Effect of Dapagliflozin on the Incidence of Contrast-Induced Nephropathy after Percutaneous Coronary Intervention in Patients with Type 2 Diabetes Mellitus: Postprint

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Abstract

Background Dapagliflozin is an effective medication for type 2 diabetes mellitus (T2DM) that also reduces the risk of T2DM nephropathy progression, decreases urinary protein, and provides cardioprotection; however, whether dapagliflozin can reduce the incidence of contrast-induced nephropathy (CIN) after percutaneous coronary intervention (PCI) in T2DM patients remains unclear. Objective To investigate the effect of dapagliflozin on the incidence of CIN after PCI in T2DM patients. Methods Based on dapagliflozin usage and following a 1:1 propensity matching principle, a total of 484 T2DM patients who underwent PCI in the Department of Cardiology at Tianjin Chest Hospital between January 2021 and December 2023 were retrospectively and consecutively enrolled, with 242 patients in the dapagliflozin group and 242 in the control group. Pre-PCI clinical data were collected and compared between the two groups, while renal function parameters including blood urea nitrogen (BUN), serum creatinine (Scr), creatinine clearance rate (Ccr), cystatin-C (Cys-C), $ 2 − *microglobulin*($2-MG), and neutrophil gelatinase-associated lipocalin (NGAL) were recorded before PCI, 48 hours after PCI, and 1 week after PCI. The primary endpoint was CIN incidence, and the secondary endpoint was perioperative renal function changes after PCI. Multivariate Logistic regression analysis was employed to evaluate the effect of dapagliflozin on CIN incidence after PCI in T2DM patients. Results The CIN incidence in the dapagliflozin group was 6.2%, significantly lower than the 12.0% incidence in the control group (2=4.900, P=0.039); the CIN risk score and B-type natriuretic peptide were higher in the dapagliflozin group than in the control group (P<0.05). Before PCI and 1 week after PCI, no statistically significant differences were observed in BUN, Scr, Ccr, Cys-C, $ $2-MG, or NGAL levels

between the two groups (P>0.05). At 48 hours after PCI, Cys-C, $ $2-MG, and NGAL levels in the dapagliflozin group were lower than those in the control group (P<0.05). Multivariate Logistic regression analysis revealed that a high CIN risk score (OR=1.213, 95%CI=1.085~1.358, P=0.001) and elevated B-type natriuretic peptide level (OR=0.338, 95%CI=1.479~10.494, P=0.006) were independent risk factors for CIN after PCI in T2DM patients, while dapagliflozin use (OR=0.338, 95%CI=0.159~0.717, P=0.005) was an independent protective factor. Conclusion Dapagliflozin use is an independent protective factor for CIN after PCI in T2DM patients, does not increase the risk of acute kidney injury (AKI) after PCI in T2DM patients, and may reduce the incidence of CIN.

Full Text

The Impact of Dapagliflozin on the Incidence of Contrast-Induced Nephropathy in Patients with Type 2 Diabetes Mellitus Underwent Percutaneous Coronary Intervention

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Abstract

Background: Dapagliflozin is an effective drug for treating type 2 diabetes mellitus (T2DM) that also reduces the risk of nephropathy progression, decreases urinary protein, and provides cardioprotective effects. However, whether dapagliflozin can reduce the incidence of contrast-induced nephropathy (CIN) after percutaneous coronary intervention (PCI) in T2DM patients remains unclear.

Objective: To investigate the impact of dapagliflozin on the incidence of CIN in T2DM patients undergoing PCI.

Methods: Using a 1:1 propensity matching principle based on dapagliflozin usage, we retrospectively and consecutively enrolled 484 T2DM patients who underwent PCI in the Department of Cardiology at Tianjin Chest Hospital between January 2021 and December 2023. The cohort consisted of 242 patients in the dapagliflozin group and 242 in the control group. We collected and compared pre-PCI clinical data for both groups and recorded renal function parameters before PCI, at 48 hours post-PCI, and at 1 week post-PCI, including blood urea nitrogen (BUN), serum creatinine (Scr), creatinine clearance rate (Ccr), cystatin C (Cys-C), β-2 microglobulin ($ $2-MG), and neutrophil gelatinase-associated lipocalin (NGAL). The primary endpoint was CIN incidence, while the secondary endpoint was perioperative renal function changes. Multivariate

logistic regression was used to analyze the effect of dapagliflozin on post-PCI CIN development in T2DM patients.

Results: The CIN incidence in the dapagliflozin group was 6.2%, significantly lower than the 12.0% observed in the control group (^{2} = 4.900, P = 0.039). The dapagliflozin group also had higher CIN risk scores and B-type natriuretic peptide levels compared to the control group (P < 0.05). No statistically significant differences were found between the two groups in BUN, Scr, Ccr, Cys-C, $ $2-MG, or NGAL levels before PCI or at 1 week post-PCI (P > 0.05). However, at 48 hours post-PCI, the dapagliflozin group showed significantly lower levels of Cys-C, $ $2-MG, and NGAL compared to the control group (P < 0.05). Multivariate logistic regression analysis revealed that a high CIN risk score (OR = 1.213, 95%CI = 1.085–1.358, P = 0.001) and elevated B-type natriuretic peptide levels (OR = 3.943, 95%CI = 1.479–10.494, P = 0.006) were independent risk factors for post-PCI CIN in T2DM patients, while dapagliflozin use (OR = 0.338, 95%CI = 0.159–0.717, P = 0.005) was an independent protective factor.

Conclusion: Dapagliflozin use is an independent protective factor against CIN development after PCI in T2DM patients. Dapagliflozin does not increase the risk of acute kidney injury (AKI) after PCI in T2DM patients and may reduce CIN incidence.

Keywords: Diabetes mellitus, type 2; Acute kidney injury; Dapagliflozin; Contrast materials; Percutaneous coronary intervention; Root cause analysis

Introduction

Contrast-induced nephropathy (CIN) is a common form of acquired acute kidney injury (AKI) that occurs following imaging examinations or percutaneous coronary intervention (PCI) using iodinated contrast agents. In 2012, the Kidney Disease: Improving Global Outcomes (KDIGO) organization defined CIN as an absolute increase in serum creatinine (Scr) of 26.5 μmol/L within 48 hours of contrast administration, or a relative increase exceeding 50% within one week [1]. CIN incidence varies depending on patients' underlying comorbidities, occurring in approximately 2% of low-risk patients but reaching 25–50% in high-risk populations such as the elderly, patients with type 2 diabetes mellitus (T2DM), and those with chronic kidney disease. CIN not only prolongs hospital stays and increases medical costs but is also associated with poor prognosis [2]. Currently, apart from hydration therapy, minimizing contrast dosage, and using low-osmolar or iso-osmolar non-ionic contrast agents, no specific effective prevention or treatment methods exist for CIN [1].

Recent clinical evidence has demonstrated that dapagliflozin not only effectively reduces blood glucose in T2DM patients but also improves renal function, decreases the risk of nephropathy progression, reduces urinary protein, and provides cardioprotective benefits, offering cardiorenal protection for T2DM pa-

tients [3]. However, early case reports suggested that dapagliflozin might increase AKI risk due to its diuretic effects, blood pressure reduction, and decreased blood volume, which could lead to reduced glomerular filtration rate. Consequently, some researchers have recommended discontinuing dapagliflozin during the peri-PCI period to reduce CIN risk [4]. Nevertheless, the impact of dapagliflozin on CIN incidence in T2DM patients after PCI remains incompletely understood. Therefore, this retrospective study aimed to investigate the effect of dapagliflozin on CIN incidence in T2DM patients undergoing PCI.

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1. Subjects and Methods

1.1 Study Subjects Based on dapagliflozin usage and using a 1:1 propensity matching principle, we retrospectively and consecutively enrolled 484 T2DM patients who underwent PCI in the Department of Cardiology at Tianjin Chest Hospital between January 2021 and December 2023. The cohort comprised 242 patients in the dapagliflozin group and 242 in the control group. Assuming a CIN incidence of 5% in the dapagliflozin group and 15% in the control group, with $\alpha = 0.05$ and power $= 0.95$, the estimated sample size was at least 227 patients per group, which our study satisfied.

Inclusion criteria: (1) Met the diagnostic criteria for T2DM in the *Guidelines for the Prevention and Treatment of Type 2 Diabetes in China (2020 Edition)* [5]: classic diabetes symptoms plus random blood glucose $\geq$ 11.1 mmol/L, or fasting blood glucose $\geq$ 7.0 mmol/L, or 2-hour oral glucose tolerance test $\geq$ 11.1 mmol/L, or glycated hemoglobin $\geq$ 6.5%; for patients without classic symptoms, confirmation on a subsequent day was required. (2) Met the PCI treatment indications in the *Guidelines for the Diagnosis and Treatment of Stable Coronary Artery Disease* [6].

Exclusion criteria: (1) Contrast agent or dapagliflozin allergy; (2) Acute or chronic infection or active bleeding; (3) Cardiogenic shock, systolic blood pressure $<$ 90 mmHg, severe heart failure, or left ventricular ejection fraction (LVEF) $<$ 30%; (4) Renal insufficiency with estimated glomerular filtration rate (eGFR) $<$ 60 $\mathrm{mL \cdot min^{-1} \cdot (1.73\ m^2)^{-1}}$ or requiring dialysis; (5) Hyperthyroidism or malignant tumors.

This study was approved by the Medical Ethics Committee of Tianjin Chest Hospital, Tianjin University (2021YS-031-01), with a waiver of informed consent.

1.2 Research Methods To minimize CIN risk, all patients received hydration therapy with 0.9% sodium chloride at 1 $\mathrm{mL \cdot kg^{-1} \cdot h^{-1}}$ for 6–12 hours before and after PCI; for patients with LVEF $<$ 45%, hydration was administered at 0.5 $\mathrm{mL \cdot kg^{-1} \cdot h^{-1}}$. Patients were also encouraged to drink plenty of water. All patients received the iso-osmolar contrast agent iodixanol (Jiangsu Hengrui Pharmaceuticals Co., Ltd., batch number: H200203DJ). The dapagliflozin

group received oral dapagliflozin (AstraZeneca Pharmaceuticals Co., Ltd., batch number: H20170119) 10 mg once daily for at least 4 weeks before PCI and continued postoperatively.

CIN risk was assessed using the risk scoring system proposed by Mehran et al. [7]: hypotension (systolic blood pressure < 90 mmHg, 5 points); intra-aortic balloon pump use (5 points); severe heart failure (NYHA class III/IV) or pulmonary edema (5 points); age ≥ 75 years (4 points); anemia (baseline hematocrit: female $< 36\%$, male $< 39\%$, 3 points); T2DM (3 points); contrast dose (1 point per 100 mL); eGFR ≤ 60 mL $\cdot$ min^{-1} $\cdot$ (1.73 m^2)$^{-1}$ [eGFR 40–60, 20–40, < 20 mL $\cdot$ min^{-1} $\cdot$ (1.73 m^2)$^{-1}$ scored 2, 4, and 6 points, respectively]. Risk categories were low (≤ 5 points), intermediate (6–10 points), high (11–15 points), and very high (≥ 16 points).

1.3 Data Collection We collected baseline demographic data, smoking and alcohol consumption habits, medical history, laboratory parameters, echocardiographic findings, PCI details (number of target vessels and stents implanted), medication history, contrast volume, and hydration volume. Smoking was defined as ≥ 1 cigarette per day for > 6 months; those who never smoked or smoked less were classified as non-smokers. Alcohol consumption was defined as ≥ 1 occasion per week for > 6 months; those who never drank or drank less were classified as non-drinkers [8].

Renal function parameters were recorded before PCI, at 48 hours post-PCI, and at 1 week post-PCI, including blood urea nitrogen (BUN), Scr, creatinine clearance (Ccr), cystatin C (Cys-C), $\$ 2-microglobulin($ \$2-MG), and neutrophil gelatinase-associated lipocalin (NGAL). Ccr was calculated as follows: (1) For males: Ccr = (140 -age) $\times$ weight (kg) $\times$ 88.4 / [72 $\times$ Scr (µmol/L)]; (2) For females: Ccr = (140 -age) $\times$ weight (kg) $\times$ 88.4 / [72 $\times$ Scr (µmol/L)] $\times$ 0.85 [9].

1.4 Endpoint Events The primary endpoint was CIN incidence, defined as an Scr increase > 26.5 µmol/L (0.3 mg/dL) within 48 hours of contrast administration, or a $> 50\%$ increase from baseline within one week [1]. The secondary endpoint was changes in renal function at 48 hours and 1 week after PCI.

1.5 Statistical Methods Data were analyzed using SPSS 19.0 software. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation and compared between groups using independent samples t-tests. Categorical variables were expressed as frequencies (percentages) and compared using ^{2} tests. Multivariate logistic regression was used to analyze the effect of dapagliflozin on post-PCI CIN in T2DM patients. Statistical significance was set at $P < 0.05$.

2. Results

2.1 CIN Incidence and Baseline Characteristics Among the 484 patients (284 males [58.7%] and 200 females [41.3%]), the mean age was 73.8 ± 10.6 years, and 44 patients (9.1%) developed CIN. The dapagliflozin group had a significantly lower CIN incidence than the control group (6.2% vs. 12.0%, P = 0.039) and higher CIN risk scores and B-type natriuretic peptide levels (P < 0.05). No significant differences were observed between the two groups in other baseline indicators (P > 0.05), as shown in Table 1 .

2.2 Changes in Renal Function Indicators Before and After PCI Before PCI, no significant differences were found between the two groups in BUN, Scr, Ccr, Cys-C, $ $2-MG, or NGAL levels (P > 0.05). At 48 hours post-PCI, the groups showed no significant differences in BUN, Scr, or Ccr levels (P > 0.05); however, the dapagliflozin group had significantly lower Cys-C, $ $2-MG, and NGAL levels compared to the control group (P < 0.05). At 1 week post-PCI, no significant differences were observed between the groups in any renal function parameters (P > 0.05), as detailed in Table 2 .

2.3 Multivariate Logistic Regression Analysis of Factors Influencing Post-PCI CIN in T2DM Patients Using CIN occurrence (yes = 1, no = 0) as the dependent variable and factors potentially influencing CIN from Table 1 (P < 0.1) as independent variables—including diabetes duration, CIN risk score, B-type natriuretic peptide, fasting blood glucose, glycated hemoglobin (actual values), diuretic use (yes = 1, no = 0), and dapagliflozin use (yes = 1, no = 0)—multivariate logistic regression analysis revealed that a high CIN risk score (OR = 1.213, 95%CI = 1.085–1.358, P = 0.001) and elevated B-type natriuretic peptide levels (OR = 3.943, 95%CI = 1.479–10.494, P = 0.006) were independent risk factors for post-PCI CIN in T2DM patients, while dapagliflozin use (OR = 0.338, 95%CI = 0.159–0.717, P = 0.005) was an independent protective factor, as shown in Table 3 .

Discussion

Traditional renal function indicators such as BUN, Scr, and Ccr are commonly used in clinical practice but are susceptible to various factors including diet, body weight, gender, and age, limiting their sensitivity and timeliness for early AKI diagnosis. Cys-C is filtered solely by the glomerulus and completely metabolized after reabsorption in the proximal tubule, making it unaffected by external factors and an ideal endogenous marker of glomerular filtration rate changes. Meanwhile, $ $2-MG and NGAL can be highly expressed and released into the bloodstream during early AKI, making Cys-C, $ $2-MG, and NGAL sensitive biomarkers for early CIN detection [10]. In this study, BUN, Scr, and Ccr showed no significant changes from baseline at 48 hours post-PCI, while

Cys-C, $ $2-MG, and NGAL levels increased in both groups, likely reflecting their superior sensitivity for detecting early renal injury.

The pathogenesis of CIN may involve renal artery vasoconstriction following contrast administration, leading to an imbalance between renal vasodilatory and vasoconstrictive factors. Contrast-induced nephrotoxic injury causes renal cortical and medullary ischemia and hypoxia, triggering inflammatory responses and oxidative stress damage through reactive oxygen species, ultimately resulting in CIN [11]. Therefore, anti-inflammatory effects and reduced oxidative stress represent potential therapeutic strategies for CIN prevention. Dapagliflozin, a sodium-glucose cotransporter 2 inhibitor (SGLT2i), effectively inhibits glucose reabsorption in renal tubules, promoting urinary glucose excretion and thereby reducing blood glucose in T2DM patients. Recent studies have demonstrated that dapagliflozin improves renal function and delays nephropathy progression in T2DM through anti-fibrotic, anti-inflammatory, and antioxidant stress effects, as well as by improving renal cortical and medullary ischemia-hypoxia [12]. Based on these mechanisms, dapagliflozin may offer potential benefits in reducing CIN incidence.

However, some studies have reported concerns that dapagliflozin might increase AKI risk by promoting glucosuria-induced diuresis, dehydration, and blood pressure reduction, which could decrease renal blood volume and trigger tubuloglomerular feedback-mediated afferent arteriolar constriction, ultimately reducing glomerular filtration rate—particularly in patients with renal insufficiency or volume depletion [13]. Consequently, some researchers have recommended discontinuing dapagliflozin during the peri-PCI period to minimize CIN risk. Nevertheless, accumulating clinical data indicate that dapagliflozin not only fails to increase AKI risk but actually reduces its incidence, demonstrating favorable renal safety. The WIVIOTT et al. study [14] showed that AKI incidence was lower in the dapagliflozin group compared to placebo (1.5% vs. 2.0%, HR = 0.69, 95%CI = 0.55–0.87). Real-world data analysis by CAHN et al. [15] demonstrated that prophylactic dapagliflozin use reduced AKI risk by up to 74% (OR = 0.47, 95%CI = 0.27–0.80). A large meta-analysis of 112 randomized controlled trials and 5 observational cohort studies revealed that dapagliflozin reduced AKI risk by 36% [16].

Evidence suggests that SGLT2i treatment causes a transient Ccr decline during the first 1–4 weeks, with Ccr levels normalizing after 4 weeks of use [17]. Therefore, this study required patients to have used dapagliflozin for at least 4 weeks before PCI while ensuring adequate hydration and encouraging fluid intake during the perioperative period to promote contrast excretion. Our results showed that the dapagliflozin group had significantly lower CIN incidence than the control group, with lower Cys-C, $ $2-MG, and NGAL levels at 48 hours post-PCI —all sensitive markers of AKI. Multivariate logistic regression confirmed that dapagliflozin use was an independent protective factor against post-PCI CIN in T2DM patients, suggesting that with adequate hydration, dapagliflozin may not increase AKI risk but rather reduce CIN incidence, offering potential renal

protection for T2DM patients undergoing PCI.

These findings are supported by PAOLISSO et al. [18], who included 646 T2DM patients with acute myocardial infarction and found that SGLT2i users had significantly lower CIN incidence than non-users (5.4% vs. 13.1%, P = 0.022), with multivariate analysis identifying SGLT2i use as an independent predictor of reduced CIN (OR = 0.356, 95%CI = 0.134-0.943, P = 0.038). Similarly, HUA et al. [19] conducted a propensity-matched analysis comparing SGLT2i users and non-users, finding CIN incidence of 4.1% in SGLT2i users versus 9.1% in controls, with significantly lower post-PCI Scr and Cys-C levels in the SGLT2i group. These data collectively demonstrate that dapagliflozin effectively improves renal function in T2DM patients after PCI, supporting that discontinuation during the peri-PCI period is unnecessary. However, whether short-term dapagliflozin use during the perioperative period effectively reduces CIN incidence requires further investigation.

This study has several limitations. First, as a single-center retrospective cohort study with a limited sample size, further research with larger datasets is needed to provide more comprehensive clinical and theoretical evidence. Second, this study cannot address whether other SGLT2i agents besides dapagliflozin have similar CIN-reducing effects. Third, due to variability in dapagliflozin treatment duration among patients, the optimal dosage and duration for CIN prevention cannot be specifically defined. Finally, the exact mechanisms by which dapagliflozin reduces CIN incidence were not elucidated. Therefore, the precise impact of dapagliflozin on post-PCI CIN in T2DM patients and the optimal dosing and timing for CIN prevention require clarification in future studies.

In summary, although numerous previous studies have proposed various CIN prevention methods with controversial results, our findings indicate that dapagliflozin—a widely used antihyperglycemic agent in T2DM patients—not only fails to increase post-PCI AKI risk but may actually reduce CIN incidence, representing a potential protective factor against CIN in this patient population.

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Author Contributions: LIU Xiaogang conceptualized the study, collected and organized data, and drafted the manuscript; YANG Shicheng organized data, provided statistical design guidance, and performed statistical calculations; FU Naikuan managed the project, provided guidance, and supervised and reviewed the manuscript; SHAO Dujing collected and organized data; ZHANG Peng collected and organized data and assisted with manuscript revision.

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