The Association between SGLT2 Inhibitors Use and the Risk of Contrast Induced Acute Kidney Injury in Patients with Type 2 Diabetes

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Abstract

Aim: Contrast-induced acute kidney injury (CI-AKI) is associated with increased risk of morbidity and mortality and specific treatments are needed. In our study, we aimed to investigate the association of sodium-glucose cotransporter-2 inhibitors (SGLT2-I) use with the development of CI-AKI due to coronary angiography (CAG) in patients with DM.

Methods: A total of 516 type 2 diabetes mellitus (DM) patients, including 250 SGLT2-I users and 266 non-SGLT2-I users, were included in our retrospective and cross-sectional study.

Results: The study groups were divided into CI-AKI (+) and CI-AKI (-). SGLT2-I use was statistically significantly higher in the CI-AKI (-) group. Multivariate logistic regression analysis showed that SGLT2-I use reduced the probability of CI-AKI by 83.8%.

Conclusions: Our findings suggest that SGLT2-Is may significantly reduce the risk of CI-AKI in diabetic patients undergoing CAG. In conclusion, the use of SGLT2-I may have a protective and preventive effect against the development of CI-AKI.

Keywords: *Contrast-induced acute kidney injury; coronary angiography; diabetes mellitus; percutaneous coronary intervention; sodium-glucose cotransporter-2 inhibitors*

1. Introduction

Percutaneous coronary intervention (PCI) is a successful treatment option for patients with coronary artery disease. In addition to its proven benefits, this procedure can also lead to some adverse events. Contrast-induced acute kidney injury (CI-AKI) is a type of acute kidney injury that occurs after the administration of contrast media for elective coronary angiography (CAG). This condition is characterised by an increase in serum creatinine levels ≥ 0.5 mg/dl or a 25% increase from baseline 48 hours after contrast media administration. The incidence ranges from 1.3% to 33.3% and is the third most important cause of hospital-acquired renal failure. CI-AKI is closely associated with longer hospital stays, more frequent readmissions and increased risk of short- and long-term morbidity and mortality. Type 2 diabetes mellitus (DM) is one of the risk factors for CI-AKI.[1,2]

Several mechanisms have been demonstrated for renal impairment after contrast media administration. The cytotoxicity of contrast media triggers apoptosis and necrosis of tubular and endothelial renal cells. The concentration and duration of the contrast medium play an important role in this process. After contrast media administration, ischaemia and hypoxic changes occur due to renal haemodynamic disturbances. Inflammatory processes and oxidative stress play a role in the deterioration of renal function.[3,4]

Although various approaches including hydration, N-acetylcysteine, sodium bicarbonate and statins have been investigated to prevent CI-AKI, the optimum approach has not yet been clearly determined.[5,6] Sodium-glucose cotransporter-2 inhibitors (SGLT2-I) are a new class of oral anti-diabetic agents.

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SGLT2-Is act by inhibiting renal reabsorption of glucose, increasing renal glucose excretion and reducing serum glycaemic levels. These drugs reduce blood pressure, the risk of cardiovascular events and kidney disease in patients with and without diabetes.[7,8] Two randomised controlled trials, the DAPA-CKD[9] and EMPA-Kidney[10] studies, have shown that in patients with chronic kidney disease, regardless of the presence or absence of diabetes, gliflozins lead to a lower risk of chronic kidney disease progression and death from renal or cardiovascular events compared with placebo. SGLT2-Is are thought to preserve renal function by inhibiting inflammation and fibrosis, reducing oxidative stress and improving tubular oxygen content. These mechanisms ultimately delay the decline in renal function and slow the progression of proteinuria.[11,12]

In recent years, several studies have been conducted to evaluate the effect of SGLT2-Is on contrast-induced nephropathy. Recent observational studies have suggested that SGLT2-Is may prevent CI-AKI in patients with diabetes undergoing coronary interventions such as CAG and PCI.[13,14] However, there are also studies that have not shown a clear nephroprotective effect of SGLT2-Is after PCI.[15] To our knowledge, there are few clinical studies focused on this issue. Since specific treatments for CI-AKI are still needed in the clinic, it is necessary to find a new treatment strategy that is an important complement to the current clinical management protocol. In our study, we aimed to investigate the association of SGLT2-I use with the development of CI-AKI due to PCI and CAG in patients with DM.

2. Materials and Methods

2.1. Study Population and Laboratory Measurements

A total of 516 type 2 DM patients, 250 of whom were using SGLT-2 inhibitors and 266 of whom were not using SGLT2-I, were included in our retrospective and cross-sectional study, and whose medical history and previous examinations did not prevent their inclusion in the study, and who were scheduled for CAG due to elective indications. Patients using SGLT2 for 6 months or more were included in the study. Patients between 01.01.2023 and 31.12.2024 were included in the study. Patients using dapagliflozin 10 mg or empagliflozin 10 mg were included in the study. Diabetes mellitus was defined as fasting blood sugar level ≥126 mg/dL or HbA1c level ≥6.5% or treatment with antidiabetic medication [16]. CI-AKI was defined as a 0.5 mg/dL increase in serum creatinine concentration 48 hours after contrast medium administration or a 25% increase in creatinine level from baseline [2].

Patients received standard hydration with intravenous infusion of 0.9% saline at a rate of 1 mL/kg/h for 12 hours before and 12 hours after the procedure. Patients who were administered other anti-CI-AKI drugs such as N-acetylcysteine and sodium bicarbonate were excluded from the study. Patients who received the same contrast material during coronary angiography were included in the study. In patients with unstable angina pectoris, myocardial infarction, chronic kidney disease, heart failure, malignancy, pregnant women were excluded from the study. The study was conducted in accordance with the Declaration of Helsinki and was approved by the institutional ethics committee. Adana City Training and Research Hospital Ethics Committee approved the study with decision number 2231 dated 03.11.2022. After 5 minutes of rest, in a dim and quiet environment, blood pressure measurements were taken from both arms using a suitable cuff and pulses were monitored. Anthropometric body weight measurements were performed. Height was measured with the feet bare and together, leaning perpendicular to the height measurement ruler. BMI was calculated as body weight (kg) divided by the square of height in meters (BMI=kg/m2). Laboratory procedures of the study were performed in the Biochemistry Laboratory of Adana City Training and Research Hospital. Venous blood was drawn from the antecubital vein after at least 8 hours of overnight fasting from the patients and the control group during routine controls. Laboratory measurements of participants were measured using automated laboratory methods (Abbott Aeroset, Minneapolis, MN) and appropriate commercial kits (Abbott).

2.2. Statistical analysis

We used SPSS 24.0 (Chicago, IL, USA) for all of our statistical analysis. We checked for normality in the distribution of continuous variables using the Kolmogorov-Smirnov test. The mean ± standard deviation was used to express continuous variables in the group data. The numerical and percentage values for the categorical variables were used. When comparing groups with normally distributed continuous variables, either Student's t-test or one way anova were employed. When comparing continuous variables that did not follow a normal distribution, the Mann-Whitney U test was employed. To compare categorical variables, the chi-square ($\chi 2$) test was employed. We identified the independent variables that have an effect on CI-AKI. Statistically significant parameters with a p value less than 0.05 were used in the multivariate logistic regression analysis that followed the univariate model in the independent determination of patients with CI-AKI. Statistical significance level was accepted as $p<0.05$.

3. Results

The study groups were divided into CI-AKI (+) and CI-AKI (-). There were 37 patients with CI-AKI (+) and 479 patients with CI-AKI (-). When both groups were compared according to demographic, clinical and laboratory findings; age, duration of DM, BMI, LDL level, total procedure time and amount of contrast medium were found to be statistically significantly higher in the CI-AKI (+) group. SGLT2-I utilisation was statistically significantly higher in the CI-AKI (-) group (table 1).

Patients with CI-AKI (-) were divided into three groups according to SGLT2-I use: SGLT2-I non-users, dapagliflozin users and empagliflozin users. When the groups were compared, it was found that the rate of CI-AKI (-) was higher in the group receiving dapagliflozin and empagliflozin compared to the group not using SGLT2-I. No statistically significant difference was found between the groups receiving empagliflozin and dapagliflozin in terms of CI-AKI (-) rate (table 2).

To determine whether the patients were CI-AKI (+), multivariate logistic regression analysis was performed using parameters with a p value less than 0.05 and shown to be statistically significant in univariate analysis. According to multivariate logistic regression analysis, SGLT2-I use reduced the probability of CI-AKI by 83.8%. Each 1 unit increase in BMI increased the probability of CI-AKI by 12% (table 3).

4. Discussion

In our study, we aimed to evaluate the effect of SGLT2-I use on the risk of CI-AKI formation after elective CAG in patients with type 2 DM. The main findings of our study were that SGLT2-I use was higher in the CI-AKI (-) group than in the CI-AKI (+) group and SGLT2- I use reduced the probability of CI-AKI by 83.8%. The reno-protective effect of SGLT2-I drugs in patients with type 2 DM is known. However, data on the effect of SGLT2-Is on CI-AKI in patients undergoing CAG are limited. SGLT2-Is, which are used in patients with type 2 DM and have known renoprotective properties, may also have favourable effects on the development of CI-AKI after CAG.

Table 1

Demographic and descriptive data of the groups

Variables	CI-AKI (+) n=37	CI-AKI (-) n=479	p
Gender (F/M), n	16/21	233/246	0.532
Age (year)	60.38±10.93	53.64±14.36	0.001
Diabetes mellitus duration (year)	3.3±1.91	2.7±1.65	0.036
Body mass index (kg/m2)	29.3±5.8	26.14±5.96	0.002
Systolic blood pressure (mmHg)	128.35±10.49	128.2±11.35	0.939
Diastolic blood pressure (mmHg)	89.92±5.2	90.26±5.12	0.694
Smoking, n	18(48.6%)	237(49.5%)	0.923
Hypertension, n	16(43.2%)	218(45.5%)	0.79
ACEi/ ARB use, n	13(35.1%)	182(38%)	0.73
Beta bloker use, n	4(10.8%)	68(14.2%)	0.568
Calcium channel blockers use, n	3(8.1%)	36(7.5%)	0.896
Statin use, n	14(37.8%)	169(35.3%)	0.755
SGLT2-I use, n	8(21.6%)	242(50.5%)	<0.001
White blood cell (10^3/ µL)	8.14±2.7	8.39±2.6	0.589
HbA1c, %	7.67±1.1	7.36±1.48	0.116
Red blood cell (106/ µL)	4.3±0.94	4.25±0.78	0.745
Hemoglobin(g/dL)	12.07±2.12	11.94±2.06	0.728
Platelet (10^3/ µL)	270.72±69.6	284.48±101.1	0.423
Fasting plasma glucose (mg/dL)	168.35±54.67	157.77±55.33	0.263
Thyroid stimulating hormone (mIU/L)	2.04±1.9	1.74±1.23	0.447
Blood Urea Nitrogen (mg/dL)	24.89±4.78	24.74±3.81	0.854
Creatinine (mg/dL)	0.8±0.13	0.77±0.18	0.176
Albumin (g/dl)	3.88±0.54	3.8±0.56	0.465
Total protein (g/dL)	6.91±0.71	6.88±0.61	0.826
C-reactive protein (mg/L)	0.75±0.75	1.06±1.05	0.167
Sodium (mmol/L)	136.74±5.07	136.2±4.31	0.474
Potassium (mmol/L)	4.4±0.69	4.53±0.66	0.272
Uric acid (mg/dL)	5.16±1.68	5.69±2.05	0.169
Triglycerides (mg/dL)	161.91±65.13	154.64±56.34	0.487
High-density lipoprotein (mg/dL)	28.76±9.7	46.63±43.48	0.023
Low-density lipoprotein (mg/dL)	149.39±24.54	124.36±37.17	<0.001
Alanine aminotransferase (u/L)	15.3±10.01	16.9±9.96	0.352
Alanine aminotransferase (u/L)	19.47±5.67	20.96±8.22	0.347
Calcium (mg/dL)	9.07±0.62	8.92±0.66	0.193
25-hydroxyvitamin D (ng/mL)	13.01±4.09	17.55±13.52	0.319
eGFR (mL/min/1.73 m2)	90±0.56	89.88±2.05	0.728
The total processing time (min)	24.16±4.66	18.82±3.08	<0.001
Amount of contrast (cc)	106.75±15.1	87.20±7.70	<0.001

CI-AKI: Contrast-induced Acute Kidney Injury, DM: Diabetes mellitus, eGFR: estimated glomerular filtration rate, HbA1c: glycated haemoglobin, ACEI/ARB, angiotensin converting enzyme inhibitors/angiotensin receptor blocker, SGLT2-I: sodium glucose cotransporter 2 inhibitors

CI-AKI has become one of the leading causes of iatrogenic kidney injury, which is closely associated with poorer prognosis and higher overall healthcare costs in patients. Common risk factors of CI-AKI have been reported to include traditional risk factors (age, chronic renal failure, heart failure and DM) and factors such as dosage and types of contrast agents, multiple operations within a short period of time. Therefore, patients with DM are at higher risk of developing CI-AKI after an elective CAG and should be given special attention.[17] The mechanisms underlying how contrast media induce CI-AKI and impair renal function encompass a variety of pathogenic processes that are not fully understood. Three main mechanisms such as medullary hypoxia, oxidative stress and inflammation are reported to be involved simultaneously.[18] Recent studies suggest that SGLT2-Is play an important role beyond glucose regulation, including inhibition of inflammation, oxidative stress reduction and prevention of medullary hypoxia.[19] These findings increase interest in studying the effects of this class of drugs on renal function tests in patients with cardiovascular diseases.

DECLARE-TIMI, DAPA-CKD and EMPA-KIDNEY studies demonstrated the renoprotective effect of dapagliflozin and empagliflozin in patients with chronic renal failure regardless of the presence or absence of diabetes.[7,9,10] In a study conducted in type 2 DM patients,

Table 2

Evaluation of patients with CI-AKI (-) according to SGLT2-I use

Variable	SGLT2-I (-) n=266	Dapagliflozin use n=139	Empagliflozin use n=111	p
CI-AKI (-)	237 (89.0%) α, β	135 (97.1%)	107 (96.4%)	0.003

CI-AKI: Contrast-induced Acute Kidney Injury, SGLT2-I: sodium glucose cotransporter 2 inhibitors, α: Significant association between the SGLT2-I (-) group and Dapagliflozin group (p<0.05), β: Significant association between the SGLT2-I (-) group and Empagliflozin group (p<0.05)

Table 3

Multivariate logistic regression analysis for detection of patients with CI-AKI (-)

Variables	Odds Ratio	95 % Confidence Interval	p
Age (year)	1.042	0.980-1.108	0.193
Diabetes mellitus duration (year)	1.215	0.895-1.649	0.211
Body mass index (kg/m2)	1.120	1.034-1.212	0.005
SGLT2-I use	0.162	0.044-0.596	0.006

SGLT2-I: sodium glucose cotransporter 2 inhibitors, CI-AKI: Contrast-induced Acute Kidney Injury

Nadkarni et al. aimed to compare the risk of AKI in relation to SGLT2-I with non- users and did not observe an increased risk of AKI in DM patients receiving SGLT2-I compared to non-users during the 18-month follow-up period. Moreover, the study reported that the risk of AKI decreased in the group treated with SGLT2-I.[20] In an in vivo study conducted in a rat model, Huang et al. reported the potential molecular mechanism by which dapagliflozin may reduce CI-AKI by regulating the HIF-1a/HE4/NF-kB pathway.[21] Huang et al. found that the risk of CI-AKI was reduced in patients with DM who used SGLT2-I.[22] Liu et al. showed that dapagliflozin effectively reduced the risk of CI-AKI and showed renoprotective effects in patients with type 2 DM and chronic renal failure undergoing elective CAG.[17] In their randomised controlled study, Hosseini et al. reported that empagliflozin significantly reduced the incidence of CI-AKI in patients undergoing PCI by improving renal function parameters such as eGFR and cystatin C.[23] In a meta-analysis performed by Rodriguez et al., it was shown that the use of SGLT2-I significantly reduced the risk of CI-AKI by up to 63% in diabetic patients undergoing CAG or PCI.[24] In a study conducted by Özkan et al. in DM patients with non-ST segment elevation myocardial infarction, SGLT2-I use may be protective against the development of CI-AKI, especially in patients with comorbid conditions such as DM.[14]

In our study, we found that the use of SGLT2-I may be protective against the development of CI-AKI. We found that the use of SGLT2-I reduced the probability of CI-AKI by 83.8%. SGLT2-I may reduce renal oxygen consumption by directly inhibiting SGLT2- mediated sodium reabsorption in the proximal tubule and alleviate renal hypoxia by increasing blood ketone levels to provide more substrates for mitochondrial energy metabolism. On the other hand, it may exert renoprotective effect by regulating tubuloglomerular feedback responses and vasodilatation of afferent arterioles by improving glomerular hyperfiltration. In addition, SGLT-2-I reduces the energy requirement of the nephron. One of the reasons why the development of CI-AKI was significantly less in patients receiving SGLT2-I in our study may be that it reduces the energy required in the renal system, makes energy utilisation more efficient and alleviates renal hypoxia. One of the mechanisms leading to CI-AKI is a reduction in renal plasma flow to the outer medulla. This ischaemia is due to an imbalance between renal vasodilatory and vasoconstrictive factors. SGLT2-I may help to correct volume loss and expand the intravenous volume by causing natriuresis, glycosuria and subsequent diuretic action. This increases the clearance of contrast media, decreases the concentration of contrast media in the tubule lumen and vasa recta and may prevent the activation of neurohormonal systems leading to medullary vasoconstriction.[14,22] In our study, there was no difference in the use of ras blockers and statins between the groups with and without CI-AKI. The fact that the use of ras blockers, which are known to slow down diabetic kidney disease progression, and statins, which have studies on preventing CI-AKI, were similar in both groups is important in showing that the use of SGLT2-I provides an additional contribution. Phase studies show that the use of SGLT2-I treatment for more than 6 months may reveal beneficial effects of cardiac and renal protection in patients with DM. Therefore, we used 6 months of SGLT2-I use as the inclusion cut-off for participants in this study. The effect of shorter duration of SGLT2-I use on the incidence of CI-AKI needs to be evaluated in future studies. We found no difference in the development of CI-AKI with the use of empagliflozin or dapagliflozin among SGLT2-Is. This suggests that SGLT2-Is may be effective in the development of CI-AKI as a group effect. In our study, we found that LDL levels were higher in the CI-AKI group than in the non-CI-AKI group. High LDL may aggravate the CI-AKI condition by increasing atherosclerosis formation, mitochondrial damage and mitochondrial oxidative stress.

Risk factors for the development of CI-AKI include age, chronic renal failure, heart failure, diabetes, obesity, dosage of contrast agents and duration of the procedure. As the duration of diabetes increases, vascular smooth muscle cell functions and endothelial functions deteriorate. This has been shown to cause atherosclerosis, impaired nitric oxide-mediated vasodilatation and inflammatory cell migration. In our study, the correlation between the increase in the duration of diabetes mellitus and the development of CI-AKI is consistent with the literature. In our study, we found that age, BMI, LDL level, amount of contrast medium and total procedure time of CAG were higher in the group with CI-AKI, again consistent with the literature. The amount of contrast medium and procedure time are important for the development of CI-AKI. In our study, although the amount of contrast medium and procedure time were longer in the group that developed CI-AKI, the fact that the amount of contrast medium and procedure time were not statistically significant in the logistic regression analysis performed to determine the development of CI-AKI shows that the use of SGLT2-I reduces the development of CI-AKI independently of these two factors. This finding is important.

Development of is a complication that increases hospital stay, mortality and morbidity rates despite successful PCI. Although many factors predicting the development of CI-AKI after CAG or PCI have been described in the literature, widely accepted treatment options for the prevention of CI-AKI are insufficient CI-AKI.[3] Pre- and post-procedural hydration therapy and high-dose statin therapy before the procedure are effective methods in CI-AKI prophylaxis. In addition, there are limited data in the literature showing that amlodipine, theophylline and phosphodiesterase-5 inhibitors may be useful.[25] On the other hand, SGLT2-I is a group of drugs that are part of the current treatment of patients with diabetes and whose cardioprotective and nephroprotective effects have been strongly recognised in the light of recent studies. SGLT2-I may be useful for renal

protection in patients who will undergo elective coronary intervention, in patients who will be exposed to contrast media and in diabetic patients who are predicted to have a high risk of developing CI-AKI due to comorbidities.

Our study had some limitations. Our study was single-centre and retrospective. Further multicentre studies with a larger number of patients are needed. In our study, we used a definition of CI-AKI based on the change in creatinine value, which is generally accepted in the literature. Another limitation is that neutrophil gelatinase-associated lipocalin and cystatin C, which are more specific laboratory markers of CI-AKI, were not evaluated.

5. Conclusion

Our findings suggest that SGLT2-Is may significantly reduce the risk of CI-AKI in diabetic patients undergoing CAG or PCI. In conclusion, SGLT2-I use may have a protective and preventive efficacy against the development of CI-AKI. SGLT2-I may be an important and promising option for the clinical management of CI-AKI in patients with DM.

Statement of ethics

Adana City Training and Research Hospital Ethics Committee approved the study with decision number 2231 dated 03.11.2022.

Conflict of interest statement

The authors declare that they have no conflict of interest.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Author contributions

HAO, EG, DDO, FNA, is the major contributor to the writing of the manuscript. HAO, BSA, BI, BBK, IA, CD, AGM, CY, TS, HES are involved in the design, conception, data collection and analysis of the study. All authors read and approved the final version of the manuscript.

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