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Effects of Dapagliflozin on Renal Function and Metabolic Outcomes in CKD Patients with Type 2 Diabetes

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Abstract

Background: Chronic kidney disease (CKD) in type 2 diabetes mellitus (T2DM) is associated with metabolic, cardiovascular, and renal complications. Sodium-glucose cotransporter-2 (SGLT2) inhibitors like dapagliflozin offer benefits across these domains.

Aim of Study: This study aims to evaluate the effects of dapagliflozin on renal function, glycemic control, body composition, and blood pressure in CKD patients with T2DM, with a focus on changes in estimated glomerular filtration rate (eGFR), body mass index (BMI), and glycated hemoglobin (HbA1c).

Patients and Methods: This prospective cohort study (March 2021–August 2024) included CKD patients with T2DM receiving dapagliflozin or other antidiabetic agents. Changes in eGFR, BMI, HbA1c, and blood pressure were assessed.

Results: Among 255 patients (dapagliflozin: 155, control: 100), eGFR decline was similar (–2.34 vs. –2.14mL/min; p=0.76). BMI (–1.05 vs. –0.13kg/m^2, p<0.001) and HbA1c (–0.89% vs. –0.57%, p=0.008) decreased more with dapagliflozin. Linear regression showed dapagliflozin did not significantly affect systolic (p=0.282) or diastolic (p=0.610) blood pressure. Adverse events were comparable.

Conclusions: Dapagliflozin improved BMI and HbA1c without significant effects on eGFR or blood pressure over 12-months of follow-up, and had an acceptable safety profile, supporting its role in CKD management in T2DM.

Key Words: *Dapagliflozin – CKD Patients – T2 DM – SGLT2.*

Introduction

CHRONIC kidney disease (CKD) represents a major worldwide health concern, especially in people with type 2 diabetes mellitus (T2DM). In diabetic patients, the progression of CKD is closely linked to inadequate glycemic control, a higher likelihood of cardiovascular complications, and higher mortality rates. Sodium-glucose cotransporter-2 (SGLT2) inhibitors, such as dapagliflozin, have been recognized for their role in slowing CKD progression and improving metabolic outcomes. Clinical trials and major guidelines now advocate SGLT2 inhibitors as a key component of CKD management in people with T2DM *[1]*.

SGLT2 inhibitors reduce renal glucose reabsorption, leading to glucosuria and natriuresis, which contribute to hemodynamic and metabolic benefits. Large-scale clinical trials have repeatedly shown the renal and cardiovascular benefits of SGLT2 inhibitors **[2-6]**.

Importantly, early studies suggested that SGLT2 inhibitors might have a detrimental effect on renal function due to an initial decline in eGFR shortly after treatment initiation *[7]*. This was observed in short-term studies that followed patients for only a few weeks to months, leading to concerns about potential nephrotoxicity. However, long-term follow-up in major trials demonstrated that this initial drop in eGFR is transient and stabilizes over time, with long-term use ultimately slowing CKD progression **[2-6]**. The delayed renal benefits of SGLT2 inhibitors are now well recognized, highlighting the importance of extended follow-up periods in assessing their impact on renal outcomes.

SGLT2 inhibitors have multiple benefits, including promoting weight loss. A meta-analysis demonstrated a notable decrease in body weight compared to placebo, with an average loss of 2.48kg after one year and 2.99kg after two years *[8]*. Notably, these agents also facilitate weight reduction in individuals without diabetes *[9]*.

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SGLT2 inhibitors have also been shown to produce a modest yet significant reduction in blood pressure among patients with T2DM and hypertension. The antihypertensive effect is likely due to osmotic diuresis, natriuresis, and reduced arterial stiffness *[10]*.

SGLT2 inhibitors are commonly well tolerated; however, they are associated with certain adverse effects. The most commonly observed are genital fungal infections, especially in women, as glycosuria creates a favorable environment for fungal overgrowth *[11]*. Urinary tract infections (UTIs) have also been observed, although the overall increase in risk remains relatively small *[5]*. Initial concerns regarding acute kidney injury (AKI) have been reconsidered, as growing evidence suggests that SGLT2 inhibitors may actually lower the long-term risk of AKI by improving renal hemodynamics and mitigating hyperfiltration **[12]**. However, a temporary decline in eGFR is commonly seen upon treatment initiation, which may have contributed to early misconceptions about renal safety *[13]*. Less frequent but serious adverse events include euglycemic diabetic ketoacidosis (DKA), which can develop even in the absence of severe hyperglycemia, *[14]* and a potential increase in lower limb amputation risk, as reported in the CANVAS trial. Nevertheless, the consistency of this finding has not been established across studies *[3]*. Despite these risks, the overall safety profile of SGLT2 inhibitors remains favorable, particularly given their well-established cardiovascular and renal benefits.

Aim of the work:

This study aims to evaluate the impact of dapagliflozin on renal and metabolic parameters in Egyptian patients with CKD and T2DM.

Patients and Methods

Study design:

This was a prospective cohort study conducted at the outpatient clinic of the Kidney and Urology Center, Alexandria, Egypt. This study was conducted from March 1, 2021, to August 31, 2024.

Participants:

Patients with CKD and T2DM who were receiving either dapagliflozin or other antidiabetic agents were included. Eligibility criteria required stable kidney function at baseline.

Inclusion and exclusion criteria:

This study enrolled adult patients (≥18 years) with chronic kidney disease (CKD) stages 3 or 4 (eGFR 15–59mL/min/1.73m^2), as defined by KDIGO guidelines *[15]*. Inclusion criteria were: (1) Initiation of dapagliflozin with at least 12 months of follow-up; (2) Stable doses of antidiabetic agents (excluding dapagliflozin) at least four weeks prior to enrollment.

Exclusion criteria included: (1) Active malignancy; (2) Pregnancy or lactation; (3) Life-limiting conditions; (4) Specific kidney diseases (polycystic kidney disease, nephrotic syndrome, lupus nephritis, ANCA-associated vasculitis); (5) Type 1 diabetes mellitus; (6) A body mass index (BMI) <18.5kg/m^2; (7) A cardiac function classification of IV according to the New York Heart Association (NYHA); (8) Recent investigational drug use (within four weeks); (9) Acute or chronic liver disease; (10) Chronic pulmonary disease; (11) Significant active blood loss within 6 months; (12) Serum iPTH >600pg/mL at screening, and (13) Major surgery within 90 days prior to screening.

Outcome measures:

Primary outcomes included changes in eGFR, BMI, and HbA1c from baseline to 12 months. Secondary outcomes included adverse event rates and predictors of blood pressure changes.

Statistical analysis:

All statistical analyses were performed using R version 4.4.2 (R Foundation for Statistical Computing, Vienna, Austria). Continuous variables were summarized as mean ± standard deviation (SD) and compared between groups using Welch's two-sample *t*-tests. For baseline variables that were not normally distributed, data were reported as median and interquartile range (IQR). Categorical variables were presented as counts and percentages and analyzed using the chi-square test or Fisher's exact test when appropriate. Linear regression models were used to evaluate predictors of changes in systolic and diastolic blood pressure. Statistical significance was defined as $p<0.05$. Figures were generated using the ggplot2 package in R.

Results

Baseline characteristics:

A total of 255 patients were included in the study, with 155 in the dapagliflozin group and 100 in the control group. The baseline characteristics of the study population are presented in Table (1).

Changes in renal and metabolic parameters:

Changes in eGFR:

Over the 12-month period, the average change in eGFR was -2.14mL/min (SD=4.93) in the control group and -2.34mL/min (SD=5.59) in the dapagliflozin group. A Welch two-sample *t*-test showed no significant difference between the two groups (t=0.3, df=229.73, 95% CI: –1.11 to 1.51, p=0.76) (Fig. 1). Fig. (2) shows the longitudinal changes of eGFR in the treatment groups over the study period. In the dapagliflozin group, an initial eGFR dip ≥10% was observed in (47.7%) of patients.

Table (1): Description of baseline characteristics.

Characteristic	Control (n=100)	Dapagliflozin (n=155)	p
Sex — n. (%):			
Males	54 (54.0%)	75 (48.6%)	0.381[a]
Females	46 (46.0%)	80 (51.6%)	
Age — years	63 (55-69)	64 (56-70)	0.092[b]
Age group (years) — n. (%):			
Age <65	57 (57.0%)	74 (47.7%)	0.149[a]
Age ≥65	43 (43.0%)	81 (52.3%)	
eGFR — mL/min [CKD-EPI 2021]	37.1 (22.9-47.0)	38.3 (27.4-49.8)	0.214[b]
eGFR group (mL/min) — n. (%):			
eGFR <30	37 (37.0%)	48 (31.0%)	0.318[a]
eGFR ≥30	63 (63.0%)	107 (69.0%)	
Hemoglobin — g/dL	11.8 (11.2-12.6)	11.9 (11.5-12.8)	0.914[b]
Anemia at baseline	66.0 (66.0%)	109.0 (70.3%)	0.468[a]
BMI — Kg/m^2	31.6 (28.1-34.3)	33.2 (28.3- 37.9)	0.138[b]
HbA1c — %	7.2 (6.8-7.9)	7.0 (6.3-8.1)	0.344[b]
SBP — mmHg	140 (130-150)	140 (130-150)	0.666[b]
DBP — mmHg	80 (80-90)	80 (80-90)	0.511[b]
Urinary ACR — mg/g	580.0 (130.0-1092.0)	698.0 (110.0-1366.0)	0.068[b]
UACR > 1000 mg/g	28 (28.0%)	58 (37.4%)	0.120[a]
Hypertension — n. (%)	73 (73.0%)	141 (91.0%)	<0.001*,[a]
IHD — n. (%)	40 (40.0%)	70 (45.2%)	0.417[a]
Heart Failure — n. (%)	15 (15.0%)	27 (17.4%)	0.611[a]
RAS inhibitors — n. (%)	46 (46.0%)	77 (49.7%)	0.566[a]
Diuretics — n. (%)	40 (40.0%)	64 (41.3%)	0.838[a]
CCBs — n. (%)	57 (57.0%)	74 (47.7%)	0.149[a]
Current smoker — n. (%)	7 (7.0%)	9 (5.8%)	0.701[a]

Data presented as median (IQR) unless otherwise indicated.
BMI : Indicates Body Mass Index.
CCB : Calcium channel blockers.
DBP : Diastolic blood pressure.
eGFR: estimated glomerular filtration rate.
IHD : Ischemic heart disease.
RAS : Renin-Angiotensin System Inhibitor.
SBP : Systolic blood pressure.

a = Pearson Chi-Square Test.
b = Independent-Samples Mann-Whitney U Test.
* = The test is significant at the 0.050 level.

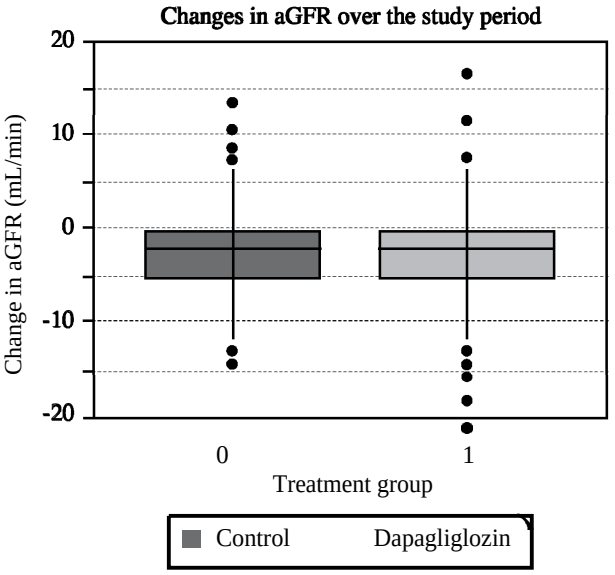

Fig. (1): eGFR changes in the treatment groups in overall cases over the study period (n=255).

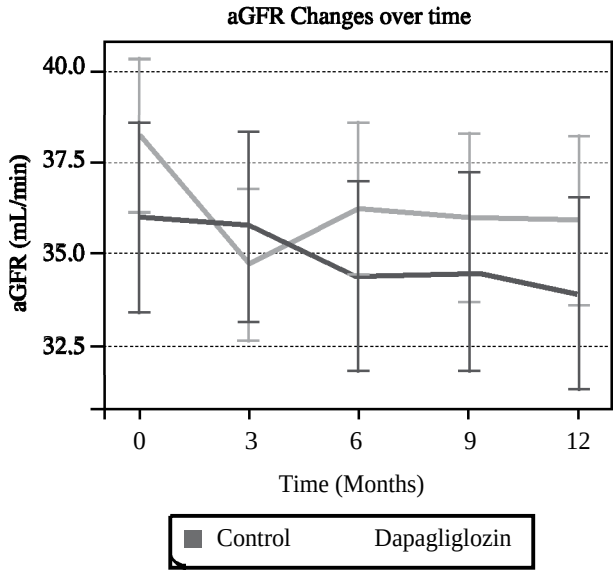

Fig. (2): Longitudinal changes of eGFR in the treatment groups in overall cases over the study period (n=255).

Changes in BMI:

The reduction in BMI was significantly greater in the dapagliflozin group compared to the control group (t=5.68, df=180.29, 95% CI: 0.6 to 1.24, p<0.001). On average, BMI decreased by 0.13kg/m^2 (SD=1.36) in the control group, whereas the dapagliflozin group experienced a larger decline of 1.05kg/m^2 (SD=1.11). These findings suggest that dapagliflozin treatment led to a more substantial reduction in BMI over the course of the study (Fig. 3).

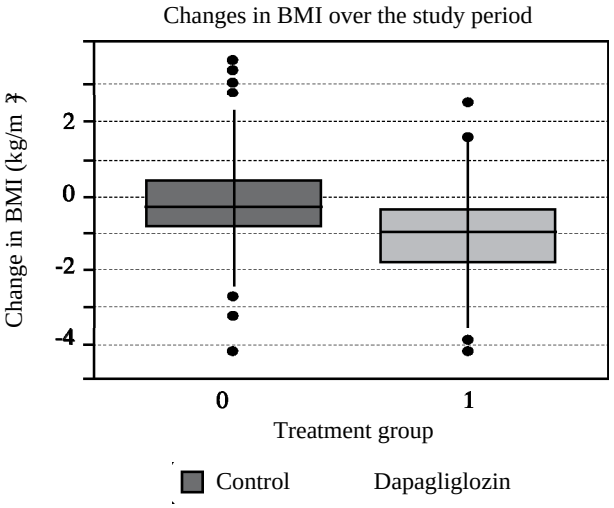

Fig. (3): BMI changes in the treatment groups in overall cases over the study period (n=255).

Changes in HbA1c:

The dapagliflozin group exhibited a greater reduction in HbA1c compared to the control group (t=2.7, df=148.41, 95% CI: 0.09 to 0.55, p=0.008). On average, HbA1c decreased by 0.57% (SD= 0.44%) in the control group, whereas the dapagliflozin group experienced a larger decline of 0.89% (SD=1.16%). These findings suggest that dapagliflozin therapy led to a greater enhancement in glycemic control (Fig. 4). Table (2) shows the antidiabetic medications used by patients in treatment groups.

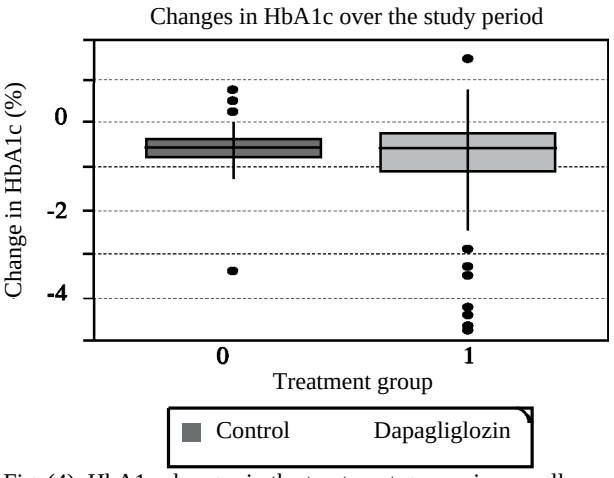

Fig. (4): HbA1c changes in the treatment groups in overall cases over the study period (n=255).

Table (2) Antidiabetic medications used by patients.

Drug class	Control (n=100)	Dapagliflozin (n=155)
Insulin	50 (50.0%)	47 (38.2%)
Metformin	46 (46.0%)	47 (38.2%)
Sulfonylurea	15 (15.0%)	43 (35.0%)
DPP-4 inhibitor	61 (61.0%)	55 (44.7%)
Thiazolidinedione	2 (2%)	3 (2.4%)
Meglitinide	3 (3%)	0 (0.0%)
GLP-1 agonist	1 (1%)	4 (3.3%)

DPP-4: Dipeptidyl peptidase 4 inhibitors.

GLP-1: Glucagon-like peptide-1 agonists.

Predictors of blood pressure changes:

Linear regression analyses were conducted in hypertensive patients (n=214) to assess predictors of systolic blood pressure (SBP) and diastolic blood pressure (DBP) changes. The SBP model explained only 4.56% of the variability (R^2=0.0456, p=0.471), while the DBP model explained 5.78% (R^2=0.0578, p=0.264), indicating weak predictive power.

Dapagliflozin treatment did not significantly affect SBP (β=–2.775, p=0.282) or DBP (β=0.848, p=0.610). Baseline eGFR showed a borderline significant association with SBP change (β=0.184, p=0.050), suggesting a weak link between higher kidney function and SBP increase. Heart failure was strongly linked to a rise in DBP (β=5.461, p=0.008), possibly reflecting increased vascular resistance. Other factors, including baseline BMI, anemia status, age, sex, and antihypertensive medications, were not significant predictors of BP changes.

Table (3) shows the antihypertensive medications used by patients with hypertension. Fig. (5) show the changes in SBP and DBP over time in hypertensivepatients in the treatment cohorts.

Table (4) show the detailed linear regression analysis of SBP and DBP Changes in hypertensive Patients.

Table (3): Antihypertensive medications used by patients with hypertension (n=214).

Drug class	Control (n=73)	Dapagliflozin (n=141)
RAS inhibitors	36 (49.3%)	73 (51.8%)
Calcium channel blockers	57 (78.1%)	71 (50.4%)
β-Blockers	37 (50.7%)	100 (70.9%)
Diuretics	33 (45.2%)	62 (44.0%)
Others	14 (19.2%)	26 (18.4%)

RAS inhibitors, Renin-Angiotensin System Inhibitor.

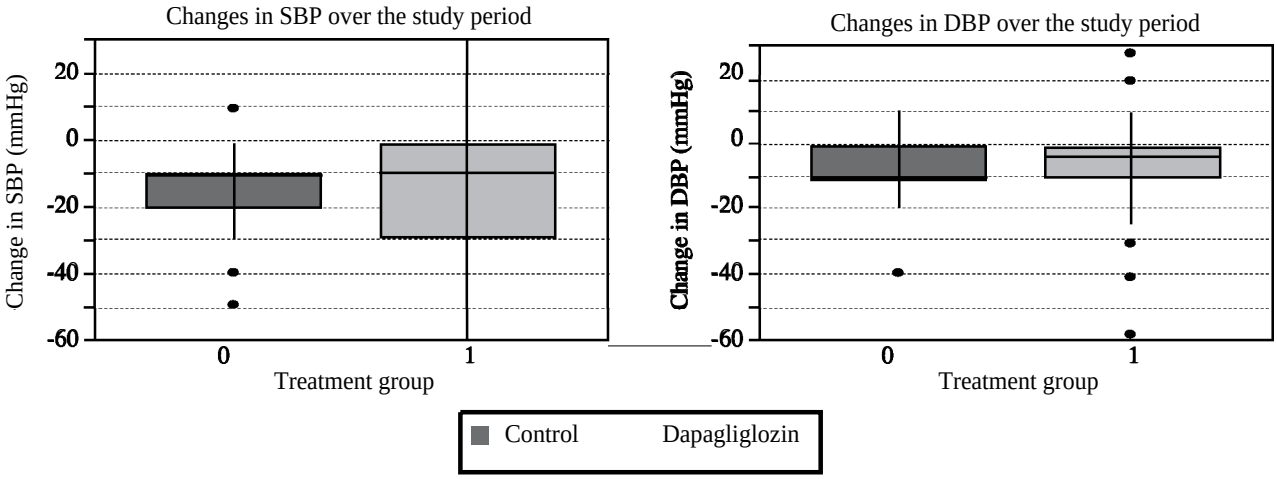

Fig. (5): Changes in blood pressure over time in the treatment groupsin patients with hypertension (n=214).

Table (4): Linear Regression Analysis of SBP and DBP Changes in Hypertensive Patients (n=214).

Variable	Model	Estimate	Std. Error	*t*-value	*p*-value
(Intercept)	SBP	-23.872	11.836	-2.017	0.045
	DBP	-9.737	7.630	-1.276	0.203
Dapagliflozin	SBP	-2.775	2.577	-1.077	0.282
	DBP	0.848	1.661	0.511	0.610
Heart Failure	SBP	3.562	3.204	1.112	0.267
	DBP	5.461	2.065	2.644	0.008
Baseline BMI	SBP	0.067	0.213	0.315	0.752
	DBP	0.108	0.137	0.791	0.430
Baseline eGFR	SBP	0.184	0.093	1.974	0.050
	DBP	0.058	0.060	0.974	0.331
Anemia at baseline	SBP	0.621	2.727	0.228	0.820
	DBP	0.474	1.758	0.269	0.788
Age	SBP	-0.046	0.114	-0.399	0.690
	DBP	-0.086	0.074	-1.166	0.245
Sex	SBP	0.379	2.827	0.134	0.893
	DBP	1.533	1.822	0.841	0.401
Antihypertensive medications:					
Use of a single medication	SBP	11.756	7.509	1.565	0.119
	DBP	5.010	4.840	1.035	0.302
Combined use of 2 medications	SBP	4.822	2.736	1.762	0.079
	DBP	0.774	1.763	0.439	0.661
Combined use of ≥3 medications	SBP	-3.785	3.769	-1.004	0.316
	DBP	0.765	2.429	0.314	0.753

For SBP: Residual standard error: 16.64 on 203 degrees of freedom, Multiple R-squared: 0.04558, Adjusted R-squared: -0.001433, F-statistic: 0.9695 on 10 and 203 DF, *p*-value: 0.4712

For DBP: Residual standard error: 10.71 on 203 degrees of freedom, Multiple R-squared: 0.05784, Adjusted R-squared: 0.01142, F-statistic: 1.246 on 10 and 203 DF, *p*-value: 0.2636.

Adverse events:

The most commonly experienced adverse event was urinary tract infection (UTI), occurring in 16.8% (n=26) of dapagliflozin patients versus 13.0% (n=13) in the control group (p=0.414). Treatment interruption was rare, with only 2 patients in the dapagliflozin group requiring temporary discontinuation. Two female patients in the dapagliflozin group experienced genital fungal infections requiring temporary treatment interruption. No cases of serious adverse events, including AKI, ketoacidosis, fractures, amputations, or hypoglycemia, were reported.

Discussion

In this prospective cohort study, we investigated the effects of dapagliflozin on renal function andmetabolic parameters, and adverse events in patients with CKD and T2DM over a 12-month period. Our findings indicate that dapagliflozin was associated with significant reductions in BMI and HbA1c compared to standard antidiabetic therapy, without accelerating renal function decline. The safety profile of dapagliflozin remained acceptable, with adverse events occurring at comparable rates between groups.

Renal function changes:

The effect of dapagliflozin on renal function has been widely studied, with major clinical trials demonstrating its long-term nephroprotective benefits. In our study, the mean eGFR decline was similar between the dapagliflozin and control groups (–2.34 vs. –2.14mL/min/1.73 m^2, p=0.76), reinforcing the notion that SGLT2 inhibitors do not accelerate CKD progression. These findings align with the results of the DAPA-CKD trial, which established that dapagliflozin slows CKD progression despite an initial eGFR dip upon treatment initiation.5 The transient eGFR decline observed in 47.7% of dapagliflozin-treated patients in our study is consistent with previous reports and is thought to result from hemodynamic changes that ultimately confer long-term renal protection **[16].**

Metabolic benefits:

Our study demonstrated that dapagliflozin significantly reduced BMI (–1.05 kg/m^2 vs. –0.13 kg/m^2, p<0.001) and HbA1c (–0.89% vs. –0.57%, p = 0.008) compared to the control group. These results align with prior studies showing that SGLT2 inhibitors promote weight loss, probably through urinary glucose excretion, natriuresis, and associated caloric loss. However, the weight loss observed in clinical practice tends to be lower than the theoretical prediction, likely due to compensatory mechanisms such as increased appetite and gluconeogenesis *[17,18]*. Similarly, the observed reduction in HbA1c with dapagliflozin is consistent with previous trials, underscoring its efficacy in promoting better glucose control in patients with T2DM *[19]*.

Blood pressure and cardiovascular considerations:

Despite evidence from meta-analyses indicating that SGLT2 inhibitors modestly lower blood pressure through osmotic diuresis and natriuresis, *[10]* our study did not find a significant effect of dapagliflozin on systolic or diastolic blood pressure, possibly due to sample size, baseline differences with more prevalent hypertension in the dapagliflozin group, or concurrent medication use. Regression analyses revealed no strong predictors of blood pressure changes, except for a weak association between baseline eGFR and SBP (p=0.050) and a significant association between heart failure and DBP increase (p=0.008). These findings suggest that the blood pressure effects of dapagliflozin may be more pronounced in specific subgroups, warranting further investigation.

Safety and adverse events:

The adverse event profile of dapagliflozin in our study was consistent with existing literature. The most common adverse events were UTIs (16.8% in dapagliflozin vs. 13.0% in control, p=0.414) and genital infections, with two cases requiring treatment interruption. Notably, no serious adverse events such as AKI, ketoacidosis, fractures, amputations, or severe hypoglycemia were reported. These findings support the overall safety of dapagliflozin, aligning with major clinical trials that have demonstrated its favorable risk-benefit profile.

Strengths and Limitations:

A major strength of our study is its prospective design, which allowed for a comprehensive assessment of dapagliflozin's effects over a clinically relevant follow-up period. Additionally, our study included a real-world CKD population, enhancing its generalizability. However, several limitations should be acknowledged. First, our study lacked randomization, which may introduce selection bias and confounding factors. Second, the sample size, while adequate for detecting differences in metabolic parameters, may have been underpowered to assess rarer adverse events.

Conclusion:

Dapagliflozin significantly reduces BMI and HbA1c in CKD patients with T2DM without affecting renal function or blood pressure significantlyover 12-months of follow-up. These results indicate that dapagliflozin does not negatively impact kidney function, but the follow-up period (12 months per patient) may be too short to fully demonstrate the expected positive outcomes. It has an acceptable safety profile, making it a viable option for this patient population. These results align with the growing body of evidence highlighting the benefits of SGLT2 inhibitors in CKD management.

Declarations:

Ethics approval and consent to participate:

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and was approved by the ethics committee at Damanhour University (Ref. No. 221PP32) before the commencement of the study. The study was conducted at the outpatient clinic of the Kidney and Urology Center, Alexandria, Egypt, from March 1, 2021, to August 31, 2024. Informed consent was obtained from all participants before their inclusion in the study.

Consent for publication:

Not applicable.

Availability of data and material:

The data sets used and analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests:

The authors declare no competing interests.

Funding:

No funding was received for this study.

Authors' Contributions:

All authors contributed to the study conception and design. Ramy Ibrahim Elwaraky conducted the research, performed data analysis, and wrote the manuscript. All authors reviewed and provided feedback on previous versions of the manuscript. All authors have reviewed and approved the final manuscript for publication.

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تأثير داباغليفلوزين على وظائف الكلى والنتائج الأيضية لدى مرضى السكرى من النوع الثاني المصابين بالقصور الكلوى المزمن

تستهدف هذه الدراسة تقييم تأثير دواء داباغليفلوزين على وظائف الكلى والنتائج الأيضية لدى مرضى السكرى من النوع الثانى المصابين بالقصور الكلوى المزمن. شملت الدراسة ٢٥٥ مريضًا (١٥٥ فى مجموعة داباغليفلوزين و١٠٠ فى المجموعة الضابطة) خلال الفترة من مارس ٢٠٢١ إلى أغسطس ٢٠٢٤.

تمت مقارنة التغيرات فى معدل الترشيح الكبيبى المقدّر (eGFR)، ومؤشر كتلة الجسم (BMI)، ومستوى السكر التراكمى (HbA1c)، وضغط الدم بين المجموعتين. لم تظهر الدراسة فرقًا ملحوظًا فى انخفاض معدل الترشيح الكبيبى المقدّر (eGFR) بين المجموعتين (−٢,٣٤ مقابل −٢,١٤ مل/دقيقة؛ p=٠,٧٦)، مما يشير إلى عدم تأثير داباغليفلوزين سلبًا على وظائف الكلى، لكن قد تكون مدة المتابعة (١٢ شهرًا لكل مريض) أقصر من أن تظهر النتائج الإيجابية المنتظرة. كما أظهرالانحدار الخطى أن داباغليفلوزين لم يؤثر بشك لملحوظ على ضغط الدم الانقباضى ($p = 0.282$) أوالانبساطى ($p = 0.610$).

من ناحية أخرى، سجلت مجموعة داباغليفلوزين انخفاضًا أكبر فى مؤشر كتلة الجسم (BMI): (−١,٠٥ مقابل −٠,١٣ كغم/م²؛ $p < 0.001$) ومستوى السكر التراكمى (HbA1c): (−٠,٨٩٪ مقابل −٠,٥٧٪؛ $p = 0.008$)، مما يعكس تحسنًا واضحًا فى ضبط السكر والوزن.

كما أظهرت الدراسة أن نسبة الآثار العكسية كانت متشابهة بين المجموعتين، مما يؤكد أمان استخدام داباغليفلوزين. وبناءً على هذه النتائج، تدعم الدراسة دور داباغليفلوزين فى تحسين المؤشرات الأيضية لدى مرضى السكرى المصابين بالقصور الكلوى المزمن.