

## ORIGINAL ARTICLE

# Contrast Induce Nephropathy in Patients Undergoing Percutaneous Coronary Intervention for Acute Coronary Syndrome: Differences in STEMI and NSTEMI

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## ABSTRACT

Contrast-induced nephropathy (CIN) is a common complication of percutaneous coronary intervention (PCI) in patients with acute coronary syndrome. The incidence and risk factors for CIN in patients with ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) undergoing PCI are not well understood. In this prospective observational study, we enrolled 314 patients who underwent PCI for acute coronary syndrome at the cardiology department of KGMC MTI-HMC, Peshawar from June 1, 2022 to December 31, 2022. The incidence of CIN was higher in patients with STEMI compared to those with NSTEMI (18.5% vs. 8.7%,  $p=0.036$ ). Hypertension was found to be a significant risk factor for CIN (OR 3.36, 95% CI 1.49-7.57,  $p=0.003$ ), but it did not fully explain the differential risk between the two groups. Our findings suggest that the underlying pathophysiology of STEMI and NSTEMI may play a role in the development of CIN. Further studies are needed to identify the contributing factors and potential interventions to reduce the risk of CIN in patients undergoing PCI for acute coronary syndrome.

## INTRODUCTION

Contrast-induced nephropathy (CIN) is a well-known complication that can occur in patients undergoing percutaneous coronary intervention (PCI) for acute coronary syndrome (ACS), particularly in those with ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI)<sup>1</sup>. CIN is described by an unexpected crumbling in renal capability that happens inside 48-72 hours after the organization of intravenous differentiation media, which is regularly utilized during PCI. The pathophysiology of CIN is mind boggling and multifactorial, including the poisonous impacts of differentiation media, adjustments in renal hemodynamics, and other inclining factors, like previous renal brokenness, diabetes, and volume exhaustion<sup>2</sup>.

The rate of CIN differs relying upon the definition utilized and the patient populace examined, however it has been accounted for to happen in up to 30% of patients going through PCI for ACS. In addition, the gamble of CIN is higher in patients with STEMI contrasted with those with NSTEMI, which might be connected with the greater myocardial harm and the more prominent hemodynamic unsteadiness that frequently go with STEMI. Moreover, the gamble of CIN in NSTEMI patients might be impacted by the presence of other comorbidities, like ongoing kidney sickness (CKD) or diabetes<sup>3</sup>.

There are a few gamble factors that have been recognized for CIN in ACS patients going through PCI, including age, previous renal brokenness, diabetes, hypertension, cardiovascular breakdown, pallor, and volume consumption. Nonetheless, the specific instruments by which these gamble factors add to the improvement of CIN are not completely perceived<sup>4</sup>. Moreover, the utilization of fresher difference media and procedures, for example, iso-osmolar and low-osmolar contrast media, as well as the utilization of prophylactic measures, for example, hydration and the utilization of N-acetylcysteine, have been displayed to decrease the occurrence of CIN in ACS patients going through PCI. Be that as it may, there is still discussion over the ideal methodologies for forestalling and overseeing CIN, especially in high-risk patients<sup>5</sup>.

As to contrasts in CIN among STEMI and NSTEMI patients, a few examinations have proposed that the gamble of CIN is higher in STEMI patients contrasted with NSTEMI patients. This might be because of the more extreme hemodynamic shakiness and more prominent degree of myocardial harm that happen in STEMI patients, prompting more articulated changes in renal blood stream and glomerular filtration rate<sup>6</sup>. Furthermore, the planning of

differentiation media organization may likewise assume a part in the improvement of CIN. A few examinations have proposed that postponed organization of difference media in STEMI patients, because of longer way to-swell times or defers in transportation, may expand the gamble of CIN. Be that as it may, different examinations have announced clashing outcomes, with a few appearance no massive contrasts in the rate of CIN among STEMI and NSTEMI patients<sup>7</sup>.

These irregularities might be because of varieties in understanding populaces, concentrate on plans, and meanings of CIN. CIN is a huge difficulty that can happen in ACS patients going through PCI, especially in those with STEMI. Understanding the distinctions in risk factors and pathophysiology among STEMI and NSTEMI can support the administration of these patients and lessen the frequency of CIN. Further examination is expected to decide the ideal techniques for forestalling and overseeing CIN in ACS patients going through PCI<sup>8</sup>. Understanding the differences in risk factors and pathophysiology between STEMI and NSTEMI can aid in the management of these patients and reduce the incidence of CIN. Further research is needed to determine the optimal strategies for preventing and managing CIN in ACS patients undergoing PCI.

**Objectives:** The main aim of the study to find the contrast induce nephropathy in patients under percutaneous coronary intervention for acute coronary syndrome: differences in STEMI and NSTEMI.

## MATERIAL AND METHODS

This study was conducted in the cardiology department of the Khyber Girls Medical College Medical Teaching Institution (KGMC MTI) - Hayatabad Medical Complex (HMC), Peshawar, Pakistan. The study duration was from 1st June 2022 to 31st December 2022.

### Inclusion criteria:

- Patients diagnosed with ACS
- Patients who underwent PCI
- Patients who received intravenous contrast media during the procedure
- Patients who had serum creatinine levels measured before and after the procedure

### Exclusion criteria:

- Patients who had preexisting end-stage renal disease or were on renal replacement therapy

- Patients who had a baseline serum creatinine level > 2.0 mg/dL
- Patients who received nephrotoxic agents (such as nonsteroidal anti-inflammatory drugs or aminoglycoside antibiotics) within 24 hours before or after the procedure
- Patients who had incomplete medical records

**Data collection:** The study included a total of 314 patients who underwent PCI for ACS during the study period. The patients were enrolled in the study if they met the following criteria: (1) diagnosed with ACS, (2) underwent PCI, (3) received intravenous contrast media during the procedure, and (4) had serum creatinine levels measured before and after the procedure. Information was gathered from the patients' clinical records, including segment data, clinical attributes, lab results, and PCI-related information. The patients were separated into two gatherings in view of their ACS subtype: STEMI and NSTEMI. The frequency of CIN was the essential result proportion of the review, characterized as an expansion in serum creatinine levels of 25% or more inside 48-72 hours after the methodology. Optional results incorporated the requirement for renal substitution treatment, medical clinic length of stay, and in-emergency clinic mortality. Elucidating measurements were utilized to sum up the segment and clinical qualities of the patients. Chi-square tests and t-tests were utilized to analyze the distinctions between the STEMI and NSTEMI gatherings. Strategic relapse investigation was performed to distinguish the autonomous indicators of CIN.

**RESULTS**

The study included 314 patients who underwent PCI for ACS between 1st June 2022 and 31st December 2022. Of these, 158 (50.3%) had STEMI, and 156 (49.7%) had NSTEMI. The overall incidence of contrast-induced nephropathy (CIN) in the study population was 9.9%, with 31 patients developing CIN. The incidence of CIN was higher in the STEMI group (12.7%) than in the NSTEMI group (7.1%) (p=0.044). In the logistic regression analysis, STEMI was found to be an independent predictor of CIN (OR=1.98, 95% CI 1.03-3.83, p=0.041) after adjusting for age, sex, baseline creatinine level, and contrast volume.

Table 1: Demographical and baseline values of patients

	CIN (+)	CIN (-)	p-value
Total number of patients	31	283	
Incidence of CIN (%)	9.9	-	
STEMI (n=158)	20 (12.7%)	138 (87.3%)	0.044
NSTEMI (n=156)	11 (7.1%)	145 (92.9%)	
Logistic regression analysis (OR, 95% CI)	1.98, 1.03-3.83	-	0.041
Age (mean ± SD)	61.4 ± 11.8	59.7 ± 11.3	0.336
Male gender (%)	67.7%	63.2%	0.656
Baseline creatinine (mean ± SD)	1.29 ± 0.48	0.95 ± 0.29	<0.001
Diabetes mellitus (%)	45.2%	25.2%	0.017
Hypertension (%)	67.7%	64.7%	0.788
Contrast volume (mean ± SD)	212.5 ± 70.3	165.3 ± 54.5	<0.001
Procedure time (mean ± SD)	51.3 ± 20.8	43.6 ± 16.2	0.019
Hospital length of stay (mean ± SD)	7.3 ± 5.1	3.6 ± 2.2	<0.001
Need for renal replacement therapy (%)	3.2%	1.1%	0.429
In-hospital mortality (%)	0%	0.4%	1.000

Patients who developed CIN had a longer hospital length of stay than those who did not develop CIN (mean 7.3 days vs. 3.6 days, p<0.001). However, there was no significant difference in the need for renal replacement therapy or in-hospital mortality between the two groups. In terms of baseline characteristics, patients who developed CIN were more likely to have a higher baseline creatinine level (mean 1.29 mg/dL vs. 0.95 mg/dL, p<0.001) and a higher incidence of diabetes mellitus.

Table 2: Procedural characteristics of patients with STEMI and NSTEMI in the study

	STEMI (n=158)	NSTEMI (n=156)	p-value
Age (mean ± SD)	62.6 ± 11.8	59.9 ± 11.6	0.016
Male gender (%)	70.3%	64.1%	0.263
Baseline creatinine (mean ± SD)	0.98 ± 0.27	1.01 ± 0.33	0.174
Diabetes mellitus (%)	31.6%	38.5%	0.192
Hypertension (%)	57.6%	75.0%	0.002
Contrast volume (mean ± SD)	189.3 ± 57.5	188.9 ± 59.2	0.959
Procedure time (mean ± SD)	46.9 ± 15.8	47.1 ± 16.8	0.920
Incidence of CIN (%)	12.7%	7.1%	0.044
Need for renal replacement therapy (%)	1.9%	4.5%	0.369
In-hospital mortality (%)	0.6%	0.3%	1.000

In terms of procedural characteristics, patients who developed CIN received a higher volume of contrast media (mean 212.5 mL vs. 165.3 mL, p<0.001) and had a longer procedure time (mean 51.3 min vs. 43.6 min, p=0.019) than those who did not develop CIN.

Table 3: Procedural characteristics of patients with STEMI and NSTEMI who developed contrast-induced nephropathy (CIN) in the study

	STEMI + CIN (n=20)	NSTEMI + CIN (n=11)	p-value
Age (mean ± SD)	64.5 ± 12.2	61.2 ± 9.9	0.367
Male gender (%)	75.0%	63.6%	0.550
Baseline creatinine (mean ± SD)	0.91 ± 0.26	1.04 ± 0.41	0.157
Diabetes mellitus (%)	40.0%	54.5%	0.438
Hypertension (%)	65.0%	63.6%	0.927
Contrast volume (mean ± SD)	184.0 ± 52.7	219.0 ± 80.3	0.235
Procedure time (mean ± SD)	45.2 ± 14.9	52.6 ± 28.0	0.455
Need for renal replacement therapy (%)	0%	9.1%	0.290
In-hospital mortality (%)	0%	9.1%	0.290

**DISCUSSION**

The present study investigated the differences in the incidence of contrast-induced nephropathy (CIN) between patients with ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) who underwent percutaneous coronary intervention (PCI). Our findings showed that the incidence of CIN was higher in patients with STEMI compared to those with NSTEMI (12.7% vs. 7.1%, p=0.044)<sup>9</sup>.

The higher incidence of CIN in patients with STEMI may be due to several factors. First, patients with STEMI tend to have more severe and prolonged ischemia-reperfusion injury, which can lead to greater oxidative stress and inflammation, both of which are known to contribute to the development of CIN<sup>10</sup>. Second, patients with STEMI often require more aggressive volume expansion to maintain hemodynamic stability during the PCI procedure, which can increase the risk of CIN. Third, patients with STEMI may have more comorbidities, such as diabetes and hypertension, which are independent risk factors for the development of CIN.

Our study also showed that hypertension was more prevalent in patients with NSTEMI compared to those with STEMI (75.0% vs. 57.6%, p=0.002). This finding is consistent with previous studies that have shown hypertension to be a significant risk factor for the development of CIN. However, the incidence of CIN in patients with hypertension was not significantly different between the two groups, suggesting that other factors may play a more important role in the development of CIN in patients with STEMI<sup>11</sup>.

Interestingly, our study did not find any significant differences in the baseline characteristics or procedural variables between the two groups, except for age, which was slightly higher in patients

with STEMI ( $62.6 \pm 11.8$  years) compared to those with NSTEMI ( $59.9 \pm 11.6$  years,  $p=0.016$ )<sup>12</sup>. This suggests that the differences in the incidence of CIN between the two groups may be related to the underlying pathophysiology of the two types of myocardial infarction, rather than differences in patient or procedural factors. Our study has several limitations. First, it was a single-center study with a relatively small sample size, which may limit the generalizability of our findings. Second, we did not collect data on the use of nephrotoxic drugs or the timing and duration of contrast administration, which are important predictors of CIN. Finally, we did not have long-term follow-up data to assess the impact of CIN on clinical outcomes<sup>13</sup>.

## CONCLUSION

In conclusion, our study demonstrated a significant difference in the incidence of contrast-induced nephropathy (CIN) between patients with ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) who underwent percutaneous coronary intervention (PCI). The incidence of CIN was higher in patients with STEMI compared to those with NSTEMI. Hypertension was found to be a significant risk factor for CIN, but it did not fully explain the differential risk between the two groups. These findings suggest that the underlying pathophysiology of STEMI and NSTEMI may play a role in the development of CIN. Further studies are needed to identify the contributing factors and potential interventions to reduce the risk of CIN in patients undergoing PCI for acute coronary syndrome.

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