

## Original Article

## Comparison of Dapagliflozin and Empagliflozin in Patients with Type 2 Diabetes and/or Chronic Kidney Disease: A Real-World Observational Study

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## Abstract

**Background:** Sodium-glucose cotransporter-2 (SGLT2) inhibitors provide renal protection in patients with diabetes mellitus (DM) and chronic kidney disease (CKD), but comparative real-world data on dapagliflozin and empagliflozin across different renal diagnoses are limited.

**Method:** In this retrospective observational study, 328 adults with DM and/or CKD, including diabetic nephropathy, glomerulonephritis, and heart failure, who received dapagliflozin or empagliflozin for at least 12 months were evaluated. Demographic, clinical, and laboratory data, including estimated glomerular filtration rate (eGFR) and urine albumin-to-creatinine ratio, were recorded at baseline and 12 months. The primary outcome was  $\geq 50\%$  reduction in albuminuria, and a  $>50\%$  decline in eGFR was analyzed as a safety endpoint.

**Results:** Of 328 patients (mean age  $60.4 \pm 11.1$  years, 43.3% female), 165 received dapagliflozin and 163 received empagliflozin. Among 298 patients with DM, 61.4% achieved  $\geq 50\%$  reduction in albuminuria at 12 months, while 86.7% of 30 non-diabetic patients reached this target. High response rates were observed in patients with isolated DM, DM+CKD, and non-diabetic CKD, including those with glomerulonephritis. Only one patient (with DM and CKD in the empagliflozin group) experienced a  $>50\%$  decline in eGFR; no such decline occurred in other subgroups. There were no significant differences between dapagliflozin and empagliflozin in albuminuria reduction or eGFR decline across diagnostic categories.

**Conclusion:** In this real-world cohort, dapagliflozin and empagliflozin were similarly effective in substantially reducing albuminuria and preserving eGFR in patients with DM, CKD, glomerulonephritis, and heart failure. These findings support the use of SGLT2 inhibitors as renoprotective therapy across diverse high-risk populations.

**Keywords:** Sodium-Glucose Transporter 2 Inhibitors, Dapagliflozin, Empagliflozin, Albuminuria, Renal Insufficiency, Chronic

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## INTRODUCTION

The prevalence of diabetes mellitus (DM) is increasing in Türkiye, as it is worldwide. With the rise in type 2 DM, the number of individuals at risk of cardiovascular disease and end-stage renal disease also continues to grow (1). According to data from the Turkish Society of Nephrology, DM remains the most common underlying cause among incident hemodialysis patients (2).

Sodium-glucose cotransporter 2 inhibitors (SGLT2i) are oral antidiabetic agents used in the management of type 2 DM. These drugs act by inhibiting the SGLT2 cotransporter in the renal proximal tubule, thereby reducing glucose reabsorption and promoting urinary glucose excretion. Beyond their glucose-lowering effects, SGLT2 inhibitors exert renoprotective benefits

independent of glycemic control. By blocking the reabsorption of glucose and sodium in the proximal tubule, they increase sodium delivery to the macula densa, which activates the tubuloglomerular feedback mechanism and induces vasoconstriction of the afferent arteriole. The resulting decrease in intraglomerular pressure is expected to reduce proteinuria (3). Considering that DM is the leading cause of chronic kidney disease (CKD), SGLT2 inhibitors (capable of improving glycemic control and lowering proteinuria) have become a preferred therapeutic option for patients with diabetic nephropathy. In Türkiye, the available SGLT2 inhibitors include dapagliflozin and empagliflozin.

It has been reported that 20–40% of individuals diagnosed with DM develop diabetic nephropathy (4).

A decline in estimated glomerular filtration rate (eGFR) and an increase in proteinuria are associated with higher mortality among patients with diabetic nephropathy (5). In a study by Coppo et al., age and proteinuria were identified as the key factors influencing renal prognosis in patients with IgA nephropathy (6). Likewise, a study on membranous glomerulonephritis demonstrated that proteinuria had a significant impact on prognosis (7). Consequently, the use of SGLT2 inhibitors, which effectively reduce proteinuria, has been increasing among patients followed for glomerulonephritis (4). In the treatment of diabetic nephropathy, SGLT2 inhibitors play a crucial role by lowering intraglomerular pressure and thereby contributing meaningfully to disease management.

Accordingly, this study aimed to evaluate and compare the effects of dapagliflozin and empagliflozin on albuminuria and decline in eGFR among patients with CKD, including those with glomerulonephritis and diabetic nephropathy.

## METHODS

### Study Design and Participants

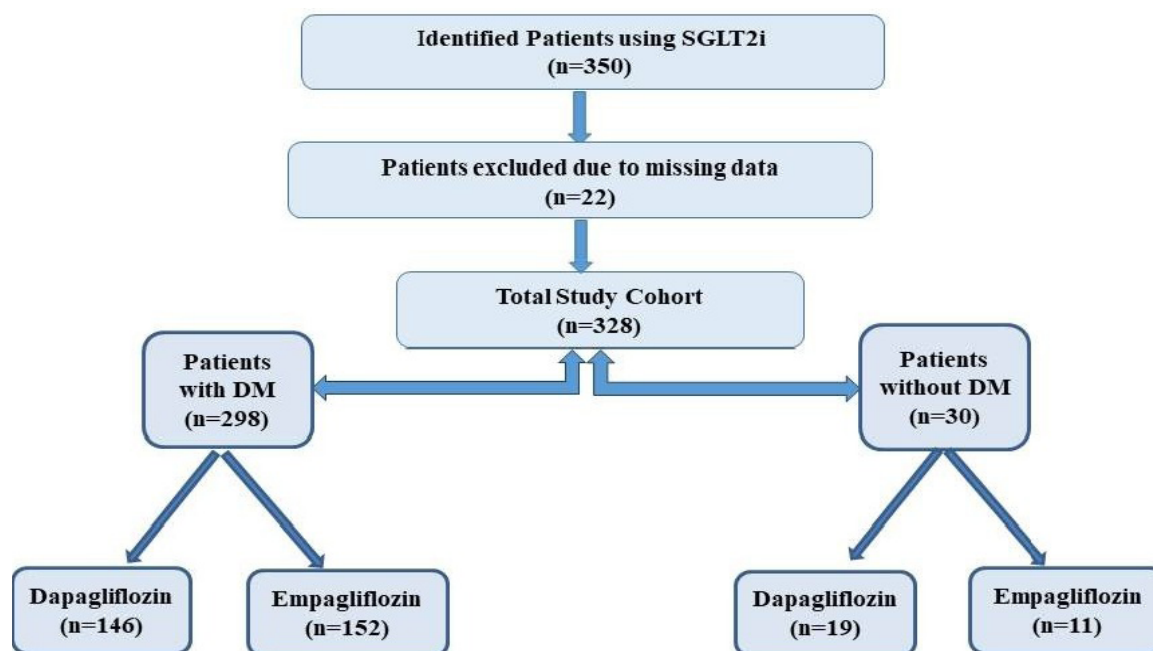
This retrospective observational study included 328 adult patients who were diagnosed with DM, CKD, including diabetic nephropathy and glomerulonephritis, and had been using a SGLT2i for at least one year at the Nephrology Clinic/Outpatient Unit of Dışkapı Training and Research Hospital between January 2022 and January 2023. Exclusion criteria was stage 5 CKD patients, type 1 DM patients and patients with missing data. The flowchart for the study is presented in **Figure 1**.

**1.** Ethical approval for the study was obtained from the Gülhane Faculty of Medicine Ethics Committee (Decision No: 146/24, Date: 12/09/2022). Individual consent to participate was waived with the approval of the ethics committee due to the retrospective design of the study.

Patients' diagnosis, treatment, and follow-up data were retrospectively reviewed from medical records and recorded. Clinical and demographic variables such as age at treatment initiation, sex, comorbidities, smoking status, body mass index (BMI), concomitant treatments (RAAS blockers, diuretics, statins) and systolic and diastolic blood pressures were collected. Laboratory parameters including serum creatinine, eGFR, spot urine albumin-to-creatinine ratio (ACR), potassium, hemoglobin, and HbA1c were also recorded. In addition, patients' 12th-month laboratory values for creatinine, eGFR, and, ACR were retrospectively retrieved and documented. eGFR was calculated with the formula CKD-EPI. CKD staging was performed according to the KDIGO 2024 CKD guideline. BMI was calculated as body weight divided by the square of height ( $\text{kg/m}^2$ ). The SGLT2i used by each patient—either dapagliflozin or empagliflozin—was recorded. All patients receiving dapagliflozin received 10 mg. Patients with DM without CKD received empagliflozin 25 mg, while patients with CKD received empagliflozin 10 mg.

## STATISTICAL ANALYSIS

Data analysis was conducted using SPSS version 26 and a p value <0.05 was considered statistically significant. Continuous variables have been summarized as mean



**Figure 1.** Flowchart of the study

**Table 1.** Baseline Characteristics of Study Population (n=148)

Parameters	Total (n=328)	Dapagliflozin (n=165)	Empagliflozin (n=163)	p value
Age, years, mean±SD	60.4±11.1	60.1±12.0	60.7±10.2	0.631
Female, n (%)	142(43.3)	74(44.8)	68(41.7)	0.567
Body mass index (kg/m <sup>2</sup> ), mean±SD	30.7±5.8	30.3±6.0	31.0±5.6	0.261
Smoking, n (%)	144(43.9)	68(41.2)	76(46.6)	0.323
Blood pressure (mmHg), mean±SD				
Systolic blood pressure	134.4±19.1	133.7±17.4	135.0±20.8	0.526
Diastolic blood pressure	79.2±12.8	77.8±12.0	80.6±13.6	0.056
Estimated GFR (mL/min/1.73 m <sup>2</sup> ), mean±SD	70.9±24.5	69.4±24.9	72.5±24.2	0.258
Stage 1–2 CKD, n (%), (eGFR≥60)	198(60.4)	97(58.8)	101(62.0)	0.411
Stage 3a CKD, n (%), (eGFR 45–59)	81(24.7)	38(23.0)	43(26.4)	
Stage 3b CKD, n (%), (eGFR 30–44)	38(11.6)	23(13.9)	15(9.2)	
Stage 4 CKD, n (%), (eGFR 15–29)	11(3.4)	7(4.2)	4(2.5)	
Serum creatinine (mg/dL), mean±SD	1.2±0.5	1.2±0.5	1.1±0.5	0.299
Hemoglobin (g/dL), mean±SD	13.5±2.0	13.3±2.0	13.7±2.0	0.065
HbA1c (%), mean±SD	8.3±1.9	8.2±2.0	8.3±1.9	0.671
Potassium (mEq/L), mean±SD	4.6±0.6	4.6±0.5	4.5±0.6	0.147
Urine albumin/creatinine ratio, mean±SD	402.8±836.6	474.5±975.6	330.2±662.4	0.124
< 30 mg/g creatinine, n (%)	75(22.9)	37(22.4)	38(23.3)	0.587
30–300 mg/g creatinine, n (%)	156(47.6)	75(45.5)	81(49.7)	
> 300 mg/g creatinine, n (%)	97(29.6)	53(32.1)	44(27.0)	
Diabetes mellitus, n (%)	298(90.9)	146(88.5)	152(93.3)	0.134
Hypertension, n (%)	251(76.5)	123(74.5)	128(78.5)	0.395
Glomerulonephritis, n (%)	26(7.9)	20(12.1)	6(3.6)	0.043
Heart failure, n (%)	87(26.5)	36(21.8)	51(31.2)	0.221
Reduced EF (≤ 40 %), n (%)	43(49.4)	15(41.6)	28(54.9)	0.149
Mildly reduced EF (41–49 %), n (%)	14(16.0)	6(16.6)	8(15.6)	
Preserved EF (≥ 50 %), n (%)	30(34.4)	17(47.2)	13(25.4)	
Concomitant medications, n (%)				
RAAS blocker	316(96.3)	161(97.6)	155(95.1)	0.231
Diuretic	199(60.7)	95(57.6)	104(63.8)	0.248
Statin	176(53.7)	83(50.3)	93(57.1)	0.220

SD, standard deviation; GFR, glomerular filtration rate; CKD, chronic kidney disease; EF, ejection fraction; RAAS, renin-angiotensin-aldosterone system.

± standard deviation, and categorical variables as frequency and percentage. Differences between groups were assessed using the appropriate statistical tests for the groups, including the independent samples t-test for continuous variables, and the Chi-square or Fisher's exact test for categorical variables.

## RESULTS

A total of 328 patients were included in the study. The mean age of participants was 60.4 ± 11.1 years and 142 (43.3%) patients were female. Among the participants, 165 (50.3%) were using dapagliflozin and 163 (49.7%) were using empagliflozin. The mean serum creatinine level was 1.2 ± 0.5 mg/dL, and the mean eGFR was 70.9 ± 24.5 mL/min/1.73 m<sup>2</sup>. Of the total patients, 298 (90.9%) had DM, 251 (76.5%) had hypertension, 87 (26.5%) had heart failure, and 26 (7.9%) had a diagnosis of glomerulonephritis.

The demographic, clinical, and laboratory characteristics of the patients, along with the comparison of these parameters between those using dapagliflozin and empagliflozin, are presented in **Table 1**.

Among the 298 patients diagnosed with DM, 74 (24.8%) did not have accompanying CKD or heart failure. A total of 210 patients (70.5%) had CKD in addition to DM, 73 (24.5%) had concurrent heart failure, and 6 (2.0%) had a diagnosis of glomerulonephritis alongside DM.

Of the 30 patients without a DM diagnosis, 25 (83.3%) had CKD, 20 (66.6%) had glomerulonephritis, and 14 (46.6%) had heart failure. The comparison of disease frequencies between dapagliflozin and empagliflozin users among patients with and without diabetes is presented in **Table 2**.

A ≥50% reduction in albuminuria at the 12th month was observed in 183 (61.4%) of the 298 patients diagnosed with DM. Among the 30 patients without a DM diagnosis,

**Table 2.** Comparison of disease frequencies between dapagliflozin and empagliflozin users among patients with and without diabetes mellitus

Patients with DM	Total (n=298)	Dapagliflozin (n =146)	Empagliflozin (n =152)	p value
DM only, n (%)	74 (24.8)	33 (22.6)	41 (27.0)	0.383
DM + CKD, n (%)	210 (70.5)	109 (74.7)	101 (66.4)	0.120
DM + GN, n (%)	6 (2.0)	2 (1.4)	4 (2.6)	0.438
Membranous GN	3 (1.0)	1 (0.7)	2 (1.3)	1.000
MPGN	1 (0.3)	0	1 (0.7)	
FSGS	2 (0.7)	1 (0.7)	1 (0.7)	
DM + Renal Transplant, n (%)	7 (2.3)	5 (3.4)	2 (1.3)	0.275
DM + Heart Failure, n (%)	73 (24.5)	33 (22.6)	40 (26.3)	0.456
Reduced EF ( $\leq 40\%$ )	31 (42.5)	12 (36.4)	19 (47.5)	0.128
Mildly Reduced EF (41–49%)	13 (17.8)	4 (12.1)	9 (22.5)	
Preserved EF ( $\geq 50\%$ )	29 (39.7)	17 (51.5)	12 (30.0)	
CKD, n (%)	25 (83.3)	15 (78.9)	10 (90.9)	0.397
GN, n (%)	20 (66.6)	18 (94.7)	2 (18.1)	0.002
IgA nephropathy	14 (70.0)	13 (72.2)	1 (50.0)	0.007
Membranous GN	5 (25.0)	4 (22.2)	1 (50.0)	
Minimal Change Disease	1 (3.3)	1 (5.5)	0 (0)	
Heart Failure, n (%)	14(46.6)	3(15.7)	11(100.0)	0.056
Reduced EF ( $\leq 40\%$ )	12(85.7)	2(66.6)	10(90.9)	0.333
Mildly Reduced EF (41–49%)	1(7.1)	1(33.3)	0 (0)	
Preserved EF ( $\geq 50\%$ )	1(7.1)	0 (0)	1(9.0)	

DM, diabetes mellitus; CKD, chronic kidney disease; GN, glomerulonephritis; MPGN, membranoproliferative glomerulonephritis; FSGS, focal segmental glomerulosclerosis; EF, ejection fraction.

26 (86.7%) showed a  $\geq 50\%$  reduction in albuminuria at the 12th month. The comparison of patients receiving dapagliflozin and empagliflozin who achieved a  $\geq 50\%$  reduction in albuminuria at 12 months across different disease groups is presented in **Table 3**.

Among patients with only DM, as well as those with

DM and renal transplantation or DM and heart failure, no patients in either the dapagliflozin or empagliflozin groups experienced a  $>50\%$  decline in eGFR at the 12th month. In patients diagnosed with both DM and CKD, none of the patients in the dapagliflozin group showed a  $>50\%$  decrease in eGFR at 12 months, whereas only

**Table 3.** Comparison of patients showing  $\geq 50\%$  reduction in albuminuria at the first year between dapagliflozin and empagliflozin groups

groups	Total		Dapagliflozin		Empagliflozin		p value
	Total	≥ %50 reduction in Albuminuria	Total	≥ %50 reduction in Albuminuria	Total	≥ %50 reduction in Albuminuria	
Patients with DM							
Only DM	74	53 (71.6)	33	22 (66.6)	41	31 (75.6)	0,255
Chronic kidney disease	210	123 (58.5)	109	64 (58.6)	101	59 (58.4)	0,126
Renal Transplant	7	3 (42.8)	5	2 (40.0)	2	1 (50.0)	0,516
Heart Failure	73	37 (50.6)	33	16 (48.4)	40	21 (52.5)	0,509
Reduced EF (≤40%)	31	14 (46.6)	12	4 (33.3)	19	10 (52.6)	0.119
Mildly Reduced EF (41–49%)	13	7 (53.8)	4	2 (50.0)	9	5 (55.5)	
Preserved EF (≥50%)	29	16 (55.1)	17	10 (58.8)	12	6 (50.0)	
Patients without DM							
Chronic kidney disease	25	21 (84.0)	15	14 (93.3)	10	7 (70.0)	0.562
Heart Failure	14	12 (85.7)	3	3 (100.0)	11	9 (81.8)	0.365
Glomerulonephritis	20	18 (90.0)	18	17 (94.4)	2	1 (50.0)	0.070
IgA Nephropathy	14	13 (92.8)	13	12 (92.3)	1	1 (100.0)	-
Membranous GN	5	4 (80.0)	4	4 (100.0)	1	0	-
MCD	1	1 (100.0)	1	1 (100.0)	0	0	-

DM, diabetes mellitus; EF, ejection fraction; GN, glomerulonephritis; MCD, minimal change disease



one patient in the empagliflozin group exhibited such a decline. Among patients without a DM diagnosis, no >50% decrease in eGFR at the 12th month was observed in either treatment group.

## DISCUSSION

The results of this retrospective cohort revealed the reduction rate of albuminuria  $\geq 50\%$  at 12 months to be notably high across multiple patient groups; including those with only DM, those with DM and CKD and those with only CKD – such as the patients with glomerulonephritis. It is important to note that there was no significant difference between dapagliflozin and empagliflozin in regards to their ability to reduce albuminuria across these subgroups – seemingly showing both molecules to be equally effective. Additionally, only one patient in the entire cohort experienced a >50% decline in estimated eGFR at the 12th month.

Overall, the proportion of patients achieving more than a 50% reduction in albuminuria was high in all diagnostic groups. Our findings support Lin et al., who previously demonstrated that SGLT2 inhibitors effectively reduce albuminuria levels (8). Similarly, a meta-analysis including 12 studies and 89,191 patients confirmed that SGLT2 inhibitors significantly lower albuminuria (9). In our cohort, none of the patients with DM showed a >50% eGFR decline after one year, aligning with the meta-analysis by Toyama et al., which included 27 studies and 7,363 patients and demonstrated that SGLT2 inhibitors attenuate the annual decline in eGFR (10). Another meta-analysis involving six randomized controlled trials further confirmed that SGLT2 inhibitors slow renal function deterioration among patients with DM and CKD (11).

According to the KDIGO guideline for the management of CKD in patients with diabetes, SGLT2 inhibitors are now recommended as the first-line therapy for all individuals with an eGFR  $\geq 20$  mL/min/1.73 m<sup>2</sup> (12). Therefore, patients with DM and/or CKD should be evaluated for the early initiation of SGLT2 inhibitors at diagnosis to mitigate the risk of renal disease progression. Large multi-center trials such as DAPA-CKD and EMPA-KIDNEY have further expanded the therapeutic scope of SGLT2 inhibitors, demonstrating significant reductions in CKD progression and albuminuria, even among non-diabetic patients (13,14). In glomerulonephritis, a study focusing on IgA nephropathy similarly reported that SGLT2 inhibitors effectively reduce proteinuria (15). In our analysis, although the number of glomerulonephritis patients was limited, these patients showed the highest albuminuria reduction rates. While SGLT2 inhibitors are increasingly incorporated into routine practice for IgA nephropathy, broader randomized prospective

studies are still needed to clarify their efficacy across other glomerulonephritis subtypes.

We also observed that among patients with heart failure (regardless of DM status) albuminuria significantly decreased and furthermore, no cases of >50% eGFR decline were identified. The DAPA-HF trial similarly showed that dapagliflozin provided superior renal outcomes compared to placebo, including reduced risk of >50% eGFR decline, end-stage kidney disease, or renal death (16). Likewise, in the EMPEROR-REDUCED trial involving 3,730 patients, empagliflozin was associated with a lesser decline in eGFR compared with placebo (17). Together, these findings support the growing evidence that the renal benefits of SGLT2 inhibitors extend to patients with heart failure and should not be overlooked in their management.

At baseline, patients using dapagliflozin and those using empagliflozin were similar in terms of demographic, clinical, and laboratory characteristics. After 12 months of treatment, both agents showed comparable efficacy, suggesting equivalent renal benefit. This is consistent with findings from Lim et al., who reported no significant difference in renal endpoints between dapagliflozin and empagliflozin (18). Another study has also demonstrated similar results between the two drugs (8). Conversely, the VERTIS-CV trial, which included non-diabetic CKD patients, found that ertugliflozin did not significantly reduce albuminuria (19). Taken together, our findings and those from the literature suggest that the renal effects of SGLT2 inhibitors are likely class effects rather than molecule-specific differences (20). However, additional head-to-head and mechanistic studies are needed to confirm this observation.

### Limitations of The Study

This study has several limitations. The retrospective design, relatively small sample size, and single-center setting may limit the generalizability of the results. The small number of glomerulonephritis cases represents another constraint. Additionally, other antidiabetic agents used by patients with DM were not recorded, which may have influenced renal outcomes. Nevertheless, the principal strength of our study lies in its inclusion of diverse patient populations and the direct comparison between dapagliflozin and empagliflozin users, providing valuable real-world evidence on the renal effects of SGLT2 inhibitors across multiple disease contexts.

## CONCLUSION

In our study, we found that SGLT2 inhibitors significantly reduced albuminuria in patients with diabetic nephropathy, heart failure, CKD, and glomerulonephritis. Furthermore, no substantial difference in efficacy was

observed between dapagliflozin and empagliflozin. Consistent with existing literature, our findings support the importance of initiating SGLT2 inhibitors in all eligible patients with appropriate indications.

## DECLARATIONS

**Ethics Committee Approval:** This study was carried out according to the ethical rules and principles of the Declaration of Helsinki. Patient data was retrospectively accessed and anonymized before analysis. Approval for the study protocol was obtained from the Ethics Committee of Gülhane Hospital (approval date: 12 September 2022; approval number: 146/24).

**Data Availability:** The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

**Competing Interests:** All authors declare that they have all participated in the design, execution, and analysis of the paper and that they have approved the final version.

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**Informed Consent:** Since the study is retrospective, informed consent form is not necessary.

**Authors' Contributions:** **MY:** Conceptualization, methodology, and writing—original draft. **EY and BIA:** Data collection and validation. **EGO:** Writing—review and editing and supervision. **MDA:** Conceptualization, project administration, and writing—review and editing. All authors read and approved the final manuscript.

**AI Assistance:** We hereby confirm that the content of this article was written entirely by the authors and does not involve the use of artificial intelligence or AI-assisted writing tools. All intellectual contributions, data analysis, and interpretations presented in this manuscript are the result of the authors' original work.

## REFERENCES

1. Afkarian M, Zelnick LR, Hall YN, et al. Clinical Manifestations of Kidney Disease Among US Adults With Diabetes, 1988-2014. *Jama*. Aug 9 2016;316(6):602-10. doi:10.1001/jama.2016.10924
2. Seyahi N, Ateş K, Süleymanlar G. *Current status of renal replacement therapy in Turkey: A summary of the Turkish society of nephrology registry report. Current Status of Renal Replacement Therapy in Turkey*. 2020;
3. Rajasekaran H, Lytvyn Y, Cherney DZ. Sodium-glucose cotransporter 2 inhibition and cardiovascular risk reduction in patients with type 2 diabetes: the emerging role of natriuresis. *Kidney Int*. Mar 2016;89(3):524-6. doi:10.1016/j.kint.2015.12.038
4. de Boer IH. Kidney disease and related findings in the diabetes control and complications trial/epidemiology of diabetes interventions and complications study. *Diabetes Care*. 2014;37(1):24-30. doi:10.2337/dc13-2113
5. Gansevoort RT, Matsushita K, van der Velde M, et al. Lower estimated GFR and higher albuminuria are associated with adverse kidney outcomes. A collaborative meta-analysis of general and high-risk population cohorts. *Kidney Int*. Jul 2011;80(1):93-104. doi:10.1038/ki.2010.531
6. Coppo R, D'Amico G. Factors predicting progression of IgA nephropathies. *J Nephrol*. Sep-Oct 2005;18(5):503-12.
7. Reichert LJ, Koene RA, Wetzels JF. Prognostic factors in idiopathic membranous nephropathy. *Am J Kidney Dis*. Jan 1998;31(1):1-11. doi:10.1053/ajkd.1998.v31.pm9428445
8. Lin YH, Huang YY, Hsieh SH, et al. Renal and Glucose-Lowering Effects of Empagliflozin and Dapagliflozin in Different Chronic Kidney Disease Stages. *Front Endocrinol (Lausanne)*. 2019;10:820. doi:10.3389/fendo.2019.00820
9. Mavrakanas TA, Tsoukas MA, Brophy JM, et al. SGLT-2 inhibitors improve cardiovascular and renal outcomes in patients with CKD: a systematic review and meta-analysis. *Sci Rep*. Sep 23 2023;13(1):15922. doi:10.1038/s41598-023-42989-z
10. Toyama T, Neuen BL, Jun M, et al. Effect of SGLT2 inhibitors on cardiovascular, renal and safety outcomes in patients with type 2 diabetes mellitus and chronic kidney disease: A systematic review and meta-analysis. *Diabetes Obes Metab*. May 2019;21(5):1237-1250. doi:10.1111/dom.13648
11. Cao H, Rao X, Jia J, et al. Effects of sodium-glucose co-transporter-2 inhibitors on kidney, cardiovascular, and safety outcomes in patients with advanced chronic kidney disease: a systematic review and meta-analysis of randomized controlled trials. *Acta Diabetol*. Mar 2023;60(3):325-335. doi:10.1007/s00592-022-01989-7
12. KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease. *Kidney Int*. Nov 2022;102(5s):S1-s127. doi:10.1016/j.kint.2022.06.008
13. Heerspink HJL, Stefánsson BV, Correa-Rotter R, et al. Dapagliflozin in Patients with Chronic Kidney Disease. *N Engl J Med*. Oct 8 2020;383(15):1436-1446. doi:10.1056/NEJMoa2024816
14. Herrington WG, Staplin N, Wanner C, et al. Empagliflozin in Patients with Chronic Kidney Disease. *N Engl J Med*. Jan 12 2023;388(2):117-127. doi:10.1056/NEJMoa2204233
15. Dong Y, Shi S, Liu L, et al. Effect of SGLT2 inhibitors on the proteinuria reduction in patients with IgA nephropathy. *Front Med (Lausanne)*. 2023;10:1242241. doi:10.3389/fmed.2023.1242241
16. McMurray JJV, Solomon SD, Inzucchi SE, et al. Dapagliflozin in Patients with Heart Failure and Reduced Ejection Fraction. *N Engl J Med*. Nov 21 2019;381(21):1995-2008. doi:10.1056/NEJMoa1911303
17. Packer M, Anker SD, Butler J, et al. Cardiovascular and Renal Outcomes with Empagliflozin in Heart Failure. *N Engl J Med*. Oct 8 2020;383(15):1413-1424. doi:10.1056/NEJMoa2022190
18. Lim J, Hwang IC, Choi HM, et al. Comparison of cardiovascular and renal outcomes between dapagliflozin and empagliflozin in patients with type 2 diabetes without prior cardiovascular or renal disease. *PLoS One*. 2022;17(10):e0269414. doi:10.1371/journal.pone.0269414
19. Cannon CP, Pratley R, Dagogo-Jack S, et al. Cardiovascular Outcomes with Ertugliflozin in Type 2 Diabetes. *N Engl J Med*. Oct 8 2020;383(15):1425-1435. doi:10.1056/NEJMoa2004967
20. Demir ME, Helvacı Ö, Yıldırım T, Merhametsiz Ö, Sezer S. Potential Role of SGLT-2 Inhibitors in Improving Allograft Function and Reducing Rejection in Kidney Transplantation. *Clin Transplant*. 2025;39(9):e70233. doi:10.1111/ctr.70233