Radiology

ORIGINAL RESEARCH • STATEMENTS AND GUIDELINES

Use of Intravenous Iodinated Contrast Media in Patients with Kidney Disease: Consensus Statements from the American College of Radiology and the National Kidney Foundation

Matthew S. Davenport, MD • Mark A. Perazella, MD • Jerry Yee, MD • Jonathan R. Dillman, MD, MS • Derek Fine, MD • Robert J. McDonald, MD, PhD • Roger A. Rodby, MD • Carolyn L. Wang, MD • Jeffrey C. Weinreb, MD

From the Departments of Radiology (M.S.D.) and Urology (M.S.D.), Michigan Medicine, 1500 E Medical Center Dr, B2-A209P, Ann Arbor, Mich 48109; Michigan Radiology Quality Collaborative, Ann Arbor, Mich (M.S.D.); American College of Radiology, Reston, Va (M.S.D., J.R.D., R.J.M., C.L.W., J.C.W.); National Kidney Foundation, New York, NY (M.A.P., D.F., R.A.R.); Section of Nephrology (M.A.P., J.C.W.) and Department of Radiology and Biomedical Imaging (J.C.W.), Yale University School of Medicine, New Haven, Conn; Department of Nephrology, Henry Ford Health System, Detroit, Mich (J.Y.); Department of Radiology, Cincinnati Children's Hospital Medical Center at University of Cincinnati College of Medicine, Cincinnati, Ohio (J.R.D.); Department of Nephrology, Johns Hopkins Medicine, Baltimore, Md (D.F.); Department of Radiology Mayo Clinic, Rochester, Minn (R.J.M.); Department of Nephrology, Rush University Medical Center, Chicago, Ill (R.A.R.); and Department of Radiology, University of Washington, Seattle, Wash (C.L.W.). Received September 13, 2019; revision requested October 8; final revision received October 28; accepted November 7. **Address correspondence to** M.S.D. (e-mail: *matdaven@med.umich.edu*).

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Intravenous iodinated contrast media are commonly used with CT to evaluate disease and to determine treatment response. The risk of acute kidney injury (AKI) developing in patients with reduced kidney function following exposure to intravenous iodinated contrast media has been overstated. This is due primarily to historic lack of control groups sufficient to separate contrast-induced AKI (CI-AKI; ie, AKI caused by contrast media administration) from contrast-associated AKI (CA-AKI; ie, AKI coincident to contrast media administration). Although the true risk of CI-AKI remains uncertain for patients with severe kidney disease, prophylaxis with intravenous normal saline is indicated for patients who have AKI or an estimated glomerular filtration rate less than 30 mL/min/1.73 m^2 who are not undergoing maintenance dialysis. In individual high-risk circumstances, prophylaxis may be considered in patients with an estimated glomerular filtration rate of 30–44 mL/min/1.73 m^2 at the discretion of the ordering clinician.

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Introduction and Background

Intravenous iodinated contrast media are commonly used with CT to evaluate disease and to determine treatment response. Although patients have benefited from their use, iodinated contrast media historically have been denied or delayed in patients with reduced kidney function due to the perceived risks of contrast-induced acute kidney injury (CI-AKI) (1,2). This is important because denying patients diagnostic testing that is indicated in a timely fashion creates potential for indirect harm related to delayed diagnosis and misdiagnosis (3).

Because of the critical role contrast media play in modern medical imaging, clinicians and diagnostic radiologists are routinely charged with balancing the potential risks of contrast media administration with diagnostic benefits (1,2). However, clinical decision making in patients potentially at risk for acute kidney injury (AKI) is often fraught with confusion, uncertainty, and heterogeneity. This is due in part to shifting perceptions regarding the true risks of modern contrast media (1–6), improvements in scientific methodology used to study these adverse events (7–13), incomplete penetrance of new knowledge into scientific practice (14), latent bias related to historical precedent (1,2), uncertainty regarding the interpretation of recently conducted well-controlled observational studies (4–6), and differences in recommendations across radiology and medical subspecialties (15–18).

In this document, joint statements are made by a multidisciplinary group of radiologists (M.D., R.M., J.W., J.D., C.W.) and nephrologists (M.P., J.Y., R.R., D.F.). These statements are endorsed by the American College of Radiology, or ACR, and the National Kidney Foundation, or NKF, to improve and standardize the care of patients with impaired kidney function who have indication(s) to receive intravenous iodinated contrast media. These opinions and recommendations are only applicable to intravenous (eg, contrast material–enhanced CT) as opposed to intra-arterial (eg, coronary artery angiography) contrast media administration, because intra-arterial administration has unique considerations that do not apply to the intravenous route of administration (eg, requirement for arterial access, atheroembolic complications, population-specific risk factors for AKI) (19).

It is important to recognize that in clinical practice, a multitude of factors are used to determine whether intravenous contrast media should be administered (eg,

Abbreviations

AKI = acute kidney injury, CA-AKI = contrast-associated AKI, CI-AKI = contrast-induced AKI, CKD = chronic kidney disease, eGFR = estimated glomerular filtration rate, IOCM = iso-osmolality contrast media, LOCM = low-osmolality contrast media, PC-AKI = postcontrast AKI

Summary

These statements are endorsed by the American College of Radiology and the National Kidney Foundation to improve and standardize the care of patients with impaired kidney function who have indication(s) to receive intravenous iodinated contrast media.

probability and necessity of an accurate diagnosis, alternative methods of diagnosis, risks of misdiagnosis, expectations about kidney functional recovery, allergic-like reaction risk). Decisions are rarely based on a single consideration (eg, risk of an adverse event specifically related to kidney impairment). Consequently, these statements should be considered in the context of the entire clinical scenario.

Nomenclature and Definitions

AKI (Fig 1) occurring within 48 hours of intravenous contrast media administration and after the exclusion of other nephrotoxic factors has been termed *contrast-induced nephropathy* (2). This definition is problematic because in most cases, "exclusion of other nephrotoxic factors" is not feasible or reliable (2). Consequently, many cases of AKI that are coincident with but causally unrelated to intravenous contrast media administration have been incorrectly attributed as contrast induced, thereby overstating the risks of intravenous contrast media administration (1–13,15). The vast majority of studies on this topic have not included a control group of similar patients who were not exposed to contrast media, making it impossible to separate causal from coincident AKI (20). However, recent (2013–present) large, well-controlled, observational series have shown that a substantial proportion of AKI that occurs after intravenous contrast media administration is not attributable to contrast media (7,8,10,11,13).

Therefore, in 2015, the ACR Committee on Drugs and Contrast Media adopted new terms that were intended to disentangle the implicit causal relationship between contrast media and AKI (15). The following terms are endorsed in these consensus statements:

Contrast-associated acute kidney injury (CA-AKI): Any AKI occurring within 48 hours after the administration of contrast media. The term *postcontrast acute kidney injury* (PC-AKI) is synonymous with CA-AKI and appears in radiology guidelines (15). Both terms imply correlative diagnosis. Neither term implies a causal relationship between contrast medium administration and an AKI event. Related AKI events that occur in clinical care and events documented in study protocols that lack a control cohort are best termed CA-AKI or PC-AKI.

Contrast-induced acute kidney injury (CI-AKI): CI-AKI is the subset of CA-AKI that can be causally linked to contrast media administration. CI-AKI implies a causal relationship between intravenous contrast media and the development of AKI (ie, contrast induced). Use of the term CI-AKI (formerly termed contrast-induced nephropathy) in clinical practice can be misleading because of the large fraction of false-positive events (ie, AKI related to concurrent nephrotoxic exposure or insults in proximity to the time of contrast media administration). Only studies with a well-matched control group can establish a potential causal relationship.

KDIGO AKI Staging			
Stage	Serum Creatinine Criteria		
1	1.5–1.9 times baseline serum creatinine		
	OR		
	Increase in serum Cr ≥ 0.3 mg/dL (≥26.5 μmol/l)		
2	2.0–2.9 times baseline serum creatinine		
3	3.0 times baseline serum creatinine		
	OR		
	Increase in serum Cr to ≥ 4.0 mg/dL (≥353.6 μmol/l)		
	OR		
	Initiation of kidney replacement therapy		
	OR		
	Decrease in eGFR to < 35 mL/min/1.73 m^2 (for patients < 18 years old)		
KDIGO CKD Staging			
Stage	GFR Criteria		
G1	≥ 90 mL/min/1.73 m^2 with markers of kidney damage		
G2	60–89 mL/min/1.73 m^2 with markers of kidney damage		
G3a	45–59 mL/min/1.73 m^2		
G3b	30–44 mL/min/1.73 m^2		
G4	15–29 mL/min/1.73 m^2		
G5	< 15 mL/min/1.73 m^2		
Stage	Albuminuria Criteria		
	AER	ACR (approximate equivalent)	
	(mg/24 hours)	(mg/mmol)	(mg/g)
A1	<30	< 3	<30
A2	30–300	3–30	30–300
A3	> 300	> 30	>300

Figure 1: Image shows Kidney Disease Improving Global Outcomes (KDIGO) staging criteria for acute kidney injury (AKI) and chronic kidney disease (CKD). Urine output criteria for AKI staging were not included. ACR = albumin creatinine ratio, AER = albumin excretion rate, Cr = creatinine, GFR = glomerular filtration rate.

Diagnosis of CI-AKI and Chronic Kidney Disease

Updated Kidney Disease

Kidney Disease Improving Global Outcomes, or KDIGO, criteria are recommended for the diagnosis of CA-AKI and, when feasible, in the context of a controlled study, for the diagnosis of CI-AKI (17,21,22). KDIGO criteria are endorsed by the NKF Kidney Disease Outcomes Quality Initiative as a consensus definition for epidemiologic and clinical research applications (17). Although serum creatinine is an imperfect biomarker for AKI, it remains the most common and practical clinical method of diagnosing AKI. Reduced urine output is another criterion to diagnose AKI by using KDIGO criteria, but change in urine output can be more challenging to as-

sess in retrospective studies because it is not always rigorously documented (7,8,10,11,13).

KDIGO criteria are recommended for the diagnosis of chronic kidney disease (CKD) (Fig 1) and are endorsed by NKF Kidney Disease Outcomes Quality Initiative (23,24). Although the estimated glomerular filtration rate (eGFR) has intrinsic error due to reliance on serum creatinine and lack of validation in patients with AKI, low muscle mass, or in patients treated with dialysis (23–25), eGFR is the most accurate and least biased method commonly available in clinical practice to stratify KDIGO CKD stage by using glomerular filtration rate ("G" based on eGFR) (Fig 1).

Because of the historical conflation of CA-AKI and CI-AKI, many relevant published studies that have used CA-AKI as an outcome have inaccurately labeled it CI-AKI (ie, contrast-induced nephropathy) (2,20). Therefore, while there is ample evidence for the existence of CA-AKI, the evidence base for CI-AKI is sparse (2,3,13,20). Several topics in this document are addressed from the perspective of generic CA-AKI due to an insufficient evidence base for CI-AKI. Where feasible, specific commentary about CI-AKI is made. These statements enable providers to make judgments in specific circumstances.

Key Questions and Joint NKF-ACR Statements

What Is the Risk of CA-AKI and CI-AKI in Patients Who Have eGFR Less Than 30, 30–44, 45–59, and Greater Than or Equal to 60 mL/Min/1.73 m^2 Undergoing Contrast-enhanced CT?

Contrast-associated AKI.—The risk of CA-AKI (coincident AKI of any cause) increases with each stepwise increase in CKD stage (7–11,13,15). Using stage I KDIGO serum creatinine criteria, the risk of CA-AKI is approximately 5% at eGFR greater than or equal to 60, 10% at eGFR of 45–59, 15% at eGFR of 30–44, and 30% at eGFR less than 30 mL/min/1.73 m^2. This risk is much higher than the risk of CI-AKI because it includes any AKI coincident to contrast media administration, regardless of contrast media exposure.

Contrast-induced AKI.—The risk of CI-AKI is substantially less than the risk of CA-AKI, but the actual risk remains uncertain in patients with severe kidney disease. Several large controlled observational studies have shown no evidence of CI-AKI regardless of CKD stage (10,11,13), whereas others found evidence of CI-AKI only in patients with severely reduced kidney function (7,8,13). In such studies, the risk of CI-AKI has been estimated to be near 0% at eGFR greater than or equal to 45, 0%–2% at eGFR of 30–44, and 0%–17% at eGFR less than 30 mL/min/1.73 m^2. These studies (1–8,10,11,13) are underpowered to establish risk in patients with severe kidney disease, differ in their conclusions about risk in patients with eGFR less than 30 mL/min/1.73 m^2 (estimated CI-AKI risk range, 0%–17%), and are observational in design (ie, only known confounders can be addressed). There are no randomized trials differentiating CA-AKI from CI-AKI in patients with eGFR less than 30 mL/min/1.73 m^2.

What Other Major Patient-related Factors Increase the Risk of CA-AKI or CI-AKI?

Contrast-associated AKI.—Multiple patient-related risk factors have been associated with CA-AKI (15–17,26). The primary risk factor is eGFR, with some studies finding an additive risk of CA-AKI from diabetes mellitus (15–17,26). Additional risk factors include nephrotoxic agents and exposures, hypotension, hypovolemia, albuminuria, and impaired kidney perfusion (eg, congestive heart failure [27]). Although multiple myeloma has long been considered a risk factor for CA-AKI, this is not supported by more recent literature (28–30).

Contrast-induced AKI.—Few studies have linked patient-related risk factors with CI-AKI. In studies that found evidence of CI-AKI, the primary risk factor was eGFR (7–11,13,15). No other putative risk factors that increase CI-AKI risk beyond eGFR alone have been confirmed in well-controlled studies of intravenous media.

Are There Clinically Relevant Differences in CA-AKI and CI-AKI Risk for Patients with Reduced Kidney Function with Intravenous Iodinated Low-Osmolality Contrast Media Compared with Intravenous Iodinated Iso-Osmolality Contrast Media?

Contrast-associated AKI.—There are no confirmed clinically relevant differences in risk of CA-AKI between low-osmolality contrast media (LOCM) and iso-osmolarity contrast media (IOCM) for intravenous applications (31). Indirect evidence suggests that the LOCM iohexol may have a higher risk compared with other LOCM, but that potential risk difference has not been confirmed (31). Randomized studies comparing LOCM and IOCM primarily analyzed intra-arterial administrations and have mixed results (31). Based on results of a 2015 systematic review and meta-analysis, any difference in risk of CA-AKI between LOCM and IOCM is not likely to be clinically meaningful (31).

Contrast-induced AKI.—No studies have directly compared risk of CI-AKI between LOCM and IOCM. However, randomized trials comparing contrast media using CA-AKI as an outcome (31) inform the risk of CI-AKI because, other than the contrast media exposure, the groups are balanced (ie, the outcome is a combination of CA-AKI unrelated to contrast media and CI-AKI, with the primary difference being the CI-AKI fraction). There is thought to be no clinically relevant difference in risk of CI-AKI between LOCM and IOCM (31).

Despite the acronym, LOCM are hyperosmolar (approximately 600 mOsm/kg) relative to both IOCM (approximately 290 mOsm/kg) and serum (approximately 290 mOsm/kg) (15). However, the dimeric structure of IOCM renders them more viscous than LOCM (15). Most modern iodinated contrast media are classified as LOCM (15). High-osmolality iodinated contrast media have higher osmolality

than do LOCM and IOCM, but high-osmolality iodinated contrast media has been replaced by LOCM and IOCM for intravenous administration in modern clinical practice (15).

Which Patients Should Undergo Prophylaxis to Prevent AKI prior to Intravenous Iodinated Contrast Media Administration?

Prophylaxis is indicated for patients who have AKI or an eGFR less than 30 mL/min/1.73 m^2 and are not undergoing maintenance dialysis (15,26,32,33). However, the evidence supporting this statement is based on data for the general prevention of CA-AKI rather than CI-AKI specifically. The risks of prophylaxis (eg, heart failure, other hypervolemic conditions) should be considered before initiation (34,35). Prophylaxis is not indicated for the general population of patients with stable eGFR greater than or equal to 30 mL/min/1.73 m^2 (35), for patients undergoing chronic dialysis, or for patients at risk for heart failure (34,35). This eGFR threshold should not be adjusted solely based on concomitant diabetes mellitus (7–11,13,15,36). In a 1:1 propensity-matched observational study of 1112 patients with stable eGFR of 30–44 mL/min/1.73 m^2, diabetes mellitus did not independently increase risk of CI-AKI in patients undergoing contrast-enhanced CT (P = .22) (31).

In individual high-risk circumstances (eg, numerous risk factors, recent AKI, borderline eGFR), prophylaxis may be considered in patients with eGFR of 30–44 mL/min/1.73 m^2 at the discretion of the ordering clinician. If a contrast-enhanced imaging study that otherwise would be preceded by prophylaxis has an emergent indication and there is insufficient time for preprocedural prophylaxis, then postprocedural prophylaxis may be considered, but there is no evidence to support this action.

When prophylaxis is indicated, isotonic volume expansion with normal saline is the preferred method (15,26,32,33). The ideal timing, volume, and rate of volume expansion is uncertain. Typical volume expansion regimens begin 1 hour before and continue 3–12 hours after contrast media administration, with typical doses ranging from fixed (eg, 500 mL before and after) to weight-based volumes (1–3 mL/kg per hour) (15,26). Longer regimens (approximately 12 hours) have been shown to lower the risk of CA-AKI compared with shorter regimens (15,37). However, longer intravenous protocols are generally impractical in the outpatient setting. Oral hydration has not been well studied for patients with eGFR less than 30 mL/min/1.73 m^2 or AKI (38,39).

Although bicarbonate is likely similar to normal saline for the prevention of CA-AKI (40), it is not preferred because bicarbonate solutions require pharmacist compounding. *N*-acetylcysteine was not shown to be effective versus placebo in a recent randomized trial of intra-arterial iodinated contrast media administration (40) and is not recommended for intravenous contrast media exposure prophylaxis. In patients with eGFR less than 30 mL/min/1.73 m^2 or AKI, cessation of nonessential nephrotoxic medications (eg, nonsteroidal anti-inflammatory drugs) may decrease the risk of CA-AKI and is recommended when feasible.

Should Screening Be Used to Identify Patients at Risk for CI-AKI?

Screening based on eGFR should be used to identify patients at potential risk of CI-AKI (15,16). Screening based on eGFR is preferred over serum creatinine–based screening (17,23,24,41). Ideally, serum creatinine measurements should undergo calibration traceable to isotope dilution mass spectroscopy (42). Following accurate calibration, eGFR should be calculated with a validated isotope dilution mass spectroscopy–traceable equation (eg, chronic kidney disease epidemiology collaboration, Modification of Diet in Renal Disease) (42).

What patient risk factors should be used to trigger eGFR measurement?—A variety of screening data elements have been considered that variably affect the sensitivity and specificity of kidney function screening (15,43,44). A personal history of kidney disease (eg, CKD, remote AKI, kidney surgery, kidney ablation, albuminuria) is the most useful element that demonstrates a requirement for kidney function determination (15,43,44). Diabetes mellitus is an optional factor for screening (43,44). Patient age and both treated and untreated hypertension are of uncertain utility as independent triggers for kidney function assessment during radiology point of care; they are sensitive indicators and confer a large false-positive rate to the identification of patients with eGFR less than 30 mL/min/1.73 m^2.

What eGFR threshold should be used by radiologists to trigger consultation with the referring clinician prior to intravenous administration of contrast media?—Patients with AKI or eGFR less than 30 mL/min/1.73 m^2 (including nonanuric patients undergoing maintenance dialysis [see below]) should prompt consideration by the referring professional and radiologist to discuss the risks and benefits of contrast media administration (15,16). Because of a lack of data supporting an additive risk of CI-AKI beyond CKD stage, the eGFR threshold does not need to be modified based on chronic diseases such as diabetes mellitus (15,16). Acute clinical risk factors (eg, volume depletion) without known AKI are beyond the scope of radiology practices to determine and are left to referring clinicians to evaluate. In general, when stable, eGFR is the best indicator of a patient's potential risk of CI-AKI (15,16).

Should Intravenous Iodinated Contrast Media Be Withheld in Patients with CKD Stages 4 or 5 Not Undergoing Maintenance Hemodialysis?

Patients with CKD stages 4 or 5 (eGFRs of 15–29 mL/min/1.73 m^2 or <15 mL/min/1.73 m^2, respectively) who are not undergoing maintenance hemodialysis are at potential risk of CI-AKI (7,8,13,15,16,26). The number needed to harm from contrast media administration (ie, one patient developing CI-AKI after x exposed patients) has been calculated in well-controlled observational studies to be as low as six and as high as infinity (ie, no harm) (1,2). Patients with CKD stages 4 or 5 have a relative rather than absolute contraindication to iodinated contrast media (15,16). If contrast media administration is required for a life-threatening diag-

nosis, then it should not be withheld based on kidney function (15). If intravenous iodinated contrast media administration is clinically indicated, then its use should be informed by consideration of the potential risks and benefits as well as alternative imaging strategies. If the decision is made to administer iodinated contrast media in this setting, then volume expansion with normal saline is indicated if there are no contraindications (see above).

If intravenous iodinated contrast media is administered in this setting, then should the patient undergo dialysis in addition to standard prophylaxis?—Because of the inherent demonstrated lack of benefit, risks, and cost, neither acute dialysis nor continuous renal replacement therapy should be initiated or have the schedule changed solely based on iodinated contrast media administration, regardless of residual kidney function (15,17,26,32,44–47).

Do Patients Undergoing Maintenance Dialysis with Residual Kidney Function Require Different Treatment than Do Those without Residual Kidney Function?

From an operational standpoint, patients undergoing dialysis who make more than 1–2 cups of urine daily (approximately 100 mL) can be considered nonanuric (17). Nonanuric patients undergoing maintenance dialysis, whether peritoneal dialysis or hemodialysis, are at increased risk of further loss of residual kidney function following nephrotoxic exposure(s). Although unproven for intravenous iodinated contrast media, loss of residual kidney function may have adverse quality-of-life and overall survival implications. Therefore, nonanuric patients with residual kidney function undergoing maintenance dialysis are considered similar to patients with AKI or eGFRs less than 30 mL/min/1.73 m^2 not undergoing dialysis with respect to the potential nephrotoxic risk of iodinated contrast media (ie, relative contraindication). If loss of residual kidney function is considered clinically important, then the risks, benefits, and alternatives should be considered, and the need for the procedure may require discussion between the referring professional and radiologist.

Are Patients with a Single Kidney at Increased Risk for CA-AKI or CI-AKI Beyond That Associated with Their eGFR?

Patients with a single normal or partially functioning kidney (eg, kidney agenesis, nephrectomy, transplant) should be managed similarly to patients with normal kidney volume (eg, two normal kidneys) (15,48). In patients with a single normal or partially functioning kidney, clinical risk should be determined based on overall kidney function (ie, eGFR) and clinical circumstances (ie, AKI). The presence of a solitary functioning kidney should not influence decision making regarding the risk of CA-AKI or CI-AKI (15,48).

If a Patient Is Determined to Be at Risk for CI-AKI, Then Should the Dose of Iodinated Contrast Media with Contrast-enhanced CT Be Reduced?

Although correlative data link higher doses of contrast media to greater risk of CA-AKI following intra-arterial administration, no analogous data exist that imply a dose-ranging toxicity for intravenous administration within the range of clinically administered doses. Consequently, if iodinated contrast media is administered to a patient at risk, then a conventional single diagnostic dose should be used (ie, volume typically used for a single diagnostic dose). Ad hoc contrast media dose reductions as an effort to mitigate risk of CI-AKI should be avoided because this practice may produce a suboptimal or nondiagnostic study. If lower doses of contrast media have been shown to be sufficiently diagnostic with specific protocols, then practices should consider lowering doses in all patients imaged with those protocols, not only patients with reduced kidney function.

Should Any of the Above Recommendations Be Altered in Patients Receiving Certain Nephrotoxic Medications or Undergoing Chemotherapy, Especially If They Have Normal Kidney Function?

In general, recommendations 1–9 should not be altered in patients receiving nephrotoxic medications or undergoing chemotherapy. This is especially true for patients who have normal eGFR or mild-to-moderate reductions in eGFR because they are not considered at risk, regardless of the drug(s) prescribed (13,15). However, monitoring eGFR in patients receiving nephrotoxic medications (eg, aminoglycosides) or undergoing chemotherapy is important before, during, and after treatment to identify incident nephrotoxicity (eg, AKI or new eGFR <30 mL/min/1.73 m^2) (49).

Is there a role for withholding certain medications prior to intravenous iodinated contrast media administration to decrease the risk of kidney injury?—In patients with AKI or eGFRs less than 30 mL/min/1.73 m^2, it may be prudent to withhold nonessential potentially nephrotoxic medications (eg, nonsteroidal anti-inflammatory drugs, diuretics, aminoglycosides, amphotericin, platins, zoledronate, methotrexate) if clinically feasible for 24 to 48 hours before and 48 hours after exposure (17,21,26).

Whether to withhold renin-angiotensin-aldosterone system inhibitors, or RAASi, is controversial. Conflicting data support increased risk for CA-AKI, no risk for CA-AKI, and potentially less risk of CA-AKI with RAASi (50–56). Many of these publications are small studies that include different routes of contrast media administration (venous vs arterial), variable definitions of CA-AKI, and inconsistent prophylactic measures. In addition, the effect on long-term kidney function is insufficiently studied. A meta-analysis of 12 studies and 4493 patients found no difference in risk of CA-AKI (odds ratio: 1.27; 95% confidence interval: 0.77, 2.11; $P = .35$) between patients receiving and patients not receiving RAASi (57). However, in stratified analysis, there was an increased risk for CA-AKI (odds ratio: 2.06; 95% confidence interval: 1.62, 2.61; $P < .001$) for chronic RAASi users who did not withhold the drug. This relationship was not present in patients new to RAASi (57). Given the lack of strong evidence demonstrating that continuing RAASi is beneficial, referring clinicians should consider withholding RAASi in patients at risk for at least 48 hours before elective contrast-enhanced

Table: Summary of Major ACR-NKF Consensus Statements on Use of Intravenous Iodinated Contrast Media in Patients with Kidney Disease, with Comparison to ACR (2018) and KDIGO (2012) Guidelines for CI-AKI

Summary
1. The terms CA-AKI or PC-AKI are recommended for use in clinical practice due to the large proportion of AKI events correlated with but not necessarily caused by contrast media administration.
a. ACR: Similar recommendation to distinguish generic PC-AKI from CI-AKI
b. KDIGO: No recommendation regarding terminology, although it is acknowledged that AKI may be caused by other things
2. CI-AKI is only feasible to diagnose in the context of a well-matched controlled study.
a. ACR: Not specifically addressed
b. KDIGO: Not specifically addressed
3. KDIGO AKI criteria are recommended for the diagnosis of AKI, and KDIGO CKD criteria are recommended for the diagnosis of CKD.
a. ACR: AKIN criteria recommended
b. KDIGO: KDIGO criteria recommended
4. The risk of CI-AKI from intravenous iodinated contrast media is lower than previously thought. Necessary contrast material–enhanced CT without a suitable alternative should not be avoided solely on the basis of CI-AKI risk.
a. ACR: Similar recommendation
b. KDIGO: Similar recommendation
5. CI-AKI risk should be determined primarily by using CKD stage and AKI. Patients at high risk include those with recent AKI and those with eGFR less than 30 mL/min/1.73 m^2, including nonanuric patients undergoing maintenance dialysis.
a. ACR: Similar recommendation
b. KDIGO: Similar recommendation, but eGFR threshold is less than 45 mL/min/1.73 m^2 instead of less than 30 mL/min/1.73 m^2
6. Kidney function screening is indicated to identify patients at high risk for CI-AKI. Personal history of kidney disease (CKD, remote AKI, kidney surgery or ablation) is the strongest risk factor indicating the need for kidney function assessment.
a. ACR: Similar recommendation, but also includes age, diabetes mellitus, and hypertension as potential risk factors to indicate kidney function assessment
b. KDIGO: Similar recommendation, but also includes age, diabetes mellitus, hypertension, multiple myeloma, gout, and proteinuria as potential risk factors to indicate kidney function assessment
7. Radiologist-clinician discussions about risks and benefits of contrast-enhanced imaging can be helpful in patients at high risk for CI-AKI.
a. ACR: Not specifically addressed
b. KDIGO: Not specifically addressed
8. There are no clinically relevant differences in CI-AKI risk between iso-osmolality and low-osmolality iodinated contrast media.
a. ACR: Similar recommendation
b. KDIGO: Similar recommendation
9. Prophylaxis with intravenous normal saline is indicated for patients not undergoing dialysis who have eGFR less than 30 mL/min/1.73 m^2 or AKI. In individual high-risk circumstances, prophylaxis may be considered in patients with eGFR of 30–44 mL/min/1.73 m^2 at the discretion of the ordering clinician.
a. ACR: Prophylaxis with normal saline recommended for patients not undergoing dialysis with eGFR less than 30 mL/min/1.73 m^2; no exception for patients with eGFR of 30–44 mL/min/1.73 m^2 and multiple risk factors
b. KDIGO: Prophylaxis with normal saline or sodium bicarbonate recommended for patients not undergoing dialysis with eGFR less than 45 mL/min/1.73 m^2; prophylaxis may include *N*-acetylcysteine
10. Prophylaxis is not indicated for patients with stable eGFR greater than or equal to 45 mL/min/1.73 m^2.
a. ACR: Prophylaxis not recommended for patients with eGFR greater than or equal to 30 mL/min/1.73 m^2
b. KDIGO: Necessity of prophylaxis is ambiguous for patients with eGFR of 45–59 mL/min/1.73 m^2
11. Kidney replacement therapy should not be initiated or have the schedule adjusted solely on the basis of contrast media administration.
a. ACR: Similar recommendation
b. KDIGO: Similar recommendation
12. The presence of a solitary kidney should not independently influence decision making regarding the risk of CI-AKI.
a. ACR: Not specifically addressed
b. KDIGO: Not specifically addressed
13. In patients at high risk of CI-AKI, ad hoc lowering of contrast media dose below a known diagnostic threshold should be avoided. Rather, the minimum routine clinical diagnostic dose should be used.
a. ACR: Not specifically addressed
b. KDIGO: Contrast media dose reduction recommended
14. When feasible, nephrotoxic medications should be withheld by the referring clinician in patients at high risk.
a. ACR: Not specifically addressed
b. KDIGO: Similar recommendation

Table *(continues)*

Table *(continued)*: Summary of Major ACR-NKF Consensus Statements on Use of Intravenous Iodinated Contrast Media in Patients with Kidney Disease, with Comparison to ACR (2018) and KDIGO (2012) Guidelines for CI-AKI

15. Data on risk of CI-AKI in pediatric patients is extrapolated from data in adult patients. Pediatric-specific research in this area is a major unmet need.
a. ACR: Similar recommendation
b. KDIGO: Not specifically addressed

Note.—ACR = American College of Radiology, AKI = acute kidney injury, AKIN = Acute Kidney Injury Network, CA-AKI = contrast-associated AKI, CI-AKI = contrast-induced AKI, CKD = chronic kidney disease, eGFR = estimated glomerular filtration rate, KDIGO = Kidney Disease Improving Global Outcomes, NKF = National Kidney Foundation, PC-AKI = postcontrast acute AKI.
Source.—References 15, 17, 21.

CT to avoid the potential for hypotension and hyperkalemia should CA-AKI develop. RAASi may be restarted if CA-AKI does not occur or following the return of kidney function to baseline.

Metformin does not increase risk of CA-AKI. If CI-AKI develops in a patient receiving metformin, then the risk of lactic acidosis is increased. Therefore, metformin should be withheld in patients with AKI or eGFR less than 30 mL/min/1.73 m^2, consistent with U.S. Food and Drug Administration recommendations to generally avoid metformin at this level of kidney function. Although the FDA also recommends withholding metformin prior to iodinated contrast media exposure for eGFR 30–59 mL/min/1.73 m^2 (58), decision making should be individualized by referring clinicians at this eGFR level because the risk of CI-AKI is sufficiently low.

If CA-AKI develops, then nonessential nephrotoxic medications should continue to be withheld until kidney function has recovered (17,21,26). In some cases, withholding a nephrotoxic drug or the delay of an indicated imaging examination while waiting for an administered nephrotoxic drug to be eliminated may carry more risk than the potential risk of CI-AKI. Therefore, decisions regarding the suspension of medications should be individualized by referring and other treating providers.

Should Any of the Above Be Altered in Infants and Children?

Kidney function measurement in infants and children is optimally evaluated by the Bedside Schwartz equation rather than by eGFR equations developed and validated in adults (59–61). In general, the aforementioned recommendations should not be altered for infants and children, but there are minimal data assessing risk of CI-AKI in this population (62). Recommendations for this population are largely based on extrapolated adult data (15). The risk of CI-AKI from intravenous contrast media in infants and children is a pressing research need.

Summary

The putative risk of administering modern intravenous iodinated contrast media in patients with reduced kidney function has been overstated. This is primarily because of the conflation of contrast-associated acute kidney injury (CA-AKI) with contrast-induced acute kidney injury (CI-AKI) in uncontrolled studies. Although the true risk of CI-AKI remains unknown, prophylaxis with intravenous normal saline is indicated for patients without contraindication (eg, heart failure) who have acute kidney injury (AKI) or an estimated glomerular filtration rate (eGFR) less than 30 mL/min/1.73 m^2 who are not undergoing maintenance dialysis. In individual high-risk circumstances, prophylaxis may be considered in patients with an eGFR of 30–44 mL/min/1.73 m^2 at the discretion of the ordering clinician. The presence of a solitary kidney should not independently influence decision making regarding the risk of CI-AKI. Ad hoc lowering of contrast media dose below a known diagnostic threshold should be avoided due to the risk of lowering diagnostic accuracy. When feasible, nephrotoxic medications should be withheld by the referring clinician in patients at high risk. However, renal replacement therapy should not be initiated or altered solely based on contrast media administration. Prospective controlled data are needed in adult and pediatric populations to clarify the risk of CI-AKI. A summary of these recommendations with comparison to existing guidelines is provided in the Table.

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