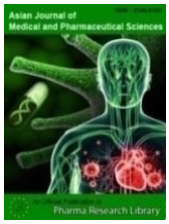

Asian Journal of Medical and Pharmaceutical Sciences
Home Page: https://pharmaresearchlibrary.org/journals/index.php/ajmps
ISSN (Online): 2348-0165 | Publisher: Pharma Research Library
A. J. Med. Pharm, Sci., 2025, 13(1): 56-62
DOI: https://doi.org/10.30904/j.ajmps.2025.4816

# Determinants of Contrast-Induced Acute Kidney Injury in High-Risk Individuals

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**A B S T R A C T**
The primary causes of contrast-induced acute kidney damage (CI-AKI) in high-risk people are examined in this study. CI-AKI was substantially correlated with factors including high contrast volume, advanced age, diabetes, and pre-existing renal disease. In patient populations that are at risk, identifying these factors is crucial for prevention and early intervention.
**Keywords:** Acute kidney injury, Contrast-induced acute kidney injury, High risk individuals

**A R T I C L E I N F O**

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**Article History:**
Received : 29 March 2025
Revised : 11 April 2025
Accepted : 24 May 2025
Published : 29 June 2025



***Citation:*** *Sk.Salma Sultana, et al. Determinants of Contrast-Induced Acute Kidney Injury in High-Risk Individuals. A. J. Med. Pharm, Sci., 2025, 13(1):56-62*

**C o n t e n t s**



## 1. Introduction

In order to better illness detection and therapy, improve the development effect, and alter the image contrast of local tissues, contrast chemicals are injected into the body through blood vessels. Iodine-containing contrast agents, sometimes referred to as iodine contrast medium (ICM), have become an essential component of illness diagnosis and have been widely employed in clinical settings in recent years due to the widespread use of computed tomography (CT) in clinical diagnosis [1] .Acute kidney injury (AKI), also known as contrast-induced acute kidney injury (CI-AKI) or contrast-induced nephropathy (CIN), is possible in 11–40% of patients treated with iodine contrast medium, according to some research [2].As the relationship between ICM and AKI has been well studied, the term CIN has lost some of its usage. Kidney Disease: Improving Global Outcomes (KDIGO)-endorsed CI-AKI has superseded the previous definition of CIN[3]. One of the main causes of hospital-acquired AKI that has an impact on patients' health in China is CI-AKI. Hydration therapy was originally thought to be the most practical, cost-effective, and efficient way to avoid CI-AKI[4].The subset of PC-AKI or CA-AKI that has a causal relationship to the delivery of contrast media is called CI-AKI. CI-AKI is advised when it is evident that ICM and AKI are causally related[5]. Hydration therapy is advised for the prevention of CI-AKI in both the 2018 ESUR CI-AKI Prevention and Treatment Guidelines[5]and the 2012 KDIGO AKI Clinical Practice Guidelines[4]. At the moment, CI-AKI has no particular treatment. Numerous randomized controlled trials (RCTs) and meta-analyses have examined a number of medications, including statins, vitamin C, sodium glucose cotransporter 2 inhibitors (SGLT2i), and N-acetylcysteine (NAC), however there isn't any conclusive evidence that they lower the incidence of CI-AKI. CI-AKI currently has very little effective treatment. Consequently, in recent years, a lot of study has focused on the mechanisms that underlie the development and progression of CI-AKI[5,6].

**Definition of CI-AKI:** A sudden decline in renal function linked to the use of iodinated contrast media is known as contrast-induced acute kidney damage (CI-AKI). This kind

of acute kidney injury is commonly seen as a side effect of percutaneous coronary intervention (PCI) and is linked to both short- and long-term negative consequences, primarily death, cardiovascular morbidity, and length of hospital stay[7].

**Pathophysiology of Contrast-Induced Acute Kidney Injury:** The two main causes of Contrast-Induced Acute Kidney Injury (CI-AKI) are direct cytotoxic effects of contrast media on renal tubular cells and hemodynamic changes that result in renal ischemia.

**Hemodynamic Effects**

- Medullary hypoxia and reduced blood flow are caused by renal vasoconstriction brought on by contrast media, particularly in the outer medulla[8].
- Renal vasoconstriction is increased by an imbalance between vasodilators (such as prostaglandins and nitric oxide) and vasoconstrictors (such as endothelin and adenosine)[9].
- Contrast drugs decrease oxygen transport and renal microcirculation by making blood and tubular fluid more viscous[1].

**Cytotoxic Effects**

- Contrast fluids cause vacuolization, apoptosis, and necrosis in renal tubular epithelial cells through direct cytotoxic actions[10].
- Reactive oxygen species (ROS) are produced in response to contrast agent exposure, which causes oxidative stress and tubular damage[11].
- Contrast agents limit ATP generation and induce death in renal tubular cells via causing mitochondrial malfunction[1].

**Characteristics and Diagnostic Standards**

- Serum creatinine (SCr) rising by at least 0.5 mg/dL or by at least 25% from baseline after 48 to 72 hours of contrast media exposure is usually referred to as CI-AKI[12].
- Acute Kidney Injury (AKIN) is defined as a decrease in urine output to ≤0.5 mL/kg/hour for at least 6 hours, or an increase in SCr of ≥0.3 mg/dL or a rise of ≥50% from baseline within 48 hours[12].
- AKI is defined by the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines as Urine output <0.5 mL/kg/hour for 6 hours, a rise in SCr of ≥0.3 mg/dL within 48 hours, or a rise of ≥50% from baseline within 7 days[13].
- After contrast exposure, serum creatinine levels usually start to climb 24 hours later, peak 3–5 days later, and then drop down to baseline in 7–10 days[14].

**Tools for Biomarkers and Diagnostics**

- Neutrophil Gelatinase-Associated Lipocalin (NGAL) is a sensitive and early biomarker for CI-AKI, and its levels rise 2–6 hours after contrast exposure[15].
- In contrast to SCr, serum cyclostatin C levels increase 24 hours after contrast exposure and are less affected by variables such as muscle mass, making it a more sensitive diagnostic for CI-AKI early diagnosis[16].
- Early tubular injury detection is made possible by the upregulation of Kidney Injury Molecule-1 (KIM-1) in proximal tubular cells after nephrotoxic insult. Urine levels of KIM-1 rise before changes in SCr[15].
- Within hours of contrast exposure, other biomarkers that show renal tubular damage include interleukin-18 (IL-18) and liver-type fatty acid-binding protein (L-FABP)[17].
- It has been demonstrated that the combination of Insulin-like Growth Factor-Binding Protein 7 (IGFBP7) and Tissue Inhibitor of Metalloproteinases-2 (TIMP-2) is more effective than other biomarkers in the early diagnosis of CI-AKI[17].

For the early diagnosis and treatment of CI-AKI, particularly in high-risk patients, these diagnostic standards and biomarkers are essential.

## 2. Risk Factors for CI-AKI

**Patient-Related Factors**

- Advanced Age: People over 65 are more vulnerable because of age-related declines in renal function and a higher incidence of concomitant conditions like diabetes and hypertension[18].
- Pre-existing Kidney Disease: The most important risk factor is chronic kidney disease (CKD), and the danger of CI-AKI rises with the severity of CKD[19].
- Diabetes Mellitus: Diabetes by itself may not raise the risk of CI-AKI greatly, but it does so when paired with chronic kidney disease (CKD)[20].
- Congestive heart failure: A higher incidence of CI-AKI is linked to congestive heart failure, which also leads to renal hypoperfusion[20].
- Volume Depletion: Low blood volume might worsen renal vasoconstriction brought on by contrast, which raises the risk of CI-AKI[19].
- Concurrent Use of Nephrotoxic Drugs: Drugs such ACE inhibitors, ARBs, and NSAIDs can affect renal autoregulation, increasing the risk of CI-AKI[20].

**Procedure-Related Factors**

- Type and Contrast Media Volume: The more contrast that is given, the higher the chance of CI-AKI. Patients at high risk may suffer kidney damage from even little amounts (~30 mL)[21].
- Route of Administration: Compared to intravenous approaches, intra-arterial delivery of contrast media carries a greater risk of CI-AKI, potentially because of increased acute intrarenal concentrations and the possibility of atheroembolism[18].

**Contrast Media-Related Factors**

**Osmolarity:** A higher risk of nephrotoxicity is linked to high-osmolarity contrast media. Particularly for patients

who already have renal impairment, low-osmolar and iso-osmolar agents are thought to be safer substitutes[22].

- Viscosity: The development of CI-AKI may be facilitated by higher viscosity contrast agents, which might increase tubular workload and decrease renal perfusion[23].
- Volume of Contrast Agent: The risk of CI-AKI is correlated with the volume of contrast media utilized in a dose-dependent manner. It's critical to limit the contrast volume, especially in high-risk individuals[18].

It is crucial to comprehend these risk factors in order to prevent and treat CI-AKI.

**Predictive Scoring Systems**

- **Mehran Risk Score**
- Development and Components: To estimate the risk of CI-AKI in patients receiving percutaneous coronary intervention (PCI), the Mehran Risk Score was invented. Eight variables are included: baseline serum creatinine or estimated glomerular filtration rate (eGFR), intra-aortic balloon pump use, hypotension, congestive heart failure, age greater than 75 years, anemia, diabetes mellitus, and contrast media volume[24].
- Risk stratification: People are divided into four risk groups according to their overall score: low (≤5), moderate (6–10), high (11–15), and very high (≥16), which correspond to a higher risk of CI-AKI[24].
- Clinical Utility: The Mehran Score is frequently used in clinical settings to detect high-risk individuals before they are exposed to contrast, enabling the use of preventive measures like hydration regimens and contrast volume limitation[25].

**Other Predictive Models**

- Contemporary Simple Risk Score: Eight clinical factors are included in this more recent model: age, clinical presentation, eGFR, congestive heart failure, diabetes, hemoglobin, basal glucose, and left ventricular ejection fraction (LVEF). When used on PCI patients, this model has shown good predictive accuracy for CI-AKI[26].
- Preprocedure Risk Score: Another model that predicts CI-AKI risk is the Preprocedure Risk Score, which emphasizes the significance of early risk assessment before to contrast exposure[27].
- Validation of Risk scores: The Mehran Score is one of several risk scores that have been validated in diverse patient populations by studies, demonstrating their usefulness in predicting CI-AKI and directing clinical decision-making[28].

**Utility in Clinical Practice**

- Risk Assessment: By using these scoring systems, physicians can evaluate the risk of CI-AKI before to contrast media procedures, which helps them make well-informed decisions and provide patient counseling[26].
- Strategies for Prevention: Preventive treatments, such as intravenous hydration, the use of low-osmolar or iso-osmolar contrast agents, and reducing contrast volume, can be implemented by identifying high-risk patients[26].
- Improvement of Outcome: By grouping patients according to their risk, these models help lower the prevalence of CI-AKI and its related problems, which eventually leads to better patient outcomes[24].

These prediction scoring systems are useful resources for managing and preventing CI-AKI.

## 3. Preventive Strategies

With an emphasis on pharmacologic therapies, contrast media selection, and hydration regimes, this provides a structured summary of preventive strategies for CI-AKI.

**Hydration Protocols**

- Intravenous Isotonic Saline: The incidence of CI-AKI can be effectively decreased by giving isotonic saline (0.9% NaCl) at a rate of 1 mL/kg/hour for 6–12 hours prior to and following contrast exposure[29].
- Sodium Bicarbonate Infusion: By alkalinizing urine and scavenging free radicals, using sodium bicarbonate solutions (e.g., 154 mEq/L) for hydration may have protective effects against CI-AKI[29].
- Customized Hydration Techniques: By tailoring hydration regimens to each patient's unique characteristics, such as left ventricular end-diastolic pressure, fluid administration can be maximized and the risk of volume overload reduced[29].

- **Use of Low- or Iso-Osmolar Contrast Media**

- Preference for Low- or Iso-Osmolar Contrast Media Use: Using low- or iso-osmolar contrast media is linked to a decreased risk of CI-AKI than using high-osmolar agents, especially in high-risk patients[30].
- Comparative Efficacy: Research has indicated that both low-osmolar and iso-osmolar contrast agents are superior than high-osmolar alternatives, with no discernible difference in CI-AKI incidence between the two[31].

**Pharmacologic Interventions**

- N-acetylcysteine (NAC): NAC's antioxidant qualities have been investigated in relation to CI-AKI prevention. Although several meta-analyses point to a possible advantage, the findings are conflicting, and its widespread application is still debatable[32].
- Statins: Because of its pleiotropic effects, which include enhancing endothelial function and lowering oxidative stress, pre-procedural statin medication may lower the incidence of CI-AKI[33].
- Sodium Bicarbonate vs. Sodium Chloride: Although the evidence is conflicting, some studies indicate that sodium bicarbonate may be more beneficial than sodium chloride in avoiding CI-AKI[33].

The incidence of CI-AKI can be decreased by putting these preventive measures into practice, particularly in high-risk individuals.

**Clinical Implications and Outcomes**

With an emphasis on both immediate and long-term effects, this is a structured summary of the clinical implications and outcomes of contrast-induced acute kidney injury (CI-AKI).

- **Short-Term Consequences**
  - Longer Hospital Stay: Because CI-AKI patients require more monitoring and control of their renal function, they frequently have longer hospital stays[34].
  - Need for Renal Replacement Therapy (Dialysis): Patients with severe CI-AKI may require dialysis, either temporarily or permanently, particularly if they already have kidney disease[35].
  - Increased In-Hospital Mortality: Patients with CI-AKI are more likely to die in the hospital, especially if they are having procedures like percutaneous coronary intervention[36].

**Long-Term Consequences**

- Chronic Kidney Disease (CKD) Progression: CI-AKI can cause renal function to deteriorate over time, raising the risk of CKD or hastening its course[37].
- Increased Long-Term Mortality: Even after renal function appears to have recovered, patients who suffer from CI-AKI remain at an increased risk of dying in the long run[38].
- Increased Risk of Cardiovascular Events: CI-AKI is associated with a higher chance of developing cardiovascular events later on, which raises morbidity and mortality[39].
- Recurrent AKI Episodes: Individuals with a history of CI-AKI are at an increased risk of experiencing acute kidney injury in the future, which further jeopardizes renal health[38].

Knowing these clinical ramifications emphasizes how crucial early intervention and preventative measures are to reducing the negative effects of CI-AKI.

**Recent Advances and Future Directions**

A comprehensive summary of recent developments and future directions in contrast-induced acute kidney injury (CI-AKI) is provided here, with an emphasis on new contrast agents, biomarkers, and preventative strategies.

**Emerging Biomarkers**

- Neutrophil Gelatinase-Associated Lipocalin (NGAL): As a possible early diagnostic for CI-AKI, neutrophil gelatinase-associated lipocalin (NGAL) can be seen in blood and urine within hours of contrast exposure, enabling an earlier identification than more conventional markers such serum creatinine[40].
- Kidney Injury Molecule-1 (KIM-1): A sensitive marker for CI-AKI detection, Kidney damage Molecule-1 (KIM-1) is increased in proximal tubular cells after damage[40].
- Interleukin-18 (IL-18): The pro-inflammatory cytokine interleukin-18 (IL-18) is elevated in the urine after renal damage and is a precursor to CI-AKI[40].
- MicroRNAs (miRNAs): The pathophysiology of CI-AKI has been linked to certain microRNAs (miRNAs), which may serve as prognostic and diagnostic indicators[41].

- **New Contrast Agents**
  - Iso-Osmolar Contrast Media (IOCM): IOCM, such iodixanol, have been shown to have a reduced incidence of CI-AKI in high-risk patients and to be less nephrotoxic than high-osmolar agents[42].
  - Low-Viscosity Contrast Agents: More recent contrast agents with lower viscosities are designed to lessen oxidative stress and renal tubular blockage, which lowers the incidence of CI-AKI[42].

**Novel Prevention Methods**

- Remote Ischemic Conditioning (RIC): RIC shows promise in lowering the incidence of CI-AKI by protecting distant organs, such as the kidneys, from ischemia-reperfusion injury through brief, non-lethal episodes of ischemia in a limb[43].
- Antioxidant Therapies: N-acetylcysteine and ascorbic acid are two examples of substances that have been researched for their ability to scavenge reactive oxygen species, which could help reduce oxidative stress-induced kidney damage in CI-AKI[41].
- Hydration Protocols: Using sodium bicarbonate solutions and other customized hydration techniques has been shown to reduce the risk of CI-AKI by inducing diuresis and diluting contrast media[44].

These developments in biomarkers, contrast agents, and prevention techniques present encouraging paths toward lowering the prevalence and severity of CI-AKI.

## 4. Conclusion

An organized summary of the conclusion is provided here: Key factors and the significance of risk assessment and prevention in contrast-induced acute kidney injury (CI-AKI) are summarized.

**Summary of Key Determinants**

- Patient-Related Factors: Congestive heart failure, diabetes mellitus, advanced age, and pre-existing chronic kidney disease (CKD) are all important risk factors for CI-AKI[45].
- Procedure-Related Factors: The risk of CI-AKI is influenced by the kind and amount of contrast media used, as well as the administration method (intravenous versus intra-arterial)[45].
- Properties of Contrast Media: Compared to low- or iso-osmolar contrast agents, high-osmolar contrast agents are linked to a higher risk of nephrotoxicity[46].

**Importance of Risk Stratification and Prevention**

- Risk stratification: It is essential to identify high-risk patients by evaluating their eGFR,

comorbidities, and other risk factors in order to put preventive measures in place[47].

- Preventive Strategies: The occurrence of CI-AKI can be decreased by following hydration guidelines, using low- or iso-osmolar contrast fluids, and decreasing contrast volume[48].
- Clinical Outcomes: CI-AKI is linked to longer hospital stays, dialysis requirements, and greater death rates, highlighting the significance of prevention[45].

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