ORIGINAL ARTICLE

Sodium–Glucose Cotransporter 2 Inhibitors in Kidney Diseases Other Than That Due to Diabetes: Benefits in Composite Renal Outcomes Driven by Immunoglobulin A Nephropathy

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Abstract

Background: Sodium–glucose cotransporter-2 inhibitors (SGLT2i) are a well-established treatment for managing diabetic kidney disease (DKD). Clinical trials, including DAPA-CKD and EMPA-KIDNEY, indicate that these drugs are probably protective for the kidneys even in cases of chronic kidney disease (CKD) without diabetes, and secondary analysis of trials suggests that the overall outcome may be driven by the benefits of IgA nephropathy (IgAN). We aimed to evaluate whether benefits in composite renal outcomes in CKD, other than those due to diabetes, are observed beyond those with IgA nephropathy.

Methods: Data were extracted from the clinical trials DAPA-CKD and EMPA-KIDNEY, including patients with and without diabetes and CKD. Kidney diseases were classified as hypertensive/renovascular nephropathy or glomerular diseases, further subdivided into IgAN, focal segmental glomerulosclerosis (FSGS), and other glomerulonephritis. The heterogeneous group labeled as other/unknown was excluded from all analyses. The composite renal outcome was analyzed from pooled data using the Mantel-Haenszel risk ratio with a random-effects model and analyzed for all groups and then reanalyzed excluding the IgAN subgroup.

Results: The first pooled analysis, including all groups, demonstrated a 22% reduction in composite renal outcomes (RR 0.78, 95% CI 0.63–0.96, $p = 0.02$). However, when the IgAN group was excluded, the analysis revealed that the renoprotective benefits were no longer significant (RR 0.84, 95% CI 0.67–1.04, $p = 0.11$).

Conclusion: The overall benefit of SGLT2i in CKD due to causes other than diabetes, in patients with or without diabetes, may be predominantly driven by the benefits of IgAN.

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Introduction

Sodium–glucose cotransporter-2 inhibitors (SGLT2i) have established benefits in diabetic kidney disease (DKD).[1–3] The CREDENCE, DAPA-CKD, and EMPA-KIDNEY studies demonstrated that SGLT2 inhibitors slow the decline in estimated glomerular filtration rate (eGFR). It has also been suggested that renal outcomes can be improved even in individuals who do not have diabetes.[4,5] In addition to DKD, the DAPA-CKD and EMPA-KIDNEY studies included several other causes of kidney disease.

Empagliflozin led to a 29% decrease in the progression of kidney disease (HR 0.71, 95% CI 0.62–0.81) in groups such as those with DKD, hypertensive/renovascular disease, glomerular disease, and other unknown renal disorders.[3] Similarly, dapagliflozin demonstrated a notable 44% decrease in the progression of renal disease (HR 0.56, 95% CI 0.45–0.68, $p < 0.001$).[2] A recent meta-analysis that combined data from clinical trials, including EMPA-REG OUTCOME, EMPEROR-Reduced, EMPEROR-Preserved, and EMPA-KIDNEY, revealed that empagliflozin reduced the progression of CKD by 30% [HR 0.70 (0.63–0.78)] and reduced the risk of kidney failure by 34% [HR 0.66 (0.55–0.79)], independent of diabetes status and etiology of kidney disease.[6]

The major etiologies of these other forms of heterogeneous renal diseases include a few specific disorders such as hypertensive nephropathy/renovascular kidney disease and primary glomerular disorders, along with other innumerable less common entities. It remains extremely difficult to determine whether the renal benefits of SGLT2 inhibitors apply uniformly to each individual kidney disease and whether the overall benefits in composite renal outcomes are predominantly driven by their effects on a particular renal condition.

In the EMPA-KIDNEY and DAPA-CKD studies, there was a significantly heterogeneous group of patients labeled as "unknown/others" with unclassified renal disease. The diagnosis of the underlying cause of kidney disease was not always confirmed through biopsy and was based on the investigators' clinical hunch and possibly included atypical and undiagnosed DKD, as well as other renal conditions.

A predetermined analysis of IgA nephropathy (IgAN) from the DAPA-CKD trial reported a significant 76% increase.[7] These observations raise the question of whether the renoprotective benefits seen in composite analyses of these heterogeneous groups of kidney diseases, irrespective of type 2 diabetes (T2DM) status, are driven primarily by the benefits derived in patients with IgAN. It is also likely that other forms of renal disease such as focal segmental glomerulosclerosis (FSGS), other forms of glomerulonephritis, and hypertensive/renovascular nephropathy might not achieve a statistically significant and clinically meaningful reduction in the primary renal outcome (HR 0.24, 95% CI 0.09–0.65, *p*-value 0.002).

In this context, we investigated whether SGLT2 inhibitors provide benefits across a broad spectrum of kidney diseases, including focal segmental glomerulosclerosis (FSGS), other glomerulopathies, and hypertensive/renovascular nephropathy, once we excluded IgAN, where benefits are highly likely. Additionally, we excluded the large group of unknown/other kidney diseases, as that group was a mixed bag of less common diseases with varied pathogenesis, from all the analyses.

Methods

For data extraction, we included only those RCTs in which SGLT2 inhibitors reported composite renal outcomes in individuals with kidney disease, regardless of whether they had diabetes, and in which the cause of

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renal disease other than DKD was reported. Therefore, we included DAPA-CKD and EMPA-KIDNEY and excluded CREDENCE, as it did not report the causes of kidney disease other than diabetes mellitus.

Data were collected by 2 independent reviewers, SR and MB. Any disagreements in the data extraction process were addressed through discussions among the investigators and consultations with the other 2 team members (PSM and SG).

We reviewed data from both primary and secondary analyses of the DAPA-CKD[2,4,7,8] and EMPA-KIDNEY[3,5] trials. We included data from subjects with CKD with or without T2DM and any form of kidney disease. The cause of kidney disease, other than possible DKD, was based on either (1) histopathological diagnosis of any renal involvement other than diabetes, (2) investigator-presumed renal disease other than that due to diabetes, or (3) investigator-suspected unknown causes of kidney disease.

The composite renal outcome was summarized as follows: (1) hypertensive/renovascular nephropathy, (2) glomerular disease: (a) IgAN, (b) FSGS, and (c) other glomerulonephritis, and (3) other/unknown kidney diseases (unclassified) from secondary analysis of EMPA-KIDNEY[5] and prespecified analyses of DAPA-CKD.[4,7,8] We calculated data for the other glomerulonephritis group in the DAPA-CKD study by subtracting the number of IgAN and FSGS patients from all patients with glomerulonephritis, as these data were not directly available.

In both EMPA-KIDNEY and DAPA-CKD studies, the primary composite renal outcome was consistently defined as either a sustained reduction in eGFR (≥40% for EMPA-KIDNEY and ≥50% for DAPA-CKD), progression to ESKD, characterized by ongoing dialysis, kidney transplantation, or an eGFR of <15 mL/minute/1.73 m^2 in DAPA-CKD and <10 mL/minute/1.73 m^2 in EMPA-KIDNEY, or death related to kidney issues.

Thereafter, for the sake of analysis, we excluded those in the other/unknown kidney disease group, as this group was extremely heterogeneous, with numerous presumed, documented, and unknown etiologies, where it is extremely difficult to explain the pathophysiological basis of the benefit of SGLT2i use. It should be noted that this heterogeneous group included a significant proportion of patients with diabetes (26.65%),[9] which could explain some renal benefits and may not reflect renal benefits due to causes other than diabetes. The original investigators in the DAPA-CKD clinical trial excluded 11 patients with FSGS from the analysis, as the diagnosis of FSGS can be made with certainty only with histopathological confirmation.[8]

First, we analyzed composite renal outcomes, including all patients with renal disease due to causes other than diabetes. In this analysis, we did not include those for whom the cause was labeled as other/unknown, for the reasons explained above. As our hypothesis was that the entire benefit of SGLT2i in kidney disease was driven primarily by IgAN, we reanalyzed the composite renal outcomes excluding IgAN to assess whether the benefits of SGLT2i were demonstrated in participants with kidney disease beyond IgAN.

Statistical Analysis

To calculate the pooled estimates of effect size, we used the relative risk, with corresponding 95% confidence intervals, derived using the Mantel–Haenszel approach, as data were collected from various primary and secondary analyses. For this analysis, a random-effects model was applied to the subgroups of kidney disease.

We evaluated statistical heterogeneity using the I^2 and χ^2 tests developed by Higgins and Thompson. Heterogeneity was categorized as high when I^2 was ≥75%, moderate when I^2 was ≥50%, and low when I^2 was ≤25%. Although the χ^2 test is not as sensitive in detecting heterogeneity, a *p*-value of <0.05 was used as a threshold to reject the assumption of homogeneity or to support the presence of heterogeneity. Statistical analyses were performed using Review Manager (RevMan) version 5.4.1, provided by The Cochrane Collaboration (2020).

Results

This meta-analysis categorized CKD into 4 distinct types based on the DAPA-CKD and EMPA-KIDNEY trials: hypertensive/renovascular nephropathy, IgAN, FSGS, and other forms of glomerulonephritis, excluding IgAN and FSGS. The study included data from 2,132 participants with hypertensive/renovascular nephropathy, 1,087 with IgAN, 299 with FSGS, and 967 with other types of glomerulonephritis. Overall, the meta-analysis included 4,485 participants. The baseline characteristics are comprehensively outlined in Table 1. The renal outcomes of SGLT2i in each CKD category for patients with and without T2DM are summarized in Table 2.

The pooled Mantel–Haenszel risk ratio for composite renal outcomes across all 4 SGLT2i-treated kidney disease groups was 0.78 (95% CI 0.63–0.96; $p = 0.02$). These findings are illustrated in Figure 1.

The Mantel–Haenszel risk ratio for combined renal outcomes, excluding IgAN, among the other 3 categories, hypertensive/renovascular nephropathy, FSGS, and other types of glomerulonephritis excluding IgAN and FSGS, treated with SGLT2i was 0.84 (95% CI 0.67–1.04; $p = 0.11$). Figure 2 provides a representation of these results.

Discussion

According to the KDIGO 2024 guidelines, it is recommended to treat adults with CKD with an SGLT2i who have an eGFR of 20 mL/minute/1.73 m^2 or higher, along with a uACR of at least 200 mg/gm (20 mg/mmol), or those suffering from heart failure, irrespective of their albuminuria levels. The level of evidence recommendation is very robust (level 1A).[10]

These recommendations are based on data published in secondary analyses of the DAPA-CKD and EMPA-KIDNEY trials.[4,5] It should be emphasized that not all patients had a histopathologically confirmed diagnosis. Only 1,508 (42%) and 500 (36%) patients without diabetes had biopsy-proven categorized renal disease in the EMPA-KIDNEY and DAPA-CKD trials, respectively.[9] In a large proportion of patients, the subclassification of renal disease was based on the assumption of the investigator or the treating clinician. A significant proportion of participants had diabetes, which could have driven the benefits of the composite renal outcome in the entire population. These limitations may have biased the final outcomes.

In both trials, there was a significantly large population of subjects with a mixed bag of renal diseases termed others/unknown diseases. This extremely heterogeneous group consists of innumerable entities, including familial nephropathy, obstructive nephropathy, chronic pyelonephritis, and many others, including unknown causes of nephropathy. Even this unknown kidney disease comprised 629 (43.7%) and 214 (51.9%) patients among the heterogeneous others/unknown group in the EMPA-KIDNEY and DAPA-CKD trials, respectively.[5,11]

Moreover, 27.4% of patients had diabetes in this combined, complex, and confusing others/unknown kidney disease group in the DAPA-CKD and EMPA-KIDNEY trials. There may be spillover benefits of SGLT2 inhibition, as diabetes mellitus increases SGLT2 expression. Even if SGLT2 inhibition is beneficial in some of these rare causes of kidney disease, the pathophysiological basis of such benefits remains unexplained. Therefore, dedicated RCTs are needed in patients with renal disease without diabetes.

Table 1: Baseline characteristics of patients included in meta-analysis

Characteristics	*Hypertensive/renovascular nephropathy*		*IgAN*		*FSGS*		*Other glomerulonephritis except IgAN and FSGS*	
	EMPA-KIDNEY	*DAPA-CKD**	*EMPA-KIDNEY*	*DAPA-CKD*	*EMPA-KIDNEY*	*DAPA-CKD*	*EMPA-KIDNEY*	*DAPA-CKD**
Number of subjects	1445	687	817	270	195	104	657	310
Age in year (SD)	68.4 (11.9)		50.6 (12.7)	51.2 (13.1)	54.3 (14.5)	54 (14.3)	56.9 (13.7)	
Sex, female (%)	430 (29.8)		282 (34.5)	88 (32.6)	67 (34.4)	34 (32.7)	247 (37.6)	
Race, *n* (%)								
White	953 (66)		361 (44.2)	108 (40)	123 (63.1)	58 (55.8)	281 (42.8)	
Black	85 (5.9)		1 (0.1)	1 (0.4)	10 (5.1)	7 (6.7)	11 (1.7)	
Asian	387 (26.8)		442 (54.1)	159 (58.9)	59 (30.3)	30 (28.8)	362 (55.1)	
Other	20 (1.4)		13 (1.6)	2 (0.7)	3 (1.5)	9 (8.7)	3 (0.5)	
Prior T2DM (%)	397 (27.5)	200 (29.1)	58 (7.1)	38 (14.1)	33 (16.9)	20 (19.2)	77 (11.7)	37 (11.9)
Systolic blood pressure, mm Hg (SD)	138 (18.4)		131.8 (15.1)	127.4 (15.1)	131.9 (15.9)	128.2 (14.9)	134.3 (16.9)	
Diastolic blood pressure, mm Hg (SD)	78 (12.2)		82.5 (10.4)	79.1 (11.0)	79.8 (10.2)	75.9 (9.3)	82.0 (11.4)	
BMI in kg/m^2	30.0 (6.3)		26.8 (5.5)	27 (5.3)	30.3 (6.9)	29.6 (6.1)	26.7 (5.6)	
eGFR in mL/minute/1.73 m^2 body surface area (SD)	35.1 (11.6)		43.3 (17.5)	43.8 (12.2)	40.9 (17.5)	41.9 (11.5)	41.9 (18.4)	
UACR mg/gm Median (IQR)	11418–623		662 (331–1265)	900 (539.6–1515.0)	902 (426–1997)	1248 (749–2211)	693 (264–1509)	

*Data not available in public domain

Table 2: Renal outcome of individual CKD category in patients (with or without T2DM) with SGLT2i

Type of kidney disease (% of patients with diabetes)		*Trial name*	*SGLT2i*		*Placebo*		*HR (95%CI)*
			Events	*Total*	*Events*	*Total*	
Hypertensive/renovascular ds (28%)		EMPA-KIDNEY	72	706	87	739	0.79 (0.58–1.08)
		DAPA-CKD	18	324	26	363	0.74 (0.4–1.36)
		Total	90	1030	113	1102	
Glomerular diseases (11.18%)	IgAN (8.83%)	EMPA-KIDNEY	51	413	67	404	**0.67 (0.46–0.97)**
		DAPA-CKD	5	137	20	133	**0.24 (0.09–0.65)**
		Total	56	550	87	537	
	FSGS (17.73%)	EMPA-KIDNEY	17	98	13	97	1.35 (0.65–2.81)
		DAPA-CKD	4	45	6	59	0.67 (0.19–2.44)
		Total	21	143	19	156	
	Other glomerulonephritis (11.79%)	EMPA-KIDNEY	47	342	59	315	0.78 (0.53–1.16)
		DAPA-CKD	12	153	19	157	0.65 (0.33–1.29)
		Total	59	495	78	472	
Other/unknown kidney disease (unclassified) (26.65%)		EMPA-KIDNEY	60	713	89	725	**0.67 (0.48–0.92)**
		DAPA-CKD	10	214	14	198	0.81 (0.35–1.83)
		Total	70	927	103	923	

In a prespecified analysis, the DAPA-CKD trial demonstrated a notable 76% reduction in the composite renal outcome (HR 0.24, 95% CI 0.09–0.65, *p*-value 0.002) for IgAN, indicating the advantages of SGLT2i in treating IgAN. Patients with IgAN experienced a dramatic reduction in the risk of adverse renal outcomes, and only 4% of patients on dapagliflozin reached the primary endpoint compared to 15% in the placebo group. At the 4 month mark, the average percentage change in UACR was −35% (95% CI −51% to −18.9%; $p < 0.001$) when comparing dapagliflozin with placebo, which was sustained throughout.[7] Taken together, these results, including a substantially reduced rate of eGFR decline (2.4 mL/minute/1.73 m^2 annually after excluding the first 2 weeks), highlight the significant therapeutic impact of SGLT2 inhibition in IgAN. It is also worth exploring SGLT2 expression in IgAN, given the significant benefits of SGLT2 inhibition. In a recent publication using in silico analysis, dapagliflozin was found to have immunological benefits in patients with IgAN. The researchers demonstrated that the genes LCN2 and AGER are colocalized and linked to the drug dapagliflozin and the

Study or Subgroup	SGLT2i Events	SGLT2i Total	Placebo Events	Placebo Total	Weight	Risk ratio M-H, Random, 95% CI
Hypertensive/Renovascular Nephropathy	90	1030	113	1102	33.6%	0.85 [0.65, 1.11]
IgAN	56	550	87	537	27.6%	0.63 [0.46, 0.86]
FSGS	21	143	19	156	11.2%	1.21 [0.68, 2.15]
Other GN except IgAN and FSGS	59	495	78	472	27.6%	0.72 [0.53, 0.99]
Total (95% CI)		**2218**		**2267**	**100.0%**	**0.78 [0.63, 0.96]**
Total events	226		297			

Heterogeneity: $Tau^2 = 0.02$; $Chi^2 = 4.66$, df = 3 ($p = 0.2$); $I^2 = 36\%$
Test for overall effect: $Z = 2.33$ ($p = 0.02$)

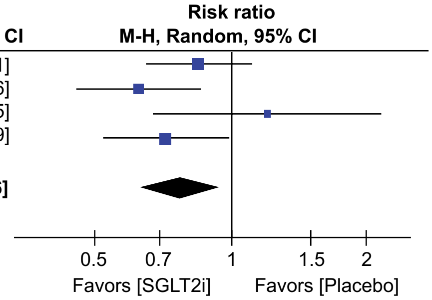

Fig. 1: Effect of SGLT2i on composite renal outcome in patients with CKD other than DKD, with or without T2DM

Study or Subgroup	SGLT2i Events	SGLT2i Total	Placebo Events	Placebo Total	Weight	Risk ratio M-H, Random, 95% CI
Hypertensive/Renovascular Nephropathy	90	1030	113	1102	49.2%	0.85 [0.65, 1.11]
FSGS	21	143	19	156	13.0%	1.21 [0.68, 2.15]
Other GN except IgAN and FSGS	59	495	78	472	37.7%	0.72 [0.53, 0.99]
Total (95% CI)		**1668**		**1730**	**100.0%**	**0.84 [0.67, 1.04]**
Total events	170		210			

Heterogeneity: $Tau^2 = 0.01$; $Chi^2 = 2.41$, df = 2 ($p = 0.30$); $I^2 = 17\%$
Test for overall effect: $Z = 1.61$ ($p = 0.11$)

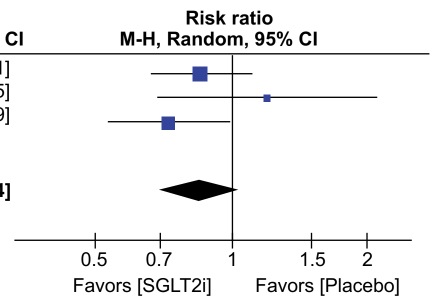

Fig. 2: Effect of SGLT2i on composite renal outcome in patients with CKD other than DKD, with or without T2DM, excluding IgAN

occurrence of IgAN. LCN2 is recognized as a risk factor, whereas AGER plays a protective role in patients with IgAN. According to the Kyoto Encyclopedia of Genes and Genomes analysis, LCN2 is part of the interleukin-17 immune signaling pathway, whereas AGER is linked to the neutrophil extracellular traps immune signaling pathway. However, no positive colocalization results were observed for the target genes with the other 2 SGLT2 inhibitors, canagliflozin and empagliflozin, in relation to IgAN occurrence.[12]

Our research revealed a statistically significant 22% decrease in composite renal outcomes for kidney diseases, even without DKD, with or without diabetes, with the use of SGLT2 inhibitors. However, this benefit was driven predominantly by the benefits in IgAN, and the benefits were no longer significant after excluding IgAN from the analysis.

This suggests that patients with other non-IgAN etiologies, such as FSGS, other forms of glomerulonephritis, and hypertensive/renovascular nephropathy, may not benefit from SGLT2 inhibition.

Dapagliflozin failed to reduce proteinuria significantly in a small cohort of 58 nondiabetic patients with IgAN, FSGS, hypertensive nephropathy, and other kidney disease in the DIAMOND trial.[13] This finding raises questions about the benefits across all CKD subtypes. The ADAPT trial is currently underway to evaluate the renal benefits of dapagliflozin in terms of the reduction of proteinuria and improvement in eGFR in nondiabetic stage 4 CKD patients. The results of this study would provide greater insights.

The potential limitations of this study are worth discussing. The meta-analysis relied on subgroup data from large trials, and inherent limitations, such as sample size and diversity within patient groups, might have influenced the robustness of subgroup conclusions. Moreover, differences in study design, baseline characteristics, and follow-up duration between the DAPA-CKD and EMPA-KIDNEY trials could contribute to variability in the observed treatment effects. Future prospective trials focusing on specific subtypes will help to address these uncertainties.

Conclusion

Our meta-analysis, which integrated data from the DAPA-CKD and EMPA-KIDNEY studies, revealed that the overall kidney protective effect of SGLT2 inhibitors in CKD not related to DKD was largely driven by the benefits of IgAN. We found an overall significant benefit of 22% in preventing the renal composite outcome in pooled data. However, this benefit disappears when patients with IgAN are excluded from the study. While the future of SGLT2i in IgAN looks promising, caution should be exercised when using these drugs in CKD other than IgAN. Further targeted studies are warranted to optimize treatment strategies for patients with CKD other than IgAN to help individualize treatment options. Until then, blanket recommendations for the use of SGLT2i in CKD, other than diabetes, should be viewed with caution.

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References

1. Perkovic V, Jardine MJ, Neal B, et al. Canagliflozin and renal outcomes in type 2 diabetes and nephropathy. N Engl J Med 2019;380:2295–2306.
2. Heerspink HJL, Stefánsson BV, Correa-Rotter R, et al. Dapagliflozin in patients with chronic kidney disease. N Engl J Med 2020;383:1436–1446.
3. The EMPA-KIDNEY Collaborative Group, Herrington WG, Staplin N, et al. Empagliflozin in patients with chronic kidney disease. N Engl J Med 2023;388:117–127.
4. Wheeler DC, Stefánsson BV, Jongs N, et al. Effects of dapagliflozin on major adverse kidney and cardiovascular events in patients with diabetic and non-diabetic chronic kidney disease: a prespecified analysis from the DAPA-CKD trial. Lancet Diabetes Endocrinol 2021;9:22–31.
5. Abat S, Abd Rahman R, Abdul Cader R, et al. Impact of primary kidney disease on the effects of empagliflozin in patients with chronic kidney disease: secondary analyses of the EMPA-KIDNEY trial. Lancet Diabetes Endocrinol 2024;12:51–60.
6. Herrington WG, Che ZJ, Sardell R, et al. Effects of empagliflozin on conventional and exploratory acute and chronic kidney outcomes: an individual participant-level meta-analysis. Lancet Diabetes Endocrinol 2025.
7. Wheeler DC, Toto RD, Stefánsson BV, et al. A pre-specified analysis of the DAPA-CKD trial demonstrates the effects of dapagliflozin on major adverse kidney events in patients with IgA nephropathy. Kidney Int 2021;100:215–224.
8. Wheeler DC, Jongs N, Stefansson BV, et al. Safety and efficacy of dapagliflozin in patients with focal segmental glomerulosclerosis: a prespecified analysis

of the dapagliflozin and prevention of adverse outcomes in chronic kidney disease (DAPA-CKD) trial. Nephrol Dial Transplant 2022;37:1647–1656.
9. Herrington WG, Wanner C, Green JB, et al. Design, recruitment, and baseline characteristics of the EMPA-KIDNEY trial. Nephrol Dial Transplant 2022;37:1317–1329.
10. Stevens PE, Ahmed SB, Carrero JJ, et al. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int 2024;105:S117–S314.
11. Wheeler DC, Stefansson BV, Batiushin M, et al. The dapagliflozin and prevention of adverse outcomes in chronic kidney disease (DAPA-CKD) trial: baseline characteristics. Nephrol Dial Transplant 2020;35:1700–1711.
12. Lv X, Shang Y, Ning Y, et al. Pharmacological targets of SGLT2 inhibitors on IgA nephropathy and membranous nephropathy: a mendelian randomization study. Front Pharmacol 2024;15:1399881.
13. Cherney DZI, Dekkers CCJ, Barbour SJ, et al. Effects of the SGLT2 inhibitor dapagliflozin on proteinuria in non-diabetic patients with chronic kidney disease (DIAMOND): a randomized, double-blind, crossover trial. Lancet Diabetes Endocrinol 2020;8:582–593.