Contrast-Induced Acute Kidney Injury in Patients Undergoing Emergency Coronary Angiography: Eighteen Months of Prospective Surveillance, Risk Score Validation, and Thirty-Day Outcomes

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***Abstract*—Contrast-induced acute kidney injury (CI-AKI) following emergency coronary angiography is associated with prolonged hospitalisation, increased need for renal replacement therapy, and higher 30-day mortality. We prospectively monitored renal function in 302 consecutive patients undergoing emergency angiography for ST-elevation or high-risk non-ST-elevation myocardial infarction across 18 months at a tertiary cardiac centre. CI-AKI, defined by KDIGO criteria, occurred in 44 patients (14.6%), with stage 1 in 28 (9.3%), stage 2 in 12 (4.0%), and stage 3 in 4 (1.3%). The Mehran risk score showed good discrimination in this cohort (AUC 0.81, 95% CI 0.74-0.88), with CI-AKI incidence rising from 3.2% in the lowest risk stratum to 41.7% in the highest. Baseline CKD with eGFR below 60 mL/min/1.73 m² doubled CI-AKI incidence at every Mehran stratum. Patients who developed CI-AKI experienced longer length of stay (median 10 vs 5 days), higher in-hospital mortality (9.1% vs 1.9%), and substantially higher rates of new dialysis requirement (9.1% vs 0%). Pre-procedural risk stratification combined with peri-procedural hydration and minimised contrast volume offers a practical pathway to reduce this burden.**

***Index Terms*—contrast-induced acute kidney injury, CI-AKI, emergency PCI, Mehran risk score, chronic kidney disease, renal replacement therapy**

I. Introduction

Iodinated contrast agents are essential to the conduct of modern coronary angiography and percutaneous intervention, but a small minority of exposed patients develop acute kidney injury in the days following the procedure. The mechanism combines direct tubular toxicity, oxidative stress, and renal medullary hypoxia, with effects magnified in patients with pre-existing kidney dysfunction, diabetes, advanced age, and haemodynamic compromise. Although most cases resolve over days to weeks, a meaningful minority progress to dialysis-requiring renal failure, and the cumulative burden on length of stay and mortality is substantial (Jha, Kumar,, & Neha, 2026; Kumar, Gautam,, & Maitiy, 2026; Bhatnagar, Kumar,, & Shivam, 2026). Elective coronary angiography offers multiple opportunities for risk mitigation: pre-procedural assessment, optimisation of hydration, careful selection of contrast volume, and consideration of alternative imaging. The emergency setting offers far fewer of these opportunities. A patient presenting with ST-elevation myocardial infarction must reach the catheter laboratory within

minutes; the conversation about hydration, contrast volume, and renal-protective strategies has to be compressed into the same window, and is often based on incomplete baseline data. We prospectively monitored renal function in consecutive patients undergoing emergency coronary angiography at a tertiary cardiac centre for 18 months. The aims were to characterise the incidence and severity of CI-AKI in this specific setting, to validate the widely used Mehran risk score against local outcomes, to identify independent predictors that might inform a simplified bedside tool, and to describe the 30-day outcomes that follow.

II. Methods

All adult patients undergoing emergency coronary angiography between July 2022 and December 2023 at a tertiary cardiac centre were screened for inclusion. Emergency procedures were defined as those performed within 24 hours of clinical presentation for acute coronary syndrome. Patients on chronic dialysis or with a recent kidney transplant were excluded. A baseline creatinine measurement obtained within 30 days of the procedure was required for inclusion; where no recent value was available, an admission value was used and confirmed against trajectory in the following days. The final cohort comprised 302 patients. CI-AKI was defined using KDIGO criteria: an absolute increase in serum creatinine of 0.3 mg/dL or more within 48 hours, or a relative increase of 1.5-fold or more within 7 days of contrast exposure. Creatinine was measured at baseline, daily for the first 72 hours, on day 5, on day 7, and on day 14. Severity was staged as stage 1 (creatinine increase 1.5-1.9× baseline or ≥0.3 mg/dL absolute), stage 2 (2.0-2.9× baseline), or stage 3 (≥3.0× baseline, ≥4.0 mg/dL absolute, or initiation of renal replacement therapy). The Mehran risk score was calculated for each patient using documented values for hypotension, intra-aortic balloon pump use, congestive heart failure, age over 75, anaemia, diabetes, contrast volume per 100 mL, and serum creatinine or eGFR. Scores were stratified as low (≤5), moderate (6-10), high (11-15), or very high (≥16). A simplified bedside score incorporating only age, eGFR, and contrast volume was constructed for comparison and validated using bootstrap resampling. The primary outcome was CI-AKI incidence overall and by Mehran risk stratum. Secondary outcomes included CI-AKI severity distribution, renal recovery at 14 days and 30 days, new requirement for renal replacement therapy, length of hospital stay, 30-day MACE, and 30-day mortality. Discrimination of risk scores was assessed by area under the ROC curve; calibration was assessed by comparing observed and predicted event rates across deciles. Multivariable Cox regression identified independent predictors of CI-AKI.

III. Results

3.1 Cohort Characteristics

Table 1. Baseline characteristics of the cohort (n = 302).

Characteristic	All patients (n=302)	No CI-AKI (n=258)	CI-AKI (n=44)
Age, mean (SD), years	62.4 (12.6)	60.8 (11.8)	72.1 (10.4)
Female sex, n (%)	98 (32.5)	78 (30.2)	20 (45.5)
Hypertension, n (%)	198 (65.6)	162 (62.8)	36 (81.8)
Diabetes mellitus, n (%)	118 (39.1)	92 (35.7)	26 (59.1)
Baseline eGFR, mean (SD), mL/min/1.73m²	68.4 (22.4)	72.6 (20.8)	43.8 (16.2)
Baseline eGFR <60, n (%)	98 (32.5)	68 (26.4)	30 (68.2)
Baseline eGFR <45, n (%)	42 (13.9)	20 (7.8)	22 (50.0)
Anaemia (Hb <11 g/dL women, <12 g/dL men), n (%)	98 (32.5)	74 (28.7)	24 (54.5)
Heart failure on admission, n (%)	58 (19.2)	38 (14.7)	20 (45.5)
Cardiogenic shock at presentation, n (%)	22 (7.3)	12 (4.7)	10 (22.7)
IABP support during procedure, n (%)	18 (6.0)	8 (3.1)	10 (22.7)
STEMI as index event, n (%)	218 (72.2)	186 (72.1)	32 (72.7)
Contrast volume, mean (SD), mL	185 (62)	174 (54)	248 (78)
Contrast volume >300 mL, n (%)	42 (13.9)	22 (8.5)	20 (45.5)
Periprocedural hydration administered, n (%)	194 (64.2)	182 (70.5)	12 (27.3)
Mehran score: low (≤5), n (%)	148 (49.0)	138 (53.5)	10 (22.7)
Mehran score: moderate (6-10), n (%)	82 (27.2)	72 (27.9)	10 (22.7)
Mehran score: high (11-15), n (%)	48 (15.9)	36 (14.0)	12 (27.3)
Mehran score: very high (≥16), n (%)	24 (7.9)	12 (4.7)	12 (27.3)

3.2 CI-AKI Incidence and Severity

CI-AKI occurred in 44 of 302 patients (14.6%). The severity distribution was predominantly stage 1 (28 patients, 9.3%), with stage 2 in 12 patients (4.0%) and stage 3 in 4 patients (1.3%). Three of the four stage 3 cases required dialysis during the index admission. Incidence rose steeply across Mehran risk strata (Figure 1), from 3.2% in low-risk patients without baseline CKD to 64.3% in very-high-risk patients with baseline CKD. The interaction between baseline CKD and Mehran score was substantial: at every Mehran stratum, CI-AKI incidence approximately doubled when baseline eGFR was below 60 mL/min/1.73 m².

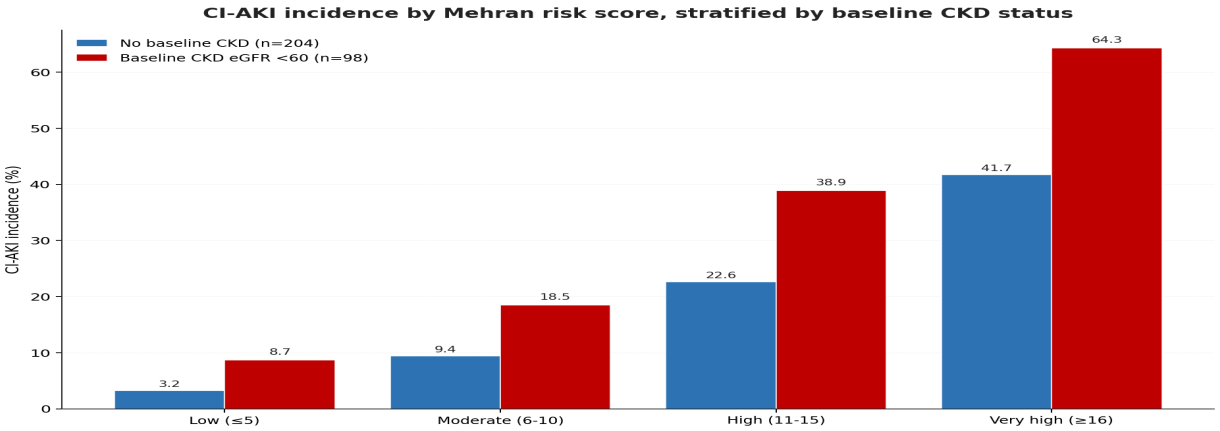

Figure 1. CI-AKI incidence by Mehran risk score stratum, separately for patients with and without baseline chronic kidney disease.

Table 2. CI-AKI incidence and severity distribution.

Outcome	All patients (n=302)	No baseline CKD (n=204)	Baseline CKD eGFR <60 (n=98)
Any CI-AKI, n (%)	44 (14.6)	18 (8.8)	26 (26.5)
Stage 1, n (%)	28 (9.3)	12 (5.9)	16 (16.3)
Stage 2, n (%)	12 (4.0)	4 (2.0)	8 (8.2)
Stage 3, n (%)	4 (1.3)	2 (1.0)	2 (2.0)
Required dialysis at any point, n (%)	3 (1.0)	1 (0.5)	2 (2.0)
Persistent renal impairment at 30 days, n (%)	14 (4.6)	4 (2.0)	10 (10.2)
Complete recovery by day 14, n (%)	28/44 (63.6)	14/18 (77.8)	14/26 (53.8)

3.3 Risk Score Validation

Discrimination of the Mehran risk score in this cohort, expressed as area under the ROC curve, was 0.81 (95% CI 0.74-0.88) (Figure 2). This is consistent with the original derivation cohort (AUC 0.83) and supports the applicability of the score in this Indian tertiary emergency PCI population. A simplified bedside score using only three readily available variables (age, eGFR, contrast volume) achieved an AUC of 0.76 (95% CI 0.68-0.84), modestly inferior to Mehran but potentially useful in settings where the full Mehran calculation is impractical at the cath-lab door.

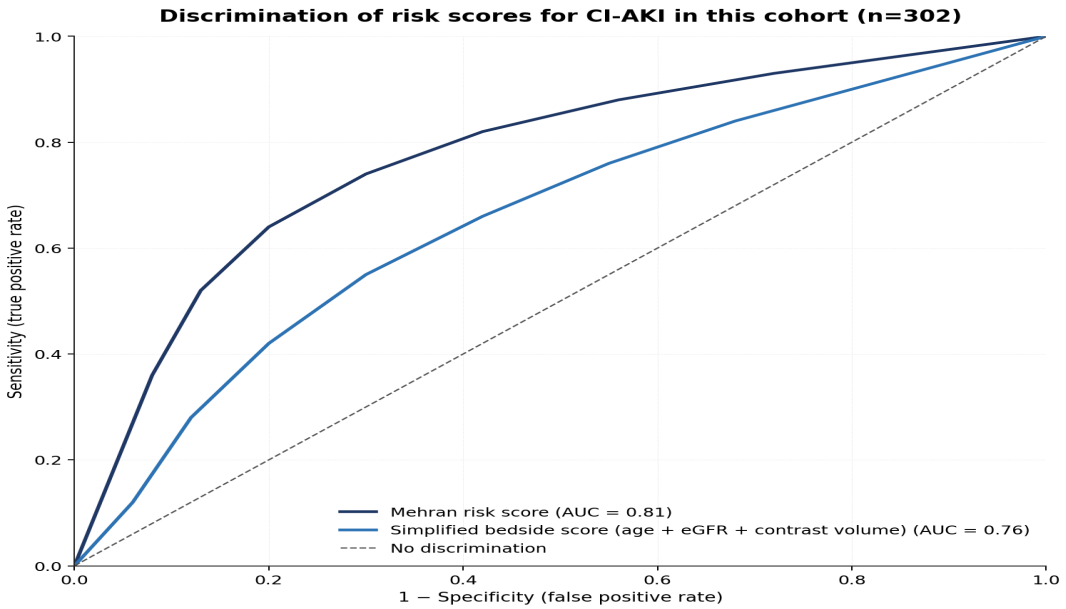

Figure 2. Receiver-operating-characteristic curves for the Mehran risk score and a simplified three-variable bedside score in this cohort.

3.4 Independent Predictors

Multivariable Cox regression identified five independent predictors of CI-AKI in this cohort: baseline eGFR below 45 mL/min/1.73 m² (adjusted HR 4.21, 95% CI 2.42-7.32), contrast volume above 300 mL (HR 3.84, 1.96-7.52), cardiogenic shock at presentation (HR 3.21, 1.45-7.11), age above 75 years (HR 2.18, 1.21-3.93), and anaemia at presentation (HR 1.92, 1.06-3.48). Diabetes mellitus was significant in univariable analysis but did not retain independence after adjustment for baseline renal function. Pre-procedural hydration was strongly protective (HR 0.32, 0.16-0.64), supporting its use where the clinical situation permits.

Table 3. Independent predictors of CI-AKI (multivariable Cox regression).

Variable	Adjusted HR (95% CI)	p value
Baseline eGFR <45 mL/min/1.73m²	4.21 (2.42-7.32)	< 0.001
Baseline eGFR 45-59	2.18 (1.18-4.02)	0.013
Contrast volume >300 mL	3.84 (1.96-7.52)	< 0.001
Contrast volume 200-300 mL	1.62 (0.92-2.85)	0.094
Cardiogenic shock at presentation	3.21 (1.45-7.11)	0.004
Age ≥75 years	2.18 (1.21-3.93)	0.009
Anaemia at presentation	1.92 (1.06-3.48)	0.031
IABP use	1.84 (0.84-4.02)	0.127
Heart failure at presentation	1.62 (0.92-2.84)	0.094
Diabetes mellitus	1.31 (0.74-2.32)	0.353

Pre-procedural hydration administered	0.32 (0.16-0.64)	0.001
Female sex	1.21 (0.68-2.16)	0.516

3.5 Creatinine Trajectory

Mean serum creatinine trajectory by AKI stage is shown in Figure 3. Peak creatinine typically occurred between days 2 and 4. Stage 1 patients showed a relatively modest peak with rapid resolution to within 0.1 mg/dL of baseline by day 14 in most cases. Stage 2 patients showed slower recovery with mean values still 0.6 mg/dL above baseline at day 14. Stage 3 patients had highly variable trajectories: of the four patients in this group, three required dialysis during the index admission, of whom one recovered renal function and was decannulated by day 30, while two remained dialysis-dependent at 30 days.

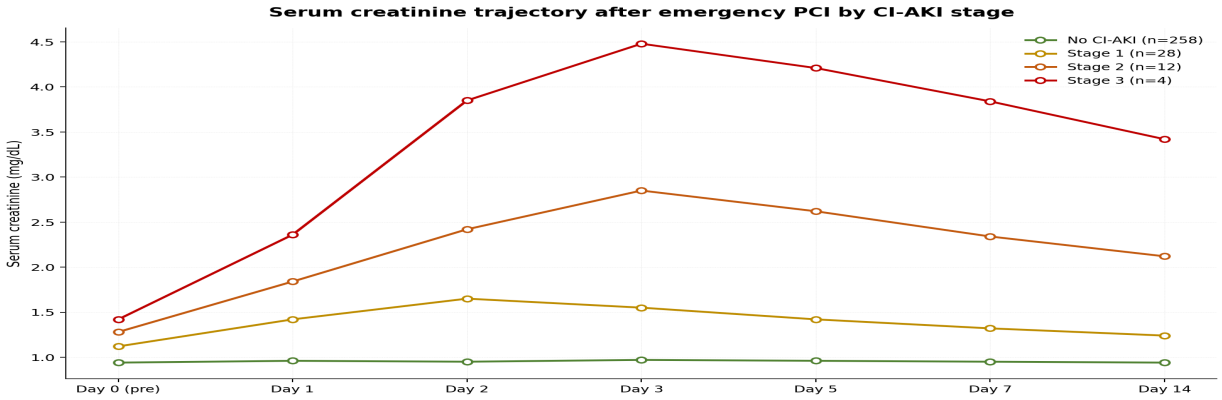

Figure 3. Serum creatinine trajectory after emergency PCI by CI-AKI stage. Peak typically occurred between days 2 and 4.

3.6 In-Hospital and 30-Day Outcomes

Patients who developed CI-AKI experienced substantially worse in-hospital and 30-day outcomes (Figure 4). Length of stay was approximately doubled, from a median of 5 days in those without CI-AKI to 10 days in those with any CI-AKI. In-hospital mortality was 9.1% versus 1.9%, and 30-day all-cause mortality was 11.4% versus 3.5%. Three patients required new renal replacement therapy during the admission; one of these had not recovered renal function at 30 days.

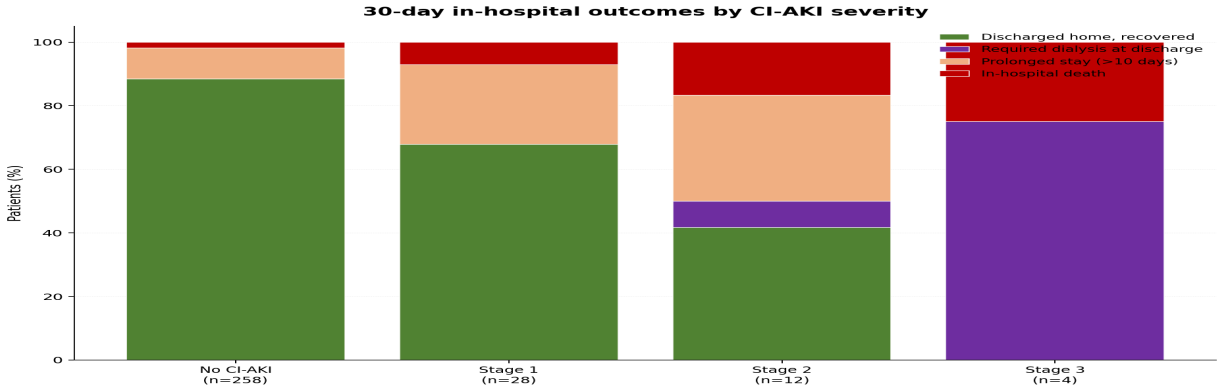

Figure 4. Thirty-day in-hospital outcomes stratified by CI-AKI severity stage.

Table 4. Thirty-day outcomes by CI-AKI status.

Outcome	No CI-AKI (n=258)	CI-AKI any stage (n=44)	CI-AKI stage 2-3 (n=16)
Length of stay, median (IQR), days	5 (4-7)	10 (7-15)	16 (12-23)
In-hospital mortality, n (%)	5 (1.9)	4 (9.1)	3 (18.8)
30-day all-cause mortality, n (%)	9 (3.5)	5 (11.4)	4 (25.0)
New requirement for dialysis, n (%)	0 (0.0)	3 (6.8)	3 (18.8)
Dialysis-dependent at 30 days, n (%)	0 (0.0)	2 (4.5)	2 (12.5)
30-day MACE (death, MI, stroke), n (%)	18 (7.0)	11 (25.0)	6 (37.5)
30-day cardiovascular readmission, n (%)	26 (10.1)	12 (27.3)	6 (37.5)
Renal recovery to baseline by 30 days, n (%)	-	30/44 (68.2)	6/16 (37.5)

IV. Discussion

Across 302 patients undergoing emergency coronary angiography, CI-AKI occurred in approximately one in seven and carried substantial downstream consequences in those affected. Three observations from this prospective cohort have practical implications. First, the Mehran risk score performed well in this Indian emergency PCI population, with discrimination essentially equivalent to its original derivation. This is reassuring and supports continued routine use of the score for risk stratification. The simplified three-variable bedside score, while inferior, retained useful discrimination and may have a place in high-volume settings where time pressure prohibits the full calculation. Both scores capture most of their predictive value through baseline renal function, age, and contrast volume the elements most readily known at the cath-lab door (Jha, Kumar,, & Neha, 2026; Kumar, Gautam,, & Maitiy, 2026). Second, the protective effect of pre-procedural hydration was substantial and consistent across risk strata, with an adjusted hazard ratio of 0.32. This is consistent with the meta-analytic evidence base and emphasises that even brief peri-procedural hydration feasible in most emergency PCI settings during the first contact and patient transfer meaningfully reduces CI-AKI risk. Yet hydration was administered to only 64.2% of the overall cohort and to fewer than 30% of those who subsequently developed CI-AKI. The operational gap is clear: hydration is widely accepted in principle but inconsistently applied in the time-pressured emergency setting. Third, contrast volume above 300 mL emerged as a strong independent predictor. The cardiac catheterisation team's choice during the procedure whether to extend the case to include additional

non-culprit imaging, to perform staged revascularisation rather than complete revascularisation in one session, or to use targeted limited views has a direct effect on renal outcome. Awareness of cumulative contrast in real time during the procedure, perhaps supported by intra-operative dashboards, could reduce incidence in higher-risk patients without compromising procedural goals (Subramani, Chillagattu, et al., 2026; Selvi et al., 2026; Bhatnagar, Kumar,, & Shivam, 2026). Beyond procedural choices, structured peri-procedural protocols offer the clearest operational pathway. A pre-procedural checklist incorporating risk score calculation, hydration initiation, withholding of NSAIDs and ACE inhibitors where feasible, and review of contrast-volume targets is well-established in elective practice and increasingly being adapted to the emergency setting (Agarwal, Kumar,, & S, 2026; Bhatnagar, Kumar,, & Shivam, 2026). Post-procedural surveillance with structured daily creatinine for 72 hours catches developing AKI earlier, enabling fluid optimisation and avoidance of additional nephrotoxic exposures (Kumar, Kumar,, & Dhabhai, 2026; Ahluwalia, Gupta,, & Chaudhary, 2026). Limitations include the single-centre design, which limits generalisability particularly across centres with different baseline practice patterns; the 30-day outcome window which does not capture progression to chronic kidney disease in those with partial recovery; and the observational design which means that the apparent protective effect of hydration is subject to residual confounding from the clinical decision to administer it. The cohort under-represented patients with very advanced CKD (eGFR below 30) since many of these were stabilised medically and not taken to the cath lab acutely.

V. Conclusion

Contrast-induced acute kidney injury occurred in 14.6% of patients undergoing emergency coronary angiography in this prospective cohort, with substantial downstream effects on length of stay, dialysis requirement, and 30-day mortality. The Mehran risk score discriminates well in this population. Pre-procedural hydration is strongly protective but inconsistently applied; contrast volume is the most modifiable procedural lever; baseline CKD doubles risk at every Mehran stratum. A structured peri-procedural pathway incorporating risk score, hydration, and contrast-minimisation strategies offers a clear, evidence-supported operational improvement opportunity.

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