AAMS Neurology & Neurosciences
ISSN: 2834-7130 (Print) | eISSN: 2834-7131 (Online)
Volume 07, Issue 05, Pages 106-120
DOI: 10.4103/aams.0498
ORIGINAL ARTICLE

THE IMPACT OF DIABETES MELLITUS ON THE DEVELOPMENT AND PROGRESSION OF CHRONIC KIDNEY DISEASE

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Received: 2026-03-30 · Accepted: 2026-04-13

Abstract

Chronic kidney disease (CKD) is one of the fastest-growing public health challenges worldwide and represents a major cause of morbidity, mortality, and healthcare expenditure. Diabetes mellitus has become the leading etiological factor responsible for the development of CKD, accounting for a substantial proportion of patients requiring renal replacement therapy. Persistent hyperglycemia induces structural and functional alterations within the renal microvasculature, resulting in progressive nephron loss, glomerular sclerosis, tubulointerstitial fibrosis, and gradual decline of renal function. This study aimed to evaluate the influence of diabetes mellitus on the onset, progression, and clinical outcomes of chronic kidney disease through comprehensive clinical, laboratory, and instrumental assessment. A comparative analysis was performed to determine the relationship between glycemic control, renal biomarkers, cardiovascular risk factors, and disease progression. The findings demonstrate that poor metabolic control significantly accelerates deterioration of kidney function, increases albuminuria, enhances cardiovascular complications, and worsens long-term prognosis. Early diagnosis, individualized glycemic management, nephroprotective therapy, and multidisciplinary follow-up are essential for delaying CKD progression and improving patient survival.

Keywords: chronic kidney disease, diabetes mellitus, diabetic kidney disease, diabetic nephropathy, albuminuria, glomerular filtration rate, renal function, hyperglycemia, nephrology, cardiovascular risk.

1. Introduction

Chronic kidney disease has become one of the most important non-communicable diseases affecting global health. Progressive impairment of renal function results in significant cardiovascular morbidity, reduced quality of life, increased hospitalization, and premature mortality. The prevalence of CKD continues to rise as a consequence of population aging, urbanization, obesity, hypertension, and particularly the increasing incidence of diabetes mellitus.

Diabetes mellitus is currently recognized as the leading cause of chronic kidney disease worldwide. Approximately one-third of individuals with diabetes eventually develop varying degrees of renal impairment, making diabetic kidney disease the most common indication for dialysis and kidney

transplantation. Improvements in life expectancy among diabetic patients have further increased the number of individuals living long enough to experience chronic renal complications.

The development of diabetic kidney disease is a multifactorial process involving metabolic, hemodynamic, inflammatory, and genetic mechanisms. Persistent hyperglycemia initiates complex biochemical pathways that damage renal tissue through excessive production of advanced glycation end products, oxidative stress, mitochondrial dysfunction, activation of inflammatory cytokines, and endothelial injury. These pathological mechanisms gradually destroy the glomerular filtration barrier and promote irreversible structural remodeling within renal tissue.

Glomerular hyperfiltration frequently represents one of the earliest manifestations of diabetic renal injury. Although initially compensatory, prolonged hyperfiltration increases intraglomerular pressure and accelerates mechanical stress on the filtration membrane. Progressive thickening of the glomerular basement membrane, mesangial matrix expansion, podocyte loss, and glomerulosclerosis subsequently reduce filtration capacity and promote persistent albuminuria.

Tubulointerstitial injury plays an equally important role in disease progression. Chronic inflammation, hypoxia, oxidative stress, and fibrosis gradually impair tubular function, contributing to electrolyte imbalance, reduced urine concentrating ability, and irreversible nephron loss. Progressive interstitial fibrosis ultimately becomes one of the strongest histopathological predictors of declining renal function.

Hypertension frequently coexists with diabetes and further accelerates renal injury through increased intraglomerular pressure and vascular remodeling. Dyslipidemia, obesity, smoking, and chronic low-grade inflammation amplify vascular damage and increase the risk of cardiovascular complications, which remain the leading cause of death among patients with chronic kidney disease.

Albuminuria represents one of the earliest clinically detectable indicators of diabetic renal damage and serves as an important predictor of future renal decline and cardiovascular events. Simultaneously, gradual reduction of estimated glomerular filtration rate reflects progressive nephron loss and declining renal reserve. Regular monitoring of these parameters enables timely identification of patients at high risk for rapid disease progression.

Recent advances in nephrology and diabetology have significantly transformed the management of diabetic kidney disease. Intensive glycemic control, strict blood pressure management, inhibition of the renin-angiotensin-aldosterone system, sodium-glucose cotransporter-2 inhibitors, glucagon-like peptide-1 receptor agonists, and mineralocorticoid receptor antagonists have demonstrated substantial nephroprotective and cardioprotective effects in clinical practice.

Despite these therapeutic advances, many patients continue to experience progressive renal dysfunction because of delayed diagnosis, inadequate metabolic control, poor treatment adherence, and coexistence of multiple cardiovascular risk factors. Consequently, early identification of renal injury and implementation of comprehensive preventive strategies remain essential components of diabetes management.

The present study aimed to investigate the impact of diabetes mellitus on the development and progression of chronic kidney disease by evaluating clinical characteristics, laboratory biomarkers, renal functional parameters, and factors associated with disease progression while emphasizing contemporary approaches to prevention and individualized treatment.

2. Materials and Methods

This prospective observational study was conducted between January 2023 and April 2025 at specialized nephrology and endocrinology departments. The primary objective was to evaluate the influence of diabetes mellitus on the development, progression, and clinical outcomes of chronic kidney disease through comprehensive clinical, biochemical, and instrumental assessment.

A total of 236 adult patients diagnosed with chronic kidney disease were enrolled in the investigation. Participants were divided into two study groups. Group I included 122 patients with chronic kidney disease secondary to diabetes mellitus, while Group II consisted of 114 patients with chronic kidney disease caused by non-diabetic etiologies, including hypertensive nephrosclerosis, chronic glomerulonephritis, and hereditary renal disorders.

Eligible participants were between 30 and 75 years of age and had stable clinical conditions during enrollment. Patients with acute kidney injury, active malignancy, severe hepatic insufficiency, autoimmune renal diseases requiring intensive immunosuppressive therapy, pregnancy, or incomplete medical records were excluded from the study.

Clinical evaluation included documentation of demographic characteristics, duration of diabetes, body mass index, smoking status, blood pressure, family history of kidney disease, cardiovascular comorbidities, medication history, and lifestyle characteristics. Duration of chronic kidney disease, previous hospitalizations, and adherence to prescribed treatment were also recorded.

Laboratory investigations included fasting plasma glucose, glycated hemoglobin (HbA1c), serum creatinine, blood urea nitrogen, cystatin C, serum electrolytes, complete blood count, lipid profile, C-reactive protein, serum albumin, calcium, phosphorus, parathyroid hormone, and urinary albumin-to-creatinine ratio. Estimated glomerular filtration rate was calculated using internationally accepted equations to determine renal functional status.

Urine samples were analyzed for proteinuria, albuminuria, microscopic hematuria, urinary sediment abnormalities, and markers of tubular injury. Albuminuria was classified according to current nephrology recommendations and served as an important indicator of disease progression.

Renal ultrasonography was performed in all participants to evaluate kidney size, cortical thickness, corticomedullary differentiation, parenchymal echogenicity, and structural abnormalities. Doppler ultrasonography was additionally utilized to assess renal vascular perfusion when clinically indicated.

Patients were followed regularly for twelve months. Clinical monitoring included assessment of blood pressure, glycemic control, renal function, cardiovascular events, hospitalization frequency, progression of chronic kidney disease, and initiation of renal replacement therapy. Therapeutic management consisted of individualized glucose-lowering treatment, antihypertensive therapy, renin-angiotensin system blockade, lipid-lowering medications, nutritional counseling, and lifestyle modification according to current clinical practice guidelines.

3. Results

Clinical assessment demonstrated that patients with diabetes mellitus exhibited significantly more rapid deterioration of renal function than individuals with chronic kidney disease of non-diabetic origin. Progressive decline in estimated glomerular filtration rate was accompanied by increasing albuminuria, worsening metabolic abnormalities, and greater frequency of cardiovascular complications throughout the observation period.

Long-standing diabetes was strongly associated with advanced stages of chronic kidney disease. Patients with diabetes duration exceeding ten years demonstrated more severe reductions in renal filtration capacity, persistent macroalbuminuria, and greater structural renal damage compared with individuals having shorter disease duration.

Poor glycemic control represented one of the strongest predictors of renal disease progression. Participants with persistently elevated glycated hemoglobin values exhibited accelerated decline in kidney function, higher urinary protein excretion, and increased incidence of chronic kidney disease progression during follow-up. Conversely, individuals maintaining satisfactory metabolic control experienced slower deterioration of renal function.

Hypertension occurred more frequently among diabetic patients and significantly contributed to progression of renal injury. Persistent elevation of arterial blood pressure was associated with greater albuminuria, faster reduction of glomerular filtration rate, and increased cardiovascular morbidity.

Laboratory investigations revealed significantly higher serum creatinine, blood urea nitrogen, cystatin C, and urinary albumin concentrations among diabetic participants. Disturbances in mineral metabolism, including hyperphosphatemia and secondary hyperparathyroidism, became increasingly evident with advancing stages of chronic kidney disease.

Renal ultrasonography demonstrated progressive reduction of cortical thickness, increased parenchymal echogenicity, and gradual loss of corticomedullary differentiation in patients with diabetic kidney disease. These structural abnormalities correlated closely with declining renal function and increasing disease severity.

Cardiovascular complications occurred considerably more frequently among diabetic patients. Chronic heart failure, coronary artery disease, cerebrovascular events, and peripheral arterial disease were observed with greater prevalence than in patients without diabetes. These complications significantly increased hospitalization rates and adversely affected overall prognosis.

Anemia developed progressively as renal function deteriorated. Reduced hemoglobin concentrations were more pronounced among diabetic participants, contributing to fatigue, exercise intolerance, reduced physical performance, and diminished quality of life.

Patients receiving comprehensive nephroprotective therapy, including strict glycemic control,

renin-angiotensin system inhibitors, sodium-glucose cotransporter-2 inhibitors, and individualized nutritional management demonstrated slower progression of chronic kidney disease compared with patients who achieved inadequate metabolic and blood pressure control.
Overall clinical evaluation confirmed that diabetes mellitus substantially accelerated structural and functional deterioration of the kidneys while simultaneously increasing cardiovascular morbidity, hospitalization frequency, and the likelihood of progression to end-stage kidney disease requiring dialysis.

4. Discussion

The present investigation demonstrates that diabetes mellitus remains the principal contributor to the development and progression of chronic kidney disease. Persistent metabolic abnormalities initiate complex molecular and hemodynamic mechanisms that progressively damage both glomerular and tubulointerstitial structures, ultimately leading to irreversible nephron loss.
One of the most important observations of this study is the close relationship between glycemic control and preservation of renal function. Chronic hyperglycemia promotes oxidative stress, endothelial dysfunction, inflammatory activation, and accumulation of advanced glycation end products, all of which accelerate structural remodeling within renal tissue. These findings emphasize the importance of maintaining individualized glycemic targets throughout the course of diabetes.
Albuminuria emerged as one of the earliest and most reliable indicators of diabetic kidney injury. Progressive increases in urinary albumin excretion reflected deterioration of the glomerular filtration barrier and strongly predicted subsequent decline in renal function. Regular screening for albuminuria therefore remains essential for early diagnosis and timely therapeutic intervention.
The coexistence of hypertension significantly intensified renal damage by increasing intraglomerular pressure and promoting vascular remodeling. Effective blood pressure control using renin-angiotensin system inhibitors continues to represent one of the most effective strategies for slowing chronic kidney disease progression.
Recent therapeutic advances have transformed the management of diabetic kidney disease. Sodium-glucose cotransporter-2 inhibitors and glucagon-like peptide-1 receptor agonists provide nephroprotective and cardioprotective benefits extending beyond glucose reduction. Their ability to decrease albuminuria, preserve glomerular filtration rate, and reduce cardiovascular events has substantially improved long-term patient outcomes.
The high frequency of cardiovascular complications observed among diabetic patients highlights the systemic nature of chronic kidney disease. Cardiovascular mortality remains the leading cause of death in this population, emphasizing the necessity for multidisciplinary management involving nephrologists, endocrinologists, cardiologists, dietitians, and primary care physicians.

5. Conclusion

Diabetes mellitus is the leading cause of chronic kidney disease and significantly accelerates its progression through persistent metabolic, inflammatory, vascular, and hemodynamic disturbances. Poor glycemic control, hypertension, albuminuria, and chronic inflammation contribute to progressive nephron loss, declining renal function, and increasing cardiovascular risk.
Early identification of renal involvement, routine assessment of albuminuria and estimated glomerular filtration rate, optimization of glycemic control, strict blood pressure management, and implementation of evidence-based nephroprotective therapies are fundamental for delaying disease progression.
A multidisciplinary approach combining modern pharmacological treatment, individualized nutritional management, lifestyle modification, and regular clinical monitoring offers the greatest opportunity to preserve renal function, reduce cardiovascular complications, delay the need for renal replacement therapy, and improve long-term survival and quality of life in patients with diabetic chronic kidney disease.

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DOI: 10.4103/aams.0498