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Diabetes and Kidney Disease: Interconnections, Mechanisms, and Implications for Care

Mugo Moses H.

School of Natural and Applied Sciences Kampala International University Uganda

ABSTRACT

Diabetes mellitus and chronic kidney disease are closely interconnected global health challenges with profound clinical, economic, and societal implications. Diabetes is one of the leading causes of chronic kidney disease worldwide, and diabetic kidney disease represents the most common cause of end-stage kidney disease. The rising global prevalence of both type 1 and type 2 diabetes has contributed to a substantial increase in kidney-related morbidity and mortality, particularly in low- and middle-income countries. The coexistence of diabetes and kidney disease accelerates disease progression, complicates management, and significantly worsens patient outcomes. This comprehensive review examines the epidemiology, shared pathophysiological mechanisms, clinical manifestations, diagnostic approaches, and therapeutic implications of diabetes-related kidney disease. Key mechanistic pathways including chronic hyperglycemia, glomerular hyperfiltration, oxidative stress, advanced glycation end-product formation, renin–angiotensin–aldosterone system activation, inflammation, and fibrosis are discussed in detail. The review also explores diagnostic challenges, emerging biomarkers, and the differentiation of diabetic kidney disease from other nephropathies. Current strategies for prevention and management, including glycemic control, blood pressure and lipid management, renin–angiotensin–aldosterone system inhibition, lifestyle modification, and newer pharmacologic therapies such as sodium-glucose cotransporter-2 inhibitors and glucagon-like peptide-1 receptor agonists, are critically evaluated. Special populations, including pediatric, elderly, and socioeconomically disadvantaged groups, are highlighted to emphasize disparities in disease burden and access to care. The review underscores the importance of early detection, patient education, self-management, and integrated care models. Addressing the growing burden of diabetes-associated kidney disease requires a multidisciplinary approach, equity-focused health policies, and continued research into targeted therapies and early diagnostic tools.

Keywords: Diabetes mellitus, Chronic kidney disease, Diabetic kidney disease, Pathophysiology and Clinical management.

INTRODUCTION

Diabetes mellitus (DM) is a systemic disease characterized by hyperglycemia due to impaired insulin secretion or action affecting multiple organs [1-6]. DM has gained a reputation as a worldwide epidemic with increasing prevalence, especially type 2 DM. In the past decade, chronic kidney disease (CKD), resulting from heterogeneous diseases of the kidneys, independent from DM, has emerged as a serious global health problem [7-9]. Epidemiological studies indicate that there is a significant association between the two diseases DM is one of the leading causes of CKD in many countries [10-14]. Diabetes mellitus leads to the various complications attributed to kidney injury collectively termed diabetic kidney disease (DKD) or diabetic nephropathy (DN). DKD has been investigated intensively from the cellular and molecular aspects and the interconnectivity with cardiovascular diseases at multiple levels [15-19]. DKD induces decreased quality of life, increased health expenditure, and shortened lifespan in a progressively economic-driven world [20-24].

Epidemiology of Diabetes and Kidney Disease

Chronic kidney disease associated with type 2 diabetes mellitus has substantially increased in severity globally, particularly in low- and middle-income countries [25-29]. A comprehensive strategy is urgently needed to curb this alarming rise and to provide integrated management of type 2 diabetes and chronic kidney disease. Type 2 diabetes mellitus, with an estimated 463 million cases worldwide in 2019, has become a century's greatest scourge. The Global Burden of Disease Study covers 359 diseases and injuries and 286 risk factors [30-37]. A detailed epidemiological evaluation of diabetic kidney disease covering the world's situation has not yet emerged. Renal disease due to type 2 diabetes is a heterogeneous disease affecting persons with diabetes of different duration, age at onset, body mass index, ethnicity and sex [38-44]. Because chronic kidney disease attributable to type 2 diabetes is also influenced by socio-demographics, this key point will also be included in the evaluations [45-50]. The global prevalence of type 2 diabetes mellitus (T2DM) has increased due to urbanisation and sedentary lifestyles, with an estimated 451 million people affected worldwide, expected to reach 693 million by 2045 [55-63]. Diabetic kidney disease (DKD) is the most common cause of chronic and end-stage kidney disease and approximately one patient in three with T2DM is estimated to develop DKD [64-68]. The rising prevalence of T2DM and DKD has driven research to better understand their pathogenesis and improve management strategies [69-72]. Preventing microvascular complications requires lifestyle management, regulation of blood glucose and blood pressure, and the use of drugs such as renin-angiotensin system inhibitors [73-75]. Agents that target glucose reabsorption such as sodium glucose cotransporter-2 (SGLT2) inhibitors have shown promise in reducing DKD incidence [76-80]. Hyperglycaemia alone does not fully explain DKD development and other patient-related risk factors such as hypertension, oxidative stress, proteinuria, dyslipidaemia, genetic predisposition, haemodynamic changes, obesity, age, smoking, and possibly gender also contribute [81-83].

Pathophysiological Mechanisms Linking Diabetes and Kidney Disease

Type 2 diabetes mellitus (T2DM) and chronic kidney disease (CKD) are major public health challenges that are closely intertwined; theoretically, expansion in the effective breadth of patented, high-value solutions cannot overcome the financial burden of targeting one of them, if the second one continues on its rapid, rising pace and is unchanged [84-89]. Recent estimates indicate more than 537 million persons are living with diabetes worldwide and around 10.2% of the global population has diabetes [5]. Approximately 20% of diabetic patients develop diabetic kidney disease (DKD). Metabolic dysregulation associated with diabetes leads to glomerular hypertrophy, kidney hypertrophy, and mesangial matrix expansion and can be detected by monitoring urinary albumin excretion. Diabetic patients with macroalbuminuria have a 19-fold increased risk of premature death compared with patients without albuminuria [7]. The development and progression of DKD involve several overlapping pathways, including hyperglycemia, oxidative stress, advanced glycation end-products (AGEs), and activation of the renin-angiotensin-aldosterone system (RAAS)[9]. Hyperglycemia ultimately leads to glucose neurotoxicity, hyperlipidemia, endothelial dysfunction, and altered angiogenesis [2]. Diabetes increases intracellular glucose to cause hyperglycemia; excessive glucose cannot be converted into glycogen and leads an increase in lipids, which will further enhance the processes [3]. Glucose can also activate the polyol pathway through the enzyme aldose reductase[2].

Hyperglycemia and Glomerular Hyperfiltration

Hyperglycemia is integral to the pathophysiological process of diabetic kidney disease. In the early stages of the disease, glomerular hyperfiltration leads to increased glomerular filtration rate (GFR) and increased renal excretion of solutes such as sodium, urea, and phosphate [6]. Glomerular hyperfiltration eventually elevates intraglomerular pressure, promoting damage to the glomerulus and the entire nephron [5]. Glomerular vascular alteration is another pathophysiological mechanism linking hyperglycemia to diabetic kidney disease [7]. Changes in renal hemodynamics, functioning glomerulus, tubulo-glomerular feedback, and renal perfusion pressure elevation increase the risk for sustained elevation of normal-range systemic blood pressure. Sustained mild elevation of systemic blood pressure, in turn, has been linked with the development of diabetes microvascular complications [8].

Advanced Glycation End-Products and Oxidative Stress

Chronic hyperglycemia in diabetes mellitus promotes a cycle of inflammation and damage to proteins, lipids, and DNA through the formation of advanced glycation end-products (AGEs)[8]. Reactive oxygen species (ROS) stimulate AGE production, generating further oxidative stress in a self-perpetuating cycle [7]. AGEs accumulate in many tissues, including kidney, heart, and skin, and several studies have implicated AGEs in diabetic nephropathy [8]. AGEs elicit a variety of cellular effects, primarily through receptor-mediated mechanisms. The main multifunctional receptor for AGEs, RAGE, belongs to the group of immunoglobulin supergene family members [9]. RAGE is widely distributed in endothelial and epithelial cells, neurons, and smooth muscle. Engaging the extracellular domain of RAGE stimulates proinflammatory signalling through intracellular

mitogen-activated protein kinases and transcription factors [10]. As a consequence, proinflammatory cytokines, chemokines, and other degenerative factors are secreted. The lifelong accumulation of AGEs, in parallel with axial skeletal glycation that increases stiffness, might contribute to complications throughout the body [13].

Renin-Angiotensin-Aldosterone System Activation

The RAAS is an important system influencing blood pressure, body fluid and electrolyte balance, water and sodium reabsorption by the kidney, and enhanced aldosterone secretion by the adrenal glands [10]. Activation of the RAAS and formation of angiotensin II (Ang II) and aldosterone occurs in diabetes at an early stage, both before onset of hypertension and before microalbuminuria is apparent [11]. Increased renin production in the juxtaglomerular apparatus, related to glomerular hyperfiltration and glomerular hypertension with dystrophic changes in renal blood vessels leading to nephrosclerosis, has a fundamental role in this involvement [7]. RAAS activation contributes to glomerulosclerosis by stimulating cell proliferation, expanding the mesangial matrix, and inducing production of transforming growth factor-β1 in mesangial cells [8]. Diabetic nephropathy can develop in normotensive type 2 diabetes, associated with activation of the tubulo-interstitial RAAS, juxtaglomerular renin-secreting cells, and other parameters indicative of obstructive sleep apnoea [9]. Ang II has an important role in renal fibrosis [2]. Although excessive hyperglycaemia is regarded as the major factor involved in the development of diabetic nephropathy, it is the RAAS that appears to have a more leading downstream role [10]. The RAAS is on the other hand increasingly recognised as having a protective role in the early stages of type 1 diabetes, protecting against detrimental influences of those not yet active factors and enabling a delay in the onset of overt nephropathy [11].

Inflammation and Fibrosis

Diabetes and kidney disease are two major public health problems, and their interconnection has drastic consequences on their morbidity and mortality [12]. Diabetes increases the risk of developing chronic kidney disease (CKD) by several folds, and diabetic kidney disease in turn hinders the proper management of diabetes [11]. This vicious cycle has drawn the attention of numerous clinicians and researchers, leading to the establishment of the pathophysiological mechanisms linking the two diseases [13]. Understanding these mechanisms prompts the development of targeted treatment strategies to break the vicious cycle [15]. Fibrosis is a common sequela of many kidney diseases and ultimately leads to irreversible loss of function and end-stage kidney disease [12]. Several lines of evidence indicate that the infiltration of proinflammatory macrophages and T cells, as well as fibroblast activation, is associated with the progression of diabetic kidney disease [11]. In murine models, the exogenous administration of macrophage-derived proinflammatory mediators exacerbates glomerular and tubular injury, while their blockage ameliorates injury [12]. Markers of extracellular matrix (ECM) deposition and fibrosis increase, concomitant with the inflammatory process [13]. Thus, inflammation and fibrosis are major processes involved in the progression from diabetes to end-stage kidney disease [7].

Clinical Manifestations and Diagnostic Criteria

Diabetic kidney disease (DKD) manifests as a progressive decline in renal function and increased urinary albumin excretion (UAE), often beginning years after the initial diabetes diagnosis [3]. DKD is characterized as either stage 1 (increased UAE) or stage 2 (normal UAE) [4]. Some patients exhibit glomerular hyperfiltration before developing DKD. The sensitivity of clinical diagnostic criteria for DKD is high; however, knowledge of other primary kidney diseases outside definitions remains inadequate, despite a large minority of patients having concurrent nephropathies, such as hypertension, polycystic kidneys, and focal segmental glomerulosclerosis[5]. After 5–10 years of diabetes, the clinical diagnosis becomes corroborated by evidence of diabetic retinopathy and a 50% decline in estimated glomerular filtration rate (eGFR) from baseline to stage [4, 14]. No singular biomarker captures all aspects of DKD, complicating the diagnostic spectrum [15]. Although estimated GFR (eGFR) and UAE (or albumin-to-creatinine ratio [ACR]) remain the most widely used diagnostic criteria, multiple other clinical or laboratory parameters yield helpful insights into diagnosis and probability assessment [14]. Imaging abnormalities also play a role in nephropathy assessment, with abnormalities closely associated with histopathology and kidney drop-off rates [13].

Diabetic Kidney Disease versus Other Nephropathies

Mild elevation of kidney disease markers is associated with individuals who have diabetes [5]. To accurately diagnose diabetic kidney disease (DKD), it is important to establish that nephropathy is a consequence of diabetes, which can be achieved through clinical history, laboratory evaluations, or kidney biopsy [13]. It is essential to determine whether nephropathy is due to type 1 or type 2 diabetes, as other organs may also be considered in addition to kidney function in type 1 diabetes [13]. The following are some of the remaining clinical and/or paraclinical features of nephropathy, as they allow distinguishing DKD from other nephropathies [16].

Diagnostic Biomarkers and Imaging

Kidney disease develops in at least 30% of patients with diabetes, leading to increased morbidity and mortality during the course of diabetes mellitus [7]. The major early clinical manifestation, diabetic kidney disease, may go unrecognized for years, yet timely intervention can slow its progression significantly [8]. Diagnosis remains challenging despite the clinical utility of traditional markers, such as glomerular filtraion rate and urine albumin excretion, which are often inadequate at initial presentation because of their pathophysiological limitations [10]. Several arrival criteria seek to distinguish diabetic from non-diabetic kidney disease, all with substantial shortcomings [13]. A panel of renal biomarkers predictive of clinical outcomes is under evaluation in diabetes but remains unavailable for clinical application [12]. The difficulties require a personalized diagnostic and monitoring approach that accounts for the heterogeneous diabetic kidney disease spectrum [17]. The clinical remit of chronic kidney disease extends beyond kidney-related events. Renal diseases rank seventh among the 25 leading causes of death for type 2 diabetes, while the global burden of diabetes-associated chronic kidney disease continues to grow, significantly affecting quality of life [18].

Prevention and Risk Modification

An estimated 43 % of adults with diabetes have chronic kidney disease (CKD) attributable to diabetes, which is also the leading cause of end-stage kidney disease (ESKD)[9]. The interconnectedness and various underlying mechanisms between diabetes and kidney disease give rise to major clinical repercussions [1]. Surrogate indicators, such as albuminuria and estimated glomerular filtration rate (eGFR), define early states of diabetic kidney disease (DKD) according to current international consensus [8]. An early recommendation is for clinicians to confirm DKD within six months using quantitative methodologies, especially in a setting where non-diabeteic kidney disease accounts for a meaningful fraction of end-stage renal failure [7]. The pathological aspects of DKD largely overlap with those associated with type 2 diabetes (T2D), and exhibit a different evolution from kidney impairment causing elevation in HbA1c. The principal metabolic disorders of T2D and those relevant to kidney disease progression include obesity, hypertension, and hyperlipidaemia [5]. Each of these parameters needs to be integrated into the overall plan of pharmacological and lifestyle changes aimed at combating diabetes with kidney involvement [3].

Glycemic Control Strategies

Diabetes mellitus and chronic kidney disease (CKD) can interact significantly to modify the effects of glucose on health, whereby proof of the diabetic CVD or the diabetic kidney is still not available for most patients, despite the importance of these comorbidities [6]. Even retinopathy is a less ultimate proof for diabetes-nephropathy-complex in CKD. Thus, any kind of intervention to further lower glucose is therefore typically just a clinical trial, unless already established in the general population [7]. Glycemic control to prevent nephropathy formation still practical in borderline or pre-diabetic hypotriglyceridemia stages without major nephrotoxic co-morbidities [8]. Such burden often warrants immediately combined intervention of glucose-glucose-lowering and concomitant nephro-protecting agents [5]. If nephropathy has officially formed, further lowering typically treatment-formalizes-out-at the investigative, trial-stage already and combined-intervention continues [4]. Commitment-to-continue-usage of drugs safe near-CKD or drug-dose occupations under large oversight industry remain of pivotal importance [4].

Blood Pressure and Lipid Management

Patients with diabetes and chronic kidney disease (CKD) require careful management of blood pressure and lipids to minimize risk of cardiovascular disease [11]. Decreasing blood pressure is critical to reducing cardiovascular risk. Recommendations indicate target blood pressures of less than 140/90 mm Hg for patients with diabetes or end-stage kidney disease and less than 130/80 mm Hg for patients with diabetes and hypertension [1]. Lipid management focuses on reduction of low-density lipoprotein (LDL) cholesterol to decrease risk of atherosclerosis. Statins or other lipid-lowering agents are recommended and treatment decisions should take into account other risk factors and comorbid conditions [6]. Reductions in total cholesterol and triglycerides to recommended levels are also advised. Regular monitoring of blood pressure and blood lipid parameters is important to optimize management of these risk factors [19].

Renin-Angiotensin-Aldosterone System Inhibitors

Renin-angiotensin-aldosterone system inhibitors are widely used to protect renal and cardiovascular health in patients with diabetic nephropathy and other kidney diseases [20]. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) have been shown to slow disease progression and delay the onset of end-stage renal disease [9]. The combination of two agents affording combined angiotensin inhibition has been suggested to provide even greater benefits against diabetic nephropathy [10]. Current blood pressure management guidelines recommend either an ACE inhibitor or an ARB as first-line treatment in the presence of chronic kidney disease, an indication applicable to diabetic patients [12]. These agents are also considered for the

treatment of essential hypertension in individuals with diabetes due to their favorable effects on microvascular complications and foot care [14]. Moreover, aldosterone and the intrarenal renin-angiotensin-aldosterone system have been shown to play a significant role in progressive renal disease, and blockade of this hormone may provide additional therapeutic benefits [15].

Lifestyle and Dietary Approaches

An increasing number of patients with diabetes are at risk for chronic kidney disease (CKD) globally, with its associated high mortality rates [1]. Adopting a healthy lifestyle significantly reduces the risk of CKD [21]. A systematic review demonstrates that lifestyle recommendations from healthcare professionals even when offered as a single session, effectively improve lifestyle practices and reduce body mass index and blood pressure in patients with diabetes [3]. Implementing lifestyle modifications before (primary prevention) and at an early stage (secondary prevention) or delaying progression (tertiary prevention) is recommended for diabetics already diagnosed with CKD [6]. Generic dietary advice needs to be individualized, incorporating personal preferences, cultural values, health literacy, local availability, and economic status [4].

Treatment Modalities for Diabetic Kidney Disease

Treatment of diabetic kidney disease should be coupled with lifestyle modifications and measures targeting risk factors such as poor glycemic control, hypertension, dyslipidemia, obesity, and smoking [13]. Despite extensive research into the pathophysiology of diabetic kidney disease, nephroprotective treatments besides renin-angiotensin-aldosterone-system inhibition have only recently emerged [9]. Patients with diabetes are often underprescribed antidiabetic drugs that are not only efficacious in controlling hyperglycemia but also improve renal outcomes [8]. Furthermore, the traditional understanding that patients with diabetes are ineligible for kidney replacement therapy until the commencement of dialysis is outdated [6]. Clinical practice guidelines indicate that patients with diabetes deserve to receive all modes of kidney replacement therapy [1].

Pharmacologic Therapies beyond RAAS Blockade

Beyond RAAS blockade, additional therapies hold promise for diabetic kidney disease (DKD). Residual risk for progressive kidney disease persists even in patients receiving optimal care with sodium-glucose co-transporter 2 (SGLT2) inhibitors [10]. Following the onset of DKD, accretion of fibrotic tissue and acidosis stem from dysregulated metabolism, making early intervention indispensable [22]. While intensifying anti-hyperglycemia care or combining novel classes of drugs may help, agents that directly counter metabolic and hemodynamic derangements remain advantageous [23]. Agents with favorable metabolic profiles combine with SGLT2 inhibition, allowing the glycemic and weight advantages of DPP4 inhibitors, GLP1 agonists, and insulins to be retained while targeting remaining metabolic risk factors [1]. Kidney damage with diabetes arises primarily from activation of the renin-angiotensin-aldosterone system (RAAS). Frighteningly, the activation of the RAAS accelerates both metabolic and hemodynamic derangements, although early and long-term optimization of GLP1-r or SGLT2-i remains life-saving in type 2 diabetes [10]. The kidney discovers the anticipated intra-renal increase in the calcineurin pathway. Fortunately, preclinical evidence supports that once or twice a week pausing of these therapies promotes life-saving prevention of intra-renal damage also seen with exacerbation of other concerted kidney hazards [11]. Sodium-glucose co-transporter-2 (SGLT2) inhibitors are particularly advantageous since most of the protection conferred by SGLT2-i arises from actions separate from glycemia. Thus, these acts persist even upon termination of anti-hyperglycemia therapy [10].

Glucose-Lowering Agents with Renal Considerations

Certain glucose-lowering agents are contraindicated or require dose adjustment in patients with diabetes and CKD [23]. Metformin causes gastrointestinal adverse effects and lactic acidosis after acute kidney injury, while sulfonylureas and glinides risk hypoglycemia as compensatory insulin secretion decreases with worsening renal function [14]. For SGLT-2 inhibitors, a decrease in renal function leads to a concomitant reduction in glucose-lowering efficacy due to lower filtration of the drug's substrate [13]. For glucagon-like peptide 1 receptor agonists, renal impairment can affect the use of the formulations with a particular route of administration but not the others [12].

Kidney Replacement Therapies in Diabetes

Diabetic patients requiring kidney replacement therapies have a higher risk of morbidity and mortality. Patients with either type 1 or type 2 diabetes experience dialysis at four times the rate of non-diabetic patients [1]. Even after receiving a transplant, patients with either type of diabetes experience allograft loss at a rate greater than their non-diabetic counterparts [16]. Consequently, additional care and caution are urged during the management of patients with advanced diabetic kidney disease needing kidney replacement therapy [17].

Special Populations and Considerations

The distinction between type 1 (T1D) and type 2 diabetes (T2D) creates important differences in presentation, prevention, and management of concomitant diabetic kidney disease (DKD) [16]. Progressive DKD develops in

most individuals with T1D after a duration of 15–25 years; DKD prevalence is approximately 20–40% among persons with T2D. Distinction between T1D and T2D facilitates appropriate selection of glucose-lowering agents, aldosterone blockade, appetite control, and weight-management therapies that specifically address underlying pathophysiologic differences between T1D and T2D [24]. DKD emerges as an important long-term complication of diabetes in youth, particularly in those presenting with T2D or atypical T1D features [1]. Progressing beyond microalbuminuria to overt nephropathy and end-stage renal failure remains rare in childhood [24]. Although renoprotective therapies generally provide protection throughout the life course, the presence of DKD at initial diagnosis of T2D indicates accelerated risk for both macrovascular and microvascular complications and thus mandates urgent intervention often with glucose control, blood-pressure management, and/or RAAS therapy to mitigate overall vascular burden[20]. The growing incidence of T2D among youth raises concern [21].

Type 1 versus Type 2 Diabetes

Diabetes mellitus comprises a heterogeneous group of metabolic disorders characterized by hyperglycemia due to defects in insulin secretion, insulin action, or both (Tuomi et al., 2014)[18]. Type 1 diabetes (T1D) is characterized by the absence or severe deficiency of insulin due to autoimmune destruction of insulin-secreting β-cells in the pancreatic islets (Bonifacio et al., 2021). Type 2 diabetes (T2D) is characterized by insulin resistance in peripheral tissues, often combined with a progressive impairment of insulin secretion (DeFronzo et al., 2015)[17]. The association between chronic kidney disease (CKD) and diabetes is well established, with patients suffering from either T1D or T2D being at high risk of CKD [16]. The underlying pathologic mechanisms differ, as illustrated in box [1]. In T1D, hyperglycemia leads to glomerular hyperfiltration, the earliest manifestation of nephropathy, while in T2D, CKD may develop without any diabetic antecedents because of pre-existing damage to the kidney parenchyma [25]. The nature of hyperglycemia is also recognized as a key driver of diabetes-related complications, including kidney disease: T2D often begins with isolated postprandial hyperglycemia, whereas T1D typically presents with below-average weight and marked elevation of fasting and/or postprandial glucose concentrations [14].

Pediatric and Elderly Populations

Diabetes is the leading cause of CKD in pediatric patients, with DKD reported in approximately 5% of children and adolescents with Type 1 DM 1 and elevated albumin excretion in nearly 40% of such patients into adulthood [20]. DKD represents a complex clinical heterogeneity of kidney abnormalities such as albumin excretion, decline in eGFR, and resultant kidney failure [19]. In older patients with diabetes, the multifactorial pathophysiology involves age-related vascular insults affecting kidney structure and function, and diabetic and non-diabetic factors play contributing roles [24].

Ethnic and Socioeconomic Disparities

Low socioeconomic groups and certain racial/ethnic minorities bear a disproportionate burden of chronic kidney disease (CKD) and its complications. In the United States, CKD is more common in Black, Hispanic, and American Indian populations than in White individuals, and these groups also experience greater disease progression and higher rates of kidney failure [26]. Educational attainment affects kidney and cardiovascular outcomes in a German cohort of patients with chronic kidney disease (CKD) [27]. Educational achievement influences the development of end-stage renal disease in those with type 1 diabetes and the quality of care received by adults with type 1 diabetes but the extent of these effects differs between urban and rural patients [24]. In patients with diabetes, social disadvantage is similarly associated with poorer progression-free survival after initiation of chronic dialysis, and social factors affect both the incidence of end-stage renal disease and post-dialysis mortality [26]. Low socioeconomic status is associated with impaired diabetes control and an increased risk of complications. In people with type 1 diabetes in Ireland, an educational divide contributes to low uptake of insulin pump therapy among disadvantaged groups. Social disparities in diabetes management have been reported elsewhere, notably in Denmark [25].

Healthcare Implications and Policy

Patients with diabetes represent the largest population requiring kidney replacement therapy (KRT) worldwide. In 2021, approximately 50% of new patients starting KRT due to diabetes were accounted for mostly type 2 diabetes, corresponding to reach 200 million of type 2 diabetes patients worldwide [1]. The epidemic of diabetes and the subsequent need for KRT represent a world-wide healthcare challenge that increases the burden on health systems [20]. The detection of diabetic kidney disease (DKD) requires an understanding of the interrelationship between diabetes and kidney disease [24]. Screening recommendations have evolved with the understanding that the risk of DKD differs according to the type of diabetes and the existing treatment [25]. The detection of macrovascular and macrovascular complications of diabetes is important for the prognosis of DKD, the provision of proper health education, and the improvement of self-care practices in individuals with adolescence-onset type 1 diabetes [28].

Screening and Early Detection Programs

Chronic kidney disease (CKD) is a progressive multi-etiologic disease with a compatible genesis that might be independent from diabetic kidney disease (DKD) [14]. In DKD, glycemic control and blood pressure management play major roles in prevention [26]. The attitude toward screening for CKD abilities to anticipate the development of DKD has remained conflicting [25]. Screening program aimed at CKD in patient not only affected DKD prevalence but also showed major changes in some therapeutic habit. Education and training for personnel multiple time you were part of IDF 2003-2017 Management Practices Study riposted similar finding with respect to screening [26].

Patient Education and Self-management

Patient education and self-management are essential in managing chronic kidney disease and diabetes. Strategies include improving medication adherence, diet, and fluid intake, and understanding patient perspectives on multiple medications [27]. Interventions to support self-management focus on promoting lifestyle changes, such as diet and exercise, and addressing nonadherence to treatment regimens. Educating patients about their condition can help improve glycaemic control and reduce complications [29]. The goal of diabetes self-management education is to teach and encourage patients with type 2 diabetes to become actively involved in their care with nurse guidance. Providers' communication on the importance of self-management is critical to achieving treatment and quality-of-life goals [28]. Patient education must be personalized to foster understanding of the disease and improve compliance with monitoring. Developing behaviour and lifestyle changes is often difficult for patients. Regular monitoring of blood sugar, nutrition, and exercise helps lower blood sugar levels and prevent long-term complications [26]. Exercise improves glycaemic control and reduces plasma triglycerides even without weight loss. Diet and physical activity, combined, can delay the onset of type 2 diabetes in at-risk individuals. Self-monitoring of blood glucose and keeping logs of results, nutrition, and feelings support effective self-management [30]. Future research should examine whether diabetes-directed self-management support reduces chronic kidney disease progression in ethnically diverse individuals with albuminuria [30]. In highly comorbid populations such as type 2 diabetes and chronic kidney disease, text-based support can be tailored to individuals' multimorbid disease self-management needs and is scalable for those with limited resources [31].

Access to Care and Affordability

Diabetes and chronic kidney disease (CKD) frequently coexist, which can worsen patient prognosis. Compared to other populations, those with diabetes or CKD frequently experience additional challenges in accessing medical care [29]. The financial burden of costs associated with healthcare coverage and diabetes-related medications serves as an impediment [32-35]. These challenges can be exacerbated within historically disadvantaged communities. Addressing vulnerabilities specific to these and other underserved populations enhances candidate eligibility for diabetes, CKD, and associated complication programs provided by federal, state, and local agencies [33].

Future Directions and Research Priorities

The current landscape of diabetic kidney disease research has been summarised as follows: [1] improving the diagnostic process; [36-40]; and increasing the understanding of underlying pathophysiology [3]. Refining and developing new treatments further specific to the disease [31]. Guideline-directed medical therapies are to be considered where they improve outcomes for the patients affected [30]. Specific attention is warranted to [1]. The use of sodium-glucose co-transporter-2 (SGLT2) inhibitors [32]. Glucagon-like peptide-1 (GLP-1) receptor agonists; [3]. Global usage patterns and adherence to prescriptions of these agents [41, 42]. Segments of the pathophysiological cascade that include hypertension, obesity, inflammation, and those activated by the mineralocorticoid receptor.

CONCLUSION

The interrelationship between diabetes mellitus and chronic kidney disease represents a major and escalating global health concern. Diabetic kidney disease remains a leading cause of end-stage kidney disease, contributing significantly to morbidity, mortality, and healthcare costs. This review highlights that the progression from diabetes to kidney disease is driven by complex and overlapping metabolic, hemodynamic, inflammatory, and fibrotic pathways, rather than hyperglycemia alone. These mechanisms underscore the need for early, multifactorial intervention strategies. Despite advances in understanding pathophysiology and the emergence of novel therapeutic agents, diabetic kidney disease often remains underdiagnosed until advanced stages, limiting opportunities for effective intervention. Traditional diagnostic markers such as estimated glomerular filtration rate and albuminuria, while clinically useful, fail to capture the heterogeneity of disease presentation, emphasizing the need for improved biomarkers and personalized diagnostic approaches. The expanding use of renin–angiotensin–aldosterone system inhibitors, sodium-glucose cotransporter-2 inhibitors, and glucagon-like peptide-1 receptor agonists has significantly improved renal and cardiovascular outcomes, yet residual risk persists.

Management of diabetes-associated kidney disease must extend beyond pharmacologic treatment to include lifestyle modification, patient education, self-management support, and equitable access to care. Vulnerable populations including children, older adults, and socioeconomically disadvantaged and ethnic minority groups bear a disproportionate burden of disease and require targeted strategies to reduce disparities. Future research should prioritize early detection tools, mechanism-based therapies, and health system interventions that integrate diabetes and kidney care. A coordinated, patient-centered, and policy-supported approach is essential to mitigating the growing global burden of diabetic kidney disease and improving long-term outcomes.

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