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REVIEW ARTICLE

Contrast-Induced Nephropathy: An Overview

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Abstract

Background This review elaborates on the definition, incidence, risk factors and treatment options for contrast-induced acute renal injury and highlights the potential therapeutic options to prevent this condition. Contrast-induced nephropathy is a subclinical and acute form of renal failure characterized by an unexplained worsening of renal function within 48–72 h after the administration of iodinated contrast media.

Methods A PubMed search was performed to identify studies published in English and focused on contrast-induced nephropathy using specific keywords:: contrast-induced nephropathy, acute renal failure, iodinated contrast agent, chronic renal failure, and percutaneous coronary intervention.

Results The risk of developing contrast-induced nephropathy increases in the presence of certain factors, including pre-existing renal dysfunction, diabetes, congestive heart failure, advanced age, and the concomitant use of nephrotoxic drugs; this risk varies from 5% in patients with mild renal dysfunction to 50% in patients with diabetes and severe renal dysfunction.

Conclusions Over recent years, many patients undergoing percutaneous coronary intervention, do not opt to receive iodinated contrast agents due to the risk of acute renal failure, thus compromising diagnostic procedures. However, recent studies have shown that contrast-induced nephropathy occurs less frequently in patients with normal renal function than in those with pre-existing chronic renal failure and/or diabetes mellitus. Furthermore, over recent years, preventive strategies using intravenous fluids, pharmaceuticals, and renal replacement therapy, have reduced the occurrence of contrast-induced nephropathy. However, as diagnostic and therapeutic intervention paradigms evolve, some questions remain unanswered.

Keywords Contrast-induced nephropathy · Acute renal failure · Iodinated contrast agent · Chronic renal failure · Percutaneous coronary intervention

Abbreviations

AKI	Acute kidney injury
CAD	Coronary artery disease
CI-AKI	Contrast-induced acute kidney injury
CIN	Contrast-induced nephropathy
CKD	Chronic kidney disease
CKD-EPI	Chronic kidney disease epidemiology collaboration
eGFR	Estimated glomerular filtration rate
ESLD	End stage liver disease
GFR	Glomerular filtration rate
MDRD	Modification of diet in renal disease
NAC	N-acetyl cysteine
NaCl	Sodium chloride
PCI	Percutaneous coronary intervention
RIPC	Remote ischemic preconditioning
ROS	Reactive oxygen species

1 Background

Contrast-induced nephropathy (CIN) is indicated by a reduction in kidney function within 48 to 72 h post- administration of an iodinated contrast medium, in the absence of another cause [1, 2]. The risk of developing CIN increases in the presence of certain factors such as pre-existing renal dysfunction, diabetes, congestive heart failure, advanced age, and the concomitant use of nephrotoxic drugs [3]. The risk of developing CIN ranges from 5% in patients with moderate renal injury to 50% in patients with diabetes and severe renal injury [4, 5]. Renal dysfunction is also indicated as

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an absolute increase in serum creatinine of 0.5 mg/dl (or greater) from the normal range or 25% (or greater) in the estimated glomerular filtration rate (eGFR) ≤ 60 ml/dl/min (renal insufficiency), creatinine estimated using the Modification of Diet in Renal Disease (MDRD) formula [6], the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula [7] or the simple Cockcroft-Gault formula [8]. Serum creatinine elevations peak on days 3–5 and return to normal range within 10–14 days [9].

Many studies have shown that a small number of patients with chronic kidney disease (CKD) who undergo angiography or percutaneous revascularization may experience worsening of kidney function [10–12]. Renal failure is associated with longer hospital stays, impediments and long-term mortality [13, 14]. Levy et al. [15] reported a notable high risk of in-hospital mortality (34% vs. 7%) for patients who develop contrast-induced acute kidney injury (CI-AKI). Therefore, a logical step forward is to use prophylactic measures to minimize the risk of developing CIN, and to identify groups of patients who have a higher risk of developing this complication. This is more important due to the increased use of radiological contrast procedures, especially in patients who require multiple follow-ups due to underlying disease and/or in patients with greater frailty and morbidity [16, 17].

Preventative measures have been suggested for patients who have a higher risk of developing CIN. Increasing the volume of contrast is the most acknowledged preventative method; however, there is no consensus as to how to accomplish this or whether to use N-acetyl cysteine as a supplement [18–21]. For CI-AKI and adverse events, it is important to determine the true risk of clinically important kidney injury and the best clinical approach to preclude CIN. In this article, we review the incidence, underlying pathophysiology, and recent approaches for the management of CI-AKI.

2 Methods and Materials

2.1 Data Sources

The PubMed search was conducted using a variety of search engines, including Google, Bing Health, WebMD, and Healthline using the following keywords: contrast-induced nephropathy, acute renal failure, iodinated contrast medium, chronic renal failure, percutaneous coronary intervention.

2.2 Definition and Incidence of CIN

2.2.1 Definition

Contrast-induced nephropathy is characterized by a serum creatinine ≥ 0.3 mg/dl (26.5 µmol/l) or a ≥ 1.5-fold increase from normal within 48–72 h of iodinated contrast media exposure [22, 23] and accounts for up to 30% of acute kidney injury (AKI) in hospitalized patients [24]. However, serum creatinine has moderate sensitivity and low specificity because it is directly influenced by fluid movement and administered drugs [25, 26].

2.2.2 Incidence of CI-AKI

According to existing literature, the incidence of CI-AKI ranges from 3.3% to 14.5% [27, 28]. A previous prospective and observational study investigated 112 patients with chronic renal dysfunction with a glomerular filtration rate (GFR) of 30 ml–60 ml/min/1.73 m^2 who underwent computed tomography (CT) with intravenous iodinated contrast material; the authors found that only 0.9% of patients developed CI-AKI [28].

A retrospective study was performed in Iceland between 2008 and 2015 and focused on patients who underwent coronary angiograms with or without angioplasty; the authors demonstrated an interaction between contrast agent dose and existing renal function, with a significant risk of AKI only at higher doses in patients with a normal eGFR [29]. In a retrospective study of 544 consecutive cardiac catheterization patients with end-stage liver disease (ESLD), Bhandari et al. [30] analyzed 179 specific cases undergoing coronary angiography and found that CI-AKI occurred in 23% of patients. Overall mortality was 52% in the CI-AKI group and 37% in the non-CI-AKI group, with a mean follow-up of 2.2 ± 3.8 years; furthermore, mortality was moderately elevated in populations other than patients with ESLD [30]. The concomitant use of nephrotoxic drugs, non-steroidal anti-inflammatory drugs, and angiotensin-converting enzyme inhibitors, drugs that are used in more than 60% of patients undergoing imaging procedures, was also found to increase the incidence of CI-AKI [31]. The current incidence of CIN is still a matter of debate and depends on the presence of certain risk factors; however, the incidence of CIN is estimated to vary between 5 and 15%. Renal insufficiency, diabetes, a reduction in effective arterial volume, and the dose of contrast agent are the main risk factors [32].

2.3 Pathophysiology

The pathophysiology of CIN remains poorly defined and poorly understood. Related mechanisms include changes in renal blood flow leading to ischemic and hypoxic damage to the renal medulla, and the production of oxygen free radicals that damage the tubular epithelium [25] (Fig. 1). Furthermore, contrast agents exert direct nephrotoxic effects on tubular epithelial cells, leading to osmotic nephrosis and reduced oxygen output.

In addition, these agents stimulate the release of vasoactive molecules (i.e., endothelin and adenosine), minimize

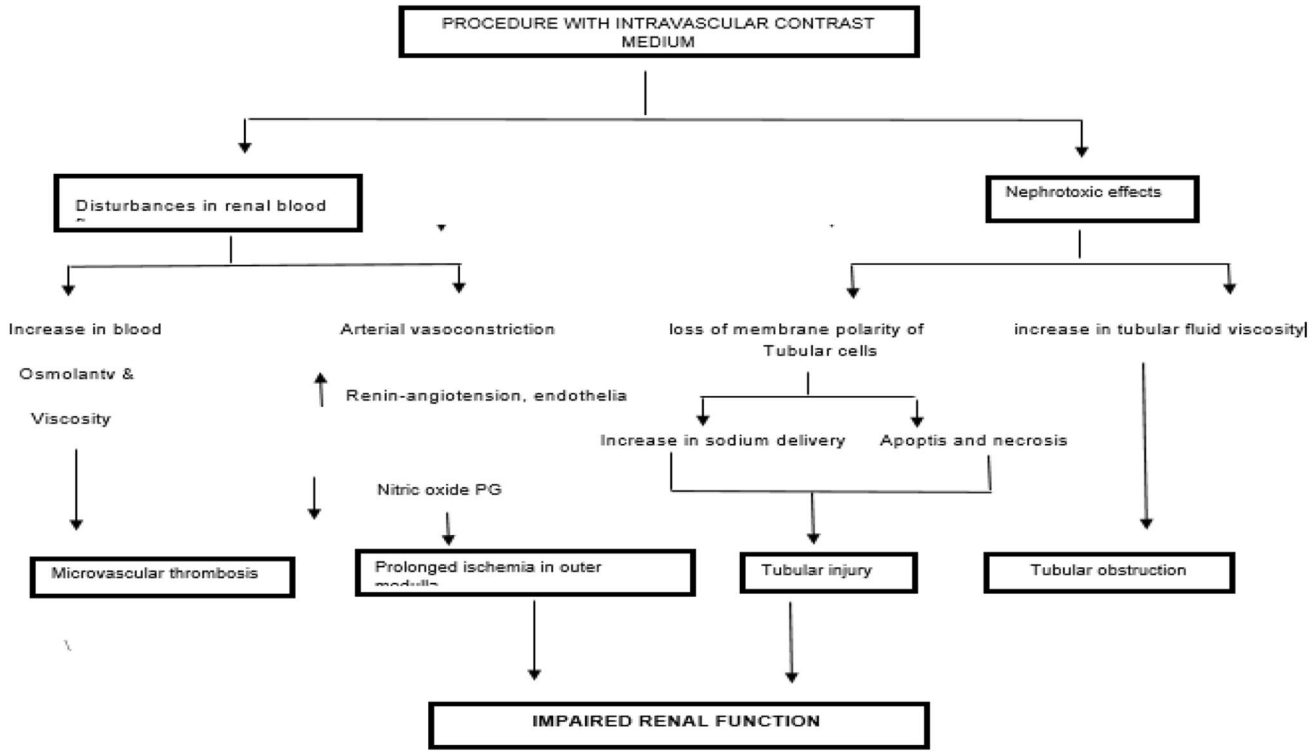

Fig. 1 Mechanisms of contrast-induced acute renal injury. Adapted from Mehran et al. [25]

the availability of vasodilators (prostaglandins and nitric oxide), and promote vasoconstriction and ischemic damage. These physiological changes cause a state of increased oxidative stress and cellular damage. In particular, the renal medulla is very sensitive to vascular changes due to its relatively low oxygen partial pressure. Chronic hypoxia and hypoxic adaptation may provide resistance to pigment-induced hypoxic tubular damage. In such circumstances, the development of renal dysfunction may reflect alterations in renal hemodynamics rather than true tubular injury [33, 34]. The accelerated osmotic load and viscosity associated with hyperosmotic contrast may also accelerate hypoxia in the renal medulla. In addition, hypo- and iso-osmotic contrast agents are highly viscous, a physical property that may also contribute to the pathogenesis of CIN [35].

2.4 Risk Factors of CI-AKI Post-Percutaneous Coronary Intervention (PCI)

Previous chronic kidney disease is the most significant risk factor for CI-AKI post-PCI. Patients with CKD are also at risk of certain complications such as ischemic heart disease and micro-embolization during angioplasty. Diabetes mellitus was found to be another significant risk factor. In patients with diabetes mellitus and chronic kidney disease, the occurrence of CI-AKI increases by twofold and can reach up to 33%. Even though there is imprecise affirmation that CI-AKI is related to the duration of diabetes or poor glycemic control, it is important to ensure strict glycemic control prior to the administration of contrast [36, 37]. Certain other factors can also increase the risk of AKI, including age, poor hydration, albuminuria, or the administration of renal toxic drugs [38] (Table 1). Age is considered an autonomous threat for CIN, especially in patients over 75 years of age [39]. Nevertheless, multiple studies have shown that age, male sex, atherosclerosis, and reduced left ventricular ejection fraction are not risk factors for CIN [40]. Toso et al. [41], Quintavalle et al. [42], and Han et al. [43] all reported that patients who developed CI-AKI had chronic renal failure, and identified this as the most significant risk factor. In other studies, the specific number of patients with CKD was not reported (e.g., Jo et al. [44]), or the number of patients with CKD represented only a small fraction of the total sample size (e.g., Xinwei et al. [45], Patti et al. [46], and Leoncini et al. [47]), or the number of patients with CKD was small (Ozhan et al. [48]).

2.5 Current Evidence for CIN

2.5.1 The PRESERVE Trial [49]

The PRESERVE Trial was a study funded by the National Health and Medical Research Council of Australia and the US Veterans Affairs for the Prevention of Serious Adverse

Table 1 Risk factors of CIN following the administration of contrast media [38]

Patient-associated risk factors	Iodinated contrast media	Renal toxic drugs
Previously existing chronic kidney disease	Intra-arterial injection high	Non-steroid anti-inflammatory
Diabetes mellitus	Osmolality high viscosity Injected	Aminoglycoside antibiotics
Old age	Volume > 100 ml multiple	Calcineurin inhibitors
Poor hydration/hypovolemia: sepsis, treatment with diuretics, decreased cardiac output	Injections –short time between injections (< 72 h)	
Hemodynamic instability		
Albuminuria		
Kahler's disease		
Anemia		
Reduced serum albumin concentration (< 35 g/l)		

Events Following Angiography and featured 5177 randomized patients who underwent coronary and non-coronary angiography to receive oral acetylcysteine versus placebo and intravenous isotonic bicarbonate versus saline. Patients with risk factors such as those who had stage 3B CKD or stage 3A CKD in the context of diabetes, were included in the study. In contrast to more prevalent consequences such as AKI, the investigators focused on major adverse kidney events (MAKE), which represented a composite end point consisting of mortality, dialysis, or a persistent 50% increase in blood creatinine at 90 days. The mean volumes of contrast and crystalloid delivered was 85 ml and 1 l, respectively. The major end point, which was experienced by 4.7% in the saline group in comparison to 4.4% in the bicarbonate group ($P = 0.62$), was the primary end point, and the trial was stopped due to the low probability of identifying a significant difference between these groups. There was no difference between patients receiving acetylcysteine and those receiving the placebo (4.6% versus 4.5%, respectively; $P = 0.88$). In addition, there were no significant differences between the main pre-specified subgroups (including stratification by baseline eGFR, albuminuria, and contrast volume) and no variations were seen in the rates of AKI, cardiovascular, or all-cause hospitalizations or adverse events. This trial did not directly address a number of issues, including the exclusion of patients with acute coronary syndromes who were undergoing PCI and the restriction of individuals who were at higher risk of developing CI-AKI. Overall, the PRESERVE study explained the potential benefits of the two therapies, thus addressing the shortfall in our understanding of these two approaches.

2.5.2 The AMACING Trial [50]

The AMACING trial was a prospective, randomized, phase 3, parallel-group, open-label, and non-inferiority trial for subjects who were at risk of CIN and carried out at Maastricht University Medical Centre in the Netherlands. High-risk patients (with an eGFR of 30–59 ml per min/1.73 m^2) over the age of 18 who were undergoing an elective procedure requiring the administration of iodinated contrast material were randomly assigned (1:1) to receive intravenous 0.9% NaCl or no prophylaxis. Patients with an eGFR of < 30 ml per minute per 1.73 m^2, a history of dialysis, or no referral for intravenous hydration were excluded. Between June 17, 2014, and July 17, 2016, 660 patients were randomized to receive either intravenous hydration (n = 328) or no prophylaxis (n = 332). For 307 (92%) of the 332 patients in the no prophylaxis group and 296 (90%) of the 328 patients in the intravenous hydration group, 2–6 day serum creatinine results were determined. Eight (2.6%) of the 307 non-hydrated patients and eight (2.7%) of the 296 hydrated individuals developed CIN. No hydration vs. hydration had an absolute difference of 0.10% (one-sided 95% CI 2.25 to 2.06; one-tailed $P = 0.4710$). In comparison to hydration, there were no cost savings with respect to non-hydration. Within 35 days, there were no deaths attributable to hemodialysis. Of the 328 patients, 18 (5%) reported problems with intravenous hydration. According to current clinical practice standards, the trial found that no prophylactic was non-inferior or cost-saving in preventing CIN when compared with intravenous hydration.

2.6 Risk Score for the Prediction of CIN

Mehran et al. [51] conducted a study to develop a simple risk score for CIN following PCI and proposed a CIN risk stratification score based on eight easily accessible characteristics, and demonstrated that a higher score was associated with a higher risk of CIN: (1) patient-related characteristics, such as an age > 75 years, diabetes mellitus, chronic congestive heart failure, or admission with acute pulmonary edema, hypotension, anemia, or chronic kidney disease; and (2) procedure-related characteristics, such as the use of elective intra-aortic balloon pump (IABP) or increasing volumes of contrast media. This risk score

Table 2 CIN risk score

Risk factors	Score
Age > 75 years	4
Hypotension[a]	5
IABP	5
CHF[b]	5
Anemia[c]	3
Diabetes	3
Contrast media volume	1 for each 100 cm^3
Serum creatinine	4
eGFR (ml/min/1.73 m^2 eGFR (ml/min/1.73 m^2 = 186 * $(Scr)^{-1.154}$ * $(Age)^{0.203}$ * (0.742 if female) * (1.210 if African American)	40–60 = 2 20–40 = 4 < 20 = 6

[a]Hypotension systolic blood pressure < 80 mm Hg for at least 1 h requiring inotropic support with medication or an intra-aortic balloon pump (IABP) within 24 h peri-procedurally

[b]CHF: congestive heart failure class III/IV by the New York Heart Association classification and/or a history of pulmonary edema

[c]Anemia baseline hematocrit value < 39% for men and < 36% for women

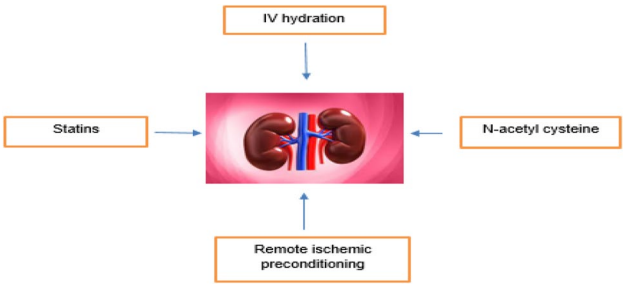

Fig. 2 Major preventive strategies for contrast-induced acute kidney injury. OCT adapted from Chandiramani et al. [40].

can be used for both clinical and investigational purposes. Table 2 provides a summary of the study's key findings.

Guo et al. [52] subsequently investigated whether the Mehren risk score was applicable in an Asian population who underwent coronary angioplasty and concluded that the best practice was to include the Mehran 2 risk score in patients who are hospitalized for coronary angiography.

2.7 The Prevention of CIN

The prevention of CI-AKI has been a long-standing subject of much interest. Research priorities for the management of CI-AKI include intravenous fluids, N-acetyl cysteine, statins, and remote ischemic conditioning [40], as shown in Fig. 2.

2.7.1 Hydration

Hydration is the cornerstone of preventive care as it can reduce the tubular content and consistency of iodinated contrast medium, reduce stimulation of the renin–angiotensin–aldosterone system, inhibit diuretic hormone synthesis, and minimize volume depletion and prostacyclin synthesis, thus reducing blood flow and causing hypostasis, renal perfusion and medullary hypoxia [19, 53]. Oral fluid intake rapidly increases diuresis by inhibiting vasopressin release and contributes to prompt and short-term renal protection. In contrast, saline suppresses the renin–angiotensin–aldosterone system, thus slowing down the response of the renal system to the intravenous administration of isotonic saline but providing long-term renal protection. It is recommended to commence with intravenous sodium bicarbonate 1.4 N or intravenous NaCl (0.9%) several hours prior to exposure to iodinated contrast medium. Sodium bicarbonate reduces the synthesis of reactive oxygen species (ROS), in addition to its effect on volumetric gain [54]. Eisenberg et al. [55] conducted a prospective study and reported that renal failure following large angiography could be prevented using the fluid intake procedure in response to the reported rate of post-angiographic acute renal failure of 12% [55].

2.7.2 N-acetyl Cysteine

By scavenging oxygen free radicals created as a result of toxic tubular damage, the antioxidant N-acetyl cysteine (NAC) has been suggested as a treatment to reduce the chance of developing CI-AKI. NAC's ability to prevent CI-AKI, however, is still debatable [56]. In this pervious study, a 154 mmol sodium bicarbonate solution was given to eligible participants in a single-center and randomized study at a rate of 3 ml/kg/h and then at a rate of 1 ml/kg/h for the following 6–12 h. NAC, as well as 154 mmol NaCl solution, was administered at a rate of 3 ml/kg/h, 2 h prior to the procedure, and at 1 ml/kg/h during the test or process for the following 6–12 h, in addition to the schedule. These authors found that NAC-based renal preventive strategies were superior at preventing CI-AKI in patients whose renal function has already been compromised. They also demonstrated the limited efficacy of bicarbonate edema alone in preventing CI-AKI. In high-risk patients, concurrent prophylaxis should be advised, including an increase of dosage and NAC, to reduce the possibility of renal damage following the administration of contrast media [57].

According to Kelly et al. [58], the most recent meta-analysis of 41 randomized studies, NAC is the most efficient treatment for CIN in patients with chronic renal failure. It has yet to be established whether this risk reduction improved clinical results. Given the known correlations of CIN with higher morbidity, mortality, and hospitalization,

NAC has been proposed as a routine preventive strategy for CIN due to its accessibility, low cost, and relative safety. Although more research is required, using NAC as a preventative measure against CIN may be warranted [59].

2.7.3 Statins

The study of Han et al. [43] reported two key advantages for the use of statins. Firstly, the sample size ($n=2998$) was higher than the total sample size ($n=2053$) of all other samples. Second, as indicators of a higher risk of CI-AKI, all patients had both diabetes mellitus and stage 2–3 CKD. There was no positive impact ($P=0.89$). Consequently, in individuals not receiving intravenous fluids, rosuvastatin can avoid CI-AKI. According to Ozhan et al. [48], patients undergoing coronary angiography may benefit from atorvastatin in terms of changes in serum creatinine (0.02 mg/dl in the atorvastatin group vs. 0.06 mg/dl in the control group, $P=0.023$). It is important to note that such minute variations in blood creatinine readings do not fall under the definition of CI-AKI and have no known clinical effects. The CI-AKI rate did not differ significantly across groups (3.3% vs. 10% or $P=0.135$). The primary finding of the trial by Barbieri et al. [60] was that both high- and low-dose statin medications were associated with a significant reduction in the risk of CIN when compared to a placebo.

2.7.4 Effects of Remote Ischemic Conditioning

Ischemic preconditioning was initially described in dogs in 1986 and in humans in 1993. This is a non-pharmacological, non-invasive approach. In order to "prime" cells against the possibility of ischemic harm in the future, this technique involves a brief sequence of ischemia/reperfusion prior to the start of protracted ischemia. This method is based on widespread, interspecies, and highly conserved endogenous cytoprotective mechanisms. Anaerobic glycolysis is enhanced when oxygen and nutrients are removed, whereas oxidative phosphorylation is suppressed. Cellular metabolism slows down after reperfusion and an "oxidative burst" occurs following significant oxygen intake. This causes the release of high levels of ROS and pro-inflammatory cytokines; these can cause cellular damage. In addition, cells reduce their metabolic activity and activate different genes in response to ischemia.

In a single-blinded, randomized controlled experiment conducted by Menting et al. [61], 76 individuals at risk of CIN were given either hydration alone, remote ischemic preconditioning (RIPC), or hydration as a pretreatment. The primary outcome measured the difference in serum creatinine between pre-contrast and 48–72 h later. RIPC had no discernible impact on the main endpoint. CIN was observed in four patients. According to Dutch findings, RIPC did not reduce post-contrast serum creatinine levels in patients at risk of CIN when used in addition to normal precautions. Nonetheless, the available data suggest that RIPC may be of benefit to patients with an extremely high or high risk of CIN.

2.8 Other Preventative Measures

2.8.1 Pharmacological Agents

Furosemide may provide renal-protective effects in the prevention of toxic renal failure by preventing tubular sodium transport and reducing the energy requirements of cells in the large ascending loop of Henle. Renal hypoxia is caused by an increase in external transport activity in the renal medulla, which also plays a significant role in the pathophysiology of CIN by increasing oxygen demand. Furosemide was therefore recommended as a kidney protector by reducing active tubular reabsorption. Moreover, furosemide increases the volume of urine produced and shortens the amount of time the contrast agent is in touch with the tubular epithelium, both of which reduce epithelial damage. However, in the absence of sufficient fluid replenishment, an increase in urinary salt excretion caused by a diuretic may cause salt depletion [62]. Hydration and furosemide were recommended as the optimal combination by Marenzi et al. [63]. According to the volume of urine produced while taking furosemide, patients were given an amount of IV fluid; these patients had better outcomes in comparison with patients who received hydration-only treatment.

Other pharmacological drugs are currently under investigation, including L-arginine, hypertonic mannitol, dopamine, theophylline, prostaglandin analogs, endothelin agonists, calcium channel blockers, furosemide, and prostaglandin analogs. However, none of these available treatments appear to be able to prevent iodinated contrast media-induced nephrotoxicity [64–66].

2.8.2 Intracellular Calcium Overload

It is known that an excess of intracellular calcium (Ca^{2+}) plays a significant role in ischemic cell damage and CIN. An increase in intracellular calcium can cause the intra-renal circulation to become vasoconstrictive and play a key role in mediating epithelial cell apoptosis and necrosis. Calcium channel blockers are therefore administered to exert a preventative effect on CIN. However, the use of calcium channel blockers has led to spurious findings in that some authors contend that it offers protection [67–69], while others claim that it offers no advantage [70–73].

2.8.3 Hemodialysis and Hemofiltration

The removal of X-ray contrast agent by hemofiltration or hemodialysis immediately after X-ray therapy was suggested by Schindler et al. [74]. Contrast agents (iopromide or iomeprol), high-flux hemodialysis, and hemodiafiltration over low-flux hemodialysis and hemofiltration have been found to successfully remove the contrast agents but do not prevent CIN in patients with CKD, the majority of whom undergo chronic dialysis [75]. Immediately following the delivery of a hypotonic contrast agent, Vogt et al. [76] tested whether prophylactic hemodialysis could prevent CIN in patients with renal impairment (baseline serum creatinine level > 2.3 mg/dl). Renal function was monitored prior to and for 6 days following the administration of contrast. The authors of this study concluded that the administration of hemodialysis as preventative care did not reduce the incidence of CIN.

2.8.4 The Impact of CIN

Current research is focused on the prevention of CIN as this condition is associated with greater morbidity, prolonged hospital stays, and higher healthcare expenses. The main objective of CIN prevention is to improve the outcomes of the healthcare team. A patient's medical history and the concurrent use of additional medications must be communicated to the nurse, pharmacist and physicians. It is crucial to obtain a thorough history of risk factors such as diabetes, heart failure, and high blood pressure. Any patient who is given a prescription for a procedure that involves the use of contrast dye needs to have their renal function and medical history examined extensively. Prior to the test, it should be ensured that any nephrotoxic medications are stopped. Before and after the test, the patient should be properly hydrated. The frequency of CIN may only be reduced via adequate communication and monitoring by the healthcare team [77–79].

2.9 Guidelines for CIN

According to the Consensus Guidelines [80] for the Prevention of Contrast-Induced Nephropathy by the Canadian Association of Radiologists, serum creatinine is not a reliable measure of renal function. Rather, the best method to reduce the incidence of CIN is the use of eGFR to assign risk levels and apply preventative activities.

When GFR < 60, the general recommendations for all patients are as follows:

- To avoid iodinated contrast media (CM) whenever possible
- To avoid nephrotoxic medications 48 h prior to the administration of CM
- To apply low- or iso-osmolar CM to prevent the use of high osmolar CM
- To reduce the amount of contrast used and avoid repeating the procedure within 72 h
- To consider acetylcysteine (AC) and avoid postponing or abandoning education with regards to AC

When GFR is 30–60, the general recommendations for all patients are as follows:

- To avoid volume contracts for patients
- To provide fluids orally or intravenously
- To provide GFR follow-up after 48 h of CM administration

When GFR is 30, the specific recommendations for patients are as follows:

- IV fluid administration (normal saline or sodium bicarbonate) for volume expansion
- To provide GFR follow-up after 48 h of CM administration

According to the guidelines published by the updated ESUR Contrast Media Safety Committee [81], only patients with an eGFR <45 ml/min/1.73 m^2 are at risk of CIN prior to the intravenous administration of iodinated contrast media. Furthermore, the risk of CIN is lower after intravenous administration than after the administration of intra-arterial contrast medium (at a similar dose). The Committee also recommended that either sodium bicarbonate or ordinary saline should be administered to expand volume and to avoid CIN.

3 Conclusion

A serious, underdiagnosed PCI consequence referred to as CI-AKI is associated with lengthy hospital stays, high rates of morbidity, and mortality. An increased occurrence of CI-AKI in post-PCI may arise from the increased use of PCI, the increased use of PCI during acute illness, or the increased use of PCI in patients with numerous co-morbidities. The most vulnerable patients are those who already have diabetes, congestive heart failure, or renal impairment. Risk evaluation and the implementation of preventative measures, such as avoiding dehydration, are essential. Early identification and treatment can reduce negative CI-AKI effects. A combination of numerous preventative measures,

as opposed to a single protective measure, may be more successful in preventing CIN; however, given its complicated pathophysiology, new treatment approaches that shield renal function from CI-AKI in patients receiving PCI require further investigation.

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Data Availability All data generated or analyzed during this study are included in this published article.

Declarations

Conflict of interest None of the authors have any conflicts of interest to declare.

Ethical Approval Not applicable.

Consent to Participate Not applicable.

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