

Review

SGLT2 Inhibitors as Nephroprotective Agents in Kidney Transplant Recipients: A Narrative Review

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Sodium-glucose cotransporter-2 (SGLT2) inhibitors have gained interest for their glycemic control and are now being investigated for cardiorenal benefits. This non-systematic literature review evaluates current evidence on the use and effects of SGLT2 inhibitors in kidney transplant recipients (KTR). A narrative literature search of PubMed/MEDLINE and Embase was conducted; publications reporting SGLT2 inhibitor use in adult KTR were included: randomized controlled trials, systematic reviews or meta-analyses, and multiple prospective and retrospective cohort studies. Included studies in this review report associations between SGLT2 inhibitor use and renal, cardiovascular, and metabolic outcomes, including lower rates of adverse cardiovascular events, improved glycemic control, reduced body mass index, blood pressure reduction confined to hypertensive recipients, and stable short-term allograft function. Because this data is predominantly observational, with modest sample sizes and follow-up frequencies, causal inference is not warranted and nephroprotective efficacy in this population remains unestablished. Further research and additional randomized controlled trials are needed to better understand the long-term effects of SGLT2 inhibitors in post-renal transplant patients.

1. INTRODUCTION

Kidney transplantation remains the definitive therapy for end-stage renal disease, conferring improved survival, reduced dialysis dependence, and lower healthcare costs.¹ In the United States, 28,142 kidney transplants were performed in 2023, reflecting a recent peak in activity.¹ However, more recent data from the United Network of Organ Sharing (UNOS) suggests a slight decline, with 27,573 kidney transplants performed in 2025 (21,052 from deceased donors and 6,521 from living donors).² Despite these fluctuations, more than 90,000 individuals in the United States remain on the waiting list.² Chronic kidney disease (CKD), a principal cause of transplantation, affects approximately 850 million people worldwide and is projected to become the fifth leading cause of death by 2050.³ Patients with end-stage renal disease on dialysis experience high morbidity and mortality, particularly among older adults.⁴

Kidney transplant recipients (KTR) face delayed graft function, rejection, surgical complications, and infections, with opportunistic infection risk highest during the first 12 months.⁵ Many factors contribute to complications, such as specific donor/recipient factors, preexisting infections, immunity, use of antimicrobial prophylaxis, and immunosuppression state.^{5,6} Maintenance immunosuppression combines calcineurin inhibitors, antimetabolites, corticosteroids, mTOR inhibitors, and costimulation blockers.⁶ Corticosteroids and calcineurin inhibitors drive post-transplant diabetes mellitus through insulin resistance and

beta-cell toxicity, which contributes to cardiovascular risk.^{6,7}

SGLT2 mediates about 80 to 90% of filtered glucose reabsorption in the proximal tubule, cotransported with sodium.⁸ Its inhibition produces glucosuria and increases distal sodium delivery to the macula densa, restoring tubuloglomerular feedback and lowering intraglomerular pressure.⁸ In addition, its effects include osmotic diuresis and natriuresis, reduced proximal tubular oxygen consumption, and a shift toward ketogenesis and fatty acid oxidation for energy production.⁸ The cardiorenal benefit demonstrated in outcome trials appears to be largely independent of glucose-lowering, which is the mechanistic rationale for the potential benefits of SGLT2 inhibitors in non-diabetic patients.^{7,8} Beyond their glucose-lowering effects, SGLT2 inhibitors have been associated with cardioprotective and nephroprotective benefits in patients with type 2 diabetes and chronic kidney disease.^{7,8} However, KTR were excluded from pivotal cardiorenal outcome trials because evidence is insufficient and immunosuppressed patients may be more vulnerable to infection.⁹ Therefore, this study aims to evaluate the potential nephroprotective role of SGLT2 inhibitors in KTR.

2. METHODS

This manuscript is a narrative review examining the role of SGLT2 inhibitors in adult KTR. The review was conducted through a narrative rather than systematic design due to

the nature of currently published evidence, consisting of observational cohort studies that have a high degree of variability among variable definitions, as well as variable follow-up. PubMed/MEDLINE and Embase were searched for articles published between 2016 and 2026, last executed in August 2026. PubMed was searched using the following MeSH: (“SGLT2 inhibitors” or “sodium-glucose cotransporter 2 inhibitors” or empagliflozin or dapagliflozin or canagliflozin or ertugliflozin) and (“kidney transplantation” or “kidney transplant recipients” or “renal transplant” or “post-transplant diabetes mellitus”). Embase was searched using equivalent terms and keywords: (“SGLT2 inhibitors” or empagliflozin or dapagliflozin or canagliflozin or ertugliflozin) and (“kidney transplantation” or “kidney transplant recipients” or “renal transplant” or “post-transplant diabetes mellitus”). Additional relevant studies were identified through manual screening of reference lists from key articles.

The search yielded 159 records from PubMed and 472 from Embase (631 total). After removing duplicates, titles and abstracts were screened for relevance. Full-text articles were assessed against the following eligibility criteria: (1) reported SGLT2 inhibitor use in adult (≥ 18 years) KTR, diabetic and/or non-diabetic; (2) included at least one outcome of interest (metabolic, renal, cardiovascular, or safety); (3) published in English in a peer-reviewed journal. Studies were excluded if the population did not include KTR, reported non-kidney or multi-organ transplantation, included only pediatric populations, or reported overlapping cohorts. After screening, 14 publications reporting SGLT2 inhibitor use in adult kidney transplant recipients met inclusion criteria: two randomized controlled trials, one systematic review/meta-analysis, three non-quantitative reviews, one prospective multicenter cohort, and seven retrospective cohort or registry-based studies. Additional references providing epidemiologic, mechanistic, guideline, and safety context were included and are not counted among the above.

Consistent with the narrative design in which this manuscript was formatted, no formal risk-of-bias instrument was implemented, excluding study quality as an eligibility criterion. Randomized controlled trials and analyses accounting for confounding were prioritized over single-center studies. Outcomes of the studies selected were synthesized narratively into metabolic, renal, cardiovascular/hemodynamic, and safety categories; no quantitative pooling was performed.

3. RESULTS

3.1. RANDOMIZED EVIDENCE

In a single-center, prospective, double-blind study, 49 KTR with post-transplant diabetes mellitus received empagliflozin 10mg daily or placebo for 24 weeks.¹⁰ HbA1c fell by a median of 0.2% with empagliflozin versus 0.1% with placebo ($p = 0.025$). The magnitude of glucose reduction was dependent on GFR and baseline HbA1c. In addition, the study showed a significant reduction in body weight of

2.5kg versus a 1kg increase in the placebo group ($p = 0.014$). In addition, there were no significant differences in adverse effects, immunosuppressive drug levels, or eGFR.¹⁰

An additional randomized, double-blind, placebo-controlled mechanistic trial enrolled 52 KTR to assess SGLT2 inhibitors' physiologic effects.¹¹ Cardiovascular and kidney protective mechanisms were assessed over 12 weeks; participants received dapagliflozin 10mg or placebo. Dapagliflozin did not lower systolic blood pressure but reduced mean arterial pressure at 1 week (-3.9 mmHg; 95% CI -7.5 to -0.2). Dapagliflozin led to placebo-adjusted reductions in iothexol-measured GFR from 4.2 mL/min per 1.73 m² at 1 week to 3.49 mL/min/ 1.73 m² (95% CI -6.33 to -0.64) at 12 weeks.¹¹

3.2. METABOLIC OUTCOMES

A systematic review and meta-analysis of KTR was associated with a significant reduction in HbA1c (-0.56% , $p = 0.007$) and body weight (-2.16 kg, $p < 0.001$) with SGLT2 inhibitor therapy.¹² A meta-analysis, comprising 21 SGLT2 inhibitor studies in 3,856 KTR, reported weight loss (SMD -0.59 ; 95% CI -1.04 to -0.15), HbA1c reduction (MD -0.33% ; 95% CI -0.55 to -0.12), increased serum magnesium, reduced uric acid, and stable kidney function.¹² In a retrospective cohort study of patients with post-transplant diabetes mellitus, Navarrete et al.¹³ reported a reduction in body mass index from 27.27 to 25.95 kg/m² ($p < 0.001$). Although HbA1c decreased from 7.22% to 7.01% , the change was not statistically significant ($p = 0.558$). In contrast, fasting plasma glucose increased from 112.6 to 125.0 mg/dL ($p = 0.03$). Lipid parameters, including total cholesterol and LDL cholesterol, were also reduced in patients receiving combination therapy with GLP-1. In a single-center cohort of 50 KTR, insulin requirements decreased by 3.7 units (SD 22.8 , $p = 0.17$), a non-significant change.¹⁴

Renal function remained stable across studies, with no significant changes in eGFR, serum creatinine, or proteinuria.¹² In a single-center retrospective study including 58 non-diabetic KTR, Rodríguez-Espinosa et al.¹⁵ reported a reduction in the rate of eGFR decline from -3.5 to -1.0 mL/min/ 1.73 m²/year following SGLT2 inhibitor initiation, with no significant change in urinary albumin-to-creatinine ratio ($p = 0.067$). A multicenter cohort reported approximately 15% proteinuria reduction after three months of SGLT2 inhibitor therapy, with greater reductions among patients with higher baseline proteinuria.¹⁶ In a separate retrospective cohort study, kidney allograft function remained stable following SGLT2 inhibitor initiation, with no significant changes in eGFR at three months (-1 mL/min/ 1.73 m², $p = 0.8831$) or six months ($+1$ mL/min/ 1.73 m², $p = 0.1478$).

In a multicenter cohort of 2,083 diabetic KTR followed for a mean of 62.9 ± 42.2 months, SGLT2 inhibitor use was associated with a lower composite of death, death-censored graft failure, and creatinine doubling (adjusted HR 0.43 ; 95% CI 0.24 - 0.78 ; propensity-matched HR 0.45 ; 95% CI 0.24 - 0.85), and an acute eGFR dip $> 10\%$ occurred in 15.6% with subsequent recovery.¹⁷

3.3. CARDIOVASCULAR AND HEMODYNAMIC OUTCOMES

Navarrete et al.¹³ reported a reduction in systolic blood pressure of approximately 6 mmHg ($p < 0.01$), with no significant change in diastolic blood pressure. In a multicenter prospective cohort of 347 KTR, Maigret et al.¹⁶ reported reductions in both systolic and diastolic blood pressure ($P < 0.00001$) after 3 months, with the antihypertensive effect confined to hypertensive recipients. Sheu et al.¹⁸ reported lower all-cause mortality, major adverse cardiovascular events, and major adverse kidney events among propensity-matched users, and Lim et al.¹⁹ reported reduced myocardial infarction and cardiovascular mortality in a propensity score-matched multicenter cohort.

3.4. SAFETY OUTCOMES

In the 347-recipient multicenter cohort, adverse events occurred in 25.9%. Bacterial urinary tract infection was the most frequent (6.6%), with pyelonephritis in 2.0%; genital mycotic infection was distinctly less common (0.6%). Treatment was discontinued in 54 recipients (15.6%), mainly for infection and graft dysfunction.¹⁶ In a prospective study of 50 KTR, urinary tract infection occurred in 14% compared to a historical rate of approximately 20%, with one genital fungal infection and no ketoacidosis, acute kidney injury, or lower extremity amputation.¹⁴ A matched analysis of 240 diabetic KTR found no excess of urinary tract infection or other adverse outcomes,²⁰ and the randomized dapagliflozin trial reported no genitourinary infections in either arm.¹¹ In a review pooling 11 early studies of 214 diabetic KTR, there were 23 urinary tract infections, 2 genital yeast infections, one acute kidney injury, one mild hypoglycemia, and no ketoacidosis or acute rejection.²¹

4. DISCUSSION

This review examines the potential association of SGLT2 inhibitors as nephroprotective and cardioprotective agents in KTR. As previously described, these agents exert their primary effect by inhibiting glucose reabsorption in the proximal convoluted tubule, leading to urinary glucose excretion. Beyond glycemic control, this mechanism contributes to downstream hemodynamic and metabolic effects associated with renal and cardiovascular protection.^{7,8} The nephroprotective effects of SGLT2 inhibitors are primarily attributed to their modulation of tubuloglomerular feedback, which reduces intraglomerular pressure and attenuates hyperfiltration.^{7,8} However, it is important to note that the mechanisms of benefit have been predominantly characterized in the general CKD population, and the extent to which they translate to KTR, who have a single functioning allograft, altered renal hemodynamics, and chronic immunosuppression, remains incompletely established.

Over recent years, the incidence of diabetes mellitus (DM) leading to end-stage kidney disease has increased, contributing to the demand for kidney replacement therapy. Glycemic control following kidney transplantation also remains a clinical challenge, particularly in the setting of

immunosuppressive therapy. Despite emerging evidence associated with the metabolic, cardiovascular, and nephroprotective properties of SGLT2 inhibitors, real-world utilization among diabetic KTR remains low, with current estimates suggesting use in only 6.5% of post-transplant patients.¹⁸ These findings suggest that SGLT2 inhibitors may represent an underutilized therapeutic option in KTR. In addition, it is important to note that the KDIGO 2022 Clinical Practice Guideline for Diabetes Management in CKD explicitly excluded KTR from its recommendation to use SGLT2 inhibitors, citing insufficient evidence on efficacy and safety in immunosuppressed populations and the potential for increasing infection risk.⁹

The two available randomized trials in KTR provide proof of concept but are limited by small samples, short follow-up, and reliance on surrogate or mechanistic endpoints. Halden et al.¹⁰ randomized 49 KTR with post-transplant diabetes mellitus (PTDM) to empagliflozin 10mg or placebo for 24 weeks; HbA1c decreased by a median of 0.2% versus 0.1% ($p = 0.025$) with a significant weight reduction of 2.5kg ($p = 0.014$), and no significant differences in adverse effects, immunosuppressive drug levels, or eGFR.¹⁰ In an additional randomized trial, 52 KTR received dapagliflozin 10mg or placebo over 12 weeks; dapagliflozin reduced mean arterial pressure at 1 week (-3.9 mmHg; 95% CI -7.5 to -0.2) and was associated with placebo-adjusted reductions in GFR of 3.49 mL/min/1.73m² (95% CI -6.33 to -0.64) at 12 weeks, consistent with expected hemodynamic effects.¹¹

Reported glycemic effects are heterogeneous, ranging from significant HbA1c and weight reduction in pooled analyses to non-significant HbA1c change in individual cohorts. These discrepancies likely reflect differences in baseline HbA1c and GFR, variability in immunosuppressive regimens, and heterogeneity in study design. That weight and metabolic benefit can occur without consistent HbA1c improvement supports the interpretation that the cardiorenal benefits of SGLT2 inhibitors in KTR, if confirmed, may be largely independent of glucose-lowering.

Renal findings are the most consistent across studies, with stabilization of graft function and reductions in proteinuria reported in diabetic and non-diabetic recipients.²² The initial transient eGFR dip described after initiation reflects reduced intraglomerular pressure and is not indicative of kidney injury; in non-transplant populations, the same phenomenon precedes slower long-term CKD progression.²³ The relative consistency of renal outcomes may reflect the direct, glucose-independent hemodynamic mechanism, which is less susceptible to confounding by immunosuppressive effects than glycemic endpoints. Nonetheless, these signals derive largely from retrospective designs and must be interpreted with caution given confounding by indication.

Beyond renal outcomes, SGLT2 inhibitors have been associated with cardioprotective effects in KTR. Sheu et al.¹⁸ reported lower rates of major adverse cardiovascular events and reduced all-cause mortality among SGLT2 inhibitor users compared to non-users. Navarrete et al.¹³ reported a reduction in systolic blood pressure of approximately

Table 1. Studies evaluating the use of SGLT2 inhibitors in kidney transplant recipients.

Reference	Design	N	Population	Follow-up	Key-outcomes
Halden et al. ¹⁰	RCT	44	KTR with PTDM	24 weeks	↓ HbA1c (−0.2%); stable eGFR; no serious AEs
Sridhar et al. ¹¹	RCT	51	KTR with PTDM	12 weeks	Stable iohexol-GFR; ↓ weight, ↓BP
Sheu et al. ¹⁸	Retrospective cohort	1,970	Diabetic KTR	Median ~ 3 years	↓ PTDM incidence (3.3% vs 8.3%); ↓ mortality (−7.46%); ↓ MACE (−9.43%); ↓ MAKE (−13.61%)
Lim et al. ¹⁹	Propensity-matched cohort	2,083	Diabetic KTR	Median ~ 5.2 years	↓ MI risk; ↓CV mortality; stable graft function
Diker Cohen et al. ²⁰	Retrospective matched cohort	240 matched pairs	Diabetic KTR	3 years	↓Renal events; adjusted HR 0.99 (NS)
Rodríguez-Espinoza et al. ¹⁵	Single center retrospective	58	Non-diabetic KTR	12 months	Slower eGFR decline ($\Delta +0.75$ mL/min/1.73 m ² /year); no significant change in UACR
Maigret et al. ¹⁶	Multicenter prospective	347	Diabetic and non-diabetic KTR	3 months	↓ proteinuria (~ 15%); ↓ BP (−5/−3 mmHg)
Navarrete et al. ¹³	Retrospective cohort	89	KTR with PTDM	12 months	↓BMI (p <0.001); ↓ LDL & total cholesterol; HbA1c statistical significant; ↑ fasting glucose

Note: AE, adverse event; CV, cardiovascular; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; KTR, kidney transplant recipient(s); MACE, major adverse cardiovascular events; MAKE, major adverse kidney events; MI, myocardial infarction; NS, not significant; PTDM, post-transplant diabetes mellitus; SGLT2 inhibitors, sodium-glucose cotransporter-2 inhibitor; UACR, urine albumin-to-creatinine ratio; UTI, urinary tract infection.

6mmHg ($p < 0.01$), with no significant change in diastolic blood pressure. Maigret et al.¹⁶ reported a reduction in systolic and diastolic blood pressure ($p < 0.00001$) after 3 months, with the antihypertensive effect confined to hypertensive KTR. Lim et al.¹⁹ reported that SGLT2 inhibitor use was associated with reduced risk of myocardial infarction and cardiovascular mortality. While these associations are encouraging, all cardiovascular findings are derived from observational studies subject to confounding by indication and should be regarded as hypothesis-generating rather than established benefits.

Because KTR requires lifelong immunosuppression, potential interactions between SGLT2 inhibitors and immunosuppressive agents are a relevant consideration. Available evidence suggests that SGLT2 inhibitors do not cause clinically significant interactions with tacrolimus, cyclosporine, or everolimus, likely owing to their minimal involvement in cytochrome P450 metabolism. Halden et al.¹⁰ reported stable immunosuppressive trough levels with empagliflozin. Nevertheless, these pharmacokinetic data are derived from small studies, and long-term interaction data remain limited.

Across studies, SGLT2 inhibitors appear well tolerated in selected KTR, though the anatomical and immunosuppression-related predisposition to infection in this population warrants caution. Bacterial urinary tract infection is the dominant adverse event and should be distinguished from

genital mycotic infection, which is uncommon and typically self-limited; discontinuation rates remain low and are driven mainly by infection or graft-dysfunction concerns. No consistent increase in acute kidney injury, diabetic ketoacidosis, or graft loss has been observed, and a matched analysis found no excess urinary tract infection, while the randomized dapagliflozin trial reported no genitourinary infections in either arm.¹¹ Data from type 2 diabetes populations show no overall increase in infection risk and possibly reduced pneumonia,²⁴ which, though not transplant-specific, is consistent with the observed profile. Additional considerations include volume depletion and euglycemic diabetic ketoacidosis; no eDKA cases have been reported in KTR studies. Reasonable practice includes monitoring renal function monthly for three months after initiation and at least quarterly thereafter, counseling on hydration, and sick-day withholding. Recipients most likely to benefit are those with established PTDM or type 2 diabetes, stable graft function ($\text{eGFR} > 30$ mL/min/1.73 m²), and cardiovascular comorbidity or proteinuria; caution is warranted with recurrent urinary infection, recent transplantation, or marginal graft function.

Adequately powered, multicenter randomized trials with hard clinical endpoints are needed to move beyond hypothesis-generating data. Priorities include: (1) diabetic versus non-diabetic recipients, the latter being the least-supported group; (2) optimal timing of initiation after

transplantation; (3) death-censored graft survival; (4) chronic rejection and any immunomodulatory effects; (5) adjudicated cardiovascular outcomes; and (6) long-term genitourinary infection risk under sustained immunosuppression.

4.1. LIMITATIONS

This manuscript is a non-systematic and narrative review. The current literature on SGLT2 inhibitors in KTR is derived predominantly from small, retrospective observational studies with substantial heterogeneity in design, patient populations, and immunosuppressive regimens. These studies are subject to confounding by indication and selection bias that cannot be fully mitigated by propensity score matching and are further constrained by short follow-up periods and a scarcity of randomized data with hard clinical endpoints such as graft survival and cardiovascular events. Consequently, the overall certainty of the current evidence is low, and the findings summarized here should be regarded as hypothesis-generating. The search implemented for this review was single-screened and not duplicated by independent reviewers, leading to an increased risk of selection and publication bias. Additional limitations of this manuscript include a lack of a formal risk-of-bias instrument and no registered protocol. Eligibility criteria and exclusion of non-English language articles and non-peer-reviewed articles further restrict available data.

5. CONCLUSION

This narrative review summarizes current evidence on the use of SGLT2 inhibitors in KTR through a non-systematic literature search. Current literature describes an increasing number of patients on renal transplant waiting lists, as well as a substantial burden of post-transplant complications, including diabetes mellitus, cardiovascular disease, and infections.

Available evidence suggests that SGLT2 inhibitors might represent a potential therapeutic option in KTR, based on reported associations with renal, cardiovascular, and metabolic outcomes. Data associate the use of SGLT2 inhibitors with reduction in blood pressure measures in hypertensive patients, allograft function stability, and lower incidence of cardiovascular events following kidney transplant. The use of SGLT2 inhibitors in the data reviewed revealed no increased risk of acute kidney injury, ketoacidosis, or graft loss. Although mechanistic pathways such as reduced renal glucose reabsorption in the proximal convoluted tubule and modulation of tubuloglomerular feedback provide biologic plausibility, most available data are derived from observa-

tional studies with limited sample sizes and short follow-up periods. Implementation of SGLT2 inhibitor therapy in this population remains low. Evidence utilized in this review consisted mostly of retrospective cohorts with limited follow-up. An important caveat to highlight is that current KDIGO guidelines do not include SGLT2 inhibitors in their recommendations for KTR.⁹ Further randomized controlled trials comparing SGLT2 inhibitors with standard therapy in post-transplant patients are needed to better define their long-term safety and efficacy. In addition, more robust evidence is required to clarify their role in routine post-transplant management.

ETHICS APPROVAL

Ethics approval was not required for this study. This is a narrative review based on previously published literature and does not involve any studies with human participants or animals performed by any of the authors.

AVAILABILITY OF DATA AND MATERIAL

This is a narrative review; all data discussed are drawn from previously published studies cited in the reference section.

COMPETING INTERESTS

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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AUTHORS' CONTRIBUTIONS

ZVM conceived the study. ZVM, CCV, and EVM collected the data. ZVM and CCV cross-checked the data. ZVM, CCV, and EVM drafted the manuscript, and all authors contributed substantially to its revision.

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