International Journal of Medicine and Pharmaceutical Research
Home Page: https://pharmaresearchlibrary.org/journals/index.php/ijmpr
CODEN (USA): IJCPNH | ISSN: 2321-2624 | Publisher: Pharma Research Library
DOI: https://doi.org/10.30904/j.ijmpr.2025.4817
Int. J. Med. Pharm. Res., 2025, 13(1): 60-65

Prevalance and Outcomes of Contrast Induced Acute Kidney Injury

Dr.Sk.Salma Sultana*[1], Lakku Navitha[2], Katari Jhansi[2], Mallam Sriraj[2], Yennameedhi Mahendra Babu[2]
[1]Professor, Department of Pharmacology, Narayana Pharmacy College, Chintareddypalem, Nellore, A.P, India
[2]Narayana Pharmacy College, Chintareddypalem, Nellore, A.P, India

A B S T R A C T

Contrast-induced acute kidney injury (CI-AKI) is a frequent complication following the use of iodinated contrast media, particularly in patients with underlying risk factors such as chronic kidney disease and diabetes. The prevalence ranges from 2% to 25%, depending on patient characteristics and procedure type. CI-AKI is linked to increased hospital stay, morbidity, and mortality. Preventive measures like adequate hydration and use of low- or iso-osmolar contrast agents can reduce its incidence. Early identification and management are essential to improve outcomes.

Keywords: CI-AKI, Contrast Media, Acute Kidney Injury, Prevalence, Outcomes, Risk Factors, Renal Complications

A R T I C L E I N F O

***Corresponding Author**
Dr.Sk.Salma Sultana
Professor, Department of Pharmacology
Narayana Pharmacy College,
Chintareddypalem, Nellore, A.P, India

Article History:
Received : 14 Mar 2025
Revised : 19 April 2025
Accepted : 21 May 2025
Published : 11 June 2025

Citation: *Sk.Salma Sultana, et al. Prevalance and Outcomes of Contrast Induced Acute Kidney Injury. Int. J. Med. Pharm. Res., 2025, 13(1):60-65.*

Contents

1. Introduction

Definition of CI-AKI

An acute decline in renal function after intravascular delivery of iodinated contrast media is the standard definition of contrast-induced acute kidney injury, or CI-AKI. following excluding out alternative causes of renal impairment, the most commonly recognized criteria are an absolute increase in serum creatinine (SCr) of ≥0.5 mg/dL or a relative increase of ≥25% from baseline within 48–72 hours following contrast exposure[1,2].

Clinical Importance and Relevance

As the third most frequent etiology of hospital-acquired acute kidney damage, CI-AKI is a major contributor to this condition. Its prevalence is linked to higher short- and long-term death rates, longer hospital stays, increased morbidity, and higher healthcare expenses. About 25–30% of CI-AKI episodes may develop to chronic renal failure, and the rate might reach up to 25% in high-risk groups, such as those with chronic kidney disease (CKD)[3,4].

Pathophysiology Overview

- The pathophysiology of CI-AKI is complex and not fully understood. Important mechanisms consist of:
- ❖ Direct Cytotoxic Effects: Iodinated contrast media can cause direct harm to endothelial and renal tubular epithelial cells, which can result in necrosis, apoptosis, and mitochondrial malfunction[4].
- ❖ Hemodynamic Changes: Contrast agents have the potential to reduce medullary blood flow, induce renal vasoconstriction, and result in hypoxia and ischemia[4].
- ❖ Reactive oxygen species (ROS) production is a contributing factor to oxidative stress, which further deteriorates renal tissues[3].

Risk Factors and Vulnerable Populations

The following patient and procedure-related factors raise the risk of CI-AKI:

- Pre-existing Renal Impairment: Individuals with chronic kidney disease (CKD) are particularly vulnerable if their estimated glomerular filtration rate (eGFR) is less than 60 mL/min/1.73 m²[5].
- Diabetes Mellitus: People with diabetes are more vulnerable, especially if they also have chronic kidney disease[5].
- Advanced Age: People over 75 are more at risk[5].
- Renal hypoperfusion may be made worse by heart failure and hypotension[4].
- High Intra-arterial Administration and Contrast Volume: Higher risk is linked to larger contrast administration volumes and arterial pathways[5].
- Concurrent Use of Nephrotoxic Drugs: Certain chemotherapeutic drugs, aminoglycosides, and NSAIDs can increase the risk[6].

Objective of the Study/Review

This study's main goal is to thoroughly examine the prevalence and clinical consequences related to CI-AKI. This entails examining existing classifications, comprehending the pathophysiological mechanisms at play, identifying sensitive populations and important risk factors, and assessing how CI-AKI affects patient morbidity and death. In order to reduce the incidence and unfavorable consequences of CI-AKI, the study intends to suggest preventive measures and inform clinical practices.

Epidemiology and Prevalence

The epidemiology and prevalence of contrast-induced acute kidney injury (CI-AKI) are presented here, with particular attention to prevalence in high-risk populations, variations between hospital-based and community occurrence, and prevalence worldwide.

Global Prevalence Rates

- Global Incidence: A meta-analysis of 1.2 million patients in 24 countries found that the pooled incidence of CI-AKI was 12.8%, with differences noted depending on patient populations and therapeutic settings[7].
- Regional Variations: The incidence rates vary by geography; Asia has higher rates (13.2%) than North America (10.4%) and Europe (12.7%)[7].
- India-Specific Data: Patient comorbidities and procedural methods are two factors that affect the incidence of CI-AKI in India, which ranges from 2.4% to 29%[5].

Hospital-Based vs. Community Incidence

- ❖ Hospital Settings: As the third most frequent etiology of hospital-acquired acute kidney damage, CI-AKI is a major contributor to this condition. Depending on risk factors and criteria, the incidence in hospitalized patients having procedures such as coronary angiography might vary from 2% to 25%[8].
- ❖ Community Settings: There is little information available on the incidence of CI-AKI in community settings. In contrast, the risk is estimated to be between 0.6% and 2.3% in the general population without predisposing conditions[9].

Prevalence in High-Risk Groups

- People with Chronic Kidney Disease (CKD): Individuals who already have renal impairment are more vulnerable. Up to 40% of CKD patients may have CI-AKI, and in certain cohorts, up to 76% may be reported in some studies[10].
- Patients with Diabetes: Diabetes mellitus considerably raises the risk of CI-AKI, particularly when it coexists with CKD. Between 10% and 34% of diabetic patients have this condition[10].
- Elderly Patients: Growing older is known to increase the risk. Certain studies have reported CI-AKI incidence rates of up to 48% in patients 70 years of age and older[10].

2. Mechanisms and Pathophysiology

Hemodynamic Effects of Contrast Media

Renal hemodynamics may be significantly altered by the use of iodinated contrast medium. Vasoconstriction of the intrarenal vasculature is one of the main effects; this is especially true for the outer medulla, which is already vulnerable to hypoxia. By decreasing renal blood flow, this vasoconstriction causes ischemia and aids in the development of CI-AKI[11].

Role of Oxidative Stress and Inflammation

One important factor in the pathophysiology of CI-AKI is oxidative stress. Reactive oxygen species (ROS) are produced during the delivery of contrast media, and these can harm endothelial and renal tubular cells. Leukocyte infiltration and further kidney damage result from an inflammatory response that intensifies this oxidative damage by activating different cytokines and adhesion molecules[12].

Direct Tubular Toxicity

Renal tubular epithelial cells may be directly cytotoxically affected by contrast media. Contrast agent exposure causes cellular apoptosis and necrosis, which compromises the integrity of the tubular epithelium, according to in vitro research. Regardless of hemodynamic alterations, this direct toxicity plays a major role in the development of CI-AKI[13].

Types of Contrast Agents and Nephrotoxicity Potential

The nephrotoxic potential of contrast agents is influenced by their osmolality and chemical makeup. In contrast to low-osmolar (LOCM) and iso-osmolar contrast media (IOCM), high-osmolar contrast media (HOCM) have been linked to an increased risk of nephrotoxicity. Low-osmolar, nonionic agents are typically used because of their reduced risk profile. Nephrotoxic potential varies even across LOCM and IOCM, though, therefore cautious selection based on patient risk factors is required[14].

Risk Factors and Predictors

Patient-Related Factors (Age, Comorbidities)

The following patient-specific traits raise the risk of CI-AKI:

- Advanced Age: Because renal function deteriorates with age, patients over 75 are more vulnerable to CI-AKI.

- Comorbidities: A higher risk is linked to diseases including diabetes mellitus, hypertension, and congestive heart failure.
- Anemia and hypotension: These are two disorders that can worsen renal hypoperfusion and raise the risk of kidney damage[15].

Procedure-Related Factors (Contrast Volume, Type, Frequency)

The following procedural factors increase the likelihood of CI-AKI:

- Contrast Volume: Giving patients a lot of contrast media raises their risk, particularly if they already have renal impairment.
- Contrast Agent Type: Compared to low- or iso-osmolar contrast agents, high-osmolar contrast agents are more nephrotoxic.
- Exposure Frequency: Several contrast exposures in a brief period of time (e.g., 48–72 hours) may increase the risk[16].

Baseline Renal Function

One important predictor of CI-AKI is pre-existing renal impairment:

- Individuals having a lower estimated glomerular filtration rate (eGFR) are more vulnerable to chronic kidney disease (CKD).
- Acute Kidney Injury (AKI): People who have had or are currently experiencing AKI episodes are more vulnerable to further renal harm from contrast agents[4].

Use of Nephrotoxic Drugs

Nephrotoxic drug usage concurrently may increase the risk of CI-AKI:

- NSAIDs (nonsteroidal anti-inflammatory drugs): These can increase susceptibility to damage by compromising renal autoregulation.
- Certain antibiotics and aminoglycosides: These are known to have nephrotoxic properties, and when taken with contrast media, they may have cumulative effects.
- Chemotherapeutic Agents: Contrast agent side effects can be made worse by certain nephrotoxic chemotherapy medications[17].

3. Clinical Presentation and Diagnosis

The clinical presentation and diagnosis of contrast-induced acute kidney injury (CI-AKI) are briefly discussed here.

Timeline of Kidney Injury Post-Contrast

After iodinated contrast media is administered, CI-AKI usually appears 24 to 72 hours later. Within seven to ten days after exposure, serum creatinine levels usually recover to normal, having typically peaked three to five days earlier. In order to distinguish CI-AKI from other causes of acute kidney damage, this temporal pattern is essential[4].

Diagnostic Criteria (KDIGO, CIN Definition)

- Acute kidney damage is defined by the Kidney Disease: Improving Global Outcomes recommendations as:
- A 48-hour rise in blood creatinine of at least 0.3 mg/dL (26.5%/L); or
- A rise in serum creatinine of at least 1.5 times the baseline that is known or assumed to have happened in the last seven days; or
- <0.5 mL/kg/h of urine for six hours.
- These standards apply to the diagnosis of CI-AKI[18].

Biomarkers and Imaging Tools

- Serum creatinine and other conventional markers are late signs of renal damage. New biomarkers provide earlier detection:
- Within hours of contrast exposure, elevated levels of neutrophil gelatinase-associated lipocalin (NGAL) may be a sign of tubular damage.
- Cystatin C is a sensitive indicator of changes in glomerular filtration rate that rises before creatinine.
- Proximal tubular injury is reflected by Kidney Injury Molecule-1 (KIM-1)[19].
- These biomarkers improve risk assessment and early diagnosis in CI-AKI.

Short-Term Outcomes

- **Need for Renal Replacement Therapy (RRT)**
  Temporary dialysis may be necessary in situations with severe CI-AKI, particularly in high-risk or intensive care unit patients. The degree of renal injury is reflected in the requirement for RRT[20].
- **Length of Hospital Stay**
  Because of problems and the requirement for prolonged monitoring, CI-AKI is linked to lengthier hospital stays. Additionally, prolongation raises medical expenses[21].
- **In-Hospital Mortality**
  In-hospital mortality is much higher for patients with CI-AKI, particularly those who had underlying CKD or cardiovascular illness[22].
- **Progression of AKI Stages**
  From mild functional deterioration to severe AKI necessitating intervention, CI-AKI can take several forms. If it is not identified and treated right away, it could worsen[4].

  LONG-TERM OUTCOMES

  An outline of the long-term consequences of contrast-induced acute kidney injury (CI-AKI) is provided below.
- **Risk of Chronic Kidney Disease (CKD)**
  The risk of developing or worsening chronic renal disease is greatly increased by CI-AKI. Over time, patients with CI-AKI frequently have a higher glomerular filtration rate drop than patients without CI-AKI[8].
- **Recurrent Acute Kidney Injury (AKI)**
  Recurrent bouts of AKI are more likely to occur in patients with a history of CI-AKI. According to studies, 25–30% of people who have had AKI may undergo more episodes, which could result in more readmissions to the hospital and worsening renal impairment[23].
- **Long-Term Dialysis Requirement**
  Even while many CI-AKI episodes conclude without the need for long-term dialysis, severe

cases can result in end-stage renal disease that necessitates continuous dialysis therapy, particularly in individuals who already have CKD[24].

- **Mortality and Cardiovascular Implications**
  A higher incidence of major adverse cardiovascular events (MACE), such as myocardial infarction and stroke, and an elevated risk of long-term mortality are linked to CI-AKI. The risk level of these events is correlated with the severity of CI-AKI[25].

Prevention and Risk Mitigation

An overview of methods for preventing and reducing the risk of contrast-induced acute kidney injury (CI-AKI)

- **Hydration Protocols**
  One of the most important ways to avoid CI-AKI is to drink enough water before the procedure. Prior to and following contrast exposure, intravenous isotonic saline is administered to help preserve renal perfusion and aid in the removal of contrast. Hydration plans that are specifically tailored to each patient's risk variables have been proven to be successful in lowering the occurrence of CI-AKI[26].
- **Use of Low-Osmolality or Iso-Osmolar Contrast Agents**
  The risk of CI-AKI is greatly impacted by the contrast media selection. Particularly in high-risk patient groups, iso-osmolar contrast agents—like iodixanol—have been linked to a decreased incidence of nephrotoxicity as compared to low-osmolar agents[27].
- **Pharmacologic Interventions (e.g., N-Acetylcysteine, Statins)**
  The use of pharmaceuticals to prevent CI-AKI has been investigated. High-dose statins have been shown to be effective in lowering the risk of CI-AKI, either by themselves or in conjunction with N-acetylcysteine (NAC). By improving endothelial function and having antioxidant qualities, these substances may have protective benefits[28].
- **Dose Minimization Strategies**
  Reducing the amount of contrast material given is essential for lowering the risk of CI-AKI. Techniques include using imaging methods that require less contrast and determining the maximum permitted contrast dose based on patient-specific characteristics[29].

Management Strategies

An outline of Contrast-Induced Acute Kidney Injury (CI-AKI) Management Strategies are:

Supportive Care

Supportive treatment is the mainstay of CI-AKI management, with an emphasis on:

- Volume optimization is the process of maintaining renal perfusion by ensuring euvolemia through the delivery of isotonic saline.
- NSAIDs, aminoglycosides, and certain chemotherapeutics are examples of nephrotoxic substances that should be avoided by stopping or reducing exposure to them.
- Monitoring and addressing electrolyte abnormalities, especially hyperkalemia and acidosis, is known as electrolyte management.
- Nutritional Support helps in supplying enough food to aid in healing[30].

Monitoring Parameters

- Ongoing monitoring is crucial to evaluate kidney function and identify deterioration.
- Serum creatinine and eGFR: Daily measurements to track renal function
- Urine output: monitoring for oliguria (<0.5 mL/kg/h) as an early indicator of worsening AKI
- Biomarkers: Emerging markers like NGAL and cy statin C may offer earlier detection, though their ro utine use is still being investigated[4].

Role of Nephrology Consultation

Nephrology experts should be consulted as soon as possible in the following situations:

- Severe AKI: especially at Stage 3 or while considering renal replacement treatment.
- Unclear Etiology: When AKI's cause is unclear.
- Despite early treatment attempts, persistent anuria or oligouria may persist.
- Complicated cases: These are individuals that rapidly deteriorate or have several comorbidities[30].

Management of Complications

Promptly addressing difficulties is essential:

- Electrolyte imbalances: Dialysis, calcium gluconate, or insulin-glucose treatment may be necessary for hyperkalemia.
- Dialysis or diuretics are used to treat volume overload if the patient is not responding.
- Metabolic Acidosis: Dialysis or bicarbonate therapy may be required.
- Infections: Close observation and timely antibiotic treatment where necessary[30].

Recent Advances and Research Gaps

An overview of the latest developments and unmet research needs in CI-AKI (contrast-induced acute kidney injury) are:

Emerging Diagnostic Tools (Novel Biomarkers)

- Serum creatinine and other conventional markers are delayed indicators of renal damage. A number of new biomarkers have been discovered recently that allow for the early detection of CI-AKI:
- Within hours after contrast exposure, elevated levels of neutrophil gelatinase-associated lipocalin (NGAL) may be a sign of tubular damage.
- A sensitive indicator of changes in glomerular filtration rate, cystatin C rises before creatinine.
- Damage to the proximal tubules is reflected by Kidney Injury Molecule-1 (KIM-1).
- In CI-AKI, these indicators improve risk stratification and early diagnosis[31].

Ongoing Clinical Trials

A number of clinical trials are being conducted to assess therapies meant to prevent or lessen CI-AKI:

- The preserve Trial evaluated the effectiveness of oral N-acetylcysteine and intravenous sodium bicarbonate in avoiding CI-AKI in patients undergoing angiography.
- The amazing trial examined the need for preventative hydration in patients receiving contrast-enhanced operations who are at risk of CI-AKI.
- The purpose of these studies is to enhance patient outcomes and preventive measures[32].

Areas Needing Further Investigation

Despite progress, further study is needed in a few areas:

- Pathophysiological Mechanisms: More research is required to fully understand the exact mechanisms of CI-AKI, including the contributions of inflammation, oxidative stress, and direct tubular toxicity.
- Standardization of terminology: Consistent research and clinical practice require a defined definition of the CI-AKI, as variations in terminology make it difficult to compare studies.
- Long-Term Results: Additional research is required to comprehend the long-term cardiovascular and renal consequences of patients who acquire CI-AKI[4].

4. **Conclusion**

This is a succinct summary of the main conclusions, clinical consequences, and suggestions for future management regarding Contrast-Induced Acute Kidney Injury (CI-AKI).

Summary of Findings

CI-AKI, which usually appears 48–72 hours after contrast media exposure, is a leading cause of acute kidney injury acquired in hospitals. It is caused by processes such as renal vasoconstriction, oxidative stress, and direct tubular toxicity. Individuals who are older, have diabetes mellitus, or have chronic kidney disease (CKD) are more vulnerable. Hydration is still the mainstay of prevention, even with studies into pharmaceutical preventives[4].

Clinical Implications

Negative consequences include extended hospital stays, higher medical expenses, and higher in-hospital and long-term mortality are linked to CI-AKI. These ramifications highlight the necessity of early detection and preventative actions in communities that are at risk[4].

Recommendations for Future Practice

- To identify high-risk people, risk assessment methods like the Mehran score should be regularly used prior to administering contrast[33].
- For prevention, hydration regimes utilizing sodium bicarbonate or isotonic saline should be used both before and after the treatment. It's also crucial to reduce the contrast exposure's frequency and volume[26].
- To find effective pharmacologic medications, confirm new biomarkers for early identification, and comprehend the long-term repercussions of CI-AKI on cardiovascular and renal health, more research is required[4].

5. Bibliography

[1] Ramachandran P, Jayakumar D. Contrast-induced acute kidney injury. Indian Journal of Critical Care Medicine: Peer-reviewed, Official Publication of Indian Society of Critical Care Medicine. 2020 Apr;24(Suppl 3):S122.

[2] Gouveia R, Bravo P, Santos C, Ramos A. Contrast-induced acute kidney injury–a review focusing on prophylactic strategies. Angiologia e Cirurgia Vascular. 2015 Jun 1;11(2):68-78.

[3] Vlachopanos G, Schizas D, Hasemaki N, Georgalis A. Pathophysiology of contrast-induced acute kidney injury (CIAKI). Current pharmaceutical design. 2019 Dec 1;25(44):4642-7.

[4] Li Y, Wang J. Contrast-induced acute kidney injury: a review of definition, pathogenesis, risk factors, prevention and treatment. BMC nephrology. 2024 Apr 22;25(1):140.

[5] Rao R, Limaye A. An Overview of Contrast-induced Acute Kidney Injury. Indian Journal of Clinical Cardiology. 2023 Dec 1;4(4).

[6] Homma K. Contrast-induced acute kidney injury. The Keio Journal of Medicine. 2016;65(4):67-73.

[7] Lun Z, Liu L, Chen G, Ying M, Liu J, Wang B, Liang J, Yang Y, Chen S, He Y, Chung EY. The global incidence and mortality of contrast-associated acute kidney injury following coronary angiography: a meta-analysis of 1.2 million patients. Journal of Nephrology. 2021 Oct;34(5):1479-89.

[8] Solomon R, Dauerman HL. Contrast-induced acute kidney injury. Circulation. 2010 Dec 7;122(23):2451-5.

[9] Theofilis P, Kalaitzidis R. Navigating nephrotoxic waters: A comprehensive overview of contrast-induced acute kidney injury prevention. World Journal of Radiology. 2024 Jun 28;16(6):168.

[10] Ravi k, ganesh k, kumar js, simon s. Wcn24-1124 incidence and risk factors of contrast induced acute kidney injury among patients undergoing coronary and peripheral angiogram in a tertiary care centre. Kidney International Reports. 2024 APR 1; 9(4): S529.

[11] Caiazza A, Russo L, Sabbatini M, Russo D. Hemodynamic and tubular changes induced by contrast media. BioMed research international. 2014;2014(1):578974.

[12] Kusirisin P, Chattipakorn SC, Chattipakorn N. Contrast-induced nephropathy and oxidative stress: mechanistic insights for better interventional approaches. Journal of translational medicine. 2020 Oct 20;18(1):400.

[13] Funaki B. Re: Contrast media-induced nephrotoxicity: identification of patients at risk and algorithms for prevention. Journal of vascular and interventional radiology: JVIR. 2001 Jul 1;12(7):894-.

[14] Katholi RE. Contrast-induced nephropathy-choice of contrast agents to reduce renal risk. Am Heart Hosp J. 2009 Jun 1;7(1):45-9.

[15] Mehran R, Aymong ED, Nikolsky E, Lasic Z, Iakovou I, Fahy M, Mintz GS, Lansky AJ, Moses JW, Stone GW, Leon MB. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. Journal of the American College of Cardiology. 2004 Oct 6;44(7):1393-9.

[16] Rudnick MR, Leonberg-Yoo AK, Litt HI, Cohen RM, Hilton S, Reese PP. The controversy of contrast-induced nephropathy with intravenous contrast: what is the risk?. American Journal of Kidney Diseases. 2020 Jan 1;75(1):105-13.

[17] Hossain MA, Costanzo E, Cosentino J, Patel C, Qaisar H, Singh V, Khan T, Cheng JS, Asif A, Vachharajani TJ. Contrast-induced nephropathy: Pathophysiology, risk factors, and prevention. Saudi Journal of Kidney Diseases and Transplantation. 2018 Jan 1;29(1):1-9.

[18] Hogas SM, Covic A. Contrast Induced Acute Kidney Injury. InContemporary Approaches to Renal Vessels Disorders: Updates and Management Strategies 2024 Nov 5 (pp. 45-59). Cham: Springer Nature Switzerland.

[19] Teo SH, Endre ZH. Biomarkers in acute kidney injury (AKI). Best practice & research Clinical anaesthesiology. 2017 Sep 1;31(3):331-44.

[20] Lameire N, Kellum JA, KDIGO AKI Guideline Work Group. Contrast-induced acute kidney injury and renal support for acute kidney injury: a KDIGO summary (Part 2). Critical Care. 2013 Feb;17:1-3.

[21] Chalikias G, Drosos I, Tziakas DN. Contrast-induced acute kidney injury: an update. Cardiovascular drugs and therapy. 2016 Apr;30:215-28.

[22] McCullough PA, Choi JP, Feghali GA, Schussler JM, Stoler RM, Vallabahn RC, Mehta A. Contrast-induced acute kidney injury. Journal of the American College of Cardiology. 2016 Sep 27;68(13):1465-73.

[23] Gameiro J, Marques F, Lopes JA. Long-term consequences of acute kidney injury: a narrative review. Clinical Kidney Journal. 2021 Mar 1;14(3):789-804.

[24] Horne KL, Viramontes-Hörner D, Packington R, Monaghan J, Shaw S, Akani A, Reilly T, Trimble T, Figueredo G, Selby NM. A comprehensive description of kidney disease progression after acute kidney injury from a prospective, parallel-group cohort study. Kidney international. 2023 Dec 1;104(6):1185-93.

[25] Ng AK, Ng PY, Ip A, Lam LT, Ling IW, Wong AS, Yap DY, Siu CW. Impact of contrast-induced acute kidney injury on long-term major adverse cardiovascular events and kidney function after percutaneous coronary intervention: insights from a territory-wide cohort study in Hong Kong. Clinical Kidney Journal. 2022 Feb;15(2):338-46.

[26] Cossette F, Trifan A, Prévost-Marcotte G, Doolub G, So DF, Beaubien-Souligny W, Abou-Saleh D, Tanguay JF, Potter BJ, Ly HQ, Menkovic I. Tailored Hydration for the Prevention of Contrast-Induced Acute Kidney Injury After Coronary Angiogram or PCI: A Systematic Review and Meta-Analysis. American Heart Journal. 2025 Jan 4.

[27] Briguori C, Colombo A, Airoldi F, Morici N, Sangiorgi GM, Violante A, Focaccio A, Montorfano M, Carlino M, Condorelli G, Ricciardelli B. Nephrotoxicity of low-osmolality versus iso-osmolality contrast agents: impact of N-acetylcysteine. Kidney international. 2005 Nov 1;68(5):2250-5.

[28] Su X, Xie X, Liu L, Lv J, Song F, Perkovic V, Zhang H. Comparative effectiveness of 12 treatment strategies for preventing contrast-induced acute kidney injury: a systematic review and Bayesian network meta-analysis. American Journal of Kidney Diseases. 2017 Jan 1;69(1):69-77.

[29] Bagai J, Beavers CJ. Quality initiatives for prevention of contrast-induced acute kidney injury [Internet]. 2018

[30] Mercado MG, Smith DK, Guard EL. Acute kidney injury: diagnosis and management. American family physician. 2019 Dec 1;100(11):687-94.

[31] González-Nicolás MÁ, González-Guerrero C, Goicoechea M, Boscá L, Valiño-Rivas L, Lázaro A. Biomarkers in contrast-induced acute kidney injury: towards a new perspective. International Journal of Molecular Sciences. 2024 Mar 19;25(6):3438.

[32] Bangalore S, Briguori C. Preventive strategies for contrast-induced acute kidney injury: and the winner is…. Circulation: Cardiovascular Interventions. 2017 May;10(5):e005262.

[33] Moitinho MS, Barbosa D, Galhardo A, Caixeta A, Santana-Santos E, Cunha M, Prado BS, da Fonseca CD. Mehran vs. Mehran2 pre-procedure: which score better predicts risk of contrast-induced acute kidney injury in patients with acute coronary syndrome?. PeerJ. 2025 Apr 15;13:e19166.