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SGLT2 Inhibitors for Renoprotection in Hypertensive Chronic Kidney Disease: Beyond Glycemic Control: A Narrative Review

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Abstract

Background: Chronic kidney disease (CKD) is a major global health burden, with hypertension being a leading driver of disease progression. Despite established renoprotective therapies, substantial residual renal risk persists. Sodium–glucose cotransporter 2 (SGLT2) inhibitors have emerged as important kidney-protective agents beyond glucose lowering. This review summarizes the mechanisms and clinical evidence supporting the renoprotective effects of SGLT2 inhibitors in hypertensive patients with CKD.

Methods: A narrative review was conducted using literature from PubMed, Google Scholar, Scopus, and ScienceDirect published between 2015 and 2025. Studies evaluating the mechanisms, renoprotective effects, and clinical outcomes of SGLT2 inhibitors in patients with CKD and hypertension were included.

Results: SGLT2 inhibitors confer substantial renoprotective benefits through mechanisms independent of glycemic control, including restoration of tubuloglomerular feedback, reduction of intraglomerular pressure, improvement of sodium homeostasis, attenuation of neurohormonal activation, and suppression of inflammatory, oxidative, and fibrotic pathways. Major trials such as CREDENCE, DAPA-CKD, and EMPA-KIDNEY consistently demonstrated reduced albuminuria, slower eGFR decline, lower risk of kidney failure, and improved cardiovascular outcomes across diverse CKD populations, regardless of diabetic status. These agents are increasingly incorporated into contemporary CKD management and may provide additive benefits when combined with other renoprotective therapies.

Conclusion: SGLT2 inhibitors provide significant renoprotection beyond glycemic control by slowing CKD progression, reducing albuminuria, preserving kidney function, and lowering the risk of kidney failure, supporting their role as foundational therapy in hypertensive CKD.

Keywords: Chronic kidney disease; Hypertension; SGLT2 inhibitors; Renoprotection; Cardiorenal protection

1. Introduction

Chronic kidney disease (CKD) has emerged as one of the fastest-growing non-communicable diseases worldwide and represents a major challenge for contemporary healthcare systems (1). Recent estimates indicate that CKD affects more than 800 million individuals globally, corresponding to approximately 10% of the world's population (2). According to the Global Burden of Disease (GBD) 2023 analysis, CKD ranked as the ninth leading cause of death worldwide in 2023, accounting for approximately 1.48 million deaths. Hypertension is among the most important drivers of CKD development and progression. Elevated systolic blood pressure has consistently been identified as one of the leading modifiable risk factors contributing to CKD-related morbidity and mortality worldwide (3). Hypertension is highly

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prevalent among patients with CKD, affecting approximately 80–85% of this population, while hypertension-related CKD accounts for more than 24 million cases globally (4).

The relationship between hypertension and CKD is complex and bidirectional. Persistent elevation of systemic blood pressure promotes intraglomerular hypertension, endothelial dysfunction, vascular remodeling, and progressive nephron loss, ultimately leading to glomerulosclerosis and tubulointerstitial fibrosis (5). Conversely, declining kidney function further exacerbates hypertension through sodium retention, activation of the renin–angiotensin–aldosterone system (RAAS), sympathetic nervous system overactivity, and impaired vascular regulation. This self-perpetuating cycle contributes to progressive deterioration of renal function and markedly increases the risk of cardiovascular events, heart failure, and end-stage kidney disease (ESKD). Among the various etiologies of CKD, hypertension remains one of the most prevalent and clinically significant contributors to progressive kidney dysfunction. Hypertensive kidney injury is characterized by sustained glomerular hypertension, vascular remodeling, endothelial dysfunction, and chronic neurohormonal activation, all of which accelerate nephron loss and increase the risk of kidney failure. Consequently, hypertensive patients with CKD represent a particularly high-risk population in whom preservation of renal function remains a major therapeutic priority (1).

For decades, renoprotective strategies in CKD have centered primarily on blood pressure control and pharmacologic blockade of the RAAS. The introduction of angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) significantly improved renal outcomes by reducing proteinuria and slowing CKD progression (6). Nevertheless, substantial residual risk persists despite optimal RAAS inhibition and guideline-directed blood pressure management. Many patients continue to experience progressive declines in estimated glomerular filtration rate (eGFR), increasing albuminuria, and eventual progression toward kidney failure. These observations have highlighted the need for additional therapeutic approaches capable of targeting the complex pathophysiological mechanisms underlying CKD progression beyond conventional hemodynamic control (7).

In recent years, sodium–glucose cotransporter 2 (SGLT2) inhibitors have emerged as one of the most important therapeutic advances in nephrology. Initially developed as glucose-lowering agents for the management of type 2 diabetes mellitus, SGLT2 inhibitors have demonstrated unexpectedly robust cardiovascular and renal benefits that extend far beyond their effects on glycemic control (8). Landmark trials, including CREDENCE, DAPA-CKD, and EMPA-KIDNEY, have consistently demonstrated significant reductions in CKD progression, decline in kidney function, kidney failure, and cardiovascular events across diverse CKD populations, including individuals without diabetes. These findings have fundamentally transformed the role of SGLT2 inhibitors from antidiabetic agents to cornerstone therapies in contemporary cardiorenal medicine (9).

Emerging evidence suggests that SGLT2 inhibitors provide renoprotective effects beyond glucose lowering through multiple hemodynamic and non-hemodynamic mechanisms. These mechanisms may be particularly relevant in hypertensive CKD, where glomerular hyperfiltration, maladaptive neurohormonal activation, endothelial dysfunction, and chronic inflammation play central roles in disease progression (10).

Despite growing evidence supporting the renal benefits of SGLT2 inhibitors in CKD, their role in hypertensive CKD and the mechanisms underlying kidney protection beyond glycemic control remain incompletely understood. As the clinical use of SGLT2 inhibitors continues to expand, a clearer understanding of these renoprotective effects is increasingly important. Therefore, this narrative review summarizes the current evidence on the renoprotective effects of SGLT2 inhibitors in hypertensive patients with CKD, focusing on the underlying mechanisms and contemporary clinical evidence.

2. Methods

This narrative review was conducted through a comprehensive literature search of electronic databases, including PubMed, Google Scholar, Scopus, and ScienceDirect. Relevant articles evaluating the renoprotective effects of sodium–glucose cotransporter 2 (SGLT2) inhibitors in patients with chronic kidney disease (CKD), particularly those with hypertension, were identified using combinations of the following keywords: “chronic kidney disease,” “CKD,” “hypertension,” “hypertensive CKD,” “SGLT2 inhibitors,” “sodium glucose cotransporter 2 inhibitors,” “renoprotection,” “renal outcomes,” “albuminuria,” “kidney function decline,” “cardiorenal protection,” and “glomerular hemodynamics.”

Articles were selected based on predefined inclusion and exclusion criteria. The inclusion criteria comprised studies published between 2015 and 2025, articles written in English, articles addressing the renal effects of SGLT2 inhibitors in CKD and hypertension. Studies available only as abstracts, duplicate publications, and studies not directly relevant to

the topic were excluded. Priority was given to landmark clinical trials and contemporary evidence evaluating the mechanisms and clinical outcomes associated with SGLT2 inhibitor therapy.

2.1. Pathophysiological Basis of Hypertension-Driven CKD Progression

Hypertension is both a major cause and a common consequence of chronic kidney disease (CKD), creating a bidirectional relationship that accelerates renal function decline and increases cardiovascular risk. Persistent elevation of systemic blood pressure exposes the renal microvasculature to chronic hemodynamic stress, resulting in structural and functional alterations that progressively impair kidney function. Over time, these maladaptive changes promote nephron loss, glomerulosclerosis, and tubulointerstitial fibrosis, ultimately leading to end-stage kidney disease (ESKD) (11).

One of the earliest and most important mechanisms underlying hypertension-driven CKD progression is glomerular hypertension. Under physiological conditions, autoregulatory mechanisms maintain relatively stable intraglomerular pressure despite fluctuations in systemic blood pressure. However, chronic hypertension gradually disrupts these protective mechanisms, leading to increased transmission of systemic pressure to the glomerular capillary network. The resulting elevation in intraglomerular pressure induces mechanical stress on podocytes, mesangial cells, and glomerular endothelial cells, triggering structural injury and progressive glomerulosclerosis. Persistent glomerular hyperfiltration further exacerbates nephron damage and contributes to the progressive decline in estimated glomerular filtration rate (eGFR) (12).

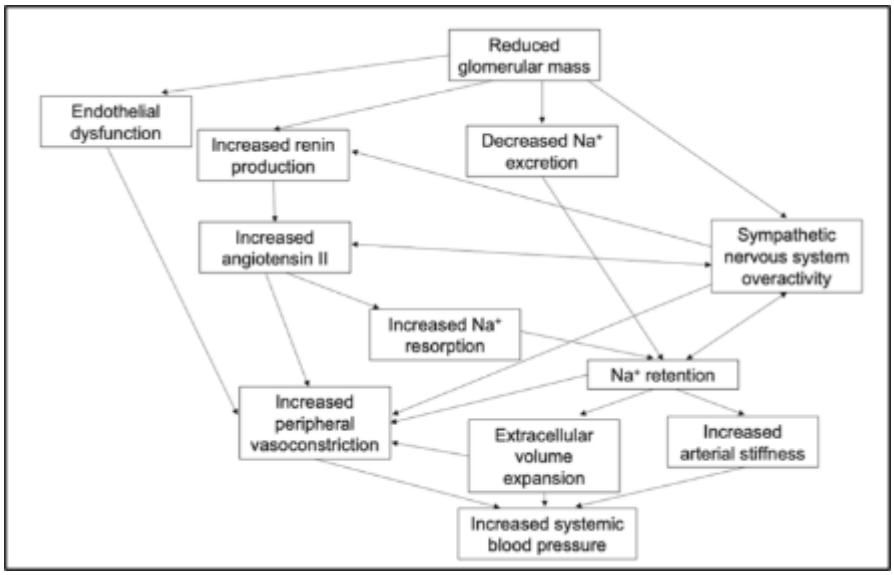

Figure 1 Pathophysiological Mechanisms of Hypertension-driven CKD Progression (1)

The renin–angiotensin–aldosterone system (RAAS) plays a central role in this process. Chronic activation of RAAS promotes efferent arteriolar vasoconstriction, thereby increasing intraglomerular pressure and worsening glomerular injury. Beyond its hemodynamic effects, angiotensin II stimulates pro-inflammatory and pro-fibrotic pathways through the upregulation of transforming growth factor-β (TGF-β), connective tissue growth factor, and extracellular matrix production. Aldosterone further contributes to renal injury by promoting inflammation, oxidative stress, endothelial dysfunction, and interstitial fibrosis. Consequently, sustained RAAS activation is widely recognized as a key driver of CKD progression in hypertensive patients (13).

In addition to neurohormonal activation, endothelial dysfunction is increasingly recognized as a hallmark of hypertension-related kidney injury. Chronic exposure to elevated blood pressure impairs nitric oxide bioavailability and promotes vascular stiffness, resulting in reduced vasodilatory capacity and impaired renal perfusion. Endothelial injury also facilitates leukocyte recruitment, inflammatory signaling, and microvascular rarefaction, all of which contribute to progressive renal damage. The resulting impairment in renal oxygen delivery may further aggravate tissue hypoxia and accelerate nephron loss (14).

Oxidative stress and chronic low-grade inflammation represent additional mechanisms linking hypertension to CKD progression. Increased production of reactive oxygen species (ROS) within the kidney contributes to cellular injury,

mitochondrial dysfunction, and activation of inflammatory pathways. Pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and monocyte chemoattractant protein-1 (MCP-1), promote immune cell infiltration and amplify tissue damage. Persistent inflammatory activity ultimately stimulates fibroblast activation and extracellular matrix deposition, leading to irreversible tubulointerstitial fibrosis, which is considered the final common pathway of CKD progression regardless of the underlying etiology. Emerging evidence also highlights the contribution of sympathetic nervous system overactivity in hypertension-driven kidney disease. Increased sympathetic activity promotes sodium retention, vasoconstriction, and further RAAS activation, thereby perpetuating hypertension and renal injury. This creates a self-reinforcing cycle in which progressive kidney dysfunction contributes to worsening hypertension, while uncontrolled hypertension accelerates structural kidney damage (15).

Hypertension-driven CKD progression results from a complex interplay of glomerular hypertension, neurohormonal activation, endothelial dysfunction, oxidative stress, inflammation, and fibrosis. These interconnected mechanisms not only contribute to progressive renal impairment but also provide important therapeutic targets for renoprotective interventions. Understanding these pathophysiological processes is essential for appreciating the mechanisms through which sodium–glucose cotransporter 2 (SGLT2) inhibitors exert kidney-protective effects beyond glycemic control (12).

2.2. Mechanisms of Renoprotection by SGLT2 Inhibitors Beyond Glycemic Control

The remarkable kidney-protective effects of sodium–glucose cotransporter 2 (SGLT2) inhibitors cannot be explained solely by their glucose-lowering properties. Increasing evidence from experimental and clinical studies indicates that these agents exert multiple complementary renoprotective actions that extend beyond glycemic control. These mechanisms target several key pathways involved in hypertension-driven CKD progression, including glomerular hyperfiltration, neurohormonal activation, inflammation, oxidative stress, and fibrosis. One of the most important mechanisms underlying SGLT2 inhibitor–mediated renoprotection is the restoration of tubuloglomerular feedback (TGF). Under normal physiological conditions, sodium delivery to the macula densa regulates afferent arteriolar tone and maintains stable intraglomerular pressure. In CKD and hypertension, impaired sodium handling and maladaptive renal hemodynamics contribute to glomerular hyperfiltration and elevated intraglomerular pressure, both of which accelerate nephron injury (16).

By inhibiting sodium and glucose reabsorption in the proximal tubule, SGLT2 inhibitors increase sodium delivery to the macula densa, triggering afferent arteriolar vasoconstriction and restoring tubuloglomerular feedback. This process reduces intraglomerular pressure and mitigates hyperfiltration-induced damage to the glomerular filtration barrier. The resulting reduction in mechanical stress on podocytes and glomerular endothelial cells is believed to be a major contributor to the long-term preservation of kidney function observed in large clinical trials. Beyond their direct renal hemodynamic effects, SGLT2 inhibitors exert modest but clinically meaningful reductions in blood pressure. Through natriuresis and osmotic diuresis, these agents promote sodium excretion, reduce extracellular fluid volume, and lower systemic blood pressure without substantial activation of compensatory sympathetic responses. Meta-analyses have demonstrated reductions in systolic blood pressure of approximately 3–5 mmHg among patients receiving SGLT2 inhibitors (17).

In hypertensive CKD, where sodium retention and volume expansion are major contributors to disease progression, improved sodium homeostasis may provide additional renal protection. Reduction in blood pressure decreases transmission of systemic pressure to the glomerular microcirculation, thereby limiting glomerular injury and slowing CKD progression. Furthermore, SGLT2 inhibitors have been shown to reduce arterial stiffness and improve vascular function, effects that may further contribute to cardiorenal protection (18).

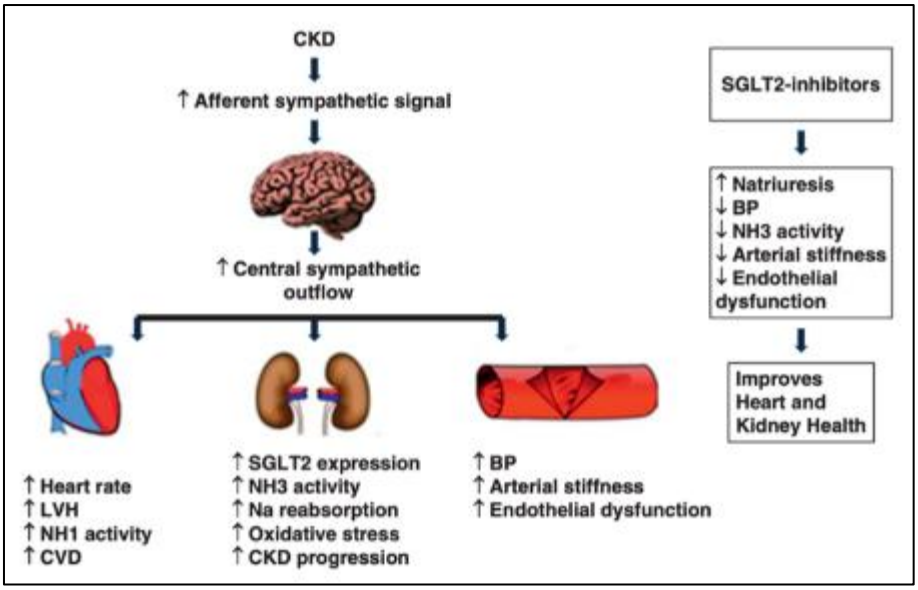

Figure 2 Effects of SGLT2 inhibitors on sympathetic activity, vascular function, and cardiorenal protection in CKD (18)

Chronic inflammation and fibrosis are central mechanisms driving progressive kidney injury regardless of the underlying cause of CKD. Experimental studies have demonstrated that SGLT2 inhibitors attenuate renal inflammation through the suppression of pro-inflammatory mediators, including interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and monocyte chemoattractant protein-1 (MCP-1). In addition, these agents appear to reduce inflammatory cell infiltration within the renal interstitium and attenuate activation of nuclear factor-kappa B (NF-κB) signaling pathways. SGLT2 inhibitors may also limit renal fibrosis by modulating transforming growth factor-β (TGF-β) signaling and reducing extracellular matrix deposition. Experimental models have demonstrated decreased expression of profibrotic markers and attenuation of tubulointerstitial fibrosis following SGLT2 inhibition. Given that fibrosis represents the final common pathway of CKD progression, these antifibrotic effects may play an important role in preserving long-term renal function (19).

Oxidative stress is increasingly recognized as a major contributor to kidney injury in both hypertension and CKD. Excessive generation of reactive oxygen species (ROS) promotes endothelial dysfunction, mitochondrial injury, inflammation, and fibrosis. SGLT2 inhibitors have been shown to reduce oxidative stress through multiple mechanisms, including improved mitochondrial efficiency, reduced oxygen consumption, and attenuation of ROS production within renal tissues. Furthermore, these agents appear to improve renal energy metabolism by promoting a shift toward more efficient substrate utilization. Enhanced mitochondrial function and improved renal oxygenation may help protect vulnerable tubular cells from hypoxic injury, a process that is particularly relevant in CKD, where chronic hypoxia is increasingly recognized as a key driver of disease progression (20).

2.3. Clinical Evidence Supporting Renoprotection in Hypertensive CKD

The renoprotective effects of sodium–glucose cotransporter 2 (SGLT2) inhibitors have been consistently demonstrated across multiple large-scale randomized controlled trials involving patients with chronic kidney disease (CKD). Although most landmark studies were not specifically designed to evaluate hypertension-driven CKD as a distinct clinical entity, hypertension was highly prevalent among enrolled participants, reflecting real-world CKD populations. Consequently, the findings of these trials provide important insights into the potential role of SGLT2 inhibitors in mitigating renal injury associated with chronic hypertension (19).

The first major evidence supporting the renoprotective effects of SGLT2 inhibitors emerged from the CREDENCE trial, which evaluated canagliflozin in patients with type 2 diabetes mellitus and albuminuric CKD receiving standard renin–angiotensin–aldosterone system (RAAS) blockade. The study demonstrated a significant reduction in the composite outcome of end-stage kidney disease (ESKD), doubling of serum creatinine, or renal death compared with placebo. In addition to slowing the decline in estimated glomerular filtration rate (eGFR), canagliflozin significantly reduced albuminuria, suggesting attenuation of ongoing glomerular injury. These findings were particularly notable because all participants were already receiving optimized background therapy, indicating that SGLT2 inhibitors provide additional renal protection beyond conventional blood pressure control and RAAS inhibition (21).

The DAPA-CKD trial further expanded the evidence base by demonstrating substantial renal benefits of dapagliflozin in patients with CKD both with and without diabetes. Dapagliflozin significantly reduced the risk of sustained eGFR decline, ESKD, and renal or cardiovascular death compared with placebo. Importantly, the magnitude of benefit was similar regardless of diabetic status, providing strong clinical support for the concept that SGLT2 inhibitors exert kidney-protective effects beyond glucose lowering. Given the high prevalence of hypertension among trial participants, these results are particularly relevant to hypertensive CKD, where glomerular hyperfiltration, endothelial dysfunction, and chronic inflammation contribute to progressive nephron loss (22).

Additional evidence was provided by the EMPA-KIDNEY trial, which evaluated empagliflozin in a broad CKD population, including a substantial proportion of non-diabetic patients and individuals with lower levels of albuminuria than those included in previous studies. Empagliflozin significantly reduced the risk of kidney disease progression and cardiovascular death while slowing the rate of eGFR decline. The consistency of benefit observed across diverse patient populations reinforced the notion that the renoprotective actions of SGLT2 inhibitors are largely independent of glycemic control and may be applicable across a wide spectrum of CKD etiologies, including hypertension-associated kidney disease (23).

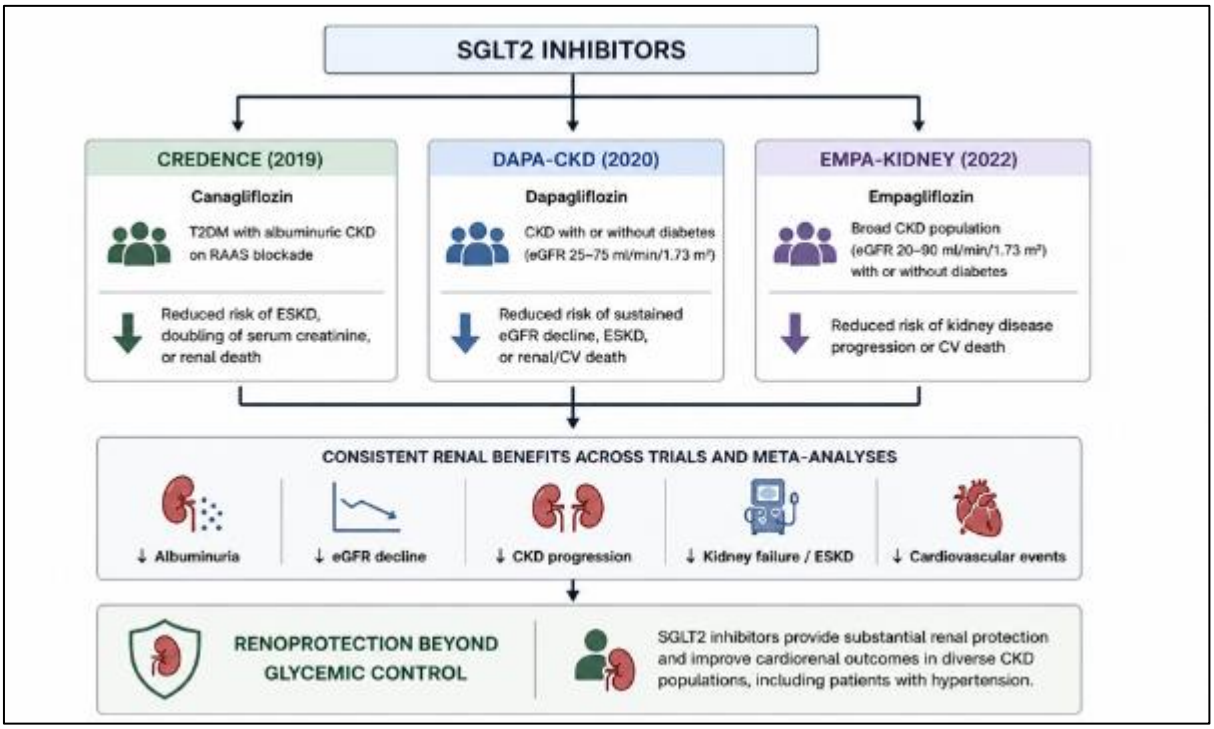

Figure 3 Clinical Evidence Supporting Renoprotection by SGLT2 Inhibitors in Hypertensive Chronic Kidney Disease

2.4. Current Clinical Implications and Future Perspectives

The growing body of evidence supporting the renoprotective effects of sodium–glucose cotransporter 2 (SGLT2) inhibitors has substantially reshaped the contemporary management of chronic kidney disease (CKD). Once regarded primarily as glucose-lowering agents for type 2 diabetes mellitus, SGLT2 inhibitors are now increasingly recognized as foundational therapies for kidney protection across a broad spectrum of CKD populations. The consistent benefits observed in major clinical trials have led to their incorporation into international guidelines, including those from the Kidney Disease: Improving Global Outcomes (KDIGO), American Diabetes Association (ADA), and European Society of Cardiology (ESC), which recommend SGLT2 inhibitors as part of standard care for eligible patients with CKD regardless of diabetic status (7).

For patients with hypertensive CKD, the clinical implications of these findings are particularly important. Hypertension remains one of the leading drivers of CKD progression, and despite advances in blood pressure control and renin–angiotensin–aldosterone system (RAAS) blockade, substantial residual renal risk persists. The consistent renal benefits observed across major trials support the incorporation of SGLT2 inhibitors into routine CKD management, particularly in patients who remain at high residual risk despite optimized blood pressure control and RAAS blockade (24).

Recent advances have also highlighted the potential benefits of combination therapy. Emerging evidence suggests that combining SGLT2 inhibitors with established renoprotective agents, including angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), and nonsteroidal mineralocorticoid receptor antagonists such as finerenone, may provide additive cardiorenal protection. As treatment strategies continue to evolve, multidrug approaches targeting complementary pathophysiological pathways may further improve long-term renal outcomes and delay progression to kidney failure. Despite these advances, several important knowledge gaps remain. Most major clinical trials have included heterogeneous CKD populations, making it difficult to isolate the specific effects of SGLT2 inhibitors in patients whose primary cause of kidney disease is hypertension. Dedicated studies focusing on hypertension-driven CKD are therefore needed to better characterize treatment responses, identify patient subgroups most likely to benefit, and clarify the long-term effects of therapy across different stages of CKD. In addition, further research is warranted to determine the optimal integration of SGLT2 inhibitors within existing antihypertensive treatment algorithms and combination renoprotective strategies (25).

Future investigations are also expected to explore precision medicine approaches, including the use of biomarkers, genetic profiling, and individualized risk stratification to guide treatment selection. A better understanding of the molecular mechanisms underlying renal protection may facilitate the development of novel therapeutic targets and improve personalization of care. Furthermore, ongoing studies examining the broader cardiorenal and metabolic effects of SGLT2 inhibitors may expand their clinical applications beyond current indications (7).

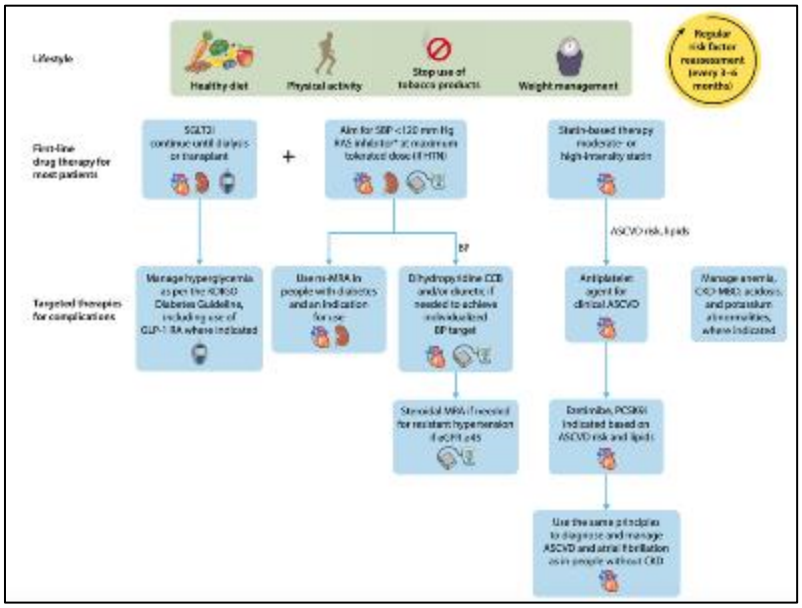

Figure 4 Integration of SGLT2 inhibitors into contemporary chronic kidney disease management and risk reduction strategies (7)

3. Conclusion

SGLT2 inhibitors provide substantial renoprotective benefits in chronic kidney disease beyond glucose lowering. Evidence from major clinical trials consistently demonstrates slower eGFR decline, reduced albuminuria, and a lower risk of kidney failure across diverse CKD populations, including individuals without diabetes. In hypertensive CKD, these benefits may complement conventional blood pressure control and RAAS blockade by addressing multiple mechanisms involved in disease progression. These findings support the incorporation of SGLT2 inhibitors as foundational components of contemporary CKD management. However, further studies specifically targeting hypertension-driven CKD are needed to better define treatment responses, optimize patient selection, and clarify long-term renal outcomes.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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