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Contrast-Induced Nephropathy Following Coronary Angiography and Percutaneous Coronary Intervention: Incidence and Associated Risk Factors

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Abstract

Background: Contrast-induced nephropathy (CIN) is a common complication associated with coronary angiography and percutaneous coronary intervention (PCI). It can result in acute renal impairment, extended hospitalization, and poor clinical results. This study will determine its prevalence and evaluate three categories of risk variables at the demographic, clinical, and procedural levels, with a particular emphasis on contrast medium dosage, diabetes, and renal function indicators.

Methods: A prospective observational cohort study included 150 adult patients having non-compulsory or acute coronary angiography and/or PCI at a tertiary care facility. Baseline

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demographic and medical records, including diabetes status, high blood pressure, serum creatinine, and estimated glomerular filtration rate (eGFR) determined using the CKD-EPI equation, had been acquired. All patients received standard prophylactic hydration with intravenous 0.9% saline before and after contrast injection. Serum creatinine levels were retested 24 hours following the surgery. CIN is characterized as a rise in serum creatinine by ≥0.5 mg/dL or ≥25% increase from baseline within 24 hours after comparison publicity. Statistical analyses were carried out in R Studio model 4.3 using Chi-square tests, impartial-samples t-tests, Spearman's rank correlation, and multivariable logistic regression.

Results: The overall CIN incidence was 58.0% (87/150). Diabetic individuals experienced CIN substantially more frequently than non-diabetic patients (66% vs. 46%, $p = 0.014$). Diabetic patients were older, had a higher prevalence of hypertension, got more contrast media, and had larger decreases in eGFR after the surgery. Contrast volume had a significant positive connection with post-procedural serum creatinine increases ($\rho = 0.65$, $p < 0.001$). After adjusting for confounding variables, multivariable logistic regression revealed that higher contrast volume and lower baseline eGFR were independent predictors of CIN, while diabetes mellitus was not.

Conclusion: CIN is a common complication for individuals undergoing coronary angiography and PCI. Increased contrast volume and poor baseline renal function were independent predictors of CIN. These findings highlight the relevance of pre-procedural renal risk classification and the appropriate use of contrast media in reducing the development of CIN and improving patient outcomes.

INTRODUCTION

The diagnostic and treatment methods for ischemic heart disease require enhancements because the condition remains one of the primary reasons people die around the world (Zaman *et. al.,* 2025). Coronary angiography and percutaneous coronary intervention (PCI) are widely used diagnostic and therapeutic procedures for the management of coronary artery disease and acute

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coronary syndromes, providing effective coronary revascularization and improving patient outcomes (Rao *et al.,* 2025; Lawton *et al.,* 2022; Ota *et al.,* 2018).

PCI procedure success relies on doctors using iodinated contrast media which they inject into patients through arterial veins even though it can lead to severe health risks. Patients with complex coronary lesions require higher contrast media doses because their advanced age makes their health condition more dangerous (McCullough *et al.,* 2016). The medical community now faces a significant challenge from contrast-induced nephropathy which medical experts refer to as CIN and contrast-associated acute kidney injury which they call CA-AKI because these conditions increase both post-procedural morbidity and mortality rates (Malik *et al.*, 2025). Contrast-induced nephropathy exists as a medical condition which leads to sudden kidney function decline with the requirement that patients develop a serum creatinine increase of 0.5 mg/dL or more, or a baseline creatinine rise of 25% or more within 24 hours after receiving contrast material (Mehran *et al.,* 2004; KDIGO, 2012).

Acute coronary syndrome (ACS) is a key spectrum of cardiovascular disorders that cause significant morbidity and mortality worldwide. Currently, percutaneous coronary intervention (PCI) is the primary diagnostic and revascularization method for patients presenting with ACS. Despite its established therapeutic efficacy, contrast-induced nephropathy (CIN)—also known as contrast-induced acute kidney injury (CI-AKI)—has arisen as a serious iatrogenic consequence in patients receiving PCI. The European Society of Urogenital Radiology (ESUR) defines contrast-induced nephropathy (CIN) as an absolute increase in serum creatinine of ≥44.2 µmol/L (≥0.5 mg/dL) or a relative increase of ≥25% from baseline within 48 to 72 hours after intravascular administration of iodinated contrast media (Jiang *et al.,* 2022; Pranata & Wahyudi, 2024). Epidemiologically, CIN accounts for a significant proportion of hospital-acquired acute kidney injury cases, trailing only reduced renal perfusion and postoperative complications as the third main cause of in-hospital renal dysfunction. The development of post-PCI CIN severely impairs the patient's prognosis and quality of life, resulting in prolonged functional decline and increased patient concern.

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Furthermore, the clinical manifestation of CIN is directly related to adverse clinical endpoints such as extended hospitalization, increased healthcare costs, an increased risk of developing major adverse cardiovascular events (MACE), and an increased likelihood of requiring long-term kidney replacement therapy (KRT) (Keaney *et al.,* 2013). Moreover, significant registry data demonstrates that the occurrence of CIN is a strong, independent predictor of both short- and long-term all-cause mortality, underscoring the significance of rigorous pre-procedural risk assessment and prophylactic actions in catheterization laboratories (Perrin *et al.,* 2012; Pranata & Wahyudi, 2024). CIN's underlying pathophysiology is complex, with a delicate balance of direct cellular cytotoxicity and severe hemodynamic alterations in the renal microvasculature. The intravascular delivery of iodinated contrast media results in a characteristic biphasic hemodynamic response in the kidneys. Because of their elevated osmolarity and hyperviscosity, these medicines generate an initial, transitory phase of vasodilation, followed by a chronic, severe vasoconstriction mediated by vasoactive factors such as endothelin and adenosine (Moitinho *et al.,* 2020).

Chronic intrarenal vasoconstriction greatly lowers local blood flow, leaving renal tissue vulnerable to severe ischemia and hypoxia. Because of its low baseline oxygen tension and the high metabolic demands necessary for active solute transport, the renal medulla is especially susceptible to hypoxia. Furthermore, contrast media cause direct cytotoxicity in renal proximal tubular epithelial cells. This exposure causes cellular vacuolization, oxidative stress via the production of reactive oxygen species (ROS), disruption of intracellular transport, and, eventually, cellular death by apoptosis and necrosis (Perrin *et al.,* 2012; Pranata & Wahyudi, 2024). Combining these factors makes clinical detection of CIN challenging; its earliest symptoms are typically moderate or do not cause evident acute discomfort, leading doctors to miss the early stages of renal impairment. Despite decades of clinical trials exploring different pharmacological methods, there is presently no feasible definitive treatment for established CIN (Keaney et al., 2013). As a result, because therapeutic options are only supportive once injury has occurred, early identification of high-risk groups and aggressive application of prophylactic strategies—such as

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optimal intravenous hydration—remain the absolute cornerstones for improving the clinical prognosis of patients undergoing PCI (Pranata & Wahyudi, 2024).

Therefore, the present study was conducted to determine the incidence of contrast-induced nephropathy and to evaluate the demographic, clinical, and procedural factors associated with its development among patients undergoing coronary angiography and/or percutaneous coronary intervention (PCI).

Materials and Methods

Study Design

The researchers executed their study through a prospective observational cohort study design. The selected design enables researchers to monitor postoperative results through their active observation of actual clinical outcomes which involve measuring serum creatinine levels after patients receive iodinated contrast media in a real medical environment.

Study Setting and Duration

The research took place at the Department of Cardiology and the Cardiac Catheterization Laboratory of a tertiary care hospital. The data collection phase lasted between six to eight months which provided enough time to recruit patients and observe procedures and complete 48-hour post-procedural monitoring for all participants.

Study Population and Sample Size

The study included adult patients who visited the cardiology department to receive diagnostic coronary angiography or percutaneous coronary intervention (PCI) procedures. The calculated sample size for this study was 150 patients. The researchers separated the study participants into two groups for their comparison purpose by creating a diabetic group and a non-diabetic group. The researchers used non-probability consecutive sampling method to select participants who met the study requirements throughout the research duration until they reached their desired sample size.

Data Collection Procedure

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A systematic proforma was used to capture baseline clinical and demographic information, such as age, sex, body mass index, comorbidities, and current drug use. Serum creatinine and blood urea nitrogen were measured from pre-procedural venous blood samples, and the CKD-EPI equation was used to estimate the glomerular filtration rate. In accordance with institutional procedure, all patients received routine intravenous hydration using 0.9% normal saline. During coronary angiography and percutaneous coronary intervention, procedural factors such as contrast type, contrast volume, procedure duration, and procedural complexity were recorded.

Following the procedure, patients were moved to the recovery ward or cardiac care unit and given post-procedural hydration in accordance with clinical recommendations. Absolute and relative variations from baseline blood creatinine were used to define contrast-induced nephropathy, and follow-up serum creatinine was evaluated at 24 hours.

Ethical Considerations

The study was approved by the Ethical Committee of Abasyn University, Peshawar, and was conducted in accordance with its ethical guidelines and regulations.

Statistical Analysis

Data were compiled and analyzed using RStudio (Version 4.3). Descriptive statistics were calculated, and differences between groups were assessed using appropriate statistical tests. A *p*-value of < 0.05 was considered statistically significant.

Results

A total of 150 patients undergoing coronary angiography and/or percutaneous coronary intervention (PCI) were enrolled in this study. Baseline Characteristics The baseline demographic and clinical characteristics of the study participants stratified by diabetic status (Table 1).

Out of 150 patients, 91 (60.7%) were diabetic and 59 (39.3%) were non-diabetic. Compared to non-diabetic patients, diabetic patients were significantly older (63.8 ± 7.1 vs 59.6 ± 8.6 years, $p = 0.003$), had a higher prevalence of hypertension (87% vs 56%, $p < 0.001$), and more frequently underwent combined angiography with PCI (75% vs 49%). They also received a significantly higher

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volume of contrast media (167.8 ± 91.0 mL vs 106.1 ± 70.1 mL, $p < 0.001$) and had lower baseline eGFR (84.4 ± 22.8 vs 111.1 ± 134.3 mL/min/1.73m^2, $p = 0.007$).

Table 1. Baseline Characteristics of the Study Participants

Characteristic	Overall (N = 150)	Non-Diabetic (N = 59)	Diabetic (N = 91)	p-value
Age (years)	62.1 (8.0)	59.6 (8.6)	63.8 (7.1)	0.003
Gender				0.048
Male	47 (31%)	13 (22%)	34 (37%)	
Female	103 (69%)	46 (78%)	57 (63%)	
Weight (kg)	71.2 (9.9)	70.5 (11.9)	71.6 (8.5)	>0.9
Height (cm)	165.9 (3.5)	166.7 (3.1)	165.3 (3.6)	0.017
Hypertension	112 (75%)	33 (56%)	79 (87%)	<0.001
Congestive Heart Failure	10 (6.7%)	3 (5.1%)	7 (7.7%)	0.7
LVEF (%)	58.0 (7.8)	59.4 (7.4)	57.1 (8.0)	0.044
Procedure Type				<0.001
Angiography	49 (33%)	30 (51%)	19 (21%)	
PCI	4 (2.7%)	0 (0%)	4 (4.4%)	
Angio + PCI	97 (65%)	29 (49%)	68 (75%)	
Contrast Volume (mL)	143.6 (88.5)	106.1 (70.1)	167.8 (91.0)	<0.001
Baseline Creatinine (mg/dL)	0.9 (0.4)	0.9 (0.3)	0.9 (0.4)	0.5
Baseline eGFR (mL/min/1.73m^2)	94.9 (86.6)	111.1 (134.3)	84.4 (22.8)	0.007
CIN	87 (58%)	27 (46%)	60 (66%)	0.014

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Incidence of Contrast-Induced Nephropathy (CIN)

Contrast-induced nephropathy (CIN) affected 87 out of 150 patients, or 58.0% of the total. CIN occurred in 66.0% (60/91) of diabetic patients and 45.8% (27/59) of non-diabetic individuals when stratified by diabetes status. There was a statistically significant difference ($p = 0.014$) as shown in table 2.

Table 2. Overall Incidence of Contrast-Induced Nephropathy and Distribution by Diabetic Status

Characteristic	Total (N = 150)	Non-Diabetic (N = 59)	Diabetic (N = 91)	p-value
CIN, n (%)	87 (58.0)	27 (45.8)	60 (66.0)	0.014

Correlation between Contrast Volume and Post-procedural Serum Creatinine Change

There was a strong positive monotonic correlation between the total volume of contrast media administered and the change in serum creatinine at 24 hours post-**procedure (Spearman's $\rho = 0.65$, $p < 0.001$) (Figure 1).**

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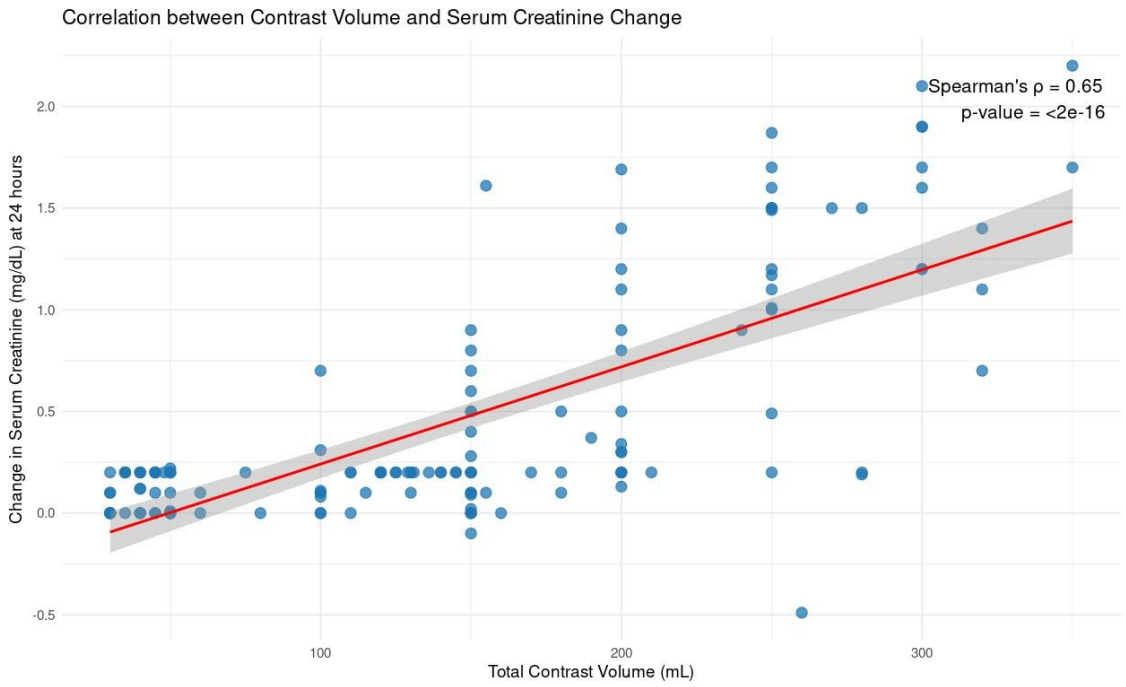

Figure 1: Correlation between Contrast Volume and Serum Creatinine Change

Early Renal Function Changes (eGFR) between Diabetic and Non-Diabetic Patients Diabetic

Patients experienced a greater reduction in eGFR at 24 hours following contrast exposure compared to non-diabetic patients. As shown in the violin plot, the diabetic group demonstrated a wider distribution and a more pronounced negative shift in eGFR change, indicating more severe early renal impairment in diabetic patients (**Figure 2).**

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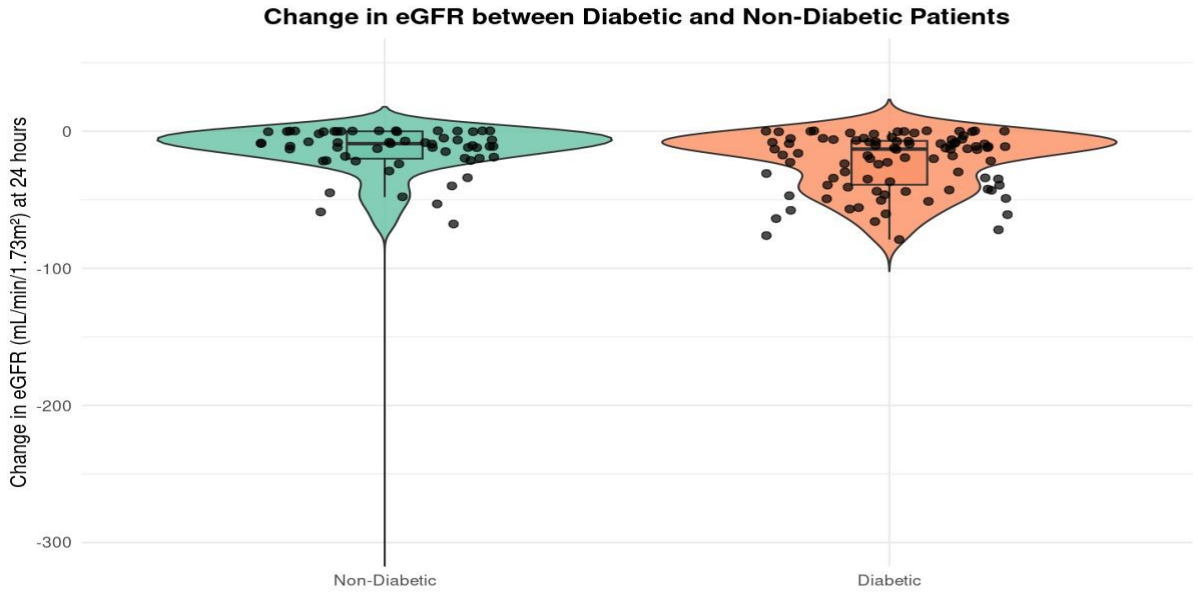

Figure 2: Change in eGFR between Diabetic and Non-Diabetic Patients

Risk Factors for CIN

To find independent determinants of contrast-induced nephropathy (CIN), multivariable logistic regression analysis was used (Table 4). Contrast volume and baseline eGFR were found to be significant independent predictors of CIN. Increased odds of CIN were linked to higher contrast volume (OR 1.02, 95% CI 1.01–1.02, $p < 0.001$), meaning that the risk increased by 2% for every 1 mL increase. Additionally, a higher incidence of CIN was strongly correlated with lower baseline eGFR (OR 1.05, 95% CI 1.03–1.08, $p < 0.001$). On the other hand, following correction, diabetes mellitus was not an independent predictor (OR 1.41, 95% CI 0.57–3.45, $p = 0.50$).

Table 3. Multivariable Logistic Regression Analysis for CIN

Characteristic	OR	95% CI	p-value
Contrast Volume (mL)	1.02	1.01, 1.02	<0.001
Diabetes Mellitus			

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Non-Diabetic	—	—	
Diabetic	1.41	0.57, 3.45	0.5
Age (years)	1.04	0.99, 1.10	0.13
Hypertension			
No	—	—	
Yes	2.24	0.85, 5.99	0.10
LVEF (%)	1.03	0.97, 1.10	0.4
Baseline eGFR	1.05	1.03, 1.08	<0.001

Discussion

The current study sought to ascertain the prevalence of contrast-induced nephropathy in patients receiving PCI and coronary angiography, as well as to pinpoint related clinical and procedural risk factors (Peer *et al.,* 2017; Azzalini *et al.,* 2019). This comparatively high percentage indicates that renal function decline following iodinated contrast exposure is still a clinically significant problem in standard interventional cardiology treatment. Contrast-related kidney injury still affects a significant percentage of patients, especially those with pre-existing risk factors, despite advancements in procedural technique, better imaging, and the widespread use of preventive measures like hydration (Mehran *et. al.,* 2004).

In the current study, patients who experienced contrast-induced nephropathy (CIN) were more likely to have diabetes mellitus than those who did not. This result is in line with earlier research demonstrating that long-term microvascular and metabolic damage puts diabetes patients at higher risk of acute kidney injury after contrast exposure. The kidney's capacity to withstand further assaults, such as iodinated contrast agents, is diminished by chronic hyperglycemia, which causes increasing glomerulosclerosis, endothelial dysfunction, and reduced renal autoregulation (Forbes *et al.,* 2013; Brownlee *et al.,* 2001). Furthermore, diabetes is often

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linked to chronic renal disease and hypertension, which raises the risk of CIN (Mehran *et al.,* 2006; Rudnick *et al.,* 2020).

Nevertheless, diabetes mellitus was not found to be an independent predictor of CIN in multivariable analysis. This is consistent with findings from other studies that indicate contrast exposure and baseline renal impairment, rather than diabetes acting as an independent risk factor, are the main causes of the observed connection (McCullough *et al.,* 2008; Davenport *et al.,* 2013). The primary mediating factor causing this risk seems to be a lower estimated glomerular filtration rate (eGFR), which is more prevalent in diabetes patients (Nash *et al.,* 2002). Therefore, rather than directly causing CIN, diabetes may be a reflection of underlying renal susceptibility.

The development of CIN was found to be strongly and consistently correlated with contrast volume. The risks of CIN were considerably higher in patients who received higher contrast volumes, indicating a clear dose-response connection. Previous research that identified contrast dosage as one of the most significant modifiable risk factors for CIN provides strong evidence for this (McDonald *et al.,* 2016). Renal vasoconstriction, medullary hypoxia, and direct tubular toxicity are the underlying mechanisms, and they all get worse as the contrast load increases (Persson *et al.,* 2005; Heyman *et al.,* 2008). These results emphasize how crucial it is to reduce contrast volume during diagnostic and therapeutic operations, especially for patients who are at high risk.

Baseline renal function was another key independent predictor of CIN. Patients with lower pre-procedural eGFR had a considerably greater frequency of CIN, consistent with extensive earlier research indicating chronic renal disease as the biggest risk factor for contrast-associated kidney injury (Barrett *et al.,* 2006; Mehran *et al.,* 2004). Reduced nephron reserve and reduced autoregulation in these individuals limit the kidney's ability to adapt for hemodynamic and toxic stress, making even modest insults clinically important (Thomsen, H. S. 2011).

Although diabetes was not an independent predictor in adjusted analysis, diabetic patients nonetheless demonstrated a higher drop in post-procedure renal function. This shows that

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diabetes may exert an indirect or synergistic effect when paired with other risk factors such as low eGFR or high contrast volume. Prior studies have similarly suggested that diabetic kidneys live in a "primed" state of vulnerability, where preclinical structural and functional problems magnify the impact of additional renal insults (Weisbord *et al.,* 2008).

Overall, these findings suggest that CIN is a complex illness in which contrast volume and baseline renal function remain the most relevant factors of risk. Careful patient selection, risk stratification, and minimization of contrast exposure are essential strategies to reduce CIN incidence in clinical practice.

Conclusion

The frequency and predictors of contrast-induced nephropathy (CIN) in patients receiving PCI and coronary angiography were evaluated in this study. 58% of patients experienced CIN, suggesting that contrast-associated renal damage is still a common consequence in standard interventional treatment. Patients with diabetes were more likely to have CIN; they also had lower baseline renal function, a larger comorbidity burden, and higher contrast volumes. These results imply that rather than being a separate causative factor, diabetes is a reflection of a general high-risk clinical profile. After the surgery, diabetic patients also showed a higher early deterioration in renal function. Contrast volume and baseline eGFR had the highest correlation with CIN out of all the factors. While lower baseline eGFR suggested decreased renal reserve and increased vulnerability to damage, higher contrast exposure was associated with an increased likelihood of renal impairment. Only contrast volume and baseline eGFR were found to be independent predictors of CIN in multivariable analysis. After correction, diabetes was not statistically significant, indicating that renal function and contrast exposure play a major role in mediating its impact.

In conclusion, baseline renal function and contrast volume are the main risk factors for CIN, which is still frequent during coronary angiography and PCI. Reducing renal consequences requires careful risk classification and minimal contrast use.

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Recommendations

Prior to coronary angiography or PCI, all patients should have their renal function evaluated. Those with diabetes mellitus or pre-existing renal impairment should receive special attention because they are more likely to experience contrast-induced nephropathy. Contrast volume should be kept to the lowest level required for safe and efficient imaging throughout the process, eliminating needless exposure whenever feasible. Appropriate peri-procedural hydration and post-procedural serum creatinine monitoring should be carried out in at-risk patients to enable early identification and prompt treatment of any decline in renal function.

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