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CASE REPORT

Contrast media induced nephropathy: case series and review of the literature focusing on management

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ABSTRACT

BACKGROUND

Contrast media administration during diagnostic and invasive procedures in high risk patients for nephrotoxicity is a common problem in clinical practice. Radiological procedures using intravascular iodinated contrast injection media have been widely used for therapeutic purposes. Contrast-induced nephropathy (CIN) is a serious complication of angiographic procedures and results from administration of contrast media (CM), which increases morbidity and mortality rates.

CASE DESCRIPTION

We present these 10 cases with high risk of CIN and diverse characteristics. A new generation iso-osmolar CM (iodixanol) was administered in these cases. Three of the cases experienced CIN events, where one patient experienced an improvement in his condition, but two other patients experienced complications and eventually died due to the underlying disease. The other 6 cases did not experience CIN after receiving CM, which was due to better preparation beforehand. One patient with a history of regular hemodialysis, underwent immediate post-operative dialysis with CM, and no evaluation of the incidence of CIN was required.

CONCLUSION

Of the 10 cases observed, 3 of them experienced CIN which was caused by the severity of the patient's condition and lack of preparation time before the CM procedure. Management of CIN is complex, starting from the pre-treatment evaluation until 72 hours or more after the CM procedure. This case series suggests that even new generation CM (including iodixanol) may be severely nephrotoxic, when administered to high risk patients. The amount of CM given must be below the maximum limit and adjusted to the patient's condition. Additionally, we review the complex mechanisms involved in management of CM nephrotoxicity.

Keywords: Acute kidney injury, contrast-induced nephropathy, contrast media, management, kidney failure

Abbreviations :

AKI: Acute kidney injury; ALARA: As low as reasonably achievable; BUN: Blood urea nitrogen; CHF: Congestive heart failure; CI-AKI: Contrast-induced acute kidney injury; CIN: Contrast-induced nephropathy; CKD: Chronic kidney disease; CM: Contrast media; CRMI: Cardiac magnetic resonance imaging; CT: Computed tomography; DM: Diabetes mellitus ; eGFR: Estimated glomerular filtration rate; ESKD: End-stage kidney disease; F: female; HCT: hematocrit ; HD: Hemodialysis; HOCM: Hyperosmolar contrast media; IPC: Ischemic preconditioning; IV: Intravenous; KDIGO: Kidney disease improving global outcomes; IOCM: iso-

osmolar contrast media; LOCM: Low osmolar contrast media; M: Male; MACD: Maximal allowable contrast dose; mOsm/kg: Milliosmol/kilogram; MRI: Magnetic resonance imaging; NAC: N-Acetylcysteine; NaCl: Sodium chloride; Na+/K+-ATPase: Sodium-potassium adenosine triphosphatase; NM/PET: Nuclear medicine/positron emission tomography; NYHA: New York heart association; PCI: Percutaneous coronary intervention; RRT: Renal replacement therapy; SBP: Systolic blood pressure; SC: Serum creatinine; US: Ultrasound

INTRODUCTION

Each year more than 80 million contrast studies are conducted worldwide by a variety of medical specialties.[1] Contrast media (CM) is used in imaging techniques to increase the difference between body tissues in the images. The ideal contrast media should reach a very high concentration in the tissues without causing adverse effects.[2] Contrast media is used for diagnostic imaging including computed tomography (CT), magnetic resonance imaging (MRI), ultrasound (US), nuclear medicine/positron emission tomography (NM/PET), coronary angiography, echocardiography, and cardiac magnetic resonance imaging (CMRI).[3]

Intravascular contrast media is a concentrated tri-iodinated benzene compound that is radio-opaque as a result of iodine group bonds. All CM agents are cytotoxic and this cytotoxicity can be aggravated by the ionic strength and osmolarity or viscosity of each specific agent.[4] Low-osmolar contrast media (LOCM) or iso-osmolar CM (IOCM) are recommended to be used to reduce the cytotoxic effects on patients.[5]

The use of radiographic contrast media is responsible for 12% of kidney insufficiency cases occurring in hospitals and is the third most common cause of acute kidney injury (AKI) after kidney hypoperfusion (42%) and postoperative kidney injuries (18%). The incidence of contrast-induced nephropathy (CIN) reported after percutaneous coronary intervention (PCI) varies between 0 and 24%, depending on the prevalence of associated risk factors, with higher incidence reported after emergency PCI. The development of CIN is associated with long hospital stays, increased morbidity and mortality, in addition to higher costs.[6]

Our reason for raising this topic is to increase overall awareness of the importance of preventing CIN in patients undergoing procedures with CM. Various conditions and comorbidities in patients require adjustments in evaluation and intervention, necessitating involvement of various experts such as cardiologists, nephrologists, and radiologists.

The aim of this case series was to add to the literature on CIN, particularly regarding its management. On the other hand, we review the currently available experimental and clinical data on the mechanisms involved in pathogenesis of CIN, because we consider that these mechanisms deserve significant interest for the development of new perspectives on the prevention of CIN in the near future.

CASE REPORT

In this case series will be reported 10 patients with high or very high-risk of CIN. Two patients were planned to undergo elective PCI, 4 patients were planned to undergo primary PCI, 3 patients were planned to undergo CT scan with contrast, 1 patient was planned to undergo arteriography, and 1 patient was planned to undergo venography. Some patients received different treatments based on the underlying disease of the patients. Most of the patients had a “standard regimen therapy” with NaCl 0.9% 1-1.5 mL/kg BW/h from 12 hours before and up to 12 hours after the procedure, with oral acetylcysteine administration 600 mg every 12 hours starting 24 hours before until 24 hours after the procedure.

In case no.5, an adult male, 54 years old, at very high risk of CIN, underwent primary PCI. There was no time to give the patient fluid therapy before the contrast procedure, and administration of oral acetylcysteine 600 mg every 12 hours was started after the procedure. This patient had a CIN event and was in an unstable hemodynamic condition, experiencing cardiac arrest twice during treatment and dying 24 hours after the procedure.

In case no 6. an adult male, 57 years old, at very high risk of CIN, underwent primary PCI. There was no time to give the patient fluid therapy before the contrast procedure, and administration of oral acetylcysteine 600 mg every 12 hours was

started after the procedure. The patient had a CIN event with increase in serum creatinine (SC) levels and accompanied by oliguria. The patient was planned to undergo hemodialysis (HD), but unfortunately the patient died of heart failure before HD could be performed.

In case no 7. an adult male, 54 years old, at high risk of CIN, underwent primary PCI. Due to time constraints, the patient was given hydration therapy with NaCl 0.9% 1-1.5 mL/kgBW/h from 6 hours before to 12 hours after the procedure and oral acetylcysteine 600 mg every 12 hours to 24 hours after the procedure. The patient had no CIN event.

In case no. 9, an adult male, 77 years old, with stage V chronic kidney disease (CKD) (kidney failure, also known as end-stage kidney disease or ESKD), at high risk of CIN, underwent venography with CM. The patient was given hydration therapy at a minimum dose of 0.5 mL/kgBW/h, considering that the patient had anuria. The patient received oral acetylcysteine 600 mg every 12 hours from 24 hours before until 24 hours after the procedure. Hemodialysis was performed as soon as the patient completed the procedure (Table 1).

Table 1. Patient characteristics

Age (years)	Gender	Risk of CIN	Type of medical procedure	Received "standard regimen therapy"	CIN event
66	F	High risk	Elective PCI	Yes	No
75	F	High risk	Elective PCI	Yes	No
48	M	High risk	Abdominal CT scan	Yes	No
52	M	High risk	Head CT scan	Yes	No
54	M	Very high risk	Primary PCI	No	Yes
57	M	Very high risk	Primary PCI	No	Yes
54	M	High risk	Primary PCI	No	No
64	F	High risk	Arteriography	Yes	No
77	M	High risk	Venography	No	Not Evaluate
78	M	High risk	Midface CT scan	Yes	Yes

Note: PCI: Percutaneous coronary intervention, CT: Computed tomography, CIN: Contrast-induced nephropathy, F : female, M: male

The image above illustrates the course of serum creatinine monitoring in all patients from before undergoing the procedure with contrast media until several days after the procedure.

After the contrast media procedure, serum creatinine levels in all patients were evaluated, generally starting from 12 hours to 72 hours after the procedure, except for patients on regular HD who did not require monitoring and underwent dialysis immediately after the contrast procedure. In some patients at very high risk HD can be started as early as 6 hours after the procedure. Serum creatinine levels before the procedure are used as a reference if there is an increase in serum levels after the procedure that meets the criteria for CIN.

Patients who received hydration and therapy as recommended had a better prognosis as indicated by the absence of CIN in these patients. In contrast, patients no. 5 and 6 who were not able to be given sufficient hydration according to protocol had a poor prognosis. Patient no. 9 was a patient with late-stage kidney disease who had undergone regular HD and no repetition of SC measurements after the contrast procedure. Patient no. 10 initially showed an increase in SC, but experienced improvement along with the continuation of the therapeutic regimen (Figure 1).

The image above illustrates the process of monitoring urine production in all patients from before the contrast media procedure until several days after the procedure.

The patients' urine production after the procedure was evaluated for signs of acute kidney injury caused by the contrast procedure. Monitoring is generally carried out up to 72 hours after the procedure. In this report most patients experienced a decrease in urine production in the first 24 - 48 hours and improved thereafter. Of the three patients who experienced CIN, 2 patients could not be evaluated because they died within 24 hours due to cardiac complications, but the other one experienced improvement in urine production after 48 hours. Patient number 9 had anuria due to CKD stage V and was on regular HD.

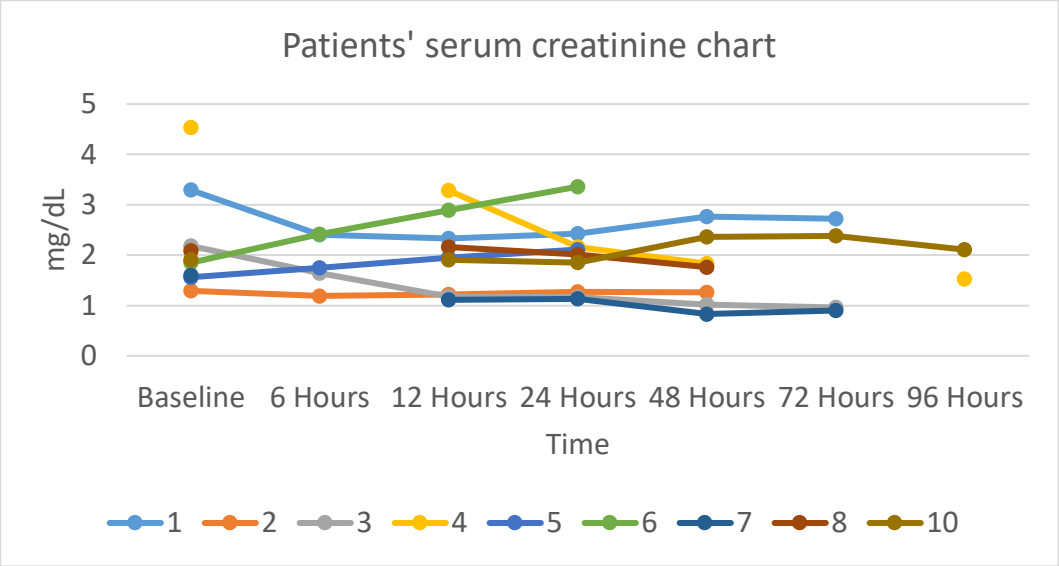

Figure 1. Creatinine serum travel chart of the patients (n=9)

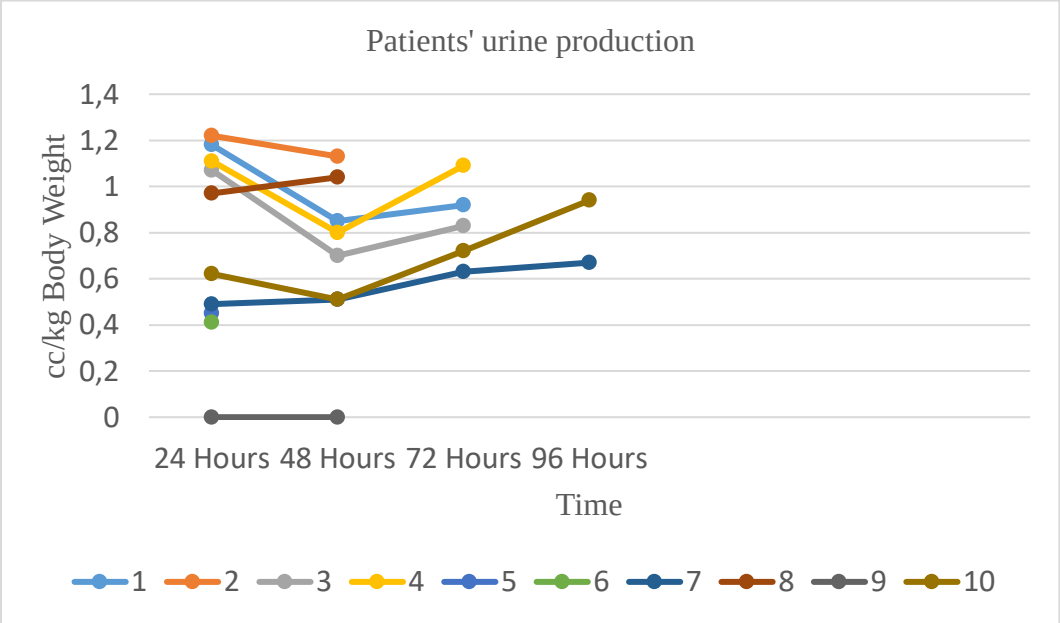

Figure 2. Patients' urine output travel chart (n=10)

Patients who received hydration and therapy according to recommendations had a good prognosis with adequate urine production. However, in patients number 5 and 6, there was time to repeat measurement of the amount of urine production. Patient number 9 had late-stage kidney disease with anuria (Figure 2). Informed consent was obtained from the study subjects, and this study was conducted in accordance with the Declaration of Helsinki. At our institution, ethical approval is not required for a case report.

DISCUSSION

Contrast media overview

A common indication for the use of CM in medical imaging is as a diagnostic and therapeutic tool. Type, osmolality, molecular structure, and viscosity of CM are important determinants of nephrotoxicity events. Contrast media is divided into 3 categories based on osmolarity (Table 2). Hyperosmolar contrast media (HOCM) has been proven to cause CIN more frequently than low

osmolar CM (LOCM). A meta-analysis mentions that IOCM has a lower nephrotoxic risk compared to LOCM although the difference is not significantly meaningful.[6] In the present case series, all patients received LOCM, i.e., iopamidol or iopromide. In patients with PCI and arteriographic measures, CM is injected through the arteries, while in patients undergoing CT scans CM is injected intravenously.

Contrast induced nephropathy

In the Kidney Disease Improving Global Outcomes (KDIGO) guideline, a slightly different term is used with the same meaning as CIN, *i.e.* contrast-induced acute kidney injury (CI-AKI), which is described as acute renal injury, characterized by an increase in SC of >0.5 mg / dL (444 mmol/L), or an increase in SC of >25%, or a decrease in eGFR of >25%, or a combination of all three definitions, in patients after exposure to intravascular contrast media, and after elimination of the possibility of other AKI causes.[8]

Contrast-induced nephropathy is defined as absolute (≥0.5 mg/dL, ≥44 μmol/L) or relative increase (≥25%) in initial creatinine serum value (SCr) at 48 to 72 hours after exposure to contrast media, in the absence of alternative causes.[12] In most cases CIN is reversible and improves on its own. Serum creatinine levels generally peak within 3-5 days and gradually return to their initial levels within 7-10 days, but some patients may develop acute kidney injuries that require dialysis. The medical team that plays the role of CIN prevention is mainly the nephrologist and the radiologist. [6,9] Contrast-induced nephropathy risk is divided into 4 categories based on the Mehran score, namely low risk, moderate risk, high risk, and very high risk, determined by patients' comorbidities.[10] In this case report, 3 patients were diagnosed with CIN. One patient (case no.10) improved with the standard therapeutic regimen, but in the other two patients (cases no. 5 and 6) their condition worsened before they received renal replacement therapy (RRT).

The pathogenesis of contrast-induced nephropathy has not been clearly understood and the presence of direct and indirect effects, as well as hemodynamic disorders, play a role in CIN (Figure 3).

The direct cause of CIN is the exposure of the renal tubular epithelium to nephrotoxic contrast agents, causing apoptosis and necrosis. Initially tubular epithelial injury is characterized by loss of cell polarity due to redistribution of Na+/K+-ATPase. As cellular injury progresses, epithelial cells detach from the basement membrane and cause luminal obstruction, increased intratubular pressure, and ultimately decreased glomerular filtration rate. The indirect effect of contrast agents involves ischemic injury from a decrease in regional or global perfusion. Contrast agents can cause locally mediated intrarenal vasoconstriction by vasoactive substances such as endothelin, nitric oxide, and prostaglandins, resulting in reduced glomerular blood flow and reduced oxygen delivery to metabolically active parts of the nephron. In addition, contrast agents increase blood viscosity, leading to a further decrease in the flow of microcirculation and changes in blood osmolality, which can damage the plasticity of erythrocytes and may increase the risk of microvascular thrombosis.[11]

Table 2. Types and physicochemical properties of contrast media [7]

Type	Name	Osmolarity (mOsm/kg water)	Ionicity	Mono/ Dimer	Amount of iodine (mg/mL)	Iodine ratio	Viscosity (mPa.s at 37^0C)
HOCM	Iothalamate	1695	Ionic	Monomer	400	3:2	2.8
LOCM	Iomeprol	720	Nonionic	Monomer	350	3:1	4.8
LOCM	Iopentol	683	Nonionic	Monomer	310	3:1	6.5
LOCM	Iopromide	586	Nonionic	Monomer	350	3:1	4.7
LOCM	Iopamidol	653	Nonionic	Monomer	300	3:1	4.6
LOCM	Ioversol	719	Nonionic	Monomer	320	3:1	6
LOCM	Iohexol	667	Nonionic	Monomer	300	3:1	5.7
LOCM	Ioxaglate	584	Ionic	Dimer	320	3:2	7.8
IOCM	Iodixanol	290	Nonionic	Dimer	320	6:1	11.4

Note: HOCM: hyperosmolar contrast media; LOCM: low osmolar contrast media; IOCM: iso-osmolar contrast media; mOsm/kg: milliosmol/kilogram, mPa.s : millipascal second

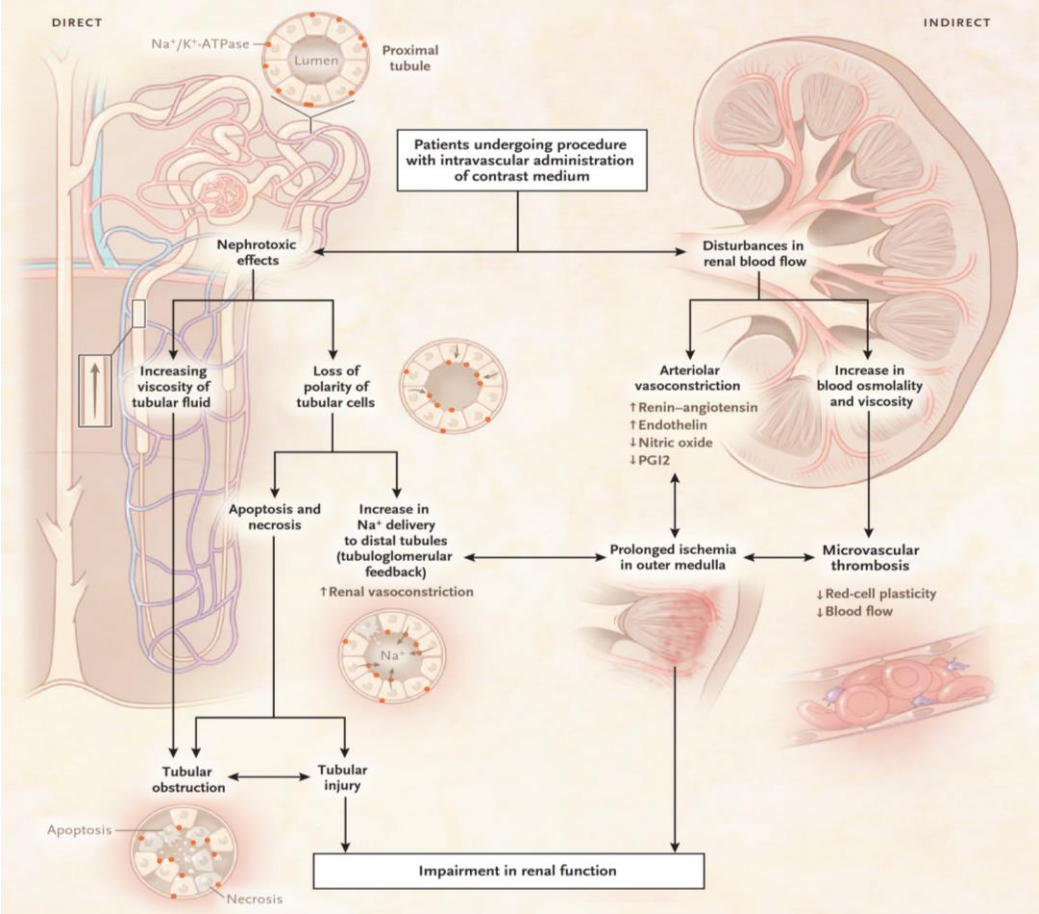

Figure 3. Pathogenesis of contrast-induced nephropathy [11]

Management

There is currently no definitive treatment available for CIN. Therefore, the benefits for CM-based diagnostic studies or intervention procedures should always be weighed against the risk of CIN. In addition, repeated CM exposure in a short period of time should be avoided wherever possible.[7] The management of patients with a high risk of CIN occurrence is preventive and, begins prior to CM exposure, and requires good peri- or pre-, intra-, and post-procedural preparation. However, in emergency cases the pre-procedural preparation cannot be given to the maximum benefit of the patient.

Before the clinical procedure is started, the clinician should assess the loss and benefit to the patient if exposed to CM. If CM exposure is judged to result in greater loss or does not change the therapy for the patient, the clinic should reconsider the indication of the CM administration. When it is decided that the patient will get CM, the status of the patient in stratification through the scoring system that is currently used is the Mehran score. Stratification of risk before contrast is given to the patient should be fully assessed and precautions should be taken in patients with decreased renal function. Implementation of preventive strategies is considered the best approach to suppress CIN events.[8,10]

Risk factors can be grouped into procedure-related and patient-related risk factors. Procedures that can increase the risk of CIN occurrence include repeated use of CM within 24 hours, intra-arterial CM injection, high CM volume, and high CM osmolarity.[12]

Risk factors of CIN can be divided into fixed and modifiable risk factors. In the management and prevention of CIN, several risk factors that can be modified before the procedure are expected to prevent the occurrence of CIN after the procedure (Table 3).

After identifying risk factors, an essential management step is to know the principles in the use of contrast materials themselves, including the use of the lowest possible contrast volume and the

selection of contrast agents with the lowest toxicity value, especially in patients at high risk of CIN. This principle goes by the name of "as low as reasonably achievable" (ALARA).[14] The maximum dose of contrast media allowed is called maximal allowable contrast dose (MACD) and is calculated with the formula 5 mL x body weight (in kg)/serum creatinine (in mg/dL), with a maximum dose not exceeding 300 mL.[15] Low CM doses are defined as <30-125 mL or <5 mg/kg, are minimally nephrotoxic, and are associated with a lower risk of CIN. However, AKI can even occur with a small volume of CM (30 mL).[16] In this case series report, ALARA principles have been applied in the provision of CM. All patients received a contrast volume below 100 mL using LOCM contrast.

Table 4 describes the Mehran CIN risk score components and their respective values, with risk stratification according to punctuation. The Mehran CIN risk score has 8 components with different values for each one. The final punctuation gives us the estimated risk of CIN, congestive heart failure, estimated glomerular filtration rate, intra-aortic balloon pump (Table 4).[17] The Roxana Mehran score predictor applies the following ten variables: a risk score of less than 6 carries a risk of 7.5% and more than 16 carries up to 57% risk.[18]

There is currently no specific treatment for CI-AKI once the injury occurs, therefore it is essential to perform risk stratification and implement all measures to prevent CIN in selected patients. The main prophylactic strategies include: reduction of modifiable risk factors (anemia, hypotension, use of nephrotoxic drugs), reduction of CM exposure, and peri-procedural oral or intravenous hydration (Figure 4). In addition, treatment with acetylcysteine, ascorbic acid, and statins have been evaluated over the years with inconsistent results.

Table 3. Patient-related CIN risk factors [13]

Fixed risk factors	Modifiable risk factors
Chronic kidney disease	Hypotension
Diabetes mellitus	Anemia
Congestive heart failure	Nephrotoxic drugs
Age	Hypercholesterolemia
Female gender	Pre-procedural dehydration/ hypovolemia
	Pre-procedural hyperglycemia
	Contrast media type and volume

Table 4. Mehran score [17]

Risk factors	Score
Hypotension (SBP <80 mmHg or >1 hour with inotropic)	5
Intra-arterial balloon pump therapy (IABP)	5
Congestive heart failure (NYHA III/IV or pulmonary edema)	5
Age >75 years	4
Diabetes mellitus	3
Anemia (male: HCT<0.39, female HCT<0.36)	3
Estimated glomerular filtration rate <20 mL/minute	6
Estimated glomerular filtration rate 20-40 mL/minute	4
Estimated glomerular filtration rate 40-60 mL/minute	2

Contrast media volume			1 per 100 cc
<5	6-10	11-16	>16
7.5%	14%	26.1%	57.3%
0.04%	0.12%	1.09%	12.6%
Light	Intermediate	High	Very high

Note: SBP: systolic blood pressure; NYHA: New York Heart Association; HCT: hematocrit

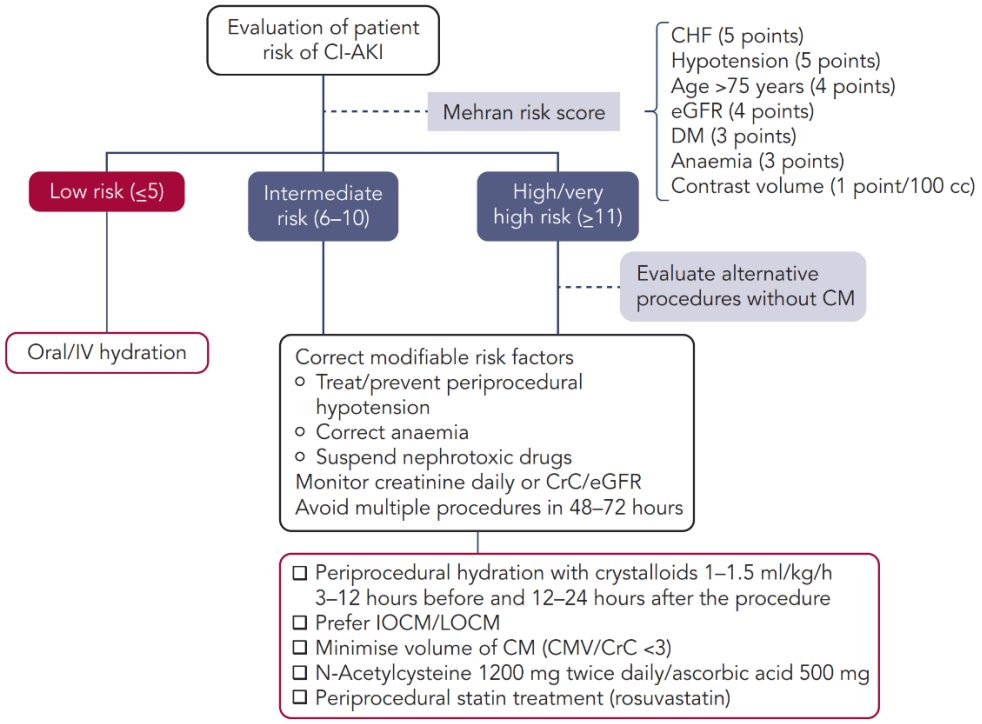

Figure 4. Patient risk assessment and management flowchart for CIN.[13]

When patients are categorized as high risk, there are several approaches that can be taken. The approaches can be classified into nonpharmacological and pharmacological. Non-pharmacological approaches include re-evaluation of possible alternative procedures, reviewing and considering discontinuation of drugs considered nephrotoxic, optimization of CM use, advanced assessment of post-procedural renal function, coronary sinus contrast removal system, remote ischemic preconditioning (IPC).[19] Drugs categorized as nephrotoxic should be stopped at least 2 days before the CM procedure.[20]

Volume expansion is a technique of administering liquids to prevent the occurrence of CIN. Kidney disease improving global outcomes does not recommend oral hydration, but recommends expanding volume with a solution of isotonic sodium chloride or sodium bicarbonate, with intravenous administration in patients at increased risk of CIN.[11] Expansion is believed to work on two things. First, intravascular space expansion and second, intravenous fluid replacement is believed to weaken the direct toxic effects of contrast agents on tubular epithelial by lowering the concentration and viscosity of contrast between tubular lumens. This weakening effect is the result of inhibition of volume mediation from the presence of proximal tubular sodium and water reabsorption as well as decreased contact time with contrasting materials related to increased tubular flow.[19]

Current clinical practice guidelines and consensus statements recommend intravenous hydration (IV) with an isotonic 0.9% NaCl of 1.0–1.5 mL/kg/h starting 3–12 hours before the CM procedure and continuing for 12–24 hours after CM exposure. For a same-day procedure, faster hydration with 3 mL/kg/h can be used at least 1–3 hours before and 6 hours after the procedure.[13] In this case report all patients received hydration with isotonic solution, but there was an incredibly significant difference in start time and hydration dose in cases number 5 and 6 which were directly proportional to the poor prognosis in those patients. In the case of number 9, this patient with a history of regular HD and anuria, was clearly unable to excrete optimal contrast through urine, therefore hydration was not administered according to standards and CM elimination function is replaced with HD immediately after procedure.

Pharmacological management includes the use of drugs to prevent the occurrence of CIN. One of the KDIGO recommended drugs is N-Acetylcysteine (NAC), because the use of antioxidant agents has been evaluated for many years. N-acetylcysteine (NAC) is a scavenger of reactive oxygen species and improves vasodilation.[11] The recommended standard oral

NAC regimen consists of 600 mg twice a day 24 hours before and up to 24 hours post procedure.[21] All patients in this case report were given NAC according to the standard dose. Several other drugs that are believed to prevent CIN include ascorbic acid and statins but were not given to the patients in this case report. In monitoring all patients who are determined to be at risk of CIN, these patients must undergo the essential measurements of serum creatinine levels (SC) after exposure to CM and measurement of urine production and fluid balance.

The optimal dialysis time for AKI is not specified definitely. In current practice, the decision to start renal replacement therapy (PRC) is most often based on a clinical picture of excess volume and a biochemical picture of an imbalance of dissolved substances (azotemia, hyperkalemia, severe acidosis). Therefore KDIGO recommends starting the RRT immediately when there is a life-threatening change in fluid, electrolyte, and acid-base balance conditions considering the broader clinical context, assessing the presence of conditions that can be improved with the RRT, and not seeing an increase in BUN and SC alone in deciding to institute RRT.[8]

In this case series, in case number 9, the patient had previously undergone regular HD. As soon as the patient was exposed to CM, the patient was immediately given HD to prevent excessive exposure to CM. In patients already undergoing dialysis, frequently cited problems with contrast administration include volume load and direct contrast toxicity to nonfunctional nephrons and remaining nonrenal tissue. These problems underlie the perceived need for emergency dialysis and contrast elimination. CM can be removed effectively and efficiently with HD. High flux dialysis membrane with blood flow between 120-200 mL / minute can eliminate almost 50% of CM in one hour and 80% in 4 hours. Even in patients with CKD, where contrast excretion is delayed, 70-80% of the contrast can be eliminated with HD therapy for 4 hours. Dialysis immediately after contrast administration has been advised for patients who are already using long-term HD and for those at very high risk of CIN. Three studies mentioned that LOCM can be safely administered to patients with late-stage CKD.[21]

The recommendation in this case series is that the approach in patient management with high risk of CIN is highly individual, with a look at the background and condition of the disease. The experience of the clinicians plays a role in decision making about the therapeutic plan and procedures in each patient.

CONCLUSION

Contrast-induced nephropathy is an ever-growing clinical problem. Until now preventive management is the main in avoiding CIN in patients, consisting of non-pharmacological and pharmacological management. Rigorous monitoring after the CM procedure plays an important role for clinicians in determining the next course of procedures for the patients. There are no standard HD guidelines in CIN, but under some conditions that are considered life-threatening, it can be decided to apply HD while paying attention to the "cost and benefit" of the procedure itself.

Conflict of Interest

There is no conflict of interest in this paper

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Author Contributions

PAM designed and collected data. PAM, YK, GWM, NPA compiled and analyzed the data. PAM, GWM wrote the manuscript. All authors have read and approved the final manuscript.

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Data Availability Statement

The data used to support the findings of this study is available from the corresponding author upon request.

Declaration of Use of AI in Scientific Writing

Nothing to declare.

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