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Remission revolution; redefining CKD management from slowdown to reversal

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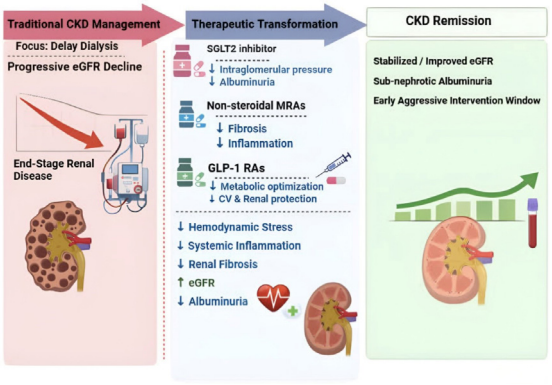
ABSTRACT



Chronic kidney disease (CKD) has historically been managed with a traditional mindset focused solely on delaying dialysis initiation rather than restoring renal function. This entrenched paradigm is shifting due to emerging evidence suggesting that significant disease regression and clinical remission are achievable goals. Here, we examine the therapeutic landscape transforming CKD management from mere slowdown to potential reversal. We discuss on sodium glucose cotransporter 2 inhibitors, non-steroidal mineralocorticoid receptor antagonists, and glucagon like peptide 1 receptor agonists alongside lifestyle modifications including dietary protein restriction and blood pressure optimization. Data indicates that early intervention targeting hemodynamic stress, systemic inflammation, and renal fibrosis can restore glomerular filtration rates (GFRs) and reduce albuminuria below nephrotic thresholds. Furthermore, clinical guidelines should evolve to prioritize remission criteria, emphasizing on aggressive early treatment windows before irreversible scarring occurs. This remission revolution necessitates a fundamental change in physician expectations and patient education, moving away from inevitable decline toward active restoration. Finally, redefining success in CKD care requires integrating these novel agents into standard practice to halt progression and actively reverse damage, offering renewed hope and improved long term outcomes for the future global CKD population while significantly reducing the economic burden associated with end stage renal disease therapy.

Implication for health policy/practice/research/medical education:
Chronic kidney disease (CKD) management has historically prioritized delaying progression rather than restoring function. This paradigm shift, termed the remission revolution, explores emerging strategies aimed at achieving structural reversal of kidney injury. Traditional therapies focusing on hemodynamic control are now complemented by regenerative medicine, anti-fibrotic agents, and precision nutrition targeting underlying metabolic dysfunctions. This review considers current evidence demonstrating that early intervention with sodium-glucose cotransporter 2 (SGLT2) inhibitors, glucagon-like peptide-1 (GLP-1) agonists, and novel anti-inflammatory pathways can induce partial remission in specific chronic kidney disease etiologies.

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Introduction

The management of chronic kidney disease is undergoing a conceptual and practical transformation from passive deceleration of decline toward an active pursuit of remission and, in selected phenotypes, partial reversal of structural and functional injury. This "remission revolution" arises from converging advances in pathophysiologic understanding, pharmacotherapy, metabolic surgery, and systems-based care that together challenge the long-held paradigm that chronic kidney disease (CKD) inexorably

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Review

progresses once established (1). The traditional focus on slowing estimated glomerular filtration rate (GFR) loss and delaying kidney replacement therapy is being replaced by strategies that target albuminuria regression, stabilization or improvement of GFR slopes, and modification of underlying drivers such as metabolic toxicity, hemodynamic overload, inflammation, and fibrosis (2). In fact, KDIGO (kidney disease: improving global outcomes) 2024 guidelines codify this shift by emphasizing albuminuria as a modifiable risk marker, recommending routine reassessment of estimated glomerular filtration rate (eGFR) and urine albumin-creatinine ratio and linking treatment intensification to changes in these parameters rather than to static staging alone (3). Within this framework, remission can be conceptualized along a spectrum that includes complete normalization of albuminuria and near-normal renal function, partial remission defined by substantial reduction in albuminuria and flattening of GFR decline, across with functional remission wherein the clinical trajectory diverges away from kidney failure despite persistent structural disease (4). This narrative review dedicated to the concept of remission revolution by redefining CKD management from slowdown to reversal.

Search strategy

For this overview, we conducted a literature search across multiple databases, including PubMed, Google Scholar, the Directory of Open Access Journals (DOAJ), Web of Science, EBSCO, Scopus, and Embase, using a variety of relevant keywords such as 'chronic kidney disease', 'SGLT2 inhibitors', 'GLP-1 agonists', 'Remission revolution', 'KDIGO 2024 guidelines' and 'albuminuria'.

Renal pathology of CKD; focus on reversible lesions

Chronic kidney disease is characterized by progressive, largely irreversible structural damage including glomerulosclerosis, tubular atrophy, interstitial fibrosis, and vascular thickening (5). However, within this chronic framework, several potentially reversible pathological lesions may coexist, and their identification is critical for preserving residual renal function (6). Acute tubular injury, often superimposed on chronic changes, represents a key reversible component; triggered by ischemia, nephrotoxins, or sepsis. This morphologic lesion manifests as tubular epithelial flattening, loss of brush border, and cast formation, yet can resolve with timely supportive care and removal of the inciting agent (6). Similarly, acute interstitial nephritis, which is frequently drug-induced by antibiotics, proton pump inhibitors, or non-steroidal anti-inflammatory drugs presents with interstitial edema and inflammatory infiltrates (7). Therefore, prompt discontinuation of the offending drug, sometimes with corticosteroid therapy, can restore function even in a chronically compromised kidney (8). Obstructive uropathy, another reversible cause, leads to tubular dilation and interstitial inflammation; relief of obstruction may halt progression and partially reverse damage if addressed before fibrosis becomes dominant (9). Malignant hypertension can induce fibrinoid necrosis of arterioles and thrombotic microangiopathy; hence, aggressive blood pressure control can reverse these vascular lesions and improve outcomes (10). Additionally, active immune-mediated glomerulonephritis flares like lupus nephritis or ANCA-associated vasculitis may accelerate CKD progression but often respond to immunosuppression, preserving nephrons (11). Even in diabetic kidney disease, early structural changes like glomerular hyperfiltration and mild mesangial expansion may partially regress with stringent glycemic and blood pressure control, sodium-glucose cotransporter 2 (SGLT2) inhibitors, or renin–angiotensin–aldosterone system (RAAS) blockade (12). Clinically, distinguishing reversible lesions from fixed fibrotic change requires integration of history, urinalysis, serology, and sometimes repeat biopsy (13). The presence of active sediment, rapid GFR decline, or systemic symptoms should prompt evaluation for treatable pathology (14). Recognizing and targeting these reversible elements offers a vital therapeutic window, potentially stabilizing kidney function, delaying dialysis, and improving quality of life. Though CKD denotes chronicity, a nuanced pathological assessment remains essential to uncover actionable, reversible processes within the broader landscape of irreversible injury (15).

The concept of remission revolution

At the mechanistic level, the possibility of remission or partial reversal hinges on the plasticity of glomerular and tubulointerstitial compartments when injurious stimuli are removed or sufficiently attenuated (16). Experimental models showed that podocyte injury, mesangial expansion, and early interstitial fibrosis can regress if hemodynamic stress, metabolic derangements, and inflammatory signaling are corrected before scar becomes fully established (5). In humans, clinical evidence for regression is most robust in conditions characterized by hemodynamic hyperfiltration, metabolic overload, or immune dysregulation where upstream drivers can be modified, including diabetic kidney disease, obesity-related glomerulopathy, and some forms of glomerulonephritis (17). Albuminuria is central in this narrative; since, reductions in urine albumin-to-creatinine ratio (UACR) correlate strongly with lower risk of GFR decline and kidney failure and are increasingly used as intermediate surrogates for remission (18). The KDIGO 2024 highlights that even modest reductions in albuminuria from high baseline levels translate into meaningful reductions in both renal and cardiovascular risk, underscoring why many contemporary trials now report remission-like outcomes such as transitions from macro- to microalbuminuria or to normoalbuminuria (19). Equally important is the recognition that GFR

responses can be biphasic, with early dips after hemodynamically active therapies like renin–angiotensin system blockers, SGLT2 inhibitors, or mineralocorticoid receptor antagonists representing beneficial reductions in intraglomerular pressure rather than harm; longer-term trajectories, not acute changes, define true reversal (20). The pharmacologic engine of the remission revolution is a multidrug platform that layers complementary agents on top of angiotensin-converting enzyme inhibitors or ARBs to achieve durable albuminuria reductions and stabilization or improvement of GFR slopes (21). Sodium–glucose cotransporter-2 inhibitors have been the first class to consistently demonstrate kidney protection beyond glucose lowering, reducing progression to kidney failure, acute kidney injury, and major cardiovascular events across a range of eGFR and albuminuria levels (22). Large contemporary new-user cohort analyses demonstrate that in real-world populations with type 2 diabetes and chronic renal failure, SGLT2 inhibitor initiation is associated with slower GFR decline and fewer composite kidney events compared with other glucose-lowering agents, with benefits extending down to advanced CKD stages (23). Mechanistically, these drugs restore tubuloglomerular feedback, reduce intraglomerular hypertension, lower proximal tubular workload, and favorably modulate inflammation and fibrosis, providing a biologic rationale for partial structural recovery when treatment is initiated early (24). The concept of remission is supported by substantial proportions of treated patients who experience large, sustained reductions in albuminuria and flattening of eGFR slopes, although complete normalization remains uncommon and is more likely in those with shorter disease duration and less chronic damage at baseline (25). Layered on this SGLT2 backbone, nonsteroidal mineralocorticoid receptor antagonism has emerged as a key antifibrotic and anti-inflammatory pillar that can deepen albuminuria responses and further modulate long-term trajectories (26). Finerenone, by selectively antagonizing mineralocorticoid receptors in the kidney and vasculature with a distinct tissue distribution profile, attenuates mineralocorticoid receptor -mediated injury and intersects with fibroblast growth factor-23 (FGF23)–Klotho pathways implicated in CKD progression (27-29). Pooled cardiovascular and kidney outcome analyses from the FIDELITY program demonstrate that finerenone, on top of optimized renin–angiotensin system blockade, reduces albuminuria and slows GFR decline in patients with type 2 diabetes and CKD, with consistent benefit across a broad spectrum of baseline GFR and albuminuria (30). Recent analyses focusing on concomitant therapy show that the effect of finerenone persists and may even be amplified when used in combination with SGLT2 inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists (28). In FIDELITY, finerenone produced greater reductions in UACR and systolic blood pressure than placebo across all subgroups, and the largest relative difference in albuminuria at 12 months was observed in patients receiving triple therapy with finerenone, an SGLT2 inhibitor, and a GLP-1 receptor agonist (31). A prespecified and real-world analysis in adults with type 2 diabetes and CKD initiating finerenone on top of background SGLT2 inhibitor and GLP-1 receptor agonist therapy reported approximately a significant reduction in UACR over six months, substantially greater than reductions seen with any single class alone, suggesting additive or synergistic effects on pathways driving proteinuria (28). Experimental work further indicates that finerenone can reverse diabetes-related down-regulation of renal GLP-1 receptors, mechanistically reinforcing the potential for combination regimens to move patients from high-risk albuminuria states toward partial or complete remission (32). Meanwhile, GLP-1 receptor agonists themselves are advancing from cardiometabolic agents to bona fide kidney drugs capable of altering CKD trajectories. Beyond their effects on glycemic control, body weight, and blood pressure, GLP-1 receptor agonists exert direct renal actions through natriuresis, reduction of oxidative stress, modulation of inflammatory cascades, and possible improvement of endothelial function (33). The FLOW trial with semaglutide provides pivotal evidence; since in people with type 2 diabetes and CKD, semaglutide reduced the risk of major kidney outcomes by about 24% compared with placebo, with benefits observed across CKD severity strata and accompanied by clinically meaningful improvements in glycemic control, weight, and blood pressure at two years (34). When these drugs are administered alongside SGLT2 inhibitors and finerenone, their complementary effects on metabolic load, hemodynamics, inflammation, fibrosis, and tubular stress create a multidimensional therapeutic environment in which regression of albuminuria and stabilization of eGFR become realistic targets for many patients (35). Importantly, the updated KDIGO 2024 clinical practice guideline embeds these pharmacologic advances within a refined risk-stratified approach that supports earlier and more aggressive intervention with remission in mind. The guideline reaffirms the centrality of albuminuria on both kidney and cardiovascular outcomes (3,19). Notably, KDIGO encourages more frequent monitoring of eGFR and albuminuria in higher-risk patients or when therapeutic decisions hinge on trajectory, aligning practice with a dynamic model where response to therapy guides escalation or de-escalation. This dynamic framework underpins remission definitions, creating space for clinicians to define success not only as delayed dialysis but as measurable shifts in albuminuria categories, improved risk grid position, and deceleration or reversal of GFR slope over multi-year horizons (3,19,36).

Focus on metabolic interventions

Beyond drug therapy, metabolic interventions illustrate the potential for partial reversal of CKD through aggressive

modification of upstream drivers. Obesity is tightly linked to incident CKD and its progression via glomerular hyperfiltration, activation of the renin–angiotensin–aldosterone axis, insulin resistance, and systemic inflammation. Some researchers believe that bariatric surgery targets these mechanisms simultaneously through profound and sustained weight loss, improved insulin sensitivity, and hormonal changes (37). Retrospective cohort data from large UK primary care databases show that bariatric surgery in severely obese patients with insulin-treated type 2 diabetes is associated with improved renal outcomes over five years, including better eGFR trajectories and protection against microalbuminuria, with signals toward reduced cardiovascular events (38). Other observational series in individuals with morbid obesity and established CKD report significant decreases in proteinuria, stabilization or modest decline in kidney function consistent with correction of hyperfiltration, and durable improvements in blood pressure, glycemic control, and inflammatory parameters over one to five years (39). More recent longitudinal analyses across CKD and non-CKD populations suggest that bariatric surgery may reduce the risk of kidney function decline and kidney failure meaningfully, and that in patients with stage 3–4 CKD, renal can actually improve over three years post-surgery (40). These findings indicate that in metabolically driven CKD, aggressive weight loss and metabolic surgery can induce a form of remission characterized by lower albuminuria, improved or stabilized kidney function, and reduced cardiovascular risk, especially when combined with optimized medical therapy. However, the side effects of bariatric surgery and its risks/benefits should be kept in mild (40,41).

The concept of re-engineering of CKD management

Translating these advances into routine care requires re-engineering CKD management around early detection, aggressive risk-factor control, multidrug combination therapy, and individualized targets that explicitly include remission metrics (42). Early identification through systematic screening of at-risk populations, like those with diabetes, hypertension, obesity, cardiovascular disease, or family history remains foundational, with urine albumin-creatinine ratio and eGFR as the core tests (43). Once CKD is detected, prompt optimization of blood pressure using renin–angiotensin system blockade, careful glycemic control, lipid management, and lifestyle measures such as salt restriction, smoking cessation, and physical activity is critical to create a platform for disease modification (44). On this background, timely initiation of an SGLT2 inhibitor is now recommended for most patients with CKD and diabetes and is increasingly considered even in non-diabetic CKD with albuminuria, while finerenone is added in those with persistent albuminuria and appropriate serum potassium and eGFR thresholds (45). In the meantime, GLP-1 receptor agonists are integrated for additional glycemic, weight, and cardiovascular benefit, and may be prioritized in patients with obesity or high atherosclerotic risk, with a growing rationale for their use primarily as kidney-protective agents (46). In parallel, clinicians should periodically reassess indications for bariatric surgery or structured weight-loss programs in appropriate candidates, recognizing their potential to shift renal trajectories (40). Accordingly, clinicians can frame therapeutic goals in terms of moving from higher-risk to lower-risk KDIGO color zones, achieving specific percentage reductions in albuminuria, and flattening individualized eGFR slopes, while remaining honest that structural scarring often limits complete reversal (3,19). Real-world data on combination therapy with finerenone, SGLT2 inhibitors, and GLP-1 receptor agonists illustrate that substantial proportions of patients can achieve appropriate reduction in albuminuria within one year, translating into lower long-term event rates (28). While these advances do not equate to cure, they justify a more ambitious therapeutic stance and offer a framework for future research into true regenerative strategies, including cellular therapies, anti-fibrotic biologics, and interventions targeting senescence and repair pathways (28). The remission revolution in CKD thus represents not a single breakthrough but an evolving synthesis of pathophysiologic insight, multidrug regimens, metabolic transformation, and dynamic risk-based care, collectively redefining what is possible for patients who previously faced a near-certain march toward kidney failure (25).

Conclusion

The excursion through the remission revolution culminates in a profound realization that CKD is no longer a one-way ticket to dialysis or transplantation. For decades, the medical community accepted progression as inevitable, focusing solely on delaying the unavoidable. However, emerging evidence now confirms that functional recovery is genuinely achievable. This transformation relies on early intervention, precision medicine, and aggressive lifestyle modification tailored to individual metabolic profiles. Patients are no longer passive recipients of care but they are active architects of their health outcomes, empowered by knowledge. By targeting underlying metabolic drivers, inflammation, and fibrosis, we can halt deterioration and restore renal function. The future of nephrology lies in personalized protocols combining pharmacological advancements with rigorous nutritional therapy. This shift demands a collaborative effort between clinicians and patients to dismantle outdated fatalism. We stand at the precipice of a new era where remission is the standard goal, not merely an exceptional outlier. Embracing this revolution requires courage to challenge established norms and commitment to sustained management. Eventually, redefining CKD management empowers individuals to reclaim their lives from kidney failure, transforming anxiety into agency.

Authors' contribution

Conceptualization: Sepideh Hajian, Azadeh Zahedi Far.
Data curation: Azadeh Zahedi Far.
Investigation: Azadeh Zahedi Far.
Resources: Sepideh Hajian.
Supervision: Azadeh Zahedi Far.
Validation: Sepideh Hajian.
Visualization: Sepideh Hajian.
Writing—original draft: Azadeh Zahedi Far, Sepideh Hajian
Writing—review and editing: Sepideh Hajian, Sepideh Hajian.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

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