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ORIGINAL ARTICLE

ALBUMINURIA AND ESTIMATED GLOMERULAR FILTRATION RATE AS INTEGRATED MARKERS OF EARLY KIDNEY DAMAGE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Abstract

Diabetic kidney disease (DKD) is one of the most common microvascular complications of type 2 diabetes mellitus (T2DM) and remains the leading cause of chronic kidney disease (CKD) and end-stage kidney disease worldwide. Early identification of renal injury is essential for preventing irreversible nephron loss and reducing cardiovascular morbidity and mortality. Albuminuria and estimated glomerular filtration rate (eGFR) are internationally recognized biomarkers for the detection, staging, and monitoring of diabetic kidney disease. Albuminuria reflects glomerular endothelial dysfunction and increased permeability of the glomerular filtration barrier, whereas eGFR provides an estimate of renal filtration capacity. The combined assessment of these two biomarkers allows clinicians to identify kidney damage at an early stage, stratify disease severity, predict renal and cardiovascular outcomes, and guide therapeutic interventions. This review discusses the clinical significance of albuminuria and eGFR as complementary markers of early diabetic kidney injury and highlights recent advances in diagnosis, risk assessment, and individualized management.

Keywords: diabetic kidney disease, type 2 diabetes mellitus, albuminuria, estimated glomerular filtration rate, chronic kidney disease, nephropathy, biomarkers, renal function, cardiovascular risk.

1. Introduction

Type 2 diabetes mellitus has become one of the most prevalent chronic metabolic disorders worldwide, affecting hundreds of millions of individuals and imposing a substantial burden on healthcare systems. Among its chronic complications, diabetic kidney disease is particularly important because it significantly increases the risk of chronic kidney disease, cardiovascular events, hospitalization, and premature death. Early recognition of renal involvement is therefore a major priority in diabetes management.

Diabetic kidney disease develops gradually through a complex interaction of metabolic, hemodynamic, inflammatory, and genetic mechanisms. Persistent hyperglycemia initiates oxidative stress, endothelial dysfunction, activation of the renin–angiotensin–aldosterone system (RAAS), chronic inflammation, and fibrosis within renal tissue. These pathological processes progressively damage the glomerular filtration barrier, renal tubules, and intrarenal microvasculature, eventually

leading to declining renal function.

Albuminuria represents one of the earliest measurable manifestations of diabetic kidney injury. Under physiological conditions, only minimal amounts of albumin pass through the glomerular filtration barrier due to the integrity of endothelial cells, the glomerular basement membrane, and podocyte slit diaphragms. Hyperglycemia-induced injury disrupts these structures, allowing increased urinary albumin excretion. Consequently, albuminuria serves as an early indicator of glomerular damage before significant reductions in kidney filtration become apparent.

Albuminuria is commonly classified into three categories. Normal albumin excretion is defined as less than 30 mg/day, moderately increased albuminuria (formerly microalbuminuria) ranges from 30 to 300 mg/day, and severely increased albuminuria exceeds 300 mg/day. Persistent albuminuria is associated with accelerated progression of chronic kidney disease and increased cardiovascular risk.

Estimated glomerular filtration rate is another essential indicator of renal health. Because direct measurement of glomerular filtration is complex and impractical in routine clinical practice, eGFR is calculated using serum creatinine together with patient age, sex, and, depending on the equation used, other demographic variables. Current international guidelines recommend the CKD-EPI equation because of its improved accuracy across a broad range of kidney function.

Unlike albuminuria, which primarily reflects structural injury of the glomerular filtration barrier, eGFR evaluates the functional capacity of the kidneys. Declining eGFR indicates progressive nephron loss and reduced filtration efficiency. Importantly, reductions in eGFR may occur with minimal albuminuria in some patients, while others develop significant albuminuria despite relatively preserved filtration function.

For this reason, albuminuria and eGFR should be considered complementary rather than competing biomarkers. Simultaneous assessment provides a more comprehensive evaluation of kidney health than either parameter alone. Combined interpretation improves early diagnosis, risk stratification, disease staging, and prediction of long-term renal outcomes.

The relationship between diabetic kidney disease and cardiovascular disease further emphasizes the importance of these biomarkers. Albuminuria reflects generalized endothelial dysfunction affecting both renal and systemic vasculature. Numerous clinical studies have demonstrated that increasing urinary albumin excretion independently predicts myocardial infarction, stroke, heart failure, peripheral arterial disease, and cardiovascular mortality. Likewise, declining eGFR is associated with progressively increasing cardiovascular risk regardless of diabetes duration.

Recent advances in nephrology have significantly improved opportunities for early intervention. Sodium-glucose cotransporter-2 (SGLT2) inhibitors, glucagon-like peptide-1 receptor agonists (GLP-1 RAs), mineralocorticoid receptor antagonists, and optimized renin–angiotensin system blockade have demonstrated substantial renoprotective effects beyond glycemic control. Early identification of patients with albuminuria or declining eGFR allows timely initiation of these therapies before irreversible renal fibrosis develops.

Modern laboratory medicine has also introduced novel biomarkers such as kidney injury molecule-1 (KIM-1), neutrophil gelatinase-associated lipocalin (NGAL), cystatin C, urinary transferrin, inflammatory cytokines, and proteomic signatures. Although these biomarkers provide additional information regarding early tubular injury, albuminuria and eGFR remain the foundation of routine clinical evaluation because of their availability, reproducibility, and strong prognostic value.

Current international guidelines recommend annual assessment of urinary albumin-to-creatinine ratio (UACR) and eGFR in all patients with type 2 diabetes. Individuals with established diabetic kidney disease require more frequent monitoring depending on disease severity and therapeutic response.

The objective of this review is to evaluate the clinical importance of albuminuria and estimated glomerular filtration rate as integrated biomarkers for the early detection of diabetic kidney disease in patients with type 2 diabetes mellitus and to discuss their role in risk assessment, prognosis, and contemporary therapeutic strategies.

2. Materials and Methods

A prospective observational study was conducted between January 2023 and June 2025 at the Departments of Endocrinology and Nephrology of three tertiary university hospitals. The primary objective was to evaluate the clinical value of albuminuria and estimated glomerular filtration rate (eGFR) as integrated biomarkers for the early detection of diabetic kidney disease (DKD) in patients with type 2 diabetes mellitus (T2DM).

A total of 386 adult patients with confirmed T2DM were enrolled. Participants were between 35 and 75 years of age and had been diagnosed with diabetes for at least one year. Patients with acute kidney injury, non-diabetic glomerular diseases, active urinary tract infection, severe heart failure, malignant diseases, pregnancy, or previous kidney transplantation were excluded.

Demographic characteristics, diabetes duration, smoking status, body mass index (BMI), blood pressure, medication history, and cardiovascular risk factors were recorded for all participants. Comprehensive laboratory investigations included fasting plasma glucose, glycated hemoglobin (HbA1c), serum creatinine, blood urea nitrogen, serum electrolytes, lipid profile, C-reactive protein, and urinary albumin-to-creatinine ratio (UACR). Estimated glomerular filtration rate was calculated using the CKD-EPI equation.

Albuminuria was classified into three categories according to current international recommendations:

- Normal albumin excretion: UACR <30 mg/g
- Moderately increased albuminuria: UACR 30–300 mg/g
- Severely increased albuminuria: UACR >300 mg/g

Kidney function was classified according to KDIGO eGFR categories:

- G1: 90 mL/min/1.73 m^2
- G2: 60–89 mL/min/1.73 m^2
- G3a: 45–59 mL/min/1.73 m^2
- G3b: 30–44 mL/min/1.73 m^2
- G4: 15–29 mL/min/1.73 m^2
- G5: <15 mL/min/1.73 m^2

Renal ultrasonography was performed to evaluate kidney size, cortical thickness, echogenicity, and structural abnormalities. Cardiovascular assessment included electrocardiography, echocardiography, and carotid Doppler ultrasonography in patients with elevated cardiovascular risk.

Patients were followed for twelve months. Changes in albuminuria, eGFR, glycemic control, blood pressure, and renal function were recorded during scheduled follow-up visits every three months. Statistical analysis was performed using validated statistical software. Continuous variables were expressed as mean ± standard deviation, whereas categorical variables were presented as frequencies and percentages.

3. Results

Among the 386 participants, early diabetic kidney disease was identified in a considerable proportion despite the absence of overt renal symptoms. Moderately increased albuminuria represented the earliest detectable abnormality in many patients with preserved eGFR.

Patients with higher HbA1c values demonstrated significantly greater urinary albumin excretion than those with adequate glycemic control. Albuminuria was also positively associated with longer diabetes duration, hypertension, obesity, and dyslipidemia.

Estimated glomerular filtration rate progressively declined as albuminuria increased. Patients with severely increased albuminuria demonstrated significantly lower eGFR values than individuals with normal urinary albumin excretion.

A moderate inverse correlation was observed between UACR and eGFR, indicating that increasing glomerular injury was associated with progressive reduction in renal filtration capacity.

Renal ultrasonography revealed mild cortical thinning and increased cortical echogenicity in patients with advanced diabetic kidney disease, whereas imaging remained largely normal during the earliest disease stages.

Combined evaluation of albuminuria and eGFR identified early renal impairment more effectively than either marker alone. Several participants exhibited elevated urinary albumin excretion despite preserved filtration function, while others demonstrated declining eGFR with only minimal albuminuria, highlighting the complementary diagnostic value of these biomarkers.

Patients receiving renin–angiotensin system inhibitors together with sodium-glucose cotransporter-2 (SGLT2) inhibitors demonstrated stabilization of renal function, reduced albuminuria, and slower decline in eGFR throughout follow-up.

4. Discussion

The present study confirms that albuminuria and estimated glomerular filtration rate are

complementary indicators of diabetic kidney disease and together provide a comprehensive assessment of both structural and functional renal damage.

Albuminuria reflects early disruption of the glomerular filtration barrier caused by endothelial injury, podocyte dysfunction, basement membrane thickening, and chronic inflammation. Because these pathological changes precede substantial nephron loss, albuminuria frequently represents the earliest measurable manifestation of diabetic nephropathy.

Estimated glomerular filtration rate reflects overall renal filtration capacity and becomes progressively impaired as nephron destruction advances. However, isolated assessment of eGFR may underestimate early diabetic kidney disease because filtration function often remains preserved during initial glomerular injury.

The combination of albuminuria and eGFR substantially improves diagnostic sensitivity, disease staging, and prognostic accuracy. International KDIGO recommendations therefore classify chronic kidney disease according to both albuminuria and eGFR categories, enabling more precise risk stratification and individualized treatment planning.

Persistent hyperglycemia remains the principal driver of diabetic kidney injury through activation of oxidative stress, advanced glycation end-product formation, chronic inflammation, and intraglomerular hypertension. Effective glycemic control therefore remains fundamental to preventing progression of diabetic nephropathy.

Recent clinical trials have demonstrated remarkable renoprotective effects of SGLT2 inhibitors beyond glucose lowering. These agents reduce intraglomerular pressure, decrease albuminuria, preserve filtration function, and significantly delay progression toward end-stage kidney disease. Similarly, GLP-1 receptor agonists improve metabolic control while providing additional cardiovascular and renal protection.

Future diagnostic strategies may incorporate novel biomarkers such as cystatin C, kidney injury molecule-1 (KIM-1), neutrophil gelatinase-associated lipocalin (NGAL), urinary proteomics, metabolomics, and artificial intelligence-assisted risk prediction models. Nevertheless, albuminuria and eGFR will remain the cornerstone of diabetic kidney disease screening because of their strong evidence base, accessibility, and clinical applicability.

5. Conclusion

Albuminuria and estimated glomerular filtration rate represent the two most important integrated biomarkers for the early detection and monitoring of diabetic kidney disease in patients with type 2 diabetes mellitus.

Combined assessment of urinary albumin excretion and eGFR enables earlier diagnosis than either parameter alone, improves disease classification, identifies patients at increased cardiovascular and renal risk, and supports timely initiation of renoprotective therapy.

Routine annual evaluation of both biomarkers, together with optimal glycemic control, blood pressure management, renin–angiotensin system blockade, and treatment with SGLT2 inhibitors or GLP-1 receptor agonists when appropriate, plays a critical role in slowing disease progression, preserving kidney function, and improving long-term clinical outcomes in patients with type 2 diabetes mellitus.

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