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ORIGINAL ARTICLE

Role of Dapagliflozin in Protection of Contrast Induced Nephropathy in Patients Eligible for Angiographic Procedures

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ABSTRACT

Background: Research showed that Dapagliflozin demonstrated significant cardiorenal protective effects that may extend to prevent Contrast-induced nephropathy (CIN), that significantly complicates percutaneous coronary angioplasty. This research aimed to assess the role of Dapagliflozin for reducing contrast induced nephropathy (CIN) incidence before coronary angiographic procedures**.**

Methods: We performed a case–control study involving 82 coronary artery disease patients undergoing PCI, 41 received dapagliflozin 10 mg daily for ≥90 days before intervention and 41 served as controls. All patients received standard hydration and non-ionic contrast. CIN was outlined as ≥25% or ≥0.5 mg/dL increase in the serum creatinine within 72 h.

Results: Dapagliflozin group were associated with lower CIN incidence than those not on therapy (12.2% vs. 34.1%, p = 0.018). On univariate regression analysis, Dapagliflozin use p = 0.039), higher baseline eGFR (p = 0.001), and higher EF (p = 0.023) were protective factors. In contrast, diabetes mellitus (p = 0.034), hypertension (p = 0.011), smoking (p = 0.024), peripheral vascular disease (p = 0.028), and higher baseline Scr (p = 0.014) were potential risk factors Multivariate analysis confirmed Dapagliflozin use and higher baseline eGFR as independent protective predictors, while diabetes mellitus, and smoking were identified as risk factors for CIN.

Conclusion: Dapagliflozin was significantly associated with lower CIN incidence in individuals with IHD undergoing elective PCI.

Keywords: Percutaneous Coronary Intervention; Dapagliflozin; Contrast Induced Nephropathy.

INTRODUCTION

Dapagliflozin can protect the kidneys from deleterious effects of (CM) contrasted media [[1], that is used for diagnostic or therapeutic angiographic interventions [2]

Angiography is a cornerstone imaging technique that provides detailed anatomical and structural visualization of the vascular system. By injecting contrast media into blood vessels and capturing sequential X-ray images. Over the past century, angiography transformed from a purely diagnostic tool into a foundation for modern interventional therapies like coronary angioplasty [3]

Iodinated contrast agents are associated with a significant drawback—the development of contrast-induced nephropathy (CIN), which ranks as the third leading cause of acute kidney injury acquired during hospitalization [4]. This risk concerns the setting of percutaneous coronary intervention (PCI), a life-saving strategy for coronary artery (CAD) that is performed for millions of patients annually worldwide [5].

Sodium–glucose cotransporter-2 inhibitors (SGLT2i) have gained recognition as a newer group of glucose-lowering drugs for managing type 2 diabetes mellitus (T2DM) [6] Apart from

their metabolic benefits, SGLT2 inhibitors have consistently been associated with preservation of renal function [7] An early decrease in eGFR, commonly described as the "initial dip," is a recognized phenomenon within 2–4 weeks of SGLT2i initiation, after which renal function generally shows partial improvement by week 12 [8].

Recent evidence suggests that the benefits of SGLT2i may extend to prevent CIN. Their proposed protective mechanisms appear independent of glucose lowering and include anti-inflammatory and antioxidant actions that could counteract pathways implicated in CIN pathogenesis [9]. Huang et al. [10] specifically revealed that dapagliflozin attenuated contrast-induced nephropathy. However, despite encouraging signals, clinical data directly linking SGLT2i therapy with reduced CIN risk remain scarce. [10]

Few studies have directly assessed Dapagliflozin in patients undergoing PCI, leaving uncertainty about its independent protective effect in this setting. Therefore, this research aimed to assess the role of Dapagliflozin for protection of contrast induced nephropathy among patients eligible for angiography.

METHODS

We performed this case control research after being approved and validated by the ethical committee of Faculty of Medicine Menoufia University (approval code 3\2023 card 15) and written consent was obtained from all the patients after they are fully informed about the study and coded by numbers to preserve their confidentiality. Study was conducted on 82 patients who presented, between April 2023 and December 2024 for percutaneous coronary intervention in the Cath lab unite of The National Institute of Diabetes & Endocrinology, Cairo., Egypt.

We included patients' age ≥ 18 years. Who were admitted for percutaneous coronary intervention with approved indication according to the latest guidelines with normal renal function. Our exclusion criteria were patients with type I DM, Patients with elevated serum creatinine and /or urea level, hypotension, shock, and other causes of renal insufficiency before or after intervention. History of diabetic ketoacidosis, Active urogenital infection, pregnant females.

Patients were categorized into two groups according to Dapagliflozin intake: Group A (Cases): Including 41 patients who received Dapagliflozin 10mg once daily for at least 90 days before intervention (DAPA group). Group B (Control): including 41 patients who did not receive Dapagliflozin or other SGLT2i (non-DAPA group). All patients were given the same regular treatment in the form of proper hydration 24-48 hours before intervention. During the procedure, all cases were injected with ted non-ionic contrast. Serum creatinine level was measured in both groups before intervention, then 1, 3 days after intervention. CIN was classified as either a ≥25% increase in serum creatinine from baseline or an absolute rise of ≥0.5 mg/dL within 72 hours of intravascular contrast exposure [11]

All enrolled patients received a comprehensive clinical assessment. The medical history captured demographic data (age and sex) along with conventional cardiovascular risk factors, including diabetes mellitus, hypertension, dyslipidemia, smoking status, and prior evidence of renal impairment. Physical examination emphasized vital parameters such as blood pressure and heart rate, in addition to routine cardiac auscultation. Baseline laboratory testing included complete blood count (CBC), serum creatinine, and blood urea, which were re-evaluated at 24- and 72-hours following PCI. Renal function was further assessed by estimating the glomerular filtration rate (eGFR) using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation [12]

Liver function tests and international normalized ratio (INR) were assessed at admission. Left ventricular ejection fraction (LVEF) was assessed using transthoracic echocardiography, assessed by M-mode, modified Simpson method, and interpreted by a

cardiologist blinded to patient group allocation [13].

To minimize CIN risk, all patients received standardized periprocedural hydration with isotonic saline (0.9% NaCl) at a rate of 1 mL/kg/h for 12 hours before and after the procedure (adjusted for patients with reduced LVEF). Only low-osmolar, non-ionic contrast media were used, and total contrast volume was recorded for each case. Nephrotoxic agents, such as nonsteroidal anti-inflammatory drugs and aminoglycosides, were discontinued at least 48 hours prior to intervention when applicable.

Statistical analysis:

The distribution of continuous variables was first tested for normality using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Depending on the data type, categorical variables were assessed using either the chi-square test or Fisher's exact test, while continuous variables were analyzed with the independent t-test for normally distributed data or the Mann–Whitney U test for skewed data. Serial changes in serum creatinine and eGFR within each group were evaluated by repeated-measures ANOVA followed by post-hoc comparisons. Quantitative variables are presented as mean ± standard deviation, whereas qualitative variables are summarized as frequencies and percentages. Logistic regression models were applied to explore risk factors for contrast-induced acute kidney injury; univariate analysis was first performed, and variables reaching significance were then entered into a multivariate model to identify independent predictors. Statistical significance was defined at a threshold of $p < 0.05$. All computations were carried out using SPSS version 26.0 (IBM, USA).

RESULTS

Eighty-two patients were analyzed, mean age 53.4 ± 6.1 years, of whom 47 (57.3%) were males and 35 (42.7%) females. Forty-one patients had received dapagliflozin for at least 90 days before intervention, while the remaining 41 served as controls. Baseline demographic and clinical characteristics, including diabetes mellitus, hypertension, smoking status, dyslipidemia, peripheral vascular disease, heart rate, and ejection fraction, were comparable between the two groups, with no statistically significant differences were observed ($p > 0.05$) (Table 1).

Renal Function Trends

At baseline, serum creatinine (Scr) and eGFR did not differ statistically significant between Dapagliflozin and non-Dapagliflozin users (1.03 ± 0.13 vs. 0.98 ± 0.17 mg/dL, $p = 0.167$; 74.9 ± 7.6 vs. 79.6 ± 15.4 mL/min/1.73 m², $p = 0.077$). Following PCI, both groups demonstrated statistically significant rise in Scr and decline in eGFR at 24 and 72 hours compared with baseline ($p < 0.001$ within groups). However, between-group comparisons showed that the Dapagliflozin cohort had statistically significant lower Scr at both day 1 (1.20 ± 0.11 vs. 1.32 ± 0.33 mg/dL, $p = 0.033$) and day 3 (1.26 ± 0.12 vs. 1.39 ± 0.35 mg/dL, $p = 0.028$). Correspondingly, eGFR decline was less pronounced in the Dapagliflozin group (Table 2).

Contrast-Induced Nephropathy Incidence

CIN occurred among 19 patients overall (23.2%). It was statistically significantly lower among Dapagliflozin group than controls (5/41 [12.2%] vs. 14/41 [34.1%]; $p = 0.018$) (Figure 1).

Our results demonstrated that DAPA group were associated with lower CIN incidence than those not on therapy. Multivariate analysis confirmed Dapagliflozin use and higher baseline eGFR as independent protective predictors, while diabetes mellitus, and smoking were risk factors for CIN

CIN vs. Non-CIN Subgroup Analysis

Patients who developed CIN had statistically significantly higher baseline Scr (1.09 ± 0.15 vs. 0.99 ± 0.14 mg/dL, $p = 0.009$) and higher post-procedure Scr at day 1 (1.49 ± 0.26 vs. 1.20 ± 0.21 mg/dL, $p < 0.001$) and day 3 (1.54 ± 0.30 vs. 1.27 ± 0.23 mg/dL, $p < 0.001$). Likewise, baseline eGFR was statistically significantly lower in the CIN group (69.4 ± 11.7 vs. 81.9 ± 12.7 mL/min/1.73 m², $p < 0.001$), with further reductions at day 1 (52.6 ± 18.7 vs. 71.9 ± 10.2 mL/min/1.73 m², $p <$

0.001) and day 3 (49.7 ± 17.7 vs. 67.5 ± 8.9 mL/min/1.73 m², $p < 0.001$) did not differ statistically significantly between CIN and non-CIN groups (Table 3). Routine laboratory tests, including CBC and liver function, were also similar across groups ($p > 0.05$). (Table 3).

Amount of dye in CIN and non-CIN patients

The contrast volume of non-CIN (141.1 ± 33.3 vs. 134.3 ± 38.8 mL, $p = 0.494$) was not statistically significantly higher than the CIN group, p=0.494, all used dye was non-ionic. (Table 4).

DAPA intake duration

The duration of Dapagliflozin therapy (5.0 ± 1.8 vs. 5.9 ± 1.8 months, $p = 0.297$) did not differ statistically significantly between those who developed CIN and those who didn't, p>0.05. (Table 5).

Predictors of CIN

On univariate regression analysis, Dapagliflozin use (OR 0.299, 95% CI 0.095–0.939; $p = 0.039$), higher baseline eGFR (OR 0.907, 95% CI 0.855–0.962; $p = 0.001$), and higher EF (OR 0.913, 95% CI 0.844–0.987; $p = 0.023$) were protective factors. In contrast, diabetes mellitus (OR 9.54, 95% CI 1.19–76.37; $p = 0.034$), hypertension (OR 5.67, 95% CI 1.49–21.50; $p = 0.011$), smoking (OR 3.57, 95% CI 1.18–10.76; $p = 0.024$), peripheral vascular disease (OR 4.07, 95% CI 1.16–14.29; $p = 0.028$), and higher baseline Scr (OR 237.0, 95% CI 3.05–18,417; $p = 0.014$) were potential risk factors (Table 4). Multivariate analysis confirmed that dapagliflozin therapy (adjusted OR 0.161 (0.039-0.666); $p = 0.032$) and DM (adjusted OR 9.794 (1.045-91.792); $p = 0.046$) and smoking (adjusted OR 8.017 (1.594-40.312); $p = 0.012$) and eGFR (adjusted OR 0.920 (0.855-0.991); $p = 0.027$) were independent predictors of CIN (Table 6).

Table 1: Demographic data, risk factors and ejection fraction of the studied groups (n=82)

Parameter	Group A (n=41)	Group B (n=41)	P value
Age (years)	53.4 ± 6.1	55.1 ± 5.5	0.189
Male, n (%)	25 (61.0%)	22 (53.7%)	0.503
Female, n (%)	16 (39.0%)	19 (46.3%)	
Diabetes Mellitus (DM), n (%)	30 (73.2%)	28 (68.3%)	0.627
Hypertension (HTN), n (%)	19 (46.3%)	26 (63.4%)	0.120
Smoking, n (%)	21 (51.2%)	20 (48.8%)	0.825
Peripheral vascular disease (PVD), n (%)	5 (12.2%)	8 (19.5%)	0.364
Cerebrovascular stroke (CVS), n (%)	3 (7.3%)	1 (2.4%)	0.305
Dyslipidemia, n (%)	40 (97.6%)	41 (100%)	0.314
Obesity (kg/m²)	28.0 ± 4.1	28.2 ± 3.7	0.805
Systolic blood pressure (SBP, mmHg)	130.0 ± 14.3	128.0 ± 16.5	0.569
Diastolic blood pressure (DBP, mmHg)	77.1 ± 9.6	77.6 ± 9.7	0.819
Heart rate (HR, bpm)	90.2 ± 9.8	87.9 ± 9.8	0.281
Ejection fraction (EF, %)	55.7 ± 7.4	52.8 ± 9.8	0.143

DM: Diabetes mellitus; HTN: Hypertension; PVD: Peripheral vascular disease; CVS: Cerebrovascular stroke; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HR: Heart rate; EF: Ejection fraction.

Table 2: Comparison of serial creatinine and eGFR measurements of the studied patients (n=82)

		DAPA (n=41)	Non- DAPA (n=41)	P value**
Creatinine (mg/dL)	pre	1.031 ± 0.13	0.985 ± 0.166	0.167
	after 1 day	1.201 ± 0.105	1.320 ± 0.334	0.033
	after 3 days	1.262 ± 0.124	1.392 ± 0.354	0.028
P value&		<0.001	<0.001	
		P_1=<0.001 P_2=<0.001 P_3=<0.001	P_1=<0.001 P_2=<0.001 P_3=<0.001	
eGFR (mL/min/ 1.73 m²)	pre	74.9 ± 7.6	79.6 ± 15.4	0.077
	after 1 day	67.8 ± 10.2	67.5 ± 18.5	0.941
	after 3 days	64.2 ± 9.1	63.0 ± 16.9	0.690
P value&		<0.001	<0.001	
		P_1=<0.001 P_2=<0.001 P_3=<0.001	P_1=<0.001 P_2=<0.001 P_3=<0.001	

eGFR: Estimated Glomerular Filtration Rate; DAPA: Dapagliflozin. Values are mean ± SD. Student t-test used for between-group comparison. & One-Way ANOVA of repeated measures.

Table 3: Comparison between CIN and non-CIN groups for creatinine, eGFR, (n=82)

Parameter	CIN (n=19)	Non-CIN (n=63)	P value
Pre-Creatinine (mg/dL)	1.089 ± 0.15	0.985 ± 0.143	0.009
Pre-eGFR (mL/min/1,73 m²)	69.4 ± 11.7	81.9 ± 12.7	<0.001
Creatinine (mg/dL) after 1 day	1.488 ± 0.258	1.197 ± 0.214	<0.001
Creatinine (mg/dL) after 3 days	1.544 ± 0.303	1.266 ± 0.230	<0.001
eGFR (mL/min/1.73 m²) after 1 day	52.6 ± 18.7	71.9 ± 10.2	<0.001
eGFR (mL/min/1.73 m²) after 3 days	49.7 ± 17.7	67.5 ± 8.9	<0.001

CIN: Contrast-induced nephropathy; eGFR: Estimated Glomerular Filtration Rate; DAPA: Dapagliflozin.

Table 4: amount of dye in CIN and non-CIN patients:

	CIN (n=19)	Non- CIN (n=63)	P value
Amount of the nonionic dye (ml)	141.1 ± 33.3	134.3 ± 38.8	0.494

Table 5: DAPA intake duration in CIN and non-CIN groups:

	CIN (n=5)	Non- CIN (n=36)	P value
DAPA intake duration (months)	3.8 ± 2.9	5.6 ± 2.2	0.113

Table 6: Univariate and multivariate regression analysis (binary logistic, enter method) for independent risk factors for post-contrast administration (n=82)

Predictor	Univariate OR (95% CI)	P value	Multivariate Adjusted OR (95% CI)	P value
DAPA use	0.299 (0.095–0.939)	0.039	0.161 (0.039-0.666)	0.032
Diabetes Mellitus (DM)	9.537 (1.191–76.367)	0.034	9.794 (1.045-91.792)	0.046
Hypertension (HTN)	5.667 (1.494–21.496)	0.011		
Smoking	3.565 (1.181–10.764)	0.024	8.017 (1.594-40.312)	0.012

Predictor	Univariate OR (95% CI)	P value	Multivariate Adjusted OR (95% CI)	P value
Peripheral vascular disease (PVD)	4.071 (1.160–14.291)	0.028		
Ejection fraction (EF, %)	0.913 (0.844–0.987)	0.023		
Pre-procedure creatinine (mg/dL)	237.0 (3.049–18417)	0.014	12.139 (0.057-2576)	0.242
Pre-procedure eGFR (mL/min/1.73 m²)	0.907 (0.855–0.962)	0.001	0.920 (0.855-0.991)	0.027

OR: Odds ratio; CI: Confidence interval; DM: Diabetes mellitus; HTN: Hypertension; PVD: Peripheral vascular disease; EF: Ejection fraction; eGFR: Estimated Glomerular Filtration Rate; DAPA: Dapagliflozin.

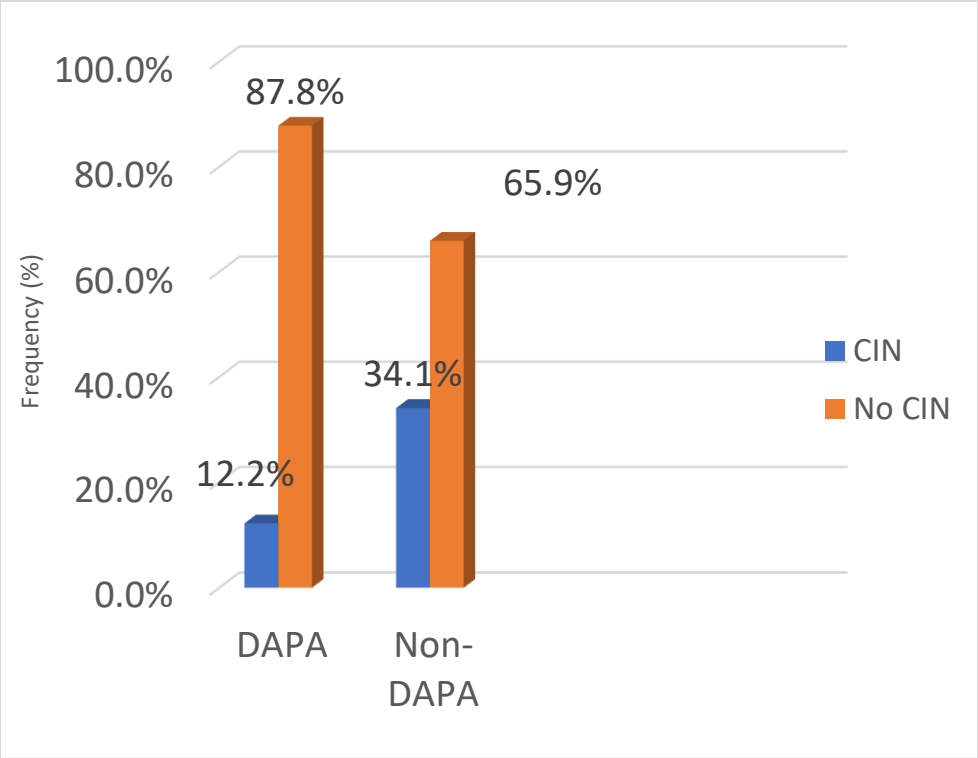

Figure 1: Bar chart displaying the rate of CIN in Group A (DAPA) vs Group B (non-DAPA) showed that 5 (12.2%) of Group A developed CIN vs 14 (34.1%) in Group B, p=0. 018.

For DAPA users: CIN: 12.2% [95% CI: 2.4%, 22.0%]. No CIN: 87.8% [95% CI: 78.0%, 97.6%]. For non-DAPA users: CIN: 34.1% [95% CI: 19.6%, 48.6%] No CIN: 65.9% [95% CI: 51.4%, 80.4%].

DISCUSSION

The present study explored whether Dapagliflozin could provide renal protection from injurious effects of (CM) contrasted media in individuals undergoing PCI, considering the expanding evidence of the dual cardiovascular and renal benefits of SGLT2 inhibitors. Our results demonstrated that DAPA group were associated with lower CIN incidence than those not on therapy (12.2% vs. 34.1%). Multivariate analysis confirmed Dapagliflozin use and higher baseline eGFR as independent protective predictors, while diabetes mellitus, and smoking were identified as risk factors for CIN. These findings suggest that Dapagliflozin may confer additional protection beyond glycemic control, potentially mitigating CIN in high-risk populations.

The PCI remains central to coronary artery disease management, restoring blood flow through balloon angioplasty and stent placement. [14], the procedure carries risks,

including CIN, which continues to represent a clinically significant complication of contrast media exposure,[15]

According to the present study, Dapagliflozin significantly was associated with lower CIN incidence, affecting only 12.2% of patients compared with 34.1% in the control group. This observation mirrors that of the meta-analysis Thakurathi et al, with a total of 4,321 patients (SGLT2i users: 1,348; non-users 2,973). The risk of CIN was significantly lower in SGLT2i users than in non-users. Creatinine levels showed a smaller increase in SGLT2i users compared to non-users , and the decline in eGFR was greater in non-users than in SGLT2i users. [16] This meta-analysis supports an additional nephro-protective effect of SGLT2i: in patients with acute MI exposed to contrast from CAG/PCI, SGLT2i reduces CIN risk and better preserves renal function[16]

Yang et al. [17], who documented a lower incidence of CIN in Dapagliflozin-treated patients versus controls (5.8% vs. 11.7%). After controlling for confounding variables, dapagliflozin remained an independent predictor of reduced CIN risk (p = 0.008). In addition, their analysis highlighted a significant decline in major adverse cardiac events among the dapagliflozin group (p = 0.009), with Cox regression confirming a protective effect (p = 0.036).

Similarly, Santos-Gallego et al. [18] observed that SGLT2i-treated patients had a smaller rise in serum creatinine and less decline in eGFR one day after PCI compared with non-users. Importantly, the incidence of CIN was substantially lower in the treated group (3.8% vs. 17.3%).

These findings were further supported by a meta-analysis involving 2,572 diabetic patients undergoing coronary intervention, of whom 512 received SGLT2 inhibitors. The pooled analysis demonstrated a 63% reduction in CIN risk among patients treated with SGLT2 inhibitors compared with non-users [1]

In ElSlalhy et al[19] Mohamady et al,.[20] Dapagliflozin therapy was associated with preservation of kidney function from contrasted agents during coronary angioplasty. The results suggested that SGLT2 inhibitors, particularly for cases with concomitant diseases like diabetes, may offer protection against the progression of CIN. Comorbid heart failure, higher baseline serum creatinine and non-use of Dapagliflozin significantly independently increased risk of CIN

On the other hand, Kültürsay et al. [21] the effect of SGLT2i therapy in diabetic patients with STEMI undergoing PCI. Initially, no significant difference in CIN incidence was observed between treated and untreated groups. However, after applying propensity score weighting, the analysis suggested a tendency toward reduced CIN risk among SGLT2i users.

Uunivariate regression analysis in our study found that the DAPA intake OR (95% CI) 0.299 (0.095-0.939) and (p=0.039), the pre-procedure eGFR OR (95% CI) 0.907 (0.855-0.962) and (p=0.001), and EF OR (95% CI) 0.913 (0.844-0.987) and (p=0.023) were significant protective factors against CIN, while DM OR (95% CI) 9.537 (1.191-76.367) and (p=0.034), HTN OR (95% CI) 5.667 (1.494-21.496) and (p=0.011), smoking OR (95% CI) 3.565 (1.181-10.764) and (p=0.024), PVD OR (95% CI) 4.071 (1.160-14.291) and (p=0.028) and the higher creatinine OR (95% CI) 237.0 (3.049-18417) and (p=0.014) were potential risk factors for CIN. While by multivariate regression analysis, Multivariate analysis confirmed that dapagliflozin therapy (adjusted OR 0.161 (0.039-0.666); p = 0.032) and DM (adjusted OR 9.794 (1.045-91.792); p = 0.046) and smoking (adjusted OR 8.017 (1.594-40.312); p = 0.012) and eGFR (adjusted OR 0.920 (0.855-0.991); p = 0.027) were independent predictors of CIN.

In contrast, diabetes mellitus as a significant risk factor for CIN Khan et al. [22] highlighted diabetic patients associated with higher CIN incidence following PCI compared with non-diabetics. These findings are in line with Tolba et al. [23], who similarly observed greater CIN risk among diabetic populations, in the current study findings also identified diabetes mellitus as a significant risk factor for CIN. Importantly,

prior research confirms that diabetes, particularly when associated with chronic kidney disease, markedly increases CIN susceptibility [24].

The current study, smoking emerged as a significant risk factor for CIN, a finding comparable to Hassan et al. [25] who noted smoking as the third most common comorbidity after hypertension and diabetes. Interestingly, Ye et al. [26] reported an opposite effect, where smokers with STEMI undergoing PCI were less likely to develop CIN compared with non-smokers, a paradoxical observation previously described in coronary artery disease as the "smoker's paradox."

The current study findings also demonstrated that preserved ejection fractions (EF) were protective against the development of CIN. This observation is in line with Xu et al. [27], who reported EF as an independent predictor of CIN in patients with heart failure. They noted a negative correlation between EF and CIN incidence, suggesting that when EF is ≥50%, the exact numerical value of EF becomes less clinically relevant in predicting CIN risk. Similarly, Wang et al. [28] that among 225 patients, 13.7% developed CIN, with lower left ventricular EF and advanced heart failure status (NYHA class >2 or Killip class >1) serving as independent predictors.

Conversely, other investigations have yielded conflicting results. Kurtul et al. [29] and Barbieri et al. [30] reported that the association between EF and CIN risk lost statistical significance. These discrepancies suggest that while EF may contribute to CIN susceptibility, its predictive value might vary depending on patient characteristics, underlying comorbidities, and methodological differences across studies.

The current study provides valuable insight into the potential renoprotective role of Dapagliflozin in patients undergoing PCI, highlighting its significantly associated lower CIN incidence. A particular strength is the homogeneity of the study population, as baseline demographic and clinical variables were well balanced between groups, allowing for a clearer assessment of Dapagliflozin's effect. In addition, renal function was systematically monitored at baseline and during follow-up, ensuring reliable detection of CIN events.

Nevertheless, several limitations should be acknowledged, including the relatively small sample size and single-center design may limit the external validity and generalizability of the findings. Second, patients with abnormal kidney function were excluded (for patient safety, to get two homogenous study group and there is a significant heterogeneity in renal function assessment methods: Cockcroft-Gault, MDRD , and CKD-EPI), which may have led to an underestimation of CIN incidence in a real-world setting where CKD is common among PCI candidates. Third, the observational nature of Dapagliflozin exposure prior to PCI may introduce selection bias, and causality cannot be definitively established. Finally, longer-term outcomes, including the impact on major adverse cardiovascular and renal events, were not assessed, and thus further large-scale randomized trials in multiple centers are warranted to answer that Dapagliflozin may be a part of therapy for the patient eligible for elective angiography or at high risk of developing CIN especially patients with concomitant indication for Dapagliflozin.

Conclusion

Dapagliflozin is significantly associated with lower CIN incidence in individuals with IHD undergoing elective PCI.

Conflict of Interest & Funding Statement: This work was completed independently, with no external funding, and the authors report no competing interests.

Data Availability: Requests for access to the underlying data that substantiate the results of this study may be directed to the corresponding author, who will consider providing them in accordance with institutional policies and applicable ethical guidelines.

Author Contributions: A.A.K. played a leading role in shaping the study design and critically reviewed the intellectual content

throughout the writing process. R.Y. offered substantial guidance in clinical interpretation and contributed to refining the final manuscript. W.F. provided expert input on cardiological perspectives and helped structure the methodology and analysis framework. E.M.E. was actively involved in data acquisition, statistical evaluation, and took the initiative in preparing the initial draft of the manuscript, ensuring coherence and clarity across all sections. All authors discussed the results, contributed to the manuscript's development, and approved the final version for submission.

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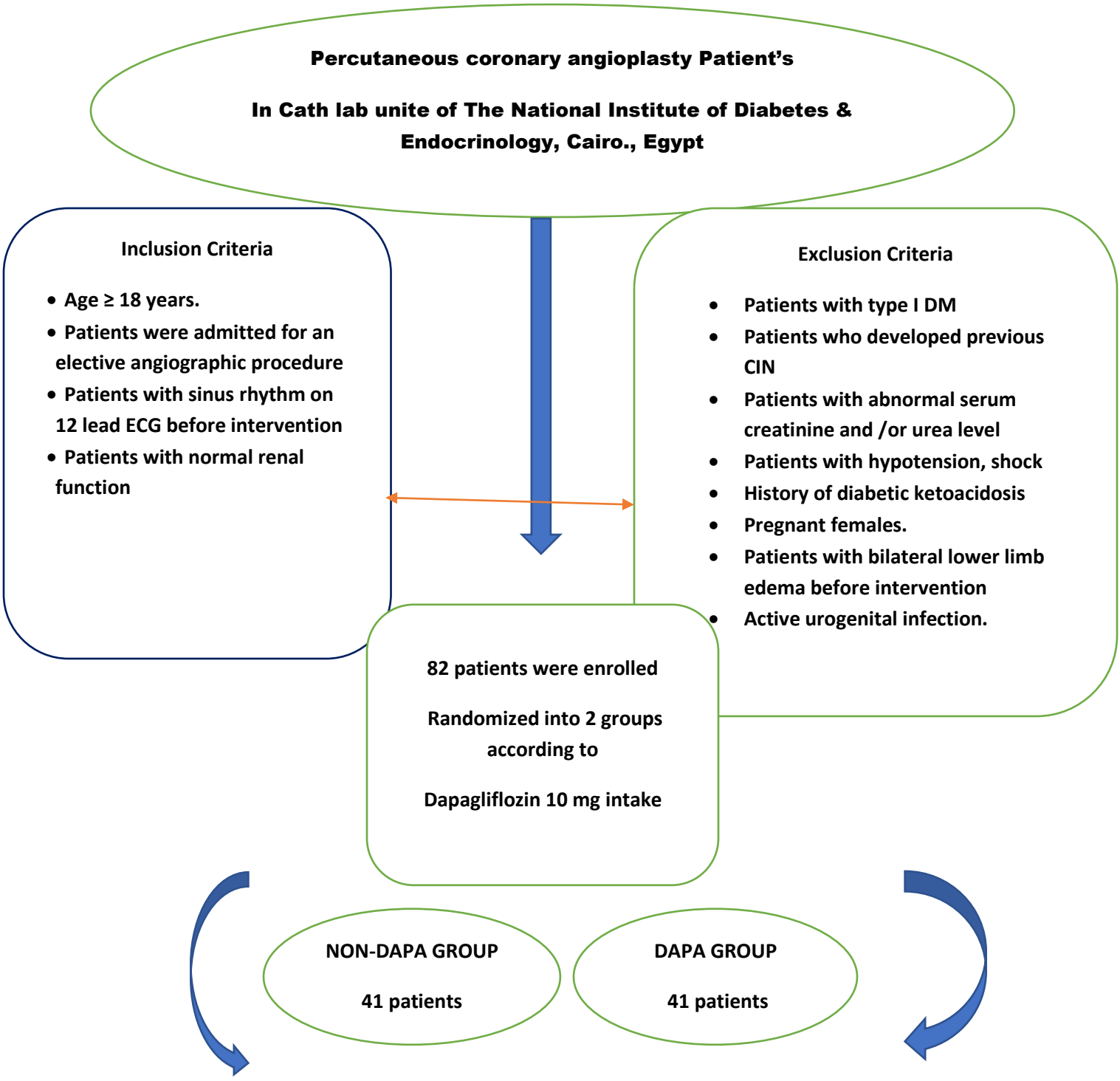

Supplementary Figure 1: Consort Flow Chart

Citation

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