

Effect of coronary angiography on kidney function in patients with normal and raised serum creatinine

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Abstract

Objective: To assess the impact of coronary angiography on patients with stable coronary artery disease and renal dysfunction.

Method: The descriptive, retrospective study was conducted from February 2022 to June 2023 at the Indus Hospital Health Network, Karachi, and comprised data from January 2015 to December 2021 of all patients regardless of age and gender who had renal dysfunction with a serum creatinine of >1.5 mg/dl who underwent coronary angiography. Data was analysed using SPSS 26.

Results: Of the 5,376 patients, 3,630 (67.5%) were male and 1,746 (32.5%) were female. The overall mean age was 55.1 ± 10.3 (range: 18-94 years). Hypertension was the most frequent comorbid 3,405 (63.3%), followed by diabetes mellitus 2,084 (38.8%). The patients' mean creatinine levels significantly increased after angiography ($p < 0.05$). Female gender, old age, diabetes, hypertension, congestive cardiac failure, ischaemic heart disease, cerebrovascular accident and glomerular filtration rate had significant association with contrast-induced nephropathy ($p < 0.05$).

Conclusion: Contrast-induced nephropathy was found to be an important cause of acute renal dysfunction. Female gender, advanced age, low glomerular filtration rate at the time of procedure, and comorbid conditions were associated with contrast-induced nephropathy.

Keywords: Contrast media, Acute kidney injury, Coronary artery disease, Nephropathy. (JPMA 76: 1623; 2026)

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Introduction

Revascularisation of the coronary arteries by percutaneous coronary intervention (PCI) is one of the preferred interventions for patients with stable coronary artery disease (CAD). Globally, CAD itself is one of the leading causes of cardiovascular morbidity and mortality, which creates an enormous burden on healthcare economics.¹ Coronary angiography (CA) is the procedure that uses an intravascular contrast medium to determine the site of occlusion. The administration of the contrast can lead to acute kidney injury (AKI).² The mechanism of AKI following the administration of contrast medium is not fully understood, but several possible mechanisms have been proposed, including haemodynamic changes that lead to a reduction in renal flow, iodine-induced tubular necrosis, medullary hypoxic injury due to impaired microcirculation, cell death due to selective intracellular signalling pathway, and, lastly, inflammation.³ Additionally, oxidative stress and renal vasoconstriction contribute to the pathogenesis of contrast-induced nephropathy (CIN), which further exacerbates renal damage. Selective intracellular signalling pathways, cell death and inflammation are also critical

factors in the development of AKI. Furthermore, the CA procedure itself imparts many complications to the patients, like myocardial infarction (MI), stroke, embolism and death, other than AKI with contrast.⁴

Although the incidence of AKI reportedly ranges 3-50%, there are differences of opinion regarding the potential nephrotoxicity of contrast medium. Intrinsically, cardiac catheterisation has procedural complications that may affect kidney perfusion, including hypotension, blood-loss, arrhythmias, fluid restriction and use of a furosemide diuretic. These complications are not present in other contrast study procedures, like computed tomography (CT) and others.⁵ Irrespective of the procedure, the use of a high volume of contrast >350 ml, or 4 ml per kg bodyweight, and repeated administration within 72 hours after initial administration have been reported to be linked with exaggerated risk.⁶

Clinical presentations like the severity of the MI (ST-segment elevation vs sub-endocardial infarction) also pose an increased risk of AKI. Additionally, CA requires direct injection of contrast into the artery, which arteriography enables, allowing for the direct delivery of the concentrated medium to the kidney compared to conventional intravenous (IV) contrast exposure.⁷

People of Indo-Asian origin have one of the highest susceptibilities to CAD in the world, and it is, therefore,

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unsurprising that CAD is now the leading cause of death in the subcontinent.⁸ Pakistan is among the low- and middle-income countries (LMICs) with a dense population of >225 million. There is a rapid change in lifestyle, especially in the urban areas of the country. One in four middle-aged adults in Pakistan has CAD, and the risks are uniformly high in the young and women.⁹ Similarly, the incidence and prevalence of chronic kidney disease (CKD) are also on the fast-paced rise in Pakistan.¹⁰ Patients with stable CAD and advanced CKD represent a high-risk population with distinctive challenges. Because these patients have been systematically excluded from most clinical trials of invasive treatment strategies, the information about the impact of cardiac catheterisation in this population is mostly unknown.¹¹ There is a dire need to review the data of cardiac catheterisation and its impact on patients with normal kidney function, as well as those with kidney dysfunction in the Pakistani population. Although several prediction models have been developed to estimate the risk of AKI following CA, they have not been validated in South Asian populations.¹² The current study was planned to fill the gap in literature by evaluating renal function changes post-CA in a Pakistani cohort.

Materials and Methods

The retrospective, descriptive study was conducted from February 2022 to June 2023 at the Indus Hospital Health Network (IHHN), Karachi, and comprised data from January 2015 to December 2021 of patients regardless of age and gender who had renal dysfunction with serum creatinine level >1.5mg/dl and underwent CA. Data of cases not having complete information was excluded.

Exemption was obtained from the ethics review board of IHHN, which is one of the largest tertiary care facilities in Karachi. It is privately run, but offers services free of charge. It has cardiology as well as nephrology services, and caters to almost 5-6 coronary catheterisations per day on average.

History, clinical examination, and laboratory investigations were retrieved from the Health Management Information System (HMIS). All the patients had been assessed by a nephrologist. The patients were hydrated with normal saline at least 12 hours (if needed, and not in heart failure or pulmonary oedema cases) before and after the sample, at least at a rate of 100-150ml/hour. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs), diuretics and metformin were stopped 24 hours before the procedure as an institutional practice. The amount of contrast used during the procedure mostly did not exceed 150ml, and, if there was a need to repeat the procedure, it was done after at least one week. The patients after discharge were followed up in

the nephrology clinic.

Data was analysed using SPSS 26. Frequencies with percentages were calculated for categorical data, while mean $\pm$ standard deviation (SD) was obtained for continuous data. Normality of continuous data was checked with histogram, box and whisker plot, and Kolmogorov-Smirnov test. Since creatinine-related data was non-normally distributed, hence non-parametric tests were used. Mann-Whitney U test was applied to compare mean differences between two independent groups, while Wilcoxon signed-rank test was used to assess changes within the same patient. The mean difference among three or more groups was evaluated with Kruskal-Wallis test. Logistic regression analysis was performed to quantify the impact of risk factors on the development of CIN, with odds ratios (ORs) and 95% confidence intervals (CIs) calculated. Univariate analysis was conducted initially, followed by multivariate modelling to adjust for potential confounders and to minimise bias. $P \leq 0.05$ was considered statistically significant.

Results

Of the 5,376 patients, 3,630(67.5%) were male and 1,746(32.5%) were female. The overall mean age was

Table-1: Demographic data of the patients (n= 5,376).

Variables		n (%)
Gender	Male	3630(67.5)
	Female	1746(32.5)
Mean Age (years)		55.1 $\pm$ 10.3
Married		5359(99.7)
Education	Uneducated	2123(39.5)
	Primary	497(9.2)
	Secondary	1667(31)
	High school-Graduate	931(17.3)
	Post-graduate	158(2.9)
Occupation	No job	899(16.7)
	Field job	2349(43.7)
	Office job	539(10)
	Retired	1320(24.6)
	House-wife	193(3.6)
	Health-professionals	76(1.4)
Smoker		813(15.1)
Comorbid	Diabetes mellitus	2084(38.8)
	Hypertension	3405(63.3)
	Ischaemic heart disease	1254(23.3)
	Congestive cardiac failure	39(0.7)
	Cerebrovascular accident	107(2)
Residence	Karachi	4257(79.2)
	Rural Sindh	908(16.9)
	Punjab	36(0.7)
	Baluchistan	117(2.2)
	KP/Azad Kashmir	58(1.1)
On aspirin		1530(28.5)

KP: Khyber Pakhtunkhwa.

Table-2: Comparison of mean creatinine values at different stages.

Comparison	Mean $\pm$ SD	p-value
Creatinine at first presentation	1.1 $\pm$ 0.64	< 0.001
Creatinine after the angiography	1.17 $\pm$ 0.6	
Creatinine at first presentation	1.1 $\pm$ 0.64	< 0.001
Creatinine at the last follow-up	1.2 $\pm$ 0.9	
Creatinine after the angiography	1.17 $\pm$ 0.6	< 0.001
Creatinine at the last follow-up	1.2 $\pm$ 0.9	

The Wilcoxon signed-rank test was applied to obtain the p-value. SD: Standard deviation.

Table-3: Comparison of change in creatinine levels stratified by gender and comorbid conditions (The differences are delta values).

Parameters	Mean $\pm$ SD	95% CI Lower-Upper	p-value
Gender			
Male	0.11 $\pm$ 0.56	0.09-0.13	<0.001
Female	0.18 $\pm$ 0.69	0.15-0.22	
Diabetes mellitus			
Yes	0.21 $\pm$ 0.75	0.17-0.24	<0.001
No	0.09 $\pm$ 0.49	0.07-0.1	
Hypertension			
Yes	0.16 $\pm$ 0.67	0.13-0.18	<0.001
No	0.09 $\pm$ 0.48	0.07-0.11	
Congestive cardiac failure			
Yes	0.7 $\pm$ 1.21	0.31-1.1	<0.001
No	0.13 $\pm$ 0.6	0.11-0.14	
Ischaemic heart disease			
Yes	0.21 $\pm$ 0.78	0.17-0.26	<0.001
No	0.11 $\pm$ 0.54	0.09-0.12	
Cerebrovascular accident			
Yes	0.45 $\pm$ 1.2	0.22-0.68	<0.001
No	0.13 $\pm$ 0.59	0.11-0.14	

The Mann-Whitney U test was applied for statistical significance; SD: Standard deviation, CI: Confidence interval.

Table-4: Effect of demographic and clinical parameters on the risk of developing contrast-induced nephropathy (CIN).

Risk factors for development of CIN n (%)			Univariate binary logistic regression				Multivariate binary logistic regression			
		CIN=969(18%)	Odds Ratio	95% CI		p-value	Odds Ratio	95% CI		p-value
				Lower	Upper			Lower	Upper	
Age (years)	< 36	15/165=(9.1)			1				1	
	36-50	194/1637=(11.9)	1.34	0.77	2.3	0.293	0.893	0.51	1.6	0.696
	51-60	353/1933=(18.3)	2.23	1.3	3.8	0.004	1.1	0.63	1.9	0.73
	61-70	307/1332=(23)	3	1.7	5.2	<0.001	1.04	0.59	1.9	0.883
	>70	100/309=(32.4)	4.8	2.7	8.6	<0.001	1.3	0.7	2.4	0.409
Diabetes mellitus	504(24.2)	1.95	1.7	2.2	<0.001	1.6	1.4	1.9	<0.001	
Hypertension	701(13.6)	1.65	1.4	1.9	<0.001	1.34	1.1	1.6	0.001	
Congestive cardiac failure	21(53.8)	5.4	2.9	10.2	<0.001	3.2	1.6	6.3	0.001	
Ischaemic heart disease	337(15.3)	2.03	1.75	2.4	<0.001	1.64	1.4	1.9	<0.001	
Cerebrovascular accident	39(36.4)	2.7	1.8	4	<0.001	1.8	1.2	2.8	0.009	
Glomerular filtration rate (GFR)	> 90	166/2069=(8)	1		1					
	60-89.99	385/2145=(17.4)	2.5	2.1	3.04	<0.001	2.4	1.97	3	<0.001
	59.99-30	354/1009=(35.1)	6.2	5.1	7.6	<0.001	5.3	4.2	6.6	<0.001
	29.99-15	48/110=(43.6)	8.9	5.9	13.4	<0.001	6.5	4.2	10	<0.001
	<15	16/43=(37.2)	6.8	3.8	12.9	<0.001	4.4	2.2	8.5	<0.001

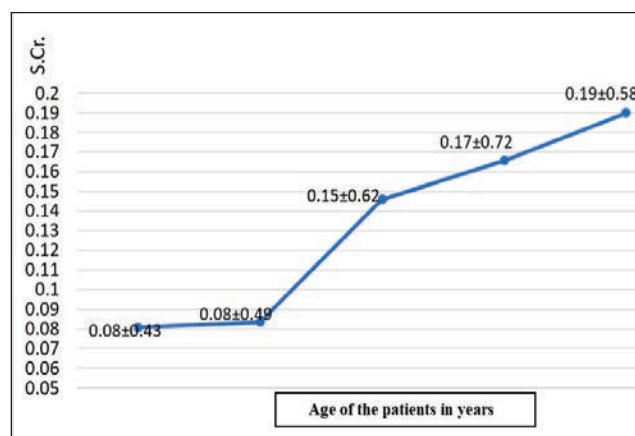
CI: Confidence interval, Age <36 is the reference category, GFR >90 is the reference category.

55.1 $\pm$ 10.3 (range: 18-94 years). Hypertension (HTN) was the most frequent comorbid 3,405(63.3%), followed by diabetes mellitus (DM) 2,084(38.8%). The majority of the patients were residents of Karachi 4,257(79.2%) (Table 1).

Pairwise comparison of the three creatinine readings revealed significant differences between each pair ($p<0.001$). The maximum increase in creatinine levels was observed at the final follow-up (Table 2).

The female gender was more prone to the creatinine rise post-CA compared to males, and among patients who had DM, HTN, congestive cardiac failure (CCF), ischemic heart disease (IHD) and cerebrovascular accident (CVA) compared to the patients who were not suffering from these comorbid conditions (Table 3).

The maximum difference in pre-CA and post-CA was found

**Figure-1** Mean differences between 1st and last creatinine(mg/dl) of the patients stratified by the age.

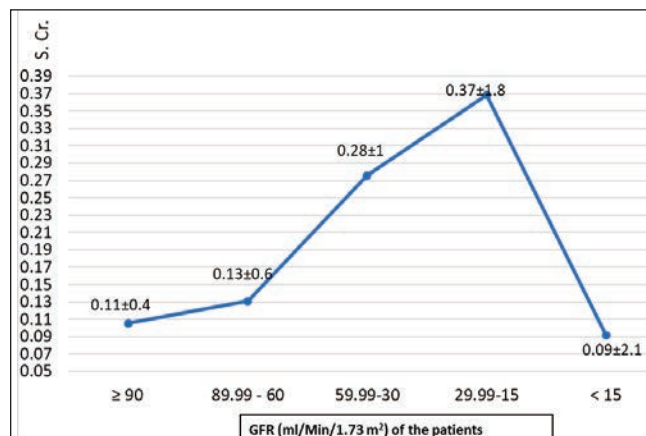


Figure-2 Mean differences between 1st and last creatinine of the patients stratified by the GFR.

among the elderly compared to the young patients ($p < 0.001$) (Figure 1).

The highest rise in mean creatinine level was noted in patients with glomerular filtration rate (GFR) 15-29.9 mL/min/1.73 m² compared to patients with other GFRs ($p < 0.001$) (Figure 2).

CIN occurred in 969 (18%) patients, with a rising trend in incidence with increasing age (Table 4).

Univariate analysis highlighted that age, comorbidities and GFR significantly influenced the development of CIN when evaluated individually ($p \leq 0.05$). The patients aged 51-60 years had 2.23 times higher risk of developing CIN than patients aged < 36 years (OR: 2.23, 95%CI: 1.3-3.8). Similarly, those aged > 70 years had 4.8 times increased risk of CIN compared to < 36 years (OR: 4.8, 95% CI: 2.7-8.6). Moreover, the risk of CIN was significantly higher in patients with CCF, and, as GFR decreased, the odds ratio for developing CIN increased correspondingly (Table 4).

Upon adjusting for confounding factors in the multivariate analysis, age became non-significant ($p > 0.05$), whereas other variables continued to show a significant association with CIN (Table 4).

Discussion

The current study, to our knowledge, is the largest study comprising patients with normal kidney function as well as patients with low GFR who underwent CA in the Pakistani population. The study sample had a good age and gender representation and diversity of socioeconomic groups, manifested by education and job status (Table 1). Although the majority of the patients belonged to Karachi city and Sindh province, there were also a good number of patients from across Pakistan. The prevalence of an acute rise in serum creatinine was found to be 822 (15.29%), while 4,554 (87.71%) patients showed no change in serum

creatinine post-procedure. There is a disparity in reporting of the incidence and prevalence of CIN, possibly due to differences in the population parameters, sample size, comorbid conditions, and route of contrast injection (IV vs intra-arterial). In a systematic review and meta-analysis of 259 studies regarding CIN, the incidence of AKI after CA was 9.06% (95% CI: 8.53-9.58%) which was derived from 120 studies, while, on the other hand, the stratified summary incidence proportion of CIN after intra-arterial contrast administration was almost similar (9.60%) (95% CI: 9.0-10.2%) that was derived from 106 studies.¹³ In another prospective consecutive cohort of 633 patients, the incidence of CIN was 11%¹⁴ and a recently published prospective study from a single centre from Pakistan reported an incidence of 22.3% among 112 patients.¹⁵

The rise of serum creatinine was significant and persistent among those who showed impairment in renal function, and it was almost at the same level till the last follow-up in the current study. The clinical impact of change in small rise in serum creatinine in the early stages of CKD is significant.¹⁶ A decline in estimated GFR (eGFR) over time is associated with a higher risk for all-cause mortality and cardiovascular disease, independent of initial eGFR and other known risk factors at baseline.¹⁷

Although there was a male preponderance in the current study, females showed more severe impairment of serum creatinine compared to the males. This female predilection was also observed in an animal model study by Eduardo et al. who compared male and female mice with contrast exposure vs no exposure, and documented that there was a more intense presence of micro-vacuoles in proximal tubules in the mice exposed to the contrast than in the control groups. Such proximal tubular vacuolation was more intensive in the female mice ($p = 0.001$).¹⁸ Similarly, in an interesting analysis of 285 patients (730 females vs 1,851 males), Barberi et al. concluded that the incidence of CIN was 12.6% and it was significantly higher among females (15.6% vs 11.4%, OR: 1.42, 95%CI: 1.11-1.82, $p = 0.004$), but, surprisingly, the outcome was not established during multivariate analysis after correction for all baseline confounders (adjusted OR: 1.14, 95%CI: 0.81-1.60, $p = 0.45$).¹⁹

The other factors that predisposed the patients to more severe outcomes were found to be HTN, DM, CVA and CAD in the current study. These factors have been reported by several studies as well-known causes for enhancing CIN risk factors.^{20,21} Age and higher serum creatinine were both found to be significantly associated with increased CIN in the current population, which was in line with literature.^{22,23} While several recent studies have supported the predictive value of traditional risk factors for CIN, a

recent systematic review found these associations to be less significant than previously thought, highlighting the ongoing variability and complexity in risk prediction.^{23,24}

The current study has various limitations, including its retrospective design, absence of a control group, and limited follow-up data. The strength of the study is its large population, data storage in the electronic medical record, a consistent institutional protocol and policy regarding CA for cardiologists, as well as preventive measures for CIN implemented and followed by nephrologists, along with an integrated follow-up in consultant clinics.

Conclusion

CIN was found to be an important cause of acute renal dysfunction, and female gender, advanced age, low GFR at the time of the procedure, and comorbid conditions, like DM, HTN and CAD, were associated with CIN.

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Author Contribution:

FS: Literature search, design, concept, data collection and final draft.

MFA & JC: Literature search, data collection and final draft.

SI: Literature search, design, concept, data analysis and final draft.

SM: Literature search and final draft.