

The Protective Effect of Dapagliflozin Against Contrast-Induced Nephropathy After Percutaneous Coronary Catheterization in Patients Moderate to High-Risk for CIN

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ABSTRACT

Background: Dapagliflozin may have a reno-protective effect on patients with chronic renal disease. Additionally, there is mounting evidence that it can reduce the incidence of contrast-induced nephropathy (CIN) and protect against acute renal injury.

Objective: To assess the role of dapagliflozin in the prevention of CIN in patients with moderate to high risk for CIN who are undergoing cardiac catheterization and PCI.

Patients and Methods: Two subgroups of 200 moderate-to-high-risk CIN patients who received percutaneous coronary intervention (PCI) were created: Group A: Included patients who were already on dapagliflozin 10mg before undergoing cardiac catheterization and PCI and Group B: Included patients who weren't on dapagliflozin. Every patient underwent a thorough history taking, physical examination, echocardiogram, twelve-lead ECG, routine laboratory tests and evaluation of their Mehran risk score.

Results: The incidence of CIN was higher in Group B according to the KDIGO definition (22% vs. 41%, $p = 0.002$), and higher post-intervention S. creatinine (1.54 ± 0.4 vs 1.8 ± 0.52 , $p = 0.03$). Hemoglobin levels were significantly lower in CIN cases of Group B compared to Group A (11.42 ± 1.95 vs. 13.6 ± 1.35 g/dL, $p = 0.006$). Patients with CIN had significantly higher HbA1c levels (mean 10.35 ± 1.05 vs. 7.97 ± 0.84 , $p < 0.001$) and higher Mehran risk scores (mean 9.36 ± 1.71 vs. 7.13 ± 1.42 , $p < 0.001$). Statin use was significantly less common among CIN cases (36.36% vs. 98.72%, $p < 0.001$). Multivariate logistic regression analysis revealed that the odds ratio of HbA1c was 47.889, with a significant logistic regression relationship between the two variables ($p = 0.004$). Odds ratio of Mehran score was 6.254, with a significant logistic regression relationship between the two variables ($p = 0.029$).

Conclusion: Dapagliflozin may have a protective effect in patients with moderate to high risk for developing CIN, especially in complex interventions with high contrast volume.

KEYWORDS: Contrast-induced nephropathy; Dapagliflozin; Cardiac catheterization.

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INTRODUCTION

Revascularization by percutaneous coronary intervention (PCI) has been shown to significantly reduce mortality in patients with coronary artery disease (CAD).¹ Contrast-induced acute kidney damage (CI-AKI), which is characterized as a new-onset or an exacerbation of renal impairment following administration of CM without other possible reasons, has been reported in a percentage of CAD patients. According to Chalikias *et al.*² the incidence of CI-AKI varies between 1.3 and 33.3%. Type 2 diabetes mellitus (T2DM) patients are significantly more susceptible to CI-AKI, a serious side event that is becoming more common in the overall population of CAD patients.³

Sodium-glucose cotransporter 2 (SGLT2) inhibitors are new medications used to treat type 2 diabetes because they specifically stop glucose from being reabsorbed in the renal tubules. According to van Baar *et al.*⁴ SGLT2 inhibitors (SGLT2i) mechanically reduce blood glucose levels, enhance glucose excretion, and prevent the kidneys from reabsorbing glucose.

In addition to lower rates of cardiovascular events and mortality, several multicenter trials demonstrated a notable decline in the incidence of worsening kidney disease.⁵ However, nothing is known about how SGLT2i affects the prevalence of CI-AKI in PCI patients. Compared to patients taking alternative glucose-lowering drugs, Current real-world studies have shown that patients on SGLT2i were less likely to develop AKI and experienced a smaller drop in their estimated Glomerular Filtration Rate (eGFR).⁶

The current study aimed to focus on dapagliflozin's potential to prevent CIN in moderate-to-high-risk individuals having cardiac catheterization and PCI.

Patients and methods

Two subgroups of 200 patients who had cardiac catheterization and PCI at Badr University Hospital and 15 of May Hospital between October 2022 and October 2023 were included in this study because they were considered to be at moderate to high risk for CIN based on the Mehran Score.

- **Group A:** Prior to receiving cardiac catheterization or PCI, patients in this group had been taking dapagliflozin 10 mg for at least four weeks.
- **Group B:** This group of patients did not receive dapagliflozin prior to PCI or cardiac catheterization.

The inclusion criteria were moderate to high-risk patients for CIN based on Mehran score who undergoing PCI or cardiac catheterization, glomerular filtration rate (GFR) ≥ 30 ml/min/1.73 m² [CKD stage G1–G3], and age more than 18. The study excluded patients with cardiogenic shock, active cancer, pregnancy or desire to get pregnant during the follow-up period, and eGFR < 29 ml/min/1.73m².

All patients were subjected to complete history taking, physical examination, including vital sign evaluation, pallor, cyanosis, jaundice, and lymph node enlargement, as well as a comprehensive examination to rule out systemic illnesses, laboratory investigations including complete blood count, liver and kidney functions, and coagulation profile, twelve-lead resting ECG, echocardiography and Mehran risk score assessment.

Kidney Function Tests:

Serum creatinine and blood urea were measured both before and 48–72 hours after the intervention. -eGFR based on the MDRD formula was calculated to determine renal function according to age, gender, race, and creatinine levels.⁷ The formula for eGFR (mL/min/1.73 m²) is (serum creatinine) $-1.154 \times (\text{age}) - 0.203 \times (0.742 \text{ if female}) \times (1.212 \text{ if Black})$.

Mehran score

The Mehran score (6 points or higher) indicated that the two patient groups were at moderate to high risk for CIN. Age, diabetes, anemia, contrast media volume, eGFR, hypertension, intra-aortic balloon pump, and congestive heart failure were among the factors that were used to calculate Mehran's score.⁸ A number of variables that have been linked to a higher risk of contrast-induced nephropathy are taken into consideration by the Mehran score. These components include:

- **Hypotension:** Low blood pressure, defined as a systolic blood pressure of less than 80 at the time of the procedure (it equals 5 points).
- **Intra-aortic balloon pump (IABP) use:** Use of an intra-aortic balloon pump to assist cardiac function during the procedure (it equals 5 points).
- **Congestive heart failure (CHF):** NYHA class III (it equals 5 points).
- **Age:** Older age was associated with an increased risk. (older than 75 years old equals 4 points)
- **Diabetes:** Presence of diabetes mellitus (it equals 3 points).
- **Anemia:** The presence of low red blood cell counts or hemoglobin levels, HCT less than 39 in men and less than 36 in women (it equals 3 points).
- **Contrast media volume:** Higher volumes of contrast media used during the procedure (each 100 ml equals 1 point).
- **Serum creatinine:** A measure of kidney function (serum creatinine more than 1.5 equals 4 points).

The sum of the scores for each factor was used to calculate the likelihood that a person will develop contrast-induced nephropathy. Four risk categories for CIN were established based on the cut-off points and intervals mentioned by **Mehran et al.**⁸.

- Low ≤ 5 pts.
- Moderate between six and ten.
- Elevated between 11 and 16.
- Really high > 16 .

Serum creatinine levels were measured for all patients after 48-72 hours.

Table 1: Mehran Score for CIN⁸

(a)	
Risk factors	Score
Hypotension	5
IABP	5
CHF	5
Age > 75	4
Anemia	3
Diabetes	3
Contrast volume (cc)	1/100 cc
Serum Cr > 1.5	4

OR		
GFR<60 mL/min/1.73 m²		2 for 40–60
		4 for 20–40
		6 for <20
(b)		
Risk score	Risk of CIN	Risk of dialysis
<5	7.5%	0.04%
6–10	14%	0.12%
11–16	26.1%	1.09%
>16	57.3%	12.6%

IABP: Intra-aortic balloon pump, CHF: Congestive heart failure, CIN: Contrast-induced nephropathy

Outcome Measurements and Follow-up:

The incidence of CIN in each of the two study arms is compared. The European Society of Urogenital Radiology (ESUR) initially defined CIN as a condition in which, within 72 hours of intravascular iodine contrast medium administration, the serum creatinine (Scr) content rose by 25%, or 0.5 mg/dL, from the baseline value without the need for surgery, nephrotoxic medications, or other interventions.^{9, 10} It is defined as an absolute rise of 0.3 mg/kg in serum creatinine content relative to baseline within 48 hours after intravascular infusion of iodine contrast medium, despite the fact that the KDIGO employed the CI-AKI in 2012.¹¹

Ethical Considerations:

The Helwan Ethical Committee reviewed and approved the study protocol (serial 25-2022). Every participant who shared in the study gave their written informed consent.

Statistical Analysis

SPSS (Version 22) for Windows was used to code, process, and analyze the gathered data. When necessary, the relevant statistical tests were applied. Statistical significance was defined as P values < 0.05 (5%). The results for qualitative components were presented as numbers and percentages, whereas the mean ± standard deviation (SD) was used for quantitative variables.

RESULTS

The contrast volume employed in our study was comparatively considerable because the majority of the patients had complex PCI treatments (LM, MVD, bifurcation lesions, and CTO operations).

With the exception of a noticeably greater incidence of diabetes in group A (Dapagliflozin users), The study groups did not differ significantly in terms of comorbidities or demographic data. The groups under investigation differed statistically significantly in terms of the anti-diabetic drugs; group A took more oral hypoglycemics. Since all of the study's diabetic patients had type 2 diabetes, neither DM duration nor HbA1c differed statistically significantly. Hemoglobin and HbA1c levels did not substantially differ between the study groups. Both the post-intervention s-creatinine and the net increase in s-creatinine were considerably lower in Group A. Regarding the injected contrast amount or material, Mehran score, pre- or post-intervention GFR, and creatinine levels before to the intervention, the A and B groups did not differ significantly. According to the ESUR criteria, which states that 20% of patients on Dapagliflozin develop CIN and 24% of group B acquire CIN, there was no discernible difference in the incidence of CIN between the two groups. However, the incidence of CIN was significantly reduced in the dapagliflozin group based on the KDIGO criteria. (Table 2).

Table 2: Demographic and clinical data among the study groups

	Group A (n=100)	Group B (n=100)	P-value
Age (years)			
Mean±SD.	60.6±9.08	60.98±7.25	0.818
Median (IQR)	62.5(54-68)	61(57-66.5)	
Range	(40-77)	(45-76)	
Gender			
Male	30(60%)	36(72%)	0.179
Female	20(40%)	14(28%)	
Comorbidities			
Smoking	50(50%)	48(48%)	1
HTN	46(46%)	46(46%)	1
DM	88(88%)	66(66%)	0.009
Hypotension	2(2%)	0(0%)	0.315
IABP	0(0%)	0(0%)	-
CHF (NYHA III)	30(30%)	32(32%)	0.829
DM duration			
Mean±SD.	15.38±7.99	16.61±7.72	0.496

Median (IQR)	14(10-20)	15(10-20)	
Range	(5-40)	(7-35)	
DM drugs			
Oral hypoglycemic only	58(58%)	34(34%)	<0.001
Insulin only	4(4%)	30(30%)	
Both	28(28%)	2(2%)	
HbA1c			
Mean±SD.	8.43±1.46	8.72±1.2	0.339
Median (IQR)	8(7.9-9)	8.2(8-9.5)	
Range	(5.5-12)	(7.5-12)	
Hemoglobin			
Mean±SD.	13.01±1.79	12.59±1.96	0.268
Median (IQR)	13.5(12-14.38)	13(10.12-14)	
Range	(9-16)	(9.5-15.5)	
Contrast volume (cc)			
Mean±SD.	287±61.29	293±67.77	0.643
Median (IQR)	300(250-300)	300(212.5-350)	
Range	(200-400)	(150-400)	
≤250	92(92%)	89(89%)	0.507
>250	7(7%)	11(11%)	
eGFR pre-intervention			
Mean±SD.	80.8±17.31	83.03±19.87	0.551
Median (IQR)	80(67.25-94.75)	84.5(72-96)	
Range	(49-130)	(1.4-130)	
eGFR post-intervention			
Mean±SD.	67.06±19.86	69.32±21.15	0.583
Median (IQR)	65(54.5-82)	74(58.25-82)	
Range	(25-130)	(30-110)	
ΔeGFR	-13.7±3.4	-15.4±2.8	0.2
Mehran score			
Mean±SD.	7.78±1.79	7.46±1.7	0.362
Median (IQR)	7.5(6-9)	7(6-9)	
Range	(6-11)	(6-12)	
Serum Creatinine pre-intervention			
Mean±SD.	1.15±0.21	1.18±0.19	0.455
Serum Creatinine post-intervention			
Mean±SD.	1.54±0.41	1.8±0.52	0.03
CIN (ESUR)	20(20%)	24(24%)	0.629
CIN (KDIGO)	22(22%)	41(41%)	0.002
Serum creatinine Δ			
Mean±SD.	0.3±0.1729	0.4±0.2828	0.03

P value less than 0.05 was considered statistically significant, HTN: Hypertension, DM: Diabetes Mellitus, IABP: Intra-Aortic Balloon Pump, CHF: Congestive Heart Failure, NYHA: New York Heart Association, HbA1c: Glycated Hemoglobin, cc: Cubic Centimeters, eGFR: Estimated Glomerular Filtration Rate, SD: Standard Deviation, IQR: Interquartile Range, CIN: Contrast-Induced Nephropathy, ESUR: European Society of Urogenital Radiology.

Regards ESUR definition: Age, gender, comorbidities (such as smoking, hypertension, diabetes, and CHF), HbA1c levels, contrast volume, and Mehran risk scores did not differ statistically significantly between CIN cases in Group A and Group B ($p > 0.05$ for all). However, Group B CIN cases had significantly lower hemoglobin levels than Group A cases (11.42 ± 1.95 vs. 13.6 ± 1.35 g/dL, $p = 0.006$). There were no statistically significant differences between the CIN and No-CIN groups in terms of age, gender, smoking, hemoglobin level, or contrast volume when comparing patients who developed CIN and those who did not (Table 3). However, those in the CIN cohort had significantly higher HbA1c levels (mean 10.35 ± 1.05 vs. 7.97 ± 0.84 , $p < 0.001$) and Mehran risk scores (mean 9.36 ± 1.71 vs. 7.13 ± 1.42 , $p < 0.001$). Additionally, statin use was significantly lower among CIN cases (36.36% vs. 98.72%, $p < 0.001$) (Table 4).

Table 3: Comparison between CIN (ESUR) cases in group A&B regarding risk factors results

	CIN cases in group A (n=20)	CIN cases in group B (n=24)	P-value
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Age (years)			
Mean±SD.	60.8±10.57	63.08±8.26	0.585
Median (IQR)	62.5(59-68.75)	66(59.25-68.5)	
Range	(40-72)	(48-72)	
Gender			
Male	14(70%)	18(75%)	0.793
Female	6(30%)	6(25%)	
Comorbidities			
Smoking	12(60%)	12(50%)	0.639
HTN	10(50%)	18(75%)	0.225
DM	20(100%)	20(83.33%)	0.176
Hypotension	0(0%)	0(0%)	-
CHF	6(60%)	8(33.33%)	0.211
HbA1c (%)			
Mean±SD.	10.55±1.04	10.15±1.08	0.41
Median (IQR)	10.25(10-11.38)	10(9.62-10.88)	
Range	(9-12)	(8-12)	
Hemoglobin (g/dL)			
Mean±SD.	13.6±1.35	11.42±1.95	0.006
Median (IQR)	14(13.12-14.38)	10.25(10-13.25)	
Range	(10.5-15)	(9.5-14.5)	
Contrast volume (cc)			
Mean±SD.	215±62.58	191.67±73.34	0.18
Median (IQR)	200(150-350)	200(150-350)	
Range	(100-400)	(100-400)	
Mehran score			
Mean±SD.	9.4±1.84	9.33±1.67	0.931
Median (IQR)	10(8.25-11)	9(9-10.25)	
Range	(6-11)	(6-12)	

P value less than 0.05 was considered statistically significant, HTN: Hypertension, DM: Diabetes Mellitus, CHF: Congestive Heart Failure, HbA1c: Glycated Hemoglobin, cc: Cubic Centimeters, SD: Standard Deviation, IQR: Interquartile Range, CIN: Contrast-Induced Nephropathy.

Table 4: Comparison between the patients who developed CIN (ESUR) in both groups and the other patients regarding risk factors results

	CIN (n=44)	group	No (n=156)	CIN group	P-value
Age (years)					
Mean±SD.	62.05±9.22		60.44±7.89		0.462
Median (IQR)	65(59-68.75)		61(55.25-66.5)		
Range	(40-72)		(40-77)		
Gender					
Male	32(72.73%)		100(64.1%)		0.428
Female	12(27.27%)		56(35.9%)		
Comorbidities					
Smoking	24(54.55%)		76(48.72%)		0.629
HTN	28(63.64%)		64(41.03%)		0.06
DM	40(90.91%)		114(73.08%)		0.079
Hypotension	0(0%)		2(1.28%)		0.594
CHF	20(45.45%)		42(26.92%)		0.097
HbA1c (%)					
Mean±SD.	10.35±1.05		7.97±0.84		<0.001
Median (IQR)	10(9.88-11)		8(7.5-8.5)		
Range	(8-12)		(5.5-10)		
Hemoglobin (g/dL)					
Mean±SD.	12.41±2		12.91±1.84		0.301
Median (IQR)	13(10.12-14)		13.25(12-14)		
Range	(9.5-15)		(9-16)		
Contrast volume (cc)					
Mean±SD.	202.27±68.1		186.54±63.28		0.338

Median (IQR)	200(162.5-350)	200(112.5-350)	
Range	(100-300)	(150-300)	
Mehran score			
Mean±SD.	9.36±1.71	7.13±1.42	
Median (IQR)	9.5(9-11)	7(6-8)	<0.001
Range	(6-12)	(6-11)	
Statin usage			
Yes	16(36.36%)	144(98.72%)	<0.001

P value less than 0.05 was considered as statistically significant, CIN: Contrast-Induced Nephropathy, HTN: Hypertension, DM: Diabetes Mellitus, CHF: Congestive Heart Failure, HbA1c: Glycated Hemoglobin, g/dL: Grams per Deciliter, cc: Cubic Centimeters, SD: Standard Deviation, IQR: Interquartile Range, Min-Max: Minimum–Maximum.

The univariate logistic regression analysis predicting CIN using odds ratios and 95% CI was displayed in **Table 5**. The two variables had a highly significant logistic regression connection ($p < 0.001$), and the odds ratio for HbA1c was 27.587. There was a highly significant logistic regression link between the two variables ($p < 0.001$), and the Mehran score odds ratio was 2.211. CIN is predicted by multivariate logistic regression analysis using odds ratios and 95% CI. There was a significant logistic regression link between the two variables ($p = 0.004$), and the odds ratio for HbA1c was 47.889 as well. There was a significant logistic regression link between the two variables ($p = 0.029$), and the Mehran score odds ratio was 6.254.

Table 5: Univariate logistic regression analysis for the prediction of CIN (ESUR)

		CIN			
		OR	95% CI		P
			Lower	Upper	
Univariate	HbA1c	27.587	5.104	149.119	<0.001
	Mehran score	2.211	1.568	3.119	<0.001
Multivariate	HbA1c	47.889	3.439	666.897	0.004
	Mehran score	6.254	1.208	32.391	0.029

P value less than 0.05 was considered statistically significant, CIN: Contrast-Induced Nephropathy, OR: Odds Ratio, CI: Confidence Interval, HbA1c: Glycated Hemoglobin.

The frequency of CIN did not significantly change between diabetic patients on dapagliflozin and those who were not (**Table 6**).

Table 6: CIN (ESUR) incidence in diabetic patients of both groups.

	Group A	Group B	p value
	<i>Diabetic</i> <i>N=88</i>	<i>Diabetic</i> <i>N=66</i>	
CIN	20(22.7%)	19(28.3%)	0.350

CIN: contrast-induced nephropathy

DISCUSSION

The incidence of CIN in the current study was 22% of the studied group (20% of dapagliflozin users and 24% of non-users); we attributed this to the relatively high contrast volume resulting from the complex coronary intervention in the majority of cases, as well as the moderate to high risk for CIN that existed prior to the intervention. We discovered that the dapagliflozin users had a significantly lower post-intervention serum creatinine at 48 hours compared to the non-users, however that, both before and after the intervention, there was no discernible change in eGFR between the two groups. Urea nitrogen, creatinine, and eGFR levels did not differ substantially between the two groups either before or 24 hours after PCI, according to Hua *et al.*¹²; however, the creatinine levels in the SGLT2i users group were significantly lower than those in the non-user group ($P < 0.05$) 48 and 72 hours after PCI. This is consistent with a research that had 242 people who used SGLT2 inhibitors and 242 people who didn't. Additionally, in a study by Paolisso *et al.*¹³, 646 patients with T2DM and AMI undergoing PCI included 111 SGLT2-I users and 535 non-SGLT2-I users. 72 hours following PCI, they found that creatinine levels were considerably lower among SGLT2-I users.

Hua *et al.*¹² revealed a significantly lower incidence of AKI using the KDIGO definition among SGLT2 inhibitor users (4.1% in users vs. 9.1% in nonusers, $P = 0.04$), but no significant difference in median peak creatinine values between the groups of users and nonusers ($P = 0.12$). These results were in line with the current study, which, using the ESUR definition, demonstrated no discernible change in the incidence of CIN between Dapagliflozin users and nonusers. Additionally, Nadkarni *et al.*¹⁴ discovered that SGLT2 inhibitor use was linked to a decreased risk of AKI both before and after propensity matching; the estimates of this association were qualitatively comparable across a number of sensitivity tests. Those on SGLT2 inhibitors did not experience

more severe AKI, as measured by changes in creatinine or peak serum creatinine. Lastly, dapagliflozin, which raises concerns about AKI, did not result in an elevated risk of AKI. Additionally, Paolisso *et al.*¹³ discovered that the overall rate of CI-AKI was much lower in SGLT2-I users than in non-users, and that SGLT2-I use was an independent predictor of a reduced rate of CI-AKI.

Furthermore, among the 646 AMI patients in the Paolisso *et al.*¹⁵ trial, 111 were SGLT2-I users and 535 were not; contrast-induced acute kidney damage was less common in SGLT2-I users ($p = 0.022$). Furthermore, compared to a placebo, dapagliflozin decreased newly developing or worsening renal illness by 24%, according to Mosenzon *et al.*¹⁶. Furthermore, Zhuo *et al.*¹⁷ found that the SGLT2 inhibitor group had a lower incidence of AKI than either the DPP-4 inhibitor group or the GLP-1RA group. They comprised 68,130 new SGLT2 inhibitor users who were paired with either new GLP-1RA or DPP-4 inhibitor users. These results suggest that SGLT2 inhibitors assist patients with cardiovascular disease by protecting their kidneys.

Out of all the patients we looked at, those who acquired CIN used statins far less frequently than those who did not (36.36% vs. 98.72%; $P < 0.001$). Our findings are consistent with a study conducted on 4386 patients by Lin *et al.*¹⁸, which discovered that the incidence of CI-AKI was much lower in the group of patients using statin medication. A meta-analysis by Wang *et al.*¹⁹ further supports the idea that statins can lower the incidence of CI-AKI in patients receiving CAG/PCI. They found that the statin group had a significantly lower incidence of CI-AKI than the control group (3.7% vs. 8.3%, RR, 0.46; $p < 0.00001$). Additionally, a meta-analysis of CI-AKI in patients with and without acute coronary syndromes by Marenzi *et al.*²⁰ found that short-term statins administered prior to therapy significantly decreased the risk of CI-AKI when compared to control.

In our study, the Mehran score of patients who developed CIN was substantially greater than that of individuals who did not (9.36 ± 1.71 vs. 7.13 ± 1.42 ; $P < 0.001$). Similar to this, in the Sato *et al.*²¹ study, Following PCI, 192 patients (8.7%) experienced CI-AKI. The incidence of CI-AKI increased gradually as the Mehran risk score grew ($p < 0.001$) following the division of the patients into four groups based on their Mehran risk score. The CI-AKI rate for the very high-risk group was more than four times greater than that of the low-risk group (OR 4.09, 95% CI: 1.72–9.17, $p = 0.002$), but the rates for the high-risk group and medium-risk groups were 1.49, CI: 0.89–2.42, $p = 0.120$, and 1.08, CI: 0.74–1.54, $p = 0.693$, respectively.

In STEMI patients receiving multi-vessel intervention, the Mehran risk score correctly classified 78% of the patients who developed CI-AKI, according to the findings of a meta-analysis conducted by Chatterjee *et al.*²² that comprised four studies. This is in line with the results of the multivariate logistic regression analysis used in this investigation, which indicated that one of the significant predictors of CIN was Mehran score (OR=6.254, C.I: 1.208-32.391; $p = 0.029$).

The current study found that patients who acquired CIN had significantly higher levels of HbA1c than patients who did not develop CIN, which is consistent with Zhang *et al.*²³ examination of 670 CAG/PCI patients' rate of preoperative HbA1c and postoperative CIN. $P < 0.001$ (10.35 ± 1.05 vs. 7.97 ± 0.84). Additionally, they found that rising HbA1c levels significantly increased the risk of CIN ($p < 0.0001$). Additionally, Qin *et al.*²⁴ discovered that patients receiving CAG who had elevated HbA1c had a greater risk of CIN than those who did not. Furthermore, Barbieri *et al.*²⁵ showed a relationship between the risk of developing CIN in people without diabetes and the HbA1c level. HbA1c was a significant predictor of CIN, per the multivariate logistic regression analysis conducted for our sample (OR=47.889, C.I: 3.439-666.897; $p = 0.004$). In a similar vein, Zhang *et al.*²³ discovered that their multivariate analysis indicated that people in the major high-risk category who had elevated HbA1c levels ($\geq 8.8\%$) were more likely to suffer from CIN disease.

Patients who received Dapagliflozin had significantly higher hemoglobin levels than those who did not (315 ± 62.58 vs. 291.67 ± 73.34 ; $P = 0.006$) among those who developed CIN in our study. This is consistent with a study by Stefánsson *et al.*²⁶ that involved 5325 patients and 14 placebo-controlled participants and found that hemoglobin levels increased steadily up to at least week 8 and remained stable in patients treated with dapagliflozin for 24 weeks. Additionally, in the Koshino *et al.*²⁷ Dapagliflozin-CKD trial, 443 (53.3%) of the patients who received dapagliflozin had their anemia corrected, indicating a link between dapagliflozin and the management or prevention of anemia in patients with chronic kidney disease (CKD), including those without diabetes.

CONCLUSION:

According to the KDIGO definition, dapagliflozin had a reno-protective effect against CIN, indicating that it may have a protective effect in this type of patient with moderate to high risk for developing CIN, particularly in complex interventions with high contrast volume, even though, under the ESUR definition, the frequency of CIN was not appreciably lower among dapagliflozin users. The current study indicates that the onset of contrast-induced nephropathy (CIN) is significantly correlated with lower hemoglobin levels, higher HbA1c values, and raised Mehran risk scores. Since statin use was much less prevalent among CIN patients, it seems to be notably protective. Furthermore, multivariate analysis revealed that the Mehran score and HbA1c were both independent predictors of CIN, highlighting the significance of risk stratification and glycemic control in patients having heart catheterization.

RECOMMENDATIONS:

More extensive multicenter studies with longer follow-up times are needed to evaluate dapagliflozin's prognostic effect on CIN. Modifiable risk factors for CI-AKI must be managed early.

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