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ORIGINAL ARTICLE

Nephrotoxic Effects of Contrast Medium in Patients Undergoing Coronary Angiography and Percutaneous Coronary Intervention

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ABSTRACT

Background: *Contrast-induced nephropathy (CIN) is a significant complication of coronary angiography and percutaneous coronary intervention, leading to increased morbidity, prolonged hospital stay, and higher healthcare costs.* ***Objective:*** *The aim of the study was to evaluate the incidence, risk factors, and outcomes of CIN in patients undergoing these procedures.* ***Method & Materials:*** *This prospective, non-randomized clinical study enrolled 100 adults undergoing coronary angiography with or without PTCA at the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh (May 2007–April 2008). Non-ionic low-osmolar contrast (Iopamidol) was used, and serum creatinine was measured at baseline and days 1–3 and 7. The primary outcome was peak creatinine rise by day 3; secondary outcomes were rises ≥0.5 and ≥1.0 mg/dL. Statistical analyses used Student's t-test and Chi-square test with p<0.05.* ***Results:*** *Among 100 patients undergoing coronary procedures, those with pre-existing renal impairment (n=50) versus normal function (n=50) had higher baseline creatinine (1.8±0.3 vs 1.1±0.2 mg/dL), more diabetes (70% vs 20%), greater creatinine rise at day 3 (0.39±0.24 vs 0.20±0.15 mg/dL), and higher CIN incidence (18% vs 2%) (all p<0.01). The 10 CIN cases occurred in older patients (67.4±4.9 vs 49.5±9.1 years) receiving higher contrast volumes (126.0±21.3 vs 104.3±35.8 ml). Age ≥70, contrast ≥100 ml, diabetes, renal impairment, and their combination were significant predictors (OR 1.24–2.89, all p<0.05). Most (96%) recovered within 3 weeks.* ***Conclusion:*** *Diabetic patients with pre-existing renal impairment, advanced age, and higher contrast volume are at the highest risk for contrast-induced nephropathy, which is usually reversible within two weeks.*

Keywords: *Nephrotoxicity, Contrast Medium, Coronary Intervention*

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INTRODUCTION

Cardiovascular diseases continue to be a major contributor to illness and death globally, with coronary artery disease (CAD) posing a particularly significant challenge for healthcare systems worldwide. Among patients with CAD, the prevalence of chronic kidney disease (CKD) is notably high, ranging between 23% and 46% according to various studies [1,2]. Individuals with CKD are at an elevated risk for cardiovascular complications compared with the general population [3,4], and cardiovascular disease accounts for over half of deaths in patients with end-stage renal disease (ESRD) [5]. The extensive adoption of coronary angiography and percutaneous coronary interventions (PCI) has markedly enhanced outcomes for patients with acute coronary syndrome (ACS) by enabling rapid revascularization and minimizing myocardial injury [6]. Since coronary angioplasty was introduced more than thirty years ago, the number of PCI procedures has steadily increased, with approximately 1.4 million catheterization procedures performed annually in the United States [7].

As the frequency of these interventions has risen, patients are increasingly exposed to radiographic contrast agents. The occurrence of renal insufficiency following coronary procedures has become more common due to the use of contrast in more complex interventional procedures [8]. Contrast media are routinely used in both diagnostic coronary angiography and PCI, and intravenous administration of iodinated contrast is a well-recognized trigger for contrast-induced nephropathy (CIN), also referred to as contrast-induced acute kidney injury (AKI) [9,10], Despite their life-saving potential, these procedures carry inherent risks, and CIN represents a significant complication, especially in patients who undergo multiple interventions [11].

Contrast-induced nephropathy is characterized by a deterioration of renal function, usually defined as a 25% relative increase in serum creatinine from baseline or an absolute rise of 0.5 mg/dL within 48–72 hours following contrast exposure [12,13]. CIN is an important cause of acute kidney injury in hospitalized patients and ranks as the third most common etiology of hospital-acquired kidney injury in those undergoing interventional procedures [14]. Most affected patients experience transient impairment, with renal function typically returning to baseline within 7–14 days [15,16]. Nevertheless, CIN is associated with higher rates of morbidity,

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longer hospitalizations, increased healthcare costs, and in severe cases, elevated mortality [17].

Several risk factors for CIN have been consistently identified. Pre-existing renal dysfunction and diabetes mellitus are considered the most important predictors [18]. Other contributing factors include advanced age, dehydration, anemia, severe congestive heart failure (NYHA class III–IV), exposure to nephrotoxic drugs, and high volumes of contrast medium. Patients undergoing PCI for myocardial infarction are particularly susceptible due to additional challenges such as hemodynamic instability, hypotension, and urgent procedural conditions [19-21]. While the overall incidence of CIN in the general population is relatively low (0.6–2.3%), it is significantly higher in patients with CKD, diabetes, or other comorbid conditions, highlighting the importance of identifying high-risk populations and implementing preventive strategies [22].

Despite extensive research on contrast-induced nephropathy, there remains considerable variability in reported incidence rates and risk factor profiles, particularly among patients undergoing coronary angiography and PCI in different clinical settings. Most studies have focused on high-income countries, with limited data available from developing countries, where patient demographics, comorbidities, and procedural practices may differ. Furthermore, while several preventive strategies have been proposed, there is still uncertainty regarding which patients are most at risk and how contrast volume, pre-existing renal function, and diabetes interact to influence outcomes. These gaps underscore the need for region-specific data to guide risk assessment and management. The purpose of the study is to evaluate the incidence, risk factors, and outcomes of contrast-induced nephropathy in patients undergoing coronary angiography and percutaneous coronary intervention.

OBJECTIVE

To evaluate the incidence, risk factors, and outcomes of contrast-induced nephropathy in patients undergoing coronary angiography and percutaneous coronary intervention.

METHODS & MATERIALS

This prospective, non-randomized clinical study was conducted at the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh, from May 2007 to April 2008. A total of 100 patients who underwent coronary angiography with or without percutaneous transluminal coronary angioplasty (PTCA) were included, selected based on predefined inclusion and exclusion criteria to evaluate the nephrotoxic effects of contrast medium.

Inclusion Criteria

- Patients aged >18 years.
- Undergoing coronary angiography with or without PTCA.
- Patients with normal or impaired renal function.

Exclusion Criteria

- History of intravascular administration of iodinated contrast medium within the previous 7 days.
- History of treatment with NSAIDs or Metformin within the previous 48 hours.
- History of intake of other nephrotoxic drugs within the previous 7 days.
- Patients receiving ACE inhibitor therapy within one week of study entry.
- End-stage renal disease requiring dialysis.
- Renal transplant recipients.
- Severe concomitant diseases (e.g., chronic heart failure, chronic liver disease).
- Pregnant women.
- Lactating mothers.

The study protocol was approved by the institutional review board, and written informed consent was obtained from all participants with strict maintenance of confidentiality. All patients underwent baseline evaluation including detailed history, physical examination, and relevant investigations; demographic variables, clinical parameters, and cardiovascular risk factors were recorded. Baseline investigations included ECG, serum creatinine, random blood sugar, echocardiography, viral markers, and coagulation profiles, with lipid profile and serum electrolytes assessed in selected patients. All procedures were performed using non-ionic, low-osmolar contrast medium (Iopamidol), with individually determined contrast volumes. Procedural data including angiographic findings, number of diseased vessels, performance of PTCA, intravenous hydration volume, contrast volume, and total iodine dose were documented. Serum creatinine was measured at baseline and on days 1, 2, 3, and 7 after contrast exposure, with extended follow-up up to the third week in selected cases, and patients were monitored for adverse events throughout follow-up. The primary outcome was the peak increase in serum creatinine between day 0 and day 3, while secondary outcomes included the number of patients with creatinine rises ≥0.5 mg/dL and ≥1.0 mg/dL. Continuous variables were expressed as mean ± SD and compared using Student's t-test, categorical variables as percentages using the Chi-square test, and a p-value <0.05 was considered statistically significant.

RESULTS

Table I summarizes the baseline demographic and clinical characteristics of the study population. The mean age, body weight, and BMI were comparable between Group I and Group II. Baseline serum creatinine was significantly higher in Group II than in Group I (1.8 ± 0.3 vs. 1.1 ± 0.2 mg/dL; p = 0.001). The mean contrast volume administered differed significantly between the groups (101.9 ± 13.8 ml vs. 111.1 ± 26.7 ml; p = 0.037). Diabetes mellitus was more prevalent in Group II (70% vs. 20%; p = 0.001), and intravenous hydration during the procedure was administered more frequently in Group II (80% vs. 28%; p = 0.001). Other baseline and procedural characteristics were similar between the two groups.

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Table – I: Demographic and Baseline Characteristics of the Study Patients (*n*=100)

Characteristics	Group I (n=50)	Group II (n=50)	p-value
Age (years)	49.8 ± 9.4	52.8 ± 11.0	[a]0.149[NS]
Sex			
Male	45 (90.0)	40 (80.0)	[b]0.161[NS]
Female	5 (10.0)	10 (20.0)	
Weight (kg)	63.2 ± 8.4	61.7 ± 8.5	[a]0.385[NS]
Body mass index (kg/m^2)	24.3 ± 3.6	24.2 ± 2.7	[a]0.847[NS]
Diabetes Mellitus	10 (20.0)	35 (70.0)	[b]0.001[s]
Base line serum creatinine (mg/dl)	1.1 ± 0.2	1.8 ± 0.3	[a]0.001[s]
Hydration given intravenously during procedure	14 (28.0)	40 (80.0)	[b]0.001[s]
Amount of hydration (ml)	750.0 ± 259.4	887.5 ± 239.9	[a]0.076[NS]
Total volume of contrast medium (ml)	111.1 ± 26.7	101.9 ± 13.8	[a]0.037[s]
Total dose of contrast medium (gm of Iodine)	40.7 ± 14.4	38.0 ± 13.8	[a]0.353[NS]
Coronary angiography performed	50 (100.0)	50 (100.0)	-
No. of diseased vessels identified			
1	21 (42.0)	19 (38.0)	[b]0.650[NS]
>1	18 (36.0)	20 (40.0)	
PTCA Performed	22 (44.0)	18 (36.0)	[b]0.364[NS]

[a] Values are expressed as mean ± SD; comparison by independent t-test.
[b] Values are expressed as number (%); comparison by chi-square test.
NS = Not significant (p > 0.05), s = Significant (p ≤ 0.05)

Table II shows the distribution of patients according to pre-procedural selected investigations. All patients in Group I had baseline serum creatinine <1.5 mg/dL, whereas in Group II, 84% had values between 1.5–2.0 mg/dL and 16% had >2.0 mg/dL, showing a statistically significant difference (p = 0.001). Elevated random blood sugar (≥11.1 mmol/L) was more frequent in Group II compared with Group I (70% vs. 20%), while normal values were more common in Group I (80% vs. 30%); this difference was also statistically significant (p = 0.001).

Table – II: Distribution of Patients by Pre-procedure Selected Investigations (*n* = 100)

Investigations	Group I (n=50)		Group II (n=50)		Total (n=100)		p-value
	No	%	No	%	No	%	
Baseline Serum Creatinine (mg/dL)							
< 1.5	50	100.0	0	0.0	50	50.0	0.001[s]
1.5–2.0	0	0.0	42	84.0	42	42.0	
> 2.0	0	0.0	8	16.0	8	8.0	
Random blood sugar (mmol/L)							
< 11.1	40	80.0	15	30.0	55	55.0	0.001[s]
≥ 11.1	10	20.0	35	70.0	45	45.0	

Values are expressed as number (%); comparison by chi-square test.
s = Statistically significant (p ≤ 0.05)

Table III compares the peak rise in serum creatinine levels from baseline to day 3 following contrast exposure between the two study groups. Group II showed a significantly greater mean increase in serum creatinine compared with Group I (0.39 ± 0.24 mg/dL vs 0.20 ± 0.15 mg/dL), with higher median and wider range values. This difference was statistically significant (p = 0.001).

Table – III: Peak Increase in Serum Creatinine from Baseline to Day 3 after Contrast Administration

Group	No. of patients	Mean ± SD (95% CI) (mg/dL)	Median (mg/dL)	Range (Min-Max) (mg/dL)	p value
Group I	50	0.20 ± 0.15 (0.16 – 0.24)	0.2	(0.0 – 0.6)	0.001s
Group II	50	0.39 ± 0.24 (0.33 – 0.45)	0.4	(0.1 – 1.1)	

Values are expressed as mean ± SD (95% CI), median, and range; comparison by independent t-test.
s = Statistically significant (p ≤ 0.05)

Table IV shows the incidence of contrast-induced nephropathy (CIN) among patient subgroups. Overall, 10% of patients developed CIN, with higher rates in Group II compared with Group I (18% vs 2%, p = 0.008). Subgroup analysis revealed that patients with pre-existing renal impairment or diabetes had increased CIN risk, with the highest incidence in those with both conditions (20%).

Table – IV: Distribution of Patients Developing CIN by Subgroups Following Coronary Angiography and PCI (*n*=100)

Sub group	Group I			Group II			p value
	No of Pts.	CIN No	CIN %	No of Pts.	CIN No	CIN %	
All patients	50	1	2.0	50	9	18.0	0.008[s]
Patients with preexisting renal impairment	0	0	0.0	15	2	13.3	-
Patients with DM	10	1	10.0	35	7	20.0	0.046[s]
Patients with pre-existing renal impairment with DM	0	0	0.0	35	7	20.0	-

CIN = Contrast-Induced Nephropathy; DM = Diabetes Mellitus.
s = Statistically significant (p ≤ 0.05); "–" indicates not applicable

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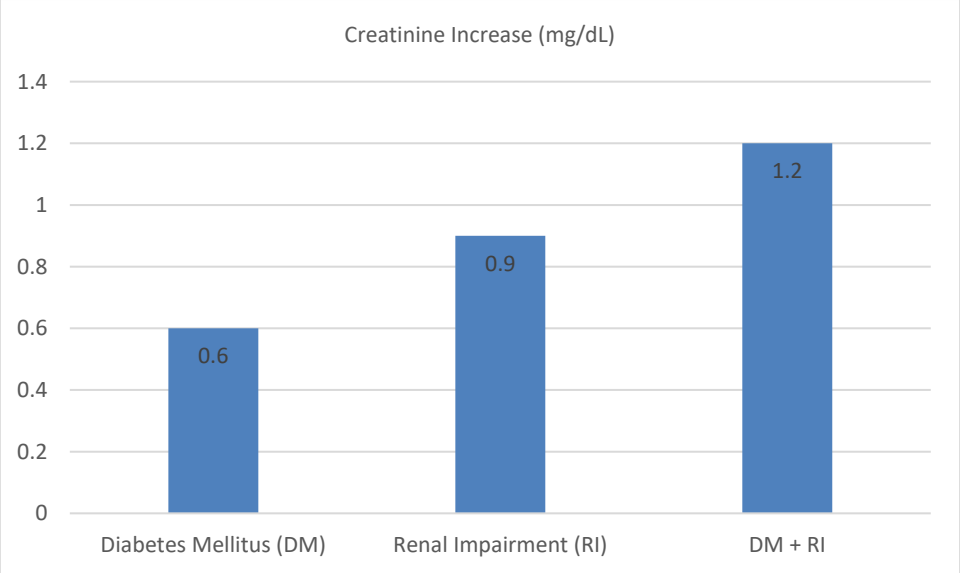

Figure – 1: Maximal Absolute Increase in Serum Creatinine by Patient Subgroup After Contrast Administration

Figure 1 illustrates the maximal rise in serum creatinine from baseline to day 3. The greatest increase occurred in patients with both diabetes and renal impairment (≥ 1.1 mg/dL), the lowest in diabetics without renal impairment (≤ 0.6 mg/dL), and intermediate in patients with renal impairment alone (0.6–0.9 mg/dL).

Table V compares patients who developed CIN (n=10) with those who did not (n=90). Patients with CIN were older (67.4 ± 4.9 vs. 49.5 ± 9.1 years), received a higher contrast volume (126.0 ± 21.3 vs. 104.3 ± 35.8 ml), had higher baseline serum creatinine (1.85 ± 0.31 vs. 1.37 ± 0.44 mg/dL), and elevated random blood sugar (16.9 ± 6.8 vs. 10.3 ± 6 mmol/L); all differences were statistically significant (p ≤ 0.006). Use of ACE inhibitors was not significantly different (p = 0.193).

Table – V: Comparison of Patients with and Without Contrast-Induced Nephropathy (CIN) (*n*=100)

Variables	Patients without CIN (n=90)	Patients with CIN (n=10)	P Value
	Mean ± SD	Mean ± SD	
Age (years)	49.5±9.1	67.4±4.9	[a]0.001[s]
Contrast volume (ml)	104.3±35.8	126.0±21.3	[a]0.006[s]
Baseline serum creatinine (mg/dL)	1.37±0.44	1.85±0.31	[a]0.001[s]
Random blood sugar (mmol/l)	10.3±6	16.9±6.8	[a]0.001[s]
ACEI used	45 (50.0)	7 (70.0)	[b]0.193[NS]

CIN = Contrast-Induced Nephropathy; ACEI = Angiotensin-Converting Enzyme Inhibitor.
s = Statistically significant (p ≤ 0.05); NS = Not significant.
a = p-value calculated using Student's t-test; b = p-value calculated using Chi-square test.

Table VI demonstrates that higher contrast volumes were associated with greater increases in serum creatinine and higher incidence of CIN in both groups. In Group I, only 1 patient developed CIN with >150 ml contrast, whereas in Group II, CIN incidence rose progressively with contrast dose (2/24 for 50–99 ml, 3/18 for 100–149 ml, 4/8 for >150 ml), all statistically significant (p = 0.001).

Table – VI: Correlation between the Contrast Volume and Change in Serum Creatinine from Baseline to Day 3

Amount of dye (ml)	Group I (n=50)			Group II (n=50)			p Value
	No. of pts receiving contrast	No. of pts showing CIN	Increase in serum creatinine (mg/dl) Mean ± SD (95% CI)	No. of pts receiving contrast	No. of pts showing CIN	Increase in serum creatinine (mg/dl) Mean ± SD (95% CI)	
50–99	17	0	0.09±0.08 (0.05–0.13)	24	2	0.27±0.20 (0.19–0.35)	0.001[s]
100–149	19	0	0.22±0.13 (0.16–0.28)	18	3	0.44±0.22 (0.34–0.54)	0.001[s]
>150	14	1	0.29±0.17 (0.21–0.37)	8	4	0.60±0.23 (0.44–0.76)	0.001[s]

CIN = Contrast-Induced Nephropathy; SD = Standard Deviation; CI = Confidence Interval; s = Statistically significant (p ≤ 0.05).
p-value calculated using Chi-square test for CIN incidence and Student's t-test for mean increase in serum creatinine.

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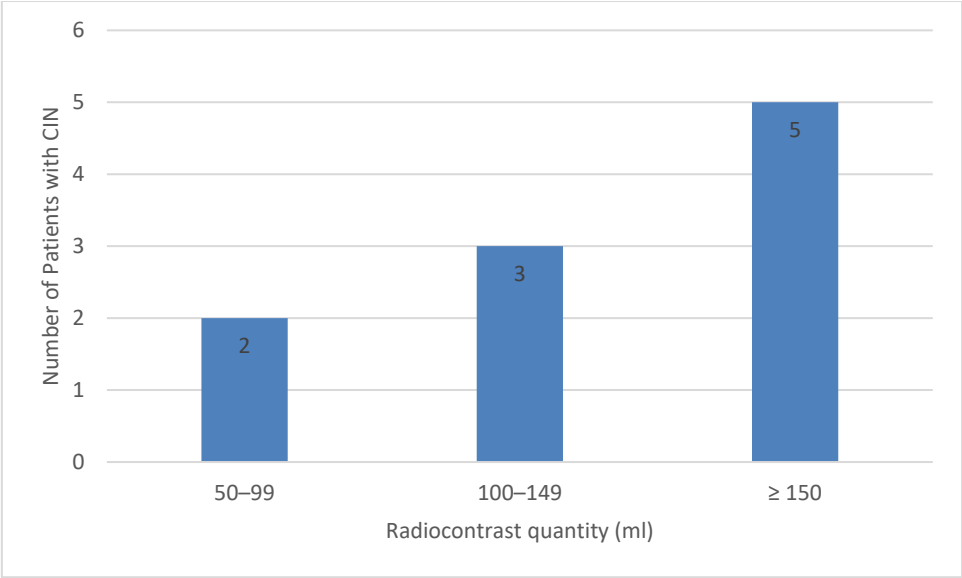

Figure – 2: Relation of Contrast Volume to Development of CIN

Figure 2 shows the incidence of contrast-induced nephropathy (CIN) by contrast volume. Among patients receiving 50–99 ml, 2 developed CIN; among 100–149 ml, 3 developed CIN; and ≥150 ml, 5 developed CIN. The incidence of CIN increased with contrast volume, and the association was statistically significant (p = 0.001).

Table VII shows that age ≥ 70 years, contrast volume ≥ 100 ml, pre-existing renal impairment, diabetes, and the combination of diabetes with renal impairment were significantly associated with CIN, with odds ratios ranging from 1.24 to 2.89, indicating these are strong predictors of nephrotoxicity (p ≤ 0.05).

Table – VII: Logistic Regression Analysis for Risk Factors of Contrast-Induced Nephropathy (CIN)

Variables	Patients (%)	No. of Patients With CIN	Incidence of CIN (%)	OR	95% CI	p Value
Age (≥ 70 years)	7	3	42.8	2.68	2.09–3.44	0.001[s]
Contrast volume (≥ 100ml)	59	8	13.5	1.24	1.01–1.54	0.045[s]
Pre-existing renal impairment with DM	35	7	20	2.89	2.32–3.59	0.001[s]
Preexisting renal impairment	15	2	13	1.9	1.59–2.27	0.001[s]
Diabetes Mellitus	10	1	10	1.73	1.48–2.02	0.001[s]

CIN = Contrast-Induced Nephropathy; DM = Diabetes Mellitus; OR = Odds Ratio; CI = Confidence Interval; s = Statistically significant (p ≤ 0.05). Logistic regression was used to identify independent risk factors associated with CIN.

Table VIII shows serum creatinine trends after contrast administration. In Group I, 62% of patients were tested in the first week, with only 3.2% showing a rise; all returned to baseline by the second week. In Group II, 88% were tested in the first week, with 11.3% showing an increase; by the third week, 96% had returned to baseline, while 4% had persistently elevated levels.

Table – VIII: Outcome of Study Patients Based on Serum Creatinine Recovery (*n*=100)

Group	Weeks after contrast administration	Serum creatinine done		Increase in serum creatinine concentration (mg/dl)		p value
		No.	%	No.	%	
Group I (n=50)	1st week	31	62	1	3.2	0.047[s]
	2nd week	1	2	0	0	
Group II (n=50)	1st week	44	88	5	11.3	0.021[s]
	2nd week	5	10	2	4.5	
	3rd week	2	4	2	4.5	

Serum creatinine recovery was monitored weekly after contrast administration. s indicates statistically significant (p ≤ 0.05). Group I = patients with normal baseline renal function; Group II = patients with pre-existing renal impairment.

DISCUSSION

This prospective study evaluated 100 patients undergoing coronary angiography and percutaneous coronary intervention (PCI) over one year to determine the incidence, predictors, and outcomes of contrast-induced nephropathy (CIN). The mean age of the study population was 51.3 ± 10.3 years, with patients in Group II being significantly older than those in Group I (52.8 ± 11.0 vs. 49.8 ± 9.4 years; p = 0.002). The majority of patients belonged to the 40–49-year age group. This distribution aligns with Chong et al., who reported a median age of approximately 59.5 years in PCI patients, highlighting that CIN predominantly affects middle-aged and older populations [23]. Male predominance was evident, with a male-to-female ratio of 5.7:1, consistent with previous

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regional reports [24] and international data, including Chong et al., where 78.7% of PCI patients were male [23]. The age range of the present cohort also parallels earlier Bangladeshi studies on ischemic heart disease [25].

Anthropometric parameters were similar between the groups, with no significant differences in mean body weight or BMI. However, diabetes mellitus was significantly more prevalent in Group II (70%) than in Group I (20%) (p = 0.001), reinforcing diabetes as a major risk factor for CIN. This observation closely mirrors Afzal et al., who reported diabetes in 66.7% of patients developing CIN after PCI [26]. Baseline renal function also differed significantly, with mean serum creatinine levels higher in Group II (1.8 ± 0.3 mg/dL) compared with Group I (1.1 ± 0.2 mg/dL; p = 0.001), underscoring the contribution of pre-existing renal impairment to CIN susceptibility.

Preventive measures, particularly intravenous hydration, were administered more frequently in Group II (80% vs. 28%; p = 0.001), reflecting standard risk-based protocols. Although the mean hydration volume was higher in Group II, this difference was not statistically significant. Interestingly, the mean contrast volume was slightly higher in Group I than Group II (111.1 ± 26.7 ml vs. 101.9 ± 13.8 ml; p = 0.03). Despite remaining within calculated safe limits, contrast exposure remained a significant determinant of CIN risk, consistent with Afzal et al., who showed increased CIN incidence with contrast volumes >200 ml [26]. These findings suggest that even moderate contrast doses can contribute to CIN risk, especially in high-risk patients.

Regarding coronary anatomy and intervention, the distribution of single- and multi-vessel disease was comparable between groups, and PTCA was performed in 44% of Group I and 36% of Group II patients. Overall, these demographic and procedural characteristics closely resemble those reported in regional and international PCI cohorts, reinforcing the generalizability of our findings.

Pre-procedural investigations highlighted the clustering of CIN risk factors. Diabetes mellitus, defined as random blood sugar ≥11.1 mmol/L, was significantly more common in Group II (p = 0.001). All Group I patients had baseline serum creatinine <1.5 mg/dL, whereas all Group II patients had serum creatinine ≥1.5 mg/dL, with 84% between 1.5–2.0 mg/dL and 16% >2.0 mg/dL (p = 0.001). These findings emphasize the combined impact of hyperglycemia and baseline renal dysfunction. Kumar et al. similarly documented a 13.1% CIN incidence among STEMI patients undergoing PCI, with diabetes and elevated baseline creatinine significantly predicting CIN, particularly in patients receiving higher contrast volumes [27]. This concordance underscores the importance of careful pre-procedural risk stratification.

The mean peak rise in serum creatinine within three days of contrast exposure was significantly higher in Group II than Group I (0.39 vs. 0.20 mg/dL; p = 0.001), indicating that elevated baseline serum creatinine predicts post-procedural renal deterioration. Applying the widely accepted definition of CIN (increase ≥0.5 mg/dL), the incidence was 18% in Group II versus 2% in Group I (p = 0.008). These findings align with Marenzi et al., who reported a CIN incidence of 19% among 208 AMI patients undergoing primary PCI, with greater post-procedure creatinine rises in those with reduced baseline renal clearance (<60 ml/min) [21]. Marenzi et al. also demonstrated that serum creatinine continues to rise during the first 48–72 hours after contrast exposure in high-risk patients, mirroring the temporal pattern observed in our cohort [21].

Subgroup analysis revealed that CIN incidence was 10% among diabetic patients with normal renal function, 13.3% in patients with pre-existing renal impairment without diabetes, and 20% in patients with both diabetes and renal impairment. These findings are consistent with previous studies: Afzal et al. reported a CIN incidence of 15% in 120 patients undergoing primary PCI, with diabetes present in 66.7% of CIN cases and pre-existing renal impairment (eGFR <60 mL/min) observed in 55.6% of cases, highlighting diabetes and baseline renal dysfunction as key risk factors [26]. Similarly, Kumar et al. found CIN occurred in 13.1% of 282 STEMI patients treated with primary PCI, with diabetes significantly associated with CIN risk, supporting the observation that diabetic patients have a higher incidence of CIN [27]. Overall, 10 patients in our study developed CIN, yielding an overall incidence of 10%, which falls within the internationally reported range (1–6% in unselected populations and up to 40–50% in high-risk patients) [28].

Patients who developed CIN were older (67.4 ± 4.9 vs. 49.5 ± 9.1 years; p = 0.001), received higher contrast volumes, had higher baseline serum creatinine, and higher admission random blood sugar. All variables except ACE inhibitor use were statistically significant. A dose–response relationship was evident between contrast volume and CIN, particularly in Group II, where incidence reached 50% among those receiving >150 ml (p = 0.001). These findings are supported by Chong et al., who reported that in a large cohort of 3,036 PCI patients with normal baseline creatinine, CIN occurred in 7.3%, with age being an independent predictor of CIN (odds ratio 6.4), indicating older patients are more susceptible to nephropathy after PCI [23]. Similarly, Kumar et al. found that among 282 STEMI patients undergoing primary PCI, CIN occurred in 13.1%, with the risk significantly associated with increased contrast volume (>200 mL) and diabetes, reinforcing that higher contrast doses contribute to CIN risk [27].

Five factors were identified as significant predictors of CIN: age ≥70 years, pre-existing renal impairment, diabetes, combined diabetes and renal impairment, and contrast volume ≥100 ml, consistent with recognized international risk determinants [29].

Regarding outcomes, no patient died or required dialysis. Renal recovery was favorable, with serum creatinine returning to baseline within two weeks in all Group I patients and in 96% of Group II patients. Only 4% of Group II patients exhibited persistent creatinine elevation beyond three weeks, consistent with prior reports that CIN is typically reversible within two weeks [30].

LIMITATIONS

The present study has several limitations:

- **Sample size and setting:** This study included a small number of patients from a single center, and the findings need confirmation through larger multi-center trials.
- **CIN definition and confounding factors:** CIN was defined based on absolute or relative increases in serum creatinine. Although 10% of patients developed CIN after contrast exposure, other factors such as hemodynamic instability may have contributed to renal impairment and influenced outcomes.
- **Limited renal function assessment:** Ideally, CIN studies should include multiple renal function variables with daily follow-up for 4–5 days post-contrast. This study used serum creatinine alone, which, while widely used for CIN definition, is less sensitive than creatinine clearance in detecting subtle renal function changes.

CONCLUSION

Contrast-induced nephropathy (CIN) is a common and clinically significant cause of iatrogenic acute renal dysfunction. Our study concludes that diabetic patients with pre-existing renal impairment are at the highest risk for developing CIN, whereas patients with normal renal function, with or without diabetes, are at low risk. Patients with pre-existing renal impairment but without diabetes have an intermediate risk. Additionally, age (≥70 years) and contrast volume were identified as independent risk factors, with nephrotoxicity positively correlated with higher contrast doses, particularly in high-risk patients. Although most patients recover spontaneously within two weeks, renal function may remain impaired in a small proportion beyond this period.

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Conflicts of interest

There are no conflicts of interest.

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