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REVIEW

Navigating nephrotoxic waters: A comprehensive overview of contrast-induced acute kidney injury prevention

Panagiotis Theofilis, Rigas Kalaitzidis

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Panagiotis Theofilis, Rigas Kalaitzidis, Center for Nephrology "G Papadakis", General Hospital of Nikaia-Piraeus "Agios Panteleimon", Nikaia-Piraeus 18454, Greece

Corresponding author: Panagiotis Theofilis, MD, PhD, Doctor, Researcher, Center for Nephrology "G Papadakis", General Hospital of Nikaia-Piraeus "Agios Panteleimon", Mantouvalou 3, Nikaia-Piraeus 18454, Greece. panos.theofilis@hotmail.com

Abstract

Contrast-induced acute kidney injury (CI-AKI) is the third leading cause of acute kidney injury deriving from the intravascular administration of contrast media in diagnostic and therapeutic procedures and leading to longer in-hospital stay and increased short and long-term mortality. Its pathophysiology, although not well-established, revolves around medullary hypoxia paired with the direct toxicity of the substance to the kidney. Critically ill patients, as well as those with pre-existing renal disease and cardiovascular comorbidities, are more susceptible to CI-AKI. Despite the continuous research in the field of CI-AKI prevention, clinical practice is based mostly on periprocedural hydration. In this review, all the investigated methods of prevention are presented, with an emphasis on the latest evidence regarding the potential of RenalGuard and contrast removal systems for CI-AKI prevention in high-risk individuals.

Key Words: Contrast-induced acute kidney injury; Contrast media; Prevention; Hydration; RenalGuard; Dyevert

Core Tip: Although periprocedural hydration is a fundamental preventive measure for contrast-induced acute kidney injury (CI-AKI), recent research suggests exploring emerging strategies such as RenalGuard and contrast removal systems. These innovative approaches show promise, particularly in high-risk individuals with critical illness or pre-existing renal and cardiovascular conditions. By staying updated on the latest evidence and incorporating these advancements into clinical practice, healthcare professionals can enhance CI-AKI prevention efforts and improve patient outcomes.

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INTRODUCTION

Contrast media (CM), administered intravascularly during radiodiagnostic procedures to improve the visibility of blood vessels, can lead to a complication known as acute kidney injury shortly after their use[1]. This condition, termed contrast-induced acute kidney injury (CI-AKI), is the third most common cause of acute kidney injury, following reduced renal perfusion and the use of nephrotoxic drugs[2]. CI-AKI is characterized by an increase in serum creatinine levels within 48-72 hours after CM injection, after ruling out other causes of kidney impairment. Typically, serum creatinine levels return to baseline within 1-3 weeks[1]. The first 24 hours following CM exposure are critical, as 80% of cases show a rise in serum creatinine within this period, and nearly all patients who develop severe renal dysfunction exhibit increased serum creatinine within the same timeframe[3].

CI-AKI is linked to serious adverse outcomes, including chronic kidney disease (CKD), heart attacks, strokes, and death, with patients who develop CI-AKI experiencing higher mortality rates within one month[4,5]. The one-year mortality rate varies depending on the severity of pre-existing renal impairment before the CM procedure, ranging from 8%-23%, and can reach up to 55% in patients who require dialysis due to CI-AKI[6,7]. This review discusses the latest evidence on CI-AKI, covering the different prevention strategies and exploring new approaches currently under investigation..

EPIDEMIOLOGY AND OUTCOMES

Incidence and risk factors

The incidence of CI-AKI ranges from 0.6% to 2.3% in the general population but may increase in the presence of risk factors[8]. With over a million CM procedures performed annually in the USA, the incidence of CI-AKI is approximately 150000 cases per year[9]. Acute kidney injury following the intravascular injection of contrast agents for diagnostic or therapeutic purposes primarily occurs in patients with underlying risk factors that heighten the kidney's vulnerability to the CM. Pre-existing renal disease with elevated serum creatinine is a significant risk factor for CI-AKI development (Table 1). According to Mehran and Nikolsky[8], the incidence of CI-AKI in patients with underlying CKD ranges from 14.8% to 55%. The risk of CI-AKI increases with higher baseline creatinine values. For plasma creatinine levels ≤ 1.4 mg/dL, 1.4-1.9 mg/dL, and ≥ 2.0 mg/dL, the risk of CI-AKI is 2%, 10.4%, and 62%, respectively[8]. The need for dialysis is 10%, compared to less than 1% in CI-AKI requiring dialysis in patients without preexisting renal disease[10-12]. Renal function in patients with preexisting renal disease and elevated serum creatinine should be evaluated before exposure to CM for CI-AKI risk assessment[13]. Patients with renal transplants are at an increased risk for CI-AKI due to the high prevalence of diabetes, renal insufficiency, and the use of nephrotoxic drugs (*e.g.*, cyclosporine and nephrotoxic antibiotics)[14]. The incidence of CI-AKI in such patients is approximately 21.2%[14].

Diabetes is a risk factor in the case of underlying renal impairment[15]. Morabito *et al*[16] detected a comparable incidence of CI-AKI in non-diabetic and diabetic patients with preserved renal function and without other risk factors who underwent coronary angiography or Percutaneous Coronary Intervention (PCI)[16]. The incidence of CI-AKI depends on the value of serum creatinine. For serum creatinine levels ≤ 2 mg/dL, 2-4 mg/dL, and ≥ 4 mg/dL, the incidence of CI-AKI was 5.7%, 29.4%, and 81%, respectively[16]. Moreover, pre-diabetes (fasting serum glucose between 100 and 125 mg/dL) increases the incidence of CI-AKI through the enhanced synthesis of reactive oxygen species (ROS) and activated renin-angiotensin–aldosterone system[17]. Toprak *et al*[17] in a study of 421 patients (137 with diabetes, 140 with prediabetes, 140 normoglycemic) who underwent coronary angiography found that CI-AKI occurred in 20% of diabetic patients, 11.4% of pre-diabetic patients, and 5.5% of normoglycemic patients. The increase in serum creatinine was significantly higher in diabetic patients (absolute increase in serum creatinine 0.33 ± 0.08 mg/dL) and pre-diabetic patients (absolute increase in serum creatinine 0.22 ± 0.32 mg/dL) than in normoglycemic patients (absolute increase in serum creatinine 0.11 ± 0.06 mg/dL)[17]. Hemodialysis was required in 3.6% of diabetic patients, 0.7% of pre-diabetic patients, and none of the normoglycemic patients[17].

Patients older than 75 years have a 2-5-fold increased risk of CI-AKI[18]. Every one-year increment after 75 years increases the risk of the occurrence of CI-AKI by 2%[18]. The etiology is multifactorial, including age-related alterations in renal function [diminished glomerular filtration rate (GFR), tubular secretion, and concentrating ability][18]. The elderly also have an increased propensity for vasoconstriction from excessive angiotensin II and endothelin and higher levels of oxidatively modified biomarkers[18]. Renal function in the elderly should be evaluated before intravascular exposure to CM[18].

Outcomes

Patients who underwent CM procedures and developed CI-AKI experienced longer in-hospital stays than patients who did not develop CI-AKI. In a study of 1111 Israeli hospitalized patients in 2006, the average in-hospital length of stay was

Table 1 Clinical pearls for contrast-induced acute kidney injury in every day clinical practice

Risk factors-complications
Serum creatinine elevation occurs within 48-72 hours after the injection of the CM, with the first 24 hours post-exposure are crucial to the development of CI-AKI
Patients with preexisting CKD, elderly, and renal transplant recipients are at increased risk for CI-AKI development
Renal function *via* serum creatinine and eGFR calculation should be evaluated in high-risk patients before exposure to CM for CI-AKI risk assessment
Other notable complications of CM exposure: Myocardial infarction, shock, stroke, death, longer in-hospital stays
Prevention
CM considerations: Apply non-ionic, hypo-osmolar CM at lowest dose, prewarm at 37 °C
Discontinue nephrotoxic drugs: Non-steroidal anti-inflammatory drugs, aminoglycosides, metformin
Periprocedural hydration with normal saline in patients at risk:
Three mL/kg/hour 1 hour before to 4 hours after the procedure
One mL/kg/hour 12 hours before to 12 hours after the procedure
Individualized use of specialized systems (RenalGuard/DyeVert) in coronary procedures

CI-AKI: Contrast-induced acute kidney injury; CM: Contrast medium; CKD: Chronic kidney disease; eGFR: Estimated glomerular filtration rate.

almost twice as long among patients with CI-AKI compared to patients without CI-AKI[19]. Turan *et al*[20] reported a mean in-hospital stay of 9 (7-16) days in 30 NSTEMI patients who underwent a CM procedure and developed CI-AKI compared to 7 (5-9) days in 282 NSTEMI patients who did not develop CI-AKI.

Patients developing CI-AKI more frequently exhibit procedural cardiac complications, including myocardial infarction (even requiring emergency coronary artery bypass grafting), hypotension, shock, use of intra-aortic balloon pump, and cardiac arrest[21]. Furthermore, procedural complications occurred more frequently in patients who developed CI-AKI and included femoral bleeding, hematoma, pseudoaneurysm, stroke, acute respiratory distress syndrome, pulmonary embolism, and gastrointestinal bleeding[21].

Patients who develop CI-AKI also have a higher in-hospital mortality rate when compared with those who do not. McCullough *et al*[10] in a study of 1826 patients undergoing coronary interventions, reported a 7.1% in-hospital mortality rate in patients with CI-AKI *vs* 1.1% in patients without CI-AKI. The in-hospital mortality rate for patients with CI-AKI requiring dialysis was 35.7%[10]. Nearly one-third of patients who require in-hospital dialysis because of CI-AKI die before discharge[10]. The increased in-hospital mortality rate was additionally reported by Rihal *et al*[21] in a large randomized clinical trial (RCT) of 7586 PCI patients (22% in patients who developed CI-AKI *vs* 1.4% in patients who did not, respectively). In-hospital mortality rates are low (approximately 0.7%) for patients with CI-AKI without preexisting renal disease and diabetes mellitus[21].

Patients with CI-AKI also experience diminished long-term survival. According to Sadeghi *et al*[22], the one-year mortality rate in CI-AKI patients is 23.3%, compared to 3.2% in those who do not develop CI-AKI. CI-AKI significantly impacts long-term survival, particularly in patients with pre-existing renal disease. Gruberg *et al*[7], in a study involving 439 patients with pre-existing renal disease undergoing coronary interventions, reported a 37.7% one-year mortality rate for patients with preexisting renal disease who developed CI-AKI, compared to 19.4% for patients who did not have pre-existing chronic renal disease and developed CI-AKI. Additionally, diabetes mellitus contributes to increased long-term mortality rates, with a one-year mortality rate of 25.9% in diabetic patients who developed CI-AKI[23]. Last but not least, the analysis of 7287 patients (476 with CI-AKI) included in a prospective multicenter registry showed that 2-year net adverse clinical events occurred at a greater frequency in those affected by CI-AKI compared to those who did not (adjusted hazard ratio: 1.88)[24]. CKD was an additional aggravating factor (CI-AKI + CKD hazard ratio 3.29 with reference no CI-AKI + no CKD)[24].

PATHOPHYSIOLOGY OF CI-AKI

CI-AKI is thought to stem from renal medulla hypoxia, culminating in acute tubular necrosis, as opposed to direct toxic damage to renal tubules[25,26]. The renal tubule toxicity induced by CM directly promoting apoptosis is attributed to the suppression of mitochondrial enzyme activity[26]. Hypoxia in the renal medulla is the result of a reduction in vasa recta perfusion, heightened oxygen consumption by epithelial tubular cells, and alterations in medullary vasculature, further diminishing blood flow in the outer renal medulla[26]. This outer region is already poorly perfused under normal circumstances, as it is located distantly from the descending vasa recta[26]. Osmosis caused by CM contributes to heightened pressure in the interstitium and enhanced mobilization of sodium due to the entrainment of water in the renal tubule[26]. Ultimately, compressed vasa recta and peritubular capillaries are noted, as well as elevated blood viscosity, which together decrease vasa recta perfusion and exacerbate the hypoxic injury in the renal medulla[27]. Additionally, water is

reabsorbed to a lesser extent in the presence of CM in the renal tubule, resulting in greater intraluminal pressure and diminished filtration from glomerular capillaries. This enhances sodium transport[28], raising oxygen consumption from the epithelial cells in the renal tubule, thus aggravating the hypoxic insult in the renal medulla[27]. Adenosine also has constrictive effects, thereby decreasing the GFR. This results in lower sodium delivery, further contributing to decreased oxygen use[29]. Medullary vasoconstriction due to CM is also evident due to the action of released endothelin and prostanoids by endothelial cells through the activation of prostaglandin E_2 receptors 1 and 3 and endothelin receptor A [30]. Endothelin-induced vasoconstriction is more pronounced in subjects with preexisting renal disease[26]. The combination of adenosine catabolism and hypoxic renal medulla generates free radicals that scour nitric oxide (NO), resulting in weakened vasodilatory response[29]. Furthermore, impaired NO bioavailability boosts sodium reabsorption, leading to greater oxygen spending and hypoxia in the renal medulla[31].

PREVENTION OF CI-AKI

CI-AKI risk stratification

Mehran *et al*[32] devised a straightforward risk score to predict CI-AKI and the need for dialysis following PCI based on their study of 8357 patients. Each risk factor is assigned a weighted integer score, and the cumulative score has a predictive power for the incidence of CI-AKI, as well as the requirement for renal replacement therapy[32]. Recently, there have been emerging prognosticators of CI-AKI, particularly in patients undergoing coronary interventions in the setting of an acute coronary syndrome, namely the Athens Score or the PRECISE-DAPT[33,34]. Nevertheless, considering that renal function significantly influences CI-AKI incidence, estimating GFR remains the most practical method for risk stratification. This is especially pertinent in patients with certain characteristics such as the elderly, and those with diabetes mellitus, arterial hypertension, and CKD[35,36].

CM considerations

Concerning CM, it is recommended that a non-ionic and hypo-osmolar or iso-osmolar CM is administered at the lowest dose possible, especially for individuals at risk of CI-AKI[37]. Notably, CI-AKI requiring dialysis after PCI did not occur with CM doses below 100 mL[38]. Additionally, the concept of zero-contrast PCI, facilitated by Intravascular Ultrasound or Optical Coherence Tomography, may emerge as a viable alternative for patients with CKD undergoing coronary procedures[39,40]. However, even doses below 100 mL of CM can potentially induce CI-AKI in patients with both CKD and diabetes mellitus, particularly when the iodine concentration of CM ranges from 140-400 mg/mL[41,42]. Safety predictors for CI-AKI extend beyond contrast volume alone. Ratios such as the volume of CM to baseline creatinine clearance less than 3, the volume of CM to baseline estimated GFR (eGFR) less than 5.1, or the grams of iodine of CM to baseline eGFR less than 1 are considered more reliable indicators[43,44]. While automated contrast injector systems reduce the administered CM volume, there is no significant difference in the incidence of CI-AKI or the need for dialysis [45]. In a recent analysis of 182196 consecutive patients who underwent PCI over a 7-year period (2010-2016) to document the trends in CM volume use, Gurm *et al*[46] noted a decline in mean CM volume from 197 to 168 mL, as well as in the mean ratio of CM volume to GFR (from 2.91 to 2.51). The percentage of patients with a ratio of CM/GFR ≥ 3 was also significantly diminished (from 36% to 25%)[46].

Optimal CM administration is based on prewarming to 37 °C, as well as adequate dilution to lower its viscosity[47]. In patients with circulatory collapse or severe congestive heart failure, CM administration should be postponed until an improvement in hemodynamic parameters is achieved[37,48]. Repeated doses of CM ought to be postponed for 48 hours in patients at low risk for CI-AKI, extended to 72 hours for high-risk patients, in cases of CI-AKI, subsequent CM administration must be avoided until resolution of the acute event[37]. If procedural delays are feasible, an interval of 2-3 weeks is deemed reasonable in such cases[49].

Discontinuation of nephrotoxic drugs

In managing the risk of CI-AKI, the cessation of concurrent nephrotoxic drugs, particularly non-steroidal anti-inflammatory drugs, aminoglycosides, amphotericin-B, high-dose loop diuretics, and antivirals, could be considered prior to and following the procedure[50]. If feasible, the examination should be postponed to mitigate the cumulative impact of nephrotoxic drugs and CM[37]. Additionally, metformin discontinuation is advised 48 hours before the procedure and can be reinstated after assessing renal function in diabetic patients with severely impaired renal function (eGFR < 30 mL/min/1.73 m^2)[50]. While metformin is not considered a direct risk factor for CI-AKI, caution is exercised due to its kidney excretion and stimulation of intestinal lactic acid production, which could lead to lactic acidosis in the event of CI-AKI [51].

There is ongoing debate regarding the discontinuation of renin-angiotensin system-blocking drugs. While some argue that there is no need to interrupt angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) before CM exposure[52], the CAPTAIN study reported a higher incidence of CI-AKI in patients who continued these medications compared to those who withheld them[53]. A meta-analysis also indicated an increased CI-AKI risk, especially in elderly patients and those with CKD[54]. Considering the controversial findings, discontinuation of ACE inhibitors or ARBs could be contemplated, particularly in these subpopulations, 48 hours before the procedure, with reinstitution post-evaluation for CI-AKI[36].

Ischemic conditioning

Another technique that has been used for CI-AKI prevention is remote Ischemic Conditioning (RIC). Walsh *et al*[55] were among the first to document its effectiveness in 40 patients in whom endovascular aortic aneurysm repair was performed. Additional RCTs have since been performed, highlighting a benefit from RIC, irrespective of timing (pre or post-ischemic)[56,57]. A recent meta-analysis of 16 RCTs proved that RIC led to lower CI-AKI rates, along with fewer major adverse cardiovascular events based on a follow-up of 6 months, with hydration defined as an important contributor[58]. Despite those encouraging preliminary findings, RIC is not considered a standard of care for CI-AKI prevention, with additional studies being awaited.

Hydration

Periprocedural hydration has long been considered a fundamental approach for preventing CI-AKI in both renal-impaired and non-impaired patients[59,60]. The positive impact of hydration was initially elucidated in a study involving 78 CKD patients who underwent angiography[61]. The underlying mechanism involves volume expansion-induced inhibition of the renin-angiotensin-aldosterone system (RAAS), leading to the attenuation of renal vasoconstriction and hypoperfusion. Simultaneously, the increased concentration of renal prostaglandins is associated with vasodilatory actions. Furthermore, the dilution of intratubular CM and increased diuresis restrict the interaction of CM with renal tubular cells, lowering the risk of direct renal tubular toxicity[62].

The optimal route of hydration, whether enteral or parenteral, for preventing CI-AKI lacks clear evidence. A meta-analysis of eight studies involving 1754 participants demonstrated the noninferiority of oral hydration compared to IV hydration in preventing CI-AKI during coronary angiography[63]. Mueller *et al*[64] concluded that prophylactic hydration with isotonic saline 0.9% (809 patients, CI-AKI incidence 0.7%) was superior to saline 0.45% (811 patients, CI-AKI incidence 2%), especially in women, patients with diabetes mellitus, or those receiving equal or more than 250 mL of CM. Standard clinical practice typically involves IV isotonic saline administration at a rate of 1 mL/kg/h, initiated 3-4 hours before and continued for 4-6 hours after the procedure[50].

The groundbreaking AMACING trial introduced controversy, suggesting that no intervention was non-inferior to IV infusion of 0.9% saline, as recommended by current guidelines for CI-AKI prevention[65]. This approach was not only cost-effective but also demonstrated no subgroup differences based on eGFR, diabetes mellitus status, or the type of procedure. However, complications arised in a significant proportion of saline hydrated patients, such as symptomatic heart failure, emphasizing the need for cautious administration[65]. Long-term results showed no differences in mortality or renal function metrics during a 1-year follow-up[66]. The CI-AKIART study, following these results, advocated for a more restricted prophylactic hydration approach, recommending its administration only in patients with eGFR < 30 mL/min/1.73 m^2[67]. Recently, in a multicenter, open-label, randomized controlled study involving 1002 patients with CKD (eGFR 15-60 mL/min/1.73 m^2) undergoing coronary angiography, Liu *et al*[68] showed that a simplified hydration protocol of normal saline from 1 hours before to 4 hours after CAG at a rate of 3 mL/kg/hour was non-inferior to the standard hydration protocol (12 hours before and 12 hours after) in terms of CI-AKI occurrence, acute heart failure, or major adverse cardiovascular events at 1 year. The analysis of subgroups revealed a similar magnitude of effect irrespective of age, sex, eGFR, contrast volume administered, contrast volume/eGFR, or the presence of congestive heart failure[68]. The rate of the CI-AKI was also lower with the simplified hydration protocol in those undergoing PCI[68].

Methods assessing intravascular volume, including left ventricular end-diastolic pressure (LVEDP) or Central Venous Pressure, may reduce CI-AKI risk in CKD or heart failure patients by guiding the degree of IV hydration[69-73]. In the randomized study of 469 patients with ST-elevation myocardial infarction undergoing primary PCI, CI-AKI rates were lower in the LVEDP-guided aggressive hydration group, without an increase in acute heart failure incidence when compared to standard hydration (0.9% saline at 1 mL/kg/hour for 6 hours after randomization)[74]. However, another recent RCT with 114 patients undergoing coronary angiography failed to demonstrate the superiority of LVEDP-guided hydration over routine hydration for preventing CI-AKI[75]. Aggressive hydration guided by the Vigileo/FloTrac system has also been attempted in a randomized controlled study of 344 patients with acute myocardial infarction undergoing urgent PCI[76]. The investigators found a remarkably lower CI-AKI incidence in the intervention group compared to control (12.1% *vs* 22.2%) associated with a significantly greater mean volume received (1910 *vs* 440 mL), without increased rates of acute heart failure[76].

A balanced hydration strategy, involving temporary forced diuresis with furosemide with matched hydration *via* the RenalGuard System, has been proposed (Table 2). Dorval *et al*[77] studied high-risk patients undergoing a CM procedure and reported lower than predicted CI-AKI rates (9.5% *vs* 14.5%-55%) with RenalGuard balanced hydration. Moreover, in patients undergoing transcatheter aortic valve implantation (TAVI), RenalGuard hydration demonstrated significant CI-AKI protection[78]. Briguori *et al*[79] proved the advantage of RenalGuard compared to LVEDP-guided hydration, demonstrating noteworthy reductions in CI-AKI occurrence and one-month major adverse events in patients undergoing vascular procedures. It should be noted that RenalGuard-guided group exhibited a higher rate of hypokalemia[79]. There have also been studies with neutral outcomes, such as the REDUCE-AKI randomized, sham-controlled clinical trial that failed to detect a benefit of RenalGuard in patients undergoing TAVI, with indications of an increased long-term mortality in the treatment group[80]. In the STRENGTH trial of patients with CKD undergoing complex coronary, structural, or peripheral procedures, RenalGuard did not offer additional protection against CI-AKI and other adverse outcomes compared to standard hydration[81]. Similarly, patients with CKD undergoing TAVI did not gain any benefit from RenalGuard in the study of Voigtländer-Buschmann *et al*[82]. According to the meta-analysis of Wang *et al*[83], the RenalGuard system can reduce the risk of CI-AKI in patients undergoing PCI but not in those undergoing TAVI. Considering the existing evidence, RenalGuard's routine use is not advised, especially in TAVI procedures, but it could be employed on a case-by-case basis (Table 2)[84-90].

Table 2 Clinical evidence on the role of RenalGuard for the prevention of contrast-induced acute kidney injury in patients at risk

Ref.	Patients	Study design	Preventive strategy	Outcome
Briguori *et al*[79]	702	RCT	RenalGuard	Less CI-AKI, PE and 1-month MAE
			LVEDP-guided hydration	
Katoh *et al*[84]	60 (Japanese)	Observational	RenalGuard	High UFR associated with less CI-AKI
Chorin *et al*[85]	300	Observational	RenalGuard	Net decrease in eGFR and CI-AKI incidence
			Isotonic saline	
Visconti *et al*[78]	48	Non-randomized	RenalGuard	Protective against CI-AKI (OR 0.71)
			SB	
Briguori *et al*[86]	400	Observational	RenalGuard	Effective in reaching the target UFR (≥ 450 mL/h)
Barbanti *et al*[87]	112	RCT	RenalGuard	Reduced incidence of CI-AKI
			Isotonic saline	
Briguori *et al*[88]	292	RCT	RenalGuard (NAC + SB)	Lower CI-AKI and in-hospital dialysis incidence
			NAC + SB	
Arbel *et al*[80]	136	RCT	RenalGuard-active	Similar CI-AKI incidence
			RenalGuard-sham	Increased long-term mortality in active group
Mauler-Wittwer *et al*[81]	259	RCT	RenalGuard	Similar CI-AKI incidence at day 3
			Isotonic saline	No difference in secondary outcomes
Voigtländer-Buschmann *et al*[82]	100	RCT	RenalGuard	Similar CI-AKI incidence
			Isotonic saline	Similar 30-day and 12-month mortality rates
Ben-Haim *et al*[89]	58	Non-randomized	RenalGuard	RenalGuard was an independent predictor of lower Renal CM accumulation score
			Isotonic saline	
			None	
Mirza *et al*[90]	1205	RCT	RenalGuard (non-automated)	Significantly lower incidence of CI-AKI with RenalGuard
			Isotonic saline	

CI-AKI: Contrast-induced acute kidney injury; LVEDP: Left ventricular end diastolic pressure; PE: Pulmonary edema; MAE: Major adverse events; UFR: Urinary flow rate; eGFR: Estimated glomerular filtration rate; SB: Sodium bicarbonate; OR: Odds ratio; NAC: N-acetyl Cysteine; CM: Contrast medium; RCT: Randomized clinical trial.

Sodium bicarbonate

Bicarbonate serves to mitigate the acidification of renal tubular fluid, diminishing pH-dependent ROS formation, and enhancing ROS neutralization. According to a CI-AKI Consensus Working Panel of KDIGO, a potential but inconsistent advantage of isotonic bicarbonate over saline solutions in preventing CI-AKI was suggested[91]. This arises from the controversy in RCTs comparing the renoprotective effects of sodium bicarbonate and sodium chloride infusions[91]. However, Brar *et al*[92], in a study involving 353 patients undergoing coronary angiography with either sodium chloride or sodium bicarbonate, explored outcomes beyond CI-AKI (death, dialysis, myocardial infarction, stroke) and found no significant benefit with sodium bicarbonate infusion.

In a meta-analysis by Jang *et al*[93], the preventive effects of saline *vs* sodium bicarbonate for CI-AKI were compared, revealing beneficial results with sodium bicarbonate administration. Notably, there was no difference in the need for renal replacement therapy and mortality, while side effects consisted of serum bicarbonate and potassium abnormalities [93]. Conversely, a meta-analysis of 14 RCTs by Zoungas *et al*[94] showed no significant difference in efficacy between sodium bicarbonate and sodium chloride. The PRESERVE trial, involving 5177 high-risk patients undergoing angiography, failed to document a benefit of sodium bicarbonate over IV saline in preventing CI-AKI, the requirement of renal replacement therapy, or death[95]. The recently reported TEATE trial showed that even though IV or oral sodium bicarbonate administration led to more frequent urine alkalization (pH > 6), the difference in the rates of CI-AKI was not significant compared to standard saline hydration[96]. Nonetheless, sodium bicarbonate is considered more convenient for emergent procedures and cost-efficient compared to saline. Thus, the decision to use it should be based on an individualized approach[91,97].

N-acetylcysteine

N-acetylcysteine (NAC) possesses antioxidant and vasodilatory properties, acting by scavenging ROS, increasing NO synthase expression, competing with superoxide radicals for NO, forming S-nitrosothiol with vasodilatory effects, inhibiting vascular cell adhesion molecule-1 expression in glomerular mesangial cells who are in charge of inflammatory cell recruitment, and promoting glutathione production[98-100]. However, a CI-AKI-protective effect remains controversial, and no evidence suggesting a reduction in the need for dialysis in patients developing CI-AKI after receiving NAC. Marenzi *et al*[101] reported an effect of NAC which was dose-dependent, but this finding has not been consistently confirmed. Moreover, Hoffmann *et al*[102] concluded showed that creatinine metabolism might be influenced by NAC administration, rather than renal function, based on a study involving 50 healthy subjects not receiving CM but administered NAC.

A meta-analysis by Adabag *et al*[103], covering RCTs with oral or IV NAC administration (10 RCTs, 1163 patients), found no significant impact of NAC against CI-AKI (NAC group: 35%, Control group: 37%). In another meta-analysis of 10 RCTs (1163 patients), the CI-AKI rates were lower in the NAC group compared to the control group (7.9% *vs* 14.3%) [104].Considering the overall evidence, however, the use of NAC is currently not recommended.

Statins

Beyond their hypolipidemic effects, statins exhibit antioxidative and anti-inflammatory actions[105], potentially conferring renoprotection. They reduce ROS formation by enhancing heme oxygenase-1 protein production, an antioxidant protein that interferes with NADPH oxidase activity[106]. Studies on the efficacy of statins in preventing CI-AKI have yielded debatable outcomes. While most studies demonstrated a benefit, others showed minimal or no protective effect against CI-AKI. It's important to note that the majority of these studies focused on patients undergoing coronary procedures, limiting the generalizability of conclusions to this specific study population.

A meta-analysis by Zhang *et al*[107] suggested that a high dose of statins could reduce the incidence of CI-AKI. Zhou *et al*[108] found that statin administration was efficient primarily in patients with significant renal dysfunction. On the contrary, Zhang *et al*[109] showed that statins had a negligible impact in CI-AKI prevention. Additionally, Liu *et al*[110] meta-analyzed 9 RCTs (2560 statin-treated patients, 2583 control), observed a 53% decreased risk of CI-AKI and a reduced need for dialysis in the statin group. In another meta-analysis of 9 RCTs, Liu *et al*[111], reported that administering high doses of atorvastatin before coronary angiography significantly lowered CI-AKI incidence compared to low-dose statins or placebo. Lastly, Li *et al*[112], in their meta-analysis of 21 RCTs involving 7746 patients undergoing coronary angiography/PCI, confirmed the efficacy of short-term statin administration for preventing CI-AKI, despite the heterogeneity of the study group.

Despite these positive findings, questions remain unanswered regarding the optimal statin choice, timing, and dosage. However, their use is permitted, especially in patients undergoing coronary procedures. Giacoppo *et al*[113], in a Bayesian meta-analysis spanning two decades (124 trials, 28240 patients, 10 different preventive regimens), established that statins were the only preventive approach consistently and significantly preventing CI-AKI compared to saline.

Other antioxidants

Ascorbic acid, with its antioxidant properties scavenging ROS and vasodilatory effects[114], has been investigated for its potential in preventing CI-AKI. However, the limited number of studies and inconsistent data currently do not support its use in this setting[115-119].

Tocopherols, specifically vitamin E, act as ROS inhibitors, enhance NO activity, and improve mitochondrial membrane function[120]. Despite positive results in some studies, the limited number of investigations assessing the efficacy of vitamin E (α or γ tocopherol) for CI-AKI prevention does not warrant its current recommendation for prophylaxis[121-123].

Allopurinol, a xanthine oxidase inhibitor, restricts ROS formation following CM exposure[124]. While some studies demonstrate its beneficial action, others do not consistently support its efficacy for preventing CI-AKI[125-128]. In a meta-analysis of 5 RCTs involving 754 patients, allopurinol showed efficacy in high-risk CI-AKI patients undergoing PCI[129]. However, further investigation through large-scale RCTs is necessary to establish allopurinol as a recommended preventive agent for CI-AKI.

Calcium channel blockers

Calcium overload contributes to the pathogenesis of CI-AKI, as the sodium/calcium exchanger system plays a role in intracellular calcium overload, especially in hypoxic conditions induced by CM injection[130]. In this context, the reversal of transport leads to intracellular Ca^{2+} overload, contributing to tubular epithelial cell apoptosis. Calcium channel blockers (CCBs) may exert a renoprotective effect by inhibiting intracellular calcium overload, as initially demonstrated by Yang *et al*[131] and Neumayer *et al*[132] in a study of 35 patients who received intravascular CM along with oral nitrendipine, resulting in significant preservation of GFR. Russo *et al*[133] further supported the efficacy of CCBs in CI-AKI prophylaxis in a study of 30 patients without risk factors during intravenous pyelography, where nifedipine administration showed positive effects. Additionally, Yin *et al*[134] documented the effectiveness of amlodipine administration prior to CM administration in a cohort of 2666 hypertensive patients, by reducing the incidence of CI-AKI as well as the long-term survival.

Conversely, Khoury *et al*[135], in a study of 85 patients undergoing radiologic examinations involving CM infusion, found no statistically significant difference in serum creatinine increase between the control and nifedipine groups. The positive effect of nitrendipine was not established by Carraro *et al*[136] in a RCT with 121 patients who underwent arteriography after its administration for CI-AKI prevention. Moreover, Arici *et al*[137] failed to demonstrate a protective effect

of amlodipine in an RCT with 29 patients undergoing coronary angiography, as it had no impact on serum creatinine levels. In conclusion, due to controversial data, the use of CCBs in the prophylaxis of CI-AKI is not recommended.

Other vasoactive agents

Atrial natriuretic peptide (ANP), an endogenous natriuretic compound produced by cardiac myocytes in atria, demonstrates increased levels following CM injection, particularly in patients with underlying diabetes or preexisting renal disease[138,139]. ANP's actions include attenuating the reduction of GFR by increasing sodium delivery to the distal nephron through tubuloglomerular feedback[138,139]. Studies evaluating ANP's protective effect in CI-AKI prophylaxis yield controversial results, necessitating further research[140,141]. Similarly, recombinant brain natriuretic peptide is being studied for CI-AKI prevention[142,143], with its actions encompassing vasodilation, reduction of preload and afterload, inhibition of cardiac remodeling, the RAAS, sympathetic nervous system, as well as adenosine and endothelin release. However, conclusive data confirming its efficacy are awaited.

Trimetazidine, a drug used in stable coronary artery disease, inhibits β-oxidation of fatty acids, leading to glucose oxidation, thereby requiring less oxygen and ensuring adequate energy utilization in ischemic conditions[144]. Despite a notable reduction in CI-AKI incidence in high-risk patients, limited RCTs hinder its current recommendation[145,146].

Prostaglandin E1 and prostacyclin (PGI2) promote vasodilation, enhancing kidney perfusion and alleviating CM-induced hypoxic injury to the medulla[147]. Earlier studies have already pointed towards their potential in CI-AKI prevention[148-152]. In a recently reported RCT of 1146 individuals undergoing PCI, alprostadil was an independent protective factor towards CI-AKI in patients at moderate and high risk according to the Mehran risk score when compared to placebo, potentially driven by an anti-inflammatory action[153]. Moreover, according to a systematic review and meta-analysis by Xu *et al*[154], alprostadil use on top of hydration was related to a lower risk of CI-AKI as well as ameliorated renal function biomarkers (serum creatinine, blood urea nitrogen, serum cystatin, neutrophil gelatinase-associated lipocalin, urine macroglobulin). Despite the available evidence, the lack of a large-scale RCT testing alprostadil's efficacy in this setting is a deterring factor in its routine use. Ongoing is a RCT assessing the effectiveness of alprostadil liposome injection for CI-AKI prevention in patients with CKD and an additional risk factor for CI-AKI undergoing PCI (NCT05475717).

Theophylline, acting as an adenosine antagonist with vasodilatory and antioxidative effects, inhibits adenosine production stimulated by CM. A meta-analysis by Dai *et al*[155] suggested a significant beneficial effect of theophylline infusion for CI-AKI prevention. However, inconsistent findings in studies by Bagshaw and Ghali[156], and Kelly *et al*[157] warrant cautious consideration, and, as of now, theophylline use is not recommended for CI-AKI prophylaxis[155-157].

Hemodialysis-hemofiltration

The removal of CM from the bloodstream can be achieved through renal replacement therapy following the CM-requiring procedure. A single hemodialysis session can eliminate 60%-90% of the administered CM, while peritoneal dialysis can also achieve similar results but requires a longer duration than hemodialysis[158]. Despite several RCTs investigating the potential protective effect of hemodialysis on CI-AKI prophylaxis, a significant reduction in the incidence of CI-AKI was not consistently demonstrated[159-163]. The reasons for the lack of benefit from hemodialysis are not fully understood, with possibilities including rapid onset of renal injury post-CM administration or potential nephrotoxicity of hemodialysis[159].

In a notable RCT of 114 patients undergoing coronary interventions, Marenzi *et al*[164] demonstrated the potential of hemofiltration in comparison to hydration. Conversely, in a small-sized trial, hemodialysis was proven superior to hemofiltration regarding CM removal[165]. A pilot study highlighted the positive impact of high-flow intermittent hemodiafiltration both prior to and following angiography of coronary and peripheral arteries, against hydration, in individuals at risk of CI-AKI[166]. The study reported no such incidents, along with a steeper renal function decline at 1 year[166]. Although hemofiltration may reduce the risk of CI-AKI, its cost, the need for intensive care unit admission, and associated risks necessitate further studies to establish its benefit and cost-effectiveness.

Contrast removal-reduction systems

A novel approach involves removing the majority of injected CM from the coronary sinuses before it enters the systemic circulation during coronary angiography. This is achieved by inserting a catheter into the coronary sinus through the right femoral vein and transferring blood into an extracorporeal contrast-absorbing column. While effective in reducing CI-AKI incidence, this technique faces challenges with a high failure rate (57%), limiting its clinical applicability[165,167]. a new contrast reduction system known as DyeVert was utilized to prevent CI-AKI by minimizing residual CM administration and aortic reflux. A study involving 96 patients undergoing coronary angiography assessed its effectiveness, demonstrating reduced CM exposure without compromising image quality[168]. In 451 patients with acute coronary syndromes that ultimately underwent diagnostic and therapeutic coronary interventions, DyeVert resulted in a lower incidence of CI-AKI (DyeVert: 8% *vs* control: 19%)[169]. In a recent study of 136 patients undergoing PCI for stable coronary artery disease, DyeVert emerged superior to LVEDP-guided hydration in terms of creatinine increase and CI-AKI occurrence[170]. DyeVert may be extremely useful in cases of chronic total occlusion revascularizations, where large volumes of CM are usually required. Tajti *et al*[171] showed that its use is feasible in this setting, significantly reducing the amount of administered CM. Notably, a UK-based cost-utility analysis revealed significant cost savings and improved quality of life with the use of DyeVert[172]. Such findings were replicated in a hypothetical cohort of 1000 patients with stage 3b-4 CKD undergoing PCI, with DyeVert being more effective and less costly compared to standard of care[173]. Last but not least, we should mention the results of the latest RCT of 550 patients with acute MI (74.5% with STEMI) undergoing PCI, who were randomized to either CM volume reduction through the DyeVert system or manual/

automatic CM injection syringe[174]. A lower CM volume was recorded in the DyeVert group (95 ± 30 mL *vs* 160 ± 23 mL) and fewer CI-AKI events (16% *vs* 24.3%, absolute risk difference: -8.3%)[174].

CONCLUSION

CI-AKI, an iatrogenic complication of procedures requiring CM administration, has an overall low incidence in the general population which increases in the presence of risk factors, namely pre-existing renal disease. Managing CI-AKI is largely dependent on its prevention. This is achieved by precise risk stratification, appropriate choice and handling of CM, and interruption of agents that are potentially associated with nephrotoxicity. Prevention is the cornerstone of CI-AKI management starting with risk assessment, application of CM-related measures, and withholding of nephrotoxic drugs. Despite the plethora of clinical trials that have been conducted in this setting, no measure appears to have an unequivocal effect in preventing CI-AKI, with hydration being the most well-characterized. Modern approaches such as the RenalGuard balanced hydration and contrast manipulation systems may be of use in certain clinical scenarios but are not widely available in everyday practice.

FOOTNOTES

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Country of origin: Greece

ORCID number: Panagiotis Theofilis 0000-0001-9260-6306; Rigas Kalaitzidis 0000-0002-7773-7575.

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