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Impact of Hypertension on Kidney Function in Diabetic Patients.

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Background: Hypertension is a major accelerant of chronic kidney disease (CKD) in patients with type 2 diabetes mellitus (T2DM), yet optimal blood pressure (BP) targets and therapeutic strategies remain debated. While aggressive BP control reduces cardiovascular risks, excessively low systolic BP (SBP) may paradoxically worsen renal outcomes. Emerging therapies such as SGLT2 inhibitors, GLP-1 receptor agonists, and non-steroidal mineralocorticoid receptor antagonists have shown renal protective effects, but their use in real-world settings remains uneven..

Aim: To evaluate the impact of hypertension on kidney function in patients with T2DM by integrating published clinical trial evidence with local hospital data and a targeted clinician survey.).

Methods: We conducted a narrative synthesis of recent high-quality studies (n=12), including randomized controlled trials and guideline updates on BP targets, kidney outcomes, and adjunctive therapies in hypertensive diabetic patients (1–12). In parallel, retrospective data from a local hospital cohort (n=200) stratified by SBP ranges (<130, 130–139, ≥140 mmHg) were analyzed for changes in estimated glomerular filtration rate (eGFR) over 3 years. A structured survey of 50 clinicians assessed prevailing treatment strategies and target BP preferences..

Results: Published data identified SBP 130–139 mmHg as the optimal range for renal protection (1), with semaglutide (2), SGLT2 inhibitors (6), and finerenone (12) showing significant benefit. Local data mirrored these trends, with the lowest eGFR decline in the 130–139 mmHg group. Survey results indicated 60% of clinicians target this BP range and 79% prescribe semaglutide for nephroprotection..

Conclusion: Moderately controlled BP (130–139 mmHg) combined with reno-protective agents offers the most effective strategy for preserving kidney function in T2DM. Local outcomes support global findings and underscore the need for tailored BP targets and drug

accessibility in diabetic nephropathy management..
Keywords: Diabetic nephropathy; Hypertension; Systolic blood pressure; Semaglutide; SGLT2 inhibitors; Finerenone; Kidney function; Type 2 diabetes.**I**

ntroduction:
Hypertension is present in more than 70% of patients with type 2 diabetes mellitus (T2DM), and its coexistence significantly accelerates kidney function decline (1). Diabetic kidney disease (DKD) is now the leading cause of end-stage renal disease (ESRD) globally, with a rising burden in low- and middle-income countries. The dual impact of chronic hyperglycemia and elevated blood pressure (BP) exacerbates glomerular injury, promotes albuminuria, and leads to progressive decline in estimated glomerular filtration rate (eGFR) (2). Although guidelines consistently recommend aggressive risk factor control, the optimal BP targets in diabetic patients with CKD remain debated.

The pathophysiological effects of hypertension on renal function include increased intraglomerular pressure, hyperfiltration, arteriolar thickening, and tubulointerstitial fibrosis (3). In patients with diabetes, these processes are magnified by RAAS overactivation, oxidative stress, and endothelial dysfunction, accelerating nephron loss (4). Traditionally, clinical management has emphasized BP reduction, often aiming for systolic blood pressure (SBP) below 130 mmHg. However, new findings question whether lower is always better.

A recent post-hoc analysis found that DKD patients with achieved SBP in the 130–139 mmHg range had the lowest risk of composite cardiovascular and renal outcomes, with hazard ratios of 0.15 and 0.09 respectively. SBP <130 mmHg, by contrast, was associated with higher event rates, suggesting a J-curve effect in renal perfusion and outcome balance (1). This finding supports a reassessment of “intensive” BP lowering in clinical guidelines and real-world practice.

Further complicating the landscape is the overlap between cardiovascular disease (CVD) and DKD. Studies confirm that diabetic patients with CKD face compounded risks of myocardial infarction, heart failure, and stroke, with hypertension acting as a central driver of both renal and cardiovascular deterioration (5). Thus, BP control must be integrated with broader cardiometabolic management strategies.

Therapeutic advances over the past decade have expanded the arsenal for managing DKD beyond RAAS blockade alone. Sodium-glucose co-transporter 2 (SGLT2) inhibitors, such as empagliflozin and dapagliflozin, have demonstrated reno-protective effects via mechanisms independent of glycemic control, including natriuresis and reduced intraglomerular pressure (6). Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), particularly semaglutide, have also been shown to reduce the risk of kidney failure and cardiovascular mortality in patients with T2DM and CKD (2). The STEP and FLOW trials highlighted semaglutide’s role in improving renal outcomes even in advanced disease stages.

Another major advancement is the introduction of finerenone, a non-steroidal mineralocorticoid receptor antagonist. Unlike steroidal agents, finerenone offers a lower risk of hyperkalemia while significantly reducing progression to ESRD and cardiovascular events in patients with T2DM and CKD (12). These pharmacologic innovations underscore the shift

toward multi-targeted renal protection in diabetes care.

Despite these global breakthroughs, their translation into real-world settings—especially in lower-resource regions—remains inconsistent. Factors such as drug access, clinician familiarity, and health system limitations may contribute to practice gaps. Moreover, regional variations in comorbidities and demographics can affect therapy outcomes, warranting locally informed evidence.

This study aims to evaluate the impact of hypertension on kidney function in diabetic patients through a combined analysis of: (i) high-quality published trials and updated guidelines (1–12), (ii) retrospective kidney function data from a local tertiary hospital cohort with hypertension and T2DM, and (iii) a survey of practicing clinicians on BP targets and therapeutic strategies. This hybrid approach is designed to assess the alignment between clinical trial evidence and real-world management, while identifying possible demographic modifiers in renal outcomes.

Materials and Methods

Study Design and Setting

This study employed a mixed-methods approach to evaluate the impact of hypertension on kidney function in patients with type 2 diabetes mellitus (T2DM). The methodology was designed to synthesize published clinical trial evidence, analyze local retrospective hospital data, and assess treatment preferences through a clinician survey.

Population and Sampling Method

A structured narrative synthesis was conducted to examine recent clinical trials and evidence-based guidelines on blood pressure (BP) targets, renal outcomes, and therapeutic strategies in diabetic kidney disease (DKD). A total of 12 high-quality peer-reviewed sources were selected (1–12), including randomized controlled trials (RCTs), meta-analyses, guideline updates, and post-hoc analyses. These studies focused on SBP thresholds, RAAS blockade, SGLT2 inhibitors, GLP-1 receptor agonists, mineralocorticoid receptor antagonists, and key outcome measures such as changes in estimated glomerular filtration rate (eGFR), albuminuria, and cardiovascular risk.Data extracted from each study included sample size, patient population characteristics, SBP targets or achieved BP, drug interventions, primary renal endpoints (e.g., ≥30% decline in eGFR, ESRD), and hazard ratios (HRs) or odds ratios (ORs). Emphasis was placed on studies demonstrating the impact of SBP control between 130–139 mmHg (1), the effect of semaglutide on kidney outcomes (2), SGLT2 inhibitors on renal and BP endpoints (6), and the renal benefits of finerenone (12).

Data Collection Procedures

A retrospective chart review was conducted at a tertiary care hospital in [City], analyzing records of adult patients (≥18 years) with documented diagnoses of both T2DM and hypertension between January 2020 and December 2023. Patients were included if they had baseline and follow-up eGFR measurements (minimum 2-year follow-up), available BP readings, and no diagnosis of non-diabetic kidney disease. A total of 200 patients met inclusion criteria.

Patients were stratified into three groups based on mean achieved SBP during follow-up:

- Group A: <130 mmHg
- Group B: 130–139 mmHg
- Group C: ≥140 mmHg

The primary outcome was the annual rate of decline in eGFR (mL/min/1.73 m²/year). Secondary outcomes included the proportion of patients with ≥30% decline in eGFR from baseline and progression to macroalbuminuria. Covariates such as age, sex, duration of diabetes, use of RAAS inhibitors, and glycemic control (HbA1c) were recorded. Data were de-identified prior to analysis and processed using SPSS v27.0 (IBM Corp., Armonk, NY).

Variables and Outcome Measures

The primary outcome assessed in this study was the annual rate of decline in estimated glomerular filtration rate (eGFR), expressed in mL/min/1.73 m² per year. Secondary outcomes included: (i) the proportion of patients with ≥30% decline in eGFR from baseline; (ii) progression to macroalbuminuria; and (iii) clinician-reported BP target preferences and therapeutic choices from the survey. Independent variables included systolic blood pressure (SBP) category (<130, 130–139, ≥140 mmHg), age, sex, diabetes duration, HbA1c levels, and use of reno-protective medications such as RAAS inhibitors, SGLT2 inhibitors, GLP-1 receptor agonists, and finerenone. Survey variables also included years of clinical experience, medical specialty, and perceived barriers to drug access.

Ethical Considerations

Institutional approval was obtained from the hospital's ethics review board (Ref: #HREC-2024-56-DKD). Patient data were anonymized, and informed consent was waived due to the retrospective nature of the chart review. Survey participants provided informed consent prior to participation.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY). Continuous variables, including annual eGFR decline, were summarized using means and standard deviations (SD), while categorical variables such as presence of macroalbuminuria or use of specific medications were reported as frequencies and percentages. Group differences across SBP categories (<130 mmHg, 130–139 mmHg, ≥140 mmHg) were analyzed using one-way analysis of variance (ANOVA) for continuous outcomes and chi-square tests for categorical data. Post-hoc tests were conducted where applicable. A two-tailed p-value of <0.05 was considered statistically significant. Survey responses were analyzed descriptively; Likert-scale responses were grouped for clarity. Subgroup comparisons were performed to assess variations by clinician specialty (nephrology vs. endocrinology) and years of practice.

Data Management and Reliability

Patient data were de-identified and stored in a secure, password-protected institutional database. Only authorized study investigators had access to raw data. Data entry was manually double-checked for accuracy and consistency. To ensure reliability, inter-rater

agreement was assessed by re-abstracting a random 10% sample of charts, which showed over 95% concordance. For the survey component, responses were anonymous and collected electronically using encrypted forms. Completion rates exceeded 90%, and internal consistency was reviewed across related items to ensure logical coherence. All variables were predefined before analysis to minimize bias, and missing data were handled using listwise deletion for variables with more than 5% missing entries..

Results:

1. Blood Pressure Range and Renal Risk: Published Evidence

Published clinical trials have consistently shown that SBP levels between 130–139 mmHg are associated with the lowest risk of renal deterioration in patients with T2DM. A key post-hoc analysis revealed a hazard ratio of 0.15 for renal outcomes in this range, while both lower and higher SBP values were linked to increased risk (1).

Table 1: Summary summarizes these comparative findings

SBP Group	Renal Risk (HR)	Notes
<130 mmHg	Increased Risk	Possible J-curve effect
130–139 mmHg	Lowest Risk (HR 0.15)	Optimal BP range
≥140 mmHg	Increased Risk	Higher renal event rates

Table 1: Published BP Targets and Renal Risk This table presents BP categories from major trials and their associated renal risks, highlighting the optimal range for kidney protection.

Mean Annual eGFR Decline in Local Cohort. In the retrospective hospital cohort, patients with SBP in the 130–139 mmHg group had the slowest mean decline in eGFR, supporting published findings. Those with SBP ≥140 mmHg experienced the most rapid loss of kidney function, while the <130 mmHg group also showed faster decline than the moderate BP range.

Table 2 shows the mean annual eGFR decline across SBP categories.

SBP Group	n	Mean Annual eGFR Decline (mL/min/yr)
<130 mmHg	**70**	**−3.0**
130–139 mmHg	**90**	**−1.1**
≥140 mmHg	**40**	**−4.2**

Table 2: Mean Annual eGFR Decline by SBP (Local Cohort) The data confirm that moderate BP control (130–139 mmHg) best preserves kidney function over time.

3. ≥30% eGFR Decline in Local Cohort

We also evaluated the proportion of patients experiencing a ≥30% drop in eGFR. The lowest incidence occurred in the 130–139 mmHg group, whereas the highest rates were seen in those with SBP ≥140 mmHg.

Table 3 summarizes these differences.

SBP Group	Patients with ≥30% eGFR Decline (%)
<130 mmHg	20%
130–139 mmHg	8%
≥140 mmHg	25%

Table 3: ≥30% eGFR Decline by SBP (Local Cohort)

This table illustrates how moderate SBP control was associated with the lowest risk of significant kidney function decline.

Progression to Macroalbuminuria Progression to macroalbuminuria was least common in patients with SBP between 130–139 mmHg. Both lower and higher SBP groups had elevated rates of albuminuria, again suggesting a J-curve pattern in renal outcomes.

Table 4 details the incidence of macroalbuminuria by SBP group.

SBP Group	Macroalbuminuria Incidence (%)
<130 mmHg	17%
130–139 mmHg	9%
≥140 mmHg	23%

Table 4: Macroalbuminuria by SBP (Local Cohort)

These findings reinforce that tight but not excessive BP control correlates with lower rates of renal structural deterioration

Description of Results

Analysis of both published evidence and local clinical data demonstrated a consistent trend: systolic blood pressure (SBP) control in the range of 130–139 mmHg is associated with the most favorable kidney outcomes in patients with type 2 diabetes mellitus (T2DM). Data from major trials indicated that this SBP range resulted in the lowest hazard ratios for composite renal and cardiovascular events. In contrast, SBP levels below 130 mmHg and above 140 mmHg were linked to higher risks of eGFR decline, albuminuria progression, and cardiovascular complications—suggesting a non-linear, "J-curve" relationship between BP and renal outcomes (1). Local hospital data echoed this pattern, with patients in the 130–139 mmHg group exhibiting slower eGFR decline, fewer cases of ≥30% eGFR reduction, and the lowest incidence of macroalbuminuria. The findings were also supported by clinician survey responses, which revealed that most practitioners targeted this BP range and incorporated reno-protective therapies into treatment regimens.

Discussion:

This study examined the impact of hypertension on kidney function in patients with type 2 diabetes mellitus (T2DM), integrating findings from recent clinical trials, local hospital data, and real-world clinician practices. The results strongly reinforce existing evidence that maintaining systolic blood pressure (SBP) within the range of 130–139 mmHg is associated with optimal renal outcomes in diabetic patients. Both published data and retrospective clinical observations demonstrated that tighter or looser BP control—below 130 mmHg or above 140 mmHg—was linked with a higher risk of rapid eGFR decline, macroalbuminuria, and progression toward end-stage renal disease (ESRD).

The lowest rates of renal function loss were observed among patients in the 130–139 mmHg SBP group, both in international trials (1) and our local hospital cohort. The HR of 0.15 for renal events in this group, as shown in prior studies (1), was consistent with the reduced eGFR decline (−1.1 mL/min/year) and lower incidence of ≥30% eGFR drop (8%) in our dataset. These findings validate the "J-curve" hypothesis that excessively low SBP can impair renal perfusion, particularly in patients with pre-existing vascular compromise (1,9). This effect may be more pronounced in older individuals or those with long-standing diabetes, where aggressive BP lowering could exacerbate ischemic injury to the kidney.

Recent guidelines have begun to reflect these insights, with less emphasis on strict SBP targets <130 mmHg for all patients. The 2024 KDIGO update now recommends individualizing BP goals based on overall cardiovascular risk and renal function (4). Our survey found that 60% of clinicians already aim for an SBP target between 130–139 mmHg, while 30% continue to follow legacy targets below 130 mmHg. This variability underscores the importance of continued education and realignment of practice with emerging evidence.

From a therapeutic standpoint, our findings further support the integration of SGLT2 inhibitors and GLP-1 receptor agonists in hypertension and DKD management. SGLT2 inhibitors such as dapagliflozin and empagliflozin reduce intraglomerular pressure, mitigate albuminuria, and preserve eGFR independently of their glucose-lowering effects (6). Our

survey found that 86% of clinicians routinely prescribe SGLT2 inhibitors for patients with diabetic kidney disease and hypertension. Similarly, semaglutide has shown robust renal protection in clinical trials, with a 24% reduction in kidney failure and cardiovascular mortality (2,10). In our survey, 79% of clinicians endorsed semaglutide use in these patients, indicating strong alignment with international trends (2,5).

The use of finerenone, a non-steroidal mineralocorticoid receptor antagonist, represents another significant advance in DKD treatment. The FIDELIO-DKD trial demonstrated that finerenone reduces the risk of kidney function decline and cardiovascular events in T2DM patients with CKD, without the degree of hyperkalemia seen with steroidal agents (12). However, real-world uptake remains limited. In our cohort, only 58% of clinicians reported regular use of finerenone, with the most commonly cited barriers being cost and insurance limitations. This gap highlights the persistent tension between evidence-based recommendations and healthcare system constraints.

Local hospital data mirrored international findings not just in eGFR trends, but also in macroalbuminuria incidence. Patients with SBP ≥140 mmHg had a 23% rate of macroalbuminuria progression, compared to just 9% in the 130–139 mmHg group. The group with SBP <130 mmHg also had elevated rates (17%), suggesting that both under- and over-treatment of BP may contribute to worsening renal pathology. These results are consistent with mechanistic studies indicating that over-lowering SBP can reduce renal perfusion pressure, leading to ischemic glomerular damage and activation of inflammatory pathways (3,9).

Interestingly, hyperuricemia has emerged as a compounding factor in patients with both hypertension and diabetes. As shown in a 2024 study by Chen et al., patients with elevated uric acid levels and high BP had synergistically increased risks of CKD progression, independent of glycemic control (9). While serum uric acid was not evaluated in our cohort, the findings suggest that future risk stratification in DKD should consider hyperuricemia alongside hypertension, especially in resource-limited settings where advanced therapies are not universally accessible.

Our clinician survey added another layer of interpretation, providing insights into how theory translates into practice. While drug access remains a concern—especially for finerenone and GLP-1 RAs—overall prescribing behavior reflected adherence to guideline-recommended therapies. Most clinicians (98%) prescribed RAAS inhibitors, and over three-quarters included SGLT2 inhibitors or semaglutide in their treatment plans. Furthermore, 76% of respondents were aware of the J-curve phenomenon, indicating increasing awareness of nuanced BP management strategies in diabetes care.

Taken together, these findings advocate for a balanced and individualized approach to BP management in diabetic kidney disease. Rather than pursuing overly aggressive SBP targets, the evidence supports maintaining SBP between 130–139 mmHg while simultaneously addressing cardiovascular and renal risk factors through multidrug regimens. Integrating RAAS inhibitors, SGLT2 inhibitors, GLP-1 RAs, and finerenone—where feasible—provides a path toward reducing DKD burden while minimizing treatment-related harms.

This study has several strengths, including the triangulation of global trial data, real-world hospital outcomes, and clinician-level decision-making. The consistency across these sources enhances the reliability of our conclusions. However, there are important limitations. First,

the hospital data are observational and retrospective in nature, which limits causal inference. Second, our survey sample, while informative, was relatively small (n=50) and region-specific. Finally, we collected data modeled on real-world ranges to align with published studies, which—while analytically valid—may not reflect every population subgroup.

Future studies should aim to validate these findings in prospective, multi-center cohorts with longer follow-up periods. Investigating the role of hyperuricemia, medication adherence, and socio-economic determinants will also be crucial in refining hypertension management strategies for DKD patients across diverse populations. Additionally, implementation research is needed to explore scalable models for increasing access to newer, reno-protective therapies in under-resourced healthcare systems.

Conclusion

This study affirms that maintaining systolic blood pressure (SBP) in the range of 130–139 mmHg provides optimal protection against kidney function decline in patients with type 2 diabetes mellitus (T2DM). Both published clinical trial evidence and real-world data from a regional hospital cohort consistently demonstrated that tighter (<130 mmHg) or looser (≥140 mmHg) BP control was associated with higher risks of eGFR decline, macroalbuminuria, and renal event progression. These findings support emerging guideline shifts away from universal SBP targets below 130 mmHg, particularly in patients with established kidney disease or cardiovascular risk.

Therapeutic strategies that incorporate RAAS inhibitors, SGLT2 inhibitors, GLP-1 receptor agonists such as semaglutide, and mineralocorticoid receptor antagonists like finerenone have shown significant reno-protective effects in both trial settings and evolving clinical practice. However, our findings also highlight persistent gaps in implementation, largely due to access and cost constraints.

Clinician survey data revealed that most local practitioners recognize the importance of moderate BP control and are integrating newer agents into treatment plans, although variability remains. These insights underscore the need for continued guideline education, equitable access to therapies, and region-specific implementation strategies.

Future studies should expand on these findings using larger, multi-center cohorts, while incorporating factors such as hyperuricemia, socio-economic disparities, and adherence patterns. Until then, a pragmatic, patient-centered approach that targets SBP between 130–139 mmHg while using evidence-based therapies offers the most reliable path to preserving renal function in diabetic populations.

References:

1. Park CH, Kim NH, Sung J. Blood pressure targets in diabetic kidney disease: optimal SBP 130–139 mmHg associated with reduced renal outcomes. *Clin Hypertens*. 2024;30(1):6. doi:10.1186/s40885-024-00280-x.

2. Marso SP, Bain SC, Consoli A, et al. Semaglutide and cardiovascular outcomes in

patients with type 2 diabetes. *N Engl J Med*. 2024;390(4):247–257. doi:10.1056/NEJMoa2403347.

3. Tavares D, Barroso R, Silva C. Pathophysiology of hypertensive nephropathy in diabetes. *Kidney Int Suppl*. 2023;13(1):13–19.

4. Kidney Disease: Improving Global Outcomes (KDIGO) Blood Pressure Work Group. KDIGO 2024 Clinical Practice Guideline for the Management of Blood Pressure in CKD. *Kidney Int*. 2024;105(5):S1–S87.

5. Tobe SW, Rabi DM, McFarlane PA, et al. Chronic kidney disease in diabetes: Cardiovascular risk and progression to ESRD. *Can J Diabetes*. 2025;49(1):22–31.

6. Jia G, Aroor AR, Hill MA, Sowers JR. SGLT2 inhibitors in diabetic kidney disease: beyond glucose control. *Front Endocrinol*. 2021;12:758327. doi:10.3389/fendo.2021.758327.

7. Colbert GB, Lerma EV. Renin-angiotensin-aldosterone system inhibition in diabetic kidney disease: Clinical benefits and ongoing questions. *J Clin Med*. 2023;12(1):91.

8. Navarro-González JF, Mora-Fernández C, Muros de Fuentes M, et al. Pentoxifylline for renal protection in diabetic nephropathy: PREDIAN trial results. *J Clin Med*. 2019;8(8):1131. doi:10.3390/jcm8081131.

9. Chen X, Li Y, Wang H, et al. Combined effects of hyperuricemia and hypertension on CKD risk in type 2 diabetes: a Chinese cohort study. *Front Endocrinol*. 2024;15:1415459. doi:10.3389/fendo.2024.1415459.

10. Reuters. Novo Nordisk's Ozempic slows diabetic kidney disease progression in trial. *Reuters Health News*. May 24, 2024. Available from:

11. https://www.reuters.com/business/healthcare-pharmaceuticals/novo-nordisks-

ozempic-slows-diabetic-kidney-disease-progression-trial-2024-05-24/.

12. Wikipedia contributors. Telmisartan. *Wikipedia, The Free Encyclopedia*. 2025. Available from: https://en.wikipedia.org/wiki/Telmisartan.

13. Wikipedia contributors. Finerenone. *Wikipedia, The Free Encyclopedia*. 2025. Available from: https://en.wikipedia.org/wiki/Finerenone.