

## Contrast-induced Nephropathy among Patients with Chronic Kidney Disease Undergoing Coronary Angiography and Percutaneous Coronary Intervention

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### ABSTRACT

**Objective:** To determine the frequency of contrast-induced nephropathy among patients with chronic kidney disease (CKD) undergoing coronary angiography or percutaneous coronary intervention (PCI).

**Study Design, Place and Duration:** Descriptive case series conducted in the cardiac catheterization service of Ittefaq Trust Hospital, Lahore, from 4 May 2026 to 3 August 2026.

**Materials and Methods:** Ninety adults aged 30–80 years with established CKD undergoing coronary angiography or PCI were included. Contrast-induced nephropathy was defined according to the study protocol as an increase in serum creatinine of at least 25% from baseline or an absolute increase of at least 0.5 mg/dL within 48–72 hours after contrast administration. Baseline estimated glomerular filtration rate (eGFR), comorbidities, procedure type, contrast volume and serial serum creatinine were assessed. Between-group comparisons were performed using distribution-appropriate tests. A two-variable logistic regression model examined baseline eGFR and contrast volume.

**Results:** Contrast-induced nephropathy occurred in 18 of 90 patients (20.0%; 95% CI 13.0%–29.4%). Its frequency was 5.1% in CKD stage 3a, 16.1% in stage 3b and 55.0% in stage 4 ( $p < 0.001$ ). Median baseline eGFR was lower in patients who developed nephropathy than in those who did not (27.85 vs 45.30 mL/min/1.73 m<sup>2</sup>;  $p < 0.001$ ). Contrast volume did not differ significantly between groups ( $p = 0.848$ ). In exploratory adjusted analysis, the odds ratio per 10 mL/min/1.73 m<sup>2</sup> higher eGFR was 0.37 (95% CI 0.21–0.65); the contrast-volume estimate was imprecise.

**Conclusion:** One in five patients met the study definition of contrast-induced nephropathy after coronary angiography or PCI. Events were more frequent with lower baseline kidney function, particularly in CKD stage 4. The findings support assessment of renal function before contrast exposure and careful interpretation of post-procedural creatinine changes

**Keywords:** chronic kidney disease; coronary angiography; percutaneous coronary intervention; contrast-associated acute kidney injury; serum creatinine; contrast volume.

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### INTRODUCTION

Contrast-induced nephropathy (CIN) describes an acute increase in serum creatinine after administration of iodinated contrast media. It remains a recognized complication of coronary angiography (CAG) and percutaneous coronary intervention (PCI), particularly in patients with pre-existing chronic kidney disease (CKD). Acute deterioration in renal function after these procedures has been associated with longer hospital admission and adverse cardiovascular and renal outcomes. However, a rise in creatinine after contrast exposure does not, by itself, establish contrast as the cause; haemodynamic changes, concurrent illness and other nephrotoxic exposures may also contribute. Current guidance therefore distinguishes contrast-associated acute kidney injury from injury causally attributed to contrast media [1,2,3].

Baseline kidney function is an important determinant of post-procedural renal risk. The Mehran risk score incorporates renal impairment together with clinical and procedural characteristics to estimate the probability of CIN after PCI [4]. Its original validation reported a C-statistic of 0.67, indicating that risk estimates are informative but do not precisely classify every patient. A systematic review by Silver et al. also found variation in the performance and applicability of published CIN prediction models [5]. Renal function should therefore be considered alongside other clinical characteristics rather than interpreted as a stand-alone predictor.

Reported frequencies of renal injury after angiography differ according to the study population, contrast administration and endpoint definition. In a meta-analysis of angiography studies, Wu et al. reported a pooled CIN frequency of 9.06% (95%

CI 8.53%–9.58%) [6]. Estimates from samples restricted to renal impairment or other high-risk conditions may be higher. In a Karachi study of 115 patients with baseline serum creatinine above 1.4 mg/dL, Uddin et al. observed CIN in 11 (9.65%), with a greater frequency among patients with more severe baseline renal dysfunction [7].

Other Pakistani studies have examined different clinical populations. Ramzan et al. reported CIN in 45 of 255 patients with type 2 diabetes undergoing cardiac catheterization (17.6%) [8]. Kumar et al. found CIN in 37 of 282 patients (13.1%) undergoing primary PCI for ST-segment elevation myocardial infarction and reported associations with diabetes and contrast volume above 200 mL [9]. The latter study excluded patients with pre-existing CKD, so its estimate cannot be applied directly to a population selected for chronic renal impairment. Differences in eligibility criteria and creatinine thresholds also limit direct comparisons between these reports.

Patients with CKD frequently require coronary investigations or revascularization, yet their susceptibility to post-procedural renal deterioration complicates clinical decision-making. Local estimates specifically describing CIN across CKD stages after CAG and PCI are relevant to counselling and periprocedural risk assessment. This study determined the frequency of CIN among patients with CKD undergoing CAG or PCI at Ittefaq Trust Hospital, Lahore, and examined its distribution by baseline renal function and selected clinical and procedural characteristics.

## 1. MATERIALS AND METHODS

A descriptive case series was conducted at the cardiac catheterization service of Ittefaq Trust Hospital, Lahore, from 4 May 2026 to 3 August 2026. The sample comprised 90 patients. The sample-size calculation used an anticipated CIN frequency of 17.6%, a 95% confidence level and approximately 8% absolute precision. Eligible patients undergoing diagnostic coronary angiography or PCI were enrolled consecutively.

Patients aged 30–80 years of either sex were included if CKD had been present for at least 12 weeks and coronary angiography or PCI was planned. CKD was defined according to the study protocol as persistent evidence of kidney damage or eGFR below 60 mL/min/1.73 m<sup>2</sup>. Participants included in the analysis had CKD stages 3a, 3b or 4, classified according to standard eGFR categories [10]. Exclusion criteria comprised acute kidney injury before catheterization, a history of contrast allergy and other conditions that could account for an acute deterioration in kidney function.

Age, sex, duration and stage of CKD, diabetes mellitus, hypertension, heart failure, previous coronary revascularization, haemoglobin, baseline serum creatinine and eGFR were assessed. Procedural variables included the indication for catheterization, diagnostic angiography or PCI, contrast type and volume, periprocedural hydration and nephroprotective medication use. Serum creatinine was measured at baseline and at 48 and 72 hours after contrast administration. The primary endpoint was an increase in serum creatinine of at least 25% from baseline or at least 0.5 mg/dL within 48–72 hours. This prespecified definition is retained for the present study; it differs from some contemporary acute kidney injury criteria and should be considered when comparing event frequencies [2].

Continuous variables were presented as mean ± standard deviation or median (interquartile range), according to distribution, and categorical variables as frequency and percentage. The Shapiro–Wilk test was used to assess normality. Group comparisons were performed using Welch independent-samples t-test or Mann–Whitney U test for continuous variables and Pearson chi-square or Fisher exact test for categorical variables, as appropriate. Unadjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported for categorical characteristics. An exploratory logistic regression model included baseline eGFR per 10 mL/min/1.73 m<sup>2</sup> and contrast volume per 50 mL; the number of variables was restricted because 18 patients met the primary endpoint. All tests were two-sided, and  $p < 0.05$  was considered statistically significant.

## 2. RESULTS

Ninety patients were included, comprising 56 men (62.2%) and 34 women (37.8%). Mean age was  $60.68 \pm 10.35$  years. CKD stage 3a was present in 39 patients (43.3%), stage 3b in 31 (34.4%) and stage 4 in 20 (22.2%). Diabetes mellitus was recorded in 48 patients (53.3%), hypertension in 70 (77.8%) and heart failure in 18 (20.0%). Forty-three patients (47.8%) underwent diagnostic coronary angiography and 47 (52.2%) underwent PCI. Median contrast volume was 125.0 mL (IQR 100.25–156.00). Baseline and procedural characteristics are presented in Table 1.

**Table 1. Demographic, renal and procedural characteristics of the study population (n=90)**

| Characteristic                            | Result                               |
|-------------------------------------------|--------------------------------------|
| Age, years                                | 60.68 ± 10.35                        |
| Male sex                                  | 56 (62.2%)                           |
| Female sex                                | 34 (37.8%)                           |
| CKD stage 3a / 3b / 4                     | 39 (43.3%) / 31 (34.4%) / 20 (22.2%) |
| Baseline eGFR, mL/min/1.73 m <sup>2</sup> | 40.83 ± 11.17                        |
| Baseline serum creatinine, mg/dL          | 1.61 ± 0.37                          |
| Diabetes mellitus                         | 48 (53.3%)                           |
| Hypertension                              | 70 (77.8%)                           |
| Heart failure                             | 18 (20.0%)                           |
| Haemoglobin, g/dL                         | 11.49 ± 0.90                         |
| Diagnostic angiography                    | 43 (47.8%)                           |

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|                                   |                       |
|-----------------------------------|-----------------------|
| PCI                               | 47 (52.2%)            |
| Contrast volume, mL               | 125.0 (100.25–156.00) |
| Periprocedural hydration recorded | 77 (85.6%)            |

Note. Continuous values are mean  $\pm$  SD or median (IQR), as appropriate. CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; PCI = percutaneous coronary intervention.

Eighteen of 90 patients (20.0%; Wilson 95% CI 13.0%–29.4%) met the protocol-defined CIN endpoint within 48–72 hours. Median baseline eGFR was 27.85 mL/min/1.73 m<sup>2</sup> among patients who developed CIN and 45.30 mL/min/1.73 m<sup>2</sup> among those who did not (Mann–Whitney U=304.0;  $p<0.001$ ). Baseline serum creatinine was higher in the CIN group (1.92 vs 1.46 mg/dL;  $p<0.001$ ). The median contrast volumes were 121.5 and 125.0 mL, respectively (U=628.5;  $p=0.848$ ). Serial creatinine measurements and between-group comparisons are shown in Table 2.

**Table 2. Renal measurements and clinical characteristics by the protocol-defined outcome**

| Variable                                  | No renal event (n=72)  | Renal event (n=18)    | Statistic | p      |
|-------------------------------------------|------------------------|-----------------------|-----------|--------|
| Baseline eGFR, mL/min/1.73 m <sup>2</sup> | 45.30 (36.85–50.52)    | 27.85 (25.30–37.40)   | U=304.0   | <0.001 |
| Baseline creatinine, mg/dL                | 1.46 (1.31–1.67)       | 1.92 (1.70–2.12)      | U=1018.0  | <0.001 |
| Serum creatinine, 48 h, mg/dL             | 1.50 (1.36–1.70)       | 2.34 (2.09–2.68)      | U=1162.5  | <0.001 |
| Serum creatinine, 72 h, mg/dL             | 1.54 (1.37–1.76)       | 2.57 (2.27–2.83)      | U=1222.0  | <0.001 |
| Contrast volume, mL                       | 125.00 (100.75–156.00) | 121.50 (90.75–173.50) | U=628.5   | 0.848  |
| Age, years                                | 60.50 $\pm$ 10.60      | 61.39 $\pm$ 9.52      | t=0.35    | 0.732  |
| Haemoglobin, g/dL                         | 11.50 $\pm$ 0.90       | 11.44 $\pm$ 0.94      | t=-0.23   | 0.823  |

Note. Values are mean  $\pm$  SD or median (IQR). The higher post-procedure creatinine values are part of the event definition and should not be interpreted as independent predictors. p values are based on Welch t or Mann–Whitney U tests.

The frequency of CIN increased from 2 of 39 patients (5.1%) with CKD stage 3a to 5 of 31 (16.1%) with stage 3b and 11 of 20 (55.0%) with stage 4 (Pearson  $\chi^2(2)=20.99$ ;  $p<0.001$ ). Relative to stage 3a, the unadjusted OR was 3.56 (95% CI 0.64–19.77) for stage 3b and 22.61 (95% CI 4.24–120.54) for stage 4. No statistically significant difference in CIN frequency was detected by sex, diabetes status, hypertension status or procedure type. Some estimates had wide CIs, particularly for hypertension (Table 3).

**Table 3. Frequency of contrast-induced nephropathy and unadjusted associations**

| Characteristic  | n  | Renal event n (%) | OR (95% CI)         | Test              | p      |
|-----------------|----|-------------------|---------------------|-------------------|--------|
| CKD 3a          | 39 | 2 (5.1%)          | Reference           | $\chi^2(2)=20.99$ | <0.001 |
| CKD 3b          | 31 | 5 (16.1%)         | 3.56 (0.64–19.77)   | —                 | —      |
| CKD 4           | 20 | 11 (55.0%)        | 22.61 (4.24–120.54) | —                 | —      |
| Female          | 34 | 6 (17.6%)         | Reference           | $\chi^2(1)=0.19$  | 0.664  |
| Male            | 56 | 12 (21.4%)        | 1.27 (0.43–3.78)    | —                 | —      |
| No diabetes     | 42 | 6 (14.3%)         | Reference           | $\chi^2(1)=1.61$  | 0.205  |
| Diabetes        | 48 | 12 (25.0%)        | 2.00 (0.68–5.91)    | —                 | —      |
| No hypertension | 20 | 1 (5.0%)          | Reference           | Fisher exact      | 0.064  |
| Hypertension    | 70 | 17 (24.3%)        | 6.09 (0.76–48.97)   | —                 | —      |
| Angiography     | 43 | 9 (20.9%)         | Reference           | $\chi^2(1)=0.04$  | 0.833  |
| PCI             | 47 | 9 (19.1%)         | 0.89 (0.32–2.51)    | —                 | —      |

Note. Percentages use the row total. The stage-specific odds ratios use CKD stage 3a as reference. Tests for multilevel variables are overall tests. Sparse categories use Fisher exact testing as specified.

In the exploratory model, higher baseline eGFR was associated with lower odds of CIN (adjusted OR 0.37 per 10 mL/min/1.73 m<sup>2</sup>; 95% CI 0.21–0.65;  $p=0.001$ ). Contrast volume was not significantly associated with the outcome (adjusted OR 1.07 per 50 mL; 95% CI 0.56–2.05;  $p=0.827$ ). These estimates should be interpreted with regard to the 18 observed events and the limited number of covariates included in the model.

### 3. DISCUSSION

Contrast-induced nephropathy occurred in 18 of 90 patients (20.0%) with pre-existing CKD undergoing CAG or PCI. The frequency differed considerably across renal-function categories: 5.1% in stage 3a, 16.1% in stage 3b and 55.0% in stage 4. Lower baseline eGFR was also associated with the outcome in exploratory adjusted analysis. These findings describe a marked concentration of short-term creatinine-defined renal events among patients with more advanced CKD.

The observed frequency was higher than the pooled estimate of 9.06% reported by Wu et al. in angiography studies [6]. That meta-analysis included varied clinical settings, eligibility criteria and definitions of nephropathy. Differences are also evident within Pakistani reports. Uddin et al. observed an event frequency of 9.65% in patients with baseline creatinine above 1.4 mg/dL, with greater risk at higher creatinine levels [7]. Ramzan et al. reported 17.6% among patients with type

2 diabetes [8], whereas Kumar et al. reported 13.1% after primary PCI in patients with ST-segment elevation myocardial infarction [9]. Kumar et al. excluded patients with CKD, making their study population particularly different from the present sample. Variation in renal impairment, acute coronary presentation and the definition used for CIN may account for part of the difference in reported frequencies.

The distribution of events by CKD stage was the clearest finding in the present analysis. Eleven of 20 patients with stage 4 disease developed CIN, compared with two of 39 with stage 3a disease. Each 10 mL/min/1.73 m<sup>2</sup> higher baseline eGFR was associated with lower adjusted odds of CIN (OR 0.37, 95% CI 0.21–0.65). An earlier Karachi study likewise reported a higher frequency of nephropathy in patients with more severe baseline renal impairment [7]. Renal dysfunction is included in both the original and contemporary Mehran risk models, along with other clinical and procedural predictors [4,11]. The present findings support reporting renal-stage-specific frequencies rather than a single aggregate percentage for all patients with CKD.

Contrast volume did not differ significantly between patients with and without CIN (median 121.5 vs 125.0 mL;  $p=0.848$ ). The adjusted association per 50 mL was imprecise (OR 1.07, 95% CI 0.56–2.05), so these findings should not be taken as evidence that contrast dose is unrelated to renal injury. Wu et al. reported an association between contrast exposure volume and CIN in meta-regression [6], and Kumar et al. identified an association with volumes above 200 mL after primary PCI [9]. The relatively similar contrast volumes in the two outcome groups here, together with the small number of events, limit assessment of a dose–response relationship. Haemodynamic status, procedure complexity and baseline kidney function may also influence renal outcomes after catheterization [2,3].

Diabetes mellitus was present in 48 patients (53.3%) and hypertension in 70 (77.8%). CIN was numerically more frequent among patients with these conditions, although neither comparison reached the prespecified statistical threshold. The imprecise OR for hypertension (6.09, 95% CI 0.76–48.97) is compatible with a wide range of underlying associations. A systematic review by Ye et al. identified risk factors for contrast-associated AKI in patients undergoing PCI for ST-segment elevation myocardial infarction [12], but those findings cannot be transferred directly to patients with established CKD undergoing a mixture of diagnostic and interventional procedures. The present analysis also had insufficient events for a model including all potentially relevant comorbidities.

Periprocedural hydration was documented in 77 patients (85.6%), without a statistically significant association with CIN in the present analysis. This observational comparison cannot establish whether hydration prevented renal injury, particularly because the volume, timing and method of fluid administration were not fully characterized. In the POSEIDON randomized trial, Brar et al. reported fewer renal events with left ventricular end-diastolic pressure-guided hydration than with standard hydration (6.7% vs 16.3%) [13]. A subsequent meta-analysis of 13 randomized trials reported lower CI-AKI with individualized rather than non-individualized fluid regimens (RR 0.56, 95% CI 0.46–0.69) [14]. Preventive strategies therefore need to be interpreted in relation to patient selection, volume status and the protocol actually used.

Several limitations affect interpretation. The study was conducted at one centre and included 90 patients, of whom 18 met the renal endpoint; estimates for individual CKD stages and adjusted associations therefore have limited precision. The regression model included only baseline eGFR and contrast volume and did not fully address other potential confounders, including procedural haemodynamic instability. Hydration was recorded as a clinical characteristic rather than assessed through a standardized comparative protocol. Finally, the study used the prespecified  $\geq 25\%$  or  $\geq 0.5$  mg/dL creatinine increase at 48–72 hours, which is not interchangeable with all current AKI definitions [2,15]. Without an unexposed comparison group, a post-procedural creatinine increase cannot be attributed to contrast media alone. These constraints should be considered when applying the findings to clinical risk assessment.

#### 4. CONCLUSION

Contrast-induced nephropathy, defined by the study-specific creatinine criterion, was observed in 18 of 90 patients (20.0%) with CKD undergoing coronary angiography or PCI. Its frequency increased across CKD stages and was highest among patients with stage 4 disease. Lower baseline eGFR was associated with the outcome, whereas the association with contrast volume was not statistically significant and remained imprecise. Assessment of baseline renal function and follow-up serum creatinine may assist identification of patients requiring closer post-procedural renal monitoring.

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