast dose should be sufficient to answer the clinical question. Unnecessary iodine exposure should be avoided, particularly in patients with severe CKD or active AKI. However, arbitrary reduction of the contrast dose below a diagnostically adequate level may produce insufficient vascular or tissue enhancement, resulting in a nondiagnostic examination, repeat imaging or delayed treatment [1].

A clear dose–response relationship between intravenously administered contrast volume and CI-AKI has not been consistently demonstrated in routine CECT. This differs from some intra-arterial cardiovascular procedures, in which large contrast volumes may accompany prolonged procedures, haemodynamic instability and direct renal arterial exposure. Contrast volume alone should therefore not be used as a substitute for assessment of baseline kidney function and the overall clinical condition.

Validated low-iodine protocols may be appropriate when they preserve diagnostic quality. Lower tube-voltage acquisition, dual-energy or spectral CT, virtual monoenergetic reconstruction and iterative or other advanced reconstruction methods can improve iodine conspicuity and may permit lower iodine doses for selected examinations [17,18]. Such protocols should be optimized for the patient's body habitus and the diagnostic task rather than applied indiscriminately.

3.5.3 Route of administration

Intravenous and intra-arterial contrast administration represent different risk settings. Intra-arterial procedures may involve first-pass renal exposure, catheter manipulation, atheroembolism, haemodynamic instability and larger or repeated contrast doses. The European Society of Urogenital Radiology therefore distinguishes intra-arterial first-pass renal exposure from second-pass exposure [2].

3.5.4 Repeated contrast exposure

Repeated contrast administration may be required in trauma, critical care, postoperative imaging, vascular emergencies and oncological assessment. Recent contrast exposure should be considered in the overall risk assessment, particularly in patients with active AKI, severe CKD, haemodynamic instability or multiple concurrent kidney insults. Nevertheless, a recent contrast examination is not an absolute reason to withhold another clinically necessary CECT examination.

When the examination is elective and clinically safe to postpone, allowing time for kidney function to stabilize may be reasonable in high-risk patients. In urgent or life-threatening circumstances, imaging should not be delayed solely to satisfy an arbitrary interval between contrast administrations. The timing should reflect the patient's kidney function, clinical condition, previous contrast dose, urgency and expected diagnostic benefit [1,2].

3.5.5 Diagnostic indication and protocol optimization

The diagnostic indication remains central to contrast-related decision-making. The potential kidney risk may be acceptable when CECT is required to diagnose active haemorrhage, vascular occlusion, bowel ischaemia, major traumatic injury, organ infarction, complicated infection or malignancy. Conversely, contrast should not be administered when enhancement is unlikely to improve diagnostic accuracy or patient management.

Protocol optimization should involve communication between the radiologist, referring clinician and CT team. The objective is to obtain adequate diagnostic information with an appropriate contrast agent, iodine dose and acquisition technique while avoiding unnecessary repeat phases or examinations. This approach is preferable to reflexively withholding contrast or reducing the dose without considering image quality.

3.6 Prevention and periprocedural management

3.6.1 Pre-contrast assessment

When kidney function is stable, eGFR is preferred to serum creatinine alone. Active AKI should be identified clinically because creatinine-based eGFR assumes steady-state function. Assessment should consider recent kidney trends, hydration, haemodynamics, comorbidities, medications and the urgency and expected value of CECT [1,2].

3.6.2 Hydration

The ACR–NKF consensus recommends prophylactic isotonic saline principally for active AKI or stable eGFR <30 mL/min/1.73 m^2 when volume expansion is not contraindicated [1]. The AMACING trial found no prophylaxis non-inferior to hydration in selected elective patients with eGFR 30–59 mL/min/1.73 m^2, with fewer hydration-related complications; however, patients with eGFR <30 were excluded [19]. Hydration should therefore be selective and individualized, particularly in heart failure or fluid-overload states.

3.6.3 Pharmacological prophylaxis

The PRESERVE trial found no benefit from sodium bicarbonate over isotonic saline or from N-acetylcysteine over placebo [20]. ESUR likewise reports no consistently effective pharmacological prophylaxis; N-acetylcysteine, diuretics, dopamine, fenoldopam, theophylline and statins should not be used routinely solely to prevent PC-AKI [21].

3.6.4 Medication, dialysis and follow-up

Non-essential nephrotoxic medication may be withheld selectively in active AKI or severe CKD. Metformin does not cause CI-AKI, but should be temporarily withheld in active AKI or eGFR <30 mL/min/1.73 m^2 and restarted after kidney function is stable [21,22]. Dialysis should not be initiated or rescheduled solely to remove contrast [1,21]. Routine post-CECT creatinine testing is unnecessary in stable patients with eGFR ≥30 and no AKI; targeted follow-up is appropriate when deterioration would alter management.

3.7 When should CECT be withheld?

The decision should balance the possibility of kidney injury against the consequences of incomplete or delayed diagnosis. In stable eGFR ≥45 mL/min/1.73 m^2, attributable risk is minimal; at 30–44 mL/min/1.73 m^2, most patients can undergo indicated CECT without prophylaxis, although diabetes and concurrent kidney insults may justify additional assessment [1,14,15].

Stable eGFR <30 mL/min/1.73 m^2 and active AKI warrant individualized review of urgency, expected benefit, kidney trajectory, haemodynamics and alternatives, but neither is an absolute contraindication. Elective imaging may be postponed when recovery is expected and delay is safe; urgent CECT should proceed when diagnostic harm from withholding contrast is greater than the plausible kidney risk [1,5,8,9,14–16].

Contrast should be omitted when it will not improve diagnostic accuracy or management, or when a timely alternative provides equivalent information. Conversely, impaired kidney function should not prevent contrast use for time-sensitive conditions such as active haemorrhage, aortic syndromes, pulmonary embolism, mesenteric ischaemia, complicated infection or cancer staging. Maintenance dialysis schedules should not be altered solely because contrast was administered [1,21].

3.8 Current guidelines and practical decision-making

Current ACR–NKF, ACR and ESUR guidance supports individualized decision-making rather than automatic exclusion from CECT. When kidney function is stable, eGFR should guide assessment; active AKI and eGFR <30 mL/min/1.73 m^2 warrant closer risk–benefit evaluation. Routine prophylaxis is unnecessary at stable eGFR ≥30 mL/min/1.73 m^2, while

isotonic saline may be considered for active AKI or lower eGFR when volume expansion is safe. Urgent, diagnostically necessary CECT should not be delayed solely because of impaired kidney function [1,22,23].

The current American College of Radiology Manual similarly treats eGFR <30 mL/min/1.73 m^2 and active AKI as the principal situations requiring enhanced assessment. It advises against lowering the contrast dose below a diagnostically adequate level solely because of impaired kidney function and does not support initiating or altering dialysis exclusively because iodinated contrast has been administered [22].

The 2025 European Society of Urogenital Radiology guidelines address prevention of CA-AKI following iodine-based contrast administration, dialysis, repeat contrast exposure and safe intervals between contrast injections [23]. These updated guidelines use CA-AKI in their principal kidney-safety recommendations, whereas the earlier 2018 European recommendations commonly used PC-AKI [2,21]. Both terms describe a temporal association and do not, by themselves, establish that contrast caused the kidney injury.

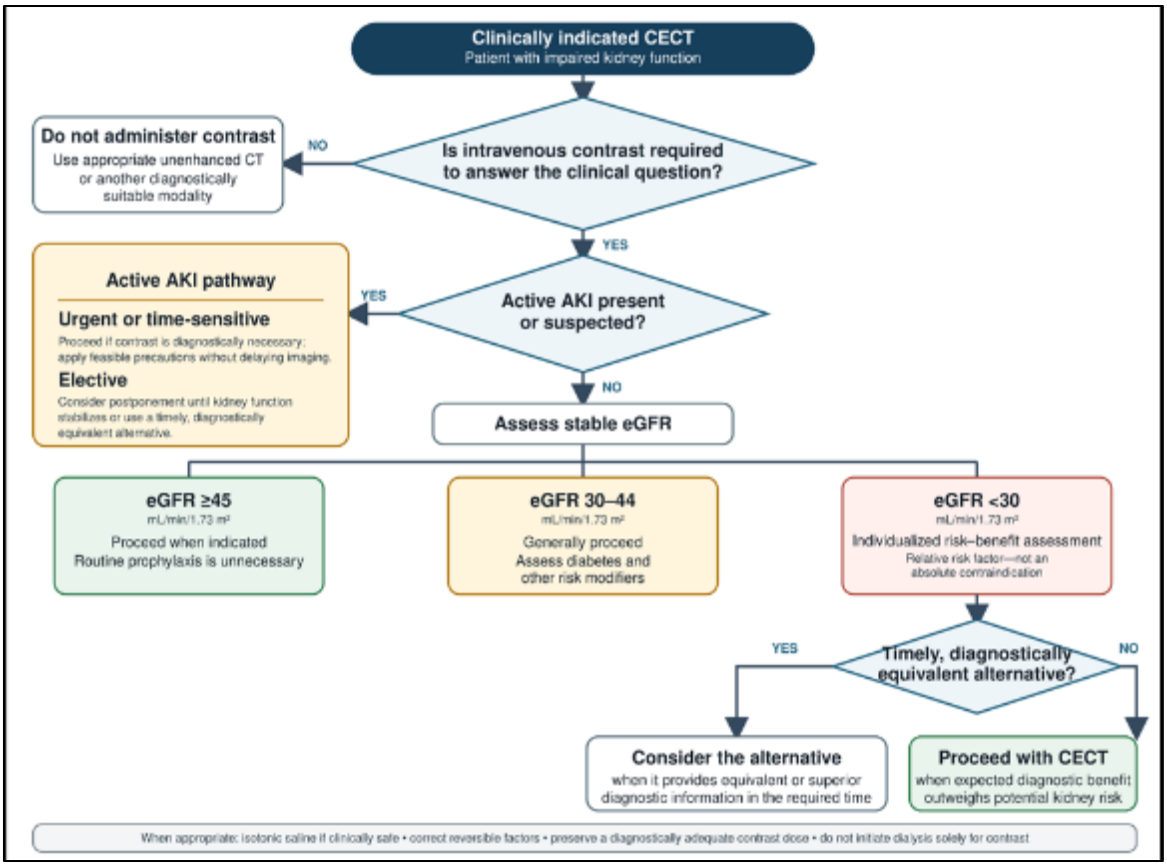

Figure 1: Practical decision-making algorithm for intravenous iodinated contrast administration in patients with impaired kidney function undergoing CECT.

3.9 Radiologist–clinician communication

Radiologist–clinician communication is most important in active AKI, stable eGFR <30 mL/min/1.73 m^2 or competing clinical risks. Discussion should establish the urgency and diagnostic question, whether contrast is essential, the stability of kidney function, reversible risk factors, feasible alternatives, hydration safety and how the result will alter management.

Formal consultation is most valuable when the diagnostic contribution of contrast is uncertain or when the potential kidney risk is substantial. In urgent conditions, communication should be prompt and should not create unnecessary delay. When CECT is essential for diagnosing active haemorrhage, vascular occlusion, bowel ischaemia, major trauma or another time-sensitive condition, the need for immediate diagnostic information may outweigh the uncertain risk of CI-AKI.

Routine consultation for every patient with mildly or moderately reduced eGFR is unlikely to improve safety and may delay appropriate imaging. Institutions should instead develop clear local protocols specifying when radiologist review is required, when hydration should be considered and when CECT may proceed without additional approval.

The final decision should be documented in the clinical or radiology record when active AKI, eGFR <30 mL/min/1.73 m^2 or another major concern necessitates individualized assessment. Documentation should briefly state the relevant kidney findings, expected diagnostic benefit, considered alternatives and agreed management plan. This supports continuity of care without treating contrast administration as an isolated decision made by either the radiologist or referring clinician alone.

3.10 Evidence gaps and future research directions

Despite substantial changes in the understanding of kidney injury following intravenous iodinated contrast administration, important uncertainties remain. Evidence is most reassuring for patients with preserved or moderately impaired stable kidney function. By contrast, patients with eGFR <30 mL/min/1.73 m^2 and those with active AKI—the groups of greatest clinical concern—remain comparatively underrepresented in controlled studies [1,5,11,14–16].

Most evidence is retrospective and therefore remains vulnerable to treatment-selection bias and unmeasured confounding. Severe CKD and active AKI are underrepresented, and creatinine-based AKI definitions may not reflect persistent injury or patient-important outcomes [1,5,11,14–16].

Future prospective, multi-centre studies should enrol adequate numbers of patients with stage 4–5 CKD and active AKI; document indication, contrast protocol, haemodynamics, hydration and concurrent nephrotoxins; and evaluate persistent kidney dysfunction, dialysis, hospital stay, mortality, diagnostic yield and harm from delayed imaging.

Research should also prioritize patient-important outcomes rather than relying predominantly on small, transient changes in serum creatinine. Relevant outcomes include persistent loss of kidney function, requirement for renal replacement therapy, prolonged hospitalization, treatment delay, cardiovascular complications and mortality. Studies should also evaluate the morbidity associated with withholding contrast, including missed diagnoses, repeat imaging, delayed intervention and inappropriate treatment.

The role of prophylactic hydration in severe CKD remains uncertain. The AMACING trial supports omission of routine hydration in selected patients with eGFR 30–59 mL/min/1.73 m^2 but excluded patients with eGFR <30 mL/min/1.73 m^2 [19]. Further trials should determine which high-risk patients benefit from hydration, the most effective regimen and how kidney protection can be balanced against pulmonary oedema and other complications of volume expansion.

Research should determine whether contrast changes clinically important outcomes and whether withholding it produces diagnostic or therapeutic harm.

4 CONCLUSION

The risk of kidney injury attributable to modern intravenous iodinated contrast media has historically been overestimated because AKI occurring after contrast administration was frequently assumed to be caused by contrast. Current controlled evidence indicates that this additional risk is minimal in patients with stable eGFR ≥45 mL/min/1.73 m^2 and remains low in most patients with eGFR 30–44 mL/min/1.73 m^2. Greater uncertainty persists in patients with eGFR <30 mL/min/1.73 m^2 and those with active AKI [1,11,14–16].

Impaired kidney function alone should not determine whether clinically necessary CECT is performed. Patients with stable eGFR ≥30 mL/min/1.73 m^2 can generally receive intravenous contrast without routine prophylactic hydration. An eGFR <30 mL/min/1.73 m^2 or active AKI should prompt individualized assessment of clinical urgency, expected diagnostic benefit, concurrent causes of kidney injury, volume status and the availability of a timely, diagnostically equivalent alternative. Neither condition should be treated as an absolute contraindication when contrast enhancement is necessary to guide diagnosis or treatment [1,22,23].

Preventive measures should be selective and proportionate to the patient's risk. Isotonic intravenous saline may be considered in severe CKD or active AKI when volume expansion is clinically safe. Routine N-acetylcysteine, prophylactic dialysis and arbitrary reduction of the contrast dose below a diagnostically adequate level are not supported. The final decision should balance the possibility of kidney injury against the potentially greater harm of delayed, missed or incomplete diagnosis.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

The authors declare that they have no financial or non-financial conflicts of interest related to this work.

Author contributions

- Mohammed Danyaro Rilwanu: Conceptualization, literature review, methodology, evidence synthesis, manuscript drafting and critical revision.
- Muhammad Abdullahi Jega: Literature review, interpretation of clinical evidence, validation and critical revision of the manuscript.
- Samaila Buhari: Review of contrast-media and CT protocol considerations, technical interpretation and critical revision of the manuscript.

All authors reviewed and approved the final version of the manuscript and accept responsibility for its content.

Data availability

No new dataset was generated or analyzed for this narrative review. All evidence discussed in the article was obtained from published sources cited in the manuscript.

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