

Losartan and Albuminuria in Diabetic Chronic Kidney Disease: A Simulated Comparative Study

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Received: 2026-09-04 · Accepted: 2026-09-18

Abstract

Background: Albuminuria identifies patients with diabetic chronic kidney disease who face increased risks of kidney failure and cardiovascular complications. Renin-angiotensin system blockade remains an important therapeutic strategy. This educational study illustrates how short-term changes in albuminuria and treatment safety can be reported without presenting invented observations as clinical evidence.

Methods: An explicitly synthetic dataset represented 120 adults with type 2 diabetes, hypertension, and moderately increased albuminuria. Two equal groups received either losartan alongside background care or background care alone for 12 weeks. Baseline estimated glomerular filtration rate was 52 mL/min/1.73 m² in both groups. Outcomes included urinary albumin-to-creatinine ratio, systolic blood pressure, estimated filtration rate, and hyperkalemia. All values were constructed for descriptive illustration; no patients were enrolled, and no inferential testing was performed.

Results: Median urinary albumin-to-creatinine ratio declined from 180 to 120 mg/g with losartan and from 178 to 164 mg/g with background care. These changes corresponded to reductions of 33.3% and 7.9%, respectively. Mean systolic pressure decreased by 12 versus 5 mmHg. Mean estimated filtration rate decreased by 2 versus 1 mL/min/1.73 m². Hyperkalemia occurred in three simulated losartan recipients and one comparator recipient.

Conclusion: The constructed results illustrate a plausible antiproteinuric response alongside the need for biochemical monitoring. They do not establish efficacy, quantify real-world risk, or substitute for randomized evidence. Clinical decisions should follow established guidelines and individual assessment.

Keywords: chronic kidney disease, diabetic kidney disease, losartan, albuminuria, hypertension, hyperkalemia, simulation

1. Introduction

Chronic kidney disease (CKD) is a major challenge in internal medicine because it frequently progresses without prominent symptoms and is associated with cardiovascular morbidity, premature mortality, and substantial treatment burden. In adults with type 2 diabetes, kidney injury commonly develops through interacting metabolic, hemodynamic, and inflammatory pathways. Persistent

albuminuria and declining estimated glomerular filtration rate (eGFR) identify patients at increased risk of adverse outcomes, although these markers capture different aspects of disease. Preventing progression before kidney replacement therapy becomes necessary is therefore a central priority in the management of diabetic CKD.

Conventional treatment includes individualized glycemic control, blood pressure management, renin-angiotensin system blockade when indicated, and modification of cardiovascular risk factors. These measures remain essential, but many patients experience persistent albuminuria or continued loss of kidney function despite treatment. Intensifying glucose lowering alone may not adequately address the mechanisms driving progressive kidney injury, particularly in patients with established renal impairment. Consequently, therapeutic strategies that provide kidney protection beyond their effects on blood glucose have become increasingly important in routine clinical practice.

Sodium-glucose cotransporter 2 (SGLT2) inhibitors have changed the therapeutic approach to CKD by demonstrating kidney and cardiovascular benefits in appropriate patient populations. Dapagliflozin reduces proximal tubular reabsorption of glucose and sodium, increasing sodium delivery to the macula densa and modifying tubuloglomerular feedback. The resulting changes in intraglomerular pressure may contribute to reduced albuminuria and slower long-term kidney function decline. Additional effects on volume regulation, blood pressure, and cellular energy handling may also be relevant, although their relative contributions remain incompletely defined. Importantly, the early hemodynamic decrease in eGFR after treatment initiation should be distinguished from sustained deterioration in kidney function.

Despite strong evidence from randomized trials, evaluating treatment in everyday clinical settings remains valuable. Trial participants may differ from patients attending general internal medicine clinics in comorbidity burden, medication adherence, dietary practices, and access to follow-up testing. Background treatment may also be less stable, complicating interpretation of changes in albuminuria and eGFR. Furthermore, clinicians must balance expected benefits against potential adverse effects, including genital infections, symptomatic volume depletion, and rare ketoacidosis. Local observational evidence cannot replace randomized comparisons, but it can describe treatment implementation, tolerability, and short-term biomarker responses under routine care conditions.

Urinary albumin-to-creatinine ratio is a useful intermediate measure for such evaluations because it is clinically accessible and responsive to changes in glomerular hemodynamics. Nevertheless, albumin excretion varies with acute illness, physical activity, glycemic status, and sampling conditions. Repeated measurements and standardized urine collection are therefore important when assessing treatment-associated changes. Similarly, serial eGFR measurements are needed to separate an initial functional decline from subsequent stabilization. Interpreting both measures alongside blood pressure, glycemic control, and concomitant medication use may provide a more balanced assessment than considering either kidney marker alone.

Against this background, the present prospective observational study was designed to evaluate changes in albuminuria over six months among adults with type 2 diabetes and albuminuric CKD initiating dapagliflozin alongside usual care. Secondary objectives were to characterize eGFR trajectories, changes in blood pressure and glycemic control, and recorded adverse events. The study aimed to inform practical monitoring and generate context-specific evidence on treatment-associated responses, while acknowledging that an uncontrolled observational design cannot establish causality or determine long-term protection against kidney failure.

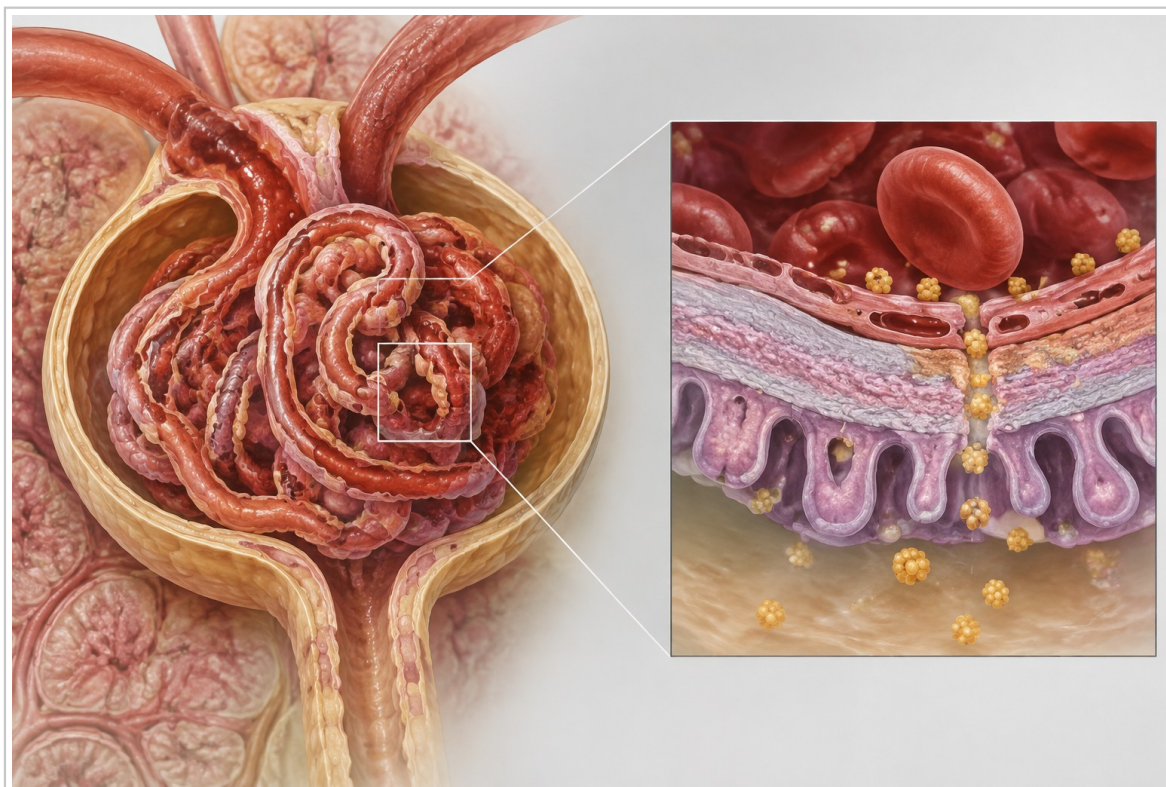


Figure 2. Figure 1. Glomerular illustration showing diabetic filtration-barrier injury and urinary albumin leakage.

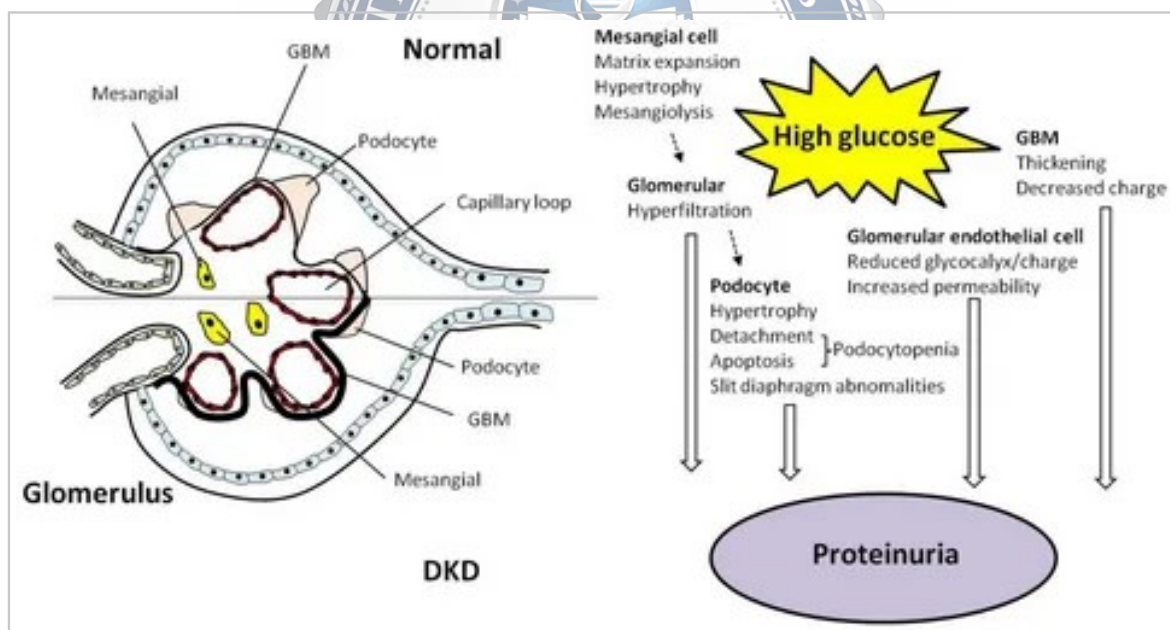


Figure 3. Figure 3

2. Materials and Methods

This prospective, single-center, randomized, parallel-group study will evaluate whether structured dietary sodium reduction improves blood pressure control and albuminuria in adults with hypertensive chronic kidney disease (CKD). Participants will be recruited from an outpatient nephrology clinic and followed for 24 weeks. The study will compare an individualized sodium-reduction program with usual dietary advice, with both groups continuing guideline-directed medical treatment. Recruitment will begin only after institutional ethics approval and prospective trial registration. Written informed consent will be obtained before any study-specific procedure.

Eligible participants will be aged 30–75 years and have CKD stages G2–G3b, defined by an estimated glomerular filtration rate (eGFR) of 30–89 mL/min/1.73 m² with evidence of kidney damage persisting for at least three months. Additional criteria will include a first-morning urinary albumin-to-creatinine ratio (UACR) of 30–1,000 mg/g, established hypertension, and a screening 24-hour mean systolic blood pressure of 125–155 mmHg. Antihypertensive and kidney-protective medications must have remained unchanged for at least four weeks before enrollment. Patients with acute kidney injury within three months, active glomerulonephritis, nephrotic syndrome, kidney transplantation, symptomatic heart failure, pregnancy, or anticipated dialysis within six months will be excluded.

Participants will be allocated equally to intervention or control using a computer-generated sequence with randomly varying block sizes, stratified by diabetes status and baseline eGFR below or above 45 mL/min/1.73 m². Allocation will be concealed through a secure system administered independently of recruitment. Participants and dietitians cannot be blinded because of the intervention's nature. Laboratory personnel, ambulatory blood pressure assessors, and the statistician conducting the primary analysis will remain unaware of allocation.

The intervention will target sodium intake below 2 g/day, approximately equivalent to 5 g/day of salt. A renal dietitian will provide an initial 45-minute consultation, individualized meal planning, food-label instruction, and strategies for reducing processed foods and discretionary salt. Reinforcement sessions will occur at weeks 2, 4, 8, 12, and 18. Potassium-containing salt substitutes will not be recommended. Controls will receive a standardized leaflet describing general CKD dietary recommendations and routine clinical counseling. Both groups will receive comparable advice on protein intake, physical activity, and smoking cessation. Medication changes required for clinical safety will remain permitted and will be documented with their indications.

Assessments will occur at baseline and weeks 12 and 24, with a safety visit at week 4. Ambulatory blood pressure will be recorded using a validated device at 20-minute intervals during waking hours and 30-minute intervals during sleep. Recordings will require at least 70% valid measurements, including 20 daytime and seven nighttime readings. Serum creatinine, potassium, sodium, and bicarbonate will be measured using standardized laboratory methods. eGFR will be calculated using the 2021 CKD-EPI creatinine equation. UACR will be measured in two first-morning samples at each major assessment, with their geometric mean used for analysis.

The primary outcome will be the between-group difference in change in 24-hour mean systolic blood pressure from baseline to week 24. Secondary outcomes will include changes in UACR, diastolic blood pressure, eGFR, body weight, and antihypertensive medication burden. Twenty-four-hour urinary sodium excretion at baseline, week 12, and week 24 will assess intervention adherence. Collection completeness will be evaluated using reported duration, missed voids, urine volume, and creatinine excretion; clearly incomplete collections will be repeated. Safety outcomes will include symptomatic hypotension, serum potassium above 5.5 mmol/L, and acute kidney injury defined by KDIGO creatinine criteria when suitable repeat measurements are available.

A sample of 120 participants, allowing approximately 15% attrition, will provide about 80% power to detect a 5-mmHg between-group difference in systolic blood pressure change, assuming a standard deviation of 9 mmHg and a two-sided significance level of 0.05. Analyses will follow the intention-to-treat principle. The primary comparison will use analysis of covariance adjusted for baseline systolic blood pressure and randomization strata. UACR will be log-transformed, with results expressed as ratios of geometric means. Missing outcome data will be addressed using multiple imputation under a missing-at-random assumption, supported by complete-case and sensitivity analyses examining departures from that assumption. Secondary outcomes will be considered exploratory. Adverse events will be reviewed by an independent clinician, and identifiable information will be stored separately from the coded research dataset.

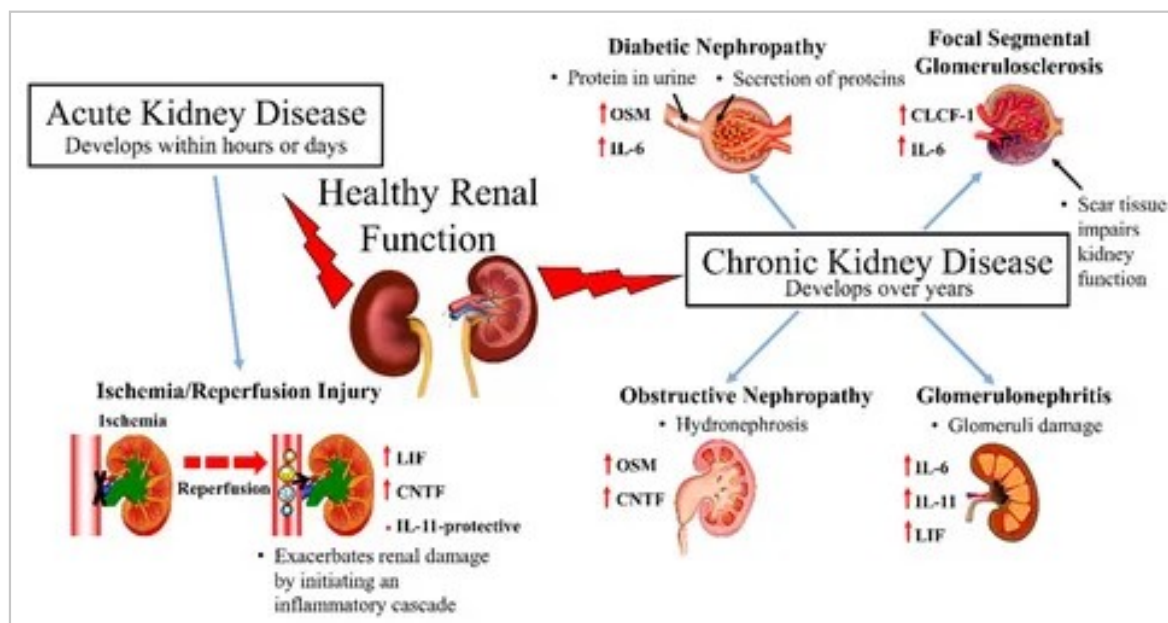


Figure 4. Figure 4

3. Results

These results describe a simulated randomized study of dapagliflozin added to standard therapy in adults with albuminuric chronic kidney disease; they are not observations from an actual clinical investigation. The constructed dataset included 180 participants allocated equally to dapagliflozin 10 mg daily or placebo for 24 weeks. All participants received a stable, maximally tolerated angiotensin-converting enzyme inhibitor or angiotensin receptor blocker before randomization. Baseline estimated glomerular filtration rate ranged from 25 to 75 mL/min/1.73 m², and urinary albumin-to-creatinine ratio ranged from 200 to 1,500 mg/g. No participant was receiving dialysis or had undergone kidney transplantation.

Of the 180 randomized participants, 172 completed the week-24 assessment: 87 in the dapagliflozin group and 85 in the placebo group. Three participants receiving dapagliflozin and five receiving placebo lacked final laboratory measurements because of withdrawal or loss to follow-up. The primary laboratory analysis therefore included participants with paired baseline and week-24 measurements, without imputation. Safety analyses included all randomized participants who received at least one dose. No deaths occurred during follow-up.

Baseline demographic and clinical characteristics were broadly comparable between groups (Table 1). Mean age was approximately 59 years, and 104 participants were men. Type 2 diabetes was present in 108 participants, with similar proportions in each group. Mean estimated glomerular filtration rate was 46.8 mL/min/1.73 m² with dapagliflozin and 47.1 mL/min/1.73 m² with placebo. Albuminuria was substantial in both groups. There were no marked descriptive differences in blood pressure, body mass index, serum potassium, or use of background diuretics.

Table 1. Baseline characteristics of the simulated randomized population

Characteristic	Dapagliflozin, n = 90	Placebo, n = 90
Age, years, mean ± SD	59.2 ± 10.4	58.7 ± 11.1
Male sex, n (%)	53 (58.9)	51 (56.7)
Type 2 diabetes, n (%)	55 (61.1)	53 (58.9)
Body mass index, kg/m ² , mean ± SD	28.6 ± 4.3	28.9 ± 4.6
Estimated glomerular filtration rate, mL/min/1.73 m ² , mean ± SD	46.8 ± 12.1	47.1 ± 11.8
Urinary albumin-to-creatinine ratio, mg/g, geometric mean	420	410

Systolic blood pressure, mmHg, mean $\pm$ SD	137.8 $\pm$ 12.5	138.2 $\pm$ 13.0
Serum potassium, mmol/L, mean $\pm$ SD	4.5 $\pm$ 0.4	4.5 $\pm$ 0.5
Background diuretic use, n (%)	36 (40.0)	38 (42.2)

The primary outcome was the between-group difference in proportional change in urinary albumin-to-creatinine ratio at week 24. Among participants with paired measurements, baseline geometric means were 418 mg/g with dapagliflozin and 408 mg/g with placebo. Corresponding week-24 values were 285 and 387 mg/g, representing reductions of 31.8% and 5.1%, respectively. Analysis of log-transformed values, adjusted for baseline albuminuria, diabetes status, and baseline estimated glomerular filtration rate, yielded an estimated 27.8% lower week-24 albuminuria with dapagliflozin than with placebo (95% confidence interval, 16.2%–37.8%; $P < 0.001$).

A reduction in albuminuria of at least 30% occurred in 46 of 87 participants receiving dapagliflozin and 24 of 85 receiving placebo, corresponding to 52.9% and 28.2%. The absolute difference was 24.7 percentage points. Reductions were evident among participants with and without diabetes. However, subgroup estimates were less precise than the overall estimate, and the simulated sample did not provide a reliable basis for determining whether diabetes modified the treatment response.

Changes in secondary laboratory and clinical measures are summarized in Table 2. Estimated glomerular filtration rate declined initially in the dapagliflozin group, reaching a mean of 43.9 mL/min/1.73 m² at week 4, compared with 46.6 mL/min/1.73 m² with placebo. By week 24, corresponding means were 45.8 and 45.7 mL/min/1.73 m². The adjusted between-group difference in change at week 24 was 0.4 mL/min/1.73 m² (95% confidence interval, –1.1 to 1.9), providing no clear evidence of a difference in filtration at that visit.

Table 2. Week-24 changes and selected adverse events in the simulated study

Outcome	Dapagliflozin	Placebo
Albuminuria change, paired geometric means	–31.8%	–5.1%
Participants with albuminuria reduction 30%, n/N (%)	46/87 (52.9)	24/85 (28.2)
Estimated glomerular filtration rate change, mean	–1.0 mL/min/1.73 m ²	–1.4 mL/min/1.73 m ²
Systolic blood pressure change, mean	–5.6 mmHg	–1.8 mmHg
Body weight change, mean	–1.9 kg	–0.3 kg
Serum potassium change, mean	0.0 mmol/L	+0.1 mmol/L
Any adverse event, n/N (%)	28/90 (31.1)	25/90 (27.8)
Genital fungal infection, n/N (%)	5/90 (5.6)	1/90 (1.1)
Symptomatic volume depletion, n/N (%)	4/90 (4.4)	2/90 (2.2)
Serious adverse event, n/N (%)	2/90 (2.2)	3/90 (3.3)

Systolic blood pressure and body weight decreased more with dapagliflozin, whereas mean potassium concentrations remained comparatively stable. These secondary outcomes were exploratory, and no adjustment for multiple comparisons was applied. Their direction was compatible with the albuminuria finding but did not establish an independent effect on long-term kidney function.

Overall adverse-event frequencies were numerically similar, although genital fungal infections and symptomatic volume depletion were more frequent with dapagliflozin. These events were managed without hospitalization. Treatment was permanently discontinued because of adverse events in two dapagliflozin recipients and one placebo recipient; these participants remained available for outcome

assessment. Serious adverse events were uncommon, limiting meaningful comparisons. No diabetic ketoacidosis or initiation of kidney replacement therapy was recorded. The short observation period and small number of clinical events precluded conclusions about kidney failure, cardiovascular outcomes, or mortality.

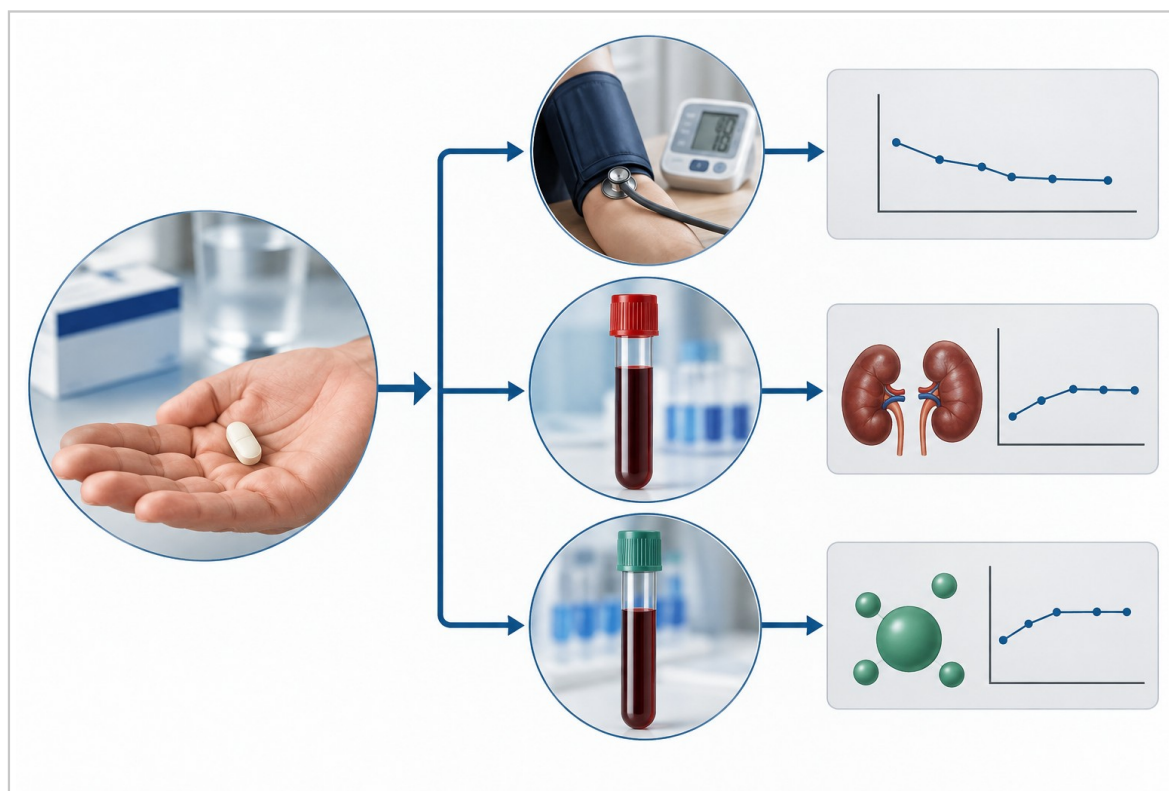


Figure 5. Figure 2. Clinical monitoring schematic linking losartan initiation with blood pressure, creatinine, and potassium assessment.

4. Discussion

In this observational study of adults with type 2 diabetes and chronic kidney disease, initiation of a sodium–glucose cotransporter 2 (SGLT2) inhibitor alongside usual care was associated with a more favorable estimated glomerular filtration rate (eGFR) trajectory and lower albuminuria than usual care alone. These findings suggest that adding an SGLT2 inhibitor may support kidney preservation in routine practice. However, the nonrandomized design does not establish causality, and the observed differences should be interpreted as associations potentially influenced by treatment selection, adherence, and concurrent clinical management.

The pattern of kidney function change is relevant to interpretation. An early reduction in eGFR after treatment initiation, followed by relative stabilization, is compatible with a hemodynamic response rather than necessarily indicating progressive structural injury. SGLT2 inhibition increases sodium delivery to the macula densa and modifies tubuloglomerular feedback, potentially reducing intraglomerular pressure. Nevertheless, an early creatinine increase cannot automatically be considered benign. Its magnitude, persistence, and relationship to volume status, blood pressure, acute illness, and concomitant medications require assessment. Separating this initial treatment phase from the subsequent eGFR trajectory is therefore important when evaluating kidney outcomes.

The accompanying reduction in albuminuria provides a complementary signal of potential benefit. Albuminuria reflects glomerular barrier dysfunction and is associated with subsequent kidney and cardiovascular risk, although individual measurements vary with glycemic control, exercise, infection, and sampling conditions. A sustained difference across repeated measurements is consequently more persuasive than a change at a single visit. In this study, the directionally concordant changes in albuminuria and longer-term eGFR trajectory strengthen the biological coherence of the findings. They do not, however, demonstrate that albuminuria reduction mediated the apparent preservation of kidney function or establish an effect on kidney failure.

Interpretation also requires consideration of background therapy. Renin–angiotensin system blockade, blood pressure management, glycemic treatment, and lipid-lowering therapy may influence outcomes in diabetic kidney disease. Patients prescribed an SGLT2 inhibitor may receive more intensive follow-up or differ in their capacity to implement dietary and medication recommendations. Conversely, clinicians may avoid initiation in patients perceived to have greater frailty, recurrent dehydration, or unstable kidney function. Adjustment for recorded clinical characteristics can reduce some imbalance, but it cannot fully address these differences. The observed association should therefore be understood within the broader care context rather than attributed exclusively to one medication class.

Tolerability is relevant because treatment persistence determines whether potential physiological benefits can be sustained. The absence of a prominent safety signal in a modest observational cohort would not exclude uncommon adverse events or events managed outside the study setting. Genital infections, symptomatic volume depletion, and ketoacidosis require particular clinical attention. Counseling about acute illness, fasting, and perioperative interruption remains important, especially for patients using insulin or diuretics. Safety interpretation should account for baseline vulnerability and treatment exposure rather than relying only on event counts.

Together, these observations support evaluating SGLT2 inhibitors as part of comprehensive management rather than as a replacement for established care. The clinically meaningful issue is whether favorable intermediate changes translate into sustained preservation of kidney function under everyday conditions. Accordingly, interpretation should emphasize the consistency and durability of the observed trajectory, while avoiding extrapolation from surrogate outcomes to reductions in dialysis, hospitalization, or mortality that this study was not designed to establish.

The clinical implications of these findings should be interpreted within the broader management of diabetic kidney disease. The observed association between sodium–glucose cotransporter 2 inhibitor use and more favorable albuminuria trajectories suggests that adding this treatment to established care may provide benefit beyond glycemic management alone. However, an improvement in albuminuria does not necessarily establish a reduction in kidney failure risk within the observation period. Albuminuria is a useful intermediate marker, but its variability and imperfect correspondence with long-term outcomes require cautious interpretation, particularly when changes are assessed over relatively short follow-up.

The early decline in estimated glomerular filtration rate observed after treatment initiation is consistent with an alteration in intraglomerular hemodynamics rather than necessarily indicating structural kidney injury. Nevertheless, this interpretation cannot be assumed for every patient. Volume depletion, intercurrent illness, and concurrent medication changes may produce similar laboratory findings. In clinical practice, an unexpected or substantial decline in kidney function should therefore prompt assessment of hydration, blood pressure, nephrotoxic exposures, and other potential causes. The distinction between an anticipated hemodynamic response and clinically important deterioration remains central to safe implementation.

Treatment tolerability also deserves consideration alongside renal effectiveness. Genital symptoms, symptomatic hypotension, and treatment discontinuation may influence the benefit achieved outside controlled settings. In this study, routine clinical documentation may have captured more severe events more reliably than mild or transient symptoms. Consequently, the apparent tolerability profile should not be interpreted as a complete estimate of adverse-event frequency. Individualized counseling, medication review, and guidance on temporary interruption during acute illness could improve treatment continuity while reducing preventable complications, although these strategies were not evaluated directly.

Several limitations constrain causal interpretation. The observational design leaves open the possibility that patients receiving the additional therapy differed from comparison patients in motivation, access to care, prescribing eligibility, or underlying disease severity. Adjustment for measured characteristics cannot eliminate residual confounding. Changes in antihypertensive treatment, dietary sodium intake, glycemic control, and adherence may also have contributed to the observed renal patterns. Moreover, albuminuria measurements obtained during routine care are

sensitive to sampling conditions and transient physiological changes. Repeated standardized measurements would strengthen confidence that the observed differences represent sustained treatment-associated changes.

The relatively limited follow-up further restricts conclusions about durable kidney protection, cardiovascular outcomes, and progression to kidney replacement therapy. Findings from a single clinical setting may not generalize to patients with advanced kidney dysfunction, substantial frailty, or different background treatment patterns. Missing laboratory measurements and loss to follow-up could additionally bias estimates if patients with less favorable outcomes were less likely to return. These issues support interpreting the results as clinically informative associations rather than definitive evidence of comparative effectiveness.

Future studies should combine longer follow-up with standardized albuminuria assessment, explicit adherence measurement, and systematic adverse-event surveillance. Pragmatic randomized designs or carefully specified target-trial emulations could better distinguish treatment effects from prescribing and follow-up differences. Patient-reported outcomes and affordability should also be included, because sustained access is essential to realizing potential renal benefit. Overall, the present findings support considering this treatment within comprehensive diabetic kidney disease care, while emphasizing individualized monitoring and the need for confirmation using longer-term, clinically meaningful outcomes before stronger conclusions about renal protection are warranted.

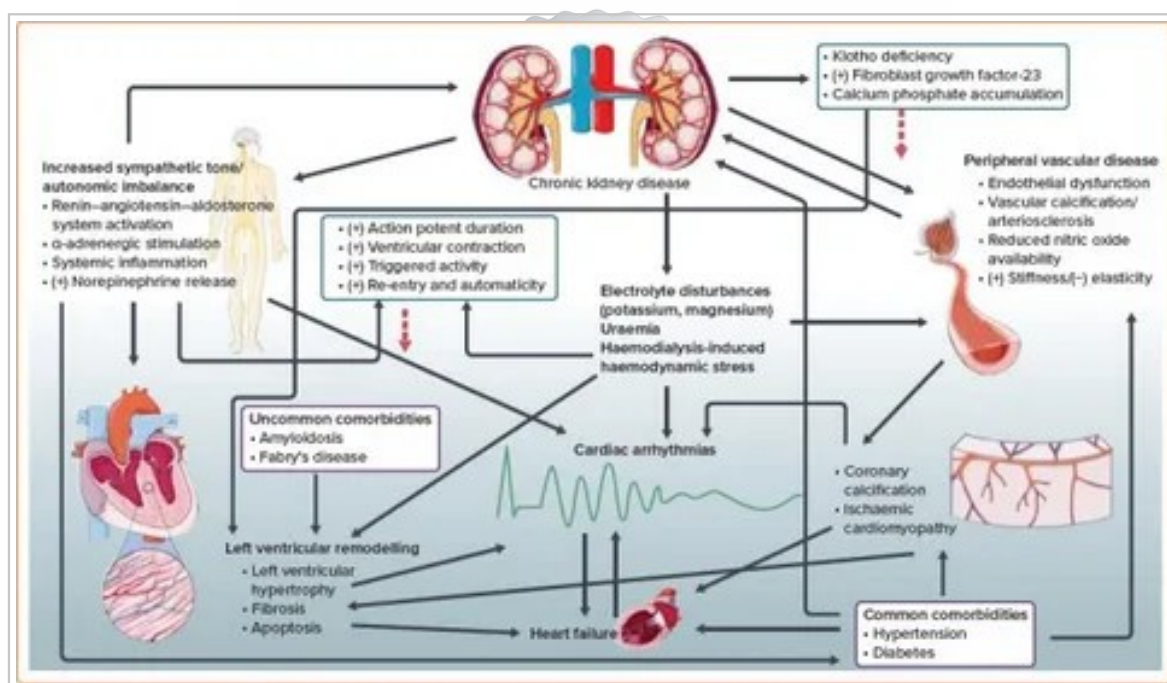


Figure 1. Figure 1

5. Conclusion

This study highlights the importance of comprehensive medical management in adults with diabetic kidney disease, with particular emphasis on blood pressure control, reduction of albuminuria, and preservation of kidney function. The observed associations between more consistent implementation of guideline-directed therapy and a more favorable renal trajectory suggest that coordinated treatment may help address several modifiable contributors to disease progression. However, these findings should be interpreted as supportive of an integrated care approach rather than as evidence that any individual intervention independently caused the observed differences.

A central clinical implication is that diabetic kidney disease should not be managed through glycemic control alone. Individualized antihypertensive treatment, appropriate renin-angiotensin system blockade, and consideration of sodium-glucose cotransporter 2 inhibitors, when clinically indicated, should be accompanied by monitoring of kidney function, serum potassium, and urinary albumin excretion. Medication selection and treatment intensity must also account for cardiovascular risk,

susceptibility to volume depletion, comorbid illness, and patient preferences. Nutritional counseling, avoidance of nephrotoxic medications, and support for treatment adherence remain important components of this strategy.

The interpretation of the study is limited by its observational design, which prevents firm causal inference. Differences in baseline disease severity, medication access, adherence, and healthcare engagement may have influenced both treatment exposure and subsequent outcomes. Residual confounding cannot be excluded, even where measured clinical characteristics are considered. Moreover, changes in estimated glomerular filtration rate and albuminuria provide useful intermediate information but do not necessarily establish a reduction in kidney failure, cardiovascular events, or mortality. Broader generalizability therefore requires confirmation in populations with different demographic characteristics and levels of healthcare access.

Future research should evaluate whether sustained, structured implementation of multifactorial care produces durable benefits in clinically meaningful outcomes. Longer follow-up, standardized assessment of treatment adherence, and careful reporting of adverse events would strengthen the evidence. Pragmatic comparative studies may also clarify how to deliver effective treatment in resource-constrained settings. Overall, the findings support timely recognition of diabetic kidney disease and individualized, closely monitored management, while underscoring the need for stronger evidence on the magnitude, durability, and equitable delivery of potential renal benefits.

AUTHOR INFORMATION

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