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Comprehensive Review

Management of Patients With Kidney Disease in Need of Cardiovascular Catheterization: A Scientific Workshop Cosponsored by the National Kidney Foundation and the Society for Cardiovascular Angiography and Interventions

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ABSTRACT

Patients with chronic kidney disease (CKD) are at an increased risk of developing cardiovascular disease (CVD), whereas those with established CVD are at risk of incident or progressive CKD. Compared with individuals with normal or near normal kidney function, there are fewer data to guide the management of patients with CVD and CKD. As a joint effort between the National Kidney Foundation and the Society for Cardiovascular Angiography and Interventions, a workshop and subsequent review of the published literature was held. The present document summarizes the best practice recommendations of the working group and highlights areas for further investigation.

Introduction

The development of chronic kidney disease (CKD) and progression to end-stage kidney disease (ESKD) continue to be growing concerns worldwide. The complex interactions between CKD and cardiovascular disease (CVD) have gained increased attention over the past 2 decades. Patients with CKD are at risk of developing CVD, whereas those with established CVD are at risk of incident or progressive CKD.[1] Compared with individuals with normal or near normal kidney function, there are fewer data to guide the diagnosis, prevention, and management of patients with CVD and CKD. Less is known about how therapeutic interventions affect outcomes in this patient population. With the global

Abbreviations: AKI, acute kidney injury; CKD, chronic kidney disease; CV, cardiovascular; CVD, cardiovascular disease; PVI, peripheral vascular intervention; TAVR, transcatheter aortic valve replacement.

Keywords: acute kidney injury; cardiovascular procedures; chronic kidney disease.

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rise of cardiometabolic risk factors such as aging, obesity, diabetes, and hypertension, larger numbers of patients with CKD are now undergoing catheterization procedures for the treatment of coronary, peripheral arterial, and heart valve disease.[2] Furthermore, arterial disease in patients with CKD tends to be more complex, with a substantial burden of atherosclerosis, arteriosclerosis, and vascular calcification. Decision-making surrounding specific procedural indications and approaches in this patient group is an evolving area of study. A key focus area for quality improvement in patients undergoing cardiovascular (CV) catheterization remains minimization of the risk of acute kidney injury (AKI), which occurs at a higher rate in this patient group than in the general clinical population.[2-4]

Across the CV and nephrology society clinical guidelines, there has been limited focus on patients with CKD undergoing catheterization or percutaneous coronary interventions (PCIs) or peripheral vascular interventions (PVIs). To better understand the available evidence on this topic, highlight areas of uncertainty, and provide consensus recommendations for health care providers, the National Kidney Foundation and the Society for Cardiovascular Angiography and Interventions (SCAI) convened a workshop of experts in the field. The present paper summarizes the best practices for managing patients with kidney disease requiring CV catheterization procedures, focusing on AKI.

Methods

A scientific workshop sponsored by the National Kidney Foundation and SCAI was convened online on September 18, 2020, and September 25, 2020. Drs Anand Prasad, Paul Palevsky, and Steven Weisbord chaired the workshop. There were 41 participating faculty identified by the Chairs, spanning the disciplines of cardiology, nephrology, vascular medicine, epidemiology, and included patient representation (Appendix). The planning committee met monthly for over a year to design the program objectives. During the day 1 plenary session, speakers provided reviews of our current knowledge of epidemiology, pathophysiology, and recent research on preventing and managing AKI in patients with CKD needing catheterization procedures. Participants were divided into 3 workgroups that met twice over the next week, charged with addressing critical questions on coronary, valvular, and peripheral vascular topics. This was followed on day 2 with the breakout group discussion, when leaders summarized their groups' deliberations and recommendations, with feedback from the entire group. Next, the group leaders provided the didactic presentations, followed by moderated discussions among the expert faculty panel. Finally, the group leaders recorded the consensus opinions and areas of uncertainty in summary reports to the Chairs. In the months following workshop completion, additional references and new literature were examined and further discussed by the Chairs for potential incorporation into the final summary manuscript.

Background: The relationship between CKD and CVD

The association between CKD and CVD prevalence and mortality exists in a graded relation with the stage of CKD, including categories of estimated glomerular filtration rate (eGFR) and the level of albuminuria.[5-8] The transition from CKD to ESKD and subsequent need for kidney replacement therapy represents a critical threshold that heralds the onset of an even higher prevalence of atherosclerosis, arteriosclerosis, and incident CVD events.[9] The CVD manifestations in patients with CKD are varied and extend beyond atherosclerosis to include cardiomyopathies (often with ventricular hypertrophy), large vessel remodeling, arrhythmias, and valvular heart disease.[10,11] The ultimate cause of death in patients with ESKD is often the result of these latter disease processes.[11]

Chronic kidney disease stages have important implications for patient prognosis across CVD phenotypes. For example, lower eGFR is associated with a rising incidence of total CVD-related mortality and risk of acute coronary syndrome (ACS).[12] In addition, lower eGFR correlates with a higher prevalence of atypical chest pain and nondiagnostic electrocardiograms—both of which confound the management of these patients.[13-15] Lower extremity peripheral artery disease (PAD) lesions are more advanced in patients with CKD with more multilevel lesions, more extensive and severe calcification, and a higher risk of limb loss.[16] Aortic stenosis likewise progresses more rapidly among patients with impaired kidney function.[11,17,18]

Having established that CKD and CVD are related, it should be emphasized that risk factor modification and prevention remain the cornerstones of managing patients with these disease processes. However, catheterization procedures may be needed for many patients with CKD to prevent or attenuate the severity of CV events. Although understanding of the indications and relative benefits of interventional procedures in this cohort is evolving, minimizing AKI in this high-risk population is of paramount importance.

The importance of AKI following catheterization procedures

Acute kidney injury as a complication of CV catheterization has been well described in the literature for over half a century. The mechanism traditionally often invoked is contrast-mediated toxicity, which can be multifactorial in etiology and can include oxidative stress, renal tubular cell injury, and renal microcirculatory vasoconstriction.[19,20] However, given the complexity of modern catheterization procedures, factors such as hypovolemia, hypervolemia, cardiogenic shock/hypotension, atheroembolism, and nephrotoxic medication use may cause or contribute to AKI. A theoretical framework that remains under investigation proposes that episodes of AKI may reduce the renal functional reserve, and over time, repeated insults could result in reduced kidney function and progression of CKD (Figure 1).[21]

More practically, a growing body of data demonstrates a clear and independent association between AKI and adverse outcomes in patients undergoing catheterization procedures. These independent associations include higher rates of inpatient mortality, bleeding, and myocardial infarction in patients undergoing coronary[22] and peripheral arterial procedures[23,24] and higher adverse events and mortality in transcatheter aortic valve replacement (TAVR) procedures.[25] Regardless of the procedure type, these adverse event signals continue to manifest in the longitudinal follow-up for months and are related to AKI severity. The issue of residual confounding remains a major limitation of these observational studies. Nevertheless, in addition to individual patients' outcomes, the occurrence of AKI significantly affects the entire health system. AKI events are associated with prolonged length of hospital stay, higher readmission rates—often for heart failure or myocardial infarction—and higher total health care costs.[2,26,27] AKI events are tracked and reported in the context of the American College of Cardiology (ACC) National Cardiovascular Data Registries (NCDR) for PCI, TAVR, and PVI.[22,23,25] Thus, AKI is now a mainstream quality metric for in-hospital CVD procedures in the United States.

The definition of AKI in the context of catheterization procedures

Defining AKI has been an evolving process over the past 2 decades. Various criteria have been used in the literature, and this variation has made the comparison of trials and data sets challenging. Table 1 summarizes the most common definitions of AKI. Contemporary criteria for AKI in a broader context of the nephrology literature use the Kidney Disease: Improving Global Outcomes (KDIGO) thresholds for the presence and severity of injury.[28] AKI following CVD procedures has similarly been subject to multiple definitions, examined first in validation studies and implemented based on consensus.[22,28-31] AKI in this context was historically linked to the use of iodinated contrast dye, giving rise to the

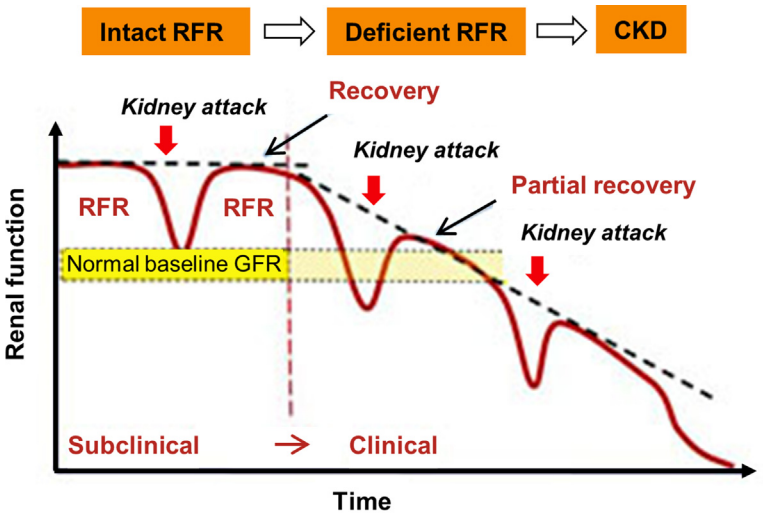


Figure 1. Theoretical construct of the renal functional reserve and kidney attacks. Small renal insults may remain subclinical if the renal functional reserve (RFR) is preserved. In the case of a true AKI event (kidney attack), there will be a transient reduction in GFR. In both cases, even if GFR returns to normal, recovery of renal function may be complete or partial. In case of partial recovery and reduced RFR, the kidney may be more susceptible to further insults and develop clinically evident AKI even in the presence of mild exposure. A progressive defective repair will then progress toward CKD. AKI, acute kidney injury; CKD, chronic kidney disease; GFR, glomerular filtration rate; RFR, renal functional reserve. Reproduced with revision from Sharma et al.[21] Copyright © 2014 Karger Publishers, Basel, Switzerland.

term contrast-induced nephropathy and later contrast-induced AKI.[32] The development of less nephrotoxic contrast agents and the increased complexity of CVD procedures has led to the recognition that AKI may be caused—all or in part—by factors other than contrast. Accordingly, the term contrast-associated AKI has been used more recently.[19]

Contrast-associated AKI has most commonly been defined in the CV literature as an increase in serum or plasma creatinine (SCr) levels by 25% or by 0.5 mg/dL at 48 to 72 hours postprocedure.[31] Subsequently, the CV community shifted to using the AKI-Network (AKIN)–based definition as reflected by the ACC NCDR registries.[22,23,25] This AKIN-based framework was first used in the NCDR CathPCI registry to track AKI rates in the US PCI population and provided the basis for a clinical risk prediction tool.[22] Subsequently, it has been extended to peripheral arterial endovascular procedures in the ACC NCDR PVI registry. In 2011, for heart valve procedures such as TAVR, the Valve Academic Research Consortium (VARC) developed an AKI definition based on the Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease classification.[33] Subsequently, in 2014, the VARC-2 recommendations used the AKIN criteria and stages,[34] and in 2021, the VARC-3 consensus document recommended using the KDIGO criteria.[35] In addition, VARC-2/AKIN AKI definitions are currently used by the Society of Thoracic Surgeons Transcatheter Valve Therapy registry. The writing committee suggests adopting the KDIGO criteria to define AKI for future studies in the context of CV procedures.

The reliance on SCr as the biomarker for these AKI definitions carries numerous limitations. Changes in SCr may be temporally delayed from the index injury event and, therefore, missed without serial laboratory testing.[36] Confounding our understanding is that not all elevations of SCr that reach the threshold definition of an AKI event may be the result of nephron injury. Fluctuations in SCr may be influenced by baseline volume status, transient alterations in hemodynamics, and assay variability.[37-39] More specific and sensitive biomarkers of AKI have been described and continue to be investigated for clinical use.[40-42] The biomarker substudy of the Prevention of Serious Adverse Events trial demonstrated heterogeneity between the AKI occurrence as measured by changes in SCr and changes in tubular injury markers, suggesting that some episodes of AKI following angiography may reflect hemodynamic effects rather than true parenchymal kidney injury.[36,43] It is likely that 4 possible renal outcomes following CVD procedures exist for a given patient: (1) no functional (SCr change) or structural (tissue level injury) change, (2) functional but no structural change, (3) structural change with no or subclinical functional change, and (4) both functional and structural change. The delineation of which patients develop a functional versus structural change remains uncertain at this time; therefore, strategies to mitigate AKI risk are best applied broadly. With the recognition that these small changes in SCr have high sensitivity but poor specificity for AKI, there has been an interest in incorporating end points that include more clinically relevant events as was done in the Prevention of Serious Adverse Events

Table 1. Definitions and staging of acute kidney injury.

	Serum creatinine criteria			Urine output criteria[b]
	KDIGO	AKIN	RIFLE[a]	
Definition of AKI	Increased by ≥0.3 mg/dL within 48 h or ≥50% within 7 d	Increased by ≥0.3 mg/dL or ≥50% within 48 h	–	<0.5 mL/kg/h for ≥6 h
Stage 1/Risk	Increased by ≥0.3 mg/dL or 1.5-1.9 times baseline	Increased by ≥0.3 mg/dL or 1.5-2.0 times baseline	Increased by ≥1.5 times baseline	<0.5 mL/kg/h for 6-12 h
Stage 2/Injury	Increased by 2.0-2.9 times baseline	Increased by >2.0 to 3.0 times baseline	Increased by ≥2.0 times baseline	<0.5 mL/kg/h for ≥12 h
Stage 3/Failure	Increased ≥3.0 times baseline; or ≥0.3 mg/dL to ≥4.0 mg/dL; or on KRT	Increased by >3 times baseline; or ≥0.5 mg/dL to ≥4.0 mg/dL; or on KRT	Increased by ≥3.0 times baseline or ≥0.5 mg/dL to ≥4.0 mg/dL	<0.3 mL/kg/h for ≥24 h or anuria for ≥12 h
RIFLE Loss	–	–	KRT dependent for >4 wk	–
RIFLE End-stage	–	–	KRT dependent of >3 mo	–

AKI, acute kidney injury; AKIN, Acute Kidney Injury Network; KDIGO, Kidney Disease Improving Global Outcomes; KRT, kidney replacement therapy; RIFLE, Risk, Injury, Failure, Loss, End-stage classification.

[a] Rather than a single definition and staging categories, the RIFLE classification provided definitions of AKI with increasing stringency but decreasing sensitivity (Risk, Injury, and Failure) and 2 outcome categories based on the duration of KRT-dependence (Loss and End-stage disease).

[b] Urine output criteria are identical for RIFLE, AKIN, and KDIGO.

Interventions that are likely beneficial to reduce AKI risk	Interventions that are of uncertain benefit to reduce AKI risk	Interventions unlikely to be beneficial to reduce AKI risk or are associated with potential harm
Identification of patients at risk for AKI (may be accomplished using risk prediction tools)	Diuresis in decompensated heart failure patients	Administration of N-acetylcysteine
Volume expansion in patients without decompensated heart failure	Forced diuresis with matched volume expansion	Hemofiltration or hemodialysis before or after contrast exposure
Identification of pre-procedure contrast dose limits, monitoring of contrast volume administrated, and reduction in total contrast dye administration	Dilution of contrast dye with saline	Prophylactic use of adjunctive drugs (antioxidants, dopaminergic agonists, natriuretic peptides, vasopressor use in non-hypotensive patients)
Avoidance of concomitant nephrotoxins	Routine use of iso-osmolar contrast agents over low-osmolar agents	Use of sodium bicarbonate infusion
Minimization of risks for procedural complications (bleeding, hypotension, conversion to emergency surgery)	Staging of interventions or serial procedures once a contrast volume limit reached	
Transfemoral approach for TAVR	Use of technologies to limit contrast use (use of dye delivery reduction devices, automated injectors, use of non-contrast imaging tools)	
	'Zero' or ultralow contrast PCI	
	Use of radial artery access as compared to femoral artery access for coronary procedures	
	Use of biplane cineangiography	
	Use of hemodynamic support devices as an adjunct to coronary interventions	
	Remote ischemic preconditioning	
	Carbon dioxide angiography for peripheral procedures	
	Use of high dose HMG-CoA reductase inhibitors (statins) for kidney protection	

Figure 2. National Kidney Foundation and the Society for Cardiovascular Angiography and Interventions Workshop Expert Opinion Recommendations for Catheterization Procedures and Acute Kidney Injury Risk Prevention. AKI, acute kidney injury; PCI, percutaneous coronary intervention; TAVR, transcatheter aortic valve replacement.

trial, such as death, the need for dialysis, or a higher threshold for creatinine change (doubling of SCr).[44]

AKI risk and the concept of "renalism"

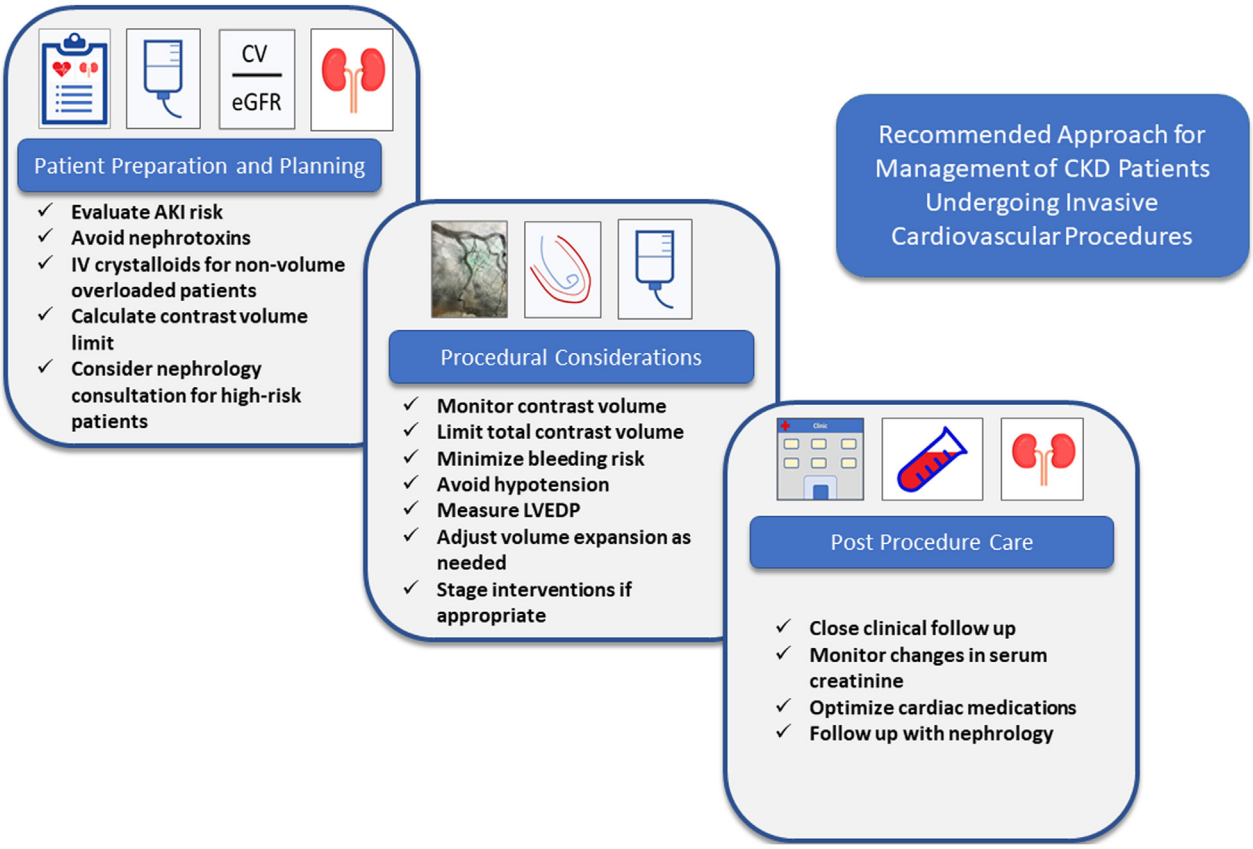
We have outlined the association of AKI with adverse clinical events and provided a framework whereby some patients may have nephron injury that could contribute to worsening long-term kidney function. In this context, the concerns related to AKI may dissuade providers and/or patients from proceeding with a catheterization procedure, even when otherwise indicated. This focus on kidney function at the expense of the overall patient's condition has been coined "renalism."[45] Several lines of evidence suggest that, despite overall benefit, patients with CKD are less likely to be treated for ACS with catheterization.[45-48] Furthermore, "renalism" may extend beyond procedural biases and influence the penetrance of evidence-based medical therapies in this population.[48] There is also preliminary evidence of "renalism" among patients with PAD in need of revascularization procedures.[49] Whether these biases exist for all CVD procedures remains to be determined. Regardless, the writing committee felt that, as a general principle, catheterization procedures deemed beneficial for overall outcomes should not be withheld from a patient solely based on AKI risk. Instead, a holistic approach involving patient-centered decision-making and strategies to prevent and manage AKI should be undertaken.

Preventive strategies

To date, there remains no effective treatment of the acute tubular injury leading to AKI following catheterization procedures, to attenuate the severity of injury once established, or to hasten recovery. Therefore, clinical management strategies should focus on risk prediction, prevention, and monitoring of outcomes. Across subspecialties and procedure subsets, there was a broad consensus among the writing group on these elements of AKI management. The recommendations of the writing group are summarized in Figure 2 and outlined in the Central Illustration. There was a strong recommendation that each institution develops an evidence-based AKI prevention protocol with input from key stakeholders, including nephrologists, cardiologists, nurses, and other involved staff. The protocol should include risk assessment tools, volume expansion strategies, and contrast administration management. Clinical outcomes should be monitored, and the local protocol should be revisited periodically. Early mixed-methods findings incorporating structured interviews with clinical care teams identified key standardized protocols associated with lower rates of AKI following PCI.[50,51] In a prospective study with >21,000 consecutive PCI procedures, when protocols were standardized and teams coached to implement those protocols, AKI was reduced by 28% in patients with pre-existing CKD. A new hybrid type 1 effectiveness-implementation cluster-randomized trial is underway to determine how best to coach teams in implementing AKI preventive protocols (IMPROVE-AKI [NCT03556293]) and launches a new era of pragmatic trials for AKI.[52]

Risk prediction

Advanced age, pre-existing CKD, diabetes, heart failure, high contrast volume procedures, and urgent or emergent procedures are all consistent predictors of AKI.[22,31] Several groups have developed formal AKI and dialysis risk prediction tools for patients undergoing cardiac catheterization procedures. Examples include the original Mehran risk score, the



Central Illustration. Recommended approach for management of patients with CKD undergoing invasive cardiovascular procedures. AKI, acute kidney injury; CKD, chronic kidney disease; CV, cardiovascular; eGFR, estimated glomerular filtration rate; IV, intravenous; LVEDP, left ventricular end-diastolic pressure.

Blue Cross Blue Shield of Michigan Cardiovascular Consortium scoring system, the ACC NCDR risk score, Veterans Health Administration based score, the ADVANCIS score, and the revised Mehran score.[22,31,53-56] No single scoring system was recommended over another by the working group; rather, the consistent use of a validated risk assessment tool by each hospital should be considered. These scores have variable penetrance in clinical use and are most often used in the cardiology (vs nephrology) literature. The drawbacks of these scores include uncertain generalizability, variable case mixture of patients at high versus low risk of AKI in the validation and testing cohorts, and lack of randomized data demonstrating benefits of implementation. The writing group considered several potential uses for risk scoring precatheterization. First, the use of these calculators provide data that better inform patients and families of likely versus perceived AKI risk. Second, having a common risk score may reduce equipoise or disagreement on treatment options among different specialty providers. Third, identifying high-risk patients may better focus prevention strategies and initiate closer follow-up of postprocedure kidney function. Finally, the implementation of risk scoring may alter overall provider or health care system behavior and improve AKI outcomes. This latter construct has been shown as a proof of concept in large health care systems, particularly when AKI risk scoring is integrated into the electronic medical record as a clinical decision tool.[57-59] Developing a validated AKI risk scoring system for PAD and TAVR procedures remains an ongoing area of research.[23]

Volume management

A significant proportion of the cardiac catheterization and AKI literature has focused on intravascular volume management. The writing group converged on several common themes. AKI risk is exacerbated in the context of hypovolemia. In most euvolemic or hypovolemic patients, volume expansion with intravenous normal saline is recommended. There is no evidence of benefit from other forms of crystalloid in this specific context.[44,60] The use of lactated Ringer's solution has been studied in critically ill patients, but this fluid strategy has not been validated for AKI prevention following catheterization procedures.[61]

Oral hydration before the procedure was believed to be helpful as an adjunctive measure but not as an alternative to intravenous hydration.[62] A reduction in the npo time and oral water given up to 2 hours before elective procedures is reasonable in patients treated under moderate sedation. Intravenous fluid administration protocols remain highly variable by the institution and differ by timing, duration, and volume of fluid administration before and after the procedure.[63,64]

The workshop group discussed the preprocedure assessment of patients' intravascular volume status. Tailoring administration by giving more fluid to hypovolemic patients and less to those who might develop volume overload or pulmonary edema was cited as an important clinical goal. Prior studies have shown efficacy in AKI reduction and safety when using techniques such as determining preprocedure total body fluid level via bioimpedance vector analysis, the left ventricular end-diastolic pressure during catheterization, and measurement of central venous pressure in patients with heart failure.[65-67] Incorporation of invasive hemodynamics to individualize volume therapy for patients was a strong recommendation. The writing group was less supportive about the role of volume expansion in patients initially presenting in volume overload states (acute decompensated heart failure). In these individuals, optimization of volume status with diuretics, staging of procedures, if possible, and obtaining invasive hemodynamic measurements were believed to be reasonable practices. The use of hemodialysis before or after catheterization to prevent AKI was not recommended. The use of noninvasive imaging assessments of volume status, such as inferior vena

cava dimensions or echocardiographic Doppler surrogates of filling pressures, remains to be better studied in the context of volume management for AKI prevention.

The proposed theoretical mechanisms for volume expansion and AKI prevention center on dilution of tubular contrast concentrations, increased urine flow rates and subsequent "clearing" of contrast from the kidneys, and potentially improved renal oxygen delivery.[68] The potential benefits of urine flow rate underlie the concept of "forced diuresis." Coupling of diuretics with volume administration matched for urine output has been shown to be beneficial in improving AKI rates in several studies of patients undergoing PCI or TAVR.[69] This strategy may be helpful but requires additional data and evaluation for broader applicability.

Contrast management

Contrast management remains a central focus for preventing AKI following catheterization procedures. The strong consensus of the writing committee was that contrast volume use should mirror the radiation exposure principle of as low as reasonably acceptable. A consistent association of higher AKI rates has been observed with increasing total contrast dose, particularly when indexed to baseline renal function and AKI risk.[70,71] Despite this relationship, paradoxically, contrast volume use by interventional cardiologists participating in the NCDR CathPCI registry remained high, even in the patients at the highest risk.[71] To achieve low volume use, the prospective planning of cases, discussions regarding contrast dose limits within teams, and intraprocedural monitoring of the volume of dye should be undertaken. The SCr level should be available before the procedure with a calculated eGFR. There was no clear statement on the method of eGFR calculation; however, eGFR (using institution-specific calculation method) should be used in lieu of creatinine clearance (Cockcroft-Gault).[72] The most recent statements from the American Society of Nephrology and the National Kidney Foundation recommend using the race-free 2021 Chronic Kidney Disease Epidemiology Collaboration equation.[73] A variety of contrast dose limits have been described in the literature. However, the total contrast volume indexed to an estimate of kidney function provides a validated ratio for determining the ideal limit.[70,74,75] Cardiology-based guidelines have recommended a contrast volume to eGFR ratio of 3.7 as an upper dose limit.[3,75,76] However, multiple studies demonstrate that risk increases substantially at lower thresholds; therefore, a ratio of 2:3 may be preferred.[70,77-79] Other studies from the NCDR CathPCI registry have suggested that a "one size fits all" approach in high-risk patients with AKI may not be appropriate, and patient-centered methods to individualize the contrast volume/eGFR ratio minimized AKI risk while allowing the maximum contrast volume for complex procedures.[57,80]

Technical advancements in contrast management over the past decade include automated contrast injectors and smart manifold syringes with contrast volume delivery modulation technology that provide real-time monitoring of contrast delivered and have been shown to reduce contrast volume delivery.[81-85] Furthermore, limited data would suggest lower rates of AKI with these devices.[86,87] In centers without these technologies, close observation of contrast amount used during a case should be undertaken by the staff, with frequent communication with the operating physician. Once a calculated contrast limit is reached, consideration should be made for staging additional procedures if clinically appropriate. The relative benefits and ideal interval duration of staging for AKI prevention remain to be determined.[88,89]

The contrast media used in most contemporary catheterization laboratories includes low- or iso-osmolar agents. Either of these agents is preferred over higher osmolar media. A substantial debate has existed in this context as to the relative merits of iso-osmolar contrast agents.[90] CV guidelines have largely stated that there are insufficient data to support the preferential use of iso-osmolar contrast agent or the use of one type of low-osmolar dye over the others. In the review of the published literature, meta-analyses would suggest a modest benefit of iso-osmolar media over low-osmolar agents in high-risk patients.[91,92] Further studies have outlined the role of iso-osmolar dyes in preventing limb pain during peripheral angiography and the potential reduction in composite renal and CV events.[93-95] However, the mechanisms for these benefits remain uncertain. There was no clear consensus from the expert panel supporting the broad option of 1 agent over the others for AKI prevention.

Apart from contrast delivery methods and staging, additional strategies were discussed to reduce procedural contrast use. These include the use of intravascular ultrasound (IVUS) or optical coherence tomography using diluted or dextran-based flush.[96] Adjunctive noncontrast–based imaging, use of intracoronary pressure wires, coregistration of multimodality imaging, and road mapping were all cited as technologies that could reduce contrast delivery to patients. The concept of "zero" or ultralow (total volume $< 1\times$ eGFR) contrast volume PCI has been shown to be feasible in selected patients.[97] Further exploration of these techniques' broad adaptability and safety remains to be determined. Each administration of contrast should result in meaningful, high-quality information, and unnecessary injections or "puffs" should be avoided. Biplane cineangiography has not uncommonly been utilized in patients with CKD; however, the body of literature to support biplane imaging to reduce contrast delivery or AKI risk remains sparse.[98-100] The tradeoffs of image quality and radiation exposure from biplane imaging should be considered. The panel also concluded that transthoracic echocardiography should be encouraged to assess left ventricular systolic function in lieu of contrast injection for left ventriculography unless absolutely needed. Lastly, dilution of contrast dye with saline may limit contrast dose; however, the impact on image quality, diagnostic accuracy, and AKI outcomes is uncertain.

Medication management

A consistent recommendation across guidelines and societal recommendations has been to limit, when possible, the use or initiation of known nephrotoxic medications in the pericatheterization period. Although the list of these agents is lengthy, nonsteroidal anti-inflammatory agents and nephrotoxic antibiotics remain common culprits. The risk-benefit of specific drugs should be examined in the context of an individual patient's condition. The expert panel discussed equipoise in the management of diuretics, renin angiotensin system (RAS) blockers, and sodium-glucose cotransporter-2 inhibitors.[101] The use of diuretics should be personalized for specific patients and clinical scenarios. RAS blockers and sodium-glucose cotransporter-2 inhibitors can lower the glomerular filtration rate in the acute period via largely transient hemodynamic changes in renal blood flow. These changes in eGFR could confound AKI diagnosis but should not be conflated with nephron injury. Three small trials have examined continuation or holding of RAS blockers before catheterization.[102-104] The studies did not use contemporary definitions of AKI and had no to modest signals of lower creatinine rise or change in eGFR. There remains insufficient evidence to stop or initiate these drugs specifically for AKI prevention. There is a risk of failure to reinitiate these therapies known to slow the progression of CKD. A clear consensus from both the cardiology and nephrology panelists was to avoid using N-acetylcysteine as this agent has no benefit in AKI prevention.

Follow-up of outcomes

Postintervention monitoring of SCr is a reasonable part of any comprehensive AKI prevention initiative. With same or next day discharges, the serial measurement of SCr levels may not be available. It is, therefore, possible that the literature underestimates the incidence of AKI

Class of Recommendation	Level of Evidence	Recommendations
1	C-LD	In patients with CKD undergoing contrast media injection for coronary angiography, measures should be taken to minimize the risk of contrast-induced acute kidney injury (AKI)
1	C-EO	In patients with STEMI and CKD, coronary angiography and revascularization are recommended, with adequate measures to reduce the risk of AKI
2a	B-NR	In high-risk patients with NSTE-ACS and CKD, it is reasonable to perform coronary angiography and revascularization, with adequate measures to reduce the risk of AKI
2a	C-EO	In low-risk patients with NSTE-ACS and CKD, it is reasonable to weigh the risk of coronary angiography and revascularization against the potential benefit
3: No benefit	B-R	In asymptomatic patients with stable CAD and CKD, routine angiography and revascularization are not recommended if there is no compelling indication

Figure 3. Recommendations for revascularization in patients with CKD from the 2021 ACC/AHA/SCAI Guideline for Coronary Artery Revascularization. AKI, acute kidney injury; CAD, coronary artery disease; CKD, chronic kidney disease; EO, expert opinion; LD, limited data; NR, nonrandomized; NSTE-ACS, non -ST-elevation acute coronary syndrome; R, randomized; STEMI, ST-segment elevation myocardial infarction.

after catheterization. Most retrospective or quality data set–derived AKI studies conducted in real-world settings have not followed SCr measurements. A minimum assessment at 24 hours and ideally at 48 to 72 hours should be considered in patients with high risk of AKI—especially inpatients. In such patients, follow-up with a nephrologist can be considered. Each catheterization laboratory should monitor the volume of used contrast, AKI incidence, and associated outcomes as part of a quality assurance program.

Specific considerations for CV disease subsets

Coronary artery disease procedures

The risks and benefits of cardiac catheterization for patients with CKD should be examined in the appropriate clinical scenario. Patients with CKD and ACS are at a high risk of adverse cardiac outcomes; therefore, emergent revascularization is indicated in patients presenting with ST-segment elevation myocardial infarction (STEMI) or shock. Similarly, an early invasive approach for non–ST-segment elevation ACS is recommended in this population. The role of revascularization in stable patients with CKD continues to evolve. The International Study of Comparative Health Effectiveness With Medical and Invasive Approaches–Chronic Kidney Disease (ISCHEMIA CKD) trial, for example, was a large prospective randomized trial to evaluate the role of revascularization (PCI or coronary artery bypass graft surgery, as appropriate) versus medical therapy in patients with advanced CKD (that is, eGFR of <30 mL/min/1.73 m^2 or on dialysis) and moderate or severe ischemia on noninvasive testing.[105] The study results demonstrated that routine invasive therapy failed to reduce the incidence of death or myocardial infarction compared with optimal medical therapy. Furthermore, a recent subanalysis of the ISCHEMIA-CKD trial suggests that in patients with concomitant chronic coronary artery disease and advanced CKD, an initial strategy including invasive CV procedures was associated with a significantly earlier initiation of dialysis than an initial conservative strategy.[106] Highlighting the earlier discussion on "renalism," these results should not be extrapolated to patients with ACS, severe angina (most patients in the trial did not have angina), left main stenosis, or patients with ischemic cardiomyopathy (left ventricular ejection fraction <35%). The key summary statements relevant to patients with CKD from the 2021 ACC/American Heart Association/SCAI Guideline for Coronary Artery Revascularization are outlined in Figure 3.[4]

Within the context of patients with CKD undergoing cardiac catheterization, the acuity of presentation increases AKI risk. Patients presenting with STEMI and/or cardiogenic shock represent a vulnerable population to kidney injury.[107] The incidence of AKI ranges from 20% to 60% across the STEMI and shock phenotypes spectrum and is an independent predictor of renal replacement therapy and mortality.[107-109] Baseline comorbidities, acute left ventricular dysfunction, and hypotension—particularly requiring vasopressor support—are all associated with AKI in these patients. The impact of hemodynamic support devices on renal function remains an ongoing area of investigation. The intra-aortic balloon pump (IABP) functions by rapid inflation of a helium-filled balloon in the descending aorta during diastole (to augment coronary filling), followed by rapid deflation during systole to augment forward flow. IABP counterpulsation can improve forward cardiac output, renal perfusion, and coronary perfusion, potentially benefiting kidney function. Conversely, the potential for vascular site bleeding, renal artery obstruction in cases of caudal migration of the IABP, and atheroembolism are potential risks to kidney function. Transvalvular hemodynamic support with the Impella (Abiomed) family of catheters has been increasingly used in the recent years. These devices actively pull blood from the left ventricle, resulting in a decrease in left atrial pressures and augmentation of cardiac output. However, these devices have inherent risks, including a large vascular entry profile and the potential for hemolysis. Their role in reducing mortality in patients with cardiogenic shock remains under investigation. In the context of high-risk PCIs, some observational data suggest a lower risk of AKI when Impella catheters are used for support,[110] whereas other data suggest a higher risk of AKI.[111]

Finally, the vascular access approach for patients undergoing cardiac catheterization has seen a marked increase in radial artery access use.[112] Radial access has been associated with improved patient satisfaction, reduced length of stays, reductions in access site bleeding, and cost savings.[112] Given the potential variation and challenges in radial catheter manipulation, less consistent arterial anatomy, and the possible need for femoral crossover, there were initially concerns about the potential use of greater volumes of contrast dye and the resultant impact on AKI risk. However, multiple studies have confirmed that radial catheterization compared with transfemoral access does not result in higher contrast volumes.[113] Furthermore, radial access for PCI is associated with lower rates of AKI in some studies—likely driven by lower bleeding and vascular complication rates.[112,114] Current clinical practice routinely leverages radial access for cardiac catheterization procedures. The 2021 ACC/American Heart Association Coronary Revascularization Guidelines state to "use radial artery access if feasible."[4] The recommendation is made on the basis of 3 studies with patients with ACS—1 randomized trial and 2 retrospective propensity analyses.[114-116] The key limitations

of these analyses are the inconsistent use of the AKIN or KDIGO AKI definitions and no control of volume expansion or contrast type between study arms. One randomized trial, Acute Kidney Injury-Minimizing Adverse Haemorrhagic Events by Transradial Access Site and Systemic Implementation of Angiox (AKI-MATRIX), failed to show a reduction in AKI with radial access when the KDIGO criteria were used as an outcome, although stage 3 AKI incidence was lower in this study.[114] The consensus from the working group was that further data (ideally prospective) are needed to better understand these relationships and to identify which patient populations benefit from transradial approach with respect to AKI outcomes.

PAD and PVIs

Patients with PAD frequently have CKD with an overlap of similar risk factors, including advanced age, diabetes, and established CVD. Patients with PAD and CKD have worse limb-specific outcomes than patients with PAD without CKD. Patients with CKD are also particularly prone to the development of vascular calcification. Calcified vasculopathy increases the risk of PVI. Calcification increases procedural complexity with patients presenting with multilevel disease, long diffuse lesions, need for atherectomy devices, and inability to expand lesions with conventional balloon and stent therapies.[117] Patients with very advanced CKD and ESKD have also not been routinely included in trials of endovascular devices. Furthermore, like the coronary procedure literature, AKI following PVI is associated with an increased risk of adverse outcomes, including mortality, myocardial infarction, progression of CKD, and major adverse limb events. AKI risk and dialysis risk are inversely associated with patients' baseline kidney function following PVI.[118]

Risk prediction tools are lacking in this space, and most current scoring methods are based on coronary procedures.[23] Given the marked differences in the pathophysiology and outcomes between patients with claudication versus chronic limb-threatening ischemia, the panel noted a need to better understand the role of clinical presentation on overall limb and renal outcomes in patients with PAD. Procedural complexity and the role of contrast as a nephrotoxin contributing to AKI risk were reinforced in the PAD procedure population.[107] Additionally, the role of atheroembolism was acknowledged as a potential understudied contributor to renal injury. Strategies to lower the contrast dose should be undertaken in these patients, and the panel recommended consideration of automated injectors, contrast delivery reduction devices, and the use of IVUS. Within experienced centers, the use of carbon dioxide imaging may have a role for these patients.[119] Appropriate contrast dosage thresholds have been studied, and keeping total contrast volume <2 to 3 times eGFR appears to be associated with a lower risk of AKI.[24] Preliminary data suggest that using iso-osmolar contrast dye in selected patients with PAD may be associated with reduced renal and composite CV events.[94] Additionally, randomized controlled trial data have demonstrated reduced patient discomfort when using iso-osmolar agents during lower extremity peripheral angiography and interventions.

The ideal protocol for volume expansion during PVI has not yet been established. Developing a validated volume expansion strategy for patients undergoing peripheral procedures was highlighted as a high priority area for further investigation. The trials that compared various intravenous fluids did have smaller subsets of patients undergoing peripheral angiography. Normal saline appears to be the most appropriate crystalloid for volume expansion in this population. The committee felt left ventricular end-diastolic pressure–guided hydration to be impractical in the peripheral context. The expert panel instead suggested fixed or weight-based saline infusion strategies. Many PVI procedures are performed in the outpatient setting and increasingly in office-based laboratories or ambulatory surgical centers. The ability to give volume for prolonged periods (overnight), either pre- or postprocedure, is less attractive given early same-day discharges. Potentially selecting patients at a higher risk of AKI for inpatient care for their PVI was suggested by the panel.

TAVR

The prevalence of aortic stenosis (AS) is growing globally, and over the past decade, TAVR has revolutionized the treatment of AS across a broad spectrum of patient subsets. Patients with CKD are at a higher risk of valvular calcification and progression of AS severity. Patients with kidney disease are also more likely to have calcification and disease in the iliofemoral vessels, most often used as access vessels for TAVR. The valvular and aortic arch calcium seen in CKD is associated with a higher risk of peri-TAVR stroke.[120] In addition, the presence of CKD can alter ventricular geometry with left ventricular concentric remodeling or hypertrophy, fibrosis, and dysfunction—all of which can confound echocardiographic assessments of AS severity.[18]

Not surprisingly, patients undergoing TAVR are also at risk of AKI, with rates as high as 20% to 30% when using sensitive definitions.[30] Elucidation of AKI risk factors in this context is continuing to evolve. To date, the role of procedural contrast volume as a key contributor to AKI remains uncertain and is variable across studies.[30,121] It should be noted that patients do have multiple potential contrast dye exposures during the workup and ultimate procedure, including diagnostic coronary angiography, PCI if needed, and computed tomographic (CT) imaging for implantation planning. Although not well studied, the committee recommends judicious use of contrast dye, spacing of dye loads when possible, and maintaining euvolemia during exposures. For annular and/or vessel sizing, modern CT low-contrast imaging protocols, the use of noncontrast CT imaging in selected patients, transesophageal echocardiography, and the use of IVUS may be helpful tools.

Contemporary data from the Society of Thoracic Surgeons/ACC National Cardiovascular Data Registry/Transcatheter Valve Therapy Registry have provided important insights into AKI and TAVR procedures.[25] Over 7 years of data analysis (2012-2018), AKI rates (AKIN stage 1) were as high as 26% in a given year, but overall AKI rates across AKIN stages declined over the sample period. In aggregate, of 107,814 patients, 11,566 (10.7%) developed postprocedural AKI. Of these patients, 9.5% experienced stage 1 AKI, 0.1% stage 2 AKI, and 1.1% stage 3 AKI. There were multiple patients and procedural risk factor associations for AKI, including the acuity of illness, the use of inotropes, alternative access, general versus moderate sedation, and need for conversion to open surgery. Contrast volume (>100 mL) was a modest risk factor for AKI, and the total volume used did not change during the study period. Most patients received <150 mL of contrast dye, with 36.7% receiving 0 to 75 mL and 43.9% receiving 75 to 150 mL. The occurrence of AKI was associated with a significantly higher risk of 1-year mortality. In summary, procedural approaches and the occurrence of complications appear to direct the AKI risk in these patients. Despite the risk of AKI, multiple studies, including observational registries and data from randomized controlled trial subsets, indicate the mortality and renal benefit of TAVR in patients with CKD.[122-124]

Perspective of a person living with kidney disease: Written by Mr Kevin Fowler

Thirty-seven million people in the United States have kidney disease, and a large proportion have CVD; thus, issues of imaging and procedures with imaging require ample attention. The recommendations provided by this group require serious consideration of how to inform patients of the risk and consequences of AKI. The subject of AKI in this scientific workshop is 1 element of a larger issue that requires immediate action by health care systems. Based upon my personal and professional experience, AKI is a concept that is poorly understood by persons with CKD and

kidney transplant recipients. I was over 6 years postkidney transplant when I learned that some antibiotics could cause AKI.

I was over 10 years postkidney transplant when I learned that high tacrolimus levels can cause AKI and accelerate allograft loss. Unfortunately, there is a significant gap in awareness and knowledge of AKI in those with the highest risk. It is important that health care professionals develop strategies to inform their patients with CKD about the risk of AKI and steps that can be done to reduce the risk. With the lack of therapeutic interventions to treat AKI, cardiologists and nephrologists must be mindful of the increased risk of CKD and protocolize prevention strategies. The COVID-19 pandemic has reinforced the need for AKI patient education and the urgent need for therapeutics. COVID-19 and AKI have disproportionally impacted the African American community, only worsening a public health crisis. In 2021, there was 1 positive advancement in addressing AKI. The Critical Path Institute launched the AKI Project. The AKI Project will focus primarily on the development of predictive tools for drug-induced kidney injury, but it is anticipated this initiative will also feed into, synergize with, and offer support for current and future efforts to develop tools to advance drug development for other causes of AKI and improve the care of patients with AKI.

Conclusions

As the population of patients with CKD continues to grow, the convergence of kidney disease and CVD will increase. In this context, the management of patients with CKD has unique challenges, particularly in preventing AKI. The potential adverse effects of "renalism" on patient outcomes should be considered part of clinical decision-making. Patient involvement and education for CKD management and AKI prevention is vital. This committee document provides consensus recommendations and highlights areas of uncertainty that warrant further scientific investigation.

Declaration of competing interest

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Ethics statement

This manuscript has adhered to the relevant ethical guidelines.

Supplementary material

To access the supplementary material accompanying this article, visit the online version of the *Journal of the Society for Cardiovascular Angiography & Interventions* at 10.1016/j.jscai.2022.100445.

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