Research Article

Incidence of Contrast-Associated Acute Kidney Injury and Need for Renal Replacement Therapy in Patients who have Undergone Elective PCI in the Last 3 Years in a Tertiary Care Hospital

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Abstract: Background: With more than 660,000 percutaneous coronary interventions (PCI) carried out each year nationwide, Post-procedure acute kidney damage, sometimes referred to as contrast-associated acute kidney injury (CA-AKI), can complicate up to 14% of all PCIs.

Objective: The aim of this study is to provide an assessment of the frequency of contract-induced acute kidney injury in patients undergoing PCI in a single healthcare center.

Materials and Methods: This is a retrospective, clinical research conducted at Dr. Ziauddin Hospital, Karachi, from 20th June 2023 to 30th August 2023. Diagnosis of ischemic coronary artery disease was made and elective or emergency percutaneous coronary intervention (PCI) was performed. SPSS 22 used to analyze the data. For the continuous variables, mean ± standard deviation (SD) or median with interquartile range (IQR) were performed.

Result: A total of 166 patients were enrolled in the study, Mean age estimation of the study population was 63 ± 9.31 years, acute kidney injury was diagnosed in 11 (6.6%) while 157 (94.5%) had no AKI incident. Serum creatinine of baseline 1.4 ± 1.2 mg/dL and 2.1 ± 21.0 mg/dL, day 03 (after 48 hours) 1.5 ± 0.2 mg/dL and 2.5 ± 1.9 mg/dL, and day 30 1.4 ± 1.1 mg/dL and 2.0 ± 1.9 mg/dL were estimated in AKI and non-Aki patients respectively, and results indicated a mean difference of 0.7 ± 0.6 mg/dL in baseline serum creatinine of AKI patients and non-AKI patients. The remaining laboratory investigations were insignificant.

Conclusion: However, CA-AKI is a concern for subspecialties especially cardiology leading to withholding of clinically indicated interventions causing an increased risk of morbidity and mortality. This study identified that patient characteristics such as comorbidities can lead to CA-AKI.

Keywords: Acute Kidney Injury (AKI), Percutaneous coronary intervention (PCI), Contrast-induced, Comorbidities, Patient, Morbidity and Mortality.

INTRODUCTION

Coronary artery disease is known as the principal reason of illness and death worldwide, with more than 660,000 percutaneous coronary interventions (PCI) carried out each year, PCI is the preferred therapy for refractory symptomatic coronary artery disease and acute coronary syndromes [1, 2]. Post-procedure acute kidney damage can complicate up to 14% of all PCIs [2, 3]. Acute renal failure (AKI) defines as rapid and normally reversible deterioration in kidney function or glomerular filtration rate (GFR) [3]. However, blood urea nitrogen (BUN) or creatinine levels may remain in normal ranges [4, 5]. The prevalence of AKI specifically inhospitalized pateints is reported as up to 7% of hospital admissions and 30% of ICU patients [6]. According to Kidey Disease Improving Global Organization (KDIGO), the definition of AKI is "Increase in serum creatinine by 0.3 mg/dL within 48 hours and/or Increase in serum creatinine to 1.5 times or more baseline within the prior seven days and/or Urine volume less than 0.5 mL/kg/h for at least 6 hours" [7-9].

Patients who acquire CA- AKI have significantly higher hospital stay duration, increased financial burden, and increased mortality rate as compared to no AKI patients with a 36% likelihood of death during hospital stay and a 12% chance of death one year after being discharged [10].

According to 3 distinct CI-AKI criteria, CI-AKI was observed in ≤15.7% of patients in patients with acute coronary syndrome.

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It is unclear if this variability results from variations in the studies or advancements in CI-AKI prophylaxis [7, 9, 10]. Contrast volume is routinely recognized as a significant determinant, and it is one of the few risk factors that may be changed in a periprocedural situation. Therefore, the core of many earlier attempts to avoid CA- AKI has been to decrease the amount of contrast utilized during PCI [7]. Nevertheless, despite widespread support for lowering contrast usage, current techniques for identifying a safe contrast volume for a specific patient are insufficient and fail to take into account many patient risk factors, such as hypertension or diabetes mellitus, which are known to increase the risk CA-AKI [11]. By providing healthcare providers with a tangible value to use for procedural planning before or even during cardiac catheterization (e.g., whether to stage a multiverse PCI procedure if a significant amount of contrast has already been used or whether to use special contrast-sparing techniques), the development of more precise safe contrast volume limits could significantly reduce CA-AKI risk [5, 7, 12].

After the treatment, the majority of patients with CI-AKI would only temporarily lose their ability to urinate, with serum creatinine and diuresis often returning to normal within 7 to 10 days. However, a significant number of studies revealed that CI-AKI is linked to an increased risk of both short- and long-term unfavorable clinical outcomes [9]. Numerous of these studies don't account for baseline risk variables, which may cause them to exaggerate the rise in mortality and length of hospital stay that is a result of renal impairment [12].

Recent epidemiological studies conducted in North America and Europe have revealed that the frequency of acute kidney injury (AKI) is rising alarmingly. According to statistics, contrast-induced acute kidney injury (CI-AKI) is the third most frequent reason for hospital-acquired renal failure [4, 6]. In individuals with normal baseline renal function, the incidence of acute renal insufficiency following PCI is 20%; however, in patients with baseline creatinine levels above 176 μmol/L(or 2 mg/dL) before PCI, it can reach up to 30% [9-12]. Patients who already have congestive heart failure, diabetes mellitus, renal insufficiency, or advanced age are more likely to develop CI-AKI after PCI [3]. The largest risk factor is preexisting renal impairment, and dehydration increases the risk [8, 11]. Although there isn't conclusive proof that CI-AKI and inadequate glycemic management or the length of diabetes are related, constricted glycemic control should be achieved before exposure to contrast media [10-12].

The purpose of this study is to assess the frequency of contract-induced acute kidney injury in patients undergoing PCI in a single healthcare center.

MATERIALS AND METHODS

This is a retrospective, clinical research conducted at Dr. Ziauddin Hospital, Karachi, from 20th June 2023 to 30th August 2023. Approval acquired from institutional research review committee to conduct the study. Patients with a diagnosis of ischemic artery disease with the help of Troponin I test results and ECG, prescribed for elective and emergency percutaneous coronary intervention (PCI). The sample size was calculated with the help of the WHO sample size calculator, keeping the total number of PCI cases from the institute as population, n=300, confidence interval as 95% and margin of error as 5%, the minimum required sample size is 168. Data was documented from patients of both genders, aged between 18 to 90 years, a pre-structured questionnaire with three parts was filled, part A contained demographic details of patients including age, gender, and comorbidities. Part B reported pre-procedure laboratory investigations and procedure details including hemoglobin, hematocrit, pre-procedure hydration, type of hydration and rate of hydration, etc. while part C documented post-procedure renal functioning tests, including urea, creatinine, Sodium, bicarbonate, chloride and estimated glomerular filtration rate as a baseline, day three and day thirty post-procedure, while 40 ml of contrast was used per patients in study.

The inclusion criteria for the study required that patients have undergone an elective PCI procedure for ACS; the exclusion criteria included the patients had only undergone coronary angiography, had received contrast medium exposure less than one week before the procedure; had undergone peripheral artery procedures, had end-stage renal disease (ESRD), dialysis and declined to take part in the study. Kidney disease improving global outcomes (KDIGO) [4] criteria of increase in serum creatinine by 0.3 mg/dL or more within 48 hours was used to assess acute kidney injury diagnosis, patient's baseline serum creatinine, and repeated creatinine after 48 hours was calculated to measure AKI.

STATISTICAL ANALYSIS

Statistical Package of Social Sciences (SPSS) version 22 was used to enter, sort, and analyze the data. For the continuous variables, we presented the baseline characteristics as mean ± standard deviation (SD) or median with interquartile range (IQR), and for the categorical variables, as count and percentage. The CI-AKI risk model was developed in the derivation cohort by utilizing logistic regression analysis to identify the independent CI-AKI interpreters. Each independent risk factor was given a weighted integer coefficient value, and the sum of the coefficients was used to construct the CI-AKI risk.

RESULT

A total of 166 patients were enrolled in the study, gender distribution indicated 111 (66.8%) of the male population and 57 (34.2%) of the female population. The mean age estimation of the study population was 63 ± 9.31 years, acute kidney injury was diagnosed in 11 (6.6%) while 157 (94.5%) had no AKI incident.

Distribution of patients according to acute kidney injury was associated with demographic details and comorbidities, indicating a higher number of the male population in AKI with 8 (4.8%) and p-value of 0.452, and chronic kidney disease as comorbid-

ity with 4 (%) followed by Diabetes mellitus and hypertension in combination in 3 (%) and Diabetes mellitus, hypertension and IHD in 3 (%), with p-value of 0.09. Use of diuretics was not significant in AKI patients, with a p-value of 0.43, while usage of metformin, NSAIDs, and Nephrotoxic medicines was non-significant as well with a p-value of 0.07, 0.93, and 0.22 respectively (Table **1**).

Table 1. Descriptive Analysis of Acute Kidney Injury in the Study Population.

Variables		Acute Kidney Injury		P-Value
		Yes (n=11)	No (n=155)	
Gender	Male	8 (4.8%)	103 (62%)	0.452
	Female	3 (1.8%)	54 (32.5%)	
Comorbidities	DM	0	23 (13.8%)	0.099
	HTN	1 (0.6%)	40 (24%)	
	DM +HTN	3 (1.8%)	55 (33.1%)	
	CKD	4 (2.4%)	27 (16.2%)	
	Psychiatric illness	0	3 (1.8%)	
Diuretics	Yes	2 (1.2%)	41 (24.6%)	0.432
	No	9 (5.4%)	116 (69.8%)	
Metformin	Yes	3 (1.8%)	13 (7.8%)	0.073
	No	8 (4.8%)	144 (86.7%)	
NSAIDs	Yes	0	1 (0.6%)	0.935
	No	11 (6.6%)	156 (93.9%)	
Nephrotoxic Meds	Yes	5 (3%)	47 (28.3%)	0.225
	No	6 (3.6%)	110 (66.2%)	

Serum creatinine of baseline, day 03 (after 48 hours), and day 30 were estimated in all patients and results indicated a mean difference of 0.7 ± 0.6 in baseline serum creatinine of AKI patients and non-AKI patients. There was no significant difference between the mean age values of both groups. However, the serum creatinine mean values identified a significant p-value in AKI and non-AKI patients with 0.014, similarly estimated glomerular filtration rate mean value was significant for day 03 on AKI and Non-AKI patients with 0.007. The remaining laboratory investigations were insignificant (Table **2**).

Table 2. Comparative Analysis of Mean Values between AKI and Non-AKI Patients.

Variables		Non-AKI	AKI	P-Value
		Mean ± SD	Mean ± SD	
Age (in years)		63 ± 9.2	62.2 ± 10.9	0.79
Creatinine	Baseline	1.4 ± 1.2	2.1 ± 2.0	0.098
	Day 3	1.5 ± 1.2	2.5 ± 1.9	0.014
	Day 30	1.4 ± 1.1	2.0 ± 1.9	0.08
eGFR	Baseline	62.9 ± 32.5	49.1 ± 28.1	0.171
	Day 3	57.8 ± 27.9	34.5 ± 17.3	0.007
	Day 30	65.8 ± 32.1	46.4 ± 33	0.054
Urea	Baseline	50.5 ± 43.6	64.9 ± 59.9	0.306
	Day 3	53 ± 44.4	68.5 ± 60.8	0.277
	Day 30	51.7 ± 43.7	66.2 ± 55.7	0.299
Sodium	Baseline	138.9 ± 4.1	139.2 ± 6.7	0.821
Potassium	Baseline	4 ± 0.5	4.1 ± 0.4	0.569
Chloride	Baseline	103.3 ± 4.7	101.1 ± 5.5	0.144
HCO_3	Baseline	23.3 ± 4.8	22 ± 5.3	0.390
Albumin (serum)	Baseline	3.8 ± 0.4	3.8 ± 0.4	0.607

DISCUSSION

The pathophysiological mechanism behind CI-AKI is complex and yet poorly understood. However, hemodynamic worsening is known to be a critical factor in the development of CI-AKI. Heart failure is a contributing factor to hemodynamic instability, which can lower effective renal perfusion pressure [13]. This can activate the sympathetic nervous system and the renin-angiotensin system, which in turn raises levels of oxygen radicals and inflammatory factors and ultimately leads to the development of CI-AKI. In our study mean age of the patients was similar to another study of CA-AKI assessment in the Pakistani population. The gender distribution was identified as similar to other studies [14]. The Acute Kidney Injury Network defines oliguria as an increase in serum creatinine of ≥0.3 mg/dL; this might become the new benchmark. A revised definition of CI-AKI has been proposed by the European Renal Best Practice (ERBP) position statement on the Kidney Disease Improving Global Outcomes (KDIGO) guidelines [4, 12]. This classification suggests a rise of serum creatinine of at least 50% or 0.3 mg/dL at 48 hours. In our study, a similar definition was used to evaluate CA-AKI and distribution suggested an overall 6.6% frequency of CA-AKI. The Contrast-Induced Nephropathy Consensus Working Panel has advocated utilizing the relative rise in creatinine to define CI-AK, regardless of whether absolute or relative increases in creatinine levels are used.

Patients with poor EF had a considerably greater risk of CI-AKI following the second stage of staged coronary revascularization

tion for acute myocardial infarction, according to new research involving 138 patients with the condition [15].

Shacham Y, *et al*. [16] observational analyses demonstrated that declining EF was a reliable independent predictor of CI-AKI. However, following many confounder adjustments, Kurtul A, *et al*. [17] demonstrated the opposite impact. A further single-center prospective observational research examined the variation in CI-AKI incidence amongst HF groups. The findings of these investigations demonstrated that the HFpEF, HFrEF, and HFmrEF groups did not differ significantly from one another.

According to published research, the prevalence of CI-AKI ranges from 3.3% to 14.5% [17]. The incidence of CI-AKI was estimated to be around 7.1% in a cohort of 985,737 patients from the National Cardiovascular Data Registry (NCDR) who were having elective or urgent percutaneous coronary intervention (PCI) [18]. In the United States (US), the overall incidence of acute myocardial infarction (AKI) fell by 26% between 2000 and 2008, according to a major national research that included 33,249 hospitalizations for AMI [19]. There was no discernible difference in the risk of CI-AKI between patients who got contrast and those who did not, according to a meta-analysis encompassing 25,950 patients who had imaging procedures [Relative Risk (RR) 0.79, 95% CI 0.62–1.02, p = 0.07] [20].

Numerous research investigations have identified risk variables linked to CA-AKI, which may be categorized as either modifiable or non-modifiable. They consist of multiple myeloma, dehydration, cardiovascular disease, use of diuretics, multiple use of contrast media within 24 hours, female sex, advanced age, and the quantity and kind of contrast media and intervention measures used during administration [21]. The risk for CA-AKI was shown to be elevated by heart failure or poor ejection fraction, age, CKD, hypotension or shock, and diabetes mellitus. Increased inpatient mortality, significant late mortality, and late chronic hemodialysis use were all linked to CA-AKI [22, 23].

Patients with a continuous rise in SCr showed the greatest manifestation of this. Age, diabetes, CKD, and reduced left ventricular performance were patient-related predictors of CA-AKI. Technical aspects such as the use of IVUS, the retrograde approach, or the applied interventional strategy did not affect the outcome; nevertheless, a longer fluoroscopy duration was linked to a higher risk of CAAKI [24-26].

The latter may serve as a sign of more intricate and difficult steps. Likely, this was not due to the contrast itself, as other CTO-PCI investigations have also noted. With more advanced stages of CKD, the rate of CA-AKI rose significantly [27]. Therefore, identifying modifiable risk factors is crucial for CA-AKI prevention. Anaemia is a significant clinical variable that may be addressed before the operation, however, this was not recognized in earlier CTO-PCI publications. As oxygen levels in the kidney's tubular system decrease during transit, anemia might exacerbate this process and result in tubular injury, which would be a mechanism of CA-AKI [28]. Prognosis and clinical outcome in patients having PCI and coronary angiography as well as other CM-enhanced CT scans have been demonstrated to be considerably impacted by CI-AKI. Numerous research investigations throughout the years have demonstrated a correlation between CI-AKI and increased rates of both in-hospital and long-term death. It's important to note that findings from research examining the relationship between CI-AKI and mortality differ significantly [29, 30].

CONCLUSION

The incidence of coronary artery disease is on the rise in developing countries, and PCI is used as a standardized procedure to treat the issue. However, CA-AKI is a concern for subspecialties especially cardiology leading to withholding of clinically indicated interventions causing an increased risk of morbidity and mortality. This study identifies the incidence of AKI after PCI in a single centre. Given the low incidence of AKI in the study population it did not identify any potential predictors of AKI. Infact the study was not powered to detect such a difference. One of the limitations of the study was lack of data on contrast volume used during PCI which is considered to be one of the determinants of AKI in PCI.

AUTHORS' CONTRIBUTION

- **Kaneez Zehra**: Objective, Subject specialist.
- **Namirah Iftikhar and Michelle Farooq**: Write-up, Data collecting.
- **Ghulam Hussain Soomro**: Data entry, Data collection.
- **Muhammad Osama Rehan Khalid**: Ethical consideration, Write-up.
- **Ashar Ekhlaq Ahmed**: Data analysis, Result interpretation.

CONFLICT OF INTEREST

None declared.

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REFERENCES

[1] Azzalini L, Spagnoli V, Ly HQ, *et al*. Contrast-induced nephropathy: From pathophysiology to preventive strategies. Can J Cardiol 2016; 32(2): 247-55.

[2] Rudnick MR, Leonberg-Yoo AK, Litt HI, Cohen RM, Hilton S, Reese PP. The controversy of contrast-induced nephropathy with intravenous contrast: What is the risk? Am J Kidney Dis 2020; 75(1): 105-13.

[3] Chandiramani R, Cao D, Nicolas J, Mehran R. Contrast-induced acute kidney injury. Cardiovasc Interv Ther 2020; 35: 209-17.

[4] Batra MK, Sial JA, Kumar R, *et al*. Contrast-induced acute

kidney injury: The sin of primary percutaneous coronary intervention. Pakistan Heart J 2018; 51(2): Available from: https://pakheartjournal.com/index.php/pk/article/view/1499

[5] Davenport MS, Perazella MA, Yee J, *et al*. Use of intravenous iodinated contrast media in patients with kidney disease: Consensus statements from the American College of Radiology and the National Kidney Foundation. Radiology 2020; 294(3): 660-8.

[6] Lameire N, Kellum JA, KDIGO AKI Guideline Work Group. Contrast-induced acute kidney injury and renal support for acute kidney injury: A KDIGO summary (Part 2). Crit Care 2013; 17(1): 205.

[7] Wang Y, Zhu B, Zhao L, *et al*. The incidence, risk factors, and outcomes of contrast-induced nephropathy in patients undergoing percutaneous coronary intervention: A prospective cohort study. Int J Cardiol 2014; 176(3): 1046-9.

[8] Machado MN, Nakazone MA, Maia LN. Acute kidney injury based on KDIGO (Kidney Disease Improving Global Outcomes) criteria in patients with elevated baseline serum creatinine undergoing cardiac surgery. Rev Bras Cir Cardiovasc 2014; 29(3): 299-307.

[9] Kusirisin P, Chattipakorn SC, Chattipakorn N. Contrast-induced nephropathy and oxidative stress: Mechanistic insights for better interventional approaches. J Transl Med 2020; 18(1): 400.

[10] McCullough PA, Choi JP, Feghali GA, *et al*. Contrast-induced acute kidney injury. J Am Coll Cardiol 2016; 68(13): 1465-73.

[11] Aijaz S, Ahmed N, Akhter Z, *et al*. Clinical characteristics and in-hospital outcome in percutaneous coronary interventions with ST elevation myocardial infarction patients developing acute kidney injury. J Pak Med Assoc 2019; 69(12): 1827-33.

[12] Ammar A, Khowaja S, Kumar R, *et al*. Significance of left ventricular end diastolic pressure for risk stratification of contrast-induced acute kidney injury after primary percutaneous coronary intervention. Pak Heart J 2022; 55(3): 247-52.

[13] Rahman G, Aziz H, Yaqoob N, Yasmin R, Tareen M, Ahmed F. Examine the risk factors for contrast-induced nephropathy in patients undergoing Percutaneous Coronary Intervention (PCI) and the prevalence of this complication. Pak J Med Health Sci 2023; 17(05): 245-7.

[14] Kim JH, Yang JH, Choi SH, *et al*. Predictors of outcomes of contrast-induced acute kidney injury after percutaneous coronary intervention in patients with chronic kidney disease. Am J Cardiol 2014; 114(12): 1830-5.

[15] Narula A, Mehran R, Weisz G, *et al*. Contrast-induced acute kidney injury after primary percutaneous coronary intervention: Results from the HORIZONS-AMI substudy. Eur Heart J 2014; 35(23): 1533-40.

[16] Shacham Y, Steinvil A, Leshem-Rubinow E, *et al*. Association between time to reperfusion and echocardiography assessed left ventricular filling pressure in patients with first ST-segment elevation myocardial infarction undergoing primary coronary intervention. Cardiol J 2014; 21(4): 357-63.

[17] Kurtul A, Gok M, Esenboga K. Prognostic nutritional index predicts contrast-associated acute kidney injury in patients with ST-segment elevation myocardial infarction. Acta Cardiol Sin 2021; 37(5): 496-503.

[18] Shibata K, Wakabayashi K, Ishinaga T, *et al*. Feasibility, safety, and long-term outcomes of zero-contrast percutaneous coronary intervention in patients with chronic kidney disease. Circ J 2022; 86(5): 787-96.

[19] Tsai TT, Patel UD, Chang TI, *et al*. Contemporary incidence, predictors, and outcomes of acute kidney injury in patients undergoing percutaneous coronary interventions: Insights from the NCDR Cath-PCI registry. JACC Cardiovasc Interv 2014; 7: 1-9.

[20] Amin AP, Salisbury AC, McCullough PA, *et al*. Trends in the incidence of acute kidney injury in patients hospitalized with acute myocardial infarction. Arch Intern Med 2012; 172: 246-53.

[21] Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pract 2012; 120(4): c179-84.

[22] Palevsky PM, Liu KD, Brophy PD, *et al*. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for acute kidney injury. Am J Kidney Dis 2013; 61(5): 649-72.

[23] Almendarez M, Gurm HS, Mariani J, *et al*. Procedural strategies to reduce the incidence of contrast-induced acute kidney injury during percutaneous coronary intervention. JACC Cardiovasc Interv 2019; 12(19): 1877-88.

[24] Mehran R, Dangas GD, Weisbord SD. Contrast-associated acute kidney injury. New Engl J Med 2019; 380(22): 2146-55.

[25] Huang J, Lyu Y, Li J, Cheng P, Jiang Y, Pu K. A renal-clearable duplex optical reporter for real-time imaging of contrast-induced acute kidney injury. Angew Chem Int Ed Engl 2019; 58(49): 17796-804.

[26] Ronco F, Tarantini G, McCullough PA. Contrast induced acute kidney injury in interventional cardiology: An update and key guidance for clinicians. Rev Cardiovasc Med 2020; 21(1): 9-23.

[27] Çınar T, Karabağ Y, Tanık VO, Çağdaş M, Rencüzoğulları İ, Öz A. The investigation of TIMI risk index for prediction of contrast-induced acute kidney injury in patients with ST elevation myocardial infarction. Acta Cardiol 2020; 75(1): 77-84.

[28] Han M, Lee HW, Lee HC, Kim HJ, Seong EY, Song SH. Impact of nutritional index on contrast-associated acute kidney injury and mortality after percutaneous coronary intervention. Sci Rep 2021; 11(1): 7123.

[29] Zeng JL, Xiang YF, Zhang LW, *et al*. Predictive value of systemic inflammation score for contrast-associated acute kidney injury and adverse outcomes among patients undergoing elective percutaneous coronary intervention. J Inflamm Res 2023; 16: 2845-54.

[30] Wang Y, Guo Y. Renal Guard system and conventional hydration for preventing contrast-associated acute kidney injury in patients undergoing cardiac interventional procedures: A systematic review and meta-analysis. Int J Cardiol 2021; 333: 83-9.

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