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How Diabetes Mellitus Leads to Dialysis: A Narrative Review

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ABSTRACT

Background: Diabetes mellitus is the leading cause of chronic kidney disease (CKD) and end-stage kidney disease (ESKD) worldwide, accounting for a substantial proportion of patients requiring dialysis or kidney transplantation. The increasing global prevalence of diabetes has resulted in a parallel rise in diabetic kidney disease (DKD), creating a significant clinical and public health burden. Understanding the mechanisms that drive progression from diabetes to dialysis is essential for improving prevention and management strategies.

Objective: This narrative review aims to examine the pathophysiological mechanisms underlying diabetic kidney disease, describe its clinical progression from early renal injury to dialysis-dependent kidney failure, and evaluate current and emerging therapeutic approaches designed to delay or prevent progression to end-stage kidney disease.

Methods: A comprehensive narrative review of contemporary literature, clinical practice guidelines, landmark clinical trials, and nephrology reference sources was conducted. Evidence relating to the epidemiology, pathogenesis, diagnosis, progression, prevention, and management of diabetic kidney disease was synthesized to provide an integrated overview of the disease continuum.

Results: Chronic hyperglycemia initiates a complex cascade of metabolic and hemodynamic disturbances, including glomerular hyperfiltration, activation of the renin–angiotensin–aldosterone system, oxidative stress, inflammation, formation of advanced glycation end-products, and progressive renal fibrosis. These mechanisms contribute to albuminuria, declining glomerular filtration rate, chronic kidney disease progression, and ultimately end-stage kidney disease requiring renal replacement therapy. Early detection through albuminuria screening and estimated glomerular filtration rate assessment remains critical for identifying patients at risk. Contemporary therapeutic strategies, including optimized glycemic control, blood pressure management, renin–angiotensin system blockade, sodium-glucose cotransporter-2 (SGLT2) inhibitors, glucagon-like peptide-1 (GLP-1) receptor agonists, and non-steroidal

mineralocorticoid receptor antagonists such as finer none, have significantly improved renal and cardiovascular outcomes and reduced progression to dialysis.

Conclusion: Diabetic kidney disease remains a major cause of kidney failure and dialysis worldwide. Early diagnosis, aggressive risk factor modification, and implementation of evidence-based Reno protective therapies are essential for slowing disease progression. Recent therapeutic advances have transformed the management of DKD and offer new opportunities to delay or prevent dialysis-dependent kidney failure. Continued research into precision medicine, regenerative therapies, and novel molecular targets may further improve outcomes and reduce the global burden of diabetic kidney disease.

Keywords: Albuminuria; Chronic kidney disease; Diabetes mellitus; Diabetic kidney disease; Diabetic nephropathy; Dialysis; End-stage kidney disease; Finerenone; GLP-1 receptor agonists; Renal replacement therapy; SGLT2 inhibitors.

1. INTRODUCTION

Diabetes mellitus has emerged as one of the most significant global health challenges of the 21st century, affecting hundreds of millions of individuals worldwide and imposing an enormous burden on healthcare systems. Beyond its effects on glucose metabolism, diabetes is a progressive multisystem disease capable of damaging nearly every organ in the body. Among its chronic complications, diabetic kidney disease (DKD) stands out as one of the most devastating because it frequently progresses to end-stage kidney disease (ESKD), ultimately requiring lifelong dialysis or kidney transplantation. Today, diabetes is recognized as the leading cause of ESKD worldwide and accounts for a substantial proportion of patients receiving renal replacement therapy. (American Diabetes Association, 2025; KDIGO, 2024)

The progression from diabetes to dialysis is neither sudden nor inevitable. Rather, it represents the culmination of years of complex metabolic, hemodynamic, inflammatory, and fibrotic processes occurring within the kidneys. As highly vascular organs, the kidneys are particularly vulnerable to chronic hyperglycemia. Persistent elevations in blood glucose trigger a cascade of pathological events, including glomerular hyperfiltration, oxidative stress, formation of advanced glycation end products, endothelial dysfunction, and activation of the renin–angiotensin–aldosterone system. Over time, these mechanisms lead to progressive injury of the glomeruli, tubules, interstitium, and renal vasculature, resulting in declining glomerular filtration rate (GFR), persistent albuminuria, and irreversible nephron loss. (Tuttle et al., 2022; Webster et al., 2022)

The global burden of diabetic kidney disease continues to rise in parallel with increasing rates of type 2 diabetes mellitus, obesity, sedentary lifestyles, population aging, and urbanization. Despite remarkable advances in glycemic management and the introduction of novel Renoprotective therapies such as sodium-glucose cotransporter-2 (SGLT2) inhibitors and non-steroidal mineralocorticoid receptor antagonists, a considerable number of patients still progress to dialysis-dependent kidney failure. Consequently, diabetic kidney disease remains a major contributor to morbidity, mortality, healthcare expenditure, and reduced quality of life worldwide. (USRDS, 2024; KDIGO, 2024)

2. EPIDEMIOLOGY AND GLOBAL BURDEN

Diabetic kidney disease (DKD) has emerged as one of the most important causes of chronic kidney disease (CKD) and end-stage kidney disease (ESKD) worldwide. It is estimated that DKD accounts for approximately 30–50% of all cases of ESKD, making diabetes mellitus the leading

cause of dialysis initiation and kidney transplantation in many countries. The burden is particularly pronounced in individuals with type 2 diabetes mellitus because of its rapidly increasing prevalence, delayed diagnosis, and prolonged exposure to asymptomatic hyperglycemia. Epidemiological studies indicate that nearly one in three individuals with diabetes will develop some degree of kidney involvement during their lifetime, highlighting the enormous public health impact of this complication. (International Diabetes Federation, 2025; KDIGO, 2024)

The global prevalence of diabetes continues to rise at an alarming rate, driven by increasing obesity, sedentary lifestyles, urbanization, population aging, and dietary changes. As a consequence, the number of patients developing diabetic kidney disease and progressing to kidney failure is also increasing. This trend has placed substantial pressure on healthcare systems worldwide, particularly because renal replacement therapies such as dialysis and kidney transplantation are resource-intensive and costly. (International Diabetes Federation, 2025; USRDS, 2024)

The burden of diabetic kidney disease is disproportionately higher in low- and middle-income countries, where access to healthcare resources remains limited. Inadequate screening programs, delayed diagnosis, poor glycemic control, restricted availability of nephrology services, and limited access to modern Renoprotectivetherapies often result in late presentation and accelerated progression to ESKD. Consequently, many patients require emergency dialysis initiation or remain untreated due to financial and infrastructural constraints. (GBD Study, 2023; KDIGO, 2024)

Beyond its clinical consequences, diabetic kidney disease imposes a substantial socioeconomic burden on patients, families, and healthcare systems. The costs associated with long-term dialysis, repeated hospitalizations, management of cardiovascular complications, and loss of productivity contribute significantly to healthcare expenditure worldwide. As the prevalence of diabetes continues to increase, preventing the progression of diabetic kidney disease has become a major global health priority aimed at reducing both mortality and the growing demand for renal replacement therapy. (USRDS, 2024; International Diabetes Federation, 2025)

3. NORMAL RENAL STRUCTURE AND FUNCTION

The kidneys are specialized organs that maintain internal homeostasis by regulating fluid balance, electrolytes, acid–base status, and waste excretion. Each kidney contains about one million nephrons, the functional units responsible for urine formation. A nephron consists of the glomerulus and a tubular system (proximal tubule, loop of Henle, distal tubule, and collecting duct), which together filter blood and selectively reabsorb and excrete substances. (Brenner and Rector's The Kidney, 2024)

The glomerulus acts as a selective filtration barrier made up of endothelial cells, the basement membrane, and podocytes, allowing passage of water and small solutes while retaining proteins and cells. The renal tubules further regulate reabsorption and secretion to maintain fluid, electrolyte, and acid–base balance. (Brenner and Rector's The Kidney, 2024; Johnson et al., 2024)

In addition to excretory functions, the kidneys have key endocrine roles, including RAAS activation for blood pressure control, erythropoietin production for red blood cell formation, and vitamin D activation for bone health. They also contribute to gluconeogenesis and hormone metabolism. (Guyton & Hall, 2021; KDIGO, 2024)

Due to their high metabolic demand and rich blood supply, the kidneys are highly

susceptible to diabetic injury, and damage to their structures leads to progressive chronic kidney disease and eventual end-stage kidney disease (KDIGO, 2024; Tuttle et al., 2022).

4. PATHOGENESIS OF DIABETIC KIDNEY DISEASE

Diabetic kidney disease (DKD) develops through a complex interplay of metabolic, hemodynamic, inflammatory, and fibrotic mechanisms triggered by chronic hyperglycemia. Persistent elevation of blood glucose initiates a cascade of pathological changes that affect glomerular, tubular, vascular, and interstitial structures of the kidney. These abnormalities progressively impair renal function, resulting in albuminuria, declining glomerular filtration rate (GFR), and ultimately end-stage kidney disease (ESKD). Understanding these mechanisms is essential for appreciating how diabetes mellitus gradually progresses to dialysis-dependent kidney failure. (KDIGO, 2024; Tuttle et al., 2022; Webster et al., 2022; Alicic et al., 2022)

4.1 Chronic Hyperglycemia and Glomerular Injury

Chronic hyperglycemia is the primary initiating factor in diabetic kidney disease. Increased plasma glucose leads to excessive filtration of glucose across the glomerulus and enhanced reabsorption in the proximal tubule through sodium-glucose cotransporter-2 (SGLT2) proteins. This increased glucose and sodium reabsorption reduces sodium delivery to the macula densa, disrupting the normal tubuloglomerular feedback mechanism. As a consequence, afferent arteriolar dilation occurs, resulting in glomerular hyperfiltration and increased intraglomerular pressure. Although initially compensatory, prolonged hyperfiltration causes mechanical stress on glomerular structures, leading to endothelial injury, mesangial expansion, and progressive nephron damage. Over time, sustained intraglomerular hypertension contributes significantly to the development and progression of diabetic kidney disease. (Heerspink et al., 2022; Tuttle et al., 2022)

4.2 Formation of Advanced Glycation End Products (AGEs)

Persistent hyperglycemia promotes the non-enzymatic glycation of proteins, lipids, and nucleic acids, resulting in the formation of advanced glycation end products (AGEs). These compounds accumulate within renal tissues and interact with specific cellular receptors known as receptors for advanced glycation end products (RAGE). Activation of the AGE–RAGE pathway stimulates oxidative stress, inflammatory signaling, and extracellular matrix production. This process contributes to thickening of the glomerular basement membrane, mesangial expansion, glomerulosclerosis, and tubulointerstitial fibrosis. AGEs also alter vascular structure and function, further aggravating renal injury. (Forbes & Thorburn, 2021; KDIGO, 2024)

4.3 Oxidative Stress and Inflammation

Excess intracellular glucose metabolism increases the production of reactive oxygen species (ROS) within renal cells. These highly reactive molecules cause oxidative damage to endothelial cells, podocytes, mesangial cells, and tubular epithelial cells.

Oxidative stress activates multiple pro-inflammatory pathways, including nuclear factor-kappa B (NF-κB), leading to increased production of cytokines, chemokines, and adhesion molecules. The resulting chronic low-grade inflammation promotes cellular injury, fibrosis, and progressive deterioration of kidney function. Oxidative stress and inflammation act synergistically and are now recognized as central drivers of diabetic kidney disease progression. (Alicic et al., 2022; Webster et al., 2022)

4.4 Activation of the Renin–Angiotensin–Aldosterone System (RAAS)

The renin–angiotensin–aldosterone system (RAAS) plays a crucial role in the progression of diabetic kidney disease. Chronic activation of this system results in preferential constriction of the efferent arteriole, thereby increasing intraglomerular pressure and exacerbating glomerular hyperfiltration.

In addition to its hemodynamic effects, angiotensin II promotes inflammation, oxidative stress, cellular hypertrophy, and extracellular matrix deposition. These actions contribute to mesangial expansion, podocyte injury, proteinuria, and tubulointerstitial fibrosis. Persistent RAAS activation, therefore, accelerates structural kidney damage and progression toward ESKD. (KDIGO, 2024; Brenner & Rector, 2024)

4.5 Podocyte Injury and Glomerulosclerosis

Podocytes are highly specialized epithelial cells that form an essential component of the glomerular filtration barrier. Their structural integrity is critical for preventing the leakage of plasma proteins into the urine.

In diabetic kidney disease, podocytes are exposed to mechanical stress from glomerular hypertension, metabolic toxicity from hyperglycemia, oxidative injury, and inflammatory mediators. These insults result in podocyte dysfunction, apoptosis, and detachment from the glomerular basement membrane. Loss of podocytes disrupts the filtration barrier, leading to albuminuria and progressive glomerulosclerosis.

Because podocytes possess limited regenerative capacity, their loss is largely irreversible and represents a critical step in the progression from early diabetic nephropathy to advanced chronic kidney disease and eventual dialysis-dependent kidney failure. (Tuttle et al., 2022; Perkovic et al., 2022)

5. EARLY FUNCTIONAL CHANGES IN DIABETIC KIDNEY DISEASE

Early diabetic kidney disease (DKD) is typically silent, with functional renal changes occurring long before symptoms appear (ADA, 2025; KDIGO, 2024).

The earliest hallmark is glomerular hyperfiltration, driven by increased intraglomerular pressure due to altered tubuloglomerular feedback and hyperglycemia-related hemodynamic changes. While initially compensatory, persistent hyperfiltration leads to progressive glomerular injury (Heerspink et al., 2022; KDIGO, 2024).

This is followed by microalbuminuria, the first clinically detectable sign of DKD, caused by endothelial dysfunction, basement membrane thickening, and podocyte injury. It may progress to macroalbuminuria if untreated (ADA, 2025; Tuttle et al., 2022).

Over time, gradual nephron loss leads to declining eGFR, eventually progressing to CKD and possibly ESKD. This sequence—hyperfiltration, albuminuria, and falling eGFR—represents the typical natural history of DKD. Early detection using UACR and eGFR, along with tight glycemic and blood pressure control and renoprotective therapies, provides a critical window to slow progression and reduce the risk of dialysis (ADA, 2025; KDIGO, 2024).

6. TRANSITION TO CHRONIC KIDNEY DISEASE AND DIALYSIS

As diabetic kidney disease (DKD) progresses, loss of functioning nephrons triggers compensatory hypertrophy and hyperfiltration in remaining nephrons to

maintain GFR. While initially adaptive, these changes become maladaptive over time, increasing intraglomerular pressure and accelerating further renal injury (Brenner & Rector, 2024; KDIGO, 2024).

With ongoing nephron loss, the kidney's ability to maintain fluid, electrolyte, and metabolic balance declines, marking progression through CKD stages 3–5. This leads to retention of metabolic waste and multisystem dysfunction. Declining GFR is associated with accumulation of uremic toxins, electrolyte disturbances (notably hyperkalemia), metabolic acidosis, anemia due to reduced erythropoietin, mineral and bone disorders, and increased cardiovascular risk (Floege et al., 2024; KDIGO, 2024).

In advanced stages, uremic symptoms such as fatigue, anorexia, nausea, pruritus, cognitive impairment, fluid overload, and refractory hypertension become prominent, often necessitating renal replacement therapy (Webster et al., 2022; KDIGO, 2024).

When eGFR falls to ~15 mL/min/1.73 m² or when significant symptoms and metabolic complications occur, dialysis or transplantation is required. This final transition can be delayed with early detection and appropriate intervention (KDIGO, 2024; USRDS, 2024).

7. STAGING OF CHRONIC KIDNEY DISEASE IN DIABETES

Accurate staging of CKD is essential in diabetic kidney disease (DKD) for assessing severity, guiding treatment, and predicting outcomes. The KDIGO system combines estimated glomerular filtration rate (eGFR) with albuminuria to stratify risk for progression and cardiovascular events (KDIGO, 2024).

Based on eGFR, CKD is classified into five stages: Stage 1 (≥90 mL/min/1.73 m² with kidney damage), Stage 2 (mild reduction), Stage 3a–3b (moderate reduction), Stage 4 (severe reduction), and Stage 5 (<15 mL/min/1.73 m², kidney failure). Risk of complications and progression to ESKD increases with advancing stage. Albuminuria is categorized as A1 (normal to mildly increased), A2 (moderately increased), and A3 (severely increased). Higher albuminuria reflects greater glomerular damage and faster disease progression. Together with eGFR, it forms the KDIGO "heat map" risk model, where low eGFR and high albuminuria (A3) indicate the highest risk of ESKD and cardiovascular events (ADA, 2025; KDIGO, 2024).

Clinically, early CKD stages focus on intensive glycemic and blood pressure control, lifestyle modification, and renoprotective therapy to slow progression. Advanced stages require management of complications (e.g., anemia, bone-mineral disorders) and timely preparation for dialysis or transplantation. Thus, CKD staging provides a structured roadmap from early DKD to potential renal replacement therapy (KDIGO, 2024; NKF, 2023).

8. PROGRESSION FROM MICROALBUMINURIA TO DIALYSIS

Microalbuminuria is the earliest clinically detectable sign of diabetic kidney disease (DKD), defined as UACR 30–300 mg/g or urinary albumin excretion of 30–300 mg/day. It is often asymptomatic but indicates early glomerular injury and can still be partially reversible with tight glycemic and blood pressure control, lifestyle modification, and Reno protective therapy. Persistent microalbuminuria strongly predicts CKD progression and cardiovascular risk (ADA, 2025; Tuttle et al., 2022).

Progression to macroalbuminuria (overt proteinuria) reflects more advanced glomerular damage due to podocyte loss,

endothelial dysfunction, inflammation, and structural changes such as glomerulosclerosis and tubulointerstitial fibrosis. This is typically associated with accelerating nephron loss and declining GFR (Heerspink et al., 2022).

As DKD progresses, adaptive hyperfiltration in remaining nephrons becomes maladaptive, increasing intraglomerular pressure and driving a self-perpetuating cycle of kidney injury, proteinuria, and declining renal function (Webster et al., 2022).

Eventually, CKD develops with worsening eGFR and metabolic complications including fluid overload, hypertension, electrolyte imbalance, metabolic acidosis, anemia, and uremia. When eGFR falls to ~10–15 mL/min/1.73 m² or symptomatic uremia occurs, renal replacement therapy (dialysis or transplantation) becomes necessary (KDIGO, 2024; USRDS, 2024).

9. CLINICAL MANIFESTATIONS OF DIABETIC KIDNEY DISEASE

Diabetic kidney disease (DKD) is often "silent" in early stages, with patients remaining asymptomatic despite ongoing renal injury, making routine screening for albuminuria and eGFR essential for early detection and intervention (ADA, 2025; KDIGO, 2024).

As kidney function declines, nonspecific symptoms appear due to uremic toxin accumulation, including fatigue, weakness, anorexia, nausea, poor concentration, and sleep disturbances (Webster et al., 2022).

Fluid and electrolyte imbalances then develop, leading to edema, hypertension, and in severe cases, pulmonary edema. Metabolic complications such as hyperkalemia, metabolic acidosis, hyperphosphatemia, and calcium imbalance contribute to arrhythmias, bone disease, and vascular calcification (KDIGO, 2024; Floege et al., 2024).

Anemia is common in advanced DKD due to reduced erythropoietin production, often worsened by iron deficiency and inflammation, resulting in fatigue and reduced functional capacity (KDIGO Anemia Guideline, 2025).

In end-stage disease, systemic and neurological features such as pruritus, cognitive impairment, neuropathy, and uremic encephalopathy may occur. Severe uremia, refractory fluid overload, and persistent metabolic disturbances ultimately necessitate renal replacement therapy (dialysis or transplantation) (KDIGO, 2024; Daugirdas et al., 2021).

10. MECHANISMS OF PROGRESSION TO END-STAGE KIDNEY DISEASE

Progression of diabetic kidney disease (DKD) to end-stage kidney disease (ESKD) is driven by persistent metabolic, hemodynamic, inflammatory, and fibrotic injury. Although hyperglycemia initiates damage, progression often becomes self-perpetuating, and renal decline may continue even after glycemic control improves. Chronic hyperglycemia promotes advanced glycation end products (AGEs), oxidative stress, and endothelial dysfunction, while RAAS activation increases intraglomerular pressure and proteinuria, accelerating structural damage (KDIGO, 2024; Tuttle et al., 2022).

Proteinuria further worsens injury through tubular toxicity and activation of inflammatory pathways, leading to the release of cytokines and growth factors that amplify renal damage. Persistent activation of profibrotic mediators such as TGF-β and CTGF promotes glomerulosclerosis and tubulointerstitial fibrosis, which represent the final common pathway of irreversible kidney failure (Alicic et al., 2022).

As nephron loss progresses, compensatory hyperfiltration and hypertrophy in remaining nephrons initially maintain GFR but ultimately become maladaptive, increasing intraglomerular pressure and accelerating further injury. Over time, these mechanisms fail, leading to declining renal function, accumulation of uremic toxins, metabolic derangements, and loss of homeostasis, culminating in ESKD. At this stage, renal replacement therapy (dialysis or transplantation) becomes necessary for survival (Perkovic et al., 2022; USRDS, 2024).

11. DIAGNOSIS OF DIABETIC KIDNEY DISEASE

Early diagnosis of diabetic kidney disease (DKD) is crucial because timely intervention can slow progression and reduce the risk of end-stage kidney disease (ESKD). Since DKD is often asymptomatic in early stages, routine screening is essential for early detection (ADA, 2025; KDIGO, 2024).

Diagnosis is based on persistent albuminuria, reduced eGFR, and exclusion of other kidney diseases. The most sensitive early marker is urine albumin-to-creatinine ratio (UACR); a persistent value ≥30 mg/g (confirmed over ≥3 months) indicates kidney damage and predicts both CKD progression and cardiovascular risk (ADA, 2025; KDIGO, 2024).

Renal function is assessed using serum creatinine and estimated GFR, commonly calculated with CKD-EPI equations. A declining eGFR reflects progressive nephron loss and increasing risk of ESKD and dialysis requirement (KDIGO, 2024; NKF, 2023).

Additional tests such as renal ultrasound, urine microscopy, serology, and kidney biopsy (in selected atypical cases) help assess severity and exclude non-diabetic causes. Early diagnosis enables prompt initiation of glycemic control, blood pressure optimization, and Renoprotective therapies, which significantly delay progression to dialysis-dependent kidney failure.

12. ROLE OF ALBUMINURIA IN PROGRESSION TO DIALYSIS

Albuminuria is a key biomarker in diabetic kidney disease (DKD), reflecting early glomerular injury due to podocyte damage, basement membrane thickening, endothelial dysfunction, and increased intraglomerular pressure. It often appears before any measurable decline in eGFR and is strongly associated with faster CKD progression, higher cardiovascular risk, and increased mortality (Tuttle et al., 2022; Heerspink et al., 2022).

Importantly, albuminuria is not only a marker but also a mediator of damage, as filtered proteins trigger tubular inflammation, oxidative stress, and profibrotic signaling, accelerating tubulointerstitial fibrosis and nephron loss.

Because of its prognostic significance, reducing albuminuria is a major therapeutic target. ACE inhibitors, ARBs, SGLT2 inhibitors, and non-steroidal MRAs (e.g., Finerenone) have all been shown to lower albuminuria and slow DKD progression. Reductions in albuminuria correlate with slower eGFR decline, reduced risk of kidney failure, and delayed need for dialysis, making it an important parameter for monitoring and treatment response (KDIGO, 2024; Perkovic et al., 2022).

13. MODERN THERAPEUTIC APPROACHES

Management of diabetic kidney disease (DKD) has shifted from glucose-focused therapy to a comprehensive strategy targeting kidney protection, cardiovascular risk reduction, and delayed progression to

end-stage kidney disease (ESKD) (KDIGO, 2024; ADA, 2025).

Glycemic control remains central, with individualized HbA1c targets to reduce microvascular damage while minimizing hypoglycemia risk. Tight glucose control helps slow glomerular injury and nephron loss (Tuttle et al., 2022).

Blood pressure control is equally critical, with a general target of <130/80 mmHg in patients with DKD and albuminuria. Hypertension management reduces intraglomerular pressure, albuminuria, and cardiovascular risk (KDIGO, 2024).

Renin–angiotensin system (RAS) blockade using ACE inhibitors or ARBs is first-line therapy, reducing proteinuria and providing direct renoprotective effects beyond blood pressure lowering, thereby slowing CKD progression (NKF, 2023; Brenner & Rector, 2024).

Lifestyle measures—including sodium restriction, weight control, exercise, smoking cessation, and avoidance of nephrotoxins—support overall metabolic and cardiovascular health.

Newer therapies such as SGLT2 inhibitors, GLP-1 receptor agonists, and non-steroidal MRAs (e.g., finerenone) have significantly improved outcomes by providing additional reno- and cardioprotection, further delaying progression to dialysis-dependent kidney failure (KDIGO, 2024).

14. ROLE OF SGLT2 INHIBITORS IN PREVENTING DIALYSIS

Sodium–glucose cotransporter-2 (SGLT2) inhibitors are a major therapeutic advance in diabetic kidney disease (DKD), initially developed for glycemic control but now recognized for strong reno- and cardioprotective effects, including slowing progression to kidney failure and reducing cardiovascular events (KDIGO, 2024; ADA, 2025; Heerspink et al., 2022).

They act by inhibiting glucose and sodium reabsorption in the proximal tubule, restoring tubuloglomerular feedback, reducing intraglomerular pressure, and decreasing hyperfiltration, a key mechanism of DKD progression (Heerspink et al., 2022; Tuttle et al., 2022; KDIGO, 2024).

In addition, they lower albuminuria, improve blood pressure, support modest weight loss, and reduce renal inflammation and fibrosis. Large trials such as DAPA-CKD, CREDENCE, and EMPA-KIDNEY confirm significant reductions in kidney failure, cardiovascular death, and heart failure hospitalization in both diabetic and non-diabetic CKD patients. Consequently, SGLT2 inhibitors are now recommended as first-line Reno protective therapy in DKD and are central to strategies aimed at delaying or preventing dialysis (Perkovic et al., 2022; Webster et al., 2022; KDIGO, 2024).

15. ROLE OF GLP-1 RECEPTOR AGONISTS

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are an important adjunct in type 2 diabetes and diabetic kidney disease (DKD), primarily used for glycemic control but also offering cardiovascular and renal benefits that help slow disease progression (ADA, 2025; KDIGO, 2024; Tuttle et al., 2022).

They enhance glucose-dependent insulin secretion, suppress glucagon, delay gastric emptying, and increase satiety, resulting in improved glycemic control and weight loss. They also exert anti-inflammatory, anti-oxidative, and anti-atherosclerotic effects that support cardio-renal protection (ADA, 2025; Mann et al., 2021; Tuttle et al., 2022).

In DKD, GLP-1 RAs reduce albuminuria and improve key risk factors such as obesity, hypertension, and cardiovascular disease, though their direct Reno protective effects are less pronounced than SGLT2 inhibitors. They are therefore considered complementary therapy within a multifactorial approach (Tuttle et al., 2022; Heerspink et al., 2022; KDIGO, 2024).

Large cardiovascular outcome trials, including LEADER, SUSTAIN-6, and REWIND, have shown reductions in major adverse cardiovascular events, which is particularly important given the high cardiovascular mortality in DKD patients (Marso et al., 2016; Gerstein et al., 2019; Mann et al., 2021).

Current guidelines recommend GLP-1 RAs for patients requiring additional glycemic control, especially those with obesity or established cardiovascular disease. Overall, they help indirectly delay progression to end-stage kidney disease by improving metabolic and cardiovascular risk profiles (ADA, 2025; KDIGO, 2024; Heerspink et al., 2022).

16. FINERENONE AND NON-STEROIDAL MINERALOCORTICOID RECEPTOR ANTAGONISTS

Finerenone is a non-steroidal mineralocorticoid receptor antagonist (ns-MRA) that represents a key advancement in diabetic kidney disease (DKD) management. Compared with steroidal MRAs, it more selectively blocks mineralocorticoid receptors with a lower risk of hyperkalemia and hormonal adverse effects, while offering cardio-renal protection beyond blood pressure control (KDIGO, 2024; Bakris et al., 2021; Agarwal et al., 2022).

It reduces inflammation, oxidative stress, and fibrosis driven by overactivation of mineralocorticoid receptors, thereby slowing structural and functional kidney damage. These effects complement ACE inhibitor/ARB and SGLT2 inhibitor therapy in DKD (Bakris et al., 2021; Perkovic et al., 2022; KDIGO, 2024).

Clinical trials, including FIDELIO-DKD and FIGARO-DKD, have shown that finerenone reduces albuminuria, slows eGFR decline, and lowers risks of end-stage kidney disease and cardiovascular events in patients with type 2 diabetes and CKD (Bakris et al., 2021; Pitt et al., 2021; Agarwal et al., 2022).

Current guidelines recommend Finerenone for patients with persistent albuminuria despite optimized standard therapy. Used alongside RAS blockers and increasingly SGLT2 inhibitors, it provides additive renal and cardiovascular protection and helps delay progression to dialysis-dependent kidney failure (KDIGO, 2024; ADA, 2025).

17. INDICATIONS FOR DIALYSIS IN DIABETIC KIDNEY DISEASE

Dialysis is initiated when kidneys can no longer maintain fluid, electrolyte, acid–base, and metabolic balance. The decision is based mainly on clinical status rather than a fixed eGFR threshold, aiming to prevent life-threatening complications and improve quality of life (KDIGO, 2024; USRDS, 2024).

Absolute indications include refractory hyperkalemia, severe metabolic acidosis, fluid overload unresponsive to diuretics, and symptomatic uremia such as encephalopathy, pericarditis, persistent nausea/vomiting, malnutrition, pruritus, and bleeding due to platelet dysfunction (KDIGO, 2024; USRDS, 2024).

Although an eGFR of ~10–15 mL/min/1.73 m² is often used for planning, initiation should be individualized based on symptoms, complications, and overall clinical condition. Patients with diabetic kidney disease often require earlier dialysis

due to faster progression and higher cardiovascular burden (National Kidney Foundation, 2023; KDIGO, 2024).

Early nephrology referral is essential to allow education, modality choice, and timely vascular access or peritoneal dialysis catheter placement. Planned initiation is associated with better outcomes, including reduced morbidity, fewer hospitalizations, and improved survival compared with emergency dialysis starts (KDIGO, 2024; National Kidney Foundation, 2023).

18. TYPES OF DIALYSIS IN DIABETIC PATIENTS

18.1 Hemodialysis

Hemodialysis (HD) is the most widely used renal replacement therapy for patients with end-stage kidney disease (ESKD). Blood is circulated through a dialyzer where excess fluid, electrolytes, and metabolic waste products are removed before being returned to the patient. Most individuals undergo HD three times weekly. In patients with diabetes, vascular access complications, peripheral vascular disease, and intradialytic hypotension are common challenges. Despite these limitations, HD remains the standard treatment because of its availability and effectiveness in solute clearance. (USRDS, 2024; Webster et al., 2022)

18.2 Peritoneal Dialysis

Peritoneal dialysis (PD) uses the peritoneal membrane as a natural semipermeable barrier for solute and fluid exchange. Compared with HD, PD provides continuous dialysis and greater hemodynamic stability, making it suitable for selected diabetic patients. However, complications such as peritonitis, protein loss, and glucose absorption from dialysate may affect long-term outcomes and glycemic control. (KDIGO, 2024; Johnson et al., 2024)

18.3 Kidney Transplantation

Kidney transplantation is considered the preferred long-term treatment for eligible patients with ESKD. Compared with dialysis, transplantation offers improved survival, better quality of life, and greater functional independence. However, diabetic recipients remain at increased risk of cardiovascular disease, infection, and recurrence of diabetic kidney disease, necessitating careful selection and long-term follow-up. (KDIGO, 2024; National Kidney Foundation, 2023)

19. Complications of Dialysis in Diabetic Patients

Diabetic patients on dialysis experience higher morbidity and mortality than non-diabetic patients due to the combined effects of hyperglycemia, cardiovascular disease, vascular calcification, neuropathy, and immune dysfunction, requiring multidisciplinary care (USRDS, 2024; Webster et al., 2022).

19.1 Cardiovascular complications

Cardiovascular disease is the leading cause of death. Accelerated atherosclerosis, left ventricular hypertrophy, vascular calcification, and hemodynamic stress during dialysis increase risks of ischemia, arrhythmias, heart failure, and sudden cardiac death (USRDS, 2024).

19.2 Infectious complications

Impaired immunity and invasive access procedures increase risk of vascular access infections, catheter-related bloodstream infections, and peritonitis in peritoneal dialysis, leading to frequent hospitalization and mortality (KDIGO, 2024).

19.3 Anemia and ESA resistance

Anemia is more severe in diabetic dialysis patients due to reduced erythropoietin,

inflammation, iron deficiency, and blood loss. ESA hyporesponsiveness is common, contributing to fatigue and cardiovascular strain (KDIGO, 2024).

19.4 Protein-energy wasting (PEW)

Chronic inflammation, poor intake, metabolic acidosis, and nutrient loss during dialysis lead to malnutrition, sarcopenia, frailty, and increased mortality risk (KDIGO, 2024).

19.5 Vascular and valvular calcification

Disturbed calcium–phosphate metabolism and chronic inflammation accelerate vascular and valvular calcification, causing arterial stiffness, hypertension, reduced coronary perfusion, and heart failure (Floege et al., 2024).

19.6 Progression of microvascular complications

Diabetic retinopathy, neuropathy, and foot complications may continue or worsen despite dialysis, necessitating ongoing glycemic control, eye care, and foot surveillance (ADA, 2025; KDIGO, 2024).

20. CARDIOVASCULAR RISK IN DIABETIC PATIENTS ON DIALYSIS

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality among patients with diabetic kidney disease (DKD) and remains a major determinant of outcomes after the initiation of dialysis. The coexistence of diabetes mellitus and chronic kidney disease creates a particularly high-risk cardiovascular environment characterized by accelerated atherosclerosis, endothelial dysfunction, chronic inflammation, oxidative stress, and vascular calcification. These pathological processes contribute substantially to the increased incidence of coronary artery disease, heart failure, cerebrovascular disease, and peripheral arterial disease in this population. **(USRDS, 2024; Webster et al., 2022; KDIGO, 2024)**

Even before the onset of end-stage kidney disease (ESKD), many patients exhibit structural and functional cardiovascular abnormalities, including left ventricular hypertrophy, arterial stiffness, and impaired vascular compliance. As kidney function declines, disturbances in calcium-phosphorus metabolism, chronic volume overload, anemia, and activation of neurohormonal pathways further increase cardiovascular burden. **(Brenner & Rector, 2024; Floege et al., 2024; KDIGO, 2024)**

Following initiation of dialysis, cardiovascular risk remains exceptionally high. Rapid intradialytic fluid shifts, electrolyte fluctuations, chronic inflammation, autonomic dysfunction, and recurrent myocardial stress contribute to ongoing cardiovascular injury. Sudden cardiac death, arrhythmias, ischemic heart disease, and heart failure are among the most common causes of mortality in dialysis-dependent diabetic patients. **(USRDS, 2024; Daugirdas et al., 2021; Webster et al., 2022)**

Given this elevated risk, comprehensive cardiovascular risk assessment and management are essential components of care. Strategies include strict volume control, optimization of blood pressure, management of dyslipidemia, correction of anemia, control of mineral and bone disorders, and individualized cardiovascular monitoring. Close collaboration between nephrologists, cardiologists, diabetologists, dietitians, and dialysis teams is critical for improving survival and quality of life. **(KDIGO, 2024; American Diabetes Association, 2025; National Kidney Foundation, 2023)**

20.1 Major Cardiovascular Complications in Diabetic Dialysis Patients

- Coronary artery disease (CAD)
- Heart failure
- Left ventricular hypertrophy (LVH)
- Cardiac arrhythmias
- Sudden cardiac death
- Cerebrovascular disease (stroke)
- Peripheral arterial disease (PAD)
- Vascular calcification

21. PREVENTION STRATEGIES TO AVOID DIALYSIS

Preventing progression from diabetic kidney disease to dialysis-dependent kidney failure remains a primary goal of modern diabetes and nephrology care. Because kidney damage often develops silently over many years, successful prevention depends on early identification of at-risk individuals and timely implementation of evidence-based interventions. The greatest opportunity to alter disease trajectory exists during the early stages of diabetic kidney disease, before substantial nephron loss and irreversible fibrosis have occurred. **(KDIGO, 2024; American Diabetes Association, 2025; Tuttle et al., 2022)**

Lifestyle modification is a key foundation for preventing diabetic kidney disease progression. This includes maintaining healthy body weight, regular physical activity, smoking cessation, dietary sodium restriction, and avoiding nephrotoxic agents. Individualized nutrition, including appropriate protein intake and metabolic control, further supports renal and cardiovascular health. Early and sustained glycemic control is one of the most effective strategies to prevent or slow diabetic nephropathy progression (ADA, 2025; KDIGO, 2024).

Strict blood pressure control and reduction of albuminuria are also critical in slowing CKD progression. Current management emphasizes Reno protective therapies, including ACE inhibitors, ARBs, SGLT2 inhibitors, GLP-1 receptor agonists, and non-steroidal mineralocorticoid receptor antagonists, when appropriate, to preserve kidney function and reduce cardiovascular risk (Heerspink et al., 2022; Perkovic et al., 2022; KDIGO, 2024).

Regular screening is crucial for detecting kidney involvement before symptoms appear. Measuring urine albumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR) enables early diagnosis of diabetic kidney disease and timely intervention. Serial monitoring also helps evaluate treatment response and identify patients at high risk of progression to end-stage kidney disease (KDIGO, 2024).

Prevention of dialysis requires a comprehensive, multidisciplinary approach including patient education, lifestyle modification, optimal glycemic and blood pressure control, cardiovascular risk reduction, and evidence-based pharmacotherapy. Early detection and proactive management can significantly delay—and in many cases prevent—the progression from diabetes to dialysis dependence (ADA, 2025; KDIGO, 2024; Tuttle et al., 2022).

21.1 Key Strategies to Prevent Progression to Dialysis

- Early screening with UACR and eGFR
- Optimal glycemic control
- Blood pressure control (<130/80 mmHg when appropriate)
- Reduction of albuminuria
- ACE inhibitor therapy
- Angiotensin receptor blocker (ARB) therapy
- SGLT2 inhibitor therapy
- GLP-1 receptor agonist therapy
- Finerenone in eligible patients
- Smoking cessation
- Weight management and regular exercise
- Dietary sodium restriction
- Early nephrology referral for high-risk patients

These preventive measures target the major metabolic, hemodynamic, inflammatory, and fibrotic pathways responsible for diabetic kidney disease progression and represent the most effective contemporary strategy for reducing future dialysis dependence. **(KDIGO, 2024; American Diabetes Association, 2025; National Kidney Foundation, 2023)**

22. FUTURE DIRECTIONS IN DIABETIC KIDNEY DISEASE

Despite major advances, diabetic kidney disease (DKD) remains a leading cause of CKD, ESKD, and dialysis worldwide. Current research is focused on targeting key mechanisms such as inflammation, fibrosis, oxidative stress, and metabolic dysfunction to slow or alter disease progression (KDIGO, 2024).

Emerging therapies include endothelin receptor antagonists, which reduce albuminuria and may slow progression by modulating vasoconstriction and fibrosis, and agents targeting TGF-β and other profibrotic pathways to limit renal scarring (Kohan et al., 2019). Additional strategies aim to reduce mitochondrial dysfunction and oxidative stress, thereby improving cellular resilience and nephroprotection (KDIGO, 2024).

Anti-inflammatory and regenerative approaches, including stem cell therapy, tissue engineering, and podocyte repair strategies, are under investigation and may eventually offer the potential to restore renal structure and function rather than only slowing decline (KDIGO, 2024).

Precision medicine is also expected to reshape DKD care through genetic profiling, biomarker-based risk prediction, and AI-assisted individualized therapy. Alongside this, digital health tools, remote monitoring, and wearable technologies may improve early detection, adherence, and long-term disease management, ultimately reducing complications and disease burden (Colhoun et al., 2021; KDIGO, 2024).

23. PROGNOSIS OF DIABETIC PATIENTS REQUIRING DIALYSIS

Prognosis in diabetic patients on dialysis is generally poorer than in non-diabetic patients due to higher comorbidity burden, particularly cardiovascular disease, peripheral vascular disease, autonomic neuropathy, and chronic inflammation, all contributing to increased morbidity and mortality (USRDS, 2024; Webster et al., 2022).

Cardiovascular disease is the leading cause of death, with higher rates of coronary artery disease, heart failure, arrhythmias, sudden cardiac death, and stroke driven by diabetes, CKD-related vascular calcification, and dialysis-related hemodynamic stress. Pre-existing cardiovascular disease strongly predicts worse outcomes (USRDS, 2024).

Infectious complications are also common due to impaired immunity and poor wound healing, leading to vascular access infections, peritonitis, pneumonia, and sepsis. Nutritional issues such as protein-energy wasting, sarcopenia, and hypoalbuminemia further worsen survival (KDIGO, 2024).

Despite this, outcomes have improved with advances in dialysis care, cardiovascular risk management, and multidisciplinary approaches. Early nephrology referral, planned dialysis initiation, optimal vascular access, and individualized diabetes control improve survival and quality of life. Kidney transplantation remains the best long-term option, offering superior survival and functional outcomes compared with maintenance dialysis, and should be considered in all eligible patients (KDIGO, 2024; National Kidney Foundation, 2023).

24. CONCLUSION

Diabetic kidney disease remains the leading cause of chronic kidney disease and kidney failure worldwide, accounting for a substantial proportion of patients requiring renal replacement therapy. Chronic hyperglycemia initiates a complex network of metabolic, hemodynamic, inflammatory, and fibrotic pathways that progressively damage renal structure and function. Without timely intervention, these pathological processes lead to albuminuria, declining glomerular filtration rate, chronic kidney disease progression, and ultimately dialysis-dependent kidney failure.

The contemporary management of diabetic kidney disease has evolved considerably over the past decade. Early detection through routine assessment of urine albumin-to-creatinine ratio and estimated glomerular filtration rate, combined with aggressive control of glycemia, blood pressure, and cardiovascular risk factors, can significantly delay disease progression. The emergence of SGLT2 inhibitors, GLP-1 receptor agonists, and non-steroidal mineralocorticoid receptor antagonists has transformed the therapeutic landscape by providing substantial renoprotective and cardioprotective benefits beyond traditional treatment approaches.

Despite these advances, diabetic kidney disease continues to impose a major global clinical and economic burden. Strengthening preventive strategies, expanding access to evidence-based therapies, promoting patient education, and encouraging multidisciplinary care are essential to reducing progression to end-stage kidney disease. Continued research into novel molecular targets, regenerative medicine, and precision nephrology may further improve outcomes and help reduce the worldwide burden of dialysis-dependent kidney failure. (KDIGO, 2024; ADA, 2025).

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